What was Killing Babies in Rostock? An Investigation of Infant Mortality Using Individual-Level Cause-of-Death Data, 1800–1904

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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 Rostock?

An Investigation of Infant Mortality Using Individual-Level Cause-of-Death Data, 1800–1904

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ABSTRACT

This paper examines the causes of infant mortality for the Hanseatic city of Rostock, Germany, between 1800 and 1904. Based on unique individual-level church records from Rostock's largest inner-city parish, St. Jakobi, we apply the novel ICD10h coding system for the first time to the German context. Using this coding system, we analyse cause-specific patterns of infant, neonatal and post-neonatal mortality in an internationally comparable way and bring new insights into the determinants of 19th-century infant mortality, which was shaped by increase and stagnation in wide parts of Germany. Our results show that Rostock experienced a stagnating infant mortality rate at a low level in international comparison during the first 40 years of the 19th century, followed by severe increases during the next 20 years and a stage of slight decline and stagnation towards the end of the study period. This suboptimal development from 1840 was strongly related to post-neonatal mortality and causes of death that are related to unfavourable sanitary conditions and/or poor nutrition, which possibly hints at worsening housing and living conditions following accelerated population growth. Our analyses also reveal that water-food borne diseases were underestimated in Rostock, even though symptomatic disease terms such as convulsions and teething, that were frequently recorded over much of the 19th century, had deviating seasonality patterns and thus cannot entirely refer to this disease group but rather to a wide field of different diseases. The applied coding scheme is a significant step forward to foster comparative international research on historical cause-specific mortality.

Keywords: Infant mortality, Neonatal and post-neonatal mortality, Causes of death, Historical demography, 19th Century, Germany

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1 INTRODUCTION

As a country that arose from many small states, Germany has experienced diverse regional contrasts over the last centuries. These contrasts were especially visible in the 19th century — a time that was shaped by far-reaching societal transformation processes like political changes, industrialisation, urbanisation and the demographic and epidemiologic transitions. These developments affected trends and regional patterns of infant mortality. For example, the German-wide infant mortality rate (IMR) never fell below 200 per 1,000 live births during the 1800s, seemingly higher than many Northern and Western European countries (Gehrmann, 2011). However, regional differences in Germany show large variations: higher rates in the south, often attributed to differences in breast feeding practices (Kintner, 1988), while the northern areas of Schleswig-Holstein and Mecklenburg had overall IMRs lower than Sweden until 1865. Conversely, while Sweden and Norway showed declines in much of the 19th century, Germany as a whole as well as its northern part experienced increases in infant mortality during the third quarter of the century, followed by stagnation in the last quarter (Gehrmann, 2011). Therefore, northern Germany increasingly lost pace with the Scandinavian countries, especially after 1875. Moreover, large variations between urban and rural areas of Germany existed; not only did the overall level of infant mortality vary, declines generally began earlier in urban areas in the south and rural areas in the north relative to their regional rural and urban counterparts (Knodel, 1977; Vögele, 1994). In fact, the largest cities in Germany experienced IMRs between 150 and 380 at the end of the 1870s, decreasing to values between 100 and 180 by 1913 (Vögele, 1993).

As a growing urban area in northern Germany, the Hanseatic city of Rostock was among the latest German regions to experience a significant decline in infant mortality, which did not occur until the 20th century (Mühlichen, 2020; Mühlichen et al., 2015). Exploring the causes that led to this suboptimal trend in Rostock might allow indications for a broader context — as the city experienced a similar trend like the rest of northern Germany — and thus contribute to the understanding of infant mortality stagnation and decline in the 19th century. In particular, cause-of-death data could help to understand what went wrong in comparison to proximate countries like Sweden. Were the diverging trends driven by particular causes that give deeper insights into the living environments of the respective time? How did cause-of-death patterns change over the course of the century?

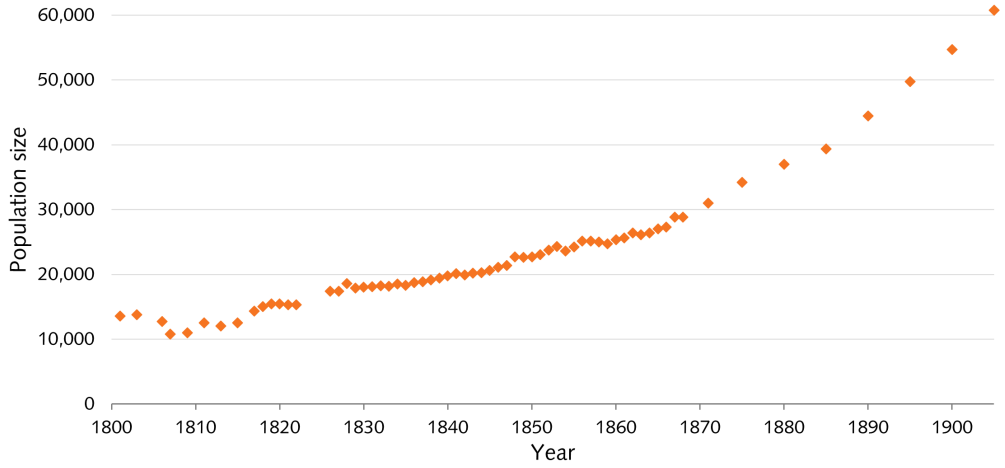
Using the recently transcribed individual-level church records from the city of Rostock (Mühlichen, 2020; Mühlichen & Scholz, 2015) and applying the newly developed ICD10h disease coding scheme (Janssens, 2021), we aim to broach these questions by calculating cause-specific rates of overall, neonatal, post-neonatal and seasonal infant mortality for the period from 1800 to 1904. In connection with the other articles of this special issue, we also aim to test the new ICD10h coding scheme and gain new insights into the causes for the different infant mortality trends across Europe.

2 BACKGROUND AND DATA

2.1 THE CITY OF ROSTOCK

Rostock is located on the Baltic coast and has been the biggest and economically most important city in the northern German Mecklenburg region. Being one of the earliest and most influential members of the Hanseatic League, the city is shaped by its port, its university and its long-standing Hanseatic trading tradition (Dollinger, 1970). Like most of northern Germany, the city was overwhelmingly Protestant, with very small numbers of Catholics, Jews and Evangelical minorities living in the city, as historical census data reveal (RAPHIS, 2016). Rostock's population grew continuously but slowly in the first two thirds of the century, followed by accelerated growth in the last third due to the emerging processes of urbanisation and (moderate) industrialisation (Manke, 2000; Szołtysek et al., 2011). During the study period, the population increased from 13,556 in 1801 to 60,790 in 1905 (Figure 1). Up to 1872, Rostock's population growth was driven primarily by in-migration (particularly by young adults from Mecklenburg's rural subclasses), while natural increase became even more important thereafter (Szołtysek et al., 2011). The share of people born outside of the city rose from 39% in 1819 to 56% in 1900. Among people of working age, the share even increased to 70% until 1900, while it remained relatively low among children (Szołtysek et al., 2009, 2011).

Figure 1 *Population development in Rostock, 1801–1905*



Source: Local statistical office of the Hanseatic City of Rostock; Mecklenburg-Schwerin State Calendar

According to previous research, infant mortality in 19th-century Rostock was slightly higher in comparison to the rural areas of Mecklenburg but lower than in most other German regions, and roughly followed the northern German trend (Brüning and Balck, 1906; Gehrmann, 2011; Mühlichen, 2020; Mühlichen et al., 2015; Paulsen, 1909; Prinzing, 1900; Toch et al., 2011). Infant mortality in Rostock remained, relative to the time, low until 1840 but shifted upwards in the early 1840s and late 1850s. This increase was followed by a period of stagnation until the early 20th century (Mühlichen, 2020; Mühlichen et al., 2015). By 1910, the total IMR of Rostock combined with its encompassing region had decreased to roughly 136 infant deaths per 1,000 live births, which was lower than the German and European means (166 and 173, respectively, as estimated based on data from Klüsener et al. (2014)). Following recurring cholera epidemics due to contaminated water, Rostock established its first waterworks in 1867, though they soon exceeded its limits due to accelerated population growth, thus requiring the construction of a new one, which was completed in 1894 (Rathaus Rostock, 2017; Uffelmann, 1889).

2.2 DATA AND METHODS

Measuring infant mortality rates (IMRs) requires data on live births and infant deaths. Since civil vital and cause-of-death registration were not yet introduced in Rostock at the time under study, we analysed the recently transcribed and harmonised individual-level burial and baptismal registers of Rostock's largest parish, St. Jakobi, for the period from 1800 to 1904. The baptismal registers of St. Jakobi include the population at risk, which contain all live births registered in the parish during the study period, and the burial registers include all infant deaths recorded in the parish. More information on the data sources are given in Mühlichen and Scholz (2015) and Mühlichen (2020). St. Jakobi was by far the largest of the four old Rostock parishes and constituted Rostock's New Town in the west of the city's historic centre. Other parishes encompassed Rostock's Middle Town (St. Marien) and Old Town (St. Nikolai and St. Petri). Between 1827 and 1875, the share of Rostock's live births born in St. Jakobi rose steadily from 39 to 61%. The parish was probably also the most heterogeneous one in terms of social structure (Mühlichen & Scholz, 2015; Szołtysek et al., 2011), as also indicated by the old popular rhyme "Marien reich, Jakobi gleich, Nikolai arm, Petri Gott erbarm", which describes St. Marien as wealthy, St. Jakobi as balanced, St. Nikolai as poor and St. Petri as particularly poor ("God, have mercy").

As common for historical demographic data, there are several limitations. First, we cannot ascertain whether there were (informal) rules for the differentiation between live births and stillbirths and whether it varied over time or between social groups. However, we could not identify any irregularities or sudden shifts in the development of live births and stillbirths during the study period. Stillbirths and unbaptised infant deaths were consistently recorded in the burial registers throughout the observation period but also in the baptismal registers, at least until 1876. Second, we cannot determine how many children left the parish and died elsewhere in the first year of life. Therefore, we cannot rule out that IMRs might be underestimated. At least, in-migrated infants are identifiable by the given place of birth. Third, causes of death were recorded in absence of any systematic cause-of-death classification. Therefore, the accuracy and consistency of the recorded causes was not optimal. However, the recorded causes became considerably more specific and diverse towards the end of the 19th century.

