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Abstract

This paper presents the first comprehensive quantitative account of epidemic cholera in 19th-century Germany. Using a new dataset based on archival sources, it documents nearly half a million cholera deaths, along with outbreak timing and population at risk, across 2,685 cities and 852 rural counties within the 1871 German Empire. I document five stylized facts: First, cholera was primarily an urban disease, with city death rates averaging 3.5 times higher than in rural areas. Second, mid-sized cities (1,000-3,000 inhabitants) were the most severely affected. Third, cholera's geographic epicenter focused on the less developed North-East territories (Central Poland), but shifted South-West over time. Fourth, outbreaks spread more rapidly across regions and within cities over time, despite declining overall mortality. Fifth, local epidemics converged in severity across locations but became more spatially clustered over time. Understanding these complex patterns requires analysis of cholera's interaction with dominant trends of 19th-century Western development, including public health reforms, urbanization, market integration, and political change. While the rich cholera historiography has long recognized these links, it merits greater attention from quantitative social scientists, including economic historians. Datasets like this one are the foundation for that engagement.

JEL Codes: J11, N33

Keywords: Historical Epidemiology, Demography, Mortality Transition

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1 The continuous significance of cholera

Epidemic cholera claimed the lives of almost half a million people in Germany between 1831 and 1895, the years spanning the country's first and last major outbreaks. This paper offers the first complete and precise account of cholera's mortality patterns, drawing on a new dataset covering 2,685 cities and 852 rural counties within the 1871 German Empire. Cholera—a highly contagious and often lethal diarrheal disease—became emblematic of industrialization, urbanization, and globalization as it swept across the globe in multiple pandemics during the 19th century.[1] While now a matter of the past in the Western world, cholera's global and historical importance has kept it at the center of a vibrant literature in medical, social, and increasingly, economic history. I argue that this literature stands to benefit significantly from a more quantitative, data-driven perspective, making use of the rich epidemiological data that archival sources offer.

At the core of this paper is a newly assembled dataset, compiled from archival and supplementary sources, and covering the territory of the 1871 German Empire. The dataset includes annual cholera deaths, population at risk, and outbreak start and end dates. Analysis of these data yields five stylized facts about cholera in Germany. First, despite notable rural outbreaks, cholera was primarily an urban phenomenon, with cities experiencing death rates 3.5 times higher than their rural surroundings. Second, mid-sized cities with populations of 1,000–3,000 were most severely affected—a pattern observed both in cross-sectional comparisons and as individual cities expanded over time. Third, the severity of cholera was initially concentrated in Germany's historical North-East (in the region that now includes central and northern Poland) before shifting to the South-West over time. Fourth, epidemics diffused more rapidly across space and developed faster within cities as time progressed. Fifth, outbreaks became increasingly localized, transforming from a broadly shared experience into rarer, regionally concentrated events.

Although cholera was not the deadliest disease of the 19th century, its periodic outbreaks instilled widespread fear and uncertainty because of their sudden onset and mysterious nature. The disease was deeply entangled with industrialization, urbanization, and other modernization processes. Research has, for example, linked cholera to 19th-century sanitary reform movements, social struggles, wars, and the emergence of global public health cooperation. Moreover, contemporary outbreaks in developing regions echo aspects of 19th-century epidemics, ensuring that historical cholera continues to attract scholarly attention.

1. Cholera is caused by the ingestion of the *V. cholerae* bacterium. Transmission occurs via fecal contamination of food, water, and fomites. Among others, John Snow demonstrated its waterborne nature in 1854, while Robert Koch first identified the bacterium in 1883, following earlier work by Filippo Pacini (Azman et al. 2013; Clemens et al. 2017; Deen et al. 2020). Historically, cholera originated in the Ganges Delta and spread across six pandemics since 1817. The seventh, ongoing pandemic began in Indonesia in 1961. Although cholera disappeared from Western countries by the early 20th century, it still causes an estimated 3–5 million cases and up to 120,000 deaths annually—mostly in less developed regions such as sub-Saharan Africa (Ali et al. 2015). Due to effective treatments, the case fatality rate has declined from around 50% in the 19th century to 1% today.

This sustained interest is reflected in a vast body of qualitative research—including detailed case histories of Victorian London (Stieva 2023), post-revolutionary Paris (Kudlick 1996), and the notorious 1892 Hamburg outbreak (Evans 1987)—as well as studies examining cholera's impact across nations and regions, such as in Africa (Echenberg 2011), the United States (Rosenberg 1987), and France (Bourdelais and Raulot 1987). Comprehensive global overviews have also been published (Pollitzer et al. 1959; Hamlin 2009a).[2]

Despite this rich literature, cholera has attracted limited engagement from quantitative social scientists. For instance, Doran et al. (2024)'s recent survey of data-driven historical pandemic studies identifies just four papers on cholera compared to 97 on the 1918/19 Influenza—a stark contrast to cholera's prominent position in historical demography and the *qualitative* research literature (Høiby 2020; Piret and Boivin 2020). This gap highlights untapped potential: explicitly causal, quantitative frameworks can assess the extent to which prominent cholera narratives generalize beyond case studies and evaluate their quantitative importance. One example is the claim that cholera epidemics spurred the development of public health and sanitation reforms in 19th-century Western societies. Although this idea gained renewed attention during the COVID-19 pandemic, questions remain about the underlying mechanisms, the necessary societal and political preconditions, and the overall role of epidemics as drivers of social change (Hamlin 2009a; Vögele et al. 2021; Kappner 2024). The benefits of a data-driven approach are evident from the growing quantitative literature on other pandemics, such as the Black Death (Jedwab et al. 2024; Siuda and Sunde 2021) and the 1918/19 Influenza pandemic (Beach et al. 2022). Developing high-quality datasets—such as the one presented here—is a crucial first step toward systematic quantitative analysis of cholera's social, economic, and political impacts. Because of its size, central European position, and rich geographic variation, Germany is an excellent starting point for this endeavor.[3]

In section 2, I outline the new dataset on Germany's historical cholera mortality, discuss the historical origins of quantitative cholera historiography, and validate the data against three independent sources. Section 3 contextualizes Germany's cholera experience by comparing its aggregate temporal and spatial patterns to those of neighboring countries and other major epidemic diseases; in that section, I also present the five stylized facts outlined above. Section 4 discusses the potential for a data-driven approach to cholera's social history by highlighting five intriguing narratives and hypotheses from the literature. Section 5 concludes.

2. See Stieva (2023) for a recent overview. In the German context, additional studies focus on Berlin and East Prussia (Dettke 1995) and on individual cities like Düsseldorf (Fliescher 1977). An early synthesis of the German literature is provided by Otto et al. (1990), and a recent meta study finds that 10% of the German medico-historical literature after 1945 was dedicated to cholera (Fangerau et al. 2023).

3. Further development of this research agenda is also supported by the growing data infrastructure in German economic history (Braun and Hesse 2024; Jopp and Spoerer 2024).

2 A dataset of German cholera epidemics

This paper presents the first comprehensive quantitative account of Germany's cholera history. The dataset documents almost half a million cholera deaths between 1831 and 1895, coded at the level of 2,685 cities and 852 time-consistent rural areas.[4] It also includes annual population estimates for these reporting units and reports start and end dates for each outbreak. This data effort builds on a long tradition in historical epidemiology, aiming at the documentation of cholera's spatial and temporal patterns. In the 19th century, European governments produced a wave of quantitative reports in the aftermath of major cholera outbreaks. An impressive example is the French government's comprehensive municipality-level report on the 1854 epidemic (Ministère des travaux publics 1862). For Germany, similar reports were published for the 1873 (Hirsch 1879) and 1892 (Kaiserliches Gesundheitsamt 1896) outbreaks, but political fragmentation before German unification in 1871 hindered comparable efforts across the entire territory.[5] A second wave of quantitative cholera research emerged in the 1930s–1950s, after cholera had largely disappeared from Western countries. This included retrospective "geo-medical" studies for various nations, such as Austria (Krebs 1941), Great Britain (Pertl 1940), Russia (Olzscha 1938), Switzerland (Teuber 1947), and France (in der Beeck 1948). For Germany, Krehnke (1937) assembled a detailed collection of quantitative evidence on major cholera events. As these studies rely heavily on the government-issued reports mentioned above, rather than archival sources, they lack both spatiotemporal detail and consistency, and often remain vague on smaller epidemics.[6] More recently, spatially and temporally consistent cholera datasets have been constructed for countries such as Italy (Forti Messina 1984; Alfani 2014), England (Davenport et al. 2019), Japan (Kuo and Fukui 2007), and India (Aslam et al. 2024). However, to my knowledge, no dataset matches the present one in terms of its spatio-temporal granularity and completeness.[7]

Constructing the dataset, I draw on a broad range of sources, including official reports, archival materials, and secondary literature. Like most other European states, Prussia and the other German territories started registering and publishing official cause-of-death statis-

4. After 1895, cholera virtually disappeared from Germany. 107 deaths were recorded between 1905 and 1910 (Kirchner 1907; Jansen 1911; Krehnke 1937). During World War I, about 1,750 deaths were registered, almost exclusively in the army (Roesle 1925; Reichskriegsministerium 1934). The last two deaths due to cholera ever registered in the German Empire were in Königsberg in 1921 (Krehnke 1937, 101). Germany has been unaffected during the ongoing seventh pandemic, except for isolated imported cases (Oprea et al. 2020).

5. For Prussia, which accounted for about half of Germany's territory before 1866, reports on major outbreaks were routinely published, although these presented highly aggregated figures, e.g. at the district or national level rather than for individual counties and cities. See Szrant (1874) for a summary of these reports.

6. This limitation is seen in the fact that Krehnke (1937) does not provide an estimate of the total cholera death toll for Germany. Similarly, Kohn's (2007, 143) encyclopedia of epidemics includes total cholera death toll estimates for many countries—but not for Germany.

7. Comprehensive data of *contemporary* cholera outbreaks is also scarce due to underdeveloped reporting facilities in many affected countries. Apart from the World Health Organization, Johns Hopkins University's Disease Dynamics program collects local incidence data, e.g. for sub-Saharan Africa (Lessler 2018).

tics only in the second half of the 19th century (e.g. 1862 in Prussia), thus well after the most important cholera waves. For this reason, it is generally not possible to comprehensively reconstruct the patterns of cholera epidemics from official vital statistics.[8] Thus, the primary source used for this dataset was archival material, consisting of official correspondence between government bodies on local outbreaks, often featuring precise tabulations. I add to this data from contemporary government-issued outbreak reports, which often provided disaggregated data for certain outbreaks and certain regions. Supplementary data were gathered from medical journals, public health treatises, and local newspapers. About 83% of the recorded cholera figures in this dataset come from such primary sources, with archival evidence covering most cases.

