Temperature, Disease, and Death in London:

Analyzing Weekly Data for the Century from 1866-1965*

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June 1, 2020

Abstract

Using weekly mortality data for London spanning 1866-1965, we analyze the changing relationship between temperature and mortality as the city developed. Our results show that both warm and cold weeks were associated with elevated mortality in the late 19th-century, but heat effects, due mainly to infant deaths from digestive diseases, largely disappeared after WWI. The resulting change in the temperature-mortality relationship meant that thousands of heat-related deaths–equal to 0.8-1.3 percent of all deaths–were averted. Our findings also indicate that a series of hot years in the 1890s substantially changed the timing of the infant mortality decline in London.

* We thank Vellore Arthi, Brian Beach, Alan Barreca, Robert Margo, Andrew Oswald, and seminar participants at the European Economic Association Meeting, the Copenhagen Business School Inequality Platform's First Workshop on Climate Change and Inequality, and the 5th Annual Meeting of the Danish Society for Economic and Social History for their helpful comments. Funding for this project was provided by National Science Foundation CAREER Grant No. 1552692.

1 Introduction

Understanding historical mortality patterns has been a long-term goal of economic historians and demographers. Studying historical mortality serves both to provide a deeper understanding of long-run economic and demographic trends as well as offering useful lessons for modern developing countries as they seek to follow the path of those that have already developed. Within this topic, one subject that has attracted considerable attention is the relationship between mortality and weather conditions, particularly temperature.[1] It has long been recognized that mortality followed seasonal patterns and that these patterns have evolved over time.[2] Recently, there has been renewed interest in this topic because it has been recognized that understanding how the relationship between temperature and mortality has evolved in the past has the potential to offer useful lessons as the world confronts the challenges posed by rising temperatures in the future.[3] Yet, our current understanding of the temperature-mortality relationship, particularly before the 20th century, remains incomplete. The key impediment to progress in this area has been a lack of detailed high-frequency mortality data that can be compared to available measures of temperature and other weather variables. As a result, most existing studies looking at the period before WWII have been forced to use annual or quarterly level data, particularly those studies that look back into the 19th century.[4]

In this paper, we introduce a new set of detailed weekly mortality observations from London, starting in 1866 and spanning a full century, which we use to advance our understanding of the historical temperature mortality relationship. These data, which we digitized from original printed

[1] See Section 2 for a review of this literature.
[2] See discussion in Section 2 of this paper.
[3] A prominent recent example using historical mortality patterns to inform our expectations of the impact of rising temperatures on mortality is Barreca et al. (2016).
[4] See, for example, Galloway (1985), Landers and Mouzas (1988), Woods, Watterson and Woodward (1988), Williams (1992), Mooney (1994), Williams and Galley (1995) and Huck (1997), all of which study the seasonality of mortality or the relationship between temperature and mortality in Britain for the period before WWII. These papers all use either annual or quarterly mortality data. One exception that does use weekly data before WWII is Carson et al. (2006), but that study does not examine patterns before 1900. For the U.S., see Fishback et al. (2011), which uses annual data from the 1930s, Anderson et al. (2020b), which uses monthly data on infant deaths from 1910-30, and Barreca et al. (2016), which uses monthly data for 1900-2004 (though most of their analysis focuses on the period after 1959). Two studies that do use high-frequency (daily) data are Petkova et al. (2014) looking at New York City and Åström et al. (2013) looking at Stockholm, but neither of those studies consider data before 1900. One study that does have high-frequency data stretching into the 19th century is Ekamper, et al. (2009), which uses data from the Dutch province of Zeeland stretching back to 1855.

source material, are unique in that they provide high-frequency observations of mortality, broken down by age group and cause-of-death, for an unusually long period. The richness of this data set, which contains over 300,000 observations, allows us to assess how the relationship between temperature and mortality evolved as London developed, as well as the role of the disease environment in influencing the health effects of temperature.[5]

We combine our mortality data with one of the longest continuously-observed single-site weather series in the world, taken from the Radcliffe Observatory in Oxford, roughly 80km outside of London. These data allow us to track temperature as well as other weather conditions in a consistent way over our study period. Using high-frequency mortality and climate data allows us to identify the mortality effects of temperature applying a fairly simple empirical strategy that exploits week-to-week variation in weather conditions, while the age and cause-of-death data shed light on the mechanisms through which both high and low temperatures influence mortality.

Our analysis shows that, in the late 19th-century and early 20th-century, both unusually cold and unusually warm weather was associated with elevated mortality. Cold weather tended to increase mortality among the elderly, typically from respiratory-related diseases (e.g., pneumonia), while warm weeks were associated with elevated mortality among infants and young children. Prior to WWI, and particularly before 1900, these warm-weather deaths were heavily concentrated in digestive diseases. However, the patterns of warm-weather deaths changed substantially over time.

A particularly interesting finding of our analysis is that the strong association between unusually warm weather and mortality in London began declining after 1900 and had largely disappeared after WWI. This disappearance is driven by a reduction in infant deaths, and specifically those due to digestive diseases. As a result, weeks with unusually warm weather became much less deadly as London developed. The reduced mortality associated with warm temperatures after WWI contrasts with the impact of cold weather, which was largely unchanged across the study period. The fact that the change in the effect of warm weather on mortality was concentrated in infant digestive diseases indicates that it was driven by changes in the underlying disease environment.

[5] The only other modern study that we are aware of using the Registrar's weekly data is Carson, et al. (2006), but they do not consider data before 1900 and their analysis of the data is somewhat limited.

Using weekly mortality data in our analysis offers three distinct advantages relative to studies based on annual, quarterly, or even monthly data. First, the weekly frequency allows us to go further in exploring the dynamics of the temperature effect, such as how the effect of a particularly hot week evolved over the following weeks. Second, using higher-frequency data allows us to avoid problems created by a lack of high-frequency population denominators. The fact that population in a location is typically only well-observed once every decade, during the census, creates an important challenge for studies using annual or quarterly data, because it is never clear whether a change in the number of deaths observed may be due to changes in the underlying at-risk population, due to migration or the presence of seasonal workers. As a result, almost all existing studies of the seasonality of mortality focus on infant deaths, which can be compared to births, data that are observed at the same frequency as the mortality statistics. We can overcome this concern with high-frequency data, because if we observe, say, a 5% increase in mortality in London during a week in which temperatures were unusually high or unusually low, relative to the weeks just before, it is implausible that the underlying population of the city could have changed by such a degree from one week to another to explain this difference. This makes it possible for us to extend our analysis beyond infant mortality, in order to assess the relationship between temperature and mortality across all parts of the age distribution. Finally, using more detailed data provides us with additional statistical power than what is available from more aggregated series.

Documenting the historical relationship between temperature and mortality also allows us to generate counterfactual assessments of the impact of rising temperatures under different conditions. As a demonstration of this, we use our results to provide counterfactual estimates of how the impact of temperature on mortality in London would have evolved without the shifts in the underlying disease environment that occurred in the early twentieth-century. These calculations show that the change in the temperature-mortality relationship that we observe after WWI, relative to before, was associated with a substantial reduction in *total mortality* equal to 0.8-1.3 percent of all deaths. These results indicate that, in environments with high infectious disease burdens, improvements in the disease environment can lead to very large reductions in the mortality impact of high temperatures.

We also conduct a second counterfactual that shows how temperature events, specifically a series of hot summers in the late 1890s, altered the timing of the mortality transition in London. In the counterfactual, we use our estimates to remove the impact of the series of unusually hot years in the 1890s on infant mortality. The results indicate that the decline in infant mortality in London would have started about five years earlier than indicated by the raw data had it not been for a series of unusually hot years. The fact that temperature events can shift the timing of London's mortality transition by several years has implications for studies, such as Anderson et al. (2020a), that rely on the timing of such transitions to identify the mechanisms behind mortality declines.

One implication of our results is that altering the underlying disease environment has the potential to help locations adapt to rising temperatures. This adaptation mechanism has been mostly ignored by the modern economics literature on climate change adaptation. Instead, existing work, which for data reasons is largely drawn from modern developed countries, has emphasized mechanisms such as electrification and the introduction of air conditioning, migration, or changes in time use patterns.[6] Yet our results show that altering the disease environment, and specifically reductions in digestive diseases, can have a substantial impact on the effect of rising temperature on mortality. These lessons may be useful as the developing world struggles to adapt to climate change.

One striking feature of our results is how much they differ from studies looking at more recent settings with lower infectious disease burdens, such as Barreca et al. (2016). For example, in their study, the impact of high temperature was due mainly to cardiovascular and respiratory diseases. In our results, the impact of high temperature is driven instead by diseases of the digestive system. A straightforward explanation for this difference is the vastly different disease environment that they study. In our data, in 1871, digestive diseases alone accounted for 1.7 deaths per thousand, 1.4 deaths per thousand in 1911, and 0.16 deaths per thousand in 1931. In contrast, in the cause-of-death data used by Barreca et al. (2016), all infectious diseases accounted for just 0.2 deaths per thousand people. To put this into the perspective of modern developing countries, in Southeast Asia and Sub-Saharan Africa in 2017, estimates from the Global Burden of Disease (Troeger et

[6] See Basu and Samet (2002) and Deschenes (2014) for reviews of this literature. On electrification and the use of air conditioning, see Chestnut et al. (1998), Braga et al. (2001), Curriero et al. (2002), Deschenes and Greenstone (2011), Barreca (2012), and Barreca et al. (2016). On the role of geographic mobility see, e.g., Deschenes and Moretti (2009) and Bohra-Mishra et al. (2014). On changes in time use, see Graff Zivin and Neidell (2014).

al., 2018) suggest that diarrheal deaths (a somewhat narrower definition than our digestive deaths, and much narrower than all infectious diseases) accounted for, respectively, 0.11 and 0.62 deaths per thousand. Thus, infectious disease deaths in modern developing countries are many times more important than they were in the environment studied by Barreca et al. (2016) and comparable, though probably not as high, as the levels observed in London in the early 20th-century.

The remainder of this study proceeds as follows. In the next section, we describe the empirical setting and related literature. The data are introduced in Section 3, followed by the analysis in Section 4 and concluding remarks in Section 5.

2 Background and related literature

In the middle of the 19th century, when our study begins, Londoners experienced what are by modern standards extremely high mortality rates, comparable to the levels faced by the very poorest urban residents in developing countries today.[7] Much of this mortality was concentrated in infants and young children, and it is useful to focus on this group for comparison purposes because this has been the main focus of existing research and, as we will see, this group plays a central role in the relationship between temperature, mortality, and the disease environment. In 1860-1899, at the beginning of our study period, out of every thousand children born in London, 158 died before age one in an average year (Williams and Mooney, 1994). While high by modern standard, this put London just above the national average (149 per thousand) and below more industrialized cities like Birmingham and Manchester (Williams and Mooney, 1994).[8] Starting from this high level, infant mortality dropped to 132 per thousand births in 1900-10, nearly halved to 66 per thousand in the 1920s, and fell to 24 per thousand in the 1950s, at which point infant mortality accounted for just 2.5% of all deaths.[9] In this pattern London was relatively similar to the average

[7] As a point of comparison to the figures mentioned for London later in this paragraph, in 2017 the infant mortality rate was 109 per thousand in Sub-Saharan Africa and 89 in South Asia, 44 in Latin America, 42 in China, and 13 in Europe. See Hug et al. (2017).
[8] See also, Lee (1991).
[9] Authors calculations using the data described in Section 3.

across British cities, though there has been some dispute about the similarity in the exact timing of the onset of the infant mortality decline.[10]

Our data show that in the 1870s, most deaths were due to infectious diseases. The largest single cause of all-age mortality was tuberculosis (14% of all deaths), followed by bronchitis (13%). Cardiovascular diseases accounted for only about 6% of deaths, followed by pneumonia (5.4%) and whooping cough (3.6%). Cancer accounted for less than 3% of deaths. Among infants the most important category was digestive diseases, which accounted for 13% of all deaths from 1876-85. By the 1950s, infectious diseases had become much less important, and overall mortality, which was concentrated among older adults, was driven by cardiovascular factors (28% of deaths), cancer (22%) and stroke (9%). Even for infants, digestive diseases were much less important, accounting for just under 3% of infant deaths (65% of infant deaths were attributable to either prematurity, birth injuries, or congenital defects).

A substantial amount of scholarly work has been focused on understanding the mortality transition in London, its causes, and how it compares with other parts of the country. Of this extensive literature, the most relevant for our study is work looking at the seasonality of deaths. A remarkable early paper in this area is Buchan and Mitchell (1875), written for the Scottish Meteorological Society, which calculated the average death contribution by each week of the year from 1845-1874 using the weekly mortality statistics produced by the Registrar General's office. These weekly data seem to have been largely overlooked by studies of historical mortality patterns in London until a recent paper by Carson, et al. (2006), which examines mortality patterns in the 20th century. Relative to their paper, our study includes several decades of data from the 19th century. This difference is important, because it means that our analysis begins before the sharp decline in infant mortality in London began (see Appendix Figure A.3). We also offer a richer analysis framework that captures the importance of lagged mortality effects as well as allowing us to calculate counterfactual mortality patterns.

Buchan and Mitchell were not the only contemporary observers to recognize the connection between season and mortality in British cities in the nineteenth and early twentieth-century. Arthur Newsholme discussed the seasonality of infant diarrheal deaths extensively in his Presidential Address to the Incorporated Society of Medical Officers of Health in 1899.[11] He continued to write on the subject when he became the

[10] See Woods, et al. (1988), Lee (1991) and Williams and Mooney (1994).
[11] Newsholme (1899).

Medical Office of the Local Government Board (e.g., Newsholme 1901). E.W. Hope, the Medical Office of Health of Liverpool undertook similar studies (Hope 1899a, 1899b), as did George Newman, the Medical Office for the district of Finsbury (Newman 1906). These and other contemporary observers, many of whom were local Medical Officers, were acutely concerned about the causes of the summer diarrhea patterns that they observed. They proposed a number of possible causes, including poor sanitation (particularly sewage removal), overcrowded living conditions, insufficient breastfeeding leading infants to be fed with contaminated milk or other food, and generally poor childcare among the working classes, often because the mother went back to work soon after giving birth. Contemporary observers generally agreed that all of these factors likely mattered, though opinions differed on whether the bulk of the blame should be placed on mothers, for a failure to adequately breastfeed or care for children, or local authorities, for their inability to provide adequate sanitation and healthy living conditions.[12]

In the intervening years, a number of other authors have studied the seasonality of mortality or how mortality was affected by temperature variation using either annual or quarterly data. Woods, Watterson and Woodward (1988), for example, run regressions comparing mean temperature to infant mortality in England and Wales for 1870-1911 (N=42) and find evidence that temperature in the third quarter of the year is positively related to mortality. After documenting substantially higher mortality in cities than in rural areas, they speculate that ``the most likely reason for this `urban effect' is that climatic conditions, especially during the third quarter of the year, interacted with poor urban sanitary environments which resulted in high levels of diarrhea and dysentery among infants…'' The same quarterly data has been used to study the seasonality of infant mortality by a number of other authors, including Mooney (1994) and Williams and Galley (1995). Wrigley and Schofield (1989, Chapter 9) also examines the relationship between their mortality data, based on Anglican parish registers, and monthly temperature. One notable feature of most of the recent work on the seasonality of mortality is that almost no attention is paid to the impact on older age groups, or the effect of unusually cold weather, two topics that we tackle in this paper.

One interesting discovery in existing work is that the seasonal nature of mortality in London appears to have been absent before the second quarter of the 19th century. While data are scarce before the beginning of general death registration in 1837, findings from the Bills of Mortality compiled by Landers and Mouzas (1988) show no evidence of a summer mortality peak in the late eighteenth or early nineteenth century

[12] See Woods, et al. (1989) for a useful review of the contemporary discussion of these topics.

(though the summer peak does appear in the early eighteenth century). A similar pattern is identified by Huck (1997) for a sample of industrial parishes in Northern England. Thus, it appears that the summer peak in infant mortality that plays such an important role in our results only emerged in British cities sometime in the first half of the nineteenth century.

Despite extensive research into the mortality decline that took place in British cities in the decades around 1900, no clear consensus has emerged as to the causes. It is clear that this decline was concentrated among infants and driven by the reduction in diseases of the digestive system, but why these diseases declined remains debated. McKeown, (1976) emphasized the importance of improving overall nutrition among the British population. McKeown's hypothesis finds some support in the work of Millward and Bell (1998), which also emphasizes the importance of improvements in the housing stock.[13] However, the importance of nutrition has been challenged by Szreter (1988), among others, who instead emphasized the role of public health measures.[14] Recent evidence from Chapman (2019) and Harris and Hinde (2019) provides support for the role of public health infrastructure. Chapman, for example, finds that between 30 and 60 percent of the mortality decline that took place in British cities from 1861-1900 can be explained by infrastructure investments.[15] However, the case for sanitary infrastructure investments playing a key role in reducing infant mortality in London is hard to make. Most of the major improvements, including water filtration and other water quality improvements, continuous water supply, and sewer access generally took place years or decades before the decline in infant digestive disease mortality that appears in our data (see, e.g., Appendix Figure A.3).[16]

One aspect of nutrition that has received particular attention, both by contemporary observers (e.g., Peters 1910) and in later research (e.g., Beaver 1973), is the availability of uncontaminated milk supplies. The importance of milk has found support in a recent study of the U.S. (Komisarow 2017) but not in others (Anderson, et al. 2020a), and the contribution of improvements in milk

[13] Harris (2004) also finds support for the role of nutrition over a longer time-frame (1750-1914).
[14] See also Szreter (2005). In fairness, while McKeown is best-known for arguing that improving living standards were critical for the overall decline in mortality, McKeown and Record (1962) recognized the critical role played by sanitary measures in reducing digestive disease mortality.
[15] A parallel literature on the role of water and sewer infrastructure exists for the U.S. See, e.g., Troesken (2002), Cutler and Miller (2005), Ferrie and Troesken (2008), Beach, et al. (2016), Alsan and Goldin (2019), Anderson, et al. (2020a) and Anderson, et al. (2020b).
[16] Another explanation, offered by Guha (1994), is that the population of cities may have become more resistant to the effects of diseases over time, through exposure or selection, though this claim is hard to substantiate. However, McKeown and Record (1962) argue that selection is unlikely to have been a critical factor, though their focus is on TB.

quality to the decline in British mortality in the decades around 1900 remains disputed (Atkins 1992). A closely connected factor is the level of maternal care, which was thought by some contemporaries to have a large influence on infant mortality during our study period (Newsholme 1899; Newman 1906), particularly among the many poorer mothers that returned to work soon after giving birth. In response, by the early twentieth century many local areas employed Health Visitors who helped educate mothers on the feeding and care of children (Fildes 1992; Mooney 1994). Evaluating the efficacy of this and other similar interventions remains challenging.

In summary, a variety of factors, including improved sanitation, water and milk quality, better general nutrition (especially for nursing mothers), improved maternal care and breastfeeding practices, and better housing conditions, have all been put forward as potential explanations for the decline in mortality in British cities in the decades around 1900. Many of these suggestions have found some empirical support, though almost all have also had their critics. It is not our purpose in this paper to attempt to settle this debate. What is clear, however, is that there was a general decline in mortality in London and other British cities commencing in the years around 1900, that this decline was concentrated among infants and young children, and that it was principally due to a decrease in digestive diseases. Taking these facts as given, in the remainder of this paper we consider the role of weather in contributing to mortality patterns in London and how the relationship was altered as the underlying disease environment changed.

Finally, this study connects to a literature in epidemiology using time-series methods to assess the impact of temperature on mortality. The large literature in this area using modern data includes papers such as Hajat, et al. (2002), which studies the mortality effects of heat using daily data for London from 1976-1996. There is also a smaller historical literature in this area, which includes, in addition to the paper by Carson, et al. (2006) on London cited above, papers by Petkova et al. (2014) looking at New York City, Åström et al. (2013) looking at Stockholm, and Ekamper, et al. (2009), which uses data from the Dutch province of Zeeland. Of these, only the Ekamper et al. paper uses data stretching into the 19th century, though their data comes from a mainly rural/agricultural setting. Our analysis approach differs substantially from the one used in these papers. In particular, we do more to assess the lag structure of temperature effects, which are ignored in many existing studies. We also provide counterfactual estimates that allow us to quantify the impact of temperature and how this impact changed over time.

3 Data

The weekly mortality data used in this study were digitized from printed reports produced by the Registrar General's office collected mainly from the LSE Library and the New York Public Library. The collection of birth, marriage and mortality data by the Registrar's Office, which commenced in the 1830s and was well-established by the 1870s, was an enormous undertaking. The system was registering as many as a million events per year in the 1840s and 1.7 million by the early 20th century, which, "considering the detail of reporting and the standard of accuracy obtained…was a very considerable achievement'' (Woods 2000, p. 36). To undertake this task, a new office was established within the civil service, headed by the Registrar General and employing a professional staff (Woods 2000, p. 31-32). These oversaw a network of local registrars and superintendent registrars drawn from among local officials or leading citizens (Woods 2000, p. 31). Within this system, it was to the collection of mortality statistics "that most attention was given and from which the greatest detail was sought'' making them the "shining star of Victorian civil registration."

The 300,000 mortality observations in our main data cover 4,540 weeks stretching from 1866 to 1965, with breaks in 1915-1918 (WWI) and 1940-1948 (WWII). We end our main study period in 1965 because the geographic area for which our data are reported changed at that point, though in Appendix A.2 we present supplementary results for 1981-2006.[17] One benefit of ending our analysis in 1965 is that we avoid the impact of air conditioning, which began to be used in some locations in London around that time.

In each week, we observe deaths broken down by cause and age group for the city as a whole, so our analysis data set is structured as a single time-series. Our mortality data come from a consistent geographic area that encompasses all of modern Central London. This area is somewhat smaller than the current Greater London administrative area, which was established in 1966, but much larger than the City of London. Administrative changes in 1966 resulted in a change in the geographic area covered by the weekly data series to the modern Greater London. This change motivates our decision to end the study period in 1965.

[17] For 1981-2006 we obtained data from the Office of National Statistics (ONS). Unfortunately, the ONS is not willing to provide breakdowns of weekly deaths by age group or cause for this period.

Our analysis focuses mainly on either all-age mortality, which is available for the full study period, or infant mortality, which is available starting in 1874. The availability of weekly mortality data with complete breakdowns by age and cause of death is unique to London. While some weekly mortality figures are reported for other cities, or for neighborhoods within London, only for London as a whole do we observe the fully detailed breakdown of age-group by cause-of-death, so this is our focus.

The cause-of-death categories require some standardization. In our data set, causes of death are reported in as many as 130 different categories in some years, and as few as 57 in others, with substantial changes in the reported categories over time. To deal with these changes and generate series that are reasonably consistent across the study period, we collapse these causes of death into more aggregated categories, such as digestive diseases (including diarrhea, dysentery, cholera, typhoid, etc.) or respiratory diseases (bronchitis, asthma, etc.). While historical cause-of-death data must be treated with some caution, within these broadly-defined categories it is likely that most diseases are correctly categorized, particularly deaths due to the most common causes. Importantly, digestive diseases, the most important category for this study, typically show clear defining features, so the classification of these diseases should be reasonably accurate, even early in our study period. The causes of death categories change substantially after WWII. For digestive diseases, which includes diarrhea, dysentery, enteritis, gastritis, typhoid and cholera, we are able to generate a consistent series up to 1965, but when we look at other causes of death we focus only on data before WWI.

The existing literature provides a vigorous discussion of potential issues in the use of the Registrar General's mortality data (see, e.g., Luckin 1980; Hardy 1994). One common concern is the completeness of the records. However, for London in the period we study, this is unlikely to be a major concern; Hardy (1994) reports that registration rates reached 98 percent in London by 1870. Another issue is the classification of causes of death, which naturally evolved over time as medical knowledge advanced.[18] Certain disease categories, such as tuberculosis, which was difficult to diagnose, have been highlighted as particularly problematic (McKeown and Record

[18] Very few (less than 1%) of the deaths in our data are unclassified. A more important issue is that many deaths were classified into general categories such as "other zymotic diseases" or "other lung diseases, etc." and other deaths may have been misclassified.

