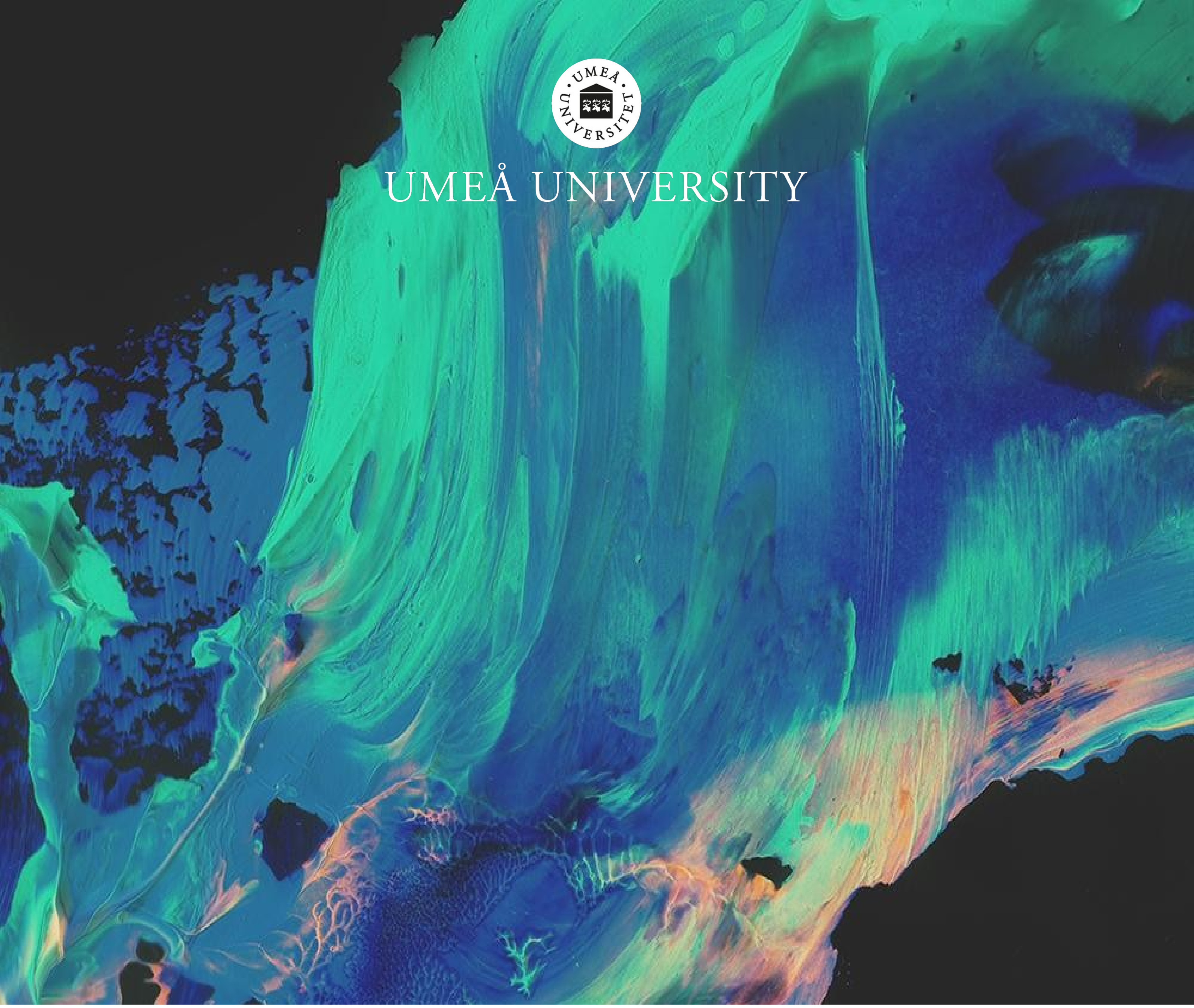

Infectious disease mortality among infants, seasonality and ambient temperature in Sweden, 1868-1892

Johan Junkka and Maria Hiltunen

CEDAR Working Papers 2022:23
Centre for Demographic and Ageing Research

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Abstract

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Abstract

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Introduction

Climate variability, such as ambient temperature, is crucial for infants' vulnerability to infectious diseases (Hedlund et al., 2014; Junkka et al., 2021; Karlsson et al., 2021). When it comes to pre-industrial societies in cold climates, very few research studies have examined the relationship between infectious disease mortality among infants and temperature. Seasonal variations, on the other hand, are well known to affect infants' vulnerability to infectious diseases (Martinez, 2018; Pappas et al., 2008). Water- and foodborne infectious diseases (WFID) such as infectious diarrhoea, increases in warm seasons (Liang et al., 2021), and airborne infectious diseases (AID), such as infectious respiratory diseases, correlates with cold winter seasons (Hare et al., 1981). Given the expected changes in climate variability (IPCC, 2021), a better understanding of the respective role of temperature and seasonality for infants' vulnerability to infectious diseases is of importance.

A review from 2014 found overall strong evidence for heat and food- and waterborne diseases in arctic and sub-arctic settings. Yet, there is weak evidence for a link between temperature and airborne infectious diseases, partially due to a lack of studies (Hedlund et al., 2014). Overall, there is strong empirical evidence for a link between temperature and WFID. For example, warm temperatures improve survival for Dysentery pathogens, which in turn increases mortality risks. The effect of 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 both seasonal variations in temperature and human behaviours. Temperature affects the immune responses to viral infections, increasing vulnerability to AID. However, AID among infants is related to both hot and cold temperatures (Xu et al., 2012), while WFID infant mortality increases (such as from infectious gastroenteritis disease) with temperatures in both high income as well as low- and middle-income countries (Xu et al., 2012, 2014).

Studies of historical populations show a strong association between temperature and infant mortality, which increases with hot but also with cold temperatures.Infant mortality in the 19th century Netherlands increased after a heatwave (Ekamper et al., 2009). Neonatal mortality has been shown to increase with both high and especially low temperatures in northern Sweden, especially among boys (Junkka et al., 2021). Regarding mortality in infectious diseases in historical populations, results are scarce to lean on. Galloway showed in a study conducted on data from England 1773-1825, that infant mortality in diarrheal diseases correlated with high summer temperatures (Galloway, 1985). Sadetskaya obtained similar results in a study from New Zealand, 1873-1916, which showed higher incidences of diarrheal diseases at high temperatures (Sadetskaya, 2015).

Not only do temperatures correlate with seasonality, seasonal changes in human behaviour affects, for example, contact rates, increasing exposure to airborne infections in the winter (Moriyama et al., 2020), or through greater social interactions and outdoor activity in the summer, increasing exposure to WFID (Hubálek, 2005; Morand et al., 2013). Infant mortality has a strong seasonal pattern. Overall, mortality peaks in the winter while some, often urban, areas also have a summer peak (Burkart et al., 2011; Rau, 2007). Studies of pre-industrial populations reveal both summer and winter peaks in mortality. In Italy, infant mortality was three times higher in winter than in summer (Breschi et al., 2000; Dalla-Zuanna & Rosina, 2011). Similar patterns have been shown for Brittain, where infant mortality peaked in winter and reached a low level in summer, except for urban areas which also showed a summer peak (Huck, 1994). The opposite pattern was found in studies in eastern Europe where infant mortality was highest in the summer (Breschi & Livi-Bacci, 1997; Tymicki, 2009). Populations in Swedish Sapmi follow the general pattern of high winter infant mortality, especially among the Sami population (Karlsson, 2018; Karlsson et al., 2019). Given that infants are especially vulnerable to infectious diseases, the observed seasonal patterns in infant mortality were likely related to seasonal variations in temperature exposures (Hare et al., 1981; Junkka et al., 2021; Karlsson et al., 2021; Scalone & Samoggia, 2018; Schumann et al., 2019).

We aim to investigate first, the association of infectious disease mortality among infants and ambient temperature, and second the association to seasonality, using historical register data from Sweden covering the period 1868-1892.

Method and data

We applied a retrospective study design using population data from digitised church records. Data was collected from the POPUM database covering the Sundsvall region, 1868-1892 (Extraction ID: U210002) consisting of digitalised parish records (Vikström et al., 2002; Westberg et al., 2016). The sample consisted of all children born over the period, 47 575 births, within 14 parishes surrounding the town of Sundsvall, see Figure 1. We selected information on 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.

Primarily driven by a growth of the working classes, working at the sawmills in parishes surrounding the Gulf of Bothnia. Infant mortality was overall high in the town of Sundsvall, and the area experienced the demographic transition during the study period with overall declining infant mortality rates (Edvinsson, 1992).

