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RESEARCH ARTICLE

Temperature- and seasonality-related infectious disease mortality among infants: A retrospective time-series study of Sweden, 1868–1892

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ABSTRACT Climate conditions, such as ambient temperatures, play a crucial role in infants' vulnerability to infectious diseases. However, little is known about how climate conditions, such as temperatures and seasonality, affect infectious disease mortality among infants in high mortality settings. The aim of our study was to investigate the association between cause-specific infant mortality and ambient temperatures and seasonality. We applied a retrospective study design using parish register data from Sweden covering the 1868–1892 period in combination with daily temperature data. Mortality due to water- and foodborne diseases, airborne infectious diseases and other causes was modelled as a function of temperature exposure in the previous 14 days using distributed lagged non-linear models. We found that airborne infectious disease mortality was not related to cold temperatures, but rather to seasonality. The summer peaks in mortality due to water- and foodborne infections were associated with high temperatures, and not with seasonality. The increased vulnerability of infants to infectious diseases at high temperatures is a significant future risk, given that global temperatures are projected to rise in the coming decades.

KEYWORDS Temperature • Seasonality • Infectious disease • Infant mortality • Retrospective study

Introduction

In the first year of life, infants' vulnerability to infectious diseases is significantly influenced by climate conditions, and particularly by the ambient temperatures (Hedlund et al., 2014; Junkka et al., 2021; Karlsson et al., 2021). Very few research studies have examined the relationship between infectious disease mortality among infants and temperature in pre-industrial societies with cold climates. There is, by contrast, ample evidence that seasonal variations affect infants' vulnerability to infectious diseases (Martinez, 2018; Pappas et al., 2008). The incidence of water- and foodborne infectious diseases (WFID), such as infectious diarrhoea, increases in warm seasons (Liang et al., 2021); while the incidence of airborne infectious diseases (AID), such as infectious respiratory diseases, increases in cold seasons (Hare et al., 1981). Given the expected changes in climate conditions (IPCC, 2021),

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it is important that we gain a better understanding of the respective roles of temperature and seasonality in infants' vulnerability to infectious diseases.

A review from 2014 found strong evidence of an association between heat and WFID in Arctic and sub-Arctic settings, but only weak evidence of a link between temperature and AID, partially due to a lack of studies on the topic (Hedlund et al., 2014). Overall, there is strong empirical evidence that temperature and WFID are linked. For example, warm temperatures improve the survival chances of dysentery pathogens, which, in turn, increase mortality risks. Warm temperatures can have both an immediate (single-day) effect as well as a cumulative lagged effect on WFID (Liang et al., 2021). The mechanisms linking cold temperatures to infectious disease mortality are more uncertain. The seasonality of respiratory viral infections is related to seasonal variations in both temperature and human behaviour. Temperature affects immune responses to viral infections, which may, in turn, increase vulnerability to AID. However, among infants, AID mortality is related to both hot and cold temperatures (Xu et al., 2012), while WFID mortality (from conditions such as infectious gastroenteritis disease) increases at higher temperatures in high-income countries as well as in low- and middle-income countries (Xu et al., 2012, 2014).

Studies of historical populations have found strong associations between temperature and infant mortality, with mortality increasing with both hot and cold temperatures. It has, for example, been shown that in the 19th-century Netherlands, infant mortality increased after a heatwave (Ekamper et al., 2009). Another study found that neonatal mortality increased with both high and especially low temperatures in northern Sweden, especially among boys (Junkka et al., 2021). Research results on mortality from infectious diseases in historical populations are scarce. Studies of historical populations in England and New Zealand have reported higher incidences of diarrheal diseases at high temperatures (Galloway, 1985; Sadetskaya, 2015).

In addition to being correlated with temperatures, seasonal changes are associated with changes in human behaviour that can influence disease exposure. For example, due to higher contact rates, exposure to airborne infections tends to increase in the winter (Moriyama et al., 2020); while higher social interaction and outdoor activity levels can increase exposure to WFID in the summer (Hubálek, 2005; Morand et al., 2013). Infant mortality follows a clear seasonal pattern. While overall mortality peaks in the winter, some areas, which are often urban centres, also have a summer peak (Burkart et al., 2011; Rau, 2007). Studies of pre-industrial populations have found both summer and winter mortality peaks. In Italy, infant mortality was three times higher in the winter than in the summer (Breschi et al., 2000; Dalla-Zuanna and Rosina, 2011). Similar patterns have been identified in Britain, where infant mortality peaked in the winter and reached a low point in the summer, except in urban areas, where there was also a summer peak (Huck, 1994). The opposite pattern was found in eastern Europe, where infant mortality was shown to be highest in the summer (Breschi and Livi-Bacci, 1997; Tymicki, 2009). People living in Swedish Sapmi, and especially the Sami population, followed the general pattern of infant mortality peaking in the winter (Karlsson, 2018; Karlsson et al., 2019). Given that infants are especially vulnerable to infectious diseases, it is easy to assume that the observed seasonal patterns in infant mortality were related to seasonal variations in temperature exposures (Hare et al., 1981; Junkka et al., 2021; Karlsson et al., 2021; Scalone and Samoggia, 2018; Schumann et al., 2019).

