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ABSTRACT

This paper examines the racial gap in infant mortality rates from 1920 to 1970. Using state-level panel data with information on income, urbanization, women's education, and physicians per capita, we can account for a large portion of the racial gap in infant mortality rates between 1920 and 1945, but a smaller portion thereafter. We re-examine the post-war period in light of trends in birth weight, smoking, air pollution, breast-feeding, insurance, and hospital births.

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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 (Atack and Passell 1994, p. 51); average height increased by about 7 centimeters for native-born white males (Costa and Steckel 1997, p. 51); and a number of deadly diseases and debilitating illnesses were all but vanquished. As the overall level of health improved, racial gaps in health outcomes persisted. For example, as shown in figures 1 and 2, the overall infant mortality rate (deaths under 1 year of age per 1,000 live births) and the absolute racial gap in infant mortality rates fell over time, but nonwhite infants were always more likely to die than whites. In fact, nonwhites were about 75 percent more likely to die than whites throughout the twentieth century. When translated into numbers of "excess" deaths, it is clear that the racial gap remained non-trivial even as it narrowed: In 1940, approximately 8,900 more nonwhite infants died than would have if they had had the same mortality rate as whites; in 1970, the "excess" was approximately 8,400 infants.[1]

Racial gaps in health status remain a serious concern for U.S. policymakers (Department of Health and Human Services 2000; Levine et al. 2001; Byrd and Clayton 2002).[2] In large part, this is because physical health is a direct determinant of well-being. Additionally, because one's health may influence educational attainment, labor force participation, and one's children's health, the consequences of intergroup health disparities may be quite far-reaching.[3] In discussions of

[1] Thus, the increase in the number of births offsets the decline in the infant mortality rate gap in generating the number of "excess" deaths. 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.

[2] Whether this concern is potentially misguided is not the focus of this paper (see Scanlon 2000). Rather, we take the concern as given and try to advance the understanding of the history of health.

[3] For example, using British data for a cohort born in 1958, Currie and Hyson (1999) found that low birth weight had an adverse long-run impact on educational attainment and labor market outcomes. Also see Grossman (1975) and Edwards and Grossman (1979) on childhood health, cognitive development, and education. Such considerations have not yet been part of the literature on the long-run evolution of racial

intergroup health differences, infant mortality is a frequently referenced barometer. It is a simple and widely available measure that is sensitive to group disparities in maternal health, resources, behavior, and environment, as well as to health-related policy, technology, and institutions.

The literature on infant mortality is voluminous and multi-faceted, but to our knowledge, previous works have not systematically examined the determinants of the racial gap over the full 1920 to 1970 period.[4] This was a remarkable period of medical and social transformation. Professional training for doctors, nurses, and midwives was upgraded and standardized; federal and state governments poured resources into improving and extending hospital and clinic systems and into funding services for 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. Finally, all of this unfolded in concert with the period's most dramatic economic and political events: the Great Migration, the Great Depression, World War II, and the Civil Rights Movement.

In this paper, we explore state-level infant mortality data in an empirical and historical framework that sheds light on both the declining overall level of infant mortality and the racial gap. In section 2, we discuss the interaction of race and infant mortality in the context of a simple model of demand for and supply of broadly-defined healthcare. This framework suggests strong links between socioeconomic characteristics, technological progress, healthcare, and infant mortality. To explore these links, we construct a panel of state-level data for whites and nonwhites in section 3, and in section 4, we outline our econometric approach and report the

gaps in labor market outcomes in the U.S. (Smith and Welch 1989, Donohue and Heckman 1991).

[4] See Rochester 1923; Shapiro et al. 1968; Chase 1972; Shin 1975; Grossman and Jacobowitz 1981; David and Collins 1997; Costa 1998; and Department of Health and Human Services 2000.

results of our analysis. We find that differences in income, education, urban residence, and the supply of physicians can account for a large portion of the racial gap in infant mortality rates from 1920 to 1945, but that they account for a declining proportion of the gap thereafter. There was a strong secular decline in infant mortality for both races, but the racial gap did not narrow continuously over time. The episodic changes in the gap are re-examined in section 5 in light of trends in birth weight, maternal characteristics, the prevalence of hospital births, air pollution, smoking and breast-feeding behavior, and insurance coverage.

2. A Framework for Exploring Infant Mortality Rates, 1920-1970

Our interpretation is guided by a simple model of infant mortality in which the likelihood of death 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.[5] This production function may evolve as technology, knowledge, and institutions change, may be shocked by changes in the disease environment, and may embody differences in "health productivity" across groups, perhaps associated with educational, environmental, or even cultural and genetic characteristics. Furthermore, although our discussion focuses on the time span from conception to one year of age, maternal health prior to pregnancy may influence infant health, and a mother's health is influenced by her own lifetime stream of healthcare. We suppose that parents allocate resources over healthcare inputs and other goods, subject to resource constraints and relative prices, and that they influence infant mortality probabilities accordingly.

For the most part, it is not possible to observe healthcare inputs directly in this period.

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

Consequently, the exact pathways through which healthcare influenced infant mortality remain unidentified.[6] Likewise, because observed socioeconomic characteristics may be correlated with unobserved health-related behaviors, we do not claim to identify true treatment effects in the paper's econometric section. Rather, we start by linking fundamental socioeconomic characteristics, such as income, education, and area of residence, to infant health outcomes, both conceptually and empirically. Then, relying on more fragmentary evidence, we map several of the intermediating connections between those characteristics and infant mortality rates in more detail.

Demand and Supply of "Healthcare"

Nonwhites' average income and wealth lagged behind whites' throughout the period under study, implying tighter budget constraints and less demand for all normal goods, including infant-related healthcare (ranging from maternal nutrition to physician services for infants). Low socioeconomic status might not only constrain expenditures on healthcare, but might also influence fundamental attitudes regarding health, sickness, death, and treatment (Beardsley 1987, p. 33). Additionally, we hypothesize that for various reasons, ranging from the population's geographic distribution to the prevalence of discriminatory practices, nonwhites might have faced higher effective costs for any given quantity of healthcare, further dampening their consumption relative to that of whites. Reflecting blacks' geographic concentration, our discussion begins with its focus on the South.

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,

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

southern real per capita income was about 64 percent of the national average (Mitchener and McLean 1999).[7] Such low average income levels not only may imply less private demand for healthcare, as suggested above, but also may imply tightly constrained public supplies of health-related services, including hospitals, subsidized care for the poor, and investments in sanitation and education.[8]

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, effort, and money) when seeking professional medical services. On the other hand, although blacks in urban areas may have benefitted from proximity to medical facilities, discriminatory practices underpinning patterns of residential segregation constrained the supply of housing for blacks, thereby raising its price and exacerbating crowded, unhealthy living conditions in the emerging ghettos (Massey and Denton 1993). Additionally, as discussed later in the paper, urban life may have been associated with health behaviors (like smoking) and environmental conditions (like air pollution) that are now known to be detrimental to maternal health and fetal development.

The shortcomings of the southern educational system through most of the twentieth century, especially for blacks, have been well documented (Margo 1990). 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 health-related information; or more directly, to the extent that hygienic and health information was transmitted through schools, blacks might have had less

[7] It is possible that the relative health of southerners affected the region's relative productivity, as argued by Brinkley (1997) for the late nineteenth century.

[8] On average, lower-income 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).

exposure to such information.[9] Similarly, educational deficiencies might have facilitated the continuing influence of superstition, folk remedies, and "lay referral" even when modern medical care and information was available (Beardsley 1987, pp. 32-35). Along these lines, in recent studies, better educated people appear to be more behaviorally responsive to health-related information than others (Kenkel 1991, Meara 2001).

Finally, 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, limiting the number and quality of black doctors, and using black patients for clinical studies with dubious ethical standards (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.[10] 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.[11] Additionally, until federal funds

[9] In this paper, we view educational attainment as an exogenous variable. See Goldman and Lakdawalla (2001) and Meara (2001) for recent discussions 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).

[10] 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).

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

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).[12]

Between 1920 and 1970, millions of southerners moved to other parts of the country. 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, as discussed in detail later in the paper, the pollution levels in urban centers were much higher than elsewhere, and pollution may be detrimental to fetal and infant health. Third, although "chain migration" patterns often ensured some support from extended family networks, striking out on one's own might have entailed additional economic and health risks. Fourth, 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 and unhealthy. And fifth, the industrial employment opportunities open to blacks, especially early in the period under study, were sometimes physically debilitating (see Maloney and Whatley 1995).

