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Research Article

**On the association between weather variability
and total and cause-specific mortality before and
during industrialization in Sweden**

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On the association between weather variability and total and cause-specific mortality before and during industrialization in Sweden

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Abstract

BACKGROUND

While there is ample evidence for health risks associated with heat and other extreme weather events today, little is known about the impact of weather patterns on population health in preindustrial societies.

OBJECTIVE

To investigate the impact of weather patterns on population health in Sweden before and during industrialization.

METHODS

We obtained records of monthly mortality and of monthly mean temperatures and precipitation for Skellefteå parish, northern Sweden, for the period 1800–1950. The associations between monthly total mortality, as well as monthly mortality due to infectious and cardiovascular diseases, and monthly mean temperature and cumulative precipitation were modelled using a time series approach for three separate periods, 1800–1859, 1860–1909, and 1910–1950.

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RESULTS

We found higher temperatures and higher amounts of precipitation to be associated with lower mortality both in the medium term (same month and two-months lag) and in the long run (lag of six months up to a year). Similar patterns were found for mortality due to infectious and cardiovascular diseases. Furthermore, the effect of temperature and precipitation decreased over time.

CONCLUSIONS

Higher temperature and precipitation amounts were associated with reduced death counts with a lag of up to 12 months. The decreased effect over time may be due to improvements in nutritional status, decreased infant deaths, and other changes in society that occurred in the course of the demographic and epidemiological transition.

CONTRIBUTION

The study contributes to a better understanding of the complex relationship between weather and mortality and, in particular, historical weather-related mortality.

1. Background

Following the Little Ice Age between the 14th and 19th centuries (Ljungkvist 2009), an increase in global average temperatures has been observed (IPCC 2013). Since the end of the 20th century, more frequent heat waves have been reported and these put stress on the elderly and sick especially, as they suffer most from heat-sensitive health concerns, including cardiovascular and respiratory diseases (Smith et al. 2013). Apart from the impact of high temperature on morbidity and mortality, the effects of cold have also been shown (Mäkinen et al. 2009; Oudin Åström et al. 2013; Näyhä et al. 2011).

Infectious diseases are related to heavy rainfall (waterborne diseases), to higher temperatures (e.g., vector-borne diseases), and to lower temperatures (e.g., influenza) (McMichael 2012; Portier et al. 2010; Semenza and Menne 2009). But also for non-communicable diseases, in particular cardiovascular diseases, including stroke and myocardial infarction, higher mortality and morbidity rates have been reported following temperature extremes at both ends of the spectrum (Portier et al. 2010; Rocklöv, Ebi, and Forsberg 2011; Zacharias, Koppe, and Mucke 2015). While there is ample evidence for health risks associated with heat and other extreme weather events today, little is known about the impact of weather patterns on population health in preindustrial societies.

Human well-being depends to a large degree on the availability of basic environmental resources, such as agricultural yields and clean water, which are influenced by climate (McMichael 2012). Most existing studies on the impact of preindustrial climate on human health focus on the role of harvest outcomes. For example, in the 17th century, at the height of the Little Ice Age, considerably lower annual temperatures led to harvest failures and food shortages. These in turn caused famines and epidemics, as well as armed conflicts, throughout Europe (Ljungkvist 2009; McMichael 2012; Parker 2013; Zhang et al. 2011).

However, there is less evidence about the role of weather patterns or weather extremes on infectious disease outbreaks beyond the pathway of harvest and food supply. Catalano, Bruckner, and Smith (2008) showed that around 1900 in Scandinavia the gestational survival of male fetuses increased when the annual temperature was higher, while longevity of males decreased (due to natural selection of less fit male babies). According to our earlier research, in Sweden between 1749 and 1859 annual average temperature and precipitation had little impact on annual death counts. But while higher springtime temperatures and precipitation decreased annual mortality, high levels of rain in autumn led to higher annual mortality (Rocklöv et al. 2014; Schumann et al. 2013). There was also evidence that older children were most vulnerable to an unfavourable climate, while there was a less pronounced impact on infants (Rocklöv et al. 2014). These findings applied to a period of slowly decreasing mortality trends before the onset of industrialization in Sweden. Much insight is to be gained from studying changes in climate impact during the economic and sociodemographic transition after 1860. Additionally, we need to know more about the sensitivity to weather conditions of different diseases. Although little reliable information on specific causes of death is available for previous centuries, analyses of disease groups – infectious and cardiovascular diseases – are likely to be more valid. These should increase our knowledge about the vulnerability of preindustrial populations to extreme weather.

