What was Killing Babies in Amsterdam? A Study of Infant Mortality Patterns Using Individual-Level Cause of Death Data, 1856–1904

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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 Amsterdam?

A Study of Infant Mortality Patterns Using Individual-Level Cause of Death Data, 1856–1904

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ABSTRACT

Based on unique individual-level cause of death data, this article presents an analysis of the development of infant mortality and the underlying cause of death pattern in the city of Amsterdam in the period 1856–1904. We contribute to the discussion on the development of infant mortality and its determinants and test the newly-constructed ICD10h coding system. First, our results demonstrate that the ICD10h and groupings of causes worked quite well for our period and city data. Second, Amsterdam moved from being one of the most lethal cities in the country to one of the healthiest for infants. These improvements in the fate of infants were brought about despite faltering progress in the provision of piped water, and an absence of modern sewerage throughout the period. For the entire period air-borne diseases were a prominent cause of death category, peaking in the 1880s and still making up the major group of diseases by 1904. Water- and food related ailments were also dominating the epidemiological pattern after the 1870s. Vague or ill-defined disease terms were frequent at the start of the study period. These observations suggest that physicians were increasingly better able and more prepared to formulate more precise disease terms by the 1900s. The seasonality analysis of the different disease groups demonstrates strong summer effects on the group of water- and food related causes of death. It testifies to the shortcomings in the city's hygienic situation and limited breastfeeding.

Keywords: Infant mortality, Causes of death, Coding historical diseases, Amsterdam, 19th Century

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1 INTRODUCTION

This article presents an analysis of the development of infant mortality and the underlying cause of death pattern in the city of Amsterdam between 1856 and 1904. The aim of this study is twofold. First of all, we aim to contribute to the discussion on the development of infant mortality and its determinants in the Netherlands in the second half of the 19th century. Most studies in this field have had to rely on mortality data only, either at the national, provincial or local level, without having access to the underlying causes of death. This makes it more difficult to answer questions relating to differences in levels of infant mortality between locations and differences in the timing of the decline in infant mortality rates in this period. This study is based on unique individual-level cause of death data so that we are able to reveal in what ways the underlying epidemiological profile changed with declining infant mortality rates. In the study period Amsterdam, and the other cities in the north-western part of the Netherlands, belonged to the group of pioneers leading the mortality decline in the country. In the mid-19th century levels of infant mortality in the city were in fact rather high, comparatively speaking, but began a steady decline from the 1880s onwards. Studying the cause of death pattern will aid in our attempts to answer questions relating to the drivers of mortality change. The second aim of this contribution is to test the European coding system for historical causes of death, the ICD10h, on individual-level cause of death data for infants. This coding system has been designed in recent years by members of the SHiP+ network; an elaboration of both the network and the ICD10h coding system is presented in the introduction to this special issue.[1] The test we are conducting here will make clear whether the coding system is able to capture in adequate ways the disease patterns for infants in Amsterdam in the study period. The study period creates in many ways a dynamic research setting for our test of the ICD10h coding system. The analysis we conduct in this paper follows closely the outline set out for all contributions in this special issue. Before we begin the analysis of what was killing babies in Amsterdam, we will first introduce the historical context of the city of Amsterdam and the dataset used for this study.

2 THE CITY AND THE DATA

Today Amsterdam is the capital city of the Netherlands and the largest city in the country. In the 17th century Amsterdam was a large sea port city, serving as an important node in global networks of trade and as the staple market of the world, making it into the wealthiest city in the western world. By the start of the 19th century however, most of that wealth and economic power had evaporated, and the city's economy went through a period of decline and stagnation lasting until the 1870s. Despite its poor social and economic condition Amsterdam still belonged to the top 20 of largest cities in Europe in the mid-19th century (Chandler & Fox, 1974, p. 361).

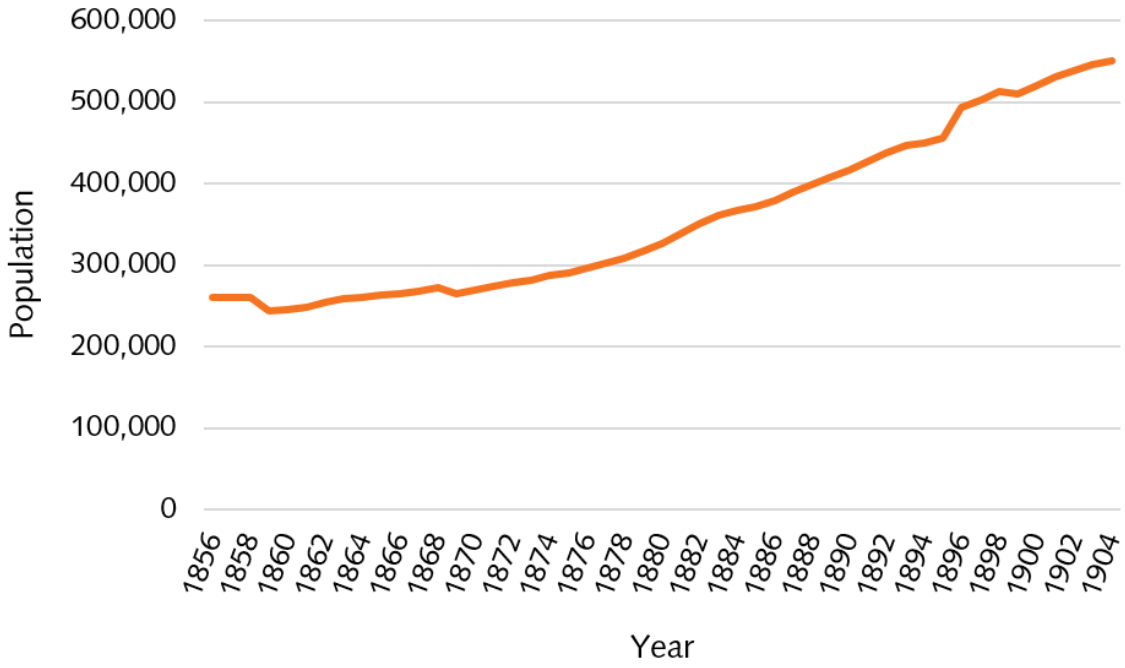
In the 1870s the city experienced a strong economic boom, driven by extensive public works — such as the construction of the North-Sea Canal and the expansion of the city's port facilities —, and the spectacular growth of the diamond industry as well as the building sector (Knotter, 1991). Building activities were strongly stimulated by the influx of large numbers of immigrants from the surrounding rural areas. Amongst them were many young adults and couples, which caused the numbers of births to rise substantially, above all in the period between the 1870s and the 1890s. Figure 1 shows that population numbers doubled between 1856 and 1904 and that the increase in total population took place from the 1870s onwards. Natural increase played an important role. The figure in Appendix 1 shows a considerable rise in the number of births taking place from the 1870s onwards until well into the 1890s.

The economic upswing of the 1870s was interrupted by years of crisis in the period 1884–1888 and 1892–1896, when unemployment in the city soared and the numbers of people in poor relief as a result of unemployment peaked from 1884 onwards and remained at a high level until 1910. During the economic upturn of the 1870s nominal wage levels in local industries had risen considerably, to level out during the 1880s after which in the 1890s a new upward trend in industrial wages began. As a result industrial wages in Amsterdam had increased by 60% between 1850 and 1900 (van Zanden, 1987). In terms of social structure, Amsterdam was typically a working class city, heavily dominated by skilled and unskilled labourers; in 1851, only 2% of the population could be classified as belonging

1 See also https://www.ru.nl/en/rich/research/networks/ship

to the elite and 20% belonged to the middle classes (Ekamper & van Poppel, 2019). The Amsterdam labour market was heavily male-dominated as a result of the orientation on shipbuilding, and the metallurgical and machine building industries. Female labour was concentrated in domestic service and in the final parts of the 19th century also in the clothing industry (Knotter, 1991; van Zanden, 1987). Mostly, this female workforce was limited to unmarried women. Especially in the western parts of the Netherlands rather low proportions of married women, below 6 or 8%, participated in the paid labour force in this period (Janssens, 2014).

Figure 1 *Total population numbers, Amsterdam 1856–1904*



Sources: These population counts are based on the census data for Amsterdam for the years 1849, 1859, 1869 1879, 1889, 1899, 1910, and the yearly population numbers from the Amsterdam Statistical Yearbooks from 1849 onwards. For each year an age and gender specific population at risk has been constructed. A working paper is available from the authors with a detailed specification of the procedure followed (Buzasi, 2021).

To study infant mortality with greater precision than previous studies did, this study uses a unique dataset based on the Amsterdam cause of death registers covering the years ranging from 1854 up until and including the year 1940. The registers were initiated in 1853 by the city authorities in response to the increasing fear amongst the population of being buried alive. Hence, the city adopted the rule that all deaths needed to be verified by a doctor before burial could take place. The members of the Amsterdam Medical Practitioners' Circle, the *Geneeskundige Kring*, seized this opportunity to also demand a systematic verification of the cause of death which was to be reported before the death certificate could be issued. Hence from 1854 onwards the city was able to start a systematic registration of all deceased individuals together with the cause they died of (Neurdenburg, 1929). The Medical Practitioners' Circle agreed to "personally monitor registration certificates, to ensure that local registrars had accurately and truthfully stated the nature of the disease" (van Poppel & van Dijk, 1997). The resulting registers were kept until the end of 1940. Apart from registering the individual cause of death, the registers also record the address of the deceased — but no names —, the date of death, his/her age, sex, marital status, occupation of the deceased, and for a limited number of years the registers also record the religious denomination of the deceased.

The Amsterdam medical doctors were in the vanguard of the Dutch hygienist movement, organised in the Dutch Society for the Advancement of Medicine, the *Nederlandsche Maatschappij tot bevordering der Geneeskunst* (NMG), established in 1849. The NMG was primarily aimed at improving the standards of medical care and the quality of medical professionals by promoting continuous training, but was also actively pressing for social and sanitary reforms (Houwaart, 1991). In addition, the society promoted the introduction of a uniform nation-wide system for the recording of causes of death based

on the British disease classification system created by William Farr. These efforts were successful in 1865 when the Dutch government issued the new Public Health Inspectorate Act and the Medical Practitioners Act, making it mandatory for medical doctors throughout the country to provide a medical certificate with every death. Also, doctors were required to state as accurately as possible what the patient had died of, but "with due regard to their oath of confidentiality" (van Poppel & van Dijk, 1997). Obviously, this latter phrase opened up the possibility that doctors resisted the recording of the precise cause of death of some or all of their patients. Starting in 1866, the medical certificates had to be handed in to the municipal civil registrars, who in turn were obliged to report monthly overviews to the provincial medical authorities. The provincial inspectors then summarised these overviews in 34 different disease categories (van den Boomen, 2021).

The introduction of the 1865 laws on the regulation of medicine and the medical profession is likely to have enhanced the quality of the ongoing registration of causes of death in Amsterdam. This can be gleaned from the fact that after approximately 1867 the registration of causes of death in the Amsterdam registers becomes more complex and detailed with doctors also reporting a second or even a third cause for a large number of cases. This is quite likely related to the introduction of new forms for the medical certificates in 1868 which differentiated between the underlying or "first disease" and the immediate cause of death, the "last disease". The Dutch government hoped this would further improve the accuracy and the completeness of the registration. Nevertheless, not all doctors accepted these new rules, which they regarded as a breach with patient confidentiality (van Poppel & van Dijk, 1997). The Amsterdam registers suggest that this also applied to some of the practitioners in the city. The academic consensus is, however, that in general the quality of the cause of death registration in the early years was much better in the more advanced urban areas of the country than in more remote rural parts. Given the prominent position of Amsterdam physicians in the national hygienist movement, we can expect the registration in the city to have been of comparatively high quality. Finally, an important step to further improve cause of death statistics nationally, was taken in 1869 with the passing of the Burial Act, which made medical certification of causes of death a national statutory requirement.

The dataset used in this study pertains to the period of 1856 up to and including 1904, due to the fact that the data for the period 1904–1940 are still in the process of cleaning, standardisation and coding. The choice of this period was further determined by the fact that the year 1855 is lacking entirely. Hence this paper takes 1856 as a starting point. Other data gaps are found for the years 1892–1894, which are also lacking in entirety, and for 1899 we found a data gap of three months. We excluded these years with data gaps from the analysis. Finally, for the year 1858 we have a small random under registration in the cause of death registers which report 103 fewer infant deaths (or 5,3%) than the total of 1,942 infants deaths recorded in the city reports of that same year. We decided to keep this year in the analysis as the missingness is very limited and seems to be highly random. The table in Appendix 2 shows that the resulting dataset consists of a robust number of events (deaths experienced by infants) and a robust number of cases (that is live births), making up the population at risk. We tested for data completeness of infant deaths in the cause of death register by comparing the total number of infant deaths by year to the number of infant deaths reported in the annual city reports (*Gemeentelijke Jaarverslagen*). These annual reports were mandatory by law for all communities in the country and were used to report to the central government on community developments. The annual city reports are regarded as highly accurate on topics such as basic population numbers. In Appendix 3 we show the results of the comparison which indicates that the infant deaths recorded in the cause of death registers follow the numbers stated in the annual city reports closely, apart from the years with the data gaps reported above. For the year 1862 there is a small discrepancy with the cause of death registers undercounting the number of infant deaths by 3% of a total number of infant deaths of 1756. It is unclear what the precise reason is for this difference. The counts for live births by year have also been taken from the city reports.

