

# Does inequality in self-assessed health predict inequality in survival by income?- Evidence from Swedish data

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## Abstract

This paper empirically addresses two questions using a large individual level Swedish data set linking mortality data to health survey data. The first question is whether there is an effect of an individual's self-assessed health on his subsequent survival probability and if this effect differ by socioeconomic factors? Our results indicate that the effect of self-assessed health on mortality risk declines with age – probably because of adjustment towards ‘milder’ overall health evaluations at higher ages – but does *not* seem to differ by indicators of socio-economic status like income or education. This finding suggests that there is no systematic adjustment of self-assessed health by socioeconomic status and therefore that the measured income-related inequality in self-assessed health is unlikely to be biased by reporting error. The second question is: how much of the income-related inequality in mortality can be explained by income-related inequality in self-assessed health? Using a decomposition method, we find that inequality in self-assessed health accounts for only about 10% of mortality inequality if interactions are not allowed for, but its contribution is increased to about 28% if account is taken of the reporting tendencies by age. In other words, omitting the interaction between age and self-assessed health leads to a substantial underestimation of the partial contribution of self-assessed health inequality by income. These results suggest that the often observed inequalities in self-assessed health by income do have predictive power for the – less often observed – inequalities in survival by income.

Keywords: health inequality; mortality risk; survival; self-assessed health; Sweden

## 1. Introduction

There is a large and fast growing literature comparing the existence of socioeconomic inequality in morbidity and mortality across a wide range of countries and populations.<sup>1</sup> The literature on socioeconomic inequality in measures combining health status and length of life like quality adjusted life years (QALYs) is much thinner.<sup>2</sup> One of the reasons for this is that mortality registrations often do not include indicators of socioeconomic status (SES) such as income, education or occupation. Another reason is probably that for reliable estimates of socioeconomic inequality in QALYs, large datasets with large spans of longitudinal follow up of both health status and mortality experience are required and the collection of such datasets is very costly and therefore relatively rarely undertaken (cf Wolfson and Rowe, 2001).<sup>3</sup> This explains why much of the literature on socioeconomic inequalities in morbidity and in mortality has developed relatively independently. Interestingly, the results obtained – at least in terms of cross-country comparisons – do not always match. For instance, Mackenbach *et al* (1997) report socioeconomic inequalities in France and Finland to be relatively high in mortality, but rather low in morbidity, and the reverse appears to be true for Denmark. However, the results based on mortality and morbidity need not necessarily coincide because they partly reflect different dimensions of health, but are strongly related in view of the consistently reported high predictive power of self-assessed morbidity for subsequent survival chances. Idler and Benyamini [(1997) for example, quote evidence from no less than 27 studies documenting that a respondent’s global health rating, based on the simple question “How is your health in general?”, with response categories ranging from “very good” or “excellent” to “poor” or “very poor” is an independent and powerful predictor of subsequent individual mortality. So, while self-assessed health (SAH) will include the evaluation of non-life threatening conditions on the one hand, and not reflect unknown future mortality risks (like e.g. accidents) on the other hand, the relationship nevertheless appears to be sufficiently strong to persist even after controlling for other mortality risk factors. Moreover, recent findings for Sweden (Burström and Fredlund, 2001) suggest

that the predictive power of SAH for mortality is unaffected by social class: SAH predicts survival equally well for high and low social classes.

This suggests that there is little reason to expect that the comparability of SAH across populations groups is problematic because of a problem which has been termed ‘state-dependent reporting bias’ (e.g. Kerkhofs and Lindeboom, 1995), ‘scale of reference bias’ (e.g. Groot, 2000) or response category cut-point shift (e.g. Sadana *et al*, 2000, Murray *et al*, 2001). Basically, it occurs if subgroups of a population use systematically different threshold levels for their SAH evaluation, despite having the same level of ‘true’ health. These differences may be influenced by, among other things, age, sex, education, language and personal experience of illness. Basically, it means that different groups appear to ‘speak different languages’ when they are responding to the same question. Evidence of significant cut-point shift by income or education would have important implications for the measurement and explanation of, for example, inequalities in SAH by income or education. If, given the same level of ‘true’ health, the assessment of reported health differs systematically by SES, this will bias the measured degree of socioeconomic inequality in health. If, for instance, those with lower SES are more inclined to report poor health at the same ‘true’ health, e.g. because of general feelings of dissatisfaction, then true socioeconomic health inequality will be overstated by using SAH. If the reverse is true, and lower SES persons are less inclined to report poor health given true health, e.g. because of lower health expectations, then true SES-related health inequality will be underestimated.