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Here, we aim to investigate how infectious disease mortality among children in their first year of life (infants) is associated with both ambient temperatures and seasonality using historical register data from Sweden and daily temperature data covering the 1868–1892 period.

Methods

We applied a retrospective study design using population data from digitised church records. Data were collected from the POPUM database, which contains digitalised parish records covering the Sundsvall region over the 1868–1892 period (Extraction ID: U210002) (Vikström et al., 2002; Westberg et al., 2016).

Study population

The sample consisted of all children born over the 1868–1892 period within 14 parishes surrounding the town of Sundsvall (see Figure S1, supplementary material available online at https://doi.org/10.1553/p-33g4-pgab). We selected information on each child's birthdate, place of birth, date of death, cause of death and last observation date. The town of Sundsvall experienced rapid population growth during the study period, which was primarily driven by an increase in the working-class population employed at the sawmills. As of 1890, the total population around the town of Sundsvall was approximately 63,353. While infant mortality was high in the town of Sundsvall, the area underwent the demographic transition during the study period, which led to a decline in infant mortality rates (Edvinsson, 1992).

Temperature and seasonality

Daily temperature data were collected from the Swedish Meteorological and Hydrological Institute (SMHI). We used observational data from 1868 to 1892 on temperature readings at three points within the day (morning, noon and afternoon) taken at the weather station in Härnösand, which is located about 40 kilometres north of the town of Sundsvall (Figure S1).

Temperatures spanned from −33.9 °C in the winter to +27.5 °C in the summer (Figure S3, supplementary material). Median monthly temperatures below zero were observed between November and March. The coldest days were observed in February, with a median daily temperature of −5.1 °C; while the hottest days were observed in July, with a median daily temperature of +15.9 °C.

From these observations, we calculated average daily temperature exposures over the past 14 days for each day between 15 January 1868 and 31 December 1892. It is important to note that there were five (non-adjacent) missing temperature readings in our data, which we approximated using non-linear interpolation to ensure the continuity and accuracy of our data.

Seasonality was measured using the day of the year from day 1 to day 366. This approach allowed us to capture the cyclical nature of seasonal variations in temperature and their potential impact on infectious disease mortality.

Infectious disease mortality

In this study, infant mortality is defined as the death of a child under one year old. Infectious disease mortality among infants was identified using data on causes of death from historical records. The POPUM population database contains information on causes of death originating from the death and burial registers. After 1860, causes of death were determined either by the parish ministers or, in cities and towns, by physicians. The primary and secondary causes were recorded in death certificates, which were transferred by the ministers to death and burial records (Rogers, 1999). Even though causes of death were recorded by members of different professions in rural and in urban areas, ministers and physicians followed the same nosological system defined in the national regulations (SFS, 1874:60, 1860:13, 1874:61). However, in cities and towns, the precision of the diagnoses was much higher, and the proportions of unknown causes of death were lower, than in rural areas.

In the present study, the ICD10h code system has been used to code and to classify the digitised historical cause-of-death (COD) data. The ICD10h system was designed for encoding historical COD data using the modern ICD10 (10th revision of the International Statistical Classification of Diseases and Related Health Problems) as a baseline. Given that medical knowledge and recording practices have changed over time, ICD10 is not fully applicable to historical data without the loss of important information. For example, the disease term "teething" was frequently used to refer to diarrheal conditions in infants. Thus, a historical contextual system of classification is necessary. The ICD10h further subdivides the ICD10 codes to retain historical information, while its use of ICD10 as a baseline allows us to link historical disease patterns to contemporary ones (Janssens, 2021). Therefore, the system enables us to compare deaths attributable to larger disease groups, such as AID, WFID and other causes of death, among infants.

We focused on deaths in which the primary cause of death (COD) was identified as any infectious disease. In cases of multiple causes of death, each COD was coded separately and then assigned to one of the three COD groups described above. If two or more infectious diseases were listed, we considered the disease that started the chain leading to death as the primary COD.

We noted that throughout the study period, a significant share of the infant deaths in our study population was not attributed to a specific cause. However, as time progressed, there was a noticeable increase in the share of infant deaths for which the specific cause of death was identified, reaching approximately 50% of all infant deaths by the 1890s (Figure S2, supplementary material).

In our analysis, we took into account the geographical contexts in which the deaths occurred, and specifically whether they took place in urban or rural areas. We found notable urban-rural differences in the proportions of unknown causes of death. In rural regions, the proportion of deaths without a known cause was high, at 75.5%. Conversely, in urban areas, this proportion was markedly lower, at just 8.3%. This discrepancy could be attributed to the higher precision of diagnoses in urban areas, which led to smaller proportions of deaths without a specified cause in cities and towns.

