# Exploring the Racial Gap in Infant Mortality Rates, 1920-1970

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Abstract: White and nonwhite infant mortality rates declined sharply between 1920 and 1970, but the ratio of nonwhite/white rates did not decline. This paper examines the racial gap using state-level panel data with information on income, urbanization, women's education, and physicians per capita. We find that these variables can explain a large portion of the persistent racial gap in infant mortality rates between 1920 and 1945, but a smaller portion thereafter. We also find that even after controlling for these characteristics, southern states had relatively high infant mortality rates during this period, especially for nonwhites.

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## 1. Introduction

Great improvements in physical health are among the twentieth century's most impressive social achievements. In the United States, life expectancy at birth increased by more than 25 years; average height increased by about 7 centimeters (for white males); and a number of deadly diseases and debilitating illnesses were all but vanquished.[1] However, even as the overall level of health for both whites and nonwhites improved, racial gaps in health outcomes were, by some measures at least, remarkably persistent (Levine et al. 2001). At the center of this paper's analysis, the overall infant mortality rate (deaths of children under one year of age per 1,000 live births) fell from 85.8 in 1920 to 7.2 by 1997, but the racial gap in infant mortality rates, when expressed as a nonwhite/white ratio (reflecting the relative risk of death), did not decline over time. In fact, Figures 1 and 2 show that while the racial gap declined in some periods, it rose substantially in others.

Thus, we (and this paper) are of two minds: it is impossible to ignore the extraordinary gains in health enjoyed by both whites and nonwhites, but the persistent gap in the relative risk of death facing the two groups is also striking.[2] In this paper, we explore state-level infant mortality data in an empirical and historical framework that accommodates and sheds light on both trends. We focus on infant mortality because it is a simple, sensitive, and frequently referenced barometer of health conditions; because it conveys useful information about families' resources and environment, as well as about the effectiveness of public health policy; and because it is available

[1] The life expectancy figures are from Atack and Passell (1994, p. 230). The height estimates are derived from Costa and Steckel (1997, p. 51). The infant mortality figures are from the United States Statistical Abstract (1947, 2000).

[2] Though our primary interest is in "black" relative to "white" health, infant mortality data are reported by "white" and "nonwhite", and so our statistical analysis proceeds accordingly.

in a reasonably consistent form for whites and nonwhites over a relatively long period of time. Furthermore, though it is beyond the scope of this paper, we are interested in the connection between childhood health (for which local infant mortality might proxy) and subsequent educational and labor market outcomes. In future work, we hope to link our exploration of long-run racial gaps in health with the existing literature on long-run racial gaps in labor market outcomes.[3]

The literature on infant mortality is voluminous, but to our knowledge, previous works have not systematically examined the determinants of long-run trends in the racial gap over the 1920 to 1970 period (e.g., see Rochester 1923; Chase 1972; Shin 1975; Grossman and Jacobowitz 1981; David and Collins 1997; Almond, Chay, and Greenstone 2001). We are especially interested in this period because it was one of remarkable medical and social transformation. Professional training for doctors, nurses, and midwives was substantially upgraded and standardized; federal and state governments poured resources into improving and extending hospital and clinic systems, as well as into making health services available to the poor; and of course, medical science advanced rapidly. At the same time, prevailing patterns of racial discrimination embedded themselves in the healthcare system, perhaps compounding the disadvantages that African Americans already faced due to their geographic distribution and relative lack of financial and educational resources. Moreover, a number of historic events may have shaped the racial gap in health outcomes including the Great Migration, the Great

[3] For example, using British data for a cohort born in 1958, Currie and Hyson (1999) have found that low birthweight has an adverse long-run impact on educational attainment and labor market outcomes. See also Grossman (1975) and Edwards and Grossman (1979) on childhood health, cognitive development, and education. Such considerations have not been part of the literature on the long-run evolution of racial gaps in labor market outcomes in the U.S. (Smith and Welch 1989, Donohue and Heckman 1991).

Depression, World War II, and the Civil Rights Movement.

In section 2, we discuss the interaction of race, discrimination, health policy, and infant mortality in the context of a simple model of demand for and supply of broadly-defined healthcare. This simple model (and much of the existing empirical literature) suggests strong links between socioeconomic characteristics, healthcare, and infant mortality. To explore these links, we construct a panel of state-level data for whites and nonwhites for five-year intervals in section 3. In section 4, we outline our econometric approach to analyzing the dataset and report the results of that analysis. We find that differences in income, education, urban residence, and the supply of physicians can account for a large portion of the persistent racial gap in the infant mortality rate from 1920 to 1945, but they account for much less of the gap thereafter. We also find some substantial differences in the coefficient estimates across race categories, perhaps reflecting relatively strong urban disamenities and relatively poor educational quality for nonwhites. Even after controlling for observable state characteristics, southern states had relatively high infant mortality rates during this period, especially for African Americans. Finally, there is a strong secular decline in infant mortality for both races, but within this downward trend, there are a few striking periods in which the racial gap in infant mortality increases or decreases sharply. These sharp swings in the infant mortality gap are re-examined in section 5. Section 6 concludes and suggests several potential extensions for future research.

## 2. Infant Mortality and Race, 1920-1970

Our discussion is guided by a simple model of infant mortality in which the likelihood of survival is influenced by the consumption of quality-adjusted units of nutrition, housing, health-

related information, and health services. We refer to this bundle of goods as "healthcare" (broadly speaking), and think of it as an input into a health production function.[4] This production function may evolve as technology advances and may embody differences in "health productivity" across groups, perhaps associated with educational, environmental, or even cultural characteristics. We suppose that utility-maximizing parents allocate family resources over healthcare and other goods, subject to resource constraints and relative prices, and that they influence infant survival probabilities accordingly.

For the most part, it is not possible to observe healthcare consumption directly and consistently in this period. Consequently, the exact pathways through which healthcare influenced infant mortality will remain, in the econometric sense, unidentified.[5] But we can observe many of the factors that determine healthcare consumption, and therefore we can garner considerable insight into the ultimate determinants of the infant mortality gap.

Nonwhites' income and wealth were considerably less than those of whites throughout the period under study, implying tighter budget constraints and less demand for all normal goods, including infant-related healthcare. Additionally, we hypothesize that for various reasons, ranging from their geographic distribution to discriminatory practices, nonwhites might have faced higher costs for any given quantity of healthcare, further dampening their consumption of broadly-defined healthcare relative to that of whites.

By itself, the geographic distribution (and redistribution) of African Americans might have

[4] See Grossman (1972), Rosenzweig and Schultz (1983), Berger and Leigh (1988), Kenkel (1991), and Goldman and Lakdawalla (2001) on household production of health.

[5] For example, low income might lead to poor nutrition, low birthweight infants, and higher risk of infant mortality. Empirically, we can observe income and mortality, but until the 1950s we do not observe birthweight.

adversely affected their health relative to that of whites. In 1920, 85 percent of African Americans resided in the South (compared to 25 percent of whites), and in 1960, 60 percent still resided there (compared to 27 percent of whites). Throughout this period, southern per capita income was relatively low: in 1940, for example, southern real per capita income was about 64 percent of the national average (Mitchener and McLean 1999).[6] Such low average income levels might imply less private demand for healthcare, as in the simple framework outlined above, but might also imply tightly constrained public supplies of health-related services, including hospitals, subsidized care for the poor, and investments in sanitation and education.[7]

Furthermore, southern blacks were relatively concentrated in rural areas, especially early in the period under study. Therefore, they often lived far from hospitals and doctors and faced considerable costs (in terms of time and effort) when seeking professional medical services. On the other hand, although blacks in urban areas may have benefitted from proximity to medical facilities, patterns of residential segregation constrained the supply of housing to blacks, thereby raising its price and exacerbating crowded, unhealthy living conditions in the emerging ghettos.

