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Racial Disparities in Mortality During the 1918 Influenza Pandemic in United States Cities

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ABSTRACT Against a backdrop of extreme racial health inequality, the 1918 influenza pandemic resulted in a striking reduction of non-White to White influenza and pneumonia mortality disparities in United States cities. We provide the most complete account to date of these reduced racial disparities, showing that they were unexpectedly uniform across cities. Linking data from multiple sources, we then examine potential explanations for this finding, including city-level sociodemographic factors such as segregation, implementation of nonpharmaceutical interventions, racial differences in exposure to the milder spring 1918 "herald wave," and racial differences in early-life influenza exposures, resulting in differential immunological vulnerability to the 1918 flu. While we find little evidence for the first three explanations, we offer suggestive evidence that racial variation in childhood exposure to the 1889–1892 influenza pandemic may have shrunk racial disparities in 1918. We also highlight the possibility that differential behavioral responses to the herald wave may have protected non-White urban populations. By providing a comprehensive description and examination of racial inequality in mortality during the 1918 pandemic, we offer a framework for understanding disparities in infectious disease mortality that considers interactions between the natural histories of particular microbial agents and the social histories of those they infect.

KEYWORDS 1918 influenza pandemic • Racial disparities • Immunological imprinting • Nonpharmaceutical interventions • Residential segregation

Introduction

The 1918 so-called "Spanish flu" pandemic was distinctive for at least three reasons, two of which are well-known. The pandemic was immensely destructive, causing 50–100 million deaths globally and perhaps 675,000 in the United States (Ewing 2021; Spreeuwenberg et al. 2018). The virus also killed in a unique W-shaped age pattern: alongside the more universally vulnerable very young and old, young adults in their late 20s and early 30s experienced relatively high mortality rates. Within the United States, a third distinctive fact about the 1918 pandemic is less well-known. As

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we show in this study, the non-White/White racial disparity in influenza and pneumonia mortality was around three fourths smaller than in prior years.

This narrowing of inequality is surprising for several reasons. It is the opposite of mortality trends observed during the COVID-19 pandemic and the HIV/AIDS pandemic (Hutchins et al. 2009), during which oppressed groups, including poor people and communities of color, have experienced comparatively high mortality owing to increased risks of disease incidence and severity.[1] The narrowing also diverges from health disparity trends throughout the early twentieth century, when non-White mortality far exceeded White mortality in every nonpandemic year (Boustan and Margo 2016; Feigenbaum et al. 2019; Jackman and Shauman 2019; Wrigley-Field 2020). And, although the available medical care at the time was quite limited, the pattern is nevertheless notable because the U.S. healthcare system was rife with racism; during the pandemic, both northern and southern cities opened Whites-only clinics (Bristow 1992:72; Byrd and Clayton 2001).

Despite being so distinctive, these reduced mortality disparities have received relatively little scholarly attention. Researchers frequently relied on anecdotal evidence from newspapers and public health reports but did not systematically measure race-specific pandemic mortalities (Gamble 2010; Schlabach 2019). Earlier studies from the 1920s and 1930s used military statistics and local surveys to examine mortality patterns for specific subgroups of the American population. In a comprehensive review of such studies, Økland and Mamelund (2019) found some evidence of a racial mortality crossover when the pandemic was most severe in the United States, during the fall of 1918: mortality rates may have been lower for Black people than for White people during those months, although it is not clear whether these findings are generalizable to the full U.S. population.

The drivers of these patterns also remain unclear. Scholars have hypothesized that Black communities may have been disproportionately exposed to a less deadly "herald wave" in the spring of 1918 (Crosby 2003; Økland and Mamelund 2019), providing partial immunity against the deadlier fall wave. Segregation may also have functioned as an accidental *cordon sanitaire* that insulated non-White communities against infection (Schlabach 2019). Still, "shockingly sparse" data (Krishnan et al. 2020:474) have prevented the verification of such hypotheses (Gamble 2010).

In this study, we provide the first comprehensive description of racial disparities during the pandemic in U.S. cities. We also examine *why* these racial disparities were strongly reduced. Integrating theories from demography, sociology, public health, and virology and immunology, and linking data from varied sources, we posit and assess four possible explanations. Two are linked to city-level characteristics—sociodemographic features and nonpharmaceutical interventions (NPIs), such as school closings—while the other two consider the life course histories of infectious disease exposure ("social immunology"; Shattuck 2021) among urban White and non-White populations of this era. The first of these social immunology explanations, proposed previously (Crosby 2003; Økland and Mamelund 2019), suggests that mortality inequalities in 1918 may have been reduced because *non-White* populations

[1] The ratio of age-adjusted non-Hispanic Black to non-Hispanic White total mortality in 2019 was 1.18; in 2020, it was 1.34 (authors' calculations from Ahmad et al. 2021 and Centers for Disease Control and Prevention 2021).

were more likely to have had a prior influenza exposure (to a less severe herald wave of 1918's new H1N1 viral strain) that was protective. In contrast, the second suggests that disparities in 1918 may have been reduced because *White* populations were more likely to have had a prior influenza exposure (to an earlier pandemic peaking in 1890) that resulted in "immunological imprinting" that proved harmful when the H1N1 pandemic influenza emerged.

We examine explanations related to city-level characteristics and social immunology using a combination of empirical tests and simulations. Our empirical tests combine data from digitized mortality records for 70 U.S. cities; linked census records that establish urban residency across multiple decades; and a novel, comprehensive data set on the timing of NPIs—such as school shutdowns, mandatory closures of theaters and bars, and bans on public gatherings—collected from historical newspaper accounts. Simulations allow us to ascertain the plausibility of speculative explanations for which no direct measurements are available. To test the immunological imprinting explanation, we employ a combination of five suggestive (but not definitive) empirical and simulation tests.

Consistent with prior work using different data (Gamble 2010; Krishnan et al. 2020; Økland and Mamelund 2019; Schlabach 2019), we find that racial disparities in pandemic mortality were almost uniformly reduced compared with prepandemic years. Turning to potential explanations for these reductions, we find that city-level characteristics, such as segregation and NPIs, fail to explain them. We also find little evidence that partial immunity due to herald wave exposure accounts for reduced racial disparities in pandemic mortality, although we speculate—based on qualitative accounts—that the 1918 herald wave may have induced relevant behavioral responses that protected Black communities during the pandemic's peak months. This explanation merits further exploration. The explanation we are unable to refute is the one based on immunological imprinting. We argue that the 1918 pandemic's reduced racial disparities may reflect a historical contingency, built on the interaction of the distinctive migration and public health experiences of non-White and White urban populations with the historical sequence of influenza strains that successively emerged between the late nineteenth century and 1918. This perspective on infectious disease mortality explicitly draws attention to the historically contingent interplay between the macrosocial histories of differently racialized groups and the microbiological characteristics of particular epidemics.

Potential Explanations for the Racial Gap in Influenza and Pneumonia Mortality in 1918

The uniquely narrow racial mortality gap was recognized in 1918. Chicago health officials noted that race-specific incidence counts were "quite contrary to what would have been expected" (Schlabach 2019:41), while a prominent Black reverend proclaimed that God had been trying "to show [the White man] the folly of the empty conceit of his vaunted race superiority, by dealing with him just as he dealt with the peoples of darker hue" (Bristow 1992:73). Mortality tables constructed in the wake of the pandemic also reveal unexpectedly high White mortality and reduced Black/White disparities in influenza mortality (Frankel and Dublin 1919).

Two potential explanations have been proposed to account for these reduced mortality disparities, although neither has been tested systematically. The first focuses on local sociodemographic characteristics, which often affect infectious disease mortality during public health emergencies (Bonds et al. 2010; Hart et al. 1998; Valleron et al. 2010; Waitzman and Smith 1998). Mortality in 1918 correlated with local illiteracy rates in Chicago (Grantz et al. 2016) and may have been elevated in communities with intense residential crowding (Mamelund 2018; Økland and Mamelund 2019). Residential segregation may also have offered temporary protection to non-White communities during the 1918 pandemic if it distanced these groups, physically and socially, from centers of community transmission (Krishnan et al. 2020; Schlabach 2019).