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Statistical analysis

We applied time-series analysis to model the relationship between daily cause-specific infant mortality, seasonality and ambient temperature exposure. Data were aggregated to count data on deaths per day by age group (0–13, 14–30, 31–365 days old). We estimated the effect of ambient temperature on the number of infant deaths using Poisson regressions.

$$E(Y) = \exp(\log(\alpha_t) + X\beta_t) \quad (1)$$

The expected number of deaths in a day Y was modelled as a function of person-days $\log(\alpha)$ and a model matrix, X, and β the corresponding matrix of coefficients. X included not only ambient temperature exposure, but several other factors as well. We adjusted for the type of parish (urban or rural) and for the age group of the individuals. In addition, we accounted for the non-linear effect of the day of the year, specified as a cubic spline with six degrees of freedom and the year as a cubic spline with three degrees of freedom (Boor, 1978; Hastie, 1991/1997).

It has been shown that ambient temperature has a lagged non-linear relationship to infant mortality (Junkka et al., 2021; Schumann et al., 2019). Thus, the association was modelled as a nonlinear function, specified as a cubic spline with four degrees of freedom, using the distributed lag linear and non-linear models framework. The lagged response was specified as a cubic spline with four degrees of freedom, with lag knots set at logged intervals (Gasparrini et al., 2010). Furthermore, in the cause-specific analysis, the models were simplified by specifying the temperature association as a linear threshold function. The threshold was set at the proximate minimum mortality temperature (MMT), as shown by the nonlinear specifications (see Figure 1). The results were presented as incidence rate ratios (IRR) with 95% confidence intervals (CI), which represented the relative risk of mortality compared to the MMT, or, for the seasonality analysis, 1 February. The models were specified and evaluated within the programming language R (R Core Team, 2021).

Sensitivity Analysis

In our study, we performed additional sensitivity analyses to account for potential biases and limitations in our data. We recognised that the causes of death were not similarly distributed by age (Figure S5), and that very few deaths from infectious diseases were recorded during the neonatal period (i.e., the first 28 days of life). To address these issues, we limited our sample to cases of post-neonatal mortality, defined as deaths occurring in infants aged between 28 days and one year old. The results from this sensitivity analysis showed patterns similar to those of our main findings (Figures S6 and S7, supplementary material).

Given the large proportions of deaths with unknown causes, we performed analyses in which we grouped these deaths into categories beyond the "other causes" classification. These analyses showed that deaths due to unknown causes have seasonal and temperature patterns that are similar to those of deaths due to known causes (Figures S8 and S9, supplementary material). Thus, the results indicated that unknown causes of death did not have a bias towards either AID or WFID mortality.

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We conducted a placebo test to further validate our findings. This test involved examining the association between temperature at a future time point (two months after death, $t + X$) and mortality risks. If the observed associations between temperature and mortality in our main analysis were due to some unobserved confounding factor, rather than a direct causal effect of temperature, we might expect to see a similar association in this placebo test. However, our placebo test (see Figure S10) showed no association between the temperature two months after death and the risk of dying from other causes, airborne infectious diseases (AID) or water and foodborne infectious diseases (WFID). These results support the validity of our main findings, which suggests that the observed associations between temperature and mortality are likely to be causal, rather than due to chance or confounding.

Results

There were 47,575 births over the period. Of these infants, 346 died from WFID and 898 died from AID within the first year of life. Infant mortality was lowest between the 75th and the 90th percentile of the temperature distribution, and thus at temperatures ranging from +11.8 to +15.5 °C (Table 1). Above and below this temperature range, infant mortality increased. Infant mortality due to WFID showed a linear relationship, with the rate increasing with the temperature. The opposite pattern was observed for mortality

Table 1 Infant mortality rates (per 1000) by temperature percentile and season

		Person years	All-cause mortality	WFID mortality	AID mortality
Total		32,968	135.43	9.58	18.88
Season					
Spring		8,272	148.3	8.10	24.50
Summer		8,317	151.9	22.50	15.80
Autumn		8,269	138.7	11.20	20.20
Winter		8,110	170.7	7.30	29.30
Temperature percentile					
0–10th	*−33.9 to −8.2 °C*	3,229	167.81	6.81	30.03
10–25th	*−8.2 to −2.1 °C*	4,978	168.71	8.44	28.52
25–50th	*−2.1 to +3.8 °C*	8,157	149.43	7.11	23.17
50–75th	*+3.8 to +11.8 °C*	8,324	140.18	11.65	21.26
75–90th	*+11.8 to +15.5 °C*	5,033	139.86	18.87	14.30
90–100th	*+15.6 to +27.5 °C*	3,295	167.50	29.74	16.99
N		47,575 births	6,443	456	898

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Figure 1 Incidence rate ratios (IRR) with 95% confidence intervals for all-cause infant mortality by ambient temperature exposures over the past 14 days.

