THE ROLE OF AMBIENT TEMPERATURE AND INFLUENZA OUTBREAKS: A CASE STUDY OF THE 1918 SPANISH FLU PANDEMIC

A thesis submitted in partial fulfillment of the requirements for the degree

MASTER OF SCIENCE

in

ENVIRONMENTAL STUDIES

By

ASHTON BASAR

APRIL 2019

at

THE GRADUATE SCHOOL OF THE UNIVERSITY OF CHARLESTON, SOUTH CAROLINA AT THE COLLEGE OF CHARLESTON

Approved by:

Dr. Brian Bossak, Thesis Advisor

Dr. Norman Levine

Dr. Matthew Nowlin

Dr. Jacob Steere-Williams

Dr. Godfrey Gibbison, Interim Dean of the Graduate School

ABSTRACT

THE ROLE OF AMBIENT TEMPERATURE AND INFLUENZA OUTBREAKS: A CASE STUDY OF THE 1918 SPANISH FLU PANDEMIC
A thesis submitted in partial fulfillment of the requirements for the degree

MASTER OF SCIENCE

in

ENVIRONMENTAL STUDIES

by

ASHTON BASAR
APRIL 2019

at

THE GRADUATE SCHOOL OF THE UNIVERSITY OF CHARLESTON, SOUTH CAROLINA AT THE COLLEGE OF CHARLESTON

The seasonal outbreak of influenza occurs around the same time every year with distinct patterns across temperate regions of the world. Pandemic outbreaks such as the 1918-1919 outbreak often referred to as "Spanish Flu" are less predictable and more lethal than the annual flu seasons. Scientists have suggested that environmental factors such as ambient (outdoor) temperature may have a role in the seasonal patterns following latitudinal lines between the northern and southern hemispheres of the globe. Pandemic strains, such as the 1918 Spanish Flu, have yet to be studied for a significant relationship between peak mortality and environmental factors. Using historical 1918 Spanish Flu data in three North American cities as case studies, daily mortality counts, and daily temperature values were analyzed for a significant relationship between peak flu activity and a change in temperature. Findings suggest that a fluctuation in temperature may occur prior to the peak transmission of a pandemic strain of influenza. Historical naval and civilian death records from Charleston, SC were also analyzed to provide further evidence of the spread of disease. The information found in this study can be utilized to inform future public health officials about epidemic and pandemic outbreaks of influenza at the local level and worldwide.

Acknowledgements

I would like to thank all my previous advisors, teachers, and mentors who have allowed me to continue expanding my education and encouraging me to share that knowledge with others. I would like to thank Dr. Bossak and the rest of my committee for leading this project and contributing their expertise to the expansive field of environmental epidemiology. Most of all, I would like to thank my parents for the love and support they have given me throughout my entire life.

Table of Contents

List of Figures

List of Tables

Chapter 1: Introduction

1.1 What is Influenza?

The annual flu season affects individuals around the world and often claims the lives of thousands of people each year (Azziz Baumgartner *et al.*, 2012). Influenza is a virus that infects cells of the human body by integrating their own DNA into the original cell structure (Skehel & Wiley, 2000). The influenza virus is unique due to its constant genetic changes and DNA mutations (Nicholls, 2006). In a single flu season, the virus can mutate multiple times leading to newly emerging strains and severities of infection (Smith *et al.*, 2009). There are three main classes of influenza that are categorized as Influenza A, B, and C, with type A being the most lethal (Nicholls, 2006). The nomenclature of influenza relies on the number of Hemagglutinin and Neuraminidase receptors required for entry into cells and are located on the outside of the virus (Skehel & Wiley, 2000). An example, H1N1 is a subtype of the Influenza A virus class that has been associated with some of the deadliest outbreaks of influenza (Zimmer & Burke, 2009). The 2009 Swine Flu pandemic and the 1918 Spanish Flu pandemic were both varying strains of the H1N1 subtypes (Zimmer & Burke, 2009). Although the structure of each of these viruses are similar in nature, the slight mutations between them are what distinguishes virulence and mortality between the two strains (Smith *et al.*, 2009).

The transmission of the virus occurs through direct contact, airborne particles, or surface to host contact (Mathews *et al.*, 2009). A unique characteristic of the influenza virus is the zoonotic nature of the pathogen that allows the virus to infect various vertebrate species (Webster *et al.*, 1992). The specific receptors on the outside of the

virus allow viral entry into mammalian cells depending on the host cell type at the time of infection (Skehel & Wiley, 2000). While influenza most commonly infects humans, it also has animal hosts including birds, swine, and other vertebrate species (Webster *et al.,* 1992).

Rapid mutation of the virus also defines specificity for treatments such as vaccines and antivirals (Mathews *et al.,* 2009). The current production of vaccines relies heavily on the monitoring of circulating viruses in other countries to prepare for future production (Gerdil, 2003). The flu vaccine takes approximately six months to produce, and around 250 million doses are distributed worldwide each year (Gerdil, 2003). However, the virus often mutates before reaching the United States and the timing of production leads to ineffective vaccinations (Layne *et al.,* 2009). Antivirals are currently being approved for production, but their efficacy has only been tested in limited clinical trials (Jefferson *et al.,* 2006). The rapid mutation of future pandemic strains may not warrant enough time for mass vaccine production and distribution (Layne *et al.,* 2009).

Most strains of viruses tend to be predictable in their patterns of transmission and lethality (Dowell, 2001). Nonpolio viruses and highly transmissible viruses such as RSV, rotavirus, and influenza all have cyclical epidemic outbreaks each year (Pons-Salort *et al.,* 2018). Scientists have monitored these diseases seasonally and have found patterns in transmission at the latitudinal and longitudinal levels (Dowell, 2001). Pandemic influenza could also be monitored in a way that could allow scientists and public health officials to effectively predict the course of an outbreak and devise quarantine protocols.

1.2 The Seasonality of Influenza

The annual flu season is predictable and relatively stable across temperate regions of the world (Azziz Baumgartner *et al.,* 2012). Countries with a temperate climate have a singular annual peak in January or February in the Northern Hemisphere and July in the Southern Hemisphere (Azziz Baumgartner *et al.,* 2012). This repetitive pattern has led researchers to try to find the cause of predictability in annual flu seasons (Tamerius *et al.,* 2011). Many researchers correlate the outbreaks with air travel, holiday and school schedules, close indoor proximity in the winter, and decreased immunity. (Chattopadhyay *et al.,* 2018). However, many of these variables would lead to a much earlier flu season instead of mid-winter months. School schedules send students back to classes in late August or early September. Holiday travel occurs about a month to two months prior to the annual outbreak. Global flights are constantly occurring throughout summer and winter months. The zoonotic nature of the pathogen has led researchers to study animal movement patterns such as bird migrations, but the viral prevalence in wild birds is low (Olsen *et al.,* 2006). Numerous studies have speculated that a change in weather attributed to the winter months may have a role linked to the spread and severity of the disease (Reichert *et al.,* 2004).

Influenza affects the entire globe at different times of the year depending on the latitude (Dowell, 2001). The virus circulates across the equator multiple times throughout the year leading to multiple outbreaks of influenza in the tropical regions of the globe (Chew *et al.,* 1998). Different strains of the flu circulate together with the introduction of newly mutated strains along with the previous strains (Finkelman *et al.,* 2009). Weather parameters such as temperature and relative humidity have been the most

researched variables associated with the seasonality of the flu (Lowen & Steel, 2014). Low humidity levels have been found to increase viral survival in many strains of influenza (Shaman & Kohn, 2009; Yang *et al.*, 2012). However, temperature has continually had the highest level of significance when compared to cases of influenza (Sundell, *et al.*, 2016). The transmission of the virus following specific latitudes across the globe still leaves researchers with missing information about the nature of the viral passage. The answer may be multi-factorial, but ambient temperature is likely a key piece to unraveling the full etiological picture of pandemic outbreaks.

1.2.1 Tropical Regions

The prevalence of influenza in tropical regions of the world does not follow a typical unimodal peak like the temperate regions of the globe (Tamerius *et al.*, 2013). Although data is limited, tropical countries tend to experience bi- or tri- modal peaks closely associated with the rainy seasons (Baumgartner *et al.*, 2012). A study of 85 countries found that 85% of temperate climate countries exhibit a unimodal distribution while 28% of tropical countries have bimodal distributions and 13% have trimodal distributions (Baumgartner *et al.*, 2012). Researchers have attributed this pattern to increased indoor proximity during the rainy months, cooler temperatures, and school schedules (Fuhrmann, 2012). Most health data are sparse in many of the tropical countries due to the lack of electronic surveillance systems (Viboud *et al.*, 2006). Recent studies have begun using newly implemented digital disease surveillance systems implemented by the Centers for Disease Control (CDC) in the United States for higher levels of accuracy (Zhao *et al.*, 2017).

Tropical regions have been studied with similar weather parameters to attempt to explain the bi- or tri- modal peaks in these areas (Tamerius *et al.,* 2013). Studies in the late 1990s concluded that the multi-modal patterns exist in these regions due to lower air temperature and lower relative humidity (Chew *et al.,* 1998). However, many tropical regions have peak flu activity during the rainy seasons and a more recent study found a significant correlation between the roles of rainfall, humidity, and temperature (Soebiyanto *et al.,* 2015). Researchers attempted to separate the areas of tropical and temperate regions by explaining that cold-dry air in the temperate regions and humid-rainy air in the tropics are the best conditions for viral transmission (Davis *et al.,* 2012; Tamerius *et al.,* 2013). Digital disease surveillance has provided a better data set to study the tropical regions, and researchers have now found a significant correlation between temperature and peak activity with no distinct difference between the humidity levels (Zhao *et al.,* 2017). Evidence suggests that in tropical and temperate regions of the world, temperature is the presiding factor that influences the distribution of influenza between individuals in repetitive seasonal patterns.

1.3 Weather Parameters and Meteorological Findings

As scientists continue to attempt to explain the seasonality of influenza and other diseases, they have focused mainly on the environmental factors that may have a role in the transmission between populations (du Prel *et al.,* 2009). Viral infections such as Rotavirus and Rubella have also shown cyclic seasonal activity with evidence linked to the environment (Dowell, 2001). Weather parameters including outdoor temperature, relative humidity, and precipitation are variables that scientists have examined to explain the phenomenon of the annual influenza outbreaks (Lofgren *et al.,* 2007). Levels of

ultraviolet light exposure have been correlated with host Vitamin D and melatonin levels but exhibit lower levels of significance with immunity against influenza (Bonilla *et al.,* 2004; Cannell *et al.,* 2006; Martineau *et al.,* 2017). Vapor pressure has also been a suggested parameter to study with seasonality, but historical data often did not record vapor pressure (Lipsitch & Viboud, 2009).

Ambient temperature continues to be statistically significant parameter associated with influenza transmission (Sundell *et al.,* 2016). In temperate regions of the world, the singular peaks in influenza activity coincide with mid-winter months were the air is dry, cold, and reduced sunlight exposure to the lowest levels of the year (Li *et al.,* 2018). Studies have attempted to re-create the winter conditions in a lab setting using in vivo models to simulate exposure and transmission (Lowen *et al.,* 2007; Lowen *et al.,* 2008). A study used guinea pigs as a model for airborne transmission of the flu by using temperature and humidity as independent variables (Lowen & Steel, 2014). Lowen and Steel initially conducted a study in 2008 and found that higher temperatures decreased airborne transmission of the virus but not through direct contact (Lowen *et al.,* 2008). Another study in 2014 indicated that both variables influenced the transmission between guinea pig cages with lower temperatures and humidity levels showing the highest rates of airborne infection (Lowen & Steel, 2014). Although there are many routes of transmission of influenza, the airborne distribution may have a larger impact on the passage of the disease between individuals.

Pandemic strains of influenza have peaked much earlier in the season than the annual seasonal strains (Table 1). Pandemics have consistently occurred around the autumn months in mid-October and November in the United States (Morens &

Taubenberger, 2009). The 1918 Spanish Flu pandemic also had peak activity in mid-October with a few smaller waves in winter and spring 1919 (He *et al.,* 2011). The highest level of activity, termed the "Herald Wave," had the greatest levels of mortality around October 1918 in most regions of the United States (Olson *et al.,* 2005). Records from Europe also showed similar patterns in the pandemic with a short spring wave, a large autumn wave, and another wave in late-winter 1919 (Pearce *et al.*, 2010).

The 2009 Swine Flu pandemic followed a similar trend with an early spring wave and a larger wave of activity in October 2009 (Smith *et al.,* 2009). Scientists have also studied weather parameters associated with the Swine Flu and found a significant correlation between decreased relative humidity and lower temperatures prior to the peak (Steel *et al.,* 2010). Although the impact of the 2009 Swine Flu was not as significant as the 1918 Spanish Flu, the H1N1 strain in both cases has led researchers to question the similarities in the pandemics (Steel *et al.*, 2010). Scientists believe that the pandemic strains propagate shortly after the end of the annual flu seasons and peak in mid-Autumn (Fox *et al.,* 2017). Similar patterns observed in future pandemics can allow preparations to begin for timely public health interventions (Willem *et al.*, 2012).

1.4 The 1918 Spanish Flu Pandemic

The Spanish Flu was one of the most virulent strains of influenza that infected over 500 million people, which was over a third of the world's population at the time (Taubenberger & Morens, 2006). The virus was one of the most lethal strains with an estimated 50 million deaths or 2% of the population in 1918 (Johnson & Mueller, 2002). Public health officials during the time thought that the flu strain originated in Spain, and thus named the disease the Spanish Flu (Barry, 2004). It was not until 2005 that the

entire genome of the virus was sequenced from a frozen lung tissue sample found in the permafrost in Alaska (Tumpey *et al.*, 2005). Scientists have found that the H1N1 strain showed high expression in respiratory regions of the body including lung and epithelial tissues, and unique expression in brain tissues (Tumpey *et al.*, 2005). Most viral strains do not permeate the blood brain barrier which may have been another reason for the higher level of lethality associated with the Spanish Flu strain. (Sheng *et al*., 2011).

1.4.1 Historical Perspective

At the time of the 1918 Spanish Flu pandemic, the First World War was coming to an end, yet another threat was on the horizon (Byerly, 2010). The United States military involvement in Europe peaked from September 1918 to November 1918, which was around the same time of the peak mortality rates from the Spanish Flu pandemic (Byerly, 2010). Researchers believe that 20% to 40% of military personnel were infected with the disease (Bylerly, 2010). The final death toll amongst military personnel and civilians was greater than the amount of deaths recorded in combat (Bylerly, 2010).

Today, scientists still have not found the original location of the flu outbreak, but many believe that the first case in the United States was introduced in a military base in Fort Riley, Kansas (Barry, 2004). Although the virus had spread rapidly across the United States and Europe around the same time, military personnel would have had to travel across the Atlantic Ocean by ship because commercial air travel was not established at the time (Byerly, 2010). The extreme virulence and rapid transmission were confirmed in military records with some medical officers falling ill after only 48 hours of exposure (Byerly, 2010).

At the time of the pandemic, molecular biology and virology did not exist and doctors in the US and Europe struggled to distinguish the new pandemic from other respiratory diseases such as pneumonia and bronchitis. (Bootsma & Ferguson, 2007). Medical records were also not recorded accurately, and some deaths were recorded as pneumonia instead of influenza (Appendix A). Most records were handwritten or typed on typewriters, and many of the records only included deaths (Appendix D). Especially in underserved communities, many of the death tolls were averaged over a long period of time or not recorded at all (Doshi, 2008). At the height of the pandemic, healthcare facilities had maximized their resources and were running out of space, especially in city centers (Acuna-Soto *et al.,* 2011). Within 10 days, many infirmaries were out of room for incoming patients and had to go to great lengths to find space and treatments for the sick (Bootsma & Ferguson, 2007). Public health officials began trying to implement quarantine measures, but many of the communities had already reached peak mortality and infection rates by the time the interventions commenced (Juckett, 2006). Studies have found that death rates in city centers were 30-40% higher than in rural areas of the globe (Chowell *et al.*, 2008). Most individuals living in rural and poorer areas died in their homes rather than in hospitals (Chowell *et al.,* 2008). Although modern medicine and health facilities have greatly advanced since 1918, this case serves as a learning opportunity for the necessary public health quarantine and social distancing measures necessary for future pandemics (Cooper *et al*., 2006).

1.4.2 History in Charleston, SC

In 1918, Charleston was a thriving small city with a population of around 60,000 people and an active military base ("Year Book," 1918). The first cases of Spanish Flu

were reported at the naval training station amongst 16 infected sailors ("The Spanish," 1918). The military base had 4,167 personnel and a recorded 1,118 cases of influenza by the end of 1919 (Sohn & Boulier, 2012). Military records indicated that 26 military personnel had confirmed deaths caused by the Spanish Flu, but actual counts may be much higher (Sohn & Boulier, 2012).

During the early 1900s, many cities were not equipped with large health facilities, yet Charleston had six major health centers during the pandemic ("Pneumonia," 1918). In the fall of 1918, a typhoid epidemic was coming to an end, yet the Spanish Flu was quickly to follow across the southeastern region ("The Spanish," 1918). Public health officials attempted to slow the progression of the disease by cancelling the Ringling Brothers Circus show and closing schools and churches ("Circus", 1918; "College", 1918). City officials freely distributed liquor to those afflicted to help 'cure' the disease (Moore, 1985). Public health officials advised citizens to refrain from sweeping their floors which would stir up dust and spread the flu (Moore, 1985). Dr. J. Mercier Green was the City Health Officer at the time and directed all public health measures in Charleston (Green, 1918).

By late October, over 5,500 records of Spanish Flu were recorded in Charleston, yet Dr. Green estimated the true count was probably doubled due to misdiagnosis (Green, 1918). As quickly as the pandemic spread throughout the city, the number of flu cases quickly dropped around mid-November 1918 ("No More," 1918). On November 6th, the city ban was lifted, and children returned to school a week later ("No More," 1918). A second wave of flu spread throughout the city again in January 1919 (Moore, 1985). Initial records suggested that the impact of the flu was even greater during the second

wave, but more recent studies have suggested a much smaller impact (Moore, 1985). There is a possibility that the second wave was a less lethal strain of the Spanish Flu caused by a mutation in the virus or a completely different strain of seasonal flu.

1.4.3 Young Adult Responses

During the annual flu season, the highest levels of mortality are shown with infant and elderly populations due to their compromised immune systems (Abedi *et al.*, 2014). A distinct characteristic of the 1918 Spanish Flu pandemic was the "W-shaped" epidemic response curve with the highest mortality rates shown in the young adult population (Taubenberger & Morens, 2006). This type of mortality curve has not existed during any other pandemic or annual flu season pattern (Langford, 2002). Researchers have attempted to explain this phenomenon with a few hypotheses attributed to a prior immunity in older adults and an overactive immune response from the young adult population (Shanks & Brundage, 2012). Studies have suggested that "immunological memory" may have protected older adults after living through a pandemic called the Russian Flu (Ahmed *et al.*, 2007). Any level of prior exposure may have given the older population the necessary antibodies to fight the virus (Ahmed *et al.*, 2007; Mathews *et al.*, 2010).

A study showed that young adults who were not involved in the war had much higher mortality rates compared to the older adults and children (Ahmed *et al.*, 2007). They also examined a control study in an isolated area of Alaska that would not have had the prior immune exposure and showed that their mortality curve did not exhibit the "W-shaped" signature pattern (Ahmed *et al.*, 2007). A similar study examined another

isolated case in Mexico that had a spring wave prior to the pandemic that took the lives of many individuals over the age of 65 (Chowell *et al.,* 2010). This suggests that the isolated areas did not have a pre-existing immune response in the older adult population that would not have been exposed to the previous Russian Flu strain.

Researchers have recently examined the immune reactions to the Spanish Flu strain and found that the outcomes in young adult populations could be attributed to a T-cell mediated response that caused an overactive immune system to begin to attack their own bodies (Shanks & Brundage, 2012). Other than age, studies have also considered gender and race as causal factors but have not found significant correlations (Viboud *et al.,* 2012). If a future pandemic follows a similar pattern of manifestation in the young adult populations, then public health officials will want to provide targeted intervention techniques for these demographic groups in each city.

1.5 The Future of Pandemic Outbreaks

The 2009 swine-origin H1N1 pandemic has been one of the few pandemic outbreaks in the twenty-first century that caused for some panic in the public health community (Smith *et al.,* 2009). In the first few weeks of the outbreak, the virus had already spread to 30 different countries and infected hundreds of people (Smith *et al.,* 2009). The mutation pathway of the virus is not well understood and may have evolved in an intermediate host before infecting humans (Reid & Taubenberger, 2003). The Swine Flu virus was thought to originate in a swine host and then mutated into the human species (Smith *et al.,* 2009). According to another study, the 1918 Spanish Flu strain may have also originated in swine and followed a similar pattern in the 2009 outbreak (Zimmer & Burke, 2009). Although the 2009 strain was not as lethal as the Spanish Flu

strain, many scientists have found similarities in the pattern of transmission, which leads future researchers to believe that the next pandemic strain will be of the H1N1-swine variety (Smith *et al.*, 2009).

A study analyzed the relationship between meteorological parameters and the transmission of the 2009 pandemic strain (Steel *et al.*, 2011). They found peak activity in October 2009 was dependent on the relative humidity and temperature like the H3N2 seasonal strain (Steel *et al.*, 2011). Since the 2009 strain peaked in mid-October like the Spanish Flu, the dependence on temperature may be another factor that allowed the rapid distribution of the virus during the fall of 1918. A more recent study in 2017 also suggested that ambient temperature and absolute humidity had a role in the transmission of the H7N9 strain of Avian Flu in China (Liu *et al.*, 2017). Studies such as these suggest that despite various strains in circulation, ambient weather factors may have a role in the transmission of the flu virus.

Chapter 2: Materials and Methods

2.1 Data Collection

Historical health records from 1918 are sparse and not easily accessible. During the time of the pandemic, many public health officials were overwhelmed by the influx of patients and did not have the time to update accurate records. Often, health officials would do house visits where they would treat entire families and did not know the outcomes of their treatments after they left the home. Some individuals would recover, while others would die in a few days. Military records are some of the most well-kept records from the time, but still may contain gaps in the coverage for individuals. The estimations of the death toll from the 1918 Spanish Flu continue to increase as more information is collected but may still not show an accurate representation of the entirety of the affected population. In order to obtain the most accurate records for this study, mortality data from the 1918 Department of Commerce, Bureau of the Census was utilized for Indianapolis, IN and Philadelphia, PA from September 1st, 1918 to December 31st, 1918 (Appendix A; Appendix B). Data from Kansas was also included in these records but were omitted due to the lack of coverage for the pandemic time span. The records are published in *Special Tables of Mortality from Influenza and Pneumonia* and separated into primary and secondary causes of influenza and pneumonia (Appendix A; Appendix B). Many individuals who were infected with the Spanish Flu developed bacterial pneumonia as a secondary factor and died from complications after the initial disease (Brundage & Shanks, 2008). This study utilized both the influenza and

pneumonia deaths as counts of mortality due to the interdisciplinary nature of the viral pathogenicity and, more practically, because the historical records conflated the two diseases in the mortality records. Mortality data was chosen due to the confirmation of influenza causality as opposed to the unknown causes in patients exhibiting flu-like symptoms. Separation of male and female deaths were also included in the data but not analyzed in this study.

In South Carolina, a medical officer at the *Sixth Naval District* in Charleston, SC kept daily records of cases treated outside of his office from September 25th, 1918 to November 12th, 1918 (Appendix C). These records were transcribed into Excel documents and used to indicate the impact of naval cases as a comparison to the civilian cases. Public records from the Charleston County Library were found in the *Return of Deaths Within the City of Charleston, SC*, and included alphabetized death records from January 1, 1907 – December 31, 1926 (Appendix D). These records were used to extract information for 314 individuals who had perished during the 1918 Spanish Flu (Appendix D). The document headings are written as: Name, Sex, Color, Date of Death, Cause of Death, Place of Death, Age, Place of Nativity, Attending Physician or Coroner, and Place of Internment (Appendix D). Records were analyzed from both white and black mortality records on each of the film reels. Each individual field was transcribed into an Excel document and then shared for future use.

The online National Oceanic and Atmospheric (NOAA) database contains a Global Historical Climatology Network (GHCN) of documents from the early 1900s that have been digitized and include daily temperature readings from most major cities across the United States. Weather stations were selected for this study based on the closest

proximity to the city center and highest amount of coverage relative to the cities of interest (Figure 3). The selected weather stations and coordinates are listed in the table below.