The aim of the present study was to analyse the impact of monthly weather on monthly total and cause-specific mortality in Skellefteå, northern Sweden, between 1800 and 1950. We also aimed to investigate whether these impacts changed in the course of demographic and socioeconomic development.

2. Material and methods

2.1 Population data

The population data for the Skellefteå region, located on the Gulf of Bothnia coast in the north of Sweden, comes from the POPLINK dataset at the Demographic Database (DDB), Umeå University. The POPLINK dataset is based on long-term parish records (registers of births, deaths, and migration, and population registers – i.e., clerical surveys (*husförhörslängder*) until the 1890s and parish registers thereafter). In this study, we used data for the period 1800–1950. The population was continuously recorded in the clerical surveys and parish registers, and the individual records have been linked, allowing us to reconstruct the biographies of people from a demographic and social perspective as long as they remained in the region (Edvinsson 2000; Wisselgren et al. 2014).

The main variables considered in this study were date and cause of death. Individual deaths were included in the analyses if the person had died within the study region and if information on month and year of death was available.

Cause of death was determined and recorded by the parish ministers from the mid-18th century in Sweden. For the first decades of the 19th century a large proportion of records have missing data on cause of death, and infant deaths were under-reported. There is probably also some under-reporting of deaths of young children, but not for other age groups. From 1911 it became mandatory to report the cause of death for all deceased persons. The validity of the given causes was then controlled by doctors. Thus the most reliable data on cause of death related to the last period in our study. Reported causes of death were assigned an ICD code at the DDB. We grouped diseases into two main categories: a) infectious (including respiratory) diseases and b) cardiovascular diseases. Specific analyses were conducted for total mortality, infectious disease mortality, and cardiovascular disease mortality respectively.

2.2 Climate data

Regular recordings of daily temperature and precipitation started in Sweden about 1860. As a proxy for Skellefteå weather, we used monthly data on precipitation and temperature from Umeå, a town 110 kilometres south of Skellefteå. For the period 1800–1859, early temperature recordings from Uppsala (roughly 600 kilometres south) and from Tornedalen (roughly 230 kilometres north) were used for the reconstruction of Umeå monthly temperature (see details in Rocklöv et al. 2014). Weather data was obtained from the Swedish Meteorological and Hydrological Institute (SMHI), with the

exception of Tornedalen data, which was taken from Klingbjer and Moberg (2003). Uppsala temperature measurements have been homogenized and validated by Moberg and Bergström (Moberg and Bergström 1997; Bergström and Moberg 2002).

The precipitation record for Umeå for the period 1748–1859 was reconstructed using bootstrapped linear regression modelling with precipitation observations from Uppsala. Cross-validation was applied to test the predictive capacity of this approach for 1860–1950, when observations were available from both locations. Modelled precipitation totals for Umeå for 1860–1950 were all within 5% of actual observations across the ensemble of regression models, with the median model differing from observations by less than 4 centimetres over the entire record. The average correlation coefficient between modelled and observed monthly precipitation time series ranged from 0.59 to 0.62, with a median of 0.61 (see Supplementary Figure 1). More details on the precipitation reconstruction method are available in the supplementary material.

Temperature values are indicated in degrees Celsius (°C) and precipitation in centimetres (cm).

Exact and reliable estimates of local harvest results are not available throughout our study period. In our analysis, we have chosen to identify years characterized by harvest failures. Harvest estimates and general descriptions of harvests were reported from the 18th century by parish ministers and later by county governors based on local reports (Hellstenius 1871). They can be used only as crude measures of yearly harvest results, but they clearly indicate years with severe crop failure. This information has been evaluated and validated by other sources, such as minutes from local parish board meetings and data on relief consignments from the government. For the period 1870 onwards, we have used data on harvests at local level from *hushållningssällskapet*, local organizations for the promotion of agriculture (Statistics Sweden 1926).