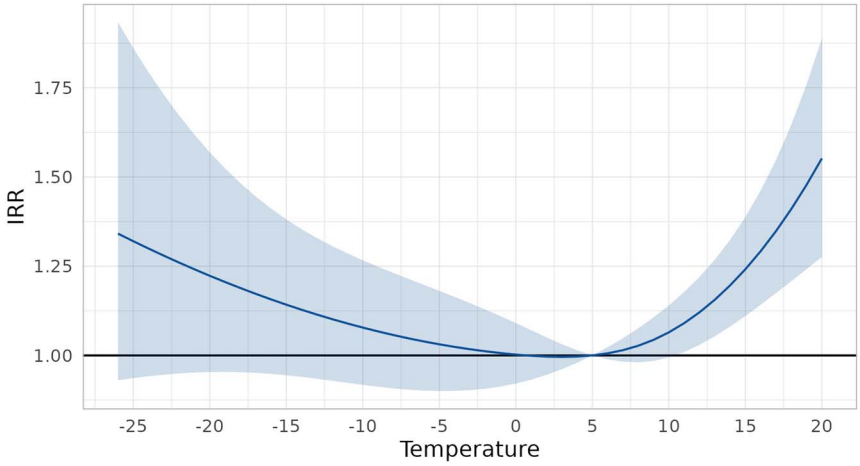
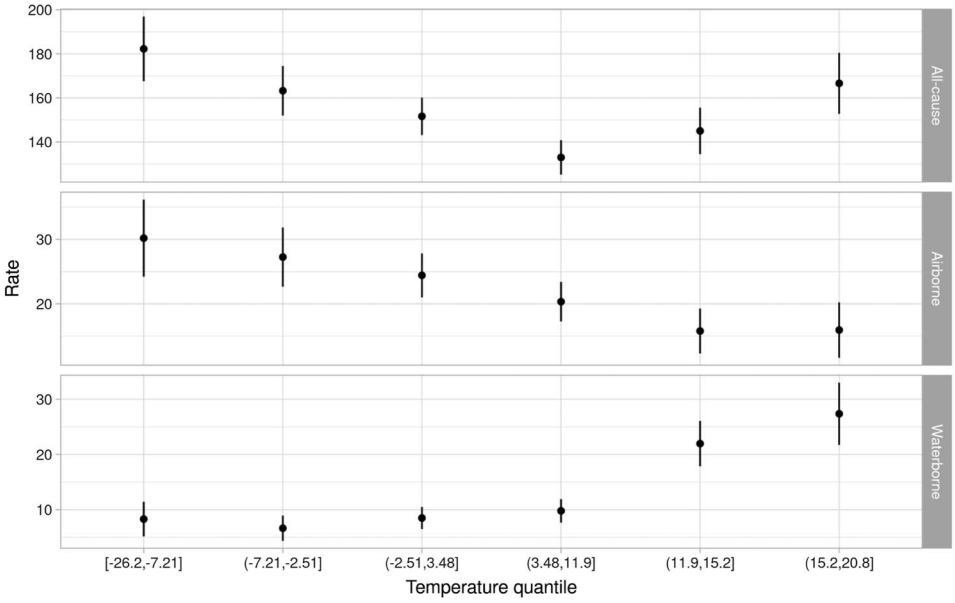


Figure 2 All-cause and cause-specific infant mortality rates by temperature percentiles (10th, 25th, 50th, 75th, 90th). CI are based on Poisson standard errors.



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due to AID, with mortality increasing as temperatures fell towards −33 °C. WFID mortality peaked in the summer while AID mortality peaked in the winter. Over the study period, IM declined, as shown in Figure S4 in the supplementary material. Most noticeable was the reduction in seasonal variations after the 1870s, when survival rates during the winter were improving.

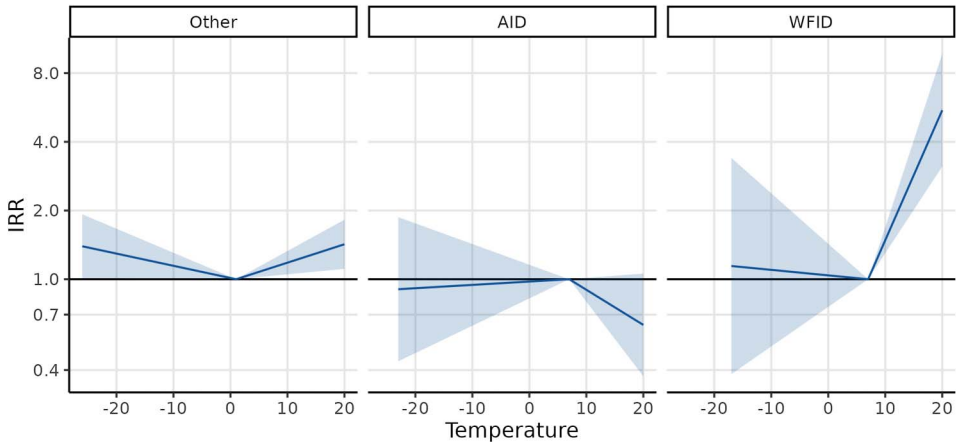
Temperature

Breaking down mortality rates by temperature exposures revealed distinct cause-specific patterns (Figure 2). For all-cause infant mortality, there was a u-shaped pattern with a MMT of around +3.5 to +12 °C, with mortality increasing above and below this range. For infant mortality due to AID, there was a linear negative relationship: as temperatures increased, infant mortality declined. Mortality due to waterborne diseases showed the opposite pattern: as temperatures increased, infant mortality increased.

