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The Long-Run and Intergenerational Impact of Poor Infant Health: Evidence from Cohorts Born During the Civil Rights Era*

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ABSTRACT

The 1960s witnessed the greatest reduction in the mortality rates of black infants relative to white infants of the last 50 years. We use these dramatic relative changes in the health of black birth cohorts to evaluate the long-run effects of early life health conditions. The analysis compares differences in the adult health and birth outcomes of black and white women born in the late 1960s to those of women born in the early 1960s, while controlling for age and year effects.

Black women born in the late 1960s have substantially lower risk factor rates as adults and are much less likely to give birth to an infant with low birth weight and APGAR scores than black women born in the early 1960s. The between-cohort gains for white women are small to non-existent, consistent with the smaller health improvements for white infants born during the 1960s. The timing of the black-white relative birth cohort improvements corresponds with the timing of the 1960s infant health gains and is robust to several tests of internal validity. For example, the between-cohort relative gains are significantly larger for black women born in Mississippi than for black women born in Alabama, exhibiting patterns much like the patterns in relative infant mortality rates in the two states during the 1960s. We conclude that the social policies that led to the infant health improvements had long-run and intergenerational health benefits. Also, studies of changes in racial health disparities over the life-cycle (and over time) could be severely biased by not accounting for the significant changes in the health of black birth cohorts.

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Introduction

The 1960s witnessed the greatest reduction in the mortality rates of black infants relative to white infants of the last 50 years. Appendix Figure 1 shows trends in nonwhite and white infant mortality rates within a year of birth (per 1,000 live births) for the United States from 1950-1990 as well as the nonwhite-white infant mortality rate (IMR) ratio.[1] Since 1950 there has been a secular increase in the black-white IMR ratio with one notable exception. From 1964 to 1971 the black infant mortality rate and the black-white ratio declined sharply relative to pre-existing trends. The black infant mortality rate fell 30 percent from 41 per 1,000 live births in 1964 to 28 in 1971, and the black-white ratio fell from 1.9 to 1.65, the only prolonged convergence in the post-World War II era.

Almond, Chay and Greenstone (2001) find that while the black-white IMR gap narrowed in all regions of the United States, the convergence rates were greatest in the rural South and varied substantially within the South. For example, Appendix Figure 2 shows trends in black and white post-neonatal mortality rates from 1941-1971 for Mississippi, Alabama, Illinois, and New York; states with large African-American populations.[2] In 1965 the black post-neonatal death rate in Mississippi was 26 per 1,000 live births -- 30 percent greater than the black rate in Alabama, 2-2.5 times greater than the black rates in Illinois and New York, and five times the rates for white infants in all four states. However, Mississippi also experienced the sharpest decline in black post-neonatal mortality after 1965, with the black infant death rate falling over 50 percent from 1965 to 1971. Almond, Chay and Greenstone (2001) argue that these dramatic changes in Mississippi were largely the result of the federal antidiscrimination effort of the mid-1960s.

These figures suggest that there were large improvements in the early health of black cohorts born during the 1960s relative to their white counterparts, and that the relative gains varied significantly across the U.S. This study uses these dramatic relative changes to evaluate the long-run and intergenerational effects of early life health conditions. In particular, we examine whether African-Americans born in the late 1960s have better health as adults and healthier infants in the 1980s and 1990s

[1] The data are from the Vital Statistics of the United States annual publications. In 1965, black births accounted for 92 percent of all nonwhite births in the U.S.

[2] Conventionally, post-neontal mortality is defined as the death rate in the period from 28 days to 1 year after birth.

than blacks born in the early 1960s. A finding of a long-run and intergenerational link has striking implications. First, it suggests that the causes of the 1960s infant health improvements (e.g., social policy) had multiplier effects that have been unaccounted for in cost-benefit calculations. Second, it would provide an alternative explanation to racial differences in behavior, medical care access, and treatment for the existence of racial health disparities. Observed black-white differences in adult and infant health could partially be the result of racial inequality in the health the adults had as infants.

The data requirements for a precise analysis of adult and infant health outcomes across narrowly-defined race, state and year of birth cells are enormous. Thus, we use the microdata contained in the 1979-2000 Natality Detail files, which are based on a census of all birth certificates in the U.S. These files provide information on the characteristics and health risk factors of the 34 million mothers born in the U.S. between 1955 and 1975 who gave birth during the 1980s and 1990s, as well as information on the birth outcomes of their infants. We link these data to the infant health conditions that prevailed in the state and year in which the mother was born. The analysis compares differences in the health and birth outcomes of black and white mothers born in the late 1960s to those of mothers born in the early 1960s. A well-known identification issue is that age, birth cohort, and survey year are collinear. To address this, we use flexible controls for mother's age and year in which she gave birth -- e.g., interactions of a cubic polynomial in age with unrestricted survey year effects -- and examine whether the estimated birth cohort effects exhibit trend breaks that correspond with the breaks in infant mortality during the 1960s.

We find mother's birth cohort effects that are very large in magnitude, particularly among African-American women. Table 1, for example, presents the estimated effects for the incidences of birth weight less than 1500 grams (first four columns) and less than 1000 grams (last four columns) among the infants of women who were born in the United States during the 1960s.[3] The regressions include birth cohort dummies, dummies for 2-year age categories of the mother, marital status, educational categories, and survey year and allow each of their effects to vary by race. The first two columns show that when race-specific birth cohort dummies are not included, it appears that the racial gap in very low birth weight

[3] Low birth weight is conventionally defined as a weight less than 2500 grams. Birth weight less than 1000 grams (2.2 pounds) is strongly associated with eventual infant death (Almond, Chay and Lee 2002).

(VLBW) grows significantly as mother's age, with teenage black mother's experiencing the best birth outcomes.

These findings change dramatically when mother's birth cohort is accounted for. In the next two columns, teenage black women are now more likely to have a very low birth weight infant than 24-25 year old black mothers. The conclusions from the results for extremely low birth weight incidence are similar. Further, the across-cohort differences in birth outcomes for black women are comparable in size and often larger than the estimated effects of age, marital status and education. Finally, black mothers born in the late 1960s have significantly better birth outcomes than black women born in the early 1960s, while there are no across-cohort improvements among white women. Thus, the improving health of black infants born in the 1960s seems to be mirrored by improved birth outcomes among black women born in the 1960s, who gave birth in the 1980s and 1990s.

This study finds that black women born in the late 1960s have substantially lower risk factor rates (e.g., diabetes and hypertension) as adults and are much less likely to give birth to an infant with low birth weight or low APGAR scores than black women born in the early 1960s. The between-cohort gains for white women are small to non-existent, consistent with the smaller health improvements among white infants born during the Civil Right Era. The timing of the black-white relative birth cohort improvements corresponds with the timing of the 1960s infant health gains and is robust to several tests of internal validity. For example, consistent with the early life health conditions hypothesis, we find that there are no across-cohort improvements in the outcomes of black women who were born outside the United States. In addition, we find that genital herpes rates – a maternal risk factor that is likely attributable to current behavior and cannot be plausibly linked to health as an infant – are higher among younger than older black birth cohorts. Finally, the between-cohort relative gains are significantly larger for black women born in Mississippi than for black women born in Alabama, exhibiting strikingly similar patterns to the patterns in relative infant mortality rates in the two states during the 1960s.

We conclude that the War on Poverty social policies that led to infant health improvements (e.g., hospital desegregation, Medicaid, Food Stamps) may have had long-run and intergenerational health benefits. Also, studies of changes in racial health disparities over the life-cycle and over time could be

severely biased by not accounting for the significant changes in the health of black birth cohorts. It appears that racial disparities in health at and soon after birth are associated with health inequalities later in life and in the subsequent generation.

Motivation and Background

In this section, we describe the federal interventions that could have induced the sharp reductions in the black-white infant mortality gap during the 1960s. We also summarize the literatures on racial health disparities and the long-run effects of early life conditions that help to motivate examining relative changes in adult and intergenerational health outcomes across birth cohorts of African-Americans. Finally, we discuss how our research design may reduce the role of omitted variables bias that could plague previous studies of the effects of early life conditions.

