What was Killing Babies in Sundsvall? A Study of Infant Mortality Patterns Using Individual Level Cause of Death Data, 1860–1892

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HISTORICAL LIFE COURSE STUDIES

What was Killing Babies?
European Comparative Research on Infant Mortality
Using Individual Level Causes of Death

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What was Killing Babies in Sundsvall?

A Study of Infant Mortality Patterns Using Individual Level Cause of Death Data, 1860–1892

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ABSTRACT

In this article, we analyse infant mortality in Sundsvall 1860–1892. The focus is on the pattern and development of causes of death with separate analyses of neonatal and post-neonatal mortality. Furthermore, we discuss the development of infant mortality in relation to possible determinants in the historical context in Sundsvall. The results show substantial differences between neonatal and post-neonatal mortality when it comes to causes of death as well as their seasonal pattern. For deaths in the first 28 days, a large proportion of the deaths were diagnosed as unknown disease or given vague and symptom-descriptive diagnoses. For post-neonatal mortality on the other hand, the dominant cause of death categories were water- and food-borne infections and air-borne infections. Water- and food-borne diseases had a very strong seasonal pattern with the peak in late summer — July and August. There is no indication that sanitary improvements in the 1880's led to fewer cases of diarrhoea. Mortality from air-borne diseases on the other hand was lowest during summer, instead peaking in the winter months.

Keywords: Individual level cause of death data, Infant mortality patterns, Historical causes of death, 19th Century Sweden, ICD10H

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1 INTRODUCTION

In this article, we analyse infant mortality in Sundsvall 1860–1892, a small port city along the Baltic Sea coast in Northern Sweden, which experienced rapid population growth as it became the centre in one of the largest sawmill districts in Europe. Sundsvall represents a somewhat different context than the other port cities in this special issue (Garrett & Reid, 2022; Janssens & Riswick, 2023; Ludvigsen et al., 2023; Mühlichen & Cilek, 2024; Pujadas-Mora, 2024; Raftakis, 2022; Sommerseth, 2023). Compared to Amsterdam, Copenhagen and Palma, for example, Sundsvall had a significantly smaller population. When our study begins Sundsvall was in the outskirts of Europe, not only in a geographical sense but also when it came to economy and communications, something that changed fundamentally during the last decades of the 19th century.

The focus of this study is on the pattern and development of causes of death among infants. By applying the same *Historic cause of death coding and classification scheme for individual-level causes of death* (ICD10h) (Reid et al., 2024a) and a common infant categorisation (InfantCat_2020) (Reid et al., 2024b), to our historical cause-of-death data, comparisons with the other articles in this special issue are made possible. The approach is mainly descriptive with the purpose to document the urban epidemiologic transition (Omran, 1971) during a period of rapid social, economic and health-related changes. Furthermore, we will discuss our analysis of the development of infant mortality in relation to possible determinants and accessible knowledge about the socioeconomic situation as well as public health in Sundsvall during the study period.

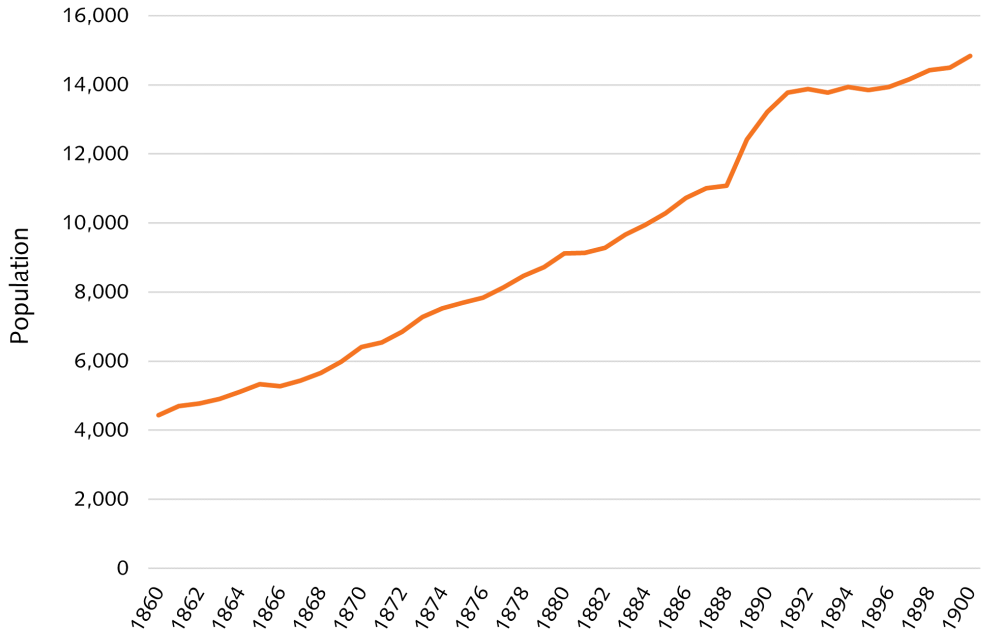
1.1 SUNDSVALL IN THE 19TH CENTURY — A SMALL TOWN IN GROWTH

In the first half of the 19th century, Sundsvall was a small town on the coast of the Baltic Sea, with a population of only a couple of thousand inhabitants (on 19th century Sundsvall, see Edvinsson (1992)). Until the middle of the century, its economy was dominated by small-scale trade, handicraft and fishing.

At the time when our study begins, Sundsvall was undergoing a rapid transformation. International demand for forest products increased and the large forest areas in northern Sweden became valuable (on the development of Sundsvall 1860–1900, see Björk and Schnell (1979) and Edvinsson (1992)). Rivers were cleared in order to facilitate deliverance of timber to the sawmills along the coast. The first steam sawmills were started around 1850, thereby starting the process of increased production of sawn products. Sundsvall was strategically located between two large rivers coming from the large forests in the inlands. This started an economic boom in the district as well as in the port city of Sundsvall, which became the commercial centre of the area. Enormous wealth was created for some, and job opportunities for others, although with less financial reward. The economic expansion brought Sundsvall into an international context. Ships from large parts of Europe served the port of Sundsvall.

The work opportunities in the Sundsvall district attracted labourers from many parts of Sweden but also from neighbouring countries, particularly from Finland. In the beginning seasonal work dominated, but over time production continued throughout the whole year. The population in the town of Sundsvall increased rapidly from a little more than 4,000 in 1860 to around 14,000 in 1900 (see Figure 1). The most expansive period was in the 1870s, which in turn caused problems with overcrowding, sanitation and access to fresh water as the local government was unable to keep up with the health requirements of the whole population. From 1875, all cities and towns in Sweden had to appoint local health boards with specific responsibilities for different aspects of health (water and sewage, sanitation, waste disposal, epidemic diseases and so on). From 1879, piped water and sewers were introduced in parts of Sundsvall. Another problem came in 1888 when large parts of the town were burnt. The following years were characterized by rapid expansion, when the town was rebuilt. From this point, only stone buildings were allowed in the city centre, leading to a large division between the wealthy centre and the poorer surrounding areas (Paulsson, 1953, p. 244). The problems also had implications on social differences in mortality, especially in child mortality where working class children had much higher mortality, less so in infant mortality and not at all in male mortality in adulthood and old age (Edvinsson, 1992, 2004; Edvinsson & Broström, 2012; Edvinsson & Lindqvist, 2011).

Figure 1 *Population development in Sundsvall 1860–1900*



Source: Bidrag till Sveriges officiella statistik A Befolkningsstatistik (1859–1912).

1.2 THE DATA

The analyses are based on digitized parish registers for the parish of Sundsvall. These data come from the POPUM database (Westberg et al., 2016) hosted by the Demographic Data Base (DDB) at the Centre for Demographic and Ageing Research (CEDAR), Umeå University. The major advantages of Swedish parish registers are that they cover the complete population (also the minorities not belonging to the Swedish Lutheran Church) and that the registers reporting the parish populations (i.e. the catechetical registers) are continuous, making it possible to follow individuals during the time spent in the parish. The registers for demographic events (births, deaths and marriages) include all events taking place in the parish even for those with a different home parish as well as events among parishioners taking place outside the home parish (for more information about Swedish parish registers, see Nilsdotter Jeub (1993)). In this article, we only include the *de jure* population in accordance with the official way the population was defined in Sweden at the time. Information on births and deaths of those that did not belong to Sundsvall was sent to their home parish. To be defined as living in a certain parish, i.e. belonging to the *de jure* population, you had to be registered in its catechetical registers, consequently, births and deaths of those not registered in the catechetical registers of Sundsvall are not included.

During turbulent periods with a lot of migration, some did not inform the authorities about their change of residence. The most expansive period resulted in a large proportion of the population not being registered, as many migrants avoided informing the authorities about their new place of residence. This means that the *de facto* population could be different from the *de jure*. However, since it is impossible to adequately estimate the *de facto* population, we restrict the cohort to the *de jure* definition. When it comes to infants, another problem appears. Those that died very young were not always entered in the catechetical registers. Our definition of the infant population for this study is therefore based on the following requirements:

1. The child was stated as being administratively belonging to the parish according to the birth register and/or the catechetical register;

2. If no such information is given, we checked if the mother was resident in the parish at the time of the child's birth (i.e. registered in the catechetical register at the time of the birth);

3. In addition, children, not born in Sundsvall who moved into the town and died within the first year of life are included in the infant population.

The definition influences the size of the population to be analysed. We have compared the study population with those from earlier studies (Edvinsson 1992, 1993, 2004) as well as with official statistics.

Compared with these earlier sources, there are some differences in approach and definitions (for example in Edvinsson (1992) a cohort approach was taken). We conclude that although some minor differences in the number of infants can be identified (a 5% difference in births and 2% difference in infant deaths), the comparisons show no significant problems with the selection of infants for the current study.

1.3 BASIC DESCRIPTIVE STATISTICS

The period studied here is 1860–1892. Due to the extensive fire in Sundsvall in 1888, some of the older parish registers were destroyed, which has determined the starting year of our study. The remaining catechetical registers from before 1860 do not provide full information on infant deaths and no information at all on causes of death.

Table 1 *Basic descriptive statistics on the population of Sundsvall 1860–1892*

Description	Total	Mean per annum
Years covered by the data	1860–1892	
Population of Sundsvall in 1860–1892	4.432–13.873	
Total number of infant deaths	1.739	53
Total number of live births	10.248	311

In some of the analyses, the period 1860–1892 is divided in approximately 10- or 5-year groups. Apart from providing similar lengths of time, they can also be considered as representing distinct characteristics in the rapid transformation of Sundsvall. During the 1860s, industrialization had just begun in the district, while the 1870s marked the decade of the most rapid economic expansion. In the 1880s, the growth stabilised, and several sanitary improvements were introduced. The final period covers the years following the devastating fire of 1888.

1.4 CODING AND CLASSIFICATION OF CAUSES OF DEATHS ACCORDING TO ICD10h

In the present study, the *Historic cause of death coding and classification scheme for individual-level causes of death*, ICD10h (version 2020), has been applied to code and classify the historical cause of death data from the POPUM database at CEDAR (Reid et al., 2024a).