The problem with testing for cut-point shift is that ‘true’ health is not directly observable and therefore the benchmark is difficult to measure in a valid and reliable way. Typically, previous researchers have relied on other indicators of SAH (Kerkhofs and Lindeboom, 1995) or chronic conditions (Groot, 2000) to test for cut-point shift. While the test of an income-SAH interaction effect in survival prediction cannot be seen as a definitive test of such cut point shifting because morbidity and mortality are related but inherently different dimensions of health status, it seems nevertheless worthwhile to explore whether their covariance is affected by such an interaction effect

between SAH and SES indicators in a mortality prediction model. The longitudinal mortality follow up in the Swedish Level of Living Conditions Surveys allows us to test for such an interaction.

The above observations lead to the following two research questions which we address in this paper: (1) Given that mortality is obviously a very objective indicator of health risk, is there a significant interaction between SAH and other mortality predictors, in particular income and education? In the absence of such an interaction, the evidence on income-related inequalities in SAH carries more weight, since it indicates that income-related inequalities in mortality, even if unobserved directly, are also likely to follow similar patterns. (2) If SAH predicts mortality, to what extent can observed socioeconomic inequalities in current SAH (e.g. by income) predict inequalities in future survival?

The methods, data and variable definitions used are outlined below. This is followed by a results section. The paper ends with some concluding remarks.

## **2. Methods**

### **2.1 Modeling mortality risk**

Because the observation of the duration until death is censored by the length of the follow-up period (cf data description in section ), we analyse the effect of SAH and other covariates on mortality risk during follow up using a Cox 's (1972) proportional hazard model. This is a flexible semi-parametric model which makes no distributional assumptions about the functional form of the baseline hazard, as is required for most other hazard functions (cf Greene, 1993). Besides depending on time, the hazard rate will depend on individual characteristics (covariates). Taking these into account, the hazard rate at time  $t$  (the number of years from inclusion in the study) for individual  $i$   $H(t;X)$  may be written as:

$$(1) \quad H_i(t|X) = h(t|0) \cdot e^{X_i\beta}$$

where  $h(t,0)$  is the unknown baseline hazard rate at time  $t$  (i.e. the hazard rate for the respective individual when all covariate values are equal to zero),  $X$  is a vector of covariates which shifts the hazard function proportionally, and  $\beta$  is a vector of parameter.

All variables except age and income are entered as dummy variables. Age is included without any transformation, which implies an exponential relationship between age and the mortality risk. The annual mortality risk increases approximately exponentially with age in the Swedish adult population (Statistics Sweden 1998). We have tested for a significant interaction between SAH and the various other covariates using both t-tests and Wald  $\chi^2$  tests.

## 2.2 Measuring and decomposing income-related inequality in mortality

If SAH plays an important role in predicting survival it follows that inequality in SAH should also play some part in ‘explaining’ inequality in survival. We use the *health concentration index* [cf. Wagstaff, van Doorslaer and Paci (1989)] as our measure of *relative* income-related health inequality. If we have a continuous cardinal measure of health  $y_i$ , the concentration curve  $L(y)$  plots the cumulative proportion of the population (ranked by income, beginning with the lowest incomes) against the cumulative proportion of health. If  $L(y)$  coincides with the diagonal, everyone enjoys the same health. If, by contrast,  $L(y)$  lies *below* the diagonal, inequalities in health exist and favour the richer members of society. The further  $L(y)$  lies from the diagonal, the greater the degree of inequality. The health concentration index,  $C$ , is defined as twice the area between  $L(y)$  and the diagonal.  $C$  takes a value of zero when  $L(y)$  coincides with the diagonal and is negative (positive) when  $L(y)$  lies above (below) the diagonal. The minimum and maximum values of  $C$  using individual-level data are -1 and +1 respectively: these occur when all the population's ill-health is concentrated in the hands of the most and least disadvantaged persons respectively.

