Revisiting the Mortality Gradient: SES and Cause-Specific Mortality in Southern Sweden in the Past 200 Years

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Abstract

Higher socioeconomic status (SES) is associated with lower mortality. This has been confirmed using different indicators across several geographical settings. Nevertheless, the timing of the emergence of the SES gradient remains an open question. This paper shows the development of SES inequalities in cause-specific mortality in southern Sweden over 200 years. We apply a cause-specific hazard model to estimate mortality differentials by SES. Our results confirm that the SES differences we see today emerged only around 1970 and demonstrates that, with few exceptions, SES differences emerged about the same time for all causes of death. For women it started earlier than for men, particularly in infectious diseases. In the nineteenth and early twentieth century we find a positive association between SES and mortality from circulatory diseases for men. We argue that habits and behaviors embedded in the different social strata played a major role behind the SES differences in mortality observed throughout the analyzed period.

Keywords

Socioeconomic status

Cause-specific adult mortality

Sweden

Nineteenth century

Twentieth century

Long-term trend

1. Introduction

Socioeconomic status (SES) is positively related to health and negatively related to mortality. Nowadays, in most developed societies there is a perfect mortality gradient by SES (Cutler et al., 2008; Elo, 2009; Marmot 2004), including egalitarian societies with a developed welfare state, such as Sweden (Erikson and Torssander 2008; Fritzell and Lundberg 2007). Reducing socioeconomic inequalities is a high priority in public health (Marmot et al., 2014), since it may improve life expectancy at the national level to a larger extent than eliminating cardiovascular diseases or cancer (Veugelers and Guernsey 1999; Veugelers, Yip, and Kephart 2001). The overwhelming and consistent evidence together with the substantial impact on public health make the study of this relationship of interest to policy makers (Marmot et al., 2014; WHO, 2008).

The mechanisms behind the SES differentials in mortality are still debated. While several studies have looked at all-cause mortality (Mackenbach et al. 1997; Torssander and Erikson 2010), focusing on specific causes of death can provide valuable insights in understanding these mechanisms (Erikson and Torssander 2008; Toch-Marquardt et al. 2014).

A much argued matter in the literature is when the SES gradient in mortality emerged (Bengtsson and Dribe, 2011; Bengtsson and Van Poppel, 2011; Dribe and Eriksson, 2018; Edvinsson and Lindkvist, 2011). While some studies have found SES differences in adult mortality far back in time, others point towards a recent emergence. In Sweden, for example, it was not until the 1970s that a full negative SES gradient emerged in adult mortality for both sexes (Bengtsson et al., 2018).

The main objective of this paper is to advance the understanding of mortality inequalities by studying SES differences in cause-specific adult mortality for men and women over a period of 200 years, focusing on when the SES gradient emerged, and in which causes of death. To our

knowledge, there is no published study analyzing the association between SES and cause-specific adult mortality over such a long period. This type of analysis has never been possible before and allows us to shed light not only on the relationship between SES and cause-specific adult mortality, but also on when and how it became to be what we observe in recent studies. Our study further contributes to the present literature by presenting sex differences in the association of interest. Recent findings have shown the importance of considering sex differences in presenting socioeconomic inequalities in all-cause and cause-specific mortality. This is mainly due to differences in the impact of SES for men and women, and to a different prevalence of social, behavioral, and biological risk factors by sex (Rogers et al. 2010; Backholer et al. 2017).

Our analysis is based on individual-level, longitudinal data from southern Sweden. We use data on occupation-based SES, family context and cause of death from population registers and vital event records, and event-history analysis with competing outcomes (causes of death). With a series of sensitivity analyses we provide evidence about the robustness of our results with respect to different aspects related to exposure and outcome.

2. Background

There are several mechanisms through which SES could affect adult mortality related to working conditions, social position, education, and income. Working conditions include job characteristics and material benefits connected to paid work. Occupational risks clearly differ between unskilled workers and higher managers, exposing the two groups to different health outcomes (Brand et al. 2007). Social position reflects characteristics such as prestige, social status and power related to occupation and it relates to control over the workplace and social networks that, when stronger and wider, allow for potentially more help when needed (Fujishiro, Xu, and Gong 2010; Marmot 2005). Higher earnings and/or education may also explain the

relationship between SES and mortality, as individuals with higher SES have access to better healthcare, resources (Cutler, Lleras-Muney, and Vogl 2008), and knowledge about prevention (Lleras-Muney 2005). SES may also have an effect on mortality through different lifestyles (Cutler, Lleras-Muney, and Vogl 2008; Marmot et al. 1991; 1978).

SES inequalities in mortality vary greatly by cause of death in contemporary societies (Elo, Martikainen, and Myrskylä 2014; Kunst et al. 1998). Cardiovascular mortality, nutritional/metabolic disorders, and respiratory diseases are strongly linked to SES (Erikson and Torssander 2008; Kunst et al. 1998) and mostly related to risk factors such as smoking, hypertension, and cholesterol (Lynch et al. 2006). Death from lung, stomach, and esophageal cancers (Smith et al. 1991), as well as unintentional poisoning and falls, and assaults are more common among disadvantaged groups, who may be exposed to negative habits and conditions (Erikson and Torssander 2008).

2.1 SES inequalities in mortality over time

While the contemporary SES gradient in mortality is consistent across contexts, results from the past are mixed. As a consequence, it is unclear whether SES differences have always been present or if they are a recent phenomenon, and, if so, when they emerged (Bengtsson and Van Poppel, 2011).

According to the Fundamental Cause Theory (FCT), inequalities in mortality by SES are more or less universal (Link and Phelan 1995). Even if the causal mechanisms vary historically and geographically, SES remains a key determinant of health and mortality (Freese and Lutfey, 2011; Lutfey and Freese, 2005; Cockerham 2013). A recent version of the FCT, which has taken demographic and epidemiological transitions (Omran 1971) into account, postulates that, as mortality declines, new diseases determine overall mortality, but overall mortality differentials go

through the same four phases (Clouston et al. 2016). First, diseases are largely non-preventable because of lack of knowledge on causal agents and treatment. Therefore SES differences in mortality are usually small. Second, SES differences arise, thanks to emerging knowledge on disease prevention and treatment, which spread asymmetrically favoring higher SES. Third, mortality among the lower SES groups starts declining and inequalities reduce as a result of more evenly distributed health innovations in the population. Last, mortality-reducing innovations become universally available and no further gains can be obtained. In some cases the disease is virtually eliminated, in others small disadvantages for low-status groups remain, due to differences in behaviors or resources (Clouston et al. 2016). Importantly, this pattern occurs for all diseases. At all stages, except before the start of the transition, high SES groups are advantaged when looking at overall mortality. Therefore, SES is a fundamental cause of overall mortality, although pathways may vary by disease.