The second existing explanation focuses on the short-term immunological history of different racial groups. Although the overwhelming majority of 1918 influenza deaths occurred in the United States between September and December, a less lethal flu strain began circulating—and raising mortality rates beyond normal seasonal fluctuations—between January and March (Hoffman 2011; Olson et al. 2005; Patterson and Pyle 1991). Recent evidence suggests that this influenza strain subsequently evolved to evade immune responses, accounting for its radically increased lethality by September (Patrono et al. 2022). Crosby (2003:229) speculated that exposure to the antigenically similar spring herald wave may have primed the immune system for a more efficient response during the fall. Non-White communities may have disproportionately benefited from such partial immunity because they were more likely than White communities to experience residential conditions that are conducive to the spread of infectious diseases, such as overcrowding and lack of access to handwashing opportunities (Mamelund 2018; Økland and Mamelund 2019). In this scenario, partial immunity induced by the herald wave might explain the comparatively small mortality disparities during the fall because it made non-White individuals disproportionately resistant.

In addition to these existing hypotheses, we propose and evaluate two additional potential explanations based on recent work in historical and social immunology. First, we extend the literature on NPIs. During the peak months of the 1918 pandemic, many U.S. cities sought to contain the spread of infections by closing schools, theaters, and bars; banning gatherings in public places; and quarantining infected persons. Cities that implemented these NPIs early and sustained them until the end of the pandemic tended to experience lower mortality than cities that delayed NPIs or lifted them prematurely (Hatchett et al. 2007; Markel et al. 2007).[2] Because non-White populations had a much higher baseline risk of infectious disease mortality,

[2] One reason that NPIs were not sustained longer is that they were often highly controversial. As one Topeka, Kansas, newspaper article put it in declaring the pandemic defeated in Topeka (after two flu-free days in April 1919): "Both [health commissioner] Porter and [health officer] Clark have been the target, during the last few months, for gobs of criticism and insubordination against what was termed the autocracy they were establishing in depriving the citizens of the privilege of freedom of action, the right to mingle with 'flu' germs" ("Topeka free of flu" 1919). There was, however, context beyond the disruption associated with NPIs; Clark had been mocked by the same paper three weeks earlier for a snobbish declaration: "'How can you expect the common people to heed influenza warnings,' snorted Doctor Clark, 'when our socially best people sometimes show just as little sense about such things?'" ("Affair riles Dr. H. L. Clark" 1919).

they may have disproportionately benefited from NPIs. This hypothesis is analogous to Troesken's (2004) argument that water and sewage improvements in southern cities disproportionately benefited Black individuals because of their higher risk of waterborne disease. In this case, evidence supporting this explanation would show that cities with early NPI onset and long NPI duration experienced reduced non-White/White mortality ratios in addition to reduced total mortality.

We additionally propose and evaluate the novel explanation that immunological imprinting from a late nineteenth-century pandemic may account for reduced racial disparities in 1918. Pandemics are transformative events that reshape populations with long-lasting epidemiological consequences (Noymer 2010). We posit that the distinctive social and geographic histories of 1918's urban populations—specifically, the disproportionately rural origins of the urban non-White population—may have produced racial differences in early-life flu exposures that translated to increased immunological vulnerability, primarily for *White* young adults, during the 1918 pandemic. This explanation builds on historical immunology research arguing that early childhood exposure to a pandemic that reached U.S. port cities in December 1889 may account for the unusually high mortality of young adults in 1918 (Gagnon et al. 2013, 2015; Hallman and Gagnon 2014; Luk et al. 2001; Mamelund 2011; Shanks and Brundage 2012; Woo 2019; Worobey et al. 2019; Worobey et al. 2014). (We refer to this previous pandemic as the "1890 pandemic," reflecting its deadliest year; it has also been widely referred to as the "Russian flu.") Extending the imprinting hypothesis to consider racialized life course exposures offers the enticing possibility of a consilience (Whewell 1858) between two of the most striking features of the 1918 pandemic: its unusual age shape and its reduced racial disparities.

This explanation turns on current hypotheses about evolving influenza A strains over the decades leading up to 1918. The 1918 flu was an H1N1 influenza; the leading hypothesis is that the 1890 pandemic was caused by an H3N8 influenza (Dowdle 2006). Because H1 and H3 hemagglutinin come from different phylogenic groups, their antibodies offer little cross-protection (Gostic et al. 2019), while first exposure to influenza within the same phylogenic group can offer long-lasting protection (Gostic et al. 2016). Virological work (Worobey et al. 2014) suggests that H3 may have remained in circulation until around 1900, when it was replaced by H1, producing a likely H1N8 outbreak in the early 1900s. The same virologists speculate that H1N8, or another influenza with hemagglutinin similar to that of H1, predominated before the 1890 pandemic. Thus, people born around 1890 would have likely had their earliest exposure to H3N8 influenza—with substantial phylogenic difference from 1918's H1N1—while those born much earlier or later would have likely had early exposure to a homotypic influenza. We also explore a variant of this imprinting hypothesis in which exposure to the 1890 pandemic need not be one's *first* influenza exposure to result in imprinting during early childhood, via a mechanism called "immunological refocusing" (Gagnon et al. 2015); in that case, the key question is who had *early childhood* exposure to the 1890 pandemic, irrespective of earlier exposures.

According to the immunological imprinting explanation of elevated young adult mortality in 1918, immune systems "imprinted" with the 1890 virus often had a dysregulated response to the 1918 virus: an abundance of antibodies that could not neutralize that virus crowded out a more effective immune response and produced deadly tissue damage that left people susceptible to secondary bacterial infections.

By hypothesizing that such imprinting was more prevalent among 1918's urban White young adults than urban non-White young adults, we extend this possibility to potentially account for reduced racial disparities.

Notably, the immunological imprinting explanation explored here presumes that the 1890 pandemic was indeed caused by an H3N8 influenza. There are two clear alternatives: an H2N2 influenza (Mulder et al. 1958) and a coronavirus (Brüssow and Brüssow 2021; Vijgen et al. 2005; Vijgen et al. 2006). Since H2 is in the same phylogenic group as H1, it is not clear that immunological imprinting with H2N2 would have been as deeply deleterious in 1918 as this explanation requires; and should the 1890 pandemic turn out to stem from a coronavirus, the imprinting mechanisms just outlined would be moot. In either of these cases, childhood exposure to the 1890 pandemic presumably could account for vulnerability in 1918 only if it produced grievous bodily harm resulting in subsequent frailty (the "long COVID" of the nineteenth century, perhaps).

Altogether, this leaves us with four potential explanations—two rooted in city-specific characteristics (*Explanation A:* segregation/illiteracy/density and *Explanation B:* NPIs) and two rooted in social immunology (*Explanation C:* protective immunity due to herald wave exposure and *Explanation D:* reduced immunity due to childhood imprinting). While the two potential explanations rooted in city-specific characteristics focus on local conditions *during* the 1918 pandemic, the explanations rooted in social immunology build on the distinctive social histories of exposure of each racial group *prior to* the pandemic. The urban White population of this era was a combination of recent immigrants from abroad, grown children of earlier immigrants, and some internal migrants, particularly through westward expansion. In contrast, many non-White urban residents had migrated only recently from rural southern areas, where they experienced comparatively low influenza exposure during childhood, to southern and northern cities, where they were forced, by law and by violence, into intensely crowded conditions rife with infectious disease (Ager et al. 2020; Økland and Mamelund 2019:15; Roberts 2009). This combination of relatively low early-life exposure followed by intense exposure after urban migration allows for the mix of potentially protective and harmful exposures that we explore.