In the following section we will first discuss a brief overview of recent contributions to the Dutch debate on infant mortality in the 19th century as well as a number of important international contributions. This overview cannot be exhaustive given the scope and ambition of the paper which concentrates on the empirical study of infant mortality in Amsterdam. In that section we also consider an overview of infant mortality rates in Amsterdam and how these evolved over time, including the development of infant mortality by major groups of causes of death. We distinguish 11 categories, based on the ICD10h coding system, used by all the SHiP studies contributing to this special issue. In Appendix 14

we list all (standardised) disease terms within each category with a frequency of over a hundred cases for the entire period. In Section 3 we will separate neonatal and post-neonatal deaths, and for each we will consider the development of major causes of death over time, in Section 4. In Section 5 we will explore the seasonality of causes of death by major group for neonatal and post-neonatal deaths separately. In Section 6 we present our conclusions. As already explained above, the statistics that we present in these sections are part of the collective analytical strategy followed by all papers in this special issue. However, we do depart to some extent from this collective strategy in the sense that we have opted to show annual mortality rates in the main body of the text rather than quinquennial rates. The quinquennial rates were unable to show the missing years with data gaps in an adequate way and as the Amsterdam dataset counts a robust numbers of cases for each year, we decided to show the annual rates in the main body of the text and relegate the quinquennial rates to the appendix.

3 INFANT MORTALITY

Survival chances for infants in the western parts of the Netherlands were generally very poor in the mid-19th century, compared to the eastern and southern parts of the country (van den Boomen & Ekamper, 2015). At least until the 1870s infant mortality rates were much higher in the coastal provinces of North Holland — the province in which Amsterdam is located —, South-Holland and Zeeland. Around the middle of the century these coastal provinces had infant mortality rates well above 200 per 1,000 live births. After the 1870s survival conditions for infants in the west improved considerably whereas developments in the south lagged behind or in some cases even deteriorated. These time lags in the decline of infant mortality rates in the southern parts of the country, with a predominantly Catholic population, were traditionally attributed to religious differences between Protestants and Catholics. The explanation for these differences between religious groups were generally sought in the field of different breastfeeding habits. Catholic mothers were less inclined to breastfeed their children (van Poppel et al., 2002). However, recent studies have revealed that religion cannot account for these regional divergences (Janssens & Pelzer, 2014; van den Boomen, 2021), or that the opposition between Catholics and Protestants loses much of its stark contrast when regional indicators are taken into account (Walhout, 2019). In another study on infant mortality in Amsterdam it was confirmed that religious differences in infant mortality only played a role where it concerned the favourable position of Jewish families and the unfavourable position of families in the orthodox Protestant minority groups. Between Catholics and liberal Protestants no significant differences were found (Ekamper & van Poppel, 2019). The sources we are using in this study, however, do not allow for an analysis of religious differences in infant mortality (Riswick et al., 2022).

The high levels of infant mortality in the western part of the Netherlands in the mid-19th century are generally contributed to poor water quality due to the strongly salinized soil conditions which made people solely dependent upon surface water (van den Boomen, 2021). Not surprisingly conditions for infants were even worse for the densely populated cities in the west, among them Amsterdam. In the recent international debate a lot of attention has been given to the impact of innovative sanitary technologies, such as piped water and sewerage, on the historical reductions in infant mortality rates. For US cities, scholars have estimated that between 1900 and 1930 clean water and sewerage reduced infant mortality levels by 44%, or even as much as 59% (Alsan & Goldin, 2015; Cutler & Miller, 2005; Cutler & Miller, 2015). Other studies for England and Wales, and for Australia, also report significant effects on the decline of mortality, and in particular for mortality due to water-related causes of death (de Looper et al., 2019; Harris & Hinde, 2019). Some scholars emphasize that the effects of these sanitary measures may differ by period and context (Peltola & Saaritsa, 2019). Effects may have been smaller for the larger pioneering cities adopting the less advanced technologies at an earlier stage, e.g. in the 19th century, than for much smaller locations which were able to introduce the more advanced infrastructures available in the 20th century.

Given the importance attached to urban sanitary conditions in the recent literature, we will discuss this factor more extensively for Amsterdam. Obviously, sanitary conditions are not solely responsible for the historical decline of infant mortality, which should be considered a multi-factorial process (Pozzi & Barona, 2012). Sanitary conditions in Amsterdam were generally reported as having been quite poor at the start of the study period, and with increasing population numbers the situation may have

deteriorated even further. As we will see below, infant mortality rates in Amsterdam at the start of the study period were rather high, which is often related to the strong contamination of the city's surface and ground water supply (van den Boomen, 2021). Poor families were dependent upon the canals for waste disposal as well as washing and other household purposes. Contemporary reports by medical doctors even lament the unsavoury practices of emptying so-called "night buckets" directly onto the streets (Jager, 2002, p. 61). In 1872 the so-called Liernur system was introduced for the removal of human faeces; the system was based on closed pipes without the use of flush water. Diffusion was slow, however, so that at the end of the 19th century only 40% of the city's inhabitants was connected to the system (van Zon, 1993). Moreover, due to the ambivalence in the sanitary policies developed by the city, an important part consisted of a so-called "temporary Liernur system" in which the faeces were still collected in big containers above ground which had to be emptied three times a week by the city services (van Zon, 1986). In 1906 a modern public sewerage infrastructure which collected both waste and rainwater was introduced. A piped water system was introduced in 1854 by a private company on the basis of yearly subscriptions. The exact amount to be paid depended upon the number of rooms in the house; nevertheless, such a yearly subscription was beyond the reach of most working-class families (Groen, 1978, p. 76). However, the water company also installed so-called standpipes throughout the city where piped water could be purchased by the bucket. The initial number of 15 standpipes installed in 1855–1856 increased steadily thereafter, so that by 1866 the city counted 56 of such outlets.

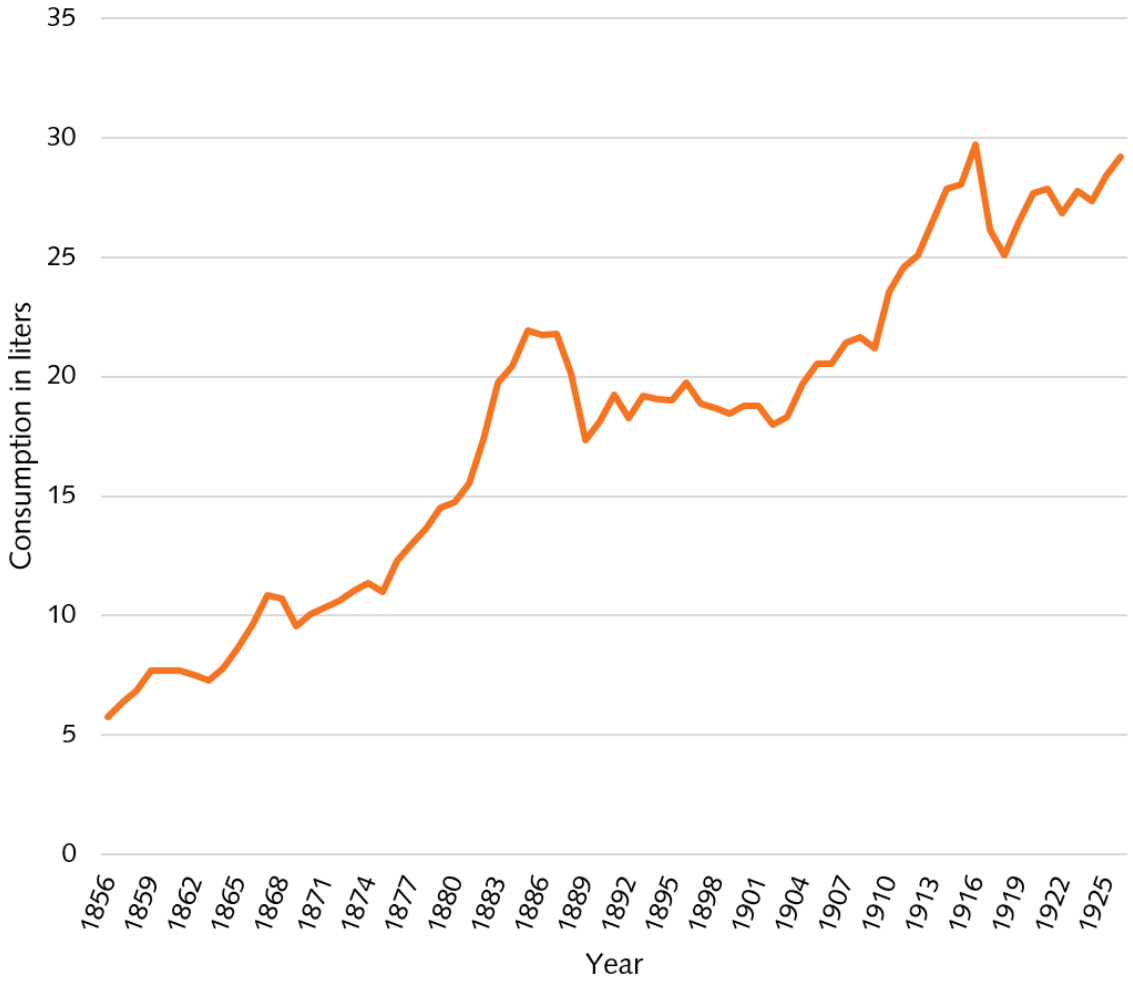
The increase in the provision of clean water is also borne out by Figure 2, which shows the consumption of piped water per capita. Between 1854 and the mid-1880s water consumption per capita rose quickly to the level of approximately 22 cubic meters per annum. This latter amount, which comes down to 22,000 litres per year or 60 litres per day, may seem quite substantial; however, amongst the subscribers were also many factories and public institutions which were probably using large quantities.[2] After the mid-1880s until 1910 the city experienced a "water crisis". The consumption of piped water per capita stagnated and even declined due to the fact that the water infrastructure ran up against its own technological limits. It was unable to deliver the water pressure required by the huge increases in population numbers, between 1879 and 1909 numbers increased by 78%, and the expanding urban economy. Water delivery problems were especially urgent during the warm summer months, so that large numbers of households were going without any water at all (Groen, 1978, p. 104). These problems were only slowly overcome after 1896 when the city council took over the water system and the provision of piped water to all of its inhabitants. The city's water crisis was also picked up in a report on US commercial relations issued by the US government in 1903, with data on water consumption for the years 1896–1899 which makes clear that the consumption level of clean water in Amsterdam was lagging seriously behind cities such as Glasgow, Hamburg, Munich and Rotterdam (Bureau of Foreign Commerce, Department of State, 1903, p. 481).[3]

High infant mortality rates (IMR) are clearly visible in our results. Figure 3 displays the annual IMR by sex for Amsterdam between 1856 and 1904, based on two datasets: the yearly city reports and the cause of death registers (the quinquennial IMRs by sex are shown in Appendix 4). The calculations based on the cause of death registers make use of the numbers of live births as stated by the city reports, but uses the number of infant deaths as given by the cause of death registers. Here, the years with data gaps, which we have discussed above in Section 2, are again visible. At the start of the period IMR was high for both boys and girls, fluctuating between roughly 180 and 230 but with mortality peaks which sometimes reached as high as 280. These mortality peaks for both boys and girls were related to the occurrence of a number of epidemic years for infants: 1858, 1871, and 1880. The years 1858 and 1871 were years with smallpox epidemics (Muurling et al., 2022). In 1866 the city was visited by cholera, but unlike the 1855 epidemic, this time without a clear impact for infants. In 1880 we see a final peak of excessive infant mortality, which was caused by a 50% increase in water- and food borne diseases, as well as a 50% increase in air-borne diseases occurring amongst infants. This final peak therefore cannot be linked to a single disease or an epidemic outbreak occurring in the city in that year, or to particular circumstances such as extreme weather conditions which might explain the enhanced IMR.

2 For instance, in 1856 the total set of subscriptions counted 1,397 private households, 40 factories and 30 public institutions (van Tijn, 1965).

3 These latter four cities had an average consumption of 225, 184, 198 and 162 liters per capita per day in the years 1898–1899. Daily consumption figures for Amsterdam varied between 80 and 85 liters per capita in the years 1896–1899. We wish to thank Daniel Gallardo Albarrán for drawing our attention to this source.

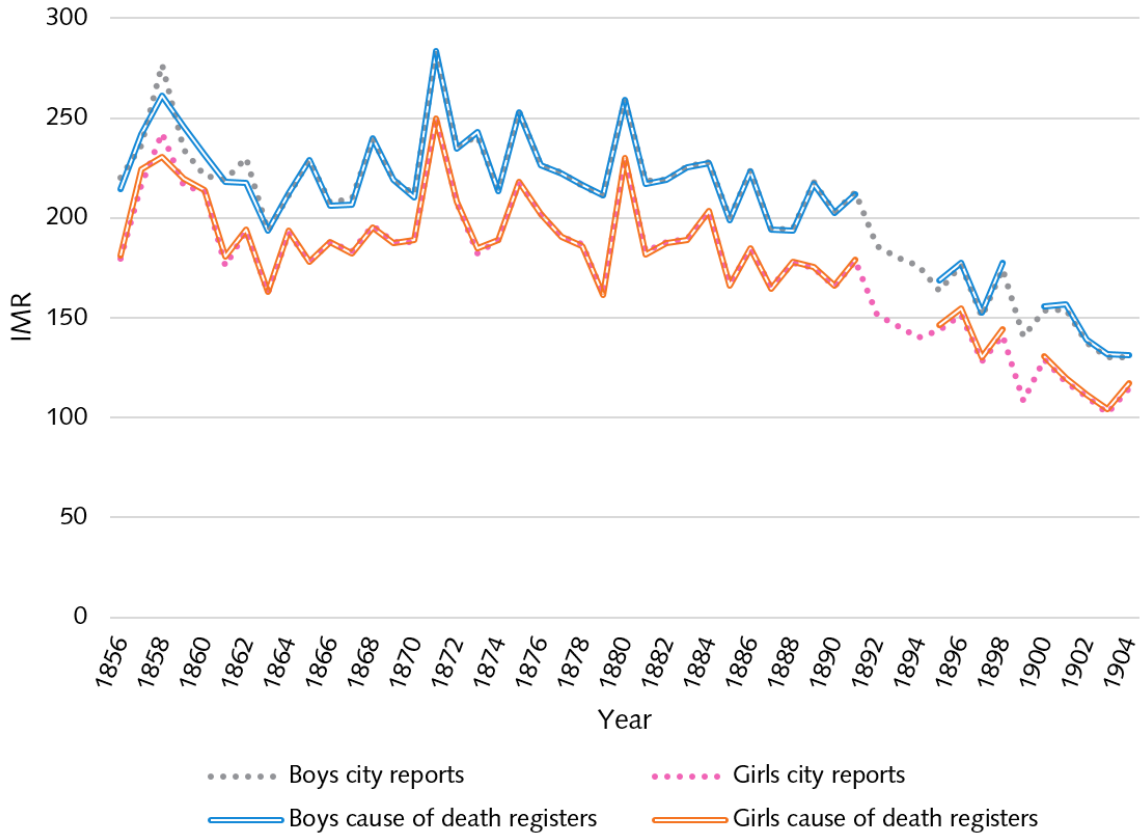
Figure 2 *Average per capita consumption of piped water, Amsterdam 1856–1926*



Sources: Calculations are based on the 'jaarverslagen van de gemeente Amsterdam' (Stadsarchief Amsterdam, toegangsnr. 15030, Collectie Stadsarchief Amsterdam: Bibliotheek, inv.nrs. 1332256–133349, de jaarverslagen van de gemeente Amsterdam), 'statistische jaarboeken' (Stadsarchief Amsterdam, 5185, Inventaris van het Archief van het Bureau voor Statistiek, inv.nrs. 859–889), and 'jaaroverzichten van de geleverde hoeveelheid water van de Gemeentewaterleiding' (Stadsarchief Amsterdam, 30031, Inventaris van het Archief van de Gemeentewaterleidingen; Afdeling Statistiek, inv.nr. 265).