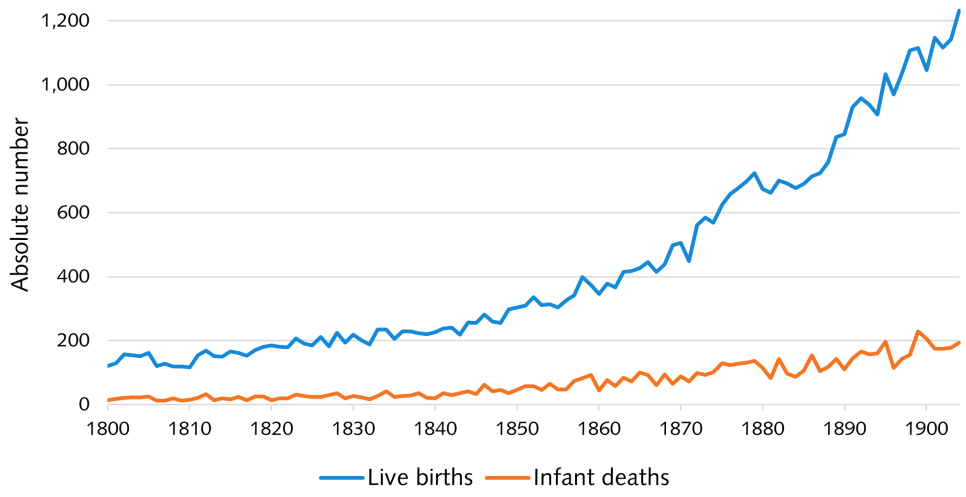
Furthermore, the diagnoses were not recorded by a medical doctor but by the pastor of the parish, who likely had some rudimentary medical knowledge, as the variety of recorded diagnoses, at least in post-neonatal deaths, and clerical statute books from the 19th century (Gesenius, 1839, 1848; Millies, 1895, 1896, 1910) indicate. Whether the pastors conferred with a physician in some instances is unclear but it is likely that the recorded cause of death is usually based on information given by the parents, e.g. with regard to symptoms (Radtke, 2002). Fourth, we cannot rule out group-specific under-reporting. However, we could not identify any problematic irregularities in this context in the burial and baptismal registers. To the contrary, it rather seems that the pastors were highly interested in gathering information on births and deaths as complete as possible, as even stillbirths and unbaptised infant deaths from all social classes and 'illegitimate' children were recorded including information on the likely or suspected father.

We drew all infant deaths by year, month, age, sex and cause of death from the burial registers and all live births every year from the baptismal registers of St. Jakobi, then calculated cause-specific neonatal, post-neonatal and overall infant mortality rates, as well as seasonality indices. We excluded both stillbirths and infants born in another place from the analyses. In total, 7,380 infant deaths (70.2 per annum) and 45,728 live births (435.5 per annum) were recorded in the study period. Figure 2 highlights the increase of the annual numbers of live births (from 121 to 1,232) and infant deaths (from 14 to 193) between 1800 and 1904.

We applied ICD10h to code and classify the diagnoses given in the burial records. ICD10h is a novel European coding system for historical causes of death that has been designed by members of the SHiP+ network (Janssens, 2021). More information on both the network and the coding system is presented in the introduction to this special issue. Concerning 'multiple' causes of death, we decided to classify them according to the first given cause, except for diagnoses where the first term refers to a symptom only and the latter one being more specific. The complete list of causes with the harmonised German expression, its English translation, the causal grouping, the ICD10h code and its frequency and share in the study period is shown in Table 1 in the appendix.

In the following sections, we provide a detailed overview of infant mortality differentiated by sex, cause of death, age at death (neonatal and post-neonatal) and month of death. IMRs as well as neonatal and post-neonatal mortality rates (NMRs and PNMRs) are calculated as infant deaths per 1,000 live births. When shown, confidence intervals are calculated under the assumption that the incidence rates follow a Poisson distribution. Neonatal mortality refers to deaths of infants aged 0 to 28 days of life, while post-neonatal mortality includes infant deaths from 29 days upwards. Seasonality indices describe the relative risk of infant mortality for each month differentiated by cause of death.

Figure 2 *Number of infant deaths and live births in St. Jakobi, Rostock, 1800–1904*



Source: Baptismal and burial registers of St. Jakobi, Rostock; authors' calculations.

3 INFANT MORTALITY

The development of infant mortality in 19th-century Rostock is shaped by three stages (Figure 3). First, there is a stage of low infant mortality in the period between 1800 and 1839, when the IMR decreased from 136 to 122 infant deaths per 1,000 live births (in quinquennial values). The second stage is characterised by an upward shift to 197 infant deaths between 1840 and 1859. The early 1840s and late 1850s particularly contributed to this shift, which peaked in 1859 with an IMR of 265 among boys and 230 among girls (see Figure 10 in the appendix for annual values). In the third stage, from 1860 onwards, infant mortality remains at this higher level — as compared to the first stage — but tends to slightly decrease, with IMR declining to 163 in the years 1900–1904.

Distinguished by sex, infant mortality was almost entirely higher among boys than for girls. This gap was rather small in the first 30 years and between 1850 and 1864, and particularly pronounced in the 1870s and between 1895 and 1904. The shift of infant mortality in the 1840s and 1850s is true for both sexes but the timing is different. For males, a slight increase is already visible in the mid-1830s, which is then followed by a major shift in the early and mid-1840s. Among females, these increases came a few years later, in the 1840s and 50s. However, the IMR for females began to decrease after the peak in 1859, whereas the IMR for males remained at a high level, then reached a further peak in the late 1870s with an IMR of 213. Subsequently, there was a sharp decline in the early 1880s but another increase at the end of the 19th century. For all years combined, the IMR was 175 for boys, 147 for girls and 161 in total.

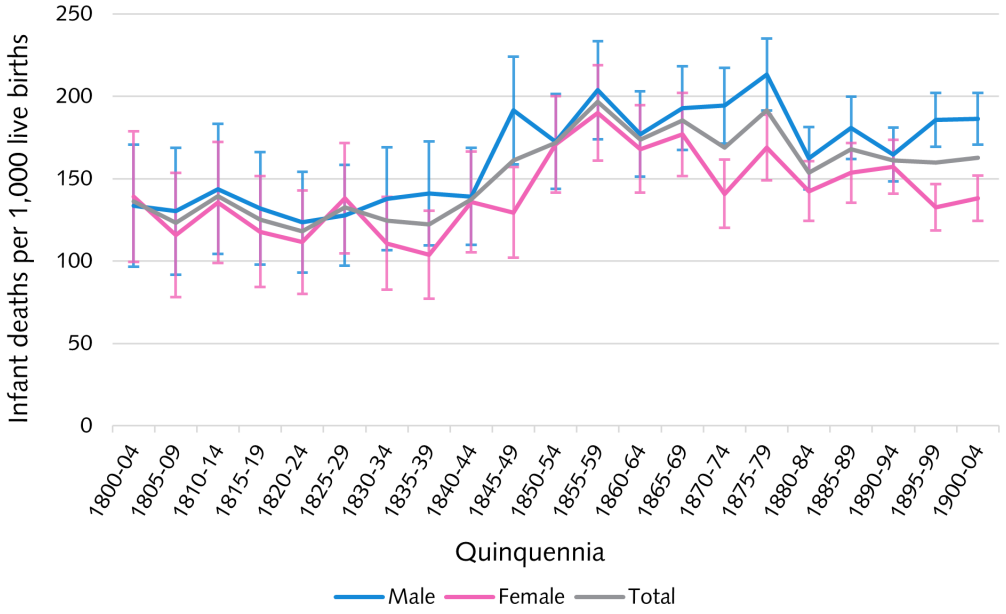
In general, most German regions showed an upward shift in infant mortality over the course of the 19th century, before ultimately beginning to decline, albeit at varying times. Only the central German region of Hessen-Nassau followed the Northern European 'model' of continuous decline in infant mortality throughout the 19th century (Gehrmann, 2011; Mühlichen, 2020). The infant mortality trend for Rostock is similar to those observed for other northern German regions like Prussia, Schleswig-Holstein and the Hanseatic cities of Hamburg and Bremen, but the timing of the three stages (low infant mortality, increases, stagnation/slight decline) is different (Gehrmann, 2011). Although situated in the western part of northern Germany, Bremen, which previously also had low levels of infant mortality relative to the rest of Germany, experienced an increase in mortality from the middle of the 19th century to a peak in 1886, before beginning its ultimate decline (Lee & Marschalck, 2012). This similar pattern of rise in infant mortality in the 1860s and 70s also occurred in Hamburg and Berlin (Gehrmann, 2011; Vögele, 1993).

In Rostock, the rise of infant mortality levels led to a diversion from some other European port cities, which either began to decline earlier, despite initially higher levels (Amsterdam and Copenhagen), or for which no mid-century uptick was observed (Trondheim) (Janssens & Riswick, 2023; Ludvigsen et al., 2023; Sommerseth, 2023). However, upward shifts during the 19th century were not unique to Germany, as the examples of Palma de Mallorca and Hermoupolis show (Pujades Mora, 2009; Raftakis, 2021, 2022).

Rostock's peak in infant mortality in 1846 coincided with the Europewide 'potato murrain' epidemic that destroyed a major part of the harvest in Mecklenburg and led to excess mortality, pauperisation and migration from rural areas to the cities or other countries (Zadoks, 2008). Thus, this might have affected Rostock's population structure through changes in mortality and migration patterns and consequently led to changes in its sanitary, housing, working and/or nutritional conditions. In the 1850s, Rostock increasingly struggled with contaminated water, which led to recurring cholera epidemics in the 1850s (Rathaus Rostock, 2017; Uffelmann, 1889). Deterioration in infant care, e.g. through declining breastfeeding practices and increasing use of substitute or contaminated food, could be a possible reason as well, even though we could not find evidence in the literature. In the following analyses, we aim to provide new insights into this development.

The differentiation by causal groups (Figure 4) shows that the increase of infant mortality in the late 1850s, as well as the slight decrease beginning in the 1880s, are largely driven by trends in the causal group of convulsions. The decrease of convulsions from the 1880s and the elimination of teething towards the end of the study period were accompanied by increases of water-food borne diseases and, to a lesser extent, air-borne diseases and weakness. The groups of congenital and birth disorders, external causes as well as other infectious, ill-defined, unknown and unspecified ('blank') diseases were largely absent. Therefore, we decided to merge the most similar of the rarest disease groups (ill-defined, unknown and blank) for our analyses. The majority of deaths in this group were blank, as detailed in Table 1 in the appendix.

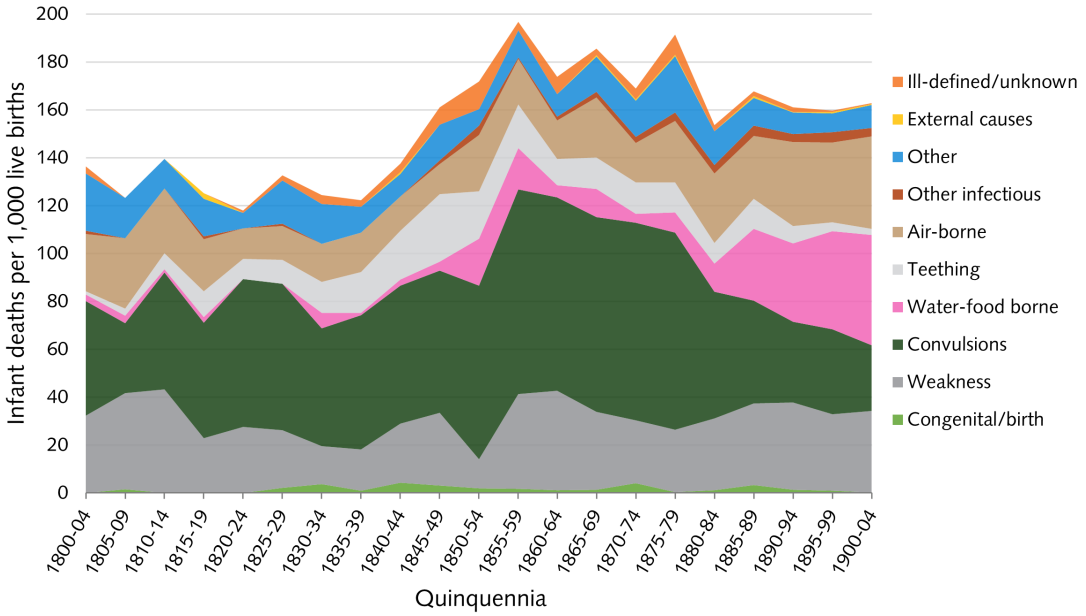
Figure 3 *Infant mortality rate by sex in Rostock, St. Jakobi parish, 1800–1904 (quinquennia)*



Note: The error bars show 95% confidence intervals.

