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ORIGINAL ARTICLE

Poverty, pollution, and mortality: The 1918 influenza pandemic in a developing German economy

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Abstract

The paper provides a detailed analysis of excess mortality during the ‘Spanish Flu’ in a developing German economy and the effect of poverty and air pollution on pandemic mortality. The empirical analysis is based on a difference-in-differences approach using annual all-cause mortality statistics at the parish level in the Kingdom of Württemberg. The paper complements the existing literature on urban pandemic severity with comprehensive evidence from mostly rural parishes. The results show that middle- and high-income parishes had a significantly lower increase in mortality rates than low-income parishes. Moreover, the mortality rate during the 1918 influenza pandemic was significantly higher in highly polluted parishes compared with least polluted parishes. Furthermore, the paper provides a detailed description of mortality statistics in Württemberg and new excess mortality rate estimates for Germany and its states.

KEYWORDS

air pollution, income, mortality, pandemics, Spanish Flu

JEL CLASSIFICATION

I14, I15, N34, Q53

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The 1918 ‘Spanish Flu’ was the deadliest influenza pandemic in modern history, likely causing 50–100 million deaths.¹ Besides the large range in the global mortality estimates, the local mortality rate estimates show enormous variation across and within countries.² To date, the factors influencing the varying regional severity of the pandemic have not been fully explored. This paper studies the local determinants of pandemic mortality in southwest Germany, using exceptionally detailed vital statistics for 1763 parishes in the Kingdom of Württemberg. The analysis focuses on the effect of poverty and air pollution on mortality. Both factors have received much attention also in current debates on regional differences in COVID-19 mortality.³

The paper makes three contributions to the literature on regional differences in pandemic severity during the 1918 influenza pandemic. First, I study the determinants of pandemic severity at an unusually disaggregated level: the median population of parishes in the sample is just 649. The analysis thus complements the existing literature on urban pandemic severity with comprehensive evidence from mostly rural parishes. The focus on rural parishes is essential because previous results indicate considerable differences in pandemic severity by the degree of urbanisation.⁴ Second, I provide the first analysis on the effect of income on regional pandemic severity for Germany and one of the first for continental Europe. This point is important as existing evidence points to significant differences in pandemic severity between Europe and the United States.⁵ Moreover, in contrast to the analysis for the United States,⁶ recent studies focusing on Europe provide mixed evidence on the effect of income on influenza mortality.⁷ Third, I assess the link between air pollution and regional pandemic mortality, motivated by considerable evidence that air pollutants can increase susceptibility to influenza infection.⁸

The previous literature has mainly focused on urban areas, but at the beginning of the twentieth century, two-thirds of the population lived in towns with fewer than 5000 inhabitants.⁹ The analysis of rural areas, however, is impeded by the lack of high-quality data. The administrative statistics for the universe of civil parishes (henceforth parishes) in Württemberg helps to fill this research gap.¹⁰ Furthermore, the Kingdom of Württemberg is of particular interest because of its development level. As a latecomer in the industrial revolution, Württemberg shares several socio-economic characteristics with other rural areas in Germany and Europe, for example, relatively low income levels and urbanisation rates and relatively high infant mortality rates. Therefore, the results can inform the general debate on the effects of income and pollution during the 1918 influenza in rural areas beyond Württemberg. Moreover, in contrast to other central European regions, there were no ground battles of the First World War in Württemberg.

¹ Johnson and Mueller, ‘Updating the accounts’, p. 115.

² For example, Johnson and Mueller estimate country-specific mortality rates between 1.2 and 445.0 per 1000 persons, for Argentina and Cameroon, respectively, and Bootsma and Ferguson estimate 4–12 excess deaths per 1000 persons in a sample of 47 US cities (Johnson and Mueller, ‘Updating the accounts’; Bootsma and Ferguson, ‘Effect of public health measures’).

³ Beach, Clay, and Saavedra, ‘The 1918 influenza pandemic’.

⁴ Clay, Lewis, and Severnini, ‘Cross-city variation’.

⁵ Bootsma and Ferguson, ‘Effect of public health measures’.

⁶ Ibid; Cilek, Chowell, and Fariñas, ‘Age-specific excess mortality’; Grantz et al., ‘Disparities’.

⁷ Karlsson, Nilsson, and Pichler, ‘Impact of the 1918 Spanish flu’; Dahl, Hansen, and Jensen, ‘The 1918 epidemic’; Carillo and Jappelli, ‘Pandemics and local economic growth’.

⁸ Jaspers et al., ‘Diesel exhaust’.

⁹ Bairoch and Goertz, ‘Factors of urbanisation’, p. 288.

¹⁰ The civil parish (or parish) is the smallest administrative unit in Württemberg and comprises towns and villages.



The paper provides a detailed description of mortality statistics in Württemberg. The official statistics are disaggregated by cause of death, age, and sex. Although these data are only available at the state level, they uncover the unique patterns of the 1918 influenza pandemic. For instance, the reported influenza mortality increased by about 3000 per cent in 1918 compared with previous years and showed a distinctive W-shape in the age-specific mortality pattern. To the best of my knowledge, data with a comparable level of detail have not been discussed for any major German state before.¹¹

The paper also relates the excess mortality rate estimates for Württemberg to newly estimated excess mortality rates of Germany and its states. Based on these estimates, the paper shows an association of income and air pollution at the national level. Moreover, the paper briefly describes the socio-economic conditions in Württemberg at the beginning of the twentieth century and the adverse effects of the First World War on food supply and pre-pandemic health. The suffering caused by the war may also explain why the 1918 influenza pandemic did not leave a lasting impression on the collective memory of the German population.

For the empirical analysis, I use annual data on all-cause mortality for the universe of parishes in Württemberg in 1914–25. I combine these data with rich socio-economic data from various population and occupation censuses. For each parish, I observe the amount of total taxable income in 1907 and calculate the average income per capita. In addition, I link the data with available information on the location of coal-fired power plants, a major source of air pollution in the early twentieth century.¹² Before the First World War, about two-thirds of the installed power plant capacity in Württemberg and neighbouring Hohenzollern was based on coal.¹³ The pollution was spatially dispersed from the power plants and affected the pollution levels of parishes in a wider radius. I exploit this fact to calculate the exposure of each parish to pollution from coal-fired power plants as a proxy for air pollution.

The empirical analysis is based on a difference-in-differences approach to estimate the effect of poverty and air pollution on pandemic mortality. The approach compares the average change in mortality rates during the influenza pandemic across parishes with high and medium income (pollution) relative to parishes with low income (pollution). The average all-cause mortality rate across parishes in Württemberg was 15.8 deaths per 1000 persons during the pandemic year 1918, corresponding to a mortality rate increase of 2.9 deaths or 23 per cent relative to the baseline in 1917.¹⁴ In low-income (pollution) parishes, the respective mortality rate increase was 3.9 (2.4).

The results show that middle- and high-income parishes recorded a significantly lower increase in mortality rates than low-income parishes by 1.3 and 0.9 deaths per 1000 population, respectively. Moreover, the mortality rate increase from 1917 to 1918 was significantly higher in highly polluted parishes compared with least polluted parishes. The estimates indicate an additional increase in the mortality rate by 1.6 deaths per 1000 population. In other words, the spike in 1918 mortality was particularly large in poor and highly polluted parishes.

¹¹ Previous studies of the 1918 influenza pandemic in Germany often focus on case studies for smaller areas or the medical debates of the time (Michels, 'Die Spanische Grippe 1918/19', pp. 4–5).

¹² There are no data on the actual air pollution levels in Württemberg available for the time period. However, the level of pollution in the late nineteenth and early twentieth century in industrialised countries is considered to be much higher than today and mainly caused by the usage of coal (Bailey, Hatton, and Inwood, 'Atmospheric pollution'; Beach and Hanlon, 'Coal smoke'; Clay, Lewis, and Severnini, 'Cross-city variation'). Throughout the text, I will use the terms 'pollution' and 'coal-fired power plant capacity' interchangeably.

¹³ Ott et al., 'Elektrizitätsversorgung', p. 7.

¹⁴ Here, the mortality rates exclude military personnel, stillbirths, and infant deaths.



Two recent articles summarise the extensive literature on the 1918 influenza pandemic. Beach et al. focus on literature about economic and health outcomes,¹⁵ and Taubenberger et al. focus on the medical and biological insights on the 1918 influenza pandemic.¹⁶ The review articles demonstrate that, although an extensive body of work has emerged over the last century, numerous questions remain unanswered to date, not least on the origin of the virus and how many deaths it caused. Widely cited estimates on the global mortality burden range between 50 and 100 million deaths.¹⁷ This large range in estimates is partially driven by sparse data for developing countries in Africa and Asia, especially China.¹⁸ However, even for industrialised countries that generally have detailed statistics in the early twentieth century, the estimates vary widely.¹⁹ According to new estimates in this paper, there may have been as many as 4.1 excess deaths per 1000 persons in Württemberg and between 5.4 and 7.0 in Germany.

The empirical analysis focuses on the impact of poverty and air pollution on mortality. Both factors have received much attention also in debates on regional differences in mortality during the COVID-19 pandemic. Several studies have analysed the effect of socio-economic differences on mortality during the 1918 influenza pandemic. However, some of these studies do not study income, but other potentially correlated measures like apartment size, social status based on occupation, and housing conditions, or illiteracy rates, home ownership, and unemployment.²⁰ These papers provide evidence for a socio-economic gradient in mortality rates. However, the measures used in these studies focus on a specific channel of the income effect. This limitation is not trivial because income differences can affect pandemic mortality through multiple channels, and the studies mentioned above potentially neglect parts of the effect.²¹

Focusing on cross-country differences in income, Murray et al. and Barro et al. find a significant negative income effect on pandemic mortality in a sample of 27 and 42 countries, respectively.²² Other studies, however, do not find a significant correlation between income levels and pandemic mortality in 1918.²³ The literature provides strong evidence on the effect of

¹⁵ Beach, Clay, and Saavedra, 'The 1918 influenza pandemic'.

¹⁶ Taubenberger, Kash, and Morens, 'The 1918 influenza pandemic'.

¹⁷ Johnson and Mueller, 'Updating the accounts', p. 115.

¹⁸ The actual range of mortality estimates is even larger; for example, Patterson and Pyle estimate a global mortality burden of 25–40 million deaths (Patterson and Pyle, 'Geography and mortality').

¹⁹ Beach, Clay, and Saavedra, 'The 1918 influenza pandemic', p. 17.

²⁰ Mamelund, 'A socially neutral disease?'; *idem*, '1918 pandemic morbidity'; Bengtsson, Dribe, and Eriksson, 'Social class'; Sydenstricker, 'The incidence of influenza'; Chowell et al., 'The 1918–1919 influenza pandemic'; Clay, Lewis, and Severnini, 'Cross-city variation'; Grantz et al., 'Disparities'.

