

What was Killing Babies in Ipswich Between 1872 and 1909?

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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 Ipswich Between 1872 and 1909?

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ABSTRACT

This paper examines the causes of infant mortality for the port town of Ipswich between 1872 and 1909. Ipswich is the only town in England for which a complete run of computer-readable, individual-level causes of death are available in the late 19th and early 20th century. Our work makes use of the ICD10h coding system being developed to contribute to two projects: Digitising Scotland (University of Edinburgh) and SHiP — Studying the history of Health in Port Cities (Radboud University, Nijmegen). We consider annual and quinquennial mortality rates amongst Ipswich's youngest residents by age, sex, seasonality and cause. The individual causes of death not only offer insight into conditions in the town, but also highlight questions concerning how best to interpret the information provided when both medical terminology and registration practices were changing over the decades of the study. Ipswich infant mortality rates very closely mirrored those of England as a whole, rather than the most unhealthy large cities, such as Liverpool or Manchester. It becomes clear that birth itself was a major cause of neonatal, even some post-neonatal, deaths. While water-borne diseases killed large numbers in the summer months, it was the ever-present airborne diseases which carried off a greater number of small victims. Although the records offer a rich vein of data to explore, some causes of death, such as convulsions and teething, remain enigmatic and require further research.

Keywords: Infant mortality, Causes of death, Neonatal mortality, Post-neonatal mortality, Victorian Ipswich, Vaccination registers

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1 INTRODUCTION

This paper explores the diseases and conditions to which the deaths of infants in the port town of Ipswich, England, were ascribed between 1872 and 1909. As part of the special issue on causes of infant death in European port cities in the late 19th century, it has two complementary aims. First, it provides an overview of infant mortality in Ipswich to compare with the other port cities covered in the companion papers. Second, the data from Ipswich provide new insights on certain aspects of infant mortality in England and Wales, allowing us to comment on these and on the various theories regarding the influences affecting the chance that a child would survive to its first birthday in the late 19th and early 20th centuries.

Woods and Shelton (1997, p. 48, Figure 12), Woods (2000, p. 275, Figure 7.12) and Galley (2021, p. 123, Figure 4) have very effectively illustrated the fact that England and Wales' infant mortality rate (IMR) remained relatively stable over the second half of the 19th century, before rising across the 1890s and then turning sharply downwards at the start of the 20th century. Woods and his associates (Woods, 2000; Woods et al., 1988, 1989) demonstrated that the increase in the IMR during the 1890s was due to an upsurge in diarrhoeal deaths as the inadequate sanitation systems and poor hygiene in urban areas were severely strained by a series of long hot, dry summers in the later part of the decade (see Hanlon et al., 2021). More recently, Galley (2021) has suggested that more analysis of the relationship between infant mortality and the weather is needed before any firm conclusions can be reached on the nature of the interaction between them. Galley also highlighted the role that increasing urbanisation played in keeping infant mortality rates relatively stable across the later 19th century, writing "even though IMRs in most places were declining, large-scale population redistribution and urbanisation mean that an increasing proportion of infants were being born in the unhealthy towns ... increasing levels of urbanisation counterbalanced the generally improving IMRs ..., and consequently the overall national rate remained relatively stable" (Galley, 2021, p. 122–123).

There has been much debate about the efficacy and timing of sanitary provision in England and Wales.¹ As a spirit of *laissez faire* prevailed, each urban authority carried out its own programme of works, allocating its own budget and carrying out the improvements at its own pace. This created a patchwork of successful and unsuccessful schemes, with no consistently detectable impact on IMR. Any improvement in the IMR that did occur was concentrated amongst children who had survived the first month of life. Despite the success of the maternal and child welfare movement in the early years of the 20th century, mortality amongst neonates — infants less than 28 days old — remained virtually unchanged until the 1930s (Reid, 2001a, 2001b). Most of what is known about infant mortality has been gleaned very largely from the aggregate statistics presented in the annual reports and decennial supplements published by the Registrar General for England and Wales between 1837, when civil registration began, and the 1920s.²

Although this paper will not address the above debates directly, it will examine over 9,000 individual infant death records from Ipswich between 1872 and 1909 to assess whether they reflect the national picture. Large numbers of individual death records for an extended time span are very difficult to obtain in England and Wales and therefore the Ipswich dataset offers a chance to take an unprecedented look at the causes of death that doctors were diagnosing in a late Victorian town. As other papers in this special issue, this paper tests out a new international historical cause of death coding scheme, ICD10h. This allows us to examine the incidence of, and patterns amongst both 'big killers' — such as airborne and water or food borne infections in general — and certain ill-defined causes of death such as 'teething', 'convulsions' and 'weakness'. The latter three causes can tell us much about medical certification and registration of cause of death at the end of the 19th century. Examination of the age, sex and seasonality patterns of such conditions will help to assess whether it is acceptable to treat these ill-defined causes as manifestations of other conditions — teething and convulsions are often assumed to represent diarrhoea, for example — and thus generate a more robust classification scheme for causes of infant mortality. We believe this detailed case study of infant mortality in the port of Ipswich will inform the understanding of the decline of infant mortality in England and Wales, as well as contributing to the wider comparison of the experience of European ports.

1 The literature on this debate is very extensive. See, for example: Bell and Millward (1988), Harris and Hinde (2019), Szreter (1988, 1997), Szreter and Mooney (1998), Torres et al. (2019), Williams (1992), Woods et al. (1988) and Woods and Woodward (1984).

2 These can be consulted at www3.histpop.org. The reports were replaced by the *Registrar-General's Statistical Review of England and Wales* from 1921.

1.1 A BRIEF INTRODUCTION TO IPSWICH

Ipswich is the 'county town', or administrative centre, of Suffolk, a largely agricultural county in East Anglia, England.³ The town is considered to be one of the oldest in England and has had a long history as a trading port, particularly with the Baltic. It was once a *kontor*, or trading post, for the Hanseatic League. It lies on the River Orwell, 10 miles upstream from today's major ports of Harwich and Felixstowe.

In the later 19th century Ipswich remained an important port, particularly for coastal trade, receiving coal and iron from elsewhere in Britain (see Figure 1). The town had developed as a centre of manufacturing, and shipbuilding flourished on the banks of the River Orwell. Iron foundries made farm machinery for use in local rural areas and for export as well as producing parts for the construction of railways and their rolling stock. Fertilizer was manufactured, and the town was home to firms processing farm produce, such as brewers, maltsters, grain millers and seed oil producers. In addition, there were printing firms, companies producing tobacco and clothing, and a substantial military presence.⁴

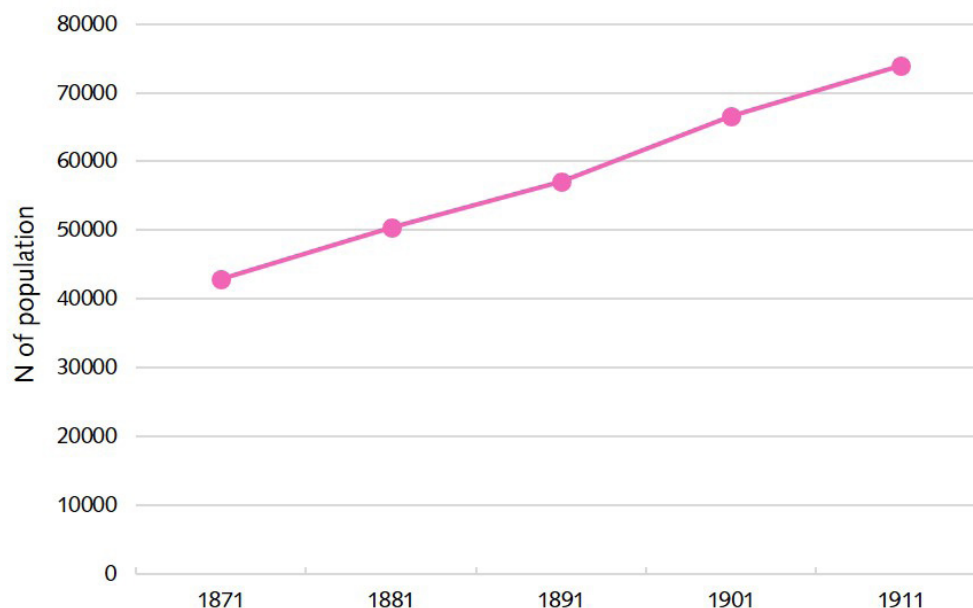
In 1871 Ipswich was home to just under 43,000 people. Over the next four decades the population grew steadily, to reach 74,000 by 1911 (see Figure 2), an increase of some 78%, rather larger than the 58% increase experienced in England and Wales as a whole.⁵ Much of Ipswich's population growth was due to natural increase, as some 69,000 births occurred in the town over the four decades, but only 43,000 deaths, 9,904 of which were to infants less than one year old.⁶ The Registrar General of births, deaths and marriages in England and Wales (1885, 1895, 1907, 1919) reported that the IMR in Ipswich was 154 deaths per 1,000 births in 1871–1880 and 137, 156, and 127 in the following three decades, while the corresponding rates for England and Wales were 163, 142, 153 and 128.⁷ The Ipswich infant mortality rates were thus very close to the national average; this was not a town notorious for high mortality, although the rates were undoubtedly higher than those in the surrounding rural districts.⁸

Figure 1 Ipswich Island site, taken from a silo around 1900



Source: Image A593, Richard Pawsey Collection, Ipswich Maritime Trust Image Archive. Reproduced with the kind permission of the Ipswich Maritime Trust.

- 3 Despite its long history, Ipswich does not have a cathedral and therefore technically remains a town rather than a city.
- 4 For further details of Ipswich's history see <http://www.localhistories.org/ipswich.html>; <https://www.britainexpress.com/counties/suffolk/ipswich.htm>.
- 5 Ipswich Registration District (RD) boundary underwent some changes 1891–1901, but these involved the loss and addition of rural areas surrounding the town, and would have had little effect on the overall population of the RD. The evolution of Ipswich Registration District boundaries 1871–1901 can be viewed at www.populationspast.org.
- 6 Figures derived from the Ipswich dataset.
- 7 These volumes can be consulted at <http://www3.histpop.org/>.
- 8 The contrast in rates can be seen at: <https://www.populationspast.org/imr/1881/#13/52.0576/1.1621/bartholomew>.

Figure 2 *Population growth, Ipswich 1871–1911*

Sources: *Census Reports for England Wales, 1871–1911*

Hall (2005) and Hall and Drake (2006) confirmed that infant mortality in Ipswich conformed to the national pattern of an increase in IMR, driven by diarrhoeal disease, during the long, hot summers of the 1890s.⁹ As in many other towns in England, a programme of sanitary improvements was initiated in Ipswich towards the end of the 19th century. Work on a network of sewers began in the 1880s,¹⁰ but the town did not have a 'comprehensive sewerage system' until 1897, and had to wait until 1907 for this to be completed (Hall & Drake, 2006, p. 167). Farmers in the agricultural areas surrounding the town had traditionally bought night soil — human excrement — from the inhabitants, to use as fertiliser. Even in the late 19th century, many residents kept the contents of their chamber pots and ash closets in privy middens against the walls of their houses, awaiting collection. It was estimated that there were 8,000 such middens in Ipswich in 1894, but by 1906 there were just 125, thanks largely to the work of the local Medical Officer of Health (MOH) (Hall & Drake, 2006, pp. 156, 167). For most of the 19th century the town's water supply was provided by private companies, although in the middle of the century it was reported that as many as half of the households were "without any supply whatever, or (had) to depend upon wells, whose impurities (were) frequently a subject of just complaint" (Glyde, 1850, p. 30). In 1892 the council took control of the water supply and "by 1900 virtually every one of the town's 14,000 households had running water".¹¹ Morgan (2002) has pointed out that all forms of horse traffic increased markedly in the last decades of the 19th century, adding to the risk of fly-borne disease, particularly in hot weather. In 1880 horse-drawn trams began to run along the main streets in Ipswich, but from 1903 the trams were electrified,¹² considerably reducing horse traffic and the amount of manure deposited on the streets. Such changes might be expected to have had a disproportionate effect on infant mortality as this age group was particularly vulnerable to diarrhoeal mortality.¹³

This paper will examine the causes of death registered for Ipswich's infants to understand the 'broad-brush' story in finer detail.

9 Interestingly, G.S Elliston, Medical Officer of Health for Ipswich noted in his 1895 report that "the weather during the Summer and Autumn of 1895 was certainly such as generally favours an excessive mortality from Diarrhoea, but was hardly sufficient to account for the alarming death-rate [that year]" (Elliston, 1896, p. 9).

10 <http://www.localhistories.org/ipswich.html>

11 <http://www.ipswich-lettering.co.uk/water.html> (accessed 30.04.2021) Harris and Hinde note that the council took out a loan of £230,000 to fund this work (with thanks for a personal communication).

