

What was Killing Babies in Trondheim? An Investigation of Infant Mortality Using Individual Level Cause of Death Data, 1830–1907

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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 Trondheim?

An Investigation of Infant Mortality Using Individual Level Cause of Death Data, 1830–1907

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ABSTRACT

This paper examines infant mortality amongst newborns in Trondheim city, 1830–1907, working specifically with individual level cause of death data. Findings show that infant mortality in the city started to drop from 1895, primarily as a result of a decline in post-neonatal mortality. At the start of the decline air-borne diseases accounted for nearly half of the deaths, and water-food borne for around one third. The drop was predominantly driven by a decline in these two causal groups, and seasonal fluctuations became less pronounced. Because of the fall in post-neonatal mortality, the relative risk of dying amongst neonates rose towards the end of the period. Although 'convulsions' accounted for 50–70% of all infant deaths between 1830 and 1860, this cause had faded away to near insignificance by the beginning of the 1900s. Here we aim to assess the extent to which this particular aspect of decline can be explained by alterations to official instructions regarding registration and in registration practice itself. This article proposes that the decline in deaths from 'convulsions' can be explained by a relabelling of such deaths into 'congenital and birth disorders' amongst neonates, and a mix of 'water-food borne' and 'air-borne diseases' amongst post-neonates. This argument is supported by the fact that the timing of the decline corresponds with the introduction of cause of death certificates issued by medical practitioners, and which most likely resulted in fewer causes of death being reported by lay informants who could only offer vague symptoms rather than informed diagnoses.

Keywords: Infant mortality, Causes of death, ICD10, 19th Century Norway, Convulsions, Causes of death registration practice

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1 INTRODUCTION

This paper explores the causes of death registered for infants in the city of Trondheim, Norway, between 1830 and 1907. It contributes to a special issue on causes of infant deaths in European port cities in the late 19th century where each paper shares a standard analytic approach and uses the same historical cause of death coding scheme (ICD10h).¹ The first part of the paper introduces the city, giving particular attention to its demographic development, the growing awareness of the need for measures to improve the health of the population, and the actions taken. Section 2 discusses the data, which are derived from the city's burial books. A principal aim of this section is the investigation of the role of priests as registrars of death, including cause of death. Section 3 presents mortality rates amongst infants, neonates and post-neonates by 12 pre-defined groups of causes. Age group specific seasonality in the 3–4 groups of causes responsible for the greatest toll of infant deaths is also examined. This section also includes a discussion of changes in the official instructions regarding cause of death registration and classification. How did such changes affect registration practices, particularly the use of terms describing symptoms rather than a specific underlying cause? A case study of the use of the term 'convulsions', which accounted for 50–70% of infant deaths in the years before the early 1860s, but only 5% by the end of the study period, is presented. The fourth section brings the paper to a close with a discussion of the findings.

Compared to infant mortality rates in other countries at the time, infant mortality rates in 19th century Norway were low. In 1840, 140 of the country's infants died out of each 1,000 live births. In the 1870s this figure began to ebb slowly, before dropping quickly from the late 1890s onwards (Backer, 1961). Nevertheless, the urban penalty was a well-known phenomenon even in this least deadly of European countries (Hubbard, 2002). Between 1876 and 1905 Norway's cities experienced infant mortality rates around 50% above those in rural areas. In the mid-1870s Trondheim was the most salubrious city, but by 1885 its infant mortality rates were second only to those of Christiania, as Oslo was then known (Backer, 1961; Hubbard, 2002).

In Norwegian historiography, there is a broad agreement that 19th century public health measures such as vaccination programs, a focus on stillbirths, the introduction of education for midwives in 1810, and the establishment of health boards for each municipality, all contributed to the country's relatively low infant mortality levels (Sogner, 2000). However, what is much less clear is what initiated the fall in mortality amongst infants, although both the role of doctors and midwives (Dyrvik, 1997; Fure 2002) and the extent of improved health amongst mothers (Fure, 2002; Sommerseth, 2018) have been suggested. The majority of micro-data studies of infant mortality in 19th century Norway have focused on rural areas, where deficient registration of cause of death by the local priest is a common feature (Sommerseth & Thorvaldsen, 2022). In comparison, church books in the cities tend to have nearly complete cause of death registration. This paper is, we believe, the first to explore long-term infant mortality by individual level cause of death in a large Norwegian city during a period which saw considerable population growth, heightened public awareness of the need for measures to improve health, and increasing action to implement these.

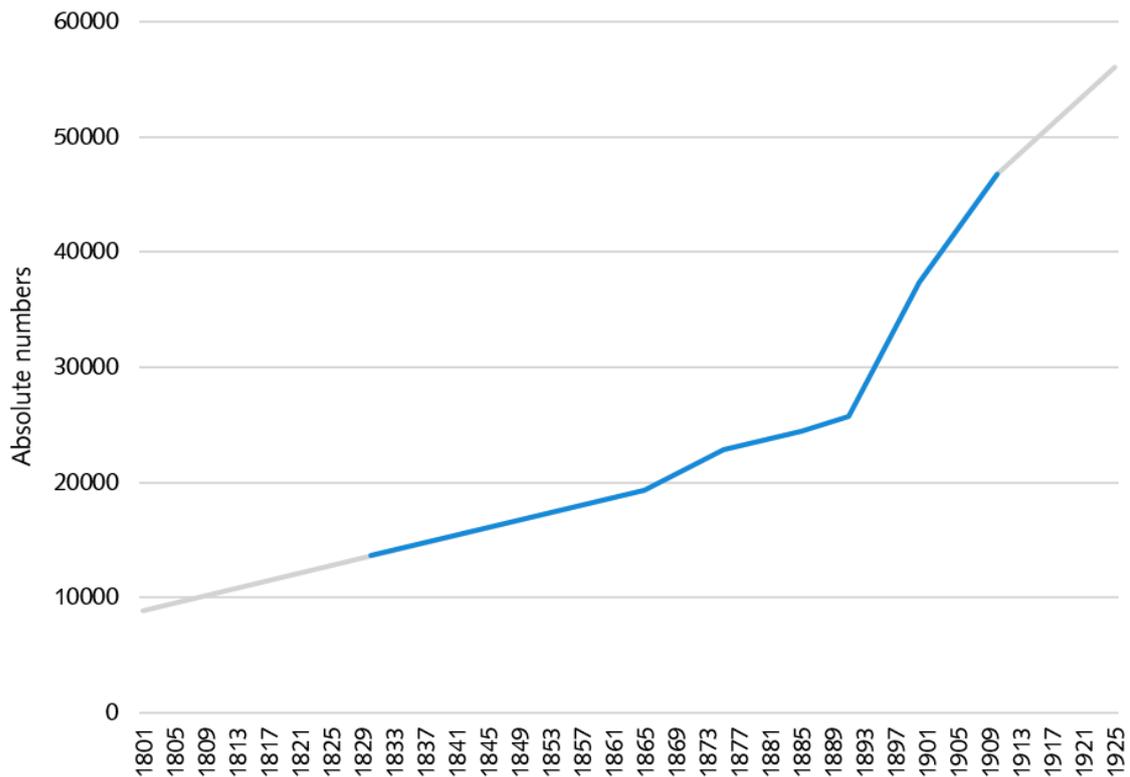
1.1 TRONDHEIM CITY

As it is today, Trondheim was the third largest city in Norway during the period covered by this study. It lies on a sheltered peninsula on the southern shore of the deeply indented Trondheim fjord, about 37 km southeast of the Norwegian Sea. By the beginning of the 19th century, exports of fish, timber and copper combined with mining, shipping, and the shipbuilding industry were mainstays of the city's economy, and this gave a vibrant, crowded atmosphere to the harbour area. Over the course of the 19th century, Trondheim became an economic centre and most of the goods entering and leaving the surrounding region had to pass through the city.

As shown in Figure 1, the population of the city increased from about 14,000 inhabitants in 1830 to 46,200 in 1910. A number of extensions to the city boundaries contributed to this increase. The two greatest extensions took place in 1864 and 1893. The first of these extended the city borders in the east and about 2,000 individuals were added to the city's population. In the chief medical officer's view, all these new citizens contributed with nothing but misery and want (NOS, 1866, pp. 129–138). In 1893 the city extended its borders further to the east, but also to the south and west. The extensions meant that while 13% of the city's population lived in its eastern area in 1845, by 1900 56% did so, making it the most crowded district of the city.

¹ For an overview of the ICD10h, see the introduction paper of the special issue.

Figure 1 Population growth in Trondheim, 1801–1920 (absolute numbers; blue line = study period)



Source: Norwegian Historical Data Centre, UiT the Arctic University of Norway, *Historical Population Register of Norway, Population censuses, Trondheim, 1801–1920*. Original sources at the National Archive of Norway.

As political and economic ideas developed at unprecedented speed, bringing unforeseen opportunities for profit both in Trondheim and beyond, it also brought increasing social inequality and growing awareness of the detrimental impacts this was having. Old bourgeois families and the new middle classes, who lived primarily in the city centre, gained their knowledge of the living conditions of the fast-growing working classes, who lived around the urban periphery, from a safe distance — through the pages of the city's newspaper, *Dagsposten*. In August 1888, an article described conditions in the eastern suburbia in very unflattering terms. The houses were portrayed as being made of planks thrown together any old how, with virtually no materials to seal the gaps, and panelled inside with boards that were not properly dried, or even just cardboard. The streets were full of mud and manure, families had to fetch tainted water from local wells, and sewage systems were non-existent. This picture contrasted unfavourably with the improvements that had been made in the public health infrastructure within the inner part of the city. These had been carried out under the Public Health Act of 1860 by the city's health board. Each of Norway's municipalities had a health board which was primarily responsible for implementing measures to reduce environmental threats to hygiene and preventing the spread of infectious diseases. Amongst their key targets were the improvement of sanitation conditions in outdoor spaces, drinking water quality, food hygiene, and the air quality in homes and public buildings (Schjøtz, 2010; Storesund, 2005).²

Early in 1860 sewers and a drainage system began to be built in the city, and by 1887 the chief medical officer was able to conclude in his medical report the "entire" inner city was now covered (NOS, 1889). While these measures were taken because city council recognized they had to take greater responsibility for public health, the provision of a water supply, — which eventually saw water piped into every house — initially grew out of their wish to improve the city's firefighting capability. Two devastating fires had hit the inner city at the beginning of the 1840s. The first, in 1841, saw 330 apartment buildings and 39 piers in the harbor burnt down, and then, a year later, a second fire led to some 3,000 people being left homeless in mid-winter. By 1863, the city had a new high-pressure water system, allowing water

2 Since the 1830s, the city's medical officers had accumulated knowledge about implementing temporary measures to prevent cholera. However, with the Public Health Act of 1860, this mandate was extended to also apply to permanent and long-term public health work.

to be piped into the houses. Nevertheless, despite water pipes gradually becoming more common in houses in central Trondheim, in the 1880s many households were still using old wells which lay in the vicinity of "sink wells and waste pits", as latrines were called (Røskaft & Østby, 2021).

In 1883, Trondheim's health board received budget approval to employ two *assistentter* or inspectors to examine outdoor privies, and the sewer and drainage systems. According to their job description, the inspectors were also to inspect rental housing, possibly reflecting the health board's concerns for the poorest families in the city. This concern also led them to appeal to the city council to impose a ban on the renting out of sub-standard, one-room dwellings in damp basements or outbuildings (Marstein, 1999). In addition, in 1890, the council employed a private company to undertake the renovation of all public buildings; they would also work on private apartment buildings if requested by a landlord. In 1892, with the threat of cholera looming, the city was divided into three districts, each with its own inspector from the health commission to ensure that all apartment building complied with the mandatory hygienic and sanitary standards measures. The health board's permanent staff of city doctors and inspectors also kept boarding houses, slaughterhouses, milk- and victualling shops, dining rooms, etc. under strict surveillance. Interestingly, these measures were kept in place and became a regular part of the city's public health work even after the threat from cholera had subsided. This supervision was meticulously documented and submitted as part of the annual medical report compiled by the city health board, which today gives us a unique insight into the city's public health priorities (NOS, 1892–1907).

After the city was extended in 1893, the city's health board faced new challenges. Each year the medical report highlighted the problematic hygienic and sanitary conditions in the new districts, noting the competition for city funds between a much-needed update of the sewerage system in the old inner city and the shocking conditions in the eastern outskirts. Some improvements to the drains and sewers were made in 1897, and by 1899 every house in the city was connected to a water pipe (NOS, 1900; NOS, 1902). Another major concern was the lack of housing. Despite considerable building activity, families arriving in the city had few options other than to rent a small and draughty basement apartment. When, in 1899, it was made obligatory to build in brick or stone, all construction stopped. As a result the already severe housing shortage became much worse, and living conditions deteriorated even further, particularly for new arrivals (NOS, 1903, pp. 210–236). The situation appears to have been at its worst in 1901; thereafter building activity increased again (NOS, 1904–1907).

