

Socioeconomic Differences in Old Age Mortality in Denmark and the USA

with Special Emphasis on the Impact of Unobserved Heterogeneity on the
Change of Mortality Differences over Age

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Rostock, March 2006

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Mais de ce que nous nous proposons avant tout d'étudier la réalité, il ne s'ensuit pas que nous renoncions à l'améliorer [...] Nous estimerions que nos recherches ne méritent pas une heure de peine si elles ne devaient avoir qu'un intérêt spéculatif. Si nous séparons avec soin les problèmes théoriques des problèmes pratiques, ce n'est pas pour négliger ces derniers: c'est, au contraire, pour nous mettre en état de les mieux résoudre.

(Emile Durkheim)

Der Herr drüben bestellt sich noch ein Bier,
das ist mir angenehm, dann brauche ich mir keinen Vorwurf zu machen
dass ich auch gelegentlich einen zische.
Man denkt immer gleich, man ist süchtig,
in einer amerikanischen Zeitschrift las ich sogar,
jede Zigarette verkürzt das Leben um sechsunddreißig Minuten,
das glaube ich nicht, vermutlich steht die Coca-Cola-Industrie
oder eine Kaugummifabrik hinter dem Artikel.

Ein normales Leben, ein normaler Tod
Das ist auch nichts. Auch ein normales Leben
führt zu einem kranken Tod. Überhaupt hat der Tod
mit Gesundheit und Krankheit nichts zu tun,
er bedient sich ihrer zu seinem Zwecke.

(Gottfried Benn)

Irgendwann sind alle gleich,
Jung und alt, ob arm, ob reich.
Das Schicksal setzt den Hobel an
Und hobelt alle gleich.

(Heino)

Introduction and overview

This dissertation is about socioeconomic mortality differences in old age and the question of how these differences change with age. Social differences in health and mortality constitute a persistent and almost universal finding in epidemiological, demographic, and sociological research. This general finding and the question of why health is poorer and life expectancy lower for people with lower socioeconomic status have been plausibly addressed and discussed by numerous empirical and theoretical studies. However, the diversity of pathways, settings, and mechanisms from social status to health and mortality is still overwhelming.

I start from the well-established finding of social health differences in order to focus on the interplay between class and health in old age (age 59+). Basically the same principles and factors are involved in old age as in other age groups, but old age additionally poses theoretical and practical problems for understanding the interplay between health and social status. The process of aging is not well-defined in biology nor in sociology. It certainly includes the dimension of physical decline, which is similar to a health decline, and the change of the social situation, which interacts with individual subjective perceptions of the body and the environment. The process of aging is very variable and depends on individual socioeconomic status. But socioeconomic status may also depend on the process of aging, e.g., on the level of health and functional ability of a person. The greater need to introduce the health dimension into the consideration of social status makes the study of social inequality in old age different from other ages. The sociological background for the analysis of social differences in old age mortality is the question of whether social inequality as such increases, decreases, or just remains stable in older ages.

In the theoretical part of this dissertation, these aspects and all other important aspects involved in the relationship between socioeconomic status and health will be discussed. For the empirical analysis, Denmark and the USA have been selected as examples of two very different types of countries. Social inequality is much higher in the USA than in Denmark and the level of social security is lower. Denmark and the USA will be treated as two “case studies” where high quality longitudinal data are available, allowing us to discover deep and revealing insights into factors involved in social mortality differences in each country. However, these two countries will not be

“compared” in a strict sense and no hypotheses will be tested concerning the impact of country-specific features on mortality features.

For the USA, I use survey data from the Health and Retirement Study (n=9,376)¹. The Danish data come from the Danish Demographic Database that compiles data from national registers (n=2,029,324). The change of social mortality differences over age will be addressed first on a simple empirical level (meaning, what does the data reveal?) and then on a more advanced level, where measurement problems and possible biases due to unobserved heterogeneity and mortality selection effects are theoretically and empirically taken into account.

The goal is first to present a comprehensive international analysis that is based on appropriate data and methods in order to rule out “avoidable” mistakes and to present results that neither overspecialize nor oversimplify the research topic. Second, this dissertation is an attempt to gain new insight into difficult and experimental questions concerning measurement and statistics. However, the estimation of the impact of unobserved heterogeneity is not a distinct field of research where suddenly numbers assume more importance than information about people, but it is still integral to the evaluation of even the simpler measurements and interpretations. The way unobserved heterogeneity in frailty is defined here has no sociological interpretation as such, but it is crucial to study the interplay between social and biological factors in old age. For this reason, the attempt will be made to draw conclusions from the new and preliminary insights garnered through simulations and experimental modeling in search of the answer to the relatively simple question of whether socioeconomic mortality differences decline with age, and for the more difficult task of explaining the pattern of mortality differences over age.

In the following section, I will give a short overview of the content of each of the ten chapters. More detailed descriptions can be found in the summaries at the end of each chapter.

Chapter 1 presents long-term trends in life expectancy in Denmark, the USA, and Germany and discusses the main contributing factors for the overall mortality decrease. Furthermore, I address the possible principles that may underlie these mortality trends and mention possible conclusions concerning the future trends in life expectancy. Then the mortality patterns of the two countries under study (USA and Denmark) will be

¹ This study consists of the health and retirement study (HRS) and the study on Assets and Health Dynamics among the oldest old (AHEAD).

described. In the case of Denmark where we find a surprisingly high mortality, possible explanations for this exceptional development will be discussed.

Chapter 2 addresses health as a sociological issue. In order to evaluate the importance of health for current debates and the functioning of social institutions, the principles of social security and welfare are presented. Esping-Andersen's classification of welfare states and other international descriptions and comparisons are presented before Denmark and the USA are discussed in greater detail. The last section about pensions connects the welfare state perspective to the notion of an individual socioeconomic status in old age.

Chapter 3 starts at the basis of the sociological concept of social inequality using definitions by Hradil and the comprehensive theoretical framework by Bourdieu. Because there is no explicit theory that includes the two dimensions, health and social inequality, I attempt to ascertain whether the concept of social inequality is open to the inclusion of health as an important social parameter. Social inequality and the meaning of health have to be integrated in order to understand the co-evolution of health and social status during the life course. Therefore, the existing hypotheses concerning the change of social inequality in old age (leveling, maintenance, accumulation) will be discussed using theoretical considerations and empirical evidence. Gender will be presented as a special dimension for the consideration of social inequality that assumes special importance in old age. The level and the kind of social inequality in Denmark and the USA will be described, illustrating the point that these two countries are very different. The various considerations from Chapter 3 concerning social inequality will be summarized and simplified by proposing a practical definition of socioeconomic status that is needed for the empirical analysis.

Chapter 4 first describes socioeconomic differences in health and mortality using findings from the literature. An international comparison, the trend over time, and gender differences will all be presented. The relationship between health and mortality will be discussed before we come to the main section about the causality between socioeconomic status and health or mortality. To present this broad and complex field of research, I chose the following procedure: five categories of causal factors for health are described, and then the concept of "fundamental causes" is presented, followed by a discussion of the hypothesis that income inequality as such influences mortality rather than the individual socioeconomic status. Since both the socioeconomic status and the health status may change and evolve throughout the whole life course, it is worth

considering a life course perspective regarding the question of causation between socioeconomic status and health. Another controversial approach that will be discussed in detail at the end of Chapter 4 is the hypothesis that a considerable amount of causality goes from health to socioeconomic status and not vice versa. Nevertheless, the life course perspective and the reverse causation hypothesis cannot be fully integrated into my empirical study.

Chapter 5 addresses the main research question of my dissertation: how do socioeconomic mortality differences change over age? The chapter is structured as follows: all arguments from the literature speaking in favor of a convergence of mortality differences are presented in the first section, including all relevant findings from the literature. After that, the arguments against a convergence are listed. A third section discusses in greater detail five important research articles that have investigated this question.

Chapter 6 starts out by dealing with empirical questions, namely measurement issues. Between the causality discussed in Chapter 4 and my own operationalization of the empirical data presented in Chapter 7, this chapter discusses various kinds of measurement problems and their consequences for empirical research in social epidemiology. Both measures of predictors and outcome (health and mortality) are presented.

Chapter 7 is the chapter where the two datasets, the data sources, the variables and the exact definitions of all categories are explained. The method of event history analysis is described here before more sophisticated methods and models are applied and explained in Chapter 9.

Chapter 8 contains the results of the analysis of socioeconomic mortality differences. It starts with traditional multivariate mean effect models for both countries and then addresses several interaction effects. The most important of these interactions is the interaction between income and age where the pattern of socioeconomic mortality differences over age can be revealed. Besides the numerous event-history models, Chapter 8 also presents an analysis of socioeconomic differences in health trajectories. The last section of Chapter 8 shows socioeconomic mortality differences separately by cause of death. To my knowledge, this analysis of the Danish data is the most comprehensive analysis of socioeconomic mortality differences in the literature. This is due to the extraordinary data features in terms of data quantity, quality and the number of variables, and to the statistical method of event-history modeling.

Chapter 9 is the most challenging chapter (for the author and for the reader). The concept of frailty and unobserved heterogeneity is presented and it is explained why this can cause a bias in the measurement of socioeconomic mortality differences at different ages. Chapter 9 is devoted to the exploration of this possible bias which can only be estimated, since it eludes measurement. This estimation can be limited by several problems having to do with empirical data. Therefore, one part of the analysis is done with simulated data while the other part is done with the Danish data. The creation of simulated data is explained in detail. Then the different analytical steps to approaching the correct estimation of the bias with frailty models are enumerated. Finally, a new method is proposed that can replace statistical models in cases where the latter cannot be applied because of left-truncation.

Chapter 10 summarizes the most important findings and draws conclusions. Here I elucidate the new insight that this dissertation has generated, and point out the questions that still remain as well as those new questions which have appeared.

The appendix includes additional formulas, an overview of ICD-classifications, the programming code in Stata, and the output of this software for the event history models used in Chapter 9.

Figures and tables are numbered continuously within each chapter, using the number of the chapter as the first digit. The English versions of citations that are originally in German, French or Danish are my own translations.

Chapter 1 Aging and mortality

1.1 Increasing life expectancy

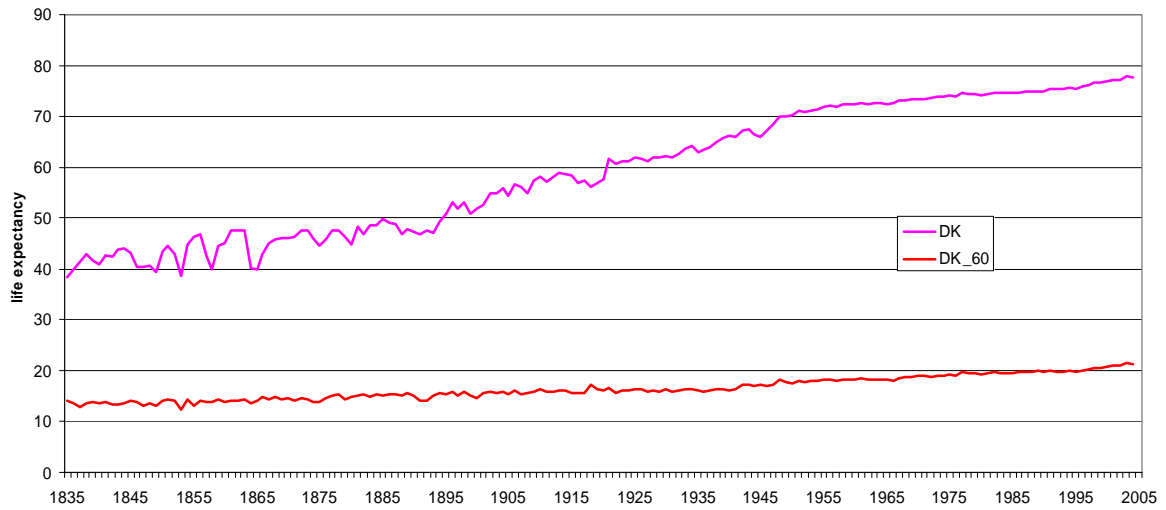
Most countries in the world have aging societies, i.e., populations where the mean age and the share of old people are increasing. The United Nations defines aging societies as societies in which more than 7 percent of the population are 65 years old or older, and aged societies as societies where 14 percent are in this age group. According to this definition, Denmark and Germany are aged societies with their respective percentages being 14.9 and 17.5 in 2003 (World Bank 2004). The USA is still an aging society with 12.4 percent of the population over the age of 64 in 2002 (ibid.) According to the United Nations, the percentage of people in the world above age 60 was 8 percent in 1950, 10 percent in 2005 and is expected to be 22 percent in 2050 (United Nations 2005:13).

This aging process consists of two distinct demographic changes, falling fertility and falling mortality in older ages. The fertility decline is based on the increased use of contraceptives and on the change of lifestyles and values which compromise between family life and childbearing on the one hand and occupational duties, insecurities and individualized self-realization on the other.

Mortality decline in older ages is also contributing to population aging. In fact, in the last few decades it was the main contributor. In the Middle Ages, and perhaps even for many thousands of years before, life expectancy was of 33 to 40 years. The highest life expectancy among countries for which data are available was 38 years in Sweden in 1751 and 44 years in 1840. For women in Sweden in 1840, life expectancy was 46 years. Since this time, life expectancy increased steadily and today the record-holders are women in Japan with a life expectancy of 85.6 years. This is a remarkable increase of 40 years of life in just a 160-year time span (Oeppen and Vaupel 2001).

Almost all countries, even very poor ones, exhibit an increasing life expectancy. The exceptions are countries with a high HIV rate and some Eastern European countries, especially Russia, that still suffer from a transition crisis. Figure 1.1 shows the increase in life expectancy at birth and at age 60 in Denmark from 1835 onwards.

Figure 1.1: Development of life expectancy at birth and at age 60 in Denmark, 1835-2004

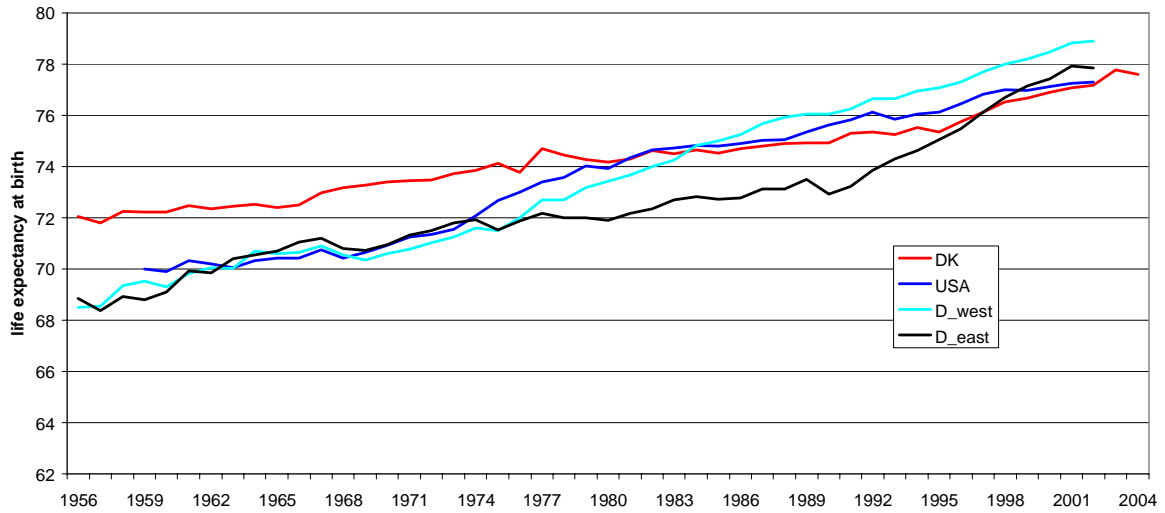


Source: Human Mortality Database²

The increase in life expectancy at birth in Denmark is enormous. It more than doubles from 1835 until 2004 and increases on average almost 3 months every year. The increase is steeper in the first part of the 20th century and slower in the second part. Naturally, the remaining life expectancy at age 60 increases more slowly. The next figure compares the life expectancy at birth in Denmark with the USA and Germany (East and West) during the period for which data is available for all three countries.

²The Human Mortality Database (HMD) is a high quality collection of recent and historical data run by the University of California, Berkeley and the Max Planck Institute for Demographic Research. It is freely available under www.mortality.org.

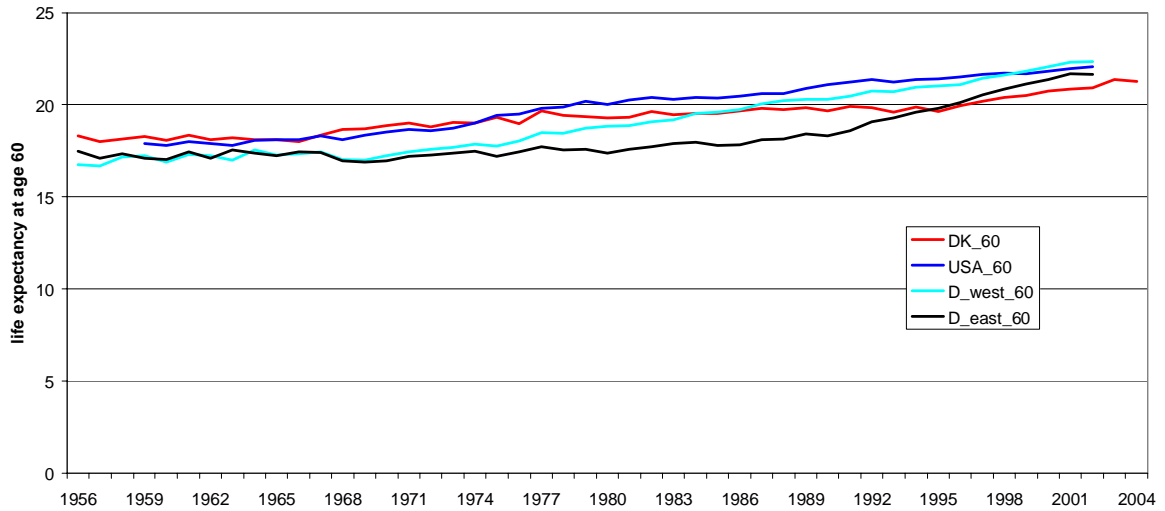
Figure 1.2: Development of life expectancy at birth in Denmark, the USA and Germany



Source: Human Mortality Database

Comparing Denmark and the USA, Denmark had a clear advantage in life expectancy from 1950 until 1980. After that, they had similar levels of life expectancy and in Denmark the increase slowed down, especially compared to West Germany. The line for Denmark shows that it lost the leading position and that since 1997 it has had a lower life expectancy than the USA and Germany. The opposite is true for West Germany: from the lowest position in 1956 it leapt ahead and since 1985 has had the highest life expectancy by far. Another remarkable pattern is shown for East Germany: since the middle of the 1970s, life expectancy increased much less than in West Germany until a maximum difference of 3.1 years was reached in 1990. After German reunification, the rate of increase was even higher than in West Germany with the consequence that life expectancy converged rapidly between East and West Germany.

What is more relevant for our analysis of old age mortality is life expectancy at age 60 because our datasets include persons aged 59 and older. The following figure, Figure 1.3, shows the same comparison for old age mortality.

Figure 1.3: Development of life expectancy at age 60 in Denmark, the USA and Germany

Source: Human Mortality Database

Most features of old age mortality are very similar to mortality at all ages, as was shown in Figure 1.2. Denmark starts with the highest life expectancy and ends up with the lowest and the opposite is true for West Germany. East Germany loses its relative position already at the beginning of the 1970s and catches up after reunification. An interesting difference between old age and all age mortality can be observed for Denmark and the USA: life expectancy at birth converged strongly between the USA and Denmark (Figure 1.2) whereas life expectancy at age 60 diverged from the late 1970s onwards (Figure 1.3). Besides the possibility that life expectancy in the USA is overestimated because of unreliable data sources, two explanations are possible: either the USA developed a mortality pattern that was rather advantageous for the elderly, or Denmark developed a pattern that implied a relative disadvantage for the elderly. The first explanation seems to have more influence because since around 1980 there is a particularly favorable trend for old age mortality in the USA compared to the overall mortality level in the USA. Of course, these considerations are based solely on the comparison of three countries and are therefore limited. Further discussion of the mortality trend in Denmark will be done in Section 1.3.

The mortality decline is due to many different cultural changes: the technical and medical ability to prevent and heal illnesses has increased enormously since the late nineteenth century, e.g., with the discovery of the tuberculosis pathogen in 1882 and penicillin in 1928. But the historical perspective shows that the overall rising living standard, the improvements of sanitary conditions, diet, education and social security

since the early eighteenth century as a consequence of the industrial revolution, is probably the earlier and more important factor for an overall increasing life expectancy (Beckett 2000:116). Vincent (1995:130f) summarizes the most important factors for the increase of longevity as “peace, potatoes and penicillin” claiming that the social situation, lifestyle and diet contribute more to longevity than medicine does. Among the improved social factors, increasing education may have been of major importance (Ross and Wu 1996:116; Himes 2000:80). More and more old persons are well-educated (Preston 1992:53), resulting in better overall health behavior.

At the beginning of the long period of increasing life expectancy, it was the decline in infant mortality which contributed most to the improvement in life expectancy. Statistically, the saved life of a baby contributes more to the overall life expectancy than the delayed death of an old person. But in the last several decades, infant mortality in rich countries has remained at such low levels that further improvements are difficult to achieve. In the last decades of the long period of mortality decline most improvements happened in old age mortality (Kannisto 1994; Vaupel et. al. 1998), which has become tractable and plastic (Vaupel 1998). Between 50 and 75 percent of the improvements in mortality are due to the decrease in the number of deaths from cardiovascular diseases that occurred in most developed countries (Jeune 2002:79).

A result of declining mortality in old age is that centenarians are the fastest growing age group in the population (Vaupel 2000). For Denmark, where good data are available, an average of 3 people per year reached the age of 100 in the decade of 1870. In 1970 there were already 43 new centenarians per year and in 1999 the number was 254. It is likely that before the nineteenth century there were no centenarians at all in a country of the size of Denmark and that reports about persons of that age are not true (Vaupel 2001).

These improvements, especially in old age mortality, point to an important factor that has to be considered in the discussion of decreasing mortality: the biological plasticity of the aging process of humans which seems to allow them to reach very high ages if the living conditions are good. This genetic and evolutionary ability has not been totally explained yet. There are several plausible models for the mechanisms of aging but no generally accepted biological theory of aging that allows us to understand exactly why and how humans and other species are aging (Vincent 1995:16). Further without a theory we do not know exactly what explains the amazing increase of human life expectancy on the one hand and the large and persisting inter-individual differences in lifespan on the other.

The interrelationship of aging and health is not well understood either. Aging is not just decreasing health; to some extent healthy aging is possible. Eventually even a healthy life will lead to death just as an unhealthy life does. On the other hand, a health decline in older ages is very likely because genetic replication, cells, tissue, organs and whole systems become more and more defective. Time, i.e. numerical age, is one factor for this decreasing robustness but the more we know about the concrete influences on aging and health the more specific exposures to risk factors are known. This knowledge of concrete causes and mechanisms may gradually replace the vague impact of time and age in our understanding and may help to avoid many of the reasons why people currently die (Ukrainitseva and Yashin 2001).

There are large gender differences in mortality. In all populations and almost all circumstances women have lower mortality than men (Verbrugge 1989; Federici et al. 1993; Luy 2002). This difference has a biological and a social component, i.e., female roles and behavior in society seem to be less harmful than the male lifestyle. Moreover, in most cases women have profited more from the mortality improvement than men, as their gains in life expectancy are higher (Myers 1996; Vaupel 1998). The gender difference in life expectancy was 1.8 years in 1920 and 8 years in 1970. Now it is slightly lower, remaining relatively stable in the range of 6 to 7 years (Hummer et al. 1998b:558; Liang et al. 2002:294). Naturally, this influences the gender composition of the population in higher ages: e.g., in the USA, there are about 50 percent more women than men at ages above 65 and three times more women at ages 85 and above (Arber and Ginn 1993:34).

Decreasing mortality does not necessarily imply an aging society, because it depends on the age group where mortality decreases. But the change of mortality that happened in most developed countries in the last decades has led to an aging population, which is perceived as a problem in many social and political fields. A contradiction is evident: technically we enable longer life and individually longer life in good health is attractive. But culturally and socially we define this as a problem because having a larger share of elderly persons in a population requires more care and financial support. It may also represent and necessitate a lifestyle that is very different from the generally accepted youth-orientated lifestyle (Fry 1996:123ff). The structure of the population in terms of age is related to the social structure and many sociological questions arise from the aforementioned changes. The increase of life expectancy has implications for one's individual life course, since it now has to be planned differently. On the aggregated

level, namely in an aging society, changes to the age and social structures generate significant implications for the welfare system (Kunst et al. 1998b).

In a democratic system the elderly will have more power and influence; however, this can be opposed by a possible increase of hostility and exclusion toward older persons (Backes 1997). The growing share of elderly persons could lead to this age group having their worse relative position (Vincent 1995:125). The question of whether there will be less age segregation and more age integration is not a simple consequence of the demographic change, but rather a question of how social negotiations and norms adjust to demographic developments (O’Rand et al. 1999:213). Social problems generated by the mere fact of having a greater proportion of elderly in society do not exist per se (Vincent 1995:126), but are – at least to a large extent – the result of a conflict between structural changes and value changes. Values like independence, youth, beauty and high performance in all areas of life are highly appreciated, but a change in the age structure of the population will increasingly make aging and functional limitations an integral part of everyday life.

The shift from a work-based to a consumption- and leisure-based society, which is related to the demographic change, does not only depend on the availability of resources but also on a fundamental change of values (Kohli 1990:389). In a society and within the life course, the distribution of work and the relative importance of one’s occupation may change (Berger et al. 2001) and develop towards a model that Dahrendorf (2003) called “Tätigkeitsgesellschaft” (a society based on activity) in contrast to the “Bezahlte-Arbeit-Gesellschaft” (a society based on paid work)(Kreckel 2004:33).

The mortality decline and improvements in life expectancy do not just make our lives longer because people are prevented from dying. The demographic trend in both the EU and in the USA shows not only declining mortality but also less disability (Lee and Edwards 2001) and improving overall health (Ziegler and Doblhammer 2005a). Thus the factors mentioned above, contributing to increasing life expectancy, seem to contribute to well-being, too, and investments in well-being are very likely to further expand our life span (Vaupel 1998).

1.2 Limits to life expectancy and the compression of morbidity and mortality

There is some uncertainty concerning the uppermost limit human life expectancy could increase to in the future, if living conditions and medical interventions continue to improve.³ Older and conservative estimates of the future development of human life expectancy still predict a leveling-off based on the assumption that humans are approaching a biological limit for their lifespan. Such upper limits have been hypothesized several times in history and very often such claims did not survive the actual increase of life expectancy for many years. If there is such a biological limit, it still seems to be quite far away. Since 1840, there is a linear increase in the maximum life expectancy recorded (i.e., the life expectancy of the country with the highest life expectancy) (Oeppen and Vaupel 2002:1029). After some years another country may take over the position of the record-holder because in a single country the trend is not linearly increasing. But several countries, one after another, line up in a straight increasing line. This trend does not show any signs of bending down or approaching a limit. Moreover, the increase is not slower but in some cases even faster in countries that already demonstrate a high life expectancy. This also speaks against being near the biological limit (Martelin et al. 1998:89; Vaupel 1998:243; Vaupel 2001). Based on these empirical findings it is likely that mortality will continue to decline (Lynch 2001:81).

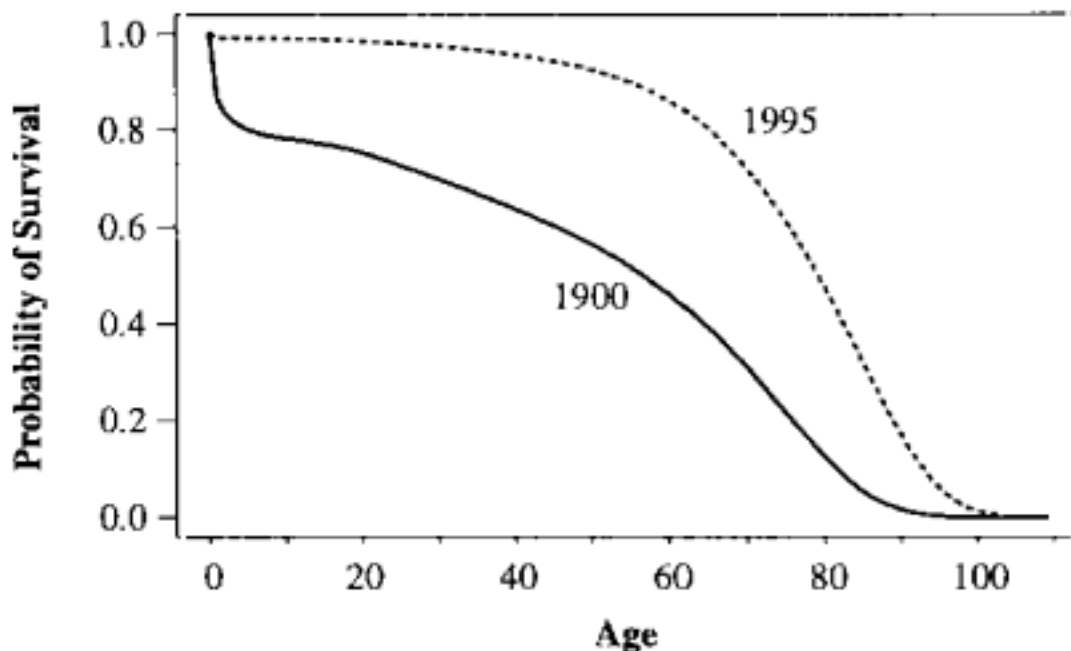
In many developed countries more than half of all women and more than one-third of all men die over the age of 80 (Manton et al. 1995). If mortality continues to decline, 50 percent of today's female newborns in rich countries like France, but probably also in Germany, Denmark or the USA, will reach their 100th birthday (Vaupel 2001).

A topic that is related to possible limits of human life expectancy, as well as to the question of how the length of life is distributed in the population and between social groups, is the "compression of mortality". It means that the variance of the age at death in the population is decreasing. This is equivalent to the so-called "rectangularization" of the survival curve, shown in Figure 1.4. Many people survive until rather old ages, and then the survival curve declines steeply, which means that in a narrow age range all persons will die (Fries 1996; Klein 1999:450). An analogous concept is the compression

³ For an overview of different perspectives on limits of human life expectancy, see Manton et al. 1991.

of morbidity which suggests that the period of poor health before death is compressed. People enjoy good health until they encounter a relatively sudden health decline and subsequent death (Kunst et al. 1998b; Lampert and Maas 2002).

Figure 1.4: Rectangularization of the survival curve of women in the USA between 1900 and 1995



Source: Wilmoth and Horiuchi 1999:477

There is a large body of literature discussing these suggestions and trying to find empirical evidence for or against compression. Findings are inconsistent with regard to the age at onset of morbidity in different decades (Crimmins et al. 1994:160). There is evidence for compression as well as for enlargement (Ross and Wu 1996; Doblhammer and Kytir 2001; Lynch 2001; Cheung et al. 2005). A very recent branch of the discussion suggests that there has been some compression, but for several decades now and continuing into the future, the survival curve will not change its shape further but will continue to shift to older ages (Canudas-Romo 2005).

Without showing supporting and opposing empirical results on this topic,⁴ I will mention three important questions related to the compression hypothesis:

⁴ A simple analysis of the change of the standard deviation of age at death in Denmark based on my Danish data set shows that the standard deviation is increasing from 1980 to 2000 for all deaths that occur above age 59. However, the coefficient of variation, which is the standard deviation divided by the mean, is slightly decreasing, because the mean age at death was also increasing in this period from 76.9 to 79.9.

1. If there is compression of morbidity, the revolutionary gain in life expectancy would increase the length of healthy lives more than the number of lives in poor health. There are indications that the gains in life expectancy since 1970 imply a higher proportion of healthy life expectancy (Hayward et al. 1998). Manton (1993) says that we have turned the relationship between life expectancy and health in the 1970s around, i.e., that longer life indeed implies better health (also see Hayward et al. 1998:212 and Dinkel 1999). This diagnosis is important for judging a possibly increasing burden of disability on individuals and society (Kunst et al. 1999; Ziegler and Doblhammer 2005b).

2. Originally, the idea of a rectangularization of the survival curve was interpreted as support for the existence of a near absolute limit of human life expectancy. If there is a maximum life span and if we approach this upper limit, then the rectangularization would show that the population is becoming homogeneously robust, the share of frail persons is getting smaller with most people dying near the upper limit of human life expectancy (Lynch 2001:93). As a model this is plausible, but the empirical evidence for the assumption that we are approaching a limit in life expectancy is missing, as has been mentioned above. Best-practice countries in terms of life expectancy do not show a slower increase in life expectancy than other countries.

3. The model of rectangularization is important for the study of social differences in health and mortality because a narrow range of ages where almost all people die is exactly the opposite of what many studies about socioeconomic mortality differences reveal: increasing rather than decreasing differences. Many findings suggest that compression happens socially different, being more evident for richer persons (House et al. 1994:214; Lampert 2000; Liang et al. 2002:305). People with higher social status are more likely to postpone the onset of diseases and to die in relatively good health whereas poor people suffer a longer period of bad health before their death at younger ages (Huisman et al. 2003:862).

After the description of general trends in mortality and the related questions, the next two sections will describe mortality in the USA and Denmark.

1.3 Mortality in the USA

The country-specific mortality of the USA shares some features with other developed countries, for example, in the transition from acute and infectious to chronic diseases as the leading cause of death or, more generally, the transition from contagious to degenerative diseases (Myers 1996). Besides that, the USA has some different mortality features: in many comparisons of rich countries concerning mortality, the USA is the richest. In a global perspective, richer countries often have higher life expectancy, but this is not a perfect correlation. For example, the USA has a relatively low life expectancy among rich countries (Kawachi and Kennedy 2001:16). In contrast, Costa Rica, which has only 10 percent of the GDP per capita of the USA, has almost the same life expectancy as the U.S., respectively 76 and 76.7 years at the end of the 1990s (Lardner 2001:87). Sweden e.g., has a life expectancy of 80 years, which would be like the USA with no heart attacks (Lardner 2001:87).

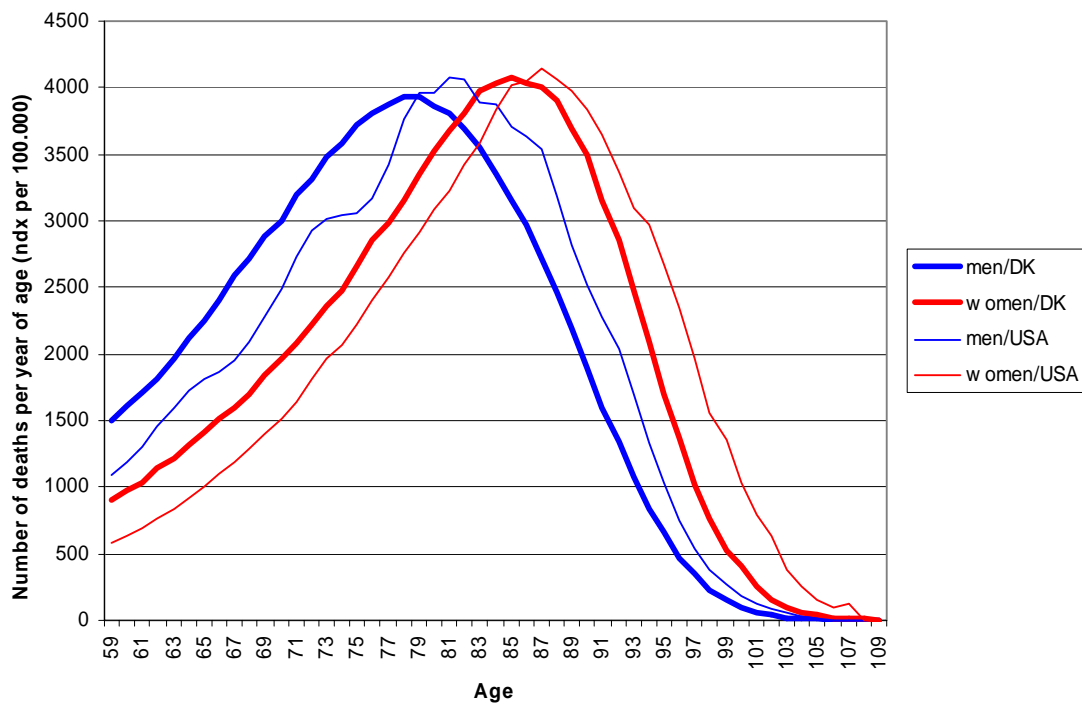
In the USA, there is a special age pattern for mortality: middle age mortality is higher than the average in the EU countries and old age mortality is lower (Vaupel 1998). Old age mortality in the USA may have been the lowest in the world up until the middle of the 1990s (Hummer et al. 1998b:571). Figure 1.2 and Figure 1.3 show that the level of old age mortality is more favorable for the USA than the level of overall mortality. Remaining life expectancy at age 80 was 8.5 years in 1999 in the USA, which was higher than in Sweden (8.1), Germany (8.0), Netherlands (7.8), Norway (7.8) but still lower than in France (8.7) and Japan (9.2)(Human Mortality Database).

Possible reasons for this difference in the relative mortality level between middle and higher ages are the following: first, compared to middle ages, older persons in the USA have better health insurance coverage (see section about social security). Second, older persons in the USA get high quality health care. Third, they are better educated than in other countries because when they were young the educational level in the USA was better than in many other countries. Therefore, they adopt healthy behavior more easily. Fourth, there are many immigrants to the USA which, in middle age, live in more unhealthy circumstances and have high mortality just as many other Americans have. This higher mortality in middle age has a selective effect that leads to a select and robust old population (Manton and Vaupel 1995).

The figures in Section 1.1 above compare the two countries under study as well as Germany in terms of life expectancy at different ages in order to give an overview of

differences in mortality at all ages and in higher ages. Figure 1.5 compares another mortality feature between Denmark and the USA, namely the life table distribution of deaths over different ages for each sex in both countries. This figure is based on the empirical data that will also be used in the empirical part. The data sets will be described in more detail in Chapter 7. Here it is noteworthy that all four distributions have a similar shape, but the age when most people die differs between the two countries and between genders. Overall women die in higher ages compared to men. Furthermore, elderly people in the USA die at higher ages than Danish elderly. Mortality differences between males and females (blue and red) are larger than differences between the two countries (thick and thin lines).

Figure 1.5: Life table density function for Denmark and the USA by gender



Note: Due to low case numbers from the HRS data and consequently a much-disrupted pattern in the curve, it was necessary to smooth the curves for the USA by employing a standard method for such data problems. This has been done with a Penalized Maximum Likelihood assuming a Gompertz distribution of the hazard.

1.4 Mortality in Denmark

As in all developed countries, life expectancy in Denmark has risen tremendously since the 19th century. In Denmark, life expectancy was a mere 38.4 years in 1835 but it shot up to 77.6 in 2004 (Human Mortality Database). As indicated above, old age mortality is higher in Denmark than in the USA. Due to a high female mortality at all ages, middle-aged women in Denmark also have higher mortality than women in the USA (Myers 1996). Besides having a relatively high mortality rate for women, Denmark shows another negative feature compared to other rich countries: life expectancy in Denmark almost stagnated from 1975 to 1995 (at least for women), because improvements in old age, comparable to other European countries, were outbalanced by problems at middle ages (Andersen and Laursen 1998; Brønnum-Hansen 2000; Jeune 2002:78). “A comparable stagnation is not seen in other western European countries” (Sundhedsministeriet (Danish Health Ministry) 1994a:102).

Andreev (1999) shows that the excess mortality in Denmark, compared to Sweden, Netherlands and Japan, occurred mainly among the middle-aged. Main causes of death for this excess mortality are lung cancer, breast cancer and respiratory diseases. Mortality from ischemic heart diseases in the period from 1975 to 1995 shows the same level and the same decline as in Sweden, in middle ages as well as in old age. This decline fits in the overall decline of cardiovascular diseases that was mentioned in Section 1.1 as the most important factor for the overall mortality decline. The underlying decline of risk factors that may have contributed to these improvements has been investigated in an epidemiological study in Denmark:

“According to comparisons of 70-year-old Danes from three different cohorts born in 1897, 1914 and 1921 participating in the Glostrup Population Studies [Sjol et al. 1998; Thomsen 1999] both the systolic and the diastolic blood pressures decreased significantly from the 1960s to the 1990s in both genders, and the proportion of medically treated hypertensive cholesterol decreased in both genders by about a quarter from the 1960s to the 1990s [Thomsen 1999; Sjol et al. 1991]. These declining trends in cardiovascular risk factors have been observed in most low-mortality countries.” (Jeune 2002:80)

Contrary to this favorable trend for the elderly, the trend in middle age mortality is rather negative in Denmark. The comparison to other European and other countries of the world shows that from 1950 until 1970, Danish life expectancy at birth was higher than in Belgium, France, West Germany, England, Italy, Spain, Japan, Australia, New

Zealand and the USA. From 1975 to 1990, almost all of these countries overtook Denmark in terms of life expectancy (Sundhedsministeriet 1994a:25ff).

What is responsible for this relatively negative trend in middle-age mortality in Denmark? Two possible reasons are discussed in the literature: alcohol and tobacco consumption. These two factors are closely linked to and can serve as an illustration of the study of socioeconomic mortality differences because consumption patterns may be a causal link between socioeconomic status and health.

Denmark has a much higher alcohol consumption per capita than the other Scandinavian countries, but a lower consumption than United Kingdom and France (Sundhedsministeriet 1994b:55). The long term development of the alcohol consumption in Denmark shows that the consumptions declined steeply from 1910 to 1940 and increased again until 1990 to about the level of 1910. In 1987 in the age group 67+ about 42 percent of Danish men and 24 percent of Danish women had one or more drinks per day (Sundhedsministeriet 1994d:22). Unlike one would expect, the social gradient of drinking behavior shows that men and women in higher occupational groups drink more alcohol (*ibid.*) This is probably due to relatively high alcohol prices in Denmark.

Concerning health damage due to alcohol, it is important to consider the levels of consumption of beer and hard alcohol in Denmark, which have been more or less stable after 1975 in contrast to the steeply increasing consumption of wine (*ibid.*:30). Although alcohol consumption as such is a health risk, in many studies a moderate level of wine consumption is associated with better health. But “it is still a matter of discussion whether the benefit of wine could be due to ethanol itself or to non-ethanol beneficial effects of antioxidants in red wine, or to a healthier lifestyle among wine drinkers” (Jeune 2002:80, for literature see *ibid.*) Typical wine drinkers also eat more healthy food like fruits, fish, cooked vegetables, salad and olive oil (Tjonneland 1998). The sharp increase in alcohol consumption starting in the middle of the century is associated with an increased alcohol-related mortality (liver cirrhosis) starting with a time lag of about two decades (Sundhedsministeriet 1994b:77).

Maybe smoking is even more responsible for Denmark’s mortality disadvantage than drinking. High mortality, especially for Danish women, has been explained by the high percentage of smokers among them (Christensen 2001:106). The proportion of male

smokers in Denmark decreased from more than 70 percent in the 1960s to less than 40 percent in the year 2000. The share among women decreased from more than 50 percent to 30 percent (Jeune 2002:80; see also Sundhedsministeriet 1994d:28). This decrease seems to be contradictive to the contribution of smoking to excess mortality. But perhaps women born between 1915 and 1945 smoked more than other women before and during the general decline in smoking, and consequently they show a higher mortality throughout the life course. Even if people quit smoking they may die of the consequences later. While the number and the percentage of smokers decreased, the amount of tobacco that was smoked increased (Sundhedsministeriet 1994d:28). This indicates that the remaining smokers consume much more than in earlier years.

Results from the Danish Health Ministry (1994d:20) show that at the beginning of the 1990s the age group with the highest smoking prevalence was ages 40 to 49 for males (almost 60 percent smokers) and ages 25 to 39 for females (almost 50 percent smokers). In the oldest age group of age 65+ they find almost 50 percent smokers among men and more than 30 percent among women. In Denmark, men and women smoke more than in other countries (Sundhedsministeriet 1994b:53).

Concerning the health damage caused by smoking, the international comparison of smoking-related mortality clearly mirrors the very high smoking rates in Denmark compared to other countries (Sundhedsministeriet 1994b:74).

Summary of Chapter 1

The increase of life expectancy is a general trend in most countries. There are medical but also cultural and social causes for increasing life expectancy. It is unknown if there is an upper limit of human life expectancy and where this limit could be. During the increase of life expectancy the mortality pattern can change towards a rectangularization of the survival curve and a compression of mortality. The changing age structure of a society will also have social consequences that affect the distribution of resources and the value system. The comparison between Denmark and the USA shows that the latter has a higher life expectancy and a more favorable trend during the last decades. In the USA, middle ages are characterized by a relatively high mortality while older ages are characterized by a relatively low mortality. Compared to other European countries and the USA, Denmark has a worse mortality trend since the 1970s, especially for women. The excess mortality occurs for middle ages and may affect mostly the cohorts born between the two World Wars. Among other factors, Denmark's mortality disadvantage can be attributed to the consumption of alcohol and tobacco since Denmark shows higher levels than many other countries.

Chapter 2 Underlying features of social differences in health and mortality

Health is a central category in the analysis of mortality and aging. Except for accidents, murders and suicides, death is the end of a process of increasing age and changing health, usually of declining health. Thus, health is a good measure for the status an individual has in this process in which the tendency of declining health can be modified, i.e., slowed down or accelerated, substantially by social and other external factors. In surveys people answer that health is perceived as the most valuable good and the most important for satisfaction (Arber and Ginn 1993). Health is also a social value and an economic resource (Hradil 1993:383). A healthy workforce and a healthy population is a precondition for economic and social well-being but, more and more, health is also perceived as the result of economic well-being, namely as a purchasable good.

Health has always been the outcome of a person's economic status, at least to some extent. But with the increasing possibility of improving one's health status through better nutrition, better environmental conditions and medical treatment (Marmot 1994), more health problems have become preventable and curable. This change largely depends on the overall wealth status of a society, individual socioeconomic status, and individual behavior. Therefore health is partly an outcome of socioeconomic status.

Given these long-term changes – first, in the conditions for obtaining a good health status through purchasable goods and services and, second, in our perception of the determinants of health – the sociological question arises:

Which socioeconomic predictors of health and mortality can be identified and how great are the resulting social differences in health and mortality? This question will be addressed as the first research topic in the empirical part of this dissertation.

In the background of this relation between the individual socioeconomic status and health there are changes in the role of health in society that will be briefly addressed in the following. The individual responsibility to care about the health outcome of our behavior increases to the extent that our ability to influence our natural environment and its interaction with the body increases. There are not only biological and medical reasons for a certain health status, but also social and behavioral factors which in principle have an alternative and are becoming more and more contingent. This means

that regarding public and individual health care, choices have to be made between different ways of practicing health care and different amounts of resources dedicated to this aim. This choice is restricted by limitations of public and individual resources and by competing goals which exist on both levels.

There is the tendency to perceive and to treat health as a purchasable good. To some extent it only stays a perception, i.e., we behave *as if* health was purchasable, but to some extent this trend materializes because healthy behavior and healthy living conditions really depend on economic categories. The result is that the increasing health expenditures and the increasing number of old persons have led to a public debate in many EU countries and in the USA about the question of whether societies can and should afford high quality health care for the elderly (e.g. Buiatti 2004). These debates are fueled by research results that specify detailed risk factors and the amount of individual responsibility for certain diseases. For example, it has been revealed that on average smokers are 40 percent more expensive for the health care system than non-smokers and that each kilogram above normal weight increases the health costs by 5 percent (Jungbauer-Gans and Schneider 2000).

Many of these considerations focus on old age because the assumption that an increasing number of elderly persons will cause both the average health level of the population to decrease and health care costs to increase is in principle correct. But as stated above, not only life expectancy but also healthy life expectancy increases, i.e., the number of years that people live in good health without expensive treatments increases, too. Studies show that the most expensive years are the last ten years before death. Within this period, the last year before death is the most expensive year (Brockmann 2002). As life expectancy increases, these years are shifted toward higher ages but the expensive period of bad health is not necessarily expanded (Zweifel et al. 1996).

Another related finding is that the overall health costs increase with the increase in GDP rather than with the share of old people (ibid.) This means that the high quality of health care, characterized by high-tech diagnoses, treatments and the use of medicine, is a driving force for the increase in health expenditures. Another reason for rising health costs, and probably the most important, is the inefficiency of the health care system. Monopolies in the medical sector and bureaucracy have successfully prevented structural reforms towards a more patient-oriented health care system (Kranich and Vitt 2003). Despite this lack of reforms, the perception of a cost-explosion in the health care system has been used to legitimize liberalization, which in turn has decreased the level

of health care for disadvantaged groups (Jungbauer-Gans and Schneider 2000:213). The main outcome of these current attempts to limit health expenditures is a deterioration in health care for poor people.

An important change may take place going from the right to be ill, which was formerly defined as an achievement of our welfare system, to the “individualized responsibility and care for one’s own health” (Jungbauer-Gans and Schneider 2000:229), based on the assumption that each individual can care for his or her own health. Such a development would neglect the realities that health is partly determined by unforeseeable or unchangeable events and genetic constitution. What is even more important is that poor and less educated people usually do not have the resources to act in a responsible manner and to pay for good health care and prevention.

For more affluent persons who can and do care a lot about their health, prevention and treatments, a different unintended consequence of the economic trend in the health care system may occur: due to a permanent reflection of one’s own health status, possible health threats, perfect diagnoses and treatments, the natural and carefree feeling about health may be lost, similar to the happiness that is destroyed when people are forced to be happy (Jungbauer-Gans and Schneider 2000). But in order to reduce social differences in health, it is most important to provide conditions, especially for disadvantaged persons, that would enable them to care about their health.

2.1 Social security and welfare systems

Health care is to a large extent organized and regulated by social institutions. The more general notion of these institutions is social security. In modern welfare states the individual depends on the welfare state at all ages but in old age the level of services is especially high, so the elderly and the process of aging depends on the welfare system (Esping-Andersen 1990). The main task of the social security system is to provide care and help for people who need it. These persons may be in a situation where they are still autonomous and active, but they just need support (empowerment). Other people, in very old age or with a very bad health status, also need to be guided and helped through everyday life because they are in need and no longer autonomous (Jungbauer-Gans and Schneider 2000). The need-model and the empowerment-model are two important directions in the understanding of the welfare system (Rosenbrock 1995).

As in the discussion of the financing of health care in the previous section, a trade-off exists between the advantages of market-shaped economized services, where those who need care are considered as sovereign consumers, and the need-model of social security. The latter approach pays attention to the fact that persons in misery may not be able to take part in a market because they do not have any power or orientation. One conclusion is that the distribution of social security cannot be organized totally by the market (Esping-Andersen 1990) and that the care of ill or weak people is a social interest per se and thus one of the duties of the welfare state (Jungbauer-Gans and Schneider 2000).

If the welfare state gives resources to people in need free of charge, this influences social inequality. In principle public health care systems that are financed by people with very different levels of income and wealth give a large share of their benefits to lower income groups, resulting in a redistribution of resources from the top-down. But social benefits do not necessarily reduce social inequality because the welfare system is another system of stratification and redistribution which has new lines of conflict (Esping-Andersen 1990). The welfare state can produce equality and/or social inequality (Beck 2005:7). For example, the German welfare state is especially conservative because it stresses and rewards traditional patterns in family and working life. This results in a disadvantage for persons who do not follow the normal life course concerning partnership and work. In such a conservative system, these persons experience an additional disadvantage throughout the welfare state (Vincent 1995:138; Ostner 1998).

Another reason why welfare state institutions do not necessarily reduce social inequality is, for example, that health insurance which is based on the principle of solidarity, can get into the following vicious circle: rich people who have to pay a lot into a health care system based on solidarity in order to support those who pay less, opt to leave the insurance system, which in turn then has less money. This decreases the quality of health care that insurance can offer to its clients, again inducing more rich people to leave. The result is that poor persons are left in an insurance system where they get worse health care and the principle of solidarity disappears (Ostner 1998:240). Andersen and Larsen (2002:3) call this “path dependency towards dualism” which may confirm the statement that welfare for the poor eventually becomes poor welfare (ibid; Korpi and Palme 1998).

The level and kind of social security is very different in different countries and in different welfare state regimes. The comparison of welfare states is its own specific

field of research and will thus not be discussed in detail here (see Esping-Andersen 1990). Table 2.1 shows important features of Esping-Andersen's main three welfare state regimes: liberal (e.g., USA, Great Britain), conservative-corporatist (e.g., Germany) and social democratic (e.g., Scandinavian countries). The two countries chosen for the empirical part of this dissertation, Denmark and the USA, fit well into this classification system in that they represent two different welfare state regimes. The following descriptions and classifications of welfare states should help to locate Denmark and the USA in the spectrum of differences. However, this dissertation will not make hypotheses about, or test empirically, the link between features on the welfare state level and social mortality differences. My analysis will only be done between individual characteristics and mortality. Denmark and the USA will be treated as two different cases for such an analysis.

Table 2.1: Comparison of three welfare state regimes

	Liberal (e.g. USA)	Conservative-corporatist (e.g. Germany)	Social-democratic (e.g. Denmark)
Employment Pathways	Educationally anchored work pathways but loose coupling between education and market. High levels of female labor force participation. Occupational segregation and high wage inequality. Part-time work is marginalized.	Education to work linkages tightly coupled via a "dual system" of partly school-based and partly firm-based vocational training. Low levels of female labor participation with occupational segregation. Part-time work highly gendered and marginalized.	Education to work linkages tightly coupled via vocational training in public schools. High levels of female labor force participation. Occupational segregation coupled with solidarity wage policy. Part-time work is not marginalized.
Family/Gender Pathways	Mixed breadwinner and role-sharing models	Breadwinner model predominates	Role-sharing model predominates
Welfare Regimes	Liberal model based on the principle of equivalence between covered employment and benefit eligibility. Life course risks receive limited protection with some based on means-tested eligibility.	Conservative-corporatist model based on the principle of social insurance. Life course risks are protected by the state.	Social democratic model based on the principle of citizenship. Life course risks are protected by the state.
Life Course Variability	High/ increasing levels of variability across tripartite phases	Low/ increasing levels of variability across tripartite phases	Medium/ increasing levels of variability across tripartite phases
Aged Inequality	High overall, Poverty high	Medium overall, Poverty low	Low overall, Poverty low

Source: O'Rand et al. 1999:188

The next table, Table 2.2, is an international comparison of some typical welfare state criteria. Countries can be similar and different in different dimensions and the USA and Denmark are not perfectly opposed types of welfare states. However, they do differ a lot

(more than one standard deviation) in most of the criteria, especially in the use of means-tested poor relief, in the share of private health spending and in benefit equality.

Table 2.2: Degree of corporatism, etatism, means-testing market influence, universalism, and benefit equality in 18 welfare states, 1980

	Corporatism: number of major occupationally distinct public pension schemes	Etatism: expenditure on pensions to government employees as percent of GDP	Means-tested poor relief (as percent of total public social expenditure)	Private pensions (as percent of total pensions)	Private health spending (as percent of total)	Average universalism (average for sickness, unemployment and pensions)	Average benefit equality (average differential between basic and maximum social benefits for sickness, unemployment and pensions)*
Australia	1	0.7	3.3	30	36	33	1.00
Austria	7	3.8	2.8	3	36	72	0.52
Belgium	5	3.0	4.5	8	13	67	0.79
Canada	2	0.2	15.6	38	26	93	0.48
Denmark	2	1.1	1.0	17	15	87	0.99
Finland	4	2.5	1.9	3	21	88	0.72
France	10	3.1	11.2	8	28	70	0.55
Germany	6	2.2	4.9	11	20	72	0.56
Ireland	1	2.2	5.9	10	6	60	0.77
Italy	12	2.2	9.3	2	12	59	0.52
Japan	7	0.9	7.0	23	28	63	0.32
Netherlands	3	1.8	6.9	13	22	87	0.57
New Zealand	1	0.8	2.3	4	18	33	1.00
Norway	4	0.9	2.1	8	1	95	0.69
Sweden	2	1.0	1.1	6	7	90	0.82
Switzerland	2	1.0	8.8	20	35	96	0.48
UK	2	2.0	n.a.	12	10	76	0.64
USA	2	1.5	18.2	21	57	54	0.22
Mean	4.1	1.7	5.9	13	22	72	0.65
Std. Dev.	3.2	1.0	5.1	10	14	19	0.22

Source: Esping-Andersen 1990:70

* Benefit differentials are based on the ratio of guaranteed basic social benefit to the legal maximum benefit possible in the system.

Table 2.3 allows us to briefly summarize similarities and dissimilarities between the USA and Denmark. It shows that, in terms of Esping-Andersen's three welfare state dimensions, both countries have a low degree of conservatism; they differ only moderately concerning liberalism but show a maximum difference in socialist democratic attributes.

Table 2.3: The clustering of welfare states according to conservative, liberal and socialist regimes attributed with cumulated index scores

	Conservatism		Liberalism		Socialism	
strong	Austria	8	Australia	10	Denmark	8
	Belgium	8	Canada	12	Finland	6
	France	8	Japan	10	Netherlands	6
	Germany	8	Switzerland	12	Norway	8
	Italy	8	United States	12	Sweden	8
medium	Finland	6	Denmark	6	Australia	4
	Ireland	4	France	8	Belgium	4
	Japan	4	Germany	6	Canada	4
	Netherlands	4	Italy	6	Germany	4
	Norway	4	Netherlands	8	New Zealand	4
			United Kingdom	6	Switzerland	4
					United Kingdom	4
low	Australia	0	Austria	4	Austria	2
	Canada	2	Belgium	4	France	2
	Denmark	2	Finland	4	Ireland	2
	New Zealand	2	Ireland	2	Italy	0
	Sweden	0	New Zealand	2	Japan	2
	Switzerland	0	Norway	0	United States	0
	United Kingdom	0	Sweden	0		
	United States	0				

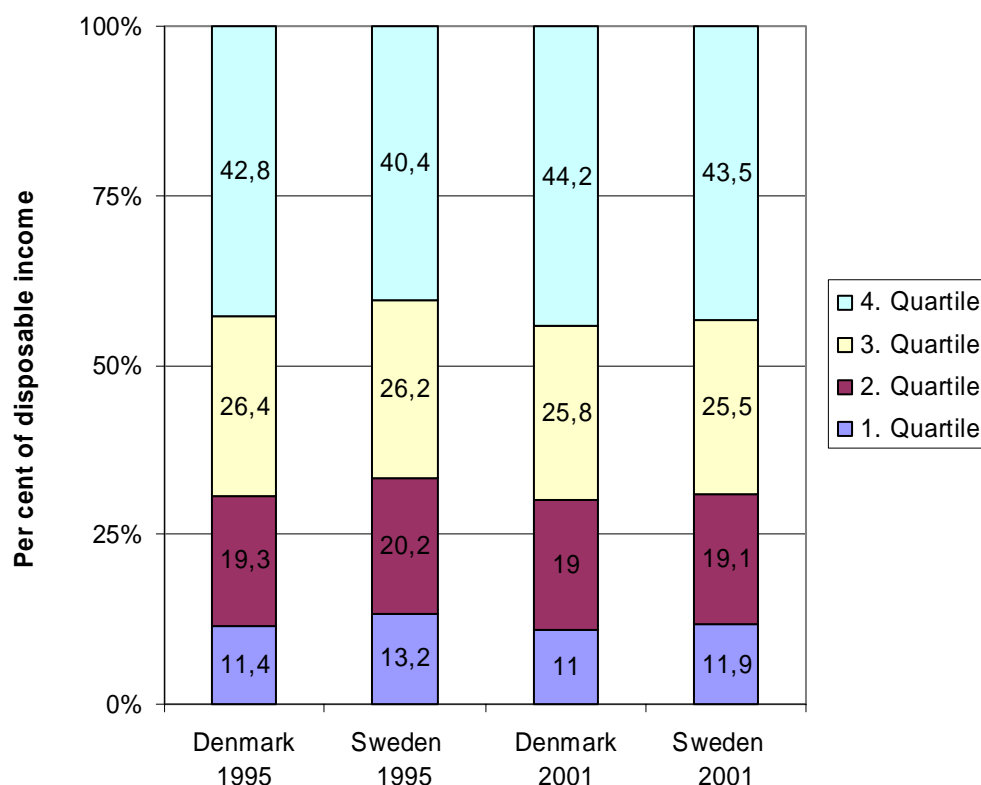
Source: Esping-Andersen 1990:74

This well-known classification by Esping-Andersen is based on information from the late 1980s. The extent to which the relative position of each welfare system has changed cannot be analyzed in detail here. In the 1980s and 1990s, Denmark moved within the class of regimes that Esping-Andersen calls Social Democratic, slightly towards the liberal pole, compared with Sweden, for example, which instead moved in the conservative direction (Andersen and Larsen 2002:2). Fewer cut-backs have been implemented in Denmark compared to Sweden (Kvist 1999:231). The USA expended a lot of effort in order to provide better access to health care for the poor (Steinkamp 1999:140) and improved policy for the elderly, which actually increased the overall availability of health care services. However, large social differences in the quality of health care remain.

O’Rand et al.’s comparison (1999:206) comes to the conclusion that the USA, Sweden, and Germany are the three most typical representatives of the three welfare state regimes. In spite of the differences between Sweden and Denmark, this comparison can also shed light on differences between the USA and Denmark to some extent:

“Perhaps a final ironic comparison can be proposed between the United States and Sweden. Both contexts imply relatively high levels of individualization, but of different kinds. Individualization in the United States is privately defined. It is expressed by the variability in the life course across all domains – education, family, and work – that are loosely coupled over the lifetime of aging U.S. cohorts. In the absence of a strong welfare system, loose coupling among these institutions tends to segment the experiences of individuals in the system. Individualization in Sweden is publicly defined as citizenship. Variability in the life course extends mainly to family and gender roles, roles that receive coherent and integrated support within the system. Solidarity as opposed to segmentation or isolation appears to be the contrasting result. In between the two systems, Germany exhibits more clearly defined gender-based pathways that emerge from tightly coupled market, state, and family systems. Workers are relatively more advantaged, but women are less at risk of poverty as a result of protection from the social insurance system” (O’Rand et al. 1999:206).

Since Sweden is taken as a representative of the Scandinavian countries many times in the literature, I will also use some of these comparisons to illustrate the principal differences between USA and Nordic countries. Of course, this comparison may not be adequate in all respects for comparing Denmark and the USA. But in cases where comparable data for direct comparisons between Denmark and the USA could not be found within the frame of this dissertation, it is worth taking information from Sweden. To evaluate the similarity between Denmark and Sweden the following figure and table compare some welfare state features of these two countries.

Figure 2.1: The share of disposable income received by each income quartile in Denmark and Sweden, 1995 and 2001

Source: NOSOSCO 2005

Table 2.4: Social and health expenditures in Denmark and Sweden in 1995 and 2002

		1995	2002
Social expenditure (percentage of GDP)	Denmark	32.2	30
	Sweden	35.8	32.5
Total health care expenditure (percentage of GDP)	Denmark	8.2	8.8
	Sweden	8.1	9
Total health care expenditure (€ per capita)	Denmark	2187	3001
	Sweden	1691	2644

Source: NOSOSCO 2005

Though they were already quite similar, Figure 2.1 shows that Denmark and Sweden became even more alike between 1995 and 2001 concerning the income distribution. In 1995, Sweden was slightly more equal than Denmark. Table 2.4 shows that the percentages of social and health expenditures from the GDP are also similar in Denmark and Sweden both in level and trend. A difference is obvious in terms of health

expenditures in absolute values per capita. In 1995 Denmark spend 29 percent more on health care per capita than Sweden; in 2002 it was still 14 percent more.

This section has shown that welfare systems and levels of social security differ considerably even among rich, developed welfare states. As far as the available information allow it, I tried to contrast welfare state features that are important for health and well-being of the elderly in Denmark, the USA, Germany and other countries. The next section will be a more detailed description and comparison of welfare rules in Denmark and the USA.

2.2 Welfare in the USA and Denmark

To compare the USA and Denmark it is worth to look at the beginning of the 20th century because at that time most of the persons in my data sets for Denmark and the USA are born. Denmark had a lower living-standard, lower child health level and a lower educational level than the USA. But in both countries this was a period of enormous improvements in the sanitary and health care systems. Table 2.5 shows some indicators for the Danish health care system and their change over time.

Table 2.5: Statistics of the Danish health care system, 1890-1939

Year	Doctors	Nurses	Hospitals	TB-Sanatorias
	Number of persons		Number of beds	
1890	941	n.a.	5600	0
1901	1350	394	7915	465
1910	1631	1182	10652	2171
1920	1918	3371	13349	2890
1930	2485	7277	16710	3446
1939	3252	12434	22781	40038

Source: Johansen 2002:175

In Copenhagen, a closed pipe system for water was used, carrying water from the wells to all consumers from 1900 on. Compared to many large European cities, Copenhagen had a high standard with respect to water supply and the sewage system. The use of water closets increased for a few decades until 1939 when about 99 percent of the households in central Copenhagen were equipped with water closets (Johansen 2002:176). In the USA, filtration, chlorination, and partly also sewage treatment and sewage chlorination began to spread throughout the cities, all of which have been

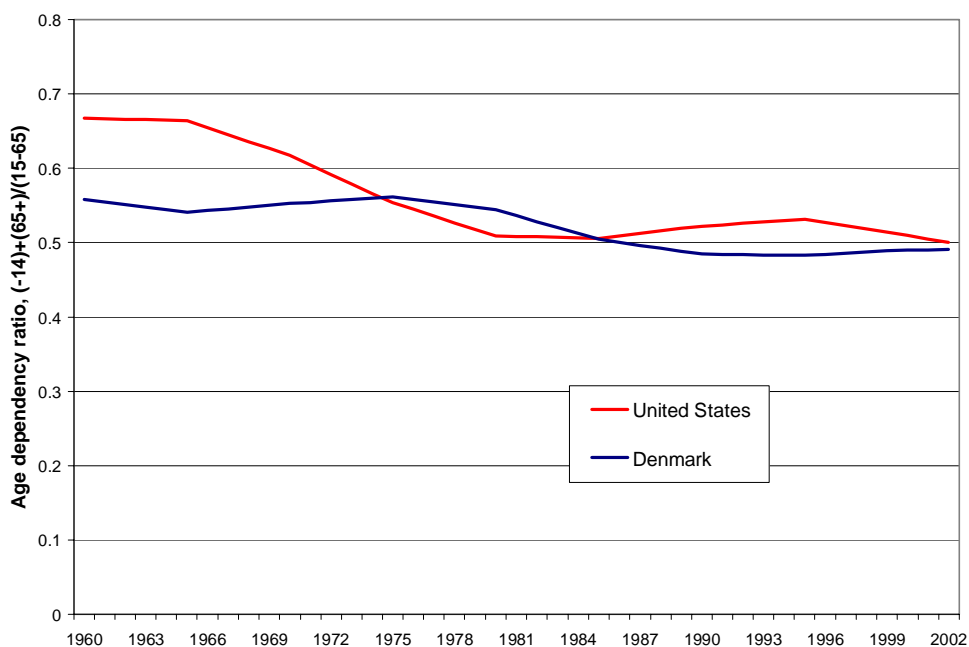
shown to be responsible for a large part of the mortality decline between 1900 and 1936 (Cutler and Miller 2005).

During the 20th century, Denmark and the USA both experienced increasing prosperity. The Danish welfare system compared to the USA certainly exerts a reductive influence on the development of social inequality with the effect that inequality in Denmark increased, but less than in other countries such as the USA (Munk 2000:4,14).

The USA and Denmark have populations of very different sizes. During our observation period in Denmark from 1980 to 2002, the Danish population increased by 5 percent from 5.1 million to 5.4 million persons. In the same period, the U.S. population increased by 27 percent from 227 to 288 Million persons (World Bank 2004). The higher rate of population growth in the USA is due in part to a higher fertility rate, but mostly it is due to more in-migration.

The age structure of the population can be described by the age dependency ratio. This is the ratio of dependents, i.e. people younger than 15 or older than 64, to the working-age population from age 15 to 64. The age dependency ratio for the two countries can be seen in Figure 2.2.

Figure 2.2: Age dependency ratio in Denmark and USA, 1960 to 2002



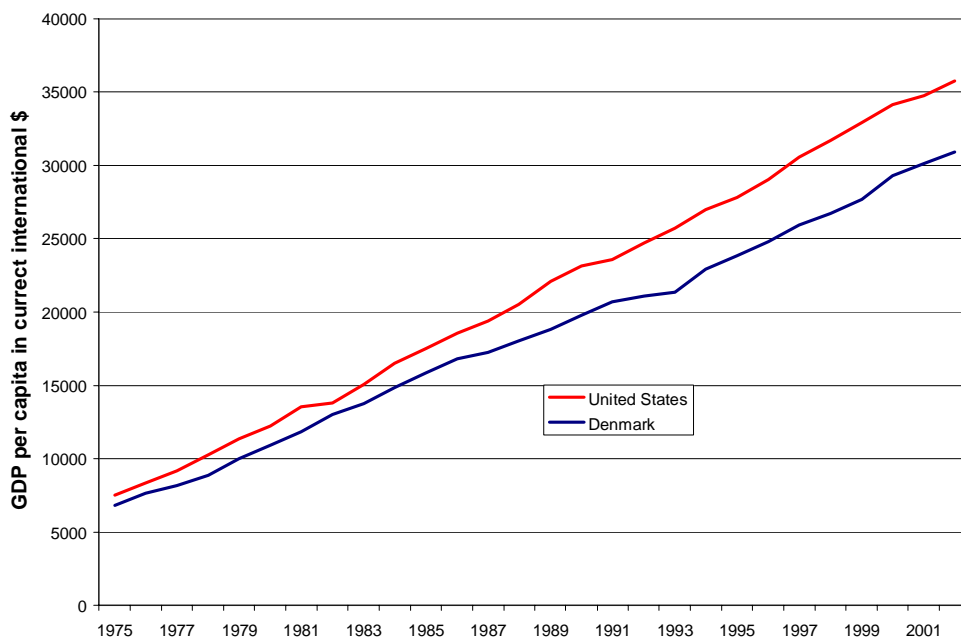
Source: World Bank 2004

This figure can be interpreted as follows: an age dependency ratio of about 0.5 like in both countries for the last twenty years means that per 100 persons between ages 15-64

(assumed to be the productive age), there are 50 persons that are “dependent”, i.e., under age 15 or over age 64. In contrast to the *old age dependency ratio* that shows the “burden” of old age dependency that increases (worsens) for most countries, Figure 2.2 just shows the *age dependency ratio* that decreases (improves) and then levels off. This means that the increasing share of old persons is more than out-balanced by the decreasing share of children that are also “dependent” persons.