1962, Hardy 1994). However, the most important category for our analysis, diarrheal diseases, is "one of the least troublesome'' according to Hardy (1994) because the fact that it represented a symptom, rather than a disease, made it relatively easy to recognize. This is particularly true in our main analysis, since we have combined diarrheal diseases with those other categories (cholera, enteric fever) which were those most likely to be confused with one another.

Our temperature data come from the Radcliffe Observatory in Oxford, 80km outside of central London. We have two primary reasons for preferring the temperature data series from Oxford over other alternatives. First, they come from a single location, while the weather data for London reported in the Registrar General's report comes partially from Greenwich Observatory and partly (after WWII) from Kew Gardens. Second, the Oxford data provide both average maximum and average minimum temperature for each week, rather than just weekly mean temperature. This additional detail is useful for focusing specifically on heat and cold-related deaths. Another advantage of using data from Oxford is that it is far enough outside of London that temperature there is unlikely to be substantially influenced by urban heat-island effects.[19] Dealing with the potential endogeneity created by urban heat-island effects is important if we want to identify the causal impact of temperature on mortality. Finally, we use some additional weather data tracking precipitation and humidity from the Greenwich Observatory and Kew Gardens.[20]

In our main analysis, we assess the impact of temperature non-parametrically by dividing weeks into temperature bins. Given London's relatively mild climate, we examine heat effects by looking at bins with maximum temperatures (in Fahrenheit) from 65-70, 70-75, 75-80 and above 80. This division provides sufficient observations in each bin to estimate effects. For cold effects, we focus on bins in which minimum temperatures were between 30-35, 25-30, or below 25. Appendix Figure A.1 shows the distributions of the minimum and maximum weekly temperatures during our sample period.

[19] The endogeneity concern here is that as the city grows it may become both warmer (the heat island effect) and less healthy. If the city becomes less healthy in a way that interacts with warmer temperatures, such as by increasing the spread of infectious diseases, then this endogeneity has the potential to bias our results

[20] Temperature data are also available from Greenwich Observatory (before WWII) and Kew Gardens (after WWII). A comparison of the three data sources shows that the Oxford temperature data track variation in the data from Greenwich and Kew Gardens closely. In Appendix A.2 we verify that similar results are obtained if we instead use temperature from Greenwich and Kew Gardens in our analysis.

Table 1 provides statistics on the temperature bins included in our analysis for the full sample as well as several sub-periods. The sub-periods that we consider are naturally defined by the breaks in the data. The first covers all of the years before the onset of WWI in 1914. The second covers the interwar period, with data from 1918-1939, while the third period covers the years just after WWII, from 1949-1965. In the analysis we pool the data after WWI to increase the sample size and because we observe similar patterns in both the interwar and post-WWII periods.

Table 1: Temperature bins used in the analysis

Bin Range (F)	Number of weeks falling into bin by period:			
	All years	Pre-WWI 1866-1914	Interwar 1919-1939	Post-WWII 1949-1965
Min temp < 25	483	242	83	86
Min temp 25-30	811	383	154	99
Min temp 30-35	1139	494	224	157
Reference weeks	1020	407	174	180
Max temp 65-70	785	351	144	121
Max temp 70-75	707	293	139	114
Max temp 75-80	516	213	95	74
Max temp > 80	435	174	83	56
Total weeks	5896	2557	1096	887

Notes: Temperature data are from the Radcliffe Observatory, Oxford, U.K. Temperature observations are in degrees Fahrenheit.

4 Empirical Specification

Our main analysis uses a lead-lag model in which we estimate the non-parametric relationship between the occurrence of unusually high or low temperatures in a week on mortality in that week as well as several previous and subsequent weeks. Estimates of the mortality response in weeks before a high or low temperature event is observed provide a check on our identification strategy; we should expect these estimates to be very close to zero, since temperature today should not impact mortality in the past (controlling for past temperature). Our estimates of the effect of high or low temperature events in subsequent weeks allow us to understand whether relatively extreme temperature events have lagged mortality effects. To estimate the effect of temperature non-

parametrically, we classify weeks into the temperature bins shown in Table 1, with weeks where the high temperature was never above 65F and the low temperature never below 35F treated as the reference category.

Our baseline empirical specification is:

$$ln(y_{wt}) = \sum_{j=-m}^{k} \sum_{q=1,q\neq 4}^{8} \alpha_j^q TEMP_{wt}^q[w = j] + \delta_w + \delta_t + X_{wt}\beta + \varepsilon_{wt} \qquad (1)$$

where lny_{wt} is the log number of total or infant deaths (or such deaths due only to certain causes) in week w of year t in London. The $TEMP_{wt}^q$ terms are a set of indicator variables, taking on the value one if the weekly temperature is in the qth temperature bin, with q=4 corresponding to the reference bin. The estimated α_j^q's quantify the non-parametric relationship between temperature and mortality for each m (week) "leads" and each k (week) "lags". Note that the leading or lagged effects are estimated while also estimating the direct effect of contemporaneous temperature. Our specification includes controls for week-of-the-year fixed effects (δ_w), year fixed effects (δ_t), and a vector of week-by-year varying weather controls (X_{wt}) (precipitation and an indicator for weeks with heavy fog).[21]

To be clear, y_{wt} is the number of deaths, not a death rate. However, because we are using a log specification with year fixed effects, the coefficients we estimate are identical to what would be obtained if we instead replaced y_{wt} with a death rate calculated using population measured annually.

Because our data are structured as a time series, serial correlation is a potential concern. However, we find that allowing for serial correlation using Newey-West standard errors results in smaller confidence intervals, indicating negative serial correlation. This most likely reflects that when there are many deaths in one week there are fewer people at risk of dying in the next week. Thus, in the main results we present more conservative robust standard errors, though results obtained using Newey-West standard errors are provided in the appendix.

Our analysis approach, which relies on high-frequency variation in time-series data, is similar to the approach commonly used in the public health literature (reviewed by Deschenes, 2014) but differs from most existing studies in the economics literature, which instead use lower-frequency panel data, often at the monthly level. This difference comes with both advantages, such as an ability to examine the structure of lagged effects in more detail, as well as disadvantages, such as limitations

[21] We examine humidity controls in the appendix. Heavy fog affected mortality through pollution (Hanlon, 2018). The fog data are drawn from that paper.

on the variety of climate conditions observed. Given this, we view our approach as complementary to the panel data approach used in studies such as Barreca et al. (2016).

One final point to note regarding our estimation strategy is how our ability to control flexibly for time effects compares to studies using lower-frequency panel data such as Barreca et al. (2016) and Geruso and Spears (2018). Typically, panel studies have an advantage in that it is possible to more flexibly control for time-varying factors by including time-period fixed effects. In studies using panel data at monthly frequency, such as the two cited above, this takes the form of month-by-year effects. However, note that our data are sufficiently rich that we are also able to estimate results while including month-by-year effects, just as in those studies. These results, which can be found in Appendix Figures A.8 and A.15, show that the inclusion of these flexible time controls has very little impact on our results. This highlights the fact that we are able to control for time effects as flexibly as any existing panel data study reliant on monthly data.

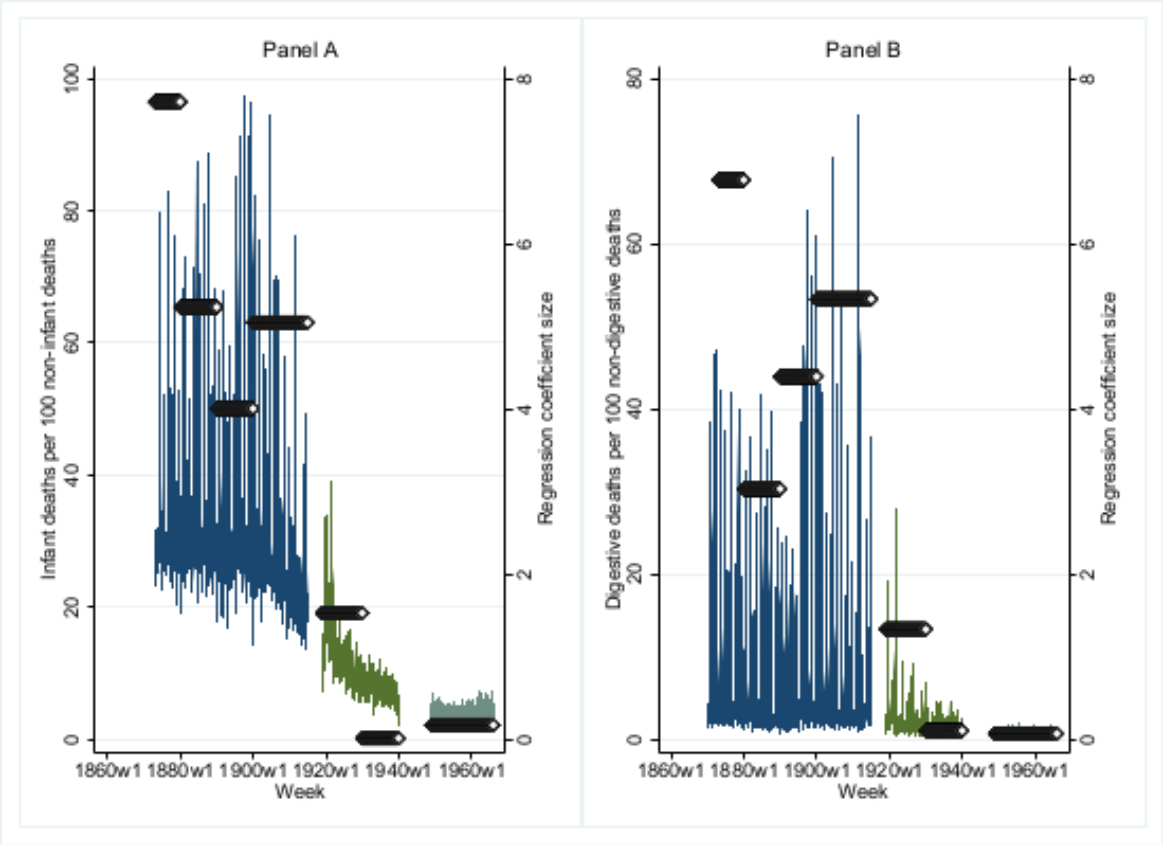
5 Results

Before presenting our main results, it is useful to preview two of the key patterns underlying our analysis. In Figure 1, we present raw data showing the change in infant deaths relative to non-infant deaths (Panel A) and digestive deaths relative to non-digestive deaths (Panel B), from 1874-1965. These data show a clear secular decline in the number of infant and digestive deaths over time, together with a reduction in the week-to-week variability in the number of deaths. The pattern for infants is not driven by secular trends related to the fertility transition; Appendix Figure A.2 shows similar results for the infant mortality rate, i.e., infant deaths divided by the number of live births in the 52 preceding weeks. In this paper, we argue that the peaks in infant and digestive mortality prior to WWI are to some extent driven by weeks with high temperatures and, as infant and digestive mortality fell, this effect was substantially reduced.

This figure also presents coefficients (thick black horizontal lines) from preliminary regressions in which we estimate the impact of weeks with temperature falling into the highest temperature bin on infant or digestive mortality in a week and the seven following weeks. These coefficients provide a summary of warm-week effects, which we will explore in more detail later. We estimate coefficients separately for each decade in order to get an initial sense of how the effect of

temperature was changing across the study period. The resulting coefficients show that the impact of high temperatures on mortality had declined substantially by the inter-war period. Motivated by these patterns, in our main analysis, which we turn to next, we partition our data into an early period (pre-WWI sample) and a late period (post-WWI sample).[22]

Figure 1: Infant deaths per 100 non-infant deaths, digestive deaths per 100 non-digestive deaths, and warm-week effects by periods



Notes: On the first y-axis in Panel A, the thin line plots infant deaths relative to total non-infant deaths (x100) over time. In Panel B, on the left-hand y-axis, the thin line plots digestive deaths relative to total non-digestive deaths (x100) over time. On the second y-axis (Panels A and B), the horizontal thick grey lines show the estimated effect of warm weeks on total mortality in that week and the next seven weeks for different periods (1874-1879, 1880-1889, 1890-1899, 1900-1914, 1919-1929, 1930-1939, and 1949-1965). These regressions include fixed effects for week-of-the year and year, as well as weekly controls for rainfall and fog events. Appendix Figure A.2 reports the corresponding results for the infant mortality rate.

[22] The period from 1900 to 1914 could be thought of as a transition period from the high infant mortality regime of the late 19th century to the low infant mortality regime that existed after WWI (see also Appendix Figures A.3-A.5). We have chosen to include this in our pre-WWI sample, though in Appendix Figures A.10 (for total mortality) and A.17 (for infant mortality) we verify that our results are not substantially affected if we instead end the early period in 1900.

Next, we turn to our main estimates of the temperature-mortality relationship for both total and infant deaths. We consider seven lags because for most series the effects of temperature on mortality dies out within seven weeks. This also roughly corresponds to the two-month exposure period considered in Barreca et al. (2016), which they argue is sufficiently long to address "harvesting" concerns, i.e., the death of individuals who were already near death. We include five leading weeks, a somewhat arbitrary choice but one that seems sufficient to establish a lack of pre-trends.

Figure 2 presents our first set of main results. Specifically, this figure presents estimated coefficients and confidence intervals for the impact of temperature on total mortality, where the black lines are estimates from the pre-WWI sample and the grey lines are estimates from after WWI. Each panel represents estimates for one leading, lagged, or contemporaneous observation, while within each panel temperature bins are represented on the x-axis. Thus, the top-left panel represents the impact of a temperature event on mortality in the week before the temperature event occurred, the top-right panel represents the effect in the week in which an event occurred, the middle-left panel represents the effect in the following week, etc.

Note first that for both the pre-WWI and post-WWI periods, the leading effects in the top-left panel are small and insignificant, so temperature in a week is not systematically related to mortality in previous weeks (see Appendix Figure A.6 for additional leads and lags). This indicates that our identification strategy is working well.

Focusing on the pre-WWI sample (i.e., the black lines), we observe a clear increase in total mortality due to cold, with deaths peaking with a one-week lag. The coefficient indicates that weeks when temperature fell into the coldest bin were followed the next week by an increase in mortality of 0.15 log points relative to weeks in the reference bin. The cold-week effect persists for 4-5 weeks.[23] If we average this cold week effect across the week in which the cold temperature is observed as well as the subsequent seven weeks, we estimate an average mortality increase of 7 percent across that eight-week period (see Appendix A.6). There is also some indication that cold

[23] It is worth keeping in mind that the impact of cold weeks may be due in part to pollution effects, since coal used for heating was an important source of pollution throughout our study period.

weather was more deadly in the later period. However, this pattern should be interpreted with caution, since it may be due to changes in the underlying age composition of the city.

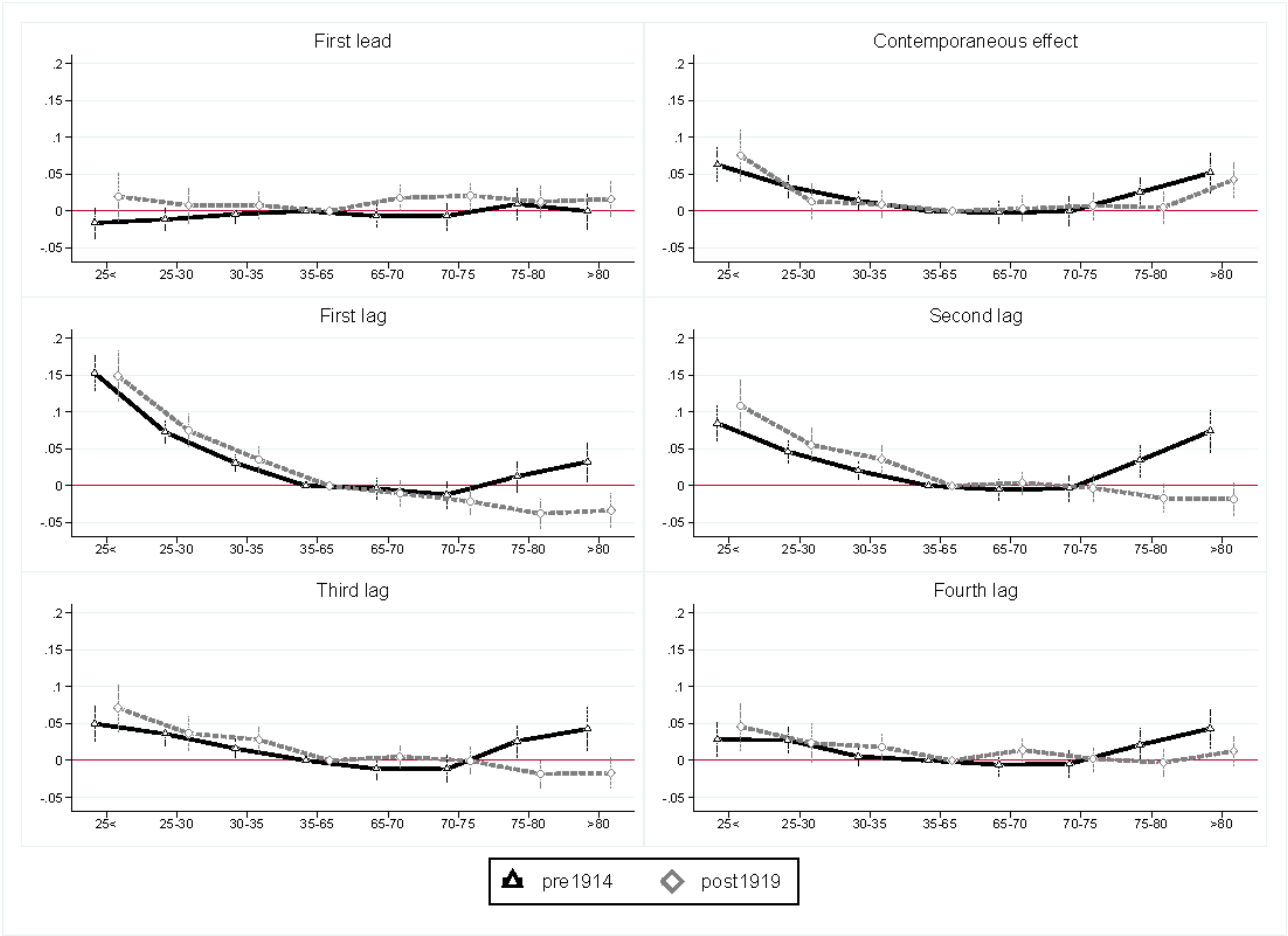
There is also evidence of an increase in mortality associated with warm weeks. For weeks falling into the hottest temperature bin, two weeks after the temperature is observed we estimate a coefficient of 0.07 (t-value = 5.15), implying an increase in total mortality of around 7 percent. Averaged across the eight-week period, the effect of temperature falling into the warmest bin was a 4 percent increase in total mortality (see Appendix A.6). Interestingly, this "warm-week" effect persists for longer than the cold-week effect. Later, we will provide evidence that this lag structure is most likely due to the fact that warm weather facilitates the spread of diseases which continue to spread and increase mortality for several weeks. This persistence suggests that either the effects we observe are not due to short-run harvesting or that any harvesting effects are more than offset by the lagged direct impacts of high temperatures. Overall, the dark lines in Figure 2 reveal that in pre-WWI London total mortality was elevated both during cold and warm weeks, though the cold-week effect is stronger in magnitude while the warm-week effect is relatively more "persistent".

The grey lines in Figure 2 show the relationship between temperature and mortality after 1919. Here we find a similar pattern for the cold-week effect as in the pre-WWI sample. The magnitude of the effect of cold weeks is almost exactly the same, a 7 percent increase in mortality averaged across the week in which cold weather occurred and the subsequent seven weeks (see Appendix A.6). However, the warm-week effect looks strikingly different. Total mortality is only elevated in warm weeks during the temperature change (coefficient of 0.042 and t-value = 3.42), and in the following weeks total mortality is unusually low, possibly indicating short-term harvesting effects. Averaged across an eight-week period, the total effect is essentially zero (see Appendix A.6). Therefore, starting in 1919, the warm-week effect is only present during the temperature change and subsequently absent: the effect of high temperature on mortality has essentially disappeared. While the data examined in Figure 2 end in 1965, Appendix Figure A.11 shows that results for 1981-2006 are similar to the post-WWI results shown in Figure 2.

The results in Figure 2 show that the effect of warm weeks can be divided into two distinct components. The first component is the small increase in mortality, of 4-5%, in the week in which warm weather occurs. This increase appears in all time periods, though it diminishes in magnitude over time. Further analysis shows that this contemporaneous effect is spread broadly across different

causes of death and age categories. The second component, which accounts for most of the mortality effect prior to WWI, is the sustained elevated mortality level in weeks following a warm week. As we discuss below, this persistent mortality effect shows a clear association with specific age groups and causes of death.

Figure 2: Temperature and total mortality before and after WWI



Notes: This figure shows the non-parametric relationship between temperature and log total mortality for the 1st lead, the current effect, and four lags. Results for four additional leads and three additional lags are reported in Figure A.6. The pre-WWI sample is from 1866-1914 and the post-WWI sample is from 1919-1939 and 1949-1965. The omitted reference weeks have minimum temperature above 35F and maximum temperature below 65F.

We have undertaken a number of robustness checks on these results. These checks, reported in Appendix A.2.2, show that our main findings are robust to, for example, including rich non-parametric controls for humidity, controlling for month fixed effects, and accounting for serial

correlation using Newey-West standard errors. One may also wonder whether the changes over time shown in Figure 2 may be due in part to shifts in the age distribution of the population of London. The best way to examine this issue is to break down results by age group, as we do in the next step of our analysis.

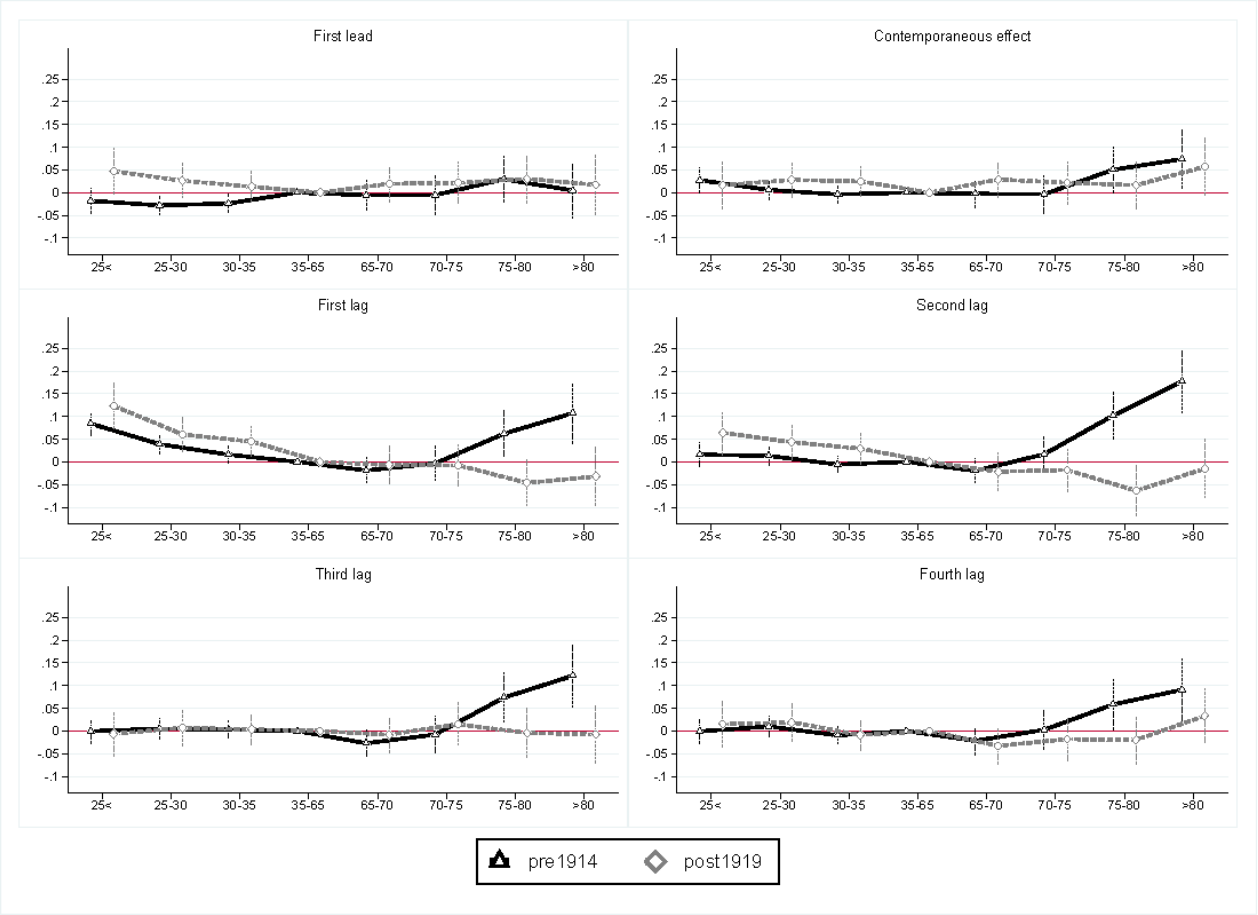
We now consider the role that infant deaths play in generating the total-mortality results. Our infant results are in Figure 3, which is structured similarly to Figure 2 but with log infant deaths as the outcome. Note that, as in the total mortality results, we see no systematic evidence of pre-trends (see additional leads in Appendix Figure A.14).

