Racial Disparities in 1918 Flu Mortality: What Drove Them & What We Can Learn for Today

Elizabeth Wrigley-Field

BWFD
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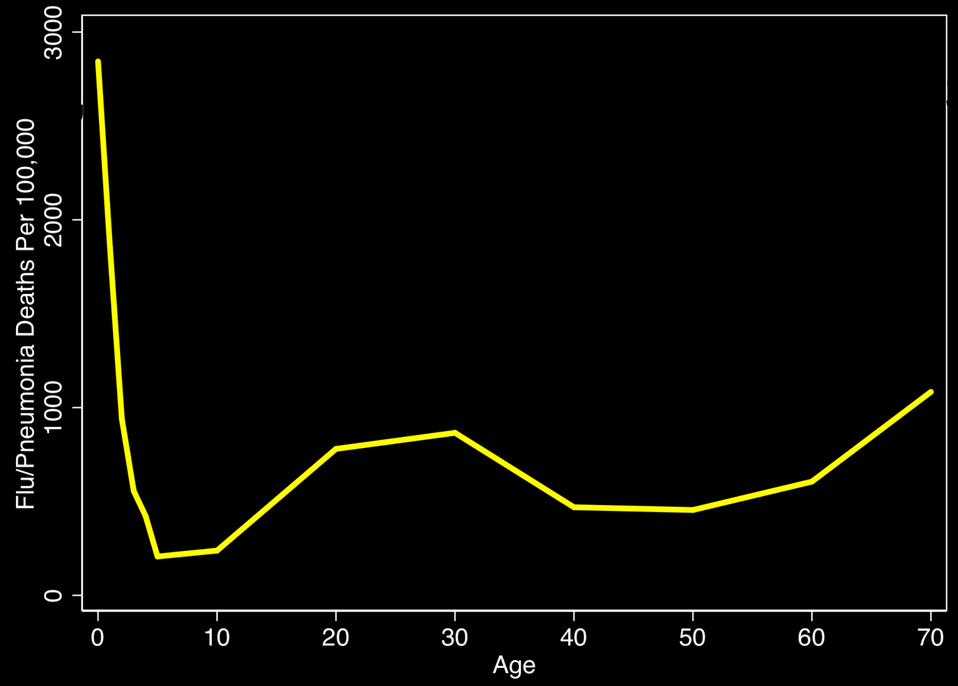
Racial Disparities in the 1918 Flu Pandemic in U.S. Cities

Collaborators:
Martin Eiermann, Courtney Boen, James Feigenbaum, Jonas Helgertz, Elaine Hernandez

What was distinctive about 1918?

1. Extreme virulence

2. Weird age pattern: Young adults hit very hard



What was distinctive about 1918?

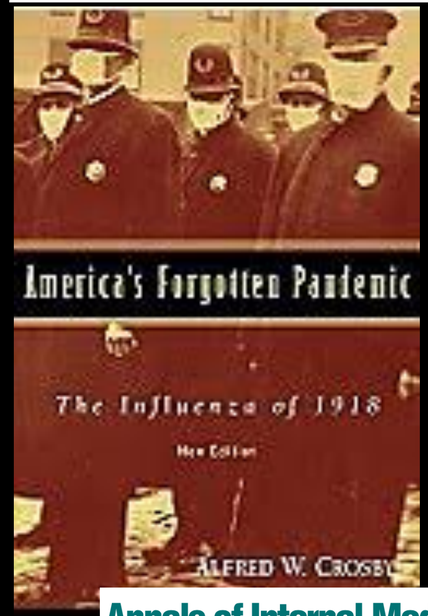
1. Extreme virulence
2. Weird age pattern: Young adults hit very hard
3. In U.S. cities, strikingly small racial disparities

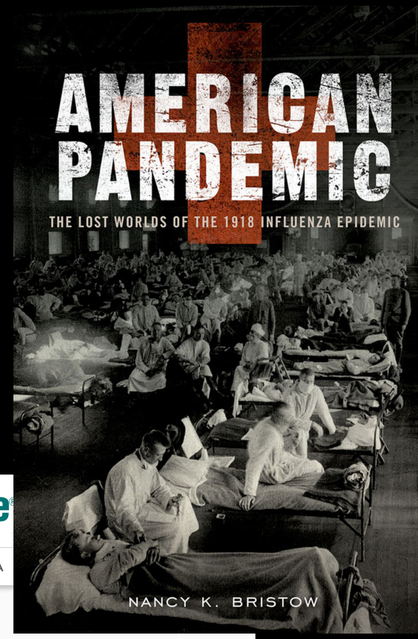
Small racial disparities in 1918

"During these terrible weeks, while the epidemic raged, God has been trying in a very pronouncedly conspicuous and vigorous way, to beat a little sense into the white man's head; has been trying to show him 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**."