12 <http://www.localhistories.org/ipswich.html>; <https://allaboutipswich.com/blog/2020/timeline>

13 See Morgan (2002).

1.2 DATA

In England and Wales large numbers of individual death certificates — and the information they contain on cause of death — are expensive to obtain, as each has to be bought separately from the General Register Office. Our study makes use of individual-level information taken from the smallpox vaccination birth registers for Ipswich and a copy of the town's death registers covering the years 1871–1910.¹⁴ In 1853 legislation was passed in England making it compulsory to have all children vaccinated against smallpox before they were three months old.¹⁵ From 1871 vaccination officers were appointed to administer the vaccination system, taking over this role from local registrars. Registrars were expected to send monthly lists of births and deaths of infants under one year old to the officers. These lists formed the basis of the vaccination birth register which recorded all the infants the vaccination officer had to account for, and the vaccination death register which noted the infants who had 'exited' the system and therefore did not need to be brought for vaccination.¹⁶

The Suffolk Record Office holds the Ipswich vaccination birth registers for 1871–1930 and 1940–1958, but instead of the vaccination death registers, listing only infant deaths, it holds copies of the civil registers of death for the town from 1851–1945, made for the local MOH (Drake & Razzell, 1999, p. 40, note 45). Ipswich is therefore the only town in England for which a significant run of birth and death certificates relating to the entire population is available to researchers. The death records include the cause of death information reported on each record: deceased's name, age at death and occupation as well as the place and cause of death. Usually, the registrar would have copied the cause of death information from the medical certificate of cause of death issued by the doctor who had attended the deceased in their last illness.¹⁷ The data from the vaccination birth and death registers for 1871–1910 were transcribed as part of a project led by Peter Razzell at the Open University which also cleaned, standardised and partially linked them to the individual census records of Ipswich from the same four decades (Razzell et al., 2007).¹⁸ It is important to note that in England and Wales stillbirths were not registered systematically until 1927, so are not included in either the birth or death registers in this dataset.

Not all births and deaths occurring in 1871 and 1910 were included in the registers transcribed, and we excluded these years from our analyses, so our calculations are based on the 65,961 births and 9,356 infant deaths that occurred in Ipswich between January 1st 1872 and December 31st 1909. The quinquennial figures presented below thus run from years ending in 2 to those ending in 6, or from years ending in 7 to those ending in 1. The final period, 1907–1909, is three rather than five years long.

1.2.1 A NOTE ON AGES

Throughout this paper 'infant', 'neonatal' and 'post-neonatal' are defined by reference to the age at death reported on each infant's death certificate. The exact age of the child was not verified by linking each death to the relevant birth. While this would be possible for the great majority of the deaths registered in Ipswich, there were some infants, particularly those who survived well into their first year before dying, who moved into Ipswich after their birth and therefore could not have their age at death

14 The Ipswich vaccination birth registers for 1871–1930 and 1940–1958 (47 volumes) are held at Suffolk Record Office, Ipswich (Ref DC/3/8/1–47). See Drake and Razzell (1999, p. 40, note 45), and Razzell et al. (2007), Study documentation.

15 For a history of smallpox inoculation and vaccination in England see Drake and Razzell (1999) and Razzell (1977), also Galley (2021).

16 The vaccination death registers were not expected to include the cause of each infant's death but, on occasion, they did.

17 The cause of death in cases where the deceased had died suddenly, unexpectedly or by violent means would have been established by a coroner. Unusually, some of the records in the Ipswich vaccination death registers include notes on the categories into which the causes of death were classified when they were tabulated for publication. Hall and Drake (2006, p. 150) suggest that the MOH added these notes to his copies of the death registers.

18 They also formed the basis of an Open University Ph.D. thesis (Hall, 2005).

verified.¹⁹ Similarly, some Ipswich-born infants would have migrated away from the town with their parents before their first birthday and we cannot tell if, or when, they died. We have assumed that the number and survival of in-migrating and out-migrating infants were roughly equal, but further investigation, involving record linkage outside the town, would be needed to confirm this.

We recognise that our results may be affected by our reliance on reported ages, as age can be reported in a number of ways, and this, in turn, is often affected by the age at which the infant died. Deaths in the first day or two of life may have been reported in hours, or even minutes, whereas those dying in the first two weeks frequently had their death reported in days. By the end of the first month of life children's ages were more likely to be reported in weeks and by the end of the first year months were most commonly used. It is not always clear where a 'cut off point' between age categories occurs. A child reported to have died aged '1 month', for example, might have been exactly a month old, having been born on the fourth of one month and dying on the fourth of the next month, but it is more likely that the informant meant that the child was 'at least one month, but not yet two months old', so could have been aged anything from 28 days, or four weeks, old up to a maximum of 61 days, or eight and a bit weeks, if the parents reckoned age by summing the number of days in each month.

All calculations below relating to age are subject to the above caveats. Infants are defined as children less than one year, or 12 months, of age. Neonatal deaths are taken as deaths occurring when the child was reported to be less than '28 days' or '4 weeks' old. Post-neonatal deaths are taken as those stated to have died aged 28–364 days, 4–51 weeks, or 1–11 months.²⁰ We do not believe that our use of 'reported ages' has a major distorting effect on the measures of neonatal and post-neonatal mortality, but comparisons with trends in other papers where age at death has been calculated more precisely, by subtracting the date of birth from the date of death, require an element of caution.

2 AN OVERVIEW OF INFANT MORTALITY RATES

As Figure A1 in Appendix A shows, births in Ipswich rose steadily from the 1870s until around 1902, although the pace of growth was less marked from 1886 onward. From 1902 the annual number of births plateaued briefly before turning downwards from 1904. As was the case elsewhere in England, fertility decline had started in the town well before 1904, but the initial stages of the decline were masked by in-migration as Ipswich grew in size and prosperity. Analysis, not shown here, suggests that there was little, if any, seasonality to the birth rate in Ipswich in this period, as befits a town with a diverse profile of industries.

Figure 3 shows that the infant mortality rate (IMR) in Ipswich fell from around 140 infant deaths per 1,000 births in 1872 to below 80 deaths in 1909. This was not a smooth or continuous process, however. A certain degree of fluctuation is to be expected with a relatively small dataset such as this, but infant mortality in the 1870s and 1890s was arguably more volatile than the period of relatively low and stable mortality in the 1880s. The volatility in the 1890s is characteristic of the 'urban-sanitary-diarrhoeal' effect felt by many English cities, during the series of long hot summers in that decade (Galley, 2021; Williams & Galley, 1995; Woods et al., 1988, 1989). From the early years of the new

19 An exercise which attempted to link the 6,874 infant deaths registered in Ipswich between the 1st of January 1872 and the 31st December 1900 to each deceased child's birth in the birth register found that in 503 cases (7.3%) no appropriate birth could be identified. In 213 of these cases (3.1% of all cases) this was because the names of the parents, needed in the linking process, were not provided on the death certificate. This left 290 deaths (4.2% of the total) where at least one parent's name was given but still the child's birth could not be identified in the Ipswich vaccination birth registers, giving an (upper) estimate of the proportion of infants dying in the town who had been born elsewhere.

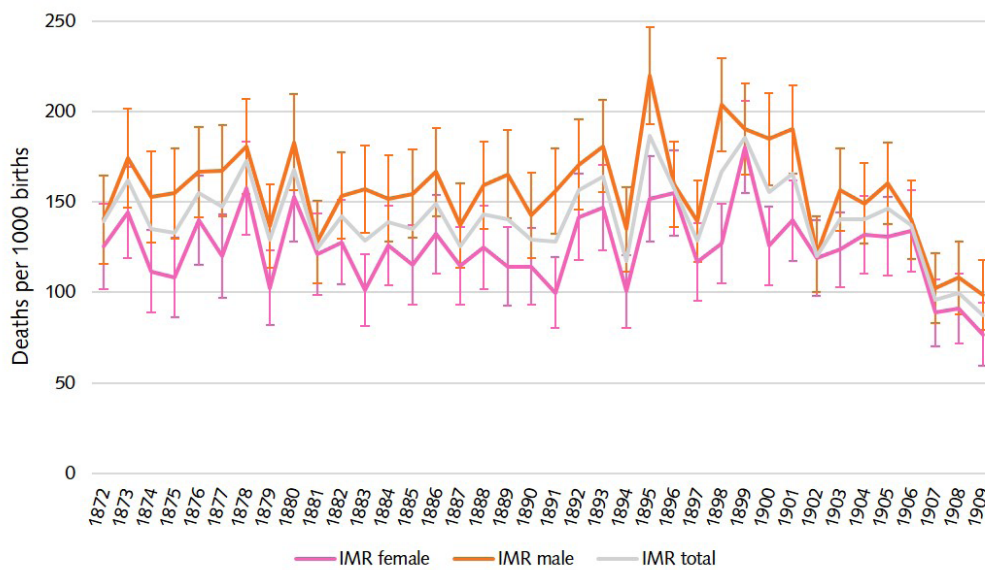
20 The way that ages were reported on the death certificates changed across our study period. Children in the second, third, and particularly the fourth weeks of life were much more likely to have had their age at death recorded in days by the end of the study period than they were at the beginning. Those dying in the second, third, and fourth months of life were more likely to have their ages recorded in weeks at the beginning of the period, but in months by the end. It is not clear whether these changes were due to a change in the way registrars recorded ages, or to changing fashions in the reporting of babies' ages. However, it is possible, given the changes, that a proportion of deaths classified as neonatal in the 1870s were assigned to the post-neonatal category in the 1890s and 1900s, affecting the mortality rates in the two age groups.

century, however, infant survival began to improve relatively steadily in Ipswich, again echoing the trend in infant mortality for other urban areas of England (Woods et al., 1988, 1989).

In most places and historical eras, and at almost all ages, males tend to have higher mortality rates than females (Maiolo & Reid, 2020; Reid et al., 2016). As shown in Figure 3, this pattern is clearly visible amongst infants in Ipswich. The error bars show that this difference was not always statistically significant, due to relatively small numbers of deaths in individual years. There is, however, some suggestion of a reduction in the difference between the sexes towards the end of the period.

The quinquennial IMRs in Figure 4 show much smoother curves, and the larger numbers in the aggregated series confirm the impressions given by the annual series. Male infant mortality was significantly higher than female across the last 30 years of the 19th century, particularly between 1897 and 1901, but the difference between the two sexes became much less marked after the turn of the century.

Figure 3 Annual IMRs, Ipswich 1872–1909



Note: The error bars show 95% confidence intervals.

Source: Calculated from vaccination birth and death registers, Ipswich 1872–1909.

Figure 4 Quinquennial infant mortality rates, Ipswich, 1872–1909*



* The final period includes only three years.

Note: The error bars show 95% confidence intervals.

Source: As Figure 3.

3 CAUSE OF DEATH

Doctors in England during our study period were instructed to fill in medical certificates of cause of death by listing the 'primary' cause of death — the disease initiating the 'train of events leading to death' — first, followed by any 'secondary' or 'contributory' causes.²¹ Under the ICD10h coding system each contributing cause of death is coded 'literally', each separate term being given its own code. For example, the term 'pulmonary tuberculosis' is coded A16.904, 'pulmonary consumption' is given A16.902, 'consumption' on its own A16.906, 'phthisis' A19.901 and 'pulmonary phthisis' A16.903. This allows the terms to be classified in a number of ways, depending on whether, say, a researcher is interested in 'tuberculous' conditions generally, or conditions specifically said to affect the 'lung', or wants to follow how one term superseded another.²² Many of the Ipswich death records listed multiple contributing causes, but in almost all such cases the second and third causes listed were symptomatic, while the first cause of death listed, COD1, could be identified as the 'underlying' cause, triggering the other causes.²³ Hence, the great majority of our analyses are based on COD1.

In Figure 5 infant deaths from Ipswich have been assigned to the 11 categories, based on the ICD10h coding system, used by all the SHiP studies contributing to this special issue (see Appendix B). These categories were designed specifically for the analysis of infant mortality, and therefore show a rather different picture from that gleaned from the published annual reports of the Registrar General for England and Wales, where the categorisation used was applied to all age groups and the 'other causes' category often contained a large number of deaths. For example, the supplement to the Registrar General's *Forty-Fifth Annual Report (1871–80, BPP 1884–85 XCII [C.4564])* recorded 2,404 infant deaths in Ipswich. Of these 389 were ascribed to 'respiratory disease', 420 to 'diarrhoea and dysentery' and 224 to 'diseases of the nervous system'. A further 1,023 (42.5% of the total) were in an 'other causes' category, with no indication of the relative contribution of particular causes.

The Ipswich death registers show that over the 1872–1909 period as a whole, the 'congenital and birth disorders' category was the largest killer of infants in Ipswich, followed by 'airborne diseases'. The 'water-food borne diseases' category was some way behind in third place with the 'weakness' category fourth. 'Convulsions' was the fifth largest category, but it should be noted that this category, like the much smaller 'teething' category, included only one specific cause, whereas the largest four categories and the 'other non-infectious' and 'other infectious' categories all contained a combination of various conditions or diseases. There were very few infant deaths in the Ipswich death registers from the study period where the cause of death was ill-defined, indecipherable or stated to be 'unknown'.