Data and Methods

First, we examine racial mortality disparities across the United States by integrating a series of annual race-specific and cause-specific mortality rates for 70 U.S. cities that is based on annual death counts collected by the Department of Commerce (Feigenbaum et al. 2019), supplementary data sets with age-specific and monthly mortality rates for a smaller number of cities ($n=20$), and measures of an array of city characteristics and public health responses to the pandemic (summarized in Table 1). Our mortality measure is based on recorded influenza and pneumonia deaths, since pneumonia was the proximate cause of many pandemic deaths. We report disparities for alternative measures in section IB of the online appendix.

Historical *Vital Statistics* from the Department of Commerce report published death counts. The coverage of these mortality data sets includes cities from states that were included in the Death Registration Area (DRA) in 1918, as well as several

Table 1 Data sets and data sources

Variables	*N* (cities)	Years Covered	Comment	Source
Annual Mortality, Cause- and Race-Specific	70	1900–1930	Limited to states and cities that were included in the Death Registration Area in 1918; pre-1918 data are missing for some cities	U.S. Department of Commerce (digitized by research team)
Annual Mortality, Cause-, Race-, and Age-Specific	20	1900–1930	Limited to states and cities that were included in the Death Registration Area in 1918; pre-1918 data are missing for some cities	U.S. Department of Commerce (digitized by research team)
Monthly Mortality, Cause- and Race-Specific	20	1910–1920	Limited to states and cities that were included in the Death Registration Area in 1918; pre-1918 data are missing for some cities	U.S. Department of Commerce (digitized by research team)
Population Counts, Cause- and Race-Specific	1,205	1900–1930	Log-interpolated values for intercensal years based on 1910 and 1930 census counts	U.S. Department of Commerce/IPUMS
City Characteristics, Including Residential Density, Residential Segregation, Illiteracy Rates, and Share of Foreign-born Residents	Up to 881	1900–1930	Density measures are estimated on the basis of 1% census samples; number of cities in the data set varies between 329 (for residential density estimates) and 881 (for residential segregation indices)	U.S. Department of Commerce/IPUMS
Childhood Urban Residency Rates, Race-Specific	601	1900–1910	Low percentage of linked census records may distort estimated rates	Census Linking Project/IPUMS
NPIs	52	1918–1919	—	Original data collection through newspapers.com
NPIs (alternative measures)	43	1918–1919	Cause- and race-specific mortality data are available for 18 cities in this data set	Markel et al. (2007)
Spring Wave Influenza Mortality (alternative measure)	45	1918	—	Crosby (2003)

Table 1 (continued)

Variables	*N* (cities)	Years Covered	Comment	Source
Infant Mortality, City-Specific	43	1910	—	Collins (1930)
Air Pollution and Infant Mortality, City-Specific	878	1915–1925	Missing data for some cities/years; air pollution is calculated from local coal-fired electricity generation capacity	Clay et al. (2019)

Note: NPI = nonpharmaceutical intervention.

additional large cities in states such as Texas (which had not yet joined the DRA but reported death counts for specific cities). While our primary data set with annual race-specific mortality rates includes large- and medium-sized cities across the United States, monthly race- and age-specific mortality estimates are available only for a reduced number of large cities. The smallest city included in our monthly data is Nashville, Tennessee, with an estimated 126,000 residents in 1918. Our findings may not extend to smaller cities or nonurban settings, or to cities that were exclusively White.

To convert these death counts into death rates, we estimate race-specific, age-specific populations for each city. Because the 1920 census likely reflected substantial population distortions resulting from the pandemic, we avoid drawing on that census in constructing our population denominators. Instead, our main measure interpolates populations (on log scale) from 1910 to 1930. (We include alternative mortality calculations that draw on 1920 data in the online appendix.) Since our main measure may be distorted by changing migration patterns, we also construct an alternative denominator by estimating population sizes from annual noninfectious death counts (details are reported in the online appendix). These two mortality measures, which make very different assumptions when estimating 1918 population counts, evince relatively close agreement: the alternative measure estimates the non-White/White ratio of flu and pneumonia mortality at 1.39 in 1918 (vs. 1.35 with our main measure) and 2.41 in 1910–1917 (vs. 2.33).

The *Vital Statistics* report deaths in "White" and "Colored" populations. In this era, non-White urban populations were overwhelmingly Black, and we interpret them as a measure of Black mortality. We measure disparities on a multiplicative scale, using the ratio of non-White to White mortality in descriptive results and estimating regressions in logit scale. Our unit of analysis is cities.

Next, we assess our four possible explanations for the racial mortality gap in 1918—A: city-level segregation, illiteracy, and residential density; B: city-level NPIs; C: partial immunity from the spring 1918 herald wave; and D: immunological imprinting. To explore the first two explanations, we combine mortality data with a wide array of city- and race-specific measures of segregation, residential density, illiteracy, air pollution, age composition, and the onset and duration of NPIs. We collected NPI data from a qualitative survey of historical newspaper records. Specifically, we recorded the number of total days that mandatory quarantine, bans on

public gatherings—which included the closure of theaters, bars, and restaurants—and school closings were in effect. We did not count NPIs implemented only in military camps, since those camps were commonly located beyond city limits and administered by the U.S. military rather than municipal authorities. Following Markel et al. (2007), we calculated total NPI duration by summing across all NPI types. We find a high correlation ($r = .9$) between our original NPI measures and those obtained by Markel et al. (2007) for cities appearing in both data sets. We also measured the number of days between the first locally recorded infection and the start of NPIs, which we treat as a measure of NPI delay. In total, we were able to expand Markel et al.'s (2007) data set on the date and duration of school closings and bans on public gatherings from 43 cities to 52 cities, 34 of which reported race-specific death counts and are included in our analyses.

City-specific calculations of segregation indices and measures of city- and race-specific illiteracy rates and residential density are based on census microdata via IPUMS (Ruggles et al. 2020), while additional city-level variables are taken from several prior studies (Clay et al. 2019; Collins et al. 1930; Crosby 2003; Markel et al. 2007), as summarized in Table 1. We use full-count census data to calculate illiteracy rates and four segregation indices: the dissimilarity index (White 1983), the divergence index (Reardon and O'Sullivan 2004), the variance ratio index (Roberto 2016), and the sequence index of segregation (Grigoryeva and Ruef 2015). We use the 1% census sample to construct indices of residential density based on the average number of persons per dwelling and the percentage of residents living in multifamily homes; we use this sample because the required variables are not reliable in the current iteration of the IPUMS USA full-count data (Ager et al. 2020).

We use these data to construct a series of bivariate and multivariate models that test the predictions of each hypothesis and control for variables such as age composition and NPI implementation, which are known to have affected pandemic mortality. Specifically, we construct multivariate linear models that regress logged mortality ratios on city- and race-specific measurements of illiteracy and residential density and city-specific racial segregation indices, controlling for logged baseline mortality that captures potential unobserved time-invariant confounders (predicted from 1910–1917 trends for each city), local age composition (measured as the percentage of residents aged 20–39), and local NPIs. Likewise, to assess the association between race-specific mortality and NPIs, we construct a series of linear models that regress logged race-specific mortalities and logged mortality ratios on NPI duration, NPI delay, and race-specific baseline mortalities and baseline mortality ratios for each city. Full details on these models (including regression equations) are reported in section II of the online appendix; power calculations for all models are reported in section IIE. Models are generally well-powered, with the exception of the analysis of the herald wave; we discuss this limitation when we report those results.

We test the partial immunity hypothesis, which posits that racial differences in exposure to the milder spring 1918 herald wave contributed to mortality patterns during the deadlier fall wave, by answering two questions: First, were non-White populations more affected by the herald wave than White populations? Second, was greater exposure to the herald wave associated with reduced mortality during the fall wave? These analyses use influenza/pneumonia mortality during January–June 1918 as a proxy for population-level partial immunity during September–December 1918.