²¹ Differences in income can affect multiple factors that influence (pandemic) mortality, like the nutritional situation, access to sanitary infrastructure, or access to healthcare (Blum, 'War, food rationing, and socioeconomic inequality'; Gallardo-Albarrán, 'Sanitary infrastructures'; Bauernschuster, Driva, and Hornung, 'Bismarck's health insurance'). For instance, it is not clear to what extent these factors are captured by social status differences of individuals. See Deaton and Weil for a detailed discussion on the association between income differences and health (Deaton, 'Health'; Weil 'Health and economic growth').

²² Murray et al., 'Global pandemic'; Barro, Ursua, and Weng, 'Coronavirus'. Basco et al. use Spanish occupation-level data and associated income to show a negative association between income and pandemic mortality rates (Basco, Domenech, and Rosés, 'Unequal mortality'). Clay et al. control for the manufacturing payroll per worker in 1900, but the results for this estimator are not reported (Clay, Lewis and Severnini, 'Pollution'). Furthermore, Mamelund shows a significant effect of wealth differences on pandemic mortality for Norwegian regions (Mamelund, 'Spanish influenza mortality').

²³ Brainerd and Siegler, 'Economic effects'; Karlsson, Nilsson, and Pichler, 'Impact of the 1918 Spanish flu'; Carillo and Jappelli, 'Pandemics and local economic growth'; Dahl, Hansen, and Jensen, 'The 1918 epidemic'.



socio-economic factors, but the effect of income-level differences on pandemic mortality is less well understood. In this paper, I show that the income level in 1907 has a statistically and epidemiologically significant effect on the relative change in all-cause mortality rates between 1917 and 1918. I provide evidence that parishes with lower income levels in 1907 experienced a significantly stronger increase in mortality rates from 1917 to 1918. The level of analysis is of particular interest because the majority of the population lived in rural areas and there is empirical evidence that the influenza pandemic had different impacts in smaller and more rural parishes.²⁴

The effect of pollution on mortality during the 1918 influenza pandemic has received less attention.²⁵ Only recently, an article by Clay et al. showed the effect of installed coal-fired electricity generating capacity on mortality in a sample of 180 US cities.²⁶ The authors find the mortality rate to increase by an additional 9.6 per cent in high-capacity cities and by 5.4 per cent in medium-capacity cities, relative to changes in low-capacity cities. Furthermore, Clay et al. find a significant effect of pollution on infant mortality.²⁷ In an extended dataset of 438 US cities with at least 10 000 inhabitants, Clay et al. test the effect of multiple factors on excess mortality in 1918, including coal-fired capacity.²⁸ Their results confirm the significant effect of pollution on pandemic mortality, but only for high-capacity cities.

The empirical strategy of the paper follows mainly Clay et al.²⁹ The main difference, however, is the unit of analysis. Clay et al. use a sample of US cities with at least 20 000 inhabitants,³⁰ while the median parish in Württemberg has a population of 649 inhabitants. The paper also contributes to a broader literature that analyses the effect of pollution on mortality, for example, Beach and Hanlon, who analyse the effect of pollution on mortality during the industrialisation in England and Wales.³¹

I | HISTORICAL BACKGROUND

On 19 October 1918, the military physician assistant (*Feldhilfsarzt*) Erich Steinthal from Stuttgart published an article about the new ‘Spanish Disease’. Steinthal closes his article with a warning that, although the press in Württemberg has taken the influenza outbreak lightly, the pandemic’s overall consequences cannot be foreseen.³² The article, published at the peak of the 1918 influenza pandemic in Stuttgart (the capital of the Kingdom of Württemberg), illustrates the perception of the pandemic as a minor problem in local media. The low level of media and public interest in the pandemic was due to the (self-)censorship of the press and the hardships of everyday

²⁴ Acuna-Soto, Viboud, and Chowell, ‘Influenza and pneumonia mortality’; Chowell et al., ‘The 1918–1919 influenza pandemic’.

²⁵ The detrimental health effects of pollution on influenza in general, however, have been studied more extensively, see for example: Jaspers et al., ‘Diesel exhaust’; Wong et al., ‘Modification’; Wu et al., ‘Air pollution’.

²⁶ Clay, Lewis and Severnini, ‘Pollution’.

²⁷ Ibid.

²⁸ Clay, Lewis and Severnini, ‘Cross-city variation’.

²⁹ Clay, Lewis and Severnini, ‘Pollution’, pp. 1190–2.

³⁰ Ibid, p. 1186.

³¹ Beach and Hanlon, ‘Coal smoke’.

³² Steinthal, ‘Beobachtungen an “Spanischer Krankheit”’, p. 368.



survival imposed by the First World War.³³ In the following section, I describe the socio-economic conditions before and during the Great War in Württemberg.

The Kingdom of Württemberg was one of the four kingdoms of the German Empire and the third largest after Prussia and Bavaria (see [figure A1](#) in the Appendix). According to the 1910 census, Württemberg had a population of about 2.4 million. Württemberg was a latecomer in the industrial revolution, and its industrialisation process still lagged behind other German states at the beginning of the twentieth century.³⁴ The share of agricultural employment in Württemberg was 41.3 per cent in 1907, while on average in the German Empire, it was 32.7 per cent.³⁵ In addition, Frank estimates that the GDP per capita in 1913 was about 92 per cent of the German average.³⁶ Thus, Württemberg's development level was comparable to other less developed German regions such as Bavaria (87 per cent) and Hanover (94 per cent) and European states such as Austria (95 per cent) and France (96 per cent).³⁷ Württemberg also shares other common measures of development with these states, for example, relatively high pre-war infant mortality rates and relatively low urbanisation rates.³⁸

Economic historians have identified multiple factors that contributed to the kingdom's economic backwardness, for example, institutions such as the guilds³⁹ and the division of property among all heirs (*Realteilung*),⁴⁰ as well as high pre-railway transport costs.⁴¹ In addition, the Kingdom lacked raw materials, such as coal or ore, which were key drivers in early industrialisation.⁴² In 1913, for example, about 1000 kg of coal was consumed per capita in Württemberg, compared with an average of 3870 kg per capita in the whole German Empire.⁴³ Despite the lack of coal deposits, the public electricity supply in Württemberg was considered very advanced compared with other German states. An official report states that, out of the 1907 parishes and localities, 1705 had a sufficient supply of electricity in March 1915.⁴⁴

When the German Empire declared war on Russia on 1 August 1914, there was hardly any publicly noticeable criticism.⁴⁵ This changed soon, however, with little success in military campaigns and an increasingly protracted war. In the German Empire, about 2 million soldiers lost their lives, and more than 4 million were wounded during the First World War.⁴⁶ In Württemberg, more than 72 000 soldiers died during the war, with over 5000 deaths due to diseases.⁴⁷ Thus, the military

³³ Witte, 'The plague'.

³⁴ Marquardt, *Geschichte Württembergs*; Flik, 'Von der Agrar- zur Dienstleistungsgesellschaft'.

³⁵ Losch, 'Berufs- und Betriebszählung vom 12. Juni 1907'.

³⁶ Frank, 'Regionale Entwicklungsdisparitäten'.

³⁷ Ibid.; Bolt and van Zanden, 'Maddison Project Database'.

³⁸ Table A16 in the online Appendix shows the corresponding values for Austria, France, and more developed European countries for comparison.

³⁹ Acemoglu et al., 'Consequences of radical reform'; Ogilvie, 'Guilds'; *idem*, *European guilds*.

⁴⁰ Flik, 'Von der Agrar-zur Dienstleistungsgesellschaft'.

⁴¹ Braun and Franke, 'Railways'.

⁴² Fernihough and O'Rourke, 'Coal'.

⁴³ Statistisches Landesamt, *Statistisches Handbuch 1914/21*; Statistisches Reichsamt, *Statistisches Jahrbuch 1924/25*.

⁴⁴ Ott, 'Grundlageninvestitionen in Württemberg', pp. 143–4. See online Appendix A2 for a more detailed discussion of coal consumption and electricity production in Württemberg.

⁴⁵ Herwig, *The First World War*.

⁴⁶ Statistisches Reichsamt, *Statistisches Jahrbuch 1924/1925*.

⁴⁷ Statistisches Landesamt, *Württembergische Jahrbücher 1919/20*.



losses account for about 3 per cent of Württemberg's pre-war population and 14 per cent of males of military age (17–45 years old).

The suffering due to the First World War, however, was not limited to the soldiers and their families but reached the whole population. Cox uses data of school-age children in Germany during the First World War,⁴⁸ and Blum uses anthropomorphic data from German Second World War soldiers to show severe malnutrition in Germany during the First World War.⁴⁹ There are multiple reasons for the crisis of food supply, including the Allied blockade and crop failures.⁵⁰

The increasingly hopeless military situation and food shortages facilitated the Kiel mutiny on 3 November 1918. It was the starting point of the German Revolution that ended the German monarchy within a few days and intensified the calls for peace. On 11 November, the Armistice of Compiègne ended the First World War's battles. However, the conditions during the revolutionary period did not improve immediately, partly because the blockade ended only in July 1919.

II | THE MORTALITY BURDEN OF THE INFLUENZA PANDEMIC

In this situation of war and food shortages, the 1918 influenza pandemic reached Württemberg. Figure 1 illustrates the monthly number of all-cause deaths for the Kingdom of Württemberg and the capital Stuttgart.⁵¹ The virus that caused the influenza pandemic might have already spread before 1918, but the excess mortality only exceeded detection thresholds worldwide in three waves in 1918 and 1919:⁵² the first wave in northern spring and summer 1918, the second wave in autumn 1918, and the third wave in spring 1919. The first influenza wave in Württemberg peaked in July 1918.⁵³ The overall number of monthly deaths, excluding military personnel and stillbirths, increased from 2688 in June 1918 to 3133 in July 1918. The average number of monthly deceased in July 1914–7 was 2798. Thus, Württemberg saw an increase in monthly deaths of 17 per cent in July (relative to June 1918) or 12 per cent (relative to July 1914–7). The spike in July 1918 was even more pronounced in Stuttgart, where the number of monthly deaths increased by 28 per cent or 37 per cent, respectively. The big difference in both measures for Stuttgart indicates that the influenza pandemic hit the city already in June 1918.

The second, deadlier wave of the influenza pandemic peaked in November 1918 (marked by the vertical dashed line in figure 1). The total number of deaths increased to 8969, an increase of 208 per cent relative to the average in November 1914–7. The mortality in Stuttgart, however, already reached its peak in October 1918. The earlier peak in the capital provides evidence for a spread of the virus from more central urban hubs to the rural hinterlands.⁵⁴ Figure 1 reveals a third wave that spread in Württemberg in April and May 1919, with peak mortality in May. The magnitude of the third wave is comparable to the first wave. The time series for Stuttgart indicates another

⁴⁸ Cox, 'Hunger games'.

⁴⁹ Blum, 'Government decisions'; *idem*, 'War, food rationing, and socioeconomic inequality'.

⁵⁰ Howard, 'Social and political consequences'. See online Appendix A3 for a more detailed discussion.

