Preliminary

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, 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 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 – e.g., Title VI of the 1964 Civil Rights Act – 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. 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) difference.[1] Since 1950 there have been small decreases in the black-white IMR difference with one notable exception. From 1964 to 1971 the black infant mortality rate and the black-white difference 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 19 to 11, the only prolonged convergence in the post-World War II era.

Almond, Chay and Greenstone (2003) 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, Figure 2 shows trends in black and white post-neonatal mortality rates from 1946-1975 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 (2003) 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 than blacks born in the

[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.

early 1960s. A finding of a long-run and intergenerational link has important 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 conventional 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 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 2005).

(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, 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 not 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 those 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 due to the 1964 Civil Rights Act, 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.

I. Motivation and Background

Here, 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.

A. 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 (2003) 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.

B. 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

[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).

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]

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 shows, it appears that this conclusion may be an artifact of better health at birth among younger than older cohorts of black women. Panels A and C of Figure 3, which plot the estimated effects of mother's age on birth outcomes unadjusted for mother's birth cohort effects, show results that are consistent with the weathering hypothesis. However, Panels B and D show that once mother's year of birth

[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 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.

effects are accounted for, black teenagers now have the worst birth outcomes and the “optimal” age for childbearing is similar by race.

C. 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 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]

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[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

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 1901-1910 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.

D. Research Design Issues

Several factors impede decisive 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

(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.

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 (2005) 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, 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

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

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.

II. 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 and low birth weight 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

[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.

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 $h^*_i \geq d_1$.

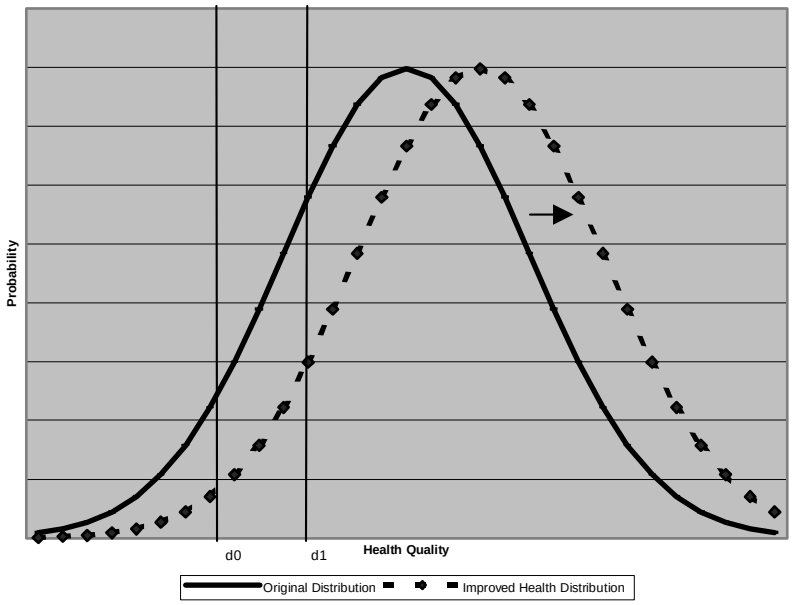
Given these health thresholds, the infant mortality rate (IMR) may be defined using the cumulative distribution function $F(h^*_i)$ as:

$$IMR \equiv F(d_0)$$

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]

[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.

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 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.

III. 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. In total, information on 75 million births is available across the survey years.

For births occurring between 1983 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]

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[14] At present, the 1995-2000 *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.

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 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

[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.

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 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 1979 and 2000 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 1955 and 1975. Panel B presents the same information for Mississippi- and Alabama-born women giving birth during the 1980s and 1990s, and Panel C does the same for New York- and Pennsylvania-born women. The overall U.S.-born sample consists of over 33 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.

IV. 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.

A. Estimating Cohort Effects and Regression Adjustment

A well-known identification issue 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 is to use flexible controls for mother's age and year in which she gave birth and examine whether the estimated birth cohort effects exhibit trend breaks that correspond with the breaks in infant mortality during the 1960s. This approach is enabled by having multiple 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 survey

[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).

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

In the analysis that compares U.S.-born (Mississippi-born) women to foreign-born (Alabama-born) women, we estimate models that include unrestricted age dummies that vary by race. Intuitively, differencing the estimates between affected and unaffected groups absorbs all effects that are constant between the two groups. This approach assumes that the age effects for black women born in Mississippi, for example, mirror those of Alabama-born black women. Below, we allow all other effects to vary for the two groups. Further, we re-estimate all of our models (and figures) using a probit model that constrains the predicted probabilities to be between zero and one. The resulting estimates (and figures) are very similar to those produced by the linear probability regression and are actually stronger. Finally, we correct the estimated standard errors for potential heteroskedasticity and clustering at the group-race-year-age level.

B. Tests of the Validity of the Findings

After obtaining estimates of the black and white year-of-birth effects, α_{rc}, based on equation (1), 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 1960-1962 or 1968-1970:

$$(2) \qquad y_{ircat} = X_{ircat}'\beta_r + \text{constant} + \theta_1 \text{Black} + \theta_2 \text{Post}_c + \theta_3 \text{Post}_c \cdot \text{Black} + \gamma_{ra} + \lambda_{rt} + e_{ircat},$$

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 1968-1970 and equal to zero if she was born in 1960-1962; and $\text{Post}_c \cdot \text{Black}$ is their

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

interaction. Thus, θ_1 measures the black-white outcome gap among women born in 1960-1962; 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 1960-1962 to 1968-1970 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. In this case, we can use a difference-in-differences-in-differences strategy that allows for unrestricted race-specific, age effects (as long as they are not interacted with native-born status).

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. Here, the issue of selective attrition discussed in Section II appears to be a first-order issue.

Finally, the 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 (2) 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 (1966-1969) more than black women born in Alabama in the late 1960s – relative to black women born in the early 1960s (1961-1964) and relative to their white counterparts. It seems unlikely that there are many variables that will exhibit these patterns. As an additional validity check, we use a similar analysis to compare New York-born women to Pennsylvania-born women, whose cohorts experienced very similar changes in black-white infant mortality rates during the 1960s.

V. Results

This section is currently being revised – the below text provides a rough summary of the results.

A. Results for the Entire United States

Figure 4 presents the estimated, mother's year-of-birth effects on the incidence of extremely low birth weight (less than 1000 grams) among infants born between 1979 and 2000, based on the specification used in Table 1 – i.e., it plots the estimates in the final two columns of the table. The figure shows that while the extremely LBW incidence is about 20 percent lower among the infants of black women born in the early 1970s relative to those of blacks born in the early 1960s. The white incidence, on the other hand, is quite similar between these cohorts. As a result, the black-white difference in extremely LBW incidence

falls by over 25 percent between the early 1960s and early 1970s birth cohorts, and the patterns are somewhat consistent with the patterns in black-white infant mortality rate differences shown in Figure 1 and in Panel A of Figure 5. This is particularly so when they are contrasted with the changes in the black-white postneonatal mortality rate.

Figure 5 also shows the raw (Panel B) and regression-adjusted (Panel C) black-white differences in the mother's year-of-birth effects for several outcomes of interest. Both Panels show large, across-cohort changes in the relative incidence of maternal risk factors, with the black-white gap mirroring the black-white gap in (postneonatal) infant mortality rates in Panel A. Indeed, the black-white gap in mother's risk factors is 70 percent smaller among women born at the end of the 1960s relative to those born at the beginning of the decade. Similar patterns are shown for the birth weight, APGAR score, and gestation length of the infants of these women. Importantly, the patterns are reversed for the black-white difference in herpes rates. In fact, black women born in the late 1960s had higher rates of herpes relative to their white counterparts than black women born in the early 1960s, suggesting that behavioral changes do not explain the patterns for the other outcomes.

Relative to white women, black women born in the late 1960s – and their infants – are indeed healthier than black women born in the early 1960s. Figures 6 and 7 present a validity check of our results. Panel A of Figure 6 depicts the raw, black-white differences in the LBW incidence of infants by mother's year-of-birth for: 1) U.S.-born black relative to U.S.-born whites; 2) foreign-born blacks relative to foreign-born whites; 3) U.S.-born blacks relative to foreign-born blacks; and 4) U.S.-born whites relative to foreign-born whites. The panel shows that only U.S.-born blacks exhibit a relative improvement in the birth weight of infants born to women themselves born in the late 1960s relative those born in the early 1960s, whether compared to U.S.-born whites or to foreign-born blacks or whites. This result is consistent with the hypothesis that changing infant health conditions faced by blacks *born in the United States* are the cause of the health improvements exhibited in the next generation of infants.

Panel B of Figure 6 presents a similar analysis for the mother's health risks prior to giving birth in the 1980s and 1990s. These patterns are even more striking, as it is clear that only U.S.-born black women

exhibit an across-cohort gain in health as an adult relative to the various comparison groups. Further, the trend breaks in both panels correspond with those in Figure 5 A.

Figure 7 shows the raw (Panel A) and regression-adjusted (Panel B) difference-in-differences – (native black minus native white) minus (foreign black minus foreign white) – in mother's year of birth effects for several outcomes of interest. In all cases, the patterns are striking and consistent with the infant health conditions hypothesis. In particular, there are sharp improvements in the relative health of U.S.-born black mothers (and their infants) who are born in the late 1960s compared to those born in the early 1960s, with patterns very consistent with the predicted trend break in year-of-birth effects. The only exceptions are the patterns for herpes rates, which is again consistent with the maintained hypothesis.

Table 3 presents results of estimating equation (2), which fits a difference-in-differences model to the estimated mother's birth cohort effects shown in the figures, for U.S.-born and foreign-born women. Thus, the estimates compare outcomes among black mothers for the 1968-1970 birth cohorts versus the 1960-1962 birth cohorts to the corresponding change for white mothers for the two groups. The first three columns present the raw, unadjusted results, while the final three columns present the regression-adjusted results. The table also presents the difference-in-differences-in-differences estimates obtained by further contrasting the relative changes in the black-white differences between U.S.- and foreign-born women.

The 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) 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 difference-in-differences-in-differences estimates contrasting U.S.- and foreign-born women show that across a range of health outcomes, across cohort gains for U.S.-born 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 30 percent lower in the 1968-70 cohorts than in the 1960-62 cohorts. 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.

Table 4 presents the difference-in-differences-in-differences results from four different regression specifications – the "Unadjusted" column is the same as column 3 of Table 3, while the "Regression 3" column corresponds to column 6 of Table 3. The main conclusion is that the results are quite robust across specifications.

B. Results for Southern versus Northern United States **(ignore, since doesn't correspond with revised figures)**

Figure 8, 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 (1) 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.

C. Comparisons between Mississippi and Alabama

Panels A and B of Figure 10 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 C and D of Figure 10 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 6 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.

D. Comparisons between New York and Pennsylvania

Figure 11 and Table 7. Bottom line – no differences for black women born in the two states. Confirms that one cannot replication the Mississippi versus Alabama findings elsewhere.

Appendix Figure 1 – find similar patterns for black-white test scores (AFQT scores among military applicants). Pretty striking.

VII. 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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Appendix: Potential Mechanisms by which Infant Health Improved During the 1960s

A. 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.[21]

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 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

[21] 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.

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,[22] 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".[23]

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 1959 to $5.7 billion in 1968. Finally, the Food Stamp program began in the early 1960s, and increased

[22] 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).
[23] Unfortunately, the BEA did not retain any documentation for the pre-1969 microfiche data.

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.