Because this proxy measure assumes equal infection fatality across racial groups and cities, it is highly imperfect, made necessary by the almost total lack of historical data on race-specific influenza exposure rates. To our knowledge, such data were collected only during the peak of the 1918 pandemic (rather than the spring) from nonrandom samples in six U.S. cities and several military camps (Britten 1932; Frost 1920; Ireland 1929). The generalizability of these surveys is severely limited, yet they indicate that, during fall 1918, Black populations may have experienced an ~30% higher infection fatality rate compared to White populations, and that race-specific exposure rates may have been as low as 5% and as high as 50%. These data suggest that the bias in our proxy measurement for spring exposure may lead us to overestimate the prevalence of herald wave infections in non-White communities, and thereby also overestimate racial differences in herald wave exposure—the key purported reason for race-specific differences in immunity. We discuss the consequences of this potential bias, and more broadly of potential noise, when we present the herald wave results.

To test the immunological imprinting explanation (for which no direct measurements are available), we use a rough empirical proxy for variation in childhood exposure to the 1890 pandemic: 1918 urban populations' urban origins, estimated from the Census Linking Project (Abramitzky et al. 2021; Abramitzky et al. 2020), working from the assumption that urban children would have had greater influenza exposure than rural children in 1890. We also develop a set of simple simulations that evaluate the effects of 1890 flu exposure on the magnitude of age-specific mortality in 1918, exploiting cohort variation in the likelihood of the 1890 pandemic being individuals' first flu exposure. These simulations allow us to answer two questions. First, how high would mortality need to be among individuals who were "imprinted" with the H3N8 virus during childhood to generate age- and race-specific mortality rates that we observe in 1918? Second, what exposure rates to the 1890 flu (and prior influenza strains) would be required to produce these imprinting rates? We assess the plausibility of the resulting parameter ranges consistent with the imprinting explanation by comparing them to empirical data obtained in other immunological studies. In particular, we focus on assessing the plausibility of the parameter ranges for 1918's urban White young adults—the key population driving reduced aggregate disparities. This two-pronged empirical and simulation-based methodological approach allows us to narrow the space of plausible explanations despite the scarcity of historical health data (particularly from the nineteenth century). The immunological imprinting tests are explained in further detail in the section Explanation D: Immunological Imprinting.

Details of all data, measures, and models are given in the online appendix. Data, codebook, and software code are available at http://doi.org/10.17605/OSF.IO/NJGHD.

Non-White and White Influenza and Pneumonia Mortality in 1918

The 1918 pandemic occurred in the context of extreme, ubiquitous racial inequality in the United States—including inequality in infectious disease mortality. Prior to 1918, non-White residents across U.S. cities (which were overwhelmingly Black) were more than twice as likely to die from influenza/pneumonia as White residents.

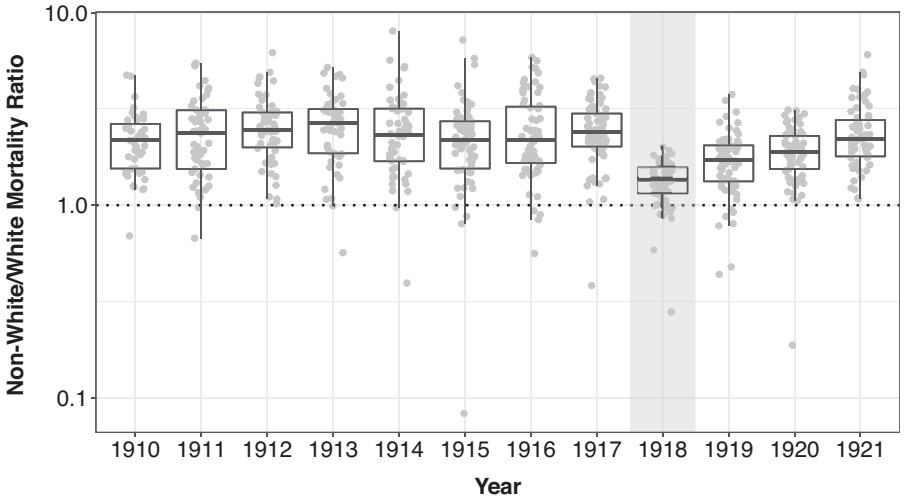

Fig. 1 Non-White/White ratios of influenza and pneumonia mortality by year. Mortality values (unlogged) are portrayed on a logged-scale *y*-axis. The 1918 pandemic (gray shading) was characterized by uniformly reduced racial disparities in mortality relative to in nonpandemic years. The dotted line indicates a mortality ratio of 1, that is, an equal number of White and non-White deaths per 100,000 individuals. The thick horizontal lines indicate city-level medians, with lines above and below indicating 75th and 25th percentiles, respectively. Vertical lines span 1.5 times the interquartile range.

Non-White populations still experienced some of the highest recorded mortality rates during the 1918 pandemic, but the non-White/White inequality in influenza mortality was around three fourths smaller than in previous years; we find the median city's non-White/White mortality ratio was 1.35 in 1918, compared with 2.33 over the pooled years 1910–1917. Equally striking is the small variation in 1918 influenza/pneumonia mortality disparities across cities (Figure 1). The 1910–1917 median standard deviation in non-White/White mortality ratios across 70 cities was 1.11, yet it shrank to 0.32 in 1918. This is unexpected, considering which subpopulations were represented in age-groups most at risk during the pandemic: the very young, the very old, and young adults. White people were overrepresented among the very young and old, but non-White people were overrepresented among young adults, who had fairly high mortality rates.

This sudden decline in the mortality ratio reflects the fact that, on average, non-White flu deaths in 1918 were around three times the prepandemic average—while White flu deaths were five times the prepandemic average. Young adults aged 10–39 experienced the largest proportional mortality increase. Figure 2 shows that the average non-White influenza/pneumonia mortality among those aged 10–39 grew from 156 deaths per 100,000 people in prepandemic years to 732 deaths in 1918, and the average White mortality increased from 42 deaths per 100,000 people to 627 deaths. In the group with the highest excess mortality during the 1918 pandemic—Whites aged 20–29—mortality was almost 20 times as high in 1918 as in the pooled 1910–1917 years.

The narrowing of racial disparities in mortality was largely due to a mortality convergence among those aged 10–19, 20–29, and 30–39 (Figure 2). These three groups

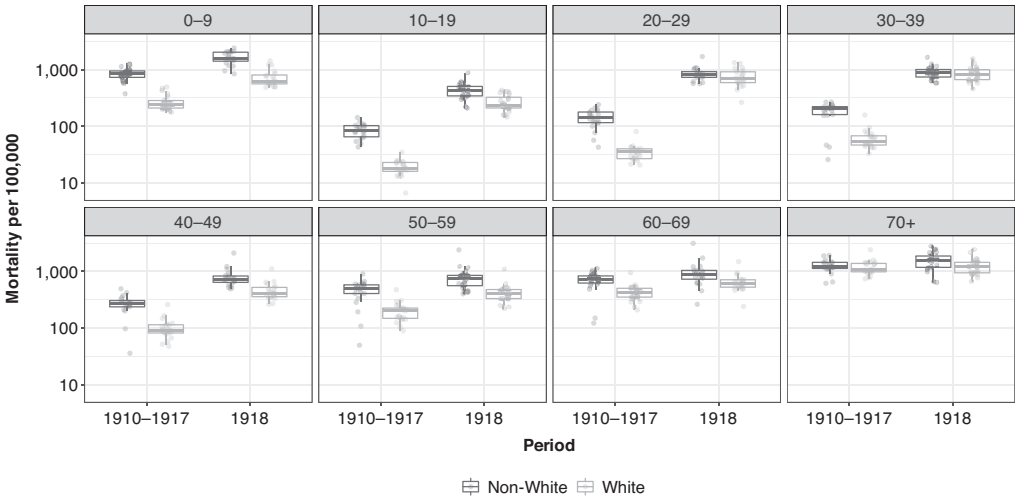

Fig. 2 Prepandemic (1910–1917) and pandemic (1918) influenza and pneumonia mortality per 100,000 individuals by race and age-group, for the 20 cities for which such data are available. Mortality values (unlogged) are portrayed on a logged-scale *y*-axis. Mortality among the very young and very old was almost uniformly high, but those aged 20–39 experienced large increases in mortality *and* the largest declines in non-White/White mortality ratios during the 1918 pandemic. See Figure 1 for description of the box-and-whisker plots.

had the highest prepandemic disparities—with the non-White/White influenza/pneumonia mortality ratios measuring 4.48, 4.15, and 3.39 respectively—but had among the smallest disparities in 1918. Disparities were notably nonexistent in the 20–29 and 30–39 age-groups. Compared with 1910–1917, non-White/White flu and pneumonia mortality disparities in 1918 declined by 78% in the 10–19 age-group, by 95% in the 20–29 age-group, and by 98% in the 30–39 age-group.