For individual-level data, it can be computed as (cf Kakwani, Wagstaff and van Doorslaer, 1994):

$$(2) \quad C = \frac{2}{\mu} \sum_{i=1}^N w_i y_i R_i - 1,$$

where  $\mu$  is the mean health of the sample,  $N$  is the sample size, and  $R_i$  is the relative fractional rank of the  $i$ th individual defined as [cf. Lerman and Yitzhaki, 1989]

$$(3) \quad R_i = \frac{1}{N} \sum_{j=1}^{i-1} w_j + \frac{1}{2} w_i \quad \text{where } w_0 = 0.$$

and thus indicates the cumulative proportion of the population up to the midpoint of each individual weight. More conveniently,  $C$  can be computed using the covariance between  $y_i$  and  $R_i$  [Lerman and Yitzhaki, 1989] as:

$$(4) \quad C = \frac{2}{\mu} \sum_{i=1}^N (y_i - \mu) \left(R_i - \frac{1}{2}\right) = \frac{2}{\mu} \text{cov}(y_i, R_i)$$

Recently, a method was proposed by Wagstaff, Van Doorslaer and Watanabe (2002) for decomposing the measured degree in inequality into its determining components. They show that for any linear regression model linking the variable of interest,  $y$ , to a set of  $K$  determinants,  $x_k$ :

$$(5) \quad y_i = \alpha + \sum_k \delta_k x_{ki} + \varepsilon_i,$$

where the  $\delta_k$  are coefficients and  $\varepsilon_i$  is an error term, the concentration index for  $y$ ,  $C$ , can be written as:

$$(6) \quad C = \sum_k (\delta_k \bar{x}_k / \mu) C_k + GC_\varepsilon / \mu = C_y + GC_\varepsilon / \mu,$$

where  $\mu$  is the mean of  $y$ ,  $\bar{x}_k$  is the mean of  $x_k$ ,  $C_k$  is the concentration index for  $x_k$  (defined analogously to  $C$ ),  $C_{\hat{y}}$  is the concentration index of predicted  $\hat{y}$  and  $GC_\varepsilon$  is the generalized concentration index for  $\varepsilon_i$ .

Equation (6) shows that  $C$  can be thought of as being made up of two components. The first is the deterministic component, equal to a weighted sum of the concentration indices of the  $k$  regressors, where the weight or “share” for, say,  $x_k$ , is simply the elasticity of  $y$  with respect to  $x_k$ . The second is a residual component, captured by the last term—this reflects the inequality in health that cannot be explained by systematic variation across income groups in the  $x_k$ . Thus (6) shows that, by coupling regression analysis with distributional data, we can partition the causes of inequality into inequalities in each of the  $x_k$ . The decomposition also shows how each determinant’s separate contribution to total income-related health inequality can be decomposed into two parts: (i) its health elasticity (i.e.  $\delta_k$ ), and (ii) its income-related inequality (i.e.  $C_k$ ). As such, the method therefore not only allows to separate the contributions of the various determinants, but also to identify the importance of each of these three components within each factor’s contribution.

We will exploit this useful property in the analysis of the contribution of inequality in self-reported health to inequality in survival of Swedish adults in section 4. Of course, in order to ‘linearize’ equation (1) it was estimated in logarithmic form as

$$(7) \quad \ln H_i(t; X) = \ln h(t|0) + X_i \beta$$

Consequently, we will be decomposing the inequality in the log of the one-year hazard rate, not in the survival probability itself. Moreover, since we will only be concerned with describing the inequality in *predicted* survival  $\hat{y}$  given the observed values of the  $X$  variable, attention will be limited to the first term in eq. (6), i.e. the deterministic component  $C_{\hat{y}}$ .

### 3. Data and variable definitions

The data used in this paper are taken from Statistics Sweden's *Survey of Living Conditions* (the ULF survey), which was linked to all-cause mortality data from the National Causes of Death Statistics and to income data taken from the National Income Tax Statistics. Since 1975, annually a random sample of adults aged 16-84 is interviewed about living conditions. In this paper we have used pooled data from the annual surveys conducted in 1980-1986 for adults aged 20-84.

After deletion of missing values, the sample size includes 43,328 individuals. The mortality experience of the sample was recorded until December 31, 1996. In Table 1 the variables in the regression analysis are defined and summary statistics are given.