Source: As for Figure 2.

Figure 4 *Infant mortality rate by cause-of-death group in Rostock, St. Jakobi parish, 1800–1904 (quinquennia)*



Source: As for Figure 2.

As a cause of death, air-borne diseases increased in the late 19th century, which is mainly related to respiratory infections like pneumonia and lung catarrh. Deaths from measles also increased during this time but to a lesser extent. The last infant death from smallpox appeared in 1871, three years prior to the introduction of mandatory immunisation against smallpox for children aged 1 to 12 in the German Empire (Thießen, 2013).

The upward shift of infant mortality from convulsions and weakness is hard to interpret, as these causal groups refer to symptoms instead of actual diseases and are therefore ill-defined, just like teething. However, given the information from literature that Rostock increasingly struggled with contaminated water in the

1850s and, as a consequence, experienced recurring cholera epidemics (Rathaus Rostock, 2017; Uffelmann, 1889), we can assume that these two causal groups may partly cover deaths from waterborne diseases associated with epidemic outbreaks. We further investigate this in the following sections.

4 NEONATAL AND POST-NEONATAL MORTALITY

The upward shifts in infant mortality in the 1840s and 1850s are larger and more considerable in post-neonatal than neonatal mortality, suggesting an increase in the effect of environmental disadvantages for infants. The post-neonatal mortality rate (PNMR) was consistently higher than the neonatal mortality rate (NMR) throughout the study period but the gap increased over the course of the 19th century, especially after the peak in 1855–1859, when the PNMR remained on a high level reaching its peak in 1875–1879, while the NMR steadily decreased over the last 40 years of the century (Figure 5). Moreover, the relative male-female gap to the disadvantage of boys was in most years stronger in neonatal mortality than in post-neonatal mortality.

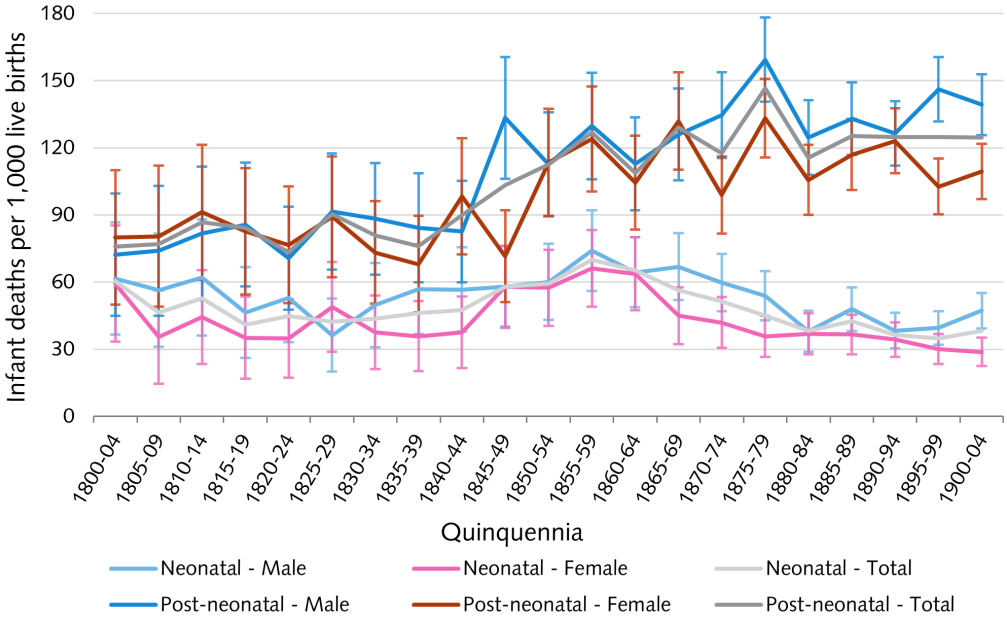
That the total share of neonatal deaths was particularly high in the first 40 years of the century makes it highly unlikely that the low infant mortality level in this time was caused by an under-reporting of early neonatal deaths, for instance by changes in differentiating live births from stillbirths. The relative stability of the total level of neonatal deaths throughout the observation period further reflects that there were likely no substantial changes to the way these deaths were recorded during the time of study.

The levels and trends of the NMR and PNMR in Rostock are very similar to Bremen and Copenhagen. The pattern of decreasing neonatal mortality and stagnating post-neonatal mortality towards the end of the century is also present in Bremen and Copenhagen, though only until 1895, when post-neonatal mortality started to drop in the Danish capital (Lee & Marschalck, 2002; Ludvigsen et al., 2023). Similar to Rostock, Copenhagen and Amsterdam experienced a slight decline of neonatal mortality after 1860 (Janssens & Riswick, 2023; Ludvigsen et al., 2023). However, in contrast to Rostock, Amsterdam showed lower neonatal and higher post-neonatal mortality for a major part of the study period beginning in 1856. Due to the decrease of the PNMR in the mid-1890s, both Amsterdam and Copenhagen reached a lower level compared to Rostock by the turn of the century (Janssens & Riswick, 2023; Ludvigsen et al., 2023). In general, both higher levels of post-neonatal than neonatal mortality and higher mortality for boys is characteristic for the 19th and early 20th centuries throughout other European cities (Garrett & Reid, 2022; Janssens & Riswick, 2023; Ludvigsen et al., 2023; Raftakis, 2022; Sommerseth, 2023). In Rostock, this sex gap was comparatively small during the first half of the 19th century.

From a cause-specific perspective, neonatal mortality was dominated by the causal groups of weakness and convulsions, though the particular level of each of these causes varied throughout the study period (Figure 6). Following the peak of infant mortality in the 1850s, the decrease of deaths related to convulsions was accompanied by an increase in water-food borne diseases and more deaths registered as weakness. As expected, due to the usual physical development of infants, the causal group of ‘teething’ is nearly non-existent in neonatal mortality showing only three cases over the entire period.

The temporary decreases of weakness in 1835–1839 and 1850–1854 are probably partially related to the temporary increases of 'blanks' (i.e. no cause recorded) which occurred at the same time (included in the group of ill-defined/unknown diseases). The decline in weakness in 1835–1839 was also accompanied by an increase in ill-defined/unknown and other causes, whereas the drop in weakness in 1850–1854 coincided with an increase in convulsions, water-food borne, air-borne and other infectious diseases. An increase in weakness in the early and mid-1840s, especially in neonatal but also, to a lesser extent, in post-neonatal mortality, contributed to the overall rise in infant mortality. The peak in neonatal mortality in 1855–1859 was also mainly driven by an increase in weakness. The share of deaths registered as weakness rose considerably from 1880, thus reducing variety in cause-of-death recording. Thus, the low variety and increasing dominance of weakness in the cause-of-death spectrum of neonatal mortality might point to a lack of interest in the causes of neonates’ deaths.

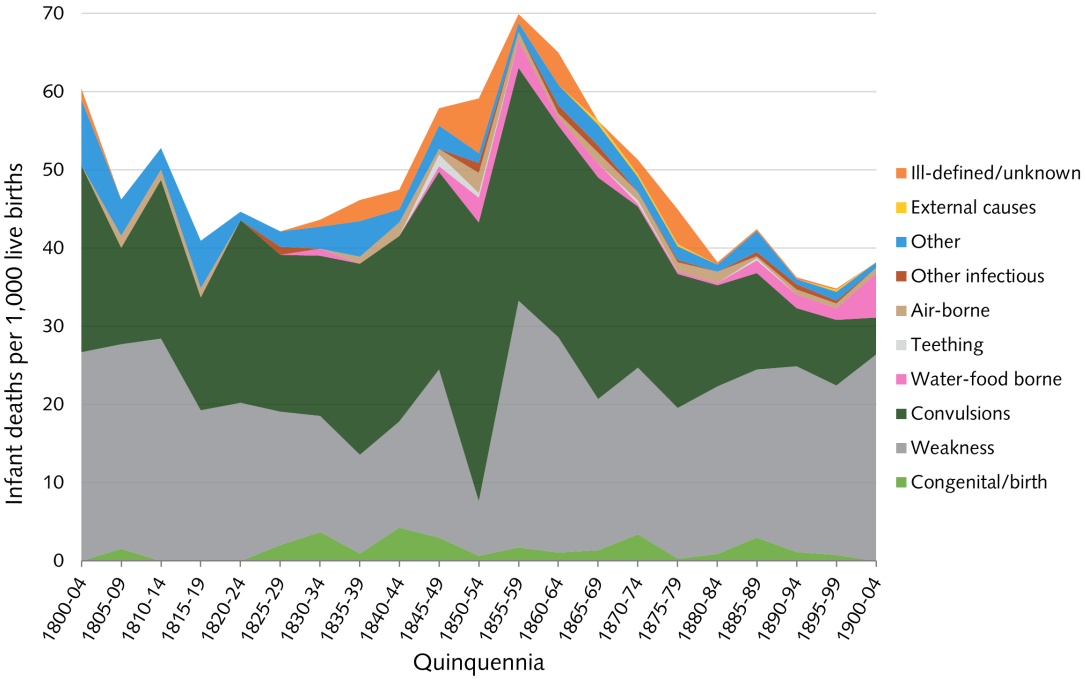
Figure 5 *Neonatal and post-neonatal mortality rate by sex in Rostock, St. Jakobi parish, 1800–1904 (quinquennia)*



Note: The error bars show 95% confidence intervals.

