ABSTRACT

THE 1878 YELLOW FEVER EPIDEMIC IN MEMPHIS, TENNESSEE:
AN HISTORICAL GEOGRAPHIC INFORMATION SYSTEMS (HGIS) APPROACH

By

Sheridan Wright Kennedy

August 2011

In 1878, one of the worst yellow fever epidemics in the history of the United States struck Memphis, Tennessee. This study uses geographic information systems (GIS) to trace the spread of yellow fever during the epidemic by identifying and analyzing areas of disease mortality across time. Methods from geography, history, and epidemiology are employed in a multidisciplinary examination of this catastrophic event. This work contributes new knowledge about the epidemic, provides a methodological expansion to the subfield of historical geographic information systems (HGIS), and it identifies and corrects an error in an important primary source. This study demonstrates the importance of GIS as a tool in historical research and shows how it can enhance the understanding of historical events.

KEYWORDS: Geographic Information Systems (GIS), Historical Geographic Information Systems (HGIS), Yellow Fever, 1878 Yellow Fever Epidemic, Memphis.
THE 1878 YELLOW FEVER EPIDEMIC IN MEMPHIS, TENNESSEE:
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SYSTEMS (HGIS) APPROACH

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CHAPTER 1

INTRODUCTION

Purpose

The purpose of this study is to examine yellow fever diffusion through Memphis, Tennessee during the 1878 epidemic. This study utilizes geographic information systems (GIS) to identify and analyze areas of disease mortality across time. Additionally, sources and methods from the fields of epidemiology and history are coupled with geography to understand these patterns. Using this multidisciplinary toolkit to study detailed historical medical records produces a new spatial history. This work contributes new knowledge about the epidemic and a methodological expansion to the subfield of historical geographic information systems (HGIS), while it identifies and corrects an error in an important primary source.

In recent years, the number of yellow fever cases has risen, along with the risk of localized epidemics (Rogers et al. 2006). The World Health Organization estimates there are 200,000 yellow fever cases annually (World Health Organization 2011). Examining the spatial and temporal patterns will not only help in future surveillance and identification of emerging yellow fever epidemics, but will enhance methods for understanding and tracking other vector-based diseases in urban environments. This study combines historical and geographical methods to better understand the 1878 yellow fever epidemic in Memphis, Tennessee.
Studies on yellow fever have examined the importance and consequences of historical epidemics without analyzing the inherently spatial components (Capers 1938; Baker 1968; Ellis 1992; Humphreys 1992; Bloom 1993; Carrigan 1994; Espinosa 2006; Nuwer 2009). Furthermore, few studies have analyzed both the spatial and temporal components of past yellow fever epidemics (Arden 2005; Curtis, Mills, and Blackburn 2007; Curtis 2008). Finally, no work has rigorously used historical sources and geospatial techniques to investigate yellow fever epidemics. Indeed, some scholars have criticized the lack of historical methods in HGIS as a barrier to the broader adoption of GIS in history (Knowles and Hillier 2008). The research presented in this thesis aims to redress these oversights.

Chapter One introduces the purpose of this thesis. To provide context for this research project, it includes the background on the city of Memphis, as well as an overview of yellow fever, and the importance of the 1878 epidemic. Chapter Two situates this thesis in the body of academic knowledge. The literature review is divided into two sections. The first section reviews the methodologies and the emerging subfield of technologically-enabled historical research. This focuses specifically on the advantages of spatial analysis and HGIS. The second section is an examination of the sub-discipline of medical geography. This reviews the history of the field, past disease mapping research, efforts to understand yellow fever through mapping, and finally GIS studies of yellow fever.

Chapter Three presents the methodology for this research. Techniques, methods, and analyses used in this study are detailed. This chapter also includes a full review, assessment, and source analysis of the data, historical records, and maps used in this
thesis. Chapter Four presents the analytical results and the associated visualizations. Chapter Five discusses the results of this study, including the new findings, the limitations uncovered, and the value of this study to geography, history, and HGIS.

**Background**

Yellow fever is thought to have originated in West Africa and been transported to the Americas by European trade ships. One of the first documented outbreaks of yellow fever was by the Spanish on the Yucatan Peninsula in 1648. In the eighteenth and nineteenth centuries, yellow fever epidemics in the United States often originated from an outbreak at a trade hub in the West Indies (e.g., Havana) or South America (e.g., Rio de Janeiro) (Hardenstein 1879; Espinosa 2006). The virus would be transported via ship to port cities of the United States. If this unintentionally imported virus came in contact with a locale containing a susceptible populace and a population of *Aëdes aegypti* mosquito (the disease vector), with the right timing, a yellow fever epidemic was possible.

The 1870s were a turbulent time in the history of the United States. The Panic of 1873, a period of financial instability, caused woes in both the United States and abroad. The country was still in a state of unrest following the Civil War and the ongoing Reconstruction. Tensions were high amid political and social strife. The U.S. military occupied Southern states through most of the decade. With both candidates claiming victory after the votes were tallied, the 1876 presidential election threatened to deteriorate into violence. Informal bipartisan negotiations led to a deal. The Compromise of 1877 ended Reconstruction (which included the withdrawal of federal troops from Louisiana and South Carolina) in exchange for Southern Democrats shifting their support to the
Republican candidate, Rutherford Hayes (Tindall and Shi 2004). The Mississippi River Valley, although economically fragile due to low cotton prices, hoped the end of Reconstruction and the prospect of federal infrastructure investment signaled the beginning of a new era of prosperity.

The Mississippi River brought life and death to local inhabitants. Cities and towns lining the banks of the river were connected to an international trade network that offered great economic possibilities. The movement of people and goods along this network was not easily halted, likewise, neither was the spread of disease. A yellow fever epidemic in 1873, five years before the outbreak examined in this study, made this readily apparent to the people of Memphis. In the late summer and early fall of that year, five thousand people contracted the disease and for two thousand of those people, it proved fatal. Cholera and smallpox outbreaks struck Memphis the same year (Billings 1881).

Among Southern cities, federal troops occupied Memphis early in the Civil War. Because of this, Memphis fared well throughout the war. Postbellum Radical Reconstruction and a notable immigration of unskilled laborers, many of whom were former slaves, embittered the middle class of Memphis (Capers 1939). Widespread political corruption and poor investments left the city government heavily in debt and ineffective by 1878. The grotesque sanitation in Memphis was talked about nationally, if not internationally (Murtough 1879; Thornton 1886). The city government relied on business owners to pay for cleanup initiatives (Keating 1879). Memphis did not have the fiscal or economic resources to enforce a stringent health code or strict quarantine. Even before the epidemic of 1878, there were motions to repeal the city charter (Capers 1939).
The weak government and poor sanitation in Memphis increased the risk of epidemic in the city.

The Virus

The yellow fever virus, of the genus *flavivirus*, is an acute viral hemorrhagic disease. Yellow fever is a communicable vector-borne disease (World Health Organization 2011). The transmission of the disease follows a *host-vector-host* pattern. When a susceptible person is infected with the agent (disease), that person becomes a host (Cromley and McLafferty 2002). While yellow fever is endemic to the tropics, the geographic range of this virus is limited only by the range of its arthropodal vector (Carter 1931). The *Aëdes aegypti* mosquito was the exclusive vector of eighteenth and nineteenth century urban yellow fever in the United States (Christophers 1960).

The Spanish called the disease *vomito negro* after one of its more gruesome symptoms (Keating 1879). This black vomit consists of partially digested blood, the result of slow internal hemorrhaging. The name *yellow fever* was also derived from a symptom of the disease: jaundice of the liver causes the infected person’s sclera (white of the eye) and skin to take on a yellowish hue. Additional symptoms of the disease include muscle aches, high fever, suppression of urine, and bleeding from nose, mouth and gums. Of all these symptoms, the hematemesis (i.e., vomiting blood; with yellow fever the blood is partially digested and has turned black due to a reaction with the gastrointestinal acid) can be the most indicative sign of yellow fever (World Health Organization 2011). Other diseases have many of the same symptoms as yellow fever, but the hematemesis has been used to distinguish yellow fever (Carter 1931). Symptoms vary greatly from person to person, complicating diagnosis.
From initial infection with the yellow fever virus, a person undergoes an incubation period which typically lasts three to six days. Near the end of this presymptomatic period, a patient develops high levels of viremia (i.e., the concentration of the virus in the bloodstream). During this three to five day period of increased viremia, a mosquito that feeds on the host will ingest the virus and, after a ten to twelve day extrinsic incubation period, spread it to any subsequent hosts on whom it feeds. After the three to six day period of incubation in the host, symptoms of the acute phase appear. These symptoms often include fever, aches, pains, and vomiting. The acute phase generally lasts three to four days, after which there is a twenty-four hour period of remission. At this point, an infected person will either convalesce or enter into the toxic phase. The common symptoms of the toxic phase are jaundice, internal and external hemorrhaging, high fever, and vomiting. The deterioration of the internal organs usually leads to death in three to seven days. If a host survives the virus, immunity to the yellow fever virus is acquired (World Health Organization 2011).

Yellow fever virus strains differ by geographic origin. Because of isolation, evolutionary changes have differentiated strains from South America, West Africa, and Central-East Africa. The most important characteristic of differentiation is virulence of the virus. However, the present-day variance in virulence of these strains is unproven (Lepiniec et al. 1994; Mutebi et al. 2001; Mutebi et al. 2004).

The yellow fever of 1878 in the United States was determined by contemporary medical practitioners to be “. . . peculiarly virulent and violent, and particularly fatal” (Keating 1879, 72). The secondary fever (known as the toxic phase in modern medicine) of the 1878 form of the disease was accelerated (Keating 1878; Maury 1881). While it
was generally understood that “. . . one attack usually [gave] immunity from a second,” (Keating 1878, 318), this did not guarantee protection from the more malignant version of yellow fever in 1878. Therefore, the size of the susceptible population was increased. Compounding the risk, poverty limited the mobility of a substantial portion of the susceptible population. This divided the host population in Memphis into two parts, mobile and immobile. Out of a population of 50,000, approximately 20,000 people did not or could not flee the city during the epidemic (Keating 1879).

Diagnosis of yellow fever is difficult and unreliable in present-day medicine without a virus-specific test of serum or cerebrospinal fluid (Steffen 2010). In the 1870s, prior to a full blown epidemic, singular cases were often misdiagnosed or disputed (Maury 1881). Yellow fever was misdiagnosed, among other things, as “hemorrhagic fever,” “typho-malarial fever,” “congestive fever,” “congestion of the brain,” or “jaundice” (Dunglison 1876; Maury 1881; Anderton and Leonard 2004). Physicians in port cities often diagnosed suspicious cases with a certain tact. The word “quarantine” was a death knell to trade merchants and associated businesspersons. If yellow fever was announced to be present in a city, quarantines were often raised against the beleaguered city (Keating 1879). Therefore, when a yellow fever case or death was announced in a trade-based city, it was generally the result of overwhelming evidence.

Transmission

Mosquitoes are the vector of yellow fever, though scientists would not confirm this until 1900. In this year, Walter Reed clinically proved the transmission of yellow fever by the vector *A. aegypti* (Reed et al. 1900). While other species in the genus *Aëdes* are known to transmit yellow fever at present, in the eighteenth and nineteenth century
urban yellow fever epidemics in the United States, *A. aegypti* was the primary vector. Characteristics and traits of *A. aegypti* vary greatly from other mosquitoes, like the malaria vector, genus *Anopheles*. In the built environment, *A. aegypti* almost exclusively breeds in water that is confined and collected artificially. This ranges from cisterns to flower vases to cracks in masonry. *A. aegypti* is equally adept to breeding in collections of water in the gaps between planks of ships. The species prefers water with moderate to no organic matter (Christophers 1960). Generally, this mosquito only makes short flights and tends to stay in one dwelling. With sufficient resources, *A. aegypti* typically does not fly more than 50 meters from its breeding area (Christophers 1960).