City	Station Code	Coordinates
Philadelphia, PA	USC00366194	40.1482, -74.953
Indianapolis, IN	US1INMR0051	39.78333, -85.76667
Charleston, SC	USW00013782	32.775, -79.9239

The locations of each city and selected weather stations are shown in **Figure 3.**

The temperature variables from 1918 were published as daily high and low temperatures and labeled as T-MAX and T-MIN for reference. The daily average values (T-MEAN) and daily temperature range values (T-RANGE) were calculated from the given parameters as further independent variables for this study. A few days in the datasets also included precipitation and snowfall but were omitted from this study due to lack of coverage in the data.

2.2 Statistical Tests

Upon initial examination of the data, the case counts, and temperature values were graphed as a scatter plot to find a correlation between increasing and decreasing temperature and the peak mortality rates in each city. The distribution of each data set was tested for normality but were not found to follow a normal distribution. To determine the overall relationship between the mortality and temperature variables, a non-parametric method was necessary for proper analysis. SPSS Software was used to conduct a Wilcoxon-Signed-Rank test. This test analyzes each individual data point and

finds the marginal distance from the mean and combines them for an overall comparison of the dataset in a non-parametric test (Field, 2014).

Most time series models of diseases are used to predict future seasons of the flu rather than finding correlations between outside variables and the accounts of flu (Lofgren *et al.*, 2007). Studies have also attempted to predict the spread of the disease by photoperiod and latitude and longitudinal relationships (Prendergast, 2011). While methods are limited, the use of autocorrelation methods for time series can determine the relationship between the two variables of infectious diseases (Imai *et al.*, 2015). According to previous studies, a lag in the temperature values is necessary to align the peak in mortality for influenza with the peak change in temperature (Sundell *et al.*, 2016). Time series analysis of one response statistics (mortality) and a covariate (temperature) requires analysis using a time series regression model (Allard, 1998). JMP Pro Software was used to conduct an ARIMA model to find the most significant lag value for comparison of the two time series data sets. The selection criteria for statistical significance was set at 0.05* and 95% confidence intervals.

2.3 GIS Mapping

ArcGIS Pro software was used to produce an initial map that would show the overall coverage of the study area with points set at each of the weather stations using their coordinates provided above (Figure 3). The overall distribution of the selected cities shows the wide span of coverage on the eastern coast via the selected areas and the variability in the latitudes and longitudes.

The data transcribed from civilian death records in Charleston, SC included individual locations of each case of mortality. Each location was transcribed into the closest estimated modern address and added as a new field into the Excel document. Out of the 315 individuals, 22 points were omitted due to the lack of a complete address in the records. The data fields were imported into ArcGIS Pro and then geocoded into individual data points to indicate a location-based image of the overall distribution of deaths across the Charleston area (Figure 12). A historical 1919 map of Charleston was downloaded from the Perry-Castañeda Library Map Collection from the United States Department of the Interior Geological Survey and used as a base map to ensure the highest degree of accuracy (Figure 12). A heat map of the point density of the number of deaths was created through a symbology modification (Figure 13).

Chapter 3: Results

3.1 Philadelphia Results

Philadelphia, PA had the largest amount of data coverage from September 1st, 1918 to December 31st, 1918 with 13,941 recorded cases of mortality over the 122-day period (Table 2). A pandemic curve was developed from this data with peak mortality of 803 cases on October 11th, 1918 (Figure 4). Daily temperature values ranged from 13°F to 86°F over the course of the season (Table 2). A gradual decrease in average temperature continued with slight variations in daily temperature (Figure 5). The greatest daily change in temperature was 43°F on October 10th, 1918, one day prior to the pandemic peak (Table 2). To compare the temperature variables to the daily counts of mortality, a non-parametric Related Samples Wilcoxon Signed Rank Test was used with manual lags of the data from 0 to 5 days and increased to 10 days for T-RANGE (Table 3). Initial results showed a significant correlation between the T-RANGE values and the cases of mortality (Table 3). In order to account for time series data and the relationship between various days, an ARIMA model was used to determine the possible lags between peak mortality and the temperature variables (Table 4). Each of the models showed significant results at the 0.01 confidence level for each of the temperature variables throughout each of the lag values (Table 4). A scatter plot of the T-RANGE and cases of mortality for the data and the 2-day manual lag with R^2 values of 0.1137 (no lag) and 0.0981 (2-day lag) (Figure 6). Figure 11 shows the relationship between the daily T-

RANGE and cases of mortality with the corresponding peaks approximately 2 days apart (Figure 7).

3.2 Indianapolis Results

The city of Indianapolis, IN had a much smaller population in 1918 than that of Philadelphia, PA and recorded only 1,033 cases of mortality during the same time period from September 1st, 1918 to December 31st, 1918 (Table 5). Peak mortality was recorded on October 18th, 1918 with 29 deaths (Figure 8). Indianapolis continued to show decreasing cases of mortality throughout the rest of November 1918 with a smaller secondary peak of 23 cases on November 24th, 1918 (Figure 8). Daily temperature values also exhibited a gradual decrease in temperature over the season with a few fluctuations in the values (Figure 9). Temperature values ranged from 17°F to 85°F with the greatest T-RANGE of 36 degrees on October 17th, 1918, one day prior to the pandemic peak (Table 5). Related-Samples Wilcoxon Signed Rank Tests showed significant results for each of the temperature values for all the manually lagged days with 95% confidence (Table 6). ARIMA models also showed significant findings with the greatest probability for 2- and 4-day lags for T-RANGE (Table 7). Significant values were also found for 0- and 5-day lags for T-MEAN and T-MAX (Table 7). T-MIN showed significant results for 2- and 4-day lags (Table 7). A scatter plot of the T-RANGE variable and cases of mortality explained the best fit of the data with an R^2 value of 0.0116 (no lag) and 0.0612 (2-day lag) (Figure 10).

3.3 Charleston Results

Data collection from Charleston, SC included two separate datasets with naval case counts and civilian mortality records (Table 8) (Table 9). Data included from the

Charleston Naval base was recorded over a 45-day span from September 25th, 1918 to November 8th, 1918 (Table 8). These records included daily visits from a medical health officer in the Sixth Naval District (Appendix C). Civilian records were recorded from the Charleston Return of Deaths reel from the Charleston Public Library from October 1st, 1918 to February 28th, 1919 (Appendix D) (Table 9). Individual records of 151 civilians including dates and locations of deaths were extracted from historical data for analysis (Table 9). The locations of each death were geocoded into ArcGIS Pro and displayed on a historic 1919 Charleston map (Figure 12). Included in the map are the 5 main hospital centers that were used for treatment; Baker Sanatorium, Charleston Orphan House, Riverside Infirmary, Roper Hospital, and St. Francis Infirmary (Figure 12). A secondary analysis of the point density was included to show the number of deaths that occurred in each region (Figure 13). ArcGIS Pro was used to integrate the points into a heat map image based on the density of deaths. The highest densities are recorded around the main hospital centers and a small region on the north east side of the peninsula (Figure 13).

Access to civilian and naval records showed a shift in the dispersal from military personnel to civilians with about a 1 to 2-week lag (Figure 16). To conduct the statistical analysis, naval and civilian records were combined for a wider range of records (n=196) (Figure 17). Combined data showed a peak on October 7th, 1918 with 38 records (Figure 17). Most cases of influenza were sparse after November 1918 with a few small outbreaks in mid-January and February 1919 (Figure 17).

Temperature data showed a gradual decrease in the seasonal temperature with a few sharp drops in temperature around mid-October and late-December 1918 (Figure 18). The largest change in temperature range occurred on January 3rd, 1919 of 34 degrees

(Table 9). During the time of the pandemic, the greatest change in temperature occurred on October 6th, 1918 of 20 degrees, one day prior to the pandemic peak (Figure 19). Scatter plots of mortality and temperature range were created to see the general relationship between the variables (Figure 20). Calculations of R^2 values did not show a strong correlation with values of 0.0014 (no lag) and 0.0009 (2-day lag) (Figure 20). Nonparametric tests for relationships between the variables showed highly significant results (<0.0001*) for each of the temperature values and mortality (Table 10). ARIMA models also showed strong correlations between each variable from 1 to 5-day lags (Table 11). Temperature range was extended to a 10-day lag and showed strong correlations from 1-9-day lags with 95% confidence (Table 11).

The detail included in the Charleston data set allowed for further analysis of the demographic data to include race, gender, and age (Appendix D). Age-related mortality from the Spanish Flu typically follows a W-shaped curve with the highest levels of mortality amongst mid-20 to 30-year-old individuals (Taubenberger & Morens, 2006). This curve was further supported by the Charleston civilian data set with the highest amount of deaths from age 18 to 40 with the highest number of deaths at age 25 (Figure 20). Records from the historical data were separated by race to include white, black, and colored individuals (Appendix D). Races were distributed by 52% white, 15% black, and 33% colored (Figure 22B). Gender was closely distributed with 43% male and 57% female individual deaths (Figure 22A).

Chapter 4: Discussion

4.1 Overall Discussion

Historical medical records from 1918 have been difficult for researchers to access due to lack of modern digitization and poorly kept records. Mortality records are the easiest to obtain and aim to confirm causality with Spanish Influenza. Often, secondary causes of influenza deaths were recorded as pneumonia and can serve as additional counts of mortality during the pandemic. Confirmed cases of mortality for the three study areas, Philadelphia, Indianapolis, and Charleston were utilized to analyze the relationship between the daily counts of mortality and the change in temperature (T-RANGE).

4.2 Philadelphia

Philadelphia, PA had the largest range of coverage and the most distinguished pandemic peak out of the three cities (Figure 4). Peak mortality was exhibited on October 11th, 1918 which follows the national scale of mortality from the heroic wave with most cities peaking in mid-October. The greatest change in diurnal temperature existed one day prior to the pandemic peak with a 43-degree difference from high and low temperatures. Without considering the time series analysis of the variables, the daily cases of mortality and diurnal temperature had a significant relationship at a one- and two-day lag at the 0.05 level (Table 3). Maximum temperature, minimum temperature, and average temperature did not produce significant results when compared to the number of deaths (Table 3). Using the T-RANGE as an explanatory variable, the data

was graphed against the cases of mortality (Figure 6). As the number of cases of mortality increased, so did the range of temperature during the mid-October months (Figure 6). However, in comparison to the entire time span, the data only explains 9% of the variability, overall and 11% when considering the 2-day lag (Figure 6). To ensure statistical tests for a time series, the ARIMA model was used to determine the possible lags and found a significant correlation between each of the lag days tested from 1 to10-day lags (Table 4). While temperature range may be a predictor for the annual flu season, the rapid transmission of the 1918 Spanish Flu may have been too lethal to determine a causal relationship between the outdoor temperature and cases of mortality in Philadelphia, PA. While the visual relationship can be shown between the peak change in temperature one day prior to the pandemic peak, the causation is not enough of an explanatory variable to be completely supported as the only causal factor (Figure 7).

4.3 Indianapolis

The city of Indianapolis had the least amount of data coverage in comparison to the other two cities. Due to the lower number of cases, the pandemic curve did not exhibit a singular strong peak, but two peaks in mid-October and mid-December (Figure 8). Although data coverage was consistent throughout the 122-day period for deaths and temperature, the accuracy of the statistical tests may not hold enough weight to fully support the hypothesis (Table 5). The Wilcoxon-Signed Rank test showed a highly significant relationship between the case count and temperature variables at the 0.05 level for all lag days (Table 6). The ARIMA model only showed significant correlations between T-RANGE and case count at 2- and 4-day lags (Table 7). Although significant values were found for T-MAX, T-MIN, and T-MEAN at varying days, the further

support from the Philadelphia case study would suggest that the T-RANGE has the greatest significance on the case count rather than the other variables (Table 3) (Table 4) (Table 7). The scatter plot of T-RANGE and cases with a 2-day lag only explained 1% and 6% of the relationship, respectively (Figure 10). Although this figure does not consider the time series relationship of the variables, the results are like those found in the Philadelphia study (Figure 6) (Figure 10). This suggests that when time is taken into consideration, the temperature has a significant relationship with the amount of mortality cases within two days prior to the pandemic peak. Again, the visual comparison of the case counts overlaid with the temperature range shows a peak in the daily change in temperature around 1 to 2 days prior to the peak (Figure 11). While the curves are not as pronounced as those in the Philadelphia data set, this pattern is similar in both case studies (Figure 7) (Figure 11).

4.4 Charleston

The data utilized in the Charleston study was initially separated by naval and civilian records and the final analysis was conducted on the combined dataset (Table 8) (Table 9). The coverage of the data in Charleston was also not as vast as the one for Philadelphia but provided a longer range of data from September 1918 to March 1919. An initial qualitative analysis between the naval and civilian datasets shows the gradual movement of the virus from the naval population to the civilians a few weeks later (Figure 16). The peak in the naval population was on October 7th, 1918 while the civilian population did not reach peak activity until October 18th, 1918 (Figure 16). The 11-day lag between the two populations suggests that the spread of the virus was most likely initiated from military personnel and transferred to the general population about a week

and a half later. However, the dataset for the naval population is a count of cases while the civilian population records mortality. A study found that the average number of days between onset of symptoms to mortality in naval and civilian populations was between 7-11 days (Klugman *et al.*, 2009). This finding further supports the spread of disease between civilian and naval populations.

While the naval base was in the upper portion of the Charleston peninsula, precise locations of the cases used in this study were not included in the original record (Appendix D). Therefore, this data was omitted from the visual map produced for the downtown Charleston region. The map created from the civilian data has confirmed address locations of each death and were geocoded in ArcGIS Pro to represent each person at their final place of death (Figure 12). The main hospital centers at the time were Baker Sanatorium, Charleston Orphan House, Riverside Infirmary, Roper Hospital, and St. Francis Infirmary which are represented on the map by individual points (Figure 12). Many of the individuals recorded in the study also perished at these locations. The selected weather station used in the study is also represented on this map (Figure 12). The widespread distribution of points shows that the virus spread rapidly and evenly throughout the downtown Charleston region (Figure 12). Overall, this map provides a new perspective on the impact of disease in the Charleston area, and could provide historians with vital information for families and historical narratives about the city.

A heat map of the density of deaths was created to show the distribution and clusters of deaths on the peninsula (Figure 13). The regions with the most deaths were located around the hospital centers due to the volume of people being served in these facilities (Figure 13). Over 40 individual deaths were recorded at Roper Hospital (Figure

13). The most unique portion of the map relates to a cluster of deaths in the top right portion of the peninsula (Figure 13). Upon closer examination of the points located in this cluster, it was found that most of these individuals were of African American descent (Figure 12). After further analysis, the historical data showed that this region was designated for Freedman Cottages built for newly freed African American slaves following the Civil War (D'Aquisto, 2006). At the time, many African Americans were either prohibited from visiting certain health facilities or were afraid to go due to prejudice following the Civil War. Many of them were forced to battle the disease in their homes instead, and thus had higher rates of mortalities in their neighborhoods. This smaller region of high density of deaths explains the demographic dispersal across the peninsula and adds to the racial disparities of this group at the time of the pandemic.

The combined dataset shows a significant peak in the case count in early October and a smaller peak in mid-January with 6 cases recorded on January 20th, 1919 (Figure 17). This city is the only case study that has extensive data coverage through the 1919 months beyond the pandemic period. This finding further supports the previously published data that suggests a second smaller wave of influenza following the herald wave in October 1918 in most cities (He *et al.*, 2011; Chowell *et al.*, 2010). Some studies have also suggested a minor wave in the summer prior to the pandemic peak, but data was not available before September 1918 for these cities (Andreason *et al*., 2008).

The specificity of the data also included ages for civilians in Charleston and was sorted into age groups from 0 to 82 years old and graphed to visualize the distribution (Figure 21A). The W-shaped curve as suggested by many other researchers was further supported from the data found in Charleston and showed the greatest number of deaths

with individuals ages 18 to 40 with the highest amount of deaths at age 25 (Figure 21A). The normal curve of mortality shows the highest influenza deaths at the youngest and oldest ages, but the Spanish Flu has continually shown a unique peak in mortality amongst young adult populations (Figure 21B). Researchers have attributed this shift in mortality to an overactive immune response of healthy young adult individuals and a lack of prior immunity (Langford, 2002; Viboud *et al*., 2012; Taubenberger & Morens, 2006). This dataset further supports this argument and contributes to the growing evidence of a young adult overactive immune response.

Historical medical records from 1918 are lacking, especially for marginalized groups of individuals. The Charleston data set was also categorized by race and gender as printed on the historical death records (Appendix D). A simple analysis of the overall distribution of deaths by gender was 43% male and 57% female in the civilian population (Figure 22A). The slightly higher number of females in the civilian population may be attributed to a large majority of males serving in the military for the war. If gender were included with the naval population as male, the overall ratio would be more even. The categorization of white and black civilians were place into separate death records and some individuals were also labeled as "C" for "colored" rather than "B" for "black" (Appendix D). The reasoning behind this categorization was due to African American class systems that separated affluent groups into "colored" and poorer groups as "blacks." The overall distribution of individuals in the set were 51% white, 15% black, and 33% colored (Figure 22B). This data set can serve as one of the most complete record systems for African Americans during the 1918 Spanish Flu pandemic.

Statistical analysis of the Charleston data was conducted on the combined civilian and naval records over the time span from October 1918 to March 1919. Initial comparison of the temperature variables and case counts found a significant correlation at the 0.05 level for all variables and all lags (Table 10). This finding from the Wilcoxon Signed Rank test suggests that the variables without respect to time are significantly different than the number of deaths in the population. The ARIMA model also showed significant values for all variables and lag days except for 0- and 10-day lags with T-RANGE (Table 11). While the dataset does not have as many recorded deaths as those in the Philadelphia dataset, the findings further suggest a significant correlation between the temperature and peak mortality from 1 to 9-day lags (Table 11).

Chapter 5: Conclusion

5.1 Research Conclusions

The findings in this study suggest that a significant relationship was found between the temperature changes (T-RANGE) and the peak mortality in the pandemic with an average of a 1 to 2-day lag in each city. The lethality of the virus caused the disease to spread rapidly throughout the population and increased mortality to a higher than average rate, especially for young adult populations.

Influenza typically takes 1-4 days to exhibit symptoms from onset to infection and can infect an isolated population of individuals within 72 hours (Moser *et al.*, 1979). The 1918 Spanish Flu strain followed a similar pattern of transmission with most individuals showing symptoms within 1-5 days, yet mortality may follow within that timeframe or shortly after (Cumpston, 1919). A two-day lag in the pandemic between the change in temperature and peak mortality may be too close for a causal factor but may have a role in the individual's immune response. A drastic change in temperature may have had a negative impact on the recovery success to the vulnerable infected population. The individuals who perished within the 2-day time frame would have already been infected with the virus at the time that the drastic change in temperature occurred. This suggests that the passage of the virus from person to person may not be influenced by the ambient temperature, but the host and behavioral responses must be further examined. A previous study that analyzed seasonal flu with a multi-factorial approach found a 3% relationship with humidity levels, no relationship with school and holiday schedules, and 27%

"between seasons" effects (teBeest *et al.*, 2013). The sudden changes in temperature (T-RANGE) found in this study may serve as an explanation for the "between season" effect found in the previous study (teBeest *et al.*, 2013).

As with any historical data set, limitations exist for data coverage and information about individuals (White, 2010). More historical records are becoming digitized and providing more information to researchers on the impact of the 1918 Spanish Flu and other pandemic diseases (White, 2010). This study provides a further count of mortality for three North American cities with a detailed count of individuals in Charleston.

5.2 Public Health Implications

The World Health Organization currently has published guidelines to follow in the event of a pandemic outbreak including quarantine measures, social distancing measures, and personal protection measures as nonpharmaceutical interventions (Group, 2006). However, local measures such as distribution of antivirals and vaccine initiatives are lacking. Modern medicine has introduced the use of antivirals to reduce the severity of influenza when treated early in the process. Antiviral Neuraminidase inhibitors have also been found to be an effective treatment for critically-ill patients with H1N1 (Louie *et al.*, 2012). While vaccines may take months to produce, antivirals may be the best alternative to provide an effective immune response during a pandemic.

Advancements in the internet activity across the globe have allowed us to track patterns in the population in a greater manner than ever before. Researchers have utilized Google to examine the number of people searching for flu-related symptoms and found a correlation between increased searches and increased hospital visits during the annual flu seasons (Dugas *et al.*, 2012).

Education of the public will also be essential in alleviating the burden of a pandemic outbreak. Household based intervention methods have also been proposed such as voluntary quarantine and keeping antivirals at home for easy access (Wu *et al.,* 2009).

5.3 Potential Public Administration Outreach

Various studies can be conducted prior to the next pandemic such as surveillance studies in hospitals as well as group cohort studies to assess the general knowledge of the population (J. Abramson & Z. Abramson, 2011). Modern surveillance techniques would allow epidemiologists to better predict future pandemics with early surveillance and possibly allow for enough time to produce a vaccine by the peak rate of transmission (Miller *et al.,* 2009). Once the vaccines are created, a question of distribution priority also comes into question when determining priorities in demographic groups. If the age-related mortality of future pandemics follows the similar pattern of the 1918 Spanish Flu, then public health officials should prioritize treating young-adult age groups over those who are younger and elderly (Miller *et al.,* 2009). Ideally, all individuals would be able to have access to the treatments, but priorities during a shortage must be planned for accordingly.

Predictions for future pandemics can be determined with monitoring software and digitization of health records. The 2009 Swine Flu pandemic was the first opportunity that surveillance techniques could be tested in the United States. A study of the southeastern coast analyzed and successfully predicted the spatial transmission of the 2009 swine flu and compared to confirmed cases (Pei *et al.,* 2017). Similar measures could be implemented and improved in future pandemics.

5.4 Future Directions

Findings that show a fluctuation or sudden change in temperature may provide new information to prepare for sudden outbreaks of influenza. However, with climate change, this also brings greater fluctuations in temperature which would therefore increase the possibility of flu-related outbreaks more often or sooner in the seasons (Ebi & Mills, 2013). One study suggested that warmer winters could lead to less influenza infections, but climate change would not cause every area of the globe to have warmer winters (Urashima *et al.*, 2003). The overall distribution of weather patterns across the globe will have high levels of variation and will require a focused approach to target areas of highest need during pandemic outbreaks.

The current distribution of vaccinations is based on the currently circulating viruses at the time of manufacturing. Twice each year, the World Health Organization gets together to determine the circulating viruses for the Northern and Southern Hemispheres (Gerdil, 2003). The FDA makes the final decision in the United States around February each year, well before the following season (Gerdil, 2003). While this method can be beneficial at times, this often leads to the lowered level of efficacy in the vaccinations each year. Viral mutations may cause the manufactured vaccinations to become ineffective in short amounts of time. The process to develop a vaccine may shorten as advancements are made in manufacturing, but the current dilemma remains the same.

The multi-disciplinary approach to combatting influenza with environmental factors is a new field of research that can be beneficial to public health and biological research. As scientists learn more about the individual susceptibility of influenza related to social aspects, we can better understand the underlying principles that influenza the

passage of the virus throughout populations. Temperature has continued to be a strongly correlated variable and should be continually researched in future studies.

References

Abedi, G. R., Prill, M. M., Langley, G. E., Wikswo, M. E., Weinberg, G. A., Curns, A. T., & Schneider, E. (2014). Estimates of parainfluenza virus-associated hospitalizations and cost among children aged less than 5 years in the United States, 1998–2010. *Journal of the Pediatric Infectious Diseases Society*, *5*(1), 7-13.

Abramson, J., & Abramson, Z. H. (2011). Research methods in community medicine: surveys, epidemiological research, programme evaluation, clinical trials. *John Wiley & Sons.*

Acuna-Soto, R., Viboud, C., & Chowell, G. (2011). Influenza and pneumonia mortality in 66 large cities in the United States in years surrounding the 1918 pandemic. *PLoS One*, *6*(8), e23467.

Ahmed, R., Oldstone, M. B., & Palese, P. (2007). Protective immunity and susceptibility to infectious diseases: lessons from the 1918 influenza pandemic. *Nature Immunology*, *8*(11), 1188.

Allard, R. (1998). Use of time-series analysis in infectious disease surveillance. *Bulletin of the World Health Organization*, *76*(4), 327.

Andreasen, V., Viboud, C., & Simonsen, L. (2008). Epidemiologic characterization of the 1918 influenza pandemic summer wave in Copenhagen: implications for pandemic control strategies. *The Journal of Infectious Diseases*, *197*(2), 270-278.

Azziz Baumgartner, E., Dao, C. N., Nasreen, S., Bhuiyan, M. U., Mah-E-Muneer, S., Mamun, A. A., ... & Widdowson, M. A. (2012). Seasonality, timing, and climate drivers of influenza activity worldwide. *The Journal of Infectious Diseases*, *206*(6), 838-846.