The most striking feature of the infant results is the large effect of warm weather on mortality in the years before WWI. The estimated effect of falling into the warmest temperature bin begins contemporaneous to the temperature observation and peaks two weeks later with an estimated coefficient of 0.18 (t-value = 4.95), implying an increase in infant mortality of around 18 percent relative to weeks with moderate temperatures. This effect persists for 6-7 weeks. Averaged across the week falling into the warmest temperature bin and the subsequent seven weeks, we estimate a 10 percent increase in infant mortality (see Appendix A.5). However, after 1919 this effect has essentially disappeared. Clearly an important change took place that substantially reduced the impact of warm weeks on infant mortality.

For the coldest group of weeks, the peak infant mortality effect for the period before WWI, which occurs with a one-week lag, has a coefficient of 0.08 (t-value = 6.21) and two weeks after the effect of cold weather largely disappears. Thus, compared to total mortality, the cold-week effect on infants is significantly smaller and less persistent. We see little evidence of this effect changing over time though, if anything, it appears to be strengthening. Remember, however, that these results reflect the impact of cold weather relative to weeks with moderate temperature, so the apparent strengthening of the cold weather effect may simply reflect a reduction of infant deaths in other weeks, rather than a stronger causal impact of cold weather on infant mortality.

Comparing the results for infants shown in Figure 3 to estimates obtained from all other age groups in Figure 4 reveals that the increase in overall mortality in warm weeks in the period before WWI is driven almost entirely by infant deaths. For all other ages, we see only a modest contemporaneous effect of high temperatures and no evidence of lagged effects. Instead, for older age groups the impact of temperature on mortality is driven almost entirely by unusually cold rather than unusually warm weather.

Figure 3: Temperature and infant mortality before and after WWI

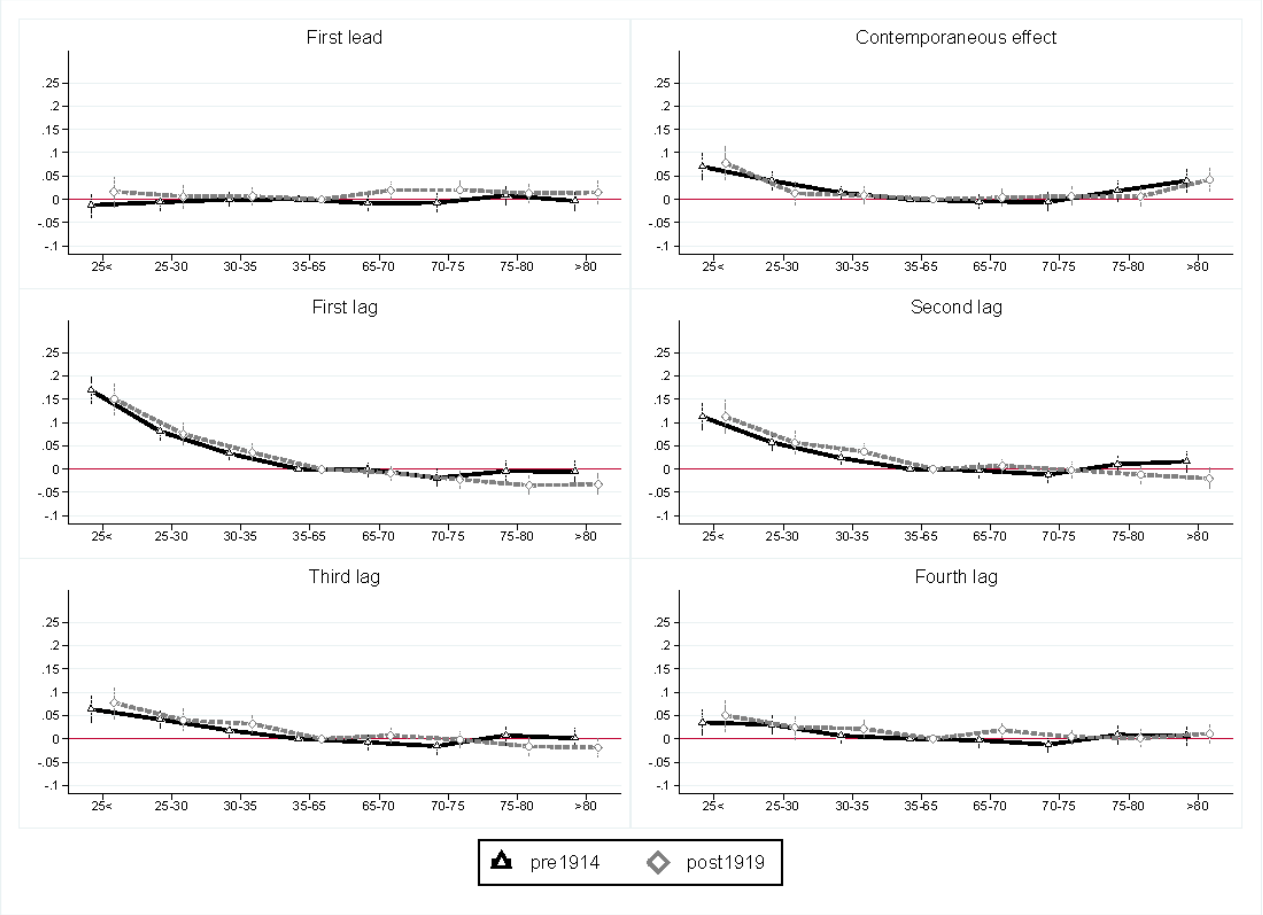


Notes: This figure shows the non-parametric relationship between temperature and log infant mortality for the 1st lead, the current effect, and four lags. Results for four additional leads and three additional lags are reported in Figure A.14. The pre-WWI sample is from 1874-1914 and the post-WWI sample is from 1919-1939 and 1949-1965. The omitted reference weeks have minimum temperature above 35F and maximum temperature below 65F.

As we did for total mortality, we have also examined the robustness of the infant mortality results. These robustness checks, in Appendix A.2.3, show that our results are robust to including rich non-parametric humidity controls, using the infant mortality rate as the dependent variable, etc. In addition, these results naturally raise questions about the relationship between temperature and births. Appendix Figure A.21 shows that, both before and after WWI, both high and low temperatures were associated with a substantial contemporaneous reduction in births, possibly partially offset by

increases in births in the following weeks. These findings tell us that changes in birth rates are not behind our infant mortality results.

Figure 4: Total non-infant deaths before and after WWI



Notes: This figure shows the non-parametric relationship between temperature and log (total - infant) mortality for the 1. lead, the current effect, and four lags. Results for four additional leads and three additional lags were estimated but are not reported. These are available upon request. The pre-WWI sample is from 1874 to 1914 and the post-WWI sample is from 1919-1939 and 1949 to 1965. The omitted reference temperature bin is $q = 4$ or $35F - 65F$.

Together, the patterns documented in Figures 2 and 3 are striking: The effect of cold weeks on total and infant mortality has been relatively stable throughout our historical sample period of around 100 years. However, high-temperature weeks were mainly related to excess total mortality–and in particular infant mortality–in the early period of the sample.

One thing that the results in Figures 2 and 3 do not reveal is whether hot or cold weeks are more deadly if they occur consecutively. We explore this possibility in Appendix A.5. The results in that

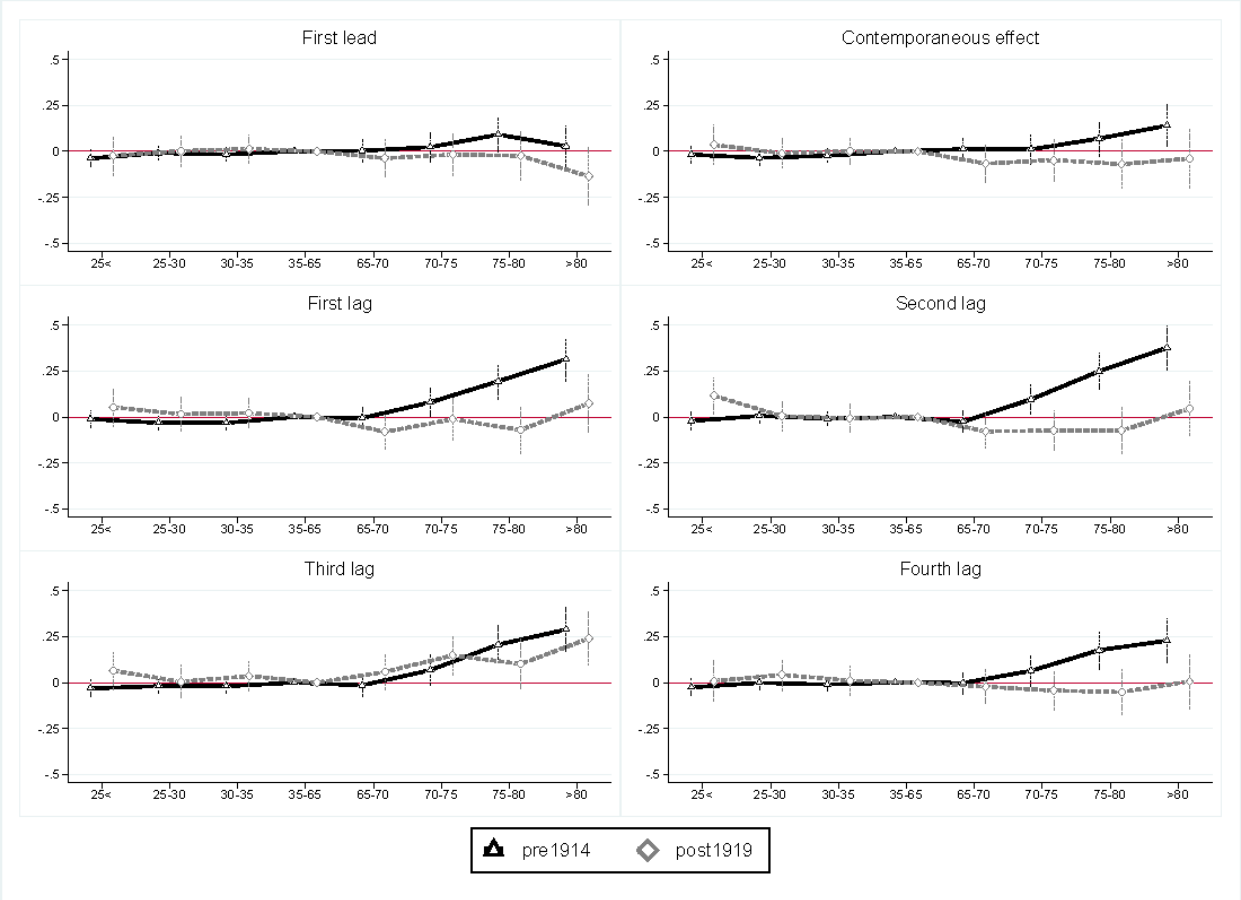
section show that for both total and infant mortality, warm weeks are more deadly when they occur consecutively, particularly when it warm for three or four weeks in a row. However, this is only true in the period before 1914. We find no evidence that consecutive warm weeks are more deadly in the later part of our sample. Interestingly, no similar pattern exists for cold weeks.

Next, we study the impact of temperature on mortality within particular cause-of-death categories. Since infants appear to be the key to the changing relationship between high temperatures and mortality, it is natural to begin our analysis by focusing on digestive diseases, the main cause-of-death for this group.

Figures 5 and A.22 plot the impact of temperature on mortality due to digestive diseases. As in the previous figures, the dark lines show estimates from the pre-WWI sample while the grey lines are estimates from the post-WWI sample. The contrast between them is clear. While the years before WWI saw large increases in digestive deaths associated with warm temperatures, this pattern essentially disappears after 1920.

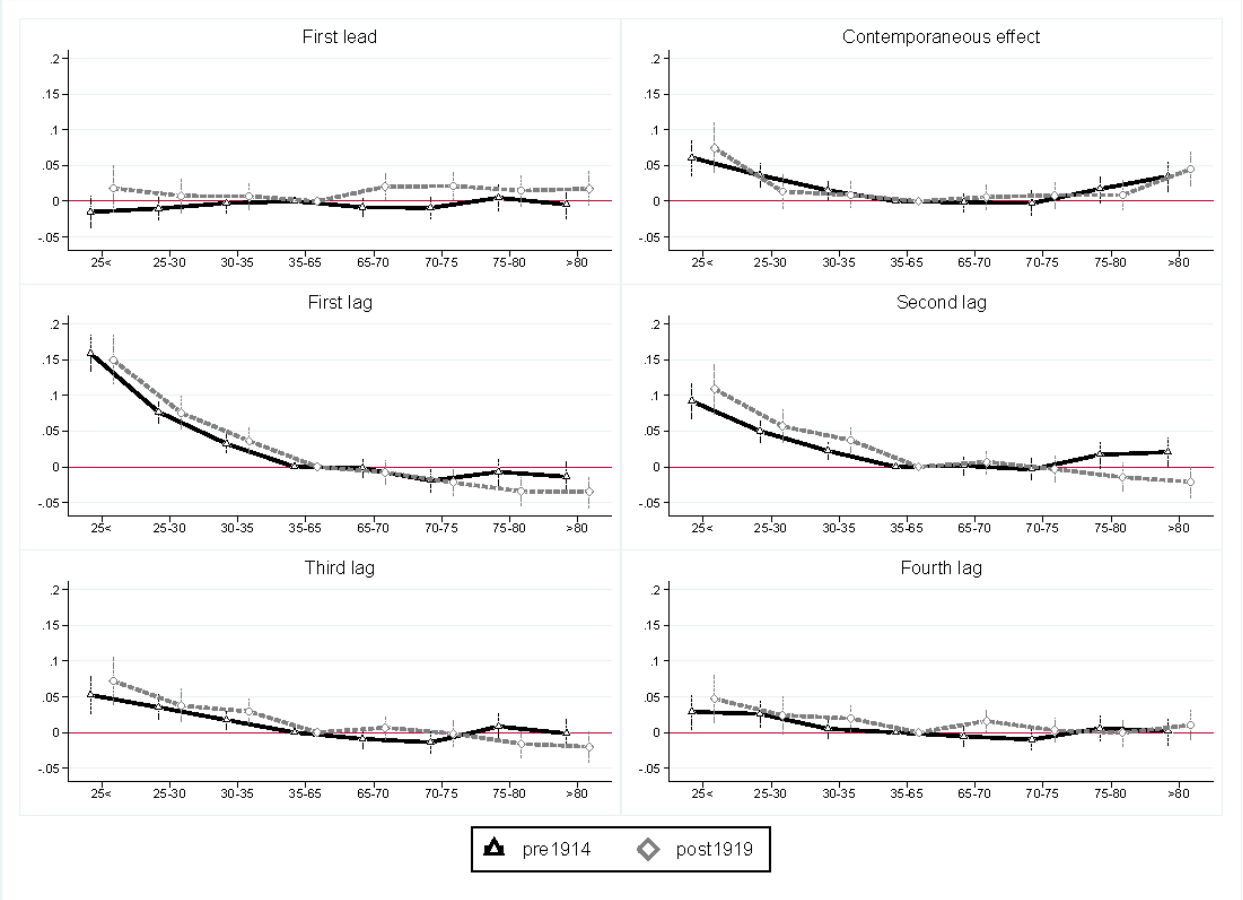
An analysis of all causes of death other than digestive diseases, in Figure 6, makes it clear that the elevated mortality observed in warm weeks in the pre-WWI period is driven largely by the digestive disease category. For all other diseases, we do not observe any persistently high level of mortality in the weeks following a warm-weather week, even in the pre-WWI period. This tells us that, with the exception of the small contemporaneous effect, the persistent impact of warm weather on mortality before WWI is due entirely to digestive disease deaths. Thus, the change in the impact of warm weeks on mortality after WWI was due mainly to the elimination of digestive diseases among infants.

Figure 5: Temperature and digestive mortality before and after WWI



Notes: This figure shows the non-parametric relationship between temperature and log digestive deaths for the 1st lead, the current effect, and four lags. Results for four additional leads and three additional lags are reported in Figure A.22. The pre-WWI sample is from 1870-1914 and the post-WWI sample is from 1919-1939 and 1949-1965. The omitted reference weeks have minimum temperature above 35F and maximum temperature below 65F.

Figure 6: Temperature and total non-digestive mortality before and after WWI



Notes: This figure shows the non-parametric relationship between temperature and log non-digestive mortality for the 1. lead, the current effect and four lags. Results for four additional leads and three additional lags were estimated but are not reported. Those are available upon request. The pre-WWI sample is from 1870 to 1914 and the post-WWI sample is from 1919-1939 and 1949-1965. The omitted reference temperature bin is $q = 4$ or $35F - 65F$.

Finally, we break our data down to look at the impact of temperature across a variety of more detailed cause-of-death categories. This analysis uses data from 1866-1914. To simplify the analysis, we estimate the effect of our highest and lowest temperature bins on mortality in a week and the seven following weeks by cause of death. This is essentially the average effect across the contemporaneous and first seven lagged coefficients for temperatures falling into the highest bin. The results are presented in Table 2. These results show that low temperatures increase mortality due to a number of causes of death, including bronchitis, cardiovascular diseases, measles, "old age", pneumonia, other respiratory diseases (which includes influenza), and tuberculosis. These results reflect the well-known association between respiratory and cardiovascular diseases and low

temperatures. At the same time, low temperatures are associated with reduced deaths due to scarlet fever and typhus.

Table 2: Effect of high temperature weeks on different causes of death before WWI

	DV: Log deaths					
Cause:	Digestive	Bronchitis	Cancer	Cardio	Childbirth	Diphtheria
Low temp.	-0.0271	0.198***	0.00297	0.0720***	0.0491	0.0250
	(0.0193)	(0.0186)	(0.0102)	(0.00971)	(0.0316)	(0.0249)
High temp.	0.257***	-0.0597***	0.0142	0.00627	-0.0309	0.0307
	(0.0327)	(0.0152)	(0.0109)	(0.00910)	(0.0315)	(0.0264)
Observations	2,328	2,328	2,328	2,328	2,326	2,327
R-squared	0.817	0.884	0.882	0.814	0.297	0.812
Cause:	Homicide	Measles	Old Age	Pneumonia	Respiratory	Scar. Fev.
Low temp.	0.0135	0.129***	0.0930***	0.129***	0.164***	-0.0710**
	(0.0458)	(0.0491)	(0.0137)	(0.0153)	(0.0211)	(0.0297)
High temp.	-0.00818	-0.0668*	0.0205	-0.0393***	-0.0346	-0.0548**
	(0.0442)	(0.0382)	(0.0150)	(0.0135)	(0.0219)	(0.0279)
Observations	1,640	2,328	2,328	2,328	2,328	2,316
R-squared	0.101	0.410	0.533	0.803	0.720	0.824
Cause:	Suicide	TB	Typhus	Whoop. Cgh.		
Low temp.	0.0380	0.0393***	-0.160***	0.0166		
	(0.0329)	(0.00805)	(0.0618)	(0.0299)		
High temp.	-0.0317	0.0284***	0.0628	-0.0299		
	(0.0329)	(0.00768)	(0.0689)	(0.0256)		
Observations	2,322	2,328	781	2,328		
R-squared	0.311	0.662	0.648	0.700		

*** p<0.01, ** p<0.05, * p<0.1 Robust standard errors in parentheses. Data cover 1866-1914. Estimates reflect the impact of a week with temperature falling into the highest bin (> 80 degrees F) in the in which the temperature occurred or the seven following weeks.

As expected, high temperatures cause a substantial increase in deaths associated with digestive diseases, as well as a more modest increase in deaths due to tuberculosis. We do not see effects of high temperatures in other categories, and a number of diseases, including bronchitis, measles and pneumonia, exhibit reduced deaths when temperatures are high. Not surprisingly, these are diseases where we see more deaths in cold conditions. It is interesting to note that scarlet fever deaths are reduced at high and low temperatures. This may reflect a competing risk story, where those who might have died of scarlet fever instead succumb to diseases like diarrhea during warm weather or measles during cold weather. Overall, the high-temperature estimates provide clear

evidence of the central role that digestive diseases play in explaining the association between high temperatures and elevated mortality before WWI.

The results presented above have implications for the impact of rising temperatures on mortality in London. In Table 3, we conduct some simple counterfactual exercises in order to assess the magnitude of these effects. Panel A of Table 3 describes our estimates of the actual number of excess deaths due to relatively hot and relatively cold weeks in different periods. These are based on the estimated coefficients obtained from applying our regression approach to the data from each period. Heat effects reflect the number of deaths associated with maximum temperatures above 80F, while cold effects are those associated with minimum temperatures below 35F, the bins for which we observe statistically significant effects before WWI. Panel A shows that we estimate that warm weeks, those falling into our top temperature bin, are associated with 58,129 excess deaths in 1876-1914, or 1.9% of all deaths. However, after WWI, we observe few deaths in weeks when temperatures are in the top bin, and by the post-WWII period weeks in the top bin were on average relatively healthy. This shift reflects the patterns described in the previous section. Unlike warm weeks, cold weeks remain associated with substantial numbers of excess deaths throughout the study period. Overall, temperature-related deaths were a major component of mortality, accounting for 10.3% of all deaths in the pre-WWI period, 13.9% in the interwar period (when influenza deaths spiked), and 7.5% of all deaths after WWII.

Next, we ask: what would mortality have looked like if the temperature-mortality relationship had not improved after WWI? To answer this, we calculate mortality in the interwar and post-WWII periods while imposing, for each age group, the temperature-mortality relationship estimated on pre-WWI data. We do this by age group in order to account for changes in the age composition of the population. These estimates are shown in Panel B of Table 3. It is important to note that this delivers conservative counterfactual estimates, since our estimates incorporate the baseline reduction in overall mortality observed during each period. I.e., we are not holding overall mortality rates at the pre-WWI period. Instead, we are simply applying the estimated percentage increase in mortality associated with warm weeks, relative to weeks with moderate temperature, from the pre-WWI period to the baseline mortality rates observed in the interwar and post-WWII periods. Since we are allowing baseline mortality rates to change, this counterfactual incorporates broad health improvements that occurred across these periods.