B. 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).

Table 1: Estimates of Mother's Year of Birth Effects for Birth Weight and Gestation Length in the U.S., 1955 to 1975 Mother's Birth Cohorts
(estimated standard errors in parentheses)

	Incidence < 1500 grams (per 1,000 births)				Incidence < 1000 grams (per 1,000 births)			
	No cohort effects		Cohort effects		No cohort effects		Cohort effects	
Coefficients	Black	White	Black	White	Black	White	Black	White
Mother Age Effects								
16-17	-1.89***	2.90***	1.25***	0.47*	-1.11***	1.17***	0.63***	0.15
	(0.24)	(0.22)	(0.24)	(0.27)	(0.16)	(0.15)	(0.17)	(0.19)
18-19	-1.92***	1.59***	0.14	-0.16	-1.22***	0.67***	-0.09	-0.01
	(0.14)	(0.13)	(0.14)	(0.18)	(0.10)	(0.09)	(0.10)	(0.12)
20-21	-1.88***	0.62***	-0.55***	-0.52***	-1.01***	0.24***	-0.29***	-0.19**
	(0.11)	(0.09)	(0.11)	(0.13)	(0.07)	(0.07)	(0.07)	(0.09)
22-23	-1.17***	0.15*	-0.44***	-0.38***	-0.56***	0.04	-0.19***	-0.14**
	(0.09)	(0.08)	(0.10)	(0.09)	(0.07)	(0.06)	(0.07)	(0.07)
24-25	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
	--	--	--	--	--	--	--	--
26-27	0.57***	-0.34***	0.08	0.53***	0.47***	-0.23***	0.13**	0.17***
	(0.09)	(0.08)	(0.09)	(0.09)	(0.07)	(0.06)	(0.07)	(0.06)
28-29	1.54***	-0.63***	0.62***	0.91***	0.81***	-0.34***	0.23***	0.34***
	(0.10)	(0.08)	(0.10)	(0.12)	(0.07)	(0.06)	(0.07)	(0.09)
30-31	2.02***	-0.42***	0.68***	1.83***	1.21***	-0.31***	0.40***	0.65***
	(0.11)	(0.09)	(0.11)	(0.16)	(0.08)	(0.06)	(0.08)	(0.12)
32-33	2.96***	-0.28***	1.16***	2.66***	1.57***	-0.15**	0.49***	1.10***
	(0.14)	(0.10)	(0.14)	(0.21)	(0.10)	(0.07)	(0.10)	(0.16)
34-35	3.71***	0.04	1.38***	3.73***	1.66***	0.04	0.28**	1.60***
	(0.18)	(0.12)	(0.18)	(0.27)	(0.13)	(0.09)	(0.13)	(0.19)
Married	-5.31***	-6.36***	-7.21***	-5.26***	-2.44***	-3.47***	-3.51***	-2.83***
	(0.12)	(0.06)	(0.13)	(0.06)	(0.09)	(0.04)	(0.09)	(0.04)
Education missing	4.53***	0.51***	4.41***	0.59***	3.47***	0.24***	3.42***	0.28***
	(0.25)	(0.08)	(0.25)	(0.08)	(0.18)	(0.05)	(0.18)	(0.05)
Less than H.S.	0.45***	1.36***	0.59***	1.26***	0.19*	0.17*	0.27**	0.14
	(0.16)	(0.15)	(0.16)	(0.15)	(0.11)	(0.10)	(0.11)	(0.10)
Some College	-0.51***	-1.17***	-1.11***	-1.03***	0.26**	-0.63***	-0.04	-0.57***
	(0.16)	(0.05)	(0.16)	(0.05)	(0.12)	(0.03)	(0.12)	(0.03)
College Grad +	-1.41***	-2.31***	-2.80***	-2.04***	0.23	-1.20***	-0.49**	-1.08***
	(0.25)	(0.05)	(0.25)	(0.05)	(0.19)	(0.03)	(0.19)	(0.03)
Year Effects	Y	Y	Y	Y	Y	Y	Y	Y
Cohort Effects	N	N	Y	Y	N	N	Y	Y
Sample Size	34,118,254		34,118,254		34,118,254		34,118,254	

Table 1 (cont'd)

	Incidence < 1500 grams (per 1,000 births)				Incidence < 1000 grams (per 1,000 births)			
	No cohort effects		Cohort effects		No cohort effects		Cohort effects	
Coefficients	Black	White	Black	White	Black	White	Black	White
Cohort Effects								
1955			40.1***	5.24***			23.2***	3.60***
			(0.65)	(0.53)			(0.48)	(0.38)
1956			40.1***	5.61***			23.3***	3.66***
			(0.63)	(0.51)			(0.47)	(0.37)
1957			39.5***	5.85***			22.9***	3.81***
			(0.61)	(0.48)			(0.46)	(0.35)
1958			40.3***	6.07***			23.5***	3.78***
			(0.60)	(0.46)			(0.45)	(0.33)
1959			39.5***	6.25***			22.9***	3.85***
			(0.58)	(0.44)			(0.43)	(0.31)
1960			39.5***	6.67***			22.5***	3.97***
			(0.57)	(0.41)			(0.42)	(0.30)
1961			39.2***	6.88***			22.6***	4.04***
			(0.56)	(0.39)			(0.42)	(0.28)
1962			38.4***	7.20***			22.2***	4.21***
			(0.54)	(0.37)			(0.41)	(0.27)
1963			37.4***	7.32***			21.6***	4.13***
			(0.53)	(0.35)			(0.40)	(0.25)
1964			36.7***	7.83***			21.0***	4.39***
			(0.52)	(0.32)			(0.39)	(0.23)
1965			36.2***	8.13***			20.7***	4.45***
			(0.51)	(0.30)			(0.39)	(0.22)
1966			35.7***	8.43***			20.6***	4.64***
			(0.51)	(0.28)			(0.38)	(0.20)
1967			35.3***	8.76***			20.3***	4.70***
			(0.51)	(0.26)			(0.38)	(0.19)
1968			34.2***	9.19***			19.5***	4.94***
			(0.50)	(0.24)			(0.38)	(0.17)
1969			33.6***	9.35***			19.3***	5.03***
			(0.50)	(0.22)			(0.38)	(0.16)
1970			32.7***	9.76***			18.8***	5.21***
			(0.49)	(0.20)			(0.37)	(0.15)
1971			32.1***	10.18***			18.4***	5.29***
			(0.49)	(0.19)			(0.37)	(0.13)
1972			31.3***	10.47***			18.1***	5.35***
			(0.49)	(0.18)			(0.37)	(0.13)
1973			30.0***	10.87***			17.3***	5.70***
			(0.49)	(0.17)			(0.37)	(0.12)
1974			29.2***	11.11***			16.9***	5.62***
			(0.49)	(0.17)			(0.37)	(0.12)
1975			28.0***	11.52***			16.2***	5.86***
			(0.49)	(0.17)			(0.37)	(0.12)

Notes: ***, **, and * indicate statistical significance at the 1, 5, and 10 percent levels, respectively. Results based on linear probability regressions. Standard errors corrected for heteroskedasticity. Sample comes from the 1979-2000 Natality Detail Files and contains the 1955 to 1975 birth cohorts. Probit and logit analyses provide similar effects.

Table 2: Sample means for black and white women born in 1955 to 1975, who give birth in 1979 to 2000 (1979-2000 Natality Detail data)

A. By region of mother's birth

	Born in U.S. South		Born in U.S. North		Born outside U.S.	
	Black	White	Black	White	Black	White
Mother's Characteristics						
Age	23.7	25.1	23.8	26.1	26.9	26.7
Teenager percent	20.3	12.0	20.6	8.3	6.2	5.8
Less than HS percent	26.9	19.5	28.3	13.5	19.1	14.4
HS graduate percent	42.4	37.5	40.8	41.6	35.4	31.1
Some college percent	16.6	17.6	20.5	20.6	21.2	18.9
College grad+ percent	5.7	15.0	6.0	18.9	14.6	21.3
Missing educ. percent	8.4	10.4	4.5	5.4	9.6	14.3
Married percent	36.5	85.6	29.0	84.4	56.2	87.5
Prenatal care percent	96.5	99.0	95.3	99.2	96.6	98.6
Care in 1st trimester %	63.8	82.4	63.6	84.5	67.3	80.6
Number of kids	2.48	2.07	2.72	2.17	2.51	2.16
Sample size	4,966,016	11,105,586	2,780,429	15,298,816	707,191	1,876,752
Mother's Medical Risk Factors (per 1,000)						
Any risk factor (excluding herpes)	284.2	259.0	298.1	250.5	247.9	203.1
Herpes	8.59	9.08	10.91	9.29	4.89	7.23
Sample size	2,251,649	5,213,299	1,516,454	8,077,189	460,120	1,048,114
Birth outcomes of infant (per 1,000)						
Birth weight < 2500 g	116.1	51.1	122.5	45.2	87.2	45.5
Birth weight < 1500 g	23.8	7.9	25.8	7.1	24.7	8.4
Birth weight < 1000 g	12.6	3.6	13.8	3.4	15.3	4.6
Sample Size	4,960,423	11,098,122	2,775,837	15,283,872	705,716	1,873,723
5-min. APGAR ≤ 8	127.9	98.7	126.0	95.4	109.8	90.0
5-min. APGAR ≤ 7	48.5	27.1	47.2	26.4	42.1	25.7
5-min. APGAR ≤ 6	27.3	12.7	26.7	12.3	24.5	13.0
Sample Size	3,465,674	7,288,667	2,189,717	12,127,974	514,659	1,210,737
Gestat. length ≤ 36 wks	174.0	82.9	169.6	72.5	130.8	74.3
Gestat. length ≤ 31 wks	40.3	12.4	40.6	11.0	31.7	11.7
Gestat. length ≤ 27 wks	16.5	4.3	17.3	4.1	15.9	4.9
Sample Size	4,750,623	10,728,924	2,670,804	14,923,308	688,659	1,790,866

Notes: Sample contains singleton births to non-Hispanic black and white mothers who are born between 1955 and 1975. Information on mother's characteristics, infant birth weight and gestation length comes from the 1979 to 2000 Natality Detail Files; mother's medical risk factors come from the 1989-2000 Natality Detail files; and APGAR scores come from the 1979-1991 and 1995-2000 Natality Detail Files. The South region consists of all Southern states. The North region consists of the Middle Atlantic (New Jersey, New York, Pennsylvania) and East North Central (Indiana, Illinois, Michigan, Ohio, Wisconsin) divisions.

B. Mississippi- and Alabama-born women

	Born in Mississippi		Born in Alabama	
	Black	White	Black	White
Mother's Characteristics				
Age	23.7	24.9	23.8	25.0
Teenager percent	20.9	12.3	19.7	12.1
Less than HS percent	32.8	20.8	27.3	22.9
HS graduate percent	39.4	35.7	42.7	38.0
Some college percent	18.7	22.4	20.5	20.0
College grad+ percent	6.1	17.1	6.5	16.2
Missing educ. percent	3.0	3.9	3.1	2.9
Married percent	34.8	88.9	38.7	89.3
Prenatal care percent	97.7	99.4	97.3	99.1
Care in 1st trimester %	66.0	85.8	63.5	83.9
Number of kids	2.55	2.02	2.43	2.01
Sample size	456,212	350,000	402,342	603,835
Birth outcomes of infant (per 1,000)				
Birth weight < 2500 g	112.7	51.6	109.7	51.3
Birth weight < 1500 g	21.3	7.5	22.2	8.1
Birth weight < 1000 g	11.0	3.4	11.5	3.6
Sample Size	455,765	349,847	401,828	603,518
5-min. APGAR ≤ 8	98.9	69.1	124.6	89.2
5-min. APGAR ≤ 7	40.3	20.3	46.1	24.0
5-min. APGAR ≤ 6	23.7	10.1	26.1	11.7
Sample Size	364,881	271,667	325,006	483,463
Gestat. length ≤ 36 wks	176.9	87.6	172.9	84.3
Gestat. length ≤ 31 wks	38.2	12.4	40.1	12.8
Gestat. length ≤ 27 wks	14.8	4.0	16.0	4.4
Sample Size	443,011	343,275	384,766	589,708

Notes: See notes to Panel A. Sample contains singleton births between 1979 and 2000 to non-Hispanic black and white women who are born in Mississippi and Alabama between 1955 and 1975.

C. New York- and Pennsylvania-born women

	Born in New York		Born in Pennsylvania	
	Black	White	Black	White
Mother's Characteristics				
Age	24.2	26.9	24.0	26.2
Teenager percent	18.7	6.0	19.5	8.0
Less than HS percent	26.9	9.1	26.4	13.2
HS graduate percent	37.6	33.4	45.0	46.7
Some college percent	20.0	20.5	18.0	18.3
College grad+ percent	6.8	22.7	6.6	19.0
Missing educ. percent	8.8	14.3	4.0	2.8
Married percent	30.0	85.3	27.0	83.4
Prenatal care percent	93.1	99.2	94.8	99.3
Care in 1st trimester %	57.9	85.5	59.8	84.5
Number of kids	2.78	2.21	2.79	2.14
Sample size	655,493	2,941,231	355,016	2,357,479
Birth outcomes of infant (per 1,000)				
Birth weight < 2500 g	124.2	43.3	123.8	45.8
Birth weight < 1500 g	26.5	6.7	26.2	7.2
Birth weight < 1000 g	13.8	3.2	14.2	3.4
Sample Size	654,259	2,936,405	354,411	2,355,969
5-min. APGAR ≤ 8	110.5	78.5	131.7	85.7
5-min. APGAR ≤ 7	44.3	21.9	46.8	23.7
5-min. APGAR ≤ 6	25.9	10.3	26.8	11.5
Sample Size	518,242	2,312,145	284,337	1,907,375
Gestat. length ≤ 36 wks	164.0	67.7	172.2	71.4
Gestat. length ≤ 31 wks	40.2	10.2	42.4	11.1
Gestat. length ≤ 27 wks	17.0	3.8	18.3	4.1
Sample Size	639,189	2,882,747	334,278	2,300,024

Notes: See notes to Panel A. Sample contains singleton births between 1979 and 2000 to non-Hispanic black and white women who are born in New York and Pennsylvania between 1955 and 1975.

