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## Disparities in death

### Inequality in cause-specific infant and child mortality in Stockholm, 1878–1926

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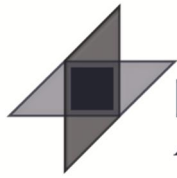
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## **Disparities in death: Inequality in cause-specific infant and child mortality in Stockholm, 1878–1926**

**Joseph Molitoris**

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## **Disparities in death: Inequality in cause-specific infant and child mortality in Stockholm, 1878–1926**

**Joseph Molitoris<sup>1</sup>**

### **Abstract**

#### **BACKGROUND**

The decline of child mortality during the late 19th century is one of the most significant demographic changes in human history. However, there is evidence suggesting that the substantial reductions in mortality during the era did little to reduce mortality inequality between socioeconomic groups.

#### **OBJECTIVE**

The aim of this study is to examine the development of socioeconomic inequalities in cause-specific infant and child mortality during Stockholm's demographic transition.

#### **METHODS**

Using an individual-level longitudinal population register for Stockholm, Sweden, between 1878 and 1926, I estimate Cox proportional hazards models to study how inequality in cause-specific hazards of dying from six categories of causes varied over time. The categories included are 1) airborne and 2) food and waterborne infectious diseases, 3) other infectious diseases, 4) noninfectious diseases and accidents, 5) perinatal causes, and 6) unspecified causes.

#### **RESULTS**

The results show that class differentials in nearly all causes of death converged during the demographic transition. The only exception was the airborne infectious disease category, for which the gap between white-collar and unskilled blue-collar workers widened over time.

#### **CONCLUSIONS**

The results demonstrate that, even in a context of falling mortality and a changing epidemiological environment, higher socioeconomic groups were able to maintain a health advantage for their children by reducing their risks of dying from airborne disease to a greater extent than other groups. Potential explanations for these patterns are suggested, as well as suggestions for future research.

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## **CONTRIBUTION**

This is the first paper to use individual-level cause-of-death data to study the long-term trends in inequality of cause-specific child mortality during the demographic transition.

## **1. Introduction**

The greatest threats to human health and longevity in historical populations were infectious diseases caused by unsanitary living conditions and unclean food and water, and there was perhaps no group at greater risk of succumbing to these threats than children (see e.g., Mercer 1986; Omran 1971; Preston and Haines 1991). Especially a scourge of industrial cities where transmission was rapid, infectious diseases caused urban mortality rates to dwarf their rural counterparts for much of the industrial era (Cain and Hong 2009; Condran and Crimmins 1980; Knodel 1977). For those children who survived beyond the critical first years of life, the severe disease burden during childhood could have long-lasting impacts on their health and well-being as well as on their individual economic outcomes (Bengtsson and Lindström 2000; Fogel 2004; Hatton 2011; Quaranta 2013). It therefore cannot be overemphasized how important the mortality and health transitions were in improving living standards in both the short and long run, as these changes not only led to greater life expectancy but also a larger share of life spent in good health. Some economists have even argued that such dramatic gains in health and life expectancy may have been important inputs into the transition to modern economic growth (Aksan and Chakraborty 2014; Cervellati and Sunde 2011; Fogel 2004; Fogel and Costa 1997; Morand 2004).

Despite these improvements, we know that reductions in child mortality were not necessarily equally distributed throughout societies. Socioeconomic disparities in all-cause mortality at younger ages were clearly present during the mortality transitions of Western populations, with wealthy groups often having substantially lower death rates than the working classes (see Antonovsky 1967; Bengtsson 2004:161; Breschi et al. 2011; Edvinsson et al. 2005; Haines 1989, 2011; Molitoris and Dribe 2016a; Ó Gráda 2004; Preston and Haines 1991: 120). Furthermore, some evidence suggests that class differences in infant and child mortality, while changing in absolute terms, may have remained virtually constant in relative terms throughout the mortality decline, particularly between the highest and lowest socioeconomic groups (Antonovsky and Bernstein 1977; Haines 2011; Molitoris and Dribe 2016a). However, it is poorly understood why differentials would persist during an era in which living standards, particularly for the lower classes, seem to have been improving by virtually any measure.

This study aims to shed light on why all-cause mortality inequality persisted by examining how socioeconomic inequality in various causes of death developed during Stockholm's mortality and health transitions. Although it is beyond the scope of the paper to exactly quantify the influence of various factors (e.g., medical care, sanitation, nutrition) on overall mortality differentials, understanding how the underlying inequalities in cause-specific mortality changed during the period can inform our understanding of the causes behind the process. This is accomplished by using data from an individual-level, longitudinal population register with cause-of-death information to estimate event history models in a competing risks framework. Such an analysis can also provide a multidimensional view of the development of living standards inequality during industrialization. Mortality's sensitivity to social, economic, and environmental conditions, particularly among children, makes it a strong indicator of living conditions, and as a result it has been incorporated into major standard-of-living indices, such as the Human Development Index (United Nations 1990). The advantage of studying cause-specific rather than all-cause mortality is that different causes of death can be more easily linked to their proximate determinants. This study is unique, as individual-level cause-of-death information is rare in historical settings, especially for a large industrial city like Stockholm. It can therefore also contribute to our understanding of social inequality during the broader urban health transition.

## **2. Background**

The sustained decline of infant and child mortality in Stockholm did not begin until the 1860s (Edvinsson, Gardarsdóttir, and Thorvaldsen 2008). Until then, infant mortality rates were consistently above 300 per 1,000 births, about 20% higher than the rates for Sweden as a whole at the start of its own mortality decline in the late 18th century. Once Stockholm's mortality rates began to fall, they did so with remarkable speed compared to the experience of the rest of the country. Within 50 years the infant mortality rate in the city had been reduced by more than two-thirds, while the Swedish population needed more than a century to achieve a reduction of the same magnitude.

Stockholm's rapid mortality transition was characterized by large socioeconomic differences in all-cause mortality at young ages. Several studies of infant and child mortality covering different areas of Stockholm have shown that between 1878 and 1925 manual workers had 70%–100% higher infant mortality than those of the upper classes and that these differentials showed no sign of disappearing (Burström and Bernhardt 2001, Burström et al. 2005, Molitoris and Dribe 2016a). The persistence of socioeconomic differences in all-cause child mortality during this period is puzzling, as it was a time in which significant sanitary reforms had tackled many of the city's health

hazards, such as unclean water and food, and economic growth had led to a threefold increase in the real wages of unskilled workers (Molitoris and Dribe 2016a). It is therefore the aim of this paper to examine how socioeconomic differences in cause-specific infant and child mortality developed in order to better understand how mortality differentials could remain in spite of historical trends, which would seemingly have removed the underlying conditions enabling the perpetuation of inequality in all-cause mortality.

Link and Phelan (1995) provide a framework to explain why, despite large declines in some of the deadliest diseases in the past, socioeconomic differences in health may persist in the long run. They argue that socioeconomic status is a “fundamental social cause” of disease. A fundamental social cause is one which has four main features. First, it influences multiple disease outcomes. Second, those outcomes are affected by more than one risk factor. Third, it involves access to economic and social resources, such as income, knowledge, political clout, and social networks, which can be used to avoid illness as well as to minimize the negative effects of disease. Fourth, the relationship between fundamental causes and health is reproduced over time as new dominant diseases emerge and as knowledge and treatments of disease are developed. It is worth noting that this is not the only framework for understanding socioeconomic differences in mortality over time, as Antonovsky (1967) argues that disparities in mortality follow a pattern of divergence and eventual convergence. This is because the early gains of industrialization first reached higher socioeconomic groups, which led to greater inequality in mortality from nutrition-dependent diseases, but later raised the living standards of the remainder of the population, thereby leading to a convergence of mortality across socioeconomic groups. I have chosen to utilize the ‘fundamental causes’ framework, as it is more explicit about how changes in all-cause mortality differentials relate to changes in the underlying distribution of cause-specific mortality.

The theory of fundamental causes thus hinges on the unequal distribution of resources that individual agents can use to alter exposure to the proximate determinants of mortality. It should be made clear here that ‘resources’ can be interpreted in a broad sense, ranging from monetary wealth to social capital. For example, different socioeconomic groups may be more or less able to afford medical care or they may have more or less beneficial social networks that may allow them to get access to better housing. The proximate determinants include maternal factors (e.g., birth intervals, age at birth), personal illness control (e.g., medical care, personal hygiene, stress management), environmental contamination (e.g., pollution), injury, and nutrient deficiency (Mosley and Chen 1984). If investments in public health, such as the increased availability of clean water, make exposure less dependent on individual resources, there should be smaller socioeconomic discrepancies in causes of death

related to those sources of exposure. Thus, the degree to which each of the proximate determinants influences overall mortality differentials is not fixed, but rather varies over time as individual resources become more or less important in altering specific sources of exposure.

Although explicitly quantifying the effects of each of the proximate determinants in relation to socioeconomic mortality differentials is beyond the scope of this paper, it is worthwhile to briefly explore how the relative importance of some of these factors may have shifted during Stockholm's mortality decline, as this will be relevant to interpreting how changes in cause-specific mortality inequality developed during the period.

## **2.1 Maternal factors**

It has been shown that children born to mothers at young ages and following short intervals have a higher risk of dying and experiencing other negative postnatal outcomes (Hobcraft, McDonald, and Rutstein 1983, 1985; Rutstein 2005; Conde-Agudelo et al. 2012). In Stockholm at the turn of the 20th century these characteristics did not differ much between socioeconomic groups (Molitoris and Dribe 2016b). For women born between 1860 and 1881 there were virtually no socioeconomic differences in either the mean age at first birth or the average closed birth interval. The mean age at first birth declined across these cohorts from just over 26 years old to slightly older than 24, but class differences in the age at first birth within cohorts never varied by more than a couple of months. Furthermore, in many cohorts it was actually working-class women who tended to enter motherhood later, not the upper classes. Regarding closed birth intervals, there was a more or less uniform upward trend in the mean time between births across cohorts, increasing from 2.5 to 3.3 years. This pattern was visible among women in all socioeconomic groups, and class differences in birth intervals never widened beyond three months. As with the mean age at first birth, in many cohorts it was upper-class women who were in the theoretically disadvantaged position, as they often had slightly shorter intervals than other groups. There were class-specific differences in maternal factors, in the sense that the working classes tended to have substantially higher fertility in the 1870s, but these differences actually disappeared over time (Molitoris and Dribe 2016b). Furthermore, higher fertility per se does not necessarily influence child health via maternal factors, but likely through other proximate determinants, such as exposure to disease resulting from residential crowding. It therefore seems unlikely that systematic socioeconomic differences in maternal characteristics played an important role in perpetuating mortality differentials.



