

YELLOW FEVER EPIDEMICS AND MORTALITY IN THE UNITED STATES, 1693–1905

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Abstract—Yellow fever epidemics struck the United States repeatedly in the 18th and 19th centuries. The disease was not indigenous; epidemics were imported by ship from the Caribbean. Prior to 1822, yellow fever attacked cities as far north as Boston, but after 1822 it was restricted to the south. Port cities were the primary targets, but the disease occasionally spread up the Mississippi River system in the 1800s. New Orleans, Mobile, Savannah, and Charleston were major targets; Memphis suffered terribly in 1878. Yellow fever epidemics caused terror, economic disruption, and some 100,000–150,000 deaths. Recent white immigrants to southern port cities were the most vulnerable; local whites and blacks enjoyed considerable resistance.

Key words—epidemics, mortality, United States, yellow fever

Yellow fever had a long history in the United States as a scourge of cities and towns, primarily coastal and river ports. From its initial appearance in Spanish Florida in 1649–1650 [1] and its first known visit to British North America when warships from the West Indies infected Boston and other towns in 1693 [2], until the last epidemic of 'yellow jack' on the Gulf coast in 1905, the United States and the English, French and Spanish territories from which it was constituted were repeatedly attacked by lethal epidemics of this deadly tropical disease. Yellow fever struck at irregular intervals, sometimes afflicting a community for several years in a row, then disappearing for a decade or forever. It always flourished in the summer and early fall, disappearing with the first frost. With its unpredictable behavior, frightening symptoms, and high case-fatality rate, yellow fever was a puzzle to doctors and laymen alike. Perhaps no disease attracted so much or such acrimonious medical debate for so long as did yellow fever, as physicians discussed and sometimes argued bitterly over the nature of the disease, its cause, prevention, and therapy, and whether it was generated locally or somehow spread from person to person and place to place [3, 4].

Yellow fever is an acute viral disease transmitted to man by the bite of the female *Aedes aegypti* mosquito. It cannot be spread by contact and is not directly contagious from person to person. Many cases in adults, and the vast majority of cases among children, are mild or asymptomatic. In clinical cases, symptoms include fever, body aches, headache, nausea, and prostration occur from 3–6 days after infection. Liver damage frequently results in jaundice and damage to capillaries can result in bleeding from the nose and mouth, stools stained dark with blood, and the most dreaded symptom, copious black vomit due to gastric bleeding. Urine production is reduced and

sometimes completely suppressed, and neurological involvement can cause confusion, convulsions, and coma. Death may occur from one or a few days after the onset of symptoms; some victims may linger for 2 weeks. Case-fatality rates range from 15 to 50% and higher; very high mortality reports may indicate that milder cases were not recognized or were not reported as yellow fever. Children seem to have strong resistance to the virus and generally suffer only mild or sub-clinical cases. Persons recovering from either clinical or inapparent cases gain lifetime immunity [5, 6].

Past epidemics were not recorded with modern medical knowledge and terminology, so it is not always possible to diagnose yellow fever (or any other disease) retrospectively. Even today, diagnosis of individual cases may require sophisticated tests. However, significant epidemics can usually be distinguished in 18th and 19th century descriptions. High fever, epidemic occurrence, extensive mortality, absence of references to rashes and diarrhea, and cases with black vomit indicate that yellow fever was present in a community.

The mosquito vector is an urban, peri-domestic insect which breeds in small bodies of water near houses, such as gutters, flower pots, water storage barrels, and other receptacles. *Aedes aegypti* is most active in warm, moist conditions, and feeds only when the temperature is above 15–17°C. It is a weak flier, generally ranging no more than a few hundred meters. Mosquitoes ingest viruses in blood meals from patients, and after an incubation period which varies from 4 days to 2 weeks depending on the temperature, can infect another victim. The vector can also feed on plant juices and other sugary fluids, but blood meals are needed for egg production. In Africa and Latin America there is also a forest cycle involving monkeys and other species of mosquitoes.

This cycle creates a reservoir of infection which can become 'urban' yellow fever when a person bitten in the forest by a 'monkey' mosquito acquires viruses and is then bitten by *A. aegypti* [7, 8]. Yellow fever can exist as an urban disease with the man-*Aedes* cycle indefinitely in tropical climates as long as there is a constant supply of non-immune people. If the cycle fails, it will be absent until infected persons come from other places to infect the local *Aedes* or until the forest cycle provides a human virus carrier. In temperate climates, as throughout the United States, adult *Aedes* do not survive the winter. Eggs hatch in the spring to create a new generation of vectors, but the virus must be introduced again [9].

Both the vector and the virus almost certainly originated in Africa and reached the New World as unintended by-products of the slave trade [7, p. 41, 10-12]. The mosquito was probably introduced early, as it travels well in ships' water barrels, but the virus seems to have come much later, after the vector was well established in the new settlements. The first clearly described epidemic in the Americas took place in Barbados in 1647 and Yucatan in 1648 and the disease quickly spread to several points in the Caribbean, notably Barbados, Martinique, St Kitts, Cuba, and Florida. The disease probably did not become permanently established then, as the next known epidemic did not take place until 1685, when Brazil was attacked. There was a flurry of activity in the 1690s and the virus was probably established in Brazil and in the Spanish mainland colonies by then, with new epidemics being generated from primate reservoirs, fresh importations from West Africa, or by circulation of the virus around the coasts and islands of the Caribbean [2, pp. 675-676, 7]. African importation probably remained important, because Cuba was free from the disease for decades prior to 1761 and yellow fever did not become endemic on smaller islands like Barbados [13].

