Temperature, Disease, and Death in London: Analyzing Weekly Data for the Century from 1866 to 1965

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Using novel weekly mortality data for London spanning 1866–1965, we analyze the changing relationship between temperature and mortality as the city developed. Our main results show that warm weeks led to elevated mortality in the late nineteenth century, mainly due to infant deaths from digestive diseases. However, this pattern largely disappeared after WWI as infant digestive diseases became less prevalent. The resulting change in the temperature–mortality relationship meant that thousands of heat-related deaths—equal to 0.9–1.4 percent of all deaths—were averted. These findings show that improving the disease environment can dramatically alter the impact of high temperature on mortality.

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 and to offer 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. It has long been recognized that mortality follows seasonal patterns and that these patterns have evolved over time. 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

The Journal of Economic History, Vol. 81, No. 1 (March 2021). doi: 10.1017/S0022050720000613

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We thank Vellore Arthi, Alan Barreca, Brian Beach, Robert Margo, Andrew Oswald, the Editor, William Collins, three anonymous referees, 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.

temperatures in the future.[1] Yet, our current understanding of the temperature–mortality relationship, particularly before the twentieth 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 nineteenth century.[2]

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 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.[3]

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 80 km outside of London. These data allow us to track temperatures 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.

[1] 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).

[2] 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 United States, see Fishback et al. (2011), which uses annual data from the 1930s; Anderson, Rees, and Wang (2020), which uses monthly data on infant deaths from 1910 to 1930; 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, Gasparrini, and Kinney (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 nineteenth century is Ekamper et al. (2009), which uses data from the Dutch province of Zeeland stretching back to 1855.

[3] 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.

Our analysis shows that, in the late nineteenth century and early twentieth century, both unusually cold and unusually warm weather were 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.

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 or 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 percent 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 0.9–1.4 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, Charles, and Rees (forthcoming), 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.[4] Yet, our results show that altering the disease environment, and specifically

[4] See Basu and Samet (2002) and Deschênes (2014) for reviews of this literature. On electrification and the use of air conditioning, see Chestnut et al. (1998), Ferreira Braga, Zanobetti, and Schwartz (2001), Curriero et al. (2002), Deschênes and Greenstone (2011), Barreca (2012), and Barreca et al. (2016). On the role of geographic mobility, see, for example, Deschênes and Moretti (2009) and Bohra-Mishra, Oppenheimer, and Hsiang (2014). On changes in time use, see Graff Zivin and Neidell (2014). There is also a separate adaptation literature looking at the impact of climate on outcomes other than mortality, such as productivity (e.g., Bleakley and Hong 2017).

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, digestive diseases alone accounted for 1.7 deaths per thousand in 1871, 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 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 substantially 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 twentieth 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 the third section, followed by the analysis in the fourth section, some counterfactual exercises in the fifth section, and concluding remarks in the final section.

BACKGROUND AND RELATED LITERATURE

In the middle of the nineteenth 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.[5] 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

[5] 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 (Hug, Sharrow, and You, 2017).

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 standards, this put London just above the national average (149 per thousand) and below more industrialized cities such as Birmingham and Manchester (Williams and Mooney, 1994).[6] Starting from this high level, infant mortality dropped to 132 per thousand births in 1900–1910, 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 percent of all deaths.[7] In this pattern, London was relatively similar to the average across British cities, though there has been some dispute about the similarity in the exact timing of the onset of the infant mortality decline.[8]

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 percent of all deaths), followed by bronchitis (13 percent). Cardiovascular diseases accounted for only about 6 percent of deaths, followed by pneumonia (5.4 percent) and whooping cough (3.6 percent). Cancer accounted for less than 3 percent of deaths. Among infants, the most important category was digestive diseases, which accounted for 13 percent of all deaths from 1876 to 1885. 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 percent of deaths), cancer (22 percent), and stroke (9 percent). Even for infants, digestive diseases were much less important, accounting for just under 3 percent of infant deaths (65 percent 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 to 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

[6] See also Lee (1991).
[7] Authors' calculations using the data described in the third section.
[8] See Woods, Watterson, and Woodward (1988), Lee (1991), and Williams and Mooney (1994).

et al. (2006), which examines mortality patterns in the twentieth century. Relative to their paper, our study includes several decades of data from the nineteenth century. This difference is important, because it means that our analysis begins before the sharp decline in infant mortality in London began (see Online Appendix Figure A.3). We also offer a richer analysis framework that captures the importance of lagged mortality effects and allows 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 centuries. 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.[9] He continued to write on the subject when he joined the 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.[10]

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, p. 360), 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

[9] Newsholme (1899).

[10] See Woods, Watterson, and Woodward (1989) for a useful review of the contemporary discussion of these topics.

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 have 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, ch. 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 nineteenth 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 (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.[11] However, the importance of nutrition has been challenged by Szreter (1988), among others, who instead emphasized the role of public health measures.[12] 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 to 1900 can be explained by infrastructure

[11] Harris (2004) also finds support for the role of nutrition over a longer time frame (1750–1914).

[12] 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) recognize the critical role played by sanitary measures in reducing digestive disease mortality.

investments.[13] 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 (e.g., Online Appendix Figure A.3).[14]

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 United States (Komisarow 2017) but not in others (Anderson, Charles, and Rees forthcoming), and the contribution of improvements in milk 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 who 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 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,

[13] A parallel literature on the role of water and sewer infrastructure exists for the U.S. See, for example, Troesken (2002), Cutler and Miller (2005), Ferrie and Troesken (2008), Beach, et al. (2016), Alsan and Goldin (2019), Anderson, Charles, and Rees (forthcoming), and Anderson, Rees, and Wang (2020).

[14] 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 tuberculosis.

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 to 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, Gasparrini, and Kinney (2014) looking at New York City, Åström et al. (2013) looking at Stockholm, and Ekamper et al. (2009) using data from the Dutch province of Zeeland. Of these, only the Ekamper et al. paper uses data stretching into the nineteenth century, though their data come 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.

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 London School of Economics 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 twentieth 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, pp. 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 (Hanlon, Hansen, and Kantor 2020) 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 Online Appendix A.2, we present supplementary results for 1981–2006.[15] 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.

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, and so on) or respiratory diseases (bronchitis, asthma, and so on).[16] 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

[15] 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.

[16] A list showing the specific causes that are included in each of the broad categories we study is available in the replication files provided on the journal website.

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 (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.[17] Certain disease categories, such as tuberculosis, which were difficult to diagnose, have been highlighted as particularly problematic (McKeown and Record 1962; Hardy 1994). However, the most important category for our analysis, diarrheal diseases, is "one of the least troublesome'' according to Hardy (1994, p. 486) 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) that were most likely to be confused with one another.

Our temperature data come from the Radcliffe Observatory in Oxford, 80 km 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 come partially from Greenwich Observatory and partly (after WWII) from Kew Gardens. Second, the Oxford data provide both average maximum and average minimum temperatures for each week, rather than just weekly mean temperatures. 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 the temperature there is unlikely to be substantially influenced by urban heat-island effects.[18] Dealing with the potential endogeneity created by urban heat-island effects is important

[17] Very few (less than 1 percent) 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.

[18] 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.

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.[19]

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 to 70, 70 to 75, 75 to 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 and 35, 25 and 30, or below 25. Online Appendix Figure A.1 shows the distributions of the minimum and maximum weekly temperatures during our sample period.

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 to 1939, while the third period covers the years just after WWII, from 1949 to 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.

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

[19] 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 Online Appendix A.2, we verify that similar results are obtained if we instead use temperatures from Greenwich and Kew Gardens in our analysis.

