

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

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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 and therefore remain a concern for current policymakers (Department of Health and Human Services 2000; 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, Figure 1 shows that while the racial gap declined in some periods, it rose substantially in others.

Thus, while we find it impossible to ignore the extraordinary gains in health enjoyed by both whites and nonwhites, 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 in a reasonably consistent form for whites and nonwhites over a relatively long period of time.

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; Department of Health and Human Services 2000; 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

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.

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

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.³ We suppose that utility-maximizing parents allocate resources over healthcare inputs and other goods, subject to resource constraints and relative prices, and that they influence infant survival probabilities accordingly. In our simple model, factors that influence infant health include income, residence, the availability of medical care, and women’s education. These variables reflect the financial, environmental, and medical resources available to families having children.

To explore the relationship between these variables and infant mortality links, we construct a panel of state-level data for whites and nonwhites for five-year intervals. 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. Income is important because it provides a measure of a household’s ability to purchase healthcare. 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, African Americans may have faced higher costs for healthcare services due to discriminatory practices within the healthcare system. Such discrimination 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).

Residence is important for two reasons. First, we include in our model the proportion of households in a state living in an urban area. Prior to 1920, it is clear that urban residence was relatively hazardous for infants’ health (see Haines, 2001). 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.⁴

³ See Grossman (1972), Rosenzweig and Schultz (1983), Berger and Leigh (1988), Kenkel (1991), and Goldman and Lakdawalla (2001) on household production of health.

⁴ See Troesken (1999 and 2001) on typhoid, water, and sewerage in U.S. cities in the late 19th and early 20th centuries.

In addition to living in an urban area, living in the South may have also influenced infant health. Compared to households living in non-southern regions, infant mortality rates in the South were higher for both blacks and whites. By itself, the geographic distribution (and redistribution) of African Americans might have 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).⁵

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.⁶ The level of education for women is also an important determinant of infant mortality for two reasons. 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.⁷ 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. To the extent that blacks' quality and quantity of education fell short of that provided whites, blacks might have found it more difficult to acquire information about medical advances and/or the availability of professional medical services.⁸ Similarly, educational deficiencies might have facilitated the influence of superstition and folk remedies.

⁵ 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.

⁶ 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. Such aspects include residential segregation/ghettoization and an unfavorable change in climate.

⁷ 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.

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

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

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

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.⁹ We add the time-period dummy variables (γ) 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.¹⁰

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

⁹ 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.

¹⁰ 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.

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.

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. The regressions suggest 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.

To what extent can racial gaps in the independent variables account for the persistent gap in infant mortality? Table 3 presents a variable-by-variable decomposition of the “explained” change in infant mortality. The explained gap is that part of the gap that can be attributed to changes in the observed characteristics between whites and nonwhites, while the “unexplained” change is that portion of the gap that occurs because of differences in the estimated coefficients between races, holding sample characteristics constant. The total gap is reported in the top row of Table 3, which provides information on how much each variable contributes to the explained change. The last row of the table shows how much of the total gap is explained solely by differences in white and nonwhite characteristics. 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.

This apparent decline in the significance of socioeconomic differences in explaining the infant mortality gap could be interpreted in several ways. It may be that unobserved factors influencing infant health diverged even as the observed socioeconomic factors converged. The time series graph of the racial gap in infant mortality rates 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). To a large extent, however, the abrupt changes in the infant mortality gap remain econometrically unexplained. Fortunately, the range of pertinent data expands in the post-1940 period,

and with it we can pursue a re-examination (albeit somewhat speculative) of these “unexplained” changes in the gap.

Table 4 reports data on neonatal (under 28 days old) and postneonatal infant mortality gaps for southern and non-southern regions. The distinction between neonatal and postneonatal data is useful because the ultimate causes of death differ substantially for the two groups (premature delivery and low birthweight dominate neonatal mortality; infectious disease and environmental factors are more prevalent in postneonatal mortality). The table also reports weights reflecting the proportion of white and nonwhite births in both regional categories, an important detail because of the dissimilar (and changing) geographic distributions of nonwhites and whites.

Clearly, the 1941-1946 period was characterized by rapidly declining levels of infant mortality for both race categories *and* a declining racial gap. Table 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.

There are two more likely contributors to the gap’s decline in the 1940s. First, as mentioned already and reflected in Table 4, 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, and consistent with the large decline in the southern nonwhite IMR, 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 supported the Emergency Maternal and Infant Care (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 (Sinai and Anderson 1948; 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, 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, at least among neonates, may be explained in large part by widening racial differences in birthweight. Birthweight statistics were reported for the first time during the 1950s, and Chase and Byrnes (1972) noted an increase in the proportion of low birthweight neonates (under 2,500 grams) among nonwhites from 10.2 percent of births in 1950 to 12.8 percent in 1960. Among whites the low birthweight proportion fell from 7.1 to 6.8 percent. Chase and Byrnes 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, or reported gestation.¹¹ Furthermore, the postneonatal gap, which is generally regarded as being fairly insensitive to birth weight, 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' birth weight. Finally, the relatively strong divergence of white and nonwhite infant mortality rates in the South recommends some regional focus for the investigation.

The rapidly changing healthcare system may have also influenced the infant mortality gap. Did white infants (especially in the South) benefit disproportionately from Hill-Burton funding of hospital expansion? Although we cannot rule this out, the available evidence appears to run against the grain of the hypothesis. In the South, the proportion of nonwhite hospital births increased from 24 to 74 percent between 1945 and 1960, whereas the white proportion increased from 68 to 97 percent. Of course, these figures say nothing about the quality of hospital care nor about the availability of prenatal care, but they do suggest a rapid increase in hospital and physician services for nonwhites. Furthermore, unlike the services available to southern blacks prior to World War II, Beardsley argues that "In new federally sponsored hospitals black patients, if still segregated, at least had benefit of modern facilities and enjoyed roughly equal treatment" (1987, p. 256).

