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When Bioterrorism Was No Big Deal

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ABSTRACT

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To better understand the potential economic repercussions of a bioterrorist attack, this paper explores the effects of several catastrophic epidemics that struck American cities between 1690 and 1880. The epidemics considered here killed between 10 and 25 percent of the urban population studied. A particular emphasis is placed on smallpox and yellow fever, both of which have been identified as potential bioterrorist agents. The central findings of the paper are threefold. First, severe localized epidemics did not disrupt, in any permanent way, the population level or long-term growth trajectory of those cities. Non-localized epidemics (i.e., those that struck more than one major city) do appear to have had some negative effect on population levels and long-term growth. There is also modest evidence that ill-advised responses to epidemics on the part of government officials might have had lasting and negative effects in a few cities. Second, severe localized epidemics did not disrupt trade flows; non-localized epidemics had adverse, though fleeting, effects on trade. Third, while severe epidemics probably imposed some modest costs on local and regional economies, these costs were very small relative to the national economy.

1. Introduction

During the seventeenth, eighteenth, and nineteenth centuries, American cities were subjected to sporadic outbreaks of epidemic disease. These epidemics were far more severe than anything that has been observed during the past one hundred years. For example, in 1721 a smallpox epidemic in Boston killed 1 of every 10 city residents (Blake 1959, pp. 73-75). A smallpox epidemic in Charleston (South Carolina) in 1698 and 1699 might have killed up to a quarter of the city's population.¹ In 1878, a yellow fever epidemic in Memphis killed 1 of every 8 city residents. The death toll in Memphis would have been even higher had two-thirds of the white population not fled the city.² In Shreveport, Louisiana a yellow fever epidemic in 1853 killed one-quarter of the city's population (Keating 1879, p. 89). Less severe outbreaks of yellow fever in 1699 and 1792 killed nearly 1 of every 10 Philadelphia residents.³ To put these numbers in context, consider the experience of Camden, New Jersey during the great influenza pandemic of 1918. At this time, Camden had a higher death rate from influenza than any other major American city (U.S. *Mortality Statistics* 1918). Influenza killed 1 of every 80 Camden

¹For numbers of deaths, see Bridenbaugh (1968b), p. 240, and Duffy (1953), pp. 140-43. For population in Charleston at this time, see Coclanis (1989), p. 114.

²On the death rate in Memphis, see Humphreys (1992), p. 5. See also, Wrenn (1987) and Ellis (1992), and Keating (1879) for similar numbers and assessments.

³Although the 1792 epidemic has been the better documented of the two outbreaks, the earlier epidemic likely killed a larger proportion of the population. On the number of deaths and rough approximations of death rates in Philadelphia from these epidemics, see Griscom (1858), p. 2; Keating (1879), pp. 77-79; Duffy (1953), pp. 138, 142, 144-45, 151-54, and 158-62. On the population of Philadelphia during seventeenth and eighteenth centuries, see Klepp (1989) and Smith (1977).

residents, one-tenth the death rate observed in Memphis a half-century earlier. Along the same lines, more people died in the Memphis yellow-fever epidemic than in the Chicago fire, the San Francisco earthquake, and the Johnstown flood combined (Bloom 1993, p. 1).

For economists, these epidemics are interesting on at least two levels. First, both smallpox and yellow fever have been identified as possible bioterrorist agents (Jahrling 2002; Peters 2002). Although the possible effects of bioterrorism have been studied extensively using computer simulations, this work focuses on epidemiological questions such as how quickly an outbreak would spread and how many people would perish (Bozette *et al.* 2003; Del Valle *et al.* 2005; Kress 2005). Less attention has been paid to the possible economic and financial repercussions of a bioterrorist terrorist attack, or even a severe localized epidemic of natural origin. Historical observers claimed that these epidemics brought economic activity to a halt, as people fled and as neighboring towns imposed quarantines and trade embargos on the infected area (Bloom 1993, pp. 150-55; Humphreys 1992, pp. 77-93). There is, however, a dearth of systematic evidence to support or reject such claims. One might also ask if epidemics capable of killing 10 to 25 percent of an urban population were also capable of inducing bank and financial panics, widespread business failures, and the permanent relocation of labor and capital away from the infected area.

Beyond the implications for national security, these epidemics might also help inform current debates in development economics. For example, it is often suggested that diseases like malaria are an important source of economic stagnation and underdevelopment in sub-Saharan Africa and other tropical climates (Sachs 2003; Sachs and Malaney 2002). Because the

epidemics considered here came at a time when American cities were at a comparatively early stage of development and were especially vulnerable to events that might disrupt economic activity, their experiences might have some relevance for the disease-underdevelopment hypothesis. Furthermore, recent research suggests that in high-mortality areas policymakers are likely to adopt institutional structures that inhibit the region's long-term growth prospects (Acemoglu, Johnson, and Robinson 2001). In light of this research, one might ask if severe epidemics in American cities also gave rise to pathological institutions. Historians, for example, have hypothesized that cities in the American South promoted residential segregation because local whites feared that epidemic disease would more easily spread from black households to white in integrated settings (Doyle 1990, pp. 282-83).

2. Preliminary Considerations

2.A. Why Epidemics Might Influence Economic Activity

The extent to which severe epidemics might alter the location of economic activity is shaped partly by the expectations of economic actors. Consider a scenario where before ever locating in city *A*, actors believe that there is a high risk of an epidemic in city *A* relative to other cities. If such expectations are realized, actors might temporarily flee the city in the midst of the epidemic but they will return once it subsides. Similarly, even an unanticipated outbreak of disease might not have a permanent effect on the location of population if actors believe the outbreak is random and independent of future outbreaks. Only if actors believe that the epidemic is telling them something new and undesirable about city *A* will it induce a permanent change in the city's population. If, for example, actors believe that epidemics are

positively correlated with one another so that a severe outbreak of disease in year t raises the probability of a severe outbreak in year $t + k$, an epidemic might induce some residents to permanently leave the city and discourage new migrants from locating there.

In addition, agglomeration economies and city size might help determine a given city's vulnerability to epidemics and its ability to recover from those epidemics. On the one hand, the presence of productivity advantages in a particular region, whether stemming from agglomeration or natural characteristics, might make it easier to attract replacement workers and recover from adverse shocks.

On the other hand, large and densely populated cities are at greater risk for experiencing serious outbreaks of epidemic diseases than small, less-dense areas. High population densities facilitate the spread of infectious disease, particularly diseases like smallpox and yellow fever (Crosby 1993; Cooper and Kiple 1993). Furthermore, while exposure to infectious diseases often confers some degree of immunity against later attacks, rapid population growth implies an ever expanding group of vulnerable individuals and/or individuals carrying new pathogens. In the nineteenth-century U.S., for example, some observers argued that epidemics were worst in boom periods when cities were attracting relatively large numbers of new and previously unexposed immigrants. There is also econometric evidence that, during the mid-nineteenth century individuals living in regions with high levels of trade and migration had, holding everything else constant, worse health than those living in less economically vibrant areas (Haines *et al.* 2003). In New Orleans and other Southern cities, yellow fever was called "strangers disease" because newly-arrived

migrants tended to suffer more from the disease than long-time city residents (Carrigan 1970; Humphreys 1992, pp. 7-8). Finally, rapid population growth might overwhelm existing public-health infrastructure such as local housing stocks and public water and sewer systems, creating an environment conducive to the spread of disease.

Institutions, particularly financial institutions, might also help determine the broader economic effects of epidemic disease. For example, deposit insurance and access to a lender-of-last-resort would have made it easier for banks to survive an onslaught of withdrawals while local populations sought liquidity to finance their temporary flight from an area experiencing a severe epidemic. Similarly, a well-developed financial network would have enabled local businesses and retailers to survive the quarantines and temporary downturns in the local economy which accompanied nearly all serious outbreaks of smallpox and yellow fever. The causality here, however, need not have been unidirectional; institutions might not only have mitigated the economic fallout of epidemics, they themselves could have been shaped by those same diseases. Epidemics that had the capacity to kill ten percent of an urban population in a few months time probably also had the capacity to generate legal and political change.

Finally, the geographic distribution of disease would have shaped the impact of particular epidemics. Suppose, for example, yellow fever and smallpox struck all places with equal frequency and severity so that cities only differed in the timing of their particular epidemics—e.g., some cities suffered an epidemic in year t , others in year $t+1$, others in year $t+2$, and so on. If this were the case, it seems unlikely that any given epidemic, no matter how severe in absolute terms, would have had a lingering effect on the development of particular

city or town, though it certainly could affect urbanization rates for the country or region as a whole. The historical evidence presented below, however, indicates that cities were not equally vulnerable to disease, and that migrants could have altered their locational choices if avoidance of disease were of primary importance. Yellow fever, for example, never struck a city in Maine, and only rarely visited Massachusetts, western Pennsylvania, and Rhode Island. In the American South, with a few exceptions, yellow fever never struck places well inland or those of even modest elevation (Toner 1873).

Consider too the difference between highly localized epidemics that struck only one city versus those that struck multiple cities simultaneously within some broader regional context. In all but one of the yellow fever epidemics discussed below, the disease did not spread beyond one or two major cities. The one exception occurred in 1878 and 1879 when an outbreak of yellow fever spread across the entire Mississippi Valley, affecting towns all over the South and lower Midwest. Cities likely would have had a harder time recovering from such widespread and non-localized outbreaks because other neighboring population centers also would have experienced significant population losses and would have been competing among one another to attract new migrants.

2.B. Smallpox

Smallpox was a viral and highly-contagious disease, typically transmitted when a vulnerable individual inhaled the virus, absorbing it into his or her upper respiratory tract. Historically, the disease might have infected up to 90 percent of the population at risk. There were two kinds of smallpox, *Variola major* and *Variola minor*. The former was more serious and

had a case fatality rate of 25 to 30 percent, while the latter produced relatively minor symptoms and had a case fatality rate of 1 percent or less. Once a vulnerable individual was exposed to smallpox, there was an incubation period of about twelve days. After this, the disease erupted in a high fever, head and muscle aches, vomiting, and convulsions. In the most severe infections, the stricken individual died quickly from a hemorrhagic disease resembling septic shock before the appearance of the rash pathognomic to smallpox. About three-quarters of all cases, however, developed a rash, with pustules eventually appearing on the face, hands, feet, and other parts of the body (Crosby 1993; Bray and Buller 2004).

Because individuals who survived smallpox exposure in their youth developed a lifelong immunity, smallpox is often thought of as a disease primarily of childhood and infancy. Consider the experience of Manchester, England between 1769-1774. Over this six year period, smallpox was ever-present in Manchester and accounted for just over 15 percent of all deaths in the city. Moreover, of the 589 smallpox-related deaths recorded over this interval only one occurred in an individual over the age of ten. Similar distributions were observed in Liverpool and Warrington (Creighton 1894, pp. 534-37). Having said this, the idea that smallpox was primarily a disease of childhood is easily overstated. Certainly in places where smallpox was endemic, individuals who lacked the capacity to fight off the disease were quickly taken from the population. But in times and places where smallpox epidemics were more sporadic, the distribution of deaths across age groups could become much flatter, and in some cases, could even be skewed toward adults and the elderly (Creighton 1894, p. 443-45; Humphreys 1897, p. 539). During an epidemic in Paris in 1850-51, 50 percent of all deaths

occurred in individuals 20 years of age or older (Creighton 1894, p. 611). In the American epidemics discussed below, adults bore the brunt of the disease.

The breadth and severity of smallpox epidemics depended, in part, on environmental conditions. Much like tuberculosis, smallpox did not thrive in regions with low population and housing densities. Smallpox also withstood dry and cool climates better than humid and warm ones (Crosby 1993). Migration patterns were important as well. Waves of in-migration to cities from rural areas, where smallpox was much less prevalent, increased the proportion of the urban population that was vulnerable to the disease and raised the likelihood of an epidemic. Put another way, migration patterns help explain why smallpox death rates often exhibited a cyclical quality over time. When the population of vulnerables was large, smallpox epidemics would strike year after year until all the vulnerables perished, only to return again once the stock of vulnerable individuals had been replenished (McNeill 1977, pp. 76-79). Many historical observers argued that the incidence of smallpox was correlated with other diseases and that it struck those in already unhealthy environments the most. Such observers emphasized the simultaneous declines in cholera, diarrheal diseases, and smallpox (Biggs 1912, pp. 210-14). This led some observers, particularly those opposed to mandatory vaccination, to claim that smallpox epidemics were caused, in part, by inadequate sewerage and poor water supplies. Although the idea that smallpox could be transmitted through sewer gas or tainted water is devoid of scientific support, many nineteenth-century scientists acknowledged that there appeared to be a correlation between smallpox and waterborne diseases (Carpenter 1882; Royal Commission on Vaccination 1898, pp. 96-98).

Although an effective vaccine was not widely available until the early nineteenth century, inoculation (also referred to as variolation) often helped to inhibit the spread of the disease in earlier time periods. A crude form of vaccination, inoculation involved deliberately infecting vulnerable individuals with mild cases smallpox. It is not clear when or where the practice of inoculation began. In China, inoculation is well documented for populations after the eleventh century and the practice likely began in the southwest of the country, along its border with India. The Chinese aged the scabs from mild smallpox cases in an effort to attenuate the virus, and then blew the scabs up the nostrils of the patient. In the Americas and Europe during the eighteenth century, various practitioners of folk medicine took pus from smallpox victims and scratched it into the skin of vulnerable individuals. Although inoculation usually prevented individuals from developing fatal forms of the disease, there were at least two problems with the practice. First, the inoculated individual became contagious and could help spread the disease as long as he or she had an active case. Second, between 1 and 4 percent of all those inoculated developed fatal cases of smallpox. In light of these risks, most people did not resort to inoculation unless in the midst of an epidemic, in which case the risks of inoculation were outweighed by the risks of contracting a more virulent strain of the disease (Crosby 1993; Hopkins 2002, pp. 109-12).

Modern forms of vaccination were developed when Edward Jenner, a scientist and practitioner of inoculation, noticed that individuals who had previously suffered from cowpox (a relatively mild pox disease spread by cattle) did not exhibit the symptoms of smallpox, even after inoculation. Building on this observation, Jenner injected several people, including his

own son, with cowpox. He then exposed these individuals to smallpox through inoculation. When the test subjects failed to develop smallpox, Jenner knew he had made a major discovery. Soon after his findings were published in the summer of 1798, they spread rapidly across Europe, parts of Asia, and North America. By 1801, England had vaccinated more than 100,000 persons, and in the years that followed, France, Germany, Russia, and Spain vaccinated millions more. Between 1808 and 1811, France alone vaccinated nearly 2 million people (Crosby 1993; Hopkins 2002, pp. 77-81).

One can get some appreciation of the effects of vaccination by looking at the evolution of smallpox in London. Figure 1 plots the death rate from smallpox in London over the period 1670 to 1840. Between 1670 and 1800, there were, on average, 2.7 deaths from smallpox per 1,000 persons every year. After the introduction of vaccination around 1800, the annual death rate averaged .684, a drop of roughly 75 percent. Although the data in figure 1 suggest a mild downward trend in the smallpox death rate before the introduction of Jenner's vaccine, there is a clear break in trend after 1800. Moreover, the variance in the death rate drops sharply. Before 1800 the smallpox death rate frequently spiked at levels as high as 5 or 6, while between 1800 and 1840, the death rate was much more stable and never rose above 2. Historical demographers have documented reductions of similar magnitudes following the introduction of vaccination for nineteenth-century India (Banthia and Dyson 1999), eighteenth-century Finland (Mielke *et al.* 1984; Pitkanen *et al.* 1989), and eighteenth-century Sweden (Skold 1996). It is important to note, however, that smallpox epidemics continued to erupt sporadically throughout the nineteenth and early twentieth centuries. Many cities and towns in England,

for example, experienced serious outbreaks of the disease during the 1870s and 1880s (Biggs 1912, pp. 276-89).

Based on a chance observation, the discovery of the smallpox vaccine was an exogenous event that reduced the relative costs of living in densely populated areas otherwise highly vulnerable to outbreaks of smallpox. Throughout the seventeenth and eighteenth centuries, rural areas were much less vulnerable to smallpox epidemics than were densely-populated cities. If smallpox epidemics represented a significant check on urban development, this discovery should have encouraged migration to urban centers in both Europe and the United States. We will exploit this prediction later in the paper to assess the impact of smallpox on urban development.

2.C. *Yellow Fever*

To nineteenth century observers, yellow fever was a mysterious and frightening disease, whose more popular historical monikers included bleeding fever, yellow Jack, and *vomito negro*, for the blackened blood thrown up by many patients. As its name implies, the disease adversely affected liver function (resulting in jaundice) and caused a high fever. Other symptoms included headache, restlessness, chills, and nausea. For those who survived, the disease reached its peak three or four days after the onset of symptoms. For those who did not, death usually came after a week of suffering. One or two days before death, the patient's kidneys would shut down and urine output would cease. Profuse internal hemorrhaging resulted in blackened vomit, and bleeding from the gums, nose, mouth, and even old bruises (Cooper and Kiple 1993; Humpheys 1992, p. 6). In an era of limited medical understanding,

medical treatments for the disease ranged from the benign to the malignant. On the latter end of the spectrum, some physicians continued to use poisons such as antimony and mercury, as well as bleeding and ice-cold baths to treat the sick (e.g., Hogg 1840). On the other end, homeopaths used bed rest, cool baths, various herbal mixtures such as Peruvian tree bark, and diet in an effort to check the disease. Life insurance companies at this time were reported to have offered their customers discounts if they would limit themselves to homeopathic treatments (Nuwer 1996, pp. 12-16). An effective vaccine to prevent the spread of the yellow fever virus was not developed until the twentieth century.

While smallpox often left parents childless, yellow fever more often leaves children parentless. In young children, yellow fever “tends to be quite mild (Bloom 1993, p. 11).” During an epidemic in Norfolk and Portsmouth, Virginia in the 1850s, hundreds of children were left parentless and an orphanage had to be created (Portsmouth Relief Association, 1859). During the 1878 epidemic in Memphis, the Catholic Church published the following appeal in prominent newspapers across the country (*New York Times*, August 24, 1878, p. 1):

The scourge of yellow fever is again upon Memphis, and many will be left orphans. St. Peter’s Asylum in this city is already overburdened. Substantial sympathy is needed. Please send contributions to Rev. J.A. Kelly, St. Peter’s Church.

Vulnerability to yellow fever also differed by race. After thousands of years of repeated exposure, Africans developed a genetic resistance to the disease which persons of European extraction did not possess (Carrigan 1970; Kiple and Kiple 1977).

In severe epidemics, case fatality rates were said to be as high as 70 to 90 percent,

though more typical reports suggest a range between 10 and 25 percent (e.g., Keating 1879, pp. 77-98; Griscom 1858; and Spinzig 1880, pp. 155-74). There is also some debate among medical historians if the high case fatality rates associated with yellow fever historically reflected poor diagnostic skills and under-reporting of the true incidence of the disease (Humphreys 1992, pp. 5-6). According to others, recent laboratory experiments indicate that yellow fever has many strains, and that the pathogenic properties of these strains differ markedly (Bloom 1993, pp. 10-11). This suggests it might have been possible to have case fatality rates as high as 90 percent in some outbreaks and rates as low as 5 or 10 percent in others.