—Rev. Francis J. Grimke

quoted in Nancy K. Bristow, *American Pandemic*





Demography (2019) 56:1371–1388
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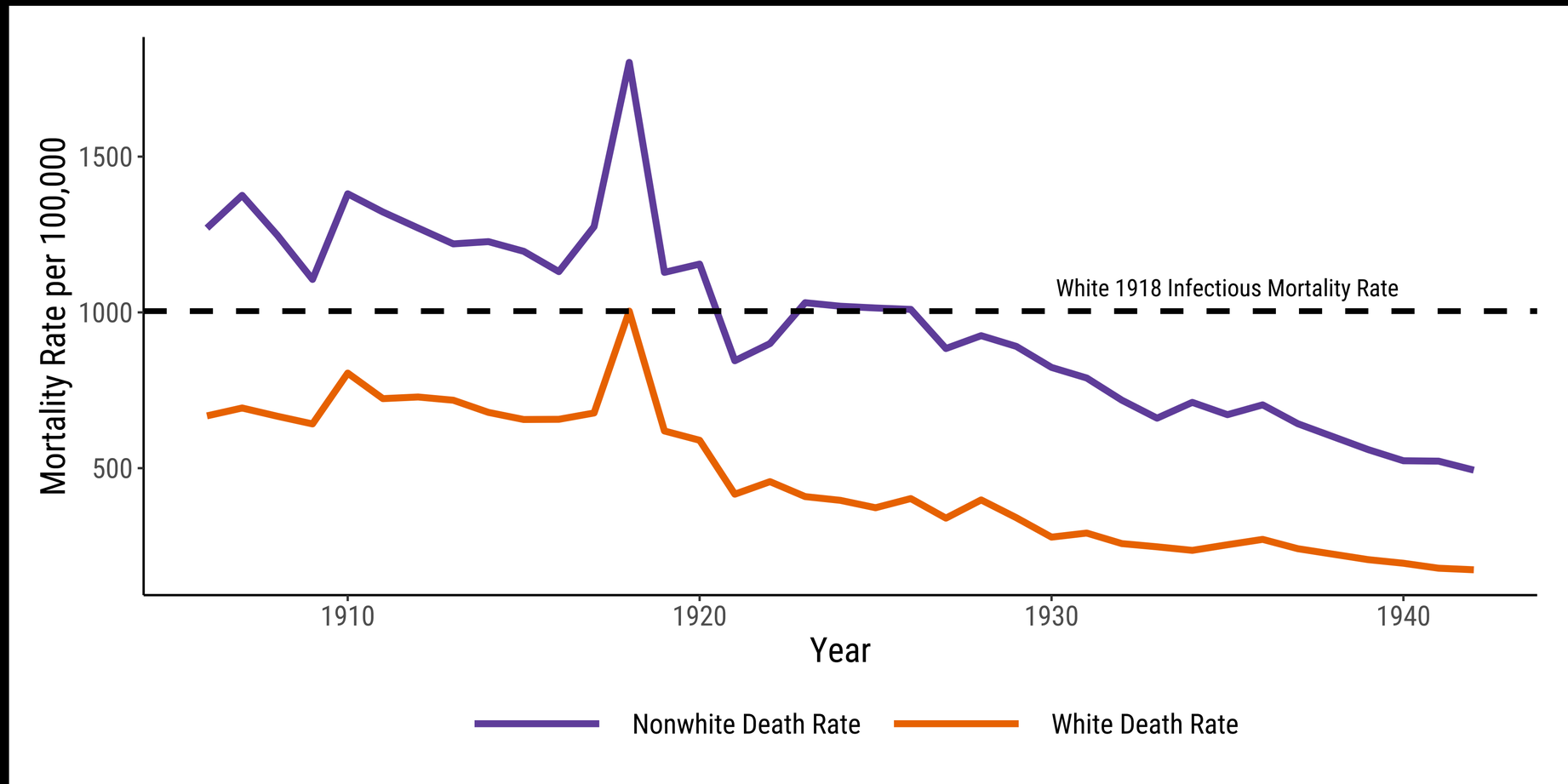
Regional and Racial Inequality in Infectious Disease Mortality in U.S. Cities, 1900–1948

