 Seminar on Economic History

My dissertation, entitled “Lowering Mortality: A Spatial History of Segregation, Environments, and Mortality Transitions in New Orleans, 1880–1915,” examines health, segregation, and community development in New Orleans during the mortality transition. I argue that residential segregation coupled with major environmental transformations in the urban landscape increased health disparities between white and black residents during a key transition into the modern health epoch. I digitized and mapped 50,000 death certificates, 40,000 property tax records, historical weather data and topographic surveys, and over 500,000 city directory records in a geographic information systems (GIS) to reconstruct the historical built and natural environs, socioeconomic landscape, and mortality patterns of New Orleans. Geospatial analysis of these sources revealed that white residents' efforts to segregate neighborhoods pushed black residents into more hazardous areas, which, even with advancements in medicine and public health during this period, resulted in larger disease burdens for black residents.

“Lowering Mortality” ties together scholarship on the late-19th/early-20th century mortality transition (e.g., Condran, Haines, Fogel, Colgrove, Higgs) with work on health and black communities during segregation (e.g., Blassingame, Samuel Roberts, Rabinowitz, Tera Hunter, Robert Bullard). I emphasize the importance of bio-environmental conditions to health outcomes, and I argue that we cannot understand the causes and consequences of the mortality transition or segregation without considering the landscape and health environments in which the segregation battles unfolded. Furthermore, the new findings demonstrate the value of spatial information embedded in underutilized sources, such as death certificates and property tax records, to study and explain the broader patterns of health burdens and systems of environmental oppression.

“Lowering Mortality” is organized into four research chapters and introduction/conclusion. The first chapter (“Mapping New Orleans”) presents a new analytical framework based on environmental conditions rather than more arbitrary political boundaries. The second chapter (“Mortality Transition”), which I’ve shared with you, identifies the primary causes of death in different age groups and explains how these changed between 1880 and 1915. My analysis disentangles the divergences in black and white mortality rates by age and disease. The results reveal the effects on life expectancy for black and white residents. The third chapter (“Mortality Terrain”) links the divergences in black and white mortality rates with spatial shifts in demographic patterns and neighborhood-specific environmental conditions. This chapter uses the analytical framework established in Chapter One to uncover the environmentally-linked mortality burdens for black and white residents. The fourth chapter (“Landscapes of Conflict”) argues that, through a combination of grassroots efforts and top-down enforcement, white resident sought to implement the color line along bio-environmental boundaries. Black residents resisted white displacement with limited success. Consequentially, white residents not only strengthened a racially-based system of social and economic oppression (i.e., Jim Crow) at the turn of the twentieth century, but they created a deeply embedded system of oppression at the intersection of disease, environment, and landscape.

At present I am revising the dissertation for publication as a book entitled “Separate but Dead: Creating Unhealthy Environments in a Jim Crow City.” Although my conclusions are based on the new geospatial datasets and results of the spatial analysis, I am rewriting chapters to focus on peoples’ stories. I am moving much of the data and quantitative results into the background while simplifying the visualizations to create a more accessible history. In addition to the analytical results, I am using the HGIS to organize and assemble contextual information on people, neighborhoods, and the city, which allows me to include rich, untapped details that situate the residents’ stories.
ABSTRACT

LOWERING MORTALITY
A Spatial History of Segregation, Environments, and Mortality Transitions in New Orleans, 1880–1915

by

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Between 1880 and 1915, three forces changed New Orleans. City engineers drained the interior of the city, opening new land for settlement. Public health officials gained powerful tools and knowledge to fight and prevent infectious diseases. And white residents developed and increasingly enforced social, economic, and residential segregation by race. The mortality differentials between black and white residents show that the color line segregated health improvements as well. By 1915, white residents of the city had experienced unprecedented health gains. Black infants, however, died at a rate 102 percent higher than white infants, and black adults died at a rate 185 percent higher than white adults. The mortality rate for black adults, moreover, increased by 25 percent, while the rate for white adults decreased by 42 percent. This dissertation uses new methods in spatial history to investigate the causes of the black and white mortality differentials in New Orleans during the Gilded Age (1880-1915). This study finds divergences in white and black mortality rates over the course of the mortality transition. Black infants and adults died at increasingly disparate rates from white infants and adults beginning in the 1890s. Public health improvements, more space, and drier residential areas lowered white mortality while fewer sanitary services, less space, and lower residential areas more prone to flooding increased black mortality. White efforts at oppression and segregation displaced black residents into these low-lying, high-density areas with heavy disease burdens. As such, this dissertation argues that at the turn of the twentieth century, white residents not only implemented a racially-based system of social and economic oppression (Jim Crow), but they created a deeply embedded system of oppression at the intersection of disease, environment, and landscape. This system of environmental oppression has sustained social and economic oppression along the color line in New Orleans, even as community, local, and national reformers have worked to dismantle the overt structures of Jim Crow.
CHAPTER 2: MORTALITY TRANSITION

In New Orleans, as in most other cities prior to the twentieth century, infectious diseases killed young and often. Infant and young children made up the plurality of the deaths in 1880, and infectious diseases caused half of all deaths. By 1915, however, both the relative contribution of child mortality to total mortality and the percentage of deaths from infectious diseases had each been halved. More broadly, scholars have identified these shifts as “the mortality transition,” which they claim enabled unprecedented population growth in the twentieth century, yet most overlooked the unevenness of the mortality transition.¹

Although city-wide mortality patterns by age and disease suggest a mortality transition in New Orleans similar to other American cities, underneath these patterns more complex changes were unfolding. The patterns in mortality for the black and white populations differed markedly in New Orleans. Careful analysis of mortality by age and race shows that shifts in black and white age-specific mortality varied in timing and magnitude. By 1915, black infants were 2.02 times

more likely to die than white infants. Black adults, in the prime of their lives, were 2.41 times more likely to die than white adults.2

The mortality transition in New Orleans was separate and unequal. Although mortality rates declined for both black and white infants between 1880 and 1915, I argue that these declines should not be considered independently.3 In 1915, black infants died at a rate equivalent to the white rate in 1880, while white infant rates had declined by 50 percent. Put another way, the mortality burden for black infants in 1915 equaled that of the pre-transition white burden. Therefore, black infant mortality did not transition in New Orleans during the period under study. For black adults, these years were even worse: the mortality rate for black adults increased in the years commonly demarcated as the mortality transition. The mortality rate for black adults rose nearly 50 percent while the rate for white adults fell 30 percent. This chapter argues that the color line divided the mortality transition. The temporal patterns of black and white deaths compared by age (i.e., not only infants) and disease reveal the large disparities that manifest during this period.

This chapter presents a macro-level analysis of the mortality transition and traces the differences in the mortality transition by age for the black and white populations of New Orleans. My datasets, constructed with historical geographic information systems and database software, as described in the previous chapter, reveal the different patterns of the mortality transition by age, by race, and by disease. The black and white populations shared similar mortality rates only in the early years (i.e., the 1880s and early-1890s). Infant mortality rates began to diverge by race in the 1890s. In the first years of the twentieth century, infant mortality rates for both groups decreased,

2 In 1915, black infants died at a rate of 2,016 per 10,000 while white infants died at a rate of 997 per 10,000. Black adults died at a rate of 208 per 10,000. White adults died at a rate of 86 per 10,000. These rates are age and race adjusted.

3 Unless otherwise noted, the mortality rates included are age and race-adjusted with census estimates. In the subsequent chapter, “Mortality Terrain,” none of the spatial rates are adjusted by age or race. This is due to the lack of a time-sensitive, spatial population dataset that includes age and race, save for the 1880 microform census dataset.
but not at the same rate. The gap between black and white infant mortality continued to expand. Meanwhile, white adults died at decreasing rates while black adults died at increasing rates. By 1915, black adults and infants died at rates double those of white adults and infants. In the mortality transition in New Orleans, white health improved while black health regressed. This chapter establishes the timeline of divergences in black and white rates by age and the major diseases proximately responsible for the divergences. In addition, this chapter includes discussion of particular characteristics of these patterns that will become significant in the following chapter’s environmental analysis.

This chapter uses three datasets to trace the mortality transition by year, cause of death, and race for New Orleans, Louisiana. The Death Certificate Index Dataset enables the examination of mortality by age and race for every year. The Geospatial Mortality Dataset supports the analysis of mortality by cause of death, race, and age for every fifth year. The Board of Health Annual Statistics Dataset provides summary statistics for the main causes of death in the city for each year. All three datasets are useful for disease incidence and rates between the sample years.

The mortality rates calculated using my datasets often differ from mortality rates listed in contemporary public health reports. Officials claimed that mortality rates continued to improve (i.e., decline), yet results from my datasets and statistical analyses indicated greater complexity. Even after testing with the most conservative estimates of the mortality, my results differ from those of contemporary public health reports. Dr. Joseph Jones, former president of the Louisiana Board of Health, shed light on the inaccuracy of public health reports in 1888: “During the past

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4 I use “proximately responsible” to denote that these are not the root causes of higher mortality in the black population. In Chapter Three and Chapter Four, I argue that white segregation efforts and environmental disenfranchisement caused higher disease and mortality burdens for black residents.

5 See “Chapter 1: Mapping New Orleans” for a detailed discussion of these records and the database-creation methodology and “References” for an index of the databases.
eight years no census of the city has been taken, and all calculations based upon any imaginary
increase of the white and colored population must be regarded as uncertain, and useful simply for
sanitary estimates and speculations.”

Public health officials in New Orleans frequently overestimated the population growth of the city, which, intentionally or not, accomplished two things. Large population growth served as a metric for the success and economic health of the city. This fit with the boosterism and city-promoting common among city leaders in the late-nineteenth century. Second, the Board of Health used population estimates to calculate mortality rates for the city. High estimates of population growth yielded artificially lower mortality rates.