Not until the 1890s did scientists discover that yellow fever was spread by mosquitos, in particular the *Aedes aegypti*. The peculiar characteristics of this mosquito help explain why yellow fever existed almost exclusively in cities and towns, and was largely unknown in rural areas. Sometimes referred to as a "cistern mosquito," *A. aegypti* is a small and gray-backed insect common throughout the American South and the Carribean. It breeds in fresh water sources that are clear and relatively free of organic activity such as cisterns, metal gutters, and buckets. Such man-made containers are attractive because the mosquitos can cement their eggs to a stable and flat surface. By the same token, the *A. aegypti* avoids marshes, swamps, lakes, and water sources otherwise polluted with mud, urine, and feces. *A. aegypti* flourishes in temperatures between 70° and 90° Fahrenheit; its activities begin to slow when temperatures dip below 70° and it will not feed when temperatures are below 60°. It becomes inert at temperatures below 50°. Nevertheless, the eggs of *A. aegypti* are robust, and historically, could survive the relatively mild winters observed in the American South. This helps to explain why

yellow fever epidemics occasionally stretched over two or three years (Bloom 1993, pp. 22-28; Carter 1914, pp. 4-10).

Often, yellow fever would lay dormant for years and then erupt suddenly, killing hundreds or thousands of people within a few months time. In Memphis, for the half-century preceding 1878, in only four years (1853, 1855, 1867, and 1873) did anyone in the city die of yellow fever. As figure 2 illustrates, similar patterns can be observed in other cities in the American South. Yellow fever epidemics struck Savannah, Georgia in 1820, 1854, and 1876 while in the intervening years, no one in the city perished from the disease. Rivaling the Memphis epidemic of 1878, the 1876 outbreak in Savannah outbreak killed as many as 1 of every 13 residents who remained in the city during the epidemic. Similarly, in Charleston, South Carolina, yellow fever epidemics struck the city on and off during the eighteenth and nineteenth centuries. In 1871 and 1872, 113 Charleston residents were killed in a yellow fever outbreak, but over the next three decades, no one else was stricken (Toner 1873; Keating 1879, pp. 80-98).

Aside from a few relatively small epidemics in Florida, Mississippi, and Louisiana, after 1880 yellow fever struck with much less frequency and severity in the American South. This shift came about quite by accident. After 1880, cities throughout the U.S. began to rapidly expand their public water systems. Although cities extended public water systems largely to combat typhoid fever and diarrheal diseases, the effect was to bring piped water to populations that had previously relied on cisterns for water. In turn, urban dwellers began abandoning their cisterns, a favorite breeding ground of the *A. aegypti* (Bloom 1993, pp. 24, 226-28; Carter

1914, pp. 16-23). If yellow fever were an important hindrance to urban development, cities that previously had been vulnerable to severe and repeated epidemics would have started to grow faster once those epidemics began to subside. Similarly, after Walter Reed demonstrated that yellow fever was spread by mosquitos in 1899, preventing outbreaks of the disease was a comparatively easy task and would have promoted urban growth in the cities and places hitherto most prone to epidemics if those outbreaks represented a significant barrier to growth.⁴

2.D. *On the Geographic Distribution of Yellow Fever*

In 1873, the president of the American Medical Association, Dr. J.M. Toner, delivered a lengthy paper titled, "On the Natural History and Distribution of Yellow Fever in the United States." Culling articles from every medical journal and government report that he could find, Toner tried to document as many outbreaks of yellow fever as possible that occurred in the United States from 1668 through the early 1870s.⁵ More than a century later, Toner's paper remains probably the most complete record historians have of yellow fever epidemics in the U.S. prior to 1874. According to Toner, there were at least 723 epidemics of yellow fever in 229

⁴Reed, it should be noted, was not the first person to maintain that yellow fever was spread by mosquitos. Working in Havana, Dr. Carlos Finlay published a paper in 1881 presenting evidence that yellow fever was spread by the *A. aegypti*. In part because his experiments were flawed, Finlay's hypothesis was largely ignored by the medical community. See Bloom (1993), pp. 20-21; and Smith (1951), pp. 101-03. On Reed's experiments and their broader importance, see Agramonte (1915).

⁵Having said this, Toner (1873, p.3) acknowledged that he undoubtedly missed a few epidemics, but at the same time, he was "confident that such localities will be found within the region of this general distribution, as here indicated."

cities and towns. With few exceptions, these towns were below 500 feet above sea level, and were coastal, bordering major rivers and their tributaries, the Atlantic Ocean, or the Gulf of Mexico. Although yellow fever bore disproportionately on places in the American South, Toner's data indicate that northern cities were not exempt from the disease.

Based on data presented in Toner's original paper, table 1 helps to highlight these patterns in greater detail. More than half of all yellow fever epidemics took place in four states: Louisiana (23 percent of all epidemics); New York (12 percent); Texas (10 percent); and South Carolina (9 percent). Moreover within in these states, epidemics were concentrated in large cities that served as major seaports. New Orleans accounted for 66 percent of all the epidemics in Louisiana, New York City for 74 percent of all epidemics in New York state, and Charleston for 81 percent of all epidemics in South Carolina. It is notable that aside from New Orleans, the American city most often afflicted with yellow fever was New York, the largest and fastest growing city in the country. Equally notable, however, is the absence of yellow fever in places like Portland, Maine and Newport, Rhode Island. Although both of these port cities were widely recognized by historical observers as especially salubrious places, neither approached New York City or New Orleans as major trading centers (Albion 1939, pp. 26, 389; Bridenbaugh 1964b, pp. 88, 442; Duffy 1953, pp. 52-57; Mason 1884, pp. 12-13, 149-53, 235-40).

Aside from New York, the diffusion of yellow fever was greatest in the Southern states. In Alabama, 11 different cities were afflicted, at one point or another, by the disease, though more than half of the epidemics in the state occurred in Mobile. In Florida, thirteen cities were struck, with three cities bearing the brunt of the outbreaks, Saint Augustine, Key West, and

Pensacola. In Louisiana, yellow fever erupted in 43 different cities and towns. Aside from New Orleans, the state's most vulnerable towns were Alexandria, Baton Rouge, Shreveport, Thibodeaux, and Washington. In Mississippi, yellow fever was also widespread, affecting at least 20 different localities between 1668 and 1874. Natchez, where yellow fever struck most often, accounted for only 24 percent of the outbreaks observed in the state. Yellow fever was most diffuse, however, in Texas, where outbreaks occurred in at least 41 places. The two Texas cities with the most frequent visitations from yellow fever were Galveston and Houston, which together account for only 28 percent of all outbreaks in the state. In later sections of the paper, we will exploit this interstate variation in the diffusion of yellow fever to draw inferences about how the disease influenced urban development.

3. Epidemics and Long-Term Growth Trajectories

One way to assess the economic effects of severe epidemics on particular cities is to examine population growth before and after the event. Population growth is an attractive metric for at least three reasons. First, reliable and regularly-reported estimates of population are available for many large American cities for the past three or four hundred years. Even for relatively small towns and rural counties population data are often available for two hundred years or more. The same cannot be said of other possible economic measures such as wages and income. Second, even if wage and income data were available, population movements are likely a more revealing source of information. To the extent that labor was mobile, migration would have mitigated the wage and income effects of productivity shocks (Beeson *et al.* 2001). Third, population movements encapsulate information not only about productivity but also

about urban amenities.

Consider first the experience of the four largest cities in colonial America circa 1770: New York; Philadelphia; Boston; and Charleston. Figure 3 demonstrates the small effect smallpox and yellow fever epidemics had on population growth in New York City. Striking in 1702, New York City's worst yellow fever epidemic killed more than five hundred residents, or roughly 10 percent of the local population. Less severe outbreaks occurred in 1743, 1795, 1798, 1803. The epidemic in 1743 killed 217 people, 2 percent of the city's population. The epidemic in 1798 killed 2,080 people, 4 percent of the city's population. New York City's worst outbreak of smallpox occurred in 1731, killing 549 people, about 6 percent of the local population.⁶ To put these numbers in context, the great influenza pandemic of 1918 killed only six-tenths of one percent of the residents living in New York City (United States, *Mortality Statistics*, 1918). The death rate associated with the yellow fever epidemic of 1702 exceeded the influenza death rate by a factor of 16.7. Nevertheless, figure 3 suggests that even the most severe outbreaks of smallpox and yellow fever failed to disrupt New York City's long-term growth trajectory. There was a small dip in the city's population in 1703, but rapid in-migration soon overwhelmed this perturbation. There is, however, some modest evidence that once the severity of yellow fever epidemics in the city began to subside after 1830 population growth became more stable.

⁶Data on the number of deaths from smallpox and yellow fever are taken from the following sources: Bridenbaugh (1968b), pp. 399-400; Duffy (1953), pp. 76-78, 81, 84-86, 88, and 91; Toner (1873), pp. 28-29; and Griscom (1858), pp. 1-18. Data for New York City's population (to calculate death rates) are from Rossiter (1970), p. 11, and Carter *et al.* (2006a), p. 1-110, Series Aa832.

Epidemics of yellow fever struck the city of Philadelphia intermittently throughout the seventeenth, eighteenth, and early nineteenth centuries. An epidemic in 1699 killed 220 people, about ten percent of the city's population. Except for a few comparatively small outbreaks in the 1740s, the city had to wait nearly a century before an equally severe epidemic struck. An epidemic in 1792 and 1793 killed just over 4,000 individuals, or about 8 percent of the city's population. A string of epidemics in 1797, 1798, and 1799 killed more than 5,800 people. At the time, Philadelphia's population was between 65,000 and 69,000. Outbreaks during the early 1800s were much less severe and generally did not involve more than a hundred people. As figure 4 shows, these epidemics do not appear to have had much an effect on population growth in the city. After 1710, the city's population grew exponentially, and while growth before 1710 was slower, the epidemic in 1699 does not appear to have altered the trend in either direction.⁷

Having said this, by 1810 New York had surpassed Philadelphia as America's largest city. Was Philadelphia's relative decline the result of the repeated yellow fever epidemics during the 1790s? It seems unlikely. As shown above, New Yorkers were immune to neither smallpox nor yellow fever. On the contrary, yellow fever struck New York City 62 times between 1668 and 1874; it struck Philadelphia only 34 times (Toner 1873, pp. 28-31). Moreover, differences in population growth between New York and Philadelphia began long before 1790,

⁷Data on the number of deaths from yellow fever are from the following sources: Griscom (1858), p. 2; Keating (1879), pp. 77-79; Duffy (1953), pp. 138, 142, 144-45, 151-54, and 158-62; Toner (1873), pp. 30-31. On the population of Philadelphia during seventeenth, eighteenth, and nineteenth centuries, see Klepp (1989), Smith (1977), and Carter *et al.* (2006a), p. 1-110, Series Aa841.

and the differences that emerged in the wake of the epidemics of the 1790s were small by historical standards. This can be seen in figure 5. Lastly, the extant historical literature attributes Philadelphia's relative decline in the long term to its failure to keep pace with the development of the Erie Canal and various railroads linking the Port of New York to the country's interior (e.g. Albion 1939, pp. 1-13, and 373-86).

Before 1800, Boston suffered epidemics of yellow fever during the years of 1691, 1693, 1795, 1796, 1798, but there are no precise data that would allow one to assess the severity of these outbreaks. Moreover, the fragmentary evidence that does exist suggests they were not especially severe (Toner 1873, pp. 25-26; Blake 1959, pp. 151-76). There are, however, data that make it possible to consider the effects of smallpox. Boston experienced severe smallpox epidemics in 1667-68, 1678-79, 1702, 1721, 1730, 1752, 1761, 1776, 1779, 1788, and 1792. The worst of these were the 1702, 1721, and 1730 outbreaks, each of which killed between 6 and 10 percent of the city's population.⁸ According to Creighton's (1894, p. 626) authoritative history of disease, these epidemics affected adults in Boston more so than children. As a means of assessing how these epidemics affected the city's long-term growth trajectory, figure 6 plots the population and overall mortality rate in Boston from 1700 through 1740. The unusual spikes in the city's death rate in 1702, 1721, and 1730 illustrate the most severe smallpox epidemics.

Although there appear to have been small drops in the city's population following the 1702 and 1721, population growth quickly recovered and returned to its long-term growth rate. By 1776,

⁸Information on smallpox in Boston comes from the following sources: Blake (1959), pp. 2, 15, 19-20, 60-61, and 74-98; Bridenbaugh 1968b, pp. 86-87, and 240-41; Duffy (1953), pp. 44-52, 57, 64, and 68. Data on population are from Rossiter (1970), p. 11.

Boston was the third largest city in the American colonies (Coclanis 1989, p. 116).

As figure 7 illustrates, Charleston experienced a wave of severe and apparently inter-related yellow fever and smallpox epidemics during the late 1690s and early 1700s. The worst of these killed roughly 25 percent of the city's population; the less severe epidemics killed only about 10 percent. Despite such carnage, Charleston grew rapidly throughout the first half of the eighteenth century and in 1776 was the fourth largest city in the colonies, behind New York, Philadelphia, and Boston (Coclanis 1989, p. 116). On the eve of the American Revolution, whites in Charleston and the broader South Carolina low country were "by far the richest single group in British North America (Coclanis, p. 7)." Interestingly, at the same time the city's epidemics from smallpox and yellow fever abated during the early nineteenth, Charleston's population growth slowed. Most economic historians attribute the slowdown in Charleston's growth to a decline in the marketability of rice, the region's primary export, and the absence of a significant inland waterway near the city (Albion 1959, pp. 102-06; Coclanis 1989, pp. 111-58). Near Charleston there are but two small rivers (the Ashley and the Cooper) that offer little access to the state's interior or the broader region. By contrast, the ocean ports at New York City, New Orleans, and Philadelphia connected to large inland waterways: the Hudson, Mississippi, and Susquehanna, respectively. Such connections made it possible to integrate international markets with a vast number of interior ones.⁹

That yellow fever constituted no great impediment to any port city that was

⁹On the significance of navigable inland water ways near major ocean ports, see Albion (1959) for a qualitative description on the importance of such connections. For quantitative evidence, see Beeson *et al.* (2001) and Rappaport and Sachs (2003).

advantageously situated is particularly clear from the history of New Orleans. No city in the United States was as susceptible to yellow fever as was New Orleans. (See table 1.) Between 1790 and 1860, there were no fewer than 29 epidemics in the city, and such epidemics routinely killed 2 to 8 percent of the local population within a few months time. In one three year period—1817, 1818, and 1819—more than 3,000 people died of yellow fever. In 1816, the city's population stood around 20,000, suggesting that 15 percent of the population was killed by this three year epidemic.¹⁰ Similarly, between 1853 and 1855, more than 13,000 people died of yellow fever, while the population was probably no higher than 130,000. This implies that around 10 percent of the local population was killed. By contrast, during the influenza pandemic of 1918, just below 8/10 of 1 percent of the city's population died of the flu. Although the frequency and severity of yellow fever epidemics dropped sharply after 1880, outbreaks continued to afflict the city until the early twentieth century. Yet none of this prevented New Orleans from growing at a rapid pace. On the contrary, figure 8, which plots the death rates from major yellow fever epidemics alongside population, suggests the city realized its quickest growth when yellow fever was at its worst. It is notable too that population growth in New Orleans does not show any bounce after the disease was eradicated in the early 1900s, and only declines with adverse shocks that occurred during the late twentieth century.

One might argue that preceding discussion focuses on cities that, *ex post*, grew unusually fast; places that were, because of natural advantages and/or agglomeration

¹⁰Data on yellow fever in New Orleans are taken from Toner (1873), pp. 23-24; Keating (1879), pp. 78-98; Carrigan (1961), p. 223; and Humphreys (1992), p. 4. Data on population are from Carter *et al.* (2006a), p. 1-111, Series Aa 861.

economies, predestined to grow quickly and overcome whatever adverse shocks came their way. There are two difficulties with this line of thought. First, it is difficult to portray Charleston's growth trajectory after the Civil War as anything but mediocre. Second, it was not only large and fast-growing cities that exhibited such resiliency. Similar patterns can be observed in smaller cities and towns throughout the deep South. Table 2, for example, presents data for three small cities in antebellum Louisiana. In 1853, a severe yellow fever epidemic affected cities and towns throughout Louisiana. In Alexandria, the pestilence killed 16 to 20 percent of the population; in Baton Rouge, it killed 5 percent; and in Shreveport, it killed close to a quarter of the population. Yet there is no evidence that the such widespread killing prevented these places from realizing substantial population growth. On the contrary, between 1850 and 1860, the population of Alexandria more than doubled; the population of Baton Rouge grew by nearly 40 percent; and the population of Shreveport grew by just over 25 percent. The Civil War might have had an effect, but even on this score the evidence is not particularly strong. Between 1860 and 1870, the population of Alexandria fell by 17 percent, and the population of Baton Rouge grew by only 20 percent.¹¹

Similarly, figures 9 and 10 illustrate the experiences of Mobile, Alabama and Natchez, Mississippi. Much like New Orleans, both Mobile and Natchez were subjected to frequent and severe epidemics of yellow fever during the first half of the nineteenth century. These

¹¹Data on population for these places are taken from various volumes of the U.S. Census for appropriate years. Data on the number of deaths, or approximate death rates, are taken from Toner (1873), pp. 21 and 25; and Keating (1879), pp. 87-90.

epidemics routinely killed 5 to 14 percent of the local population within a few months time.¹²

In Natchez, population growth exhibited no break in trend after 1850, when yellow fever in the city subsided.¹³ This suggests that the severe epidemics observed in the city between 1800 and 1840 did little to slow growth or initiate decline. Notice too that growth in Natchez exhibited a significant break in trend between 1870 and 1880, long after the most serious yellow fever epidemics in the city had ceased. We will return to a discussion of the causes of this stagnation later in the paper. In Mobile, population grew rapidly during the antebellum period, and experienced no slowdown until after the Civil War and after the incidence of yellow fever subsided. Historians attribute Mobile's stagnation after 1865 to the shallowness of Mobile Bay, which required periodic dredging to be accessible to anything other than shallow-draft sailing ships (Summersell 1949, p. 17). The construction of railroad lines to competing ports in New Orleans and Pensacola helped to further undermine the city's position (Summersell 1949, p. 46-47).

¹²Data on population in Mobile are gathered from the following sources for the antebellum period: DeBow (1854), p. 192 and Amos (1985), pp. 1, and 11-12. For the postbellum and early twentieth century periods, data are from Carter *et al.* (2006a), p. 1-113, series Aa917. Population data for Natchez during the antebellum period are from: DeBow (1854), p. 193; and Darby and Dwight (1833), p. 332. Data for later years are from various *Census* volumes. Data on yellow fever for both cities are from Toner (1873), pp. 18-19, and 26-27.