The dependent variable in the regression analysis is the survival time in years and the survival status at the end of follow-up. The date of death was recorded for all subjects who had died by December 31, 1996.<sup>4</sup> The survival time is estimated as the number of years from the interview date to the date of death. The censored survival time of persons alive at the end of 1996 is estimated as the number of years from the interview date to December 31, 1996. The average censored survival time in the sample is 12.3 years, and 79.8% were still alive at the end of follow-up on December 31, 1996.

In the categorical SAH rating question the individuals rated their own current overall health status on a three-point scale (poor, fair or good health). This type of categorical health measure has been shown to capture important information about the individual's health and to be an important predictor of mortality (Idler and Benyamini, 1997; Wannamethe and Shaper, 1991). We expect the mortality risk to decrease with increasing SAH.

Our (permanent) income measure consists of the aggregate of two components: annual disposable income and the annuity of net wealth. The disposable income of the household (the income of the individual included in the study plus the income of the spouse/cohabitant) in the interview year is obtained from the National Income Tax Statistics.<sup>5</sup> From the same source, we also

have data about the taxable net wealth (total taxable assets minus total liabilities) of the household during the interview year.<sup>6</sup> The taxable net wealth is converted to net wealth at market value through an approximation (see Gerdtham and Johannesson, 2001), and the annuity of net wealth is based on the life-expectancy for men and women of different ages in Sweden and a 3% interest rate (Statistics Sweden, 1998). Income was converted to 1996 prices using the consumer price index and for married or cohabiting individuals the income of the household is divided by two to obtain an income per adult person in the household. Thus we assume that the household income is evenly divided between the spouses. The mortality risk is expected to decrease with higher income.

In the Survey of Living Conditions, respondents were asked two questions about functional ability. First they were asked if they could run a short distance (e.g. 100 metres) if they were in a hurry, and in the second question the respondents were asked if they could climb stairs without difficulty (this question was only asked to individuals who were unable to run a short distance). These two questions divide the respondents into three classes of functional ability: (1) those capable of running a short distance and climbing stairs without difficulty (i.e. no limitations in functional ability); (2) those unable to run a short distance but are able to climb stairs without difficulty (i.e. some limitations in functional ability); and (3) those unable to run a short distance and unable to climb stairs without difficulty (i.e. severely limited in functional ability). We expect the mortality risk to increase with decreasing functional ability.

We also include a dummy variable for persons diagnosed with hypertension. Elevated blood pressure is one of the most important risk factors of cardiovascular disease, and we expect hypertension to increase the mortality risk (MacMahon *et al.*, 1990). In addition we include the following demographic variables: age (at the time of inclusion in the study), gender and education. We expect the mortality risk to increase with age and to be higher for men than women (Statistics Sweden 1998) and to decrease with education. Finally, we have included six dummy variables for the year of inclusion into the study, to control for any differences between the populations included in different years.

## 4. Results

### 4.1 Effect of SAH on mortality risk

The Cox regression results of three model specifications with and without interaction terms with the SAH dummies are presented in Table 1. We can see that, as expected, being male and of higher age, and having functional limitations or high blood pressure increases mortality risk, whereas SAH, income and education all show protective effects on survival of Swedish adults. When SAH interactions are included, some of these coefficients become non-significant, in particular for blood pressure, education and income. The Wald  $\chi^2$  tests show that the interactions between SAH and age, gender and blood pressure *are* statistically significant but the interactions with income, education and functional status are not. Apparently, reporting good SAH is less protective for males and for those with known high blood pressure and the protective effect decreases with age. This suggests that SAH reporting is affected by these characteristics: males and hypertensives seem inclined to report better health at the same mortality risk as females and non hypertensives. Similarly, with increasing age, individuals are more likely to report good health at the same mortality risk. This seems fairly clear and intuitively plausible evidence of interaction with SAH reporting by these three characteristics.

An important result is that we do *not* observe similar interactions with respect to income and education. The  $\chi^2$  tests show that the interactions terms between SAH and income or education are not significant at the 5% level. This suggests that reporting poor or fair SAH has the same effect on mortality for individuals at different SES. As a result, inequalities in SAH by income or education will be mirrored in inequalities in mortality risk (or survival). Obviously, the estimated degree of (relative) inequality in self-reported morbidity and mortality need not necessarily be the same, because of the abovementioned lack of a one-to-one relationship between both health dimensions (morbidity and mortality). Some SAH inequality, reflecting non-fatal diseases, will not translate into any mortality inequality at all and some mortality inequality will not be associated with SAH, but it does mean that empirical evidence on socioeconomic inequality in SAH has strong implications for

the likelihood of socioeconomic inequality in mortality. In the next section we attempt to quantify this contribution.