Figure 2.3 shows a central category for the overall welfare of a country, GDP per capita. In 1975 Denmark and the USA are close together in terms of GDP per capita. Both countries experience a very linear increase, but the slope is steeper for the USA which makes its GDP per capita almost \$5,000 higher than for Denmark in 2002. Both countries have GDPs that are among the highest in the world.

Figure 2.3: GDP per capita, USA and Denmark, from 1975 to 2002



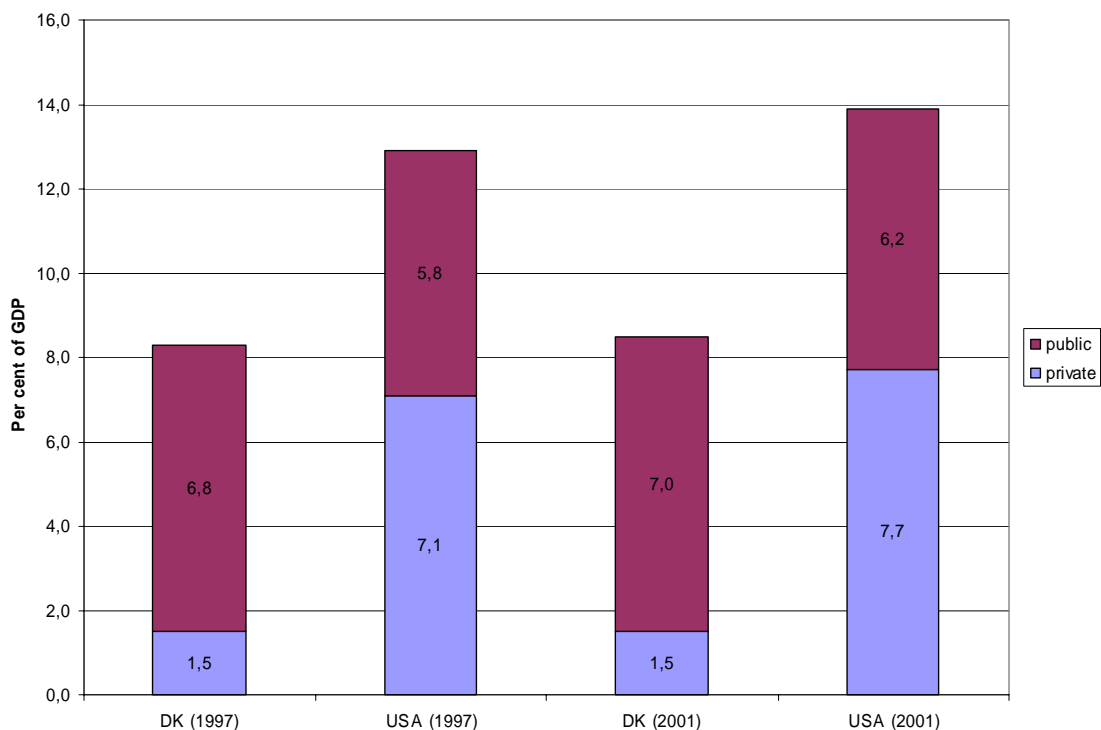
Source: World Bank 2004

In the following I will give a description of the welfare system for elderly people in the USA and in Denmark that focuses on the health and age relevant aspects. One big difference is that in the USA about 15 percent of the population does not have health insurance. This number changed only very little between 1990 and 2004 (Bureau of the Census). Besides that, in the USA generally people have to pay higher co-payments. However, the insurance coverage for people over age 65 is about 99 percent and this

holds almost stable from 1990 to 2004. This is about the percentage of coverage in Denmark and Germany for all ages. Thus, among the elderly in the USA there are as few uninsured people as in rich European welfare states. The difference is that being insured in the Medicare program (see below) in the USA (as are 96 percent of the population above age 65) and even more so in the Medicaid program (as 9 to 10 percent of the population above age 65 are) does not guarantee the same high level of health care as in Germany or Denmark.

The percentage of the GDP that the USA spends on health care increased from 12.4 to 13.9 percent from 1997 to 2001 (Bureau of the Census, World Bank 2004). This share includes both public and private health expenditures. In Denmark public and private expenditures together held stable from 1997 to 2001 at about 8.4 percent of the GDP (Manton et al. 1995; World Bank 2004). Important differences are that the USA spends more of its GDP on health and that the share of private spending is higher than in Denmark, namely higher than the public portion.

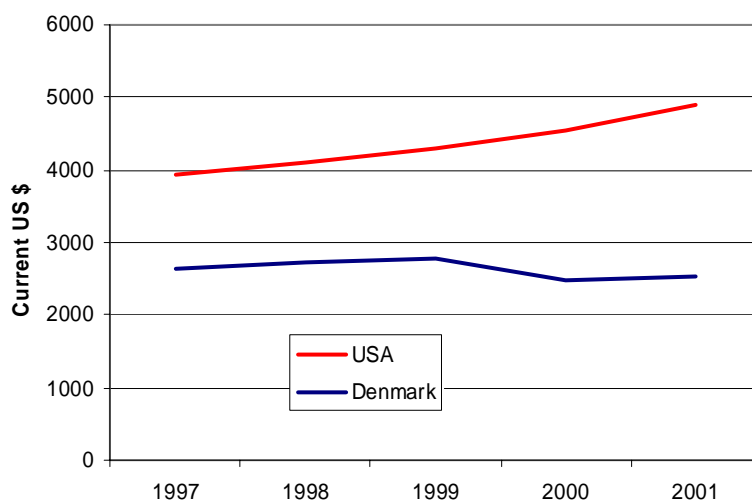
Figure 2.4: Public and private health expenditures as share of GDP in USA and Denmark



Source: World Bank 2004

Figure 2.5 below shows these numbers in absolute amounts. In Denmark, the health care expenditure was about \$2,500 per capita per year and \$4,887 in the USA in 2001. In the USA this number increased by about 25 percent in just 4 years while it remained stable with minor fluctuations in Denmark. Although more money is spent on health care in the USA and in general U.S. health policies and social policies are orientated towards the elderly, the quality of the services is not better than in other rich countries including Denmark. The USA spends more money on health care per capita than any other country and has the most advanced medical system (Cutler 2003:2; Cutler 2004). Thus the health care system in the USA can be regarded as relatively inefficient in its resource allocation. National health expenditures are rising but the results are not improving and not consistently better than in countries that spend less (Williams 2001:81; Kaplan 2001:145).

Figure 2.5: Health expenditures per capita in U.S. \$ for the USA and Denmark from 1997 to 2001



Source: World Bank 2004

In the USA, like in several other countries, many improvements in the health and mortality of elderly people have been made since the inception of Social Security in the 1930s (Kaplan 2001:139). Since then, different laws against aged poverty have been made (Sattler 1994; O’Rand 1996). An institution that sharply improved the health care coverage for the elderly and that makes a difference in health care between older and younger persons in the USA is Medicare. It is the largest public health care program and is devoted to all persons aged 65 and older as well as to permanently handicapped

persons. It costs more than 2 percent of the GDP (Lee and Edwards 2001). Medicare covers hospital and related services (Klein and Unger 2001) and tends to address acute illnesses rather than prevention (Adams and McFadden 2002).

In principle, Medicare pays for the necessary health care but 70 percent of those who are entitled to Medicare have additional private health insurance, called Medigap (Sattler 1994:182). People who receive Medicare have to pay on average \$3,000 in co-payments per year (Kneesebeck et al. 2003). Generally, persons over age 65 spend 23 percent of their income for out-of-pocket health care costs, which is more than before the start of the Medicare program in 1965 (Crystal 1996:404, 392ff). Altogether, 43 percent of all health care costs of the elderly are paid out-of-pocket (Crystal 1996:404). Medicare does not prevent poorer persons from getting the worst health care. Doctors are paid 25 to 45 percent less for the treatment of a Medicare patient than for other patients with the result that on average Medicare and Medicaid patients get worse doctors and worse treatments (Moon 1995; Kneesebeck et al. 2003; Silveira et al. 2005). Medicaid is another health benefit program which is a health insurance program for low-income people, like certain low income families with children, aged, blind or disabled people on supplemental security income and people who have very high medical bills. It was implemented in 1966 (Moon 1995). Of those who are eligible for Medicare, 15 percent are also in Medicaid, which means that they do not have to pay the co-payments for Medicare services. The services offered by Medicaid differ considerably between states within the USA. Only 42 percent of the people living under the poverty line receive Medicaid payments or services (Sattler 1994:183). Additional to health benefits, people may be eligible for SSI (Supplemental Security Income) which was about \$484 per month in 1997 for a single person over age 65 with no other income or assets (O'Rand et al. 1999:46). Research results show that Medicare did not have a large impact on one's overall health status or on health or mortality differences between social groups (Auerbach and Krimgold 2001:151).

Conceptionally, the Danish welfare system belongs to the so-called Nordic welfare state model of which the ideal form can be described by the following features (Kvist 1999:232):

1. **Comprehensiveness:** the scope of public policy is broad; the state has a larger role vis-à-vis the market and civil society than is the case in other countries.

2. Full employment: policies are committed to contributing to full (i.e., more) employment and/or preventing unemployment, particularly long-term unemployment.
3. Equality: policies are committed to contributing to equality between groups based on gender, age, class, family situation, ethnicity, religion, region and so forth.
4. Universality: right to basic social security benefits (in cash and kind) in a wide range of social contingencies and life situations.
5. High-quality benefits: services are of a high quality, and provided by welfare professionals.
6. Generous benefits: cash transfers are generous, in particular for low-income groups, to allow for a “normally” accepted standard of living.

The Danish health care system can be described as more generous than the system in the USA, but this observation is far from complete: “there are 6- to 12-month waits for cataract and hip surgeries. The wait for cardiac procedures exceeds three months. The effects of such delays are not benign for persons who are 80 years old or older” (Manton et al. 1995:1233).⁵

2.3 Pensions

Besides special benefits and payments for health problems and health prevention, income in old age is important for a person’s socioeconomic status and overall level of health care. This is especially the case when private co-payments are high, like in the USA, but many other material aspects other than health care benefits contribute to a good health status, with the consequence that in all welfare systems, the income of the elderly is very important. This income consists mostly of a pension from the state, an employer, or a private pension plan. Historically, receiving a pension in old age as a mass phenomenon is not very old. There are estimates that only 20 percent of workers retired before death in the early 20th century (O’Rand et al. 1999:99ff). Nowadays, the average age at death is 10 to 20 years above the average pension age, thus many people receive pensions for many years.

⁵ For a detailed discussion of features of the Danish welfare state including its transition, a possible crisis and international comparisons, see Cave and Himmelstrup 1995; Andersen 1997; Hansen 2002; Hussain 2002.

For an empirical study of socioeconomic status in old age it is important to consider possible income sources in old age because they may be more diverse and more difficult to take into account than the income of working people. The following table shows the relative importance of different income sources by income level for all aged persons and for aged single women from the USA, Sweden and Germany.

Table 2.6: Within deciles aggregate income composition for the aged and for single aged women in the USA, Germany and Sweden

	All aged persons			Single Women 65+		
	decile 1	decile 5	decile 10	decile 1	decile 5	decile 10
United States (1994)						
Social retirement	69.7	65.7	18.6	68.6	34.3	26.8
Means-tested income	17.8	0.9	0.1	18.8	1.5	0.2
Occupational pensions	3.7	14.7	20.1	2.7	6.6	21.2
Earnings	2.6	9.6	37.9	0.6	1.9	17.2
Capital Property	6.1	9.2	23.2	9.3	5.8	34.6
Sweden (1992)						
Social retirement*	76.8	90.6	71.2	79.0	74.6	84.2
Means-tested income	15.8	0.6	0	13.7	13.1	0.1
Earnings	0.4	1.7	16.5	0	0.1	5.1
Capital Property	7.0	7.1	12.4	7.3	12.1	10.6
Germany (1989)						
Social retirement	73.9	83.5	26.4	83.1	86.6	40.7
Means-tested income	8.9	0.1	0.3	5.3	1.3	0
Occupational pensions	3.7	7.5	31.3	5.1	3.3	37.3
Earnings	6.5	6.5	33.6	1.3	0	5.3
Capital Property	7.1	2.4	8.4	5.3	3.8	16.7

Source: O'Rand et al. 1999:201. Percentages are rounded from figures reported in the Luxembourg Income Study Database (Smeeding 1997).

* Social retirement in Sweden includes occupational pensions, which average about 8 percent of income.

In all countries and both groups social retirement income makes up the largest share in the income composition. This share decreases considerably from about 70 percent to about 20 percent when we go from the poorest income decile to the richest. But this is only true for the USA. This high level of state responsibility for pensions makes them account for more than 10 percent of the GDP in many countries. But of course this percentage differs depending on the pension system (Esping-Andersen 1990:79,103). Means-tested income plays a role for poor persons and single women in Sweden with an average income. The share of occupational income increases with the income group in the USA and Germany. In Sweden, this type of income is included in social retirement. Therefore the share of social retirement income for rich people is as high as for the poor. The share of earnings increases with the income level, which shows that rich persons have better possibilities of earning money after age 65. Lastly, the importance of capital property is highest for the richest income decile in the USA.

Denmark, and the other Scandinavian countries as well, started with a universal “flat-rate” pension regime with a classical Social Democratic notion of citizenship (Andersen and Larsen 2002:2). In 1994 there was a major reform of the social assistance and pension scheme where the pensions became almost fully taxable. This reform brought an income-test against earnings on the basic amount of the national pension, but nothing changed concerning the right of all citizens to receive a guaranteed minimum pension. As mentioned in Section 2.2 this reform implied less generosity and a slight liberalization of the Danish system, which however can still be subsumed under Esping-Andersen’s category of Social Democratic welfare regimes. Universal old-age pensions together with relatively generous benefits are characteristics of the Nordic welfare-state model. It means that normally the elderly have access to social care regardless of an individual’s previous work and contribution record (Kvist 1999:246).

An important parameter of the pension system is the age where persons can retire and expect to get payments. In the last decades the pension period has been extended at both ends: people tend to retire earlier and they get pension up to higher ages because they live longer (O’Rand et al. 1999:34; Lee and Edwards 2001). This is true as a general trend since the 1950s, but the retirement age still differs considerably between countries. In the USA, early eligibility for retirement benefits for men at age 62 was introduced in 1961 (Gruber and Wise 1999:14). From 1970 to 1985 public incentives encouraged people to retire early, while after that period, people were encouraged to retire later (Quadagno and Hardy 1996:341). The legal retirement age in the USA has been 65 for many years. However, beginning with people born in 1938 or later, that age will gradually increase until it reaches 67 for people born after 1959. In all systems early retirement usually leads to lower pensions (O’Rand et al. 1999:45).

The eligible age for retirement is 65 in many countries, as in Germany and the USA. In the European Union it is lowest in Italy with age 59 and highest in Denmark with age 67. Besides this official age, labor force participation in old age in the USA has been increasing in some periods and has always been much higher when compared to European countries. In the USA, 50 percent of men between ages 60 and 64 still work. In Germany the respective figure is only one-third (Niejahr 2003). There are high participation rates in the USA, Sweden and Great Britain and low rates in Germany and France. The departure rate from work shows the same pattern: it is 25 percent in the USA and 60 percent in France and Germany. Denmark is in the middle, because unlike

Sweden, it encouraged early retirement for a long time until the pension reform of 1994 (O’Rand et al. 1999). The actual retirement age in Denmark and Germany is about 61 (Abrahamson and Wehner 2003:18). In the USA it is higher and many people continue to work part-time after retirement.⁶

It is unclear how much public pensions reduce social inequality in old age. Evidently, those persons with a high working income also get high pensions (Crystal 1996: 395). Moreover, they get these pensions for a longer period because they live longer (Menchik 1993). The trend towards private pension schemes increases inequality in old age and especially gender differences because those with small or no income will also be the ones who can not invest in their future pension.

Persons with higher education retire later, but this does not mean that they are forced to work in older ages. It rather means that they have occupations that on average require less physical performance and are suitable for the elderly. Persons with a higher socioeconomic status also work longer because they also have a better health status. Relative to their health status they retire earlier than persons with lower status, i.e., when they retire they have better health (O’Rand et al. 1999:129).

The comparison of some broad categories of the welfare system in Denmark and the USA shows that the two countries that will be studied in the empirical part are very different in terms of welfare rules. It will be interesting to see whether these substantial differences in the level of welfare translate into social differences in health and mortality. In Section 1.1 we already saw that the overall level of mortality speaks in favor of the USA, which is the richer of the two countries, but the USA has a lower level of explicit social welfare. The empirical analysis in this study will provide information about the size of social mortality differences in each country. However, a comparison of these differences in a strict sense, and the analysis of factors on the level of the welfare state, is not possible within the scope of this dissertation.

⁶ For a description of the Danish pension system and an international comparison see Hauschild 1999; Andersen and Larsen 2002; Abrahamson and Wehner 2003.

Summary of Chapter 2

The relation between health and mortality on the one hand and society, i.e. social mechanisms, on the other is the main topic of this dissertation. Due to the increasing possibilities of influencing health by medical treatments and individual behavior, health has become an important subject in the discussion of social distribution processes, welfare and individual responsibility. Due to the economization of health and health care, social differences in the quality of health care are increasing. On an international level, different welfare systems provide different levels of social security including different levels of health care. The elderly especially depend on public services provided by the welfare system. Denmark and the USA belong to different categories of welfare systems and in many regards they represent opposite poles concerning the level of social security. Different dimensions of the welfare system of the two countries are compared and considered. These include the pension system, the Medicare program in the USA, and the income composition of the elderly. Although in principle welfare institutions tend to redistribute resources from the rich to the poor in order to reduce social inequality, in many cases this effect is marginal.

Chapter 3 Concepts of social inequality

For the analysis of socioeconomic differences in health and mortality, a basic definition and understanding of social inequality is needed. Societies consist of individuals, which are all different. The notion of social inequality only refers to differences in such parameters which have an influence on the social position of an individual. These characteristics are resources or goods in a broad sense that are much in demand in the society. The unequal distribution of these goods must not be natural or accidental, e.g., body size, but must be systematically made by a social process. If this systematically unequal distribution occurs regularly between the same social groups, this inequality will be perceived as inequity and can become a social problem (Hradil 2001:29). Hradil summarizes his definition of social inequality as follows:

“Social inequality exists when people frequently receive more of a society’s ‘valuable goods’ than others owing to their position in the social network of relationships.” (Hradil 2001:30). This definition implies that differences in eye color, body height, physical handicap, etc. cannot be called social differences or social inequality because they are not the result of a social process. To be precise, even height is not purely biological or genetic since it also depends on class. But what is more important is that such characteristics have a social meaning and can imply serious social advantages or disadvantages for individuals (Goldman 2001b:23). Characteristics like height show that physical attributes pose a special problem with regards to a clear definition of social inequality. Of course society cannot be blamed for an individual’s body height or a handicap, but if we look at a more complex characteristic like beauty or health, there are many ways in which these “resources” are distributed by social mechanisms. This will be discussed later.

According to Hradil it is important to differentiate between legitimate and illegitimate inequality. Sociological research focuses not only on illegitimate inequalities which are generally considered unjust, e.g., income differences between men and women with the same qualification level, but also on generally accepted differences, e.g., income differences between persons with very different levels of qualification (Hradil 1987a:16).

The so-called dimensions of social inequality specify which goods contribute to social inequality because they are in great demand in society and unequally distributed. Hradil

differentiates between four basic dimensions (material wealth, power, prestige and education) and four “new” dimensions of social inequality: working conditions, housing, environmental and leisure conditions. Other, somewhat similar resources could be added to this list, e.g., social capital, security and mobility. The term “new” has often been criticized (e.g., Dietz 1997:72ff). Hradil concedes that some of these dimensions are not really new, but the interest in these dimensions has increased to the extent that the work sphere, which the traditional dimensions focus on, has lost its relative importance (Hradil 1987b:117).

The social situation or the social status of an individual is the result of the interplay of many different dimensions. The most important dimensions are mentioned above as dimensions of social inequality. The term “social status” is older and focuses more than the term “social situation” on a hierarchic social structure. Both terms designate objective living conditions rather than subjective perceptions and interpretations.

Another categorical level in Hradil’s theory entails the determinants⁷ of social inequality, such as gender, age, cohort, occupation, region of residence, ethnic group or nationality and living arrangements, i.e., marital status and number of children (Hradil 1987a:40).

“The determinants of social inequality denote social positions of individuals in networks of social relations [...]; these positions do not represent advantages or disadvantages as such but very likely produce them” (Hradil 2001:34).

Material wealth as one dimension of social inequality is central to the analysis of social inequality because in modern market societies it is material wealth and money especially that is necessary for a high standard of living. Besides material goods, money can also buy immaterial goods like security, health, housing conditions, etc., at least to a large extent. Hradil calls these “chances for conversion” and uses arguments similar to Bourdieu, who assumes a mutual convertibility of economic, cultural and social capital (Bourdieu 1983:190,197; Woll-Schumacher 1994:228). Huster describes income in a close relationship and as a precondition for the satisfaction of many different kinds of needs (Huster 1993:43).⁸

⁷ In German the first term in use was “Zuweisungsmerkmale” and then “Determinanten”.

⁸ The Hypothesis of the value change towards a so-called post-materialism (Inglehart 1977) cannot be taken as a counter argument to this central role of material wealth because post-materialism has developed together with increasing levels of material wealth as an additional orientation which should not interpreted as anti-materialism (Reusswig 1994:25; Schultz and Weller 1996:25f).

Before I present Bourdieu's system of social classification it is worth mentioning a general compromise that all such systems have to make and that is very clearly described by Steinkamp (1993:114f):

“An ever increasing differentiation in the classification of ever increasing aspects of unequal living conditions affecting ever shrinking population groups could in the end lead to the absurd consequence of the “total individualization of social inequality”, where a *common* concern no longer would be identified. Such an approach would – as Geiger [1980] already clearly saw – ‘establish no order to the diversity of phenomena at all, but rather mirror the disorder of reality as accurately as does a photographic image’”

This means in order to reveal structures and dynamics in social relations it is necessary to classify phenomenon and characteristics.

A systematic classification of resources that play a role in social inequality has been suggested by Bourdieu (1979). He defined economic, cultural, and social capital and for a more detailed approach also a fourth kind of capital, symbolic capital (Bourdieu 1979, 1983).⁹

Since Bourdieu's theory of capital, structure, habitus and practice is one of the most comprehensive and widely used concepts to describe and explain social structure, social inequality and classes, it will be introduced in more detail below. Bourdieu offers an evolution of two earlier theories, first by Marx, who defined classes by their position in the economic system and for whom the labor class was opposed with hostility to the capitalists in a historically determined process (Marx 1969). Secondly, Weber's class theory already used more cultural phenomenon such as lifestyle and he defined classes by their life chances and opportunities. Bourdieu reintegrates two aspects of social inequality, which have been separated by Weber and designated with the German terms *Klasse* and *Stand*. The latter could be described by wealth and prestige combined with a certain way of life. It is true though, that Weber (1985:535) has already established this connection between *Klasse* and *Stand*:

“The differences between the social classes enter the most manifold relationships with corporative differences, and property as such in the long run gains corporative [ständische] importance, this with exceeding regularity.”

⁹ Especially in the American literature Coleman is sometimes considered to be the first author who described social capital (e.g. Coleman 1988). However, Bourdieu introduced this term first in 1979. Another author who established the notion of social capital is Putman.

3.1 Pierre Bourdieu's model of structure, habitus and practice

The special feature in Bourdieu's theory is that he introduces a dimension of structure (the space of social positions) and a dimension of practice (the space of lifestyles), which is mediated by "habitus", a kind of class-specific way of producing forms of practice (Bourdieu 1982:277). Accrediting to individuals a strategic and spontaneous room for maneuver, still limited by structural constraints, Bourdieu points to a middle way between structural determinism and the subjective voluntarism of acting (1987:105). The latter perceives the individual to be able to take conscious, free, and rational decisions. At the same time, a middle way is taken that aims to dissolve the traditional cleavage in sociological theory between objectivism and subjectivism (Schwingel 1995:68,92).

3.1.1 Three kinds of capital

A central term in Bourdieu's social model is capital (see Bourdieu 1983). The term can be used to highlight existing differences between societal groups (in particular, when capital is perceived in terms of accumulation), including durable and determining differences in power and the influence of particular groups. These differences reach beyond the effects of currently available resources to the detriment of equal opportunities. In principle, capital can be regarded as accumulated work. It has a tendency to survive, i.e., it can be seen as a kind of stock-building for work carried out over a relatively long period (even covering generations), which again secures resources for a certain period. Capital can reproduce itself and make profit, i.e., grow. Bourdieu does not limit the term capital to a purely economic definition but broadens its application to include the social exchange of social goods, e.g., prestige. The unequal distribution of capital forms the basis of its effect, namely making profits and instituting rules conducive to capital reproduction, and this applies to all areas affected by capital.

1. Economic capital

It corresponds to the common usage of the term capital and denotes money or possession, i.e., goods that can be converted directly into money. This sort of capital is institutionalized through vested titles and dominates other forms of capital because it can be easily converted into economic capital, among other things. Economic capital

constitutes a unit that penetrates all other forms of capital. Bourdieu states that it is not only economic capital that can be used selfishly and that has power. His observation applies equally to the two kinds of capital mentioned below.

2. Cultural capital

a) Incorporated cultural capital. This capital is in principle body-bound and requires a process of internalization, i.e., education (Bourdieu 1983:186). Education requires that time and socially constituted “libido” (deprivation, making sacrifices) be invested. If a family has a high level of cultural capital, the accumulation of cultural capital has already begun in early childhood through socialization. According to Bourdieu, this form of capital transfer is the least transparent (ibid.:188). Families with different levels of cultural capital need to invest different amounts of time and economic capital in order to further accumulate cultural capital. From the necessity of investing personal time in the individual acquisition of education, it follows that, “of all measures for cultural capital the least inaccurate are those that use *education duration* as a yardstick” (ibid.:186).

b) Objectified cultural capital. This kind of capital includes objects such as works of art or machines which are material carriers of culture capital. Incorporated cultural capital is again needed to make use of these objects or benefit from them.

c) Institutionalized cultural capital. It consists of, for example, titles or academic degrees that officially confirm a person’s cultural capital, thus establishing a clear demarcation line between the carriers of institutionalized cultural capital and self-taught people. It certifies cultural competence and legally guarantees a conventional value, acknowledgment, and a certain level of power. Investment in education requires economic capital and can be reconverted into economic capital e.g., by way of titles. It is necessary, however, that the title is relatively rare. The central meaning of educational degrees and titles as forms of cultural capital is based on the fact that degrees “sanction in a durable manner the individual position in the distributional structure of cultural capital” (Bourdieu 1987:228). Generally, degrees are important in transforming one form of capital into another.

3. Social capital

“Social capital constitutes the totality of current and potential resources connected to a durable network of more or less institutionalized *relations* characterized by mutual knowing or acknowledgement; in other words, social capital is a resource based on the *affiliation to a group*” (Bourdieu 1983:190).

The total capital of a group serves as security and lends credit-worthiness to the individual member. The exchange of relations as regards social capital requires mutual recognition. Profits arising from affiliation to a group form the basis of solidarity, which in turn facilitates the making of these profits, causing the effect of multiplication and profit increase. Networking and investment of other kinds of capital, e.g., time and money, is in turn necessary to reproduce social capital.

3.1.2 The space of social positions

The central item of Bourdieu’s social model is the space of objective social positions, which consists of three dimensions:

1. The volume of capital

In Bourdieu’s illustration of social space (see Figure 3.1), the axis y represents the quantity of economic and cultural capital (Bourdieu 1982:212f). For reasons of presentation Bourdieu omits the dimension of social capital in this diagram (which would be the third dimension, turning surface into space). Instead, he uses the development and the career of a status group over time as a third descriptive parameter.

2. The structure of capital

The x-axis represents the ratio of economic to cultural capital (with more economic capital on the right and more cultural capital on the left).

3. The social career

This aspect describes the extent to which the share of each population group identified changes over time and whether or not this group experiences upward or downward social mobility (Schwingel 1995:103).

In this space, groups of individuals that have similar positions can be identified, which means that they carry relatively similar social positions. These positions are indicated and labeled with typical job titles that statistically correspond to the social positions.

3.1.3 The space of lifestyles

Whereas the space of socioeconomic positions described above represents the structural dimension in Bourdieu's model, the space of lifestyles shows the dimension of practice, in which the economic and cultural constraints are objectified into behaviors and objects (Bourdieu 1982:137). According to Bourdieu, a lifestyle is "the unity of variety and the manifoldness of all forms of practice" (ibid.:175). Empirically, Bourdieu uses extensive statistics and interviews which elicit detailed information on the food, music, cars, literature, housing conditions, kinds of sport and leisure activities preferred by the interviewed persons in order to describe their lifestyles (ibid.:800ff).

He assigns this information to the position of individuals within the space of social positions, thus empirically evidencing the existence of a homology between the space of social positions and that of lifestyles (ibid.:286), i.e., a systematic relationship (ibid.:11) between the objective living conditions (economic and cultural resources) and lifestyles. According to Bourdieu, this relationship is not strictly causal or absolutely necessary. He calls it "structural causality of a network of factors" (ibid.:184) and "the causality of that which is probable" (Bourdieu 1981:173). Theoretically, the homology of the two spaces is justified by the theory of habitus (see next section). In the following figure, the two spaces just described are shown as if they were on two transparencies laid on top of each other. The space of social positions is in black and the space of lifestyles is in grey. As indicated by the four thick frames in the figure, the vertical dimension denotes the amount of capital and the horizontal dimension means more economic (on the right) or more cultural capital (on the left). For the present purpose it is not necessary to read or to understand every detail on this social map. The most important message is that there is a typical and probable relation between social positions and lifestyle.

3.1.4 Habitus

Habitus, as the link between the two spaces discussed above, can be defined roughly as mentality, predisposition, attitude, appearance, habit, or way of life (Schwingel 1995:54). It is a class-specific principle for producing forms of practice (Bourdieu 1982:277ff). Accordingly, the forms of practice (= lifestyles) are systematic products of the habitus (ibid.:281), which is also the individual way that experiences are perceived and classified. The theory of the habitus aims to clarify how social practice is created and how it is experienced.

“[So] ‘capital owners’ incorporate their resource equipment through the habitus, i.e. the given structure of capital structures the social perception and judgment of the habitus, which in turn shapes the forms of practice and thus [...] the lifestyles” (Konietzka 1995:80)¹⁰

The habitus is socially conditioned and “ensures the active presence of earlier experiences, which is reflected in the form of the patterns of perception, thinking, and action in each organism” (Bourdieu 1987:101). The three patterns have a combined influence and lead to the habitus being a system of durable action-generating dispositions. This process depends on the position of the individual in the social structure. Differences in the habitus therefore are always an expression of structural social inequality and classes competing.

Generally, habitus is thus the congruent incorporation of external conditions (Bourdieu 1987:50) and for the lower classes in particular the incorporation of external obligations (Bourdieu 1982:138). Although habitus is not innate, it is a social necessity that has become “second nature” to humans (Bourdieu 1992:84) or “a virtue” developed from necessity (Bourdieu 1982:585).

Although these descriptions sound deterministic, Bourdieu particularly stresses the individual’s strategic room for maneuvering as well as the variations, limited merely by perception, thought and action, which nevertheless do not determine concrete practices

¹⁰Considering the numerous quasi-definitions of the habitus offered by Bourdieu, which supplement each other, also this summary cannot prevent defining the habitus by means of many descriptions. Despite the inflationary use of the term “structure” the definition of the habitus as “structured and structuring structure” is very precise (Bourdieu 1982:279f). In principle, this is stated in Konietzka’s quotation and becomes more easily understandable when the so-defined habitus is opposed to the space of objective social positions as only “structuring structure”. On this very abstract level it becomes clear that Bourdieu regards the objective living conditions as the starting point (structuring structure), the habitus as the mediating operator [Vermittlung] (structured and structuring structure) and the lifestyle as the result of complicated processing processes [Verarbeitungsprozessen] (structured structure).

(Bourdieu 1987:103). This is similar to a speaker who can form an infinite number of sentences from a limited repertoire of words and grammar (Schwingel 1995:64). Bourdieu thus stresses again and again the importance of habitus as a mediator between resources and lifestyle.

Taste as part of the habitus depends on one's position in the space of social positions, thus depending also on income. However it remains stable under short term changes, again disproving a direct influence of the structural position on behavior.

“[that] the taste unfolds its own, lasting effectiveness, is never as obvious as when it survives its preconditions. This can be seen at those craftsmen and little businessmen, who, according to their own words, do not know what to do with their money” (ibid.:587).

Taste does not have much to do with health but the same logic can be applied to the understanding of individual health behavior that partly depends on personal preferences and the trade-off between competing goals, e.g., enjoyable consumption and health. Hradil suggests analyzing the habitus in order to understand the impact of socioeconomic status on health, which would guide the way from “abstract to concrete, from objective to subject-orientated, from descriptive to explaining social epidemiology” (Hradil 1993:390). Existing elaborations of the habitus concept for the analysis of social differences in health will be described at the end of Section 3.2.

3.1.5 Classes

According to Bourdieu, grouping in a society is based on a neighboring position in the space of social positions. Within these classes one can assume and find similar dispositions and interests and a similar habitus. But Bourdieu only accepts the term class if these similarities also exist subjectively and consciously in the view of the class members. Designations of groups coming from outside which may have a political background do not form classes.

“A social class is not only defined by its position in the economic system, but also by the habitus of the class [i.e. also the self-perception], which is ‘normally’ (i.e. with high statistical probability) associated with this position” (Bourdieu 1982:585).

Much simplified, Bourdieu differentiates between three large classes:

1. The bourgeoisie, whose habitus is based on the principle of distinction (“more existence than appearance”) (Bourdieu 1982:405ff; Reusswig 1994:68).
2. The lower middle class whose “characteristic” is to pretend (“more appearance than existence”).
3. Workers and farmers, whose habitus is oriented towards the necessary (“low existence and low appearance”).

3.2 The importance of Bourdieu’s theory

The concept of habitus is helpful in understanding the pathways of the dependence of health and mortality on social status. These pathways are not uniform in the sense that there are not merely either direct repercussions of economic or educational constraints on health, or just the opposite: a purely behavioral link between class and health outcome which could be explained by autonomous choices between healthy and unhealthy alternatives. It is the interplay between structure and agency which is addressed by the concept of habitus. As will be discussed in Section 4.4.2.4, it is not justified to understand socioeconomic differences in health and mortality as a result of differences in behavior. Even in a broad definition of behavior that would include not only smoking, drinking and physical exercise but also diet, responsiveness to preventive health care services and compliance as a patient, the observed differences cannot be explained by this factor. There are class-specific exposures and maybe even differences in the impact of unhealthy exposures (see Section 4.4.2.2). To find a sociological explanation for social differences in health, it would be promising to reconstruct the health-relevant interplay between structure and agency, and between living conditions and the way the individual perceives and reacts to them. Bourdieu does not focus on health outcomes when he illustrates the functioning of the habitus, but his empirical studies entitled “Distinction” (1979, in French) include a survey of differences in sport and eating habits between the upper-middle class and the working class. He reveals structural differences and differences in preferences concerning healthy behaviors. The following description of the dependence of consumption on income may serve as an analogous illustration, at least for situations where socioeconomic differences in health and mortality are based on individual behavior and preferences.

“if it really seems, as if there would be a direct relationship between income and consumption, it is because the taste almost always evolves from the same economic conditions, in whose framework it acts, so that a causal effect can be attributed to income, which however has this effect only *in combination* with the habitus, that produced it [the taste]. Indeed the *influence* of the habitus shows up clearly when different consumer habits correspond to the same income, which only becomes understandable under the condition that other criteria participate” (Bourdieu 1982:590).

Bourdieu’s theory is one of the most influential concepts in social structure and lifestyle research. According to critics his capital theory reduces the “social to the economic” (Honneth 1984) and his proximity to structuralism leads to a deterministic conception of men (Hradil 1989; Müller 1992). Also, his empirical evidence is no longer up to date (Blasius and Winkler 1989). A basic problem of sociological research that tries to classify individuals according to some measured qualities is expressed by Girtler:

“in this sense there are no ‘fine differences’¹¹ between the social layers, as proposed by Bourdieu, because humans, as potentially ‘respectable people’ do not let themselves be assigned a ‘layer’. They even successfully refuse to accept the classifying and typifying sociologist” (Girtler 1989:441).

In my opinion Bourdieu does not make the mistake of a structural determinism. He shows clear structures, but stresses at the same time that they only exist through the execution of individual or collective practices: “no physical agents, no practice; no practice, no objective structure” (Schwingel 1995:71). By that, Bourdieu can make necessary abstractions without losing the contact between practice and everyday life that he integrates with extensive and illustrative material. A clear advantage of Bourdieu’s theory is that he does not understand lifestyles as a “modern” concept which could replace class models in the course of social differentiation (like, e.g., Hradil 1987a; Schulze 1992:17). He rather integrates lifestyles explicitly into his class model. In doing this he finds many connections between cultural phenomena, matters of taste and preferences on the one hand and structural dimensions of inequality on the other. Thus, he does not lose sight of the important question of the class affiliation of lifestyle carriers.

¹¹ The German title of Bourdieu’s most important book is „Die feinen Unterschiede“ published in 1982, originally “La distinction” in French, published in 1979.

The use of Bourdieu's theory for the analysis of socioeconomic differences in health and mortality is limited by the fact that health is essentially different from other valuable goods in society like wealth or education. To the extent that health is distributed by social processes it shares the two important characteristics with the classical dimensions of inequality, namely being much in demand and being distributed by social processes. But this extent, to which health is socially distributed, is very difficult to measure and leaves much room for different interpretations and opinions. On the one hand, no one would doubt that unhealthy working and living conditions are socially distributed, but on the other hand the individual health outcome can never be reduced to the result of the sum of unhealthy living conditions. Genetic factors, individual health behavior and pure chance can intervene in the relationship between social status and health.

Bourdieu's theory gives further insight into the second of these factors, individual health behavior. He shows that lifestyle, with its numerous health relevant aspects, including nutrition, drinking, smoking, drugs, priorities and preferences for healthy versus unhealthy alternatives in every day life, is class specific. Thus we can think of all these health relevant factors as part of a lifestyle, which, according to Bourdieu, is the product of the habitus. In this way sociological theory can be used to explain health and mortality differences, but this explanation does not extend to all differences in health. There are also direct causal pathways from external and internal physiological factors to health and mortality that are not class-related or influenced by behavior.

Bourdieu's model has also been introduced here as one of many examples that show the relative importance of different dimensions of social status. It is theoretically and empirically well-proven that Bourdieu's three forms of capital describe an individual's social position very well. The more simplifying use of education and material wealth as only two predictors of social status used by Bourdieu is at least precise enough to use it as operationalization in an empirical study (see Section 6.1.)

In the empirical part of this paper the most complicated and innovative part of his theory, the habitus, is not translated into an empirical operationalization for two reasons: first, theoretically the habitus is not able to represent all intermediate steps that play a role in the causal pathway from social status to health and mortality. The habitus is defined as mentality, predisposition, attitude, appearance, habit, or way of life and includes some incorporated and fixed qualities. But this concept would be largely over-interpreted if extended to health constitution or genetic endowment. Second, there was

practically no information available in the two chosen datasets for the empirical part of this dissertation that could have been used to describe the habitus of the persons. Thus, far from being “applied” in my empirical research, Bourdieu’s theory serves as a background for understanding how far-reaching social influences are on health and mortality. His concept offers enough differentiation and complexity to rule out both simplifications of either separating health from the social world entirely or of treating health as just another social resource like education or income.

What we can learn from Bourdieu’s theory for the analysis of social differences in health and mortality is that social conditions can be incorporated and embodied, which makes the distinction between social and biological realms difficult. Epidemiologists have also stressed this complex interplay:

“Human bodies in different social locations become crystallized reflections of the social experiences within which they have developed. The socially-patterned nutritional, health, and environmental experiences of the parents, and of the individuals concerned, influence birthweight, height, weight, and lung function, for example. These biological aspects of bodies (and the histories of bodies) should be viewed as frozen social relations, rather than as asocial explanations of health inequalities which, once accepted, exclude the social from consideration [...] aspects of bodily form can influence social trajectory in the same way that social experience become embodied.” (Davey Smith et al. 2001:115)

Given the similarities between the incorporation of social structure and practices in Bourdieu’s theory and the health-relevant incorporation of (social) experiences mentioned above, Bourdieu’s theoretical framework offers the opportunity of extending our understanding of social inequalities in health. It may constitute a fruitful theoretical contribution to future research in social epidemiology and the sociology of health. This work cannot be conducted here because my research focuses on a different question; however, Section 4. 4 discusses the causality between socioeconomic status, health and all other factors. The next research step would be the integration of these factors into a theoretical framework. This framework may be similar to the habitus concept but designed for social epidemiology which aims to explain health lifestyles by describing the relation between agency and structure in health relevant fields.¹² Cockerham and colleagues are pioneers in this field of research.¹³ They propose a health lifestyle

¹² The aim to move beyond the agency-structure debate is probably too ambitious. Archer (1995:1) points out: “The vexatious task of understanding the linkage between ‘structure and agency’ will always retain this centrality because it derives from what society intrinsically is.”

¹³ E.g. Cockerham et al. 1993; Cockerham et al. 1997; Cockerham 2005.

paradigm based on Weber and Bourdieu where life choices (agency)¹⁴ and life chances (structure)¹⁵ interact and result in dispositions to act (habitus). These dispositions materialize through practices (action) and become health lifestyles (reproduction)(Cockerham 2005:57). That health is a relevant dimension in lifestyles can be seen by the simple observation that many individual health lifestyles are either generally positive or negative (Cockerham 2005:56). Positive health behaviors are clustered along two dimensions: promoting wellness and avoiding risk (ibid.)

3.3 Health as a dimension of social inequality?

At the beginning of Chapter 3, I suggested that the question of whether health can be regarded as a dimension of social inequality on its own depends on the question of whether health is unequally distributed by a social process. This notion can never be completely accepted given the numerous health factors that are not social. According to Hradil, it is not health but rather the circumstances of health that are socially and unequally distributed and which can be called social inequalities (Hradil 1993:377). He differentiates the following health-relevant sub-dimensions of social inequality: objective living conditions like income, wealth, education, dwelling, partnership and household patterns. These dimensions are not mutually exclusive, e.g., money is related to dwelling and education and the higher the socioeconomic status the higher the chance to be partnered in old age (Mayer and Wagner 1996:267).

According to Hradil health can definitely be understood as a consequence of social inequality, but it is not easy to say whether health can also be found in levels and categories of Hradil's system of definitions that do contribute to the creation of social inequality. At these more basic levels are the determinants of social inequality. These are social positions of individuals, e.g., sex, age, occupation, region of living, generation or ethnic group, which do not imply an advantage or disadvantage as such but which result in (dis-)advantages with a high probability (Hradil 2001:34). At the other end of the spectrum between cause and effect, Hradil says that the consequences of social inequality are the perceivable advantages and disadvantages in the living conditions, ways of thinking and behavior that let you really feel the (dis-)advantages. Obviously, health would be among these consequences. But health does not only follow

¹⁴ The German term used by Weber is "Lebensführung".

¹⁵ Life chances can be roughly equated with structure because the higher the socioeconomic status the higher the life chances, i.e. probabilities for satisfaction).

the pattern of social inequality, it produces and reproduces it (Jungbauer-Gans and Schneider 2000:228).

The problem of defining health as a dimension of social inequality can also be seen in the two central aspects of the theory of social inequality: 1. localization of resources, and 2. social relations (Kohli 1990:391). Strictly speaking, health does not have much to do with these aspects, thus it seems justified to keep health and mortality on a different analytical level from income or education. But why not define health as a valuable resource and years of life as the most valuable of all resources? Although this resource is simultaneously socially and biologically determined, the close relationship between the classical dimensions of social inequality and their effect on health make inequality in health and mortality a very good indicator for social inequality (Valkonen 1996:64). Preston and Elo (1995:476) understand mortality as one of the most central indicators of social and economic well-being and a fundamental indicator of social inequality. Following this argumentation, the distinction between dimensions of social inequality, health-relevant sub-dimensions and health as a consequence of social inequality change from being a principal to being a gradual difference. Level of education, e.g., is not exclusively distributed by social processes just as health is not exclusively distributed by non-social processes.

In old age the role of health in social inequality is especially important because health declines systematically with age. Health in old age is not only determined by age, as there are large health differences between people of the same age. But the link between age, aging and health is so close that health is part of one of Hradil's determinants of social inequality, namely age. Of course, numerical age is socially important as it defines which social norms and expectations apply to a person. But aging and health are dimensions that are implicitly addressed through general norms and concrete age regulations, for example, that above a certain age people should not drive or work, etc.

The question of to what extent differences in health and mortality can be attributed to social inequality leads to the question of whether these differences are unjust and can be called social inequity. Even more than the term social inequality, the notion of inequity includes a normative dimension. Not all differences are unjust or unfair (Elkeles and Mielck 1997; Kunst 1997:207) and most people would agree that there will always be social inequality. What should be aimed for is equity rather than total equality. The term equity again is hard to define and concepts differ, e.g., in the degree to which they are based on principle or performance-based equity.

Differences in living conditions and in conditions of choice (e.g., educational differences) can be unjust. The consequences of a choice (e.g., smoking that leads to cancer) are not unjust if and only if the conditions of choice are equal and when there is an alternative, i.e., when there is free (informed, deliberate, unconstrained) choice (Hertzman et al. 1994:77; Stronks 1997:22ff). Lifestyle is not really a choice nor is health behavior entirely a choice. Here the interplay between structure and agency as discussed in the description of Bourdieu's concept can be seen again. Even rational choices are not voluntary (Giddens 1976:16; Dannefer 1992:42).

In a specific situation there might be an alternative and a free choice in principle, e.g., the choice not to smoke a specific cigarette in a specific moment. But the systematically worse health behavior in lower social classes show that the conditions of choice, e.g., knowledge about consequences, independence from group pressure, and alternative ways of expressing feelings, lifestyle and becoming integrated, differ between classes. Therefore, health behavior is not just a matter of choice and cannot be attributed completely to individual responsibility.

Once the structural origin of many risk factors in lower social classes and the empirical evidence that health differences are systematically related to and partly caused by social status is accepted, health differences can be called unjust. Again, the reason that health status is not accepted as a dimension for social inequality is that there are still biological and random predictors of health that cannot be regarded as unjust (Murray et al. 2001). There are individual choices that no one else other than the individual can be made responsible for. But the simple fact that a lower class person knows, or should know, that smoking is unhealthy is not enough to call the risky choice to smoke a "free choice" and to negate the question of whether the large social health differences can be called unjust.¹⁶

Different social inequalities are differently perceived and accepted in society. The acceptance of e.g., income inequality is rather high (Berger and Schmidt 2004:7), especially between groups with different educational levels, because it is perceived as a motivation for better occupational performance. Assumably the acceptance for health and mortality differences between social classes is much lower because good health as a reward of great effort in education or occupation is much less plausible than in the case of income.

¹⁶ For the analysis of the association between class and smoking see Graham 1994.

Finally, it is important to consider the social circumstances that should apply to such theoretical reasoning. Huge international differences in the general level of wealth, sanitary conditions and health care make it difficult to agree on one logical framework of definitions for health and social inequality. In a wealthy society the health dimension is one possible dimension of showing social advantages and disadvantages. Rich people have better health on average than poor people. But these are gradual differences and even for a lower class person in a rich society it is relatively easy to maintain a good health status with the help of social services. If a health problem occurs, the quality of life and the overall well-being of these persons can still be high. In contrast to this, in one of the unhealthiest slums somewhere in the “third world” or in an arid region of Africa it is almost sure that people with low social status will get diseases that are avoidable elsewhere, that they will subsequently die at young ages. Only exceptional cases can escape this destiny that is a direct result of social distribution processes. It is not only that these persons lose their good health easily, very often health is the only resource that they can invest in order to survive. In such a situation where social destiny is almost identical with health destiny, the factors mentioned above – that make the health dimension principally different from class-relevant entities because they partly depend on genes, free choice, coincidence, etc. – are irrelevant. Thus, the above considerations are more useful for the analysis of socioeconomic health differences in rich countries such as Denmark or the USA.

The next section will also take the global perspective and discuss another theoretical consequence of very different levels of wealth and welfare in different societies: the problem of absolute versus relative deprivation.

3.4 Relative deprivation

The concept of relative deprivation was originally formulated by Stouffer (1949) and is now used rather arbitrarily in the literature. It stresses two aspects. First, disadvantage in status depends on a comparison to other persons. Second, for this comparison, individual perceptions and interpretations of social inequality are important in addition to objective differences and objective under-supply. In rich countries like the USA and Denmark, relative deprivation plays an important role in the assessment of social inequality which in principle is always relative because statements like “a poor or less educated person” are relative to the social structure the person lives in.

Regarding rich instead of poor countries it is plausible that social constraints for health become more important than material ones. Relative deprivation is linked to social and psychosocial problems whereas absolute deprivation is linked to material problems. Material deprivation can be defined as the inability to participate fully in society and have control over one's life (Marmot 1999:23). This definition suggests that there should be a threshold of material wealth above which such deprivation is avoided. But this threshold is not absolute; it rather depends on the overall level of wealth in a society which determines the necessary means to participate in social life. The fact that there are health and mortality differences even between the rich and the very rich groups of a society speaks for the importance of relative deprivation and psychosocial factors (Marmot 2000:362). Thus, the notion of poverty becomes relative: you know what others have and in principle deprivation is possible on all absolute levels of wealth (Vågerö and Illsley 1995:226).

The independent existence of relative deprivation and its impact on individual well-being is an explanation for the fact that there is a social health gradient in all societies, even the richest, and second for the observation that relative deprivation (and social health differences) can grow even if the absolute average level of wealth increases (Vågerö and Illsley 1995:227). Such an upward shift of all social classes may imply that disadvantaged groups are getting smaller, which implies progress in overall well-being, average values for income, education, health and mortality. But inequality may still stay the same or even increase (Kunst 1997:57). Investigations for many countries show that social health inequalities are increasing while in some countries they remain stable (e.g. Gustafsson and Johansson 1999; Valkonen 2001:8826, see Section 4.2.2)

This paradoxical development can be found in many modern countries: on the one hand the prosperity level has risen enormously in many countries since World War II; on the other hand the divergence between rich and poor people has continued to increase at least since the 1980s. Both statements as such are correct. However, it is important to differentiate between levels of the respective diagnosis in order to avoid that one diagnosis is abused for the refutation of the other, like that the persistent problem of poverty would be ignored.

For the rise of prosperity of the entire population the metaphor of an elevator by Ulrich Beck has become famous:

“more life-years, less working years and more financial playroom – these are the corner-stones, in which ‘the elevator effect’ is expressed in the biographic framework of the people. A radical change in the relationship between work and life took place - with constant relations of inequality” (Beck 1986:124).

Gerhard Schulze uses similar expressions: “both the rich and the poor have become richer” (Schulze 1993:191). But there are also contradictory opinions which state that there was no common upwards shift but rather there was much more improvement for the middle and upper classes than for the poor with the result that social inequality increased (e.g. Geißler 1996:321).

The fact that an objective improvement can be combined with a deterioration of the relative social status stresses the importance of the interplay between objective versus subjective social status considerations. Schulze (1993:183) asks:

“What becomes of social inequality during a long period of prosperity? [...] social reality depends on how humans process their life circumstances subjectively; subjective conceptions are for their part considerably determined by objective conditions” (Schulze 1993:183).

Summarizing the relationship between objective situation and subjective perception Schulze says that “the happiness (Glück) of the people does not rise proportionally to their prosperity” (Schulze 1993:192). In the relationship of absolute and relative social status I consider the following aspects to be important: The debate about a generally rising level (education level, prosperity level, etc.) should not obscure the view of the internal differentiation of this process. This differentiation cannot only weaken the association but can also change a general trend for certain groups into the opposite trend. Therefore, generalizations like the elevator metaphor, the transitions from scarceness to affluence and from obligations to choices diagnosed by Schulze (1992) or consumers making free decisions (Lüdtke 1989:54) do not adequately describe increasingly differentiated and different social situations.

3.5 Social inequality among the elderly

It is difficult to consider social inequality among elderly persons because many of the classical parameters and dimensions for identifying social inequality are based on the labor market. After leaving the labor market these positions can only have an after-effect and it is unclear how strong these effects are relative to new and current living

conditions. One of the conditions which unifies all pensioners and that still refers to the logic of the labor market is the large amount of leisure time. This quality of the elderly has been used to draw a line between them and younger persons in the social structure (O'Rand et al. 1999:36). The absence of paid work is a feature of most elderly people's lives. But of course considering this criterion alone would portray the elderly as a homogeneous group without paying enough attention to its internal distributions and inequalities. For this inequality the extent to which the classical descriptors of inequality among younger people are still valid and useful has to be revealed. Furthermore it is interesting to observe which new dimensions (if any) become important for an aging individual and also for an aging society. The question to ask is whether there is a different kind of social inequality in old age (Vincent 1995).

It is important to analyze social inequality among the elderly because they are often considered a group that is growing and causing a financial problem for the entire society. This burden is real and the discussion is necessary but how this burden can be distributed in a fair way depends very much on the diagnosis of the wealth distribution and thus on social inequality within the group of elderly people. If wealth is very unequally distributed within the group of old-aged people, it is not plausible to suggest that either the young have to subsidize the old or vice versa.

Hradil understands age as a determinant of social inequality and as argued above I would add that aging as well as health are relevant for an individual's social status, because both can considerably reduce all three of Bourdieu's types of capital (Woll-Schumacher 1994:222). Within one social group older persons are likely to be more deprived (Vincent 1995:31). Older persons are economically inactive, they are more likely to be single because of widowhood, and many of them live in nursing homes (Martelin 1994:1276). On average they are less engaged in social activities but they do not have less money than younger persons.

Age is not only a biological but also a social variable and a social category that determines social roles, norms, and expectations (Arber and Ginn 1993). It is unclear if there is a loosening or a strengthening of the relation between age and social roles over time (O'Rand et al. 1999:2). Theoretically, society could become more and more age integrated as age loses its power to regulate individual life (O'Rand 1996b:192).

What makes social inequality in old age different from in younger ages is the fact that more biological processes are involved. It is difficult to separate these biological

processes from social mechanisms because they interact with socioeconomic status. The fact that more biological processes are involved in social inequality does not necessarily mean that the overall importance of biological aspects relative to social determinants is increasing. On the one hand a universal health decline takes place for everyone at a certain age and this may affect social status, but this change is neutral to social inequality as a whole. On the other hand the aging process is very variable and interacts with many social factors such as socioeconomic status. It is conceivable that the aging process makes old age inequality different and even more acute than inequality in younger ages. Still aging process is not neutral to social inequality nor is it a simple continuation of class differentiation (Steinkamp 1993:15; Backes et al. 1998:174).

Here are some examples of how the aging process can have different consequences for different social status groups: persons with higher social status get more institutionalized help because they have more money and get along better with the administration (Woll-Schumacher 1994:241, 246). To the extent that care is privatized, access to care depends more on private money and thus inequality of access will increase (ibid.) When minimally educated persons have to change jobs because of age or health problems they experience a downgrading. Persons in high positions are more likely to be offered a suitable job at a high level according to their needs (ibid.:225). It is unclear whether people in lower classes have more social contacts because they have more children (which additionally are more likely to live nearby) or whether they have fewer social contacts because the size of networks and the number of friends outside the family is positively correlated with social class. Social contacts, especially in the family, are not necessarily positive (ibid.:238).

Other unanswered questions are, e.g., whether better educated persons are more able to cope with illness and the threat of death than lesser educated persons (ibid.:236) and whether old people suffer more from social disadvantages and are thus less satisfied than young people (Dannefer 1987).

Woll-Schumacher summarizes the interplay in which socioeconomic status influences the process of aging. Aging and health have repercussions on socioeconomic status as follows: Aging is only really bad for low status groups. Aging reduces the advantages of high social status and increases the disadvantages of low social status (ibid.:248).

After this overview of general features of social inequality in old age and the related open questions, three distinct scenarios of how social inequality changes with age

(which also means within the life course) can be specified (Mayer and Wagner 1996:253ff; O’Rand et al. 1999:36ff). When we look at the change of differences over age we consider differences within and between age groups. This approach reflects the understanding that aging is open to social influences that may increase or decrease variability over age. The very ambitious theoretical goal of this approach, that cannot be further followed here, is to combine the social and individual levels by finding a social explanation for individual developments and their variability (Dannefer 1987:226ff).

3.5.1 Status leveling hypothesis

It is possible that aging works as a leveler of social status because biological processes assume dominance over social determinants and eventually everybody must die regardless of social class (Liang et al. 2002:295). Thus there may be stable social inequality in old age but it has less of an effect on social status and social activities, except perhaps in the case of the impact of education which is increasing (Mayer and Wagner 1996:266). A different assumption within the status leveling hypothesis is that the welfare state actually reduces socioeconomic differences in old age through benefits and social security (Ross and Wu 1996:107). This is also called the redistribution hypothesis which stresses that in many industrial countries inequality among the elderly is less pronounced than among younger groups (O’Rand et al. 1999:11). It assumes that, with the change in the main source of income from earnings to annuities from social security, the latter of which has a progressive redistributive structure, social inequality in old age is reduced (Crystal and Shea 1990).

The status leveling hypothesis is sometimes presented with a slightly different argumentation under the name of age dependency hypothesis (Mayer and Wagner 1993:525ff; Mayer and Wagner 1996:254). It claims that one’s social situation changes with age. This may work through social ascriptions to certain ages (Kohli 1990), institutionalized rules, e.g., concerning labor force participation and pensions (Mayer and Müller 1989) or, like the above argument, through the dominance of physiological factors over social conditions. An example of physiological factors taking priority is when illness and disability limit mobility and the quality of life to such a low level that the social inequality in what elderly people can do and reach is limited, too. Or, as Mayer and Wagner (1996:255) phrase it, the playroom is so restricted by illness and disability that individual resources cannot compensate for this (see also Backes et al.

1998:83). Mayer and Wagner point out that such a leveling is likely if health, health care and wealth are relatively independent from each other (Mayer and Wagner 1996:255), which we will see later is not the case.

Generally, an analysis of the change of social inequality with age has to consider the possibility that besides our attempt to find objective descriptors for inequality, equality or inequality can also be ascribed socially to a certain age group. This social ascription may then become socially meaningful and change our perception of inequality in old age (Mayer and Wagner 1996:253f).

3.5.2 Status maintenance hypothesis

This hypothesis assumes that there is continuity between the social status of people in middle age and old age with the result that the social structure and the degree of social inequality in old age are not very different from younger ages. This status maintenance is based first on the influence of the working age on the retirement age through external structures where the individual has a persisting position. Second, status maintenance can be based on internal dispositions like learning behavior, habits and one's own self-concept (Kohli 1990; O'Rand et al. 1999:69). This continuity theory (e.g. Atchley 1989) has some support in the empirical finding that wage inequality converts more or less into pension inequality (Pampel and Hardy 1994; O'Rand et al. 1999:9) and that there is a high correlation between an individual's working income and pension (Kohli 1990:395). This leads to the conclusion that there is also a continuation of social inequality from middle to older ages (Backes et al. 1998:84).

Kohli claims that the assumption that a simple continuity exists is not satisfying and discusses possibilities of understanding elderly people as a class of their own. Lepsius' (1979:197ff) idea of the "Versorgungsklasse" (a class whose social status is defined by entitlement to social benefits) is an example; this in addition to Weber's "Erwerbs- and Besitzklasse" (a class based on employment and property). But this concept would still be concentrated on income and the income source. The theoretical possibility that the elderly constitute a class that is different from other age-defined classes has never been realized (Kohli 1990:397). Kohli concludes that the age limit is not very useful in terms of social class theory. A possible reason for the absence of an age-defined class is that it is unlikely for individuals to belong to a class in younger ages and to change the class just by reaching a certain age. And it is even more unlikely that all persons of an age

group belong to a single class whose common characteristic is age rather than income, education, or lifestyle, etc.

But for a deeper understanding of social structure in old age it is not enough to justify continuity with the argument that class and habitus are life-long attributes: “Continuity must be documented and explained” (Kohli 1990:398). This would be an ambitious research problem, one that cannot be started here. But there are certainly many unanswered questions in this field. Kohli states that continuity and discontinuity are not the right alternatives. Instead, he asks for an analysis of the structural conditions that allow continuity and discontinuity and he suggests to include time in our theoretical constructs (“Verzeitlichung des theoretischen Apparates”)(Kohli 1990:399).

Given the important relation between individualization and social inequality in sociology, another question is whether there is more or less individualization for old people. If it is also true for them that lifestyles become more independent from living conditions, as Hradil has claimed (Hradil 1987:122) and if they increasingly depend on formal institutions and less on family, it is likely that there is individualization in old age, but maybe of a different kind or to a different degree. Finally, it would be helpful to gain more insight into the way different biographical age-structured pathways and an assumed path-dependency determine old persons’ life chances and life choices besides the increasing importance of age that has been discussed above.

3.5.3 Cumulative advantage hypothesis

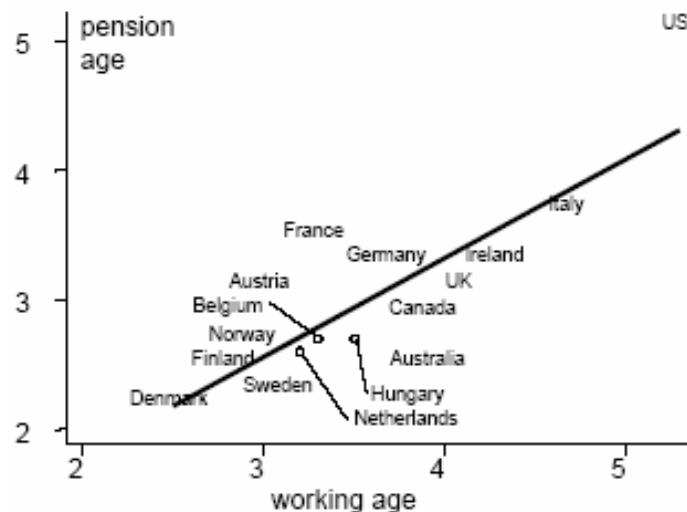
For this hypothesis the basic assumption is that there is an accumulation of social (dis-) advantages over the life course (Crystal and Shea 1990). According to the logic of the accumulation of capital (see Bourdieu’s theory of capital in Section 3.1.1), it is plausible that a higher social status would allow an individual to achieve more and more advantages. This would lead to higher social inequality in old age.¹⁷

The empirical proof for this scenario is difficult. Social inequality in old age is likely to be underreported because the sources that are important for elderly are the most underreported (Crystal 1996:392). In old age wealth is more important than income (Bäcker et al. 2000:303) and in the USA home ownership contributes most to the assets of a household (O’Rand et al. 1999:55). In the USA income inequality among the elderly is higher than among younger people (O’Rand et al. 1999:69) but in many

¹⁷ The accumulation of health differences is discussed in detail in Section 5. 2, argument 6.

countries, like Denmark, e.g., income inequality in old age is less than in younger ages because of the progressive structure of the pension system. But this is not necessarily true for wealth and an individual's overall financial status (O'Rand et al. 1999:200f). In Germany, e.g., wealth is more unequally distributed than income and this is true more so in old age than in young age (Bäcker et al. 2000:308). In the USA both income and wealth are more unequally distributed in old age than in younger ages (O'Rand 1996a:231; O'Rand et al. 1999:69).

Figure 3.2: Income inequality among pension-age and working-age populations in 16 countries: ratio of 90th to 10th percentile of income

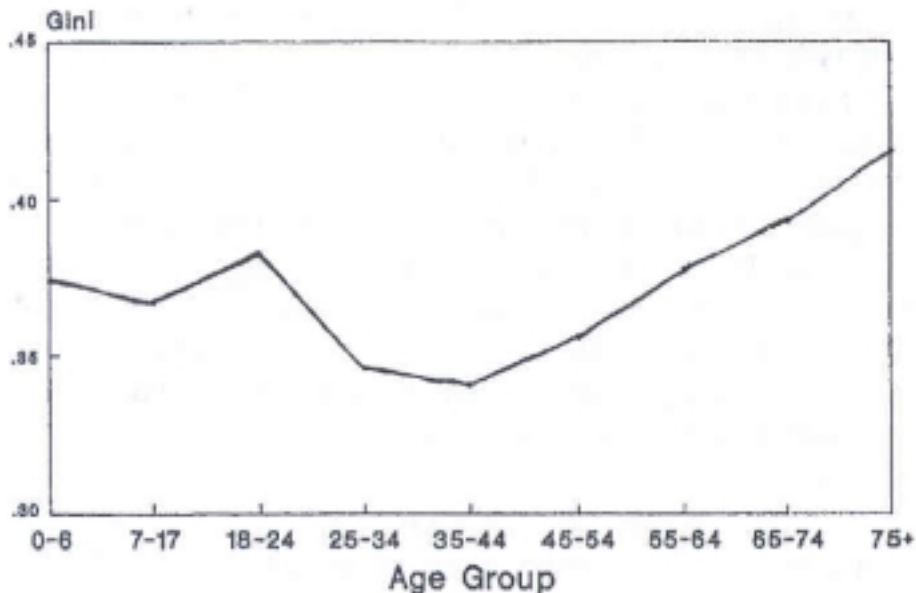


Source: Förster and Pellizzari 2000. The data are from 1994 and 1995, with the exception of Italy (1993). Fitted values are estimated from the equation: pension-age ratio = 0.9874 + 0.8655*working-age ratio with standard errors of 0.5568 and 0.1803 respectively.

In this graph the mean inequality at working age is 3.5 and at pension age it is slightly smaller at 3. This means that the persons in the 90th income percentile receive three and a half times more income than those in the 10th percentile, whereas at pension age they receive just three times more. There is a clear correlation between inequality in middle age and in later life. The graph also shows that Denmark has the lowest inequality in both dimensions, inequality in pension age being slightly lower than the inequality for working age. The USA is on the other end of the spectrum of countries with the highest level of inequality in both dimensions. The other feature of the USA, the fact that income inequality increases with age as mentioned above, cannot be seen in this graph. In both age groups the ratio of the 90th to the 10th percentile of income is slightly more than 5. However, the data point for the USA, like for France, Austria and Belgium, is

well above the regression line. In these countries income inequality in old age is not smaller than in working ages. Figure 3.3 below shows clearly that the inequality in income (Gini-index) increases with age in the USA.

Figure 3.3: Gini-index for the USA at different ages



Source: Crystal and Shea 1990:440

Another aspect that contributes to an accumulation of inequality is the fact that certain inequalities only become visible and effective with a poor health status. For example, the question of whether a person can afford to pay for help in the household only becomes crucial when the person is disabled. This is another example where health interacts with social status. It shows that it is difficult to stop the discussion of social inequality in old age without discussing inequalities in health. However, this section tries to consider social inequality and discusses three possibilities for how this inequality could change with increasing age (leveling, maintenance and accumulation). We will see in Chapter 5 that there are the same three hypotheses for the discussion of health differences and for the discussion of mortality differences. The concrete pathways for how health can affect social status and vice versa will also be discussed later.

A field of research that is related to social inequality in old age deals with the question of whether individual differences increase with age. This perspective focuses on the life course, collective historical experiences, and the psychological rather than sociological question of to what extent an old individual is the product of biography. If individual

characteristics and personality, which do not necessarily belong to the category of social inequality, become more pronounced in old age, the question remains: are these differences due to social influences and differences or is this a triumph of the individual over the social environment? (Thomae 1983, discussion in Kohli 1990:394f). This question cannot be addressed here.

Dannefer (1987:224) mentions another rather psychological aspect of inequality by showing that life satisfaction increases with age. This could mean that subjective social inequality also decreases with age (buffering), maybe because of a legitimization of the biography as a preparation for death. Small groups, e.g., couples, also become more equal and thus reduce overall heterogeneity. There is a complex interaction between groups and levels for which either increasing or decreasing heterogeneity can be assumed (Dannefer 1987:226). But this subjective “creation” of homogeneity is only a reaction to an existing objective heterogeneity and inequality and shall not be further discussed in this sociological analysis. Keeping a sociological perspective regarding this problem is justified here because, regardless of individual characteristics like personality or the interpretation of one’s own biography, individuals in old age still belong to social groups where members share the same social position.