Barry, J. M. (2004). The site of origin of the 1918 influenza pandemic and its public health implications. *Journal of Translational Medicine, 2*(1), 3.

Bonilla, E., Valero, N., Chacín-Bonilla, L., & Medina-Leendertz, S. (2004). Melatonin and viral infections. *Journal of Pineal Research, 36*(2), 73-79.

Bootsma, M. C., & Ferguson, N. M. (2007). The effect of public health measures on the 1918 influenza pandemic in US cities. *Proceedings of the National Academy of Sciences, 104*(18), 7588-7593.

Brundage, J. F., & Shanks, G. D. (2008). Deaths from bacterial pneumonia during 1918–19 influenza pandemic. *Emerging Infectious Diseases, 14*(8), 1193.

Byerly, C. R. (2010). The US military and the influenza pandemic of 1918–1919. *Public Health Reports, 125*(3_suppl), 81-91.

Cannell, J. J., Vieth, R., Umhau, J. C., Holick, M. F., Grant, W. B., Madronich, S., ... & Giovannucci, E. (2006). Epidemic influenza and vitamin D. *Epidemiology & Infection, 134*(6), 1129-1140.

Chattopadhyay, I., Kiciman, E., Elliott, J. W., Shaman, J. L., & Rzhetsky, A. (2018). Conjunction of factors triggering waves of seasonal influenza. *Elife, 7*, e30756.

Chew, F. T., Doraisingham, S., Ling, A. E., Kumarasinghe, G., & Lee, B. W. (1998). Seasonal trends of viral respiratory tract infections in the tropics. *Epidemiology & Infection, 121*(1), 121-128.

Chowell, G., Bettencourt, L. M., Johnson, N., Alonso, W. J., & Viboud, C. (2007). The 1918–1919 influenza pandemic in England and Wales: spatial patterns in transmissibility and mortality impact. *Proceedings of the Royal Society B: Biological Sciences, 275*(1634), 501-509.

Chowell, G., Viboud, C., Simonsen, L., Miller, M. A., & Acuna-Soto, R. (2010). Mortality patterns associated with the 1918 influenza pandemic in Mexico: evidence for a spring herald wave and lack of preexisting immunity in older populations. *The Journal of Infectious Diseases*, *202*(4), 567-575.

"Circus Problem up to Governor," *Charleston Evening Post*, 27 Sept. 1918, 13; "Circus Tours Limited," *Charleston Evening Post*, 30 Sept. 1918, 4.

"College Closes for the Present," *Charleston News and Courier*, 4 Oct. 1918, 8; "Red Cross to Help Fight Influenza," *Charleston News and Courier*, 4 Oct. 1918, 6;

Cooper, B. S., Pitman, R. J., Edmunds, W. J., & Gay, N. J. (2006). Delaying the international spread of pandemic influenza. *PLoS Medicine, 3*(6), e212.

Cumpston, J. H. L. (1919). Influenza and maritime quarantine in Australia (No. 18). *Issued under the Authority of the Minister for Trade and Customs*, AJ Mullett, Government printer.

D'Aquisto, L. (n.d.). *"Freedmans Cottage" Project Collection* [Scholarly project]. Retrieved from https://avery.cofc.edu/archives/Freedmans_Cottage.pdf

Davis, R. E., Rossier, C. E., & Enfield, K. B. (2012). The impact of weather on influenza and pneumonia mortality in New York City, 1975–2002: a retrospective study. *PLoS One*, *7*(3), e34091.

Doshi, P. (2008). Trends in recorded influenza mortality: United States, 1900–2004. *American Journal of Public Health*, *98*(5), 939-945.

Dowell, S. F. (2001). Seasonal variation in host susceptibility and cycles of certain infectious diseases. *Emerging Infectious Diseases*, *7*(3), 369.

Dugas, A. F., Hsieh, Y. H., Levin, S. R., Pines, J. M., Mareiniss, D. P., Mohareb, A., ... & Rothman, R. E. (2012). Google Flu Trends: correlation with emergency department influenza rates and crowding metrics. *Clinical Infectious Diseases*, *54*(4), 463-469.

du Prel, J. B., Puppe, W., Gröndahl, B., Knuf, M., Weigl, F., Schaaff, F., ... & Schmitt, H. J. (2009). Are meteorological parameters associated with acute respiratory tract infections?. *Clinical Infectious Diseases*, *49*(6), 861-868.

Ebi, K. L., & Mills, D. (2013). Winter mortality in a warming climate: a reassessment. *Wiley Interdisciplinary Reviews: Climate Change*, *4*(3), 203-212.

Field, A. (2014). *Discovering Statistics Using IBM SPSS Statistics* (4th ed.). London: Sage Publications.

Finkelman, B. S., Viboud, C., Koelle, K., Ferrari, M. J., Bharti, N., & Grenfell, B. T. (2007). Global patterns in seasonal activity of influenza A/H3N2, A/H1N1, and B from 1997 to 2005: viral coexistence and latitudinal gradients. *PloS One*, *2*(12), e1296.

Fox, S. J., Miller, J. C., & Meyers, L. A. (2017). Seasonality in risk of pandemic influenza emergence. *PLoS Computational Biology*, *13*(10), e1005749.

Fuhrmann, C. (2010). The effects of weather and climate on the seasonality of influenza: what we know and what we need to know. *Geography Compass*, *4*(7), 718-730.

Gerdil, C. (2003). The annual production cycle for influenza vaccine. *Vaccine*, *21*(16), 1776-1779.

Green M. J., Report of the Health Officer, *City of Charleston, Year Book*, 1918.

Group, W. H. O. W. (2006). Nonpharmaceutical interventions for pandemic influenza, national and community measures. *Emerging Infectious Diseases*, *12*(1), 88.

He, D., Dushoff, J., Day, T., Ma, J., & Earn, D. J. (2011). Mechanistic modelling of the three waves of the 1918 influenza pandemic. *Theoretical Ecology*, *4*(2), 283-288.

Imai, C., Armstrong, B., Chalabi, Z., Mangtani, P., & Hashizume, M. (2015). Time series regression model for infectious disease and weather. *Environmental Research*, *142*, 319-327.

Johnson, N. P., & Mueller, J. (2002). Updating the accounts: global mortality of the 1918-1920" Spanish" influenza pandemic. *Bulletin of the History of Medicine*, *76*(1), 105-115.

Juckett, G. (2006). Avian influenza: preparing for a pandemic. *American Family Physician*, *74*(5).

Kilbourne, E. D. (2006). Influenza pandemics of the 20th century. *Emerging Infectious Diseases*, *12*(1), 9.

Klugman, K. P., Astley, C. M., & Lipsitch, M. (2009). Time from illness onset to death, 1918 influenza and pneumococcal pneumonia. *Centers for Disease Control.*

Langford, C. (2002). The age pattern of mortality in the 1918-19 influenza pandemic: an attempted explanation based on data for England and Wales. *Medical History*, *46*(1), 1-20.

Layne, S. P., Monto, A. S., & Taubenberger, J. K. (2009). Pandemic influenza: an inconvenient mutation. *Science*, *323*(5921), 1560-1561.

Linder, F. E., & Grove, R. D. (1943). *Vital Statistics Rates in the United States, 1900-1940*. US Government Printing Office.

Lipsitch, M., & Viboud, C. (2009). Influenza seasonality: lifting the fog. *Proceedings of the National Academy of Sciences*, *106*(10), 3645-3646.

Li, Y., Wang, X. L., & Zheng, X. (2018). Impact of weather factors on influenza hospitalization across different age groups in subtropical Hong Kong. *International Journal of Biometeorology*, *62*(9), 1615-1624.

Liu, T., Kang, M., Zhang, B., Xiao, J., Lin, H., Zhao, Y., ... & Ma, W. (2018). Independent and interactive effects of ambient temperature and absolute humidity on the risks of avian influenza A (H7N9) infection in China. *Science of the Total Environment*, *619*, 1358-1365.

Lofgren, E., Fefferman, N. H., Naumov, Y. N., Gorski, J., & Naumova, E. N. (2007). Influenza seasonality: underlying causes and modeling theories. *Journal of Virology*, *81*(11), 5429-5436.

Louie, J. K., Yang, S., Acosta, M., Yen, C., Samuel, M. C., Schechter, R., ... & Uyeki, T. M. (2012). Treatment with neuraminidase inhibitors for critically ill patients with influenza A (H1N1) pdm09. *Clinical Infectious Diseases*, *55*(9), 1198-1204.

Lowen, A. C., & Steel, J. (2014). Roles of humidity and temperature in shaping influenza seasonality. *Journal of Virology*, *88*(14), 7692-7695.

Lowen, A. C., Steel, J., Mubareka, S., & Palese, P. (2008). High temperature (30 C) blocks aerosol but not contact transmission of influenza virus. *Journal of Virology*, *82*(11), 5650-5652.

Lowen, A. C., Mubareka, S., Steel, J., & Palese, P. (2007). Influenza virus transmission is dependent on relative humidity and temperature. *PLoS Pathogens*, *3*(10), e151.

Martineau, A. R., Jolliffe, D. A., Hooper, R. L., Greenberg, L., Aloia, J. F., Bergman, P., ... & Goodall, E. C. (2017). Vitamin D supplementation to prevent acute respiratory tract infections: systematic review and meta-analysis of individual participant data. *bmj*, *356*, i6583.

Mathews, J. D., McBryde, E. S., McVernon, J., Pallaghy, P. K., & McCaw, J. M. (2010). Prior immunity helps to explain wave-like behaviour of pandemic influenza in 1918-9. *BMC Infectious Diseases*, *10*(1), 128.

Mathews, J. D., Chesson, J. M., McCaw, J. M., & McVernon, J. (2009). Understanding influenza transmission, immunity and pandemic threats. *Influenza and Other Respiratory Viruses*, *3*(4), 143-149.

Miller, M. A., Viboud, C., Balinska, M., & Simonsen, L. (2009). The signature features of influenza pandemics—implications for policy. *New England Journal of Medicine*, *360*(25), 2595-2598.

Moore, J. H. (1985). Charleston in World War I: Seeds of Change. *The South Carolina Historical Magazine*, *86*(1), 39-49.

Morens, D. M., & Taubenberger, J. K. (2009). Understanding influenza backward. *Jama*, *302*(6), 679-680.

Moser, M. R., Bender, T. R., Margolis, H. S., Noble, G. R., Kendal, A. P., & Ritter, D. G. (1979). An outbreak of influenza aboard a commercial airliner. *American Journal of Epidemiology*, *110*(1), 1-6.

Nicholls, H. (2006). Pandemic influenza: the inside story. *PLoS Biology*, *4*(2), e50.

"No New Flu Cases Up to 12 Yesterday," *Charleston News and Courier*, 7 Nov. 1918, 8; "Schools Open Monday," *Charleston News and Courier*, 8 Nov. 1918, 3.

Olsen, B., Munster, V. J., Wallensten, A., Waldenström, J., Osterhaus, A. D., & Fouchier, R. A. (2006). Global patterns of influenza A virus in wild birds. *Science*, *312*(5772), 384-388.

Olson, D. R., Simonsen, L., Edelson, P. J., & Morse, S. S. (2005). Epidemiological evidence of an early wave of the 1918 influenza pandemic in New York City. *Proceedings of the National Academy of Sciences*, *102*(31), 11059-11063.

Pearce, D. C., Pallaghy, P. K., McCaw, J. M., McVernon, J., & Mathews, J. D. (2011). Understanding mortality in the 1918–1919 influenza pandemic in England and Wales. *Influenza and Other Respiratory Viruses*, *5*(2), 89-98.

Pei, S., Kandula, S., Yang, W., & Shaman, J. (2018). Forecasting the spatial transmission of influenza in the United States. *Proceedings of the National Academy of Sciences*, *115*(11), 2752-2757.

Pons-Salort, M., Oberste, M. S., Pallansch, M. A., Abedi, G. R., Takahashi, S., Grenfell, B. T., & Grassly, N. C. (2018). The seasonality of nonpolio enteroviruses in the United States: Patterns and drivers. *Proceedings of the National Academy of Sciences*, *115*(12), 3078-3083.

Prendergast, B. J. (2011). Can photoperiod predict mortality in the 1918-1920 influenza pandemic. *Journal of Biological Rhythms*, *26*(4), 345-352.

"Pneumonia Ward Roper Hospital," *The Charleston Evening Post*, October 22, 1918.

Reichert, T. A., Simonsen, L., Sharma, A., Pardo, S. A., Fedson, D. S., & Miller, M. A. (2004). Influenza and the winter increase in mortality in the United States, 1959–1999. *American Journal of Epidemiology*, *160*(5), 492-502.

Reid, A. H., & Taubenberger, J. K. (2003). The origin of the 1918 pandemic influenza virus: a continuing enigma. *Journal of General Virology*, *84*(9), 2285-2292.

Saunders-Hastings, P., & Krewski, D. (2016). Reviewing the history of pandemic influenza: understanding patterns of emergence and transmission. *Pathogens*, *5*(4), 66.

Shaman, J., & Kohn, M. (2009). Absolute humidity modulates influenza survival, transmission, and seasonality. *Proceedings of the National Academy of Sciences*, *106*(9), 3243-3248.

Shanks, G. D., & Brundage, J. F. (2012). Pathogenic responses among young adults during the 1918 influenza pandemic. *Emerging Infectious Diseases*, *18*(2), 201.

Sheng, Z. M., Chertow, D. S., Ambroggio, X., McCall, S., Przygodzki, R. M., Cunningham, R. E., ... & Taubenberger, J. K. (2011). Autopsy series of 68 cases dying before and during the 1918 influenza pandemic peak. *Proceedings of the National Academy of Sciences*, *108*(39), 16416-16421.

Skehel, J. J., & Wiley, D. C. (2000). Receptor binding and membrane fusion in virus entry: the influenza hemagglutinin. *Annual Review of Biochemistry*, *69*(1), 531-569.

Steel, J., Palese, P., & Lowen, A. C. (2011). Transmission of a 2009 pandemic influenza virus shows a sensitivity to temperature and humidity similar to that of an H3N2 seasonal strain. *Journal of Virology*, *85*(3), 1400-1402.

Smith, G. J., Vijaykrishna, D., Bahl, J., Lycett, S. J., Worobey, M., Pybus, O. G., ... & Peiris, J. M. (2009). Origins and evolutionary genomics of the 2009 swine-origin H1N1 influenza A epidemic. *Nature*, *459*(7250), 1122.

Soebiyanto, R. P., Clara, W. A., Jara, J., Balmaseda, A., Lara, J., Moya, M. L., ... & Kiang, R. K. (2015). Associations between seasonal influenza and meteorological parameters in Costa Rica, Honduras and Nicaragua. *Geospatial Health*.

Sohn, K., & Boulier, B. L. (2012). Estimating Parameters of the 1918-19 Influenza Epidemic on US Military Bases. *Journal of Applied Business and Economics*.

Sundell, N., Andersson, L. M., Brittain-Long, R., Lindh, M., & Westin, J. (2016). A four year seasonal survey of the relationship between outdoor climate and epidemiology of viral respiratory tract infections in a temperate climate. *Journal of Clinical Virology*, *84*, 59-63.

Tamerius, J. D., Shaman, J., Alonso, W. J., Bloom-Feshbach, K., Uejio, C. K., Comrie, A., & Viboud, C. (2013). Environmental predictors of seasonal influenza epidemics across temperate and tropical climates. *PLoS Pathogens*, *9*(3), e1003194.

te Beest, D. E., van Boven, M., Hooiveld, M., van den Dool, C., & Wallinga, J. (2013). Driving factors of influenza transmission in the Netherlands. *American Journal of Epidemiology*, *178*(9), 1469-1477.

Tamerius, J., Nelson, M. I., Zhou, S. Z., Viboud, C., Miller, M. A., & Alonso, W. J. (2010). Global influenza seasonality: reconciling patterns across temperate and tropical regions. *Environmental Health Perspectives*, *119*(4), 439-445.

Taubenberger, J. K., & Morens, D. M. (2006). 1918 Influenza: the mother of all pandemics. *Emerging Infectious Diseases*, *12*(1), 15.

"The Spanish Flu at Training Camp," *Charleston Evening Post*, 17 Sept. 1918, 11

Tumpey, T. M., Basler, C. F., Aguilar, P. V., Zeng, H., Solórzano, A., Swayne, D. E., ... & Garcia-Sastre, A. (2005). Characterization of the reconstructed 1918 Spanish influenza pandemic virus. *Science*, *310*(5745), 77-80.

Urashima, M., Shindo, N., & Okabe, N. (2003). A seasonal model to simulate influenza oscillation in Tokyo. *Japanese Journal of Infectious Diseases*, *56*(2), 43-47.

Viboud, C., Alonso, W. J., & Simonsen, L. (2006). Influenza in tropical regions. *PLoS Medicine*, *3*(4), e89.

Viboud, C., Eisenstein, J., Reid, A. H., Janczewski, T. A., Morens, D. M., & Taubenberger, J. K. (2012). Age-and sex-specific mortality associated with the 1918–1919 influenza pandemic in Kentucky. *The Journal of Infectious Diseases*, *207*(5), 721-729.

White, P. (2010). Making use of secondary data. *Key Methods in Geography*, *2*, 61-76.

Webster, R. G., Bean, W. J., Gorman, O. T., Chambers, T. M., & Kawaoka, Y. (1992). Evolution and ecology of influenza A viruses. *Microbiological Reviews*, *56*(1), 152-179.

Willem, L., Van Kerckhove, K., Chao, D. L., Hens, N., & Beutels, P. (2012). A nice day for an infection? Weather conditions and social contact patterns relevant to influenza transmission. *PloS One*, *7*(11), e48695.

Wu, J. T., Riley, S., Fraser, C., & Leung, G. M. (2006). Reducing the impact of the next influenza pandemic using household-based public health interventions. *PLoS Medicine*, *3*(9), e361.

Yang, W., Elankumaran, S., & Marr, L. C. (2012). Relationship between humidity and influenza A viability in droplets and implications for influenza's seasonality. *PloS One*, *7*(10), e46789.

"Year Book," 1918, *City of Charleston, South Carolina*. Charleston, (1919)

Zhao, N., Cao, G., Vanos, J. K., & Vecellio, D. J. (2018). The effects of synoptic weather on influenza infection incidences: a retrospective study utilizing digital disease surveillance. *International Journal of Biometeorology*, *62*(1), 69-84.

Zimmer, S. M., & Burke, D. S. (2009). Historical perspective—emergence of influenza A (H1N1) viruses. *New England Journal of Medicine*, *361*(3), 279-285.

Figures

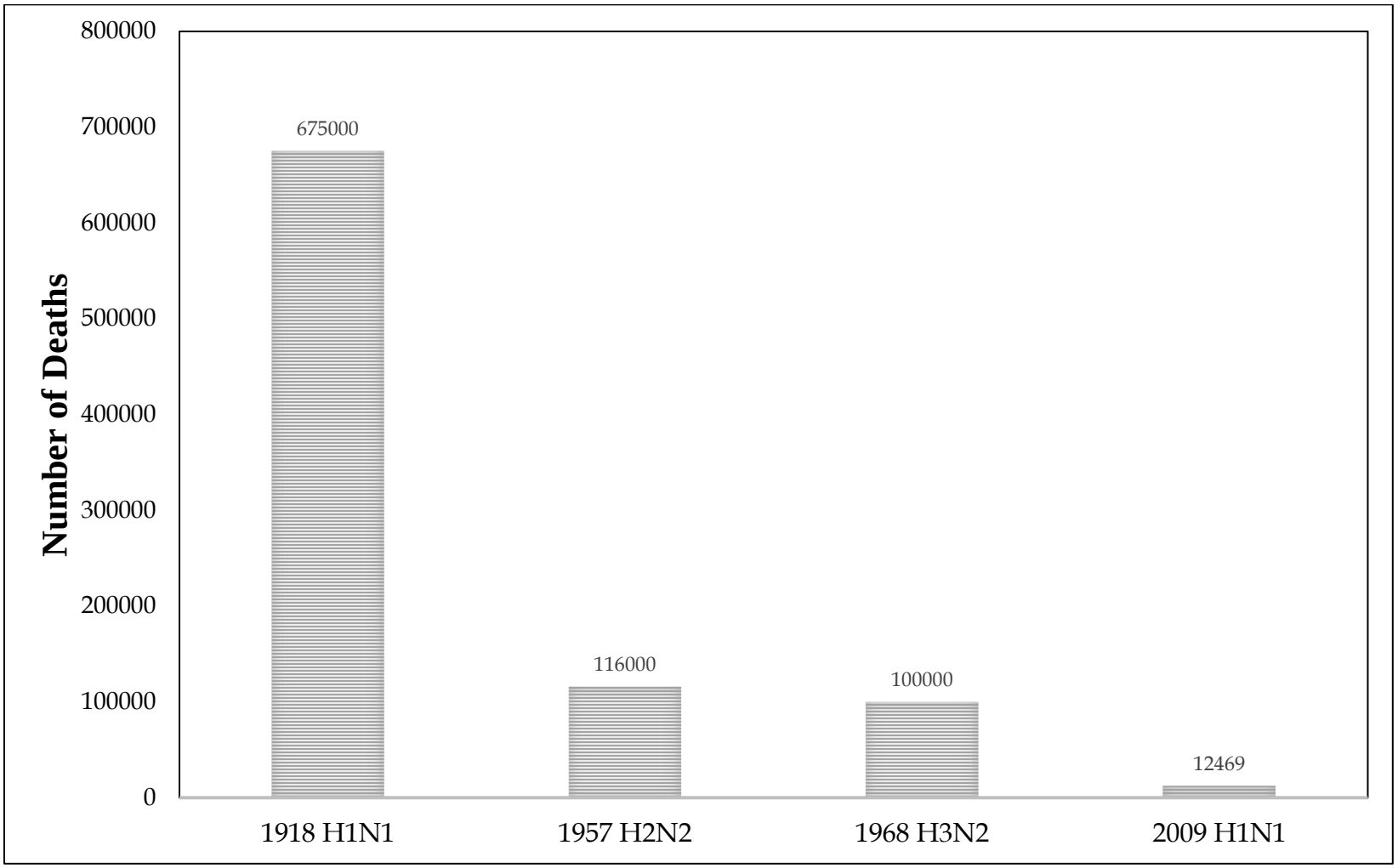


Figure 1. Mortality Impact of Previous Flu Pandemics in the United States (Saunders-Hastings and Krewski, 2016)

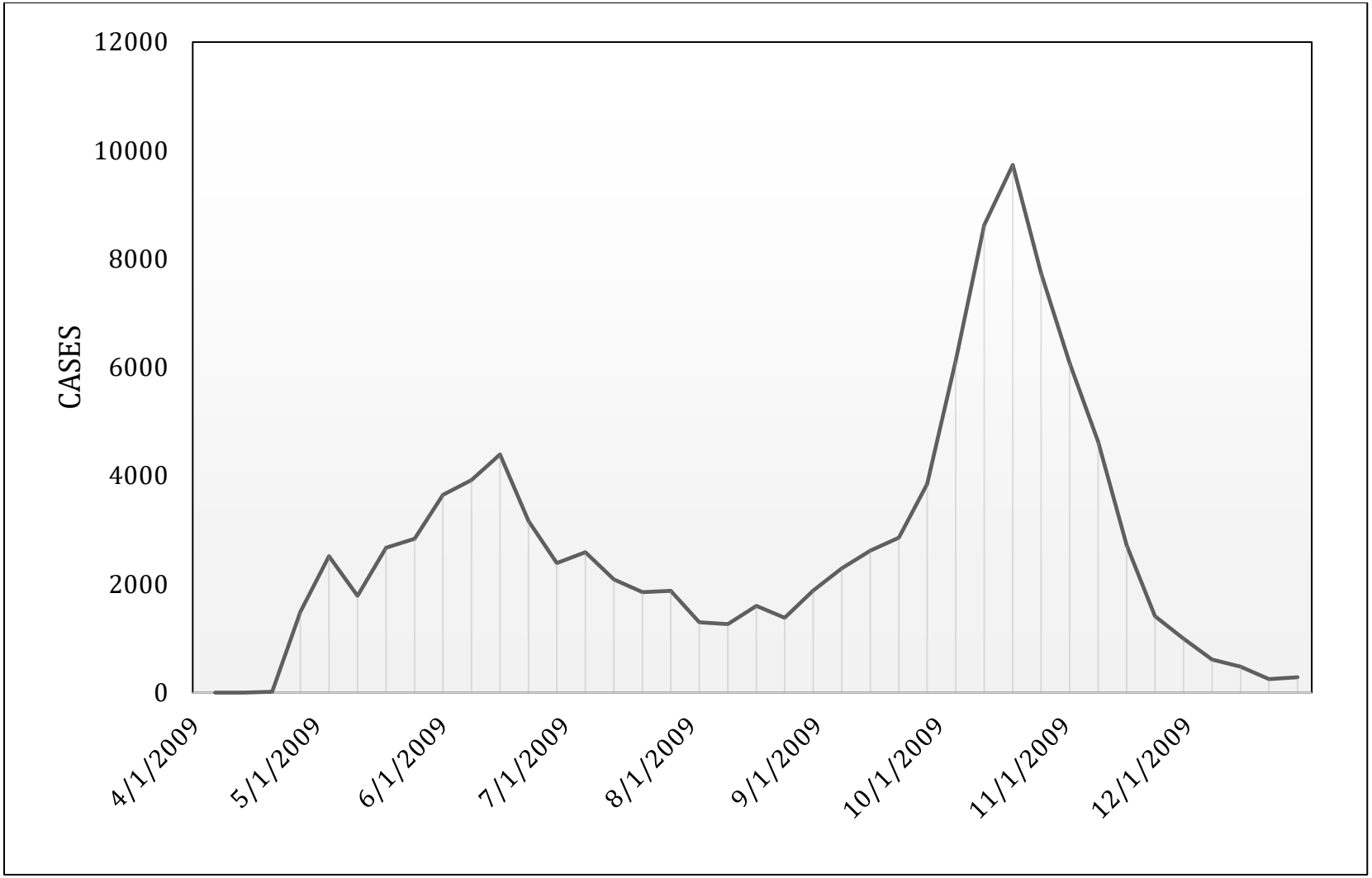


Figure 2. Pandemic Curve of 2009 Swine Flu Pandemic in the United States (WHO, Flu Net Available from: http://www.who.int/influenza/gisrs_laboratory/flunet/en/)