#### 4.2 Contribution of inequality in SAH to inequality in survival

Table 2 presents the results of a decomposition analysis of the restricted and preferred Cox models along the lines proposed in equation (6).<sup>7</sup> It combines the estimated regression coefficients from Table 1 with information on the means and concentration indices of the explanatory variables to compute the ‘contributions’ of each of the variables – or to sets of dummy variables – to the total estimated income-related inequality. Remember that the concentration index of the dependent variable (i.e.  $C_j$ ) records the degree of inequality in the logarithm of the predicted hazard rate and as such is not directly comparable to concentration indices of other measures of mortality. Because the log of the hazard rate has a *negative* mean (hazard values are between 0 and 1) the interpretation of the concentration index is slightly more complex than usually: its estimated *positive* value of 0.0345 is then to be interpreted as indicating a *negative* association with income rank; Swedish adults with higher income have lower mortality probabilities, not higher.

The extent to which each of the explanatory variables is unequally distributed by income is reflected by their  $C$  values. We see that, unsurprisingly, income, education and male gender are positively associated with income rank while the opposite is true for the health variables and age. But their contribution to the measured degree of inequality in mortality obviously also depends on their estimated effect on mortality, as embodied in the Cox regression coefficients. The total effect can be measured in “units” of the concentration index as shown in the column with ‘contribution to  $C$ ’. Especially from the contributions expressed as percentages of total inequality it is clear that by far the greatest impact is due to age. This results from the fact that age is the strongest mortality predictor *and* from the fact that older individuals (in Sweden) are less wealthy. Looking at the restricted model without interactions first, we see that almost 70% of inequality is accounted for by the unequal distribution of age across income, while SAH and functional limitations account for 10% and 13.8%

respectively. The contributions of income (5.2%) and education (4.3%) to income-related inequality in mortality are much smaller than similar figures reported in van Doorslaer and Koolman (2000) and Van Doorslaer and Jones (2001) for inequality in self-reported health. However, none of these (cross-sectional) studies could control for initial health. In this paper we can control for initial health status to some extent by including SAH, functional limitations and elevated blood pressure at the time of the interview. This essentially means that we are looking at the effects on *changes* in health over time (as expressed by the mortality experience) for individuals who were initially in the similar health states, but differ with respect to income. Finally, the negative contribution of being male is explained due to the fact that males have higher mortality risk but are somewhat more concentrated among the higher income ranks.

The shifts in percentage contributions when comparing the restricted model with the (preferred) model allowing for interactions with SAH are interesting: the (separate) percentage contribution of age is reduced to 57.9% while that of SAH increases to 27.76%. Interestingly, the percentage contribution of the interaction between age and SAH is negative and equal to -4.82%. This can be interpreted as follows: not allowing for the fact that SAH reporting becomes 'milder' with increasing age leads to an underestimation of the effect of SAH and an overestimation of the effect of age on mortality. When this is taken into account, the effect of SAH becomes stronger and the effect of age becomes weaker. A similar effect (but of the opposite sign) can be observed with respect to the interaction between SAH and gender: not allowing for the fact that males tend to be milder in their SAH judgement also leads to a (slight) overestimation of its negative contribution to income-related inequality in mortality. Finally, allowing for interaction with SAH even results in a change in sign of the (fairly small) contribution of elevated blood pressure. The percentage contributions of the other variables, including income and education, are rather unaffected by the inclusion of the SAH interactions.