Under the 1860 Public Health Act, annual statistics on the number of medical doctors, midwives, dentists, and vaccinators were compiled for all Norwegian cities and rural areas. The population census taken in 1865 estimated that there were 14 medical doctors (one per 1,400 of the population) and 10 midwives (one per 550 women aged 18–49) in Trondheim. The city authorities clearly did not manage to improve medical coverage in line with the enormous population growth; by 1910 there was only one doctor for every 1,500 patients. Admittedly the number of midwives had increased three-fold, so that there was one midwife for every 430 women aged 18–49.

1.2 DATA

The information in the Trondheim-database has been extracted from the Historical Population Register of Norway and covers the period 1830–1907 (Sommerseth & Thorvaldsen, 2022). It includes data from the baptism and burial registers recorded by the priests in each of the city's parishes, yielding a total of 50,461 baptisms and 6,820 infant burials. This gives a mean of 647 live births per year, with a sex ratio of 104, and 88 infant deaths per year on average. About 98% of the city's population belonged to the Church of Norway and most of the remainder were members of organized dissenting congregations (Danielsen, 1997). Under the Dissenter Act of 1891, those heading dissenting congregations were ordered to keep their own registers; previously they had reported all vital events to their local Church of Norway priest. Unfortunately, the database does not include dissenting congregations after 1891. In addition to dates of birth and death, and the sex of the child, the main variable of interest is the causes of death recorded in the burial registers.

In Norway, historical cause of death data originate from two different sources: the individual level data entered in the burial registers, and the yearly aggregates published by the Ministry of Health. Over the period covered here there were changes in the way causes of death were recorded in the two sources and in the following paragraphs I will discuss this evolution, paying particular attention to how it developed in the cities. We need to ask two questions: why did cause of death appeared in the church

registers of burials and what type of instructions were given to the priests filling in the registers, who would have had little medical training.

For centuries, Church of Norway registers have had a twofold agenda. As well as recording baptisms and burials back to the late 17th century, an early 18th century act instructed each parish priest to report the annual numbers of births and deaths in his parish to the authorities as the basis for population statistics; the reporting of marriages was implemented later (Backer, 1947). In addition, from 1794 the Church contributed to the earliest survey of the extent of infectious diseases or, to be more precise: "scurvy, venereal or other dangerous infectious diseases". The statistics were reported annually, and the reports also contained a description of the public health precautions that were taken and the regulations with which doctors, priests, and landowners were expected to comply (Thurmann, 1851).

Less than a decade later, in 1803, the annual reports became the responsibility of the medical profession, rather than the priests. As well as reporting six other aspects of health and health services the doctors had to document "which diseases that have raged the most, particularly smallpox, itch, venereal disease and *radesyge*"³ (Schjønsby, 2005). In the following years, we find numerous complaints from the central authorities that the reports were incomplete. Around 1820 there were about 100 general medical practitioners in Norway, and given that there were four times this number of clergymen, the practical solution was to ask the priests to once again record additional information in their well-organised church registers. From 1820 onwards, therefore, church registers included a specific field in which the priest had to report "Om død af Smitsom Sygdom, og da hvilken, eller ved ulykkelig hændelse" [If [the deceased was] dead by infectious disease, the name of the disease, or if by an accident].

In the following years, Norway's leading medical journal, *Magazin for Lægevidenskaben*, included numerous discussions concerning the need for medical statistics. Most of these discussions were addressed in the published minutes from the frequently held meetings of Christiania's Medical Society. Clearly inspired by the statistical congresses held in Brussels in 1853 and in Paris in 1855, three main concerns were addressed. First, the current annual medical reports were considered useless in the battle against ongoing epidemics as they were not collated and published until it was too late (NML, 1855). Secondly, the cause of death statistics presented in the annual reports from 1853 onwards were of poor quality; the first report for example only covered about 20% of all deaths. Thirdly, if the statistics were to be used by doctors, it was important that the central authorities in different countries worked together, following an agreed set of principles, in order to produce comparable statistics (Levi, 1854; NML, 1855).

A solution to the first of these concerns, which would also partly address the second, was proposed during one of the Medical Society's meetings in the spring of 1855 (NML, 1855, pp. 116–117). It was noted that experience of publishing lists of data on a weekly basis had been gained during the severe cholera outbreaks in 1853, so, as early as the autumn of 1855, monthly rather than annual reports of the *constitution epidemica* were published for each of the medical districts of Christiania. These reports contained aggregated counts of both morbidity and mortality by sex. The experience of collating these reports formed a valuable basis for the next steps in the improvement of cause of death registration. However, it was nearly five years before those steps were taken between January 1860 and January 1861.

Undoubtedly, the Public Health Bill presented to the Norwegian parliament in 1859, which recommended measures to deal with epidemic and contagious diseases and that permanent health boards for each municipality should be set up, was pivotal to the setting up of a national registration system. However, improving the system for the collection of cause of death appears to have been a challenge. As Director of the government's Medical Office, and the Secretary responsible for guiding the Public Health Act through Parliament, Christian Thorvald Kierulf, stated:

There are still many doctors who either do not, or only very incompletely, report deaths known to them from their practice. [...] The reports are heterogeneous in that some use Latin, others Norwegian disease terms, some state sex and age as well as the day or month of the death, others do not. Due to this incompleteness and great disparity, it has not yet been possible to think of creating age columns in the yearly medical reports, and even the sex column is still useless. Furthermore, the cause of death statistics have the disease terms arranged alphabetically, as it is impossible to collect them into any system. (NML, 1860, pp. 447–449).

3 Translates as a 'crippling skin disease', see Kveim Lie (2007).

One promising system or registration, recently implemented in Swedish cities, was to have parish priests, whenever they were notified of a death, request a certificate stating the cause of death signed by a medical practitioner. As almost all deaths were buried in their local churchyard, this made it easier to compile a continuous series of statistics, contributing to more reliable annual measures. The discussion following the suggestion that this system should be followed in Norway, provides a rare glimpse into contemporary medical notions regarding registration practice and the quality of cause of death data which could be obtained from the "church books", i.e. the death registers. Although the form on which priests reported the annual number of births and deaths occurring in their parish did not require the priests to report causes of death, priests in Christiania had done so for quite some time, although in many cases, the vague terms used had undoubtedly been given to the priest by whichever family member had informed him of the death. This was also true in other places, as one member of the Society emphasised: "In several places, causes of death are supposedly registered in these books, as far as they were known, but the registration is done by unskilled people, and is thus very unreliable" (NML, 1860, pp. 447–449). Kierulf was far more optimistic, as he had few doubts that even in the rural districts, medical practitioners could, in collaboration with the priests and the deceased's relatives, provide reliable information about the cause of death, even if they themselves had not attended the deceased during his or her last illness (NML, 1860, pp. 447–449). One solution to the issues outlined above, was presented by Kierulf during a Medical Society meeting in early November 1860. He had been working on a publication to provide members of Norway's health boards with guidance so that all involved had the same priorities and were aware of the public health regulations laid out in the newly published Public Health Act of 1860. His creative suggestion was that, in order to avoid bureaucracy, he would add a chapter about disease and cause of death statistics to his guide that would then be distributed to the health boards. The additional chapter initially applied only to the cities.⁴

Kierulf's guide to death registration reflects the work done by the Medical Society in Christiania. One particular point is of great importance to our understanding of the way cause of death was to be recorded in the burial register. Within four days of the end of each month, or within eight days of the end of each quarter, the priest had to forward the medical cause of death certificates which he had collected to the Medical Officer of Health who then compiled lists of infectious diseases and cause of death statistics for the city. In addition to the deceased's full name, occupation, marital status, address, age and death date, each certificate recorded both the primary and secondary causes of death. If no doctor was present at the death, one was supposed to be appointed to determine the cause of death. The notification of cause of death was not considered to be complete unless signed by a medical practitioner (NML, 1861, pp. 709–711).

In conjunction with the publication of Kierulf's guidance, the Ministry of the Interior issued a circular to all doctors which stated clearly that several measures had to be put in place to make cause of the death statistics more detailed and comparable. The most crucial of these was the introduction of a standardized nomenclature of diseases. The circular defined a total of 122 causes of death and provided instructions on which Norwegian and Latin terms should be used to describe them (NML, 1861, pp. 460–465). Again, as in the preparatory work for the monthly *constitution epidemica* and the development of the death certificate, a committee of the Christiania Medical Society was appointed to present a first draft of the nomenclature. In undertaking this work, the Committee emphasised, they sought to avoid the level of detail that characterized the lists proposed by Farr and D'Espine at the international Statistical Congresses. Instead they followed the recent approach taken in Sweden, which was to make the death certificate short and simple and to organise the causes of death roughly according to whether they occurred in epidemic, sporadic or endemic form. They did not, however, adopt the category headings used in the Swedish nomenclature. Stillbirths were not included in the Norwegian nomenclature as the relevant statistics were compiled mainly by midwives, and deaths from accident or violence were less detailed than in the Swedish version (NML, 1861, pp. 283–284).

Furthermore, when an extensive revision of the church books was undertaken in 1877, the old instruction from 1820 to state if the "death was caused by an infectious disease, [if so] name the disease, or [if caused] by an accident" was replaced with the heading *Opgiven Dødsarsag*, indicating that the "cause of death [stated on the certificate]" should be entered. As collaboration between the Church and the medical profession was well established by this date, this change in wording, handed down from the Ministry of the Interior, was designed to further improve the medical statistics. This revision did not result in any change in the causes of death appearing in the burial books of Trondheim, since they had included

4 Similar system was made compulsory in rural areas in 1892, see Backer (1947).

"cause of death given" since the early 19th century. Finally, another major revision of the nomenclature was introduced in 1896, when the causes of death were organized in groups partly defined by aetiology and partly by the organ, or organic system, most affected by the disease (NML, 1897, pp. 110–128).

To sum up, prior to 1861 there was no official guidance in Norway as to how cause of death should be entered in the burial registers other than the relevant column title, which emphasised deaths from infectious disease and external causes. Even in this period, however, it was generally the practice in Trondheim to register all causes of death, although it was reported that many of the causes of deaths in both rural and urban areas were provided by lay informants. In 1861, the causes of death registered were standardized through the use of a newly issued nomenclature; a classification system which stood unchanged until 1896. From 1861 onwards, priests' offices across Norway's cities became hubs for the collection and collation of death certificates each month. The system of delivery of monthly reports and each month's crop of death certificates to the local health board, was implemented in Trondheim in 1862 (NOS, 1863, pp. 92–98). From then on, the causes of death reported in the burial registers of Trondheim and other Norwegian cities, were largely defined by medical practitioners.

2 INFANT MORTALITY OVERVIEW

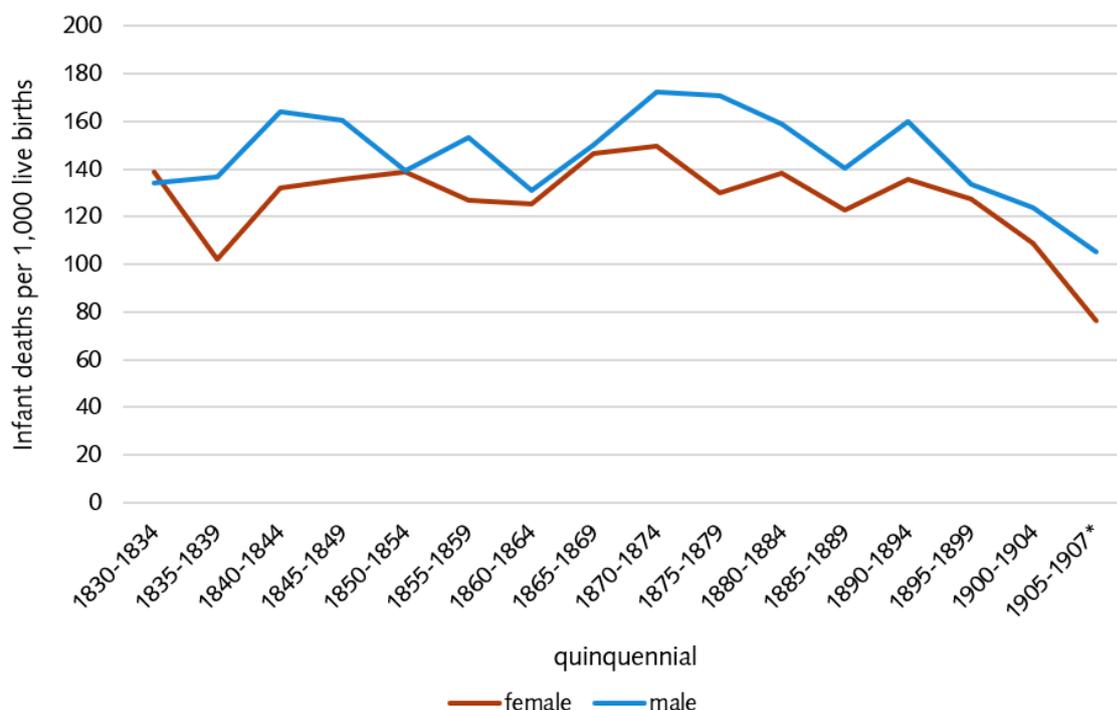
Over the 78-year period of this study, the annual absolute number of infant deaths reported in Trondheim varied considerably, from 12 deaths in 1830 to 216 in 1901. On the one hand, these variations mirror the increased number of births resulting from the steady population growth in the city, which meant that there were more newborns at risk of dying shortly after birth. On the other hand, and as shown in appendix A3, the infant mortality rate varied from one year to the next; in some years it was as "low" as 60 deaths per 1,000 births while in other years more than 25% of infants died. Such marked fluctuations became rarer from around the 1870s, and from the mid-1890s mortality started to decline. It should be borne in mind that the fluctuations up until the 1870s could be explained by smaller numbers (see appendices A1 and A2). However, the major peaks that can be discerned in 1840, 1865, and 1870 in appendix A3 were, according to the annual medical reports, the result of multiple epidemic outbreaks occurring either in combination or in series across the year.