The three hypotheses or scenarios presented in this section are not mutually exclusive. Some pathways that lead to a leveling of social inequality with age may exist together with other processes that increase inequality. A simple empirical view on how inequality changes with age can only reveal the combined net effect of all involved processes. This is why it is important to have a collection of theoretically possible explanations for an empirical finding. Research in this field has to be detailed enough to allow for evaluation of and discrimination between different explanations. My contribution to this field is to explore the change of differences in health and mortality over age.

3.6 Gender differences in old age

As described in the introduction of this chapter, Hradil understands gender as a determinant of social inequality. “It is not an advantage as such to be a man. But considerable advantages are associated with the male gender in our society” (Hradil 2001:34). With respect to the difficulty of defining the exact theoretical meaning of sex

for social inequality, sex resembles health as was discussed in Section 3.3. Analogous to the above citation by Hradil, we can say: “You don’t have to die earlier when you are poor, but you probably will¹⁸”. Hradil’s distinction between something that is not a disadvantage as such, but in most cases results in a disadvantage, is not satisfying. One reason for this is again that sex, like health, has a biological component that cannot easily be integrated into a social explanation. Unlike for the interplay between health, aging and social status, I will make no attempt to explain or interpret the role of sex for the definition and the empirical analysis of social inequality in old age but will instead just give descriptive information on gender differences.

One basic fact is that women have a life expectancy that is about six years longer than male life expectancy (Luy 2002). In Denmark and the USA, women on average live about 5 years longer than men. Because of this there are more women than men in the elderly population. In heterosexual partnerships the woman is likely to be the younger partner. This, combined with the higher life expectancy, makes it very likely that a wife survives her husband, resulting in a lot of single women in old age, who moreover have a lower chance than men of getting married again (Woll-Schumacher 1994:237). Another consequence is that most old men have a younger and healthier wife to take care of them but most old women do not (Backes et al. 1998:86).

In the last century, female labor force participation substantially increased because of shifts in the economy towards the service sector and the increase of part-time jobs, among other reasons (O’Rand et al. 1999:60). But still women have a work life that is irregular and on average consists of fewer working years compared to men. This, combined with their higher risk of living alone in old age, results in a higher poverty risk for females. Backes (1997:212) says that being old and female sums up to a double inequality.

3. 7 Description of social inequality – USA and Denmark

In the 1950s and to a lesser extent in the 1960s, there was a period of great prosperity and economic growth in the USA that was comparable to the German “Wirtschaftswunder”. After that, a falling living standard can be observed for a large part of the population from 1960 to 1986 (Pappas et al. 1993:107). Other sources even

¹⁸ Bourdieu’s appealing notion of “the causality of the probable” (Bourdieu et al. 1981:173) is no solution to this problem of identifying causality.

find that income decreased for the majority of US-Americans from 1979 to 1995 (Ostner 1998:234). How these descriptions fit together with the linear increase of the GDP shown in Figure 2.3 is hard to say. Maybe the inequality increased so much that the increase of GDP only went to certain segments of the population. From 1980 to 1994 the family income of the richest 40 percent of the population increased and the income of the poorest 40 percent decreased so that in the 1990s the richest 1 percent of the population owned 40 to 50 percent of all wealth. The total gain in net financial wealth from 1983 to 1989 was distributed very unequally: 66 percent went to the richest 1 percent, 37 percent was received by the next 19 percent in the wealth distribution and the poorest 80 percent of the population lost 3 percent on average (Wolff 1995).

Concerning inequality, there are substantial differences by state in the USA. In New Hampshire and Utah, the poorest half of the population gets about 24 percent of all income but in Louisiana, New York and Mississippi, they only get about 18 percent respectively (Kawachi and Kennedy 2001:19f). The level of inequality in the 1990s in the USA was as high as in the Great Depression of the early 1920s (ibid.:86). In terms of the distribution of material wealth, the USA is very unequal, namely the most unequal country in the industrialized world (ibid.:19). In 1994, out of 260 million people in the USA, 38 million (14.5 percent) lived in poverty, i.e., they had an annual income of lower than \$7,500 for an individual (Lynch and Kaplan 2000:24; US Census Bureau). In 2003, out of 288 million Americans, 36 million lived in poverty (12.5 percent, with a poverty threshold of \$9,400). The percentage of poor people that are elderly is lower than for the total population. Even if they are not poorer, people above age 65 are sometimes called disadvantaged because of their limited access to other resources (e.g., social and cultural capital). Other disadvantaged groups are women, widows, singles and people with poor health and/or low education (O’Rand 1996a:232). In cross-sectional data, the economic status of elderly people declines with age while poverty rates increase. A relatively low financial status of the very old may be due to the fact that these people had to spread a given amount of resources over an unexpectedly long lifetime (Soldo et al. 1997:2). Second, people in old age may have spent a large part of their resources on health care and, third, the decreasing level of wealth is perhaps a cohort effect in cross-sectional data. People above age 80 belong to cohorts with an overall lower lifetime earning than subsequent generations and the inflation of the 1970s may have reduced the real value of their private pensions (ibid.)

Concerning the time trend, during the mid-1980s, the real non-home assets of the elderly increased but during the early 1990s the net worth of the elderly declined (O’Rand et al. 1999:51). Out of 100 aged couples, 75 live in their own home, 80 percent of them mortgage-free (Elder and Caspi 1990:102). Although there is no special poverty among the aged any more, wealth is very unevenly distributed. This has also been shown by Smith (1995:2) with the HRS/AHEAD data sets that will be used for the empirical analysis of this dissertation. In the USA a clear trend toward a widening of social inequality can be diagnosed (O’Rand et al. 1999:1).

Despite the declining wealth from retirement age to old age in cross-sectional data, the elderly are absolutely better off than middle aged, especially at the lowest income groups. But they are also especially unequal compared to younger groups because the income sources characterized with a great deal of inequality (pension, savings) become more important relative to the equal sources (Social Security, Medicare) (i82 O’Rand et al. 1999:69). This may seem counterintuitive because some of these equal sources are especially implemented for the elderly but it seems that these benefits cannot outbalance the unequal effect of other sources, e.g., pension (O’Rand et al. 1999:46ff).

Thus even if, as some authors argue, income inequality decreases after age 65 because the more equal Social Security benefits get more important, this is more than outbalanced by the increasing importance of wealth which is more unequally distributed than income (Crystal 1996:392) and increasingly unequally distributed with age (for discussion and literature see Crystal 1996:396). The same argument comes from Crystal and Shea (1990:441):

“The three legs of elder support are Social Security, pension, and assets. [...] private and public employee pension income and asset income outweigh the equalizing effect of Social Security pensions and of means-tested benefits like SSI. [...] This system, which benefits from taxation advantages which create enormous ‘tax expenditures’ [...] is a major element in the generation of inequality among elderly people.”

Concerning the inequality between genders, similar trends of modernization can be found in both countries. In both the USA and Denmark the female labor force participation rate is high compared to other countries. Figure 3.4 shows the trend for Denmark from 1940 to 1990. There is a steep increase in the participation rate for non-married women from 1960 to 1990 but the rate for married-women decreased from 1940 until 1970. The percentage of women in the total labor force was astoundingly

similar between Denmark and the USA: it was 32 percent in both countries in 1960 and 46 percent in both countries in 2002 (World Bank 2004). In the 1980s, Denmark had a slightly higher proportion of women in the labor force.

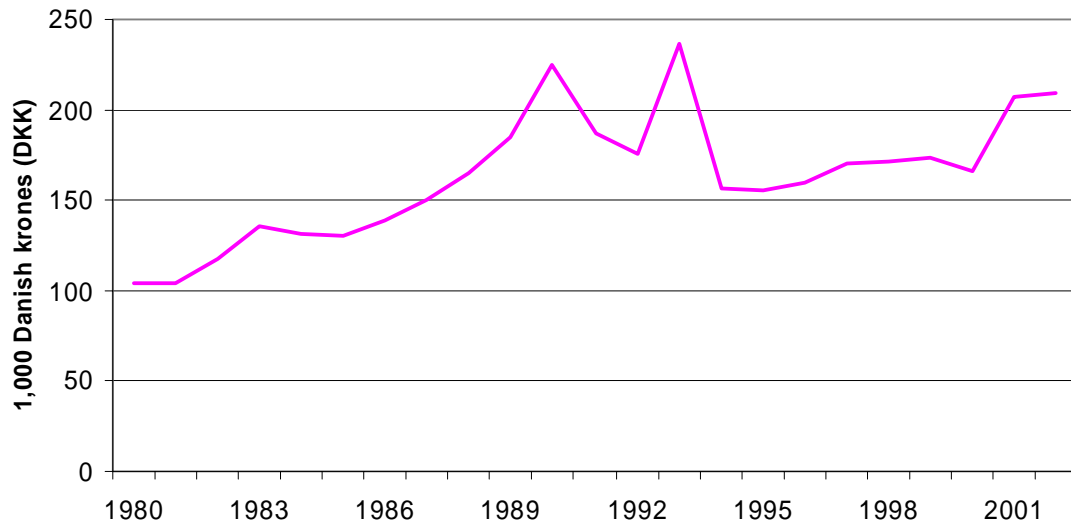
Figure 3.4: Labor force participation rates of married women, unmarried women and men in Denmark



Source: Sundhedsministeriet 1994d:47

For Denmark the large set of register data allows to describe with great exactitude the distributions in the population. Figure 3.5 shows the standard deviation of the distribution of annual gross-income for all Danes above age 58. This measure for the inequality in the income distribution clearly increases over time from just above 100,000 in 1980 to more than 200,000 DKK in 2001. The beginning of the 1990s was a period of especially high income inequality.

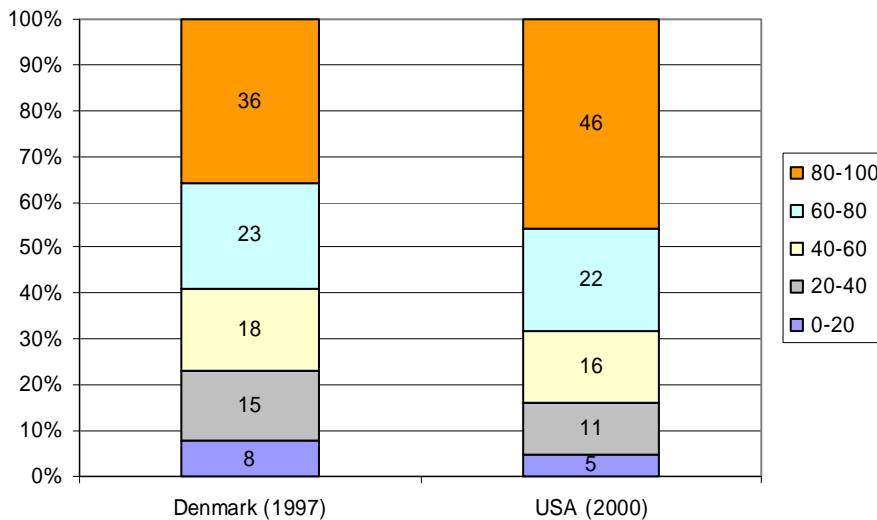
Figure 3.5: Standard deviation of income distribution in Denmark, 1980-2002



“The USA and Denmark can be seen as the two extreme cases regarding government intervention to reduce poverty” (Hussain 2002:2). USA and the Scandinavian countries are also at opposite poles in terms of income inequality (Kunst 1997:125ff). The Gini-Indices for income are: USA 40.8 (2000), Germany 28.3 (2000), Sweden 25.0 (2000) and Denmark 24.7 (in 1997). Denmark, with Hungary and Japan, has the lowest value of income inequality among all countries measured by the World Bank. On the other end of the spectrum there are countries that are much more unequal than the USA. The world leader in income inequality is Namibia with a Gini-Index of 70.7 in 1993.¹⁹ The next figure also shows the latest available data from the World Bank for the income distribution of Denmark and the USA.

¹⁹ Gini-Indices are not measured every year and the above numbers are the latest available.

Figure 3.6: The share of disposable income received by each income quintile in Denmark and the USA, 1997 and 2000



Source: World Bank 2004

All but the highest income quintile in Denmark get a higher share of the total income than in the USA. This advantage for the poor and the middle income groups in Denmark is substantial in the lowest income quintile and gets smaller towards higher income groups. On the other hand the richest 20 percent of the population in the USA receive 10 percent more from their total income than in Denmark.

Not only is the overall income inequality higher in the USA than in Denmark, inequality is especially high among the elderly in the USA. U.S. elderly are perhaps the most unequal of all age groups in all industrialized countries (O’Rand et al. 1999:2) and from that group, elderly single women are among the worst off. Their minimum benefit as a share of the median older income is much lower than in Sweden and Denmark (O’Rand et al. 1999:205). In Denmark fewer aged persons live in poverty.

3.8 Definition of socioeconomic status (SES)

Since socioeconomic position is the key concept for which the relationship between health and mortality will be described and analyzed in this dissertation, it is necessary to give a definition of the term socioeconomic status. Socioeconomic status means control and desired resources (a19 Oakes and Rossi 2003:775). To a large extent this definition is the result of the detailed discussion of social inequality because social inequality means nothing more than inequality in socioeconomic status.

“The better or worse position of an individual at the higher or lower end of a dimension of social inequality is usually called ‘status’. In the newer sociological literature about inequality this notion is used for all dimensions of social inequality. Accordingly, a prosperity status, a power status and a prestige status can be differentiated. However, in the older sociological literature on stratification the notion of ‘status’ refers solely to the position in hierarchy of prestige” (Hradil 2001:33).

A differentiation between social status and economic status is only plausible insofar as some indicators that are used to operationalize socioeconomic status are economic or financial variables and others are social. They are social in the sense that they involve a person’s relationship to other people and cannot be directly translated into economic categories (Link and Phelan 1995:81). But beyond that, there is no consistent distinction between social position and economic position because the economic position depends on social distribution mechanisms which are based on one’s relation to others.

Bourdieu’s theoretical framework stresses the need to describe and understand these different dimensions together. He uses cultural, economic, and social capital in mutual interaction. These three sorts of capital represent the three main dimensions of social status and they can be subdivided into more detailed dimensions (see Section 3.1.1). Cultural status is rarely used as a concept because cultural capital, e.g. education and qualifications, does not directly imply a status level as such but is used to accumulate capital and convert one capital form into another. Although these dimensions all belong together for a complete description of social status, Bourdieu ascribes special importance to economic capital because it can be easily converted into other sorts of capital and therefore is especially valuable. Within this economic dimension income is used by Bourdieu because it has a central meaning for a person’s economic status. Although this central meaning of income may be different for elderly persons, it may still be the best single indicator for this dimension, especially if different kinds of income sources are considered.

As in the definition of social status it is also true for the definition of social inequality that economic inequality can not really be separated from social inequality. Any valuable resources that are socially distributed are possible dimensions of social inequality. The reason why the economic resources cannot be separated from social resources is that the former are socially distributed so that economic or material conditions become social conditions (Link and Phelan 1995:81).

The notion of status compared to the similar term of position makes allusion to different layers which structure society vertically. There is an ongoing discussion of whether these layers actually structure society in the sense that they build classes or if all similarities on the aggregated level are just statistical categories and no real social groups. I think what is real, both statistically and socially, are the differences. Thereby, it is always possible to represent these differences on a continuum with no visible breaks. But it is not just a statistical artifact to oppose two different social groups and describe them by their average socioeconomic characteristics. As long as significant and important correlates with these socioeconomic indicators can be found, e.g. health or mortality, these differences do not only have a statistical but also a social meaning.

Concerning the practical operationalization of socioeconomic status for the empirical part of this dissertation, more detailed information and justification for the chosen independent variables will be given in Section 6.1. Only a broad description and justification of my approach should be given here: I tried to include as many plausible variables as available in the analysis to get a somewhat complete picture of the socioeconomic determinants of health. Of course, many desirable items are not available in my data or are not even measurable in principle. But I think it is better to look at many different contributions and influences in relation to each other and with the respective interactions than to either define groups by a single parameter, e.g. occupation, or construct an index for social status. Such an index can include different dimensions and weight them according to their relative importance but afterwards we must assume that these indicators with their relative importance stay the same in all models for both sexes, all ages, all causes of death, etc. This assumption is not plausible and therefore keeping different dimensions separate seems to be the better alternative in a situation where the impact of a multidimensional concept like socioeconomic status on health and mortality is going to be analyzed as a first part of the empirical analysis.

The main dimensions are similar to those identified by Bourdieu to be important descriptors of socioeconomic status, namely income and education. Social capital could not be treated as a dimension with equal emphasis because very little information about social capital was available in the two datasets.

Summary of Chapter 3

When some people get more of the “valuable goods” in a society than others it is called social inequality. There are different kinds of goods that can be unequally distributed. In Bourdieu’s theoretical framework these goods are classified as economic, cultural or social capital. According to him the amount and composition of capital defines the individual socioeconomic status. This position, i.e., the available amounts of different kinds of capital, determines to a large extent the health-relevant living conditions, the individual habitus and, as such, lifestyle, behavior and taste. Although Bourdieu’s theory is not made to explain social health differences, it shows how far-reaching the influence and predictive power of socioeconomic status is.

In sociological theory it is not health but rather health conditions that are considered a dimension of social inequality. It is questionable if this distinction holds, since worse health conditions normally lead to worse health, and lower status groups have systematically worse health and higher mortality. In the section about relative deprivation the effect of an overall increasing level of wealth on the meaning of social inequality is discussed. Social inequality among the elderly is of special importance to this study. In older ages health is more important for living conditions and has to be connected to our understanding of social inequality. Considering this interplay between social inequality and health over the life course, three hypotheses are possible about the change of social inequality over age: status leveling, status maintenance and cumulative advantage. Empirical findings show that social inequality is rather stable or even increasing with age. The comparison between Denmark and the USA concerning inequality shows that the USA is much more unequal than Denmark. The concept of socioeconomic status is defined very similarly to the definition of social inequality: the same resources that define social inequality (income, education, prestige, etc.) also define individual socioeconomic status.

Chapter 4 Socioeconomic differences in health and mortality

4.1 Socioeconomic differences in health

The finding that lower classes have worse health is widely accepted as a fact (Lundberg 1991a; Townsend and Davidson 1992; Thorslund and Lundberg 1994; Mackenbach and Kunst 1997). Likewise, Vågerö and Illsley (1995:220) state:

“...it is almost universally agreed in the academic literature that social class differences in health are real, a property of social relations in all societies, and not the by-product of measurement errors or errors of definition. Measurement problems may affect the size and pattern of differences but do not cast doubt on their existence.”

Whereas nowadays, socioeconomic health differences are taken as a universal and persistent phenomenon, some decades ago there were different opinions about the possible chances for improvement: “...there is every indication that in modern Western countries, the relationship between social class and the prevalence of illness is certainly decreasing and most probably no longer exists” (Kadushin 1966:410).

But the reduction of health inequalities has not taken place and is still an aim of social policy. The World Health Organization (WHO) has proposed the “health for all” target for countries in the European region. “By the year 2000, the differences in health status between countries and between groups within countries should be reduced by at least 25%, by improving the level of health of disadvantaged nations and groups” (WHO 1985). While the health status of many social groups in many societies has definitely improved, a process that can hardly be expressed in percentages, international and social health differences did not decline. But there are considerable variations in health inequalities between time periods and places (Mackenbach et al. 1999; Kaplan 2001:140).

The beginning of social epidemiology, which addresses social differences in health, goes back to Friedrich Engels’ descriptions of the British labor class in 1845 (Engels 1987). Since then, systematic health differences between social groups have been described repeatedly. These differences exist between the social position one inhabits

and the average health status of social groups. Logically, we are dealing with social groups – not health groups – showing such differences: the intra-group health variation is higher than the inter-group variation (Vincent 1995:19; Nichols 2001:134; Knesebeck et al. 2003:19).

Concerning prominent risk factors and diseases responsible for social health differences, there is a historically dynamic social distribution of these factors. Smoking and obesity were “privileges” of the rich until the early 20th century. After this, the overall living standard increasingly made it possible for the poor to adopt this behavior (Davey Smith et al. 1994:442). Since then, lower class people are at higher risk of smoking, drinking and obesity (Kunst 1997:140). Accordingly, coronary heart disease and stroke are not a businessman’s disease anymore but rather are much more common in lower classes (Wilkinson 1994:66). It is doubted that these diseases have ever been a businessmen’s disease. An indication is that before 1950 cardiovascular mortality was higher for males in high social status groups and lower for women with a higher status. After 1950 this pattern reversed, but only for men, with the result that today higher status means less risk for men and women (Lauderdale 2001). Many diseases of affluence reversed their social distribution: heart disease, stroke, hypertension, obesity, and duodenal ulcers are more common among poor people than among rich (Wilkinson 1997:593).

Even if the association between social status and risk factors like smoking has reversed, lower status groups have always had worse health than upper status groups (Davey Smith et al. 1994:443). This means that smoking was less dangerous for the rich than for the poor and could not outbalance other health threats that lower class persons experience. In the last two centuries, the major diseases and causes of death have changed from infectious diseases to chronic diseases. It is remarkable that the social health gradient is the same after this total reversal of causes and after a general mortality decline due to improving living conditions (Vågerö and Illsley 1995:234).

The health gradient that exists between social groups can be observed throughout the social gradient: even rich persons are less healthy than very rich persons (Wilkinson 1997:593). But the impact of education, income, and wealth is also non-linear, i.e., in upper classes the positive impact of certain additional resources is lower than in lower classes (Smith and Kington 1997:115ff; Goldman 2001b; Smith 1999; Mackenbach et al. 2005). This can be explained by the concept of a ceiling effect: it is very difficult to improve health further than to a healthy status, and additionally it would be difficult to measure this further improvement.

Researchers wondered if the social health gradient is continuous or if there are important thresholds (Hummer et al. 1998b:558): it seems to be continuous (Goldman 2001b), so the gradient is important at every level but not to the same degree at every level (Lynch and Kaplan 2000:22ff). It is only in a few studies that the rich no longer exhibit this gradient (Siegrist 2001:363).

Socioeconomic health differences change over time: most evidence speaks for an increasing health gap in the USA (i44 Marmot 1999:19; i69 Smith 1999:158) in spite of rising income and societal efforts to act against social differences in health. A widening gap can also be found for the UK (Lampert 2000:161; Chandola et al. 2003a:2063). Interestingly, health differences are not consistently smaller in egalitarian countries (Kunst 1997:142; Valkonen 2001:8826). Adda et al. (2003) find comparable socioeconomic health differences in the UK, Sweden and the USA (Adda et al. 2003:59).²⁰

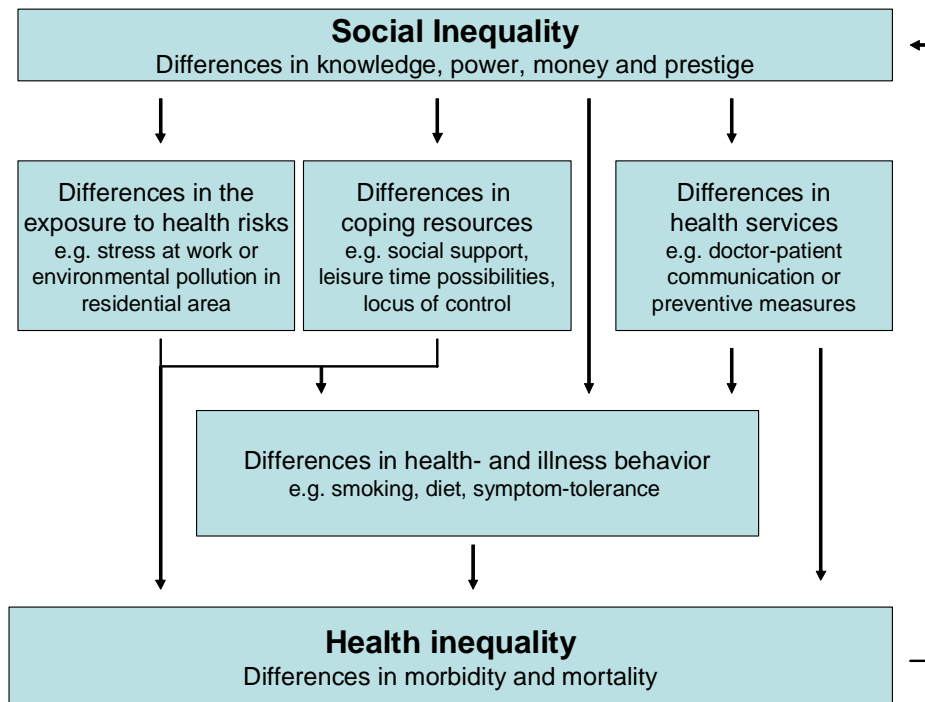
Today risk factors like smoking, exercise and the Body-Mass-Index (BMI) are more correlated with education than they were thirty years ago because disparities in health knowledge have increased (Lauderdale 2001). But it bears repeating that there are more health differences *within* socioeconomic groups than *between* groups. Thus even if income or other resources were equally distributed, there would be large health differences between individuals.

When increasing health differences between social groups are found over time, what has to be considered is that the share of persons in lower groups may have decreased so that actually fewer persons are affected by worse health or higher mortality levels (Marmot 1994:26). Reducing social inequality in health is not a zero-sum game where a health improvement for the lower status groups would result in a loss for the upper classes. Instead, the whole society would benefit from reduced inequality through reduced health care costs, increased overall well-being, and higher productivity (Glyn and Miliband 1994; Davey Smith 1996:988).

Figure 4.1 suggests some causal links between social inequality and social inequality in health. Since health is an approximation of mortality, this figure also serves as a causality scheme for the discussion of social mortality differences found in the following section, wherein I will discuss this causality in much greater detail.

²⁰ See Section 4. 2. 1 and Section 5. 3 for international comparisons.

Figure 4.1: Causal pathway from social inequality to health inequality



Source: Elkeles and Mielck 1997:140

4.2 Socioeconomic differences in mortality

Socioeconomic mortality differences are the central research topic of this dissertation. Before I present findings from the literature about such differences, it is worth listing the reasons why research on mortality differences is important. It can help to:

1. Identify disadvantaged groups and improve their health (Arber and Ginn 1993:229)
2. Find causes of diseases and changes in mortality.
3. Extend life expectancy by identifying beneficial conditions for longevity (for all three points, see Martelin 1996:112).

Health is the number one value and the single most important factor predicting life satisfaction (Arber and Ginn 1993:33) but to be alive is even more important, so inequality in mortality is as such a very important topic of study. A simple but noteworthy aspect is that since all persons have to die, research about inequalities can only look at postponement of death or at compression of morbidity (House et al. 1994:214).

Lower socioeconomic status groups have a higher relative mortality level (Klein 1993b; Mayer and Wagner 1996:268). Poor groups of people have generally two to three times higher death rates than rich ones (Wilkinson 2001:31). The difference in life expectancy for Dutch men between the highest and lowest educational groups is four years (Stronks 1997:3). In Germany, men in the lowest income quartile have a life expectancy that is about 6 years shorter than life expectancy of men in the highest quartile, while for women this figure is about four years (Reil-Held 2000:1).²¹ In the 1980s, white men in the USA with a family income lower than \$10,000 had a life expectancy of 6.6 years lower than those with an income higher than \$25,000 (Smith 1999:147). But it is not necessary to compare extreme income groups to find these differences. Muenning et al. (2005:2022) show that the bottom 80 percent of adult income earners have a life expectancy 4.3 years lower compared to the top 20 percent of income-earners. Expressed in health adjusted life years, this difference is 5.8 years (ibid.)

Within-country differences are at times much higher than international differences, e.g., the male mortality rate of those under age 65 is higher in Harlem, New York, than it is in Bangladesh (McCord and Freeman 1990). The socioeconomic mortality gradient exists at all levels of social status. But just as for socioeconomic differences in health, there is evidence that the relationship between socioeconomic status and mortality is non-linear (Backlund et al. 1996; Wilkinson 2001:31). At the higher end of the income distribution, an additional amount of income lowers mortality less than at the lower end of the income scale. But there are still advantages even at the higher end of the social scale (Liang et al. 2002:304).

Reflecting the “health for all” target of the WHO cited above, Valkonen et al. (1993:70) identify a level objective and a distribution objective concerning mortality:

1. Mortality should decline particularly for those causes of death and age groups in which Finland [or any other country] has lagged behind other countries with a similar level of development (‘level objective’).
2. Socio-economic mortality differences should shrink, which requires a lowering of mortality faster than average among less fortunate groups (‘distribution objective’).

²¹ For an overview of studies on social mortality differences in Germany, see Schepers and Wagner (1989), Mielck and Helmert (1994) and Becker (1998). Additionally, there are more recent studies by Klein (1999) and Klein and Unger (2001) and Unger (2003). The latter two also offer a comparison between the USA and Germany.

A fundamental assumption for research into social mortality differences is that these differences really are social and not biological (Hummer et al. 1998b:556). This means that, although it cannot be excluded that mortality differences between social groups may have a biological basis, I assume that these differences develop due to social mechanisms. Research on socioeconomic status and health and mortality is also conducted to rule out other explanations (Oakes and Rossi 2003:770), by showing concrete causal pathways where the mechanisms that produce different health and mortality levels are explained by social variables. From a sociological point of view, such a concept of social difference implies that these differences are contingent. That is, in principle they could be brought about by social change, although these social differences are observed in all societies. This idea should not lead to the expectation that individual differences in health and mortality would decline to zero if, theoretically, all social inequality would be abolished. The analysis of socioeconomic mortality differences should bear in mind that, “in a world of genetic diversity there is no presumption that under ideal conditions, heterogeneity as we have defined it would disappear” (Hertzman et al. 1994:68). The term heterogeneity here is more neutral than inequality or inequity and it especially recognizes biological diversity that is not socially determined.

When systematic differences between social groups are juxtaposed against individual diversity, it is important to note that individual differences cannot explain group differences (Marmot 1999:21). The fact that intra-group differences are larger than inter-group differences, as mentioned earlier, reminds us not to ascribe all differences to social causes and backgrounds. Socioeconomic status does not explain much variance in morbidity or mortality (Mayer and Wagner 1996:269) but socioeconomic status is a very strong predictor for mortality – maybe the strongest after age and sex. Socioeconomic mortality differentials are larger than differences between other subpopulations defined by region, location (rural-urban), or marital status (Valkonen 2001:8825).

The analysis of social differences in mortality reveals that general shifts or improvements, e.g., the amazing gains in life expectancy or the possible compression of morbidity, do not happen uniformly for all members of society. Under conditions of massive social inequality it is possible that morbidity compression or the postponement of health decline and death is only realized for higher status groups (House et al. 1994:214).

4. 2. 1 International comparison

Socioeconomic mortality differentials are smallest in the Netherlands, Denmark, Norway and Sweden. The United Kingdom lies somewhere in between these, whereas large differences exist in France, Italy, USA, and Finland. In spite of its record high level of social inequality, the USA does not consistently have higher differences than other countries that are more equal (Kunst 1997:61ff). In the 1980s the USA showed the same level of differences in mortality as Scandinavia (Kunst 1997:138, 211ff). It is plausible that socioeconomic mortality differences reflect differences in social position and thus social differences in mortality can be taken as an indicator for social inequality (Valkonen 1996:64). Comparing socioeconomic inequalities in health in ten European countries, Kunst et al. (2005) find that Scandinavian countries are more equitable between 1980 and 1990 than other countries. They conclude that these more egalitarian welfare states were, “able to buffer many of the adverse effects of economic crisis on the health of disadvantaged groups” (ibid.:295). But the U.S. example shows that the link between social inequality and socioeconomic mortality differences is not very tight. In the case of the USA, Kunst (1997:204) hypothesizes that the “spirit of classlessness” outbalances some of the actual inequality.

Some authors have tried to find evidence for the effect of egalitarian policy on health and mortality differences. Many results support this idea but some findings where more egalitarian countries do not show smaller differences in health and mortality suggest that an interpretation in the above manner is not easy. A less consistent social health pattern is found in the USA than in Germany (Knesebeck et al. 2003:1649), although there are only very few studies about social differences in health and mortality in Germany because of a lack of appropriate data. But from a European perspective it can be stated that in spite of a more pronounced egalitarianism in northern Europe, mortality differences in these countries are not consistently smaller than in other countries (Kunst 1997:125). At least this shows that practicing egalitarian policy cannot entirely remove the problem of socioeconomic mortality differentials (Kunst 1997:142). In fact, after the Medicare program was implemented in the USA in 1965, and after the National Health Service was started in Great Britain in 1946, mortality differences even increased (Pamuk 1985; Preston and Elo 1995:491).

“Socioeconomic differences in mortality in countries with more egalitarian policies are not small from an international perspective. Nor are they small from

an historical perspective: since the 1960s, socioeconomic differences in mortality have increased in northern Europe as well as in the United States [...] The findings do *not* imply that egalitarian socioeconomic policies cannot help to reduce socioeconomic differences in mortality. It is more likely that mortality differences in the Nordic countries would have been larger in the absence of egalitarian policies, or that mortality differences in the United States would have been smaller if income inequalities in this country would have been as small as in the Nordic countries.” (Kunst 1997:142)

Research findings showing socioeconomic differences in mortality in the USA have already been mentioned and cited several times because the USA is one of the most studied countries and results can be found very easily in the literature (see Chapter 5). More scarce are results from Denmark, and therefore I will present such results in the following part. Comparisons on the order of magnitude of mortality differences between countries are difficult because usually there are no two datasets that are exactly comparable; at least this is the case for comparisons between the USA and Europe. An attempt to make a comparison of old age health differences between Germany and the USA was carried out by Knesebeck et al. in 2003. They find steeper social health differences in Germany than in the USA.

Large-scale statistics on socioeconomic mortality differences in Denmark that use register data of the whole population are often based on a classification system that uses occupational status as criteria. For example the Danish Health Ministry uses the following groups: self-employed in agriculture, other self-employed, helping relatives in agriculture, other helping relatives, white-collar, skilled blue-collar, unskilled blue-collar and an undefined group of economically active persons (Sundhedsministeriet 1994c:33). Mortality differences between these groups are shown in the following table.

Table 4.1: Mortality differences between occupational groups in Denmark 1986-1990 (all occupational groups=1)

	Men		Women	
	Relative Mortality	CI (95%)	Relative Mortality	CI (95%)
Self-employed, agriculture	0.71	0.68-0.74	0.94	0.77-1.15
Self-employed, other	1.06	1.03-1.09	1.15	1.07-1.24
Helpers, agriculture	-	-	0.70	0.64-0.77
Helpers, other	-	-	1.01	0.93-1.10
Lower white-collar	0.81	0.78-0.84	0.93	0.84-1.03
Middle white-collar	0.89	0.86-0.92	0.90	0.86-0.95
Upper white-collar	1.02	0.99-1.05	0.97	0.94-1.00
Skilled blue-collar	0.97	0.94-1.00	0.98	0.82-1.17
Unskilled blue-collar	1.22	1.19-1.25	1.05	1.02-1.08
Undefined group	2.50	2.35-2.65	1.24	1.17-1.32

Source: Sundhedsministeriet 1994c:39

We see more or less the expected pattern that higher occupational status groups have lower mortality. The exceptions are, first, that self-employed men and women that do not work in agriculture have a surprisingly high mortality which can be due to the fact that this group is very heterogeneous and includes not only wealthy employers and industrialists but also small, self-employed persons with only a few, if any, employees. Second, the order of the subgroups within the white collar employees is not always according to the assumed social status of workers. Concerning the comparison to other European countries, Denmark has the lowest manual/non-manual mortality ratio in Europe (Kunst et al. 2000).²²

4. 2. 2 Trend over time

In general, mortality in upper and lower socioeconomic groups has decreased over time. Since mortality fell more in higher groups (Valkonen 1996:54), mortality disadvantage of lower groups has increased in spite of their absolutely declining mortality level (Wilkinson 1994:71). This results in increasing relative differences but also in stable or decreasing absolute differences. There are some findings showing a more dramatic development in lower status groups and thereby also suggesting increasing absolute differences: Barnett et al. (1999) find that mortality from coronary heart disease among black persons' mortality did not decline at all between 1984 and 1993, but instead increased in lower status groups, except for in the highest status group. Elo and Drevenstedt (2004) mention that the difference in life expectancy between black and white persons in the USA increased with substantial fluctuations from 6.7 years in 1960 to 8.2 years in 1995. The authors point out that in the mid-1980s black male life expectancy declined, which is, "highly unusual in a developed country at the end of the 20th century" (Elo and Drevenstedt 2004:269). In the UK all causes of mortality for persons aged 15 to 44 in the second and third lowest income quintiles did not decline between 1981 and 1991. Mortality did increase in the lowest income quartile (Geyer 1997:37). But besides this exceptional mortality increase which implies increasing

²² More results based on finer and broader occupational categories including the unemployed and the differentiation of different causes of death can be found in the publications by the Danish Health Ministry (Sundhedsministeriet 1994c), in Andersen and Laursen (1998) and in Andersen et al. (2005). Since the focus on occupational groups is not ideal for studying elderly persons, these results will not be further discussed here. A study on socioeconomic differences in life expectancy and health expectancy that uses educational groups based on survey data is Brønnum-Hansen et al. (2004). The present dissertation is the most comprehensive analysis of socioeconomic differences in old age mortality in Denmark in terms of number of variables and size of the study population.

absolute mortality differences, the increasing disparity due to the different pace of improvement is a common finding. Pappas et al. (1993:103) show increasing mortality differences between income and educational groups from ages 25 to 64 in the USA between 1960 and 1986 for both sexes. However, Preston and Elo (1995:486) only find male educational mortality differences increasing between 1960 and 1980 and narrowing differences for women.

Other studies confirm increasing socioeconomic mortality differences in the USA (Lynch 2003), and slightly increasing differences for men in Denmark from 1970 to 1990, differently for different causes of death (Sundhedsministeriet 1994c:43ff). In many different countries the same trend towards increasing differences has been found (Pamuk 1985; Marmot and McDowall 1986; Elkeles and Mielck 1997; Lauderdale 2001:552; Goldman 2001a).²³ Valkonen (2001:8826) concludes that relative differences in mortality increased during the 1980s in all countries where data are available (e.g., United States, Nordic countries, and France).

Factors that may contribute to these increasing differences are the following:

1. Davey Smith et al. (2001:114) showed that important causes of death are also those that show a large class gradient. It is possible that socioeconomic mortality differences increased because causes of death that are more unequally distributed got more important over time relative to other causes (Davey Smith et al. 2001:114). Additionally, the most important cause of death contributing to the general mortality decrease is cardiovascular disease. This cause of death is at the same time the cause that contributed most to the increase of socioeconomic mortality differences (Feldman et al. 1989). During this trend, upper classes benefited more because they were faster in adopting recommended health behavior, including diet and lifestyle choices, as well as in getting better medical treatment (Valkonen et al. 1993:71; Preston and Elo 1995:490; Valkonen 2001:8826).
2. Biological determinism gets weaker relative to social differentiation, which then dominates and gets more impact on mortality relative to biological influences.
3. As a supplement to argument number two, it can be argued that in general increasing social inequality in many countries and differential access to health care causes mortality differentials to increase (Pappas 1993; Lynch 2003:31). While this may well

²³ More literature can be found in Kunst (1997:142) and Lampert (2000:161).

be true, it still does not explain why mortality differentials also increased in countries where social inequality decreased, e.g., in Finland (Valkonen 2001:8826).

The link between increasing social inequality and increasing mortality differentials is, although not proven, at least plausible. What is more surprising to note is the increase of socioeconomic mortality differences in a period of increasing levels of wealth, economic growth, and improvements in medicine (Kunst 1997:9f).

A puzzling aspect of the increase of mortality differences is that women have been less affected by the widening social mortality gradient. At least for the USA, two explanations are offered by Preston and Elo (1995:490): first, during the last decades the female labor participation rate increased and second, more women than men are entitled to get payments from Medicaid or other benefits.

4. 2. 3 Gender differences

In spite of the higher life expectancy of women, they have on average worse health than men, both in terms of self-rated health and functional status (Verbrugge 1984 and 1989; Arber and Ginn 1993:37; Christensen 2001:102; Liang et al. 2002). Surprisingly, some research findings suggest that though women have the same probability of contracting illnesses, their overall health status is worse than men's (Klein 1999:452). This would imply that they recover less easily from diseases than men do. Research findings differ concerning the social health gradient for men versus women: some studies reveal slightly more pronounced social differences for men (Liang et al. 2002, Goldman 2001a) whereas others show the same gradient (Arber and Ginn 1993:33). Klein (1999:461) showed that in the lowest social status group there are no gender differences in mortality.

A group that has both a higher life expectancy and a worse health status than another group is remarkable and counterintuitive because in comparisons of social and many other conceivable groups (e.g. region, biological differences, etc.), the disadvantaged group normally has both worse health and higher mortality. Gender differences concerning health and mortality do not fit with the simple logic of advantage or disadvantage which predicts that a group always has both higher morbidity and higher mortality.

It is not yet known why a health disadvantage for women exists. It may be due to biological differences, i.e., genetically, men and women have different physical constitutions and health and mortality trajectories. An explanation for a portion of the differences is that women have a different self-assessment regarding their body. They perceive more problems, have more sorrows and are more prone to depression (Delbès and Gaymu 2002:900ff). Women understand their bodies better, admit to having illnesses more readily, and rank their health worse than men in investigations, and they also allow more treatments (Oakes and Rossi 2003:103) and generally exhibit better health behavior (Luy and Di Giulio 2005). If such differences in health behavior play a role, it means that they are not successful in terms of health improvement but rather in terms of a longer life. The shortest notion for these gender differences would be that “women suffer, men die” and this is so because of an interesting and still unexplained interplay of physiological, mental, and behavioral differences.

One explanation that could integrate the disparate findings of better health but higher mortality for men is mortality selection. If men have higher mortality throughout their lives, maybe because of a different physical constitution and a more stressful role in society (Klein 1999), it is possible that the average health status of the surviving men is better than that of women because the unhealthy men already died.

Concerning mortality, a large body of literature shows a weaker socioeconomic mortality gradient for women.²⁴ If, due to data limitations, women are classified according to their husbands' occupations, they show steeper gradients than if their own occupational classifications are used (Moser et al. 1990). Arber and Ginn (1993) do not find such measurement differences above age 65. Educational mortality differences are a lot larger for men than for women. This is mainly because men receive greater rewards from education in terms of money. That is, if money is controlled for, both sexes have the same educational mortality gradient (McDonough et al. 1999:20).

More than two decades ago, Goldthorpe used the husband's class to categorize the women they were married to (Goldthorpe 1983:468). He argued that men have a “directly determined position within the class structure” because they are involved in the labor market more intensely and for a longer period. A classification problem does not occur in my analysis because I have individual information for men and women. If only the unmarried are analyzed, the social mortality gradient for men and women is the

²⁴ Pappas 1993; Koskinen and Martelin 1994; Martelin 1994; Backlund et al. 1996; Elo and Preston 1996; Mackenbach et al. 1999; Goldman 2001; Valkonen 2001; Liang et al. 2002.

same (Bassuk et al. 2002:531). This means that one's marital status probably has an effect on the impact of education or income on mortality. It seems that single women and single men are exposed to similar risks because they have similar lifestyles whereas married women live very differently from single persons.

Besides this, there are the following explanations for a steeper gradient for men: they die more of causes that are more unequally distributed (Valkonen et al. 1993:72; Mackenbach et al. 1999:1804; Valkonen 2001:8826). Men react more dangerously to stress and other challenges. The general tendency for them is to drink alcohol whereas women in such situations tend to eat more and get obese, which is the less harmful health risk (Mackenbach et al. 1999:1804). Of course there is also an interplay between class and gender because women on average have lower status than men and among women there is less social inequality. Because of these differences in the level and distribution of men's versus women's social status it is difficult to say whether female mortality really depends less on socioeconomic status (Klein 1993c).

4.3 Mortality versus morbidity

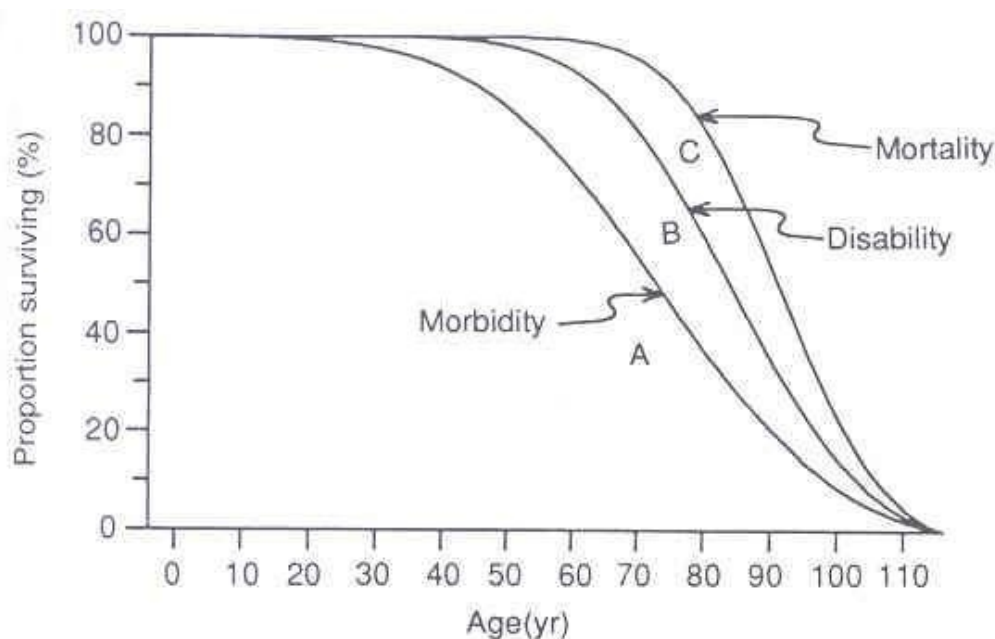
In the previous sections morbidity and mortality were discussed in separate sections if this was possible. They are two distinct phenomenon and so many studies, theories, and empirical findings address either mortality or morbidity exclusively. A severing of mortality from morbidity is possible, but of course both belong to the same process where in most cases declining health precedes death. Except for accidents and homicides a person dies from the consequences of an illness or due to a physical failure. The same factors and maybe also the same pathway may lead from socioeconomic status to bad health and from socioeconomic status to death (Kåreholt 2000:3). For example, Backlund et al. (1996:13) show that income has the same association with mortality and morbidity.

In the following I will justify why my main research focus is on mortality and describe implications of this decision: mortality is a reliable picture of public health (Valkonen et al. 1993:12ff). It is also the most objective health measure (Markides and Black 1996:165; Kåreholt 2000:2), and this objectivity remains so across classes (Ferraro and Farmer 1999). It is a measure for social and economic well-being and mortality differences are a fundamental indicator of social inequality (Preston and Elo 1995:476). Aiach (2000:84) describes two important features of mortality as a health indicator:

“Mortality has been the basic parameter for the study of social inequality in health. This is for two reasons, a practical and a theoretical one. First, different from a disease, death has to be declared officially to the administration, which facilitates comparative studies. Second, death can be understood as the end of a process in which all elements of social and mental life interact. Therefore differences in life-span are the synthetic expression of all social inequalities between hierarchical social groups. The use of this indicator is not a stopgap but corresponds well to a strong theoretical exigency.”

In spite of the tight linkage between health and mortality, one cannot necessarily extrapolate from health to mortality, and maybe not even vice versa (van Doorslaer and Gerdtham 2003). Ferraro and Farmer (1996:324) present the surprising finding that having a chronic illness can be associated with lower mortality if controlling for other health indicators. Several studies have shown that in Denmark socioeconomic differences in health expectancy are larger than in life expectancy while the opposite seems to be true for France and Finland (Mackenbach 1997; Brønnum-Hansen 2000:194). Generally, socioeconomic inequality in health is mirrored in socioeconomic inequality in mortality, but some morbidity is not translated to mortality and vice versa (van Doorslaer and Gerdtham 2003). To measure only mortality means to neglect the burden of bad health (Smith and Kington 1997:122). The following illustration, Figure 4.2, is one possible representation of the relationship between morbidity, disability, and mortality.

Figure 4.2: Survival curves for different transitions in the aging process



Source: Jette 1996:105

Figure 4.2 shows three survival curves for each of the three events: falling ill, becoming disabled, and death. All curves start at 100 percent of the population and describe the decline in the proportion of a given status (healthy, not disabled, alive). The areas separated by the curves represent (from left to right) the status of good health (A), poor health (B) and disability (C). The probability is on the Y-axis, so at age x the probability to be in good health is y , namely the value on the Y-axis of the morbidity curve. If lower mortality just means a postponement of death, i.e. a shift of the survival curve for the event of death to the right, there is more morbidity. If mortality falls because people are healthier, area A increases, there is less morbidity, and probably all curves, including the curve for mortality, shift to the right (Crimmins et al. 1994:160, see Section 1.2 about compression of morbidity). In conclusion, mortality is a good health measure, but it depends on the compression argument, namely on the relative shape of the curves in Figure 4.2. Finally, the measurement of the transition from life to death as a single event can never fully reflect a trajectory, namely the complex process of declining health.

4.4 Causality from socioeconomic status to health and mortality

What is a cause for a disease? “For an exposure to be a cause, it must be true for at least one exposed, that he or she would not get the disease in question at the time he or she did, had he or she not been exposed” (Olsen 2003:86). In a situation where an event is caused by many factors simultaneously, a cause may only be sufficient given that all other causes are present. Causes are not globally sufficient or necessary, but apply only to a specific situation. It follows that a prediction of a certain health outcome or a prediction of death is almost impossible, but only probabilistic. Only this is certain: that the event had causes and if all these causes would coincide again, the event would happen again. All factors together are deterministic, but this is a theoretical situation since in almost all situations not all causes are known (ibid.)

There are proximal causes which lead to the disease, and distal causes which cause exposures and determinants (Olsen 2003:88). Identifying social groups between which mortality differentials are high shows that these groups are different in a way that makes a difference for mortality. It does not mean that the parameter used to differentiate between the groups is really causal for mortality. The parameter may be a risk indicator

and not a risk factor (Müller 1993:5f), and even a risk factor does not necessarily provoke a disease or death.

To identify causality, three requirements need to be considered: 1. causality includes a specific chronological order, i.e., the cause and effect cannot be contemporaneous (Hertzman et al. 1994:74), 2. explanatory power and 3. invariance of the relationship over time (Hoover 2003:121). In principle, the first point is simple for an analysis of mortality because the event of death always happens after the cause. But concerning causes for health, the availability of longitudinal data and the possibility of revealing associations does not mean that causality can be directly observed (Campbell and Alwin 1996:39). Causes and indicators can both have latency (Hertzman et al. 1994:83). To obtain plausible assumptions about causality, it is possible to look at many possible factors and compare their impact on mortality. If a plausible pathway is found that explains mortality differences in different settings, in different periods and in the presence of different choices of covariates in a model, certain factors can be accepted as causes for mortality. By definition, social differences in health and mortality can be found by comparing social groups, but causality in a strict sense can only be assumed.

The following discussion of causal pathways to mortality starts with the most proximal cause for mortality that may be available in a data set: the cause of death. Then a classification of other causes is proposed, and finally a concept of distal causes, the fundamental causes, will be discussed.

4. 4. 1 Cause of death

When a person dies, one or several causes of death are usually recorded by a medical doctor on the death certificate. These causes are classified according to the International Classification of Diseases (ICD). This classificatory system is changed and adjusted every few years or so. In the Danish registers ICD-8 was used until 1995, and thereafter ICD-10 was applied.

The analysis of socioeconomic mortality differences can profit from taking the causes of death into consideration because they show specific risk factors that contribute differently to socioeconomic differences for all causes of mortality (Kunst 1997:127). “Differently” means that there are socioeconomic gradients of different magnitude for each cause of death. In middle age, the causes of death with the largest differences e.g.,

for manual versus non-manual workers are respiratory diseases, accidents and violence (Valkonen 1996:61).

We cannot only look at the social differences in the mortality of one cause, but also at the relative contribution of that cause to social mortality differences. A rare cause with high inequality can have the same impact on the overall socioeconomic mortality differences as a common cause with less inequality (Valkonen 1996:62). Elo and Drevenstedt (2004) analyze the contribution of different causes of death on mortality differences between black and white persons in the USA. They find that HIV/AIDS and homicide are the largest contributors to mortality differences between black and white people.

Leading causes of death for the elderly are cancer, heart disease, stroke and accidents. The contribution of cardiovascular disease (CVD) has declined much, but it is still the most important cause of death (Jeune 2002:79). Coronary heart disease (CHD) is the leading cause of death in the USA since 1921, but mortality of this cause declined since 1961 (Lauderdale 2001:559). Generally, it has been proposed that the most important causes of death also have the sharpest social gradient (Davey Smith et al. 2001:115). Deaths from alcohol and tobacco are also distributed especially unequally (Stolpe 1997). Causes of death associated with drinking are cirrhosis and alcoholism; cancers of the mouth, esophagus, larynx and the liver; breast cancer for women; and injuries and external deaths for men (Thun et al. 1997). The causes of death directly associated with smoking are mainly lung cancer and other diseases of the respiratory system. One special finding for lung cancer is that this cause of death shows more social differentiation than smoking behavior does, which is an indication that lung cancer cannot entirely be explained by smoking as such, but also by other social differences, e.g., diagnosis and treatment (Davey Smith et al. 2001:115).

Sometimes the ICD codes are taken as an indicator for certain living conditions or health behavior. This may be plausible in some cases, e.g., lung cancer is much more common among smokers. But logically, such a procedure tries to extrapolate from the effect to the cause and is therefore questionable (Valkonen and Martelin 1999:220).

The impact of a single cause of death is always relative because the causes interact (Myers 1996:99). If a single cause of death could be eliminated, the impact of other causes would increase because every person will eventually die of some cause.

Socioeconomic mortality differences, i.e., higher death rates for lower classes, are evident for all causes of death except for breast cancer for women and cancer of the

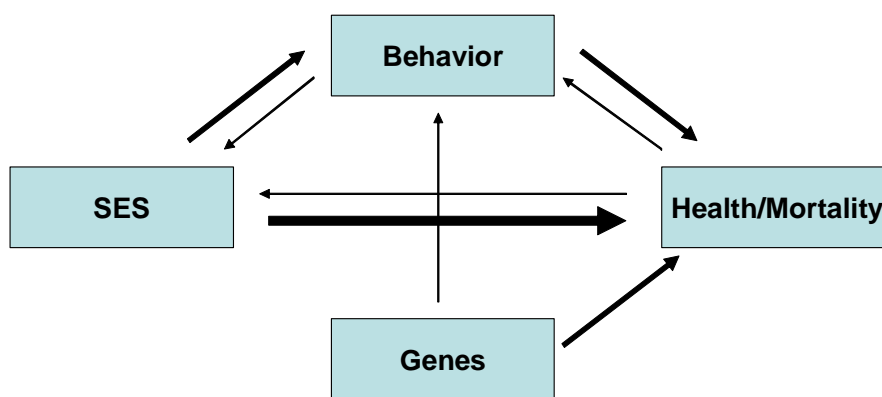
intestines and rectum for men that sometimes are found to be more common in upper classes (Valkonen 1996:61). Socioeconomic mortality differences are usually greater for causes amenable to medicine (Lauderdale 2001:559). Kennedy et al. (1996) find that, although the contribution of treatable causes of death to overall mortality is rather small, mortality differences between income groups were larger for treatable causes of death. Causes of death can be complicated by data problems. In Germany an estimate of 40 percent of all death certificates are wrong because the person who fills out the death certificate is not able or willing to take the appropriate measures to find out the correct cause of death (Süddeutsche Zeitung 2./3.10.2001). In situations where up to three causes of death per death are recorded and available in a dataset, the question arises of whether it is advisable and possible to use this additional information to disentangle co-morbidity and gain insight beyond a first cause of death. In many cases the first cause is a simple overall cause, e.g., cardiovascular disease that is assumed for many old persons but is not the exact description of the physical condition leading to death. Despite the fact that lower class persons have on average a higher co-morbidity, these further causes of death have deliberately been neglected by some researchers (e.g., Hayward et al. 1998:199), and have been shown not to be important for the analysis of the socioeconomic mortality gradient (Kåreholt 2000:27).

4. 4. 2 Factors influencing health and mortality

How do we make a systematic and exhaustive list of factors that have an impact on health and mortality? We could say that death is the end of a process where all factors had an effect throughout a long period of the life course. Then this process would be very similar to what we call “life” and to take life as the process that leads to death is not very promising in analytical terms. Thus, for analytical research it is necessary to simplify the universe of possible factors to a limited number of risk factors, e.g., BMI, weight, smoking, alcohol, leisure time, physical activity, social support, marital status (Davey Smith et al. 2001:99). These factors may be the only available variables in a concrete data set or study, and of course the availability of data can constrain the choice of factors. Nevertheless there should be theoretical and empirical considerations that lead to such a choice. Before I discuss several factors in detail, the following figure, Figure 4.3, gives an overview of causality that focuses on the interplay between socioeconomic status, behavior, and genes. The problem of differentiating between

structural constraints and free choice has been discussed in Section 3.3 and the meaning of genetic differences for the study of social differences will be discussed below. This figure serves as a schematic orientation for the discussion of the most important factors in the following sections.

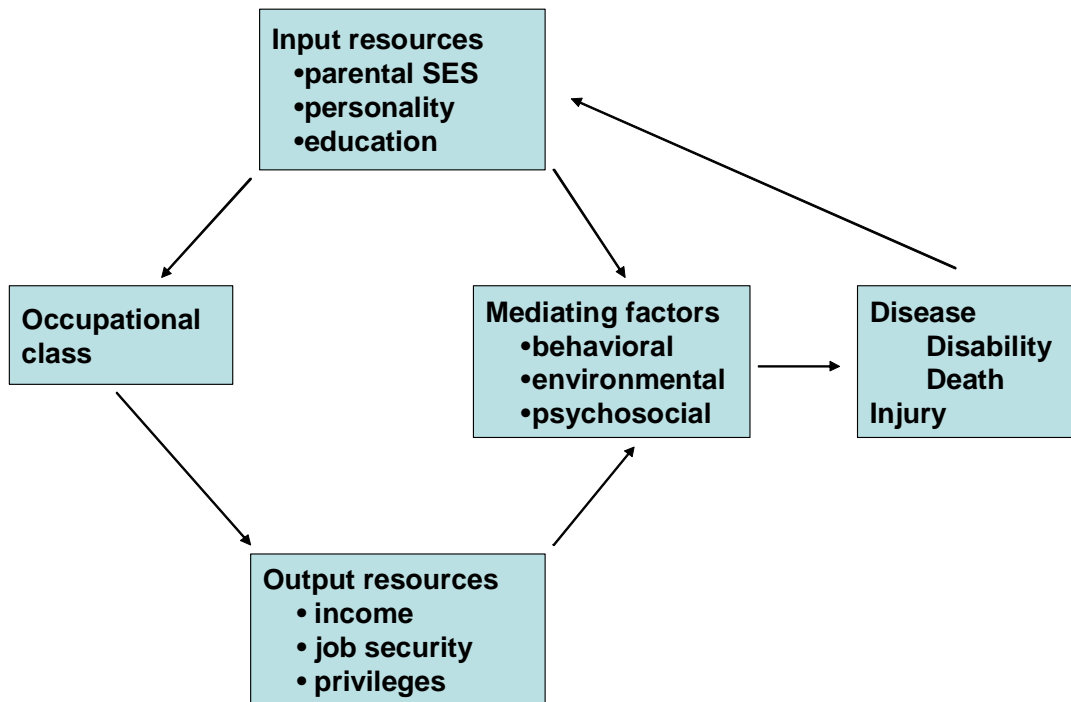
Figure 4.3: Causality between socioeconomic status (SES) and health/mortality



As this figure shows, I suggest that none of the possible causes go only in one direction. Except for genetic endowment, which is fixed, and death as an absorbing status, all factors can be the cause and the effect of other factors. Besides the main causality going from socioeconomic status to health, there is a side line that works via behavior. The small arrows also allow for “unconventional”, indirect effects of e.g., genes on social status via behavior or health. The reverse causality from health to socioeconomic status will be discussed in Section 4. 4. 7.

The following causal scheme proposed by Kunst et al. (1998a:478) is more detailed concerning socioeconomic status, differentiating between the resources of input and output. However, this causality scheme does not assume causality from behavioral and psychosocial factors to socioeconomic status.

Figure 4.4: The relative position of different socioeconomic variables to health



Source: Kunst et al. (1998a:478)

In the empirical part of this dissertation I have to accept a limited number of available variables. But in this theoretical part it is worth considering a broad range of different influences on mortality, even if they cannot be included in the empirical analysis. I suggest the following number of categories that help to classify and understand the variety of mortality predictors. The shortest of many possible lists of categories includes the following five categories. They will be described now, paying attention to empirical evidence as well as to theoretical problems.

1. Genetic constitution
2. Natural/physical environment
3. Structural and material conditions
4. Behavioral and cultural factors
5. Psychosocial circumstances

4.4.2.1 Genetic constitution

Studies revealing that life expectancies of monozygotic twins have a higher correlation than life expectancies of dizygotic twins clearly show that genes have an impact on mortality (McGue et al. 1993; Herskind et al. 1996). Monozygotic twins share the same genetic make-up and the same social background, at least in childhood, whereas dizygotic twins only share this social background (Lampert and Maas 2002:220). Their genomes are similar because of the common parents but not identical. Because of this difference comparative studies between monozygotic and dizygotic twins are able to estimate the relative contribution of genes and (social) environment. The results suggest that the variability of mortality after age 30 may be explained up to 25 percent or less by genes and to another 25 percent by factors that are fixed until the age of 30 years (Christensen and Vaupel 1996; Vaupel et al. 1998; Vaupel 1998; Vaupel 2000:42). This means that within the scope of socioeconomic mortality differences we do research on about 50 percent of the variability, maybe more if living conditions before age 30 belong partly to one's socioeconomic status, maybe less because not every external factor that influences mortality after age 30 depends on one's socioeconomic status²⁵.

In a simple (uncontrolled) analysis, the parents' age of death has an impact on mortality (biological hereditary), but controlling for the parent's education reduces this influence because parents also transfer a part of their social status to the children (social hereditary), which again is correlated with their life span (Klein 1995). Such interplay between social factors and genes is probably true for many determinants of mortality and complicates the identification of social versus genetic factors. Examples for such interactions are gender and race.

1. Gender or sex, basically a genetic and biological variable, has a major impact on how an individual comes under social influences. This term "gender" includes both biological sex and social roles and allows, in principle, for extreme cases where an individual changes its gender. Men suffer higher mortality than women, and a part of this increased risk is due to certain behaviors and roles. Different roles for men and women in society also imply that they come under qualitatively different mortality risks. Some studies find that for men, education, income and occupational prestige are important mortality predictors whereas for women, only income is of major importance (Bassuk et al. 2002:520). Others do not observe gender differences in the impact of

²⁵ The impact of genes and theories about the hereditary of the life span are discussed in Lampert (2000:164), Steinkamp (1993:115), and Jeune (2002:83).

education or income on mortality (McDonough et al. 1999:17). Lampert (2000:167) finds gender differences in working life and health behavior. As mentioned in Section 4.2.3 the social mortality gradient is higher for men.

2. Racial mortality differences also include genetic differences. Estimates suggest that racial mortality differences can be explained to more than 60 percent by social differences (Smith and Kington 1997:117). Racial mortality differences have been described such that being black in the USA means having a health status of a white person who is five years older (Menchik 1993:434).

It is difficult and ethically problematic to say that socioeconomic status also has a genetic background. But in my opinion it is plausible to assume that at least to some extent, genes also contribute to an individual's social status. Height and beauty, which both have a genetic component and a social meaning, may illustrate how in principle such a causal relationship between genes and the socioeconomic status may work. Health is another factor in a possible causation from genes to social status. But even if such pathways cannot be excluded, there is clear evidence that mortality differences caused by social factors independent of the genes, are much larger. Moreover, they are certainly large enough to be addressed by research and policy and are also large enough to rule out the assumption that social health differences represent a "natural" difference that cannot be changed.

4.4.2.2 Natural and physical environment

There are physical and chemical factors in the environment that influence mortality, e.g., a healthy climate or the existence of healthy food (Hertzman et al. 1994:76ff; Henke and Müller 2002). Maybe these factors can explain some exceptional cases where people in poor countries have a life expectancy that is not much lower or is even higher than in rich countries, e.g., Costa Rica with 76 years compared to the USA with 76.6 years at the end of the 1990s. But generally only a few of these natural and physical factors are not mediated by social factors. For most factors there is a social gradient in the use of or in the exposure to environmental conditions. Biological constraints interact with social behavior and social processes, e.g., with sexual or sanitary behavior in a region with harmful viruses and/or disease risks (Vincent 1995:19; Marmot 2000:349). However, it is important to point out that every description of a social mortality risk factor, inside or outside the human body, must take

into account that eventually death is the result of biological processes. Therefore, any causation must be biologically plausible (Marmot 1999:27).

Before looking at factors that can be understood as social in a strict sense, one logical distinction is important. For the obviously higher mortality risk for lower socioeconomic status groups, the question is whether a higher exposure to concrete risk factors is the reason for higher mortality or, conversely, whether these groups have a higher vulnerability and susceptibility which lead to a higher impact of health threats. The idea of a higher level of exposure would point to the macro level, with factors for higher susceptibility found on the micro level (Marmot 2000:360). House et al. (1994:221) claim that it is a different exposure level that results in socioeconomic mortality differences, but the impact of such exposures increase with age. Stronks (1997) and Hertzman et al. (1994:76ff) claim instead that it is different levels of susceptibility. House et al. (1994) and Adler (2001:59) say that education as an indicator for socioeconomic status influences both exposure and impact. This is plausible because it fits with the sociological understanding of social structure where in lower classes, not only is exposure higher, but also the resources for coping with it are scarce. Mediated by behavior and personality, this increases the impact of unhealthy exposures.

4.4.2.3 Structural and material conditions

The most important determinants of mortality are age and sex. Other important predictors are race, income, education and occupation. All these factors are principally different from each other (Oakes and Rossi 2003:275ff) because of their different interplay between social and biological elements. Material conditions like income and also occupation, which I treat as a material condition, are less connected to the biological world, whereas age and sex are to a large extent biologically determined. Nevertheless, sex and age are parameters of the social structure.

Only some of the structural conditions are material conditions because the social structure is also built of non-material differences, e.g., education. According to Bourdieu, social capital is also a structural factor because it defines a person's position in the social structure. But I treat it as a psychosocial factor and discuss it below. A category of "structural factors" is maybe too broad if all dimensions that also would be used for a definition of the social structure as such are included. Therefore, in the literature structural differences tend to be operationalized rather by objective and more

material measures which are easier to measure and represent the so-called objective living conditions. But this is a tendency rather than a sign that structural indicators are necessarily objective or material. That a strict classification of structural conditions is difficult can be seen by the fact that one of the subdimensions of Bourdieu's cultural capital (as such a non-material dimension) is 'objectified cultural capital', which means that it is materialized into objects. Its counterpart is incorporated cultural capital, i.e., education. The most important of the material living conditions are mentioned below.

Material conditions explain a large part of socioeconomic mortality differences (Schrijvers et al. 1999) and are possibly more important than behavioral factors (Kunst et al. 1999:203). Of course such relative statements are problematic because it is difficult to separate material from behavioral factors under the assumption that part of the behavior is caused by material factors and, to a less extent, also vice versa. If all structural dimensions together define the socioeconomic position of an individual, these factors describe the living conditions and influence the thinking and the health behavior as part of the lifestyle and thus influence health and mortality. In the following I will discuss three important factors that belong to the group of structural and material factors: income, education and medical care.

Income is widely used as a measure for material well-being (e.g. Adler et al. 1994; Davey Smith 1996; Adler 2001; Klein and Unger 2001). Its importance for health and mortality is based on the ability to buy healthy food, good housing in a safe environment, quality health care, medical treatment, and other goods that are directly or indirectly relevant to maintaining a good health status (Grundy and Holt 2001:895f; Lampert and Maas 2002:222). These factors can be called the direct consequences of financial status on health. Vincent (1995) describes two consequences of insufficient financial resources especially for the elderly: first, material deprivation and second, less social contact (Knesebeck et al. 2003:1643). Klein and Unger (2001) mention four points as being responsible for the income-mortality connection: 1. working conditions, 2. behavior, 3. material conditions, and 4. medical services. While all these factors may be helpful in explaining the income-mortality gradient, at least the factors of social contact and behavior are not direct impacts of material wealth on health. But it seems that money has a central role for many health relevant goods, services and also behaviors and social conditions. Thus, many detailed factors could be subsumed into a group of health relevant consequences of income even if they are not material factors in

a strict sense. Some of these factors have been used in other studies as independent predictors of mortality with their own theoretical justification, either because information on income was not available (e.g., in studies where housing conditions are measured instead), or because they allow to gain additional insight into the pathway from income to health (e.g., the access to health care).

Education is an example of a structural factor because it is used to define the individual position in the social structure. However, education is not material but rather psychosocial. According to Bourdieu it is incorporated cultural capital. Education is important for receiving knowledge about health risks and healthy behavior and in providing cognitive skills for dealing with complex information such as the association of behavior on one's personal health and the institutions of the health care system. Better education promotes less stress as well as better coping and preventive behavior (Hummer et al. 1998b:560; Kåreholt 2000:222).

The enormous increase of the average education level in the last century implies that people know more about health than before, something which may have contributed to the overall increase in life expectancy.

Lynch (2003:12) discusses the trends of educational mortality differences in the 20th century and suggests that money may have taken over the role of education in determining social mortality differences. The association between education and mortality can be largely explained by material factors (Menchik 1993:436), and by behavior that depends on material factors (Schrijvers et al. 1999). Davey Smith et al. (1998:158) suggest that education is associated with health and mortality via: 1. common background factors that influence both education and health (indirect selection), 2. health knowledge, and 3. income, living and working conditions and behavior. Higher education means higher income, inner qualities like self-efficacy, and better health behavior (Preston 1992:53; Ross and Wu 1995, 1996).