*Aëdes aegypti* generally feeds during the afternoon, but will feed at night in lighted rooms. The optimal feeding conditions are around 82° Fahrenheit with low wind. Between 63° and 90° Fahrenheit, the mosquito will feed, but these are the minimum and maximum temperature ranges. Exposure to temperatures near freezing is debilitating and often lethal to the insect (Christophers 1960). This explains the seasonality of yellow fever in North America (summer to fall). Even with the transmission vector and mechanics of yellow fever unknown in the 1870s, people recognized the ameliorating nature of cold weather. Seldom found throughout the history of humankind, winter was embraced as a saving grace in cities like Memphis.

To become a vector of yellow fever, *A. aegypti* first feeds on a person with viremia and then undergoes an extrinsic incubation period. As previously stated, this period lasts approximately ten to twelve days (Christophers 1960). This is the time it takes for the RNA virus strand to move from the mid-gut of the mosquito to the salivary glands. At the same time, the RNA strand is replicating (World Health Organization
When a female mosquito feeds on a person, she first injects an anticoagulant via saliva into a person’s bloodstream (Christophers 1960). In this action, the blood-meal is made easier to withdraw with the aid of the anticoagulant, while the yellow fever virus RNA strand is injected as well (World Health Organization 2011). After the extrinsic incubation period, the mosquito can spread the virus throughout the remainder of its life. Six or seven weeks is considered the upward bounds for the effective epidemiological survival period of *A. aegypti* (Soper 1967).

Similar to the discussion about the yellow fever virus, its vector, *A. aegypti*, is thought to have originated in Africa and spread to the Americas on European ships (Chastel 2008). The geographic distribution of *A. aegypti* varied considerably throughout the nineteenth century. Yellow fever records can be used to track the historical distribution of *A. aegypti*. With no *A. aegypti* population, yellow fever transmission did not occur in the United States during this time period (Bloom 1993). When a group of people infected with yellow fever arrived in a locale and no new cases of yellow fever occurred, it can be assumed there was no substantial *A. aegypti* population (Bloom 1993). At the end of the eighteenth century and the beginning of the nineteenth century, *A. aegypti* was found in the port cities of the Southern and Eastern United States and sporadically about the Eastern interior (Keating 1879; Carter 1931). This distribution estimate is based on the locations of yellow fever epidemics during the time period. The species moved inland along the Mississippi River during the first half of the century. Vicksburg had a population of *A. aegypti* by 1839. However, this population was not well established, if present at all in Memphis during the summer and fall of 1853 (Bloom 1993). Over fifty yellow fever-infected people from New Orleans traveled to Memphis
in this year, but no new cases occurred. However, by 1867, the spread of yellow fever in Memphis substantiates the establishment of a large population of *A. aegypti* (Keating 1879; Carter 1931; Bloom 1993).

Residential construction practices played a roll in the spread of yellow fever. In 1878, breeding areas preferred by *A. aegypti*, like cisterns, numbered in the thousands in Memphis (Billings 1881; Christophers 1960). Over 1,200 dwellings were single-story, one or two-roomed buildings (Billings 1881). These structures are important in examining the prevalence of multi-death residences (Curtis 2008). This is in part because infected mosquitoes in this type of dwelling had few barriers separating them from the residents and the virus transmission can be more readily apparent.

Likely due to the oceanic and meteorological phenomenon known as El Niño, a wet and mild winter and spring led up to a warm summer in 1878. This created extremely favorable conditions for *A. aegypti* propagation (Diaz and McCabe 1999). The conditions in Memphis were ideal for a yellow fever epidemic.

**History of the 1878 Memphis Epidemic**

Reports of a yellow fever outbreak in the West Indies reached Memphis by early May of 1878 (Keating 1879). In late May and June, yellow fever lurked in New Orleans, but was not announced. The virus entered New Orleans through infected crewmembers and passengers on merchant ships servicing fever ports in the West Indies (Hardenstein 1879; White 1881). The quarantine stations set up to protect New Orleans from this pestilence failed primarily due to nondisclosure (by crewmembers) of early yellow fever symptoms to station physicians along with the inability of these physicians to diagnose presymptomatic cases (New Orleans Board of Health 1879; Ellis 1992). With rumors
spreading up river of yellow fever deaths in New Orleans, the president of the Memphis Board of Health, Dr. Robert W. Mitchell, began preparations for the defense of Memphis (Maury 1881; Hicks 1964). Based on the patterns of the 1867 and 1873 spread of yellow fever, he knew that virulent yellow fever in New Orleans usually meant Memphis was next. Owners of riverboats and steamers, giving priority to commerce, ignorantly began spreading yellow fever up the Mississippi River to Memphis and beyond.

In early June, Dr. Mitchell proposed that a quarantine station be set up to protect the city, but he was met with resistance from other physicians (Hicks 1964). With the propagation mechanics of yellow fever unknown and the state of public health/medicine inchoate, disagreement abounded. The medical community primarily was split between two opinions: one argued that yellow fever was imported into local environments, and the other side thought the disease to be of local origin and primarily due to poor sanitation and gases from aerated detritus (Keating 1879; Ellis 1992). As to the pressing issue of whether or not to quarantine the city, nearly half the physicians in Memphis believed that quarantine was not the right decision; thirty-two Memphis physicians were successful in petitioning the General Council not to impose quarantine. Memphis was heavily in debt and the physicians believed the proposed quarantine was either unnecessary at the time or an exercise in futility. With his recommendations rejected, Dr. Mitchell tendered his resignation to the General Council (Keating 1879; Hicks 1964).

After the Memphis yellow fever epidemics in 1867 and 1873, the city health officials began to view New Orleans as an early warning system for yellow fever. When there was a large outbreak of yellow fever in New Orleans, Memphis health officials tried to mitigate the risk of yellow fever appearing in their city. Upon request in June of 1878,
New Orleans health officials promised to notify Memphis officials immediately of any yellow fever cases in New Orleans (Keating 1879; Hicks 1964). Health officials in Memphis and around the Mississippi River Valley anxiously awaited news from New Orleans, though June passed without any official reports of yellow fever. In the July 26th newspapers, Memphis (including the health officials) and most of the United States read the first official declaration of the existence of new yellow fever cases in New Orleans (Bennett 1878; Maury 1881). Memphis quickly established quarantine for rail and river arrivals, but having chosen to be reactive instead of proactive, it was already too late (Hicks 1964). Unbeknownst to Memphis, by the last week of July, the virus was already active in the city (Maury 1881).

Anxiety and fear from the notion of yellow fever reaching Memphis started building in the population towards the end of July. On August 9th, rumors of a yellow fever outbreak in Grenada, Mississippi, excited the population of Memphis to a near panic (Keating 1879; Knights of Honor 1879). Grenada and Memphis were directly linked by a railway route. In Memphis, Kate Bionda, a trattoria proprietor at 212 Front, was taken with fever on this very same day (see Chapter Four, Figure 5, Location A). She died in the afternoon of August 13th, and the following day the Board of Health announced her death (Hicks 1964). It was the first confirmed case of yellow fever contracted in the city. The Board of Health responded to her death by disinfecting the buildings and streets within a half block and cordoning off the area directly adjacent to her residence (Hicks 1964).

The Board of Health recorded seven yellow fever related deaths for the 14th of August. The next day, it reported twenty-two new cases in the city. On August 16th, the
Board of Health met and decided to start making arrangements to evacuate all of the people residing in the infected district, which encompassed the First, Second, and Eighth wards along Poplar, Washington, and Adams (for city maps, see Chapter Four, Figure 5 and Appendix B, Figure 27). The city established refugee camps along the railroad lines and within ten miles outside of the city limits, and the Board of Health recommended citizens minimize contact with the infected district. Also, citizens residing in the infected district were encouraged to relocate to the refugee camps (Hicks 1964). On August 21st, isolated cases began appearing further south in the city. These cases were attributed to interactions with the infected district. Citizens also disregarded and destroyed the Board of Health’s barricades around infected residences. By August 23rd, yellow fever overcame attempts at confinement by city health officials, crossed Madison, and continued south. The southern boundary of the infected district was first set at Jefferson, then moved to Madison, and finally abandoned altogether. After this containment policy failed, the Board of Health officially declared yellow fever to be epidemic in the city of Memphis. By August 24th, many parts of the city had yellow fever. Officials realized that they would not be able to contain yellow fever in one district, a strategy which had proven more successful in the 1873 epidemic. The uninfected people remaining in the city were strongly encouraged to leave (Hicks 1964).

By the last week of August, an estimated 25,000 people had left Memphis. At the end of the first week in September, an additional 5,000 people had moved out of the city, many relocated to the refugee camps (Hicks 1964). An estimated 20,000 people remained in the city (Keating 1879). Around September 1st, the overwhelming morbidity and mortality had substantially reduced the ability of the Board of Health to accurately
report the number of new cases and deaths (Hicks 1964). The number of new cases and
deaths continued to rise throughout the first half of September. Dr. Mitchell estimated
six hundred new cases on September 7th (Keating 1878). The overworked Board of
Health shut down on September 11th because quorum could not be met (Hicks 1964).
The epidemic peaked on September 14th with unofficial reports listing more than two
hundred deaths on that Saturday, alone (Knights of Honor 1879).

Yellow fever appeared in most of the suburbs and in the countryside of Memphis
by September 18th (Keating 1878). Inside the city limits, new cases and deaths from
yellow fever noticeably dropped on September 20th (Knights of Honor 1879). The
Avalanche (2 October 1878) described the virus as spreading east into the country along
the rail lines (Gallaway and Pollard 1878). By October 2nd, the virus had infected people
within a twelve mile radius from the city (Knights of Honor 1879). The decrease in
yellow fever incidences inside the city limits was attributed to the near complete
disappearance of the susceptible population, whether by flight, death, or acquired
immunity (Murtough 1879). In mid-October, the temperature cooled, marking the
beginning of the end of the active Aëdes aegypti population, and thus, the spread of
yellow fever. The Board of Health encouraged the refugee population to remain away
from Memphis until multiple hard freezes end the threat of yellow fever (Hicks 1964).
On October 29th, the Board of Health officially declared the epidemic over (Keating
1879).*

* Retrospectively, a man with the surname Robinson was identified as the index
case in the city of Memphis (Keating 1879; Tyner 1879; Maury 1881). Stepping back to
around July 15th, Robinson traveled about fifty miles downriver from Memphis to
Moon’s Landing (Maury 1881). He returned to Memphis on July 21st and immediately
was taken with a fever. His wife treated him at their residence. He showed symptoms
Conclusion

Chapter One provided the background of the 1878 yellow fever epidemic in Memphis, Tennessee. Through the combination of methods from epidemiology, history, and geography, this study is designed to uncover new perspective on an historical event, develop an understanding of the spatiality of yellow fever diffusion in an urban environment, and contribute to the growing use of HGIS. Next, the etiology of the yellow fever virus was discussed, along with its pathology. To better portray the spatial aspects of the virus spread, the characteristics and physical limits of *Aëdes aegypti*, the vector of urban yellow fever, were presented. Historical context was provided through a brief account of the days before the epidemic. Lastly, the epidemic and its spatial dimensions were summarized using only textual resources. Before presenting a detailed discussion of the methods and analytical techniques used to track and explain the onset, spread, and effects of the yellow fever epidemic, the following chapter reviews the associated with the acute phase of yellow fever. Robinson survived the disease, but while he recovered, other cases started to appear in close proximity to his residence. From this city block, bounded by the streets of Poplar, Washington, Main, and Second, yellow fever quickly spread east and west along Poplar and Washington; these streets were major transportation arteries in the city (Maury 1881; Hicks 1964).

The area along Alabama Street was initially infected by two or three people from the *Golden Crown* steamboat. They stayed in Patrick Winter’s home at 36 Alabama (Maury 1881). Several different accounts of the circumstances surrounding this event exist (Keating 1879; Knights of Honor 1879; Murtough 1879; Tyner 1879; Maury 1881). Regardless of the particulars, yellow fever emerged along Alabama during the first week of August. From this area, the fever spread northeast to the intersection of Alabama and Johnson, and then along Johnson.