The estimates in Panel B suggest that, had the pre-WWI temperature-mortality relationship persisted into the later periods, there would have been an additional 9,018 heat-related deaths in the interwar period and 8,903 deaths in the decades after WWII, equal to a 0.8- 1.3% increase in overall mortality. This provides a direct estimate of how many heat-related deaths were averted as a result of the changes in the temperature-mortality relationship that took place in the early 20th-century.

Table 3 Estimated and counterfactual effects of temperature on mortality by period

Period:	**Before WWI**	**Interwar**	**Post WWII**
Actual deaths	3,075,021	1,102,963	671,621
Panel A: Estimated actual deaths due to temperature by period			
Heat-related	58,129	2,822	-4,207
(share of all deaths)	0.019	0.003	-0.006
Cold-related	257,741	150,514	54,269
(share of all deaths)	0.084	0.136	0.081
Total temp-related deaths	315,869	153,336	50,062
(share of all deaths)	0.103	0.139	0.075
Panel B: Estimated heat-related deaths imposing the pre-WWI temp-mortality relationship			
Heat-related		11,840	4,695
Diff vs. panel A		9,018	8,903
(diff. as share of all deaths)		0.008	0.013
Implied additional deaths per year		432	555
Panel C: Impact of 1.5C increase using temp-mort relationship observed in each period			
Heat-related deaths	32,061	1,057	-1,701
Cold-related deaths	-80,554	-47,913	-14,688

Finally, we provide an assessment of the impact that rising temperature would have had under the different mortality regimes we have observed. In Panel C of Table 3 we present counterfactual mortality given the mortality patterns observed in each period, but imposing an increase in average temperatures of 1.5 degrees Celsius, a common reference level used in the climate change

literature.[24] These results show that had this temperature increase occurred in the pre-WWI period, we would have expected an additional 32,061 heat-related deaths, equal to a 1% increase in total mortality. This would have been more than offset by a reduction of 80,554 cold-related deaths. However, in the later periods, the increase in heat-related deaths resulting from this temperature increase was quite small, or even negative. This provides a direct illustration of how much the impact of a rise in temperature can vary across environments with different underlying disease burdens.

Another benefit of being able to generate results using counterfactual temperature patterns is that we can examine the impact of temperature on the timing of the mortality transition. A number of papers, such as Cutler and Miller (2005) and Anderson et al. (2020a) compare the timing of public health interventions to annual mortality data, to see if they correspond to the timing of infant mortality declines. In the context of London, however, it has been argued that the timing of the decline in infant mortality may have been substantially affected by several hot summers that occurred in the 1890s (Woods, et al., 1988; Woods 2000, p. 296). Our data allow us to assess this argument.

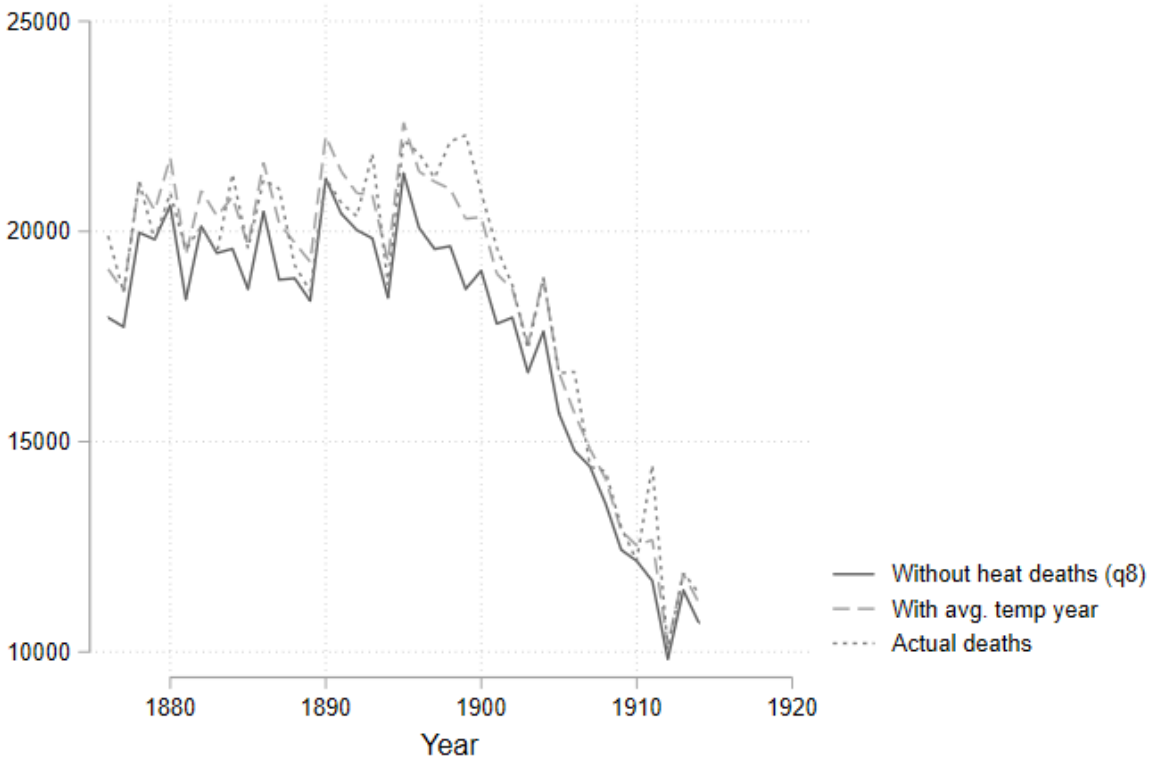
To do so, we use our estimates of the impact of temperature on mortality to generate two counterfactual infant mortality series. In the first, we completely remove the (contemporaneous and lagged) impact of weeks with temperatures falling into the top bin. In the second, we replace the pattern of high-temperature weeks in all years with that observed in an average high-temperature year.

These counterfactuals are compared to the actual pattern of infant deaths in Figure 7. The main take-away from this figure is that under either counterfactual scenario, the decline in infant mortality in London begins several years earlier than the true infant mortality decline. Whereas actual infant mortality in London peaked in 1899, under the counterfactuals the decline begins after 1895. The difference is due to the unusually high number of warm weeks that occurred in the late 1890s. This result confirms the suspicions of demographers such as Woods (2000). It also tells us that we have to be very careful to control for the impact of temperature in studies that compare the timing of an intervention, such as water filtration or chlorination, to annual mortality patterns, since temperature variation can alter the timing of mortality declines by several years.

[24] This change is implemented in the simplest possible way, by shifting the observed temperature distribution up by 1.5 degrees C. Of course, the variance of the temperature distribution is also likely to be impacted by climate change, with additional implications for mortality.

Figure 7 also provides a sense of the extent to which temperature variation influenced mortality in different years. Specifically, the vertical distance between the "Actual deaths" line and the "Without heat deaths (q8)" line describes the number of deaths in a year due just to the effect of temperatures falling into the top temperature bin. It is clear that this varies substantially over time, with hot weeks making a particularly large contribution to total deaths in the late 1890s.

Figure 7: Actual and counterfactual infant deaths



6 Discussion

This study presents uniquely detailed long-run mortality data and shows how they can be used to enrich our understanding of the relationship between temperature and mortality in the past, and how this relationship evolved over time. One striking feature of our results is the extent and timing of the change in the impact of high temperatures on mortality in the period after WWI, relative to the nineteenth century, and the contrast between this change and the stability of the mortality impact of cold weather. While these changes are not surprising, the richness of our data allow us to provide

quantitative estimates of how this change affected mortality in London. Our results suggest that deaths would have been higher by 0.8-1.3 percent in the 1918-1965 period if the temperature mortality relationship had resembled the one we observe in the decades before 1914.

Our results also offer lessons that may be useful for assessing the impact of climate change in modern developing countries. A recent study, Geruso and Spears (2018), shows that high temperatures have much larger mortality effects in developing countries than in the developed world. Our results help shed light on the mechanisms behind these findings, by highlighting, quantitatively, how differences in the disease environment alter the relationship between temperature and mortality. One implication of our results is that interventions that alter the disease environment have the potential to play an important role in helping less developed countries adapt to rising temperatures.

Finally, our results illustrate the importance of accounting for temperature effects when studying the mortality transition. In particular, our counterfactual estimates show that the infant mortality decline in London would have occurred several years earlier had it not been for a series of hot summers in the 1890s. This confirms, quantitatively, the suspicion of previous work, and highlights how important accounting for temperature effects can be to identifying the mechanisms behind mortality transitions.

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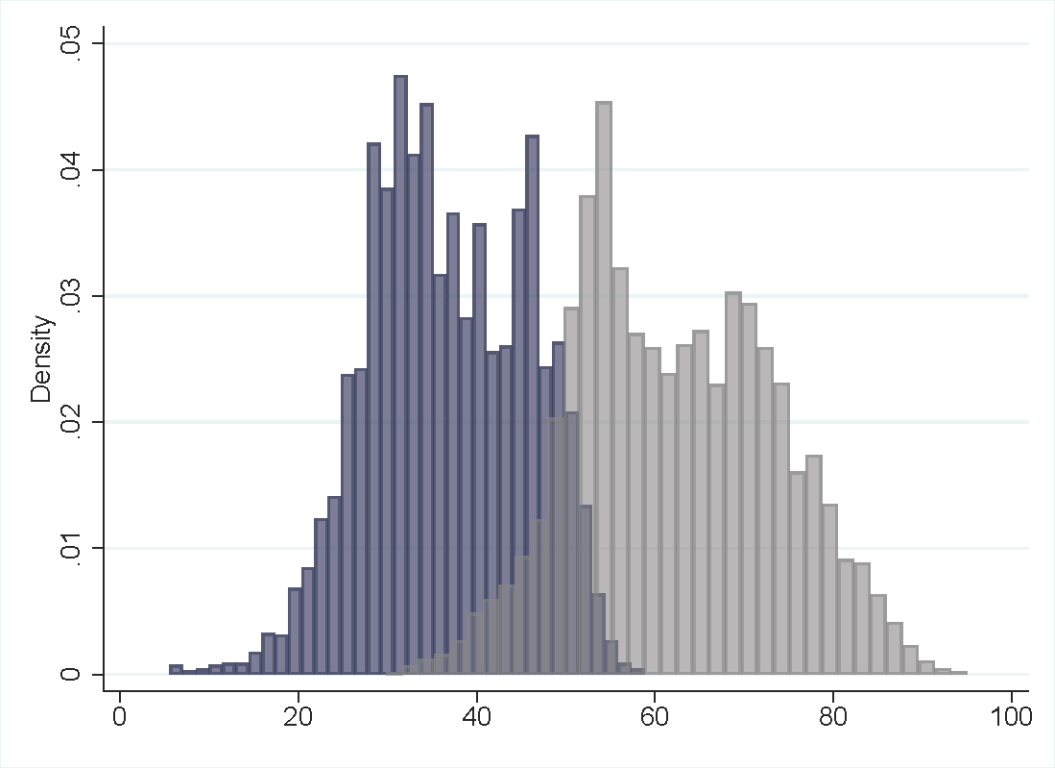
A Appendix – For Online Publication Only

A.1 Additional data description

Figure A.1: Distributions of weekly minimum and maximum temperatures

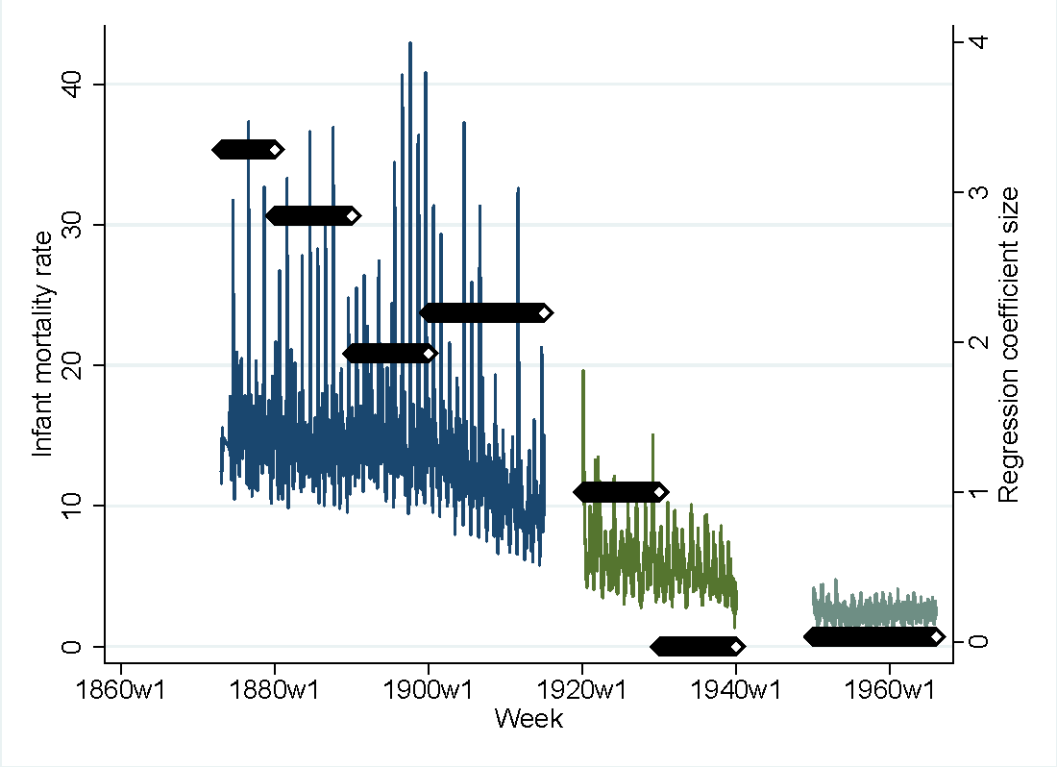


Notes: This figure shows the distributions of weekly and maximum temperatures during our sample period.

A.2 Supplement to the main analysis results

Figure A.2 shows the development of the infant mortality *rate* and how this variable was influenced by warm weeks by different periods. This supports the evidence presented in Figure 1 by showing that our results are not driven by any scale effects coming from fertility declines.

Figure A.2: Infant mortality rate and warm-week effect by period

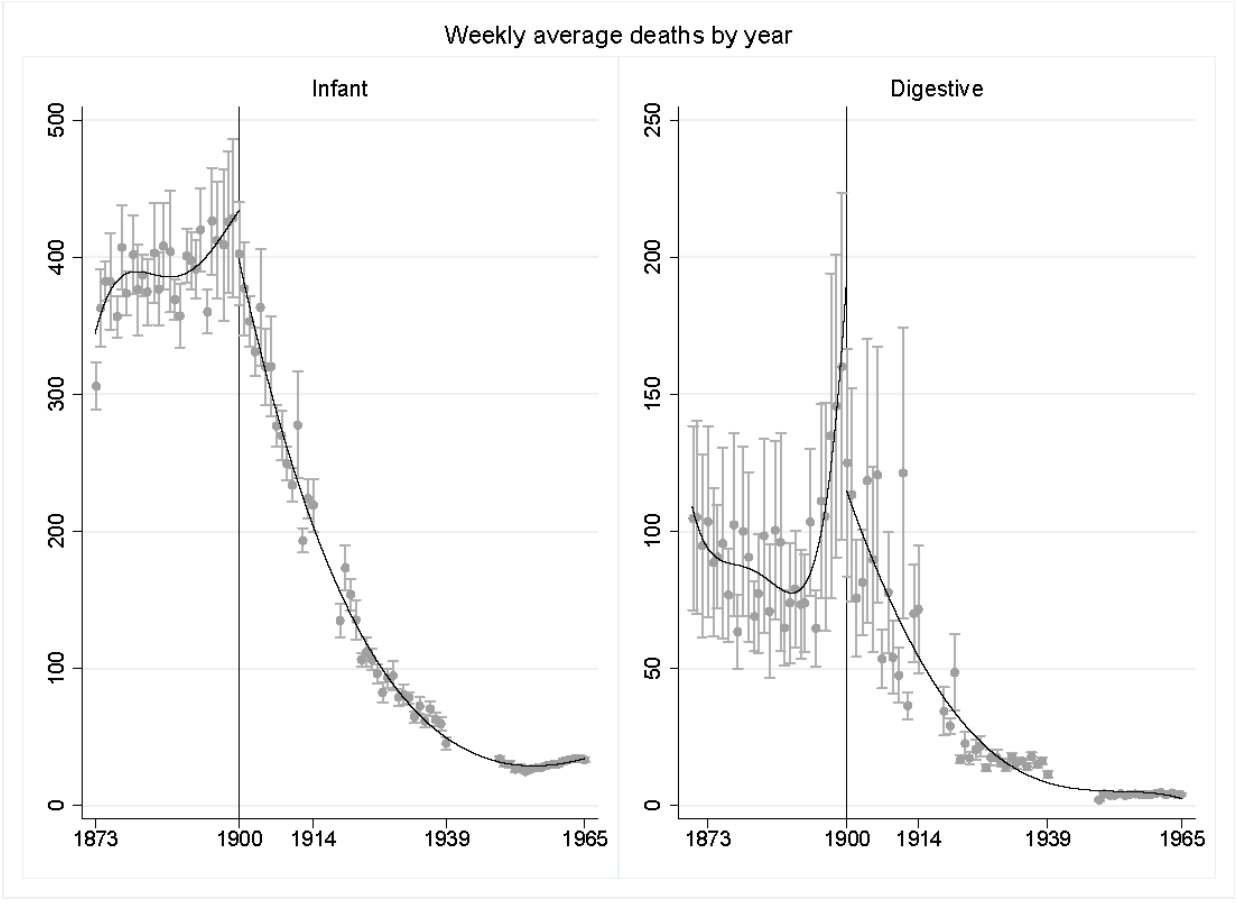


Notes: This figure shows the development of the infant mortality rate, which is calculated as the number of infant deaths during a particular week and scaled with the average number of births over the past 52 weeks (x100). The thick horizontal grey lines show warm-week estimates for different periods (1874-1879, 1880-1889, 1890-1899, 1900-1914, 1919-1929, 1930-1939, and 1949-1965). The regressions use the average of the contemporary effect and seven lags and control for week-the-year fixed effects, year fixed effects, weekly rainfall, and weekly fog events.

A.2.1 Timing of the decline in infant digestive disease deaths

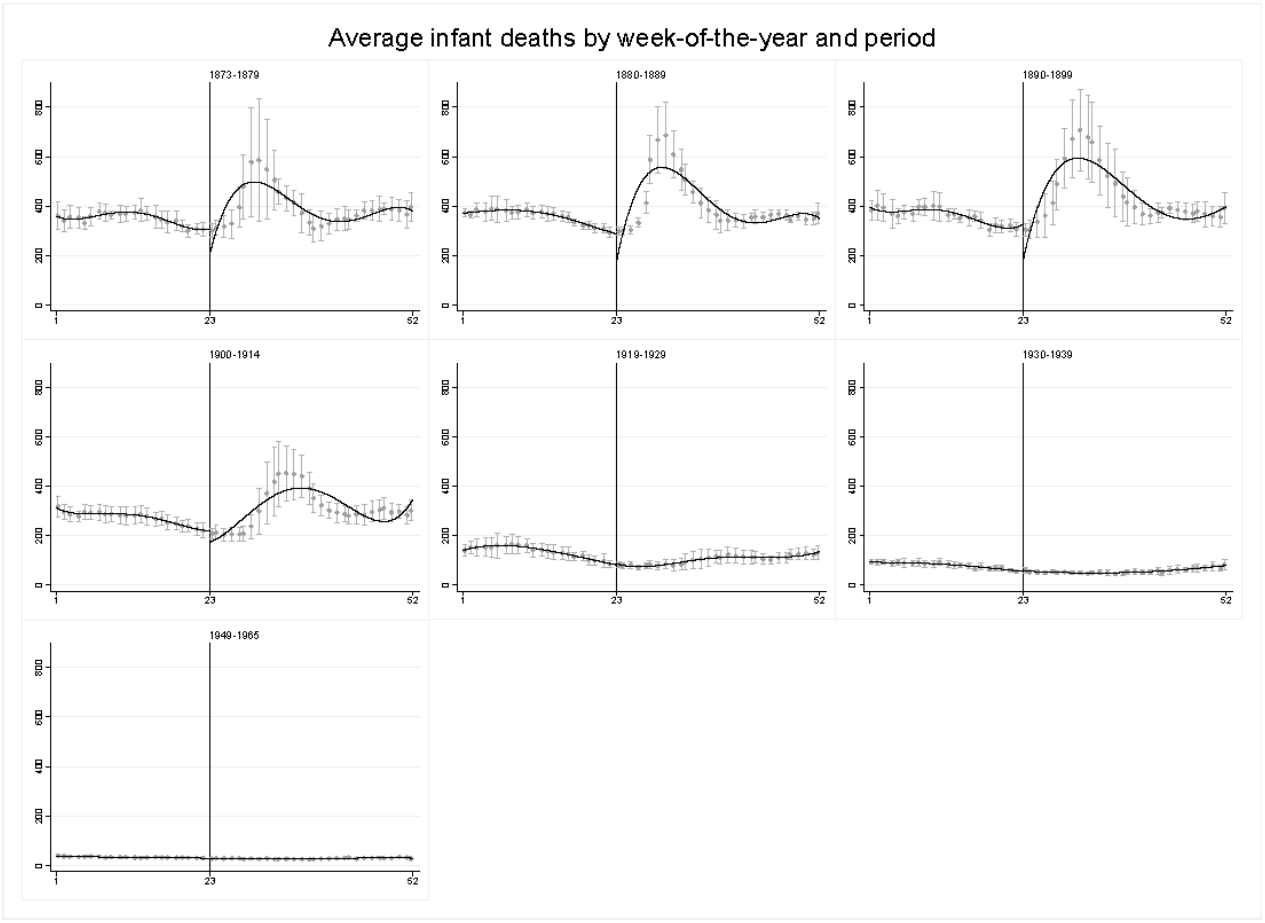
Here we present some additional evidence on the timing of the decline in infant digestive deaths. First, Figure A.3 presents results plotting out the pattern of infant and digestive disease deaths by year. Second, Figures A.4-A.5 describes the changing distribution of infant and digestive mortality across weeks of the year by different periods.