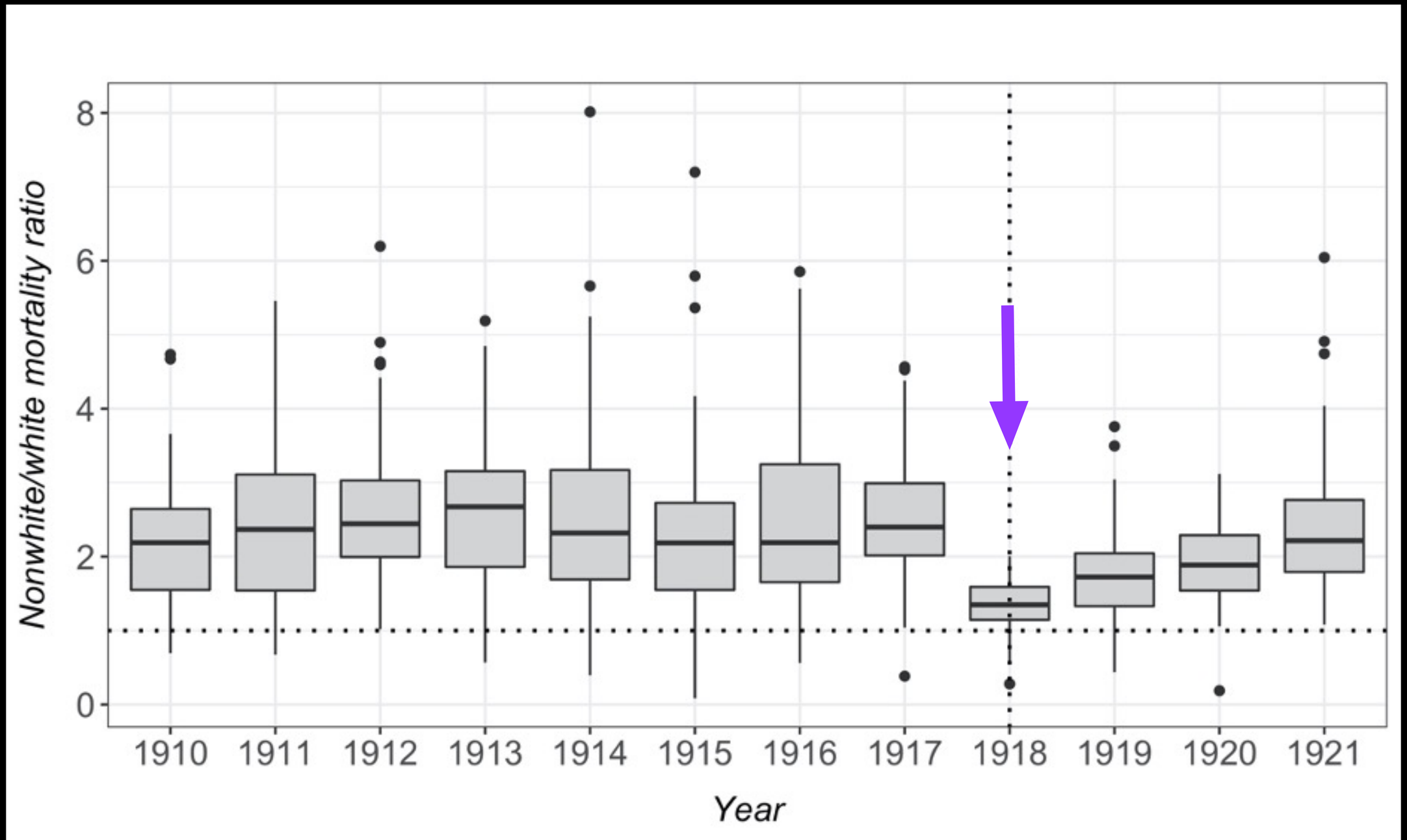
James J. Feigenbaum [1,2] · Christopher Muller [3] · Elizabeth Wrigley-Field [4]



Descriptive Results:

Yes, the disparities were small (relatively)

Small racial disparities in 1918



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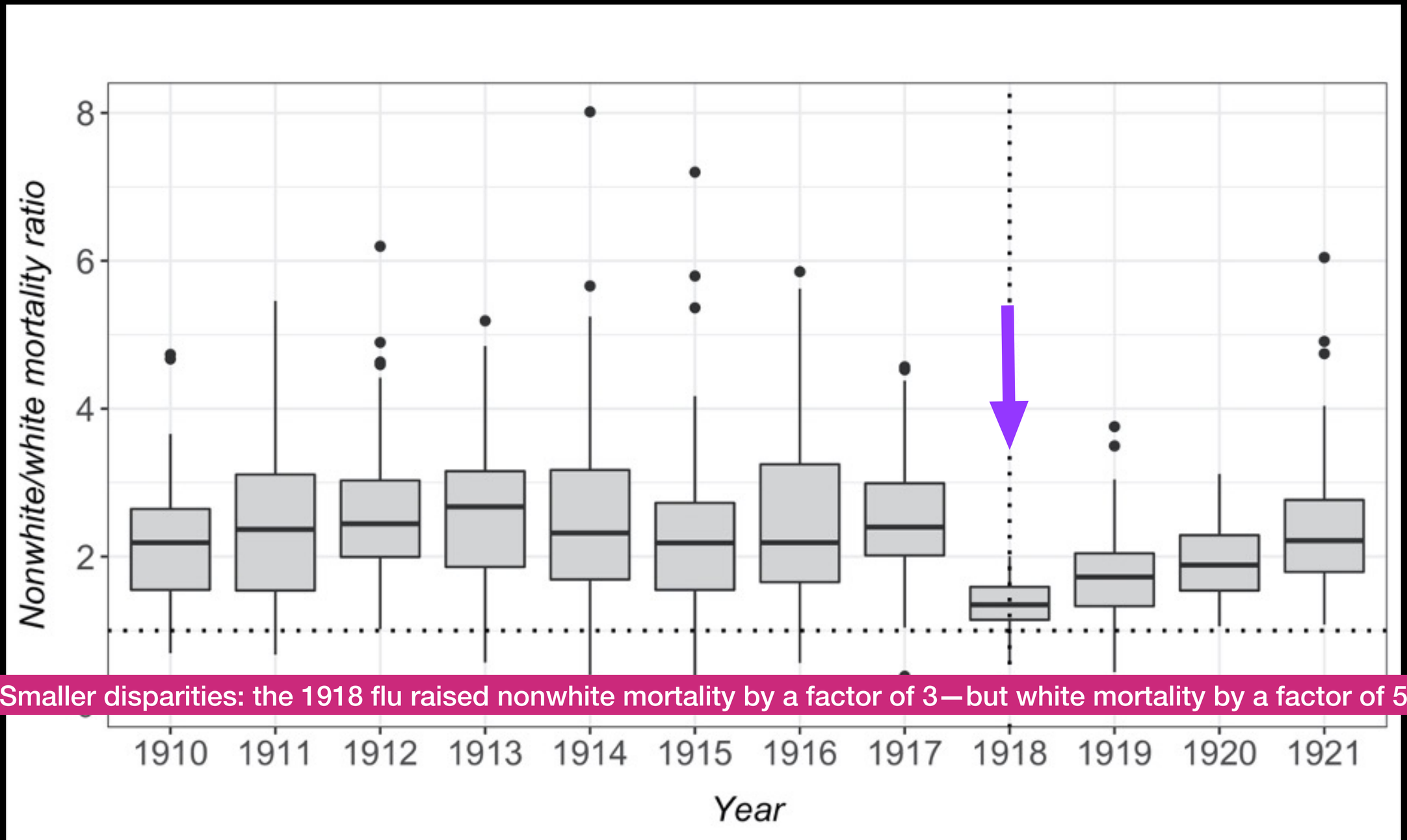