Black-White Infant Mortality and Federal Interventions in the 1960s

Figures 1 and 2 show that the late 1960s are the key period for improvements in the relative health of black infants over the past 50 years. The mid-1960s also witnessed a dramatic shift in federal policies regarding access to medical care, which could explain the significant convergence in black-white infant mortality rates after 1964. Health care expenditures accounted for the largest and fastest-growing share of the War on Poverty and Great Society programs (Davis and Schoen 1978). Major initiatives to improve the health of poorer people, such as the Medicaid program, were initiated in the mid-1960s, leading to dramatic changes in the provision of health services. Two of the more notable federal interventions though were Title VI of the 1964 Civil Rights Act and the 1963 and 1965 expansions to the maternal and infant care component of Title V of the 1935 Social Security Act.

Title V of the 1935 Social Security Act established the Maternal and Child Health (MCH) Services Program. The Maternal and Infant (M&I) Care component of MCH targeted federal dollars to improve the health of mothers and infants from families with low income levels and diverse racial and ethnic heritages and those living in rural areas without access to care. In 1963 and 1965, amendments to Title V resulted in dramatic increases in MCH funding of maternal and infant care projects (Davis and

Schoen 1978). An expressed purpose of the amendments was to reduce infant mortality rates among the poor in central cities and rural areas by improving prenatal and postpartum care. Although the MCH program attempted to allocate more funds to states with low incomes, "Southern states with high incidence of poverty and large rural populations, such as Mississippi, Louisiana, Texas, and Georgia, received one-fourth to one-tenth the average expenditure per poor child of [certain areas in the North]" (Davis and Schoen 1978:147-148).

Title VI of the 1964 Civil Rights Act prohibited discrimination and segregation in institutions receiving federal financial assistance, including all public hospitals. One goal of Title VI was to eliminate racial discrimination in access to medical care, particularly in the South. Although the original enforcement of Title VI by the Department of Health, Education, and Welfare was weak and disorganized, there is a consensus that the 1965 Medicare Act gave Title VI real bite. First, the Act withheld Medicare certification and funding from all hospitals that could not provide evidence of integrated facilities and equality of care. Second, Title VI enforcement was now under the purview of the Office of Equal Health Opportunity (OEHO) in the Surgeon General's Office, which took an aggressive approach to auditing hospitals in the South (Smith 1999:128). The combination is believed to have resulted in a dramatic integration of Southern hospitals in the last half of 1966.

Almond, Chay and Greenstone (2001) find that the integration of hospitals played a causal role in the decline in black infant mortality in Mississippi during the 1960s. A key piece of supporting evidence is the finding that the reduction in black post-neonatal death shown in Figure 2 was driven by a remarkable decrease in causes of death considered preventable by medical treatment, such as diarrhea and pneumonia. In addition, this reduction was concentrated in the Mississippi counties that contained the most racially segregated hospitals before the passage of Title VI. We return to a discussion of the various programs implemented during the 1960s before the conclusion.

Reasons for Racial Health Disparities and Policy Implications

The large racial disparities in health in the United States are well-established. For example, today African-Americans are twice as likely as white Americans to die from heart disease and 34 percent more

likely to die from cancer. The racial inequality in infant health is stark as well, with black infants dying at 2.5 times the rate of white infants within a year of birth and experiencing even larger disparities in the incidences of low (less than 2500 grams) and very low (less than 1500 grams) birth weights.

By contrast, the underlying causes of these disparities are not well understood. The literature has examined the roles of: 1) racial differences in behavior and lifestyles -- e.g., tobacco, alcohol, and drug use, diet, exercise, and obesity; 2) racial differences in access to medical care, due to income and socioeconomic stratification, racial discrimination or segregation, differential technology diffusion, and geographic segmentation; and 3) racial differences in the quality of care and the treatments prescribed conditional on access (e.g., Schulman et al., 1999). However, these studies have found large black-white gaps in outcomes even after controlling for observable measures of these differences. With respect to infant health, studies have found that a wide variety of risk factors explain only a small portion of the racial disparity in adverse birth outcomes.[4] For example, although college-educated black and white women have similar prenatal care usage patterns and similarly low rates of tobacco and alcohol use during pregnancy, there remain large disparities in the incidences of very low birth weight and infant mortality (Schoendorf, et al. 1992).

We examine another potential explanation for these anomalous health inequalities. Differences in the early health conditions faced by black and white infants could result in health differences as these infants age into adulthood and in differences in the birth outcomes of their children. Schoendorf, et al. (1992) hypothesize that the poorer birth outcomes among college-educated blacks may partially reflect the poorer health these women had as children. As we discuss below, studies have found an association between health at birth and health as an adult. In addition, it is widely accepted that racial differences in women's health can result in disparities in birth outcomes (Geronimus 1996). Thus, the poor birth outcomes among well-educated black women could plausibly be due to an intergenerational pass through (e.g., Emanuel, Hale, and Berg 1989).[5]

[4] The risk factors studied include maternal age, socioeconomic status, marital status, parity, smoking, mental health, alcohol and substance use, adequacy of prenatal care, and genetics (e.g., Kleinman and Kessel 1987, Kempe et al. 1992).

[5] In an interview for the NewsHour on PBS, Dr. David Satcher – Surgeon General of the United States from 1998-2002 – discusses these potential intergenerational links. He states, "… even for the higher socioeconomic group

The existence of long-run and intergenerational effects of differences in initial health has striking implications. First, it suggests that policies that impact early health have additional health benefits in the future and in future generations that are not accounted for in conventional cost-benefit analyses. In the context of this study, it implies that the antidiscrimination and social policies of the War on Poverty had substantial multiplier effects. Second, pre-existing racial disparities in health arising from unequal access to medical care or differential exposure to poor conditions could result in disparities in the next generation, suggesting that infants do not start on a level playing field with respect to health at birth.

Finally, studies of changes in racial health disparities over the life-cycle and over time could be severely biased by not accounting for significant changes in the health of black birth cohorts. For example, based on the observation that racial inequality in birth outcomes is lowest among teenage mothers and greatest in young and middle adulthood, some have concluded that African-American women experience greater health deterioration as they age than white women (a.k.a., the weathering hypothesis).[6] This literature concludes that the optimal age for childbearing occurs at a younger age for black than for white women. As Table 1 and Figure 3 show, it appears that this conclusion may be an artifact of better health at birth among younger than older cohorts of black women. The results are very consistent with the weathering hypothesis when mother's birth cohort is not controlled for. However, when one accounts for mother's year of birth, black teenagers now have the worst birth outcomes and the optimal age for childbearing is similar by race.

Literature on Early Life Health Conditions

It is well known that environmental conditions affect health and mortality. This effect is thought to be strongest during the earliest periods of life, when growth is most rapid. Rather than being temporary effects which dissipate over time, it has been argued that early environmental conditions have

there are still disparities … We believe that some of these are multigenerational to the extent that some experiences that people have as children, especially girls who grow up to be women, of course, affect the outcome of their pregnancies." (transcript at http://www.pbs.org/newshour/bb/health/jan-june02/satcher_1-21.html)

[6] The "weathering hypothesis" (Geronimus 2001) posits that the significant worsening of health among black women is the result of the cumulative impact of repeated exposure to social, economic, and political stressors and barriers. "Allostatic load", an index for the long-term effect of repeated physiological response to stressors, provides the biological basis for the weathering hypothesis.

permanent effects on health. Particularly during the critical period of fetal development, the body may be "programmed" for susceptibility to disease later in life (Barker 1998). When the fetal environment is unfavorable, a triage in the nutrient supply occurs where the brain is given priority over other organs, such as the heart, which can suffer permanent damage as a result. These injuries manifest themselves later in life in increased morbidity and accelerated mortality.[7] Animal and epidemiological studies generally confirm that such early health injuries can manifest themselves later in life. A 2001 *British Medical Journal* editorial states that the Barker linkage is "no longer just a hypothesis".