The regression model (Figure 1) that adjusted for seasonality and the long-term trend showed that being exposed to temperatures over +5 °C in the past 14 days was associated with higher mortality. At the 99th percentile temperature exposure of +19 °C, the IRR was 1.48 (95% CI 1.24, 1.76) compared to the reference at the minimum mortality temperature of +5 °C. Temperature exposures below +5 °C did not show any significant association with the IRR of infant mortality, while the IRR was 1.17 at −17 °C (95% CI 0.95, 1.45).

The adjusted Poisson regression models that broke down the associations by causes showed significantly different patterns (Figure 3). No association was found between

Figure 3 Incidence rate ratios (IRR) with 95% confidence intervals for cause-specific infant mortality by ambient temperature exposures over the past 14 days. IRR are on a log scale.



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AID mortality and ambient temperature exposures over the past 14 days. By contrast, a strong positive association was observed between WFID mortality and temperatures above +7 °C, with an IRR of 5.50 (95% CI 3.12, 9.71) at +20 °C (the 99th percentile temperature exposure), while no association was found between WFID mortality and cold temperatures. For mortality related to other causes, the IRR increased slightly at temperatures both +1 °C hotter and +1 °C colder than the MMT; at −17 °C (first percentile), the IRR was 1.26 (95% CI 1.01,1.59); and at +19 °C (99th percentile), the IRR was 1.40 (95% CI 1.10, 1.77).

The associations were not uniform across lag time. Figure 4 shows the IRR of cause-specific infant mortality at exposure to temperatures of −20 °C and +20 °C (the first and the 99th percentile daily temperature exposures) over the past 1–14 days. The lagged IRR confirms that AID was not associated with any temperature changes in the past 14 days. Exposure to high temperatures (+20 °C) was associated with an increase in the IRR of WFID mortality 10 days after exposure, to 1.19 (95% CI 1.06, 1.33). A small positive association was found between mortality due to other causes and exposure to heat two days after exposure, with an IRR of 1.13 (95% CI 1.01, 1.27).

Figure 4 Lagged incidence rate ratios (IRR) with 95% confidence intervals for cause-specific infant mortality by exposure to ambient temperatures of −20 and +20 °C over the past 1–14 days. IRR are on a log scale.

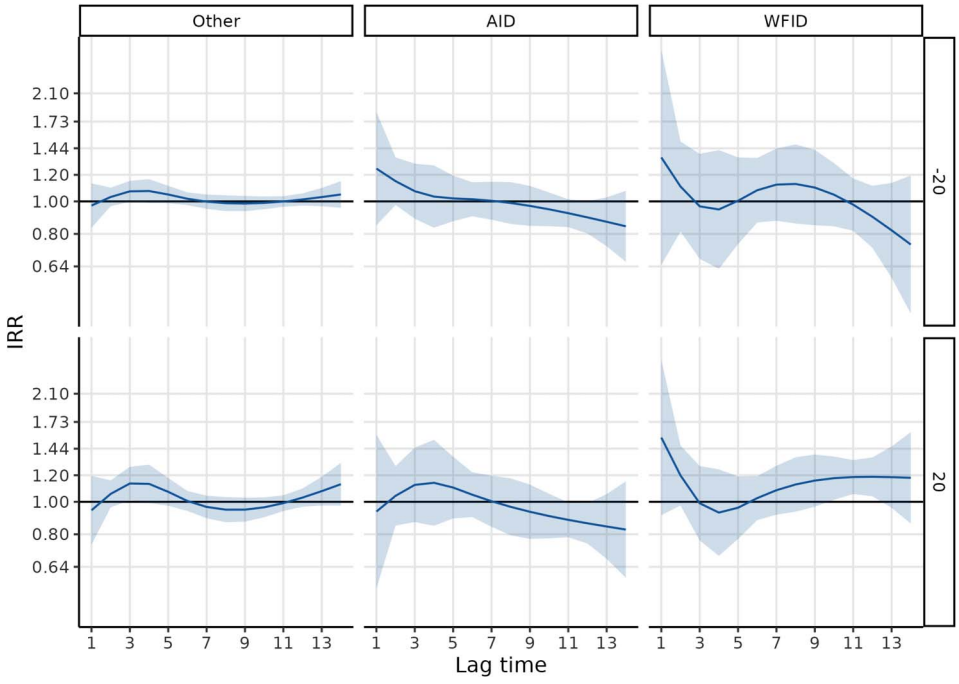

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Table 2 Historical causes of death and corresponding ICD10 and ICD10h categories