Table 3: Difference-in-differences estimates of 1960-1962 to 1968-1970 change in birth cohort effects, for United States-born and foreign-born women
(estimated standard errors in parentheses)

	1968-1970 minus 1960-1962 Birth cohort differences (per 1,000 live births)					
	Unadjusted black-white differences			Regression-adjusted black-white diffs		
	U.S.-born	Foreign-born	U.S. – Foreign	U.S.-born	Foreign-born	U.S. – Foreign
Mother's Health						
Health Risk Factor	-41.22***	-2.56	-38.67***	-32.72***	-4.26	-28.46***
	(1.03)	(2.63)	(2.83)	(1.32)	(3.66)	(3.89)
Herpes	4.34***	3.89***	0.44	5.55***	4.42***	1.13*
	(0.21)	(0.45)	(0.50)	(0.27)	(0.63)	(0.69)
Infant Health Outcomes						
Birth Weight < 2500 g	-12.12***	0.02	-12.14***	-32.71***	-3.57*	-29.13***
	(0.44)	(1.32)	(1.39)	(0.68)	(1.97)	(2.09)
Birth Weight < 1500 g	-1.03***	0.62	-1.65**	-8.06***	-3.54***	-4.52***
	(0.21)	(0.70)	(0.73)	(0.32)	(1.06)	(1.10)
Birth Weight < 1000 g	-0.25*	0.41	-0.65	-4.34***	-2.01**	-2.33***
	(0.15)	(0.55)	(0.57)	(0.24)	(0.83)	(0.87)
5 min. APGAR ≤ 8	-11.11***	4.34**	-15.45***	-24.57***	-6.58**	-17.98***
	(0.55)	(1.83)	(1.91)	(0.88)	(2.94)	(3.07)
5 min. APGAR ≤ 7	-5.14***	1.03	-6.17***	-11.38***	-2.55	-8.83***
	(0.35)	(1.13)	(1.18)	(0.55)	(1.83)	(1.91)
5 min. APGAR ≤ 6	-3.10***	0.31	-3.41***	-6.82***	-0.69	-6.12***
	(0.26)	(0.86)	(0.89)	(0.41)	(1.39)	(1.45)
Gestat length ≤ 36 wks	-7.90***	-2.24	-5.67***	-22.44***	-5.55**	-16.89***
	(0.53)	(1.61)	(1.70)	(0.80)	(2.43)	(2.56)
Gestat length ≤ 31 wks	-0.28	0.25	-0.54	-8.05***	-4.10***	-3.95***
	(0.27)	(0.81)	(0.86)	(0.41)	(1.22)	(1.29)
Gestat length ≤ 27 wks	0.01	0.55	-0.54	-3.80***	-2.16**	-1.65*
	(0.18)	(0.58)	(0.60)	(0.27)	(0.87)	(0.91)

Notes: See notes to Table 2. ***, **, and * indicate statistical significance at the 1, 5, and 10 percent levels, respectively. Results based on linear probability regressions. Standard errors corrected for heteroskedasticity. Sample contains the 1960-1962 and 1968-1970 birth cohorts. The "Regression-adjusted" specifications allow for unrestricted dummies for survey year, marital status, educational categories, and two-year age categories that vary by race and for US-born and foreign-born women.

Table 4: Difference-in-differences-in-differences estimates for United States-born versus foreign-born women,
(estimated standard errors in parentheses)

Outcomes (per 1,000)	Unadjusted	Regression 1	Regression 2	Regression 3
Mother's Health				
Health Risk Factor	-38.67***	-23.86***	-26.16***	-28.46***
	(2.83)	(2.84)	(3.26)	(3.89)
Herpes	0.44	1.00**	1.35**	1.13*
	(0.50)	(0.51)	(0.58)	(0.69)
Sample Size	6,070,029	6,070,029	6,070,029	6,070,029
Infant Health Outcomes				
Birth Weight < 2500 grams	-12.14***	-13.08***	-29.02***	-29.13***
	(1.39)	(1.42)	(1.87)	(2.09)
Birth Weight < 1500 grams	-1.65**	-1.65**	-4.47***	-4.52***
	(0.73)	(0.75)	(0.99)	(1.10)
Birth Weight < 1000 grams	-0.65	-0.49	-1.98***	-2.33***
	(0.57)	(0.59)	(0.78)	(0.87)
Sample Size	11,708,986	11,708,986	11,708,986	11,708,986
5 min. APGAR <= 8	-15.45***	-14.62***	-16.43***	-17.98***
	(1.91)	(1.95)	(2.71)	(3.07)
5 min. APGAR <= 7	-6.17***	-5.14***	-8.54***	-8.83***
	(1.18)	(1.20)	(1.69)	(1.91)
5 min. APGAR <= 6	-3.41***	-2.80***	-5.41***	-6.12***
	(0.89)	(0.91)	(1.29)	(1.45)
Sample Size	8,446,258	8,446,258	8,446,258	8,446,258
Gestat length $\leq$ 36 wks	-5.67***	-13.02***	-21.73***	-16.89***
	(1.70)	(1.73)	(2.26)	(2.56)
Gestat length $\leq$ 31 wks	-0.54	-2.95***	-5.80***	-3.95***
	(0.86)	(0.87)	(1.16)	(1.29)
Gestat length $\leq$ 27 wks	-0.54	-1.36**	-2.16***	-1.65*
	(0.60)	(0.61)	(0.82)	(0.91)
Sample Size	11,346,865	11,346,865	11,346,865	11,346,865

Notes: See notes to Tables 2 and 3. ***, **, and * indicate statistical significance at the 1, 5, and 10 percent levels, respectively. Results based on linear probability regressions. Standard errors corrected for heteroskedasticity. Sample contains the 1960-1962 and 1968-1970 birth cohorts. Regression 1 allows for unrestricted dummies for age, survey year, marital status, and educational categories that vary by race but constrains them to be the same for US- and foreign-born mothers. Regression 2 only constrains the race-specific age effects to be the same by US-born and foreign-born status. Regression 3 includes interactions of race-specific, two-year age categories with US-born and foreign-born status.

Table 5: Difference-in-differences estimates of 1960-1962 to 1968-1970 change in birth cohort effects, for United States-born from the National Health Interview Surveys
(estimated standard errors in parentheses)

Table 6: Mississippi- and Alabama-born mother's, difference-in-differences-in-differences estimates (estimated standard errors in parentheses)

Infant outcomes (per 1,000)	Unadjusted	Probit 1	Probit 2
Birth Weight < 1000 grams	-1.62**	-1.63***	-2.00***
	(0.67)	(0.55)	(0.57)
Birth Weight < 1500 grams	-2.47**	-2.43***	-2.51***
	(1.07)	(0.85)	(0.94)
Birth Weight < 2500 grams	-4.21	-3.68*	-4.63*
	(3.71)	(2.22)	(2.47)
Sample Size	802,353	802,353	802,353
5 min. APGAR <= 6	-2.64	-2.56**	-3.63***
	(1.66)	(1.09)	(1.16)
5 min. APGAR <= 7	-4.56*	-4.58***	-6.12***
	(2.62)	(1.60)	(1.70)
5 min. APGAR <= 8	-6.79	-6.91**	-5.92*
	(5.19)	(3.00)	(3.15)
Sample Size	641,488	641,488	641,488

Notes: ***, **, and * indicate statistical significance at the 1, 5, and 10 percent levels, respectively. Entries are the implied probability effects derived from probit models. Standard errors are corrected for heteroskedasticity and clustering at the state-race-year-age level. Sample contains the 1961-1964 and 1966-1969 birth cohorts. Probit 1 allows for unrestricted dummies for age, survey year, marital status, and educational categories that vary by race but constrains them to be the same for Mississippi- and Alabama-born mothers. Probit 2 only constrains race-specific age effects to be the same by state of mother's.

Table 7: New York- and Pennsylvania-born mother's, difference-in-differences-in-differences estimates (estimated standard errors in parentheses)

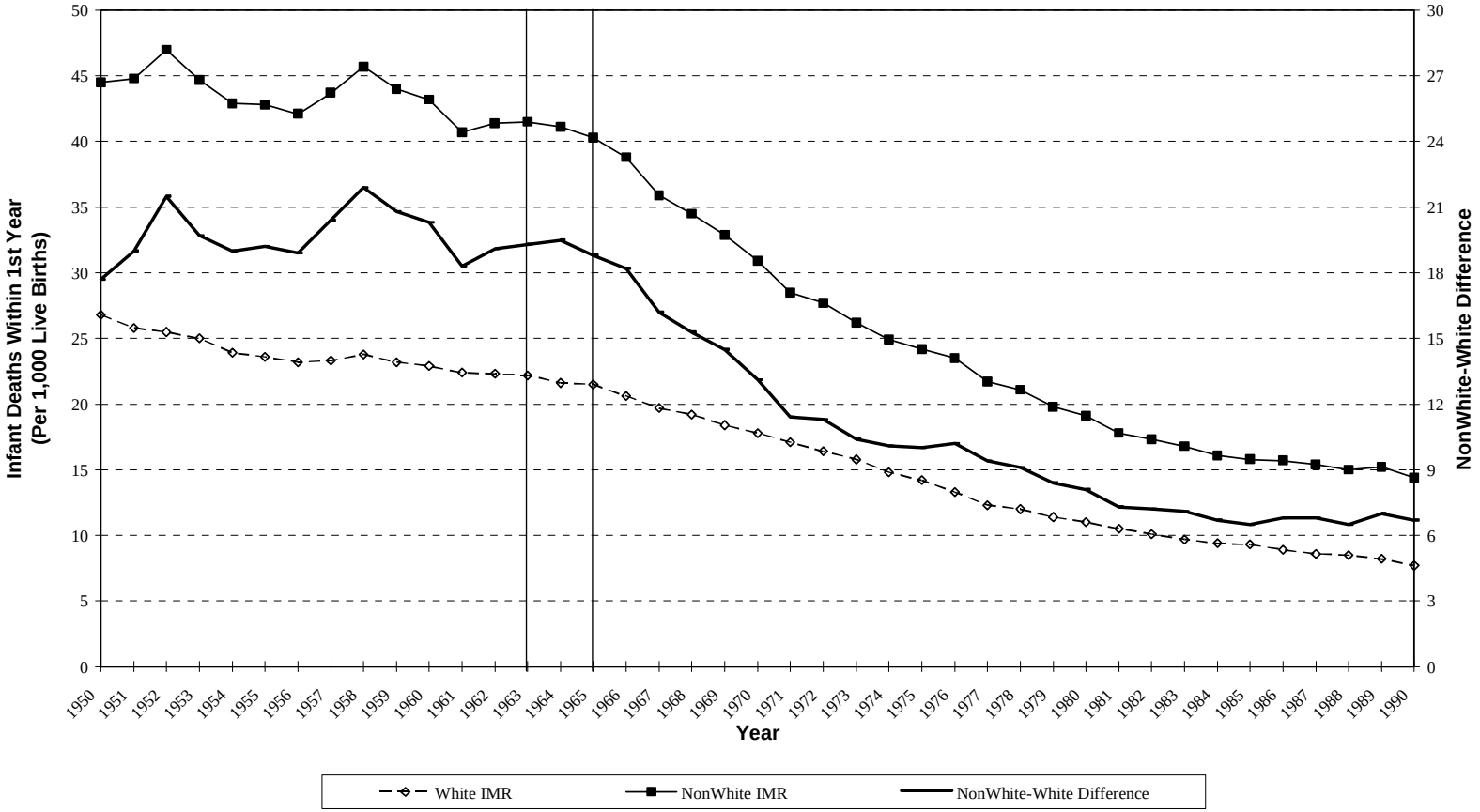
Infant outcomes (per 1,000)	Unadjusted	Probit 1	Probit 2
Birth Weight < 1000 grams	-0.33	-0.33	-0.28
	(0.31)	(0.28)	(0.30)
Birth Weight < 1500 grams	-0.42	-0.44	-0.33
	(0.47)	(0.41)	(0.45)
Birth Weight < 2500 grams	-0.93	-0.49	-0.62
	(1.25)	(1.13)	(1.22)
Sample Size	2,848,329	2,848,329	2,848,329
5 min. APGAR <= 6	-0.32	-0.41	-0.90
	(1.17)	(0.58)	(0.64)
5 min. APGAR <= 7	1.12	0.93	-1.29
	(2.16)	(1.01)	(1.04)
5 min. APGAR <= 8	4.92	4.56**	-2.80
	(5.08)	(2.07)	(1.79)
Sample Size	2,197,296	2,197,296	2,197,296

Notes: ***, **, and * indicate statistical significance at the 1, 5, and 10 percent levels, respectively. Entries are the implied probability effects derived from probit models. Standard errors are corrected for heteroskedasticity and clustering at the state-race-year-age level. Sample contains the 1961-1964 and 1966-1969 birth cohorts. Probit 1 allows for unrestricted dummies for age, survey year, marital status, and educational categories that vary by race but constrains them to be the same for New York- and Pennsylvania-born mothers. Probit 2 only constrains race-specific age effects to be the same by state of mother's.