In contrast, racial disparities in influenza/pneumonia mortality among the very young and the very old were considerably less affected by the pandemic. At ages 70+, non-White mortality was (and remained) roughly comparable to White mortality. This low inequality likely reflected the operation of selective mortality at younger ages (Vaupel and Yashin 1985), given the high non-White mortality rates of this era. And among children aged 0–9, non-White mortality was (and remained) higher than White mortality. Non-White children experienced a median of 890 influenza/pneumonia deaths per 100,000 during the pooled 1910–1917 years and a median 1,571 deaths per 100,000 in 1918, compared with a prepandemic median of 268 deaths per 100,000 and a pandemic median of 608 deaths for White children. Because of the high mortality among these younger ages, most of the absolute difference between non-White and White death counts in 1918 can be traced to differences among children aged 0–9 (Figure 2).

Potential Explanations for Reduced Disparities in 1918

A summary of findings from the suggestive tests conducted for each of the possible explanations for reduced racial disparities in 1918 urban pandemic mortality appears in Table 2. We describe the results pertaining to each of these explanations in turn.

Table 2 Possible explanations for reduced racial disparities in 1918 urban pandemic mortality

Research Questions and Testable Predictions	Results	Conclusions
City-Level Organization and Action		
A. Were levels of residential segregation, residential density, and illiteracy associated with non-White/White mortality ratios?		Cities with high prepandemic infectious disease mortality are also hit hard by the pandemic, but city demographics are unlikely to explain the specific mortality signature of the 1918 pandemic.
A1. Cities with high prepandemic mortality are hit harder by the 1918 pandemic	A1. Yes	
A2. Illiteracy, density, and segregation associated with mortality disparities in 1918	A2. No evidence in support	
B. Did nonpharmaceutical interventions (NPIs) offer protections and disproportionately benefit non-Whites?		NPIs help to explain cumulative total mortality but are unlikely to explain reduced racial disparities.
B1. NPI implementation associated with race-independent (total) mortality	B1. Yes	
B2. NPI implementation associated with race-specific mortalities and mortality disparities	B2. No evidence in support	
Social Immunology		
C. Did racial differences in herald wave exposure generate differences in partial immunity, disproportionately protecting non-White communities?		Partial immunity is unlikely to explain reduced fall disparities, though herald wave exposure may have mattered for nonimmunological reasons (e.g., behavioral changes). (Note: Low statistical power may reduce ability to detect associations; calculations are based on potentially noisy/biased proxy measures.)
C1. Non-Whites had greater spring wave exposure	C1. Yes	
C2. Greater spring wave exposure associated with lower fall wave mortality	C2. Mixed evidence (inconsistent)	
D. Did racial differences in early childhood flu exposure to the 1890 influenza virus produce greater influenza mortality among Whites compared with non-Whites in 1918?		We cannot rule out 1890 exposure as a major driver of high mortality among White young adults, and thus reduced disparities, in 1918. If 1890 exposure is to *fully* account for such high mortality, historical flu exposure and immunological imprinting parameters need to line up in relatively narrow ways, albeit ones consistent with some historical influenza literature. (Note: We are unable to directly measure imprinting.)
D1. Reduced 1918 disparities are driven by cohorts who would have had 1890 exposure	D1. Yes	
D2. Proportion of city residents with urban origins is greater for White than for non-White populations	D.2. Yes	
D3. Proportions of city residents with urban origins positively associated with mortality	D3. Yes (though only suggestive)	
D4. Aggregate mortality in the 20–29 and 30–39 age bands is consistent with "reasonable" mortality among the hypothetically imprinted	D4. Yes (but only if imprinting is close to ubiquitous in some cohorts)	

Table 2 (continued)

Research Questions and Testable Predictions	Results	Conclusions
D5. Estimated imprinting rates are consistent with "reasonable" historical influenza attack rates	D5. Yes (but the parameter space is highly constrained for the 20–29 age-group)	

Explanation A: Segregation, Illiteracy, and Residential Density

We assess the link between sociodemographic indicators and mortality patterns by testing whether levels of residential segregation and non-White/White ratios of residential density and illiteracy were associated with non-White/White mortality ratios in 1918. We expect that more highly segregated cities and cities with smaller racial disparities in illiteracy and residential density experienced smaller racial disparity in influenza/pneumonia mortality.

We observe that the race-specific baseline mortality—that is, the expected mortality in a city in 1918, given its mortality trends in the years 1910–1917—was a strong predictor of 1918 observed mortality. This suggests that communities that were generally vulnerable to infectious diseases remained so during the pandemic. But we find no evidence that segregation, illiteracy rates, or residential density were associated with racial disparities during the pandemic (see Table S1 in the online appendix). Our sociodemographic predictors from 1918 were also statistically indistinguishable from coefficients for nonpandemic years between 1910 and 1930. Although city characteristics and sociodemographic indicators may explain general infectious disease mortality rates during the early twentieth century, our findings suggest that they are insufficient to explain the specific mortality patterns and mortality disparities of the 1918 pandemic.

These findings are robust to the substitution of alternative segregation and density indices and to the use of logged race-specific mortality and excess mortality rather than logged mortality ratios as the dependent variable. In an additional model, we also include a measure of local coal-fired powerplant capacity as a proxy for air pollution, which has been found to be predictive of 1918 influenza mortality in an earlier study (Clay et al. 2019). This also does not predict 1918 non-White/White disparities.

Explanation B: Nonpharmaceutical Interventions

As in prior studies that used weekly mortality data (Hatchett et al. 2007; Markel et al. 2007), we find evidence that early NPI onset—that is, a shorter delay between the first locally reported infection and the implementation of NPI measures—was associated with lower annual non-White mortality. For White populations, we observe a similarly positive, but statistically insignificant, association between NPI delay and annual mortality. At first glance, these findings may suggest a slight but potentially

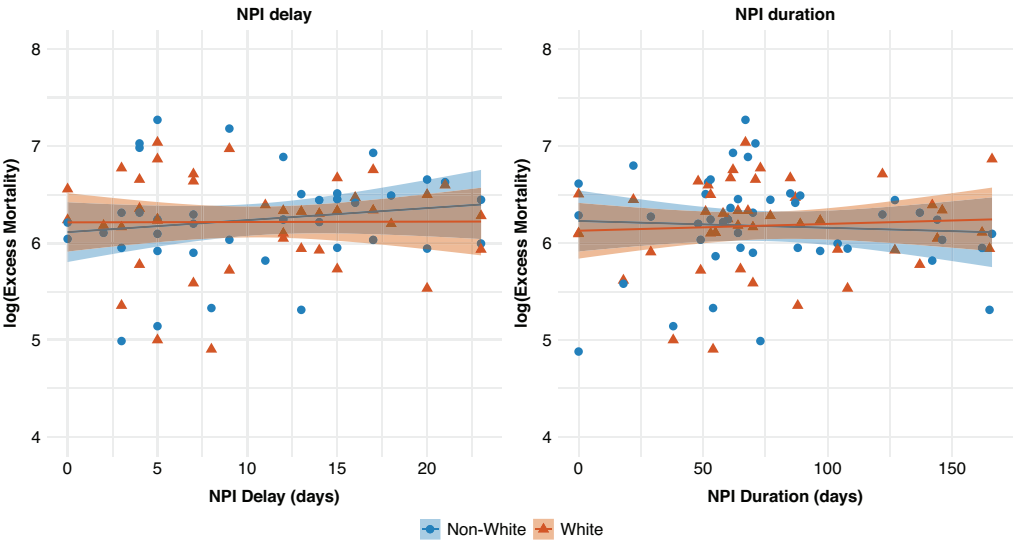

Fig. 3 Race-specific influenza and pneumonia excess mortality in 1918 relative to in prepandemic years, as a function of nonpharmaceutical interventions (NPIs). Linear regression trend lines and shaded 95% confidence bands are shown separately for the two populations.