Consistently, city officials overestimated the population. After the federal government enumerated

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6 Joseph Jones, twice president of the Board of Health and a well-known public health figure of the time, discussed these issues in *Medical and Surgical Memoirs: Containing Investigations on the Geographical Distribution, Causes, Nature, Relations and Treatment of Various Diseases 1855-1890* (New Orleans: Self-Published, 1890), 311–12. Jones suggested election data as a proxy for population, but this method had limitations even before the Louisiana State Constitution of 1898 disenfranchised African Americans. Samuel Chaille, another president of the Board of Health and a major figure in the national public health community, discussed the city’s knack for overestimating the population in an 1888 article entitled “Life and Death Rates—New Orleans and Other Cities Compared” in the 1888 issue of the *New Orleans Medical and Surgical Journal*. Chaille criticized the tradition of overestimating the city’s population: “Three very serious evils result [from overestimates]: a popular expectation is cultivated which the Census disappoints, and this disappointment induces many to discredit the Census; further, an overestimated population necessitates an underestimated death-rate, and thereby, inadequate appreciation of insanitary conditions; and still further, an overestimated population implies more voters than there are, and thus ‘ballot-box stuffing’ is encouraged” (pg. 88). Chaille suggested that the overestimates by the Board of Health did not matter as much for national statistics because other cities overestimated their populations as well, and so the death-rates remained justly comparable. See *The New Orleans Medical and Surgical Journal* (J.A. Gresham, 1889).


8 I used death certificates and the census population figures. I used a linear model of population growth to calculate the annual population between census years. This method of interpolating intercensal year population estimates is commonly used by demographers. I compared these figures to the Board of Health reports. Before 1896, the death certificates documented an average of 25 percent fewer deaths than the Board of Health aggregate tallies and 8 percent fewer from 1896 to 1915. Death certificates for black people averaged 45 percent fewer than aggregate records and white people averaged 17 percent fewer. Representation of black deaths in the death certificates remained near 70% in 1890 and 1900 (1890 black = -29% and white = -14%; 1900 black = -27% and white = -5%), which indicates that the mortality rate divergences of the 1890s are not explained by improvements in record keeping. In the 1900s, white records approach 100% representation while black records averaged 19% underrepresentation. This change marked later shifts in recordkeeping and the reorganization of the Louisiana Board of Health into the New Orleans Board of Health in 1898.
and released census figures every ten years, public health officials in New Orleans returned to previously published mortality reports to revise the mortality rates to reflect the less-than-estimated population of New Orleans.9

1. CAUSES OF DEATH

In the 1880s, physicians and public health officials developed and refined disease classifications systems.10 The recently formed, but short-lived National Board of Health worked to standardize vital statistics record keeping and the disease classification system. The Louisiana Board of Health began classifying and consolidating mortality statistics for New Orleans after the reorganization of the board in 1877.11 Although they attempted specificity, often physicians diagnosed cause of death accurately at the categorical-level.12

The most prevalent causes of death in New Orleans in the 1880s were infectious diseases, followed by diseases of the digestive, respiratory, nervous, and circulatory systems. Chart 1 illustrates the aggregate mortality transition in New Orleans. As was the case in other American

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9 Therefore, studies on mortality in New Orleans should not rely on mortality rates published in the Board of Health reports between census years. For the Board of Health Annual Statistics Dataset, this project used the disease-specific counts of mortality from the Board of Health reports and then independently calculated mortality rates.


11 Rudolph Matas, The Rudolph Matas History of Medicine in Louisiana, ed. John Duffy, vol. 2 (Baton Rouge, LA: Louisiana State University Press, 1962); see Appendix O in John S. Billings, “Annual Report of the National Board of Health 1880” (Government Printing Office, 1881), 537; Also see Louisiana Board of Health, “Annual Report of the Board of Health,” Annual Report of the Board of Health (New Orleans: State of Louisiana General Assembly, 1882), iii. Jones stated, “Such classification, consolidation and publication of the vital and mortuary statistics of the city was imperatively demanded, not merely to rescue the records from speedy destruction, but also for the purpose of furnishing a firm foundation for the comparison of future progress in sanitary science, and the estimation of the actual effects of the various measures which may, in the future be executed for the improvement of the sanitary condition of the people.”

12 Leonard and Anderton, “Grammars of Death.”
cities, the number of deaths from infectious diseases declined from 50 percent of deaths in 1880 to 25 percent in 1915.13

Chart 1. General Causes of Death in New Orleans, 1880-1915. The causes of death are classified by general category, which shows the decreases in the quinquennial proportion of infectious disease deaths and the increases in chronic causes of death. Data Source: Geospatial Mortality Dataset

13 These groupings are based on the 10th revision of the International Statistical Classification of Diseases and Related Health Problems (ICD 10). Note that certain infectious diseases, such as influenza/pneumonia (J09-J18) and neonatal infections (P35-P39), are classified outside of the general infectious disease category (A00-B99). Some of the changes show changes in diagnosis rather than changes in mortality patterns. For instance, Chart 2 shows the eradication of senility in 1910. In reality, public health officials discontinued the use of the generalized diagnosis of senility because it was broad and vague.
Chart 2. Death Rates by Disease, 1880–1920. The annual mortality rates for the leading causes of death in New Orleans. This chart shows the insignificant influence of many familiar diseases on mortality rates during this period. For example, after the 1878 yellow fever epidemic, only two relatively small outbreaks occurred (1897 and 1905). Conversely, the smallpox epidemic of 1883, which is seldom written about, killed a massive number of people. A page-sized version of this chart is available in Appendix C. Source: Board of Health Annual Statistics and U.S. Census Population Estimates.

Among infectious diseases, tuberculosis killed the largest number of people, followed by diarrheal diseases, pneumonia, malaria, tetanus, and diphtheria.14 Chart 2 “Death Rates by Disease, 1880-1920” tracks deaths by disease. Despite the spikes visible in deaths from smallpox and pneumonia, tuberculosis clearly dominated the disease landscape until the 1910s.

From 1883 to 1884, smallpox broke out in the city. People without previously acquired immunity or vaccination are the most susceptible to infection. Among the susceptible residents in the 1880s, smallpox killed a large percentage of children. Mortality rates jumped for age groups between 1-15 years in this epidemic, making 1883-84 the deadliest years for children of both races. The 1883-84 outbreak made smallpox the leading cause of death for the year (see Chart 2) but apart from a brief appearance in 1900, it did not rank in the top ten causes of death again. While imported epidemic diseases killed large numbers of people in short periods of time, more people died from infectious diseases endemic to the city, such as tuberculosis and dysentery.

These aggregate pictures of the mortality transition in New Orleans hide deeper and highly significant differences between the health outcomes of black and white residents of the city. These patterns emerge as we focus on mortality by age. Black and white mortality rates diverged the most between infants (under age one), adults (ages 15-45), and seniors (65 years and older). Infant and adult mortality rates diverged by race to such a great extent as to have significant ramifications for average age at death and health outcomes.

2. MORTALITY BY AGE GROUP

A. INFANT MORTALITY

In the nineteenth century, the first year of life was the most hazardous.\(^\text{15}\) In the first year of life, infants were susceptible to a wide range of fatal illnesses and conditions. Infants were completely at the mercy of their environment and those controlling their environment. Infants did not choose where they lived or traveled, what they ate or drank, or with whom they came in contact.

\(^{15}\) Compared to current (2013) life tables for the United States, babies born in 1880 had the same probability of dying within one year as someone in their late 90s today. “Actuarial Life Table,” US Government, Social Security Administration, 2013, https://www.ssa.gov/oact/STATS/table4c6.html.
Public health officials of the nineteenth century knew this. As such, officials used infant mortality as a “barometer for the public health” of neighborhoods, cities, and regions.16

In New Orleans, the rate of black infant mortality remained similar to white infant mortality until the 1890s. However, decreases in white infant mortality and increases in black infant mortality in the 1890s resulted in a rapid divergence. This pattern is clearly visible in Chart 3 and Chart 4.

Chart 3. Infant Mortality Rates by Race, 1880–1915. This chart illustrates the divergence between black and white rates in the 1890s. Data Source: Death Certificate Index

16 For example, Ernest Hart, *The Sanitary Record*, vol. 2 (London: Smith, Elder & Co., 1875), 423 “…children are specially sensitive to any unwholesome surroundings, they may be looked upon as a delicate hygienic barometer in indicating the sanitary conditions of a neighbourhood.” This held true throughout the period. Thirty years later, the Louisiana State Board of Health Report stated, “the progressive reduction in the death rate of infants, that ‘barometer of public health...’” *Biennial Report of the Louisiana State Board of Health. 1906/07*, 120.
Although diarrheal diseases, recorded as enteritis, inflammation of the bowels, chronic diarrhea, or cholera infantum (not to be confused with *Vibrio cholera* — Asiatic Cholera) caused seven percent of the deaths in the population older than one year, they caused 16 percent of infant deaths in 1880.\(^{17}\) Five years later, this proportion increased to 21 percent and by 1890, it reached 24 percent of the total infant deaths.

The mortality rates for white infants had a strong positive correlation with the diarrheal rates for white infants.\(^{18}\) The mortality rates for black infants remained similarly correlated to the

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\(^{17}\) Combination of A04, A09, and K52 (ICD10 codes).

\(^{18}\) Positive correlation between the monthly number of white infant deaths and white infant deaths from diarrhea. \(r^2 = 0.79; \ p < 0.0001\) (n=96); This finding supports Condran and Lentzner’s assumption in “Early Death: Mortality among Young Children in New York, Chicago, and New Orleans,” *The Journal of Interdisciplinary History* 34, no. 3 (January 1, 2004): 315–54. to an extent. Condran and Lentzner (2004) suggest that overall mortality patterns reflect infantile diarrhea mortality patterns. I found that this was true for white infantile diarrhea throughout this period but not entirely true for black infantile diarrhea. The next chapter, “Mortality Terrain,” provides evidence for this difference.
diarrheal rates for black infants until around 1905. Several different factors affected infant diarrheal rates during this period, but leading causes related to poor sanitation and contaminated food and water.

Tetanus neonatorum (A33), historically known as trismus nascentium, ranked as the second most prevalent cause of death (14 percent) among infants in 1880 (see Chart 5). Tetanus neonatorum is a neonatal bacterial disease contracted during birth in unsanitary conditions (often through a contaminated tool used to cut the umbilical cord). Deaths from tetanus occurred typically in the first two weeks after birth. Physicians recognized unsanitary birthing conditions as the cause of neonatal tetanus before 1880, even though the bacteria, *Clostridium tetani*, was not isolated and identified in humans until 1889.