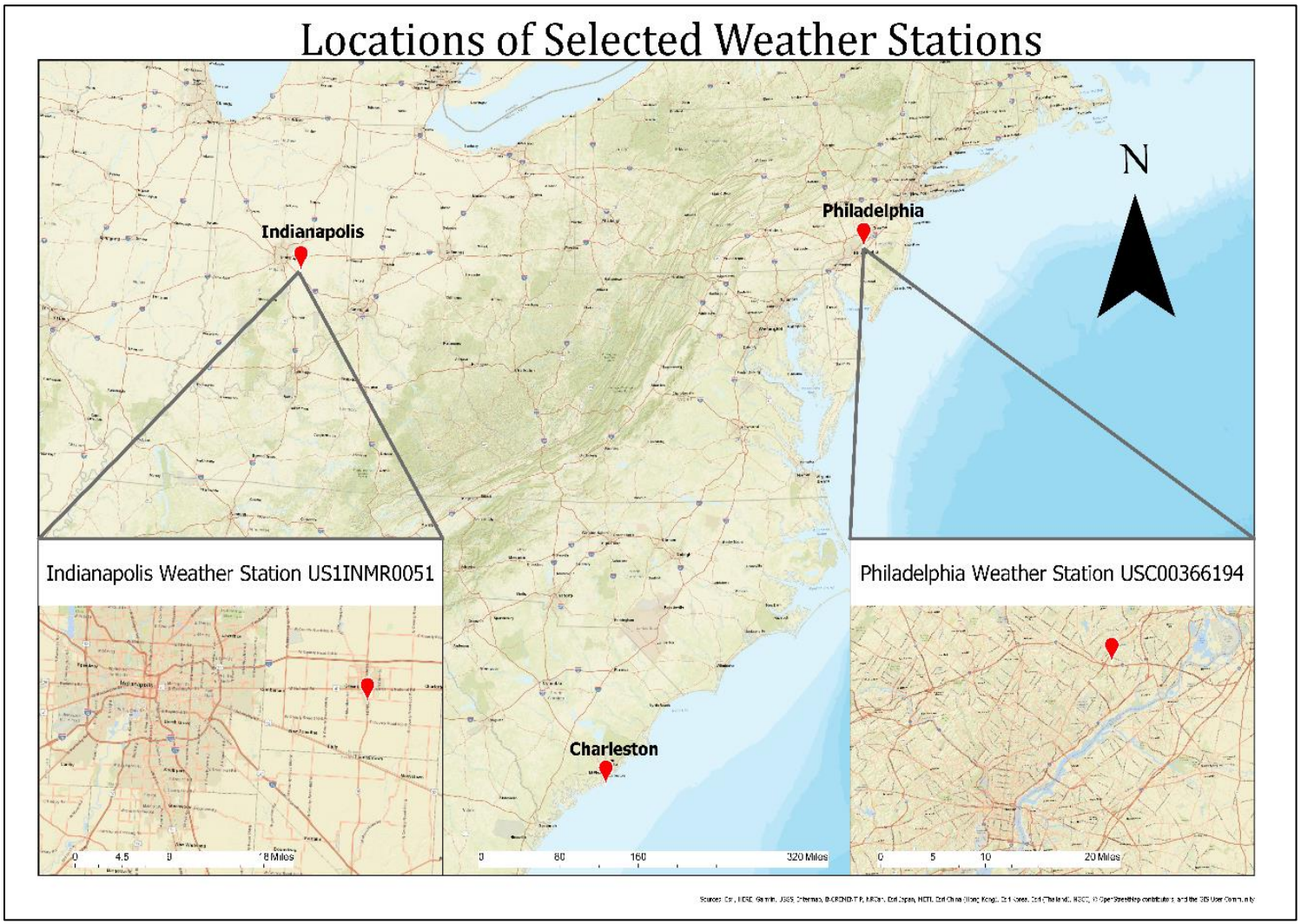


Figure 3. Map of Study Area and Selected Weather Stations in Indianapolis and Philadelphia

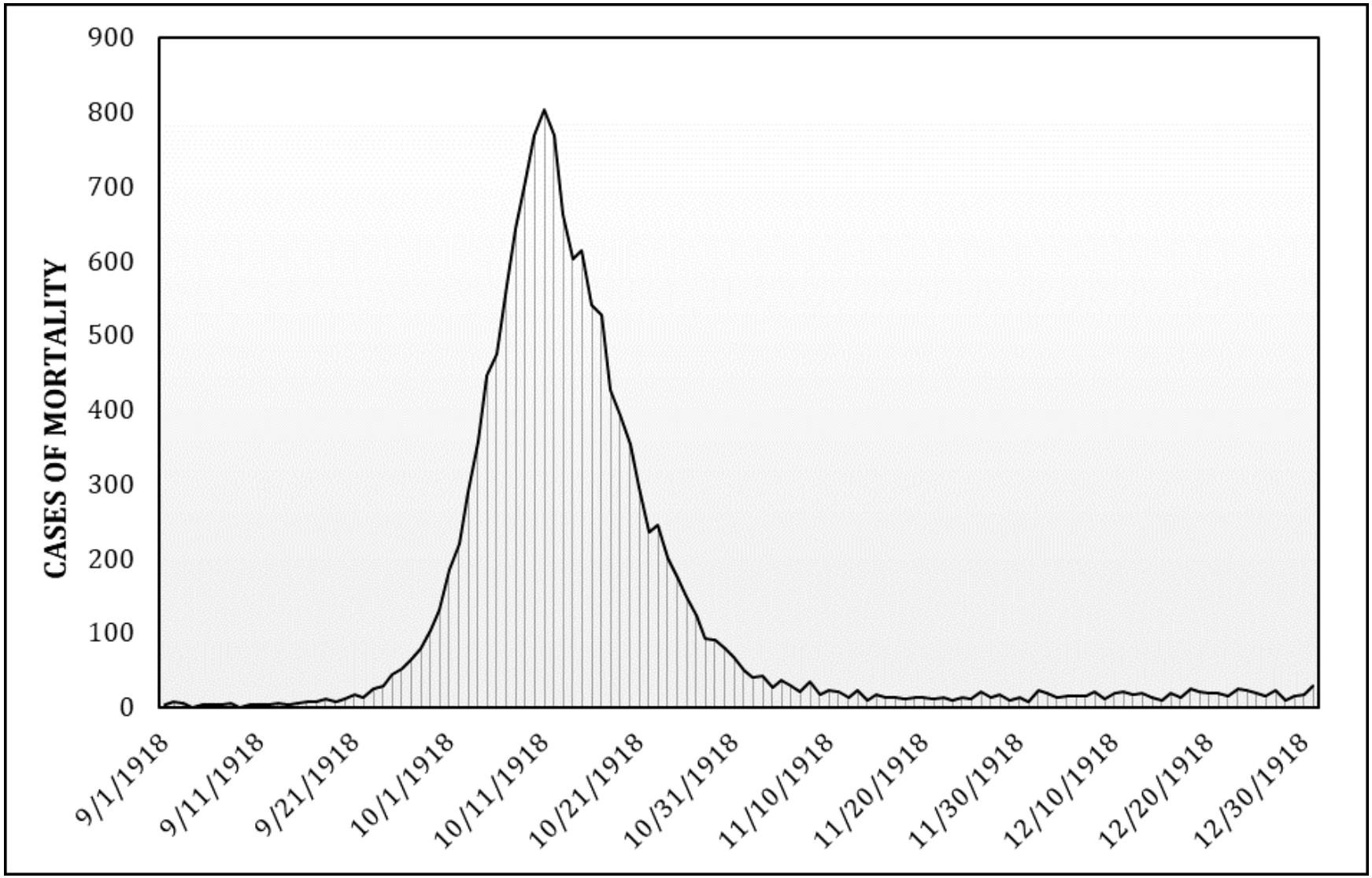


Figure 4. Pandemic Mortality Curve for Philadelphia, PA 1918

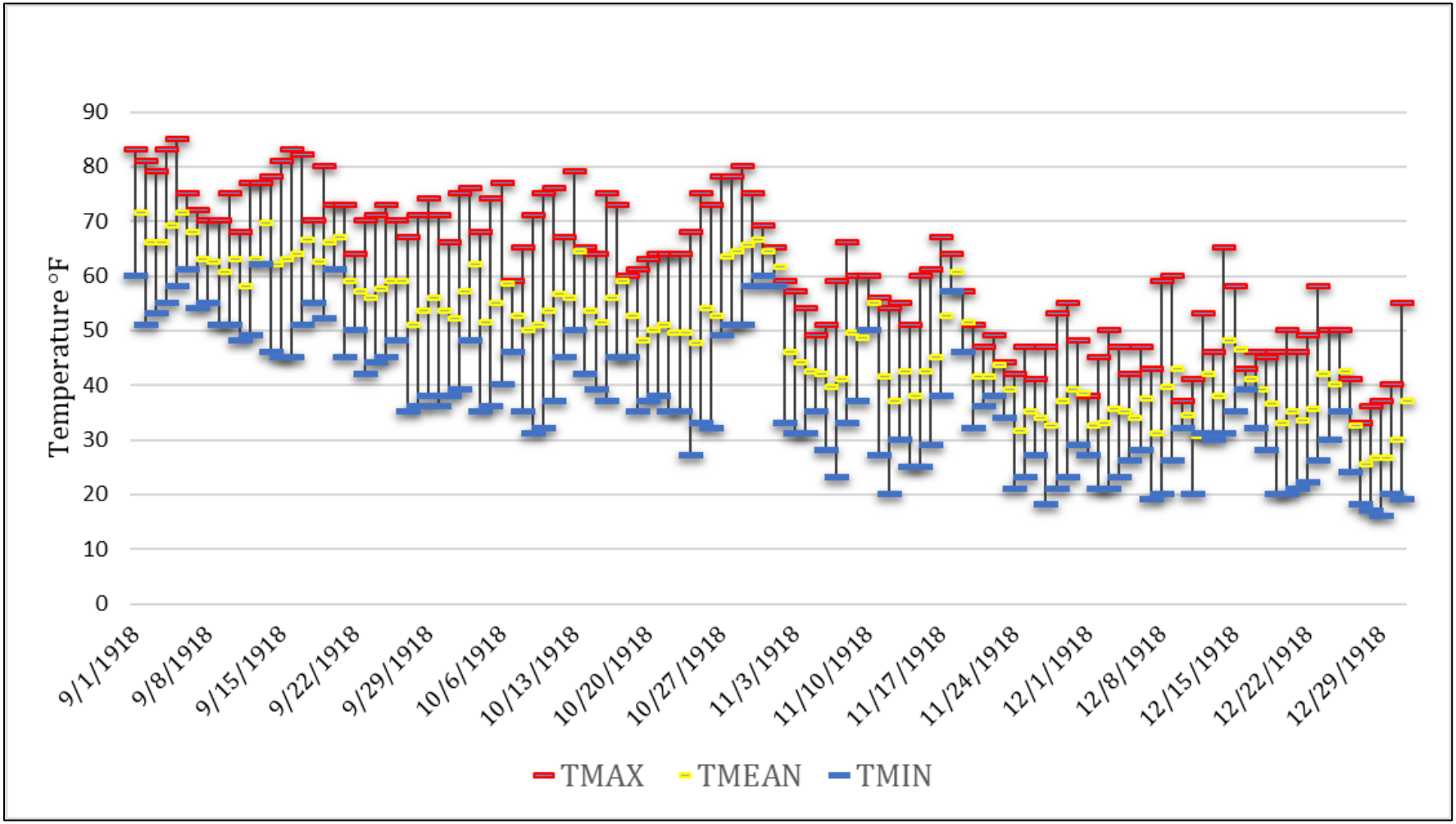


Figure 5. Daily High, Low, and Mean Temperature Values for Philadelphia, PA 1918

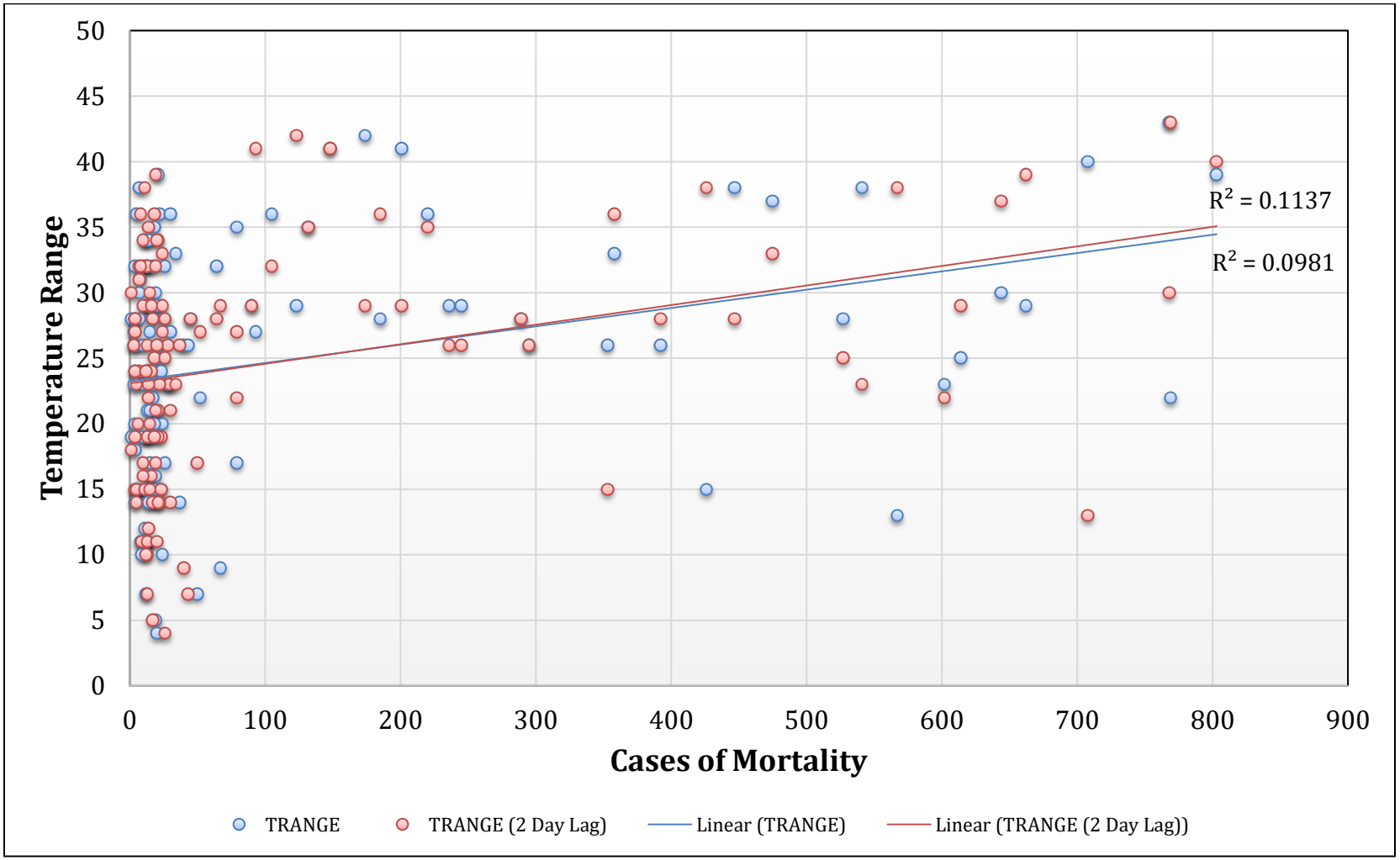


Figure 6. Scatter Plot of Relationship between Mortality and Change in Temperature in Philadelphia, PA 1918