## 2.2 Personal illness control

Given the state of the medical knowledge of the time, it is also hard to imagine that differences in personal illness control would be responsible for class differences in almost any cause of death, especially prior to the 20th century. Until the late 19th century, smallpox was the only major disease for which an effective therapy existed, and vaccination had already been mandatory for all Swedish children since 1816, suggesting that there should have been no socioeconomic advantage in receiving treatment. Furthermore, smallpox was practically nonexistent in Stockholm after the city's last serious epidemic in 1874 (Nelson and Rogers 1992). In fact, in the following 25 years of the 19th century there were only 38 registered deaths from smallpox (Stockholm City Research and Statistics Office 2004).

Tuberculosis, on the other hand, was rampant in Stockholm in the late 19th and early 20th centuries (Puranen 1984: 274), especially among children, yet there were no effective treatments for it. At the turn of the 20th century, treatment for tuberculosis consisted of living in sanatoria for months, if not years. These patients were prescribed to breathe fresh air and take walks (Wallstedt and Maeurer 2015). The only way that access to these facilities may have influenced mortality would have been by removing infected individuals from their families, thereby limiting families' exposure. Considering that these were typically small institutions and that the first state sanatorium was only opened in 1900 (Wallstedt and Maeurer 2015), their influence on mortality inequality most likely was limited.

Of course, personal illness control was not solely related to the use of medical care. Although the importance of advances in general hygienic practices has generally been assigned a secondary role in the mortality decline (McKeown, Brown, and Record 1972), there is evidence that these practices could also have served to reduce mortality in the past and that they may have differed along socioeconomic lines in England and Wales (Razzell 1974). For example, there may have been differences in general cleanliness across groups in terms of washing food or bathing. In Stockholm, it is unclear to what extent these practices varied across groups. Based on schoolchildren's essays from 1912, well after the start of this study's coverage, the general view of hygienic living placed a good deal of emphasis on the importance of keeping a home well-lit with sunlight and full of fresh air, and it is easy to imagine how access to these would vary across socioeconomic groups. After all, both of these would be highly contingent on the physical structures in which individuals resided.<sup>2</sup> Unfortunately, it is difficult to separate these factors from the role of environmental contamination as a proximate determinant, as many preventative measures, for example, washing food or

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<sup>2</sup> An example of one such essay may be found at: <http://www.stockholmskallan.se/Soksida/Post/?nid=29297>.

bathing, will only be effective illness control strategies if they can be done with clean water.

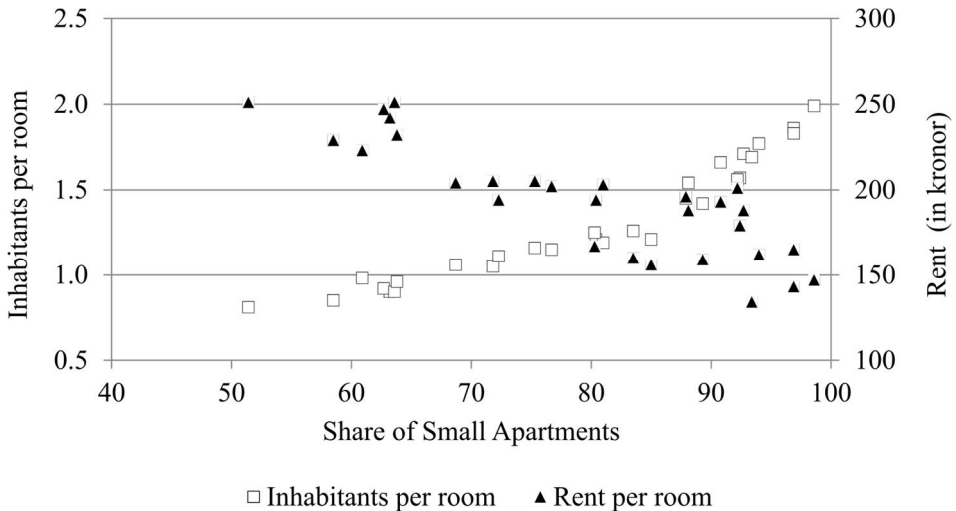
### **2.3 Environmental contamination**

Differences in environmental exposure to disease were likely much more important for sustaining mortality inequality across socioeconomic groups than differences in maternal factors or medical care. As examples let us consider two common environmental sources of disease in developing contexts: unclean water and residential crowding. The early phases of the expansion of Stockholm's water network disproportionately favored the wealthier neighborhoods in Stockholm. This is significant, as recent work on the Estonian city of Tartu has shown that educational differences in infant mortality could largely be accounted for by differences in access to clean water (Jaadla and Puur 2016). The city's first water mains were laid in 1861, primarily near the royal residence and in the well-to-do surrounding areas. Several water pumps were distributed throughout the city and could be used free of charge, but individual property owners were responsible for financing connections to the main network for direct access to the home. This served as an entry barrier to many working-class families and resulted in early declines in child mortality from waterborne diseases among the city's wealthier inhabitants relative to other socioeconomic groups (Burstrom et al. 2005). Compounding the problem was the fact that most working-class areas of the city did not have any water mains until several decades after the first pipes were laid (Hansen 1897), meaning that, even if families in these areas could afford to have direct access to clean water, they did not have the possibility of doing so without moving to more expensive districts. By around the turn of the 20th century, piped water had reached most of the population, and this coincided with the equalization of infant and child mortality from some waterborne diseases across socioeconomic groups (Burstrom et al. 2005), suggesting that individual resources may have become less important in securing clean drinking water than previously.

As water access for all improved, residential crowding increasingly became a characteristic of the working classes, leading to heightened risks of airborne infection (e.g., diphtheria, measles, tuberculosis). In the two heavily working-class districts of Kungsholmen and Södermalm, over 90% of apartments were classified by the city government as "small apartments" (i.e., one to three rooms and a kitchen) in 1910 (Stockholm Stads Statistiska Kontor 1915). On the other hand, in the wealthy districts of lower Norrmalm and Östermalm many fewer apartments fitted this description (about 69% and 63% respectively). Though the number of small apartments is not in itself a sign of poor living conditions, it is a good indication of the potential for

residential crowding. Figure 1 shows that the share of small apartments in Stockholm's districts in 1910 had a strong positive correlation with the number of inhabitants per room. Unsurprisingly, districts with a large share of small apartments also tended to have lower rents per room, suggesting that individual resources continued to be extremely important in avoiding exposure to diseases associated with crowding – which also happened to be the deadliest diseases of the day.

**Figure 1: Inhabitants per room and rent per room by the share of small apartments in Stockholm's districts, 1910**



Notes: 'Small apartments' refers to apartments with one to three rooms and a kitchen.

Source: Stockholm Stads Statistiska Kontor (1912). *Statistisk Årsbok för Stockholms Stad 1912 (1910, 1911)*. Kartogram 2, 3, & 5.

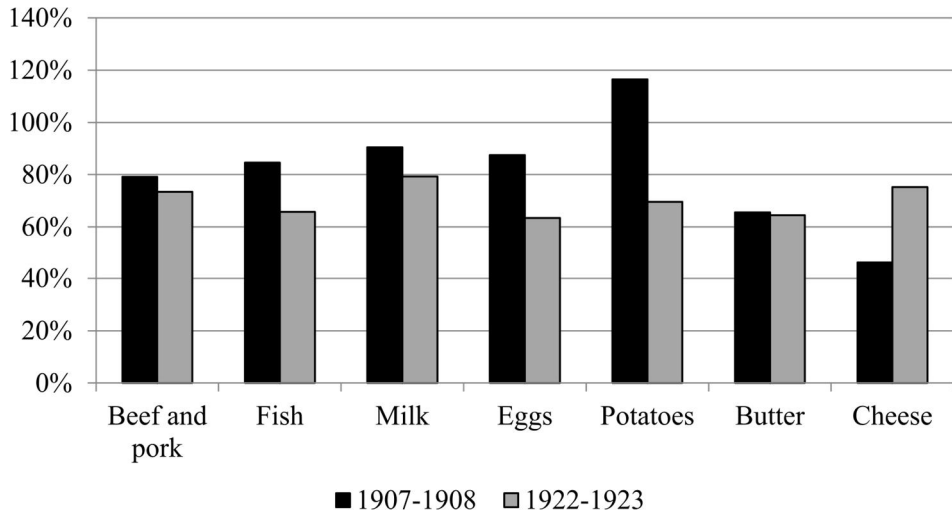
## 2.4 Nutrient deficiency

Finally, nutritional differences in terms of the quantity and quality of calories consumed varied considerably between socioeconomic groups and may have also played an important part in perpetuating mortality differentials. Many of the deadly diseases that plagued Stockholm were very much influenced by their host's nutritional status and especially their protein consumption (Rice et al. 2000). These included some of the most lethal airborne killers, like tuberculosis, measles, and pertussis (Lunn 1991). A survey conducted in 1907–1908 concerning the consumption patterns of working-class households in Stockholm showed that, as one would expect, wealthier families spent

much lower proportions of their income on food, but also consumed about 20% more protein per capita (Stockholms Stads Statistiska Kontor 1910). It appears that this discrepancy widened during the first decades of the 20th century. Despite improvements in nutrition for all income groups, a similar survey conducted in 1922 showed that the gap in protein consumption had increased to about 30% (Stockholm Stads Statistiska Kontor 1927), suggesting that nutritional inequality was present in Stockholm well into the 20th century. Figure 2 shows the per capita consumption of low-income households (less than 1,300 kronor) as a share of the per capita consumption of middle-income households (more than 2,000 kronor) as reported in the 1907–1908 and 1922–1923 surveys, respectively. What is striking is how the relative differences in consumption widened over only a decade and a half. The growing disparity is especially clear for arguably the most important and relatively inexpensive source of protein for working class families, eggs. In 1907–1908, higher-income households purchased about 14% more eggs per capita, and this gap had increased to 37% by 1922–1923. That households with higher levels of income had better nutrition is not surprising, but that this gap increased over time may be an important reason why airborne mortality differentials failed to disappear.

The above discussion highlights the dynamic nature of the relationship between individual resources and exposure to the proximate determinants of mortality. It also suggests that, even if overall mortality differentials remained constant during the mortality decline, the underlying distribution of causes of death should have shifted over time. In particular, causes that were more highly dependent on personal resources, such as airborne diseases via housing and nutrition, should have become more differentiated along class lines than those for which resources became less advantageous, such as waterborne disease due to the expansion of the municipal water supply. At the same time, causes of death for which therapies were ineffective or whose risk factors were poorly understood, such as deaths resulting from perinatal causes, should not have disproportionately influenced the working classes. The following sections will examine how the distribution of causes of death changed between 1878 and 1926 and how socioeconomic differences in specific causes evolved.