2.3 Statistical methods

The associations of monthly mortality with monthly mean temperature and monthly cumulative precipitation respectively were modelled using generalized additive regression assuming an overdispersed Poisson distribution of monthly death counts. To model the association between weather variables and mortality we used a three-month moving average (MA) of monthly temperatures and precipitation, lag02, as well as lagged periods of three to six months (lag36) and seven to 12 months (lag712), as the main exposure variables. These lags were chosen as they take into account the immediate as well as the somewhat delayed effects of temperature and precipitation on mortality. We expected to capture most of the medium-term impact (weeks to months) of temperature and precipitation on deaths in this time span. We included a smooth

function of time trend, which was allowed 1 degree of freedom (DF) per year to control for unmeasured confounding from variability in mortality due to seasonality and longer time trends. Furthermore, we adjusted for known periods with mortality extremes that were not caused by climate, namely the war between Sweden and Russia (high mortality in September 1809) and the Spanish flu (high mortality due to infectious disease from October 1918 to January 1919).

We investigated total mortality as well as cause-specific mortality due to infectious and cardiovascular diseases for three separate periods, 1800–1859, 1860–1909, and 1910–1950. The periodic stratification of analyses was done in order to make possible assessment of changes in the exposure response relationships over time in the context of socioeconomic development and epidemiological transitions. We stratified analyses in groups of cause of death and time period. Moreover, we stratified the analyses on sex and age for the different time periods. In additional analyses we adjusted for harvest failure the previous year. Relative risks (RR) were presented with 95% confidence intervals (CI).

We assumed the relationship between monthly mortality and temperature and precipitation to be approximately linear. To further investigate if this assumption was valid we performed sensitivity analyses where the different lags were modelled as smooth functions (with 5 DF each).

To investigate if temperature and precipitation distributions, as well as mean temperatures and mean precipitation levels, changed between the three periods, which would affect our assessment of temporal variation in weather-related mortality impacts, we performed two-sample Kolmogorov–Smirnov tests and one-way analyses of variance and considered a p -value < 0.05 to be statistically significant.

SAS version 9.2 was used to create datasets. R version 3.1.2 and the MGCV add-on package was used for statistical modelling (R Core Team 2015, Wood 2006). The R-code used is available in the supplementary material (Supplementary Material 2 R-code).

3. Results

Recorded monthly temperatures, as well as the three-month moving average of monthly mean temperatures, are presented in Table 1. The last period was the warmest and the period 1860–1909 the coldest. The distribution of mean temperatures during the last period differed significantly from the two previous ones. Moreover, the last period had statistically significantly higher observed monthly mean temperatures than the second period. When comparing the means of the lag02 temperature variable, the last period had statistically significantly higher monthly mean temperatures than the two previous

ones. Precipitation distribution and mean values were similar across the observed values in the last two periods (1860–1909 and 1910–1950), while the distribution and reconstructed values for the first period were significantly lower than the two later ones (Table 1).

Table 1: Descriptive statistics of temperature and precipitation presented by period

TEMPERATURE °C								
PERIOD	MEAN	SD	MIN.	MAX.	MEAN LAG02	SD LAG02	MIN. LAG02	MAX. LAG02
1800–1859	2.1	9.1	-21.0	18.6	2.1	8.1	-15.3	16.4
1860–1909	1.8	8.7	-21.2	18.3	1.8	7.8	-15.3	15.6
1910–1950	3.1	8.5	-17.2	19.1	3.1	7.6	-14.0	16.9
PRECIPITATION CM								
PERIOD	MEAN	SD	MIN.	MAX.	MEAN LAG02	SD LAG02	MIN. LAG02	MAX. LAG02
1800–1859	4.3	2.1	1.0	17.0	4.3	1.6	1.3	9.9
1860–1909	4.8	3.2	0.2	24.8	4.7	2.1	0.9	13.8
1910–1950	4.8	3.3	0	16.5	4.8	2.2	0.7	12.4

Notes: Lag02 is the three-month moving average of monthly mean temperatures and cumulative precipitation for lags 0–2. SD is the standard deviation, min. is minimum, max. is maximum.

In total, 58,947 deaths with valid information on month and year of death were recorded in the region over the entire study period. Population size grew considerably between 1800 and 1950, as did the monthly death counts. Reported infectious disease deaths remained higher than cardiovascular deaths (Table 2).