Medical care. An important consequence of material wealth is access to medical care. This access can be understood as a consequence of the individual material situation, but also as a result of the overall level of wealth in a society and the health care system. The latter factor may be more important if individual differences in wealth do not play a major role in a generous and comprehensive welfare system. We also have to differentiate between formal access to health care, i.e., the right to get help based on

legal regulations, and the actual use of and the response to health care. Even if the former would allow poor people to get the same services as rich people, the latter would still cause social differences because lower social groups sometimes ignore health care services and have problems understanding and following the advised treatments (Hertzman et al. 1994:76ff; Lampert 2000:164).

Statements about the importance of health care can be found in numerous epidemiological studies and also in economic research (Arber and Ginn 1993:34). For the USA, House et al. (1994:224) conclude that differential access is not very important for the elderly since Medicare provides comprehensive services (see also Goldman 2001a). This argument can be contrasted with Preston and Elo (1995), who say that Medicare is not of major importance because there is no change of health inequalities after 1965, the year when Medicare was implemented. Moreover, the trend in health inequality is worse for elderly to whom this program is dedicated. They conclude that access to health care is still socially different in spite of Medicare.

The Whitehall studies I and II in the UK showed that the socioeconomic health gradient is not due to access to health care (Smith 1999:158.) Other authors conclude that unequal access to health care is not crucial (Preston and Elo 1995:491). This is also true for old age (Knesebeck et al. 2003:1643, 1650). Other empirical findings concerning the overall importance of health care for social mortality gradients reveal only limited importance. According to Deaton and Paxson, medical care explains only 10 percent of the impact of income on mortality (Deaton and Paxson 2001:132), and Smith (1999:148) and Adda et al. (2003:59) suggest that access to health care does not explain health differences. Marmot (1994) argues that health care is not an important explanation because, first, the mortality improvement that was higher in upper classes is the result of a decline of non-amenable deaths and, second, because the argument that better health care services decrease socioeconomic health differences does not hold for cross-national comparisons.

Hurd et al. (2001:196) reject the impact of the socioeconomic status on mortality via differences in access to health care and argue that health care utilization is not important because the socioeconomic mortality gradient disappears when health is controlled for. This means that given a certain health status there are no social differences in mortality anymore. Thus, the health care, which should be mostly effective when people are ill, is not differently effective for different social groups.

Other authors go even further and claim that health care is not responsible for social health differences because health care is not very important for health at all. Inadequacies in health care account for only 10 percent of premature mortality, whereas health behavior and lifestyle wholly account for 50 percent (Adler 2001:59).

But there are other researchers who claim that the health care system and the use of health care are indeed important (Müller 1993:83; Elkeles and Mielck 1997:139ff). Medical improvements over the last decades may have contributed substantially to the decline in old age mortality and to the increase in life expectancy. Their role may be small but crucial in decreasing health disparities in the future (Lurie 2001:91).

To conclude, it is fair to say that general health care cannot outbalance other unequal forces and change the trend of persisting or increasing health inequalities. This is partly because the health care system is not concentrated on prevention so that the development of an illness is affected by factors other than health care (Adler 2001:59f). Once a disease is developed there is not much inequality left and it is too late to have a substantial impact on health inequalities.

4.4.2.4 Behavioral and cultural factors

From the outset it is worth discussing two problems for the separation of behavioral factors from material factors:

First, many factors that are material in principle also include a behavior. For example, food is material but individual habits concerning diet are behavioral (Klein and Unger 2001:97). I decided to treat this aspect as a behavior and I call it obesity, but it is also possible to put it in a different category of factors. The health care system is a material factor but the individual use of health services, which is a behavior, is just as important (Grundy and Holt 2001:895f). Here I decided to stress the material part and therefore the health care system is subsumed under “material factors”.

Second, material factors like income may influence behavior and therefore it is difficult to separate the impact of income from the impact of behavior. Stronks (1997:163) suggests that 30 to 40 percent of health differences are due to behavior, but also points out that this impact cannot be separated from living conditions because it is not a free choice (ibid.:168). Other estimates suggest that the identifiable health behavior explains only 25 percent of the impact of income on mortality (Deaton and Paxson 2001:132). The Whitehall II study showed that the socioeconomic health gradient is not due to

behavior (Smith 1999:158) because only a small part of the gradient could be explained by smoking, physical activity, blood pressure and cholesterol (Valkonen 1996:64).

These findings suggest that “the poor behave poorly” (Lynch et al. 1997) in a very comprehensive way and therefore including observable indicators for health behavior in a statistical model only slightly reduces the socioeconomic health and mortality gradients. Hertzman et al. (1994:78) mention another statistical aspect: since poor health behavior is not just a choice but mainly an outcome of socioeconomic status, not controlling for socioeconomic status can result in an overestimation of the influence of the behavior, e.g. smoking. In their very interesting book chapter, Hertzman et al. also implicitly allude to the question of whether one could interpret behavioral differences within a social group as free choice, whereas behavioral differences between social groups cannot be interpreted as free choice (Hertzman et al. 1994:77). This is a simple but plausible way to understand the problem of “choice under constraint” which was discussed in Section 3.3.

One can learn from Bourdieu’s elaboration of the relationship between structure on the one hand, and lifestyle and culture on the other, that both are connected (Bourdieu 1979; Vågerö and Illsley 1995:221). Emphasizing the structural origin of behavior is not equal to a purely materialistic or deterministic argument (Vågerö and Illsley 1995:221). Behavior corresponds to one’s individual position in the social structure, but to some extent there are situations where people can choose without constraints from their position or their habitus. Consequently, health status is never 100 percent predictable or determined by one’s social status. In all other situations where the behavior does depend on structural factors, the behavior may still be interesting and important in an empirical analysis but it is not an independent causal factor. Rather, it is the consequence of more fundamental causes. The opposite idea of health behavior being a result of free individual and rational choices becomes even less convincing when we look at the aggregated level and observe systematic differences in health behavior between social groups. Lower social groups almost always have worse health behavior and this is not just the sum of individual phenomenon, it is social structure (House et al. 1994).

The emphasis on a specific health behavior, an attitude, certain habits, and a habitus all being related to material resources but form a different level of health relevant differences characterizes the class approach and makes it different from the material deprivation approach (for a comparison and discussion see Arber and Ginn 1993:34).

The most common behavioral factors studied in epidemiology are smoking, drinking, obesity and physical activity:²⁶

1. Smoking. Almost all studies find worse health and higher mortality for smokers versus non-smokers (Smith and Kington 1997:143; Lampert 2000:164). Smoking leads to cancer, cardiovascular and heart diseases (ibid.:134). High mortality rates for those who quit smoking show that people often do not quit until very late, when they may already be ill (Hummer et al. 1998a). This can bias the measurement of mortality differences between smokers and non-smokers, especially if past smoking behavior is not taken into account.

2. Alcohol. Drinking alcohol increases the risk of injuries and cirrhosis (Smith and Kington 1997:134; Lampert 2000:164). In contrast to the clear negative findings about tobacco, many studies do not find higher mortality or worse health for drinkers. This has been explained by a beneficial effect of moderate drinking (Smith and Kington 1997:143; Jeune 2002:79ff, see Section 1.3) and possible selection effects: Persons may stop drinking when they know that they have a serious health problem. Thun et al. (1997) even find that death rates for cardiovascular disease (CVD) were lower for drinkers (those who consumed one or more drinks per day) and moreover, the level of intake does not seem to matter. But overall death rates are lowest for those who drink one drink per day.

3. Obesity. Being obese cannot be described entirely as a behavior because there are also diseases leading to obesity, but generally and for our purposes it can represent the intake of too much – and probably the wrong types – of food, in addition to a lack of physical exercise. Obesity can lead to heart disease, hypertension, diabetes and osteoarthritis. Obese persons have more mobility problems and more problems with Activities of Daily Living (ADL). Obesity is often measured with the Body-Mass-Index (BMI)²⁷. The WHO overweight levels based on the BMI are: I. 25-30, II. 30-40, III. above 40 (WHO 1995).

Generally, obesity rates increase over time and obese persons have higher mortality. But the relationship between BMI and morbidity or mortality has a J-shape or a U-shape, meaning that being underweight and overweight both imply higher mortality. Women

²⁶ For smoking and drinking, the example of Denmark already served as an illustration of their possible impact on mortality in Section 1. 4 .

²⁷ The Body Mass Index is the weight of a person in relation to height. It is calculated by dividing the body weight (in kilograms or pounds), divided by the squared body height (in meters or feet).

are more obese but less overweight (light obesity) than men. It has been shown that low social status groups and less educated persons have a higher BMI. This relationship is more pronounced for women (Smith and Kington 1997:128).

The association between obesity and mortality changes over age. At age 50 the heaviest persons have the highest mortality, obesity rates decline with age, and the maximum limits for a healthy BMI increases with age (Himes 2000:77). In old age there is an interesting change in the association between obesity and health which may be due to the fact that in old age low weight is an indicator for health problems (reverse causation bias)(Greenberg 2001). The elderly have an increased probability of weight loss (Losonczy et al. 1995:314) and obesity may not be harmful for them or may even be negatively correlated with mortality. Normally, weight loss can be based on good health (e.g., diet or sports), but in old age it is mostly negative (Losonczy et al. 1995:320) because weight loss may well be related to muscle loss or bone mineral density loss (Greenberg 2001:1076). Losonczy et al. (1995:319) show that after controlling for illness-related weight loss, the thinnest persons have the lowest mortality. When weight changes are controlled for, BMI is no longer predictive of mortality (ibid.)

4. Physical activity. Sports have been found to be practiced more in higher social groups. Of course, this finding has to be balanced with the fact that lower class persons more often have an occupation that requires physical activity (Sundhedsministeriet 1994d:25). But not all physical activity on the job is as healthy as physical leisure time activity like sports and outdoor activities. Moreover, in old age there is not much effect leftover from one's occupation, but habits (including bad ones) concerning physical leisure time activities probably survive until older ages. Habits are an important aspect that is only rarely mentioned in epidemiological literature, an exceptional example being Klein (1996:372).

Thus, the overall assumption of a positive correlation between social status and beneficial physical activity is justified, although physical exercise may be especially biased by cause and effect because unhealthy persons may be unable to exercise (Smith and Kington 1997:136).

Health behavior does not only include the four items presented above (that are, incidentally, relatively easy to measure and often included in health surveys). Health behavior also consists of "illness behavior". This notion does not only include the use of health care (Grundy and Holt 2001:895f) but also the way symptoms are perceived,

evaluated, acted upon or not. Its realm also includes preventive behavior and different reactions to acute illness (Krause 1990:227). All this is different in different social groups and may also be different in old age. But knowledge concerning what factors “illness behavior” depends on in old age is rare. There are findings suggesting that social control over health behavior decreases with age (Tucker et al. 2004) and that married persons have more control and responsibility over their health behavior (Müller 1993:79). By implication, the latter finding would also result in worse health behavior among old people, especially elderly women, because they are more likely to be alone.

4.4.2.5 Psychosocial circumstances

Psychological factors consist of a mixture between individual predispositions and characteristics (personality) on the one hand, and social factors like social capital, integration, and support on the other hand (Christensen 2001:94). The first component also includes the concept of habitus as a relatively stable individual way of perceiving and reacting on experiences. The two dimensions can interact, e.g., in the case of stress. This group of factors is not totally distinct from other groups, e.g., some psychosocial explanations are based on material explanations (Stronks 1997:166) and of course some forms of cultural capital can be subsumed here under psychosocial circumstances rather than under structural conditions.

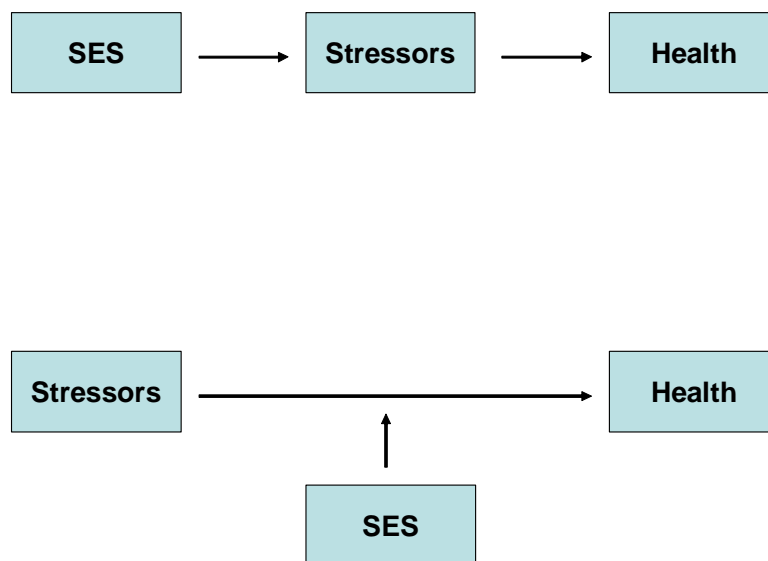
Some indicators for psychosocial factors used in the literature are based on vague concepts that are difficult to measure like empowerment, relative social status, integration, stress, and control, as those proposed by Grundy and Holt (2001:896). But Beckett (2000:116) claims that e.g., social support, stressors and self-efficacy are important health determinants and as indicators they are superior to traditional risk factors like smoking, drinking and exercise. This section will focus on stress, social capital, marital status, and children.

1. Stress can be caused by objective living conditions like financial problems or unemployment (Lampert and Maas 2002:222; Beckett et al. 2002), or also by the mere perceived danger or risk of something, such as losing a job. The latter has been shown in a study where workers’ health worsens already when they are informed of an impending crisis of their employer but before they actually become unemployed (Wilkinson 1994:71). Stress can also come from the more subjective psychosocial environment (Knesebeck et al. 2003:1643) that may be influenced by relations to other

persons in the social environment like relatives, neighbors, etc. Negative and stressful relations to other persons are conceptionally very close to the concept of social capital, but with a negative sign.

Analogous to my description of behavior as an independent health relevant factor, it is difficult to identify the proper impact of stress given the identifiable factors which have a causal link to stress and occur prior to stress. The causal pathway between socioeconomic status, stress, and health is not obvious because it is not obvious that lower classes have more stress, but there are good reasons to assume this (Lardner 2001:87; Stronks 1997:79; Adler et al. 1994; Steinkamp 1993; Brunner 1997). If we agree that people with lower socioeconomic status experience more stress, the causality would go from social status via stress to health. If the amount of stress does not depend on social status, the impact of stress on health is mediated by socioeconomic status because the ability to cope with stress is higher in higher social status groups. Figure 4.5 shows these two different causal pathways, focusing on the different relative position of stress and socioeconomic status but not on other possible causal pathways.

Figure 4.5: Simplified pathways between socioeconomic status (SES), stress and health



Source: Stronks 1997:79

Stronks (1997:79) compares these two models and claims that the first is more plausible. Likewise, Huisman et al. (2004:439) argue that low status increases exposure and decreases the ability to cope with stress:

“Low status groups are arguably more likely to be exposed to stressful environments during their lifetime, and these, in turn, reduce individuals’ reserve capacity for managing stress, thereby increasing vulnerability to negative emotions and cognitions with effects on health.”

Stress originates not primarily in objective problems but in the subjective way of coping with problems (Steinkamp 1993:117). Stress stems from unsuccessful coping strategies. Since objective problems, stressful situations, and harmful life events are more common in lower classes and because these classes also have less ability and fewer resources to cope with these problems (Steinkamp 1993:115; Lampert 2000:164), it is plausible that both of these disadvantages accumulate in lower classes (Elder and Caspi 1990). Lower classes have less self-efficacy, control, and competence (House et al. 1994:214). The Black Report (Townsend and Davidson 1992) finds that low social status implies a lack of control and in turn a higher risk of illness (Steinkamp 1993:118). On the contrary, upper classes tend to see their environment as coherent and controllable and therefore less stressful (Geyer 1997:38f).

Besides these possible class differences in the exposure and impact of stress, there are basic problems with this concept. The relationship between stress and a possible danger to health is not linear, i.e., a small amount of stress is physiologically healthy and hardens (hormesis) (Christensen 2001:93). To define the turning point at which stress becomes harmful is very difficult because this also implies that a single stressor, e.g., working environment, can be either positive or negative (Elder and Caspi 1990:26).

Correspondingly, there are different opinions concerning the impact of stress on health and the usefulness of this concept in epidemiology. For example, Deaton and Paxson (2001:132f) and Sloan et al. (2005) find this research on stress promising whereas Davey Smith et al. (2001:114) find little support for a general susceptibility entrained by stress.

2. Social capital. The concept of social capital has been used for many different purposes. First proposed by Bourdieu in the late 1970s, the notion has also become famous through publications by Coleman (1988) and Putnam (1995) who use slightly different concepts of social capital to study different topics. The relationship between

social capital and health and mortality has been studied by Kawachi et al. (1997), Lochner et al. (1999), Kawachi and Berkman (2000), Mielck and Bloomfield (2001), Herzog et al. (2002), and Grundy and Sloggett (2003). Some findings suggest that social capital is not very important for overall health (Beckett et al. 2002:206), but rather for mental health (Steinkamp 1993:117). The protective effect of social contacts may be higher for women than for men (Beckett et al. 2002:194).

Kawachi and Berkman (2000:184) propose three ways that social capital can affect health: via behavior, via the influence on access to health care and through psychosocial processes. Less social capital means less support and a lack of control (Marmot 1994:43). Social capital is helpful when a person needs information, connections, and emotional and practical help. There is no good measure for social capital on the individual level because both the structure and network of social relationships are essential to this concept and it is difficult to measure it individually (Kawachi and Berkman 2000:176). Religious activity can be an indicator for social capital (Bassuk et al. 2002:521). The effect of religion on mortality is an independent branch of research but it is mentioned here with social capital because some of this assumed causality works in a way similar to social capital. Religious attendance is associated with lower mortality. The causation may work via social networks, social control, communication, financial assistance and social norms from religion, e.g., being religious is associated with less cigarette and alcohol consumption (Rogers et al. 2000:10).

3. Marital status is also a classic social structural variable, but it is presented here as a psychosocial factor because its impact on health works to a large extent via psychological factors. Married persons have better health and lower mortality than never married, divorced, and widowed persons (Klein 1993a:109). The reasons for this finding are diverse: support from a close person, emotional well-being, mutual control over health status and health behavior, and taking responsibility for one's own health causes a mortality advantage for married persons (Klein 1993b:724f; Müller 1993:79). The mortality differences between marital status groups are mediated by the socioeconomic status, e.g., the decrease of income after the loss of the spouse is greater for low status groups (O'Rand et al. 1999:67). Klein (1993a:109) also suggests that rich persons suffer less from widowhood.

Marriage has been found to provide different benefits for men and women. Unfortunately, these findings about marriage do not all point in the same direction.

Thus, it is still unclear who benefits more and at which age from marriage (see Brockmann and Klein 2004). Klein (1999) shows that women profit more from marriage, while results for the USA show that only men have an advantage through marriage (Klein and Unger 2001). More advantages for men in old age have been explained with the fact that they are more likely to have a younger spouse who may well have better health and be able to care for her husband (Beckett et al. 2002:206). But generally, if a partnership also implies negative consequences or at least less positive consequences for one partner, it can be related to the level of stress and the position in the relationship which is different between genders. The situation for widows is also different from the situation for widowers. Couples tend to have less social contact, so after the death of a spouse social relations change and women may have more social capital to rely on because there are many more widows than widowers. The experience of the death of the spouse is more common for women than for men. Accordingly, Christensen (2001:95) finds fewer disadvantages in mortality for widows compared to widowers. On the other hand, women lose more money than men after becoming widowed (Delbès and Gaymu 2002:884) and widows mostly give help to friends and relatives while widowers mostly receive help from them.

Another insecurity concerning the relationship between health and marital status is the direction of causality. It is not clear whether married persons are healthier because of the partnership or if they are married because they have better general health than those who do not marry (selection into marriage). Hummer et al. (1998b:566) discuss this issue. The majority of findings and arguments speak against the selection hypothesis (Klein 1993b:728; Goldman 2001a; Blane et al. 1993:8f).

4. Children. Considering parenthood as a psychosocial factor, it is again obvious that it belongs to different categories of health factors. Having children is as much a structural variable – i.e., it expresses one's individual location in the social structure – as it is a psychosocial variable. Maybe it also reflects a behavior since having children in developed countries is at least partly the result of a decision, although this decision is most likely not made because of the positive effect parenthood has on health and mortality. The notion of “reproductive behavior” stresses this aspect. Treating parenthood as a psychosocial variable is justified by the fact that it usually coincides with marital status, which I also classified as a psychosocial factor. Together these two factors represent the aspects of family planning and family formation.

Having children is a beneficial psychosocial factor. It is also not only a predictor for health but also an outcome of one's health status, since unhealthy persons are less likely to have children. Most importantly, the net effect of parenthood on health seems to consist of a negative influence on physical health that does not only affect mothers but also fathers (Christensen 2001:82), and social gains in different stages of the life course. The gains seem to outbalance the costs because parents very often have lower mortality than childless persons (Doblhammer 2000; Beckett et al. 2002; Jeune 2002:77f). But there are also studies that do not find this relationship (Lampert and Maas 2002:239).

The above presentation of a wide range of possible factors being grouped into classes of factors is for analytical purposes, to provide a better understanding of the principal differences between them. But this should not neglect the fact that in reality many factors contribute to health and to health differences. Almost all of them belong to different categories of factors. These may not only be additive but they may interact in complex ways.

4. 4. 3 Fundamental causes

The notion of fundamental causes or a single fundamental cause comes from a concept that stresses the existence of underlying factors which are the real causes for more proximate risk factors like health behavior or stress. Some authors criticize the kind of epidemiological research that concentrates on proximate determinants (e.g., Link and Phelan 1995:81). They say that certain cultural values make us focus on individual risks and responsibilities. Thereby we could ignore the risk of blaming the victim by identifying many different proximate determinants of higher mortality instead of revealing the basic risk factors that make people adopt poor health behavior or an unhealthy lifestyle. Wilkinson (1992:1084) suggests:

“The point, after all, is not to identify each separate risk factor in an attempt to account for the myriad of separate contributions to the lower class health disadvantage [...] but to identify points at which it is possible to intervene in the social processes which make almost all the common causes of mortality and morbidity more common in the lower classes.”

House et al. (1994:230) express it in a different way:

“The distal cause may operate to produce the same outcome through different intervening variables or mechanisms at different times or places. If one intervening variable or mechanism is not relevant or operable, another may substitute for it, maintaining equifinality in the link between distal cause and outcome [...] Chronic diseases and their risk factors have replaced infectious diseases and their risk factors as the major cause of morbidity, disability, and mortality, and have come to be characterized by the same socioeconomic gradient.”

The assumption is that the social stratification system, i.e. social inequality as such, produces socioeconomic differentials in health and mortality and that, e.g., access to helpful resources is a fundamental cause. Many of the classic risk factors in epidemiology are part of the mechanism but they are not the underlying causes (Link and Phelan 1995:81). Proximate determinants like the Body-Mass-Index, drinking, or smoking are not causes because they just relate the socioeconomic status to health and mortality (Kunst et al. 1999:219), and their distribution is caused by social structure (Stronks 1997:169). Fundamental causes cannot be explained by tracing a specific pathway (Link and Phelan 1995:88). Even if this criticism sounds radical and anti-positivistic, many research findings point in this direction, which is worth discussing in more detail.

A very similar social gradient is found for nearly all diseases and causes of death, indicating a common underlying factor or factors (Hertzman et al. 1994:69). This factor causes a general susceptibility of persons in lower social positions (Marmot 2000:364). Many studies have shown that including a risk factor in a statistical model does not change the effect of socioeconomic status on mortality (Smith and Kington 1997:143f). For example, in the Whitehall study, only a small part of the social mortality gradient for lung cancer and coronary heart disease (CHD) could be explained by smoking, activity, blood pressure, and cholesterol (Valkonen 1996:64). This means that smoking behavior, for example, is not a fundamental cause (Davey Smith et al. 2001:90) because even if it is controlled for, different classes get cancer differently (ibid.:110). Moreover, the same social gradient for coronary heart disease is found for smokers and nonsmokers (Marmot 1999:22). Smoking's association with health did not change, but its association with socioeconomic status did (Link and Phelan 1995:87), so the role of smoking as an explanation for social health differences is only temporarily correct and not fundamental. Another example, namely higher mortality in lower classes given the

same heart problem, has been explained with differences in the quality of care, diagnoses, and appropriate changes in health behavior (Kåreholt 2000:19). Japanese men are twice as likely to smoke as men in the USA, but they have lower rates of lung cancer and a longer life expectancy (Lardner 2001:88).

The idea that one or a few fundamental features and disadvantages express themselves constantly in many different kinds of health disadvantages is similar to Bourdieu's understanding of capital. This capital can operate in the form of resources like knowledge, money, power, prestige, and social connectedness and is transportable from one situation to another even if health-related situations with their specific risk factors change (Link and Phelan 1995:87). Progress in the theoretical understanding of social differences in health could be made if the concept of fundamental causes was made more concrete, maybe by taking Bourdieu's concept of capital as a theoretical and empirical guideline.

Concepts like the fundamental cause concept and Bourdieu's capital theory that are characterized by an assumption of an almost omnipotent resource, should be taken with a grain of salt because they may lapse into structural determinism (Link and Phelan 1995:81). This theoretical framework leaves practically no playroom for a change in individual destiny and underestimates the individual chances to influence one's health outcome in a specific situation. Moreover, this theoretical approach offers only a vague idea of what a fundamental cause could be, and whether we actually look for one or for many fundamental cause(s) (Kunst et al. 1999:201). The definition given above as an example that "helpful resources" is a fundamental cause is not satisfactory for an empirical analysis. However, there are good reasons to point out that each cause of death as the most proximate factor is linked to different specific proximate causes but not necessarily to different fundamental causes (Hummer et al. 1998b:568). The resulting research questions also lead in an interesting direction: Why do socioeconomic differences exist for all causes of death? Why can some groups in society manage to postpone death from almost all causes when others cannot cope with any of them?

The fundamental cause approach has different implications for policy compared to the proximate determinants approach. Following the fundamental cause approach one could argue that specific measures against proximate risks are only modestly useful as long as fundamental cause persist (Kunst 1997:126). Instead, more money from health care should be spent on housing and education (Nichols 2001:135). Instead of having a large impact on one specific risk factor like smoking, this alternative would have little impact

on all diseases (Link and Phelan 1995:89). Traditional health promotion focuses on proximate factors and may therefore not be successful (Davey Smith et al. 2001:91). This strategy tries to change proximate determinants like smoking, but it may not change anything with regard to the overall health level of disadvantaged groups (Link and Phelan 1995:86). The idealistic idea is that not just behavior but the causes for behavior should be changed (ibid.:88). So we come to the fundamental question of whether social health differences should be mainly addressed by medical or by social responses. Medical response or individual choice will probably not solve the problem, although they can help. “Aspirin can relieve a headache, even if the cause is poverty” (Marmot 1999:17).

Policy implications of the different opinions in this debate will not be further discussed here²⁸. One implication for this study is that the idea of a fundamental cause approach supports my empirical approach in that it uses socioeconomic status, i.e., the fundamental position of a person in the social structure as a predictor for health and mortality (Kunst 1997:195ff). The idea of a fundamental cause also supports my proposal that mortality differences are indicators of social inequality (Valkonen 1996:64).

4. 4. 4 Different levels of social determinants

When possible social factors influencing health and mortality are considered, it is not enough to look only at the individual level. The concentration on individual characteristics which are also applied in the empirical part of this study is based on two reasons. First is the pragmatic reason that the data is collected mainly at the individual level. Only very little information about the family and the household is offered. Data which could be collected independently from the original data I use (HRS and Danish registers), and that would describe the social level, e.g., infant mortality rate, level of unemployment, income inequality, etc., would be very difficult to match to the individual information. Moreover different statistical methods would be necessary for such a multilevel analysis.

Second, there is the theoretical reason that all social mechanisms influencing health and mortality must have an effect on the individual because health and death are purely individual. It is worth keeping in mind that the definition of social factors implies that

²⁸ For literature and a further discussion of both approaches, see Hummer et al. (1998b:563).

they come from the interaction of and communication between persons, i.e., from society. In the case of mortality, somewhere in the causality chain social factors are transferred to a strictly individual level. For this study the reduction to individual variables is based on the assumption that the measured individual variables can really describe the effect of social factors. An example that illustrates this rationale is unemployment: Durkheim (1982 [1895]:129) pointed out that society is more than the sum of its parts. It is plausible that a high unemployment rate in a society has an effect on individual well-being that goes beyond the effect of individual unemployment for the individual. The effect of high overall unemployment is not zero for employed persons. A high unemployment rate is more than the sum of its parts and affects the whole society (Marmot 2000:360). But in this example, the difference between employed and unemployed persons is still substantial and the variable for individual unemployment grasps much of the effect of unemployment. Following this compromise and restricting my empirical analysis to the use of individual level indicators, I propose to keep in mind that information about the individual level of income, education, etc. is derived from larger social and economic processes that shape the distribution of these resources in society (Lynch and Kaplan 2000:22).

4. 4. 5 The Wilkinson hypothesis

A prominent and controversial example for the impact of a social phenomenon on the aggregated level on health is the hypothesis that higher social inequality and especially income inequality in a society is responsible for higher mortality. The following pathways have been suggested to explain this relationship. Income inequality is the cause for a lack of social capital, cohesion, social trust, self-esteem and a cause for disinvestment in social capital. It can also be a cause for stress (Wilkinson 1992; Kawachi et al. 1997; Fiscella and Franks 1997; Kåreholt 2000:10). Perceived deprivation, hopelessness, “underclass fatalism” (Elkeles and Mielck 1997:139), depression, isolation, insecurity, and anxiety are all results of relative poverty and can additionally cause worse health and higher mortality. Other similar explanations do not see the causal link between income inequality and mortality via social capital or psychosocial factors, but instead take income inequality as a parallel phenomenon and as an indicator for disinvestment in social capital (Kaplan et al. 1996): those societies that do not care about inequality and tolerate it are also those who disinvest in social

capital and have a low level of it. Those societies do not care about the poor either and have a higher mortality (Kennedy et al. 1996). In this second explanation, income disparity is just an indicator for a lack of social capital, which has been shown by Kawachi et al. (1997:1497). Maybe there is a common background factor for both phenomena, low social capital and high mortality (ibid.)

According to both hypotheses, the income distribution is more important than the overall level of income (Hertzman et al. 1994:70). The underlying idea is similar to the argument mentioned above. The well being of the population is more than the sum of individual risk factors; social determinants are not individual (Kawachi and Berkman 2000). More equal countries have been shown to be more cohesive and better integration is known to benefit health (Wilkinson 1997:593).

To answer the question of whether it is poverty or rather income inequality that affects health, many studies have investigated the association between income inequality and mortality while controlling for the level of income. Kaplan et al. (1996) study the 50 United States of America in 1980 and 1990 and find a correlation between the income share of the poorest 50 percent of the population and mortality, controlling for median income. They find that different percentages of black people cannot explain the relationship. They suggest that income inequality is a common background factor for worse health and disinvestment in social capital. The same logic is applied by Wilkinson (1992) who found an association between income inequality and mortality, even when controlling for poverty.

A large number of authors support the hypothesis that higher inequality leads to higher mortality, even after controlling for possible confounding variables (Mcisaac and Wilkinson 1997; Goldman 2001a; Dunn 2005). Mcisaac and Wilkinson (1997:51) say that this association is true for younger ages but is spread over most of the life course. Some studies even find that when income of the poor is held constant, infant mortality is higher when the rich receive more income (Waldmann 1992).

Another argument that has been tested with empirical research is that the absolute level of the Gross National Product (GNP) does not explain differences in life expectancy between rich countries, but the income distribution within countries explains it.²⁹ This again supports the assumption that the relative position within a society is more important than the absolute material (international) standard (Goldman 2001:130). This could be because, first, mortality is more related to relative inequality within countries

²⁹ See Leon 2001 for a discussion of the findings.

than to absolute differences between them. Second, mortality is lowest in countries with less inequality and less relative deprivation, and third, “most of the long-term rise in life expectancy seems unrelated to long-term economic growth rates” (Wilkinson 1997:591). An example mentioned by Wilkinson (1992) is the United Kingdom versus Japan. Japan has a very low level of inequality (Wilkinson 1992:1083) and according to Wilkinson, the UK would have two more years of life expectancy if it was as equal as Sweden or Japan.

There is a clear international correlation between mean income or GDP per capita and life expectancy. This curvilinear relationship levels off for rich countries. Among the rich countries there is much less of a relationship between wealth and life expectancy. This suggests that inequality plays a role (Wilkinson 1994:62ff). Inequality may be more important for mortality than economic growth (ibid.:61) which has not lowered social mortality differences in the past. But maybe the distribution matters more than the level in rich countries because there it is not easy for additional money to improve health much (Marmot 1994). The correlation between the Gini-index and life expectancy does not disappear if the share of GDP that is spent on medical care is controlled for (Wilkinson 1994:68).

Some studies address the question of whether an observed association depends on the choice of measurement of the income inequality. Kennedy et al. (1996) and Kawachi and Kennedy (1997) compare several measures for income distribution and find that most of them are associated with mortality. The authors conclude that inequality has an impact on mortality net of the level of income, and further, the association is not a matter of how it was measured.

In this debate about income inequality and mortality there are contradictory findings and opinions. Osler et al. (2002) find an association between income inequality and mortality in a large sample of inhabitants of Copenhagen. However this association disappears after controlling for income level. Fiscella and Franks (1997) also find that the correlation between income inequality and mortality disappears if household-level income is controlled for and conclude that poverty – and not income inequality – is the important factor. They say that the inequality question is interesting but that other indicators like family income are more important. Wilkinson replied to their criticism with a commentary in the same issue of the *British Medical Journal* (Wilkinson 1997). In the discussion, the question was raised of on what level the individual subjective

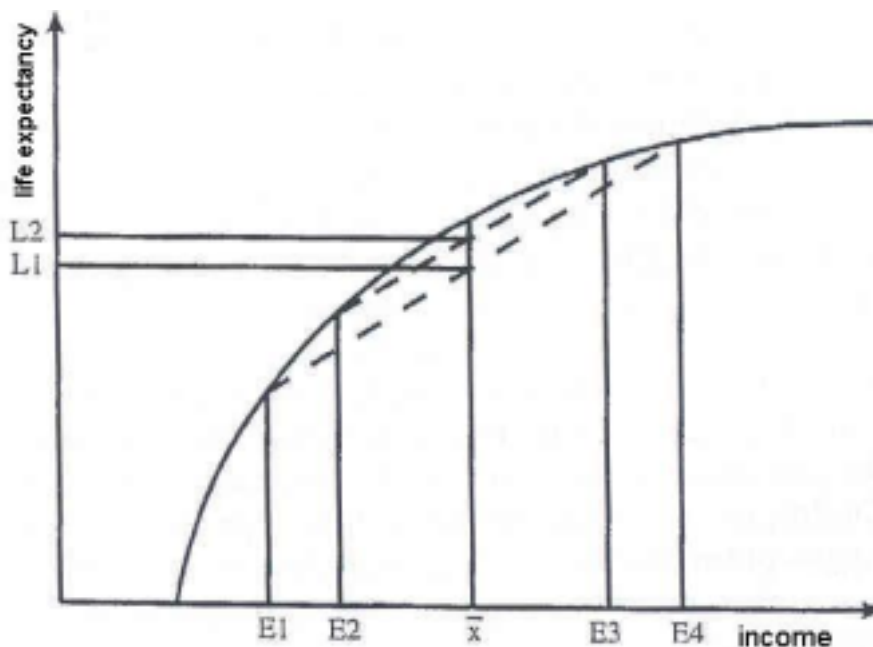
comparison between one's own income and other people's income status actually happens. Is it the neighborhood where the feeling of inferiority originates, or rather through the countrywide perspective where income is disproportionately distributed to rich persons that one does not know personally?

Deaton and Paxson (2001:131) give a weak counter-argument to Wilkinson's hypothesis:

“when mortality was falling the most rapidly in the late 1970s and early 1980s [...] [in the USA], inequality of income was also rising rapidly [...] It is hard to understand why, if income inequality is so important in explaining mortality differences across states in the United States, as well as differences between the United States and other developed countries, mortality should have fallen most rapidly just when inequality was rising most rapidly.”

These broad correlations on the macro-level may not be suitable for ruling out the above hypothesis. A more substantial criticism presented by the same authors, Smith (1999), Wagstaff et al. (2000), and Mielck and Bloomfield (2001), is based on the curvilinear association between income and life expectancy in Figure 4.6.

Figure 4.6: The association between income and life-expectancy (schematic)



Source: Mielck and Bloomfield 2001:29, changed

Because of the shape of the curve there is a negative correlation between the mean and the variance. In countries where income is unequally distributed which graphically lie

between E1 and E4, there is a lower life expectancy (L1) than in countries where the income is between E2 and E3, although both have the same average income. This is because the mean life expectancy is pulled down by those with low income more than it is pulled up by those with high income.

Another reproach against the Wilkinson hypothesis is that this hypothesis makes an ecological fallacy. This criticism cannot be rejected nor accepted easily because income inequality cannot be measured on the individual level and is, as such, an ecological variable which reflects a property of the population and not of the individual.

A literature review by Wagstaff and van Doorslaer (2000) gives a good overview of several hypotheses on the association between inequality and health. Mellor and Milyo (2002) offer a review of the book edited by Auerbach and Krimgold (2001) that has been cited several times above and which is dedicated to looking at the relationships between income, socioeconomic status, and health as well as to the Wilkinson hypothesis. According to their review, there is no clear evidence showing whether to accept or to reject this hypothesis. I would like to close this section by offering a few different citations that present more modest opinions about this hypothesis than either absolute verification or falsification:

“The disconnect between economic prosperity and well-being in the United States tell us that it is not just economic growth that matters, but also distribution of economic benefits Kawachi and Kennedy.” (2001:26)

“It would be foolhardy to say that inequality ‘causes’ sickness. But perhaps not a great deal more foolhardy than to say that carcinogens ‘cause’ cancer.” (Lardner (2001:88)

“No further undifferentiated economic growth is needed because it helps to remove the material but not the social problems even if it provides more luxury.” (Wilkinson 1994:61)

“...could it be said that each individual’s desire for more income is more a desire to improve his relative standing in society than it is a desire for a higher level of material consumption? [...] it would mean that is not legitimate to sum up individual desires for more income into an aggregated societal demand for economic growth.” (ibid.:73f)

4. 4. 6 Life course perspective on the causality from socioeconomic status to health

“The ‘life course’ is the individual experience of the collective social process of aging. Life courses are social because they have general and observable patterns which are part of the structure of society [...] the life course is both an individual and a social process of ageing.” (Vincent 1995:9)

One’s social situation and health status in old age depend in many ways on previous circumstances. Therefore the comprehensive study of health and mortality determinants needs to consider the life course (van Wissen and Dykstra 1999:269). Because of its time dimension, the relationship of cause and effect is also related to the life course approach, which is an organizing, synthesizing approach but not a theory (ibid.:273). Social theory of the life course is ambitious, and so is social theory of death, partly because of biology and partly because it is very difficult to relate the coexisting principles of determinism, path dependency, and openness to current conditions. Maier (2002) gives the advice not to broaden the field by including everything, but to carefully consider it and deepen the insights into the process that leads to death. Naturally, it is difficult to define what is relevant for this process, if in principle one’s whole life, and even one’s parent’s life, could be important (van Wissen and Dykstra 1999).

Income, marital status, and gender roles vary over the life course. Smoking, alcohol, diet, stress and health care factors can have latency. Hummer et al. (1998b:556) suggest the following sources of information to take care of the longitudinal nature of the mortality process: 1. historical information, 2. prospective longitudinal data, and 3. retrospective data (Hummer et al. 1998b:566). Elder and Caspi (1990) go even further and want a special explanatory model for each cohort including the intra-generational transmission of behavior to explain an individual’s personality. Instead of following such ambitious suggestions, I will give a short overview of research findings on the relationship between childhood health and health at old age which will serve to illustrate the complexity and difficulty of this research approach.

Childhood conditions such as parental socioeconomic status, education or epidemics in childhood can affect adult and old age health outcomes. It is often difficult to get information about the social and health status during childhood for persons who are now old. Proxies that are used for this purpose, e.g., education as an indicator for childhood quality (Davey Smith et al. 2001:94) or height as an indicator for childhood health, may be unreliable (Blackwell et al. 2001; Grundy and Sloggett 2003:936, 940).

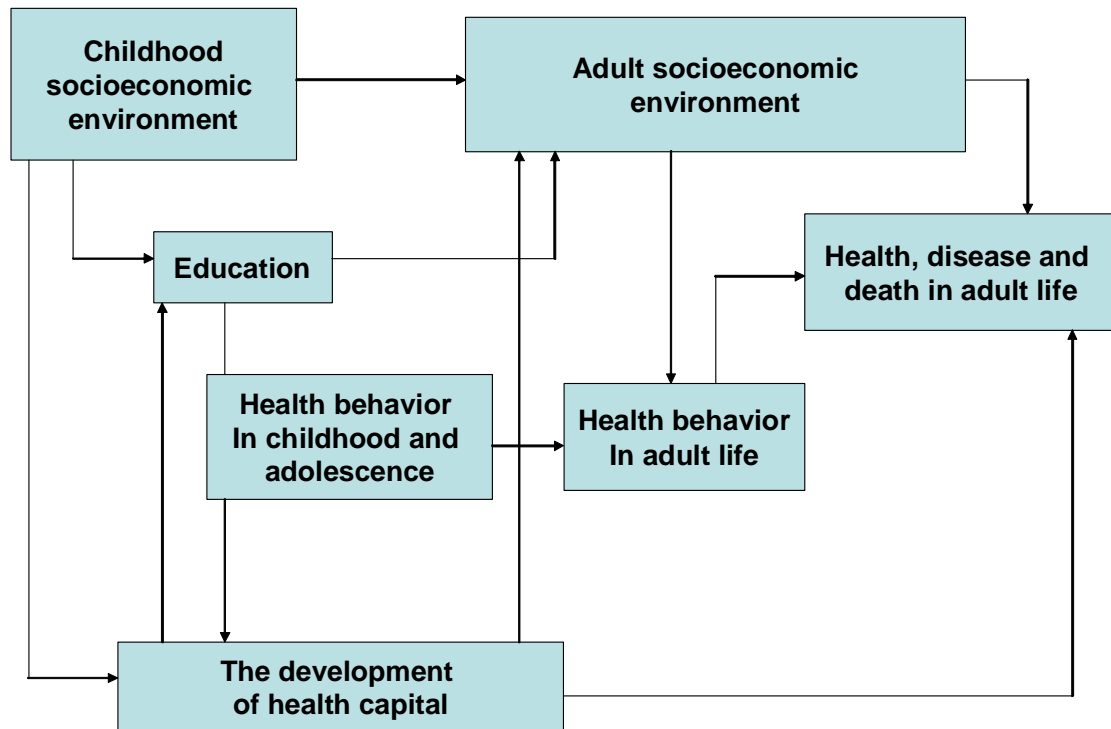
Two different ways for early life to exert an influence on later life are plausible: first, cohorts that were affected by high overall infant mortality or specific health threats may be selected and thus have a lower subsequent mortality. On the other hand, it is also possible that the health status of such cohorts is affected without mortality selection, i.e., without people dying, which results in worse health and higher mortality in higher ages. The first process is called selection and the second debilitation (Doblhammer 2004:53ff).

One model that describes the life course effect of socioeconomic status on health suggests that differences in exposure are small in younger ages when education or income do not have time to affect living conditions and health. Then the importance of SES increases throughout mid-life until it again decreases later in life because of the welfare system and disengagement from main stratifying systems, e.g., the labor force. This is why House et al. (1994:221, 228) suggest that socioeconomic health differences are narrow in early adulthood, wider in middle age and smaller again in old age. What is not convincing about this model is that it suggests that there are no differences in the socioeconomic positions of children only because their income and education have not yet had an effect. The socioeconomic status of their parents, however, cannot be excluded as an important factor, i.e., Smith (1998:195) finds that childhood poverty is more important for coronary heart disease in later life than adult poverty is.

Concerning the suggested narrowing of mortality differences in old age, House et al. find that chronic conditions and limitations in functional status occur at older ages in upper classes. They interpret this as a postponement of aging in upper classes (ibid.:221). This age gap in the aging process between lower and upper social groups implies social differences in health and mortality even in high ages.

The following causality scheme proposed by Kuh et al. (2004:374) shows the possible interaction between different factors in early and middle phases of the life course.

Figure 4.7: Causal relationship between socioeconomic status, health behavior, and health over the life course



Source: Kuh et al. (2004:374), changed

Having a poor childhood has a different impact on the subsequent health of boys and girls (Elder and Caspi 1990:27ff) in that boys are vulnerable at an earlier age. The problem with such statements is that childhood conditions influence both subsequent socioeconomic status and subsequent health. Davey Smith et al. (1997) argue that childhood is important for life opportunities. The pathway to health via adult socioeconomic status can, in principle, be revealed if socioeconomic status is controlled for. But this creates the risk of hiding the impact of childhood conditions because they are correlated with subsequent socioeconomic status (Grundy and Sloggett 2003:940). But in some studies, childhood health affects old age health even when controlling for early and later socioeconomic status (Blackwell et al. 2001). Other studies show that the effect of a father's social class on female mortality (i.e., his daughter) does not exist (Kåreholt 2000:11), and that childhood is not very important as a predictor of adult health (Lynch et al. 1994).

A different life course effect has been suggested by Kåreholt (2000): with statistically insignificant results, the author shows that those with a “good” childhood have a smaller

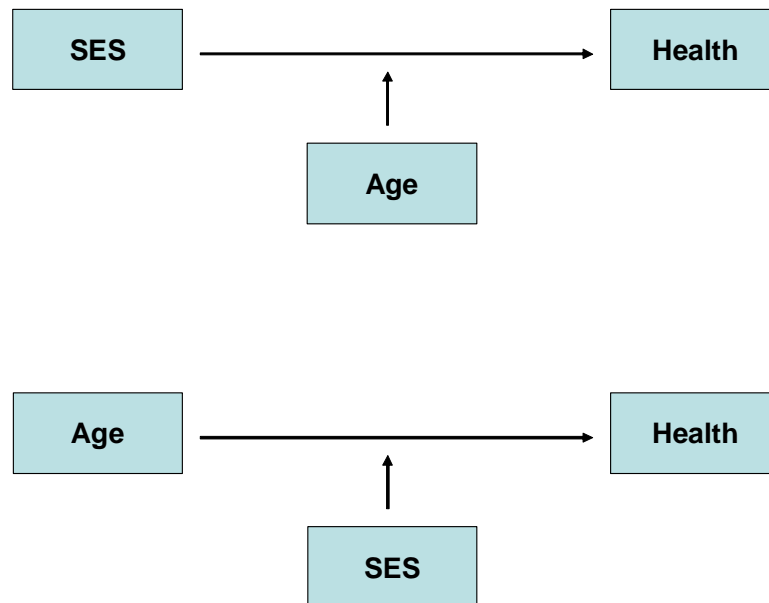
socioeconomic mortality gradient in older ages than those with a “bad” childhood (Kåreholt 2000:1). This would mean that childhood does not influence later health in a positive or negative direction but that it is important for a robustness that limits the subsequent susceptibility against health threats.

If childhood is important for this study of the USA and Denmark it should be considered that the former was much richer and more equal than Denmark in the early 20th century when most of the persons used in my empirical analysis were born (Kunst 1997:140). But to use macro-level data in addition to individual information is a different approach with a different research focus than I follow here. Moreover, there is some indication that the effect of socioeconomic status on health is not biased when childhood is not controlled for (Blackwell et al. 2001).

The study of socioeconomic differences in health and mortality over the life course is interesting because there may be different causes and pathways for different ages (Davey Smith et al. 2001: 113): e.g., disengagement works especially among the elderly (Bassuk et al. 2002:520). But the overall picture is that there is mixed evidence about the relative importance of different parts of the life course for mortality (q10 Vaupel 1998), maybe because we cannot avoid observing the combined effects of many stages in the life course (Kåreholt 2000:2). This means that there are remaining life course effects from childhood and adult ages in addition to new effects from old age (Huisman et al. 2003).

To conclude on this point: age, socioeconomic status, and health interact. The first schematic and simplistic causality model could be that social status influences health while age intervenes. This model is also a model for the research question of this study: to find out if the impact of social status on mortality changes with age. The second model is that age influences health while social status intervenes (see Figure 4.8 below). At any rate, biology intervenes in both age and health (House et al. 1994:213f).

Figure 4.8: Two schematic and simplified representations of the interplay between socioeconomic status (SES), age and health



The exact relationship between these models cannot be explored here. In spite of these remaining research questions, we are reminded of the general overall pattern, which besides being correct, helps to simplify the complicated life course considerations from above: socioeconomic status is rather stable while health generally declines over the life course (House et al. 1994:226).

4. 4. 7 Reverse causation and health selection

Until now in this text and very often in the literature, socioeconomic health differences are treated as health differences that are caused by social differences. But we can also think of health differences that cause social differences. This section will give an overview of a long and ongoing discussion on the causal direction between health and social status. The direction from social status to health is sometimes called causation while the reverse direction is called reverse causation. In some cases the latter is also called health selection because persons are selected into different status groups according to their health status via social mobility. Other names for the latter direction are: health-related social mobility, occupational or social drift, (selective) drift

hypothesis (Elkeles and Mielck 1997), social selection, and discrimination on the basis of health (Goldman 2001b). Since we are looking at two different models for causation, I will use the term reverse causation in the following.

The theme of the mentioned scientific discussion is over the extent to which reverse causation contributes to the observed social health gradient (Lichtenstein et al. 1993; MacIntyre 1997). Strictly speaking, there can be no reverse causation in the research of the socioeconomic mortality gradient because death cannot be the cause for anything, but it is possible that health determines social status and if this is true the social mortality gradient is partly due to reverse causation. Like for the causal direction from socioeconomic status to health discussed above, there are several plausible pathways from health deterioration to a decline in social status, income, or wealth. A status decline may be due, first, to health expenditures or, second, to overall higher consumption because ill people do not expect to live for a long time. Maybe some people also intentionally “spend-down” or transfer their capital to children to become eligible for Medicaid coverage.

Besides these two factors, Smith (1998) suggests that a health event can cause higher health expenses and a lowered ability to work, both decreasing financial well-being (Smith 1998:195). Using HRS and AHEAD data, he studies the predictors and consequences of the onset of new health events (Smith 1999; Smith 2003; Smith 2004) and finds that persons are less wealthy after a health decline. Generally, there are relatively small costs for even severe illnesses. A new major illness means about \$5,000 higher health expenditures with insurance and \$10,000 more without. In many cases where there is no major illness, persons without insurance spend less out of pocket. Insurance may influence both health care utilization and expenditures with the consequence that people without insurance consume less health care and pay less out of pocket. Moreover, they may spend less because they are on average poorer than those with insurance. The idea that the financial status of unhealthy people falls is supported by Soldo et al. (1997:3), who find that unhealthy elderly do not save, whereas healthy elderly do.

Smith concludes that the combination of medical expenses together with income reduction may be the reason for the wealth decline after a health decline. Low income households are more likely to stop working after a health shock (Smith 2003:8ff). Additionally, he suggests that there may be increasing general consumption when people get ill. It is actually unclear whether people save more or less when they are ill

(Smith 1999:150). One special feature of his analysis is that he controls for initial health status, although he himself states that the probability of a new onset depends very much on prior health status (Smith 2003:3). This analytical step will be criticized below.

Adams et al. (2003a) also use data from the AHEAD study and apply a series of probit models. The authors state that there is an absence of a direct causal link between social status and mortality when initial health is controlled for. But they also find a modest causation from social status to health, which they attribute to common genetic and behavioral background factors. They identify causality from health to wealth, something which disappears after working age. The causation from social status to health exists for chronic rather than for acute health problems.

This influential work by Adams et al. was subject to some criticism, part of which was published simultaneously in the same journal issue. Adda et al. (2003:61) argue that the rejection of a direct causal link from social status to mortality is partly incorrect. According to the results from Adams et al., for most of the causes of death that have been tested, such a causal link cannot be rejected. Their causality test actually tests between direct causality on the one hand and no causality or indirect causality on the other hand. They do not test between causality and no causality. Since the pathway from social status to mortality is likely to be a process that develops via risk factors and bad health, the assumption of a direct causal link is not useful or at least it includes a different hypothesis (Adda et al. 2003:62). Using the same data, Adda et al. come to the conclusion that there is no causality from health to socioeconomic status.

Poterba (2003) criticizes Adams et al.'s use of a definition of causality that implies that the relationship between social status and health must not vary over time in order to be considered a causal relation. This is not plausible because, e.g., Medicare can change this causal relationship (Poterba 2003:67). Also, Hoover (2003) mainly criticizes their causality tests and that they controlled for health status. Regardless of whether the causality goes from health to social status *and* mortality or if it goes from social status via health to mortality, the correlation between social status and health is destroyed by controlling for health status (Hoover 2003:123f).³⁰

Hurd et al. (2001) try to eliminate the impact of health on income via work while Smith and Kington (1997) take the level of work to control for reverse causation. They conclude that the causation direction is mainly health to income (Smith and Kington 1997:158). Another attempt to unravel the causation direction was applied by Davey

³⁰ Martelin (1996:127) also argues against controlling for health.

Smith et al. (1990:269): they exclude persons who were unhealthy at the beginning of the study. By doing this the causality from bad health to a lower social status can be largely reduced if not eliminated. The authors find that this does not greatly affect the mortality differentials. The same results were reached when the same test was done by Blane et al. (1993:9) and Beckett et al. (2002:196).

Even if there is an ongoing debate about the causation direction, it is fair to say that most epidemiological research shows that reverse causation is not of major importance (Goldman 2001a:10086). Several studies have investigated the health selection hypothesis (e.g. Fox et al. 1985; Blane et al. 1993; Lundberg 1991b; Kåreholt 2000; Chandola et al. 2003a). While there is some evidence for a certain health-related social mobility that may exist at labor market entry (Power et al. 1998; Smith 1999), by far most epidemiological studies conclude that health selection is not of major importance for explaining social gradients in health and mortality³¹. However, studies in the field of economic research (e.g. Smith) very often come to different conclusions.

The epidemiological and sociological findings seem to be justified first, by findings of only a small degree of health-related mobility that matters mostly at labor market entry (Blane et al. 1993:11; Davey Smith et al. 1994:439), and by the general observation that accumulation and continuities in social status dominate selection and mobility. There is also an opposite kind of health selection, namely the selection of unhealthy people into physically light occupations, which do not have a lower but often a higher status (Otterblad Olausson 1991). Health related mobility cannot contribute much to the social mortality gradient because there are rather stable social differences, and the overwhelming majority of people do not move up or down considerably (Fox et al. 1985; Davey Smith et al. 1994:439; Valkonen 1996:64). Moreover the mobile persons have a significantly different mortality from those who have always been in a certain social group: upward mobility is associated with lower mortality (Mare 1990:384) whereas downward mobility implies a higher mortality level than would have been the case if the person was always in the lower group (Kåreholt 2000:15). This again makes it implausible that social differentiation is created to a large degree by health-related mobility (Chandola et al. 2003a:2060).

³¹ Fox et al. 1985; Lundberg 1991; Wilkinson 1992; Blane et al. 1993; Dahl and Kjaersgaard 1993; Davey Smith et al. 1994; House et al. 1994; Marmot 1994; Backlund et al. 1996; Marmot and Shipley 1996; Ross and Wu 1996; Power et al. 1996; Kunst 1997; Kåreholt 2000; Marmot 2000; Becket and Elliott 2001; Deaton and Paxson 2001; Goldman 2001a, 2001b; Chandola et al. 2003; Koivusilta et al. 2003;

Nevertheless, the question is not whether there is only one specific source of causation at work and at which ages it exists. It is likely that at any age there will be both causation directions which are not exclusive (Goldman 2001b). It is important to notice that causation works not only from social status to health. Also, there is no sudden “knockout blow” in the debate (Smith 1999:165). Without a doubt the process can be described as a co-evolution of health and social achievement (Vågerö and Illsley 1995:219). The question is: which direction contributes considerably to the social gradient of health and mortality? Although this question cannot be further investigated in this dissertation, it might be useful to add two more perspectives to this problem. First, different answers on this question would have different implications for a demand for policy reactions. If socioeconomic status causes social health differences, policy actions should focus on income redistribution, education, employment and lifestyles. If health differences are causally prior to social differences, then the access to health care and health services should be improved (Adda et al. 2003:57). Second, the question of the direction of causation is embedded in the life course perspective on social mortality gradients, as will be discussed in the next chapter.

4. 4. 8 Both causation directions considered together in a life course perspective

The same evaluation of the impact of both causation directions can be done from the life course perspective because, as mentioned above, it is likely that the relative impact of both causality directions changes with age. Generally, there is an additive relationship between the two directions of causation, i.e., health selection increases the social health gradient (Fox et al. 1985:2). However, this conclusion is not straightforward, as discussed by Goldman (2001:121f). Also Kunst (1997:140) claims that mobility, may it be health-related or not, can increase and decrease the gradient. An increase is possible because the accumulation of social and health disadvantages is stronger in certain groups of the population than in others. A decrease may happen because in principle, mobility allows a lower class person to move upwards because of good health, and upper class persons to move downward because of bad health. The latter may seem logically plausible, but it is less realistic than the assumption of an increasing gradient because of accumulation. Even if reverse causation increases the gradient, the determining influence of one factor that is analyzed in an empirical analysis is reduced, i.e., reverse causation causes measurement biases (Hertzman et al. 1994:77).

The problem with the reverse causation hypothesis is that it neglects to define an origin for health inequality in a life course perspective. Therefore, Smith refers to Barker (1997), who showed the impact of conditions in utero for later health. But Smith himself finds the term “fetal programming” to be too harsh (Smith 1999:160). Two arguments can be used against the idea that conditions very early in life are the origin of health inequalities: first, concerning the Barker hypothesis, it must not be overlooked that a large part of health determinants in utero are caused by the social status of the mother or the parents. Second, a high crisis of mortality (e.g., in Russia) shows that current conditions have a strong immediate influence on health and mortality.

Health is usually good in young ages e.g., teenagers show only small health differences (Stolpe 1997). But if health is poor at young ages, one’s inert health constitution, as well as class can account for this. Blane et al. (1993) find a social gradient in health already in childhood. From this it can be concluded that there may have only been a brief period of time in which the social status could affect health, but it is certainly even less realistic to assume that in childhood health already had a repercussion on the social status. It rather suggests the influence of parental social status and moves the life course perspective on the social health gradient beyond one individual life course. Also Goldman (2001:123f) points out that there is selection between generations and intergenerational mobility. This makes the interplay between social and biological influences on health and social status even more complicated. For later childhood, Koivusilta et al. (2003) have shown that causation goes from parental socioeconomic status via health behavior to education rather than directly from health to education to later socioeconomic status (Koivusilta and Rimpela 2003).

For the functioning of the mechanism of health selection, there must be a certain degree of social mobility that is most likely true for younger ages (Fox et al. 1985:6). On the other hand, there is only a small degree of health variation in these ages, making the assumption of a lot of health-related mobility again unlikely. Whereas a precise amount of mobility is difficult to measure, there are findings stating that there are only small mobility differences between the USA and the EU (Kunst 1997:140) and that mobility is more important for men (Kåreholt 2000:10).

Smith (1998:196) claims that after age 40, the impact of health on socioeconomic status is important, maybe the most important direction peaking in older working ages (ibid.:158). Smith (2003) claims that in middle-age, health influences labor, income, and wealth. His argument is that people in their fifties have more health problems than

money problems and therefore, that health is causally prior to money (Smith 2003:3) is not convincing because it does not really favor one causation direction over the other. It seems that the increased mobility in younger ages and more (diverse) health problems in higher ages do not fit together to support the hypothesis of health selection. On the contrary, these two features appear in the wrong order in the life course: according to the hypothesis of health selection, health diversity should emerge prior to mobility.

In another text, Smith repeats the claim that reverse causation occurs mostly in early old ages. He finds that in the HRS data, the direction is from bad health to low income in early retirement, and in AHEAD (the older sample at age 70+) it is from low income to bad health (Smith and Kington 1997:147). For persons in the HRS (ages 51-61), the effect of socioeconomic status on health is reduced by one-half if the effect of health on socioeconomic status is controlled for (ibid.:149).

Furthermore, Huisman et al. conclude that selection effects cannot be excluded as a contributory factor for health differences in old age. If there is such an impact, it is likely to be highest in middle-age (Huisman et al. 2003:872). If this effect is additive to the effect of socioeconomic status on health, it means that in principle a mortality convergence could be due to the diminishing effect of reverse causality from younger old to old ages. By that, the issue of reverse causality would become part of my main research question about convergence or divergence. But because too many strong assumptions have to be made to let reverse causality affect my main question, and because I have no tool to further analyze the problem of reverse causality, this possibility of explaining a convergence in old age will not be included in the discussion of changes of mortality differences in old age in Chapter 5.

To simplify again the differentiated life course pattern of causation, it is likely true that for old age it is lifetime socioeconomic status that influences health and, to a certain degree, lifetime health that influences socioeconomic status. This means that it is the accumulated experience in both dimensions rather than only during a certain period (Smith 1999:149). Another aspect that relativizes the importance of the debate on the causal direction is that in analytical terms, it might be important if socioeconomic status causes health or if health influences status. Still the practical importance is questionable: in most cases a downward social mobility will be followed by deteriorating health and deteriorating health by downward mobility. These two logically distinct processes are just two elements of the same process, namely accumulation of advantages or disadvantages. This accumulation is based on the mutual negative or positive influence

of social status and health. The two directions may not have the same importance for the creation of social health differences but they are both indicators of a dysfunction of the social security system.

4. 4. 9 Indirect selection

Another interesting causal model is called indirect selection (Blane et al. 1993; Martelin 1994; Smith 1999:148; Goldman 2001b). The idea is that there are common background factors that influence both health and social status. This may be the parents, schooling, physical characteristics (O'Rand et al. 1999:64f), or the lifestyle that influences both income and health. The same logical model is applied to explain the relationship between unemployment and mortality: unemployment is not causal for mortality. Rather, it is bad health that causes both unemployment and mortality (Hummer et al. 1998b:558; Valkonen and Martelin 1999:221).

Some authors describe this causation as being more important than the model based on mobility (Fox et al. 1985; Davey Smith et al. 1994; Valkonen 1996:64). Other authors describe it as being unimportant (House et al. 1994:228; Marmot et al. 1995:198; Goldman 2001a:10068). Blane et al. (1993:12) say that this causal model has some meaning in that it is more likely than direct selection. Also, it would lead to an accumulation of social health differences. Hurd et al. (2001) find converging mortality differences between wealth groups in old age. They conclude from the weakening impact of wealth that the causality direction is not from socioeconomic status to mortality. Instead they suggest that there is an indirect selection from something unknown to both socioeconomic status and mortality. Generally, in empirical and theoretical research, this causal model is only rarely discussed and tested, maybe because it is even more difficult to verify than it is to disentangle the opposite causation directions discussed in the previous section.

Summary of Chapter 4

Socioeconomic differences in health and mortality constitute a clear and persistent finding. They can be found in all countries, but not to the same degree. Research findings indicate that these differences increased during the past few decades and that they are larger for men than for women. Health and mortality are two aspects of the same process. Mortality, expressing only the event of death, cannot substitute the measurement of a complex health trajectory, but objectivity and availability make it a widely accepted and valuable health measure. The description of the complex causality between socioeconomic status and health includes the following aspects: the most proximate cause is the cause of death. Five other categories of causes are proposed here to structure the interrelated universe of health-related factors: genes, natural and physical environment, structural and material conditions, behavioral and cultural factors, and psychosocial circumstances. An advanced perspective on causal factors for mortality goes beyond the identification of factors. It suggests the differentiation between proximate and fundamental causes, the latter being less evident but more important because of their persisting influence even under changing proximate risk factors. Together with these different levels of factors, different levels can be differentiated by which causal factors are effective. Most research focuses on individual socioeconomic status. Besides this, the hypothesis is discussed of whether inequality as such increases mortality. Finally, another alternative to the classic social causation model has been presented: the hypothesis that in a life course perspective the relationship between health and social status can only be understood if both causal directions, from socioeconomic status to health and vice versa, are considered.

Chapter 5 Change of socioeconomic mortality differences with age

Interest in the topic of a possible change of socioeconomic mortality differences in old age is rising due to a number of open-ended theoretical and methodological questions related to this issue. Except for a very limited number of studies showing no socioeconomic mortality differences in old age (Valkonen 1993), there is general agreement that differences in health and mortality also exist in old age. However, different results and assumptions exist for the question of whether these differences are larger or smaller in old age than in younger age groups. Decreasing differences have been reported by the majority of studies. In principle, there are three possibilities which are analogous to the three ways social inequality can change, as mentioned in Section 3.5 : divergent, convergent or constant relative differences. In this section I will present different hypotheses, research findings and explanations that support each of these possibilities.

It is important to note that this consideration is for relative mortality differences. The overall level of mortality increases so steeply with age for all social groups, that absolute mortality differences between social groups will increase in most cases in old age (Martelin 1996). The distinction between relative and absolute differences sometimes causes confusion because some authors just speak about increasing differences referring to absolute differences and compare these findings with findings for relative differences (e.g., Liang et al. 2002:304 referring to Marmot and Shipley 1996). Marmot and Shipley (1996) study absolute mortality differences and Huisman et al. (2004) interpret absolute social mortality differences as avoidable numbers of death. This interpretation is based on the strong assumption that health and mortality disadvantages of lower social status groups are avoidable and would disappear if all persons had the same social status. In his dissertation only relative differences are analyzed.

Concerning the two countries under study here, converging health and mortality differences between workers and salaried employees from age 35 to age 60 have been found in Denmark (Andersen and Laursen 1998). Converging mortality differences for older ages have been found in other Scandinavian countries (Otterblad Olausson 1991;

Martelin 1994, 1996; Martelin et al. 1998). For the USA, a number of studies have shown converging differences in old age (Kitagawa and Hauser 1973; Haan et al. 1987; House et al. 1994; Backlund et al. 1996; Elo and Preston 1996)³². On the other hand, Silveira et al. (2005) show that there are substantial differences concerning the burden of illness between categories of wealth even in the last year of life. For Germany one of the rare researchers to address this question is Stolpe (1997) who uses death certificates from the city of Bochum's population and finds decreasing differences. Some studies specify the maximum social mortality differences between age 30 and age 45 (Valkonen 1996:57; Kunst 1997).

In the following two sections I will present arguments that support convergence, and respectively divergence, of social mortality differences in old age.

5.1 Arguments for convergence

1. Aging works as a leveler of social differences because biological processes assume dominance over social determinants and eventually everybody must die, regardless of social class (Liang et al. 2002:295).

It is possible that old age mortality is generally more biologically and genetically determined than mortality in young ages (Klein 1995:315; Mayer and Wagner 1996:273). This assumption is analogous to the leveling of social inequality because of the impact of biological aging that has been discussed in Section 3.5.1. It can be illustrated by the idea that a death between age 40 and 50 is more likely to be caused by some abnormal social situation and living conditions than a death at age 80 where all people are approaching death. However, a death at age 40 can also have a genetic background and the question of whether a person survives until age 70 or until age 80 can depend very much on social factors. Thus the question is whether relative social mortality differences that are defined as being caused by social factors and being independent from the overall level of mortality necessarily decline when we approach "normal" ages at death. A supporting argument is that genetic determination becomes more important in old age (Christensen 2001:79) and the health status depends more on

³² For other countries and further discussion of this issue see Fox et al. 1985; Marmot and Shipley 1996; Mustard 1997; Shkolnikov et al. 1998; Breeze 2000; K areholt 2000; Lampert and Maas 2002; Grundy and Sloggett 2003.

age with increasing age (Lynch 2003:10). This suggests limits to the “plasticity of aging and mortality”. This plasticity is one of the most important recent findings in mortality research (Vaupel et al. 2003; Maier and Scholz 2004).

The extent to which genes determine observable processes and events in the individual life course is very difficult to measure. This question can not be answered here. This first argument describes the possibility that socioeconomic mortality differences converge because social factors in old age have less impact relative to other determinants (House et al. 1994:218) and that advancing age works as a leveler (Dowd and Bengtson 1978). This would consequently result in a weaker association between class and mortality in old age.

2. The welfare state reduces socioeconomic differences in old age through benefits and social policy.

This second argument is based on the idea that the welfare state decreases social inequality in old age by spending a major part of its payments and benefits on the elderly, thereby contributing to a certain redistribution between social groups (see Chapter 2). This effect of the social system could decrease social mortality differences either fundamentally by reducing social inequality or just at the level of the symptom through health related services. This explanation has been used by House et al. (1994:221), Mayer and Wagner (1996:273), Backes et al. (1998:83), Bassuk et al. (2002:522), and Knesebeck et al. (2003).

3. The impact of past stratifying and health relevant experiences, e.g., working conditions, fades out at old age.

The main idea of this argument is that differential exposure to health-damaging factors between social classes is not constant over age. In older ages most people disengage from the main stratifying systems, e.g., labor force (House et al. 1994:228), which means that the life course leads to a fading out of differences (Mare 1990). If working conditions throughout the life really play this important role in middle age it could lead to a convergence of mortality differences in old age. If on the other hand general living conditions are important we would not expect a convergence (Klein 1999).