To the extent that blacks' quality and quantity of education fell short of that provided whites, blacks might have found it more difficult (effectively more costly) to acquire information about medical advances and/or the availability of professional medical services.[8] Finally, in

[6] It is certainly possible that the relative health of southerners affected the region's relative productivity, as argued by Brinkley (1997) for the late 19th century. In this draft, however, we view income as exogenous variable.

[7] On average, poorer states had fewer hospital facilities per capita (Lave and Lave 1974). But even if southern income per capita had been as high as elsewhere, political economy considerations suggest that public spending on health initiatives might have remained low in the South in this period (see Alston and Ferrie 1999).

[8] In this paper, we view educational attainment as an exogenous variable. See Goldman and Lakdawalla (2001) for a recent discussion of the literature linking health and education. In an international context, several empirical studies of infant mortality and children's health emphasize

addition to having lower incomes and less education than whites and being geographically concentrated in poorly served areas, African Americans may have faced higher costs for healthcare services due to discriminatory practices. Discrimination in the healthcare system took many forms, including restricting blacks' access to healthcare facilities and limiting the number and quality of black doctors (Johnson 1949, Seham 1964, Beardsley 1987, Smith 1998).

The available evidence suggests that southerners, especially blacks, lagged well behind the rest of the country in the consumption of healthcare during much of the period under study. In 1945, only about 25 percent of nonwhite southern births and 68 percent of white births occurred in hospitals, compared to about 81 percent of nonwhite and 91 percent of white births outside the South.[9] In 1940, the southern level of physicians per capita was only about 60 percent of that in the rest of the country, almost surely implying less consumption of physician services. Also in 1940, only 35 percent of southern homes had flush toilets (an indicator of housing quality and sanitation), about half the level for the rest of the country.[10] And until federal funds became available, political and economic considerations severely limited the scope of southern public health efforts, particularly where blacks would have been the primary beneficiaries of such efforts (Beardsley 1987, pp. 128-185).[11]

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the importance of women's education. See Subbarao and Raney (1995).

[9] Washington D.C. is not included in the averages. 1945 is the first year for which we have such data by race and state, but Tandy reports that in 1935, only 17 percent of all black births occurred in hospitals compared to 40 percent of all white births (1937, p. 327).

[10] Physician data are from the occupational tables of the 1940 U.S. Census of Population. Toilet facilities data are from the 1940 U.S. Census of Housing, Volume II, Part 1, p. 79.

[11] Though wary of using expenditure figures to gauge the quantity of services provided, state government spending per capita on public health and hospitals was lower in the South than elsewhere (particularly the Northeast) throughout the period under study (see Financial Statistics of States and continuing publications).

Although blacks' geographic distribution and whites' discriminatory practices might have reduced the supply of healthcare to African Americans, philanthropic organizations and government agencies may have effectively increased the supply over time. In the first half of the century, the Rockefeller Sanitary Commission for the Eradication of Hookworm (1909-1915), the Rosenwald Fund, and the Duke Endowment all made efforts to improve children's health and education (Beardsley 1987, pp. 51, 114-127). Between 1926 and 1942, the Rosenwald Fund contributed to black hospitals in both the North and the South, funded public health training and research, contributed to thousands of black schools in the South, and was an early participant in the battle against tuberculosis and syphilis. The Duke Endowment, established in 1924, was instrumental in funding hospitals and healthcare for the poor in the Carolinas.

Perhaps more importantly, the federal government became increasingly engaged in the study, funding, and provision of healthcare for infants and mothers over the course of the century. Congress established the Children's Bureau in 1912 to "investigate and report . . . upon all matters pertaining to the welfare of children", with special attention to the issue of infant mortality (Bradbury 1956, p. 87). Under the auspices of the Children's Bureau, the Sheppard-Towner Act of 1921 provided matching grants to states for educational materials and instruction on maternal and infant nutrition, care, and hygiene. By the time the Sheppard-Towner funding expired in 1929, the Children's Bureau had sponsored nearly 3,000 centers for prenatal care and more than 3 million home visits by nurses (Lemons 1969; U.S. Dept. of Labor 1931). Although it was discontinued, Bradbury argues that Sheppard-Towner laid a foundation for subsequent federal health programs (1974, p. 26).

The Social Security Act of 1935 featured a federal-state program for maternal and child

health that, in terms of both spending and scope of activity, eclipsed Sheppard-Towner. Furthermore, from 1943 to 1949, the Children's Bureau administered the Emergency Maternal and Infant Care (EMIC) program which promised free medical care to the pregnant wives and newborn children of relatively low-ranking servicemen. EMIC handled approximately 1.5 million cases (Bradbury 1956, p. 60), and in some places (particularly the South) the program directly elevated the standard of obstetric care through its process of review and approval of medical facilities (Beardsley 1987, p. 175-176). An indirect and long-standing benefit of the EMIC program was that it cast light on the general inadequacy of the nation's hospital system. In response, Congress launched the colossal Hill-Burton program (1946-1971) to inventory and modernize existing hospitals and to fund new construction in areas with relatively few hospital beds per capita (Lave and Lave 1974).

Over the same period, large numbers of southerners, especially blacks, moved to other parts of the country (Collins 1997). These migrants and their children might have benefitted from the non-South's relatively abundant supply of healthcare services, relatively egalitarian education system, and relatively high levels of pay. But some aspects of the relocation might have offset such benefits. First, adjusting to the change in disease environment might have been difficult, particularly for those moving from rural to urban areas and/or from warm- to cold-weather climates. Second, although "chain migration" patterns often ensured some support from extended family networks, striking out on one's own might have entailed substantial economic and health risks. Third, the period under study coincides with a rising level of racial residential segregation in American cities (Cutler, Glaeser, and Vigdor 1999), and the emerging African-American ghettos were often crowded, unclean, and unhealthy. Fourth, the industrial employment

opportunities open to blacks, especially early in the period under study, were sometimes physically debilitating (see Maloney and Whatley 1995).

## 3. Data

We use variation across states and over time to measure the impact of several factors on white and nonwhite infant mortality rates. In the absence of family- or child-level data, we use state-level averages for whites and nonwhites at five-year intervals from 1920 to 1970. At each point in time, our dataset includes (by race and state) the infant mortality rate, estimates of real per capita income, the proportion of the population residing in urban areas, average years of education for women between 20 and 40 years of age, and physicians per capita. These variables reflect the financial, environmental, and medical resources available to families having children. The variables that intermediate the connection between these resources and infant mortality (such as birthweight) will be discussed when and where evidence is available, but they do not enter the econometric explorations directly.

The infant mortality rate is calculated as the number of deaths of children under one year of age per 1,000 live births in the relevant calendar year; thus, it requires both death and birth information. In the United States, this information was not collected systematically until the twentieth century, though in response to public health crises, some cities and states began tracking mortality earlier (Haines 2001). A national death-registration area, covering just ten states and some cities in other states, was established in 1900. A national birth-registration area was established in 1915, also with geographically limited coverage. Over time, states were added to the official registration areas as they passed laws requiring the registration of deaths and births,

and as they demonstrated to the Census Bureau that they met minimum requirements for completeness of coverage (90 percent).

By 1933, all states were covered, but there is reason to believe that the extent of under-reporting was sometimes significant (Linder and Grove 1943; Demeny and Gingrich 1967). Using 1940 census data to verify the birth-registration data, the Census Bureau found that about 93 percent of all births were indeed registered, but in some states the proportion had fallen well below 90 percent (as low as 76 percent in Arkansas). If infant deaths and births were under-reported by the same proportion, then the infant mortality measures would be unbiased; otherwise, the rate would be mismeasured. If relatively poor states had relatively weak registration systems, and if infant mortality rates were systematically underestimated for those states, we would observe a spurious positive correlation between average income and infant mortality. For the most part, we must take the infant mortality data at face value, but we will discuss some specifications that incorporate the information from the Census Bureau's 1940 accuracy check.