Much of the epidemiologic evidence supporting the "Fetal Origins" hypothesis comes from analyses of micro data that link birth records to adult health outcomes. This literature has found a significant association between infant birth weight and the development of chronic diseases in adulthood, such as diabetes, hypertension, and cardiovascular disease.[8] For example, several studies have documented a strong correlation between infant birth weight and adult death due to ischaemic heart disease (Barker, et al., 1989, Vagero and Leon 1994). Research in the economics literature has also found that low birth weight is correlated with lower educational attainment, poorer self-reported health status, and reduced employment and earnings among adults.[9] However, for reasons discussed below, some have seriously questioned the causality of these documented birth weight associations.[10]

Social scientists have also explored this proposition in a number of population-based cohort studies (see literature reviews by Elo and Preston 1992 and Smith 1999). Leading work in this vein is by Barker, who has studied the association between health conditions in British localities between 1901-

[7] For example, it is widely accepted that acute rheumatic fever, an upper respiratory infection, among infants and children will cause permanent damage to the valves in the heart if left untreated by antibiotics. This damage may become apparent later in life as rheumatic heart disease and lead to premature death as an adult. This was a common occurrence among the generation born before the widespread use of antibiotics in the late 1930s and 1940s.

[8] See Barker (1992), Barker, et al. (1989, 1993), Vagero and Leon (1994), Leon, et al. (2000), and Innes, et al. (2002). Rasmussen (2001) provides a review of the literature.

[9] See, for example, Behrman, Rosenzweig, and Taubman (1994), Currie and Hyson (1999), and Behrman and Rosenzweig (2001).

[10] Comparing twins to singletons, Williams and Poulton (1999) and Christensen, et al. (1995) find no evidence of an effect of birth weight on either blood pressure or adult mortality, respectively. Phillips, et al. (2001) summarizes the studies of the effects of twins' birth size on long-run morbidity and mortality. Susser and Levin (1999) and Kramer (2000) provide commentary questioning the causal effect of birth weight. Rasmussen (2001) discusses several quasi-experiments, including Stein, et al. (1975), that provide mixed empirical evidence on the fetal origins hypothesis.

1910 and mortality from various causes in these same regions between 1968 and 1978. He finds that neonatal mortality rates, which can reflect fetal nutrition, are associated with subsequent mortality from stroke and that post-neonatal mortality (mortality from 1 month to 1 year of age and tied to the early pathogen environment) is strongly correlated with subsequent death from heart disease.

Costa (2000) uses data on military veterans to look at the effect of disease exposure in childhood and during military service on subsequent health outcomes. She concludes that reduced rates of infectious disease early in life may help account for the increase in the survival rate of older men during the 20th Century. Similarly, a recent Penn State study (Hayward, et al., 2001) using the National Longitudinal Study of Older Men finds that after accounting for demographic differences, men's mortality is affected by childhood conditions.

Finally, Case, Lubotsky, and Paxson (2001) focus on the link between economic status and health among children. Children from low-income families experience more chronic health conditions which become more pronounced as the children near adulthood. Income offers a protective effect in wealthier families that mitigates the impact of chronic health conditions. The authors argue that the SES-health gradient thus has its origins in childhood, and that the intergenerational transmission of socioeconomic status may occur in part as a result of the inferior health path of children from poorer families.

Research Design Issues

Several factors impede convincing research on the long-term effects of conditions in utero and during early infancy. First, longitudinal data that include information on initial health conditions and subsequent morbidity and mortality are scarce. For example, Costa uses the size of the serviceman's city of residence and city of enlistment as a proxy for the health conditions servicemen encountered early in life. Hayward, et al. only have information on childhood conditions at age fifteen and not on the conditions of early infancy that are thought to be most influential. Case, et al. also cannot link childhood health conditions to adult morbidity and mortality. Second, there may be substantial slippage in matching early health conditions to the outcomes of the adults presumed to be affected. For example, the enlistment city or city of residence Costa uses in assigning childhood health conditions may differ from

where servicemen were born, and approximately half of those in Barker's sample moved from their region of birth over their lives (Elo and Preston 1992).

Finally, and most importantly, convincing analysis requires variation in early health conditions that is not confounded by other factors that might also affect adult health, such as income and family background. Since infant birth weight is strongly correlated with the race, socioeconomic status, and behavior of the mother during pregnancy, the longitudinal micro studies establishing birth weight effects may be severely biased by omitted variables. For example, Almond, Chay, and Lee (2002) find that much of the observed association between birth weight and eventual infant death can be attributed to genetic and other unobserved influences. With respect to the cohort studies, Barker's raw geographic correlations utilizing only place of birth may also suffer from omitted variables bias. Similarly, Costa does not use variation in infant health measures across birth cohorts.

In the absence of a convincing research design, previous research may be documenting correlations that are artifacts of other influences that vary with early health conditions.[11] This study, on the other hand, attempts to address several of these issues. First, we can directly link proxies for the infant health conditions that prevailed to the actual place and year in which the mother was born, separately for black and white women. Further, we use the differential changes in black and white infant health across states of birth during the 1960s to mitigate the role of omitted variables. For example, Appendix Figure 2 suggests that the across-cohort improvement in the relative health of black infants was significantly greater in Mississippi than in Alabama. Also, it seems unlikely that there were differential improvements in the family backgrounds and home environments of these infants.[12] Thus, we test the hypothesis by examining whether black women born in the late 1960s have better health and birth outcomes in the 1980s and 1990s than black women born in the early 1960s, and whether these across-cohort improvements are greater than those for white women. We also test whether the across-cohort relative gains are greater for Mississippi-born black women than for their Alabama-born counterparts.

[11] It is worth noting that the Economics literature examining the intergenerational correlation in income and wealth suffers from similar omitted variables issues.

[12] In fact, Almond, Chay and Greenstone (2001) find that proxies of family background, such as age and marital status of the mothers giving birth, actually worsened for black infants relative to white infants during the 1960s.

Conceptual Framework

Our study explores the long-run effects of 1960s infant health changes on maternal health and infant health of the subsequent generation. Unfortunately, the infant health of individual mothers is not observable, nor is it observed for her birth cohort at large. Instead, we only have information on the infant mortality rates for a mother's particular birth cohort. At first pass it may not be apparent how infant mortality rates register cohort health. However, infant mortality rates respond predictably to changes in the unobserved distribution of cohort health. Moreover, the infant mortality proxy has the appealing feature that it biases results against finding positive long-term effects of improved infant health, as measured by decreases in infant mortality.

Infant mortality rates for a given birth cohort reflect two distinct pieces of information: a) the unobserved distribution of initial cohort health, and b) the health threshold which must be exceeded in order for newborns to survive infancy.

The effect on cohort health generated by changes in the infant mortality threshold (b) is perhaps the more intuitive determinant of mortality. Infants who survive a particular mortality threshold will have better unobserved health than those infants who died. If the infant mortality rate is high because more infants of marginal health are dying, infants who survive infancy will be especially healthy. To the extent that this health threshold effect is at play, we would expect that cohorts exposed to higher early-life infant mortality rates to be more positively selected and therefore in better initial health. If intertemporal health linkages exist, these cohorts will also be observed to have better health as adults. In contrast, if infant mortality rates fall because fewer infants of marginal health are dying (that is, the health threshold for infant survival has become less stringent), then surviving infants will be in poorer average initial health. To the extent that such a selection effect generated the large observed decreases in infant mortality during the 1960s, intertemporal health linkages would cause these cohorts to be in *worse* health as adults.

Shifts in the unobserved distribution of initial cohort health will also generate changes in the infant mortality rate. If, for example, maternal nutrition improves, one might expect that unobserved fetal and infant health would improve. Fewer infants would fall below the threshold at which infant death

occurs, and the infant mortality rate would decrease. If intertemporal health linkages exist, these cohorts would be observed to have better health as adults. Albeit implicitly, improvements in the underlying health distribution are generally the focus of empirical work on long-term health linkages.

For this distributional effect of changes in cohort health to be observed, it must overwhelm the selection effect generated by changes in the survival threshold. This implies that estimated long-term health benefits due to improved fetal and infant health will be underestimated when infant mortality rates are used to proxy for cohort health due to the selection effect.