⁵¹ Statistisches Landesamt, *Statistisches Handbuch 1914/21*; *idem*, *Statistisches Handbuch 1922/26*.

⁵² Taubenberger, Kash, and Morens, 'The 1918 influenza pandemic'; Johnson, 'Aspects of the historical geography'. However, this pattern was not universal; for example, some areas might have experienced a fourth wave in 1920 (Johnson and Mueller, 'Updating the accounts', p. 107).

⁵³ Bogusat notes that the first influenza infections in Württemberg were recorded already in March 1918. These are the earliest records of the 1918 influenza in Germany (Bogusat, 'Influenza-Epidemie', p. 445).

⁵⁴ Clay, Lewis, and Severnini, 'Cross-city variation'.

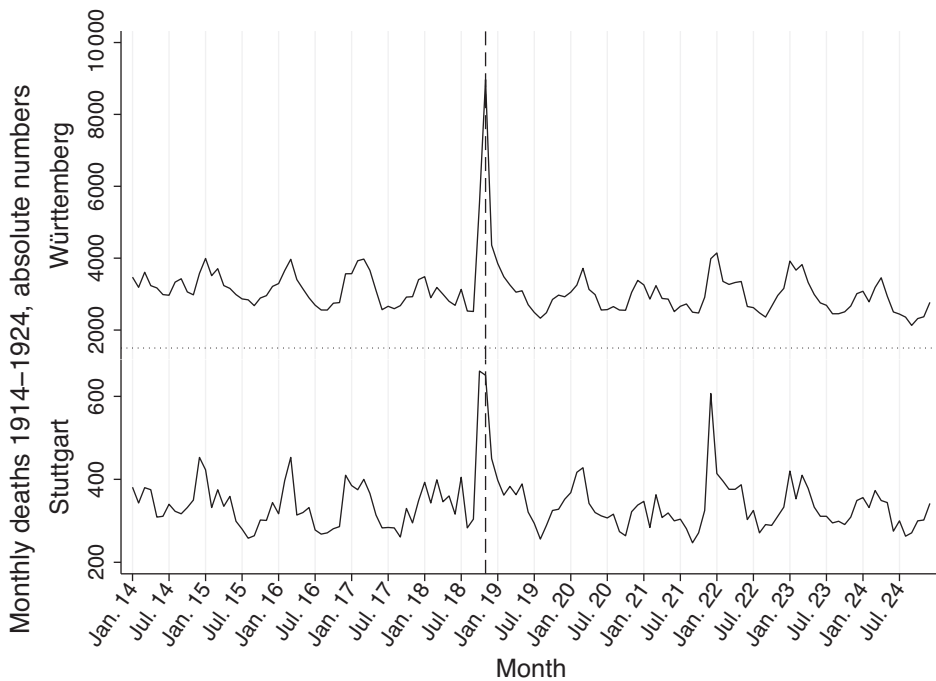


FIGURE 1 Absolute number of monthly deceased 1914–24. The number of deceased does not include military personnel or stillbirths. *Source:* Statistisches Landesamt, *Württembergische Jahrbücher* 1919/20; Statistisches Landesamt, *Württembergische Jahrbücher* 1925/26. Author's design

severe mortality increase in December 1921. This peak in mortality coincides with an influenza wave also documented for other towns.⁵⁵ However, the 1921/2 influenza had a more common ‘U-shaped’ age-specific mortality pattern and is generally not associated with the Spanish Flu.

The monthly data allow us to identify the onset of the pandemic and to distinguish the severity of the different waves in line with previous studies. Unfortunately, these data do not allow a further breakdown by cause of death, age, or sex. Therefore, I revert to annual state-level data published by the statistical office in Württemberg.⁵⁶ Figure 2 shows several annual mortality statistics by sex and age groups for Württemberg, excluding military personnel and stillbirths. Figure 2a shows the total number of all-age influenza deaths per year and distinguishes between male and female deaths. In 1914–7, the statistics report on average 103 male and 134 female deaths per year due to influenza. These numbers increase sharply in 1918 to 2941 male and 4322 female deaths. Thus, the statistics indicate 7026 excess influenza deaths in 1918, relative to the average of 1914–7. In 1919, when the third wave hit Württemberg, the official statistics record 525 male and 692 female influenza deaths.

⁵⁵ Lubinski, ‘Grippepandemie in Breslau’.

⁵⁶ Statistisches Landesamt, *Württembergische Jahrbücher* 1919/20; *idem*, *Württembergische Jahrbücher* 1925/26.

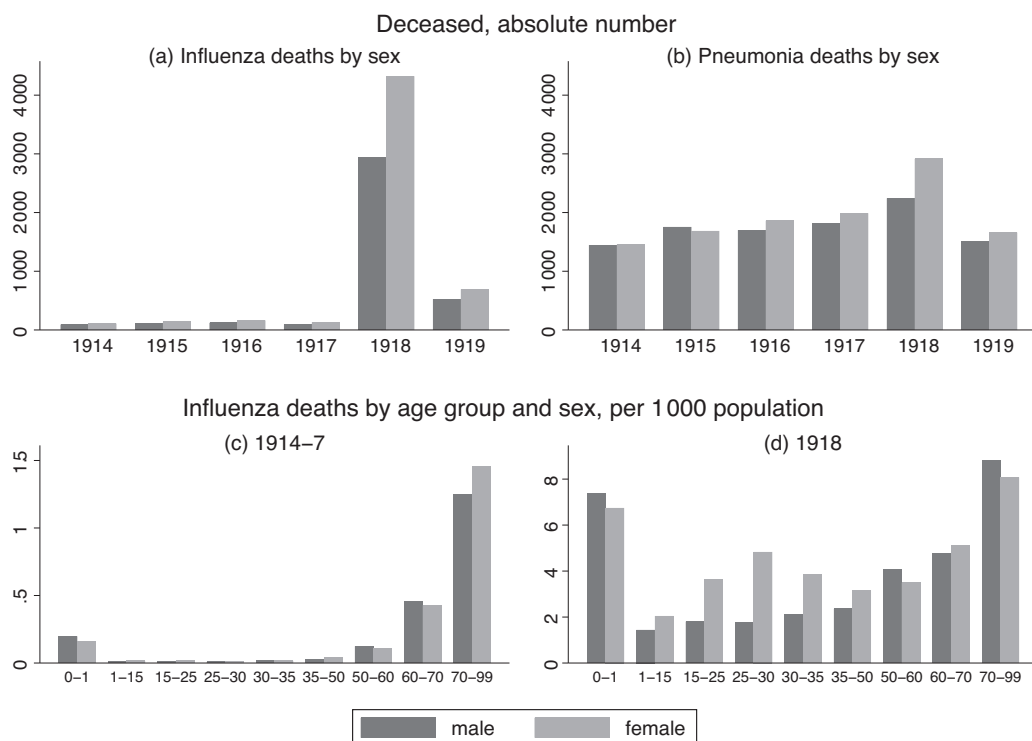


FIGURE 2 Influenza and pneumonia statistics for Württemberg. (a, b) Annual influenza and pneumonia deaths by sex for the years 1914–9. (c) The annual influenza mortality rate by age group averaged over the years 1914–7. (d) The influenza mortality rate by age group in 1918. The number of deceased does not include military personnel and stillbirths. Source: Statistisches Landesamt, *Württembergische Jahrbücher* 1919/20. Author's design

The influenza mortality statistics might suffer from under-reporting, especially due to cases of influenza that were wrongly assigned to pneumonia.⁵⁷ To evaluate the magnitude of this misreporting, figure 2b shows the annual number of pneumonia deaths by sex for 1914–9. In 1918, there are 2242 male and 2914 female deaths assigned to pneumonia, while the average for 1914–7 is 1679 and 1747, respectively. This provides suggestive evidence that a considerable amount of influenza deaths have been assigned to pneumonia.⁵⁸

The reported differences in male and female influenza and pneumonia deaths should not be mistaken as differences in influenza mortality rates by sex. They are mainly driven by the exclusion of military personnel. When influenza and pneumonia deaths of military personnel are included, the differences between the sexes decrease significantly (see figure A7a and b in the online Appendix).

Figure 2 (c and d) reports the influenza mortality rate per 1000 population by nine age groups and sex. Figure 2c shows the average mortality rates for the years 1914–7, while figure 2d shows

⁵⁷ Additionally, pneumonia was often caused by an initial influenza infection. Thus, although pneumonia might have been correctly diagnosed, some cases were caused by influenza.

⁵⁸ Figure A7 (e and f) in the Appendix shows pneumonia mortality rates per 1000 population by age group and sex, for the years 1914–7 and 1918, respectively. The increase in mortality rates specifically among young adults in 1918, in line with the W-shaped age-specific mortality pattern of the Spanish Flu, provides further evidence for the false assignment of cases.



the influenza mortality rates for 1918. The age distribution of the census in 1910 is used to calculate the age-group-specific influenza mortality rates for each year.

The average influenza mortality rate per 1000 population in 1914–7 is 0.2 for infants, almost zero for people aged 1–50 years, and increases significantly for people above age 50. The highest mortality rate is 1.4 and observed for people above age 70. The average influenza mortality rate per 1000 population in 1918 is about 3.0 and thus much higher than in the previous years.⁵⁹ The highest influenza mortality rates in figure 2d are observed for infants (7.4 for males and 6.7 for females) and people above age 70 (8.8 for males and 8.1 for females). The true infant mortality rate in 1918 is even higher because figure 2d neglects the decline in birth rates during the war (see online Appendix A7 for further details on fertility and infant mortality). Dividing the total number of influenza deaths of infants younger than 1 year by the number of births in 1917 increases the influenza mortality rate to 11.4. The age-specific distribution of influenza mortality in 1918 is commonly described as W-shaped, that is, high mortality rates among the youngest and oldest population groups, but also relatively high mortality rates among young adults, peaking at about age 27.⁶⁰ Figure 2d matches the W-shaped mortality pattern, but only for females. Again, this is due to the exclusion of influenza deaths of military personnel.⁶¹

In general, the cause-specific mortality statistics describe a pattern of the Spanish Flu in Württemberg that is in line with the findings for other countries and regions. Yet, the data also demonstrate the difficulties in accounting for influenza mortality of military personnel and insufficient diagnostics at the time. Therefore, most scholars use all-cause mortality to calculate excess mortality during the influenza pandemic. However, the estimates for the excess mortality in Germany vary considerably. Johnson and Mueller estimate the death toll of the influenza pandemic in Germany to be about 225 000 and the excess mortality per 1000 population to be 3.8.⁶² Patterson and Pyle estimate a range of 4.2–5.0.⁶³ Ansart et al. use monthly all-cause mortality statistics and estimate the cumulative excess mortality rate at 7.3.⁶⁴ Murray et al. and Barro et al. compare the annual all-cause and influenza-related mortality in 1918–20 with the average in the 3-year periods before and after 1918–20.⁶⁵ The resulting cumulative excess mortality rates are 7.6 and 7.8, respectively. Thus, the estimates for Germany range from 3.8 to 7.8, that is, they vary by a factor of about two. The variation in the estimates can be explained by different datasets, estimation methods, and definitions.