Several studies support this theory, by showing that higher SES is protective for several preventable diseases associated with higher education or more resources (Mackenbach et al. 2015; Phelan, Link, and Tehranifar 2010). However, most of the studies are based on contemporary data (Phelan et al., 2010). Findings from the late nineteenth and early twentieth centuries are more mixed. Some studies indicate SES differences in all-cause mortality, others show small or no inequalities, casting doubts on the validity of FCT in this period (Bengtsson, Dribe, and Helgertz 2018; Blum, Houdaille, and Lamouche 1990; Chapin 1924; Dribe and Eriksson 2018; Pamuk 1985; Schenk and van Poppel 2011).

The FCT emphasizes the importance of cause-specific mortality (Clouston et al., 2016) but the evidence, particularly in the past, is still inconclusive. There are mixed results on mortality from infectious diseases (Ferrie, 2003, 2001; Leonard et al., 2015) and from cardiovascular, respiratory, and digestive conditions (Breslow and Buell 1960; Costa 2015; 2000;

Crombie and Precious 2011; Eli 2015; Lilienfeld 1956; Logan 1952; Stamler et al. 1960) in the nineteenth and early twentieth century and in the mid-twentieth century (Bainton and Peterson 1963; Dow and Rehkopf 2010; Kitagawa and Hauser 1968; Pell and Fayerweather 1985; Rogot and Hrubec 1989; Vågerö and Norell 1989; Weires et al. 2008).

Based on the FCT, we expect SES differences in adult mortality from the end of the nineteenth century onwards and differences in cause-specific mortality over time. Moreover, such changes should follow the preventability of causes of death, as higher SES groups are able to decrease their mortality risk through earlier access to prevention and interventions (Link and Phelan, 1995; Phelan, et al., 2004; Phelan et al., 2010), because of higher education and income, and better social context (Masters, Link, and Phelan 2015). Several studies have found that SES is indeed more strongly related to preventable mortality (Masters, Link, and Phelan 2015; Phelan et al. 2004; Song and Byeon 2000). Interestingly, SES differences have been found also for non-preventable causes (Ericsson, et al., 2019; Song and Byeon, 2000).

SES differences in non-preventable mortality are not expected to have changed considerably over time. On the one hand, SES differences in preventable mortality are expected to have decreased over time in Sweden, because education has become more egalitarian and more people have access to medical care and knowledge. However, recent findings suggest that, for the last decades, SES differences in preventable mortality have been increasing (Lumme et al., 2018; Masters et al., 2015).

The theoretical framework also suggests that the preventability of diseases changes over time. For instance, SES differences in infectious diseases should be present in the past, when higher SES was related to better living conditions, but not recently, when preventive measures and knowledge reached all social strata. Circulatory and respiratory diseases are largely driven by lifestyle so SES differences over time are expected to follow stratum-specific behavioral changes.

3. Data and Method

We use individual-level longitudinal data from the Scanian Economic-Demographic Database (SEDD) (Bengtsson et al, 2018). It contains information for five rural and semi-urban parishes and a port town in *Skåne*, the southernmost region of Sweden. Individuals are followed across generations from 1813 until 2014. The data for the port town of Landskrona only start in 1922. The data are unique in covering a period of 200 years for which cause-specific mortality can be studies longitudinally at the individual level.

For the period up to 1968, information about demographic events and occupation come from population registers (continuously updated records with individual-level information for each household), vital events registers, poll-tax registers, and annual income- and taxation registers. For the period 1968-2014 data come from administrative registers managed by Statistics Sweden (*SCB*) and the National Board of Health and Welfare (*Socialstyrelsen*), which have been linked to the historical data. Information on migration in and out of the area allowed to precisely calculate the population at risk.

The population under study is representative of most rural and semi-urban areas at the time of study (Dribe, Helgertz, and van de Putte 2015). Moreover, previous studies of overall mortality found patterns of SES differences similar to Sweden as a whole (Bengtsson, Dribe, and Helgertz 2018; Dribe and Eriksson 2018; Lazuka, Quaranta, and Bengtsson 2016).

The linkage of the historical and contemporary data allows us to follow individuals in the area under study from 1968, even after migrating out to another place within Sweden, and their children and grandchildren throughout Sweden. We exploit this extension of the sample in one of the sensitivity analyses.

The period is divided into four sub-periods: 1813-1921, 1922-1967, 1968-1989, 1990-2014. This reflects changes in data sources and availability, and in societal and epidemiological environment of the sample. The first period encompasses a pre-/early industrial society with mortality moving from pre-transitional levels to a continuously increasing life expectancy (Figure 1). Towards the end of this period better hygienic measures started to spread. In addition, restrictions on alcohol production and sale were gradually introduced (Sundin and Willner, 2007, p.139). In the second period, the basis of the modern Swedish welfare state are laid. Cigarette smoking spread from upper and middle classes to other social groups. The last two periods are characterized by rapid economic growth, and consolidation and widening of welfare policies.

Figure 1 here

3.1. SES measure

Occupational status was updated annually between 1815 and 1968 and between 2001 and 2011 (the occupation in the last three years is assumed to be the same as in 2011); in the period in between occupation is available at census years (1970, 1975, 1980, 1985, 1990). Occupational notations have been coded in HISCO (van Leeuwen et al. 2002) and grouped into a 12-category classification: HISCLASS (van Leeuwen and Maas, 2011). In the analysis, we aggregate HISCLASS categories into three groups: non-manual, manual, and farmers. We also run the analysis on a six-category grouping. While HISCLASS mainly measures the social class position, we used it as a proxy for SES, as neither income nor education are available for the entire period.

One of the advantages of using HISCLASS is that it provides a grouping that is relevant over the entire time span. For the period after 1970 it has been shown to produce results that are comparable to other class schemes (Bengtsson et al., 2018). At the same time, when studying earlier periods, HISCLASS captures both social stratification and mobility and demographic

inequalities (Dribe et al. 2017; Dribe, Hacker, and Scalone 2014; Dribe and Helgertz 2016). While it may be problematic to use the same classification over 200 years, it has been shown that occupational hierarchies remain quite stable over time, particularly when using broad SES groups as we do here (Treiman 1976).