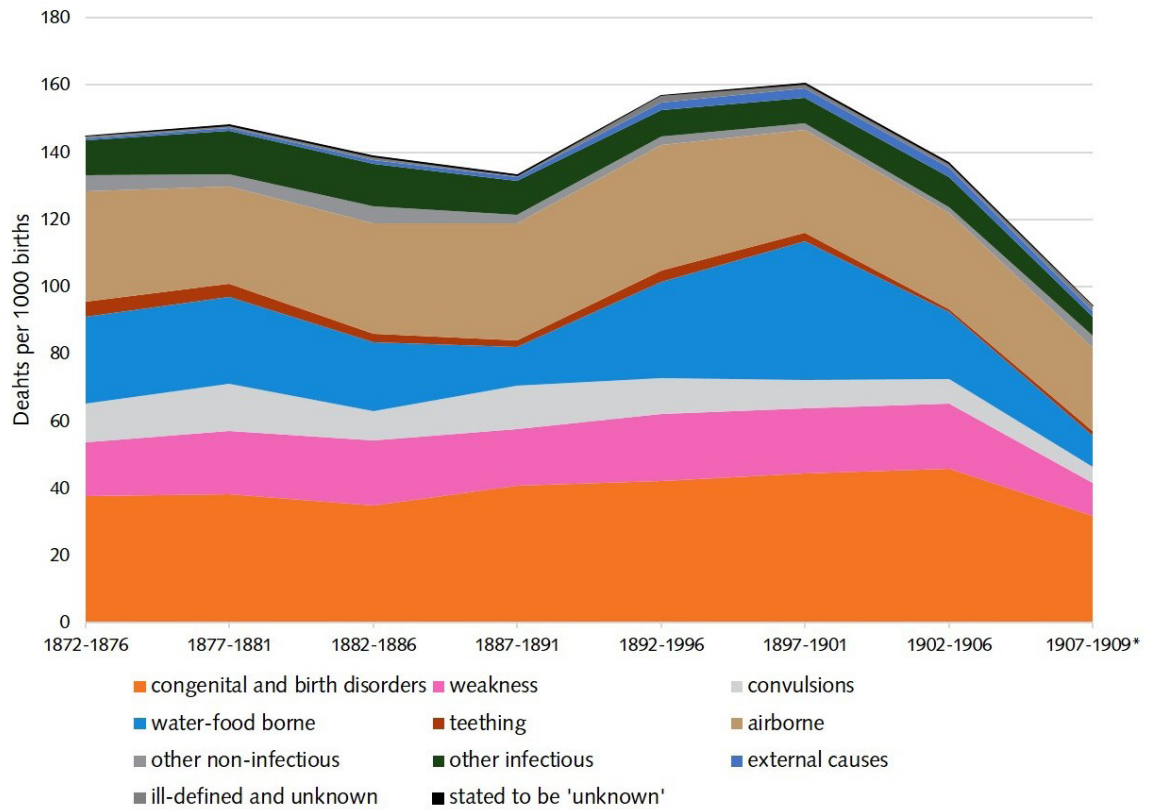
Figure 5 and Table 1 show that until the turn of the century the trends in IMR in Ipswich were dominated by fluctuations in 'water-food borne diseases', but that all causes contributed to the decline thereafter. The different causes were concentrated at different ages of infancy, and overall patterns in cause of death groups disguise more complex patterns in the neonatal and post-neonatal periods. We therefore leave our discussion of most causes of death for the age-specific sections of the paper.

21 From as early as the 1840s doctors in England and Wales were provided with printed Medical Certificates of Cause of Death forms on which to record their diagnoses of what had killed the person whose cause of death they were certifying. An early form and the instructions which accompanied it can be found in: Registrar-General of England and Wales (1846), p. 20; see www3.histpop.

22 Harris (2008, p. 180) quotes Anderton and Leonard (2004) as arguing that "literal causes of death offer a more adequate picture of disease as understood at the time".

23 Not every doctor listed causes in the expected order. There were 445 infant deaths in Ipswich between 1872 and 1909 which contained the cause of death 'measles'. In 415 cases (93%) 'measles' was listed first as the 'primary' cause. In 11 of the remaining cases another infectious disease — mainly whooping cough — was given as the primary cause. In a further eight cases a pre-existing condition such as TB, heart disease or debility was given as the primary cause, with measles proving too much for the little victim. In the remaining 10 cases (2.2%) the deceased had succumbed to pneumonia or bronchitis, presumably brought on by measles. The doctor should probably have listed measles as the primary cause of death in the latter cases. Taking COD1 causes as the underlying cause of death, the infant death rate from measles in Ipswich was 6.39 per 1,000 births in 1872–1909. The rate calculated by including all deaths mentioning measles for the same period is 6.85, but as just explained, not all cases would have had measles as their primary cause and the true rate lay somewhere between 6.39 and 6.85.

Figure 5 *Quinquennial IMRs by causal group, Ipswich 1872–1909**



* The final period includes only three years.

Source: As Figure 3.

Table 1 *Quinquennial infant mortality rates by causal group, Ipswich 1872–1909*

COD category	1872–1876		1877–1881		1882–1886		1887–1891		1892–1896		1897–1901		1902–1906		1907–1909*	
	IMR	N	IMR	N	IMR	N	IMR	N	IMR	N	IMR	N	IMR	N	IMR	N
congenital and birth disorders	37,7	291	38,2	311	35,0	303	40,9	351	42,1	370	44,4	405	46,0	438	31,7	173
weakness	16,1	124	18,8	153	19,2	166	16,7	143	19,9	175	19,3	176	19,2	183	10,1	55
convulsions	11,5	89	14,3	116	8,9	77	12,8	110	10,7	94	8,4	77	7,3	70	4,8	26
water-food borne	25,7	198	25,7	209	20,5	177	11,8	101	28,6	251	41,4	377	19,9	190	9,2	50
teething	4,5	35	3,8	31	2,4	21	1,9	16	3,4	30	2,5	23	0,9	9	1,3	7
airborne	32,8	253	29,1	237	32,9	285	34,7	298	37,5	329	30,4	277	28,6	273	24,7	135
other infectious	4,9	38	3,4	28	4,9	42	2,6	22	2,5	22	2,1	19	1,6	15	3,7	20
other non-infectious	10,2	79	13,0	106	12,8	111	10,0	86	7,9	69	7,5	68	9,0	86	5,5	30
external causes	0,6	5	1,0	8	1,2	10	1,2	10	2,1	18	3,0	27	2,6	25	1,8	10
ill-defined and unknown	0,5	4	0,6	5	0,9	8	0,3	3	2,1	18	1,3	12	1,5	14	1,5	8
stated to be 'unknown'	0,3	2	0,1	1	0,1	1	0,2	2	0,1	1	0,2	2	0,2	2	0,2	1
Total	144,9	1118	148,1	1205	138,8	1201	133,2	1142	156,8	1377	160,5	1463	137,0	1305	94,4	515

* The final period includes only three years.

Source: As Figure 3.

Before moving on, however, we discuss the role of changing reporting practices on trends in some of the cause of death groups. Did deaths from 'teething' and 'convulsions' decline because doctors were no longer reporting them, or no longer reporting them as the first cause of death on a death certificate? Both of these conditions are symptoms caused by the true underlying cause of death and, as such, represent deaths from that cause. For example, both contemporary doctors and historians have often interpreted infant deaths from 'teething' as really due to diarrhoea or to a synergistic complex of poor nutrition during weaning and an enteric infection known as 'weanling diarrhoea' (de Looper et al., 2019; Lewis, 1979; Sawchuck et al., 1985).

'Convulsions' has also been identified as a symptom of diarrhoea, related to dehydration, but could accompany the final stages of many fatal diseases (Kintner, 1986; Reid & Garrett, 2012; Williams, 1996). It is difficult to establish reasons for the declines in 'teething' and 'convulsions' from the figures in Table 1 as the numbers of deaths from these causes are relatively small. However, further insight can be gained from comparing the time trends, age-patterns and seasonality of these diseases with those of 'water-food borne diseases', points we will return to later.

The 'congenital and birth disorders' category was dominated by deaths from 'premature birth' and 'congenital debility'. Doctors may have become more confident in predicting mothers' delivery dates, thus allowing them to state that a birth was 'premature', but Galley (2021), for one, considers this very unlikely. By the early 1900s, "Suggestions to Medical Practitioners respecting Certificates of Causes of Death" were being issued by the Registrar General.²⁴ These indicated that 'premature birth' was an acceptable term for use on a death certificate, whereas 'debility' was an "Indefinite or Undesirable Term when used without further particulars" and the doctor was expected to supply the "disease causing this condition" (Collingridge, 1903, Appendix 5). In the early 1870s 'prematurity' was rarely given as a cause of death, but by the early 1900s it accounted for over 20 deaths per 1,000 births. The increased use of the term 'prematurity' could therefore simply have been the result of changing directives to doctors. Further study of such directives could cast a helpful light on registration and medical certification practices. There were further significant overlaps between the 'congenital and birth disorders' and 'weakness' categories, partly because the categorisation places 'congenital debility' and 'debility from birth' in the former, and 'debility' in the latter. ICD10h has separate codes for each of these terms because 'debility' was also used for debility associated with old age. The highest number of infant deaths from 'debility' occurred on day one of life, however, suggesting that when this cause was given for an infant death, it could well be counted in the 'congenital and birth disorders' category. While 'debility' was often explicitly stated to have been present 'from birth', deaths from 'weakness' occurred throughout the first year of life. Nevertheless, 'weakness' and 'congenital and birth disorders' could probably be combined when infant deaths are being considered.

Use of further terms in the 'weakness' category, such as 'atrophy', and 'marasmus', were concentrated at older ages within the first year of life. 'Marasmus' deaths did not appear in any number until the third week of life and were most numerous in the second, third, and fourth months of life, although numbers remained substantial across the rest of the first year. 'Atrophy' followed a broadly similar pattern to 'marasmus' but at a much lower number of cases. Both 'atrophy' and 'marasmus' are described in contemporary sources as forms of 'wasting' (Payne, 1884). Both could have been linked to infant feeding. Artificially fed infants were more likely to have died from diarrhoea, but were also at risk of wasting due to the lack of appropriate milk substitutes. Adulteration of milk powder with flour, chalk and other substances was not unknown, and some infants were fed solid food they could not digest, leading to malnutrition (Reid, 2006). Ironically, it is also possible that children who were already failing to thrive when breastfed might have been moved to artificial food in an attempt to improve their health.

3.1 INDIVIDUAL CAUSES WITHIN CAUSAL GROUPS

3.1.1 'AIRBORNE DISEASES'

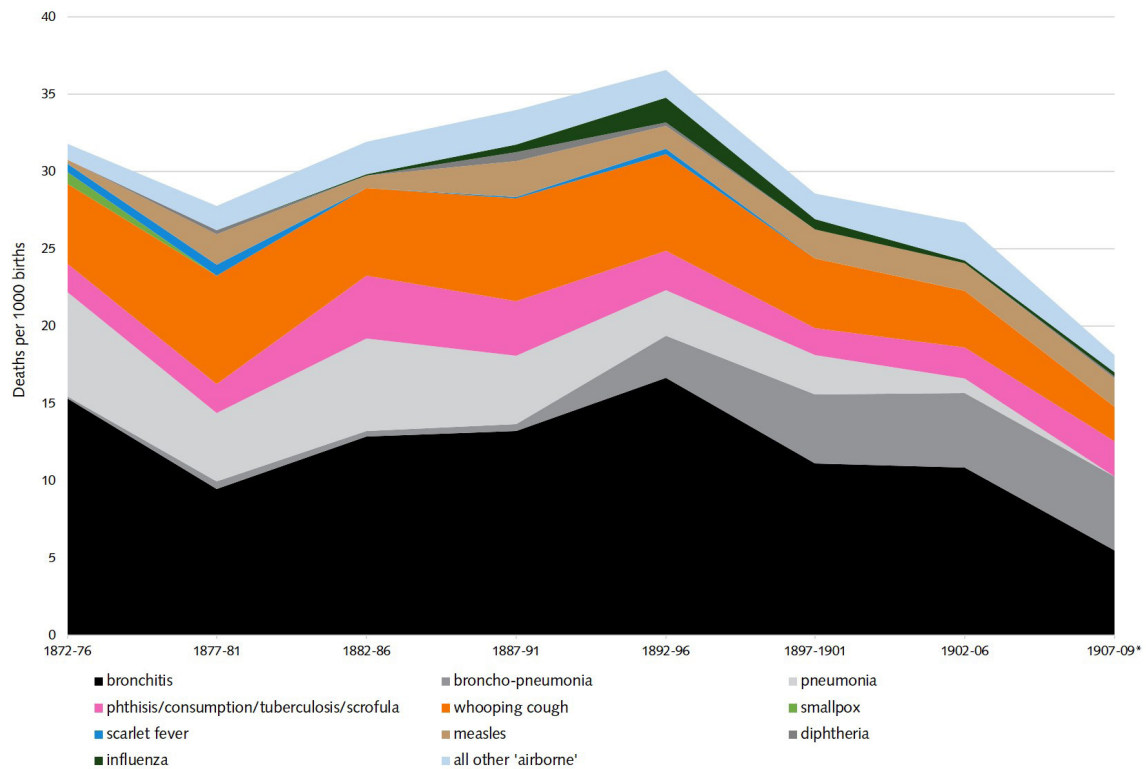
Figure 6 includes all deaths from the 'airborne disease' category in the first year of life, most of which occurred after the first four weeks. The 'airborne' category is made up of what may be thought of as three major groups of diseases and a catchall 'other airborne causes' (light blue in Figure 6). The largest group comprises 'bronchitis' (black), 'pneumonia' (light grey) and 'broncho-pneumonia' (dark grey). There were virtually no cases of 'broncho-pneumonia' recorded in Ipswich until 1892–1896, but thereafter

24 For example, "Suggestions" from 1902, published by the General Register Office, London, can be viewed at the National Records for Scotland, ref. GRO5_814, p. 9.

this cause gradually replaced 'pneumonia'; almost certainly a reflection of changing nomenclature, reminding us to be vigilant for such changes as they might affect our perception of the history of certain conditions. The second group is made up of 'tubercular diseases' (pink), variously described as 'phthisis', 'consumption', 'tuberculosis' (or TB) and 'scrofula'. This was not a major killer of infants. The third major group comprises 'infectious diseases' and includes 'smallpox' (bright green), only visible in the early 1870s, showing the effectiveness of the Vaccination Acts described above, 'scarlet fever' (bright blue), 'measles' (light brown), 'diphtheria' (white), 'influenza' (dark green) and, accounting for the most deaths amongst infants in this group, 'whooping cough' (orange). Woods and Shelton (1997, pp. 76–78), among others, have highlighted the importance of whooping cough as an infectious disease affecting infants, and this is reflected clearly in Figure 6. Why infants might be more susceptible to this disease than to other infectious diseases of childhood merits further investigation. It is also relevant that over 30% of 'whooping cough' deaths registered in Ipswich had 'convulsions' as a contributing cause, so the decline in whooping cough deaths may have helped to reduce those from convulsions.