A household's ability to pay for food, housing, and medical services depends in large part on its income and wealth.[12] Because the Census first inquired about income in 1940, compiling any such measure by race and state for the full 1920 to 1970 period is difficult. We have constructed measures of per capita income (by race, state, and year: $Y_{W, it}$ and $Y_{NW, it}$) which ultimately depend on estimates of a few variables in the following two equations. The first is simply a weighted average of white and nonwhite income per capita.

[12] See Steckel (1988) and Ferrie (2001) for micro-level studies of wealth and mortality in the 19th century. See Rochester (1923) for a detailed study of households in Baltimore in 1915. We cannot observe wealth during this paper's period of study.

(1) $$Y_{it} = \theta_{it} Y_{W,it} + (1 - \theta_{it}) Y_{NW,it}$$

(2) $$Z_{it} = Y_{W,it} / Y_{NW,it}$$

$Y_{it}$ is nominal income per capita in state i at time t, as reported by the Bureau of Labor Statistics (after 1929) and by Mitchener and McLean (1999) in 1920. $\theta$ is the proportion of the population that is white. Z is the ratio of white income per capita ($Y_{W,it}$) to nonwhite income per capita ($Y_{NW,it}$). We estimate Z for each state and census year using the detailed occupational information for individuals reported in the Integrated Public Use Microdata Series (IPUMS, Ruggles and Sobek 1997).[13]

Given measures of $Y_{it}$ and $Z_{it}$, and using the number of IPUMS observations for each race category to measure $\theta$, we have enough information to calculate $Y_{W,it}$ and $Y_{NW,it}$. Essentially, we are simply using Z and $\theta$ to figure out how to split the state's income between whites and nonwhites. Although estimates of $Y_{it}$ are available in every year, Z and $\theta$ can only be estimated directly for census years, and we interpolate between those years to get mid-decade figures. These parameters appear to change relatively slowly over time, so their interpolation seems reasonable. This procedure yields estimates of nominal income per capita by race, state, and year. To get real income estimates, we adjust for price level changes over time using national consumer price indices (U.S. Department of Commerce 1975).

[13] We assign every person in the microdata samples an income proxy based on his or her observed characteristics. This "income proxy" is the median value of total annual income earned by workers of that sex, of that race, in that region of the country, in that three-digit occupation category in 1960. We cannot use the 1940 Census because only wage and salary income is reported in that year. We cannot use the 1950 Census because only "sample line" observations have total income reported, and there are not enough observations for meaningful sex/race/region/occupation estimates. In spirit, combining one year's income distribution across occupations with another year's occupational distribution to estimate the racial income gap is similar to the exercise undertaken in Smith (1984).

Education may affect infant mortality rates both indirectly, through its effect on earnings, and directly, through its effects on health-related knowledge and responsiveness to that knowledge.[14] Therefore, even though education and income tend to be highly correlated, we include measures of both in the regressions, focusing on the educational attainment of women between 20 and 40 years of age. Again, estimating race- and state-specific education levels is a challenge because the Census did not inquire about years of education prior to 1940. Instead, the Census reports literacy rates, but there is no simple way to move from literacy rates to average years of education or vice versa.

We estimated years of education for the years prior to 1940 by using the individual-level information contained in the 1940 IPUMS sample (Ruggles and Sobek 1997). For example, to estimate years of education for women who were 20 to 40 years old in 1930 for each state, we calculated the average level of education for women who were 30 to 50 in 1940 for each state. This approach clearly has defects: first, it does not account for migration (or death); second, it does not account for upward biases in self-reported education as age rises. But it does provide a much richer measure of educational attainment than the literacy measure, and on the basis of previous studies of women's education and infant health, it can certainly be argued that focusing on young women is a useful sample restriction.[15]

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[14] See Berger and Leigh (1989) or Kenkel (1991) for discussions of the empirical connection between health and schooling in modern data, including potential omitted variable issues. Even after adjusting for selection on the basis of unobservable characteristics (e.g., rate of time preference or ability), Berger and Leigh find that education has a significant direct effect on health. Kenkel finds that even after controlling for health knowledge, education appears to have a strong positive effect on health-related behavior.

[15] At this point, we have not attempted to adjust the data for differences in the quality of education across races or states. Margo (1986) discusses the pitfalls of comparing "years of education" across race groups and over time at length. He argues that blacks educated in the late 19th century had substantially less schooling relative to whites than suggested by the 1940 census

Prior to 1920, it is clear that urban residence was relatively hazardous for infants' health. Haines notes that "A variety of circumstances contributed to the excess mortality of cities: greater density and crowding, leading to the more rapid spread of infection; a higher degree of contaminated water and food; garbage and carrion in streets and elsewhere not properly disposed of; larger inflows of foreign migrants, both new foci of infection and new victims; and also migrants from the countryside who had not been exposed to the harsher urban disease environment" (2001, p. 3). The observed gap between urban and rural mortality rates dissolved over the course of the late nineteenth and early twentieth century as public health initiatives took root, as medical science progressed, as water and food supplies improved, and as urban hospitals increased the volume and quality of available healthcare.[16]

In the regression framework outlined below, we enter Census measures of the urban proportion of the population by race and state, with an adjustment to the data in 1960 to reflect a non-negligible change in the Census definition of "urban".[17] Interpretation of the urban effect is complicated by the fact that price level adjustments to the nominal income measures do not distinguish between more and less urban states. If prices are higher in urban areas, then real income might be systematically overestimated for more urban states relative to less urban states. Thus, the urban variable may capture not only health disamenities associated with urban life, but

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data.

[16] See Troesken (1999 and 2001) on typhoid, water, and sewerage in U.S. cities in the late 19th and early 20th centuries.

[17] The change was introduced in 1950, and in that year figures for both "new" and "old" definitions of "urban" are reported. The 1960 urban figures are scaled according to the ratio between the "new" and "old" measures for each state and race in 1950. The figures from 1930 to 1950 come from the 1950 state-level census volumes. The figures for 1920 comes from the 1940 state-level census volumes. For the sake of consistency, we made an effort to avoid using the 1930 volumes because in them, Mexicans are counted as nonwhite, but in other years they are counted as white.

also the relatively high cost of living in urban areas.

The Census occupation tables report the number of physicians in each state, by race.[18] *Ceteris paribus*, we expect a larger number of physicians per capita to lower the effective cost of medical care, and to therefore lower the rate of infant mortality.[19] A higher proportion of doctors in the population could also increase general awareness of public health issues (and remedies), although it is interesting to note that physician groups (e.g., the American Medical Association) often opposed government health initiatives that, in their view, infringed on physicians' private practice (including Sheppard-Towner). Also, to the extent that doctors are attracted to areas with plentiful and state-of-the-art medical facilities, the physicians per capita variable will reflect the supply of medical facilities (Lave and Lave 1974, p. 2). Of course, there is no "quality-of-training" or "vintage-of-knowledge" adjustment built into this measure of health service supply, and so we must rely on time-period dummy variables to capture such trends.

Table 1 reports summary statistics for our basic data set of 281 state-year observations for each race category (weighted by population). For the sake of comparability, the samples consist of the same collection of state-year cells for both whites and nonwhites. Some states had very few nonwhite residents and could not be included in the nonwhite sample, and we have trimmed states from the white sample to match. State-year observations are weighted by the relevant racial group's population, so that each observation is weighted by (approximately) the number of

[18] The census also reports the number of nurses and medical technicians, but those occupational categories are more difficult to compile in a consistent manner.