disproportionate impact of NPIs on non-White mortality. Yet we find no statistically significant effect of NPI onset or NPI duration on non-White/White mortality ratios, and no evidence that coefficients for the association between NPIs and mortality were significantly different for non-White and White populations (Figure 3; see Table S2 in the online appendix). These null findings are robust to different model specifications and to the substitution of NPI data from other sources (Hatchett et al. 2007; Markel et al. 2007) for our measures. It therefore appears unlikely that the reduced mortality disparities during the 1918 pandemic were due to pronounced racially disparate impacts of ostensibly race-neutral measures such as NPIs.

Explanation C: Partial Immunity

We test the partial immunity explanation by answering the two following questions.

Question C1: Were Non-White Communities More Affected by the Herald Wave Than White Communities?

Cities that experienced high influenza mortality during the 1910–1917 period were also more likely to experience high mortality during the 1918 spring wave. Alongside this city-specific mortality penalty, non-White populations experienced a more severe spring wave than White populations (Figure 4). In comparison with the period 1910–1917, median non-White mortality increased by 50% while median White mortality increased by 30%. As a result, the median non-White/White mortality ratio in the spring of 1918 was 2.79, compared with 2.43 during the pooled period 1910–1917. This made the spring of 1918 among the most unequal periods recorded

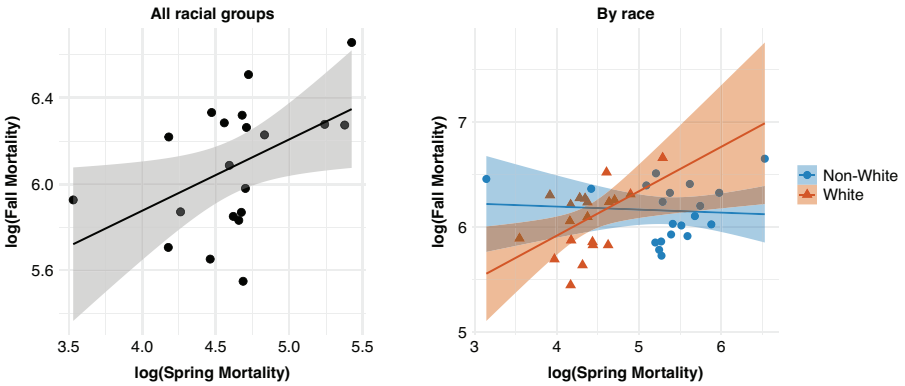

Fig. 4 Fall 1918 influenza and pneumonia mortality as a function of spring 1918 influenza and pneumonia mortality, logged, for all racial groups and by race. Linear regression trend lines and shaded 95% confidence bands are shown for the full population (left panel) and separately for non-White and White populations (right panel).

in our prepandemic data, tying mortality disparities observed during the 1912 spring wave and exceeding disparities during the (least unequal) 1915 spring wave by 42%.

Question C2: Across Cities, Was Greater Exposure to the Herald Wave Associated With Lower Mortality During the Deadlier Fall Wave?

We predict a race-independent negative spring/fall association, since populations that experienced greater mortality in the spring should be partially immune to the pandemic viral strain and therefore experience lower mortality during the fall. Yet we find the association between spring and fall mortality to be indistinguishable from zero for non-White populations and either indistinguishable from zero or positive for White populations, depending on the control variables included in the model (Figure 4). This null finding is robust to changes in the dependent variable (e.g., using mortality ratios rather than race-specific mortality and using spring/fall mortality data published in the appendix to Crosby 2003); to a substitution of NPI data taken from Markel et al. (2007) for our original data; to different specifications of the herald wave's duration; and to a stratification of our model into multiple tiers of herald wave severity (all results are shown in Table S4 in the online appendix). These findings make it unlikely that mortality disparities during the pandemic can be explained by greater partial immunity among non-White populations as a result of herald wave exposure.

Overall Assessment of the Analysis of Partial Immunity

Although we are able to examine the association between spring exposure and fall mortality more systematically than were prior studies, our analysis has two major limitations. First, the relatively small number of cities included in the sample limits the statistical power of our models. The power of models that test the association between spring wave exposure and fall mortality ranges from 0.21 to 0.62: well below a conventional threshold of 0.8. This means that we would be able to

distinguish only large effects, and it leads us to treat our null finding as merely suggestive evidence that needs to be corroborated through future historical work. Second, if infection fatality rates were higher in non-White populations, then excess deaths among those populations should imply fewer infections than the same excess deaths in White populations. The exposure inferred from mortality would then be erroneously high for non-White populations versus White populations, and our estimates of disparities in herald wave exposure would be upwardly biased.

The potential bias in overstating exposure disparities would lead us to overstate one key premise of the herald wave hypothesis (C1 in Table 2), but the potential noisiness could contribute to disguising a clear signal of the other key premise, namely, a race-independent negative association between spring exposure and fall mortality (C2 in Table 2). Given that such an association is essential to the partial immunity hypothesis, the potential overstatement of non-White/White exposure disparities is a less acute limitation than the models' low statistical power and potential cross-city measurement error. Perhaps the best summary of the total state of evidence that can be given is that, at the end of our analysis, it remains the case that—across all literature on the 1918 pandemic—there is no clear empirical evidence *for* the herald wave hypothesis.

We are also unable to determine whether statistically significant differences between non-White and White spring/fall mortality associations are artifacts of our models or hint at potential race-specific effects of herald wave exposure. Such race-specific effects would lend themselves to multiple possible interpretations. The distributions of White and non-White populations' spring exposure barely overlap (Figure 4), leaving the possibility that genuinely protective partial immunity was discernible only at the high exposure levels of non-White populations. Alternatively, potentially race-specific effects of herald wave exposure may reflect behavioral, rather than immunological, responses. Because the regular hospital system often refused to admit African Americans or triaged them into substandard care, public health campaigns in non-White communities relied more heavily on community-based prevention and education (Gamble 2010; Krishnan et al. 2020; Schlabach 2019). Those initiatives, as well as the persistent experience of high infectious disease mortality, may have contributed to greater lay knowledge about infectious diseases in urban non-White communities (Økland and Mamelund 2019:13) and precipitated greater vigilance during the pandemic even as mainstream newspapers initially played down the severity of infections (Crosby 2003).

Explanation D: Immunological Imprinting

No direct measure of exposure to the 1890 influenza virus is available, so as an alternative, we conduct suggestive tests of five predictions (summarized in Table 2) based on necessary conditions for the hypothesis that immunological imprinting explains reduced 1918 disparities (outlined in Table 3).