**Figure 2: Per capita consumption in low-income households as a share of per capita consumption in middle-income households for specific commodities in 1907–1908 and 1922–1923**



Notes: Low income refers to individuals with an annual salary less than 1,300 kronor and middle income refers to individuals with a salary of more than 1,950 kronor. These categories were provided by the original surveys. A value of 100% signifies that the per capita consumption of a particular good was equal across income groups in the respective survey, while a value of 50% would indicate that low-income households consumed half the amount of a specific good per capita.

Source: For 1907–1908: Stockholms Stads Statistiska Kontor (1910). *Statistisk Undersökning Angående Lefnadskostnaderna i Stockholm, Åren 1907–1908*. Stockholm: K.L. Beckmans Boktryckeri. For 1922–1923: Stockholms Stads Statistiska Kontor (1927). *Statistisk Undersökning Angående Levnadskostnaderna i Stockholm 1922/1923*. Stockholm: K.L. Beckmans Boktryckeri.

### 3. Data

This study uses longitudinal, individual-level register data from the Roteman Database, which is maintained by the Stockholm City Archives. The data was collected between the years of 1878 and 1926 and includes all individuals who lived in the city during that time. This was done by dividing the city into districts, which were each assigned an administrator to maintain the local records. Administrators would record all movements to and from their district and also events such as births, deaths, and marriages. Individuals would be entered into the local registers upon entering a district and their information would be updated annually at the time of census registration (Geschwind and Fogelvik 2000). By the end of the Roteman system in 1926, the number of registration districts in the city had expanded from 16 to 36, due to the expansion of the

city's borders and to the subdivision of existing districts. To account for this, the 36 districts have been condensed into seven time-invariant districts: Gamla Stan, Norrmalm, Kungsholmen, Östermalm, Södermalm-East, Södermalm-West, and Brännkyrka. At present, data from 26 of the 36 original districts have been digitized. For each individual in the database there is information on birth date and place, location of residence within Stockholm, occupational titles, sex, and position in the household (i.e., servant, lodger, child, etc.). The present work will utilize a subsample of the database that contains individual-level causes of death in addition to the abovementioned information.

Socioeconomic groups were defined by the occupation of the head of household, which could be the mother, father, or, in rare circumstances where the child was adopted by another member of the family, another relative such as an uncle or aunt. Occupations were coded using the Historical International Standard Classification of Occupations (HISCO) (van Leeuwen, Maas, and Miles 2002) and then classified according to HISCLASS (van Leeuwen and Maas 2011), which groups occupations according to the level of skill required, the degree of supervision, and the rural or urban character of work. The original HISCLASS scheme produces 12 distinct categories of workers. However, to avoid problems with small numbers in the multivariate analysis later on, these 12 categories have been condensed into three: 1) white-collar/non-manual workers (HISCLASS 1–5), 2) skilled manual workers (HISCLASS 6-8), and (3) low-skilled/unskilled manual workers (HISCLASS 9-12). In addition, a fourth category was included for those who had no occupational information available. Among white-collar workers, the majority (about 70%) worked as lower clerical and sales personnel while the remainder had higher management positions or other professions requiring advanced education and experience.

The data used in this study is a subset of the larger database and includes all children observed at any point between birth and age 10 during the coverage period (January 1, 1878 to December 31, 1926) in a district with any digitized cause-of-death information, as well as any individuals sharing the same household (e.g., family members, boarders). Of the 26 digitized districts, 16 had some causes of death recorded, but 6 of these were excluded from the analysis because they had small shares of deaths with recorded causes. The remaining 10 districts were in the time-invariant districts of Gamla Stan, Södermalm-East, and Södermalm-West. In total, 193,833 children and over 25,400 infant and child deaths can be observed between 1878 and 1926 in this subsample, or about 50% of all the deaths occurring under age 10 in the entire Roteman Database. Individual cause-of-death information is available for nearly 16,500 children, or 65% of the observed deaths occurring in the districts with any digitized causes of death. The large number of deaths missing a recorded cause is due to the fact that only a portion of records have been digitized to date. The only selection that was introduced

into the digitization of death records was that it was based on district of residence in Stockholm at the time of death (see Appendix Table A-1 for a description of the district selection). In some cases, primary and secondary causes of death are listed, but because this was by no means the norm, this study only utilizes primary cause-of-death information.

It should be noted that historical sources on causes of death are not without problems (see e.g., Risse 1997; Rosenberg 1989). Besides changing definitions of diseases over time, changes in administrative procedures can also affect both the quality and quantity of reporting. Starting in 1749, parish priests in Sweden were required by the Tabular Commission (*Tabellverket*) to record this information when their parishioners died (Rogers 1999). The difficulty in correctly identifying many causes of death, in addition to the time constraints faced by priests as a result of their wide-ranging responsibilities, could at times lead to under-registration of causes and ambiguous terminology. By the time the present data began (i.e., 1878), the Central Bureau of Statistics (*Statistiska centralbyrån*) had required for nearly twenty years that urban cause-of-death information be based on death certificates signed by a physician (Rogers 1999). Although this does not ameliorate all problems with the data, it suggests that that information present in the records is as reliable as can be expected for the time. Nevertheless, it is clear from some of the terminology used that doctors were not always able to easily identify the cause of death. There is perhaps no better example for children than the commonly used cause of death, “congenital weakness.”

Changes in the quality and quantity of reporting causes of death obviously have a critical influence on the results of this study. It is certainly plausible that individuals from different social strata were more or less likely to have reported causes based on, for example, differences in the extent to which they may have interacted with health care institutions. Likewise, these differences in coverage may well have changed over time. In order to investigate these potential pitfalls, a logistic regression was used to estimate predicted probabilities of having a reported versus unreported cause of death for all child deaths occurring during the study period (see Figure A-1 in the Appendix). The probabilities are all conditional on the same covariates that will be included in the survival models later in the analysis. Panel a) of the figure shows that there were indeed improvements in coverage over time. Individuals dying later in the data’s coverage were more likely to have a recorded cause of death than those born earlier. Of course, this is to be expected, as medical knowledge and the role of formal medicine expanded rapidly during this period. This is not in itself a major concern, as long as differences in coverage did not change at different speeds across socioeconomic groups. Panels b) and c) show socioeconomic differences in the predicted probability of having a missing cause of death over time. In almost all periods the differences in the predicted probabilities of dying from an unknown cause of death were not statistically significant.

In a few isolated periods, children in the two blue-collar groups were slightly less likely to have an unreported cause of death than those in the white-collar group, but overall it appears that there was generally no substantial difference in cause-of-death reporting across socioeconomic groups.

Individual causes of death were classified by mode of transmission using the scheme proposed by Bengtsson and Lindström (2000). The scheme was developed for another historical Swedish population and was more successful in classifying the causes of death for Stockholm than contemporary classification systems (e.g., ICD-10), especially regarding obsolete medical terminology. Their classification system contains nine categories, which capture deaths from airborne, food, and waterborne and other infectious diseases, as well as noninfectious causes such as cancer, accidents, and diabetes, and unspecified causes. This paper has condensed these from nine to five categories: 1) airborne infectious diseases, 2) food and waterborne infectious diseases, 3) other infectious diseases, 4) noninfectious diseases and accidents, 5) perinatal causes, and 6) unspecified causes. Of those categories with defined causes of death, airborne infectious diseases were by far the largest group (Table 1). This category included common killers like pneumonia, diphtheria, and tuberculosis. The next largest was the food and waterborne disease category, which contained diseases such as cholera, typhoid fever, and diarrhea. Next was the noninfectious disease and accidents group, which included a plethora of conditions and diseases such as cancers, accidents, and chronic digestive disorders. These causes were clustered together due to the fact that they all have unspecified etiology and were relatively uncommon in isolation. A collection of perinatal causes accounted for the second smallest group and often included rather vague descriptions such as “born prematurely.” Nevertheless, for the under-one age group this category of causes contributed to many deaths. Finally, a catch-all group of other infectious diseases was the smallest category of causes. It included infections passed through means other than respiration or ingestion, such as congenital syphilis, which is transferred via the blood from mothers to fetuses, and peritonitis, which was a common result of a burst appendix.



**Table 1: Examples of diseases and conditions present in cause-of-death categories and their distribution for children between ages 0 and 9**

	Percent	Disease/Condition
Airborne infectious disease	38.9	Pneumonia, bronchitis, scarlet fever, diphtheria, tuberculosis
Food and waterborne infectious disease	11.1	Gastroenteritis, cholera, diarrhea, typhoid fever, dysentery
Other infectious disease	2.1	Congenital syphilis, sepsis, erysipelas, peritonitis
Non-infectious disease and accidents	8.4	Accidents, convulsions, rickets, nephritis, cancers
Perinatal causes	4.4	Premature birth, sclerema neonatorum, cleft palate
Unspecified or ambiguous	35.2	

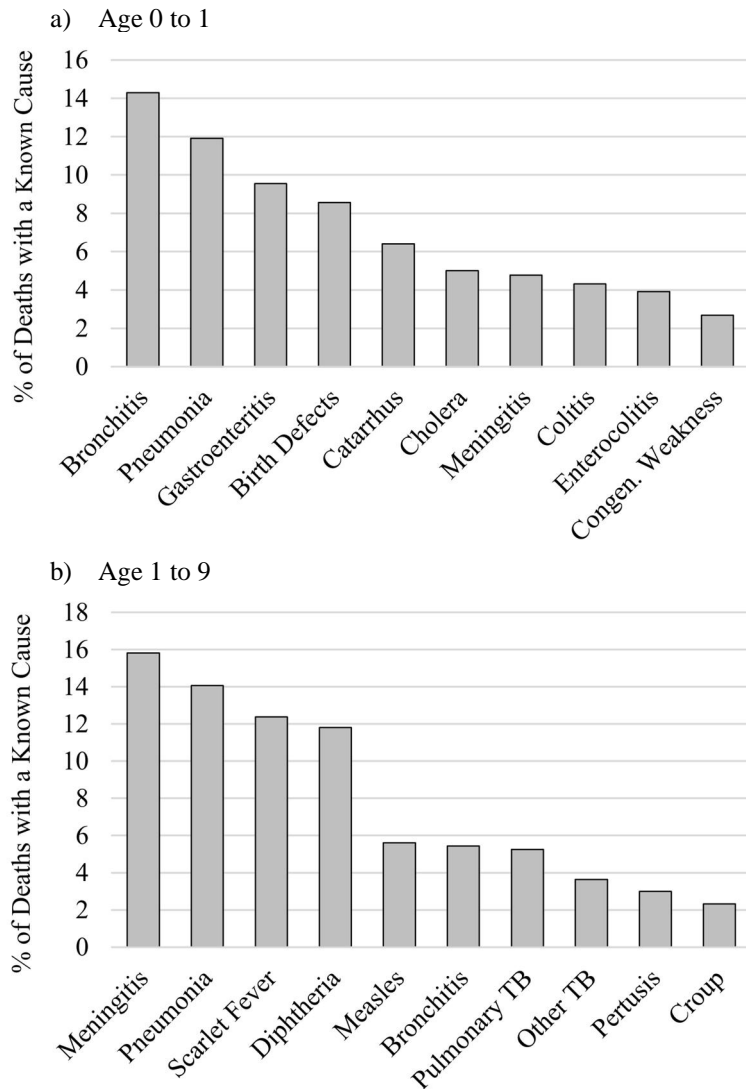
*Notes:* Percent column refers to the percentage of dying individuals age 0 to 9.