TABLE 1
TEMPERATURE BINS USED IN THE ANALYSIS

	Number of Weeks Falling into Bin by Period			
Bin Range (°F)	All Years	Pre-WWI 1866–1914	Interwar 1919–1939	Post-WWII 1949–1965
Min temp <25	412	242	83	87
Min temp 25–30	635	383	155	97
Min temp 30–35	881	497	225	159
Reference weeks	755	404	172	179
Max temp 65–70	616	351	144	121
Max temp 70–75	546	293	139	114
Max temp 75–80	382	213	95	74
Max temp >80	313	174	83	56
Total weeks	4,540	2,557	1,096	887

Source: Authors' calculations using temperature data from the Radcliffe Observatory, Oxford, UK.

weeks where the high temperature was never above 65°F and the low temperature never below 35°F treated as the reference category.

Our baseline empirical specification is

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

where $\ln y_{wt}$ 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 $TEMP_{wt}^{q}$ term is 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 quantifies 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).[20]

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.

[20] We examine humidity controls in the Online Appendix. Heavy fog affected mortality through pollution (Hanlon 2018). The fog data are drawn from Hanlon's paper.

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 Online 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 Deschênes (2014)) but differs from most existing studies in the economics literature, which instead uses 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, and disadvantages, such as limitations 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 Online 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.

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 to 1965. These data show a clear secular decline in the number of infant and digestive deaths over time, together with a reduction in the

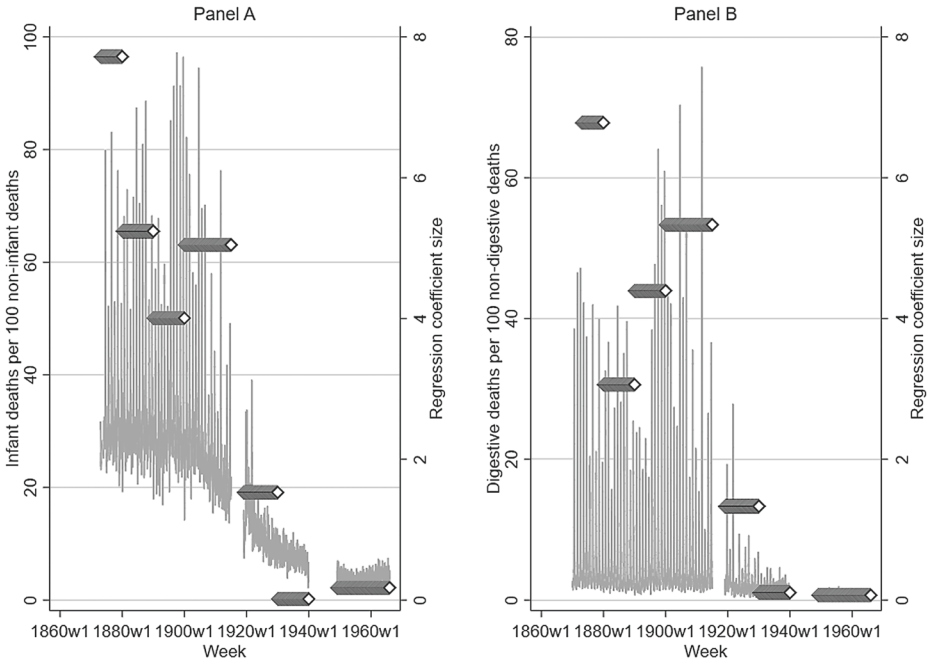

FIGURE 1
INFANT DEATHS PER 100 NON-INFANT DEATHS,
DIGESTIVE DEATHS PER 100 NON-DIGESTIVE DEATHS,
AND WARM-WEEK EFFECTS BY PERIODS

Notes: On the first *y*-axis in Panel A, the thin line plots infant deaths relative to total non-infant deaths (×100) over time. In Panel B, on the left-hand *y*-axis, the thin line plots digestive deaths relative to total non-digestive deaths (×100) over time. On the second *y*-axis (Panels A and B), the horizontal thick gray 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. Online Appendix Figure A.2 reports the corresponding results for the infant mortality rate.
Source: Authors' calculations.

week-to-week variability in the number of deaths. The pattern for infants is not driven by secular trends related to the fertility transition; Online Appendix Figure A.2 shows similar results for the infant mortality rate: 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.[21] 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 interwar 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]

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 die 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: 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 gray 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 first panel represents the impact of a temperature event on mortality in the week before the temperature event occurred, the second panel represents the effect in the week in which an event occurred, the third panel represents the effect in the following week, and so on.

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

[21] This can be thought of as estimated for the impact of heat on mortality contemporaneously and for seven lagged weeks and then taking the average across the eight coefficients obtained. See Online Appendix A.6 for details on the exact specification.

[22] The period from 1900 to 1914 could be thought of as a transition period from the high infant mortality regime of the late nineteenth century to the low infant mortality regime that existed after WWI (see also Online Appendix Figures A.3–A.5). We have chosen to include this in our pre-WWI sample, though in Online 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.

