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Publication Date
2022-05-01

DOI
10.1257/pandp.20221068

Peer reviewed

AEA Papers and Proceedings 2022, 112: 199–204
https://doi.org/10.1257/pandp.20221068

1918 Every Year: Racial Inequality in Infectious Mortality, 1906–1942[†]

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In the first half of the twentieth century, racial inequality in the rate of death from infectious disease was immense. In every year from 1906 to 1920, Black Americans in cities died from infectious diseases at a rate higher than that of urban White Americans during the 1918 influenza pandemic (Feigenbaum, Muller, and Wrigley-Field 2019). Put differently, pandemic-level infectious mortality was a regular experience for urban Black Americans in the early twentieth century. Even by the 1940s, urban Black Americans' risk of death from infectious disease remained above levels urban White Americans experienced in the 1920s.

In this paper, we assess the extent to which this vast inequality in mortality was driven by three broad causes of death: tuberculosis (TB), influenza/pneumonia (flu), and waterborne/foodborne (waterborne) diseases. We decompose infectious mortality into each disease's relative impact on total infectious mortality to determine which causes were most influential. We chose these causes for three reasons: (i) because they are the subject of extensive prior research on mortality in this era; (ii) because their etiology and characteristic demographics differ in important ways; and (iii) because flu and TB were the most common infectious causes of death in this period. Establishing which of these three broad causes contributed most to racial inequality in infectious mortality should help scholars to narrow their search for the drivers of persistent racial inequality in mortality in the early twentieth century.

Our results suggest that racial inequality in infectious mortality was primarily driven by TB and flu—the two major respiratory causes of death. Waterborne causes, by contrast, played a very minor role in explaining the disparity. We also show that racial inequality in TB mortality and flu mortality grew over the period, even as rates of death from these two causes declined among both White and non-White Americans. The starkest increase in the non-White–White infectious mortality ratio appears just after the 1918 influenza pandemic.

These findings are consistent with previous research suggesting that racial inequality in mortality in the early twentieth century stemmed from Black Americans' unique pattern of rural-to-urban migration and residential segregation (Roberts 2009; Zelner, Muller, and Feigenbaum 2017). The fact that respiratory causes of death composed such a large share of the total disparity—and that waterborne causes composed such a small share—suggests that the crowded and poorly ventilated housing that Black Americans were forced into was an important driver of their extreme risk of death from infectious disease. Future research should examine the relationship between segregation and racial inequality in infectious mortality in closer detail.

I. Data

To measure city-level mortality, we digitized city-level deaths by cause from the *Vital Statistics of the United States*. We combine these city-level death counts with population

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[†]Go to https://doi.org/10.1257/pandp.20221068 to visit the article page for additional materials and author disclosure statement(s).

counts generated using the IPUMS Restricted Complete-Count Census Microdata (Ruggles et al. 2018).

Our mortality data have two important limitations. First, states were not required to report vital statistics until 1933 (Haines 2006). Thus, our data cover an unbalanced panel of cities. Second, not all cities reported mortality separately for White and non-White Americans. Our conclusions thus are limited to those cities that did. The cities we study were larger, had larger Black populations, and were more likely to be in the South than other Death Registration Area (DRA) cities. In online Appendix Tables A1 and A2, we report characteristics of the cities in our sample based on when they entered the DRA and whether they reported racial classifications.

We focus on deaths from TB, flu, and waterborne diseases because of the relative importance these causes are accorded in previous literature. For most of the first half of the twentieth century, TB ranked among the top three causes of death among Black Americans in cities (Roberts 2009). A large share of urban Black Americans were recent migrants from rural areas who were pushed into segregated, crowded, and poorly ventilated housing, creating the ideal conditions for TB to spread (Acevedo-Garcia 2000; Ager et al. 2020; Karbeah and Hacker 2021). Flu, another airborne disease, was the other most prevalent cause of infectious mortality during this period (Armstrong, Conn, and Pinner 1999). While recent research has begun to systematically explore racial inequality in flu mortality during the 1918 pandemic (Eiermann et al. 2021), less is known about racial inequality in flu mortality generally.

