Population Association of America
2009 Annual Meeting, Detroit

Session 169: Comparative Mortality Experience
Chair: Patrick Heuveline
Discussant: France Meslé

How are causes of death shaping the differences in population health inequality between the United States and Sweden?

Paper presented by:
Claudia Nau
Glenn Firebaugh
Pennsylvania State University
Department of Sociology and Population Research Institute

ABSTRACT

This paper investigates the cause-specific underpinnings of differences in population health inequality between the United States and Sweden. Population health inequality is conceptualized as population heterogeneity in the age at death and is measured as the spread of the mortality age distribution. We use a decomposition technique to derive measures that quantify the degree to which the inequality difference between these two countries is due to specific causes having a higher spread in their patterns, different average timings of death and higher levels of mortality. We show which causes are the main contributors to international differences in inequality and through which mechanisms they do so. Cross-country differences in these features of cause specific mortality are likely to be reflective of, and might inform us on, national idiosyncrasies in the distribution of risk factors. Since susceptibility to specific causes and risk exposure vary by sex, the decomposition is partitioned by sex.

INTRODUCTION

This paper investigates how cause-specific mortality underlies the cross-country difference in population health inequality between the United States and Sweden. Population health inequality is conceptualized as heterogeneity in the age at death and measured as the variation of the age-distribution of life table deaths. Ryan Edwards and Shripad Tuljapurkar (2005) used the standard deviation of the distribution of ages at death occurring after age 10 (s_{10}) to measure population health inequality, and found that in the United States inequality in population health was significantly higher than in Sweden. They showed that these trends could not be explained well with the common predictors of health inequality such as income, education and race. This study investigates the epidemiological underpinnings of the differences in population health inequality. Our aim is to show which causes of death are underlying national differences in population health and through which mechanisms these causes operate to increase the spread of the age at death distribution.

Prior research has shown that the secular decline in mortality was accompanied by a massive decrease in the heterogeneity of life-spans (Wilmoth and Horiuchi 1999; Kannisto 2000). The decline of within-population inequality lost momentum in the 1950s (Wilmoth and Horiuchi 1999) after levels of infant and child mortality had decreased sufficiently to leave future mortality reductions to be made at older ages. A comparison of the United States with other developed nations reveals the nations poor state in terms of population health. The US presents the highest variability in adult ages at death when compared to six other developed nations (Wilmoth and Horiuchi 1999; Edwards and Tuljapurkar 2005) and its life-expectancy ranks among the lowest of 21 industrialized countries (White 2002). Sweden, in contrast, had until recently the longest lived population, and remains the leader in population homogeneity in terms of the age at death (Edwards and Tuljapurkar 2005).

Shkolnikov et al. (2003) present an age-cause specific decomposition of the absolute difference of two countries' Gini coefficients to describe the cause-specific underpinnings of cross-country differences in the lengths of life spans. They demonstrate their method by decomposing the difference of the Gini coefficients of the US and the UK. Using six broad cause groups they show that the excess inequality of the US was largely due to over-mortality from external causes in young adult ages and from cardiovascular diseases in the middle age range.

Edwards and Tuljapurkar (2005) advocate the use of the spread of the distribution of the age at death because it revealed previously undetected trends in population health inequality. We wish to use this straight-forward measure of heterogeneity to contribute to a better understanding of the epidemiological underpinnings of the US health inequality. We will present a method which will allow us to firstly identify the causes of death which contribute most to the inequality differential of the United States and Sweden and secondly, to quantify the degree to which the inequality gap is due to specific causes of death showing more within-cause inequality, differences in the average timing and higher levels of mortality. Causes of death have distinct age patterns which depend on their etiology and the presence of risk factors in the population. Consequently, national differences in cause-specific age-patterns, i.e. the within-cause variability of the age at death and the average timing, as well as differentials in the proportion of deaths from a particular cause are likely to reflect national idiosyncrasies in the distribution of risks. These features of cause-specific mortality are therefore the starting point of this analysis. In addition to identifying cause-specific mechanisms contributing to the inequality gap between the United States and Sweden, our method will also allow us to assess whether the concept of general susceptibility could serve as an explanation for the observed population heterogeneity. The concept of general susceptibility suggests that the cumulative effect of life-stresses and unfavorable environments increases a person's susceptibility to disease and death in general

(Thurlow 1967; Alter and Riley 1989; Kunitz 2002). Under this assumption, cause-specific mortality is supposed to be reflective of the population's composition in terms of frailty (Himes 1994). If the observed difference in the spread of the life spans would reflect that the US population is generally more frail and susceptible to death, we would expect a wide variety of causes to have age distributions that are more spread out. The latter would reflect that causes of death were striking on higher proportions of vulnerable individuals at all ages. Susceptibility to specific causes of death and risk exposure, particularly through risk behavior, both vary by sex (Krieger 2002). Accordingly, we consider features of cause-specific mortality separately for men and women.

Our method allows us to isolate the contributions of differences in within-cause inequality, average timing and cause-specific levels of mortality to the inequality gap of two countries. Practically, we decompose the absolute difference in the variation of the age distribution of adult life table deaths of a country pair into three parts: the cause-specific marginal effects of within and between variance, and the effect of the allocation of deaths across causes. These marginal effects measure what proportion of the difference in the variation of a country dyad would have persisted if, *ceteris paribus*, for example, only the within variance of a specific cause had been different.

DATA

This study uses mortality and population data from the World Health Organization Mortality Database (WHO2007b) for Sweden and the United States for 2001. The MDB provides the underlying cause of death which is "the disease or injury which initiated the train of morbid events leading directly to death, or the circumstances of the accident or violence which produced the fatal injury" (WHO 2007a). Mortality data has been reported by the "relevant national

authority" (WHO 2007a) to the World Health Organization. For the United States and Sweden all deaths registered through their vital registration systems in 2001 have been reported. The relevant population denominators have been provided by the respective national authority along with their mortality data to the WHO (WHO 2007).