Figure A.3: Timing of infant and digestive disease mortality declines



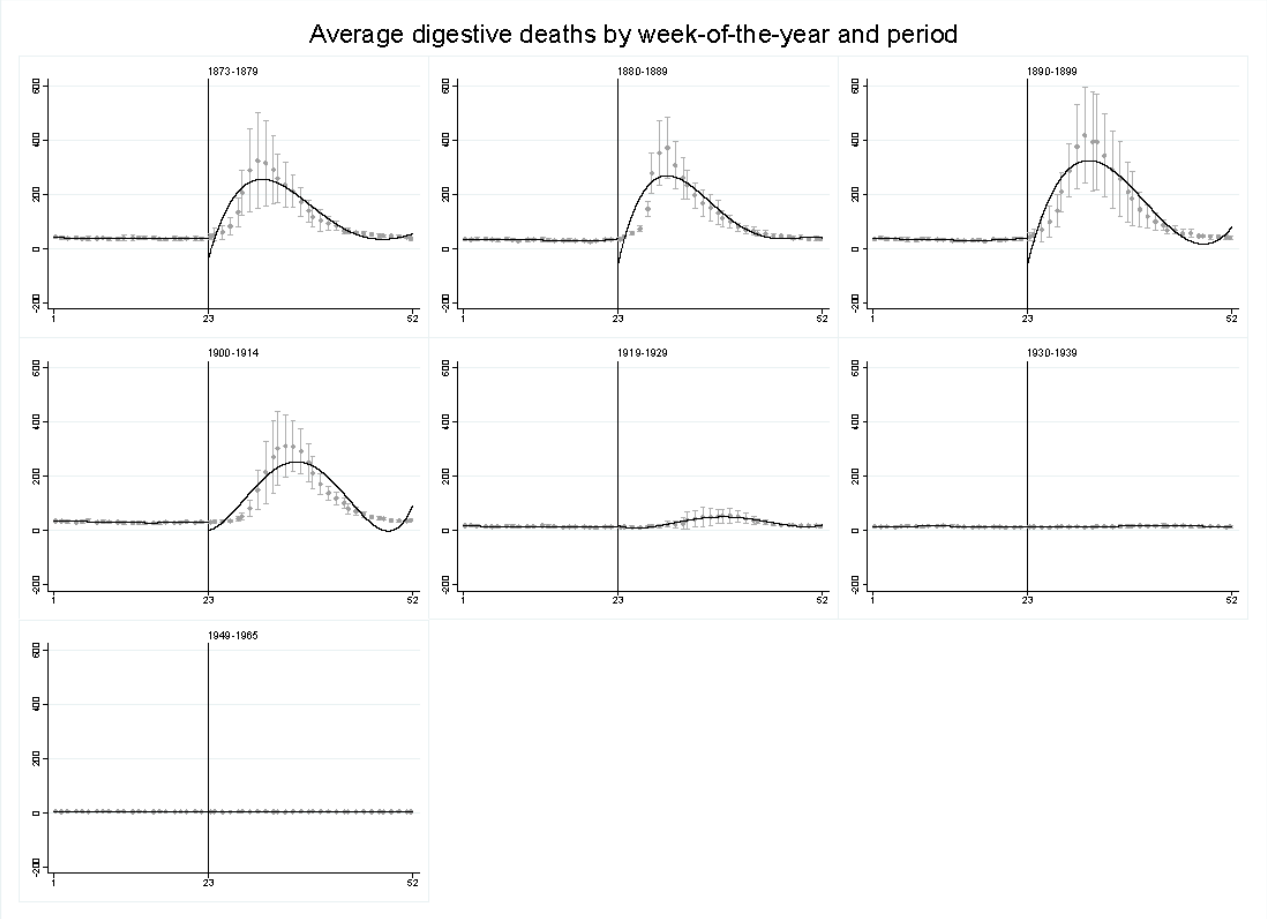
Notes: This figure shows the weekly average number of infant and digestive deaths by year (1873-1965), including 95 percent confidence bands, calculated for each year. The smooth black solid lines are fourth order polynomial fits for before/after the years 1900. The vertical lines are placed at year 1900.

Figure A.4: Distribution of infant deaths across weeks, by period



Notes: This figure shows the average number of infant deaths by week of the year, where 1 is the first week of January and 52 is the last week of December, for the periods: 1873-1879, 1880-1889, 1890-1899, 1900-1914, 1919-1929-1939, and 1949-1965 (averages and 95 confidence bands are based on these periods). The vertical lines are placed at week 23, which is typically around the first week of June.

Figure A.5: Distribution of digestive deaths across weeks, by period

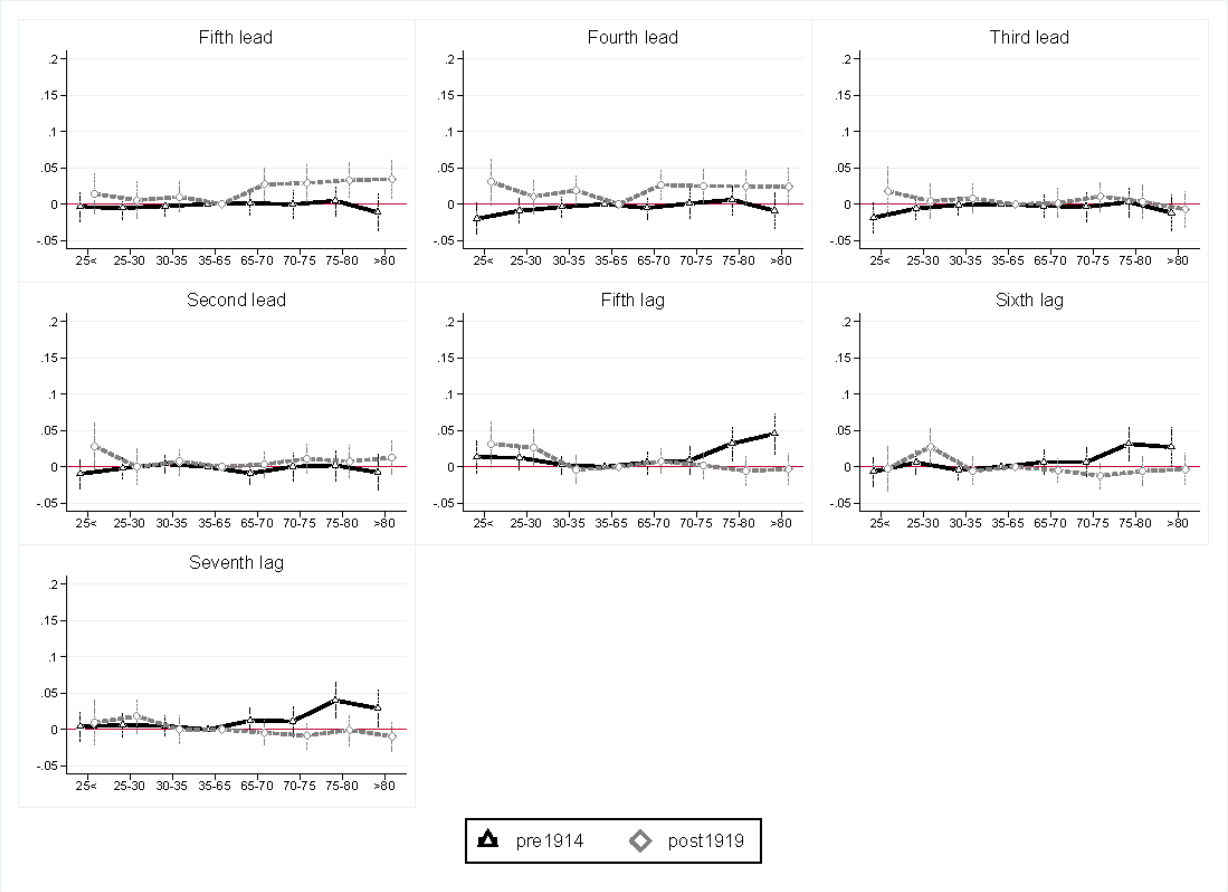


Notes: This figure shows the average number of digestive deaths by week of the year, where 1 is the first week of January and 52 is the last week of December, for the periods: 1873-1879, 1880-1889, 1890-1899, 1900-1914, 1919-1929-1939, and 1949-1965 (averages and 95 confidence bands are based on these periods). The vertical lines are placed at week 23, which is typically around the first week of June.

A.2.2 Additional total mortality results

Figure A.6 reports additional leads from our main total mortality regression results. Specifically, the figures plot the results for weeks 2-5 weeks before the week in which a particular temperature event is observed. These come from our main regression specification and are estimated at the same time as the remaining leads and lags presented in Figure 2. The main purpose here is to provide some additional evidence on the estimated pre-trends in weeks before a weather event is observed. The essentially flat and generally statistically insignificant estimates show that, as we would expect, weather events in a week are not systematically correlated with mortality in previous weeks.

Figure A.6: Additional leads and lags for total mortality regressions

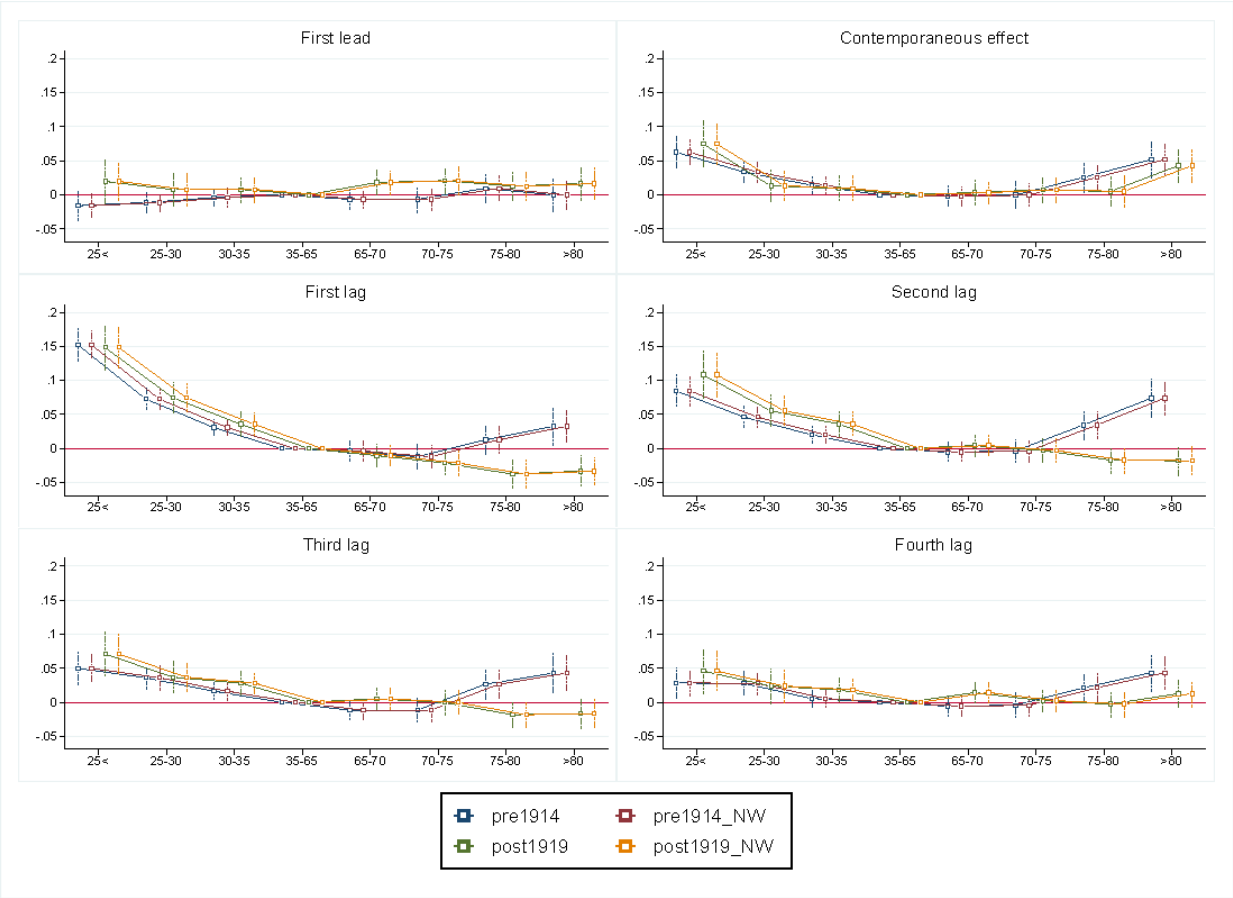


Notes: This figure shows the non-parametric relationship between temperature and log total mortality for leads 2-5 and lags 5-7 which were not reported in Figure 2. The pre-WWI sample is from 1866 to 1914 and the post-WWI sample is from 1919-1939 and 1949 to 1965. The reference weeks have minimum temperature above 35F and maximum temperature below 65F.

One potential concern in our analysis is that there may be serial correlation in the data, which are structured as a time series. A standard way to address serial correlation concerns is to allow correlation across temporally adjacent weeks using Newey-West standard errors.

In Figure A.7 we estimate results allowing correlation across time periods within seven weeks of each other and compare these to the results obtained from the robust standard errors presented in the main text. These results show that allowing for serial correlation typically leads to smaller confidence intervals, indicating that there may be mild negative serial correlation in the data. This should not be surprising given that we are studying mortality, because a high number of deaths in one week will reduce the population at risk of dying in the following week.

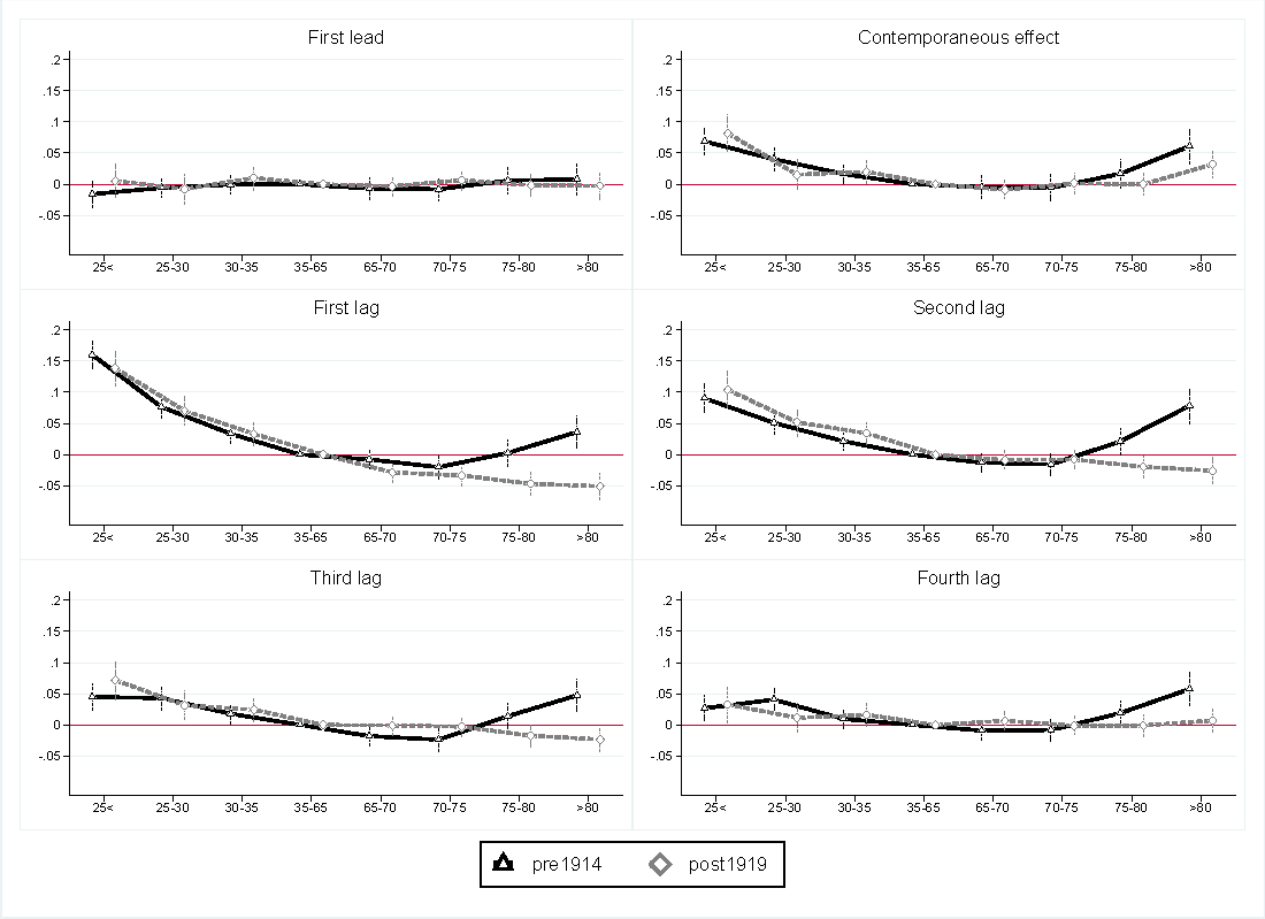
Figure A.7: Temperature and total mortality with Newey-West standard errors



Notes: This figure shows the non-parametric relationship between temperature and log total mortality for the first lead, the current effect, and four lags with robust Newey-West SEs allowing correlated errors across seven weeks. The length of the lag allowed in the Newey-West standard errors is based on the standard rule-of-thumb $T^{1/4}$ where T is the number of time periods in the analysis. We use the T from the longest sub-period that we analyze, the pre-WWI sample, which implies a lag length of seven. For consistency we apply this length to all of the time periods we study. The pre-WWI sample is from 1866-1914 and the post-WWI sample is from 1919-1939 and 1949-1965. The reference weeks have minimum temperature above 35F and maximum temperature below 65F.

It is also interesting to study results where we include month-by-year effects. Including such a rich set of fixed effects is possible due to the high-frequency nature of our data, though we do not use this as our main specification because it is likely to be over-controlling in a way that biases our results toward zero. It is worth noting that this specification allows us to control for time effects as flexibly as is possible in existing studies using monthly panel data, such as Barreca et al. (2016). In fact, because our study contains only one location, this specification is actually more flexible, since it is equivalent to including month-by-year-by-location effects in a panel data study, though that level of flexibility is impossible in panel data studies using monthly data at the location level. The results, in Figure A.8, show that even with this very flexible set of time effects we still obtain results that are similar to those presented in the main text, though the reduction in degrees of freedom means that we now have slightly larger standard errors. In summary, these results illustrate that we can control as flexibly for time effects as any existing panel data study that is reliant on monthly data.

Figure A.8: Temperature and total mortality with month-by-year fixed effects



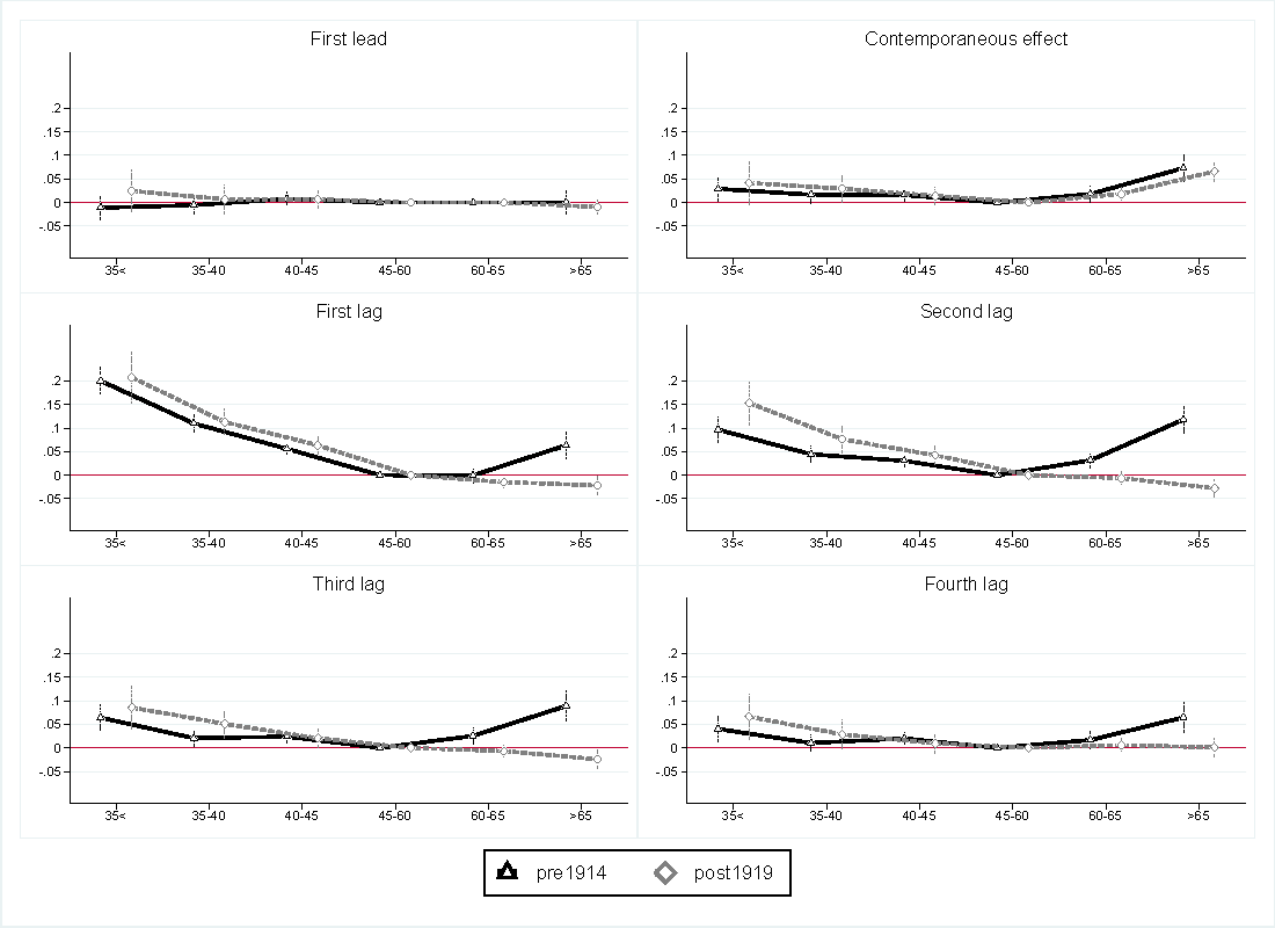
Notes: This figure shows the non-parametric relationship between temperature and log total mortality as in Figure 2, but also controlling for month-by-year fixed effects.

In the main analysis, we use temperature measures from Oxford rather than those available from London. In the next set of results, we show that the findings obtained from the Oxford data are essentially unchanged if we instead use temperature data from London. Note, however, that the London temperature comes from two different locations, Greenwich before WWII and Kew Gardens after. Also, only mean temperature is available, rather than average high and low temperatures for the week. This means that we have to work with different temperature bins in these results. The bins that we use are: under 35F (q1), 35-40 (q2), 40-45 (q3), 45-60 (q4, the reference bin), 60-65 (q5), and above 65 (q6). Note that, because we have to change our bin definitions, the coefficients will not be directly comparable to those shown in the main text.

Figure A.9 presents the estimated coefficients obtained when using the mean weekly temperature from London locations. Overall, the patterns shown in this figure are similar to those obtained from

our preferred Oxford temperature data. We see no evidence of pre-trends in the top-left panel. In the top right-panel, we see that both warm and cold weeks exhibit increased mortality, with the hot effects being stronger. The remaining panels show that the warm week effect persists strongly in the period before WWI but is essentially absent after WWI, while the effect of cold weather is similar in both periods and, if anything, somewhat stronger in the post-WWI period. Overall, these results confirm the patterns identified in our main specifications.