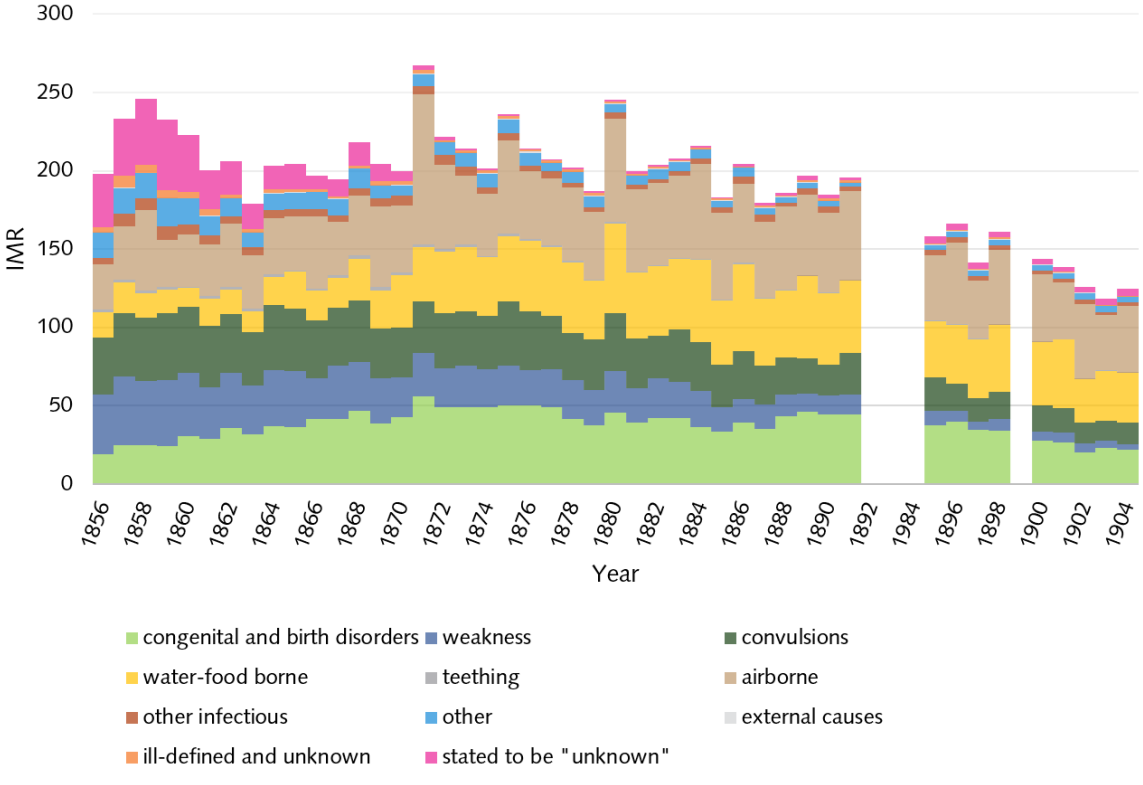
Clearly, the decline in infant mortality in Amsterdam firmly and definitively set in after the early 1880s, coming down from a rather high IMR of around 200. This development is similar for boys and girls. However, we can also see that in the 1870s, with the expansion of the city and the fast increase in population numbers, IMR is only slightly elevated, when compared to the 1860s. The potentially harmful effects this process of rapid urban expansion may have had on the health of Amsterdam's infants seem to have remained modest. By 1904 the mortality rates infants were facing in Amsterdam had come down to levels as low as 116 for girls and 131 for boys, which were in line with the situation in the countries that had been leading the secular mortality decline, such as the Scandinavian countries (Klüsener et al., 2014). Compared to many other cities in the Netherlands at the start of the 20th century, especially in the southern part of the country, the situation for infants in Amsterdam had begun to look quite favourable. As reported above, infant mortality levels in some of the southern parts of the Netherlands were much higher at that time. For instance, in the southern factory town of Maastricht IMRs were still above the 200 level until 1910, and for many of the surrounding communities the situation was no better (Murkens et al., 2022; van den Boomen & Ekamper, 2015).

Figure 3 *Infant mortality rates for Amsterdam, 1856–1904, by sex*



Sources: Calculations based on Amsterdam Cause-of-Death Database and Statistisch jaarboek der gemeente Amsterdam 1905–1909 (1910, pp. 85 and 108).

Figure 4 *Annual infant mortality rates, Amsterdam 1865–1904, by causal group*



Sources: Calculations based on Amsterdam Cause-of-Death Database and Statistisch jaarboek der gemeente Amsterdam 1905–1909 (1910, p. 85).

Over the entire period we see a clear gender gap in infant survival, with higher hazards for boys than for girls, although the gap narrows somewhat towards the end of the period. There are, however, no diverging trends between boys and girls. Over the whole period, the gender difference in infant survival is significant ($p = 0.02$). Gender gaps in infant mortality are often seen as exposing burdens and hardships caused by structural gender inequalities and vulnerabilities due to differential values attached to baby boys and girls. Therefore, this result may seem surprising as the Netherlands generally counts as a country with relative gender equality. However, our results tie in with those found for children aged 1–4 years old for the country as a whole which show that girls had significantly higher survival than boys (Poppel et al., 2009). The better survival chances of girls in infancy are a well-known phenomenon in the literature, and are deemed to be related to genetic mechanisms causing a greater vulnerability for boys in the first year of life (Waldron, 1998).

Figure 4 shows the area chart of annual IMRs by causal group (Appendix 5 shows the area chart of quinquennial infant mortality rates by causal group, and the numbers behind this chart can be found in Appendix 6). In Appendix 14 we list all (standardised) disease terms within each category with a frequency of over a hundred cases for the entire period. A number of important conclusions can be drawn from this graph. First, over the entire period the number of causal groups decreases sharply, so that by 1904 we are left with three major cause of death categories, namely air-borne diseases, water- and food-borne diseases, and congenital and birth disorders, although the first two groups are most dominant, making up 45% and 32% of all deaths respectively. The causal groups that disappeared more or less completely, already by the 1880s, are first of all the unknown and ill-defined cases. This ties in with the fact that from 1865 onwards it became mandatory for doctors to certify each death before a death certificate could be issued. Another old and vague historical term, namely "weakness", was also disappearing after the 1880s, whilst the term "convulsions", although not disappearing entirely, was strongly declining in importance. At the beginning of the 20th century 14% of infant deaths were attributed to convulsions, whilst this was a major group at the beginning of the period, making up 39% of all deaths. Secondly, we see an increase in the water- and food-borne causes, especially from the 1870s onwards, and also in the group of air-borne causes. Finally, the term "teething" was hardly used at all by Amsterdam physicians and disappeared completely after the 1890s. The cause of death data that we have for the city of Maastricht from 1864 onwards show that here too diagnostic practices did not include the use of the term teething (Murkens, 2023; Murkens et al., 2022).

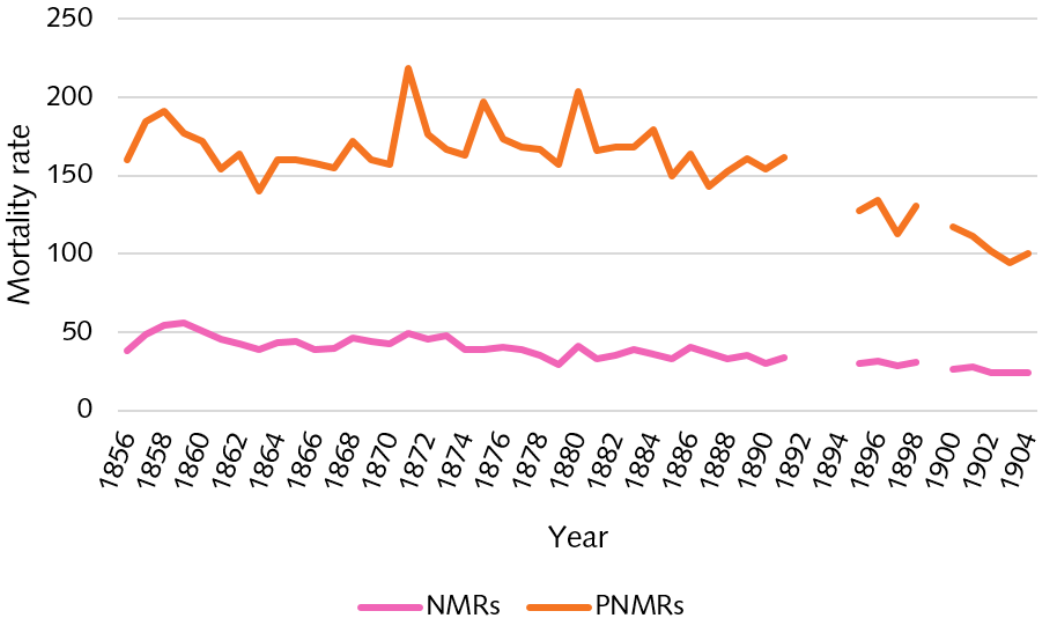
The changes shown in Figure 4 clearly indicate that diagnostic practices were moving away from the older medical terminology in the study period to be replaced by medical terms that we might recognise today as causes of death. It is important to stress that this process of diagnostic change also applied to infants, a group of patients doctors were traditionally least interested in due to their high death rates and low economic interest to the family economy (Rutten, 1986). The substitution of the old and vague medical terms for more modern disease terms raises the question whether this could have driven the increases in the air-borne and water- and food-borne disease categories. Could it be, for instance, that convulsions were somehow related to water- and food-borne diseases? This is often assumed to have been the case in the 18th- and 19th-century period (Kintner, 1986; Radtke, 2002; Walhout, 2010). And could it be that the term weakness was replaced by more precise terms from the congenital and birth disorders group? We will return to these questions below, for instance when examining the seasonality measures.

4 NEONATAL AND POST-NEONATAL MORTALITY

The decline in infant mortality that we have seen in the sections above is reflected in the development of both neonatal mortality rates (NMR) and post-neonatal mortality rates (PNMR). Neonatal deaths are defined as deaths occurring when the child was reported to be less than 28 days or less than 4 weeks old. Post-neonatal deaths are defined as deaths having occurred aged 28–364 days or 1–11 months. In Figure 5 we see a decline over time in both indicators, with neonatal mortality declining from 49,2 to 25,4 per 1,000 live births, and post-neonatal mortality declining from a level of 178,0 to 104,8 per 1,000 live births. However, the decline was somewhat stronger for neonates than for post-neonates. Between 1856 and 1904 the mortality rates for neonates and post-neonates declined by 48% and 41% respectively. This is different from the finding by van Poppel et al. (2005) that over

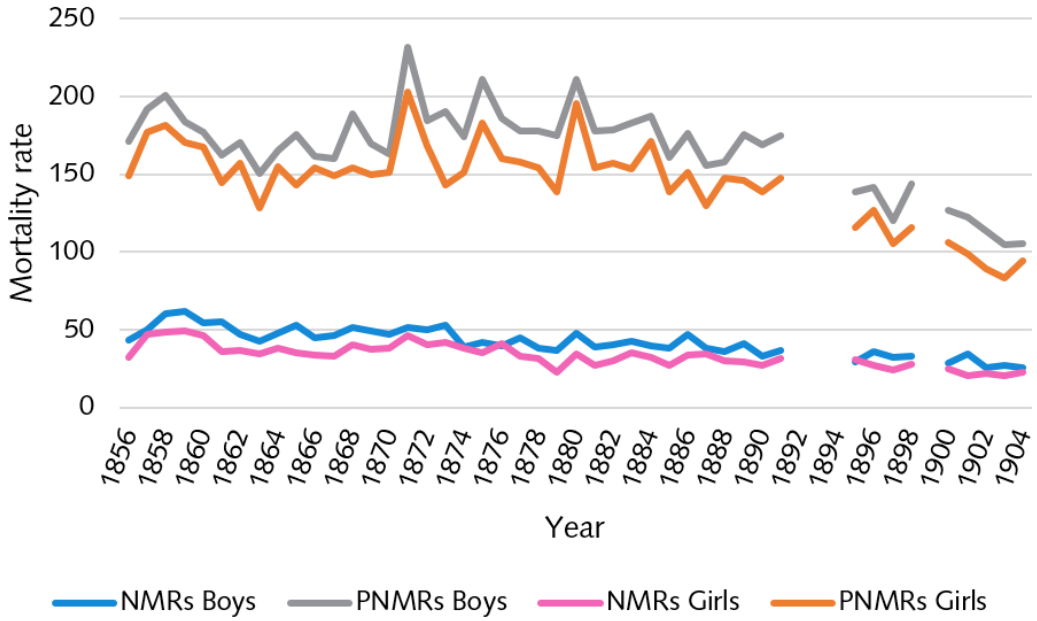
the course of the 19th century the level of neonatal mortality changed little in three provinces of the country, namely Zeeland, Utrecht and Friesland. This suggests that the mostly endogenous conditions governing the level of neonatal mortality were improving faster in Amsterdam as compared to these latter provinces. That Amsterdam was running ahead of the country as a whole in the decline of neonatal mortality is also suggested by the fact that in the late 1920s the national neonatal mortality rate was around 24 per 1,000 live births, a level Amsterdam had already reached by 1904 (de Haas, 1956). Between 1870 and the mid-1880s the PNMR in Amsterdam went up a little before beginning a steady decline towards 1904. This PNMR pattern is largely comparable to the ones found for the three provinces studied by van Poppel et al. (2005), in which PNMRs declined after the 1870s.