Institutional Interventions

Against this backdrop, the Rockefeller Sanitary Commission for the Eradication of Hookworm (1909-1915), the Rosenwald Fund, and the Duke Endowment all made efforts to

[12] 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).

improve poor children's health and educational status (Beardsley 1987, pp. 51, 114-127).[13] Perhaps more importantly, the federal government became increasingly engaged in the study, funding, and provision of healthcare for infants and mothers (see Meckel 1998). 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 (U.S. Dept. of Labor 1931; Lemons 1969). Although it was discontinued, the Sheppard-Towner program laid the political and administrative foundations for subsequent and more extensive federal health initiatives (Sinai and Anderson 1948; Bradbury 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. In the late 1930s, proposals to expand further the government's role in funding and providing universal maternal and infant healthcare met with staunch resistence from the American Medical Association (Meckel 1998, p. 223-224). Nevertheless, from 1943 to 1949, the Children's Bureau administered an extensive Emergency Maternal and Infant Care (EMIC) program which funded medical care for the pregnant wives and infant children of low-ranking servicemen. EMIC handled approximately 1.5 million cases, and at its peak, EMIC funding covered roughly 1 of

[13] 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.

every 7 U.S. births (Bradbury 1956, p. 60; Sinai and Anderson 1948, p. 83). Just as importantly, in some places (particularly the South) the program directly elevated the standard of prenatal and obstetric care through its process of review and approval of medical facilities and services (Sinai and Anderson 1948; Beardsley 1987, p. 175-176).[14] Another 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 (like much of the South) with relatively few hospital beds per capita (Lave and Lave 1974).

The Technology of Birth and Infant Health Care

The twentieth-century transformation of medicine put hospitals and physicians at the center of maternal and infant healthcare. The care provided in hospitals influenced the infant mortality rate in several ways, which we discuss in three parts: the process of birth itself, care specific to neonates (less than one month old), and care that strongly influenced post-neonatal mortality (after one month but before one year old). Improvements in medical care had clear implications for the overall level of infant mortality, but the implications for the racial gap in mortality rates are less obvious and may hinge upon the extent of differential access to high quality care, a topic we take up later in the paper.

In 1900 only about 5 percent of all births occurred in hospitals (Wertz and Wertz 1989, p. 133). American women favored giving birth in their homes, often with the assistance of midwives rather than physicians. At the time, the benefits of hospital and physician-attended birth were

[14] For example, drawing from interviews in a southern state, Sinai and Anderson (1948) report, "Not a few physicians practicing in the area have asked what the prenatal visits are for and what they are supposed to do at such visits. They have practiced for years without giving any thought to prenatal care" (p. 165). The value of prenatal exams in detecting eclampsia, venereal disease, and malformed pelvises was apparent to specialists by 1910 (Wertz and Wertz 1989).

slim. Good hospitals strove to be aseptic, but general practitioners were poorly trained and probably no better at delivering newborns than midwives (Wertz and Wertz 1989, p. 145). After 1900, the accumulation of clinical experience at hospitals and the rise of specially trained obstetricians increased the advantages of hospital births, particularly for complicated deliveries. As the population urbanized, pain-relieving procedures were perfected, insurance coverage spread, and customs changed, birth became a predominantly hospital-based activity (Leavitt 1986). By 1955, 94 percent of deliveries took place in hospitals where new drugs for speeding up delivery, X-ray machines for detecting pelvic deformities, fetal-heart monitoring equipment, and cesarean sections were common (Wertz and Wertz 1989).

Great strides in caring for unhealthy neonates, particularly those of low birth weight (under 2,500 grams), were made relatively recently. Modern neonatal intensive care units emerged in the late 1960s after decades of struggle to improve the lot of premature infants, and since then, progress has been rapid by historical standards. At mid-century, the outlook for premature infants was far less optimistic: special nurseries equipped with incubators existed in many cities in the 1940s (Corwin 1952, Cone 1985), but mortality rates remained relatively high. In 1950, 17.4 percent of low birth weight infants died as neonates, and in 1960, 17.2 percent died (Chase 1972, p. 9), but only 9.6 percent died in 1983 (CDC 2001). Even so, it is possible that the slow accumulation of clinical experience (e.g., in feeding) and technological advance (e.g., in incubators and drug therapies) made a significant impact on infant mortality rates prior to 1950 (Costa 1998). Cone (1985, p. 51, citing Schwartz and Kohn 1921) suggests that approximately 30 percent of low birth weight infants died as neonates in 1920.

New technologies made a large impact on post-neonatal mortality rates by mid-century. In particular, two leading causes of post-neonatal infant death, pneumonia and gastroenteritis,

could be effectively treated in hospitals by the mid-1940s (Almond, Chay, and Greenstone 2001). Infants with pneumonia benefitted from intravenous fluid replacement and (after 1945) antibiotics, as well as from assistance with breathing. Intravenous fluid replacement was also especially helpful for infants with gastroenteritis since they could dehydrate rapidly otherwise. Overall, the post-neonatal mortality rate fell from about 29 to 9 per 1,000 births between 1930 to 1950, and the post-neonatal proportion of all infant deaths fell from 45 to 30 percent. Again, we explore potential differences across races in the new technologies' impact later in the paper.

3. State-Level Data

In the absence of family- or infant-level data, we exploit variation across states and over time (at five-year intervals) to measure the empirical strength of the links between socioeconomic variables and infant mortality. 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, and average years of education for women between 20 and 40 years of age. The dataset also includes a measure of 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 birth weight) will be discussed when and where evidence is available (see section 5), but they do not enter the econometric exploration directly.

The infant mortality rate is 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 94 percent of white births were indeed registered, and 82 percent of nonwhite births were. By 1950, 99 percent of white births were registered and 94 percent of nonwhite births were (Vital Statistics 1950, p. 113). The under-registration of births is not a problem (for the purposes of this study) if births and infant deaths were under-reported by the same proportion within states. Otherwise, the infant mortality rate would be mismeasured, and if this mismeasurement is not random, it could confound the analysis below. For example, *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 income and infant mortality. Unfortunately, no checks exist on the completeness of death registration. So, for the most part, like Shin (1975), we must take the infant mortality data at face value, but we will check some specifications that incorporate information from the Census Bureau's 1940 accuracy study.

A household's ability to pay for food, housing, and medical services depends in large part on its income and wealth.[15] Because the census first inquired about income in 1940, compiling

[15] See Steckel (1988) and Ferrie (2001) for micro-level studies of wealth and mortality in the nineteenth 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.

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.

(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. θ is the proportion of the population that is white. In equation 2, 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).[16]

Given measures of Y_{it} and Z_{it}, and using the number of IPUMS observations for each race category to measure θ, we have enough information to calculate $Y_{W, it}$ and $Y_{NW, it}$. Essentially, we are simply using Z and θ 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 θ 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.

[16] 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).

To get real income estimates, we adjust for price level changes over time using national consumer price indices (U.S. Department of Commerce 1975). [17]

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.[18] Therefore, even though education and income tend to be strongly 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 pre-1940 census reports literacy rates, but there is no simple way to move from literacy rates to average years of education.

We estimated years of education for the years prior to 1940 by using the individual-level information contained in the 1940 IPUMS sample. 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

[17] It might be argued that the lower end of the income distribution is most relevant to the study of infant mortality. We constructed measures of the proportion (between 0 and 1) of the male labor force employed in relatively low-ranked occupations according to the IPUMS occscore variable (including unemployed). The measures are highly correlated with the log average income measures used in the regressions (0.77 for whites, 0.87 for nonwhites). When added to the base regressions, the coefficient for whites is statistically insignificant, but for blacks is significant and negative (-0.42, t-stat = 2.89).

[18] See Berger and Leigh (1989) or Kenkel (1991) for discussions of the current 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.

education and infant health, it can certainly be argued that focusing on young women is a useful sample restriction.[19]

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.

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".[20] 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.

[19] 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 nineteenth century had less schooling relative to whites than suggested by the 1940 census data.

[20] 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.

Thus, the urban variable may capture not only health disamenities associated with urban life, but also the relatively high cost of living in urban areas.

The census occupation tables report the number of physicians in each state.[21] *Ceteris paribus*, we expect a larger number of physicians per capita to lower the effective cost of medical care, and therefore to lower the rate of infant mortality.[22] 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 for each race category. Some states had very few nonwhite residents, and so the nonwhite sample includes fewer state-year cells than the white sample. Each state-race-year observation is weighted by the number of births that it represents. 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

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

[22] 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.

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 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.[23] 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. Pritchett and Summers (1997) chose a similar functional form in their analysis of an international panel dataset, as did Flegg (1982).[24] Later, we split the sample at 1950 and allow the coefficients to vary between the early and later periods.

Table 2 reports regression results from three different log specifications. Columns 1 and 2 correspond to the basic specification described by equation 3. Columns 3 and 4 add state dummy

[23] 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 that the regressions' explanatory variables are endogenous. In theory, an instrumental variable approach could help circumvent these concerns, but plausible instruments are scarce in this paper's context.