¹³The picture is changed, however, if instead of using a logarithmic scale (which allows one to better compare percentage changes rather than absolute ones) an ordinary linear scale is used. With linear scaling, there appears to be an upturn in growth beginning around 1850. Yet even with linear scaling it is clear that the frequent yellow fever epidemics in Natchez did not cause growth to stop or decline.

4. Dead Towns

Still one might object that an underlying selection problem remains: the analysis continues to focus only on towns that survived. Two books, *Dead Towns of Georgia* and *Dead Towns of Alabama*, can help address this concern. These books provide an independent assessment of the factors that caused populations to abandon small cities and towns in the American South. Written in 1878 by Charles C. Jones, *Dead Towns of Georgia* describes the history of ten now extinct localities in Georgia. Jones argued that epidemic disease played, at most, a secondary role in the demise of these places, and he never once mentioned yellow fever specifically. More important forces included the development of railways and sea ports in competing towns; battles between the Indians and British colonists; the relocation of army forts; and the movement of county seats and the state capital (Jones 1878).

In his more recent and exhaustive treatment of dead towns in Alabama, Harris (2001, pp. 63-65) recounts the history of 112 colonial and state towns.¹⁴ Of all these towns, in only one—Blakeley—does Harris attribute its extinction to yellow fever. Located on the northern end of Mobile Bay, Blakeley was located about five miles southeast of Mobile. Incorporated in 1814, Blakeley reached a population of “several thousand” by 1820. According to Harris, the town was abandoned after a series of yellow fever epidemics struck the area in 1819, 1822, 1826, and 1828. Other historians, however, argue that the people who fled the town during the 1819 and 1822 epidemics quickly returned, and that the later epidemics did little to alter the

¹⁴Harris also describes the history of 83 Indian towns and 47 army fort sites. These are not included in the discussion as their rise and fall were determined, almost exclusively, by the vagaries of war and changing military strategies.

town's pre-existing downward trajectory (Amos 1984, pp. 4-6; Parker 1974). Harris also argues that most of the people who permanently left Blakeley moved to Mobile. Yet these apparent refugees of yellow fever were no less vulnerable to the disease in Mobile than they had been Blakeley. Indeed, the same epidemics that struck Blakeley also affected Mobile, and the death rates from yellow fever observed in Mobile over this period were astounding: in 1819 alone, 274 Mobilians were killed by the pestilence, well over a tenth of the local population (Toner 1873; Keating 1878, pp. 79-98). Put another way, if people fled Blakeley because of yellow fever, relocating to Mobile was an odd choice: of all cities in the American South, only Charleston and New Orleans were struck more frequently by the disease than was Mobile (see table 1).

Notably, Harris (2006) and an earlier commentator, Lewis (1847), describe the history of three dead towns that survived serious outbreaks of yellow fever only to succumb to more mundane economic shocks and natural disasters later in their evolution. Cahaba was chosen to be the site of Alabama's first state capital in 1817, and in the years immediately following this decision, Cahaba realized stupendous growth. Unfortunately, soon after this wave of migration began the town was struck by an epidemic of some sort of "malarious" disease that appears to have been yellow fever. Lewis (1847) estimated that no less than 12 percent of the city's population was killed by the epidemic in 1821-22. Nevertheless, Cahaba continued to expand and thrive, even after the state capital was moved to Tuscaloosa in 1826. The decision to move the capital was motivated not by disease, but by a catastrophic flood in 1825. Not until after the Civil War and another destructive flood did Cahaba expire (Harris 2006, pp. 66-67).

Similarly, during the 1820s, the incipient towns of St. Stephens and Claiborne were struck by the same mysterious fever that afflicted Cahaba (Lewis 1847). St. Stephens, once the capital of territorial Alabama, never recovered when Cahaba was chosen to be the capital of the newly formed state. Yellow fever only hastened its decline. Claiborne withered only after the Civil War, when the broader region's cotton economy stagnated (Harris 2006, pp. 71-72, and 101-03.)

Another evidentiary source that avoids the selection problem outlined above is data on counties. As long as counties are defined with time-consistent borders, they will cover the universe of all geographic areas within a particular region and will therefore include the population groups that grew as well as those that did not. Accordingly, the analysis that follows considers how county-level growth responded to yellow fever epidemics. The analysis focuses on 46 counties in Louisiana between 1850 and 1860, a decade in which yellow fever epidemics struck twelve separate counties in the state.¹⁵ The counties considered all have time-consistent borders, but do not include the City of New Orleans which has already been considered and, given its size, would bias the results against finding any effect. If yellow fever undermined growth, the counties that were afflicted with yellow fever should exhibit slower growth than those that were not afflicted, after controlling for initial county size. To test this, a variant on the standard growth model is estimated:

¹⁵All population data are from the U.S. *Census*.

$$(1) \quad \Delta P_i = \alpha + \beta P_i + \gamma Y_i + e_i,$$

where, ΔP_i is the percentage change in population the county between 1860 and 1850;¹⁶ P_i is the natural log of population in the county in 1850; Y_i equals one if the county was struck by one or more yellow fever epidemics between 1850 and 1860, and zero otherwise;¹⁷ and e_i is a random error term.

Table 3 reports descriptive statistics and reveals an important pattern. As of 1850, counties that were not afflicted with yellow fever anytime between 1850 and 1860 had on average one-half the population of the counties that were. That the non-yellow fever counties were on average half as populous as the yellow fever counties at the beginning of the test period suggests that the former were growing at relatively slow pace before the epidemics hit during the 1850s. As a result, they might not be the best control group. To address this concern, we adopt two crude matching procedures. In the first, we exclude from the regressions all non-yellow fever counties that experienced negative population growth between 1850 and 1860; in the second, we also exclude non-yellow fever counties with populations below 7,758, which is the population in 1850 of the smallest county afflicted with yellow fever. Results are reported in table 4. In the full sample of counties, the coefficient on the yellow fever dummy is positive, small, and statistically indistinguishable from zero. For

¹⁶More precisely, $\Delta P_i = \ln(\text{population in county } i \text{ in } 1860) - \ln(\text{population in county } i \text{ in } 1850)$. Because county borders are time invariant in this sample, this is equivalent to estimating the change in population density.

¹⁷Data on yellow fever in Louisiana during this period are from Toner (1873), pp. 21-25; and Keating (1879), pp. 80-98.

the two matched samples, the results are very much the same. In three of the four regressions, the coefficient on the yellow fever dummy is positive (the wrong sign), and in all of the regressions the coefficient is, in terms of absolute value, close to zero and statistically insignificant.

5. Urbanization and the Eradication Smallpox and Yellow Fever

One might further explore the effects of smallpox and yellow fever on urban development by asking how exogenous reductions in their severity and prevalence affected urbanization patterns. The focal points of this analysis are Jenner's discovery of the smallpox vaccine 1799, and Walter Reed's experiments in 1899 demonstrating that yellow fever was spread by mosquitos. Both of these discoveries were unanticipated creative breakthroughs, largely independent of developments in the particular countries and places considered here. Moreover, both the smallpox vaccine and Reed's experiments made it much easier for individuals and local governments to deal with smallpox and yellow fever in constructive and low-cost ways. In light of this, if a significant number of potential urban migrants were avoiding locating in large cities out of fear of epidemic disease, these two discoveries would have reduced such fears and promoted increased urbanization.

Because smallpox was a much more severe and persistent problem in Europe cities than in American ones, it is more likely that Jenner's discovery affected urbanization in Europe than in the U.S. Accordingly, the focus will be on Europe, at least initially. Figures 11-12 plot urbanization rates in Austria, Denmark, France, Germany, the Netherlands, Russia, and

Sweden over time.¹⁸ There is no evidence of a sharp upturn in urban migration immediately following the discovery of the smallpox vaccine. On the contrary, it was forty to fifty years after the discovery of the vaccine that urbanization rates began to rise, and most observers attribute this change to industrialization (e.g., McCloskey 1985, pp. 55-57; Weber 1899, pp. 315-17).¹⁹ In contrast to continental Europe, urbanization rates in Great Britain were rising at an exponential rate long before 1800. The strong trend toward urbanization in England and Wales is depicted in figure 13; urban growth in Scotland is illustrated in figure 14. Along with observed levels, a pre-vaccination/pre-1800 trend line is also plotted. In England and Wales, Jenner's discovery was followed by a notable decline in urbanization between 1800 and 1820, and it was not until 1850 that the urbanization rate reached the level predicted by the historical (pre-1800) trend. In Scotland, there is evidence that urbanization rates began to rise above their predicted levels soon after 1800, though even here one does not observe large deviations from trend until 1850.

Within a decade of Reed's experiments with mosquitos, yellow fever was eradicated from the American South. The last epidemic occurred in 1905, but the death rates in this outbreak (which affected mainly Louisiana and Texas) were a fraction of those observed 50 to 100 years earlier. The death rate in New Orleans, example, was 1.46 deaths per 1,000 persons;

¹⁸Data on urbanization in Continental Europe prior to 1800 are from DeVries (1984), p. 39. For the later periods, data are from Weber (1899), pp. 67-112.

¹⁹One might argue that eradicating smallpox created a more stable urban environment which in turn set the stage for European industrialization. Whatever the merits of such a claim, it is certainly could not be characterized as an orthodox interpretation of the origins of the Industrial Revolution.

in an outbreak of yellow fever in New Orleans in 1819 the observed death rate was 81, or 55 times greater than the 1905 outbreak.²⁰ In light of this one would expect urbanization rates in those American states where yellow struck frequently and severely and affected a large number of places, Reed's discovery would have promoted a shift away from rural areas (where the disease rarely occurred) toward cities and towns (where it had previously flourished). To test this hypothesis, the following measure of a given state's relative level of urbanization (RU) is constructed:

$$(2) \quad RU_{it} = (\% \text{ Urban}_{it}) / (\% \text{ UrbanUS}_t),$$

where $\% \text{ Urban}_{it}$ is the percentage of the population in state i in year t that lives in an urban area (any place with a population greater than 2,500 persons); and $\% \text{ UrbanUS}_t$ is the percentage of the entire U.S. population in year t that lives in an urban area.

Figures 15-17 plot relative urbanization rates in five states where yellow fever epidemics were severe, frequent, and diffuse prior to 1900: Alabama; Florida; Louisiana; Mississippi; and Texas.²¹ Despite the historical ravages of yellow fever in these states and the suddenness of Reed's discovery, there is, at best, modest evidence that urbanization increased in these states following 1900. Figure 15 plots the relative urbanization rates of Mississippi and Alabama over the nineteenth and twentieth centuries. For both states relative urbanization fell

²⁰See Humphreys (1992), p. 163 for the number of deaths in 1905, and Toner (1873), p. 23 for the number of deaths in 1819.

²¹Data on state-level urbanization rates are from Carter *et al.* (2006a): Alabama, p. 1-180, Series Aa2246 and Aa2251; Florida, p. 1-213, Series Aa3012 and Aa3017; Louisiana, p. 1-249, Series Aa 3747 and Aa3752; Mississippi, p. 1-276, Series Aa4307 and Aa4312; Texas, p. 1-348, Series Aa 5874 and Aa 5879.

in the two decades after the Civil War, but starting in 1880, this downward trend was reversed. No significant break in trend was exhibited in either state in the two decades immediately following 1900. Figure 16 plots the relative urbanization rate in Louisiana from 1810 through 1990. Here there is stronger evidence of a change in trend in the decades immediately after 1900. Beginning in 1840, urbanization in Louisiana began to decline relative to the rest of the country, and although this trend began to moderate around 1870, it was not until 1900 that the state's relative urbanization levels stopped falling. This change must have been driven by urban development outside New Orleans because as shown in figure 8 the rate of population growth in that city exhibited no break in trend around 1900.

The long-term evolution of relative urbanization in Florida and Texas is plotted in figure 17. In both states, one observes a steep upward trend in relative urbanization beginning around 1870. This trend appears to have been interrupted during the 1890s but recovered after 1900. Growth in relative urbanization stagnated only after World War II. The interruption in urban development during the 1890s is worth exploring. Between 1891 and 1900, coastal states in the American South were subjected to a series of unusually active and severe hurricane seasons (United States 1976, p. 448). The worst of these was a hurricane that struck Galveston, Texas in 1900. Killing six thousand people, or about 14 percent of the local population, the hurricane was not, in terms of human lives, any more costly than a series of yellow fever epidemics which struck Galveston between 1839 and 1868 (James 1908; Toner 1873; Smith 1951). Nevertheless, this single hurricane had a much greater effect on the city's development than did multiple yellow fever epidemics.

The relative effects of yellow fever and hurricanes on Galveston are highlighted by figures 18 and 19. Figure 18 plots both the death rate from yellow fever and the city's population from 1830 through 1920. Despite four epidemics which killed between 10 and 14 percent of the local population, and three smaller epidemics which killed only 3 to 6 percent, Galveston's population steadily expanded from its through 1870. When the epidemics subsided after 1870, population growth did pick up. But this change was comparatively small when one considers the changes wrought by the hurricane 1900. Within two years of the hurricane, population in the city was halved, falling from around 42,000 to 21,00. It was not until 1920 that the city's population levels recovered to the levels observed in the summer of 1900. (The hurricane struck in September.) Moreover, if one predicts the city's 1920 population based on historical trends before the hurricane, Galveston's population would have been 61 percent greater than it actually was. See figure 19.²² There is no evidence from Galveston or any of the other cities considered thus far that yellow fever ever had such effects on urban development.²³ There is, however, one city where repeated yellow fever epidemics appear to have undermined long-term economic development in ways that came close to rivaling the

²²The city's observed population in 1920 was 44,255, while the population level predicted by the quadratic regression model described in the notes to figure 19 suggests that the city's population would have been 71,117 in the absence of the hurricane.

²³The recovery patterns observed in Galveston are similar to those observed in Hiroshima and Nagasaki following the dropping of the atom bombs. It took 10 to 30 years for these cities to return their pre-atom bomb levels. See Davis and Weinstein (2001). One plausible interpretation of the results in Galveston then is that destroying capital has more severe economic repercussions than simply destroying labor, likely because labor is more mobile than capital.

1900 hurricane in Galveston.

6. Memphis

After the Civil War, the single most important event in nineteenth-century Memphis was the yellow fever epidemic that struck the city in 1878. Killing one of every eight Memphis residents, this was among the worst epidemics (from any disease) to ever strike an American city. As the epidemic raged in the summer of 1878, one-third of the black population and two-thirds of the white population fled the city. Of the blacks that remained in the city, 80 percent contracted yellow fever; of these, 10 percent died. Of the whites that remained in the city, 98 percent contracted the disease; of these, 70 percent died. Only five years earlier, in 1873, a combined epidemic of yellow fever and cholera killed at least 2,000 Memphis residents, about 5 percent of the local population. Nor were these the first epidemics in the city. The year Memphis was chartered (1826), it was afflicted by smallpox, and in the years to follow there were repeated visitations of smallpox, cholera, dysentery, influenza, and yellow fever. Although it is difficult to know whether Memphis was really any worse than other developing American cities in terms of disease, there was a widespread belief that it was. Even the city's own newspaper conceded that "all visitors" saw Memphis as "the filthiest and most deathly appearing town in the Union."²⁴

Memphis certainly seemed an unhealthy place. Lying only 260 feet above sea level, the city was surrounded by swamps, with a miserable bayou that ran through the city and served

²⁴This account is drawn from the following sources: Ellis (1864), (1874); Keating (1879), pp. 99-144; Tennessee State Board of Health (1880); and Toner (1873).

as a sort of all-purpose sewer. The city's climate—hot and wet—seemed conducive to the proliferation of miasmatic poisons. There were only a few miles of private sewers and most residents dumped their waste in cesspools, privy vaults, or in open ditches; there was no system of storm water drainage or public garbage disposal; except for a small, poorly-run private company, there was no public water supply. In 1880, a government investigation found evidence that 72 percent of the city's private wells were contaminated by human waste; hundreds of buildings in the city had standing water in their basements or cellars from two to eighteen inches deep; and the few city streets that were not dirt, were made of rotting wood that emitted a foul smell (Ellis 1964; Keating 1879, pp. 99-144).

When the yellow fever epidemic of 1878 erupted, very few observers could not help believing that all of this filth had something to do with it. In response, officials in Memphis launched a massive campaign to cleanse the city of disease-causing fomites and poisons. In the midst of the epidemic, officials hired teams of people (largely African American given their elevated immunity status and relatively dire economic circumstances) to empty all privy vaults and disinfect them with carbolic acid. The wastes of yellow fever victims were treated with acid and disposed of into impervious containers. The towels, blankets, sheets, curtains, furniture, and mattresses of yellow fever victims were washed with carbolic acid; when these items were too soiled to be cleaned, they were burned. Hundreds of houses so unsanitary that they were suspected of harboring poisons, were burned. Mail entering and leaving the city was fumigated; trains and boats entering and leaving the city were quarantined, as were their passengers. Refugee camps were set up outside the city, and canons and large fires were set

off at night to disinfect the air (Tennessee State Board of Health, 1880, pp. 83-109, 319-27; Keating 1879, pp. 99-144).

By the time the epidemic subsided in the winter of 1879, the city was bankrupt. After revoking the city's charter, the state also stripped Memphis of its name. The city was now referred to as "The Taxing District of Shelby County." Memphis would not be re-chartered as a city until 1891. While yellow fever was probably the single most important precipitating event in the city's fall into bankruptcy (Keating 1879, pp. 100-02), it is important to put this in a broader context. Other cities typically did not become financially insolvent as a result of yellow fever: why did Memphis? As early as 1870, Memphis was already having trouble meeting its obligations and it had to issue new debt in order to repay the old (BeJach 1948). Compounding these difficulties was population flight and plummeting real estate values. After the epidemics of 1873 and 1878, people fled the city *en masse*. On January 1, 1880, the population of the city formerly known as Memphis stood at 30,659, three-quarters of its population in 1870 (40,226).²⁵ Such flight must have resulted in a non-trivial reduction in the city's tax base.