Some empirical results seem to support this idea. Marmot and Shipley (1996:1178) show a weakening of the social mortality gradient after retirement rather than with age. Also Klein (1993b:724) and Stolpe (1997:59) interpret decreasing class differences in mortality with age as an indication of the importance of working conditions. On the contrary, Fox et al. (1985:6) show that ten years after retirement the social mortality gradient is as steep as before retirement, i.e., there are 50 percent higher death rates in the lowest five social classes compared to the upper class. They argue that if the gradient is as strong 10 years after retirement, it must be the current environment that causes the gradient.

4. The observed mortality differences get smaller in old age but only on the aggregate level because the surviving population is more homogeneous due to unobserved heterogeneity and selective mortality.

Preston (1992:50) describes the impact of selective mortality as follows:

“The diagonal march of birth cohorts across the grid of age and time is at once the most mundane and the most profound process known to demography. Cohorts begin the march with their own unique endowment of social and biological attributes. Along the diagonal, they experience the normal process of development and aging: they absorb the wars, epidemics, recessions, and booms of their time; and they witness the attrition of their members in ways that transform the composition of survivors.

The lockstep progression of cohorts into new age-time blocks affords an opportunity for prediction that is rare in the social sciences. Although we have few clues about what changes will occur in per capita income over the next 20 years, or in the political climate or the fertility rate, we have a great deal of information about changes in the type of people who will occupy a particular age group. For older ages, especially, many characteristics of the pertinent cohorts have been largely determined and are directly observable. The major uncertainty is how the composition of each cohort will change as a result of selective mortality.”

The last sentence stresses the problem: a measurement of socioeconomic mortality differences in different age groups that does not take into account the effect of unobserved heterogeneity shows the correct mortality differences between social groups at the aggregated level. But if this result is biased by the compositional change over age, it hides a possible change of the impact of socioeconomic status at the individual level.

Vaupel (2001:10078) describes this artifact caused by unobserved heterogeneity:

“All populations are heterogeneous. Some individuals are frailer than others, innately or because of acquired weakness. The frail tend to suffer high mortality, leaving a select subset of particularly strong or resistant survivors. This creates a fundamental problem for analyses of oldest-old mortality: as a result of compositional change, death rates increase more slowly with age than they would in a homogeneous population.”

If the increase of death rates is slowed down because of unobserved heterogeneity, this effect of slowing down is stronger for groups with higher mortality because higher mortality means more selection and more compositional change (Horiuchi and Wilmoth 1998:393). As a result, an observed mortality convergence between social groups in old ages could be an artifact of selective mortality, which selects frail individuals out of the population, especially in lower status groups, which in turn makes the mortality of the lower status group similar to the mortality of the higher status group.

The logic of this process is analogous to the well-known mortality crossover between black and white people in old age. In very old age black persons seem to have lower mortality than white persons which is the opposite racial mortality relation of in all other ages (Markides and Machalek 1984; Arber and Ginn 1993:35; Ferraro and Farmer 1996; Beckett 2000).³³ Results suggesting the functioning of this mechanism are offered in many studies (see Nam 1995) as applied to different groups. Besides racial groups smokers can also have lower mortality than non-smokers in older ages³⁴. The crossover probably occurs not because smoking becomes healthy in old age but because very old smokers are selected and are very robust persons with low frailty and low mortality, even lower than that of non-smokers. The logic of a racial mortality crossover can be described as follows:

“[...] higher early mortality in disadvantaged populations leads to greater selective survival of biologically robust members of minority populations at advantaged ages than is the case with advantaged populations. This is not to imply any advantages in the aging process enjoyed by minority or other disadvantaged populations, but rather their great disadvantage in the sense that mortality

³³ There has been a discussion of whether the black and white crossover is partly due to report defaults (Goldman et al. 1995:1726; Nam 1995) but the above interpretation was supported using the Medicare Master Beneficiary Record files from the Social Security Administration. This is a high quality documentation of births and deaths in the USA that showed the racial mortality crossover to be real and occurring at age 87 for women and age 88 for men (Kestenbaum 1992, 1997; Hummer et al. 1998b:558).

³⁴ I have also found a mortality crossover between smokers and non-smokers in the HRS data that will be used in the empirical part of this study (results not shown).

disproportionally removes them from the older population” (Markides and Black 1996:155).

This theory has created some concern in the literature about socioeconomic mortality differences as well. However, valid measurements of the relative importance of compositional change versus individual change are methodologically difficult to achieve and are therefore still lacking. In the literature this idea has generated the following evaluation concerning its impact on socioeconomic mortality differences in old age. House et al. (1994:228) and Lynch (2003:10) wonder if mortality convergence is due to mortality selection. Kitagawa and Hauser (1973) mention the selection hypothesis and Robert and House (1994) apply the idea of selective survivorship explicitly to the narrowing of health and mortality differentials by socioeconomic status. Other authors regard this idea as one possible explanation for a mortality convergence (Kerstenbaum 1992; House et al. 1994; Mayer and Wagner 1996:273; Lampert 2000:165). Some studies consider controlling for mortality selection important (Lynch 2003:14) and speak about a “considerable impact” (Arber and Ginn 1993:35). Some authors mention the possibility that if persisting differences over age are found without controlling for unobserved heterogeneity, the unbiased result where the effect of unobserved heterogeneity is taken into account could even show increasing differences over age (Thorslund and Lundberg 1994:67).

The methodological and computational implications of unobserved heterogeneity and mortality selection constitute an important research question and are addressed in Chapter 9. Here I only intended to present this argument as one possible explanation of why most research findings show converging mortality differences in old age while in fact the pattern could be different.

5.2 Arguments for divergence

In the following I will mention some contradictory research findings and arguments, namely those supporting stable or increasing social mortality differences in old age. Increasing mortality differences in old age have only been found by Otterblad Olausson (1991). This study, like other studies, finds convergence for men but a different pattern for women. The analysis is based on a sample of Swedish persons who were economically active when they were in their working ages. Due to the selection out of

the labor force before retirement, which is stronger among manual workers, the socioeconomic mortality differences in old age may be underestimated (ibid.:438).

Recent results by Huisman et al. (2004) using an international comparison also show that relative mortality differences by education and housing tenure did not decline with age for women in some countries. This may be because converging factors apply more to men than to women. Increasing health differences have been found by Ross and Wu who find that the impact of education on objective health increases with age while it exerts a stable impact on self-rated health, and by Lynch (2003:24) who finds an increasing impact of education on health.

Empirical evidence for increasing differences may be so rare because observable differences are the net result of many different (converging and diverging) factors and possibly also of measurement errors. In this section empirical findings for stable mortality differences are taken as indicators that mortality differences do not necessarily have to decline. Then the list of arguments from above will be continued with arguments supporting not only stable differences but a mortality divergence.

Stable mortality differences are found by Huisman et al. (2003:871) and Fox et al. (1985). Many other studies talk about “persisting” differences or they say that the association between class and health remains “continuous” into old age. This can be misleading because they show declining differences but stress the fact that there are still mortality differences in old age (e.g., Berkman and Gurland 1998:81; Thorslund and Lundberg 1994:67). The following arguments support increasing mortality differences with age:

5. The impact of past unhealthy experiences, e.g., unhealthy working conditions or smoking, is postponed till older ages.

Most social conditions and behaviors take time before they begin to affect health. Health decline itself is a process where accumulation until death can take many years. Therefore, a time lag between an experienced disadvantage and its effect on health or the time of death is plausible (House et al. 1994; Lauderdale 2001). To my knowledge this postponement is a general assumption for the causality model but the exact time lag of the impact of social disadvantages on mortality has never been measured.

6. Past experiences, e.g., education, accumulate and may interact with other factors, e.g., economic and social capital. The health outcome of this accumulation is incorporated into the “health stock”.

A model for the accumulation of advantages is the so-called “Matthew-effect”. The name goes back to the quotation from Jesus in the Gospel of Matthew.³⁵ Merton (1968) applied this logic to explain unequal developments and increasing inequality over time in the careers of scientists. Since then, this example has been used to illustrate the logic of cumulative advantage. Here Ross and Wu (1996:106) state this logic:

“Increasing inequality in careers may be due to the accumulation of resources. Certain scientists, such as those from prestigious schools, publish more, which leads to better jobs with resources like computers, graduate research assistants, libraries, departmental colleagues, more time to do research, which leads to more grants, publications, citations, and so on in a self-amplifying process [...] Cumulating resources may explain cumulative advantage generally.”

Dannefer elaborated on this principle and described a social theory of cumulative advantage (Dannefer 1987, 2003; more literature in O’Rand 1996b:189). The accumulation of disadvantages has been described by Beckett et al. (2002:194), Lynch (2003) and Ross and Wu (1996:106f) and the cumulative advantage hypothesis is described in Section 3.5.3. This accumulation is regarded as one reason why social mortality differences could increase with age (Mare 1990). This principle has also been called “double jeopardy”, indicating that old age and low social status represent two disadvantages that accumulate (Markides and Black 1996:155). To illustrate this principle Markides and Black have described the differentiation of cohorts with age because of their different pathways where certain events and processes lead individuals in increasingly different directions. Convergence is often found between educational mortality differences with age when taken at a cross section, but within cohorts these differences diverge (Lauderdale 2001:555f). Moreover, accumulation of disadvantages is plausible because indicators for inequality have their highest correlation in the highest and lowest classes.

Accumulation is strengthened by the interaction between subjective and objective problems: poor people have more serious life events (Geyer 1997:38f) which are also a heavier subjective burden for them. Life events again are connected to health (Beckett et

³⁵ “For unto every one that hath shall be given and he shall have abundance; but from him that hath not shall be taken away even that which he hath.”

al. 2002:192). An elaboration of the concept of health relevant accumulation can be found in Mirowsky and Ross (2003). Empirical epidemiological studies addressing a cumulative effect of socioeconomic status are rare; yet Næss et al. (2004) and Singh-Manoux et al. (2004) both find accumulation.

7. Vulnerability increases in old age and makes differential exposures more harmful (House et al. 1994:221).

This argument means that the impact of low class disadvantages increases with age because of more biological variability and vulnerability. While differential exposure may decline with age, the increasing impact due to biological variability and vulnerability may outbalance this with the result that mortality differences increase (House et al. 1994:221; Stronks 1997:80ff).

5.3 Results from the literature for divergence versus convergence

Above I presented different research results, some showing stable results, but most showing decreasing mortality differences with age. Besides that we see that there are plausible arguments both for diverging and converging mortality differences. Knesebeck et al. (2003:1644) assumes that inconsistent results for this research question are due to 1. different measures for predictors or outcome variables, e.g., health versus mortality, 2. consideration of different causes of death and 3. different health care systems in different countries that are more or less effective in reducing health differences between social groups (see also Bowling 2004:439). In their international comparison they find a less consistent pattern between socioeconomic status and health in the USA than in Germany. Moreover these differences decrease with age in the USA but not in Germany.

In the following I will discuss five selected studies in greater detail which not only find a pattern over age using good data but which discuss and try to identify specific factors, including mortality selection, that contribute to the observed pattern.

1. Ferraro and Farmer (1996) test the three alternatives of double jeopardy, aging as a leveler and persistent health inequality between white and black US-Americans with 15-year longitudinal data from the National Health and Nutrition Examination Survey

(NHANES). Double jeopardy in this context means that “persons are faced with a double burden of racial and age discrimination or ‘multiple hazards’” (Ferraro and Farmer 1996:319). The opposite assumption would be the age as leveler hypothesis based on the assumption that “aging brings such basic challenges to health and functional ability that racial inequality is not important” (Ferraro and Farmer 1996:319). The authors apply a method very similar to what I use in the empirical part, namely event-history analysis for the event of death with an interaction between age and a variable for social status, which is race in their case.

With this technique the interaction between age and race shows that mortality differences between white and black decrease with age. Then they apply longitudinal regression to predict different health indicators and they scored the deceased persons as having zero health. This is a questionable representation of death in a model but it allows for an inclusion of deceased persons in a model which shows health differences over age between social groups. This model is much less affected by selective mortality because deceased persons stay in the model. It reveals a significant interaction between age and race which shows that racial health differences are not decreasing but increasing with age.

Besides this, the study gives further support for the double jeopardy hypothesis: first, the black do not only have worse health throughout the observation period, they also have a steeper health decline. Second, among people with a heart condition black people are more likely to be disabled by this condition. While the results for racial differences are only partly comparable to social differences, the important message from the study by Ferraro and Farmer (1996) is that “the hypothesis specified by *aging* as leveler (of individual differences) should be recast as *selective survival* as leveler among populations” (ibid.:325). This is exactly what argument 4 from above suggests.

2. The study by Beckett (2000) is entirely dedicated to the question of whether converging health inequalities in old age are an artifact of mortality selection. She uses a ten-year follow-up from the same data source as Ferraro and Farmer (NHANES). In the third part of her analysis she estimates ordinal logit models to describe the age pattern of educational differences in health, similar to the approach by Ross and Wu (1996). These differences decrease with age. Then she tests whether this result is robust against including decedents in the model and finds that the convergence is not due to mortality selection. When including decedents in a health model, it is necessary to

estimate the health of decedents. The problem is that she uses models for survivors for this prediction which may overestimate the hypothetical health status of the dead persons. This procedure may, just as the method used by Ferraro and Farmer (1996), produce a bias because the hypothetical health of dead persons is fixed (in the study by Ferraro and Farmer) or estimated on a potentially higher level (by Beckett) than it would be without the death. The result can be an artificial health convergence between decedents and survivors because the latter are made more similar to survivors. For this and other criticisms of her study see Noymer (2001) and Lynch (2003:31).

In a reaction to the criticism from Noymer, Beckett and Elliott (2001) modified the strategy. They impose even more health-based selection in the model and do not see more convergence in health differences with age. They conclude again that convergence is not caused by selection.

3. Liang et al. (2002) analyze educational mortality differences over age with a panel study of health and well-being of older adults (60+) in Japan with four waves from 1990 to 1999. Their main finding is an educational mortality crossover for men. They understand this crossover as an extreme case of convergence in old age and as evidence against the cumulative advantage hypothesis. They propose two different explanatory scenarios, both leading to convergence and eventually crossover. First, like the normal selection hypothesis introduced above, less educated people are more likely to die young, leaving a selected group of robust individuals. They additionally describe the unlikely condition that from the more educated persons, a larger proportion survives which “may have a higher burden of disease” (Liang et al. 2002:305). Second, morbidity is much more compressed for the well educated, which would also cause a mortality convergence in old age because in a relatively narrow age range, the well-educated experience high mortality.

It is not plausible that the authors consider a convergence or crossover that is due to mortality selection to be evidence against the cumulative advantage hypothesis. When the observed converging pattern is due to mortality selection it instead supports the hypothesis of accumulation because the idea of mortality selection implies that the real pattern without mortality selection would show increasing differences, stable differences or at least less mortality convergence.

4. Huisman et al. (2003) study socioeconomic differences in morbidity among the elderly in eleven European countries with data from the first wave of the European Community Household Panel from 1994. They use education and income as predictors for self-assessed health, limitations in daily activity and long-term disability. Inequality in morbidity decreases with age for women but not in all countries for men. Substantial differences persist for both sexes even in the highest age group of 80+. In a similar international comparison of the same eleven European countries, Cavelaars et al. (1998) found no convergence of health differences between educational groups. In the study by Huisman et al. (2003) Danish men show the smallest health inequalities in old age. As a general picture educational differences are similar to income differences. The authors give possible explanations for declining health differences: first, income inequalities are slightly smaller in old age which also reduces health differences. Second, income may not be an accurate measure for socioeconomic status in old age.

5. Huisman et al. (2004) published an article a year later that is similar to Huisman et al. (2003) but that looks at social differences in mortality. They use data from mortality registries linked with population census data of eleven countries and regions of Europe. Predictors are education and housing. Mortality differences either did not decrease or hardly decrease in England and Wales for men and in Belgium, Switzerland, Austria and Turin for women. Absolute differences, which they interpret as avoidable numbers of death, increase with age.

What is the reason for different findings in different countries and between men and women? First of all, the authors agree that many factors influence the age pattern of inequalities in mortality and therefore it is no surprise that countries and sexes differ in this age pattern because they differ in some or many of the related factors (ibid.:475). These factors can be: 1. the pattern over age, i.e., the question of whether mortality differences decline with age or not, depends on the level of differences at the starting age. For example, women have a lower overall level of social mortality differences and thus it is less likely that these differences decline with age. 2. "Social inequalities in smoking vary strongly by age group, with larger inequalities observed among younger than among older generations. This age dependency of inequalities in smoking may have influenced the age dependency of inequalities in mortality in many European populations". By that, social differentials in smoking behavior can be responsible for differences in the age pattern of social mortality differences between countries. 3. A

similar argument is proposed for the consumption of alcohol. To the extent that social differences in drinking behavior contribute to social mortality differences, as is the case in middle age especially in the northern countries, declining differences in drinking behavior with increasing age can contribute to lower social mortality differences in old age (ibid.:475). To conclude, depending on the country, these factors may or may not cause a mortality convergence because such behavior is more common in lower status groups (which increases the gradient) and less common among old people (which decreases the gradient).

This chapter has shown that several factors possibly contribute to declining social mortality differences with increasing age. Several other factors may contribute to the persistence of and increase in these differences. The empirical part of this dissertation aims to identify the empirical pattern over age in Denmark and the USA (Section 8.4) and to apply different models to disentangle the factors involved and their relative influence (Sections 8.5 to 8.8). The most important question of whether or not unobserved heterogeneity influences the observed pattern of social mortality differences over age (argument 4) will be addressed in Chapter 9.

Summary of Chapter 5

Chapter 5 addresses the main question for the empirical part of this dissertation: Does the socioeconomic mortality gradient increase or decrease with increasing age? Most previous research finds converging socioeconomic mortality differences with increasing age. This finding has been explained by the following ideas respective arguments: 1. Biological aging works as a leveler, 2. the welfare state reduces old age inequality, 3. the effect of experiences from earlier life fades out, 4. the observed convergence is an artifact of unobserved heterogeneity and mortality selection.

If the last factor has a substantial impact it is possible that the age pattern net of the effect of mortality selection shows constant or increasing social mortality differences. This pattern could be based on the assumption that 1. the effect of past unhealthy experiences is postponed until older ages, 2. there is mutual accumulation of (dis-) advantages in health and social status, 3. increased vulnerability in old age leads to a higher impact of differential exposures.

From the five articles described in detail in Chapter 5 the first concludes that it is selective survival that works as a leveler and not aging (argument 4). The second does not find evidence for mortality selection. The third article diagnoses mortality selection and even a crossover but argues on a different level. The authors do not link this finding to the question of whether actual cumulative mechanisms are hidden by mortality selection. The main contribution of the last two articles is that they show and try to explain international differences concerning the change of the social morbidity and mortality gradient over age. The eleven countries under study include some examples where the gradient does not decrease with age. The aim of this dissertation is to show the change of socioeconomic mortality differences over age in Denmark and the USA and to check which of the mentioned factors influence this pattern.

Chapter 6 Measures

6.1 Measures of predictors

An empirical study of the relationship between health or mortality and socioeconomic status has the task of finding an operationalization of the latter, based on a definition of socioeconomic status or social class. The conceptualization of social class is often rather vague in the literature³⁶. In addition to the definition of socioeconomic status that was proposed in Section 3.8, this section gives an overview of different ways to operationalize this concept.

It is not trivial to remind oneself that measurable items like income and years of schooling are only indicators for the larger background concept of social status (Elkeles and Mielck 1997). These items are either intermediary steps in the causal chain between socioeconomic status and health and mortality or they determine the social status together with other factors. Therefore they can only account for a part of the entire socioeconomic status (Marmot 2000:364). In other words, income, occupation or education each represents a different dimension of socioeconomic status (Kunst et al. 1998a:478). Maybe even socioeconomic status is only a proxy for something that really influences health and mortality and that we do not know yet (Link and Phelan 1995:84). If individual level indicators are used it should be kept in mind that they are derived from larger social and economic processes that shape the distribution of indicators like education, occupation and income. In terms of social inequality and one's position in society, which is always relative to others, the distribution is of major importance for the relation between the independent and dependent variables. Therefore, information on the individual socioeconomic status does not cover every social or economic influence on health (Oakes and Rossi 2003:770).

The discussion about the right indicator or the right set of indicators for social status has not yet come to any fixed conclusion; in fact the debate over socioeconomic status will probably only end when social research ends (Oakes and Rossi 2003:770). But we can be self-critical when we find that research on the measurement of socioeconomic status as such has not increased much in the last decades whereas research that uses existing measures as predictors of health has increased from almost zero in the early 1960s to

³⁶ The problems related to an insufficient conceptualization of social class are discussed in Goldman (2001:7ff).

230 articles per year in recent years. This is the result of an electronic analysis of literature databases by Oakes and Rossi (2003). The authors comment:

“This is not because the SES measurement problems are solved. Rather, it is because few have paid attention to the problem. Almost everyone has put the cart before the horse [...] we believe that correct conventional measures of SES, however well implemented, may be limited indicators of the social and economic forces that affect health” (Oakes and Rossi 2003:771).

Empirically, education, occupation and income are by far the most commonly used indicators. From all the articles in the *American Journal of Epidemiology* (AJE) between 1982 and 1985, 45 percent used education, 22 percent occupation and 15 percent income to measure what is sometimes called “the big four” representing socioeconomic status, namely money, power, prestige, and knowledge (Oakes and Rossi 2003:772). Only a few studies used all three of them (e.g., Sorlie et al. 1995), but there is agreement that only one indicator is not satisfactory (i40 Hummer et al. 1998b:560). This is because different dimensions of socioeconomic status may have different pathways to health for different groups of persons or different ages: individual occupation may be useful to study the economically active people and income or measures on the household level better for inactive people (Chandola et al. 2003b).

If income, education and occupation are considered in their relative importance, two traditions of classification can be found: British researchers (e.g., Goldthorpe 1974) focused more on hierarchical employment relationships, whereas the “American approach” (Oakes and Rossi 2003:772) started from the idea that education is important for getting a job and is like an input into the labor market with the income as the output, the reward from the occupation (see Figure 4.4). Therefore, studies from the United Kingdom more often use occupation as an indicator of socioeconomic status, whereas studies in the USA take education or, if available, income (Davey Smith et al. 1998:153; Kunst et al. 1998b:3).³⁷

Besides theoretical reasoning the availability of data from a large number of persons may be the decisive factor in the choice of variables. In cases where the preferred variables are not available, alternatives like standard of housing can be accepted since it is still a better alternative compared to, e.g., car ownership (Martelin 1994:1275).

Absolute measures describe the effect of a certain resource (e.g., one more year of schooling increases the probability of surviving to age 80 by a certain percentage),

³⁷ For a comprehensive overview of measures of socioeconomic status see Lynch and Kaplan (2000).

whereas, e.g., income quintiles measure the total impact of income because they take existing inequality into account. Therefore they reveal higher differences not only if the impact is higher but also if income is more unequally distributed (Mackenbach and Kunst 1997:759). Mackenbach and Kunst (ibid.:767) formulate three requirements for a good measurement of socioeconomic inequality in health:

“that it reflects the socio-economic dimension to inequalities in health; that it reflects the experience of the entire population (rather than just, say, social classes I and V); and that it be sensitive to changes in the distribution of the population across socio-economic groups”

The last requirement can be fulfilled by using percentiles that change over time. This is done in the case of income measurement in the empirical part of this study and allows to define, e.g., the poorest 10 percent of the population regardless of possible changes of the income distribution over time.

Furthermore the operationalization of socioeconomic status should consider theoretical assumptions about how social status affects health (Lynch and Kaplan 2000:19) that have been discussed in Section 4. 4. 2. Concerning the relation between socioeconomic status and health or mortality the problem arises that the measure for social status can be an outcome of health (Grundy and Holt 2001:895), which relates the question of the right measurement to the discussion about reverse causality in Section 4. 4. 7.

6. 1. 1 Income

Income is a very concrete measure for socioeconomic status. It is in principle easy to measure, but the information may be difficult to get and sometimes biased, depending on the source of the information. Examples for studies with income as an indicator for social status are Menchik (1993), Kawachi et al. (1997), Smith and Kington (1997a) and Kunst et al. (1998b). The measurement of income, and other household-based information, has to address the problem that arises between the individual level and the household level. In cases where individual income is the only available information, large biases may occur because some members of a household may have zero income but live from their partner's large income. In the more common situation where information about the household income is available, it is necessary to adjust for the household size. This sounds trivial but this rule is not always followed (e.g., see Bassuk

et al. 2002:521), maybe due to the lack of information about the household size. The normal way is to divide the household income by a (weighted) number of household members which results in the household equivalent income (Knesebeck et al. 2003). But even then it is not sure that the income is equally distributed in the household. McDonough et al. (1999:19) assume that men get more than women from the available household income.

Huisman et al. (2003:872) argue that income is not a good measure for socioeconomic status in old age. First, to measure the impact of income over age one has to control for the changing income distribution. This can be done by using age specific income percentiles that would account for the different income distribution in old age³⁸. Additionally, this kind of measurement fulfills the requirement that income should be measured in relation to the poverty level (Lynch and Kaplan 2000) and it may be even better because in some cases the official poverty level is different for older ages. However, the assumption that old people need less money is not generally justified (Crystal 1996:391).

Second, unlike a normal middle age working income, the income sources of elderly people may be more diverse and not accurately represented by a broad measure for household income. This, of course, depends on the definition of income in the questionnaire or other data sources. In modern welfare states it is important to include transfer incomes in the definition of income (Steinkamp 1993). Backes et al. (1998:177) mention the following possible components of old age income in Germany: pension, social benefits, employer pension, gains from assets, inheritance, family, reduced prices. Even if the available data about income and the data processing is of good quality it is difficult to compare the role of this indicator in different countries. For example, income is the best health predictor for the elderly in Germany compared to education, occupational status, assets and home ownership, which are not consistently related to health at ages 60 and above (Knesebeck et al. 2003). The association between social status and health is less consistent in the USA and also diminishes with age, which is not the case in Germany. The conclusion that money plays a more important role in Germany than in the USA and that the age pattern of this influence is really different between these two countries is still based on many assumptions, because many unobserved factors may confound this measurement. Avlund et al. (2003) show for Denmark that among different indicators for socioeconomic status in old age, the

³⁸ I have tested this measurement for the empirical analysis, see Section 8.4.

material indicators (income and housing tenure) were much stronger predictors for health than for example education or occupation.

6. 1. 2 Wealth

Wealth is more unequally distributed than income during old age due to accumulation processes and it has an influence on mortality even net of income and education (Hummer et al. 1998b:560). Depending on the exact measurement of income, wealth may also be more important for those elderly that do not only have an income but live from other capital gains that are more difficult to measure. In the prediction of mortality wealth shows a higher gradient than income (Bassuk et al. 2002:530).

If the measurement of both income and wealth is exact, it is astounding how their relative impact on health can be empirically confirmed: Smith and Kington (1997:142) show with data from the Health and Retirement Study (HRS) that \$1 of wealth has 10 percent of the effectiveness of \$1 of income, which, according to the authors, shows the actual interest rate of about 10 percent at which persons can get gains from their capital.

6. 1. 3 Education

Without a doubt education measures a very different dimension of the socioeconomic status, although all dimensions are correlated. Even if this correlation is very high and both measures would describe the social status equally well, the most important differences between financial measures and education is that the latter is normally a time constant variable that describes a formal grade of education acquired some decades ago (in the case of the elderly). In the USA this measure tends to include vocational education in the number of years of schooling, whereas elsewhere rather an ordinal measurement of educational grades, professional grades or a metric scale of number of years of schooling is used (Knesebeck et al. 2003).

The measurement of education relatively early in the life course has important implications for the use and the meaning of this variable, both advantages and disadvantages. It has advantages because it reflects the social status decades ago (Hertzman et al. 1994:84) and in the youth (Davey Smith et al. 2001). This implies that education is robust against the ups and downs of working life and also against reverse causality. Unlike actual cognitive ability, the education of a person as it is usually

measured will hardly be affected by declining health (Preston and Elo 1995:477; Beckett 2000:116). To be precise, it is possible that health affects education, namely health in early adulthood (O’Rand et al. 1999:129; Lynch and Kaplan 2000; Goldman 2001:120ff) but this is less important for the use of this variable for the study of old age. Education has further advantages because it is equally valid for both sexes and for the measurement of groups that may not have a normal income for some periods in their life, e.g., unemployed (Lynch and Kaplan 2000; Preston and Elo 1995:477). Education to a large extent comes before occupation and income both chronologically and causally (Ross and Wu 1996:105), although it is again partly determined by the social background of the family.

The disadvantages of education as an indicator for socioeconomic status partly consist of the same features as described above: If a measure is fixed very early in life it cannot represent the change of status or the current conditions. The social status, level of knowledge and intellectual ability may change over the course of 50 years. The distribution of education for the elderly is skewed, because fewer people born at the beginning of the 20th century had higher education (Huisman et al. 2003). The range of measurable differences for education is not as great as with income implying fewer categories and fewer definable differences between them (Beckett 2000:116; Grundy and Holt 2001:896).

As stated above, the decision for one measure cannot be made with theoretical considerations alone. To unpack socioeconomic status in several indicators will always be safer and better as a first explorative approach in data analysis (Deaton and Paxson 2001). The problem of different points in time where the different indicators are valid (e.g., education, occupation and wealth) is also best addressed if several measures are included. By that, different phases in the life course are considered (Kunst et al. 1999:219). After that, the best indicator can be chosen based on the results obtained from this explorative step.

6. 1. 4 Occupation

Occupation is the third most important measure for social status. For the study of elderly (here defined as persons aged 59 and older) the problem is obvious, that most of them do not work anymore. So the direct influence of occupation on health occurred mostly in the past, although this influence may be very important because it has to do

directly with the body and exposure to the working environment. Some authors prefer occupational status to education and income and there is also empirical evidence for this preference (Chandola et al. 2003b:56), but it applies mostly to younger ages. Huisman et al. (2003) skip this indicator in their study of elderly people because it is much less important than education and income.

Valkonen et al. (1993) is an example of a study that uses occupation and education to define socioeconomic groups for adults. Also for adults, Davey Smith et al. (1998) test whether education or occupational social class is better as a discriminator of socioeconomic differences in mortality and smoking behavior. They conclude that occupational class is better and argue against the interpretation that cultural (education) rather than material resources (occupation) determine social health differences (ibid.:158). Of course, one could argue here if it is correct to interpret occupation as a predominantly material measure (Vågerö and Illsley 1995:220).

Occupation is difficult to measure. Complicated systems with more or less categories have been developed to bring a structure in the diversity of occupations. There are e.g., nine major occupational categories in the 1960 US census (Mare 1990:369) and 501 detailed occupations in the same classifications system from 1990 (Warren and Kuo 2003:326). The Danish register uses categories that were applied during the last Danish census in 1970. In some cases this structure mirrors occupational prestige and thus indirectly also social status. However, it is difficult to make groups of different exposure to health threats at work because the dimension of unhealthy working conditions may not be congruent to the dimension of occupational status or prestige. For example, lower status non-manual workers may have unhealthier working conditions and higher subsequent mortality than skilled manual workers (Kunst 1997:32).

Occupation is also prone to misreporting especially if data is collected after retirement or after death from the death certificate (Breeze 2000:175). Additional complications emerge from frequent job changes. The longest occupation may be the best information to use (Hummer et al. 1998b:566). But for elderly people the last occupation is more commonly recorded which may have a lower status compared to the previous job and therefore may give a wrong picture (Kåreholt 2000:4). Finally, occupation is problematic because some people do not have one, e.g., many women in older cohorts (Ross and Wu 1996).

Concerning the empirical results for the importance of occupation in mortality studies there is evidence that occupation is related to income and education (Warren and Kuo

2003:501). More specifically, several studies find that occupation does not have much of an effect on health net of education and income (Sorlie et al. 1995; Bassuk et al. 2002:522; Warren and Kuo 2003:326).

6. 1. 5 Classification of women

A special problem poses the classification of women in a classificatory system for socioeconomic status. Especially in older cohorts, women have much less attachment to the labor market, so that a classification based on occupation and also income may fail to represent a women's status correctly. Smaller socioeconomic health and mortality differences for women can partly be explained by the assumption that most classifications do not fit women as well as they fit men (Kåreholt 2000:20). The old-fashioned way to classify women is to use the husband's characteristic (e.g., occupation) because his status is supposed to be most influential for the whole household. But of course this logic does not apply anymore in cases when women have their own occupational career or they are not married. In more recent studies the husband's education is taken only to impute missing information for the wife (Grundy and Holt 2001:896). Goldman (2001b) tests the difference between the old and the new "individualistic" approach to the measurement of women's socioeconomic status and does not find large differences between the two approaches, the individualistic approach being slightly better (ibid.) However, Bassuk et al. (2002) conclude that the old classification has more disadvantages.

6. 1. 6 Social capital

In Bourdieu's theoretical framework social capital is one of three sorts of capital, besides economic and cultural capital. This concept is more amorphous and more difficult to operationalize. Good and still practicable measures are e.g., number of friends, trust in other people, quality of neighborhood, and family ties. Bourdieu describes social capital also as prestige and network of relationships. This means that social capital is not an individual characteristic but the quality of a group, a community or society. The consequence for the measurement would be that the respondent alone, i.e., on the individual level, can not give sufficient information on his or her social capital.

Among the variables for social capital those mentioned above are only rarely used in epidemiological studies. It is also justified to subsume the number of children and marital status into this category. Marital status is a measure of position within the social structure (Goldman 2001b) and even if being widowed, single or childless is not a disadvantage in social status as such, it often has negative consequences, especially for elderly people. Marital status and having children is equally important for providing emotional well-being and help through social ties, resources that belong to the category of social capital and have been found to promote health (Seeman 1993; Kawachi and Berkman 2000; Grundy and Sloggett 2003; Tucker et al. 2004).

All of the mentioned indicators have certain measurement problems because generally the availability of information about persons is limited and the concept of social class or socioeconomic status is rather vaguely described. It is not arbitrarily defined but still there is no single correct and perfect theoretical basis or measure for it. Thus all possible measures for socioeconomic status have their problems (Huisman et al. 2003; Martelin 1994) and they tend to be more problematic in old age (Grundy and Holt 2001:896). One basic assumption that I propose that makes this concept easier to operationalize is that there are really different statuses in society in the sense that they can be understood hierarchically. This plausible assumption asks for quantifiable descriptors that fit with the logic of *more* or *less* or even a dose-response principle. This is also the basic feature of the notion “capital” used to describe social positions. In most cases the use of the term class includes this hierarchy but it also refers to a subjective and symbolic dimension³⁹ (Vågerö and Illsley 1995:234) that in principle allows for horizontal characteristics in the description of the social structure. It is difficult to say what the term class adds to the analysis of social health differences. If we want to show social health gradients empirically, it is unavoidable to base the measurement on a single dimension because the logic of a gradient or a hierarchy is one-dimensional at least in principle. If all indicators would perfectly correlate, any class measure would do, but in reality where just mid-level or strong correlations exist a reasonable and justified choice of what measures to use has to be made (Vågerö and Illsley 1995:234).

³⁹ The symbolic dimension of a class was first conceptualized by Marx with his notion of a “Klasse für sich” as being different from a “Klasse an sich” (Marx 1867:789ff).

6. 1. 7 Health behavior

There is a group of variables for which it is difficult to say if they are predictor or outcome variables, namely variables for behavior, health behavior and proximate indicators for the latter e.g., Body-Mass-Index (BMI). In terms of the causality chain between social status and mortality they are intermediary variables. In many studies they are used as control variables in order to see if, for example, smoking explains mortality differences between income groups or not. From a theoretical point of view they do not belong to the concept of social status but it can be justified to treat them as part of the larger concept of class (Goldman 2001b:131). Bourdieu illustrates that the habitus and the life-style are class-specific. Empirical results support the view that e.g., lower classes care less about their health and engage more in health-damaging behavior like smoking and drinking (Lynch et al. 1997). But it would be exaggerated to say that the social status determines if someone smokes or not. Thus, social status and behavior can not easily be separated but these concepts should not be equated either (Davey Smith et al. 1994:446). A reasonable strategy is probably to analyze the impact of control variables like drinking and smoking to see how tight their relation to social status is and to see if the relation between status and mortality persists after controlling for these variables. But caution must be exercised when interpreting the results: if health behavior is partly a result of the social situation, controlling for behavior may hide some impact of social status because the impact of status is “controlled away” (Martelin 1996:127; Hoover 2003:123).

To conclude this section about the measurement of predictors for health and mortality it should be mentioned that another way to address the problem of measuring socioeconomic status is possible but has not been applied in this analysis. It is possible to construct an index for socioeconomic status, where different dimensions and the individual levels in these dimensions contribute (additively) to a total index score for each individual. The dimensions used in such an index can be weighted according to the results of a factor analysis that can show their single contributions to socioeconomic mortality differences⁴⁰. This procedure and the index are able to summarize the complicated multidimensional concept of socioeconomic status and its measurement. Under certain conditions it is possible to use an index for the comparison of social

⁴⁰ For an example of an index for socioeconomic status in an epidemiological study, see Tello et al. 2005.

mortality differences between groups or countries. The problem is that the relative contribution of each single measure for social status has to be explored statistically and then it has to be fixed for the construction of the index. Differences of the relative importance of single dimensions between groups or changes over age or time can not be considered. Moreover, an index-based analysis gives much less insight into the causality of socioeconomic differences in health and mortality than do many different flexible multivariate models wherein it is possible to test the impact of separated factors (see Section 3. 8).

6. 2 Measures of outcome

Another group of variables that can be understood both as control variables and as outcome variables are health variables. On the one hand, health and mortality are closely related. One extreme but justified point of view is to consider mortality as a health indicator (see section 4.3). On the other hand, there is normally not enough information about death to understand the process that leads to this event. So the health trajectory including both worsening and improving health status is the more complicated and the more interesting process to analyze compared to the one event of death. However, this event has other advantages concerning exactitude and objectivity that has been discussed in Section 4.3. In the empirical part of this dissertation there will be a discussion of how and why health measures are used as control variables in the analysis of socioeconomic mortality differentials. Measurement issues are discussed in the following.

6. 2. 1 Health

There are objective and subjective health measures. Objective health measures are e.g., limitations in Activity of Daily Living (ADL) and the Body Mass Index (BMI). There is much agreement on what ADLs to measure (Katz et al. 1983; Rogers et al. 1990; Crimmins et al. 1994), e.g., preparing a meal, getting out of bed, using the telephone, etc., but not on how to scale it. The following questions remain: is having difficulty enough to elicit a point on the score for ADL? Is the need for human or technical assistance a good criteria? And is it most important that people can manage to practice these activities at all, regardless of the kind and the amount of help they need? A common pair for the definition of a limitation in ADL is having difficulties but not

using human help to do it. The use of help again depends on the social situation. This shows that it is difficult to measure ADL independent of social status, but this can not be discussed in detail here (Jette 1994:937).⁴¹

Activities of daily living and the Body Mass Index are relatively objective measures in the sense that e.g., a true value for BMI is an objective measure of the body. However, if the questions for ADL or BMI in a survey are answered by the respondent, he or she can still determine if going to the toilet is a problem or not and can still intentionally or unintentionally give a wrong body weight (Himes 2000:77). Thus, to some extent this measure also belongs to the subjective measures.

The most common subjective health measure is self-rated health, e.g., the question of, “how do you rate your general health?” It is probably so widely used because it is easy to ask and has many convincing theoretical and empirical features. The fact as such that this measure is widely used is an advantage because of higher comparability between studies. Unfortunately the answer scheme differs between studies. Sometimes five categories are used, e.g., in the Health and Retirement Study (HRS) or by Helweg-Larsen et al. (2003). Even if the same number of categories is used, the value labels are not always the same. HRS uses “excellent, very good, good, fair and poor” and e.g., the categories in Knesebeck et al. (2003) range from “bad” to “very good”. Sometimes only four categories are applied (e.g., very good, good, fair, poor). Methodological studies show that the scaling of categories has a significant impact on the results (Blinkert 1978) which may not affect the comparison among respondents in one study but certainly the comparison between studies.

Generally self-rated health is considered to be a very good if not the best single health measure and predictor for mortality (Idler and Benyamini 1997). Self-rated health measures health, as defined by the World Health Organization, rather comprehensively as not only the absence of disease but also as a state of well-being (Ross and Wu 1996:109). Ferraro and Farmer (1999) claim that self-rated health is a better mortality predictor than health evaluated by a physician maybe because individuals are better informed about their health than anyone else (Mackenbach et al. 2002). Self-rated health depends more on current conditions: Arber and Ginn (1993:43) shows that income, car ownership and housing ownership (current conditions) are more related to the subjective health among the elderly whereas past occupational status is more related to disability. This measure also catches undiagnosed diseases and co-morbidity, which are the rule

⁴¹ For more information about Activities of Daily Living (ADL) see Reuben et al. (1992).

and not the exception in old age, when people have on average three conditions (Idler and Benyamini 1997:28). In a status with co-morbidity “the whole is more than the sum of the parts” (Idler and Benyamini 1997:28).

Many elements are included when a person judges his or her own health status: health trajectory, family history, severity of current illnesses, possible symptoms of undiagnosed illnesses and social relations (Idler and Benyamini 1997; Helweg-Larsen et al. 2003).

The measurement of self-rated health may be biased because people compare themselves with their friends (Smith and Kington 1997:122). This “compared” self-assessment may still be important information but of course the trend over age and other comparisons of the results are difficult to interpret if the assessment is implicitly based on a comparison because the reference is unknown. Jylhä (1994) offers an interesting citation from an interview situation where the respondent is asked explicitly to compare her own health status with others. It is obvious that collecting valid information about self-rated health can be very difficult.

“Interviewer: Now, if you compare your health with that of other people you know of your own age, is your health better, about the same or worse?

Respondent (85 year-old woman): [...] they’ve taken one leg away, you can’t really say you’re healthy.

Interviewer: [...] Is it hard for you to compare your own health with that of other people of your own age, would you say it is ...

Respondent: Well most of them are dead, aren’t they? (laughter)

Interviewer: So you’re in a better shape than they are

Respondent: Well I suppose you can’t say it is poor, except that I’ve lost my eyesight. Did I tell you that I can hardly see anything?” (Jylhä 1994:988)

Finally, it is not clear if self-rated health is biased by gender, age or social class. Arber and Ginn (1993:37) find that in old age gender differences in self-rated health are smaller than with functional measures of health. This may either be because women underestimate or men overestimate their health. Van Doorslaer and Gerdtham (2003:1625) offer two findings: first, among men and women with the same mortality risk, men tend to rate their health better and, second, older people tend to rate their health better than younger people with the same level of mortality. Liang et al. (2005)

show that self-rated health only slightly decreases between age 60 and 85 and even seems to improve after age 85. But there are many different results:

“Roughly one-third of the studies reviewed showed that older people evaluated their health more positively, roughly one-third showed that older people evaluated their health more negatively, and one-third showed no relationship between SRH and age” (Helweg-Larsen et al. 2003:1238).

It can be assumed that elderly people are more likely to include non-physical aspects in their assessment (Helweg-Larsen et al. 2003:1242).

Concerning the class bias it is likely that self-assessment is influenced by one's social situation. There are findings showing that the more objective the health measure is, the more ill the poor are, i.e., the greater are social health differences (Thorslund and Lundberg 1994:66; Kåreholt 2000:2,6). One possible explanation is that subjective measures are biased because lower class persons judge their health better than people in higher classes because lower class people are not so aware and sensitive towards physical problems and do not worry so much about health problems. Stronks (1997:171) finds the opposite result: objective measures reveal less differences. That would mean that lower class persons underestimate their health status compared to upper class persons. This could be because people include their overall happiness and satisfaction in their health judgment. Maybe these biases balance each other out. It is plausible that questions like “Has a doctor told you that you have hypertension?” measures a specific health dimension, namely communication with a doctor and the ability to remember health problems. This has been found to be lower for poor classes (Smith and Kington 1997:127). Besides that there is scant evidence that self-rated health is class-biased (Power et al. 1998; van Doorslaer and Gerdtham 2003:1628; Arber and Ginn 1993:37).

To conclude, self-rated health measures something more and something less than a physician's evaluation (Idler and Benyamini 1997) and possibly both self-rated health and objective measures are biased to some extent (Adda et al. 2003:61). Thus, a combination of both may be the best solution. For any health measure it is important to have several categories for health measurement because a dichotomized measure for a health outcome can produce convergence over age as an artifact of floor and ceiling effects that reduce variation in health (Ross and Wu 1996:115).

6. 2. 2 Mortality

The basic event for the measurement of mortality is death. For a single person, this information only allows us to define two different statuses, alive and dead, as well as the age of death. For a sample or a population, a life table can be constructed allowing computing survival chances and mortality risks based on the aggregated age pattern of all recorded deaths. Mortality can be expressed with different measures, which are all in principle results of a life table: life-expectancy and mortality rates with the related measures probability of dying or chances of survival in a certain age interval.

Life-expectancy is a good measure because it is concrete, e.g., we can say that a newborn in a certain country has a life expectancy of 76.3 years or a 70-year old person a remaining life expectancy of 13 years. Despite this superficial simplicity, life expectancy is a very complex measure that is based on strong assumptions and it is not really accurate (Müller 1993:73). When life expectancy for a newborn is computed from a life table the assumption is that this child will pass through all ages experiencing the same age-specific mortality risks as all different ages experience it under current conditions. The child's life course can only be represented by a synthetic cohort, where current conditions are extrapolated in the future, although this child in 60 years will experience different health and mortality risks from today's 60-year old persons. Some studies therefore use different mortality measures in some cases in order to compare the results for e.g., probability of death and life-expectancy (e.g., Lauderdale 2001:552).

Death rates are a measure of mortality where the number of occurrences (deaths) is divided by the number of people exposed to this risk. In the case of a general mortality rate all persons are exposed to the risk of dying and this exposure is not measured as the number of persons but as person-months or person-years. A mortality rate of 0.15 with respect to the age interval from age 60 to age 70 means that out of 1000 persons 150 die between age 60 and 70.

This study compares groups, i.e., it focuses on mortality differences. These differences can be expressed as absolute differences, e.g., between age 60 and 70 out of 1000 rich persons, 100 persons die and out of 1000 poor persons 200 persons die. More interesting for the analysis of mortality differences over age are relative mortality differences, i.e., relative mortality risks or rate ratios. To compute such a rate ratio the mortality rate for the poor of 0.2 from above is divided by the rate for the rich (0.1) and

the rate ratio (RR) would be 2. This means that poor people have a two-fold higher mortality than rich people.

The statistical models used for the analysis in the empirical part of this dissertation are based on a slightly more complicated representation of mortality. A Gompertz function is computed that shows the increase of mortality rates over age. Then the difference between the curve for the rich and the curve for the poor people is expressed with a rate ratio just as in the example above (see description in method section). These rate ratios basically describe mortality differences between groups. Thus strictly speaking they only show an association between belonging to a certain group and mortality but often rate ratios are defined as having predictive power in the sense that belonging to the richer group decreases mortality by e.g., 50 percent (Marmot and Shipley 1996).

Naturally, dividing up the population into groups has a major impact on these rate ratios. Comparing two extremes gives higher rate ratios than comparing two halves (Marmot and Shipley 1996). Moreover, rate ratios have the problem of not accounting for group size and for changing group sizes (Anand et al. 2001:55). Thus, we may observe that mortality or health differences between educational groups have increased during the last decades, but that the share of lower educated persons is smaller, which could be important for the overall judgment of the observed inequality in this case (Marmot 1994:198).

There are other measures for mortality differences that will not be used in this study because the way these measures take group size into account can not solve the methodological problems that occur because of changing group sizes due to mortality selection. Changing group size due to a changing distribution of education or income over time and in different cohorts is not the main focus of this study. An example for such a measure is the Relative Index of Inequality (RII). This is a regression-based index that compares the mortality rate at the lower end of the income distribution, for example, with the mortality rate at the higher end of the distribution, just as a normal rate ratio would do. But it takes into account the group size and the position of the group relative to all other groups by regressing the mortality rate on a measure of its relative position, namely the proportion of the population that has a higher income. A description of this and other sophisticated measures including a comparison of their respective outputs can be found in Mackenbach and Kunst (1997).

There are also measures that are used to integrate the information about mortality and health. Active life-expectancy is the number of years that a person can expect to live

without disability. For example, Hayward et al. (1998:206) show that if men at age 70 in the USA have a remaining life expectancy of 11.2 years, these years can be predicted to be divided up into 9.7 active and 1.5 inactive years. For women it would be 14.9 years overall remaining life expectancy, of which 11.9 years were active and 3.0 inactive years. Generally years of healthy life is a good measure because it considers mortality and morbidity simultaneously (Diehr et al. 1999), but on the other hand as an alternative outcome measure it is unable to exactly explain the relation between health trajectory and time of death. This combined measure depends on definitions and many possible biases that mortality as pure information does not suffer from. Finally, the use of such integrated measures requires both a high quantity and a high quality of health measures.

Summary of Chapter 6

This chapter about the measurement describes the well-known difficulty in finding a perfect and universally accepted measure for socioeconomic status. This multidimensional concept is still vague and can at best only be approximated by good indicators, but it cannot be measured perfectly. The discussion of the measures of income, wealth, education, occupation and social capital includes a description of different traditions in using these concepts, their specific problems and their relative importance. Different indicators for the measurement of health are proposed. Especially the widely used measure “self-rated health” shows some very positive features, e.g., the inclusion of personal feelings and sensations, which have to be balanced with possible class or gender biases. These biases have neither been consistently confirmed nor rejected in the literature. The measurement of mortality is less affected by subjectivity. The differences between life expectancy, healthy life expectancy, and mortality rates (the latter which is used as the outcome variable in the empirical part) are briefly described.

Chapter 7 Data and methods

7.1 The Health and Retirement Study

The data for the USA come from the Health and Retirement Study (HRS) and a sub-study, the study of Assets and Health Dynamics among the oldest old (AHEAD). These are two representative studies conducted by the Institute of Social Research (ISR), University of Michigan, and supported by the National Institute on Aging (NIA). They were started separately in 1992 and 1993 respectively and then combined in 1998, with a follow-up every second year (Soldo et al. 1997). Since HRS focuses on retirement ages and AHEAD on the ages 70+, I merged them with the help of data sets prepared by RAND (for information, see <http://hrsonline.isr.umich.edu>). This resulted in a sample of 9,376 persons born before 1934 (aged 59 to 107) and surveyed from 1992 to 2000, with 2,608 deaths during observation. I excluded black persons from the analysis because the small number of them in the sample would only show general racial mortality differences, which is not the purpose of this study. By the same token, it would not be possible to analyze their specific age trajectory of social mortality differences.

Institutionalized persons were already excluded in the original baseline sample but surveyed in the institution during the follow-up interviews. This may cause a bias (Arber and Ginn 1993:35). For example, kinless or single persons, persons with poor health and women are more likely to be in a nursing home and thus they are more likely to be underrepresented in the sample (Soldo et al. 1997:4; Grundy and Sloggett 2003:936). Huisman et al. (2003) tested this bias and found that samples that exclude institutionalized persons underestimate socioeconomic health differences in older ages. The HRS sample, however, only omits them at baseline but follows them in the institutions. This can be seen in the following table.

Table 7.1: Proportion of elderly living in a nursing home, USA and HRS Dataset

Age group	Population (USA)		HRS, wave 1998		HRS, wave 2000	
	Men	Women	Men	Women	Men	Women
65-74	1.0	1.1	0.4	0.4	0.8	0.8
75-84	3.1	5.1	2.0	3.0	2.1	4.1
85+	11.7	21.7	10.0	15.3	12.3	21.6

Source: National Nursing Home Survey, my own calculations

The proportion living in a nursing home at older ages is indeed substantial, but the fact that HRS respondents are followed at the time they move into institutions makes the percentages in the HRS sample also very high at least in the later waves. From wave to wave (in the above examples after 6 and 8 years of observation) the numbers come closer to the overall numbers for the USA. This is still not really representative and in principle there is a bias, i.e., an under-representation of females, ill persons, single persons and probably also of persons with low socioeconomic status. This bias may produce an underestimation of socioeconomic mortality differences in old age. But in the HRS wave of the year 2000 the differences are negligible. It is unlikely that my results are biased substantially by this slight under-representation.⁴²

The original HRS sampling procedure over-samples the population of Florida and blacks. I do not use special weights to compensate for this because first, black people are excluded from the analysis anyway and second, there is no reason to believe that the population of Florida is systematically different from the U.S. population beyond the characteristics that the numerous variables in the models control for. When the research focus is on multivariate modeling with relative risks as the outcome and not on descriptive statistics for the finite population, weights are shown to have a small impact on the results (Hoem 1989). If one uses the correct model for the data, i.e., a model that is specified correctly, the use of weights is not necessary (Campbell and Alwin 1996:45). Helweg-Larsen et al. (2003:1240) suggest not using weights if the correction is less than 1 percent.

Missing values were almost entirely imputed when the data were prepared and combined by RAND. Rules for and results of this imputation can be found on the homepage cited above. Data coming directly from HRS datasets where information for single waves were missing, it was imputed in a straightforward manner using information from the previous wave, or, if applicable, the mean of two waves.

Concerning the health status and the mortality of the respondents, Soldo et al. (1997:14) find that the baseline health profile of the AHEAD sample is consistent with cross-sectional data from larger national surveys.

⁴² This is also claimed by Hurd et al. (2001:6) and shown by Adams et al. (2003a:18), who compare the level of mortality between official life tables for the U.S. and the AHEAD sample. Between the first two waves mortality in the AHEAD sample is lower than in the U.S., maybe due to under-representation of the institutionalized, but in subsequent waves it is about the same.

7.2 The Danish Demographic Database (Danish registers)

The Danish data are register data from the Danish Demographic Database that was implemented in the year 2000. It is maintained by Statistics Denmark, the central statistical office of Denmark (www.dst.dk). It combines data from different registers from 1980 onwards. Registers cover the entire Danish population, providing annual information. The information from these different sources can be linked by an individual personal identification number.⁴³ The dataset includes 1,090,897 women and 938,427 men, thus representing a total of 2,029,324 persons aged 59 years or older. They are observed from 1980 to 2002. This means that the birth cohorts 1874 to 1933 are followed over 23 years and the cohorts from 1934 to 1941 for a shorter period (starting from the lower horizontal line in a Lexis-diagram).

Unlike the HRS data the Danish data include the whole population, i.e., also the institutionalized persons. However, the information about which persons move or live into an institution is not readily available in the Danish registers. Thus, this variable cannot be included in the analysis. To give an idea of the share of institutionalized persons in Denmark: the strategy in Denmark since the 1980s has been to stay “as long as possible in your own home”. This meant that from 1990 to 1997 about 19 percent of persons aged 67+ receive home help. In the same period, the proportion of persons aged 80+ living in service flats or institutions decreased from 24.6 to 22.6 percent (Kvist 1999:248).

A problematic feature of both the HRS data and the Danish data is right censoring and left truncation. Right censoring means that not all persons are observed until they die which is not problematic for the statistical models used in this study. More serious is the limitation because of left truncation, which means that relative to the defined starting age of the observation (age 59), we start to observe some individuals at much higher ages. The consequence is that we do not know how many persons of the older cohorts already died before observation. This also implies that the sample in the case of the HRS data is not a real random sample, because persons with a high mortality risk and other characteristics that are associated with higher mortality are more likely to be already dead than persons with a lower mortality risk (Klein 1993a:105). This problem will be discussed in detail in Chapter 9.

⁴³ For detailed information about the Danish Demographic Database, see Petersen (2000).

7.3 Variables in the HRS data

The variables allow a detailed and time varying measurement of socioeconomic status, health status and some control variables. Except for education, having children, and parents' mean age at death, all variables in the following list are included as time-varying variables.

Education is measured in years of education (levels: 0-7, 8-15, 16+).

Wealth includes all assets of the household in which the person lives (bank account, real estate, shareholdings, etc.) and is measured on three levels: lowest quartile, second lowest quartile and above median wealth.

Income is the net annual household income divided by a weighted number of persons living in the household (net equivalent income). The weight is 1 for the first person and 0.7 for all other persons in the household. Income is measured on the same three levels as wealth: lowest quartile (below \$8,839 per year, which is \$737 per month), second lowest quartile (up to \$14,732) and above median income.

Parents' mean age at death is the mean age at death of both parents (levels: below 75 and 76+). Under certain conditions, it captures the genetic constitution that is transferred from parents to their children; see discussion section.

Children is an indicator of whether the subject has any children of his or her own (levels: yes, no). This variable measures one aspect of social capital, i.e., whether a child is likely to look after the old person. However, it can not just be treated as a social status variable as it measures many different things. For example, having numerous children can be an indicator of low social status and may be a cause for higher mortality whereas having no children may be a consequence of bad health (Doblhammer 2000).

Labor force status. This variable differentiates between working, being retired/disabled and not being in the labor force. While the labor force status is to a large extent a function of age and health (which I control for by using other variables), it additionally captures information on social status and every-day life – information that is predicting mortality.

Marital status is not a social status measure in a strict sense but it is related to socioeconomic status. Firstly, marital status depends partly on social status, e.g., persons with a low social status are more likely to live alone (Goldman et al. 1995, O'Rand 1996b). Secondly, marital status has a high impact on social status in the sense that divorce or widowhood is often followed by a loss of economic and/or social capital. Moreover, marital status has an influence on health and mortality independent of socioeconomic status. In this analysis, I combine divorced with never married persons because these are both very small groups that show a similar level of mortality.

Health behavior is an additive index focusing on three items that have shown to be important correlates of health: 1. physical activity (the persons were asked if they engage in vigorous physical activity once a week or more), 2. ex-smoker, 3. current smoker. From the resulting four different categories of this score (-1 to 2), the last two (with the worst health behavior) have been collapsed into one category because both of them were small.

Self-rated health. The question on self-rated health is posed with the five traditional categories: excellent, very good, good, fair, poor. I merged the first two categories, because their meanings differ only slightly.

Objective health is another additive index that includes four items: 1. being in a hospital for more than 10 days per year, 2. limitations in activities of daily living (ADL), 3. a body mass index (BMI) at baseline < 21.4 for men and < 19.5 for women (=lowest decile), 4. loss of weight of more than 10 percent of the body weight between two waves (=two years). From the resulting five different categories of this score (0 to 4), the last two (with the worst objective health) have been collapsed into one category because both of them were very small. In principle, these items are reliable and objective descriptors of health status, but the information as such is based on the respondents' answers and not on objective measurements or tests.

It was necessary to construct indices for health behavior and objective health because, given the limited number of cases, all interesting variables for the dimension of health would be too numerous to be included into the model.

Some variables have been tested in previous models and then skipped because they did not show significant results after controlling for other variables. The omitted variables

are: occupational group, parents' education, going to church, children living nearby, drinking, high BMI, a gain in body weight of 10 percent and more.

To illustrate the frequencies for all categories of the variables, Table 7.2 shows the number of person-years separately for men and women. In event-history analysis, persons-years is more exact information than number of cases. Table 7.3 shows the corresponding person-years for the different age groups used in the analysis.

Table 7.2: Person-years for the categories of the variables

		male	female
parents' age at death	-75	11,645	15,564
	76+	11,876	13,751
education	0-7	2,456	2,688
	8-15	16,312	23,249
	16+	4,752	3,377
children	No	1,872	3,073
	Yes	21,649	26,241
labor force status	Work	6,442	3,895
	retired/disabled	16,954	18,591
	not in lab force	124	6,829
marital status	married	19,263	13,953
	widowed	2,846	12,830
	divorced/never	1,413	2,532
wealth (percentiles)	0-25	4,600	8,721
	25-50	5,452	7,337
	50-100	13,469	13,256
income (percentiles)	0-25	8,339	14,480
	25-50	5,940	6,645
	50-100	9,242	8,189
health behavior (act,exsmoke,smoke)	good	2,466	4,987
	Fair	9,356	15,558
	poor	11,699	8,770
self rated health	excel/very good	8,694	10,638
	good	7,536	8,854
	Fair	4,823	6,372
	poor	2,468	3,450
objective health (hospital,adl,thin,loss)	excel/very good	16,663	18,603
	good	5,095	7,713
	Fair	1,450	2,502
	poor	313	497
sum over all categories of one variable:		23,521	29,315

Table 7.3: Person-years by age group

Age	male	female
59-69	9,612	8,044
70-79	8,668	11,775
80-89	4,618	7,934
90+	623	1,562
sum:	23,521	29,315

7.4 Variables in the Danish data

The variables in the Danish Demographic Database that have been chosen for this analysis are similar to the variables in the HRS dataset. The use of a category for “not known” for most of the variables follows the principle that it is better to have such a category in a model than to drop all persons where only some information is missing. Generally, the register data have a very low percentage of missing data. Where missing data can be imputed without strong assumptions, e.g., when income is missing only for some years, this has been done. It follows a description of the variables, their measurement and the treatment of missing values and other exceptions (for all levels of all variables, see Table 8.2.)

Education is measured in years of schooling (levels: -7,-8,-9,-10, and 11+). The variable for education is problematic because it is only available for persons born after 1920. As a consequence, there is no information about education for persons above age 82. The information was collected for all persons in the last Danish census in 1970 and later considered to be unreliable for persons above age 50 at the time of the census. These persons are coded as *education not known*, thus mainly old persons are included in this category. Tests of models without education and models restricted to persons younger than age 83 show that the information systematically missing for education neither changes the results for the other variables nor for the other analytical steps in this study. This is mainly because education has no great importance as a social predictor for mortality. Thus, it would not be justified to exclude either the variables for education or to exclude all persons born in 1920 or earlier from the analysis.

Wealth includes all assets of the household in which the person lives (bank account, real estate, etc.) and is measured on four levels representing the four wealth quartiles.

This is one level more than in the HRS data. There the number of categories should be kept as small as possible because of the small sample. Shareholdings are included in the measurement of wealth since 1995.

Income is the individual gross annual income. It is measured in six categories based on percentiles (0-10, 10-25, 25-50, 50-75, 75-90, 100). Since the absolute amount of Danish Kroner that define these groups changed considerably from 1980 to 2002, I use adjusted income limits for each year. For 1980 the respective income limits in Danish Kroner are: 11,097; 27,941; 36,871; 75,680; and 117,667. These amounts increase gradually till 2002 where it is 77,793; 101,292; 118,560; 176,289; and 262,610. If 77,793 DKK is divided by 7.42 to get the amount in EURO in 2002 and again divided by 12 for the months of a year, the poorest 10 percent of the Danish population above age 59 had a maximum gross income of 874 EURO per month.

Children is an indicator that, unlike in the HRS data, does not only mean that the persons have children of their own but that children are currently living in the household (levels: yes, no).

Source of main income in the Danish data is comparable to *labor force status* in the HRS data. This variable shows whether the persons surveyed receive the main part of their income from a normal pension, an early retirement pension, normal wages or salaries, income from a business of their own, or from transfer income (e.g., unemployment or sickness benefits).

Marital status is measured in the traditional four categories: married, divorced, widowed, and never married.

Days in hospital is the only health measure that I obtained from the Danish register system. This variable can only be an approximation yet when compared to the detailed information about different aspects of health in the U.S. data, it shows surprisingly similar results. Thus, the analysis that uses health as a variable will be repeated for Denmark; this in order to compare the results with the U.S. results, but in other cases the health analysis will be limited to the USA because the meaning of the variable days in hospital is different from a real health measure. The variable measures the days spent in hospital in one year on six levels.

I also performed a test where days in hospital are used in the HRS data. In most cases this health indicator showed similar results to the more precise health

indicators that are available in this dataset, namely self-rated health and objective health measures (results not shown).

Occupation was excluded in the analysis of the HRS data mainly because of the need to limit the number of variables and categories due to insufficient sample size. For the Danish data, there is no such need, thus the impact of occupation on mortality is shown in the first models. But to keep the analysis for the two countries comparable it is excluded in other models. Occupation was measured in the 1970 population census in Denmark, which was the last census before the census system was replaced by the register system. The categories are based on the distinction between skilled and unskilled on the one side, and manual and non-manual on the other (see Table 8.2).⁴⁴

Type of dwelling provides a distinction between different types of housing that may have an impact on health and mortality beyond the overall living standard. It also provides some information about the degree of urbanization. A *single house with garden* is the typical suburban type of dwelling that most elderly people in Denmark live in. An *apartment* is typical for larger cities whereas *country house* stands for a rural area. Inhabitants of nursing homes which have high mortality are placed in the category *shared dwelling*. But since this group of persons is not exclusively in this category, a further interpretation of this group in terms of institutionalization is not possible.

Square meters is the size of the dwelling per person, i.e. divided by the number of persons living in the dwelling.

For both countries, age is controlled for by using a Gompertz-shaped baseline risk function. Sex is controlled for by running separate models for each sex. I also checked and found that period or cohort effects do not bias the presented results in either country.

For the HRS data Table 7.2 and Table 7.3 show how many person-years are lived in each category of the variables and in each age group. Besides the descriptive information about the sample, such information is important for judging the level of significance which in some cases is not satisfactory. For the Danish data this information is not shown because the overall case number is very high and all

⁴⁴ By that the system of occupational classifications in my data set is not easily comparable to the International Standard Industrial Classification of all Economic Activities (ISIC) or to the International Standard Classification of Occupations (ISCO).

categories include enough person-years. This is also the reason why I will use a category “not known” for some variables for Denmark but not for the USA. For the Danish register data minimal effort was spent imputing information in the rare cases where it was missing in the Danish register system. In the HRS case it is much more important to impute information in order to keep as many respondents as possible in the analysis.

7.5 Method

Here only the basic method of event-history analysis will be introduced. Specific model applications and other methods will be presented in Chapter 9 when statistical problems of unobserved heterogeneity are discussed and analyzed. Theoretical advances in life-course research and the increasing attention that is paid to age and aging (Elder and Caspi 1990) have required a technique such as event-history analysis. It is perhaps the most important methodological innovation besides multi-level-modelling (van Wissen and Dykstra 1999:271).⁴⁵

I apply event-history analysis with a model for the force of mortality as the outcome variable. The force of mortality, $\mu(x)$, is a hazard rate that can be understood as an instantaneous death rate at age x (Horiuchi and Wilmoth 1998:394). The models include a baseline for the basic time variable age that has a Gompertz shape, and coefficients for the multiplicative impact of categorical variables on the baseline risk of dying. The risks are computed as rates, based on occurrences (deaths) and exposures (person-months) for specific combinations of variable levels. The results are displayed as rate ratios.

I used Stata 8 and 9 as well as aML 2.04. Among the advanced software packages Stata can best handle the problem of left-truncation. I used aML only to compare and check the results from Stata. In aML the models have a piecewise linear baseline risk. The baseline for age covers the age range from 59 to the highest age, whereas the observation period is only 8 years, namely from 1992 to 2000, or 22 years (1980-2002) for the Danish data. Thus, the cohorts are not real cohorts but partly synthetic ones in the sense that in spite of the longitudinal data, no individual in the data set is really observed from age 59 to ages above 67 (USA) or to ages above 81 (Denmark).

As described above, the analysis of mortality is limited by the fact that persons who entered the study after age 59 are left-truncated, i.e., only the period at risk after the

⁴⁵ A textbook for this technique is Blossfeld and Rohwer 1995 and examples for its application to the analysis of mortality are, e.g., Mare 1990, Crimmins et al. 1994 and Klein 1999.

respondents have entered the sample can be considered. Stata allows us to take into account left-truncated cases by distinguishing between “time under risk” and “time under observation”. Here “time under risk” starts at age 59 for all persons and “time under observation” starts at the individual age of entry. Stata computes the individual probability that a person survives from age 59 to the age of entry based on the known characteristics of this person and other persons who are observed from age 59 onwards. Different models are used in different steps to draw conclusions about the causal relationships between the predictor variables and impact they exert on mortality. Relative mortality rates are computed using different interactions. The general formula for the model is:

$$\mu_i(t | X_{1i}, \dots, X_{ki}) = \mu_0(t) \exp\left(\sum_k \beta_k X_{ki}(t)\right) \quad (1)$$

μ_i is the individual force of mortality that depends on time (t), which is age in the model, and the individual characteristics that may or may not change with time. These characteristics are represented by X_{ki} , which is the value of the k th covariate for the i th individual. This hazard is equal to the baseline hazard μ_0 , which is the hazard of a standard individual that has, e.g., value 0 for all covariates in the model, times the effect of the individual variable combination, $\exp(\dots)$. The parameters β_k denote the effect of a unit change in the covariates X_k on the logarithm of the hazard holding constant all other covariates. $\exp(\beta_k)$ for the categorical variables expresses the hazard of the group that has $X_k = 1$ as a proportion of the baseline hazard. For example, if $\exp(\beta_k) = 1.15$, the group for which $X_k = 1$ has 15 percent higher mortality than the group that has $X_k = 0$ (Mare 1990:371).

The baseline hazard is specified as a Gompertz function. The Gompertz function has been used since 1825 and a modification by Makeham has also been used since 1860. Many biological theories of aging support the idea that the age pattern of human mortality follows a Gompertz curve (Manton et al. 1986:638). Some of these theories are discussed in Strehler (1977) and Economos (1982). The Gompertz and the Weibull hazard function are most often used to represent the age pattern of mortality and

senescence. The Gompertz is especially common for describing adult human mortality.⁴⁶

The mortality hazard following the Gompertz model is defined as follows:

$$\mu(t) = \alpha e^{\beta t} \quad (2)$$

The shape parameter β is supposed to express biological senescence, and relates the mortality increase to age (t) and is the percent increase in the mortality rate per year. The scale parameter α is a constant over age and expresses the environmental mortality factors, e.g., stress (Manton and Vaupel 1992:2). Among others, two theoretical assumptions of the Gompertz model for mortality are 1. as age increases, “the ability to resist environmental stress declines and mortality increases” and 2. “physiological damage accumulates as a linear function of time” (ibid.:2f). Thatcher et al. (1998:50) compare the fit of six different models to the best available empirical mortality data and find that the Gompertz model overestimates mortality in very high ages (above age 95). They find the same phenomenon for the Weibull distribution that would be an alternative in Stata. The distributions that the authors find to be more exact in very high ages (logistic or quadratic) are not readily available in Stata. I consider the Gompertz model to fit my data sufficiently well, especially for the analysis of mortality differences, with the hazard ratio as the outcome where the total fit of the baseline function is less important.