Smallpox and whooping cough seem to have been epidemic in Trondheim in 1840. In the medical report for 1865, the city's health was described as bad, with no less than six epidemics — scarlet fever, whooping cough, typhoid fever, pneumonia, malignant sore throat, and diarrhoea — all sweeping through the population between January and June, with respiratory diseases frequently present throughout the year. 1870, which was the last year in which the infant mortality rate rose above 250, saw epidemics of measles, diarrhoea, and *cholera nostras*. In addition, as in 1865, respiratory diseases were more frequently seen than in other years. More than half of all infant deaths during these most lethal of years were related to outbreaks of epidemic disease. One way to assess the impact these outbreaks had on levels of, and trends in, infant mortality, is to calculate rates as 5-year moving averages, as shown in appendix A3. Interestingly, none of the major peak years contributed to a significant short-term increase in the mortality rate, thanks to the substantial drop in the mortality rate that followed each peak. Looking at the trend over time in appendix A3, however, one could argue that while the mortality rates continued to show large fluctuations, the prospect of a prolonged reduction in the mortality rate remained limited. Once the mortality rate started to drop the annual variation became less pronounced.

Figure 2 shows quinquennial infant mortality rates by sex in Trondheim between 1830 and 1907. The figure shows the expected excess male mortality across the period, although this is not statistically significant.⁵ Up until 1860–1864 infant mortality fluctuated between 120 and 140 per 1,000 live births, from then until 1890–1894 mortality was slightly higher, especially amongst boys. From 1895 onwards, mortality began to decline although, according to the official statistics, it would not fall below 60 deaths per 1,000 live births until 10–15 years after the end of this study.

⁵ Based on confidence intervals of 95%.

Figure 2 Quinquennial infant mortality rates by sex, Trondheim 1830–1907



* The final period includes only three years.

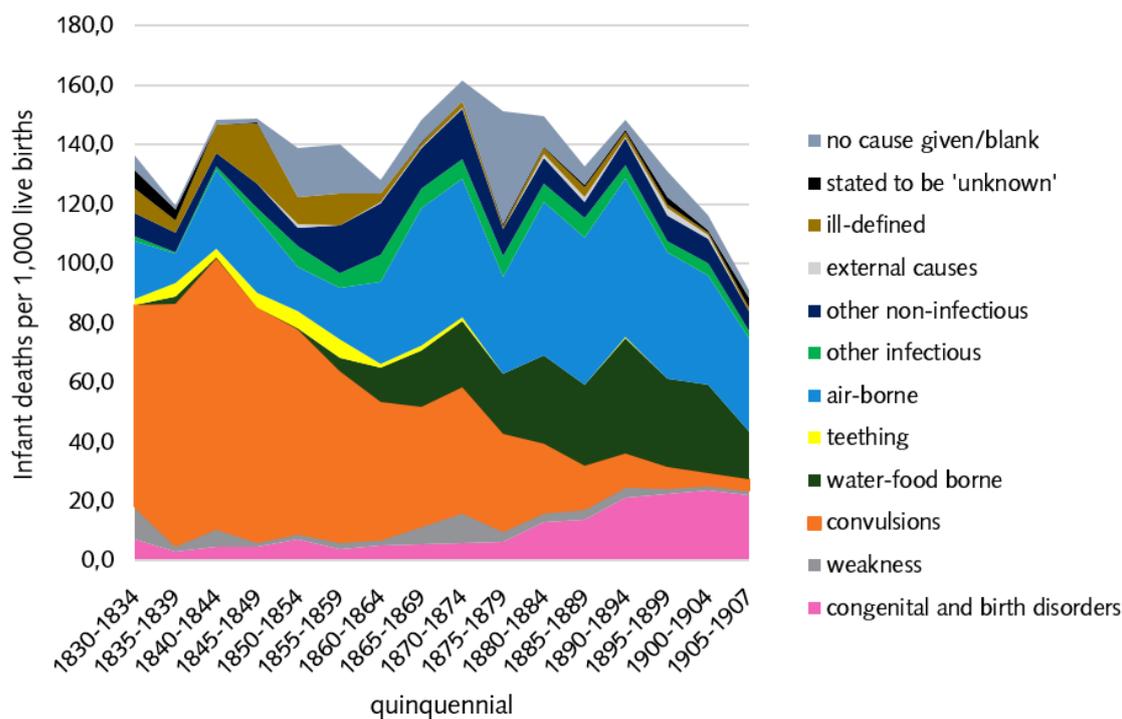
Source: Norwegian Historical Data Centre, UiT the Arctic University of Norway, Historical Population Register of Norway, Church books, Trondheim, 1801–1920. Original sources at the National Archive of Norway.

3 CAUSES OF DEATH

In Figure 3, infant deaths in Trondheim have been allocated to 12 cause of death categories, based on the ICD10h coding system. Unlike the International Classification of Disease (ICD), the standard tool for classifying diseases in current-day societies, ICD10h gives more room to causes of death found in historical sources. Of particular interest in this special issue on infant mortality are the historical causal groups of 'teething', 'convulsions', and 'weakness'.

The main causal groups displayed in Figure 3 are 'convulsions', 'air-borne', 'water-food borne', and 'congenital and birth disorders'. The topmost cause in the graph, 'no cause given', represents those cases where the priest left the cause of death blank in the burial register. Peaks in missing causes of death occurred in 1850–1859, when approximately 17% were missing, and in 1875–1879 when 37% were missing. During the first of these periods, the instruction in the register clearly stated that the priest was to register the cause of death only if it was related to an epidemic disease or an accident. A review of all the registers in Trondheim shows that while some priests registered a cause of death for nearly all the deceased, others just registered whether or not death was caused by an accident. In 1875–1879, the majority of missing information is the result of events in a single parish. Suddenly, after a funeral in July 1876, the priest of this parish stopped entering causes of death in the register, despite having faithfully done so on the previous 280 pages. This state of affairs continued, apart from a couple of registered accidents, until November 1877, when the priest once again began to register deaths after a new register was sent with the new registration guidelines issued that year. The new instructions stated that 'cause of death given' was to be registered, and from that point onwards almost every death registered in Trondheim included a medically certified cause.

Figure 3 *Quinquennial infant mortality rates by causal groups, Trondheim 1830–1907*



Source: See Figure 2.

Note: For figures underlying this graph see Appendix A4.

During the two first decades shown in Figure 3, 'water-food borne diseases' hardly appear as a cause of death. From the mid-1860s, however, they emerge as the third most frequent causal group. From then on, relative to all other causal groups, the proportion of the city's infants dying of 'water-food borne diseases' increased until it peaked in 1890–1894, when around 25% of deaths in the first year of life were ascribed to this causal group. Although the 1890–1894 peak coincided with the start of the continuous decline in the city's infant mortality rate, water-food borne diseases did not contribute to the decline until 1905–1907. Deaths from 'congenital and birth disorders' followed a similar pattern, accounting for about 5% of all deaths before 1879, but over the next three decades mortality attributed to 'weakness from birth' and 'premature birth' increased rapidly. By the end of the study period they accounted for around 20% of all infant deaths. By 1865, 'air-borne diseases' had become the most lethal group of causes, with bronchitis, pneumonia, and whooping cough being responsible for 64% of such deaths.

Figure 3 also shows that from being the most dominant cause of death at the start of the study period, *kramper*, or 'convulsions', gradually diminished. By 1865–1869 this cause had been surpassed by the rapidly increasing 'air-borne diseases'; in 1880–1884 it was overtaken by 'water-food borne' diseases and, five years later, by 'congenital and birth disorders'. Of the 1,552 infants who died from convulsions (23% of all infant deaths in Trondheim), 87% were noted as succumbing to the Norwegian vernacular term *krampe* or *kramper*, 6% to 'convulsions', and 3% to 'eclampsia'. The remaining 4% of 'convulsions' deaths were described in terms which were only seen once, as many of them included additional descriptive details. Even although use of *krampe* and *kramper* gradually declined during the period covered here, these terms are still seen in the modern nosology.⁶

As outlined in section 1.2 above, medical death certificates were issued in Trondheim from 1862 onwards and from that date were collected by the priests when they were notified of a death. We can therefore assume that from 1862 the causes of death in the burial registers were copied by the priest from the medical certificates. Before 1862, the quality of registration was probably more mixed, as the causes of death recorded were provided by a mixture of lay informants and medical practitioners. Infant mortality from 'convulsions' fluctuated between 57 and 90 per 1,000 births across the 1830s, 1840s and 1850s,

6 In the ICD10 classification system 'convulsions' corresponds to 'Other and unspecified convulsions' (R56.8) in the sub section "General symptoms and signs" in chapter XVIII "Symptoms, signs and abnormal clinical and laboratory findings, not elsewhere classified".

but then declined from 46% in 1860 to less than 5% by the end of the study period. 'Convulsions' went from accounting for more than half of all infant deaths each year at the start of this period to a mere 5% at the beginning of the 20th century. It is difficult to assess if this change was caused by a true decline in deaths involving convulsions, or whether diagnostic and/or registration practices improved. The following discussion considers the evolution of the term 'convulsions' in both the *Magazin for Lægevidenskaben* and the series of cause of death statistics published in the yearly medical reports in order to glean a better understanding of the use of this centuries-old term.

Until 1860, the term *krampe* (hereafter 'convulsions') was not explicitly used as a main cause of death in the annual medical reports. Instead, the term 'eclampsia infantum' was more often seen. 'Convulsions' was, on the other hand, frequently used in the descriptive section of the medical reports to refer to a symptom or to describe a secondary cause associated with diseases such as whooping cough, croup, diarrhoea and cholera, scarlet fever, bronchitis and, occasionally, measles. Two points are worth making. First, 'convulsions' was more often mentioned as a secondary cause if epidemics of one or more other diseases were occurring. When, for instance, whooping cough swept through Buskerud, a province in southern Norway, in 1860, most of the cases were described as being mild in nature, but some were complicated by both pneumonia and, amongst the youngest children, 'convulsions'. Most of the latter cases ended fatally. Secondly, reference to 'convulsions' as a symptom or as a secondary cause seldom appeared in the text of the medical reports after 1870.

From 1861 'eclampsia infantum' was replaced by 'convulsions' and sometimes 'convulsions infantum' in the annual national cause of death statistics, and the term was also integrated into the Norwegian nomenclature published that year (NML, 1861).⁷ In the following years several provinces reported 'convulsions' as a cause of death under the heading 'brain inflammation' in the descriptive section of the medical report. In 1863 'convulsions' was classified as belonging to a group of causes which comprised 'brain inflammation, tuberculous meningitis and convulsions in childhood'. This group formed one of the most frequently encountered cause of death categories in several provinces. From 1886 *kramper i smaabørnsalder*, 'convulsions in childhood', was listed as a single, independent cause of death and as a result it fell from being among the top 4 causes on the list to number 15 out of the 23 causes listed. It should be noted that the vast majority of deaths from *kramper i smaabørnsalder* occurred amongst infants rather than older children.

Finally, in 1896, 'convulsions' was reclassified in the annual national and provincial cause of death statistics. A new nomenclature was introduced, restructuring the previous classification system. Previously, the causes of death had been listed in alphabetic order. The new list was organized into four classes: A) infectious- nutritional- and constitutional diseases, distinguished as to whether they were 1) acute, or 2) chronic; B) diseases of specific organs, which were divided into sub-groups: 1) brain and spinal cord, 2) circulation, 3) respiratory, 4) intestinal, 5) liver, 6) urinary organs, 7) sex organs, 8) skin and external diseases; C) diseases of pregnancy and childbirth, and D) unnatural causes. Under this new system the term 'convulsions in childhood' appeared in Class B, subgroup 1 as disease of the 'brain and spinal cord'. In the instructions accompanying the new nomenclature, it was emphasized that if there was any doubt as to whether a disease should be registered as an infectious disease or as a disease of a specified organ, then it should be registered as an infectious disease (NML, 1897, pp. 110–128).

In short, even though mortality attributable to 'convulsions' was trending downwards there are no indications that this medical term had disappeared from medical terminology. The term was listed as one of 122 ways of dying in the 1861 nomenclature, and it continued to appear within a significant group of causes in the national cause of death statistics. A more detailed classification was implemented in 1896, but by that time, 'convulsions' was rarely given as a cause of death in Trondheim.