[19] We recognize that the number of physicians could respond endogeneously to health conditions. Suppose for example that states with poor health outcomes make an effort to train or attract additional physicians. This could lead to a spurious positive correlation between infant mortality and physicians per capita.

births that it represents.[20] Not surprisingly, on average, nonwhites in this sample had lower incomes, less education, and higher infant mortality rates than whites. Though initially less urban than whites, nonwhites urbanized quickly during this period, and by 1970 the proportion of nonwhites in urban areas exceeded the proportion of whites. The physicians-per-capita variable is identical within state-year cells for nonwhites and whites, and so the relatively low average value for nonwhites is driven primarily by differences in the groups' distributions across states.

## 4. Accounting for the Racial Gap in Infant Mortality

Our basic regression equation is expressed in log-log form, implicitly assuming constant elasticities between the dependent and independent variables:

$$\ln IMR_{it} = (\ln X_{it})\beta + \gamma_t + e_{it} \quad (3)$$

where i indexes states, t indexes time periods, and X is a set of race-state-year characteristics. We run the regressions separately for whites and nonwhites, thereby allowing the coefficients to differ between race categories.[21] We add the time-period dummy variables ($\gamma$) to absorb unobserved period-specific factors influencing infant mortality (such as advancing medical technology), and in some specifications we add region or state dummy variables to absorb area-specific effects.

[20] Results are similar when regressions weight each period equally and then partition the weight within periods based on population, as are results from regressions that weight each period by the number of observations it contributes to the regression and then partition weight within the period by population.

[21] Two common econometric issues deserve mention. First, there may be measurement error in the independent variables, particularly for income and education. Even if measurement error in a particular variable is random, it implies some degree of attenuation bias (towards zero) to that variable's coefficient and an unknown direction of bias to the other coefficients (Greene 1993, pp. 279-284). Second, because health and economic conditions are interrelated in complex ways, one could argue plausibly that the regressions' explanatory variables are endogenous. In theory, an instrumental variable approach could help circumvent these concerns.

Pritchett and Summers (1997) chose a similar functional form in their analysis of an international panel of infant mortality rates, as did Flegg (1982), but there does not appear to be a strong consensus in the literature on the most appropriate functional form.[22]

Table 2 reports regression results from three different specifications. Columns 1 and 2 correspond to the basic specification described by equation 3. Columns 3 and 4 add state dummy variables to the specification, and columns 5 and 6 include a dummy variable for the southern region (rather than a full set of state dummies). In general, the coefficients reported in Table 2 have the expected signs: *ceteris paribus*, higher levels of income, women's education, and physicians per capita tend to lower infant mortality rates, whereas higher levels of urbanization are correlated with higher infant mortality rates. Though the time-period dummy variables clearly absorb a great deal of the variation, the X variables' coefficients (income, education, urban, and physician supply) in columns 1, 2, 5, and 6 often exceed the 10 percent level of statistical significance. When we forfeit a great deal of the cross-state variation by including state-fixed effects (columns 3 and 4), the standard errors of the coefficient estimates increase, and some of the coefficients change noticeably in magnitude, particularly for whites.

Comparing the coefficients across racial groups (columns 1 and 2), it appears that urban residence was more detrimental for nonwhites than for whites, and that education was less beneficial for nonwhites than for whites. These gaps persist, though their magnitudes change, in columns 3, 4, 5, and 6, when state or region dummies are included in the regressions. The comparatively large urban coefficient for nonwhites in Table 2 might reflect the poor living conditions common to many nonwhite urban neighborhoods. The comparatively low returns (in

[22] In this case, $P_E$ tests of the linear versus log-linear form favor the log-linear approach (Greene 1993, pp. 321-322). Additionally, link tests, essentially regressions of the dependent variable on fitted values and fitted values squared, reveal no evidence of misspecification.

terms of mortality) to years of education for nonwhites might reflect the relatively low quality of education received by nonwhites (see Margo 1986, 1990).

Columns 5 and 6 include a southern state dummy variable to provide a sense of how different, on average, infant mortality was in the South compared to elsewhere, when accounting for state-level differences in income, education, urbanization, and physicians per capita. In both columns, the coefficient on the southern dummy variable is positive, implying that the relatively poor health outcomes in the region were not entirely due to differences in our set of observable state characteristics. The southern white infant mortality rate is only about 5 percent higher than elsewhere (*ceteris paribus*), but the nonwhite rate is about 16 percent higher.

There is evidence that southern states had less complete registration statistics than others, at least in the pre-war period (Linder and Grove 1943). If this translated into a systematic under-reporting of infant mortality, then the "South" coefficients may understate the true degree of southern mortality disadvantage. We tested this hypothesis by adding the estimated proportion of all births registered in 1940 (from Linder and Grove) to the regressions specified in columns 5 and 6 (results not shown). In the white sample, the south coefficient increased to about 8 percent; in the nonwhite sample, the south coefficient increased to 22 percent. The unobserved heterogeneity between regions that generates the positive "South" coefficient could be driven by any number of factors, including the extent of state and local public health efforts, the climate and disease environment, or even the quality of physicians, education, or urban sanitation.

Even with controls for income, education, urban residence, and supply of physicians, a very strong secular trend in infant mortality is manifested in the downward march of the coefficients on the time-period dummies. Two aspects of the time period coefficients are

especially interesting. First, even as the Hill-Burton program pumped funds into the system, the secular decline in infant mortality stagnates for nonwhites from 1950 to 1965 and for whites between 1955 and 1965. This suggests that the mid-century plateau evident in Figure 1 is not due to adverse movements in the independent variables offsetting an underlying downward trend. Second, during some intervals, the nonwhite time coefficient fell by substantially more than the white coefficient, and during others the white time coefficient fell by substantially more than the nonwhite one. We discuss these uneven movements at some length in the next section.

To what extent can racial gaps in the independent variables account for the persistent gap in infant mortality? Figure 3 plots a counterfactual white infant mortality rate which, at each point in time, is the product of the regression coefficients from column 1 and the average *nonwhite* characteristics at that time. That is, the figure plots the predicted level of white infant mortality supposing that the white population had had the observed characteristics of the nonwhite population. For comparison, we also plot the actual white and actual nonwhite levels of infant mortality. From this perspective, the *entire* racial gap in infant mortality can be accounted for by differences in characteristics up to 1945, but by 1970, only about a third of the gap can be explained. As is common in such exercises, using the nonwhite coefficients in combination with the white characteristics delivers somewhat different results, but the main point still stands: up to 1945, a large portion of the infant mortality gap can be accounted for by differences in population characteristics, but after 1945, the unexplained portion of the gap widens considerably.

Table 3 decomposes the infant mortality gap, variable by variable, for each year using the white coefficients. Through most of the period under study, the largest contributor to the racial gap in infant mortality is the gap in education levels of women aged 20 to 40. Up to 1935, the

difference in years of education among whites and blacks accounts for more than 70 percent of the observed infant mortality gap. But the racial gap in years of education narrowed quickly over time, and by 1970, differences in years of education can account for only about 14 percent of the infant mortality gap. Up to 1945, the racial gap in income accounts for about 26-30 percent of the overall infant mortality gap, but by 1970 income accounts for only 16 percent of the gap. All else equal, whites' greater likelihood of urban residence tended to narrow the infant mortality gap early in the period under study, but the nonwhite population urbanized relatively quickly and surpassed the white urban proportion by 1960. Finally, the geographic distribution of physicians accounts for 7 percent of the infant mortality gap early in the period under study, but with the redistribution of nonwhites (and to some extent, of physicians), the contribution diminished over time.

## 5. Explaining the "Unexplained" Changes in the Racial IMR Gap

The time series graph of the racial gap in infant mortality rates (Figure 2) features two periods of abrupt decline (1941-1946 and 1966-1971) and one period of rapid widening (1948-1958). The two episodes of decline coincide almost exactly with the only two periods of substantial decline in the racial wage gap (for men) since 1920 (Donohue and Heckman 1991), and in fact, Table 3 suggests that income and educational convergence helped drive much of the convergence in infant mortality during the 1940s. To a large extent, however, the abrupt changes in the infant mortality gap remain econometrically unexplained and warrant additional examination and speculation.