⁵⁹ This is a lower bound of the true influenza mortality rate in 1918 because it neglects the influenza deaths among military personnel and does not account for wrongly assigned pneumonia deaths. On the other hand, it uses the population of the 1910 census as the denominator, which causes an upward bias in the mortality rate. When I use the average total population of 1918 as the denominator and include influenza deaths among military personnel and excess pneumonia deaths in 1918, the adjusted influenza mortality rate per 1000 population increases to 4.2 (*Statistisches Landesamt, Württembergische Jahrbücher 1919/20; idem, Statistisches Handbuch 1922/26*).

⁶⁰ Taubenberger, Kash, and Morens, 'The 1918 influenza pandemic', p. 10. Figure A8 in the online Appendix documents a similar age-specific mortality pattern for Germany and its states. The W-shaped curve of age-specific influenza mortality, however, is not always observed (Cilek, Chowell, and Fariñas, 'Age-specific excess mortality').

⁶¹ When I use the age distribution of the civilian population in 1916, that is, exclude military personnel from the denominator, the gap between male and female mortality rates disappears and the W-shaped mortality pattern can also be observed for males (see online Appendix figure A7, c and d).

⁶² Johnson and Mueller, 'Updating the accounts', p. 113.

⁶³ Patterson and Pyle, 'Geography and mortality', p. 14.

⁶⁴ Ansart et al., 'Mortality burden', pp. 100–1.

⁶⁵ Murray et al., 'Global pandemic', p. 2212; Barro, Ursua, and Weng, 'Coronavirus', pp. 3–4.

**TABLE 1** Influenza-related excess mortality 1918–20 in Württemberg by age group and sex

Age group	0–1	1–15	15–25	25–30	30–35	35–50	50–60	60–70	70–99	Mean
Female	7.0	2.2	5.6	7.4	7.0	5.5	5.9	5.6	7.4	4.9
Male	5.4	1.3	3.3	3.1	3.0	3.2	4.9	4.6	3.6	2.9
Total	6.2	1.8	4.5	5.3	5.0	4.4	5.4	5.2	5.7	3.9

Note: The table shows excess mortality rates per 1000 population 1910 by age group and sex. Excess mortality rate based on the definition by Barro et al. using all influenza-related deaths of the civilian population (Barro, Ursua, and Weng, 'Coronavirus').

Source: Statistisches Landesamt, *Württembergische Jahrbücher* 1919/20; Statistisches Landesamt, *Württembergische Jahrbücher* 1925/26. Author's calculations.

Based on the estimation method in Ansart et al., the excess mortality rate per 1000 population in Württemberg is 3.8.⁶⁶ The following estimates are based on the methods by Murray et al. and Barro et al.⁶⁷ The excess mortality rate estimates for Württemberg are 2.3 and 3.9, respectively. Table 1 presents excess mortality rates by age group and sex for Württemberg, using all influenza-related deaths. The mortality rates for females and the total mortality rates show that young adults aged 25–30 years were among the prime victims of the 1918 influenza. However, this pattern does not hold for males because the data exclude military personnel.

In addition, I re-estimate excess mortality rates for Germany and its states using mortality and population statistics published in *Statistisches Jahrbuch für das Deutsche Reich*.⁶⁸ Using these alternative sources, the estimates for Württemberg are slightly higher, with 2.5 all-cause and 4.0 influenza-related excess deaths per 1000 population. For Germany, the respective estimates are 5.4 and 5.9 excess deaths per 1000 population, with a range of 2.5–8.0 and 4.0–7.3 across the German states (see table A3 in the online Appendix). Thus, the estimates for Württemberg are at the lower end of the estimates for Germany. The estimates presented here may underestimate the true pandemic excess mortality because they exclude military personnel, that is, highly susceptible young adult males. Including influenza-related mortality of military personnel increases the German estimate from 5.9 to 6.3. The respective excess mortality rate estimates for females are 7.0 and 6.8, and provide an upper bound (see online Appendix A4 for further details).

The variation in regional excess mortality rates raises the question of underlying factors. In the contemporary public perception, there was a link between influenza mortality, food shortages, and the poor health situation caused by the war.⁶⁹ On the other hand, the relatively high mortality rate observed among healthy young adults seemingly contradicts this explanation.⁷⁰ Bootsma and Ferguson show the effectiveness of non-pharmaceutical interventions (NPIs) on pandemic mortality in US cities.⁷¹ These measures included the closure of schools and churches or mandated mask-wearing. However, NPIs cannot explain regional differences in Germany because stringent measures were rarely introduced or were short-lived.⁷²

⁶⁶ Ansart et al., 'Mortality burden'.

⁶⁷ Murray et al., 'Global pandemic'; Barro, Ursua, and Weng, 'Coronavirus'. Unfortunately, I could not determine the exact definitions used by Johnson and Mueller and Patterson and Pyle (Johnson and Mueller, 'Updating the accounts'; Patterson and Pyle, 'Geography and mortality'). See online Appendix A4 for further details.

⁶⁸ Statistisches Reichsamt, *Statistisches Jahrbuch* 1919–25.

⁶⁹ Michels, 'Die Spanische Grippe 1918/19', p. 14.

⁷⁰ Bogusat, 'Influenza-Epidemie'; Taubenberger, Kash, and Morens, 'The 1918 influenza pandemic'.

⁷¹ Bootsma and Ferguson, 'Effect of public health measures'.

⁷² Witte, 'The plague'; Michels, 'Die Spanische Grippe 1918/19', p. 20.

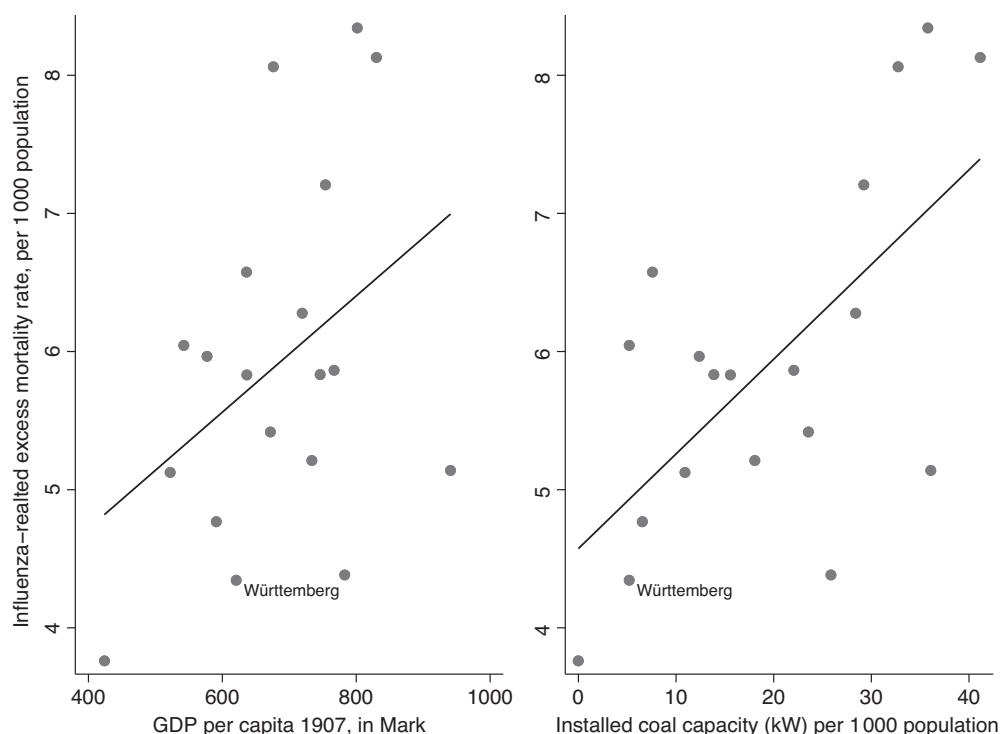


FIGURE 3 Association of income and pollution with influenza-related excess mortality 1918–20 for German states and Prussian provinces. The graph shows the association of income (left) and pollution (right) with influenza-related excess mortality 1918–20 for German states and Prussian provinces. The influenza-related mortality rate per 1000 population includes all deaths from influenza, pneumonia, other diseases of the respiratory organs, tuberculosis, and whooping cough. The excess mortality rate is the sum of deviations in 1918–20 from the average in 1921–3. The income level is measured by GDP per capita in 1907. Pollution is calculated as the installed coal-fired capacity 1913 in kW per 1000 population. The lines indicate fitted values based on univariate OLS regressions. *Source:* Herzig, Feherbach, and Drummer, ‘Elektrizitätsversorgung Deutschlands’; Frank, ‘Regionale Entwicklungsdisparitäten’; Statistisches Reichsamt, *Statistisches Jahrbuch* (1919–25). Author’s design

Previous studies have discussed the effect of income and air pollution on regional differences in pandemic severity. Figure 3 shows that both factors correlate with excess mortality rates of German states and Prussian provinces.⁷³ Univariate linear regressions indicate a positive and statistically significant association between influenza-related excess mortality rates and GDP per capita in 1907 (0.004, s.e. 0.002), and installed coal-fired power plant capacity in 1913 (0.069, s.e. 0.020). However, in a regression with both factors, the association between income and excess mortality becomes negative (−0.005, s.e. 0.003), while the pollution effect remains positive and statistically significant (0.111, s.e. 0.035). These results necessitate a further analysis of the two factors.

⁷³ Data for the states Mecklenburg-Schwerin and Mecklenburg-Strelitz are missing. Furthermore, Berlin is excluded from the analysis because it was the only metropolis of Germany and had significantly different characteristics. However, the results hold qualitatively if Berlin is included (see [figure A9](#) in the online Appendix).

III | DATA

The remainder of the paper focuses on the effect of income and air pollution on pandemic mortality rates during the 1918 influenza pandemic. For the estimations, I use digitised annual data on all-cause mortality for the universe of parishes in Württemberg in 1914–25.⁷⁴ In each parish, an official coroner (*Leichenbeschauer*) issued the death certificates. The statistical office gathered and checked the mortality data before publication. There is no evidence that this process changed during the war or that major complications arose. The official mortality statistics used in the analysis can therefore be considered of high quality.

I aggregate parishes to take border changes during the sample period into account.⁷⁵ Unfortunately, the mortality statistics on the parish level are missing for the county *Hall* and two parishes. Thus, there are 1763 civil parishes (*Gemeinden*) in the resulting dataset. The median parish had a land area of 8.7 km² and 649 inhabitants in 1910.