Figure 5 *Annual neonatal and post-neonatal mortality rates, Amsterdam 1856–1904*



Sources: Calculations based on Amsterdam Cause-of-Death Database and Statistisch jaarboek der gemeente Amsterdam 1905–1909 (1910, p. 85).

Figure 6 *Annual neonatal and post-neonatal mortality rates, by sex, Amsterdam 1854–1904*



Sources: Calculations based on Amsterdam Cause-of-Death Database and Statistisch jaarboek der gemeente Amsterdam 1905–1909 (1910, p. 85).

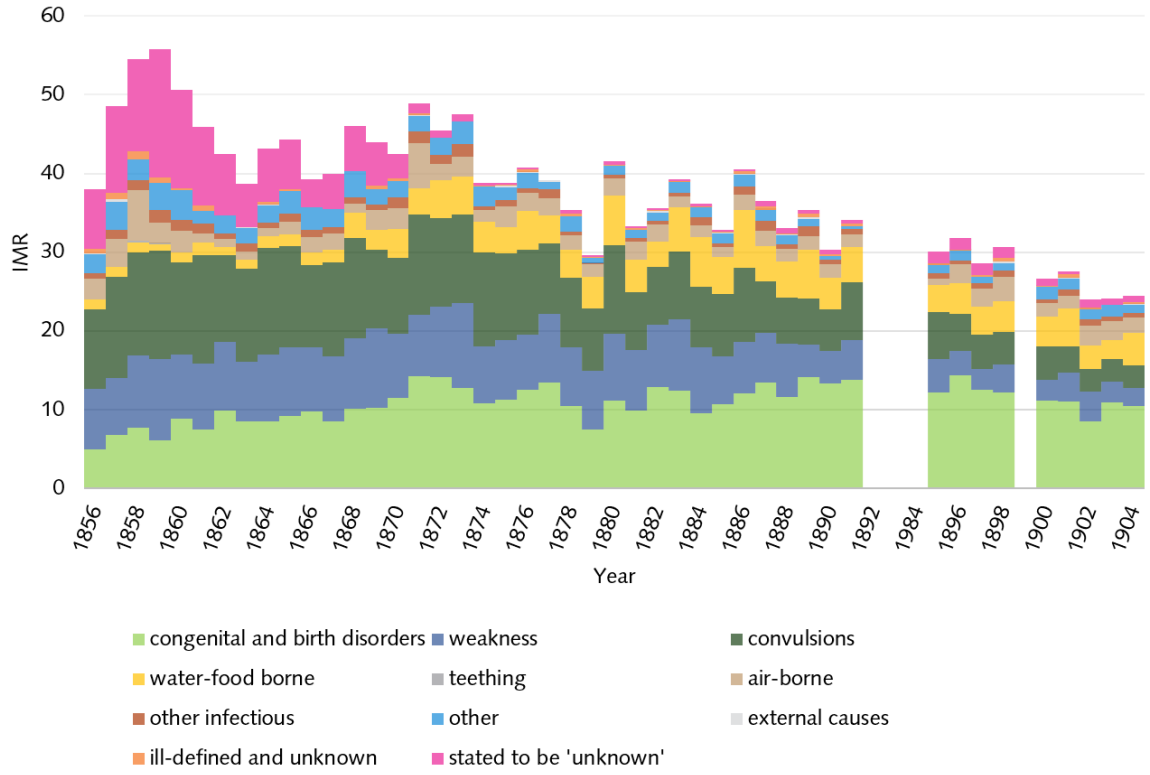
For both annual NMR and PNMR there are sex differences, see Figure 6, with mortality rates being higher for boys than for girls. Appendix 7, showing the quinquennial mortality rates, demonstrates the same. However, proportionately speaking the gender gap is mostly larger for the neonates than for the post-neonates. The table shown in Appendix 6 displays the size of sex differences for both groups. We find here that there are clearly fluctuations in sex difference for neonates between different time periods, but there is no clear trend visible of widening or narrowing gaps in mortality rates between boys and girls over time. However, we do see a widening of sex difference for post-neonatal deaths over time: between 1856 and 1904 there is a modest increase in the survival disadvantage of boys aged 2 to 12 months, going up from 1.10 to 1.20.

We next turn to an investigation of the annual cause of death patterns for neonatal mortality and post-neonatal mortality, which are shown in Figures 7 and 8 below. The quinquennial neonatal and post-neonatal graph is included in Appendix 9 and 11, and the numbers behind them can be found in Appendix number 10 and 12. For neonates we find that in the 1850s and 1860s a considerable number of deaths were registered as "unknown" for the cause of death. This practice disappeared however after the 1860s, which was undoubtedly the result of the improvements in cause of death registration introduced by the national Public Health Inspectorate Act and the Medical Practitioners Act of 1865. Throughout the study period three groups of causes dominated the neonatal pattern: congenital and birth disorders, weakness and convulsions. Between 1856 and 1870 the number of deaths due to congenital and birth disorders increased from 6 to 13 deaths per 1,000 births, which was probably related to the disappearance of those deaths from an unknown cause. The two most frequently used disease terms in the group of congenital disorders were "paedatrophia", and "atrophia infantum". Most likely, for many deaths in the first four weeks of life doctors were now resorting to these latter terms as a substitute for "unknown". Probably many of these babies were already weak from birth, of low birth weight, and unable to gain strength, especially if mothers were also of frail health and unable to breastfeed the new infant. A study by Ward (2003) in two maternity clinics in the city of Utrecht between 1880 and 1940 revealed the prominence of low birth weight, and maternal health and welfare as a risk factor for perinatal death. For neonatal deaths in Amsterdam disease terms such as weakness and convulsions began to disappear after the 1880s, whilst congenital disorders remained a dominant category. Other causal groups were of minor importance for neonatal mortality. Over the entire period very few neonatal deaths were due to air-borne diseases, and water- and food borne diseases were also not very prominent although this latter group became slightly elevated after the 1870s.

For post-neonatal mortality the picture is different, even though here the impact of the new 1865 mandatory regulations on the cause of death registration is also visible. Before that moment in time, the vague categories with old historic terms cover the majority of post-neonatal infant deaths. In the first period 1856–1859 these categories taken together, that is all deaths due to congenital and birth disorders, weakness, convulsions, teething, ill-defined and unknown, accounted for 62,4% of all deaths in this age group. By the end of the period studied here these old disease terms "only" accounted for 28,7% of all PNM deaths in Amsterdam. Once registration improved in the 1870s, the largest groups of post-neonatal deaths were water- and food borne ailments and air-borne diseases. In the early 1880s air-borne disease rates had become the largest group with a rate of 54.6 and water- and food borne disease came second with a rate of 43.1. By the beginning of the 20th century these two cause of death categories accounted for 67,1% of all post-neonatal deaths in the city.

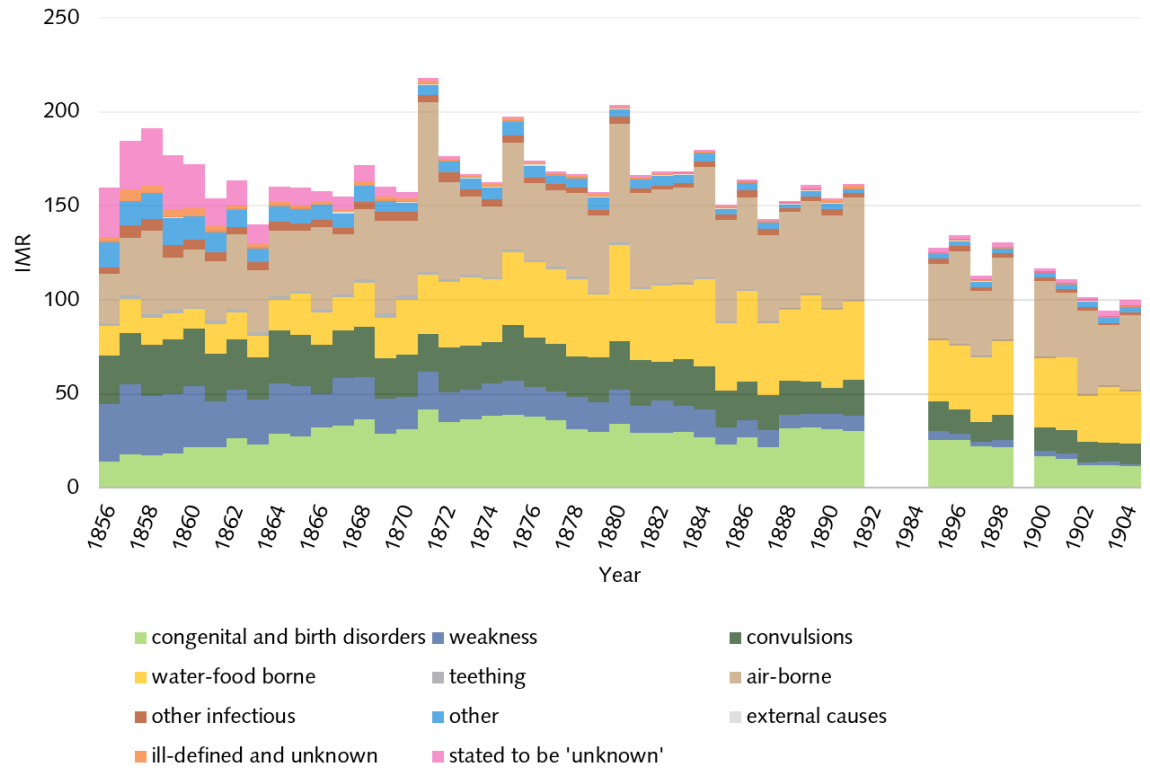
These changes in disease terms are undoubtedly related to changing diagnostic practices, and perhaps also to an increased willingness amongst doctors to invest diagnostic efforts into infant deaths. However, Amsterdam doctors only slowly became more interested in the health of infants and how infant health could be supported by correct feeding practices. Moreover, their judgements may sometimes also have had adverse effects. For instance, when in 1857 the Amsterdam Medical Practitioners' Circle debated best practices for infant feeding, its members decided that breast feeding was to be preferred. Yet, they did not at all object to cowmilk and were unable to decide whether cowmilk needed to be boiled before using it to feed infants. Even in the final years of the 19th century medical students at the University of Amsterdam were not instructed on the topic of infant feeding practices (Verdoorn 1981, p. 199). It is evident that for post-neonatal deaths the mortality-raising effect of artificial feeding is quite strong, in particular during the first six months of the infant's life (Reid, 2002). It was only after 1900 that specialised health care for infants and medical attention to the prevention of feeding disorders began to emerge in the Netherlands. For instance, in Amsterdam a specialised children's health care centre was established in 1904 which initially catered exclusively for infants from socially disadvantaged families (de Pree-Geerlings et al., 2001).

Figure 7 *Annual neonatal mortality rates by causal group, Amsterdam 1856–1904*



Sources: Calculations based on Amsterdam Cause-of-Death Database and Statistisch jaarboek der gemeente Amsterdam 1905–1909 (1910, p. 85).

Figure 8 *Annual post-neonatal mortality rates by causal group, Amsterdam 1856–1904*



Sources: Calculations based on Amsterdam Cause-of-Death Database and Statistisch jaarboek der gemeente Amsterdam 1905–1909 (1910, p. 85).

Before we turn to seasonality patterns in neonatal and post-neonatal deaths, two final remarks should be made on some specific causal groups for neonates and post-neonates. First, although the causal group of congenital and birth disorders is of a lesser importance for post-neonatal deaths than for neonates, we would have expected this group of causes to become of even lesser importance for post-neonates than what we actually find. After all, when surviving the first month of life, exogenous factors are expected to become more important. Still, if we break up the post-neonatal period, we see that the proportion of deaths due to birth disorders is lower in infants of months 7–12 than in infants of months 1–6. These results are shown in Appendix 13, and we offer some reflections on this in the conclusions.

Secondly, we find sex differences within the different causal groups for neonatal and post-neonatal deaths. We see that a male disadvantage is primarily located in the causal group of congenital and birth disorders for neonatal deaths, whilst for post-neonatal deaths the largest male disadvantages are related to water- and food borne and air-borne diseases. It is unlikely that male disadvantages occurring in the causal group of congenital and birth disorders are the result of parental neglect in any way, and are probably related to the higher biological frailty of male babies in the first year of life which is also corroborated by other Dutch studies (Walhout, 2019).