Occupation is used as a time-varying variable until the age of 65. Between 65 and 90 we consider the highest recorded occupation between ages 50 and 65, because it should represent the occupation at the peak in terms of SES. Moreover, occupations after age 65 could be misleading because of retirement. For married individuals we select the highest SES within the couple. In a sensitivity analysis we used individual SES regardless of marital status. All individuals are under observation until death, out-migration (outside the parishes before 1968 and outside Sweden afterwards), or end of 2014.

3.2. Outcome variable

The outcome variable is cause-specific adult mortality (ages 30 to 90). For the period before 1968 the causes of death, that were originally recorded as text strings from the parish registers, have been coded in ICD-10 (Hiltunen and Edvinsson 2018). The causes of death for the period after 1968 contained in the administrative register "*Dödsorsaksregistret*" are coded in ICD-8 between 1969 and 1986, in ICD-9 between 1987 and 1996, and in ICD-10 from 1997 onwards.

We group ICD codes according to two classifications. First, we divide causes of death into preventable and non-preventable following the Avoidable Mortality in the European Union (AMIEHS) classification (see also Ericsson et al. 2019). Second, since the preventability of diseases has changed over time, we added a more stable and objective measure of cause-specific mortality, following ICD chapters: (1) infectious and parasitic diseases (including pneumonia and

influenza), (2) circulatory diseases (3) respiratory diseases (including lung, larynx, trachea, bronchus, lip, oral cavity, pharynx cancers) (4) other cancers (5) external causes (6) other and ill-defined causes of death and (7) missing causes of death.

3.3. Statistical method

To estimate differences in cause-specific mortality by SES and how they have developed over time, we use a cause-specific hazard model, which estimates the effect of covariates on the mortality hazard from a specific cause. Each model represents a separate Cox regression in which the event corresponds to a specific cause of death. Individuals who die from a different cause are right censored.

The exponential of the regression coefficient represents the relative change in the cause-specific mortality hazard due to a one unit change in the independent variable (Austin and Fine 2017). Results can be interpreted as the change in the rate of mortality from the disease of interest due to a change in a given covariate among subjects who are still alive. The cause-specific hazard model is well suited for etiological studies (Austin, Lee, and Fine 2016; Koller et al. 2012; Lau, Cole, and Gange 2009).

The analysis was performed by period, separately for men and women. We further control for birth year, marital status (never married, married, and previously married), and parish of residence. We control for migration status in the second, third, and fourth periods. In the initial period there were few people born outside Sweden. We evaluated the assumption of proportional hazards using a test based on the scaled Schoenfeldt residuals (‘estat phtest’ in STATA). For preventable diseases in the third and last periods the test indicated non-proportionality for both men and women, mainly affecting the NA category. A log-log plot indicated that the hazard lines for non-manual and manual occupations were mostly parallel, except for very early ages. The

same holds for non-preventable mortality in the last two periods for women. To deal with this issue we run a sensitivity analysis by dividing the sample into two age groups.

4. Results

Table 1 shows the descriptive statistics of the analyzed sample by gender. Firstly, the changing SES structure over time, with an increasing share of non-manual workers and a decreasing share of farmers, indicates the large societal changes taking place during the period under consideration as Sweden developed from an agricultural society into a modern welfare society (e.g. Schön, 2010).

Table 1 here

Secondly, the parishes of residence have been grouped according to geographical proximity. From 1922 the industrial city of Landskrona is included and it incorporates the largest portion of individuals. The migration indicator highlights a trend of increasing foreign-born population similar to the entire country. Lastly, the cause-of-death groups partially reflect the shift from infectious diseases to man-made diseases, particularly when looking at groups (1), (2), and (3). Tables 2 and 3 report hazard ratios for all-cause, non-preventable and preventable mortality for men and women, respectively.

4.1. All-cause mortality

Results are similar to previous findings for the same area (Bengtsson et al., 2018). In the two more recent periods, after 1968, there are clear SES differences which are even stronger in the last decades (HR=0.571, $p<0.001$). Interestingly, in the second period all-cause mortality is positively associated with SES: the non-manual occupations group shows a 15% higher mortality hazard (HR=1.145, $p<0.001$) than manual workers. In the first period we do not find any

difference between SES groups; only famers show a statistically significant lower mortality than manual workers. While this holds for men, women show a significant advantage in mortality for the non-manual workers in the second period, which increases in the most recent period.

Table 2 and 3 here

4.2. Non-preventable and preventable diseases

For both genders non-manual workers and farmers have an advantage in the last two periods, regardless of preventability. These findings are consistent with the pattern found for the entire country (Ericsson et al. 2019). In the first two periods, we do not find any statistically significant association between SES and mortality for non-preventable mortality, neither for men nor for women. However, in the second period there is a positive association between SES and preventable mortality for men. While women in the non-manual group have lower mortality from preventable causes (HR=0.901, $p<0.05$) men in this group have higher mortality (HR=1.150, $p<0.01$). Supplementary tables 1 and 2 report the estimates for the ill-defined causes in the first two periods and for the missing group in the first period, for men and women respectively. While in the second period there are no significant patterns, men in non-manual occupations and farmer women have a lower hazard in the first period. In both cases the most frequent reported cause of death is “old age”.

In tables 4 and 5 we present the result for the cause-specific mortality using the more detailed subdivision.

Table 4 and 5 here

4.3. Infectious and parasitic diseases

We find evidence for lower mortality for non-manual workers for the last two periods (HR=0.686, $p<0.05$ and HR=0.531, $p<0.001$ for the third and fourth period, respectively). A similar pattern was found in studies analyzing data covering the whole country (Erikson and

Torssander 2008; Weires et al. 2008). In the earlier periods we do not find any statistically significant differences by SES, even though the coefficients suggest a lower mortality for non-manual workers. Women show a pattern similar to men with the exception that the advantage in the second period for the non-manual workers is significant (HR=0.737, $p<0.05$).

4.4. Circulatory diseases

Among men, in the last two periods there are clear and significant differences by SES which widen from the 1968-1989 period (HR=0.748, $p<0.001$) to the final period (HR=0.576, $p<0.001$). Other studies of the entire country for the same period show a similar overall pattern (Kunst et al. 1998; Toch-Marquardt et al. 2014; Weires et al. 2008). Interestingly, in the first two periods, the non-manual workers display a statistically significantly higher mortality (HR=1.248, $p<0.001$ for the second period and HR=2.554, $p<0.001$ for the first period). Women show a similar outcome for the most recent period. The difference with respect to men is that already from the 1920s women in the non-manual group had a lower risk of dying than women in the manual category.