More formally, the two distinct types of information conflated in the infant mortality rate can be considered in a stylized latent variable model of initial health. Let h^*_i be the unobserved health of individual i which is fixed from birth. In the figure below, the probability distribution of h^*_i is given by the solid black line, with individuals in poor initial health being on the left and healthier individuals on the right. If h^*_i falls below a survival threshold d_0 (depicted in the figure by the leftmost vertical line), then the individual will die within the first year of life. Individuals with $h^*_i \geq d_0$ survive to adulthood. These adults will be physically disabled during the follow-up period if $d_0 \leq h^*_i < d_1$ – that is, there initial health falls between the two vertical black lines in the figure. Individuals suffer neither death nor disability if $h^*_i \geq d_1$.

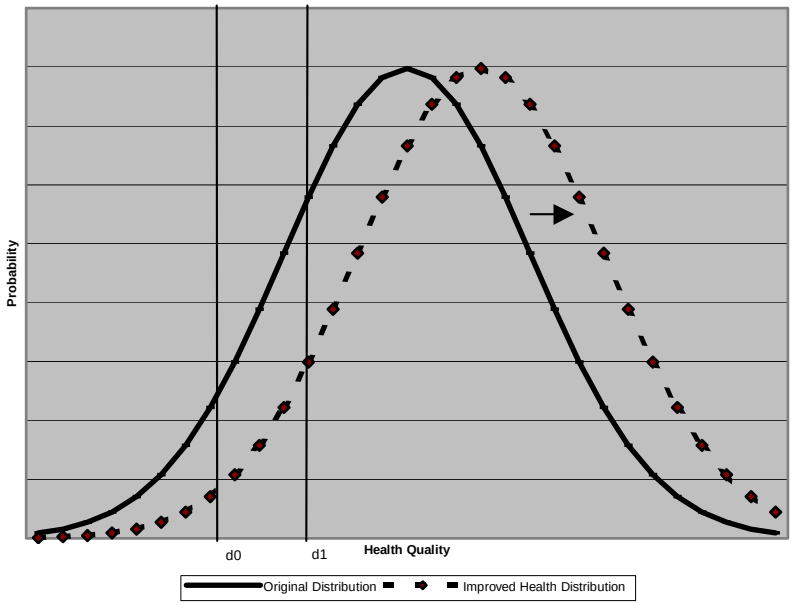
Given these health thresholds, the infant mortality rate (IMR) may be defined using the cumulative distribution function $F(h^*_i)$ as:

$$IMR \equiv F(d_0)$$

That is, the infant mortality rate is given by the share of the health distribution to the left of d_0. The adult disability rate (ADR) is given by the share of persons surviving infancy that have initial health below d_1:

$$ADR \equiv \frac{F(d_1) - F(d_0)}{1 - F(d_0)}$$

The Unobserved Distribution of Individual Health



Improvements in the probability distribution for health at birth, $f(h^*_i)$ (depicted in the figure above as an increase in the mean μ of the solid black distribution to the new dotted distribution) generate decreases in both the infant mortality rate and the adult disability rate. Therefore:

$$\frac{\frac{\partial ADR}{\partial \mu}}{\frac{\partial IMR}{\partial \mu}} > 0$$

The adult disability and infant mortality rates will move in the same direction when shifts in the probability distribution of unobserved health occur. Depending on the pdf chosen to model the distribution of initial health, the relationship between induced decreases in both IMR and ADR caused by shifts in the initial health distribution can be concave or convex. If $h^*_i \sim N(\mu,\sigma)$, then the relationship between IMR and ADR is convex.[13]

As described above, decreases in infant mortality may also occur due to reductions in the threshold at which infants survive, d_0. If improvements in birth practices and infant care (such as a

[13] In contrast, while decreases in the variance of normally-distributed unobserved health will also induce a positive relationship between IMR and ADR, this relationship will be concave.

transition from birth at home to birth in hospital) affect infant survival conditional on initial health, then decreases in infant mortality may also be substantially affected by leftward shifts in d_0. As:

$$\frac{\frac{\partial ADR}{\partial d_0}}{\frac{\partial IMR}{\partial d_0}} < 0$$

leftward shifts in d_0 (for a fixed d_1) will exert a countervailing effect on ADR by causing the expected value of health for adults at the follow-up period to deteriorate when the infant mortality rate falls. To the extent that infant mortality falls due to improvements in birth technologies or improved access to health care that does not affect the distribution of unobserved health, adult disability should *increase* as a result. Therefore, for improvements in the underlying health distribution to be apparent empirically when the infant mortality rate falls, they must overwhelm the selection effect on adult disability risk.

Data Description

This study requires health measures for those black and white adults who were born in the United States during the 1960s, as well as measures of the birth outcomes of their newborns. Further, our research design entails an examination of these adult and infant health outcomes across narrowly-defined race, state, and year of birth cells. Thus, the data requirements for a precise analysis are enormous. To address these needs, we use data derived from U.S. birth certificates.

The primary datasets used in the analysis are the 1979-2000 natality microdata produced by the National Center for Health Statistics (NCHS). These files provide detailed information on the *universe* of births occurring each year in the United States as reported on birth certificates. Among the analysis years, 1986 had the fewest births (with 3,760,997) and 1990 the most (with 4,163,150). In total, information on 75 million births is available across the survey years.

For births occurring between 1985 and 1991, the linked birth/infant death mircrodata are used. The linked files contain detailed information on each live birth occurring in the United States, and match it to any corresponding vital statistics record of subsequent infant death (deaths during the first year of life). For the 1992 to 1994 data years, NCHS suspended linking of natality and mortality records – thus,

for births during these years, the unlinked natality detail microdata are used. For births between 1995 and 2000, the annual perinatal microdata are used. The perinatal files also contain the complete set of information on live births and link this information to corresponding infant and fetal death records.[14]

The natality portion of the microdata provides socioeconomic and demographic information for each mother giving birth in the United States. This information includes maternal age in years, race and ethnicity of the mother, educational attainment, marital status, and most importantly for this analysis, the mothers' place of birth. The state in which each mother was born is identified, and together with age, is used to group birth records into state and year of birth cells that are then linked to the infant mortality rates that prevailed in those cells. The natality files also identify births occurring in the United States among mothers who were themselves born outside the U.S. (Canada, Mexico, various U.S. territories, or the "Remainder of the World"). In the analysis sample, approximately four percent of births were to non-Hispanic white mothers themselves born outside of the United States. Approximately eight percent of non-Hispanic black mothers were foreign-born. Since these foreign-born women were not exposed to the infant health conditions that prevailed in the U.S., we use their outcomes as a test of the internal validity of our findings for U.S.-born women.

The natality portion of the microdata also provide detailed information on aspects of maternal health, characteristics of the pregnancy, as well as information on the initial health of the newborn infant. Information on maternal health is contained in seventeen "Medical Risk Factors," which NCHS began collecting with the expansion of the birth certificate form in 1989. These risk factors are described in greater detail below. Information on the gestation length and birth weight of the infant are available in each of the thirteen survey years. The five-minute APGAR score (also described below) is available for the years in which natality files are linked to the infant death files: 1983-1991 and 1995-2000.[15]

Not all of the 75 million natality records from the 1979-2000 survey years are used in the analysis. First, due to the focus of this study, only mothers with birth years between 1955 and 1975 are

[14] At present, the 1995-1997 *period* linked birth/infant death microdata files are used in the analysis. This means that only infant deaths that occur in the same survey year as the birth are linked to corresponding birth records. In future analyses, the birth *cohort* linked birth/infant death microdata will be used.

[15] APGAR score for births occurring between 1992-1994 will be available in future analyses. Currently, it is not provided on NCHS public-use files that use the SETS data-extraction software.

kept. Year of birth is constructed as the difference between survey year and maternal age. As age is only measured in integers, there will be measurement error in the year of birth. While there is information on the month in which the birth occurred during the 1979 to 2000 interval, without additional information on age, year of birth can still not be exactly determined.[16] Note, as mothers on average will be their reported age plus ½ a year, and because in a census of births for a given calendar year, births on average occur at the beginning of July, the year of birth constructed here will be centered at January 1st.