Figure 20 depicts the cumulative effects of the epidemics of 1873 and 1878 on population growth in Memphis. Three series are plotted, one for observed population levels, another for predicted levels based on the rate of growth realized between 1850 and 1870, and a third based on observed population growth in a sample of 74 cities that in 1860 and 1870 resembled Memphis in terms of population levels and pre-1860 growth rates. The predicted

²⁵On the population of Memphis, see Carter *et al.* (2006a), p. 1-110, Series Aa854.

population levels indicate that Memphis was well below its expected population in 1880 by 22,000 to 25,000 people (38 to 41 percent below its expected level). However, the model based on pre- 1871 growth patterns in Memphis suggests that the city had recovered from the 1873 and 1878 epidemics by 1890. In contrast, the model based on the growth experience of similar cities suggests that Memphis did not recover until 1930. Although the city's growth rate is not radically different from the predicted rate, the observed population level appears 20 to 30 thousand people lower than it otherwise would have been throughout the late nineteenth and early twentieth century.²⁶

The yellow fever epidemics in Memphis also appear to have altered the relative attractiveness of the city among blacks and whites. In part because African Americans had an innate resistance to the disease, they were much less likely to flee the city during epidemics and much more likely to return once the outbreaks had subsided. There is evidence that following the 1878 epidemic the proportion of the Memphis population that was black rose relative to the levels observed in other Tennessee cities. This can be seen in figure 21, which plots the black population share in Memphis and the average black-population share in three other Tennessee cities, Chattanooga, Knoxville, and Nashville. In Memphis, the black-population share rose from 38 percent in 1870 to 44 percent 1880; the average black population share in other Tennessee cities also rose but not as quickly. Moreover, after 1880, the black

²⁶Capers (1939), pp. 75-89, argues that based on Memphis' advantageous geography, particularly its location on the Mississippi River, it would have experienced rapid economic growth in the absence of the 1878 epidemic. Wrenn (1987) is deeply skeptical of this view, arguing that many other cities also experienced severe yellow fever epidemics but suffered no lasting impact from the disease.

population share in Memphis continued to rise and by 1900 stood at 49 percent, while the average black population in other Tennessee cities fell by 2 percentage points, from 36 to 34 percent. These patterns were reflected in the desegregation of the Memphis police force. Prior to the 1878 epidemic, Memphis like all other Southern cities, did not hire black policemen. A desirable and well-paying job by the standards of the day, it was reserved for whites. But in the midst of the yellow fever epidemic, the city ran short of healthy white men and were forced to turn to African Americans. Memphis remained one of the few Southern cities to use black police officers throughout the late nineteenth century (Rousey 1985).

7. The 'Filthiest, Most Deathly Appearing Town' in the Union?

The results in figure 20 suggest that two years after the epidemic the population of Memphis was 40 percent below what it would have been. In the longer term, there is mixed evidence that the 1878 epidemic induced a permanent, one-time reduction in the city's population on the order of 20 to 30 thousand persons. Relative to the other cities considered here, this is clearly an unusual result. Nor is there evidence that in the other cities and areas considered above that black in-migration rose relative to white, as figure 21 suggests was the case for Memphis.

There are two broad classes of hypotheses that might explain the unique effects of the 1878 epidemic: perhaps the epidemic itself was different from prior outbreaks; or perhaps the City of Memphis differed from other cities stricken by yellow fever. There is something to both lines of thought. On the one hand, the 1878 epidemic was different. In contrast to previous yellow fever epidemics, which were localized and typically affected no more than one or two

major cities, the 1878 epidemic affected the entire Mississippi valley— Mississippi, Alabama, Tennessee, Louisiana, Kentucky, Arkansas, Missouri, and Ohio all felt its effects. Because the effects of the 1878 epidemic affected a much broader geographic area than prior outbreaks of yellow fever, it is possible that Memphis and other cities hit particularly hard would have had a harder time attracting new migrants to replace those who perished.

On the other hand, there is much anecdotal evidence to suggest that Memphis differed from other cities in terms of its reputation as an unhealthy place, particularly regarding its vulnerability to yellow fever. Perhaps the 1873 and 1878 epidemics cemented this reputation into the minds of potential migrants. In addition, the change in the racial composition of Memphis documented in figure 21 is consistent with a reputation-based explanation. If whites, who were more vulnerable to yellow fever, genuinely believed that they were at greater risk in Memphis they would have avoided the place; because blacks enjoyed greater immunity to yellow fever, they would have been more willing to live in Memphis. It is also possible that Memphis differed from other cities in how it responded to the epidemic. As explained below, there is some limited evidence to suggest that ill-advised policy responses to epidemics might have had lasting and adverse effects on growth.

Although it is difficult to construct a single clean test that could discriminate among these two classes of explanations, it is possible to expressly test the first hypothesis that the non-localized nature of the 1878 epidemic accounts for the unusual and comparatively severe effects observed in Memphis. If this hypothesis is correct, other places that were afflicted by yellow fever in 1878 would have exhibited changes in population growth and racial

composition similar to what was as observed in Memphis. To test this, county- and city-level data from Mississippi are used in regressions predicting the population growth rate between 1870 and 1880. The predictors are the death rate from yellow fever in 1878 and population in 1860 and/or 1870. As above, the estimating strategy will employ a modified version of the standard growth model. The samples used in the estimation include 38 cities and towns, and 52 counties with time-consistent boundaries. The 1878 epidemic seriously affected twelve of the cities in this sample, located in five separate Mississippi counties. As shown in table 5, the death rates observed in the worst-hit of these twelve cities rivaled, or exceeded, those observed in Memphis. In Greenville, for example, there were 338 deaths per 1,000 persons, while in Grenada, Holly Springs, and Hernando there were 178, 123, and 110 deaths per 1,000 persons, respectively. Despite losing one-third of its population to yellow fever in 1878, Greenville's population more than doubled between 1870 and 1880, while Grenada grew modestly and Holly Springs and Hernando declined.²⁷

Reported in table 6, the results offer, at best, a modicum of support for the hypothesis that the 1878 epidemic affected Memphis differently because this particular epidemic was much less localized than prior outbreaks. For the sample of 38 Mississippi towns, neither regression (1) nor (2) indicates that the severity of the 1878 yellow fever epidemic (as measured by the death rate) adversely affected population growth. In both specifications, the coefficient on the yellow fever variable is positive (the wrong sign), close to zero, and statistically

²⁷Data on population are from the U.S. *Census*. Data on yellow fever in Mississippi in 1878 are from Keating (1879), pp. 92-97; Nuwer (1996), pp. 145-56; and Spinzig (1888), pp. 155-74.

insignificant. However, cities in Mississippi that were not affected by the 1878 epidemic were, on average, 46 percent the size of those that were. This suggests that non-yellow fever cities might have been growing slower than yellow fever cities before the epidemic, and that it would be desirable to construct a control group with historical growth rates more similar to the yellow fever cities. This is done with two crude matching exercises. In the first, all non-yellow fever cities that experienced negative population growth between 1870 and 1880 are dropped (five cities). In the second, all non-yellow fever cities with a population less than 377 are also dropped (another six observations). As shown in table 6, these matching exercises do little to improve the case that the 1878 epidemic undermined urban growth among this sample of towns and cities.

For the sample of 52 counties, the Census contains more detailed data on population by racial group and it is, therefore, possible to ask if yellow fever not only lead to depopulation but whether it also altered the racial composition of Mississippi counties. Table 7 reports descriptive statistics for this sample of counties. There is evidence that the counties most immune to yellow fever tended to be less populous and slower growing areas. In terms of the white population, non-yellow fever counties were about 20 percent smaller than yellow fever counties; in terms of black population, they were more than 60 percent smaller. As above, this suggests that the full contingent of non-yellow fever counties might not act as the best control group. To address this possibility, we pursue two matching procedures, excluding non-yellow fever counties that experienced negative growth between 1870 and 1880, and excluding non-yellow fever counties with populations below the minimum black or white population level

observed in yellow fever counties.

Table 8 reports the regression results. Here there is stronger evidence that the 1878 epidemic had repercussions beyond Memphis. For the white population, the full sample suggests that population growth in yellow fever counties was 9 percent lower than in non-yellow fever counties, though the result is statistically insignificant. The matched samples, suggest white population growth was 11 to 12 percent lower in yellow fever counties; these results are statistically significant at the 4 and 6 percent levels. For blacks, the estimated coefficients suggest reductions of population growth on the order of 7 to 9 percent in the matched samples, though the results are not statistically significant. While the relative sizes of the black and white coefficients are consistent with the hypothesis that yellow fever altered the racial composition of Mississippi counties, formal statistical tests fail to reject the hypothesis that population growth rates were the same for both groups in yellow fever counties.

Another way to explore why the experience of Memphis in 1878 differed so much from the experiences of other cities struck by yellow fever is to look at population growth in cities and towns that were similarly situated to Memphis—specifically, towns that were also located on the Mississippi River, or a major tributary, and were geographically close to Memphis. If Memphis was unable to recover because the 1878 epidemic affected the broader Mississippi Valley region, one should observe unusually slow population growth in similarly-situated neighboring towns. Alternatively, if the 1878 epidemic induced depopulation in Memphis because it established the city’s reputation as an unhealthy and dangerous place, one would observe unusually rapid growth in neighboring and similarly-situated towns. We are in the

midst of compiling a data set that will enable us to explore such changes in a comprehensive and systematic way, but that effort is not yet complete and at present all we can do is discuss the experiences of a handful of cities and towns.

Consistent with a reputation-based explanation, the following cities experienced unusually high rates of population growth after the 1878 epidemic: Little Rock, Arkansas; Pine Bluff, Arkansas; and Greenville, Mississippi. As table 9 shows, Little Rock's population expanded by 91 percent between 1880 and 1890, and 48 percent between 1890 and 1900; Pine Bluff's population grew by 54 percent between 1870 and 1880, and 211 percent between 1880 and 1890; and Greenville's population grew by 146 percent between 1870 and 1880, and by 204 percent between 1880 and 1890. All three cities might plausibly be considered geographic substitutes for Memphis. Greenville is located on the Mississippi River, 147 miles south of Memphis; Pine Bluff and Little Rock are located on the Arkansas River, a major tributary off the Mississippi, and by water, are about two hundred miles from Memphis. Moreover, although these cities were affected by the 1878 epidemic, there is no record of a serious epidemic of yellow fever erupting in any them prior to 1878 (Toner 1873).

8. Natchez, Mississippi

Juxtaposed to nineteenth-century Memphis, the most intriguing city in the Mississippi Valley is Natchez, Mississippi. Located on the Mississippi River about 300 miles south of Memphis and 150 miles north of New Orleans, Natchez is a convenient entrepot for river trade and transportation. As explained earlier, the population of Natchez grew at an exponential pace during the antebellum period, despite severe and repeated yellow epidemics. Moreover,

Natchez was not affected by the 1878 yellow fever epidemic; there is no record of a single individual in the city perishing as a result of the outbreak. Yet the population of Natchez fell sharply immediately following the 1878 epidemic and, much like Memphis, appears to have never fully recovered from that drop. As figure 22 shows, the population of Natchez fell by about 2,000 people, or 22 percent between 1870 and 1880. This drop was historically unprecedented. If one predicts the city's subsequent growth path, by either an exponential model based on population growth between 1810 and 1870 or a linear model based on growth between 1850 and 1870, it appears that that 2,000 person deficit was never recouped and might have even widened over time. Why would Natchez—a city that had survived the Civil War, repeated epidemics prior to 1878, and tornados and large fires that destroyed vast areas of the city—suddenly succumb to a broader regional epidemic that, at least in terms of death, bypassed the city entirely? Answering this question can help explain the experience of Memphis as well.

Existing explanations of the post-1880 stagnation of Natchez are not especially compelling. James (1968, pp. 160-62) attributes the economic stagnation of Natchez to an unusually closed political and economic culture that discouraged “ambitious entrepreneurs and yeoman” from locating in the city. According to James, this cultural hostility to change and openness emerged slowly over the course of the earlier nineteenth century. But it is not clear how such a cultural explanation could account for the sudden and discrete change in the city's growth trajectory observed after 1878. Rowland (1925, pp. 680-81) suggests a multitude of possible causes, including the removal of the state capital from neighboring Washington to

Jackson, the decline of cotton in the broader region, and the lingering effects of a tornado in 1840 which left the town in ruins. The central problem with Rowland's explanation is that he uses events that took place between 1800 and 1850 to account for a disruption that occurred thirty to fifty years later. More promising lines of inquiry could probably be found in the years immediately surrounding the shift in the city's growth profile. In particular, it is useful to consider the draconian steps Natchez officials took to prevent yellow fever from spreading to their city during the 1878 epidemic.

Cities throughout the American South imposed severe quarantine measures during the yellow fever epidemic of 1878, going so far as to block ordinary mail from entering their cities; fumigating all parcels entering their cities; passing laws that punished anyone who transported goods or people from an infected city with a sentence on a chain gang; stopping all railroad traffic into their cities; banning the importation of mattresses, jute bags, and woolen goods; and requiring all ships that were potential carriers of yellow fever to wait in harbors thirty days or more before docking at their ports. A newspaper editorial in Vicksburg, Mississippi recommended the death penalty for anyone violating quarantine laws. It was also not uncommon for ordinary citizens to organize shotgun posses to prevent strangers from entering their towns. However severe these measures might sound, the existing literature on yellow fever in the American South suggests the City of Natchez was renowned for the unusual strictness and efficacy of its quarantines; "closed up like an oyster" was the way one newspaper editor put it. Posting armed guards at its docks, it prevented all ships from landing on its shores and forbade any outside goods from entering the city. It simultaneously refused

to handle cotton (or any other commodity) from farmers in neighboring towns and counties who might have wished to export from the city's docks. The quarantine measures put in place by Natchez authorities also lasted longer than those imposed by other cities.²⁸

If the quarantine measures in Natchez were significantly more restrictive and well-enforced than measures in other cities this might explain its anomalous growth trajectory after 1878. Although we do not have any quantitative data that would allow us to assess the extent to which quarantine measures in Memphis were as severe as those observed in Natchez, there is qualitative evidence to suggest that in the wake of the 1878 epidemic Memphis officials imposed rules on cotton shipments into the city that merchants and farmers alike found costly and unreasonable.²⁹ Another possibility is that because Memphis stood at the center of 1878 epidemic, its trading partners and neighboring cities and towns viewed it as the primary source of the infection. As such, goods and people shipping out of Memphis might have been subject to much more severe quarantine measures than those arriving from places on the

²⁸See Bloom (1993), p. 142; Humphreys (1992), p. 137; and Board of Councilmen (1859), p. 26. Vicksburg reputedly engaged in quarantine measures that were as severe and stifling as those observed in Natchez. Vicksburg exhibited a population trajectory similar to that observed in Natchez, both before and after the 1878 epidemic. Many observers argued that quarantining trade, particularly as was done in Natchez and Vicksburg, was unjust and did little prevent the disease from invading the city that the quarantine was designed to protect. See, for example, Cartwright (1856) and Board of Councilmen (1859).

²⁹Memphis allowed baled cotton and cotton seed to enter the city, but prohibited loose lint, loose cotton, and seed cotton. Authorities were reportedly very aggressive in enforcing these prohibitions. See the following issues of the *New York Times*: September 17, 1879, p. 2; September 25, 1879, p. 1; September 26, 1879, p. 1; and October 11, 1879, p. 4. It also notable that prior to the 1878 epidemic, Memphis does not appear to have been particularly aggressive in using quarantines to forestall the disease. See Board of Councilmen (1859), p. 26, where individual testimony indicates that Memphis had "never" imposed a quarantine prior to 1869.

periphery of the epidemic. In light of Memphis's reputation as the "filthiest, "most deathly appearing" town in the United States, this would not have been an unreasonable response. If the disruptions in trade and economic activity that followed such quarantines were especially severe in Memphis, it also seems possible that those disruptions could have had persistent and lingering effects.

9. Yellow Fever, Quarantines, and Trade Flows

Historians of yellow fever tend to argue that the disease imposed enormous costs on regional economies (e.g., Bloom 1993, pp. 125-28; Carrigan 1961, pp. 405-18; Humphreys 1992, pp. 8, 13, 83-89). Aside from the costs associated with the loss of life, the single most important component of these losses was the disruption and diversion of trade as a result of quarantines. While the previous discussion of Natchez suggests quarantines might well have been costly, it is far from clear that their adverse effects were as pervasive and severe as the current literature suggests. There are, on the contrary, many reasons to believe that quarantines were not typically so effective. For example, state health board's often ordered cities and towns to impose quarantines, but lacked the power and authority to enforce such orders (Nuwer 1996, p. 47). When cities imposed their own quarantines, enforcement was costly and politicians beholden to local business interests were often in no hurry to have their deeds match their words of assurance to the general public (Nuwer 1996, pp. 42-45). There is also evidence to suggest that shippers and traders found low-cost ways of evading quarantines by misrepresenting themselves, their places of origin, or by altering the ports where they chose to dock (Board of Councilmen 1859, pp. 349-56). Moreover, evasive strategies might have

involved nothing more untoward than weighting until the winter or early spring (when yellow fever abated and quarantines were lifted) to transport non-perishable commodities like wool, cotton, and jute.

To assess the effects of yellow fever on trade, data for ports in and around nine major cities are collected. Originally published by the U.S. government in the *Annual Reports of Commerce and Navigation*, these data are from DeBow (1854, pp. 186-87) and Albion (1935, pp. 390-91). The data are annual and cover the period 1790 through 1860. Because of oddities in government reporting, the data on exports span the entire 1790-1860 period, or come very close to doing so, for all cities. The data on imports do not begin until around 1820 for all of the cities. The ports are in and around the following cities: Mobile, Alabama; Savannah, Georgia; New Orleans, Louisiana; Boston, Massachusetts; Baltimore, Maryland; New York, New York; Philadelphia, Pennsylvania. Over the 1790-1860 study period, there were 67 yellow fever epidemics in the cities considered here (Keating 1878, pp. 72-98; Toner 1873). The distribution across of epidemics across cities was as follows: Mobile, 4; Savannah, 2; New Orleans, 17; Boston, 6; Baltimore, 13; New York, 5, Philadelphia, 6; Charleston, 12; and Norfolk, 2.

With these data, a panel is created and variants of the following model are estimated:

$$(3) \quad Q_{it} = \alpha_i + \tau + \lambda_i + \delta Y_{it} + v_{it},$$

where, Q_{it} is the natural log of the value of imports or exports into or out of ports surrounding city i in year t in constant 1800 dollars; α_i is a vector of port dummies; τ is a vector year dummies; λ_i is a vector of port-specific time trends; Y_{it} is dummy variable equal to one if there was an epidemic of yellow fever in port i in year t , and zero otherwise; and v_{it} is a random

error term. Note that both exports and imports are considered in this framework. Yellow fever might have reduced imports because quarantines made it harder and more costly for foreign suppliers to ship their goods into the U.S., or it might have prompted them to ship elsewhere in the U.S. away from ports with yellow fever. Exports might have fallen with yellow fever epidemics for two reasons. First, foreign ports often imposed their own quarantines on goods originating from U.S. ports with or near yellow fever outbreaks. Second, as was the case in Natchez, local authorities simply might have sealed off their ports entirely, and prevented all trade whether it was coming or going.

The results are reported in table 10. Regarding exports, the coefficient on the yellow fever dummy is consistently positive, suggesting that epidemics might have been associated with unusually high levels of export activity. See regression (1). This result, however, is driven by atypical trade patterns in Mobile, Alabama, which are discussed in greater detail below. Once Mobile is dropped from the sample, or one includes city-specific polynomial time trends in the model, the evidence of a positive correlation is much reduced. See regressions (2) and (3). An objection to the analysis thus far is that the effects of epidemics might have had lagged effects. Although there is little qualitative evidence to sustain such claims, models with a lagged yellow-fever dummy are also estimated. Adding a lag of the yellow fever dummy suggests that yellow fever years were associated with high export activity, followed by a year of unusually low activity. See regression (4). Again, though, this appears to have been related to the atypical trade patterns in Mobile, and dropping that city from the analysis reduces (though it does not completely eliminate) the pattern. See regression (5). The results of

imports are very similar. The coefficient on the yellow fever dummy is consistently positive and insignificant, suggesting that yellow fever years were not associated with significant drops in foreign imports. See regressions (6) and (7). Adding a lag of the yellow fever dummy does not alter this conclusion. See regression (8).