4.5. Respiratory diseases and cancers

The period after 1968 is again characterized by a lower mortality in the non-manual category for both men and women. Before 1968, results show small and statistically insignificant differences by SES. The only exception is for mortality due to other cancers for men in the first part of the twentieth century (HR=1.203, $p<0.05$). In this instance the most frequent diseases are malignant neoplasms of stomach, prostate, rectum, colon, pancreas (about 60% of cases in total).

4.6. External causes

In the last two periods the difference between the non-manual and manual categories is particularly evident, but clear inequalities are present throughout the period of analysis. Similar results were found by Kunst et al. (1998) and Toch-Marquardt et al. (2014). The SES advantage in mortality from external causes is present in the earlier period as well (for women in the first period, the number of events was too small for a meaningful analysis). For this cause of death group it is interesting to note the large protective effect of being married for men and the higher hazard for residing in a more urban area (Landskrona) in the first half of 1900.

4.7. Other and ill-defined causes of death.

In the last two periods, digestive system diseases and endocrine, nutritional, and metabolic disorders are among the most frequent causes for both men and women, together with genitourinary system diseases for men and dementia for women. Among these other causes of death, the non-manual workers have lower mortality for both genders in the last two periods. Before 1970, only men in the second period show statistically significant differences with both the non-manual workers and the farmers having higher mortality. In this case, the most frequent causes are again diseases of the digestive and genitourinary systems, and nutritional and metabolic disorders. Death from "old age" is also relatively frequent in the other causes of death group.

4.8. Analysis with the six-category SES classification

Supplementary tables 3 and 4 report results for the more detailed SES groups. For the last two periods, these tables show a pattern consistent with the one found previously, which provides further evidence for a clear SES gradient with a progressively higher mortality from higher to lower SES, across genders and preventability. For women, a less pronounced SES gradient is

present also in the second period, but it is statistically significant only for higher managers and professionals (HR=0.775, $p<0.05$). For men, there is no clear gradient but the positive association between SES and preventable mortality is confirmed, in particular for lower managers, professionals, and clericals (HR==1.176, $p<0.01$).

Supplementary tables 5 and 6 show mortality inequalities in cause-specific mortality using the more detailed grouping. After 1970, the SES gradient is present for all causes of death, for both men and women (only for infectious diseases and external causes for women in the third period the gradient is not completely clear but the direction of the association remains the same). Looking at circulatory diseases for men, we see the distinct reverse association in the first two periods that shifted in period three and four (a similar pattern can also be observed for other cancers).

4.9. Sensitivity analyses (Results not shown)

In order to check the robustness of our findings we run a set of sensitivity analyses. First we check whether the results are robust to variations in the sample. We do this by running the regressions considering only the five parishes, which allows to have a consistent sample throughout the four periods. Secondly we run the regression including also individuals that after 1968 are living in Sweden but not in the study area. We also provide estimates excluding the foreign-born population in order to check for possible biases introduced by international migration. Then, we test for the robustness of the findings by changing the definition of the exposure from family SES to individual SES. We also check for differences among age groups by splitting the analyses in mortality for ages between 30 and 69, and between 70 and 90. We further analyze SES differences in preventable and non-preventable mortality by using an alternative classification of preventability, namely, the one provided in Phelan et al. (2004).

Finally, we check the sensitivity of our results by using a different SES measure: we run the models using HISCAM scores as a continuous variable as well as HISCAM quartiles calculated by sex and periods. The overall results and patterns described above are robust and do not fundamentally change in any of the tests.

5. Discussion and conclusions

In this paper we studied SES inequalities in cause-specific adult mortality between 1813 and 2014. To our knowledge, in the published literature there is no other study showing how the SES gradient in adult mortality developed over a long period of time for specific causes of death.

In accordance with previous studies, our findings indicate a consistent pattern of mortality advantage for higher-SES groups from the 1970s onwards. Higher-SES groups show a lower mortality risk both for non-preventable and preventable mortality. Similar results have been found in previous studies (e.g., Ericsson et al., 2019). While the lack of variance between non-preventable and preventable causes could indicate that the grouping of causes of death is not well-suited to capture factors affecting SES groups differently, the pattern does not change when using another grouping following Phelan et al. (2004).

In terms of the development of SES inequalities in cause-specific mortality, we find that current SES differences have not been present for very long. Only in the last fifty years did higher-SES groups show a clear advantage compared to lower-SES groups for both genders. Furthermore, the emergence of the SES gradient is a recent phenomenon that happened roughly at the same time for all causes of death considered.

When looking at causes of death, we find a particularly interesting pattern for circulatory diseases in which higher SES men show a higher mortality in the first period that decreases in the second and develops into an advantageous position in the last two periods. A possible explanation

for such a mortality trend, which is also present for other cancers and other causes of death, is related to behavior and life style differences between SES groups, and that sharply changed throughout the period. A healthy diet, smoking, and drinking are often described as potential causes of the mortality gradient, as unhealthy habits are more common in lower-SES groups (Elo 2009; M. Marmot 2005). The same logic can be applied to historical contexts. The difference is that the unhealthy behavior, such as a heavy diet, tobacco smoking, and inactive lifestyle were more common in the higher-SES groups (Dribe and Eriksson 2018). Similarly, Razzell and Spence (2006) focused on behavioral characteristics to explain health differences by SES in pre-twentieth century England. Moreover, men were more exposed to these risks than women (Edvinsson and Lindkvist 2011) and this could explain the gender differences that we find in mortality from circulatory diseases. Especially for the historical period, early-life conditions could also play a role in explaining these results. To the extent that lower SES was associated with higher mortality in early ages, it may have selected stronger individuals, who had lower mortality in adult ages. However, a previous study using sibling fixed-effects models to control for the early-life environment found the same reverse association between SES and life expectancy at age 40 (and age 60), which does not suggest that selection mechanisms can explain this pattern (Dribe and Eriksson 2018).

We also find that the SES differences emerge earlier for women than for men, in particular in infectious diseases. In the second period non-manual workers already show a statistically significant advantage while we find no differences for men. One explanation for this pattern could be the fact that working class women had less bargaining power within the household leading them to be discriminated against in terms of nutrition, and hence more severely affected by infectious disease (Hinde 2015; McNay, Humphries, and Klasen 2005). The second period saw an important reduction in maternal mortality due to improvements in hygienic

conditions, institutionalized maternal health care, and the introduction of sulfa drugs (Sundin and Willner, 2007 p.155). If these improvements benefited women with higher SES more than it did for women with low SES it could explain the earlier emergence of SES differences particularly for infectious diseases. However, previous studies for the nineteenth century have found no class differences in maternal mortality (Andersson, Bergström, and Högberg 2000) and when preventive services for maternal health spread on a large-scale over the country it seemed to have especially benefited lower-SES groups (Burström 2003).