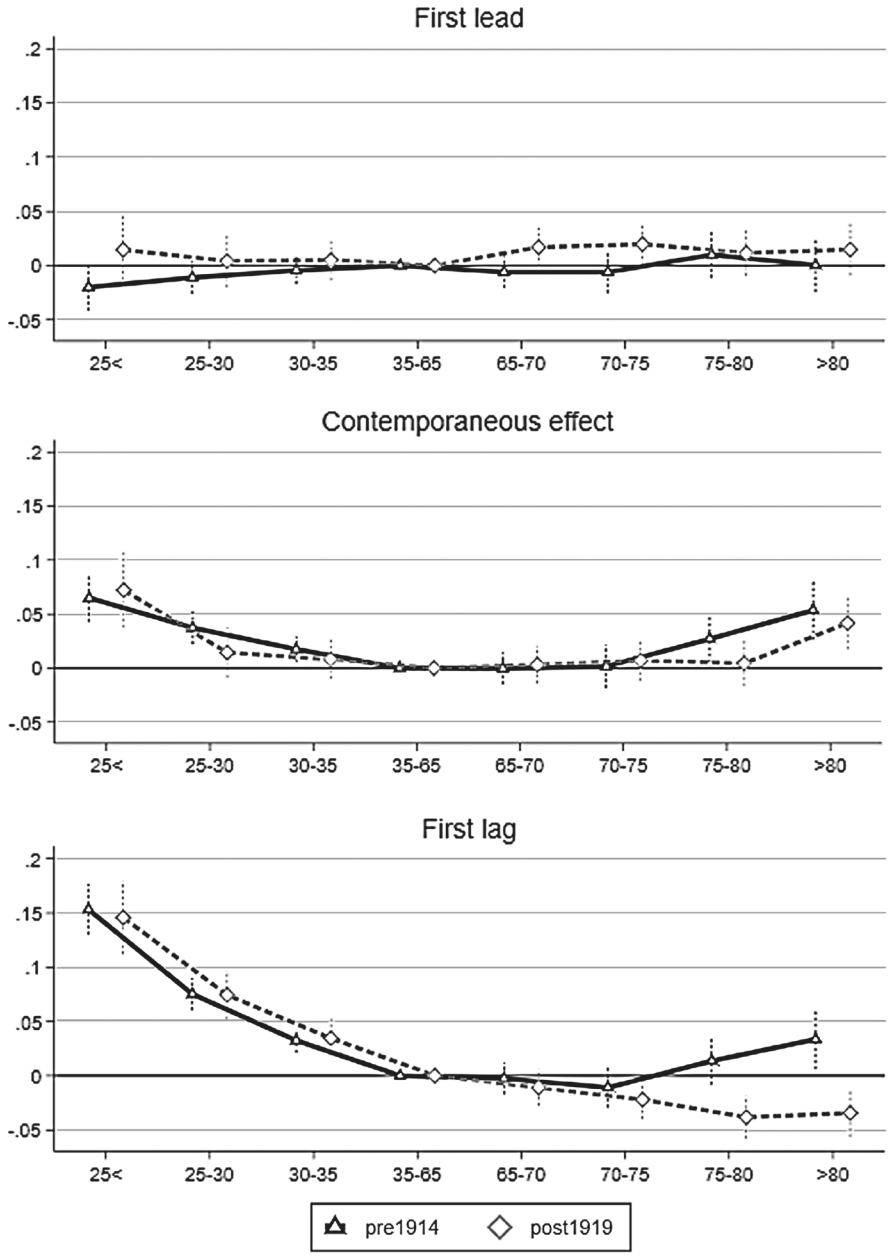

FIGURE 2
TEMPERATURE AND TOTAL MORTALITY BEFORE AND AFTER WWI

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

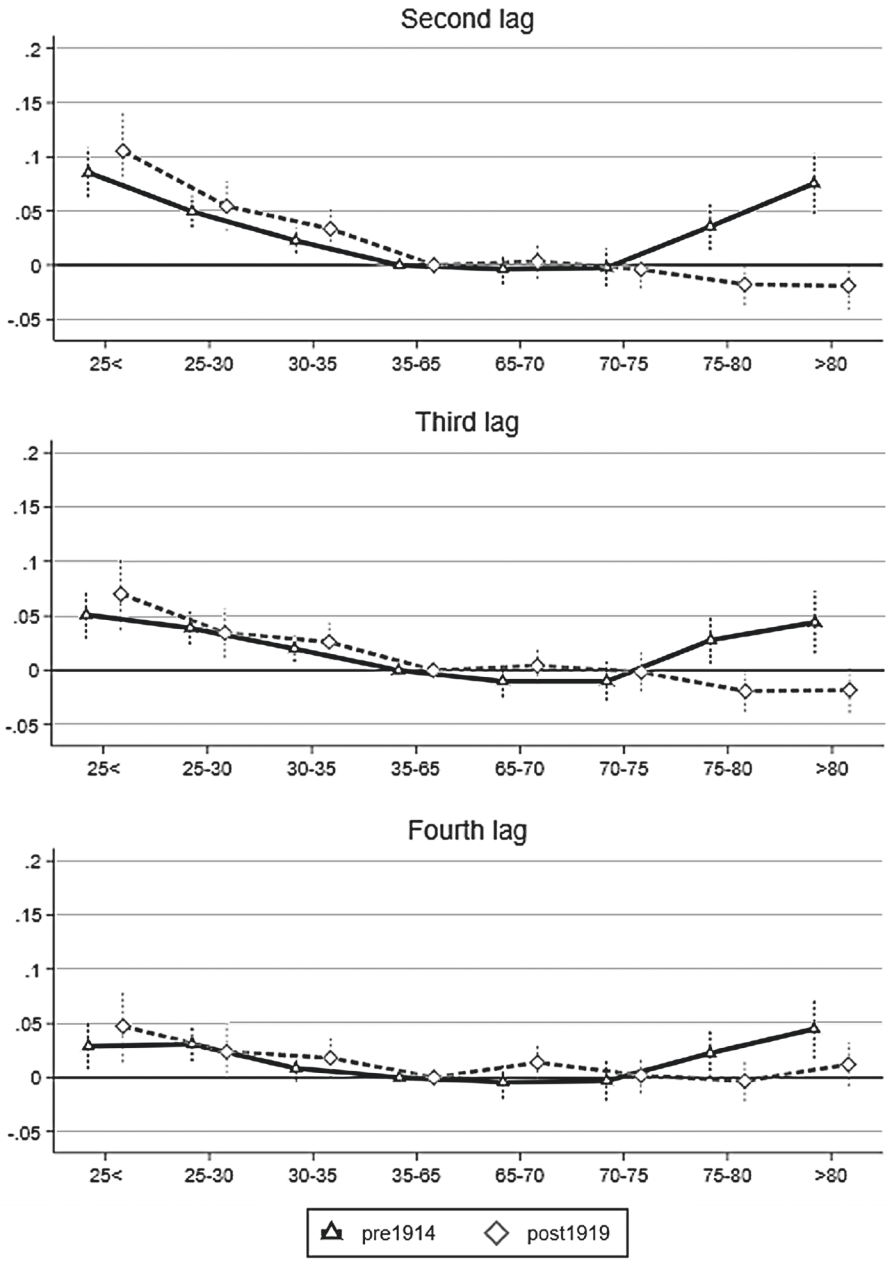

FIGURE 2 (CONTINUED)
TEMPERATURE AND TOTAL MORTALITY BEFORE AND AFTER WWI

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

Focusing on the pre-WWI sample (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 estimate this cold-week effect across the week in which the cold temperature is observed as well as the subsequent seven weeks, we obtain an average mortality increase of 7 percent across that eight-week period (see Online Appendix A.6).[24] There is also some indication that cold 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 0.075 (t-value = 5.24), implying an increase in total mortality of around 7 percent. Across an eight-week period starting with the week in which a temperature event occurred, the average effect of temperature falling into the warmest bin was a 4 percent increase in total mortality (see Online Appendix A.6).[25] 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 that 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 during both cold and warm weeks, though the cold-week effect is stronger in magnitude while the warm-week effect is relatively more "persistent."

The gray 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

[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.

[24] This 7 percent can be thought of as similar to taking the average across the q1 temperature bin coefficients estimated for the contemporaneous period and for lags 1–7 in Figure 2, though it is obtained using a regression approach, explained in Appendix A.6, so that we are also able to generate confidence intervals.

[25] As explained in footnote 24, this 4 percent can be thought of as similar to taking the average across the estimated coefficients for the contemporaneous period and lags 1–7 for temperature bin q8 in Figure 2.

averaged across the week in which cold weather occurred and the subsequent seven weeks (see Online 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 = 3.36), 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 Online 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, Online 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 percent, 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.

We have undertaken a number of robustness checks on these results. These checks, reported in Online 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 Online 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