One concern with these regressions is that they do not consider the longer term impact of yellow fever. If, for example, repeated yellow fever epidemics caused shippers to gradually move their business to other ports and trading centers that were not so unhealthy. Two sources of evidence suggest this is an unlikely scenario. First, as explained above, Newport, Rhode Island and Portland, Maine had reputations as among the most healthy port cities in the United States yet they never developed to be serious competitors with New York, Charleston, and New Orleans, three cities especially vulnerable to smallpox and yellow fever. Second, we can modify the panel data analysis above to explore the longer term effects of yellow fever on trade flows. To implement this modified approach, we estimate the following model:

$$(4) \quad Q_{it} = \alpha_i + \tau + \lambda + \epsilon_{it}.$$

This model differs from equation (3) in two ways. First, rather than estimating separate time trends for each city, it imposes a single overall trend that captures the long-term evolution in trade common to all cities. Second, the dummy variable for yellow fever is dropped. Once this model is estimated, the predicted trade value is subtracted from the observed value, and the residual is plotted over time.

Figures 23a-23i plot the residuals for the log of exports for each of the port cities. Residuals associated with yellow fever years are distinguished by a large X; residuals

associated with non-yellow fever years are denoted by small circles. These figures highlight the changes in trade not explained by a common time trend, year shocks common to all cities, static fixed effects unique to each city. Savannah and New Orleans are the only cities that exhibit patterns consistent with the hypothesis that yellow fever disrupted long-term trading relations. Two epidemics around 1820 are associated with a sharp change in trend; prior to 1820, exports in Savannah were rising relative to other port cities. After the epidemics, exports stagnated. In New Orleans, exports grew rapidly between 1800 and 1820, when yellow fever was relatively infrequent, but stagnated after 1840, when yellow fever epidemics became more frequent. Having said this, several other cities exhibit this pattern of stabilization after 1840, and in those cities the change cannot be attributed to yellow fever (see Savannah, Baltimore, Philadelphia, Charleston, and Norfolk).

By contrast, exports in Baltimore and Boston are relatively high during yellow fever years, and only begin to trend downward after the epidemics subside. The rise of New York, and the roughly simultaneous decline in Philadelphia, correspond to the completion of the Erie canal in 1825 and the subsequent development rail lines linking cities on the Erie canal to other interior markets (Albion 1939, p. 417). Similarly, the stagnation observed in Mobile after 1840 corresponds to the completion of rail lines in the South that allowed shippers to bypass Mobile and ship directly from New Orleans and Pensacola, Florida (Summersell 1949, p. 46-47).

Another possible objection to the analysis thus far is that it focuses on the antebellum period, ignoring the single greatest yellow epidemic in American history: the 1878 outbreak that struck Memphis, New Orleans, and cities and towns throughout the Mississippi Valley.

Citing estimates constructed during the nineteenth century, Bloom (1993, p. 279-80) argues that the costs of the epidemic totaled around \$200 million. Although these costs are not particularly large when considered in relation to the national economy — total GDP in 1878 was \$134 billion, suggesting that the epidemic cost less than 2/10 of 1 percent of total GDP — at a local or regional level such estimates could imply a high level of economic disruption.³⁰ Again citing historical estimates, Bloom claims that the City of New Orleans alone lost \$100 million from the epidemic, mainly in the form of lost and diverted trade.³¹ New Orleans, in other words, was said to have born one-half of the entire economic burden of the 1878 epidemic. Having said this, there are two reasons to question the estimates Bloom and other historians put forward. First, in the years surrounding, but not including, the 1878 epidemic total trade (exports plus imports) in New Orleans never exceeded \$100 million (United States, Bureau of Statistics 1893, pp. 76-79). Had the epidemic eradicated all trade in the year, which not even the most pessimistic observers believed, it could still not account for the entire estimated costs of the outbreak. Second, the econometric work above produced no evidence that prior epidemics adversely affected trade, and it is not immediately obvious why the 1878 epidemic would have been so different.

To assess the effects of the 1878 epidemic on trade in New Orleans, variants of the following equation are estimated:

$$(5) \quad N_t = \alpha + \beta US_t + \gamma Y_t + \tau T + e_t$$

³⁰For GDP data, see Carter *et al.* (2006b), p. 3-24, Series Ca10.

³¹Carrigan (1961, p. 403) offers the identical statistic.

where N_t is the natural log of exports or imports leaving or entering the Port of New Orleans in year t ; US_t is the natural log of exports or imports leaving or entering all ports in the United States except New Orleans in year t ; Y_t is a dummy variable that assumes a value of one for the year of the epidemic and zero otherwise; T is a time trend; and e_t is a random error term.

Export and import data cover the period 1867 through 1893; have been collected from the United States *Statistical Abstract* for the year 1893; and are in constant 1882 dollars. All models have been estimated with Newey-West standard errors, adjusted for heteroscedasticity and autocorrelation with a 2 year lag. Standard interpretations regarding the economic effects of the 1878 epidemic on New Orleans suggest a negative coefficient on the yellow fever dummy that is large in absolute value and robust to a variety of reasonable econometric specifications.

The results are reported in table 11. The first regression suggests that after controlling for a linear time trend and changes in overall U.S. trade, the 1878 epidemic reduced exports out of the Port of New Orleans by 16 percent. If we allow trade to follow a non-linear time trend, stronger evidence of a negative effect emerges, with the coefficient on the epidemic dummy rising to -.179. (See regression 2.) If we include dummy variables for the years immediately preceding and following the epidemic, the epidemic appears to have reduced trade by about 14 percent. (See regressions 3 and 4.) Notice, however, that in the year preceding the 1878 epidemic, exports out of the Port of New Orleans were 10 to 18 percent above their predicted levels. The evidence of an effect on imports is much less robust. Although two specifications (regressions 6 and 8) yield estimates suggesting that the epidemic reduced imports into New Orleans by 33 to 35 percent, once we allow trade to follow a non-

linear time trend those estimates are reduced to 9 percent in one case and 0 in the other. (See regressions 7 and 9.) Certainly the estimates reported here suggest the 1878 epidemic had a non-trivial effect on trade out of, and perhaps into, the Port of New Orleans. Nevertheless a 14 percent reduction in exports does not support the claims of local businessmen and merchants in New Orleans that the epidemic had a calamitous effect on the city's economic life.

Figures 24 and 25 put these regression results in broader evolutionary context, and further highlight the modest nature of the effects of the 1878 epidemic. Figure 24 (25) plots the observed log of exports (imports) over time; the large X denotes the year of the epidemic. A trend line is also plotted in both figures. Trends are estimated with the following model:

$$(6) \quad N_t = \alpha + \beta US_t + \tau' T^3 + \tau'' T^4 + e_t.$$

The only differences between this equation and equation (5) above are that the epidemic dummy is dropped and trade is allowed to follow a non-linear time trend. The model is estimated for all years except those of the epidemic and the year immediately preceding and following the outbreak. For exports, the epidemic is associated with a trade level well off the predicted trend line; for imports, the observed trade level and predicted trade level are nearly identical. Moreover, notice that in neither figure is there evidence that the epidemic caused trade in New Orleans to begin trending downward, as has been suggested by some observers (Humphreys 1992, pp. 82-83). On the contrary, soon after the 1878 epidemic, imports began trending upwards, reversing a steady eight-year trend of decline.

10. Of Cash Constraints and Cotton

Quoting nineteenth-century business leaders, Carrigan (1961, pp. 408-11) argues that whenever yellow fever broke out businesses in New Orleans were cash constrained and in danger of defaulting on their loans and debt obligations. All merchants in New Orleans could do was hope that “northern creditors” would understand “the dreadful scourge paralyzing the arms of trade.” In the absence of such understanding, businesses in the city were doomed because their own customers had fled the city, making it impossible to collect on outstanding accounts or to make new sales. Consistent with this line of argument, there is anecdotal evidence that even merchants in cities largely untouched by yellow fever felt the financial pinch of the disease and fell into default as a consequence. In one case, the largest dry goods wholesaler in St. Louis, Missouri (who sold mainly to retailers in and around Memphis) had to suspend operations ostensibly because of the 1878 epidemic, though this enterprise’s financial troubles appear to have started many years earlier (*New York Times*, October 28, 1878, p. 1). A few months after the 1878 epidemic subsided, state legislators in Tennessee passed a law requiring all creditors to grant businesses in the throws of severe epidemics a grace period on their debt obligations.³² Similar legal institutions protected borrowers in New York City.³³ Likely a response to merchants and businessmen unable to raise funds in a time of crisis, the passage of such legislation is consistent with capital market imperfections and widespread

³²See *Lewis Hanauer v. J.A. Anderson et al.* 84 Tenn. 340 (1886), pp. 342-43, for a description of the legislation and the broader decision for the court’s interpretation.

³³See *Tunno and Cox v. Lague*, 2 Johns. Cas. 1 (1800).

liquidity constraints.

Nevertheless, there is also evidence to suggest that most businesses found a way to survive yellow fever epidemics, no matter how severe. Business leaders in Memphis reported that while in the midst of the 1878 epidemic sales dropped to almost nothing, once winter returned and the yellow fever subsided, the population came back to the city. Within a few months time, businesses were reporting sales sufficient to recoup all of their losses from the previous summer and fall (*New York Times*, March 19, 1879, p. 2). There is also anecdotal evidence that creditors granted borrowers grace periods and a limited amount of debt forgiveness if the debtor found himself cash constrained because of a yellow fever epidemic.³⁴ Furthermore, while laws like those observed in Tennessee and New York are consistent with the hypothesis that businesses were constrained in the midst of an epidemic, they are equally consistent with the hypothesis that well-devised institutions mitigated the adverse financial effects of epidemics.³⁵ Finally, it is notable that for all the yellow fever epidemics discussed above the extant historical record contains no evidence of financial or banking panics in any form.

³⁴See, for example, *W.K. Henderson Iron Works & Supply Co. v. Howard*, 119 La. 555 (1907).

³⁵In general, courts throughout the United States showed a great deal sympathy towards business enterprises strapped for cash because of yellow fever, and devised legal rules to protect businesses in such contexts, particularly regarding their rights to recover damages from insurance companies. See *Maggrath and Higgins v. Church* 1 Cai. R. 196 (1803, New York); *LeRoy, Bayard and M'evors v. Gouverneur* 1 Johns Cas. 226 (1800, New York); *Littell v. Zuntz* 2 Ala. 256 (1841); and *Traylor & Co. v. Hughes*, 88 Ala. 617 (1889). On the other hand, see *Thomas Ridge v. Scottish Commercial Insurance Company*, 77 Tenn. 507 (1882), holding that a manufacturer could not recover losses from an insurance company when its factory burned down in the midst of a yellow fever outbreak.

Data on business failures can bring the broader economic repercussions of the 1878 epidemic into sharper focus. Figure 25 plots the number of businesses failing in the United States from 1866 through 1881. Although business failures peak in 1879, on the heels of the epidemic, that peak did not represent a significant deviation from the prior historical trend, which was upward. Figure 26 plots business failure rates in four regions: the Northeast; the South; the Middle States; and the West. Although both the Northeast and the South show a sharp upturn in the failure rate in 1878, that change was no greater in the South than in the Northeast, even though the latter was not affected by epidemic. There is stronger evidence of an effect if one considers the business failure rate in Tennessee relative to the failure rate in other Southern states. As shown in figure 26, during the epidemic the ratio of Tennessee's business failure rate to the rate in other Southern states rose from .09 to .16, an increase of nearly 80 percent over the prior year's ratio. Having said this, .16 is not much higher than the rates observed in 1876, 1880, and 1881, which varied between .12 and .15. Put another way, 194 Tennessee businesses failed in 1878, and 152 failed in 1879. By contrast, 158 businesses failed in 1876, while 187 failed in 1882. These numbers, while not definitive, contrast sharply with the prevailing historical wisdom, and suggest that accounts of widespread business failure and distress as a result of the 1878 epidemic are probably overstated.

The historical literature suggests that the 1878 epidemic, as well as other yellow fever outbreaks, caused serious disruptions in the American cotton market.³⁶ There are good reasons

³⁶For example, describing a yellow fever epidemic during the 1850s, Carrigan (1961, p. 407) writes: "[T]he dreadful epidemic had completely deranged all business operations. Little produce of any kind was sent to New Orleans from the interior. Shipments of cotton were

to think that an epidemic as widespread and severe as the 1878 outbreak of yellow fever could have had such effects. There were reports that as people fled infected areas, no one was left to pick the cotton and it was left to rot on the plant or in storage (e.g, Bloom 1993, pp. 137-38; Nuwer 1996, p. 168).³⁷ In addition, the three states most affected by the epidemic— Louisiana, Mississippi, and Tennessee—produced roughly one-quarter of the country’s cotton and ports in these states, particularly Memphis and New Orleans, served as clearinghouses for the Southern cotton crop. Figure 27, however, plots cotton production and prices in United States from 1866 through 1886 and reveals little evidence of serious disruptions. On the contrary, the years the epidemic most plausibly affected, 1878 and 1879, are associated with unusually low cotton prices and atypically high levels of production for this twenty year period. These data comport well reports in the popular press which tend to indicate that the epidemic had little immediate or lasting impact on the cotton market to any significant degree.³⁸

halted because of sickness in the port city and the river towns.”

³⁷The following report from the *New York Times* (October 2, 1878, p. 5) is representative: “The cotton remains unpicked in the fields. As an instance of this may be mentioned the fact that Joseph B. Ferguson, a large farmer three miles from [Vicksburg, Mississippi], usually pressing 200 bales of cotton, says there are 75 cases of fever on his and adjoining plantations, and he has not more than 1,000 pounds of cotton picked.”

³⁸A report in the *New York Times* (September 1, 1878, p. 1) two months after the fever began stated that cotton receipts at the Memphis exchange were up from the prior year. Reports in early 1879, indicate lower than usual cotton prices and average production levels. See *New York Times*, May 24, 1879, p. 3.

11. Conclusions

Based on the experience of American cities, it is difficult to argue that a bioterrorist attack would have serious economic repercussions in either the short or long term. The largest and fastest growing cities in early America—places like New York, Philadelphia, New Orleans, Charleston, and Boston—were forged in an era when epidemics of smallpox and yellow fever routinely killed 10 to 25 of the local population. Such carnage, however, did little to slow growth in these areas. Moreover, when the epidemics subsided during the late nineteenth century, there is no evidence that population growth increased. Nor did discoveries like the smallpox vaccine or the revelation that yellow fever was spread by the mosquito have an effect on urbanization rates in areas most vulnerable to these diseases. Other natural disasters, such as the hurricane that struck Galveston in 1900, appear to have had much more severe and lasting effects. Nor is there evidence that in smaller and slower growing cities—places like Alexandria, Baton Rouge, and Shreveport, Louisiana— that yellow fever epidemics undermined population growth.

The only places considered here where yellow fever appears to have had significant negative effects on long term growth were Memphis, Tennessee and Natchez, Mississippi. These adverse effects stemmed from three sources: reputation—the 1878 epidemic confirmed to all observers that Memphis truly was the “filthiest, most deathly appearing town in the Union”; from draconian quarantine policies that permanently disrupted established trading relationships; and the broad regional nature of the 1878 outbreak. Having said this, even an epidemic like that which struck Memphis and the broader Mississippi Valley in 1878 did little

to undermine economic performance, as measured by cotton production and business failures, in the American South or the country as whole. To the extent the epidemic disrupted trade in New Orleans those effects were fleeting and not nearly as severe as many previous historical commentators have suggested — the estimates above suggest the outbreak reduced exports from the Port of New Orleans by 14 to 18 percent during the year of epidemic. Furthermore, previous epidemics in New Orleans and elsewhere appear to have had almost no effect on trade.

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Table 1. Yellow Fever in the United States

| State | Frequency of epidemics | | Intrastate concentration | | | |
|----------------|------------------------|------------------|--------------------------|---------------|------------------|------------------|
| | No. | % of U.S. total* | No. of cities struck | Worst city | No. of epidemics | % of state total |
| Alabama | 45 | 6 | 11 | Mobile | 28 | 62 |
| Arkansas | 6 | 1 | 6 | — | — | — |
| Connecticut | 15 | 2 | 9 | New Haven | 6 | 40 |
| Delaware | 5 | 1 | 4 | Wilmington | 2 | 40 |
| Florida | 56 | 8 | 13 | Pensacola | 22 | 39 |
| Georgia | 13 | 2 | 4 | Savannah | 9 | 69 |
| Illinois | 1 | 0 | 1 | Cairo | 1 | 100 |
| Louisiana | 166 | 23 | 43 | New Orleans | 66 | 40 |
| Maryland | 15 | 2 | 2 | Baltimore | 14 | 93 |
| Massachusetts | 16 | 2 | 6 | Boston | 10 | 63 |
| Mississippi | 55 | 8 | 20 | Natchez | 13 | 24 |
| Missouri | 3 | 0 | 2 | Saint Louis | 2 | 66 |
| New Hampshire | 1 | 0 | 1 | Portsmouth | 1 | 100 |
| New Jersey | 5 | 1 | 5 | — | — | — |
| New York | 84 | 12 | 17 | New York City | 62 | 74 |
| North Carolina | 11 | 2 | 5 | Wilmington | 4 | 18 |
| Ohio | 3 | 0 | 2 | Cincinnati | 2 | 66 |
| Pennsylvania | 43 | 6 | 9 | Philadelphia | 34 | 79 |
| Rhode Island | 12 | 2 | 5 | Providence | 5 | 42 |
| South Carolina | 64 | 9 | 7 | Charleston | 52 | 81 |
| Tennessee | 4 | 1 | 1 | Memphis | 4 | 100 |
| Texas | 72 | 10 | 41 | Galveston | 10 | 14 |
| Virginia | 28 | 4 | 10 | Norfolk | 18 | 64 |

* U.S. total is 723 epidemics from 1668 through 1873. *Source:* Toner (1873), pp. 18-36.

Table 2. Yellow Fever in Three Small Louisiana Cities

| Year | Alexandria | | Baton Rouge | | Shreveport | |
|--|------------|----------|--------------|----------|-------------|----------|
| | Population | % change | Population | % change | Population | % change |
| 1840 | — | — | 2,269 | — | — | — |
| 1850 | 672 | — | 3,905 | 72.1 | 1,728 | — |
| 1860 | 1,461 | 117.4 | 5,428 | 39.0 | 2,190 | 26.7 |
| 1870 | 1,218 | (16.6) | 6,498 | 19.7 | 4,607 | 110.4 |
| Proportion of the local population killed in the yellow fever epidemic | | | | | | |
| 1853 | 1/5 to 1/6 | | roughly 1/20 | | roughly 1/4 | |

Sources: see text.