[24] The relationship between infant mortality and income and education is clearly non-linear. More formally, in this case, P_E tests of the linear versus log form favor the log specification (Greene 1993, pp. 321-322). Additionally, link tests, essentially regressions of the dependent variable on fitted values and fitted values squared, do not suggest misspecification. The relative stability of the coefficient estimates when the sample is split at 1950, particularly for whites, also suggests that the specification is reasonable.

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).[25] 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.[26] 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).

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. For

[25] The nonwhite results were sensitive to outlying observations from Arizona, New Mexico, South Dakota, and Montana, all of which had low black/nonwhite ratios. The nonwhite results reported in table 2 omit these states, representing approximately 1 percent of total nonwhite births in the sample. The white results were not sensitive to the inclusion of these states, and they are included in table 2's regressions.

[26] Interestingly, adding a variable for the percentage of the population (not logged) residing in relatively large urban areas (over 100,000 population) to columns 1 and 2 returns negative, but not particularly large, coefficients (-0.0018 for nonwhites; -0.0006 for whites). This is consistent with benefits from proximity to large medical centers. The other coefficients are relatively stable.

whites (column 5), there is no significant difference in southern infant mortality after accounting for the state's economic characteristics. For nonwhites, however, the infant mortality rate is nearly 15 percent higher in the South, ceteris paribus.

There is evidence that southern states had less complete birth 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 remained insignificant. In the nonwhite sample, the South coefficient increased slightly to 16.5 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 the quality of physicians, education, or urban sanitation.

Even with controls for income, education, urban residence, and supply of physicians, a 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 healthcare system, the secular decline in infant mortality stagnates for nonwhites from 1950 to 1965 and for whites between 1955 and 1965. Thus, 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 more than the white coefficient, and during others the white time coefficient fell by more than the nonwhite one. We discuss these uneven movements at length in the next section.

To what extent can racial gaps in the independent variables account for the gap in infant mortality? The answer depends somewhat on whether coefficients from the white or the nonwhite regression are used to weight racial differences in characteristics (Oaxaca 1973). For example, given the racial gap in educational attainment, the white coefficient on education would suggest that a relatively large portion of the gap is accounted for educational differences, whereas the nonwhite coefficient would suggest that a smaller portion is so explained. Rather than arbitrarily choosing one set of coefficients or the other, we use a simple average of the two sets of coefficients. In figure 3a we plot the proportion of the gap that is accounted for by racial differences in observed characteristics at each point in time. From this perspective, around 80 percent of the gap can be accounted for by differences in characteristics up to 1945, but by 1970, only about a third of the gap can be explained.

Table 3 decomposes the infant mortality gap, variable by variable, for each year. 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 accounts for more than 70 percent of the observed infant mortality gap (for example, in 1930, 0.343/0.493 = 0.70). But the racial gap in years of education narrowed quickly over time, and by 1970, the educational gap can account for only about 12 percent of the infant mortality gap. Up to 1945, the income gap accounts for about 25 percent of the overall infant mortality gap, but by 1970 income accounts for only 15 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 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.

This apparent decline in the significance of socioeconomic differences in explaining the infant mortality gap could be interpreted in different ways. It may be that unobserved factors influencing infant health diverged even as the observed socioeconomic factors converged. Alternatively, it may be that the relationship between the observed characteristics and infant mortality changed in the postwar period, and that consequently, the coefficients estimated over the full 1920-1970 period are inaccurate descriptors of the 1950-1970 experience. To test this latter hypothesis, we re-estimated regression equations separately for the 1920 to 1945 and the 1950 to 1970 periods, thereby allowing the coefficients to change from the early to the later period. Results are reported in table 4. Although the coefficients do change somewhat from the early to the late period, the only instances in which the changes are nearly statistically significant are in the nonwhite sample for the urban (p-value = 0.09) and physician (p-value = 0.06) coefficients.[27] The differences in the point estimates are non-trivial, however, and therefore, in figure 3b we constructed a two-part series which is similar in nature to that presented in figure 3a, but which is based on the separate sets of coefficients for the early and late periods. The pattern of figure 3b is strikingly similar to that of figure 3a: from 1950 to 1970, observable characteristics explained less and less of the racial infant mortality gap.

The premise of a related hypothesis is that socioeconomic characteristics might still be strong predictors of post-neonatal mortality in the postwar period, but weak predictors of neonatal mortality. Coupled with the declining proportion of all infant deaths in the post-neonatal category, perhaps this is why the socioeconomic characteristics appear to lose their explanatory

[27] To facilitate testing of the significance of the coefficient changes, we ran the regressions with data for the entire period and interactions for the variables in the post-1945 period. The coefficient estimates are identical to those reported in table 4. The p-values reported in the text pertain to the coefficient estimates on the interaction terms.

power for overall infant mortality. After running separate regressions for neonatal and post-neonatal mortality rates from 1950 to 1970 (results not shown), we find that socioeconomic characteristics do explain more of the post-neonatal gap than the neonatal gap. But for both rates, the proportion of the gap accounted for by socioeconomic characteristics declines over time (from about 63 to 27 percent for neonates; and from about 90 to 40 percent for post-neonates).

These findings fit reasonably well with recent work suggesting that differences in socioeconomic characteristics could explain only part of the racial infant mortality gap in the late twentieth century (e.g., see Miller 2001). In the next section, we discuss several variants of the first broad hypothesis – that changes in unobserved determinants of infant health tended to widen the racial infant mortality gap even as indicators of socioeconomic status converged.

5. Exploring the "Unexplained" Changes in the Infant Mortality Gap

After 1940, 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 prolonged stagnation (1948-1965). The two episodes of rapid decline coincide almost exactly with the only two periods of significant decline in the racial wage gap (for men) (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 postwar changes in the infant mortality gap remain econometrically unexplained and invite additional investigation.

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 markedly for the two groups (low birth weight dominates neonatal mortality; infectious disease is more prevalent in

postneonatal mortality). Table 5 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 each regional category, 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 (figure 1) *and* by a declining racial gap (figure 2). Figure 4 shows that the racial gap declined especially sharply in the postneonatal category, and this 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 5, it is apparent that the strong declines in southern nonwhite neonatal and postneonatal mortality dominate all other movements during the period.

Movement in the racial gap reversed course after 1948. While white infant mortality continued to fall for both neonatal and postneonatal infants through the 1950s, albeit slowly compared to the 1940s, the nonwhite rates were nearly constant. This was especially true in the South, where the nonwhite infant mortality rate apparently increased slightly between 1950 and 1960 while the white rate declined by 6.1 per 1,000 births.

After nearly twenty years of stagnation, the nonwhite-white infant mortality gap plunged from 1966 to 1971. This decline has three important features. First, the overall decline was somewhat stronger for postneonates (from 9.0 to 4.8) than for neonates (from 9.2 to 6.6). Second, the decline in nonwhite mortality outside the South (by 8 per thousand births) was nearly as large as in the South (by 10 per thousand births), and about half of nonwhite births were outside the South by 1965. Third, whereas the decline in nonwhite postneonatal mortality is the

dominant feature of the southern data, the decline in nonwhite neonatal mortality is the dominant feature of the nonsouthern data.

In the previous section's econometric investigation, neither the 1950-1960 nor the 1965-1970 changes in the racial gap were explained by relative movements in the observed X variables. The remainder of this section assembles a wide range of fragmentary evidence in discussing the unobserved factors that might have influenced the post-war gap. In doing so, it identifies several potentially fruitful areas for additional, more detailed research.

Wartime Developments

The development of penicillin and other antibiotics was the single greatest medical advance of the 1940s, and it had a strong impact on the level of infant mortality, especially for postneonatal infants (older than 28 days old) (CDC October 1999). Thus, figure 4 might appear to suggest that nonwhites benefitted disproportionately from the new antibiotics. Along these lines, nonwhite post-neonatal deaths due to pneumonia (treatable with penicillin) and influenza (grouped with pneumonia statistically) fell from 13 to 7 per 1,000 live births between 1941 and 1946, whereas white fatalities fell from 5 to 3 per 1,000. However, the timing and magnitude of the racial gap's decline implies that penicillin cannot be the whole story behind the 1941-1946 convergence, 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 additional likely contributors to the gap's decline in the 1940s. First, as mentioned already and reflected in table 3, there was 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 mortality rate, 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) positive spillovers to the general quality of infant care (Sinai and Anderson 1948; Beardsley 1987, pp. 173-175).

The potential importance of the EMIC program is relatively clear – it offered free healthcare to more than a million relatively poor women and children, and it demanded that hospitals and physicians meet or exceed certain standards of care. The potential impact of the broader effort to combat disease is perhaps less obvious, but through the first decades of the century, chronic maternal disease, particularly syphilis, was probably the chief cause of premature delivery (and therefore the chief cause of neonatal death) (Cone 1985, p. 47). Given that syphilis and other sexually transmitted diseases were more prevalent among blacks than whites (Byrd and Clayton 2002, pp. 159, 233), black neonates might have disproportionately benefitted from the disease's decline.

