On the Seasonality of Disease: Implications for the Effects of Climate Change on Health

Karen Clay, CMU and Werner Troesken, Pitt

0. Introduction

Demographers have long recognized the connection between climate and disease, particularly the connection between the seasons and various infectious diseases. For example, Sir Leonard Rogers (1926) studied the connection between the monsoon season and smallpox in India during the nineteenth and early twentieth century. His data showed that during and immediately following periods of high humidity smallpox rates tended to be much lower. North (1909) used data from New York City to show that diarrheal diseases such as typhoid fever peaked during the summer and early fall, that respiratory diseases such as pneumonia, bronchitis and diphtheria peaked during the winter months of January, February and March, and that febrile childhood diseases such as scarlet fever and measles peaked during the late winter and early spring. Still others examined how environmental temperatures affected the viability of disease-causing bacteria and viruses, such as the typhoid bacillus (e.g., Houston 1912).

In this paper, we reconsider the seasonality of disease. Through this reconsideration, we hope to establish the robustness of the seasonality of disease and to better understand the mechanisms that underlie the connection between climatic variation and disease. In particular, the paper is divided into three parts. Part I explores the seasonal patterns in deaths from six

causes: pneumonia, typhoid fever, scarlet fever, diphtheria, infant mortality and smallpox. We find that these infectious diseases are highly sensitive to seasonal variation in temperature or rainfall but that the effects of temperature are not uniform or linear. Part I also asks why seasonal variation in temperature affects disease rates. The central finding here is that exposure to organic and inorganic pathogens depends heavily on temperature. Part II looks for evidence of what demographers and epidemiologists refer to as harvesting such that unusually high or low temperatures are killing only the weakest and most unhealthy individuals, people who would have died anyway even without the extremes in temperature. Part II also asks a related question: whether poor socioeconomic groups were more vulnerable to changes in temperature than wealthier groups. Part III explores how public health interventions altered seasonal disease patterns and helped protect populations against the diseases related to temperature change.

1. On the Seasonality of Disease

Using monthly data from nineteenth century Chicago, Figures 1 and 2 demonstrate the sensitivity pneumonia to seasonal changes in temperature. Figure plots the average monthly pneumonia rate for the period 1871 through 1894. Pneumonia is an acute inflammation of the lung tissues (excluding the airways) that has bacteria, viral, and other pathogenic causes. Notice that pneumonia rates peak in the winter and early spring (January through April), decline in the late spring and early summer, and begin rising again during the fall and early winter. The magnitude of this seasonal variation is quite large. The average pneumonia rate is roughly 3 times greater January, for example, than it is in July, August, and September. Using the same data, figure 2 plots the monthly pneumonia rate against monthly temperature. There is a clear and strong negative correlation between pneumonia and temperature (R^2 = .315; β = -.230; t-

statistic = 11.23).

Figure 3 plots the average infant mortality rate in Chicago from 1871 through 1906 by month. Notice that infant mortality rates spike in July and decline steeply thereafter. Infant mortality rates are roughly twice as high in July as they were in the preceding months. Figure 4 plots the monthly infant mortality rate against monthly temperature. The effects of temperature appear to be non-linear: below 60 degrees, temperature appears to have little affect on mortality, but once monthly temperatures rise above 60 degrees, the infant mortality rate rises steeply. As explained in greater detail below, diarrheal diseases account for 37 percent of all infant deaths in Chicago; airborne diseases (pneumonia, bronchitis, and influenza) for 23 percent of all deaths; and congenital defects for 20 percent.

Figure 5 plots average typhoid rates by month in Chicago over the 1871-1906 period. Typhoid is mainly a waterborne disease and most epidemics were started because healthy individuals drank water tainted by the wastes of infected individuals. Typhoid rates rise rapidly during early summer, peak in the late summer and early autumn, and then quickly fall off in the late fall and winter. Typhoid rates are roughly twice as high in the peak summer and fall months as they are during the winter months. Figures 6 and 7 plots monthly typhoid rates against temperature; temperature, though, has a two month lag. Temperature is lagged because typhoid has a long incubation period and because it usually takes several weeks for the disease to kill its victims. Both figures reveal a strong positive correlation between lagged temperature and typhoid rates.