Causes of death are coded in the 10th Revision of the International Classification of Disease[1] and are aggregated into 16 categories which are adapted from the "List of 39 Selected Causes of Death" of the NCHS Instruction Manual Part 9, (NCHS 2007). The cause-groups of Suicide, Homicide and Drug Induced Deaths have been coded according to definitions from the Technical Notes of the National Vital Statistics Report for the year 2001 (Anderson and Smith 2003). A detailed description of the cause-grouping is given in Appendix A.

The MDB provides the age at death aggregated into five year age categories with the open ended age interval being 95+ years. For calculating the mean age and the variance of the age distribution we assume that individuals died, on average, at the mid-point of the age interval. The WHO population database provides the population denominators for calculating the overall and cause-specific mortality rates. Population data for the 90-95 year interval and the open ended age category were missing for the United States. We are using estimates for the population size of these age groups provided by the Human Mortality Database (HMD 2009)[2].

Overall and Cause specific mortality rates were used to calculate single and multi-decrement life tables for each country. Using life table deaths solves the problem of differences in population size and age composition across national populations which would otherwise influence our results. The life table reflects the mortality experience of an imaginary birth cohort

[1] "The ICD is the international standard diagnostic classification [...] It is used to classify diseases and other health problems recorded on many types of health and vital records including death certificates and health records." WHO (2007). "International Classification of Diseases." Retrieved 01/17/09, from www.who.int/classifications/icd/end/index.html.

[2] We could not use the HMD as source for mortality data since it does not provide information on cause-specific mortality.

which has undergone at each age of its lifetime the age specific mortality rates of the selected year. The multi decrement life table allows calculating how deaths have been distributed across the 16 cause-categories assuming that the imaginary cohort had been subject to the age-cause specific rates of 2001. Ryan Edwards and Shripad Tuljapurkar used for their calculations data from the Human Mortality database which provides information on the age at death in 1 year intervals up to age 110. As described above the data from the World Health Organization is limited to 5 year age intervals with the open ended age interval being 95+ years. In order to establish comparability and test the limitations which the WHO data format sets to our calculations we first reproduced Edward's and Tuljapurkar's results on the 1 year age interval data of the HMD and recalculated then s_{10} using 5 year age intervals up to age 110 with life table data provided from the HMD and compared these results to the s_{10} calculated on the WHO data. The results are shown in Table 1, the last column shows the conditional standard deviations calculated on the data from the World Health Organization Mortality Database. Overall differences of the country specific s_{10} across datasets are small. For the United States, for instance, using 5-year age intervals up to age 110 increases s_{10} by a maximum of .06 years. While using the WHO data with 5 year age groups and the open ended age interval of 95+ years decreases s10 in comparison to Edwards' and Tuljapurkar's results with a maximum difference of .19 years.

Like Edwards and Tuljapurkar (2005), our analysis excludes deaths that occurred at ages younger than 10 years because of the high influence of differences in infant and childhood mortality on the spread of the mortality age distribution. All measures are calculated on multi-decrement life table deaths in order to eliminate effects of differences in population size and age-structure on the variation of the age distribution. Variations, mean ages and variances reported hereafter are calculated on life table deaths occurring after age 10 if not noted otherwise.

METHOD

The method presented here is an adaptation of a method suggested by Douglas L. Anderton for considering the role of mean age and incidence changes of specific causes of death in the extension of the life-span (Nau and Beemer 2004). For this analysis we decompose the absolute difference in the variation of the mortality age distribution of two countries into the marginal effects of the spread of each cause (marginal effect of the difference in the within variance), its average timing (marginal effect of the difference in the between variance) and its allocation of deaths (effect of the difference in the level of mortality). For notational convenience, the equations below demonstrate the marginal effects for both sexes jointly. The partitioning by sex, practically, doubles the number of cause groups. The overall population mean age at death is that of both sexes jointly.

In order to identify and measure which parts of the US mortality experience are unfavorable we need a "best case scenario" to which we can compare the US mortality regime. Out of seven countries investigated by Edwards and Tuljapurkar (2005), Sweden proved to have an age distribution with the lowest spread and serves therefore as the reference age distribution against which the US will be compared. By investigating differences in the causes of death for Americans and Swedes we can pinpoint more precisely the proximate sources of the greater variability in age at death among Americans. Observe that differences between Americans and Swedes with respect to cause of death can contribute to the greater American variability in age at death in three principal ways.

(1) First, and most obviously, Americans might tend to be more susceptible to die at higher rates of a cause of death that strikes at all ages (i.e. that is highly variable with respect to age), or one that is centered far off the overall mean. Even if the variance and average timing of all causes

would be the same in the United States and Sweden– but the allocation of deaths across causes would favor those that fall further off the average mean or are among those with higher spread, heterogeneity would be increased through a different distribution of deaths across causes. Death due to accidents comes to mind. Suppose the mean and variance for age at death due to accidents were exactly the same for the US and Swedish populations. In both countries traffic accidents disproportionately affect the young. The cause-specific mean age is therefore in both countries much lower than the overall population mean age at death. Then, a higher rate of accidental deaths in the United States would contribute to the US's overall greater variability in the age at death. We call this the *allocation effect*.

(2) Alternatively, suppose Swedes and Americans would die at the same rate of a particular disease – no allocation differences, therefore no allocation effects – but the variability in age at death tends to be greater for each cause in the United States. We call this the *spread effect*: Differences between the United States and Sweden in the cause-specific variability in age at death would suggest that in the US the population is vulnerable to die from this specific cause across a wider age range. If we would find an increased spread in most or all causes, this would support the general susceptibility hypothesis.

(3) Suppose, thirdly, that cause-specific death rates are the same for Americans and Swedes (no allocation effects) and that the cause-specific age distributions have the same shape (no spread effects) but the age distribution are centered around different means. In that case variability in age at death will be greater in the country where the means are more spread out. We call this the *timing effect*. A positive high timing effect for the US indicates that on average this cause strikes at ages that are more different from the overall population mean than in Sweden.

For each cause or cause-group, then, we can calculate the allocation effect, the spread effect, and the timing effect. These effects indicate how much of the inequality difference of the United States and Sweden would have persisted if the two countries had only differed in respect to, for example, the spread of a specific cause. We also need an *interaction effect* to account for the effect of the simultaneous differences in allocation, spread, and timing. All three effects and the interaction effect sum to the gross contribution of a particular cause. The gross contribution is the proportion of the inequality gap of the country dyad that would have persisted if the mortality regime of the two countries had differed only in terms of this cause group.