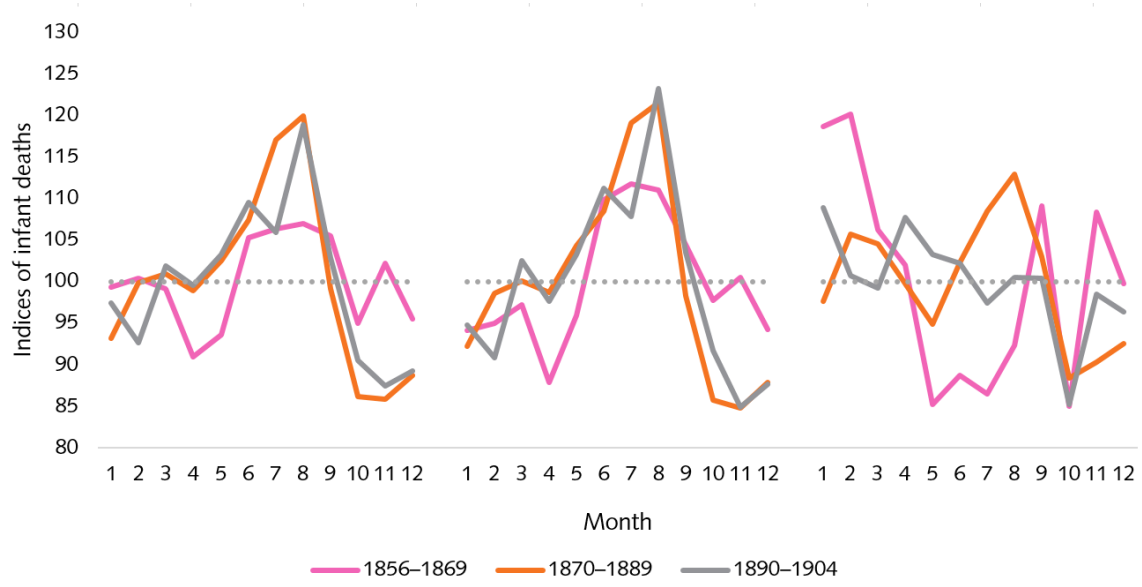
5 SEASONALITY OF NEONATAL AND POST-NEONATAL MORTALITY

Investigating seasonality patterns by causal group is very useful in the attempt to gain a better understanding of historical disease terms and changing diagnostic practices, as well as when one seeks to examine potential determinants that might have been driving the development of infant mortality. In Figure 9 we first depict the seasonality indices for infant, neonatal and post-neonatal mortality for different periods between 1856 and 1904. We can conclude that there is a clear seasonality effect for infants with mortality peaking in the summer months, which is even stronger in the later periods between 1870 and 1904. This pattern is driven by deaths of post-neonatal children which are showing high peaks in the summer months of July and August. For neonatal deaths the pattern is erratic, there is a small summer peak but only for the period 1870–1889, and there seems to be a peak in the winter months of January and February, but only for the period 1856–1869. The only other study on seasonality patterns for neonates and post-neonates in the Netherlands in the period 1830–1889 (Engelen & Hsieh, 2011) shows a clear summer peak for post-neonates, but none for infants dying in the first week of life. This is in line with our results. One other study on infant mortality as a whole, without distinguishing between neonates and post-neonates, showed that summer peaks were still the dominant pattern in the Netherlands as late as 1910–1912 (de Haas, 1956).

Summer peaks in (post-neonatal) infant mortality are often regarded as an indirect indication that mothers were not, or only for a very brief period, breastfeeding their infants. Indeed, summer peaks are not always found in 19th-century infant mortality patterns in the Netherlands. For the Dutch province of Friesland, for instance, summer peaks were not found, which typically coincides, not surprisingly, with a much lower level of IMR for that province. The authors explicitly relate this to the assumed higher level of breastfeeding in the province of Friesland (van Poppel & Mandemakers, 2002). The Amsterdam summer peak in post-neonatal mortality is a clear indication that many infants were weaned at an early stage, and also that for many of these babies hygienic provision of artificial feeding was lacking. The steady expansion of piped water in the city after the 1860s did apparently not radically change these feeding practices related hazards to infant survival. Amsterdam physicians in 1908 also explicitly related the occurrence of summer peaks in infant mortality to the lack of breastfeeding by mothers and the poor hygiene in artificial feeding practices (Saltet, 1908).

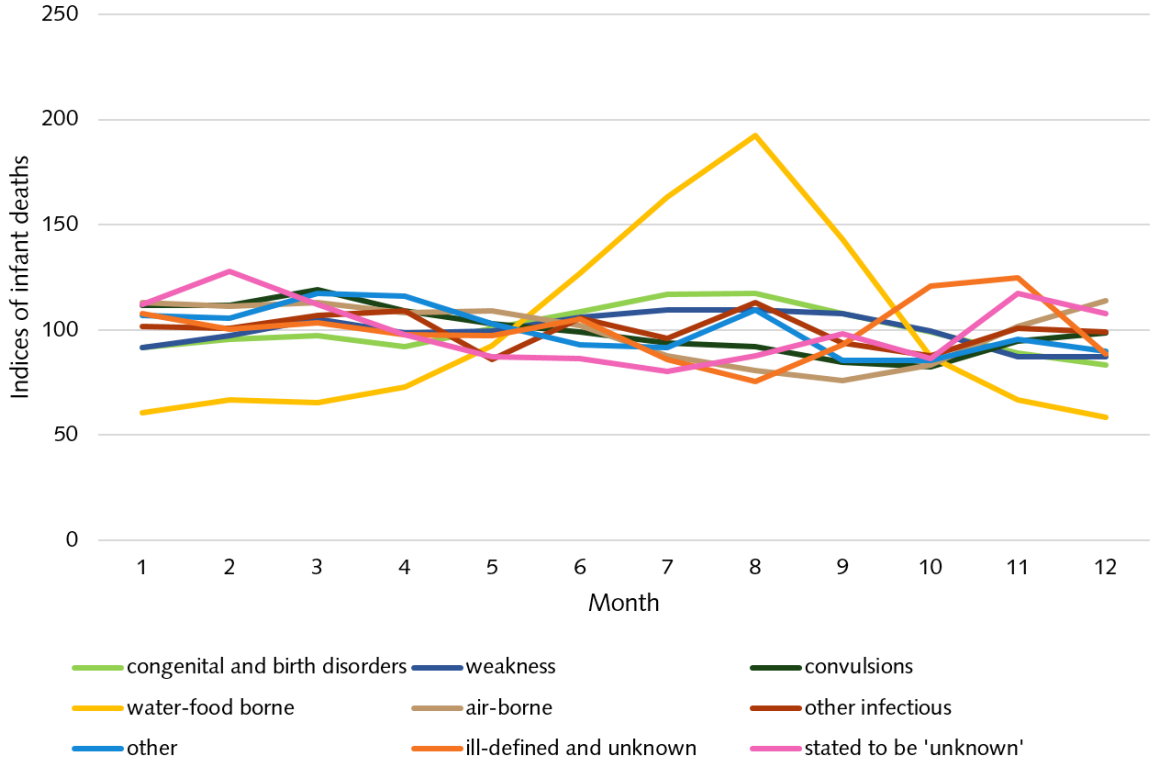
The question whether summer peaks in infant mortality are in any way related to feeding practices can be further explored in Figure 10 which shows the seasonality pattern by causal groups. For the sake of clarity, minor causal groups which did not show any effect of seasonality have been left out of this graph. Figure 10 clearly shows that the seasonality pattern in infant mortality is driven by water- and food borne diseases which typically peak in the months of July and August. For all other causal groups we do not see a clear pattern of seasonal fluctuations, although the group of air-borne diseases does show a slight dip in the summer months, a pattern which we would expect. It is noteworthy that the causal group of convulsions, which is often regarded by historical demographers as being related to water- and food borne ailments, does not in any way display a summer peak (Kintner, 1986).

Figure 9 *Seasonality indices for infant deaths, post-neonatal deaths, neonatal deaths by month, Amsterdam 1856–1904*



Source: Calculations based on Amsterdam Cause-of-Death Database.

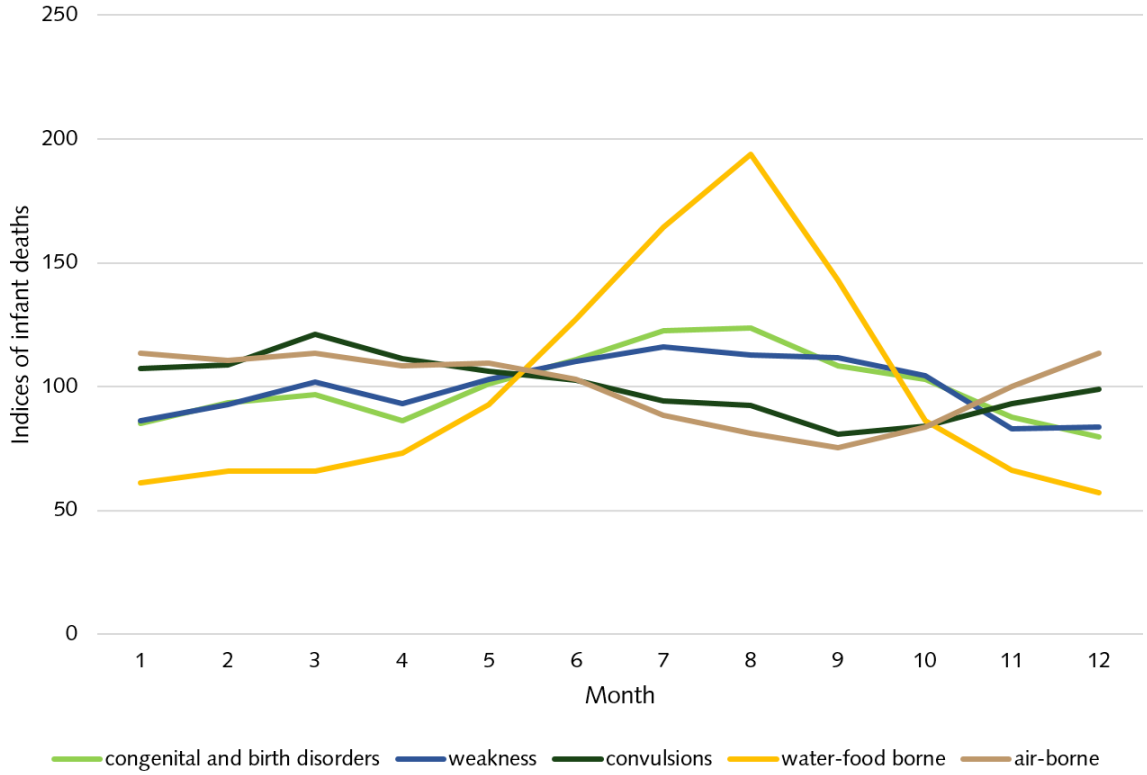
Figure 10 *Seasonality indices for infant deaths by main causal groups, all years combined*



Source: Calculations based on Amsterdam Cause-of-Death Database.

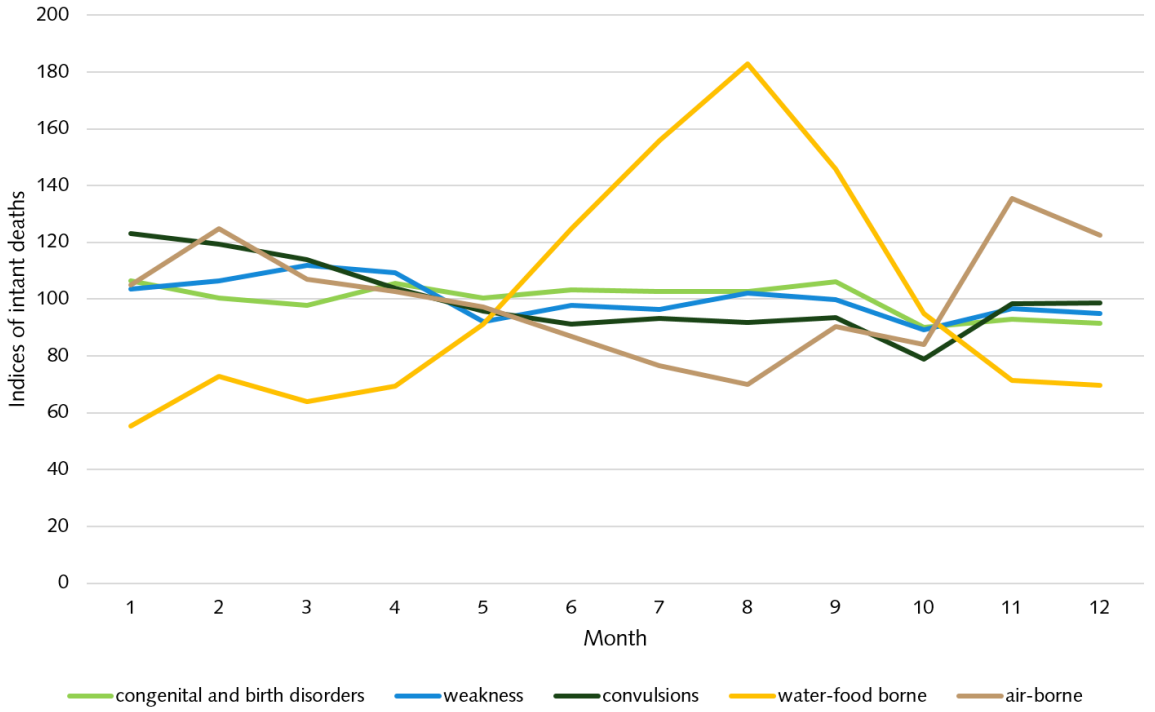
The seasonality indices for neonatal deaths by main causal groups are shown in Figure 11. It is clear that for neonatal children there is also a strong summer seasonality pattern for water- and food borne diseases. These strong summer effects suggest that even the youngest infants could be affected by a lack of breastfeeding. Obviously, these summer peaks may be the result not only of mothers unwilling to breastfeed their infants, but also because some of them may not have been able to provide breastfeeding as a result of failing health or the necessity to engage in paid work outside the home. For the neonates we see a small seasonality effect for air-borne diseases, with a dip in the summer period and a jump in the winter months of November and December. Again, for convulsions, all seasonality effects are absent.