Diphtheria seems to follow an apposite seasonal pattern. Again using data form Chicago, figure 8 plots the average diphtheria rate by month. Rates peak during the early winter and

bottom out during the mid-summer. The seasonal variable is large so that diphtheria rates in the summer are roughly double rates during the winter. Figure 9 suggests that scarlet fever follows the same seasonal pattern as diphtheria, peaking in the winter and bottoming out during the summer.

Infectious diseases are not only correlated with temperature, but also rainfall. In the graphs that follow, we document the connection between rainfall and smallpox in India.

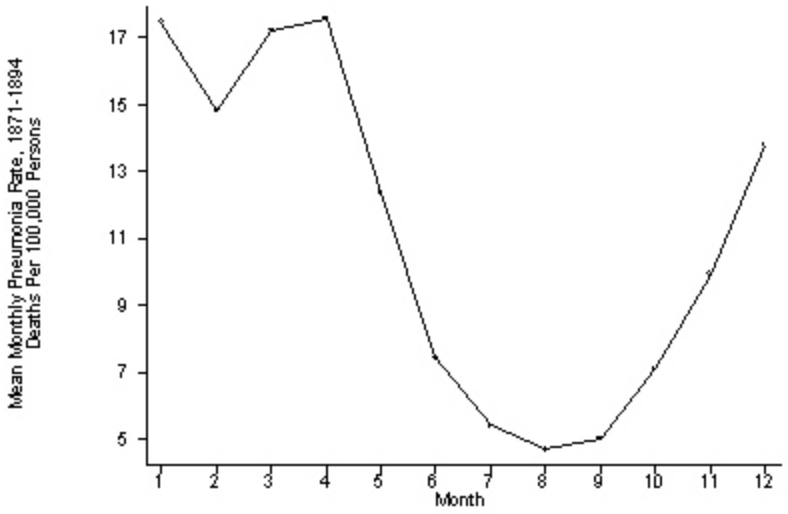

Figure 1

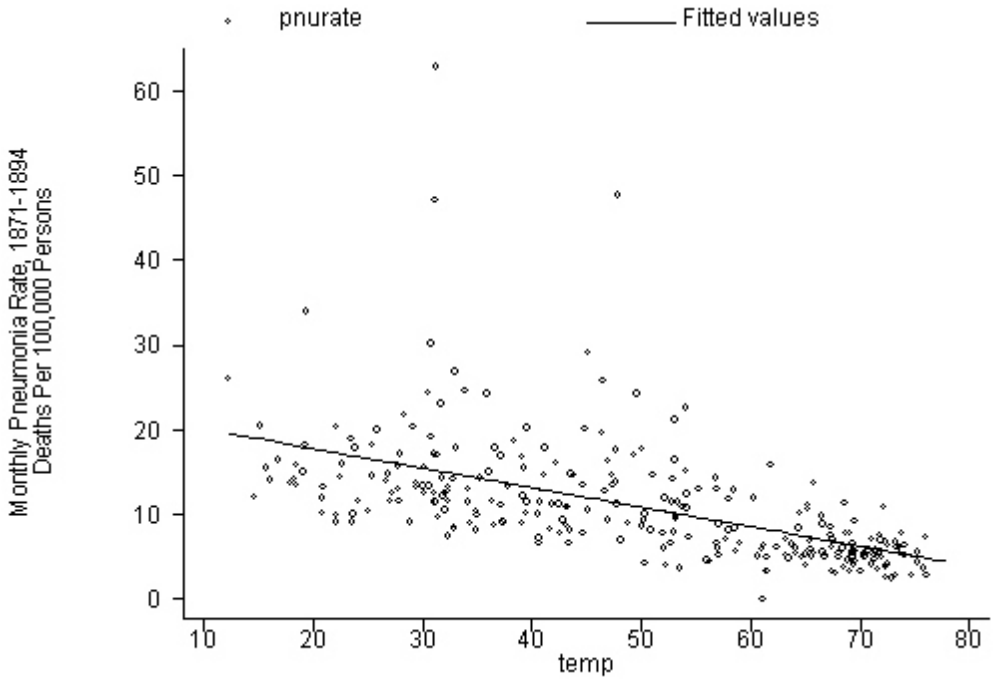

Figure 2