Smaller disparities: the 1918 flu raised nonwhite mortality by a factor of 3—but white mortality by a factor of 5

Race-specific urban mortality measures

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	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
Population counts, cause- and race-specific	1205	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 dataset 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 dataset	Markel et al. (2007)
Spring wave influenza mortality (alternative measure)	45	1918		Crosby (2003)
Infant mortality, city-specific	43	1910		Collins (1930)
Air pollution, 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)

Table 1. Datasets and data sources

Population denominators

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- Main measure: Population counts interpolated (log scale) 1910-1930
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- Alternative measure: Predict population size from non-infectious death counts
  - Estimate population sizes from non-infectious death counts in 1906-1910 (Census-calibrated population sizes), with city fixed effects and linear time trends; all race-specific
  - Predict out-of-sample population sizes for just before the 1918 pandemic

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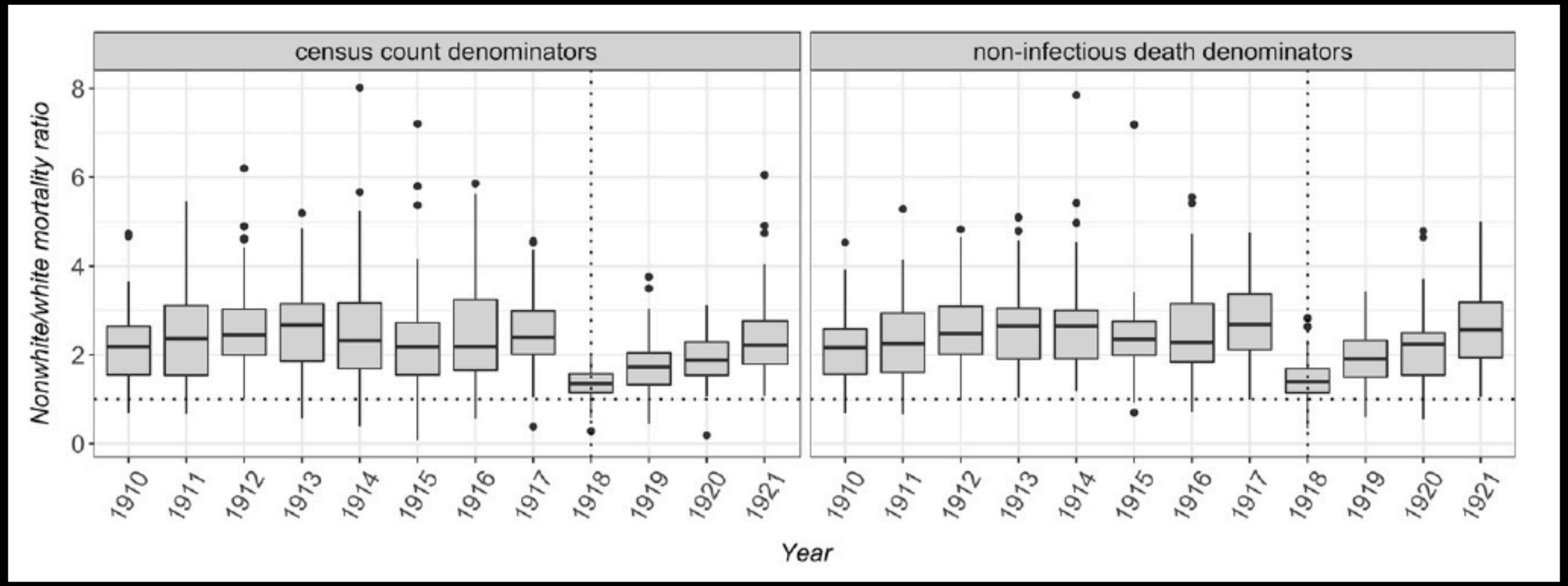