ICD10h has been designed to aid the coding and classification of causes of death recorded on historic individual death records. The ICD10h system is based on the 10th revision of the International Classification of Diseases – 2016 version (ICD10-2016) and combines ICD10 codes (without modification) with new codes for archaic/historic terms.

The ICD10h code system has a structure that follows the ICD-10 system's four-digit alphanumeric code extended by two decimal places. The purpose of this extension is to provide scope for assigning special codes to archaic/historic terms that have been interpreted as synonyms for the same disease. For more information on the construction of the system, see Janssens (2021) and Reid, Garrett, Hiltunen Maltesdotter, and Murkens (2024).

The 2020 version of ICD10h is the beta version of the now published 2024 version of the ICD10h coding system. The 2020 version of ICD10h can be reverse engineered using the ICD10h version 2024 and the transfer table between ICD10h2020 and ICD10h2024.

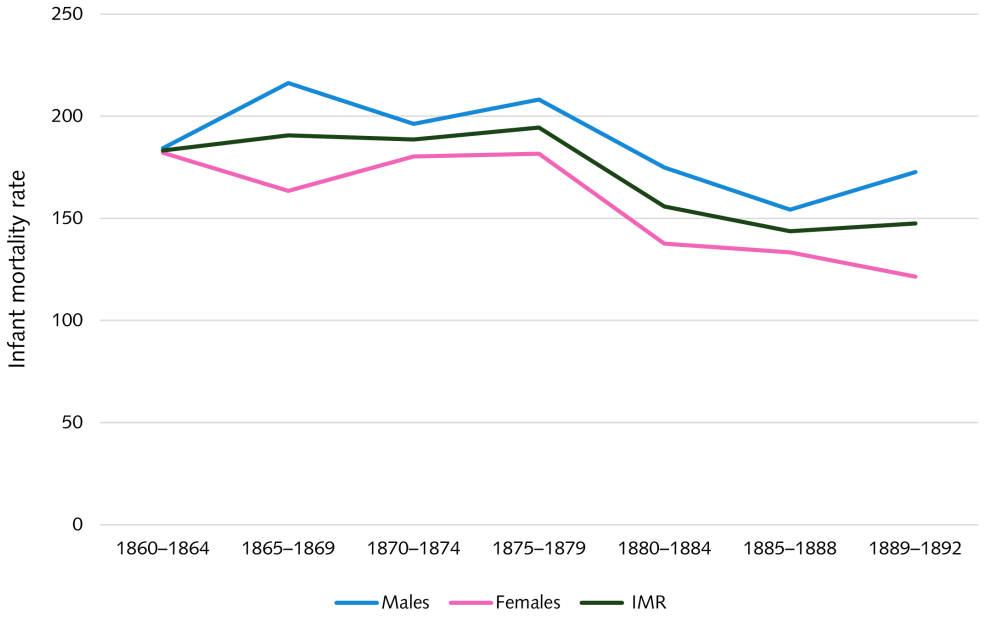
2 OVERVIEW OF INFANT MORTALITY RATES

Epidemic diseases frequently flourished in Sundsvall, some of them increasing the number of infant deaths significantly. In 1862 diphtheria and measles caused many child deaths, two years later scarlet fever ravaged. In 1866 a cholera epidemic (although primarily affecting other age groups than infants) caused large problems. After some decades of very low smallpox mortality, the disease came back in 1872–1873. During the following two years, 1874–1875, scarlet fever returned. Adding to these larger epidemics, there were frequent outbreaks of diseases like whooping cough and typhoid fever as well

as less violent epidemics of diphtheria, scarlet fever and measles. Figure 2[1] shows the development of infant mortality for boys and girls during our studied era in 5-year periods (for yearly rates see Figure A2 in the appendix). The level of infant mortality was slightly below 200 per thousand up until around 1880. Thereafter the survival of infants improved, resulting in levels between 100 and 150 per thousand. The mortality decline coincided with the sanitary improvements at this time, suggesting a possible causal relation.

Sweden has been characterized by lower female mortality across almost all age groups, not least in infant mortality, since the earliest time of available data (Edvinsson, 1992; Willner, 1999). Newborn boys have always been more vulnerable. This was also the case in Sundsvall, with statistically significant differences. There is no clear trend in the differences, although the female advantage became larger after the fire in 1888 when the survival of infant boys decreased.

Figure 2 *Quinquennial infant mortality rates by sex, Sundsvall 1860–1892*



Note: A Students T-test on the difference in infant mortality rates between males and females resulted in a P-value = 0.0028.

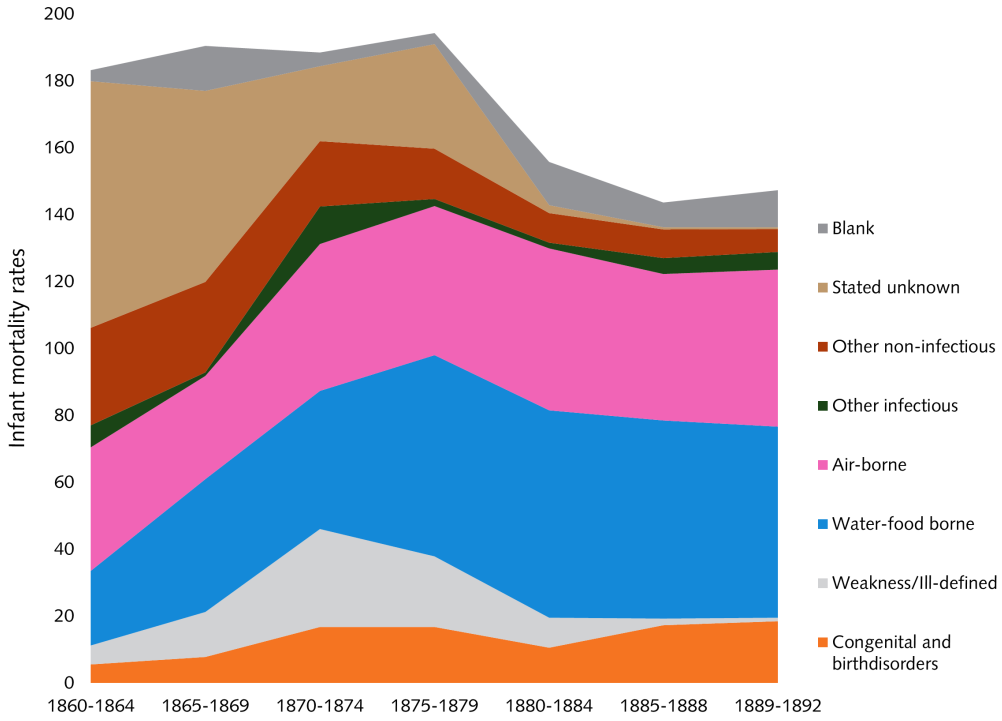
Source: The data set was based on the POPUM database, parish of Sundsvall, 1860–1892 (Demographic Data Base, CEDAR, Umeå University, 2021).

3 CAUSES OF DEATH

From 1860, it became mandatory to have a physician certifying the cause of death for every fatality in towns and cities (Rogers, 1999; Svensk Författningssamling, 1860). Consequently, the information on causes of death reflected the best medical knowledge in Sweden at the time. During the study period, it became increasingly common to report multiple causes, often in Latin. Causes of death reported for the infants were usually less precisely stated than for fatalities in other age groups (Edvinsson, 1992), a common feature for reports of historical causes of death not only in Sweden but in other countries as well. Many of the causes described symptoms or only gave the location of the disease, for example diarrhoea or chest disease. There are however also causes that were easily diagnosed and distinguished at the time, at least by physicians, for example epidemic diseases such as smallpox, measles, whooping cough and dysentery.

1 The results on which the figures in this article are based on are presented in tables in the appendix A and B.

Figure 3 *Quinquennial infant mortality rates by causal groups, Sundsvall 1860–1892*



Note: The final period includes only four years.

Source: As Figure 2.

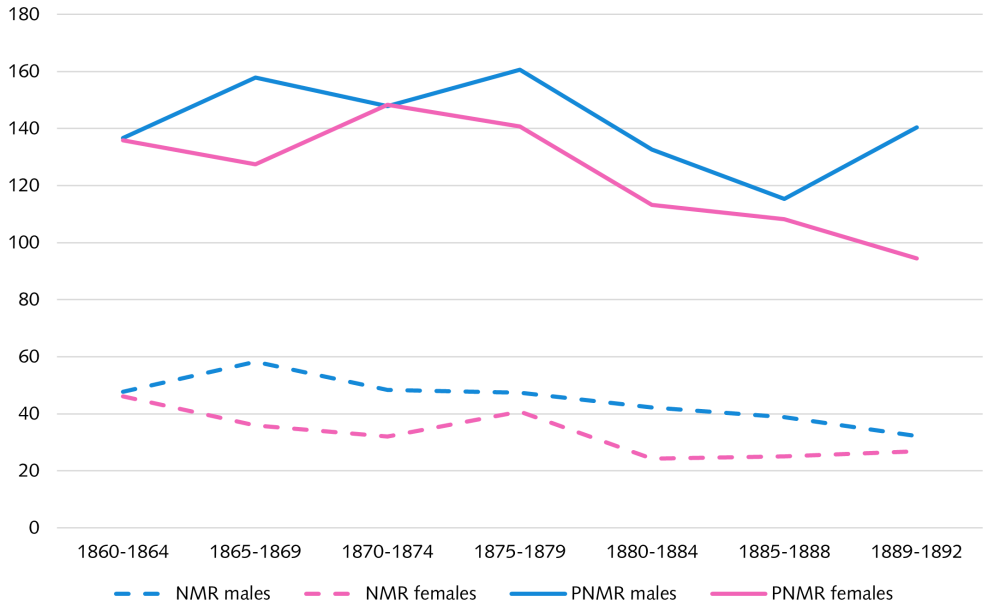
The improving quality of the death certificates can be seen in Figure 3.[2] Unknown and weakness/ill-defined causes accounted for a large proportion of all infant deaths in the 1860s, but both the actual numbers and their proportion of all deaths diminished in the following decades. The vague diagnosis "weakness" was used mainly in the 1870s. The largest cause of death groups were air-borne infections, water- and foodborne infections and congenital disorders. Towards the end of the study period, mortality in these groups increased and represented a large majority of all deaths. It is difficult to estimate how much of this increase can be attributed to improved diagnosis of infant mortality, but the increase may indicate that the previously unknown and vague diagnoses have hidden many of the deaths in the other groups. In any case, it seems likely that improved diagnosis is part of the explanation.

4 NEONATAL AND POST-NEONATAL MORTALITY — AN OVERVIEW

Neonatal mortality (first 28 days) is considered to be associated with physical fitness of the baby as well as of the mother and circumstances at delivery, while post-neonatal mortality (from day 29 and the rest of the first year) is increasingly dependent on conditions in the environment. The newborn child gets protection from infections through the mother, and if the child is breastfed, the protection is maintained. When the child gets older, the protection vanishes, making it exposed to threats from infections and epidemic diseases in the local environment (Bourgeois, 1946; Brändström, 1984; Reid, 2001, 2002).

