Climate and disease in historical urban space. Evidence from 19th-century Poznań, Poland

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Abstract. This study examines the relationship between temperature levels and precipitation amounts as explanatory variables for the probability of death due to waterborne and airborne diseases in historical urban space. To date, the literature has not focused on the climatological epidemiology of 19th-century Polish urban areas. We used individual mortality data from Poznań parish death registers between 1850–1900. Each deceased individual was assigned the average monthly temperature values and precipitation amounts in the month of death, LAG1 (one month lagged) temperature and LAG1 rainfall, and place of residence. We studied the relationship between weather conditions and mortality using formalized statistical models reflecting the discrete nature of the response data (via multinomial logistic regression). Lagged monthly average temperature levels and lagged monthly average precipitation amounts were better predictors of airborne and waterborne disease mortality than the concurrent (non-lagged) monthly averages. The lagged effects of temperature and precipitation on waterborne and airborne diseases were significant (except for the smooth lagged average monthly temperature effect for airborne diseases). There was also significant spatial heterogeneity (differences among city quarters) in the prevalence of deaths due to waterborne and airborne diseases.

1 Introduction

A large body of literature has evidenced the existence of a relationship between climate, or more precisely, its components such as temperature and precipitation, and the health status and well-being of humans (e.g., Diaz et al., 2001). The relationship between temperature level and precipitation amounts is observed in correlations between various disease rates, cause-specific death rates, and weather variations on different time scales – over weeks, months, or years. More recently, there has been greater interest in the impact of extreme temperatures (extreme highs and/or extreme lows) on health and risk of death. Healthy people, without comorbidities, have an effective heat regulation system that allows the body to effectively cope with thermal stress. However, in particularly sensitive groups: the elderly, infants, and people with comorbidities of the circulatory system and respiratory system, thermoregulation at extremely high or low temperatures is disturbed (Calleja-Agius et al., 2021).

Recent research has shown that 37% of warm-season heat-related deaths can be attributed to anthropogenic climate change, with increased mortality observed on every continent (Vicedo-Cabrera et al., 2021; Zhao et al., 2022). Prolonged periods of hot days are responsible for the increase in heat-related death rates (e.g., Hajat et al., 2007; D'lppoliti et al., 2010; Tong et al., 2012; Kent et al., 2014; Chen et al., 2015), especially due to cardiovascular and respiratory diseases (Basu and Samet, 2002; Donaldson et al., 2003; Basu, 2009), mental and nervous systems disorders (Stafoggia et al., 2006), diabetes, and kidney and urinary system diseases (Conti et al., 2005; Rey et al., 2007). Studies have also proven a link between extremely high ambient temperature during pregnancy and high prevalence of hypertension, eclampsia, and cataract in infants (Poursafa et al., 2015), perinatal brain injury (Kasdorf and Perlman, 2013), a high prevalence of preterm births (Lajinian et al., 1997; Flouris et al., 2009; Basu et al., 2010; Auger et al., 2014; Ha et al., 2017), and a significant increase in infant mortality (Rooney et al., 1998; Kysely and Kim, 2009; Basagaña et al., 2011; Nitsche et al., 2011). In turn, extremely low temperatures lead to an increased incidence of respiratory infections (Alberti and Díaz, 1997; Huynen et al., 2001; Mäkinen et al., 2008). Excess morbidity and mortality during periods of low temperatures are observed especially in elderly people (Alberdi et al.,1998; Huynen et al., 2001; Ballester et al., 2003).

A link between weather patterns and population health has also been noted in relation to historical times. Research by Åström and a team (2016) showed that mortality decreased over time as a result of improvements in nutritional status, a decline in infant deaths, and positive changes in the course of demographic and epidemiological transition. Also indicated was a broader context for weather factors deterioration: a decrease in agricultural production, which translated into an increase in grain and food prices, in turn reducing the comfort of societies and leading to demographic crises and annual variations in births, marriages, and deaths (Imhof, 1976; Lee, 1981; Galloway, 1985, 1986, 1994; Pfister and Wanner, 2021). The annual seasonal patterns of mortality were thus shaped by climate components, such as temperature or precipitation. Summer surpluses of deaths were usually caused by infectious diseases, especially cholera, dysentery, and typhoid, while in infants, there was a strongly marked summer peak associated with nutritional diarrhoea or other diseases of the digestive system (e.g., Woods et al., 1988, 1989; Landers, 1993; Liczbińska, 2009a, b, 2011, 2015; Vögele, 2010; Budnik and Liczbińska, 2015). Temperature and humidity were also shown to be associated with malaria incidences in historical Europe, for example in Denmark (Ingholt et al., 2022), Sweden (Chen et al., 2021), and Finland (Hulden and Hulden, 2009), or plague (Krauer et al., 2021). In autumn and winter higher frequencies of morbidity were recorded more often than in the warm months of the year, due to diseases of respiratory tract, such as pneumonia and bronchitis, and diseases of circulatory system. Diseases of these two systems were frequent causes of excess deaths in autumn and winter, until early spring (Landers, 1993; Liczbińska, 2009a, b, 2011, 2015; Budnik and Liczbińska, 2015). Meanwhile, Swedish studies of historical populations (Junkka et al., 2020; Karlsson et al., 2021) have indicated that the impact of environmental factors on human health is highly complex and largely dependent on the specific local context, with many additional, often unknown, contributing determinants of vulnerability.

Although the relationship between climate components, such as temperature, precipitation, humidity, and health status is well-documented in the literature, this has concerned only contemporary and/ or selected populations from the past. There exists a gap in such research regarding Eastern and Central Europe, although such studies are crucial in order to analyse long-

term developments often associated with climate change. When it comes to acute infectious diseases, until the end of the 19th century and for the first decades of the 20th century, studies on morbidity and mortality from infectious diseases were limited to elucidating risk/causative factors. The work on the cholera epidemic in the 1850s in relation to ecological conditions in an urban space, conducted by John Snow, is considered to be a starting point for systematic epidemiological research on infectious diseases (Fisman, 2007). Previously, miasmatic theories were prevalent, ascribing outbreaks of cholera and typhoid fever to unspecified germs which came to the surface with rising groundwater (Vögele, 2001). In this context, rainfall was considered to be a major variable. Such theory provided the scientific framework for construction of expensive drainage and sewerage systems in the cities. With the rise of bacteriology towards the end of the 19th century, cholera and typhoid became associated with specific waterborne pathogens and a concrete aetiology. As a consequence, adequate central water supply systems became crucial for municipalities. For historical populations, to the best of our knowledge, there are no studies pointing to the relationship between the risk of death from waterborne and airborne diseases and the local weather/climate, as described by temperatures and precipitation patterns. Neither has the literature been focused on the epidemiology of the 19th-century Polish urban space in a climatological context.

The present study examines the relationship between temperature values and precipitation rates as explanatory variables and the probability of death due to waterborne and airborne diseases in a historical urban space as responses. We focused on two types of infectious diseases due to their high burden for the 19th-century populations, and the growing evidence from contemporary populations that they are related to climate/weather. We studied the relationship using formalized statistical models reflecting the discrete nature of the response data via multinomial logistic regression.