In addition, all multiple birth records have been excluded from the analysis. Such births constitute approximately two percent of all births. In future analysis, such births could potentially be included (and weighted by the inverse of plurality). Finally, all mothers with a Hispanic origin are excluded (regardless of race). We now describe the measures of maternal risk factors, the newborn's gestation length, birth weight, and APGAR score used in the analysis, along with the survey years in which they are available.

Birth Weight and Gestation Length (available 1979-2000)

In the analysis, birth weights are grouped according to weights less than 2500 grams (the conventional threshold for "low birth weight"), less than 1500 grams, and less than 1,000 grams. In 1991, approximately 7 percent, 1.5 percent, and 0.5 percent of births fell into these respective categories.

APGAR score (available 1979-1991, 1995-2000)

While birth weight is the most commonly used proxy for infant health, other health measures reported on the birth certificate also reflect an infant's health. In particular, we analyze five-minute APGAR score as an alternative initial health measure to birth weight. NCHS describes the APGAR score as a "predictor of the infant's chances of surviving the first year of life" and a "summary measure of the infant's condition" (NCHS *Vital Statistics* Technical Appendix, 1990). Almond, Chay, and Lee (2002) find evidence that the five-minute APGAR score is a better predictor of infant health and mortality than

[16] For 1989 on, the restricted-access versions of the natality microdata include the month, day, and year of maternal birth. Subject to a review of their confidentiality policy, NCHS will be providing new natality files for these birth years that include mothers' exact year of birth for the purposes of this project.

birth weight. APGAR scores range from 0 to 10 and are calculated from five separate tests of newborn health made both one and five minutes after birth. The five component factors are each scored a 0, 1 or 2, and then summed to calculate the APGRAR score. The five health factors are:

- Heart Rate
- Respiratory effort
- Muscle tone
- Reflex irritability
- Color

In 1991, five minute APGAR score was reported on approximately 99 percent of birth certificates. Reporting rates were lowest (with around six percent missing) in Connecticut and Oklahoma. For the analysis, APGAR scores are grouped in the following ranges: less than 9, less than 8, and less than 7. In 1991, approximately 10 percent, 3 percent, and 1.5 percent of births had APGAR scores in these respective ranges.

Risk Factors (available 1989-2000)

Beginning in 1989, seventeen "Medical Risk Factors" for the mother were collected as part of the expanded birth certificate. The reported factors are: Anemia, Cardiac Disease, Diabetes, Genital Herpes, Hydramnios/Oligohydramnios, Hemoglobinopathy, Hypertension (chronic), Hypertension (pregnancy-associated), Eclampsia, Incompetent cervix, Previous infant 4,000+ grams, Previous preterm or small-for-gestational-age infant, renal disease, Rh sensitization, Uterine bleeding, other medical risk factors.

In 1991, approximately ninety-six percent of birth certificates had complete information on the medical risk factors. Generally, reporting has improved over time, although there is substantial variation in reporting by the state in which the 1989-1997 birth occurs. More than ten percent of birth records were missing information on medical risk factors in the following birth registration states: Connecticut, Maryland, Oklahoma, Texas, and Washington.

In this study, we focus on three medical risk factors. We use the "other medical risk factor" category when it is not missing. Secondly, we created an indicator variable for "labeled risk factor", which is equal to one when any one of fifteen medical risk factors (excluding other risk factors and

herpes) is reported. This "labeled risk factor" variable is missing whenever one of the component factors is not reported. Finally, we analyze genital herpes as a "control" outcome. That is, since genital herpes is likely attributable to current behavior and lifestyle and cannot be plausibly linked to health as an infant, we use changes in it across birth cohorts as another test of the internal validity of our findings.

Table 2 presents summary information on the samples analyzed in this study. Panel A presents sample means for women giving birth between 1985-1997 who were born in the United States, the North (here defined to be the Middle-Atlantic and East North Central states), and the South between 1959 and 1970. Panel B presents the same information for Mississippi- and Alabama-born women giving birth during the 1980s and 1990s. The overall sample consists of over 23 million birth records. The Southern and Northern (as defined) states account for 88 percent of all black births. There are racial disparities in demographic and socioeconomic characteristics, "labeled" and "other" maternal risk factors, and large disparities in the incidences of low birth weight and low APGAR scores. This study examines whether these disparities were lower among black women born in the late 1960s as compared to black women born in the early 1960s.

Approach to Estimating Cohort Effects and Tests of Validity

Here, we describe our approach to estimating mother's birth cohort effects and the validity tests we use to determine whether these estimated cohort effects are driven by changes in early life health conditions.

Estimating Cohort Effects and Regression Adjustment

A well-known identification issue that arises is that mother's age, birth cohort, and survey year are perfectly collinear. For example, in a single survey year such as 1991, unrestricted dummies for mother's age would exactly absorb the mother's year of birth. In another example, one might like to compare the outcomes of a black, married, high school graduate, who is 25 years-old and born in 1969 to a demographically identical black woman who is 25 years-old and born in 1963. Note, however, that the former mother is observed giving birth in 1994 while the latter mother gave birth in 1988. Thus, a

comparison of the birth outcomes of these two women may be biased by any changes in the technology of birth between 1988 and 1994, such as the advent of artificial pulmonary surfactant therapy.[17]

This problem is widespread and exists in any economic or epidemiologic study that estimates either life-cycle or cohort effects. Conventional approaches to this problem in the economics literature involve: 1) placing parametric restrictions on the age, time, or cohort effects; 2) modeling the effects as functions of observable variables; and/or 3) assuming additive separability of the effects.[18] Our approach, on the other hand, is to use flexible controls for mother's age and year in which she gave birth -- e.g., interactions of a cubic polynomial in age with unrestricted survey year effects -- and examine whether the estimated birth cohort effects exhibit trend breaks that correspond with the breaks in infant mortality during the 1960s. This is possible since we have multiple (1985-1997) survey years over which we observe outcomes for birth cohorts of pregnant women.[19]

Let i index the individual, r index the race of the individual, c index the individual's birth cohort, a index the individual's age, and t index the survey year of the observation. One linear probability model that we estimate is:

$$(1) \qquad y_{ircat} = X_{ircat}'\beta_r + \alpha_{rc} + \gamma_{ra} + \lambda_{rt} + e_{ircat},$$

where y is the outcome of interest; X contains dummies for the marital status and educational categories (less than HS graduate, HS graduate, some college, college graduate or more, and missing education) of the mother, with effects, β_r, that are allowed to vary by race; α_{rc} are unrestricted race-specific mother's birth cohort dummies (c = 1955-1975); γ_{ra} are race-specific dummies for the age categories of the mother (16-17, 18-19, 20-21, 22-23, 24-25, 26-27, 28-29, 30-31, 32-33, 34-35); λ_{rt} are unrestricted race-specific

[17] It should be noted that surfactant therapy, which treats respiratory distress syndrome in premature infants, would have the effect of reducing infant mortality rates but increasing the incidence of very low birth weight.
[18] See Brugiavini and Weber (2002). In the additively separable case, the effects are often modeled using smooth functions such as polynomials.
[19] Thus, we essentially have "synthetic" birth cohorts that can be observed as they age. However, we do not have repeated observations on the outcomes for the same woman as she ages that would be available in prospective longitudinal data (although some women are observed more than once due to multiple births over the sample frame).

survey year effects (t = 1979-2000); and e is the stochastic error term.[20] This is the regression model underlying the results in Table 1.

We also estimate a more unrestricted linear probability model:

(2) $y_{ircat} = X_{ircat}'\beta_r + \alpha_{rc} + f_r(age) + \lambda_{rt} + f_r(age)\cdot\lambda_{rt} + e_{ircat}$,

where f is a flexible function that is allowed to vary by race, and $f_r(age)\cdot\lambda_{rt}$ are interactions of this function with unrestricted race-specific survey year indicators. This model allows the age profile of the outcomes to be different in each survey year. Below, we specify f as a cubic function in age, but we found similar results (and smaller sampling errors) when we interacted age categories with every survey year. Thus, the estimated mother's birth cohort effects are deviations from "smooth" age effects in each survey year, which is clearly very demanding on the data. Here, the existence of sharp breaks in the early health of birth cohorts, such as shown in Figure 2, is crucial for identifying subsequent breaks in adult health and initial health of the subsequent generation.