This requires a different empirical approach, one that cannot by itself identify the forces

underlying the abrupt movements in the infant mortality gap, but which certainly does suggest where to look for explanations. Figure 4 reports separate series for neonatal (under 28 days old) and postneonatal infant mortality gaps, a useful distinction because the ultimate causes of death differ substantially for the two groups (premature delivery and low birthweight dominate the neonatal deaths; disease and environmental factors are more prevalent in the postneonatal deaths). For ease of interpretation, we have kept the components in their non-log form in Figure 4 . Table 4 takes an additional step by breaking up the neonatal and postneonatal components into southern and non-southern regions for the 1940 to 1970 period. The table also reports weights reflecting the proportion of white and nonwhite births in both regional categories, an important detail because of the dissimilar geographic distributions of nonwhites and whites.

Clearly, the 1941-1946 period was characterized by rapidly declining levels of infant mortality for both race categories (figure 1) *and* by a declining racial gap (figure 2). Figure 4 shows that the racial gap declined especially sharply in the postneonatal category. The shrinking gap in postneonatal mortality accounts for most, but not all, of the overall racial convergence in infant mortality rates: the postneonatal gap fell from 21 to 9 while the neonatal gap fell from 13 to 8. Comparing 1940 with 1945 in Table 4, it is apparent that the strong declines in southern nonwhite neonatal and postneonatal mortality dominate all other movements during the period.

The development of penicillin was perhaps the single greatest medical advance of the 1940s, and it had a substantial impact on the level of infant mortality, especially for postneonatal infants (older than 28 days old) (CDC 1999). Figure 4 might appear to suggest that nonwhites benefitted disproportionately from new antibiotics, but the timing and magnitude of the racial gap's decline implies that penicillin cannot be the whole story, or perhaps even a large part of it.

In particular, the racial gap fell mostly *before* the mid-1940s, that is, before antibiotics became widely available for civilian use.

There are two more likely contributors to the gap's decline in the 1940s. First, as mentioned already and reflected in Table 3, there was substantial improvement in nonwhites' absolute and relative economic and educational status. Second, and more speculatively, given the concentration of African Americans in the South, the federal government's expanded military and administrative presence in that region might have disproportionately benefitted nonwhites. During the war, government efforts virtually eliminated malaria, aggressively targeted venereal disease and tuberculosis, and, as described above, supported the EMIC program for the wives and children of low-ranking servicemen, a program with (potentially) substantial positive spillovers to the general quality of infant care (Beardsley 1987, pp. 173-175).

Unfortunately, the downward drop in the racial gap in infant mortality reversed itself completely between 1948 and 1958. While white infant mortality continued to fall for both neonatal and postneonatal infants, albeit slowly compared to the 1940s, the nonwhite rates were nearly constant. Table 4 reports that in the South, where the majority of nonwhite births occurred, the infant mortality rate increased slightly between 1950 and 1960. At the same time, the southern white rate declined by 6.1 per 1,000.

The widening infant mortality gap may be explained in part by widening racial differences in birthweight. Birthweight statistics were reported for the first time during the 1950s, and Chase (1972) noted an increase in the proportion of low birthweight neonates (under 2,500 grams) among nonwhites from 9.7 percent of births in 1950 to 12.9 percent in 1960. Among whites the low birthweight proportion fell from 7.0 to 6.8 percent. Though these changes in the weight

distributions may appear small, they might have had important consequences for the racial gap in infant mortality because the neonatal mortality rate of low birthweight infants was very high: around 160 per thousand births during the 1950s (for nonwhites) compared to about 10 per thousand for neonates weighing more than 2,500 grams (Chase 1972, p. 9).[23]

The rising proportion of low birthweight infants among nonwhites might have been a proximate cause of the widening infant mortality gap in the 1950s, but the ultimate causes remain unidentified. Chase and Byrnes (1972) were not able to explain the rising proportion of nonwhite low-birthweight infants by changes in the distributions of age of mother, plurality, hospital delivery, measurement error, and reported gestation. Furthermore, the postneonatal gap, which is generally regarded as being fairly insensitive to birthweight, also increased, and so it seems that even in the proximate sense, there must be more to the story than adverse changes in the distribution of nonwhites' birthweight. Finally, the relatively strong divergence of white and nonwhite infant mortality rates in the South recommends a regionally focused investigation.

Two hypotheses related to unobserved environmental quality come to mind. First, Chay and Greenstone (1999, 2001) have argued that environmental conditions, in particular air pollution, might be causally linked to birthweight and infant mortality. If so, then the massive redistribution of blacks out of the rural South might have had adverse health consequences which tended to offset health improvements associated with more accessible hospital care and higher incomes. The "urban" variable used in the econometric exploration should have captured some of this effect, but "urban" is a coarse characterization of environmental quality. Second, the rapid

[23] A rough calculation suggests that, *ceteris paribus*, a shift of 3.2 percent of births into the low birthweight category, given the difference in mortality rates between the weight categories, could raise the nonwhite neonatal mortality rate by 4.8 deaths per thousand births [0.032(160 - 10) = 4.8]. We view this as an upper bound. Unfortunately, when we inquired, the National Center for Health Statistics reported that the data underlying the Chase study cannot be found.

suburbanization of white families might have improved the environmental quality enjoyed by "urban" whites relative to "urban" nonwhites who were generally excluded from new suburban developments (Collins and Margo 2001). Both of these hypotheses extend beyond the bounds of this paper, but may prove worthy of future research.

Two other hypotheses suggest differential racial trends in quality of care or in prenatal behavior (e.g., smoking). For example, perhaps southern whites benefitted disproportionately from Hill-Burton funding of hospital expansion; or perhaps the rapid urbanization of nonwhites was accompanied by the emergence of "bad" ghettoes which adversely influenced a host of nonwhite socioeconomic outcomes (see Cutler and Glaeser 1997; Collins and Margo 2000). Again, these hypotheses require substantial additional research.[24]

The racial gap took a temporary plunge between 1966 and 1971. The decline was somewhat stronger for postneonates (from 9.0 to 4.8) than for neonates (from 9.2 to 6.6). Based on a detailed analysis of Mississippi, Almond, Chay and Greenstone (2001) argue that the desegregation of southern hospitals and the consequent improvement in rural blacks' access medical care played an important role in the gap's decline. The process of desegregation appears to have been driven largely by a combination of federal Civil Rights legislation and Medicare certification requirements. Compression of the infant mortality gap after 1965, however, was not confined to the rural South, suggesting that while hospital desegregation may explain a substantial part of the decline, it is probably not a complete explanation.

[24] There is a large medical literature on genetic versus behavioral/environmental/socioeconomic origins of the disparity in the distributions of black and white birthweights. See David and Collins (1997).

## 6. Conclusion

Health and economic status are interconnected not only contemporaneously, but also intertemporally through families, communities, and institutions. History, therefore, contains valuable information for understanding intergroup health disparities and for evaluating and formulating policy responses to those disparities. In this case, we focus on the racial gap in infant mortality rates between 1920 and 1970, a period which witnessed the expansion of the healthcare system, the Great Migration of African Americans, the emergence of the Civil Rights Movement, and curiously enough, a persistently large gap in the relative risk of death between nonwhite and white infants.

Clearly, the rapid descent in infant mortality rates during this period benefitted both whites and nonwhites. At each point in time, however, nonwhites were disadvantaged in terms of income, education, and location relative to whites. Using a panel of state-level race-specific data, we found that a large portion of the racial gap in infant mortality rates can be accounted for by differences in those characteristics, especially between 1920 and 1945. But between 1945 and 1970, group differences in observable characteristics lost much of their explanatory power – that is, characteristics converged, but for some reason infant mortality rates did not.