*Source:* See Data section.

Although Stockholm's children were exposed to a vast array of deadly diseases during the late 19th and early 20th centuries, only a handful were responsible for the great majority of deaths. Figure 3 shows the ten most common causes of death among Stockholm's infants and children between 1878 and 1926. For infants, these ten causes made up about 71% of all deaths with a known cause, while for children they accounted for almost 80%. Infants tended to die from a wider variety of conditions. Deaths from the airborne illnesses of bronchitis and pneumonia alone accounted for just over a quarter of all infant deaths. Diseases of the digestive system were also common killers for infants of the day, evidenced by the high incidence of gastroenteritis, cholera, colitis, and enterocolitis, which together accounted for 23% of all deaths with a known cause. In addition to these, a non-trivial share of infant deaths was attributable to perinatal causes, like birth defects and congenital weakness, which made up about 11% of all deaths.

Children beyond infancy tended to die from a much more consistent set of causes. Beyond age 1, children were easily most likely to succumb to airborne infectious diseases. In fact, just four causes (meningitis, pneumonia, scarlet fever, and diphtheria) accounted for about 54% of all deaths for children in the age group 1–9. All of the top ten causes of death among this age group were also related to airborne infectious diseases, particularly tuberculosis.

**Figure 3: Ten most common causes of death as a percentage of all deaths with a known cause for infants and children**



Notes: Figures do not include deaths from unknown causes. For infants, the ten most common causes account for 71.4% of all deaths with a known cause (n = 10,515). For children, this figure is 79.3% of all deaths with a known cause (n = 8,547). TB refers to tuberculosis.

Source: See Data section.

#### **4. Patterns and trends in child mortality in Stockholm**

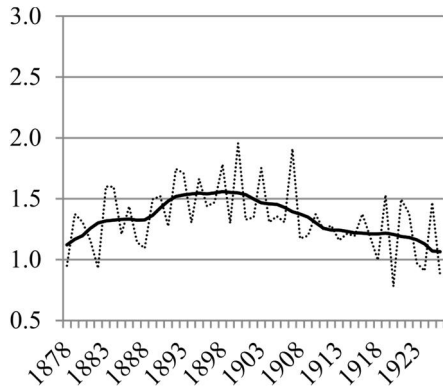
Between 1878 and 1926, the relative difference in age-specific mortality rates between high and low socioeconomic-status groups remained virtually unchanged. Figure 4 shows the development of mortality rates for the age groups 0–1 and 1–9 of the two manual worker groups in relation to those experienced by white-collar workers. At the start of observation, under-1 mortality rates for skilled workers' children were only about 10% higher than for white-collar workers' children (panel a). As mortality declined, this gap widened until around 1900, when the trend was reversed. By the end of the data's coverage, the differences between skilled and white-collar workers had begun to converge, and infant children of skilled workers only had about 6% higher rates. This pattern of divergence and convergence was different from that exhibited by lower-skilled groups (panel b). The mortality rates for children below age 1 of low-skilled and unskilled workers were consistently higher than those of white-collar workers.

Mortality differentials for children between ages 1 and 9 developed somewhat differently. The children of skilled workers consistently had mortality rates between 60% and 80% higher than those of white-collar workers until around the time of the First World War, after which rates between the groups rapidly converged (panel c). The mortality rates of low- and unskilled workers' children, on the other hand, began to show signs of slow convergence towards the rates of white-collar workers, but were still about 50% higher by 1926 (panel d).

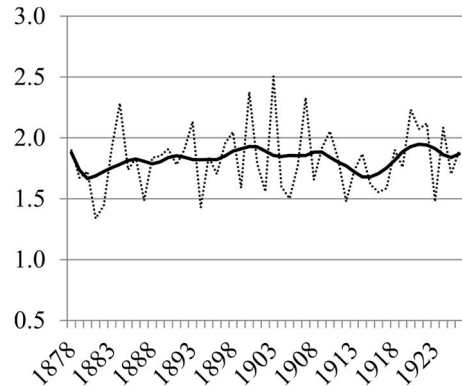
**Figure 4: Age-specific mortality rates relative to white-collar workers by socioeconomic group.**

Age 0 to 1

a) Skilled workers

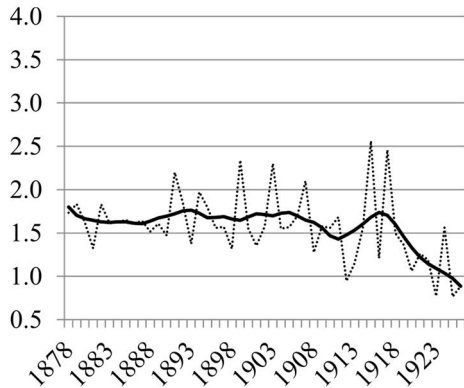


b) Low- and unskilled workers

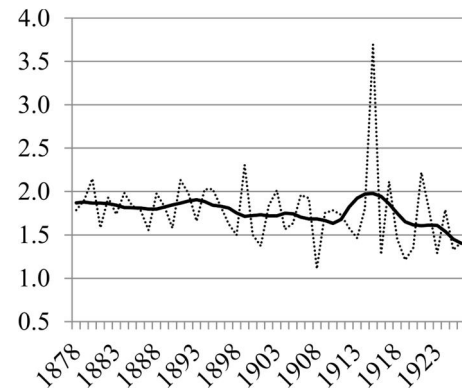


Age 1 to 9

c) Skilled workers



d) Low- and unskilled workers



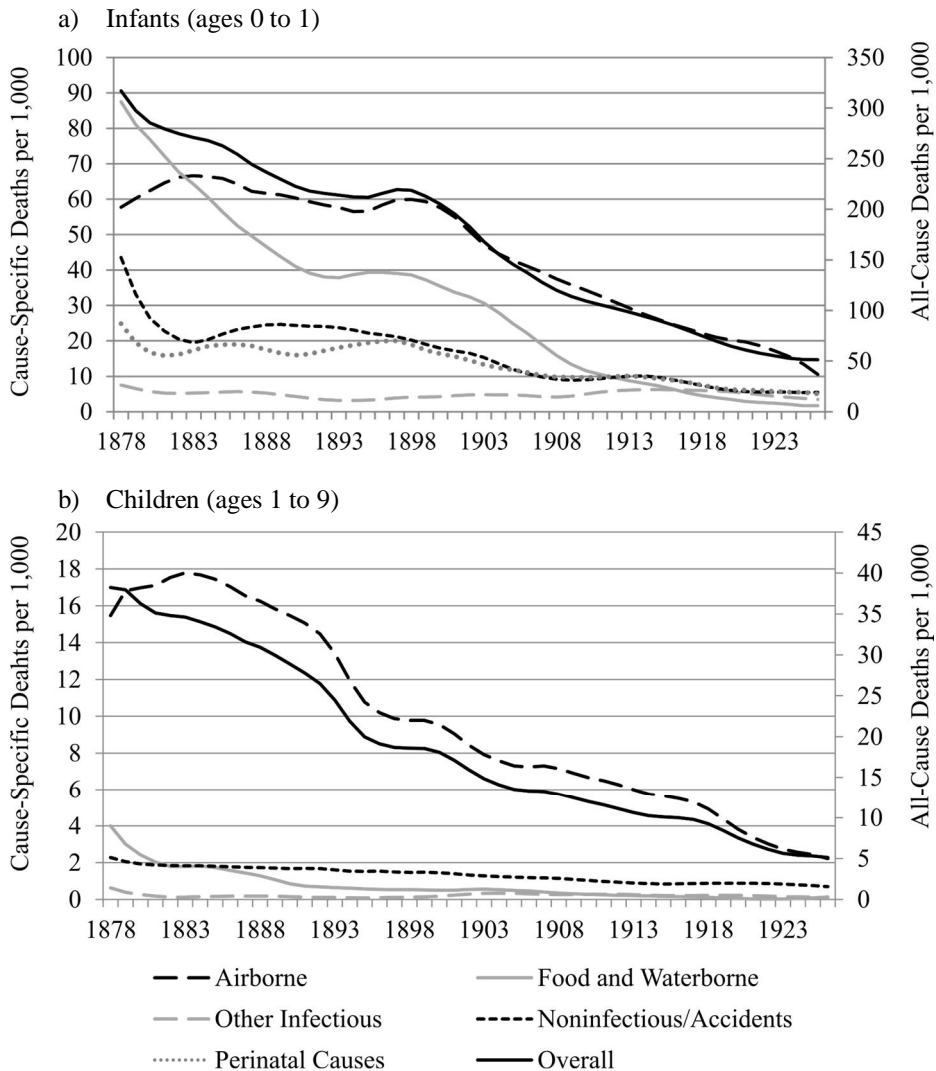
..... Ratio — Lowess-Smoothed Trend

Notes: Figures presented above are the ratios of the corresponding class's infant mortality in each year to that experienced by the white-collar groups. A locally weighted (LOWESS) regression was used to estimate the smoothed trend using a bandwidth of 0.25 and has been superimposed to clarify the long-term developments of the mortality differentials. LOWESS is a non-parametric approach to smoothing data series that can aid in identifying deterministic trends.

Source: See Data section.