The sum of squares (SS) or variation of the age-distribution of death in a nation *n* is

$$\theta_n = \sum_c \sum_i \left(x_{n,c,i} - \bar{X}_n \right)^2 \quad (1)$$

Where *x* is age at death, *n* indexes the nation (SW and US will refer to Sweden and the United States), the subscript *i* indexes individuals, *c* is the set of all causes and $\bar{X}_n$ is the population mean age at death of nation *n*. The variation is our "measure of choice" to be decomposed because it is a function of (a) cause-specific within and between variance in age at death and (b) proportion of cases within each cause.

In order to identify the cause-specific features which are underlying the US population's excess in health inequality we compare its cause-specific mortality regime to that of Sweden by decomposing the absolute difference of the two countries' variations:

$$\Delta\theta = \theta_{US} - \theta_{Sw} \quad (2)$$

The sum of squares contributed by a specific cause $a \in c$ will be denoted with $\theta_{n,a} = \sum_{i \in a} \left(x_{n,a,i} - \bar{X}_n \right)^2$. Using these notational conventions we can write the gross contribution of a specific cause to the absolute difference in the variation of the United States and Sweden as:

$$Gross\ Contribution = \frac{\theta_{US,a} - \theta_{SW,a}}{\theta_{US} - \theta_{SW}} = \frac{\Delta\theta_{\ a}}{\Delta\theta} \quad (3)$$

The numerator is the difference in the variation of Sweden and the United States which is due to cause *a*. It is scaled by the absolute difference of the variation in the distribution of ages at death of Sweden and the United States to yield the proportion of the inequality gap that would persist if our two countries had only differed in the mortality experience of cause *a*. The gross contribution can be decomposed into the marginal effects of differences in the within variance, between variance, the weighting of between and within variance and an interaction effect accounting for the simultaneous difference of the variances and the weighting. These marginal effects will be presented next.

The spread effect is defined as the marginal effect of the differences in the within variance of a particular cause *a* across two countries. It is the amount of the inequality gap that is due to the difference in the within-variance of a cause. Computationally, the spread effect of a specific cause *a* is defined as:

$$\text{Spread Effect} = \frac{\left(\sigma^2(within)_{US,a} - \sigma^2(within)_{SW,a}\right) \times n_{sw,a}}{\Delta\theta} \quad (4)$$

The spread effect is the proportion of the inequality gap that would have persisted if US deaths occurring from cause *a* had occurred with its actual within cause inequality but with the levels of mortality of Sweden.

The timing effect is the proportion of the inequality gap that would have persisted if the two countries had only differed in terms of their distance of the cause-specific mean of cause *a* to their respective overall mean age at death:

$$\text{Timing Effect } = \frac{\left(\sigma^2(between)_{US,a} - \sigma^2(between)_{SW,a}\right) \times n_{sw,a}}{\Delta\theta} \quad (5)$$

Next is the a*llocation effect,* it is the proportion of the difference in inequality that would have been observed if both countries had only differed in terms of the allocation of deaths to cause a.

$$\text{Allocation Effect} = \frac{\left(n_{US,a} - n_{Sw,a}\right) \times \left[\sigma^2(between)_{Sw,a} + \sigma^2(within)_{Sw,a}\right]}{\Delta\theta} \quad (6)$$

Finally, the interaction effect accounts for the simultaneous differences in the variances and their weighting:

$$\text{Interaction Effect} = \frac{\Delta n_a \times \left(\Delta\sigma^2(between)_a + \Delta\sigma^2(within)_a\right)}{\Delta\theta} \quad (7)$$

Spread, timing, allocation and interaction effects sum to the gross contribution of a specific cause. All gross contributions sum to 100% of the absolute difference in the variation of the United States and Sweden.

Before we begin the presentation of our results it is important to note that the decomposition is informative only to the extent that the cause-groups are aggregations of causes which are pertinent in terms of the etiology of the disease. Our cause-grouping consists of 15 cause-categories and one residual group. The residual category aggregates all causes which we could not consider separately. Its cross-country differences in the mean age, spread and allocation are therefore hard to interpret. It will therefore, not be considered hereafter. Its gross contribution accounts for not more than 11.76%. We therefore conclude that we captured most of the cause-specific dynamics underlying the inequality gap in one of the 15 specific cause-groups. The decomposition offers a wealth of information; we will focus on those cause-groups which showed the highest gross contributions.

RESULTS

Figure 1 shows the age distributions of life table deaths occurring after age 10 for the United States and Sweden. It is obvious that the US mortality age distribution reflects less favorable mortality conditions than the Swedish: Population heterogeneity of the age at death is higher (conditional standard deviation of 12.81 years in the US compared to a Swedish s_{10} of 15.06 years) and the mean age of life table deaths occurring after age 10 is lower in the United States than in Sweden (77.81 years in contrast to 80.23 years).

Table 2 shows the results of our decomposition. The cause-specific spread effects for men and women are listed in column 1 and 2, the timing effects in column 3 and 4, allocation effects in column 5 and 6 and interaction effects in column 7 and 8. The sex-specific gross contribution of each cause can be found in column 9 and 10 and the gross contributions for both sexes jointly are presented in (column 11). The cause-specific gross contribution for both sexes is the row total of the spread, timing, allocation and the interaction effects of both sexes for that particular cause. It is the proportion of the inequality gap that would have persisted if Sweden and the United States would have differed only in their mortality experience from this particular cause. The gross contributions of all causes and both sexes sum to 100% of the inequality gap.

In the United States men and women contribute to almost equal parts to the inequality gap with Sweden. According to the female gross contribution, 49% of the inequality difference would have persisted if only US women had differed in their mortality experience from Swedish women.