Infant Mortality and Birth Weight

The widening infant mortality gap after 1948, at least among neonates, may be accounted for in large part by widening racial differences in birth weight. Comprehensive birth weight statistics became available during the 1950s, and Chase and Byrnes (1972) noted an increase in the proportion of low birth weight neonates (under 2,500 grams) among nonwhites from 10.2 percent of births in 1950 to 12.8 percent in 1960 and 13.8 percent in 1966. Among whites, the low birth weight proportion fell from 7.1 in 1950 to 6.8 percent in 1960 and then increased

slightly to 7.2 in 1966. 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 birth weight 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). In fact, table 6 shows that given the 1950 distribution of nonwhite neonatal mortality rates (across the ten reported weight categories), the change in the distribution of nonwhite birth weights from 1950 to 1960 by itself would raise the nonwhite neonatal infant mortality rate by 5.7 per thousand live births. This upward force was offset by declining mortality rates *within* each weight category.

An immediate concern is that the apparent shift in the nonwhite birth weight distribution is a statistical artifact associated with the increasing proportion of nonwhite births occurring in hospitals. This is impossible to disprove, but table 7 shows that the proportion of low birth weight nonwhite infants increased everywhere, including regions (outside South) where birth registration rates were very high in 1950 and where there were relatively small changes in the proportion of infants delivered in hospitals. We do have some concerns regarding the classification and reporting of fetal deaths versus (low birth weight) neonatal deaths, but the historical fetal death data are difficult to interpret due to inconsistencies in their collection (Grove and Hetzel 1968, p. 48). Chase and Byrnes (1972) evaluate the measurement error hypothesis from a variety of other perspectives and conclude that the leftward shift is unlikely to be solely an artifact of the data.

Even if the rising proportion of low birth weight infants among nonwhites is the proximate cause of the widening neonatal infant mortality gap in the 1950s, the ultimate causes remain

unidentified.[28] Furthermore, the postneonatal gap, which is generally regarded as being fairly insensitive to birth weight, also increased during the 1950s, 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.

Importantly, between 1965 and 1970, the relative movements in the proportion of low birth weight infants were small – there were slight declines for both whites and nonwhites. Therefore, the observed convergence of infant mortality rates in the late 1960s appears to have had little to do with changes in infants' relative health at birth. Institutional and technological changes that may have contributed to improved mortality rates conditional on health at birth are discussed below.

Environmental Quality

At least one provocative hypothesis related to environmental quality and infant health lends itself to investigation.[29] Chay and Greenstone (1999, 2001) and Wolpaw Reyes (2001) have argued that environmental conditions, in particular air pollution, might be causally linked to infant mortality. If so, then the massive redistribution of blacks out of the rural South might have had adverse health consequences that tended to offset health improvements associated with proximity to hospital care and/or higher incomes. The "urban" variable used in the econometric exploration should have captured some of this effect, but it is a coarse characterization of environmental quality.

[28] For some discussion of the medical literature on genetic versus behavioral, environmental, and socioeconomic origins of the disparity in the distributions of black and white birth weights, see David and Collins (1997).

[29] A second hypothesis, which we cannot evaluate empirically with the available data, is that within urban areas, the rapid suburbanization of white families improved the environmental quality enjoyed by "urban" whites relative to "urban" nonwhites who were generally excluded from new suburban developments.

Are the links between African Americans' geographic redistribution, pollution exposure, and infant mortality strong enough to matter? Given the existing data, a precise assessment of this issue is infeasible, but it is possible to get some empirical perspective on the magnitudes involved using just three key pieces of information. First, we need measures of air quality. The earliest available Environmental Protection Agency (EPA) measures of ambient air quality are for 1960, before the Clean Air Act of 1970 induced large declines in air pollution (EPA 1973). The data for total suspended particulates (TSP) are reported separately for urban and non-urban areas. Second, we need to know the proportion of births (by race) for urban versus non-urban residents at different points in time. Third, we need Chay and Greenstone's (2001) point estimate of infant mortality's response to TSP air pollution.

This approach builds in a bias that may work against finding a large pollution effect. In particular, Chay and Greenstone's studies focus on the early 1970s and early 1980s, by which time, unhealthy infants were benefitting from significant technological advances in neonatal intensive care. As noted above, 17.4 percent of low birth weight (under 2,500 grams) infants died as neonates in 1950 (Chase 1972, p. 9), compared to 9.6 percent in 1983 (CDC 2001). Thus, the response of infant mortality to pollution might have been larger before 1970 than after.

In 1960, the gap between non-urban and urban (central-city) TSP levels was quite large, approximately 25 versus 110 micrograms per cubic meter (EPA 1973, p. 48). That is, around mid-century, a rural to urban migrant might well have quadrupled her exposure to air pollution. Among blacks, the proportion of births to metropolitan area residents increased from about 32 percent to about 75 percent between 1940 and 1970.[30] Chay and Greenstone (2001) estimate that

[30] In 1970, Vital Statistics reports births by metropolitan status for blacks (as opposed to nonwhites). In 1940, the metropolitan area definitions were not in place, but they have been retroactively imposed on the 1940 IPUMS data (Ruggles and Sobek 1997). We estimate the proportion of black metropolitan area births in 1940 as the proportion of very young (under three years of age) black children resident in metro areas.

circa 1970 an increase of 1 microgram of TSP per cubic meter increased infant mortality by 0.05 to 0.08 per 1,000 live births. Thus, choosing the mid-point of Chay and Greenstone's range, the redistribution of nonwhites to places with worse air quality tended to increase the nonwhite infant mortality rate by about 2.4 per thousand live births. In any case, the impact of environmental quality on infant mortality is certainly non-trivial – summed over time, it would amount to thousands of lives. At the same time, given that the overall nonwhite infant mortality rate fell by nearly 43 per thousand live births between 1940 and 1970, the magnitude of the pollution effect was not particularly large compared to other forces at work.

Health-Related Behavior: Smoking and Breast-Feeding

A separate set of hypotheses suggests that differential racial trends in health-related behavior could have influenced the infant mortality figures. For example, nationally, per adult consumption of cigarettes increased dramatically between 1920 (665 per year) and 1945 (3,449 per year) and peaked during the 1960s (at about 4,300 per year) (Giovino et al. 1994). Using 1988 data, Meara (2001) documents a strong correlation between smoking during pregnancy and the probability of having a low birth-weight infant. Therefore, if nonwhites' cigarette consumption increased at a faster rate than whites' consumption, particularly among young women, then the nonwhite birth weight distribution may have tended to shift leftward relative to the white distribution.

The earliest statistical characterization of smoking by race (to our knowledge) suggests that by 1955 the proportion of young nonwhite women who smoked was as high as that of whites (35.6 versus 34.2 percent among 25-34 year old women), though white women were more likely to smoke heavily (Haenszel et al. 1956, p. 36). The cross-sectional evidence also suggests that

the farm population was less likely to smoke than the urban population, implying that the rapid urbanization of blacks from 1920 to 1970 might have been accompanied by a relatively rapid increase in cigarette consumption. However, it appears that from 1950 to 1965 the smoking proportion of white and black young women was nearly equal (Burns et al. 1997), and at the same time, the low birth weight proportion of nonwhites continued to increase relative to whites (Chase and Byrnes 1972, pp. 40-41). The data tell us nothing about smoking by *pregnant* women (or their spouses), but the evidence that does exist does not offer much support for the hypothesis that differential trends in cigarette smoking drove the divergence in birth weight distributions during the 1950s and early 1960s.

This period was also marked by a dramatic decline in the proportion of mothers who breast-fed their infants, and again, the magnitude of the change in behavior appears to have been larger for nonwhites than for whites. According to a large survey of ever-married women aged 15 to 44 in 1973 (Cycle I of the National Survey of Family Growth) regarding the feeding of their first-born infant, approximately 73 percent of (firstborn) nonwhite infants born before 1950 were breast-fed (for any duration) whereas only 14 percent of those born between 1966 and 1970 were (Hirschman and Hendershot 1979). For whites, the drop over the same period was from 56 percent to 29 percent. Using recent micro-level data, Forste, Weiss, and Lippincott (2001) have documented a strong correlation between breast-feeding and post-neonatal infant mortality even after controlling for race and low birth weight. If this correlation is truly causal (which is not demonstrated by Forste et al.), then the precipitous decline in breast-feeding among blacks might have propped up the nonwhite infant mortality rate through the mid-twentieth century.