Another infected person, William Warren/Varne, also violated the quarantine by sneaking into Memphis off the *Golden Crown*. Again, there was considerable variation in the details of this case among the sources, but the consensus was that he died from yellow fever sometime between August 3rd and August 5th. Just prior to his death, William Warren was sent from the Memphis City Hospital to the quarantine station on the lower end of President’s Island (Keating 1879; Murtough 1879; Maury 1881).
existing literature on relevant historical methods, HGIS, medical geography, and yellow fever.
CHAPTER 2
LITERATURE REVIEW

Introduction

In order to examine the geography of the 1878 yellow fever epidemic in Memphis, this study draws from existing research in history and geography. Specifically, the current study is informed by quantitative history and medical geography through the use of geographic information systems (GIS). To visualize the spatial nature of the epidemic, historical sources (both maps and documents) are combined into a GIS. Connecting medical geography with history, GIS affords the means to incorporate a large number of historical sources and apply a variety of epidemiological and spatial analysis methods.

Chapter Two is divided into two major sections. Section One examines the historical research methods used in this study, including the linking of methods in history and geography through the growing specialty of historical geographic information systems (HGIS). Section Two discusses the sub-discipline of medical geography, which provides this study’s spatial-analytical framework and then concludes with a review of relevant research on yellow fever.

Section I: Advances in Historical Research Methods
Part One: Quantitative History

Historical sources form the backbone of all historical research. These sources vary from textual to oral traditions but, in essence, they all represent forms of
recordkeeping and record interpreting. This activity has been practiced throughout the history of civilization. In fact, human history is differentiated from prehistory by the existence of written records. Prior to database management software, researchers had limited capacities for the statistical analysis of records (Healey and Stamp 2000). Detailed analysis of large sets of unaggregated records was impractical. Inherently, quantitative sources in certain fields of study make these areas more readily approached with quantitative methods.

Shortly before the 1930s, researchers began using quantitative methods to study economic, fiscal, and demographic records (Furet 1971). These studies included statistical analyses and other mathematical formulations and computations of quantitative historical datasets (Clapham 1926; Usher 1932; Griffin 1939). The 1950s witnessed the marriage of quantitative methods and economic theory. This type of economic history became known as cliometrics (Marczewski 1968). The increased research in cliometrics coincided with the exponential increase in computing technology (Williamson and Mokyr 1994; Greif 1997). Technology transformed quantitative research. From punch cards to digital spreadsheets to network computing (e.g., World Wide Web), advances in technology have enabled an expansion in the scope of historical research.

The increases in computing capabilities of machines allow for unprecedented management of large historical datasets (Furet 1971; Healey and Stamp 2000). The data management capabilities of computing technology have increased more than a million-fold in the last forty years (Campbell-Kelly and Aspray 1996). With this new found ability for massive data storage, researchers have enjoyed unprecedented capabilities and flexibility in data manipulation and editing. Computer spreadsheet applications greatly
aid researchers’ work with datasets. Network data storage has not only expanded the individual capacity of computing machines, but it has also created an easily accessible avenue for information/data dissemination. Computer networking has increased the availability of historical datasets (Presnell 2007).

Network data sharing and the Internet have revolutionized information and data dissemination. Online forums, discussion boards, and electronic mail have greatly increased the amount of communication and information sharing between historians (Ayers 1999). Electronic archives offer unprecedented access to primary and secondary sources alike (Presnell 2007). Online clearinghouses provide historical datasets which are not only more readily available (than hardcopies), but may already be entered into digital spreadsheets. Digital datasets can greatly reduce the resource requirements for research projects. With this exponential increase in available information and data, researchers potentially have many more sources at their disposal, but they must still carefully analyze the quality of their sources. Database technology may aid in this type of analysis, and it also greatly extends the range and type of data analysis.

Analysis of data has the potential to unlock new historical knowledge. The technological limit of research using historical datasets is decreasingly set by hardware. Software is now the primary defining factor for the bounds of data analysis. Software packages, such as SPSS by IBM, JMP by SAS, and Excel by Microsoft, have broad analytical capabilities, with many popular statistical formulas and algorithms programmed into these packages (Fotheringham, Brunsdon, and Charlton 2004). In these programs, researchers have many options among a large set of semi-automated functions (tools) for data analysis from which to choose. Many of these tools are user-
customizable, and writing programming code can be used for more intricate customizations. Through statistical software packages, researchers can use analytical tools quickly and easily, which allows for potentially more computations to be performed at a minimal cost of project resources (i.e., time, money, labor) (Fotheringham, Brunsdon, and Charlton 2004). Additionally, statistical software packages have proven their ability to present data visually (Goebel and Gruenwald 1999; James et al. 2004; Friendly 2008).

For researchers, the visualization of historical data is an important function of computers. The visualization of data as a mode of quantitative study has been traced back to medieval times (Friendly 2008), though it has been revolutionized by the computer (Thomas 2001). Through increased automation, the process of graphing/charting data is simplified, accurate, and quick. Computers remove constraints of traditional data visualization, allowing for more advanced techniques, such as animation of change, three-dimensional work, and dynamic data models. This can increase the efficiency of exploratory data analysis. Presenting visual results, especially through these advanced methods, has limited effectiveness in traditional print media. Multimedia presentations, whether through PowerPoint by Microsoft, supplemental digital material, or the World Wide Web, have proven far more effective in conveying knowledge in these types of visualizations (Tosatto and Gribaudo 2009). In addition, these advanced visualizations can be effective teaching tools (Thomas 2001; Knowles and Hillier 2008; Tosatto and Gribaudo 2009).

Quantitative history has benefited from these advances in computing technology. Improved data management capacity enlarges the maximum size for operable datasets
and makes data more readily available. Statistical software packages provide researchers with a large array of analytical tools. The use of computers allows researchers to visualize data, more readily explore datasets, and present findings in a wider variety of methods and venues to academic and public audiences alike. Digital history, which has become the broad subheading for most technology-enabled historical research, has begun to change historical scholarship. Historians are working to create electronic articles which have “. . . enhanced accessibility, readability, and connectivity without compromising the professional craft of historical narrative” (Thomas 2001, 419). Also, with the progress of technology, historical researchers are now able to incorporate more perspectives in their work. The spatial component of an historical event is unlocked for the historian by geographic information systems (GIS), and historical data can be placed in its spatial context.

Part Two: Historical Geographic Information Systems (HGIS)

Historical researchers have long recognized the connection between time and space (Darby 1953; Knoerl 1991; Earle 1992; Bailey 2009). However, many historical researchers did not pursue the use of spatial analysis in the past because of difficulties and basic inabilities to incorporate spatial elements effectively (Amsden 1979; Ell and Gregory 2001). In the 1960s and the 1970s, more historical researchers began incorporating forms of basic spatial analysis into their work (Lampard 1961, 1965; Howard 1976). These researchers generally collaborated with geographers and statisticians to overcome the complex mathematical formulas and computations which were required to produce basic results. Also, it was generally difficult to visually display the results of research. Before the technology of computer cartography was easily
accessible, mapmaking and graphing often proved to be a resource-intensive process. More historical research projects with basic spatial analytical methods were undertaken by geographers in the sub-discipline of historical geography, which is based on inquiry into past space (Pitcher 1973; Williams 1974; Earle 1992; Prince 1997). However, until the past decade, new methods and ways to combine geography and history, like GIS, have not been widely adopted by researchers studying the past.

In the discipline of history, as in any of the social sciences and humanities, some researchers are wary of new methods (Gregory 2002; Fotheringham, Brunsdon, and Charlton 2004). This is, in part, due to the fine boundaries that may exist between disciplines. Also, many historians practice traditional research methods and, new methods, especially ones driven by technology, are viewed as less-than-rigorous shortcuts which denigrate the value of the results and, consequently, the discipline (Boonstra 2009). Historians have offered little formal criticism of GIS probably because HGIS scholarship is rarely presented in strictly historical publications and forums. Multidisciplinary audiences appreciate the scholarship produced with GIS, while historians may overlook it (Knowles and Hillier 2008; Boonstra 2009).

Past studies by historians which incorporate or attempt to incorporate spatial dimensions into the research are small in number. However, the proliferation of GIS technology is a positive step forward in increasing the abilities of historical researchers. The capabilities of GIS enable historical researchers to more readily incorporate the spatial dimensions into their work and to renew the real-world spatial relationships that exist in their topic of study (Knowles and Hillier 2008). This advance can provide a more complete picture of the past, and it certainly provides a different perspective on the
past. Geographic information systems technology opens up a new range of methods and techniques which historians may choose to employ.

The ability researchers have to visualize spatial and temporal data and attributes in GIS is one of the primary benefits of this technology. Exploration of a dataset is simple and fast in a GIS, and new ideas and theories can develop through this type of study (Knoerl 1991). This process is often referred to as exploratory data analysis. Geographic information systems software, such as ArcGIS by ESRI, offers different types of data visualization methods, creating flexibility and operability for the approach chosen by the researcher. Some of these visualization methods include displaying attribute data across space and time, displaying real-world proximity of features, and restoring of three-dimensional elements to the study environment (Knoerl 1991; Owens 2007; Bailey 2009).

Researchers using GIS in historical studies have generally focused on one of three areas of study: economic (Bales 1991; Healey and Stamp 2000; Thomas and Ayers 2003; Knowles and Healey 2006; Fyfe and Holdsworth 2009; Atack et al. 2010), agricultural (O'Kelly 2007; McLeman et al. 2010; Towers 2010), or demographic (Lee 1996; Gregory 2000; Gregory and Ell 2005; DeBats 2008; Fyfe 2009; Gregory 2009). This pattern mirrors the early historical studies that used quantitative methods. These areas have the most readily available data and are the easiest with which to work (Furet 1971). Some studies have used GIS in historical research to test the validity and accuracy of conventional historical knowledge (Diamond and Bodenhamer 2001). This type of revisionist work is beneficial to the academic body of knowledge as a whole. In addition, historical research using GIS is expanding into other areas of study, such as religion.
(Diamond and Bodenhamer 2001), economics of disaster (Paskoff 2008), health (Orford 2002; Hinman, Blackburn, and Curtis 2006; Tuckel et al. 2006; Curtis, Mills, and Blackburn 2007; Curtis 2008), and politics (DeBats 2009; Alhasanat et al. 2010). This expansion may accelerate when simplified GIS software more suited for the integration of space and time becomes available to historical researchers.

The merging of spatial and temporal analytical components into one system has developed slowly in comparison to the exponential growth in computing power over the past thirty years. Software that does incorporate time analysis functions are often for real-time data, rather than historical datasets (Wilson et al. 2010). The future of GIS technology will likely see special applications or extensions of software, specifically tailored for social sciences and humanities researchers. Ideally, this software will increase the flexibility researchers have in the management and display of temporal layers (Padilla 2008). Spatio-temporal analysis techniques, such as SaTScan (Kulldorff 1997), will be incorporated into large packages. CrimeStat (Levine 2004) is one example of the compilation of analytical functions, but the (geo)graphic abilities are limited. Historical researchers need functions in software with the ability to manipulate spatial and temporal layers outside of a strictly linear timeline. The ability to compare different time periods with each other and identify cyclical patterns will also be important to historians (Fyfe 2009). Lastly, the abilities of GIS software to animate data with optional automated functions are lacking, but are important for understanding historical networks (Owens 2007).

Tracing the movement of a person, thing, or idea across space and time inevitably leads to mapping networks. Mapping historical networks can help researchers better
understand the variables influencing past events. Geographic information systems software is already being used to study historical networks (Owens 2007; Atack et al. 2010; Bill Lane Center for the American West 2011). Geographic information systems programs, like ArcGIS, have a limited capacity in conducting advanced analysis of these networks. The analytical tools for network analysis, included in software like ArcGIS, are designed for decision-based functions (e.g., the most efficient route for a delivery truck), rather than modeling functions (e.g., probability of disease transmission routes). Therefore, researchers studying historical networks have often developed their own analysis software to supplement the GIS analysis (Owens 2007; Weaver et al. 2007; Fyfe 2009; Bill Lane Center for the American West 2011). Temporal analysis tools tailored to work in conjunction with GIS software can aid in the study of the past, especially for studying population mobility and activity space, urban disease diffusion patterns, commodity and resource flow, and information networks (Mayhew 2004; Findlen, Edelstein, and Coleman 2010; Bill Lane Center for the American West 2011).