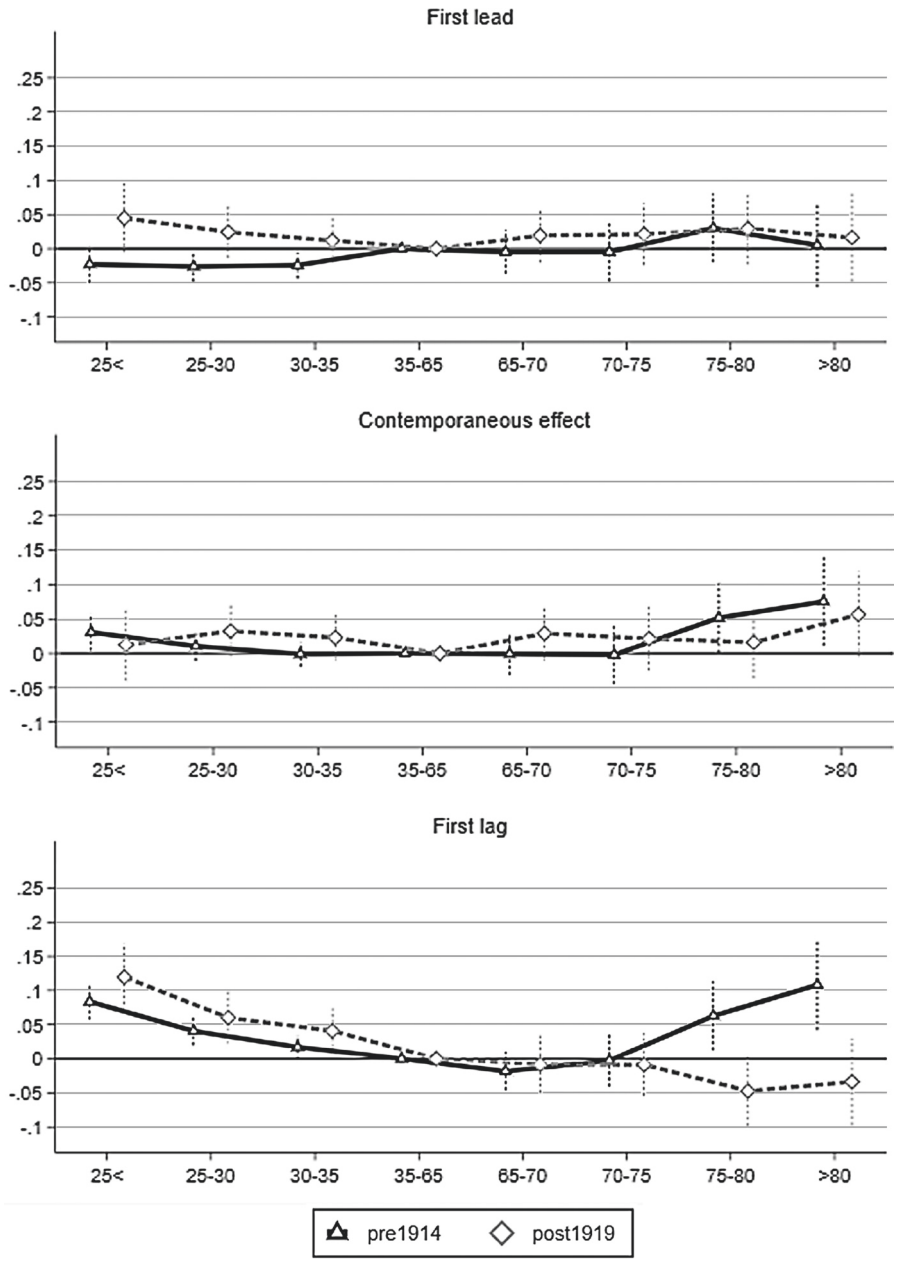

FIGURE 3
TEMPERATURE AND INFANT MORTALITY BEFORE AND AFTER WWI

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

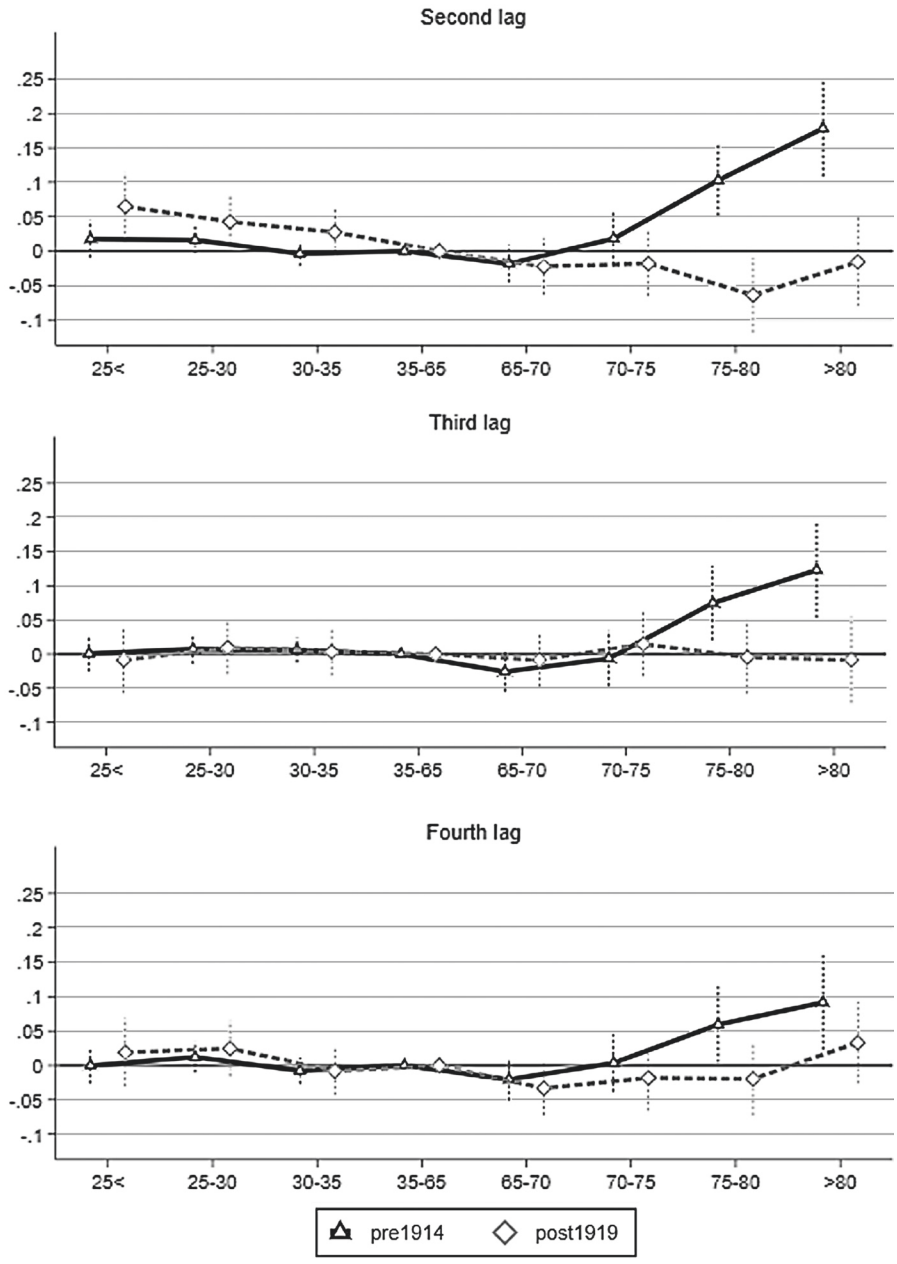

FIGURE 3 (CONTINUED)
TEMPERATURE AND INFANT MORTALITY BEFORE AND AFTER WWI

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

the temperature observation and peaks two weeks later with an estimated coefficient of 0.18 (t-value = 4.99), 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 Online Appendix A.6). However, after 1919, this effect essentially disappears. 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.

As we did for total mortality, we have also examined the robustness of the infant mortality results. These robustness checks, in Online 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, and so on. In addition, these results naturally raise questions about the relationship between temperature and births. Online 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.