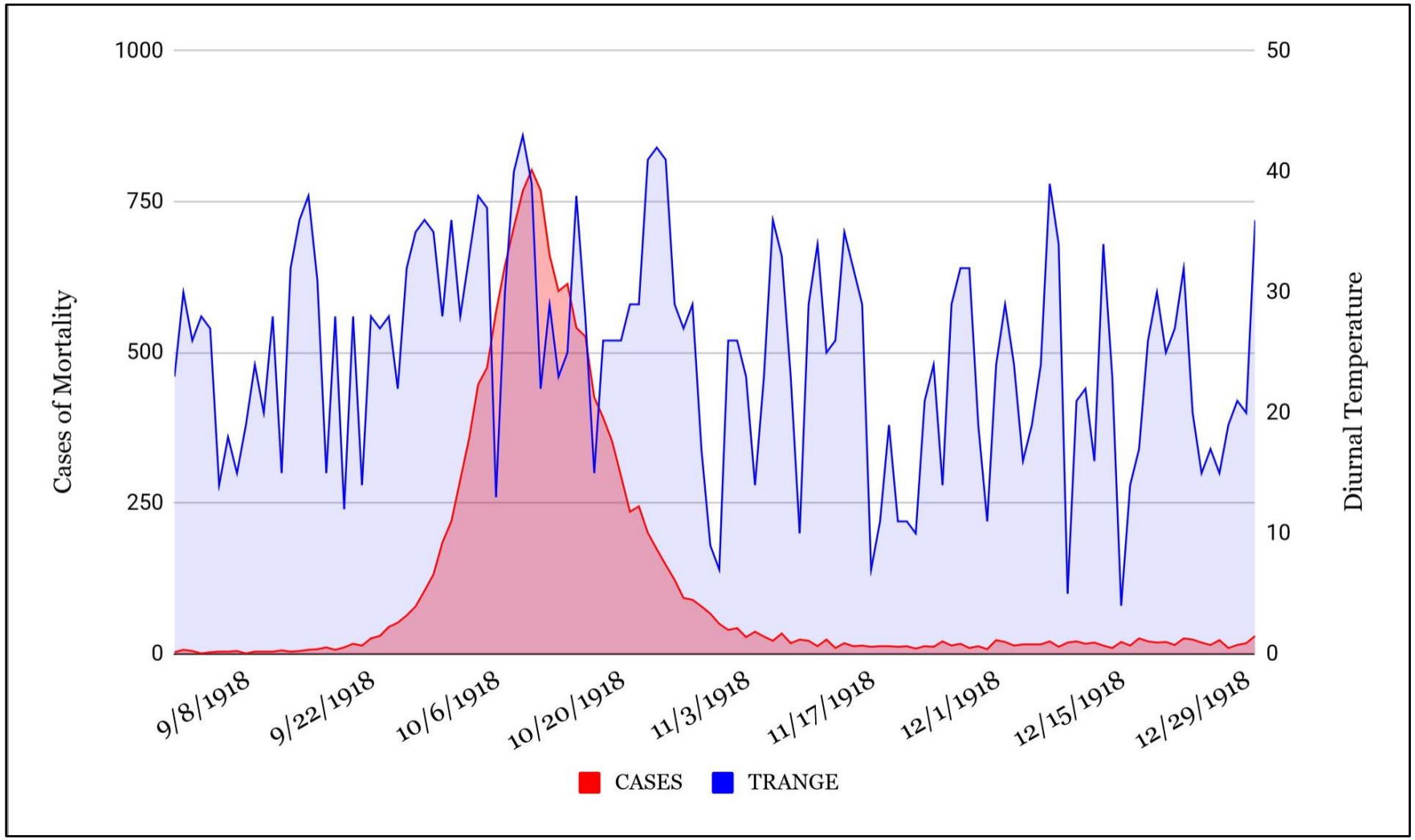


Figure 7. Overlay of Philadelphia Mortality and Temperature Range

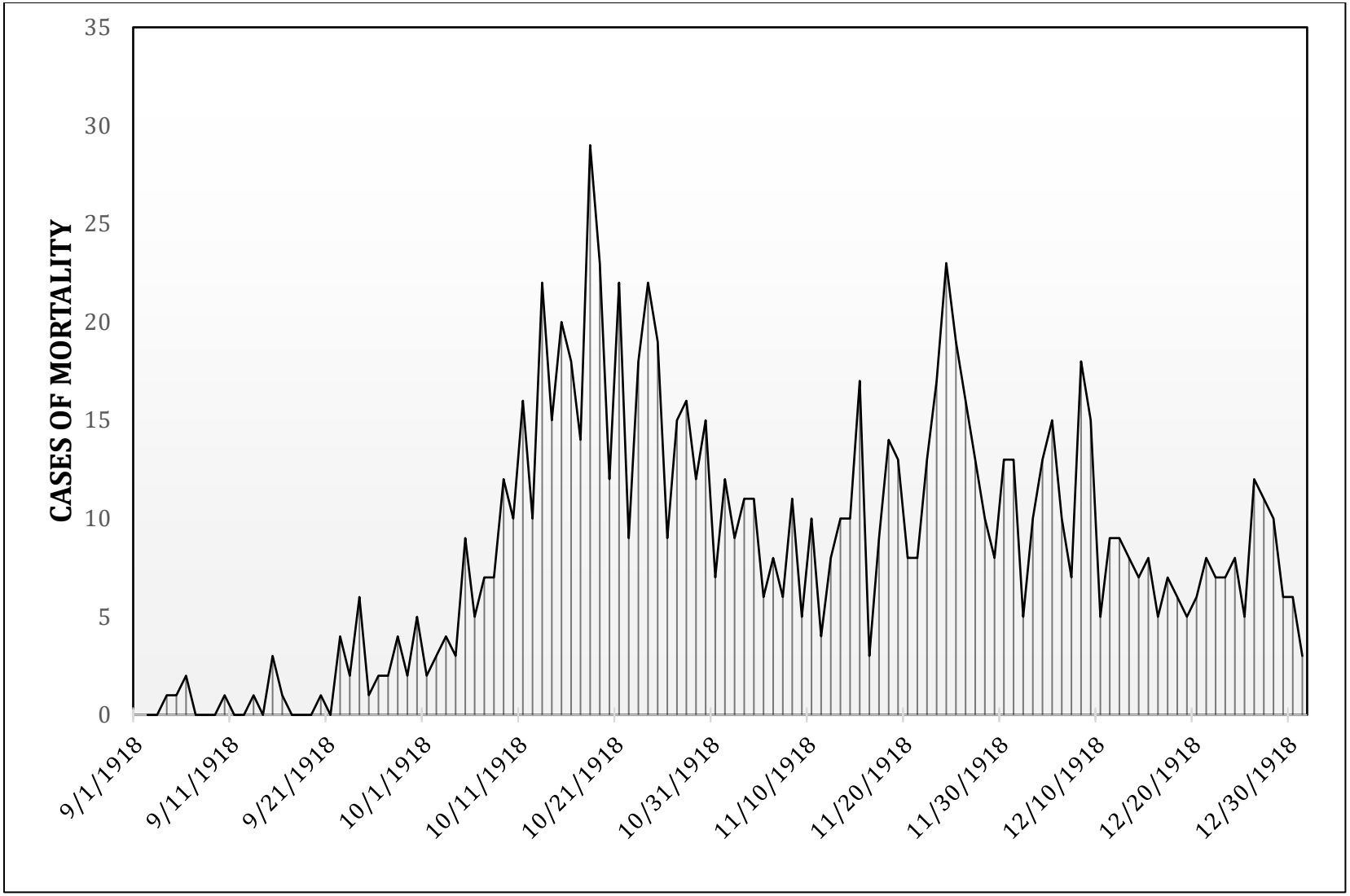


Figure 8. Pandemic Mortality Curve for Indianapolis, IN 1918

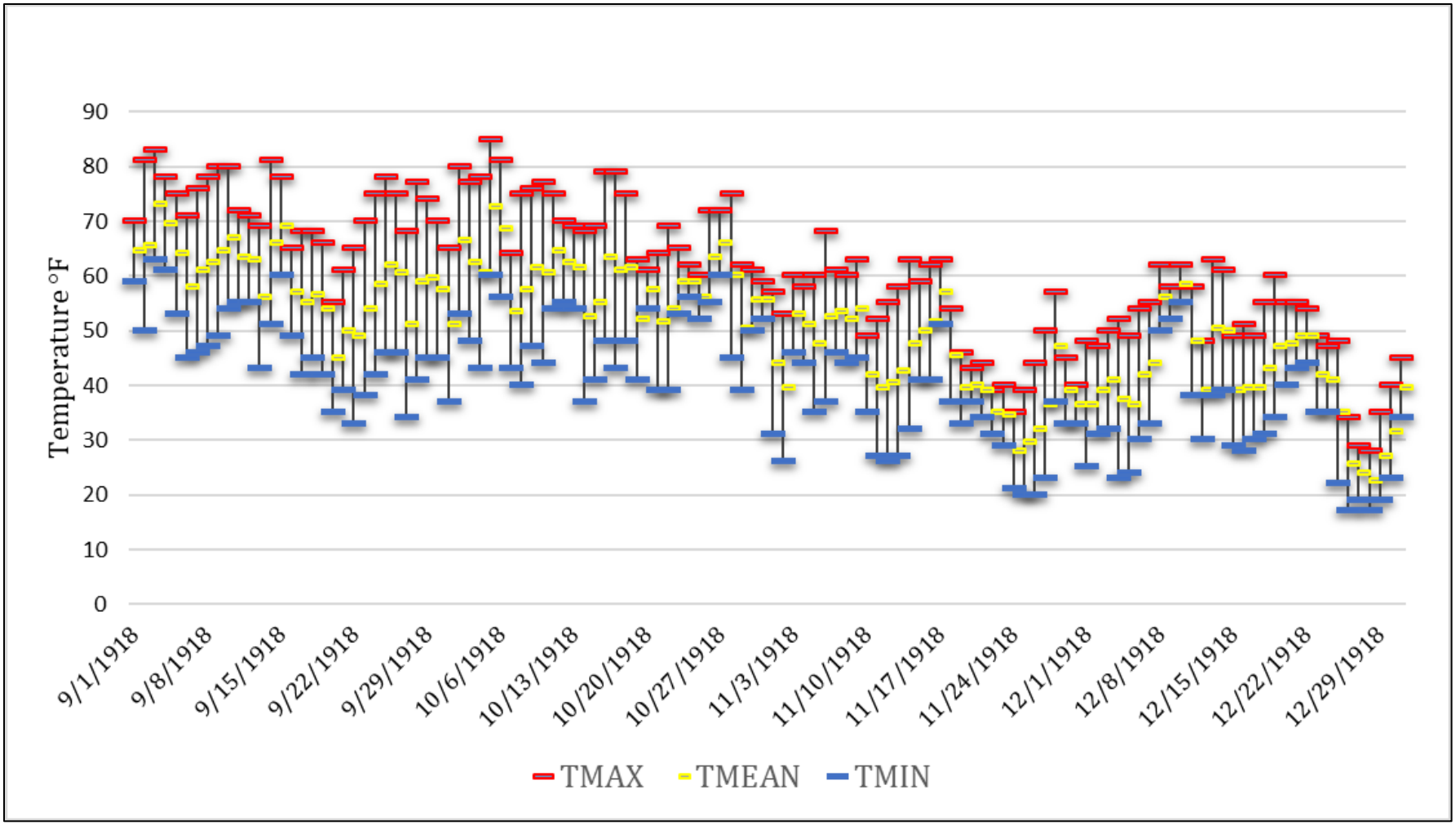


Figure 9. Daily High and Low Temperatures in Indianapolis, IN 1918

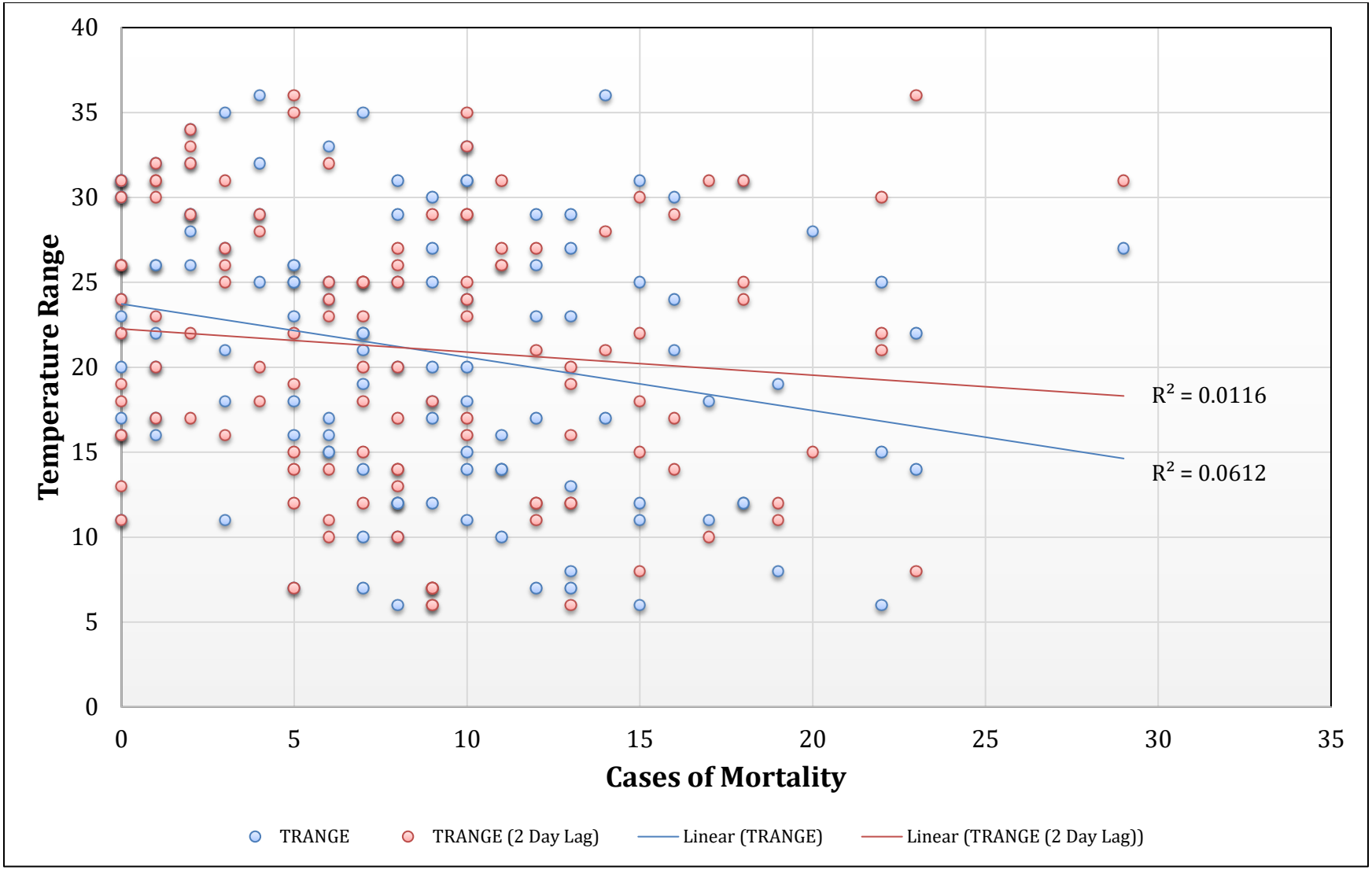


Figure 10. Scatter Plot of Relationship between Mortality and Change in Temperature in Indianapolis, IN 1918

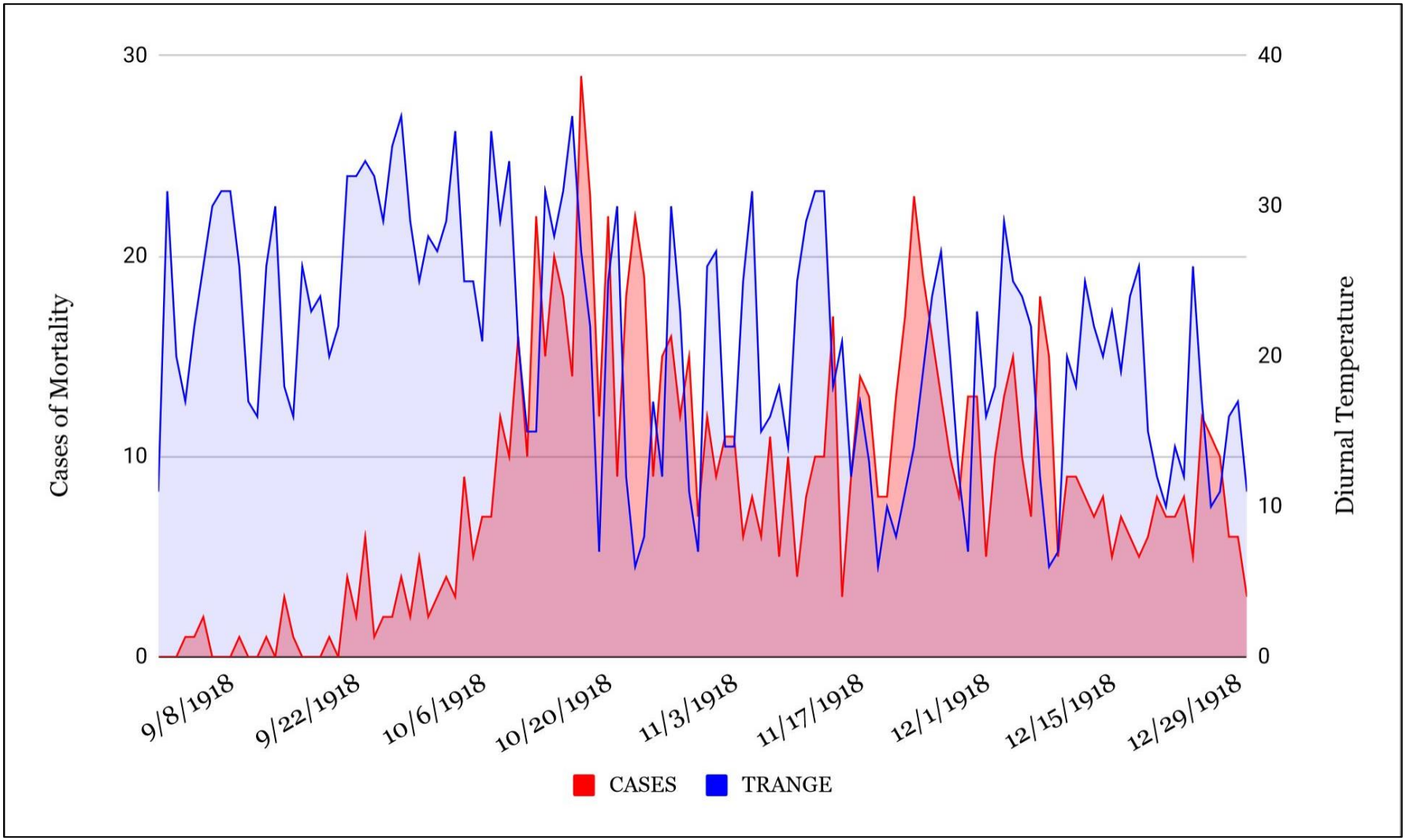


Figure 11. Overlay of Indianapolis Mortality and Temperature Range

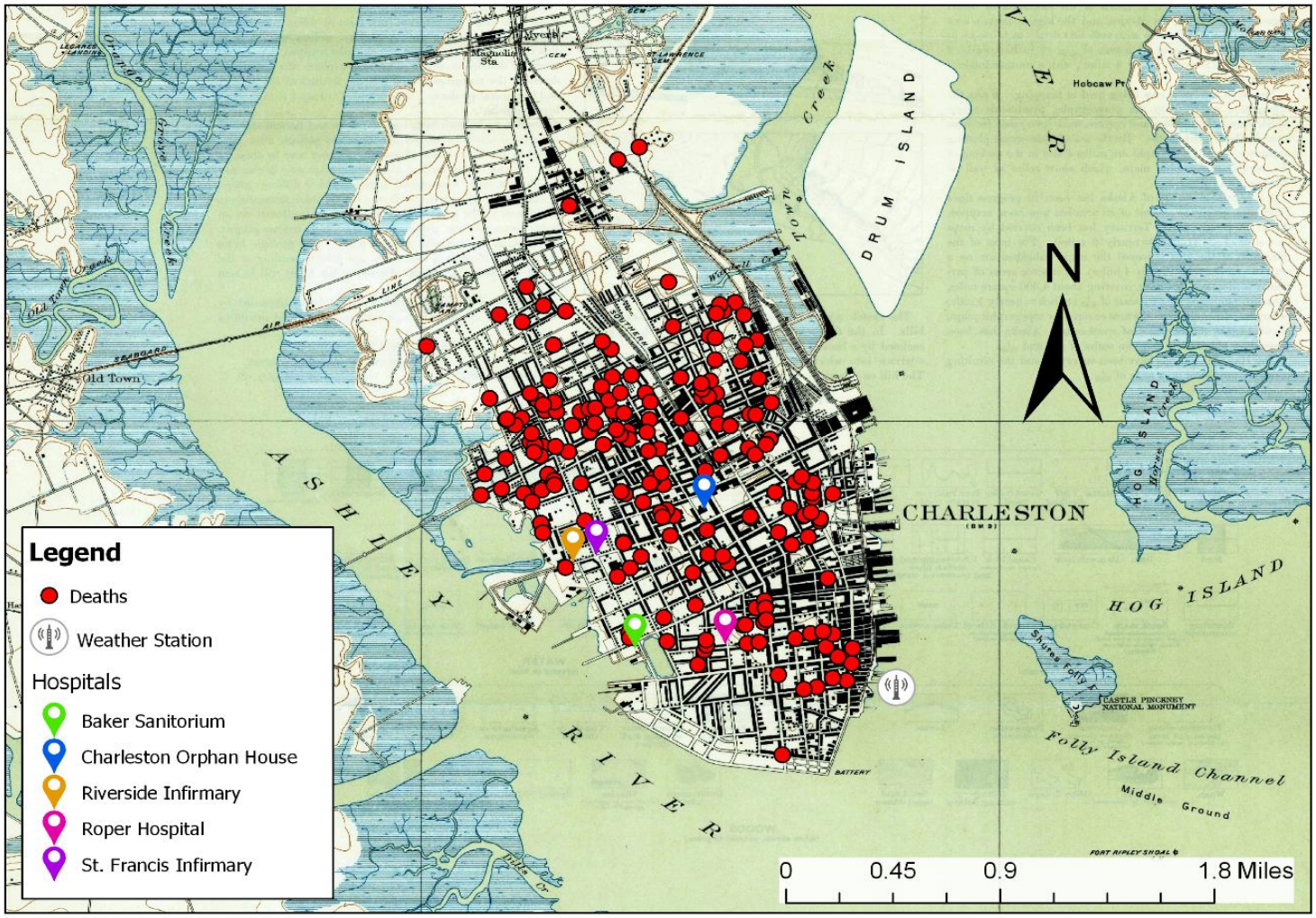


Figure 12. Distribution of 1918 Spanish Flu Deaths, Health Centers, and Weather Station in the Downtown Charleston Region