Figure 11 *Seasonality indices for neonatal deaths by main causal groups, all years combined*



Source: Calculations based on Amsterdam Cause-of-Death Database.

Figure 12 *Seasonality indices for post-neonatal death by main causal groups, all years combined*



Source: Calculations based on Amsterdam Cause-of-Death Database.

Finally, we consider seasonality indices for post-neonatal deaths as displayed in Figure 12. The causal group of air-borne diseases displays a seasonality pattern similar to the pattern found for neonates, with a small though clear dip in the summer months. Not surprisingly, the pattern for water- and food borne diseases is repeated for this group of infant deaths. For the two causal groups weakness, and congenital and birth disorders a small summer peak is also visible — which suggests that at least some cases in this causal group

may have been related to water- and food borne diseases —, but for convulsions there is no summer peak. This conclusively demonstrates that for infant deaths in Amsterdam in this period the disease term convulsions was not related to water- and food borne problems, regardless of the particular month in which the infant had died. This finding is contrary to what has been reported for Britain where deaths from convulsions were closely associated to deaths from infantile diarrhoea (Cheney, 1984). Still, Garrett and Reid in this special issue (2022) find that in Ipswich in 1870–1910 there was also no seasonality pattern to convulsions. This suggests that there, as was the case in Amsterdam, the term convulsions was not related to water- and food borne diseases. Apart from variation in medical terminology between countries, 19th-century diagnostic practices could therefore also vary within one and the same country. This is evident when viewing the results related to convulsions in the study of causes of death for infants in the industrial city of Maastricht (Murkens et al., 2022). In this southern city in the Netherlands, at least until the 1870s, deaths due to convulsions did exhibit a clear summer peak, which disappeared as the term convulsions became increasingly outmoded.

6 CONCLUSIONS

What was killing babies in Amsterdam in the period 1856–1904, and how did infant health improve in this period? It is evident that during this time period mortality rates for infants went down significantly. In the 1850s Amsterdam infants were suffering mortality rates that belonged to the highest rates of the country. Until the 1880s one-fifth to one-quarter of all infants did not survive the first year of life, with boys being particularly vulnerable. From time to time mortality peaks pushed rates up even further, but these peaks abated after 1880. Between 1880 and 1890 a decisive turning point was reached as rates began to decline steadily, bringing IMR down to levels as low as 116 for girls and 131 for boys. This moved Amsterdam from being one of the most lethal cities in the country to one of the healthiest for infants. In addition, it brought the health conditions in the city in line with the situation at that moment in the countries that had been leading the secular mortality decline, such as the UK and Scandinavia. The increasing chances of survival applied to both the neonatal and the post-neonatal period.

It is particularly noteworthy that the considerable improvement in survival rates for infants happened at a time when the city experienced a turbulent period of urban expansion, enhanced immigration levels and significant economic growth. We do not clearly see any upward pressures on IMR of this urban turbulence in the 1870s, although it may be that without some of this turmoil IMR might have gone down earlier. In addition, we have found that these improvements in the fate of infants were brought about despite faltering progress in the provision of piped water, and a total absence throughout the period of modern sewerage. During the first decades of our study period, clean piped water was mostly only available for the better off part of the population, and during the 1880s the city even went through a proper water crisis when water provision halted while population numbers were increasing rapidly. Even at the end of the study period the clean water situation in Amsterdam compared unfavourably to a number of other major cities in Europe.

Our analysis has also shown what disease groups babies were dying of. At the start of the study period the disease terms, as stated in the cause of death registers, were frequently quite vague or ill-defined, and for some babies the entries in the registers were stated as "unknown". From the later 1860s onwards registration practices improved due to new national legislation, which is also reflected in the reduction of vague or symptomatic terms such as "marasmus", "weakness" or "convulsions". Another vague term can be found in the category of birth disorders, which mainly contains the term "paedatrophia", which only disappears after the 1900s. This also applies to the category of weakness, basically consisting of the term "atrophia". This suggests that physicians were increasingly better able and more prepared to find more precise disease terms by the 1900s.

For the entire period air-borne diseases were a prominent cause of death category, peaking in the 1880s and still making up the major group of diseases by 1904. Water- and food related ailments were also dominating the epidemiological pattern but only after the 1870s, whilst before that time these diseases were probably "hiding" in other categories and behind more vague disease terms. However for both groups we see a significant decline setting in after 1894.

The seasonality analysis of the different disease groups has clearly demonstrated the strong summer effects on the group of water- and food related causes of death, which continued to be in operation

until the end of the period. This provides confidence in the attribution of the disease terms to this causal group. Moreover, it also testifies to the shortcomings in the city's hygienic situation, as well as the fact that many mothers were not breastfeeding for a long time after birth. No summer peaks were found for the categories weakness or convulsions, so that we can safely state that neither of these two terms were in any way related to water- and food born health conditions. It is evident though that diagnostic traditions, even within the Netherlands, may differ so that convulsions elsewhere in the country did have a relationship with water- and food borne problems. Still, in a recent qualitative study of the use of the term convulsions for young children by 19th-century doctors in the Netherlands, it was convincingly argued that doctors regarded the term convulsions as a symptomatic term covering a wide range of infant and children's health conditions (Wienholts, 2022).

The second aim of this study was to test the ICD10h, the coding system devised to deal with long run historical cause of death data. We find that the groupings of causes worked quite well for our period and city data. As the table in Appendix 14 shows the groupings do not really mask important single causes that are dominant. Most single causes count for less than 1% within the different groupings. We see only few causes that stand out without being really dominant, except perhaps the cause "paedatrophia" in the group of birth disorders. Relating to this latter term, it is unclear what the difference is with the term "atrophia", which is subsumed in the category weakness when dealing with infant deaths. Both causes refer to a slowly wasting away of infants. Perhaps these two causes should be taken to "mean" or to refer to the same thing? The findings relating to the term "convulsions" demonstrate quite convincingly that it is important to keep this term separate from other causes of death when studying infant deaths. The same may be true for the terms relating to weakness.

Furthermore, we also want to offer an observation on the use of multiple cause entries in the Amsterdam registers (Appendix 15). We find that in the 1850s and early years of the 1860s infants were rarely given multiple causes of death. However, from 1865 onwards it became mandatory to have a formal confirmation of each death by a physician. Perhaps this contributed to a strong rise in the proportion of infant deaths with more than one cause, so that in the 1870s and 1880s about 20% of all infant deaths had more than one cause. Interestingly, this proportion declined again, reaching the level of only 10% of all cases in the period 1900–1904. As multiple causes are also a relevant issue for other age groups, we are conducting a separate study on this issue to explore patterns and changing traditions in this particular diagnostic practice.

Finally, this study clearly demonstrates, even though most analyses are only descriptive, the great potential of individual-level cause of death data for researchers. This type of data not only reveals the ways in which the underlying epidemiological profile changed during the decline of infant mortality, but it also offers indications as to what may have caused these changes. In the case of Amsterdam, for instance, we find that infant mortality declined rapidly in a period which was also characterised by vast urban expansion and a faltering progress of sanitary provisions such as piped water. This suggests that other factors may have been at play in bringing down these mortality levels, such as improved maternal health, declining fertility and improved living standards in terms of income and nutrition. Future studies could build on our results in several ways. For instance, analyses should be extended beyond infancy, as the one year cut-off point is artificial and mortality rates were also high in the second and third year of life. Moreover, a variety of hypotheses on epidemiological change and health inequalities can be tested with unprecedented precision and accuracy. Among them are for instance the theories by Omran (2005), McKeown (1976), the fundamental causes theory (Clouston & Link, 2021), and theories which conceptualise long run health inequalities as a process based on divergence-convergence-divergence (Mackenbach, 2020).

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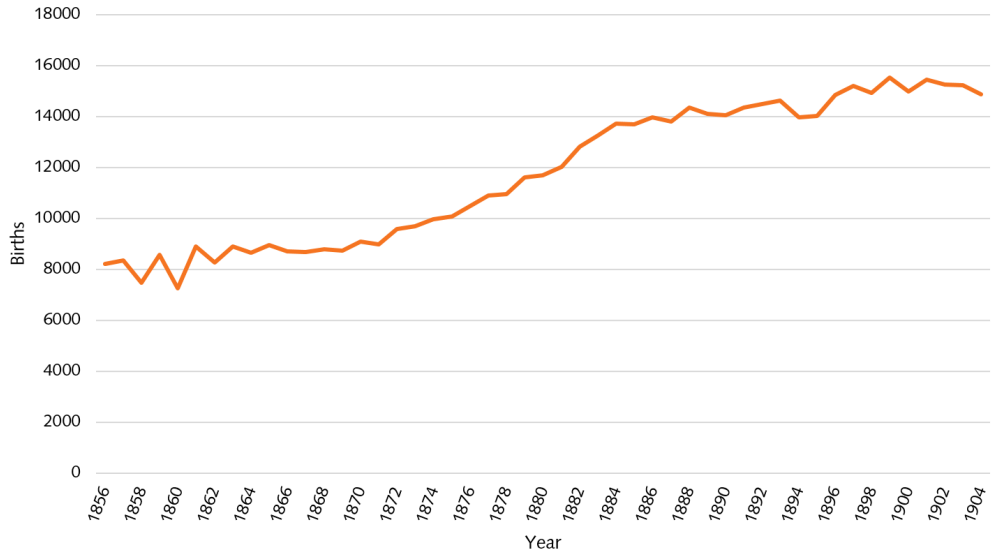
APPENDIX 1 NUMBER OF LIVE BIRTHS, AMSTERDAM 1856–1904



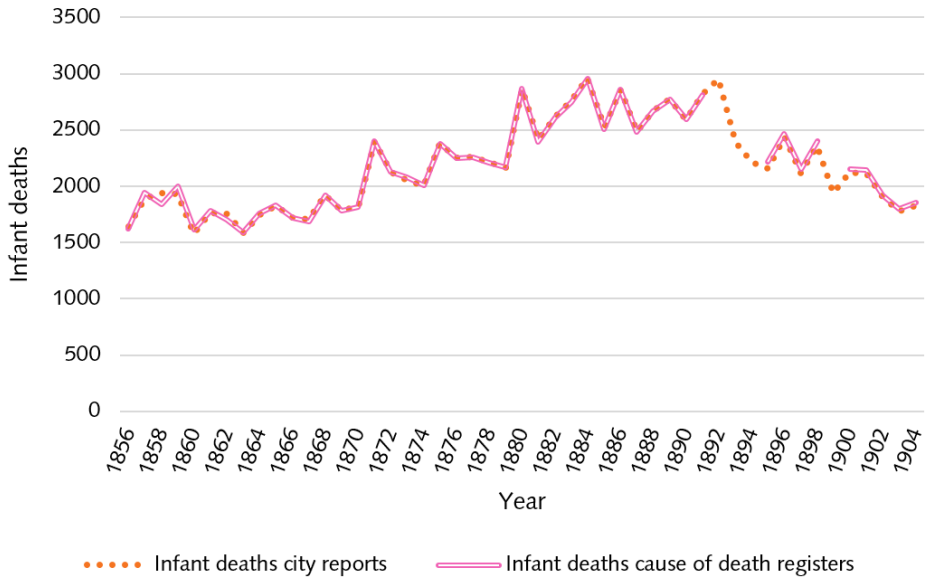
Source: Statistisch jaarboek der gemeente Amsterdam 1905–1909 (1910, p. 85).

APPENDIX 2 BASIC DESCRIPTIVES OF THE DATA SET USED IN THIS STUDY

Basic descriptives	
Years covered by the data	1856–1904
Population of Amsterdam in 1856 and 1904	259,873; 551,415
Total number of infant deaths 1856–1904	107,105
Total number of live births 1856–1904	574,968

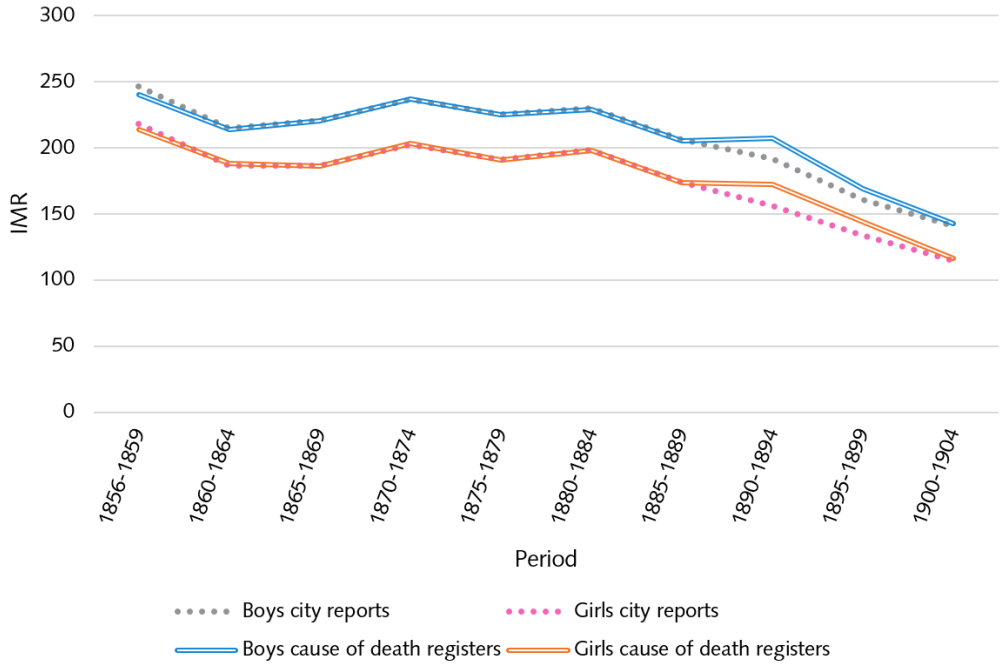
Source: Calculations based on Statistisch jaarboek der gemeente Amsterdam 1905–1909 (1910, pp. 85 and 108).