## 5. Conclusion

In this paper we have sought to provide an empirical answer to two questions using linked morbidity and mortality data for Sweden. The first question was: what is the effect of an individual's SAH reporting on his subsequent survival probability and does this effect differ by other characteristics, in particular income or education? We find that SAH does have a substantial predictive power for survival and that its effect differs by age, sex and hypertensive status, but not by income, education or functional limitation status. The SAH effect declines with age – probably because of adjustment towards ‘milder’ overall health evaluations at higher ages – and it is lower for males than for females, and for hypertensives than for non hypertensives, probably for similar reasons. The fact that the SAH effect does *not* seem to differ by SES indicators like income or education suggests that there is no systematic adjustment of SAH reporting by SES and therefore that the measured income-related inequality in SAH is unlikely to be biased by such reporting tendencies. This confirms earlier findings for Sweden suggesting that the predictive power of SAH on mortality does not differ by occupational group (Burström and Fredlund, 2001).

The second question addressed was: to what extent can the measured degree of income-related inequality in estimated mortality be explained by income-related inequality in SAH? Using a recently proposed method of decomposing concentration indices of inequality into the contributions of its determining factors, we found that inequality accounts for only about 10% of mortality inequality if interactions are not allowed for, but its contribution is increased to about 28% if account is taken of the reporting tendencies by age. In other words, omitting the age-SAH reporting interaction leads to a substantial underestimation of the partial contribution of SAH inequality by income.

What does this evidence tell us? One, it indicates that – if the Swedish evidence can be extrapolated to other countries, cultures and contexts – the often reported high predictive power of SAH for survival is *not* SES-dependent but it *is* dependent on age and sex. This implies that if

measures of income-related inequality in self-reported health are appropriately standardised for age-sex differences, they do carry important implications for expected (age-sex standardised) inequalities in mortality by income. Secondly, while the measured degree of inequalities in SAH cannot just be extrapolated to the degree of inequality to be expected in mortality – because of non-fatal illness and non-illness related mortality – the results suggest that countries with higher degrees of inequality in SAH are, all else equal (especially demographic structure), also likely to have higher degrees of inequality in mortality (in the same population). *Ceteris paribus*, countries with higher inequalities in SAH can also be expected to have higher inequality in mortality, and vice versa. This finding adds weight to studies documenting inequalities in supposedly “soft” health measures such as SAH by stressing the expected link with “harder” measures such as mortality. It also implies that where studies, such as the one by Mackenbach *et al* (1997), report conflicting country rankings for inequalities in SAH and in mortality, the differences are either due to the fact that “not all else is equal” or to the fact that these studies often have to rely on very different samples or cohorts per country. If a sufficient number of cross-sectional studies such as the Swedish ULF surveys were able to follow up the mortality experience of their samples, a higher degree of similarity between morbidity and mortality based studies is to be expected.

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## Tables

**Table 1: Descriptive statistics of variables (N = 43,328)**

<b>Variable</b>	<b>Mean</b>	<b>St. Dev</b>
<b>Dependent variable:</b>		
Survival time <sup>a</sup>	12.312	3.578
Survival status (=1 if dead at the end of follow-up)	0.201	.401
<b>Independent variables:</b>		
Male	.492	.500
Age	48.34	18.14
No functional limitations <sup>b</sup>		
Some functional limitations	.111	.314
Severe functional limitations	.111	.314
Poor health <sup>b</sup>		
Fair health	.200	.400
Good health	.733	.442
Elevated blood pressure	.088	.284
Annual income <sup>c,f</sup>	118985.9	64684
Less than secondary education <sup>b</sup>		
Short secondary education ( $\leq 2$ years)	.293	.455
Secondary education ( $> 2$ years)	.091	.288
University education	.171	.376
Included in the study 1980 <sup>b</sup>		
Included in the study 1981	.163	.370
Included in the study 1982	.154	.361
Included in the study 1983	.140	.347
Included in the study 1984	.151	.358
Included in the study 1985	.138	.345
Included in the study 1986	.100	.300

<sup>a</sup>The number of life-years from inclusion in the study to the end of follow-up.

<sup>b</sup> Baseline category in the regression analysis.

<sup>c</sup> Persons born abroad whose parents are current or previous foreign citizens.

<sup>d</sup> Persons born in Sweden whose parents are current or previous foreign citizens.

<sup>e</sup> Per adult person in the household in 1996 Swedish Crowns.