⁴⁶ For an empirical evaluation of different parametric models, see Thatcher et al. (1998) and Kannisto (1999).

Summary of Chapter 7

The data for the USA come from the Health and Retirement Study (HRS) and include 9,376 persons aged 59+ followed from 1992 to 2000. The advantage of this data is a very comprehensive set of variables collected in interviews. For Denmark I use data from the Danish Demographic Database that is based on the Danish register system. Registers include the whole population officially living in Denmark. The link between different registers by a personal identification number allows for the collection of variables that are as detailed and valuable as in the HRS data, but for many more persons. The concrete variables in both datasets and all their categories are described in detail including a frequency table for the data from the USA where some cells do not have satisfactory numbers of cases for a detailed analysis. The general method of event-history analysis is described as the main method applied in Chapter 8 as well as a general notation of the hazard regression model based on a Gompertz baseline for the mortality increase with age.

Chapter 8 Results on socioeconomic mortality differences (discussion included)

8.1 The impact of socioeconomic factors on old age mortality

8.1.1 USA

Table 8.1 shows the relative risks of dying. The underlying models are without interactions and separate for men and women. The baseline mortality risk that increases with age following a Gompertz-curve is not shown. The baseline risk roughly doubles with every ten years of age. Model 1 only contains the univariate results of each variable separately. The first category of each variable is the reference category that always has a value of 1. The categories below this reference category show relative mortality risks, relative to the reference category. All variables show the expected association with mortality and all of them are significant, except for marital status for women and having children for men. Surprisingly, men with 8 to 15 years of education do not have a significantly lower mortality compared to those with 0 to 7 years.⁴⁷

In Model 2, all variables that directly or indirectly describe socioeconomic status are included simultaneously while health variables are excluded. Naturally, the mortality differences between the levels of most of the variables become smaller than they were in Model 1. For example, in Model 1 the highest educated men have a 41 percent lower mortality than lowest educated men. This advantage is neutralized in Model 2, where the highest group has 99 percent of the mortality risk of the lowest group, which is a clearly insignificant difference. The differences between many categories become smaller in Model 2 as compared to Model 1 because other variables are controlled for. This means that only those educational mortality differences remain that can be found within one category of the other variables for which we are controlling. If wealth and income are controlled for, higher education no longer has a positive separate impact. Men with an intermediate level of education even have a significantly higher mortality than lower educated men (see discussion below). Having children reduces mortality for

⁴⁷ When the results in Table 8.1 and Table 8.2 are presented, it is not possible or necessary to mention all possible arguments, explanations, and the related literature for all covariates included. It is advantageous to use these covariates because the more variables that are controlled for, the more exactly the main variables like income and education can be interpreted. However, to integrate all the available knowledge about all possible factors would be a different, independent research undertaking.

women but not for men. Further, the retired, the disabled, and persons who are not in the labor force have a higher mortality than those who still work.

Table 8.1: Event history models of socioeconomic predictors for mortality, USA

		MODEL 1		MODEL 2		MODEL 3	
		male	female	male	female	male	female
parents' age at death	-75	1	1			1	1
	76+	0.86 ***	0.77 ***			0.92	0.87 **
education	0-7	1	1	1	1	1	1
	8-15	0.94	0.78 ***	1.20 **	0.92	1.37 ***	1.03
	16+	0.59 ***	0.63 (***)	0.99	0.86	1.31 (**)	0.94
children	no	1	1	1	1	1	1
	yes	0.93	0.83 **	0.98	0.85 **	0.99	0.87 *
labor force status	work	1	1	1	1	1	1
	retired/disabled	2.48 ***	3.36 ***	2.24 ***	3.02 ***	1.54 ***	2.17 ***
	not in labforce	3.17 ***	1.83 ***	2.54 ***	1.63 **	1.97 **	1.20
marital status	married	1	1	1	1	1	1
	widowed	1.05	1.10	0.95	0.90	1.01	0.91
	divorced/never	1.46 ***	1.17	1.25 **	0.80 *	1.22 *	0.77 **
wealth (percentiles)	0-25 (poor)	1	1	1	1	1	1
	25-50	0.88 *	0.71 ***	0.92	0.78 ***	1.05	0.91
	50-100	0.54 ***	0.57 (***)	0.65 ***	0.72 (***)	0.87 (*)	0.90
income (percentiles)	0-25 (poor)	1	1	1	1	1	1
	25-50	0.75 ***	0.60 ***	0.86 **	0.67 ***	0.95	0.75 ***
	50-100	0.52 ***	0.54 (***)	0.72 (***)	0.65 (***)	0.82 (**)	0.74 (***)
health behavior (act,exsmoke,smoke)	good	1	1			1	1
	fair	2.21 ***	3.34 ***			1.73 ***	2.40 ***
	poor	4.38 ***	4.62 (***)			2.78 (***)	2.95 (***)
self rated health	excel/very good	1	1			1	1
	good	1.58 ***	1.65 ***			1.32 ***	1.44 ***
	fair	2.60 ***	2.68 ***			1.85 (***)	1.92 (***)
	poor	6.11 ***	4.52 ***			3.38 ***	2.6 (***)
objective health (Hospital,adl,thin,loss)	excel/very good	1	1			1	1
	good	2.08 ***	1.76 ***			1.36 ***	1.22 ***
	fair	3.56 (***)	3.43 ***			1.74 (***)	1.98 ***
	poor	5.03 (***)	4.77 (***)			2.27 (***)	2.39 (***)

* : p<0.1; ** : p<0.05; *** : p<0.01

Stars in brackets mean that the value for the rate ratio is significantly different from 1 but not from the previous variable level.⁴⁸

Model 1 contains the univariate results of each variable separately,

Model 2 is multivariate including indicators for SES,

Model 3 adds the health variables and parents' age at death to Model 2.

Widows do not display a significantly different mortality from married persons. Men who are divorced or who have never married have a higher mortality whereas women in the same group have a lower one. Interestingly, the relative mortality risk of divorced or never married women turned from an insignificantly higher mortality according to the

⁴⁸ For an interesting historical analysis of traditions of reporting significance levels in sociology, see Leahey 2005.

univariate results of Model 1 to a significantly lower mortality risk in Model 2 (see discussion). Finally, income and wealth have a strong diminishing impact on mortality. One intermediate step between Model 2 and 3 is not shown here: it adds only health behavior to the socioeconomic variables and shows that the measured aspects of health behavior (physical activity, being an ex-smoker, and being a smoker) change the coefficients only slightly. They do not remove the significance of any socioeconomic variables. This means that socioeconomic mortality differences to a large extent cannot be explained by physical activity or smoking.

Model 3 is the full model, where the three health variables and also the parents' mean age at death are added. Controlling for health means that we see the remaining impact of socioeconomic status on the transition from a given health status to death. This perspective will be developed further in Section 8.4. Technically, controlling for health means controlling for an intermediate step in the causality chain from social status via health to mortality. This is problematic because of the risk of "controlling away" social differences, since health is already correlated with social status (Martelin 1996:127; Hoover 2003:123). But as a single model among others it helps us gain insight into the interplay between social status, health, and mortality by comparing different models.

A high parents' mean age at death significantly reduces the mortality of women. This supports the assumption that common family factors (genes or acquired characteristics) contribute to longevity. Parents' education included in the model as an indicator of their social status does not change the impact of their age at death (results not shown). Thus, the factors that are passed on from one generation to the next seem to be genes or those family characteristics that are not closely correlated with education.

In the full model, wealth is no longer significant but most of the other socioeconomic mortality predictors still are. This indicates that the transition from a given health status to death is also influenced by socioeconomic status. This interim finding will be further analyzed in Section 8.5 where interactions between health and income are presented. In the modeling of complex processes like those between socioeconomic status, health, and mortality, it is likely that some of the variables are intermediate variables for others and that they are not independent from each other. In this study, I find the highest correlations between wealth and income ($r= 0.47$), and between wealth and education ($r= 0.40$). As to the health variables, there are very low correlations between objective health and health behavior (men: 0.09, women 0.16), low correlations between health

behavior and self-rated health (0.22 and 0.14) and strong correlations between self-rated health and objective health (0.39 and 0.44).

For social status as for health, it is clear that multiple interrelated dimensions have to be measured. This is justified as long as the different variables reveal interesting differences in the results of the model (showing that they do in fact represent different dimensions), and as long as these results are interpreted with caution. There will be no further discussion of the results of Table 8.1. here, but there will be in the next section about Denmark in order to compare and discuss the results from both countries together. However, one feature seems to be specific to the USA, namely the excess mortality of middle educated men. This finding is surprising, but it has also been observed elsewhere (e.g. Liang et al. 2002), and has been interpreted as an educational mortality crossover due to selective mortality (ibid.:305). The authors suggest that low educated persons have a higher mortality which leads to a selected and strong group of survivors. These remaining low educated persons are more selected than the high educated persons with lower mortality. The latter may additionally be able to postpone the onset of disease and then later have a higher mortality than low educated groups⁴⁹. Hurd et al. (2001:8) also find higher mortality for middle educated men in the AHEAD sample which is part of the same data set that I use. They say that they do not have a better explanation than mortality selection for this.

An alternative explanation is that, holding income constant in the model, higher education means that the aforementioned education is not translated into higher income. This could be because the person never obtained a job that matches the educational level or he lost his job and thus experienced downward mobility, a move that may have been health-related. This interpretation is supported by the fact that the excess mortality for middle educated men concentrates on lower income and poorer health groups (results not shown). Given that the excess mortality of middle educated men is combined with low rather than with high income, it is not likely that this phenomenon reflects the health-damaging stress of upward mobility. One possible conclusion is that education as a measurement of socioeconomic status, besides having several advantages, has the disadvantage of being too stable across the life course. However, this explanation does not reveal why this pattern is found only for men, but perhaps it is because education has more of an impact for women than for men (Lauderdale 2001), which can be seen from the results showing a small but positive impact of higher education for women.

⁴⁹ This mechanism has been explained in Section 5. 1.

8.1.2 Denmark

Table 8.2: Event history models of socioeconomic predictors for mortality, Denmark

		MODEL 1		MODEL 2		MODEL 3	
		male	female	male	female	male	Female
education	-7	1	1	1	1	1	1
	-8	0.99	0.99	1.04	1.01	1.01	0.98
	-9	0.98	0.91	1.08	0.96	1.04	0.96
	-10	0.80	0.79	0.98	0.88	0.98	0.91
	11+	0.73	0.78	0.92	0.87	0.95	0.93
	not known	1.11	0.95	1.19	1.04	1.09	0.97
children	No	1	1	1	1	1	1
	Yes	0.70	0.87	0.72	0.73	0.76	0.76
main income	pension	1	1	1	1	1	1
	early pension	1.02	0.99	0.93	0.89	1.11	1.12
	wages	0.82	0.82	0.84	0.77	1.08	1.01
	business income	0.74	0.84	0.82	0.90	0.98	1.04
	transfer income	1.55	1.51	1.20	1.18	1.22	1.31
	not known	1.88	2.44	1.06	1.01	1.17	1.14
marital status	married	1	1	1	1	1	1
	divorced	1.51	1.38	1.50	2.46	1.26	1.94
	widowed	1.23	1.18	1.33	2.22	1.19	1.88
	never married	1.34	1.23	1.23	2.16	1.23	1.89
occupation	unskilled manual helper	1	1				
	skilled manual non manual	0.97	0.75				
	self-employed	0.95	0.91				
	not known	0.79	0.82				
		0.80	0.87				
		1.08	0.97				
wealth (percentiles)	0-25 (poor)	1	1	1	1	1	1
	25-50	1.08	0.98	1.03	0.97	0.96	0.95
	50-75	0.96	0.89	1.09	1.05	1.06	1.06
	75-100	0.77	0.76	1.04	1.09	1.08	1.15
	not known	1.29	2.73	1.19	2.25	1.17	1.99
income (percentiles)	0-10 (poor)	1	1	1	1	1	1
	10-25	0.59	0.64	0.64	0.69	0.62	0.63
	25-50	0.33	0.32	0.32	0.26	0.36	0.28
	50-75	0.36	0.39	0.36	0.31	0.41	0.32
	75-90	0.32	0.36	0.33	0.28	0.38	0.28
	90-100	0.25	0.32	0.28	0.23	0.32	0.24
	not known	11.26	13.69	9.15	5.60	3.02	2.40
days in hospital	0-3	1	1			1	1
	4-7	3.00	2.76			2.97	2.81
	8-14	3.71	3.22			3.63	3.28
	15-30	6.47	5.11			6.21	5.16
	31-61	13.24	9.75			12.05	9.35
	62+	28.68	22.11			23.50	17.63
dwelling	single house	1	1				
	apartment	1.34	1.21				
	terraced house	1.26	1.24				
	country house	0.88	0.97				
	shared dwelling	2.47	2.76				
	other/not known	7.23	8.57				
square meters	0-29	1	1				
	30-59	0.79	0.68				
	60-79	0.72	0.61				
	80+	0.65	0.53				
	not known	4.22	7.54				

The level of significance is not shown for the Danish results because with about 1 million cases for each sex, virtually all differences larger than 1 or 2 percent are statistically significant. Besides that, there is no fundamental meaning of significance in this case because I do not observe a sample but rather the whole population at older ages. These results, taken from the Danish population above age 58, are real and exact results from the Danish population and therefore do not need confidence intervals.

Table 8.2 presents the relative risks of dying for Denmark in the same way as Table 8.1 did for the USA. As in Table 8.1 the model on the left in Table 8.2 (Model 1) shows the univariate results for each variable separately. Concerning education, there are not many differences in mortality between the different lower educated groups. Those with 11 or more years of schooling have a mortality that is about 25 percent lower than for those with up to 7 years. Having children in the household seems to be more beneficial to men than to women, maybe because elderly men receive help from their children more so than elderly women. The variable source of income reveals, as expected, that those who still work have a lower mortality, but this difference disappears when health is controlled for, as in Model 3. Receiving transfer income is combined with higher mortality but this disadvantage also gets smaller when health is controlled for. Marital status shows the normal pattern: married persons have the lowest mortality, followed by widowed persons, for whom living without a partner seems to be less dangerous than for never married persons, and especially so for the divorced, who have the highest mortality because their single status is associated with a greater number of personal problems and an abrupt decline in the social network (see discussion below). As to occupation, mortality declines for the higher occupational status. The group of male helpers is negligibly small with 0.06 percent of all men and does not have as significant a mortality advantage as female helpers compared to the reference category of unskilled manual workers.

The wealth quartiles show a lower mortality only for the wealthiest quartile, in contrast to the USA results where already the second quartile has a lower mortality than the poorest. The opposite is true for income in Denmark: here, one has to look at the lower end of income distribution to find significant mortality differences. From the 25th percentile upwards there are no longer any large mortality differences. This is also different from the USA where, at least for men, mortality differences are still large between the second quartile and the persons above the median. The interpretation is that because in Denmark the level of income is high and income is more equally distributed than in the USA, there is a smaller fraction of persons, about 25 percent, that have financial problems serious enough to affect health and mortality. The disadvantaged group in Denmark is also smaller because medical services in Denmark rely less on individual income than in the USA. The variable for days spent in hospital shows a very

steep mortality gradient where even within the period of one week in hospital there are extreme mortality differences.

Compared to the reference category for dwelling, that is “single house with garden,” those living in an apartment or a terraced house have a higher mortality. The countryside is combined with lower mortality which also has been found elsewhere (Menchik 1993:434). Shared dwelling is combined with very high mortality. As mentioned above, this is probably due to the fact that many nursing home residents are in this category. The size of the dwelling also shows a clear mortality gradient where larger apartments or houses are associated with lower mortality.

Model 2 includes a number of variables for socioeconomic status that were also used to analyze the HRS data. Some major effects of these control variables on the hazard ratios will be described here. The two variables that describe the housing situation are not included in order to keep the model comparable to the HRS model. The mortality difference of about 25 percent between the highest and lowest educated persons in Model 1 reduces to about 10 percent when income and wealth are controlled for. This is similar to the results for the USA and shows that the univariate impact of education on mortality is due to the fact that higher educated persons have better jobs and a higher income. When the latter variables are controlled for, education has much less of an own impact on mortality. Some impact remains, possibly because people of higher education have knowledge and behavior conducive to better health.

The disadvantage combined with getting transfer income is reduced by more than half if financial variables are controlled for and the higher mortality of the persons where the main source of income is unknown is also neutralized. In Model 2 there is a surprising change of the results for marital status: the disadvantage of all single women compared to married women steeply increased after controlling for the financial variables. I can not offer a valid explanation for this effect, but it is at least a possible and logical conclusion from the modeling procedure that in Denmark single women in all three groups (divorced, widowed, and never married) have a relatively wealthy status, so that they only have a mortality about 25 percent higher than that of the married women in Model 1. When income and wealth are controlled for, this positive effect cannot hide the real disadvantage any longer, the latter which appears to be much higher than for men. This more than twofold mortality is partly due to a worse health status because in Model 3, which controls for health, this disadvantage declines.

Unlike in the HRS results, the advantage of being wealthier disappears if income is controlled for, which means that it is income rather than wealth that is important for health and mortality. If wealth does not translate into income it may even have a slightly negative impact, since the rate ratios are well above 1 for the wealthier groups. Finally, it is impressive how robust the hazard ratios for income are against the inclusion of control variables: the gradient stays basically the same in all three models.

The differences between different kinds of dwellings do not change when control variables are added to the model (results not shown), thus the differences seem to be caused by the kind of dwelling, really, and not just by related differences in social status or health. The opposite is true for the clear mortality gradient that exists between different sizes of dwellings: this gradient disappears when social variables are controlling for. Thus, in a univariate model, square meters are only an indicator for social status and do not affect health and mortality on their own.

Model 3 includes days spent in hospital as a control for health. This further slightly reduces some hazard ratios but has the most significant effect on the hazard ratio of those who still work compared to pensioners. In Models 1 and 2 active persons have a lower mortality but in Model 3 it turns out that this can be entirely explained by a better health status.

The surprising result that single women in the USA have fewer disadvantages than men and, conversely, that single Danish women have more disadvantages than men would need a study of its own focused on this topic. Here and in Section 8.2 only tentative explanations can be offered. The finding that the relative mortality risk of divorced or never married women turned from an insignificantly higher mortality according to the univariate results of Model 1 for the USA, to a significantly lower mortality risk in Model 2 may be due to an under-representation of institutionalized unhealthy women in the sample. But it more likely shows a real disadvantage for married women. The scope of my presentation does not allow for a detailed discussion of the underlying reasons associated with this. But the fact that the sex difference emerges only after controlling for income and wealth may indicate that married women profit from higher material resources. Besides, they do not have an advantage or may even have a disadvantage when married net of the other factors in my analysis. Grundy and Slogett (2003:940) argued that women experience less of a disadvantage of being single than men because they engage less in unhealthy behavior when in this situation (Johnson et al. 2000), and are more likely to support their singlehood with social networks (Goldman et al. 1995;

Brockmann and Klein 2004:579). In addition, they may even suffer in marriage, where they are likely to be the younger and healthier partner whose role it is to care for the ill spouse (Beckett et al. 2002). This explanation fits for the results of the USA. Given opposite results for Denmark, namely a larger disadvantage of single women compared to single men, it is questionable if this explanation holds. I do not know if gender situations in the two countries are really very different, or if other unknown factors are responsible for these differences between the USA and Denmark. Other differences between the genders and the two countries concerning marital status will be shown in Section 8. 2, that presents the interaction between marital status and age.

Besides the numerous findings and considerations that have been presented and that could further be mentioned here, the main finding from this analytical step is that income for Denmark and income and wealth for the USA are the most important socioeconomic predictors for mortality. This is because it shows the steepest gradient and still does so in models where many other covariates are included. Thus, in most parts of this study I will concentrate on income as an indicator for socioeconomic status; this is because a choice for one dimension is necessary for the application and presentation of some analytical procedures that could not be done with a multivariate design, and because this variable has a much greater influence on mortality than all other variables. In fact, for both countries educational mortality differences decreased a lot after financial variables were included in the model whereas mortality differences between income and wealth groups remained relatively stable (Table 8.1 and Table 8.2). Concerning the bias due to reverse causality from health to socioeconomic status discussed in Section 4. 4. 7, income may have a disadvantage compared to education because the income level is possibly more affected by health problems than the formal level of education. But in spite of this possible advantage of education compared to income, education is not a better choice as an indicator for social status because the results only show very small mortality differences that can be attributed to educational differences. Moreover, the advantage of education being a constant value over the life course implies the disadvantage of being unchangeable in cases where the social situation changes and where income would reflect the new social status because income can change not only because of bad health but also because of a decline in social status. My findings suggest that higher income, as a consequence of higher education, has a much stronger direct impact on mortality than education. This is different from results

by Smith (2003 and 2004). He finds that financial variables only have a small impact on the onset of disease, whereas education is important for new health events. I see three possible explanations for this inconsistency between these different findings: first, there are differences between pre-retirement ages as analyzed by Smith (e.g. 2003:22), and mostly retirement ages analyzed here. Second, since my multivariate analysis concentrated on the event of death, it cannot be excluded that there are differences between the predictors of health and the predictors of mortality. Third, there may be differences between the predictors of the onset of disease and the predictors of overall health status. There are reasons to believe that controlling for baseline health status hides the influence of socioeconomic status on mortality. When initial health status is controlled for, only subsequent health changes and their predictors are considered. So the fact is ignored that the baseline health status is already, among other things, the result of socioeconomic status (Martelin 1996:127; Hoover 2003:123). There are different findings supporting my results: Davey Smith et al. (1998) find that educational mortality differences disappear after controlling for occupational social class and Menchik (1993) shows with data for older men from the USA that controlled for income, the effect of education greatly diminishes. House and Zimmer (2002) also find with USA data that income is much more important than education. However, education still has some impact on the onset of disease. Goldman et al. (1995:1721) summarize such findings when they write:

“an interesting and consistent finding from several U.S. studies is that educational attainment appears to have a greater effect on health at younger ages than older ages and is often not a significant predictor for old-age mortality (e.g., Kitagawa and Hauser [1973], Menchik [1993] [...])”

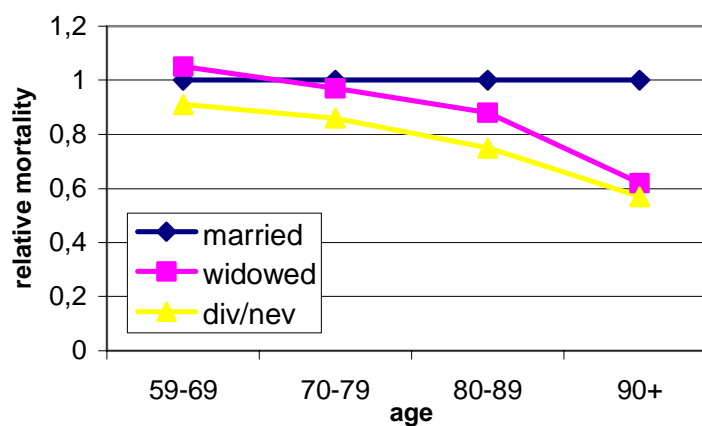
To conclude, education may be a necessary condition, but not a sufficient one, for low mortality. That education only reduces mortality when combined with high income (Kunst et al. 1998b) will be shown in Section 8.3 regarding the interaction between income and education.

The results in Table 8.1 and Table 8.2 are based on the assumption that the relative risks below or above 1 reduce or increase mortality as a multiplicative factor over the whole age range and apply to all levels of all other variables. This assumption is too simplistic. Therefore, the interactions in the following sections will give a more accurate picture of the influence of selected variables.

8.2 Interaction between marital status and age

To give more information about the finding from the previous section – that single Danish women have a much higher mortality than married women – and why these results are so different from the USA, this section presents interaction models between marital status and age. These models also include other socioeconomic covariates but concentrate on the interaction between two factors. I analyze how the impact of marital status on mortality changes with age. This is done by including dummy variables in the model that represent the different marital statuses in four different age groups. The following figures represent this interaction by showing married persons as the reference group that equals 1 in all age groups. Colored lines below this reference line indicate lower mortality than married persons and above the reference line they express higher mortality. Figure 1.1 shows a clear interaction, i.e., the relationship between the three marital statuses is different for different ages.

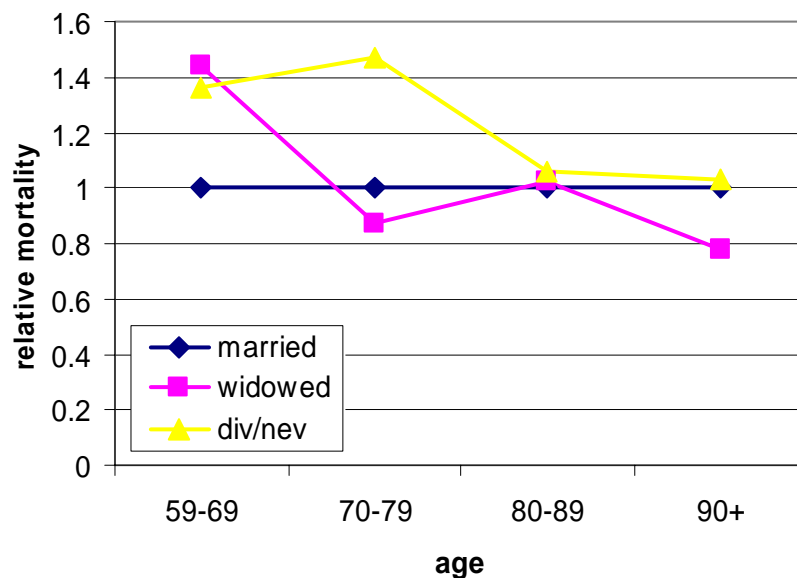
Figure 8.1: Female mortality with interaction between age and marital status, USA (married=1)



Single women in the USA have increasingly lower mortality than married women when we go from age 59 to age 90+. However, only the two data points for the age group 90+ are significantly lower than 1 at the 95 percent level. Perhaps being single is not the advantage as such, but rather that being married at high ages is less of an advantage because, as suggested above, a married women above age 80 is likely to have an older husband that she has to take care of.

Figure 8.2 shows the same interaction for men. The relative mortality level of single persons decreases with age but for men this decrease happens on a higher level relative to married persons: single men have a mortality advantage but they lose it at higher ages whereas single women become more and more advantaged compared to married women as they get older. Here again, the analysis is hampered by a low level of statistical significance: only the data point for divorced or never married men at ages 70 to 79 is significantly different from 1.

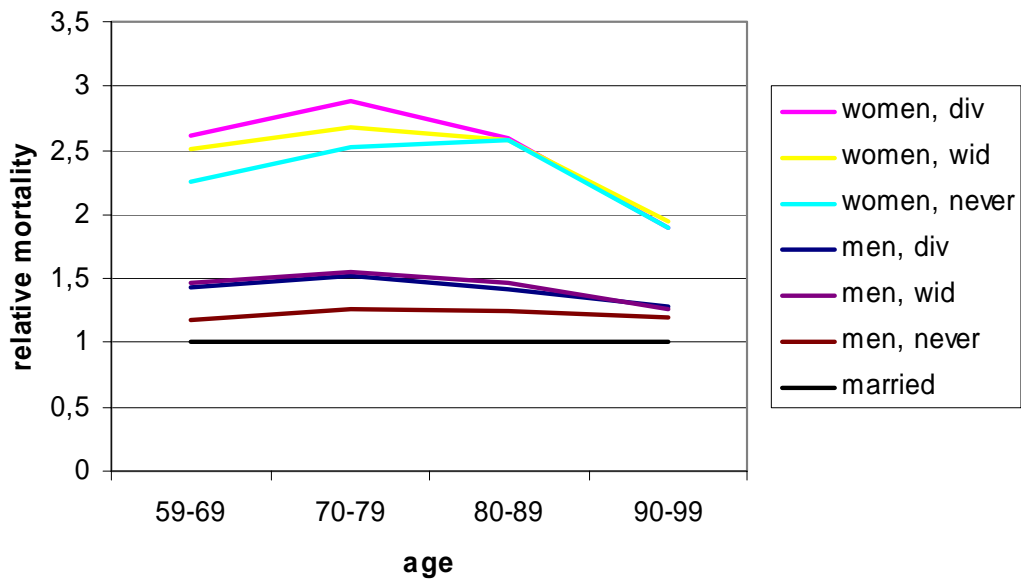
Figure 8.2: Male mortality with interaction between age and marital status, USA (married=1)



The overall result for the USA is that single women have fewer disadvantages than single men and that the age pattern differs between genders.

The next figure, Figure 8.3, shows the same analysis for Denmark. Given the larger number of cases it is possible to keep never married and divorced persons separate.

Figure 8.3: Mortality for men and women with interaction between age and marital status, Denmark (married=1 for both sexes)



Note: Figure 8.3 combines the information for men and women into one figure because the lines are at a very different level and do not hinder each other. Moreover, due to the different mortality levels, this figure also uses a different scaling on the y-axis compared to Figure 8.1 and Figure 8.2.

Mortality differences between different marital statuses converge with increasing age for both sexes with the opposite relationship between men and women compared to the USA: single women are more disadvantaged than single men. Thus the above argument that married women in old age suffer more than married men because they have to help their older husband does not hold for Denmark. Maybe there is less of a burden of care in Denmark because such work is done by public services. But this explanation cannot fully explain the differences between the USA and Denmark because then there should be very similar mortality differences for men and women and not an advantage for men. The overall message from Figure 8.3 for Denmark is that, unlike in the USA, women profit much more from being married than men. As mentioned in Section 4.4.2.5 research findings for this question are ambiguous and do not help to judge the present findings.

8.3 Interaction between income and education

The results in Table 8.1 and Table 8.2 suggest that once income is controlled for, educational differences in mortality are only small. This picture may be overly simplistic. It is worth looking at the interaction between these two predictors.

Figure 8.4: Female mortality with interaction between education and income, USA (low income/low education=1)

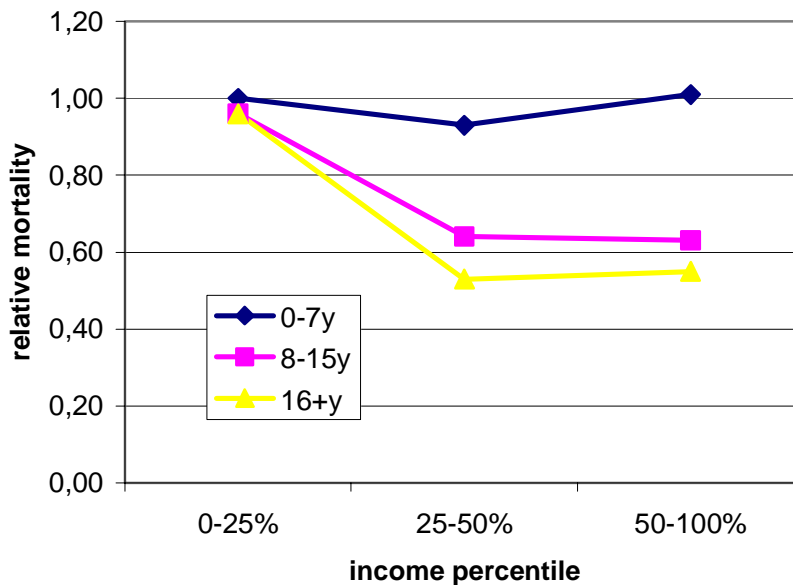


Figure 8.4 shows the mortality level for all possible combinations of three income levels with three educational levels relative to the reference category, which is comprised of poor persons with low income (=1). For women, more income is only beneficial when combined with middle or higher education. The line for lower educated women does not go down for higher income groups. Besides that, higher education is beneficial only in combination with wealth of at least an average level because there are almost no mortality differences between educational groups when people are poor. When wealth instead of income is used in such a graph, the result is very similar. This means that beyond the result of Model 2 where the financial variables removed the positive influence of higher education, for women, these two different resources have a complementary impact on mortality, i.e., both are necessary to have a mortality advantage.

In Figure 8.4, only the data points below the 0.8 line are significantly lower than 1 on the 95 percent level. This means that the decrease of the pink and yellow line (for persons with middle and high education) is significant, but their distance to the upper line for low educated persons is not. This is because the rare combination of low education and high income only shows 78 person-years and the confidence intervals of educational groups in the middle and on the right-hand side of the graph overlap. This shows again that the overall level of significance is comparatively low due to an insufficient sample size. But the pattern of the significant differences nevertheless supports the above interpretation, which claims that significant mortality advantages due to high income only occur among more educated groups.

The corresponding graph for males in the USA and in Denmark is not shown or discussed here. This is because in Denmark, there is no similar pattern and men in the USA do not show this interaction. The figure for men in the USA is dominated by the surprising excess mortality of men with intermediate education (figure not shown). Since I do not know the reason for this mortality pattern (see discussion), an interaction between education and wealth for men would not provide deeper insights.

The relatively high mortality of women with high education but a low income can be understood when education is considered as input, and income (even in retirement ages) is seen as output from the labor market. This group may suffer from not being successful in translating their education into material wealth, or else they lost their original occupational status. This would simply indicate that income is a stronger mortality predictor than education. The presence of the other group with high mortality, women with low education and high income, seems to indicate that this is not a general rule. Besides the order of importance between income and education as two dimensions of social status, there is a disadvantage of persons with an inconsistent social status, which means being on different levels in different dimensions of the social status (Siegrist et al. 1990). The above pattern was not found for Denmark.

8.4 Interaction between socioeconomic status and age

To address my central question, i.e., whether socioeconomic mortality differences remain stable, increase or decline with increasing age, it is necessary to run interactions between age, i.e., the basic time variable of the model, and a variable for socioeconomic status. In the following analysis, I will use income as an indicator for socioeconomic

status. This is because it has the highest independent impact on mortality (see Table 8.1 and Table 8.2). The analysis with the other indicators for socioeconomic status (not shown) sometimes show the same results, and sometimes less consistent results, than with income, but they never reveal very different or opposite patterns. Figure 8.5 shows the mortality for men with an interaction between age and income. Note that the graph uses a reference line for the lowest income group that equals one at all ages, i.e., it does not show the increase of mortality with age but only the relative differences between the three income groups.

To ensure that the pattern presented over age is not influenced by period and cohorts effects, I tested these models with period and cohort as control variables. The results do not differ significantly if period or cohort control variables are included in the model.

Figure 8.5: Male mortality with interaction between age and income, USA (low income=1)

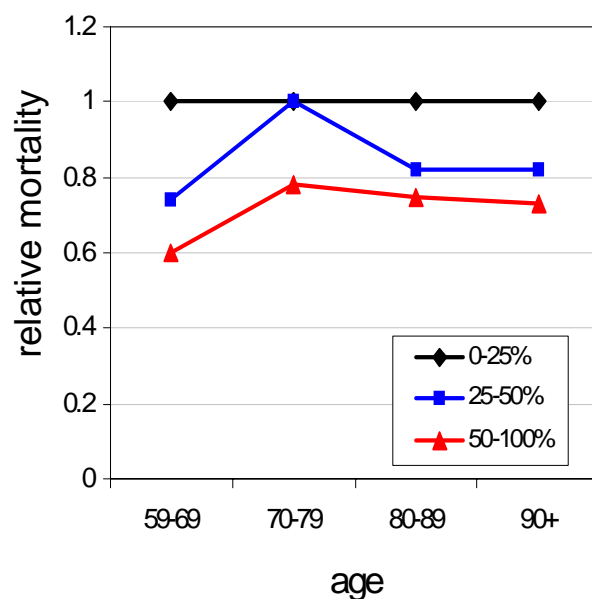
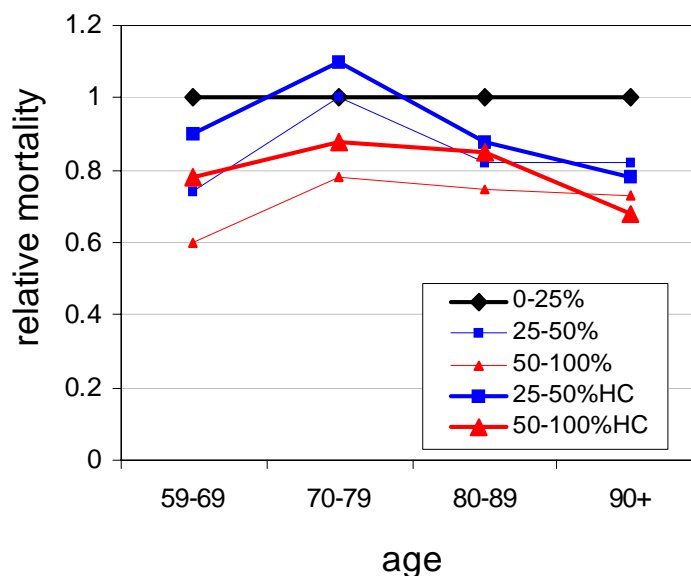


Figure 8.5 is based on Model 2 in Table 8.1 and controls for other socioeconomic variables and additionally includes an interaction between age and income. Just like the results in Table 8.1, the graph based on the interaction model reveals that men with the highest income have a significantly lower mortality than those with the lowest income. The upper bounds of the confidence interval for the rich group (red line) for the four age groups are 0.84, 0.99, 0.95 and 1.16 respectively. The confidence interval for the oldest group is wider because of a low number of cases in this group. Those with a middle

income also display a lower mortality, but this is not significant at the 95 percent level. Far from significant in this graph are the fluctuations of differences over age groups. This suggests that mortality differences between income groups are relatively stable over age and obviously not declining with increasing age. Again, the level of significance is not satisfactory, but here the differences in the oldest age group are non-significant because of the wide confidence interval due to low case numbers and obviously not because of a mortality convergence in old age.

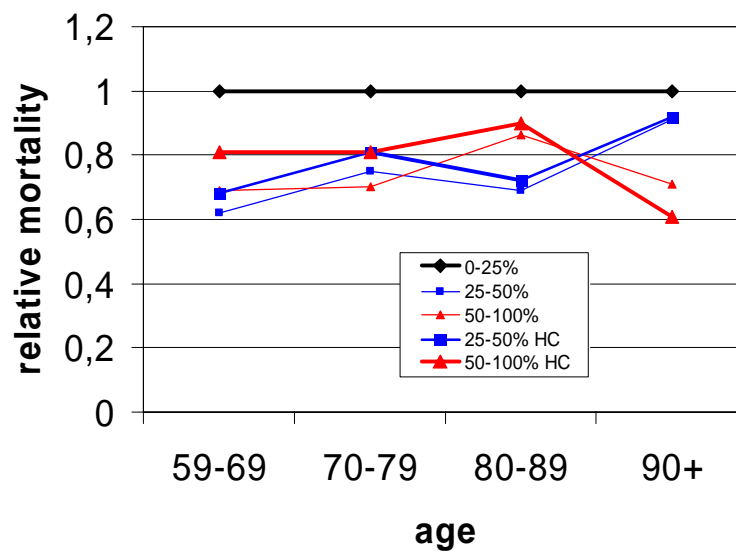
Figure 8.6 repeats Figure 8.5 (thin lines) and shows the same interaction but based on Model 3, which additionally controls for health variables (thick lines).

Figure 8.6: Male mortality with interaction between age and income, USA (low income=1) health controlled



If controlling for health, the lines for middle and higher income get closer to the reference line. This effect is limited to younger age groups, with the consequence that mortality differences between poor and middle/high income groups tend to increase with age. But due to the small sample, this increase is still far from being significant. The discussion of what it actually means to control for health will be carried out in the following section after the same kind of model has been presented for women in the USA and for Denmark.

Figure 8.7: Female mortality with interaction between age and income, USA (low income=1)



The figure for women combines the two steps that were used to present the results for men: the thin lines in the figure above show the interaction based on Model 2 without controlling for health. There is a slight convergence of mortality differences over age that shows fewer fluctuations than the graph for men. The thick lines represent Model 3, which controls for health. Social mortality differences in younger ages become smaller after controlling for health. All data points are significantly lower than 1 except for the middle income group at age 90+. The fluctuations between age groups or the differences between the middle and the higher income group (the blue and the red line) are not significant.

The results for the USA reveal a certain pattern over age and an impact of health as a control variable on his pattern. But as mentioned already, the significance is not satisfactory but will be better for the following results for Denmark.

The presentation of the Danish results will start with models and figures that are as comparable to the results for the USA as possible. This means that I will use the same income categories and the same control variables as in the figures above.

Figure 8.8: Male mortality with interaction between age and income, Denmark (low income=1) for comparison with USA

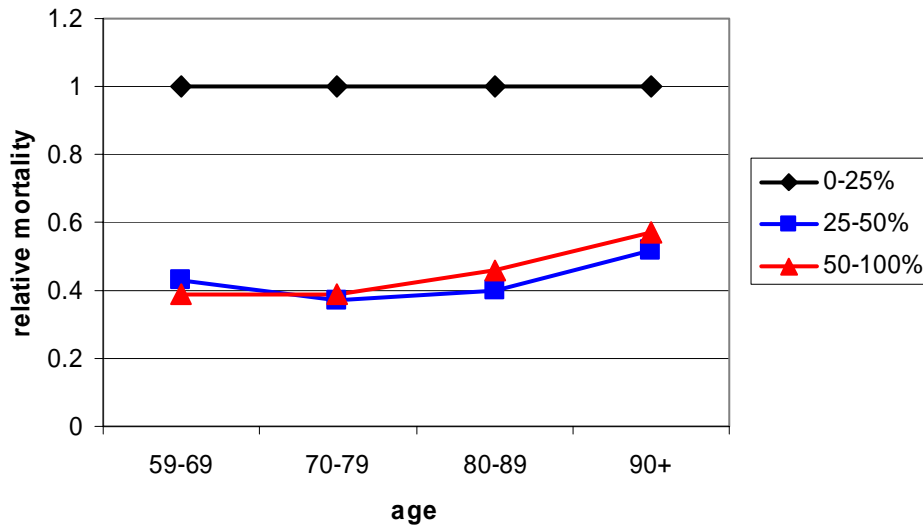
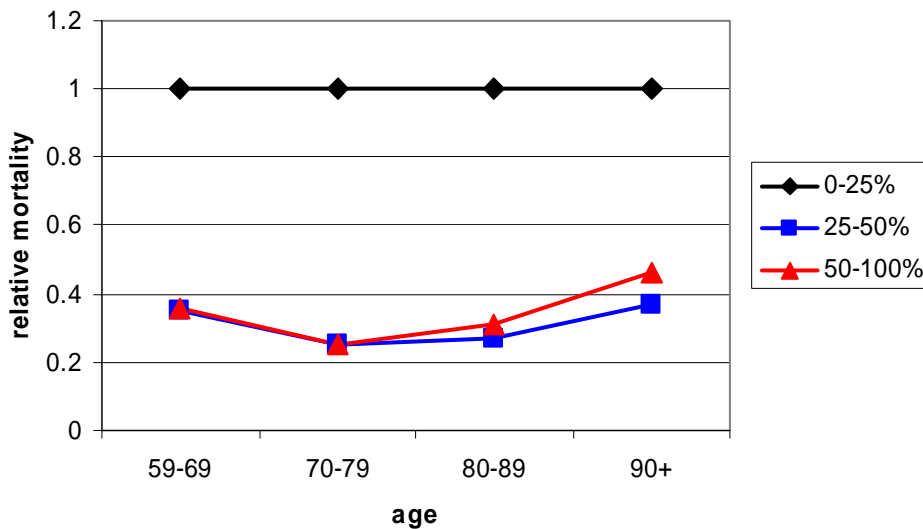


Figure 8.9: Female mortality with interaction between age and income, Denmark (low income=1) for comparison with USA

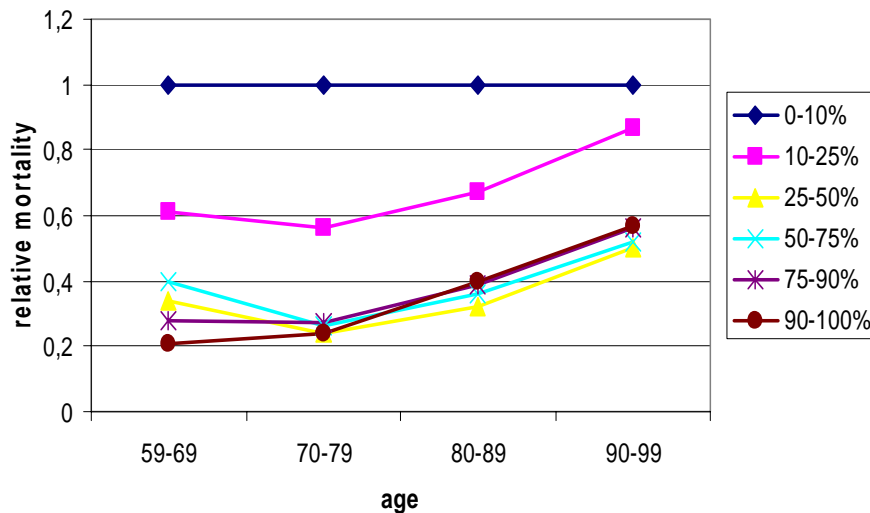


Mortality differences between income groups are much larger in Denmark than in the USA when controlling for the same covariates. Another difference is that in Denmark, mortality differences only exist between the lowest income group and the rest and not between the middle and highest income groups. In the USA, there is also a mortality difference between the middle and the high income groups, but only for men. In addition, social mortality differences are even larger for women than for men. This

finding is different from the majority of epidemiological studies finding higher socioeconomic mortality differences for men.

In the HRS data, the number of income groups for this analysis has to be small because of the small sample size. However, with the Danish data many more income categories can be used to show significant differences. The following figures repeat the analysis from above but show more income categories, thereby exhausting the possibilities of the Danish register data. In the following models and pictures, a comparison with the USA is not my major interest and therefore the larger number of income categories will be used in order to allow a more detailed analysis.

Figure 8.10: Male mortality with interaction between age and income, Denmark (low income=1)



The models with more income groups show that there are substantial mortality differences only between the poorest 10 percent, the next poorest 25 percent, and the rest. This means that between those who get an average income in Denmark and the richest 10 percent of the population, there is almost no mortality difference.

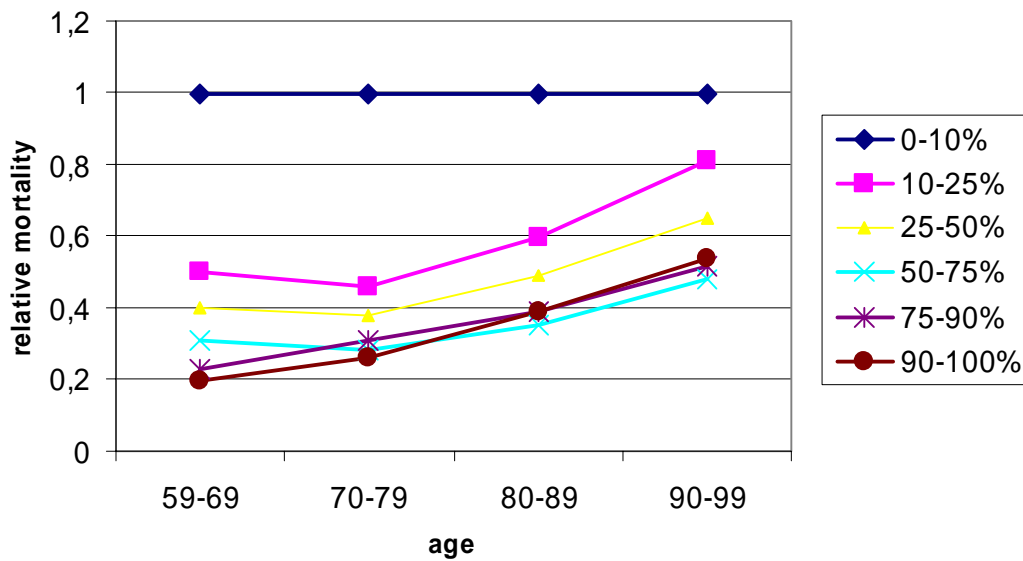
Before I show the results for women, Figure 8.11 shows the result of a test of the income measure. Until now income was measured with period-adjusted income percentiles, i.e., for each year, the income percentile in Danish kroner is used to define the income group for a person for one year. An alternative measurement is to adjust the income measurement also for age. This means that the percentiles are taken from the income distribution of one year and a specific age group. Two different theoretical understandings of income inequality are behind these two measurement methods: for the first measurement, it is assumed that age makes no difference for the definition of

low income. The same absolute amount of Danish kroner would define the poorest ten percent of the population at all ages. As a consequence, there are more than 10 percent in the lowest income group in old age because in old age it is more common to have low income. The second way to measure income differences assumes that the absolute need for money changes with age and as such the characteristic of inequality is age-specific. It is plausible that at least the subjective material deprivation, and the according comparison with other persons, happen within one age group. To take this into account we have to adjust for the changing income distribution in old age. Table 8.3 shows the changing income distribution with age that is obtained when the same absolute amount of income is applied to define income groups at different ages. Between ages 59 and 70, the poorest group is very special because it is only 4 percent of the population at this age. Over the age of 90, already 17 percent of the age group are in the lowest income group. It is worth checking if this change in the distribution has an effect on the results concerning mortality differences. Figure 8.11 shows the same model as in Figure 8.10 but based on the second income measurement.

Table 8.3: Income distribution in Denmark at different ages using a fixed income limit (percent)

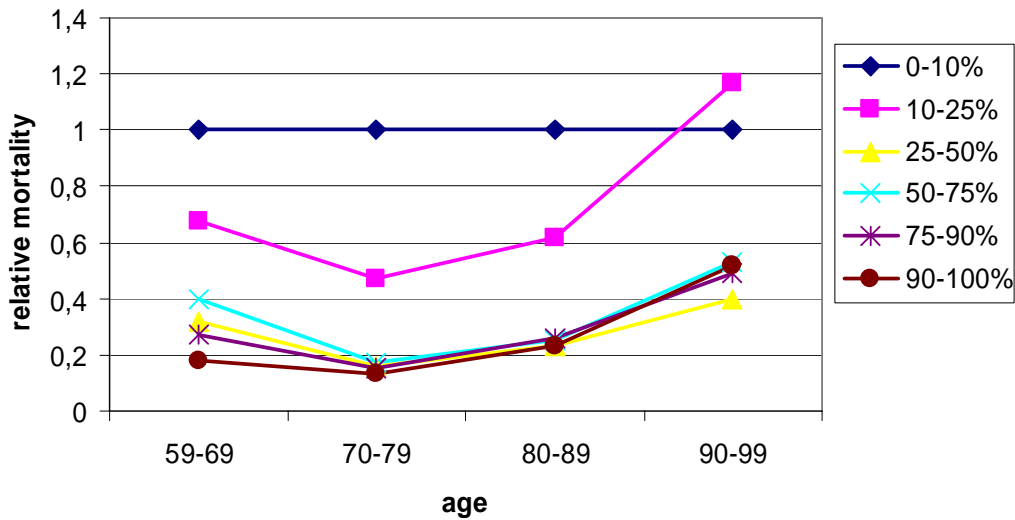
Income percentile	Age group			
	59-69	70-79	80-89	90+
0-10	4	5	8	17
10-25	6	17	20	15
25-50	9	30	35	35
50-75	29	24	20	20
75-90	27	14	11	9
90-100	25	10	6	4
Sum:	100	100	100	100

Figure 8.11: Male mortality with interaction between age and income, Denmark (low income=1), age adjusted income percentiles



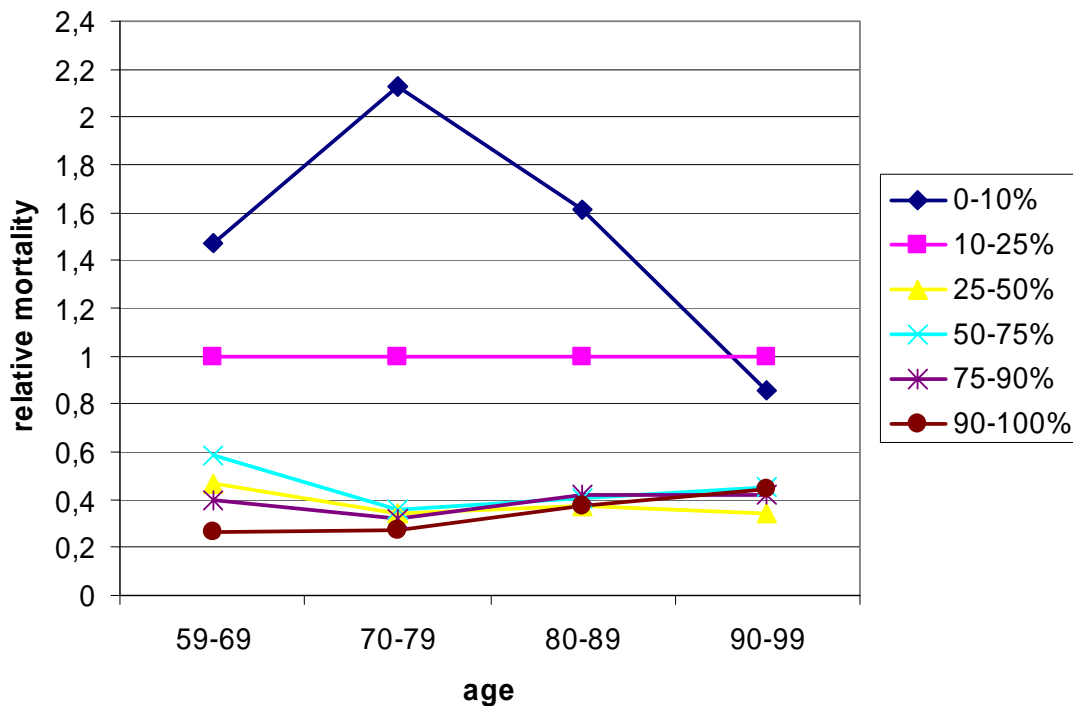
The difference between the two measurements is that the group with an income between the 25th and the 50th percentile is different and has a higher mortality than the remaining richer groups in the lower part of the graph. The second measurement reveals larger mortality differences between the two poorest groups because the poorest group with the new measurement is smaller and thus more extreme. Besides these small differences the overall mortality gradient and also the pattern over age is the same, so the first and simpler income measurement can be used without losing important information. The next figure shows the results for Danish women.

Figure 8.12: Female mortality with interaction between age and income, Denmark (low income=1)



The comparison between genders (between Figure 8.10 and Figure 8.12 above) shows no substantial differences. For women there are approximately the same mortality differences and the same convergence with increasing age. The difference is that the increase of mortality differences between the first two age groups is much stronger than it is for males.

The representation that is used in all figures of this section to present relative mortality differences has the disadvantage that the overall visible pattern depends on the choice of the reference category. It is straightforward and plausible to take the lowest or the highest status groups as the reference category. But in Figure 8.12 where all lines except the straight reference line follow the same U-shape pattern, it is worth thinking about the logic of relative differences and the impact of the choice of the reference category on the displayed pattern. As an example, Figure 8.13 below shows the same data, but here the second lowest income group is the reference category.

Figure 8.13: Female mortality with interaction between age and income, Denmark (second lowest income group=1)

Although it is the same relative mortality, the impression is different: there is still a clear convergence but it is more clear now that the U-shape pattern from Figure 8.12 may depend solely on the lowest income group. I do not have an explanation for why poor Danish women in their seventies have a much higher mortality disadvantage than at younger and older ages. Since all figures shown in this section do not change substantially when cohort is controlled for, it is unlikely that these fluctuations represent historical influences. I assume that possible changes of mortality differences around the retirement age have other reasons and another theoretical background than the pattern in high ages that is to be analyzed here. Thus, a possible non-monotonic pattern over age will not be considered in the further analysis.

Following the order of figures for the USA, the next step is to show the impact of controlling for health status on the age pattern of social mortality differences. Figure 8.14 and Figure 8.15 show the results for Danish men and women that are based on Model 2 which controls for days in hospital.

Figure 8.14: Male mortality with interaction between age and income, Denmark (low income=1) health controlled

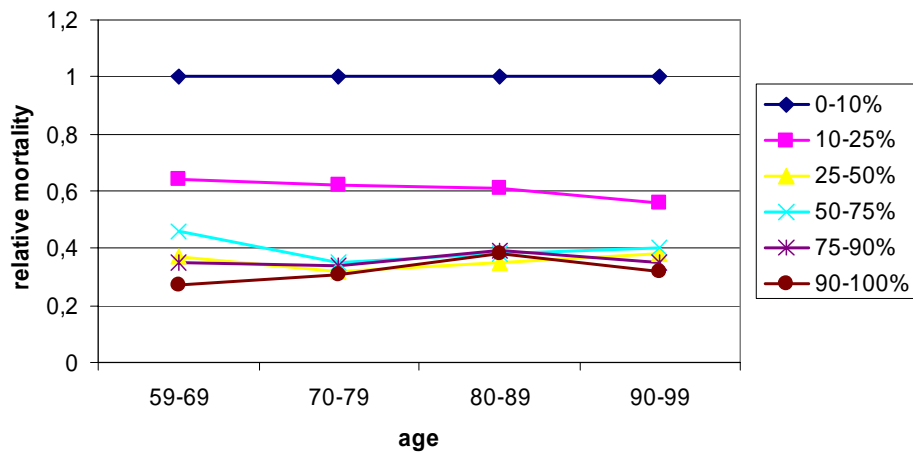
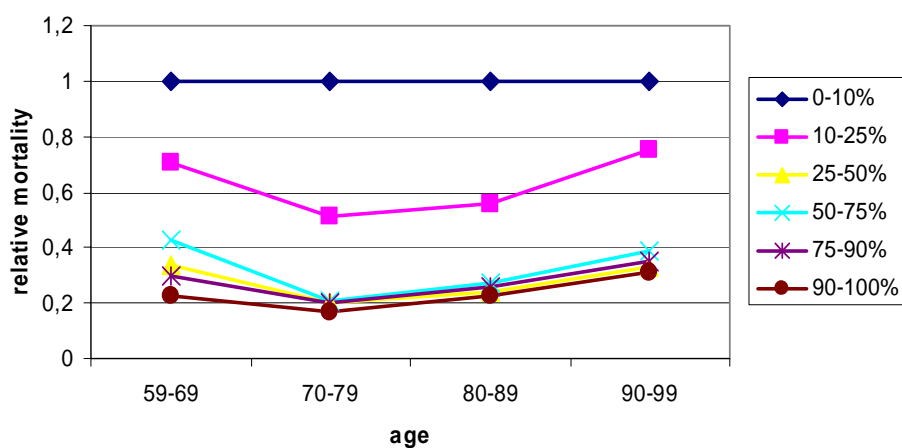


Figure 8.15: Female mortality with interaction between age and income, Denmark (low income=1) health controlled



The results for Danish men are very clear, especially in comparison to the results from the USA: there is a certain mortality convergence in Figure 8.10, but after controlling for health there is no convergence left. However, for women both models are more difficult to describe. First, there is the U-shape pattern, for which I do not have an explanation that would fit into the simplified divergence/convergence logic. Second, controlling for health removes much, but not all, of the convergence. In both countries the change of the pattern, if controlling for health, goes in the same direction but the result is still different: the mortality differences increase (insignificantly) over age in the USA (Figure 8.6), they are stable for men in Denmark and some converging pattern remains for Danish women in the figure above.

The main result from this section is first, that socioeconomic mortality differences converge with increasing age in Denmark, but they do not clearly converge in the USA (see Hoffmann 2005b). The latter may be due to having worse data from the USA, where the pattern is less reliable because of a low level of significance. Still, it is possible that there really is less convergence in the USA than in Denmark (see discussion in Section 8. 7). Second, controlling for health has basically the same impact on this pattern in both countries and for both sexes; the pattern converges less, converges not at all, or even diverges after controlling for health. The analytical step of controlling for health and the obtained results allow for two different interpretations:

1. The first interpretation takes health as an intermediate variable between socioeconomic status and death. If this intermediate variable is controlled for, the remaining mortality differences reveal the impact of e.g., income on the mortality risk given a certain health status. This means that this impact is net of the social health differences that contribute to social mortality differences because ill persons are more likely to die. The results suggest that the transition from a given health status still depends to a certain degree on social status. This was already shown in Model 3 for the USA and Model 3 for Denmark without interactions.

2. Concerning the pattern over age, controlling for health means that increasing age is analytically separate from worsening health⁵⁰. When the age pattern in models that do not control for health are considered, there is a change over time for individuals that get older and are likely to experience worsening health. Instead, in models that control for health, we just observe the changes that occur with increasing age. Given the considerable differences in the age pattern between these two perspectives, especially in Denmark, I can conclude that socioeconomic mortality differences converge with worsening health, but not with increasing age. To support this interpretation, the next section shows the interaction between health and income.

⁵⁰ In the data there may not be an individual that really experiences no health decline that could be observed, but the method of event history analysis splits up the histories of individuals into small time pieces where for an individual, the constellation of the values of the included variables changes from one piece to the next. By that, it is possible to estimate how the change of certain variables over age would occur if health was constant over age.

8.5 Interaction between health and income

This interaction shows how the impact of social status on mortality changes when health declines. This is the dimension that has been neutralized by controlling for health in the previous section. Figure 8.16 shows an interaction between self-rated health and income. Age is still controlled for, as it is in all models.

Figure 8.16: Male mortality with interaction between income and health, USA (low income=1)

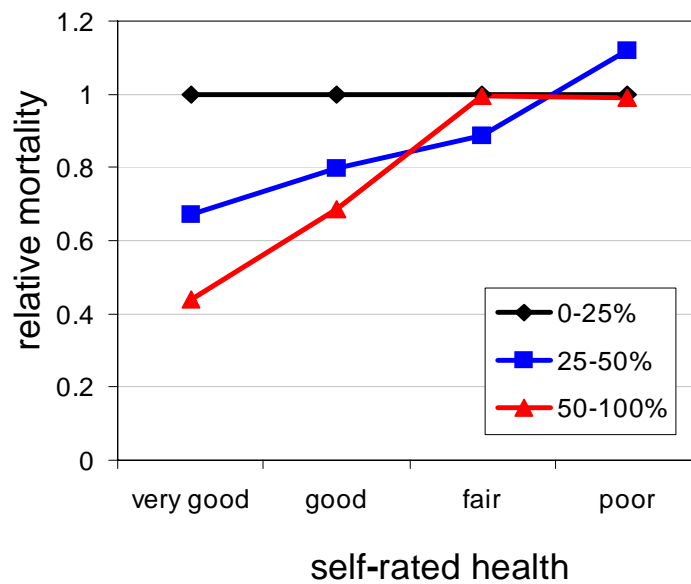


Figure 8.16 shows that income matters a lot for mortality when the person is in good health and that it has no impact when the person is in poor health. This means that poor health levels out socioeconomic mortality differences. The mortality difference between the lowest and the other income groups is only significant at the 95 percent level when people are in very good health (rate ratio (RR): 0.45, confidence interval (CI): 0.32-0.62; RR: 0.69, CI: 0.47-0.95).

Figure 8.17: Female mortality with interaction between income and health, USA (low income=1)



The same interaction for women shows the same general pattern. Income has the largest impact on mortality when health is very good. For both sexes there is even the same crossover at the health status “fair health”. The middle income group with this health status has lower mortality than the two other income groups. However, this crossover is far from being significant. It is worth mentioning here that the pattern for men and women does not depend on the choice of health indicator. The same result is obtained in an interaction between income and objective health (results not shown). In the Danish data set there is no such choice; the only available health indicator is “days in hospital”. Figure 8.18 and Figure 8.19 below show the same interaction for Danish men and Danish women. They reveal the same pattern: among persons who spend zero to three days in hospital per year, there are large mortality differences by income group, differences which are smaller for persons with worse health. This decline is less pronounced for women.

Figure 8.18: Male mortality with interaction between income and health, Denmark (low income=1)

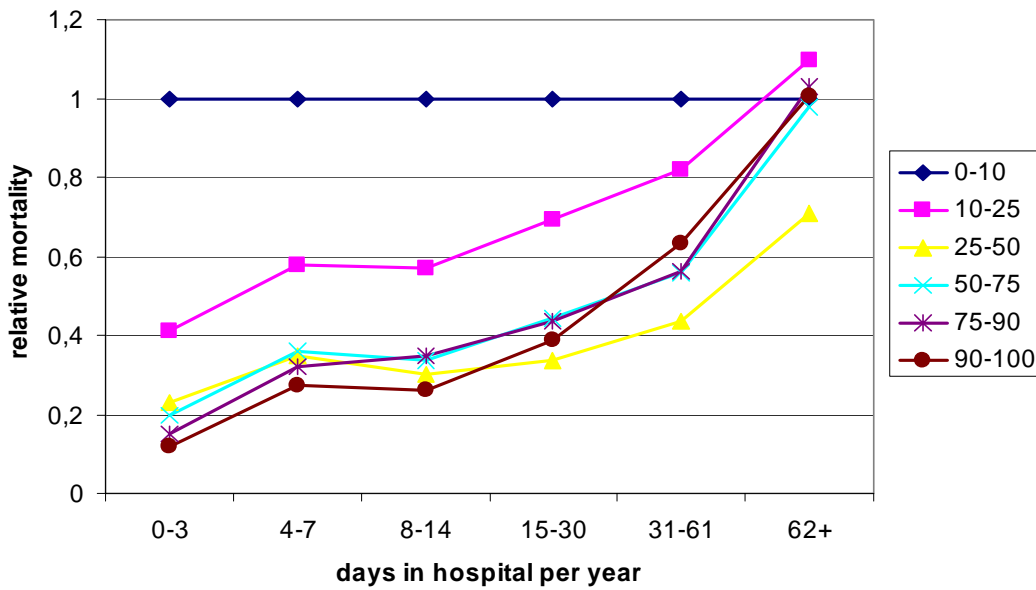
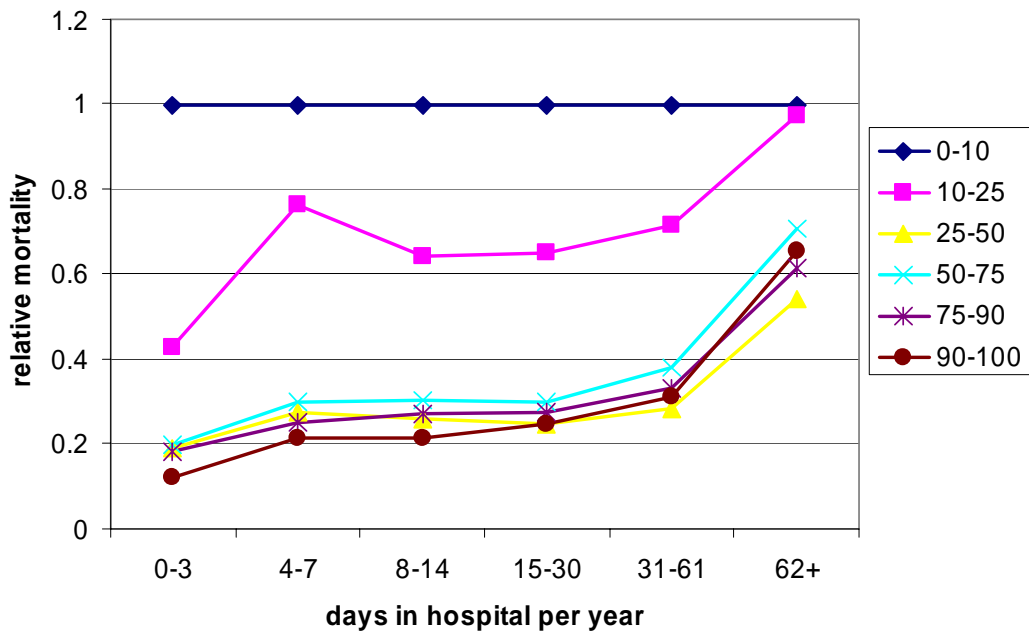


Figure 8.19: Female mortality with interaction between income and health, Denmark (low income=1)



Model 3 for the USA and Model 3 for Denmark showed that, controlling for health, the impact of socioeconomic status on mortality is smaller. The interactions between age and income in Figure 8.6, Figure 8.7, and Figure 8.14 reveal that the impact of income does not decrease with age as such. I suggested that the decrease occurs with worsening health. This assumption has been supported with the interaction between income and health in Figure 8.16 to Figure 8.19. These interactions show that income matters a lot when the person is in good health but that it has almost no impact when the person is in poor health (Hoffmann 2005b). This means that poor health evens out socioeconomic mortality differences and that the convergence of mortality differences with increasing age is mainly due to declining health. On average, health is worse in old age, thus mortality differences between income groups are smaller.

Socioeconomic status in old age may still influence the transition from bad health to death (Kåreholt 2000:14), e.g., men from lower social classes have a higher mortality risk than white-collar men when both groups have a heart problem (ibid.:36). But the results show that the gradient weakens considerably with declining health. This is plausible if the process from good health via bad health to death is considered: much of the social differentiation in this process has already occurred when a disease is developed. The subsequent individual pathway from bad health to death may still be open to social influences, but a considerable part of the trajectory is already determined by the health status. The impact of income on a good health status via direct material welfare and income-related non-material aspects is higher than its impact on a bad health status via different medical treatments (Klein and Unger 2001). Thus income is much more important and beneficial when it supports a good life lived in good health than when it is used for purchasing good medical care and expensive drugs when a person is already ill.