APPENDIX 3 NUMBER OF INFANT DEATHS AMSTERDAM 1856–1904, IN DIFFERENT SOURCES



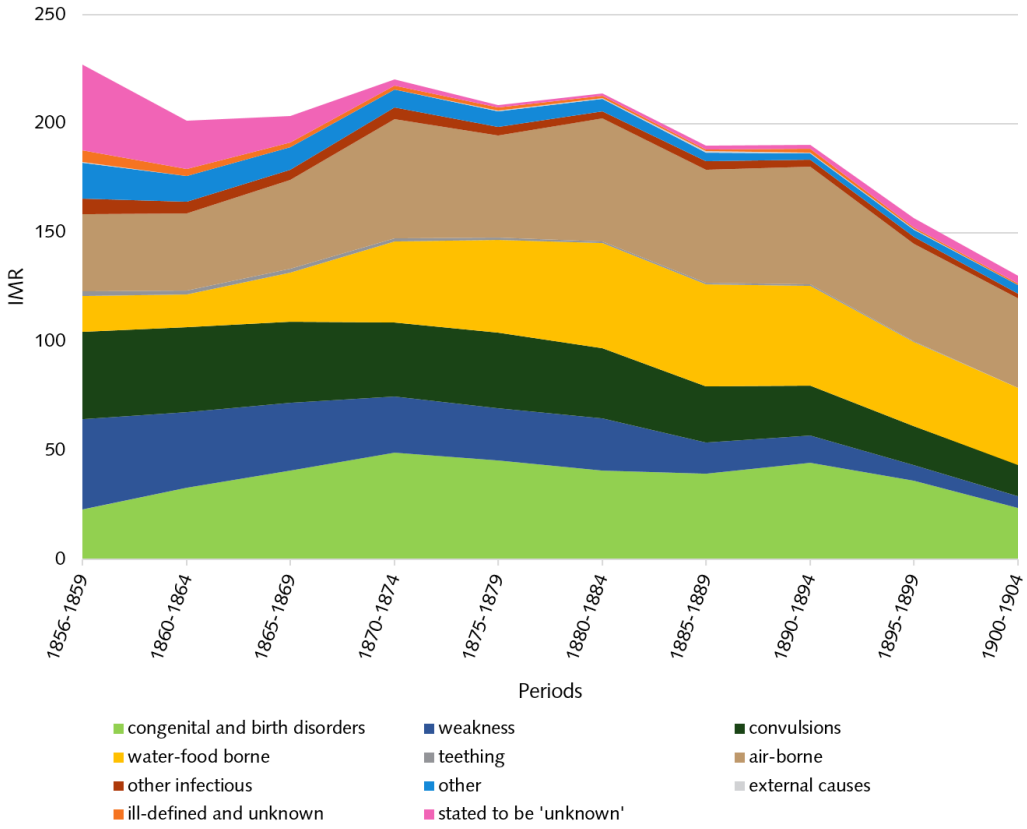
Sources: Calculations based on Amsterdam Cause-of-Death Database and Statistisch jaarboek der gemeente Amsterdam 1905–1909 (1910, p. 108).

APPENDIX 4 QUINQUENNIAL IMRS AMSTERDAM 1856–1904 BY SEX



Sources: Calculations based on Amsterdam Cause-of-Death Database and Statistisch jaarboek der gemeente Amsterdam 1905–1909 (1910, pp. 85 and 108).

APPENDIX 5 QUINQUENNIAL IMRs AMSTERDAM 1856–1904 BY CAUSAL GROUP



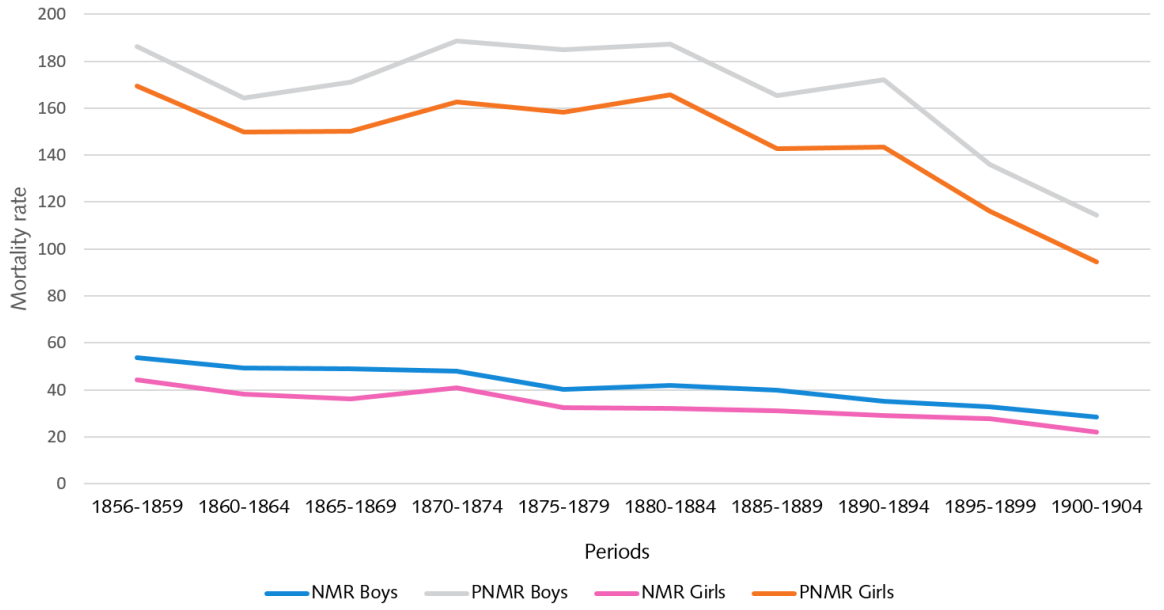
Sources: Calculations based on Amsterdam Cause-of-Death Database and Statistisch jaarboek der gemeente Amsterdam 1905–1909 (1910, p. 85).

APPENDIX 6 QUINQUENNIAL IMR BY CAUSAL GROUP, AMSTERDAM 1856–1904

	1856–1859		1860–1864		1865–1869		1870–1874		1875–1879		1880–1884		1885–1889		1890–1894		1895–1899		1900–1904	
infantcat	IMR	N	IMR	N	IMR	N	IMR	N	IMR	N	IMR	N	IMR	N	IMR	N	IMR	N	IMR	N
congenital and birth disorders	23.2	755	32.8	1379	41.0	1800	49.1	2325	45.5	2460	40.8	2593	39.4	2755	44.4	1261	36.4	2146	23.9	1811
weakness	41.4	1351	34.7	1456	31.1	1363	25.7	1216	23.9	1291	24.1	1528	14.3	1001	12.4	353	7.0	415	5.1	389
convulsions	39.9	1300	39.1	1640	37.1	1628	33.9	1602	34.7	1877	31.9	2027	25.9	1812	23.1	655	17.8	1053	14.5	1102
water-food borne	16.6	540	15.1	636	22.5	986	37.3	1763	42.5	2297	48.3	3066	46.7	3267	45.8	1301	38.6	2280	35.2	2666
teething	1.9	62	1.6	67	1.6	70	1.5	72	1.1	60	0.9	54	0.6	42	0.4	11	0.2	12	0.1	7
air-borne	35.7	1164	35.3	1484	40.8	1791	54.6	2585	46.9	2535	56.5	3587	51.9	3628	54.0	1533	44.7	2637	40.9	3097
other infectious	6.9	226	5.5	232	4.6	203	5.5	260	3.7	200	3.2	203	3.8	268	3.3	95	3.3	193	2.3	173
other	16.7	544	11.7	492	10.3	453	7.9	375	7.2	390	5.7	362	4.2	291	2.9	82	3.4	198	3.8	289
external causes	0.2	8	0.2	10	0.2	10	0.3	13	0.5	26	0.4	27	0.5	32	0.3	8	0.4	23	0.3	21
ill-defined and unknown	5.2	171	3.0	128	2.1	92	1.7	82	1.5	80	1.1	67	0.8	57	1.8	52	0.7	43	0.5	38
stated to be "unknown"	39.4	1286	22.2	931	12.2	537	2.9	135	0.9	48	1.1	70	1.8	127	1.7	48	4.1	240	3.6	270

Sources: Calculations based on Amsterdam Cause-of-Death Database and Statistisch jaarboek der gemeente Amsterdam 1905–1909 (1910, p. 85).

APPENDIX 7 QUINQUENNIAL NEONATAL AND POST-NEONATAL MORTALITY RATES BY SEX, AMSTERDAM 1856–1904



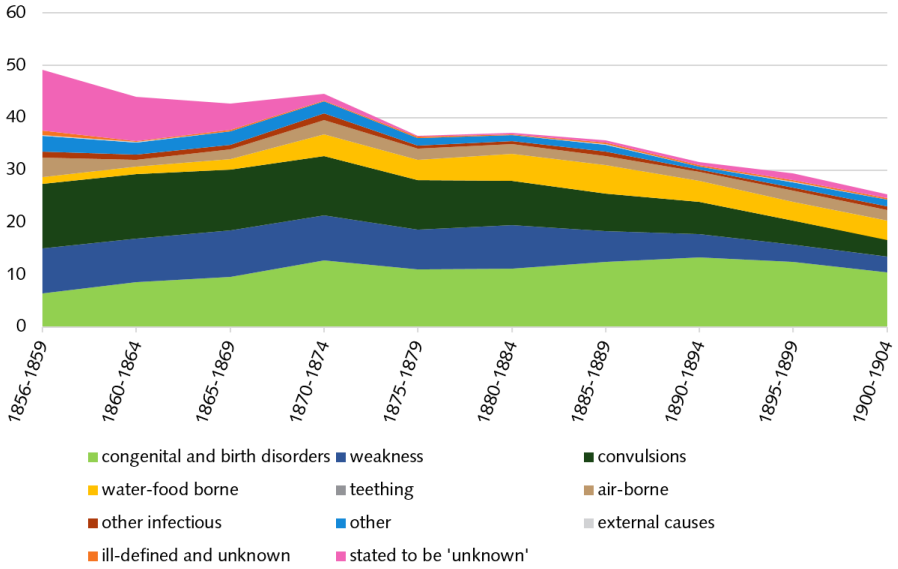
Sources: Calculations based on Amsterdam Cause-of-Death Database and Statistisch jaarboek der gemeente Amsterdam 1905–1909 (1910, p. 85).

APPENDIX 8 QUINQUENNIAL NMR AND PNMR, THE PERCENTAGE OF INFANT DEATHS, AND THE RATIO MALE/FEMALE IN THE NEONATAL PERIOD AND POST-NEONATAL PERIOD AT EACH TIME PERIOD

	NMR			PNMR			% of infant deaths		Male-female ratio	
	Total	Boys	Girls	Total	Boys	Girls	neonatal	post-neonatal	neonatal	post-neonatal
1856–1859	49.2	53.9	44.4	178.0	186.4	169.4	21.7	78.3	1.21	1.10
1860–1864	44.0	49.4	38.2	157.4	164.5	149.9	21.8	78.2	1.30	1.10
1865–1869	42.7	49.1	36.0	160.9	171.2	150.1	21.0	79.0	1.36	1.14
1870–1874	44.6	48.0	41.0	175.8	188.6	162.5	20.2	79.8	1.17	1.16
1875–1879	36.5	40.3	32.5	171.9	185.0	158.1	17.5	82.5	1.24	1.17
1880–1884	37.1	41.9	32.0	176.9	187.4	165.9	17.3	82.7	1.31	1.13
1885–1889	35.6	40.0	31.1	154.3	165.3	142.7	18.8	81.2	1.28	1.16
1890–1894	62.9	35.1	29.2	158.0	172.0	143.4	28.5	71.5	1.20	1.20
1895–1899	35.2	32.9	27.5	126.3	136.0	116.2	21.8	78.2	1.19	1.17
1900–1904	25.4	28.4	22.1	104.8	114.6	94.4	19.5	80.5	1.28	1.21

Sources: Calculations based on Amsterdam Cause-of-Death Database and Statistisch jaarboek der gemeente Amsterdam 1905–1909 (1910, p. 85).

APPENDIX 9 QUINQUENNIAL NMRS AMSTERDAM 1856–1904 BY CAUSAL GROUP



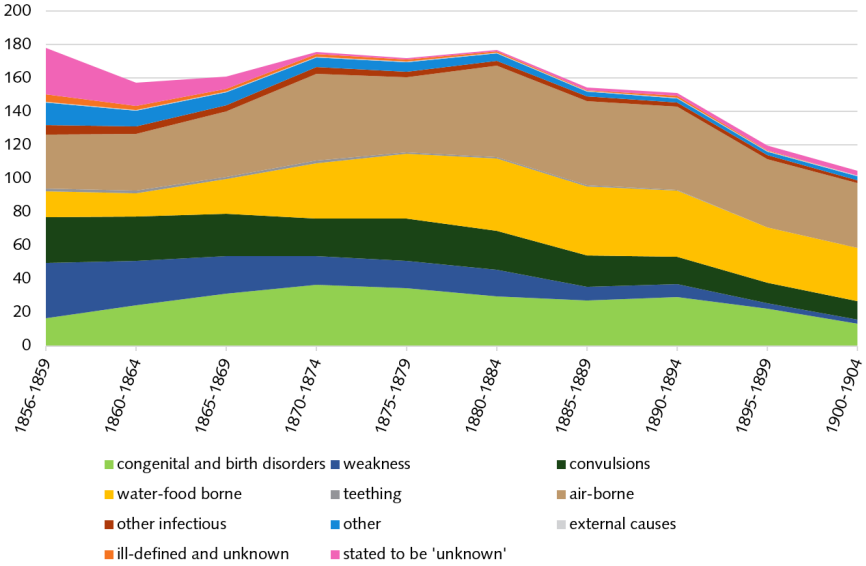
Sources: Calculations based on Amsterdam Cause-of-Death Database and Statistisch jaarboek der gemeente Amsterdam 1905–1909 (1910, p. 85).