Remarkably, *Magazin for lægevidenskaben*, is relatively silent on the topic of 'convulsions' or 'convulsions in childhood' — except for the annual reports compiled by Frans Christian Faye, the obstetrician who founded Christiania's maternity hospital in 1846. In the era before 1860, when the term 'convulsions' was used solely as a symptomatic term or as a secondary cause of death in the annual medical reports, Faye summarized an article, published in the *Edinburgh Monthly Journal of Medical Science*, which was written by J. Y. Simpson, professor of midwifery at the University of Edinburgh. Simpson's article, "Chloroform in Infantile Convulsions, and Other Spasmodic Diseases", is in the form of an essay, giving details of a number of the cases he had seen, followed by comments and a short discussion. Other

7 As previously stated the nomenclature included both Norwegian and Latin disease terms, which in this case refers to *kramper* and *convulsiones*.

than recommending the topic of the article to readers of the Norwegian journal, Faye did not make any remarks or reflections on its content, suggesting that he assumed that readers would be familiar with its subject. Instead, he started, as Simpson had, by asserting that of all the diseases affecting children there were few which were more fatal than convulsions. Regarding the different forms of the disease, Simpson was content to quote the general opinion of pathologists, that "by far the greatest proportion of infantile convulsive attacks are [...] usually traceable to some irritation of a distant surface or part, such as the stomach, bowels, teeth, etc." (NML, 1852, pp. 461–466). Faye confirmed his lack of certainty concerning the disease's aetiology 25 years later, in his maternity hospital report of 1877, where he noted 18 cases of convulsions amongst infants, half of which were fatal. The causes of these convulsions, he went on to argue, were apparently diverse, as infants easily developed convulsive movements as a symptom of an internal complaint or of a secondary illness (NML, 1877, pp. 745–746).

To summarize, the above discussion reveals that 'convulsions' appeared somewhat differently when we compare the cause of death statistics with the narrative section of the medical reports. In the annual cause of death statistics, 'convulsions' were only reported for infants throughout most of the 19th century. In 1896, 'convulsions' was removed from the cause of death statistics and replaced by 'convulsions in childhood', which was classified as a disease of the 'brain and spinal cord'. It is reasonable to assume that the extension of the age group to cover all children in the annual statistics,⁸ was an acknowledgement that children older than one year of age also died of 'convulsions', although admittedly to a lesser extent than infants. This awareness is evident in the descriptive sections of the medical reports, where 'convulsions' are referred to as 'convulsions in childhood' throughout the study period.

Even though 'convulsions' was one of the most frequently reported causes of death amongst infants and children, it was only fleetingly discussed in Norway's leading medical journal. This silence is intriguing. In the statistics, 'convulsions' was more precisely defined as a symptom caused by disorders of the brain and spinal cord; reflecting, it is reasonable to assume, greater medical knowledge and/or increased use of diagnostic terminology. One question remains, however: why were there so few publications addressing the association between 'convulsions' and its possible causes? Finally, as mentioned above, the collaboration between the clergy and the doctors was formalised at the start of the 1860s with the introduction of printed cause of death certificates and the national nomenclature. As a result the burial registers came to reflect current medical discourse, at least in Norway's cities.

4 NEONATAL AND POST-NEONATAL MORTALITY — AN OVERVIEW

The observed decline in infant mortality from about 1895 seen in Figure 2 was, as shown in Figure 4, mainly the result of a decline in post-neonatal mortality. Trondheim experienced post-neonatal mortality which was on average 10‰ higher than that of other cities in Norway, but all cities experienced similar rapid declines at around the same time (Backer, 1961).⁹ Figure 4 demonstrates that both neonatal and post-neonatal mortality were higher among boys than girls, although in neither case was the difference statistically significant.¹⁰ Boys were much more likely to experience post-neonatal mortality rates exceeding 120 deaths per 1,000 live births (see appendix A5) than girls. Furthermore, the trends in post-neonatal mortality for both sexes saw greater fluctuations than the trends in neonatal mortality, emphasising that after the first month of life the exogenous environment became an increasingly important influence on children's survival chances.

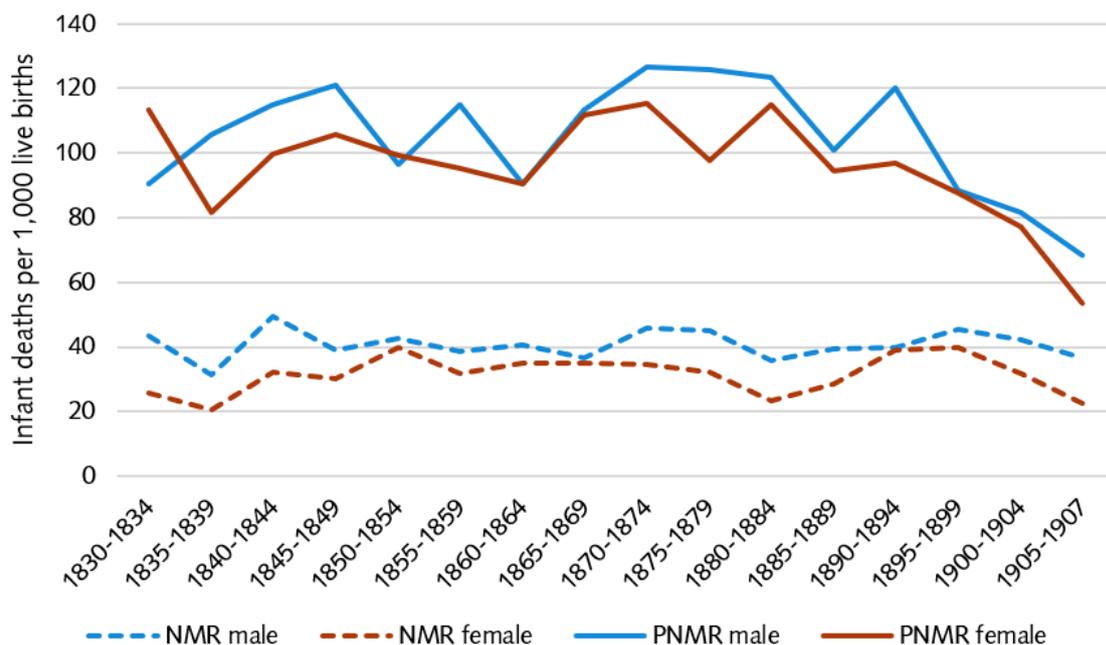
While neonatal mortality for boys fluctuated around 40 per 1,000 live births, rates of 20 to 40 per 1,000 were more common amongst girls, but there was no clear trend in the survival chances of newborns of either sex across the study period. That said, Norway's national statistics shows a continuous decline in neonatal mortality in the country's cities from around 1895, and the decline visible during the last three quinquennia of our study could therefore represent the beginning of improvement in survival across the first month of life (Backer, 1961). On average, 26.7% of the infant deaths observed occurred to neonates, however, as a consequence of falling post-neonatal mortality, the relative risk of dying during the first month of life increased towards the end of the study period (see appendix A6).

8 Deaths by age-groups 0–1, 1–5, 5–10, etc., were provided in the statistics from 1867 onwards.

9 Norwegian official statistics have published number of deaths by age in months for urban and rural environments starting in 1876.

10 Based on confidence intervals of 95%.

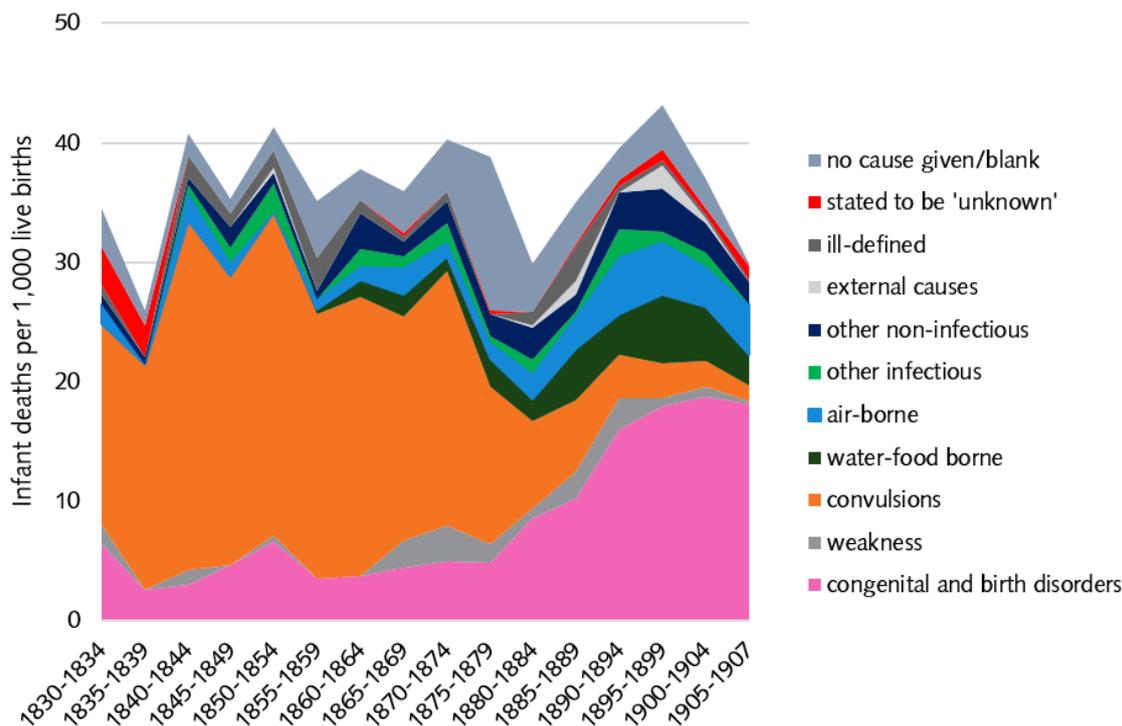
Figure 4 *Quinquennial rates of neonatal (NMR) and post-neonatal (PNMR) mortality, by sex, Trondheim 1830–1907*



Source: See Figure 2.

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

Figure 5 *Quinquennial rates of neonatal (NMR) mortality, by causal group, Trondheim 1830–1907*



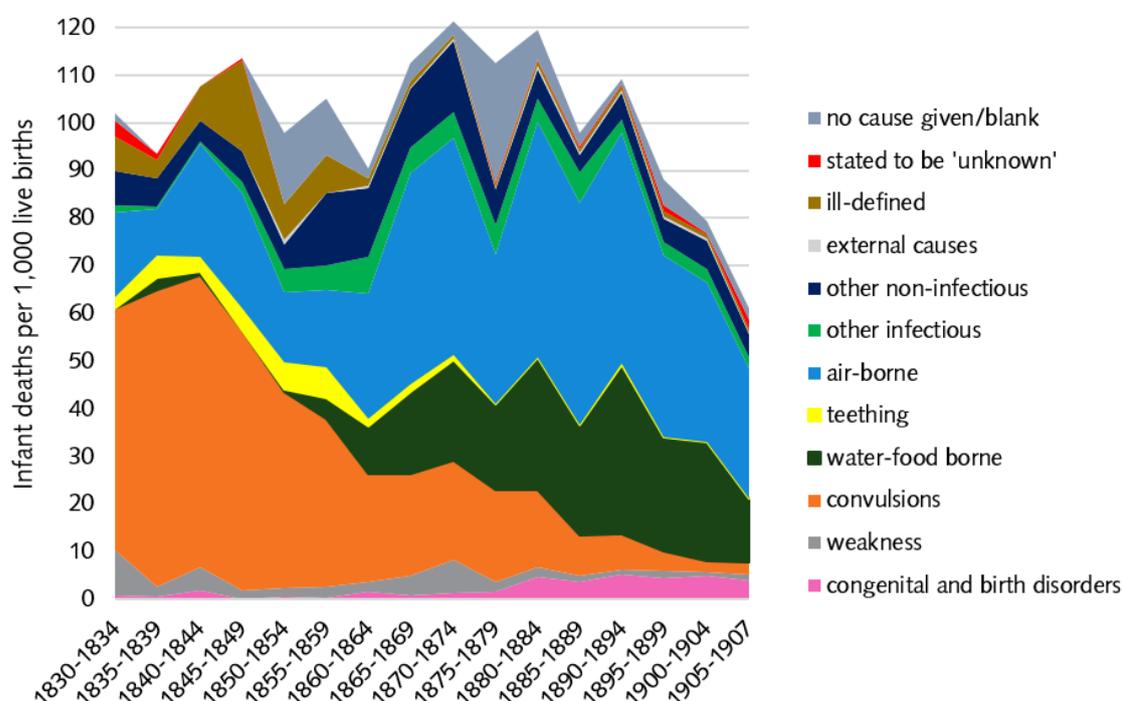
Source: See Figure 2.

Note: For figures underlying this graph see appendix A7.