Waterborne and foodborne diseases offer an instructive contrast to these respiratory diseases. Starting in the nineteenth century, cities undertook major infrastructural projects, such as water and sewer systems, that combatted these diseases (Ferrie and Troesken 2008; Beach et al. 2016; Alsan and Goldin 2019).[1] Werner Troesken has argued that it was hard to exclude Black households from nearby water mains (Troesken 2004).[2] If Troesken is correct about the difficulty of segregating water systems, waterborne causes of death should not have been a major source of the racial disparity in death from infectious disease over the period of our study.

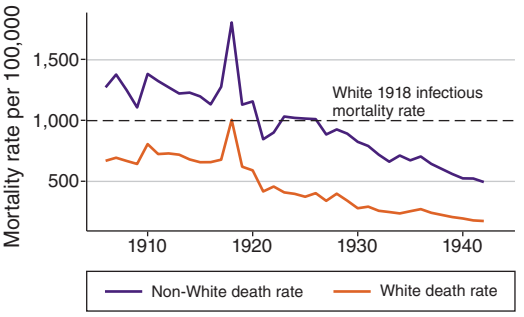

Figure 1. Infectious Mortality, Non-White and White Americans

Notes: The graph shows the weighted median of infectious mortality in US cities. Infectious mortality rates are per 100,000. Online Appendix Figure A1 reports the weighted means instead of the weighted medians.

Source: Mortality data from published volumes of the *Vital Statistics of the United States.*

We classified deaths using the causes reported in the historical *Vital Statistics*. We present the detailed causes of death included in each major cause in online Appendix Table A3. To summarize inequality in mortality, we calculate the median rate of mortality (from infectious causes overall or from specific categories) separately for White and non-White Americans, weighting by city population. This weighting captures the mortality experienced by the median person in the cities in our sample, rather than the median city. As we show in the Appendix, our results are robust to using weighted means instead.

II. Results

Figure 1 shows the stark difference in non-White and White Americans' rate of death from infectious disease from 1906 to 1942. Through 1920, the level of non-White infectious

[1] The extent to which these systems reduced mortality is the subject of ongoing debate (Cutler and Miller 2005; Anderson, Charles, and Rees, forthcoming; Beach 2022).

[2] Anderson et al. (2021) find that water purification efforts reduced the Black–White infant mortality gap. Beach, Parman, and Saavedra (2022) show that more segregated cities were slower to control typhoid despite being faster to introduce waterworks.

mortality was higher than White infectious mortality during the 1918 influenza pandemic. Feigenbaum, Muller, and Wrigley-Field (2019) show that a similar pattern characterized all US regions when examined separately.[3]

For a given cause of death to be a major contributor to racial inequality in infectious mortality, the ratio of non-White-to-White deaths from that cause had to be high, and the cause had to compose a large share of all infectious deaths. We examine these conditions in Figure 2. In panel A, we split the mortality rates from infectious causes into TB, flu, and waterborne mortality, omitting other infectious causes for clarity. Panel A shows that respiratory diseases played a major role in non-White mortality and a less important role in White infectious mortality. For both groups, rates of death for waterborne causes were lowest. In fact, in most years, the non-White mortality rate from waterborne causes was lower than the White mortality rate from flu.

Panel B displays the ratio of non-White-to-White mortality for each major cause of death. Here, deaths from respiratory causes continue to stand out. The racial disparity in deaths from TB rose dramatically after the influenza pandemic, reaching ratios of four to one by the end of the 1920s. The racial disparity in flu mortality also increased after the pandemic, but quickly leveled off.[4] In line with the results shown in panel A, the racial disparity in waterborne mortality was comparatively low throughout the entire period.

The post-1918 jump in racial disparity in TB mortality coincided with a drop in TB deaths among both White and non-White Americans (panel A), possibly because people who would have died of TB were instead killed in the pandemic (Noymer 2011). Zelner, Muller, and Feigenbaum (2017) show that the estimated per-pulmonary-case risk of TB infection fell for White Americans but rose for Black Americans after the pandemic. This slowed the relative decline in the non-White TB death rate, widening the disparity in TB mortality.

When we counterfactually hold non-White mortality from each major cause at the corresponding White mortality rate, this lowers but does not eliminate the total racial disparity in infectious mortality. We plot the adjusted rates and ratios in Figure 3.