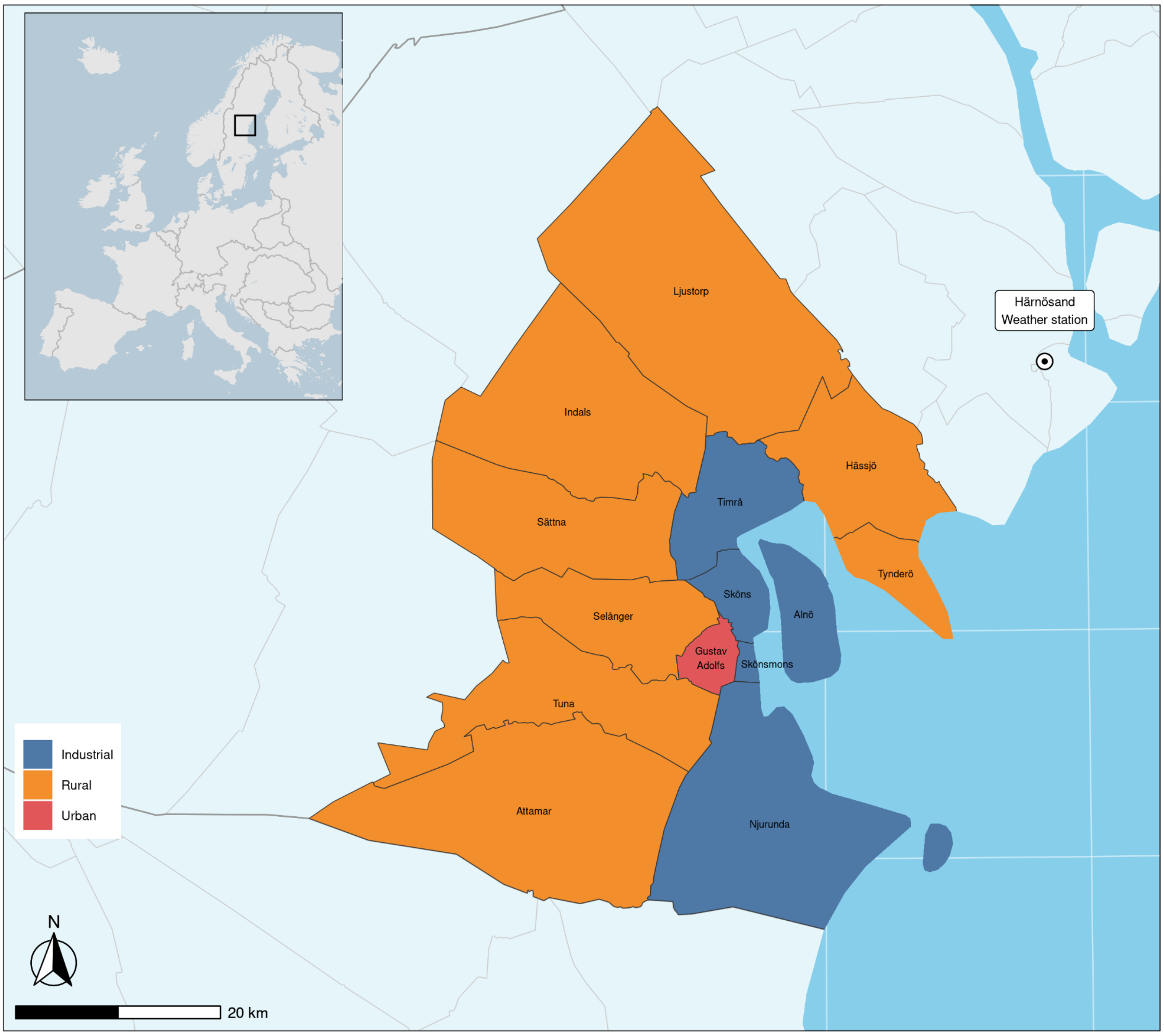

Figure 1: *Map of sample area in 1890.*

Cause-specific infant mortality

In the present study, the ICD10h-code system has been applied to code and classify the historical cause of death data from the POPUM database at CEDAR. In the population database POPUM there is information on causes of death originating from the death and burial registers. After 1860 causes of death were determined by either the parish ministers or, in cities and towns, by a physician, and the primary and secondary causes were recorded in death certificates. The causes of death were then transferred by the ministers to death and burial records (Rogers, 1999). Even though causes of death was recorded by different professions in rural and urban areas, ministers and physicians followed the same nosological system of the national regulations where the nomenclature to use was defined (Svensk Författningssamling, 1860:13, 1874:61,1891:80). However, the precision of diagnosis was much higher in cities and towns than in rural areas, and the proportion of unknown causes of death was lower in the city compared to the countryside.

The ICD10h system, developed within the international SHiP network, is designed for encoding historical cause of death data, using the international ICD10 (2016) classification system as a baseline. Given that historical medical knowledge and recording practises have changed over time, the ICD10 system is not fully applicable for historical uses 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, while the usage of the modern ICD10 as a baseline allows for a connection of historical disease patterns to contemporary ones (Janssens, 2021). Therefore, the system allows for the comparison of larger disease groups such as AID, WFID and other causes of death among infants.

Airborne infectious diseases included causes of death such as whooping cough, pneumonia, smallpox, diphtheria and measles. Approximately this group of diseases corresponds to ICD10 chapter 10 (J00-J99) and 1 (A00-B99). Causes of death such as gastrointestinal inflammation, diarrhoea and cholera were categorised as water- and foodborne infectious diseases corresponding to ICD10 chapter 11 (K00-K93) and 1 (A00-B99). The most common causes of death grouped in the category of "other diseases" were convulsions, congenital- and birth disorders and external causes.

In the case of deaths where several diseases were listed on the death record, it was not always that the one we have assigned as the primary cause of death was mentioned first. Thus, in cases of multiple causes of death, we have coded each cause of death separately and then assigned the disease to one of the 4 infant groups described. To decide which disease to choose as the primary cause of death, the following rules have been applied:

1. if one of the causes is an infectious disease, then use this to categorise the primary cause of death; i.e. 'air-borne', 'other infectious' or 'water-food borne'

2. else, if only other causes were given such as external causes, teething, or birth disorders, use ‘other’.
3. If no cause was given use ‘unknown’

If there were two or more infectious diseases listed, we have considered how they might have formed the chain of diseases which led to the death and taken the disease starting that chain.

In rural areas in particular, the cause of death for infants was rarely reported, but over time the proportion of specified causes of death increased and reached 50 percent in the 1890s. (figure A1, appendix).

Temperature data

Daily temperature data were collected from the Swedish Meteorological and Hydrological Institute (SMHI). We used observational data on temperatures at three points within the day (morning, noon and afternoon), from the weather station in Härnösand from 1868 to 1892, located about 40 kilometres north of the town of Sundsvall (Figure 1). Temperatures spanned from -33.9 ℃ in the winter to +27.5 ℃ in the summer (Figure A2, appendix). Median monthly temperatures below 0 were observed between November to March, with the coldest days observed in February (median -5.1 ℃). The highest temperatures were observed in July with a median daily temperature of +15.9 ℃. From these observations, we calculated average daily temperature exposures over the past 14 days for each day between January 15, 1868, to December 31, 1892.

Statistical methods

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 of deaths per day by age group; 0-13, 14-30,31-365 days old. We estimated the effect of ambient temperature on the count of infant deaths using Poisson regressions.

$$E(Y) = exp(log(\alpha\ ) + X\beta)$$

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. Within *X*, in addition to ambient temperature exposure, the non-linear effect of day of the year was specified as a cubic spline with six degrees of freedom and year as a cubic spline with three degrees of freedom (Boor, 1978; Hastie, 1991/1997).

Ambient temperature has been shown to have 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 4 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 as shown by the nonlinear specifications (see Figure 3). The models were specified and evaluated within the programming language R (R Core Team, 2021).

Results

Infant mortality reached a minimum between the 75th and 90th percentile, with temperatures +11.8 - +15.5 ℃, see table. Above and below this temperature range, infant mortality increased. IM due to Water- and foodborne infectious diseases show a linear relationship, where the rate increases with temperature. The opposite pattern was seen for mortality due to airborne infectious diseases, where mortality increased as temperatures fell towards -33 ℃.

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 - -8.2* ℃	3,229	167.81	6.81	30.03

10-25th	*-8.2 - -2.1* °C	4,978	168.71	8.44	28.52
25-50th	*-2.1 - +3.8* °C	8,157	149.43	7.11	23.17
50-75th	*+3.8 - +11.8* °C	8,324	140.18	11.65	21.26
75-90th	*+11.8 - +15.5* °C	5,033	139.86	18.87	14.30
90-100th	*+15.6 - +27.5* °C	3,295	167.50	29.74	16.99
N		47,575 births	6,443	456	898

Over the study period, IM declined, as seen in Figure A3. Most noticeably was the reduction in seasonal variations after the 1870s, as survival during the winter improved.

Temperature

Breaking down mortality rates by temperature exposures we see quite different cause-specific patterns (Figure 2). For all-cause infant mortality, there was a u-shaped pattern with a minimum mortality temperature (MMT) around +3.5 - +12 °C, above and below this range infant mortality increased. For mortality due to airborne infectious diseases there was a linear negative relationship, as temperature increased, infant mortality declined. Mortality due to waterborne diseases show the opposite pattern, as temperatures increase so did infant mortality.