The Board of Health reported the rate of tuberculosis deaths among infants as almost nil although some primary infectious were likely reported as pneumonia instead of tuberculosis. Tuberculosis deaths for children and youths were low as well, but public health officials attributed the low numbers to frequent misdiagnosis. Many of the tuberculosis milk-related deaths were

19 Positive correlation between the monthly number of black infant deaths and black infant deaths from diarrhea. \( r^2 = 0.71; p < 0.0001 \) (n=96)
20 Chapter 3, Mortality Terrain, examines environmental causes of mortality differentials.
21 The same bacillus causes tetanus neonatorum and traumatic tetanus, the latter of which commonly is associated with stepping on a rusted nail.
22 Age at death for both black and white infants who died from tetanus neonatorum averaged 9 days.
recorded as diarrhea and pulmonary tuberculosis deaths as bronchitis or broncho-pneumonia. As such, changes in the rates of tuberculosis deaths among infants are more difficult to assess. Still, post-primary tuberculosis killed slowly, often wearing down the victim over a span of years, and thus, tuberculosis killed very few people before their first birthday. Other bacterial diseases had an outsized effect on infants. Infants died at the highest rates from diarrheal diseases, neonatal tetanus, pneumonia, and malnutrition.

The differences in black and white rates of diarrheal diseases and pneumonia increased from 1880 to 1915 (Chart 5). Black rates remained high while white rates decreased. The differences in rates of tetanus and malnutrition increased as well, but then began to converge in the early-twentieth century. The rates of diarrheal diseases and pneumonia comprised the majority (72 percent) of the difference between the overall black and white infant mortality rates in 1915.

Physicians are much more careful in their certificates of cause of death and indeed until science has rendered it less difficult to obtain precise information in many instances” (pg. 33). After medical researchers developed a diagnostic test in 1909 (not to be confused with lab tests for tuberculosis-contaminated milk, which appeared in the 1890s, or the chemical tests for adulterated milk developed in the 1880s, or the anti-adulteration of milk ordinance passed in 1879), official records show an increase in the number of infant deaths attributable to tuberculosis.

From Roberts’ *Infectious Fear*, 20, “Humans may contract the bovine form through the ingestion of bovine tissue or cow’s milk in which the bacillus is present. After the turn of the century, however, and particularly after the implementation of milk-purification regulation, most human tuberculosis was caused by the human form of the bacillus, the mycobacterium bacillus, or M. tuberculosis. Most often, M. tuberculosis originated with inhalation, producing pulmonary tuberculosis.”

Post-primary tuberculosis is reinfection after surviving the initial infection. Roberts (Roberts, 22.) states, “Primary infection is more likely to be fatal in very early infancy (before six months) or in adults (risk increasing with age). Children over the age of two stand a very high chance of surviving primary infection, and those between two and five (and even as late as ten) may experience these symptoms as nothing more than a very bad cold.”

Chart 4 shows that in 1915 the black infant mortality rate was 102% higher than the white infant rate. The black infant mortality rate for diarrheal diseases was 35% higher, and for pneumonia, it was 38% higher. Combined, diarrhea and pneumonia rates were 73% higher for black infants than white infants (72% of the total differential). The early neonatal (7 days) differential was 20% higher for black infants, primarily from premature birth and tetanus with almost no diarrheal or pneumonia-related deaths.
Chart 5. Infant Mortality Rates by Race for the Most Prevalent Diseases, 1880–1915. These charts show the black and white infant mortality rates by leading causes of death. The disease-specific rates are stacked, which means the two charts also show the overall mortality rates for black and white infants (summation of rates for every cause of death in any given year). Data Source: Geospatial Mortality Dataset.
Identifying the seasonality of the mortality disparity helps to pinpoint the differentials in health and to narrow causative factors. Recurrent high mortality rates in specific months and seasons across multiple years indicate seasonal influences on the mortality rates. In the 1880s, white and black infants died at similar rates from causes of death that primarily struck in summer months, namely diarrhea and malnutrition. Tetanus and pneumonia, however, killed black infants at much higher rates than white infants in the winter months. Mortality rates for black infants spiked twice a year in the 1880s and 1890s, while the mortality rates for white infants spiked once a year. Black infant mortality rates in the winter (November to January) exceeded white rates and constituted a large share of the differential in mortality rates. This black infant mortality spike in winter months only began to taper off after 1910.29

Infant mortality spiked in the summer months for both black and white populations. The summer spike in mortality was comparable for black and white infants from 1880 to 1896. During this period, infant mortality patterns show that environmental conditions connected with mortality did not differ by race as much as in later years, and as a result, the black and white residents of the city experienced similar mortality rates. After 1896, the summer spikes for white infants declined while the summer spikes in black infant mortality increased to unprecedented levels until 1901. From 1901 to 1908, the black infant summer mortality rates fluctuated, but never reached the levels of the late 1890s.

29 Black infants died at higher rates in winter months when smallpox was endemic in the city. The charts are available in Appendix D.
Furthermore, the rates at which black and white infants died from diarrheal diseases followed a similar seasonal pattern. The highest rates of diarrheal deaths occurred in the summer months. White infant mortality peaked in May and black infant mortality in June (see Appendix C, Chart 36). The deaths in May and June alone accounted for 46 percent of all the black infantile diarrhea deaths and 49 percent of all the white deaths from this cause. The average age of death during the summer—six months—was older than the annual average—five months.\textsuperscript{30} Older average ages of infant deaths in the summer suggest an infant’s summer was a critical and precarious time. In this time, months with higher death rates tended to

\textsuperscript{30} Comparison of means, t-test: difference 19 days, p-value < 0.0041
have older average ages of death and vice versa. For instance, the average age of death in December, the month with the lowest average mortality rates, was 2 months younger (than the annual average) for black infants and 3 months younger for white infants.\textsuperscript{31} In these pre-transition years, the monthly mortality rates were comparable as well, demonstrating some level of hazard/disease-exposure similarity.

As the city population continued to outgrow the nonexistent sanitary infrastructure, the 1890s became a period of deteriorating health conditions for all.\textsuperscript{32} White infant mortality rates from diarrheal diseases nearly doubled in the 1880s, rising from 357 deaths per 10,000 to 659 deaths per 10,000 white infants. The black infant mortality rate for diarrheal diseases matched the white rate by the end of the decade and surpassed the white rate in the early 1890s.\textsuperscript{33} White rates

\textsuperscript{31} Average month age at death for white infants in the 1880s (1880, 1885, 1890)

- Black: annual average = 159 days; May = 192 days; June = 180 days; July = 155 days; December = 120 days
- White: annual average = 147 days; May = 166 days; June = 170 days; July = 182 days; December = 97 days

\textsuperscript{32} It was also a period of improving vital statistics, which likely explain some of the steep mortality increases. See Footnotes 8–11. See Colten (2006) on the urban expansion and landscape transformation projects during this period.

\textsuperscript{33} Although the black rate dropped in 1885, this decrease was the result, in part, of the 1883-84 smallpox epidemic, which had a greater effect on the smaller black population. This drop may have been caused by a lower black birth rate in the years following the 1883-1884 smallpox epidemic. Chart 2 shows the large spike in smallpox deaths during the epidemic. Appendix C, Chart 34 shows the results of this epidemic: more deaths in the child-bearing age group in 1883-1884. Subsequently, the black infant deaths decreased until 1889.

Over the decade, black and white deaths per year peaked in 1883 due to the smallpox epidemic. Smallpox deaths in the two years leading up to the epidemic totaled 6 deaths. The number of smallpox deaths jumped to 415 in 1882, 1,266 in 1883, and 292 at the tail of the epidemic in 1884. Since these years were not part of the sample years, I do not have race-specific mortality data for the smallpox epidemic, but overall, black mortality increased during the epidemic and other diseases made up a smaller percentage of the total deaths. Overall, diarrheal diseases reached a decade-low of 590 deaths per 10,000 people in 1883.

In the first wave of the epidemic, smallpox was responsible for 7 percent of the deaths. Smallpox was responsible for 17 percent of deaths (1,266 deaths) at the height of the epidemic. Smallpox hit the black population hardest February through April of 1883 and the white population seven months later from November to January of the following year. Louisiana Board of Health and Joseph Jones, \textit{Contagious and Infectious Diseases: Measures for Their Prevention and Arrest; Small Pox (Variola) Modified Small Pox (Varioloid) Chicken Pox (Varicella) Cow Pox (Variolae Vaccinnae) Vaccination, Spurious Vaccination. Prepared for the Guidance of the Quarantine Officers and Sanitary Inspectors of the Board of Health of the State of Louisiana} (L. Jastremski, state printer, 1884).
of diarrheal deaths peaked in the early 1890s and then declined.\textsuperscript{34} Between 1890 and 1894, black rates of infantile diarrhea increased from 640 deaths per 10,000 people to 770 per 10,000. Black rates peaked in the late 1890s and declined until 1905. After 1905, the black rates began to slowly rise again, reaching 674 deaths per 10,000 in 1915.

Although rates for both populations declined through 1905, the rate of white infantile diarrheal deaths declined much more rapidly than did the rate of black infantile diarrheal deaths. Annual black mortality rates from diarrhea averaged 42 percent higher than white rates between 1894 and 1905. The differentials in mortality rates were not caused by irregular outbreaks of diarrheal diseases in the black population. If this was the case, the seasonal pattern of diarrheal deaths would have changed, yet these patterns remained the same between 1894 and 1905. Black rates increased during the usual May-June summer spike while white rates remained similar for May and decreased slightly for June.\textsuperscript{35} Although the white death rate declined and the black rate remained higher than in the earlier years, the share of deaths in May and June remained stable, increasing slightly to 50 percent for white infants and 48 percent for black infants.

By 1915 black infants died at more than twice the rate of white infants (see Chart 4). The divergence in rates of black and white infant deaths increased in the late 1890s and again after 1905, and it was largely due to diarrheal diseases. Black infants died from diarrheal diseases at a rate 1.42 times higher than the rate for white infants in 1894-1905. This differential increased to 1.54 in 1910 and 2.09 in 1915. This means that for every 209 black infants who died from diarrheal

\textsuperscript{34} See Chart 3; Death Certificates Index. Since white infant mortality correlates with white infantile diarrhea mortality rates, for the years interceding the sample years, the index dataset approximates the infantile diarrhea patterns. White infant mortality spiked in 1890, 1892, 1896, and 1899, while still trending downward.