Figure A.9: Total mortality results using temperature measured in London

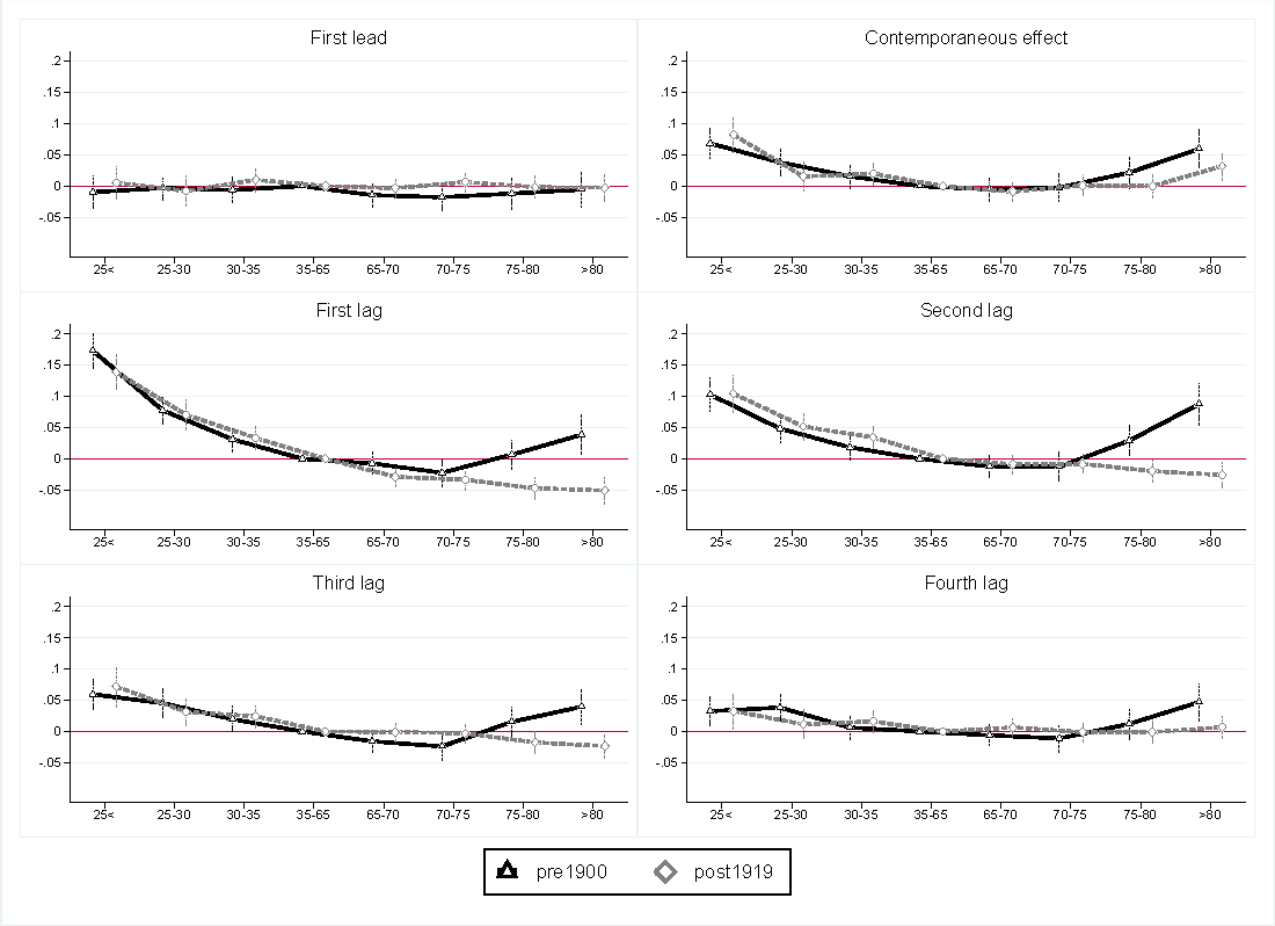


Notes: This figure shows the non-parametric relationship between temperature and log total mortality for the first lead, the current effect, and four lags. The temperature bins are based on mean weekly temperature measured in Greenwich Observatory before WWII and Kew Gardens after WWII. The pre-WWI sample is from 1866-1914 and the post-WWI sample is from 1919-1939 and 1949-1965. The omitted reference weeks have mean temperature from 45-60F.

In the main analysis, we have included the period from 1901-1914 as part of our early period. However, as shown in Figures A.3-A.5, this is perhaps better thought of as a transition period from

a high infant and digestive mortality regime of the late 19th century to the lower mortality regime the existed after WWI. Thus, we may be worried that the inclusion of 1900-1914 in our early period is influencing those results. As a check on this, in Figure A.10, we present results in which we excluded 1900-1914 from the early sample (those years are dropped from the analysis, not included in the later sample period). Overall, we can see that this has little impact on our results.

Figure A.10: Total mortality results with the early period ending in 1900



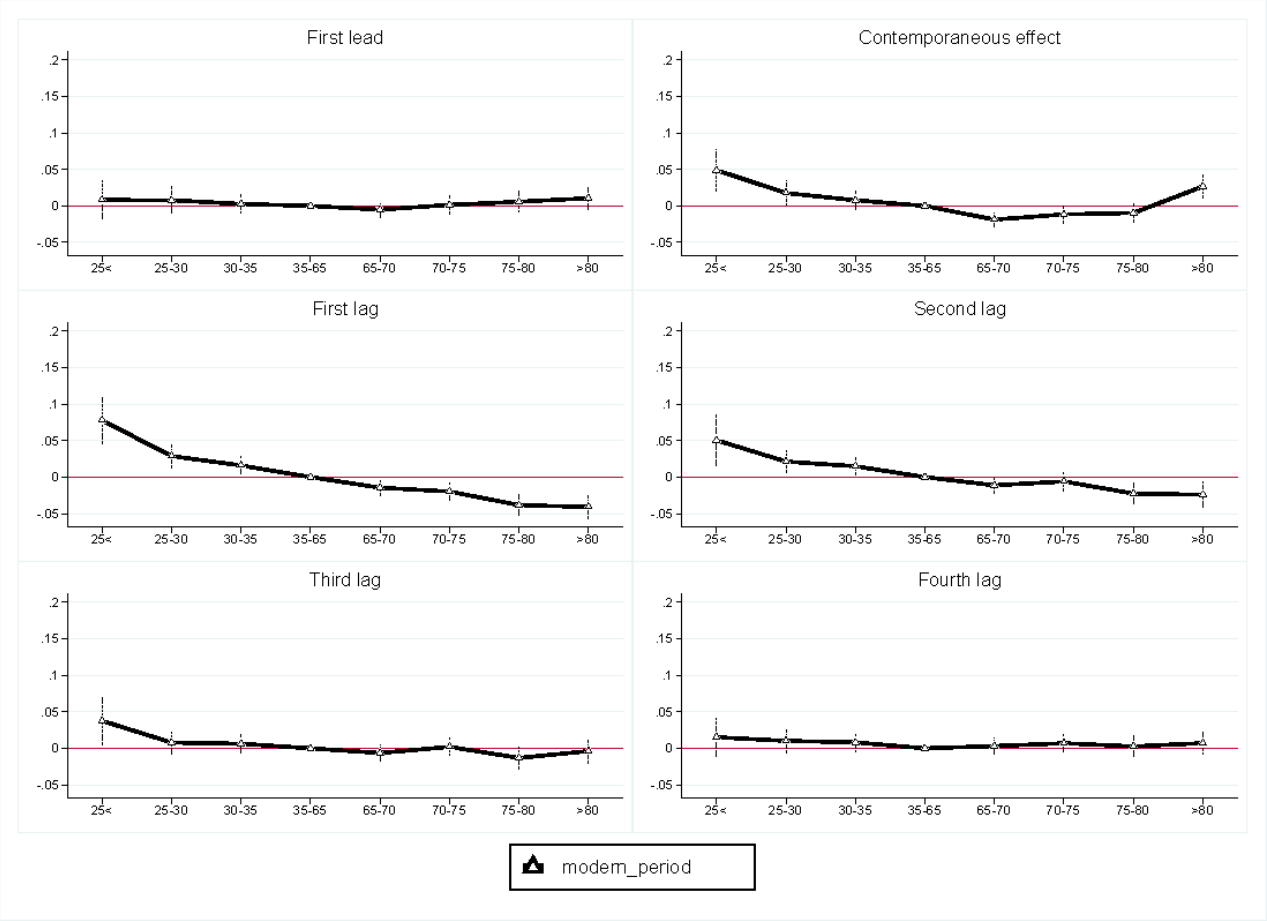
Notes: This figure shows the non-parametric relationship between temperature and log total mortality for the first lead, the current effect, and four lags. The pre-WWI sample is from 1866-1900 and the post-WWI sample is from 1919-1939 and 1949-1965. The omitted reference weeks have minimum temperature above 35F and maximum temperature below 65F.

In the next set of results, we study the temperature-mortality relationship in more recent data stretching from 1981-2006. These estimates, in Figure A.11, are very similar to what we observe in the period from 1919-1965. As in that period, we see an uptick in mortality in the week in which there is

an unusually warm temperature event, followed by reduced mortality for the next couple of weeks. The effect of unusually cold weeks remains positive, though the magnitude is smaller than what we observed in the 1919-1965 period.

It is worth noting that these results may be influenced by changes in the age profile of London across this period. Unfortunately, we cannot explore this possibility further because the U.K. Office of National Statistics has been unwilling to provide weekly mortality data by age group for London during this period and the data do not appear in printed records such as those produced for the years before 1970.

Figure A.11: Total mortality results for the modern period (1981-2006)



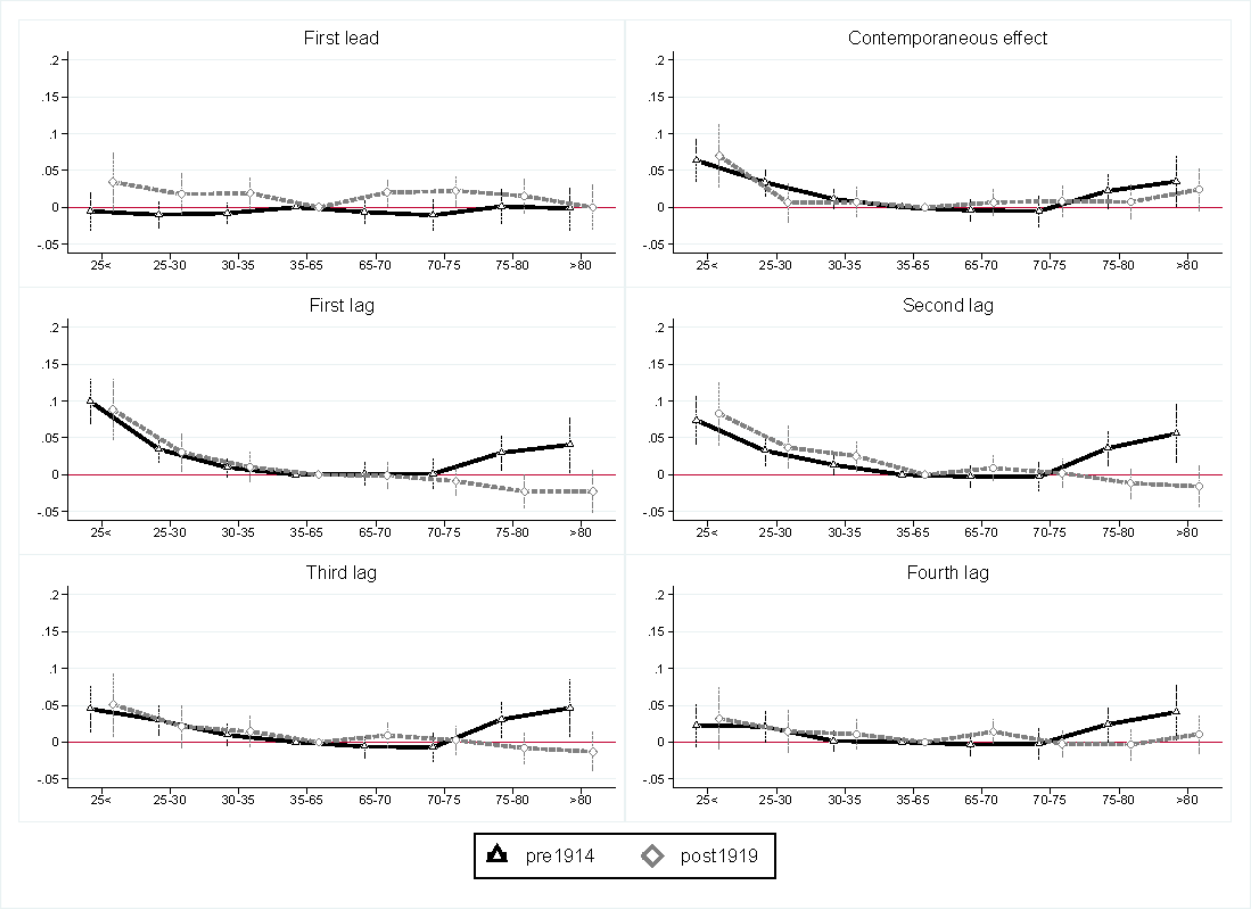
Notes: This figure shows the non-parametric relationship between temperature and log total mortality for one lead, the current effect, and four lags, using data from 1981 to 2006. The reference weeks have minimum temperature above 35F and maximum temperature below 65F.

In the next set of results, we consider the possibility that variation in humidity may be influencing our estimates of the temperature-mortality relationship. Existing work by Barreca (2012)

suggests that humidity can impact mortality in addition to just temperature, and that there may be an interaction between temperature and humidity. Thus, this is an important possibility to consider.

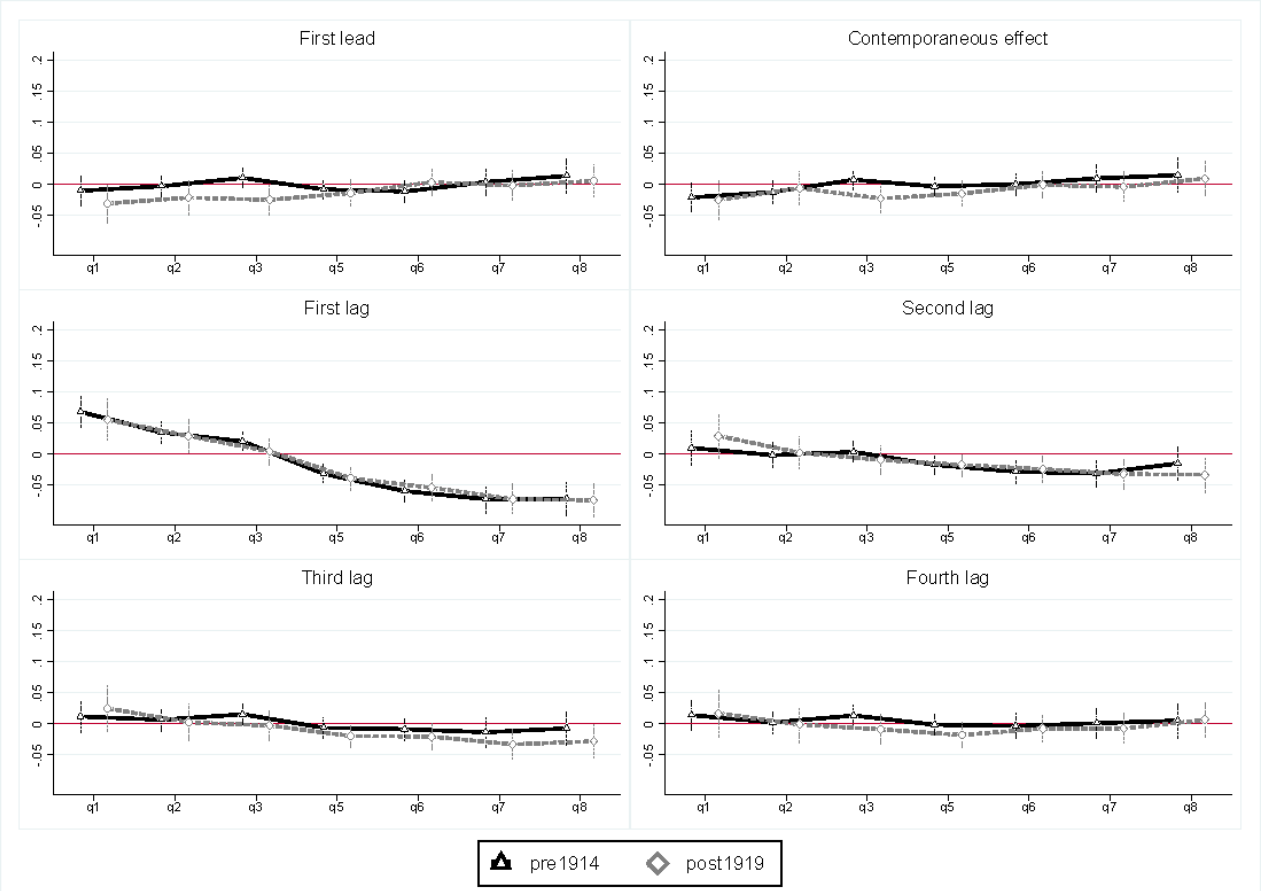
As a first step in assessing this issue, we use the relative humidity, temperature and pressure to calculate absolute humidity in each week. We then divide weeks into eight equally-sized bins based on average humidity level and construct a full set of five leads and seven lags for each of these absolute humidity bins (leaving out the middle bin as the reference). Figure A.12 presents regression results describing the relationship between temperature and total mortality obtained from a specification that includes the full set of binned absolute humidity variables (including leads and lags). These results are notably similar to those obtained in our main analysis specification, which suggests that controlling for humidity does not have a major impact on the estimated temperature mortality relationship. Figure A..13 reports the estimated humidity effects from the same regression. The pattern of elevated mortality at low humidity levels matches existing results from Barreca (2012).

Figure A.12: Total mortality results controlling for a full set of leads and lags of absolute humidity



Notes: This figure shows the non-parametric relationship between temperature and log total mortality for the first lead, the current effect, and four lags as in Figure 2, but also controlling for the non-parametric relationship between mortality and absolute humidity for a set of five leads, the contemporary effect, and seven lags, as well as the interaction between the highest temperature bin and the highest humidity bin. The main humidity estimates are reported in Figure A.13. The pre-WWI sample is from 1866-1914 and the post-WWI sample is from 1919-1939 and 1949-1965. The reference weeks have minimum temperature above 35F and maximum temperature below 65F.

Figure A.13: Humidity and total mortality before and after WWI controlling for a full set of leads/lags of temperature

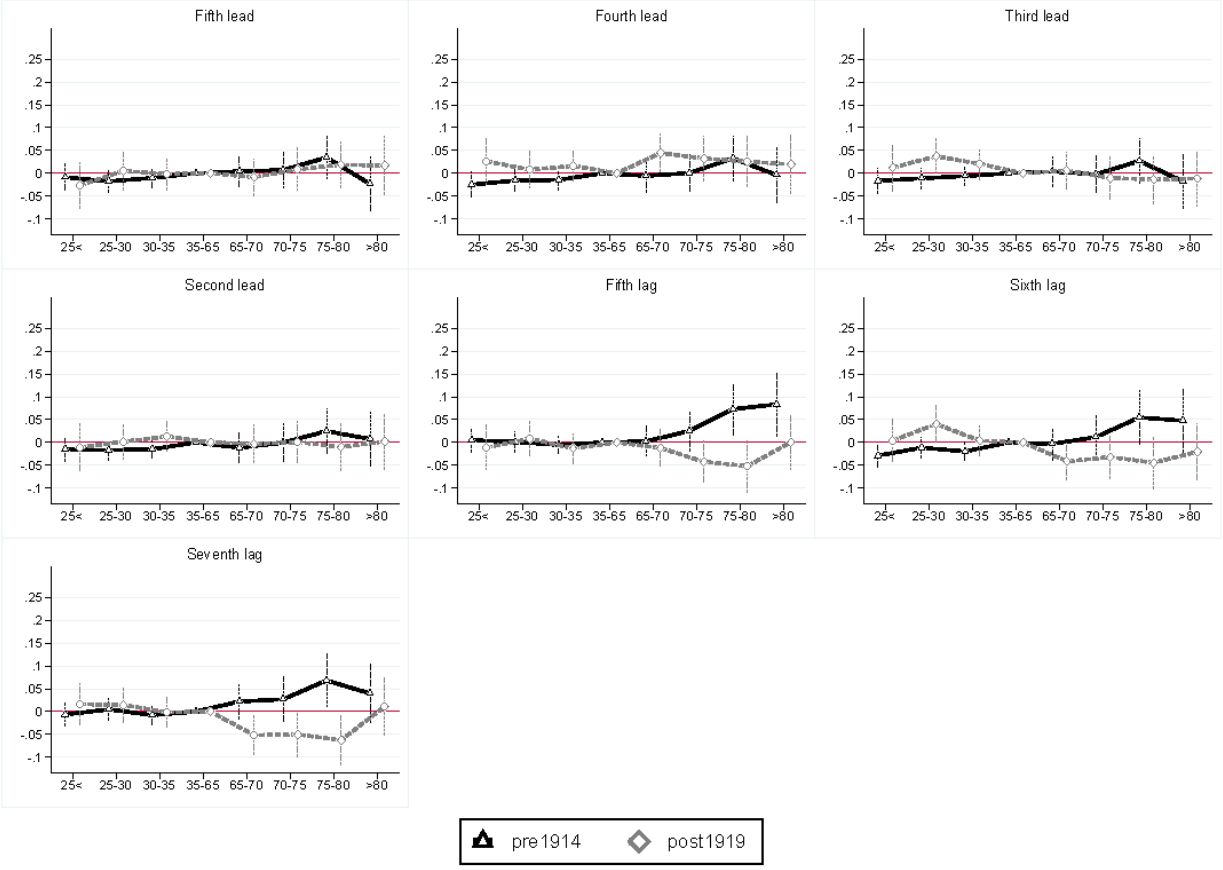


Notes: This figure shows the non-parametric relationship between absolute humidity and log total mortality for the first lead, the current effect, and four lags from the same regression as in Figure A.12. The pre-WWI sample is from 1866-1914 and the post-WWI sample is from 1919-1939 and 1949-1965.

A.2.3 Additional infant mortality results

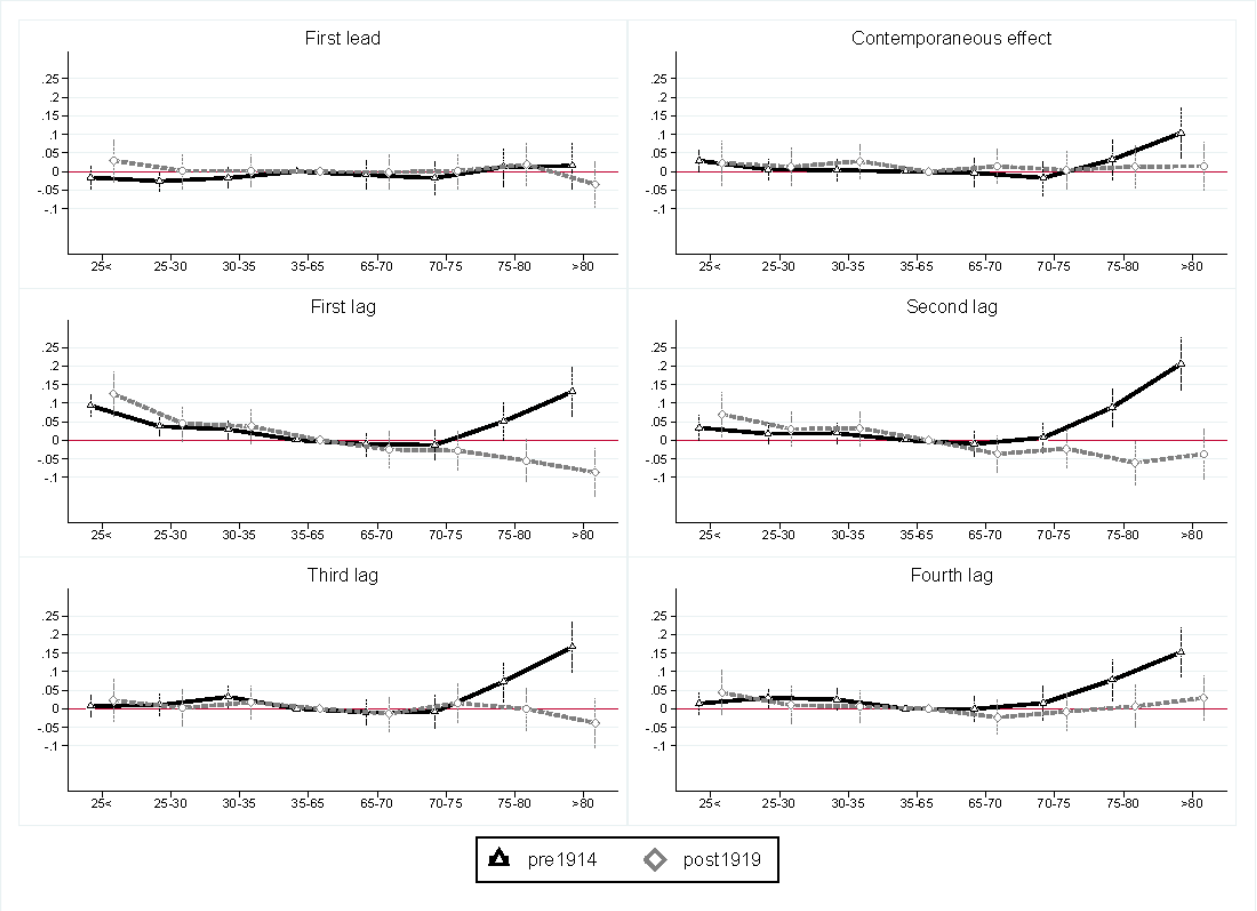
Next, we present additional infant mortality results. Figure A.14 reports leads 2-5 and lags 5-7 for log infant mortality, showing that there is no evidence of pre-trends in this specification, for example. Figure A.15 shows that our infant mortality results are largely unchanged if we include month-by-year fixed effects in the specification. Figure A.16 presents results using temperature data from London rather than Oxford. Figure A.17 presents estimates where the early period ends in 1910 rather than 1914. Figure A.18 shows that our infant results are robust to controlling for humidity as was also the case for total mortality, and Figure A.19 reports the humidity estimates from this specification. The patterns of the humidity estimates are similar to the results for total mortality, but somewhat weaker in numerical magnitude for the first lag. As our final robustness check for infant mortality, Figure A.20 shows that our results are not driven by any scale effects coming from the ongoing fertility transition during this period, as was also the main message coming from Figure A.2.