Working to advance historical studies further, projects to develop interactive and reactionary simulation modeling programs (known as complex adaptive systems) are currently underway (Owens 2007). The goal is to recreate event actors and variables in a software program and study the interactions which created the historical outcome. This approach aims to combine space, time, objects, people, and networks to permit the exploration and study of a system or event. To create such a system, many techniques and methods are borrowed from multiple disciplines (Lansing 2003). The historical studies, in turn, can provide knowledge, insight, and feedback about these methods and topics.
The applicability of HGIS work to modern issues makes it more valuable to a larger audience. Historical datasets are valuable not just to scholars studying the past, but to researchers and policymakers working towards solving current problems. For example, in public health and epidemiology, historical datasets are used to calibrate disease surveillance and detection models (Fotheringham, Brunsdon, and Charlton 2004; Bouden, Moulin, and Gosselin 2008). Additionally, researchers can use studies of the past to build profiles for types of diseases in different environments. Historical research can provide more information and data to help solve current problems and mysteries. Quantitative and increasingly HGIS scholarship are proving invaluable to planners, administrators, and officials from different fields (Jacquez et al. 2005; Hinman, Blackburn, and Curtis 2006; Curtis, Mills, and Blackburn 2007; Xing et al. 2009).

Just as historians’ ability to understand the past first expanded through the use of quantitative analysis, it is now expanding through the application of spatial analysis and GIS. Improvements in merging time with space in GIS will lead to even greater abilities of researchers to examine the past. As in history, GIS are aiding researchers in medical geography.

Section II: Medical Geography

Part One: The Sub-discipline

Health is more than an absence of disease; it is the well-being of a person physically, socially, and emotionally (Cromley and McLafferty 2002). These modalities are innately intertwined with a spatial dimension as well. People have long recognized the existence of a spatial dimension in disease (Meade, Florin, and Gesler 1988). The sub-discipline of medical geography is based on this interconnectedness of health and
space. Medical geography has been described as containing approximately six to eight distinct, but interrelated approaches of study (Pyle 1977; Paul 1985). These approaches range from health-care geography to disease ecology, to disease mapping and diffusion (Paul 1985). Presently, an increasing number of researchers are able to incorporate multiple, interrelated approaches to the study of health (Paul 1985; Cromley and McLafferty 2002; Trevelyan, Smallman-Raynor, and Cliff 2005; Bouden, Moulin, and Gosselin 2008).

Researchers first used computers for performing mathematical and statistical analyses of disease characteristics, modeling disease spread, and basic spatial analysis functions (Cliff 1981; Mielke et al. 1984; Duncan, Scott, and Duncan 1993; Wilson 1993). Advances in technology have paved the way for great progress in geographic medical research. Geographic information systems are transforming the way disease is studied.

Technologies like GIS greatly increase the capacity to work with large, epidemic datasets in a spatial environment. Geographic information systems allow for the import, rectification, management, manipulation (i.e., editing), analysis, and presentation of large datasets. Researchers utilizing GIS can greatly enhance their study of the spatial relationships of disease. Data containing a unique spatial identifier or reference can be placed in a real-world coordinate system (Tuckel et al. 2006). Almost all spatial analysis techniques are based on the ability to measure distance between two or more features. With health data accurately placed in a real-world coordinate system, this is possible. Health hazards can be modeled in a GIS to investigate and examine temporal and spatial patterns (Cromley and McLafferty 2002). Medical geographers have used GIS to
facilitate the search for correlations and possibly causations between health and environmental factors (Kulldorff, Feuer, and Miller 1997; Jacquez et al. 2006). Geographic information systems are currently being used in medical geography and public health to examine infectious diseases and further investigate environmental factors which are related to health problems.

Use of GIS in research by public health professionals and medical geographers is increasing (Chang et al. 2009). In recent years, medical geographers and public health researchers have embraced GIS and other spatial/temporal analysis packages (e.g., SaTScan (Kulldorff 1997; Savory et al. 2010); Knox Test (Knox 1964; Aldstadt 2007); comparison of spatial analysis methods and techniques (Ball et al. 2008)). The analytical power of GIS easily surpasses the capabilities prior researchers had for studying the spatiality of disease (Cromley and McLafferty 2002; Fotheringham, Brunsdon, and Charlton 2004). Medical geography research has explored a variety of health topics, but accurate and efficient disease surveillance and monitoring systems have become a major area of study for researchers working in the applied field (Savory et al. 2010). Geographic information systems technology unlocks a world of potential in historical health records and is important for better understanding present disease through past epidemics.

Quantitative epidemiological studies often rely on health records and reports (Cliff, Haggett, and Smallman-Raynor 1998). In the 1800s, medical and public health professionals began keeping detailed vital records (Anderton and Leonard 2004). They also began to realize the value of keeping detailed public health records during epidemics. By the late 1870s, there was a strong movement in the United States medical
community to standardize recordkeeping during epidemics (Harris 1880). Public health agencies and organizations began publishing official reports containing mortality records from epidemics. In these epidemic records, spatial locators were frequently among the attributes recorded. This spatial component has allowed medical geographers to examine the spatiality of past epidemics (Smallman-Raynor and Cliff 2001; Trevelyan, Smallman-Raynor, and Cliff 2005; Hinman, Blackburn, and Curtis 2006; Curtis, Mills, and Blackburn 2007). Epidemic datasets have often been incorporated into GIS for spatial analysis.

Geographic information systems, built with historical epidemic datasets, have been used to study the 1887–1888 smallpox epidemic in the United Kingdom (Cain 2004), eighteenth century epidemics in Martigues, France (Seguy et al. 2005), the 1916 poliomyelitis (“polio”) epidemic in the northeastern United States (Trevelyan, Smallman-Raynor, and Cliff 2005), early-twentieth century typhoid outbreaks in Washington, D.C. (Hinman, Blackburn, and Curtis 2006), the 1918 Spanish Influenza pandemic (Tuckel et al. 2006), 1896–1906 bubonic plague epidemics in India (Yu and Christakos 2006), late-nineteenth century cholera epidemics in Japan (Kuo and Fukui 2007), the 1889–1890 Russian Influenza epidemic in Sweden (Skog, Hauska, and Linde 2008), and multiple epidemics in the Ming and Qing dynasties in China (Cheng, Li, and Yang 2009).

Studying major historical epidemics with GIS has built new knowledge and understanding of history, disease, people, and place.

Part Two: Mapping Yellow Fever

The mapping of any disease, including yellow fever, is based on the contemporary medical understanding of that disease. This medical knowledge directly influences the
methodology and the interpretation of the results. Throughout the eighteenth and nineteenth centuries, medical professionals debated many important issues regarding yellow fever (Hillary 1759; Keating 1879). Among these were the following unanswered questions: Is yellow fever contagious? Is yellow fever imported or indigenous? How is the disease transmitted? What causes the disease (miasma, germ, or another mode)? Yellow fever maps created at the time of the epidemic often reflected the researcher’s position on these questions (Shannon 1981; Paul 1985).

By the end of the eighteenth century, some physicians started integrating spatial notions of disease into their research practices. For example, in 1792, Leonhard Finke created an unpublished supplemental map showing the disease regions of the world (Finke 1792; Koch 2005). This is one of the first known worldwide disease maps, but it has since been lost to posterity (Barrett 2000). At the same time, other disease mapping techniques were being pioneered. Publishing his findings in 1798, Valentine Seaman mapped the locations of yellow fever cases (from the 1795 epidemic) near a harbor in New York City (see Appendix C, Figure 31)(Seaman 1798; Paul 1985; Koch 2005). He used a new mapping technique (the spot map) in an attempt to prove the miasma theory (Koch 2009). The subfield of medical geography traces its modern roots to these studies. Maps are useful tools for visualizing disease outbreaks (Stevenson 1965; Cliff 1981).

With increases in the understanding of disease transmission in the mid-nineteenth century, in part due to the development of germ theory, disease mapping became a mainstream epidemiological methodology (Paul 1985; Cliff, Hagget, and Smallman-Raynor 1998). Yellow fever was one of the first diseases mapped, but disease mapping proved much more fruitful with cholera (Paul 1985). Cholera outbreaks in European
Cities were mapped throughout the 1830s and 1840s, but John Snow’s 1854 map of the cholera outbreak in a London neighborhood is the most prominent map from this early era of medical geography. Snow used this map to support his theory on the source of cholera in one neighborhood (Broad Street pump in Soho) (Koch 2009). Snow, with his map, successfully supported his theory on the source of cholera, but he also demonstrated the great power of maps for studying disease. After success in mapping cholera, medical researchers applied the same methodology to a variety of other diseases. William T. Sedgwick mapped typhoid fever in Pennsylvania towns. Smallpox, measles, and plague were some of the other diseases mapped during this time (Koch 2005). In the mid-nineteenth century, large-scale maps of yellow fever, unlike cholera, did not prove successful in identifying the cause or source of the disease.

Yellow fever has been the focus of many studies since its first appearance. The transmission mechanics of yellow fever were unknown throughout the nineteenth century. Carlos Finlay published a paper in 1881 which proposed the mosquito as the vector of yellow fever (Reed et al. 1900). He also uncovered the species of mosquito capable of transmission, but little attention was given to his work until the time of Walter Reed’s research (Reed et al. 1900). In 1898, Henry Carter studied the infection and transmission timeline for yellow fever (Carter 1900). By accurately detailing the timeline of the spread of yellow fever, Carter found an extrinsic incubation period (Carter 1901). Through a combination of the works by Finlay and Carter, Walter Reed successfully proved the vectored spread of yellow fever by the *Aedes aegypti* mosquito (Reed et al. 1900). Subsequent to these finding, *A. aegypti* has been the subject of immense amounts of research (Christophers 1960; Soper 1967; Gubler 1989; Scott et al. 1993; Reiter et al. 1999).

Early maps of yellow fever showed individual cases throughout city districts (see Appendix C, Figure 31) (Seaman 1798). These did little to advance the contemporary understanding of yellow fever transmission. Regional yellow fever maps proved much more useful during the mid-nineteenth century. In 1856, Alexander K. Johnston published an atlas which contained a map showing the distribution of diseases worldwide. An inset map showed the yellow fever districts in North America (see Appendix C, Figure 32) (Johnston 1856). Maps were used as a type of public travel advisory to show regions of yellow fever in the Western hemisphere. Larger scale maps of yellow fever often showed infected districts in cities (e.g., Appendix C, Figure 33. “The Fever Lines of Memphis”). However, since the transmission mechanics of yellow fever were unknown until around 1900, physicians and other public health officials did not use these maps to decisively stop the spread of the disease. The last major yellow fever outbreak in the United States occurred only a few years after the mosquito was definitively proven to be the vector of yellow fever. The 1905 outbreak of yellow fever in New Orleans (see Appendix C, Figure 35) was halted by mosquito eradication efforts. Researchers began mapping mosquito populations at this time (See Appendix C, Figure 34). Maps locating mosquito populations showed the direct relationship between mosquitoes and areas of disease incidence (Koch 2005). Though analog disease mapping can show basic spatial relationships and patterns, analytical options are limited. On a
paper map of Memphis, Nazor (1992) located the approximate residences of yellow fever victims for the 1878 epidemic. Beyond the overall distribution of cases, not much else is apparent (see Appendix C, Figure 36). However, with modern computing technology, advanced spatial analysis can reveal even the most subtle spatial traits of a disease.