The column totals give a first overview of the forces shaping the inequality difference of the United States and Sweden. The biggest gross contributors to the inequality gap are spread effects. That is, 63.39% of the observed inequality would have persisted if causes had only differed in terms of their within-cause variance. This gross contribution is split almost equally

between men and women (34 % contributed by males and 39.39% contributed by females). A closer look at the cause-specific spread effects will show, however, that within-cause inequality is similar for men and women in only three cause-groups (Infectious Diseases, Cancers and Heart Disease). Two of these disease groupings are main contributors to the inequality differential and will be discussed below. We may conclude that for these three cause groups the risks underlying the higher within cause-inequality in the United States are not highly sex-specific.

The second most important force shaping the inequality difference of our country dyad are male allocation effects. The latter sum to a gross contribution of 12.27%. The sum of female allocation effects constitutes less than half of this amount. Male and female high allocation effects can be found in the same cause groups; males, however, display higher effects, throughout. Timing effects are generally low, with the exception of female Heart Disease. This cause group is the only notable contributor to the inequality gap through a different average timing of deaths. Interaction effects which account for the simultaneous difference in the spread, allocation and average timing of deaths are generally low, suggesting that for most causes only one mechanism is at work. The only notable exceptions are female Heart Disease, male Homicide, male Infectious Diseases and the Residual cause-category. The interaction effects of the former three cause groups will be addressed below. The column totals give us a first impression of the broad dynamics which are underlying the inequality gap of our country dyad. Spread effects, like all other effects of notable size, are concentrated within a small number of causes. We conclude therefore, that cause-specific risks and not general susceptibility are a more likely explanation for increased within cause inequality in the United States. Under the general susceptibility hypothesis we would have expected to track increased spread effects in a host of causes which should take advantage of, and strike on, a generally more vulnerable population.

In terms of cause-specific mortality, Heart Disease, Traffic Accidents and Homicides contribute most to the inequality gap (with gross contributions of 27.09%, 15.86% and 13.5%, respectively). These cause groups are followed by Infectious Diseases and the Residual category (with gross contributions of 10.18% and 11.76%). Heart Disease stands out as the major contributor to the inequality gap. According to its gross contributions, 27.09% of the observed difference in the variation of the two countries' age at death distributions would have persisted if the country dyad had only differed in terms of its mortality from Heart Disease. The spread effects of men and women in the United States are remarkably similar, which suggests that the risk factors increasing the vulnerability to heart disease along the age range are not highly sex specific risk factors. Figure 2 compares the male and female age distributions of the age at death from Heart Disease of both countries. The US and Swedish distributions are strikingly different from each other and the distribution of life-spans is more similar within gender than within country (which is the case for most cause-groups). The female conditional mean ages at death amount to 85.32 and 84.35 years for Sweden and the United States, respectively. For males they are 79.44 and 77.03 years. Despite this within gender similarity, we see that US male and female age distributions are more spread out than the Swedish. It is obvious that in the US younger age groups are more affected than in Sweden; for women mortality in the oldest age group of 95+ also is higher than in Sweden. In the United States more women but less men succumb to Heart Disease. The gross effect on the inequality gap is moderate, however (female allocation effect of 2.19%). For men the allocation effect is -2.38%. This brings to mind the role of competing risks. The US has a higher variation in its population age-distribution than Sweden; if men die less from Heart Disease; this is most likely due to the fact that they already succumbed to competing risks at younger ages. Negative allocation effects need to be interpreted with care, their net effect on inequality will largely depend on what individuals die of instead. The average timing for male

deaths from Heart Disease in the US is very similar to that of Swedish deaths when compared to their respective population (timing effect of -.003). Women in the United States, however, contribute to the inequality difference by falling further off their population mean age than Swedish women (timing effect of 4.31%). US women's mean age at death of Heart Disease is similar to that of Swedish women (84.35 years and 85.33 years, respectively). In both countries, the mean age of female Heart Disease is above that of their respective population. Since the US population mean age is notably lower than the Swedish, inequality in the US is increased because women show similarly favorable average timing in deaths from Heart Disease than Swedish women.

The second and third most important contributors to the inequality gap between Sweden and the United States are two external cause groups of death: Traffic Accidents and Homicides. In sum, 29.3% of the observed difference in inequality would have persisted if the US had differed only in the mortality from these two causes. Traffic Accidents have a gross contribution of 15.86%, with men giving rise to two thirds and women to one third of this amount. Within cause inequality and average timing of Traffic Accidents in the US are similar to those observed in Sweden. The gross contribution to the inequality gap is largely driven by a difference in the allocation of deaths to this cause (male allocation effect of 12.25% and female allocation effect of 5.43%).

Homicide is the second external cause which contributes strongly to the difference in mortality inequality of our country dyad. Similarly to Traffic Accidents, low spread and timing effects indicate that the within cause inequality of the age distribution and the average timing contribute little to the inequality gap. The allocation effects, however, are 8% for males and 2.3% for women. In the US, men and women are more likely to be younger than in Sweden when they become victims of homicides (conditional mean age at death for American males and females

35.50 and 42.99 years and 47.4 to 49.2 years for Swedish). The reason why this difference does not show in the timing effect is that the allocation of deaths to this cause is low in Sweden. Thus, the between variance is weighted little in the calculation of the timing effect (0.57%). The interaction effect (2.81%) has to account for the simultaneous difference in timing and allocation between the United States and Sweden.

Infectious Diseases present a gross contribution of 10.18%. The male gross contribution is twice as high as that of females (6.66% compared to 3.51%). It is driven by a simultaneous difference in the allocation of deaths (male allocation effect of 1.52%), average timing (timing effect of 1.05%) and within cause inequality (spread effect of 1.44%). Consequently, the interaction effect is high, accounting for 2.66%. The female gross contribution stems from a positive spread effect (1.56%) and higher allocation of deaths to this cause (1.53%). Having several high marginal effects for men and women suggests that Infectious Diseases present a very different picture in the United States than in Sweden. Figure 3 shows the age distribution of deaths from Infectious Diseases for Swedish and American men and women. In Sweden Infectious Diseases tend to affect the old ages. The shape of the US age distributions are strikingly different. Both, the male and the female distribution of life-spans show much higher mortality in young adult and mid-ages. Male mortality reaches a second peak in the age group of 45-50. Apparent differences in the coding and reporting of 3 digit diagnoses paired with our limited medical knowledge limit our ability to compare the leading infectious diseases in each country.[3] The most prominent diseases in this cause group for males and females in the United States are various forms of Septicemia, Acute hepatitis and HIV related deaths. For Sweden we

[3] Sweden has only 55 different diagnosis reported for infectious diseases while the United States reports 216 diagnoses which leads us to believe that the US and Sweden have different coding or reporting practices for Infectious Diseases

can only say that the cause with the highest incidence were Septicemia, "Other and unspecified infectious diseases" and "Enterocolitis due to clostridium difficile".