It has been suggested that the rise of the sugar industry created ideal conditions for mosquito breeding and the transmission of yellow fever. Containers used for sugar processing were excellent breeding sites; mosquitoes could feed on sugary water, and the plantations were inhabited by susceptible Europeans and potential African donors of virus. In addition, sugar caused a general growth in population in trading towns, a rise in the volume of shipping and in the numbers of European sailors, and leaking sugar casks provided food to supplement the sailors' blood [14]. In any case, it is clear now, as it was to some earlier observers, that yellow fever was frequently introduced by sick seamen. This is the basic mode of introduction and spread in the United States; the virus was repeatedly imported into harbor towns in the summer in the bodies of people or ship-transported mosquitoes and then spread among the local *Aedes* and human populations. And, at least until the advent of the railroad and river steamer, it rarely ventured inland. Even then, except in the

Mississippi Valley, it remained primarily a disease of port cities and their immediate environs. In contrast to many other diseases, yellow fever did not find America's harbors a gateway to the hinterland.

Yellow fever occurred in the United States throughout the 18th and 19th centuries in large and small outbreaks. Some decades were almost free of recorded epidemics; at other times the disease struck widely year after year. Most of the 18th century epidemics took place in the small but growing Atlantic port cities of the colonies; by the end of the century towns on the Gulf coast began to be attacked. After initial importations from the West Indies in the 1690s through 1702, yellow fever seems to have rarely ventured north of Charleston until the 1740s. However, as Table 1 shows, that port was attacked on several occasions. There were some epidemics there and further north during the 1740s, but the disease was absent from 1762 until 1793, when a terrible epidemic devastated Philadelphia, then the capital of the new Republic. The disease was active up and down the coast from 1794 to 1805, striking cities as far north as Portland, Maine (1801) and Portsmouth, New Hampshire (1798) reported small outbreaks. It is probable that the lull from 1762-1793 was directly linked to the absence of yellow fever in the Lesser Antilles during this period [21, pp. 675-676]. The series of epidemics beginning in 1794 was caused by the dramatic upsurge in yellow fever in the West Indies linked to Anglo-French wars, the Haitian revolt, and the large-scale introduction of susceptible European troops into the region [15]. Indeed, the Philadelphia epidemic was almost certainly introduced by French refugees fleeing the wars in Haiti.

After 1805, there was another lull in yellow fever activity along the Atlantic coast until 1819. Northern ports like Boston, New York, Philadelphia and Baltimore were attacked until 1822, when, except for occasional cases caught in marine quarantine stations, yellow fever ceased to afflict the coast north of Virginia. However, as Table 2 indicates, yellow fever remained very active further south, especially in Charleston, and was increasingly common along the Gulf coast. New Orleans, by far the largest city on the Gulf and growing rapidly as the trade entrepot for the whole Mississippi Valley, hardly had a year without at least a few cases. The climax came in the 1850s, when a series of epidemics cost tens of thousands of lives in the Crescent City. Hastily erected local quarantines failed to prevent the spread of yellow fever into dozens of nearby communities and as far afield as Alexandria and Baton Rouge, Louisiana and Vicksburg and Jackson, Mississippi. Yellow fever was also epidemic elsewhere along the Gulf and Atlantic coasts during the early 1850s, with devastating epidemics as far north as Norfolk and Portsmouth, Virginia.

The Civil War period was relatively quiet, despite the presence of large numbers of vulnerable Northern

troops in New Orleans and along the Carolina coast. Except in a few Union strongholds like Key West and New Bern, North Carolina, the naval blockade and the wartime reduction in foreign trade in Union-held areas apparently protected both citizens and, to the chagrin of some Confederates, the occupying garrisons, against viral importation.

There was another spurt of yellow fever activity beginning in 1867. Steamships helped spread yellow fever far up the Mississippi and its tributaries in 1873, with both Memphis and Shreveport, on the Red River, suffering severely. Cases were reported as far north as Cairo, Illinois, which had 13 fatalities [16]. The great epidemic which swept up the Mississippi

from the Gulf to Memphis and St Louis in 1878 took about 16,000–20,000 lives, and the toll may have been even higher [17–21].

After that disaster, yellow fever occurred only in sporadic outbreaks, mostly along the Gulf coast, until its final appearance in 1905. The reasons for its demise are unclear, but probably many factors were at work. Changes in ship design and sailing patterns, better quarantines, urban improvements including piped water supplies, increasing use of window screens, and, after the discovery of the mosquito vector in 1900, measures specifically directed against *Aedes aegypti* breeding sites all played a role.