We found that higher monthly temperatures were associated with lower total mortality for the shorter lags during all our investigated periods. This pattern was not as strong for the longer lags. The association between higher temperatures and total and cause-specific mortality at different lags was largest in the first period and less pronounced from 1860 onwards.

Higher monthly precipitation totals were also associated with lower total mortality at lag 0–2 months during all periods. In the first period (1800–1859), infectious disease and cardiovascular disease mortality were impacted more strongly by precipitation levels at longer lags than at lag 0–2. In the latter two periods, a clear association between levels of precipitation and mortality was not present.

Table 2: Descriptive statistics of cause-specific mortality

PERIOD	POPULATION SIZE			MORTALITY (TOTAL)		MONTHLY NUMBER OF DEATHS				
	MEAN	MIN.	MAX.	N		MEAN	SD	MEDIAN	MIN.	MAX.
1800–1859	10,446	5,658	17,200	Total	10,744	14.9	10.8	13	0	198 ^A
				Infectious	446	0.6	2.02	0	0	25
				Cardiovascular	236	0.3	1.0	0	0	19
1860–1909	24,396	17,246	33,678	Total	23,193	38.7	13.8	37	7	93
				Infectious	5,572	9.3	7.9	8	0	64
				Cardiovascular	669	1.1	1.2	1	0	6
1910–1950	45,883	33,714	62,041	Total	25,010	50.8	13.6	49	27	179
				Infectious	7,663	15.6	10.5	14	0	140 ^B
				Cardiovascular	577	1.2	1.3	1	0	7

Notes: ^AThe 198 deaths in September 1809 were related to the war between Sweden and Russia, otherwise the maximum number of total deaths was 60 (October 1809) and 48 (August 1857).

^B140 infectious disease deaths due to Spanish flu in November 1918 (September 43, October 84, December 58 and January 1919 55). Otherwise the largest amount of infectious disease deaths was 50 (January 1931).

N is the total number of deaths in each period, SD is the standard deviation, min. is minimum, max. is maximum.

In general, patterns for infectious and cardiovascular disease mortality related to temperature and precipitation were similar to those of total mortality. However, the estimates were less precise, particularly before 1860. Age and sex did not modify the association between temperature or precipitation and total mortality during any of the investigated time periods (Table 3).

Adjustment for harvest failure the previous year for the first two periods (1800–1859 and 1860–1909) slightly modified the effect estimates for temperature and total mortality; estimates for precipitation were unchanged. The association of harvest failure the previous year and total mortality was not statistically significant at the 5% level (Supplementary Table 1).

Sensitivity analyses illustrate the relationship between mortality and temperature and precipitation (Supplementary Figures 2–7, where the logarithms of the relative risks of mortality are plotted against temperature and precipitation) respectively when relaxing the assumption of linearity. According to the analysis, the assumption of linearity does not appear generally invalid, although few deviations are observed for specific lag and time periods.

Table 3: Relative risks and 95% confidence intervals of mortality associated with a unit increase in monthly temperature and precipitation