Additional estimates were derived from later historical treatises. These are often based on archival material that is no longer available.[9] In a few cases, primary or secondary sources suggest an outbreak for a specific year in a specific region, but no quantitative data at the required granularity is available. In these cases, I approximated local death counts using excess crude mortality, other close correlates, or data available at coarser spatial levels. As a general rule, the absence of evidence for an outbreak across a wide range of sources was taken as evidence for the absence of cholera. This is a reasonable assumption for Germany, where cholera's high public profile and reporting standards precluded deliberate cover-ups or war-related underreporting, unlike in other prominent cases like Portugal (1833), Hungary (1848/49), and Italy (1911).

Cholera death counts were transcribed verbatim from original sources, without adjustment for values that appeared unexpectedly high or rounded. However, I corrected unambiguous summation errors in the process. Although reporting errors could arise from misattributed causes of death, cholera's distinct symptoms meant it was generally recognizable, and local authorities had strong incentives to report fatalities accurately. While underreporting was common for non-fatal cases (due to asymptomatic cases or comorbidities, Ali et al. 2015), the same does not apply to fatalities (Hardy 1994, 478). In Germany, unlike e.g. in England, officials took care to distinguish epidemic cholera from the milder, endemic "European cholera" (or *cholera nostras*) and other diarrheal diseases.[10]

This dataset divides the German territory as of 1871 into a time-constant set of 852 territorial units ("counties") to facilitate comparability over time and statistical analysis. The imposition of time-constant reporting units implies some anachronistic choices, given Germany's administrative reorganization and territorial expansion over the study period.[11] Apart from

8. An important exception is England, which started compiling cause-of-death statistics at the local level right after the first cholera outbreak, allowing detailed cholera mappings (Stieva 2023; Davenport et al. 2019).

9. In a few instances, conflicting estimates from multiple sources exist for a specific outbreak. In these cases, I generally used the primary evidence, except in cases where revised estimates in the secondary literature explain the source of disparity.

10. In the context of historical European plague epidemics, Roosen and Curtis (2018) have recently pointed out the problematic nature of large-scale quantitative datasets.

11. For example, my dataset includes all outbreaks in the Imperial territory of Alsace-Lorraine, even though

these rural units, 2,685 cities were defined using a compromise between different requirements. With few exceptions, all location that possessed a town charter or were considered a "titular city" at any point between 1831 and 1914 are included. I extend this set of cities with other locations listed in the German city encyclopedia (Keyser et al. 1939–2003).[12]

Apart from fatality counts, my dataset also reports outbreak start and end dates. This information was typically reported along death counts, but where it was missing, alternative sources were cross-referenced. Preference was given to dates unambiguously marking the first and last fatalities in each location. In some cases, however, dates mentioned in original sources might have correspond to the first and last diagnosed case, or the day it was officially registered. Given cholera's short incubation period and high fatality rate, any discrepancy between case onset and death should be minimal.[13] The dataset also includes annual population estimates for each unit from 1831 to 1895, facilitating mortality *rate* calculations. Population data were collected primarily from the official reports for the territorial, and later Imperial, censuses conducted at 3–5 year intervals over much of the 19th century. However, detailed population data (including counts for individual cities) is scarce for some non-Prussian German territories before 1871, making interpolations necessary.[14] While some sources report age- or sex-specific death counts (and populations), such information was not included in the dataset due to a lack of systematic evidence.[15]

In appendix section A, I conduct three quantitative validity checks on my dataset: First, I demonstrate that cholera death rates are highly correlated with all-cause excess mortality rates derived from independent civil registration data. Second, I validate local cholera outbreaks against a risk model that incorporates three reliably measurable environmental and infrastructural factors–temperature, rainfall, and water quality–and their interactions. Third, I compare both mentions of cholera outbreaks, and death counts for these, to an alternative source that contains selective reports on cholera outbreaks in Germany. In appendix section B, I detail all sources and assumptions, territory by territory.

it was only annexed into Germany in 1871. Furthermore, the reporting units in this territory are the counties defined by the German Empire in 1871, which do not perfectly align with the former French arrondissements.

12. Ploeckl (2017) discusses the city definition in historical contexts, and 19th-century German in particular.

13. My database reports the start and end dates of each outbreak, but does not include the richer temporal information sometimes reported in sources, such as weekly death counts. Thus, without further assumptions, it cannot, be used to define at what exact date the peak of an outbreak occurred.

14. See Michel (1985), Matzerath (1985), Gehrmann (2009), Pfister (2020), and Pfister and Fertig (2024) for comprehensive treatments on German population statistics for the 19th century. Earlier censuses did not always include the so-called "military population", generating a source of bias in my population estimates that I did not attempt to correct.

15. Liczbińska (2021) and Liczbińska and Vögele (2023) point out important age- and sex-specific differences in cholera incidence in 19th century Poznan, then a part of the German Empire.

3 Cholera in Germany: Basic patterns and five stylized facts

This section explores the basic spatial and temporal patterns of cholera outbreaks in 19th-century Germany, using the dataset described earlier. To put cholera into perspective, table 1 provides mortality estimates for major pandemic outbreaks in German history. Care should be taken when comparing per-capita mortality across pandemics, as the duration of each differs. The 2nd (1831–1838), 3rd (1848–1861), and 4th (1865–1874) cholera pandemics produced death tolls comparable to the 1918/19 Influenza pandemic, though cholera spread over multiple years rather than a few months. The devastating 1870–1873 smallpox outbreak, exacerbated by the Franco-Prussian War, resulted in a similarly high death toll.

Table 1: Mortality during important pandemics in Germany

Disease	Period	Deaths (per capita)	Source
Plague	1347–1353	ca. 3 mio. (20–25%)	Jedweb et al. (2022, 137)
Smallpox	09/1870–06/1873	181,067 (0.44%)	Reichsgesundheitsamt (1925, 52)
Influenza	10/1918–03/1919	263,328 (0.41%)	Förtsch and Rösel (2021)
COVID-19	03/2020–05/2023	174,490 (0.21%)	World Health Organization (2025)
Cholera	05/1831–01/1838	63,603 (0.21%)	*this study*
Cholera	01/1848–01/1861	221,124 (0.62%)	*this study*
Cholera	08/1865–11/1874	175,937 (0.43%)	*this study*

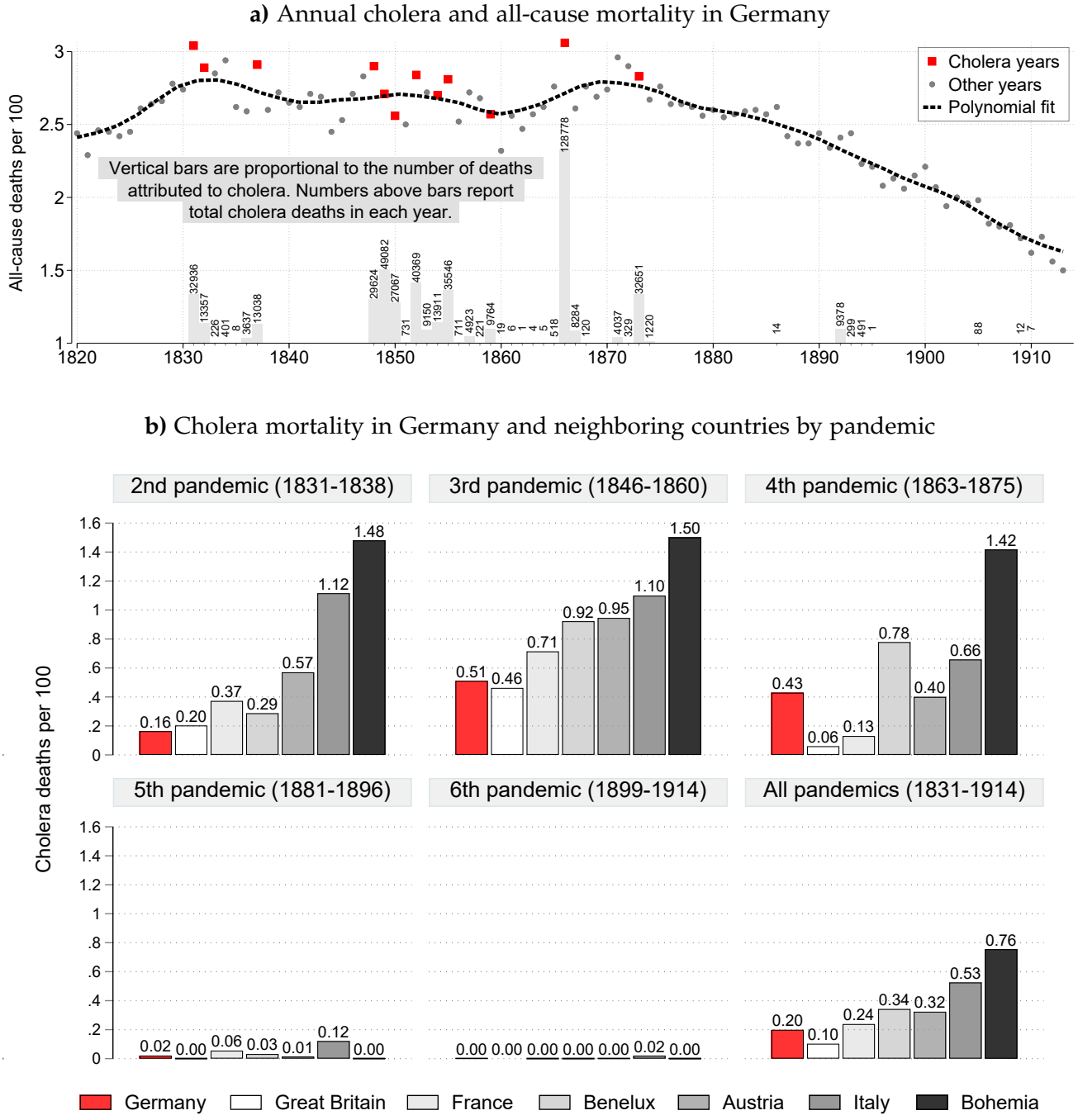
Note: Mortality estimates refer to Germany's respective territory in each period. Deaths per capita are computed using the mean population during each outbreak, or directly taken from the quoted sources. Other important epidemic outbreaks in German history include typhoid fever during the 30 years war (1618–1648), the Russian flu (1889–1890), diphtheria (19th century) and AIDS (20th century). The COVID-19 pandemic was ongoing as of January 2025, though the WHO ended the global emergency in May 2023.