Table 1. Major yellow fever epidemics, 1693–1805

| Year | Place | Deaths | Mortality and Remarks | Year | Place | Deaths | Mortality and Remarks |
|------|--|---------------------|---------------------------------------|------|---|-----------------------------------|-----------------------|
| 1693 | Boston, MA Charleston, SC Philadelphia, PA | | 1st certain epidemics in Brit. N. Am. | 1796 | Charleston New Orleans New York Norfolk Philadelphia | 300 | 1st epidemic? |
| 1699 | Charleston Philadelphia | 179 220 | ca 7% ca 7–11% | 1797 | Charleston New York Philadelphia Providence | 1300–1700 45 | |
| 1702 | New York, NY | 570 | ca 10% | 1798 | Boston New York Norfolk Philadelphia Portsmouth Wilmington, DL | 200 2080 3500 100 250 | |
| 1728 | Charleston | | | 1799 | Baltimore Charleston New Orleans New York Norfolk Philadelphia | 4 239 300 76 1000 | |
| 1732 | Charleston New York Philadelphia | '100s' 70 259 | ca 2–3% | 1800 | Baltimore Boston Charleston New Orleans New York Norfolk | 1197 184 21 250 | 4.6% 0.6% 2% |
| 1737 | Norfolk, VA | | | 1801 | Baltimore New York Norfolk Savannah, GA | | |
| 1739 | Charleston | | | 1802 | Baltimore Boston Charleston Norfolk Philadelphia Wilmington | 60 96 307 86 | 0.7% |
| 1741 | Boston New York Norfolk Philadelphia | 15 250 | | 1803 | Charleston New Haven New York Norfolk Philadelphia | 600–700 200 195 | < 1% |
| 1743 | New Haven, CN New York Philadelphia | 217 | ca 2% | 1804 | Charleston New Orleans Norfolk | 148 | |
| 1745 | Charleston New York | | | 1805 | Baltimore Boston New Haven New York Philadelphia Providence | 340 300–400 | |
| 1747 | New York Norfolk Philadelphia | | | | | | |
| 1748 | Charleston | | | | | | |
| 1762 | Charleston Philadelphia | | | | | | |
| 1764 | Pensacola, FL | | | | | | |
| 1765 | Mobile, AL Pensacola | 125 | | | | | |
| 1793 | New York Philadelphia Portsmouth, NH | ca 5000 | ca 8–9% | | | | |
| 1794 | Baltimore, MD Charleston New York Norfolk Philadelphia Providence, RI | 360 | | | | | |
| 1795 | Baltimore Boston Charleston New York Norfolk Philadelphia Providence | 700–730 | ca 2% | | | | |

Compiled from Augustin D. G. C. Yellow fever epidemics of the late eighteenth century in Baltimore. *Maryland St. Med. J.* 21, 47–49; Dowell, Hirsch and specific sources cited-in and text for particular cities.

Table 2. Major yellow fever epidemics, 1808–1905

| Year | Place | Deaths | Mortality and Remarks | Year | Place | Deaths | Mortality and Remarks |
|------|------------------|---------|-----------------------|------|-----------------------|---------------|---------------------------|
| 1808 | St Marys, GA | 45 | 9% | 1849 | Charleston | 125 | |
| 1811 | New Orleans | 500 | | | Mobile | 50 | |
| | Pensacola | | | | New Orleans | 737 | |
| 1817 | Charleston | 272 | | 1852 | Charleston | 310 | 0.7% |
| | New Orleans | 800 | | | New Orleans | 415 | |
| 1818 | Charleston | 115 | | | Savannah | 19 | |
| | New Orleans | 115 | | 1853 | Baton Rouge, LA | 200 | |
| 1819 | Baltimore | 2287 | 3.6% | | Galveston | 535 | 6.7% |
| | Charleston | 117 | 0.5% | | Key West | 112 | |
| | Mobile | 274 | ca 10% | | Mobile | 1072 | 12.6% |
| | New Orleans | 2190 | ca 4% | | New Orleans | ca 9000 | ca 9%; highest total |
| | New York | 37 | | | Norfolk | 1600 | ca 11% |
| | Savannah | 50 | | | Pensacola | | |
| 1820 | New Orleans | | | | Savannah | | |
| | Philadelphia | 83 | | 1854 | Charleston | 627 | 1.3% |
| | Savannah | 700–729 | ca 9% | | Galveston | 404 | 5.1% |
| 1821 | Baltimore | | | | Mobile | | |
| | Norfolk | | | | New Orleans | 2425 | |
| | St Augustine, FL | 140 | | | Pensacola | | |
| | Savannah | | | | Savannah | 1040 | 6.9% |
| | Wilmington, NC | | | 1855 | New Orleans | 2670 | |
| 1822 | Baltimore | | | | Norfolk | 1807 | ca 12% |
| | New Orleans | | | | Portsmouth, VA | 1000 | ca 12% |
| | New York | | | 1856 | Charleston | 211–238 | |
| | Pensacola | | | 1857 | Jacksonville, FL | | |
| 1824 | Charleston | 235 | | | New Orleans | 199 | |
| | Mobile | | | 1858 | Charleston | 717 | |
| | New Orleans | 118 | | | Galveston | 873 | 8.7% |
| 1825 | New Orleans | 49 | | | Mobile | | |
| 1827 | Charleston | 64 | | | New Orleans | 4854 | |
| | Mobile | | | | Savannah | | |
| | New Orleans | 109 | | 1859 | Galveston | 186 | |
| | Pensacola | | | 1862 | Key West | 71 | Northern troops |
| 1828 | Charleston | 26 | | | Wilmington, NC | 446 | Blockade runner |
| | New Orleans | 130 | | 1864 | Galveston | 265 | Blockade runner |
| 1829 | Key West, FL | | | | New Bern, NC | 700 | Northern troops |
| | Mobile | | | 1867 | Galveston | 1150 | 5.2% |
| | New Orleans | 130 | 4% | | Memphis, TN | 250 | |
| 1830 | Charleston | 39 | | | New Orleans | 3107 | 1.5% |
| | New Orleans | 117 | | | Pensacola | 34 | |
| 1834 | Charleston | 49 | | 1870 | Galveston | 16 | |
| 1835 | New Orleans | 284 | | | Mobile | | |
| 1837 | Mobile | 350 | | | New Orleans | 587 | |
| | New Orleans | 442 | | 1871 | Charleston | 213 | |
| 1838 | Charleston | 351 | | 1873 | Memphis | 1224–2000 | 2000 more probable; 5% |
| 1839 | Charleston | 134 | | | Mobile | 27 | |
| | Galveston, TX | 250 | 25% | | New Orleans | 225 | |
| | Mobile | 650 | | | Pensacola | 62 | |
| | New Orleans | 452 | | | Shreveport, LA | 759 | |
| | Pensacola | | | 1874 | Pensacola | 371 | ca 10% |
| 1841 | Key West | 26 | | 1876 | Brunswick, GA area | 112 | |
| | New Orleans | 594 | | | Charleston | 26 | last epidemic |
| | Pensacola | | | | Savannah | 1066 | ca 3% |
| | St Augustine | 26 | | 1878 | Greenville, MS | 299 | |
| 1842 | Mobile | 60 | | | Memphis | 5150 | ca 10% |
| | New Orleans | 211 | | | Mobile | | |
| 1843 | Mobile | 240 | | | New Orleans | 4046 | ca 2% |
| | New Orleans | 487 | | | Vicksburg, MS | 1000+ | |
| 1844 | Galveston | 400 | 6.7% | | Entire region | 16,000–20,000 | |
| | Mobile | | | 1879 | Memphis | 583 | |
| | New Orleans | 148 | | | New Orleans | | |
| 1845 | New Orleans | 146 | | 1882 | Pensacola | 192 | |
| 1846 | New Orleans | 160 | | 1887 | Key West | 62 | |
| 1847 | Galveston | 200 | 3.3% | 1888 | Jacksonville area | '100s' | |
| | Mobile | 76 | | 1897 | Louisiana, Gulf towns | 454 | |
| | New Orleans | 2259 | 1.9% | 1905 | New Orleans, | 437 | Last U.S. epidemic |
| 1848 | Mobile | 75 | | | Pensacola, scattered | | |
| | New Orleans | 850 | | | Gulf towns | | |