Disease group	ICD10 main category	Historical cause of death examples	ICD10h category
Air-borne	A	Tuberculosis of the lungs, pneumonia, whooping cough, etc.	A16-19, A30, A36-40
	B	Chicken pox, smallpox, measles, rubella, etc.	B01-03, B05-06, B26
	G	Meningitis	G00, G03
	H	Abscess of external ear, abscess of internal ear, meningitis following otitis media, acute mastoiditis	H60, H66, H70
	J	Influenza, inflamed throat, bronchopneumonia, etc.	J00, J02, J04-5, J11, J18, J20-22, J30, J36, J85, J86
	R	Cough, pleurisy, inflammation of lungs	R05, R09
Water-/food-borne	A	Cholera, choleraic diarrhoea, typhoid fever, dysentery, tuberculosis intestines, etc.	A00, A01, A05, A09, A18
	K	Chronic atrophic gastritis, colitis, dyspepsia, indigestion, stomach catarrh, teething, etc.	K00, K29-31, K52, K63
Other causes	E, F, J, K, P, Q, R, S, T, U, V, W, X, Y	Congenital and birth disorders, convulsions, external causes, teething, weakness or unknown	

Seasonality

In terms of seasonality, all-cause IM was highest in the winter months (December-February), followed by in the summer months (June-August); see Table 2. AID and WFID mortality displayed opposite patterns. AID mortality peaked in the winter and was lowest in the summer, while WFID mortality peaked in the summer and was lowest in the winter.

The Poisson regression models confirmed these seasonal patterns; see Figure 5. IRR and CI were calculated compared to the day of the year with the lowest observed cause-specific mortality. For all-cause mortality, the unadjusted IRR peaked in the winter months of January and February and in the summer months of July and August. However, after adjusting for temperature exposures, the IRR reached a low point in the summer season, and the IRR peak in the winter months increased (Figure 6).

The seasonal models uncovered a strong association between seasonality and cause-specific mortality (Figure 6). AID mortality followed a strong u-shaped seasonal pattern, with mortality reaching a low point in the summer (August) and a peak in the winter (December-March). The opposite, bell-shaped pattern was found for WFID mortality, with mortality reaching a peak in the summer and a low point in the winter (February).

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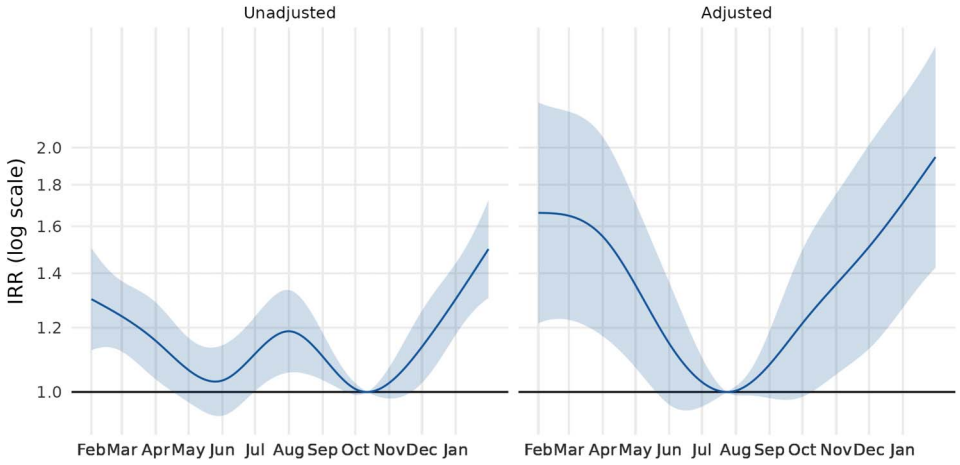
Figure 5 Incidence rate ratios (IRR) with 95% confidence intervals for infant mortality by day of the year from 1 February to 31 January. IRR are on a log scale. The reference day is set at the day of the year with the lowest mortality. Adjusted for year and temperature.



The association between the seasons and mortality from other causes followed the same patterns as those for all-cause mortality.

After adjusting for the day-of temperature and a seven-day average lagged temperature, the seasonal patterns increased for AID mortality and for mortality from other causes, while they decreased for WFID mortality. The model showed no significant relationship between the seasons and WFID mortality, which suggests that the seasonal patterns were primarily due to temperature exposure rather than to seasonal exposure.

Discussion

Infants' vulnerability to infectious diseases is associated with variations in climate conditions, such as ambient temperatures and seasonality (Hedlund et al., 2014; Junkka et al., 2021; Karlsson et al., 2021). We provide new evidence on the link between ID mortality among infants and climate conditions for a 19th-century Swedish population living in a cold climate with high mortality. Specifically, we studied the associations between ambient temperatures and seasonality and infant mortality from all causes, water- and foodborne infectious diseases, and airborne infectious diseases between 1868 and 1892.