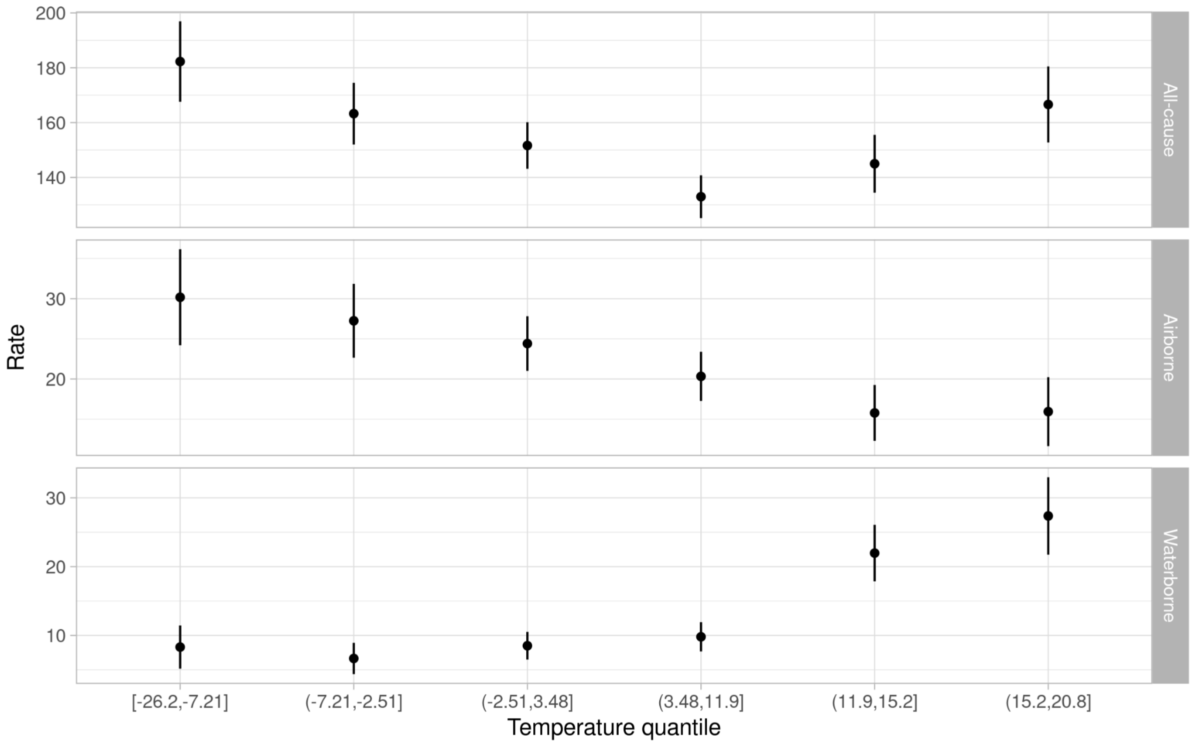

***Figure 2**: All-cause and cause-specific infant mortality rate by temperature percentiles (10th,25th,50th,75th,90th). CI based on Poisson standard errors.*

The regression model (Figure 3), adjusting for seasonality and the long-term trend, shows that temperature exposures over +5 ℃ in the past 14 days, was associated with higher mortality. At the 99th percentile temperature exposure of +19 ℃, the incidence rate ratio (IRR) was 1.47 (CI 1.24-1.76) compared to the reference at the minimum mortality temperature at +5 ℃. Temperature exposures below +5 ℃ did not show any significant association with IRR of infant mortality, IRR was 1.17 at -17 ℃ (CI 0.95-1.45).

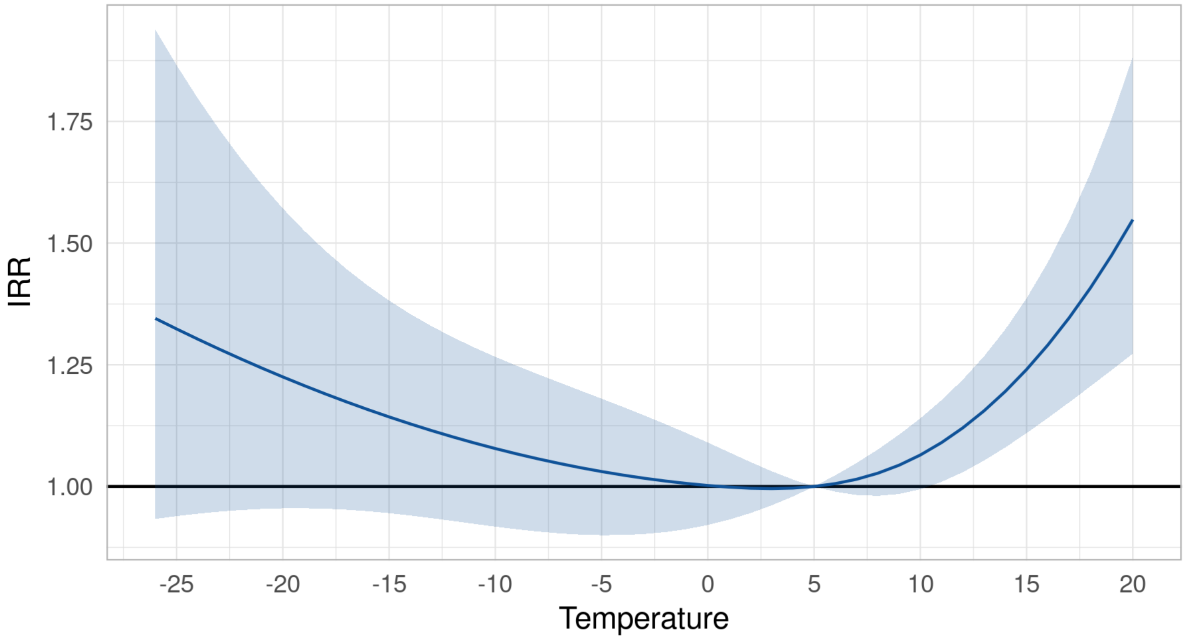

Figure 3*: Incidence rate ratios (IRR) with 95 percent confidence intervals for all-cause infant mortality by past 14 days ambient temperature exposure. .*

Breaking down the association by causes, the adjusted Poisson regression models show significantly different patterns (Figure 4). AID mortality showed no association with ambient temperature exposures over the past 14 days. WFID mortality had a strong positive association with temperatures above +7 °C, with an IRR of 5.52 (CI 3.13-9.74) at +20°C (the 99th percentile temperature exposure), and no association with cold temperatures. Mortality related to other causes who a slight increase in IRR with both heat and cold temperatures compared to the MMT at +1°C, at -17°C (1st percentile) IRR was 1.25 (CI 1.01-1.55) and at +19°C (99th percentile) IRR was 1.40 (CI 1.10-1.77).

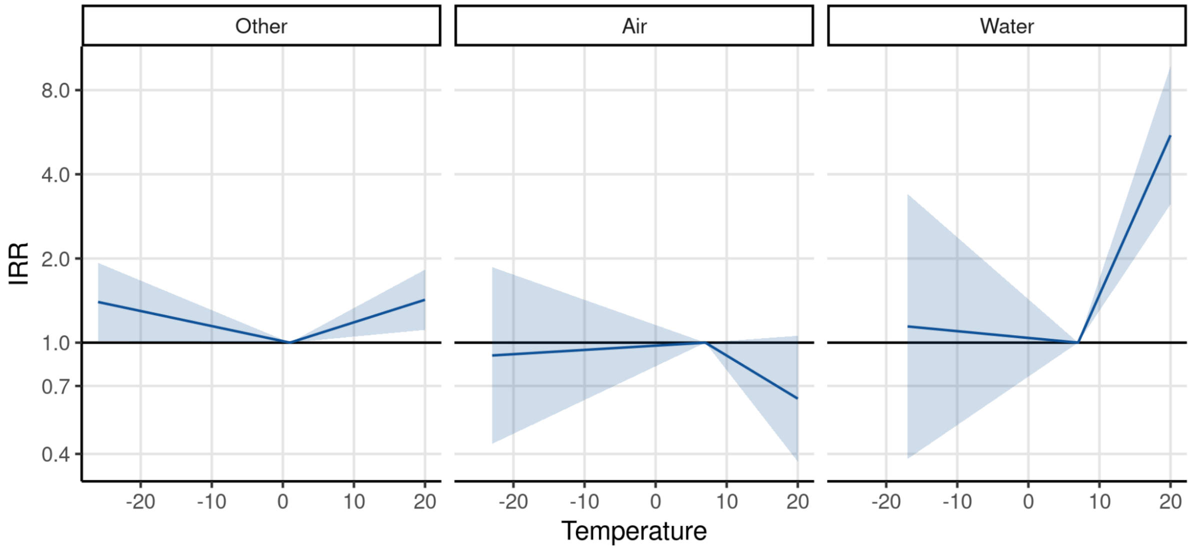

Figure 4: *Incidence rate ratios (IRR) with 95 percent confidence intervals for cause-specific infant mortality by past 14 days ambient temperature exposures. IRR on a log scale.*

The associations were not uniform across lag time. Figure 5 shows IRR of cause-specific infant mortality at -20 ℃ and +20 ℃ (the 1st 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. Heat temperature (+20 ℃) was associated with increased IRR of WFID mortality after 10 days to 1.19 (CI 1.06 - 1.33). Mortality due to other causes did show a small positive association to heat two days after exposure, with an IRR of 1.13 (CI 1.01-1.27).