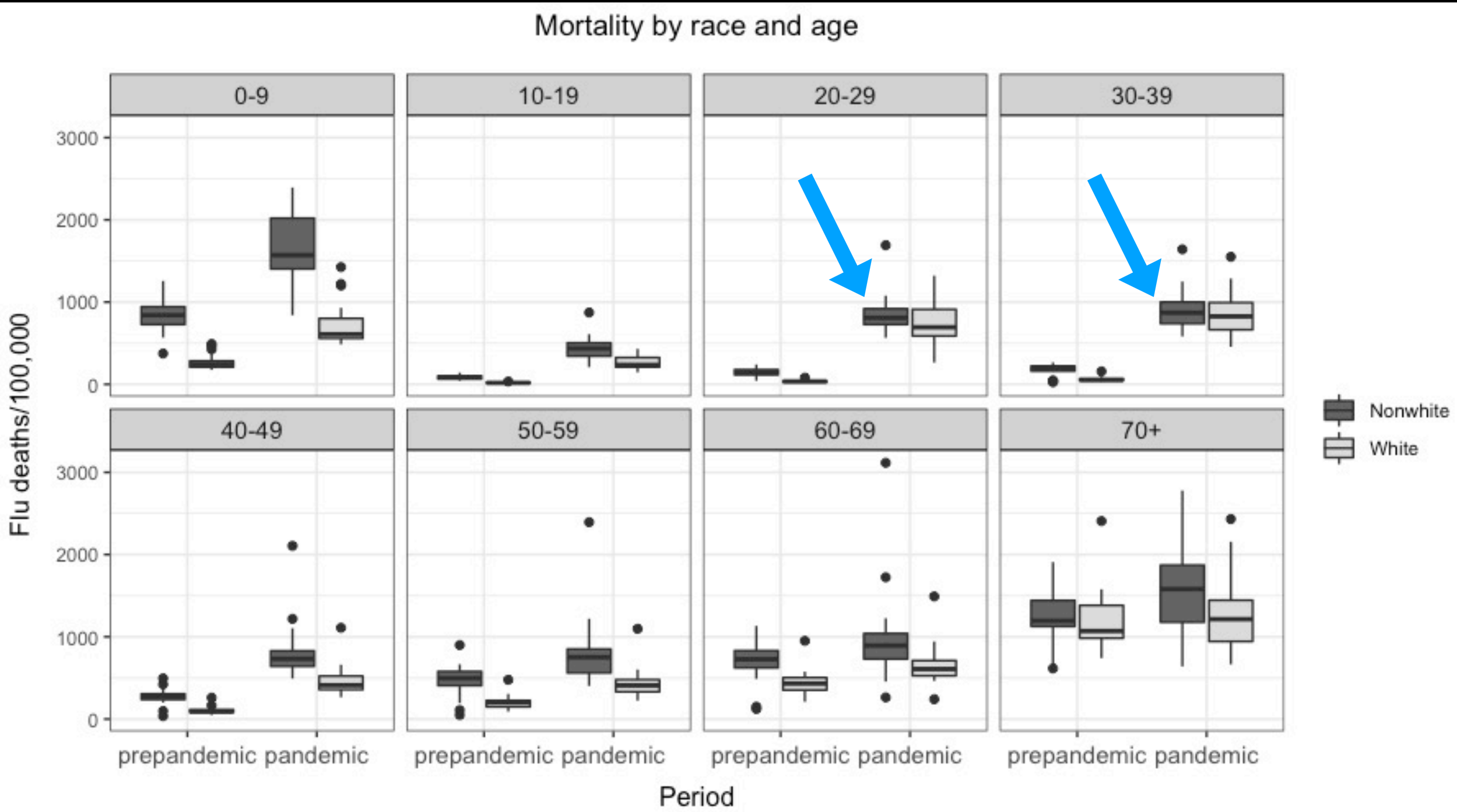
Mortality by race and age
0-9
10-19
20-29
30-39
40-49
50-59
60-69
70+
3000
2000
1000
0
Flu deaths/100,000
prepandemic
pandemic
Period
Nonwhite
White

Potential Explanations

for small racial disparities

Potential Explanations:

City-Level Factors

Why were the 1918 disparities so small?

- Did non-pharmaceutical interventions (school closures, mask mandates) help non-whites more because they were generally at greater risk?
- Is city-level racial segregation associated with larger or smaller disparities?

  (Segregation measures: Dissimilarity index, divergence index, variance ratio index, and sequence index)

Why didn’t NPIs matter more?

"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.”

— a Topeka, Kansas newspaper article in 1918

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Potential Explanations:

Social Immunology

The social immunology of the 1918 flu

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The distinctive social history of the white and nonwhite populations interacts with the distinctive natural history of the 1918 virus

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Hypothesis	Prior exposure protective or harmful?	Which prior exposure?	Exposure in which years?	Youngest cohort likely to be affected	Oldest cohort likely to be affected	Canonical citations [only H1 citations are about racial disparities]
H1: Spring wave	Protective	1918 flu	1918	No limit	No limit	Crosby (2003), Okland and Mamelund (2019)
H2: Early “1918” flu	Protective	1918 flu	1915-1918	No limit	No limit	Worobey (2019)
H3: H1 partial immunity	Protective	Any H1 virus	1900-1918 and pre-1889	No hard limit, but 1889-99ish; older is more plausible	No hard limit, but younger is generally more plausible	Worobey et al. (2014)
H4: Fetal harms	Harmful	1890 flu (“Russian”)	1889-1893	1894 birth	1890 birth	Gagnon et al. (various)
H5: Original antigenic sin (immunological imprinting)	Harmful	1890 flu (“Russian”)	1889-1893	1892 or 1893 birth?	~1885 birth? Younger is more plausible	Worobey et al. (2014, 2019), Gagnon et al. (various)
H6: Original antigenic sin plus immunological refocusing	Harmful	1890 flu (“Russian”)	1889-1893	1892 or 1893 birth?	1879-1880 birth?	Gagnon et al. (various)

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Immunological imprinting hypothesis

- History of circulating flu strains: Leading hypotheses (Worobey et al. 2014)
  - Before 1890: H1N8 or similar flu circulates
  - 1890 pandemic (~1889-1892ish): H3N8 virus
  - 1900 onward: H1N8 or similar flu circulates
  - 1918 pandemic, whenever it began: H1N1
- Key fact: H1 and H3 are phylogenetically distant
- Hypothesis: First exposure to H3N8 in its pandemic form shaped the immune system in ways that were harmful in 1918

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Our extension:

Did non-white urban populations have less 1890ish childhood flu exposure than urban white populations, because they were less likely to grow up in cities?