2 The city of Poznań in the 19th century

After the Congress of Vienna in February 1815, the city of Poznań became the capital of the Grand Duchy of Poznań, which was in territorial and administrative terms a part of the Prussian state. The Prussian authorities soon decided to turn the city into a fortress, which inhabited the city's spatial development for long years (Trzeciakowska and Trzeciakowski, 1987; Matusik, 2021). With time, the city started to suffer from the lack of free space and at the end of the 19th century it was "suffocating" within the surrounding walls. After 1867, the major barriers inhibiting the influx of population from rural areas disappeared as all previously applicable charges for permanent stay registration were abolished, while after 1870, the demand for labour force increased that is why Poznań became an attractive destination mainly for the population of Great Poland's rural areas looking for jobs in the city. Due to the lack of space in the overcrowded city centre, the incoming migrants settled mainly on the outskirts (Matusik, 2021). The area of 943 ha was enclosed within the city walls and did not change until the beginning of the 20th century (Kruszka, 2004). In the 1860s, 1870s and 1890s the city was inhabited by over 53,000, 60,000, and 73,000 inhabitants, respectively (Galloway, 2007), which means that within the fortress the density per km^2 increased with time along with the increase of the population size, and in the years mentioned above it was over 5,500, 6,300 and 7,700 people per km^2, respectively. At the beginning of the 20th century the fortress was demolished, the urban area expanded to 3,300 ha,

and the population increased to over 117,000 people, what resulted in the decline of the population density to 3,500 per km^2 (see also: Kruszka, 2004).

100   The ecological conditions in Poznań deteriorated increasingly and in parallel with the population increase. High population density and lack of urban infrastructure deteriorated "urban ecology", and this contributed to frequent epidemics of infectious diseases. Particularly dangerous were epidemics of cholera, repeating in 1831, 1837, 1848, 1852, 1855 and 1866. They caused massive and rapid changes in population numbers within a short period of time (Piankowski, 1987; Liczbińska, 2021). There were also outbreaks of measles (in 1857, 1861 and 1869), scarlet fever (1863 and 1874), and smallpox (1871)

105 (Liczbińska, 2009a, b). In Poznań, in the years when epidemics occurred most often, i.e., in 1850–1874, infant mortality rate was at the level of 315 per 1,000 live births[1]. For comparison, in 1895–1904, after implementation of water supply system and sewage system, infant mortality declined to 199 per 1,000 live births[2]. Life expectancy at birth until the end of 1880s did not exceed 30 years[3].

3 Meteorological observations in the 19th-century Poznań

110 The first systematic air temperature observations in Greater Poland took place after the creation of a measurement network by the Prussian Meteorological Institute established in Berlin in 1847. The first temperature measurements began in Poznań on January 1, 1848, in the meteorological station at 1 Pocztowa Street (Smosarski, 1925; Kolendowicz et al., 2019). This station operated until March 1862, but in April that year, it was moved to 1 Grobla Street and operated there until September 1867.

115 At the station on Grobla Str., the thermometer was set at 2.5 m above ground level (AGL). In October 1867, the observations were moved to 1 Zielona Str. and continued being conducted there until July 1885. In August 1885, the location of the station was changed from 1 Zielona Str. to 2 Zielona Str. In that station, temperatures were measured with a thermometer set at 6.2 m AGL. The station at 2 Zielona Str. operated until September 1892, when it was moved to 3 Długa Street, where the thermometers were placed at 8.6 m AGL. The station on Długa Street operated until December 1911 (Smosarski, 1925). Until

120 1892, temperature measurements were taken with a thermometer without a cover and placed outside the window. From September 1892, zinc sheet sheaths were used on thermometers to protect the measurements against atmospheric factors such as wind, sun, and humidity, which distorted the results (Smosarski, 1925). Until 1884, measurements were taken three times a day: at 6 am, 2 pm, and 10 pm. In December 1884, the hours of observation were changed to 7 am, 2 pm, and 9 pm. This continued until May 1919. Meteorological observations in Poznań were carried out by professors at the Municipal Real School:

125 first by Spiller, and from April 1862 – by Magener. In 1889, meteorological measurements were taken by the Physical Institute of the Royal Academy (Smosarski, 1925). Initially, a Mahlmann rain gauge was used to measure precipitation, which until September 1867 was placed in the yard at 1 Grobla Str., at 2.6 meters AGL. From October 1867 to 1873, the rain gauge was

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[1]authors' calculations based on data derived from Poznań parish birth and death books for years 1850–1874.
[2]authors' calculations based on data derived from Prussian Statistical Yearbooks (*Preussische Statistik…,* 1875–1913); see also Liczbińska, 2009a, b, 2011, 2015.
[3]authors' calculations based on data derived from Prussian Statistical Yearbooks (*Preussische Statistik…,* 1875–1913).

in the garden at 1 Zielona Str., and then moved to Pocztowa Str. After 1880, Osnaghi's then Hellman's rain gauges were introduced into precipitation observations. Those rain gauges were placed on poles at 1 m AGL. The measurements were taken
130 once a day: at 2 pm, and after 1885, at 7 am. In the 1890s, a meteorological station also operated in Jeżyce, which at that time was a village near Poznań. The thermometers were set at 9.5 m AGL, and the rain gauge was placed in the garden, at 1m AGL. A pharmacist named Wildt was the manager of the station. Observations were taken three times a day: at 7 am, 2 pm, and 9 pm (Smosarski, 1925).

4 Dataset

Data on mortality were obtained from parish death registers for the years from 1850 to 1900, deposited in the State Archives in Poznań. Each individual entry contained the date of death (day, month, year), place of residence, and cause of death. Records for the following parishes were used: St. Margaret, St. Mary Magdalene, Holy Cross, and St. Martin. For the analysis, 35,005 individual entries containing the cause of death recorded were used. The specific causes of death were categorized as follows:
140 1) Waterborne diseases (WBD): cholera, typhoid fever, dysentery, diarrhoea; 2) Airborne diseases (ABD): measles, smallpox, whooping cough, diphtheria, croup, influenza, measles, scarlet fever, rash; 3) other causes of death (diseases of the circulatory, excretory, and nervous systems not related to waterborne and airborne diseases). Therefore, we can consider the three-cause exhaustive death classification as an instance of multinomial distribution. For each deceased person, the following meteorological data were assigned: the average monthly temperatures in the month of death and in the month preceding the
145 month of death (LAG1, or one-month lagged temperature), and the average rainfall in the month of death and the month preceding the month of death (LAG1 rainfall). The data on temperatures and precipitation for Poznań for the years 1850–1900 were taken from the monograph by Władysław Smosarski: *"Temperature and precipitation in Greater Poland"*, published in 1925 by the Ministry of Religious Affairs and Public Education, in Poznań. Five quarters were distinguished within the fortification ring (1–5) (Figure 1). Quarters 1–4 included the area on the left bank of the Warta River, while quarter 5
150 encompassed the streets on the right bank of the Warta River (Piankowski, 1987). To each individual deceased person, a quarter identification (quarter number) was assigned, based on their address.