Temperature (change in mortality per °C increase in monthly mean temperatures)					
Lag 02					
Period	Total	Infectious	CVD	Males	Females
1800–1859	0.983 (0.976–0.990)	0.964 (0.928–1.001)	0.951 (0.919–0.985)	0.996 (0.986–1.007)	0.978 (0.964–0.993)
1860–1909	0.989 (0.984–0.994)	0.990 (0.977–1.003)	1.017 (1.001–1.033)	0.988 (0.979–0.997)	0.991 (0.982–1.000)
1910–1950	0.989 (0.986–0.993)	0.978 (0.971–0.985)	0.985 (0.969–1.002)	0.990 (0.986–0.995)	0.989 (0.985–0.994)
Lag 36					
Period	Total	Infectious	CVD	Males	Females
1800–1859	0.970 (0.957–0.984)	0.895 (0.834–0.960)	0.882 (0.826–0.941)	0.993 (0.972–1.015)	0.964 (0.936–0.993)
1860–1909	0.997 (0.988–1.006)	1.010 (0.986–1.034)	1.009 (0.981–1.038)	0.998 (0.977–1.015)	1.003 (0.984–1.022)
1910–1950	1.000 (0.994–1.007)	0.991 (0.978–1.004)	0.973 (0.943–1.003)	1.004 (0.997–1.012)	1.000 (0.990–1.009)
Lag 712					
Period	Total	Infectious	CVD	Males	Females
1800–1859	0.972 (0.954–0.989)	0.852 (0.778–0.933)	0.851 (0.782–0.926)	1.002 (0.974–1.030)	0.963 (0.926–1.002)
1860–1909	0.995 (0.983–1.006)	1.016 (0.986–1.047)	1.034 (0.996–1.072)	0.998 (0.974–1.022)	0.999 (0.975–1.024)
1910–1950	1.003 (0.995–1.011)	1.000 (0.983–1.018)	0.980 (0.942–1.020)	1.008 (0.998–1.019)	1.002 (0.990–1.014)
Precipitation (change in mortality per 1 cm increase in monthly precipitation)					
Lag 02					
Period	Total	Infectious	CVD	Males	Females
1800–1859	0.961 (0.932–0.992)	1.005 (0.873–1.156)	0.848 (0.714–1.007)	0.972 (0.941–1.004)	0.960 (0.923–1.000)
1860–1909	0.984 (0.971–0.996)	0.974 (0.948–1.001)	0.967 (0.930–1.006)	0.974 (0.960–0.989)	0.991 (0.976–1.007)
1910–1950	0.990 (0.982–0.999)	0.971 (0.955–0.987)	0.977 (0.938–1.018)	0.987 (0.977–0.997)	0.993 (0.982–1.004)
Lag 36					
Period	Total	Infectious	CVD	Males	Females
1800–1859	0.976 (0.923–1.031)	0.701 (0.531–0.924)	0.683 (0.495–0.942)	0.990 (0.939–1.044)	0.988 (0.920–1.062)
1860–1909	1.018 (0.998–1.039)	1.003 (0.988–1.081)	0.971 (0.926–1.018)	1.012 (0.989–1.035)	1.021 (0.997–1.046)
1910–1950	1.025 (1.014–1.035)	1.031 (1.011–1.052)	1.009 (0.962–1.058)	1.022 (1.010–1.034)	1.027 (1.014–1.040)
Lag 712					
Period	Total	Infectious	CVD	Males	Females
1800–1859	0.969 (0.902–1.042)	0.642 (0.452–0.912)	0.648 (0.428–0.982)	1.004 (0.937–1.076)	0.973 (0.886–1.069)
1860–1909	0.992 (0.967–1.018)	1.003 (0.947–1.063)	1.091 (1.032–1.153)	0.990 (0.961–1.019)	0.992 (0.962–1.023)
1910–1950	0.991 (0.978–1.004)	0.977 (0.951–1.003)	0.997 (0.939–1.060)	0.991 (0.975–1.006)	0.990 (0.974–1.007)
Age 0–6+					
Period	Total	Age 0–4	Age 5–19	Age 20–59	Age 60+
1800–1859	0.983 (0.976–0.990)	0.998 (0.981–1.015)	0.998 (0.973–1.023)	0.992 (0.978–1.005)	0.981 (0.967–0.993)
1860–1909	0.989 (0.984–0.994)	0.994 (0.980–1.008)	0.994 (0.978–1.010)	0.997 (0.988–1.006)	0.982 (0.974–0.990)
1910–1950	0.989 (0.986–0.993)	0.982 (0.974–0.989)	0.993 (0.982–1.004)	0.997 (0.992–1.002)	0.988 (0.983–0.992)
Lag 36					
Period	Total	Age 0–4	Age 5–19	Age 20–59	Age 60+
1800–1859	0.970 (0.957–0.984)	0.982 (0.949–1.016)	0.956 (0.910–1.005)	0.991 (0.966–1.018)	1.000 (0.976–1.025)
1860–1909	0.997 (0.988–1.006)	1.015 (0.985–1.045)	0.981 (0.950–1.013)	1.001 (0.983–1.019)	1.002 (0.986–1.018)
1910–1950	1.000 (0.994–1.007)	1.000 (0.986–1.014)	0.982 (0.963–1.003)	1.001 (0.991–1.011)	1.008 (1.000–1.016)
Lag 712					
Period	Total	Age 0–4	Age 5–19	Age 20–59	Age 60+
1800–1859	0.972 (0.954–0.989)	1.001 (0.957–1.046)	0.945 (0.890–1.011)	0.991 (0.957–1.026)	1.000 (0.968–1.032)
1860–1909	0.995 (0.983–1.006)	1.016 (0.978–1.056)	0.980 (0.940–1.021)	1.013 (0.990–1.037)	0.991 (0.970–1.012)
1910–1950	1.003 (0.995–1.011)	1.005 (0.987–1.024)	0.986 (0.960–1.012)	1.007 (0.995–1.020)	1.007 (0.997–1.018)
Precipitation (change in mortality per 1 cm increase in monthly precipitation)					
Lag 02					
Period	Total	Age 0–4	Age 5–19	Age 20–59	Age 60+
1800–1859	0.961 (0.932–0.992)	0.959 (0.912–1.009)	0.957 (0.886–1.035)	0.970 (0.930–1.010)	0.975 (0.939–1.012)
1860–1909	0.984 (0.971–0.996)	0.986 (0.965–1.008)	0.998 (0.973–1.024)	0.968 (0.952–0.984)	0.978 (0.961–0.995)
1910–1950	0.990 (0.982–0.999)	0.978 (0.961–0.996)	0.995 (0.970–1.021)	0.988 (0.976–1.001)	0.996 (0.985–1.007)