I combine these data with rich socio-economic data from various population and occupation censuses, digitised by Braun and Franke.⁷⁶ In particular, the population data are based on censuses in 1910, 1919, and 1925.⁷⁷ To get annual population figures for each parish, I linearly interpolate the population between census years.⁷⁸ On the basis of the annual mortality statistics and interpolated population data, I calculate the annual all-cause mortality rate per 1000 population. To prevent biased estimates due to the large changes in fertility during the First World War, I subtract the number of infants deceased at age 1 and below from the total number of deaths. Furthermore, I exclude mortality of military personnel. At the parish level, the data do not differentiate combat-related deaths from other causes of deaths. The inclusion of combat-related deaths would induce measurement error in the dependent variable and thus increase estimated standard errors. On the other hand, I neglect influenza-related deaths of military personnel. However, the resulting estimation bias is likely to be negligible, as only a small fraction of deaths among military personnel were caused by the pandemic.⁷⁹ Thus, the main dependent variable is defined as the number of deaths above age 1 per 1000 population, excluding deaths among military personnel and stillbirths.⁸⁰ Henceforth, I will refer to this variable as mortality rate (MR).

The two explanatory variables of interest are the parish income and air pollution levels. Income per capita is measured as the total taxable income of natural persons in 1907, that is, income net

⁷⁴ Staatsarchiv Ludwigsburg, Germany, E 258 VII Bü 120.

⁷⁵ Figure A9 in the online Appendix shows parish borders. I digitised parish borders from Kommission für geschichtliche Landeskunde in Baden-Württemberg and Landesvermessungsamt Baden-Württemberg and used information on border changes from Statistisches Landesamt Baden-Württemberg; Kommission für geschichtliche Landeskunde in Baden-Württemberg and Landesvermessungsamt Baden-Württemberg, *Historischer Atlas*; Statistisches Landesamt Baden-Württemberg, *Volkzählungen*.

⁷⁶ Braun and Franke, 'Railways'.

⁷⁷ Statistisches Landesamt Baden-Württemberg, *Volkzählungen*.

⁷⁸ Alternatively, I project the population for each parish based on the annual number of births and deaths and only interpolate the residual that is due to migration. The estimation results using this alternative population measure are virtually identical. The results are not reported for the sake of brevity but can be received on request.

⁷⁹ Only 1% of the 1914–9 deaths of military personnel were attributed to influenza (Statistisches Landesamt, *Württembergische Jahrbücher* 1919/20).

⁸⁰ The main results are, however, robust to the inclusion of infant mortality and controlling for deceased military personnel (see online Appendix section A5).



of tax allowances and other deductions, divided by total population.⁸¹ To measure pollution, I link the data with available information on the location of coal-fired power plants, a major source of air pollution in the early twentieth century.⁸² Therefore, and under the assumption that the use of coal in electric power plants is correlated with the total use of coal – both driven by the energy demand and the relative price of coal-generated energy – the installed coal-fired power plant capacity is used to proxy air pollution. The location of power plants in 1914 is taken from a map by Ott et al.⁸³ The map includes all 594 power plants in Württemberg and neighbouring Baden and Hohenzollern. I geo-referenced the map using geographic information software to get the location of each power plant. In addition, the map provides information on the type of power generation and the installed maximum capacity. For each parish, I calculate the installed capacity of power plants using steam (*Dampfkraft*) or a mixture of steam and other means of power generation (*gemischter Antrieb*) in megawatts (MW) within 50 km.⁸⁴

For the later estimation, I generate dummies for the terciles of income 1907. Hence, these dummies indicate parishes in the sample with low, medium, and high average income. Independently, I generate dummies for the terciles of installed coal-fired power plant capacity within 50 km. The average income per capita is 320.7 *Mark* (see table 2). However, low-income parishes have an average income of 226.2 *Mark* per capita. The average income increases by 86.4 and 197.2 *Mark* per capita for medium- and high-income parishes. The average installed coal-fired power plant capacity within 50 km is 8.2, 30.0, and 65.0 MW for the low, medium, and high tercile, respectively. The average over all parishes is 34.3 MW.

Column (1) of table 2 summarises the mean and standard deviation of the main variables in the dataset. The average mortality rate in 1914–25 is 11.8 deaths per 1000 population, with a standard deviation of 5.8. Columns (2) and (3) show the mean difference in mortality rates between low-income parishes and medium- and high-income parishes, respectively. In parishes with a medium income, there are on average 0.6 fewer deaths per 1000 population compared with parishes in the low-income tercile. The difference is statistically significant at the 1 per cent level (s.e. 0.098). Also, parishes in the high-income tercile have a significantly lower mortality rate. However, the mortality rates of the three groups evolve largely in parallel over time, as shown in figure 4.

Columns (4) and (5) of table 2 show the average difference in mortality rates between parishes with low and medium and low and higher coal-fired capacity, respectively. There is no statistically

⁸¹ Königliches Statistisches Landesamt, *Württembergische Gemeindestatistik*. Parish-level income data are not available for later years. However, per capita income in 1907 and 1917 are highly correlated at the county level (correlation coefficient of 0.905, rank correlation coefficient of 0.790).

⁸² Clay, Lewis, and Severnini, 'Pollution', p. 1180. Data on actual air pollution are not available for Württemberg in the early twentieth century. However, the usage of coal was the main driver of pollution during this period (Bailey, Hatton, and Inwood, 'Atmospheric pollution', Beach and Hanlon, 'Coal smoke').

⁸³ Ott et al., 'Elektrizitätsversorgung'.

⁸⁴ The source refers to steam-powered power plants (*Dampfkraft*); it is made clear that this is a synonym for coal-fired power plants (Ott et al., 'Elektrizitätsversorgung'). Excluding power plants that use a mixture of steam and other means of power generation causes an attenuation bias (see online Appendix A5). Clay et al. use a similar radius of 30 miles (approximately 48.3 km) (Clay, Lewis, and Severnini, 'Pollution', *eisdem*, 'Cross-city variation'). The radius is chosen because power plant emissions disperse locally and Levy et al. show that about 40% of primary fine particulate matter (PM_{2.5}) exposure is located within 50 km of modern coal-fired power plants (Levy et al., 'Using CALPUFF'). The dispersion radius, however, depends on the height of the smokestack, which was likely lower in early-twentieth-century Württemberg. Thus, an even higher share of total exposure would have occurred within 50 km. See also the discussion of dispersion models in online Appendix A6.

**TABLE 2** Descriptive statistics and mean differences

	All	Income		Coal	
	(1)	Medium	High	Medium	High
	(1)	(2)	(3)	(4)	(5)
Outcome					
MR per 1000 pop. 1914–25	11.766 (5.774)	−0.565 [0.098]	−0.669 [0.100]	−0.227 [0.101]	−0.054 [0.097]
Excess MR per 1000 pop. 1918	4.094 (6.455)	−1.700 [0.384]	−1.805 [0.387]	0.218 [0.386]	1.311 [0.363]
Explanatory variables					
Tax income per capita 1907, <i>Mark</i>	320.642 (97.029)	86.403 [1.844]	197.157 [3.755]	−34.376 [5.281]	−49.465 [5.527]
Coal capacity within 50 km (MW)	34.315 (25.169)	−8.146 [1.436]	−15.516 [1.401]	21.784 [0.642]	56.726 [0.249]
Controls					
Pop. 1910, log	6.556 (0.853)	0.179 [0.038]	0.344 [0.053]	−0.126 [0.047]	0.299 [0.050]
Pop. density 1910, log	4.412 (0.663)	−0.090 [0.032]	−0.055 [0.041]	0.078 [0.034]	0.645 [0.035]
Industry empl. share 1907 (%)	11.071 (7.529)	2.432 [0.366]	3.773 [0.441]	0.643 [0.404]	3.942 [0.433]
Establishment size 1907, log	2.053 (1.475)	0.243 [0.058]	0.840 [0.092]	−0.084 [0.073]	0.210 [0.090]
Hydro capacity within 50 km (MW)	22.441 (15.044)	−6.554 [0.910]	−11.202 [0.830]	13.568 [0.735]	15.516 [0.637]
Birth non-local share 1900 (%)	26.246 (11.575)	2.854 [0.561]	12.550 [0.600]	−6.684 [0.662]	−8.876 [0.655]
Pop. age 25–30 share 1910 (%)	7.004 (0.620)	0.105 [0.034]	0.219 [0.036]	0.057 [0.033]	0.250 [0.036]
Female pop. share 1910 (%)	51.465 (2.732)	−0.138 (0.152)	−0.891 (0.168)	1.322 (0.167)	0.816 (0.155)
Dist. to nearest hospital (km)	6.214 (3.363)	−0.221 [0.189]	−1.282 [0.191]	0.923 [0.203]	−0.178 [0.185]
Dist. to nearest medical doctor (km)	4.086 (2.525)	−0.181 [0.137]	−0.570 [0.149]	−0.037 [0.157]	−1.309 [0.142]
Railway station 1910, dummy	0.279 (0.448)	0.104 [0.024]	0.247 [0.025]	−0.025 [0.025]	0.066 [0.027]
Road access 1848, dummy	0.483 (0.500)	0.083 [0.029]	0.197 [0.029]	−0.096 [0.029]	−0.026 [0.029]
River access, dummy	0.078 (0.268)	0.071 [0.013]	0.095 [0.015]	0.047 [0.015]	0.028 [0.015]
Dist. to military base 1918 (km)	5.607 (3.412)	−0.078 [0.182]	−0.567 [0.201]	−0.631 [0.210]	−2.524 [0.186]
Number of parishes	1763	588	587	592	583

Note: The table presents average values and associated standard deviations in parenthesis below for all 1763 parishes in the dataset (column (1)). Columns (2)–(5) show mean differences between parishes with lowest income per capita in 1907 (columns (2) and (3)) and lowest installed coal-fired power plant capacity within 50 km (columns (4) and (5)), relative to medium (columns (2) and (4)), and high levels (columns (3) and (5)). The standard errors of a two-sided mean difference *t*-test are in brackets below.

Sources: See descriptions in Section III.

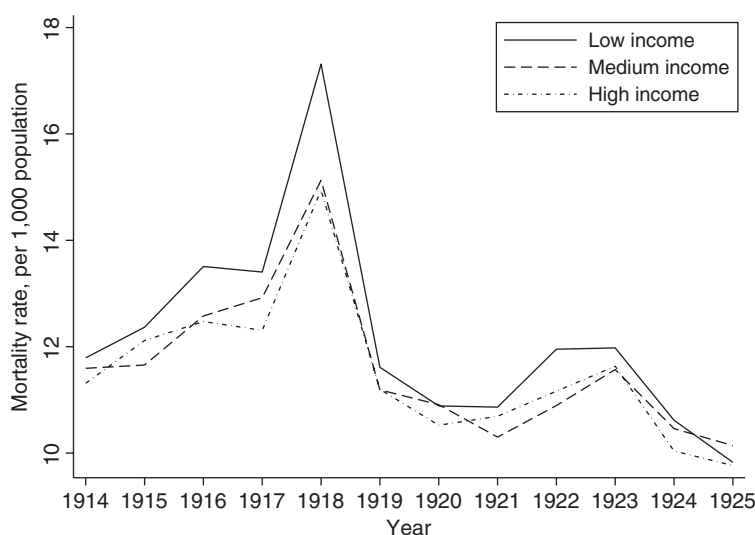


FIGURE 4 Mortality rate 1914–25 by income tercile. The figure shows the annual average mortality rate per 1000 population in parishes for the years 1914–25 by terciles of income 1907. *Source:* Königliches Statistisches Landesamt, *Württembergische Gemeindestatistik*; Statistisches Landesamt Baden-Württemberg, *Volkszählungen*; Staatsarchiv Ludwigsburg E 258 VII Bü 120. Author's design

significant difference in average mortality rates by coal-fired capacity. Moreover, figure 5 shows that the average mortality rate of all three groups moves in parallel over the period 1914–25, but not in 1918.