2 Diagnoses such as convulsions (9 cases), teething (2 cases) and other ill-defined causes as well as the category of external causes (6 cases) were rarely used in Sundsvall during the study period. We have therefore reported them together with weakness (category Weakness/Ill-defined). In order to make it possible to compare with results from other port cities, we present the full distribution of causes of death in Appendix B.

Figure 4 Quinquennial rates of neonatal (NMR) and post-neonatal (PNMR) mortality, by sex, Sundsvall 1860–1892



Note: The final period includes only four years.

Source: As Figure 2.

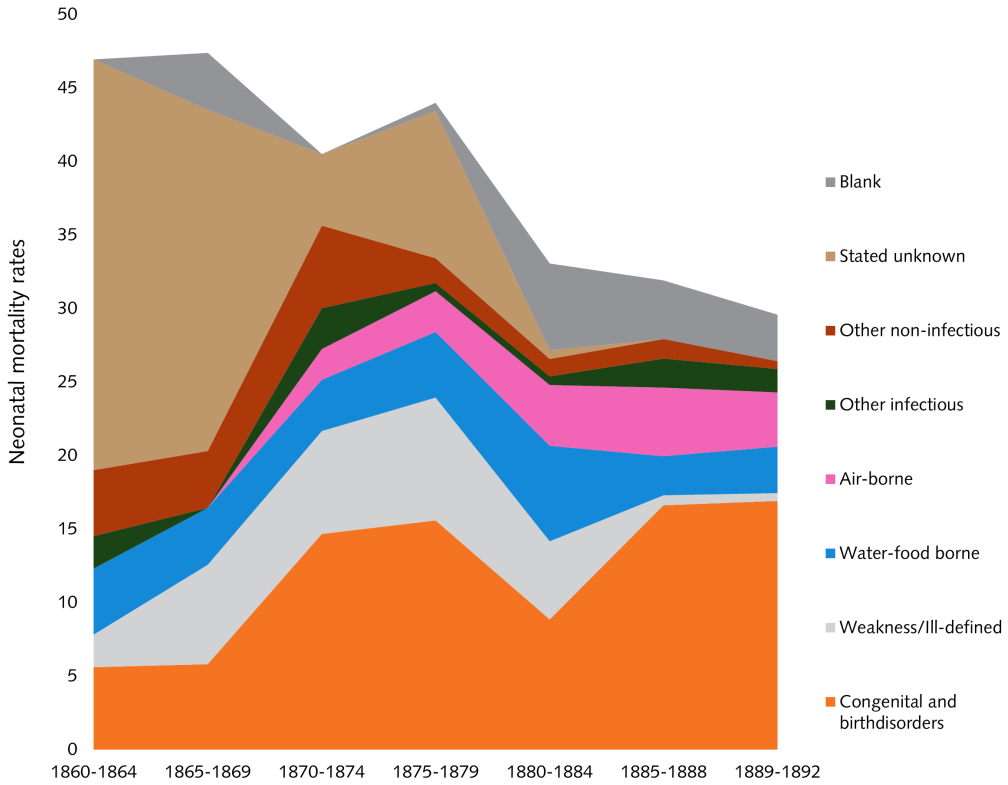
Figure 4 shows the development of neonatal (NMR) and post-neonatal mortality (PNMR) by gender in Sundsvall. In both ages, boys were more exposed to death. When it comes to the post-neonatal age group, there was no initial decline in mortality. There was a modest increase until the 1870s, with slight differences in timing between boys and girls. However, a substantial decline followed, indicating that health conditions for this age group worsened during Sundsvall's most expansive period. During this period, overcrowding increased and poor sanitary conditions caused large health problems, for example in mortality among children older than one year (Edvinsson, 1992). The timing of the decline coincides with the introduction of health measures and sanitary improvements, for example the introduction of piped water and sewerage in the late 1870s.

In contrast to post-neonatal mortality, where we observed a clear turning point around 1880, neonatal mortality does not show such a shift. Instead, we notice a steady and continuous improvement in survival among newborn infants from this time onward. On the one hand, this may be due to the same conditions as for post-neonatal mortality but can also be explained by improvements in midwifery. Around 1880, the new hygienic techniques of asepsis and antisepsis were introduced, which improved the survival of the mothers, and it is likely that these were also beneficial for the newborn's ability to survive (Andersson et al., 2000; Högberg, 1985, 2004).

5 NEONATAL AND POST-NEONATAL MORTALITY RATES BY CAUSE OF DEATH

In order to better understand what determined the patterns and developments of infant mortality, information on causes of death is of vital importance. From 1860, all deaths taking place in Swedish urban settings were expected to be diagnosed by a physician. The central authorities in Sweden at this time developed a nosology for registration of causes of death, which was inspired by and partly similar to those from other countries such as Belgium and the United Kingdom. The nosology of 1860 was used until 1874 when a new version was introduced which in turn was followed by the version published in 1891 (Svensk författningssamling, 1860, 1874, 1891).

Figure 5 *Quinquennial rates of neonatal mortality, by causal group, Sundsvall 1860–1892*



Note: The final period includes only four years.

Source: As Figure 2.

Figure 5 shows the cause of death patterns in neonatal mortality, using the same categories as in Figure 3. What immediately stands out during the period 1860–1869, is the large proportion of deaths attributed to unknown or ill-defined causes of death such as weakness and similar diagnoses, which make up almost two thirds of all neonatal deaths. This, of course, complicates the in-depth analysis of causes of death during the period 1860–1869. Weakness was a rather common cause for a couple of periods in the 1860s and 1870s. The incomplete and unsatisfactory reporting of causes probably reflects the circumstance that doctors were rarely called upon in cases of illness in infancy and consequently made the diagnosis postmortem, often only through information from relatives (Edvinsson, 1992; Reid & Garrett, 2012), but it may also be due to their inability to correctly diagnose the causes of death among infants. However, the proportion of unknown and poorly defined causes of death decreased markedly from the late 1880s onwards, and at the same time an increase in deaths from congenital diseases and birth disorders is noted. It is quite possible that improved diagnostics explain this change, i.e. a transfer from the unknown/ill-defined group to the congenital. Reported deaths from water- and food-borne infections were rather stable over the whole period, while air-borne infections increased.

To summarize the cause of death information for the neonatal period, we conclude that several uncertainties remain. In the 1860s, a large proportion of the diagnoses were recorded as unknown diseases, or were given vague and symptom-descriptive diagnoses, but we can clearly see that the diagnostic methodology improved over time. This may shed light on how the medical profession and the families dealt with health problems among infants, giving less attention to their ailments and problems compared to other age groups. Despite the problems mentioned here, we believe that cause of death information can still provide valuable insights into the health of the very young, provided it is carefully evaluated and considered alongside other available information. For example, it is possible that the high mortality from water- and food-borne infections in the four periods from 1870 to 1888 reflects the troublesome sanitary conditions during the period of most rapid growth in Sundsvall.

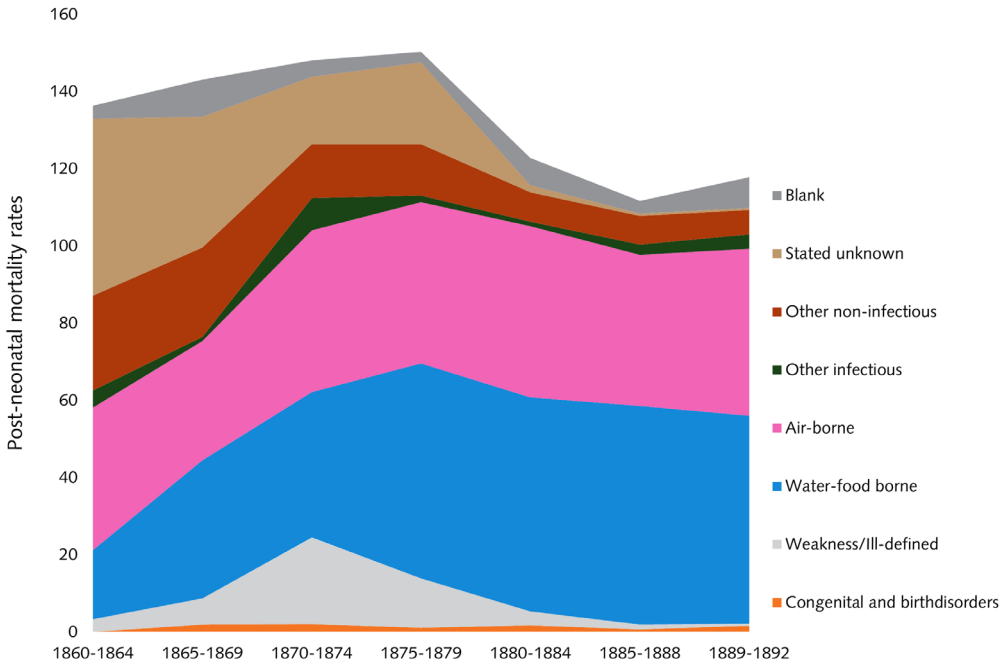
For post-neonatal mortality on the other hand, unknown causes are less prominent, except for the 1860s and to some extent the 1870s (Figure 6). Diseases comparatively easy to identify, such as smallpox, scarlet fever, whooping cough were more common in this age group. This is not to say that all diagnosed causes of death during the post-neonatal period were clear and precise.

The dominant cause of death categories were water- and food-borne infections and air-borne infections. Infant diarrhoea was a very common cause of death, maybe reflecting the sanitary situation of the town but could also be an artefact of the strong decline in unknown cases (see also the discussion in section 7). However, another important influence on this is the extent of breastfeeding versus artificial feeding and in particular the timing of weaning. This complicates the interpretation of the development of diarrhoeas. Other studies have shown that bottle feeding was more common in the county where Sundsvall is located than in most other counties in Sweden (Brändström, 1984; Edvinsson, 1992). Local physicians in Sundsvall reported frequently in the 1860s and 1870s about the widespread use of artificial feeding (on breastfeeding patterns, see Edvinsson (1992)). The age pattern of infant deaths do not indicate that many infants were never breastfed. However, analyses are not conclusive about age at weaning. A fairly high mortality from diarrhoea during the second to sixth month of life indicates early weaning. According to later reports from these physicians, the problem became smaller towards the end of the century. There were large campaigns to promote breast-feeding in Sweden at the end of the 19th century. However, it has been difficult to prove whether these campaigns really changed the behaviour in Sundsvall (Edvinsson, 1992). Nevertheless, it would be expected that sanitary improvements led to fewer cases of diarrhoea among infants in the years following 1880, but this study shows that these diseases increased in prevalence among children of post-neonatal age.

Mortality from air-borne infections was the other major killer in the post-neonatal age group. Although the level of mortality in air-borne diseases increased slightly from 1865 onwards, the increase was modest compared with water- and food-borne diseases. These two groups contributed the majority of all deaths in the later periods. As discussed above, we expect a higher mortality rate in epidemic air-borne diseases during the post-neonatal period, since the immunity transferred from the mother at birth and through breastfeeding declines with age.

The cause of death pattern for neonatal and post-neonatal mortality shows no significant difference between boys and girls (see Table A6 in appendix). Boys experienced higher mortality in the dominant disease groups in both neonatal and post-neonatal age.