Compiled from Augustin, Baker, Charleston Report, Dowell, Duffy, *Sword of Pestilence*, and Nineteenth Century; Hirsch R. M., Yellow fever: the Shreveport epidemic of 1873. *Jl Northern Louisiana Hist. Soc.* 17, 65–79, 1986. Vickers E. D. and Vikers F. N., Notations on Pensacola's medical history. *J. Florida Med. Assoc.* 61, 83–105, 1974; sources for cities in text.

Tables 1 and 2 show major outbreaks and, when available, estimates of fatalities. The data are compiled from three types of sources: contemporary medical articles and reports commissioned by governmental agencies; compilations of contemporary literature by Augustin, Dowell and Hirsch [22–24]; and recent secondary studies. It must be kept in mind that not all epidemics labelled as yellow fever, especially in the early decades of the 18th century, were necessarily yellow fever, and that small outbreaks were very incompletely recorded. Deaths and especially cases were seldom systematically tabulated. There are often discrepancies between contemporary figures. Some deaths due to yellow fever may have been caused by other conditions, and *vice versa*. Totals for particular towns may not include deaths among refugees who fled to more salubrious locales, and usually do not account for scattered deaths due to importation into smaller neighboring communities. Deaths in smaller communities infected from the major ports could run into the thousands, as was demonstrated in the great epidemics of 1853 and 1878 [19, pp. 161–168, 25]. In addition, deaths in small outbreaks, most of which are not shown here, were less likely to be assigned to yellow fever. Children were generally considered immune, and indeed they usually do contract mild cases, but some no doubt perished of yellow fever without being counted as victims [26].

Another problem is that contemporary estimates of mortality during at least some large epidemics seem to have been too low. For example, Cary listed 4041 victims in Philadelphia in 1793 by getting the names of persons buried in churchyards or the 'Potter's Field', but a modern student estimates the real toll at over 5000 [27, 28]. Official figures for New Orleans in the terrible 1853 epidemic were 7848 dead; the author of an excellent recent monograph puts the true number at almost 9000 and possibly a bit higher [22, p. 65, 25, p. 167]. Similarly, one contemporary estimate of mortality in Memphis in 1878 put the toll at 4396; another writer, apparently using later data, gave figures of 4204 whites and 946 blacks for a total of 5150. The author of a recent study of the epidemic accepts the latter number [18, p. 116, 20, p. 241, 29].

Finally, mortality rates are difficult to tabulate, not only because of uncertainties about the numbers of deaths, but also about the sizes of rapidly growing towns in years between censuses. In addition, New Orleans and other ports had large 'floating' populations of boat men, laborers and drifters. Even more important is the problem of the large but uncounted numbers of urban dwellers who always fled once it became clear that yellow fever was epidemic. Denominators, the populations at risk, are as imprecise as numerators, the numbers of yellow fever deaths. The death estimates presented here and in the tables for individual cities are therefore conservative estimates based on sometimes very rough data.