Table 2 also shows the excess mortality rate in 1918. The excess mortality rate for parish i is the difference between the observed mortality rate in 1918 and the predicted mortality rate. The prediction for 1918 is based on a model with parish-fixed effects and parish-specific linear trends, estimated for the sample periods 1914–7 and 1919–25.⁸⁵ Figure A10 in the online Appendix illustrates the variation of excess mortality rates across parishes. The figure also shows that there is no clear spatial pattern of excess mortality in Württemberg. The average excess mortality rate in 1918 is 4.1 deaths per 1000 population. The magnitude of this estimate is thus comparable to the estimates presented in Section II. The differences in columns (2) to (5) provide first unconditional evidence of the effect of income and pollution on excess mortality during the 1918 influenza pandemic.

The control variables are log population, log population density in 1910, and the ratio of industrial employment over 100 population in 1905, based on the occupation census 1907.⁸⁶ The occupation census comprises parish-level information on the number of full-time gainfully employed persons (self-employed and dependent) in agriculture, industry, and trade and transport. I also use occupation census data to calculate the establishment size in industry as the average number of persons employed in an establishment (*Hauptbetrieb*) in 1907,⁸⁷ as well as the installed capacity of hydroelectric power plants within 50 km⁸⁸ and the share of inhabitants in 1900 that was born in

⁸⁵ Clay, Lewis, and Severnini, 'Cross-city variation'.

⁸⁶ Königliches Statistisches Landesamt, *Württembergische Gemeindestatistik*.

⁸⁷ Ibid.

⁸⁸ Ott et al., 'Elektrizitätsversorgung'.

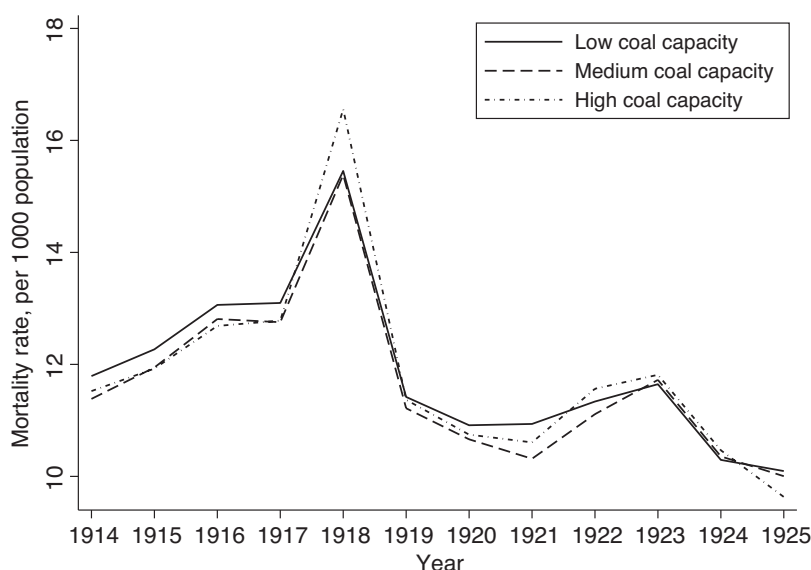


FIGURE 5 Mortality rate 1914–25 by coal capacity tercile. The figure shows the annual average mortality rate per 1000 population in parishes for the years 1914–25 by terciles of installed coal-fired capacity within 50 km. *Source:* Ott, Herzig, Allgeier, and Fehrenbach, ‘Elektrizitätsversorgung’; Statistisches Landesamt Baden-Württemberg, *Volkszählungen*; Staatsarchiv Ludwigsburg E 258 VII Bü 120. Author’s design

another parish.⁸⁹ As additional demographic control variables, I include the share of female population⁹⁰ and the share of the 1910 population aged 25–30 at the county level,⁹¹ as well as binary variables that indicate a railway station in the parish in 1910,⁹² access to a river navigable in 1845, and connection to a paved road in 1848.⁹³ I digitised the location of each military base in Württemberg in 1918 and calculated the distance to the nearest military base in kilometres.⁹⁴ Finally, I digitised the location of hospitals in 1918 and medical doctors in 1911 and calculated the shortest distance for each parish.⁹⁵

Table 2 presents descriptive statistics for the control variables. Parishes with higher income have a higher population size, are more industrialised, with on average larger establishments, and have better access to transport and health infrastructure. At the same time, parishes with high coal-fired capacity are larger, more densely populated, and more industrialised. Moreover, they have more hydroelectric capacity installed within 50 km and are further from the nearest military base. Overall, the control variables capture significant differences between parishes by income and pollution level.

⁸⁹ Statistisches Landesamt Baden-Württemberg, *Volkszählungen*.

⁹⁰ Ibid.

⁹¹ Königliches Statistisches Landesamt, *Württembergische Jahrbücher* 1912.

⁹² Königliches Statistisches Landesamt, *Hof- und Staats-Handbuch* 1911.

⁹³ Kunz and Zipf, *HGIS*. After 1848, the road network was considerably expanded, so that controlling for road access at a later stage would no longer be informative about the centrality of a parish.

⁹⁴ von Moser, *Die Württemberger im Weltkrieg*.

⁹⁵ Ott, ‘Grundlageninvestitionen’; Königliches Statistisches Landesamt, *Hof- und Staats-Handbuch* 1911.



IV | ESTIMATION STRATEGY

The empirical analysis is based on a difference-in-differences approach that compares the average change in mortality rates during the influenza pandemic across parishes with high and medium income (coal-fired capacity) relative to parishes with low income (coal-fired capacity).⁹⁶ The empirical model is specified by the following equation:

$$MR_{it} = \sum_{t=1914}^{1925} \beta_{1t} MI_i \cdot d_t + \sum_{t=1914}^{1925} \beta_{2t} HI_i \cdot d_t + \sum_{t=1914}^{1925} \beta_{3t} MP_i \cdot d_t + \sum_{t=1914}^{1925} \beta_{4t} HP_i \cdot d_t + \sum_{t=1914}^{1925} \gamma_t \overline{MR}_{i,1910-3} \cdot d_t + \delta X_i \cdot I_{\{1918\}} + d_i + d_t + d_{kt} + \varepsilon_{it}, \quad (1)$$

where MR_{it} is the mortality rate in parish i and year t . The mortality rate is regressed on binary dummy variables that indicate parishes with medium income (MI_i) and coal-fired capacity (MP_i), and high-income (HI_i) and coal-fired capacity (HP_i), each interacted with a set of time-fixed effects d_t . The coefficients β_{jt} are normalised, such that $\beta_{j,1917} = 0$. Thus, the estimator β_{jt} captures the differential change in the mortality rate from 1917 to year t in medium-income parishes relative to the change in low-income parishes, conditional on pre-pandemic characteristics.⁹⁷ The model is flexible enough to account for effects in years other than 1918, for example, during the spring wave in 1919.

To control for pre-pandemic parish characteristics, equation (1) includes the average mortality rate in 1910–3 of parish i ($\overline{MR}_{i,1910-3}$), interacted with a set of time-fixed effects d_t and a set of time-invariant parish-specific control variables X_i that are interacted with an indicator variable $I_{\{1918\}}$. The indicator variable $I_{\{1918\}}$ is one for the pandemic year 1918 and zero otherwise. Furthermore, equation (1) includes parish-fixed effects d_i that control for any time-invariant parish characteristics, for example, geographic factors. The time-fixed effects d_t and district times year-fixed effects d_{kt} control for influences on mortality that vary by time and district, like weather shocks. The standard errors ε_{it} are clustered at the county level.

The control variables in X_i are as specified in table 2 and can broadly be grouped into two categories. The first includes variables that control for socio-economic development and related pre-pandemic health differences between parishes. Population size and density, industrial employment share, firm size, installed hydroelectric capacity, the share of non-local born inhabitants, the population share aged 25–30, the share of female population, and access to health infrastructure can be grouped into this first category. Population size and density are included because there is empirical evidence that larger cities in the United States might have been able to implement more effective NPIs or had a higher immunity in the second wave due to an earlier exposure to the virus.⁹⁸ On the other hand, densely populated areas could have enhanced the spread.⁹⁹ The

⁹⁶ Hornbeck and Clay et al. use a similar empirical strategy (Hornbeck, ‘American dust bowl’, Clay, Lewis, and Severnini, ‘Pollution’).

⁹⁷ The interpretation of the estimators β_{jt} is analogous. The results are robust to changes in the baseline year (see the discussion below).

⁹⁸ Acuna-Soto, Viboud, and Chowell, ‘Influenza and pneumonia mortality’; Clay, Lewis, and Severnini, ‘Cross-city variation’.

⁹⁹ Mills, Robins, and Lipsitch, ‘Transmissibility’; Chowell et al., ‘The 1918–1919 influenza pandemic’.



transmission of the virus might have been also higher if people had more contact in their workplace. Therefore, I control for the average establishment size.

Several studies document the effect of pre-pandemic health and demographic factors on pandemic mortality.¹⁰⁰ Here, I include the average mortality rate in 1910–3, the share of population born outside of the parish, the share of population aged 25–30, and the share of female population to control for this effect.¹⁰¹ A higher average mortality rate in 1910–3 indicates a poorer local health environment or a different age structure of the population or both. As shown above, the age and sex structure are of particular interest because young adults were among the prime victims of the 1918 influenza and there are considerable differences in mortality rates by sex caused by the war. The share of the non-local-born population accounts for workers who migrated to economic centres during the rural flight. These (internal) migrants were younger and poorer and had worse health and housing conditions than the average local population.¹⁰² Moreover, they tended to have a lower immunity because they had less exposure to pathogens.¹⁰³ The inclusion of the industrial employment share and establishment size controls for adverse health outcomes of industrial employment caused by the relatively low level of occupational safety. Although there was no cure for the 1918 influenza, historical accounts document the beneficial effects of care. I control for this effect by access to health infrastructure, measured as the shortest distance to the nearest parish with a medical doctor and shortest distance to a hospital.