Three features of panel A stand out. First, panel A underscores the very minor contribution that waterborne causes made to the high non-White infectious mortality rate. When the waterborne mortality rate for non-White Americans is set to White levels, there is almost no visible downward movement in the non-White infectious mortality rate. Second, airborne causes of death play a much more significant role in the non-White-to-White disparity. Prior to the 1918 influenza pandemic, adjusting TB mortality has the clearest impact on non-White mortality. However, post-1918, adjusting non-White mortality by either flu or TB mortality has similar effects on the non-White mortality rate. Through all of these cause-by-cause adjustments, we still observe a substantial gap between non-White and White mortality. Even when we adjust for the largest killer, TB, the inequality in infectious mortality remains very large.

The ratios of adjusted non-White-to-White mortality shown in panel B yield similar conclusions. Panel B shows the same counterfactual adjustment as in panel A, but in ratios of non-White-to-White mortality. One aspect that panel B demonstrates more clearly than panel A is the apparent increase in the racial disparity in infectious mortality post-1918. The racial disparity increases even as infectious mortality declines in absolute terms. Adjusting non-White airborne mortality to White levels via either of the two major airborne causes (separately) does reduce the total disparity in infectious mortality, but even with this adjustment, non-White mortality is still 1.5–2 times that of White mortality. Adjusting non-White mortality from waterborne causes to White levels has

[3] The high concentration of the Black population in the South makes it challenging to disentangle racial inequality from regional inequality. However, we find that the pattern of our results holds within regions, suggesting that non-White–White disparities are not simply artifacts of regional differences in racial demographics.

[4] TB may have played a larger role than flu in Black households due to its different transmission pattern: TB generally takes longer and requires closer contact to contract than flu. Due to the within-family clustering of TB, it was initially thought to be a hereditary disease (Ott 1996). The discrepancy between racial disparities in TB and flu deaths may reflect intense racial segregation in housing combined with less intimate Black–White contact outside of the home. Alternatively, it could reflect the differential age distributions of TB and flu deaths, as TB deaths were concentrated at young adult ages while flu deaths occurred primarily at very young and very old ages.

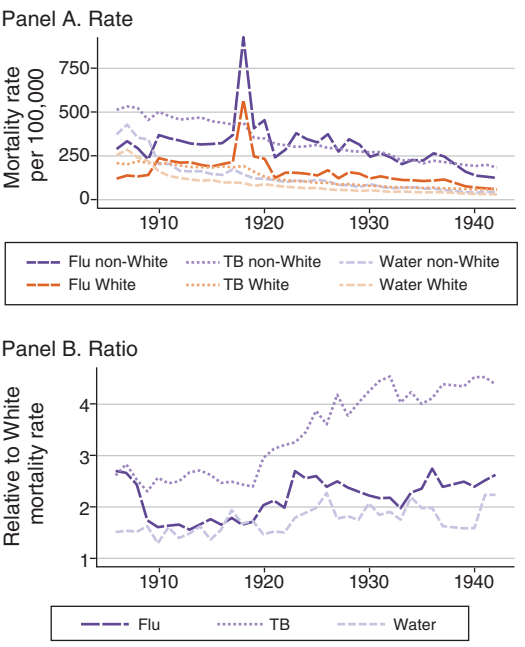

Figure 2. Racial Disparity by Cause

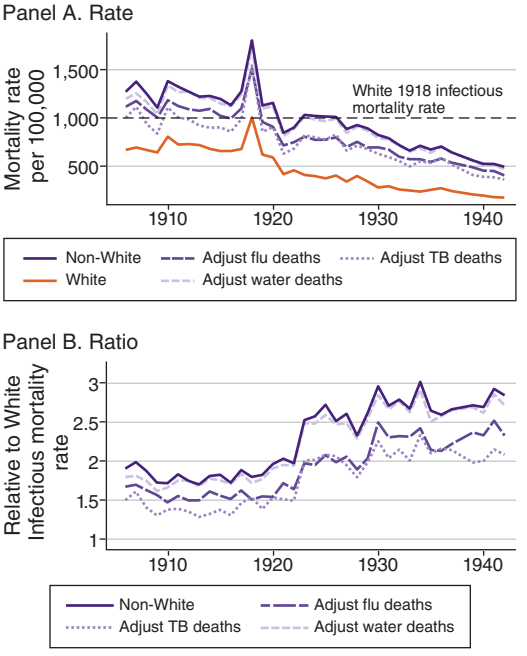

Figure 3. Counterfactual non-White Mortality

Notes: Weighted medians of infectious mortality in US cities. In each version of panel A, the infectious mortality rates are per 100,000. In each version of panel B, the ratio of non-White-to-White mortality is shown. Figure 2 shows the crude rates. Figure 3 considers the counterfactual impact of adjusting each noted cause of non-White mortality to the levels of White mortality. Means instead of medians reported in online Appendix Figures A2 and A3.