5 Methods

All analyses were based on multinomial logistic regression (Hosmer and Lemeshow, 2000; Harrell, 2015). We modelled the
155 probabilities of death due to waterborne diseases (WBD), airborne diseases (ABD), and other causes of death as a function of several covariates simultaneously. Modelling in the multinomial logistic regression was conducted with a flexible GAM (Generalized Additive Model; Hastie and Tibshirani, 1990; Wood, 2017) approach, which made it possible to capture both parametric (e.g., linear) and general smooth effects (e.g., penalized-spline-implemented curves). We explored various models and came up with a model structure where the probability of death depended on the year in which it occurred (allowing for
160 flexible/smooth/nonparametric annual trends – as we did not have any a priori information about the shape of the trends as mathematical functions), one-month-lagged average temperature values and one-month-lagged precipitation amount.

Interesting to note is that the one-month lagged models for both temperature and precipitation patterns were always better (in terms of AIC, Akaike Information Criterion; Burnham and Anderson 2002) than non-lagged models, so we used the lagged version. The covariate effects can be different for different categories of causes of death (model allows for cause-specific actions of a concrete covariate). Due to the well-known identifiability reasons (Hosmer and Lemeshow, 2000), the multinomial model of the three possible response categories (waterborne, airborne, other causes of death) in fact has two dimensions and not three, as this third category constitutes a baseline. We therefore chose "the other causes of death" category as the baseline. We fitted two modifications of the model – one non-spatial or purely temporal (without distinguishing different city quarters) and the other spatial-temporal (allowing for varying incidence among quarters). As a sensitivity check, the quarter model was then implemented in two ways – as a GMRF (Gaussian Markov Random Field; Rue and Held, 2005), and in the fixed quarter effects version. Since the results of the two were very similar, we preferred the simpler fixed-effects quarter version and present here its results only. The models were fitted in R (R-core Team, 2023), using the mgcv package (Wood, 2017). They were implemented with complexity-penalized splines for smooth components (Harrell, 2009; Wood, 2017), estimated via penalized likelihood with penalization constants being held at their REML (restricted maximum likelihood; Harville, 1977; Wood et al., 2016) estimates.

6 Results and discussion

6.1 Precipitation and temperature effects on the probability of deaths from waterborne and airborne diseases

The detailed parametric and non-parametric effects for monthly precipitation amounts and temperature levels on the probability of deaths due to waterborne diseases and airborne diseases are summarized in Table 1. All effects in the model are significant, except for the smooth lagged average monthly temperature effect for airborne diseases (AIC=7899 for the one-month-lagged model and AIC=8479 for non-lagged model). The growing lagged precipitation rates increased the probability of death both from waterborne and airborne deceases. The lagged monthly average precipitation effect was much greater for deaths from waterborne diseases than for deaths from airborne ones. Smooth effect of lagged monthly average temperature value was larger for deaths due to waterborne diseases than deaths caused by airborne diseases. Similarly, the annual trends were more significant for waterborne diseases than for airborne diseases. The results from the purely temporal model are presented in Figures 2–3. Note that the lagged average temperature effect was larger for waterborne diseases and of different shape than for airborne diseases (Figures 4–5). For waterborne diseases the increased overall effect occurred much faster in higher temperatures (approximately above 15°C) than in lower (colder) ones (Figure 4). In low temperatures, the probability of deaths from waterborne diseases was least in the lowest temperatures (Figure 4). Many epidemiological studies of contemporary populations from different regions of the world have shown that precipitation and temperature are major environmental factors linked to the prevalence of waterborne diseases (e.g., Singh et al., 2001; Hashizume et al., 2008; Drayna et al., 2010; Eisenberg et al., 2013; Carlton, et al., 2014; Chowdhury et al., 2018; Levy et al., 2018; Deshpande et al., 2020). High temperatures and humidity favour multiplication and survival of waterborne pathogens and their vectors (Singh et al., 2001; Semenza and Menne, 2009; Drayna et al., 2010; Carlton et al., 2014, 2016). Periods of heavy rainfall are also related to the prevalence

of waterborne diseases by increasing the likelihood of water contamination from sewages (Moors et al., 2013).They flush faecal material into waterways and into surface drinking water and enhance the multiplication of intestinal pathogens. Heavy rains increase hydrologic transport-driven iron availability in environmental waters, leading to increased microbial growth (Faruque et al., 2005); they can flush accumulated pathogens into surface water directly, through runoff, and indirectly, through the mobilization of bacteria in soil (Carlton et al., 2014). The lags after rainfall and temperature measurements for waterborne diseases, as obtained for the 19th-century Poznań, have been also observed in contemporary populations, for example, in Hong Kong (Wang et al., 2018), the United States (Drayna et al., 2010), and New Zealand (Lai et al., 2020). This phenomenon could be related to the incubation periods of waterborne pathogens, which vary in length in various types of pathogens. The incubation period of *Vibrio cholerae* is 1–5 days (Azman et al., 2013; Eisenberg et al., 2013). For other diarrhoea-causing pathogens, mainly rotavirus and pathogenic *Escherichia coli*, the incubation period is usually around 1week (Eisenberg et al., 2003), while lags reported in cholera endemic settings range from several weeks to 2 months (Hashizume et al., 2008, 2010). Bacillary dysentery symptoms begin within 2 to 10 days of infection, whereas amoebiasis starts more gradually and usually lasts about 2 weeks (*Infectious Intestinal Disease…*, 2012). Meanwhile, *Salmonella typhi* and *Salmonella paratyphi* incubate between 1 to 3 weeks (*Infectious Intestinal Disease…*, 2012). A similar maximum three-week incubation period was seen for *Listeria monocytogenes* (*Infectious Intestinal Disease…*, 2012). In the 19th-century Poznań longer lags in the effects of temperature and precipitation for waterborne diseases might have been caused through the accumulation of bio-organic materials at the bottoms of the water reservoirs, transport-driven with rainfalls from discharged sewage, and then their decay under the high ambient and water temperature. Delayed effects of measured temperatures and precipitation for waterborne diseases could also have been caused through plankton blooms trigged by nutrients driven into the waters with heavy rains. The city was in a network of three rivers with plankton blooms from the spring through summer months until autumn. Biological material stayed at the bottoms of the rivers until autumn, when the temperature was still high (Kaniecki, 2004). The annual sanitary reports on water cleanliness in the Warta River highlighted the contamination with such substances as nitric acid, chlorine, ammonia, and hydrogen sulphide. This did not prevent people from using water from the river for consumption (Kaniecki, 2004). Poznań at that time suffered from poor sanitation conditions and lack of access to clean water. Heavy rains contaminated surface and groundwater by sewage, which was then used by Poznań inhabitants for drinking, cooking, and washing clothes. The sanitary infrastructure was non-existent practically until the end of the 19th century, thus favouring the spread of infections with urban sewage. Water was drawn from shallow wells which were often polluted with harmful sewage discharged directly to the moats. There were also street gutters with ineffective drainage full of still, contaminated water during the summer heat periods (Kaniecki, 2004; Liczbińska, 2011). The sewage system was opened in 1896 (Kaniecki, 2004). This was very late compared with other European cities, such as Hamburg (1842), London (1850s), Paris (1850s), Leipzig (1860), Frankfurt (1867), Zurich (1867) and Prague (1868) (Kappner, 2019). A modern water supply system was only completed in 1866. Meanwhile, Paris had tap water supply since 1802, London since 1808, and Berlin since the 1850s (Kappner, 2019). In Poznań it was systematically developed with the increase in the number of inhabitants and dwellings; between 1889 and 1913, it extended from 29 to 176 km (Sobczak, 1979). Unfortunately, it was of little significance when compared with Berlin,

230   where the water supply network at that time was of 661,246 metres (Vögele, 1998), and by 1892, 100% of Berlin's lots were provided with tap water (Kappner, 2019).