DISCUSSION

Our goal was to describe the cause-specific underpinnings of the inequality difference between the United States and Sweden. We found that the causes contributing most to the inequality difference of our country dyad were Heart Disease, Traffic Accidents, Homicide, and Infectious Diseases. These results support the findings by Shkolnikov, Andreev et al. (2003) who compared the mortality inequality between the United States and the United Kingdom. Our research adds to the understanding of the epidemiological underpinnings of high levels of US health inequality by quantifying the magnitude of the cause-specific contributions, by describing the mechanism through which cause categories operate to increase inequality, and by showing how these mechanisms operate by gender.

All Cause-groups under investigation here, except Suicide, either contribute little or positively to the inequality gap between the United States and Sweden. This draws a pessimistic picture of the overall public health situation in the United States. The mechanisms which led causes to contribute to the mortality inequality gap were, however, disparate. We could not find evidence for the general susceptibility hypothesis. Two of the causes which were significant contributors to the inequality gap, Heart Disease and Infectious Diseases, showed spread effects that were remarkably similar for men and women. We suggested that this finding was hinting to non-sex specific risk factors underlying within cause inequality of these cause groups. Cancers also showed similar spread effects for men and women, their increased within-cause inequality impacted the mortality gap less, however. Their positive spread effect was buffered by negative allocation and timing effects.

Both, men and women showed high allocation effects in the same cause-groups. Male effects, however, were higher throughout than female's. The causes contributing most to the inequality gap by presenting high and positive allocation effects were Traffic Accidents, Homicide and Infectious Diseases. National levels of Traffic Accidents have been shown to depend on a multitude of factors such as speed limits, driving density, alcohol consumption, expenditures on high-way safety, seat belt laws and average distance traveled (Rockett and Smith 1989; Zlatoper 1991). Several of these factors (e.g. expenditure on high-way safety and traffic laws) are structural and should raise the risk of traffic accidents for both genders. Others, such as alcohol consumption and distance traveled are more likely to differ for men and women. Michaud and Murray (2001) show that Traffic Accidents are the second most important cause-group contributing to premature disability and death of US males. Pampel (2001) discusses how differential mortality from traffic accidents is likely to reflect gender inequality in terms of risk and routine behaviors (e.g. work related driving). Thus, higher levels of mortality from traffic accidents in US men and women suggests higher structural risks while the gender gap is likely to reflect common differences in risk behavior and exposure.

The second external cause contributing through high allocation effects to the inequality gap is Homicide. Men presented notably higher allocation effects than women. Homicide rates in the US are among the highest in industrialized nations, while the Swedish are among the lowest (Gartner 1990; Pampel and Williamson 2001). Gartner (1990) models the variation of homicide across 18 developed countries including the US. She finds that material deprivation, more cultural heterogeneity, more family dissolution and higher exposure to official violence explained homicide excess in the United States and elsewhere. These factors help to explain why homicide incidence is higher for both men and women in the United States than in Sweden. Smith and Brewer (1992) investigate homicide in a sample of US central cities and argue that social

disorganization increases the risk of homicide victimization for men and women by creating a general climate of violence. They suggest that the same factors have higher explanatory power for men since men are more likely than women to engage in "illegal economic activities" in the context of social disorganization and are thus exposed to greater risks of becoming the victim of violence.

The cause-group of Infectious Diseases contributed positively to the inequality gap through all three effect types (with the exception of a small negative timing effect for women). After graphing the age distribution we found a striking over-mortality of 25-55 year old men in the US. We have some indications that this over-mortality might be at least partially due to deaths from sexually transmitted diseases. We can say with some confidence however, that with Homicide, Traffic Accidents and certain Infectious Diseases, non-degenerative causes of deaths in general, and external causes in particular, play an important role in shaping the inequality gap of our country dyad. We can also say that these causes of death contribute to the inequality gap in the first place through higher levels of mortality and only in the case of infectious diseases through higher within cause variance.

Heart Disease was a major force contributing to the inequality gap. Diseases of the Heart are the leading cause of death in the United States and in most developed countries (Mokdad, Marks et al. 2004). Shkolnikov, Andreev et al. (2003) find that diseases of the heart contribute notably to the inequality differential between the United States and the UK by increasing mortality in the age range of 40-59. Our findings suggest that Heart Disease increases inequality in the age at death mainly through higher within-cause inequality in both genders. The magnitude of spread effects of men and women are similar, which suggest that the risk factors contributing to increased susceptibility along the age range in the US are not gender-specific. We found a

positive timing effect for women which could be explained by American women having a similarly favorable mean age at death from Heart Diseases as Swedish women.

The decomposition method presented here offers a convenient tool for considering the cause-specific underpinnings of health inequality because it summarizes age-cause and sex specific information into an acceptable number of indicator measures that have a straightforward interpretation. The gross contributions allowed us to measure the role of a specific cause in shaping the inequality differential of the two countries, while the marginal effects serve as an indicator measure for the presence and magnitude of three cause-specific mechanisms underlying the inequality differential.

There are some caveats that need to be considered for the interpretation of the marginal effects. The first is related to the fact that the main-effects are weighted measures. Thus, if in the United States a cause displays a high within-variance and high levels of mortality but in Sweden this cause has both, low within (and possibly between variance) and low levels of mortality, the marginal effects are small. Then, a significant part of the cause-specific contribution to the inequality differential will be captured in the interaction effect which accounts for the simultaneous difference of the variances and the number of life table deaths. As shown in our results section, the marginal effects are best used to gain an overview of the dynamics underlying the inequality gap of a country dyad and should then be followed up with selective comparisons of the actual age-distributions, mean ages and variances.