As mentioned above, our results are robust to a series of sensitivity analysis, however a number of limitations should be considered. The linkage between the historical and contemporary sources is based on the personal identity number that was given to everyone alive in 1947. This limitation only affects the results for the contemporary period which have been shown to be comparable with other studies that have full count Swedish population. Another limitation, common to all analyses of cause-specific mortality, stems from the reliability of the causes of death. For the modern period, this may be a problem particularly when the death event is due to more than one disease. However, it has been shown that, the quality of information contained in the Swedish cause of death register is relatively high (Brooke et al. 2017) and that when causes of death are grouped at the chapter level (as it was mainly done in this paper) the accuracy is even higher (Eriksson et al. 2013). For the historical period we have reasons to believe that the reporting of causes of death is fairly reliable. Before 1911, priests who were in charge of the reporting received basic medical training and, in rural areas, where a medical doctor was not present, they were the point of reference for health issues. In the twentieth century, instead, the reliability increased even more for the fact that every cause of death had to be approved by a trained physician who evaluated the report about circumstances surrounding the death of individuals written by the clergymen before the cause of death was actually registered. Before the

early twentieth century, when the reporting of the underlying causes of death was requested for every deceased, there is a higher share of people with missing information. In our study this affects only the first period and, in terms of differences by SES, only men (column 3 in Supplementary table 1).

In conclusion, SES has not always been a "fundamental cause" of mortality, but only emerged as a crucial determinant during the second half of the twentieth century, and especially after 1970. Moreover, when the gradient emerged it did so for both genders and all causes of death. In addition, our findings raise some doubt that it was always the better-off who had lower mortality. They point towards a more nuanced picture in which the impact of SES depends on which coping mechanisms each SES group exploits to avoid risk factors at each point in time. Life style factors and behavioral habits were most likely important mechanisms creating SES differences in mortality over the last two centuries.

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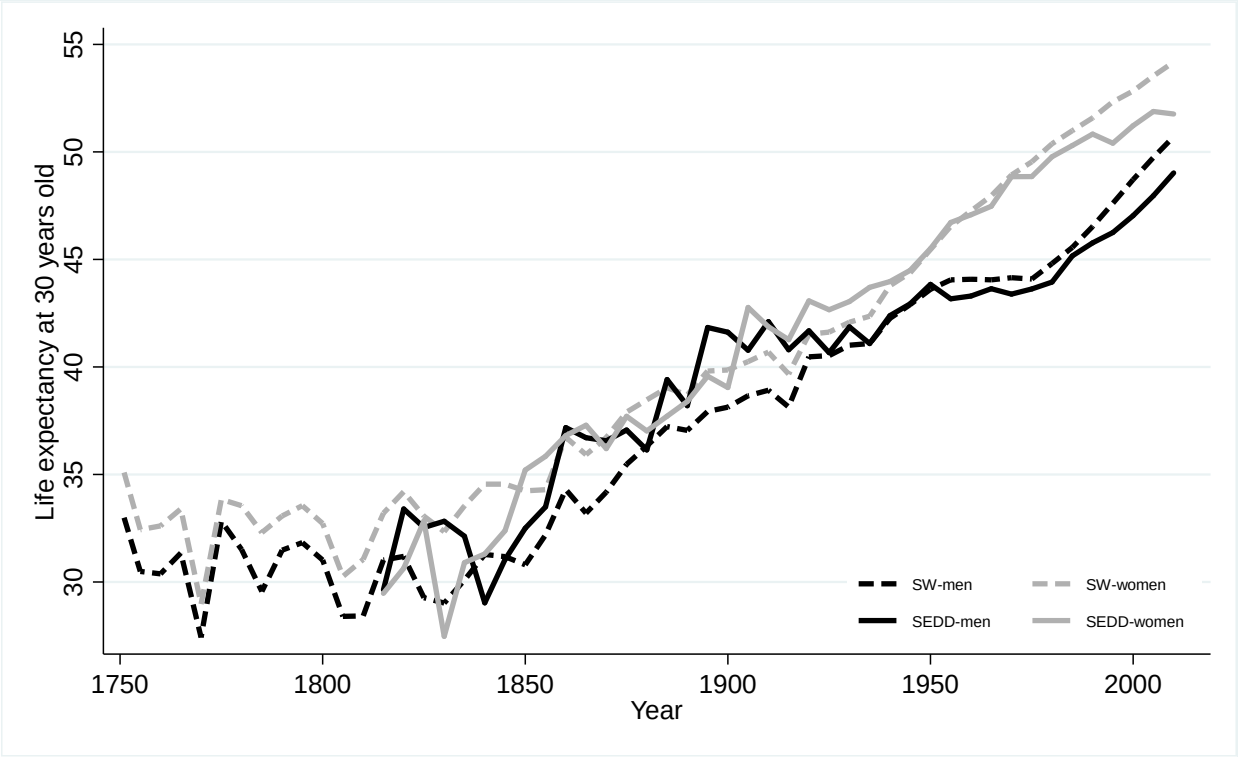
Figures

Figure 1.
Period life expectancy at 30 years old in 5 years intervals for men (black) and women (gray).



NOTE: Period life expectancy for Sweden (dashed) was calculated using data from the Human Mortality Database while for SEDD (solid) it was obtained by calculating the area under the Kaplan Meier survival curve.

Tables

Table 1.
Descriptive statistics for men and women, 30 to 90 years old (five parishes and, from 1922, Landskrona).