Figure A.14: Additional leads and lags from the infant mortality regressions



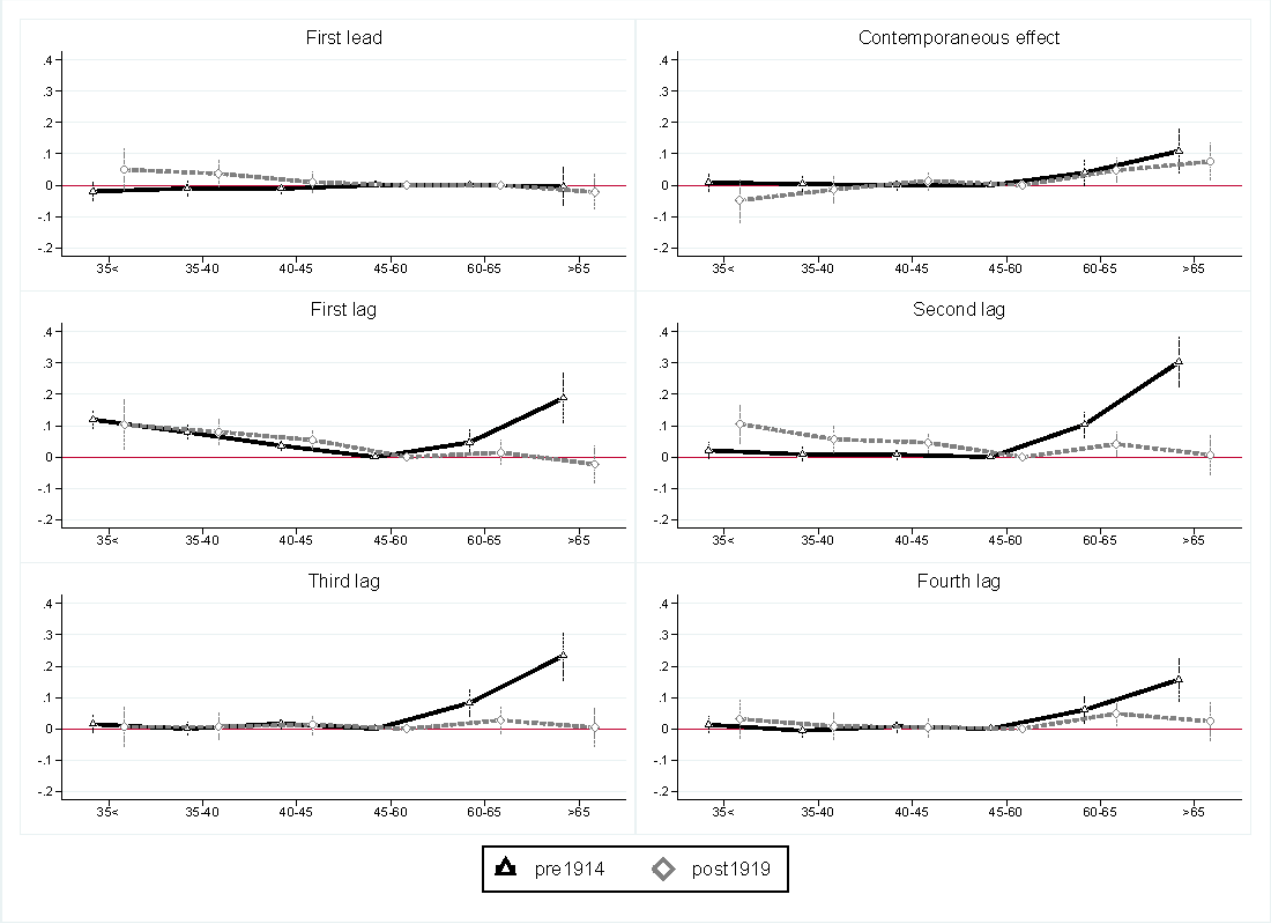
Notes: This figure shows the non-parametric relationship between temperature and log infant mortality for leads 2-5 and lags 5-7 which were not reported in Figure 3. The pre-WWI sample is from 1874 to 1914 and the post-WWI sample is from 1919-1939 and 1949-1965. The omitted reference temperature bin is $q = 4$ or $35F - 65F$.

Figure A.15: Temperature and infant mortality before and after WWI, controlling for month- by-year fixed effects



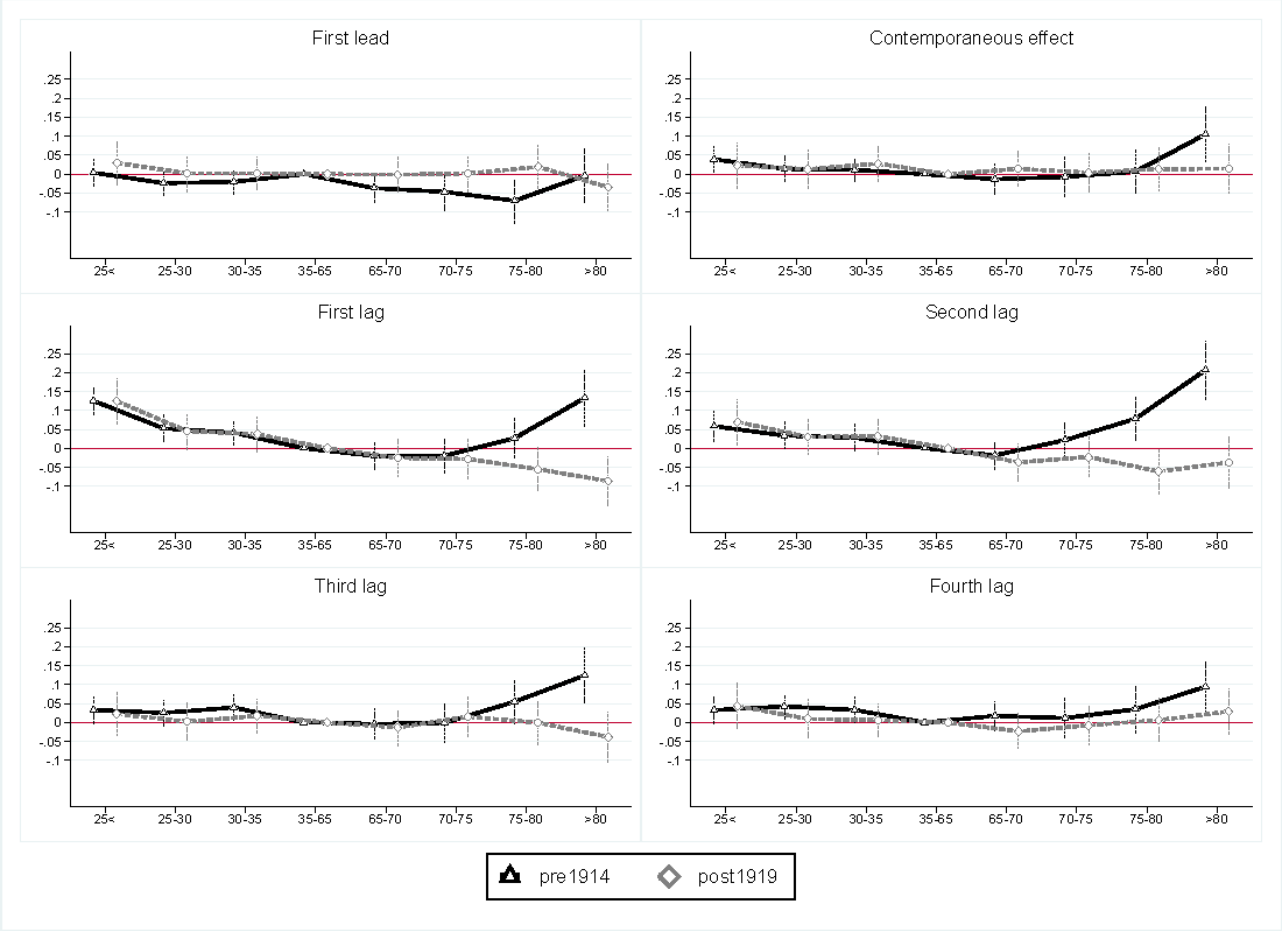
Notes: This figure shows the non-parametric relationship between temperature and log infant mortality as in Figure 3, but also controlling for month-by-year fixed effects.

Figure A.16: Infant mortality results using mean weekly temperature measured in London



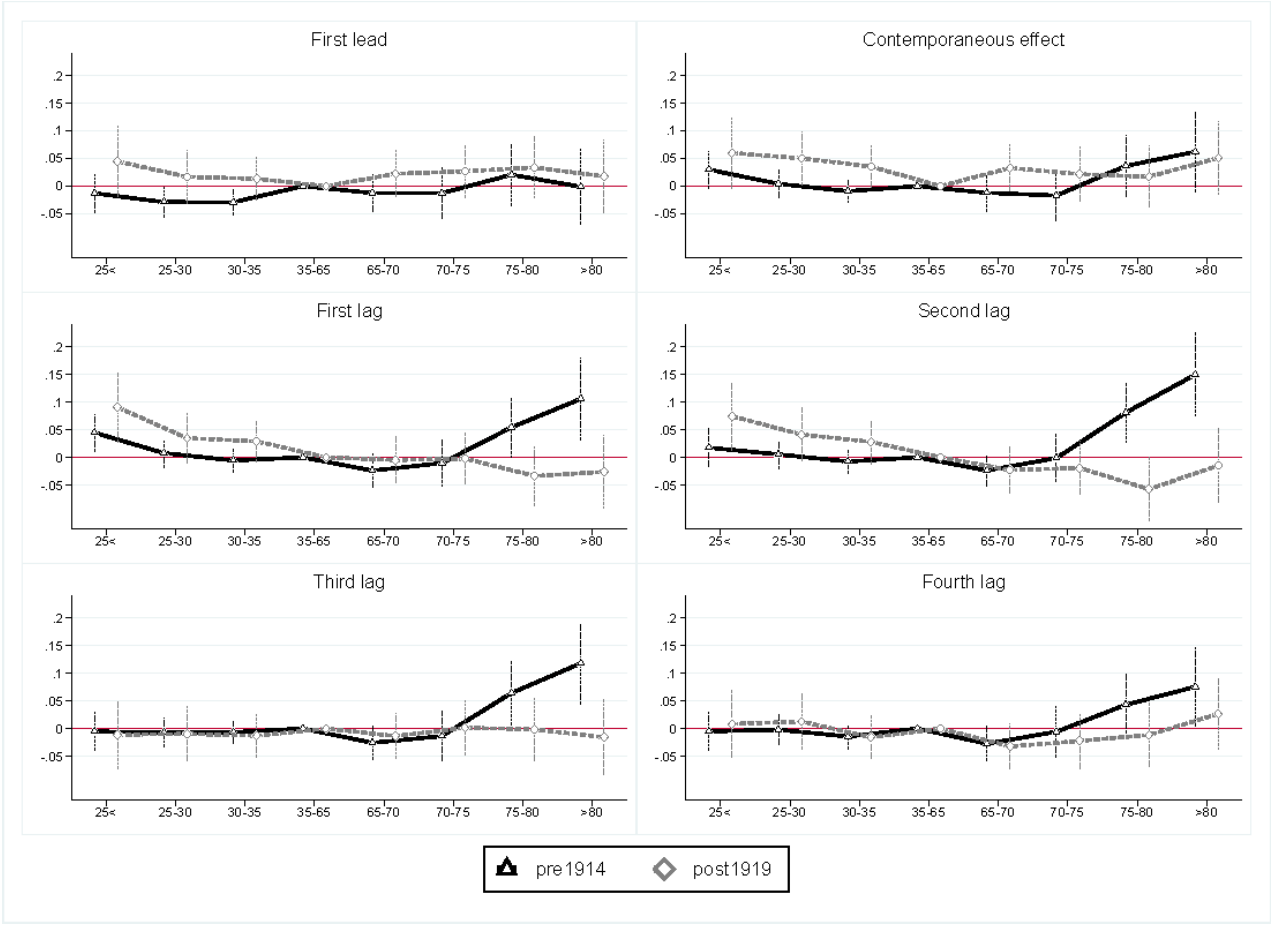
Notes: This figure shows the non-parametric relationship between temperature and log infant mortality for the first lead, the current effect, and four lags. The temperature bins are based on mean weekly temperature measured in Greenwich Observatory before WWII and Kew Gardens after WWII. The pre-WWI sample is from 1866-1914 and the post-WWI sample is from 1919-1939 and 1949-1965. The omitted reference weeks have mean temperature from 45-60F.

Figure A.17: Temperature and infant mortality excluding data from 1900-1914



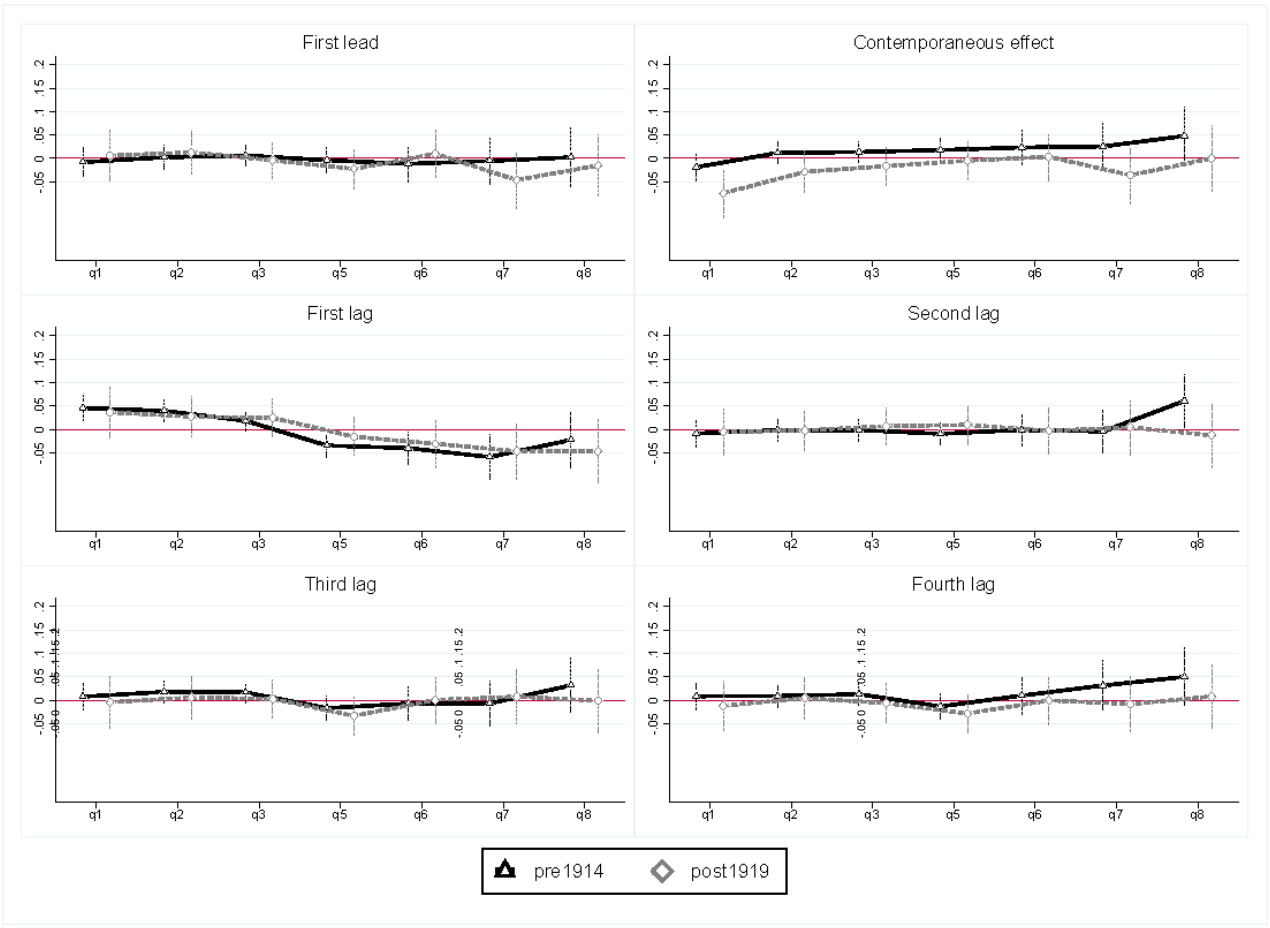
Notes: This figure shows the non-parametric relationship between temperature and log infant mortality for the first lead, the current effect, and four lags. The pre-WWI sample is from 1866-1900 and the post-WWI sample is from 1919-1939 and 1949-1965. The omitted reference weeks have minimum temperature above 35F and maximum temperature below 65F.

Figure A.18: Temperature and infant mortality controlling for a full set of leads and lags of absolute humidity



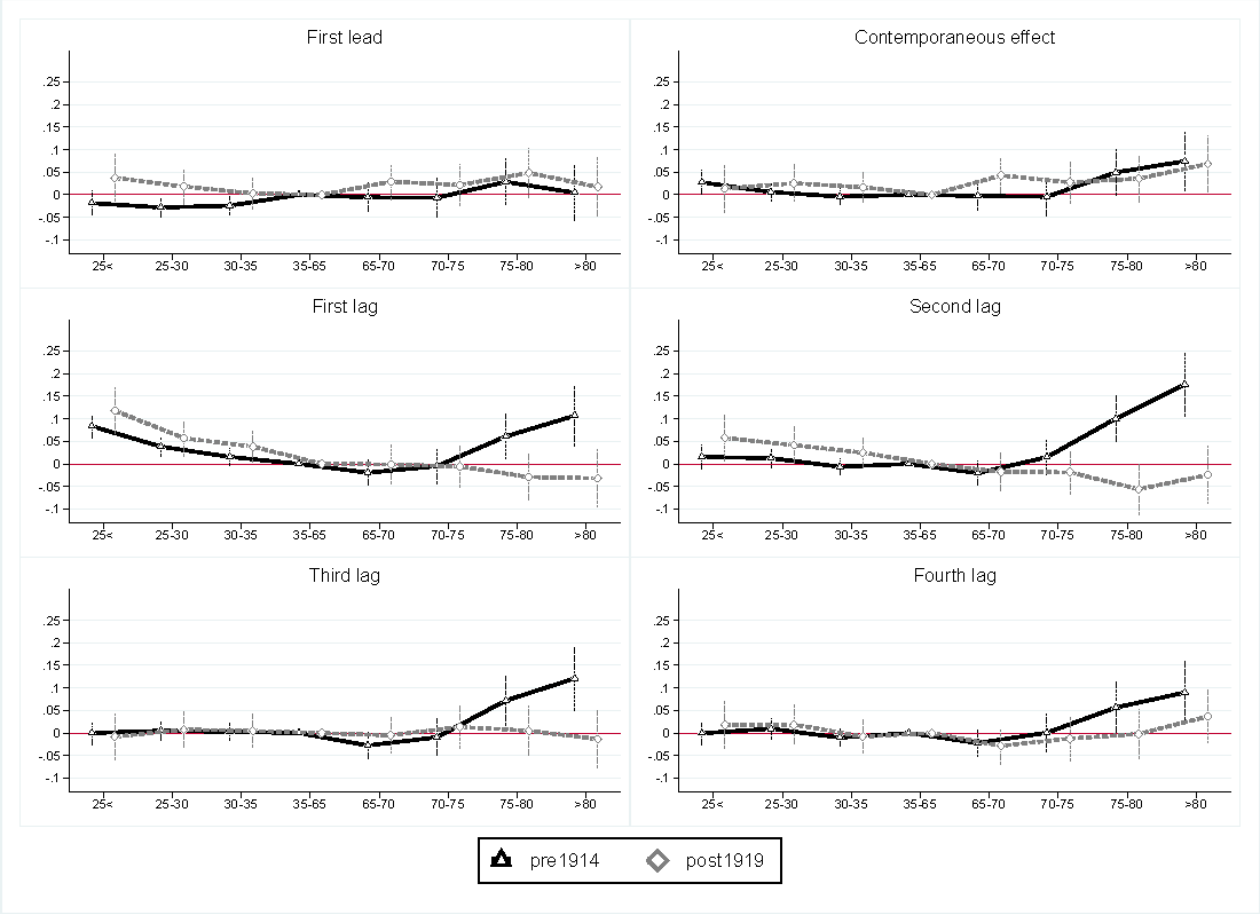
Notes: This figure shows the non-parametric relationship between temperature and log infant mortality for the first lead, the current effect, and four lags as in Figure 3, but also controlling for the non-parametric relationship between mortality and absolute humidity for a set of five leads, the contemporary effect, and seven lags. The main humidity estimates are reported in Figure A.19. The pre-WWI sample is from 1874-1914 and the post-WWI sample is from 1919-1939 and 1949-1965. The omitted reference temperature bin is $q = 4$ or $35F - 65F$.

Figure A.19: Humidity and infant mortality before and after WWI controlling for full set of leads/lags of temperatures



Notes: This figure shows the non-parametric relationship between humidity and log total mortality for the first lead, the current effect, and four lags from the same regression as in Figure 18A. The pre-WWI sample is from 1866-1914 and the post-WWI sample is from 1919-1939 and 1949-1965. The omitted reference humidity bin is $q = 4$ or $35F - 65F$.