For both infants and children, the period of observation witnessed large declines in overall and cause-specific mortality (Figure 5). The age-specific mortality rate among infants ( $m_0$ ) was just over 300 per 1,000 person-years lived (PYL) at the start of the period, about three times higher than the highest infant mortality populations in the modern world. Even for a historical population, the mortality conditions for infants in Stockholm were rather extreme. The importance of certain types of diseases to overall infant mortality varied significantly over time. Until the early 1880s, food and waterborne diseases claimed the highest number of lives, with rates near 90 per 1,000 PYL. But mortality from food and waterborne illnesses decreased rapidly in the late 19th century as clean water became more freely accessible. Mortality from airborne infections, on the other hand, remained stubbornly high, fluctuating around 60 per 1,000 PYL until near the turn of the 20th century, when it began its dramatic descent over the following decades. Death rates from airborne infections would eventually be reduced by a factor of six during the first quarter of the 20th century. Deaths from perinatal causes and noninfectious diseases and accidents were also persistent until around the same time, though at a significantly lower level than those from airborne or food and waterborne diseases. Death rates for infectious diseases with other modes of transmission were low at about 5 per 1,000 PYL, but remained more or less unchanged over the period. Virtually all major infectious diseases saw a short-term resurgence around 1899 during a measles epidemic, but decreased quickly again thereafter. By the end of observation all cause-specific mortality rates except for those related to other infectious diseases had decreased by at least 80%, and the relative importance of each cause changed considerably. Deaths rates from food and waterborne diseases became the lowest of all categories of causes, whereas airborne infectious diseases had become the most common cause of death by a substantial margin. Infants living in 1926 were about two times more likely to die from airborne infection than from either perinatal causes or noninfectious diseases, three times more likely than from other infectious diseases, and six times more likely than from food or waterborne diseases.

**Figure 5: Locally weighted smoothed estimates of infant (age 0 to 1) and child mortality (age 1 to 9) by cause of death**

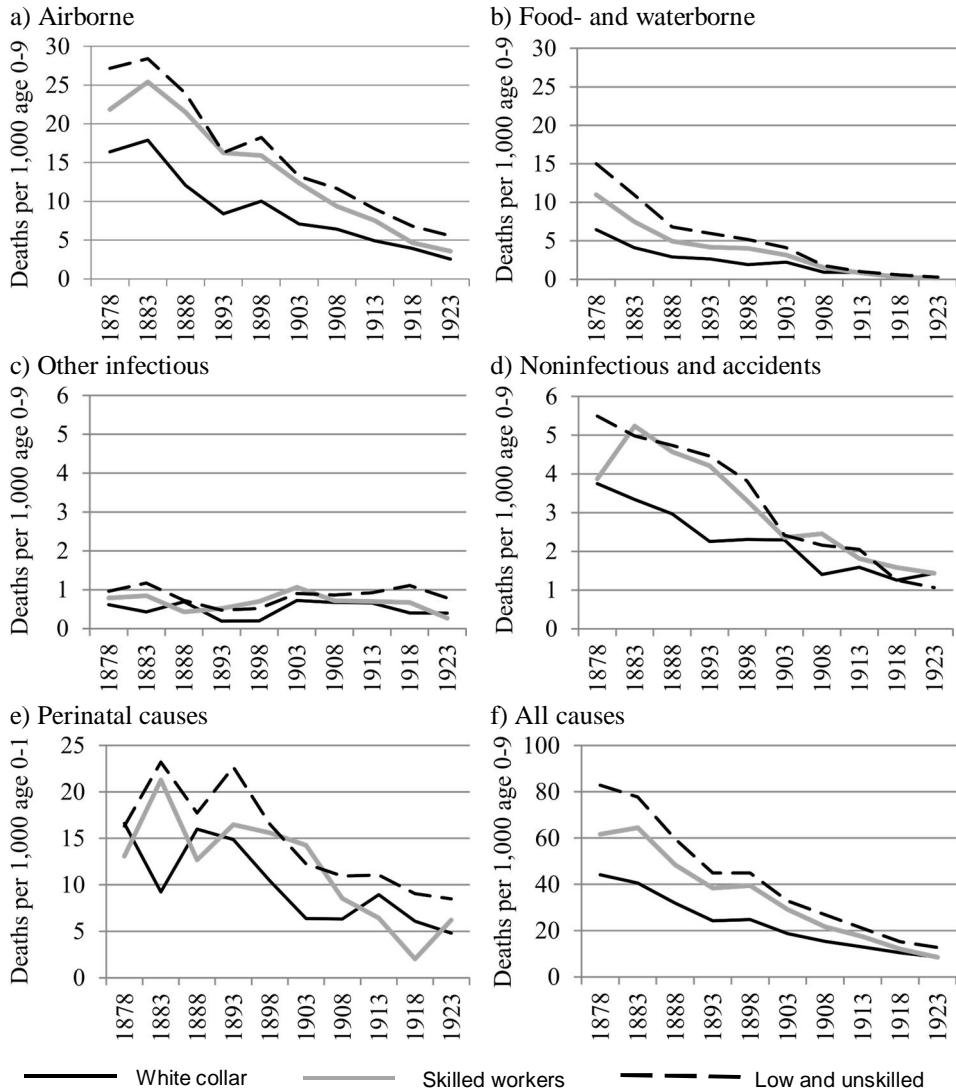


Notes: Estimates were obtained using a locally weighted regression with a smoothing bandwidth of 0.25 for annual data. Rates are calculated per 1,000 person-years. 'Overall' includes all causes of death, including those with unknown causes.  
 Source: See Data section.

As already alluded to above, the distribution of causes of death among children (ages 1 to 9) was much different from that of infants. Airborne diseases were easily the most dominant cause of death during the entire period, with rates between seven and eight times larger than any other group of causes. Deaths from all other causes were under 2 deaths per 1,000 PYL for nearly the entirety of the period. From this figure it is clear that the decline in child mortality was predominantly due to decreasing airborne disease mortality rates, while for infants the mortality decline was characterized by sequential decreases in different groups of causes. Early on, decreases in food and waterborne mortality and deaths from noninfectious diseases were largely responsible for improvements in infant survival, while later in the decline decreasing mortality from airborne diseases and perinatal causes became relatively more important.

All socioeconomic groups experienced declines in the mortality rates of most categories of causes, but there were differences in terms of how socioeconomic inequality in cause-specific mortality developed. Figure 6 presents cause-specific mortality rates for ages 0–9, except for perinatal causes, which reflects ages 0–1, as no children beyond age 1 died from this cause. The children of white-collar workers had lower mortality from all causes and the difference was most apparent for airborne diseases. As overall mortality declined, some causes of death appear to have converged across socioeconomic groups, such as those from food and waterborne disease and noninfectious diseases and accidents. On the other hand, class differences in airborne disease mortality and perinatal causes continued to exist until the end of observation in 1926, suggesting that these causes may have become relatively more important than other causes in perpetuating socioeconomic differentials in all-cause infant mortality, as shown in Figure 4 (panel b). But the rates presented so far have not taken into account many of the other characteristics that may be important in explaining mortality differentials and which may be correlated with socioeconomic status. It is possible that socioeconomic differences in cause-specific mortality were not due to class per se but rather to compositional differences between groups. For example, it may be that there were proportionally more children born out of wedlock among the lower classes over time, which would serve to inflate socioeconomic differentials in mortality. In order to account for compositional differences between groups, I will now use event-history models to examine mortality differentials.

**Figure 6: Cause-specific death rates per 1,000 person-years at ages 0–9 by socioeconomic status**



Notes: Panel e) refers to only age 0–1. Panel f) includes all deaths, including those without a specified cause.  
 Source: See Data section.



## 5. Multivariate methods

Before implementing an event history analysis it is necessary to accurately identify the population at risk. For this study, individuals who were present at any time in Stockholm between ages 0 and 9 were included in the analysis and censored upon reaching their 10th birthday. This age grouping was taken to avoid problems with small numbers that may arise when including interaction terms in the models. Because causes of death have not been digitized for all districts in Stockholm, only individuals in those districts with recorded causes (i.e., Gamla Stan, Södermalm-East, Södermalm-West) will be considered at risk. As a result, individuals may become at risk only if they were living in any of the included districts between the specified ages. A child would be right censored if he/she moved out of one of these districts before reaching age 10, even if they still resided in Stockholm. However, the child would not be censored if he/she moved from one district with cause-of-death information to another with that information, as district of residence is treated as a time-varying covariate. Individuals could also be left-truncated if they were born outside of the analysis districts and moved into one of them before reaching age 10. Ultimately, the exposure of interest is therefore the number of person-years lived until age 10 in any of Stockholm's districts with digitized causes of death.

To analyze how socioeconomic inequalities in cause-specific mortality evolved during industrialization, this study adopts a competing risks framework. Standard semi-parametric event history models are a useful way of analyzing covariate effects on mortality risks within a population for which longitudinal information is available. Furthermore, they can easily deal with individuals being censored prior to death. One of the crucial assumptions of these models, however, is that censoring does not influence failure times. For example, assume that we were interested solely in the risk of all-cause child mortality between 1878 and 1926 and that all children could be fully observed until either death or the end of the observation period. In this case, any censoring that occurred due to the termination of the study period should have no bearing on a child's risk of dying. That is, no child's risk of dying would be altered purely because data were no longer collected. On the other hand, in the study of cause-specific mortality the existence of multiple possible causes of death can violate this assumption. If the interest is in, say, the cause-specific hazard of dying from tuberculosis, but some individuals are censored because they die from cholera, then obviously censoring will not occur independently of failure times, as dying from one cause will make it impossible to die from another. The existence of competing risks therefore will change the standard interpretation of the coefficients in a semi-parametric model. As such, the present analysis will estimate the following cause-specific hazards of dying using Cox proportional hazards models:

$$h_i^J(t) = h_0^J(t)\exp(\beta X_i) \quad (1)$$

where  $h_i^J(t)$  is an individual's instantaneous risk of dying from cause  $J$  at time  $t$ . This is determined by the product of the baseline hazard of dying from cause  $J$  ( $h_0^J$ ) and a vector of covariates,  $\exp(\beta X_i)$ . To estimate this model, separate Cox regressions were fitted for each cause of death. This means that six models were estimated with the binary dependent variables indicating deaths from: 1) airborne disease, 2) food and waterborne disease, 3) other infectious disease, 4) noninfectious diseases or accidents, 5) perinatal causes, and 6) unknown causes. By using Cox models in a competing risks framework, deaths from causes other than the cause of interest for each regression are treated as a form of censoring. This differs from the commonly used subdistribution hazard model offered by Fine and Gray (1999), which allows individuals dying from competing causes to remain in the risk population (they would remain at risk infinitely for all causes but that from which they died). Both are valid approaches, but have different interpretations. Coefficients in the subdistribution hazard model would be interpreted as the effects of covariates on the cumulative probability of a specific cause of death. On the other hand, in the cause-specific hazard model the coefficients may be interpreted as the influence of the covariates on the instantaneous risk of dying from cause  $J$  among those still living and present in the risk population at time  $t$ . It has repeatedly been argued that the cause-specific hazard model is more appropriate for studying the etiology of diseases (Lau, Cole, and Gange 2009; Austin, Lee, and Fine 2016), and because this study is specifically interested in the role of socioeconomic status as a fundamental cause of disease, it seems most appropriate to adopt the cause-specific hazards approach for the analysis.