	Men				Women			
	1813-1921	1922-1967	1968-1989	1990-2014	1813-1921	1922-1967	1968-1989	1990-2014
SES-6 (%)								
Higher managers/professionals	2.5	8.1	7.8	10.8	2.5	7.6	7.6	9.4
Lower managers/professionals/clerical	6.4	20.4	38.3	39	5.5	23.4	39.8	42
Foremen and medium skilled workers	10	26.7	20.5	15.2	9.2	23.1	17.1	10
Lower skilled workers/farm workers	25.9	24.1	24	22.8	34.5	26.3	23.3	24.2
Unskilled workers/farm workers	27.5	15.5	3.8	3.8	22.9	12.2	6.4	7.3
Farmers and fishermen	24.2	4.4	3	1.4	20.4	3.8	2.6	1.2
NA	3.6	0.8	2.6	7	5.1	3.6	3.2	6
SES-3 (%)								
Non-manual	8.8	28.5	46.1	49.9	7.9	31	47.4	51.4
Manual	63.4	66.3	48.3	41.8	66.6	61.6	46.8	41.4
Farmers	24.2	4.4	3	1.4	20.4	3.8	2.6	1.2
NA	3.6	0.8	2.6	7	5.1	3.6	3.2	6
CIVIL STATUS (%)								
Never married	16.2	18.9	13.7	22.6	18	23.3	9.1	14.8
Currently married	73.4	75.1	73.1	58	65.4	66	63.9	52.5
Previously married	10.4	6	13.3	19.4	16.6	10.7	27	32.7
MIGRANT STATUS (%)								
Born in Sweden	98.8	95.6	87.4	81.3	99.1	95.9	88.7	81.4
Born outside Sweden	1.2	4.4	12.6	18.7	0.9	4.1	11.3	18.6
PARISH OF RESIDENCE (%)								
Hög, Kävlinge	28.6	12.4	11.2	14.6	28.1	12.1	11.1	15
Halmstad, Sireköpinge, Kågeröd	71.4	11.5	6.6	6.3	71.9	10.1	5.8	5.7
Landskrona	0	76.1	82.2	79.1	0	77.8	83.1	79.3
COHORT (mean)								
Birth year	1827.3	1897.6	1925.8	1948.1	1827.3	1896.5	1923.8	1945.9
CAUSE OF DEATH GROUP (%)								
Infectious and parasitic	14.5	10.2	3.4	3.8	14	10.4	4.4	4.5
Circulatory system	4.9	38.3	52.4	42.7	4.7	39.1	49.9	41.2
Respiratory system and lung cancer	6.8	4.3	8.1	11.8	5.3	3.1	4.9	9.5
Other cancers	2.4	17.1	19	21.8	4.2	19.9	24.9	23.8
External causes	4.1	8.4	7.6	5.2	0.8	3	4.5	3.4
Other and ill-defined causes	35.7	20.7	9.1	14.7	36.8	23.6	11.4	17.5
Missing	31.6	0.9	0.2	0.1	34.2	0.8	0.1	0.1
CAUSE OF DEATH PREVENTABLE (%)								
Non-preventable causes	23	28.8	25.6	32.9	20.3	27	26.3	34.9
Preventable causes	18	61.6	73.9	65.8	16.6	60.6	73.5	64.5
Ill-defined causes	27.4	8.6	0.3	1.2	28.9	11.6	0.1	0.6
Missing	31.6	0.9	0.2	0.1	34.2	0.8	0.1	0.1
Deaths	2009	4633	4786	6115	2129	4330	3699	5323
Time at risk	99016.7	344954.4	274102.4	379639	104349.9	366401.3	291272.2	399771.6

Table 2.
All-cause and non-preventable vs preventable mortality, men, ages 30-90 (five parishes and, from 1922, Landskrona).

	All-cause mortality				Non-preventable causes				Preventable causes			
	1813-1921	1922-1967	1968-1989	1990-2014	1813-1921	1922-1967	1968-1989	1990-2014	1813-1921	1922-1967	1968-1989	1990-2014
Non-manual	0.883	1.145***	0.696***	0.571***	1.117	1.106	0.712***	0.604***	1.299	1.150**	0.694***	0.556***
Manual (ref.)	1	1	1	1	1	1	1	1	1	1	1	1
Farmer	0.884*	1.149	0.747***	0.694***	1.099	0.990	0.861	0.656**	0.827	1.209*	0.714***	0.719***
NA	1.107	1.076	0.862	1.073	1.324	1.361	1.176	1.309*	1.427	0.920	0.718	0.954
N of subjects	8141	25354	24369	34266	8141	25354	24369	34266	8141	25354	24369	34266
N of failures (deaths)	2009	4633	4786	6115	463	1336	1227	2011	361	2856	3536	4024
Time at risk (person years)	99016.7	344954.4	274102.4	379639.0	99016.7	344954.4	274102.4	379639.0	99016.7	344954.4	274102.4	379639.0

*p<0.05, **p<0.01, ***p<0.001

Table 3.
All-cause and non-preventable vs preventable mortality, women, ages 30-90 (five parishes and, from 1922, Landskrona).

	All-cause mortality				Non-preventable causes				Preventable causes			
	1813-1921	1922-1967	1968-1989	1990-2014	1813-1921	1922-1967	1968-1989	1990-2014	1813-1921	1922-1967	1968-1989	1990-2014
Non-manual	0.848	0.906**	0.675***	0.635***	0.919	0.944	0.682***	0.667***	0.853	0.901*	0.672***	0.618***
Manual (ref.)	1	1	1	1	1	1	1	1	1	1	1	1
Farmer	0.942	0.993	0.629***	0.589***	1.298*	0.847	0.731	0.523***	0.988	1.075	0.590***	0.618***
NA	1.225	1.082	0.858	1.150*	1.209	1.199	0.723	1.314*	1.268	1.077	0.905	1.065
N of subjects	8131	24213	23252	33018	8131	24213	23252	33018	8131	24213	23252	33018
N of failures (deaths)	2129	4330	3699	5323	432	1170	971	1856	353	2624	2720	3431
Time at risk (person years)	104349.9	366401.3	291272.2	399771.6	104349.9	366401.3	291272.2	399771.6	104349.9	366401.3	291272.2	399771.6

*p<0.05, **p<0.01, ***p<0.001

Table 4.
Cause-specific mortality, men, ages 30-90 (five parishes and, from 1922, Landskrona).

	Infectious and parasitic				Circulatory system				Respiratory system and lung cancer			
	1813-1921	1922-1967	1968-1989	1990-2014	1813-1921	1922-1967	1968-1989	1990-2014	1813-1921	1922-1967	1968-1989	1990-2014
Non-manual	0.839	0.948	0.686*	0.531***	2.554***	1.248***	0.748***	0.576***	0.883	1.094	0.535***	0.477***
Manual (ref.)	1	1	1	1	1	1	1	1	1	1	1	1
Farmer	1.026	1.214	0.738	0.946	0.633	1.032	0.750**	0.689***	0.792	1.174	0.584	0.342***
NA	1.142	1.759*	0.881	1.331	1.107	0.766	0.796	0.843	1.072	0.670	0.929	1.141
N of subjects	8141	25354	24369	34266	8141	25354	24369	34266	8141	25354	24369	34266
N of failures (deaths)	291	474	163	233	99	1775	2510	2609	136	199	390	719
Time at risk (person years)	99016.7	344954.4	274102.4	379639.0	99016.7	344954.4	274102.4	379639.0	99016.7	344954.4	274102.4	379639.0

$^{*}p<0.05$, $^{**}p<0.01$, $^{***}p<0.001$

Table 4 – continued.