\textsuperscript{35} Change between 1880s and 1894-1905:

Black: overall = +4.7%; May = +2.16%; June = +0.4%; July = +0.8%

White: overall = -2.5%; May = -0.4%; June = -0.8%; July = -0.3%
diseases in 1915, if they had had the same advantages as white infants, more than half (109) of these black infants would have survived.

Meanwhile, the seasonality of infant deaths flattened, becoming more evenly distributed across the calendar. In 1910-1915, the share of infantile diarrhea deaths in May and June dropped from 48 percent (1894-1905) to 34.3 percent for black infants and from 50 percent (1894-1905) to 36.2 percent for white infants. Although May and June remained the peak months between 1910 and 1915, the proportion of black infant deaths in August through December were distributed within 2 percent or less of the expected value for each month. White infant deaths from diarrhea remained slightly more seasonal between 1910 and 1915, but overall averaged half a percent lower than black rates each month from May to December.

Diarrheal mortality rates for black infants exceeded those of white infants in the summer (May, June, July) and fall months (September, October) between 1894 and 1905. In the 1910s the summer mortality rates decline for both black and white infants, but the fall rates did not decline for black infants. Instead, these rates increased slightly to around 50 deaths per 10,000 people for each month, August to December. For these months, white rates remained around 25 deaths per 10,000. The lack of parity in infant mortality rate declines demonstrates increasing differences in the health experiences (socio-environmental inequalities). Compared to white infants, mortality burdens from diarrheal diseases became much higher for black infants by the 1910s.

Recent studies have found strong correlations between rises in temperature and dysentery in developing areas. For each 1-degree (Celsius) increase, dysentery incidence increases approximately 10 percent.\(^36\) Condran and Lentzner state, “[t]he summer heat not only multiplied

the bacteria in the environment but also increased the likelihood that young children would die from losing fluids through perspiration; replenishing fluids, in the nineteenth century, often introduced new pathogens through contaminated water.”

Scholars have identified the decline in summer deaths of infants from diarrheal diseases as a main factor in the mortality transition. But, in New Orleans, the average summer mortality rate only decreased for white infants. Black infants died in the summer from diarrheal diseases at higher average rates in the 1910s than in the 1880s. Declines in white infant diarrheal death rates in the summer indicated substantial healthier environments, while the lack of lower rates of black infant deaths from diarrheal diseases shows greater inequality in disease burdens.

The seasonal pattern of diarrheal deaths in the summer weakened from 1880 to 1915, while the winter seasonality of pneumonia strengthened. Diarrheal disease rates diverged by race in the 1890s, while the major divergence in pneumonia infant mortality rates occurred after 1905. Unlike the similarity in diarrheal deaths by race in the 1880s, pneumonia rates were uneven in the 1880s and became increasingly unequal through the first decades of the twentieth century.

Pneumonia caused a low proportion (6.6 percent) of infant deaths in 1880. Still, pneumonia ranked consistently in the top three causes of death for black infants, while for white infants it did


39 Possible exogenous factors played a role also, as New Orleans was not a closed system. The large influx of African American migrants in the 1910s may have skewed the mortality rates to some extent, yet the subsequent chapter argues that these rates were connected with environmental hazards in the low-lying areas of the city. Residential segregation confined black residents within these low-lying, environmentally burdened areas of the city, which had deadly health consequences.
not reach the top three until 1900. Black infants died from pneumonia at twice the rate of white infants. In 1880, pneumonia caused 9.5 percent of black infant deaths at a rate of 264 deaths per 10,000 black infants compared to five percent of white infant deaths at a rate of 107 deaths per 10,000 white infants.

![Chart 7. Infantile Pneumonia Mortality Rate by Race. Black rates (blue line) increased while white rates (red line) remained level. Source: Geospatial Mortality Dataset](image)

Distinct monthly patterns did not define pneumonia in the 1880s, and seasonality by race varied little. The first six months of each year had the highest rates for both black and white infants. Sixty-five percent of pneumonia deaths occurred in the first six months (expected value 50 percent), spread across winter, spring, and early-summer months.
Black infant mortality rates from pneumonia increased gradually from 256 deaths per 10,000 to 379 deaths per 10,000 between 1890 and 1905. After 1900, black infants died from pneumonia at increasingly disparate rates compared to white infants. This divergence increased sharply between 1905 and 1910, during which time black infant pneumonia mortality rates spiked, rising to 620 deaths per 10,000, while the white infant mortality rates remained below 200 deaths per 10,000 throughout the entire period. In the 1910s, pneumonia caused (1 in 4) nearly 25 percent of black infant deaths at an average mortality rate of 572 deaths per 10,000, while it caused 15 percent of white infant deaths at a much lower average rate of 156 deaths per 10,000.

Chart 8. (A) Monthly Infant Mortality Rate (J18) by Race, 1890–1900, and (B) Monthly Infant Mortality Rate (J18) by Race, 1905–1915. The seasonal patterns of pneumonia (J18) increased substantially among black infants (blue line) after 1900, while white infant rates (red line) did not experience a similar increase. Date source: Geospatial Mortality Dataset

In the 1890s, white and black infant mortality from pneumonia shared almost identical seasonal distributions. Deaths from pneumonia shifted more towards winter months after the 1880s, with October-March comprising 68 percent of black deaths and 65 percent of white deaths. Yet, the white rates remained flat across the months while the black rates increased significantly in the winter months (Chart 8A).
The seasonal similarities of white and black deaths ended in the 1900s. Black infant mortality from pneumonia increased drastically during the winter and spring months of 1905-1915. Black deaths showed very strong seasonal correlations, with the greatest proportion from November-April (Chart 8B). Black deaths in these 6 months of winter and spring comprised 72 percent of the total annual deaths, while only 54 percent of white infantile pneumonia deaths occurred — only 4 percentage points higher than expected. White deaths from pneumonia occurred in greater monthly proportions than black deaths from May to September, due to dips in black mortality rates rather than increases in white rates. From 1880-1915 pneumonia remained a consistent cause of unequal mortality between black and white infants.40

Unlike infantile diarrhea and pneumonia, tetanus neonatorum and marasmus declined for both black and white infants between 1880 and 1915. Black and white rates of tetanus neonatorum and marasmus approached zero in 1915, although the decline for white infants occurred much earlier than for black infants. Furthermore, the seasonality of these causes of death provide clues to the causes of the disparities.

40 This increase may have been connected, in part, with poor ventilation in homes. Poorly-ventilated homes burning solid fuel significantly increase risk for pneumonia in children under 5.

World Health Organization states, “More than 50% of premature deaths [in 2012] due to pneumonia among children under 5 are caused by the particulate matter (soot) inhaled from household air pollution.” (Factsheet 292, “Household Air Pollution and Health.”)

The temperature in the winters of 1905, 1910, and 1915 averaged 1.8 to 3.7 degrees (F) colder than the average winter temperature for the city, 1889-1994. Colder winter temperatures necessitated more fires for warmth, perhaps causing increases in pneumonia deaths as well. This is a topic for future research.
Infantile Tetanus Mortality Rate by Race

The rate of neonatal tetanus for white infants (red line) began to decline in 1890s. The black infant rate (blue line) declined after 1900. Data source: Geospatial Mortality Dataset

Tetanus neonatorum ranked as the second most prevalent cause of deaths among infants until 1905. The seasonality of tetanus neonatorum deaths corresponded with the seasonality of births in the city. The greatest proportion of births occurred in the fall and winter, while the fewest births occurred in spring and early summer (see Chart 10B). These patterns relate to the ebbs and flows of the city population around port business cycles and fears of yellow fever in the summers. Babies born in January were conceived the previous April, which was typically just before threats

41 White births peaked from August to October (average 9.7% of total births each month) and black births in August/September and December/January (average 9.5% of total births). White and black births were low (average 6% of total births) in April, May, and June.
of yellow fever menaced the city. Furthermore, babies born in August were conceived in November, the end of the seasonal threat of yellow fever.42

The mortality rate for infantile tetanus remained consistent for white infants and increased for black infants in the 1880s. Medical textbooks from the time noted that the disease was more prevalent with black newborns than with white newborns, and New Orleans was no exception.43 Black infant deaths from the disease fluctuated around 15 percent of the total black infant deaths at a death rate of 4 to 5 percent from 1880 to 1900. The black rate was twice the rate of white infant mortality from tetanus. The percentage of white deaths from tetanus gradually decreased from 13 percent to 11 percent at a mortality rate around 2.5 percent between 1880 and 1890. White and black rates of tetanus diverged significantly beginning in the early 1890s.

The distinct seasonal patterns of infantile tetanus in the 1880s gave way to instability in the 1890s. White rates declined by over 50 percent between 1894 and 1905, and the autumn rise in mortality became minimal. Black rates increased, however, in the late-spring and summer months and remained elevated through October.44

42 The yellow fever season was really the Aedes aegypti season — the primary arthropodal (mosquito) vector of yellow fever. A. aegypti become inactive and do not feed (mode of yellow fever transmission) in cold temperatures.


44 Black infantile tetanus rates were also elevated in January.
Between 1900 and 1905, the black rates of tetanus began to decrease. This corresponded with changes in medicine, care, and facilities at Charity Hospital. In the late 1890s, Charity Hospital worked to develop an antitoxin treatment program for tetanus. Physicians attested to the value of this treatment in 1896 and stated, “Our hospital mortality from this disease will be very materially reduced in both races by this treatment.” In 1900 the House Surgeon of Charity Hospital reported, "The Colored Maternity Service had been more fully equipped with requirements of asepsis." The obstetrics ward at Charity Hospital delivered babies from 248 mothers in 1900, over half of whom were women of color (N=136; 10 percent of total black births in the city). This marked a large increase from the low of 56 births (5 percent of total births) to women of color at Charity Hospital nine years earlier.