<sup>f</sup> Annual income=annual disposable income + annuity of net wealth

**Table 2: Regression results from the Cox model (N = 43,328)**

Covariate	General model		Preferred model		Restricted model	
	Coeff <sup>b</sup>	Z	Coeff	z	Coeff	z
Male	0.446*	8.8	0.437*	8.74	0.596*	26.78
Age	0.077*	28.98	0.078*	31.47	0.090*	77.79
Some functional limitations	0.196	1.84	0.326*	10.26	0.350*	11.04
Severe functional limitations	0.537*	5.55	0.530*	15.5	0.544*	15.84
Good health	-1.959*	-3.04	-2.024*	-9.93	-0.676*	-19.87
Fair health	-1.548*	-2.16	-1.291*	-5.74	-0.399*	-12.74
Elevated blood pressure	-0.025	-0.37	-0.035	-0.53	0.140*	5.17
Annual income <sup>a</sup>	-0.052	-1.11	-0.051*	-2.82	-0.050*	-2.74
Short secondary education ( $\leq 2$ years)	-0.114	-1.41	-0.025	-0.76	-0.024	-0.74
Secondary education ( $> 2$ years)	-0.061	-0.48	-0.146*	-2.71	-0.145*	-2.7
University education	-0.078	-0.58	-0.110*	-2.26	-0.131*	-2.69
Male*Good health	0.179*	2.96	0.184*	3.08		
Male*Fair health	0.186*	2.96	0.202*	3.28		
Age*Good health	0.016*	5.33	0.017*	6.15		
Age*Fair health	0.012*	3.47	0.010*	3.42		
Some functional limitations*Good health	0.118	1.03				
Some functional limitations*Fair health	0.177	1.48				
Severe functional limitations*Good health	-0.025	-0.22				
Severe functional limitations*Fair health	-0.012	-0.11				
Elevated blood pressure*Good health	0.204*	2.6	0.216*	2.75		
Elevated pressure*Fair health	0.187*	2.41	0.194*	2.51		
Annual income*Good health	-0.002	-0.04				
Annual income*Fair health	0.008	0.14				
Short secondary education ( $\leq 2$ years)*Good health	0.031	0.34				
Short secondary education ( $\leq 2$ years)*Fair health	0.207*	2.12				
Secondary education ( $> 2$ years)*Good health	-0.189	-1.28				
Secondary education ( $> 2$ years)*Fair health	0.040	0.25				
University education*Good health	-0.108	-0.73				
University education*Fair health	0.099	0.6				
Included in the study 1981	0.084*	2.58	0.085*	2.61	0.087*	2.67
Included in the study 1982	0.096*	2.59	0.099*	2.66	0.102*	2.74
Included in the study 1983	0.083*	2.11	0.083*	2.12	0.088*	2.23
Included in the study 1984	0.044	1.11	0.044	1.11	0.048	1.2
Included in the study 1985	0.079*	1.91	0.081	1.95	0.085*	2.04
Included in the study 1986	0.029	0.58	0.029	0.59	0.039	0.79
-Log-Likelihood	80998.34		81001.85		81036.38	
Likelihood ratio $\chi^2$ test of all coefficients=0	20302*		20295*		20226*	
LR $\chi^2$ test of all SAH interactions=0	76.73*		19.58*		-	
LR $\chi^2$ test of SAH interaction with MALE=0	10.44*		57.36*		-	
LR $\chi^2$ test of SAH interaction with AGE=0	28.37*		12.13*		-	
LR $\chi^2$ test of SAH interaction with FUNC=0	6.80		39.47*		-	
LR $\chi^2$ test of SAH interaction with BLOOD=0	7.42*		8.21*		-	
LR $\chi^2$ test of SAH interaction with INCOME=0	0.06		-		-	
LR $\chi^2$ test of SAH interaction with EDUC=0	12.52		-		-	

<sup>a</sup> Natural logarithm of annual income.

<sup>b</sup> \* Significant at 5% level.