The models will be used to investigate how socioeconomic status influenced the risk of dying from each cause and how differences between groups developed over time. The main independent variable is socioeconomic status as defined in section 3. In addition, the models control for temporal variation in mortality by including year and season dummies and personal characteristics such as birth order, sex, and whether or not the child was born within wedlock. Furthermore, they will control for indicators for some of the proximate determinants. These include the number of other individuals in the household and district of residence within Stockholm, which capture elements of environmental contamination and crowding, and mothers' age at birth and length of the previous birth interval, which captures the influence of maternal factors. The number of individuals in the household and district of residence are both time-varying covariates. It must be made clear, however, that controlling for the district of residence will only partially account for differences in environmental conditions, as it will also account for differences in digitization between districts. As such, the coefficients estimated for the district dummies should be interpreted cautiously. Unfortunately, the model has no way

of measuring the other two proximate determinants, nutrient deficiency and personal illness control. To examine how the association between class and causes of death evolved, the models were then extended by including interactions between social class and period dummies. The proportionality assumptions of all models were tested using the method offered by Grambsch and Therneau (1994), which utilizes Schoenfeld residuals. In all models, the main predictor variable, socioeconomic status, showed no signs of nonproportionality. Summary statistics of the covariates may be found in Table 2.

**Table 2: Distribution of covariates used in Cox proportional hazards models**

Variable	Share of exposure	Person years at risk	Deaths	Variable	Share of exposure	Excluding First-borns	Person years at risk	Deaths
Socioeconomic status:				Time since last birth:				
White collar	20.9	235,737	3,325	<18	13.1	23.4	147,397	4,480
Skilled workers	26.7	301,725	6,525	18–23	10.2	18.2	114,540	3,100
Low- and unskilled	46.0	519,022	13,764	24–29	9.3	16.6	104,836	2,691
Unknown	6.4	71,741	1,802	30–35	5.7	10.2	64,544	1,667
Birth order	2.4			36–41	3.6	6.4	40,272	1,072
Female	50.3	567,230	11,858	42–47	2.6	4.6	29,042	733
Born out of wedlock	10.0	112,683	6,508	48–53	1.9	3.4	21,534	534
People in household:				54–59	1.5	2.8	17,409	423
1 to 3	14.4	161,908	5,735	60+	7.1	12.7	80,004	1,901
4 to 6	45.0	507,559	10,473	Unknown/na	45.1	1.8	508,646	8,815
7 to 9	25.6	289,177	5,888	Period:				
10+	15.0	169,581	3,320	1878–1882	13.4		151,556	3,339

**Table 2: (Continued)**

Variable	Share of exposure	Person years at risk	Deaths	Variable	Share of exposure	Excluding First-borns	Person years at risk	Deaths
Mother's age at birth:				1883–1887	9.4		105,496	4,117
<20	2.9	33,151	712	1888–1892	10.2		114,791	3,751
20–24	19.3	218,285	4,736	1893–1897	10.7		120,995	3,250
25–29	28.6	322,636	6,449	1898–1902	10.3		115,979	3,302
30–34	24.0	270,627	5,826	1903–1907	10.4		117,306	2,375
35–39	15.7	176,902	4,212	1908–1912	11.3		127,061	2,094
40–44	6.5	72,912	1,988	1913–1917	10.9		122,996	1,677
45–49	1.3	14,574	442	1918–1922	8.8		98,804	1,055
50+	1.0	11,753	401	1923–1926	4.7		53,241	456
Unknown	0.7	7,386	650					
District:				Season:				
Gamla Stan	9.4	106,222	2,354	Spring	20.3		229,299	6,342
Södermalm-East	50.3	567,734	13,592	Summer	24.2		272,948	6,943
Södermalm-West	40.3	454,267	9,470	Autumn	24.8		280,216	5,745
				Winter	30.6		345,762	6,386

Notes: Figures for categorical variables refer to the share of total exposure accounted for by the respective category. For birth order, the mean value is presented. 'Time since Last Birth' only applies to non-first-born children. All first-borns were assigned an interval of 'unknown/NA.' The distribution excluding first-borns is presented to illustrate that very few were missing information on the length of the preceding birth interval.

Source: See Data section.

## 6. Event history results

Table 3 shows the results of the Cox models with no interactions. Before discussing the results, it should be noted for these and all of the following models that, theoretically, clustering of deaths within families could lead to an underestimation of the standard errors. In practice, however, there is so little cause-specific clustering occurring that there were virtually no differences in the models' significance tests when clustering standard errors at the family level. It is clear from Table 3 that a socioeconomic gradient existed for mortality from almost all causes of death in favor of the children of white-collar workers, especially compared to the children of low- and unskilled blue-

collar workers. The differences between groups were the largest for the airborne disease and food and waterborne disease categories. For airborne disease mortality, the children of skilled blue-collar workers were 42% more likely to die than those of white-collar workers, and the gap was even larger among the low- and unskilled blue-collar workers, whose children had a 61% higher risk of dying. The socioeconomic differences regarding mortality from food and waterborne diseases were of a similar magnitude. For most other causes of death the differences between groups were smaller, but still significant in both statistical and real terms. For example, the cause-specific hazards for other infectious diseases were only 26% and 35% higher for the skilled and unskilled groups, respectively, although the estimates for the skilled-workers group was not statistically different from the white-collar group at any conventional significance level. The risk of dying from noninfectious diseases was 25% higher for skilled workers' children and 28% higher for low- and unskilled workers' children compared to the white-collar group. There were also differences in dying from perinatal causes, but they were only statistically significant for the children of low- and unskilled workers, who had a 29% higher risk of dying from these causes. That a difference even existed is surprising, as the period of study is one in which prenatal care was virtually absent. It may well be, however, that the existence of a socioeconomic gradient with respect to perinatal causes may reflect differences in maternal nourishment and health.

**Table 3: Cause-specific hazard ratios from Cox proportional hazards models**

	Airborne	Food- and water-borne	Other infectious	Non-infectious	Perinatal causes	Unknown
Socioeconomic status:						
White-collar	(ref)	(ref)	(ref)	(ref)	(ref)	(ref)
Skilled workers	1.42***	1.37***	1.26	1.25***	1.13	1.23***
Low- and unskilled	1.61***	1.60***	1.35***	1.28***	1.29***	1.32***
Unknown	1.06	1.10	1.11	0.83*	1.13	1.26***
Birth order	1.13***	1.15***	1.14***	1.11	1.15***	1.07***
Female	0.94***	0.94*	1.14	0.76***	0.80***	0.84***
Born out of wedlock	1.93***	2.83***	2.73***	2.12***	2.82***	3.35***
People in household:						
1 to 3	(ref)	(ref)	(ref)	(ref)	(ref)	(ref)
4 to 6	0.89***	0.67***	0.64***	0.78***	0.78***	0.75***
7 to 9	0.82***	0.55***	0.49***	0.76***	0.74***	0.70***
10+	0.76***	0.50***	0.64***	0.71***	0.54***	0.67***

**Table 3: (Continued)**

	Airborne	Food- and water-borne	Other infectious	Non-infectious	Perinatal causes	Unknown
Mother's age at birth:						
<20	1.13*	0.86	0.63*	0.85	1.27	0.93
20–24	(ref)	(ref)	(ref)	(ref)	(ref)	(ref)
25–29	0.94*	0.89**	0.84	0.91	0.91	0.88***
30–34	1.02	0.92	0.82	0.94	0.86	0.82***
35–39	0.99	0.91	0.77	0.94	1.16	0.87***
40–44	1.07	0.96	0.82	0.97	1.25*	0.93
45–49	1.04	1.25	0.59	0.75	0.79	1.00
50+	0.98	1.55***	0.42**	0.97	0.62	1.06
Unknown	3.36	3.12***	2.81***	3.54***	3.55***	2.42***
Time since last birth (in months):						
<18	0.83***	1.32***	1.00	1.10	2.01***	1.08
18–23	1.06	1.16	1.00	1.08	1.08	1.05
24–29	1.01	0.93	1.02	0.95	1.03	0.99
30–35	(ref)	(ref)	(ref)	(ref)	(ref)	(ref)
36–41	0.95	0.98	0.98	1.12	1.19	1.20**
42–47	0.96	0.90	1.12	0.94	1.09	1.10
48–53	0.85	1.10	1.16	0.95	1.22	1.02
54–59	0.88	0.91	1.13	0.75	1.09	1.12
60+	0.75***	0.96	1.30	0.89	0.99	1.15*
Unknown/na	0.59***	0.70	0.81	0.81	1.15	0.92
District:						
Gamla stan	(ref)	(ref)	(ref)	(ref)	(ref)	(ref)
Södermalm-east	0.72***	1.39***	0.94	0.68***	0.72***	1.40***
Södermalm-west	0.98	1.33***	1.20	1.13	0.85	0.68***
Failures	9,883	2,811	538	2,132	1,116	8,936
Individuals	193,833	193,833	193,833	193,833	193,833	193,833
Person-years	1,128,224	1,128,224	1,128,224	1,128,224	1,128,224	1,128,224
Chi2	4,452.9	3,156.3	296.8	954.4	650.6	5,942.9

\* Denotes significance at the 10% level, \*\* Denotes significance at the 5% level, \*\*\*Denotes significance at the 1% level

Notes: A cause-specific hazard ratio of 1.5 indicates that the instantaneous risk of dying from the selected cause is about 50% higher than the reference category. For full model specification and inclusion criteria, see Methods section. Estimates of period and season hazards have been omitted from the output for brevity.

Source: See Data and Methods sections.

It is also worth briefly mentioning some of the findings from the control variables, as there were both remarkable consistencies and surprising patterns across causes of death. The cause-specific hazards for birth order consistently showed that higher-order births had a heightened risk of dying from all causes. An increase in birth order by one was associated with an increase in the risk of dying from nearly all causes by about 13%–15%, suggesting that later-born children were at greater risk of all causes of death. Female children were less likely to die from all causes except other infectious disease,

for which there was no statistically significant difference between sexes. The difference was most obvious when it came to perinatal causes and noninfectious disease. Girls had a 20% and 24% lower risk of dying from these causes than boys, respectively. Children born out of wedlock had substantially higher risks of dying from all causes relative to those born within marriage, and this association was present independent of socioeconomic and observable maternal characteristics. These children had cause-specific risks of dying that were two to three times as high as those born to married parents. Interestingly, maternal characteristics were very weakly related to most specific causes of death. Children born to mothers who were above age 40 at birth and those born following intervals of less than 18 months had a significantly higher risk of dying from food and waterborne diseases, perhaps due to reduced capacity for breastfeeding. Furthermore, children born following very short intervals also had a much higher risk of dying from perinatal causes, which may be a result of these births being premature (Miller 1989). Otherwise, there were no consistent effects of maternal age or birth spacing on cause-specific hazards.