Source: Mortality data from published volumes of the *Vital Statistics of the United States.*

almost no impact on non-White mortality past 1910.[5]

Finally, Table 1 reemphasizes the importance of flu and TB for explaining the racial disparity in mortality. Table 1 reports the average and maximum percentage reduction in the racial disparity for each major cause of mortality over 1906–1942. Counterfactually adjusting non-White TB mortality to White levels has the clearest impact on the racial disparity—a reduction of 43 percent. Adjusting flu to White levels reduces the disparity by 30 percent on average. None of the other listed causes affect the racial disparity by more than 6 percent on average. Counterfactually adjusting waterborne causes, syphilis, whooping cough, bronchitis, and malaria all lower the racial disparity by 1–6 percent.

III. Conclusion

This article decomposes racial inequality in infectious mortality into three broad causes of death, allowing us to trace possible explanations for why Black Americans were so much more likely than White Americans to die of

[5] This last result needs to be qualified because of an important change in the cause of death coding implemented in 1910. Beginning in that year, diarrheal deaths above age 2 were no longer recorded separately, but were collapsed into "all other causes," and thus are not included as waterborne (or other infectious) deaths here. Our results therefore show that diarrheal deaths below age 2, as well as specific waterborne causes such as typhoid and (late in the series) polio, were not important drivers of non-White–White mortality inequality. However, diarrheal deaths beyond early childhood may have continued to affect racial inequality in infectious mortality. As Table 1 shows, the maximum reduction in the disparity associated with waterborne causes was 17.8 percent; this occurred before 1910, with all diarrheal deaths included.

Table 1—Reduction in Racial Disparity by Cause

Adjusted cause	Reduction in disparity (%)	
	Average	Max
Tuberculosis	43.2	60.1
Influenza/pneumonia	30.1	43.8
Waterborne/foodborne	5.8	17.8
Syphilis	3.5	15.5
Whooping cough	2.1	7.6
Bronchitis	1.3	8.7
Malaria	1.0	8.4
Puerperal septicemia	0.9	3.3
Measles	0.2	3.2
Scarlet fever	−0.4	0.7
Diphtheria	−0.7	3.7

Source: Mortality data from published volumes of the *Vital Statistics of the United States.*

infectious diseases in the first half of the twentieth century. We show that racial inequality in infectious mortality was primarily driven by respiratory causes of death, especially TB and flu. Waterborne mortality, in contrast, plays almost no direct role.[6]

These findings point to the importance of the built environment as a source of health disparities. Relative to housing, by the beginning of the twentieth century, there was less variation in access to high-quality water and sewer systems. Many of these systems were built in the late nineteenth century, before our period of study (Beach 2022). The lesser degree of segregation in water and sewer systems thus could be a major reason for the relatively low degree of racial inequality in death from waterborne diseases.

Residential segregation, in contrast, was stark in cities across the country. Black rural-to-urban migrants were often forced into the most crowded and worst-ventilated housing—conditions conducive to the spread of airborne infections like TB and flu (Acevedo-Garcia 2000; Ager et al. 2020; Karbeah and Hacker 2021). The large disparities in mortality from TB and flu relative to waterborne causes provides further suggestive evidence that residential segregation was an important cause of Black Americans' pandemic-level rate of death from infectious disease in the early twentieth century. Future research should use more granular data, like death records, to track how unequal access to safe housing contributed to racial inequality in death from infectious disease.

[6]The Mills-Reincke Phenomenon—or the multiplier linked to the elimination of typhoid through waterworks (Beach 2022)—could imply that typhoid reductions had larger consequences for the total racial disparity than suggested here if they resulted in reduced airborne deaths. However, during the period of our study, most cities already had waterworks in place. The widespread adoption of city-level waterworks potentially explains the smaller observed disparity in waterborne causes of death in the early twentieth century. If we extended our series into the nineteenth century, we expect our findings would likely show a larger disparity in waterborne causes of death (Beach 2022; Anderson et al. 2022).

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