	Other cancers			External causes				Other and ill-defined causes			
	1922-1967	1968-1989	1990-2014	1813-1921	1922-1967	1968-1989	1990-2014	1813-1921	1922-1967	1968-1989	1990-2014
Non-manual	1.203*	0.794**	0.642***	0.686	0.744*	0.434***	0.499***	0.787	1.200*	0.652***	0.565***
Manual (ref.)	1	1	1	1	1	1	1	1	1	1	1
Farmer	1.099	0.837	0.819	1.139	0.981	0.736	1.248	0.894	1.385*	0.704	0.625*
NA	0.683	0.693	1.254	2.010	0.433	0.873	1.215	1.203	1.544	1.109	1.112
N of subjects	25354	24369	34266	8141	25354	24369	34266	8141	25354	24369	34266
N of failures (deaths)	792	909	1333	82	388	366	317	718	961	437	896
Time at risk (person years)	344954.4	274102.4	379639.0	99016.7	344954.4	274102.4	379639.0	99016.7	344954.4	274102.4	379639.0

$^{*}p<0.05$, $^{**}p<0.01$, $^{***}p<0.001$

Table 5.
Cause-specific mortality, women, ages 30-90 (five parishes and, from 1922, Landskrona).

	Infectious and parasitic				Circulatory system				Respiratory system and lung cancer			
	1813-1921	1922-1967	1968-1989	1990-2014	1813-1921	1922-1967	1968-1989	1990-2014	1813-1921	1922-1967	1968-1989	1990-2014
Non-manual	1.078	0.737*	0.620*	0.741*	1.369	0.945	0.670***	0.668***	0.148	0.832	0.635**	0.551***
Manual (ref.)	1	1	1	1	1	1	1	1	1	1	1	1
Farmer	1.178	1.110	0.570	0.266	1.043	1.258	0.806	0.661**	1.025	0.485	0.161	0.547
NA	1.256	0.841	0.716	1.769	1.827	1.053	0.897	1.271*	0.897	1.241	0.473	0.577
N of subjects	8131	24213	23252	33018	8131	24213	23252	33018	8131	24213	23252	33018
N of failures (deaths)	297	451	161	241	101	1692	1844	2193	112	136	180	508
Time at risk (person years)	104349.9	366401.3	291272.2	399771.6	104349.9	366401.3	291272.2	399771.6	104349.9	366401.3	291272.2	399771.6

*p<0.05, **p<0.01, ***p<0.001

Table 5 – continued.

	Other cancers				External causes				Other and ill-defined causes			
	1813-1921	1922-1967	1968-1989	1990-2014		1922-1967	1968-1989	1990-2014	1813-1921	1922-1967	1968-1989	1990-2014
Non-manual	0.885	0.950	0.692***	0.647***		0.884	0.680*	0.515***	0.850	0.886	0.687***	0.598***
Manual (ref.)	1	1	1	1		1	1	1	1	1	1	1
Farmer	0.802	0.713	0.413**	0.606*		0.431	0.612	0.882	0.870	0.918	0.622	0.473**
NA	1.800	1.282	0.776	1.052		1.806	1.352	0.604	1.057	1.050	0.829	1.345*
N of subjects	8131	24213	23252	33018		24213	23252	33018	8131	24213	23252	33018
N of failures (deaths)	90	862	920	1267		132	168	181	784	1024	423	930
Time at risk (person years)	104349.9	366401.3	291272.2	399771.6		366401.3	291272.2	399771.6	104349.9	366401.3	291272.2	399771.6

*p<0.05, **p<0.01, ***p<0.001

Supplementary Table 1.
Ill-defined and missing groups, men, ages 30-90 (five parishes and, from 1922, Landskrona).

	Ill-defined causes		Missing
	1813-1921	1922-1967	1813-1921
Non-manual	0.606*	1.247	0.632*
Manual (ref.)	1	1	1
Farmer	0.823	1.358	0.833
NA	0.954	1.118	0.903
N of subjects	8141	25354	8141
N of failures (deaths)	551	397	634
Time at risk (person years)	99016.7	344954.4	99016.7

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Supplementary Table 2.
Ill-defined and missing groups, women, ages 30-90 (five parishes and, from 1922, Landskrona).

	Ill-defined causes		Missing
	1813-1921	1922-1967	1813-1921
Non-manual	0.887	0.817	0.748
Manual (ref.)	1	1	1
Farmer	0.720**	1.086	0.909
NA	1.179	0.929	1.279
N of subjects	8131	24213	8131
N of failures (deaths)	615	503	729
Time at risk (person years)	104349.9	366401.3	104349.9

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Analysis with the six-category SES classification

Supplementary table 3.
Non-preventable vs preventable mortality, men, 30-90, more detailed SES groups (five parishes and, from 1922, Landskrona).

	Non-preventable causes				Preventable causes			
	1813-1921	1922-1967	1968-1989	1990-2014	1813-1921	1922-1967	1968-1989	1990-2014
Higher managers/professionals	1.616	1.023	0.567***	0.595***	1.242	1.083	0.624***	0.467***
Lower managers/professionals/clerical	0.909	1.086	0.740***	0.604***	1.471	1.176**	0.692***	0.538***
Foremen and medium skilled workers	1.000	0.921	0.903	0.912	1.081	0.961	0.936	0.819***
Lower skilled workers/farm workers (ref.)	1	1	1	1	1	1	1	1
Unskilled workers/farm workers	1.118	0.988	1.294*	1.471***	1.159	1.064	1.051	1.282***
Farmers and fishermen	1.145	0.963	0.862	0.656**	0.890	1.224*	0.702***	0.685***
NA	1.393	1.322	1.179	1.329*	1.550	0.930	0.705	0.919
N of subjects	8141	25354	24369	34266	8141	25354	24369	34266
N of failures (deaths)	463	1336	1227	2011	361	2856	3536	4024
Time at risk (person years)	99016.7	344954.4	274102.4	379639.0	99016.7	344954.4	274102.4	379639.0

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Supplementary table 4.
Non-preventable vs preventable mortality, women, 30-90, more detailed SES groups (five parishes and, from 1922, Landskrona).