Table 3. Louisiana Counties, 1850-60: Descriptive Statistics

| Variable | $Y_i = 1$ | | $Y_i = 0$ | |
|--|-----------|----------|-----------|----------|
| | μ | σ | μ | σ |
| ΔP_i : $\ln(\text{Pop1860}) - \ln(\text{Pop1850})$ | 0.197 | 0.324 | 0.343 | 0.249 |
| P_i : $\ln(\text{Pop1850})$ | 9.468 | 0.365 | 8.747 | 0.427 |
| L_i : Pop1850 in thousands | 13.783 | 5.353 | 6.850 | 2.779 |
| No. of obs. | 12 | | 34 | |

Table 4. Louisiana Counties, 1850-60: Determinants of Population Growth (ΔP_i)

| Variable | Full sample | | Sample A | | Sample B | |
|------------|------------------------------|------------------------------|------------------------------|------------------------------|------------------------------|------------------------------|
| | (1) | (2) | (3) | (4) | (5) | (6) |
| P_i | -.202 ^a (.095) | ... | -.216 ^a (.089) | ... | -.664 ^b (.146) | ... |
| L_i | ... | -.031 ^b (.010) | ... | -.033 ^b (.001) | ... | -.046 ^a (.011) |
| Y_i | -.001 (.111) | .069 (.110) | -.020 (.104) | .052 (.102) | .043 (.088) | .043 (.089) |
| Constant | 2.223 ^b (.831) | .556 ^b (.083) | 2.263 ^b (.780) | .601 ^b (.078) | 6.443 ^b (1.34) | .793 (.121) |
| Adj. R^2 | .107 | .181 | .164 | .258 | .491 | .478 |
| N | 46 | 46 | 44 | 44 | 23 | 23 |

Sample A excludes all counties not experiencing a yellow fever epidemic between 1850 and 1860, and experiencing negative population growth over the same period.

Sample B excludes all counties not experiencing a yellow fever epidemic during the decade and experiencing negative population growth over the decade, or which had a population in 1850 less than 7,758 (the minimum observed population level for counties experiencing a yellow fever epidemic).

^a - significant at the .05 level or higher (two-tailed test).

^b - significant at the .01 level or higher (two-tailed test).

Sources: see text.

Table 5. Yellow Fever and Population Growth in Mississippi, 1870-80

| Town | Yellow fever* | Population | |
|---|---------------|------------|--------|
| | | 1880 | 1870 |
| Passchrist | 11.8 | 1,410 | 1,951 |
| Carrollton | 13.3 | 394 | 377 |
| Jackson | 20.3 | 5,204 | 4,261 |
| Meridian | 33.6 | 9,110 | 2,709 |
| Biloxi | 47.2 | 1,540 | 954 |
| Canton | 91.7 | 2,083 | 1,963 |
| Vicksburg | 92.3 | 11,814 | 12,443 |
| Hernando | 109.6 | 583 | 730 |
| Holly Springs | 128.4 | 2,370 | 2,406 |
| Grenada | 172.8 | 1,914 | 1,887 |
| Greenville | 338.2 | 2,191 | 890 |
| <i>Average: cities w/ yellow fever</i> | 96.3 | 3,510 | 2,777 |
| <i>Average: cities w/o yellow fever</i> | 00.0 | 1,322 | 1,207 |
| <i>% Change, 1870-80: cities w/ yellow fever</i> | — | | .264 |
| <i>% Change, 1870-80: cities w/o yellow fever</i> | — | | .096 |

* - deaths per 1,000 persons during the Mississippi Valley yellow fever epidemic of 1878.

Sources: see text.

Table 6. Mississippi Towns, 1870-80: Determinants of Population Growth (ΔP_i)

| Variable | Full sample | | Sample A | | Sample B | |
|------------|------------------------------|------------------------------|------------------------------|-----------------------------|------------------------------|-----------------|
| | (1) | (2) | (3) | (4) | (5) | (6) |
| P_i | -.145 ^a (.070) | ... | -.159 ^a (.073) | ... | -.137 (.103) | ... |
| L_i | ... | -.054 ^b (.030) | ... | -.052 (.034) | ... | -.041 (.038) |
| Y_i^* | .001 (.001) | .001 (.001) | .001 (.001) | .020 (.102) | .001 (.001) | .028 (.113) |
| Constant | 1.223 ^a (.472) | .350 ^b (.095) | 1.418 ^b (.478) | .448 ^b (.089) | 1.260 ^b (.708) | .773 (.108) |
| Adj. R^2 | .107 | .037 | .083 | .011 | -.008 | -.027 |
| N | 38 | 38 | 33 | 33 | 27 | 27 |

* - in the context of this table, Y_i equals the town's death rate from yellow fever during the 1878 epidemic, measured as deaths per 1,000 persons.

Sample A excludes all towns not afflicted by the 1878 yellow fever epidemic and experiencing negative population growth between 1870 and 1880.

Sample B excludes all towns not afflicted by the 1878 yellow fever epidemic and experiencing negative population growth between 1870 and 1880, or which had a population in 1870 less than 377 (the minimum observed population level for towns experiencing the 1878 epidemic).

^a - significant at the .05 level or higher (two-tailed test).

^b - significant at the .01 level or higher (two-tailed test).

Sources: see text.

Table 7. Mississippi Counties, 1870-80: Descriptive Statistics

| Variable | $Y_i = 1$ | | $Y_i = 0$ | |
|---|-----------|----------|-----------|----------|
| | μ | σ | μ | σ |
| <i>For black population:</i> | | | | |
| $\Delta P_i: \ln(\text{Pop1880}) - \ln(\text{Pop1870})$ | 0.275 | 0.176 | 0.351 | 0.218 |
| $P_i: \ln(\text{Pop1870})$ | 9.563 | 0.249 | 8.220 | 0.998 |
| $L_i: \text{Pop1870 in thousands}$ | 14.580 | 3.467 | 5.574 | 4.719 |
| <i>For white population:</i> | | | | |
| $\Delta P_i: \ln(\text{Pop1880}) - \ln(\text{Pop1870})$ | 0.113 | 0.235 | 0.241 | 0.171 |
| $P_i: \ln(\text{Pop1870})$ | 8.583 | 0.701 | 8.390 | 0.587 |
| $L_i: \text{Pop1870 in thousands}$ | 6.437 | 4.245 | 5.095 | 2.571 |
| No. of obs. | 5 | | 47 | |

Sources: see text.

**Table 8. Mississippi Counties, 1870-80:
Determinants of Black and White Population Growth (ΔP_i)**

| Variable | Full sample | | Sample A | | Sample B | |
|-------------------------------------|------------------------------|------------------------------|-----------------------------|------------------------------|-----------------|------------------------------|
| | Blacks | Whites | Blacks | Whites | Blacks | Whites |
| Population in 1870 (000s) | -.145 ^a (.070) | -.029 ^b (.001) | -.009 (.010) | -.026 ^b (.001) | -.004 (.020) | -.004 ^b (.001) |
| Population in 1860 (000s) | . . . | .001 (.002) | .005 (.012) | .003 (.002) | .001 (.001) | .006 (.020) |
| =1 if yellow fever struck county | .001 (.001) | -.088 (.078) | -.066 (.111) | -.114 ^c (.071) | -.086 (.111) | -.121 ^d (.064) |
| Constant | 1.223 ^a (.472) | .380 ^b (.050) | .388 ^b (.048) | .392 ^b (.046) | .260 (.221) | .479 ^b (.050) |
| Adj. R^2 | .107 | .164 | -.029 | .164 | -.167 | .198 |
| N | 38 | 52 | 52 | 52 | 52 | 52 |

Sample A excludes all counties not afflicted by the 1878 yellow fever epidemic and experiencing negative population growth between 1870 and 1880.

Sample B excludes all counties not afflicted by the 1878 yellow fever epidemic and experiencing negative population growth between 1870 and 1880, or which had a population in 1870 less than 377 (the minimum observed population level for towns experiencing the 1878 epidemic).

^a - significant at the .05 level or higher (two-tailed test).

^b - significant at the .01 level or higher (two-tailed test).

^c - significant at the .06 level or higher (one-tailed test).

^d - significant at the .04 level or higher (one-tailed test).

Sources: see text.

Table 9. Population Growth in Similarly-Situated Places

| Year | Greenville, Miss. | | Little Rock, Ark. | | Pine Bluff, Ark. | |
|------|-------------------|----------|-------------------|----------|------------------|----------|
| | Population | % Change | Population | % Change | Population | % Change |
| 1870 | 890 | — | 12,380 | — | 2,081 | — |
| 1880 | 2,191 | 146.2 | 13,522 | 9.2 | 3,203 | 53.9 |
| 1890 | 6,658 | 203.9 | 25,874 | 91.4 | 9,952 | 210.7 |
| 1900 | 7,642 | 14.8 | 38,307 | 48.1 | 11,496 | 15.5 |
| 1910 | 9,610 | 25.8 | 45,941 | 19.9 | 15,102 | 31.4 |

Sources: see text.

Table 10. Yellow Fever and Trade

| Variable | Ln(exports) | | | | Ln(imports) | | | |
|--|-----------------------------|----------------|----------------|-----------------------------|-----------------|----------------|----------------|----------------|
| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) |
| =1 if yellow fever in city i in year t (Y_{it}) | .127 ^a (.066) | .041 (.050) | .054 (.058) | .124 ^a (.069) | .051 (.060) | .067 (.071) | .002 (.070) | .069 (.072) |
| Yellow fever dummy lagged one year (Y_{it-1}) | ... | ... | ... | -.079 (.069) | -.020 (.059) | ... | ... | .027 (.055) |
| Year dummies | yes | yes | yes | yes | yes | yes | yes | yes |
| City fixed effects | yes | yes | yes | yes | yes | yes | yes | yes |
| City-specific time trends | yes | yes | yes | yes | yes | yes | yes | yes |
| City-specific polynomial ^b | no | yes | no | no | no | no | yes | no |
| Mobile excluded | no | no | yes | no | yes | no | no | no |
| Adjusted R^2 | .867 | .926 | .892 | .866 | .892 | .967 | .970 | .967 |
| No. of observations | 584 | 584 | 541 | 548 | 501 | 241 | 241 | 241 |

^a - significant at the .10 level or higher (two tailed test).

^b - for each city the following polynomial time trend is included: $\text{year} + \text{year}^2 + \text{year}^3$. This allows trade to evolve in a non-linear fashion and for that evolution to differ across each city.

Sources: see text.

Table 11. Yellow Fever and Trade in New Orleans, 1866-1893

| Variable | Ln(exports) | | | | Ln(imports) | | | |
|-------------------------------|------------------------------|------------------------------|------------------------------|------------------------------|------------------------------|-----------------------------|------------------------------|------------------------------|
| | (1) | (2) | (3) | (4) | (6) | (7) | (8) | (9) |
| =1 if year of epidemic | -.163 ^d (.060) | -.179 ^d (.053) | -.144 ^b (.064) | -.138 ^b (.061) | -.355 ^d (.082) | -.088 (.143) | -.333 ^d (.092) | .002 (.130) |
| =1 if year b4 epidemic | ... | ... | .185 ^c (.074) | .101 ^c (.070) | ... | ... | .130 ^c (.082) | .447 ^d (.124) |
| =1 if year after epidemic | ... | ... | .018 (.045) | .021 (.053) | ... | ... | -.330 ^c (.116) | -.180 ^b (.087) |
| Ln(U.S. exports) [†] | .333 ^d (.229) | .310 ^d (.210) | .399 ^a (.255) | .408 ^b (.239) | ... | ... | ... | ... |
| Ln(U.S. imports) [‡] | ... | ... | ... | ... | .970 ^b (.415) | 1.41 ^d (.410) | .399 ^a (.255) | 1.70 ^d (.319) |
| Linear time trend | yes | no | yes | no | yes | no | yes | no |
| Non-linear time trend* | no | yes | no | yes | no | yes | no | yes |
| F-statistic | 23.8 | 36.9 | 3.63 | 2.94 | 28.2 | 48.0 | 4.48 | 23.8 |
| No. of observations | 27 | 27 | 27 | 27 | 27 | 27 | 27 | 27 |

Notes:

^a - significant at the .10 level or higher (one tailed test); ^b - significant at the .05 level or higher (one-tailed test); ^c - significant at the .01 level or higher (one-tailed test); and ^d - significant at the .001 level or higher (one-tailed test).

^{†,‡} - U.S. exports and imports exclude New Orleans.

* - the non-linear time trend includes: year³ + year⁴. Year and year-squared are collinear and are dropped from the regression.

Sources: see text.

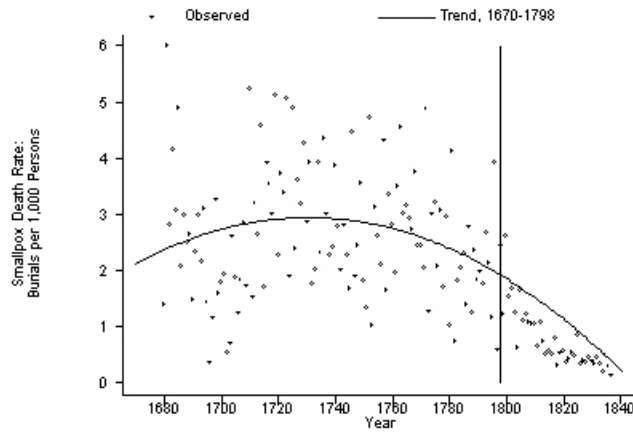


Figure 1. Smallpox in London: 1680-1840

Source: Vaccination Commission (1890), pp. 289-92.

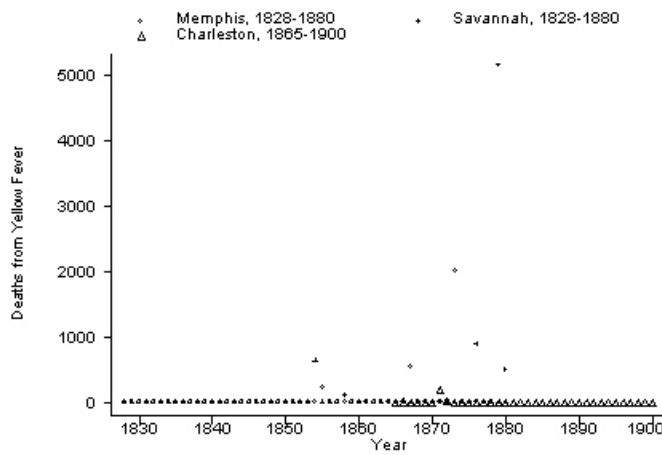


Figure 2. Yellow Fever in Memphis, Savannah, and Charleston

Sources: Keating (1878), pp. 77-98; Toner (1873); and Myers (1901), pp. 18-19.

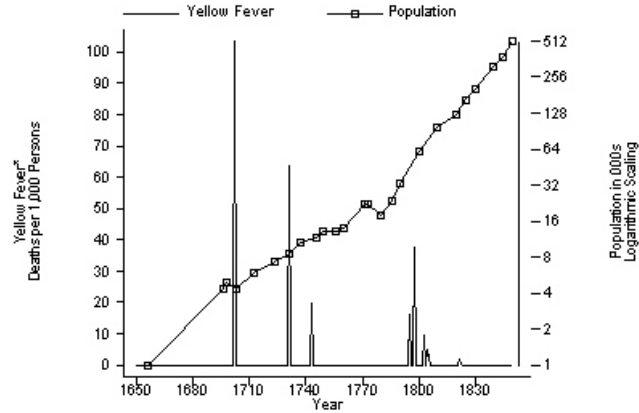


Figure 3. New York City: Yellow Fever and Growth, 1650-1840.

Note: In 1731 a smallpox epidemic struck the city, killing 549 residents; that epidemic is plotted (it appears as the second spike). The death rate from influenza during the great pandemic of 1918 was 6.79 per 1,000 persons.

Sources: Bridenbaugh (1968b), pp. 399-400; Duffy (1953), pp. 76-78, 81, 84-86, 88, and 91; Toner (1873), pp. 28-29; Griscom (1858), pp. 1-18; Rossiter (1970), p. 11; and Carter *et al.* (2006a), p. 1-110, Series Aa832.

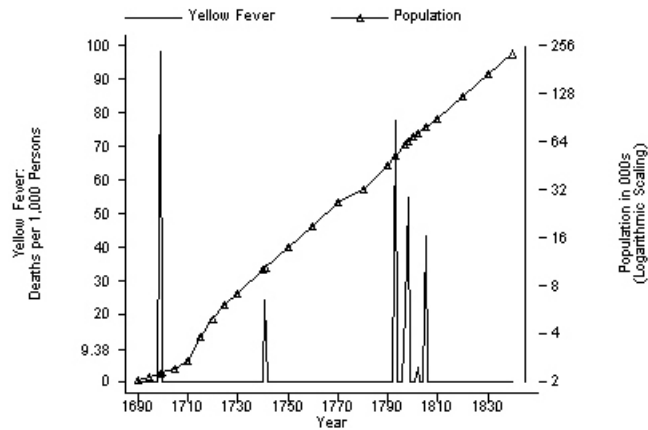


Figure 4. Philadelphia: Yellow Fever and Growth, 1690-1840

Sources: Griscom (1858), p. 2; Keating (1879), pp. 77-79; Duffy (1953), pp. 138, 142, 144-45, 151-54, and 158-62; Toner (1873), pp. 30-31; Klepp (1989); Smith (1977); and Carter *et al.* (2006a), p. 1-110, Series Aa841.

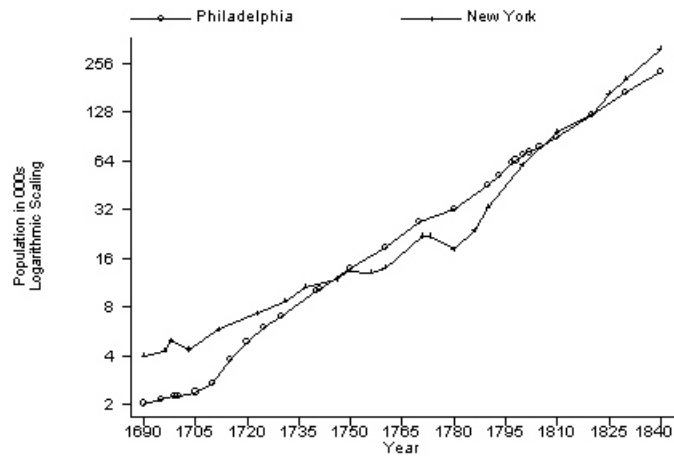


Figure 5. Population Growth in New York and Philadelphia

Sources: Klepp (1989); Smith (1977); and Carter *et al.* (2006a), p. 1-110, Series Aa841 and Series Aa832.; Bridenbaugh (1968b), pp. 399-400; Duffy (1953), pp. 76-78, 81, 84-86, 88, and 91; and Rossiter (1970), p. 11.