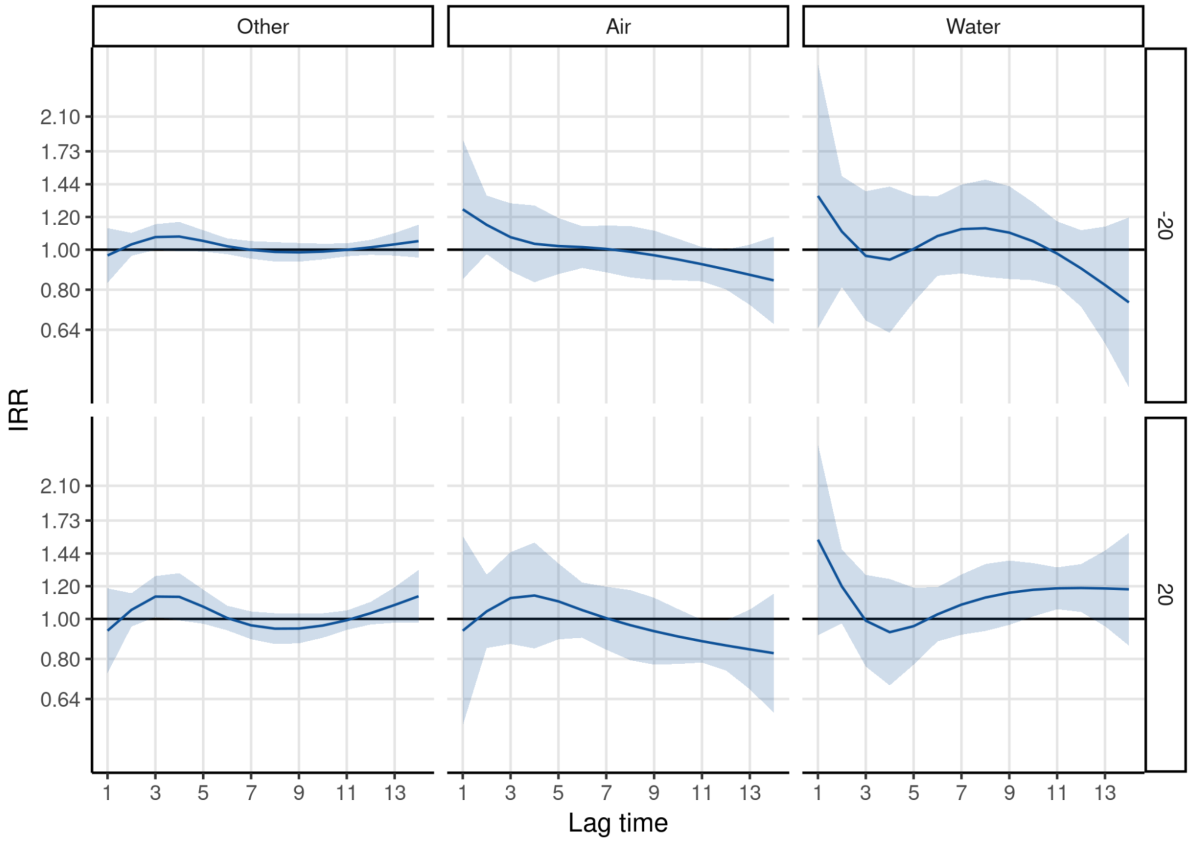

***Figure 5:** Lagged incidence rate ratios (IRR) with 95 percent confidence intervals for cause-specific infant mortality at -20 and +20 ℃ ambient temperature exposures over the past 1-14 days. IRR on a log scale.*

Seasonality

In terms of seasonality, all-cause IM was highest in the winter (December-February) followed by the summer months (June-August), see Table 1. AID and WFID mortality had opposite patterns. AID mortality peaked in the winter and was lowest in the summer, while WFID mortality peaked in the summer and reached a low in the winter.

The Poisson regression models confirm the seasonal patterns. IRR and CI were calculated compared to the day of the year with the observed minimal cause-specific mortality. The unadjusted IRR peaked for all-cause mortality in the winter months January-February and in the summer months July-August. However, adjusting for temperature exposures the IRR reaches a low point in the summer season and the IRR peak in the winter months increase (Fig 6).

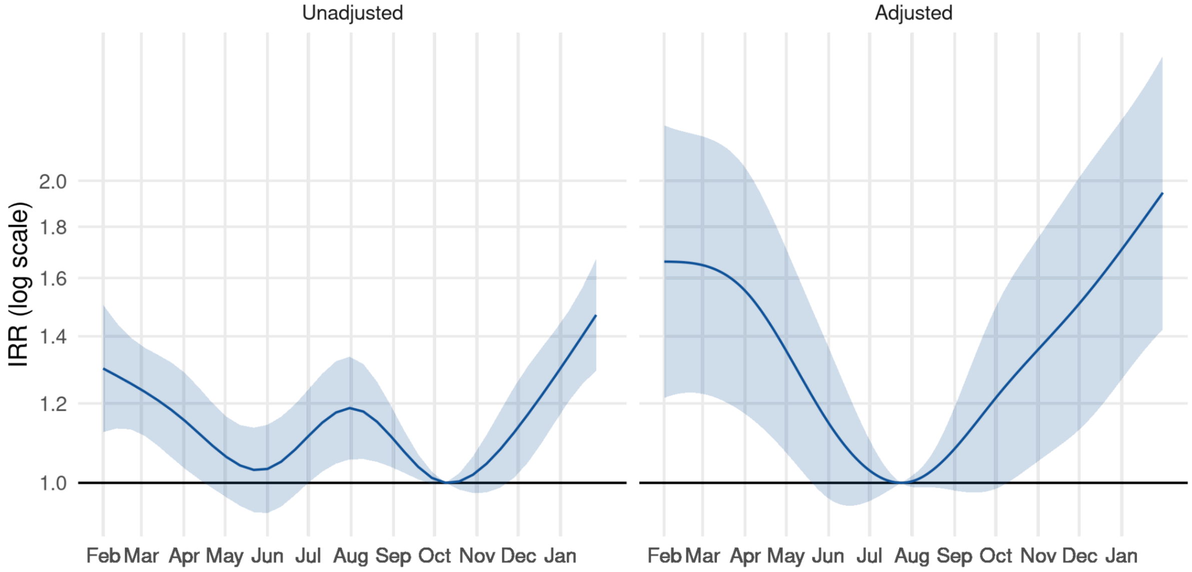

Figure 6: *Incidence rate ratios (IRR) with 95 percent confidence intervals for infant mortality by day of the year from February 1st to January 31st. IRR on a log scale. Reference day set at day of the year with minimum mortality. Adjusted for year and temperature.*

The seasonal models show a strong association with cause-specific mortality (Figure 7). AID mortality followed a strong U-shaped seasonal pattern with minimum mortality in summer (Aug) and a peak in winter (dec-mar). The opposite, bell-shaped, pattern was found for WFID mortality, where mortality peaked in summer and reached a minimum in the winter (Feb). The seasonal association with other causes followed the same patterns as all-cause mortality.

Adjusting for day-of temperature and a seven-day average lagged temperature, the seasonal patterns increased for AID mortality and other causes, while it decreased for WFID mortality. The model showed no significant seasonal relationship to WFID mortality, results that suggest that the seasonal pattern was primarily an effect of temperature exposure rather than seasonal exposure.