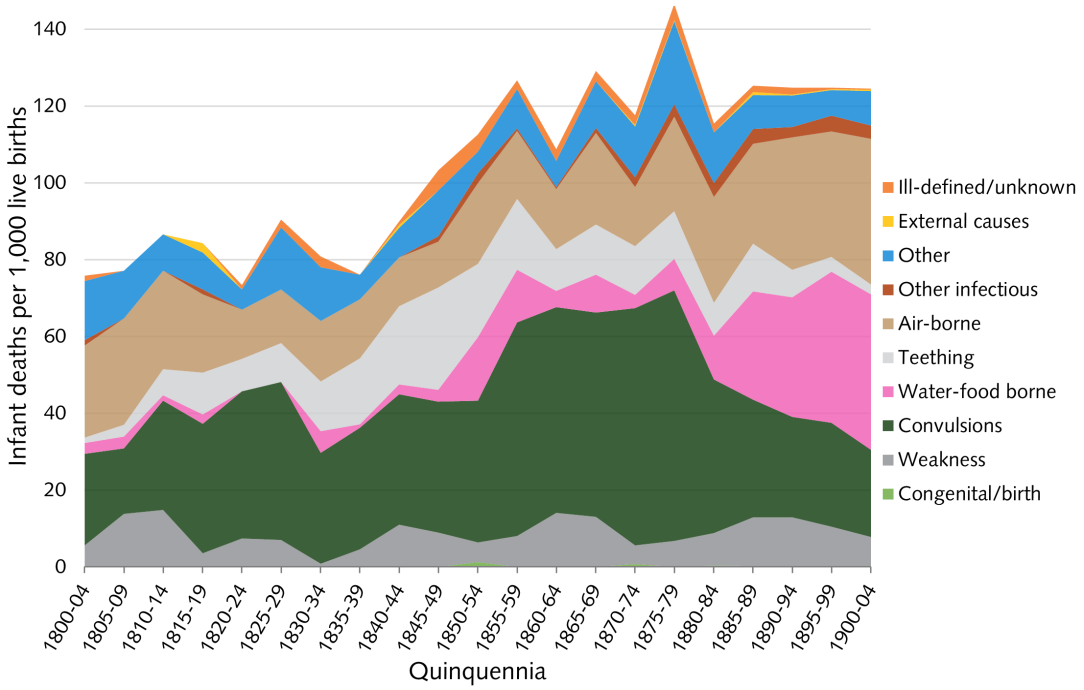
Source: As for Figure 2.

Figure 6 *Neonatal mortality rate by cause-of-death group in Rostock, St. Jakobi parish, 1800–1904 (quinquennia)*



Source: As for Figure 2.

Figure 7 *Post-neonatal mortality rate by cause-of-death group in Rostock, St. Jakobi parish, 1800–1904 (quinquennia)*



Source: As for Figure 2.

The pattern in post-neonatal mortality (Figure 7) is more diverse, with convulsions, water-food borne and air-borne diseases dominating the cause-of-death spectrum. Naturally, teething is more pronounced in post-neonatal mortality than in neonatal mortality. In post-neonatal deaths, increases in teething in the 1840s contributed most to the rise of infant mortality in this time. Convulsions were contributing the most to the high level of post-neonatal mortality between 1855 and 1879. They were overall more frequent in post-neonatal mortality than in neonatal mortality: 60% of all convulsions were recorded in the post-neonatal stage, 40% in the neonatal one, while the share of convulsions in the cause-of-death spectrum was widely similar in neonatal and post-neonatal mortality over the study period. Until the 1880s, convulsions represent the highest share of post-neonatal deaths as a cause. By contrast, the causal group of weakness was closely linked to neonatal mortality only and showed comparatively small numbers in post-neonatal mortality. Like in overall infant mortality, convulsions and teething decreased considerably towards the end of the study period, while at the same time water-food borne and, to a lesser extent, air-borne diseases gained in importance. In fact, the decline of convulsions from 1880 seems to directly relate to the increase in air and water-food borne diseases as causal groups. Convulsions are a symptom that can be caused by a variety of diseases, which include gastro-intestinal infections (e.g. cholera, dysentery or diarrhoea), air-borne infections (e.g. whooping cough or tuberculous meningitis), neurological diseases (e.g. encephalitis or epilepsy), other infections (e.g. neonatal tetanus) and metabolic disorders (Kintner, 1986; Lee & Marschalck, 2002; Prausnitz, 1901; Preston & Haines, 1991; Radtke, 2002; Schlossmann, 1897; Wasserfuhr, 1869). Therefore, the rise of water-food borne and air-borne diseases from 1885 onwards is not peculiar but rather may reflect improved conventions in cause-of-death attribution.

The plateau in infant mortality during the period from 1855 to 1879, which was strongly driven by convulsions, occurred at a time when Rostock struggled with contaminated water and cholera epidemics (Rathaus Rostock, 2017; Uffelmann, 1889). For this reason, a connection of convulsions with water-food borne diseases, which later in the century become a more frequently diagnosed causal group, seems likely, at least for this period. The pattern of increased water-food borne mortality was present across other port cities as well, such as Amsterdam, Trondheim and Copenhagen (Janssens & Riswick, 2023; Ludvigsen et al., 2023; Sommerseth, 2023). As the subsequent decline in infant mortality began, not only in Rostock, but also in Trondheim and Copenhagen, a decline in convulsions can be observed, which was, however, as opposed to Rostock, offset by an increase in deaths to congenital and birth disorders from 1880 onwards (Ludvigsen et al., 2023; Sommerseth, 2023).

Even though the construction and expansion of Rostock's waterworks in 1867 and 1894 most likely improved the sanitary conditions, infant mortality among neonates, who were predominantly breastfed only declined after 1860, similar to the case of Bremen (Lee & Marschalck, 2012; Uffelmann, 1889). While the construction of the waterworks was intended to provide universal access to clean water for the entire population, this goal likely became increasingly unrealistic following the growth of the city, which was partly driven by the in-migration of pauperised workers from the rural hinterland. Therefore, it is likely that the growing urban and industrial environment, coupled with non-universal access to clean water, increased housing density and increased probability to get in contact with infected people, hindered a sooner reduction of post-neonatal mortality. Moreover, in the whole of Germany and especially in urban areas, there is a documented general decline in both the overall percentage of breastfed infants, as well as average length of breastfeeding during the latter part of the 19th century (Kintner, 1985). During this period, additional feeding practices were often less nutritious and unsanitary relative to breastfeeding, therefore resulting in higher infant death rates (Vögele, 1994). As the combination of water-food borne diseases and convulsions remained on a high level in the late 19th century in spite of (presumably) improved water quality, it is also possible that factors like poor hygiene and malnutrition, perhaps due to a reduced level and duration of breastfeeding, played a part (Lee & Marschalck, 2002).

Weakness is a symptomatic cause, which did not decrease over time in the study, but is also quite common during this time period across Germany (Kintner, 1986; Vögele, 1994). In neonatal mortality, these diagnoses mostly refer to congenital and birth disorders, while in the context of post-neonatal mortality, weakness also includes terms like wasting and inanition, even though these terms were rarely recorded compared to 'weakness'. In the latter case, weakness likely refers to a wide field of different diseases, such as water-food borne, metabolic or cardiovascular diseases, similar to the findings for Derbyshire, England (Reid, 2001, 2002).

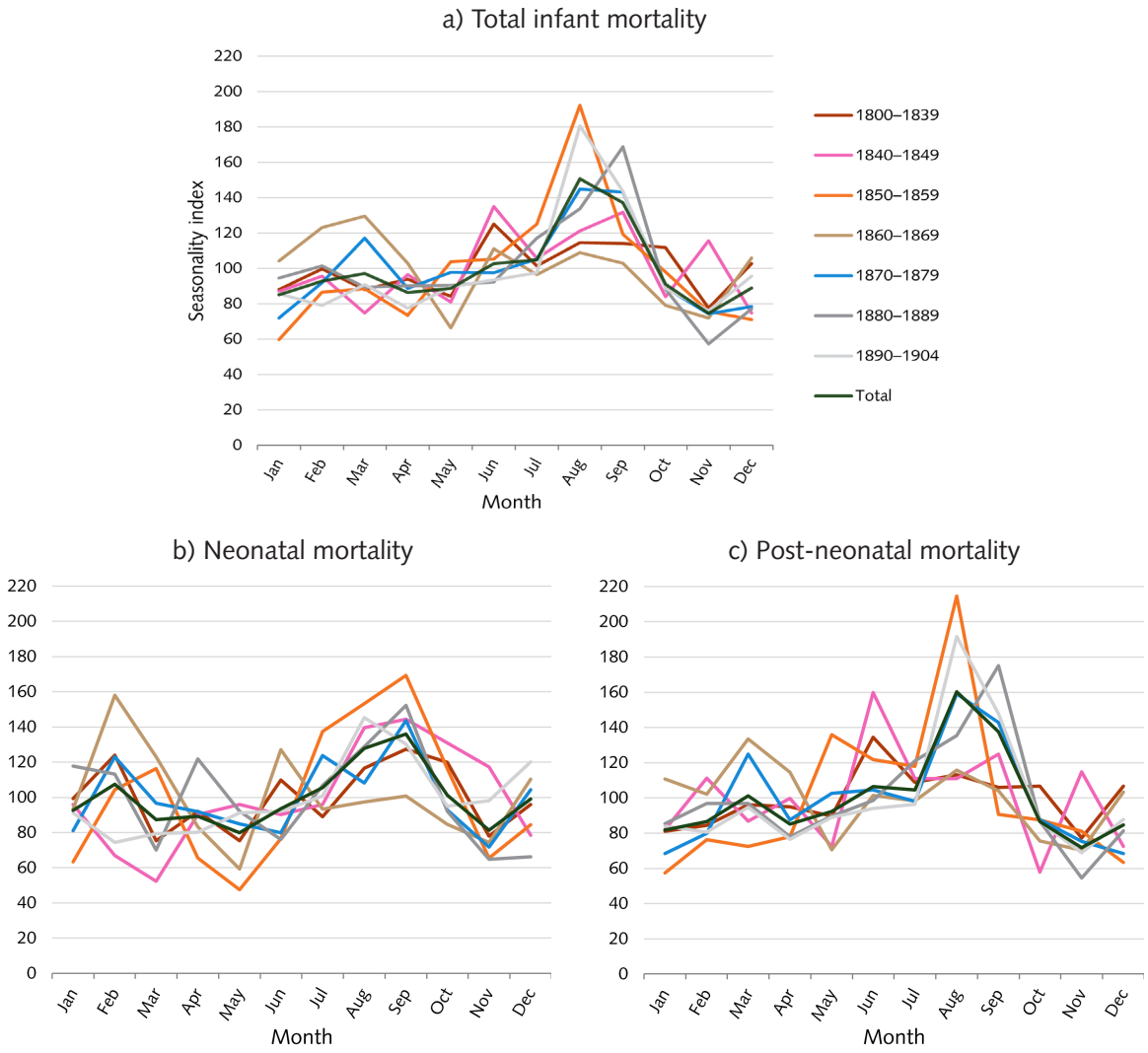
5 SEASONALITY OF NEONATAL AND POST-NEONATAL MORTALITY

The monthly distribution of infant deaths shows a strong summer peak, particularly in August and September (Figure 8). This is true for most decades, except for 1800–1839 and 1860–1869, and to a greater extent for post-neonatal mortality as compared to neonatal mortality. Winter peaks were also evident in several decades and generally more pronounced in neonatal than in post-neonatal mortality. However, with the exception of 1860–1869, they were less pronounced than the summer peaks. Over the course of the century, seasonal patterns became clearer. While seasonal patterns showed considerable variation in the first decades of the century, they increasingly stabilised from 1870 onwards showing strong summer peaks relative to the other times of the year.