Analysis D1: Impact of Young Adults on Mortality Disparities

We find that the reduced disparities in 1918 were driven by the age bands that would have been most likely to have first flu exposure to the 1890 pandemic strain (Figure 5): reduced disparities reflect strikingly high mortality among urban Whites in their 20s

Table 3 Summary of evaluation of the "imprinting explanation": The idea that racially differential patterns of early childhood exposure to the 1890–1892 influenza explain the reduced racial disparities in 1918 pandemic mortality

Necessary Conditions for the Imprinting Explanation	Analysis	Result
1. Reduced aggregate disparities in 1918 were driven by cohorts that could have been exposed to the 1890 virus during critical developmental periods.	Empirical	Yes: Reduced disparities were driven by cohorts aged 20–39 in 1918.
2. Either a. relevant cohorts of urban White 1918 populations had greater exposure to the 1890 flu than relevant cohorts of urban non-White 1918 populations *or* b. relevant cohorts of these populations had similar exposures, which swamped other factors that tended to produce higher non-White influenza mortality in other years.	Empirical	Suggestive yes (2a): Using urban origins as a proxy for childhood influenza exposure, young adult urban White populations had greater exposure than young adult urban non-White populations. Suggestive no (2b): City-level factors were not less predictive of disparities in 1918 compared to prior years, failing to provide evidence of imprinting "swamping" other factors (analysis reported in the online appendix).
3. Populations with greater 1890 flu exposure had higher mortality in 1918.	Empirical	Suggestive yes: Using urban origins as a proxy for childhood influenza exposure, city populations with greater exposure had higher 1918 mortality.
4. 1890 influenza exposure was sufficiently a. prevalent *and* b. deleterious to survival in 1918 to account for reduced racial disparities in the relevant cohorts.	Simulation	Not disproven: Simulations find that, to account for reduced disparities, imprinting would need to be highly prevalent among urban Whites in relevant cohorts, but that this prevalence could result from plausible attack rates.

Note: The leftmost column lists necessary conditions for the explanation to hold. Conditions 1–3 are evaluated, respectively, by tests D1–D3 of Table 2. Conditions 4a and 4b are jointly evaluated through test D4, and the outcome of test D4 is further evaluated through test D5.

and 30s (as already shown in Figure 2). A counterfactual estimate of mortality disparities suggests that up to three fourths of the decline in mortality disparities in 1918 (compared with the 1910–1917 period) was due to reduced disparities among those aged 20–29 and 30–39 (details are provided in section IIIA in the online appendix).

The key role of these two age-groups is consistent with the hypothesis that immunological imprinting accounts for reduced racial disparities. However, that hypothesis seemingly leaves the relatively small disparities at ages 10–19—while not as small in absolute terms or relative to prepandemic values as those for the 20–29 and 30–39 age-groups—as an anomaly. Yet, from a broader perspective in which deleterious imprinting works hand in hand with helpful H1 exposures (Worobey et al. 2014:8110–8111), this age-group may be less anomalous: they may have been likely candidates for H1 exposure in the years before the 1918 pandemic, particularly for non-White children living in appallingly poor urban conditions. Urban White children and teenagers may have been more likely to be fully immunologically naive to H1N1.

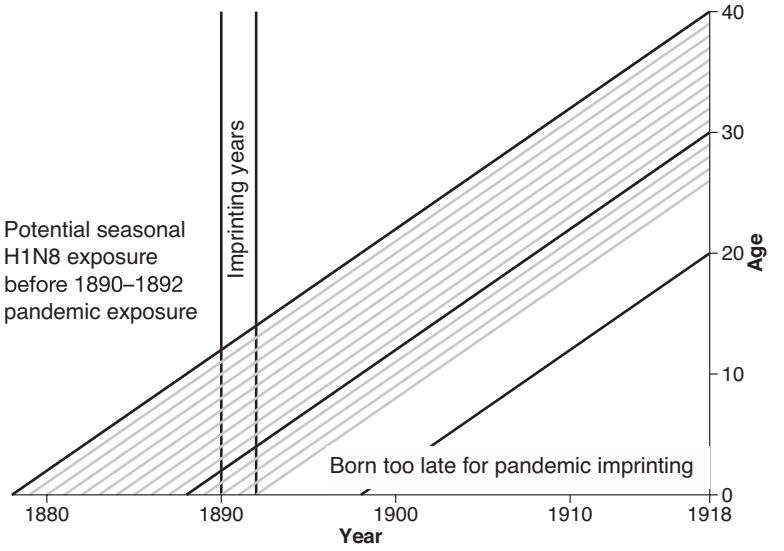

Fig. 5 Lexis diagram illustrating cohorts that might have been immunologically imprinted by the 1890–1892 pandemic flu virus, depicted in gray

Analysis D2: Differential 1890 Exposure

Since no direct measure of exposure to the 1890 virus is available, we use urban residency during childhood and adolescence as a proxy for 1890 flu exposure, given greater infectious disease prevalence in urban compared with rural areas (Feigenbaum et al. 2020). We find that such urban origins were more prevalent among the 1918 urban White population than among the 1918 urban non-White population (Condition 2a in Table 3; see discussion of alternative Condition 2b in section IIIA in the online appendix). Specifically, among cohorts aged 20–39 in 1918 and residing in cities in 1910, we estimated the national proportion with long-term urban residency at nearly half of the non-White population compared with nearly two thirds of the White population. We estimated the proportion with long-term residence in cities of at least 100,000 residents (whose influenza exposure may have been heightened; Feigenbaum et al. 2020) at around one fifth of non-White and more than one third of White residents. Thus, we find that it is indeed plausible that urban White young adults in 1918 may have had higher childhood exposure to the 1890 pandemic than urban non-White young adults in 1918, owing to the White individuals' greater chances of having spent childhood in a city. Because these calculations require the linking of individuals across multiple censuses, they include considerable uncertainty owing to the small percentage of linkable persons; we discuss this limitation, and other choices made in constructing this measure, in section IIIA in the online appendix.

Analysis D3: Effect of 1890 Exposure on Mortality in 1918

We find that cities with a larger proportion of longtime urban residents had higher 1918 mortality. Specifically, we tested whether the estimated city-level variation in

the portion of the total, White, and non-White populations with urban origins was associated with greater 1918 mortality in total and in the 20–29 and 30–39 age-groups, with and without controls for NPI timing and duration. This association is in the expected direction (i.e., having more residents with urban background is associated with greater mortality) in all models. We also find that having a higher proportion of residents with urban origins is associated with reduced non-White/White mortality ratios, although the coefficient becomes statistically insignificant when we control for NPIs (full regression results are reported in Table S5 in the online appendix). Given the small samples and imprecise measures, we consider these results to be broadly suggestive but hardly definitive.

Analysis D4: Simulated Individual-Level Effect Sizes

To evaluate whether the imprinting explanation rests on plausible assumptions about the prevalence and consequences of imprinting, we simulate cohort mixtures of imprinted and nonimprinted individuals. We find that the imprinting explanation need not require imprinting prevalence or individual-level effects (Conditions 4a and 4b in Table 3) that are clearly implausible. Specifically, our simulations answer two questions. First, how high would the mortality of the imprinted individuals need to be, as a function of the imprinted percentage in each cohort, to generate the observed age-group-specific, race-specific mortality rates? Assuming an upper limit on "reasonable" mortality rates imposes a lower limit on what portion of this 10-year age-group must have been imprinted. When combined with assumptions about how many single-year cohorts were partially imprinted, this also generates lower limits on within-cohort imprinting. These lower bounds on the portion of a birth cohort that would need to be imprinted can be evaluated for plausibility. Second, what rates of exposure to previous flus could generate these cohort imprinting rates? High rates of imprinting imply high rates of 1890 pandemic exposure and either low rates of prior H1N8 exposure (i.e., the seasonal flu variants circulating before 1890) or high rates of immunological refocusing, all of which can likewise be evaluated for plausibility. The simulated answers to this second question, in particular, are exploratory. Our goal is not to definitively evaluate these immunological hypotheses (with and without refocusing) as an explanation of 1918's reduced racial disparities, but rather, by clarifying their implications, to construct a foundation that would allow them to be evaluated in future historical virological work.