Overall, we found that all-cause mortality and WFID mortality were strongly associated with high ambient temperatures, but were not associated with cold temperatures. We also observed that AID mortality was not associated with temperature after adjusting for time trends and seasonality. After adjusting for temperature exposure, we found a clear seasonal

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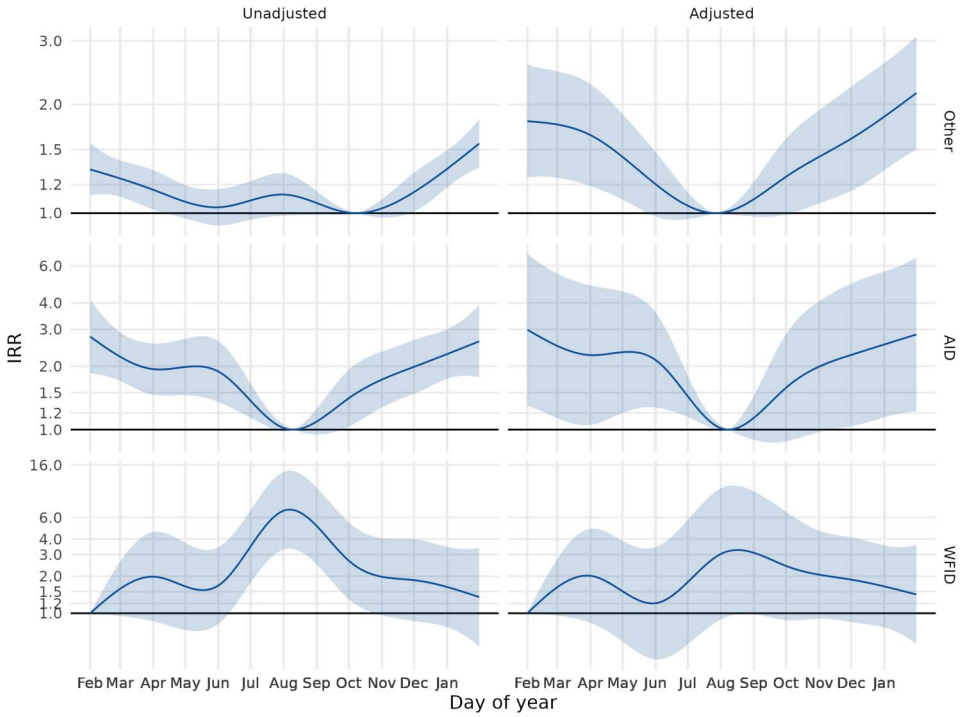
Figure 6 Incidence rate ratios (IRR) with 95% confidence intervals for cause-specific mortality among infants by day of the year from 1 February to 31 January. IRR are on a log scale. The reference day is set at the day of the year with the lowest mortality. Adjusted for year and temperature.



pattern for all-cause mortality and AID mortality, with deaths reaching a low point in the summer and peaking in the winter. Although the opposite pattern was found for WFID mortality, with a peak being observed in the summer, the association was significantly reduced after adjusting for temperature exposures, and did not differ by day of the year. Infant deaths were observed at lags of 14 days after exposure to temperatures down to −33.9 °C. During the study period, the average monthly minimum temperature in the winter months was below −15 °C. Cold temperatures have been shown to affect infant survival, largely due to the impact of hypothermia or AID (Derosas, 2009; Xu et al., 2012). Although ID mortality was not found to be related to cold temperatures in our study, mortality due to other causes was. At −17 °C, the IRR of death was 1.26 (95% CI 1.01, 1.59) compared to a temperature of +1 °C, with a peak in mortality three days after exposure. It is possible that the peaks in mortality due to other causes were driven by hypothermia and sudden infant death syndrome, which often have an immediate effect on mortality, especially among neonates (de Almeida et al., 2014). In 19th-century northern Sweden, infants were vulnerable to very low temperatures, particularly in the first weeks of life

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(Junkka et al., 2021; Schumann et al., 2019). As living standards (such as indoor heating, nutrition and income) were low and infant mortality was high in late 19th-century Sundsvall, the ability of families to protect infants against cold were limited (Edvinsson, 1992). However, our failure to find an association between AID mortality and cold temperatures suggests that mere exposure to cold temperatures was not enough to have driven high AID mortality among infants.

During our study period, the monthly average maximum temperatures in the summer months were above +19 °C. The highest numbers of infant deaths were observed during periods when the temperatures had reached +27.8 °C in the previous 14 days. It has been suggested that the high level of heat-related infant mortality observed in historical populations was attributable to gastrointestinal diseases caused by the contamination of food and water (Ekamper et al., 2009; Junkka et al., 2021). Our findings support these conclusions, as we showed that WFID were strongly related to heat exposure. Like in studies of modern populations, we found that heat exposure had a lagged effect on AID mortality, peaking after about 10 days (Xu et al., 2014). The results also showed higher IRR (albeit with high uncertainty) of AID mortality in the first few days after exposure, which could be related to the immediate effects of hyperthermia and heat stress (Auger et al., 2015; Basagaña et al., 2011; de Almeida et al., 2014). However, it should be noted that although we found that mortality due to other causes was positively associated with temperatures above + 1 °C, we observed no significant lagged patterns.