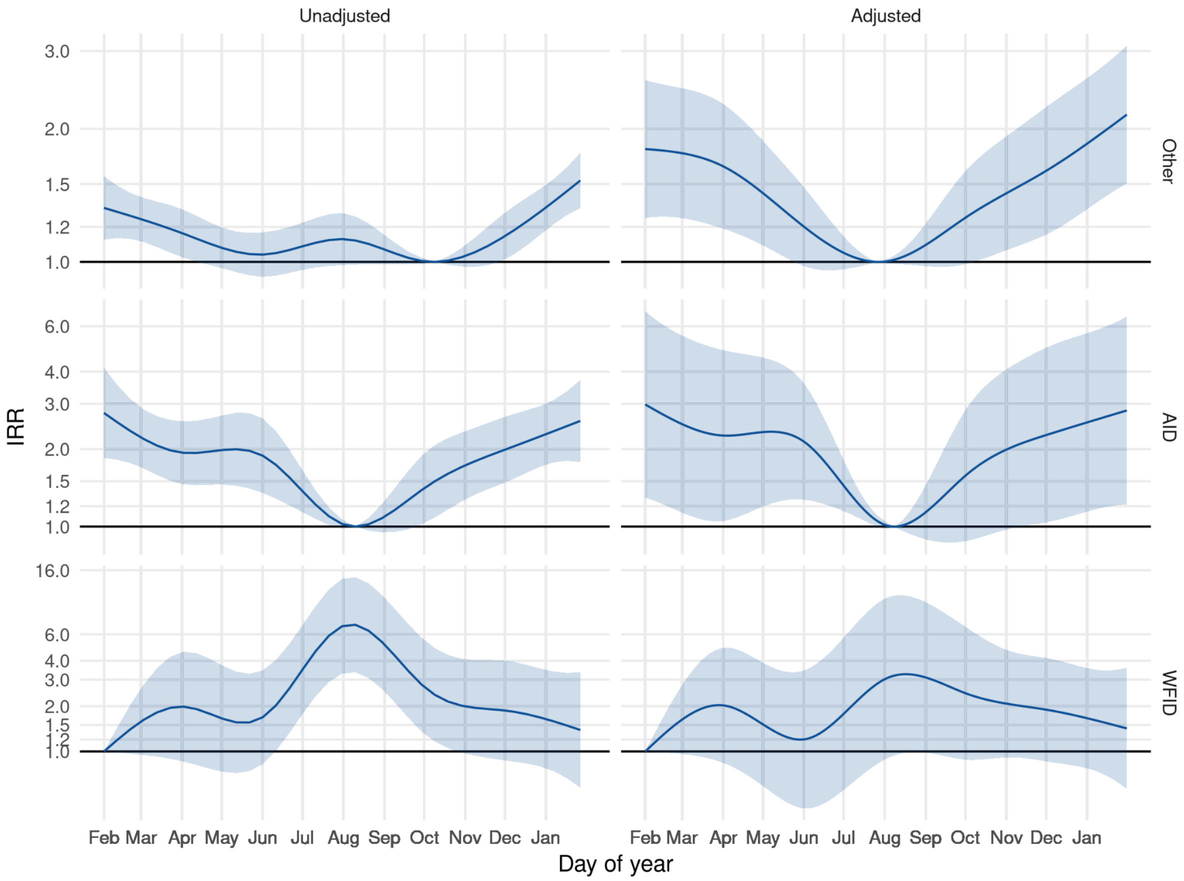

Figure 7: *Incidence rate ratios (IRR) with 95 percent confidence intervals for cause-specific mortality among infants by day of the year from February 1st to January 31st. IRR on a log scale. Reference day set at day of the year with minimum mortality. Adjusted for year and temperature.*

Sensitivity analysis

We have performed additional sensitivity analyses. Causes of death were not similarly distributed by age (see Figure A4), and very few infectious diseases deaths were recorded during the neonatal period - the first 14 days. Sensitivity analysis limiting the sample to post-neonatal mortality show similar results (Figure A5, and A6). Given the large proportion of unknown causes-of-death we performed analyses grouping these deaths into another category different from “other causes”, these analyses showed that unknown causes of death and deaths due to known other causes have similar seasonal and temperature patterns (Figure A7 and A8).

Discussion

Infant vulnerability to infectious diseases is associated with climate variability, such as ambient temperature and seasonality (Hedlund et al., 2014; Junkka et al., 2021; Karlsson et al., 2021). We provide new evidence on the association between ID mortality among infants and climate variability for a 19th-century Swedish population living in a cold climate with high mortality. Specifically, we studied the association between ambient temperature and seasonality, all-cause, water- and foodborne infectious disease, and airborne infectious disease infant mortality, between 1868 and 1892.

Overall, we found a strong association between high ambient temperature and all-cause mortality and WFID mortality, and no association with cold temperatures. AID mortality did not show any association with temperature while adjusting for time trends and seasonality. Adjusting for temperature exposure, we found a strong seasonal pattern for all-cause mortality and AID mortality, reaching a low in summer and peaking in winter. Although the opposite pattern was found for WFID mortality, with a peak in the summer, after adjusting for temperature exposures the association was significantly reduced, showing no difference by day of the year.

Infant deaths were observed at 14 days lagged temperatures down to -33.9 ℃, and the winter months had an average monthly minimum of below -15 ℃ during the study period. Cold temperatures have been shown to affect infant survival due to hypothermia or AID (Derosas, 2009; Xu et al., 2012). Although ID mortality was not related to cold temperatures in our study, mortality due to other causes was. At -17 °C the IRR of death was 1.25 (CI 1.01-1.55), compared to a temperature of +1 °C with a peak in mortality three days after exposure. Possibly, the peak 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). Infants in 19th century northern Sweden are vulnerable to very low temperatures, in particular in the first weeks of life (Junkka et al., 2021; Schumann et al., 2019). Living standards (such as indoor heating, nutrition and income) were low and infant mortality was high in late 19th-century Sundsvall, and thus the ability of families to protect infants against cold were limited (Edvinsson, 1992). Yet, we found no association between AID mortality and cold temperatures, suggesting that cold temperatures exposure in itself was not enough to drive high AID mortality among infants.

During our study period, the monthly average maximum temperatures in the summer months were above +19 ℃, with infant deaths observed at 14 days lagged temperatures at a maximum of +27.8 ℃. The reported heat-related high infant mortality in historical populations has been suggested to be related to gastrointestinal diseases due to the contamination of food and water (Ekamper et al., 2009; Junkka et al., 2021). Our findings support these conclusions, WFID were strongly related to heat exposure. As in studies of modern populations, heat exposures had a lagged effect on AID mortality, peaking after about 10 days (Xu et al., 2014). The results also show heightened IRR (although with high uncertainty) of AID mortality in the first few days, which could be related to 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 despite that death due to other causes showed a positive association with temperatures above + 1 ℃ there were no significant lagged patterns.

We found that seasonality had a significant association with AID mortality, peaking in the January-February, an association that strengthened when adjusting for lagged temperature exposures. These findings suggest that AID mortality was primarily driven by seasonality rather than low temperatures. Possibly, changes in human behaviours during the winter, such as indoor activities, increased exposure to viral infections (Moriyama et al., 2020). Increased indoor pollution during the winter due to the usage of wood-burning stoves as heating (Bergman, 2010) could also have increased the risk of viral infections (Ciencewicki & 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 outdoor activities during the summer such as proximity to animals or eating fresh food increased WFID exposures (Tymicki, 2009). Although 19th century Sundsvall experienced rapid urbanisation (Edvinsson, 1992) the city was relatively small compared to many European cities where the urban summer peak has been observed in contemporary populations (Huck, 1994; Tymicki, 2009). Thus, the absence of 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 diseases mortality and its relationship to seasonality and temperature in a 19th-century population. Without cause-specific data, previous studies have only been able to speculate on the mechanisms driving the climate variability - infant mortality relationship, which we have been able to address (Ekamper et al., 2009; Huck, 1994; Junkka et al., 2021; Schumann et al., 2019; Tymicki, 2009). Yet, historical cause-specific data on infectious diseases has 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 at large simpler to differentiate than individual conditions, using the ICD10h coding system, tailored for historical cause-of-death data (Janssens, 2021).

We limited our measurements of climate variability to seasonality and ambient temperature, thereby overlooking the importance of humidity. Especially for WFID, humidity in combination is an important factor for exposure and mortality (Martinez, 2018; Pappas et al., 2008). Our study also lacks data on indoor environmental conditions such as air pollution, thus we are not able to disentangle different forms of seasonal variations in behaviour and environment which could drive the seasonal patterns.

Conclusions

Our findings showed that both seasonality and high temperatures were important for infants' vulnerability to infectious diseases. We found that airborne infectious disease mortality was not related to cold temperatures but rather to seasonality, and that summer mortality peaks due to water- and foodborne infections were associated with high temperatures and not with seasonality. Although further research is necessary, the increased vulnerability to infectious diseases of infants at high temperatures is a significant future risk, given the expected global warming in the coming centuries.