Appendix Table 1:

Year Of Birth	Survey Year																					
	1979	1980	1981	1982	1983	1984	1985	1986	1987	1988	1989	1990	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000
1955	24	25	26	27	28	29	30	31	32	33	34	35										
1956	23	24	25	26	27	28	29	20	31	32	33	34	35									
1957	22	23	24	25	26	27	28	29	30	31	32	33	34	35								
1958	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35							
1959	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35						
1960	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35					
1961	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35				
1962	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35			
1963	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35		
1964		16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	
1965			16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35
1966				16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34
1967					16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33
1968						16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32
1969							16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31
1970								16	17	18	19	20	21	22	23	24	25	26	27	28	29	30
1971									16	17	18	19	20	21	22	23	24	25	26	27	28	29
1972										16	17	18	19	20	21	22	23	24	25	26	27	28
1973											16	17	18	19	20	21	22	23	24	25	26	27
1974												16	17	18	19	20	21	22	23	24	25	26
1975													16	17	18	19	20	21	22	23	24	25

Figure 1: Trends in the Infant Mortality Rate by Race, 1950-1990



Note: Alaska included in 1959 and Hawaii in 1960.
Source: Various Issues of *Vital Statistics of the United States* .

Figure 2: Postneonatal Mortality Rates by Race for Selected States, 1941-1971

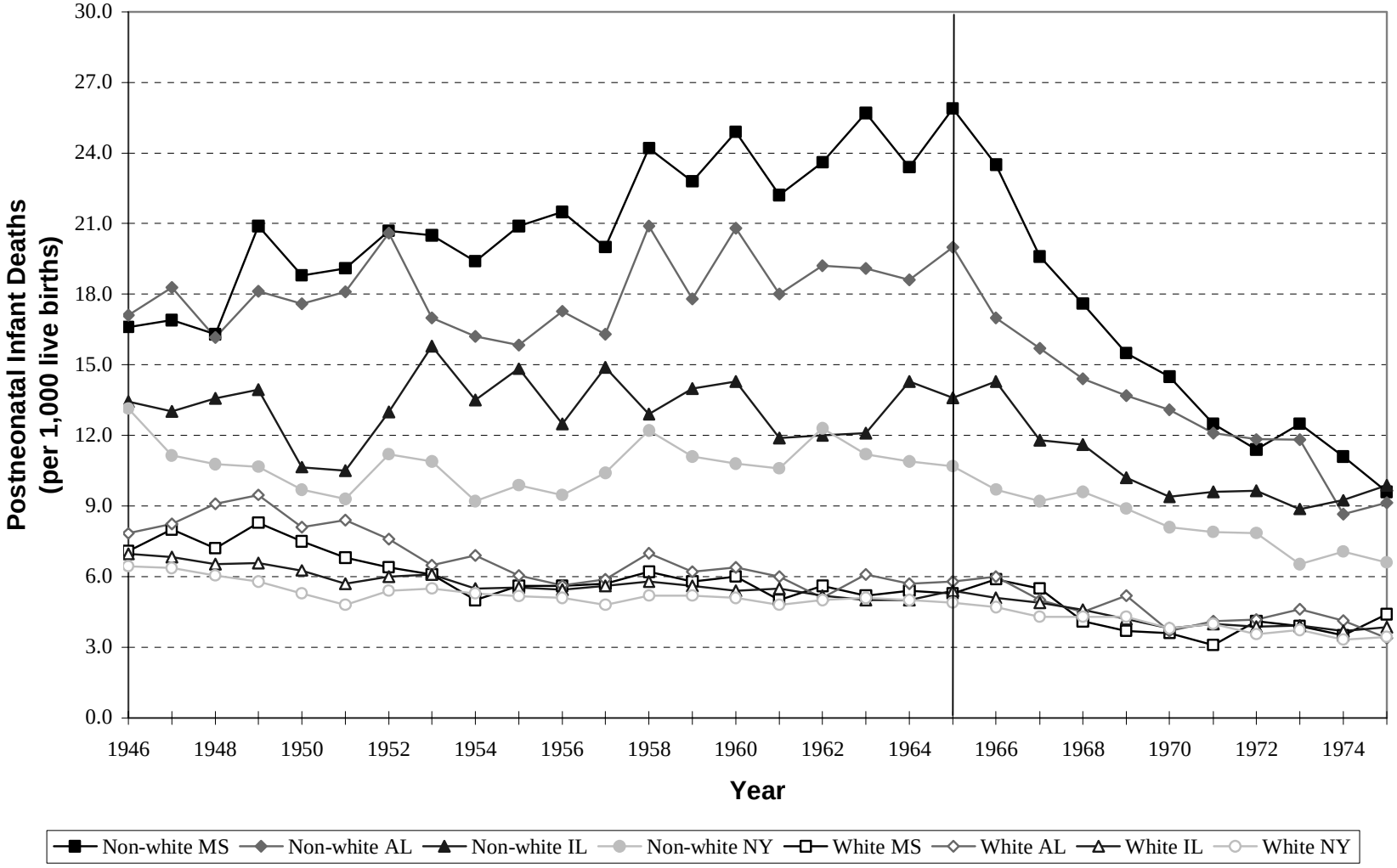
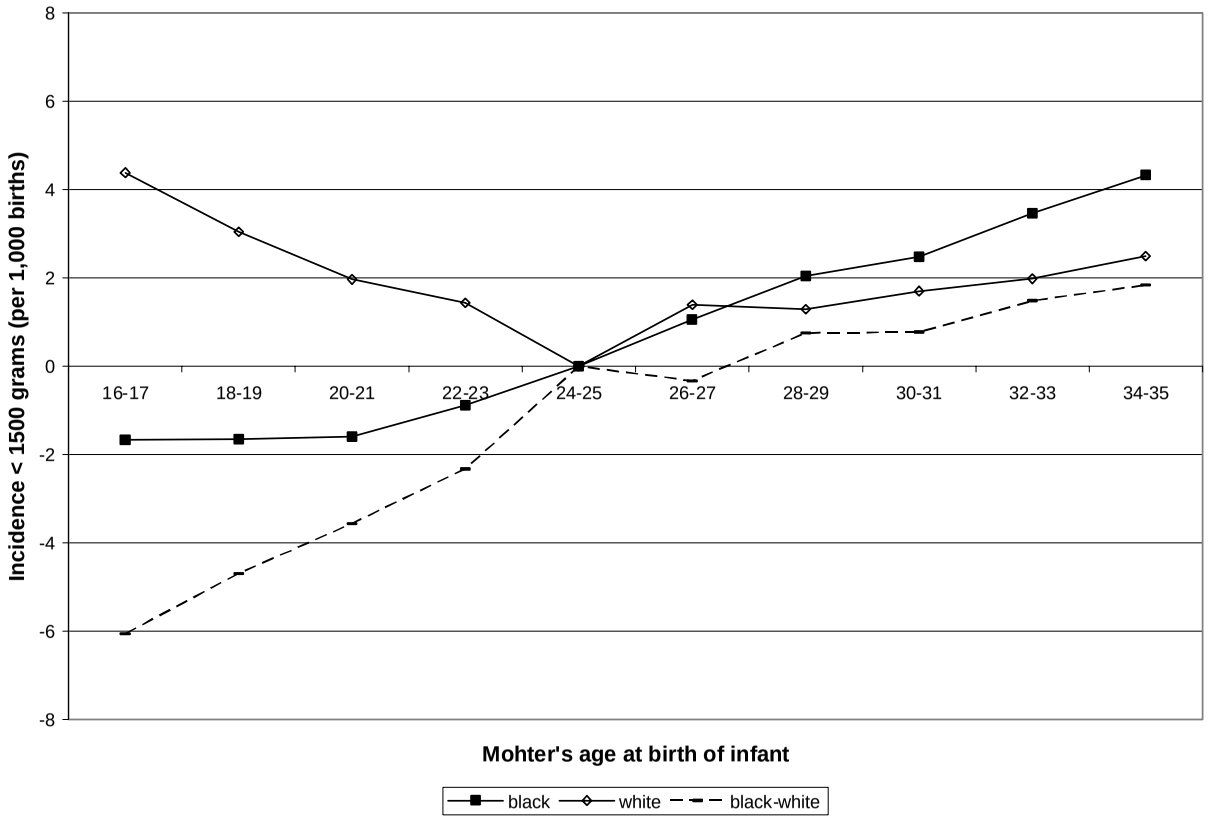
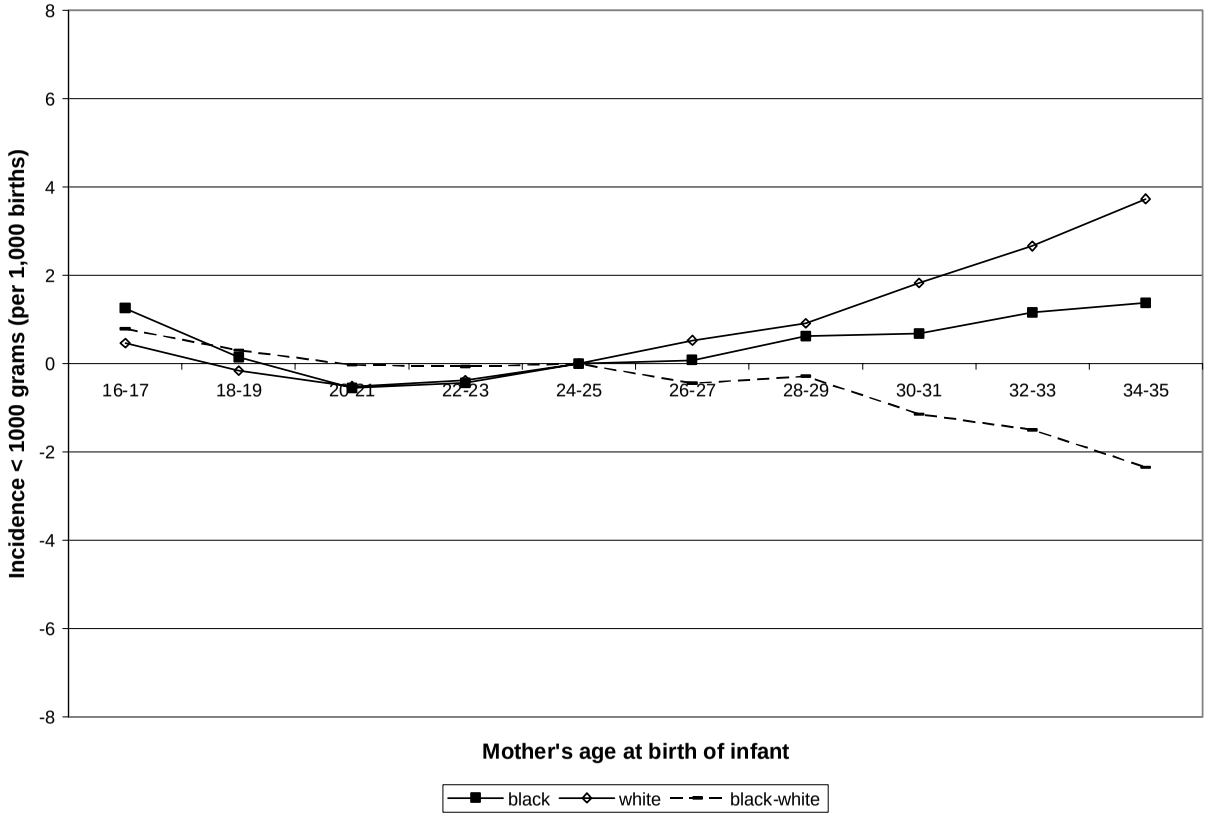


Figure 3: Mother's Age Effects in Infant Health in the United States,
Women born in 1955 to 1975, who gave birth in 1979 to 2000