Tests of the Internal Validity of the Findings

After obtaining estimates of the black and white year-of-birth effects, α_{rc}, based on equations (1) or (2), we examine whether they exhibit trend breaks that match the location and timing of the trend breaks in black-white infant mortality rates during the 1960s. The early life health conditions hypothesis would imply that black women born in the late 1960s have better health and birth outcomes than black women born in the early 1960s and that the across-cohort gains are smaller for white women. This suggests a difference-in-differences regression specification that estimates the difference in the across-cohort improvements for black women born in the 1960s relative to their white counterparts:

To implement this framework, we estimate the following equation based only on the sample of black and white women born in either 1961-1963 or 1967-1969:

(3) $y_{ircat} = X_{ircat}'\beta_r + \text{constant} + \theta_1\,\text{Black} + \theta_2\,\text{Post}_c + \theta_3\,\text{Post}_c\cdot\text{Black} + \gamma_{ra} + \lambda_{rt} + e_{ircat}$,

[20] Interacting all of the regression coefficients with race allows us to control for and examine the "weathering hypothesis", for example.

where Black is an indicator equal to one if the mother is black; $Post_c$ is an indicator equal to one if the mother was born in 1967-1969 and equal to zero if she was born in 1961-1963; and $Post_c \cdot Black$ is their interaction. Thus, θ_1 measures the black-white outcome gap among women born in 1961-1963; θ_2 measures the across-cohort outcome change among white women; and θ_3 measures the difference between black and white women in the improvement in outcomes from the 1961-1963 to 1967-1969 birth cohorts – i.e., the difference-in-differences estimate.

Below, we find that the across-cohort relative gains in the health and birth outcomes of black women born in the 1960s are large and significant and that their timing corresponds with the timing of the 1960s infant health gains. To examine whether these findings may be artifacts of influences other than early life health conditions, we perform several tests of internal validity. First, we examine the across-cohort outcomes of black and white women who gave birth in the U.S. during the 1980s and 1990s but were themselves born outside the U.S. during the 1960s. Foreign-born black women presumably were not affected by the infant health gains experienced by native-born blacks during the 1960s. Thus, a finding of similar across-cohort gains among foreign-born black women would suggest that the results for native-born African-Americans are attributable to race-specific omitted factors and not to changes in the early health conditions of U.S.-born blacks.

Second, we also examine whether there are across-cohort improvements in maternal risk factors that cannot be plausibly linked to health as an infant. The natality data contain information on one such risk factor – genital herpes – that is presumably the result of behavior and lifestyle choices and not childhood health conditions. Thus, a finding of reduced herpes rates among black women born in the late 1960s relative to those born in the early 1960s would suggest that the across-cohort improvements in other outcomes are the result of changes in adult behavior and lifestyle and not changes in infant health conditions. Third, Southern states had a significantly larger reduction in the black-white infant mortality gap after 1964 than Northern states. As a result, we also examine whether the across-cohort relative gains are greater for Southern-born black women than for Northern-born black women.

Finally, our most convincing test of causality utilizes the differential changes in black-white infant mortality rates between Mississippi and Alabama during the 1960s. Based on infant mortality

rates, the across-cohort improvement in the relative health of black infants was significantly greater in Mississippi than in Alabama. We examine whether the across-cohort relative gains in birth outcomes during the 1980s and 1990s are greater for Mississippi-born black women than for their Alabama-born counterparts. We also examine whether their patterns correspond to the patterns in relative infant mortality rates in the two states during the 1960s. In this analysis, we estimate a difference-in-differences-in differences regression equation, which augments equation (3) to include indicators for state-of-birth and interactions of state-of-birth with all of the variables. Here, our conclusions will only be biased by omitted variables that exhibit similar trend breaks that happened to impact black women born in Mississippi in the late 1960s more than black women born in Alabama in the late 1960s (relative to black women born in the early 1960s and relative to their white counterparts). It seems unlikely that there are many variables that will exhibit these patterns.

Results

This section is currently being re-written – the below text does not match the order of the new tables and figures, although the conclusions are the same.

Results for the Entire United States

Table 3 presents results for the incidence of maternal risk factors, estimated using equation (1). The first four columns assess birth cohort effects for risk factors "labeled" on the birth certificate. Findings are similar to those presented in Table 1. The steep age profile for risk factors found for black women in column one flattens when differences by birth cohort are accounted for. Black mothers exhibit much great across-cohort improvements in the incidence of risk factors (column three) than white mothers (column four). Thus, the pattern of "weathering" as black mothers age is apparently an artifact of younger birth cohorts of black women being healthier than older cohorts. Results for "other" risk factors (columns five through eight) show a similar pattern. The cohort effects estimated for black mothers are much larger in magnitude than the age effects and comparable to the marital status and education effects. Estimates of survey year effects are also sensitive to the birth cohort effects. Like the results presented in Table 1 for birth weight, results estimated for maternal risk factors indicate that that black women born in the late 1960s are healthier than black women born in the early 1960s.

Figure 5 shows trends over the same interval for rates of maternal risk factors. Panel A shows the raw (unadjusted) black-white differences in the incidence of "lableled" maternal risk factors by mother's year of birth, which fall in birth year to approximately equal levels for blacks and whites in 1970. Panel B shows regression-adjusted differences (using estimated coefficients reported in Table 3), showing a similar pattern. Panels C and D plot the analogous rates for risk factors not labeled on the birth certificate. Figure 2 indicates that the timing of the across-cohort relative improvements in black maternal health roughly corresponds with the relative improvements in cohort infant mortality rates in Figure 1. Relative to white women, black mothers born in the late 1960s are indeed healthier than black mothers born in the early 1960s.

Figure 3 shows the raw (unadjusted) black-white differences in birth outcomes by mother's year of birth – very low birth weight incidence (Panel A) and extremely low birth weight incidence (Panel B). Again, it is clear that black women born in the late 1960s have better birth outcomes than black women born in the early 1960s. The timing of the improvements does not correspond quite as well with Figure 1. Figure 4 presents regression-adjusted birth outcomes by mother's year of birth. Here, regression equation (2) is used (where a cubic in age is interacted with each survey year). All panels show across-cohort changes in black-white birth outcomes that correspond very well with the patterns in Figure 1. We conclude that the infants of black women born in the late 1960s are healthier than the infants of black women born in the early 1960s and that the patterns are consistent with changes in early life health conditions being the cause.

Table 4 presents results of equation (3), which fits a difference-in-differences model to the estimated mother's birth cohort effects shown in the figures. Thus, the estimates compare outcomes among black mothers for the 1967-69 birth cohort versus the 1961-63 birth cohort to the corresponding change for white mothers. The first three columns contain the unadjusted results; the last three columns give the regression-adjusted results. (Regression-adjustment uses the equation (1) specification, but is not sensitive to using equation (2)). Results indicate significant cohort improvements in maternal health for black woman born in the late 1960s. Maternal risk factors (labeled and other risk factors) as well as birth outcomes (birth weight, 5-minute APGAR score, gestation length, and likelihood of infant death) all improve for mothers born in the late 1960s versus those born in the early 1960s. A less consistent pattern is observed for white mothers.

The shaded columns of Table 4 present the difference-in-differences estimates. Across a range of health outcomes, cohort gains for blacks were larger than for whites. The estimated improvement is large. For example, the estimates suggest that the black-white gap in very low birth weight incidence was 20 percent lower in the 1967-69 cohort than in the 1961-63 cohort. Only one factor worsened across black cohorts relative to whites: the herpes rate. This is consistent with our hypothesis that infant health changes account for the observed cohort patterns, and if anything suggests that sexually-related behaviors of black women born in the late 1960s are moving in opposition to the other outcomes.