As in Rostock, seasonality in Copenhagen for neonatal and post-neonatal mortality became more evident in the last decades of the 19th century, especially visible in high summer peaks (Ludvigsen et al., 2023). However, while seasonality in Rostock was generally correlated between neonates and post neonates — even if neonates experienced lower summer peaks and higher winter peaks — this pattern was not ubiquitous. Other cities such as Copenhagen and Amsterdam observed almost no seasonality for neonates (Janssens & Riswick, 2023; Ludvigsen et al., 2023), and seasonality patterns in other cities were reversed from the patterns observed in Rostock (Garrett & Reid, 2022; Raftakis, 2022; Sommerseth, 2023).

Taking causes of death into account, the summer peak in neonatal mortality (for all years combined) was widely related to water-food borne diseases, which were recorded about 3.5 times more frequently in August (372) and September (336), respectively, as compared to the monthly average (Figure 9b). This pattern — linked to epidemic outbreaks — was also found for Trondheim, Amsterdam and Ipswich (Garrett & Reid, 2022; Janssens & Riswick, 2023; Sommerseth, 2023). In the most frequent causal groups of weakness and convulsions, Rostock experienced slight summer peaks as well. Air-borne diseases, which may be associated with winter illnesses, rose in February and March to about 1.75 times the average. Congenital and birth disorders were most frequently recorded in June (2.5 times the average). The other cause-of-death groups were very rare and should therefore be interpreted with caution.

Figure 8 *Seasonality index for neonatal, post-neonatal and total infant deaths by month of death in Rostock, St. Jakobi parish, 1800–1904 by period*

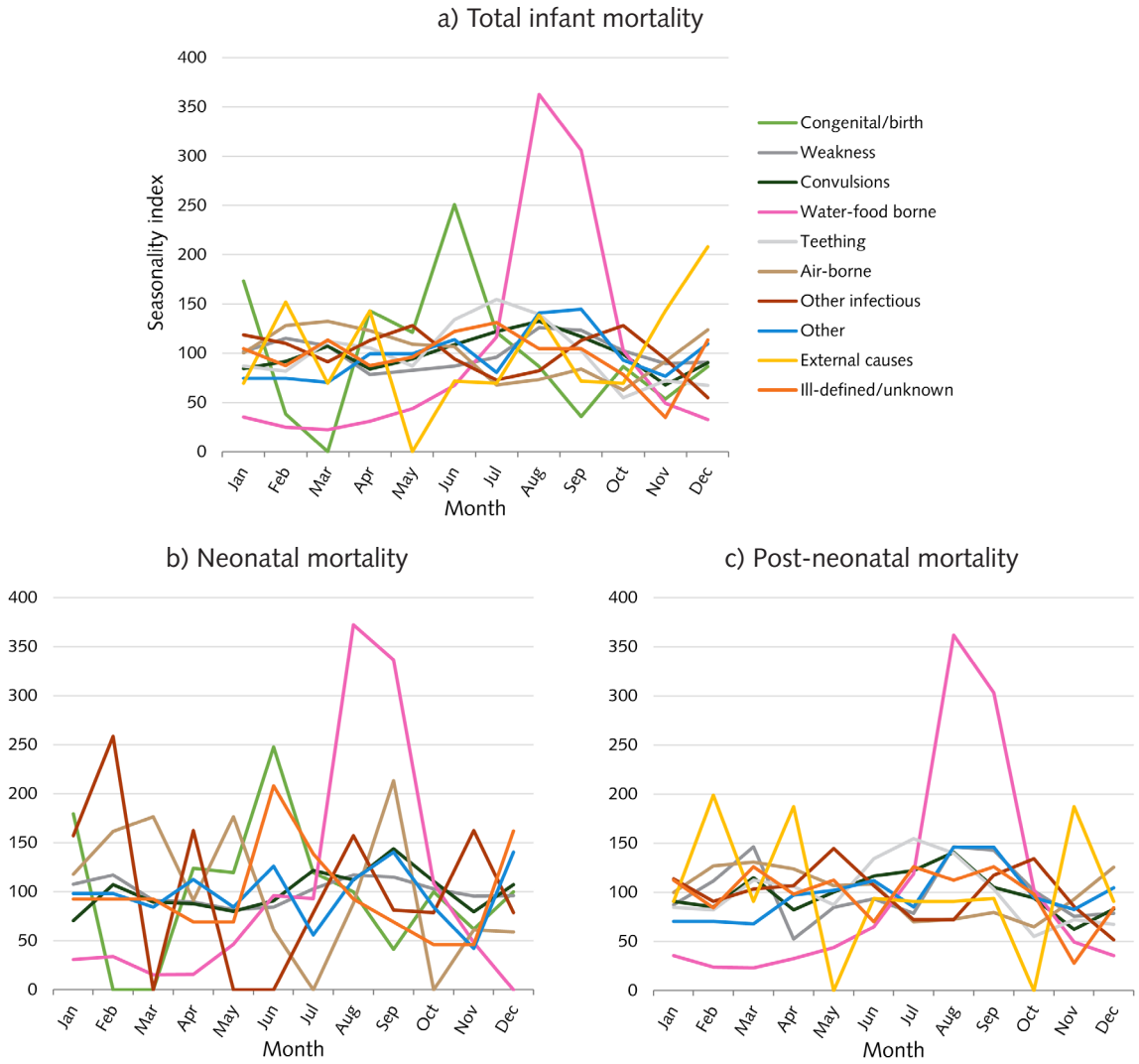


Source: As for Figure 2.

Very similar to neonatal mortality, the summer peak in post-neonatal mortality was most pronounced in water-food borne diseases, showing a seasonality index of 362 in August and 303 in September (Figure 9c). Moderate summer peaks were evident in teething, convulsions, weakness and other non-infectious diseases, peaking each at about 150. More detailed analyses by decade revealed little variation for convulsions and teething, mostly peaking at values between 150 and 200 in one of the warm months of the year. The winter peak was most pronounced in air-borne diseases peaking at 130 (in December, February and March) and in weakness (150 in March), but this is to a much lesser extent than what was observed in neonatal mortality.

The seasonal pattern shows that there was a moderate summer peak in convulsions in neonatal and post-neonatal mortality but not nearly as strong as for water-food borne diseases. The same is true for teething in post-neonatal mortality. Therefore, these two causal groups most likely do not completely refer to water-food borne diseases. On the other hand, a stronger connection to air-borne diseases is unlikely, as there is only a weak winter peak in convulsions and teething. Overall, the results indicate that there are likely some infant deaths 'diagnosed' with both convulsions and teething that were in fact related to water- or food-borne infection, particularly in post-neonatal mortality. Nonetheless, there are likely also cases in these two symptomatic causal groups that relate to causes that show a completely different seasonal pattern, such as air-borne and other diseases. This might be an indication that these two causal groups were used as some kind of container category for various causes that were not fully understood at that time but had — at least in the case of convulsions — similar symptoms.

Figure 9 *Seasonality index for neonatal, post-neonatal and total infant deaths by month and cause of death in Rostock, St. Jakobi parish, 1800–1904 (all years combined)*



Source: As for Figure 2.

Figures 11 and 12 in the appendix show the seasonal distribution of causes of death for four sub-periods. These are, on the one hand, the first two stages of infant mortality development identified in chapter 3 (1800–1839 and 1840–1859). On the other hand, we divided the third stage in two parts due to changes in the cause-of-death spectrum (1860–1879 and 1880–1904). These figures have to be interpreted with caution, however, as many numbers are low, particularly with regard to the earlier periods and neonatal mortality. Overall, the seasonal trends do not differ substantially by period. There were strong summer peaks in water-food borne diseases and moderate summer peaks in convulsions and (post-neonatal) teething in every period, while seasonal peaks in weakness varied by period.

6 CONCLUSIONS

This paper presents a comprehensive analysis of infant mortality trends in the Hanseatic city of Rostock over the course of the 19th century. Until around 1840, the level of infant mortality was similar to that or slightly below northern countries like England and Wales, which, as similar to most German regions, did not experience decline until the early 20th century, and Sweden, in which, however, infant mortality decreased almost continuously over the 19th century (Gehrmann 2011; McKeown & Record, 1962). Rostock's infant mortality rate (IMR) then exhibited a significant upward shift in the 1840s and

50s, thus developing a gap compared to Northern European countries. However, similar trends have been observed for other German regions like Prussia, Schleswig-Holstein and the Hanseatic cities of Hamburg and Bremen. While these trends appear to be common in parts of Germany, the upward shift occurred with different timing and pace (Gehrmann, 2011).

The increase in Rostock's IMR in the middle of the century developed in both neonatal and post-neonatal mortality, but to a greater extent in the latter group, suggesting a growing effect of environmental disadvantages for infants following accelerated population growth coupled with surging population density. While neonatal mortality decreased from 1860 onwards, post-neonatal mortality remained high until the end of the study period in 1904. It is possible that migration to the city played a role in this increase and stagnation in more than one way. For example, higher fertility is often associated with higher infant mortality. If the growing population of Rostock and/or the large number of migrants to the city during the 1800s had generally higher fertility than previously, it is likely that there is some association between higher fertility and the lack of decline in infant mortality, especially in the face of unsanitary and crowded living conditions (Woods, 2003). In addition, migration contributed to a change in the socio-economic composition of Rostock's population. On the one hand, in-migration was widely associated with relatively poor young adults from the rural hinterland (Szołtysek et al., 2011). On the other hand, two new city quarters were established outside the city walls in the 1850s due to the continued growth of the city. The upper class of the city was particularly attracted to Steintor-Vorstadt, a mansion district close to Rostock's Old and Middle Town areas, while Kröpeliner-Tor-Vorstadt, which is close to Rostock's New Town (St. Jakobi) was intended for the working class. Even though the place of residence did not necessarily determine the belonging to a church parish, as people were free to choose, it is possible that the share of people with a lower social status increased in St. Jakobi as a result of this.

Our analyses also show that the reasons for the diverging trends for Rostock compared to the proximate Northern European countries are probably related to worsening sanitary conditions in the middle of the century, as shown by peaks in post-neonatal deaths from water-food borne diseases and convulsions, which also at least partly refer to water-food borne diseases. In spite of the efforts to improve Rostock's water quality by constructing and expanding Rostock's waterworks, the combined level of convulsions, teething and water-food borne diseases in neonatal and post-neonatal mortality did not generally decrease after 1880, suggesting the importance of other contributory factors, such as deficits in parental care regarding hygiene and nutrition. During the time of the study, Rostock's population grew in an accelerated way. The rise of air-borne diseases in the last quarter of the century may thus be seen as a hint of worsening housing and living conditions, which further contributed to the widening gap compared to the 'vanguard' regions in Northern Europe.