The decline in 'airborne diseases' after 1892–1896 was driven principally by 'bronchitis' and 'whooping cough'. Figure 6 suggests that the analysis of infant mortality might be informed by a separation of the 'airborne diseases' causal group into separate 'pneumonia-bronchitis' and 'childhood infectious diseases' categories, as is sometimes done in other analyses (e.g. Reid, 2002).

Figure 6 *Quinquennial IMRs from diseases within the 'airborne diseases' category, Ipswich 1872–1909*



* Final period is only three years long.

Source: As Figure 3.

3.1.2 WATER-FOOD BORNE DISEASES

Infant deaths caused by diseases in the water-food borne category also occurred predominantly amongst those aged over one month. Between 1872 and 1886 85% of all water-food borne infant deaths in Ipswich were ascribed to 'diarrhoea', but after 1886 an increasing proportion of deaths in this category were attributed to 'gastro-enteritis' or 'infantile diarrhoea'. Between 1897 and 1901 there were 205 infant deaths from diarrhoea, but 104 from 'gastro-enteritis' — compared to just 18 deaths from this cause in the previous 15 years — and 23 from 'infantile diarrhoea'. From 1902 to 1909 there were 97 deaths from 'diarrhoea' in the town, but 56 deaths from 'infective colitis', 36 deaths from 'enteritis' and 26 from 'gastroenteritis'. Thus, in 1897–1901 only 57.4% of water-food borne diseases were attributed to 'diarrhoea', by 1902–1906 this had fallen to 45.3% and by 1907–1909 to just 22%. The Registrar General's Office had been waging a war with doctors for some time, instructing them not to return 'diarrhoea' as a cause of death as it was symptom of other conditions, not a single disease. For instance, in the Decennial Supplement to the Registrar General's *Sixty-Fifth Annual Report* (1907, p. lxxxiii) covering the decade 1891–1900, the then superintendent of statistics, John Tatham, complained that diarrhoea was 'unsatisfactory' as a the cause of death category. Nevertheless, he also noted that, "...it is satisfactory to note that many of the deaths which formerly would have been attributed to 'diarrhoea' simply ... are now coming to be referred very generally to 'infective enteritis' or its authorised equivalent 'epidemic diarrhoea'" (p. lxxxv). When he considered diarrhoeal diseases amongst infants in his report, Tatham included all forms of diarrhoea, enteritis, gastro-enteritis, gastritis and gastro-intestinal catarrh in his calculations; rather more conditions than were included in the 'diarrhoeal diseases' category elsewhere in the decennial supplement. It thus appears that by the beginning of the 20th century, doctors were at last beginning to follow the practice advised by the Registrar General's Office and the proportion of deaths certified as being due to 'diarrhoea' dwindled. Deaths from almost all 'water-food borne diseases' were falling after 1901, both in Ipswich and more generally, but it would appear that 'diarrhoea' may have declined particularly rapidly as a result of changing certification practices.

4 NEONATAL AND POST-NEONATAL MORTALITY

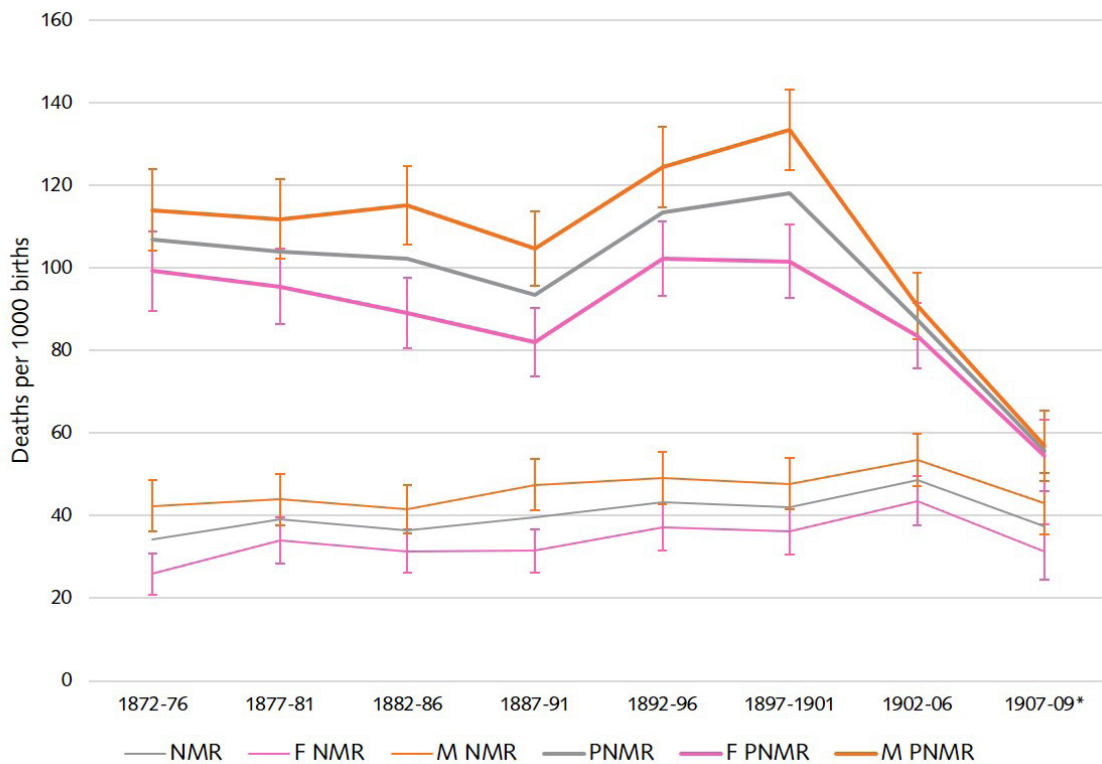
The figures above refer to all infants reported as dying aged up to '11 months and 31 days', but the picture is rather different if neonatal deaths and post-neonatal deaths are viewed separately.

In Ipswich, between 1872–1876 and 1902–1906, the neonatal mortality rate (NMR) rose by just over one third, from 35.9 deaths to 48.7 deaths per 1,000 live births (see Table 2). Over the same period the post-neonatal mortality rate (PNMR) fell by just over a sixth, despite a marked rise across the 1890s. Both the NMR and PNMR fell between 1902–1906 and 1907–1909, although the decline in the PNMR was the larger by some margin. Neonatal mortality thus became a much larger component of overall infant mortality during our period, making up 40% of the overall IMR in 1907–1909 compared to only 20.7% in 1872–1876.

Figure 7 indicates that over time there was little change in the rates and differentials between the sexes in the first 28 days of life, with male rates being consistently (and mostly significantly) higher than females. There was also a male disadvantage in PNMR, which was particularly marked in the 1880s and 1890s.

The combination of the apparent disappearance of the differential in PNMR between the sexes and the decline in PNMR across the first decade of the 20th century in Ipswich is interesting. England and Wales did not start to publish national mortality rates for the first month of life until 1906, but for 1906–1910 the ratio of female to male infant mortality was 0.77 for neonatal mortality and 0.84 for post-neonatal mortality, indicating that males retained a post-neonatal disadvantage in the national population ([Registrar-General \(1919\)](#), 1901–1910, Tables 12 and 13). In the English county of Derbyshire between 1917 and 1922, males had a 25% higher chance of dying in the post-neonatal period, a disadvantage which was particularly prominent in the risk of death from bronchitis and pneumonia ([Reid, 2002](#), pp. 156, 159). It therefore seems that the reduction in the sex difference in Ipswich was not a national phenomenon, although the decline in PNMR undoubtedly was ([Woods et al., 1988](#)), making the situation in Ipswich worthy of further investigation.

Figure 7 *Quinquennial rates of neonatal (NMR) and post-neonatal (PNMR) mortality for males (M) and females (F), Ipswich 1872–1909*



Note: The error bars show 95% confidence intervals.

* The final period includes only three years.

Source: As Figure 3.

Table 2 *Quinquennial rates* of IMR, NMR and PNMR (where sex of the child is known)**, with % of infant deaths which were neonatal and post-neonatal, Ipswich 1872–1909*

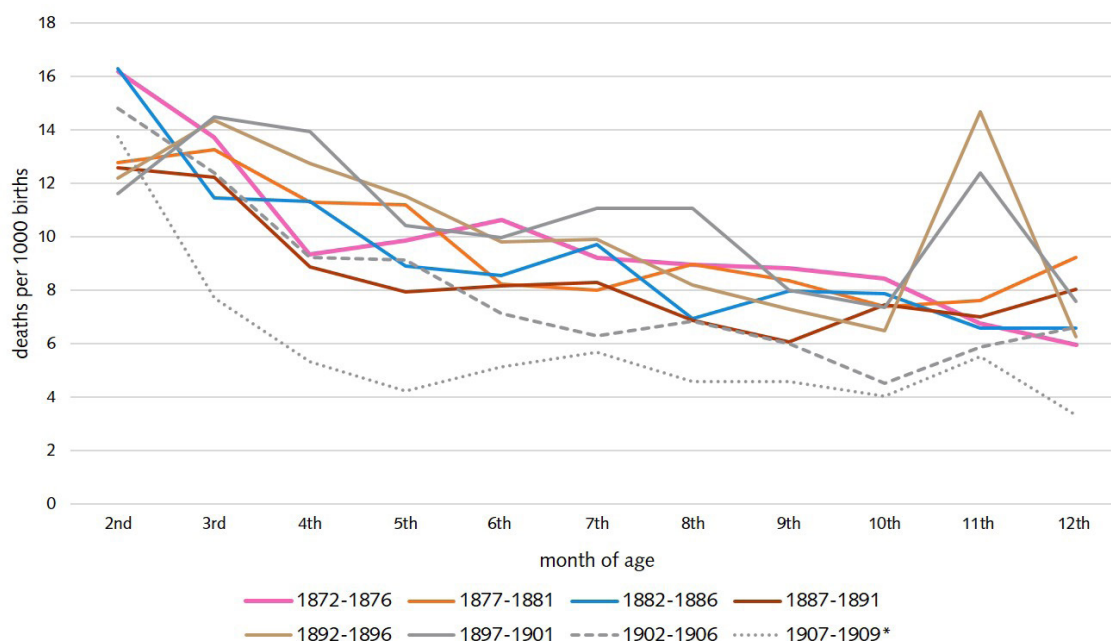
Quinquennium	IMR	NMR	PNMR	% of infant deaths	
				neonatal	post-neonatal
1872–1876	142.2	35.9	106.3	25.3	74.7
1877–1881	144.0	39.7	104.3	27.6	72.4
1882–1886	138.8	36.6	102.2	26.4	73.6
1887–1891	133.2	39.6	93.5	29.8	70.2
1892–1896	156.7	43.2	113.6	27.5	72.5
1897–1901	160.2	42.1	118.1	26.3	73.7
1901–1906	136.3	48.7	87.6	35.7	64.3
1907–1909	93.7	37.8	55.9	40.0	60.0

* The final period includes only three years.

** In the 1872–1876 and 1877–1881 quinquennia there were a small number of deaths where the sex of the child was not recorded, in most cases when the child died very quickly after birth. For this reason the IMRs in this table differ slightly from those in Table 1.

Source: As Figure 3.

Figure 8 *Post-neonatal mortality rates, for babies in the nth month of life, Ipswich: quinquennia 1872–1909*



* The final period includes only three years.

Source: As Figure 3.

The decline in infant mortality in Ipswich was therefore driven mainly by deaths in the post-neonatal period, although the initial part of this decline may have been simply a return to 1880s levels, before an additional, new decline set in from 1906 onwards. While seeking reasons for the decline post-1899, we must not lose sight of the fact that PNMRs in Ipswich, as in England and Wales as a whole, were already relatively low in the 1880s; the 1890s were a particularly lethal decade for infants, unrepresentative of the previous decade.

Figure 8 shows post-neonatal mortality rates by month of life for both sexes combined in successive quinquennia from 1871–1876 to 1907–1909. It is noticeable that the decline in PNMR between 1897–1901 and 1902–1906 was not concentrated in any particular month of life. Indeed the rates for the latter quinquennium lie very close to those of 1887–1891, reinforcing the impression that the elevated rates of the 1890s were an aberration. In contrast, the decline between 1902–1906 and 1907–1909 was particularly marked in the third, fourth, and fifth months of life.