We intend to improve and extend this work by incorporating more information on the mediating factors that link infant mortality and the population characteristics observed here, by incorporating data (and histories) for smaller geographic units, and by developing more explicit empirical connections between public health efforts and subsequent health outcomes.
We would also like to make a connection to the post-1970 period, though doing so may be complicated by the introduction of Medicaid, the legalization of abortion, and the proliferation of

very effective birth control methods (Grossman and Jacobowitz).

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Table 1: Summary Statistics, 1920-1970

| | White | Nonwhite |
|---|---|---|
| Panel A: Summary statistics of log state-year values (entered in regressions) | | |
| Ln Infant Mortality | 3.4462 | 3.9358 |
| | (0.4768) | (0.4212) |
| Ln Income | 7.5917 | 6.8052 |
| | (0.4357) | (0.6704) |
| Ln Education | 2.3419 | 2.1009 |
| | (0.1174) | (0.2625) |
| Ln Urban | 4.0954 | 3.9879 |
| | (0.3207) | (0.5245) |
| Ln Physicians | -6.6525 | -6.8232 |
| | (0.2653) | (0.3125) |
| N | 281 | 281 |
| Panel B: Summary statistics of state-year values | | |
| Infant Mortality | 35.43 | 56.32 |
| | (18.88) | (27.12) |
| Income | 2159 | 1104 |
| | (837.2) | (659.8) |
| Education | 10.47 | 8.44 |
| | (1.183) | (2.045) |
| Urban | 62.81 | 60.75 |
| | (16.67) | (26.32) |
| Physicians | 0.0013 | 0.0011 |
| | (0.0003) | (0.0004) |
| South | 0.3071 | 0.6401 |
| | (0.4621) | (0.4808) |
| N | 281 | 281 |

Notes: Each observation represents a particular race/state/year cell. Observations are weighted by population. Standard deviations are in parentheses. The regressions in the next table are run using log values of variables. Figures in Panel B are reported for ease of interpretation. Note that the log figures in Panel B will not equal the figures in Panel A (because the log of an average values is not equal to the average of log values).

Sources: See the text for description of the dataset.

Table 2: Infant Mortality Regressions, 1920-1970

| | White | Nonwhite | White | Nonwhite | White | Nonwhite |
|---|---|---|---|---|---|---|
| Income | -0.1407 | -0.1397 | 0.0014 | -0.1742 | -0.1356 | 0.0628 |
| | (0.0886) | (0.1434) | (0.1394) | (0.1770) | (0.0317) | (0.1766) |
| Education | -0.8416 | -0.4254 | -0.3746 | -0.3654 | -0.6770 | -0.4318 |
| | (0.2829) | (0.2203) | (0.3700) | (0.2612) | (0.2857) | (0.2198) |
| Urban | 0.0969 | 0.3073 | -0.0855 | 0.5682 | 0.1280 | 0.2573 |
| | (0.0780) | (0.1423) | (0.1884) | (0.2362) | (0.0711) | (0.1379) |
| Physicians | -0.1376 | -0.2057 | -0.3156 | -0.1666 | -0.1221 | -0.2350 |
| | (0.0455) | (0.1098) | (0.0945) | (0.2147) | (0.0402) | (0.1081) |
| South | ----- | ----- | ----- | ----- | 0.0539 | 0.1573 |
| | | | | | (0.0317) | (0.0629) |
| 1925 | -0.1438 | -0.1818 | -0.1671 | -0.1676 | -0.1530 | -0.1762 |
| | (0.0211) | (0.0599) | (0.0284) | (0.0614) | (0.0208) | (0.0601) |
| 1930 | -0.2767 | -0.3365 | -0.3231 | -0.2995 | -0.2947 | -0.3078 |
| | (0.0328) | (0.0778) | (0.0413) | (0.0928) | (0.0320) | (0.0795) |
| 1935 | -0.4058 | -0.5324 | -0.4728 | -0.5148 | -0.4328 | -0.4953 |
| | (0.0525) | (0.0912) | (0.0566) | (0.1151) | (0.0466) | (0.0922) |
| 1940 | -0.5226 | -0.6201 | -0.6407 | -0.6139 | -0.5582 | -0.6174 |
| | (0.0811) | (0.0788) | (0.0809) | (0.1033) | (0.0710) | (0.0762) |
| 1945 | -0.6252 | -0.7933 | -0.8131 | -0.7999 | -0.6701 | -0.8819 |
| | (0.1104) | (0.0918) | (0.1201) | (0.0779) | (0.0947) | (0.0970) |
| 1950 | -0.8873 | -1.0290 | -1.0825 | -1.0612 | -0.9390 | -1.1006 |
| | (0.1152) | (0.0860) | (0.1213) | (0.0918) | (0.0984) | (0.0837) |
| 1955 | -0.9637 | -1.0199 | -1.1833 | -1.0695 | -1.0220 | -1.1122 |
| | (0.1175) | (0.0869) | (0.1357) | (0.0971) | (0.0997) | (0.0867) |
| 1960 | -0.9622 | -0.9681 | -1.1960 | -1.0335 | -1.0266 | -1.0668 |
| | (0.1256) | (0.0930) | (0.1470) | (0.1089) | (0.1089) | (0.0921) |
| 1965 | -0.9640 | -0.9749 | -1.2266 | -1.0470 | -1.0361 | -1.1082 |
| | (0.1443) | (0.1058) | (0.1722) | (0.1202) | (0.1258) | (0.1132) |
| 1970 | -1.1028 | -1.1827 | -1.3968 | -1.2588 | -1.1824 | -1.3537 |
| | (0.1728) | (0.1209) | (0.2000) | (0.1323) | (0.1506) | (0.1356) |
| Constant | 5.8960 | 3.9867 | 3.4625 | 3.3551 | 5.4800 | 2.5960 |
| | (0.9152) | (1.1183) | (1.1628) | (1.3395) | (0.8792) | (1.3664) |
| State Dummies | No | No | Yes | Yes | No | No |
| $R^2$ | 0.97 | 0.90 | 0.98 | 0.95 | 0.98 | 0.91 |
| N | 281 | 281 | 281 | 281 | 281 | 281 |
| Mean Dep. Var. | 3.446 | 3.936 | 3.446 | 3.936 | 3.446 | 3.936 |

Notes: Observations are weighted by population. Robust standard errors with adjustments for state clustering are in parentheses.

Sources: See the text for description of the dataset.

Table 3: Accounting for the Racial IMR Gap, 1920-1970

| | 1920 | 1925 | 1930 | 1935 | 1940 | 1945 | 1950 | 1955 | 1960 | 1965 | 1970 |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Total IMR Gap | 0.462 | 0.452 | 0.499 | 0.458 | 0.525 | 0.460 | 0.504 | 0.586 | 0.636 | 0.630 | 0.562 |
| Gap "Explained" by: | | | | | | | | | | | |
| Income | 0.120 [0.261] | 0.119 [0.264] | 0.147 [0.295] | 0.139 [0.303] | 0.141 [0.269] | 0.122 [0.265] | 0.119 [0.236] | 0.112 [0.192] | 0.109 [0.171] | 0.100 [0.158] | 0.092 [0.164] |
| Education | 0.374 [0.808] | 0.324 [0.718] | 0.375 [0.751] | 0.347 [0.756] | 0.328 [0.624] | 0.282 [0.614] | 0.243 [0.483] | 0.184 [0.314] | 0.136 [0.214] | 0.103 [0.163] | 0.076 [0.136] |
| Urban | -0.056 [-0.122] | -0.045 [-0.101] | -0.044 [-0.088] | -0.035 [-0.076] | -0.029 [-0.054] | -0.018 [-0.038] | -0.009 [-0.018] | -0.002 [-0.004] | 0.003 [0.004] | 0.008 [0.013] | 0.012 [0.022] |
| Physicians | 0.036 [0.078] | 0.028 [0.063] | 0.032 [0.063] | 0.034 [0.074] | 0.038 [0.072] | 0.032 [0.070] | 0.029 [0.057] | 0.022 [0.038] | 0.018 [0.028] | 0.013 [0.020] | 0.009 [0.016] |
| Total "Explained" Gap | 0.474 | 0.426 | 0.510 | 0.484 | 0.479 | 0.419 | 0.382 | 0.316 | 0.266 | 0.224 | 0.189 |

Notes: The "Total IMR Gap" is the difference between the average (weighted by population) log white and nonwhite infant mortality rates in each year. Each component of the "Gap Explained By" section is the product of the relevant coefficient from Table 2, column 1 and the difference in the variable's average value for whites and nonwhites (in that year). The percentage of the year's total gap accounted for by each component is listed in square brackets.