Figure 5 shows the quinquennial neonatal mortality rates by causal group, demonstrating that two groups, 'convulsions' and 'congenital and birth disorders', dominated the picture.¹¹ Up until 1865–1869, 'convulsions' accounted for 50–70% of all neonatal deaths, but by the end of the study period, this proportion had shrunk to around just 5%. From around 1875 the proportion of 'air-borne diseases' increased from 2–5% of all neonatal deaths to about 10%. A similar increase can be seen for 'water-food borne diseases', a class of cause of death which did not appear in the burial registers until the mid-1850s. While the 'congenital and birth disorders' group made up about 10% of neonatal deaths up until 1875–1879, thereafter this share increased rapidly, and by the end of the period this group accounted for around half of all neonatal deaths; the majority succumbing to 'weakness from birth' and 'premature birth'. The extent to which these changes relate to changes in the registration of premature births, particularly of those babies who lived for no more than a few minutes, remains to be investigated. On the other hand, given the coincidence between the drop in 'convulsions' deaths and the rise in 'congenital and birth disorders', it is likely that the great bulk of deaths from 'convulsions' found their way into the 'congenital and birth disorders' category, slowly from 1860–1864, but then more rapidly, before stabilizing at around 5% of the total in the final years covered by the study.¹² To assess these trends in greater detail we will look at the seasonality pattern by causal groups in section 6.

Figure 6 shows the quinquennial post-neonatal mortality rates by causal group 1830–1907. Three causal groups dominated the graph, albeit to varying degrees: namely 'convulsions', 'air-borne' and 'water-food borne' diseases. As amongst neonatal deaths, post-neonatal deaths from 'convulsions' showed a mark decline, but this started earlier, in the mid-1840s. The combination of decreasing mortality from 'convulsions' and a simultaneous increase in the rates of death from 'air-borne' and 'water-food borne' diseases, observed amongst neonates is also seen in the post-neonatal period. 'Teething' diminished as a cause of death after 1875, suggesting that deaths formerly attributed to this cause were later attributed to 'water-food borne diseases'. One way to test this hypothesis might be to compare the seasonality of these causes, but sadly the association between 'teething' and the summer peaks in 'water-food borne diseases' could not be evaluated due to small numbers.

Figure 6 *Quinquennial rates of post-neonatal (PNMR) mortality, by causal group, Trondheim 1830–1907*



Source: See Figure 2.

Note: For figures underlying this graph see appendix A8.

11 'Teething' is excluded as a causal group due to zero cases.

12 5% is a level which in fact equals the median level of 'convulsions' reported among the 151 member countries of WHO around 2000 (Mathers et al., 2005).

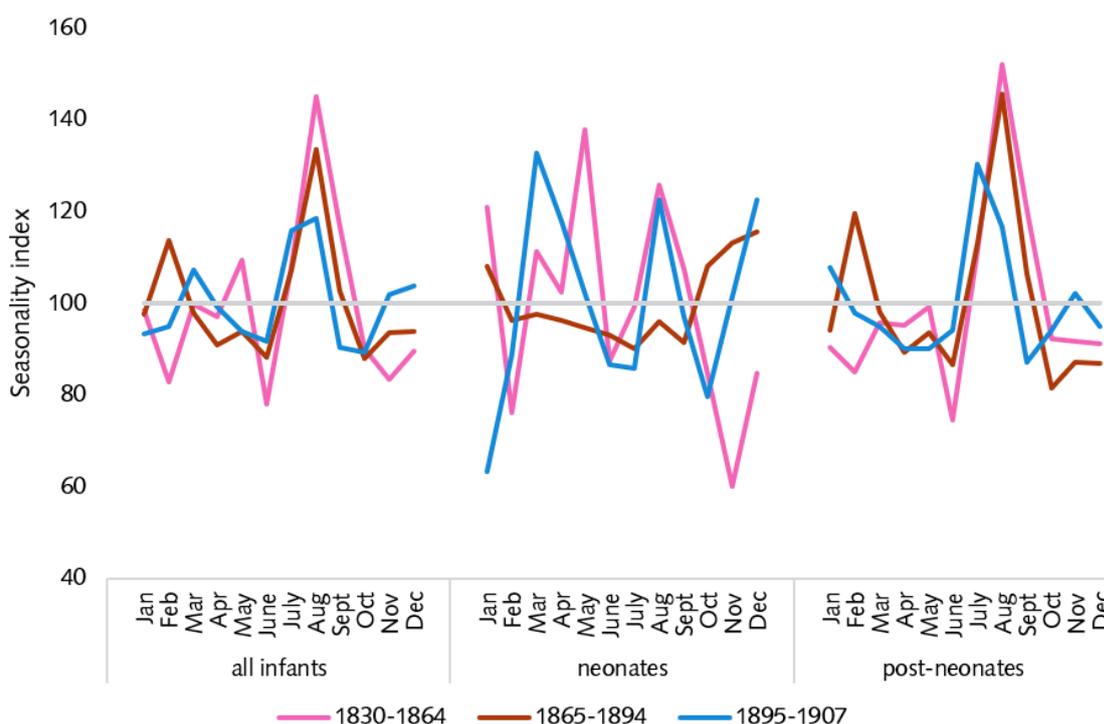
From 1895, when post-mortality started to decline, 'air-borne infectious diseases' accounted for nearly half of such deaths, and 'water-food borne' for around one third. During the following two decades, 'air-borne' post-neonatal mortality rates declined from around 50 to 27 per 1,000 live births, and 'water-food borne' from 35 to 13 per 1,000.

As previously mentioned, boys consistently had both higher neonatal and post-neonatal mortality than girls. The most noticeable sex difference in mortality by causal group is observed in the neonatal period, when boys have considerably higher rates of death from 'convulsions' and 'congenital and birth disorders'. Boys also had higher rates of 'convulsions' in the post-neonatal period (see appendix A9).

6 SEASONALITY OF NEONATAL AND POST-NEONATAL MORTALITY

Figure 7 shows seasonality amongst all infant, neonatal, and post-neonatal deaths in Trondheim. To capture trends over time, three periods characterized by differences in causes of death registration, mortality levels and sociodemographic features are compared. The first period, 1830–1864, was the era when the majority of infants were said to die of 'convulsions' and the city authorities were just beginning to implement the infrastructures needed to improve hygiene and sanitation. In the second period, 1865–1894, the city was hit by a severe economic depression and unemployment rates were high (Mykland, 1996). Nevertheless, the city's health board was more experienced at putting measures in place to prevent the spread of contagious diseases, and the water- and sanitary systems were gradually extended during this period until they covered all areas within the city boundaries. Despite this progress, the city experienced higher infant mortality during these decades than in the previous period, particularly amongst post-neonates. Between 1865 and 1894 diarrhoeal diseases contributed 28% of all post-neonatal deaths, while in the previous period this figure had been just 7.8%. The mortality peak in 1890–1894, shown in Figures 2 and 4, appears to have been primarily driven by an increase in post-neonatal mortality which was caused by a series of summers which were described by contemporary medical practitioners as being unusually hot and dry (NOS, 1893, 1896, 1897). During this quinquennium nearly 50% of all deaths amongst post-neonates in Trondheim were caused by diarrhoeal disease, suggesting that the city still lacked adequate water and sanitation water systems. The final period, 1895–1907, began one year after the city's boundaries had seen a marked expansion. It was in this period that infant mortality in the city began to decline.

Figure 7 *Seasonality indices for deaths amongst infants, neonates and post-neonates, for selected periods, Trondheim 1830–1907*

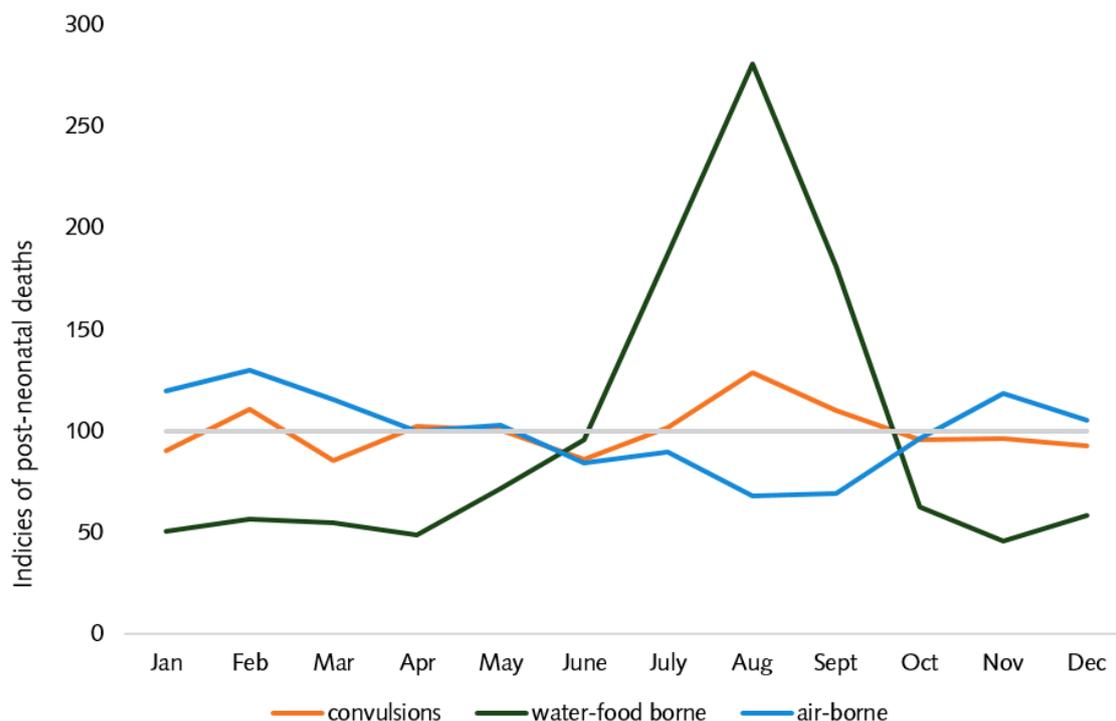


Looking at the seasonal fluctuations in overall infant mortality in Figure 7, there is a sharp peak in August in each of the three periods, although there were 45% more deaths than expected (if deaths were evenly distributed across the year) in 1830–1864, but only 18% more in 1895–1907. The August peak was clearly more pronounced amongst post-neonates; the seasonality index for this group lying at around 145–152 in the first two periods but decreasing to 116 in the third. The gradual mortality decline that occurred in 1895–1907, was thus accompanied by reduced seasonal fluctuation. Any change in seasonality over time was less clear amongst neonates, but this may have been because of small numbers.¹³ When all years are combined (see appendix A10), mortality amongst neonates was higher during spring, i.e. March–May, than in the summer months of June and July, but a peak similar to that amongst post-neonates, if rather more muted, occurred in August. The autumn months, September–October, saw relatively low numbers of infant deaths overall and, except for December, the winter season between November and February also seems to have been relatively favorable.

Figure 8 shows the seasonality of post-neonatal deaths by the three main causal groups: 'convulsions', 'air-borne', and 'water-food borne' diseases. 'Water-food borne' diseases in this age group were found to have a particularly concentrated seasonal pattern, with seasonality indices of between 180 and 280 in the months of July, August and September, but virtually no cases other parts of the year. The 'air-borne diseases' have a quite different seasonal profile; the seasonality index being highest between November and February. Interestingly, 'convulsions' are more evenly distributed across the 12 months, except for a relatively small peak in August.

Figure 9 shows the seasonality of neonatal deaths for the four main causal groups: 'congenital and birth disorders', 'convulsions', 'air-borne' and 'water-food borne' diseases. The most notable feature is the pronounced peak in 'water-food borne' diseases during July, August and September. The seasonal profile of 'air-borne' deaths amongst neonates has a characteristic u-shape, as high numbers of deaths from these causes occurred in the late autumn and throughout the winter, but were much less prevalent during spring and summer. Interestingly, 'convulsions' and 'congenital and birth disorders' had roughly similar seasonal distributions, showing little concentration, apart from for a slight increase in the former during May.