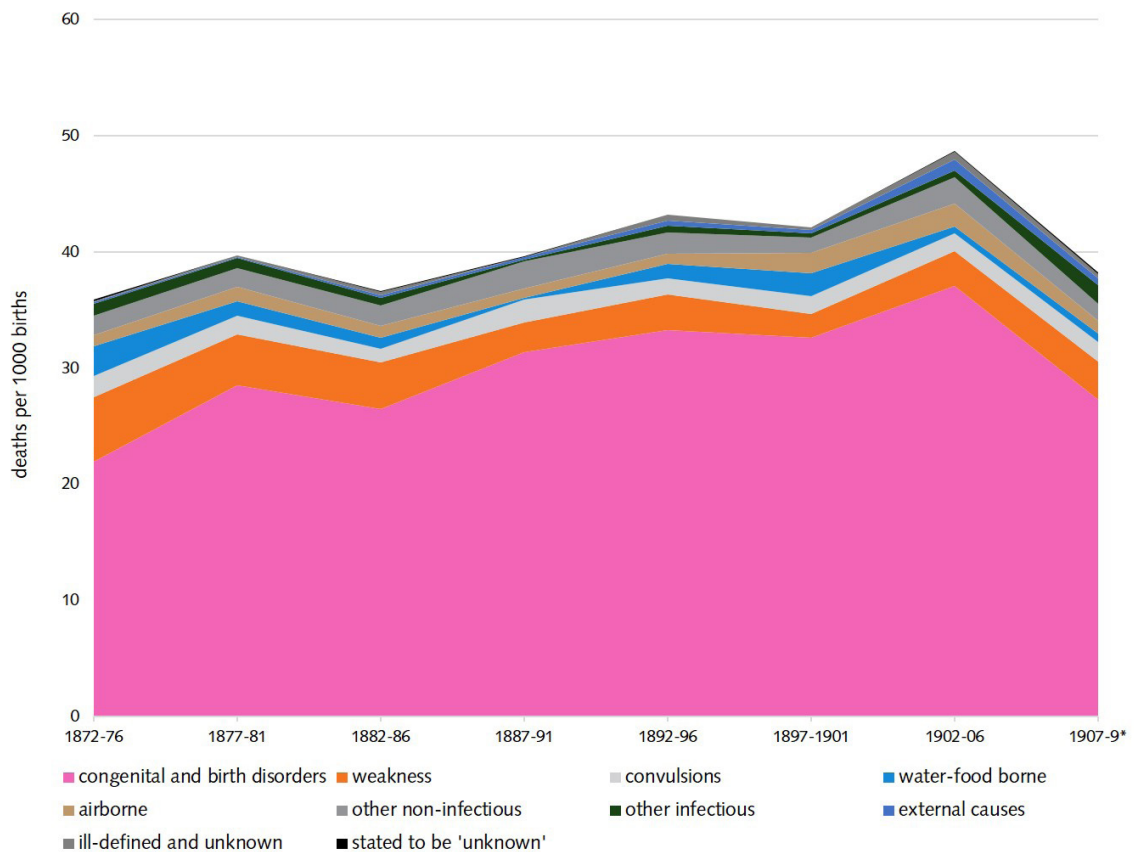
5 NMRs AND PNMRs BY CAUSE OF DEATH

5.1 NEONATES

As Figure 9 shows, 'congenital and birth disorders' dominated deaths amongst Ipswich's neonates. The risk of death from this causal group rose from just under 22 to 37 deaths per 1,000 births between 1872–1876 and 1902–1906. There was then a substantial fall in the following three years, 1907–1909. 'Weakness', 'convulsions', and 'water-food borne diseases' also took a toll on infants aged under four weeks, but the numbers were relatively small, so any fluctuations in the figures could just be random (see Table 3).

The increase in mortality from 'congenital and birth disorders' could be interpreted as showing that the health of, and the standard of care given to, pregnant women and labouring mothers were declining, but it could also indicate an improvement in these factors, if more pregnancies were surviving to term. Sadly, as noted previously, stillbirths were not registered in Ipswich or England and Wales at this period, making it impossible to determine which of these scenarios was true.

Figure 9 *Quinquennial neonatal mortality rates (NMRs) by cause of death category, Ipswich 1872–1909*



* The final period includes only three years.

Source: As Figure 3.

Table 3 *Quinquennial neonatal mortality rates by SHIP infant COD category, Ipswich 1872–1909*

CoD category	1872–1876		1877–1881		1882–1886		1887–1891		1892–1896		1897–1901		1902–1906		1907–1909*		N of deaths < 28 days 1872–1909
	NMR	N	NMR	N	NMR	N	NMR	N	NMR	N	NMR	N	NMR	N	NMR	N	
congenital and birth disorders	21,9	14	28,5	232	26,5	229	31,4	269	33,3	292	32,6	297	37,0	353	27,3	146	1987
weakness	5,6	43	4,4	36	4,0	35	2,6	22	3,1	27	2,1	19	3,0	29	3,3	18	229
convulsions	1,8	14	1,6	13	1,2	10	2,0	17	1,4	12	1,5	14	1,5	14	1,6	9	103
water-food borne	2,6	20	1,2	10	0,9	8	0,1	1	1,3	11	2,0	18	0,6	6	0,7	4	78
airborne	0,9	7	1,2	10	1,0	9	0,8	7	0,9	8	1,8	16	2,0	19	1,1	6	82
other infectious	1,0	8	0,9	7	0,7	6	0,1	1	0,6	5	0,3	3	0,6	6	1,6	9	45
other non-infectious	1,7	13	1,6	13	1,7	15	2,3	20	1,8	16	1,3	12	2,2	21	1,5	8	118
external causes	0,1	1	0,1	1	0,2	2	0,2	2	0,5	4	0,3	3	0,9	9	0,5	3	25
ill-defined and unknown	0,1	1	0,1	1	0,2	2	0,0	0	0,5	4	0,2	2	0,6	6	0,4	2	18
stated to be 'unknown'	0,1	1	0,0	0	0,1	1	0,1	1	0,0	0	0,0	0	0,1	1	0,2	1	5
total NNM rate	35,9	277	39,7	323	36,6	317	39,6	340	43,2	379	42,1	384	48,7	464	37,8	206	2690
% infant deaths to neonates		25,3		27,6		26,4		29,8		27,5		26,3		35,7		40,0	28,6

* The final period includes only three years.

Source: As Figure 3.

These causes, and weakness, were most directly influenced by standards of maternal health and net nutrition, and by obstetric practices (Reid, 2001a, 2001b). Woods (2009) proposed that reductions in infectious disease amongst mothers may have led to declining rates of perinatal mortality. The Midwives Act in 1902 introduced the compulsory training, registration and monitoring of midwives for the first time in England and Wales, and improving obstetric standards are likely to have had a beneficial impact on infant mortality from prematurity, birth trauma and other conditions. Other research has indicated that in the early 20th century infants delivered by trained midwives were less likely to be stillborn or to die in the first month of life (Reid, 2012). The early years of the 20th century also saw the birth of the British maternal and child welfare movement (Dwork, 1987). This was galvanised by the publication, in 1904, of the Report of the Inter-departmental Committee on Physical Deterioration, which was itself inspired by the shocking rates of rejection of potential recruits from all social classes amongst those attempting to sign up to serve in the Boer War. The mother and child welfare movement included a variety of measures to improve the health and survival of the youngest children, including ante-natal care, schools for mothers, infant welfare clinics, and health visiting. However until the Maternity and Child Welfare Act of 1918, most of these were private philanthropic efforts or patchily enacted due to permissive legislation, bound together in a 'mixed economy of welfare' (Al-Gailani, 2020).

5.2 POST-NEONATES

Figure 10 and Table 4 present PNMRs by cause of death group. It is clear that trends in overall IMR were dominated by PNMR, and the largest causal groups were airborne and water-food borne diseases.

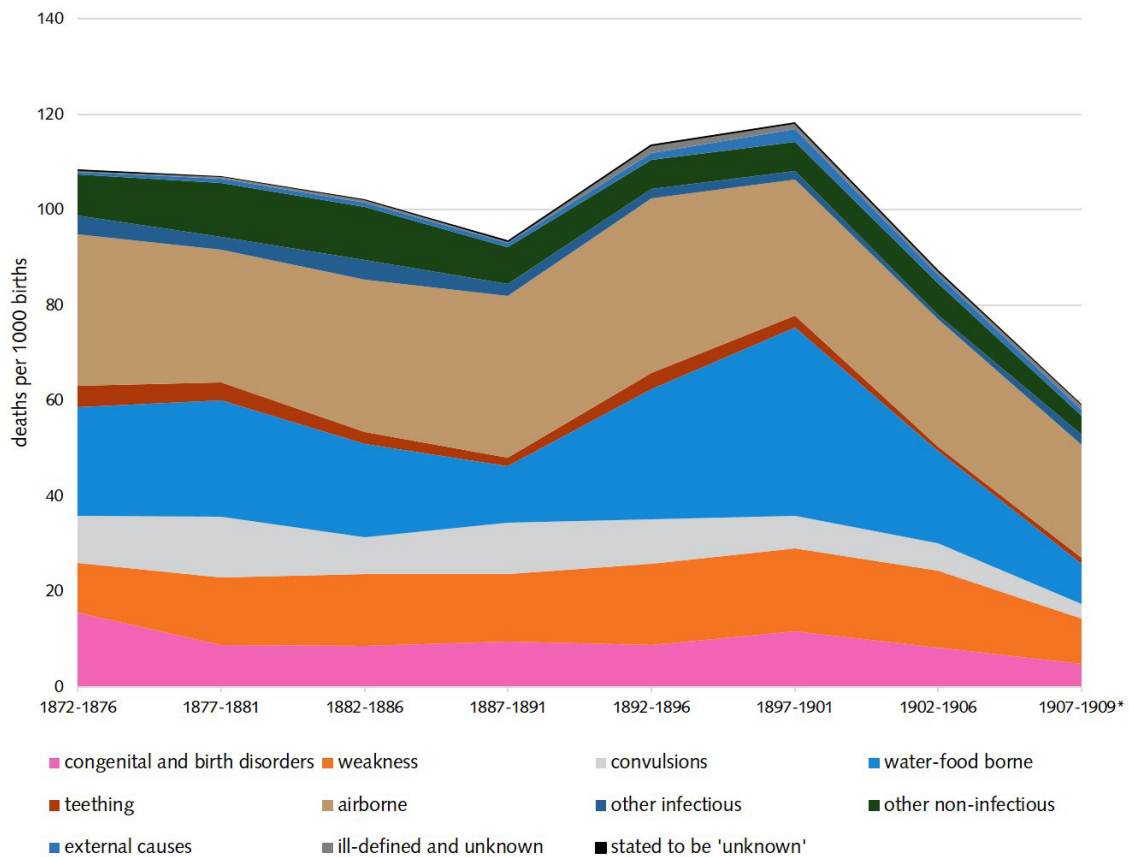
Most causes of post-neonatal mortality saw little change in the 1870s and the 1880s, but in the early 1890s 'airborne diseases' began a slow downward movement, and from the late 1890s 'congenital and birth disorders' also began to decline. After 1902–1906 mortality from almost all causes saw a reduction. The 'water-food borne diseases' was the only group of causes to see a major increase during the 1890s, confirming Ipswich suffered from an 'urban-sanitary-diarrhoeal' effect, and it was this rise that made the subsequent decline of this category, from a peak in 1897–1901, look so impressive. If the peak in this group had not occurred then the change in PNMRs between 1887–1891 (11.7 deaths per 1,000) and 1907–1909 (8.3) would have been relatively small (see Table 4), and the decline in PNMRs, both from 'water-food borne diseases' and overall, would appear much more gradual and longer-term than their sharp downturns after 1901 suggest. Deaths from 'water-food borne diseases' may have begun to decline in the 1880s, thanks to improvements in sanitation, hygiene and water supply (Bell & Millward, 1998; McKeown, 1976; McKeown & Record, 1962; Szreter, 1988, 1997). Hinde and Harris (2019) have demonstrated that loans for public works to urban sanitary authorities exhibited a first peak in the late 1870s and a second, larger peak in the 1890s. The town council in Ipswich negotiated loans amounting to some £44, 500 in the 1870s, £51,000 in the 1880s, and £280,000 in the 1890s in order to fund improvements in the town's water and sanitation systems.²⁵ Given that such loans would not have translated into immediate improvements in mortality, as the work would have to have been organised and completed, the chronology of this expenditure may go some way to explain the two phases of water-food borne post-neonatal mortality in Ipswich.

The initial reduction was overturned, however, by a set of circumstances specific to the 1890s. First, a series of hot, dry summers reduced the length of the life-cycle of the housefly, the main vector responsible for the spread of diarrhoea (Morgan, 2002). Secondly, it is likely that the increased use of horse drawn transport in Ipswich, coupled with increased stabling and additional waste in the streets, would also have multiplied habitats for flies. Finally, it has been argued that although improvements in urban water and sanitation provision may not have kept up with urban growth, without these improvements the mortality increase in the 1890s might have been even larger (Hinde & Harris, 2019; Szreter & Mooney, 1998; Torres et al., 2019; Williams & Galley, 1995; Woods, 1985). The 'urban-sanitary-diarrhoeal' effect mainly affected post-neonatal infants because most infants in this period were exclusively breastfed during their first months of life, and this almost certainly protected infants in the neonatal period (Fildes, 1998; Reid, 2002).²⁶ Temperatures in the 1900s were more moderate and sanitary systems in the South-East of England were apparently able to cope, at least until 1911, when another hot summer strained them once more (Galley, 2021).

25 We are grateful to Bernard Harris (Harris & Hinde, 2019; Hinde & Harris, 2019) who provided us with the information from which these figures were calculated.

26 We have not been able to ascertain breastfeeding rates in Ipswich itself, but the median percentage of infants who were exclusively breastfed during the first month of life in 23 English towns was 83% (Fildes, 1998, p. 255).

Figure 10 *Quinquennial post-neonatal mortality rates (PNMRs) by cause of death category, Ipswich 1872–1909*



* The final period includes only three years.

Source: As Figure 3.