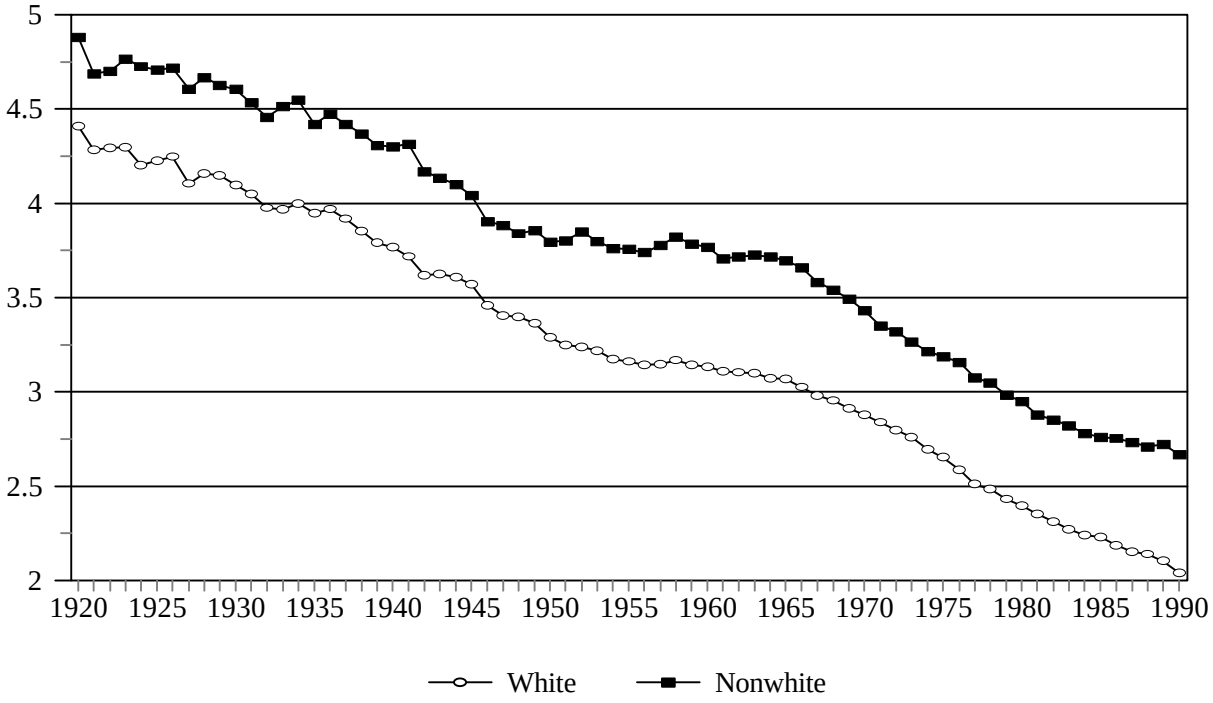
Sources: See the text for description of the dataset.

Table 4: Neonatal and Postneonatal Mortality, 1940-1970

| | 1940 | 1945 | 1950 | 1955 | 1960 | 1965 | 1970 |
|---|---|---|---|---|---|---|---|
| White, South | | | | | | | |
| Neonatal | 30.1 | 24.7 | 20.8 | 18.3 | 18.1 | 16.8 | 14.5 |
| Postneonatal | 21.7 | 16.1 | 9.8 | 7.0 | 6.4 | 5.9 | 4.2 |
| Sum | 51.8 | 40.8 | 30.6 | 25.3 | 24.5 | 22.7 | 18.7 |
| Weight | 0.31 | 0.30 | 0.29 | 0.28 | 0.28 | 0.28 | 0.29 |
| White, Nonsouth | | | | | | | |
| Neonatal | 25.9 | 22.6 | 18.8 | 17.5 | 16.9 | 15.8 | 13.4 |
| Postneonatal | 14.1 | 10.8 | 6.5 | 5.5 | 5.4 | 5.3 | 3.9 |
| Sum | 40.0 | 33.4 | 25.3 | 23.0 | 22.3 | 21.1 | 17.3 |
| Weight | 0.69 | 0.70 | 0.71 | 0.72 | 0.72 | 0.72 | 0.71 |
| White, National | 43.6 | 35.6 | 26.8 | 23.6 | 22.9 | 21.5 | 17.7 |
| | | | | | | | |
| Nonwhite, South | | | | | | | |
| Neonatal | 40.6 | 30.8 | 27.3 | 26.4 | 27.0 | 25.0 | 22.0 |
| Postneonatal | 35.2 | 25.1 | 18.3 | 17.7 | 20.0 | 18.1 | 11.0 |
| Sum | 75.8 | 55.9 | 45.6 | 44.1 | 47.0 | 43.1 | 33.0 |
| Weight | 0.78 | 0.74 | 0.67 | 0.61 | 0.56 | 0.51 | 0.49 |
| Nonwhite, Nonsouth | | | | | | | |
| Neonatal | 36.4 | 34.3 | 28.2 | 28.3 | 26.9 | 26.3 | 21.7 |
| Postneonatal | 31.1 | 24.9 | 14.6 | 12.7 | 11.9 | 11.6 | 8.3 |
| Sum | 67.5 | 59.2 | 42.8 | 41.0 | 38.8 | 37.9 | 30.0 |
| Weight | 0.22 | 0.26 | 0.33 | 0.39 | 0.44 | 0.49 | 0.51 |
| Nonwhite, National | 73.9 | 56.8 | 44.7 | 42.9 | 43.4 | 40.6 | 31.5 |

Note: The “weight” is the proportion of the relevant race-category’s births in that region.