The second category of control variables captures the potential difference in the exposure to the virus and the timing of onset. Parishes with better access to transport infrastructure might have been more exposed to the virus and might have had an earlier onset.¹⁰⁴ However, the direction of this effect on pandemic mortality is unclear. An earlier onset of the pandemic might have increased mortality because the virulence may have declined over time.¹⁰⁵ On the other hand, very remote parishes might have even escaped the pandemic.¹⁰⁶ Therefore, I include binary indicators that control for access to the railway, central roads, and navigable waterways. I also control for the distance to the nearest military base because the spread of the virus was likely accelerated by the movement of troops.¹⁰⁷

For a causal interpretation of the effect of income and coal capacity, it must hold that, conditional on control variables, the expected change in pandemic mortality rates would have been the

¹⁰⁰ Bootsma and Ferguson, 'Effect of public health measures'; Clay, Lewis, and Severnini, 'Cross-city variation'.

¹⁰¹ Clay, Lewis, and Severnini, 'Pollution'; eisdem, 'Cross-city variation'.

¹⁰² Table 2 shows that the average mortality rate is lower in parishes with higher income and medium pollution. Thus, one channel of the income effect could be the effect on pre-pandemic health differences, if the model does not capture the pre-pandemic health sufficiently. Likewise, the estimator of the pollution effect would be upward biased, if less healthy individuals sorted into highly polluted parishes and the pre-pandemic health differences are not captured by the model in equation (1). The insignificant differences in the average mortality rate for high-pollution parishes, however, do not indicate such a selective migration pattern.

¹⁰³ Mamelund, 'Spanish influenza mortality'.

¹⁰⁴ Hogbin, 'Railways'.

¹⁰⁵ Clay, Lewis, and Severnini, 'Cross-city variation'. The higher case fatality rates in the second wave could have been due to an increased frequency of secondary bacterial pneumonia rather than an increased virulence of the influenza virus (Taubenberger, Kash, and Morens, 'The 1918 influenza pandemic'). At the same time, more central parishes might have seen a stronger first wave and thus had a higher immunity in the second, deadlier wave (Acuna-Soto, Viboud, and Chowell, 'Influenza and pneumonia mortality'; Clay, Lewis, and Severnini, 'Cross-city variation').

¹⁰⁶ Erköreka, 'Safe villages'.

¹⁰⁷ Patterson and Pyle, 'Geography and mortality'.

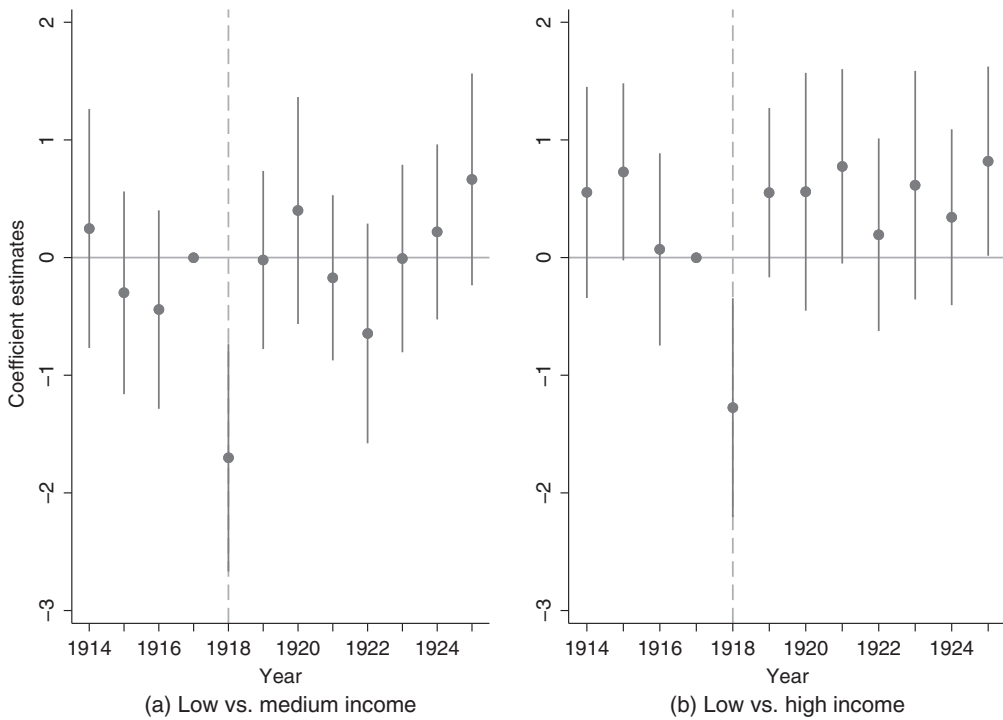


FIGURE 6 Estimated difference in mortality rate changes by income. The graph depicts differences in mortality rates between parishes with middle and low income per capita levels (left) and high and low income per capita levels (right), as estimated in an event study regression. Differences are expressed relative to the baseline difference in 1917. Point estimates are marked by a dot. The vertical bands indicate the 95 per cent confidence interval of each estimate. The dashed vertical line indicates the pandemic year 1918. Author's design

same across parishes with low, medium, and high income (coal capacity) in the absence of the difference in income (coal capacity). In terms of the difference-in-differences model, this assumption is referred to as the common trend assumption. It must also hold that there is no unobservable factor that influences mortality and correlates with income and coal capacity. Since I control for parish-fixed effects, time-fixed effects, and time- times district-fixed effects, these unobservable factors would also need to vary over time at the sub-district level to bias the estimates.

V | THE EFFECT OF INCOME AND POLLUTION ON MORTALITY

Figure 6 shows the differential changes in average mortality rates between middle- and low-income parishes (left panel) and high- and low-income parishes (right panel) from 1914 to 1925, relative to the baseline year 1917. The results are based on a reduced version of equation (1), excluding the indicators for pollution, the control variables in X_i , and county- times year-fixed effects.¹⁰⁸ The vertical bars in figure 6 indicate 95 per cent confidence intervals. The pandemic year 1918 is indicated by a vertical dashed line.

¹⁰⁸ Formally, this renders equation (1) to: $MR_{it} = \sum_{t=1914}^{1925} \beta'_{1t} MI_i \cdot d_t + \sum_{t=1914}^{1925} \beta'_{2t} HI_i \cdot d_t + \sum_{t=1914}^{1925} \gamma'_t \overline{MR}_{i,1910-3} \cdot d_t + d_i + d_t + \varepsilon_{it}$.

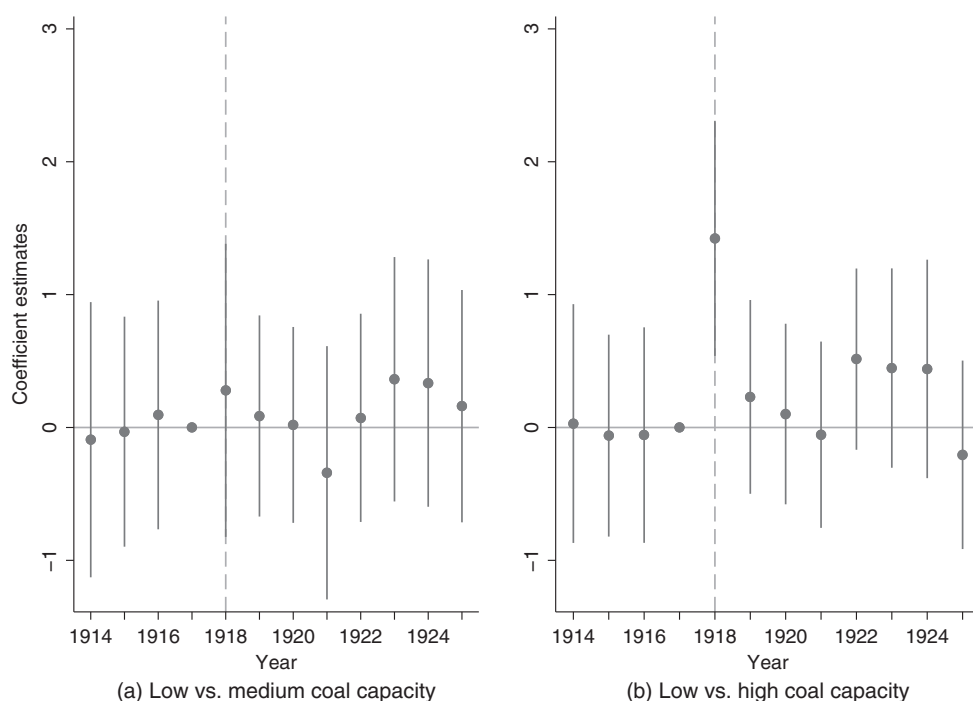


FIGURE 7 Estimated difference in mortality rate changes by coal capacity. The graph depicts differences in mortality rates between parishes with middle and low coal capacity levels (left) and high and low coal capacity levels (right), as estimated in an event study regression. Differences are expressed relative to the baseline difference in 1917. Point estimates are marked by a dot. The vertical bands indicate the 95 per cent confidence interval of each estimate. The dashed vertical line indicates the pandemic year 1918. Author's design

In low-income parishes, the mortality rate increased from 13.4 in 1917 to 17.3 in 1918, an increase of 3.9 deaths per 1000 persons or 29 per cent. The mortality rate increase in middle-income parishes relative to low-income parishes is significantly lower by -1.7 deaths per 1000 population. The same holds for high-income parishes with a point estimate of -1.3 . Both estimates are statistically significant at the 1 per cent level. Thus, the spike in 1918 mortality was particularly large in poor parishes.

Similarly, figure 7 compares the changes in mortality between parishes in the low tercile of installed coal-fired capacity and parishes in the medium (left panel) and high tercile (right panel) from 1914 to 1925, relative to the baseline year 1917. The estimation is based on a reduced model as in figure 6, but with indicators for medium and high coal capacity MP_i and HP_i instead of income indicators. There is no significant difference in the mortality rate changes in parishes with medium coal capacity compared with parishes with low coal capacity. Parishes with high levels of installed coal-fired capacity, on the other hand, have a significantly stronger increase in mortality rates. In these parishes, the mortality rate increases by an additional 1.4 deaths per 1000 population. The effect is statistically significant at the one per cent level.

