Climate, Mortality, and Adaptation: A Century of Weakly Evidence from London*

W. Walker Hanlon — NYU Stern School of Business and NBER

Casper Worm Hansen — University of Copenhagen

PRELIMINARY

February 6, 2019

Abstract

Using novel weekly climate and mortality data from 1866-2006, we study the changing relationship between temperature and mortality in London and quantify the implications of these changes for the effect of rising temperature on health. Our results show that warm weeks were much more deadly in the period before WWI due to a high burden of digestive disease deaths among infants. After 1920, public health improvements nearly eliminated these effects. As a consequence of this shift in the temperature-mortality relationship, over 10,000 deaths that would have been caused by rising temperatures in the interwar period were averted, as well as an additional 4,800 deaths in the period from 1949-65. These results reveal the important role that public health infrastructure improvements can play in helping cities adapt to rising temperatures.

*Funding for this project was provided by National Science Foundation CAREER Grant No. 1552692.

Author contact information: Hanlon, NYU Stern School of Business, Henry Kauffman Management Center 7-160, 44 West 4th St., New York, NY 10012; whanlon@stern.nyu.edu. Hansen, University of Copenhagen, Department of Ecconomics; casper.worm.hansen@econ.ku.dk

1 Introduction

The effects of climate change depend crucially on how humans adapt to rising temperatures. Recent research has studied a number of potential adaptation strategies, including electrification and the use of air conditioning (Chestnut et al., 1998; Braga et al., 2001; Curriero et al., 2002; Deschênes and Greenstone, 2011; Barreca, 2012; Barreca et al., 2016), geographic mobility (Deschenes and Moretti, 2009), and changes in time use (Graff Zivin and Neidell, 2014). One form of adaptation that has attracted less attention is alteration of the underlying disease environment in a location through public health improvements. This omission is notable given the central role that public health interventions, such as the provision of clean water, safe food, and sanitation, play in reducing overall mortality (Cutler and Miller, 2005; Ferrie and Troesken, 2008; Alsan and Goldin, ming). Our results show that the disease environment, specifically the presence of infectious diseases of the digestive system, can have powerful effects on the impact of rising temperatures on health. This evidence helps us better understand and quantify how, and why, the effects of rising temperatures will differ in developing versus developed countries. In addition, these findings suggest that investments that reduce the infectious disease burden, such as improvements in water, sanitary, and public health infrastructure, have an important role to play in climate change adaptation. This will be particularly true in developing countries, where the infectious disease burden remains high.

To study these issues we take advantage of a setting where we can observe at a high level of detail the relationship between temperature and mortality across a period in which the underlying disease environment in a location changed substantially: London from the middle of the 19th century through 2006. At the beginning of our study period, London was an extremely unhealthy environment with a high infectious disease burden, on par with very poor modern developing countries. By the end of our study period, investments in public health infrastructure, as well as medical advances, rising income, and improving nutrition, made London a much healthier place to live. Thus, this setting offers the opportunity for understanding how the relationship between temperature and mortality evolves as a location develops.

Our study takes advantage of uniquely detailed high-frequency mortality data that allow us to assess how the relationship between temperature and mortality evolved across this period, as well as the role of the disease environment in influencing the health effects of temperature. Our mortality data are unique in that they are observed at relatively high frequency (weekly) starting in 1866 and extending, with some breaks, until 2006. For much of this period we are also able to observe mortality broken down by age group and cause of death. We combine these with one of the longest continuously-observed single-site weather series in the world, taken from the Radcliff Observatory in Oxford, as well as additional rich weather data from the Royal Observatory at Greenwich. These data allow us to track temperature as well as other weather conditions in a consistent way over our study period. Together, these data series allow us to look at how high temperatures interact with different infectious diseases and how reductions in the infectious disease burden across the study period influenced the effect of high temperature events overall and for different age groups. Using high-frequency mortality and climate data allows us to identify the mortality effects of temperature using a fairly simple identification 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 results show that while cold weeks were associated with more deaths throughout the study period from 1866-1966, warm weeks were much more deadly in the period before WWI due to a high burden of digestive disease deaths among infants.

Our study contributes to a long line of work examining the relationship between temperature and mortality. Reviews of this literature are provided by Basu and Samet (2002) and Deschenes (2014). Most of the papers in this literature use data from modern developed countries.[1] Fewer studies use data from developed countries before the 1960s or from developing locations. Data availability is typically a major impediment in these settings. As a result, our understanding of the relationship between temperature and mortality in developing settings remains limited, as does evidence on how this relationship evolves as countries develop. Our study aims to fill in this gap in our existing knowledge.

The possibility that the level of development may substantially alter the relationship between temperature and mortality is suggested in existing work by Hajat et al. (2005), which looks at this relationship in London, Sao Paolo and Delhi using data from 1991-1994. They find stark differences between the impact of high temperatures in these locations. For example, in New Delhi a high proportion of deaths (48%) occurred among children younger than 15, while in London only 1% of the deaths associated with high temperatures occurred among this population. A number of forces might be behind these differences. For example, Barreca et al. (2016) provide evidence that air conditioning can play an important role in reducing heat-related mortality. As a result, it is difficult to separate out the effect of the disease environment in cross-country study designs, such as the approach used in Hajat et al. (2005), from other factors such as the availability of air conditioning (particularly in hospitals or elderly homes) or the institutional environment. Our setting allows us to consider the

[1] Examples using U.S. data include (Davis et al., 2003), Deschenes and Moretti (2009) and Deschênes and Greenstone (2011).

effect of the disease environment where, for most of our study period, air conditioning was essentially absent.

Our study is closely related to an important recent study by Barreca et al. (2016) using historical U.S. data. However, our setting offers some important advantages relative to their study. One important advantage is that we can track mortality by detailed cause-of-death across our full study period. Because of the limitations of historical U.S. mortality data, their cause of death results are based on much less detailed categories and are only available after 1959. Because this falls after many of the most important public health improvements, such as clean water, had already been implemented in the U.S., it is not possible for them to examine the impact of these improvements on the temperature-mortality relationship. Similarly, their mortality data does not include age information prior to 1959, so they cannot study how these improvements affected the impact of temperature across different demographics. Second, the higher temporal frequency of our data allow us to study patterns, such as the lag structure of effects, that cannot be examined in their data. Third, we have more detailed climate data which includes factors such as rainfall and humidity which are not present in their data. Research by Barreca (2012) suggests that controlling for other factors, such as humidity, is important.