In the case of airborne diseases, our research showed the temperature effects to be non-monotonic: both increasing and decreasing (Figure 5). The highest probability of death was noted in middle temperatures (local maximum of the effect curve at around 12 centigrade – when the temperatures are neither too low or too high, mostly in fall and spring). Then the

235   probability showed a decrease as the temperature increased. When the temperature decreased from 12°C, the probability of deaths due to airborne disease first decreased (to local minimum of around freezing point). With further decrease of temperature, the airborne disease probability tended to increase, but since there were not sufficient data with very low temperatures (there were not as many extraordinarily cold years), the super-cold temperature effect had to be estimated with a very high level of uncertainty (very wide 95% confidence intervals about the estimate) to be safely interpretable. This is

240   probably due to the wide temperature spectrum necessary for survival of airborne diseases' agents. Tang (2009) has suggested that bacteria within the same structural classification (e.g., Gram-negative) may vary in how they respond to temperature and humidity. The research of Harper (1961) showed that low temperatures of 7–8°C were optimal for airborne influenza survival. This was corroborated by Lowen and colleagues (Lowen et al., 2007), who proved that influenza was most readily transmitted through the air in cold and dry conditions. Other studies have shown that temperatures above about 24°C appear to reduce the

245   survival of airborne bacteria (Tang, 2009). This has been confirmed in Gram-negative, Gram-positive, and intracellular bacteria such as *Pseudomonas* (Handley and Webster, 1995), *Salmonella* (Dinter and Muller, 1988), *Serratia* (Ehrlich et al., 1970), *Escherichia* (Wathes et al.,1986), *Bacillus* (Ehrlich et al., 1970), *Bordetella* (Stehmann et al., 1992), *Chlamydia* (Theunissen et al., 1993). Regarding fungi, which are also airborne disease agents, studies have confirmed their pathogenic relationship with high temperatures and humidity and also with low winter-temperatures and dry air (Oliviera et al., 2005).

250   Airborne agents, such as viruses, bacteria, or fungi, are transmitted as small particles suspended in the air through breathing, talking, coughing, sneezing, or raising dust (Božič and Kandič, 2021). The dynamics of their survival and spread is influenced by the ambient temperature and humidity and favoured more by population density than sanitary and epidemiological factors. Hence, lagged effects of average temperatures for airborne diseases were smaller than for waterborne diseases. Moreover, many airborne agents, such as viruses causing influenza, have a relatively short infectivity timescale which resulted in a very

255   short lag. For example, the incubation period for influenza is typically 1–4 days (Belshan at al., 2014).

6.2 Spatial (quarter) differences in the probability of deaths from waterborne and airborne diseases

The area of the city of Poznań enclosed within its fortress walls was divided into 5 quarters superintended by 5 police stations (Piankowski, 1987). Quarters 1–4 were located on the left bank of the Warta River, while quarter 5 – on the right bank (Figure

260   1). The quarters differed in terms of the number of inhabitants and their social and economic composition, infrastructure, hygienic conditions, etc. It is for this reason that we decided to introduce the city quarters to the model. The results are presented in Table 2. The effects of average precipitation amounts and temperature levels on the probability of death due to waterborne and airborne diseases were significant, except for lagged monthly average temperature effect upon airborne diseases. This

model suggested that there were substantial differences in mortality rates due to waterborne and airborne diseases in different quarters. Parametric coefficients for monthly rainfall rates and direct effects of city quarters on the probability of deaths due to waterborne diseases (WBD) and airborne diseases (ABD) are demonstrated in Table 3. All effects were statistically significant. The smooth and lagged effects were very similar to those from the previous models (Figures 2–5) and therefore not presented graphically here. Figure 6 demonstrates spatially varying probability of death due to waterborne diseases in the 5 city quarters. In quarter 1 probability of deaths caused by waterborne diseases was higher than in quarter 2, while in quarter 3 it was higher than in quarter 4, but lower than in quarter 5. We should recall once again that in 1850 Poznań had over 44,000 inhabitants, which 10 years later had increased to 50,000, then over 60,000 in the 1870s and 1880s, and at the end of the century – over 73,000 (Galloway, 2007). In the area, which had not changed size since the construction of the fortress, the population density increased with population number, so that at the end of the 19th century there were over 7,700 persons per km^2 (Kruszka, 2004). Such overcrowded space presented a considerable epidemiological problem related to the lack of sanitary infrastructure and clean water supply. A street was at the same time a gutter, and every water reservoir became a source for drinking water. Since underground waters were shallow, they were heavily contaminated by leaks from cesspits and sewers. Waste was poured onto the streets or courtyards, from where it flowed down the sewers to the wells. This continues up until the mid-19th century, when water was drawn mainly from the rivers, shallow wells, sources located near the cemeteries, and shallow ditches supplied with water from ponds. The first wooden waterworks were built in 1847. These drew water from the spring in Winiary and distributed it to the wells at Świętokrzyski Square, Bernardyński Square, and to the suburb of St. Adalbert. The total length of that aqueduct was 2.5 km, which was insufficient for the supply of clean water to all inhabitants (Kaniecki, 2004). According to data collected by the police heads during the cholera epidemic in 1866, quarters 1, 2, 3 and 4 were inhabited by 8513, 8631, 11095 and 9194 people, respectively, while quarter 5 was inhabited by 7,706 people (*Polizeipräsidium Posen…*,1866–1868). Quarters: 1, 2 and 5 were located in the neighbourhood of the Warta River, which for their many inhabitants could potentially have been a source of drinking. The highest probability of death due to waterborne diseases was observed in quarters 1 and 5, and to our surprise – in quarter 3. The Market Square area and neighbouring streets (a part of quarter 1) were the only places in the city with access to potable water. This was related to the fact that in the 16th century the fountains with fresh spring water supplied by pipelines from outside the city were built there. The rest of Poznań inhabitants used polluted water sources located in the vicinity of their houses (Kaniecki, 2004). Moreover, the surplus of deaths from waterborne diseases in quarter 1 and quarter 3 was related to the presence of hospitals in those quarters. The first was established in the 1820s in the former Bernardine Convent (in quarter 1), and the second was the city hospital on Szkolna Street, opened in 1854 (in quarter 3) (Kaniecki, 2004). At that time hospitals were unsanitary and overcrowded, which, in times of epidemics, made them rather more "places of dying" than "places of recovery". In quarter 5 which at that time included the most dilapidated districts as Chwaliszewo, Śródka, Zagórze, Zawady and Ostrówek, and inhabited by the poorest part of Poznań society, drinking water was taken straight from the rivers, moats, and ponds (Kaniecki, 2004). The cholera-specific mortality rates in 1866 for quarters 1, 2, 3, 4 were 2–3 deaths per 100 people, while for quarter 5, 6 deaths per 100 people (Piankowski, 1987). This example of the 1866 cholera epidemic shows that despite quarter 5 having the lowest population size