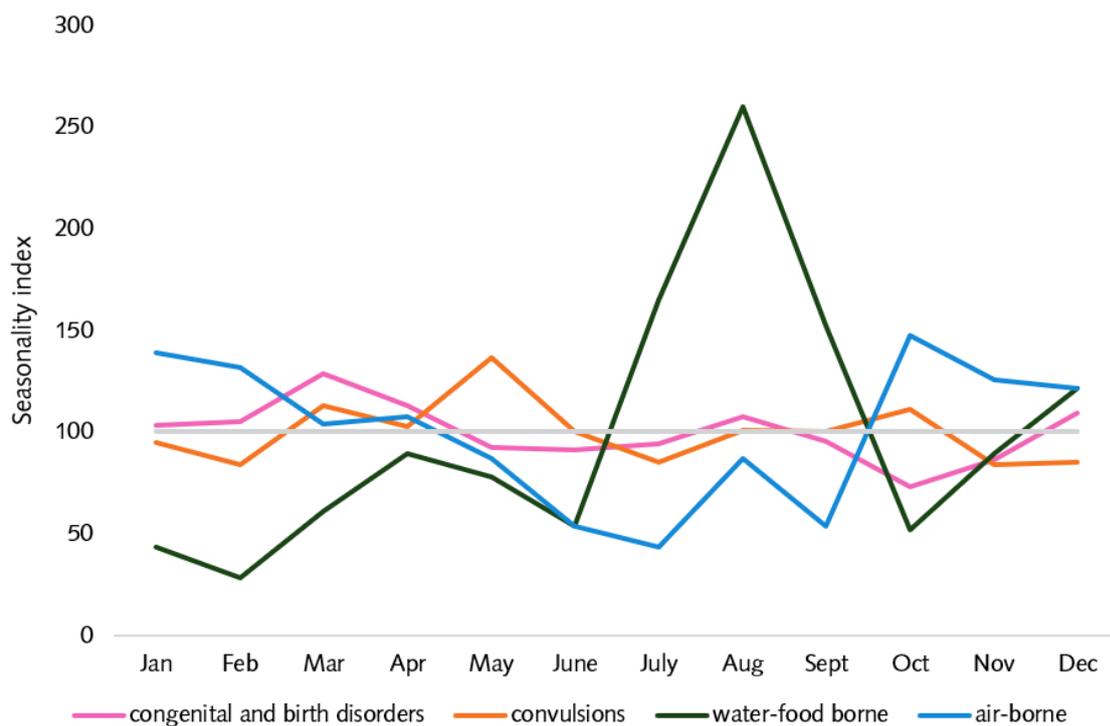
Figure 8 Seasonality indices for selected causal groups of post-neonatal mortality, Trondheim 1830–1907, all years combined



Source: See Figure 2.

13 There were 40 deaths per month on average 1830–1864, 65 per month 1865–1894 and 48 per month 1895–1907.

Figure 9 Seasonality indices for selected causal groups of neonatal death, Trondheim 1830–1907, all years combined



Source: See Figure 2.

7 DISCUSSIONS AND CONCLUSIONS

Using information extracted from Trondheim's burial registers, this study has explored cause of death patterns amongst the city's infants, between 1830 and 1907. It has also investigated continuity and change in the official instructions regarding cause of death registration and the role of priests as registrars of cause of death.

Up until 1860–1864 infant mortality in Trondheim fluctuated between 120 and 140 deaths per 1,000 live births. Thereafter, until 1890–1894, mortality was rather higher, especially amongst boys. From 1895 onwards, mortality began to fall, mainly as a result of decreasing post-neonatal mortality. The date of onset and the speed of the mortality decline were similar to those observed in Norway's other big cities. However, despite having Europe's lowest infant mortality at the time, Norway still suffered from a marked urban penalty. Even at the end of the study period infant mortality rates in the cities lay around 50% above those found in rural areas.

At the start of the decline in infant mortality, 'air-borne diseases' accounted for nearly half of the deaths amongst post-neonates, and 'water-food borne' for around one third. The ensuing fall in rates was predominantly driven by declines in these two causal groups. Furthermore, the relative trends in mortality amongst neonates and post-neonates resulted in a significant change in the distribution of deaths over the first year of life, with an increasing proportion of deaths occurring during the first month. Although levels of neonatal deaths in Trondheim remained relatively stable over the study period, the distribution of causes altered. Deaths from 'convulsions' and 'congenital and birth disorders' saw the greatest change. By the beginning of the 20th century 'convulsions' had faded away to near insignificance, and deaths from causes such as 'debility from birth', 'prematurity', 'birth injury' and 'congenital malformation' had come to dominate. Seasonality indices for both neonates and post-neonates revealed that deaths from 'water-food borne diseases' were concentrated in the summer, and as mortality declined seasonal fluctuations from these causes were reduced.

With the passing of the Public Health Act in 1860, several measures were put in place to improve hygiene and sanitation in the city. These eliminated some of the basic hazards to health, yet more than 30 years went by before infant mortality started to decline. Several circumstances may have contributed to this. As mentioned above, Trondheim's city boundary expanded in 1864 and again in 1893, and a possible explanation for the excess mortality seen in 1865 might be the 2,000 or so newly included inhabitants who, according to the chief medical officer, brought with them nothing but misery and want. Furthermore, during the economic difficulties of the 1870s and early 1880s, many families faced wage reductions and unemployment. During this period, when conditions for everyone were hard, registered deaths from diarrhoeal diseases were four times higher than in 1830–1864. 'Water-food borne diseases' continued to be a major threat during the unusually hot and dry summers of the early 1890s when more than 30% of all deaths amongst post-neonates was caused by diarrhoeal diseases.

One puzzling question is why did infant mortality not increase in 1895 or the following years when the new inhabitants brought into the city by the expansion of its boundary in 1893 were described as being in "misery and want", just like their predecessors in 1864? Quite to the contrary, infant survival increased substantially. No clear association can be discerned between the timing of the decline and the introduction of public health measures. The city's budget was constrained, and its health board faced the dual challenges of an outdated sewage system in the old inner city and appalling conditions in the peripheral districts. Furthermore, due to a severe housing shortage, those who moved into the city around 1900 had little option but to rent sub-standard apartments. The contemporary medical practitioners painted a picture in their reports of a city clearly plagued by growing pains. On the other hand, there were some positive developments: there was more effective supervision of the hygienic and sanitary measures instituted by the city's health authorities, water pipes ran into all houses by 1900, and provision of midwives had increased by the same date. More research is required before the complex associations between these developments can be fully understood.

One of the aims of this article was to examine how the official instructions regarding cause of death registration and classification changed over time, and to investigate the role of priests as registrars of cause of death. 'Teething', 'convulsions', and 'weakness' are particular causes of interest in this special issue, and this article has focused on the use of the term 'convulsions' which up until late 1860s accounted for 50–70% of all infant deaths in Trondheim. Based on a close reading of the documents describing the relevant laws and directives, the contents of Norway's leading medical journal at the time, and contemporary medical statistics, the conclusion was reached there were no indications that 'convulsions' disappeared as a diagnostic term at the time when mortality attributed to this cause went into decline.

While 'water-food borne diseases' exhibited a strong summer peak, a similarly convincing seasonal pattern was not found in 'convulsions' in Trondheim, suggesting that the term was not used solely as to refer to a symptom unambiguously connected to diarrhoea and dehydration, as has been suggested by previous studies (Guttormsson & Garðarsdóttir, 2001; Kintner, 1986; Knudtsen, 1997; Thompson, 1984; Wolleswinkel-van den Bosch et al., 1997). If there had been a connection, a significant increase in deaths from 'convulsions' would be expected in the summer months.

Although the issue of small numbers has prevented a more detailed examination of the seasonal patterns of cause of death over time, there is ample evidence for an alternative explanation to the decline of deaths caused by 'convulsions'. From the point when deaths from this cause began to decrease, several measures were taken to improve cause of death registration. Most notable of these was the introduction of death certificates, which included a cause of death certified by a medical practitioner. The certificates had to be brought, or delivered, to the priests' office before burial could proceed. This meant that the causes of death entered in the Trondheim's burial registers were most likely copied from the medical certificates. Use of the certificates ended, or at least reduced, the number of lay informants offering vague causes of death such as 'convulsions'. In a similar vein, Reid et al. (2015) have suggested that the introduction of medically certified cause of death certificates was a key factor in the decline of deaths from 'old age'. It certainly seems from the changing patterns of cause of death presented in this article, that medical practitioners in Norway were increasingly likely to attribute deaths among neonates to 'prematurity', 'congenital malformation' or other conditions and diseases closely related to birth, rather than to 'convulsions'. Amongst post-neonates, improvements in diagnosis and increased medical certification resulted in 'convulsions' deaths being increasingly re-attributed to the 'air-borne' or 'water-food borne' categories (see Figure 6). 'Air-borne' and 'water-food borne' diseases had, as a consequence, been under-reported in the period before 1865. This throws considerably doubt on

the suggestion above that deaths from diarrhoeal diseases were four times higher as a proportion of all deaths in 1865–1894 than in the previous 35 years, particularly as the relabeling of 'convulsions' to other causes was considerably more complex than previously thought.

The development of the ICD10h coding system means that various sources of data can be reconciled into formats that are compatible and comparable over time and space. The obvious strength of ICD10h is that it offers a transparent, harmonized coding system which can be used across a range of countries. It also allows time series analysis to be carried out over long periods, offering the possibility that historical cause of death registers might be linked to contemporary ones. Potentially, this would allow investigation of the role of heredity in disease; of the social, cultural, and economic reproduction of good or bad health; and of many other topics where research questions remain unanswered. A major challenge when constructing a historical variant of the globally recognised ICD10 system is that the latter is not designed to capture change over time. Each edition of the ICD (there have now been 11 since the first release at the beginning of the 20th century) is a product of lengthy negotiations between medical professionals, policy makers, economists, and researchers (Kveim Lie & Greene, 2020) and represents the medical knowledge or cultural context of a particular time.

The different editions of the ICD thus represent the understanding of medical knowledge and/or the cultural context at a particular time. Identification of a disease, giving it a name, is part of a complex decision-making process involving not just the principles of taxonomy and classification, but also an understanding of its characteristics which in turn leads to a course of action or treatment (Larsen, 2021). In this article, I have sought to show how one condition, namely *kramper*, or 'convulsions', which had been recognised by doctors throughout history was, with the introduction of medical certificates of cause of death, re-classified to other diseases which doctors of the day considered to provide a more precise cause of death. Such change is not captured by ICD10h.

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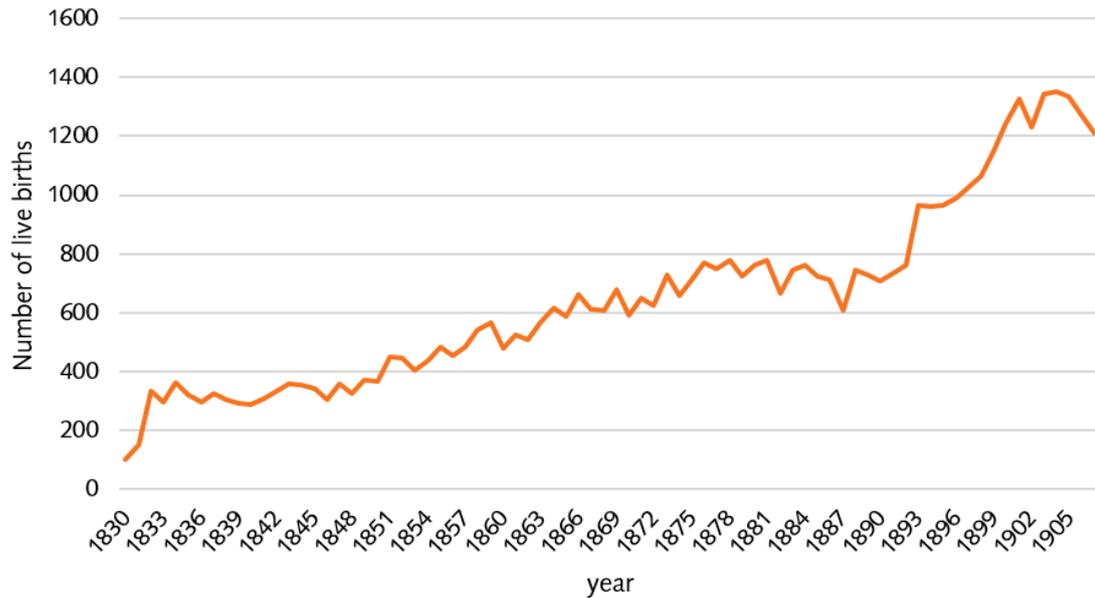
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APPENDIX

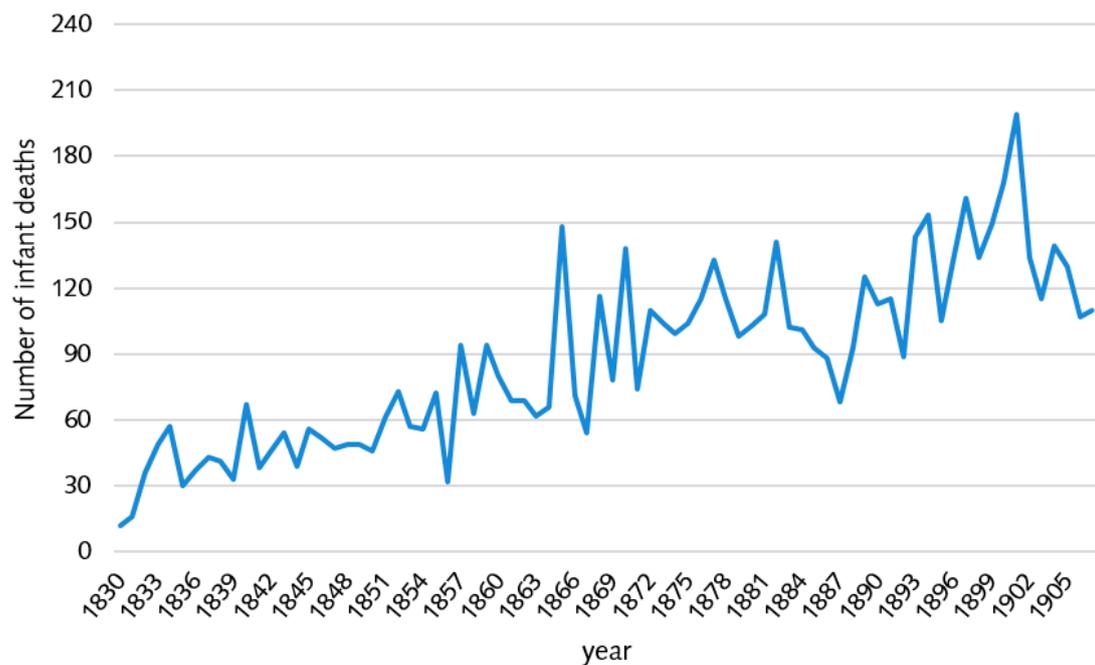
A1 NUMBER OF LIVE BIRTH PER YEAR, TRONDHEIM 1830–1907



Source: Norwegian Historical Data Centre, UiT the Arctic University of Norway, Historical Population Register of Norway, Church books, Trondheim, 1801–1920. Original sources at the National Archive of Norway.