A second major change in the healthcare system in this period was the rise of health insurance, often provided as a benefit through one's employer (Thomasson 2001). Again, race-specific information is sparse through most of the period, but prior to 1940, relatively few people of either race were covered by health insurance (about 16 percent in 1940). By 1962, however, about 74 percent of whites had hospital insurance whereas only 46 percent of nonwhites did (Hoffmann 1964). As noted above, there was substantial racial convergence in the proportion of births in hospitals between 1945 and 1960, but it is possible that the racial insurance gap had implications for the quality and frequency of prenatal and postnatal care.

In conclusion, we seek to understand the disparity between black and white infant mortality rates from 1920-1970. 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 mention several hypotheses regarding unobserved

¹¹ The potential influence of measurement error is particularly worrisome. Chase and Byrnes (1972, pp. 21-24) evaluate the hypothesis that better reporting was responsible for the shift in the nonwhite birth weight distribution from several perspectives. They conclude that the shift is not a statistical artifact, though it is impossible to rule out measurement error completely.

factors that could plausibly have driven the gap's changing character. Proximately, the neonatal gap appears to have been strongly influenced by a leftward shift in the nonwhite birthweight distribution during the 1950s, but the postneonatal gap also failed to narrow. Differences in access to health care and other behavior traits may help to explain the post-1950 differences, but without better data, our discussion remains speculative and open to more detailed empirical research.

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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 (0.4768)	3.9358 (0.4212)
Ln Income	7.5917 (0.4357)	6.8052 (0.6704)
Ln Education	2.3419 (0.1174)	2.1009 (0.2625)
Ln Urban	4.0954 (0.3207)	3.9879 (0.5245)
Ln Physicians	-6.6525 (0.2653)	-6.8232 (0.3125)
N	281	281
Panel B: Summary statistics of state-year values		
Infant Mortality	35.43 (18.88)	56.32 (27.12)
Income	2159 (837.2)	1104 (659.8)
Education	10.47 (1.183)	8.44 (2.045)
Urban	62.81 (16.67)	60.75 (26.32)
Physicians	0.0013 (0.0003)	0.0011 (0.0004)
South	0.3071 (0.4621)	0.6401 (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 (0.0886)	-0.1397 (0.1434)	0.0014 (0.1394)	-0.1742 (0.1770)	-0.1356 (0.0317)	0.0628 (0.1766)
Education	-0.8416 (0.2829)	-0.4254 (0.2203)	-0.3746 (0.3700)	-0.3654 (0.2612)	-0.6770 (0.2857)	-0.4318 (0.2198)
Urban	0.0969 (0.0780)	0.3073 (0.1423)	-0.0855 (0.1884)	0.5682 (0.2362)	0.1280 (0.0711)	0.2573 (0.1379)
Physicians	-0.1376 (0.0455)	-0.2057 (0.1098)	-0.3156 (0.0945)	-0.1666 (0.2147)	-0.1221 (0.0402)	-0.2350 (0.1081)
South	-----	-----	-----	-----	0.0539 (0.0317)	0.1573 (0.0629)
1925	-0.1438 (0.0211)	-0.1818 (0.0599)	-0.1671 (0.0284)	-0.1676 (0.0614)	-0.1530 (0.0208)	-0.1762 (0.0601)
1930	-0.2767 (0.0328)	-0.3365 (0.0778)	-0.3231 (0.0413)	-0.2995 (0.0928)	-0.2947 (0.0320)	-0.3078 (0.0795)
1935	-0.4058 (0.0525)	-0.5324 (0.0912)	-0.4728 (0.0566)	-0.5148 (0.1151)	-0.4328 (0.0466)	-0.4953 (0.0922)
1940	-0.5226 (0.0811)	-0.6201 (0.0788)	-0.6407 (0.0809)	-0.6139 (0.1033)	-0.5582 (0.0710)	-0.6174 (0.0762)
1945	-0.6252 (0.1104)	-0.7933 (0.0918)	-0.8131 (0.1201)	-0.7999 (0.0779)	-0.6701 (0.0947)	-0.8819 (0.0970)
1950	-0.8873 (0.1152)	-1.0290 (0.0860)	-1.0825 (0.1213)	-1.0612 (0.0918)	-0.9390 (0.0984)	-1.1006 (0.0837)
1955	-0.9637 (0.1175)	-1.0199 (0.0869)	-1.1833 (0.1357)	-1.0695 (0.0971)	-1.0220 (0.0997)	-1.1122 (0.0867)
1960	-0.9622 (0.1256)	-0.9681 (0.0930)	-1.1960 (0.1470)	-1.0335 (0.1089)	-1.0266 (0.1089)	-1.0668 (0.0921)
1965	-0.9640 (0.1443)	-0.9749 (0.1058)	-1.2266 (0.1722)	-1.0470 (0.1202)	-1.0361 (0.1258)	-1.1082 (0.1132)
1970	-1.1028 (0.1728)	-1.1827 (0.1209)	-1.3968 (0.2000)	-1.2588 (0.1323)	-1.1824 (0.1506)	-1.3537 (0.1356)
Constant	5.8960 (0.9152)	3.9867 (1.1183)	3.4625 (1.1628)	3.3551 (1.3395)	5.4800 (0.8792)	2.5960 (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.

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)