Cholera had a significant impact on all-cause mortality, as shown in panel a of figure 1. The graph plots annual mortality rates in Germany from 1820 to 1914, highlighting years in which cholera accounted for more than 1% of recorded deaths. Many of the deadliest years—1831 (3.04 deaths per 100), 1848 (2.9), and 1866 (3.06)—were such "cholera years". This long-run perspective also suggest that cholera's emergence coincided with elevated mortality rates beginning in the 1830s, while its decline after the mid-1870s aligned with a long-term reduction in mortality. Multiple factors contributed to Germany's mortality transition, but cholera's rise and fall played a crucial role.[16]

Panel b of figure 1 compares Germany's cholera incidence to neighboring countries, including Great Britain, France, the Benelux states, Austria, Italy, and Bohemia (modern Czech Republic). During the 2nd and 3rd pandemics, Germany experienced relatively low cholera mortality compared to neighboring countries, including the United Kingdom in

16. Other infectious diseases, such as smallpox, typhoid fever, and tuberculosis, also shaped mortality patterns in 19th-century Germany. However, systematic data on these diseases is limited, except for smallpox (Spree 1998; Otto et al. 1990).

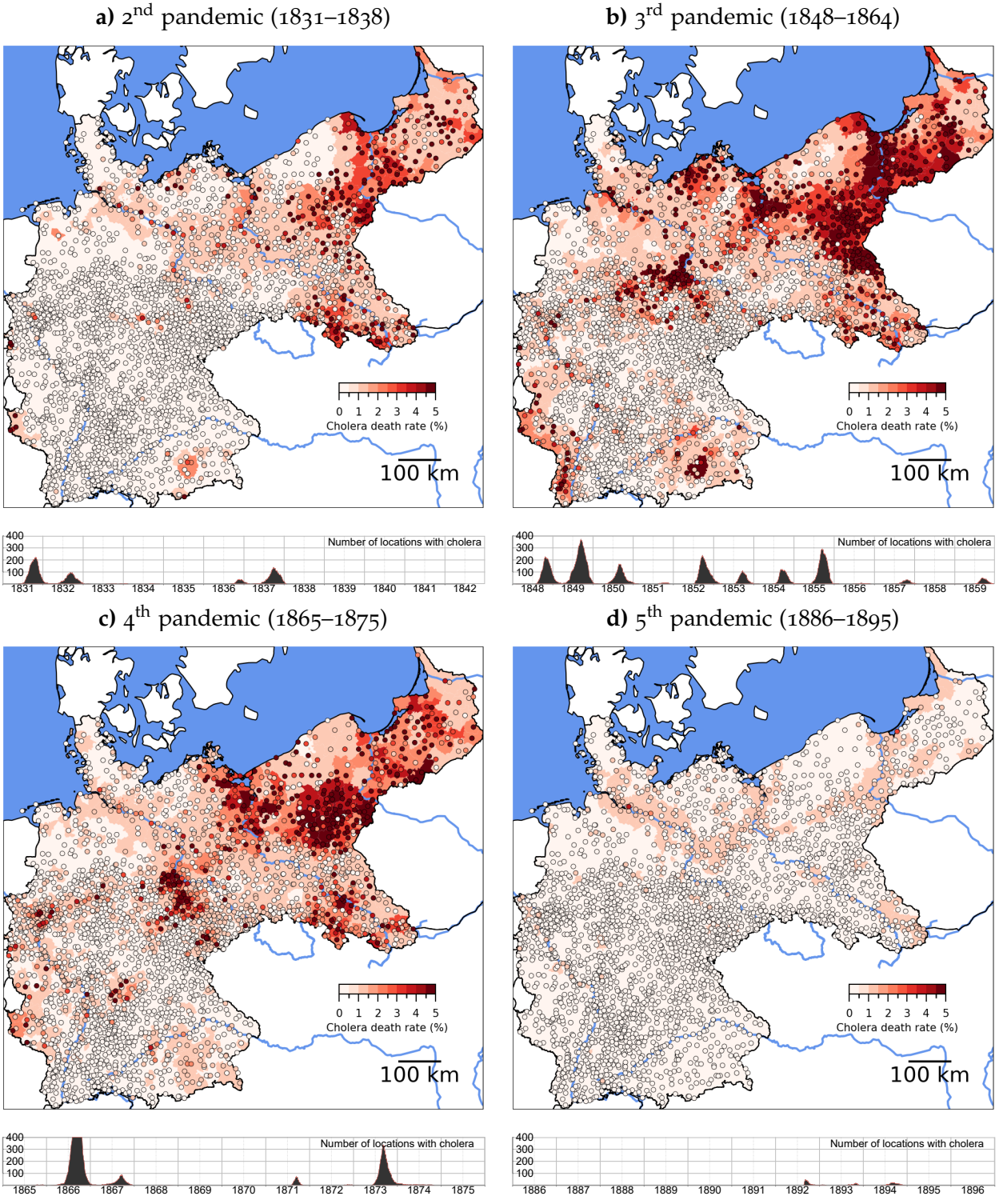
Figure 1: Cholera mortality in Germany and neighboring countries, 1820–1914



Note: Panel a shows annual all-cause deaths per 100 for Germany in 1871 borders. Years in which cholera deaths account for at least 1% of all deaths are depicted with a red square marker. The dashed line results from a local polynomial regression. Vertical bars in the lower part of the figure are proportional to the number of cholera deaths in each year; the associated labels give the death count. All-cause deaths and population counts are calculated from Mitchell (1975, 104–116). Panel b shows cholera deaths per 100 per pandemic for seven historical European states. The population at risk is measured in 1830, 1850, 1870, 1890, and 1910. The bottom-right bar chart presents the population-weighted average over pandemics per country.

1831–1838. However, by the 4th pandemic, Germany's incidence surpassed that of France and Britain, placing it in a middle position. Like most of Western Europe, Germany saw

Figure 2: The spatial and temporal distribution of cholera in Germany



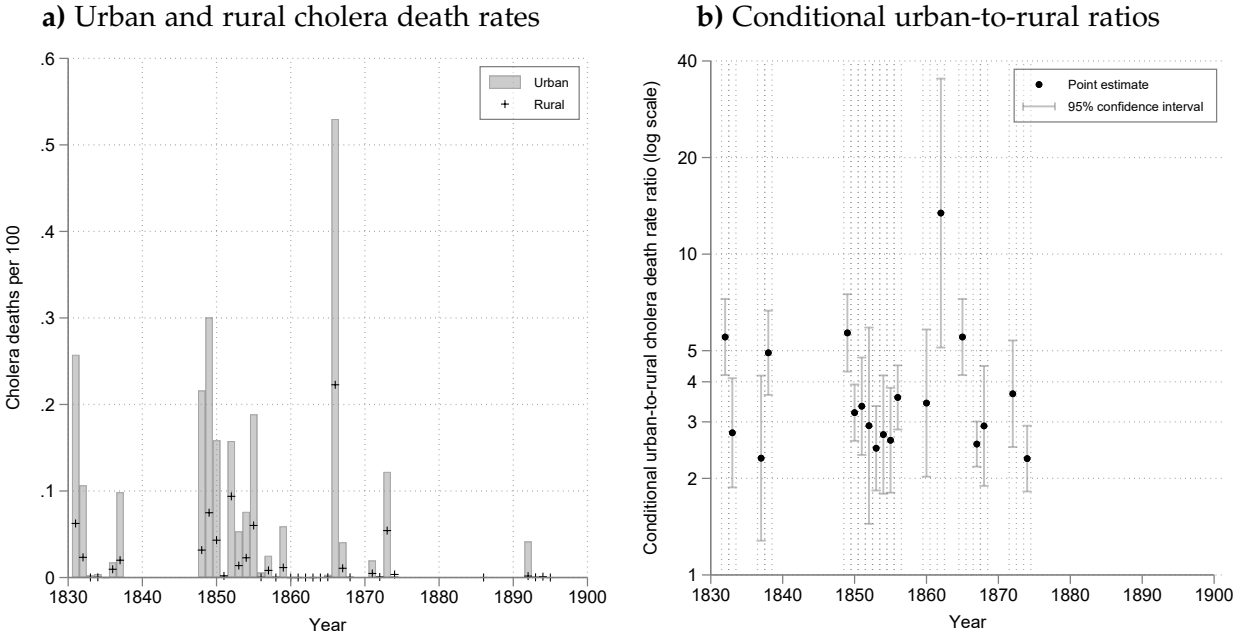
Note: Each panel shows maps of 2,685 cities and 852 rural counties in Germany, colored by cholera mortality, calculated as cholera deaths divided by mean population during each pandemic. Black lines indicate national borders; blue lines mark major rivers (Rhine, Elbe, Oder, Vistula, and Neman). Below each map, a histogram displays the number of locations with active outbreaks per day during the pandemic. The Oct. 1866 epidemic peak in panel c, with over 800 active locations, is cropped to better show variation across other pandemics.

little cholera activity during the 5th and 6th pandemics (1881–1896, 1899–1914). Aggregating across all pandemics, Germany's overall cholera incidence was at the lower end.

Turning to the spatial and temporal patterns within Germany, figure 2 maps variation in outbreak intensity across Germany during the four major pandemics. Below each map, histograms show the number of locations (cities and rural counties) with active cholera cases by day, illustrating the temporal trajectory of outbreaks. These graphs highlight both commonalities between pandemics, and significant spatial and temporal heterogeneity across them. The remainder of this section condenses these patterns into five stylized facts.

Cholera was disproportionately urban. Cholera was predominantly an urban phenomenon with city death rates consistently outpacing rural ones—at times by an order of magnitude. Figure 3 presents two complementary measures for this "urban cholera penalty".