Figure 6 *Quinquennial rates of post-neonatal mortality, by causal group, Sundsvall 1860–1892*



Note: The final period includes only four years.

Source: As Figure 2.

6 SEASONALITY

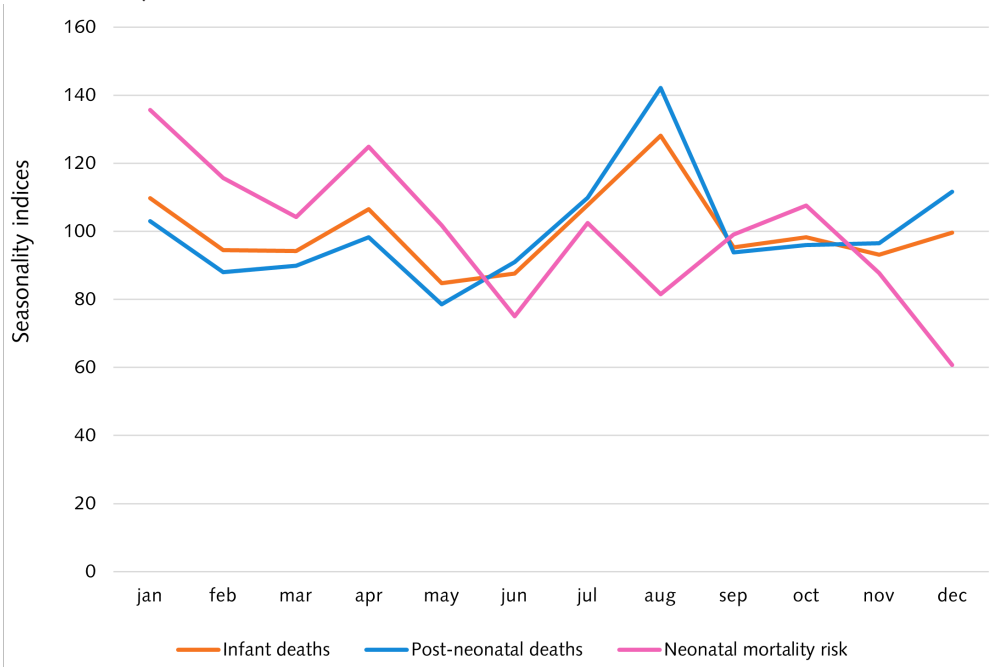
In this final analytical section, we look at the seasonality of mortality. For infant mortality and post-neonatal mortality, the deaths are put in relation to the number of births during the whole year, i.e. not controlled for possible seasonality in births. Such seasonality has little influence in these two measures. This is, however, not the case for neonatal mortality. Months with many births will obviously tend to have more infant deaths since the mortality risks are highest immediately after birth and the following weeks. Thus, for neonatal deaths we have used the neonatal mortality risk that takes into account the seasonal variation of births.

Seasonality for neonatal mortality risk has been calculated as follows:

$$\frac{no\ of\ deaths \div 0.5 \times (no\ of\ births + (no\ of\ births\ in\ previous\ month \times (no\ of\ days \div no\ of\ days\ in\ previous\ month))}{no\ of\ deaths\ in\ year \div no\ of\ births\ in\ year} \times 100$$

Figure 7 presents seasonal mortality for the entire period studied (1860–1892). We see some distinct differences according to infant age. Since the majority of infant deaths took place during the 11 months of post-neonatal age, the infant and post-neonatal mortality show a strong resemblance. They both have a marked summer peak (July and August). This is not the case for the neonatal period where mortality is lower during the summer months. All age groups (IMR, NMR and PNMR) show a peak in late winter and early spring (April), which is a season when mortality was traditionally high in Sweden, see *Bidrag till Sveriges officiella statistik. A. Befolkningsstatistik* (1859–1912).

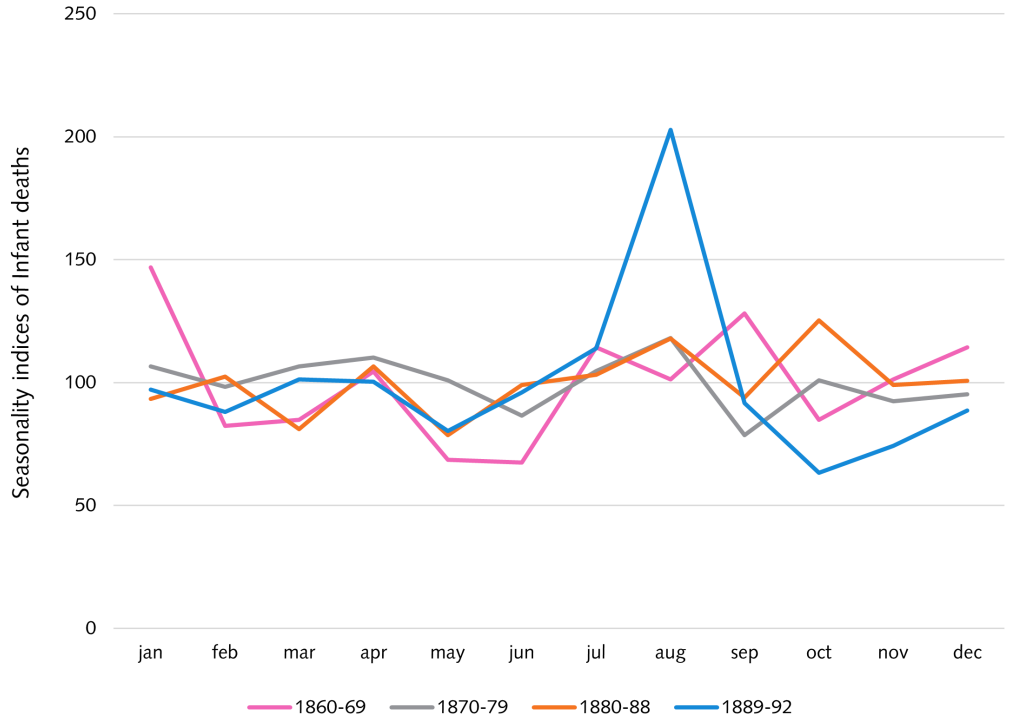
Figure 7 *Seasonality indices of infant deaths, neonatal mortality risk and post-neonatal deaths, by month, Sundsvall 1860–1892*



Source: As Figure 2.

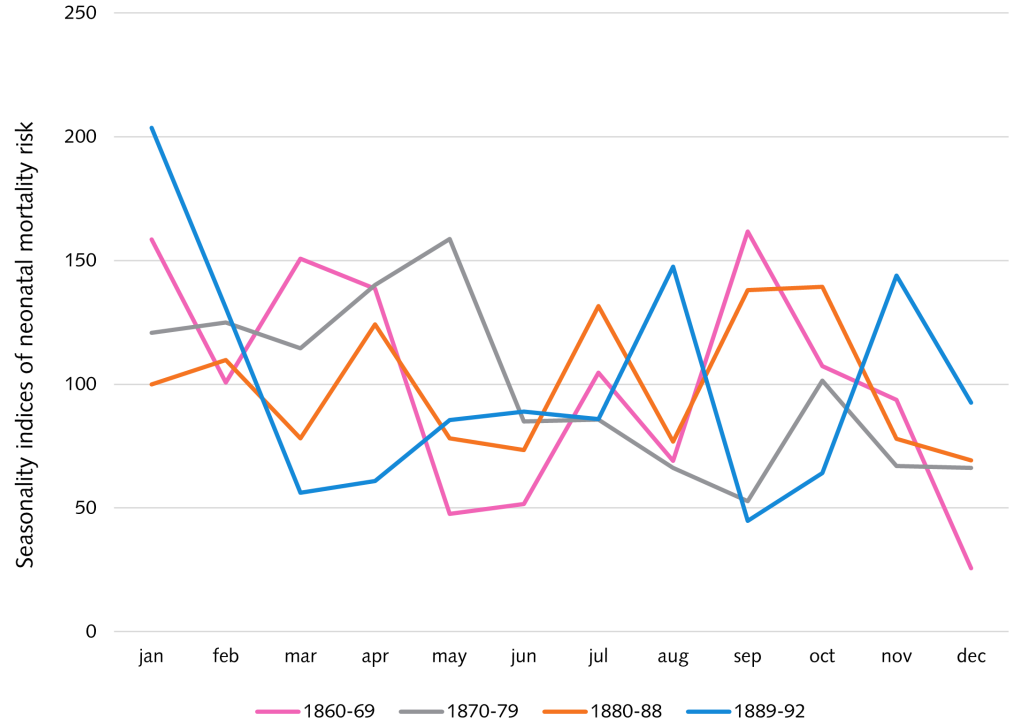
Figures 8a-c describe the seasonal variation of deaths divided by age group and approximate 10-year periods. For infant mortality, there were some changes over time (Figure 8a). The most apparent one took place in the period that followed the great fire of 1888 had very high mortality in late summer. As shown in figure 2, there was only a marginal increase in infant mortality during this period, but something made conditions worse during late summer. We know that large parts of the population lived in provisional and overcrowded houses during the first years after the fire. Furthermore, the local press reported several problems with the water quality at this time (Edvinsson, 1992). However, the peak in mortality among infants and post-neonatal children seen in August during the period 1889–1892 is not found in the earlier time periods, where the seasonal variation was relatively small, see Figures 8a and 8c. For the periods 1860–1888, it is not possible to identify any differences between the periods, nor can any consistent pattern of seasonality be discerned. As we expected, the pattern of post-neonatal mortality resembles that of infant mortality, but with somewhat higher mortality during the months July to September and December to January (Figure 8c). When it comes to neonatal mortality (Figure 8b), the number of deaths is relatively small in our dataset, making it difficult to identify any general seasonal pattern or trend over time.

Figure 8a *Seasonality indices for deaths amongst infants, for selected periods, Sundsvall 1860–1892*



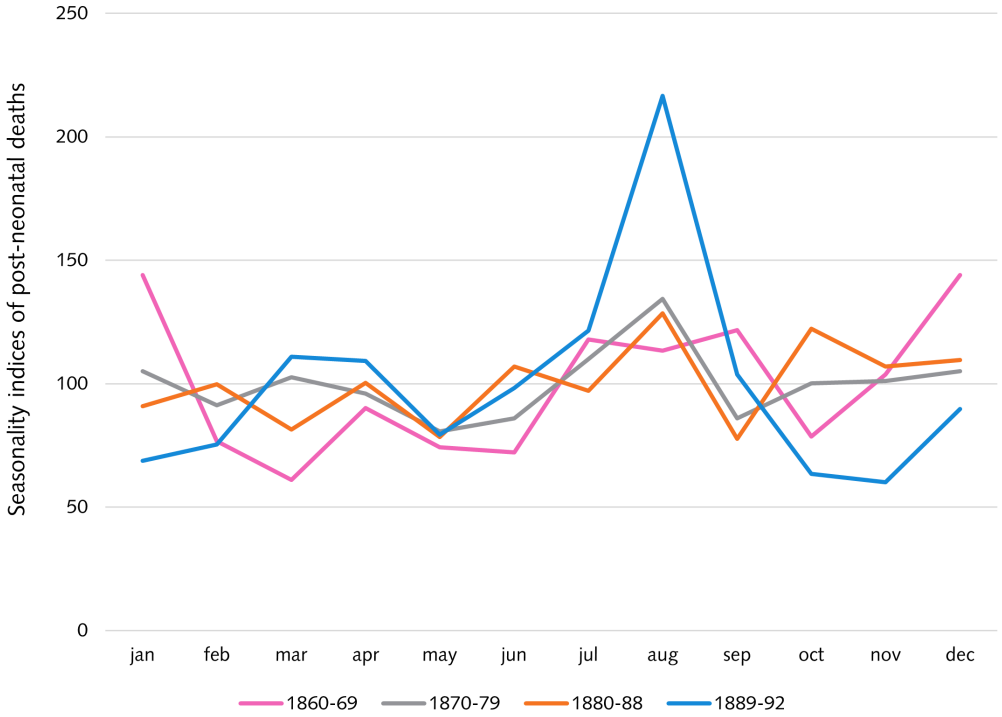
Source: As Figure 2.