It is impossible to give more than an approximate, more or less impressionistic statement of the overall

mortality toll. A count of deaths noted by Augustin, which does not give numbers for a majority of epidemics even though he does seem to have contemporary estimates for what appear to be most of the larger outbreaks, gives 16,634 for 1693–1799 and 65,521 for 1800–1893, for a grand total of 82,155. To this must be added 454 in 1897 [21, p. 23], and 437 or more in 1905, and a few thousand for low estimates for several major epidemics. Conservative addition of the estimates presented in Tables 1 and 2 produces a sum of about 100,000 deaths, three quarters of which occurred after 1805. Given the potential totals in known epidemics for which no fatality data are listed, it is reasonable to estimate that from 1693 to 1905 yellow fever killed at least 100,000 Americans, and the total could well be 150,000 or more.

Another approach to the problem of overall mortality is to examine the record of some key cities. Tables 1 and 2 show that northern ports such as Philadelphia and Baltimore suffered greatly into the first quarter of the nineteenth century, but that yellow fever did not cause epidemics north of Virginia after 1822. The reasons for this are unknown; quarantines may have played a role, but in most places these were poorly enforced or ignored altogether. Meanwhile, on the southern part of the Atlantic coast, Norfolk and Savannah had a few very bad outbreaks, but neither had nearly as many epidemics as Charleston.

Epidemics seem to have become increasingly centered on the Gulf Coast by the early 1800s. Close and expanding commercial connections with the West Indies, Mexico, and northern South America, together with the growth of New Orleans and other urban ports, help to explain this phenomenon. Warm climates for most of the year also gave *A. aegypti* a long season to transmit infection. The Spanish outposts of Pensacola and Mobile were occupied by Great Britain in 1763; it is perhaps due to the presence of susceptible English soldiers that their first recorded epidemics took place in 1764 and 1765. Unfortunately, while both cities were frequently struck, and Mobile was second only to New Orleans in this regard, mortality data are not available for either city for most epidemics. The toll for Mobile in the eleven epidemics for which deaths are known was over 3000. In 1853 the town lost about one resident in eight, despite the fact that most of the white population fled. Galveston was hit hard in 1839, only two years after it was founded, and periodically suffered serious outbreaks. Its experience is well documented; the city lost about 4279 residents in ten epidemics between 1839 and 1870. Key West, primarily a naval base and fishing port, had its first epidemic in 1829, again, shortly after its founding. Like Galveston, it also had several later bouts with yellow fever, but its population was much smaller and the U.S. Navy probably had more efficient quarantines. Ports along the Mississippi River system sometimes

suffered as well. For example, Natchez Mississippi, a city with fewer than 3000 inhabitants in 1830, had 1256 deaths in 7 epidemics between 1817 and 1839, and was struck 5 more times between 1844 and 1858. Only New Orleans, however, had enough people, including newcomers, and enough trade with Havana, Mexico, and other potential sources of infection to support almost annual visits from 'yellow jack.'

The experiences of the two most frequently hit cities, Charleston and New Orleans, are shown from 1817 to 1905 in Table 3. Charleston was first struck in 1693 and had several poorly documented epidemics during the 18th and early 19th centuries; it had its last yellow fever in 1876. The history of the disease in New Orleans did not begin until the 1790s, but it was the chief victim in 1905, the last epidemic in the continental United States. Both communities obviously suffered severely, but New Orleans, by far the larger city and with a much higher number of susceptible immigrants and transients, had more than ten times as many deaths and more frequent epidemics as well.

Not all residents of a city were equally vulnerable when yellow fever struck. Men died in greater numbers than women; adults were more likely to contract fatal cases than children. Poor people seemed to be singled out in some epidemics, but not, as contemporary miasmatic theory held, because they lived in crowded, filthy conditions. The poor were vulnerable partly because they often lived near the docks, where

we now know that infected ships would bring the disease first [28, pp. 8–18, 30], and partly because many of the poor were immunologically naive newcomers. But 18th and 19th century medical and lay opinion, while aware of differential mortality among the poor, generally recognized three groups with different risks of death from yellow fever. New arrivals, whether from the northern states or from Europe, were at greatest risk. Locally-born whites were relatively, though not totally safe, and blacks seemed to get fewer and milder cases than either white group.

Eighteenth-century observers frequently noted the high degree of immunity among native citizens of cities like Charleston or New Orleans and the extreme vulnerability of 'unseasoned' newcomers to the local 'climate.' While most recognized that survivors of attacks were immune to re-infection, this was by no means universally accepted, and there was no conception of the importance of the acquisition of immunity through mild attacks in childhood until the 1850s and later. In any case, it was believed well into the 19th century that new residents could become 'acclimatized' to local fevers without a life-threatening illness and could gradually acquire the immunities conveyed by native birth. Such resistance, dubbed 'city creolism' by a prominent 19th century student of yellow fever, was described in the 1850s for New Orleans, Mobile, Charleston, Havana, and Vera Cruz, but it did not extend into the countryside. Rural dwellers were very vulnerable if they went to