As for factors related to environmental exposure, there was a strong negative effect of the number of people in the household on all kinds of mortality. This is a surprising finding, as one would suspect that this would have especially facilitated the transmission of infectious disease. It may be, however, that this variable does not capture crowding as much as it captures wealth effects, as wealthier families tended to have larger households via live-in servants. Furthermore, there is no possibility to control for the size of the dwelling, which is equally important to understand the effects of crowding. Some work using the Stockholm Housing Censuses to adjust for the number of inhabitants per m<sup>2</sup> has found that the effect of crowding on child mortality was particularly severe among lower socioeconomic groups (Bernhardt 1995). Unfortunately, it is not possible to make this adjustment with the current data. There were also large differences in the risk of dying between districts of the city, though one should not put too much emphasis on the geographic differences as these capture variability in the digitization process as much as they capture differences in the disease environment.

To explore how socioeconomic inequalities in cause-specific mortality evolved over time, an interaction term between socioeconomic status and period was included in the model (Figure 7). It is clear that the magnitude of mortality differentials varied substantially for different causes of death. Children of white-collar workers were clearly the least likely to die from airborne diseases and this advantage was largely maintained throughout the period of observation. Over time, the risk of skilled workers' children dying showed signs of divergence and eventually slow convergence with those of the reference group, but differences remained statistically significant even at the end of observation. By the period 1923–1926 the children of skilled workers had a 64% higher

chance of dying from this category of diseases than those of white-collar workers. At the start of observation the difference in risks was only 22%. The gap between low-skilled and unskilled workers' children and the reference group continuously widened between 1878 and 1926. In the first decade of observation, children from the lowest class were 44% more likely to die from airborne illness than children from the highest class. By 1926 they were more than twice as likely to die from these diseases, and this pattern was quite different from the development of inequality in other causes of death.

Mortality differentials from food and waterborne illness declined in the long run, but only after diverging for several decades. Between 1878 and 1882 the skilled and low- and unskilled blue-collar workers were about 60% and 75% more likely to die from these diseases, respectively. But the gap between the white-collar workers and the other groups widened in the following two decades, reaching its greatest difference at the turn of the 20th century during a period of deadly epidemics. The risk of dying from food and waterborne disease had converged by the second decade of 20th century, first for the children of skilled workers and later for those of low- and unskilled workers. There is some indication that mortality differentials from this group of causes widened again after the First World War, but these estimates were statistically insignificant and based on relatively few cases. These results are consistent with the findings of Burström et al. (2005), who also documented a widening and subsequent closing of diarrheal mortality differentials between socioeconomic groups in Stockholm in the same period. It is also suggestive of class differences in obtaining access to piped water, in which wealthier households were earlier users of sanitation infrastructure than poorer households, likely due to the high costs of connecting buildings to water mains in the earlier years of the water network.

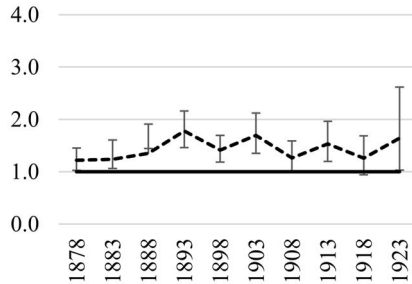
Differences in mortality caused by infectious diseases of other modes of transmission were generally null. Apart from a period of brief divergence for the children of low- and unskilled workers between 1918–1923, there was no statistical difference in the cause-specific hazards of any group compared to those of white-collar workers' children.



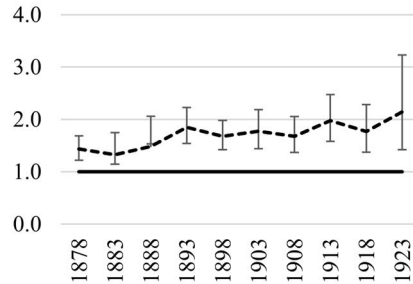
**Figure 7: Socioeconomic differences in cause-specific mortality, 1878–1926**

1) Airborne

a) Skilled workers

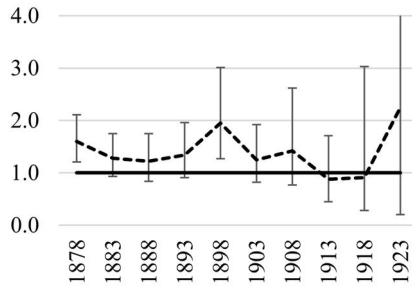


b) Low- and unskilled workers

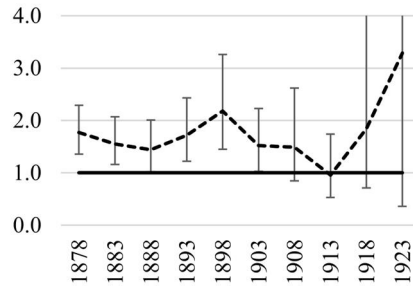


2) Food- and waterborne

a) Skilled workers

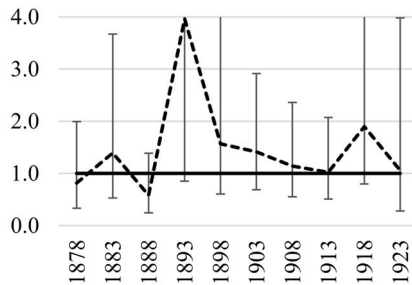


b) Low- and unskilled workers

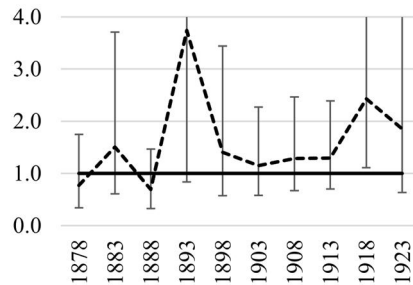


3) Other infectious

a) Skilled workers



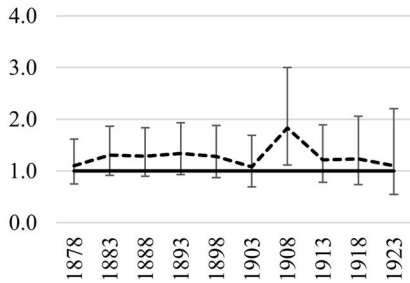
b) Low- and unskilled workers



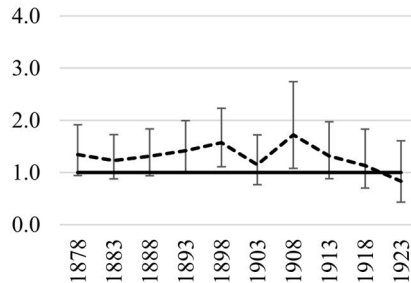
**Figure 7: (Continued)**

4) Noninfectious and accidents

a) Skilled workers

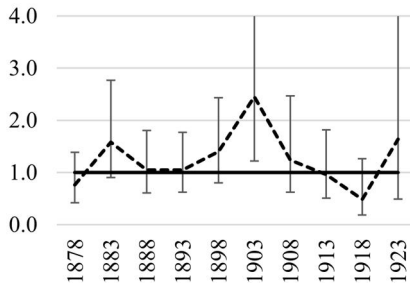


b) Low- and unskilled workers

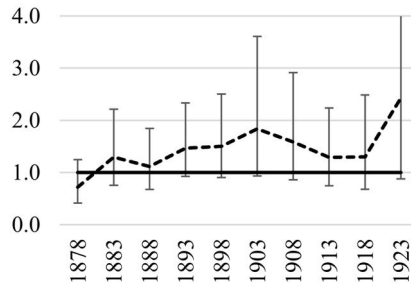


5) Perinatal causes

a) Skilled workers

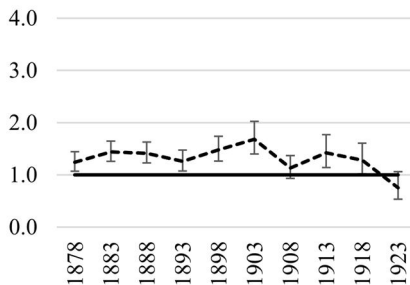


b) Low- and unskilled workers

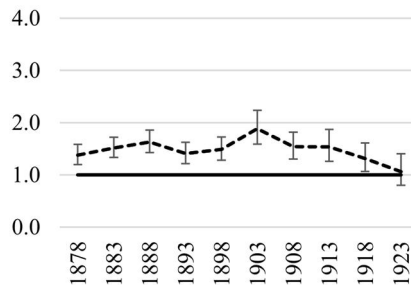


6) Unknown causes

a) Skilled workers



b) Low- and unskilled workers



Notes: Figures presented as hazard ratios. The solid line represents the cause-specific hazard of children of white-collar workers. 95% confidence intervals are reported. All other causes of death are calculated for ages 0 to 9.

Source: See Data and Methods sections.

Inequality in mortality from noninfectious diseases also disappeared during the study period. There were few statistically significant differences in the risk of dying from this group of causes for the children of skilled workers, though the point estimates indicated an elevated risk of dying for this group. Nevertheless, differences between groups converged by the end of the period. As for the children of low-skilled and unskilled workers, most estimates prior to 1908–1912 were statistically significant at least at the 10% level and indicated a 20%–70% higher risk of dying than that experienced by white-collar workers' children. In the following decades, however, these risks also converged across groups in both statistical and real terms. The convergence observed here was largely due to declining mortality from accidents, seizures, rickets, and nephritis.

Finally, class differences in mortality from perinatal causes were generally non-existent. The only exception to this was in the period 1903–1907, which saw particularly deadly outbreaks of many infectious diseases, including measles, scarlet fever, pertussis, diphtheria, and tuberculosis, and which may have indirectly affected the incidence of perinatal causes via changes in maternal health. However, apart from short-term differences, inequalities in death from congenital causes were largely absent. The point estimates for low-skilled and unskilled workers' children seem to be trending towards greater inequality towards the end of the period, although the estimates remained statistically insignificant at any conventional level.

## 7. Discussion

This paper has demonstrated how socioeconomic cause-specific mortality differentials evolved during Stockholm's mortality transition, and how these differences contributed to inequality in all-cause child mortality. Prior to 1900, class differences in all-cause mortality were due to a combination of differential mortality rates from airborne, food and waterborne, and, to a lesser degree, noninfectious diseases. However, from the end of the first decade of the 20th century, socioeconomic mortality differentials were almost entirely driven by differences in airborne disease mortality, apart from episodic divergences in mortality risks from other causes. For all other causes of death, mortality inequality had either disappeared by this point or, in the case of perinatal causes, had not been present to begin with.