Maternal Characteristics: Illegitimacy, Age, and Birth Order

The available evidence indicates that the mortality rate of "illegitimate" infants was higher than for other infants, a reflection of the socioeconomic characteristics of the mothers, as well as their age and attitude towards the pregnancy (Shapiro et al. 1968; Dept. of Health and Human Services 2000). To the extent that illegitimacy rates diverged across races, the racial infant mortality gap might have been influenced accordingly. The Vital Statistics coverage for this variable is limited both geographically and temporally, but the major post-1940 trends are readily discerned. In 1940, the nonwhite proportion of illegitimate births was 10 times the proportion among whites in the states reporting the variable (15.6 percent versus 1.55 percent). Both proportions increased slowly up to 1960 (18.5 percent versus 1.69 percent), and then increased sharply to 1970 (35.2 percent versus 5.45 percent).[31] Rough estimates of the elevation in neonatal mortality risk associated with illegitimacy suggest that the pre-1960 trends are unlikely to have had much effect on the racial infant mortality gap, but the post-1960 impact could have been substantial.[32] If so, then the convergence that did occur during the late 1960s is all the more remarkable.

Although illegitimate status may convey useful independent information about infant health (often signaling an unintended pregnancy), to a large extent, its influence reflects the age of the mothers. In particular, in the period under study young mothers (under 20 years old) were much more likely to deliver low birth weight infants than other women. Therefore, differential racial shifts in maternal age distributions could widen the infant mortality gap. Column 4 of table 8 reflects the impact of changes in mothers' age distribution (1950 to 1967) on the proportion of

[31] These figures are taken at face value from the Vital Statistics volumes, without adjustments for states entering and leaving the sample. A similar trend emerges when we use fixed samples of states.

[32] Shapiro et al. (1968, p. 66) cite figures from a study on New York City in the early 1960s. Setting the neonatal mortality rate for illegitimate nonwhite infants at 41 and for others at 25, the shift in the nonwhite proportion of illegitimate infants would have a small impact for 1940-1960, but a sizable impact from 1960-1970 (assuming a fixed mortality rate gap between illegitimate and other infants).

children with low birth weight, given the probability of low birth weight in each age category in 1950. For both nonwhites and whites, the impact is small (see "sum" rows). Column 6 captures the impact of changes in the probability of low birth weight, given the age distribution of mothers in 1950. For whites, the impact is again small, but for nonwhites the impact was relatively large, reflecting apparent increases in the likelihood of low birth weight in *every* age category for nonwhite mothers.

The risk of neonatal mortality may vary with birth order: in particular, at mid-century, high order infants (fifth and above) had elevated risks of death. Therefore, different racial distributions over the birth-order range might have influenced the racial gap in infant mortality rates. Again, however, table 9 demonstrates that in 1960 differences in mortality rates within birth-order categories were far more important than differences in the distribution of births across categories.

The Supply-Side of Healthcare: Access, Insurance, and Technology

An alternative set of hypotheses concentrates on the rapidly changing healthcare system. In particular, it is possible that the extension of the hospital system through the Hill-Burton program, the ascendance of private health insurance, and the emergence of new technologies did not have race-neutral impacts on infant mortality rates.

Did white infants benefit disproportionately from Hill-Burton funding of the hospital system's expansion? Although we cannot rule it out, the available evidence runs against the grain of this 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 and postpartum care, but they do suggest a rapid increase in hospital and

physician services for nonwhite infants. 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).[33]

A second major change in the healthcare system in this period was the rise of private health insurance, often provided as a benefit through one's employer (Thomasson 2001). 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 9 percent in 1940). By 1962, however, about 74 percent of whites had hospital insurance whereas only 46 percent of nonwhites did (Hoffmann 1964).

Did the racial insurance gap have implications for the quality of maternal and infant care?[34] In the absence of data on the treatment intensity of infants by race and insurance status, it is not possible to assemble a direct test of the hypothesis. However, it is worth noting that *within* weight classes (that is, roughly conditional on health at birth), the decline in neonatal mortality in the 1950s was larger for nonwhite infants than for white infants.[35] On the basis of such evidence, it would be difficult to argue that white newborn infants benefitted more from improvements in medical technology than nonwhite newborns, even with the insurance gap. Among post-neonates,

[33] In a recent paper on healthcare in the Mississippi Delta, however, Almond, Chay, and Greenstone (2001) argue that racial segregation in the hospital system was associated with lower quality care for blacks. This line of argument is not inconsistent with blacks' benefitting as much as (or even more than) whites from the system's expansion because the ex post racial gap in healthcare consumption, even with segregation, may have been considerably smaller than the pre-Hill-Burton gap.

[34] In a recent study of changes in Medicaid eligibility, Currie and Gruber (1997, p. 32) found evidence that "suggests that insured and uninsured populations have differential access to NICUs [neonatal intensive care units] and related interventions, and that this difference has real implications for health outcomes." Whether insurance had an infant health prior to the establishment of modern NICUs (in the 1960s) is an open question.

[35] Between 1950 and 1960, the neonatal mortality rate for nonwhites under 2,500 grams at birth fell from 164.7 to 154.8; for whites it increased from 175.8 to 177.4. For nonwhites over 2,500 grams, it fell from 11.9 to 7.7; for whites it fell from 7.1 to 5.1 (Chase 1972, p. 9).

the story might have been different. Improvements in post-neonatal death rates from influenza, pneumonia, other respiratory diseases, and gastro-intestinal diseases, all of which were treatable in hospitals, were not larger for nonwhite than for white infants during the 1950s even though nonwhites started with higher levels of mortality from these causes (Grove and Hetzel 1968).[36]

Institutional and technological changes might have been central to the relative improvement of nonwhite infant mortality rates in the late 1960s. First, Almond, Chay and Greenstone (2001) argue that the desegregation of southern hospitals, driven largely by a combination of federal Civil Rights legislation and Medicare certification requirements, played an important role in the racial mortality gap's decline in the South, particularly in rural areas. Second, according to Cone (1985), relatively rapid technological progress in the treatment of low birth weight infants began in the mid-1960s with the institution of modern NICUs. Given the relatively high proportion of low birth weight nonwhite infants, and supposing that financial or insurance constraints did not bar access to the new technology, such advances could have differentially benefitted nonwhite infants. Finally, a direct effect of Medicaid on the racial mortality gap, in addition to its indirect influence through facilitating hospital desegregation and (perhaps) access to new technologies, is certainly plausible and worthy of future research.

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. In this case, we focus on the racial gap in infant mortality rates between 1920 and 1970, a period which witnessed the

[36] The cause-of-death data must be interpreted with care because the residual category for nonwhites fell over time, implying that increases in certain disease categories may be due to more complete classification.

expansion of the healthcare system, the Great Migration of African Americans, the emergence of the Civil Rights Movement, and a significant and persistent gap in the mortality rates of 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. This finding suggests that either unobserved factors worked to offset the tendency toward convergence in infant mortality rates, or the relationship between the observed characteristics and infant mortality changed significantly after World War II. Comparing figures 3a and 3b, we concluded that the latter possibly seems less likely.

We then went on to investigate several hypotheses regarding factors that could have influenced the post-war gap. Proximately, the neonatal gap may have been strongly influenced by a leftward shift in the nonwhite birth weight distribution during the 1950s, but the postneonatal gap, which is rather insensitive to weight at birth, also failed to narrow. In any case, birth weight itself is an not an ultimate cause of infant mortality; rather, it is an intermediating variable between the ultimate causes and the mortality outcome. Digging deeper, we discussed trends in maternal characteristics, environmental quality, hospital births, smoking, breast-feeding, insurance coverage, and technology. Given the existing data constraints for the pre-1970 period, these trends are suggestive, but they are far from conclusive, and they remain promising avenues for more detailed historical and 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	3.450	3.900
	(0.481)	(0.404)
Ln Income	7.557	6.352
	(0.434)	(0.482)
Ln Education	2.346	2.114
	(0.119)	(0.259)
Ln Urban	4.049	3.991
	(0.327)	(0.521)
Ln Physicians	-6.689	-6.831
	(0.257)	(0.314)
N	478	344
Panel B: Summary statistics of state-year values		
Infant Mortality	35.71	54.03
	(19.59)	(25.87)
Income	2085	1124
	(806.5)	(655.1)
Education	10.52	8.549
	(1.194)	(2.030)
Urban	60.11	60.85
	(16.72)	(26.21)
Physicians	0.001286	0.001136
	(0.000329)	(0.00037)
N	478	344