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The immunological imprinting hypothesis requires…

1. Small disparities are driven by cohorts whose first flu exposure could have been ~1890-1893

2. Urban non-white members of those cohorts had less exposure ~1890-1893 than urban white members

3. First flu exposure ~1890-1893 was associated with worse outcomes in 1918 at the city level

4. First flu exposure ~1890-1893 was sufficiently prevalent in those cohorts to account for small disparities

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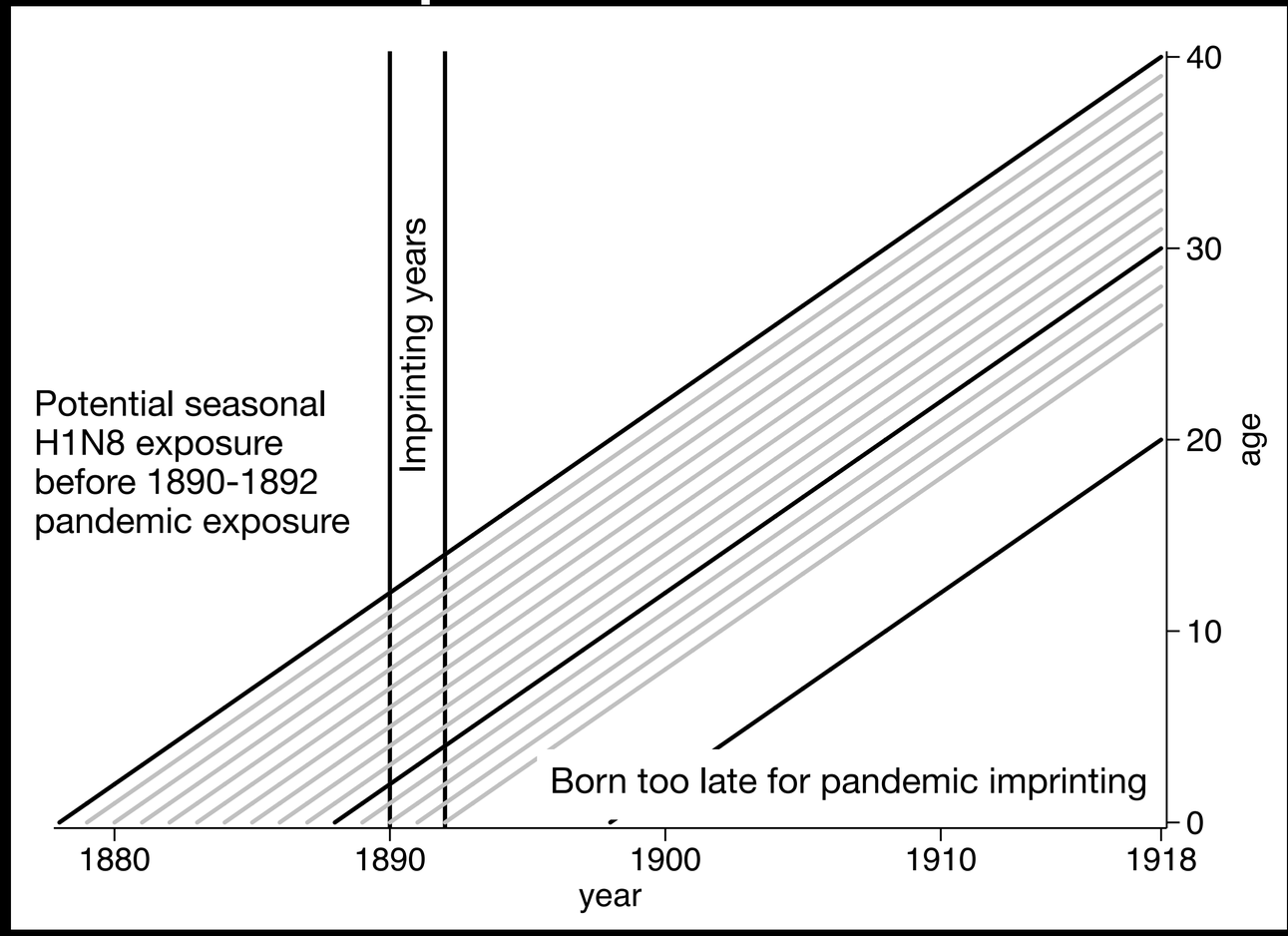