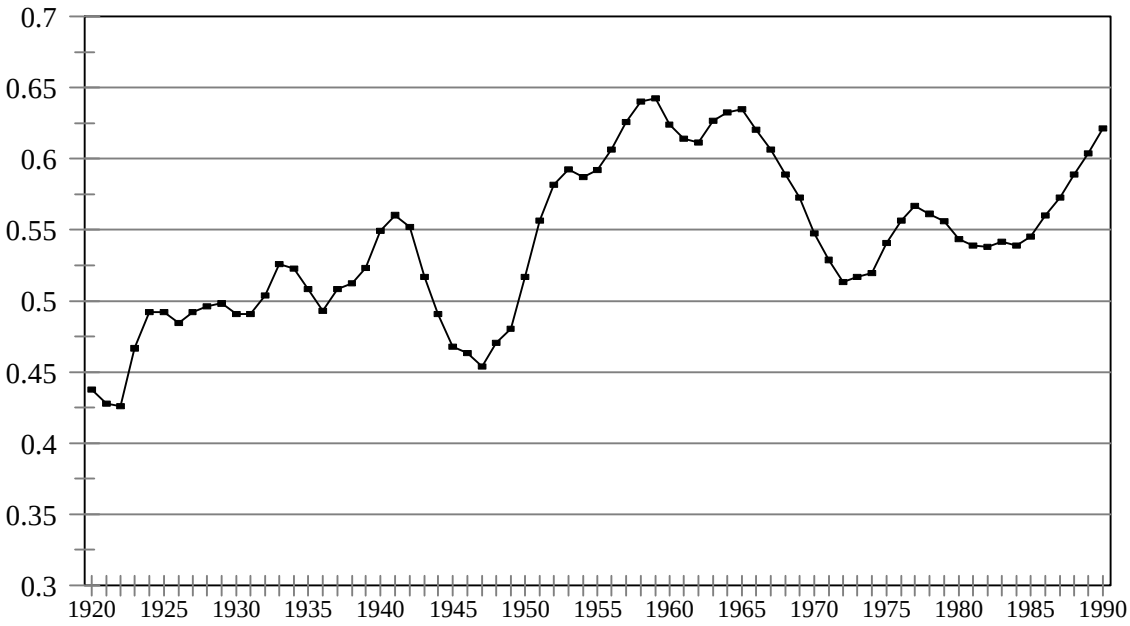
Figure 1
Ln Infant Mortality, 1920-1990




Notes: The area of coverage changes over time as states joined the birth- and death- registration areas. The series include Alaska from 1959, Hawaii from 1960, and exclude New Jersey in 1962-63 (missing data by race). From 1932 to 1934, the series count Mexican-Americans as nonwhite, but in other years they are counted as white. Mortality rates were calculated by place of occurrence prior to 1939 and by place of residence thereafter.

Sources: Compiled from Linder and Grove (1943) and Vital Statistics of the United States (various years).

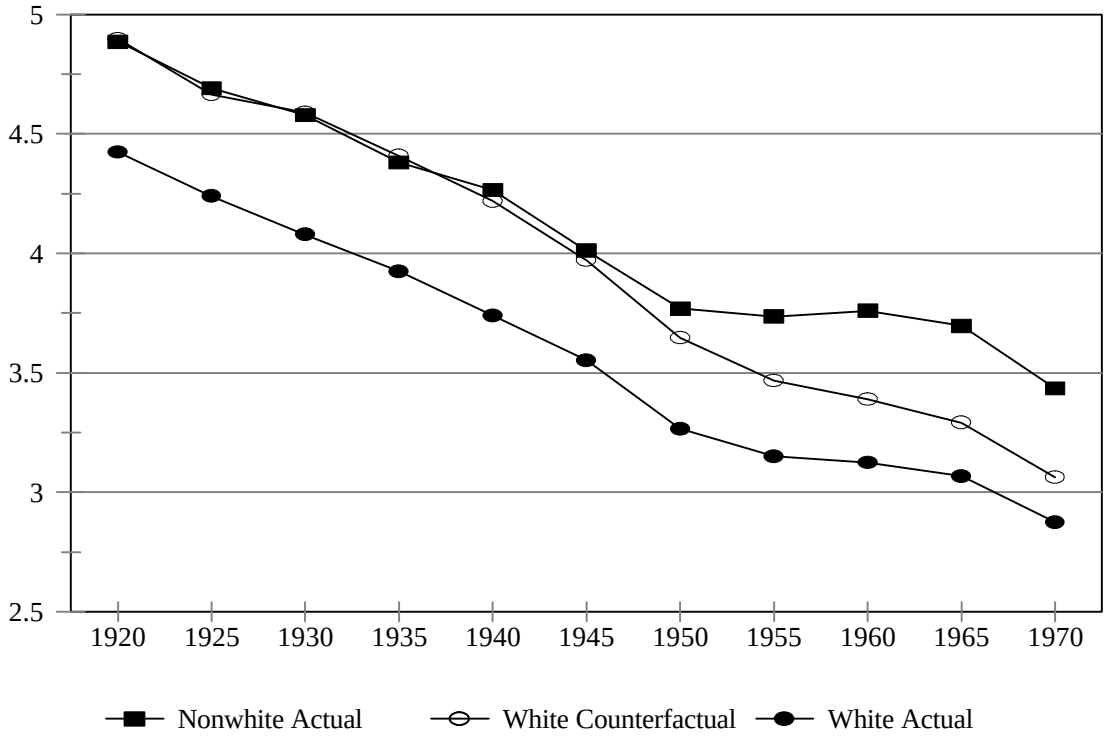
Figure 2:
Racial Gap in Log Infant Mortality




Notes: The graph plots a three-year moving average of the nonwhite-white gap in log infant mortality rates.

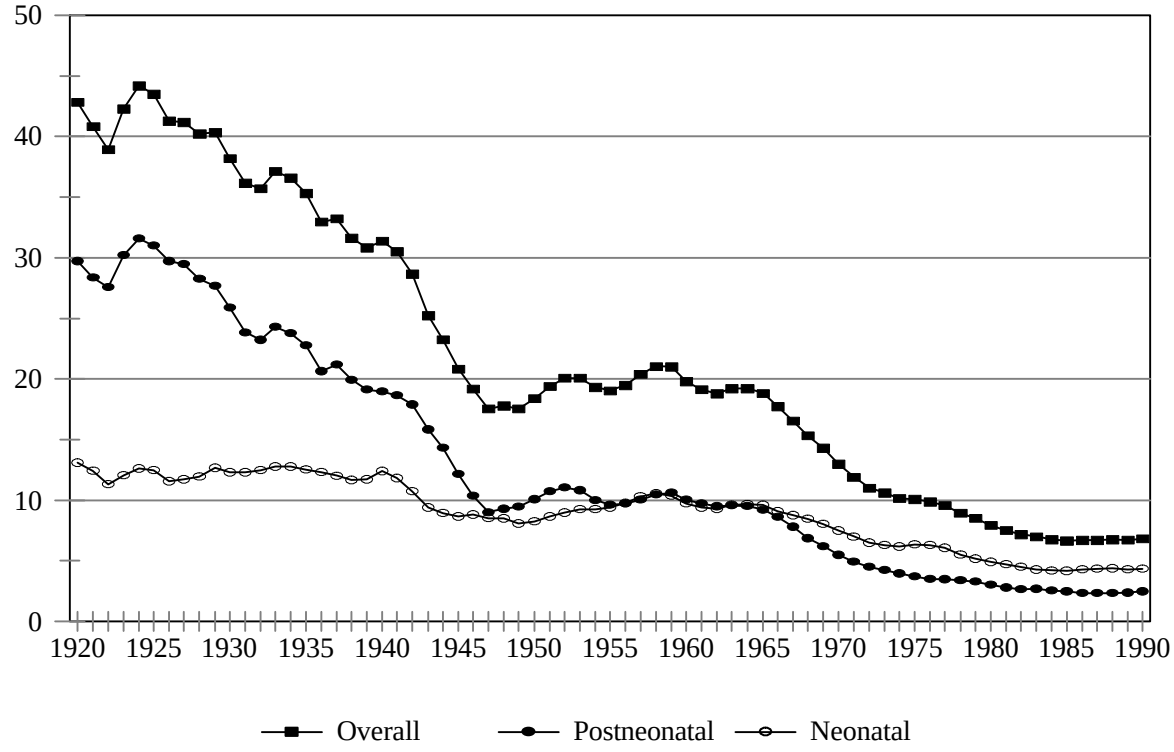
Sources: Compiled from Linder and Grove (1943) and Vital Statistics of the United States (various years).

Figure 3: Actual and Counterfactual Infant Mortality Rates




Notes: The counterfactual series is the product of the coefficients from Table 2, column 1 and the average nonwhite characteristics in each period.
Sources: See the text for a description of the dataset.

Figure 4: Infant Mortality Gap (Levels)




Note: The graph plots three-year moving averages.