Notes: Each observation represents a particular race/state/year cell. Observations are weighted by the number of births. 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	-0.1265	-0.1363	-0.0131	-0.1574	-0.1249	0.0680
	(0.0442)	(0.0623)	(0.0657)	(0.1068)	(0.0437)	(0.0807)
Education	-1.0199	-0.4508	-0.6918	-0.3418	-0.9659	-0.4577
	(0.1039)	(0.1092)	(0.2085)	(0.1312)	(0.1312)	(0.1094)
Urban	0.1046	0.2857	-0.0648	0.5729	0.1106	0.2330
	(0.0329)	(0.0591)	(0.0695)	(0.1297)	(0.0354)	(0.0570)
Physicians	-0.1332	-0.1842	-0.3601	-0.1636	-0.1314	-0.2210
	(0.0308)	(0.0527)	(0.0693)	(0.1071)	(0.0305)	(0.0544)
South	-----	-----	-----	-----	0.0129	0.1489
					(0.0193)	(0.0338)
1925	-0.1280	-0.1612	-0.1468	-0.1634	-0.1307	-0.1557
	(0.0313)	(0.0799)	(0.0325)	(0.0808)	(0.0318)	(0.0778)
1930	-0.2305	-0.3095	-0.2725	-0.2886	-0.2360	-0.2790
	(0.0302)	(0.0769)	(0.0318)	(0.0794)	(0.0315)	(0.0769)
1935	-0.3431	-0.5032	-0.4023	-0.5034	-0.3515	-0.4640
	(0.0351)	(0.0833)	(0.0408)	(0.0850)	(0.0366)	(0.0823)
1940	-0.4542	-0.5761	-0.5529	-0.5962	-0.4653	-0.5712
	(0.0428)	(0.0785)	(0.0487)	(0.0835)	(0.0436)	(0.0784)
1945	-0.5637	-0.7590	-0.7153	-0.8028	-0.5778	-0.8484
	(0.0487)	(0.0814)	(0.0608)	(0.0781)	(0.0508)	(0.0859)
1950	-0.8134	-0.9857	-0.9691	-1.0612	-0.8294	-1.0571
	(0.0492)	(0.0771)	(0.0636)	(0.0767)	(0.0508)	(0.0804)
1955	-0.8917	-0.9690	-1.0620	-1.0704	-0.9096	-1.0606
	(0.0478)	(0.0781)	(0.0706)	(0.0798)	(0.0523)	(0.0830)
1960	-0.8903	-0.9232	-1.0702	-1.0450	-0.9100	-1.0210
	(0.0496)	(0.0818)	(0.0752)	(0.0851)	(0.0554)	(0.0872)
1965	-0.8934	-0.9243	-1.0898	-1.0620	-0.9153	-1.0566
	(0.0570)	(0.0839)	(0.0858)	(0.0920)	(0.0630)	(0.0930)
1970	-1.0312	-1.1347	-1.2476	-1.2806	-1.0553	-1.3033
	(0.0643)	(0.0859)	(0.0966)	(0.1002)	(0.0707)	(0.0992)
Constant	6.1471	4.2068	3.8247	3.1985	6.0077	2.7701
	(0.4796)	(0.5366)	(0.7677)	(0.7669)	(0.5239)	(0.6700)
State Dummies	No	No	Yes	Yes	No	No
R^2	0.96	0.89	0.98	0.95	0.96	0.90
N	478	344	478	344	478	344
Mean Dep. Var.	3.450	3.900	3.450	3.900	3.450	3.900

Notes: State-race-year cells are weighted by the number of births. Robust standard errors are in parentheses.

Sources: See the text for description of the dataset.

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

	1920	1925	1930	1935	1940	1945	1950	1955	1960	1965	1970
Total Log Gap	**0.455**	**0.462**	**0.493**	**0.447**	**0.521**	**0.454**	**0.496**	**0.583**	**0.626**	**0.630**	**0.559**
Gap "Explained" by:											
Income	0.110	0.106	0.134	0.131	0.132	0.116	0.111	0.102	0.097	0.091	0.082
Education	0.337	0.293	0.343	0.329	0.307	0.265	0.223	0.166	0.121	0.093	0.069
Urban	-0.106	-0.079	-0.082	-0.069	-0.056	-0.037	-0.014	0.000	0.011	0.019	0.029
Physicians	0.034	0.027	0.034	0.038	0.042	0.037	0.029	0.022	0.016	0.012	0.007
Total "Explained" Gap	**0.375**	**0.347**	**0.429**	**0.429**	**0.425**	**0.381**	**0.349**	**0.290**	**0.245**	**0.215**	**0.187**
Total "Unexplained" Gap	**0.080**	**0.115**	**0.064**	**0.018**	**0.096**	**0.073**	**0.147**	**0.293**	**0.381**	**0.415**	**0.372**

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 coefficients from table 2, columns 1 and 2 and the difference in the variable's average value for whites and nonwhites (in that year).
Sources: See the text for description of the dataset.

Table 4: Infant Mortality Regressions, 1920-1945, 1950-1970, and 1920-1970

	White 1920-1970	**White 1920-1945**	**White 1950-1970**	**Nonwhite 1920-1970**	**Nonwhite 1920-1945**	**Nonwhite 1950-1970**
Income	-0.1265	-0.1486	-0.1106	-0.1363	-0.0666	-0.1849
	(0.0442)	(0.0689)	(0.0604)	(0.0623)	(0.1021)	(0.0698)
Education	-1.0199	-1.0014	-1.0238	-0.4508	-0.4283	-0.5182
	(0.1039)	(0.1302)	(0.1962)	(0.1092)	(0.1345)	(0.1850)
Urban	0.1046	0.1550	0.0730	0.2857	0.3444	0.1627
	(0.0329)	(0.0591)	(0.0405)	(0.0591)	(0.0790)	(0.0724)
Physicians	-0.1332	-0.2094	-0.1000	-0.1842	-0.2732	-0.0494
	(0.0308)	(0.0679)	(0.0326)	(0.0527)	(0.1045)	(0.0519)
Year Dummies	Yes	Yes	Yes	Yes	Yes	Yes
State Dummies	No	No	Yes	Yes	No	No
R^2	0.96	0.85	0.80	0.89	0.78	0.66
N	478	238	240	344	156	188
Mean Dep. Var.	3.450	3.950	3.102	3.900	4.374	3.674

Notes: State-race-year cells are weighted by the number of births. Robust standard errors are in parentheses.
Sources: See the text for description of the dataset.

Table 5: 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.

Table 6: Nonwhite Birth Weight and Neonatal Infant Mortality, 1950-1960

	1	2	3	4
	1950 Nonwhite Neonatal Mortality Rate	1950 Nonwhite Distribution	1960 Nonwhite Distribution	Col.1* (Col.3-Col.2)
< 1,000 g	821	0.006	0.010	3.28
1,000 - 1,500	507	0.009	0.011	1.01
1,501 - 2,000	196	0.020	0.025	0.98
2,001 - 2,500	50	0.068	0.083	0.75
2,501 - 3,000	15	0.214	0.253	0.59
3,001 - 3,500	10	0.354	0.371	0.20
3,501 - 4,000	11	0.228	0.189	-0.43
4,001 - 4,500	13	0.069	0.046	-0.30
> 4,500 g	20	0.033	0.013	-0.40
Sum		1.00	1.00	**5.68**

Notes: Column 4 reflects the impact of the change in birth weight distribution on the neonatal infant mortality rate.
Sources: Neonatal IMR is from Chase (1972, p. 9). Birth weight distributions are from Chase and Byrnes (1972, p. 41).

Table 7: Percentage of Live Births Under 2,500 Grams, by Region

	1950	1965
Nonwhite		
Mid-Atlantic	13.3	15.6
EN Central	12.7	14.5
WN Central	10.7	13.5
South Atlantic	9.8	13.7
ES Central	9.0	13.3
WS Central	9.2	13.8
U.S.	10.4	13.8
White		
Mid-Atlantic	7.4	7.3
EN Central	6.8	6.9
WN Central	6.3	6.3
South Atlantic	7.8	7.6
ES Central	6.9	7.3
WS Central	6.9	7.3
U.S.	7.2	7.2

Note: Low birthweight infants are born at under 2,500 grams (approximately 5.5 pounds).
Source: Vital Statistics of the United States.

Table 8: Age of Mother and Incidence of Low Birth Weight, 1950-1967

	1	2	3	4	5	6
	Percent LBW 1950	Age Dist. 1950	Age Dist. 1967	Col.1* (Col.3-Col.2)	Percent LBW 1967	Col.2* (Col.5-Col.1)
Nonwhite						
Under 15	14.7	0.007	0.010	0.044	19.5	0.034
15-19	12.0	0.206	0.269	0.756	15.7	0.762
20-24	9.6	0.326	0.324	-0.019	13.2	1.174
25-29	8.4	0.232	0.196	-0.302	11.8	0.788
30-34	8.8	0.132	0.116	-0.141	12.6	0.502
35-39	9.0	0.075	0.063	-0.108	13.3	0.323
40-44	8.9	0.020	0.020	0.000	12.2	0.066
45 and over	7.4	0.002	0.001	-0.007	10.8	0.007
Sum				**0.223**		**3.656**
White						
Under 15	15.9	0.001	0.001	0.000	12.5	-0.003
15-19	8.0	0.104	0.149	0.360	8.5	0.052
20-24	6.9	0.318	0.382	0.442	6.7	-0.064
25-29	6.5	0.297	0.257	-0.260	6.5	0.000
30-34	7.0	0.174	0.127	-0.329	7.0	0.000
35-39	7.5	0.084	0.065	-0.143	8.3	0.067
40-44	7.5	0.021	0.019	-0.015	9.1	0.034
45 and over	5.7	0.001	0.001	0.000	8.1	0.002
Sum				**0.055**		**0.088**

Notes: Column 4 reflects the impact of changes in the age distribution on the proportion of low birth weight infants (under 2,500 grams). Column 6 reflects the impact of changes in the likelihood of low birth weight within age categories on the overall proportion of low birth weight infants. Summing column 4 and column 6 gives the total change in the percentage of infants born with low birth weight.
Source: Data are from Chase and Byrnes (1972, pp. 17-18).