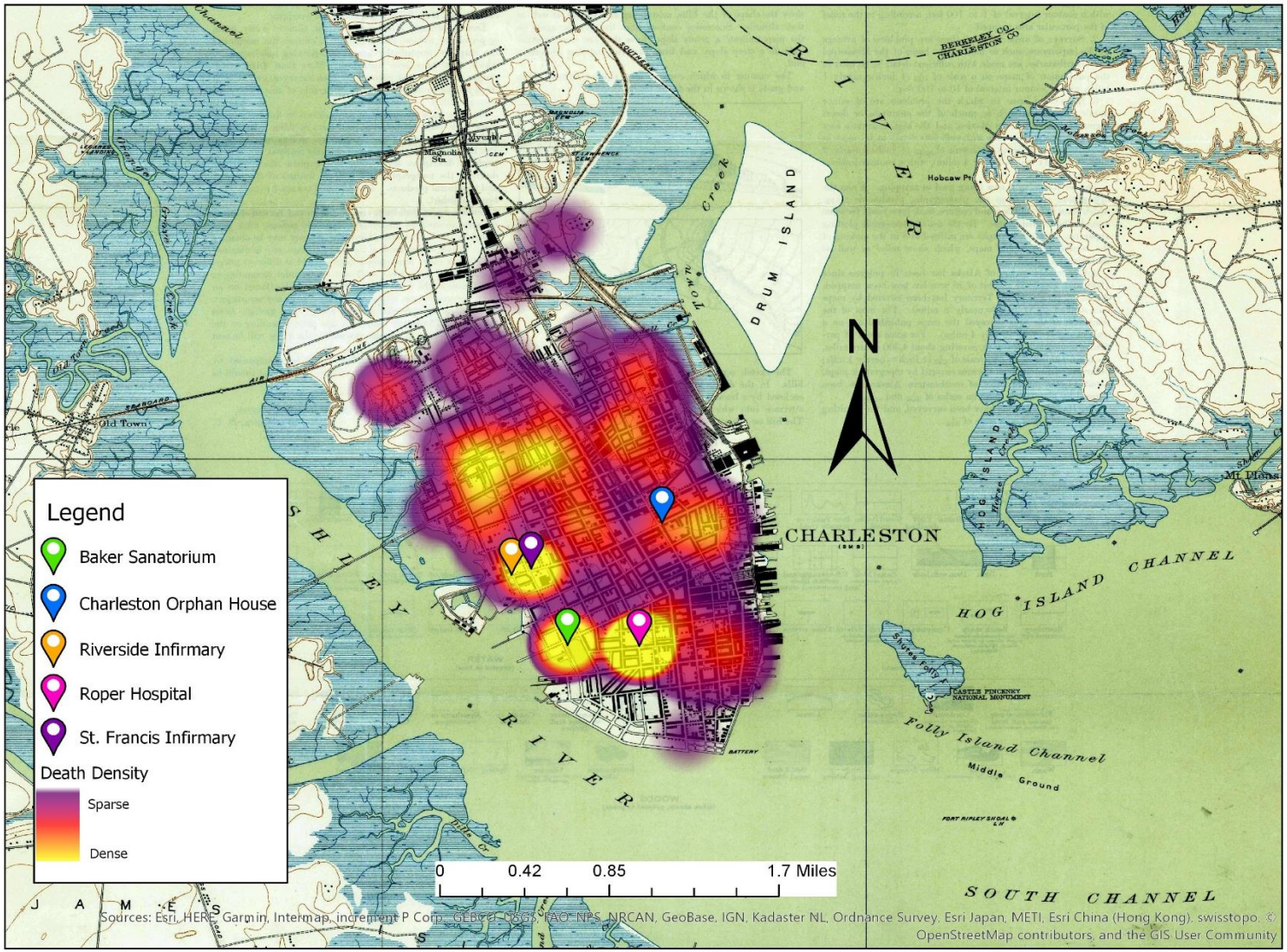


Figure 13. Heat Map of Flu Death Density in Charleston, SC 1918

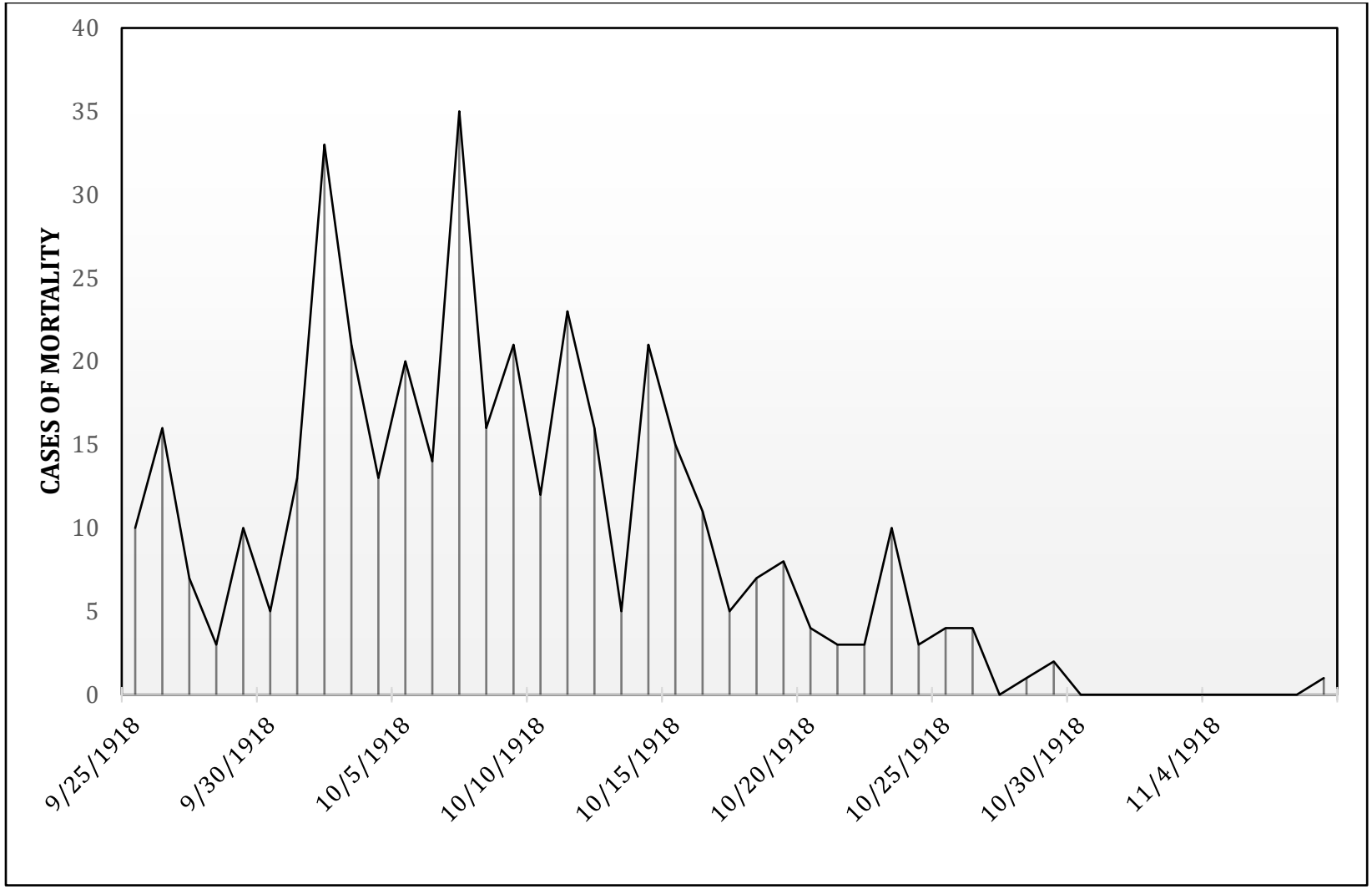


Figure 14. Pandemic Mortality Curve for Naval Data in Charleston, SC 1918

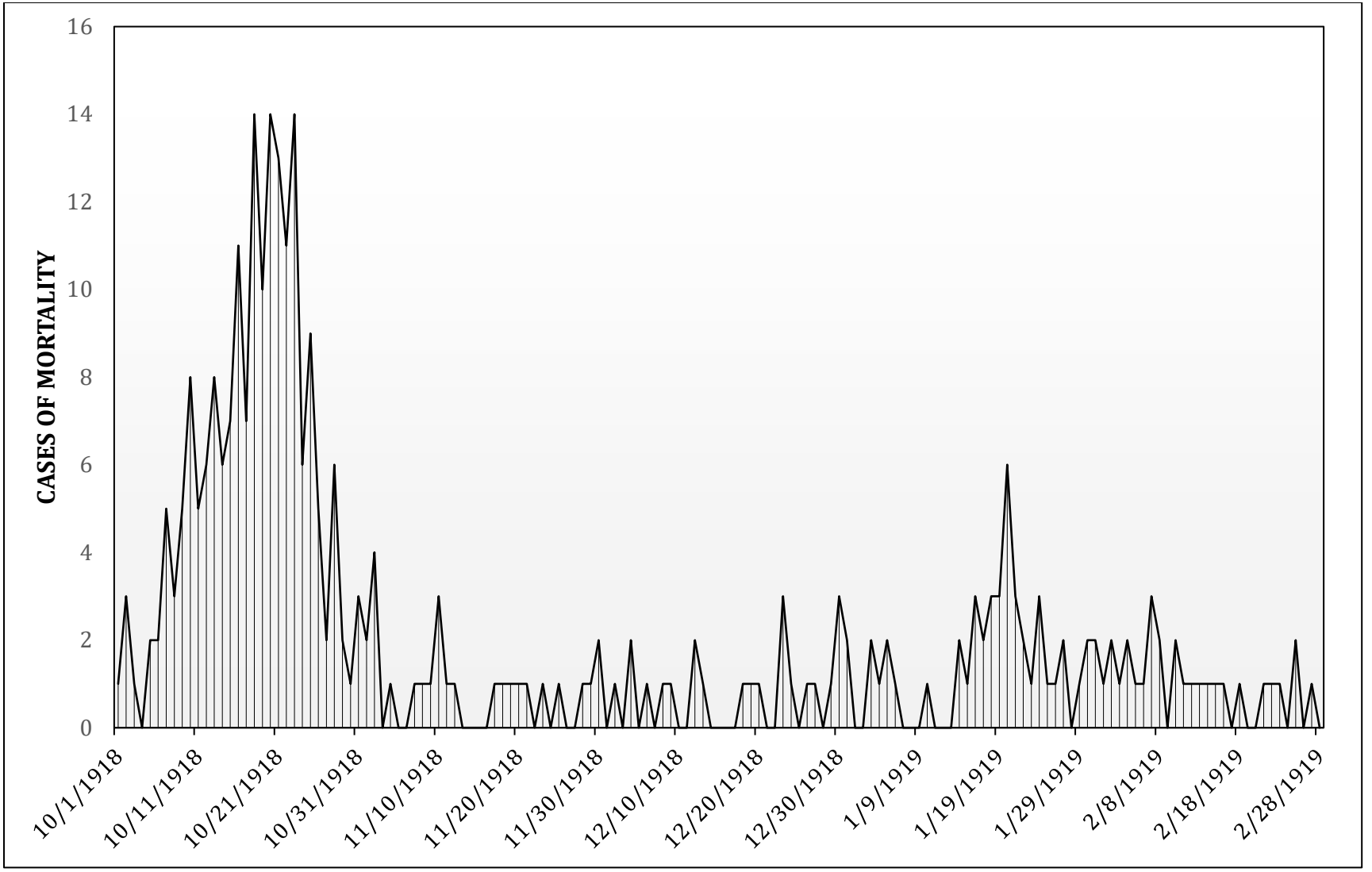


Figure 15. Pandemic Mortality Curve for Civilian Data in Charleston, SC 1918

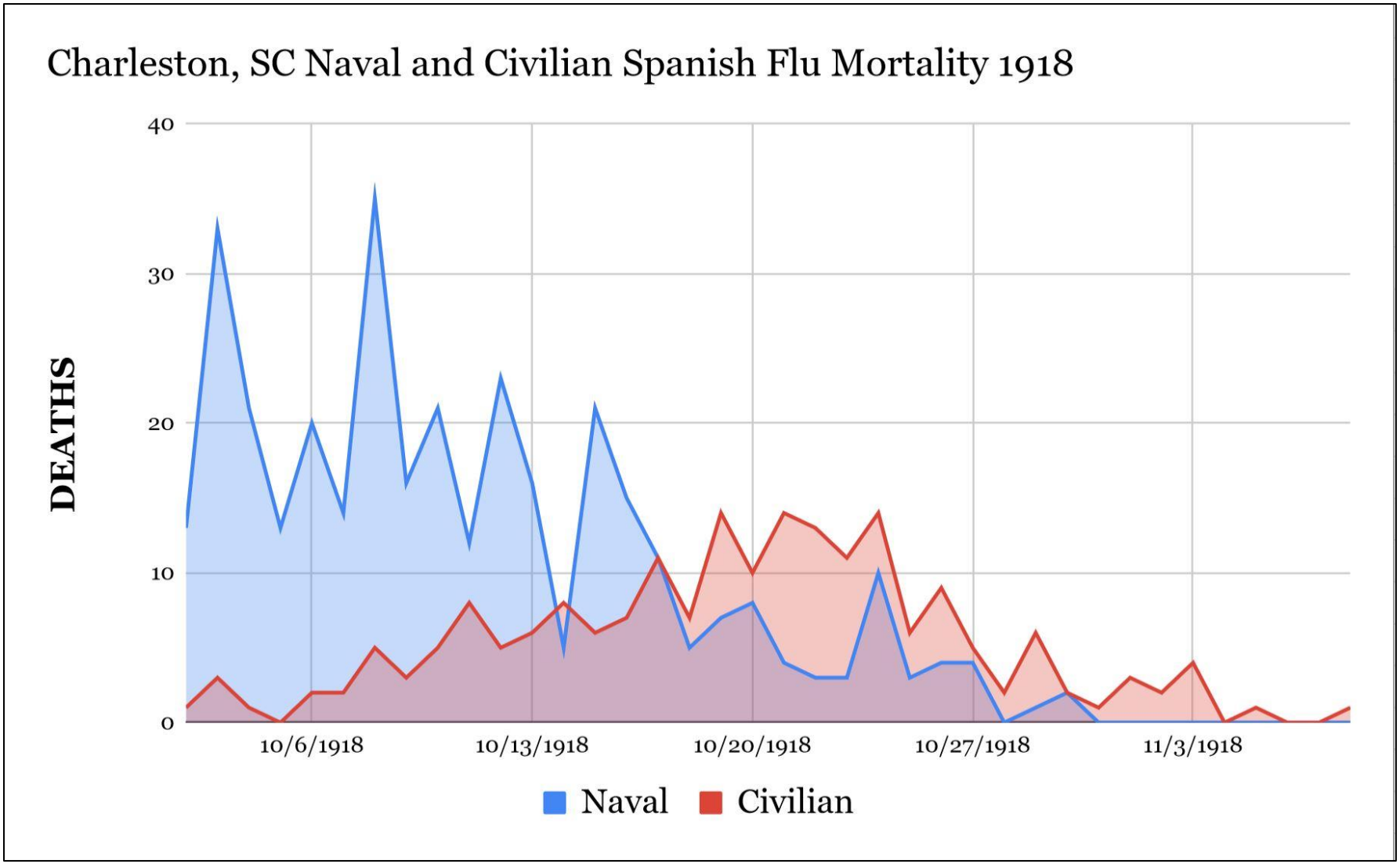


Figure 16. Overlay of Mortality Data for Naval and Civilian Data in Charleston, SC 1918

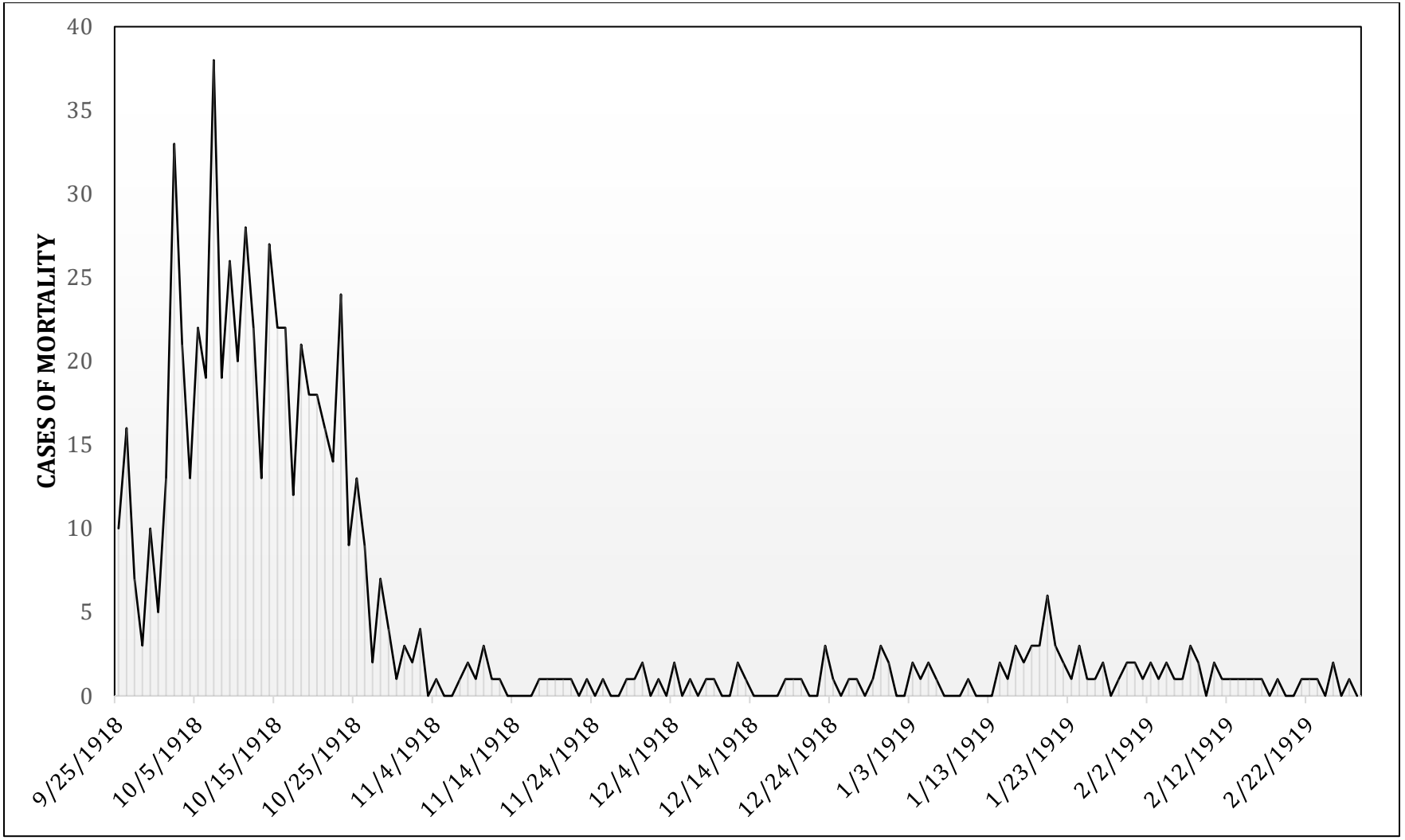


Figure 17. Combined Pandemic Mortality Curve for Charleston Naval and Civilian Combined Data in 1918

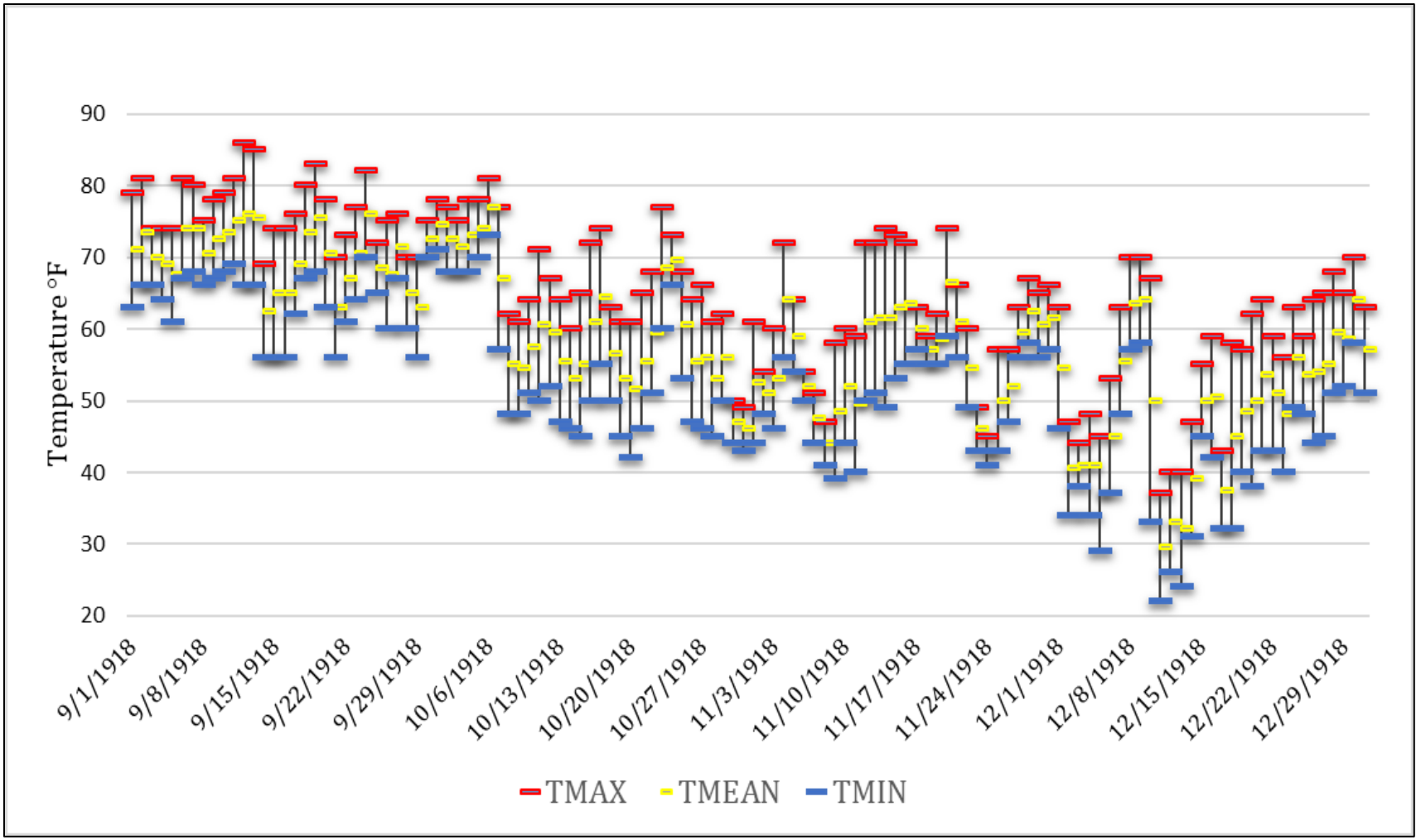


Figure 18. Daily High and Low Temperatures in Charleston, SC 1918

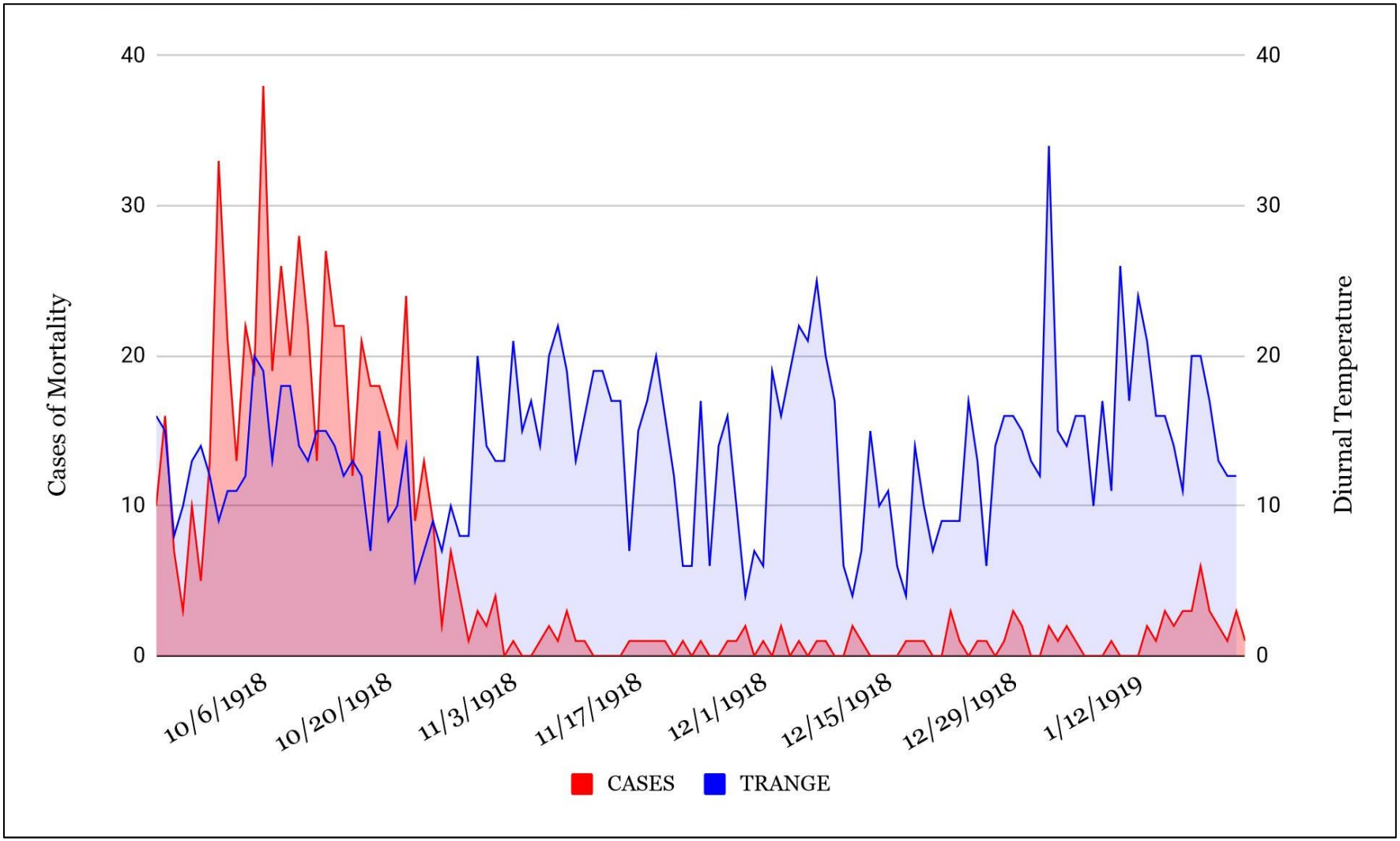


Figure 19. Overlay of Charleston Civilian and Naval Mortality and Temperature Range

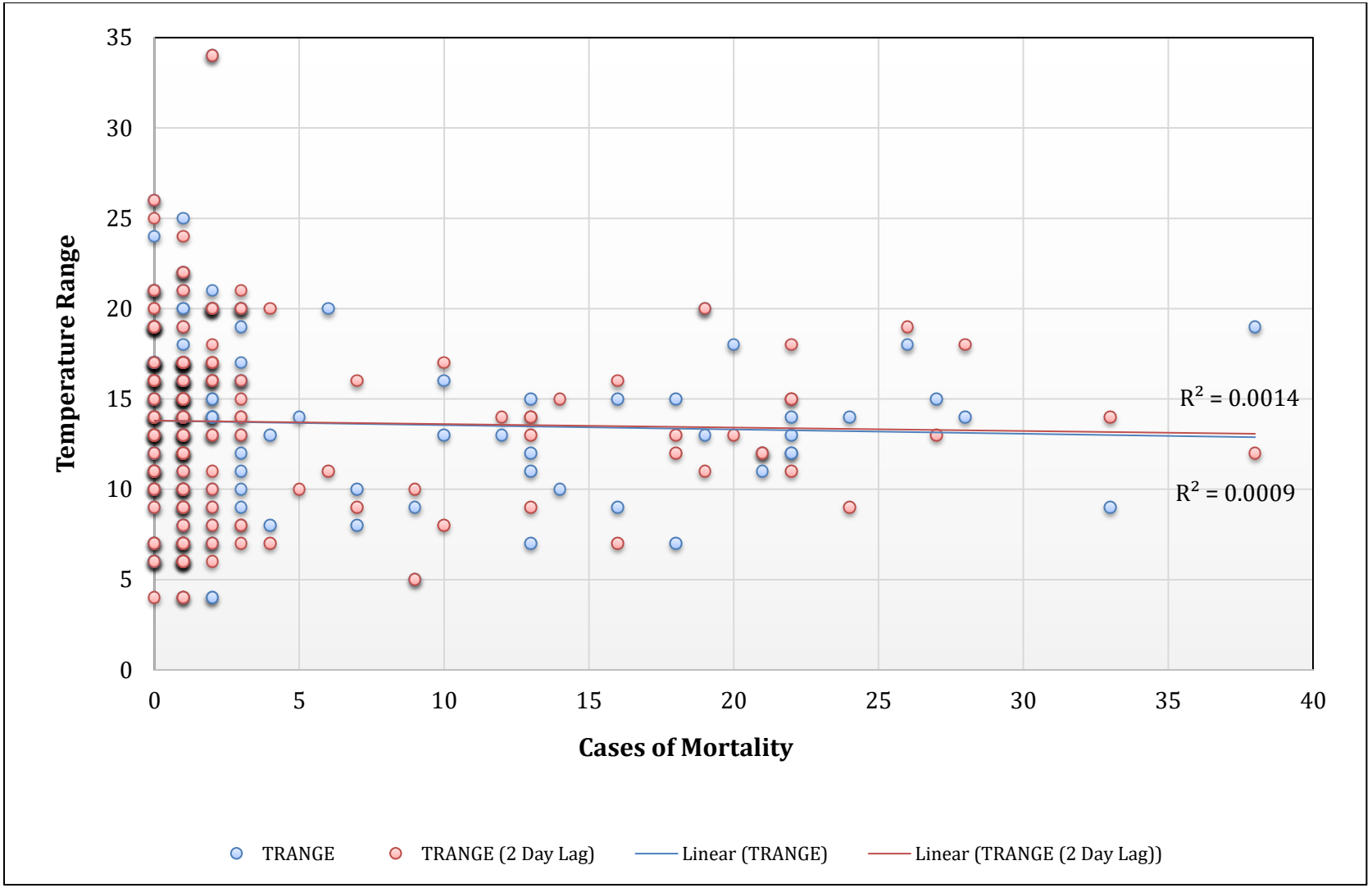


Figure 20. Scatter Plot of Relationship between Mortality and Change in Temperature in Charleston, SC 1918

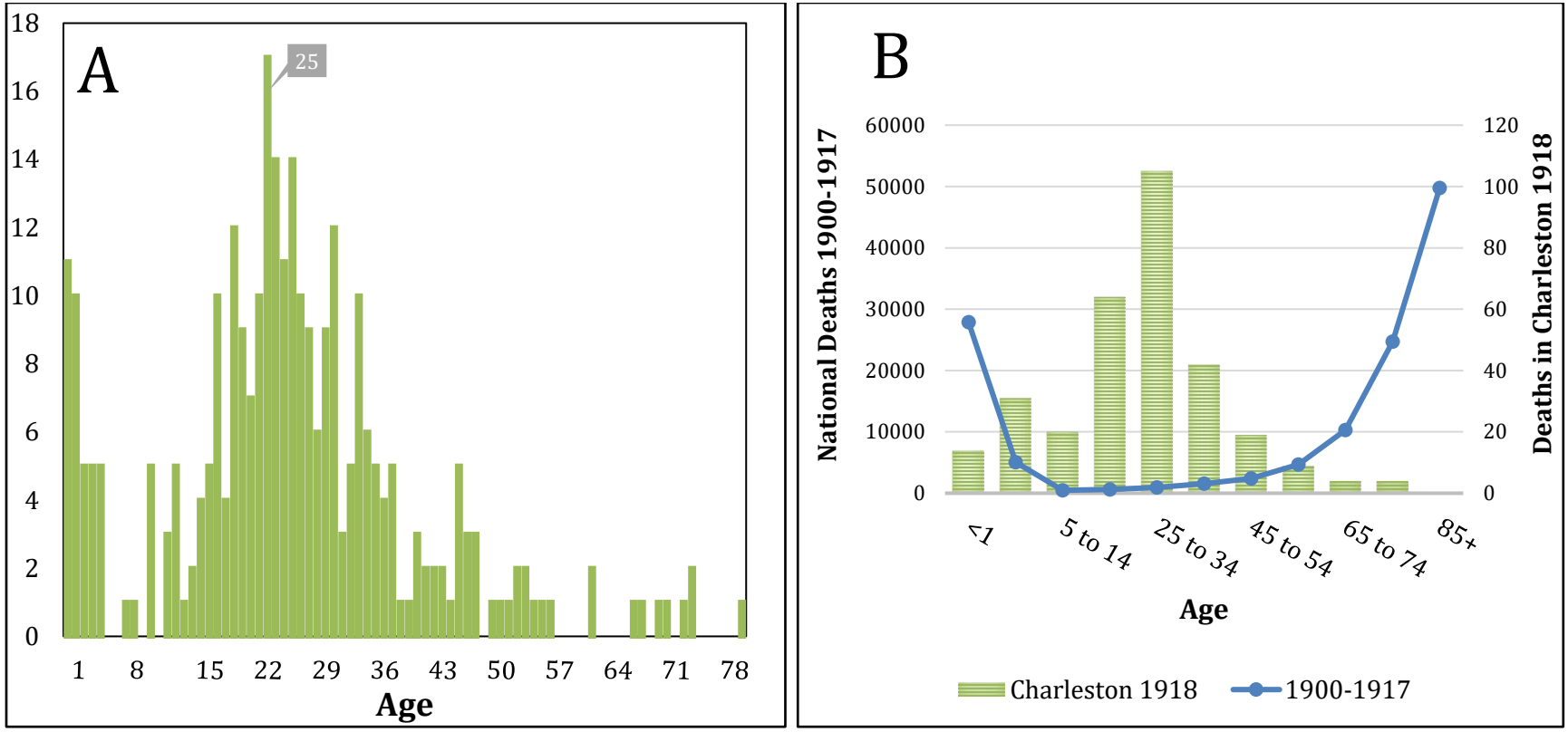


Figure 21. (A) Charleston Civilian Population 1918 Spanish Flu Deaths by Age and (B) Comparison to Previous National Counts of Deaths from 1900-1917 (Linder & Grove, 1943)