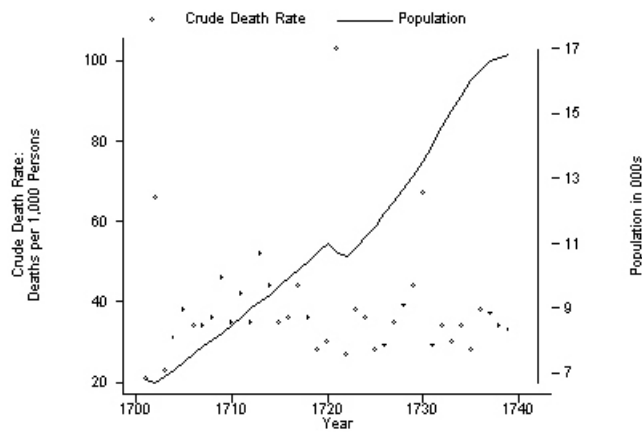


Figure 6. Boston: Smallpox and Growth, 1700-1740.

Sources: Blake (1959), pp. 247-49; and Rossiter (1970), p. 11.

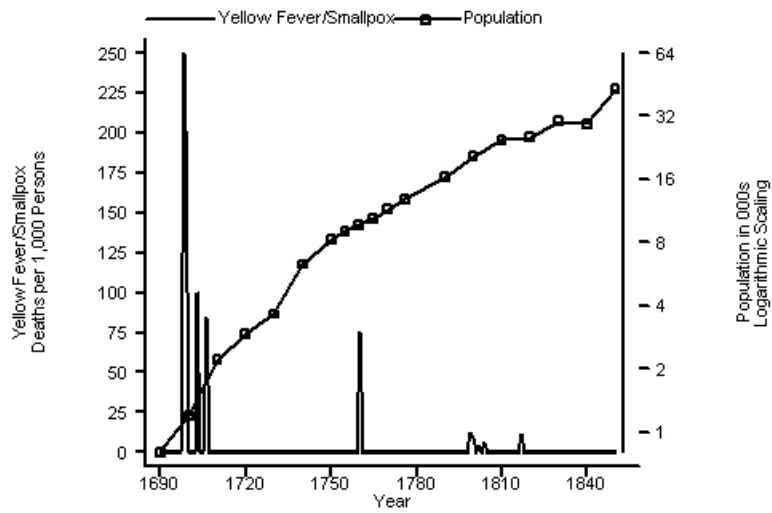


Figure 7. Charleston, S.C.: Yellow Fever, Smallpox, and Growth, 1690-1840

Sources: Bridenbaugh (1968b), p. 240; Duffy (1953), pp. 140-43; Toner (1873), pp. 31-33; Coclanis (1989), p. 114; Rossiter (1970), p. 11; and DeBow (1854), p. 192.

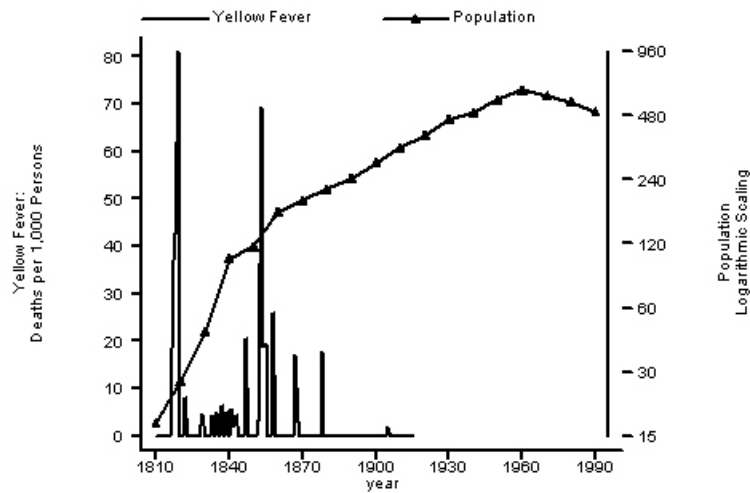


Figure 8. New Orleans: Yellow Fever and Growth, 1810-1990

Sources: Toner (1873), pp. 23-24; Keating (1879), pp. 78-98; Carrigan (1961), p. 223; Humphreys (1992), p. 4; and Carter *et al.* (2006a), p. 1-111, Series Aa 861.

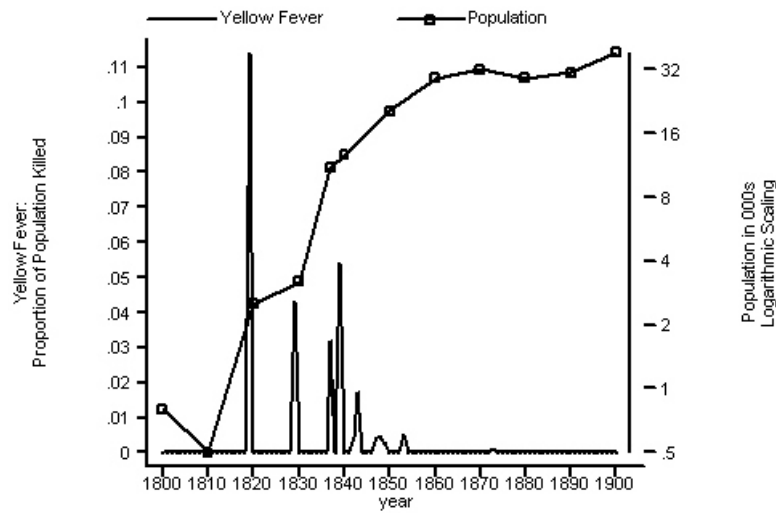


Figure 9.
Yellow Fever and Growth, 1800-1900

Mobile, Ala.:

Sources: DeBow (1854), p. 192; Amos (1985), pp. 1, and 11-12; Carter *et al.* (2006a), p. 1-113, series Aa917; Toner (1873), pp. 18-19.

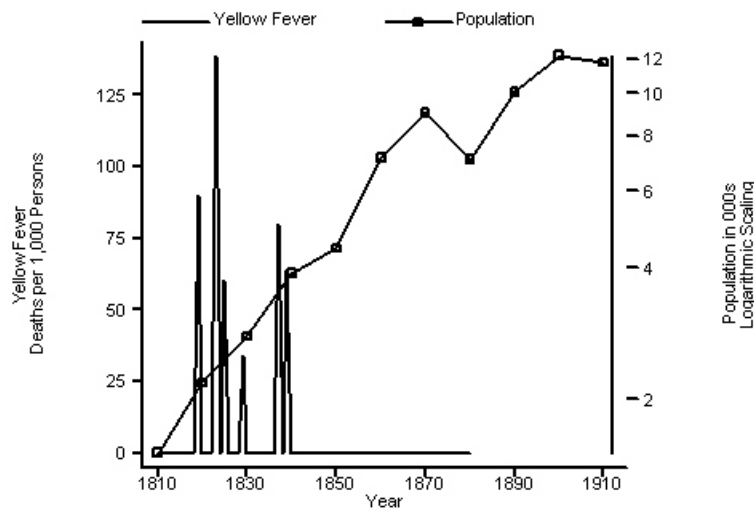


Figure 10. Natchez, Miss.: Yellow Fever and Growth.

Sources: DeBow (1854), p. 193; Darby and Dwight (1833), p. 332; various *Census* volumes; and Toner (1873), pp. 18-19.

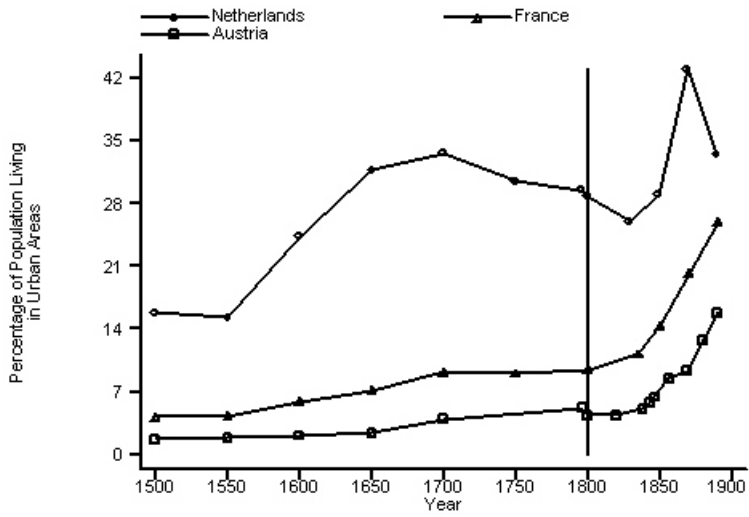


Figure 11. Urbanization in Western Europe, 1500-1900

Sources: DeVries (1983), p. 39; and Weber (1899), pp. 67-121.

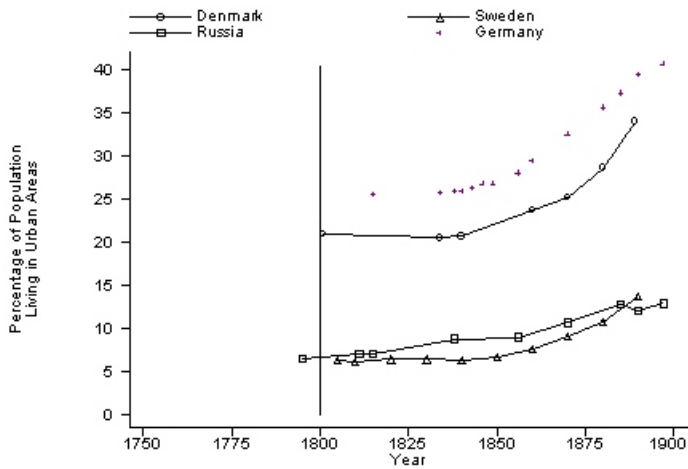


Figure 12. Urbanization in Scandinavia, Russia, and Germany, 1750-1900

Sources: DeVries (1983), p. 39; and Weber (1899), pp. 67-121.

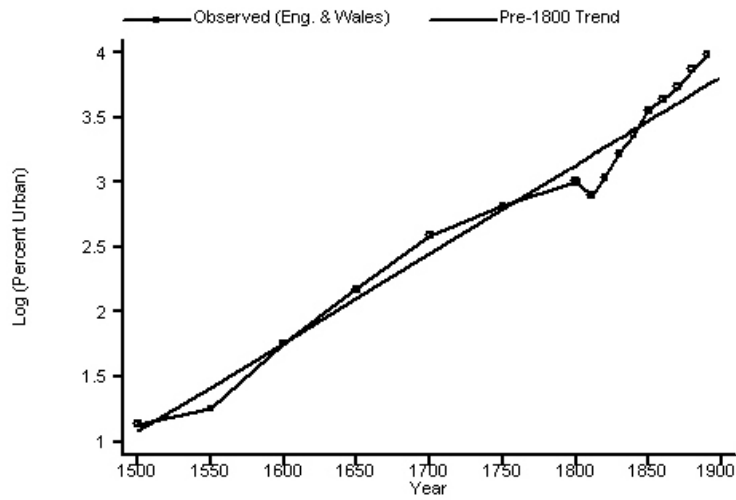


Figure 13. Urbanization in England and Wales, 1500-1900

Sources: DeVries (1984), p. 39; Weber (1899), pp. 67-121.

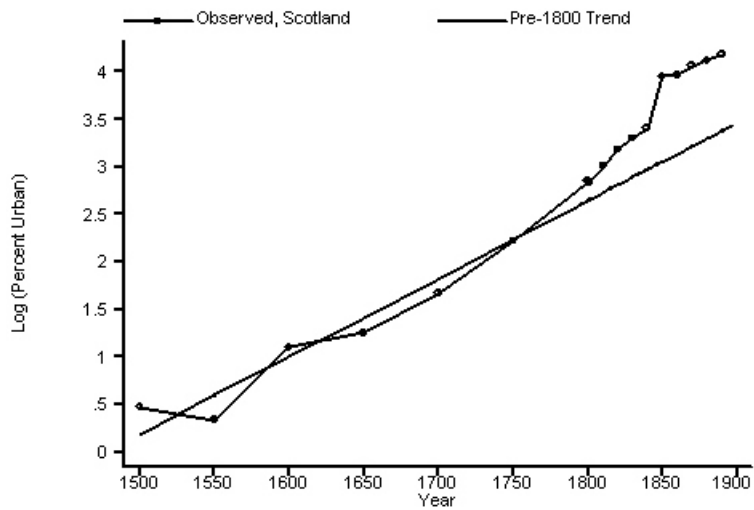


Figure 14. Urbanization in Scotland, 1500-1900.

Sources: DeVries (1983), p. 39; and Weber (1899), pp. 67-121.

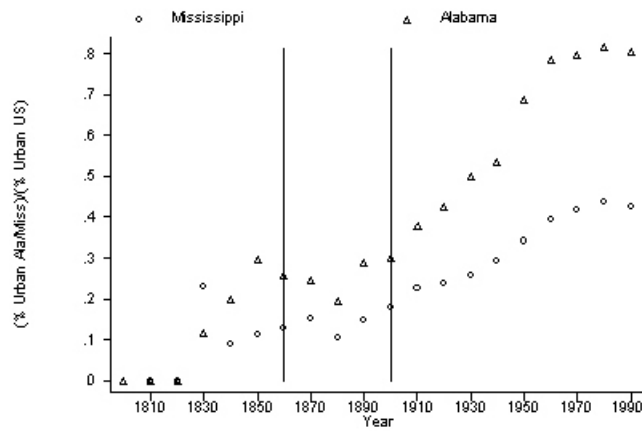


Figure 15. Relation Urbanization in Alabama and Mississippi

Sources: Carter *et al.* (2006a), p. 1-180, Series Aa2246 and Aa2251; and p. 1-276, Series Aa4307 and Aa4312

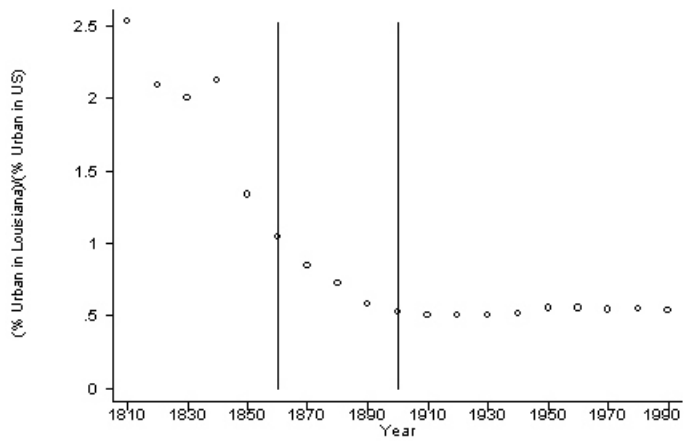


Figure 16. Relative Urbanization in Louisiana

Sources: Carter *et al.* (2006a), p. 1-249, Series Aa 3747 and Aa3752.

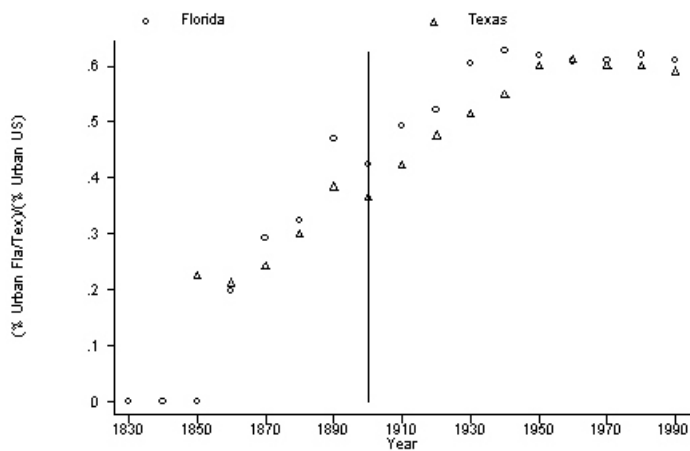


Figure 17. Relation Urbanization in Florida and Texas

Sources: Carter *et al.* (2006a), p. 1-213, Series Aa3012 and Aa3017; and p. 1-348, Series Aa 5874 and Aa 5879.

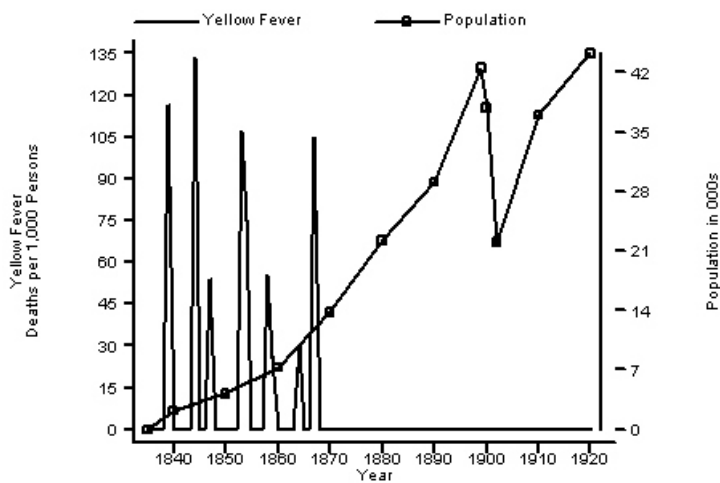


Figure 18. Yellow Fever, Hurricanes and Growth: Galveston

Sources: Toner (1873), pp. 34-35; DeBow (1854), p. 193; James (1908); and various *Census* volumes.

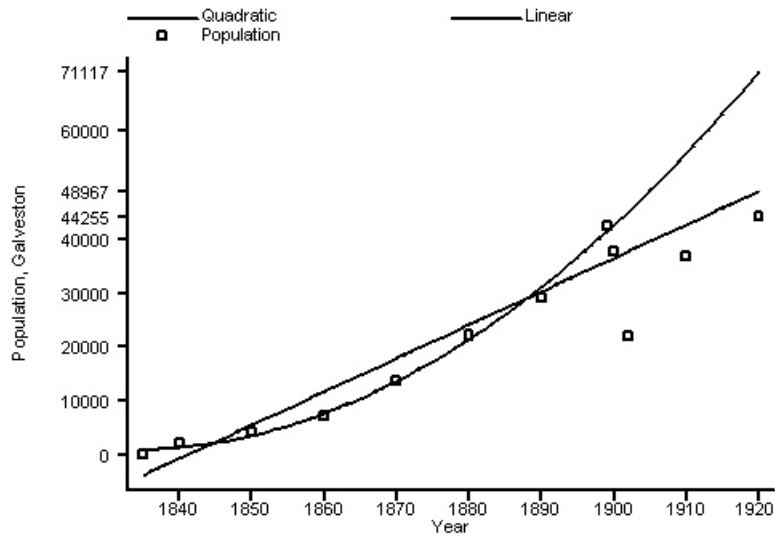


Figure 19. Population Growth in Galveston Following the Hurricane

Note: For the linear trend, population is regressed against year for all years preceding 1900. The results from that regression are:

$$Population_t = -1141.6 + .62 \times Year$$

t-statistic (8.83) (8.95)
Adjusted - $R^2 = .919$; No. of obs. = 8

For the quadratic trend, population is regressed against year for all years preceding 1900. The results from that regression are:

$$Population_t = 30998.5 - 33.8 \times Year + .0092 \times Year^2$$

t-statistic (7.61) (7.75) (7.89)
Adjusted - $R^2 = .993$; No. of obs. = 8

From both the figure and the adjusted R -squareds, it is clear that the quadratic model fits the pre-1900 data much better than the linear model.