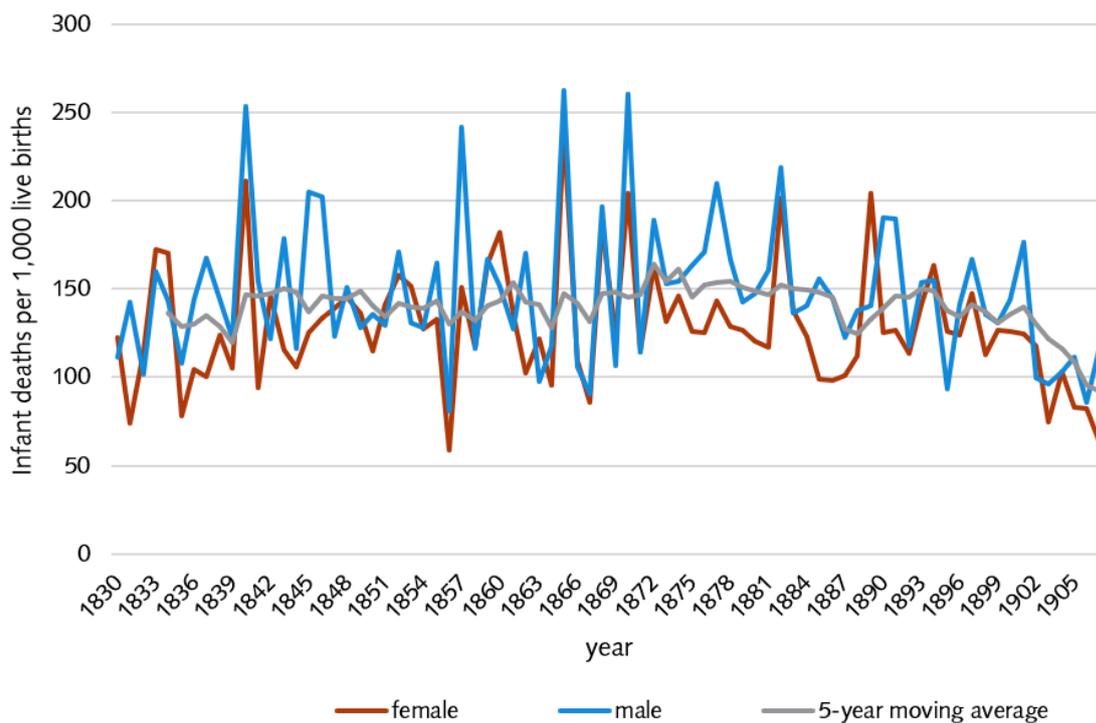
Note: The stagnation of births in the period 1882–1892 might be explained by a large amount of emigration to the United States. Between 1880 and 1895 about 5,500 people, around 20–25% of the population, left the city (Danielsen, 1997).

A2 NUMBER OF INFANT DEATHS PER YEAR, TRONDHEIM 1830–1907



Source: See Figure A1.

A3 ANNUAL INFANT MORTALITY RATES, BY SEX, TRONDHEIM 1830–1907



Source: See Figure A1.

A4 QUINQUENNIAL INFANT MORTALITY RATES (IMR) BY CAUSAL GROUP; TRONDHEIM 1830–1907

	1830– 1834	1835– 1839	1840– 1844	1845– 1849	1850– 1854	1855– 1859	1860– 1864	1865– 1869	1870– 1874	1875– 1879	1880– 1884	1885– 1889	1890– 1894	1895– 1899	1900– 1904	1905– 1907*
congenital and birth disorders	7.2 (9)	3.2 (5)	4.9 (8)	4.7 (8)	7.1 (15)	4.0 (10)	5.2 (14)	5.4 (17)	6.2 (20)	6.4 (24)	13.2 (49)	13.9 (49)	21.1 (87)	22.3 (116)	23.9 (155)	22.0 (84)
weakness	11.2 (14)	1.9 (3)	6.1 (10)	1.8 (3)	2.4 (5)	2.4 (6)	2.2 (6)	6.4 (20)	10.2 (33)	3.7 (14)	3.2 (12)	3.4 (12)	3.9 (16)	2.5 (13)	1.4 (9)	1.6 (6)
convulsions	67.4 (84)	81.2 (125)	90.6 (149)	78.8 (134)	68.3 (144)	57.3 (145)	46.0 (124)	40.0 (126)	42.2 (137)	32.4 (121)	23.4 (87)	14.5 (51)	11.1 (46)	6.9 (36)	4.3 (28)	3.9 (15)
water-food borne	0.0 (0)	2.6 (4)	0.6 (1)	0.0 (0)	0.5 (1)	4.7 (12)	11.5 (31)	19.1 (60)	22.2 (72)	20.9 (78)	29.6 (110)	27.6 (97)	38.8 (160)	29.6 (154)	29.5 (192)	16.0 (61)
teething	2.4 (3)	4.5 (7)	3.0 (5)	4.7 (8)	5.7 (12)	6.3 (16)	1.5 (4)	1.6 (5)	1.2 (4)	0.0 (0)	0.0 (0)	0.0 (0)	0.2 (1)	0.0 (0)	0.0 (0)	0.0 (0)
air-borne	19.3 (24)	9.7 (15)	26.2 (43)	25.3 (43)	14.7 (31)	17.0 (43)	27.4 (74)	46.4 (146)	46.8 (152)	32.6 (122)	51.7 (192)	49.5 (174)	55.5 (229)	42.9 (223)	37.2 (242)	31.5 (120)
other infectious	1.6 (2)	0.6 (1)	1.2 (2)	3.5 (6)	7.1 (15)	5.1 (13)	9.3 (25)	6.4 (20)	6.8 (22)	7.0 (26)	6.2 (23)	6.5 (23)	4.8 (20)	3.5 (18)	4.0 (26)	2.4 (9)
other non-infectious	8.0 (10)	6.5 (10)	4.9 (8)	8.2 (14)	6.2 (13)	16.2 (41)	17.4 (47)	13.7 (43)	16.9 (55)	9.4 (35)	9.4 (35)	5.1 (18)	9.7 (40)	8.9 (46)	8.5 (55)	6.6 (25)
external causes	0.0 (0)	0.0 (0)	0.0 (0)	0.0 (0)	1.4 (3)	0.0 (0)	0.4 (1)	0.3 (1)	0.3 (1)	0.3 (1)	1.1 (4)	1.7 (6)	0.5 (2)	2.5 (13)	0.9 (6)	0.3 (1)
ill-defined and unknown	8.0 (10)	3.9 (6)	9.1 (15)	20.0 (34)	9.0 (19)	10.7 (27)	2.6 (7)	1.6 (5)	1.8 (6)	0.8 (3)	2.4 (9)	3.4 (12)	1.5 (6)	1.3 (7)	1.1 (7)	1.0 (4)
stated to be 'unknown'	6.4 (8)	3.9 (6)	0.0 (0)	0.6 (1)	0.0 (0)	0.0 (0)	0.0 (0)	0.3 (1)	0.0 (0)	0.8 (3)	0.3 (1)	0.9 (3)	0.7 (3)	2.3 (12)	0.6 (4)	3.1 (12)
no cause given/blank	4.8 (6)	1.3 (2)	1.8 (3)	1.2 (2)	16.6 (35)	16.6 (42)	4.8 (13)	7.3 (23)	7.1 (23)	37.7 (141)	10.2 (38)	6.3 (22)	3.6 (15)	8.9 (46)	5.1 (33)	2.6 (10)

* The final period includes only three years. Source: See Figure A1.

Note: Each cell provides the IMR and, in parentheses, the number of deaths on which it is based.

A5 QUINQUENNIAL RATES OF NEONATAL (NMR) AND POST-NEONATAL (PNMR) MORTALITY, TRONDHEIM 1830–1907



Source: See Figure A1.

A6 QUINQUENNIAL RATES OF NEONATAL (NMR) AND POST-NEONATAL (PNMR) MORTALITY, WITH THE PERCENTAGE OF INFANT DEATHS IN THE NEONATAL PERIOD AND POST-NEONATAL PERIOD FOR EACH QUINQUENNIUM, TRONDHEIM 1830–1907

Period	NMR	PNMR	%NM	%PNM
1830–1834	34.5	101.9	25.3	74.7
1835–1839	26.0	93.6	21.7	78.3
1845–1849	35.3	113.5	23.7	76.3
1850–1854	41.3	97.7	29.7	70.3
1855–1859	35.2	105.1	25.1	74.9
1860–1864	37.8	90.5	29.5	70.5
1865–1869	35.9	112.5	24.2	75.8
1870–1874	40.3	121.2	25.0	75.0
1875–1879	38.8	112.4	25.7	74.3
1880–1884	29.9	119.6	20.0	80.0
1885–1889	35.0	97.8	26.3	73.7
1890–1894	39.5	109.1	26.6	73.4
1895–1899	43.1	88.0	32.9	67.1
1900–1904	36.9	79.3	31.8	68.2
1905–1907	29.9	61.1	32.9	67.1

Source: See Figure A1.

A7 QUINQUENNIAL RATES OF NEONATAL (NMR) MORTALITY BY CAUSAL GROUP, TRONDHEIM 1830–1907

NMR	1830– 1834	1835– 1839	1840– 1844	1845– 1849	1850– 1854	1855– 1859	1860– 1864	1865– 1869	1870– 1874	1875– 1879	1880– 1884	1885– 1889	1890– 1894	1895– 1899	1900– 1904	1905– 1907
congenital and birth disorders	6.4 (8)	2.6 (4)	3.0 (5)	4.7 (8)	6.6 (14)	3.6 (9)	3.7 (10)	4.4 (14)	4.9 (16)	4.8 (18)	8.6 (32)	10.2 (36)	16.0 (66)	17.9 (93)	18.8 (122)	18.1 (69)
weakness	1.6 (2)	0.0 (0)	1.2 (2)	0.0 (0)	0.5 (1)	0.0 (0)	0.0 (0)	2.2 (7)	3.1 (10)	1.6 (6)	0.8 (3)	2.3 (8)	2.7 (11)	0.8 (4)	0.8 (5)	0.3 (1)
convulsions	16.9 (21)	18.8 (29)	29.2 (48)	24.1 (41)	27.0 (57)	22.1 (56)	23.4 (63)	18.7 (59)	21.2 (69)	13.1 (49)	7.3 (27)	6.0 (21)	3.6 (15)	2.9 (15)	2.2 (14)	1.3 (5)
water-food borne	0.0 (0)	0.0 (0)	0.0 (0)	0.0 (0)	0.0 (0)	0.4 (1)	1.5 (4)	1.9 (6)	1.2 (4)	2.4 (9)	1.9 (7)	4.3 (15)	3.4 (14)	5.8 (30)	4.6 (30)	2.6 (10)
air-borne	1.6 (2)	0.0 (0)	2.4 (4)	1.2 (2)	0.0 (0)	0.8 (2)	1.1 (3)	2.2 (7)	1.2 (4)	1.3 (5)	2.2 (8)	2.8 (10)	4.8 (20)	4.4 (23)	3.4 (22)	4.2 (16)
other infectious	0.0 (0)	0.0 (0)	0.6 (1)	1.2 (2)	2.4 (5)	0.0 (0)	1.5 (4)	1.0 (3)	1.5 (5)	0.5 (2)	1.1 (4)	0.3 (1)	2.2 (9)	0.8 (4)	1.1 (7)	0.0 (0)
other non-infectious	0.8 (1)	0.6 (1)	0.6 (1)	1.8 (3)	0.9 (2)	0.8 (2)	3.0 (8)	1.3 (4)	1.8 (6)	1.9 (7)	2.7 (10)	1.4 (5)	3.2 (13)	3.7 (19)	2.5 (16)	1.8 (7)
external causes	0.0 (0)	0.0 (0)	0.0 (0)	0.0 (0)	0.5 (1)	0.0 (0)	0.0 (0)	0.0 (0)	0.0 (0)	0.0 (0)	0.3 (1)	1.1 (4)	0.0 (0)	1.9 (10)	0.5 (3)	0.0 (0)
ill-defined and unknown	0.8 (1)	0.0 (0)	1.8 (3)	1.2 (2)	1.4 (3)	2.8 (7)	1.1 (3)	0.3 (1)	0.9 (3)	0.0 (0)	1.1 (4)	2.8 (10)	0.5 (2)	0.4 (2)	0.3 (2)	0.3 (1)
stated to be 'unknown'	3.2 (4)	2.6 (4)	0.0 (0)	0.0 (0)	0.0 (0)	0.0 (0)	0.0 (0)	0.3 (1)	0.0 (0)	0.3 (1)	0.0 (0)	0.3 (1)	0.5 (2)	1.0 (5)	0.5 (3)	1.0 (4)
no cause given/blank	3.2 (4)	1.3 (2)	1.8 (3)	1.2 (2)	1.9 (4)	4.7 (12)	2.6 (7)	3.5 (11)	4.3 (14)	12.8 (48)	4.0 (15)	3.4 (12)	2.7 (11)	3.7 (19)	2.5 (16)	0.3 (1)

Source: See Figure A1.