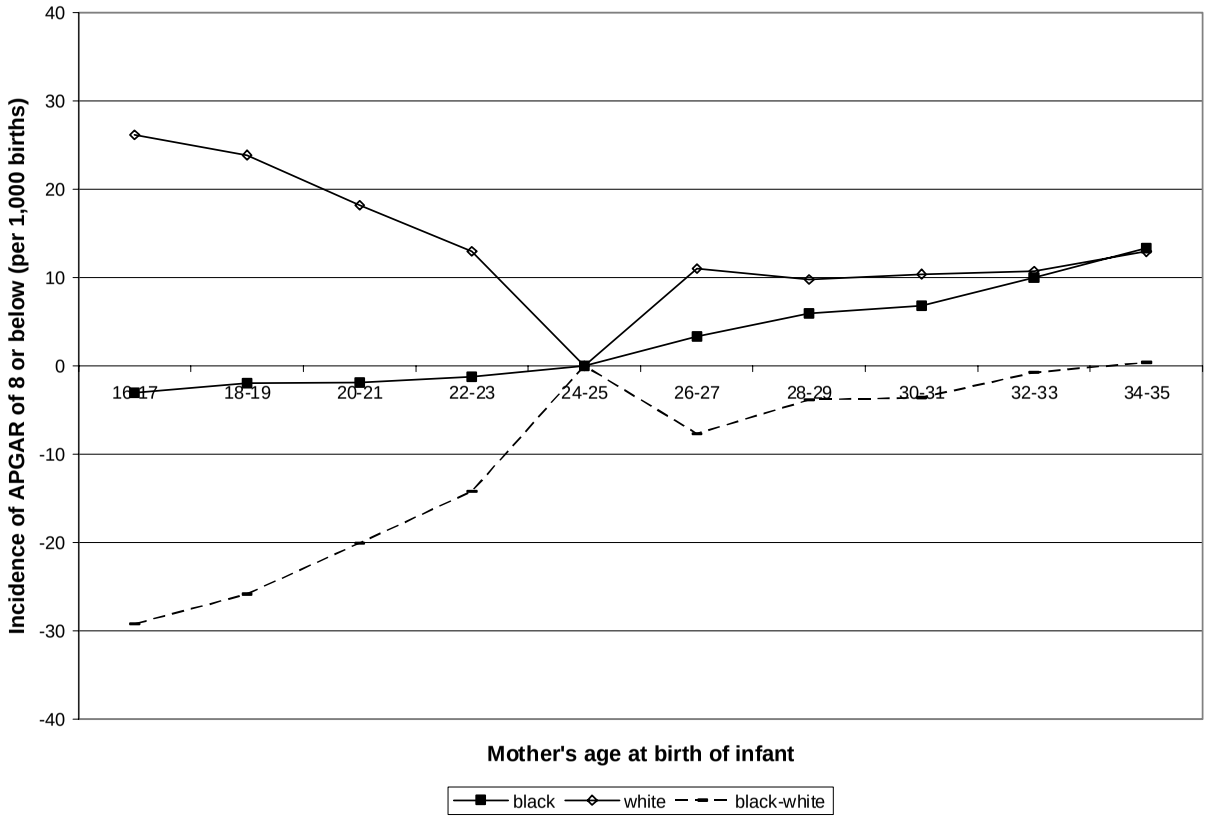
A. Incidence of infant birth below 1500 grams – no controls for year-of-birth of the mother



B. Incidence of infant birth below 1500 grams – controls for year-of-birth of the mother



C. Incidence of infant with 5-minute APGAR of 8 or below – no controls for mother's year-of-birth



D. Incidence of infant with 5-minute APGAR of 8 or below – controls for mother's year-of-birth

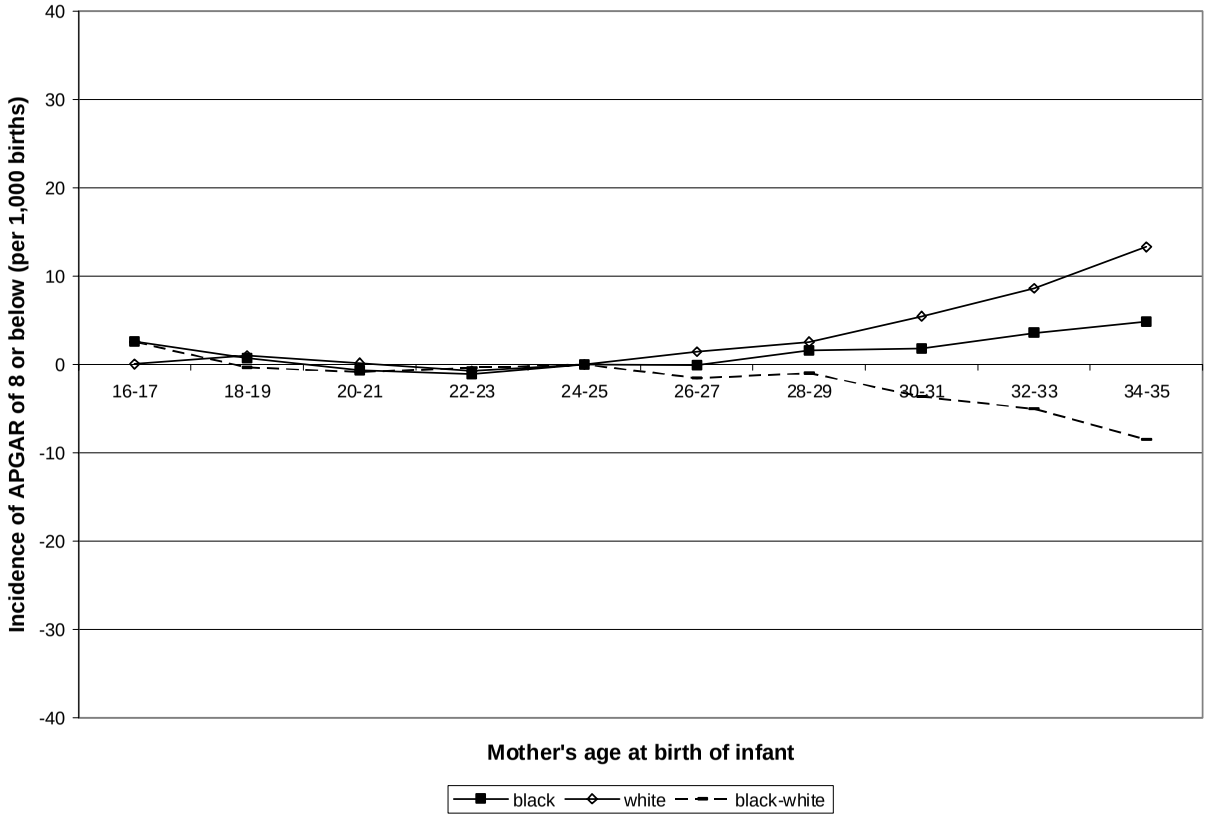


Figure 4: Regressions-adjusted mother's year-of-birth effects on risk of extremely low birth weight (less than 1000 grams)

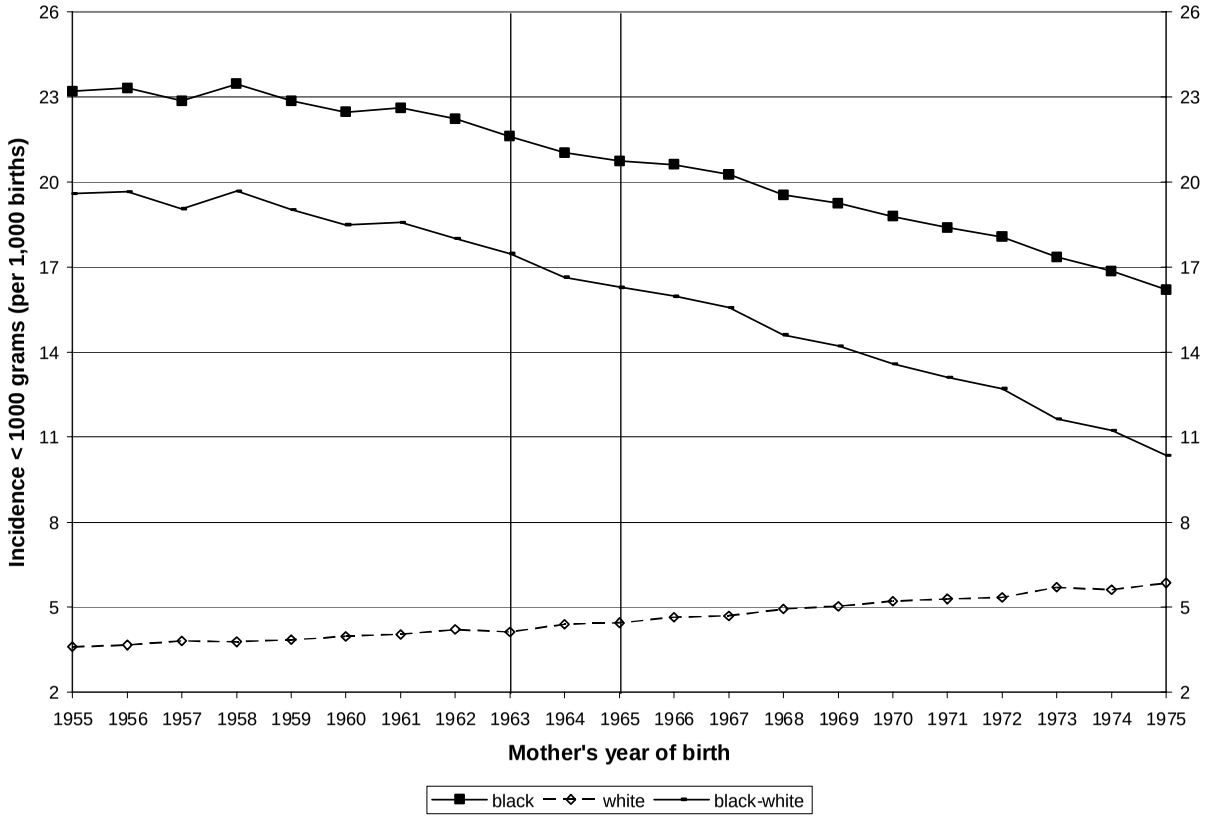
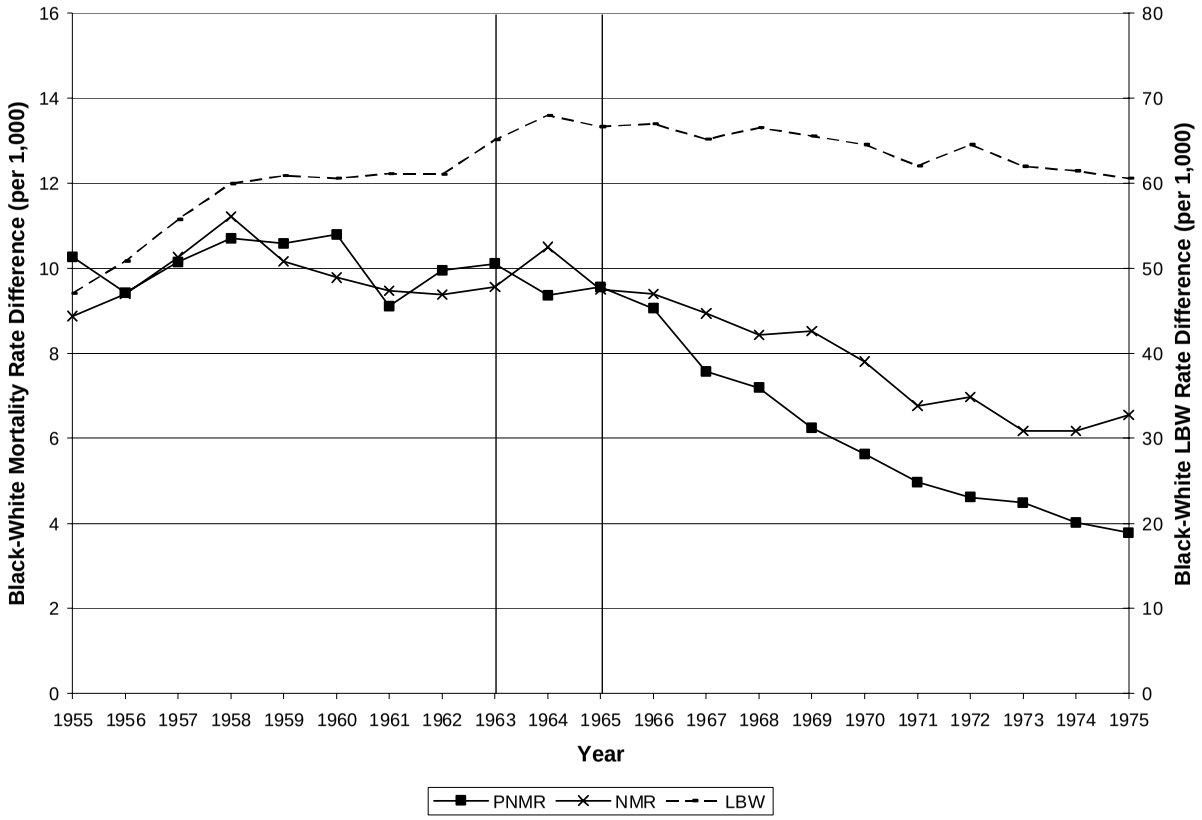
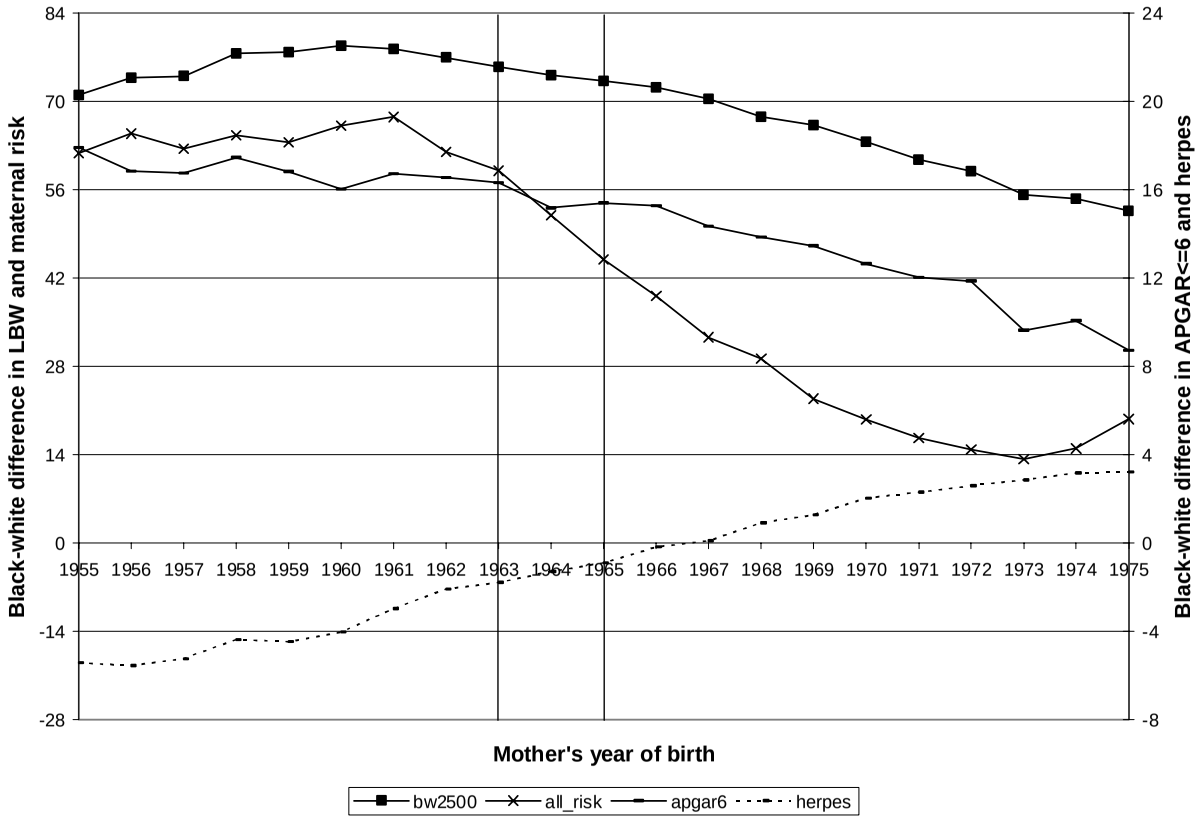