2 Background

In the middle of the 19th century, when our study begins, Londoners faced an extremely high disease burden, comparable to some of the worst disease burdens faced by poor urban residents in developing countries today. A variety of factors, including lack of access to clean water, poor sewage infrastructure, unsafe food, and ineffective medical care, combined to generate very high rates of mortality. Much of this mortality was concentrated in young children, and it is useful to focus on this group for comparison purposes because, as we will see, this group plays a central role in the relationship between temperature, mortality, and the disease environment. In 1876-1885, near the beginning of our study period, out of every thousand children born in London, 152 died before age one; infant mortality accounted for one quarter of all deaths. 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. As a point of comparison, 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.[2] Thus, in terms of infant mortality

[2]See Hug et al. (2017).

London during our study period covers a range that spans from some of the most unhealthy modern developing environments to levels comparable to modern developed countries.

This enormous decline reflected a wide variety of factors, including improved sanitation, water quality, nutrition, medical care, and housing. These factors, which were particularly important in the fight against infectious diseases, are reflected in dramatic shifts in the most important causes of death. 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 percent of deaths, and cancer just under 3 percent. Among infants the most important category was digestive diseases, which accounted for 13 percent 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). Because we have access to high-frequency data broken down by age and cause-of-death, our analysis is able to examine how these changes influenced the impact of temperature on mortality.

3 Data

Most of the mortality data used in this study were digitized from reports produced by the Registrar General's office. The data cover 4,540 weeks stretching from the beginning of 1866 to the end of 1965, with breaks in 1914-1918 for the First World War and 1939-1948 for the Second World War. Overall, this data set includes over 350,000 observations. We supplement our historical data with more recent weekly mortality data covering 1981-2006 provided by the U.K. Office of National Statistics (ONS).[3]

Our historical data were collected by a network of trained local officials (Registrars). Particular effort was put into the mortality data because this was an interest of early Registrar Generals such as Dr. William Farr, and even more so in London. As a result, the death statistics are considered to be the "shining star of Victorian civil registration" (Woods, 2000). Our data are structured as a time-series covering a consistent geographic area that encompasses all of modern Central London.[4] In each week, we observe the number of deaths

[3]Our historical data come from a consistent geographic area covering all of central London. The ONS data cover the larger Greater London Administrative Region.

[4]This area is somewhat smaller than the current Greater London administrative area, which was estab-

in each cause-of-death by age-group cell for the city as a whole.[5] The mortality data include breakdowns of deaths by age group, though with some changes over time in the reported age groups. 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 1876.

In our historical 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 reasonable accurate, even early in our study period. The causes of death categories change substantially after WWII, so when looking at causes of death we focus on data before 1940.

We use two sources of weather data in this study. Our main source of temperature data comes from the Radcliffe Observatory in Oxford. This observatory, located about 80km outside of central London, provides consistent continuous daily maximum and minimum temperature readings across the full study period. To construct our primary explanatory variable, we collapse these to weekly maximum and minimum temperature readings and then classify weeks into different temperature bins. Specifically, we construct bins identifying weeks with minimum temperatures below 25 degrees F, from 25-30, and from 30-35 as well as weeks with maximum temperatures from 65-70, 70-75, 75-80 or above 80 degrees F. These bins are chosen to reflect the range of temperatures observed in London while allowing sufficient observations in each bin to obtain precise estimates. Our reference category is the set of mild weeks where neither the minimum or maximum temperatures fall into one of these bins.

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. The third period covers

lished in 1966, but much larger than the City of London. This administrative changes in 1966 motivates our decision to end the study period in 1965.

[5]While some 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.

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 1918-1939	Post-WWII 1949-1965	Modern 1981-2006
Min. temp < 25	483	242	83	86	72
Min. temp 25-30	811	383	154	99	175
Min. temp 30-35	1,139	494	224	157	264
Reference weeks	1,020	407	174	180	259
Max. temp 65-70	785	351	144	121	169
Max. temp 70-75	707	293	139	114	161
Max. temp 75-80	516	213	95	74	134
Max. temp >80	435	174	83	56	122
Total weeks	5,896	2,557	1,096	887	1,356

Note: Temperature observations are in degrees Fahrenheit.

the years just after WWII, from 1949-1965 while the last "modern" period, which uses the mortality data provided by ONS, covers 1981-2006.[6]

An important point to take from Table 1 is that we can observe temperature rising throughout our study period. For example, the ratio of the number of weeks in the top temperature bin to the bottom temperature bin increase from 0.61 in the pre-WWI period to 1.04 in the modern period. Similarly, the ratio of weeks in the top three bins relative to the bottom three bins rises from 0.61 in the pre-WWI period to 0.69 in the interwar period, 0.71 in the decades just after WWII, and 0.82 in the modern period. Later we will directly estimate the implications of this rise for mortality in London.

We also draw on newly-digitized weekly weather data from the Greenwich Observatory, just East of Central London covering the period from 1866 up to WWII and data from Kew Gardens, just West of Central London for 1849-66. These data include weekly mean temperatures as well as additional weather information–rainfall, humidity and barometric pressure–that provide useful control variables. A comparison of the three data sources shows that the Oxford temperature data track variation in the data from Greenwich and Kew Gardens closely. However, we prefer to use temperature data from Oxford in our main analysis for three reasons. First, this series is reported consistently across the full study period. Second, the Oxford series is unlikely to be influenced by urban heat island effects, which is a potential concern when relying on temperature measures from London. Avoiding these effects allows us to attribute long-run temperature trends to global climate change, rather than the expansion of the London urban area. Third, the Oxford series contains

[6]The gap in coverage from 1965-1981 arises because hard-copy reports end during this period and the ONS has not been willing to construct weekly series from their records.

weekly maximum and minimum temperatures, allowing us to identify weeks with very cold and warm weeks, while the Greenwich series only contain weekly average temperatures.

4 Empirical Specification

Our baseline empirical specification assesses the non-parametric temperature-mortality relationship using the following lead-lag model:

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

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 $TEMP_{wt}^q$ terms are a set of indicator variables, taking on the value one if the weekly temperature is in the qth temperature bin. The omitted reference weeks (i.e., reference bin $q = 4$) are the ones that do not contain weekly minimum temperatures below 35 degrees F or weekly maximum temperatures above 65 degrees F. As also mentioned in the previous section, $q = 1$ is the lowest temperature bin defined as weeks with minimum temperatures below 25 degrees F, while $q = 8$ is the highest temperature bin with weeks containing maximum temperatures above 80 degrees F.

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". Thus, these coefficients describe the leading, contemporaneous, or lagged relationship between temperature and mortality across a range of temperature bins. 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 exogenous weather controls (X_{wt}), including humidity, precipitation, and an indicator for weeks with heavy fog.[7] The error term ε_{wt} allows for arbitrary heteroskedasticity.