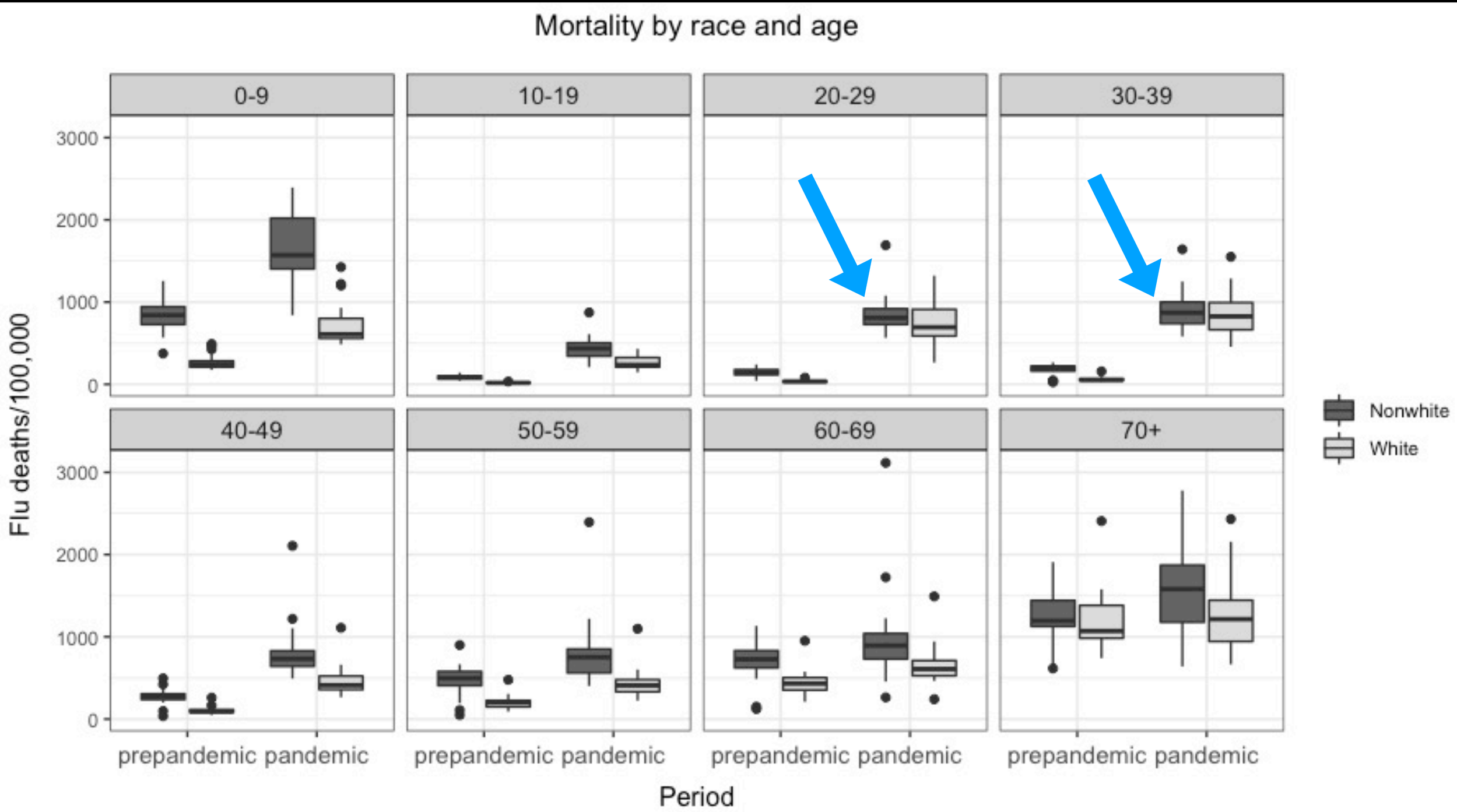
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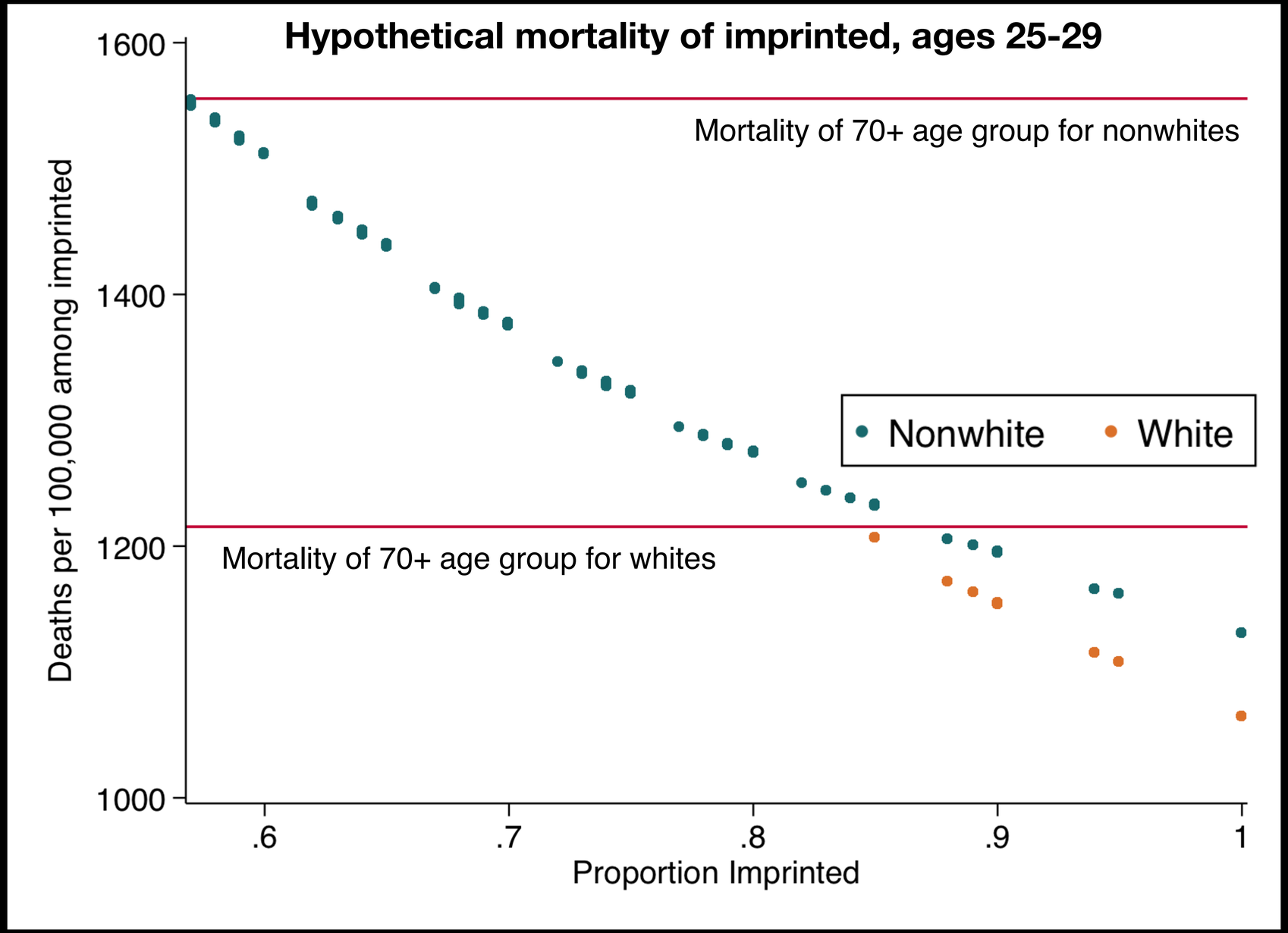