In the summer and fall of 1878, the Mississippi River Valley experienced the worst yellow fever epidemic in the history of the United States (Bloom 1993). While large yellow fever outbreaks afflicted Southern states in 1853, 1867, and 1873, these episodes paled in comparison to the 1878 epidemic. An estimated 50,000 people died from yellow fever in about a four month period, from July to October (Carter 1931). Large numbers of deaths occurred in New Orleans, Vicksburg, and Memphis. Out of these cities Memphis was the hardest hit by yellow fever. When yellow fever struck Memphis, out of a population of 50,000, approximately 30,000 citizens fled within the initial two weeks of the first confirmed local case of the virus (Hicks 1964; Baker 1968). Yellow fever spread quickly in Memphis, which caused chaos throughout the city. With 20,000 citizens remaining in the city, an estimated 19,600 contracted yellow fever and 5,150 of these cases proved fatal (Keating 1879).

Few studies of the 1878 yellow fever epidemic have utilized GIS. Curtis, Mills, and Blackburn (2007) used GIS to examine this epidemic in New Orleans. Their project employed a string of queries to identify possible index cases of yellow fever in the city. In a later project, Curtis (2008) used GIS to study the clustering of deaths at different scales and along cultural lines in the same epidemic. Arden (2005) examined the 1878 yellow fever epidemic in Memphis. However, his study was largely based on a single source (i.e., Keating 1879); no other historical sources were utilized. Arden (2005)
assumed his dataset (which contained all locatable deaths in Memphis) was a random sampling of the epidemic, spatially and temporally. His study used a variety of spatial analysis techniques to explore the GIS, though with a more rigorous historical approach, not only could the GIS benefit, but the understanding gained from the results could have been greater. Historical understanding relies on perspective; perspective is based on the sources.

This study reconstructs the 1878 yellow fever epidemic in Memphis, Tennessee with a large number of historical sources and resources. These documents, along with geographic and medical sources, are used to inform this study, create the GIS, interpret the results, and situate the knowledge. The combination of rigorous historical, epidemiological, and geographical methods and techniques is the basis for this study.
CHAPTER THREE

METHODS

Sources

More than 130 years have passed since the events of the 1878 yellow fever epidemic unfolded in Memphis. To study the spatiality of this epidemic, historical sources were used to reconstruct the circumstances in Memphis that year. The historical nature of this study demanded a reliance on a number of these materials, the accuracy, detail, and extent of which required evaluation. The geographic information system created for this study was made possible by three groups of sources. Identifying and obtaining the sources which existed in each group was a key step in the research process. The first set of sources was the medical records from the 1878 Memphis yellow fever epidemic. The contemporaneous maps of Memphis made up the second set of sources. The third key component to this study was the group of spatial reference sources. These sources provided the foundation of the GIS, but without the primary textual sources, this study would have been incomplete. Of these sources, some were readily available, while others proved more difficult to obtain.

This thesis traces its roots to the New Orleans yellow fever studies by Curtis, Mills, and Blackburn (2007) and Curtis (2008). The latter article provided a lead for the 1878 yellow fever epidemic in Memphis. Cited in Curtis’s (2008) article, the first source this thesis started with was John Keating’s (1879) *The Yellow Fever Epidemic of 1878 in Memphis, Tenn.* Keating, having been in Memphis during the 1878 epidemic, wrote a
broad ranging and detailed account of the event. To reconstruct the 1878 spatial progression of yellow fever in Memphis, mortality records were crucial. Keating’s (1879) book includes an index of yellow fever mortalities in Memphis. This index contains the names of over 5,000 individuals who died from the virus, the deceased’s residential address, the skin color of the deceased, and the date of each person’s death. Biases and flaws are inherent in all historical sources and Keating’s mortality index is no exception. To assess the quality of the index, more sources were required. Additionally, to better understand this event, the historical setting and human environment needed to be grasped.

The section of *The Yellow Fever Epidemic of 1878 in Memphis, Tenn.* titled “Incidents of the Epidemic” is Keating’s account of the epidemic, along with a compilation of newspaper stories from during the event. Among the newspaper stories included, the majority were from the *Avalanche* and the *Appeal* (Gallaway and Pollard 1878; Keating 1878). Both newspapers reported on the epidemic, and so they were important sources for this study. The Tennessee State Archives provided a microfilm collection of yellow fever related material, which included newspaper clippings from both presses (Tennessee State Archives). Also, a trip to the Memphis Public Library bore a plethora of sources, including microfilm for the full *Appeal* newspaper archive. Issues from both newspapers contained daily lists of yellow fever victims. Additional sources of the death records were found.

The Memphis and Shelby County Room at the Memphis Public Library has a substantial yellow fever collection. The yellow fever victim burial records from the Calvary Catholic Cemetery (Jones and Rennie 2003) are a part of this collection, and
once located, they were scanned into a digital format. The collection also has work by local genealogist Louise Nazor. She compiled, from a variety of sources, a large list of victims of the 1878 epidemic. A visit to Elmwood Cemetery, the main cemetery of Memphis in 1878, achieved copies of their burial records from that year (Elmwood Cemetery 1878). Official county death records from the epidemic were obtained from the Office of the Shelby County Register of Deeds (Shelby County Register 1878). This office also provided a variety of data, such as the Shelby County birth records, the Shelby County marriage records, the 1878 mayor’s relief distribution accounting records, the city directories for the 1870s, and the probate records of Shelby County. The yellow fever collection of the library at the University of Memphis contained a number of valuable human sources, such as personal letters and diaries. This collection also had rare sources, such as Dr. Dromgoole’s (1879) book on the epidemic. Memphis Heritage, Inc., a historic preservation group, had rich architectural sources, some of which will be useful in future research.

The original plan for these extra sources was either to corroborate or correct the Keating index. Along with quality assurance, they eventually played an even larger role in supplementing the index with more attribute information. While there were some errors found in the index, Keating’s complete list of the dead proved to be a good starting point for the mortality database. This source was assessed through the comparison with other sources, primary and secondary alike. Sources referenced or utilized included digital copies of the original vital records of the municipality (Shelby County Register 1878), burial records from two of the prominent cemeteries in Memphis (Elmwood Cemetery 1878; Jones and Rennie 2003), other contemporaneous books and reports.
containing substantial lists of individuals that died (Dromgoole 1879; Knights of Honor 1879; Murtough 1879), names of the dead published in local newspapers during the epidemic (Gallaway and Pollard 1878; Keating 1878), and records compiled by a local genealogist (Nazor 1992). This study benefited from access to a variety of sources.

In particular, the vital records of the city of Memphis added multiple new layers of information to the master dataset. While Keating’s death list has four attributes per record, the “Register of Deaths in the City of Memphis” contains ten additional attributes for each victim. Along with the name, date of death, skin color, and address, the Register has the following attributes for each victim: age, sex, marital status, citizenship, nativity, occupation, cause of death, physician in attendance, sexton, and city ward number. Age, nativity, and cause of death will play key roles in future studies (Anderton and Leonard 2004; Curtis 2008). The name, race, date of death, and address were used to verify the Keating records. Inconsistencies between the sources were further investigated in city directories, newspaper accounts, marriage certificates, and birth records. If no probabilistic solution could be found, the Register of Deaths record was trusted. The reasoning behind this was that while Keating’s list has a substantially greater number of records, the Register of Death files are official government records. They are potentially more accurate than the Keating index because of a conservative recordkeeping policy, and they contain more detail on each individual. The Keating index was used as the dataset foundation because of the size of the set. It was easier to remove records, than it was to figure out which records were absent. However, as is common in HGIS investigations, the dataset used for analysis represents a sample rather than a complete listing of all cases.
Without an individual’s residential address (or an alternate spatial reference), their death could not be studied in a discrete spatial context. To fill in missing address information in the Keating index, Memphis city directories were used. These proved invaluable as a source for creating spatial profiles of individuals. With the relative spatial locator, maps were needed to geographically place the event.

Contemporaneous maps of Memphis were a challenge to identify and obtain. Many map collections and libraries were consulted. Among the map collections consulted were the Geography and Map Reading Room at the U.S. Library of Congress, the Memphis and Shelby County Room at the Memphis Public Library, the Special Collections at the University of Memphis, the Cartographic Information Center at Louisiana State University, the map library at California State University: Long Beach, and the Office of the Shelby County Register of Deeds. The Library of Congress provided the 1888 Sanborn maps and two perspective drawings of the city (1870 and 1887). The Shelby County Register’s office provided a second set of the 1888 Sanborn maps, along with the 1897 Sanborn map-set. Also, among the maps provided were digital copies of Nazor’s yellow fever map (Appendix C, Figure 36), a city annexation map, and an 1886 paving map of the city. The Memphis and Shelby County Room had a large collection of historical maps of the city. Many maps were obtained using a scanner to digitize the hardcopies. Three maps were the most relevant to this study, being from 1872, 1878, and 1881. The 1872 and 1881 maps were of Memphis, but the 1878 map was of the extent of the yellow fever epidemic in the United States. Other maps were found in various locations on the Internet through web searches. The 1875 map and the 1880 sewer/topographical map of Memphis were on a variety of websites, but for this
study the maps were downloaded from, respectively, the Memphis Memories website and
the University of Texas at Austin library website (Berryhill 2008; UT Austin 2011). The
only map of Memphis found to be published between 1877 and 1879 came from an 1878
edition of the New York Herald newspaper (Appendix C, Figure 33)(Bennett 1878).
However, the base layers for this map were recognized as being an 1872 map (Murray
1872). The aforementioned 1875 and 1881 maps had a large enough scale to be useful in
corroborating and referencing the high quality Sanborn maps.

Maps from the Sanborn Fire Insurance Company were used as a main reference
layer in this study. Sanborn maps have proven spatially accurate, and the maps were
drawn to a large scale, which allowed the inclusion of a high level of detail (Curtis 2008;
Logan et al. 2011). Though spatially accurate, the first Sanborn map of Memphis was not
drawn until 1888. It focuses on Memphis proper, the area inside the original city limits
near the river (for the city boundary, see Appendix B, Figure 27). Sheets from the 1897
Sanborn map-set were used to fill in peripheral areas (Sanborn 1888, 1897). Smaller-
scale maps from 1875 (Williamson 1875) and 1881 (Sholes 1881) were used to assess
and identify the changes in the city in the 1880s and 1890s (as reflected in the 1888 and
1897 Sanborn maps). Major changes in the street network did not occur (Walker 1898).
To further ensure accuracy and evaluate the Sanborn maps, the 1878 Sholes’ City
Directory was used to verify that the address ranges and street names remained static
(Sholes 1878). The Sholes’ directory was a source for city ward boundaries as well.
With a large set of city directories published around the time of the 1878 epidemic, many
names, businesses, and streets were cross-referenced in multiple sources.
Methods

Database and GIS creation are the two most resource-consuming stages of HGIS projects (Bernhardsen 1999; Knowles 2000; Gregory and Healey 2007). This project was not an exception. First, the tabular data was inputted. Each individual mortality record, from August through November of 1878, was entered into a Microsoft Excel spreadsheet. These sources consisted of more than 5,000 records with approximately ten attributes for each record. Some of the attributes included in the spreadsheet are: unique record identifier; first name; last name; occupation; date of mortality; race; age; address number; address street; place of death; relatives.

Second, the historic maps were georeferenced. The small-scale reference maps from 1875 and 1881 were georeferenced with the U.S. Census 2000 TIGER street files. The street network in the First through Fourth Wards was similar to the modern network and resulted in reasonably accurate georectified maps. The large-scale Sanborn Fire Insurance maps from 1888 and 1897 were georeferenced with the 1881 map. Because the 1881 map was drawn to a larger scale and had a higher resolution than the 1875 map, it was the first choice when georeferencing the Sanborn maps. The TIGER files and the 1875 map were referred to when the Sanborn maps and the 1881 map did not match up properly. This was rarely necessary. In total, over sixty-five Sanborn maps were georeferenced to cover the entire study area.

Third, addresses of fever victims were digitized. Each mortality address was heads-up digitized based off the addresses on the georeferenced Sanborn maps. The digitized address points were given an identification attribute that linked each one with the corresponding mortality record. The mortality records were joined to the address
points based on the identification attribute. This created the Yellow Fever Victims (YFV) feature class.