Estimating equation (1) allows us to tests for "pre-trends" by checking if there are any mortality differences across temperature bins for all m leads ($\hat{\alpha}_j^q = 0\ \forall j < 0$); that is, all estimates of the lead temperature bins should *not* be statistically significant different from zero. If supported, this pre-trend (or falsification) test simply reveals that future temperature changes are *not* able to predict mortality changes in the present nor past. The estimated temperature-bin coefficients in the k lags (α_j^q's $\forall j \geq 0$) provide insights into the mortality

[7] Heavy fog affected mortality by increasing pollution levels. See Hanlon (2018).

dynamics of temperature changes.

An important difference between our work and previous studies within the economics literature is that we use higher-frequency time-series data. Most studies within economics utilize panel data with lower temporal frequency. Our approach is more similar to studies within the public health literature which also use a high-frequency time-series approach, though typically with daily data over short time spans in modern developed countries (see Deschenes (2014) for a review of this literature). This difference comes with both benefits and drawbacks. For example, focusing on only one location reduces the variety of climate conditions observed in our study and limits the generalizability of our results into starkly different environments, such as the tropics. However, it also means that we are able to study the impact of development on the temperature-mortality relationship among a population that remains relatively similar over time. We are also able to examine the lag structure of effects and consider the impact of repeated events. Overall, we view the high-frequency time-series approach used here as complementary to the panel data approach used in existing studies such as Barreca et al. (2016).

5 Results

This subsection reports estimates of the temperature-mortality relationship for both total and infant deaths. Because we are interested in how these effects change over time, we partition our data into an early and a late period. For our main results we use WWI as the cutoff between these periods, though we have also considered alternative cutoff dates.

Our baseline specification includes $m = 5$ leads, $k = 7$ lags, along with the current effect ($j = 0$). We consider seven lags because for most series the effects of temperature on mortality dies out within seven weeks. Our choice of five leading weeks is somewhat arbitrary, but this length seems like a reasonable number of weeks to establish the lack of pre-trends. Since each lead/lag includes seven temperature-bin estimates, these configurations imply that we end up estimating 91 coefficients ($\alpha^1_{-5}, .., \alpha^8_7$) for each mortality outcome. Such results are not well-suited for being presented in normal regression tables, so we present our estimated coefficients and their corresponding t-statistics visually, using event-study style surface plots. These provide a useful way to describe the broad patterns reflected in our coefficient estimates. To augment these we also include the corresponding 2D plots for each lead or lag, which make it easier to assess the magnitude and statistical significance of individual coefficient estimates.

Panel A of Figure 1 starts by plotting estimates for the temperature bins for each lead and lag for log total deaths before WWI. One horizontal axis shows the leading and lagged effect of temperature: "$week = -5$, for example, " indicates five weeks before a current temperature change and "$week = 7$" indicates seven weeks after. The other horizontal axis reports the different temperature bins. Panel B presents the t-statistics that correspond to the coefficients described in Panel A and Figure 2 presents the corresponding 2D plots for each lead and lag.

The first feature to note in this graph is that there is no evidence that temperature in a week is systematically related to mortality in previous weeks: the estimated lead effect coefficients are always small and mostly statistically significant at conventional levels. This is true across all of the temperature bins that we study. This tells us that our identification strategy is working well.

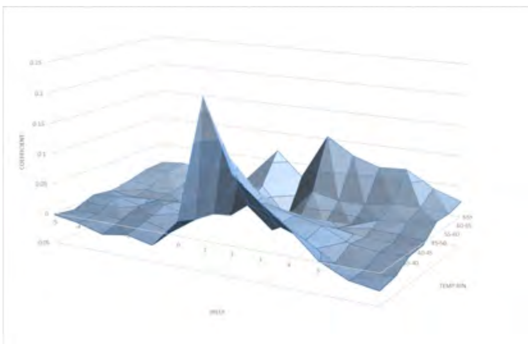
We also observe a clear increase in total mortality due to cold, with deaths peaking in the week just after the temperature is observed. The estimated coefficient indicates that weeks when temperature falls into the coldest bin experienced an increase in mortality of 0.15 log points relative to weeks in the reference bin. This effect is statistically significant at the 99% confidence level. The effect of cold weeks persists for around 4-5 weeks, with a monotonically decreasing effect.

There is also evidence of a smaller increase in mortality associated with warm weeks. Specifically, for weeks falling into the hottest temperature bin two weeks after the temperature is observed, $\hat{\alpha}_2^8 = 0.17$ (t-value = 5.14), implying that weeks in the warmest temperature bin (above 80 degrees F bin) are associated with an increase in total mortality of around 7 percent two weeks after the temperature change. Interestingly, this "warm-week" effect seems to persist longer than the cold-week effect (i.e., until around 6-7 weeks after the temperature change). 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. Overall, the top two panels of Figure 1 reveal that in pre-WWI London total mortality was elevated both during cold and warm weeks. However, the cold-week effect is significantly stronger in magnitude, while the warm-week effect is relatively more "persistent".

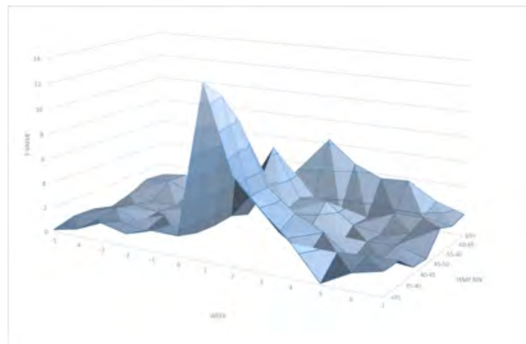
The bottom two panels of Figure 1 present the same type of results, but estimated only using data after 1920. Here we find a similar pattern for the cold-week effect as in the pre-WWI sample, while the warm-week effect looks strikingly different. In particular, total mortality is only elevated in warm weeks during the temperature change ($\hat{\alpha}_0^8 = 0.052$ and t-value = 4.09), and in the following weeks ($0 < j \leq 3$) total mortality is even lower or statistically insignificant ($j \geq 4$). Therefore, starting in 1920, the warm-week effect is only