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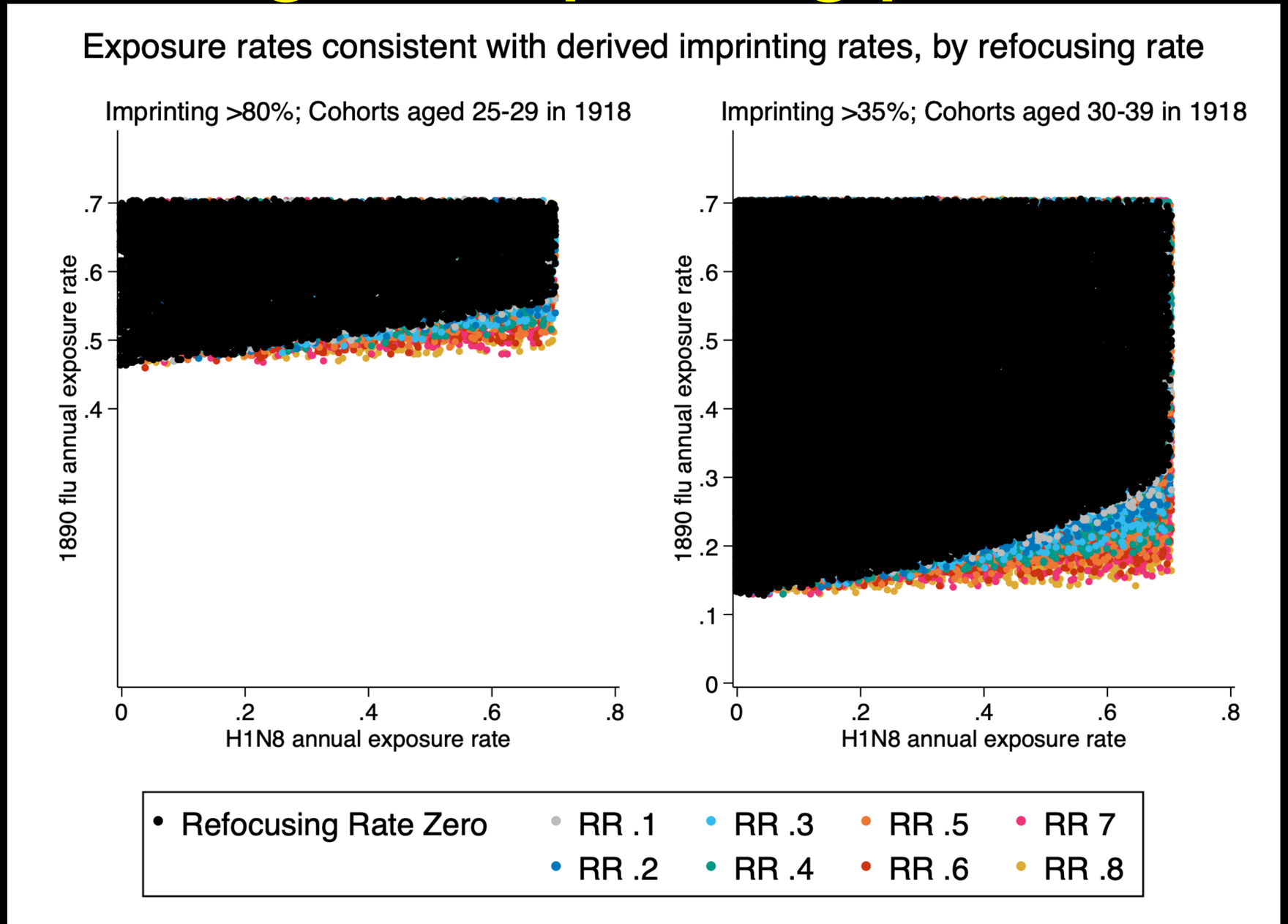

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