Figure A.20: Temperature and the infant mortality rate before and after WWI



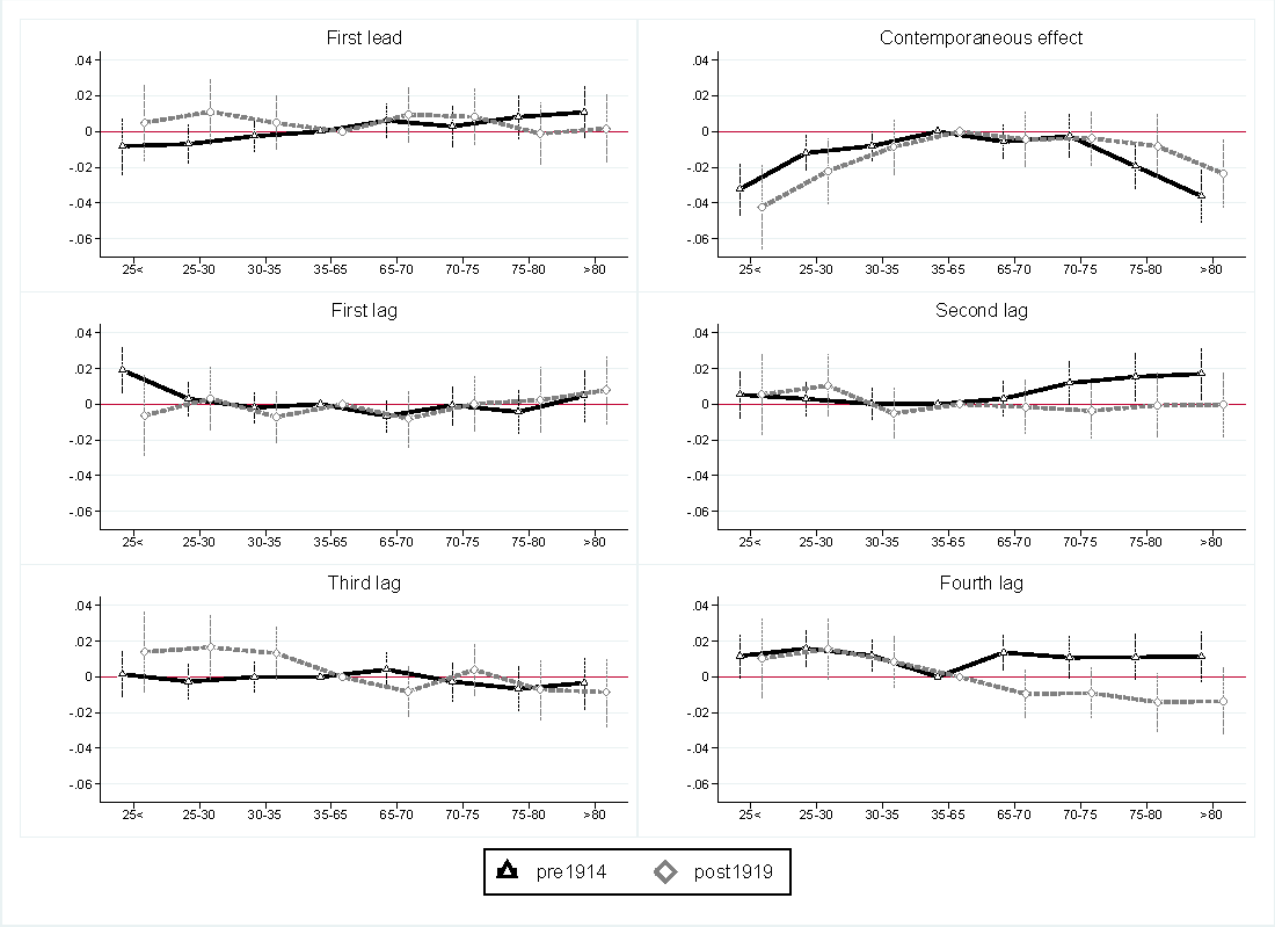
Notes: This figure shows the non-parametric relationship between temperature and the log infant mortality rate for the first lead, the current effect, and four lags. The denominator of the infant mortality rate uses the number of births the past 52 weeks. The pre-WWI sample is from 1874-1914 and the post-WWI sample is from 1919-1939 and 1949-1965. The omitted reference temperature bin is $q = 4$ or $35F - 65F$.

A.3 Analysis of temperature and births

Figure A.21 reports the relationship between live births and temperature before and after WWI. The most notable feature from this graph is that the number of live births are lower during weeks with cold and warm temperatures, but this relationship appears to have been stable over our study period. This finding is consistent with literature from modern developed countries showing that exposure to temperature extremes is associated with an increased risk of stillbirth (Strand et al., 2011; Auger et al., 2017). While existing work focuses primarily on hot temperatures, our findings provide some indirect evidence that low temperatures may also be associated with an increase in stillbirths.

The results in Figure A.21 relate to a relatively small literature looking at the relationship between ambient temperature and contemporaneous births. One recent paper in this area is Auger et al. (2014). That study, which uses data from Montreal, CA covering 1981-2010, looks at the relationship between the number of births in a week and temperature in the previous week. The authors find evidence that temperatures above 32 C in the past week are associated with an increase in births at term of around 4%, with no impact on pre- term births. It is somewhat difficult to compare these results with our findings, since we study a broader set of contemporaneous and lagged effects, rather than just a single one-week lag. However, we do find some evidence of a weak lagged increase in births, which seems consistent with their results, though this effect is strongest two weeks after a high temperature event. Similar patterns are observed in other studies, such as Ha et al. (2016). Note that this literature differs from work, such as Barreca (2017) and Barreca et al. (2018), which focuses mainly on the impact of temperature on conception.

Figure A.21: Temperature and live births before and after WWI

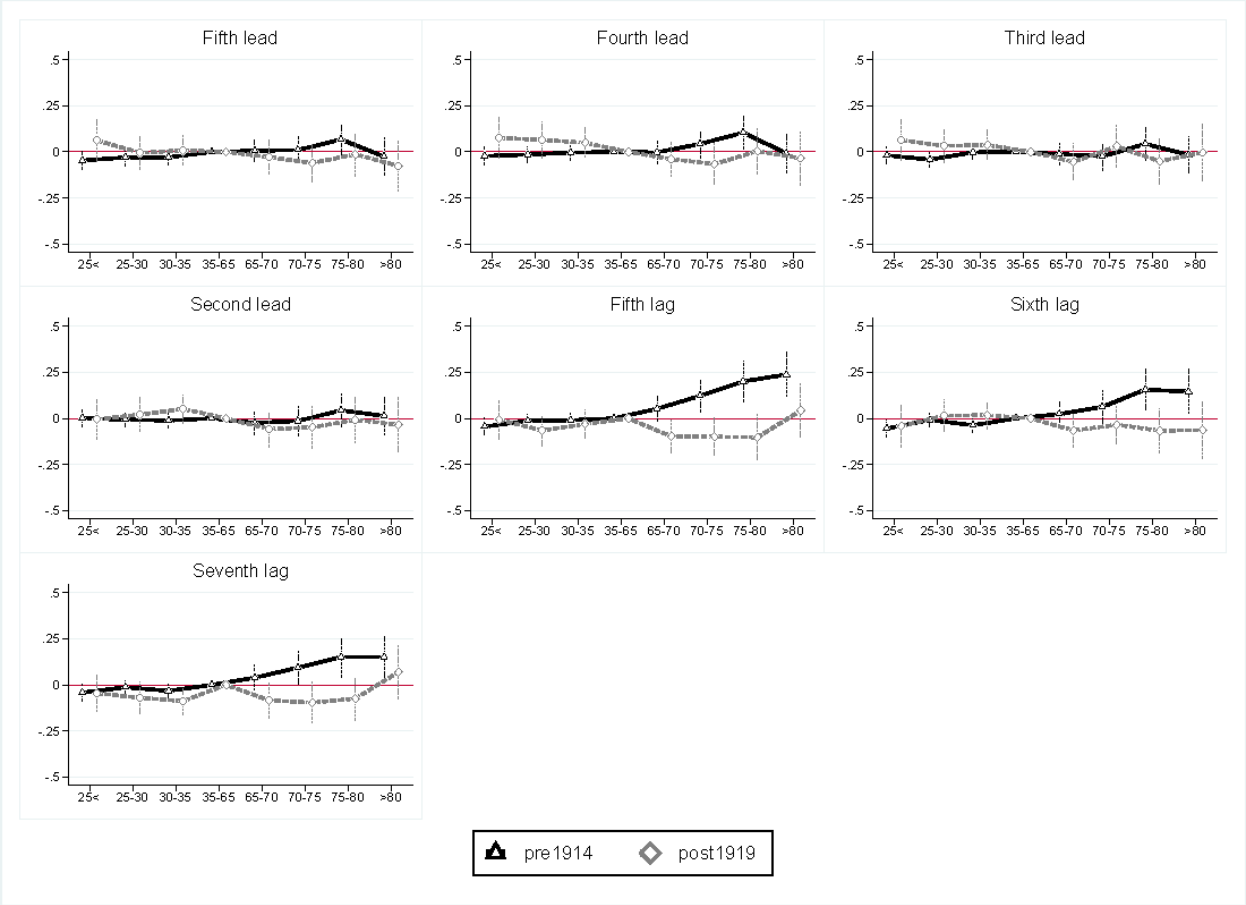


Notes: This figure shows the non-parametric relationship between temperature and log live births for the first lead, the current effect and four lags. The pre-WWI sample is from 1866 to 1914 and the post-WWI sample is from 1919-1939 and 1949-1965. The omitted reference temperature bin is $q = 4$ or $35F - 65F$.

A.4 Additional analysis of causes of death

This section presents additional results looking at digestive disease mortality data. First, Figure A.22 reports the remaining leads, not reported in the paper, for digestive mortality.

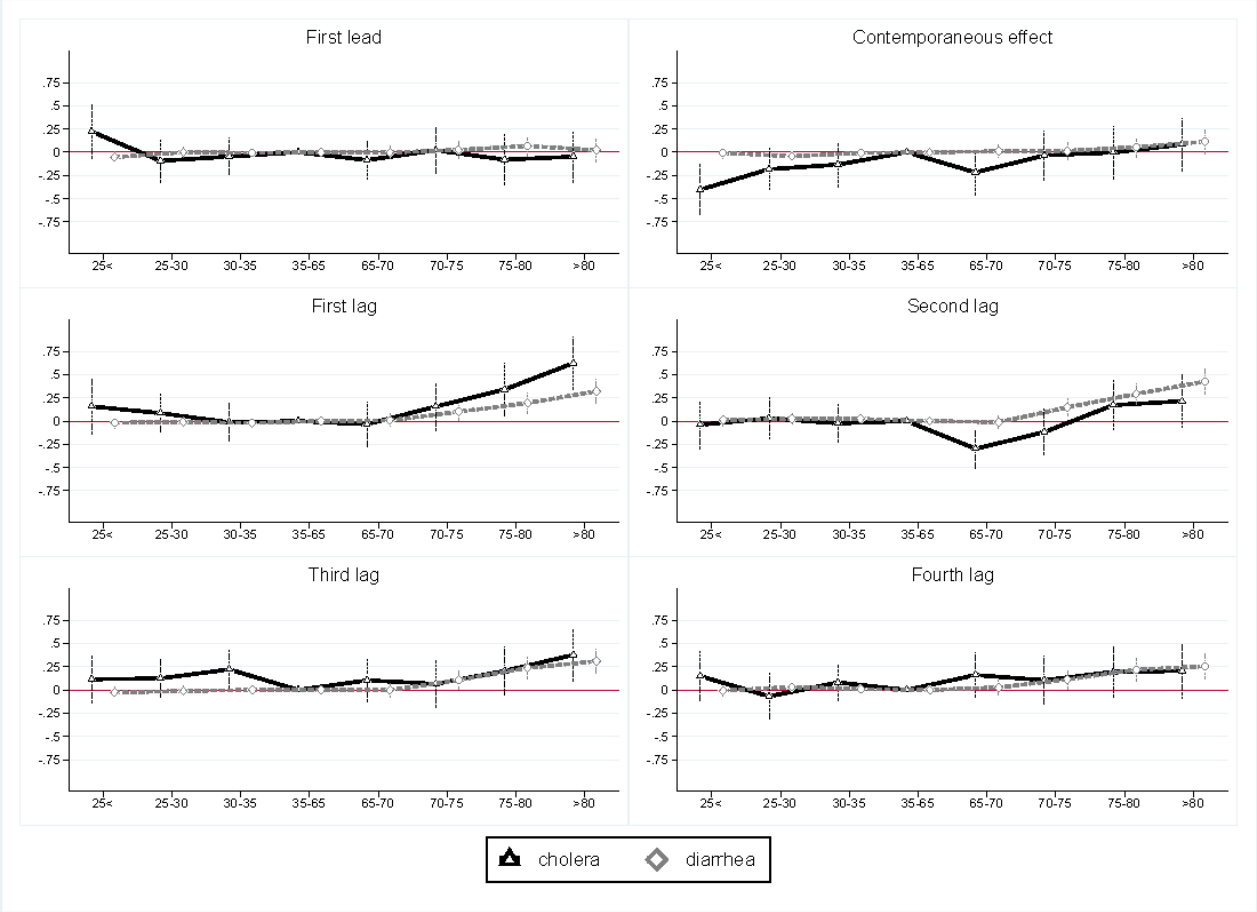
Figure A.22: Additional leads and lags for the digestive disease mortality regressions



Notes: This figure shows the non-parametric relationship between temperature and log digestive mortality for four additional leads and three additional lags which were not reported in Figure 5. The pre-WWI sample is from 1870 to 1914 and the post-WWI sample is from 1919-1939 and 1949-1965. The omitted reference temperature bin is $q = 4$ or $35F - 65F$.

Next, Figures A.23 and A.24 present additional results breaking down digestive disease deaths into subcategories. These results focus on data prior to 1911, both because this allows us to construct more consistent series and because some of these categories, such as cholera, had effectively disappeared by the end of that period. The results in Figure A.23 show that both cholera and the (much larger) diarrhea and dysentery cause of death groupings exhibited increased mortality associated with hot weather. The estimates for the heat effect on cholera deaths are large, but recall that this was applied on a much lower baseline level of cholera deaths.

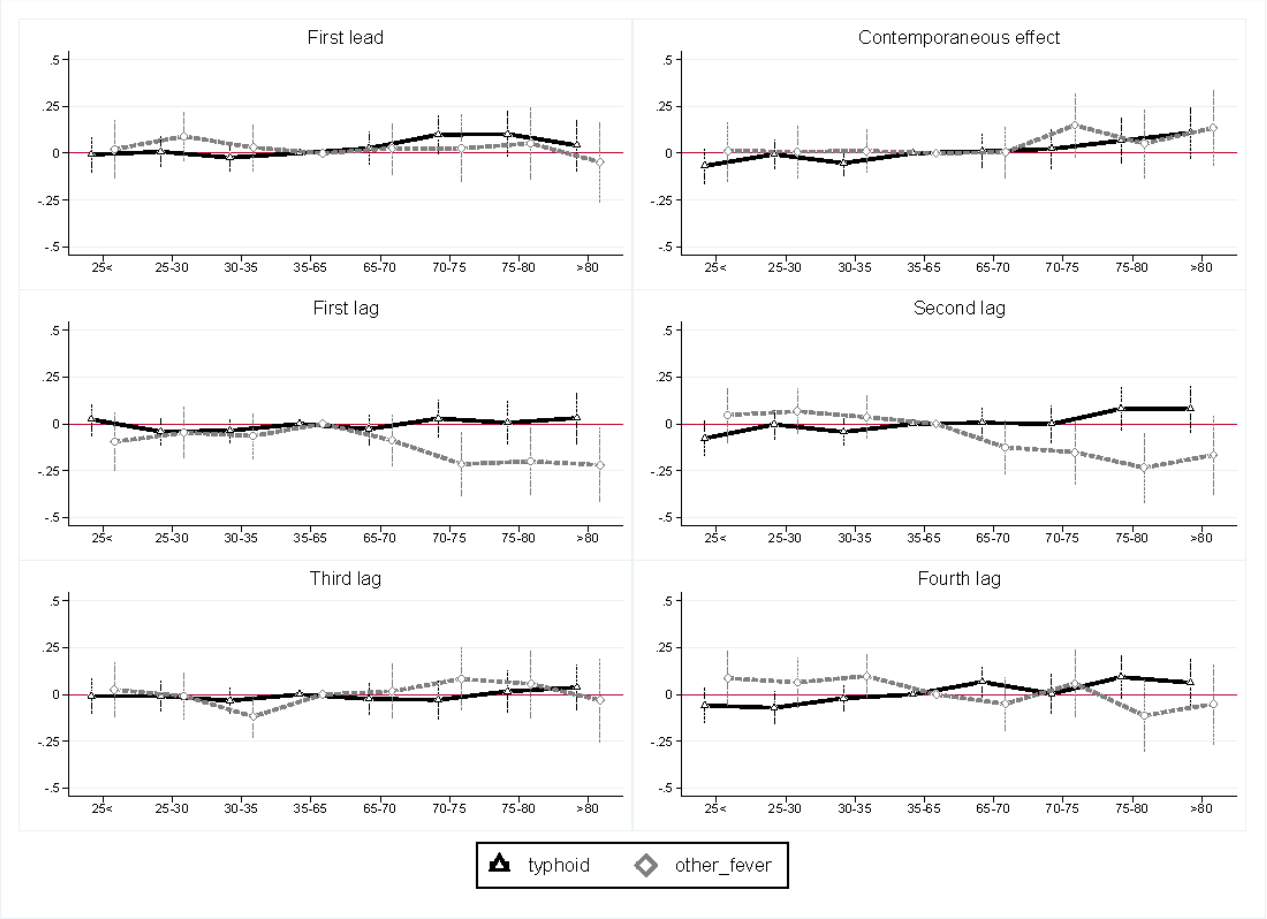
Figure A.23: Results for cholera and the diarrhea & dysentery categories, 1870-1910



Notes: This figure shows the non-parametric relationship between temperature and log cholera mortality or log mortality from diarrhea (which includes dysentery, enteritis and gastritis) for the first lead, the current effect and four lags. The pre-WWI sample is from 1866 to 1914 and the post-WWI sample is from 1919-1939 and 1949-1965. The omitted reference temperature bin is $q = 4$ or $35F - 65F$.

In the next set of results, Figure A.24, we present estimates for deaths due to typhoid fever and, for comparison, results from other fevers (which are not included in the digestive disease category). These results show that typhoid deaths were not strongly associated with warmer temperature. Only in the contemporaneous week do we see evidence (not statistically significant) of an increase in typhoid deaths. Typhoid appears to make no contribution to the lagged impact of temperature on mortality, which formed the bulk of the impact of high temperature on mortality in Figure 5. Deaths due to other fevers also show no increase during periods of high temperature, and there is evidence of negative lagged effects which probably operate through competing risk effects.

Figure A.24: Temperature and deaths due to typhoid or other fevers, 1870-1910



Notes: This figure shows the non-parametric relationship between temperature and log typhoid fever mortality or log mortality due to other fevers (including simple continued fever and remittent fever) for the first lead, the current effect and four lags. The pre-WWI sample is from 1866 to 1914 and the post-WWI sample is from 1919-1939 and 1949-1965. The omitted reference temperature bin is $q = 4$ or $35F - 65F$.

A.5 Effect of cumulative weeks of high or low temperature

In this appendix, we explore the additional impact of having multiple weeks of hot or cold temperature in a row. While the results in our main analysis allow lagged temperature effects, they do not explicitly incorporate the possibility that having several weeks of unusually cold or warm weather may have cumulative effects. To capture these cumulative effects, we construct additional variables reflecting whether a particular warm or cold week has been preceded by one, two or three similar weeks. In constructing these variables, we consider a hot week any week falling into the top two temperature bins and a cold week any week falling into the bottom two bins. We then add these variables into our standard regressions, so that the resulting coefficients reflect the additional effect of having multiple weeks of high or low temperatures in a row, beyond the direct effect of having high or low temperature in a week or in a previous week captured by our standard temperature variables.

The results for total mortality are presented in Table A.1. The results in Columns 2 and 4 look at the impact of having two hot or two cold weeks in a row in, respectively, the pre-WWI and post-WWI periods. Columns 2 and 5 look across three week windows while Columns 3 and 6 look across four-week windows. These results provide evidence that in the pre-WWI period, multiple weeks of high temperature resulted in additional deaths, beyond the direct effect of temperature in a week measured previously. This effect appears most strongly when we have two or three weeks of high temperature in a row. No similar pattern of cumulative effects appears for low temperatures, or for high temperatures in the post-WWI period. Table A.2 presents similar results for infant mortality. Here we see similar patterns with even stronger cumulative effects of high temperatures in the pre-WWI period.

Table A.1: Estimates of the cumulative effect of multiple hot or cold weeks in a row on total mortality

	DV: Log total mortality					
	Pre-WWI			Post-WWI		
	(1)	(2)	(3)	(4)	(5)	(6)
High temp for two weeks	0.0192			-0.0171		
	(0.0163)			(0.0126)		
Low temp for two weeks	-0.0146			0.0134		
	(0.0128)			(0.0196)		
High temp for three weeks		0.0339**			0.00644	
		(0.0171)			(0.0116)	
Low temp for three weeks		-0.00500			0.00929	
		(0.0131)			(0.0208)	
High temp for four weeks			0.0516**			0.0203
			(0.0203)			(0.0135)
Low temp for four weeks			0.00101			0.0239
			(0.0153)			(0.0235)
Observations	2,498	2,498	2,498	1,889	1,889	1,889
R-squared	0.715	0.716	0.716	0.849	0.849	0.849

Notes: Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1. Regressions also include a full set of temperature bin indicator variables for five leads, the current period, and seven lags, as well as weekly controls for rainfall and fog events.

Table A.2: Estimates of the cumulative effect of multiple hot or cold weeks in a row on infant mortality

	DV: Log infant mortality					
	Pre-WWI			Post-WWI		
	(1)	(2)	(3)	(4)	(5)	(6)
High temp for two weeks	0.0587			-0.0958***		
	(0.0418)			(0.0360)		
Low temp for two weeks	-0.00148			-0.0402		
	(0.0155)			(0.0293)		
High temp for three weeks		0.122**			-0.0523	
		(0.0478)			(0.0341)	
Low temp for three weeks		-0.00696			-0.0297	
		(0.0150)			(0.0310)	
High temp for four weeks			0.176***			0.00852
			(0.0534)			(0.0387)
Low temp for four weeks			-0.00314			-0.00292
			(0.0166)			(0.0349)
Observations	2,101	2,101	2,101	1,889	1,889	1,889
R-squared	0.728	0.730	0.732	0.908	0.908	0.908

Notes: Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1. Regressions also include a full set of temperature bin indicator variables for five leads, the current period, and seven lags, as well as weekly controls for rainfall and fog events.

A.6 Summary effects across eight-week periods

In this appendix, we present estimates of summary effects of a hot or cold temperature event across the week in which it occurs and the following seven weeks. These provide a useful summary of the magnitude of the effects that we observe averaged across an eight-week period, starting with the week in which exposure occurred. The estimating equation for this exercise is,

$$ln(y_{wt}) = \sum_{q=1,q\neq 4}^{8} \beta_q TEMP_{wt}^{q} + \delta_w + \delta_t + X_{wt}\eta + \varepsilon_{wt}$$

where $TEMP_{wt}^{q}$ is an indicator that takes a value of one if the temperature in any week between *wt* and *wt-7* fell into the q'th temperature bin and zero otherwise. Thus, the coefficient β_1 reflects the impact of having temperature fall into the lowest temperature bin in a week, averaged across that week and the next seven weeks. Similarly, β_8 reflects the impact of having temperature fall into the highest temperature bin in a week, averaged across that week and the next seven weeks. These provide a useful way to summarize the contemporaneous and lagged effects (out to seven weeks) of high or low temperature events.

The results obtained when applying this approach to total mortality, infant mortality, and digestive disease mortality are presented in Table A.3, focusing only on the highest and lowest temperature bins. Columns 1 and 2 present, respectively, results for total mortality before and after WWI. In both periods, cold weeks increase total mortality by about 7 percent. Warm weeks raise total mortality by about 4 percent in the earlier period, but have essentially no effect after WWI. Columns 3-4 focus on infant deaths. These show that warm weeks increased mortality by about 10 percent in the pre-1914 period, but that this decreased to a statistically insignificant 2.3 percent after 1919. We observe only mild evidence of an increase in infant mortality associated with cold weeks. We estimate similar patterns when focusing on the infant mortality rate (i.e., infant mortality relative to births in the past 52 weeks) in Columns 5-8. Finally, in Columns 9-10 we look at the impact of temperature on deaths due just to digestive diseases. These jump by 23 percent after warm weeks in the period before 1914. In the later period we observe a much smaller increase of around 7.7 percent. For cold weeks, we observe no meaningful effect on digestive disease deaths.

Table A.3: Estimated effects across eight-week windows

DV:	**Log total deaths**		**Log infant deaths**		**Log infant mort. Rate**		**Infant mort. Rate**		**Log digestive deaths**	
Lowest temp bin	0.0704***	0.0772***	0.0139	0.0245*	0.0118	0.0253	0.237	0.165*	-0.0341*	0.0279
	(0.00772)	(0.0102)	(0.0118)	(0.0147)	(0.0118)	(0.0154)	(0.185)	(0.0925)	(0.0195)	(0.0338)
Highest temp bin	0.0416***	-0.00611	0.101***	0.0230	0.101***	0.0243	1.729***	0.149	0.283***	0.132***
	(0.00767)	(0.00814)	(0.0168)	(0.0181)	(0.0167)	(0.0184)	(0.288)	(0.0960)	(0.0337)	(0.0422)
Observations	2,498	1,900	2,101	1,900	2,101	1,808	2,101	1,808	2,289	1,876
R-squared	0.607	0.804	0.662	0.903	0.595	0.849	0.545	0.762	0.818	0.755

Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.