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

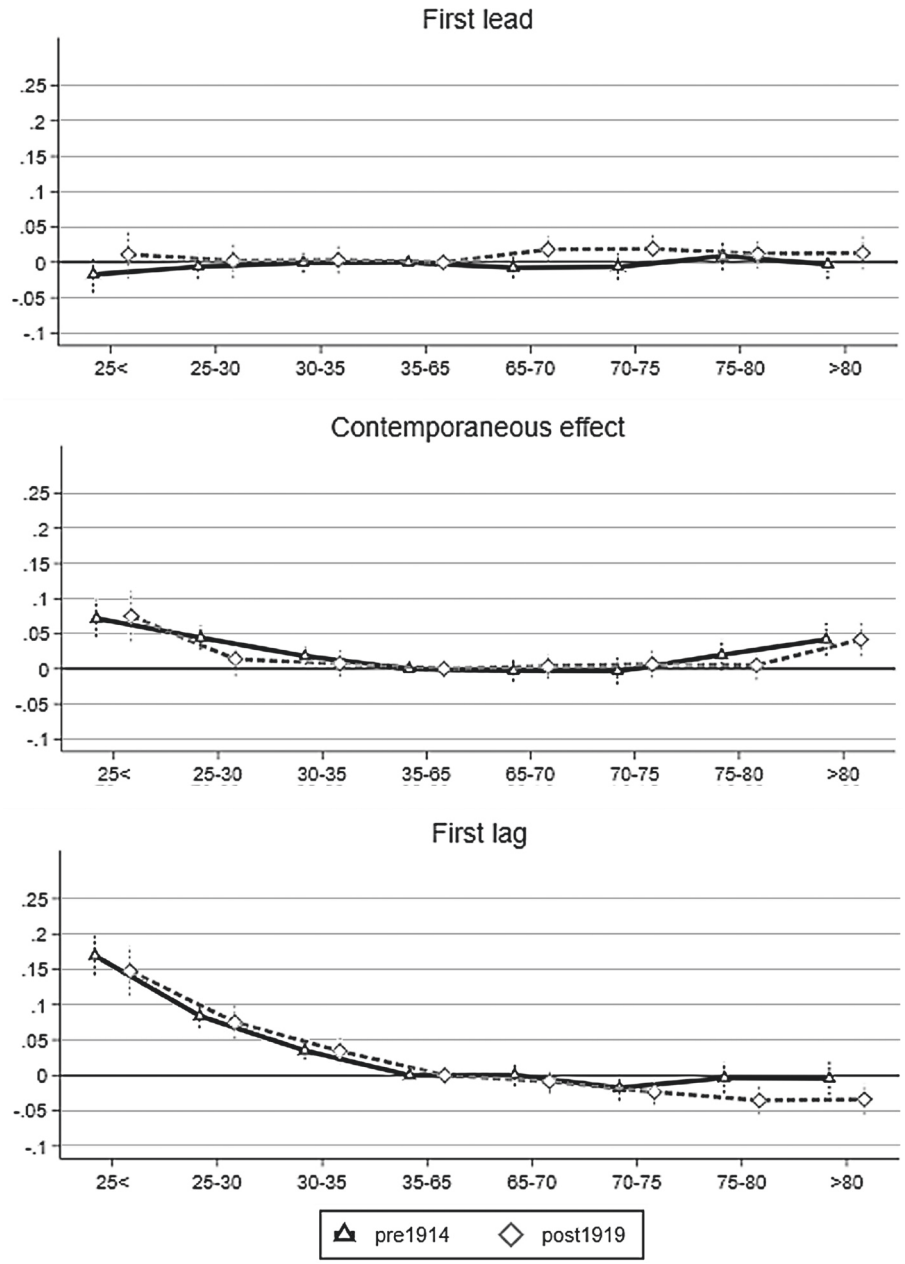

FIGURE 4
TOTAL NON-INFANT DEATHS BEFORE AND AFTER WWI

Notes: This figure shows the non-parametric relationship between temperature and log (total – infant) mortality for the first 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 to 1939 and 1949 to 1965. The omitted reference temperature bin is $q = 4$ or 35–65°F.
Source: Authors' calculations.

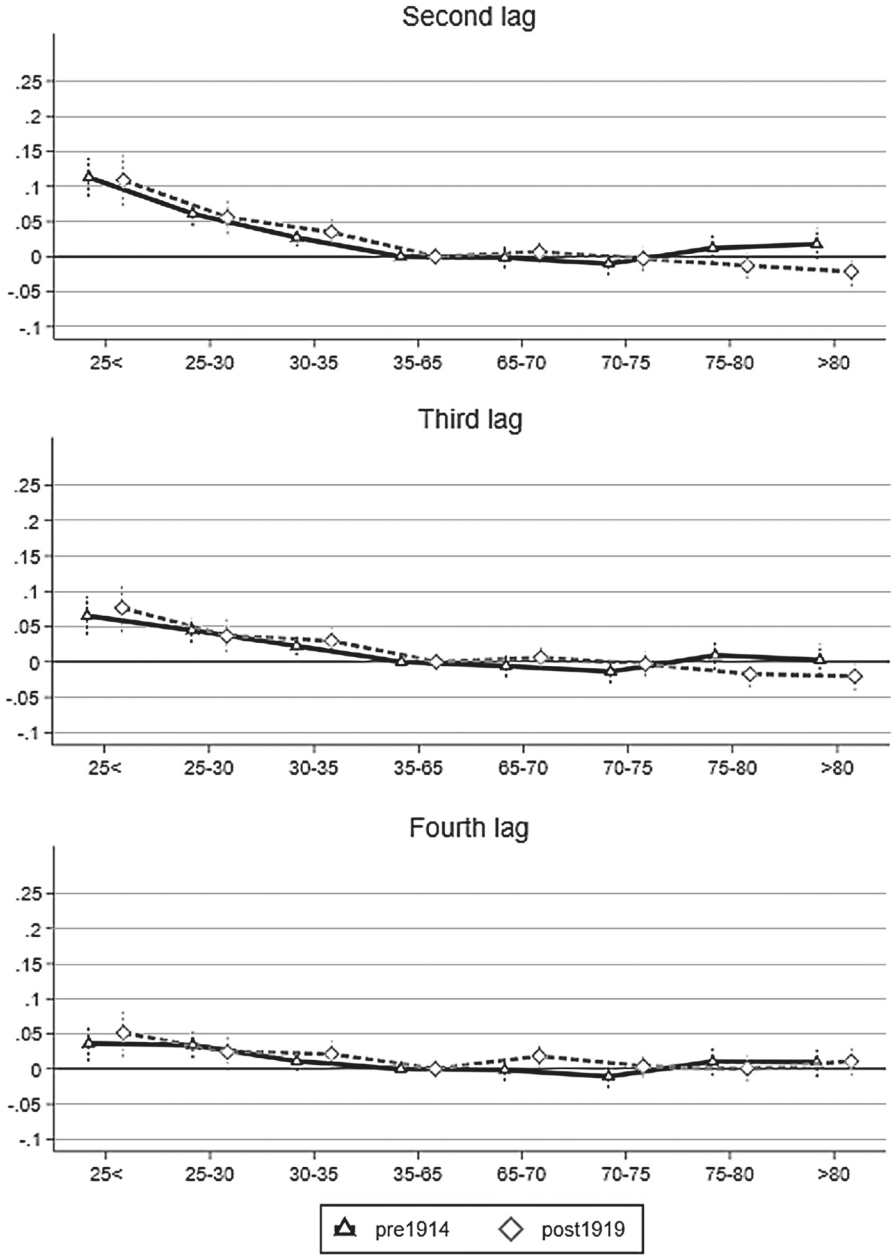

FIGURE 4 (CONTINUED)
TOTAL NON-INFANT DEATHS BEFORE AND AFTER WWI

Notes: This figure shows the non-parametric relationship between temperature and log (total – infant) mortality for the first 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 to 1939 and 1949 to 1965. The omitted reference temperature bin is $q = 4$ or 35–65°F.
Source: Authors' calculations.