Table 4 *Quinquennial post-neonatal mortality rates by SHiP infant COD category, Ipswich 1872–1909*

	1872–1876		1877–1881		1882–1886		1887–1891		1892–1896		1897–1901		1902–1906		1907–1909*	
	PNMR	N	PNMR	N	PNMR	N	PNMR	N	PNMR	N	PNMR	N	PNMR	N	PNMR	N
congenital and birth disorders	15,7	121	9,0	73	8,6	74	9,6	82	8,9	78	11,7	107	8,4	80	4,9	27
weakness	10,4	80	14,1	115	15,1	131	14,1	121	16,9	148	17,2	157	16,2	154	6,7	37
convulsions	8,3	64	12,3	100	7,7	67	10,8	93	9,3	82	6,9	63	5,8	55	3,1	17
water-food borne	22,7	175	23,8	194	19,5	169	11,7	100	27,3	240	39,4	359	19,3	184	8,3	46
teething	4,4	34	3,8	31	2,4	21	1,9	16	3,4	30	2,5	23	0,9	9	1,3	7
airborne	31,6	244	26,2	213	31,9	276	33,9	291	36,6	321	28,5	260	26,7	254	23,4	129
other infectious	3,9	30	2,5	20	4,2	36	2,4	21	1,9	17	1,8	16	0,9	9	2,0	11
other non-infectious	8,4	65	11,2	91	11,1	96	7,7	66	6,0	53	6,0	55	6,8	65	4,0	22
external causes	0,4	3	0,9	7	0,9	8	0,9	8	1,6	14	2,6	24	1,7	16	1,3	7
ill-defined and unknown	0,4	3	0,5	4	0,7	6	0,3	3	1,5	13	1,1	10	0,7	7	1,1	6
stated to be 'unknown'	0,1	1	0,1	1	0,0	0	0,1	1	0,1	1	0,2	2	0,1	1	0,0	0
total PNMR rate	106,3	820	104,3	849	102,2	884	93,5	802	113,6	997	118,1	1076	87,6	834	55,9	309
% infant deaths to post-neonates		74,7		72,4		73,6		70,2		72,5		73,7		64,3		60,0

* The final period includes only three years.

Source: As Figure 3.

Table 5 *NMRs, PNMRs and numbers of deaths of males and females, Ipswich 1872–1909*

	female NMR		male NMR		female PNMR		male PNMR	
	NMR	N	NMR	N	PNMR	N	PNMR	N
congenital and birth disorders	25,1	810	34,5	1162	9,4	303	10,1	339
weakness	3,0	96	3,9	131	12,5	403	16,0	540
convulsions	1,3	43	1,8	60	7,7	249	8,5	285
water-food borne	0,9	29	1,4	48	21,2	685	23,2	782
teething					2,1	67	3,1	104
airborne	1,3	41	1,2	41	26,9	870	33,2	1118
other infectious	0,7	22	0,7	23	1,8	58	3,0	102
other non-infectious	1,4	44	2,2	74	6,7	215	8,8	298
external causes	0,3	10	0,4	15	1,4	44	1,3	43
ill-defined and unknown	0,2	8	0,3	10	0,8	25	0,8	27
stated to be 'unknown'	0,1	4	0,0	1	0,1	2	0,1	5
total	34,3	1107	46,4	1565	90,4	2921	108,5	3658

Source: As Figure 3.

Returning briefly to the marked decline in mortality between 1902–1906 and 1907–1909 amongst infants in their third, fourth and fifth months of life (see Table 2), the largest element of this was a decline in deaths from 'water-food borne diseases', although deaths from 'convulsions', 'congenital and birth disorders' and 'weakness' also saw marked declines,²⁷ partially offset by an increase in deaths from 'airborne diseases'. Mothers would have begun to introduce solid foods to infants in this age group, so hygiene practices, or recommendations about the best 'follow-on' foodstuffs, may have undergone a marked shift. Alternatively, breastfeeding patterns may have changed somewhat at the beginning of the 20th century. If more infants were being breastfed for longer, or not being introduced to solid food until later, this might help explain the decline in mortality in months three to five. The year 1907 was also the year of the Notification of Births Act, which encouraged local authorities to establish a team of health visitors to visit new mothers and deliver advice on feeding and hygiene among other factors (Dwork, 1987; Reid, 2001b). It should be noted that male and female PNMRs fell at the same rate at each month of life between 1902–1906 and 1907–1909; so whatever was causing survival rates from month three to month five to improve between these dates affected both sexes to the same degree.

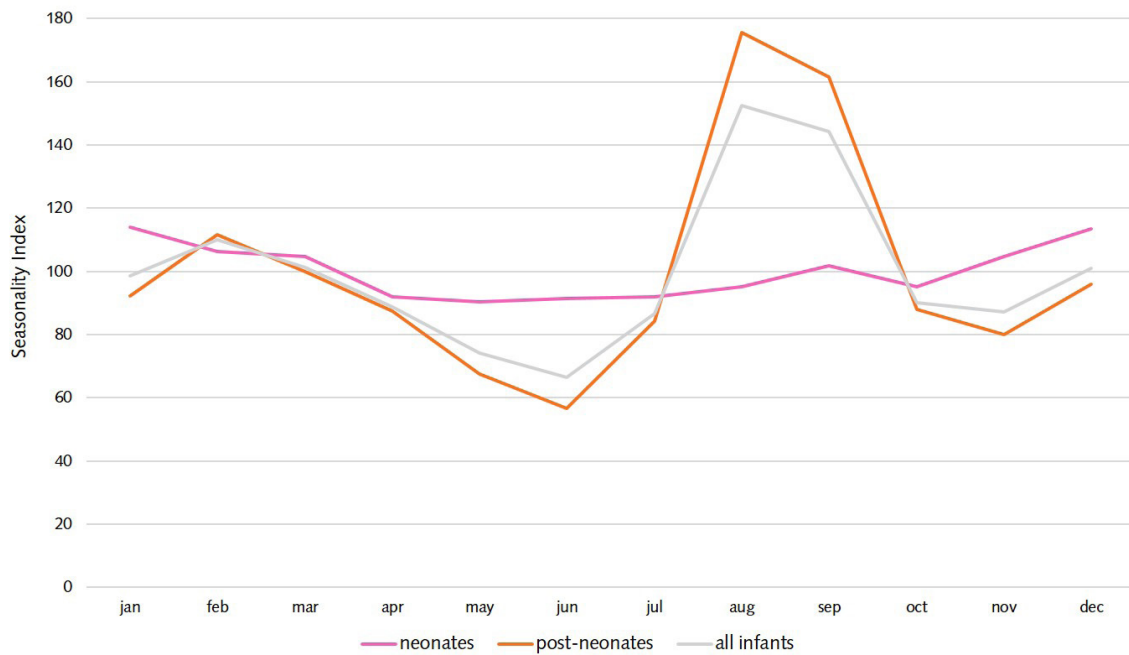
In line with the wider literature, Table 5 emphasises that across our period, male infants suffered higher rates of NMR and PNMR from almost every cause. In line with other evidence, compared to females, males were particularly susceptible to 'congenital and birth disorders' in the neonatal period and 'airborne diseases' and 'weakness' in the post-neonatal period, possibly as a consequence of their poor condition as neonates (Reid 2001a, 2002). The relative susceptibility of the sexes to particular categories of disease could, of course, vary over time.

6 SEASONALITY

When we calculate seasonality indices for neonates and post-neonates, as shown in Figure 11, it becomes clear that neonatal deaths in Ipswich were distributed relatively evenly across the year, but post-neonatal deaths in our study period had a marked August–September peak with compensating troughs in May–July and November.

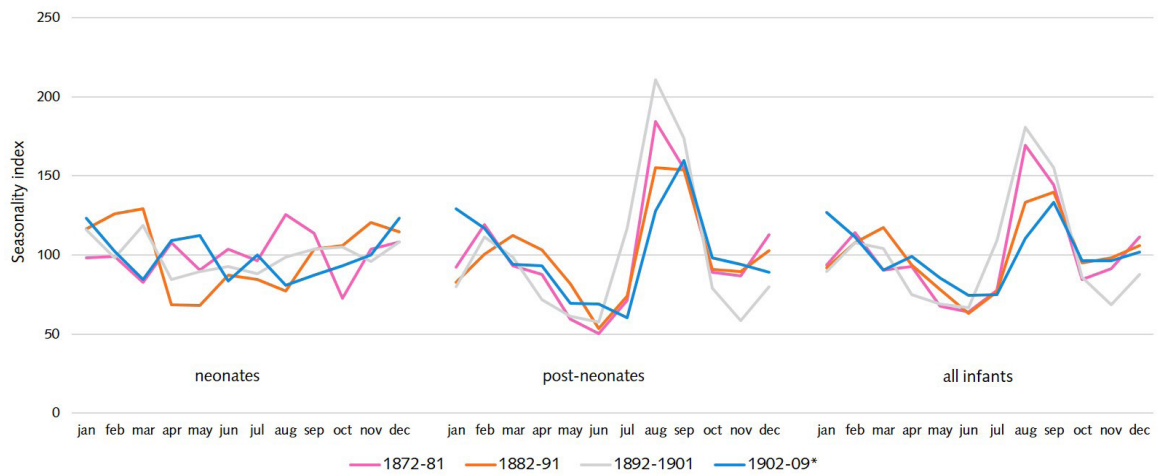
²⁷ Water-food borne deaths in the third, fourth and fifth months of life fell from 7.5 per 1000 births in 1902–1906 to 2.6 in 1907–1909. The equivalent figures for congenital and birth disorders 1.4 and 0.9; for convulsions, 2.5 and 0.5; and for weakness, 6.2 and 2.2. Deaths from airborne diseases rose from 5.0 to 5.5 per 1000 births.

Figure 11 *Seasonality indices for deaths amongst infants, neonates and post-neonates, Ipswich 1872–1909*



Source: As Figure 3.

Figure 12 *Seasonality indices for deaths amongst infants, neonates and post-neonates; by decade, Ipswich 1872–1909*



Note: Decades run from years ending 02 to years ending 01; the final period, 1902–1909 contains only eight years.

Source: As Figure 3.

As there were approximately 2.5 times more post-neonatal deaths (6,636) than neonatal (2,690), the overall IMR also shows a characteristic August–September peak. The smaller number of neonatal deaths means that when we consider seasonality by decade in Figure 12, the neonatal indices are much more 'noisy'.

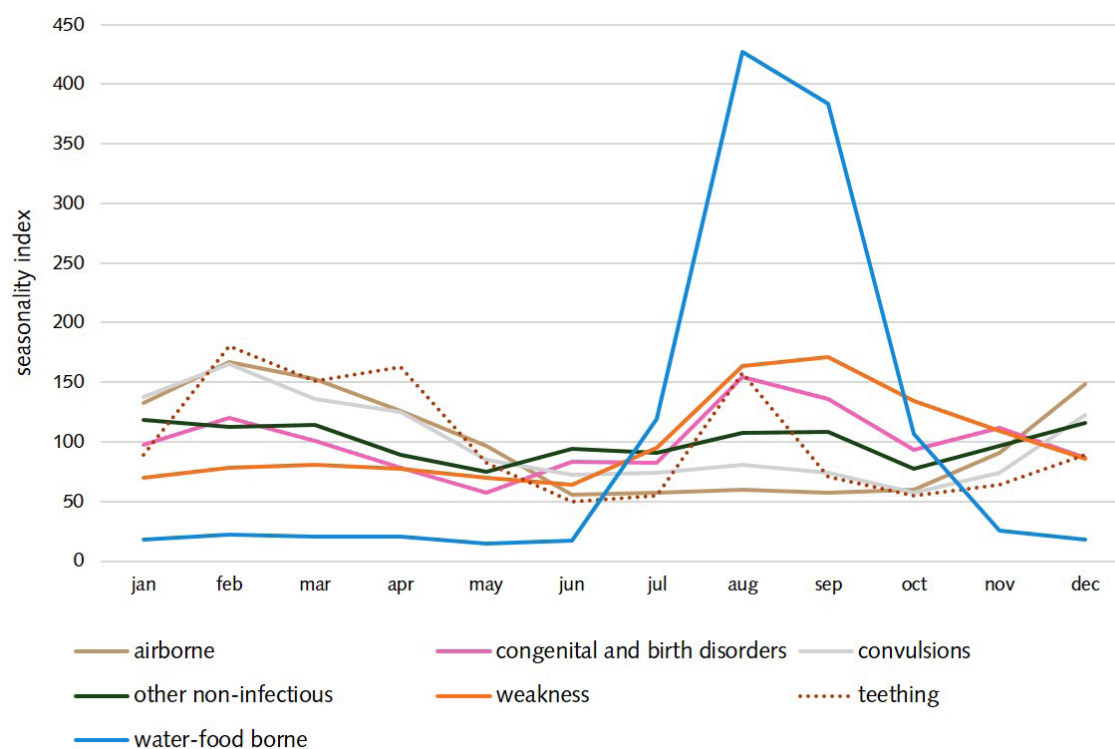
It is clear, nevertheless, that there was no consistent seasonal pattern to neonatal deaths. The August–September peak in post-neonatal and overall infant deaths is evident in every decade, but the level changed markedly; the peak in the 1890s was not only higher, but began earlier in the year with the rates starting to climb in July rather than August.