Appendix

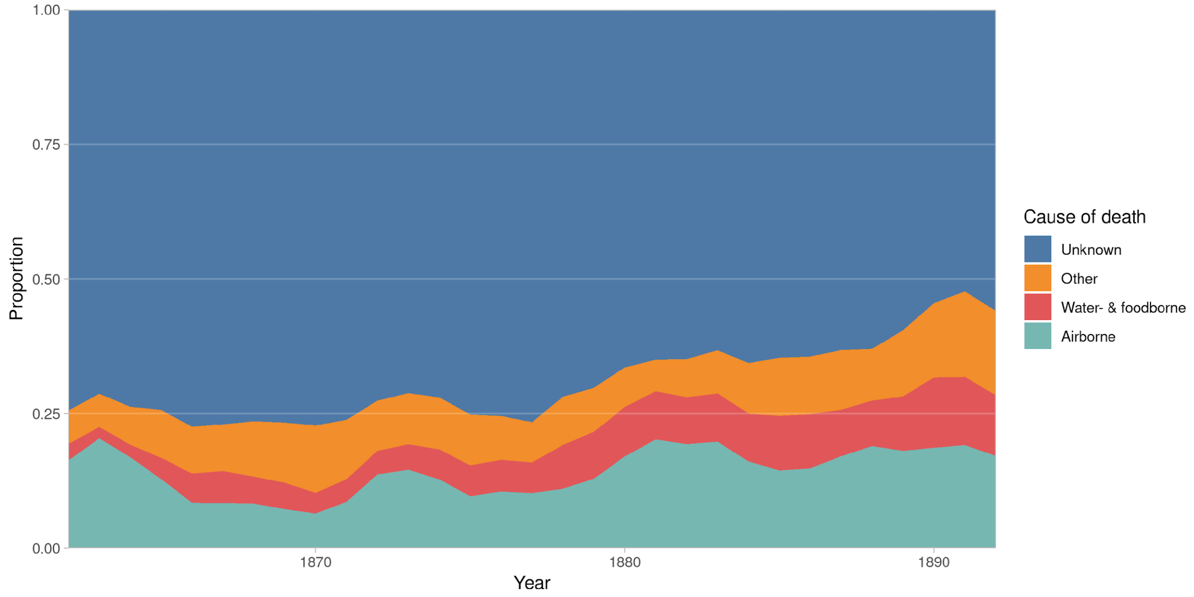

Figure A1: *Distribution of cause of death by year, 1860-1892*

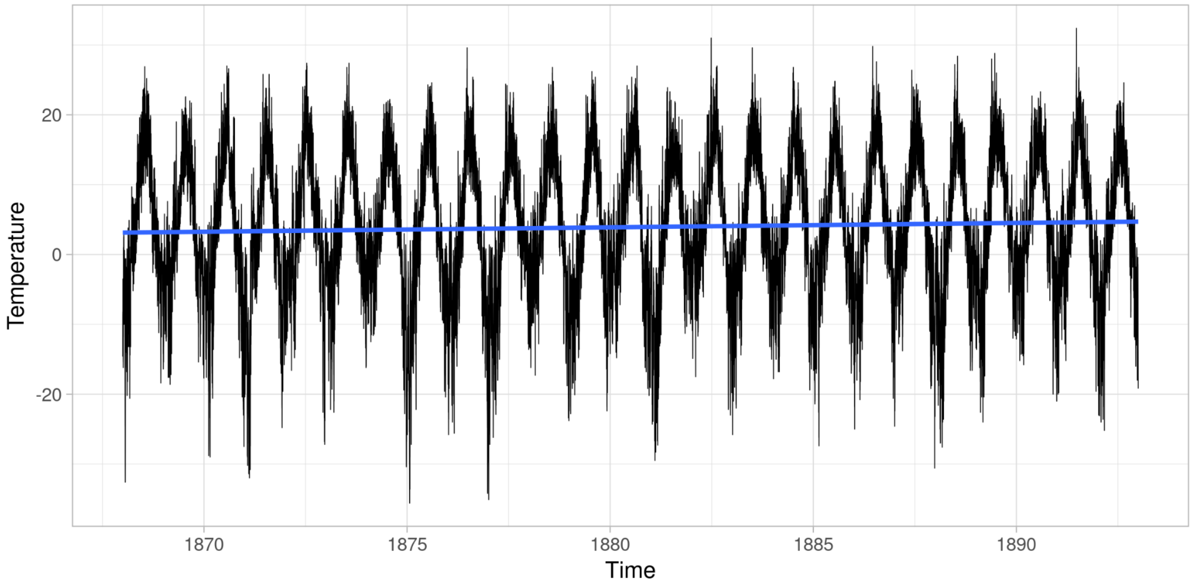

Figure A2*: Thrice daily temperature at 7:00, 13:00 and 20:00 at Härnösand weather station, 1868-1892*

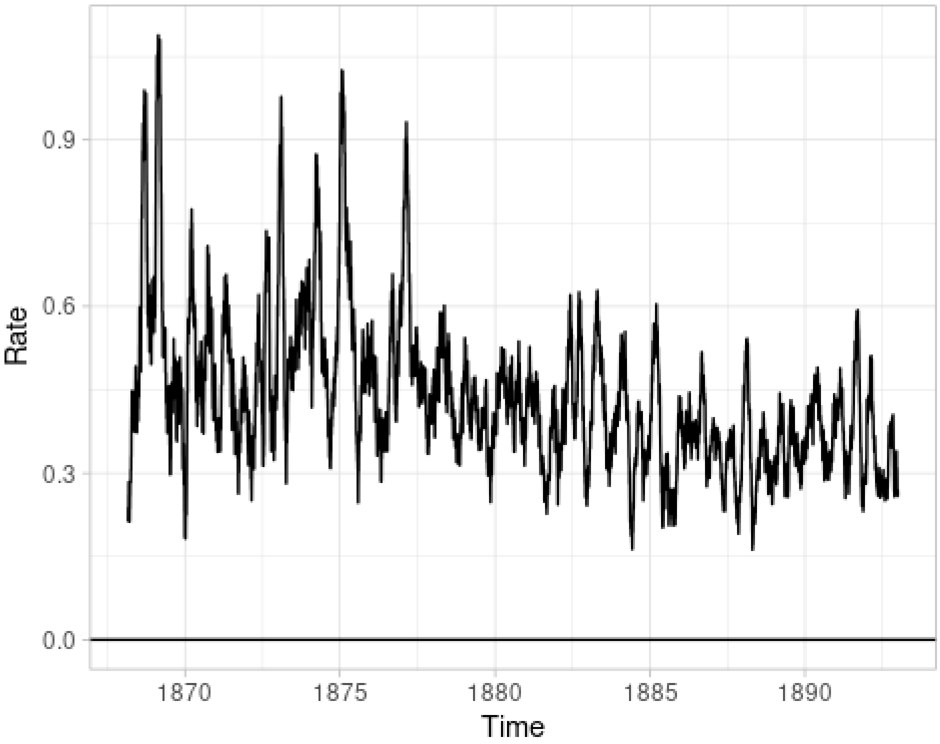

***Figure A3:** Daily mortality rate 1866-1896, averaged over the past 60 days.*

Table A1: Incidence rate ratios (IRR) with 95% confidence intervals (CI) for infant death by 14 day average temperature exposure at percentiles, compared to minimum mortality temperature (AID = +7 ℃, WFID = +7 ℃, Other = +1 ℃)

Cause	Percentile	Temperature	RR	CI low	CI high
AID	1st	-17	0.919	0.514	1.645
	10th	-8	0.949	0.659	1.365
	25th	-4	0.962	0.737	1.256
	50th	+1	0.979	0.847	1.132
	75th	+9	0.931	0.860	1.009
	90th	+14	0.780	0.590	1.031
	99th	+18	0.677	0.437	1.049
WFID	1st	-12	1.111	0.469	2.636
	10th	-5	1.069	0.620	1.845
	25th	+1	1.034	0.787	1.358
	50th	+11	1.691	1.420	2.014
	75th	+15	2.861	2.017	4.058
	90th	+17	3.720	2.403	5.759
	99th	+20	5.518	3.127	9.738
Other	1st	-17	1.250	1.008	1.549
	10th	-8	1.118	1.004	1.245
	25th	-3	1.051	1.002	1.102
	50th	+2	1.019	1.006	1.032
	75th	+12	1.227	1.063	1.416
	90th	+15	1.297	1.081	1.557
	99th	+19	1.397	1.105	1.767

Table A2: Incidence rate ratios (IRR) with 95% confidence intervals (CI) for infant death by lag in days at +20 and -20 ℃ (the 1st and 99th daily temperature exposures), compared to minimum mortality temperature (AID = +7 ℃, WFID = +7 ℃, Other = +1 ℃)

		Other		AID		WFID	
Tempera.	Lag	IRR	CI	IRR	CI	IRR	CI
-20	0	0.969	0.832 - 1.128	1.252	0.848 - 1.849	1.350	0.646 - 2.821
	1	1.030	0.966 - 1.098	1.148	0.975 - 1.352	1.106	0.812 - 1.507
	2	1.073	0.999 - 1.153	1.072	0.887 - 1.295	0.966	0.674 - 1.385
	3	1.076	0.992 - 1.167	1.033	0.834 - 1.280	0.946	0.630 - 1.421
	4	1.050	0.989 - 1.115	1.020	0.873 - 1.193	1.004	0.746 - 1.352
	5	1.019	0.975 - 1.065	1.014	0.903 - 1.140	1.079	0.865 - 1.347
	6	0.999	0.951 - 1.049	1.004	0.881 - 1.144	1.122	0.876 - 1.437
	7	0.988	0.936 - 1.043	0.989	0.857 - 1.141	1.127	0.859 - 1.478
	8	0.985	0.935 - 1.038	0.969	0.845 - 1.112	1.100	0.849 - 1.424
	9	0.990	0.947 - 1.034	0.947	0.843 - 1.063	1.047	0.843 - 1.301
	10	0.999	0.964 - 1.036	0.922	0.838 - 1.015	0.978	0.818 - 1.171
	11	1.013	0.972 - 1.056	0.896	0.802 - 1.001	0.901	0.728 - 1.115
	12	1.030	0.967 - 1.097	0.869	0.735 - 1.028	0.821	0.593 - 1.137
	13	1.049	0.957 - 1.150	0.843	0.660 - 1.076	0.745	0.464 - 1.197
+20	0	0.936	0.739 - 1.186	0.936	0.552 - 1.587	1.555	0.913 - 2.649
	1	1.052	0.959 - 1.154	1.043	0.850 - 1.279	1.198	0.975 - 1.472
	2	1.133	1.010 - 1.270	1.123	0.870 - 1.451	0.990	0.767 - 1.278
	3	1.131	0.991 - 1.290	1.139	0.848 - 1.530	0.929	0.690 - 1.249
	4	1.070	0.973 - 1.177	1.103	0.892 - 1.363	0.960	0.775 - 1.190
	5	1.005	0.938 - 1.076	1.050	0.900 - 1.224	1.026	0.882 - 1.193
	6	0.965	0.893 - 1.042	1.004	0.842 - 1.197	1.083	0.916 - 1.281
	7	0.947	0.869 - 1.032	0.966	0.794 - 1.174	1.127	0.935 - 1.358
	8	0.948	0.873 - 1.029	0.934	0.775 - 1.125	1.157	0.967 - 1.384
	9	0.964	0.900 - 1.032	0.907	0.778 - 1.056	1.176	1.014 - 1.365
	10	0.993	0.940 - 1.048	0.883	0.784 - 0.995	1.186	1.055 - 1.332

11	1.032	0.970 - 1.099	0.863	0.751 - 0.991	1.188	1.040 - 1.357
12	1.081	0.979 - 1.193	0.844	0.676 - 1.054	1.185	0.960 - 1.463
13	1.135	0.980 - 1.313	0.826	0.593 - 1.151	1.180	0.862 - 1.614

Table A3: Incidence rate ratios (IRR) with 95% confidence intervals (CI) for infant death by month (first day in month) compared to the minimum mortality day (AID = July 8th, WFID = Jan 1th, Other = June 26th). Adjusted for seasonality and timetrend.