Table 3. Yellow fever mortality in Charleston and New Orleans, 1817–1905

| Year | Charleston | New Orleans | Year | Charleston | New Orleans |
|------|------------|-------------|--------|------------|-------------|
| 1817 | 272 | 800 | 1846 | — | 160 |
| 1818 | 115 | 115 | 1847 | — | 2804 |
| 1819 | 117 | 2190 | 1848 | — | 872 |
| 1820 | — | + | 1849 | 125 | 737 |
| 1821 | — | — | 1850 | — | 102 |
| 1822 | 2 | 239 | 1851 | — | 16 |
| 1823 | — | 1 | 1852 | 310 | 415 |
| 1824 | 235 | 118 | 1853 | — | ca 9000 |
| 1825 | 2 | 49 | 1854 | 627 | 2425 |
| 1826 | — | 5 | 1855 | — | 2670 |
| 1827 | 64 | 109 | 1856 | 211 | 74 |
| 1828 | 26 | 130 | 1857 | 13 | 199 |
| 1829 | — | 215 | 1858 | 717 | 4854 |
| 1830 | 30 | 117 | 1859 | — | — |
| 1831 | — | 2 | 1867 | — | 3107 |
| 1832 | — | 18 | 1870 | — | 587 |
| 1833 | — | 210 | 1871 | 213 | 55 |
| 1834 | 49 | 95 | 1872 | — | 40 |
| 1835 | 25 | 284 | 1873 | — | 225 |
| 1836 | — | 5 | 1876 | 26 | — |
| 1837 | — | 442 | 1878 | — | 4046 |
| 1838 | 351 | 17 | 1879 | — | + |
| 1839 | 134 | 452 | 1897 | — | 288 |
| 1840 | 22 | 3 | 1905 | — | 437 |
| 1841 | — | 594 | Totals | 3687 | 40,171 |
| 1842 | — | 211 | | | |
| 1843 | 1 | 487 | | | |
| 1844 | — | 148 | | | |
| 1845 | — | 2 | | | |

— = no epidemic.

+ = epidemic, but no mortality report.

Sources: Augustin D. *Sword of Pestilence*. Charleston S. C. City Council, *Report of the Committee of the City Council of Charleston, Upon the Epidemic Yellow Fever, of 1858*, p. 47, 1859.

town and whenever yellow fever spread out from the city [16, pp. 8–16, 30–32].

As early as 1745 a Charleston resident noted in his medical thesis that sailors, working people, and heavy drinkers were much more likely to contract yellow fever than the rich, but concluded that those at greatest risk were 'newcomers' and 'foreigners.' Long-term residents and those born in Charleston had little to fear [33]. The Baron de Pontalba, writing in 1796 to his wife in Europe from his post in the midst of what is supposed to have been New Orleans' first yellow fever epidemic, assured her that "No Creole has been attacked; only those who are not acclimated are stricken." English and American residents and newcomers from France often died, but by the end of the epidemic, only a few natives had perished [34]. The local people must have acquired immunity elsewhere in the Caribbean or, more likely, from undocumented epidemics prior to 1796.

Yellow fever was often called the 'strangers' disease' in 19th century America [16, 35], and for good reason. Virtually every contemporary description and modern study notes the greater vulnerability of new residents and temporary visitors, whether they were from northern states, interior portions of the south, or from Europe. In Charleston, a city commission study of the 1858 epidemic included retrospective data on deaths from fevers, including yellow fever, with categories for age, race, sex and nativity. Unfortunately, these data are lumped together for all fevers, but in epidemic years, when the vast majority of 'fever' deaths were due to yellow fever, this information can be applied to yellow fever as a rough approximation. In 1824, 235 of the 281 (83.6%) 'fever' deaths were ascribed to yellow fever. Whites accounted for 259 of these, although blacks constituted about half of the population. Native whites had 47 deaths, other U.S. natives had 75, and 137 (53%) were foreigners. In 1854, yellow fever accounted for 93.9% (627/675) of all fever deaths; 96.1% (649) of the victims were white; 72.9% (473) were foreigners and 120 (18.5%) were from other states. The 1858 epidemic showed a similar pattern; 717/763 (93.4%) of fever deaths were blamed on yellow fever. Whites accounted for 711 (93.8%) of deaths. Foreigners comprised 485 (68.2%) of these; native whites 99 (13.9%), and other U.S. citizens made up the rest. Few children died and the excess of male deaths is explained by the large numbers of foreign and non-native adult males at risk. Many of the deceased persons of foreign birth were Irish refugees from the Great Famine [36].

Similar patterns prevailed elsewhere, once a pattern of epidemics had been established. Figure 1 illustrates differential mortality by place of origin in Savannah in 1820 [37]. The high number of deaths among natives of Ireland is noteworthy; in 1819 an outbreak was confined to Irish passengers on an immigrant ship, but 50 died. This experience led to the passage of an act to prevent the arrival of

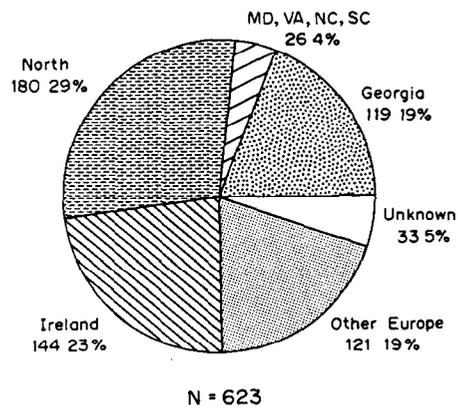


Fig. 1. Yellow fever deaths among whites in Savannah, 1820, by nativity. Russell, Official Register 23.

immigrant ships in the summer months, but the measure did not prevent the 1820 epidemic [38].