Moreover, the cause-specific analyses based on the newly developed ICD10h coding scheme reveal that medical knowledge and accuracy in diagnoses improved over the course of the 19th century in Rostock, as shown by the partial shift from 'symptomatic' diseases to more specific diseases. This is best visible in the groups of convulsions and teething that started to decrease considerably from the 1880s onwards, while water-food borne diseases rose, seemingly replacing them. Nonetheless, while it is therefore likely that convulsions and teething often referred to water-food borne diseases, diverging seasonal mortality patterns indicate that deaths from additional causes are also included in these two groups. The group of 'other non-infectious' diseases also exhibited a moderate summer peak. Given that within this group are several gastro-intestinal diseases, such as 'stomach disorder', this may actually also refer to some water-food borne diseases, and thus might be coded differently.

In neonatal mortality, the variety of diagnoses is considerably lower than in post-neonatal mortality, with the term 'weakness' dominating the causal spectrum. In other Northern European cities, such as Amsterdam, Copenhagen, Ipswich and Trondheim, the spectrum was rather dominated by congenital and birth disorders. This could indicate that neonatal deaths were given less attention, resulting in rather unspecific 'diagnoses', or that knowledge about congenital and birth disorders, which were likely behind the term 'weakness' in many neonatal deaths, was less prevalent in Rostock or at least among the pastors who recorded these deaths into the church registers.

As discussed, it is possible that the true extent of water-food borne diseases is highly underestimated, yet it remains difficult to quantify the full extent to which convulsions and other symptomatic causes may contribute to this underestimation. In this sense, the coding of causes of death on historical

records remains limited to what specifically is transcribed, as well as the contextual interpretation of that cause. Nonetheless, the new ICD10h coding scheme is a significant step forward to foster comparative international research on historical cause-specific mortality.

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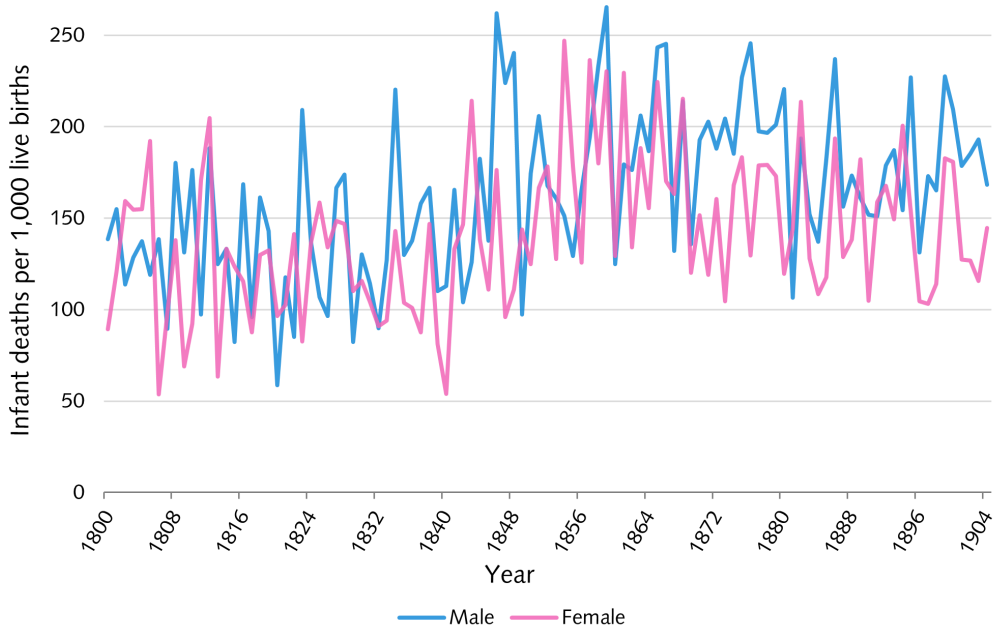
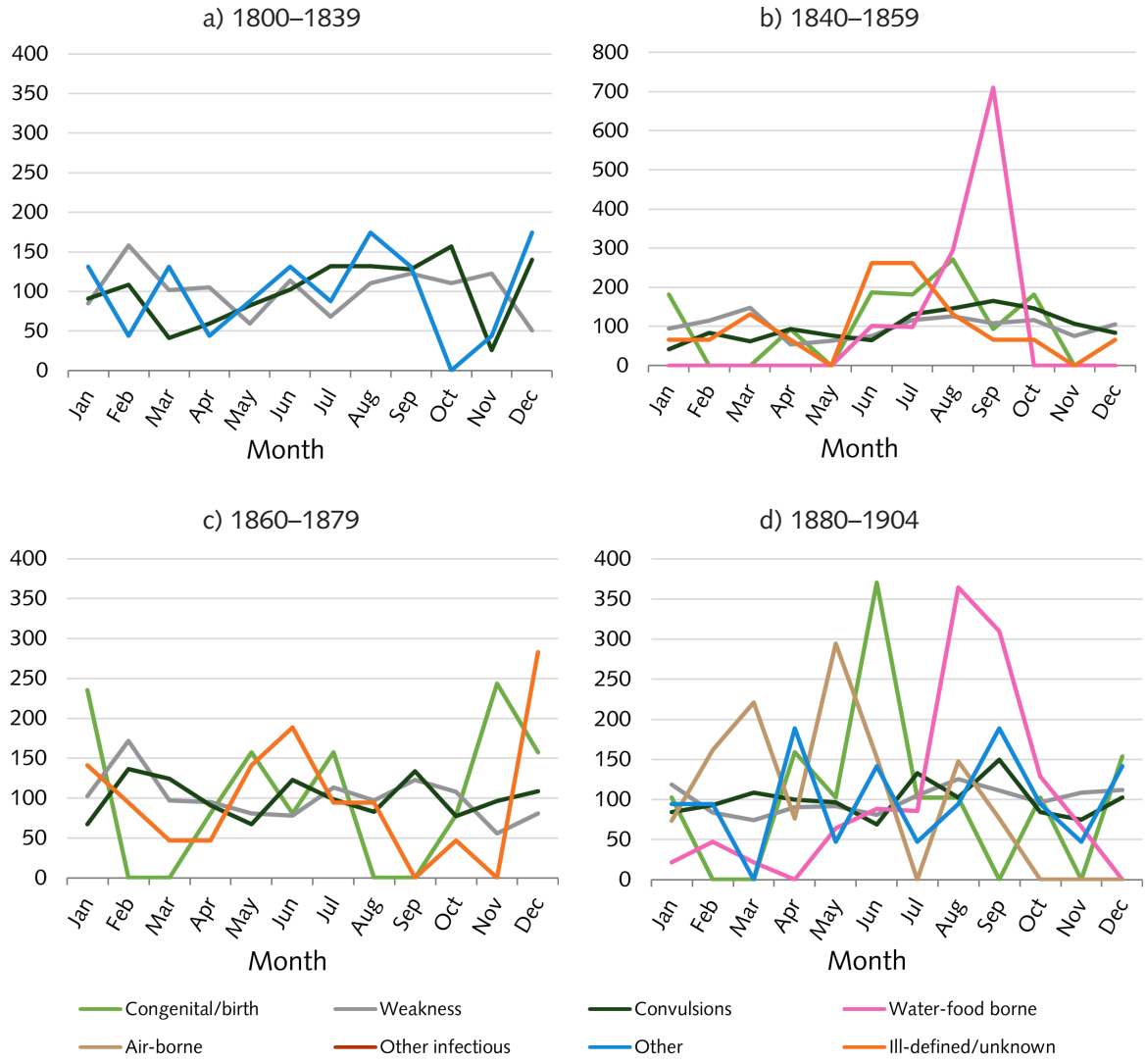
APPENDIX

Figure 10 *Infant mortality rate by sex in Rostock, St. Jakobi parish, 1800–1904*



Source: As for Figure 2.

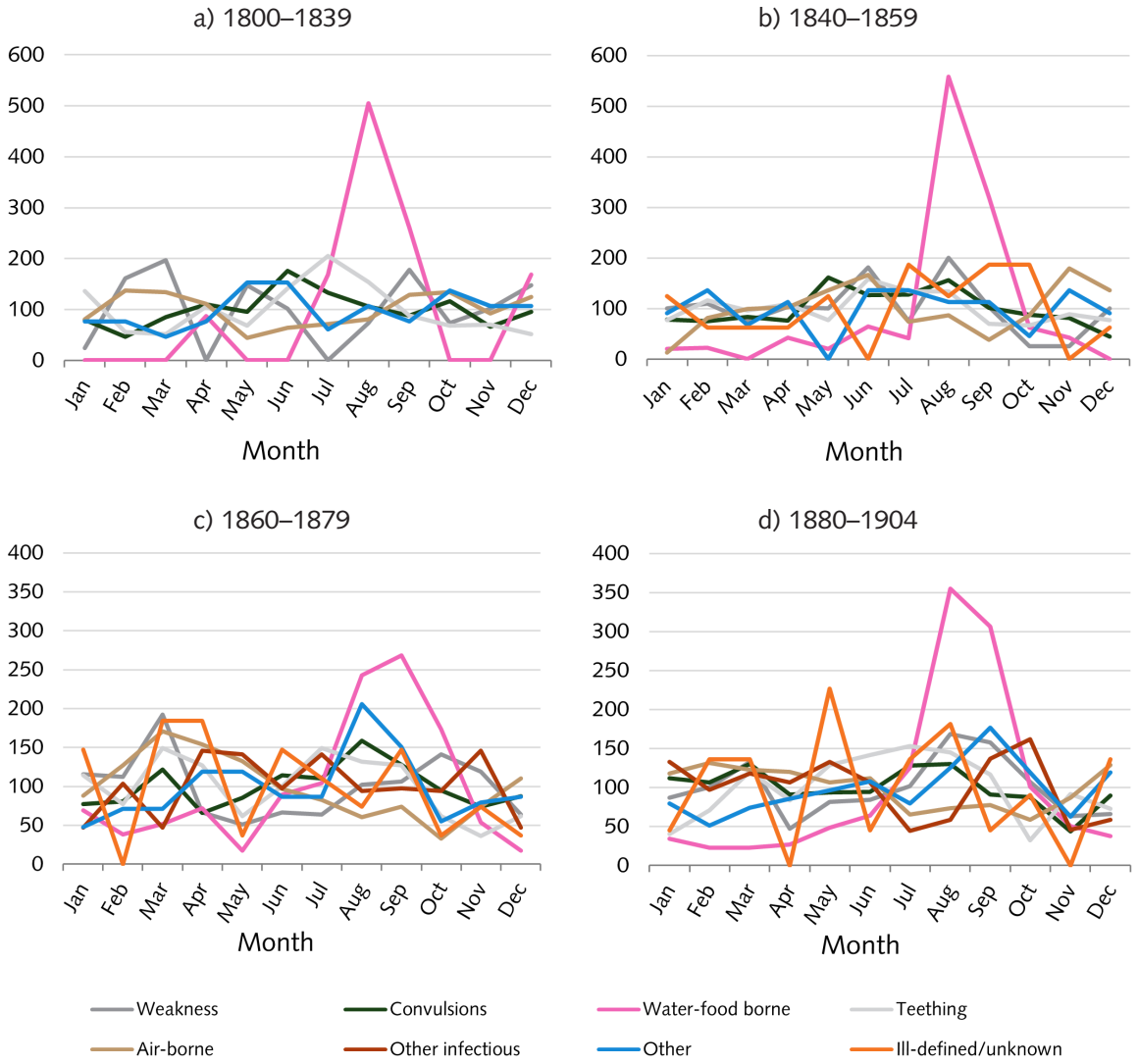
Figure 11 *Seasonality index for neonatal deaths by month, cause of death and period in Rostock, St. Jakobi parish, 1800–1904*



Note: The range for the seasonality index on the y-axis is different on panel graph b. Causal groups with less than one case per month on average are not shown. For each period, teething and external causes were too rare to be included.