	AID		WFID		Other	
Month	IRR	CI	IRR	CI	IRR	CI
Feb	2.981	1.297 - 6.854	1.000	1.000 - 1.000	1.801	1.257 - 2.579
Mar	2.498	1.132 - 5.511	1.631	0.961 - 2.769	1.762	1.255 - 2.472
Apr	2.257	1.052 - 4.844	2.022	0.840 - 4.871	1.645	1.189 - 2.276
May	2.329	1.196 - 4.534	1.513	0.589 - 3.885	1.431	1.089 - 1.878
Jun	2.137	1.271 - 3.593	1.203	0.419 - 3.453	1.201	0.978 - 1.475
Jul	1.442	1.148 - 1.810	1.778	0.555 - 5.699	1.048	0.962 - 1.142
Aug	1.016	0.981 - 1.051	3.018	0.883 - 10.314	1.002	0.993 - 1.011
Sep	1.137	0.893 - 1.449	3.100	0.981 - 9.792	1.092	0.978 - 1.220
Oct	1.577	0.878 - 2.832	2.438	0.882 - 6.735	1.259	0.988 - 1.604
Nov	1.991	0.973 - 4.075	2.077	0.912 - 4.733	1.430	1.063 - 1.924
Dec	2.267	1.039 - 4.950	1.895	0.859 - 4.177	1.607	1.153 - 2.240
Jan	2.539	1.143 - 5.642	1.662	0.772 - 3.579	1.852	1.316 - 2.606

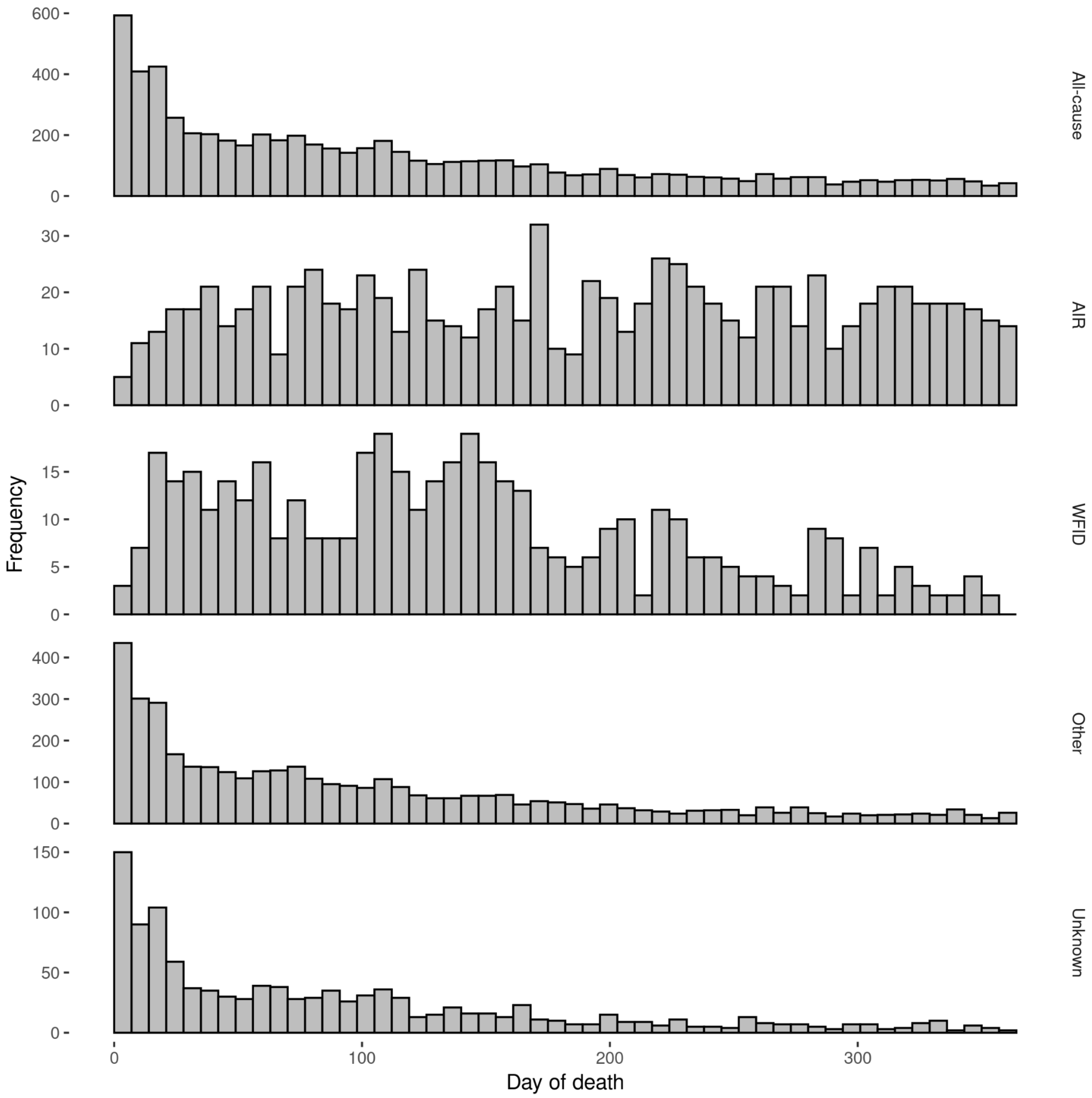

Figure A4: *Distribution of day of death by Cause-of-death.*

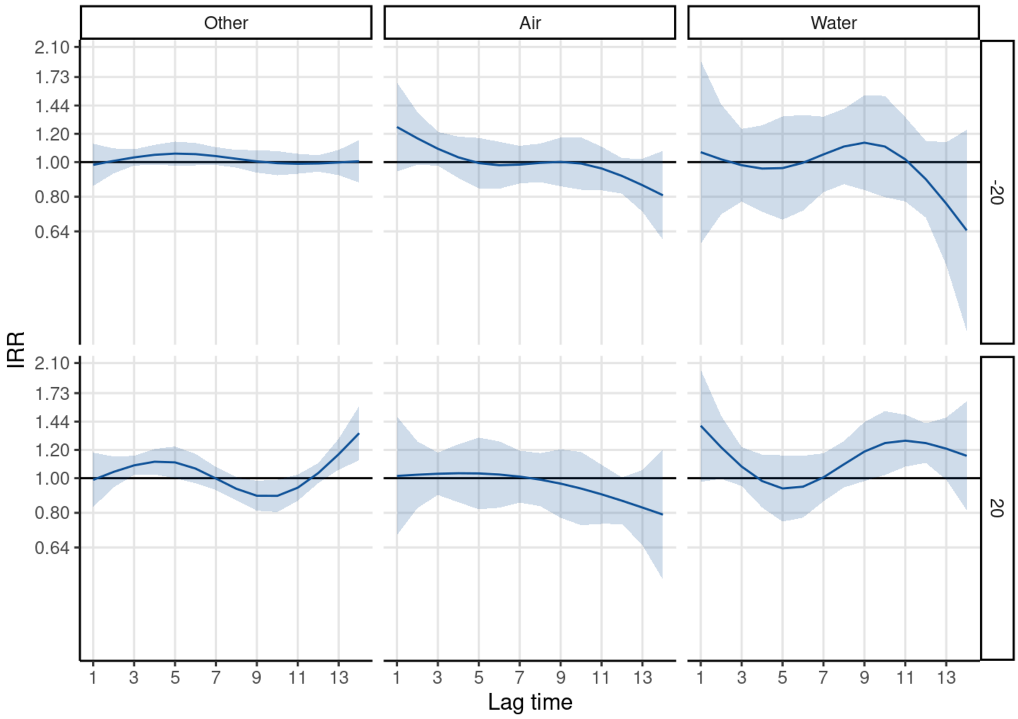

Figure A5: *Incidence rate ratios (IRR) with 95 percent confidence intervals for cause-specific post-neonatal mortality by past 14 days ambient temperature exposures. IRR on a log scale.*