We found a significant association between seasonality and AID mortality that peaked in January-February, and became stronger after adjusting for lagged temperature exposures. These findings suggest that AID mortality was primarily driven by seasonality rather than by low temperatures. It is possible that changes in people's behaviours during the winter, such as a tendency to engage in more indoor activities, increased their exposure to viral infections (Moriyama et al., 2020). It is also possible that higher levels of indoor pollution during the winter due to the use of wood-burning stoves for heating (Bergman, 2010) increased the risk of viral infections (Ciencewicki and Jaspers, 2007). Furthermore, our findings indicate that the summer peak in WFID mortality was not related to seasonality, but rather to heat exposure. It has been suggested that because people engaged in more outdoor activities during the summer, their proximity to animals and their consumption of fresh food likely increased, which might, in turn, have increased their exposure to WFID (Tymicki, 2009). Although 19th-century Sundsvall experienced rapid urbanisation (Edvinsson, 1992), the city was relatively small compared to many European cities for which a summer peak in mortality has been observed in contemporary populations (Huck, 1994; Tymicki, 2009). Thus, our failure to observe a seasonal effect might be related to the low population density of the study area.

Strengths and limitations

Ours is among the first studies on infectious disease mortality and its relationship to seasonality and temperature in a 19th-century population. Without access to cause-specific data, previous studies have only been able to speculate about the mechanisms driving the relationship between the climate and infant mortality, which we have been able to

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address (Ekamper et al., 2009; Huck, 1994; Junkka et al., 2021; Schumann et al., 2019; Tymicki, 2009). Nonetheless, historical cause-specific data on infectious diseases have important limitations. The medical knowledge and nomenclature used by parish ministers and provincial doctors during the 19th century differ from modern medical diagnoses (Revuelta-Eugercios et al., 2021). Although the historical causes of death are focused on descriptions of symptoms, infectious and non-infectious diseases are, in general, easier to differentiate than individual conditions using the ICD10h coding system, which is tailored for historical cause-of-death data (Janssens, 2021). Furthermore, additional sensitivity analyses were performed that limited the sample to post-neonatal mortality and differentiated between unknown and other causes of death. The results of these analyses were similar to those of the main findings (Figures S5, S6, S7, S8, S9 in the supplementary material).

As we limited our measurements of climate conditions to seasonality and ambient temperatures, the importance of humidity was overlooked. Especially for WFID, humidity in combination with heat is an important factor in exposure and mortality (Martinez, 2018; Pappas et al., 2008). Our study also lacked data on indoor environmental conditions, such as on levels of air pollution. Thus, we were not able to disentangle different forms of seasonal variations in behaviour and the environment that might have driven the seasonal patterns.

Conclusions

Our findings suggest that both seasonality and high temperatures were important factors in infants' vulnerability to infectious diseases. We found that AID mortality was not related to cold temperatures, but rather to seasonality; and that peaks in summer mortality due to WFID were associated with high temperatures, and not with seasonality.

These findings have broader implications beyond the historical context of our study. They contribute to our understanding of the complex relationships between climate factors and infectious disease mortality among infants. Such insights are particularly important in the context of ongoing climate change, which is expected to significantly alter temperature patterns, which could, in turn, affect disease dynamics.

For instance, while an association between temperature and WFID is unlikely to be found in contemporary Sweden, where there is easy access to clean sources of water, the evidence provided could be informative for other areas of the world, where such access is not as readily available. As Ledberg (2020) pointed out, seasonal fluctuations in mortality have been declining in Sweden, particularly among the elderly, and this trend might be present in infants as well.

Moreover, our study contributes to our understanding of how climate change might affect infectious disease patterns. The increased vulnerability to infectious diseases at high temperatures of infants living in cold climates is a significant risk factor, given the expected levels of global warming in the coming decades. This issue is particularly relevant for populations living in regions where the medical advancements and infrastructure may not be sufficient to mitigate the risks associated with increasing temperatures.

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However, caution is needed when applying our findings to current climate change challenges. The medical knowledge of and the nomenclature used by parish ministers and provincial doctors during the 19th century differ from those of modern physicians making medical diagnoses.

In conclusion, our study underscores the importance of considering both seasonality and temperature when analysing the historical and the future impacts of climate on infectious disease mortality among infants. Further research is needed to deepen our understanding of these relationships and their implications for public health in the context of ongoing climate change.

Supplementary material

Available online at https://doi.org/10.1553/p-33g4-pgab
Supplementary file 1. Figures S1–S10, Tables S1–S3.

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