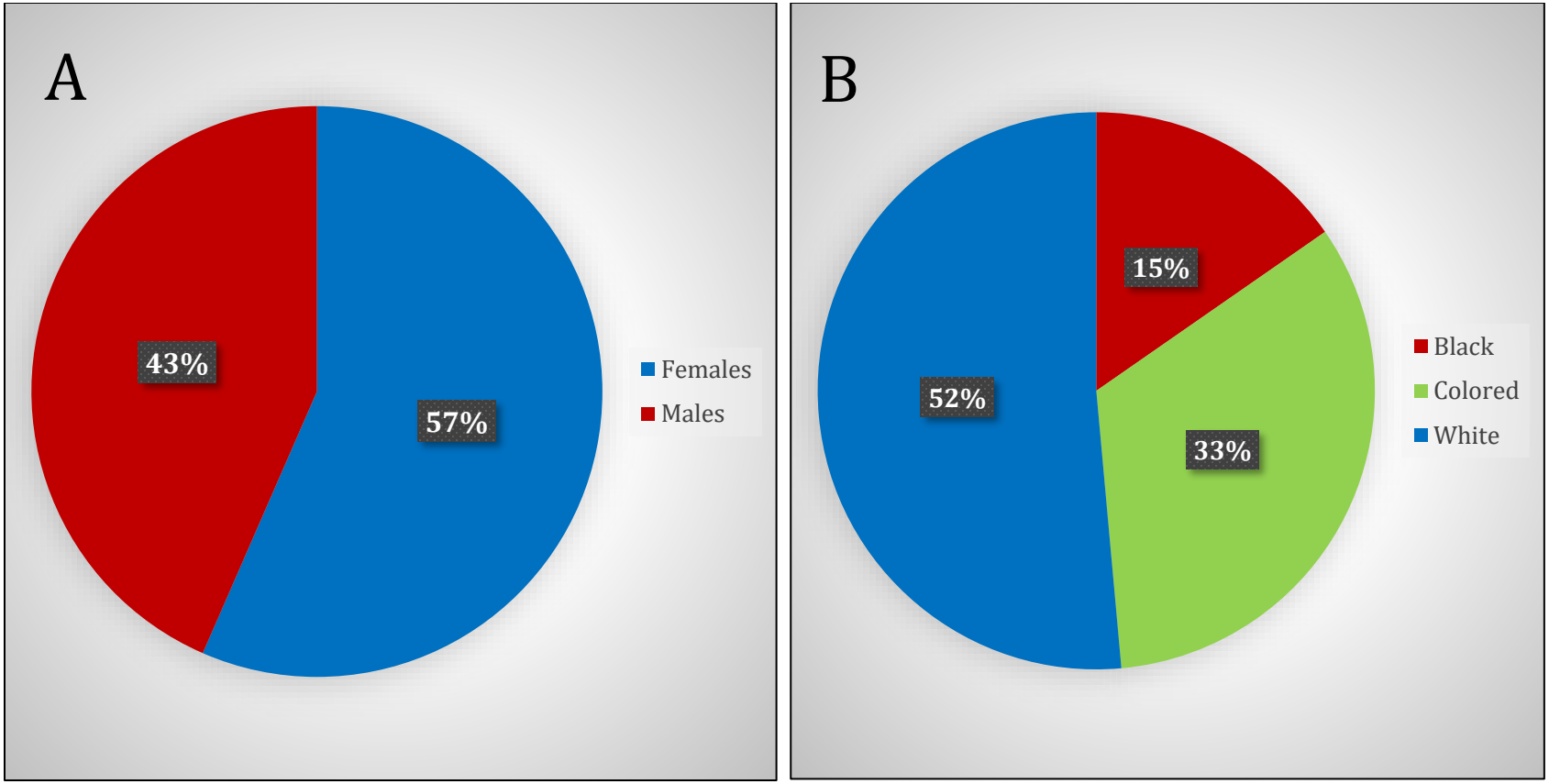


Figure 22. Gender (A) and Race Distributions (B) for Charleston 1918 Spanish Flu Mortality Data

Tables

Table 1. Timing of Previous Pandemic Outbreaks and Mortality Scale (Kilbourne, 2006)

Year	Common Name	Influenza Strain	Peak Month in US	Estimated Mortality in US	Estimated Global Mortality
1918	Spanish Flu	H1N1	October	675,000	50 million
1957	Asian Flu	H2N2	October	116,000	1.1 million
1968	Hong Kong	H3N2	October	100,000	1 million
2009	Swine Flu	H1N1pdm09	October	12,469	575,400

Table 2. Descriptive Statistics of Influenza Mortality and Temperature for Philadelphia, PA in 1918

Variable	N	Minimum	Maximum	Mean	Std. Deviation
DATE	122	9/1/1918	12/31/1918	10/31/1918	-
CASES	122	1	803	114.3	199.36
T-MAX	122	35	86	63.2	13.44
T-MIN	122	13	64	38.3	12.69
T-MEAN	122	24	74	50.8	12.31
T-RANGE	122	9	43	24.9	8.78

Table 3. Related-Samples Wilcoxon Signed Rank Test for Philadelphia, PA. Significance level 0.05*

Variable	Lag (Days)	p-value
T-MAX	0	.071
	1	.080
	2	.080
	3	.079
	4	.077
	5	.094
T-MIN	0	.995
	1	.917
	2	.917
	3	.946
	4	.863
	5	.860
T-MEAN	0	.247
	1	.242
	2	.242
	3	.253
	4	.285
	5	.295
T-RANGE	0	.023*
	1	.012*
	2	.012*
	3	.013*
	4	.015*
	5	.018*
	6	.022*
	7	.038*
	8	.047*
	9	.079
	10	.055

Table 4. Auto Correlated ARIMA Models for Philadelphia, PA

Variable	Lag (Days)	Estimate	Std Error	t Ratio	Prob>|t|
T-MAX	0	110.657	24.392	4.54	<0.0001*
	1	-1.638	0.086	-19.04	<0.0001*
	2	-2.079	0.133	-15.60	<0.0001*
	3	-1.866	0.124	-15.04	<0.0001*
	4	-1.280	0.094	-13.59	<0.0001*
	5	-0.555	0.072	-7.70	<0.0001*
T-MIN	0	110.657	24.392	4.54	<0.0001*
	1	-1.638	0.086	-19.04	<0.0001*
	2	-2.079	0.133	-15.60	<0.0001*
	3	-1.866	0.124	-15.04	<0.0001*
	4	-1.280	0.094	-13.59	<0.0001*
	5	-0.555	0.072	-7.70	<0.0001*
T-MEAN	0	110.657	24.392	4.54	<0.0001*
	1	-1.638	0.086	-19.04	<0.0001*
	2	-2.079	0.133	-15.60	<0.0001*
	3	-1.866	0.124	-15.04	<0.0001*
	4	-1.280	0.094	-13.59	<0.0001*
	5	-0.555	0.072	-7.70	<0.0001*
T-RANGE	0	109.775	36.482	3.01	0.0032*
	1	-1.456	0.095	-15.20	<0.0001*
	2	-1.786	0.147	-12.07	<0.0001*
	3	-1.908	0.218	-8.75	<0.0001*
	4	-2.083	0.259	-8.02	<0.0001*
	5	-1.950	0.280	-6.96	<0.0001*
	6	-1.89	0.248	-7.65	<0.0001*
	7	-1.61	0.195	-8.25	<0.0001*
	8	-1.43	0.146	-9.83	<0.0001*
	9	-1.30	0.121	-10.78	<0.0001*
	10	-0.47	0.101	-4.64	<0.0001*

Table 5. Descriptive Statistics of Influenza Mortality and Temperature for Indianapolis, IN in 1918

Variable	N	Minimum	Maximum	Mean	Std. Deviation
DATE	122	9/1/1918	12/31/1918	10/31/1918	-
CASES	122	0	29	8.47	6.30
T-MAX	122	28	85	61.41	13.00
T-MIN	122	17	63	40.34	10.97
T-MEAN	122	22.5	73	50.87	11.34
T-RANGE	122	6	36	21.07	7.99

Table 6. Related-Samples Wilcoxon Signed Rank Test for Indianapolis, IN 1918. Significance level 0.05*

Variable	Lag (Days)	p-value
T-MAX	0	<0.000*
	1	<0.000*
	2	<0.000*
	3	<0.000*
	4	<0.000*
	5	<0.000*
T-MIN	0	<0.000*
	1	<0.000*
	2	<0.000*
	3	<0.000*
	4	<0.000*
	5	<0.000*
T-MEAN	0	<0.000*
	1	<0.000*
	2	<0.000*
	3	<0.000*
	4	<0.000*
	5	<0.000*
T-RANGE	0	<0.000*
	1	<0.000*
	2	<0.000*
	3	<0.000*
	4	<0.000*
	5	<0.000*
	6	<0.000*
	7	<0.000*
	8	<0.000*
	9	<0.000*
	10	<0.000*

Table 7. Auto Correlated ARIMA Models for Indianapolis, IN 1918

Variable	Lag (Days)	Estimate	Standard Error	t Ratio	Prob>|t|
T-MAX	0	8.029	1.347	5.96	<0.0001*
	1	-0.353	0.092	-3.81	0.0002*
	2	-0.369	0.102	-3.59	0.0005*
	3	-0.414	0.107	-3.86	0.0002*
	4	-0.409	0.112	-3.65	0.0004*
	5	-0.473	0.110	-4.28	<0.0001*
T-MIN	0	7.898	2.333	3.38	0.0010*
	1	-0.665	0.493	-1.35	0.1809
	2	1.300	0.319	4.07	<0.0001*
	3	0.527	0.798	0.66	0.5104
	4	-1.181	0.268	-4.40	<0.0001*
	5	0.620	0.704	0.88	0.380
T-MEAN	0	8.029	1.347	5.96	<0.001*
	1	-0.353	0.092	-3.81	0.0002*
	2	-0.369	0.102	-3.59	0.0005*
	3	-0.414	0.107	-3.86	0.0002*
	4	-0.409	0.112	-3.65	0.0004*
	5	-0.473	0.110	-4.28*	<0.0001*
T-RANGE	0	7.898	2.333	3.30	0.0010*
	1	-0.665	0.493	-1.35	0.1809
	2	1.300	0.319	4.07	<0.0001*
	3	0.527	0.798	0.66	0.5104
	4	-1.181	0.268	-4.40	<0.0001*
	5	0.620	0.704	0.88	0.3809
	6	1.203	0.639	1.88	0.0626
	7	-0.889	0.514	-1.73	0.0872
	8	-1.01	0.735	-1.38	0.1696
	9	0.168	0.352	0.48	0.6331
	10	0.092	0.356	0.26	0.7947

Table 8. Descriptive Statistics of Influenza Mortality and Temperature for Charleston Naval Base, SC in 1918

Variable	N	Minimum	Maximum	Mean	Std. Deviation
DATE	45	9/25/1918	11/8/1918	10/17/1918	-
CASES	45	0	35	8.78	8.94
T-MAX	45	60	86	74.64	6.31
T-MIN	45	45	73	61.69	7.68
T-MEAN	45	53	77	68.17	6.75
T-RANGE	45	5	21	12.96	3.94

Table 9. Descriptive Statistics of Influenza Mortality and Temperature for Charleston, SC Civilian Data in 1918

Variable	N	Minimum	Maximum	Mean	Std. Deviation
DATE	151	10/1/1918	2/28/1918	12/13/1918	-
CASES	151	0	14	2	2.95
T-MAX	151	37	86	63.65	10.06
T-MIN	151	22	73	49.93	10.50
T-MEAN	151	29.5	77	56.79	9.98
T-RANGE	151	4	34	13.72	4.93

Table 10. Related-Samples Wilcoxon Signed Rank Test for Charleston, SC Combined Naval and Civilian Data Sets in 1918. Significance level 0.05*

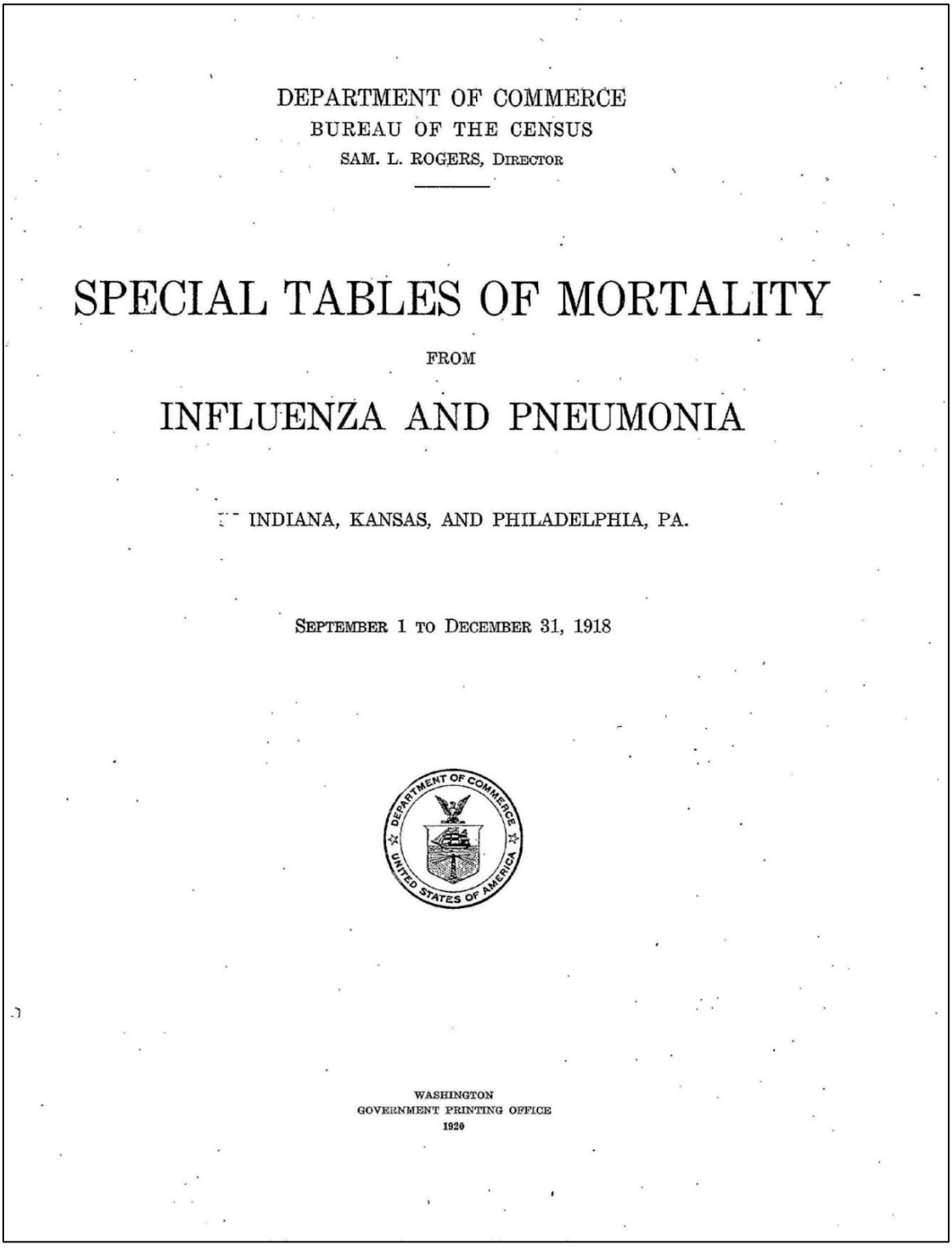
Variable	Lag (Days)	p-value
T-MAX	0	<0.000*
	1	<0.000*
	2	<0.000*
	3	<0.000*
	4	<0.000*
	5	<0.000*
T-MIN	0	<0.000*
	1	<0.000*
	2	<0.000*
	3	<0.000*
	4	<0.000*
	5	<0.000*
T-MEAN	0	<0.000*
	1	<0.000*
	2	<0.000*
	3	<0.000*
	4	<0.000*
	5	<0.000*
T-RANGE	0	<0.000*
	1	<0.000*
	2	<0.000*
	3	<0.000*
	4	<0.000*
	5	<0.000*
	6	<0.000*
	7	<0.000*
	8	<0.000*
	9	<0.000*
	10	<0.000*

Table 11. Auto Correlated ARIMA Models for Charleston, SC Combined Naval and Civilian Data Sets in 1918

Variable	Lag (Days)	Estimate	Std Error	t Ratio	Prob>|t|
T-MAX	0	4.583	1.342	3.41	0.0008*
	1	-0.353	0.082	-4.31	<0.0001*
	2	-0.399	0.092	-4.31	<0.0001*
	3	-0.367	0.084	-4.32	<0.0001*
	4	-0.363	0.078	-4.61	<0.0001*
	5	-0.630	0.090	-6.95	<0.0001*
T-MIN	0	4.697	1.155	4.07	<0.0001*
	1	-0.566	0.079	-7.10	<0.0001*
	2	-0.638	0.108	-5.89	<0.0001*
	3	-0.563	0.107	-5.23	<0.0001*
	4	-0.351	0.084	-4.18	<0.0001*
	5	-0.505	0.075	-6.71	<0.0001*
T-MEAN	0	4.697	1.155	4.07	<0.0001*
	1	-0.566	0.079	-7.10	<0.0001*
	2	-0.638	0.108	-5.89	<0.0001*
	3	-0.563	0.107	-5.23	<0.0001*
	4	-0.351	0.084	-4.18	<0.0001*
	5	-0.505	0.075	-6.71	<0.0001*
T-RANGE	0	4.583	1.342	3.41	0.0008*
	1	-0.353	0.082	-4.31	<0.0001*
	2	-0.399	0.092	-4.31	<0.0001*
	3	-0.367	0.084	-4.32	<0.0001*
	4	-0.363	0.078	-4.61	<0.0001*
	5	-0.630	0.090	-6.95	<0.0001*
	6	-0.422	0.080	-5.25	<0.0001*
	7	-0.521	0.087	-5.94	<0.0001*
	8	-0.433	0.094	-4.59	<0.0001*
	9	-0.440	0.086	-5.10	<0.0001*
	10	-0.114	0.107	-1.06	0.2921

Appendices

Appendix A. Special Tables of Mortality of Influenza and Pneumonia: Indiana Kansas, and Philadelphia, PA 1918



DEPARTMENT OF COMMERCE
BUREAU OF THE CENSUS
SAM. L. ROGERS, DIRECTOR

SPECIAL TABLES OF MORTALITY

FROM

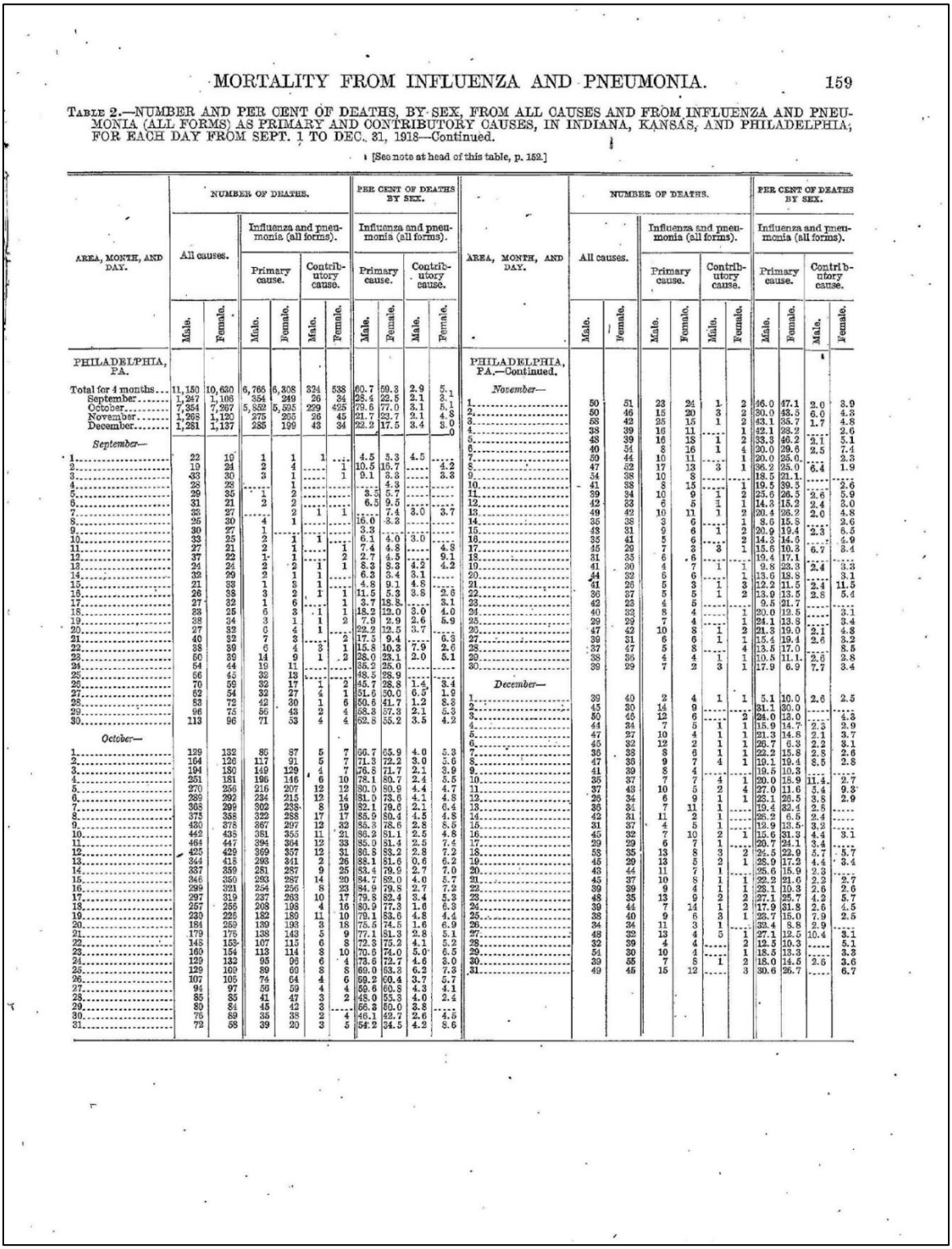
INFLUENZA AND PNEUMONIA

INDIANA, KANSAS, AND PHILADELPHIA, PA.