Notes: Each cell provides the NMR and, in parentheses, the number of deaths on which it is based. The causal group 'teething' is not shown as there were zero cases of deaths from this cause.

A8 QUINQUENNIAL RATES OF POST-NEONATAL (PNMR) MORTALITY BY CAUSAL GROUP, TRONDHEIM 1830–1907

NMRs	1830– 1834	1835– 1839	1840– 1844	1845– 1849	1850– 1854	1855– 1859	1860– 1864	1865– 1869	1870– 1874	1875– 1879	1880– 1884	1885– 1889	1890– 1894	1895– 1899	1900– 1904	1905– 1907
congenital and birth disorders	0.8 (1)	0.6 (1)	1.8 (3)	0.0 (0)	0.5 (1)	0.4 (1)	1.5 (4)	1.0 (3)	1.2 (4)	1.6 (6)	4.6 (17)	3.7 (13)	5.1 (21)	4.4 (23)	5.1 (33)	3.9 (15)
weakness	9.6 (12)	1.9 (3)	4.9 (8)	1.8 (3)	1.9 (4)	2.4 (6)	2.2 (6)	4.1 (13)	7.1 (23)	2.1 (8)	2.2 (8)	1.1 (4)	1.2 (5)	1.5 (8)	0.8 (5)	1.3 (5)
convulsions	50.6 (63)	62.4 (96)	61.4 (101)	54.7 (93)	41.3 (87)	35.2 (89)	22.6 (61)	21.3 (67)	20.9 (68)	19.3 (72)	16.2 (60)	8.5 (30)	7.5 (31)	4.0 (21)	2.2 (14)	2.6 (10)
water-food borne	0.0 (0)	2.6 (4)	0.6 (1)	0.0 (0)	0.5 (1)	4.3 (11)	10.0 (27)	17.2 (54)	20.9 (68)	17.9 (67)	27.7 (103)	23.3 (82)	35.4 (146)	24.1 (125)	24.9 (162)	13.4 (51)
teething	2.4 (3)	4.5 (7)	3.0 (5)	4.7 (8)	5.7 (12)	6.3 (16)	1.5 (4)	1.6 (5)	1.2 (4)	0.0 (0)	0.0 (0)	0.0 (0)	0.2 (1)	0.0 (0)	0.0 (0)	0.0 (0)
air-borne	17.7 (22)	9.7 (15)	23.7 (39)	24.1 (41)	14.7 (31)	16.2 (41)	26.3 (71)	44.2 (139)	45.5 (148)	31.3 (117)	49.6 (184)	46.6 (164)	48.5 (200)	38.1 (198)	33.5 (218)	27.3 (104)
other infectious	1.6 (2)	0.6 (1)	0.6 (1)	2.4 (4)	4.7 (10)	5.1 (13)	7.8 (21)	5.4 (17)	5.2 (17)	6.4 (24)	4.8 (18)	6.3 (22)	2.7 (11)	2.7 (14)	2.9 (19)	2.4 (9)
other non-infectious	7.2 (9)	5.8 (9)	4.3 (7)	6.5 (11)	5.2 (11)	15.4 (39)	14.5 (39)	12.4 (39)	15.1 (49)	7.2 (27)	6.2 (23)	3.7 (13)	5.8 (24)	5.0 (26)	5.8 (38)	4.7 (18)
external causes	0.0 (0)	0.0 (0)	0.0 (0)	0.0 (0)	0.9 (2)	0.0 (0)	0.4 (1)	0.3 (1)	0.3 (1)	0.3 (1)	0.8 (3)	0.6 (2)	0.5 (2)	0.6 (3)	0.5 (3)	0.3 (1)
ill-defined and unknown	7.2 (9)	3.9 (6)	7.3 (12)	18.8 (32)	7.6 (16)	7.9 (20)	1.5 (4)	1.3 (4)	0.9 (3)	0.8 (3)	1.1 (4)	0.6 (2)	1.0 (4)	1.0 (5)	0.8 (5)	0.8 (3)
stated to be 'unknown'	3.2 (4)	1.3 (2)	0.0 (0)	0.6 (1)	0.0 (0)	0.0 (0)	0.0 (0)	0.0 (0)	0.0 (0)	0.5 (2)	0.3 (1)	0.6 (2)	0.2 (1)	1.3 (7)	0.2 (1)	2.1 (8)
no cause given/blank	1.6 (2)	0.0 (0)	0.0 (0)	0.0 (0)	14.7 (31)	11.9 (30)	2.2 (6)	3.8 (12)	2.8 (9)	24.9 (93)	6.2 (23)	2.8 (10)	1.0 (4)	5.2 (27)	2.6 (17)	2.4 (9)

Source: See Figure A1.

Note: Each cell provides the PNMR and, in parentheses, the number of deaths on which it is based.

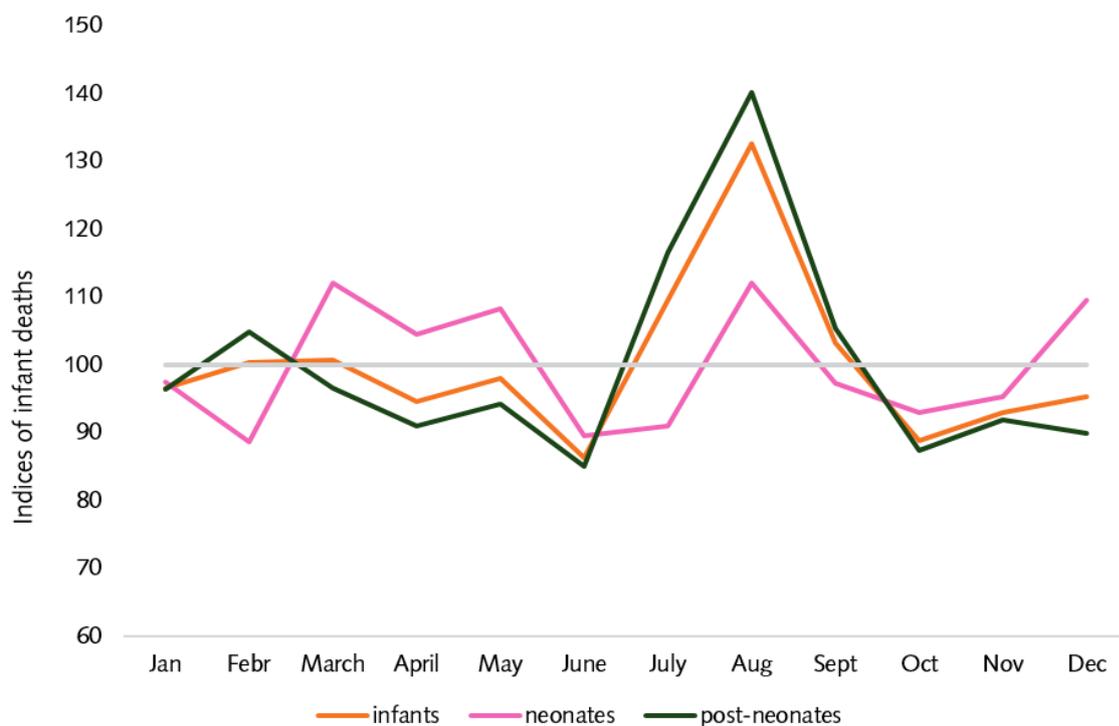
A9 FEMALE NMR, MALE NMR, FEMALE PNMR, MALE PNMR BY CAUSAL GROUP, TRONDHEIM 1830–1907

Causal groups	Male NMR	Female NMR	Male PNMR	Female PNMR
congenital and birth disorders	11.8 (314)	8.2 (210)	2.7 (71)	2.9 (73)
weakness	1.2 (31)	1.1 (29)	2.3 (60)	2.3 (60)
convulsions	12.6 (335)	9.8 (252)	19.8 (526)	17.0 (436)
water-food borne	2.6 (68)	2.4 (62)	18.2 (484)	16.3 (418)
teething	0.0 (0)	0.0 (0)	1.3 (34)	1.2 (31)
air-borne	2.5 (66)	2.4 (62)	34.1 (907)	32.2 (824)
other infectious	0.8 (20)	1.1 (27)	4.2 (113)	3.5 (90)
other non-infectious	2.2 (59)	1.8 (46)	7.9 (211)	6.7 (171)
external causes	0.3 (8)	0.4 (10)	0.6 (15)	0.2 (5)
ill-defined and unknown	1.1 (30)	0.5 (13)	2.7 (71)	2.4 (61)
stated to be 'unknown'	0.6 (15)	0.4 (10)	0.6 (16)	0.5 (13)
no cause given/blank	4.0 (106)	2.9 (73)	5.4 (144)	5.0 (129)

Source: See Figure A1.

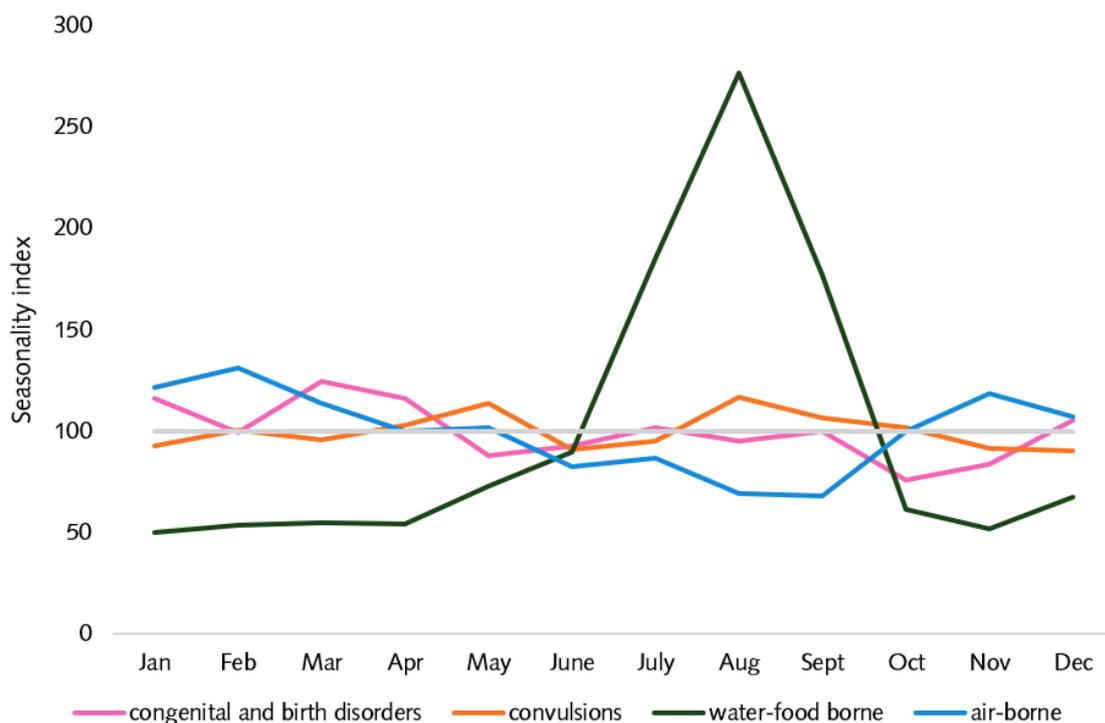
Note: In parentheses, absolute numbers of deaths.

A10 SEASONALITY INDICES FOR DEATHS AMONGST INFANTS, NEONATES AND POST-NEONATES, TRONDHEIM 1830–1907, ALL YEARS COMBINED



Source: See Figure A1.

A11 SEASONALITY INDICES FOR THE TOP-FOUR CAUSE OF DEATH GROUPS AMONGST INFANTS, TRONDHEIM 1830–1907, ALL YEARS COMBINED



Source: See Figure A1.