To address the first question, we first leverage the sharper age cutoff in the 20–29 age-group: individuals who were younger than 25 in 1918 were too young to have been exposed to the "Russian" flu, without antibodies developed *in utero* or while breastfeeding, in 1890–1893 (using 1893 as a generous cutoff date for the pandemic). Given the immense aggregate 1918 mortality in the 20–29 age-group, we estimate that this restriction implies very large individual-level imprinting effects indeed: to fully account for the greater aggregate mortality in this age-group compared with others, 1890 imprinting would need to increase individual-level mortality by a factor of at least 1.9 in non-White populations and 3.75 in White populations. Constraining the individual-level effect to be on the smaller end of the range of effect sizes essentially implies that the cohorts aged 25–29 had close to universal 1890 imprinting among

urban White individuals. For example, urban White people aged 25–29 who were imprinted by the 1890 virus in childhood would have to have had greater mortality than urban White people aged 70+ in 1918, unless at least 85% of urban Whites in the 25- to 29-year-old cohort had been imprinted (see Figure S2 in the online appendix; details, including equations, are given in section IIIBi). For comparison, serological evidence from the Netherlands (not from a specifically urban population) found that about half of the 1893 birth cohort showed evidence of early H3 imprinting (Dowdle 1999).

Since the mortality jump among those aged 20–39, compared with surrounding age-groups, is smaller in the non-White population, this same constraint is compatible with a far greater range of 1890 cohort imprinting, ranging below 60%. To the extent that the urban White population's greater likelihood of urban childhood corresponds to a higher prevalence of imprinting, the smaller mortality increase for non-White compared with White young adults is compatible with these differences in origin.

Analysis D5: Historical Plausibility of Estimated Imprinting Rates

To provide some rough, exploratory calibration of the individual-level flu attack rates implied by these cohort exposures, we simulated annual rates of childhood exposure to H1 strains before 1890, rates of exposure to an H3N8 influenza in 1890–1892 for urban and rural populations, and rates (including 0%) of immunological refocusing conditional on exposure to H3N8 after exposure to an H1 strain. These simulations suggest that, for immunological imprinting to account for the dramatic spike in urban White young adult mortality, either the rate of exposure during the 1890–1892 pandemic or the rate of immunological refocusing given exposure in that pandemic must have been very high (see Figure S3 in the online appendix; details, including equations, are given in section IIIBii)—though not outside the boundaries of the attack rates estimated for the 1890 flu (Valleron et al. 2010), or for pandemic influenza attack rates generally (Mathews et al. 2009:147; Saunders-Hastings and Krewski 2016:7).

Discussion and Conclusion

Analyses of city-level mortality data suggest that racial disparities during the 1918 pandemic were unexpectedly small across cities. The excess mortality for non-White compared with White populations shrank 74% (from 133% to 35% excess), driven primarily by unusually high mortality among Whites aged 20–39. These findings are surprising, since racial disparities in infectious disease mortality were staggeringly large during the early twentieth century (Feigenbaum et al. 2019), and since public health responses to the pandemic routinely provided substandard care to non-White communities (Bristow 1992; Byrd and Clayton 2001).

In 1918, public health officials attempted to explain reduced mortality disparities with racial pseudo-science that cast non-White populations as naturally immune to infectious diseases (Schlabach 2019). Subsequent scholarly works took a very different perspective and focused on race-specific immunological histories and the spatial

geography of urban life in the early twentieth century (Crosby 2003; Grantz et al. 2016; Krishnan et al. 2020; Økland and Mamelund 2019), although none tested these hypotheses systematically.

Our findings, which are based on analyzing race-specific mortality and a host of city-level characteristics for up to 70 cities, do not support two of the most frequently cited hypotheses: partial immunity in non-White communities stemming from higher exposure to a spring herald wave and reduced non-White exposure as a result of residential segregation. In particular, prior studies (Crosby 2003; Økland and Mamelund 2019) have proposed the herald wave hypothesis as a plausible, but empirically unsubstantiated, explanation for reduced racial disparities during the fall pandemic. We test it more systematically than prior studies and find no evidence to support it. However, limitations owing to scarce historical data prevent us from conclusively rejecting the hypothesis; these partial immunity results are merely suggestive because of the limited statistical power of our models. We also do not find support for the hypothesis that nonpharmaceutical interventions, such as school closings, which reduced overall mortality (Hatchett et al. 2007; Markel et al. 2007), explain reduced mortality disparities.

Instead, our findings produce a diverse and suggestive set of evidence suggesting that the unique migration and public health histories of non-White and White populations may have intersected with the virology of the 1918 influenza to produce especially high mortality among White young adults, thereby contributing to the unexpectedly reduced mortality disparities. This explanation draws on the idea that "immunological imprinting" to the 1890 influenza pandemic in early childhood may have driven immunological responses in 1918, nearly three decades later (Gagnon et al. 2013; Worobey et al. 2014). We hypothesize that the disproportionately urban origins of urban White young adults in 1918, in contrast with the fact that non-White young adults had often migrated from rural areas, may have resulted in differential exposure to the 1890 flu pandemic during critical childhood developmental windows. This exposure then produced a unique racial patterning of vulnerability in 1918. We attempt to refute this novel explanation for reduced racial disparities in five empirical and simulation analyses; all five instead produce results broadly compatible with the explanation—although its mechanisms would have had to operate nearly universally in key cohorts to fully account for the distinctively high mortality among White young adults.

More broadly, these results establish that distinctive exposure histories among different racial groups and racial patterns of mortality can be used to develop and evaluate hypotheses in historical virology. Some of the specific analyses reported here will likely be obviated if future virological work confirms the still-speculative hypothesis that the 1890 pandemic was caused by a coronavirus (Brüssow and Brüssow 2021; Vijgen et al. 2006; Vijgen et al. 2005) rather than an H3N8 influenza (Dowdle 2006; Worobey et al. 2014). In that case, patterns of prior H1 influenza exposure will remain relevant to outcomes in 1918, but patterns of exposure to the 1890 pandemic may not, unless their mechanism is not immunological imprinting but rather long-term morbidity stemming from childhood exposure to the coronavirus.

Our analyses also leave room for competing hypotheses to be resuscitated or adapted. For example, non-White communities may have changed their behavior to

reduce their influenza exposure in the fall of 1918. These populations, excluded from the regular public health system but accustomed to high infectious disease mortality, regularly relied on community-based education and prevention to reduce infections and may have been more circumspect during the pandemic's peak (Krishnan et al. 2020; Schlabach 2019). While our data cannot test this hypothesis directly, our (underpowered) findings of a possible race-specific association between herald wave exposure and pandemic mortality offer suggestive support to explanations that reflect the agency and social action of Black communities.

More broadly, this work offers a general approach to analyzing racial disparities in infectious disease mortality that is widely applicable to a variety of periods, contexts, and microbes. We treat racial categories as socially constructed proxies for group-specific social histories that reflect broader structures of inequality, not as ready-made biological or demographic facts. The mortality signature observed in the 1918 pandemic results from the interaction of a particular microbe with those racialized social histories of exposure. Understanding the patterns and drivers of inequality in infectious disease mortality therefore requires examining how structural and institutional arrangements shape social and microbial exposures over time.

Perhaps most importantly, simplistic comparisons between 2020, 2021, or 2022 and 1918 are misguided because the specific virology of COVID-19 and the historically rooted vulnerabilities of different social and racialized groups differ from those of the early twentieth century. It is notable that mortality at the peak of the 1918 pandemic was substantially higher than monthly mortality rates recorded during the COVID-19 pandemic (Weinberger et al. 2020). More generally, while racial disparities are usually conditioned by contexts of structural racism, their proximate causes may differ across pandemics. For example, in the 1918 pandemic, Black populations had lower morbidity but higher case fatality (Krishnan et al. 2020; Økland and Mamelund 2019), but studies of the COVID-19 pandemic return ambiguous results about the relative importance of exposure and immunological susceptibility (Navar et al. 2021; Ogedegbe et al. 2020; Rentsch et al. 2020; Zelner et al. 2021). The details of one pandemic's course cannot be imported wholesale to understand another. The enduring lesson is, rather, the broader framework of attending to the intersection between the natural history of a microbe and the social histories of the populations that are exposed to it. ■

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