Figure 1: Temperature and total mortality before and after WWI

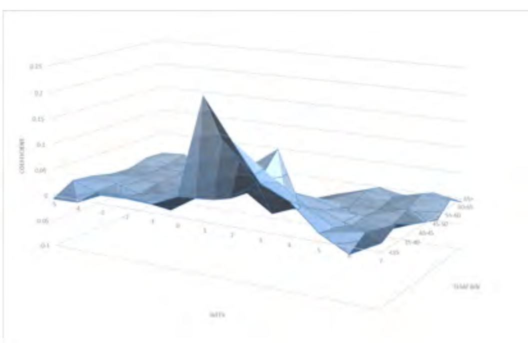
A. Coefficient estimates before WWI



B. T-statistics before WWI



C. Coefficient estimates after WWI



B. T-statistics after WWI

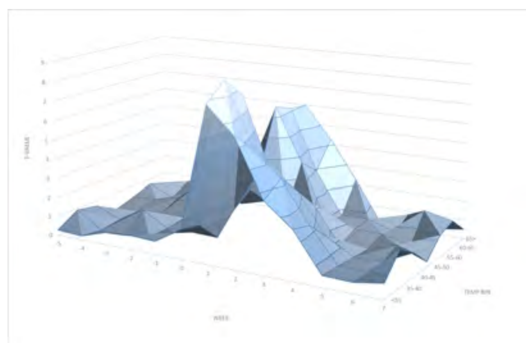
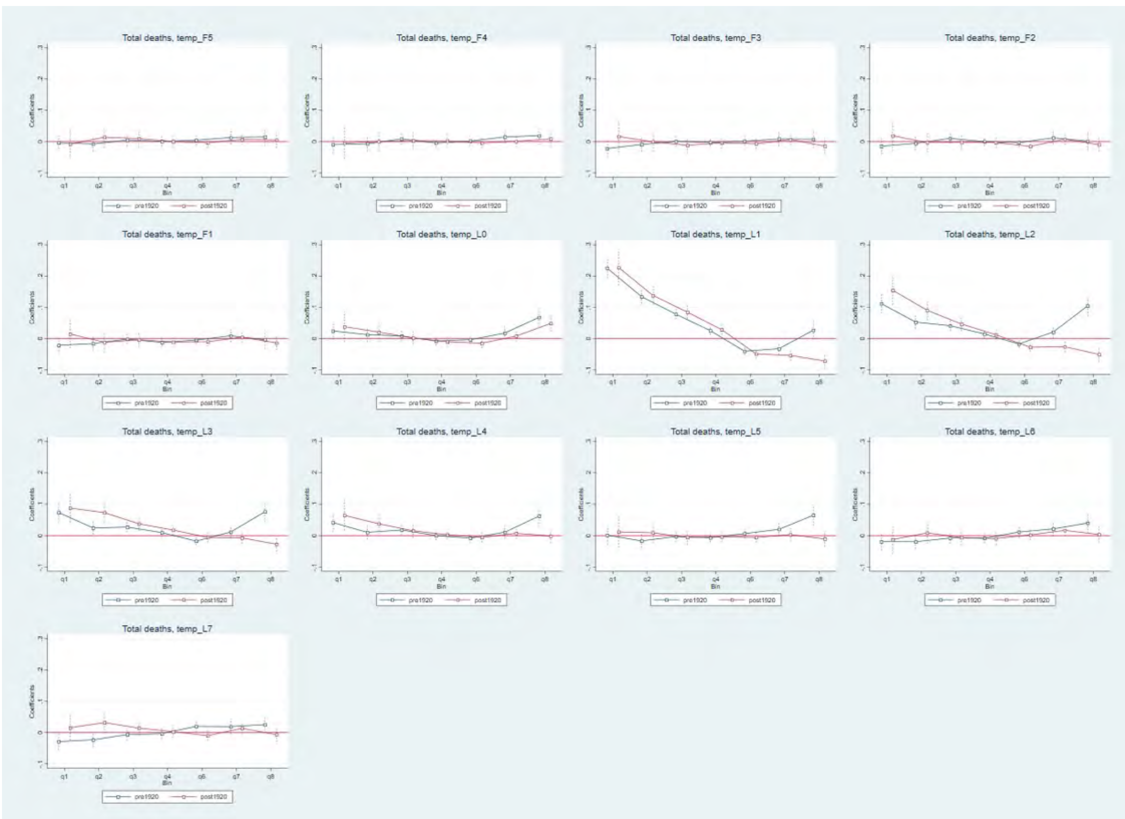


Figure 2: 2D event plots for total mortality, pre- and post-WWI



present during the temperature change and subsequently absent, meaning that most of the effect of temperature on total mortality is due to cold weeks.

Next, we consider the role that infant deaths play in generating the total-mortality results. Figure 3 presents 3D plots of the relationship between temperature and infant mortality, while Figure 4 presents the corresponding 2D plots for each lead and lag. Panel A of Figure 3 presents the estimates for (log) infant deaths prior to WWI, while Panel B presents the corresponding (numerical) t-values. Panels C and D present the coefficients and t-statistics for the 1920-1965 period. There is no systematic evidence of pre-trends in the infant mortality results; the lead temperature-bin estimates are rather small in magnitude and mostly statistically insignificant.

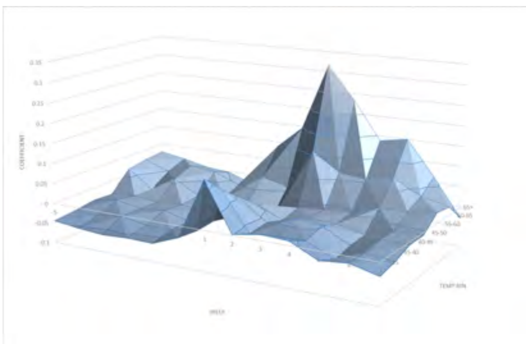
The most striking feature of the infant results is the large effect of warm weather on mortality in the years before WWI. The estimated effect of weeks in the warmest temperature begins contemporaneous to the temperature observation and peaks two weeks later with an estimated coefficient of $\hat{\alpha}_2^8 = 0.18$ (t-value = 4.99), implying an increase in infant mortality of around 18 percent relative to weeks with moderate temperatures (the 4th omitted bin). This effect persists for 6-7 weeks before becoming statistically insignificant. However, after 1920, this effect has essentially completely disappeared. This indicates that an important change in the relationship between temperature and infant mortality took place during our study period, substantially reducing the impact of warm weeks on infant mortality. Comparing the results for infants shown in Figure 3 to estimates obtained from all other age groups (i.e, total minus infant deaths), available in Appendix Figure 6, reveals that the increases in overall mortality in warm weeks in the period before WWI is driven almost entirely by infant deaths.

For the coldest group of weeks, the peak infant mortality effect for the period before WWI, which occurs with a one-week lag, is $\hat{\alpha}_1^1 = 0.08$ (t-value = 5.98) and two weeks after the effect the cold effect largely disappears. Thus, compared to total mortality, the cold-week effect on infants is significantly smaller and less persistent. From 1920 to 1965, we observe relatively little change in the impact of cold weather on infant mortality, in terms of either the magnitude or the statistical significance of the estimated coefficient. As mentioned in the data section, we were not able to obtain the number of weekly infant deaths for the modern period.