The equalizing effect of worse health does not mean that social inequalities no longer exist after health has become poor. It rather changes to focus on health differences that are already caused by socioeconomic status. Social inequality, in old age more than in younger ages, is just incorporated into a more or less severe health decline and therefore there is no longer social inequality in the transition from poor health to death. Thus, the question of social inequality in health is not only analogous to but it becomes part of the question of social inequality in mortality. Research findings reveal clear socioeconomic health differences at old age, as was referred to in Section 5. 3. Liao et al. (1999) show

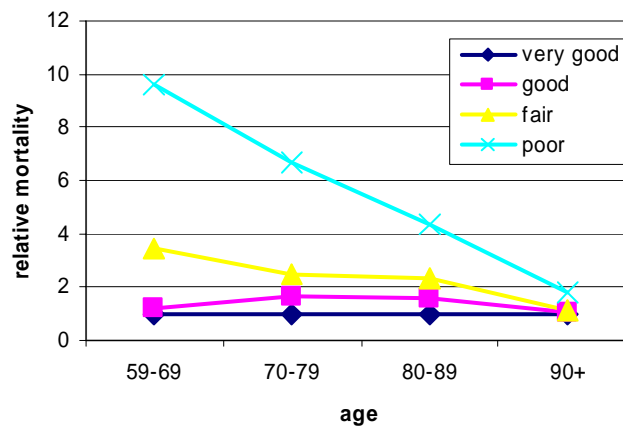
that having higher socioeconomic status means having lower morbidity, less disability, and more quality of life, even in the last year of life.

In my study, I can only make an attempt to analyze health inequalities which reveal increasing health differences because from an already unequally distributed health at onset, the rate for health deterioration is also higher for low income groups (see Section 8. 8). I will come back to socioeconomic health inequalities after the next section in which the last interaction between health and age will be presented.

8. 6 Interaction between health and age

Concerning the question of whether socioeconomic mortality differences decline with age or not, it is, finally, important to see whether the impact of health status on mortality is stable across age groups. Again, four graphs will be presented, for men and women in both countries.

Figure 8.20: Male mortality with interaction between age and self-rated health, USA (very good health=1)



The interaction between age and self-rated health reveals that mortality differences between health groups are very large in younger age groups (ages 59 to 69). When all socioeconomic variables and the other health variables are controlled for, men with a poor self-rated health status at this stage have an almost ten-fold higher mortality than those with a very good self-rated health. The figure below for women shows even higher relative risks. The convergence in both figures for the USA is not due to self-estimation by the respondents. The same interactions based on the objective health

measure show an even stronger convergence (results not shown), but for consistency, the self-rated health measure is used here as in all other interactions.

Figure 8.21: Female mortality with interaction between age and self rated health, USA (very good health=1)

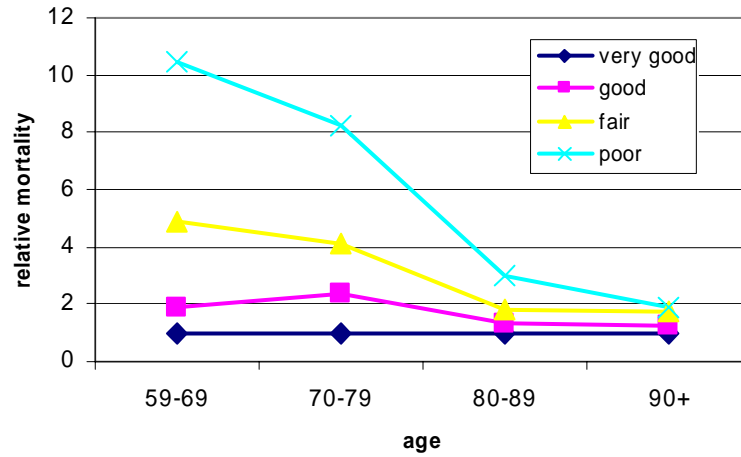


Figure 8.22: Male mortality with interaction between age and days in hospital, Denmark (best health status=1)

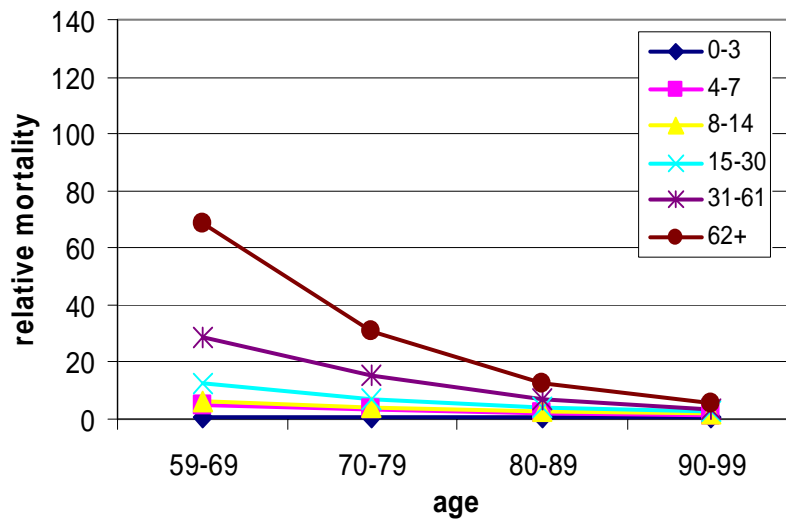


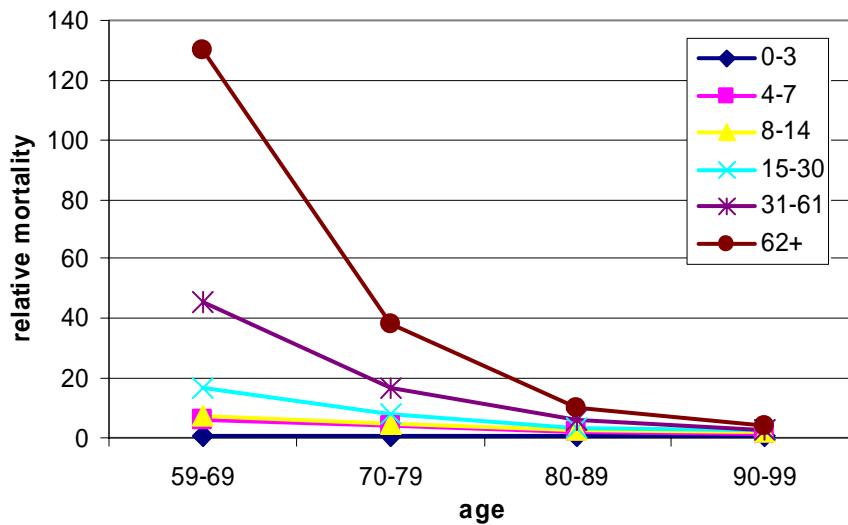
Figure 8.23: Female mortality with interaction between age and days in hospital, Denmark (best health status=1)

Figure 8.22 and Figure 8.22 show that in Denmark, men who spend 62 days or more in hospital have a 70-fold higher mortality (for women, the corresponding figure is 130-fold) than those who only spend a few days in hospital. These mortality differences converge very strongly in older age groups.

The results from the interaction between age and health suggest that in very old age, mortality depends less on morbidity than in younger ages. This has been found in other studies: Helweg-Larsen et al. (2003), after controlling for many variables, find no relationship between self-rated health and mortality over age 55. Van Doorslaer and Gerdtham (2003) find that self-rated health predicts mortality in old age much less than in younger ages and Hayward et al. (1998:197) show that the mortality difference between active and inactive persons decreases with age. The authors also show that at younger ages many more deaths occur out of a poor health status and that in old age many persons are relatively healthy before they die (ibid.:206).

These results mean that due to a health status that, on average, declines with age, health differences do not necessarily translate into mortality differences. In old age it is more common that people die of a very minor physical problem without being considerably ill for a certain period beforehand or without having a treatment in a hospital.⁵¹

⁵¹ It is worth recalling the measurement conditions for health in our data. In the HRS survey where an interview takes place every second year, to measure a bad health status before a person dies requires that this person gave this information in the last interview. This may have been up to two years before the time of death, i.e., only considerable and persisting health problems show up in the data. In Denmark, only health conditions that lead to hospitalization are included in our health measure.

8.7 Discussion of the findings on the age pattern of social mortality differences

In the USA, mortality differences between income groups do not clearly increase or decrease between age 59 and the highest ages. The low level of statistical significance in the small sample prevents a more detailed interpretation of the age pattern shown in the figures for the USA. In Denmark, there is a convergence of mortality differences. In both countries there is less convergence or even divergence after controlling for health status (Hoffmann 2005a). These results allow a tentative evaluation of the arguments listed in the introduction, which leads to the following interpretation:

Age increases for everyone. This means a convergence of socioeconomic mortality differences with age would actually indicate that the impact of socioeconomic status decreases with age as a result of an equalizing welfare state policy, or due to the temporal distance to unequal health experiences e.g., during one's work life (arguments 2 and 3 in Section 5. 1). But instead, I find that socioeconomic mortality differences are stable across age groups (which supports arguments 5 to 7) and that instead of age, poor health is the equalizer for social differences, maybe as a result of a universal shift from social to biological determinants of mortality as health decreases (argument 1).

The suggested arguments do not seem to be mutually exclusive, e.g., accumulating social differences and the dominance of bad physical conditions over social conditions could possibly occur simultaneously. So maybe the observed pattern over age is the combined effect of accumulation of socioeconomic status and health on the one hand, and domination of physical conditions over social factors contributing to the transition from poor health to death on the other. The third argument, and other similar explanations that are based solely on the temporal distance to working age or on numerical age, can be ruled out according to the findings showing that increasing age as such does not lead to converging mortality differences.

Social mortality differences are substantially larger in Denmark than in the USA. This is a surprising finding given the lower level of social inequality in Denmark. Among other reasons the finding by Kåreholt (2000:1) mentioned in Section 4. 4. 6 may be interesting in this regard: The overall level of wealth was higher in the USA than in Denmark when the elderly from today were born. Therefore social mortality differences in later life may be smaller in spite of the high current level of social inequality.

For men the complementary result is that above the median income, Denmark shows fewer mortality differences than the USA does. But this can not balance out the overall

finding that mortality differences are so much higher in Denmark. The slight convergence over age (significant for Denmark but not significant for the USA) is due to poor health rather than to old age because it disappears when health is controlled for. The first answer to the central question of my research, i.e., whether socioeconomic mortality differences decrease with age or not, is a modification of the question by the identification of two aspects of increasing age. Both of these aspects increase mortality but have very different implications for the impact of social status on mortality. The first aspect, increasing numerical age, seems to be trivial but, in fact, some of the arguments used to support the hypothesis of mortality convergence are based on numerical age. These arguments can now be rejected. The second aspect is declining health, where my finding that money matters less in poor health rejects the assumption that money is of major importance to people in bad health in order to get good treatments to prevent them from dying. It is more convincing to think of social mortality differences as a process that already started with social differences in health.

Concerning declining health, the problem remains: the theoretically simple scenario that a socially mixed sample will experience a simultaneous health decline that would level social differences in mortality will practically never happen. The health decline of upper class persons will either be delayed, will start on a higher health level, or will be slower. Therefore, it is difficult to say if the potentially leveling impact of a health decline is actually effective. This is because poor health is likely to be, to a large extent, the result of low socioeconomic status and thus it is unequally distributed.

To conclude on this point: even if it is plausible to assume that increasing age is generally combined with worsening health, it is worth keeping these two dimensions of aging separate for analytical purposes (Hoffmann 2005a). This is because age increases for everyone but health decline is very different for different social groups.

Irrespective of the question of whether health is controlled for or not, until now it is not possible to interpret the observed convergence as a decreasing impact of socioeconomic status on mortality. Later in Chapter 9 I will try to analyze the extent to which the observed mortality convergence is the result of the impact of unobserved heterogeneity. Health status and health decline are important for the impact of social status on mortality. Therefore, the next section will attempt to measure how socioeconomic health differentials change over age.

8.8 Socioeconomic differences in the health trajectory

The question is whether the health decline with age is equally distributed between social groups, enough to result in a leveling of the mortality between social groups. In a follow-up from 1988 to 1994, Grundy and Glaser (2000) find that not only the initial level of disability, but also the onset and the progression of disability differs by social status between age 55 and 69. Hemingway et al. (1997:1273) find that, “socioeconomic status is associated inversely with baseline functioning and, independently, with decline in health”.

I would like to report here three aspects of health distribution. First, health declines generally with age: the correlation between age and average health during the study is 0.20*** for self-rated health, and 0.34*** for objective health. There seems to be an adjustment for age in the self-estimation of health, which results in a lower correlation with age compared to the objective measure. But despite the general health decrease with increasing age, health is unequally distributed between income groups. Table 8.4 shows the other two aspects of the health distribution: first, the average self-rated health status at the beginning of the observation and, second, the experience of health deterioration during the observation period, both by the three income groups from above. A transition from good to bad health here means that at the beginning of the observation period, a person was in either the best or the second-best category of either self-rated or objective health and has since moved down at least two levels by the end of observation.

Table 8.4: Distribution and deterioration of health in different income groups by age, USA

	age in	n=	low income	middle income	high income
	1992				
Percentage having very good or good health at the beginning of observation	59-68	3140	58.2	78.4	88.7
	69-78	4114	54.9	74.9	80.6
	79-102	2122	52.6	69.8	73.3
Percentage that experiences a health deterioration	59-68	2408	13.1	9.6	6.5
	69-78	2799	18.1	13.7	11.8
	79-102	1273	22.6	23.9	19.1

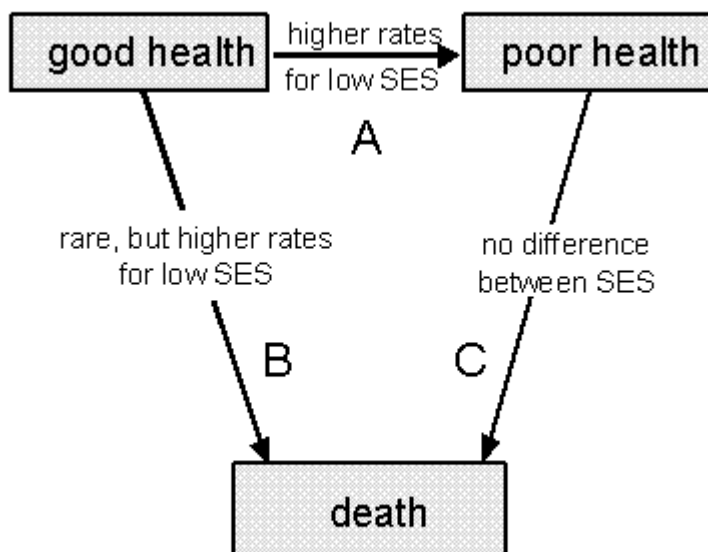
Pearson's chi-square test has been applied to the original two-way tables (not shown) and the differences in the table are significant at the 99 percent level, except for the last row (see text).

It is difficult to measure how large health differences are and even more to measure how these differences change with age. But it is obvious that even if health generally declines with age, first, people with lower income initially have a lower health status and, second, they are more likely to experience a health decline. The number of cases for the analysis of health decline is smaller than that for the analysis of health at onset because only healthy persons can be considered for a possible health decline. In the oldest age group (the last row of the table), healthy persons are especially rare and selected, which may help explain why the differences between income groups are not significant. The first finding, that persons with low income have worse health at the beginning of observation, is not surprising. It reflects the well-known income health gradient that exists at all ages. The second finding, that persons with worse health are also more likely to experience a steeper health decline, is the only plausible consequence from the first finding. It is not plausible to assume that healthier persons have a steeper health decline just because they did not have this decline before (Lynch 2003:32).⁵²

Figure 8.24 neglects the age dimension for a moment and shows the relationship between health, socioeconomic status (SES), and death, summarizing the findings from Table 8.4 (Transition A), and from the interaction between health and income (transitions B and C).

⁵² It is not possible to do a parallel analysis for Denmark because the health indicator in the Danish data (number of days in hospital) is less valid. Therefore, if a ratio of days in hospital between rich and poor people is computed, it would possibly be an over-interpretation to observe this ratio over age.

Figure 8.24: Transitions between good health and death



It is not obvious from my findings how age intervenes in this constellation. On the one hand, the interactions between income and age, when health is controlled for, show that the impact of social status is constant over age. On the other hand, the impact of health on mortality decreases with age, as shown by the interaction between health and age. To answer this question, a very good measurement of socioeconomic health differences across age groups and maybe a multi-process model for health decline and mortality would be advantageous, but both go beyond the scope of this study at the present stage.

8.9 Socioeconomic mortality differences by cause of death

The following presentation of a cause-specific analysis of socioeconomic differences in mortality will be mainly about Denmark because only for Denmark can I use the variable ‘cause of death’ in my own analysis.⁵³ In Section 4. 4. 1, the general role of causes of death and their importance for the analysis of socioeconomic mortality differences was pointed out. The following analysis will start with a brief description of the development of causes of old age mortality in the USA taken from David Smith

⁵³ The information about causes of death is theoretically available for the HRS data, but it is restricted data which is only accessible after a long procedure of applications and security checks. Moreover, this data is only rarely made available to researchers outside of the USA.

(1998). Table 8.5 shows the mortality rates for four common causes of death in two age groups from 1950 to 1990.

Table 8.5: Changes in mortality rates from common causes of death, USA, age 70-74 and age 85-89, deaths per 100,000 persons per year

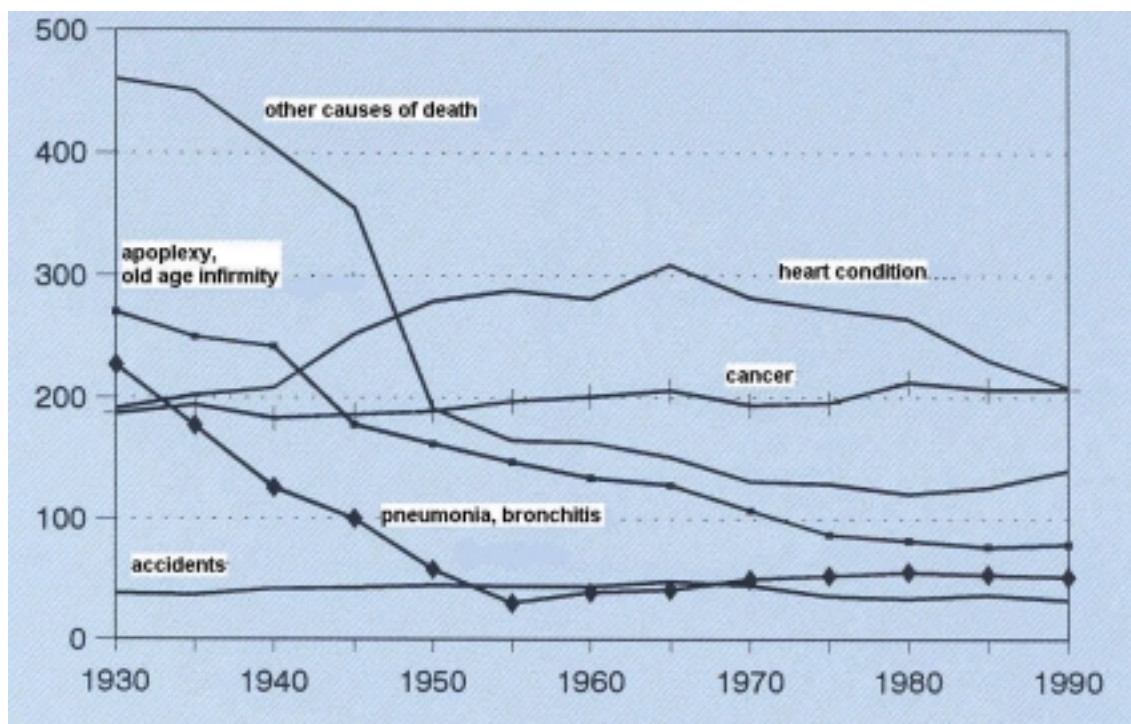
Cause of death	1950	1960	1970	1980	1990	1990 as percent of 1950
Age 70-74						
All causes	5170	4721	4376	3683	3266	63.2
Circulatory diseases	2540	2994	2603	1935	1408	55.4
Ischemic heart disease	1660	1680	1785	1171	799	48.1
Cerebrovascular diseases	743	639	518	302	195	26.2
Cancer	833	819	857	941	1013	121.6
Age 85-89						
All causes	18086	18563	15413	13499	11883	65.7
Circulatory diseases	12625	10191	11336	9023	6709	52.3
Ischemic heart disease	5354	7030	6788	4712	3413	63.7
Cerebrovascular diseases	2840	3500	2849	1265	1257	44.3
Cancer	1466	1557	1424	1631	1670	113.9

Source: David Smith (1998:332)

All causes of mortality declined considerably for both age groups between 1950 and 1990. Mortality from circulatory diseases, ischemic heart diseases, and cerebrovascular diseases decreases significantly starting in the 1960s and 1970s. Cancer mortality slightly increases, perhaps due to the fact that persons surviving other diseases have a higher risk of eventually dying of cancer.

Figure 8.25 shows a more complete and detailed picture for mortality at all ages in Denmark which reveals a similar trend: a peak in mortality from heart conditions in the 1960s and a more stable, trend in cancer mortality. Other significant changes happened in the first half of the 20th century: the decrease in mortality from other causes and from apoplexy and old age infirmity partly reflects improvements in the designation of diseases, while the decline in pneumonia and bronchitis is due to antibiotics and better medical treatments of such diseases.

Figure 8.25: Development of all-age mortality by different causes of death in Denmark, age-standardized mortality rates



Source: Sundhedsministeriet 1994a:55, changed

My analysis of the causes of death for Denmark is based on the information about the first cause of death of a person. A second and third cause of death were also available, but were not taken into account. The causes of death are classified according to the International Classification of Diseases (ICD) by the WHO (2004). When a person dies in Denmark, all civic information about the deceased is collected, including the unique personal identification number which identifies all residents in Denmark. Additionally, the underlying and contributory causes of death, manner of death, and possible results from an autopsy or other examinations, are registered. This information is stored in the Danish register of causes of death (Juel and Helweg-Larsen 1999:354).

In this register, all deaths from 1969 to 1993 were classified according to the Eighth Revision of the Manual of the ICD (ICD-8), and from 1994 on they switched directly to ICD-10. Due to these modifications in the classification, cause-specific mortality statistics are not fully comparable over the years (Juel and Helweg-Larsen 1999:354).

I used a translation key proposed by Janssen and Kunst (2004) in order to connect ICD-8 and ICD-10.⁵⁴ The remaining inconsistencies can be accepted because the analysis does not show time trends but rather models that show social differences. It is very

⁵⁴ This concordance table can be found in the appendix. I completed this information by personal communications with the authors about groups and subgroups of causes of death.

unlikely that the bias is large enough to justify the restriction of this analysis to the years 1980 to 1993. The following ten categories of causes have been used (in the order of the ICD classification):

1. Infections and parasitic diseases
2. Cancers
3. Diabetes
4. Alcohol-related causes
5. Mental disorders
6. Circulatory diseases, including ischaemic heart failure, heart failure, other heart diseases and cerebrovascular diseases
7. Pneumonia and Influenza
8. Liver and kidney diseases (except for alcoholic liver diseases) and ill-defined symptoms
9. Accidents, injuries and other external causes
10. Suicide

The following figures (Figure 8.26 to Figure 8.31) are based on event history models that take into account a single cause of death. This means only a death of a specific cause is considered as an event. When a person dies of a different cause of death, the case is censored. Besides this different definition of the event, the models and the results are the same as in the previous sections. The following figures are based on models without covariates in order to show the simple mortality hazard over age for the ten causes separately. Since the absolute risk levels are very different for different causes, the presentation will be divided into two different figures, one for common and one for rare causes, for each sex separately. Cancer (red line) is between “common” and “rare” causes and appears in both kinds of figures, which allows for a comparison between the two different graphs.

Figure 8.26: Hazard curves for common causes of death, Danish men

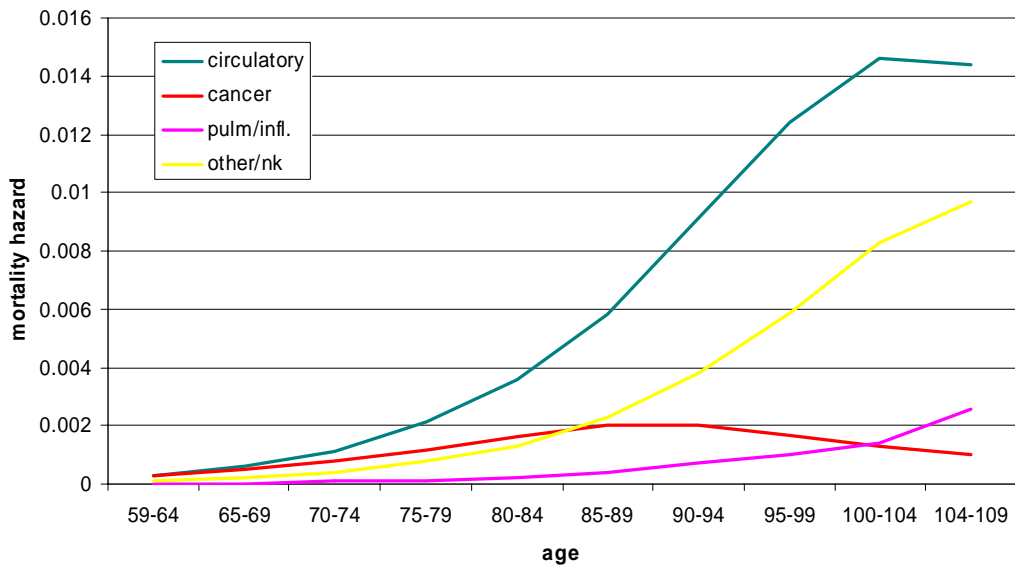
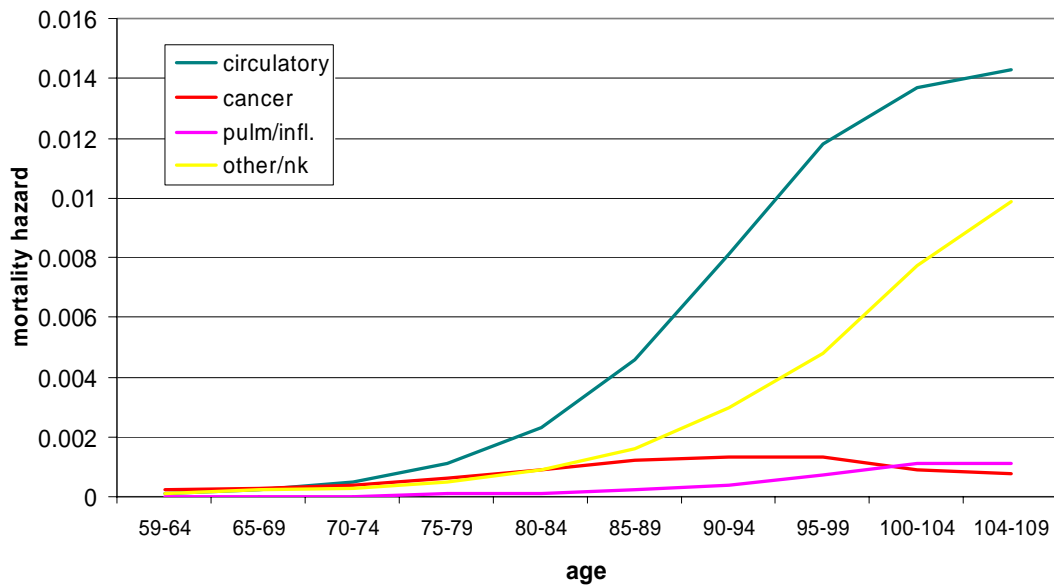


Figure 8.27: Hazard curves for common causes of death, Danish women



There are no significant differences between men and women concerning the three common causes of circulatory diseases, “other/not known”, and cancer, other than that women have a lower risk of dying of cancer.

Figure 8.28: Hazard curves for rare causes of death, Danish men

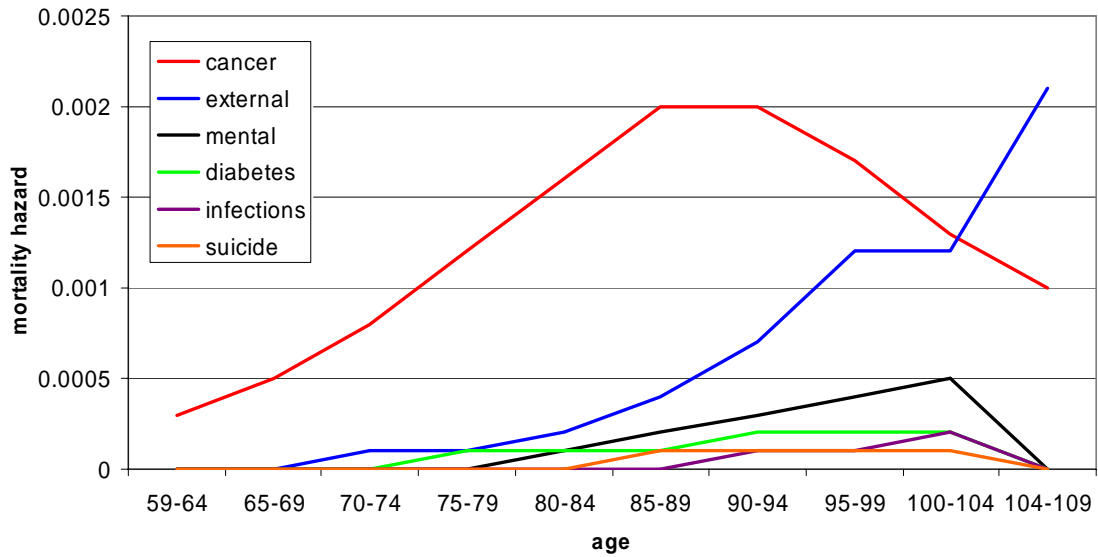
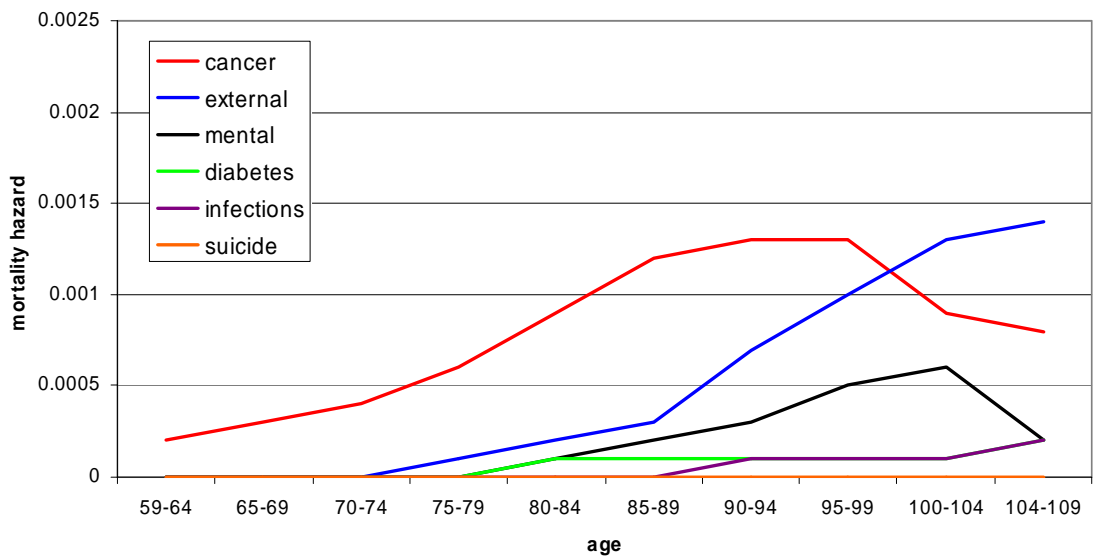


Figure 8.29: Hazard curves for rare causes of death, Danish women



Without going into detail, the general pattern, i.e., the order of magnitude of the different causes, is very similar between men and women. Generally, there are causes where the mortality risk peaks at some age, e.g., cancer around the age of 90, and other causes where the mortality risk increases til the last age group. For the first group, the explanation is that if a person survives a certain age group, the risk of dying of cancer,

for instance, decreases whereas the risk of dying from other causes continues to increase.

The next two figures continue the analysis of socioeconomic mortality differences with the income gradient in mortality by cause of death. This is done again with event history models, but here the models include the same covariates as the models in the previous sections: education, marital status, children in household, occupational status, source of income, wealth and income. As in the figures in Section 8. 4, the lines represent income levels (percentiles), the poorest 10 percent of the population being the reference category that is always 1. The causes of deaths on the x-axis are ordered from the most to the least frequent cause of death, except for other/not known. The results do not confirm the idea mentioned in Section 4.2.2 that the most common causes of death have a more unequal distribution between income groups.

Figure 8.30: Income mortality gradient for different causes of death, Danish men

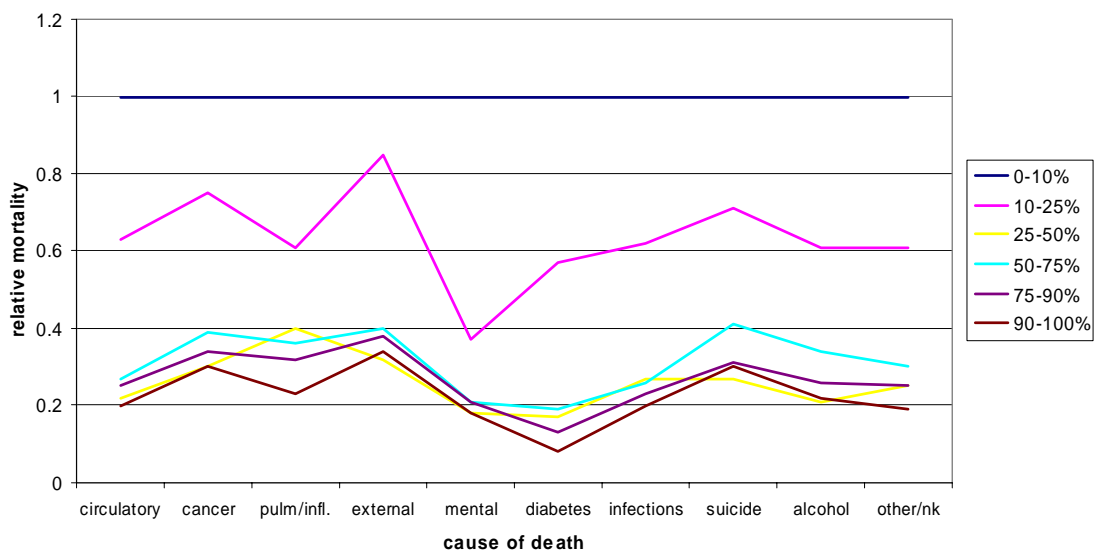
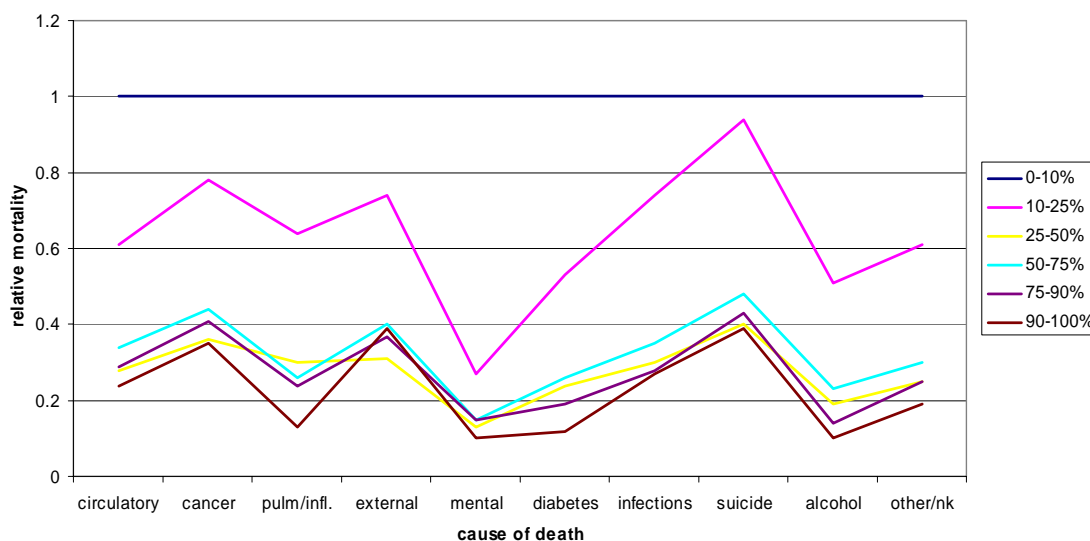


Figure 8.31: Income mortality gradient for different causes of death, Danish women

Note: The lines in these figures do not represent a connected pattern, e.g. a development over time or across categories, as lines usually do. However, differences between the gradients for different causes of death can be displayed more easily using lines compared to box-plots with 6 markers.

The interpretation of the two figures above is that a substantial mortality gradient between income groups exists for all major groups of causes of death. There are differences in the steepness of this gradient: mental disorders and diabetes seem to depend more on income than cancer, and alcohol-related deaths for men show a higher social gradient than for women. But in continuation of Section 4.4.3 (fundamental causes), the conclusion is that the disadvantage of being in a lower social status group, which is represented here by income level, is a very fundamental disadvantage that cannot easily be attributed to certain causal pathways, certain risk factors, or certain causes of death.

The separation of different causes of death can also contribute to the understanding of the pattern of social mortality differences over age. It has been argued that a change of these differences with age can be due to the change of causes of death. If more equally or more unequally distributed causes of death become dominant for mortality in higher ages, social differences could accordingly decrease or increase with age (Dobhammer et al. 2005). According to the findings above, the impact of deaths from circulatory diseases, other or unknown causes, and external causes steeply increases with age. However, these causes do not have an especially low or high social mortality gradient. Thus it seems unlikely that the age pattern of social mortality differences is influenced by the composition of causes of death changing with age.

Summary of Chapter 8

The main results from the mean effect models for the USA and Denmark are first, that in both countries the high educational mortality gradient is substantially reduced if income is controlled for. This suggests that educational mortality differences revealed by a univariate model are due to the fact that higher education is combined with higher income. But given a certain income, education only has a minor impact on mortality. Second, among the variables included in the model, income is the most powerful independent predictor for mortality. However, wealth has also a substantial effect on mortality that is partly independent from income.

Several interaction models were applied to get further insight into the interplay between variables. The interaction between marital status and age reveals that in the USA, with increasing age unmarried women (both the widowed and the divorced/never married) have increasingly lower mortality than married women. For men the mortality disadvantages that exist for single men disappear in high ages. In Denmark, singles generally have higher mortality than married persons, but this disadvantage is much higher for women. For women in the USA an interaction between income and education suggests that only women having both high education and high income have a mortality disadvantage.

The most important interaction between income, the indicator for socioeconomic status, and age reveals the amount of socioeconomic mortality differences and the age pattern of these differences. Social mortality differences are substantially larger in Denmark. The results for the USA show that the income mortality gradient does not change significantly over age, the level of significance being unsatisfactory in some of the graphs. In contrast to this, there is a significant convergence of social mortality differences with increasing age in Denmark. Also clearer in Denmark than in the USA, including health in the model causes a change of the age pattern of social mortality differences. Controlling for the normal health decline with age, mortality differences are stable across age groups, which means that it is not increasing age but worsening health that is the leveler between social groups. This can also be seen in the next group of interactions between health and income: with a good health status mortality differences between income groups are substantial and they do not exist for people in poor health. This is true for both sexes in both countries. The last group of interactions includes

interactions between age and self-rated health and shows that the health status is much less predictive for relative mortality at old age than at a younger age.

Section 8.7 uses the findings in Chapter 8 to discuss the arguments listed in Chapter 5. Most importantly, arguments that are based on age as a leveling factor are not confirmed whereas the idea that once an illness has developed, social differences are much less important does seem to be true. A thoughtful interpretation of this finding cannot be that worsening health levels out social differences. Rather, social mortality differences decrease only after the socioeconomic status already had an effect on the health status. To explore this argument, Section 8.8 compares the decline of health with age between income groups in the USA. The health at onset is not only worse for poor groups, the subsequent health decline is also faster than in higher income groups. The last section of this Chapter differentiated between 10 causes of death. The income mortality gradient in Denmark is different for different causes of death but there is a steep gradient for all major causes of death.

Chapter 9 Unobserved heterogeneity

The true change of the impact of income over age for the individual can only be shown after a successful estimation of unobserved heterogeneity. Until now I have presented results where the pattern of socioeconomic mortality differences over age is possibly biased by unobserved heterogeneity and mortality selection. Since we know in what direction the heterogeneity bias works, it is possible to conclude that if there is a bias, then the results in Section 8. 4 underestimate socioeconomic mortality differences in old age. As a consequence they would overestimate the convergence. Thus the question is whether the slight mortality convergence between social groups shown in the previous chapters is true or not. Of course it is true in the sense that if the existing population at old age is considered to be divided into income groups, then the mortality differences correspond to what is shown in the graphs. But it may be unreal in the sense that the observed convergence cannot be interpreted as a decreasing impact of social status on mortality with age because on the individual level, the impact does not necessarily decline.

This chapter is an attempt to analyze and measure the heterogeneity bias. It is a presentation of the most important steps, results, and conclusions that I got from many different attempts to answer this question, which involves difficult theoretical, methodological, and computational problems.

First, I will address the theoretical basis for the concept of unobserved heterogeneity, namely the distribution of frailty in a population. Frailty models and their meaning will be presented. Second, I will explain why and how I simulated different datasets for testing different attempts to address computational and methodological problems. Third, I try to apply these methods to my real datasets and present a new method that could substitute for advanced statistical models in cases where they cannot be used.

9.1 Frailty

As a follow-up to Section 5. 1, where I mention the selection hypothesis as an argument for why the mortality convergence can occur, the following section will explain the theoretical background of this argument in greater detail. In the statistical models that were used until now in this study, the strategy was to include as many observable

characteristics of the individuals as possible in the form of variables. The collected variables, for which an influence on the mortality risk has been theoretically and empirically shown elsewhere, mutually control for each other in the model and their isolated impact on mortality is shown.

Now we must consider unobserved characteristics, those which are not included in the dataset and those which are very difficult to observe and hardly available at present, for example genetic constitution or physical robustness against disease. All these factors can have an impact on mortality and result in an individual health constitution that is not observable. It is called frailty.

“Frailty, z , represents combined effects of genetic, environmental, and lifestyle characteristics of the individual upon his/her risk of senescent mortality. These characteristics are presumed to remain relatively stable over the age range of the study [age 50-99].” (Horiuchi and Wilmoth 1998:400)

Whereas a wide definition of frailty could be that frailty is the result of all unobserved factors that influence mortality, we must be stricter for logical and analytical reasons and make the following assumptions:

1. All socioeconomic factors are either measured by the available covariates or they sufficiently correlate with them, so that they are indirectly included in the model and controlled for.
2. All socioeconomic factors that had an impact on health during the life course before the age of 59 and those that influence the time of death are also sufficiently correlated with the variables we observe for ages above age 59.
3. Independent of this life-long universe of socioeconomic factors, each individual has a more or less favorable fixed genetic constitution which partly determines his or her frailty.
4. Frailty can also be acquired, i.e., it can also be determined by environmental factors during the life course until age 59. Opposed to the socioeconomic factors included in the model, these other environmental factors are not systematically related to socioeconomic status.
5. The incorporation of influences that may affect frailty mostly happens before age 59, so for simplicity, frailty is kept constant from age 59 until death.⁵⁵ Using a constant

⁵⁵ Yashin et al. (1994) discuss the two opposed models of fixed versus acquired frailty and find that the two theoretically different models fit their mortality data equally well. This shows that empirical evidence for either a predominant genetic or environmental component of frailty is difficult to obtain.

frailty above age 59 does not mean that susceptibility to disease and death is constant over age. Of course it increases with age, but this increase is already captured by the baseline hazard that increases with age.⁵⁶

These assumptions, especially the assumption that social variables are more or less observed and biological variables are not, may seem to be artificial because of the borderline between factors influenced by the social status and other factors. But somewhere this borderline must lie and conceptually it is important to be aware of this difference. Moreover, the theoretical setting of such a difference is necessary because, although social and biological factors jointly determine health and mortality, reasonable assumptions about their differences are the only way to analyze their complex interplay. If the above definition of frailty by Horiuchi and Wilmoth was applied literally, it would cause a problem for the analysis of socioeconomic mortality differences: the mechanism of selective survival that leads to decreasing mortality differences between two groups in high ages is only plausible and only works if frailty is distributed independent from the measured socioeconomic status. This interplay is described by Mayer and Wagner (1996:273):

“[it is] plausible, that higher rates of morbidity and mortality in lower status groups lead to the survival beyond age 70 of relatively healthy individuals in these groups. By that, these [groups] become more similar to [...] other social groups.”

Concerning this example: if we assumed that high frailty is caused by low social status (acquired frailty), it would not be logical to expect selected healthy persons to be in low status groups in old age who have the same or even a lower frailty than persons with a higher social status. At least some determinants of health and mortality must be independent of social status. This does not say anything about the relative importance of social versus other influences on mortality. It just claims that there are social and other determinants of mortality and that, for analytical purposes, it is necessary to estimate, on the one hand, social influences on health and, on the other hand, the possible differences in frailty.

For research that is not about socioeconomic differences this distinction may not be as crucial. Therefore the above definition of frailty by Horiuchi and Wilmoth and the

⁵⁶ More sophisticated models with frailty varying over age or time are discussed in Yashin et al 1985; Yashin and Iachine 1995a and 1995b.

description by Vaupel (2001:10078) cited in Section 5.1 are generally also open to acquired frailty. But, as Yashin et al. (2001:5) point out, frailty is assumed to be independent from other covariates in the model. This implies that frailty does not have a sociological meaning in a strict sense but that it reflects biological variability within social groups. By that it is essential for the analysis of the interplay between social and biological factors.

The most important feature of frailty in a population and the main parameter used to introduce this concept in a statistical model is the distribution of frailty. For the assumption of an individually constant frailty in a population, the absolute level of frailty is not important and could not be expressed on a realistic scale anyway. By convention, the mean frailty is set to 1 at the starting age, which is age 59 in this study. Some individuals have lower frailty and some have higher frailty (e.g., 0.8 or 1.3), which forms a frailty distribution in the population. The mean and the variance of the frailty distribution would logically decrease with age because mortality tends to select the frail individuals first (which decreases the mean), and makes the population more homogeneous (which decreases the variance). A specific frailty distribution, the Gamma frailty distribution, will be introduced in the next section.

The normal way to estimate the amount of unobserved heterogeneity is to use a frailty model. Frailty modeling in general tries to take into account the individual susceptibility to diseases and death in the analysis of survival data. Based on the proportional-hazard model which is explained in Section 7. 5, the individual mortality hazard at age x of an individual with frailty z is equal to the baseline risk at age x times the individual frailty z :

$$\mu(x, z) = z\mu_0(x) . \quad (3)$$

Vaupel et al. (1979) show that the observed average mortality hazard at the population level at a certain age $\bar{\mu}(x)$ is equal to the unobserved individual hazard $\mu(x)$ times the average frailty of those alive at this age $\bar{z}(x)$:

$$\bar{\mu}(x) = \bar{z}(x)\mu_0(x) . \quad (4)$$

Under the assumption of a gamma distributed frailty, $\bar{z}(x)$ is equal to the observed survivorship function s (survival from the starting age to age x) to the power of σ^2 , which is the degree of heterogeneity, namely the variance of the frailty distribution at the starting age:

$$\bar{z}(x) = \bar{s}(x)^{\sigma^2}. \quad (5)$$

In general, the frailty term is used in event-history modeling to account for omitted variables (Yashin et al. 2001:6). Here I interpret it as suggested above, as omitted variables that influence mortality and that are independent from socioeconomic status. Included in the basic model specification from Section 7.5, we obtain:

$$\mu_i(x) = z_i \mu_0(x) e^{c\beta(x)}. \quad (6)$$

The baseline risk depends on age x and there are one or more constant variables c . The interaction between a variable and age is expressed with the coefficient β , which is different for different ages. The best fit of such a model is found in an iterative process of Maximum Likelihood Estimation (MLE).⁵⁷

Frailty models can encounter identifiability problems. A model is identifiable if the parameter values uniquely determine the probability distribution of the data and the probability distribution of the data uniquely determines the parameter values. In other words, only if there is a one to one correspondence between the probability distribution of the data and the values of model parameters is the model identifiable. If the number of unique model parameters is higher than the number of independent pieces of observed information, the model is not identifiable, because there are too many variables given the amount of observable information (Huang 2005).

Even when a model is in principle identifiable, the estimation of unobserved heterogeneity is easier in a multi-process or a multi-level setting. In my case there is only one level, one process, and one event. Therefore, sufficient observation time and sufficient variation in time-varying variables is needed. Additionally, some assumptions

⁵⁷ For a general definition and explanation of maximum likelihood estimation, see Lynch (2001:84) and for the derivation of the likelihood, see Gutierrez (2002:33f).

have to be made: frailty models become identifiable if the shape of the baseline hazard is defined (e.g. Gompertz), and a choice concerning the frailty distribution is made (e.g. Gamma). In principle, frailty models are also identifiable without a defined shape of the baseline risk if observed covariates are included (Yashin et al. 2001:11).

It is misleading to describe the estimation procedure as a simple estimation of the amount of hidden heterogeneity in the data. It is important to note that, as an unobservable quality of the population, heterogeneity cannot be *measured* in a strict sense. The result for z will always depend on the assumptions concerning the model, i.e., the shape of the mortality hazard, the kind of frailty distribution, and the proportionality of the hazards.

“The estimates of frailty distribution depend on the choice of a functional form for $\mu_0(x)$ [...] Two survival models with different ‘degrees’ of heterogeneity describe the same data equally well. An illusion that the ‘amount of heterogeneity’ in the population can ultimately be estimated contradicts the fact that this ‘amount’ is determined by the conditions of identifiability.” (Yashin et al. 2001:12)

Yashin et al. (2001:11) give an example, estimating a value of 0.50 for the variance of the frailty distribution for Swedish females born in 1862 based on a Gamma-Gompertz model (for explanation, see below). However, when they assume that the underlying hazard has the form of a logistic curve, the estimate of frailty is zero.

The model estimation is based on the observable individual characteristics, i.e., time of death and independent variables, and a residual category where unobserved mortality relevant factors are included. These factors constitute the frailty of an individual.

The output of a frailty model gives no information about the individual amount of frailty or about whether a population or a subgroup has a high or low frailty. Rather, the distribution of the frailty in the population is described in terms of the variance of this distribution σ^2 . The higher the variance of the frailty distribution, the higher is the unobserved heterogeneity. In the following I will describe the application of frailty models to simulated data.

9.2 Reasons to use frailty models on simulated data

The use of frailty models is not straightforward. The structure, quality, and quantity of the data all have an impact on the estimation procedure. Applying frailty models with different software packages on my two empirical datasets, I encountered various problems. Therefore, I decided to create simulated datasets. Simulated data offer an opportunity to test and analyze statistical methods and to find out why the measurement of real data may not be satisfactory. In simulated data, observable differences between individuals and also differences in frailty can easily and deliberately be constructed and will be treated by the program as the unobserved heterogeneity that exists in real data. The advantage is that all individual and aggregated information in the simulated data is known and so is the correct result of any estimation procedure. The disadvantage, of course, is that simulated data will never be like real data. Results obtained from simulations can tell us a lot about our methods, but will never replace the empirical results.

The problems that I found with real data are the following:

1. For the HRS data from the USA, both statistical packages that I used (Stata and aML) were unable to identify unobserved heterogeneity in the estimation procedure. The estimated variance of the frailty distribution was almost zero. I have no reason to believe that unobserved heterogeneity in the sample is really close to zero because even in models with very few variables, heterogeneity was not found. This estimation failure is most likely due to the small sample size ($n=9,376$), an insufficient observation time, or insufficient variation in time-varying variables (Panis 2004, 2005).

Thus, the first reason for simulating data was to check under what conditions a program was able to identify unobserved heterogeneity. The frailty models in Stata applied to the much larger Danish data set were more successful but encountered the problem of left-truncation that will be described in the following.

2. Reasons to doubt the capability of a standard software package for showing the correct pattern of socioeconomic mortality differences over age arise from the fact that both of my datasets, as almost all survey and register data, are left-truncated. That means that some people are observed from age 59 onwards and others come under observation at much higher ages. For the latter groups, i.e., the older cohorts, it is unknown how much mortality selection already happened and what frailty distribution

had existed when this old cohort was at the age of 59. Just a positively selected subgroup with low average frailty can be observed.

Stata accounts for left truncation in the manner described in Section 7. 5 and technically, it is possible to include the term for unobserved heterogeneity in the model. But to fully explore the selection hypothesis, a longitudinal perspective must be taken into account, i.e. we need to make assumptions about past mortality experienced differently by the social subgroups of the cohorts included in the observation period. This is necessary to correct for a systematic difference in the decrease of average frailty over age between social groups or generally between groups with different mortality levels.

According to the basic idea of an individually constant frailty, average frailty in a population decreases with age because individuals with high frailty die earlier. This decrease is faster in low socioeconomic groups because mortality is higher. The resulting difference in the average frailty between socioeconomic groups in high age biases the usual measurement of mortality differences. Because of the unobservable past of the old cohorts, left-truncation is an additional problem for the estimation of unobserved heterogeneity. The way in which Stata addresses the combined problem of unobserved heterogeneity and left-truncation is described in Gutierrez (2002:42).

To see if Stata really accounts for left-truncation also in terms of the changing frailty distribution and in cases where we have possibly changing mortality differences over age, it was necessary to run these models on simulated data where it is easy to simulate the same data with and without left-truncation. In the next section, the data simulation will be explained.

9.3 Data simulation

This section is structured in eight steps showing the data simulation in detail and giving a description of the resulting dataset.

1. An arbitrary but large enough number of persons are created, in my case 1,000,000, which is comparable to the Danish data for only one sex.⁵⁸

⁵⁸ In some models, I used a simulated dataset of only 10,000 persons to have almost the same size as in the U.S. dataset in order to see if the size of the sample as such makes a difference for the estimation of frailty. The results (not shown) suggest that a smaller sample does not prevent estimation, but it does give less significant results. Therefore in the following, the large Danish dataset will be compared to a large simulated dataset.

2. The year and month of birth are each chosen randomly between January 1891 and December 1920. This is comparable to the Danish data, with the difference being that in the Danish data I also included younger cohorts that had their 59th birthdays later than 1980.

3. Randomly, each individual is assigned a value for frailty.⁵⁹ This value comes from a single draw from a random variable z which is assumed to be gamma-distributed starting at the age of 59. The mean of z at the starting age is set to 1. The random variable z follows the gamma distribution at all ages, but the mean and the variance change with age due to selective survival. For a description and discussion of the gamma distribution and the chosen values for its variance, see below.

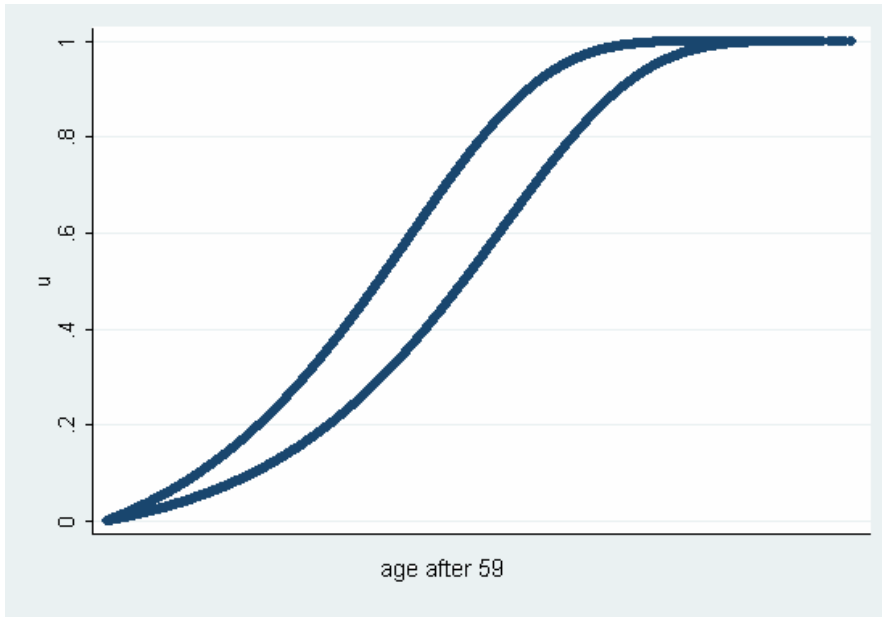
4. The population is divided into two halves, one is rich and the other is poor. The poor individuals are assigned a higher α ($\alpha = 0.012$) in the Gompertz-model, which means that they have a higher intercept thus a higher mortality at all ages (α for the rich=0.006). The second parameter β which determines the steepness that the mortality increases with age, is the same for rich and poor, namely 0.11. These are plausible assumptions taken from large-scale empirical parameter estimation (Thatcher et al. 1998, Appendix p.11). With the following formula, the inverse of the cumulative distribution function (cdf) of the Gompertz-model, the individual life spans after age 59 are computed.

$$\text{Remaining life-years after age 59} = F^{-1}(t) = \frac{1}{\beta} \log\left(1 - \frac{\beta}{\alpha} \log(1 - u)\right)$$

Since α and β are the same for all individuals of one social group, all persons in one social group would die at the same age. Only the term u makes a difference between individuals: it represents random numbers between 0 and 1. The following Figure 9.1 shows how u and the life span above age 59 are related. Given the cumulative distribution function $F(t)$, which is related to the survival function according to $F(t)=1-S(t)$, randomly assigned values for u , on the y-axis, correspond to a certain age at death.

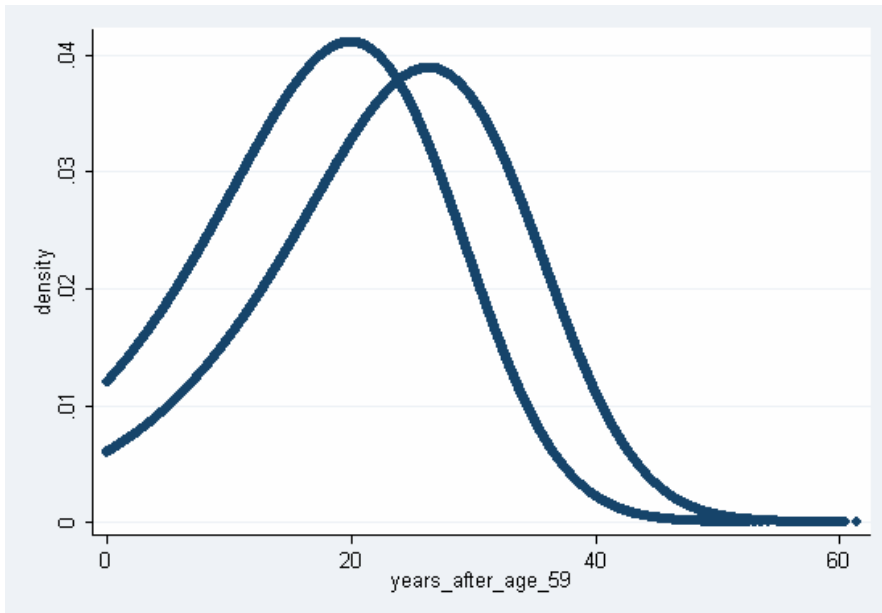
⁵⁹ Randomly means that the computer assigns a value to each person by random selection. It means first that each person has the same chance to have a certain degree of frailty and second that this frailty is independent of this person's characteristics concerning other variables.

Figure 9.1: Cumulative density functions for two socioeconomic groups



This results in two probability density distributions for rich and poor persons, shown in Figure 9.2. They can be understood as the number of deaths at a certain age, like Figure 1.5 in Section 1.3.

Figure 9.2: Probability density functions for two socioeconomic groups



The actual mortality hazard is obtained by $\mu(t) = \frac{f(t)}{S(t)}$ and is shown in Figure 9.3.

Figure 9.3: Hazard functions for two socioeconomic groups (arithmetic scale)

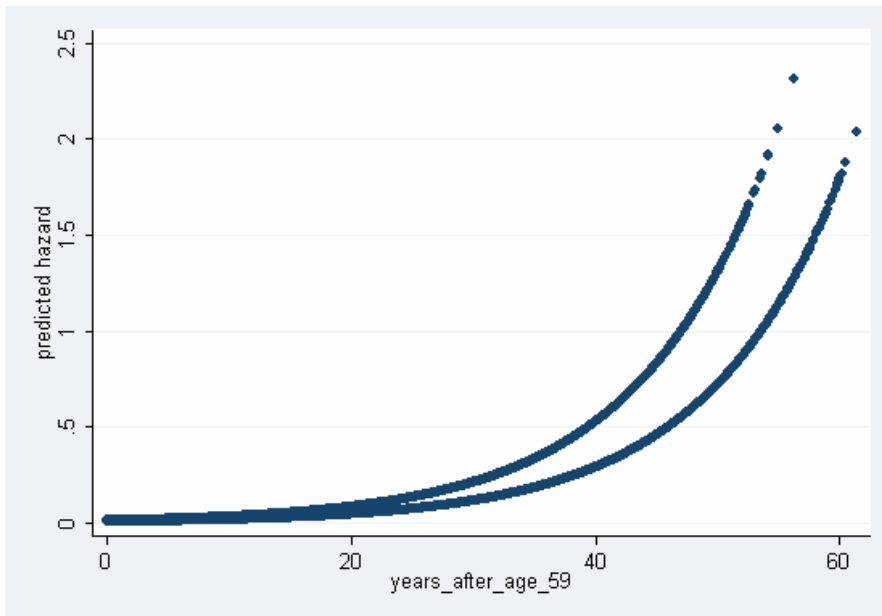
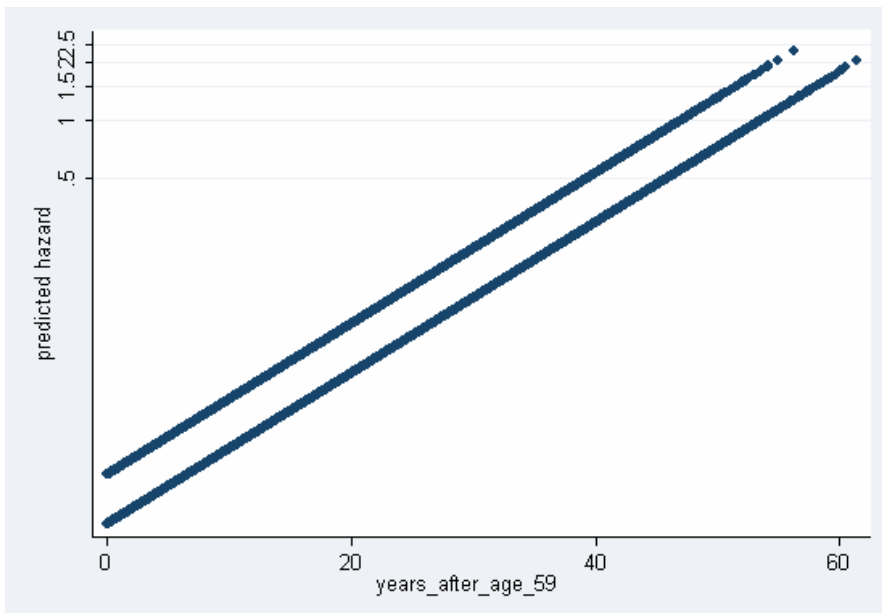


Figure 9.4 below shows the same hazard functions but on a logarithmic scale. With this scaling, the lines become two straight lines with a constant distance between each other representing the constant mortality ratio between rich and poor over age.

Figure 9.4: Hazard functions for two socioeconomic groups (log-scale)



5. As in the real data, the observation of the simulated individuals starts at age 59. Different versions of this simulated dataset will be used for analytical purposes: first is the ideal version where all individuals are observed from age 59 to their death. In this case there is no left-truncation or right censoring. A second version simulates left-

truncation by observing all individuals from 1980 onwards, which is also the start of observation in Denmark. By that some individuals will be observed from age 59 but others will come under observation at much higher ages, e.g., 95. For the latter group, it is unknown how much mortality selection has happened, which is exactly the problem with both real datasets used in this study. Additionally, right censoring is simulated by stopping the observation in the year 2000.

6. Since the change of social mortality differences over age is the desired information, this pattern must be included in the simulation. As described above, in the simplest version of the data, $\alpha_{poor} = 2 \times \alpha_{rich}$. This means that the hazard ratio between these two groups is 0.50 at all ages, i.e., rich people have 50 percent lower mortality than poor people. In a more complicated version of the dataset there is an interaction between age and income in the sense that the mortality differences between the two social groups decrease with increasing age.⁶⁰

7. The next step is the inclusion of frailty in the data simulation. The idea that some individuals have higher frailty and some have lower frailty implies that frailty in a population follows a distribution. The gamma model for the frailty distribution has been used by numerous researchers (e.g., Manton and Stallard 1981; Vaupel and Yashin 1983). The other common distribution, inverse Gaussian, was introduced as a frailty model by Hougaard (1984). Manton et al. (1986:637) claim that both distributions have the special and advantageous feature that the distributions will have the same mathematical form even after the frailer individuals have died. This is especially important in the case of left-truncated data, where it is necessary to make assumptions about the initial frailty distribution in a cohort. The mean frailty declines with age in both models as well (ibid.) The difference between the two distributions is that the coefficient of variation, which is the standard deviation, divided by the mean, declines for the inverse Gaussian but is constant for the gamma model.

Manton et al. (1986:639) compare the gamma and the inverse Gaussian frailty distribution and find a better fit to human mortality in models with the gamma

⁶⁰ The exact Stata codes for all steps of the simulation can be found in the appendix.

distribution. They study high quality Medicare data from the USA from ages 65 to 94 and estimate values for the amount of unobserved heterogeneity.⁶¹

Manton et al. measure the unobserved heterogeneity for different combinations of hazard models (Gompertz vs. Weibull) and frailty distributions (inverse Gaussian vs. Gamma). These different combinations also have different levels of heterogeneity, which implies that the assumption about a reasonable value for the variance of the frailty distribution is specific to the model specification. These values can also only be compared between models based on the same assumptions. I chose the Gompertz baseline because it is a widely used function that has been proven to fit mortality data in old age sufficiently well. Second, I decided to use a gamma distribution for the frailty distribution because it has been shown to fit better to mortality data than the inverse Gaussian distribution (Manton et al. 1986:639) and because it has a very flexible shape. The choice of the hazard model makes much more difference to the amount of unobserved heterogeneity than the choice of the frailty distribution does. Manton et al. conclude:

“the estimated coefficients of variation at about age 90 years are also relatively insensitive to the selected form of the frailty distribution. The bias generated in estimating β by ignoring heterogeneity appears to be greater than the bias induced by selecting a reasonable model of the frailty distribution. (Manton et al. 1986:641)”

In other words, it is better to take unobserved heterogeneity into account although the choice of the distribution may be difficult and not always definite, than not to take it into account.

The so-called Gamma-Gompertz Model, i.e., the combination of the Gompertz Model for the mortality increase with age and the Gamma distribution for the frailty, is frequently described and used for the analysis of unobserved heterogeneity (e.g., Manton et al. 1981 and 1986; Horiuchi and Wilmoth 1998). The model that is supposed to describe mortality only after age 59 takes two factors for the individual mortality into account. The first is the Gompertz-shaped mortality hazard for rich versus poor individuals. These two mortality levels are for “standard” individuals with a frailty of 1. Second, the individual amount of frailty, z , is taken into account, which is independent of age and income and follows a gamma distribution.

⁶¹ To express heterogeneity, they use the squared coefficient of variation, which is equal to the variance only if the mean is 1. If the mean frailty gets below 1 in a more and more selected group in very old age, the squared coefficient of variation is higher than the variance.

8. To draw random individual values for frailty from the gamma distribution, it is necessary to define the variance of this distribution which represents the amount of heterogeneity in the population. For my preferred combination, Gompertz baseline and Gamma frailty, Manton et al. (1986:640) find squared coefficients of variation of 0.211 for males (S.E.= 0.015) and 0.288 for females (S.E. = 0.016). The coefficient of variation is defined as the standard deviation divided by the mean ($cv = \frac{\sigma}{x}$). It follows

that the coefficient of variation is equal to the standard deviation if the mean frailty is 1. In an earlier work, Manton et al. (1981) found similar results.⁶² These are comparable to those from Manton et al. (1986), namely 0.254 and 0.352 for the USA (men and women respectively) and 0.313 and 0.358 for Sweden (men and women respectively)(Manton et al. 1981:399).

Horiuchi and Wilmoth (1998:402) apply a model that is slightly more complicated, considering two types of mortality: background mortality and senescent mortality.⁶³ Their results are values of 0.080 and 0.160 (men and women) in Sweden and 0.142 and 0.188 (men and women) in Japan.

Based on the same parametric approach, Barbi (2003:7) finds a value of 0.097 for Italian men and 0.147 for Italian women. In all three studies, unobserved heterogeneity is larger for women than for men, which could be the result of lower female mortality, which leaves women less selected and thus more heterogeneous. The following table summarizes the empirical findings.

Table 9.1: Results for the variance of the frailty distribution from the literature

	men	women
Manton et al. 1981, USA	0.254	0.352
Manton et al. 1981, Sweden	0.313	0.358
Manton et al. 1986	0.211	0.288
Horiuchi and Wilmoth 1998	0.080	0.160
Barbi 2003	0.097	0.147

⁶² Their heterogeneity parameter k has to be transformed with $\sigma^2 = \frac{1}{k}$, where k is the slope parameter of the frailty distribution. See appendix for the gamma distribution.