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46 *CHR* 1900, pg. 32
Chart 11. Births at Charity Hospital by Race, 1885-1915. Black births (blue line) in Charity Hospital outpaced white births (red line) at the end of the first decade of the twentieth century. This matched the decline in black infants deaths from tetanus neonatorum. Data Source: Charity Hospital Annual Reports

Chart 12. Average Distance of Tetanus Neonatorum (A33) Deaths to Charity Hospital. Based on spatial analysis of tetanus neonatorum deaths, this chart shows that the black infants who died from the disease lived increasingly farther away from Charity Hospital. White mothers used Charity Hospital less and less for child birth.
In 1903, the hospital continued to expand service to women and children of color—both in gynecological services (offered in the new ward) and pediatrics (in a new children's ward). Charity Hospital’s expansions of services began to show results over the first decade of the twentieth century. Women and newborns of color particularly benefited, with black births in the hospital outpacing the white births in 1909.\textsuperscript{47} In a five-year span, 1905 to 1910, black births at Charity Hospital increased from 8 percent (120/1478) to 13 percent (205/1612) of total estimated black births (Chart 11), and the mortality rate of tetanus neonatorum (Chart 9) for black infants dropped by more than 50 percent. Moreover, Chart 12 shows that the black infants who died from tetanus neonatorum in the later years increasing lived farther away from Charity Hospital. The percent of white mothers who used Charity Hospital for childbirth declined during this period, and the death certificates show a total of 21 white infants died from tetanus neonatorum in 1910 and 1915 combined. These two facts explain the break in the geographic link between distance away from Charity Hospital and tetanus neonatorum deaths. The matching timelines of increases in the percentage of black babies born in Charity Hospital, implementation of antiseptic in the delivery rooms at Charity Hospital, increases in the distance from Charity Hospital of tetanus neonatorum deaths, and the large declines in the rate of tetanus neonatorum among black infants, suggest causality.

From 1910 to 1915, white tetanus neonatorum nearly disappeared city-wide, finishing 1915 with a mortality rate of 14 deaths per 10,000. Black rates declined significantly, but ended the year

\textsuperscript{47} Throughout this period, three percent of white babies born in New Orleans were born in Charity Hospital. Carolyn Leonard Carson found that black women used doctors and hospitals for childbirth soon after they arrived in northern cities during the Great Migration. Based on the maternity ward records from Charity Hospital, this seems to have been the case in New Orleans as well. City-wide, there were few hospitals from which to choose. For a history of Charity Hospital, see John E. Salvaggio, \textit{New Orleans' Charity Hospital: A Story of Physicians, Politics, and Poverty} (LSU Press, 1992). For more on black childbearing in hospitals, see Carolyn Leonard Carson, “And the Results Showed Promise... Physicians, Childbirth, and Southern Black Migrant Women, 1916-1930; Pittsburgh as a Case Study,” \textit{Journal of American Ethnic History} 14, no. 1 (1994): 32–64.
at 106 deaths per 10,000, with most of the deaths having occurred in August and September, mirroring the original (1880-1890) seasonality of the disease. The advances in medical knowledge, care, and facilities improved birthing conditions and neonatal care, contributed to these declines.\textsuperscript{48}

Severe malnutrition, also known as marasmus, caused five percent of the total infant deaths in 1880. This made it the fifth leading cause of death overall, the fourth among white infants and the seventh among black infants. Despite the rankings, the black and white infant mortality rates for malnutrition were similar in the 1880s. Both rates doubled over the course of the decade and

48 By 1915, Charity Hospital attended to 23 percent of all babies of color born in New Orleans.
showed strong seasonal patterns, peaking in May and June each year. The white rate peaked in 1890 at 229 deaths per 10,000. As with infantile diarrhea and tetanus, the white rate began to decline in the early 1890s while the black rate did not drop until the second half of the decade.

Marasmus and infantile diarrhea shared a similar seasonality as well, peaking in May and June. Poor quality food and drink could cause malnutrition, diarrhea, or both. The seasonal effects of marasmus mortality remained evenly distributed between black and white infants in the 1880s. Black infants died in slightly higher percentages than white infants in October, January, and February while black rates dropped sharply in March and September.

Additionally, deaths from malnutrition followed the ebbs in the New Orleans business cycle. November through April marked the most active periods of business in New Orleans and business slowed to a crawl in the summer month. After harvest, cotton shipments through the city created seasonal highs of economic activity in the winter. The threat of yellow fever and the high temperatures in summer months encouraged people, who had the means and mobility, to spend the summers away.

The moribund business of the summers led to scarcity in employment opportunities. This meant seasonal instability for laborers, yardmen, loaders, and longshoremen among the many other occupations connected with the port. Low summer income lessened the purchasing power of the

49 Other oral pathway could introduce the disease to an infant’s body as well (e.g., a baby puts a contaminated object in their mouth).

50 For the antebellum period, Walter Johnson discusses this business cycle in Soul by Soul: Life inside the Antebellum Slave Market (Cambridge, Mass.: Harvard University Press, 1999), 49. While he explains the seasonality in relation to the slave markets of New Orleans, the seasonality of harvests for cotton shipments did not change after emancipation.

lower-class population in New Orleans over the summer. As quoted in Arnesen, both black– and white– union leaders claimed,

‘We poor men can not [sic] take so much a single dollar from our daily wages to aid or support a sick wife or child.’ Moreover, the seasonality of dock work - a theme that for decades would continue to dominate discussions of wage scales on the waterfront — meant that longshoremen could obtain employment only for a ‘few months in the year.’

This occurred at the same time of year that the risk of contaminated food increased substantially with the rising temperatures of summer.

In addition to food quality and availability, patterns of breastfeeding influenced the seasonal patterns of infant mortality. As the largest monthly cohort of infants, those babies born between August and October of each year, grew older, breastfeeding tapered off. Public health officials recognized the strong connection between artificial feeding and infant mortality, and they warned against the risks of artificial feeding. By May, the largest cohort of infants born, between August and October, ranged in age from seven to nine months old. Around this stage of development, breastmilk no longer provided all an infant’s nutritional requirements. This necessitated solid foods in the infant’s diet, and thereby increased pathways for disease.

In summary, both black and white babies died at high rates from infantile diarrhea in New Orleans. The clear seasonal variation and these seasonal conditions, especially weather, match

54 “Weaning from the Breast,” *Paediatrics & Child Health* 9, no. 4 (April 2004): 250. “Delaying the introduction of solid foods much beyond six months of age is also likely to put the infant at risk for iron deficiency anemia and other micronutrient deficiencies.”

Contemporary studies have found incidence of marasmus increases from the 0-5 month age group to the 6-11 month group, regardless of seasonality. For example, see Hallgeir Kismul et al., “Incidence and Course of Child Malnutrition According to Clinical or Anthropometrical Assessment: A Longitudinal Study from Rural DR Congo,” *BMC Pediatrics* 14, no. 1 (January 28, 2014): 22, https://doi.org/10.1186/1471-2431-14-22.
modern studies on the seasonality of infantile diarrhea and dysentery. As we shall see in the next chapters, rates of infantile diarrhea responded to changes in the environment and in public health infrastructure, as well as to access to uncontaminated water in months with large bacterial loads. Divergence in black and white rates in the 1890s and again in the 1910s are correlated, as we shall see, with the new drainage systems that opened new land in the city. Black and white spatial separation and eventual segregation in the city allowed for further divergences in environmentally-connected mortality rates.

Tetanus neonatorum occurred in the first two weeks after birth. The seasonality of tetanus neonatorum again showed the connection to the seasonality of births. Rates reflected the state of medical knowledge and access to sanitary birthing conditions. These rates converged in the later years, coinciding with the improvements in care and use of Charity Hospital by black mothers.

Marasmus exhibited strong patterns tied to age, which related to food availability and quality. As infants aged and their mothers weaned them from breast feeding, mortality from marasmus increased. Modern patterns of mortality from marasmus, with increased incidence at ages 6-11 months, match the patterns in New Orleans, yet the seasonality of marasmus varies due to local conditions. These variances relate to differences in dry and wet seasons, economic cycles unique to each city, food availability and quality, and possible comorbidities or misdiagnosed enteric diseases. From 1900 to 1910, black infant mortality from marasmus diverged from white mortality. By 1915, the rates converged just above zero. The period of divergence had strong seasonality with the summer, with slightly elevated rates of black infant deaths from marasmus in December and January. This differed from the pattern of infant deaths caused by diarrheal diseases, which did not increase in rate during the winter.
Pneumonia rates diverged for black and white infants from 1905-1915. Black deaths from pneumonia fell into a strong seasonal pattern. Pneumonia death rates for black infants increased in winter and spring months. While the seasonality is typical, even today, of pneumonia incidence, the large influxes of rural labors who crowded into boarding houses and other lodgings during winter months, thereby increasing population density, created more pathways for infants to contract pneumonia. Black adults died from pneumonia at increasingly higher rates in the mid-1890s, which coincided with the gradual increases in black infant mortality from pneumonia. Additionally, from 1905 onward, black adult deaths fell into a strong winter seasonality, matching the pattern of black infant deaths from pneumonia.

**B. Adult Mortality**

The diseases of infancy in New Orleans did not change radically over the mortality transition, instead, the rates at which infants died from these diseases declined. This differed from adult patterns, in which infectious diseases gave way to chronic diseases. Diseases that caused the greatest disparities in the black and white adult mortality rates were different from the diseases with the biggest disparities between black and white infants. Rates of adult deaths from diarrheal diseases averaged below five deaths per 10,000 throughout the study period. Malnutrition and tetanus killed a minuscule number of adults.