Important geographic and civil features were digitized from the historical maps. These included the historical street network, the railroad lines, the hydrological features, the ward boundaries, and municipal buildings. These feature classes, coupled with the YFV feature class, formed the foundation of the historical geographic information system created for this study.

Analysis

Multiple factors influenced the decision to use a spatial sample for this study. The consistency of locatable addresses for mortality records decreased as the epidemic moved further east from the Mississippi River and the city core. This was due to decreased accuracy in recording deaths on the outskirts of town. Also, because of the time between the epidemic in 1878 and the creation of the Sanborn maps in 1897 for the eastern parts of Memphis, changes were more likely. The epidemic started well within Memphis proper and as the disease spread outward, the mortality rates increased. This led to less consistency in the reporting of deaths, beginning in early September (Hicks 1964). A contemporary physician said, “The rapid spread and malignity of the disease soon ran ahead of the records, and beyond the death list, nothing is certain” (Hardenstein 1879, 22). These issues were instrumental in the selection of the neighborhoods by the river for this study. Not only was the dataset more complete for this area, but the area also contained a large portion of the yellow fever cases during the beginning weeks of the epidemic (August and early September).
The map from the September 13th front page of the *New York Herald*, which showed the area of greatest mortality, was used as a guide to set the outer boundaries of the study area (see Appendix C, Figure 33) (Bennett 1878). The area was modified to a rectangle, which became the basis of the sample area. This was done to create a focused study area and uniform the distortion (due to edge effects) in the kernel density analysis. The outer edge is essentially a buffer area designed to decrease analytical distortion in the areas important to the start of the epidemic (Gatrell et al. 1996). With the study area defined, three types of basic spatial analyses were performed on the YFV feature class.

First, basic analyses computed on the dataset assessed the proximity of the disease events. These are common functions for determining the distribution of point data (McGrew and Monroe 2009). Clustering was assessed by calculating maximum, minimum and average Euclidean distances to the nth event for the dataset. This was used as a quick analysis of the proximity of multiple death locations (Jacquez 1996). It provided raw measurements of mortality proximity, which were useful in supplementing the mainly graphical results of the Kernel Density analysis.

Kernel Density (KD) analysis is useful for generalizing point data to create estimated values of intensity across space, and it is commonly used in disease mapping (Cromley and McLafferty 2002). KD analysis is a non-parametric statistic which measures the number of events which occur in a user-defined search window as the window moves across a defined space. The number of events located within the search window is divided by the area of the search window to provide a normalized estimate of intensity for that area (Fotheringham, Brunsdon, and Charlton 2004). This calculation is either performed at each event location or at the centroid of each cell in a raster layer.
The results are returned in a raster (a continuous surface) containing intensity values. A Universal Transverse Mercator (UTM) projection was used to avoid distortion caused by the curvature in a geographic projection. KD surfaces can effectively illustrate the variations between the disease (mortality) intensities of different areas (Cromley and McLafferty 2002).

This study used a KD analysis across time to identify areas of high mortality intensity. A weighted field was not used for the KD analysis, so the results were strictly based on the geospatial proximity of the mortalities. Bandwidths of 50-meters and 200-meters were used to calculate KD surfaces. These parameters were designed to reveal specific and general areas of mortality intensity during the epidemic (Cromley and McLafferty 2002). Fifty meters is considered the average maximum flight range of \textit{Aëdes aegypti} in an urban environment (Christophers 1960). Two hundred meters was the equivalent of two Memphis city blocks (each divided down the middle by an alley) in 1878. The 200-meter KD analysis generalized the point locations to visualize areas of high mortality intensity. This approach primarily served to estimate the intensity of death taking into account the deaths in adjacent city blocks. This is designed to limit the influence of error from unreported and/or misreported deaths while providing a rough estimate across time. Additionally, the 200-meter KD analysis visualized the general spatial movement of yellow fever through the study area.

The temporal parameters of the KD analysis were designed on a sliding-baseline method (Xing et al. 2009). The sliding-baseline method created a relatively smooth transition from one time period to the next, avoiding drastic changes in KD values due to a major shift in the timeframe. The epidemic was divided into thirteen partially
overlapping periods. KD analysis was run on each of the thirteen periods of the epidemic. The first KD raster showed the intensity of deaths from the first to third weeks of the epidemic. The second KD raster showed intensity from the second to fourth weeks. The time period shifted forward by one week, but retained two-thirds of the time from the previous period. This was done for thirteen weeks, ending at the official conclusion of the epidemic (August 1st–October 29th).

Every step of the research process is important for obtaining viable results. Medical, historical, cartographical, and geographical sources were the foundation of this research, and therefore they were of crucial importance. The process used to gather adequate sources was described. Methods for GIS creation were detailed. This chapter ended by reviewing the analyses and computations to which the data were subjected. In the following chapter, the results of this study are presented.
CHAPTER 4
RESULTS

Introduction and Overview of Symbology

Each yellow fever death is symbolized by a point on the map. Each dot is situated on the map based on the associated victim’s home address. The color of the dot indicates the stage during which an individual died. Three stages (seven days each) make up a period, which is twenty-one days. Deaths occurring in Stage 1 (the first seven days of the period) are symbolized by red dots (e.g., Stage 1 of Period II is August 7th–13th). Orange dots indicate yellow fever deaths during Stage 2: the middle seven days (e.g., Stage 2 of Period II is August 14th–20th). Yellow dots indicate deaths which occurred during Stage 3: the last seven days of the time period (e.g., Stage 3 of Period II is August 21st–27th).

Each twenty-one day time period, by design, overlaps the previous time period by fourteen days. This creates a gradual transition through the epidemic timeline and links the study periods together. Using this sliding baseline method helps reduce errors stemming from mutually exclusive temporal groupings.

Additionally, each map has a raster layer which is the result of a kernel density analysis of the mortality locations of the period. A 200-meter search radius was used for calculating the intensity of mortality across the area. The intensity classification is based on equal intervals of 5 (deaths). Values of 0 (0 deaths within 200 meters) are considered null, which excludes these areas from the symbology. The intensity range is from Level 1 to Level 12, with 1 as the lowest intensity (5 or less deaths within 200 meters) and 12
the highest intensity of death (greater than 55 deaths within 200 meters). The equal interval range was chosen to methodically show the number of deaths (within five) in each area.

![Map Legend](image)

FIGURE 1. Map legend for the maps in Chapter Four.

Each map has a reference grid (Columns A, B, C; Rows 1–6). This grid is designed to help orient the reader. For example, A,1 directs the reader to Column A, Row 1. Hyphenations are used to reference large locations; for example, A–B,1–2 directs the reader to Columns A and B, Rows 1 and 2. A forward slash references the intersection between grid guides (e.g., A/B, 1/2 directs the reader to the intersection of Columns A and B, intersection of Rows 1 and 2). Along with the reference grid, zones are used to designate important parts of the study area. These zones were created based on the areas of high intensity shown to have existed at different times in the epidemic by the 200-meter kernel density analysis. Figure 5 shows the locations of these zones.
FIGURE 2. Number of yellow fever deaths in the study area for each period of the epidemic.

FIGURE 3. Number of yellow fever deaths in each zone (see Figure 5 for zone locations).
FIGURE 4. The daily total of yellow fever deaths in the Memphis Study Area.
FIGURE 5. Reference map for the study area showing water features (light blue), streets (black), wards (green), yellow fever zones (red), and proposed start locations of the epidemic (blue). Location A is the block of Kate Bionda’s residence at 212 Front. Location B is the block of 277 Second, the locale in which the epidemic began, according to Keating (1879). Location C is the area of 177 Second, the locale in which this study showed the epidemic to have begun (see Chapter Five for further discussion on this point). The black lines are streets and the gray lines are alleyways.
Spatial Patterns of the Epidemic


FIGURE 8. Period II histogram, August 7–27.

FIGURE 9. Period II map, August 7–27.

FIGURE 11. Period III map, August 14–September 3.


FIGURE 15. Period V map, August 28–September 17.

FIGURE 17. Period VI map, September 4–September 24.

FIGURE 19. Period VII map, September 11–October 1.
FIGURE 20. Period VIII histogram, September 18–October 8.

FIGURE 21. Period VIII map, September 18–October 8.
Period I: August 1–August 20

Period I deaths are shown in Figure 6 and Figure 7. The deaths in Period I primarily occurred in Stage 2 and Stage 3. All but two deaths in this period occurred between August 12th and August 20th. These dates are used as parameters for the following descriptive statistics. In 9 days a total of 72 deaths occurred. August 17th had the highest incidence with 18 deaths and August 19th had the smallest with 3. The average number of yellow fever deaths per day was 8.

The kernel density analysis identified three areas of moderate intensity of deaths during Period I. These areas were situated near Washington and Main (Zone 1), Alabama and Johnson Avenue (Zone 2), and Poplar and Bayou Gayoso (Zone 3). Areas with slightly raised levels of mortality intensity were located along Jefferson Avenue, west of Fourth \(B,4\), and the blocks between Front and Main, south of Commerce \(A,2\). Deaths appeared in the Fifth and Fourth Wards on August 19th and 20th, but the intensity of death remained low.

Period II: August 7–August 27

Period II deaths are shown in Figure 8 and Figure 9. All of the deaths in Period II occurred after August 10th. The first 5 days of minimal returns were excluded from the descriptive statistics. Over the remaining 16 days there were 166 deaths. The highest number of mortalities in one day was 20, on August 27th, and the lowest number of mortalities was 3, on August 19th. There were, on average, 10.4 mortalities each day during this period.

High intensity areas of mortalities were located near Washington and Main (Zone 1), Alabama and Johnson Avenue (Zone 2), and Poplar and Bayou Gayoso (Zone 3). Of
these zones, Zone 2 had the most concentrated intensity of deaths. In Zone 2, the average distance of each death from the nearest neighboring death was 9 meters and 41 meters to the nearest five deaths. Zone 3 had the largest area of high intensity. The area had a Level 9 intensity (41–45 deaths within 200 meters) covering approximately 3450 m². Zone 3 was also the only locale to have deaths occur every day from August 12th to August 27th.

South of Jackson between Front and Main (Zone 4) was an area of moderate intensity. A fairly linear pattern of deaths ran between this zone and the high intensity area of Zone 1. Other areas of moderate intensity extended outward from Zone 3. A tight grouping of deaths was located on Market, east of Bayou Gayoso (B,2). In Stage 3, yellow fever deaths occurred south of Madison in the three following areas: on Madison between Front and Third (A,5); along DeSoto between Gayoso and Monroe (B, 5/6); and near the intersection of Monroe and Wellington (C,5). The three areas had slightly elevated levels of intensity, but more importantly, these new deaths were indicators of the emergence of yellow fever in the southern portion of the study area.

Period III: August 14–September 3

Period III deaths are shown in Figure 10 and Figure 11. Over the 21 days in Period III, a total of 417 deaths were recorded in the Study Area. Stage III alone accounted for 268 deaths. Period III had an average of 19.9 deaths per day. The overall increase in number of deaths during this period was relatively steady, but there were a few days which experienced noticeable jumps in the daily death totals. These occurred on August 17th, August 29th, and September 2nd. The range of deaths for the days in this period was 47. August 19th had the smallest number of deaths (3) and the greatest
number of deaths (50) occurred on September 2\textsuperscript{nd}. Throughout the 1878 epidemic, the peak of yellow fever deaths in the study area occurred on September 2\textsuperscript{nd} as well.

Four major zones of yellow fever mortality are clearly identifiable for the end of August and the beginning of September. In the Poplar/Bayou Gayoso area (Zone 3), the number of yellow fever deaths between August 28\textsuperscript{th} and September 4\textsuperscript{th} doubled the total number of yellow fever deaths in the area thus far in the epidemic. During this period, new yellow fever deaths north of Washington peaked at more than 190 recorded deaths. Zone 4 was the place where yellow fever prevalence from Zone 1 and Zone 2 collide. This prevalence and intensity of yellow fever spilled over Jackson and moved towards Chelsea. The gap between Zone 1 and Zone 3 was also closed during this period. The remaining susceptible population in the area was enveloped by yellow fever.