Differential mortality during the 1835 epidemic in New Orleans, where repeated invasions had given local survivors ample opportunity to acquire immunity, was especially graphic. Native Creoles had death rates of 3.6/1000; persons from elsewhere in the Caribbean basin had a rate of 6.1; residents of other southern states 13.2; individuals from other northern and western states died at rates ranging from 30.7 to 44.2/1000; immigrants from France and Great Britain had rates of 48.1 and 52.2 respectively; and Netherlanders, presumably a small and very unlucky group, led the list with a yellow fever death rate of 328.9/1000 [24, p. 341]. Irish immigrants, often very poor, made up a disproportionate share of the victims in many epidemics, especially but not solely after the immigration wave of the late 1840s and early 1850s. Yellow fever thus took its heaviest toll among the non-acclimated poor, giving the more established segment of the urban population a sometimes false sense of security.

Urban blacks also had a special immune status. Moultrie, the Charleston writer cited above, noted in 1745 that 'Ethiopians,' despite their poverty and inclination to drink, were 'seldom attacked' [33, p. 774]. Black immunity was also noted in Spanish and French Louisiana, where people of African descent were often employed as nurses [39]. Free blacks played a crucial and much appreciated role in relief measures during the 1793 Philadelphia epidemic. Their immunity was only relative, however, as a number of Afro-Americans died [27, pp. 63, 121–161, 28, pp. 100–107]. The Savannah epidemic of 1820 killed roughly 15% of the white population, but only 2.7% of the blacks [37, p. 23]. During the 1853 New Orleans epidemic, the overall yellow fever death rate for whites was 74/1000; the rate for blacks was only 2/1000 [40].

The 1808 outbreak which hit the small community of St Marys, Georgia also showed a remarkable difference in racial vulnerability. Of the 350 whites, 87 (25%) got sick, and 42 died, for a case mortality rate

of 48%, despite the fact that most of the white population fled when the epidemic became severe. Most of the community's 150 blacks stayed. They suffered 45 cases for a 30% morbidity rate, higher than the white rate, but only 3 died [41].

Morbidity and mortality rates like these supported a modified view of black resistance. As time went on, blacks seemed to suffer morbidity rates not much lower than those among whites, but they generally contracted much milder cases and had significantly lower mortality rates. These phenomena were noted in 1873 at Shreveport and 5 years later during the great Memphis epidemic, where 946 blacks did succumb [42]. Other examples of the relative racial immunity of persons of African descent are illustrated in Fig. 2.

There is still some dispute over the nature of black resistance to yellow fever but, as Kiple and Kiple argue, there appear to be two factors involved, acquired and genetic [40, pp. 416-36]. Blacks in West Africa generally acquired immunity to yellow fever by mild childhood cases, which explains their high degree of resistance compared to European traders, missionaries, or officials. Like their white counterparts in cities with frequent importations of the disease, blacks continued to acquire immunity in this fashion in the Americas. The relative stability of urban black populations, both slave and free, allowed them to share 'native' immunity with whites. Therefore, in cities like Charleston, New Orleans and, presumably, Mobile, local blacks and local whites shared low morbidity and mortality rates. In Louisiana during the terrible epidemics of the early 1850s, both blacks and whites in New Orleans suffered much lower morbidity and mortality than members of their groups in or from rural areas. Clearly, persons of African descent were not

immune, especially those not native to port cities, "yet they suffer little from it and very rarely die" [16, pp. 329-331].

Black resistance to yellow fever, not yet accepted by many medical writers, was also noted in other parts of the Americas. The tremendous disparity in mortality of blacks in Haiti compared to British and French troops probably reflects genetic differences, but the results of differential prior exposure can not be excluded. The low death rates for blacks in Rio de Janeiro in 1849-1850 cannot be explained in this way because the disease had not been reported in the Brazilian capital for decades [43]. Dowler also cites testimony from a Brazilian physician that blacks in Rio had high morbidity but enjoyed much lower mortality than whites during the epidemic [16, p. 331]. The historical record clearly suggests the need to study the idea that many African populations have evolved genetic defenses against the worst effects of yellow fever.

The situation was very different for newcomers or in cities where yellow fever had not occurred recently. Most adults had not acquired childhood immunity, and morbidity rates were high for both groups. Blacks, however, had much lower case-mortality rates, a fact which cannot be explained by past exposure, and certainly is not a consequence of better therapy. It is reasonable to assume that over hundreds of years of exposure in Africa, Africans and their descendants in the New World underwent natural selection for resistance to yellow fever and that they have genetic defenses which whites lack. After the Civil War, as blacks became more mobile and started moving in larger numbers to southern cities, they also began to fall sick more often from yellow fever, just like white 'strangers'. They did not, however, die as frequently.