Figure 8b *Seasonality indices of neonatal mortality risk, for selected periods, Sundsvall 1860–1892*



Source: As Figure 2.

Figure 8c *Seasonality indices for deaths amongst post-neonates, for selected periods, Sundsvall 1860–1892*



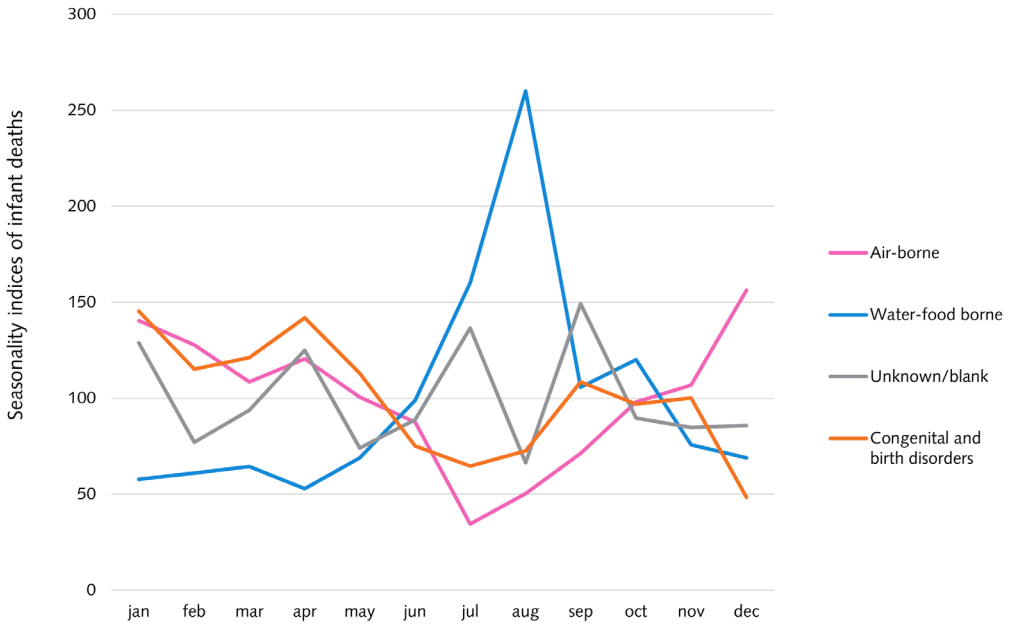
Source: As Figure 2.

To better understand the seasonal pattern of infant mortality, we now turn to an analysis of the seasonal variation for the underlying causes of death. In this part we focus on the cause of death groups that have the highest death rates (air-borne infections, water- and foodborne infections, congenital and birth disorders and unknown causes) since the number of cases in the other groups is too few to allow any meaningful analysis of seasonal variations. Figure 9 shows the seasonal pattern of infant mortality in four different cause of death groups for the whole study period. As expected, water- and food-borne diseases had a very strong seasonal pattern with the peak in late summer — July and August. Consequently, the peak in total infant mortality during these months is to a large extent explained by the high mortality in infant diarrhoea and other water- and food-borne infections. We know that there were large sanitary problems in Sundsvall at this time, as there was a general problem with Swedish urban living conditions at this time (Edvinsson, 1992). The summer peak in the late 19th century was particularly present in urban environments, but not at all or less pronounced in the Swedish countryside (Edvinsson, 1992; Nilsson, 1994).

Mortality from air-borne diseases on the other hand was at its lowest during summer. The peaks in mortality from these diseases were instead concentrated in the winter months. One explanation for this may be that more time was spent indoors during the winter months, which increased the risk of spreading air-borne diseases. The "unknown disease" causal group does not display any remarkable pattern and do neither resemble the pattern of water- and foodborne diseases nor air-borne diseases. Since post-neonatal deaths contributed to 70–80% of all infant deaths, we observe, as expected, the same pattern for infants 29 days and older (Figure 10).

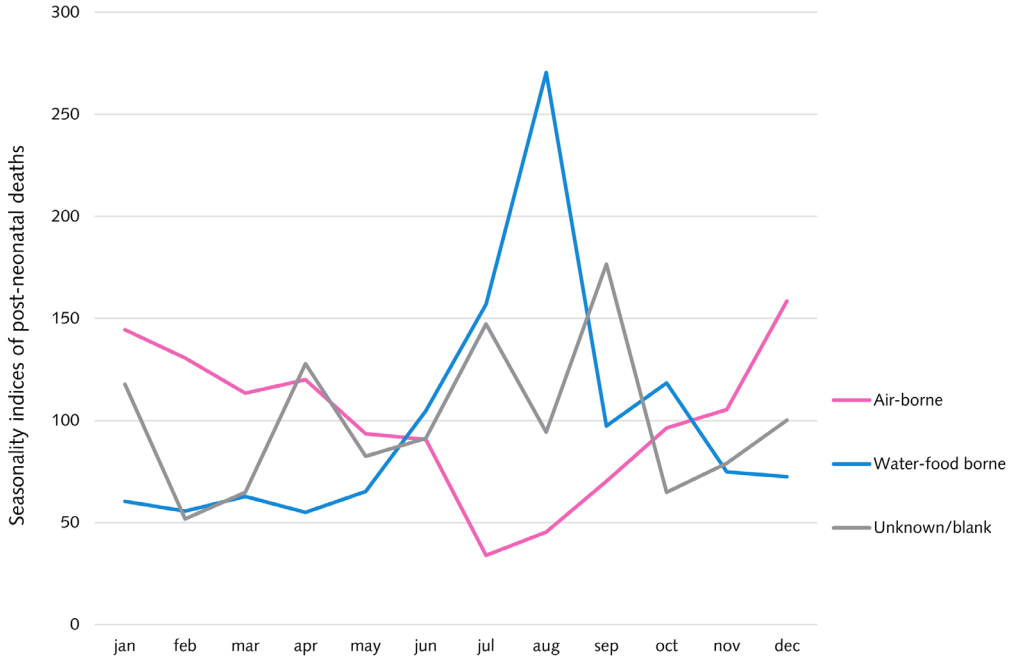
Figures 10 and 11 illustrate the fact that neonatal mortality had a very different seasonal pattern in causes of death. As we saw in previous analyses (Figure 5), the largest cause of death groups during the first weeks of life were congenital and birth disorders and unknown causes, with a concentration of the first group from the 1870s onwards and the second in the 1860s. By looking at the seasonal pattern for neonatal mortality in Figure 11, we can conclude that deaths in both of these cause of death groups mostly occurred during winter and autumn, while the number of deaths were low during summer months. This may indicate that they represent similar conditions, suggesting that congenital and birth disorders were hidden behind the term unknown disease in the 1860s. However, more thorough analyses are required to confirm this assumption. We also notice that the less frequent water- and food-borne diseases show a seasonal pattern similar to that in the post-neonatal age, with a rise during summer. In contrast, the seasonal pattern is less clear for airborne diseases, apart from a peak in May, but considering that the number of deaths is relatively few, it is difficult to draw any definite conclusions.

Figure 9 *Seasonality indices of infant deaths, by selected causal groups, Sundsvall 1860–1892*



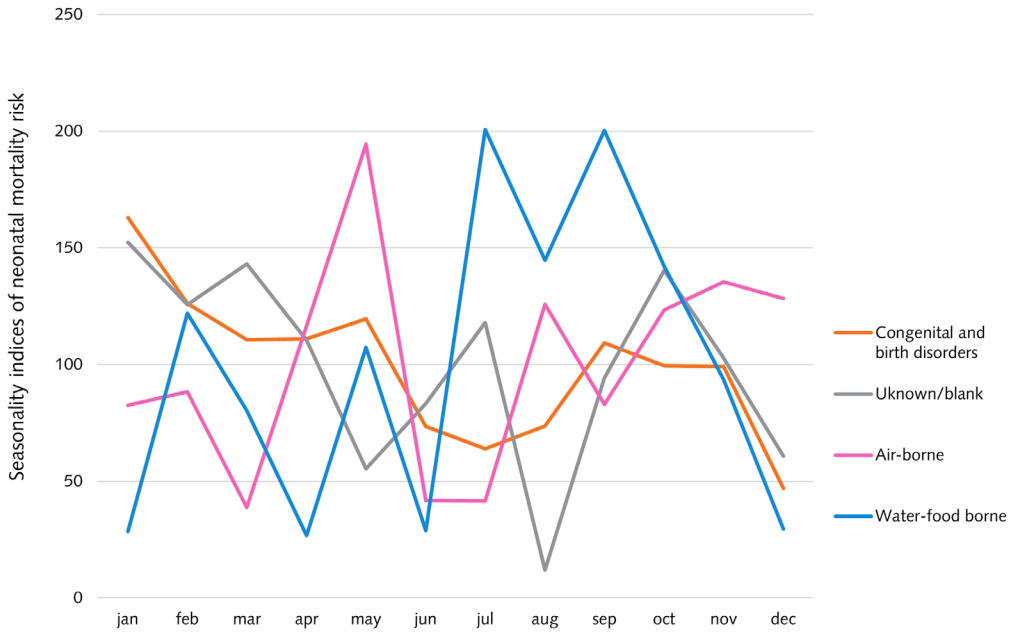
Source: As Figure 2.

Figure 10 *Seasonality indices of post-neonatal deaths, by selected causal groups, Sundsvall 1860–1892*



Source: As Figure 2.

Figure 11 *Seasonality indices of neonatal mortality risk, by selected causal groups, Sundsvall 1860–1892*



Source: As Figure 2.

7 REFLECTIONS AND DISCUSSION

Based on the results from this study, in this last part of the article we will present some reflections on how analyses of cause of death data can contribute to our understanding of health and living conditions during the 19th century.

The discussion is divided into two parts. The first part deals on a general level with historical analyses of cause of death data. By putting the analyses of cause of death data in relation to their context, a city in constant change, we show the possibilities of creating a broader picture of the complex living conditions of the time. We also address both the opportunities and challenges inherent in working with historical cause of death data. The second part offers some of our reflections on the implementation of an international, common coding system as well as a classification scheme for historical causes of death, designed for use in different countries and in different languages.