Figure 3: Urban excess cholera mortality over time



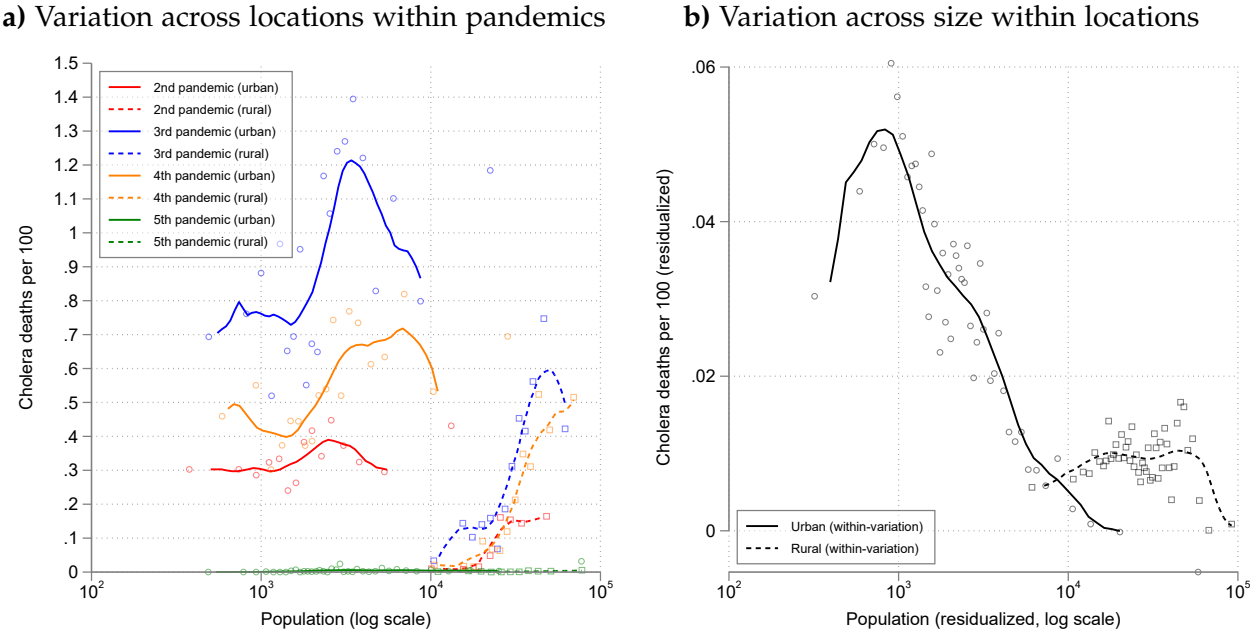
Note: Panel a displays annual average cholera death rates as percentages in urban areas (gray bars) and rural areas (black pluses). Panel b plots exponentiated coefficient estimates from a panel regression of logged death rates on a year-interacted urban dummy variable, controlling for time and county fixed effects. For example, a value of 3.5 indicates that cholera death rates were 3.5 times higher in urban areas than in their immediate rural hinterlands, conditional on time-constant cross-location heterogeneity and overall time trends. Gray spikes in panel b denote 95% confidence intervals, and coefficients with a significance level below 1% are omitted. Ratios are displayed on a logarithmic scale for clarity.

Panel a of figure 3 displays the annual average cholera death rate (in %), separately for urban and rural areas. Urban areas consistently exhibited much higher mortality rates than rural areas. This difference scaled with overall cholera severity and was most pronounced during major outbreaks (e.g., 1831, 1849, 1852, and 1866). Panel b offers a refined perspective by showing conditional urban-to-rural cholera death rate *ratios*. These are estimated in a regression model that compares cities specifically to their immediate rural hinterlands. This

approach controls for shared local conditions—geography, climate, and some socioeconomic factors—and isolates the urban-specific cholera penalty (see figure note for details). On average, urban areas experienced cholera death rates 3.5 times as high as those in their rural surroundings. This *relative* "urban penalty" remained mostly stable over time but reached a factor of 15 during the particularly devastating 1866 outbreak.

Mid-sized cities were most affected. Not all cities were affected equally by cholera; population density played a critical role. Panel a of figure 4 presents binned scatter plots showing the variation in cholera death rates across city size, separately for each pandemic. Polynomial regression fits (thick lines) exhibit an inverse U-shape, particularly during the 2nd and 3rd pandemics (1831–1838 and 1848–1864, respectively). Death rates peaked in mid-sized cities with approximately 3,000 inhabitants, while both smaller and larger cities experienced significantly lower mortality. In contrast, rural areas (dashed lines) displayed a more monotonic upward trend, with higher death rates in more populous rural regions.[17]

Figure 4: Population size and cholera mortality



Note: Panel a shows average cholera death rates by population size bins, separately for urban (circles and solid lines) and rural (squares and dashed lines) areas. Pandemic-specific death rates were calculated by pooling all cholera deaths and dividing by mid-pandemic population. Panel b shows *residualized* cholera death rates, pooling over all years (1831–1895), but controlling for location and region-specific time fixed effects. This reflects within-location differences over population size levels, abstracting from time-constant local factors and shocks common to all locations in a region. Fit lines stem from local polynomial regressions, and population size is shown on a logarithmic scale for clarity.

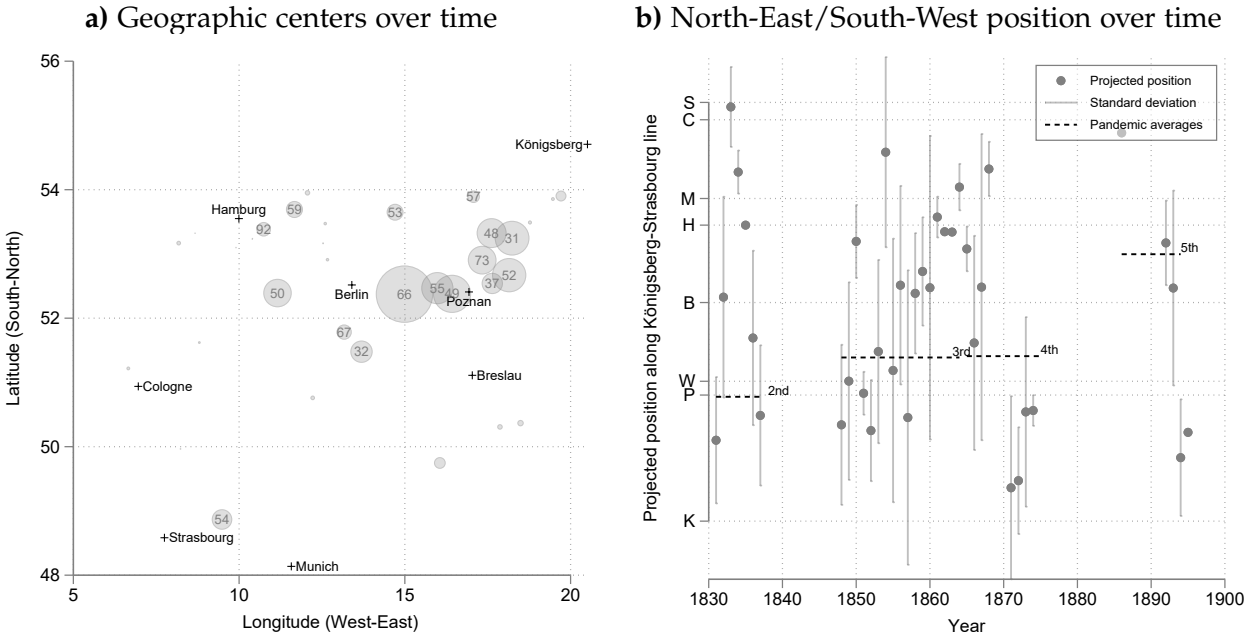
Panel b of figure 4 shifts the focus from comparing differently sized locations during a

17. Population size should be interpreted as an imperfect proxy for population *density*, as systematic data on cities' areal extent is unavailable. For rural areas, population density can be computed but is less informative due to substantial variation in the share of uninhabited land.

given pandemic to comparing a single location to itself at different population size levels. Specifically, the panel shows residualized death rates and population levels after controlling for all time-constant location characteristics and region-specific time trends in a two-way fixed effects model. The inverse U-shape persists: for an average city, death rates increased with population size up to a threshold (1,000 inhabitants), then declining as population size grew larger. A similar, though less pronounced, pattern is visible for rural areas.

Cholera began in the North-East and shifted South-West over time. Figure 2 highlights a strong Eastern bias in cholera exposure, with high mortality clusters in Imperial Germany's easternmost territories, particularly around Central Poland. Figure 5 further explores this geographical bias and reveals a gradual South-West shift in cholera's epicenter over time.

Figure 5: Cholera was heavier in the North-East, but shifted West over time



Note: Panel a shows the geographic centers of annual outbreaks, computed as mean latitude and longitude coordinates, weighted by the cholera death rate. Circle sizes indicate the overall severity of each outbreak, with larger circles representing more deadly years. For major outbreaks, the year is also labeled within the circles (e.g., "50" for 1850). Black crosses mark the locations of eight large cities for reference. Panel b projects the annual geographic centers onto a diagonal axis from Königsberg in the North-East to Strasbourg in the South-West. The gray spikes depict the average bidirectional squared deviations from this projected center, capturing the spatial dispersion of each year's outbreak. Horizontal dashed black lines show pandemic-specific average positions, weighted by each epidemic's overall death rate. Letters on the vertical axis indicate city locations: **K**önigsberg, **P**oznan, **W**rocław (Breslau), **B**erlin, **H**amburg, **M**unich, **C**ologne, and **S**trasbourg.

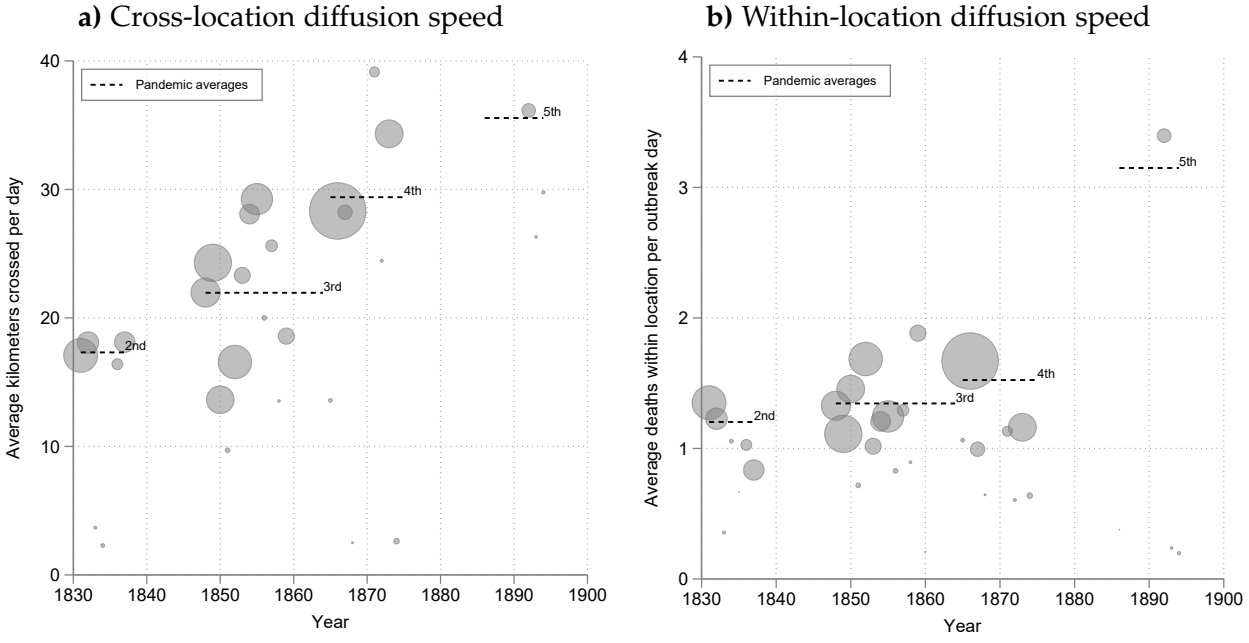
Panel a of figure 5 shows the geographic center of each outbreak, calculated as the mean latitude-longitude coordinate of all affected locations, weighted by cholera death rates. Most outbreaks were concentrated in Germany's North-Eastern provinces near Poznan, including the most severe ones (e.g. 1831, 1848/49, and 1866). However, secondary waves following major outbreaks (e.g., 1832, 1850, and 1867) shifted toward Central Germany, while smaller

outbreaks in 1853, 1859, and 1892 focused on Northern Germany. The 1854 outbreak, centered in the South-West (Bavaria and Alsace-Lorraine), stands out as a notable exception.