New deaths emerged in the Fifth Ward at Beale/DeSoto and Beale/Hernando (Zone 5, collectively). The intensity of deaths in Zone 5 increased during Period III. During the time span of nine to fourteen days after the early yellow fever death in this neighborhood on August 19\textsuperscript{th}, at least eight yellow fever deaths occurred within a 120 meter radius of that residence. Later incidences of yellow fever (deaths on the 22\textsuperscript{nd} and 23\textsuperscript{rd} of August) on DeSoto aided in creating an infect population of mosquitoes in the area.

**Period IV: August 21–September 10**

Period IV deaths are shown in Figure 12 and Figure 13. The number of daily yellow fever deaths leveled off after September 2\textsuperscript{nd}. Period IV’s range was 41 deaths. The lowest number of deaths during this period (8) happened on August 24\textsuperscript{th}. September 2\textsuperscript{nd}, with a count of 50 deaths, was again the deadliest day of the period. The total
number of deaths occurring in Period IV was 625; the average number of deaths per day was 29.8.

By September 11th, yellow fever deaths occurred within almost every block of the study area. The total number of new deaths in the section of the study area north of Washington decreased during Period IV. No new deaths were recorded in the original area of intensity at Alabama and Johnson Avenue (Zone 2). Near Poplar at Bayou Gayoso (Zone 3), the number of new deaths decreased. The combined Zone 1 and Zone 4 continued to experience large numbers of new deaths. With the Mississippi River on the west and the susceptible population already decimated on the east, yellow fever spread from Zone 1 to the south and Zone 4 to the north.

In Period IV, the section of the study area south of Washington surpassed the northern area in total number of new deaths. Near Second and Jefferson (Zone 6) there was an increase in the intensity of deaths, and a noticeable area of intensity started to emerge. Along Main, a corridor of death continued to intensify.

Zone 5 also emerged as an area of noteworthy intensity. The number of new yellow fever deaths in Zone 5 during this period was four times larger than the total number of prior deaths. Additionally, a grouping of deaths near Jefferson and Bayou Gayoso (C,4) emerged while the disease continued to spread east into portions of the Fifth, Seventh, and Eighth Wards. In Period III, a tight grouping of mortalities appeared on the edge of the study area near Monroe and Wellington (C,5). The first yellow fever death in this area was a lone case in Period I, but more deaths appeared in subsequent periods. In Period IV, yellow fever had spread outward from this area.
Period V: August 28–September 17

Period V deaths are shown in Figure 14 and Figure 15. Before September 13th, yellow fever deaths numbered between 30 and 50 a day. After September 13th, the daily number of deaths did not exceed 30 again throughout the remainder of the epidemic. The final day, September 17th, had the fewest number of yellow fever deaths (16) in Period V. A difference of 34 deaths separated this day from September 2nd, the day of the highest incidence (50 deaths). On average 33.3 deaths occurred each day. Total deaths occurring between August 28th and September 17th were 700.

Between September 11th and September 18th, new deaths covered a wide north-south range, with the majority of these having occurred to the west of Third. A large number of new deaths occurred between Bayou Gayoso and Bayou DeSoto (Zone 5). Period V was the last time in the epidemic that there were large numbers of new, wide-spread yellow fever deaths.

At the Washington/Bayou Gayoso area (Zone 3), a noticeable break in new yellow fever deaths occurred. Additionally, few new deaths were recorded in the area along Bayou Gayoso, just south of the aforementioned area. This likely indicates a substantial waning in the susceptible population in these areas.

The large area of high intensity shared by Zone 1 and Zone 4 (along Main, between Jackson and Washington) continued to expand south in Period V. This was the last period in which this large continuous area of high intensity existed. South of Zone 1, the number of deaths increased near Jefferson and Second (Zone 6). A large number of deaths occurred in very tight groups in Zone 6.
The yellow fever deaths in Zone 5 reached a high level of intensity during Period V. West of Zone 5, the emerging group of yellow fever deaths along Main, likewise increased in intensity, but to a lesser degree.

**Period VI: September 4–September 24**

Period VI deaths are shown in Figure 16 and Figure 17. Overall, Period VI showed a gradual but constant decline in the number of yellow fever deaths per day. Forty-five deaths occurred on September 4th, which was the highest for this period. September 23rd, the penultimate day of this period, had the lowest number of deaths (4). Over these 21 days, an average of 23.4 deaths per day occurred, although the bulk of the deaths occurred in the first half of the period. In total, 491 deaths occurred between September 4th and September 24th.

The number of new yellow fever deaths noticeably decreased from September 18th onward. The new deaths which did occur in Period VI were located around previous areas of intensity. The high intensity of death was limited to Zone 5 and Zone 6. By this point in the epidemic, not a block remained in the study area that had not been touched by yellow fever. The complete diffusion of yellow fever throughout the study area had annihilated the susceptible population. The intensity of the once-massive hotspots designated Zone 1 and Zone 4 had decreased dramatically. Only a very small number of new deaths occurred in these areas.

The Beale/DeSoto hotspot at Zone 5 shifted north during Period VI. New deaths continued to occur, but with less frequency and amplitude. The relatively new hotspot at Zone 6, near Jefferson and Second, was the primary location of new deaths in the study area. Most of these new deaths were scattered around the area of highest intensity. Zone
6 was more retail and business than the area above Washington; this explains the scattered and subsequently lower levels of mortality intensity.

Period VII: September 11–October 1

Period VII deaths are shown in Figure 18 and Figure 19. In Period VII, after September 17th the number of deaths by day dropped and remained below 15. This period averaged 13.3 deaths each day. The range of deaths was 29, with a high of 32 deaths occurring on both September 11th and 12th, and a low of 3 deaths occurring on September 25th. In the study area, a total of 280 deaths occurred during this period.

The continued decline in the intensity of mortality north of Washington is evident in Period VII. The other hotspots decreased in intensity as well. Few new deaths occurred in Zone 5, while the Beale/Main area experienced a small resurgence. A handful of new deaths occurred in Zone 6, but these deaths were loosely distributed. As September came to a close, yellow fever deaths continued to occur in the study area, but appeared only in small numbers.

Period VIII: September 18–October 8

Period VIII deaths are shown in Figure 20 and Figure 21. Deaths from yellow fever in Period VIII ranged from 3 to 13 deaths each day. The days in this period averaged 6.8 deaths. A total of 142 deaths occurred between September 18th and October 8th. The dramatic decrease in daily yellow fever incidence had begun to level off.

New deaths from yellow fever did not appear in close proximity. The last traces of spatially focused yellow fever were quickly fading. The intensity of deaths decreased further in Period VIII. Between September 30th and October 9th, no new deaths were recorded in the study area north of Winchester.
Finale: October

The remaining three weeks of the epidemic were marked by the small ebbs and flows of yellow fever in the study area (see Appendix B, Figure 30 for the histograms and maps for the remaining time periods in October). Widening the analysis to include Shelby County and possibly the entire Mississippi River Valley could better show the epidemic in October. By the end of September, yellow fever deaths in the study area had permanently dropped into the single-digits. By October, few yellow fever deaths were recorded for the study area, likely due to the absence of a susceptible population. During this time, however, yellow fever was continuing to wreak havoc in the suburbs and rural communities outside of Memphis. The virus traveled deep into the heart of the countryside by way of the railroad lines. Towards the end of October, low temperatures limited the activity of *A. aegypti* in Memphis and across the South. A small number of new yellow fever deaths in the study area were recorded between October 23rd and October 30th. On October 29th, the Howard Association officially announced the end of the epidemic (Hicks, 1964). Of the few yellow fever deaths after October 30th, over one half occurred at an address which had a prior yellow fever death late in the epidemic. These isolated cases of yellow fever were likely caused by infected resident mosquitoes. See Appendix A, Figure 26 for a histogram of the entire epidemic and Appendix C, Figure 29 for the full time-series of yellow fever maps.
CHAPTER 5
DISCUSSION AND CONCLUSION

Discussion

By integrating geography and history through historical geographic information systems, this study contributes new spatial knowledge of the 1878 yellow fever epidemic in Memphis, Tennessee. This critical examination assesses and utilizes primary sources for first-hand accounts of the epidemic, while it mines these sources for additional data as well. Combined, historical source analysis and geospatial techniques produce a new historical and geographical understanding and body of knowledge associated with the 1878 yellow fever epidemic.

Due to the rapid spread and onset of panic and confusion during the 1878 yellow fever epidemic in Memphis, an adequate single source of death records does not exist. Past research on the epidemic did not utilize all the available sources. Furthermore, new technology is now available for studying geospatial phenomenon. This study assessed, analyzed, and combined multiple mortality, cartographic, and historical sources into one GIS to reconstruct the epidemic in Memphis. Through Kernel Density analysis, a broad view of the diffusion of the disease was geographically represented.

The epidemic escalated in August of 1878. The northern section of the study area was inundated with yellow fever. An estimated ninety-nine percent of people in this initial area contracted yellow fever (Dromgoole 1879). During late-August and early-September the area could no longer contain the virus. All sense of boundaries vanished
and yellow fever swept towards the southern part of the study area and north towards Chelsea. Making matters worse, the Fourth, Fifth, and Seventh wards already had enclaves of people infected with yellow fever (likely from cross-town contact during the early stages of the epidemic (Hicks 1964)). The small areas had initially appeared unimportant, but the little pockets of yellow fever broke out in full force from their peripheral locations at the same time the virus exploded from the northern area. At this point, yellow fever had a large portion of the city encircled by infected populations of *A. aegypti*. The bulk of the yellow fever momentum had built up in the northern “Pinch” district, while the small pockets of yellow fever surrounding the study area intensified. The quick north-south burst of yellow fever engulfed the Third, Fourth and Fifth wards. The struggle by the Memphis Board of Health to contain yellow fever in the “Pinch” district was lost within the first week (August 14th–23rd), if not before. Survival became the main focus of the people living in the study area. The entire area was almost completely void of susceptible inhabitants by the middle of September.

The network diffusion of yellow fever progressed methodically in the northern part of the study area, but also jumped to enclaves in the south. This type of disease spread is known as mixed diffusion (Cromley and McLafferty 2002). On September 4th, the Board of Health burned barrels of tar and sulfur along Main in an attempt to decrease the disease incidence (Hicks 1964). The number of new deaths near Main had increased from 26 deaths August 21st–27th, to 64 deaths August 28th–September 3rd, to 71 deaths September 4th–10th. Additionally, the high intensity areas visible along Main (north of Washington) support the Board of Health’s understanding of the prevalence of yellow fever in this area. One explanation for this concentration of deaths along Main, was that
yellow fever spread along the Blue Line street railroad (Hicks 1964). This ran north-south on Main, between Fort Pickering and Chelsea.

Contrary to widespread belief at the time, yellow fever does not appear to have spread along Bayou Gayoso. The road networks, especially Main and Second, were much more important than Bayou Gayoso in spreading the disease north and south. Apart from these two streets, yellow fever appears to have been primarily spread along the roads perpendicular to the Mississippi River. The rapidity in the disease spread along certain roads indicates that these roads were major parts of the activity space and mobility network of the surrounding population.