Figure 13 graphs the seasonality indices for causes of death with over 600 cases across the 1872–1909 period, plus 'teething', of which there were only 172 cases. As might be expected, 'congenital and

birth disorders' and 'other non-infectious diseases' show very little seasonal pattern, while 'airborne diseases' had a distinct winter peak and a long trough from June to October. 'Water-food borne diseases' had an extremely concentrated seasonal peak with hardly any cases from November to June, but large numbers of deaths from July to October. The term 'summer diarrhoea' is often encountered in the literature, and in Ipswich very few deaths from 'water-food borne' diseases occurred at any other time of the year, although the term 'summer diarrhoea' only appears as a cause of death on seven death certificates. It has been suggested that because of the association with summer, doctors may not have certified diarrhoea as a 'primary' cause of death outside the summer months,²⁸ intensifying the seasonality of deaths from 'water-food borne diseases'.

Perhaps surprisingly, given the findings of previous studies (such as Kintner, 1986; Reid & Garrett, 2012; Williams, 1996), 'convulsions' did not show a summer peak, along with the 'water-food borne diseases', but followed the seasonal pattern of 'airborne diseases' very closely. 'Teething' too appears to have been more prevalent in winter, albeit with a suggestion of a small rise in cases in August, when deaths from 'water-food borne diseases' were at their height. It looks as though the association between teething and diarrhoea may also be suspect, at least in Ipswich. The close associations between 'teething' and 'convulsions' and between 'convulsions' and 'whooping cough' seem to be borne out by the seasonality patterns shown. 'Weakness' was the only cause in Figure 13 to have a seasonal pattern akin to that of the 'water-food borne diseases', suggesting that 'weak' children were more likely to succumb to gastro-intestinal problems, although it possible that they had become debilitated because of an initial stomach or intestinal problem.

Figure 13 Seasonality indices for selected categories of infant mortality, Ipswich 1872–1909

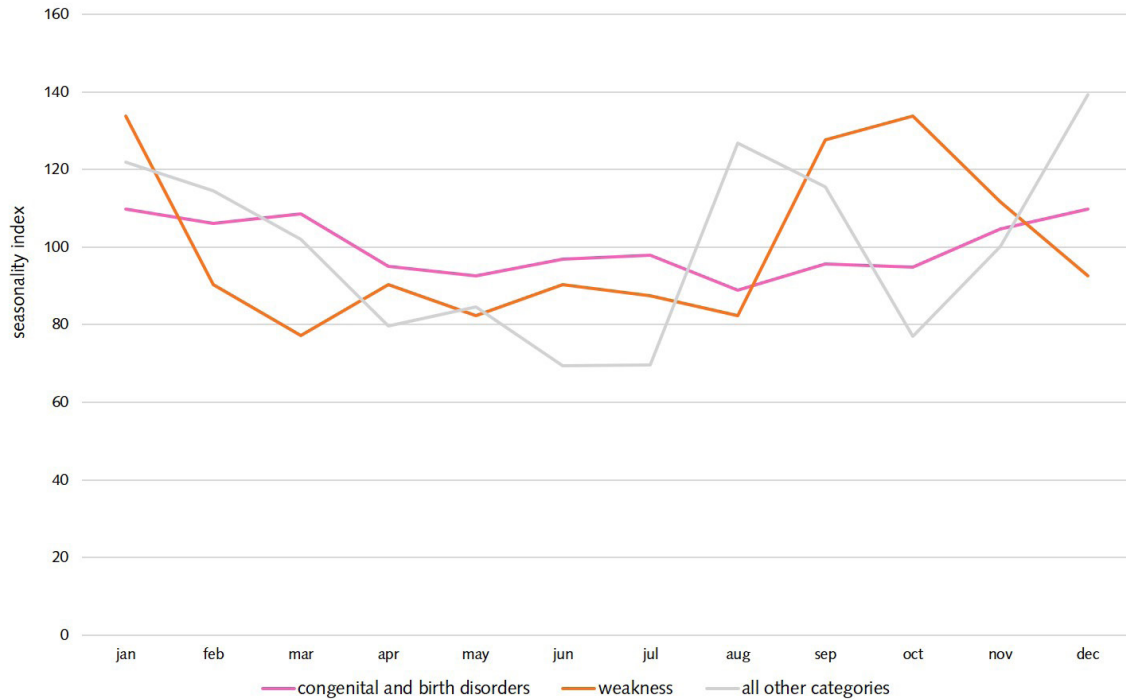


Note: Only categories with more than 600 cases 1872–1909 are shown, apart from teething which had 172 cases.

Source: As Figure 3.

28 We are grateful to Chris Galley for pointing us to this possibility. By the early 1900s doctors were being increasingly encouraged only to certify deaths from 'epidemic diarrhoea' and to see 'non-epidemic diarrhoea' as a symptom rather than a 'primary cause' of death ("Suggestions to Medical Practitioners respecting Certificates of Causes of Death" (dated October 1911). TNA RG 41/1 GRO instructional circulars regarding the registration of births, deaths and marriages, 1908 to 1933). This may have meant that the seasonal pattern was uppermost in their mind. In Ipswich, 'epidemic diarrhoea' is not seen as a cause of death until 1902. Between then and the end of 1909 63 deaths were certified as being from this cause; four of these took place in July, 15 in August, 36 in September, seven in October and one in November.

Figure 14 Seasonality indices for selected categories of neonatal mortality, Ipswich 1872–1909



Note: There were 1,987 neonatal deaths from congenital and birth disorders, 229 from weakness and 474 from all other categories — 2,690 in all.

Source: As Figure 3.

We have not supplied a graph of the seasonality of cause of death categories with more than 500 cases amongst post-neonates as the patterns, unsurprisingly, reflect those amongst all infants in almost all cases because, as we have seen, there was little seasonality amongst neonatal deaths. The 'congenital and birth disorders' was, however, one causal group where the seasonality differs between all infants and those surviving beyond the first month of life. Unlike neonates, post-neonatal infants were most likely to succumb to these conditions in the summer, following a pattern akin to that of 'water-food borne diseases' and 'weakness'.

'Congenital and birth disorder' was the only neonatal cause of death category containing more than 600 cases. There was very little seasonal pattern to these 1,987 deaths, except a small excess in December–March. The only other cause of death category with over 200 cases in this age group was 'weakness'. Neonatal deaths from this category had a late summer peak in September–October as well as a peak in January. All other 474 cases of neonatal death taken together show a similar 'dual' peak pattern, suggesting that new-borns faced particular dangers in both the summer and winter months. In some places the seasonality of neonatal deaths might be closely related to peaks or troughs in the number of births, but the lack of seasonality to births in Ipswich suggests this is unlikely to have been a factor here.

7 REFLECTIONS

To return to the question addressed by this paper: what was killing infants in Ipswich during our study period? In the 1880s the town's infants appear to have had a better chance of survival than those living there a decade earlier. This appears, from Figure 5, to have been due to a decline in exposure to, or the lethality of, 'water-food borne' diseases, suggesting that either weather conditions were less conducive to the spread of disease; sanitary improvements were beginning to having an impact; infants were stronger; or a less virulent mix of diseases was circulating. An interesting aspect of this decline is that, according to Figure 4, it was greater amongst boys than it was amongst girls. Like many other urban areas in England and Wales, Ipswich saw a significant upswing in deaths from 'water-food borne diseases' in the 1890s; any previous improvements in sanitation proved insufficient to mitigate

the effects of hot, dry summer weather in the growing town. It would be interesting to map the deaths from 'water-food borne diseases' from quinquennium to quinquennium to see if it is possible to pinpoint particular sanitary hazards within the town, and how these may have altered over time. A combination of more benign weather conditions, the removal of the town's privy middens, changes in its transport system and further sanitary improvements appear to have combined to reduce infant mortality from 'water-food borne diseases' in the 1900s. It is, again, interesting to note that each of the swings in this cause of death category, whether upwards or downwards, appears to have been more pronounced amongst male infants; a feature worthy of further research. Greater understanding of the 'water-food borne disease' trajectory is particularly important to our understanding of infant mortality decline, as its fluctuations may be masking the success of early measures to reduce the loss of infant life. The individual cause of death information has shown, however, that we must not consider the term 'diarrhoea' on its own as a marker of poor sanitation as medical certification practices relating to this condition changed significantly over the course of our study period.

We should also not lose sight of the fact that although deaths from 'water-food borne diseases' rose dramatically during the summer months of most years, highlighting failings in the local sewage, cleansing and water systems, they were concentrated into just two months of the year. For this reason they did not carry off as many infants as the 'airborne diseases' which were both a more constant threat and more difficult to contain. Another major killer of infants in Ipswich was the birth process itself: in every quinquennia 'congenital and birth disorders' was the largest of the causal groups used here, never accounting for less than a quarter of infant deaths. As post-neonatal mortality declined, this group formed an increasing proportion of deaths in the first year of life, making up a third of all such deaths by the years immediately prior to 1910. Increasing numbers of pregnancies where the child would not previously have survived to term may also have ended in a live birth, only for the infant to die very shortly thereafter.

Our investigations into infant mortality in the port town of Ipswich between 1872 and 1909 are still preliminary, and some observations require further investigation. 'Convulsions' and 'teething' do not appear to have been as closely associated with 'water-food borne diseases', especially 'diarrhoea', as some previous authors have assumed. More research is required to try to understand exactly what doctors meant by each of these terms. Preliminary analysis (not shown here) indicates that when multiple causes of death were listed on a certificate 'convulsions' and 'teething' were seen in conjunction with a wide variety of other causes, and therefore neither can be interpreted as the symptom of just one other specific cause; they may each have to remain as an individual category of cause of death.

The terms referring to certain causes of death, were 'replaced' by others over time (e.g. 'pneumonia' by 'broncho-pneumonia' and 'debility from birth' by 'prematurity'). It is also possible that the meaning of particular terms mutated over time and further understanding of how doctors were construing the terms involved would be useful.²⁹ The category 'airborne diseases', as used here, could usefully be broken into 'respiratory ailments' (including bronchitis, pneumonia, and broncho-pneumonia) and 'infectious diseases of childhood'. We could look at how the 'airborne infectious diseases' are related to 'other infectious diseases' and possibly regroup these. In particular, the importance of whooping cough as a cause of deaths amongst infants, and its relationship to other conditions, requires further exploration.

We need a clearer understanding of the registration process, particularly the instructions issued by the Registrar General to doctors certifying causes of death. When, for example, were doctors instructed not to describe particular 'symptoms', such as 'diarrhoea', as underlying causes of death? Do we see changes in registration practices immediately after these instructions were issued, or did they take time to work down to the 'grass roots' doctors who filled out the medical certificates of death? (Reid et al., 2015). What role did registrars play in this process? Finally, sensitive analysis of multiple CODs on individual certificates might help the understanding of how certain terms changed over time as well as facilitating comparisons between different locations or countries.

In conclusion, although Ipswich was a port, the diseases and conditions killing infants born in the town do not seem to have been very different from those seen in other towns in late-19th century England. Infancy brought its own dangers, regardless of the comings and goings around the town's docks and wharves. Thankfully, those dangers appear to have diminished as the 19th century gave way to the 20th, both in Ipswich and in England and Wales as a whole.

29 We intend to use the categorisation that the Ipswich MOH entered next to the COD entries in the vaccination death registers to explore this further.

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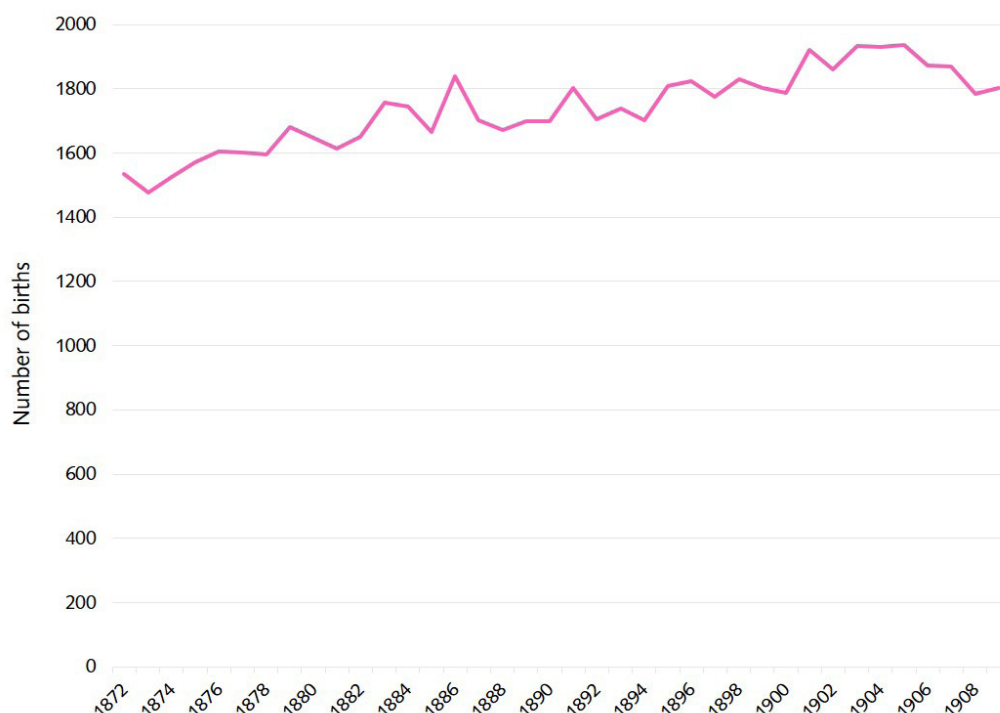
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APPENDIX A BIRTHS IN IPSWICH

Figure A1 The annual number of births occurring in Ipswich, 1872–1909



Source: Vaccination birth registers, Ipswich.

Note: Stillbirths were not registered in England until 1927, so all births shown above are assumed to have been live births.