Figure 5: Black-white differences in the United States, by year and by year-of-birth

A. Black-white differences in infant mortality rates and low birth weight incidence in U.S.



B. Raw black-white differences in outcomes by mother's year of birth (per 1,000)



C. Regression-adjusted black-white differences in outcomes by mother's year of birth (per 1,000)

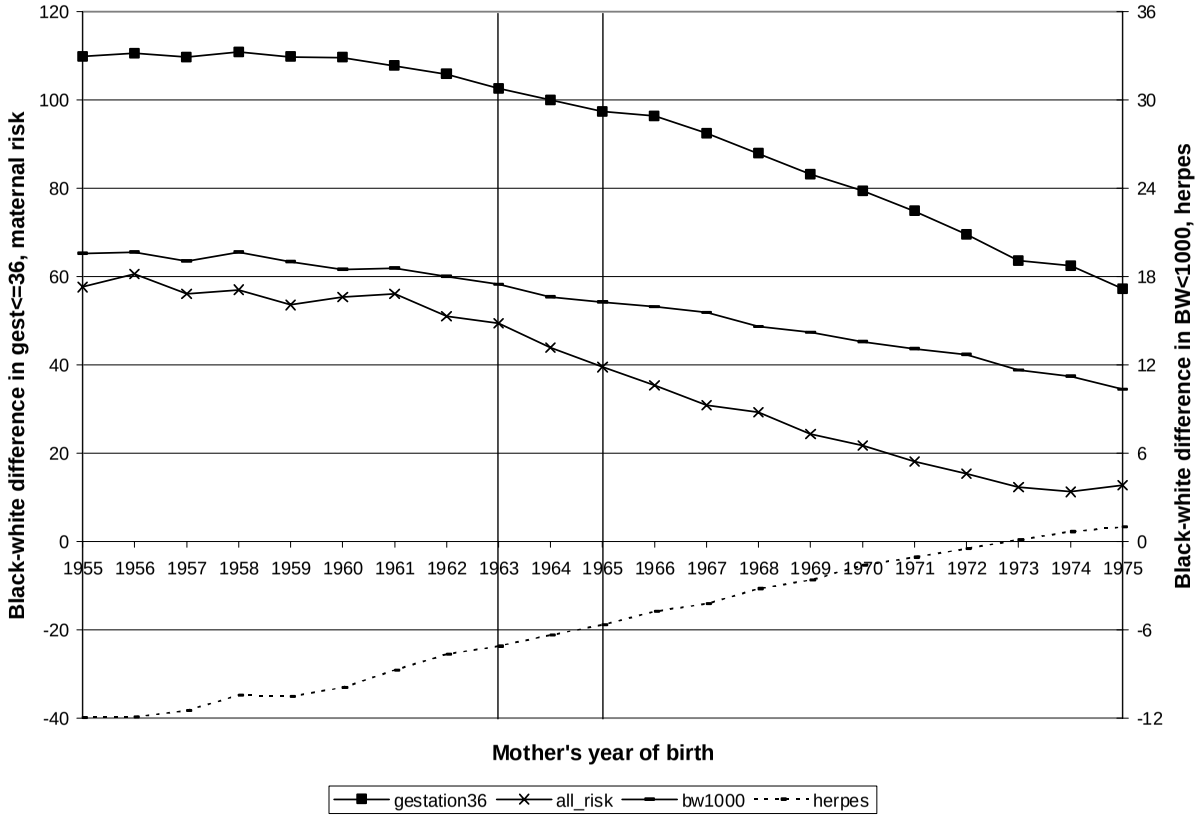
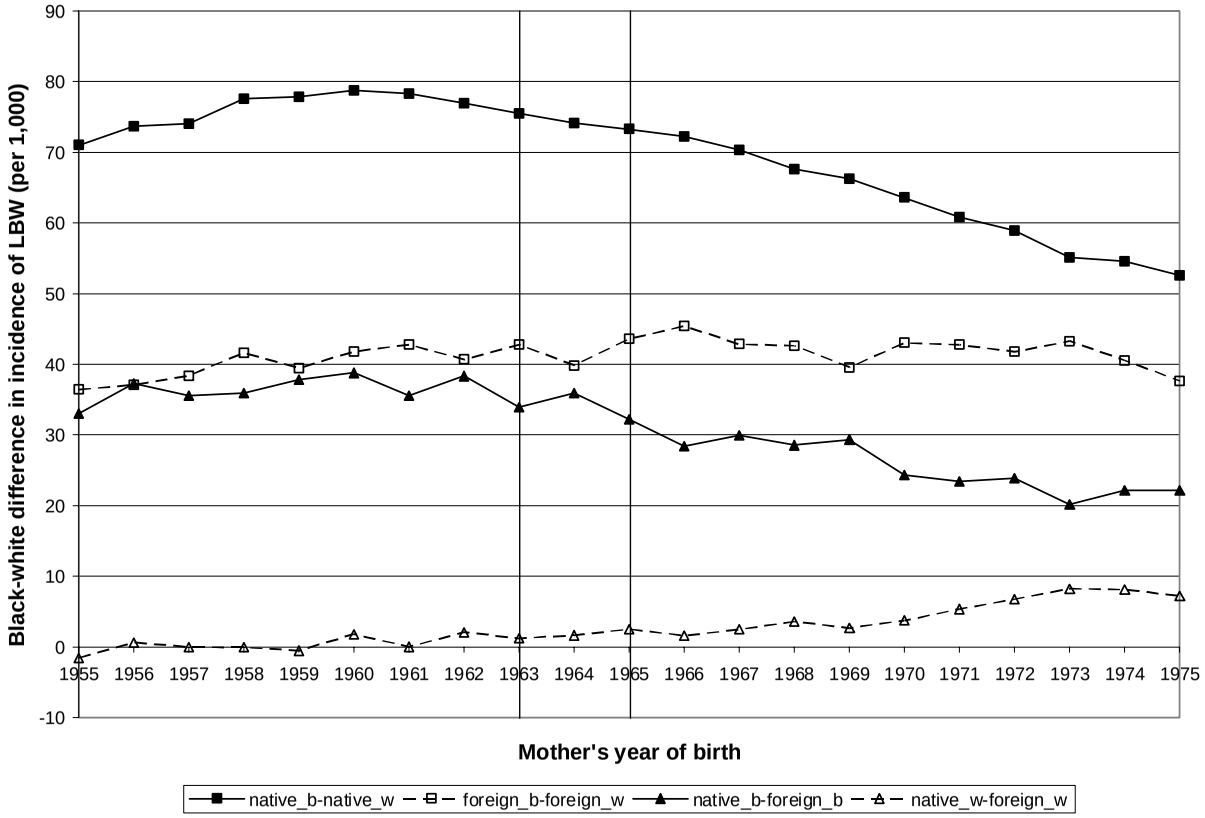


Figure 6: Native versus Foreign-born year-of-birth effects in outcomes of interest

A. Black-white, native-foreign, raw differences in low birth weight incidence (less than 2500 grams)



B. Black-white, native-foreign, raw differences in incidence of maternal health risk

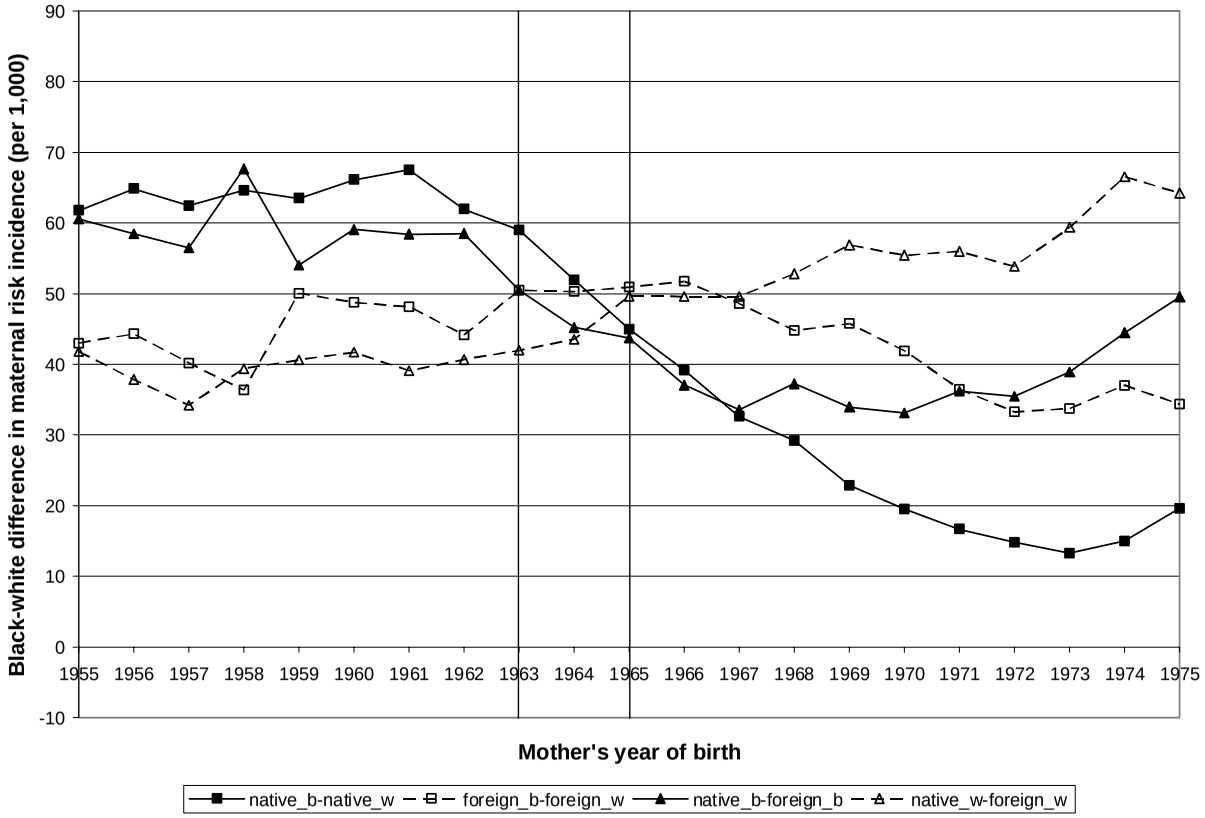
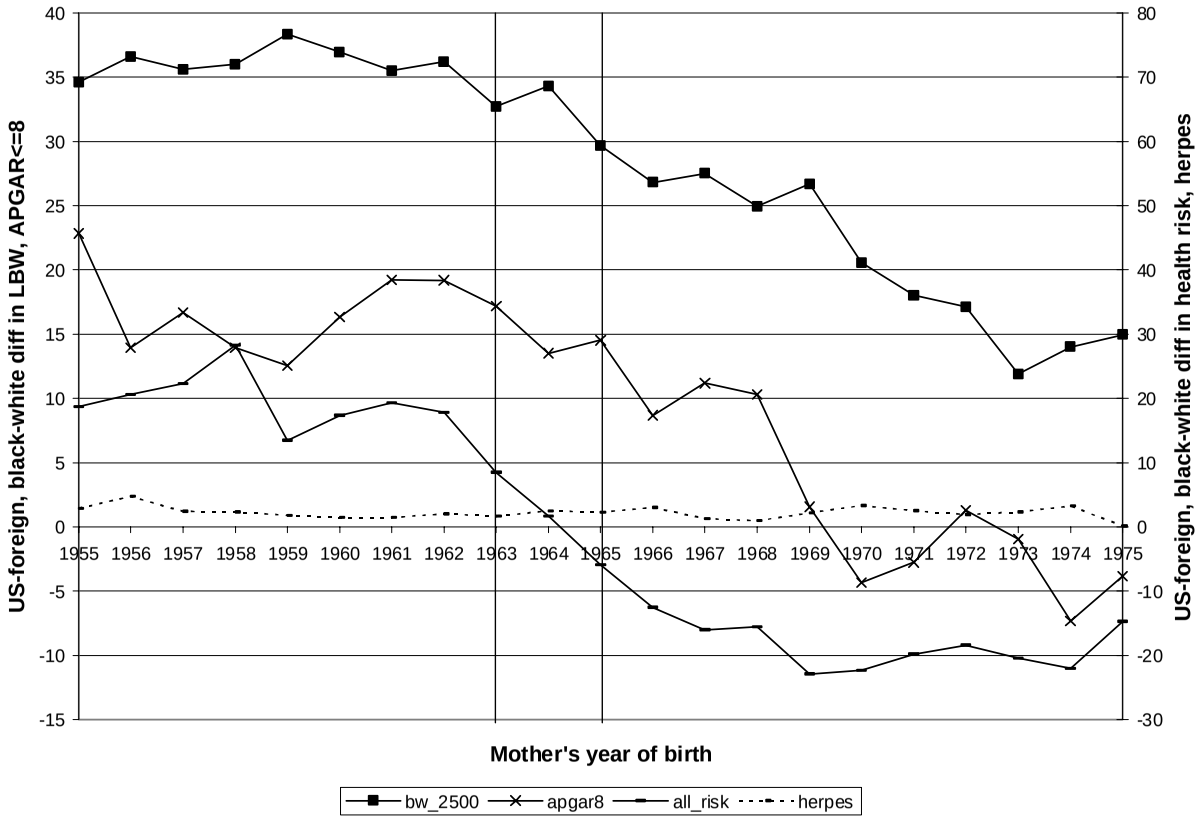