This is also important because of another short coming of our measure: its symmetric nature. The latter poses a conceptual problem when studying health inequality. We cannot detect whether a cause contributes to inequality by causing more deaths below or beyond the overall mean age at death. Technically, both scenarios contribute to population heterogeneity i.e. inequality. Practically, however, when we are studying health inequality because we want to gain

understanding that can serve to reduce inequality, we should be able to differentiate between these two scenarios. Each has very different public health implications. In our analysis Heart Disease, for instance, contributed positively to the inequality gap because US women were stretching the overall age-pattern of mortality into higher ages.

When we interpret differences in the cause-specific age distributions as reflections of risk exposure, we implicitly assume that the US and the Swedish population have the same potential for a homogeneous age distribution of deaths. The United States population in the year 2000 was, however, composed by 10.4% of foreign born individuals (Singh and Siahpush 2002). Immigrants differ in terms of their health status, health history, and health habits. This should influence population heterogeneity. The direction of the effect on population heterogeneity is not clear, however. First generation immigrants have been found to have a mortality advantage compared to native born whites of similar socio-economic status (Franzini, Ribble et al. 2001; Frisbie, Cho et al. 2001; Singh and Siahpush 2002), while there is evidence that this advantage decrease with length of stay (Frisbie, Cho et al. 2001; Singh and Siahpush 2002; Salant and Lauderdale 2003). In order to understand the contribution of immigration to population heterogeneity we needed to analyze foreign and native born individuals in both countries separately.

This research is only a snapshot of national mortality experiences. Time trends in cause-specific contributions to inequality could be especially revealing because a high variance of the overall age pattern might be a transitory stage. Medical and Public Health advances tend to be introduced first to the top of the social hierarchy (Link and Phelan 1995). Therefore, an increase in population heterogeneity might be a transitory state until progress has spread down the social ladder. A longitudinal analysis could help to test the role of such diffusion processes in shaping health inequality.

Furthermore, it would be important to conduct a cohort analysis. We used a synthetic cohort to infer lifetime risk patterns. Mortality is influenced by health experiences across the life course and these vary according to the period an individual is born in. Thus, ignoring that lifetime health experience varies across periods might bias our results (Finch and Crimmins 2004). A cohort perspective might yield new results, especially in regard to the general susceptibility hypothesis.

Heart Disease was the only leading cause of death (in terms of mortality levels) which was also a main contributor to the inequality differential. Its contribution was mainly driven through differences in it's within cause inequality. Traffic Accidents and Homicides contributed as external causes of death through higher levels of mortality to the difference in population heterogeneity. Finally, there were Infectious Diseases whose role demands further investigation, particularly into the male over-mortality at young and mid-ages. Other leading causes of deaths (in terms of mortality levels), such as Cancers and Cerebrovascular Diseases did not contribute notably to the inequality gap. In this paper we focused on cause-groups which presented the highest gross contributions to the inequality differential of the US and Sweden. The effects table has more to offer, however. There is a second group of causes with moderate gross contributions such as Alzheimer's, Diabetes and Chronic Lower Respiratory Diseases which await exploration. The next step will be to apply our method to other countries in order to study main contributors and mechanisms in a cross-national comparison.

Tables and Figures

Table 1: Comparison of conditional standard deviation calculated on varying data formats, for data for the US and Sweden, 2001 from the Human Mortality Database (HMD) and the World Health Organization Mortality Database (WHO)

data source	S_{10} HMD	S_{10} HMD	S_{10} WHO
age interval	1 year	5 year	5 year
open ended interval	110+	110+	95+
Sweden	12.861	12.929	12.813
United States	15.163	15.202	15.062

Figure 1: Comparison of Swedish and US age distributions of life table deaths occurring after age 10, 2001 (WHO Mortality Database)

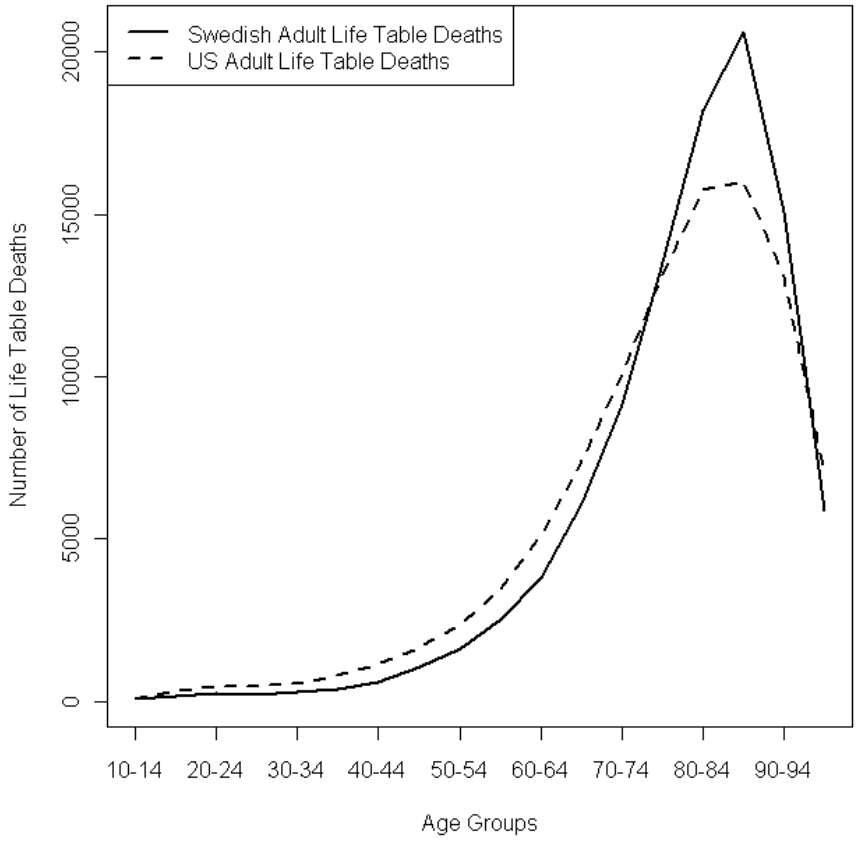


Table 2: Results of the decomposition of the inequality gap by cause and gender between the United States and Sweden, effects are presented as % of the inequality gap of the country dyad