Source: As for Figure 2.

Figure 12 *Seasonality index for post-neonatal deaths by month, cause of death and period in Rostock, St. Jakobi parish, 1800–1904*



Note: The range for the seasonality index on the y-axis differs between the upper and lower panel graphs. Causal groups with less than one case per month on average are not shown. For each period, congenital/birth disorders and external causes were too rare to be included.

Source: As for Figure 2.

Table 1 *List of infant causes of death in Rostock, St. Jakobi parish, 1800–1904*

German term	English translation	Causal group	ICD10h	N	%
Blattern	Smallpox	Air-borne	B03.000	7	0.07
Bräune	Diphtheria	Air-borne	A36.000	18	0.18
Bronchialeiterung	Bronchial suppuration	Air-borne	J40.000	1	0.01
Bronchialkatarrh	Bronchial catarrh	Air-borne	J40.000	1	0.01
Bronchitis	Bronchitis	Air-borne	J40.000	3	0.03
Brustentzündung	Pneumonia	Air-borne	J18.900	7	0.07
Brustfellentzündung	Pleurisy	Air-borne	J18.900	2	0.02
Brustfieber	Pneumonia	Air-borne	J18.900	1	0.01
Brustkatarrh	Bronchial catarrh	Air-borne	J40.000	6	0.06
Brustkrankheit	Chest disease	Air-borne	J40.000	113	1.10
Brustkrankheit und Zahnen	Chest disease and teething	Air-borne	J40.000	1	0.01
Brustleiden	Chest disease	Air-borne	J40.000	13	0.13
Brustübel	Chest disease	Air-borne	J40.000	13	0.13
Brustverschleimung	Bronchial catarrh	Air-borne	J40.000	55	0.54
Croup	Croup	Air-borne	J05.000	2	0.02
Diphtherie	Diphtheria	Air-borne	A36.900	27	0.26
Drüsenkrankheit	Glandular disease	Air-borne	A18.208	19	0.19
Drüsenleiden	Glandular disorder	Air-borne	A18.208	3	0.03
Drüsenübel	Glandular disorder	Air-borne	A18.208	1	0.01
Eiterung am Ohr	Suppurative ear	Air-borne	H66.000	1	0.01
Erkältung	Common cold	Air-borne	J00.000	1	0.01
Erkältung und Zahnen	Common cold and teething	Air-borne	J00.000	1	0.01
Grippe	Influenza	Air-borne	J11.100	7	0.07
Halsbräune	Diphtheria	Air-borne	A36.000	16	0.16
Halsentzündung	Pharyngitis	Air-borne	J02.900	3	0.03
Halskatarrh	Throat catarrh	Air-borne	J02.900	1	0.01
Hirnhautentzündung	Meningitis	Air-borne	G03.900	6	0.06
Husten	Cough	Air-borne	R05.000	21	0.21
Husten und Zufall	Cough and accident	Air-borne	R05.000	1	0.01
Influenza	Influenza	Air-borne	J11.100	7	0.07
Katarrh	Catarrh	Air-borne	J40.000	4	0.04
Kehlkopfkatarrh	Laryngitis	Air-borne	J04.000	1	0.01
Kehlkopfleiden	Laryngitis	Air-borne	J04.001	1	0.01
Keuchhusten	Whooping cough	Air-borne	A37.900	145	1.42
Keuchhusten und Krampf	Whooping cough and convulsion	Air-borne	A37.900	1	0.01
Krampfhusten	Convulsive cough	Air-borne	R05.000	1	0.01
Krupp	Croup	Air-borne	J05.000	1	0.01
Luftröhrenentzündung	Tracheal inflammation	Air-borne	J04.100	4	0.04
Luftröhrenkatarrh	Tracheal catarrh	Air-borne	J04.100	14	0.14
Luftröhrenverschleimung	Tracheal inflammation	Air-borne	J04.100	6	0.06
Lungenentzündung	Lung inflammation	Air-borne	J18.901	354	3.46
Lungenkatarrh	Lung catarrh	Air-borne	J18.902	174	1.70

Lungenverschleimung	Lung inflammation	Air-borne	J18.901	19	0.19
Masern	Measles	Air-borne	B05.900	51	0.50
Masern und Zahnen	Measles and teething	Air-borne	B05.900	1	0.01
Pocken	Smallpox	Air-borne	B03.000	4	0.04
Rachenbräune	Diphtheria	Air-borne	A36.000	12	0.12
Rippenfellentzündung	Pleurisy	Air-borne	J18.900	2	0.02
Scharlach	Scarlatina	Air-borne	A38.002	12	0.12
Scharlachfieber	Scarlet fever	Air-borne	A38.000	1	0.01
Scharlachfrieseln	Scarlet strophulus	Air-borne	A38.004	13	0.13
Schwindsucht	Consumption	Air-borne	A16.906	12	0.12
Skropheln	Scrofula	Air-borne	A18.201	8	0.08
Stickhusten	Whooping cough	Air-borne	A37.900	2	0.02
Tuberkulose	Tuberculosis	Air-borne	A16.905	1	0.01
Windpocken	Chicken pox	Air-borne	B01.900	3	0.03
Zahnen und Brustkrankheit	Teething and chest disease	Air-borne	J40.000	2	0.02
Zahnen und Masern	Teething and measles	Air-borne	B05.900	1	0.01
Zahnhusten	Dental cough	Air-borne	R05.000	1	0.01
[Keine Angabe]	[Blank]	Blank	R99.090	90	0.88
Bald nach der Geburt gestorben	Died soon after birth	Congenital/birth disorders	P96.908	3	0.03
Darmblutung	Intestinal bleeding	Congenital/birth disorders	P54.300	1	0.01
Frühgeburt	Premature birth	Congenital/birth disorders	P07.300	20	0.20
Gespaltener Kopf	Cleft head	Congenital/birth disorders	Q75.900	1	0.01
Gleich nach der Geburt gestorben	Died soon after birth	Congenital/birth disorders	P96.908	22	0.22
Kein After	Absence of anus	Congenital/birth disorders	Q42.300	1	0.01
Kein Anus	Absence of anus	Congenital/birth disorders	Q42.300	1	0.01
Kein Hirnschädel	Absence of vault of skull	Congenital/birth disorders	Q75.901	1	0.01
Keine Backen im Mund, sehr kurze Zunge	Absence of cheeks in mouth, very short tongue	Congenital/birth disorders	Q38.600	1	0.01
Knochenverschiefung	Bone deformity	Congenital/birth disorders	Q68.800	1	0.01
Kopfschaden	Head deformity	Congenital/birth disorders	Q67.400	1	0.01
Nabelentzündung	Omphalitis	Congenital/birth disorders	P38.000	4	0.04
Organischer Fehler	Organic failure	Congenital/birth disorders	Q89.901	1	0.01
Rückenleiden	Spinal disorder	Congenital/birth disorders	Q76.400	1	0.01
Rückenmarksleiden	Spinal cord disorder	Congenital/birth disorders	Q06.800	3	0.03
Rückgratsfehler	Spinal malformation	Congenital/birth disorders	Q76.400	2	0.02
Verschlossener After	Closed anus	Congenital/birth disorders	Q42.300	1	0.01
Wirbelsäulenschwellung	Spinal swelling	Congenital/birth disorders	Q67.500	1	0.01
Wolfsrachen	Cleft palate	Congenital/birth disorders	Q35.900	1	0.01
Zugewachsener After	Absence of anus	Congenital/birth disorders	Q42.300	1	0.01
Backenkrampf	Cheek convulsion	Convulsions	R56.806	7	0.07
Krampf	Convulsion	Convulsions	P90.000	2369	23.16
Krampf und Gelbsucht	Convulsion and jaundice	Convulsions	P90.000	1	0.01
Krampf und Schwäche	Convulsion and weakness	Convulsions	P90.000	1	0.01
Krampf und Zahnen	Convulsion and teething	Convulsions	P90.000	1	0.01

Magenkrampf	Gastric convulsion	Convulsions	R56.807	65	0.64
Armbruch	Arm fracture	External causes	S42.300	1	0.01
Durch die Amme erdrückt	Crushed by the wet nurse	External causes	Y20.004	1	0.01
Durch Operation bei der Entbindung	Surgery during delivery	External causes	Y60.000	1	0.01
Im Bett erdrückt	Crushed in bed	External causes	Y20.004	1	0.01
Knochenfraktur	Bone fracture	External causes	T14.200	1	0.01
Krampf in Folge des Verbrennens	Convulsion as a result of burning	External causes	T95.900	1	0.01
Operation	Surgery	External causes	Y65.800	9	0.09
Unglücksfall	Misadventure	External causes	X59.900	1	0.01
Verblutung und Mord	Exsanguination and murder	External causes	Y09.002	1	0.01
Blutung	Bleeding	Ill-defined	R58.000	1	0.01
Blutverlust	Loss of blood	Ill-defined	R58.002	1	0.01
Drüsen	Glands	Ill-defined	R59.001	29	0.28
Eiterung	Suppuration	Ill-defined	R68.803	1	0.01
Fieber	Fever	Ill-defined	R50.900	3	0.03
Innere Blutung	Internal bleeding	Ill-defined	R58.001	1	0.01
Kinderkrankheit	Childhood disease	Ill-defined	R68.100	1	0.01
Wassersucht	Hydropsy	Ill-defined	R60.901	6	0.06
Augenübel	Disorder of eye	Other	H57.900	1	0.01
Ausschlag	Rash	Other	R21.000	3	0.03
Bauchfellentzündung	Peritonitis	Other	K65.900	2	0.02
Blasenausschlag	Blistering eruption	Other	L10.900	1	0.01
Bleichsucht	Anaemia	Other	D64.900	1	0.01
Blinddarmentzündung	Appendicitis	Other	K37.000	1	0.01
Blutarmut	Anaemia	Other	D64.900	4	0.04
Blutbrechen	Blood vomiting	Other	K92.000	1	0.01
Blutkrampf	Blood convulsion	Other	R56.809	2	0.02
Blutschwamm	Haemangioma	Other	D18.000	1	0.01
Blutstockung	Blood stasis	Other	I82.901	1	0.01
Brechen	Vomiting	Other	R11.000	1	0.01
Brechleiden	Nausea	Other	R11.000	1	0.01
Bruch	Hernia	Other	K46.900	3	0.03
Brustbräune	Angina pectoris	Other	I20.900	1	0.01
Brustgeschwüre	Lung ulcers	Other	J98.402	1	0.01
Brustkrampf	Asthma	Other	J45.900	49	0.48
Darmgeschwüre	Intestine ulcers	Other	K63.300	1	0.01
Darmverschleimung	Bowel ulceration	Other	K56.600	3	0.03
Darmverstopfung	Bowel obstruction	Other	K59.000	1	0.01
Drüsenverschleimung	Glandular ulceration	Other	R59.001	1	0.01
Englische Krankheit	Rickets	Other	E55.001	11	0.11
Entzündung	Inflammation	Other	R22.901	5	0.05
Entzündung des Hüftgelenks	Inflammation of the hip joint	Other	M16.900	1	0.01