compared to the other quarters, the higher mortality in this quarter can be attributed to the lack of drinking water supplies, the very low levels of population hygiene, and no (systematized) isolation of ill people and carriers. The spatially varying probability of death due to airborne diseases in Poznań is presented in Figure 7: its level was highest in quarter 5. Just as the mortality from waterborne diseases is dependent mainly on the level of hygiene, the mortality from airborne diseases is influenced by a number of other factors: housing and working conditions, standard of living, nutrition, but also individual characteristics of the immune system (Liczbińska, 2009a). The standard of living in quarter 5 might well have been the lowest of all. This district was inhabited by the poorest social strata: small craftsmen and labourers, whose wages did not reach the minimum subsistence level (Liczbińska, 2015). These inhabitants earned 500 to 600 marks a year on average, most of which was spent on rent, the rent for one room being 40–56% of the salary of the lowest earners even up to the beginning of the 20th century. After paying their rent, a labourer had less than 1 mark per day to support the whole family (Łuczak, 1965). The workday in the overcrowded industrial plants became increasingly longer and working conditions deteriorated. In many craft workshops, the twelve-hour workday was often extended to 16 hours (Liczbińska, 2015). In 1900, nearly 45% of the Poznań population still lived in one-room dwellings, often cramped, damp and unheated, the single room sometimes being shared by 5 to 12 people (Liczbińska, 2015). These overcrowded places likely facilitated the spread of airborne agents and led to the increase in morbidity from respiratory diseases, especially in the autumn and winter. The poor health conditions of labourers were further exacerbated by malnutrition affecting their immune systems (Munteanu and Schwartz, 2022; Wu et al., 2019). Quarters 1–4, on the other hand, were inhabited by the wealthier part of Poznań society: artisans of various trades, including shoemakers, tailors, bricklayers, and carpenters, and a more professional group represented by the owners of taverns, restaurants, wine bars and beer halls located in the Market Square. Those comprising the intelligentsia (doctors, teachers, officials, etc.) and merchants also resided quarters in 1– 4 (Trzeciakowska and Trzeciakowski, 1987; Matusik, 2021).

7 Conclusion

Individual demographic data on causes of death have been shown to provide a deep insight into the relationship between mortality and its various climatic, ecological and social determinants. The extensive body of literature has highlighted that poor urban ecology in 19th-century populations was related to the lack of sewage system and clean water intakes as the main factors responsible for death surpluses. Furthermore, studying the impact of meteorological phenomena on infectious diseases in past populations is of key importance in understanding their health status and adaptation to environmental conditions. In our study, using monthly average temperature values and precipitation rates, we demonstrated the influence of climate on the probability of death from infectious diseases. We pointed out the complexity of the relationship between precipitation rates and air temperatures and waterborne and airborne diseases in city districts that differed in hygiene and standards of living. Our findings underline the need to account of climatic factors when evaluating the probability of death due to infectious diseases in urban spaces. Further research is required to uncover to what extent socioeconomic and biophysical factors can modify climate-disease relationships. While the burden of infectious diseases has been in decline since the second half of the 19th

century as a result of improved hygiene, medical advances, and improved quality of life, the effects of climate change may have slowed up the progress in reducing the impact of these diseases.

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Data on temperatures were drawn from the monograph of Władysław Smosarski, *Temperature and precipitation in Greater Poland*, published in 1925 by the Ministry of Religious Affairs and Public Education, in Poznań.

Data are appended to the paper as a separate file.

Author contributions. GL, MB, JV designed the study. MB provided the statistical analysis and prepared figures 2–7. GL provided cartographical figure 1 and the biological context. GL and JV provided the historical analysis and context. All authors discussed the methods and results and provided comments on the paper.

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600 Table 1. Parametric and non-parametric effects for monthly precipitation amounts and temperature levels on probability of death due to waterborne diseases (WBD) and airborne diseases (ABD)

Summary parametric effects			
Effect name	Estimate	Standard error	z-value
Intercept (WBD)	-2.517	0.039	-63.768
Lag 1 monthly precipitation (WBD)	0.005	0.0007	6.866
Intercept (ABD)	-1.124	0.026	-43.389
Lag 1 monthly precipitation (ABD)	0.001	0.0005	2.325
Summary of non-parametric effects			
Effect name	Equivalent degrees of freedom (effect complexity)	Ref.df	Chi.sq
Annual trend (WBD)	8.857	8.999	1492.51
Lag 1 monthly temperature (WBD)	8.045	8.754	1408.99
Annual trend (ABD)	6.431	7.577	199.53
Lag 1 monthly temperature (ABD)	3.361	4.206	7.944

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Table 2. Parametric and non-parametric effects for monthly precipitation amounts, temperature levels and city quarters on probability of death due to waterborne diseases (WBD) and airborne diseases (ABD)

Summary parametric effects			
Effect name	Estimate	Standard error	z-value
Intercept (WBD)	-2.523	0.039	-63.772
Lag 1 monthly precipitation (WBD)	0.005	0.0007	6.484
Intercept (ABD)	-1.129	0.026	-43.580
Lag 1 monthly precipitation (ABD)	0.001	0.0005	2.385
Summary of non-parametric effects			
Effect name	Equivalent degrees of freedom (effect complexity)	Ref.df	Chi.sq
Annual trend (WBD)	8.957	8.999	1473.049
City quarter (WBD)	3.647	4.000	52.106
Lag 1 monthly temperature (WBD)	8.054	8.758	1413.774
Annual trend (ABD)	5.401	6.549	135.340
City quarter (WBD)	3.736	4.000	100.579
Lag 1 monthly temperature (ABD)	3.311	4.145	7.313

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Table 3. Parametric coefficients for city quarters and monthly precipitation amounts on probability of death due to waterborne diseases (WBD) and airborne diseases (ABD)

Summary parametric effects			
Effect name	Estimate	Standard error	z-value
Quarter 1	-2.349	0.049	-47.959
Quarter 2	-2.701	0.077	-35.096
Quarter 3	-2.413	0.065	-37.322
Quarter 4	-2.696	0.052	-51.871
Quarter 5	-2.516	0.062	-40.344
Lag 1 monthly precipitation (WBD)	0.005	0.001	6.848
Quarter 1	-1.103	0.033	-33.357
Quarter 2	-1.144	0.048	-23.598
Quarter 3	-1.086	0.043	-25.034
Quarter 4	-1.303	0.034	-37.927
Quarter 5	-0.896	0.039	-22.925
Lag 1 monthly precipitation (WBD)	0.001	0.000	2.389