Table 3: (Continued)

Temperature (change in mortality per °C increase in monthly mean temperatures)					
Lag 36					
Period	Total	Age 0–4	Age 5–19	Age 20–59	Age 60+
1800–1859	0.976 (0.923–1.031)	0.975 (0.891–1.066)	0.812 (0.709–0.929)	0.954 (0.891–1.020)	1.096 (1.032–1.165)
1860–1909	1.018 (0.998–1.039)	1.032 (0.997–1.067)	0.989 (0.951–1.028)	0.993 (0.970–1.017)	1.018 (0.994–1.043)
1910–1950	1.025 (1.014–1.035)	1.042 (1.020–1.064)	1.014 (0.983–1.046)	1.007 (0.993–1.022)	1.035 (1.022–1.049)
Lag 712					
Period	Total	Age 0–4	Age 5–19	Age 20–59	Age 60+
1800–1859	0.969 (0.902–1.042)	1.039 (0.925–1.166)	0.857 (0.721–1.018)	0.936 (0.858–1.022)	1.002 (0.925–1.085)
1860–1909	0.992 (0.967–1.018)	0.992 (0.950–1.036)	1.025 (0.975–1.076)	1.001 (0.970–1.032)	0.944 (0.914–0.975)
1910–1950	0.991 (0.978–1.004)	0.978 (0.952–1.006)	1.027 (0.988–1.068)	1.008 (0.989–1.026)	0.981 (0.965–0.997)

Notes: Lag02 is the three-month moving average of monthly mean temperatures and monthly cumulative precipitation for lags 0–2. Lag36 is the four-month moving average of monthly mean temperatures and monthly cumulative precipitation for lags 3–6. Lag 712 is the six-month moving average of monthly mean temperatures and monthly cumulative precipitation for lags 7–12. CVD represents cardiovascular mortality. During 1800–1860 the total number of deaths of males and females in the different age groups does not add up to the total number of deaths due to information missing in the registers.

4. Discussion

Our study investigated the association of temperature and precipitation with total infectious disease and cardiovascular disease mortality in Skellefteå, northern Sweden, between 1800 and 1950. This period was characterized by profound societal, demographical, and epidemiological transitions in Sweden (Sundin and Willner 2007), so our intention was also to identify potential temporal changes in weather vulnerability.

Results indicated on the whole that both higher temperature and higher precipitation were associated with lower mortality – warmer weather and more rain or snow decreased monthly death counts in most periods and in most lags up to 12 months after exposure.

Vulnerability to adverse weather conditions was more pronounced in the first periods (1800s) than in the last (1900s). Weather impacts were thus largest before the onset of industrialization, when the region of Skellefteå was still a predominantly agricultural society and when average temperatures were lower. This was the case for both temperature and precipitation, and for mortality by infectious and cardiovascular diseases.