Figure 7: Native versus Foreign-born difference in black-white differences for outcomes

A. Raw difference-in-differences for LBW, APGAR≤8, maternal health risk and herpes (per 1,000)



B. Regression-adjusted difference-in-differences for outcomes (per 1,000)

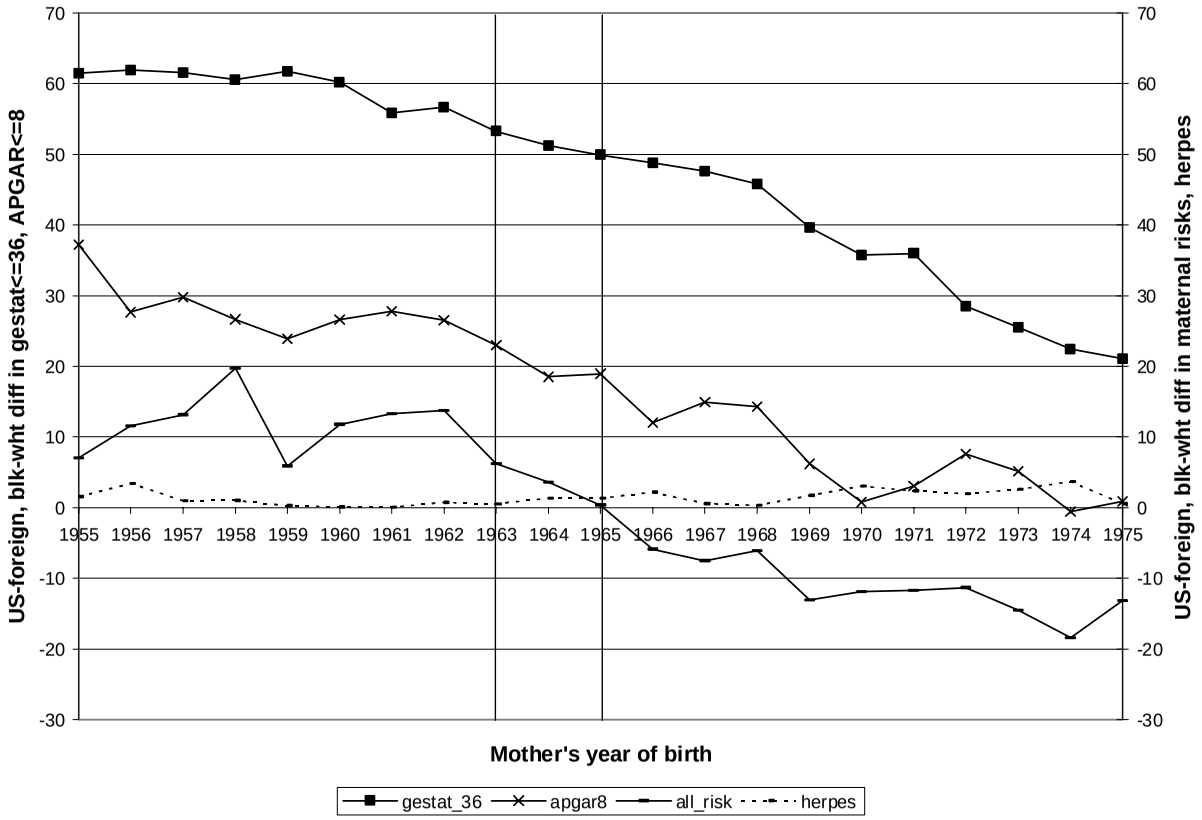
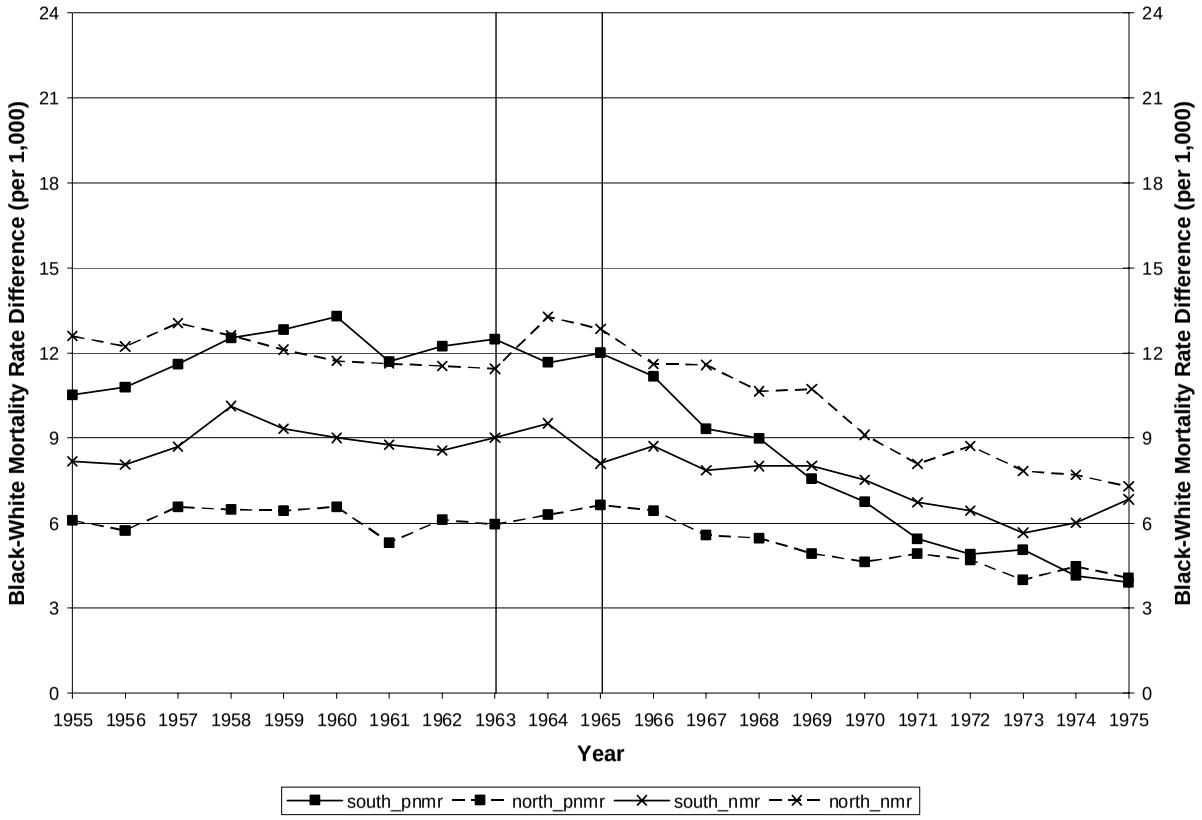


Figure 8: Infant mortality and low birth weight (less than 2,500 grams) incidence in South and Non-South (Mid-Atlantic, East North Central)

A. Black-White differences in infant mortality rates in South and Non-South



B. South-Non-South, black-white difference in infant mortality and low birth weight incidence

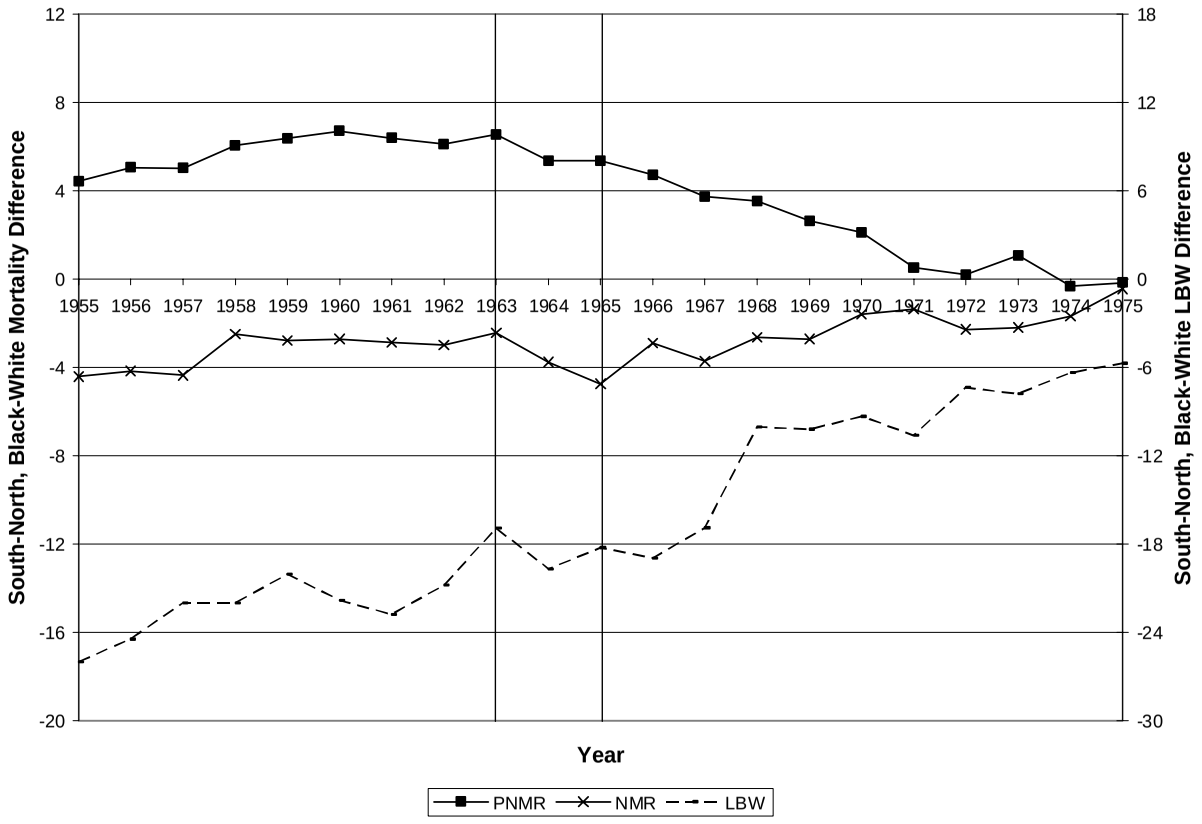
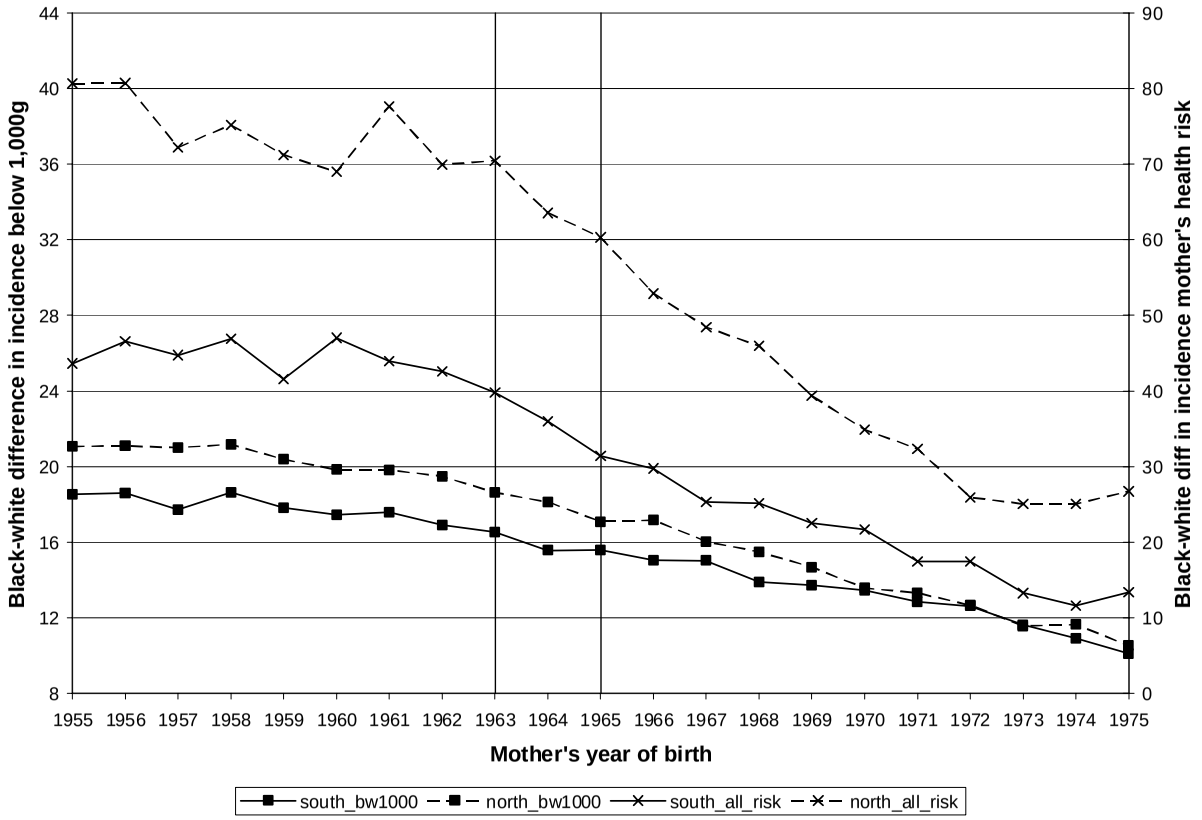