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56 Infants rarely die from chronic diseases because chronic diseases, by definition, take a long time to contract and/or to progress, and infants are only infants for a year.
Chart 14. Adult (15-45 years) Mortality Rate by Race, 1880-1915. In the 1880s, the black and white mortality rates were disparate but positively correlated. Years in which black adults died high rates were the same years in which white adults died at high rates (and vice versa). In the late-1890s, however, the black and white adult mortality rates began to diverge. By 1915, the black rate was more than double the white rate. Data Source: Death Certificate Index

Adults died at an average annual rate of 120 deaths per 10,000 adults from 1880 to 1895. These averages remained consistent over the course of the period studied, while the mortality rate of tuberculosis declined for both races. Infants and children endured large burdens from disease. People who survived into adulthood, especially while remaining in the same disease environment, acquired resilience to many of the survived maladies and afflictions. As such, the prevalent causes of death for adults differed from those of infants and children, and seasonal factors had a much lower effect on the rates at which adults died. Adults died from tuberculosis, pneumonia, heart disease, and malaria. When present in the city, epidemic diseases caused spikes in the adult mortality rate as well (e.g., smallpox 1884, 1899-1900; yellow fever 1905).
Death from pulmonary tuberculosis, at the time known as phthisis pulmonalis or consumption, indicated proximity to infected people, lack of prior exposure, poor living conditions, and/or poor environment.\textsuperscript{57} Humans are susceptible to bovine tuberculosis as well. This strain of tuberculosis infected through proximity to cows, tainted beef, and/or contaminated milk.\textsuperscript{58} Physicians recognized the threat of tuberculosis, but they lacked effective methods to treat the disease. As one physician noted in 1881, “Phthisis is certainly one of the most destructive of diseases. In no case can recovery be anticipated.”\textsuperscript{59} In the United States, the 1880 census reported that tuberculosis caused 12 percent of all deaths and 14 percent of deaths in cities.\textsuperscript{60}

Tuberculosis in New Orleans caused 15 percent of deaths at a rate of 350-400 per 100,000 people in 1880. On average, tuberculosis victims died at age 37 in the US and at age 35 in New Orleans. Black victims died slightly younger, at age 32, while white victims averaged 36 years.\textsuperscript{61} A higher percentage of black deaths (18 percent) were attributed to tuberculosis than were white deaths (13 percent).\textsuperscript{62} The death rate of tuberculosis peaked in 1884, with just under 1,000 deaths in the year (958) and a rate of 423 per 100,000.

\textsuperscript{57} Samuel Roberts (Roberts, Infectious Fear, 22.) states, “Massive exposure or a weakening of the body (by age, other diseases, mental or physical stress, malnutrition, or continual overexertion), however, may prevent the nodule from fully healing.”

\textsuperscript{58} Roberts (Roberts, 20.) states, “Humans may contract the bovine form through the ingestion of bovine tissue or cow’s milk in which the bacillus is present. After the turn of the century, however, and particularly after the implementation of milk-purification regulation, most human tuberculosis was caused by the human form of the bacillus, the mycobacterium bacillus, or M. tuberculosis. Most often, M. tuberculosis originated with inhalation, producing pulmonary tuberculosis.”


\textsuperscript{60} “Tenth Census of the United States” (Washington, D.C.: U.S. Department of the Interior, 1881) Volume 12, pg. LXIII. Other contemporaneous medical textbooks estimated that tuberculosis caused as many as 25 percent of all deaths. See Hartshorne, Essentials of the Principles and Practice of Medicine, 231.

\textsuperscript{61} Comparison of means, t-test: difference 1824 days (5 years), p-value < 0.0002

\textsuperscript{62} The contemporaneous association between tuberculosis and black people may have led to over reporting and diagnosis of tuberculosis in black communities. See Roberts, Infectious Fear; Tera W. Hunter, To ‘joy My Freedom: Southern Black Women’s Lives and Labors After the Civil War (Cambridge, Mass.: Harvard University Press, 1997).
Two years after the 1880 US Decennial Census reported tuberculosis as the biggest killer in New Orleans, Robert Koch published a paper identifying a type of bacteria, tuberculosis bacillus, as the disease agent. Koch’s work contributed to the commencement of the bacteriological revolution, but it wasn’t until the sanitary efforts of the 1920s and 1930s, followed by the discovery of streptomycin and other antibiotics in the 1940s, that tuberculosis was finally brought under control in the United States.

Public health initiatives in New Orleans focused on preventative measures to target tuberculosis. Early efforts by the Board of Health centered on sanitarian ideals, as officials worked to remove filth and other contaminants while opening areas to clean air and sunlight. An 1881 medical textbook stated, “under improved hygiene and medical treatment, the mortality from phthisis appears to be declining.” Health officials conducted sanitary inspections of properties, privies, and drinking water, attempted to remove livestock from within the city limits, notified and fined property owners for health violation, and fumigated and disinfected properties. The Board of Health developed sanitary regulations to limit dairy farms in the city and tested milk for contamination. While many public health initiatives targeted tuberculosis at this time, these efforts showed small gains.

In the first years of the new century, medical research on tuberculosis continued to advance, yet tuberculosis remained the leading cause of death in the United States as well as New Orleans.

64 Roberts, *Infectious Fear*.
65 Duffy, *The Sanitarians*.
68 Condran and Lentzner.
In a 1901 paper, Koch identified a distinct strain of tuberculosis found in cows, and city health officials increased their efforts in campaigns against contaminated milk. In 1909 a physician characterized tuberculosis as, “the most universal scourge of the human race.” Tuberculosis caused approximately 10 percent of deaths nationwide, yet remained at 15 percent in New Orleans, unchanged from the share of deaths reported in 1880.

From all causes of death, black adults had higher mortality rates than white adults. Between 1880 and 1895 differentials in mortality rates for black and white adults ranged from a high of 31 percent in 1882 to a low of 13 percent in 1886. On average the black mortality rate was 30 percent higher than white mortality rates. Although black adults had consistently higher mortality rates than white adults, the rates remained closely connected from the 1880s to the mid-1890s. Similarly, in years when the TB mortality rate for black adults increased, so too did the rate for white adults. This strong positive correlation suggests similar epidemiological conditions.


70 For 1880 to 1895: WhiteAdult = 0.562985*BlackAdult + 0.0026609; \( r^2 = 0.76 \); p-value = <0.0001. Future analysis will test these correlations by month.
Chart 15. Adult Tuberculosis Mortality Rate by Race, 1880-1915. Black adults (blue line) died at much higher rates from tuberculosis than white adults (red line). Data Source: Geospatial Mortality Dataset

Tuberculosis created the largest proportions of differences in black and white mortality rates. From 1880 to 1895, tuberculosis caused 43 percent of adult deaths in the black population at a rate of 61 deaths per 10,000 people. Over the same period, tuberculosis caused 34 percent of deaths in the white population at 36 deaths per 10,000. Black rates averaged 1.7 times higher than white rates. No other causes of death averaged more than 10 percent of the total deaths in these years.

Some causes of death had low rates in the pre-transition years but contributed to the divergence in rates in later years. These causes of death included pneumonia, which killed an annual average of 10 black adults in 10,000 and 8 white adults. Malaria was recorded as a
secondary cause in many deaths but as the primary cause in only 7 black and 6 white deaths per 10,000. In the early years, a very small number of deaths resulted from heart disease or physical trauma. Heart disease killed less than 2 black adults and 1 white adult per 10,000 black/white people. Physical trauma killed less than 1 black adult and 3 white adults per 10,000 black/white people.71

The year 1896 marked the first time the mortality rate of black adults exceeded the rate of white adults by more than 50 percent. Black mortality peaked in 1900 at 200 deaths per 10,000 annually, however the black and white rates had only begun to diverge. The differential rate remained near 50 percent in the first five years of the new century. A yellow fever epidemic in 1905 killed white adults disproportionately, resulting in a brief drop of the black-white rate differential to 34 percent. After which, the black mortality rate continued to increase, averaging more than twice the white rate from 1906 to 1915. By the 1910s, the black rate consistently doubled the white rate. By 1915 the differential reached an unprecedented level — the mortality rate for black adults exceeded the rate for white adults by 142 percent.

After 1895 the epidemiological conditions for black and white residents changed in New Orleans. The adult mortality rates for the black and white populations diverged in the late-1890s and the rates no longer correlated. The mortality rates of tuberculosis, pneumonia, heart disease, and physical trauma diverged by race, while rates of malarial and diarrheal mortality declined to near zero. Rates of tuberculosis, pneumonia, and heart disease declined amongst white adults while the rates increased amongst black adults. Physical trauma rates increased for both races, but the

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increases for black adults outpaced those for white adults, reaching double the rate of white mortality by 1915.

While tuberculosis decreased in share of total deaths for black and white adults (31 percent of black deaths and 25 percent of white) at the turn of the century, the differential between black and white rates increased. The tuberculosis rate among black adults was double the rate among white adults in 1900. The average black tuberculosis rate stagnated while the white rate continued to decline. By 1915 the tuberculosis rate for black adults approached triple the rate of white adults.\textsuperscript{72}

\textsuperscript{72} See Footnote 62 for the perception of tuberculosis and the black population.
Likewise, pneumonia differentials increased significantly in the late 1890s. Black rates of pneumonia increased from 10 deaths to 15 deaths per 10,000 adults by 1900. White rates remained under 10 deaths per 10,000. In 1915 black rates topped 22 deaths per 10,000 people, while white rates dropped to 4 per 10,000, a black-white rate differential of 5.5 times. Pneumonia was the only cause of death among adults that showed a strong seasonal pattern. Between 1905 and 1915, black adults died from pneumonia in winter months, with the peak of deaths in February. To a lesser extent, white adults experienced elevated rates of pneumonia in the same months, but the peak occurred in March. Elevated rates in winter months matched elevated rates among black infants.
In addition to poor indoor ventilation affecting death rates during cold months, influenza deaths were frequently recorded as pneumonia.73

Physical trauma ranked as the third most common cause of death. Causes of deaths in this category spanned a wide range of afflictions, including drownings, burns, gunshot and knife wounds, and railroad injuries to name a few. Black adults died at a higher rate; 22 black adults per 10,000 died from trauma compared to 15 white adults per 10,000.74

![Chart 17. (A) Adult Trauma Mortality Rate by Race, 1880-1915, and (B) Adult Heart Disease Mortality Rate by Race, 1880-1915 (B). In the 1890s, black adults (blue line) died at increasingly higher rates from trauma than white adults (red line). In the 1900s, heart disease showed a similar increase among black residents. Data Source: Geospatial Mortality Dataset](chart17.png)

Black adults died increasingly from heart disease and other circulatory diseases after the turn of the century while the mortality rate of white adults remained level. Heart disease caused few deaths before 1900. From 1900 to 1915, however, heart disease deaths steadily increased in

73 The 1918 influenza pandemic appeared in the New Orleans’ records as influenza and pneumonia deaths. During this period, physicians classified many deaths from different respiratory infections as pneumonia, so grouping pneumonia and influenza in the dataset would likely overrepresent the prevalence of influenza in many years.