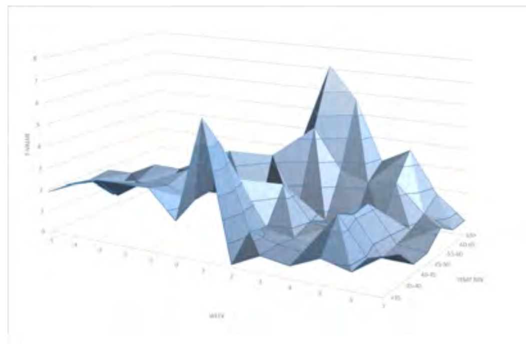
The patterns documented in Figure 1-4 are striking: The effect of cold weeks on total and infant mortality has been relative stable throughout our historical sample period of around 100 years. However, high-temperature weeks were mainly related to excess total mortality–and in particular infant mortality–in the early period of the historical sample.

Figure 3: Temperature and infant mortality before and after WWI

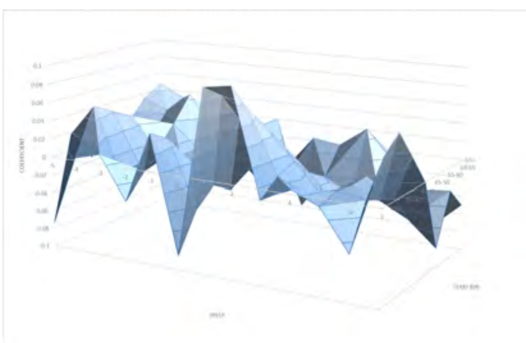
A. Coefficient estimates before WWI



B. T-statistics before WWI



C. Coefficient estimates after WWI



D. T-statistics after WWI

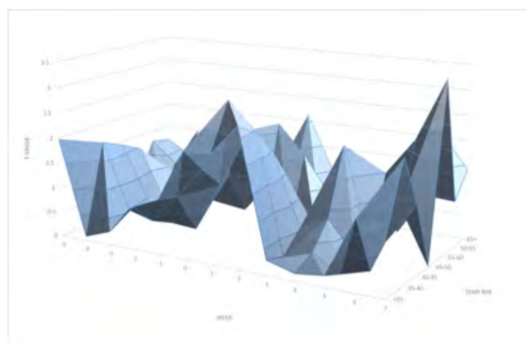
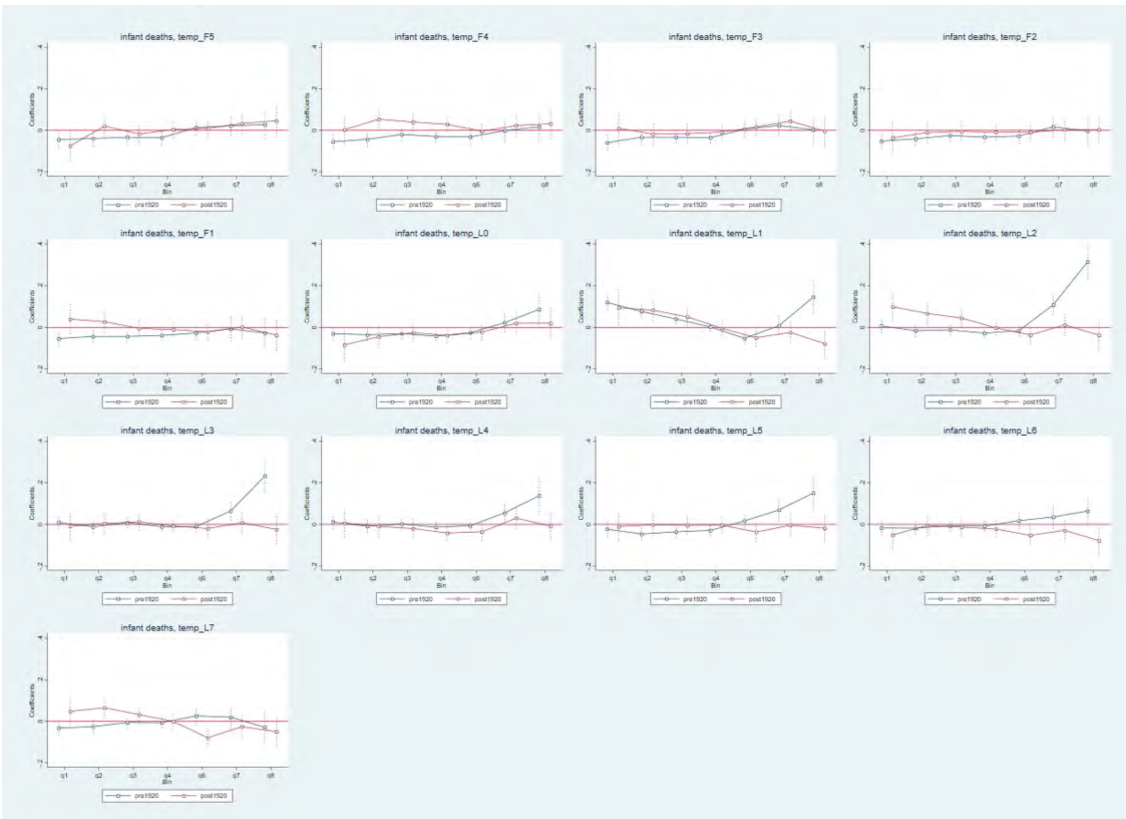


Figure 4: 2D event plots for infant mortality, pre- and post-WWI



One possible explanation of this time changing high-temperature mortality relationship is public-health investments. For example, as demonstrated in Alsan and Goldin (ming), the combinations of investments in sewerage and clean-water technologies reduced infant mortality of, e.g., digestive causes and in particular so during the spring and summer months. If the interaction with public-health investments is a viable explanation, we should expect to see high-temperature weeks influencing causes of deaths that were potentially affected by public-health investments, such as digestive causes.

To understand the factors driving changes in the temperature-mortality relationship, we now study the impact of temperature on mortality within particular cause-of-death categories. Since we have pinpointed infants as the main driver of increased mortality due to high temperatures in the period before WWI, it is natural to now focus the analysis on digestive diseases, the main cause-of-death for infants. Digestive diseases are also of interest because eliminating these was the primary focus of the many of the largest public-health interventions that took place during the study period, particularly those focused on improving water quality, general hygiene, food safety, and sewage removal.