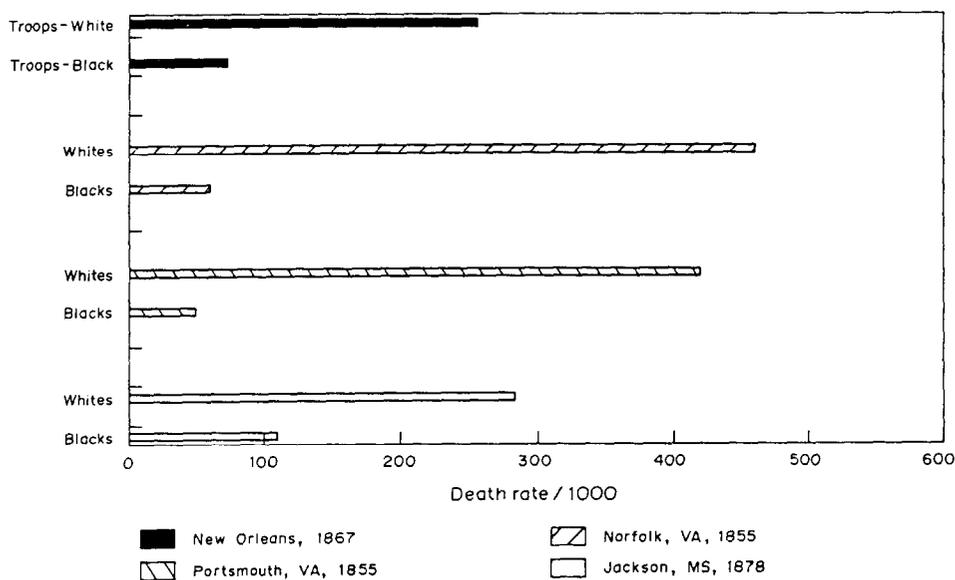


Fig. 2. Case-mortality rates by race, four cities. Kiple and Kiple, 426, 427; Power, 168, 182.



Fig. 3. Sites of major yellow fever outbreaks in the United States.

Yellow fever killed at least *ca* 100,000–150,000 people in the United States from 1693–1905. Since the disease originated in Africa, yellow fever, like falciparum malaria and hookworm (Necator) infection, was a kind of 'biological revenge' for the slave trade; a revenge which killed many more whites than the number of blacks imported [44]. But much more than sheer numbers of victims was involved. Yellow fever struck in epidemics which were unpredictable in space and time, and claimed most of its victims from one segment of the population, white newcomers.

Yellow fever could and did disrupt cities for months. Outbreaks caused panic, the flight of up to or more than half of the population, including a disproportionate percentage of the white and the wealthy, and discouraged outsiders from entering the city. Afflicted places were often almost isolated by vigorous quarantines erected by frightened neighbors, especially after contagionist ideas became more fashionable in the 1870s. Severe epidemics reduced most of those who stayed into corpses, convalescents, or persons preoccupied with caring for the sick aiding survivors, and burying the dead. Urban governments had to drastically increase expenditures for measures ranging from street cleaning to feeding orphans and burying the dead. Yellow fever was, in short, bad for business.

It is impossible to calculate the direct and indirect economic costs of yellow fever epidemics. In the 1793 and 1797 epidemics in Philadelphia, Presidents Wash-

ington and Adams and their cabinets joined a substantial segment of the population in flight, adding political to economic disruption [28, pp. 113–117, 45]. A medical writer estimated that epidemics cost New Orleans about 45 million dollars in expenditures and lost business during the relatively quiet years 1846–1850 [46]. The charitable Howard Association spent \$163,000 to care for victims of the 1853 epidemic in and around New Orleans [25, pp. 127, 172]. The city made a remarkably rapid recovery from the disaster [47], but there were long-term consequences for trade and the rate of immigration and population growth. Norfolk's 1855 epidemic cost an estimated \$5 million in lost business in addition to hundreds of lives, including perhaps 15% of the white population. Financial and demographic recovery took five years [46, pp. 567–568].

New Orleans lost an estimated \$10 million in trade because of the 1878 epidemic; medical care and funeral costs were put at over \$850,000 [48, 49]. Memphis suffered much more, both economically and in loss of life. Population declined from 48,000 in 1870 to 33,000 in 1880 and took years to recover, and local business leaders put the economic loss at \$100 million [20, p. 261]. Over \$1.1 million was spent on relief by the Howard Association [29, p. 63]. The mass exodus of white citizens forced the municipal authorities to hire black policemen, an unusual experiment which was continued successfully for many years after the crisis [50]. One contemporary observer

put the total cost of the 1878 Mississippi Valley epidemic at 25,000 lives and \$200 million [18, p. 15]. Both figures are probably high, but the toll includes death and disruption over a wide area. Even in Jackson, Mississippi, well away from the river, there were 84 deaths and 490 cases, which cost the city an average of \$27, including burial fees [19, p. 182]. The human and economic costs, and the by now conclusive evidence of importation, were powerful stimuli for the establishment and enforcement of stronger quarantine measures [49, p. 191, p. 206].

The real costs of yellow fever are impossible to measure accurately, either in human or economic terms. It doubtlessly played a role in retarding urban and commercial growth and discouraged some immigrants from trying their luck in Dixie, but other factors were much more important. The disease did not kill nearly as many people in the southern states as malaria, pneumonia, or any of a number of more common but less spectacular causes of infant, child or adult mortality, but it attracted much more medical, political, and public debate than any of these quieter killers. Yellow fever was one of the most feared diseases in a region with an unusually high burden of infectious diseases, but it was not even native to the country. As Carrigan has observed, despite its southern distribution after 1822, yellow fever was hardly a typical southern phenomenon. It attacked cities in an overwhelmingly rural region, preferentially struck northern and foreign immigrants in the part of the country which attracted the fewest newcomers, and spared blacks where they were most concentrated [51]. Still, yellow fever was one of the diseases which not only helped to shape the image of the South as a poor, backward part of the nation, but also helped to create that reality.

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