Entzündung des Zellgewebes	Inflammation of cellular tissue	Other	L03.900	1	0.01
Erbrechen	Vomiting	Other	R11.000	2	0.02
Frieseln	Strophulus	Other	R21.002	26	0.25
Furunkulose	Furunculosis	Other	L02.902	4	0.04
Gehirnblutung	Cerebral bleeding	Other	I61.900	2	0.02
Gehirnkrampf	Cerebral convulsion	Other	I67.801	15	0.15
Gehirnkrankheit	Brain disease	Other	G93.900	6	0.06
Gehirnlähmung	Brain paralysis	Other	G80.900	1	0.01
Gehirnleiden	Brain disorder	Other	G93.900	7	0.07
Gehirnschlag	Stroke	Other	I64.003	6	0.06
Gehirnverschleimung	Cerebral ulceration	Other	G06.001	1	0.01
Gelbsucht	Jaundice	Other	R17.000	6	0.06
Genickstarre	Meningism	Other	R29.100	1	0.01
Geschwür	Ulcer	Other	L98.409	12	0.12
Gliederkrankheit	Rickets	Other	E55.000	1	0.01
Halsgeschwulst	Throat ulcer	Other	J39.201	1	0.01
Halskrankheit	Throat disease	Other	J39.200	2	0.02
Halsleiden	Throat disorder	Other	J39.200	1	0.01
Hautausschlag	Rash	Other	R21.000	2	0.02
Hautentzündung	Skin inflammation	Other	L08.900	2	0.02
Hautkrankheit	Skin disease	Other	L98.900	3	0.03
Herzentzündung	Heart inflammation	Other	I51.801	1	0.01
Herzfehler	Heart failure	Other	I50.900	1	0.01
Herzkrampf	Angina pectoris	Other	I20.900	1	0.01
Herzlähmung	Heart paralysis	Other	I21.906	1	0.01
Herzleiden	Heart disease	Other	I51.900	9	0.09
Herzschlag	Heart apoplexy	Other	I21.904	8	0.08
Herzschwäche	Weak heart	Other	I50.901	2	0.02
Innere Geschwüre	Internal ulcers	Other	L94.401	2	0.02
Karbunkel	Carbuncle	Other	L02.901	1	0.01
Kopfeiterung	Suppurative inflammation of head	Other	R22.002	1	0.01
Kopfentzündung	Head inflammation	Other	R22.001	1	0.01
Kopfkrampf	Head convulsion	Other	I67.803	10	0.10
Kopfkrampf und Zahnen	Head convulsion and teething	Other	I67.803	1	0.01
Kopfkrankheit	Head disease	Other	G93.900	27	0.26
Kopfleiden	Head disease	Other	G93.900	1	0.01
Kopfschlag	Stroke	Other	I64.003	1	0.01
Kopfübel	Head disorder	Other	G93.900	1	0.01
Krampfschlag	Cerebral convulsion	Other	I67.801	2	0.02
Leberkrankheit	Liver disease	Other	K76.900	2	0.02
Leberleiden	Liver disease	Other	K76.900	1	0.01
Leberschwäche	Liver insufficiency	Other	K76.902	1	0.01
Lebervergrößerung	Enlarged liver	Other	R16.000	1	0.01

Luftkrampf	Asthma	Other	J45.900	1	0.01
Lungenkrampf	Asthma	Other	J45.900	2	0.02
Lungenkrankheit	Lung disease	Other	J98.400	5	0.05
Lungenlähmung	Lung paralysis	Other	J98.405	3	0.03
Lungenleiden	Lung disorder	Other	J98.400	22	0.22
Lungenschlag	Pulmonary embolism	Other	I26.900	5	0.05
Lungenschwäche	Lung insufficiency	Other	J98.406	2	0.02
Lungenübel	Lung disorder	Other	J98.400	9	0.09
Magenblutung	Gastric bleeding	Other	K92.200	1	0.01
Magenerweichung	Gastromalacia	Other	K31.806	2	0.02
Magengeschwulst	Gastric ulcer	Other	K25.900	1	0.01
Magenkolik	Gastric colic	Other	R10.402	1	0.01
Magenkrankheit	Gastric disease	Other	K31.902	12	0.12
Magenleiden	Gastric disorder	Other	K31.902	57	0.56
Magenreizung	Irritated stomach	Other	K31.906	1	0.01
Magenschwäche	Gastric insufficiency	Other	K30.002	5	0.05
Magenübel	Gastric disorder	Other	K31.902	60	0.59
Nabelbruch	Umbilical hernia	Other	K42.900	1	0.01
Nierenkrankheit	Kidney disease	Other	N28.900	2	0.02
Nierenleiden	Kidney disorder	Other	N28.900	6	0.06
Nierenschlag	Kidney stroke	Other	N28.806	1	0.01
Nierenvereiterung	Kidney suppuration	Other	N15.901	1	0.01
Schlag	Stroke	Other	I64.003	28	0.27
Schlag und Zahnen	Stroke and teething	Other	I64.003	1	0.01
Schlagfluss	Stroke	Other	I64.004	8	0.08
Schwamm	Sponge	Other	L98.409	36	0.35
Schwamm und Ausschlag	Sponge and rash	Other	L98.409	1	0.01
Schwamm und Brustkrankheit	Sponge and chest disease	Other	L98.409	1	0.01
Schweisbeutel	Sudoral sac	Other	L98.909	1	0.01
Unterleibsentzündung	Inflammation of lower body organs	Other	K65.902	3	0.03
Unterleibslähmung	Paralysis of lower body	Other	A80.300	1	0.01
Unterleibsleiden	Disorder of lower body organs	Other	R19.800	1	0.01
Wasserkopf	Hydrocephalus	Other	G91.900	4	0.04
Zahn und Frieseln	Tooth and strophulus	Other	R21.002	1	0.01
Zellgewebeeiterung	Suppuration of cellular tissue	Other	L03.900	1	0.01
Zellgewebekrankheit	Disease of cellular tissue	Other	L03.900	1	0.01
Zu große Leber	Enlarged liver	Other	R16.000	1	0.01
Faulfieber	Spotted fever	Other infectious	A75.000	1	0.01
Gehirnentzündung	Cerebral inflammation	Other infectious	G04.900	85	0.83
Gehirnkatarrh	Cerebral catarrh	Other infectious	G04.900	2	0.02
Gesichtsrose	Facial erysipelas	Other infectious	A46.004	1	0.01
Kopfrose	Head erysipelas	Other infectious	A46.005	1	0.01

Laufende Rose	Running erysipelas	Other infectious	A46.000	1	0.01
Mundfäule	Stomatitis	Other infectious	K12.100	1	0.01
Mundkrampf	Lockjaw	Other infectious	A35.001	1	0.01
Nabelrose	Umbilical erysipelas	Other infectious	A46.006	1	0.01
Rose	Erysipelas	Other infectious	A46.000	17	0.17
Rose Ausguss	Erysipelas drain	Other infectious	A46.000	1	0.01
Rose der Neugeborenen	Erysipelas	Other infectious	A46.000	1	0.01
Rotlauf	Erysipelas	Other infectious	A46.000	2	0.02
Syphilis	Syphilis	Other infectious	A50.900	9	0.09
Venerisches Gift der Amme	Venereal poison of the wet nurse [Syphilis]	Other infectious	A50.900	1	0.01
Zahnen	Teething	Teething	K00.700	115	1.12
Zahnen und Krampf	Teething and convulsion	Teething	K00.703	3	0.03
Zahnfieber	Dental fever	Teething	K00.702	2	0.02
Zahnkrampf	Dental convulsion	Teething	K00.703	344	3.36
Tot gefunden	Found dead	Unknown	R99.000	6	0.06
Unbekannt	Unknown [stated to be]	Unknown	R99.001	2	0.02
Brechdurchfall	Cholera	Water-/food-borne	A00.900	579	5.66
Brechruhr	Cholera	Water-/food-borne	A00.900	29	0.28
Cholera	Cholera	Water-/food-borne	A00.900	24	0.23
Cholerine	Cholera	Water-/food-borne	A00.900	7	0.07
Darmentzündung	Enteritis	Water-/food-borne	A09.009	7	0.07
Darmkatarrh	Intestinal catarrh	Water-/food-borne	A09.010	37	0.36
Darmkrankheit	Enteritis	Water-/food-borne	A09.009	1	0.01
Darmleiden	Enteritis	Water-/food-borne	A09.009	1	0.01
Diarrhoe	Diarrhoea	Water-/food-borne	A09.002	10	0.10
Dickdarmkatarrh	Intestinal catarrh	Water-/food-borne	A09.010	1	0.01
Durchfall	Diarrhoea	Water-/food-borne	A09.002	85	0.83
Hitzige Brustkrankheit	Heated chest disease	Water-/food-borne	A01.000	1	0.01
Kindertyphus	Typhoid fever	Water-/food-borne	A01.000	1	0.01
Magendarmentzündung	Gastroenteritis	Water-/food-borne	A09.008	1	0.01
Magenentzündung	Gastritis	Water-/food-borne	K29.700	26	0.25
Magenfieber	Gastric fever	Water-/food-borne	A01.003	1	0.01
Magenkatarrh	Gastric catarrh	Water-/food-borne	K31.905	99	0.97
Magenverschleimung	Gastric inflammation	Water-/food-borne	K29.701	4	0.04
Ruhr	Dysentery	Water-/food-borne	A04.900	14	0.14
Schleimfieber	Typhoid fever	Water-/food-borne	A01.000	5	0.05
Typhus	Typhoid fever	Water-/food-borne	A01.000	1	0.01
Verdauungsstörung	Digestive disturbance	Water-/food-borne	K30.000	1	0.01
Wurmfieber	Enteritis	Water-/food-borne	A09.009	2	0.02
Abzehrung	Wasting	Weakness	R64.002	11	0.11
Auszehrung	Wasting	Weakness	R64.002	90	0.88
Entkräftung	Inanition	Weakness	R64.001	11	0.11
Schwäche	Weakness	Weakness	R53.002	1299	12.70