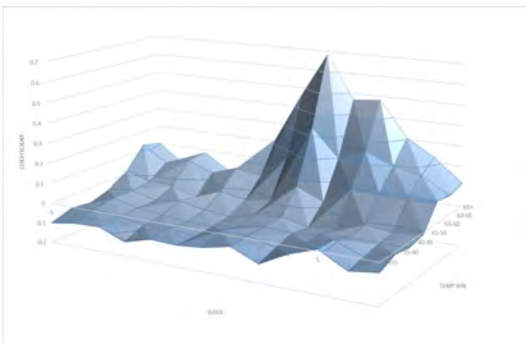
Figure 5 plots the impact of temperature on mortality due to digestive diseases. The top panels focus on the years before WWI, while the bottom panel focuses on years starting in 1920 and ending in 1965. The contrast between the top and bottom panels is striking. While the years before WWI saw large increases in digestive deaths associated with warm temperatures, this pattern essentially disappears after 1920. If we instead look at all causes of death other than digestive diseases (Appendix Figure 7), it becomes clear that the elevated mortality observed in warm weeks in the pre-WWI period are driven almost entirely by the digestive disease category. Thus, the change in the impact of warm weeks on mortality after WWI must be due to the elimination of digestive diseases among infants.[8]

Existing work suggests that a primary driver in the reduction of infant digestive disease deaths was improvements in access to water (Cutler and Miller, 2005; Ferrie and Troesken, 2008), particularly when combined with better sewer infrastructure (Cutler and Miller, 2005; Alsan and Goldin, ming). While the main water and sewer infrastructure in London was constructed between the 1850s and 1870s, further improvements across the late-nineteenth and early-twentieth centuries were required in order to bring down digestive disease deaths. For example, it was not until after 1900 that nearly all Londoners had access to a constant water supply, which Troesken et al. (2019) shows was important for bringing down digestive disease mortality.

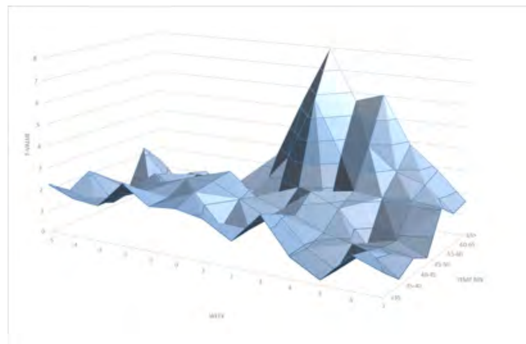
[8]We also observe weaker impacts of warm weather on deaths due to bronchitis and tuberculosis, not reported.

Figure 5: Temperature and digestive disease mortality

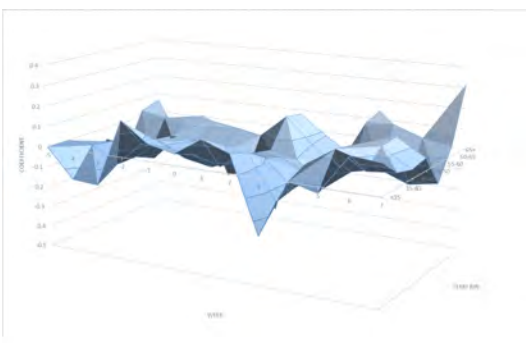
A. Coefficient estimates before WWI



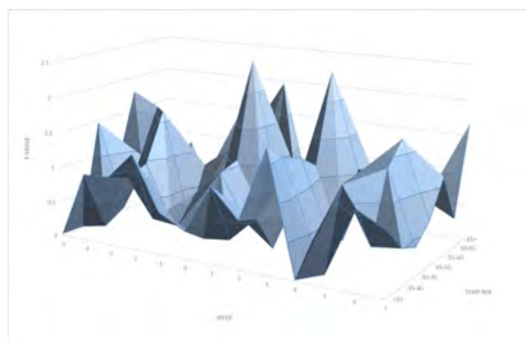
B. T-statistics before WWI



C. Coefficient estimates after WWI



D. T-statistics after WWI



6 Implications

The results presented above have implications for the impact of rising temperatures on mortality in London. We can study these effects directly because we observe a clear increase in temperature in London across our study period (see Table 1). In particular, average daily maximum temperature in our data rises by more than one degree F across the period we study, from an average of 62.0 in the pre-WWI period to 63.5 in the modern period. Similarly the average minimum temperature increased from 36.4 to 38.7.[9] Note that, since we are using data from Oxford, rather than stations in London, it is reasonable to interpret this increase as a result of global climate change, rather than an urban heat island effect.

As a starting point, Panel A of Table 2 describes our estimates of the actual number of excess deaths due to relatively hot and relatively cold weeks in different periods. These are obtained by using the estimated coefficients obtained from applying Eq. 1 to the data from each period. We calculate the heat effect as the number of deaths associated with the two top temperature bins and the cold effect as the number of deaths associated with the three lowest temperature bins, since those are the categories associated with statistically significantly elevated mortality during the period before WWI. For comparability, we use the same bins to assess the effects of hot or cold weeks on temperatures in later periods as well, even though weeks falling into the high-temperature bins actually become relatively healthy after WWI.

Panel A of this figure shows that we estimate that warm weeks, those falling into our top two temperature bins, are associated with 93,000 excess deaths in the period from 1876-1914, or about 3.0% of all deaths. However, after WWI, we actually observe fewer deaths in weeks when temperatures are in the top two temperature bins. This shift reflects the patterns described in the previous section. Unlike warm weeks, cold weeks remain associated with substantial numbers of deaths throughout the study period. We estimate that excess mortality associated with cold weeks accounted for 8.8% of deaths in the pre-WWI period and that this rose to 13.7% in the interwar period before falling to 8.3% just after WWII. Only in the modern period was there a substantial reduction in the share of mortality associated with particularly cold weeks, to 4.2% of all deaths.

What would mortality have looked like if the temperature-mortality relationship had not improved after WWI? To answer this, we calculate what mortality in the interwar,

[9] Our daily maximum and minimum temperature information comes from Oxford, but we see the same increases in the data from our London stations. For example, mean weekly temperature in the data from our London stations rises from an average of 49.5 in the pre-WWI period to 50.4 in the interwar period and 50.8 in the two decades after WWII.

post-WWII, and modern periods of our sample but imposing the temperature-mortality relationship estimated on pre-WWI data. For the inter-war and post-WWII periods we do this by age group, which means our estimates account for the impact of changes in the age composition of the population. In the modern period this is not possible because the Office of National Statistics has not been willing to provide the necessary mortality data by age group, so for that period this calculation applies the all-age mortality effects and therefore does not account for the effect of changes in the age composition of the population.