In this article, we have provided a description of mortality among infants with a focus on how the causes of death change over time and vary by season, and we have illustrated the gender differences. To further deepen the understanding of the living conditions, we will now combine these results with information about the extensive and rapid change that was taking place in Sundsvall. This will serve as an example of how these outcomes may contribute to understanding the decline in urban mortality in the late 19th century.

At this time, Sundsvall was in many respects different from other European port cities. Compared to cities like Amsterdam or Copenhagen, Sundsvall was a small town in the European periphery. Nevertheless, even if the population figures were comparatively low, it shared many of the characteristics of other and more populated cities, both with and without a port function. Mortality was high due to high population density, rapid population increase, sanitary problems and problems with overcrowded housing. The urban penalty (Kearns, 1988) was a reality even in comparatively small places. The latter half of the 19th century was a turbulent time in the history of Sundsvall. From being a small town in the mid-north of Sweden, it became the centre of a rapidly expanding saw-mill district. This brought many advantages to people living there or migrating to the area, but it also caused many problems, not least for health.

Infant mortality in Sundsvall was high in the beginning of our study period but declined substantially from around 1880 onwards. The decline might be attributed to sanitary measures taken around the time of the onset of the decline. The role of public health and sanitary improvements in the mortality transition has been discussed for a long time (McKeown, 1976; Szreter, 1988). As early as the end of the 19th century, the measures taken by the health boards and consequent sanitary improvements were suggested, by the physician Carl Lindman (1897), as an explanation for declining Swedish infant mortality rates. Lindman collected infant mortality rates for the 20 largest Swedish cities and towns from 1876 to 1895, and he argued that the tipping-point leading to lower mortality largely coincided with a variety of public health measures. The possible impact of these measures on health and mortality has continued to be discussed (see for example Szreter (1988) and van Poppel and van der Heijden (1997)). Several studies have been performed on the Swedish development, and although much speaks in favour of improved sanitation for improved survival, the exact pathways as indicated by several in-depth analyses of causes of death are not always clear (Edvinsson, 1992; Edvinsson & Nilsson, 1999; Edvinsson & Rogers, 2001; Helgertz & Önnerfors, 2019; Lazuka et al., 2016; Macassa et al., 2006; Nilsson, 1994).

In the present study we have shown that the diseases, i.e. diarrhoeas and other gastro-intestinal disorders, that could be expected to have declined the most due to these sanitary improvements, in reality did not decrease at all. In fact, it is not always the case that sanitary improvements have had exclusively positive effects. Contemporary comments from Sundsvall complained about the water quality when piped water was introduced. Furthermore, the sanitary improvements did not reach the whole population. Piped water and sewers were in the first decades only available in the central parts of Sundsvall, while the conditions remained bad in the outskirts where the workers and poor people lived (Edvinsson, 1992). It is also possible that deaths in infancy are less driven by sanitary conditions than in other age groups, as suggested by Davenport et al. (2019). Gastro-intestinal problems may signify something different in infancy compared to other age groups, such as breastfeeding practices.

However, the strong late summer peak after the fire in 1888 might indicate that new problems arose during these years, although infant mortality continued to decline. This peak was predominantly caused by waterborne diseases. In general, the problems after the fire did not cause any increased mortality in Sundsvall, but may have made infants, particularly in post-neonatal age, more exposed to gastro-intestinal disorders in late summer. Overcrowded living conditions were common and there were problems with the quality of piped water.

Our analyses have shown both the possibilities as well as the limitations of using historical cause of death data on historical problems. We would like to stress that causes of death must always be considered within their historical context and should be interpreted together with factors, for example, age at death and seasonality and also be related to contemporary medical knowledge and how this information was reported at that time. We may conclude that the analysis of the demographic development and what the citizens died of, is important for understanding of the living conditions in Sundsvall.

Finally, we offer some reflections on the use of a common international scheme for coding and classifying causes of death. There are great advantages of such a research tool for enabling comparisons are clear and require no further justification. At the same time, it is equally important to be aware of the pitfalls and limitations inherent in such international systems, and to consider their possible implications. Before beginning the task of coding and analysing historical cause of death data, it is essential to reflect on the following: What were the prevailing historical concepts and contemporary medical knowledge? What significance did health and diseases hold in the society of that time and for the age group being studied? What conclusions can be drawn and what consequences will these have for the assignment of codes and for the interpretation of historical health conditions?

It is important to keep in mind that assigning a code to a sometimes vague diagnosis, does not necessarily make it more informative or specific. Coding simply allow us, in a consistent way, to arrange the diagnoses according to a structure that aims to facilitate analysis and comparison with data from other sources. Structuring historical information via coding and aggregation, means that certain linguistic nuances in the information by necessity are lost. A challenge faced during the coding work is to interpret what different diagnoses actually stood for and how they have been used in different historical contexts. To interpret older diagnoses that are no longer used in modern medical terminology, we have used national reference literature such as contemporary medical dictionaries and published nomenclatures (Cronberg, 2018; Sundberg, 1926; Svensk författningssamling, 1860–1931;

Wernstedt, 1935, 1943, 1955, 1959). In practice, this can in some cases lead to similar archaic disease names being assigned different codes in different countries. This further emphasizes the importance of the development and validation of an international coding system for historic causes of death taking place in an international collaboration where consistent coding and comparability between datasets is sought. This is not to say at it is impossible or that the development of an international system is a fruitless mission. On the contrary, such a system is a valuable research tool, but we must recognize its limitations in order to be able to use it properly. In the following we address some issues with historical coding and classification of causes of death in relation to the analyses of the present article.

The first problem relates to the content in the historical sources regarding the specificity of the reported diagnoses. In the 1860s explicit causes of death were often missing or stated as "unknown", this was particularly the case for neonatal mortality. However, this did not occur randomly, instead it was more common in some age groups, in certain settings and in different times. Our experience with data from 19th century Sundsvall is that deaths among infants often lacked specific information on cause of death. What we can see, however, is that the frequency of specific and relevant diagnoses markedly increased over time. The way the specificity in the data shifts in time and space creates a challenge for comparative analyses.

Another problem with the content in historical sources concerns cases with vague diagnoses. These are often difficult and sometimes impossible to interpret in any meaningful way. So similar to the problem with missing information, we need to relate to the problem and try to understand the implications of it. A couple of examples of such diagnoses that were common for infants in many countries are "teething" and "convulsions". This was also the case in Swedish rural settings, but in the area under study here, these diagnoses were rarely used, and in the case of teething perhaps due to the fact that teething was not included in the cause of death nomenclature at the time and therefore not considered as a professional term by the late-19th-century physicians.

When it comes to research practice, the coded causes of death must be organised and classified according to a research purpose, for example with the aim of identifying the determinants of high mortality. In this article, we have used the InfantCat_2020 with a limited number of categories intended to be used specifically for diseases that caused death in infancy (Reid et al., 2024b). These categories seem reasonable on the whole but can of course always be discussed. With regard to the category of air-borne diseases, one could consider whether to separate pneumonia, bronchitis and other respiratory diseases from the group air-borne infections. Since the latter usually appear as recurring epidemics, a division into two groups would better capture epidemics of various kinds.

Another question concerns some more or less vague diagnoses that have been coded as symptoms, i.e. placed in Chapter XVIII in the coding scheme. This is the case with some of the diagnoses with the suffix fever. In 19th-century Sweden, the term "nervfeber" (in English *nervous fever*) could refer either to typhus or typhoid fever, but in most cases the latter. This illustrates one of the difficulties that arise when working with interpreting historical diagnoses, namely, how to deal with disease designations whose meaning is ambiguous. Fever ("feber") is another diagnosis that is placed in the symptoms chapter. This term was rarely used in Sundsvall at the time of our study, but in older times fever was often used during epidemics. A concentration of "feber" deaths therefore often indicates a ravaging epidemic. If we present causes of death while treating "nervfeber" and "feber" only as symptoms, we would miss important aspects of historical mortality. There are two possible ways to address this inadequacy. Either a decision is made already at the coding stage, i.e. one interprets the term and assigns a code that seems to match the historical coding scheme, or it is taken care of in connection with its use in research by moving the diagnosis to a more appropriate category. The best is to use the latter alternative, something that is facilitated by ICD10h. Any user of the classification system should consider possible necessary adaptions in order to the special historic and national context.

A good approach is to critically test and evaluate the validity of the code system by applying it to different types of research questions. It is clearly the case that the coding system that has been applied here, on 19th-century cause of death data for infants, works excellently as a common structure that makes data from the same time period more accessible for analyses of cause-specific mortality. Through a research-driven evaluation, the code system can continue to be developed and improved so that it provides the best conditions for comparative analyses of the health situation in historic Europe.

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Svensk författningssamling. (1911). Kungliga Medicinalstyrelsens cirkulär till samtliga läkare i Riket angående uppgifter om dödsorsaker, Bilaga 1. Dödsorsaksnomenklatur för ordnande och statistisk bearbetning av dödsbevisen [Circular from the Royal Swedish Medical Board to all physicians in the Kingdom regarding information on causes of death. Appendix 1. Cause of death nomenclature for the organization and statistical processing of death certificates]. Stockholm. *Svensk författningssamling, 58*,1–14. P. A. Norstedt & söner.