Figure 5 presents a validity check of our results. The top panels plot outcomes for native-born blacks, native-born whites, foreign-born blacks, and foreign-born whites. The only group in this set experiencing an improvement in maternal risk factor rates over the 1959-1970 birth interval is native-born black mothers. The bottom panel, which plots differences between native black and native white mothers, foreign black versus native white mothers, and foreign black versus foreign white mothers, underscores this pattern. Panels B highlights the across-cohort improvement in maternal risk factors for native-born black women. This pattern is consistent with the infant health conditions prevailing in the United States during the 1960s being the causal factor in the subsequent health changes.

Panels C and D of Figure 5 show that, if anything, herpes rates were worsening across cohorts of black women born in the United States. Panel D highlights the across-cohort deterioration in herpes infection for black mothers. Considering Panels B and D together, our hypothesis of cohort health improvement for native-born black mothers predicts that only one of the six lines should show improvements across birth cohorts: and it happens to be the line that indeed shows improvement; maternal risk factors for native-born black mothers improve when compared with native-born white women (Panel B). Panels E and H make these same comparisons for infant birth weight in the 1980s and 1990s. Panels E and F (reflecting low birth weight incidence) and G and H (incidence less than 1000 grams) show that, again, only native-born black women exhibit across cohort improvements in birth outcomes. This pattern is observed even though the incidence of extremely low birth weight (< 1000 grams) is actually higher among foreign-born black women than among native-born black women.

Figure 6 looks at the above patterns by maternal education. Panel A plots the across-cohort change in maternal health (Panel A) by education category: less than high school, high school graduate, and some college or more. The top panel plots these patterns in year of birth for black mothers born in the United States, while the bottom panel plots this pattern for white mothers. Panel A shows that the largest across-cohort gains were clearly among less-educated black women. To the extent that less-educated black women are from poorer family backgrounds (e.g., intergenerational correlation in education) and the War on Poverty programs had larger effects on disadvantaged families, then these findings are consistent with those policies underlying these changes. Panel B plots the cohort changes by

education group for the incidence of birthweight less than 1500 grams. While black mothers exhibit across cohort-improvement for each of the three education groupings, the largest gains are found among mothers who did not complete high school. [21]

Results for Southern versus Northern United States

Figure 7, panel A compares differences in infant mortality rate by race during the 1960s between northern states (New Jersey, New York, Pennsylvania, Indiana, Illinois, Michigan, Ohio, Wisconsin) and states in the South. The differences by race decrease in both northern and southern states, but the improvement for black infants is clearly larger in the South than in northern states.

Panels B plots the difference by race in the incidence of birthweight below 1000 grams during the 1980s and 1990s by the region in which the mother was born. These differences are regression-adjusted using equation (2) and allow all regression coefficients to be different in the North and South. Greater across-cohort gains in the incidence of birthweight below 1000 grams are observed among black women born in the South than among black women born in northern states. The same patterns of larger improvements for black mothers born in southern states is observed for the likelihood that an infant's APGAR score was less than 8 (Panel C) or below 7 (Panel D). Also, there is a strong correspondence in the timing of the breaks for black infant health in the 1980s and 1990s with the patterns in Panel A for infant health during the 1960s. These patterns are consistent with health at the beginning of mother's life being a driving force in the birth outcomes of their infants.

Comparisons between Mississippi and Alabama

Tables 5 presents the estimated effects of birth cohort on the incidence of low birthweight for mothers born in Mississippi. (Results are based on regression equation (1).) Similarly, Table 6 presents

[21] Unfortunately, black and white infant mortality rates during the 1960s cannot be separately-calculated by the characteristics of the family for this period (given the lack of mortality microdata). In addition, we do not know the characteristics of the parents of the mothers giving birth during the 1980s and 1990s.

analogous results for mothers born in Alabama. All coefficients are allowed to vary by state. Results are similar to those presented in Table 1: black mothers show large across-cohort improvements in the incidence of low birthweight of their children. Ignoring these cohort effects biases the age-profile of maternal health, as well as the evolution of low birthweight rates over time (given by the survey-year coefficients).

Panel A of Figure 8 plots the black-white differences in infant mortality rates during the 1960s in Alabama and Mississippi, respectively. It is clear that the black relative improvements in infant mortality are much greater in Mississippi than in Alabama. It should be noted that these are the two neighboring states in the South with the greatest difference in changes in relative mortality rates during the 1960s. This makes Alabama and Mississippi good states for testing the hypothesis that improvements in infant health affect maternal health.

Panels B through F of Figure 8 plot the regression-adjusted black-white differences in outcomes by mother's year of birth for the incidence of low birth weight, low APGAR score, and maternal risk factors. (These figures are all based on the same regressions used in Tables 5 and 6, and therefore allow the effects to vary by state.) Across the set of outcomes, it is clear that the across-cohort relative gains in birth outcomes are larger for Mississippi-born black mothers than for mothers born in Alabama. Further, the patterns by mother's year of birth correspond remarkably well with the patterns in Panel A for infant health changes in Mississippi and Alabama. These figures provide very strong evidence that the relative improvements in infant health conditions are causing relative improvements in the birth outcomes of the women who benefited from the improved conditions. Also, the figures suggest that a difference-in-differences-in-differences regression specification may be a good way of estimating these gains.

Table 7 shows the regression specification we use to estimate the difference-in-differences estimates for Mississippi. For Mississippi, it is clear that there are significant across-cohort improvements in birth outcomes for black women: the incidence of birthweight below 2500 grams and below 1000 grams both show significant decreases for black mothers born between 1967 and 1969 versus those born between 1961 and 1963. White women born in Mississippi show no across-cohort

improvement in these outcomes. We use the same specification to construct differences-in-differences estimates for mothers born in Alabama.

Table 8 presents all of the differences estimates for Mississippi and Alabama. Column two presents the black-white cohort differences in Mississippi, while Column four presents the analogous estimates for mothers born in Alabama. The final column presents the difference-in-differences-in-differences estimate comparing black-white, across-cohort differences in Mississippi and Alabama. The first set of rows present the unadjusted differences; the second set of rows present the regression-adjusted differences based on the specification used in Table 7.

All of the estimates show that black mothers born in Mississippi in the late 1960s had significantly better birth outcomes than: 1) black mothers born in Mississippi in the early 1960s (column one); 2) than the across-cohort gains for Mississippi-born white women (column 2); and 3) than the relative across-cohort gains for Alabama-born black women (last column). It should be noted that regression adjustment only serves to increase the size of the estimated effects. Also, the magnitudes of the effects are quite large. The estimates imply that among Mississippi-born women the black-white gap in very low birth weight incidence was 40 percent lower in the 1967-69 cohort than in the 1961-63 cohort. The improvement was 50 percent for the incidence of extremely low birth weight (less than 1000 grams).

Table 9 presents the Mississippi versus Alabama difference-in-differences-in-differences estimates based on various specifications. The first column presents the unadjusted estimates (also reported in the first set of outcomes in the last column of Table 8). The second column reports estimates based on regression equation (1) and constrains the control variables to have the same effects for Mississippi- and Alabama-born women. The third column is based on regression equation (2), and also constrains the variable effects to be the same by mother's state of birth. The final column allows the effects of the control variables to be different by mother's state of birth (the same specification as in Table 8). The results show that the difference-in-differences-in-differences estimates are not sensitive to the way we control for mother's age and year of the birth, and if anything, the estimates increase the fewer restrictions we impose.

Potential Mechanisms by which Infant Health Improved During the 1960s

Increases in Federal Transfer Payments (REIS data)

There has been relatively little work on the proximate causes of decreased infant mortality during the 1960s. Given the magnitude of the changes in infant mortality and the fact that several key federal programs that could have had an impact on infant health, including Medicaid and Food Stamps, were initiated during this period, this is somewhat surprising. While it is not the focus of this paper, the relationship between the 1960s infant mortality decline and the large increase in federal transfer programs can be assessed in future work using 1960s data on transfer payments from the Bureau of Economic Analysis and information on infant mortality from annual print volumes of the *Vital Statistics of the United States*. These two sets of information are currently being converted to an electronic format for each county in the United States.