SEPTEMBER 1 TO DECEMBER 31, 1918

WASHINGTON
GOVERNMENT PRINTING OFFICE
1920

Appendix B. Example Excerpt from Department of Commerce Census Records in Philadelphia, PA 1918



MORTALITY FROM INFLUENZA AND PNEUMONIA. 159

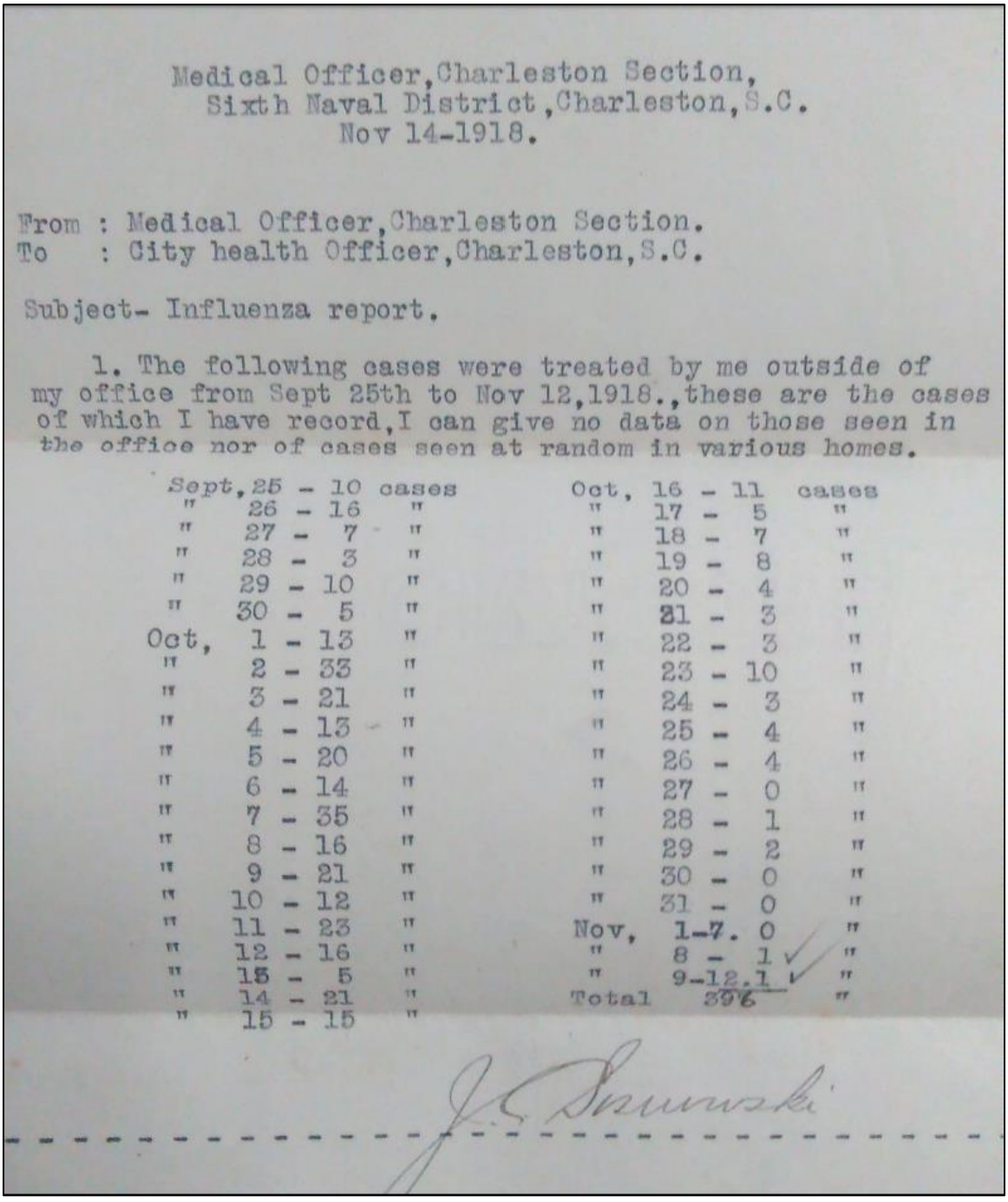
TABLE 2.—NUMBER AND PER CENT OF DEATHS, BY SEX, FROM ALL CAUSES AND FROM INFLUENZA AND PNEUMONIA (ALL FORMS) AS PRIMARY AND CONTRIBUTORY CAUSES, IN INDIANA, KANSAS, AND PHILADELPHIA, FOR EACH DAY FROM SEPT. 1 TO DEC. 31, 1918—Continued.

[See note at head of this table, p. 152.]

AREA, MONTH, AND DAY.	NUMBER OF DEATHS.						PER CENT OF DEATHS BY SEX.			
	All causes.		Influenza and pneumonia (all forms).				Influenza and pneumonia (all forms).			
			Primary cause.		Contributory cause.		Primary cause.		Contributory cause.	
	Male.	Female.	Male.	Female.	Male.	Female.	Male.	Female.	Male.	Female.
PHILADELPHIA, PA.										
Total for 4 months	11,150	10,630	6,765	6,308	324	538	60.7	59.3	2.9	5.1
September	1,247	1,106	354	249	26	34	28.4	22.5	2.1	3.1
October	7,354	7,267	5,332	5,525	229	425	72.5	77.0	3.1	5.8
November	1,268	1,120	275	205	26	45	21.7	23.7	2.1	4.0
December	1,281	1,137	285	199	43	34	22.2	17.5	3.4	3.0
September—										
1	22	19	1	1	1	...	4.5	5.3	4.5	...
2	19	24	2	4	...	1	10.5	16.7	...	4.2
3	33	30	3	1	...	1	9.1	3.3	...	3.3
4	28	28	...	1	...	...	...	4.3	...	...
5	29	35	1	2	...	...	3.5	5.7	...	...
6	31	21	2	2	...	...	6.5	9.5	...	...
7	35	27	...	2	1	1	...	7.4	3.0	3.7
8	25	30	4	1	...	...	16.0	3.3	...	...
9	30	27	1	...	...	...	3.3	...	...	...
10	33	25	2	1	1	...	6.1	4.0	3.0	...
11	27	21	2	1	...	1	7.4	4.8	...	4.8
12	37	22	1	1	2	...	2.7	4.5	...	9.1
13	24	24	2	1	1	1	8.3	4.2	4.2	4.2
14	32	29	2	1	1	...	6.2	3.4	3.1	...
15	21	33	1	3	1	...	4.8	9.1	4.8	...
16	26	38	3	2	1	1	11.5	5.3	3.8	2.6
17	27	32	1	6	...	1	3.7	18.8	...	3.1
18	33	25	6	3	1	1	18.2	12.0	3.0	4.0
19	38	34	3	1	1	2	7.9	2.9	2.6	5.9
20	27	32	6	4	1	...	22.2	12.5	3.7	...
21	40	32	7	3	...	2	17.5	9.4	...	6.3
22	33	39	6	4	2	1	15.8	10.3	7.9	2.6
23	50	39	14	9	1	2	28.0	23.1	2.0	5.1
24	54	44	19	11	...	...	35.2	25.0	...	...
25	66	45	32	13	...	...	48.5	28.9	...	...
26	70	59	32	17	1	2	45.7	28.8	1.4	3.4
27	62	54	32	27	4	1	51.6	50.0	6.5	1.9
28	53	72	42	30	1	6	50.6	41.7	1.2	8.3
29	96	75	56	43	2	4	58.3	57.3	2.1	5.3
30	113	96	71	53	4	4	62.8	55.2	3.5	4.2
October—										
1	129	132	86	87	5	7	66.7	65.9	4.0	5.3
2	104	126	117	91	5	7	71.7	72.2	3.0	5.6
3	194	180	149	129	4	7	76.8	71.7	2.1	3.9
4	251	181	196	146	6	10	78.1	80.7	2.4	5.5
5	270	256	216	207	12	12	80.0	80.9	4.4	4.7
6	289	292	234	215	12	14	81.0	73.6	4.1	4.8
7	368	299	302	238	8	19	82.1	79.6	2.1	6.4
8	375	358	322	288	17	17	85.9	80.4	4.5	4.8
9	430	378	367	297	12	32	85.3	78.6	2.8	8.5
10	442	436	381	355	11	21	86.2	81.1	2.5	4.8
11	461	447	396	364	12	33	85.9	81.4	2.5	7.4
12	425	429	369	337	12	31	86.8	78.2	2.8	7.2
13	344	418	293	311	2	26	88.1	81.6	0.6	6.2
14	337	359	281	287	9	25	83.4	79.9	2.7	7.0
15	346	350	293	287	14	20	84.7	82.0	4.0	5.7
16	299	321	254	256	8	23	84.9	79.8	2.7	7.2
17	297	319	237	263	10	17	79.8	82.4	3.4	5.3
18	257	255	208	198	4	16	80.9	77.3	1.6	6.3
19	230	225	182	189	11	10	79.1	83.6	4.8	4.4
20	184	253	139	193	3	18	75.5	74.5	1.6	6.9
21	179	175	138	143	5	9	77.1	81.3	2.8	5.1
22	148	155	107	115	6	8	72.3	75.2	4.1	5.2
23	160	154	113	114	8	10	70.6	74.0	5.0	6.5
24	129	132	95	98	6	4	73.6	72.7	4.6	3.0
25	129	109	89	69	8	8	69.0	63.3	6.2	7.3
26	107	105	74	64	4	6	69.2	60.4	3.7	5.7
27	94	97	56	59	4	4	59.6	60.8	4.3	4.1
28	85	85	41	47	3	2	48.0	55.3	4.0	2.4
29	80	84	45	42	3	...	56.3	50.0	3.8	...
30	75	89	35	38	2	4	46.1	42.7	2.6	4.5
31	72	58	39	20	3	5	54.2	34.5	4.2	8.6

AREA, MONTH, AND DAY.	NUMBER OF DEATHS.						PER CENT OF DEATHS BY SEX.			
	All causes.		Influenza and pneumonia (all forms).				Influenza and pneumonia (all forms).			
			Primary cause.		Contributory cause.		Primary cause.		Contributory cause.	
	Male.	Female.	Male.	Female.	Male.	Female.	Male.	Female.	Male.	Female.
PHILADELPHIA, PA.—Continued.										
November—										
1	50	51	23	24	1	2	46.0	47.1	2.0	3.9
2	50	46	15	20	3	2	30.0	43.5	6.0	4.3
3	58	42	25	15	1	2	43.1	35.7	1.7	4.8
4	38	39	16	11	...	1	42.1	28.2	...	2.6
5	48	39	16	13	1	2	33.3	46.2	2.1	5.1
6	40	54	8	16	1	4	20.0	29.6	2.5	7.4
7	50	44	10	11	...	1	20.0	25.0	...	2.3
8	47	32	17	13	3	1	36.2	25.0	6.4	1.9
9	34	38	10	8	...	...	18.5	21.1	...	...
10	41	38	8	13	...	1	19.5	39.5	...	2.6
11	39	34	10	9	1	2	25.6	26.5	2.6	5.9
12	42	33	6	5	1	1	14.3	15.2	2.4	3.0
13	49	42	10	11	1	2	20.4	26.2	2.0	4.8
14	35	38	3	6	...	1	8.6	15.8	...	2.6
15	33	31	9	6	1	2	20.9	19.4	2.3	5.5
16	35	41	5	6	...	2	14.3	14.6	...	4.9
17	45	29	7	3	3	1	15.6	10.3	6.7	3.4
18	31	33	6	6	...	...	19.4	17.1	...	...
19	41	30	4	7	1	1	9.8	23.3	2.4	3.3
20	44	32	6	6	...	1	13.6	18.8	...	3.1
21	41	26	5	3	1	3	12.2	11.5	2.4	11.5
22	36	37	5	5	1	2	13.9	13.5	2.8	5.4
23	42	23	4	5	...	...	9.5	21.7	...	...
24	40	32	8	4	...	1	20.0	12.5	...	3.1
25	29	29	7	4	...	1	24.1	13.8	...	3.4
26	47	42	10	8	1	2	21.3	19.0	2.1	4.8
27	39	31	6	6	1	1	15.4	19.4	2.6	3.2
28	37	47	5	8	...	4	13.5	17.0	...	8.5
29	38	36	4	4	1	1	10.5	11.1	2.6	2.8
30	39	29	7	2	3	1	17.9	6.9	7.7	3.4
December—										
1	39	40	2	4	1	1	5.1	10.0	2.6	2.5
2	45	30	14	9	...	...	31.1	30.0	...	...
3	50	43	12	6	...	2	24.0	13.9	...	4.3
4	44	34	7	5	1	1	15.9	14.7	2.3	2.9
5	47	27	10	4	1	1	21.3	14.8	2.1	3.7
6	45	32	12	2	1	1	26.7	6.3	2.2	3.1
7	36	33	8	6	1	1	22.2	15.8	2.8	3.0
8	47	36	9	7	4	1	19.1	19.4	8.5	2.8
9	41	39	8	4	...	...	19.5	10.3	...	...
10	35	37	7	7	4	1	20.0	18.9	11.4	2.7
11	37	43	10	5	2	4	27.0	11.6	5.4	9.3
12	26	34	6	9	1	1	23.1	26.5	3.8	2.9
13	36	34	7	11	1	...	19.4	32.4	2.8	...
14	42	31	11	2	1	...	26.2	6.5	2.4	...
15	31	37	4	5	1	...	12.9	13.5	3.2	...
16	45	32	7	10	2	1	15.6	31.3	4.4	3.1
17	29	29	6	7	1	...	20.7	24.1	3.4	...
18	53	35	13	8	1	2	24.5	22.9	5.7	5.7
19	45	29	13	5	2	1	28.9	17.2	4.4	3.4
20	43	44	11	7	1	...	25.6	15.9	2.3	...
21	45	37	10	8	1	1	22.2	21.6	2.2	2.7
22	39	39	9	4	1	1	23.1	10.2	2.6	2.6
23	48	35	13	9	2	2	27.1	25.7	4.2	5.7
24	39	44	7	14	1	2	17.9	31.8	2.6	4.5
25	38	40	9	6	3	1	23.7	15.0	7.9	2.5
26	34	34	11	3	1	...	32.4	8.8	2.9	...
27	49	33	13	4	5	1	27.1	12.5	10.4	3.1
28	32	39	4	4	...	2	12.5	10.3	...	5.1
29	54	30	10	4	...	1	18.5	13.3	...	3.3
30	39	55	7	8	1	2	18.0	14.5	2.6	3.6
31	49	45	15	12	...	3	30.6	26.7	...	6.7

Appendix C. Historical Naval Records from the Sixth Naval District in Charleston, SC November 14, 1918



Medical Officer, Charleston Section,
Sixth Naval District, Charleston, S.C.
Nov 14-1918.

From : Medical Officer, Charleston Section.
To : City health Officer, Charleston, S.C.

Subject- Influenza report.

1. The following cases were treated by me outside of my office from Sept 25th to Nov 12, 1918., these are the cases of which I have record, I can give no data on those seen in the office nor of cases seen at random in various homes.

Sept,	25 - 10	cases	Oct,	16 - 11	cases
"	26 - 16	"	"	17 - 5	"
"	27 - 7	"	"	18 - 7	"
"	28 - 3	"	"	19 - 8	"
"	29 - 10	"	"	20 - 4	"
"	30 - 5	"	"	21 - 3	"
Oct,	1 - 13	"	"	22 - 3	"
"	2 - 33	"	"	23 - 10	"
"	3 - 21	"	"	24 - 3	"
"	4 - 13	"	"	25 - 4	"
"	5 - 20	"	"	26 - 4	"
"	6 - 14	"	"	27 - 0	"
"	7 - 35	"	"	28 - 1	"
"	8 - 16	"	"	29 - 2	"
"	9 - 21	"	"	30 - 0	"
"	10 - 12	"	"	31 - 0	"
"	11 - 23	"	Nov,	1-7. 0	"
"	12 - 16	"	"	8 - 1	"
"	13 - 5	"	"	9-12.1	"
"	14 - 21	"	Total	396	"
"	15 - 15	"			

J. S. Szumski

Appendix D. Example Excerpts from Historical Charleston Civilian Death Records Recorded from Charleston Public Library Reel 2F: Return of Deaths – White – Jan 1, 1907 – Dec. 31, 1926

Return of Deaths Within the City of Charleston, S. C.

No.	Name	Sex	Color	Date of Death	Disease or Cause of Death	Place of Death	Age Yrs.	Mos.	Days
117	Beelow John Charles	M	White	Jan. 29	Angina Pectoris	70 Colonial	48	1	17
70	Bissonette Guy R.	"	"	" 10	Arterio Sclerosis	71 Mazyck	78	8	25
58	Baker, Mrs. Daisy P.	F	"	" 8	[illegible]	St Francis Inf.	27	–	14
154	Boinest Daniel	M	"	" 25	Heart Disease of Valv.	1 Halsey	75	9	11
20	Bill Mrs. Lillian B.	F	"	" 3	Influenza – Pneumonia	Baker San.	36	4	18
127	Bilgore, Mrs. Elizabeth L.	"	"	" 20	...... Asphyxia	4 Hampton Pl.	27	1	20
113	Banov Katie	"	"	" 20	Pneumonia – Influenza	595 King	14	6	25
125	Bowick Robert T.	M	"	" 20	......	43 Cannon	30	—	
24	Butler, Mrs. Annie G.	F	"	" 3	Ovarian Cyst	Baker San.	48	—	
111	Bum Lillian Olive	"	"	" 18	Tuberculosis	17 Chinquepin	22	6	19
114	Baskin Joseph A.	M	"	" 16	Acute Cardiac Dilatation; Acute Int. Obstruction – Tumor of Colon	Roper Hospital	65	2	13
344	Bequest, Mrs. Mary Condon	F	W	Mch. 4	Cancer Breast	Roper Hospital	52	—	
415	Blackman Henry Potter	M	"	" 21	Myocarditis	75 Pitt	69	11	15
390	Bishop, Mrs. Caroline E.	F	"	" 12	Pellagra	156 St Philip	71	—	
498	Burbage, Mrs. Mary F.	F	"	April 8	Brights Disease Chr.	789 St Philip	66	—	
559	Baker William H.	M	"	" 27	Diabetes Mellitus	72 Cannon	49	6	1
566	Bresch John Ulrich	"	"	" 24	Heart Disease of [illegible]	Riverside Inf.	40	2	18
523	Bold Francis H.	"	"	" 18	Meningitis Fulminating	Baker San.	5	—	
Washington State	Bahr David F.	"	"	" 17	Gunshot Wound	Washington State	32	—	
692	Burden William B.	M	"	May 26	Apoplexy – Brights Dis. Chr.	Roper Hospital	50	—	
698	Burnet Barnwell Rhett	"	"	" 28	Gangrene of leg – amputation	169 Queen	74	8	19
711 Amarillo	Bohlcke J.C. H.	"	"	" 30	Thrombosis – Femoral Artery	Riverside Inf.	63	5	15
Texas	Burn, Miss Georgia	F	"	" 15	Central softening	Amarillo Texas	72	—	
791	Bleckley Baby	M	"	June 18	Ante Partum asphyxia	St Francis Inf.	—	30 minutes	
755	Bremer Adaline	F	"	" 8	Arterio Sclerosis	Temple Home	71	2	—

ty of Charleston, S. C. For 1919

Disease or Cause of Death	Place of Death	Age Yrs.	Age Mos.	Age Days	Place of Nativity	Attending Physician or Coroner	Place of Interment
Angina Pectoris	70 Colonial	48	1	17	City	A. J. Buist	Magnolia
Arterio Sclerosis	7 Mazyck	78	8	25	Humboldt, Kan.	Dan. L. Maguire	Riverview
Ac. Delantia Tympanic Renal Suppression	St Francis Inf.	27	-	14	Hampton, S.C.	G. D. Wilson	St Lawrence
Heart Disease of Valv.	1 Halsey	75	9	11	City	Allen J. Jervey	Magnolia
Influenza - Pneumonia	Baker San.	36	4	18	St. Stephens S.C.	J. A. Ball	St Stephens S.C.
.... Influenza	4 Hampton Pl.	27	1	20	City	J. C. Mitchell	St Lawrence
Pneumonia - Influenza	595 King	14	6	25	"	K. J. Pearlstine	K.K.B.S.A. 3 Mile
....)	48 Cannon	30	—		"	....	Bethany
Ovarian Cyst	Baker San.	48	—		Orangeburg S.C.	J. T. Hiott	Orangeburg S.C.
Tuberculosis	17 Chinquapin	22	6	19	City	J. Aus. Finger	Magnolia
Acute Cardiac Dilatation / Partial Int. Obstruction - Tumor of Colon	Roper Hospital	65	2	12	Camden S.C.	J. A. Ball	Florence S.C.
Cancer Breast	Roper Hospital	52	—		Sullivan Isl.	C. P. Aimar	Mt. Pleasant
Myocarditis	75 Pitt	69	11	15	City	J. H. Martin	Magnolia
Pellagra	156 St Philip	71	—		"	Henry Deas	......
Brights Disease Chr.	789 St Philip	66	—		Summerville S.C.	J. S. Rhame	Jedburg S.C.
Diabetes Mellitus	72 Cannon	49	6	1	Sumter S.C.	....	Magnolia
Heart Disease of Valv.	Riverside Inf.	40	2	18	City	J. P. DesChamps	Bethany
Meningitis Fulminating	Baker San.	5	—		"	R. M. Pollitzer	....
Gunshot Wound	Washington State	32	—		—	Mabel Pr. Reg.	Magnolia
Apoplexy - Brights Dis. Chr.	Roper Hospital	50	—		City	S. McF. Mood	Held for shipment
Gangrene of leg - amputation	169 Queen	74	8	19	Beaufort S.C.	Edw. Rutledge	Magnolia
Thrombosis - femoral artery	Riverside Inf.	63	5	15	Germany	A. J. Buist	Bethany
Central softening	Amarillo Texas	72	—		—	E. A. Lemans	Magnolia
Ante Partum asphyxia	St Francis Inf.	—		20 min.	City	D. L. Maguire	Magnolia
Arterio Sclerosis	Ittmagle Home	71	2	—	Germany	Chas. D. Butte	Bethany

Return of Deaths Within the City of Charleston, S. C. For

No.	Name	Sex	Color	Date of Death	Disease or Cause of Death	Place of Death	Yrs.	Mos.	Days
1660 / 1853	Bramble Aug. Evans	M	W	Oct. 17	Aneurism Aorta	86 Huger	69	—	
1859	Bruce Dr. James P.	"	"	" 13	Bronchitis – Pneumonia	516 King	78	10	19
1851	Berry Lucy	F	"	" 17	Dementia Precox	Roper Hospital	39	—	
1792	Bowers Henry Preston	M	"	" 14	Diphtheria	Baker Sm.	10	7	8
1796	Bryan J. P. Kennedy	"	"	" 25	Indigestion Acute	47 S. Battery	66	1	15
1804	Burbage Elz. E.	F	"	" 24	Influenza	58 N. King	—	7	—
1786	Branham Mrs. Sarah A.	"	"	" 28	..... – Childbirth	84 Mary	24	3	14
1756	Borronich B. Chas.	M	"	" 23	..... – Pneumonia	Charleston Orphan House	5	9	8
1776	Benton Thomas E.	"	"	" 25	..... – ..... [illegible]	Royal Mills Hospital	3	7	7
1566	Bunch Ralph Waldo	"	"	" 25	..... –	Chas. Orphan House	4	5	25
1649	Bellom Martha A.	F	"	" 10	Pneumonia Broncho – Influenza	141 Cannon	—	7	25
1578	Beard Mrs. S. H.	"	"	" 19	....	Roper Hospital	33	—	
1683	Boland Claude Wm.	M	"	" 16	.....	73 B. George	6	—	12
1803	Bailey Mrs. Janie	F	"	" 19	.....	Roper Hospital	28	—	
1647	Barnes Mrs. Elz. C. R.	"	"	" 26	..... Ectar – Syphilis	.....	34	—	
1565	Baker Virginia	"	"	" 14	Senility	40 Calhoun	85	—	
accident 1644	Bates Alford S.	M	"	" 15	Accident – Gunshot Wound in Chest	R. Hospital	18	—	
accident 1805	Benson John Wesley	"	"	" 19	..... – Skull Fracture Automobile	.....	17	—	
1813	Boyle Inf. of Carolina E.	F	"	" 29	Stillborn	150 Ashley	—		
Phil. Pa.	Bassett Burton B.	M	"	" 11	Influenza	Phil. Pa.	19	—	
Kansas City Mo.	Boette Ernest A.	"	"	" 10	Pneumonia Broncho – Influenza	Kansas City Mo.	20	—	
Newport News Va.	Bradley David B.	"	"	" 17	.....	Camp Eustis Newport News Va.	21	—	
Chicago Ill.	Bryan Arthur	"	"	" 29	.....	Chicago Ill.	34	1	7
Edgewood Md.	Bennett Wm. A.	"	"	" 15	.....	Base Hospital Edgewood Md.	—		
Columbia S.C.	Belding Mrs. Annie Mary	F	"	" 24	Pellagra	State Hospital Columbia S.C.	76?	—	
1846 / 1920	Bailey Elizabeth M.	F	"	Nov. 6	Cerebro-Spinal Fever Ep.	Roper Hospital	9	—	
1869	Bolchoz Mrs. Jessie L.	"	"	" 20	Influenza – Pneumonia	21 Ashburne	60	—	
1858	Blandell John	M	"	" 9	Pneumonia – Influenza	Roper Hospital	32	—	
1813	Brown (Infant of Jessie)	"	"	" 3	Stillborn	St. Francis Inf.	—		
Summerville S.C.	Bernesteine Rosa	F	"	" 14	Influenza Pneumonia	Summerville S.C.	1	—	