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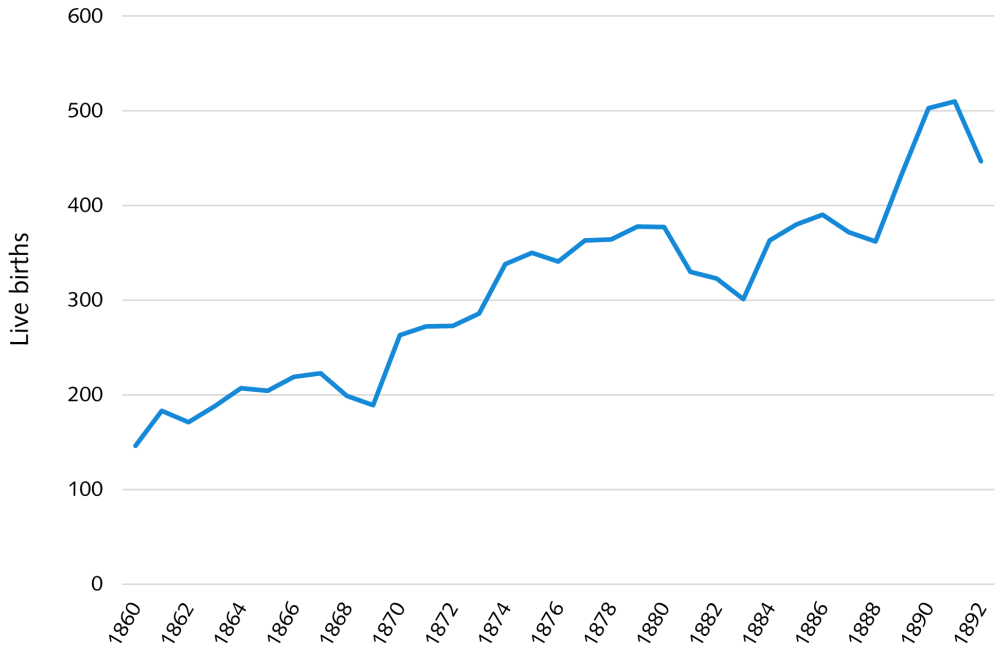
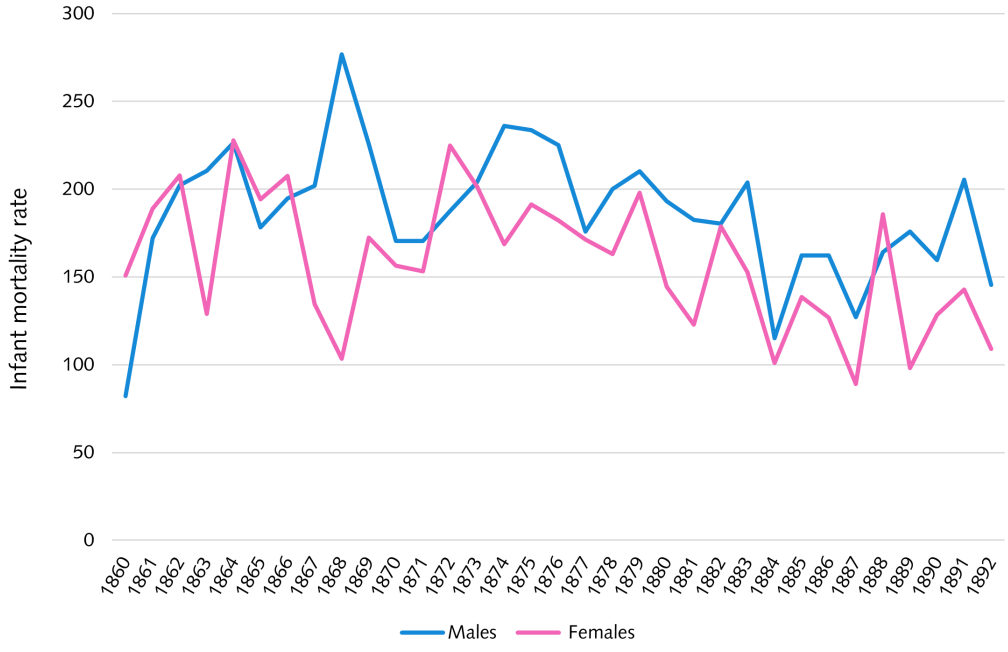
APPENDIX A

Figure A1 *The annual number of live births occurring in Sundsvall, 1860–1892*



Source: The data set was based on the POPUM database, parish of Sundsvall, 1860–1892 (Demographic Data Base, CEDAR, Umeå University, 2021).

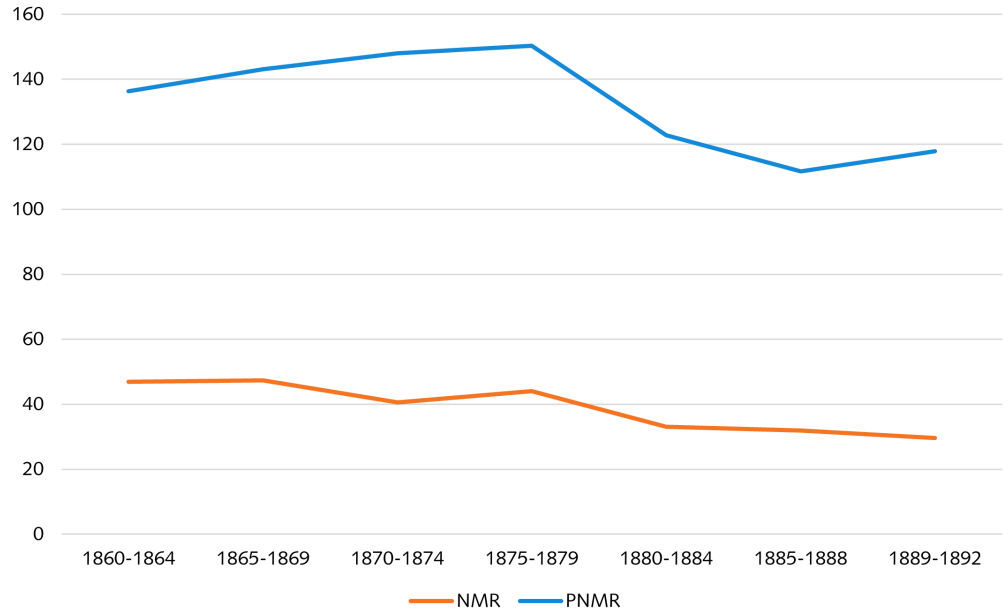
Figure A2 *Annual infant mortality rates, Sundsvall 1860–1892*



Note: A Students T-test calculated on the difference in mortality between males and females resulted in a P-value = 0,00044.

Source: As Figure A1.

Figure A4 *Quinquennial neonatal- and post-neonatal mortality rates, Sundsvall 1860–1892*



Note: The final period includes only four years.

Source: As Figure A1.

Table A2 *Quinquennial rates of IMR, NMR and PNMR, with % of infant deaths which were neonatal and post-neonatal, Sundsvall 1860–1892*

5-year period	IMR	NMR	PNMR	% of infant deaths neonatal	% of infant deaths post-neonatal
1860–1864	183	47	136	26%	74%
1865–1869	191	47	143	25%	75%
1870–1874	189	41	148	21%	79%
1875–1879	194	44	150	23%	77%
1880–1884	156	33	123	21%	79%
1885–1888	144	32	112	22%	78%
1889–1892	147	30	118	20%	80%

Notes: Infant mortality rates (IMR), neonatal mortality rates (NMR) and post-neonatal mortality rates (PNMR).The final period includes only four years.

Table A3 *Quinquennial infant mortality rates by causal group, Sundsvall 1860–1892*

Period	1860–1864		1865–1869		1870–1874		1875–1879		1880–1884		1885–1888		1889–1892	
Causal group	**IMR**	**N**	**IMR**	**N**	**IMR**	**N**	**IMR**	**N**	**IMR**	**N**	**IMR**	**N**	**IMR**	**N**
Congenital and birth disorders	5.6	(5)	7.7	(8)	16.8	(24)	16.7	(30)	10.6	(18)	17.3	(26)	18.5	(35)
Weakness	1.1	(1)	9.7	(10)	26.5	(38)	18.9	(34)	3.0	(5)	0.7	(1)	0	(0)
Convulsions	0	(0)	0	(0)	2.1	(3)	1.1	(2)	1.8	(3)	0.7	(1)	0	(0)
Water-food borne	22.3	(20)	39.7	(41)	41.2	(59)	60.1	(108)	62.0	(105)	59.2	(89)	57.1	(108)
Teething	1.1	(1)	0	(0)	0.7	(1)	0	(0)	0	(0)	0	(0)	0	(0)
Air-borne	36.9	(33)	30.9	(32)	44.0	(63)	44.5	(80)	48.4	(82)	43.9	(66)	47.0	(89)
Other infectious	6.7	(6)	1.0	(1)	11.2	(16)	2.2	(4)	1.8	(3)	4.7	(7)	5.3	(10)
Other non-infectious	29.1	(26)	27.1	(28)	19.6	(28)	15.0	(27)	8.9	(15)	8.6	(13)	6.9	(13)
External causes	1.1	(1)	0	(0)	0	(0)	1.1	(2)	1.2	(2)	0	(0)	0.5	(1)
Ill-defined	2.2	(2)	3.9	(4)	0	(0)	0	(0)	3.0	(5)	0.7	(1)	0.5	(1)
Stated unknown	73.7	(66)	57.1	(59)	22.3	(32)	31.2	(56)	2.4	(4)	0.7	(1)	0.5	(1)
Blank	3.4	(3)	13.5	(14)	4.2	(6)	3.3	(6)	13.0	(22)	7.3	(11)	11.1	(21)
Total	183.2	(164)	191	(197)	189	(270)	194	(349)	156	(264)	144	(216)	147	(279)

Notes: Infant mortality rate (IMR). Number of deaths (N). The final period includes only four years.

Table A4 *Quinquennial rates of neonatal mortality, by causal group, Sundsvall 1860–1892*

Period	1860–1864		1865–1869		1870–1874		1875–1879		1880–1884		1885–1888		1889–1892	
Causal group	**NMR**	**N**	**NMR**	**N**	**NMR**	**N**	**NMR**	**N**	**NMR**	**N**	**NMR**	**N**	**NMR**	**N**
Congenital and birth disorders	5.6	(5)	5.8	(6)	14.7	(21)	15.6	(28)	8.9	(15)	16.6	(25)	16.9	(32)
Weakness	0	(0)	6.8	(7)	5.6	(8)	6.7	(12)	2.4	(4)	0.7	(1)	0	(0)
Convulsions	0	(0)	0	(0)	1.4	(2)	1.1	(2)	1.8	(3)	0	(0)	0	(0)
Water-food borne	4.5	(4)	3.9	(4)	3.5	(5)	4.5	(8)	6.5	(11)	2.7	(4)	3.2	(6)
Teething	0	(0)	0	(0)	0	(0)	0	(0)	0	(0)	0	(0)	0	(0)
Air-borne	0	(0)	0	(0)	2.1	(3)	2.8	(5)	4.1	(7)	4.7	(7)	3.7	(7)
Other infectious	2.2	(2)	0	(0)	2.8	(4)	0.6	(1)	0.6	(1)	2.0	(3)	1.6	(3)
Other non-infectious	4.5	(4)	3.9	(4)	5.6	(8)	1.7	(3)	1.2	(2)	1.3	(2)	0.5	(1)
External causes	1.1	(1)	0	(0)	0	(0)	0.6	(1)	0.6	(1)	0	(0)	0	(0)
Ill-defined	1.1	(1)	0	(0)	0	(0)	0	(0)	0.6	(1)	0	(0)	0.5	(1)
Stated unknown	27.9	(25)	23.2	(24)	4.9	(7)	10.0	(18)	0.6	(1)	0	(0)	0	(0)
Blank	0	(0)	3.9	(4)	0	(0)	0.6	(1)	5.9	(10)	4.0	(6)	3.2	(6)

Notes: Neonatal mortality rate (NMR). Number of deaths (N). The final period includes only four years.