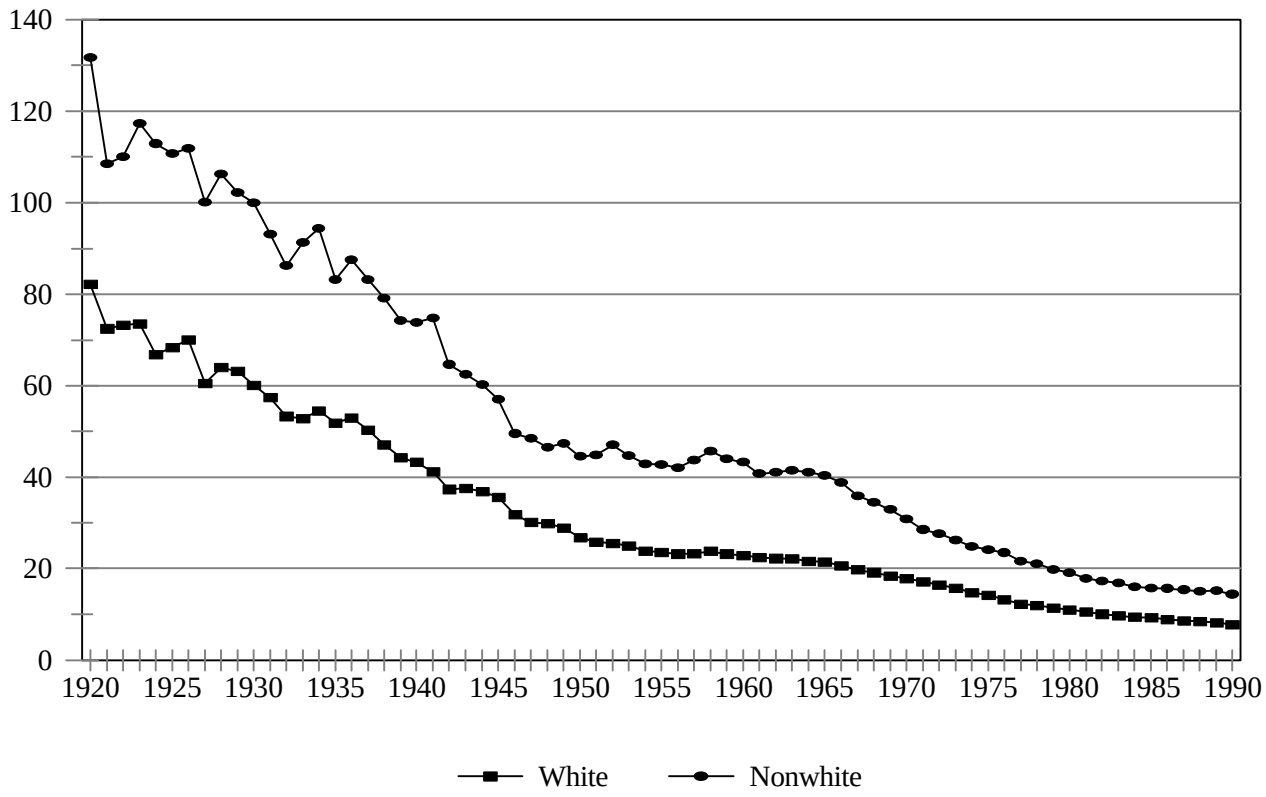
Table 9: Birth Order and Neonatal Mortality, 1960

	1	2	3	4	5	6
	White Neonatal Mortality Rate	Nonwhite Neonatal Mortality Rate	White Birth Order Distribution	Nonwhite Birth Order Distribution	(Col. 4 - Col. 3)* Col. 2	(Col. 2 - Col. 1)* Col. 3
First	15.0	25.7	0.262	0.210	-1.34	2.80
Second	16.1	27.2	0.248	0.183	-1.77	2.75
Third	15.9	25.8	0.196	0.153	-1.11	1.94
Fourth	17.2	25.4	0.127	0.121	-0.15	1.04
Fifth +	21.8	28.0	0.167	0.334	4.68	1.04
Sum					**0.31**	**9.57**

Notes: The table is based on a simple decomposition of the racial gap in neonatal mortality rates. Column 5 reflects the importance of differences in the birth order distributions conditional on mortality rates within categories. Column 6 reflects the importance of differences in mortality rates conditional on birth order. Using alternative weights for the decomposition (multiplying by column 1 rather than column 2 in column 5, and by column 4 rather than column 3 in column 6) would yield a sum of 0.95 in column 5 and 8.85 in column 6.

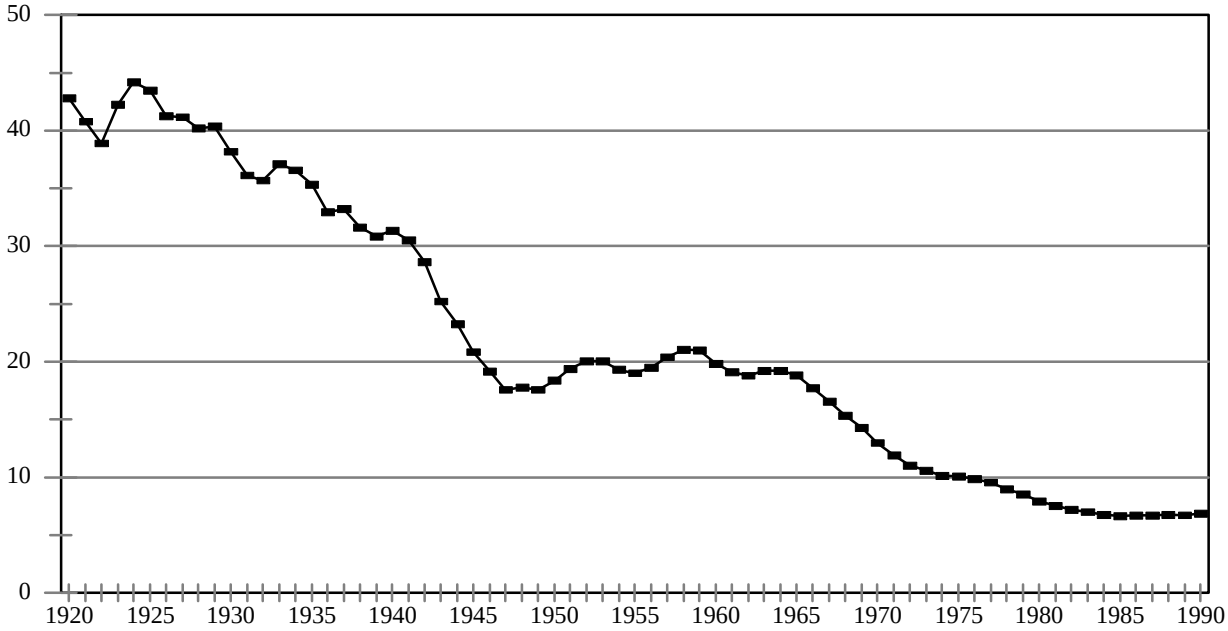
Source: Underlying data are from Chase (1972, p. 27).