	Non-preventable causes				Preventable causes			
	1813-1921	1922-1967	1968-1989	1990-2014	1813-1921	1922-1967	1968-1989	1990-2014
Higher managers/professionals	1.191	1.026	0.508***	0.526***	0.747	0.775*	0.610***	0.438***
Lower managers/professionals/clerical	0.730	0.910	0.670***	0.694***	0.967	0.949	0.665***	0.659***
Foremen and medium skilled workers	0.888	0.898	0.722***	0.809*	1.322	1.014	0.712***	0.844**
Lower skilled workers/farm workers (ref.)	1	1	1	1	1	1	1	1
Unskilled workers/farm workers	0.967	1.118	1.222*	1.295***	0.953	1.038	1.409***	1.306***
Farmers and fishermen	1.265	0.847	0.688	0.527***	1.019	1.089	0.575***	0.631***
NA	1.184	1.190	0.695	1.333*	1.318	1.094	0.907	1.093
N of subjects	8131	24213	23252	33018	8131	24213	23252	33018
N of failures (deaths)	432	1170	971	1856	353	2624	2720	3431
Time at risk (person years)	104349.9	366401.3	291272.2	399771.6	104349.9	366401.3	291272.2	399771.6

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Supplementary table 5.
Cause-specific mortality, men, 30-90, more detailed SES groups (five parishes and, from 1922, Landskrona).

	Infectious and parasitic				Circulatory system				Respiratory system and lung cancer			
	1813-1921	1922-1967	1968-1989	1990-2014	1813-1921	1922-1967	1968-1989	1990-2014	1813-1921	1922-1967	1968-1989	1990-2014
Higher managers/professionals	1.039	0.942	0.426	0.560	3.422**	1.237*	0.687***	0.515***	1.699	0.623	0.339***	0.357***
Lower managers/professionals/clerical	0.966	1.077	0.896	0.533***	2.236*	1.204**	0.730***	0.546***	0.441	1.146	0.579***	0.488***
Foremen and medium skilled workers	1.176	0.920	1.228	0.964	1.285	0.976	0.909	0.805***	0.912	0.879	0.981	0.863
Lower skilled workers/farm workers (ref.)	1	1	1	1	1	1	1	1	1	1	1	1
Unskilled workers/farm workers	1.370	1.473**	1.559	1.366	0.899	0.925	1.036	1.284**	1.233	0.842	1.064	1.411*
Farmers and fishermen	1.200	1.409	0.854	0.958	0.643	0.995	0.728**	0.651***	0.843	1.072	0.584	0.334***
NA	1.373	2.032*	1.027	1.363	1.121	0.739	0.773	0.808	1.153	0.610	0.927	1.131
N of subjects	8141	25354	24369	34266	8141	25354	24369	34266	8141	25354	24369	34266
N of failures (deaths)	291	474	163	233	99	1775	2510	2609	136	199	390	719
Time at risk (person years)	99016.7	344954.4	274102.4	379639.0	99016.7	344954.4	274102.4	379639.0	99016.7	344954.4	274102.4	379639.0

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Supplementary table 5 – continued.

	Other cancers				External causes			
	1813-1921	1922-1967	1968-1989	1990-2014	1813-1921	1922-1967	1968-1989	1990-2014
Higher managers/professionals		1.401*	0.688*	0.665***		0.526*	0.343***	0.340***
Lower managers/professionals/clerical		1.309*	0.810*	0.610***		0.747	0.417***	0.590***
Foremen and medium skilled workers		1.097	0.923	0.830*		0.808	0.801	1.105
Lower skilled workers/farm workers (ref.)		1	1	1		1	1	1
Unskilled workers/farm workers		1.290*	1.243	1.529***		0.995	1.188	1.497
Farmers and fishermen		1.246	0.835	0.791		0.925	0.697	1.353
NA		0.774	0.692	1.242		0.405	0.828	1.321
N of subjects		25354	24369	34266		25354	24369	34266
N of failures (deaths)		792	909	1333		388	366	317
Time at risk (person years)		344954.4	274102.4	379639.0		344954.4	274102.4	379639.0

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Supplementary table 6.
Cause-specific mortality, women, 30-90, more detailed SES groups (five parishes and, from 1922, Landskrona).

	Infectious and parasitic				Circulatory system				Respiratory system and lung cancer			
	1813-1921	1922-1967	1968-1989	1990-2014	1813-1921	1922-1967	1968-1989	1990-2014	1813-1921	1922-1967	1968-1989	1990-2014
Higher managers/professionals	0.892	1.039	0.889	0.520	1.656	0.811	0.608***	0.582***		0.397	0.509	0.231***
Lower managers/professionals/clerical	1.199	0.726*	0.570*	0.735	1.258	1.013	0.672***	0.682***		0.754	0.585**	0.626***
Foremen and medium skilled workers	0.967	1.043	0.736	0.779	1.500	1.029	0.784***	0.869		0.802	0.687	0.761
Lower skilled workers/farm workers (ref.)	1	1	1	1	1	1	1	1		1	1	1
Unskilled workers/farm workers	1.058	1.247	1.534	1.041	0.755	1.080	1.286***	1.166*		0.500*	1.057	1.541***
Farmers and fishermen	1.200	1.202	0.577	0.256	1.038	1.293*	0.791	0.663**		0.394	0.146	0.564
NA	1.273	0.891	0.745	1.694	1.889	1.085	0.897	1.278*		1.033	0.436	0.609
N of subjects	8131	24213	23252	33018	8131	24213	23252	33018		24213	23252	33018
N of failures (deaths)	297	451	161	241	101	1692	1844	2193		136	180	508
Time at risk (person years)	104349.9	366401.3	291272.2	399771.6	104349.9	366401.3	291272.2	399771.6		366401.3	291272.2	399771.6

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Supplementary table 6 – continued.

	Other cancers				External causes			
	1813-1921	1922-1967	1968-1989	1990-2014	1813-1921	1922-1967	1968-1989	1990-2014
Higher managers/professionals	1.358	0.806	0.474***	0.376***		1.294	0.933	0.611
Lower managers/professionals/clerical	1.013	0.975	0.685***	0.655***		0.888	0.619*	0.620*
Foremen and medium skilled workers	1.792	1.007	0.604***	0.640***		1.102	0.670	1.199
Lower skilled workers/farm workers (ref.)	1	1	1	1		1	1	1
Unskilled workers/farm workers	1.344	0.932	1.586***	1.358***		1.327	1.692*	1.788**
Farmers and fishermen	1.014	0.702	0.387***	0.577*		0.480	0.605	1.061
NA	2.177	1.270	0.769	1.022		1.974	1.407	0.737
N of subjects	8131	24213	23252	33018		24213	23252	33018
N of failures (deaths)	90	862	920	1267		132	168	181
Time at risk (person years)	104349.9	366401.3	291272.2	399771.6		366401.3	291272.2	399771.6

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$