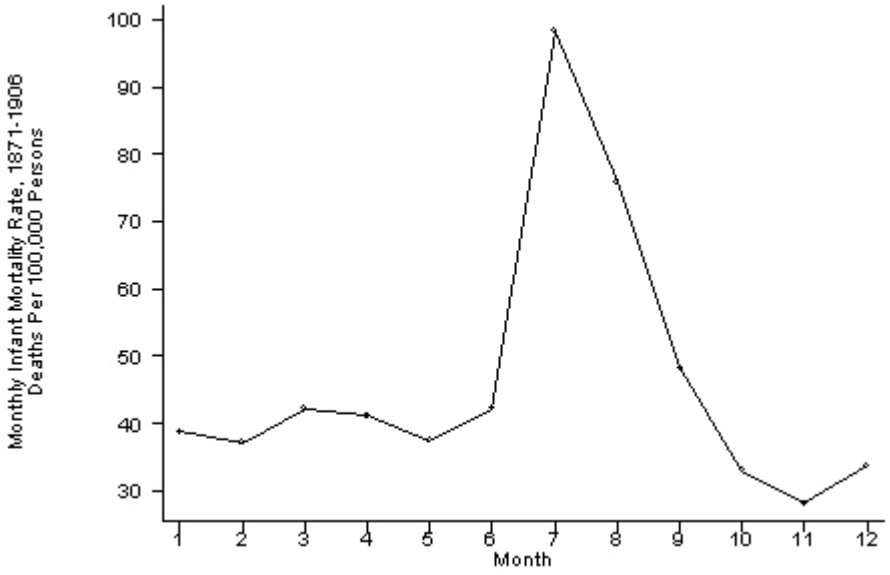

Figure 3

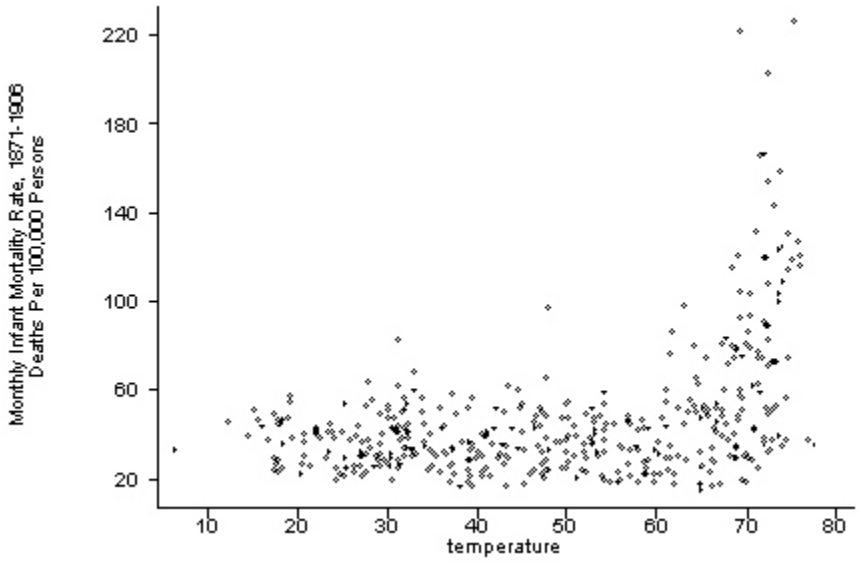

Figure 4

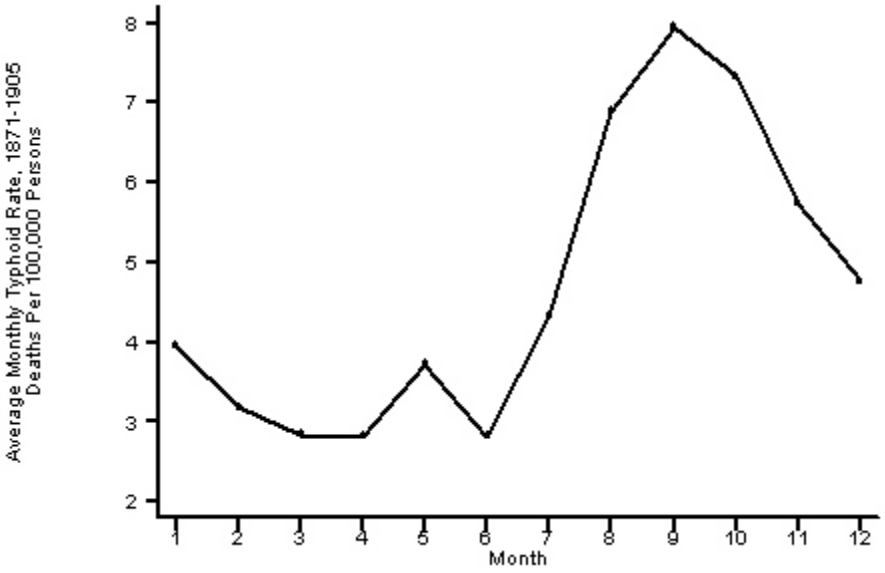

Figure 5

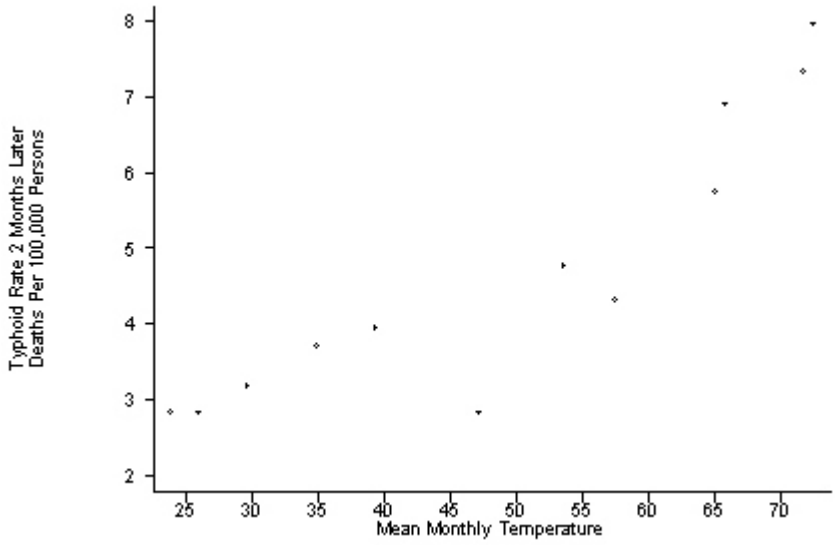

Figure 6

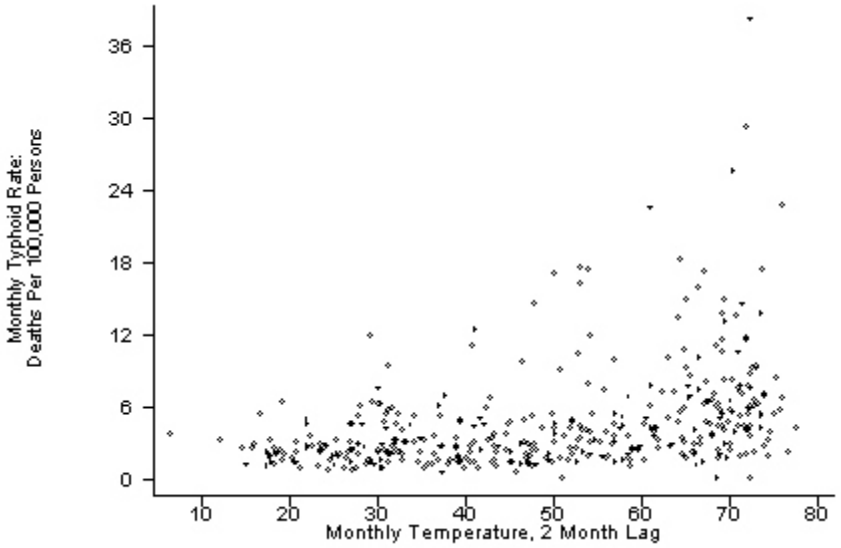
Monthly Typhoid Rate:
Deaths Per 100,000 Persons
0
6
12
18
24
30
36
10
20
30
40
50
60
70
80
Monthly Temperature, 2 Month Lag

Figure 7