Figure 1: Infant Mortality Rates, 1920 - 1990



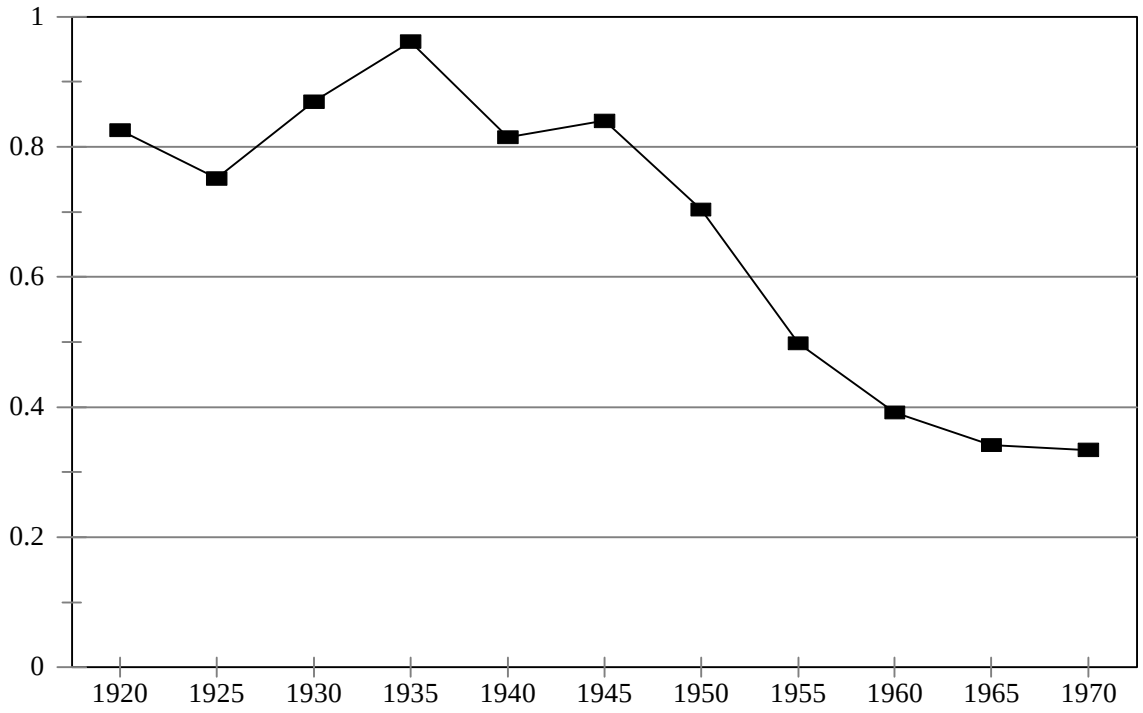
Notes: The area of coverage changes over time as states joined the birth- and death- registration areas. The series include Alaska from 1959, Hawaii from 1960, and exclude New Jersey in 1962 and 1963 (missing data by race). From 1932 to 1934, the series count Mexican-Americans as nonwhite, but in other years they are counted as white.
Sources: Compiled from Linder and Grove (1943) and Vital Statistics of the United States (various years).

Figure 2: The Racial Gap in Infant Mortality Rates



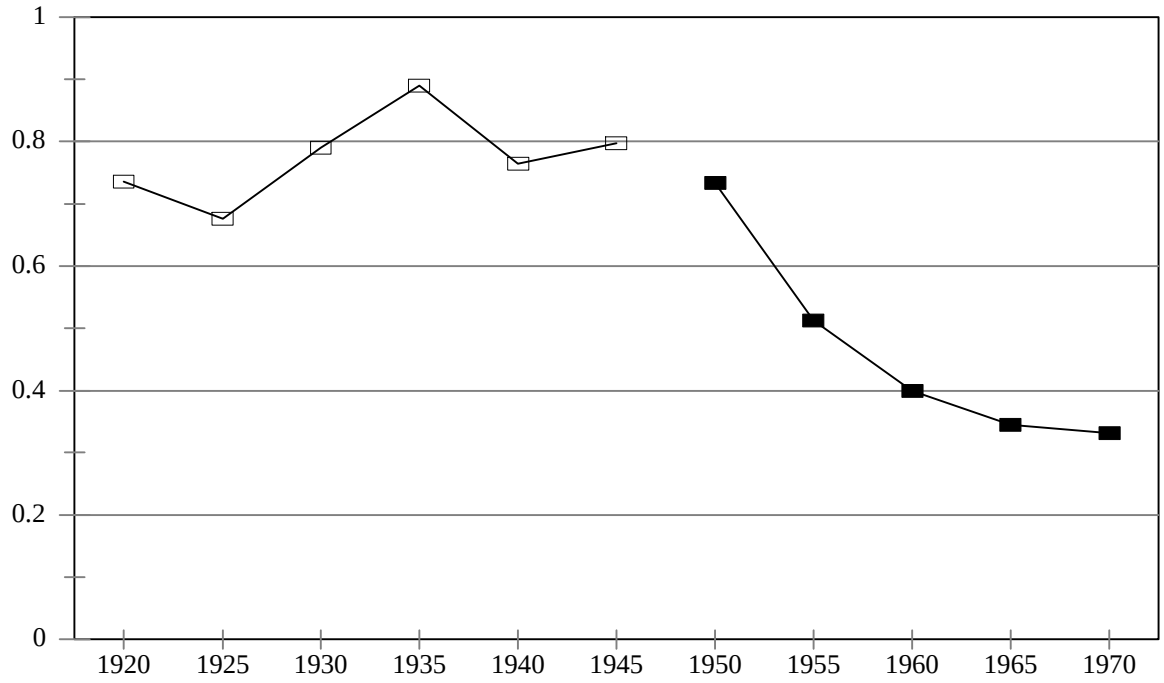
Notes: The graph plots a three-year moving average of the nonwhite-white gap in infant mortality rates.
Sources: Compiled from Linder and Grove (1943) and Vital Statistics of the United States (various years).

Figure 3a: Proportion of Infant Mortality Gap Accounted for by Covariates, 1920-1970



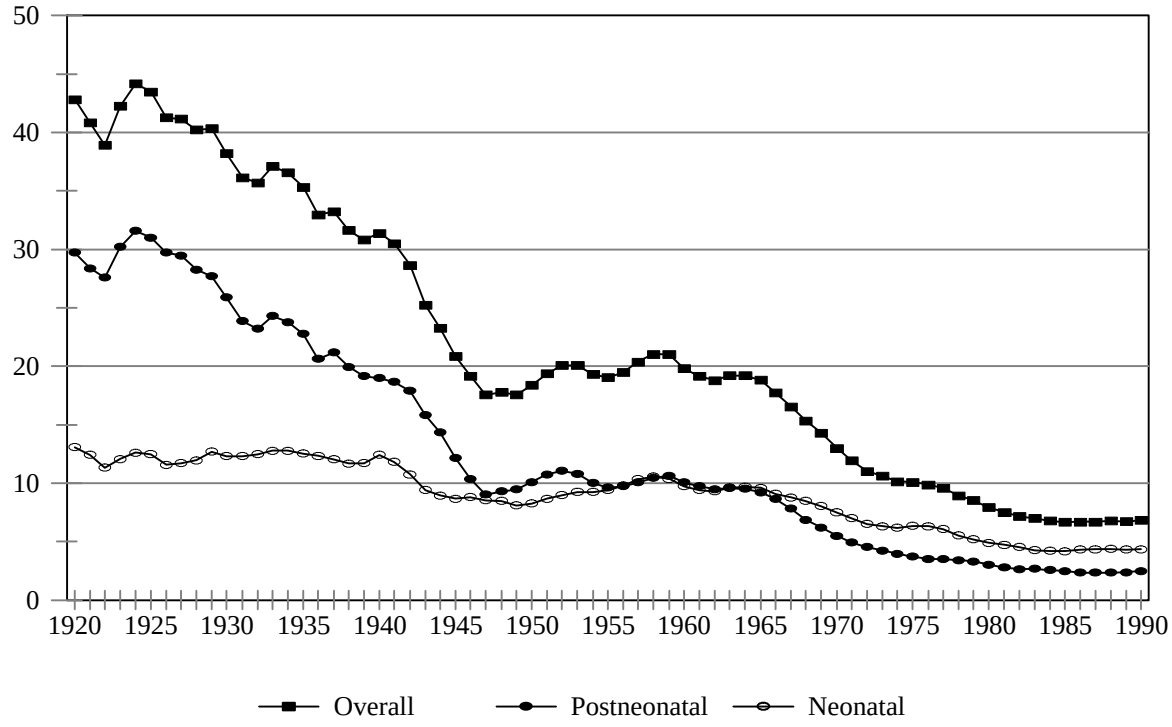
Notes: The "accounted for" part of the gap is the product of the racial gap in observed characteristics and the simple average of the regression coefficients for whites and nonwhites. The regressions were estimated with data covering the full 1920 to 1970 period. The "proportion" is then the "accounted for" part divided by the total gap at each point in time.
Sources: See the text for a description of the dataset and the regression coefficients.

Figure 3b: Proportion of Infant Mortality Gap Accounted for by Covariates, 1920-1945 and 1950-1970



Notes: The "accounted for" part of the gap is the product of the racial gap in observed characteristics and the simple average of the regression coefficients for whites and nonwhites. Regressions were estimated separately for the 1920 to 1945 and the 1950 to 1970 periods. The proportion is then the "accounted for" part divided by the total gap at each point in time.
Sources: See the text for a description of the dataset.

Figure 4: The Racial Gap in Infant Mortality Rate, Neonatal and Postneonatal



Note: The graph plots three-year moving averages. Not all states are covered until 1933.
Source: Vital Statistics of the United States, various years.