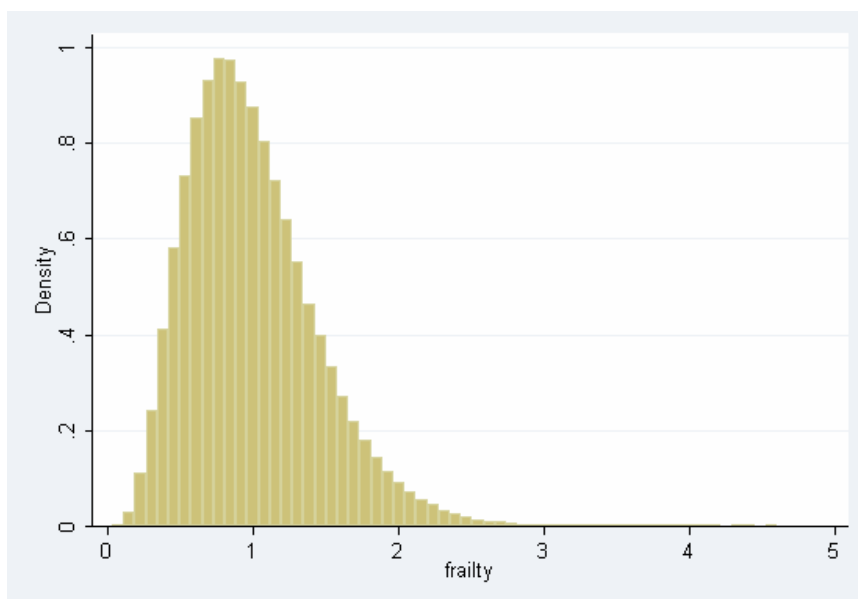
⁶³ From their results for parameter α one can compute the variance of the frailty distribution with the same transformation as above ($\sigma^2 = \frac{1}{\alpha}$).

Given these results from the literature, it is safe to assume a variance of 0.2, which is in the lower range of the results above. Data can be simulated based on this chosen value. Given the mentioned basic problems for the estimation and quantification of unobserved heterogeneity, it is safer to choose a relatively low level for this simulated demonstration. For simplicity, I will not differentiate between men and women.

The advantage of this simulated data for my analytical purposes is that the following features of the hypothetical population are known:

1. The hazard ratio of the two social groups in the population is 0.50, with the rich persons having 50 percent lower mortality. In another version of the dataset, the hazard ratio declines by 5 percent with every ten years of age, 0.50, 0.55, 0.60 and finally 0.65 for above the age of 90. Thus, there is mortality convergence between social groups.
2. The degree of unobserved heterogeneity is 0.2, defined as the variance of the frailty distribution. In my simulation with 1 million individuals this Gamma distribution has a variance of 0.2 and ranges from the minimal individual value of 0.033 to the maximum of 4.600. By definition, it has a mean of 1 at the starting age. The shape of the Gamma distribution is very flexible: the lower the variance, the more symmetric and the more similar the Gamma distribution is to a normal distribution. Figure 9.5 shows that most persons have a frailty of about 0.8 and very few have very high values of 3 or more.

Figure 9.5: Gamma Frailty distribution



The simulated data has the following mortality features: age at death ranges from 0.00028 months (=12 minutes) after the 59th birthday to 61 years after the 59th birthday, which is age 120. This extreme age is caused by the random simulation of 1 million persons, where statistically some people reach extreme ages.⁶⁴ The mean age at death is 80.2 which is slightly higher than life expectancy for women in Denmark (80.0 in 2003) and still lower than overall life expectancy in Japan that was 82.0 years in 2002 (Human Mortality Database).

9.4 Frailty models with simulated data

Table 9.2 and Table 9.3 show the results of seven different event-history models with increasing complexity:

Model 1 only includes the binary variable for income (rich vs. poor).

Model 2 includes income and controls for unobserved heterogeneity.

Model 3 is like Model 2, but is applied to left-truncated and right-censored data.

Model 4 is applied to a dataset where the hazard ratio between rich and poor decreases with age, and therefore includes an interaction between income and age to reveal these differences over age, like the figures for the interaction between income and age in Section 8. 4.

Model 5 is like Model 4 and additionally controls for unobserved heterogeneity.

Model 6 is also based on data with changing hazard ratios over age. Additionally the data is left-truncated and right-censored.

Model 7 is like model 6, but is a constraint model that imposes a fixed value for the degree of unobserved heterogeneity, namely exactly the amount of unobserved heterogeneity that has been imposed on the data during the data construction.

⁶⁴ However, this extreme age was surpassed in reality by Jean Calment, the oldest person, who was 122.5 years old when she died in 1997.

Table 9.2: Results from Model 1 to 3 (simulated data with constant mortality ratio between rich and poor of 0.50)

	Model 1		Model 2		Model 3	
	Without control for unobserved heterogeneity		With control for unobserved heterogeneity		Left truncated data, with control for unobserved heterogeneity	
	CI (95%)		CI (95%)		CI (95%)	
cases	1,000,000		1,000,000		663,482	
failures	1,000,000		1,000,000		520,417	
person-years	21,188,969		21,188,969		7,721,473	
poor	1		1		1	
rich	0.55	0.55-0.55	0.50	0.50-0.50	0.50	0.50-0.51
theta (σ^2)			0.202	0.197-0.207	0.203	0.194-0.212
log-likelihood	-767975		-763307		-188727	

Model 1 suggests that rich persons have a 45 percent lower mortality than poor ones. However, Model 2 that controls for unobserved heterogeneity reveals the true mortality relation, namely 50 percent lower mortality for rich persons as it was defined in the data simulation. It also reveals the degree of heterogeneity that was imposed to the data, namely $\theta = \sigma^2 = 0.2$. The substantial deviation from 0.50 in Model 1 is the effect of this unobserved heterogeneity.

Model 3 is based on left-truncated and right-censored (LTRC) data. Left-truncation can be a substantial problem for event-history analysis and especially for the analysis of mortality selection. As explained above, left-truncation means that for some individuals, observation begins in later ages than for others. For those coming under observation e.g., at age 80, it is unknown which individuals of this cohort already died. Normally, both the observable and unobservable characteristics of persons who died before the observation starts are unknown. But this knowledge is necessary, first, to assess the degree of mortality selection and to know how selected the 80-year old survivors are, and second to correct the bias that is due to mortality selection and that was shown in the above models.

I use the version of the simulated data described above where the persons are born between 1891 and 1920, as in the first dataset, but observation starts for all persons in 1980, with the consequence that the observation starts at different ages. The following models are performed to test whether Stata is able to reconstruct the mortality experienced differently by rich and poor persons in the past, i.e., before observation. Of course, Stata cannot observe the unobservable; it rather models the unobserved mortality of persons with unknown characteristics based on the observed mortality from age 59 onwards. The goal is to reconstruct the frailty distribution as it existed among the

left-truncated cases when they were at the starting age of 59. Technically, the data must be setup for Stata with a differentiation between the start of the risk, age 59 where the mortality risk is assumed to begin in the model, and the start of observation. The start of observation is the age at which a person is first observed. Therefore, there are far fewer than one million persons included because many persons already died before 1980. Not all of the persons die until the year 2000, when the observation stops. Consequently there are fewer deaths than cases in Model 3.

The result of Model 3 is that Stata is able to take left-truncation into account. It gives the same correct results as in Model 2.

Table 9.3: Results from Models 4 to 7 (simulated data with an increasing mortality ratio between rich and poor every 10 years of age: 0.50, 0.55, 0.60, and 0.65; all models include an interaction between income and age)

	Model 4		Model 5		Model 6		Model 7	
	Without control for unobserved heterogeneity		with control for unobserved heterogeneity		left truncated data, with control for unobserved heterogeneity		left truncated data, constraint model with $\sigma^2 = 0.200$	
	CI (95%)		CI (95%)		CI (95%)		CI (95%)	
cases	1,000,000		1,000,000		670,680		670,680	
failures	1,000,000		1,000,000		519,424		519,424	
pers.-years	21,493,474		21,493,474		7,907,918		7,907,918	
poor	1		1		1		1	
rich (59-69)	0.50	0.50-0.51	0.50	0.49-0.50	0.51	0.50-0.52	0.50	0.49-0.52
rich (70-79)	0.58	0.58-0.59	0.55	0.55-0.56	0.58	0.57-0.58	0.57	0.56-0.57
rich (80-89)	0.67	0.67-0.67	0.60	0.59-0.60	0.66	0.65-0.66	0.63	0.62-0.63
rich (90+)	0.78	0.77-0.79	0.64	0.63-0.66	0.76	0.74-0.77	0.71	0.69-0.72
theta (σ^2)			0.206	0.194-0.219	0.069	0.060-0.078	0.200	
log-likelih.	-779756		-777826		-202814		-203195	

Models 4 to 7 in Table 9.3 all include an interaction between age and income and are all applied to a data set where such an interaction is built-in. From the data construction, it is known that the hazard ratio between rich and poor declines with age. It is 0.50 from age 59 to 69, 0.55 from age 70 to 79, 0.60 from age 80 to 89 and 0.65 at higher ages.

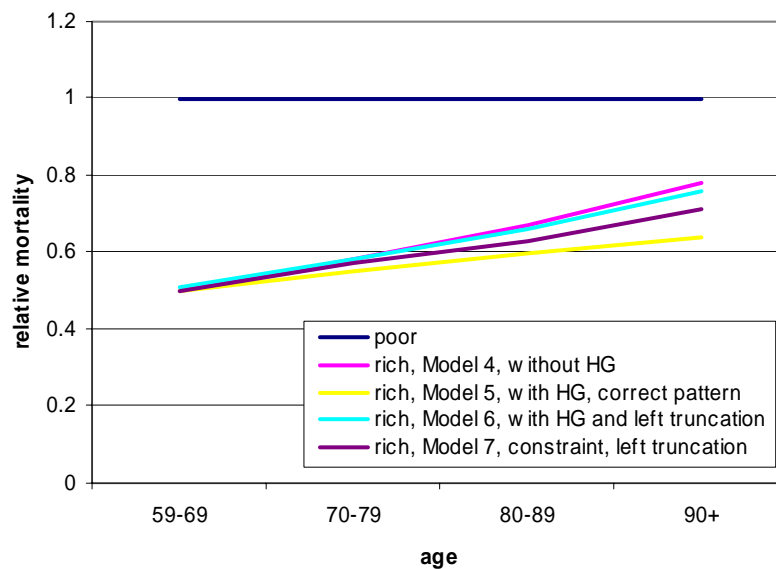
Figure 9.6 below plots the results of Models 4 to 7. The output of the four models is the hazard ratio between rich and poor persons in four different age groups. The question is whether the correct pattern over age can be revealed when controlling for unobserved heterogeneity.

Model 4 simply includes an interaction between age and income. The results of this model (pink line) are wrong, because they show a much stronger convergence over age

than is constructed in the data. Model 5 controls for unobserved heterogeneity and reveals the correct pattern over age and the correct amount of heterogeneity.

Model 6 is the same model, only applied to left-truncated data. Due to left-truncation, Stata is unable to estimate the correct amount of heterogeneity. It estimates 0.069 instead of 0.2. Accordingly, the hazard ratios for higher ages, which suggest a strong convergence over age, are wrong. One way to deal with this failure to correctly estimate the degree of heterogeneity is to impose a fixed value for the variance of the frailty distribution in a constraint model. The model is then estimated under the condition that there is a certain amount of heterogeneity. This “assumption” about the amount of heterogeneity is unproblematic because the amount of heterogeneity in a Gompertz-Gamma model is known from the data simulation. Model 7 shows the results of this attempt. Under the constraint where $\sigma^2 = 0.2$, the estimation process still finds the wrong parameter values. The red line of Model 7 is closer to the correct pattern (the yellow line), but it is still wrong.

Figure 9.6: Mortality differences between rich and poor at different ages according to Models 4 to 7, simulated data (HG=controlled for unobserved heterogeneity)



Under the condition of left-truncation it is not possible to estimate the correct amount of heterogeneity if there are changing hazard ratios over age and if a model with an interaction between age and income is used to detect them. Nor is it possible to impose the correct value for the frailty distribution in a constraint model in order to obtain correct parameter estimates.

The seven models in this section show that if there is unobserved heterogeneity in the data, it biases the age pattern of mortality differences. Stata is able to correct for this heterogeneity and to show the correct pattern unless there are changing hazard ratios with age (which is most likely the case in real data) combined with left-truncation (which occurs in most survey or register data).

9.5 Frailty models with real data: left-truncated data compared to single cohorts from Denmark

There are fundamental differences between the simulated and the real data: first, the distribution and the variance of frailty in the real population is not known as in the simulated data and, second, the exact shape of the baseline mortality hazard is unknown. The Gompertz-model that fits the data well is only an approximation. But still, the estimation of unobserved heterogeneity in real data is possible under certain conditions as discussed in Section 9. 1. Only based on an estimation of unobserved heterogeneity is it possible to correct the bias in the measurement of social mortality differences over age.

The analysis in this section applies frailty models to the large left-truncated dataset from Denmark. Then the same models are applied to single cohorts (without left-truncation) in order to rule out the impact of left-truncation, and next the results are compared. A single cohort can be observed for as many years as the data set allows it. In our case this is 23 years, from January 1980 to December 2002. For men, the cohort born in 1914 has been chosen because the members of this cohort will consequently be followed from age 65 to 88, the age range where most men in Denmark died. In this age range with the highest number of deaths, the compositional change is also supposed to be highest. For women, this is the age range from 71 to 94, thus I chose the cohort born in 1908 in order to observe as much compositional change as possible in the observation time from 1980 to 2002. The resulting two datasets have the following features: the cohort of men born in 1914 includes 23,169 persons, of which 19,748 or 85.2 percent die during observation. There are 23,386 women born in 1908 and 20,965 or 89.6 percent of them die during observation.

In the following, the analysis of each of these two cohorts will be presented in three steps: first, in order to compare left-truncated data with cohort data the normal dataset

with many different cohorts will be analyzed, as was done in Section 8. 4, but in the narrow age ranges mentioned above and with only two income categories. Second, the single cohort will be analyzed accordingly and, third, a model that controls for unobserved heterogeneity will be applied to see if heterogeneity can be estimated and if the age pattern of social mortality differences changes after taking unobserved heterogeneity into account.

Figure 9.7 is based on a model that controls for other socioeconomic variables just as in the models and figures in Section 8. 4. But in order to allow a better overview of even small differences in the age pattern, the model uses only two income categories. Moreover, the age range is narrower. This figure is supposed to remind us that there is only a small convergence because the age range is smaller than in the original analysis in Section 8. 4.

Figure 9.7: Male mortality with interaction between age and income, Denmark (low income=1) left-truncated data

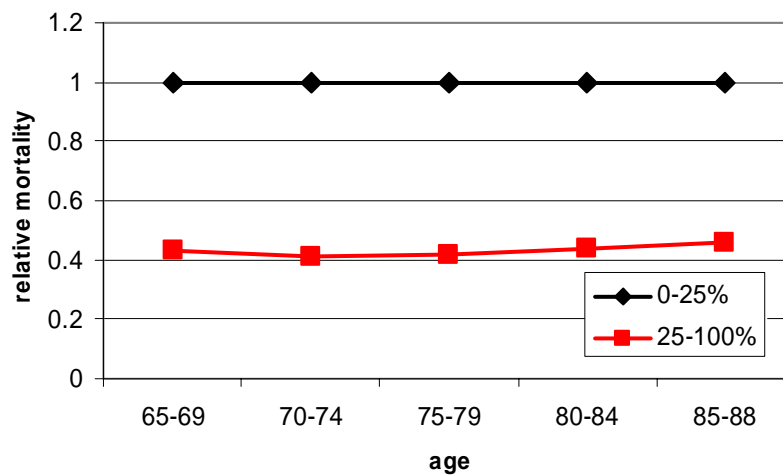


Figure 9.8 below is based on the dataset of one single cohort. The mortality convergence around age 80 is significant, although it seems to be a feature of this very cohort because it cannot be found in many other cohorts (results not shown). Thus the age pattern of mortality differences in this specific cohort, or in any other single cohort, is not of special interest here. The next step is the model that controls for unobserved heterogeneity. In this model, a degree of unobserved heterogeneity of $\sigma^2=0.115$ is estimated (CI=0.055-0.241). The corrected line in the figure is only slightly different from the uncorrected line, the rate ratio for the richer group in the age group 85-88 being 0.68 instead of 0.70, which is a non-significant difference.

Figure 9.8: Male mortality with interaction between age and income, Denmark, one cohort (low income=1, HG=controlled for unobserved heterogeneity)

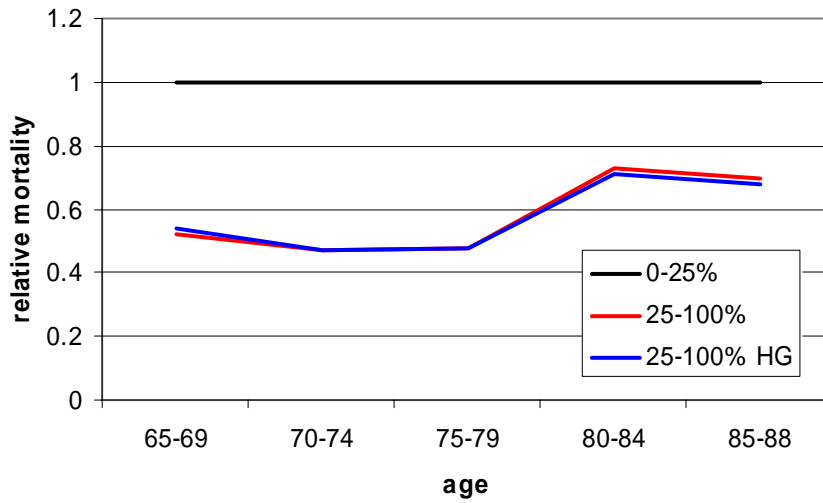


Figure 9.9 below gives the same information as Figure 9.7, i.e. based on left-truncated data, but for women. For women, a different age range is chosen to analyze mortality selection because deaths of women occur at higher ages than for men.

Figure 9.9: Female mortality with interaction between age and income, Denmark (low income=1), left-truncated data

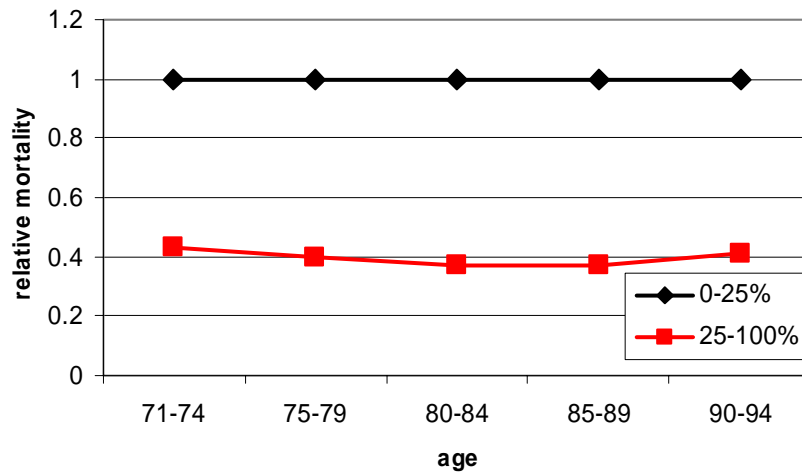
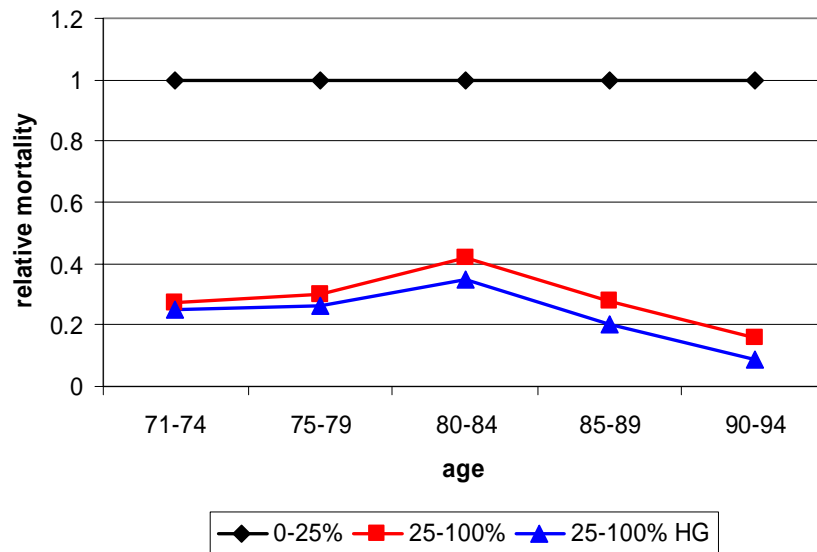


Figure 9.10 below is based on another single cohort, namely women born in 1908. Just like men born in 1914, this cohort shows an age pattern that is different from the overall pattern over age. Again, the focus is not on the pattern over age in this specific cohort, because this pattern seems to vary substantially between single cohorts and between men and women. This cohort is taken as an example and the focus is on the change that

occurs when unobserved heterogeneity is controlled for. In this cohort an unobserved heterogeneity of $\sigma^2=0.627$ (CI=0.472-0.833) is estimated. Accordingly, the deviation of the corrected from the uncorrected line in Figure 9.10 is larger and statistically significant at the 95 percent level in the last three age groups.

Figure 9.10: Female mortality with interaction between age and income, Denmark, one cohort (low income=1, HG=controlled for unobserved heterogeneity)



Even in cases where much unobserved heterogeneity is found (0.627), the deviation is relatively small. But it is important to note that the relatively small deviation of the corrected age pattern from the biased age pattern is partly due to the fact that we only observe 23 years of age. An age range of 40 years would reveal a higher impact of unobserved heterogeneity.

This section has confirmed the results from the simulated data, namely that the estimation of unobserved heterogeneity and an according correction of the age pattern of social mortality differences is possible if left-truncation is absent. In the former section this absence was due to the ideal conditions in the simulated data set. In this section left-truncation was excluded by using single cohorts. In many situations these solutions are not available. Therefore, the following Section 9. 6 suggests a simple method to circumvent the problem of left-truncation.

9.6 A new method to take unobserved heterogeneity into account (and its comparison to a Stata model)

The previous section has shown that left-truncation is a substantial obstacle for the application of frailty models and for taking into account the impact of unobserved heterogeneity on the age pattern of (social) mortality differences. Because almost all survey or register data is left-truncated, this is a substantial problem which may only start to disappear in many years, when existing panel surveys and registers will have existed long enough to follow persons through their whole period of senescence.

In the following, I suggest a method to take this impact into account that works without a statistical model and that can also easily be applied to left-truncated data. This method is a simplified version of what Stata does when it estimates an interaction between age and income controlling for unobserved heterogeneity. Since this desirable way to run a statistical model does not work with left-truncated data, I propose a simple method that can be used for any dataset from which a certain amount of unobserved heterogeneity is known or can be assumed.

I will present this method and show how it works by applying it to simulated data and then comparing the results to the results of the Stata model (in this section). This allows us to evaluate the procedure because in simulated data, the correct outcome is known. Finally, this method is applied to real Danish data (next section). I will not apply the method to the HRS data because the principle is the same and the Danish data are generally of much better quality.

The method works as follows: in order to reveal the pattern of mortality differences between two groups over age, for each group the survival function and the mortality hazard in the lexis-trapezoid a) in Figure 9.11 is computed directly from the data.⁶⁵ An amount of unobserved heterogeneity is chosen that either has been estimated empirically from the data or has to be assumed based on theoretical considerations or empirical findings from other datasets. I will keep the value 0.2 that was used in the examples above. This allows us to compute $\bar{z}(x)$ in equation (5) and then $\mu(x)$ in equation (4), which represents a hazard net of the impact of frailty. With equation 7, this

⁶⁵ To compute the survival and the hazard function, I used the predict-command after the model estimation in Stata (with the streg-command). Since the baseline hazard is specified as Gompertz, the predictions will also be based on a Gompertz hazard. For a detailed description of the calculation and formulas, see Stata Corporation (2005:228). With only small differences, survival and hazard functions from a life-table could be used as well.

is done for the next ten years of age, but the frailty from the first age group is multiplied by the frailty from the second age group:

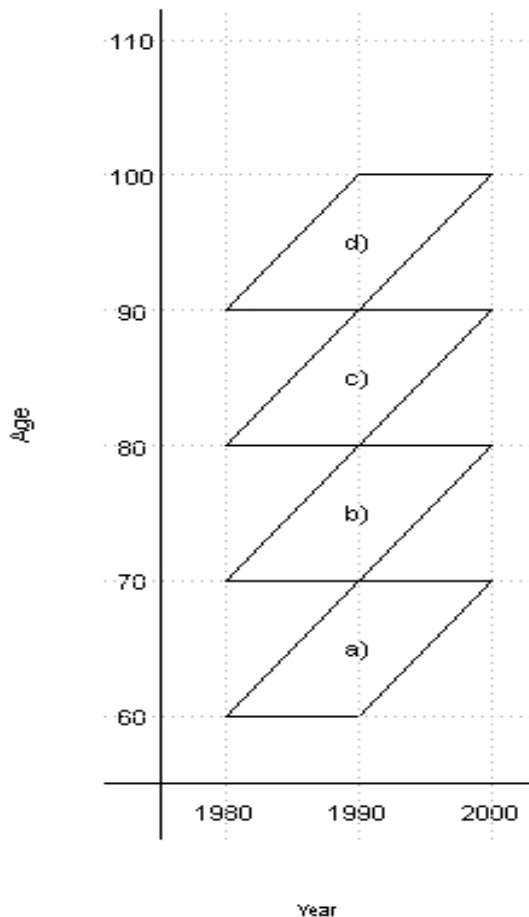
$$\bar{\mu}(x) = \bar{z}(x)\mu(x), \quad (\text{equation 4 from Section 9. 1})$$

$$\bar{z}(x) = \bar{s}(x)^{\sigma^2}, \quad (\text{equation 5 from Section 9. 1})$$

$$\bar{\mu}(x+10) = \bar{z}(x)\bar{z}(x+10)\mu(x+10). \quad (7)$$

This calculation is repeated for the third age group, then the fourth, and so on until the last age group, and each time all values for the frailty from the younger age groups are multiplied. The number of steps depends on the number of age groups. One assumption has to be made, namely that the divergence of frailties occurring from ages 60 to 70 (in lexis-trapezoid a) in Figure 9.11) because of different selective forces in different socioeconomic groups, is the same as the process that happened to the persons in lexis trapezoid b) before they were observed (the assumption of a synthetic cohort). The same values for heterogeneity are used for different age groups, even if this may be a simplistic assumption given the theoretical understanding that heterogeneity decreases with age by selective mortality.

The logic of my approach is analogous to a synthetic cohort, which lets us reconstruct the differential change of frailties in different social groups over the whole age range of the sample. This approach is able to reveal the corrected and thus higher social mortality differences at older ages.

Figure 9.11: Lexis Diagram

At the beginning of Chapter 9, I described my reasons for believing that the normal way of analyzing mortality differences across age and, correspondingly, the figures in Section 8.4 underestimate socioeconomic mortality differences at high ages. This bias may result in a converging pattern over age that is entirely due to selection processes, and not due to a decreasing impact of social status on mortality at the individual level.

In the following, I will compare the correction effect of the proposed method with the ideal correction that Stata is able to achieve in case of non-truncated data. The simulated dataset without left-truncation that is the basis for Model 5 is used because this is the model where Stata showed the correct pattern over age. The proposed method is insensitive against left-truncation and should reveal the same results as Stata does.

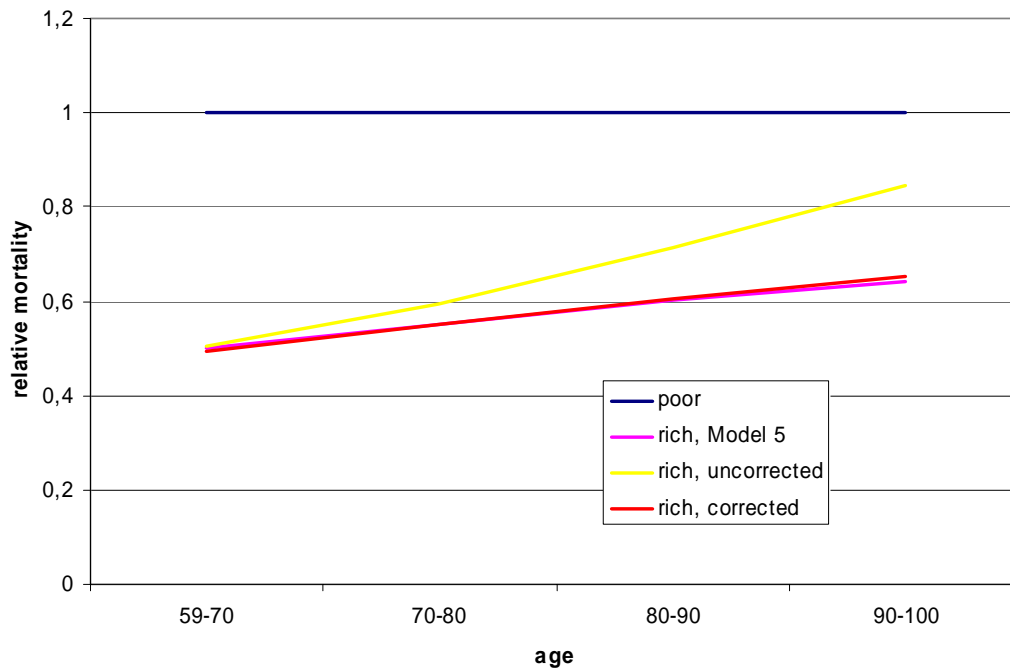
Table 9.4 contains the necessary information to apply each step of the procedure. From the left to the right side of the table there is the age group, the survival from the beginning to the end of each age range, the hazard rate (deaths divided by exposures),

the assumed degree of heterogeneity σ^2 , and the resulting average frailty \bar{z} . The average frailty decreases with increasing age because the individuals with higher frailty die earlier. This decrease is steeper in the poor group because mortality is higher. The next column contains the corrected hazard, which is the hazard divided by the frailty according to equation (4), and for the older age groups it is the hazard divided by the product of the frailties of all younger age groups according to equation (7). The column with uncorrected rate ratios (RR) just contains the hazard of the rich divided by the hazard of the poor for each age group. By that, the rich group becomes the reference category equal to 1 at all ages. The column with corrected RR is the same but based on the corrected hazards. The graph of these numbers can be seen in Figure 9.12.

Table 9.4: Calculation of rate ratios based on an assumed degree of heterogeneity based on simulated data

	Age	Survival	Haz. Rate	σ^2	\bar{z}	corr. Haz.	uncorr. RR	corr. RR
Rich:	59-70	0.881	0.020	0.2	0.975	0.020	0.506	0.493
	70-80	0.708	0.056	0.2	0.933	0.061	0.594	0.551
	80-90	0.402	0.140	0.2	0.833	0.185	0.713	0.606
	90-100	0.131	0.285	0.2	0.666	0.565	0.845	0.651
Poor:	59-70	0.778	0.039	0.2	0.951	0.041	1	1
	70-80	0.551	0.094	0.2	0.888	0.111	1	1
	80-90	0.260	0.197	0.2	0.764	0.306	1	1
	90-100	0.080	0.338	0.2	0.603	0.868	1	1

The lowest (pink) line in Figure 9.12 shows the pattern revealed by Model 5, which is the same as in Figure 9.6 and is correct. The figure also shows the pattern given by the results in the last column of Table 9.4 above (red line). The results from the simple method are almost the same as the results from the sophisticated model. The yellow line shows the pattern obtained by taking the simple hazard ratios for each age, which is the wrong and biased pattern over age.

Figure 9.12: Mortality differences (rate ratios) between rich and poor at different ages, simulated data

The result of this comparison is that the proposed method can be used in the common case when left-truncation prevents Stata or other statistical packages to take the impact of unobserved heterogeneity into account. This method does not require special data quantity or quality.

9.7 Application of the new method to real (Danish) data

To apply this method to the Danish data it was necessary to make a number of simplifications to the data compared to the multivariate time-varying measurement of the six different income groups used for the models in Section 8. 4. I computed the average income over time for each person and divided the population into a poor group, the poorest income quartile, and a rich group, that is, the remaining 75 percent. The alternative, namely to make two groups of 50 percent each, would not be a better option because only the poorest 25 percent really show higher mortality. These changes in the measurement of the socioeconomic status without control variables resulted in a slightly different pattern over age, but Figure 9.13 shows that with the new binary and time constant measurement of income, mortality differences between income groups still

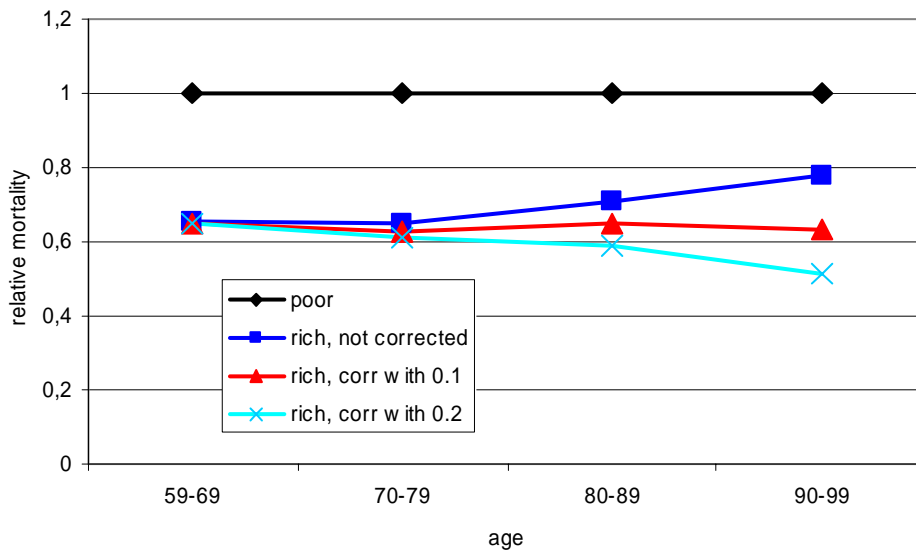
converge in higher ages. In the following, the results of the new method applied to the Danish data are described.

Table 9.5: Calculation of rate ratios based on an assumed degree of heterogeneity based on the Danish data

	Age	Survival	Haz. Rate	σ^2	\bar{z}	corr. Haz.	uncorr. RR	corr. RR
Rich:	59-69	0.879	0.00098	0.2	0.975	0.00100	0.655	0.647
	70-79	0.649	0.00246	0.2	0.917	0.00269	0.646	0.608
	80-89	0.256	0.00689	0.2	0.761	0.00905	0.708	0.590
	90-99	0.016	0.01792	0.2	0.437	0.04097	0.777	0.514
Poor:	59-69	0.822	0.00149	0.2	0.962	0.00155	1	1
	70-79	0.513	0.00381	0.2	0.875	0.00436	1	1
	80-89	0.140	0.00974	0.2	0.675	0.01443	1	1
	90-99	0.005	0.02306	0.2	0.347	0.06655	1	1

Figure 9.13 shows the uncorrected rate ratios (RR), the correction that is based on the assumption $\sigma^2 = 0.2$ (on which the calculations in Table 9.5 are based), and another correction based on the assumption $\sigma^2 = 0.1$.

Figure 9.13: Male mortality with interaction between age and income, Denmark



Even moderate and realistic assumptions about the degree of heterogeneity in a population can have an important impact on the age trajectory of social mortality differences.

As to the method, single calculations in single steps were applied to impose the assumed degree of heterogeneity and to keep track of the consequences and changes. This implies that there is a drawback, namely that the impact of income on mortality is analyzed in a much simpler way than a model such as in Section 8. 4 would do, namely time-constant, dichotomous and univariate. There is a trade-off between observing as much heterogeneity as possible by including many variables in sophisticated models on the one hand, and keeping the procedure simple to be able to observe the decisive changes in unobserved heterogeneity and frailty on the other hand. The first strategy has many advantages with results that can be seen in Section 8. 4. But as shown in the present chapter, the estimation of unobserved heterogeneity is problematic and depends on many data characteristics. So, the simple method is a way to take heterogeneity into account when the models do not work. It may be necessary to use the simplified method in cases where left-truncation and/or a small sample size do not allow a correction as shown above.

The results of this chapter help to identify and understand the problem of left-truncation, which occurs in almost all survey and register datasets and which makes it very difficult if not impossible to estimate frailty and even to impose an assumed value for the variance of the frailty distribution in order to show correct mortality differences in high ages. To relieve this problem a simple method is proposed, successfully evaluated, and applied to the Danish data. This method is based on assumptions similar to those made for a synthetic cohort, but it is robust against the problems of left-truncation. It does not allow estimating unobserved heterogeneity, but if the amount of heterogeneity is estimated, its impact can be shown and corrected.

Summary of Chapter 9

The presented estimations of unobserved heterogeneity and its impact on socioeconomic mortality differences in old age represent the attempt to empirically test argument 4 from Chapter 5. This argument suggests that the observed mortality convergence in old age does not reflect the decreasing impact of social status on mortality, but rather the changed composition in a selected population. This idea is based on the concept of frailty, i.e., unobserved individual factors that influence mortality. This frailty has a certain distribution in the population and on average those individuals with low frailty will survive to high ages. This, in turn, may decrease the observed social mortality differentials.

To show that this mechanism works in principle, I simulate a dataset where the social mortality differences between two different groups (rich and poor) and the “unobserved” characteristics are known. The models show that Stata can correct for unobserved heterogeneity, and show the correct mortality differences in all age groups, net of the impact of mortality selection. However, with left-truncated data this is not possible. This finding is confirmed by the same models applied to real data from Denmark: single cohorts without left-truncation are analyzed in comparison to left-truncated data to show that it is possible to estimate and take into account unobserved heterogeneity if there is no left-truncation. For left-truncated datasets where a correction for the compositional change is needed, I propose a method that works without a statistical model and reveals similar results.

Chapter 10 Conclusion

The main results from Chapter 8 with respect to the age pattern of socioeconomic mortality differences are:

1. Mortality differences between income groups are much larger in Denmark than in the USA. The comparison between these two countries once again illustrates the surprising fact that the level of social inequality does not consistently correlate with the level of social inequality in health or mortality (see discussion in Section 4.2.1). At least the research in this field seems to demonstrate this absence of a clear correlation (e.g., Huisman et al. 2003, 2004).

2. A simple interaction between age and income as an indicator for socioeconomic status shows only a very small convergence of mortality differences with age. In Denmark, where the pattern is much more reliable due to the large data set, the mortality of the upper 75 percent of the income distribution approaches the mortality of the poorest 25 percent only by about 15 percent: from age 59 to age 99 the rate ratio changes from 0.40 to 0.55 for men and from 0.25 to 0.40 for women. It is difficult to compare these findings with other studies from other countries because measures for the amount of convergence are not very common. But it is obvious that the convergence that this dissertation tries to explain is very small.

3. More specifically, I found that socioeconomic mortality differences are stable across age. The slight convergence mentioned above happens as health deteriorates rather than with increasing age. I propose the empirical and theoretical separation of these two different dimensions of aging: increasing age and worsening health. These processes are closely linked, e.g., for almost all persons health deteriorates as age increases. But the difference is that age increases monotonically for everyone whereas health deteriorates very differently in different social groups. The result of this perspective is that poor health is a leveler for social differences in mortality. Increasing age is only a leveler to the extent that it implies worsening health. Simplifying again, it is possible to conclude that social mortality differences decline with age because average health is worse in higher ages.

But the interpretation is not complete by saying that poor health levels out social mortality differences. This is because mortality cannot be separated from health. From a comprehensive perspective on social differences in health and mortality, mortality being

a useful indicator for health, the result is seemingly paradoxical: poor health reduces social inequality in mortality (an indicator for poor health). Naturally, this problem can be solved by considering health and mortality as belonging to one process. People with lower social status do not show much higher transitions rates from poor health to death than persons with a high status because they already incorporated the unfavorable conditions into their relatively worse health.

Besides this social explanation, there may also be a physiological one. It is possible that the development from poor health to death is much more determined and path-dependent than the change from good to poor health. Therefore, social conditions, including the level of medical care, have much less influence on mortality when a person is already ill.

Both underlying explanations allow us to conclude: when health is poor, it is too late to do something about socioeconomic mortality differences. The impact of socioeconomic status and income in a good health status via direct material welfare and income related non-material aspects is higher than its impact in a bad health status via different medical treatments. Thus income, and probably also other aspects of socioeconomic status, are much more important and beneficial when it buys a good life in good health than when it must purchase good medical care and expensive drugs because a person is already ill.

The conclusion for research on the origins of social mortality differences is that the focus on mortality differences is not sufficient for finding its origins. The origins can only be found in health differences. The conclusion for social policy in general and medical care in particular is that investment in prevention is much more effective than investment in treatment, and this is not only so when we want to reduce social health differences but probably also when we aim at improving the overall health status.

The proposed distinction between the age and health dimensions allows us to evaluate the arguments in Chapter 5. Argument 3, which is based solely on the time dimension, seems to be less plausible. But argument 1, suggesting the dominance of physiological processes over social influences, applies to people in poor health. More interesting is the opposition between arguments 1 versus 3 and 5 versus 7 because they support opposite age patterns of social mortality differences (convergence or divergence).

Chapter 9 analyzes whether the impact of unobserved heterogeneity is the key to deciding between these two possibilities and to explaining the observed age pattern of social mortality differences (argument 4). If the convergence is due to mortality selection, then we observe it just because we do not successfully control for unobserved

heterogeneity. The issue is a question of the order of magnitude: does mortality selection really bias the measurement enough to call the observed convergence an artifact? This dissertation provides the following answers. Generally, the magnitude of the bias depends on the amount of unobserved heterogeneity in the data. When we look at a plausible conservative estimation of this amount and at estimation from our data, we see the following results:

Stata models and the proposed simplified method which have been applied to simulated data suggest that the assumed mechanism can produce a large bias. With empirical data the result is mixed: the simplified method shows a bias as large as with simulated data. Stata models could only be applied to a special selection of the real data, namely the two single cohorts. The size of the bias found in these single cohorts is much smaller.

It is unlikely that this difference in the order of magnitude of the bias is due to differences between the simplified method and Stata because both reveal the same results when applied to the same simulated data. The difference between simulated and real data is an unlikely explanation as well, because the simplified method reveals about the same bias for both types of data. It is more likely that selecting only one cohort from the Danish data has an unintended effect on the results beyond the intended elimination of left-truncation. Thus, I consider the results from the analysis of the single Danish cohorts to be less valid than the other results because of the smaller age range, the smaller sample and because only one specific cohort was selected.

The overall conclusion is that unobserved heterogeneity probably causes a large bias in the measurement of social or other mortality differences in older ages. This should be taken into account in research that tries to measure and explain possible changes of mortality differences with age.

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Appendix

A. Additional formulas

Theoretical Gompertz density function:

$$f(t) = \alpha e^{\beta t} e^{-\frac{\alpha}{\beta}(e^{\beta t} - 1)}$$

Theoretical Gompertz density function with Gamma distributed frailty (Vaupel et al. 1979:452):

$$f(x) = \frac{\alpha e^{\beta x} k \lambda^k}{\left(\lambda + \frac{\alpha}{\beta} e^{\beta x - 1} \right)^{\lambda + 1}}$$

Gamma distributed frailty

Vaupel et al. 1979 and Manton et al. 1981 assume a Gamma distributed frailty while Flinn and Heckman 1982 assume a normal distribution. The choice of the frailty distribution is less crucial than the choice between different options for the hazard rate function, discussed in Section 9.3 (Manton et al. 1986:643).

Probability density function (pdf) with gamma distributed frailty:

$$f_0(z) = \frac{\lambda^k z^{k-1} e^{-\lambda z}}{\Gamma(k)}$$

λ and k are parameters of the distribution. $\frac{1}{\lambda}$ is the scale parameter, which defines the spread of the distribution and k is the shape parameter, which influences the peakedness

of the distribution. For further information about the features of the gamma distribution, see Casella and Berger (1990:100ff), where the shape parameter is called α and the scale parameter is called β .

Simulating the dataset, I used two different values for k ($k=5$ and $k=10$). Since I defined the mean of the distribution (\bar{z}) to be 1, it follows that $k = \lambda$ because $\bar{z} = \frac{k}{\lambda}$. This means that there are two different σ^2 , 0.1 and 0.2, because $\sigma^2 = \frac{k}{\lambda^2}$. The gamma distribution can look very different. If $k=1$, then it is identical to the exponential distribution, and if k is higher, then it becomes increasingly similar to the normal distribution.

B. Causes of death

Concordance table used for bridging revision 8 and 10 of the International Classification of Diseases (ICD)

Cause of death	ICD-6 and ICD-7	ICD-8	ICD-9	ICD-10
Infectious and parasitic diseases	001–138	000–136	001–139	A00–B99
Cancer of the oesophagus	150	150	150	C15
Cancer of the stomach	151	151	151	C16
Cancer of the colorectum	153–154	153–154	153–154	C18–C21
Cancer of the pancreas	157	157	157	C25
Cancer of the upper respiratory tract	140–148, 160, 161	140–149, 160, 161	140–149, 160, 161	C00–C14, C30–C32
Cancer of the lung	162–163	162	162	C33–C34
Cancer of the breast	170	174	174–175	C50
Cancer of the prostate	177	185	185	C61
Cancer of the bladder	181	188	188	C67
Cancer of the kidney	180	189	189	C64–C66, C68
Cancers, unspecified	198–199, 230–239	195–199, 230–239	195–199, 235–239	C76–C80, C97, D37–D48
Other cancers	Rest ^a (140–239, 294)	Rest (140–239)	Rest (140–239)	Rest (C00–D48)
Diabetes mellitus	260	250	250	E10–E14
Dementia and Alzheimer disease	304–306	290, 293	290, 331	F00, F01, F03, G30
Ischaemic heart disease	420	410–414	410–414	I20–I25
Other heart diseases	400–402, 410–416, 421–422, 430–434, 440–447	390–398, 400–404, 420–425, 427–429	390–398, 401–405, 416, 420–429	I00–I13, I15, I27, I30–I52
Cerebrovascular diseases	330–334	430–434, 436–438	430–434, 436–438	I60–I69
Other circulatory diseases	Rest (400–468)	Rest (390–458, excluding 435 & 446)	Rest (390–459, excluding 435 & 446)	Rest (I00–I99)
Pneumonia/influenza	480–483, 490–493	470–474, 480–483, 485–486	480–487	J10–J18
Chronic obstructive pulmonary disease	501, 502, 526, 527, 241	490–493, 518	490–494, 496	J40–J47
Senility	794	794	797	R54
Other symptoms and ill-defined conditions	Rest (780–795)	Rest (780–796)	Rest (780–799)	Rest (R00–R99)
Other diseases	Rest (001–795)	Rest (000–796)	Rest (001–799)	Rest (A00–R99)
Accidental fall	E900–904	E880–887	E880–888	W00–W19, X59
Other external causes	Rest (E800–999)	Rest (E800–999)	Rest (E800–999)	Rest (V01–Y98)

Source: Janssen et al. 2004:906

C. Stata code

The following Stata code simulates a data set with left-truncation, right censoring and decreasing mortality differences between rich and poor. This program will create a data file of about 250 MB and then runs several different models. It creates the more complicated of the two datasets used in chapter 9, namely with mortality ratios between rich and poor that decrease with age. Therefore, only Model 4 to Model 7 from Chapter 9 will run with the Stata code shown below. To run Model 1 to Model 3, Section 3 and Section 8 in the following program have to be skipped and in Section 10 the simpler `setset` command has to be used.

```
Diss_final_2.do
```

```
set more off
clear
set mem 500m
```

```
*1. create cases
```

```
set obs 1000000
gen id=_n
gen rich=1
replace rich=0 if id <=500000
gen birthyr=int((1920-1891)*uniform()+1891)
gen birthmo=int((12)*uniform()+1)
gen u=uniform()
```

```
*2. define 2 groups with different mortality:
```

```
gen b=0.11
gen a=0.006
replace a=0.012 if rich==0
```

```
*3. define decreasing mortality differences with increasing age:
```

```
gen a2=0.010909091
gen a3=0.01
gen a4=0.009230769
gen time1=1/b*log(1-b/a*log(1-u))
sum time1
histogram time1, bin(50) start(0)
```

```
*4. create the theoretical Gompertz density function to compare with:
```

```
gen checktime1=a*exp(b*time1)*exp(-a/b*(exp(b*time1)-1))
scatter checktime1 time1
gen k=5
gen L=k
rndgam 1000000 k 0.2
sum xg
histogram xg
save disstestrn.dta, replace
```

```
*5. the density function with hg (=heterogeneity):
```

```
gen timehg=1/b*log(1-b/(xg*a)*log(1-u))
histogram timehg, bin(50) start(0)
sum timehg
```

*6. the theoretical density function with gamma distributed frailty:

```
gen checktimehg2=a*exp(b*timehg)*k*(L^k)/((L+a/b*(exp(b*timehg)-1))^(L+1))
sum checktimehg2
scatter checktimehg2 timehg
```

*7. express all dates relative to January 1910:

```
gen lifeinmo=round((timehg+59)*12)
gen birthtime=(birthyr-1910)*12+birthmo
gen deathtime=birthtime+lifeinmo
gen deathyr=int(1910+(deathtime/12))
gen deathmo=deathtime-(deathyr-1910)*12+1
gen age=deathyr-birthyr
gen mort=1
gen begin=(1980-birthyr)*12+1-birthmo-708
gen end=(deathyr-birthyr)*12+1+deathmo-birthmo-708
gen check=lifeinmo-708
gen test=0
```

*8. new simulation of IA between age and income in 10 year steps, at age 70 and 80 and 90 with HG:

```
gen timehgnew=timehg
gen step_2=1 if rich==0 & timehgnew>11
sum timehgnew if step_2==1
gen timehg_2=1/b*log(1/(xg*a2)*(-b*log(1-u)-(xg*a)*(exp(b*11)-1)+(xg*a2)*exp(b*11)))
sum timehg_2
replace timehgnew=timehg_2 if step_2==1
gen step_3=1 if rich==0 & timehgnew>21
sum timehgnew if step_3==1
gen timehg_3=1/b*log(1/(xg*a3)*(-b*log(1-u)-(xg*a)*(exp(b*11)-1)-(xg*a2)*(exp(b*21)-exp(b*11))+(xg*a3)*exp(b*21)))
sum timehg_3
replace timehgnew=timehg_3 if step_3==1
gen step_4=1 if rich==0 & timehgnew>31
sum timehgnew if step_4==1
gen timehg_4=1/b*log(1/(xg*a4)*(-b*log(1-u)-(xg*a)*(exp(b*11)-1)-(xg*a2)*(exp(b*21)-exp(b*11))-(xg*a3)*(exp(b*31)-exp(b*21))+(xg*a4)*exp(b*31)))
sum timehg_4
replace timehgnew=timehg_4 if step_4==1
```

*9. make left truncation until 1980 and right censoring in 2000:

```
gen age1980=1980-birthyr-birthmo/12
gen beginnew=1980-birthyr-birthmo/12-59
gen age2000=2000-birthyr-birthmo/12
gen endnew=age2000-59
```

*10. stset-command for ideal observation from age 59 onwards:

```
*stset timehgnew, id(id) fail(mort)
```

*or, alternatively, stset-command for observation from 1980 to 2000 (left truncated and right censored data!):

```
stset timehg, id(id) fail(mort)origin(time test) entry(time beginnew)
exit (time endnew)
```

```
stsplot timeband, at(11 21 31 41)
```

```

gen tp1 =(_t<=11)
gen tp2 =(_t>11 & _t<=21)
gen tp3 =(_t>21 & _t<=31)
gen tp4 =(_t>31 & _t<=41)
gen tp5 =(_t>41)
gen tp1rich=tp1*rich
gen tp2rich=tp2*rich
gen tp3rich=tp3*rich
gen tp4rich=tp4*rich
gen tp5rich=tp5*rich

compress
save disstestrun.dta, replace

*11. run models:

*Model 1:
streg rich, d(gom)
*Model 2 and 3:
streg rich, d(gom) frailty(gamma)
*Model 4:
streg tp1 tp2 tp3 tp4 tp1rich tp2rich tp3rich tp4rich, d(gom)
*Model 5 and 6:
streg tp1 tp2 tp3 tp4 tp1rich tp2rich tp3rich tp4rich,
d(gom)frailty(gamma)shared(id)
*Model 7 with the constraint that theta=0.2:
constraint 1 [ln_the]_b[_cons]=-1.6094379
streg tp1 tp2 tp3 tp4 tp1rich tp2rich tp3rich tp4rich,
d(gom)frailty(gamma)shared(id)constraints(1)

*To calculate the survival and the hazard for the method in Chapter 9,
there are the following commands in Stata than can be applied after a
model estimation:

predict xb, xb
predict s, s
predict haz,haz

*These following commands apply the formulas and yield identical
results. Gamma is the parameter of the Gompertz function that is part
of the Model output.

gen mys=exp((-exp(xb)/e(gamma))*(exp(e(gamma)*_t)-exp(e(gamma)*_t0)))
gen myhaz=exp(xb)*exp(e(gamma)*_t)

```

D. Models

Model 1:

streg rich, d(gom)

```
          failure _d:  mort
analysis time  _t:  timehg
              id:   id
```

Fitting constant-only model:

```
Iteration 0:  log likelihood = -1176336.2
Iteration 1:  log likelihood = -975236.48
Iteration 2:  log likelihood = -810069.55
Iteration 3:  log likelihood = -809162.34
Iteration 4:  log likelihood = -809161.51
Iteration 5:  log likelihood = -809161.51
```

Fitting full model:

```
Iteration 0:  log likelihood = -809161.51
Iteration 1:  log likelihood = -769082.16
Iteration 2:  log likelihood = -767975.99
Iteration 3:  log likelihood = -767975.21
Iteration 4:  log likelihood = -767975.21
```

Gompertz regression -- log relative-hazard form

```
No. of subjects =      1000000          Number of obs   =      1000000
No. of failures =      1000000
Time at risk    = 21188969.01
Log likelihood  =  -767975.21          LR chi2(1)       = 82372.59
                                          Prob > chi2      =  0.0000
```

```
-----+-----+-----+-----+-----+-----+-----+
      _t | Haz. Ratio  Std. Err.      z    P>|z|    [95% Conf. Interval]
-----+-----+-----+-----+-----+-----+-----+
      rich |   .5517943   .0011372  -288.51  0.000    .54957   .5540276
-----+-----+-----+-----+-----+-----+
      gamma |   .0907873   .0000992   915.34  0.000    .0905929 .0909817
-----+-----+-----+-----+-----+-----+-----+

```

Model 2:

. streg rich, d(gom) frailty(gamma)

failure _d: mort
analysis time _t: timehg
id: id

Fitting Gompertz model:

Fitting constant-only model:

Iteration 0: log likelihood = -1504695.4
Iteration 1: log likelihood = -908710.76 (not concave)
Iteration 2: log likelihood = -843330.86
Iteration 3: log likelihood = -807348.74 (not concave)
Iteration 4: log likelihood = -805714.36
Iteration 5: log likelihood = -804156.09
Iteration 6: log likelihood = -804068.49
Iteration 7: log likelihood = -804065.97
Iteration 8: log likelihood = -804065.97

Fitting full model:

Iteration 0: log likelihood = -936048.55 (not concave)
Iteration 1: log likelihood = -783285.8
Iteration 2: log likelihood = -769048.04
Iteration 3: log likelihood = -764149.07
Iteration 4: log likelihood = -763476.57
Iteration 5: log likelihood = -763306.94
Iteration 6: log likelihood = -763306.6
Iteration 7: log likelihood = -763306.6

Gompertz regression -- log relative-hazard form
Gamma frailty

No. of subjects = 1000000 Number of obs = 1000000
No. of failures = 1000000
Time at risk = 21188969.01
Log likelihood = -763306.6 LR chi2(1) = 81518.73
Prob > chi2 = 0.0000

Table with 6 columns: _t, Haz. Ratio, Std. Err., z, P>|z|, [95% Conf. Interval]. Rows include rich, gamma, /ln_the, and theta.

Likelihood-ratio test of theta=0: chibar2(01) = 9337.22 Prob>=chibar2 = 0.000

Model 3:

streg rich, d(gom) frailty(gamma)

failure _d: mort
analysis time _t: (timehg-origin)
origin: time test
enter on or after: time beginnew
exit on or before: time endnew
id: id

Fitting Gompertz model:

Fitting constant-only model:

Iteration 0: log likelihood = -502482.84 (not concave)
Iteration 1: log likelihood = -437221.27
Iteration 2: log likelihood = -215789.34 (not concave)
Iteration 3: log likelihood = -210895.72
Iteration 4: log likelihood = -209013.68
Iteration 5: log likelihood = -208854.51
Iteration 6: log likelihood = -208830.4
Iteration 7: log likelihood = -208829.75
Iteration 8: log likelihood = -208829.75

Fitting full model:

Iteration 0: log likelihood = -275984.57 (not concave)
Iteration 1: log likelihood = -204923.61 (not concave)
Iteration 2: log likelihood = -195094.82
Iteration 3: log likelihood = -191554.47
Iteration 4: log likelihood = -189308.42
Iteration 5: log likelihood = -189100.31
Iteration 6: log likelihood = -188729.86
Iteration 7: log likelihood = -188726.89
Iteration 8: log likelihood = -188726.86
Iteration 9: log likelihood = -188726.86

Gompertz regression -- log relative-hazard form
Gamma frailty

No. of subjects = 663482 Number of obs = 663482
No. of failures = 520417
Time at risk = 7721472.687
Log likelihood = -188726.86 LR chi2(1) = 40205.77
Prob > chi2 = 0.0000

Table with 7 columns: _t, Haz. Ratio, Std. Err., z, P>|z|, [95% Conf. Interval]. Rows include rich, gamma, /ln_the, and theta.

Likelihood-ratio test of theta=0: chibar2(01) = 2403.42 Prob>=chibar2 = 0.000

Model 5:

```
streg tp1 tp2 tp3 tp4 tplrich tp2rich tp3rich tp4rich, d(gom) frailty(gamma)
> shared(id)
```

```
failure _d: mort
analysis time _t: timehgnew
id: id
```

note: tp4 dropped due to collinearity

Fitting Gompertz model:

Fitting constant-only model:

```
Iteration 0: log likelihood = -1506289.5 (not concave)
Iteration 1: log likelihood = -1090862.5 (not concave)
Iteration 2: log likelihood = -881876.94
Iteration 3: log likelihood = -810877.56
Iteration 4: log likelihood = -808414.64
Iteration 5: log likelihood = -806546.92
Iteration 6: log likelihood = -806529.92
Iteration 7: log likelihood = -806529.77
Iteration 8: log likelihood = -806529.77
```

Fitting full model:

```
Iteration 0: log likelihood = -937639.18 (not concave)
Iteration 1: log likelihood = -794152.83
Iteration 2: log likelihood = -780894.49 (not concave)
Iteration 3: log likelihood = -780835.68 (not concave)
Iteration 4: log likelihood = -780349.56 (not concave)
Iteration 5: log likelihood = -780170.02 (not concave)
Iteration 6: log likelihood = -780003.47 (not concave)
Iteration 7: log likelihood = -779728.18 (not concave)
Iteration 8: log likelihood = -779505.21 (not concave)
Iteration 9: log likelihood = -779289.22 (not concave)
Iteration 10: log likelihood = -779169.06 (not concave)
Iteration 11: log likelihood = -779091.67 (not concave)
Iteration 12: log likelihood = -779019.9 (not concave)
Iteration 13: log likelihood = -778921.33 (not concave)
Iteration 14: log likelihood = -778819.04 (not concave)
Iteration 15: log likelihood = -778719.22
Iteration 16: log likelihood = -778376.83
Iteration 17: log likelihood = -778086.82
Iteration 18: log likelihood = -778010.67
Iteration 19: log likelihood = -777979.68
Iteration 20: log likelihood = -777947.06
Iteration 21: log likelihood = -777919.29
Iteration 22: log likelihood = -777843.36
Iteration 23: log likelihood = -777838.31
Iteration 24: log likelihood = -777836.7
Iteration 25: log likelihood = -777834.14
Iteration 26: log likelihood = -777832.11
Iteration 27: log likelihood = -777831.99
Iteration 28: log likelihood = -777828.37
Iteration 29: log likelihood = -777828.15
Iteration 30: log likelihood = -777828.14
Iteration 31: log likelihood = -777827.71
Iteration 32: log likelihood = -777827.63
Iteration 33: log likelihood = -777827.28
Iteration 34: log likelihood = -777827.2
Iteration 35: log likelihood = -777826.96
Iteration 36: log likelihood = -777826.9
Iteration 37: log likelihood = -777826.75
Iteration 38: log likelihood = -777826.71
Iteration 39: log likelihood = -777826.6
Iteration 40: log likelihood = -777826.57
Iteration 41: log likelihood = -777826.5
Iteration 42: log likelihood = -777826.48
Iteration 43: log likelihood = -777826.43
```

```

Iteration 44: log likelihood = -777826.41
Iteration 45: log likelihood = -777826.37
Iteration 46: log likelihood = -777826.36
Iteration 47: log likelihood = -777826.33
Iteration 48: log likelihood = -777826.33
Iteration 49: log likelihood = -777826.3
Iteration 50: log likelihood = -777826.3
Iteration 51: log likelihood = -777826.28
Iteration 52: log likelihood = -777826.28
Iteration 53: log likelihood = -777826.27
Iteration 54: log likelihood = -777826.26
Iteration 55: log likelihood = -777826.25
Iteration 56: log likelihood = -777826.25
Iteration 57: log likelihood = -777826.24
Iteration 58: log likelihood = -777826.24
Iteration 59: log likelihood = -777826.23
Iteration 60: log likelihood = -777826.23
Iteration 61: log likelihood = -777826.23
Iteration 62: log likelihood = -777826.23
Iteration 63: log likelihood = -777826.22
Iteration 64: log likelihood = -777826.22
Iteration 65: log likelihood = -777826.22
Iteration 66: log likelihood = -777826.22
Iteration 67: log likelihood = -777826.22
Iteration 68: log likelihood = -777826.22
Iteration 69: log likelihood = -777826.21
Iteration 70: log likelihood = -777826.21
Iteration 71: log likelihood = -777826.21
Iteration 72: log likelihood = -777826.21
Iteration 73: log likelihood = -777826.21
Iteration 74: log likelihood = -777826.21
Iteration 75: log likelihood = -777826.2
Iteration 76: log likelihood = -777826.2
Iteration 77: log likelihood = -777826.2
Iteration 78: log likelihood = -777826.2
Iteration 79: log likelihood = -777826.2
Iteration 80: log likelihood = -777826.2

```

Gompertz regression --

log relative-hazard form
Gamma shared frailty

Number of obs = 2537623
Number of groups = 1000000

Group variable: id

No. of subjects = 1000000
No. of failures = 1000000
Time at risk = 21493473.66

Obs per group: min = 1
avg = 2.537623
max = 4

Log likelihood = -777826.2

LR chi2(7) = 57407.14
Prob > chi2 = 0.0000

_t	Haz. Ratio	Std. Err.	z	P> z	[95% Conf. Interval]
tp1	1.309889	.015156	23.33	0.000	1.280518 1.339933
tp2	1.178846	.0140696	13.79	0.000	1.15159 1.206747
tp3	1.08146	.0105193	8.05	0.000	1.061038 1.102276
tplrich	.4970846	.002595	-133.90	0.000	.4920244 .5021968
tp2rich	.5511296	.0023446	-140.05	0.000	.5465534 .555744
tp3rich	.5957138	.0032101	-96.13	0.000	.5894552 .6020389
tp4rich	.6445098	.0055556	-50.96	0.000	.6337125 .6554911
gamma	.110873	.0004527	244.93	0.000	.1099858 .1117602
/ln_the	-1.57803	.0309249	-51.03	0.000	-1.638641 -1.517418
theta	.2063813	.0063823			.1942438 .2192773

Likelihood-ratio test of theta=0: chibar2(01) = 3859.89 Prob>=chibar2 = 0.000

Model 6:

```
streg tp1 tp2 tp3 tp4 tprich tp2rich tp3rich tp4rich, d(gom) frailty(gamma)
> shared(id)
```

```
failure _d: mort
analysis time _t: (timehgnw-origin)
origin: time test
enter on or after: time beginnew
exit on or before: time endnew
id: id
```

note: tp4 dropped due to collinearity

Fitting Gompertz model:

Fitting constant-only model:

```
Iteration 0: log likelihood = -408925.19
Iteration 1: log likelihood = -352393.94
Iteration 2: log likelihood = -220174.63 (not concave)
Iteration 3: log likelihood = -216363.78
Iteration 4: log likelihood = -215895.32
Iteration 5: log likelihood = -215710.26
Iteration 6: log likelihood = -215704.46
Iteration 7: log likelihood = -215703.78
Iteration 8: log likelihood = -215703.78
```

Fitting full model:

```
Iteration 0: log likelihood = -264907.8
Iteration 1: log likelihood = -216931.52 (not concave)
Iteration 2: log likelihood = -208895.1
Iteration 3: log likelihood = -206175.33
Iteration 4: log likelihood = -203240.41
Iteration 5: log likelihood = -203089.11
Iteration 6: log likelihood = -202829.73
Iteration 7: log likelihood = -202816.15
Iteration 8: log likelihood = -202814.1
Iteration 9: log likelihood = -202814.03
Iteration 10: log likelihood = -202814.03
```

Gompertz regression --

```
log relative-hazard form      Number of obs      = 1436650
Gamma shared frailty         Number of groups    = 670680
```

Group variable: id

```
No. of subjects = 670680      Obs per group: min = 1
No. of failures = 519424      avg = 2.14208
Time at risk = 7907918.319    max = 3
```

```
LR chi2(7) = 25779.49
Log likelihood = -202814.03    Prob > chi2 = 0.0000
```

_t	Haz. Ratio	Std. Err.	z	P> z	[95% Conf. Interval]
tp1	1.295485	.0185322	18.10	0.000	1.259667 1.332321
tp2	1.278651	.0131571	23.89	0.000	1.253122 1.3047
tp3	1.19538	.0098218	21.72	0.000	1.176283 1.214786
tprich	.5070125	.0059702	-57.68	0.000	.4954452 .5188499
tp2rich	.5789955	.0029723	-106.45	0.000	.5731991 .5848506
tp3rich	.655545	.0029289	-94.52	0.000	.6498295 .6613107
tp4rich	.7563666	.0060481	-34.92	0.000	.7446049 .7683142
gamma	.0935801	.0005318	175.97	0.000	.0925378 .0946224
/ln_the	-2.677596	.0658194	-40.68	0.000	-2.8066 -2.548593
theta	.0687282	.0045236			.06041 .0781916

Likelihood-ratio test of theta=0: chibar2(01) = 244.46 Prob>=chibar2 = 0.000

Model 7:

```
streg tp1 tp2 tp3 tp4 tp1rich tp2rich tp3rich tp4rich, d(gom) frailty(gamma)
> shared(id) constraints(1)
```

```
failure _d: mort
analysis time _t: (timehgnew-origin)
origin: time test
enter on or after: time beginnew
exit on or before: time endnew
id: id
```

note: tp4 dropped due to collinearity

Fitting Gompertz model:

Fitting constant-only model:

```
Iteration 0: log likelihood = -408925.19
Iteration 1: log likelihood = -352393.94
Iteration 2: log likelihood = -220174.62 (not concave)
Iteration 3: log likelihood = -216363.78
Iteration 4: log likelihood = -215895.28
Iteration 5: log likelihood = -215710.26
Iteration 6: log likelihood = -215704.46
Iteration 7: log likelihood = -215703.78
Iteration 8: log likelihood = -215703.78
```

Fitting full model:

```
Iteration 0: log likelihood = -208467.81
Iteration 1: log likelihood = -203288.53
Iteration 2: log likelihood = -203202.95
Iteration 3: log likelihood = -203195.95
Iteration 4: log likelihood = -203195.25
Iteration 5: log likelihood = -203195.17
Iteration 6: log likelihood = -203195.16
Iteration 7: log likelihood = -203195.16
Iteration 8: log likelihood = -203195.16
Iteration 9: log likelihood = -203195.16
```

Gompertz regression --

```
log relative-hazard form      Number of obs      = 1436650
Gamma shared frailty         Number of groups    = 670680
Group variable: id
```

```
No. of subjects = 670680      Obs per group: min = 1
No. of failures = 519424      avg = 2.14208
Time at risk = 7907918.319    max = 3
```

```
Log likelihood = -203195.16    Wald chi2(7) = 25615.43
Prob > chi2 = 0.0000
```

(1) [ln_the]_cons = -1.609438

_t	Haz. Ratio	Std. Err.	z	P> z	[95% Conf. Interval]
tp1	1.326324	.0173566	21.58	0.000	1.292738 1.360782
tp2	1.233179	.0116271	22.23	0.000	1.210599 1.256179
tp3	1.122111	.0087091	14.84	0.000	1.105171 1.139311
tp1rich	.5049394	.0059871	-57.63	0.000	.4933403 .5168112
tp2rich	.5681632	.0029605	-108.50	0.000	.5623903 .5739954
tp3rich	.6294616	.0028014	-104.01	0.000	.6239948 .6349763
tp4rich	.7055393	.0058599	-42.00	0.000	.6941472 .7171184
gamma	.1040252	.0003739	278.20	0.000	.1032924 .1047581
/ln_the	-1.609438
theta	.2