While this study reveals a broad picture of the progression of the epidemic, it has uncovered a good deal of individual-level information. The beginning of the epidemic has been a frequent topic of study. Most sources acknowledge the first official yellow fever case, Kate Bionda (of 212 Front), who died on August 13\textsuperscript{th}, was almost certainly not the first actual case in Memphis. In John Keating’s book on the 1878 yellow fever epidemic in Memphis, often considered a definitive work on the event, he identified 279 Second as the address where the virus entered the city. Keating’s (1879, 107) description of the start of the epidemic follows:

It was ascertained, after the epidemic was fairly established, that many cases had occurred before her’s [sic] [Kate Bionda]. Mrs. C. W. Ferguson, boarding at the residence of Attorney-General G. P. M. Turner, 279 Second Street, states that on the 21\textsuperscript{st} of July a colored man came up the river, whose wife was cook for Mr. Turner. This woman had a residence in the yard back of the Turner house, and abutting on an alley which runs from Second to Main Street. Her husband had been taken with a severe chill on the boat on the morning of the day on which he landed, and when he reached his home had a very high fever for several days. For this his wife treated him with hot teas, and he recovered. Subsequently, and about ten days after his arrival, Mr. Turner’s two children were taken with well-marked cases of yellow fever. One of them died, and the other
recovered. In the meantime, a young man named Willie Darby, an employé of Farrell, the oyster-dealer, who lived at 277 Second Street, and who was in the habit of passing to his meals through the alley infected by the colored man, although he slept in the third story of his house, was taken with the fever, but recovered. He was nursed by his aunt, and was not visited by a doctor. His was the second case; it occurred on the 25th of July. The good woman who saved his life took the fever and died, as did nearly all who lived in the house or in the house near by. Mrs. Zack (white), who resided on the opposite side of the street, died of the fever on the 5th of August, and her brother-in-law, taken on the 10th, died on the 13th, the day before Mrs. Bionda died.

The 277 Second Street locale was examined in the GIS. No alley was found to run between Second and Main on the 277–288 block of Second. The adjacency of Court Square to the north caused the irregularity of no east-west alleyway in the block. This was confirmed after referring back to the original Sanborn maps and the “Guide to Streets and Avenues in Memphis and Suburbs” in the Sholes’ City Directory. Willie Darby, purportedly of 277 Second, did not have an alley through which to pass. The location identified by Keating is presented in Figure 22, along with the kernel density layer of yellow fever deaths for the first twenty days of August. No deaths were recorded in the immediate area. Only one death was located within 200 yards of the 277 Second Street block. This logic cast doubt on the description by Keating. Since the people listed in the description probably did exist and, if they themselves did not die, likely had family members die during the epidemic, the areas identified (through the kernel density analysis) as having had a high intensity of mortality early in the epidemic were the next places to search. Specifically, the area sought needed to have an alley between Second and Main Street, a similar grouping of surnames (Turner, Ferguson, Darby), and multiple deaths early in the epidemic.
FIGURE 22. A map showing the intensity of yellow fever deaths in the initial stages of the epidemic and the block of 279 Second Street (red circle), described by Keating (1879) as the location the epidemic began. The black lines are streets and the gray lines are alleyways.

When it became clear that 277 Second was probably not the epicenter of the epidemic, alternative addresses were considered. Because switching 177 to 277 seemed like a logical mistake, this was the first location investigated. This inquiry found that 177...
Second Street was one house over from an alleyway that connected Second and Main Street. As Figure 23 shows, the mortality intensity was high in this area during the beginning of the epidemic.

FIGURE 23. A map showing the intensity of yellow fever deaths in the initial stages, the underlying deaths (black), the block of 279 Second Street described by Keating (1879), and the 179 Second Street block with the associated cases (yellow).
While there were no victims with the surname Turner in this area, Mrs. Jenny Darby died at 177 Second and Harry W. Ferguson resided next door at 179 Second Street (he died in Camp Joe Williams). Mrs. Darby was likely the aunt of Willie Darby. Annie G. Ferguson was listed in the 1879 Sholes’ directory as the widow of C. W. Ferguson and living at 179 Second. She was likely the interviewee to which Keating referred. Along with Harry, four other Ferguson’s were listed in the 1878 Sholes’ directory as living at 179 Second. Besides the Ferguson’s and the Darby’s, the other names did not appear to match.

Keating’s notation “(white)” appeared to identify Mrs. Zack as a white woman. She lived on the opposite side of the street from either Darby or Ferguson and died from yellow fever. Her brother-in-law died on August 13th. At 181 Second, which was across the alley from the Ferguson house, a woman recorded as Mrs. Jennie White (age 25) died on the 12th of August from yellow fever. Perhaps Keating mistakenly took Mrs. Zack White to be Mrs. Zack, who was white. The 1878 Sholes’ directory lists a Z. T. White living at 181 Second Street. Additionally, Shelton M. White (age 27), who also resided at 181 Second, died on August 13th. Mrs. Jennie White was listed as married, and therefore was likely an in-law of Shelton White (who was listed as single). Shelton White died on the same day listed by Keating as the death of Mrs. Zack (white)’s brother-in-law. Figure 24 illustrates the aforementioned deaths.

In addition to not being an area of high mortality intensity at the beginning of the epidemic, the 277 block of Second Street does not appear, according to the 1878 Sholes’ directory, to have been residential. William Crosbie owned a saloon at 277 Second, but he lived at 237 DeSoto. Frederick Stillman, William Shuttleworth, and Edward Frederick
FIGURE 24. Names of victims around 177 Second Street. The order of deaths starts with Mrs. Jennie White and continues counterclockwise around the listed names. The names in bold are mentioned or related with Keating’s (1879) account.

owned businesses at 279 Second, and all lived elsewhere. Stillman was a kalciminer (whitewash painter) and lived on the west side of Manasses, two streets south of Marr. Shuttleworth was a painter and lived on the northwest corner of Manassas and Ohio. Frederick was a paperhanger and decorator and lived at 55 Orleans. All of these
residences were so far from the 277 block of Second that they were outside of the study area. At 281 Second, John Haberling, who resided at 296 Hernando, ran a stencils business. Nothing in the 277 block seems to match the description of the start of the epidemic. The most likely explanation is that Keating mistakenly switched the 277 block of Second Street for the correct 177 block. In the 1878 Sholes’ directory, G. M. P. Turner was listed as renting a room on the south side of Court Street, east of Third. This was close to the 277 block of Second. In tracking Turner’s residences throughout the 1870s and 1880s (using a range of city directories), he appears to have moved residences almost annually. He did seem to have a sustained work address at 166 Main for multiple years. A possible factor in the Keating error is that the Memphis Board of Health office was located at 266 Second. Since Keating did live in Memphis, it is quite possible that it was simply a transcription error.

Regardless of the cause of the error, Keating’s mistaken account has been spread by other works on the epidemic. Arden (2005) includes the entire Keating passage on the initial spread of the disease from the 277 block (Keating 1879, 107; Arden 2005, 32). While Arden does ask why the cases have been overlooked, he does not use the GIS he built to investigate the account. More recently, in her book *The American Plague*, Crosby (2007) propagates Keating’s account without question.

With GIS, this mistake was uncovered. After discovering this error, two sources were identified which were, at least partially, corroborated by the findings of this study. Dr. Tyner (1879) lists early cases of yellow fever at 179 Second Street, but because of many blaring mistakes in his account of the epidemic, little reliance can be placed on this source. Dr. Richard Maury, a prominent physician in Memphis at the time of the
epidemic, published a paper entitled “The Sanitary Necessities of Memphis and the Yellow Fever of 1878” in the *First Report of the State Board of Health of the State of Tennessee, April 1878–October 1880*. In this paper, Maury identified the same victims listed in the GIS database (see Figure 24) and confirmed that they lived in the 177–181 address range of Second Street (Maury 1881, 87–89). Maury’s conclusions on the locations of the initial foci of the epidemic are supported by the results from the kernel density analyses found in Chapter Four of this study. His basic spatial descriptions of the epidemic were the most comprehensive and accurate among all of the sources consulted for this study. Since Maury’s paper was published in a report by the Tennessee Board of Health, it was more difficult to discover. This may explain why past scholars did not recognize the inconsistencies between the accounts of the epidemic.

However, based on preliminary findings from research which will be part of a future study after this thesis, Maury was incorrect in another part of his paper. He asserted that the deaths in July and early August, which could have been misdiagnosed cases of yellow fever, did not have any spatial relationship to the subsequent foci of the epidemic. The deaths mapped out in Figure 25 were diagnosed as malarial fever, typhomalarial fever, hemorrhage in the lungs, congestive fever, congestive lungs, or congestion of the brain. The residences of some of these deaths appear to be located in close proximity to areas of substantial yellow fever deaths. More research is underway to examine these apparent spatial correlations.
FIGURE 25. Deaths from July (black) compared to the early areas of high intensity in August.

Conclusion

This study has contributed both to geography and history. Historians are given a framework for incorporating spatial components in their research. Additionally, this thesis details the processes of identifying and acquiring cartographic and spatial sources.
Geographic information systems have been shown to be a powerful tool for investigating spatial components of historical events. The main contribution of this thesis is the discovery and correction of the error in Keating’s (1879) account of the start of the epidemic. Also, other mistakes have been identified in commonly cited sources and will be further detailed in future work.

Keating’s (1879) book has been referenced and cited in a large majority of studies on nineteenth century yellow fever in the Southern United States. This error has never been addressed. The identification and especially the rectification of this error were only possible through the reconstruction and analysis of the city and yellow fever epidemic with GIS. Geographic information systems are a powerful tool which can greatly benefit historical research.

Collaborative research is best conducted when all researchers understand the fundamental methods in use. For historians, this means learning GIS software, understanding the geographic theories behind spatial analysis, and understanding the functions of these analytical techniques. This will situate historians in a position to conduct powerful historical research through an incorporation of more elements of the past.

Geographers who engage in HGIS would be similarly well-served by training in methods for conducting historical scholarship. Incorporating textual historical material can provide more capacity for reflective thought. The sheer fact that, if nothing else, humans have created the existing records of an historical event requires any researcher studying the past to engage in an analysis and assessment of the available sources. Just as GIS benefits historical research in providing an additional perspective on the topic, so
too should available historical sources be used by geographers to gain new perspectives on the subject. The historical research methods, source identification and collection processes, and historical repositories detailed in this thesis are a guide for geographers interested in studying the past.

Through this study it has become apparent that the obstacles in using GIS for historical research are mainly in the presentation of information. Tracing yellow fever in a spatial context, while maintaining the linear flow of time, is a challenge. In switching from a dynamic research environment (GIS) to a static presentation environment (paper), knowledge is not easily transferred. Vast quantities of rich spatial and visual data are created, and these often prove difficult or too extensive to show on paper. Paper also lacks the ability to show animations and three-dimensional virtual environments, which have proven to be some of the most effective tools for presenting this epidemic. These issues encourage supplemental websites and media to reinforce the findings of GIS aided historical studies.

The start of the yellow fever epidemic in Memphis will continue to be investigated using HGIS. This will build on the research presented in Figure 25. Weather data may be incorporated into the research as well. Also, the social and economic elements associated with the 1878 epidemic will be studied. The methods used by Curtis (2008) to examine yellow fever spread through cultural networks in New Orleans will be applied to Memphis, and then the results will be compared to New Orleans. Additionally, the epidemic’s persistence on the population and the city will be tracked. Consideration will be given to how the cultural, economic and civic structures of Memphis were altered by the epidemic.
APPENDICES
APPENDIX A

HISTOGRAM
FIGURE 26. Two histograms showing the yellow fever deaths by day in the study area compared to the deaths in the whole city.
APPENDIX B

SUPPLEMENTAL GIS MAPS
FIGURE 27. Reference map for the city of Memphis; City Wards are signified by red and the study area by yellow.
FIGURE 28. (same as Figure 1) The map legend for Figures 13 through 14. See Chapter Four for further explanation on the methodology, the map legend and the symbology.
FIGURE 29. A kernel density time series showing the spread of yellow fever in the study area.
FIGURE 30. Maps and histograms for Periods IX–Period XI, showing the frequency of deaths and the areas of mortality intensity for October.
APPENDIX C

HISTORICAL YELLOW FEVER MAPS
FIGURE 31. Valentine Seaman’s map of yellow fever cases in 1795 on the East River in New York City. The number on each building represents one case of yellow fever and the day the case occurred.
FIGURE 32. Johnston’s 1856 map of yellow fever in the Americas.
FIGURE 33. The 13 September 1878 front page of the New York Herald showing the areas in Memphis with yellow fever cases.
FIGURE 34. An early twentieth century map of mosquito populations in New Orleans.
FIGURE 35. 1905 yellow fever cases by block in New Orleans.


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