APPENDIX B INDIVIDUAL STANDARDISED CAUSES IN THEIR CAUSAL GROUPS

The table below shows the number of cases of individual cause of death (standardised to ICD10h descriptions) amongst infants dying aged less than one year in Ipswich, 1872–1909, and each cause's percentage contribution to the total. Causes contributing more than 1% of the total (18 causes contributing 80.94% of all infant deaths) are highlighted in colour. 'Primary' causes (COD1) only considered. The congenital and birth disorders grouping accounts for 28.3% of all infant deaths, airborne diseases 22.1%, water-food borne 16.9%, weakness 12.7%, convulsions 7.0%, other non-infectious conditions 6.7%, other infectious diseases 2.2%, teething 1.9% and external causes 1.2%. Ill defined and unknown causes make up less than 1% of the total.

Table A1 Individual standardised causes in their causal groups

SHiP grouping	ICD10h description	Number	% of all infant deaths
airborne	acute laryngitis	18	0.19
airborne	acute mastoiditis	1	0.01
airborne	acute tracheitis	3	0.03
airborne	bronchitis acute	116	1.20
airborne	bronchitis unspecified	708	7.31
airborne	bronchopneumonia	172	1.78
airborne	capillary bronchitis	47	0.49

airborne	chickenpox	2	0.02
airborne	consumption	1	0.01
airborne	cough	1	0.01
airborne	croup	6	0.06
airborne	diphtheria	10	0.10
airborne	inflammation of lungs	6	0.06
airborne	influenza	30	0.31
airborne	measles	114	1.18
airborne	membranous croup	4	0.04
airborne	meningitis, cerebro-spinal	5	0.05
airborne	meningitis, unspecified	71	0.73
airborne	mumps	1	0.01
airborne	otitis media	3	0.03
airborne	phthisis	33	0.34
airborne	pleurisy	1	0.01
airborne	pleuropneumonia	1	0.01
airborne	pneumonia	256	2.64
airborne	scarlet fever	15	0.15
airborne	scrofula	7	0.07
airborne	smallpox	8	0.08
airborne	tonsillitis	1	0.01
airborne	tuberculosis	57	0.59
airborne	tuberculous meningitis	76	0.78
airborne	whooping cough	365	3.77
congenital and birth disorders	atelectasis	45	0.46
congenital and birth disorders	atrial septal defect	4	0.04
congenital and birth disorders	birth asphyxia unspecified	2	0.02
congenital and birth disorders	birth injury unspecified	9	0.09
congenital and birth disorders	cleft palate	6	0.06
congenital and birth disorders	congenital condition unspecified	3	0.03
congenital and birth disorders	congenital debility	1341	13.85
congenital and birth disorders	congenital disease	3	0.03
congenital and birth disorders	congenital heart disease	1	0.01
congenital and birth disorders	congenital heart malformation	22	0.23
congenital and birth disorders	congenital hydrocephalus	3	0.03
congenital and birth disorders	congenital ichthyosis	1	0.01
congenital and birth disorders	congenital malformation anus	7	0.07
congenital and birth disorders	congenital malformation intestine	1	0.01
congenital and birth disorders	congenital malformation rectum	4	0.04
congenital and birth disorders	congenital malformation unspecified	8	0.08
congenital and birth disorders	congenital pyloric stenosis	1	0.01
congenital and birth disorders	congenital weakness	62	0.64
congenital and birth disorders	difficulty feeding	3	0.03
congenital and birth disorders	encephalocoele	1	0.01
congenital and birth disorders	hare lip	2	0.02

congenital and birth disorders	immature infant	8	0.08
congenital and birth disorders	infantile atrophy	2	0.02
congenital and birth disorders	infantile marasmus	3	0.03
congenital and birth disorders	multiple birth - twin	2	0.02
congenital and birth disorders	neonatal jaundice	8	0.08
congenital and birth disorders	neonatal mastitis	1	0.01
congenital and birth disorders	ophthalmia neonatorum	1	0.01
congenital and birth disorders	premature infant	1131	11.68
congenital and birth disorders	scleroma neonatorum	1	0.01
congenital and birth disorders	slow foetal growth, unspecified	7	0.07
congenital and birth disorders	spina bifida	35	0.36
congenital and birth disorders	stillborn	1	0.01
congenital and birth disorders	umbilical haemorrhage of newborn	5	0.05
congenital and birth disorders	want of breast milk	4	0.04
convulsions	convulsions	664	6.86
convulsions	infantile convulsions	16	0.17
external causes	accidental suffocation or strangulation in bed	74	0.76
external causes	bitten or struck by other animal	1	0.01
external causes	burns accidental	4	0.04
external causes	burns undetermined intent	8	0.08
external causes	exposure to cold	1	0.01
external causes	fall unspecified	1	0.01
external causes	foreign body in respiratory tract	2	0.02
external causes	lack of food	2	0.02
external causes	manslaughter	1	0.01
external causes	murder	2	0.02
external causes	neglect	1	0.01
external causes	poisoning narcotics	1	0.01
external causes	scalding undetermined intent	1	0.01
external causes	suffocation undetermined intent	19	0.20
external causes	unspecified violence	3	0.03
ill-defined and unknown	anasarca	1	0.01
ill-defined and unknown	artificial feeding	5	0.05
ill-defined and unknown	bad feeding	15	0.15
ill-defined and unknown	cold	1	0.01
ill-defined and unknown	collapse	1	0.01
ill-defined and unknown	coma unspecified	1	0.01
ill-defined and unknown	constitutional disease	4	0.04
ill-defined and unknown	dropsy	3	0.03
ill-defined and unknown	enlarged glands	1	0.01
ill-defined and unknown	fever unspecified	6	0.06
ill-defined and unknown	haemorrhage	4	0.04
ill-defined and unknown	haemorrhage internal	2	0.02
ill-defined and unknown	natural causes	21	0.22
ill-defined and unknown	obstruction	2	0.02

ill-defined and unknown	poor nutrition	7	0.07
ill-defined and unknown	unclear	2	0.02
other infectious	blood poisoning	1	0.01
other infectious	candida stomatitis	12	0.12
other infectious	candidiasis (thrush) unspecified	19	0.20
other infectious	congenital syphilis	106	1.09
other infectious	encephalitis	1	0.01
other infectious	erysipelas	24	0.25
other infectious	necrotising ulcerative stomatitis	3	0.03
other infectious	poliomyelitis	1	0.01
other infectious	pyaemia	1	0.01
other infectious	ringworm	1	0.01
other infectious	septicaemia	4	0.04
other infectious	stomatitis	9	0.09
other infectious	syphilis rupia	2	0.02
other infectious	syphilis secondary	4	0.04
other infectious	syphilis unspecified	21	0.22
other infectious	tetanus	1	0.01
other infectious	tetanus neonatorum	1	0.01
other infectious	tuberculosis	1	0.01
other non-infectious	abscess of breast	1	0.01
other non-infectious	abscess/ulcer of tongue	1	0.01
other non-infectious	abdominal abscess	1	0.01
other non-infectious	abscess buttock	1	0.01
other non-infectious	abscess groin	1	0.01
other non-infectious	abscess head	3	0.03
other non-infectious	abscess limb	1	0.01
other non-infectious	abscess neck	2	0.02
other non-infectious	abscess spine	1	0.01
other non-infectious	abscess trunk	3	0.03
other non-infectious	abscess unspecified	12	0.12
other non-infectious	acute vulvitis	1	0.01
other non-infectious	anaemia	3	0.03
other non-infectious	apoplexy	1	0.01
other non-infectious	asphyxia	15	0.15
other non-infectious	atrophy of intestine	2	0.02
other non-infectious	bowel inflammation	1	0.01
other non-infectious	brain abscess	1	0.01
other non-infectious	brain congestion	13	0.13
other non-infectious	brain disease	5	0.05
other non-infectious	brain effusion	1	0.01
other non-infectious	brain haemorrhage	3	0.03
other non-infectious	brights disease	3	0.03
other non-infectious	bronchial catarrh	7	0.07
other non-infectious	bronchial congestion	1	0.01

other non-infectious	bronchitis chronic	1	0.01
other non-infectious	cellulitis other	1	0.01
other non-infectious	cellulitis unspecified	4	0.04
other non-infectious	cervical abscess	1	0.01
other non-infectious	circumcision	1	0.01
other non-infectious	complications of labour unspecified	3	0.03
other non-infectious	compression of brain	1	0.01
other non-infectious	congestion of kidney	1	0.01
other non-infectious	congestion of liver	3	0.03
other non-infectious	cyanosis	9	0.09
other non-infectious	dermatitis	1	0.01
other non-infectious	disease of larynx	1	0.01
other non-infectious	disease of mouth	1	0.01
other non-infectious	disease of spinal cord	1	0.01
other non-infectious	eclampsia	5	0.05
other non-infectious	eczema	10	0.10
other non-infectious	enlarged liver	1	0.01
other non-infectious	epilepsy	3	0.03
other non-infectious	epistaxis	1	0.01
other non-infectious	erythema	1	0.01
other non-infectious	gangrene	2	0.02
other non-infectious	gastric disease unspecified	1	0.01
other non-infectious	gastro-intestinal haemorrhage	2	0.02
other non-infectious	haematemesis	2	0.02
other non-infectious	haemophilia	1	0.01
other non-infectious	heart disease unspecified	6	0.06
other non-infectious	heart failure	6	0.06
other non-infectious	hepatitis	4	0.04
other non-infectious	hernia unspecified	1	0.01
other non-infectious	hodgkins disease	1	0.01
other non-infectious	hydrocephalus	40	0.41
other non-infectious	inflammation unspecified	2	0.02
other non-infectious	inflammation navel	1	0.01
other non-infectious	inflammation skin	1	0.01
other non-infectious	inguinal hernia	1	0.01
other non-infectious	intrapartum haemorrhage	1	0.01
other non-infectious	intussusception unspecified	9	0.09
other non-infectious	jaundice	28	0.29
other non-infectious	kidney disease	1	0.01
other non-infectious	laryngeal spasm	45	0.46
other non-infectious	liver disease unspecified	1	0.01
other non-infectious	long labour	5	0.05
other non-infectious	lung congestion	124	1.28
other non-infectious	lung disease unspecified	3	0.03
other non-infectious	lymphadenitis	1	0.01

other non-infectious	malabsorbtion of food	4	0.04
other non-infectious	malignant neoplasm pelvis	1	0.01
other non-infectious	malnutrition	29	0.30
other non-infectious	melaena	2	0.02
other non-infectious	meningeal brain haemorrhage	1	0.01
other non-infectious	mesenteric disease	84	0.87
other non-infectious	naevus	1	0.01
other non-infectious	neglect	1	0.01
other non-infectious	nephritis	10	0.10
other non-infectious	nonspecific lymphadenitis	1	0.01
other non-infectious	obstruction of bile duct	1	0.01
other non-infectious	other non-infective condition of lymph nodes	4	0.04
other non-infectious	pemphigous	7	0.07
other non-infectious	perforation of intestine	1	0.01
other non-infectious	pericarditis	1	0.01
other non-infectious	periostitis	1	0.01
other non-infectious	peritonitis	3	0.03
other non-infectious	poor circulation	4	0.04
other non-infectious	psoriasis	1	0.01
other non-infectious	pupura haemorrhagica	1	0.01
other non-infectious	purpura	1	0.01
other non-infectious	respiratory failure	1	0.01
other non-infectious	rickets	13	0.13
other non-infectious	sarcoma pelvis	1	0.01
other non-infectious	stomach disease unspecified	1	0.01
other non-infectious	stomach inflammation	1	0.01
other non-infectious	strangulation intestines	6	0.06
other non-infectious	syncope	15	0.15
other non-infectious	tetany	1	0.01
other non-infectious	throat ulcer	1	0.01
other non-infectious	tumour bones of skull and face	1	0.01
other non-infectious	tumour head face neck	1	0.01
other non-infectious	tumour illdefined site	1	0.01
other non-infectious	ulcer of intestine	3	0.03
other non-infectious	ulcer unspecified	1	0.01
other non-infectious	umbilical hernia	1	0.01
other non-infectious	vomiting	17	0.18
other non-infectious	weak heart	3	0.03
stated to be 'unknown'	stated 'unknown'/unclear	12	0.12
teething	dentition	151	1.56
teething	teething	33	0.34
water-food borne	bowel catarrh	1	0.01
water-food borne	cholea	1	0.01
water-food borne	choleraic diarrhoea	5	0.05
water-food borne	colitis	2	0.02

water-food borne	constitutional weakness/debility	10	0.10
water-food borne	diarrhoea	1213	12.53
water-food borne	dysentery	7	0.07
water-food borne	dyspepsia	5	0.05
water-food borne	enteritis	73	0.75
water-food borne	gastric catarrh	13	0.13
water-food borne	gastritis acute	2	0.02
water-food borne	gastritis unspecified	17	0.18
water-food borne	gastro-enteritis	144	1.49
water-food borne	indigestion	2	0.02
water-food borne	infantile diarrhoea	38	0.39
water-food borne	tabes mesenterica	98	1.01
water-food borne	tubercular diarrhoea	1	0.01
water-food borne	tuberculosis of intestines	7	0.07
weakness	asthenia	41	0.42
weakness	atrophy	221	2.28
weakness	cachexia	1	0.01
weakness	debility	129	1.33
weakness	exhaustion	8	0.08
weakness	inanimation	32	0.33
weakness	marasmus	784	8.10
weakness	wasting	15	0.15
Total N of infant deaths		9682	100.00

Source: Calculated from copies of the death registers, Ipswich 1872–1909