For present purposes, it is nevertheless helpful to consider in general terms how infant health conditions may have changed during the 1960s. As noted above, infant mortality rates can change for two substantively distinct reasons 1) changes to the (unobserved) distribution of individual health 2) changes to the health threshold at which newborn infants survive infancy. Obviously, many factors changed during the 1960s that could affect either the unobserved distribution of health or the health thresholds for infant survival. That we observe large decreases in infant mortality for African American infants over this period indicates that large changes indeed occurred in either the initial health distribution or the survival thresholds. However, because we observe improvements in subsequent maternal health for mothers born during the late 1960s, it appears that improvements in the unobserved distribution of health were more important.[22]

As we can observe these improvements in cohort health in the aggregate (that is among persons born in the United States) we would ideally like to investigate the causes of improved initial health in data

[22] Alternatively, if the health threshold at which infants survived shifted to the right (due to a decrease in access to medical care or a deterioration in medical technology), this same pattern could be observed.

series with national coverage. However, national data from the 1960s on factors that might conceivably have led to improvements in initial health are scarce. *Disaggregated* data series providing such information, which might conceivably permit analysis of which groups or geographic regions were more likely to be affected by public policies, institutional changes, etc., are even more difficult to obtain.

The Bureau of Economic Analysis (BEA) produces annual time series of economic data at state and local levels as part of its Regional Economic Information System. Among these data series, BEA collects information on transfer payments,[23] including, medical insurance payments, Food Stamps payments, and other federal expenditures that could impact infant health. Such series are of particular interest given their large expansion under the War on Poverty and Great Society programs.

Transfer payments in aggregate more than doubled during the 1960s, reaching $59 billion in 1968 (in 1968 dollars). While most of this increase was driven by the expansion of the Social Security program, the inception of major health policies, including Medicare, Medicaid, and Food Stamps can be investigated for their impact on infant health.

BEA produces electronic data on transfer payments at the national, state, and county level back to 1969. Before 1969, no such electronic data exist. BEA has, however, provided microfiche of transfer payments at the national, state, and county level for 1959, 1962, 1965, 1966, 1967, and 1968. For the purposes of this research project, these microfiche are currently being converted to an electronic format. These data include a breakdown of transfer payments into approximately twenty payment categories. *A priori*, the most likely expenditure candidates to exert an effect on infant health are the "Medical Insurance Payments", "SSI, AFDC, General Assistance Payments", and "Food Stamps Payments".[24]

The Medical Insurance Payments category records zero payments until 1966, when the Medicare and Medicaid programs were established. Payments were approximately one billion dollars in 1966, $4.3 billion in 1967, and $5.7 billion in 1968 (all figures in unadjusted dollars). SSI, AFDC, and General Assistance payments started at a higher initial level and increased more modestly – from $3.2 billion in

[23] Transfer payments are defined as "payments to persons for which they do not render services in the current period" (U.S. Department of Commerce, Bureau of Economic Analysis).
[24] Unfortunately, the BEA did not retain any documentation for the pre-1969 microfiche data.

1959 to $5.7 billion in 1968. Finally, the Food Stamp program began in the early 1960s, and increased rapidly, but Food Stamps was a comparatively small program and did not exceed $100 million in payments until 1967.

A systematic analysis of the regional variation in the expansion of transfer payments cannot be conducted until the 1960s microfiche data have been converted to an electronic format. However, initial results obtained by looking at the state levels of transfer payments suggest substantial regional variation in the expansion and importance of these programs.

For example, in terms of per-capita medical insurance payments, New York State experienced approximately twice as large an increase between 1965 and 1968 as did Mississippi or Alabama, and approximately forty percent faster growth than Pennsylvania. In contrast, Food Stamp payments increased much more rapidly in Mississippi between 1965 and 1968 than in other states; per-capita expenditures were more than five times as high as in neighboring Alabama in 1968. Per-capita food stamp expenditures in New York were essentially zero throughout the 1960s.

In future work, we will investigate the inception and expansion of these programs at the county level within each state, matching these expenditure series to data on infant and maternal mortality. The analysis of BEA data should provide information on the role of federal transfer programs in generating improvements in infant and thereby cohort health during the 1960s. As the effect of these programs on cohort health has not previously been considered, it will potentially provide more comprehensive estimates of the benefits of these signature federal programs.

Alternative Potential Mechanisms Presented in Mississippi Paper

We will also investigate the role of other key 1960s changes that may have affected infant health outcomes. In particular, we will explore the role of expanded access to medical care for African Americans with the desegregation of southern hospitals during the 1960s. In Mississippi, where hospitals were strictly segregated until the mid-1960s, it appears that the inception of the Medicare program created

a large financial incentive for hospitals to comply with Title VI of the 1964 Civil Rights Act, which prohibited racial segregation and discrimination in institutions receiving federal funding. We plan to match information on certification for the Medicare program for each hospital in the United States provided by the annual American Hospital Association guides to the county-level data on infant and maternal mortality rates provided by the annual *Vital Statistics* volumes in order to further evaluate this hypothesis.

Additional factors that will also be explored include the expansion of the maternal and infant care component of the Maternal and Child Health program (not recorded separately in the BEA data). This program explicitly aimed to improve the health of mothers and infants from families with low income levels and diverse racial and ethnic heritages and those living in rural areas without access to care. Moreover, the 1963 and 1965 amendments to Title V resulted in dramatic increases in MCH funding of maternal and infant care projects (Davis and Schoen 1978). It appears, however, that this program was likely to have been more important in Northern and urban areas than in the South, where the improvement in infant health was the most dramatic. Finally, previous work has pointed to the expansion of the network of community health centers as important for infant health during the 1970s. The role of these centers during the 1960s will also be evaluated, although the number of centers opened during the 1960s was quite limited (Goldman and Grossman 1988).

Conclusion

Results presented above indicate large improvements in the health of black mothers born in the late 1960s when compared with the health of black mothers born a few years earlier. In contrast, white mothers exhibit small to negligible gains across these birth years. These cohort differences correspond to the large relative improvement in black infant health during the 1960s. Moreover, these cohort improvements are largest where infant health improved the most during the 1960s: black mothers born in southern states exhibit the largest cohort improvements, and black mothers born in Mississippi exhibit larger cohort effects than black mothers born in neighboring Alabama. Validity checks afforded by the data further suggest that improved infant health among black infants during the 1960s in the United States was behind these cohort changes. For example, improvements in cohort health are not observed for black mothers born abroad who did not experience the 1960s improvement in infant health in the U.S. Additionally, the health measure collected on the birth certificate that is more likely to be related to behavior as adults rather than early-life health, the incidence of genital herpes, goes in the opposite direction from the cohort effect among black mothers.

Future work on this topic will expand the coverage of natality detail files to additional years (e.g. including births between 1983 and 1984, 1998 and 1999). This will improve the age coverage of birth cohorts at either end of the 1959-1970 birth interval, as well as permitting analysis of additional birth years. In the process of expanding the data coverage, we will also allow for additional factors which might be related to birth outcomes, including birth order and prenatal care, as well as looking at additional health outcome measures, such as the existence of abnormal conditions in newborn infants. We will also use the county-level federal expenditure information provided by the REIS data along with the timing of certification of each hospital in the U.S. for the Medicare program to evaluate the initial "treatment" stage when infant health improved.

Finally, we also intend to look at additional outcome measures. We will analyze outcomes for both men and women using the 2000 Census Long-form data. In addition, our hypothesis would seem to explain the "anomaly" of reductions in the black-white test achievement gap in the 1980s. Appendix

Figure 4 (from Hanushek 2001) shows a pronounced reduction in the science, math and reading score differences between black and white students at age 17. These 17-year-olds were born in the 1960s and the black relative achievement gains, like the changes in maternal health at the U.S. level, mirror the relative gains in infant health for these birth cohorts. We will use the local variation in infant health conditions during the 1960s as well as the expansion in social programs that may have enabled their improvement to further investigate the role of infant health across the range of adult outcomes.

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