Source: see text.

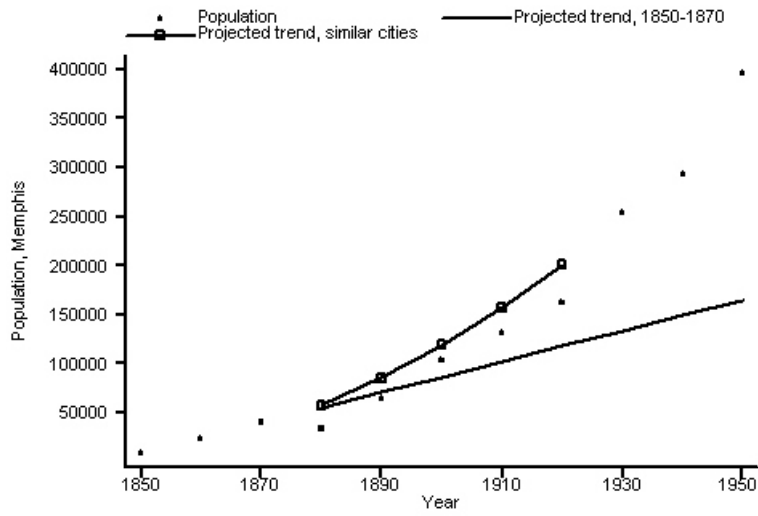


Figure 20. Population Growth in Memphis

Source: see text.

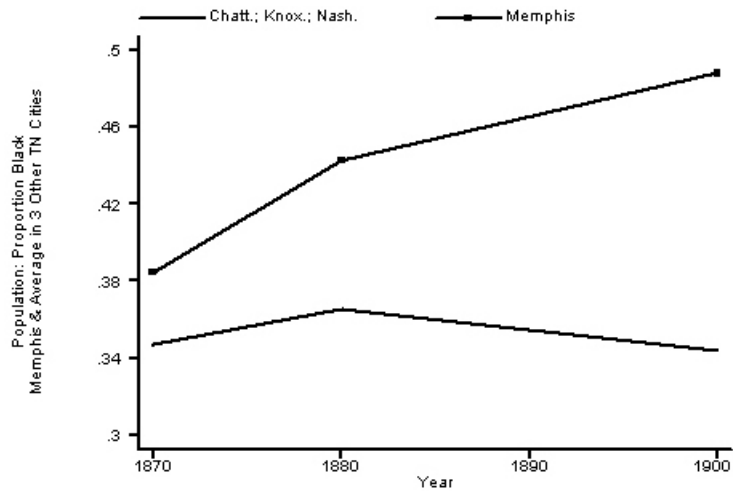


Figure 21. Racial Composition of Memphis After Yellow Fever

Source: see text.

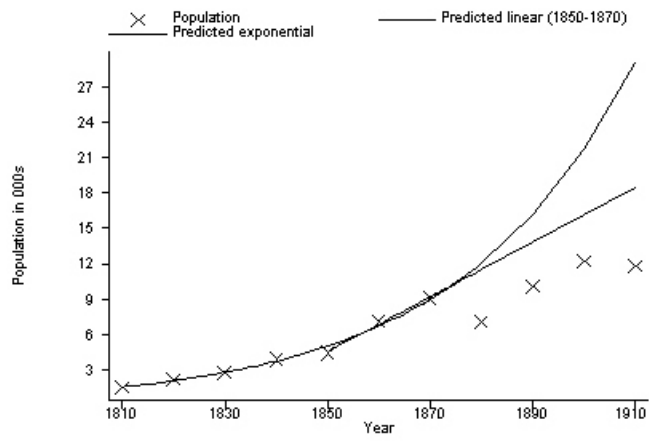


Figure 22. Population Growth in Natchez: Observed and Predicted

Source: see text.

Figures 23a through 23i

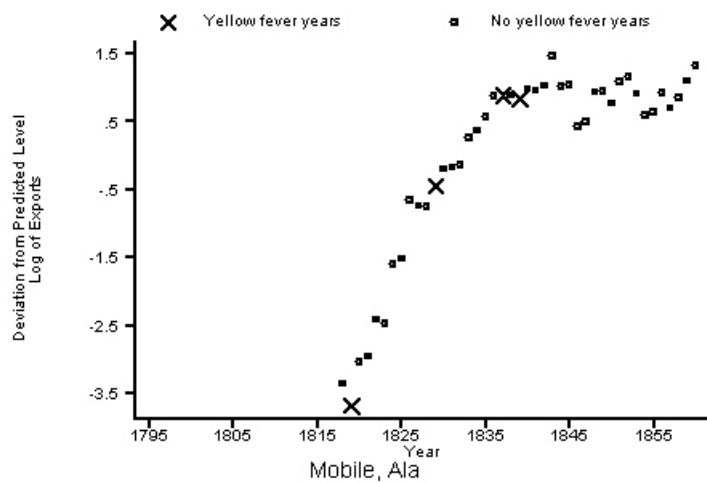


Figure 23a

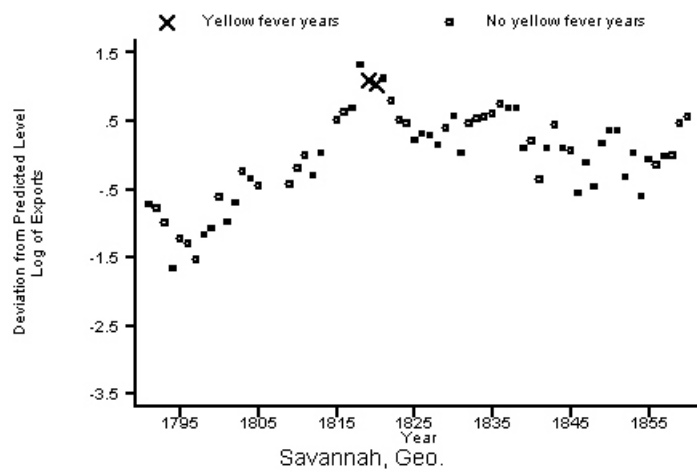


Figure 23b

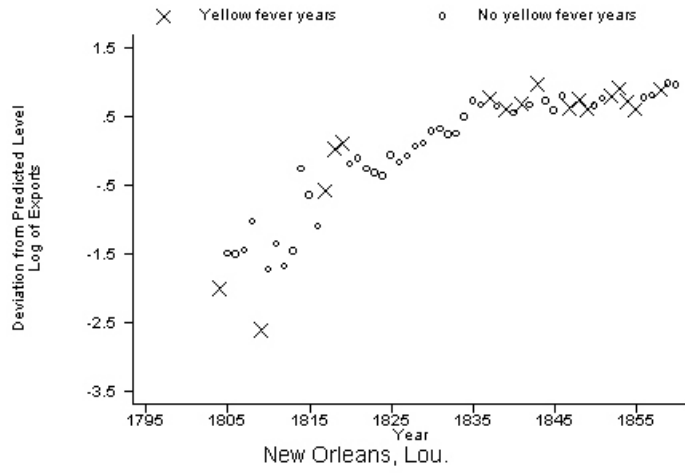


Figure 23c

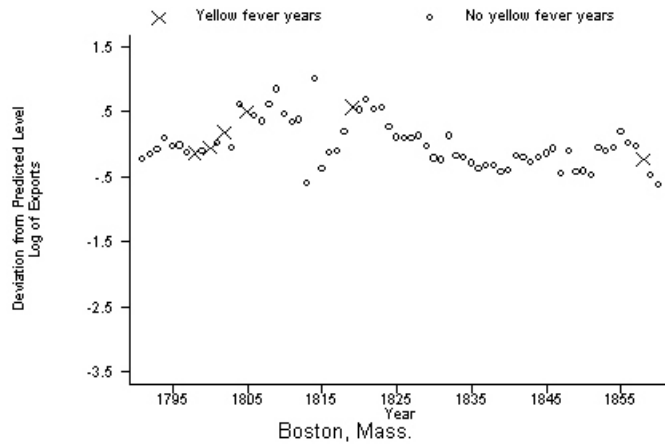


Figure 23d

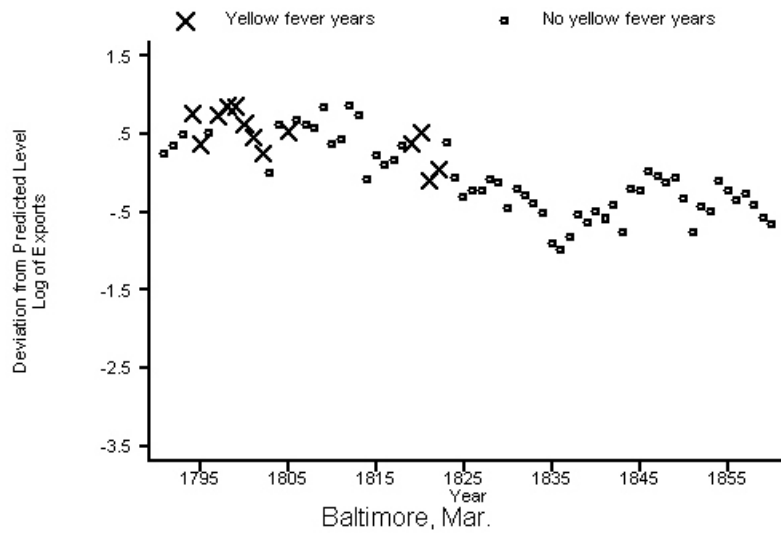


Figure 23e

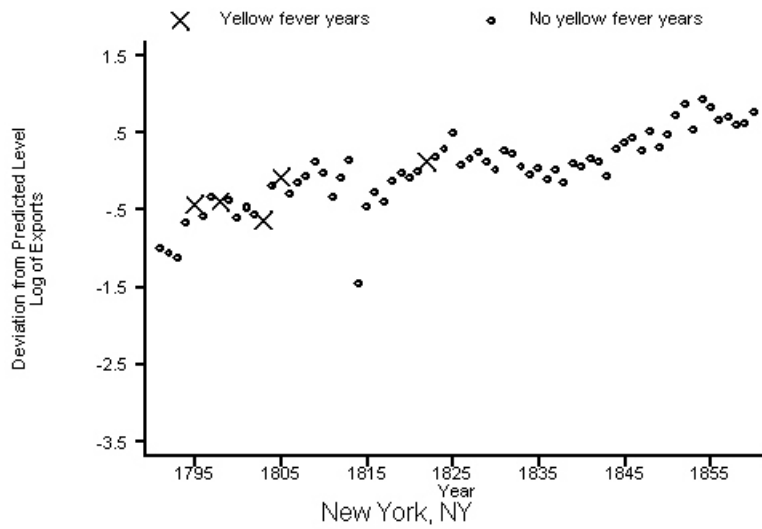


Figure 23f

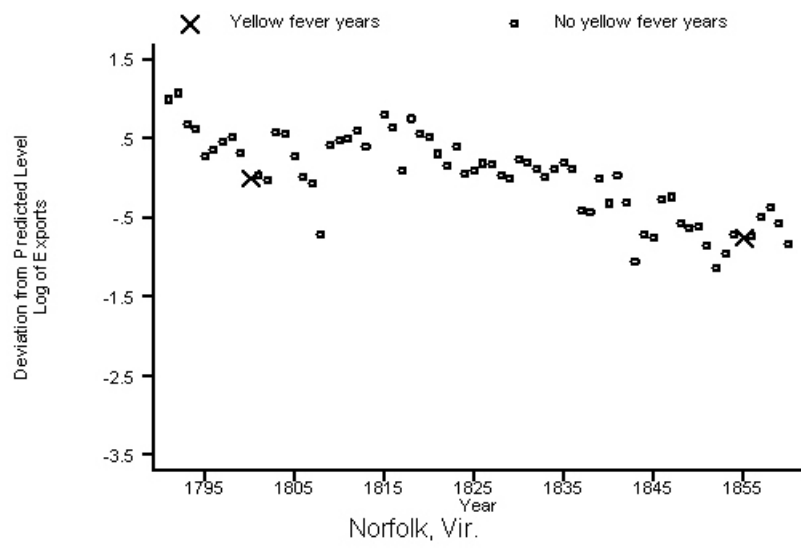


Figure 23i

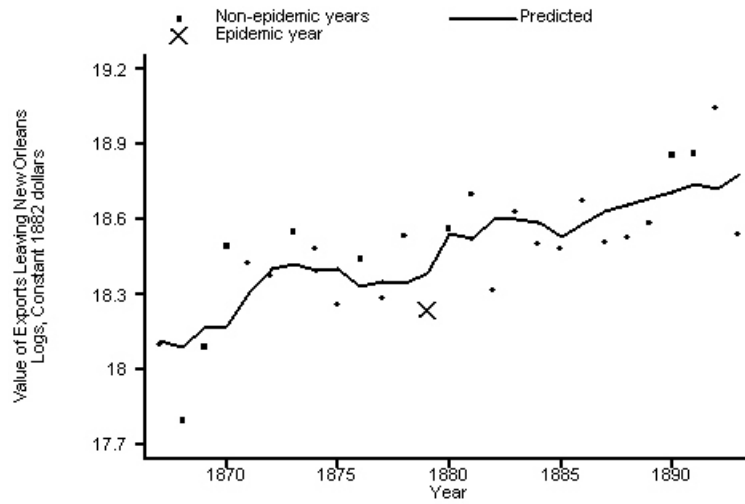


Figure 24. Exports in New Orleans, 1866-1893

Source: see text.

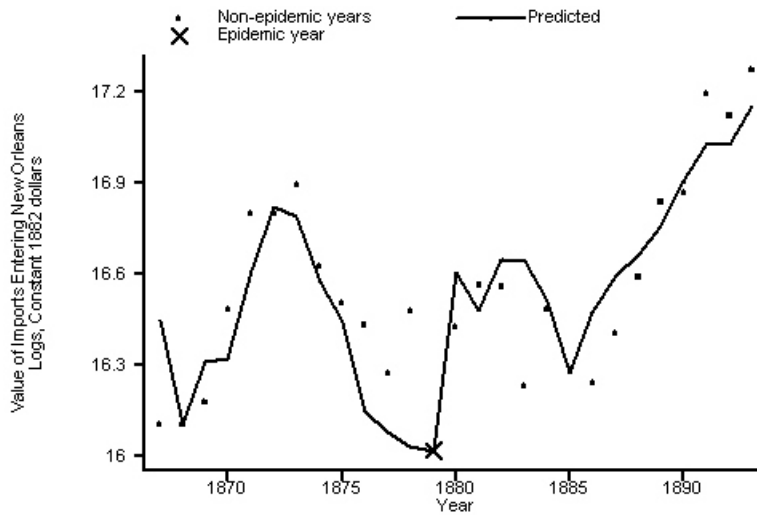


Figure 24. Imports in New Orleans, 1866-1893

Source: see text.



Figure 25. Business Failures in the United States

Source: Commercial and Financial Chronicle.

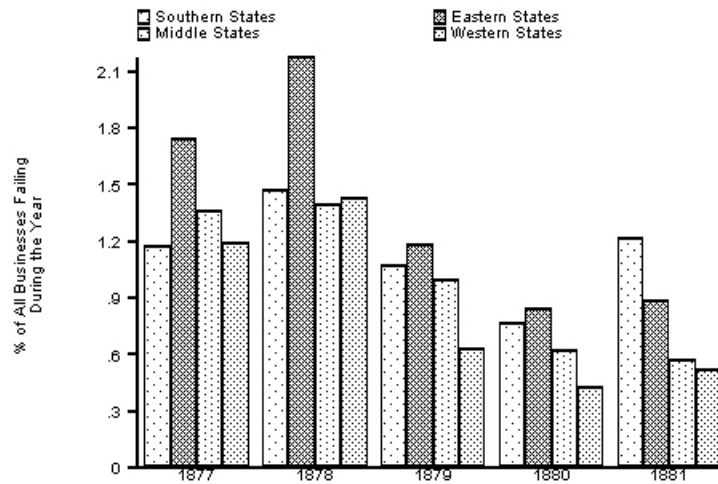


Figure 26. Regional Variation in Business Failures

Source: Commercial and Financial Chronicle.

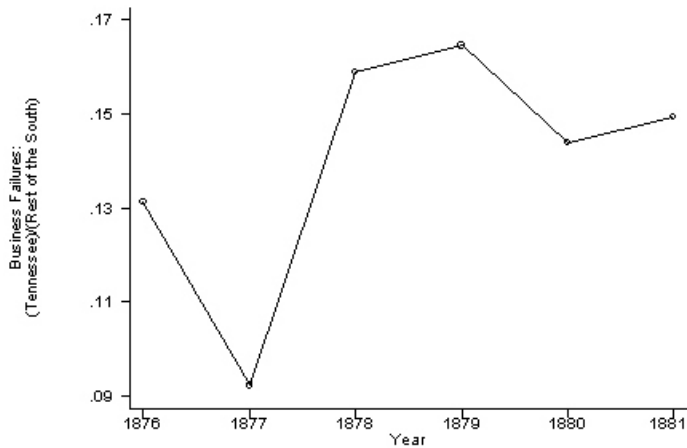


Figure 27. Business Failures in Tennessee

Source: *Commercial and Financial Chronicle*.

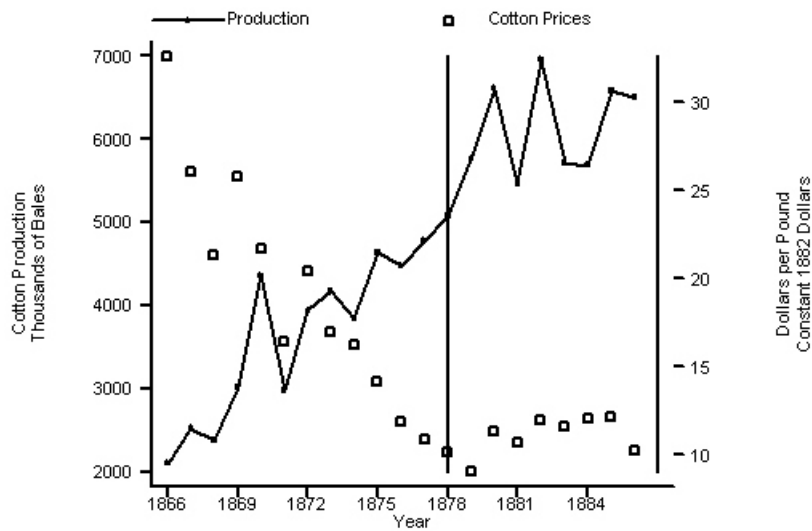


Figure 28. Cotton Prices and Production in the United States, 1866-1886

Source: Carter *et al.* (2006b), p. 4-111, Series Da756 (cotton production); and Carter *et al.* (2006b), pp. 3-208-209, Series Cc222 (cotton prices).