These estimates are shown in Panel B of Table 2. 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 or cold weeks, relative to weeks with moderate temperature, from the pre-WWI period to the baseline mortality rates 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, the result would have been an additional 15,424 heat related deaths in the interwar period, 5,599 in the decades after WWII, and 84,739 deaths in the period from 1981-2006, though the last figure should be interpreted with caution because it does not reflect the effect of changes in the age composition of the population. These additional heat related deaths would have accounted for a 3-4% increase in overall mortality. This is an important result, because it provides a direct estimate of how many heat-related deaths were averted as a result of changes in the temperature-mortality relationship, which from our previous results we know were driven primarily by sharp reductions in digestive disease deaths among infants.

For cold related deaths, imposing the pre-WWI temperature-mortality relationship would have reduced deaths substantially in the interwar period. In part this may just reflect progress in reducing deaths that were not associated with temperature during this period. In the decades just after WWII, there would have been little change in mortality. This highlights the fact that the impact of cold weather on mortality changed relatively little between the pre-WWI period and the decades just after WWII. However, by the modern period the impact of cold weather on mortality had been substantially reduced. As a result, applying the pre-WWI temperature-mortality relationship to the modern period implies a substantial increase in mortality: just under 54,000 additional deaths or an increase of around 3%.

It is also possible to provide a direct assessment of the impact that the increase in temperature in London across the study period had on mortality. To do so, we calculate the number of deaths that would have occurred in the decades after WWI given the temperature-mortality relationship observed during those periods, but with temperatures that resembled those observed in the nineteenth century.

Panel C of Table 2 presents the results of this exercise. These show that the increase in temperature in London from 1870-90 to the interwar period *reduced* overall mortality by roughly 6,970 deaths or 0.6% of all deaths. The effect of rising temperature in the post-WWII period was about 700 averted deaths, or 0.1% of total mortality, while in the modern period around 22,500 deaths were averted, equal to about 1.3% of total mortality. That rising temperatures reduced overall mortality should come as no surprising given the strong and persistent impact of cold temperatures on mortality and the almost non-existent effect of warm weeks on mortality after WWI.[10]

Note, however, that the beneficial effects of rising temperatures on mortality in London depended crucially on the virtual elimination of heat-related deaths in the period after WWI. What if temperatures had risen, but the temperature mortality relationship in London remained as it was before WWI? Answering this question is useful, because it gives us a sense of the extent to which improving the underlying disease environment affected the impact of rising temperatures. We answer this question in Panel D of Figure 2, by comparing the impact of the temperature rise observed from the 1870-90 period to the later periods on mortality given the temperature-mortality relationship estimated in the later periods, relative to the counterfactual impact assuming that the temperature-mortality relationship remained as it was prior to WWI.

The results, in Panel D, show that improvements in the temperature-mortality relationship meant that there were 2,591 fewer deaths associated with the rise in temperatures observed from the mid-19th century to the interwar period. This "adaptation effect" was equal to 1.6% of temperature-related deaths or 0.23% of all deaths during that period. The adaptation effect has a smaller impact in the post-WWI period, in part because temperatures were relatively low in that decade. However, in the modern period we estimate that adaptation averted 3,199 deaths that would have occurred as a result of rising temperatures, equal to 5.5% of temperature-related deaths or 0.19% of all deaths.

[10]This finding is in line with results from other studies, such as Hsiang et al. (2017), which estimate that climate change will decrease mortality in the Northern regions of the United States, where the climate is similar to that in London.

Table 2: Estimated and counterfactual effects of temperature on mortality by period

Period:	**Before WWI** (1876-1914)	**Interwar period** (1919-1939)	**Post-WWII** (1949-1965)	**Modern** (1981-2006)
Actual deaths	3,131,433	1,102,952	671,621	1,719,324
Panel A: Estimated actual deaths due to temperature by period				
Heat-related deaths	93,108	2,808	-8,468	-14,816
(as shr. of all deaths)	0.030	0.003	-0.013	-0.009
Cold-related deaths	275,583	151,163	55,897	72,617
(shr. of all deaths)	0.088	0.137	0.083	0.042
Total temp-related deaths	368,690	153,970	47,429	57,801
(as shr. of all deaths)	0.118	0.140	0.071	0.034
Panel B: Estimated deaths imposing the pre-WWI temperature-mortality relationship				
Heat-related deaths	–	15,424	5,599	69,923
Difference vs. Panel A	–	12,616	14,066	84,739
(diff. as a share of all deaths)		0.011	0.021	0.049
Cold-related deaths	–	120,413	84,414	126,530
Difference vs. Panel A	–	-30,750	28,517	53,913
(diff. as shr. of all deaths)	–	-0.028	0.042	0.031
Panel C: Impact of rising temperatures on mortality in London				
Heat-related deaths	–	2,013	-224	-1,874
Cold-related deaths	–	-8,983	-479	-20,697
Total temp. related deaths	–	-6,970	-703	-22,570
(shr. of all deaths)	–	-0.006	-0.001	-0.013
Panel D: Deaths due to rising temp. averted through changes in the temp-mort relationship				
Excess deaths	–	2,591	653	3,199
(share of temp-related deaths)		0.017	0.014	0.055

7 Conclusions

Our results show that the relationship between temperature and mortality in London evolved substantially from the mid-19th century to the mid-20th century. In the 19th century, warm weeks were associated with substantial excess mortality. These excess deaths were concentrated among infants and due primarily to digestive diseases such as diarrhea. By the interwar period, these warm-weather digestive deaths had been almost completely eliminated due to improvements in water treatment, sewage removal, food quality, general hygiene, and medical care.

We document and quantify the impact that these changes in the temperature-mortality relationship had on the impact of rising temperatures in London. Between the mid-19th century and the mid-20th century, measured temperatures in London increases by over one degree Fahrenheit. These rising temperatures actually reduced mortality by about 2%, because in London's temperate climate unusually cold temperatures account for more deaths than unusually warm conditions. However, a substantial portion of these gains would have been eliminated had the underlying temperature-mortality relationship not improved. Thus, our estimates offer a stark example of how public health improvements can help a location deal with the effects of rising temperatures. That we can find substantial effects even in a place where the climate is as mild as London suggest that in modern developing countries, which are almost always hotter than London, we should expect modifications of the disease environment to be even more important.

The primary implication of our results is that public health improvements can be a powerful tool for adapting to rising temperatures. The mortality patterns in London during early part of our study period are similar to those observed in poor developing countries today, and the challenges that those countries face, such as providing clean water and safe food, are the same challenges that London faced. Our results show that beyond the direct benefits of public health improvements, these improvements also provide the additional benefit of helping countries adapt to rising temperatures. At the same time, our findings make it clear that the health impacts of rising temperature are likely to be more severe in locations with poor public health infrastructure.

References

Alsan, M. and C. Goldin (Forthcoming). Watersheds in child mortality: The role of effective water and sewerage infrastructure, 1880-1920. *Journal of Political Economy*.