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 Online Appendix A.5. The results in that appendix show that, for both total and infant mortality, warm weeks are more deadly when they occur consecutively, particularly when it is 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, though note that our cause-of-death analysis looks at deaths across all age groups.

Figures 5 and A.22 plot the impact of temperature on all-age mortality due to digestive diseases. As in the previous figures, the dark lines show estimates from the pre-WWI sample, while the gray 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 (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.

Finally, we break down our data to look at the impact of temperature across a variety of more detailed cause-of-death categories. This analysis uses data from 1866 to 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

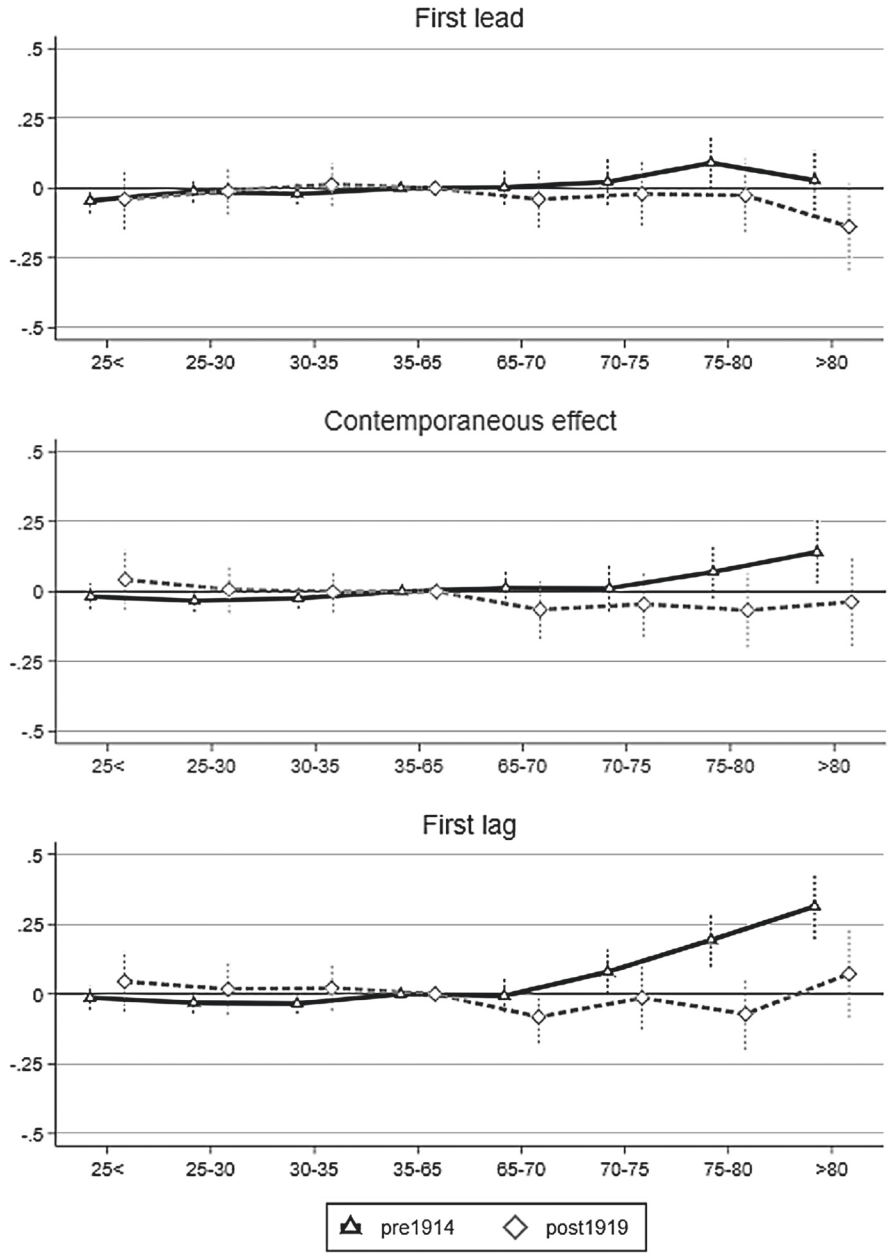

FIGURE 5
TEMPERATURE AND DIGESTIVE MORTALITY BEFORE AND AFTER WWI

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