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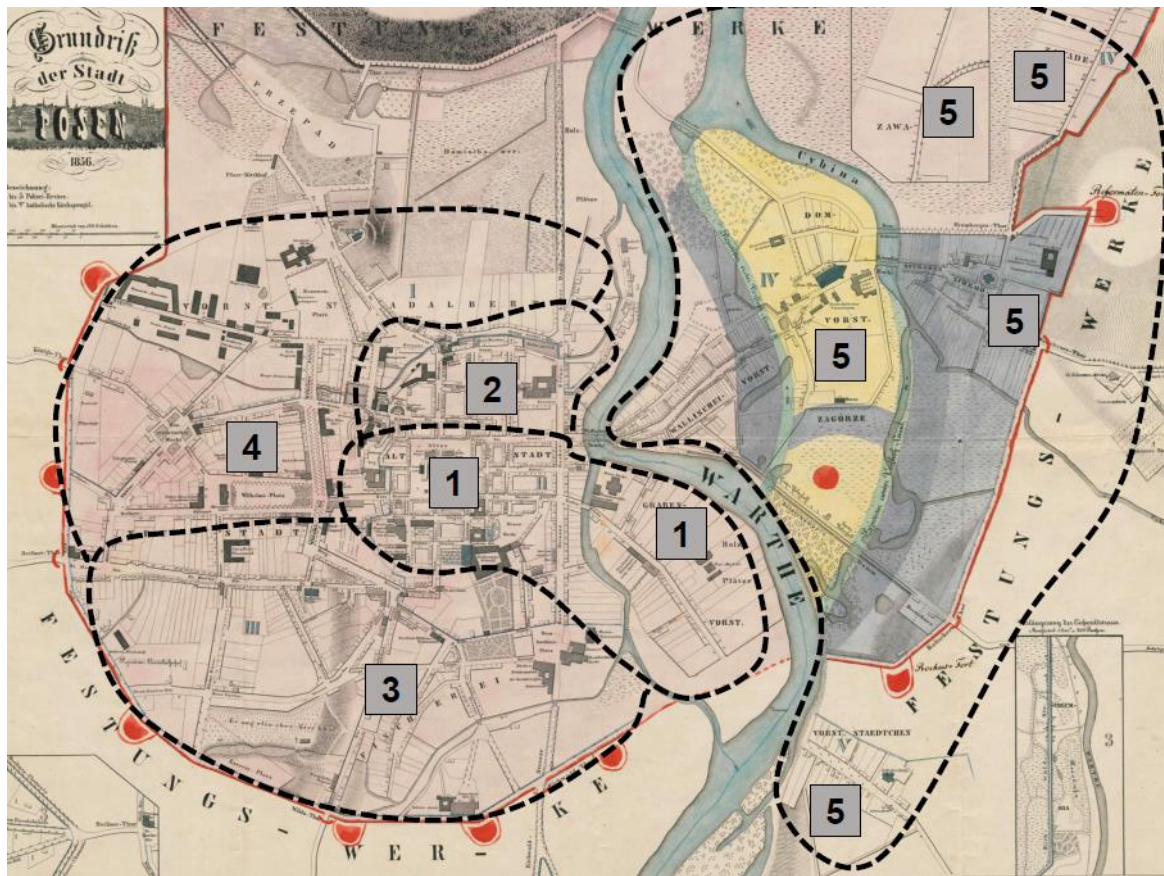


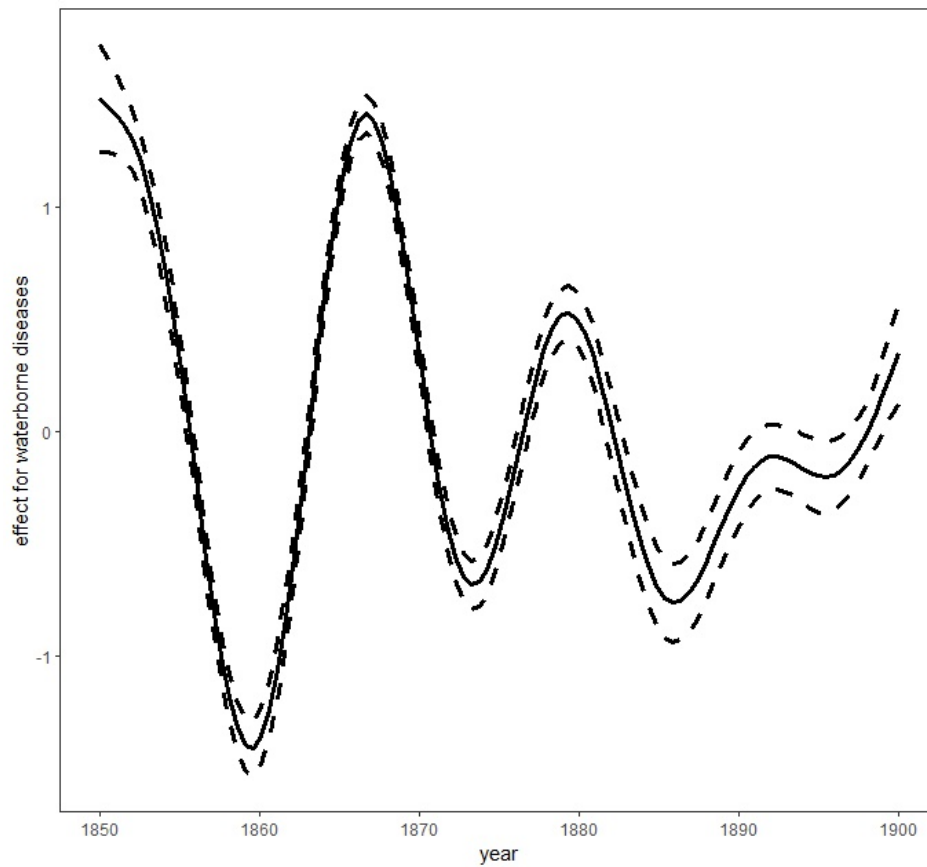
Figure 1. City of Poznań divided into five quarters. Map produced by Grażyna Liczbińska based on data collected while authoring the paper and file from https://cyryl.poznan.pl/kolekcja/plany-poznania-do-1945-roku-biblioteka-raczynskich/ (Last accessed: 19 March 2023).

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Figure 2. Smooth trends (smooth curves and their 95% asymptotic pointwise confidence limits) for waterborne diseases. Figure produced by Marek Brabec based on data collected for the project supported by the National Science Centre, Poland, Grant: *Cholera epidemic in 1866 as a turning point in the history of Poznań,* No. UMO-2021/41/B/HS3/00594.