Focusing on the most apparent spatial trend, panel b projects the geographic centers onto a diagonal axis running from North-Eastern Königsberg to South-Western Strasbourg (see figure 5 note for details). Although year-to-year variation (gray points) and within-year dispersion (gray spikes) are substantial, the pandemic-specific averages (dashed black lines) reveal a clear South-West drift over time. Each pandemic saw distinct outbreaks in the North-Eastern territories, but cholera's overall geographic focus moved West over time.

Cholera's diffusion speed increased across and within locations. Figure 6 examines the temporal dynamics of cholera's diffusion, showing how its spread accelerated both across space and within locations over the 19th century. While cholera death rates declined over time, diffusion speed increased, indicating growing efficiency in outbreak propagation. Panel a measures cross-location diffusion speed as the average kilometers traveled per day by cholera in each year (see figure 6 note for details). Dashed black lines, indicating averages per pandemic, suggest acceleration over time. In the 1830s, cholera traveled at an average speed of 18 km/d. In 1892, this had increased to 36 km/d. Circles are proportional to the severity of each outbreak, indicating that deadlier outbreaks also diffused faster.

Figure 6: The acceleration of cholera diffusion across and within locations

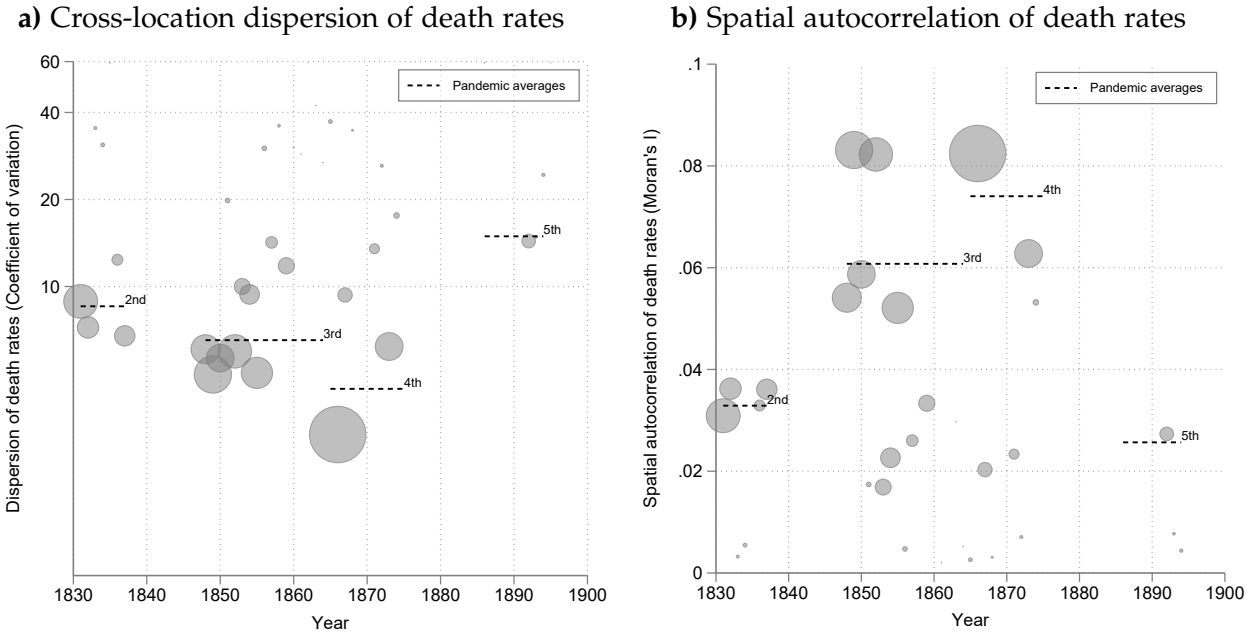


Note: Panel a shows cross-location diffusion speed, i.e. average location-pairwise distance (in kilometers) divided by the time (in days) between the start of their outbreaks. Panel b depicts within-location diffusion speed, measured as the average number of deaths per day during an outbreak spell. Circle sizes in both panels are proportional to the overall death rate; dashed black lines indicate pandemic-specific averages, weighted by each outbreak's overall death rate. Uncertain dates have been approximated, with "early", "mid", "late", and "unknown" days within a known month coded as days 5, 15, and 25, and 15. Results remain similar when these observations are dropped instead. Observations with unknown outbreak months have been dropped.

Panel b of figure 6 focuses on *within*-location diffusion speed, measured as the average number of deaths per day within each affected location during an outbreak spell. Similar to cross-location trends, within-location diffusion also accelerated over time, though less dramatically. In earlier outbreaks, daily deaths averaged around 1.2, but by 1892, this number had almost tripled, indicating more intense and faster local outbreaks despite the overall reduction in mortality. Circle sizes again correspond to outbreak severity, reinforcing the observation that larger and deadlier outbreaks were associated with faster diffusion.

Outbreaks grew more uniform across locations but clustered in space. Figure 7 asks how similar local cholera outbreaks were across locations. In panel a, the coefficient of variation (CV) measures the relative dispersion of cholera death rates across locations within a given year. The decline in the CV over time suggests that locations became more similar in terms of local outbreak intensities. This trend suggests an overall convergence in the severity of cholera's impact across locations over the 19th century, except for the last epidemic, which was highly dispersed with its disproportionate impact on Hamburg.

Figure 7: The cross-location similarity of outbreaks



Note: Panel a shows outbreak-specific coefficients of variation (CV) for death rates, i.e. their standard deviation over their mean. A higher CV suggests a more dispersed distribution. Panel a uses a logarithmic scale for clarity. Panel b shows outbreak-specific Moran's I estimates for death rates, computed using inverse straight-line distance weighting. A higher Moran's I suggests stronger spatial clustering of similarly affected locations. Circle sizes in both panels are proportional to the overall severity of each outbreak; dashed black lines indicate pandemic-specific averages, weighted by each outbreak's overall death rate.

Focusing on the spatial dimension of dispersion, panel b of figure 7 plots annual Moran's I values for cholera death rates. These measure the spatial autocorrelation of outbreaks, with higher values indicating stronger spatial clustering of similarly impacted locations.

While the variation over time is high, pandemic-wise averages (black dashed lines) suggest that highly impacted locations increasingly clustered in specific regions rather than being more evenly distributed across space.[18] The opposing patterns in panels a and b suggest a complex trend with respect to the concentration of outbreaks. While locations generally converged in terms of outbreak severity (via the CV), outbreaks also became more spatially clustered over time (via Moran's I).

4 Cholera, society and the economy: New data, new research

The previous section discussed the spatiotemporal patterns of Germany's cholera epidemics in abstract terms. To better understand these patterns, they must be contextualized within the broader social, economic, and political transformations of the 19th century. Such change expressed itself in numerous dimensions: Germany, like other Western countries, underwent industrialization and urbanization (Pfister 2022; Lehmann-Hasemeyer and Wahl 2024), demographic change (Pfister and Fertig 2020), growing market integration (Hornung 2015; Wolf 2009), expanding international trade (Hungerland and Wolf 2024), mass migration (Bräuer and Kersting 2024), and increasing cultural homogenization (Kersting and Wolf 2024). These trends influenced and were influenced by epidemic dynamics. For instance, cholera's disproportionate impact on mid-sized cities suggests that early urban growth came with severe health costs before ultimately laying the groundwork for effective sanitation as cities expanded. Moreover, the accelerating diffusion of cholera, along with its gradual westward shift, indicates that transport networks, trade integration, and internal migration changed epidemic patterns in complex ways.

Exploring these and other claims within a rigorous quantitative framework remains an open challenge for future research. I conclude the paper by highlighting five major narratives and ongoing interdisciplinary research programs that explore cholera's relationship with broader societal transformations. What unites these perspectives is not a lack of compelling hypotheses, theoretical foundations, or anecdotal evidence, but rather the absence of systematic engagement by quantitative social scientists—largely due to the scarcity of structured historical data of the kind presented in this paper.

The origins of public health and sanitary innovation Pandemic shocks are often associated with significant innovations, investments, and institutional reforms. However, the conditions under which such changes occur and the mechanisms driving them remain contested. Quantitative-causal frameworks are well-suited to investigate and generalize these

18. Note that the high number of non-affected locations in every epidemic does not affect Moran's I's interpretation as a measure of spatial autocorrelation. To calculate Moran's I, observed spatial patterns are compared to counterfactual scenarios of randomness, where the distribution of realized local death rates is spatially permuted. Locations with zero-death rates enter this randomization like any other observation. I obtain similar values when all zero-rate observations are dropped.

dynamics. For example, recent studies suggest the 1918/19 Influenza pandemic spurred formal healthcare expansion (Esteves et al. 2022), reinforced religious and scientific convictions (Berkes et al. 2024), and undermined social trust (Aassve et al. 2021).[19] Cholera historiography contains similarly intriguing claims that, however, have not yet been explored by quantitative social scientists.

Historians highlight 19th-century cholera epidemics as pivotal to the rise of public health and major sanitary investments (Hamlin 2009b).[20] Yet, key aspects of this narrative remain unclear. First, cholera's perceived importance often exceeded its actual mortality impact relative to endemic diseases like typhoid (Pelling 2022). Second, while rich anecdotal evidence links cholera to public health advocacy and reform, these processes spanned decades, often extending beyond cholera's major mortality episodes (Hennock 2000). Finally, the failure to replicate similar "sanitary revolutions" outside the Western world suggests that factors such as ideology, political institutions, economic incentives, and medical knowledge mediate the cholera-sanitation nexus (Hamlin 2009a; Kappner 2024).

In contrast to the now decade-spanning literature on Influenza and the Black Death, researchers have only very recently begun to explore cholera's role driving role in public health in settings that abstract from single case studies. For example, Gallardo-Albarrán and Kappner (2025) identify capital-skill production complementarities as a main driver of elites' willingness to finance sanitary infrastructure in face of cholera epidemics. Petroff (2024) finds that 19th century British expert's opinions on the contagiousness of cholera were shaped by their economic stakes in oversea trade relations.