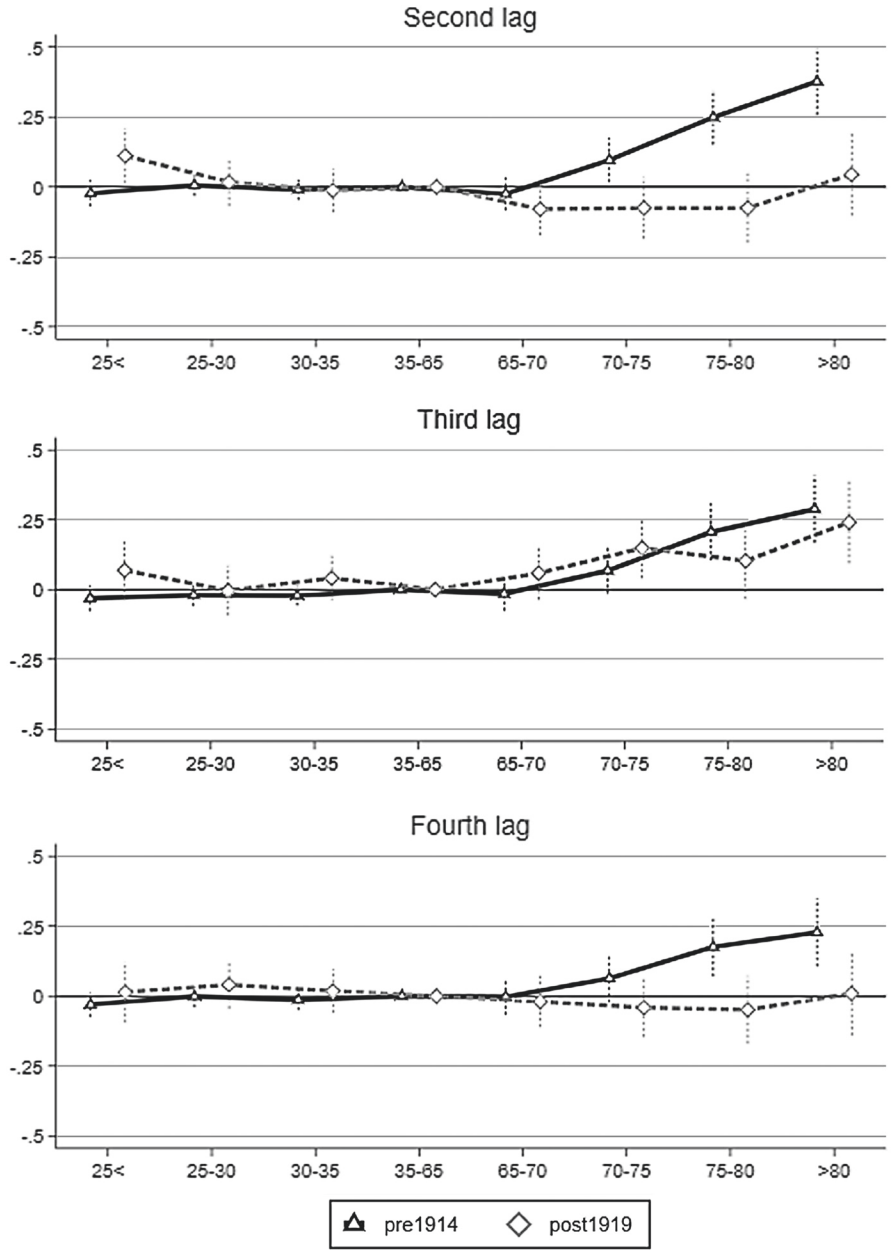

FIGURE 5 (CONTINUED)
TEMPERATURE AND DIGESTIVE MORTALITY BEFORE AND AFTER WWI

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

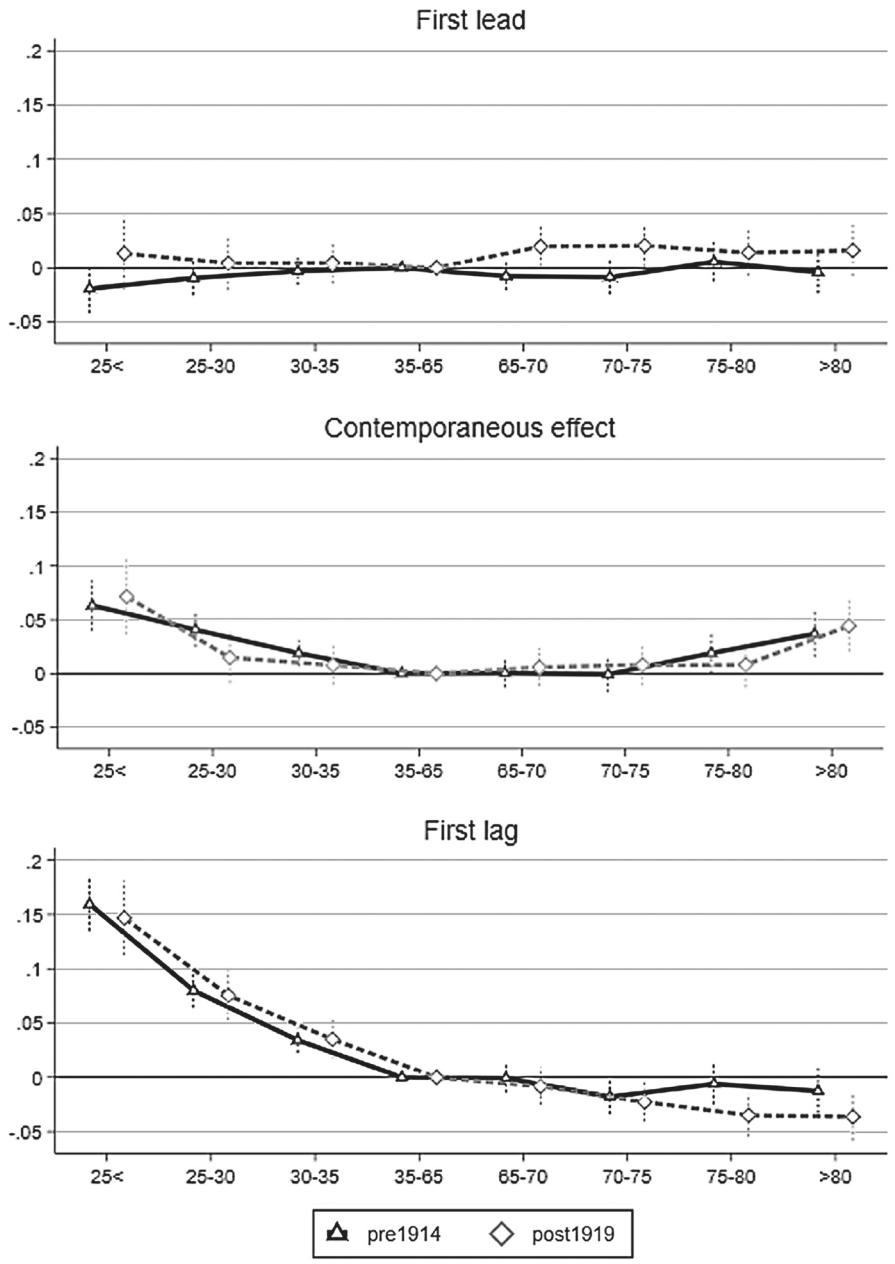

FIGURE 6
TEMPERATURE AND TOTAL NON-DIGESTIVE MORTALITY BEFORE AND AFTER WWI

Notes: This figure shows the non-parametric relationship between temperature and log non-digestive mortality for the first 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 to 1939 and 1949 to 1965. The omitted reference temperature bin is $q = 4$ or 35 to 65°F.
Source: Authors' calculations.

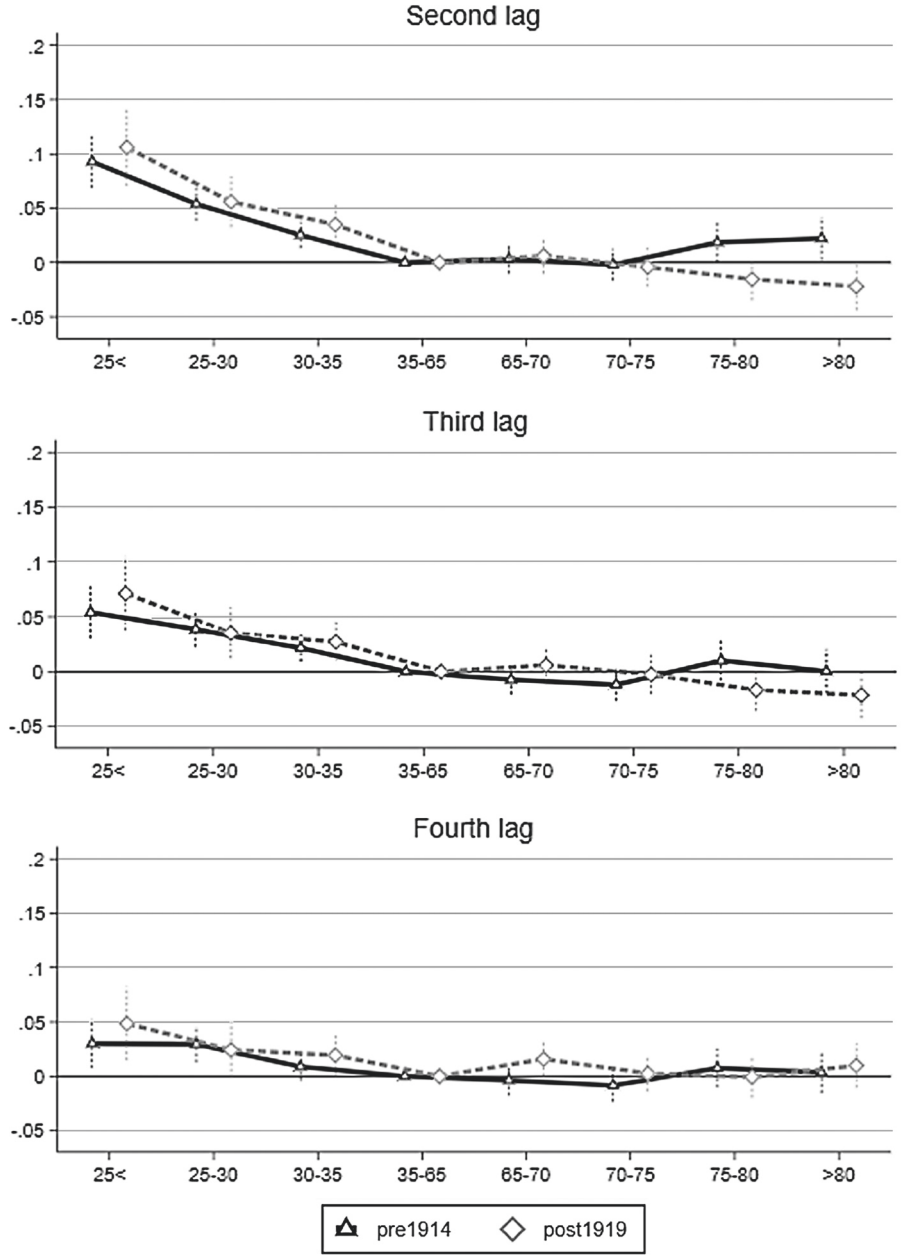

FIGURE 6 (CONTINUED)
TEMPERATURE AND TOTAL NON-DIGESTIVE MORTALITY BEFORE AND AFTER WWI

Notes: This figure shows the non-parametric relationship between temperature and log non-digestive mortality for the first 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 to 1939 and 1949 to 1965. The omitted reference temperature bin is $q = 4$ or 35 to 65°F.
Source: Authors' calculations.