In another study, conducted in southern Sweden between 1766 and 1860, Bengtsson and Broström (2010) found lower winter temperature to be associated with increased mortality, but only in adults; furthermore, they did not find any impact of summer temperature on mortality. Their analyses were adjusted, however, for food

prices and other covariables, which might hide some of the temperature effects that we observed.

During the 19th century, the local economy in the Skellefteå region was dominated by agriculture, primarily animal husbandry, on land owned by freeholders. New settlements were established in the previously sparsely populated interior of Skellefteå (Fahlgren 1953). Mortality was high overall, although lower than in most other parts of Sweden (Edvinsson 2004), and fertility was high as well (Coale and Watkins 1986). People in the region experienced several crises during the century: for example, in connection with the Finnish War of 1808–1809, when Russian troops occupied large parts of Västerbotten County. The remote nature of this region and its harsh climate made the population vulnerable to harvest failures, such as during consecutive years of bad harvests in the 1830s and in 1867.

For this preindustrial period between 1800 and 1859, we found quite large impacts of temperature and precipitation on monthly mortality at both shorter and longer lags up to one year. Lower temperatures increased infectious disease and cardiovascular disease mortality mostly after a lag of three to 12 months. This delayed effect may be explained by two causal pathways. First, low summer temperatures can result in poorer harvest outcomes, which would lead to a decreased nutritional status the next spring when people ran out of resources. Appleby (1980), Ljungkvist (2009), Zhang et al. (2011), and McMichael (2012), among others, provided evidence that in preindustrial societies during the Little Ice Age epidemics were related to harvest failures, which in turn are linked to environmental and climatic conditions. This assessment is supported by our analyses on the relationship of harvest outcomes with climate and mortality in the Skellefteå region. Here we found that the largest risk for harvest failure was a cold summer, and harvest failure significantly increased annual death counts in the following year (Schumann, Edvinsson, and Rocklöv 2014). In our present study, harvest failure the previous year slightly changed the association between temperature and mortality towards 1, but not between precipitation and mortality. Thus agricultural production might have functioned as a mediating factor between climate and mortality, explaining the observed long-term effects up to one year.

Second, colder periods may be associated with overall decreased immune status particularly of young children, making them more prone to contract communicable diseases (Bengtsson and Dribe 2002; Edvinsson 2004). Before and during industrialization, infectious diseases were the major cause of death in northern Sweden, especially among children. These causes of death included diarrhoeal and gastrointestinal diseases, which were often water- and food-borne, but also respiratory infections, such as tuberculosis and influenza (Sundin and Willner 2007). This hypothesis is supported by the strong association between higher temperature and lower infectious disease mortality, with a lag of up to 12 months in our data. As for many

other associations, the short-term effect of temperature on infectious disease mortality was greatest during the first period (1800–1859), but persisted even after that.

We have also shown earlier that in preindustrial Sweden high levels of precipitation during springtime decreased annual mortality, while precipitation during autumn increased mortality (Rocklöv et al. 2014; Schumann et al. 2013). Our present analyses do not consider seasonal differences in weather–mortality impacts, so these opposing effects might be disguised. However, in the first period, 1800–1859, the impact of precipitation on infectious diseases occurred mainly with a lag of three to 12 months. This gives support to the hypothesis that food yields following heavy rains in spring contribute to improved nutritional status, leading to lower mortality the next year. Rain in autumn, on the other hand, can cause more short-term increases in mortality due to contamination of food and water with pathogens or due to crowding and the spread of airborne diseases.

At the end of the 19th century mortality decreased and infectious disease epidemics became less frequent in Sweden, although they were still the dominant cause of death (Hofsten 1986; Sundin and Willner 2007). The period 1860–1910 was the coldest in our study region and it was also wetter than the decades before. Despite this, impacts of temperature and precipitation on total and cause-specific mortality were lower than in the first period. At the same time, socioeconomic development, including improved living standards, better sanitary conditions, less overcrowding, and improved child care, led to increased life expectancy and declining infant and child mortality (mainly caused by infectious diseases) (Brändström, Edvinsson, and Rogers 2002; Sundin and Willner 2007). Better social organization and improved communications also mitigated the effects of harvest failures towards the end of the century. Higher living standards may have contributed to reduced climate vulnerability.