Epidemic shocks, political crisis, and social change Epidemic shocks can ignite far-reaching political and social dynamics, though their directions and magnitude are difficult to predict as demonstrated by the COVID-19 pandemic (Eichengreen et al. 2024). Cholera, due to its salience, wide geographic spread, and focus on modern mass societies, provides a promising historical lens to examine how epidemics shape society and politics. Recently, Jedwab et al. (2021) and Cordell et al. (2023) emphasized cholera's role as one of the most conflict-prone epidemics in history, unleashing social clashes, scapegoating, and the infamous "cholera riots" across Europe and the world (Cohn 2017). This perspective builds on an earlier tradition that, starting with McGrew (1960), has linked 19th-century European cholera epidemics to revolutionary and disruptive events, identifying them as both catalysts for and amplifiers of political change (Briggs 1961; Rosenberg 1966; Evans 1988; Briese 2003).

19. See Beach et al. (2022) for a survey of Influenza's societal effects. Similarly, literature links the 1347–1353 plague to institutional reforms across Europe, and smallpox to the adoption of vaccination, particularly in military contexts.

20. The effectiveness of these investments, especially waterworks and sewers, in reducing mortality and waterborne diseases has been demonstrated in quantitative studies for the US (Ferrie and Troesken 2008; Alsan and Goldin 2019), Britain (Aidt et al. 2023; Davenport et al. 2019), and Germany (Vögele 1998; Spree 1998; Gallardo-Albarrán 2020; 2024b). See Gallardo-Albarrán (2024a) and Harris and Helgertz (2019) for surveys.

Cholera also played a role in major international turning points, such as the 1830/31 Polish November Uprising (Ross III 2015), the 1848 Revolutions and civil wars (Fazekas 2024), and the Crimean War (1853–1856) (Smallman-Raynor and Cliff 2004). On a brighter note, Europe's shared exposure to cholera spurred early international sanitary cooperation, laying the groundwork for the eventual creation of the World Health Organization (Lee and Dodgson 2000; Harrison 2006; Huber 2006; 2020).

Despite this rich historical backdrop, scholars have barely begun to explore cholera's sociopolitical impacts within explicitly quantitative-causal frameworks. A comparison to the well-studied 1918/19 Influenza pandemic demonstrates the dormant potential. For example, Bauernschuster et al. (2025) and Foertsch and Roesel (2023) show that exposure to the pandemic caused voters in Weimar Germany to reward parties seen as competent in public health while punishing governments that demonstratively neglected health infrastructure.

Globalization, growth, and epidemic burden The relationship between trade, globalization, and the spread of epidemic diseases has long intrigued economists and gained renewed attention during the COVID-19 pandemic (Boerner and Severgnini 2012; Antràs et al. 2023). Cholera, in particular, is often described as *the* disease of the First Globalization, with its pandemics closely tied to the expansion of global trade networks during the 19th century (Harrison 2013; 2015; Webb 2015).[21] Within countries, growing transport connectivity and labor mobility also facilitated the spread of diseases (Pyle 1969; Patterson 1994; Tang 2017; Bogart 2022).

Cholera's wide geographic reach was undeniably a byproduct of globalization—and, by extension, economic progress. Yet, it has also been portrayed as *the* symbol of pre-sanitary poverty, disproportionately affecting dense and impoverished urban areas (Evans 1988; Davenport et al. 2019; Glaeser and Cutler 2021). A useful framework to further explore this duality is provided by Troesken (2015), who distinguishes between "diseases of poverty", such as typhoid, and "diseases of commerce", such as smallpox. Institutional features conducive to economic growth—such as trade openness and labor mobility—can simultaneously mitigate poverty-related diseases while exacerbating commerce-related ones.[22] Cholera occupies an dynamic position within this dichotomy: while trade and connectivity facilitated its spread, it also spurred sanitary investments in highly integrated economies like Germany, where cities grew rich from trade and labor inflow.

The relationship between epidemic shocks, economic activity, and trade restrictions has been studied extensively in the context of the 1918/19 Influenza pandemic. For instance,

21. Recognizing cholera's connection to commerce, 19th-century governments initially imposed deeply unpopular quarantine and border control measures, though these were ultimately ineffective (Baldwin 1999; Poczka 2017). This tension between cross-border economic activity and epidemic containment gained new relevance during the COVID-19 pandemic (Eckardt et al. 2020).

22. This framework has since been applied to interpret government policies during the COVID-19 pandemic (Koyama 2021).

Boberg-Fazlic et al. (2021) argue that exposure to the Influenza pandemic led to increases in tariffs, while Clemens and Ginn (2020) find that migration barriers failed to limit mortality during four historical Influenza pandemics, including 1918/19. Correia et al. (2022) show that interaction-reducing non-pharmaceutical interventions (NPIs) in the US did not suppress economic activity beyond the direct costs of Influenza itself. Finally, Clay et al. (2018) and Franke (2022) show that local Influenza outbreaks were severely amplified by industrial atmospheric pollution. Despite cholera's similarly complex entanglement with trade and economic growth, similar empirical studies do not yet exist.

The macroeconomic effects of epidemic shocks How do epidemic shocks impact macroeconomic variables such as real wages and output? The COVID-19 pandemic has revived interest in whether evidence from historical epidemics can inform predictions about the macroeconomic consequences of future outbreaks (Eichenbaum et al. 2021; Donadelli et al. 2021; Jordà et al. 2022). Much of this literature focuses on the 1918/19 Influenza pandemic and the Black Death, with cholera receiving relatively little attention (Arthi and Parman 2021; Beach et al. 2022; Doran et al. 2024; Callegari and Feder 2022).[23]

Overlooking cholera in this context represents a missed opportunity. Unlike pre-modern plague epidemics, 19th-century cholera outbreaks occurred during a period of economic transition, affecting both Malthusian economies and those shifting toward modern economic institutions characterized by economies of scale, human capital accumulation, and sustained growth.[24] Moreover, in contrast to the 1918/19 Influenza pandemic, which saw widespread use of non-pharmaceutical interventions (NPIs), cholera epidemics largely played out in a "laissez-faire" policy environment, with few interventions apart from the initial quarantines and border control instances noted earlier (Baldwin 1999; Evans 1987).[25] Cholera epidemics also differed in their temporal dynamics, unfolding over multiple waves of varying magnitude across the 19th century, rather than constituting a one-time shock.

Potential avenues of research extend beyond real wages and output. For instance, Alfani (2022) provides evidence that cholera epidemics increased inequality in 19th-century France. Ogasawara and Inoue (2021) find that early-life exposure to cholera in Japan resulted in significant long-term health costs. Finally, Franck (2024) shows that cholera-induced labor scarcity stimulated technological innovation in the French agricultural sector. These find-

23. A notable exception involves urban housing markets: Ambrus et al. (2020) show that exposure to the 1854 Soho cholera outbreak in London persistently depressed local property values, while Francke and Korevaar (2021) document temporary declines in house prices following cholera shocks in 19th-century Paris.

24. The economics literature on the Black Death is summarized by Jedwab et al. (2022). Central themes include wage and output dynamics within Malthusian economies (Clark 2016; Jedwab et al. 2024), long-term demographic effects (Voigtländer and Voth 2013a; 2013b), and the influence on political institutions like serfdom (Bosshart and Dittmar 2024; Raster 2023).

25. The literature on the 1918/19 Influenza pandemic has extensively analyzed NPIs, especially in the US. For example, Correia et al. (2022) argue that NPIs did not reduce industrial production; Berkes et al. (2024) find no harm to innovation activity from social distancing; and Ager et al. (2024) suggest school closures had limited effects on educational attainment.

ings, which plausibly echo expected macroeconomic effects of epidemic shocks in modern contexts (e.g., COVID-19), highlight the broader potential of studying cholera to understand how economies respond to epidemics.

Cholera, climate change and resource management Cholera remains a significant global threat. Studies suggest that it may become more severe in the future as climate change expands cholera's oceanic habitats, clean water resources grow scarcer, and urbanization accelerates (Girotto et al. 2024). However, many of the hydrological, climatic, and socioeconomic factors that influence cholera evolve slowly over time. Moreover, epidemiological risk assessment models require extensive data. For these reasons, historical quantitative evidence plays an increasingly important role in outbreak modeling and speculation about cholera's future epidemic significance.

Of course, the limitations of historical comparisons must be acknowledged. Societal and economic structures have changed significantly, with important implications for cholera epidemiology (Hamlin 2009a). The modern El Tor biotype differs from earlier cholera strains in both longevity and fatality, and treatment methods have improved and become more accessible (Hsiao et al. 2018). However, these limitations must be weighed against the opportunities historical research provides. Historical data offers rich variation across time and space, including fluctuations in climate and geography that contemporary data cannot deliver. A recent example of the insights that historical data can generate is Shackleton et al. (2023), who study the relationship between changing climate conditions and cholera in Kolkata over 120 years. Other examples are the use of historical records from London, Baltic port cities, and Denmark to estimate key parameters and transmission mechanisms within epidemiological models (Tien et al. 2011; Chan et al. 2013; Phelps et al. 2017; Phelps et al. 2018).

5 Conclusion

This paper delivers a comprehensive quantitative account of 19th-century cholera mortality in Germany, documenting nearly half a million deaths across 2,685 cities and 852 rural counties. Cholera emerges as an important contributor to the mortality transition in Germany, with mortality levels comparable to those in other Western European countries and of similar quantitative importance to more extensively studied pandemics, such as the 1918/19 Influenza pandemic. The paper distills cholera's complex spatiotemporal dynamics into five stylized facts: cholera was predominantly urban, with city death rates averaging 3.5 times higher than rural ones; mid-sized cities were the hardest hit; the disease's geographic center shifted from the North-East to the South-West over time; outbreaks spread more rapidly across regions and within locations in later pandemics; and cholera mortality became increasingly spatially clustered, despite a decline in overall death rates.

The complex patterns identified in this study are not conclusions in themselves but a starting point for a more systematic engagement with the rich qualitative cholera historiography. Over decades, social and medical historians, demographers, and geographers have linked cholera to major modernization processes, including public health reform, political change, globalization, and economic development. This literature is rich in ideas, but rests primarily on case studies and anecdotal evidence as comprehensive data on historic cholera epidemics is scarce. As demonstrated with other pandemics–such as the Black Death and Influenza–quantitative social scientists, including economic historians, can substantially advance this literature by applying causal frameworks and quantitative methods to high-quality datasets like the one presented here.