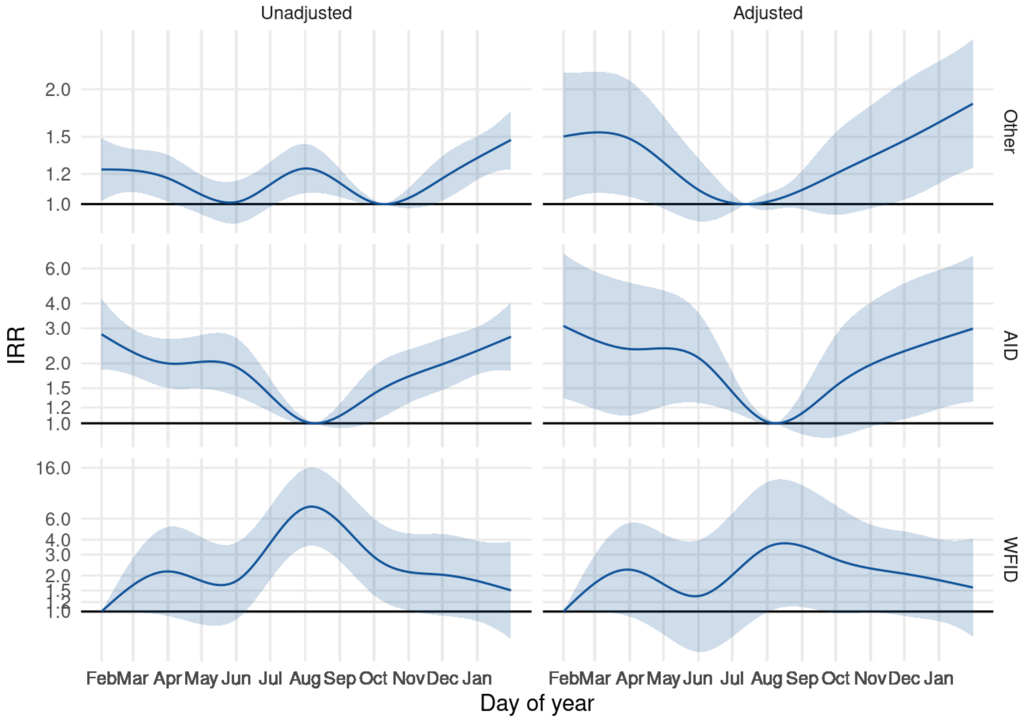

Figure A6*: Incidence rate ratios (IRR) with 95 percent confidence intervals for cause-specific post-neonatal mortality by day of year from February 1st to January 31st. IRR* *on a log scale. Reference day set at day of year with minimum mortality. Adjusted for year and temperature.*

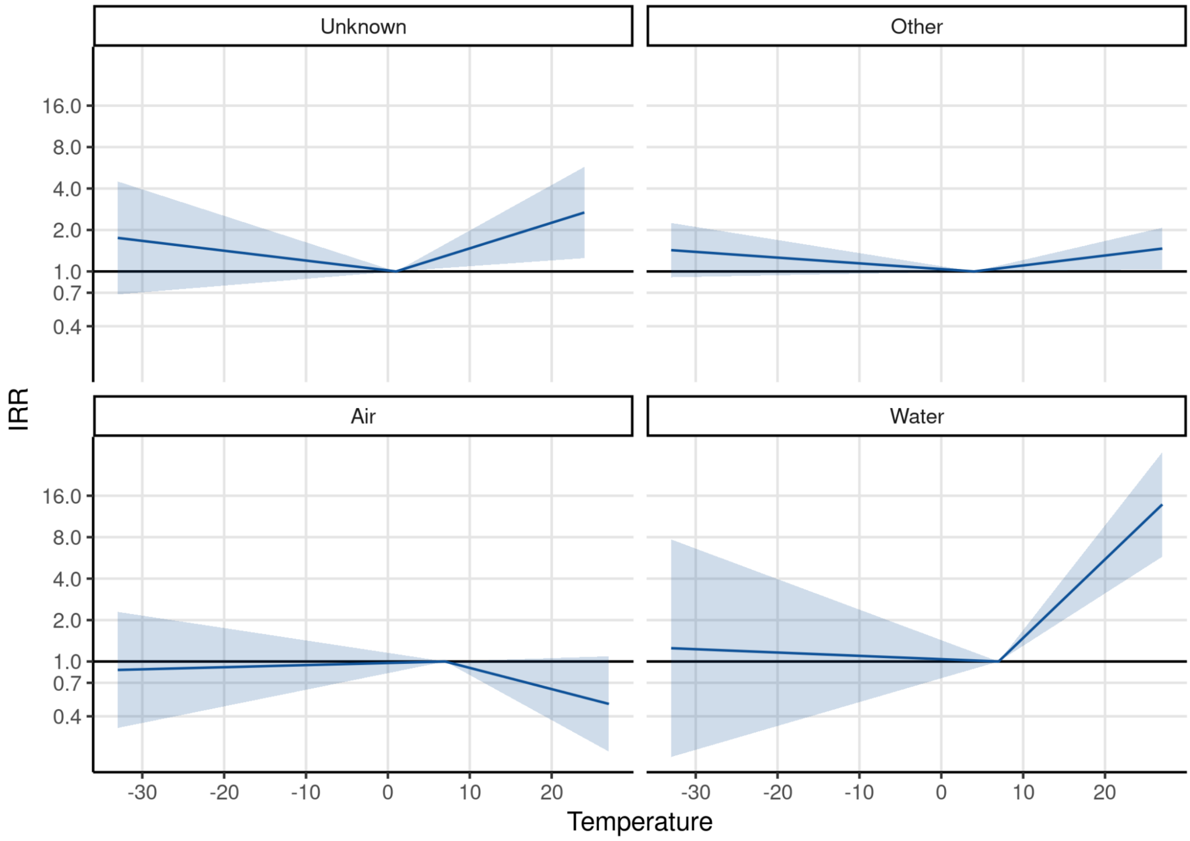

Figure A7: *Incidence rate ratios (IRR) with 95 percent confidence intervals for cause-specific infant mortality by past 14 days ambient temperature exposures, including Unknown causes of death. IRR on a log scale.*

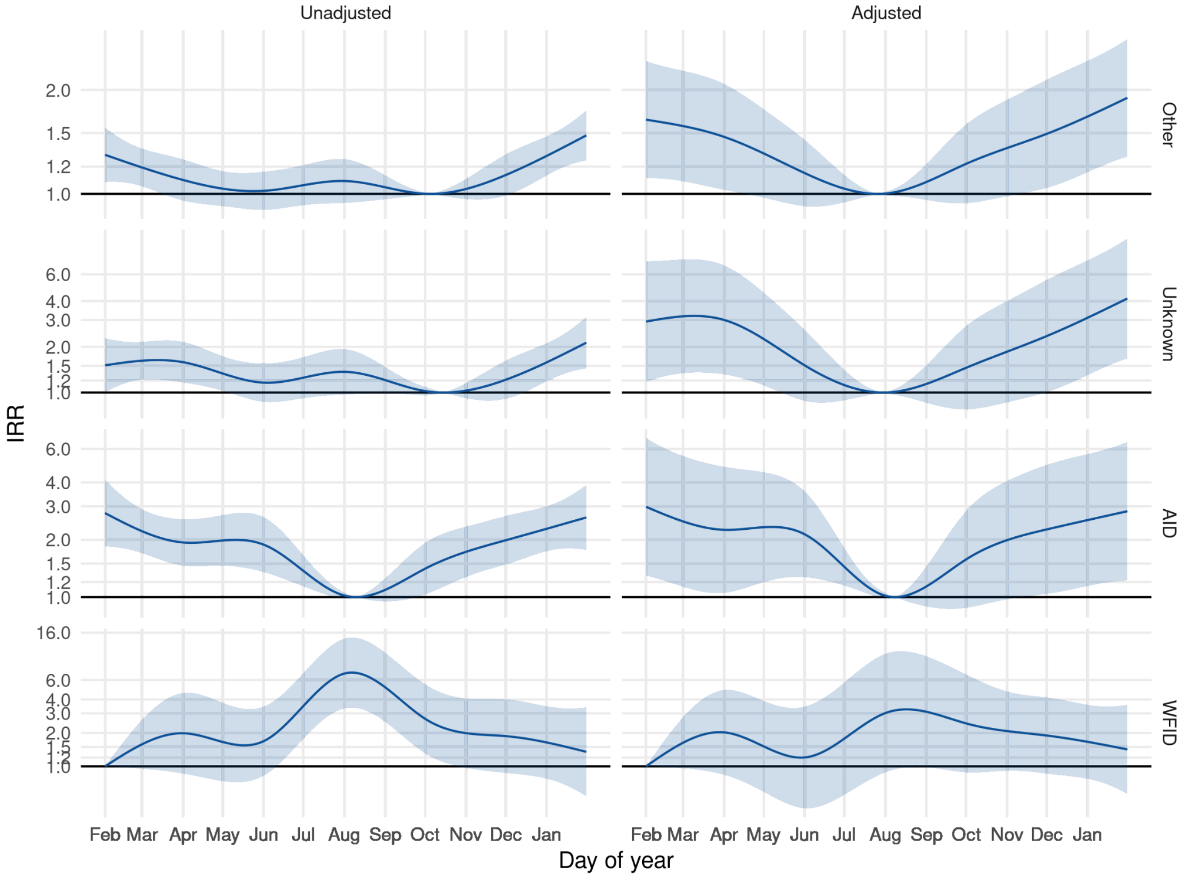

Figure A8: *Incidence rate ratios (IRR) with 95 percent confidence intervals for cause-specific infant mortality by day of year from February 1st to January 31st. IRR on a log scale. Reference day set at day of year with minimum mortality, including Unknown causes of death. Adjusted for year and temperature.*

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