**Table 3: Decomposition analysis of concentration index of mortality by income**

Covariate	Mean	CI	Restricted model (without interactions)			Preferred model (with interactions)		
			Coef	Contrib	% contrib	Coef.	Contrib	% contrib
Male	0.492	0.021	0.596	-0.00101	-2.93%	0.437	-0.00074	-2.10%
Age	48.339	-0.033	0.090	0.02369	68.70%	0.078	0.02024	57.90%
Some functional limitations	0.111	-0.262	0.350	0.00168		0.326	0.00155	
Severe functional limitations	0.111	-0.308	0.544	0.00307	13.77%	0.530	0.00297	12.93%
Good health	0.733	0.068	-0.676	0.00560		-2.024	0.01662	
Fair health	0.200	-0.163	-0.399	-0.00215	9.99%	-1.291	-0.00691	27.76%
Elevated blood pressure	0.088	-0.176	0.140	0.00036	1.04%	-0.035	-0.00009	-0.25%
Annual income <sup>a</sup>	11.600	0.019	-0.050	0.00180	5.22%	-0.051	0.00181	5.17%
Short secondary education ( $\leq 2$ years)	0.293	0.047	-0.024	0.00005		-0.025	0.00006	
Secondary education ( $> 2$ years)	0.091	0.107	-0.145	0.00023		-0.146	0.00023	
University education	0.171	0.324	-0.131	0.00120	4.31%	-0.110	0.00100	3.68%
Male*Good health	0.371	0.075				0.184	-0.00084	
Male*Fair health	0.090	-0.120				0.202	0.00036	-1.38%
Age*Good health	32.446	0.064				0.017	-0.00577	
Age*Fair health	11.679	-0.204				0.010	0.00409	-4.82%
Elevated blood pressure*Good health	0.041	-0.061				0.216	0.00009	
Elevated blood pressure*Fair health	0.036	-0.267				0.194	0.00031	1.14%
Included in 1981	0.163	-0.029	0.087	0.00007		0.085	0.00007	
Included in 1982	0.154	-0.025	0.102	0.00007		0.099	0.00006	
Included in 1983	0.140	-0.004	0.088	0.00001		0.083	0.00001	
Included in 1984	0.151	0.007	0.048	-0.00001		0.044	-0.00001	
Included in 1985	0.138	0.051	0.085	-0.00010		0.081	-0.00009	
Included in 1986	0.100	0.104	0.039	-0.00007	-0.09%	0.029	-0.00005	-0.04%
Conc ind log hazard rate				0.03449	100.00%		0.03496	100.00%
Mean log hazard rate				-6.054			-6.106	

## Notes

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<sup>1</sup> Recent examples of cross-country comparisons of socioeconomic inequality in self-reported morbidity include e.g. Van Doorslaer *et al* (1997), Kunst and Mackenbach (1995) and Cavelaars *et al* (1998), and in mortality include Kunst and Mackenbach (1994), Mackenbach *et al* (1997) and Wagstaff (2000).

<sup>2</sup> One recent exception is Gerdtham and Johannesson (2000) who estimated the income-related inequality in Sweden with respect to both life-years and QALYs. They found that inequalities in both favoured the higher income. For men (women) in the youngest age-group (20-29 years) the number of QALYs was 43.7 (45.7) in the lowest income decile and 47.2 (49.0) in the highest income decile. They also found that, at all ages, the relative inequality in life-years or QALYs was higher inequality in health status (measured by time trade-off QALY weights).

<sup>3</sup> Exceptions include, for instance, the National Child Development Study in the UK, or the Panel Study on Income Dynamics in the US, but such long panel studies are often restricted to particular cohorts of certain age groups and therefore unsuitable for global, population-wide inequality estimates.

<sup>4</sup> If individuals are lost to follow-up this could lead to attrition bias. Our mortality data were taken from the National Causes of Death Statistics which record all deaths of individuals registered as living in Sweden. Thus the only possibility for an individual in our study to be lost to follow-up would be if s/he has permanently emigrated during our observation period (and such individuals could only be a cause of bias if they have died during our observation period). Due to the low rate of emigration from Sweden (about 0.3% annually during our observation period (Statistics Sweden 1998)) attrition bias is not a major concern in our study.

<sup>5</sup> Disposable income consists of income from capital, income from employment and business and all income transfers (e.g. pension payments, unemployment benefits, paid sick-leave, housing assistance, etc) net of taxes. Capital income consists of interest rates, dividends and capital gains.

<sup>6</sup> Taxable net wealth includes property owned by the individual (including own homes and apartments), financial assets (e.g. bank savings, stocks, bonds, debts), business inventories, jewellery, and exterior inventories (e.g. cars, boats, etc).

<sup>7</sup> The decomposition analysis results obtained when instead of a Cox model a Weibull accelerated failure time model was used (cf. Greene, 1993) were very similar.