To motivate such research, I outline five major "cholera narratives" that imply causal claims deserving empirical, data-driven engagement. First, cholera is framed as crucial catalyst for sanitary investments, medical research and the development of epidemiological methods. The political motivations, economic incentives, and cognitive frameworks driving the relevant decision makers remain vague and controversial, however. Second, cholera is associated with political crises, wars, and social unrest, such as the 1848 revolutions and "cholera riots" throughout the 19^{th} century. To what extent these events were driven by, rather than directly driving cholera outbreaks warrants further analysis. Third, cholera exemplifies the epidemic risks of globalization, having spread along trade and migration routes and prompting limited but important experiments in quarantine, standardized reporting, and international health coordination during the First Globalization. Fourth, the disease's economic impacts, including potential effects on wages, inequality, and technological change, remain largely unexplored despite cholera's occurrence at a key transitional period from Malthusian stagnation to modern economic growth. Lastly, cholera is highly sensitive to changing climatic and environmental conditions. Analysis of historical epidemic patterns, including their integration in modern epidemiological frameworks, should advance our understanding of cholera's future.

As highlighted by these examples, the relevance of a data-driven research agenda extends beyond historical analysis. Globalization, urbanization, and climate change continue to shape epidemic risks, and cholera remains a persistent threat in many developing regions. Insights gained from historical cholera epidemics can inform contemporary policy debates on public health resilience, crisis management, and the trade-offs between market integration and epidemic risk. Future exploration of Germany's comprehensive 19^{th}-century cholera history—based on the dataset presented in this paper—will deepen our understanding of how societies generate, and respond to health shocks. This research agenda can build on cholera's extensive qualitative historiography, more developed literatures such as those on the Black Death and Influenza, and growing interest from the modern public health and epidemiology community.

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A Dataset validation

In this section, I validate the German cholera dataset against three independent sources of information. First, I demonstrate that regional annual excess mortality between 1816 and 1914 is highly predictive of cholera epidemics identified in the dataset. Second, I show that cholera events and associated death rates are well-predicted by an established risk model, based on the interaction of climate and water supply facilities. Third, I compare mentions of cholera events between the dataset and the German city encyclopedia (*Städtebuch*), documenting a high degree of agreement between both sources.

Regional excess mortality strongly correlates with cholera epidemics. Excess all-cause mortality can be a useful proxy for cause-specific epidemic death counts. To assess excess mortality, I collect population and all-cause deaths data for each state in the dataset (see appendix section B for details). Given their size, each Prussian district is treated as a separate "state", resulting in 63 regional entities. All-cause mortality, derived from vital statistics, are available annually from 1816 to 1914, though some states have gaps in early years.[26]

I regress annual deaths $D_{r,t}$ on state-specific base levels and time trends, adjusting for the population at risk $P_{r,t}$ within a conditional exponential means model. Due to the observed mortality transition—a sudden, consistent downward trend in overall mortality—, I allow the time trends to vary around a state-specific break year B_r, estimated through Supremum Wald tests. The regression model is as follows:

$$D_{r,t} = \exp\left(\alpha_r + \beta_r^{\text{Base}} \times t + \beta_r^{\text{Post}} \times t \times \mathbb{I}[t \geq B_r]\right) \times P_{r,t} + \varepsilon_{r,t} \, .$$

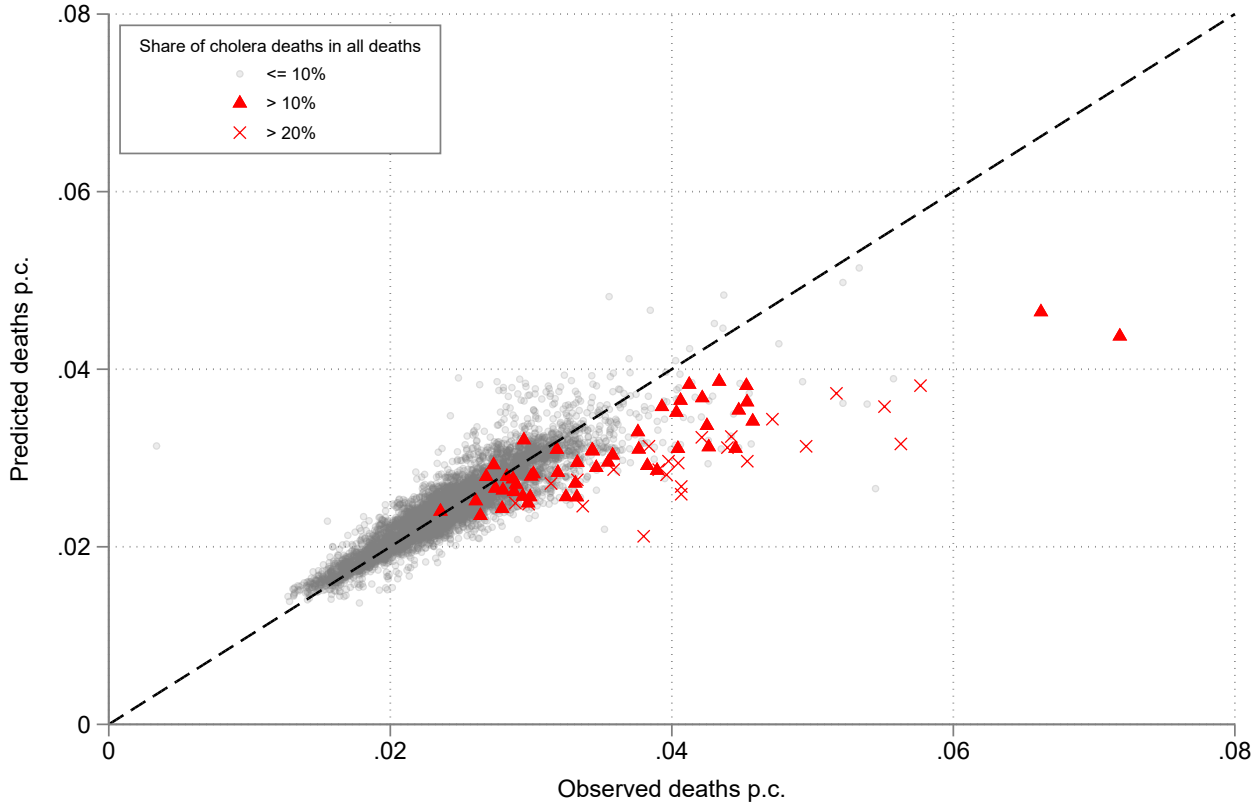
Using the fitted regression model, I obtain *predicted* deaths $\hat{D}_{r,t}$ for each state-year.

In figure 8, I compare these predictions to observed deaths, expressed in per-capita terms to accommodate different state sizes. The tight, symmetric dispersion around the 45°line indicates that the observed mortality dynamics between 1816 and 1914 are well captured trough state-specific time trends, allowing for differential onset of the mortality transition. However, observations further away from the identity line indicate unexpected excess or deficit mortality (below or above the 45°line, respectively). Among these, I highlight state-years with cholera accounting for more than 10% (triangles) or 20% (crosses) of the observed deaths, according to my dataset. Almost all of these "cholera years" show high excess mortality, among them the most extreme years.[27]

26. Data is available for 5753 out of 6237 possible state-year cells. Population counts are typically measured at three- or five-year steps. To estimate population between census years, I apply log-linear interpolations.

27. While almost all "cholera years" are years of high excess mortality, the reverse is not true. This is because other epidemics, such as the 1870–1873 smallpox outbreak, also generated excess mortality in some years. The four state-years where cholera accounted for more than 10% of all deaths but did *not* result in all-cause excess mortality are 1849 (Posen and Trier districts in Prussia), 1853 (Lübeck), and 1866 (Trier district in Prussia).

Figure 8: Excess mortality and "cholera years"



Note: This graph plots observed per-capita deaths for 5753 state-year observations against predicted deaths (also in per-capita terms) according to a time trend regression model (see text). Observations highlighted in red are "cholera years", in which cholera accounts for more than 10% (triangles) or 20% (crosses) of all deaths. Out of the 49 (24) state-years in which cholera accounted for 10–20% (more than 20%) of all deaths, 45 (24) state-years also experienced excess mortality, i.e. more observed deaths than predicted deaths.

Climate and infrastructure risk factors predict cholera outbreaks. I test whether the observed mortality patterns align with established environmental and infrastructural risk factors. Studies demonstrate that cholera incidence correlates with climatic, hydrological and infrastructural conditions. Most evidence comes from coastal and tropical areas near cholera's endemic regions, such as India and East Asia (Akanda et al. 2011; Xu et al. 2015), but comparative studies from Africa, the Arabian Peninsula, and the Caribbean have also linked cholera risk to environmental factors (Rebaudet et al. 2013; Eisenberg et al. 2013). Among the most established risk factors are high temperatures, heavy rainfall, and contaminated water supplies (Campbell et al. 2020; Escobar et al. 2015).[28]

The specific role of these three factors can vary substantially across locations, time and context.[29] However, at the most basic level, a large body of evidence links cholera risk

28. Apart from environmental risk factors, a large literature is concerned with individual, social and human-made risk factors like poverty. While these should not simply be seen as proximate factors whose role could be reduced to more fundamental geological risk factors, I have no data to incorporate them into my framework.

29. For instance, specific drivers may differ between endemic and epidemic settings (Pascual et al. 2002; Shackleton et al. 2024; Ruiz-Moreno et al. 2007). In epidemic regions, rainfall may facilitate bacterial spread,

to warm temperatures, heavy rainfall and poor sanitary conditions (Fernández et al. 2009; Herrador et al. 2015; Brumfield et al. 2021; Usmani et al. 2021; Shackleton et al. 2024; Sikder et al. 2023).[30] Jutla et al. (2013) present a simple integration of these risk factors in epidemic (i.e. non-endemic) settings. Their framework posits that if all three conditions are met—high spring temperatures (inducing bacterial growth, low river levels and salination), followed by heavy summer rains (causing cross-contamination of water reservoirs), in regions with vulnerable water supplies—the likelihood of a cholera outbreak is elevated. If any condition is absent, cholera risk diminishes. This model's simplicity, focusing on a limited set of factors, allows for the inclusion of interactions and temporal structure while remaining tractable. Moreover, each factor has been tested across diverse settings (Usmani et al. 2021).[31]

I use Jutla et al.'s (2013) model to indirectly test the plausibility of the German cholera dataset: If the model captures basic epidemic probabilities, and the data is accurate, cholera epidemics should be most severe in locations and at times subject to the presence of all three risk factors. To test this implication across locations i (in regions r) and years, I define dummy variables for the *absence* of risk factors: Temperature risk is absent ($T_{i,t} = 1$) when spring temperatures are below their 15-years rolling average. Rainfall risk is absent ($R_{i,t} = 1$) when summer precipitation is below its 15-years rolling average. Finally, water quality risk is absent ($W_{i,t} = 1$) once a city builds a safe piped water supply.[32]

The estimation equation reads

$$C_{i,t,r(i)} = \sum_{j=1}^{7} \beta_j \cdot \mathbb{I}_j \left[T_{i,t}, R_{i,t}, W_{i,t}\right] + \alpha_i + \lambda_{r(i)\times t} + \varepsilon_{i,t} \ ,$$

where $j = 1, \ldots, 7$ represents the seven possible combinations of risk-lowering factors—low spring temperature (T), low summer rainfall (R), and safe water infrastructure (W)—with the absence of all three as the omitted base category. Here, C is either an outbreak dummy indicator or the cholera death rate. Location fixed effects (α_i) and region-year effects ($\lambda_{r(i)\times t}$) control for unobserved, time-invariant risk factors and regional shocks.

while in endemic areas, low river flows and tidal intrusion–facilitated by low precipitation–often play a more prominent role. Evidence suggests that heavy rainfall after a period of low river flow could also promote outbreaks in epidemic settings (Jutla et al. 2013). Variability in risk factors is also observed over time; environmental influences on cholera outbreaks in Bengal, for instance, shifted between the late 19th and 21st centuries, suggesting the pathogen's adaptability to changing conditions (Shackleton et al. 2023). Additionally, complex interactions among factors–often not fully captured in linear models–can lead to variable outcomes across regions (Pascual et al. 2002; Emch et al. 2008).