Cause Categories	**Spread Effect**		**Timing Effect**		**Allocation Effect**		**Interaction Effect**		**Gross Contribution**		**Gross Contribution**
	Males	Females	Males	Females	Males	Females	Males	Females	Males	Females	Both sexes
Infectious	1.439	1.558	1.046	-0.083	1.523	1.068	2.655	0.97	6.663	3.513	10.176
Cancers	3.249	3.399	-0.816	-2.165	-0.902	-1.272	-0.072	-0.048	1.46	-0.086	1.374
Diabetes	0.199	0.999	0.164	-0.159	0.93	0.969	0.137	0.429	1.43	2.238	3.668
Alzheimer's	-0.019	-0.12	0.266	1.087	0.392	1.501	0.277	1.535	0.915	4.004	4.919
Heart	11.349	11.288	-0.003	4.308	-2.383	2.19	-0.891	1.227	8.073	19.013	27.086
Cerebrovascular	2.831	4.21	0.154	2.205	-2.674	-3.017	-1.129	-1.818	-0.818	1.58	0.762
Influenza/Pneum	0.77	1.177	0.099	0.45	-0.432	0.097	-0.126	0.046	0.311	1.77	2.081
Chron Low Resp	0.595	0.38	-0.005	0.031	1.453	2.274	0.445	0.44	2.49	3.125	5.615
Nephritis	0.308	0.344	-0.019	0.002	0.702	0.922	0.304	0.754	1.295	2.022	3.317
Maternal Mort	0	0.002	0	0.007	0	0.264	0	0.025	0	0.296	0.296
Traffic	-0.123	0.029	-0.678	-0.044	12.25	5.426	-0.972	-0.033	10.478	5.378	15.856
Suicide	0.622	-0.059	0.963	1.285	-0.974	-2.393	-0.12	-0.617	0.492	-1.784	-1.292
Homicide	-0.146	-0.084	0.565	0.102	7.969	2.227	2.805	0.063	11.193	2.308	13.501
Drug Induced	-0.014	-0.112	-1.331	0.312	1.154	0.917	-0.205	0.117	-0.396	1.234	0.838
Other Accidents	3.02	1.495	1.495	-0.048	-2.849	-1.322	-1.295	-0.447	0.372	-0.323	0.049
Residual	9.9	14.88	0.851	-1.472	-3.892	-4.502	-1.765	-2.244	5.094	6.662	11.756
Sum	33.98	39.386	2.751	5.818	12.267	5.349	0.048	0.399	49.052	50.95	100.002

Figure 2: Comparison of Swedish and US Life Table Deaths from Heart Disease by sex, 2001, (WHO Mortality Database)

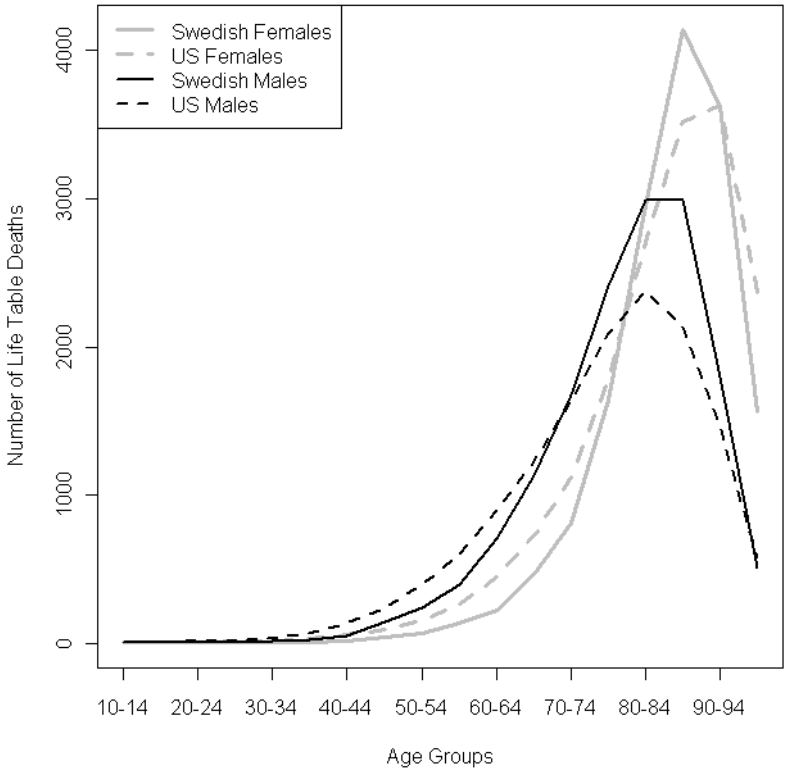
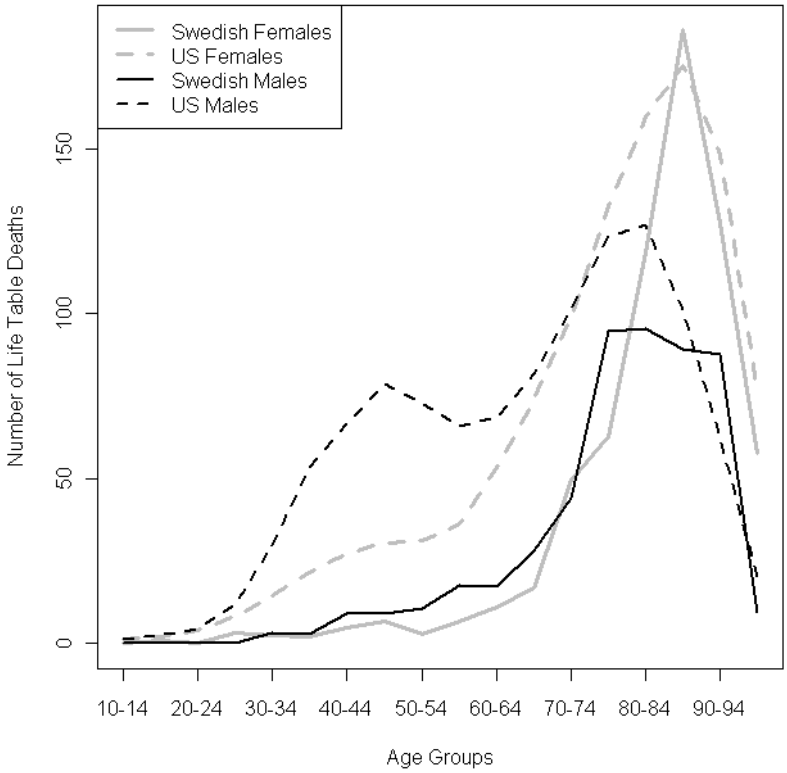