Thus, figures 6 and 7 indicate a lower mortality rate increase for parishes with higher income and less pollution between 1917 and 1918. Additionally, the figures show that the estimates for the pandemic year 1918 deviate strongly from all other years. There is no general difference in mortality rate changes that distinguish the different parish groups, other than in 1918. Indeed, only 3 out of the 44 reported estimates in figures 6 and 7 are statistically significant at the 10 per cent



TABLE 3 Baseline results – DiD estimates

Mortality rate 1914–25								
	Base year 1917						Base year 1914	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Income medium × 1918	−1.702*** (0.483)	−1.354*** (0.470)			−1.568*** (0.501)	−1.306*** (0.478)	−1.835*** (0.492)	−1.628*** (0.525)
Income high × 1918	−1.276*** (0.465)	−0.998* (0.524)			−1.014** (0.502)	−0.893* (0.531)	−1.607*** (0.568)	−1.617** (0.644)
Coal medium × 1918			0.279 (0.552)	0.423 (0.481)	0.086 (0.554)	0.370 (0.480)	0.081 (0.571)	0.262 (0.486)
Coal high × 1918			1.423*** (0.442)	1.665*** (0.514)	1.157** (0.471)	1.627*** (0.530)	0.981* (0.563)	1.166** (0.567)
Parish FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Controls	No	Yes	No	Yes	No	Yes	No	Yes
Year × district FE	No	Yes	No	Yes	No	Yes	No	Yes

Note: The table presents panel regression estimates of the effect of taxable income 1907 and installed coal-fired power plant capacity within 50 km on the differential change in mortality rates. Regressions (1) and (2) show the change in medium- and high-income parishes between 1917 and 1918 relative to the change in low-income parishes. Regressions (3) and (4) show the change in parishes with medium and high coal capacity between 1917 and 1918 relative to the change in parishes with low coal capacity. Regressions (5) and (6) include income and coal capacity measures. Regressions (7) and (8) show the differential change between 1914 and 1918. All regressions include a full set of year- and parish-fixed effects, as well as the average mortality rate 1910–3 interacted with year-fixed effects. Regressions (2), (4), (6), and (8) include the full set of pre-treatment control variables X_i , each interacted with an indicator variable for the year 1918 and year- times district-fixed effects. Number of observations: 21 156. Standard errors clustered at the county level are in parentheses. FE, fixed effects. ***, **, and * denote statistical significance at the 1, 5, and 10 per cent level, respectively.

Sources: See descriptions in Section III.

level (not counting the 1918 estimates), and all show the opposite sign.¹⁰⁹ Out of the 12 estimates for the pre-pandemic period, only one is statistically significant. These estimates provide suggestive evidence for the common trend assumption to hold because there is no systematic difference in mortality rate changes in non-pandemic years.

Table 3 columns (1) and (3) show the point estimates and standard errors for the interactions of medium- and high-income and coal capacity indicators with the indicator for 1918. The results correspond to the results in figures 6 and 7. Column (2) includes the full set of control variables as well as district- times year-fixed effects. The estimates for the income effect in column (2) are slightly lower but remain statistically and epidemiologically significant.

Table 3 columns (3) and (4) show the results for coal-fired capacity. The point estimate is slightly larger in column (4), where I include the full set of controls and district- times year-fixed effects. The change in mortality rates is almost identical for parishes with low and medium coal capacity, an average increase of about 2.4 and 2.6 deaths per 1000 population between 1917 and 1918.

Column (5) includes the measures for income and coal capacity simultaneously, and column (6) adds the full set of control variables. Thus, the estimates in column (6) are based on the full model as specified in equation (1). Conditional on pre-pandemic characteristics and the installed

¹⁰⁹ If the mortality rate in the reference year 1917 is higher in poor and highly polluted parishes, this would downward bias the 1918 estimates.



coal capacity, the change in the all-cause mortality is 1.3 less in medium- relative to low-income parishes. Likewise, the average change in the mortality rate is lower by 0.9 deaths per 1000 population for parishes in the high-income tercile compared with the low-income tercile. On the other hand, parishes with the highest level of installed coal-fired capacity had, on average, an additional increase of 1.6 deaths per 1000 population. These effects are large compared with an average increase in the mortality rate between 1917 and 1918 of 2.9.

The virus that caused the influenza pandemic might have already spread before 1918.¹¹⁰ Moreover, the mortality rate in 1917 was relatively high owing to starvation and the harsh winter. If the mortality rate in 1917 is negatively correlated with income and positively correlated with coal-fired capacity, the estimates in columns (1)–(6) would represent a lower bound. Columns (7) and (8) present results using 1914 as the baseline year instead of 1917. The income estimates indicate a stronger effect, while the estimates for pollution are lower. Thus, using 1917 as reference year might lead to more conservative income estimates, while it biases pollution effect estimates upwards. However, in both specifications, the pollution and income effect are statistically significant.

Table A13 in the online Appendix reports the associated control variable estimates, most of which are not statistically significant. However, the share of non-local-born population and the railway dummy have a significant effect in all specifications. Railway access reduces the mortality rate increase by about 0.9 deaths. The lower increase in mortality rates is likely due to the higher exposure during the early, less lethal wave and the acquired immunity.¹¹¹ An increase in the share of the non-local-born population by one standard deviation increases the mortality rate by an additional 0.5 deaths. The non-local-born population includes workers that migrated to industrial centres. These workers are on average younger and poorer, and often experienced poor housing conditions. Thus, the results are in line with earlier studies that show the adverse effects of low social status and poor housing.¹¹² In addition, these rural migrants tend to have lower immunity because they were exposed to fewer pathogens in their earlier lives.¹¹³

In the previous estimations, I control for a variety of pre-pandemic factors. However, to address potential concerns that the results are driven by model specifications, characteristics of the sample, or the construction of variables, I perform several robustness checks (see online Appendix A5 for a detailed discussion). For example, I re-estimate the main specifications from table 3, but only for the period 1914–8. This specification allows the parish-fixed effects to control for any time-invariant parish-specific effects of the First World War. In the robustness checks, I also control for war casualties, include infant mortality in the dependent variable, test different pollution measures, use income and pollution quintiles or their actual units of measurement, and allow for spatial correlation of the standard errors. Furthermore, I control for the effect of wind patterns on air pollution (see online Appendix A6). All robustness checks confirm the beneficial effect of higher income levels and the adverse effect of higher levels of coal-fired capacity on pandemic mortality rates.

The paper contributes to the understanding of the adverse effects of low income and high pollution during the 1918 influenza pandemic in rural areas. However, there are a few urban centres

¹¹⁰ Johnson, 'Aspects of the historical geography', pp. 119–21.

¹¹¹ Acuna-Soto, Viboud, and Chowell, 'Influenza and pneumonia mortality'; Clay, Lewis, and Severnini, 'Cross-city variation'; Mamelund, 'Spanish influenza mortality'.

¹¹² Bengtsson, Dribe, and Eriksson, 'Social class'; Sydenstricker, 'The incidence of influenza'; Chowell et al., 'The 1918–1919 influenza pandemic'.

¹¹³ Mamelund, 'Spanish influenza mortality'.



in Württemberg. I truncate the sample on the basis of population size in 1910, that is, I exclude the largest and smallest 1 (5) per cent of parishes from the sample. The truncation reduces the average population size in the sample from 1354.4 to 972.9 (809.4). The results are in line with the baseline results (see online Appendix [table A14](#)). Thus, the effects are not driven by a significantly different health environment in the few urban centres or outliers in small villages' mortality rates.

In addition, I re-estimate the main specifications but exclude parishes close to urban centres (defined as towns with a population above 10 000 inhabitants). Table [A15](#) in the online Appendix presents the results. Panel A excludes parishes within 6 km from urban centroids (approximately the tenth percentile) and panel B parishes within 10 km (approximately the 25th percentile). Both specifications provide estimates in line with the previous results. Yet, the adverse effect of high coal capacity increases to 2.130 (s.e. 0.768) in the full model for parishes more than 10 km from urban centres (panel B, column (6)). This result indicates the importance of pollution externalities for rural areas, that is, rural parishes do experience pollution from the urban centres, but not the associated economic benefits. However, the excluded parishes are on average richer and more polluted, so the results should be interpreted with caution.

Finally, Section A8 in the online Appendix discusses results using data aggregated at the county level. The aggregated dataset comprises 63 counties. The point estimates for income and coal-fired capacity show the expected sign; however, none of these estimates is statistically significant. The statistical insignificance might be due to the smaller sample size but also due to the aggregation itself masking within-county heterogeneity. Therefore, the county-level estimates emphasise the importance of complementing existing studies at the aggregate level with analyses using more finely grained data.

VI | CONCLUSION

This paper analyses mortality in the 1918 influenza pandemic in the Kingdom of Württemberg and the effect of income and pollution on pandemic severity. The Spanish Flu reached the southwest German state during the hardships of the First World War. The suffering due to starvation and the war, causing the death of more than 72 000 military personnel in Württemberg, in combination with a (self-)censored press could explain why the pandemic received little public attention and did not leave a lasting impression in the collective memory of the population. To put this in perspective, the 72 000 deaths of military personnel account for about 30 deaths per 1000 inhabitants in 1910, whereas the estimates of the pandemic mortality rate in Württemberg presented in the paper range between 2.3 and 4.1 excess deaths per 1000 persons.

However, the 1918 influenza pandemic led to a significant increase in all-cause mortality rates in Württemberg of 23 per cent relative to 1917. The paper shows that this increase was larger in poor and highly polluted parishes. Parishes with high levels of coal-fired power plant capacity within 50 km faced an additional increase of 1.6 deaths per 1000 population relative to medium- and low-polluted parishes. Moreover, the relative increase in mortality rates was lower in medium- and high-income parishes by 1.3 and 0.9 deaths per 1000 population, relative to low-income parishes. However, the focus on coal-fired power plant capacity might render the pollution effects at the lower bound because it is an imperfect measure of the actual local pollution levels.

The data show that the mortality burden of the 1918 influenza pandemic was lower in Württemberg compared with other German states, even though Württemberg was relatively poor by German standards. The newly calculated estimates for the German excess mortality rate are between 5.4 and 7.0. One reason might be the relative backwardness of Württemberg's economy, resulting in low usage of coal compared with other German states and foreign countries.



For instance, in Clay et al.'s sample of US cities, the average installed coal-fired capacity within 30 miles (48.3 km) was 182.8 MW, while it was only 34.3 MW (within 50 km) in Württemberg.¹¹⁴ Moreover, Clay et al. showed that cities with medium coal capacity had on average an installed capacity over 50 MW, while in Württemberg, only parishes in the highest tercile reached this level.¹¹⁵ In line with this argument, the effect for parishes with medium coal capacity is insignificant in Württemberg, whereas Clay et al. find an effect also for cities with medium coal capacity.¹¹⁶ This suggests that the pollution level must exceed a certain threshold to negatively affect mortality in the 1918 influenza pandemic. Thus, the lack of coal that contributed to Württemberg's economic backwardness might have been beneficial in reducing the pandemic's death toll.

Previous studies have found a clear north–south gradient in excess mortality for European countries during the 1918 influenza, that is, higher excess mortality in the south compared with the north, but no explanation for this pattern.¹¹⁷ The results above show that income-level differences can provide such an explanation. The correlation coefficient between excess mortality rates and log GDP per capita in 1910 for 14 European countries is -0.7 and statistically significant at the 1 per cent level.¹¹⁸ Therefore, the results for Württemberg might encourage future research at the European level.

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¹¹⁴ Clay, Lewis, and Severnini, 'Pollution', p. 1188.

¹¹⁵ Ibid., p. 1188.

¹¹⁶ Ibid.

¹¹⁷ Ansart et al., 'Mortality burden', p. 104.

¹¹⁸ The 'countries' are Bulgaria, Denmark, England & Wales, Finland, France, Germany, Italy, the Netherlands, Norway, Portugal, Scotland, Spain, Sweden, and Switzerland. I assign the British GDP per capita to England & Wales and Scotland (Ansart et al., 'Mortality burden'; Bolt and van Zanden, 'Maddison Project Database').



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