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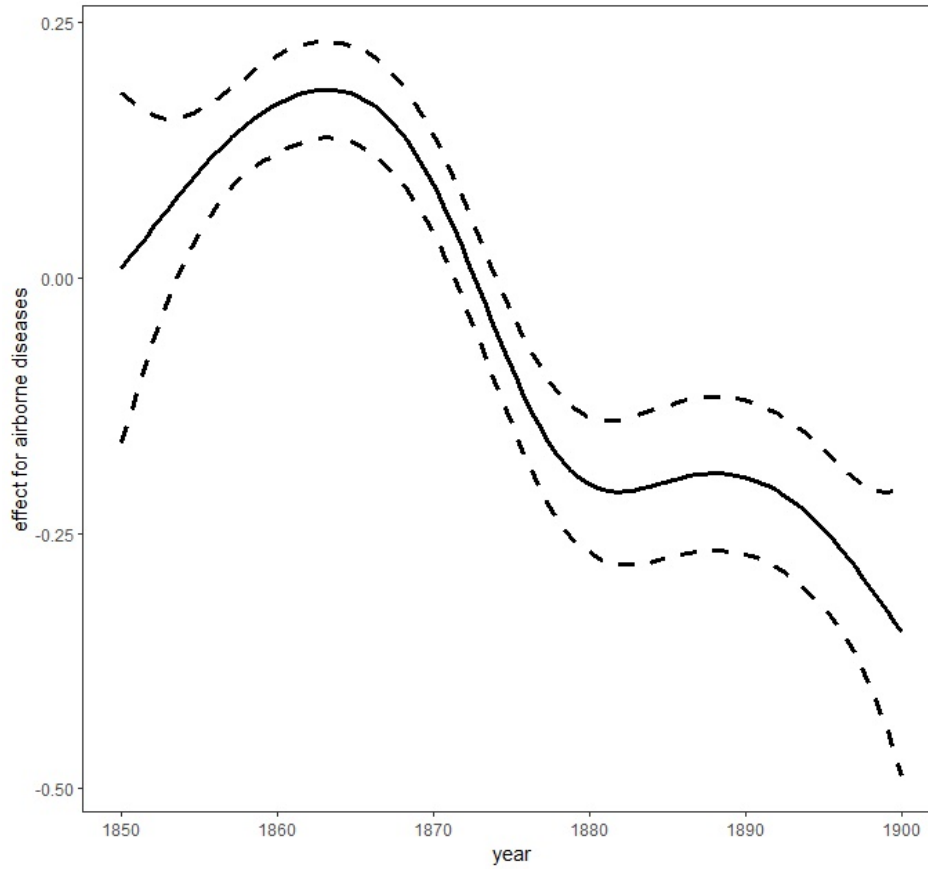
Figure 3. Smooth trends (smooth curves and their 95% asymptotic pointwise confidence limits) for airborne diseases. Figure produced by Marek Brabec based on data collected for the project supported by the National Science Centre, Poland, Grant:
685   *Cholera epidemic in 1866 as a turning point in the history of Poznań,* No. UMO-2021/41/B/HS3/00594.

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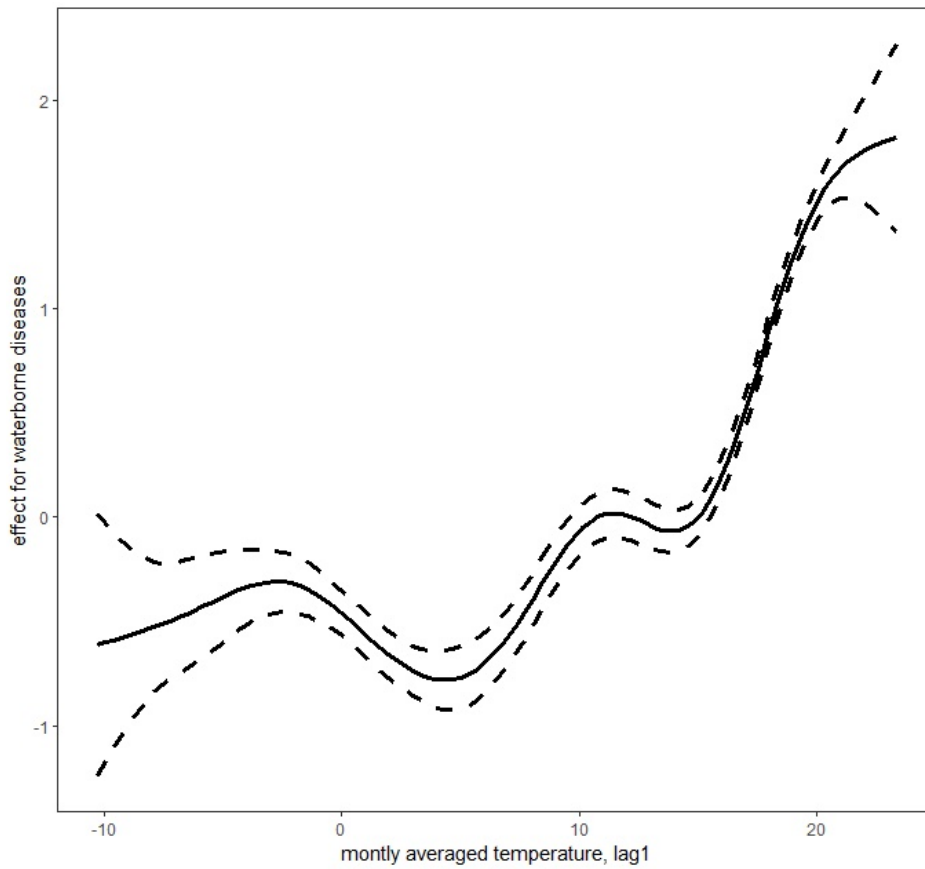
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Figure 4. Lagged mean temperature effect for waterborne diseases. Figure produced by Marek Brabec based on data collected for the project supported by the National Science Centre, Poland, Grant: *Cholera epidemic in 1866 as a turning point in the history of Poznań,* No. UMO-2021/41/B/HS3/00594.

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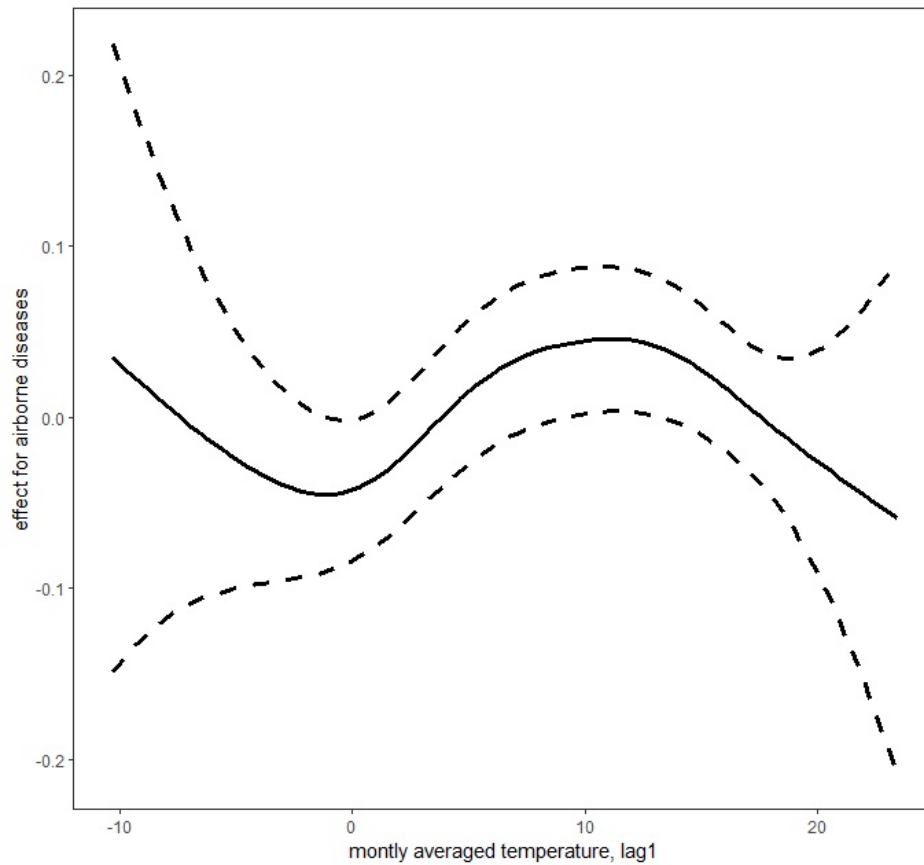
Figure 5. Lagged mean temperature effect for airborne diseases. Figure produced by Marek Brabec based on data collected for the project supported by the National Science Centre, Poland, Grant: *Cholera epidemic in 1866 as a turning point in the history of Poznań,* No. UMO-2021/41/B/HS3/00594.