APPENDIX 10 QUINQUENNIAL NMR BY CAUSAL GROUP, AMSTERDAM 1856–1904

	1856–1859		1860–1864		1865–1869		1870–1874		1875–1879		1880–1884		1885–1889		1890–1894		1895–1899		1900–1904	
infantcat	NMR	N	NMR	N	NMR	N	NMR	N	NMR	N	NMR	N	NMR	N	NMR	N	NMR	N	NMR	N
congenital and birth disorders	6.4	208	8.6	362	9.6	419	12.7	600	11.0	592	11.2	708	12.4	866	13.3	470	12.4	879	10.4	787
weakness	8.6	280	8.2	346	8.9	389	8.6	406	7.7	414	8.3	527	5.9	416	4.5	159	3.3	230	3.0	229
convulsions	12.5	406	12.4	521	11.6	509	11.3	537	9.4	510	8.4	534	7.1	497	6.1	214	4.7	331	3.3	247
water-food borne	1.2	38	1.3	56	2.1	91	4.1	196	3.9	209	5.2	327	5.5	383	4.0	142	3.5	245	3.6	276
teething	0.1	2	0.0	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0	0
air-borne	3.7	122	1.3	56	1.9	82	2.8	134	2.1	114	1.9	120	1.7	119	1.7	61	2.1	151	2.0	154
other infectious	1.2	38	1.0	42	0.8	37	1.2	57	0.6	32	0.6	37	0.9	64	0.5	19	0.7	48	0.7	50
other	3.0	99	2.3	98	2.6	116	2.3	110	1.4	78	1.1	73	1.2	86	0.5	18	1.0	71	1.3	99
external causes	0.1	4	0.0	2	0.0	0	0.0	1	0.1	4	0.1	5	0.1	10	0.0	0	0.1	7	0.1	5
ill-defined and unknown	0.8	25	0.3	11	0.2	7	0.1	7	0.2	10	0.1	8	0.3	18	0.3	10	0.2	17	0.2	15
stated to be 'unknown'	11.7	382	8.4	352	5.1	224	1.3	61	0.2	11	0.2	15	0.5	33	0.7	23	1.3	95	0.8	59

Sources: Calculations based on Amsterdam Cause-of-Death Database and Statistisch jaarboek der gemeente Amsterdam 1905–1909 (1910, p. 85).

APPENDIX 11 QUINQUENNIAL PNMRS AMSTERDAM 1856–1904 BY CAUSAL GROUP



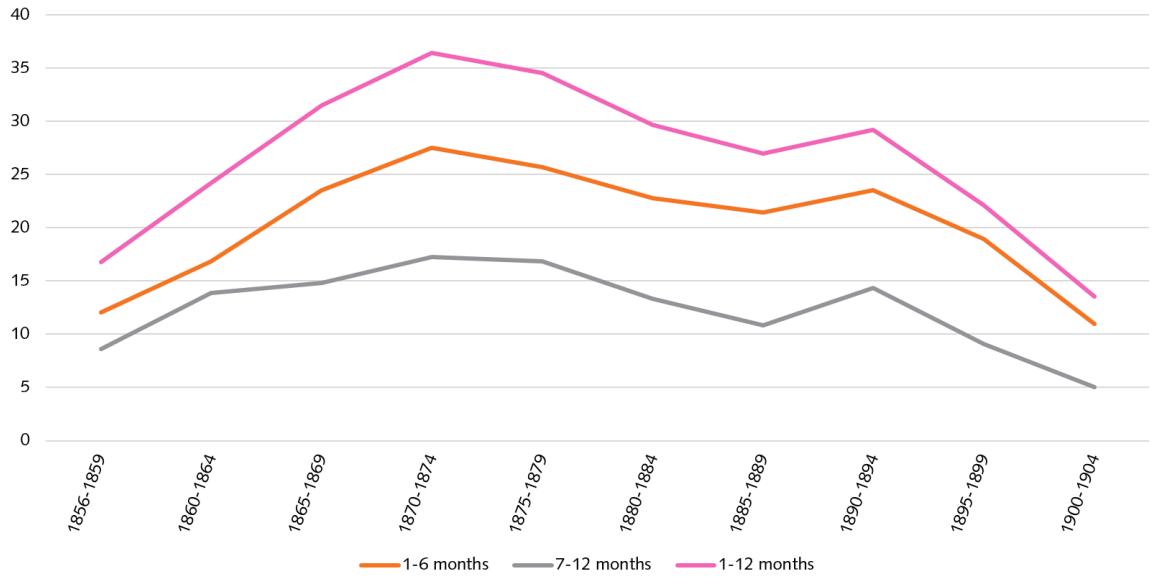
Sources: Calculations based on Amsterdam Cause-of-Death Database and Statistisch jaarboek der gemeente Amsterdam 1905–1909 (1910, p. 85).

APPENDIX 12 QUINQUENNIAL PNMR BY CAUSAL GROUP, AMSTERDAM 1856–1904

	1856–1859		1860–1864		1865–1869		1870–1874		1875–1879		1880–1884		1885–1889		1890–1894		1895–1899		1900–1904	
infantcat	PNMR	N	PNMR	N	PNMR	N	PNMR	N	PNMR	N	PNMR	N	PNMR	N	PNMR	N	PNMR	N	PNMR	N
congenital and birth disorders	16.8	547	24.2	1017	31.5	1381	36.5	1725	34.6	1868	29.7	1885	27.0	1889	29.2	1034	22.1	1563	13.5	1024
weakness	32.8	1071	26.4	1110	22.2	974	17.1	810	16.2	877	15.8	1001	8.4	585	7.6	268	3.6	251	2.1	160
convulsions	27.4	894	26.7	1119	25.5	1119	22.5	1065	25.3	1367	23.5	1493	18.8	1315	16.5	585	12.3	869	11.3	855
water-food borne	15.4	502	13.8	580	20.4	895	33.1	1567	38.6	2088	43.1	2739	41.2	2884	39.5	1396	32.8	2315	31.5	2390
teething	1.8	60	1.6	67	1.6	70	1.5	72	1.1	60	0.9	54	0.6	42	0.4	14	0.2	13	0.1	7
air-borne	32.0	1042	34.0	1428	39.0	1709	51.8	2451	44.8	2421	54.6	3467	50.2	3509	49.6	1755	40.6	2869	38.8	2943
other infectious	5.8	188	4.5	190	3.8	166	4.3	203	3.1	168	2.6	166	2.9	204	2.7	94	2.5	177	1.6	123
other	13.6	445	9.4	394	7.7	337	5.6	265	5.8	312	4.6	289	2.9	205	2.4	85	2.3	160	2.5	190
external causes	0.1	4	0.2	8	0.2	10	0.3	12	0.4	22	0.3	22	0.3	22	0.2	8	0.3	23	0.2	16
ill-defined and unknown	4.5	146	2.8	117	1.9	85	1.6	75	1.3	70	0.9	59	0.6	39	1.4	50	0.4	31	0.3	23
stated to be 'unknown'	27.7	904	13.8	579	7.1	313	1.6	74	0.7	37	0.9	55	1.3	94	1.7	59	2.6	181	2.8	211

Sources: Calculations based on Amsterdam Cause-of-Death Database and Statistisch jaarboek der gemeente Amsterdam 1905–1909 (1910, p. 85).

APPENDIX 13 CONGENITAL AND BIRTH DISORDERS MORTALITY RATE BY MONTH IN INFANCY



Sources: Calculations based on Amsterdam Cause-of-Death Database and Statistisch jaarboek der gemeente Amsterdam 1905–1909 (1910, p. 85).

APPENDIX 14 ALL STANDARDIZED CAUSES BY GROUP AND FREQUENCIES (FREQUENCY > 100)

Groupings	Cause of death in source	Number	%
air-borne	bronchitis	1796	1.80
air-borne	pneumonia	1690	1.70
air-borne	bronchitis capillaris	1438	1.44
air-borne	meningitis	1321	1.33
air-borne	pneumonia catarrhalis	1069	1.07
air-borne	scrophulosis	966	0.97
air-borne	bronchopneumonia	889	0.89
air-borne	pneumonie	746	0.75
air-borne	tussis convulsiva	721	0.72
air-borne	bronchitis acuta	618	0.62
air-borne	meningitis tuberculosa	613	0.62
air-borne	morbilli	483	0.49
air-borne	variolae	438	0.44
air-borne	meningitis cerebralis	352	0.35
air-borne	kliertering	265	0.27
air-borne	pneumonia crouposa	243	0.24
air-borne	bronchopneumonia/oedema pulmonum	227	0.23
air-borne	bronchitis/convulsiones	213	0.21

air-borne	meningitis acuta	207	0.21
air-borne	longontsteking	206	0.21
air-borne	kinkhoest	190	0.19
air-borne	tussis quinta	186	0.19
air-borne	pneumonia acuta	157	0.16
air-borne	croup	151	0.15
air-borne	tuberculosis	137	0.14
air-borne	mazelen	134	0.13
air-borne	laryngitis crouposa	133	0.13
air-borne	angina membranacea	125	0.13
air-borne	bronchitis/oedema pulmonum	123	0.12
air-borne	meningitis basilaris	123	0.12
air-borne	bronchitis capillaris/oedema pulmonum	117	0.12
air-borne	diphtheritis	112	0.11
air-borne	pneumonia lobularis	107	0.11
air-borne	tussis convulsiva/pneumonia	105	0.11
air-borne	morbilli/pneumonia	102	0.10
congenital and birth disorders	paedatrophia	13032	13.09
congenital and birth disorders	atrophia infantum	1481	1.49
congenital and birth disorders	atelectasis pulmonum	450	0.45
congenital and birth disorders	atrophia neonatorum	228	0.23
congenital and birth disorders	paedatrophia/convulsiones	203	0.20
congenital and birth disorders	onvoldragen kind	189	0.19
congenital and birth disorders	paedatrophia/collapsus	165	0.17
congenital and birth disorders	marasmus infantum	154	0.15
congenital and birth disorders	icterus neonatorum	138	0.14
congenital and birth disorders	gebrekkige ontwikkeling	133	0.13
congenital and birth disorders	vitium cordis congenita	106	0.11
congenital and birth disorders	vroegtijdige geboorte	105	0.11
convulsions	convulsiones	6294	6.32
convulsions	stuipen	3851	3.87
convulsions	eclampsia	2163	2.17
convulsions	eclampsia infantum	1389	1.39
convulsions	convulsien	885	0.89
other	hydrocephalus acutus	495	0.50
other	hydrocephalus	182	0.18
other	vitium cordis	165	0.17
other	gastromalacia	109	0.11
other infectious	encephalitis	243	0.24
other infectious	lues congenita	184	0.18
other infectious	syphilis congenita	125	0.13
other infectious	febris intermittens/convulsiones	113	0.11

stated to be "unknown"	onbekend	2900	2.91
stated to be "unknown"	geen opgave	685	0.69
stated to be "unknown"	causa ignota	135	0.14
teething	dentitio difficilis	213	0.21
water-food borne	enteritis	1792	1.80
water-food borne	tabes meseraica	1578	1.58
water-food borne	gastro enteritis	1570	1.58
water-food borne	catarrhus intestinalis	1123	1.13
water-food borne	gastro enteritis acuta	1108	1.11
water-food borne	diarrhoea	969	0.97
water-food borne	catarrhus intestinorum	876	0.88
water-food borne	enteritis acuta	548	0.55
water-food borne	dyspepsia	412	0.41
water-food borne	enteritis chronica	364	0.37
water-food borne	catarrhus gastro intestinalis	360	0.36
water-food borne	catarrhus intestinalis acutus	300	0.30
water-food borne	cholera infantum	293	0.29
water-food borne	diarrhoea infantum	288	0.29
water-food borne	dyspepsia / atrophia	272	0.27
water-food borne	gastro enteritis chronica	188	0.19
water-food borne	gastro enteritis infantum	176	0.18
water-food borne	phthisis meseraica	175	0.18
water-food borne	catarrhus intestinalis chronicus	117	0.12
water-food borne	enteritis/convulsiones	115	0.12
water-food borne	diarrhoea/convulsiones	101	0.10
weakness	atrophia	6989	7.02
weakness	debilitas	678	0.68
weakness	uittering	393	0.39
weakness	levenszwakte	392	0.39
weakness	marasmus	289	0.29
weakness	zwakte	279	0.28
weakness	algemene zwakte	190	0.19
weakness	atrophia/convulsiones	126	0.13
weakness	inanitio	112	0.11
weakness	aangeboren lichaamszwakte	107	0.11

Source: Calculations based on Amsterdam Cause-of-Death Database.

APPENDIX 15 MULTIPLE CAUSE ENTRIES BY PERIOD AND PERCENTAGE OF THE TOTAL

Period	Number	Totaal	%
1856–1859	214	7407	2.9
1860–1864	347	8455	4.1
1865–1869	799	8933	8.9
1870–1874	2262	10428	21.7
1875–1879	2380	11264	21.1
1880–1884	2807	13584	20.7
1885–1889	2757	13280	20.8
1890–1894*	1000	6464	15.5
1895–1899*	1571	10526	14.9
1900–1904	1052	9863	10.7

** Some years are missing in these periods: 1892–1894, and 1899.*
Source: Calculations based on Amsterdam Cause-of-Death Database.