74 Trauma deaths could provide an effective comparison of non-disease environmental hazards; however, these deaths seem to have been severely underreported in earlier records. Additionally, non-disease death and elderly deaths, while perhaps showing the concentrations of different age groups, could be used as an assumed random distribution within a population, which would provide a base-line to test for spatial significance of clustering in infectious disease deaths.
the black adult population, and while earlier deaths from heart disease were likely misdiagnosed, increases in chronic diseases were part of the mortality transition. By 1915 heart disease killed 15 black adults per 10,000 — the same rate as deaths from trauma — while it killed only 3 white adults per 10,000.

People who survived childhood acquired immunities through exposure to common infectious diseases, such as smallpox. Typically, people in the prime of their lives were the most resilient to infectious disease and had the lowest mortality rates. Adults died at the lowest rates of any age group, second only to youth (5-15). Before the mortality transition, elevated adult mortality rates occurred only during epidemic years. High rates of adult mortality could create feedback loops from the loss of labor, which had the potential to weaken or collapse entire societies. At the local level, high adult mortality rates can create family and community instability due to the loss of a parent and/or primary earner.75 While the adult-aged people had some of the lowest mortality rates in New Orleans, the large rate differential between black and white adults exposes the effects of racial residential segregation in determining health experiences and outcomes. The mortality rate for black adults diverged from that of white adults in the late-1890s, negatively affecting the black population and creating increasing disparities between the white and black communities.

While adults died from tuberculosis, heart disease, and pneumonia, seniors died at much higher rates from heart disease, pneumonia, and kidney disease. Mortality rates diverged between black and white seniors. The mortality rate for white seniors in 1915 exceeded the 1880 rate by 17

percent while the rate for black seniors increased more than 125 percent during the 35 years. In the 1880s the mortality rate for seniors averaged 252 deaths per 10,000. White seniors had a higher mortality rate than black seniors in 8 of the 10 years of the decade. Mortality rates for black and white seniors correlated strongly from 1880 to 1895.

Chart 18. Senior (45-65 years) Mortality Rate by Race, 1880-1915. In the mid-1890s, the mortality rate for black seniors (blue line) diverged from the rate for white seniors (red line). Data Source: Death Certificate Index

Like other age groups, mortality rates for seniors increased in the late-1890s. After 1894, however, the mortality rate for black seniors increased to a much greater extent than for white seniors due to higher rates of cardiovascular diseases, pneumonia, and kidney diseases. Black rates jumped by over 100 deaths in one year, increasing from 254 deaths per 10,000 in 1894 to 357 deaths per 10,000 in 1895. The 1890s rates peaked at 477 deaths per 10,000 for black seniors and 346 deaths per 10,000 for white seniors during the smallpox epidemic of 1899, but the black rate continued to grow throughout the first two decades of the twentieth century. The black rate climbed
above 400 deaths per 10,000 in 1906 and nearly reached 500 deaths per 10,000 by 1914. White rates, meanwhile, leveled off at the end of the nineteenth century and remained steadily around 300 deaths per 10,000 annually.

C. OTHER AGES

Similar black and white mortality rates in other age groups show that environmental hazards did not affect the other age groups to the same extent as infants and adults. As the next chapter will discuss, environmental hazards associated with different age groups, settlement patterns of families, and acquired immunity explain the similarities in black and white mortality rates for the other age groups. The next chapter argues that infants were more vulnerable to and at risk of diarrheal diseases spread through flooding, and adults lived in higher-density areas that had increased risks for communicable diseases such as tuberculosis. The mortality rates of other age groups remained more closely correlated between the black and white populations than the rates among infants, adults, and seniors. Mortality rates for children (1-5), youths (5-15), and elders (65+) played little role in the overall divergence in black and white rates. Black children and youths died at higher annual rates than the corresponding white rates, but the differentials remain steady. The black and white elderly died at equal rates throughout this period. This section explains the mortality rates in these age groups.
Chart 19. Early Childhood (1-5 years) Mortality Rate by Race, 1880-1915. Data Source: Death Certificate Index
Chart 20. Youth (5-15 years) Mortality Rate by Race, 1880-1915. Data Source: Death Certificate Index

Chart 21. Elderly (65+ years) Mortality Rate by Race, 1880-1915. Data Source: Death Certificate Index
White and black childhood (1-5 years old) mortality rates remained remarkably correlated through much of the last decades of the nineteenth century, yet black rates consistently exceeded white rates, especially in epidemic years. For instance, during the 1883-1884 smallpox epidemic, black children died at an average rate of 525 deaths per 10,000 while white children died at rates of 365 per 10,000. These large differences primarily in epidemic years defined much of the childhood mortality during this time.

Black children died at a rate of 470 deaths per 10,000 compared to white children at 320 deaths per 10,000 in the first half of the 1880s, but after the smallpox epidemic of 1883-1884, the black rate averaged 300 deaths per 10,000 and the white rate averaged 220 deaths per 10,000 until 1901. Chart 19 and Chart 2 show that the spikes in 1891, 1893, 1896, and 1899-1900 coincided with smallpox outbreaks in the city. From 1901 to 1907, black and white rates of childhood mortality remained low, only to increase again during the 1907 smallpox outbreak. After 1908, the childhood mortality rate began to increase in the black population, while the white mortality rate continued to decline gradually. Compared to other age groups, the black and white childhood mortality rates shared the strongest positive correlation over the 35-year period.

The youth (ages 5 to 15 years) mortality rate consistently ranked as the lowest among the age groups. While youth mortality rates for the black population were consistently higher than white mortality rates, the small portion of deaths in this age group limited the effects on overall mortality. Apart from a few exceptional events, such as the 1883-1884 smallpox outbreaks, very few people between the ages of 5 and 15 died in New Orleans (see Chart 20). In 1883 the mortality rate for black youths peaked at 170 deaths per 10,000 and the rate for white youths reached 70

\[ \text{RateWhiteChild} = 0.691009 \times \text{RateBlackChild} + (-0.000683643); r^2 = 0.702203; \text{Standard error: } 0.0040555; \text{p-value} < 0.0001 \]
deaths per 10,000, but these rates represented a small fraction of the childhood mortality rates in these years (5.6 percent and 3.3 percent, respectively).

The mortality rates for white and black elders remained equivalent through the mortality transition. Annual changes in mortality rates did not differ greatly by race. Members of the population who reached 65 years or older had survived many infectious diseases, and black elders had survived larger mortality penalties. As such, black elders tended to die at slightly lower rates than white elders in two-thirds of the years covered (24 of the 36 years).

The percent of deaths in the older age groups increased steadily between 1880 and 1915. Deaths of people age 65 and over increased from 10 percent in 1880 to over 20 percent in 1915. These increases reflect the mortality transition to longer life spans, which meant more deaths occurred in the oldest age group.

In summary, infant mortality rates diverged in the 1890s, with increases in black diarrheal rates and declines in white diarrheal rates. Tetanus rates declined for both groups, although the declines in rates of black tetanus lagged behind declines in white rates. Mortality rates for black and white children and youth increased in smallpox epidemic years—1883 and 1900—while rates increased for children in 1895 and 1907 as well. Mortality rates for black and white children and youth remained remarkably similar until the 1910s. In the 1910s, black mortality rates in both age groups increased while white rates continued to decrease.

Black adults and seniors died in very similar annual patterns to white adults and seniors before 1894. In the late-1890s, however, the mortality rates for black adults and black seniors

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77 Trend Lines Model of 3-year moving average: $r^2 = 0.754985$; Standard error = 0.0036604; p-value: $< 0.0001$.
Equation: Moving Average of RateWhiteElder = 0.642541*Moving Average of RateBlackElder + 0.0370665
diverged from the white rates in these groups. For black adults, these divergences were the proximate result\(^{78}\) of increases in tuberculosis rates and pneumonia rates.

The black and white elderly populations (age 65 and older) of New Orleans proved to be the outliers for mortality rates. At no time did the black elderly mortality rate increase significantly above the white rate. In fact, the white elderly mortality rate was higher than the rate for black elders in many years. Rates peaked for black and white elders in years with smallpox epidemics (1883–1884; 1898–1899) and an influenza epidemic (1893-94).\(^{79}\)

3. Average Age at Death

The uneven mortality transition had large ramifications for residents of New Orleans. White mortality rates declined earlier and to a greater extent than black mortality rates. Rates diverged the most in infant (<1 year), adult (15-45 years), and senior (45-65 years) age groups. Specific diseases influenced the mortality rate divergences. Infantile diarrhea increasingly killed black infants while the rate for white infants declined. Tuberculosis and pneumonia rates for black adults increased while rates for white adults remained unchanged. The results of these differences were a younger average age at death for black people overall and declines rather than increases in the average age at death for black adults in the prime of their lives. While black mortality patterns showed hints of a mortality transition, the black population was more disadvantaged in 1915 than 1880. This was certainly true politically and socially, with the rise of Jim Crow laws and racial segregation in the 1890s and 1900s, but the extent of the health disparities has garnered less attention from scholars.

\(^{78}\) I use “proximate result” to denote that these are not the root causes of disease burdens for black adults.

\(^{79}\) This topic, similarities in black and white elderly mortality rates, is an area for future research.
In 1880 the average age at death was 26 years old in New Orleans, but the average age increased to 41 years by 1915. Yet, scholars have been quick to point out that this statistic obscures more than it clarifies.\textsuperscript{80} Death rates differed by age group, and some groups had a larger effect on the average age. The decline in infant and childhood mortality had an outsized effect on this increase. High rates of infant and early-childhood mortality skewed average ages in 1880, but by 1915 the effect had been reduced. Without infant deaths (under 1 year) in the calculation, the age of death in 1880 averaged 36. Moreover, removing early childhood deaths (1-5 years) reveals an average age of death almost 20 years older for people who survived their first five years of life. By removing the deaths under 5 years old, the improvement in average age of death across the mortality transition becomes much less pronounced — 44 years in 1880 and 49 years in 1915.\textsuperscript{81}

\textbf{Chart 22. White Average Age at Death, 1880-1915. Data Source: Death Certificate Index Dataset}


\textsuperscript{81} Future research will include life expectancy from a life table analysis.
The effect of infant and early childhood deaths on the average age of death decreased over time. Infant and childhood deaths each weighed down the average age of death by almost 10 years in 1880, but by 1915 infant mortality weighed down the average by 6 years while childhood mortality was responsible for only 2.5 years. This highlights the substantial decline in childhood mortality over this time period. These improvements in early-life mortality rates, however, were not evenly distributed.