30. Some exceptions include the low precipitation in Iran's Qom region (Asadgol et al. 2019) or an inverse temperature relationship in 19th-century France (Franck 2024). Furthermore, incomplete sanitary infrastructure can *increase* mortality risk from waterborne diseases in some contexts (Kappner 2024).

31. Shackleton et al. (2024) offer a more refined model that allows for context-dependent effects of rainfall, but this is beyond the scope of my exercise here due to lack of data.

32. These location- and year specific dummies are constructed using data from Luterbacher et al. (2004) (temperature), Pauling et al. (2006) (rainfall), and Gallardo-Albarrán and Kappner (2025) (water supplies in cities). Data on supply structures in rural areas is not available—I assume that those were always subject to water quality risk. In table 2, I show that results hold when focusing on cities exclusively.

Table 2: Regression results from a cholera risk factor model (Jutla et al. 2013)

	Dep. var.: Outbreak (0/1)			Dep. var. Death rate (%):		
	(1) All locations	(2) Cities	(3) River cities	(4) All locations	(5) Cities	(6) River cities
Baseline rate						
All risk factors present	0.06***	0.05***	0.09***	0.02***	0.04***	0.05***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
Marginal deviation						
$T(\checkmark)R(\text{✗})W(\text{✗})$	-0.01	0.00	-0.01	0.00	-0.00	-0.01
	(0.29)	(0.68)	(0.44)	(0.60)	(0.71)	(0.78)
$T(\text{✗})R(\checkmark)W(\text{✗})$	-0.01*	0.01	-0.01	-0.00	-0.01	-0.04*
	(0.08)	(0.19)	(0.37)	(0.12)	(0.37)	(0.06)
$T(\text{✗})R(\text{✗})W(\checkmark)$	-0.02***	-0.04***	-0.04***	-0.04***	-0.02***	-0.02***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
$T(\checkmark)R(\checkmark)W(\text{✗})$	-0.00	0.02*	-0.01	0.01	-0.01	-0.02
	(0.95)	(0.06)	(0.62)	(0.40)	(0.48)	(0.45)
$T(\checkmark)R(\text{✗})W(\checkmark)$	-0.04***	-0.04***	-0.06***	-0.03***	-0.02*	-0.01
	(0.00)	(0.00)	(0.00)	(0.00)	(0.10)	(0.57)
$T(\text{✗})R(\checkmark)W(\checkmark)$	-0.02**	-0.02***	-0.03*	-0.04***	-0.02***	-0.06***
	(0.02)	(0.01)	(0.08)	(0.00)	(0.00)	(0.01)
$T(\checkmark)R(\checkmark)W(\checkmark)$	-0.01	0.01	-0.04*	-0.02**	-0.02	-0.03
	(0.53)	(0.58)	(0.06)	(0.03)	(0.12)	(0.19)
R-squared	0.63	0.63	0.83	0.34	0.40	0.78
Observations	229706	174261	19955	229706	174261	19955
Locations	3536	2683	307	3536	2683	307

Note: Table shows estimation results from a regression model that predicts cholera outbreaks or death rates based on dummy variables that indicate the absence of three risk factors: Low spring temperature (T), low summer precipitation (R), and safe water supplies (W). The baseline rate is the conditional outbreak probability (cols. 1–3) or death rate (cols. 4–6) in locations that have none of these three risk-lowering factors present. The coefficients listed as marginal deviations indicate the change to the baseline probabilities or rates under different combinations of risk-lowering factors, with ✓ indicating their presence. The "River cities" sample (cols. 3 and 6) includes all cities whose center is within one kilometer of a river. Estimates are conditional on location and region-year fixed effects. Locations are weighted by population to account for their varying size. Standard errors are clustered at the location level and presented in parentheses. See text for further details.

The regression results in table 2 indicate that locations meeting none of the three risk-lowering factors had an outbreak probability of 6–9% (cols. 1–3) and death rates of 2-5% (cols. 4–6), with cities showing higher cholera incidence (cols. 2 and 5), especially those whose center is within one kilometer of a river (cols. 3 and 6). Below the baseline rates, the table shows marginal deviations (percentage point changes) from these baseline outcomes when one or more of the three risk factors is *not* met. The model's prediction is that any absence of risk factors lowers cholera risk. The findings broadly support this prediction as

most coefficients are negative and many are significant.[33] This, in turn, indirectly validates the quality of the dataset employed for the estimation.

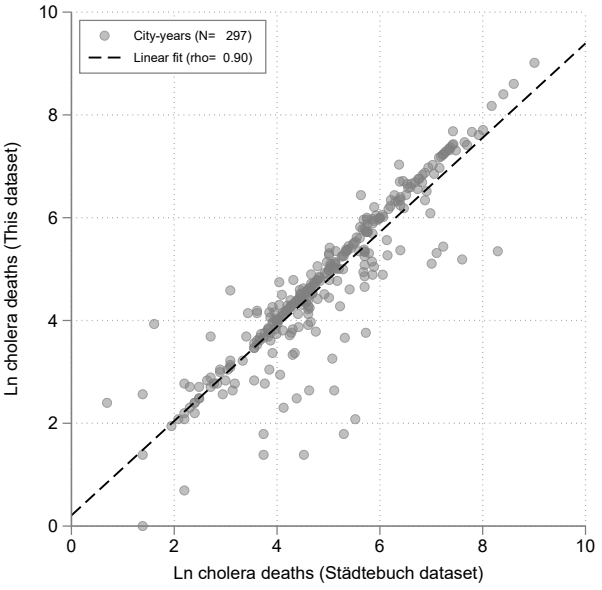
Cholera events in the dataset are well represented in the German city encyclopedia. While the dataset presented in this paper is the first systematic sources on cholera events in German cities, the multi-volume German city encyclopedia (*Städtebuch*) contains sections on historically significant epidemic events for a large sample of cities throughout history (Keyser et al. 1939–2003). I rely on a recent transcription of these books by Bogucka et al. (2019) and compare whether epidemic events mentioned in this source are also found in my dataset. This is a meaningful validation, because the mention of a significant cholera epidemic in the city encyclopedia indicates that the respective authors referenced this event across multiple sources and deemed the information reliable. In figure 9, I present several comparisons between both datasets.

Figure 9: Comparison of cholera event mentions and death counts with the *Städtebuch*

a) Crosstable of event counts

Outbreak according to *Städtebuch*	Outbreak according to this dataset: **A** Yes	**B** No	Σ
X Yes	545 0.36%	139 0.09%	684 0.45%
Y No	2,858 1.87%	149,273 97.68%	152,131 99.55%
Σ	3,403 2.23%	149,412 97.77%	152,815 100%

b) Scatter plot of logged death counts



Note: This figure presents a comparison between cholera epidemics data mentioned in the German city encyclopedia (Keyser et al. 1939–2003; Bogucka et al. 2019) and my dataset. Panel a is crosstable, counting city-year instances according to whether cholera outbreaks are mentioned in my dataset (column A) or not (column B), and whether they are mentioned in the city encyclopedia (row X) or not (row Y). For example, 545 outbreaks are mentioned in both sources, representing 0.36% of all city-year instances. Panel b plots death counts reported in both datasets, based on 297 cholera epidemics for which such data is provided in the encyclopedia. Epidemics that are located on the dashed black 45°line have the same magnitude reported in both datasets.

33. Coarse spatial resolution in climate data and simplified water infrastructure proxies likely add noise to the estimates. Moreover, the estimation approach presupposes that any outbreaks would occur in summer. This is true for most, but not all, outbreaks. Beyond statistical precision, the estimated coefficients should not be interpreted causally, though the model's fixed effects structure blocks many unobserved confounders.

In panel a of figure 9, I compare mentions of cholera events in 2,351 cities across both datasets. I register all "Cholera" and "Brechruhr" events in the city encyclopedia for the time span 1831–1895 (65 years), yielding 152,815 city-years.[34] The cross-tabulation shows that, out of the 684 outbreaks mentioned in the city encyclopedia, 545 are also featured in my dataset, suggesting that these sources broadly align. Moreover, a large share of the 139 events *not* listed in my dataset is explained by one-year deviations, e.g. when the same outbreak is coded under the years 1831 or 1832 in the two dataset, respectively. Beyond this "positive check", the cross-tabulation also implies that 2,858 outbreaks filed in my dataset are not mentioned in the encyclopedia. This is expected as reporting in the latter source depends on the subjective assessment of the respective authors, who often focused on "significant" epidemic events and did not necessarily aim at comprehensiveness. Given these limitations, the high odds ratio of agreement between both sources, $\frac{545/2858}{139/149273} \approx 19.1$, is a strong validation for my data source.

In panel b, I compare cholera death counts across both datasets for 297 city-years for which the city encyclopedia reports these. The high correlation (with an R-squared of 0.9) indicates agreement, although the city encyclopedia reports slightly higher death counts on average.

34. I discard all "Brechruhr" events registered before 1831, as these cannot relate to epidemic cholera. While my dataset includes 2,685 cities, some of them are not featured in the city encyclopedia. In particular, all cities in the territories Germany ceded to Poland immediately after World War 1 are not included.

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B Territory-by-territory sources list

The online appendix provides a detailed list of all data sources, territory by territory. It also highlights important assumptions, limitations and data anomalies. Furthermore, it prompts annual cholera death counts separately per territory, and visually contextualizes these cholera outbreaks within each territory's general mortality trend.

Please find the online appendix here.

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