TABLE 2
EFFECT OF HIGH-TEMPERATURE WEEKS
ON DIFFERENT CAUSES OF DEATH BEFORE WWI

	Dependent Variable: Log Deaths					
Cause:	Digestive	Bronchitis	Cancer	Cardio	Childbirth	Diphtheria
Low temp	–0.0276	0.199***	0.00315	0.0719***	0.0489	0.0248
	(0.0193)	(0.0186)	(0.0102)	(0.00971)	(0.0316)	(0.0249)
High temp	0.257***	–0.0590***	0.0144	0.00621	–0.0311	0.0306
	(0.0328)	(0.0152)	(0.0109)	(0.00910)	(0.0315)	(0.0264)
Obs.	2,328	2,328	2,328	2,328	2,326	2,327
R^2	0.817	0.884	0.882	0.814	0.297	0.812
Cause:	Homicide	Measles	Old Age	Pneumonia	Respiratory	Scarlet Fever
Low temp	0.0134	0.129***	0.0932***	0.130***	0.164***	–0.0704**
	(0.0458)	(0.0491)	(0.0137)	(0.0153)	(0.0211)	(0.0297)
High temp	–0.00846	–0.0663*	0.0206	–0.0386***	–0.0344	–0.0543*
	(0.0442)	(0.0382)	(0.0150)	(0.0135)	(0.0219)	(0.0279)
Obs.	1,640	2,328	2,328	2,328	2,328	2,316
R^2	0.101	0.411	0.533	0.803	0.720	0.824
Cause:	Suicide	Tuberculosis	Typhus	Whooping Cough		
Low temp	0.0373	0.0394***	–0.160***	0.0196		
	(0.0330)	(0.00805)	(0.0618)	(0.0299)		
High temp	–0.0323	0.0285***	0.0628	–0.0272		
	(0.0329)	(0.00767)	(0.0690)	(0.0256)		
Obs.	2,322	2,328	781	2,328		
R^2	0.311	0.662	0.648	0.699		

Notes: Robust standard errors in parentheses. Data cover 1866–1914. Estimates reflect the impact of a week with temperatures falling into the highest bin (>80°F) in the week in which the temperature occurred or the seven following weeks. ***$p < 0.01$, **$p < 0.05$, *$p < 0.1$.
Source: Authors' estimates using Registrar General's data.

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.

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 such as 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.

COUNTERFACTUALS

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 80°F, while cold effects are those associated with minimum temperatures below 35°F, 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 59,773 excess deaths in 1876–1914, or 1.9 percent 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 are, 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: equal to 9.2 percent of total deaths before WWI, 13.5 percent in the interwar period, and 8.4 percent after WWI. Overall, temperature-related deaths were a major component of mortality, accounting for 11.2 percent of all deaths in the pre-WWI period, 13.7 percent in the interwar period (when influenza deaths spiked), and 7.7 percent 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

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	59,773	2,395	–4,398
(share of all deaths)	0.019	0.002	–0.007
Cold related	283,263	148,394	56,329
(share of all deaths)	0.092	0.135	0.084
Total temp-related deaths	343,036	150,789	51,931
(share of all deaths)	0.112	0.137	0.077
Panel B: Estimated Heat-Related Deaths Imposing the Pre-WWI Temp–Mortality Relationship			
Heat related		12,493	5,053
Diff vs. Panel A		10,098	9,451
(diff as share of all deaths)		0.009	0.014
Implied additional deaths per year		484	589
Panel C: Impact of 1.5°C Increase Using the Temp–Mortality Relationship Observed in Each Period			
Heat-related deaths	33,011	880	–1,808
Cold-related deaths	–86,317	–45,159	–14,385

Source: Authors' estimates using Registrar General's data.

conservative counterfactual estimates, since our estimates incorporate the baseline reduction in overall mortality observed during each period. That is, 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 10,098 heat-related deaths in the interwar period and 9,451 deaths in the decades after WWII, equal to a 0.9–1.4 percent 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 twentieth century.

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°C, a common reference level used in the climate change literature.[26] These results show that had this temperature increase occurred in the pre-WWI period, we would have expected an additional 33,011 heat-related deaths, equal to a 1 percent increase in total mortality. This would have been more than offset by a reduction of 86,317 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, Charles, and Rees (forthcoming), 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, Watterson, and Woodward 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 takeaway 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 peaks 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

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

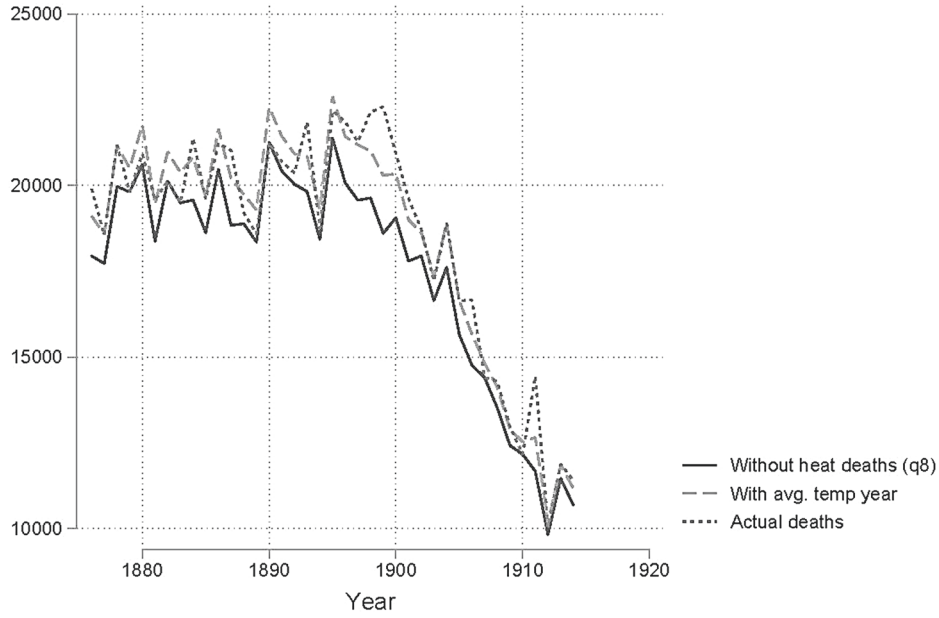

FIGURE 7
ACTUAL AND COUNTERFACTUAL INFANT DEATHS

Sources: Authors' calculations based on Registrar General's data. See text for further details.

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.

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.

CONCLUSIONS

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 allows us to provide quantitative estimates of how this change affected mortality in London. Our results suggest that deaths would have been higher by 0.9–1.4 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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