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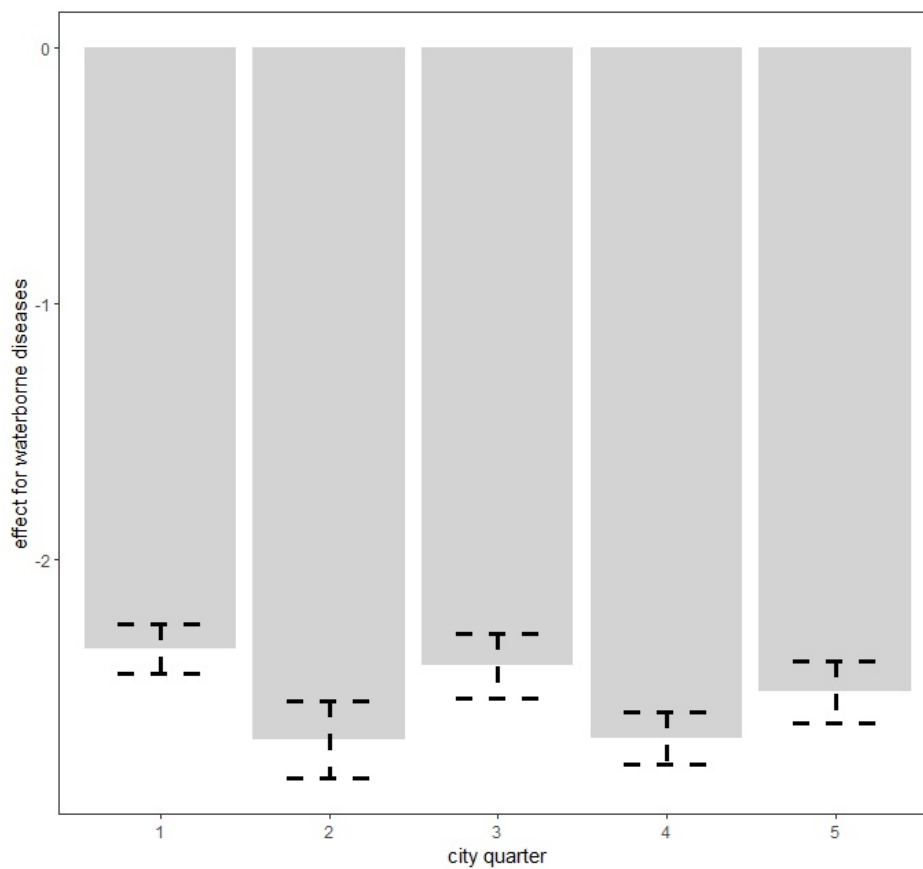
Figure 6. Spatially varying probability of death due to waterborne diseases in Poznań quarters. Figure produced by Marek Brabec based on data collected for the project supported by the National Science Centre, Poland, Grant: *Cholera epidemic in 1866 as a turning point in the history of Poznań*, No. UMO-2021/41/B/HS3/00594.

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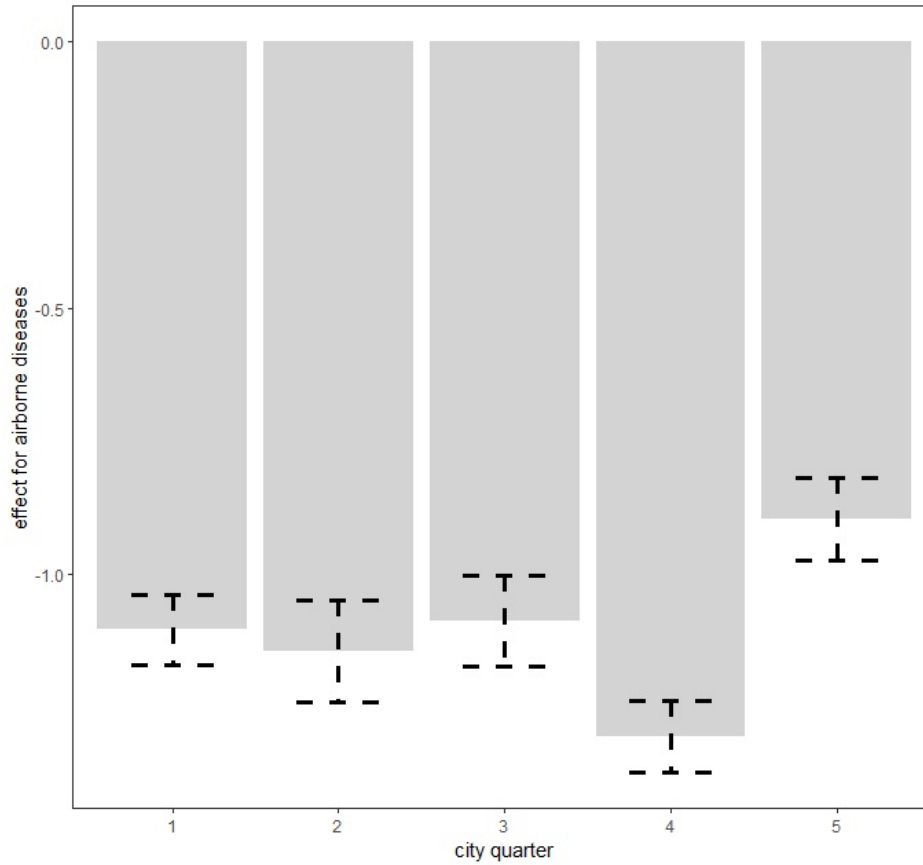
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Figure 7. Spatially varying probability of death due to airborne diseases in Poznań quarters. Figure produced by Marek Brabec based on data collected for the project supported by the National Science Centre, Poland, Grant: *Cholera epidemic in 1866 as a turning point in the history of Poznań,* No. UMO-2021/41/B/HS3/00594.

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