Table A5 *Quinquennial rates of post-neonatal mortality, by causal group, Sundsvall 1860–1892*

Period	**1860–1864**		**1865–1869**		**1870–1874**		**1875–1879**		**1880–1884**		**1885–1888**		**1889–1892**	
Causal group	**PNMR**	**N**	**PNMR**	**N**	**PNMR**	**N**	**PNMR**	**N**	**PNMR**	**N**	**PNMR**	**N**	**PNMR**	**N**
Congenital and birth disorders	0	(0)	1.9	(2)	2.1	(3)	1.1	(2)	1.8	(3)	0.7	(1)	1.6	(3)
Weakness	1.1	(1)	2.9	(3)	20.9	(30)	12.2	(22)	0.6	(1)	0	(0)	0	(0)
Convulsions	0	(0)	0	(0)	0.7	(1)	0	(0)	0	(0)	0.7	(1)	0	(0)
Water-food borne	17.9	(16)	35.8	(37)	37.7	(54)	55.7	(100)	55.5	(94)	56.5	(85)	53.9	(102)
Teething	1.1	(1)	0	(0)	0.7	(1)	0	(0)	0	(0)	0	(0)	0	(0)
Air-borne	36.9	(33)	30.9	(32)	41.9	(60)	41.8	(75)	44.3	(75)	39.2	(59)	43.3	(82)
Other infectious	4.5	(4)	1.0	(1)	8.4	(12)	1.7	(3)	1.2	(2)	2.7	(4)	3.7	(7)
Other non-infectious	24.6	(22)	23.2	(24)	14.0	(20)	13.4	(24)	7.7	(13)	7.3	(11)	6.3	(12)
External causes	0	(0)	0	(0)	0	(0)	0.6	(1)	0.6	(1)	0	(0)	0.5	(1)
Ill-defined	1.1	(1)	3.9	(4)	0	(0)	0	(0)	2.4	(4)	0.7	(1)	0	(0)
Stated unknown	45.8	(41)	33.8	(35)	17.5	(25)	21.2	(38)	1.8	(3)	0.7	(1)	0.5	(1)
Blank	3.4	(3)	9.7	(10)	4.2	(6)	2.8	(5)	7.1	(12)	3.3	(5)	7.9	(15)
Total	136.3	(122)	143.1	(148)	148.0	(212)	150.3	(270)	122.8	(208)	111.7	(168)	117.8	(223)

Notes: Post-neonatal mortality rates (PNMR). Number of deaths (N). The final period includes only four years.

Table A6 *Neonatal mortality rates, post-neonatal mortality rates and number of deaths by causal group and sex, Sundsvall 1860–1892*

Causal group	Male		Female		Male		Female	
	NMR	N	NMR	N	PNMR	N	PNMR	N
Congenital and birth disorders	14.6	75	11.2	57	1.4	7	1.4	7
Weakness	4.1	21	2.2	11	5.4	28	5.7	29
Convulsions	1.0	5	0.4	2	0	0	0.4	2
Water-food borne	5.3	27	2.9	15	51.6	265	43.6	223
Teething	0.0	0	0.0	0	0.2	1	0.2	1
Air-borne	2.9	15	2.7	14	43.0	221	38.2	195
Other infectious	1.2	6	1.6	8	3.3	17	3.1	16
Other non-infectious	2.1	11	2.5	13	14.4	74	10.2	52
External causes	0.2	1	0.4	2	0.4	2	0.2	1
Ill-defined	0.4	2	0.2	1	1.2	6	0.8	4
Stated unknown	9.0	46	5.7	29	15.4	79	12.7	65
Blank	3.1	16	2.2	11	5.3	27	5.7	29
Total	43.8	225	31.9	163	141.5	727	122.1	624

Note: Neonatal mortality rate (NMR), post-neonatal mortality rate (PNMR).

APPENDIX B

In this study all causes of deaths have been assigned to a ICD10h code (Reid et al., 2024a) and subsequently categorised in 12 causal groups according to the InfantCat2020 from Reid et al. (2024b). In cases where multiple causes were listed on the death record, we have prioritised the "underlying" cause by means of a set of prioritising rules provided to all SHiP studies participating in this special issue as described below.

A death for which there are several causes is first assigned an ICD10h code to each cause of death. To subsequently assign the infant death to only one of the causal groups that we use here, the following rules have been used to identify the "primary" cause of death and determine in which of the groups that death should be placed:

i. ignore symptomatic causes, unless they are the only causes given (e.g. causes of death assigned a code from ICD-10 Chapter XVIII);
ii. if one of the causes is an infectious disease, use this to categorise the infant death;
iii. otherwise take the first cause of death;
iv. if there are two or more infectious diseases listed, consider how they might have formed a chain of diseases which led to the death, and take the disease starting that chain.

In Table A1 infant deaths from Sundsvall have been assigned to the 12 causal groups, InfantCat2020, based on the ICD10h coding system used by all the SHiP studies contributing to this special issue.

Table B1 *Infant deaths per ICD10h and causal group in Sundsvall 1860–1892*

Causal group (InfantCat2020)	ICD10h	ICD10h description	Number of infant deaths	% of all infant deaths
air-borne	A16.200	Tuberculosis of lung	2	0,12
air-borne	A16.905	Tuberculosis	3	0,17
air-borne	A17.000	Tuberculous meningitis	6	0,35
air-borne	A18.201	Scrofula	1	0,06
air-borne	A36.000	Pharyngeal diphtheria	2	0,12
air-borne	A36.200	Laryngeal diphtheria	10	0,58
air-borne	A36.203	Membranous laryngitis	1	0,06
air-borne	A36.900	Diphtheria, unspecified	8	0,46
air-borne	A37.900	Whooping cough, unspecified	32	1,84
air-borne	A38.000	Scarlet fever	12	0,69
air-borne	A38.002	Scarlatina	7	0,40
air-borne	A38.003	Scarlatina with complications	1	0,06
air-borne	B03.000	Smallpox not known if vaccinated	31	1,78
air-borne	B05.201	Measles complicated by bronchitis or laryngitis	1	0,06
air-borne	B05.900	Measles, no mention of complication	17	0,98
air-borne	G03.900	Meningitis, unspecified	57	3,28
air-borne	J02.900	Pharyngitis	1	0,06
air-borne	J02.901	Sore, inflamed throat	1	0,06
air-borne	J04.000	Acute laryngitis	4	0,23
air-borne	J05.000	Croup	2	0,12
air-borne	J18.000	Bronchopneumonia	12	0,69
air-borne	J18.800	Pleuropneumonia	6	0,35
air-borne	J18.802	Bröstfeber	4	0,23
air-borne	J18.900	Pneumonia	97	5,58
air-borne	J20.900	Bronchitis acute	22	1,27
air-borne	J21.901	Capillary bronchitis	82	4,72

air-borne	J36.001	Quinsy	1	0,06
air-borne	J40.009	Bronchitis, unspecified no age or duration	21	1,21
air-borne	R09.101	Inflammation of lungs	1	0,06
congenital and birth disorders	J98.100	Pulmonary collapse	4	0,23
congenital and birth disorders	P07.300	Prematurity, infant	83	4,77
congenital and birth disorders	P38.000	Omphalitis of newborn	1	0,06
congenital and birth disorders	P96.903	Congenital debility	45	2,59
congenital and birth disorders	Q03.900	Congenital hydrocephalus, unspecified	2	0,12
congenital and birth disorders	Q05.900	Spina bifida, unspecified	6	0,35
congenital and birth disorders	Q24.900	Congenital heart malformation	1	0,06
congenital and birth disorders	Q76.400	Other congenital malformations of spine, not associated with scoliosis	1	0,06
congenital and birth disorders	Q89.900	Congenital malformation, unspecified	3	0,17
convulsions	R56.800	Convulsions	9	0,52
external causes	T14.900	Injury, unspecified	1	0,06
external causes	W75.000	Accidental suffocation and strangulation in bed	1	0,06
external causes	W84.000	Suffocation NOS	2	0,12
external causes	Y09.002	Murder	1	0,06
external causes	Y20.002	Suffocation, undetermined intent	1	0,06
ill-defined and unknown	R50.900	Fever unspecified	2	0,12
ill-defined and unknown	R50.905	Nervfeber	3	0,17
ill-defined and unknown	R60.900	Oedema, unspecified	1	0,06
ill-defined and unknown	R99.000	Unclear	7	0,40
stated to be 'unknown'	R99.001	Stated to be 'unknown'	219	12,59
no cause given/blank	R99.090	No cause given	83	4,77
other	G91.900	Hydrocephalus	16	0,92
other	G93.600	Brain oedema	1	0,06
other	G93.900	Brain disease, unspecified	1	0,06
other	G95.900	Disease of spinal cord, unspecified	1	0,06
other	I21.904	Heart apoplexy	2	0,12
other	I30.900	Acute pericarditis, unspecified	1	0,06
other	I46.900	Cardiac arrest	7	0,40
other	I51.900	Heart disease, unspecified	4	0,23
other	I61.900	Brain haemorrhage	1	0,06
other	I64.003	Slag	9	0,52
other	J39.200	Disease of pharynx, throat	2	0,12
other	J40.000	Bronchitis, catarrhal	13	0,75
other	J42.000	Bronchitis, chronic	7	0,40
other	J98.400	Lung disease, unspecified	8	0,46
other	J98.404	Chronic pneumonia	1	0,06
other	J98.900	Chest disease, unspecified	16	0,92
other	J98.901	Catarrh	3	0,17
other	K08.900	Disorder of teeth and supporting structures, unspecified	11	0,63
other	K52.900	Noninfective gastroenteritis and colitis, unspecified	8	0,46
other	K59.900	Functional intestinal disorder, unspecified	1	0,06
other	K63.900	Bowel disease	7	0,40
other	K92.905	Various disorders of digestive system	4	0,23
other	L02.900	Abscess, unspecified	1	0,06

other	M86.909	Caries	1	0,06
other	N12.000	Tubulo-interstitial nephritis, unspecified	3	0,17
other	N61.000	Breast inflammation	15	0,86
other	O15.900	Eclampsia, puerperal	2	0,12
other	R09.301	Phlegm	1	0,06
other	R17.000	Jaundice	1	0,06
other	R21.000	Rash	1	0,06
other	R22.901	Inflammation, unspecified	1	0,06
other infectious	A33.000	Tetanus neonatorum	2	0,12
other infectious	A41.905	Septicaemia, unspecified	3	0,17
other infectious	A46.000	Erysipelas	4	0,23
other infectious	A50.900	Congenital syphilis	11	0,63
other infectious	A53.900	Syphilis, unspecified as late or early	5	0,29
other infectious	A64.000	Venereal disease unspecified	1	0,06
other infectious	B37.900	Candidiasis (thrush), unspecified	7	0,40
other infectious	G04.900	Encephalitis	14	0,81
teething	K00.700	Teething (diarrhoea)	2	0,12
water-food borne	A00.900	Cholera	5	0,29
water-food borne	A01.003	Gastric fever	1	0,06
water-food borne	A03.900	Shigellosis, unspecified	1	0,06
water-food borne	A09.000	Colitis, infective origin	25	1,44
water-food borne	A09.001	Infantile cholera/summer diarrhoea	9	0,52
water-food borne	A09.002	Diarrhoea	42	2,42
water-food borne	A09.004	Gastric catarrh	222	12,77
water-food borne	A09.008	Gastroenteritis	1	0,06
water-food borne	A09.009	Enteritis	3	0,17
water-food borne	K29.000	Acute haemorrhagic gastritis	7	0,40
water-food borne	K29.500	Chronic gastritis, unspecified	58	3,34
water-food borne	K29.700	Gastritis, unspecified	132	7,59
water-food borne	K30.000	Dyspepsia	4	0,23
water-food borne	K52.300	Colitis, non-infective	20	1,15
weakness	R53.003	Debility	27	1,55
weakness	R64.003	Atrophy	49	2,82
weakness	R64.004	Tvinsot	13	0,75
Total N of infant deaths			**1739**	**100,00**

Source: The data set was based on the POPUM database, parish of Sundsvall, 1860–1892 (Demographic Data Base, CEDAR, Umeå University, 2021).