Barreca, A., K. Clay, O. Deschenes, M. Greenstone, and J. Shapiro (2016). Adapting to climate change: The remarkable decline in the u.s. temperature-mortality relationship over the 20th century. *Journal of Political Economy 124*(1), 105–159.

Barreca, A. I. (2012). Climate change, humidity, and mortality in the united states. *Journal of Environmental Economics and Management 63*(1), 19–34.

Basu, R. and J. M. Samet (2002). Relation between elevated ambient temperature and mortality: a review of the epidemiologic evidence. *Epidemiologic reviews 24*(2), 190–202.

Braga, A. L. F., A. Zanobetti, and J. Schwartz (2001). The time course of weather-related deaths. *Epidemiology 12*(6), 662–667.

Chestnut, L. G., W. S. Breffle, J. B. Smith, and L. S. Kalkstein (1998). Analysis of differences in hot-weather-related mortality across 44 us metropolitan areas. *Environmental Science & Policy 1*(1), 59–70.

Curriero, F. C., K. S. Heiner, J. M. Samet, S. L. Zeger, L. Strug, and J. A. Patz (2002). Temperature and mortality in 11 cities of the eastern united states. *American journal of epidemiology 155*(1), 80–87.

Cutler, D. and G. Miller (2005, Feb.). The role of public health improvements in health advances: The twentieth-century united states. *Demography 42*(1), 1–22.

Davis, R. E., P. C. Knappenberger, P. J. Michaels, and W. M. Novicoff (2003). Changing heat-related mortality in the united states. *Environmental health perspectives 111*(14), 1712.

Deschenes, O. (2014). Temperature, human health, and adaptation: A review of the empirical literature. *Energy Economics 46*, 606–619.

Deschênes, O. and M. Greenstone (2011). Climate change, mortality, and adaptation: Evidence from annual fluctuations in weather in the us. *American Economic Journal: Applied Economics 3*(4), 152–185.

Deschenes, O. and E. Moretti (2009). Extreme weather events, mortality, and migration. *The Review of Economics and Statistics 91*(4), 659–681.

Ferrie, J. P. and W. Troesken (2008). Water and chicago‘s mortality transition, 1850-1925. *Explorations in Economic History 45*(1), 1 – 16.

Graff Zivin, J. and M. Neidell (2014). Temperature and the allocation of time: Implications for climate change. *Journal of Labor Economics 32*(1), 1–26.

Hajat, S., B. G. Armstrong, N. Gouveia, and P. Wilkinson (2005). Mortality displacement of heat-related deaths: A comparison of delhi, sao paulo, and london. *Epidemiology 16*(5), 613–620.

Hanlon, W. (2018, April). London fog: A century of pollution and mortality, 1866-1965. NBER Working Paper No. 24488.

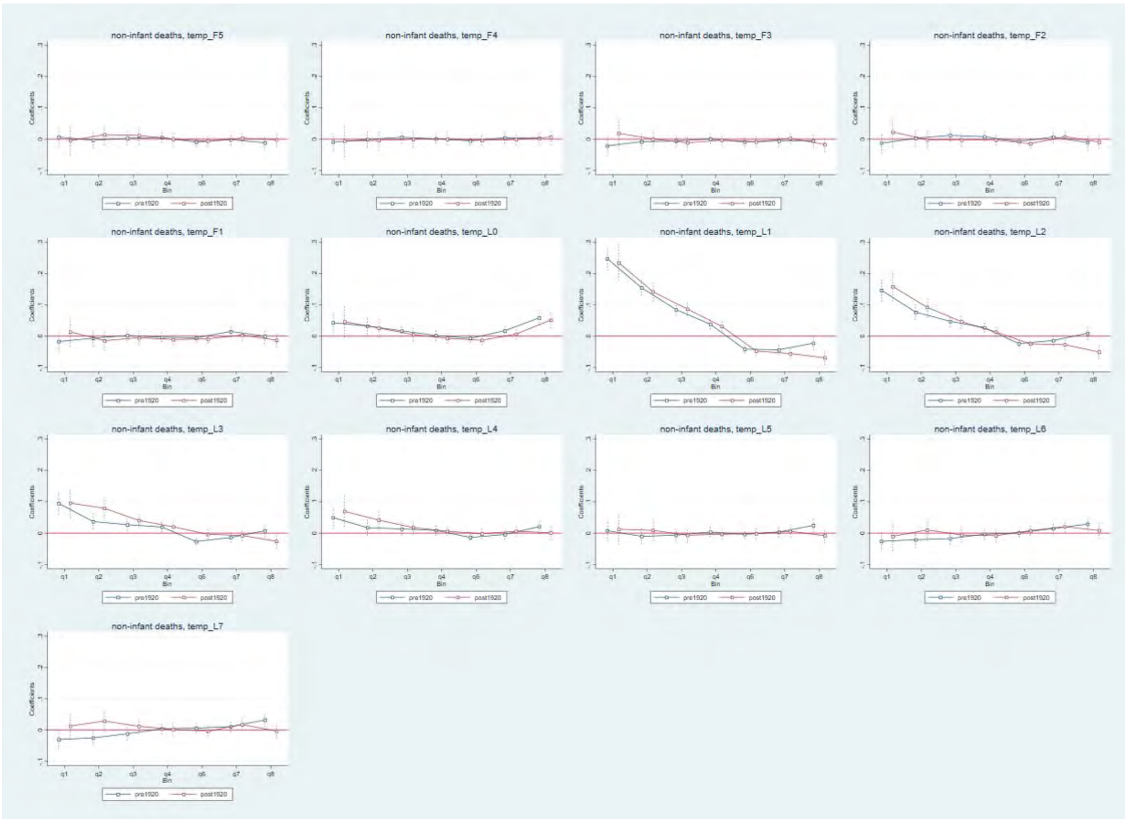
Hsiang, S., R. Kopp, A. Jina, J. Rising, M. Delgado, S. Mohan, D. Rasmussen, R. Muir-Wood, P. Wilson, M. Oppenheimer, et al. (2017). Estimating economic damage from climate change in the united states. *Science 356*(6345), 1362–1369.

Hug, L., D. Sharrow, and D. You (2017). Levels and trends in child mortality. United Nations Children's Fund.

Troesken, W., N. Tynan, and Y. Yuanxiaoyue (2019, January). What are the health benefits of constant water supply? evidence from london, 1860-1910. Mimeo.

Woods, R. (2000). *The Demography of Victorian England and Wales*. Cambridge, UK: Cambridge University Press.

Figure 6: 2D event plots for non-infant mortality, pre- and post-WWI



A Results in 2D plots

Figure 7: Total and non-digestive mortality, pre-WWI