Figure 3: Comparison of Swedish and US Life Table Deaths from Infectious Diseases by sex, 2001 (WHO Mortality Database)



Appendix A - Cause-grouping

In this Appendix we describe the cause-grouping established for this analysis. The Mortality Database (WHO 2007b) provides us with 2-3 digit codes of the International Classification of Disease 10th Revision (ICD10). The ICD10 is a detailed cause-classification "for all general epidemiological, many health management purposes and clinical use"(WHO 2007c). In order to conduct an epidemiologic-demographic study it is necessary to aggregate pathologies to cause-groups. These categories serve the practical purpose to lower the number of causes under consideration while constituting entities which, from an epidemiological point of view, are sensible. We assume that the "similarity of disease processes connotes a similarity in physical and environmental conditions affecting the incidence of the disease"(Preston, Keyfitz et al. 1972) . Therefore, the principal criterion for aggregating pathologies is their etiology, the sum of processes leading to the disease. Whenever this is not possible, cause-groupings default to organize causes by their symptomatology.

In addition to reducing the number of cause-groups, broad cause-categories also fulfill the purpose to buffer errors and national differences in recording and coding habits of causes of death. Death is a clearly defined event, the data collection of the underlying cause, nevertheless, does not only dependent on the biological processes leading to death. Diagnosis, recording and coding of causes of death are also scientific, cultural and administrative processes which might vary across countries. Therefore, cause-groups need to be established in a way in which they can accommodate inaccuracies in diagnostics, recording and coding schemes (D'Amico, Agozzino et al. 1999) .

Cause coding and recording is an inexact science and establishing a pertinent and robust cause-grouping is a difficult task even for specialists. Therefore, we serve ourselves the work of the specialists of the National Center for Health Statistics who established ICD10 cause-listings of different lengths. We used the 39-cause grouping, the shortest NCHS list, as point of departure to create a list of causes that was apt in length and contents to our analysis. We first aggregated the 39 categories established by the NCHS into 9 cause-groups. These nine categories were the 9 leading causes of death as enumerated in the National Vital Statistics Reports for the year 2001 (Anderson and Smith 2003) and maternal causes of death. Next we specified three cause-groups which had attracted our attention in our preliminary analysis by their cross-national differences in mortality. These were Suicide, Homicide and Drug Induced Deaths. ICD-codes for these categories were aggregated according to the

Technical Appendix of the National Vital Statistics Report for the year 2001 (Anderson and Smith 2003).

The resulting cause-grouping is shown in Table A along with the corresponding ICD codes for each category and a description of its contents. When ICD headings and sub-headings are used to describe the contents of a cause-group this is indicated through quotes.

Table A: Correspondence of the 15 specific cause-categories used in the analysis with ICD10 codes as available in the Mortality Database (MDB) and description of their contents

Cause-group	**ICD 10 code range** (as available in the MDB)	**Contents of Cause-group,** (ICD-titles are quoted)
Infectious Diseases	A00-B99	Chapter 1: "Certain infectious and parasitic diseases"
All Cancers	C00-D48.9	Chapter 2: "Neoplasms"
Diabetes	E10-E14.9	All codes for "Diabetes Mellitus"
Alzheimer's	G30-G30.9	All codes for "Alzheimer's Disease"
Heart Diseases	I00-I09.9 I11-I11.9, I13-I13.9 I20-I25.9 I00-I09.9 I26-I51.9	 "Hypertensive heart disease with or without renal disease" "Ischemic Heart Disease" "Acute Rheumatic Fever Chronic rheumatic heart disease" "Pulmonary heart disease and diseases of pulmonary circulation" "Other forms of heart disease"
Cerebrovascular Diseases	I60-I69.8	"Cerebrovascular Diseases"
Influenza and Pneumonia	J10-J189	"Influenza and Pneumonia"
Chronic Lower Respiratory Diseases	J40-J47	
Nephritis, Nephrosis, Nephrotic Symptom	N00-N07.9 N17-N19 N25-N27.9	
Maternal Mortality	O00-O99.8	
Traffic Accidents	V02-V04.9, V09.0, V09.2 V12-V14.9, V190-V19.2, V19.4-V19.6	All deaths that occurred in relation with a motor-vehicle
All Other Accidents	Remaining codes V01-Y99	Remainder of sub-chapter "Accidents"
Suicide (excludes self-poisoning)	X60-X84.9 Y87.0	"Intentional self-harm" "Sequelae of intentional self-harm"
Homicide	X85-Y09.9 Y871	"Assault" "Sequelae of assault"

Cause-group cont.	ICD 10 code range cont.	Contents of Cause-group cont.
Drug Induced Deaths	F11.0-F11.5, F11.7-F11.9, F12.0-F12.5, F12.7-F12.9, F13.0-F13.5, F13.7-F13.9, F14.0-F14.5, F14.7-F14.9,F15.0-F15.5, F15.7-F15.9,F16.0-F16.5, F16.7-F16.9,F17.0, F17.3-F17.5,F17.7-F17.9,F18.0-F18.5, F18.7-F18.9, F19.0-F19.5, F19.7-F19.9 X40-X44.9	Deaths due to psychoactive substances related to "Mental and behavioural disorders" "Intentional self-poisoning by and exposure to noxious substances" "Event[Poisoning] of undetermined intent"

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