Differential mortality rates resulted in a large loss of lives and life-years for the black population. The black population of New Orleans lost an increasingly higher number of life-years compared to the white population. The timing of the divergence is important for narrowing the potential social, economic, and environmental causes.

Black and white residents of New Orleans could expect to die at similar ages in 1880. White average age at death exceeded black average age at death by about 5 to 7 percent in the different age groups. For all ages, black average age at death was 25 years while white average
age at death was 27 years — an 8 percent difference. Excluding infant deaths, black people died at age 34 and white people died at 36 — a 6 percent difference. Without deaths under 5, black people averaged 43 years at death, and white people averaged 45 years — a 5 percent difference.

Black and white residents of New Orleans died at similar average ages until the 1890s, when improvements in the average age at death for black residents lagged behind those for white residents. From 1890 to 1900, the average age at which white residents died improved from 32 to 36 years old. Over the same period, the average age at death for black residents improved by just one year, increasing from 30 to 31 years old. The average age at death for older age groups remained steady for white people while the average age at death for black people declined.

By 1915, the black and white average ages at death had become increasingly different. White people in New Orleans died at an average age 26 percent-or almost a decade-older than black people. Black average age at death for all ages was 35 years while white was 44 years. Excluding infant deaths, white people died at an average age of 50 years while black people averaged 41 years—a 22 percent difference. For people who lived past the age of 5, white people averaged 52 years and black people averaged 43—a 21 percent difference.
Overall, white average age at death improved while black average age at death stagnated. For some age groups of the black population, average age at death declined over the course of the mortality transition. For example, black people over the age of 15 died on average at age 46 years in 1880, but by 1915 this average age decreased to 44 years. The corresponding white population averaged 48 years in 1880 and 54 years in 1915. The divergence of average age at death—a change from 4 percent to 23 percent difference—reflects the increasing disease burdens for black residents of New Orleans, which the next chapter will show were connected with environmental conditions and segregation.

The large disparities in mortality rates by race revealed separate and unequal health experiences in New Orleans. The mortality transition unfolded unevenly, benefitting white residents and penalizing black residents. The factors that precipitated the mortality transition — economic, environmental, social, political, or technological — discriminated by race. Yet, factors
alone do not discriminate. The people driving these forces created life for white residents and death for black residents. This chapter has framed the problem and reconstructed the divergences in health at the foundational level, through timing, seasonality, age, and cause of death across the mortality transition. In the next chapter, we shall see that flooding hazards and population density were key proximate factors in the mortality rate divergences for black and white infants and adults.

\(^{82}\) Again, these should not be confused with the root causes of the black and white mortality divergences. I argue in Chapters Three and Four that segregation and white displacement of black residents triggered the mortality divergences.
Chart 32. Annual Mortality Rate (per 10,000) by Disease, New Orleans, 1880–1920.
Chart 33. Distribution of Mortality by Age and Race, New Orleans, 1880–1915.
Chart 34. Average Age at Death for Black Residents of New Orleans, 1880–1915. The line shows the average age at death for all black residents. The blue gradient represents the number of black residents who died at each age throughout this period. Darker areas indicate more deaths in the corresponding age group (y-axis).

Chart 35. Average Age at Death for White Residents of New Orleans, 1880–1915. The line shows the average age at death for all white residents. The red gradient represents the number of white residents who died at each age throughout this period. Darker areas indicate more deaths in the corresponding age group (y-axis).
Chart 36. Monthly Infant Mortality Rate for Diarrheal Diseases by Race, 1880–1890.

Chart 37. Monthly Infant Mortality Rate for Diarrheal Diseases by Race, 1894–1905.

Chart 38. Monthly Infant Mortality Rate for Diarrheal Diseases by Race, 1910–1915.
REFERENCES

DATASETS

CONSTRUCTED IN THIS STUDY

"Geospatial Mortality Dataset"

Source: Louisiana/New Orleans Board of Health Death Certificates
Fields: Date of Death, Surname, Given name, Age, Sex, Marital Status, Race (listed as “color” in original records), Nativity, Locality, Father Place of Birth, Mother Place of Birth, Years in City, Residential Address, Occupation, Cause of Death, Secondary Cause of Death.
Time Range: 1880, 1885, 1890, 1894, 1900, 1905, 1910, 1915
Spatial Unit: Residential Address
Total records: 42,750 (39,133 geocoded)
Notes: Geocoded by address; 1894 used in place of 1895 due to city-wide change in address system beginning in 1895

"Property Tax Value Dataset"

Source: Louisiana/Orleans Parish Property Tax Rolls
Fields: Year, Municipal District, Ward, Assessment District, Block, Property Tax Value (toteda by block)
Time Range: 1880, 1885, 1890, 1895, 1900, 1905, 1910, 1915
Spatial Unit: City Block
Total records: 45,048 (44,405 block values)
Notes: Tabular join to Geospatial Block Dataset

"Geospatial City Directory Dataset"

Source: Soards’ City Directories
Fields: Year, Name, Address, Municipal District
Time Range: 1879, 1886, 1890, 1894, 1900, 1905, 1910, 1915
Spatial Unit: Residential Address
Total records: 627,915 (588,032 geocoded)
Notes: Geocoded by address and municipal district (zonal field); 1879 and 1886 used in place of 1880 and 1885 due to poor quality microfilm in the latter years.
"Geospatial Block Dataset"
Source: Robinson Fire Insurance Atlas 1883
Fields: Municipal District, Ward, Block
Time Range: 1880s-1890s and 1900s-1910s (two periods)
Spatial Unit: Block
Total records: 5,631
Notes: I georeferenced Robinson maps and heads-up digitized block footprints.

"Geospatial Historical Topography Dataset"
Source: Contour Map of New Orleans 1895 (created as part of the Report on the Drainage of the City of New Orleans)
Fields: Elevation
Time Range: 1895
Spatial Unit: 1-foot contours
Notes: Georeferenced map and heads-up digitized the contour lines. After which, I used the Topo-to-raster geoprocessing tool to create a digital elevation model (DEM).

"Geospatial Church Dataset"
Source: Soards’ City Directories
Fields: Year, Church name, Denomination, Race (listed as “color” in original records), Address
Time Range: 1879, 1886, 1890, 1894, 1900, 1905, 1910, 1915
Spatial Unit: Church address
Total records: 1,597 (479 unique churches)
Notes: Geocoded by address and merged across years by name and location

"Board of Health Annual Statistics Dataset"
Source: Annual Report of the Louisiana Board of Health and population data from the US Census
Fields: Deaths by Age and Race, Deaths by Cause of Death, Population by Age and Race
Time Range: 1880–1920 (annual)
Spatial Unit: City
Total records: 40 years, aggregated
Notes: Interpolated population by age and race for years between the US census; imputed mortality rates by age and race; imputed mortality rates by cause of death.
"Charity Hospital Admissions Dataset"

Source: Annual Report of Charity Hospital
Fields: Admissions by Race, Deaths by Race, Births by Race, Admissions by Nativity and State of Birth, Admissions by Occupation
Time Range: 1880–1919 (annual)
Spatial Unit: City
Total records: 40 years, aggregated
Notes: Imputed percent of total deaths and birth in Charity Hospital for each category; imputed mortality rates by age and race; imputed mortality rates by cause of death; imputed distribution of occupations; imputed distribution of nativities and places of birth.

CLEANED AND PROCESSED IN THIS STUDY

"Death Certificate Index Dataset"

Source: Board of Health Death Certificates indexed by the Works Progress Administration in the 1930s, transcribed by the New Orleans Volunteer Association (NOVA), and stored on USGenWeb
Fields: Date of Death, Surname, Given name, Age, Race, Sex
Time Range: 1877–1915 (all records)
Spatial Unit: City
Total records: 231,135
Notes: I standardized the date field, removed duplicates, and corrected errors.

"Historical Weather Dataset"

Source: U.S. Historical Climatology Network, Audubon Station (ID 166664)
Fields: Year, Month, Precipitation (total), Temperature (mean), Temperature (mean maximum), Temperature (mean minimum)
Time Range: 1890–1994 (monthly)
Spatial Unit: City
Notes: I joined the historical weather data to each death record (by month and year). Additionally, I aggregated deaths by age, race, disease, elevation, and month and joined these data to the weather data.
"Birth Dataset"

Source: Contained in the Louisiana State Archives, compiled by the NOVA and USGenWeb.
Fields: Date of Birth, Baby’s name, Father’s name, Mother’s name, Family name, Sex, Race (listed as “color” in original records), Volume, Page
Time Range: ~1840–1904 (all records)
Spatial Unit: City
Total records: 136,719
Notes: I standardized the date field, removed duplicates, and corrected errors.

RECEIVED AND INTEGRATED INTO THIS STUDY

"1880 Microform Census Dataset"

Source: Tenth US Census via IPUMS via the UTHGIS Project
Fields: Age, Race, Occupation, Residential Address (for the other fields included, see the Urban Transition Historical GIS Project)
Time Range: 1880
Spatial Unit: Residential Address
Total records: 216,359 (212,704 include addresses)
Notes: I joined the dataset to the Geospatial Block Dataset. Additionally, I used IDW to calculate population density estimates across the city.

ARCHIVAL REPOSITORIES

Louisiana State Archives
New Orleans Public Library's Louisiana Division & City Archives
Family Search
Ancestry.com
IPUMS
The Historic New Orleans Collection
National Historical GIS
Urban Transition Historical GIS Project
Fondren Library
Historical Weather Network (US Department of Energy, Office of Science)
USGenWeb
NewsBank America’s Historical Newspapers
Proquest Sanborn Map Collection
State Library of Louisiana Digital Repository
Louisiana Digital Library
Internet Archive
HathiTrust Digital Library
US Census

PERIODICALS

*Collier’s Weekly*
*The Century Illustrated Monthly Magazine*
*The Daily Picayune*
*The Harlequin*

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