The patterns identified in this study reveal a complex picture of the development of living standards during a Western mortality transition. This study provides evidence that mortality inequality from food and waterborne infectious diseases declined during the period, and this was likely due to a combination of clean water interventions, the intensification of food inspections, and the sanitation revolution in general during the

late 19th century. At the same time the results show that other causes of death became even more unequally distributed. In the case of Stockholm, it was the much more common and lethal airborne infectious diseases such as diphtheria, measles, pneumonia and tuberculosis that filled this niche. Hazards of other causes for which etiology was unclear, such as those associated with premature births and congenital conditions, were statistically indistinct across socioeconomic groups over time. All of these findings are consistent with the theory of fundamental causes (Link and Phelan 1995): Conditions which were poorly understood were not stratified by socioeconomic status, while those for which personal resources became less important for avoiding exposure to proximate determinants became less so over time.

However, the question remains of in what ways the greater resources of higher-status individuals reduce their children's exposure to airborne diseases and allow them to maintain a health advantage. Unfortunately, the analyses in this study were only able to account for some of the proximate determinants of mortality and therefore cannot identify precisely the influence of various health inputs. However, based on the discussion in Section 2 and the findings from the event history models, some broad conclusions can be made to guide future research in answering this question.

Even after including controls for some of the proximate determinants (i.e., maternal factors, environmental exposure), inequality in cause-specific hazards persisted. Admittedly, this study did not have the ability to control for all of the necessary elements in a satisfactory way and, although this was not the study's intention, it is an area for future research to address. For example, a much better individual-level measure of environmental exposure in the present work would have been inhabitants/m<sup>2</sup>, in order to adequately capture residential crowding. Unfortunately, there was no housing information to make such a calculation. It would be interesting to see if a socioeconomic gradient in cause-specific mortality could still be identified if measures of all of the proximate determinants could be adequately incorporated in addition to the socioeconomic characteristics. Based on the earlier discussion of the proximate determinants, it is doubtful that medical interventions played a substantial role during this period. Nevertheless, this study could not adequately account for this factor and it may have been a source of inequality, especially towards the end of the period of observation. On the other hand, there is stronger evidence that nutritional status was becoming less equal during this period. As was shown earlier in Figure 2, the gap in protein consumption between low- and middle-income families grew significantly during the period, in spite of the fact that absolute consumption increased. It would also be worthwhile to study how the development of nutritional inequality developed during the period, and its role in perpetuating differences in mortality from specific diseases.

Ultimately, the results of this paper show how all-cause mortality differentials can persist despite dramatic improvements in epidemiological conditions. As shown in Figure 4, there were virtually no changes in the mortality gap between white-collar and low- and unskilled blue-collar workers' children over this period, yet we saw that differences in most causes of death actually disappeared. From a development perspective the case of Stockholm's mortality transition is instructive, as it highlights that in order to reduce overall inequalities in health it is necessary to eliminate barriers to good health from a variety of perspectives. In spite of some of the greatest achievements in the history of public health, such as the distribution of clean water and disposal of sewage, growing inequality in other conditions emerged as those from food and waterborne illness disappeared. Whether or not these findings generalize to other industrial cities of the era remains to be seen.

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## Appendix

**Table A1: Record digitization status, cause of death registration status, and share of deaths under age 10 with a recorded cause for the districts of the Roteman System**

Time-invariant district	Districts	Digitized	Causes of death digitized	Included in final analysis	Share of deaths with recorded causes	Share of deaths with recorded causes in aggregated districts
Bromma	Brommaroten					0
Brännkyrka	Enskederoten	X				0
	Liljeholmsrotten	X				
	Årstaroten	X				
Gamla Stan	Storkyrkoroten	X	X	X	70.8	70.8
Kungsholmen	Kungsbroroten	X	X		19.1	18.7
	Kronobergsrotten	X	X		16.8	
	Karlsvikrotten	X	X		23.3	
	Kristinebergsrotten	X	X		15.5	
Normalm	Klararoten 2	X	X		0.1	0.1
	Klararoten 3	X	X		0.1	
Södermalm-East	Sofiaroten	X	X	X	60.5	60.2
	Stadsgårdsrotten	X	X	X	56.1	
	Katarina mellanrotten	X	X	X	61.3	
	Nytorgsrotten	X	X	X	64.4	
	Helgaroten	X	X	X	61.9	
Södermalm-West	Maria kyrkorote	X	X	X	79.2	79.9
	Tantoroten	X	X	X	78.7	
	Skinnarviksrotten	X	X	X	82.9	
	Heleneborgsrotten	X	X	X	80.7	
Vasastan	Tegnérrotsrotten					0
	Observatorieroten					
	Teknologrotten					
	Vasaroten					
	Norrtullsrotten					
	Karlbergsrotten					
	Birkaroten					

**Table A1: (Continued)**

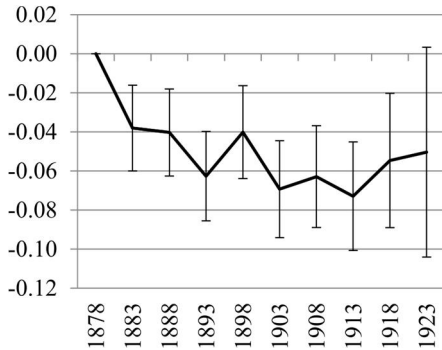
Time-invariant district	Districts	Digitized	Causes of death digitized	Included in final analysis	Share of deaths with recorded causes	Share of deaths with recorded causes in aggregated districts
Östermalm	Jakobsroten	X				0
	Roslagsroten					
	Humlegårdsroten	X				
	Nybroroten	X				
	Artilleriroten	X				
	Narvaroten	X				
	Vanadisroten					
	Karlaroten	X				
	Eriksbergsroten	X				

*Note:* The 36 districts of the Roteman System may be found under 'District Name'. Not all of these existed for the entirety of the Roteman System (1878-1926), as some were the result of the city's geographical expansion while others emerged out of subdivisions of existing districts as the city became more densely populated. As a result, they have been condensed into time-invariant districts in the analysis. As can be seen, only a portion of these (26 out of 36) have had their records digitized. A even smaller share has had any cause-of-death information registered (16 out of 26) and because of the large shares of missing causes in 6 of those districts, only 10 were included in the final analysis. These were from the time-invariant districts of Gamla Stan, Södermalm-East, and Södermalm-West.

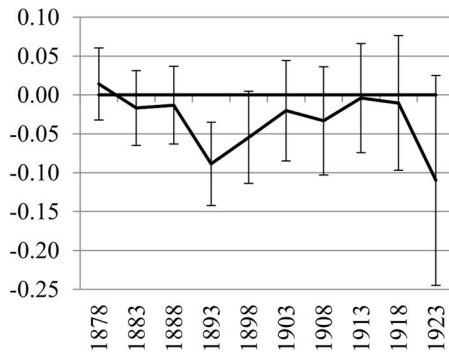
*Source:* See Data section.

**Figure A-1: Comparison of predicted probabilities of having an unknown cause of death by socioeconomic status and period**

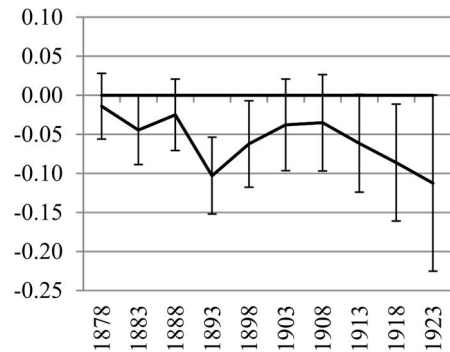
a) All groups relative to 1878–1882



b) Skilled workers

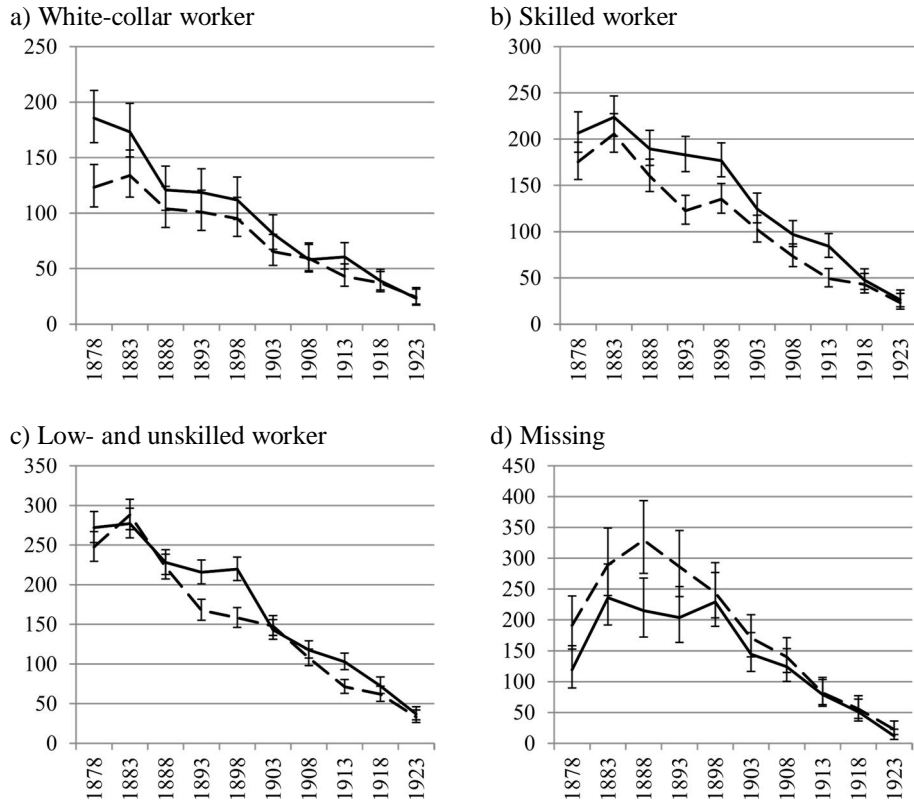


c) Low- and unskilled workers



Notes: Predicted probabilities were generated from a logistic regression. Error bars represent 95% confidence intervals; if they overlap with zero it suggests that there was no statistically significant difference in the probability of having a missing cause of death compared to white-collar workers in the respective period. The estimation sample included all deaths occurring between ages 0 and 9 during the study period 1878–1926. The dependent variable was an indicator variable equal to 1 if a death between ages 0 and 9 had no cause attributed to it and 0 otherwise. The independent variables were socioeconomic status, period of death, district of death, number of people living in the household, length of previous birth interval, month of death, illegitimacy status, age of child at death, and mother's age at birth. In addition, the models controlled for an interaction between socioeconomic status at death and period of death.

**Figure A2: Age-specific mortality rates ( $_{10}m_0$ ) of unrecorded and recorded causes of death by socioeconomic status and period**



Notes: Solid line refers to age-specific mortality rates from recorded causes. Dashed line refers to rates from unrecorded causes. 95% confidence intervals are represented by error bars.