In a previous study, we found that in preindustrial Skellefteå it was children aged three to nine years who were most affected by unfavourable weather conditions (Rocklöv et al. 2014). This age-specific vulnerability and decreasing child mortality trends may partly explain declining health impacts of weather in our present study.

The population of Skellefteå increased rapidly during the first part of the 20th century and an urban centre developed. The opening of mines in the interior and the construction of water plants changed the old agricultural setting to one increasingly dominated by industrial production and an expanding service sector. Reforms connected to the establishment of the welfare state made life safer. Life expectancy increased and infant mortality declined from about 100/1000 around 1900 to around 25/1000 in 1950. Although communicable diseases like tuberculosis still took many lives (Puranen 1984), cardiovascular diseases (including myocardial infarction, cardiac failure, and stroke) and cancers became the dominant causes of death (Sundin and Willner 2007).

In this period, the first half of the 20th century, total mortality in Skellefteå was hardly affected by temperature. For infectious disease mortality, we found only short-term impacts, while cardiovascular mortality was affected to some degree also at longer lags. Contemporary studies have shown that cardiovascular mortality is related to low temperature (Hajat, Kovats, and Lachowycz 2007; Huynen et al. 2001), which was also the case for our preindustrial and industrializing setting in northern Sweden. Thus the decline in weather vulnerability in cardiovascular diseases from the first to the second period (which may be attributed to improved living standards) was less pronounced in the last period. One reason might be that the emergence of lifestyle-related cardiovascular risk factors (sedentary occupation, smoking) produced a more susceptible population which counterbalanced benefits in living standards. The changing age distribution in the population, caused by longer life expectancy and decline in fertility, is likely to explain, too, weather impacts on cardiovascular mortality.

5. Strengths and limitations

This study has relied on unique digitized parish records on individual vital data, including the dates and causes of death of almost 60,000 individuals between 1800 and 1950. Most studies on historical populations have so far focused on long-term fluctuations in climate and mortality (years, decades, or centuries) (Galloway 1986; McMichael 2012; Zhang et al. 2011), while our analyses relied on the use of monthly data for temperature, precipitation, and death counts. This data gave us the possibility of also investigating short-term impacts, which might provide insights into causal pathways. Furthermore, the time range from the early 1800s to the mid-20th century allowed for the observation of temporal changes in weather vulnerability during the epidemiological transition.

A limitation of this study was the low number of total and cause-specific monthly death counts occurring during the first period, resulting in our risk estimates being less precise and requiring cautious interpretation.

Specific causes of death cannot be assumed to be highly reliable when comparing data from different times in the registers because of progress in the medical sciences and common beliefs among laypeople. Therefore there is great uncertainty about both the reliability and the validity of diagnoses made by ministers during the 19th century when an exact measure of cardiovascular mortality and other diseases was lacking (Bengtsson 2002; Bengtsson and Lindström 2000). However, the available information can be used as a proxy. Another source of uncertainty in the results arises from the statistical method used to estimate historical monthly precipitation data for Umeå. In

the absence of any other available information, we surmised that the reconstruction method was adequate to inform subsequent mortality modelling; supporting indicators include correlation coefficients strongly suggestive of a linear relationship between Umeå and Uppsala precipitation totals, as well as close agreement between observed and modelled precipitation totals. The modelled precipitation totals had less variance than observations and thus the reconstructed data for the historical period are biased towards the mean.

Although the statistical models controlled for trend, it is possible that not all the seasonal effects were captured.

6. Conclusions

In this study of a community in northern Sweden before and during industrialization, we were able to show short- and medium-term impacts of monthly weather on total and cause-specific mortality. We found that both higher temperature and precipitation were associated with lower mortality: that is, monthly death counts with a lag of up to one year were negatively correlated with warmer weather and more precipitation. The decreasing risk of mortality related to adverse weather conditions from the early 1800s to the middle of the 20th century demonstrates that contextual factors such as dependence on agricultural production, living standards, and lifestyle, as well as demographic factors, can contribute substantially to the climate and weather vulnerability of a population.

Future studies should look into seasonal variations in weather impacts to provide more detailed insights into potential causal pathways for communicable and cardiovascular diseases.

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