Figure 9: Maternal health risk and infant birth weight by mother's year of birth in South and Non-South

A. Regression-adjusted black-white differences in South and Non-South



B. Raw and regression-adjusted, South-Non-South, black-white differences

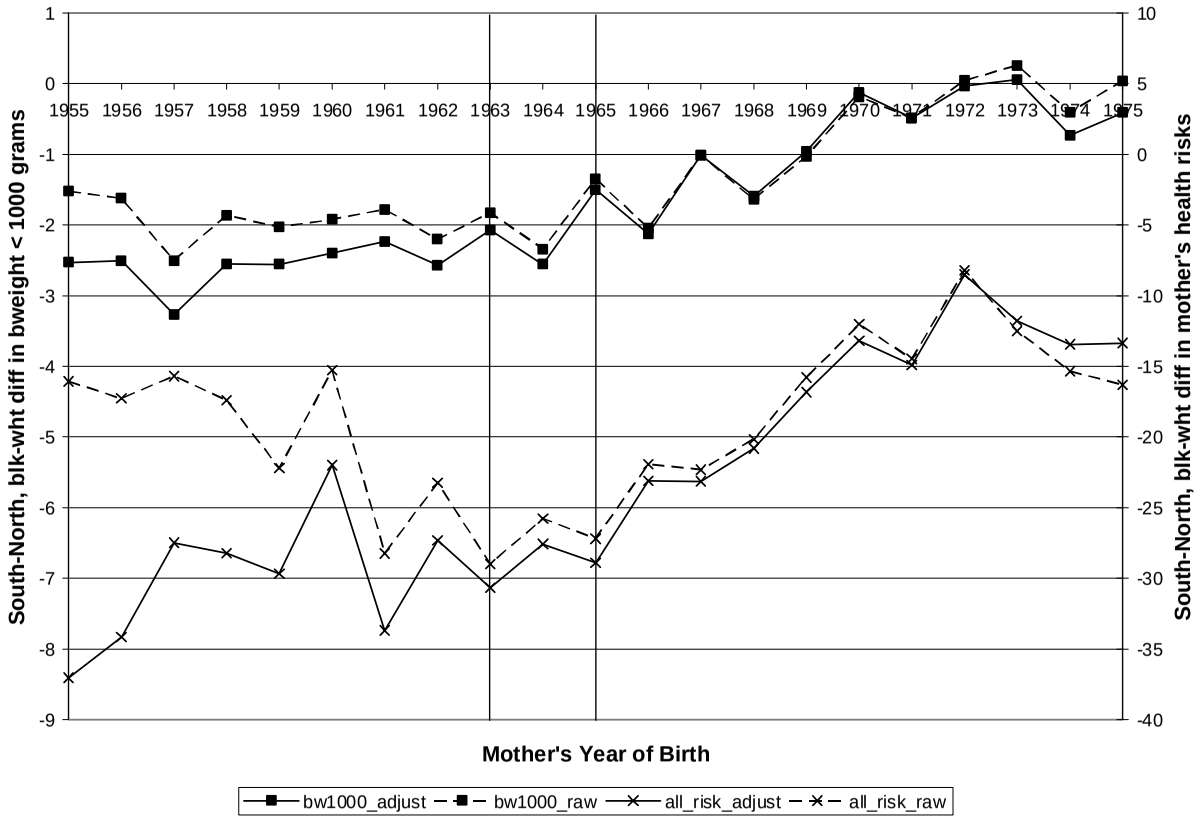
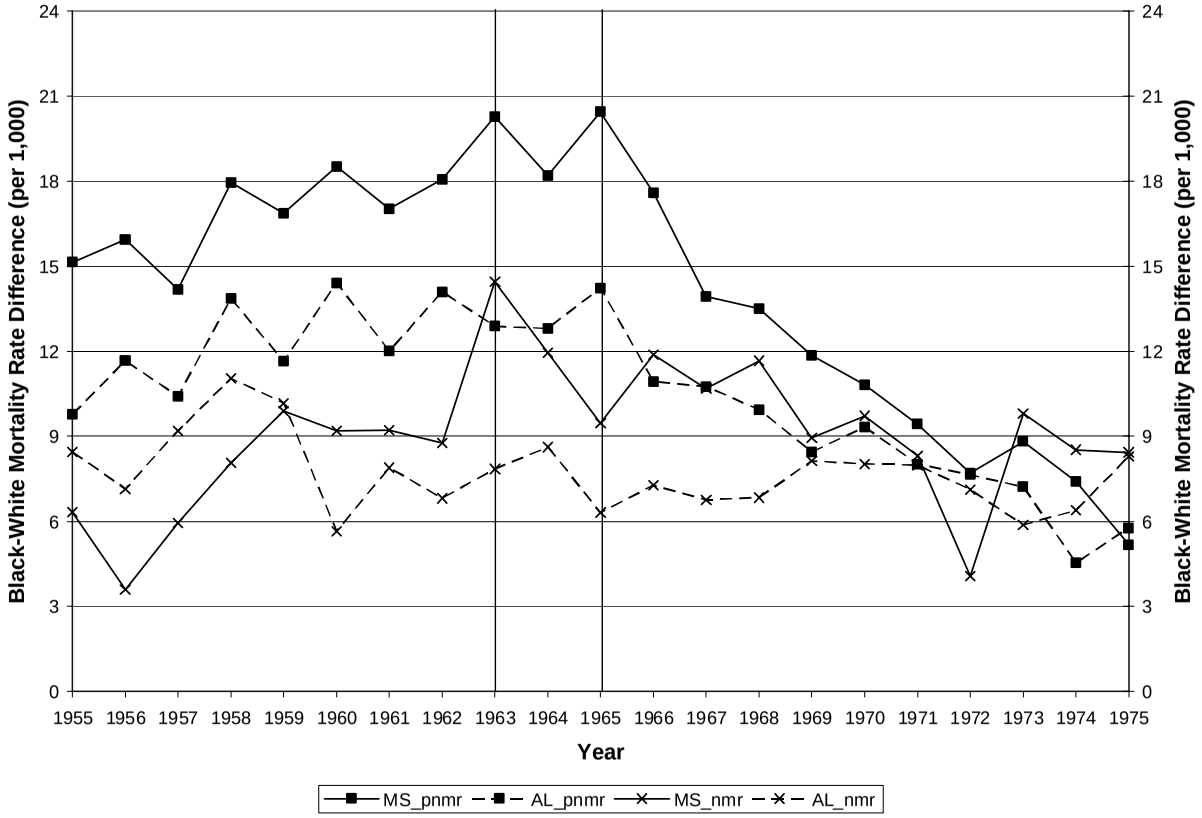
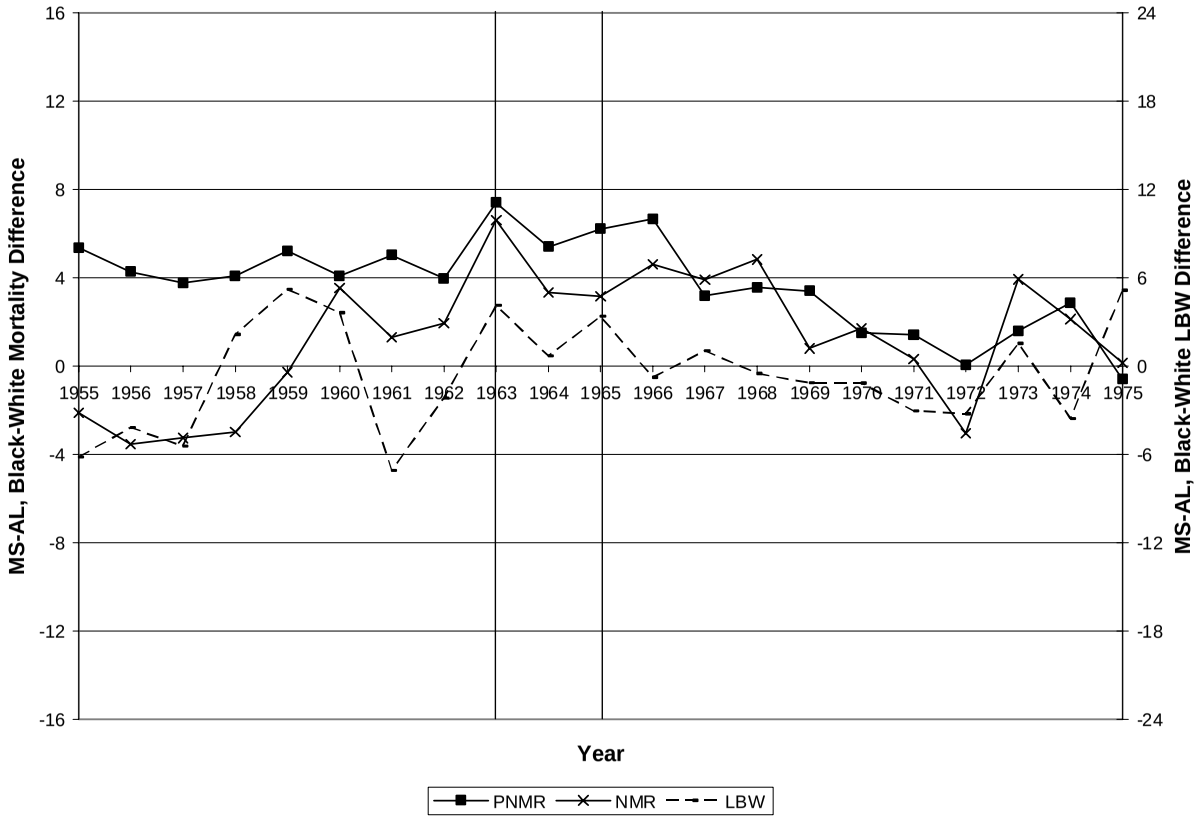


Figure 10: Comparisons of Mississippi and Alabama,

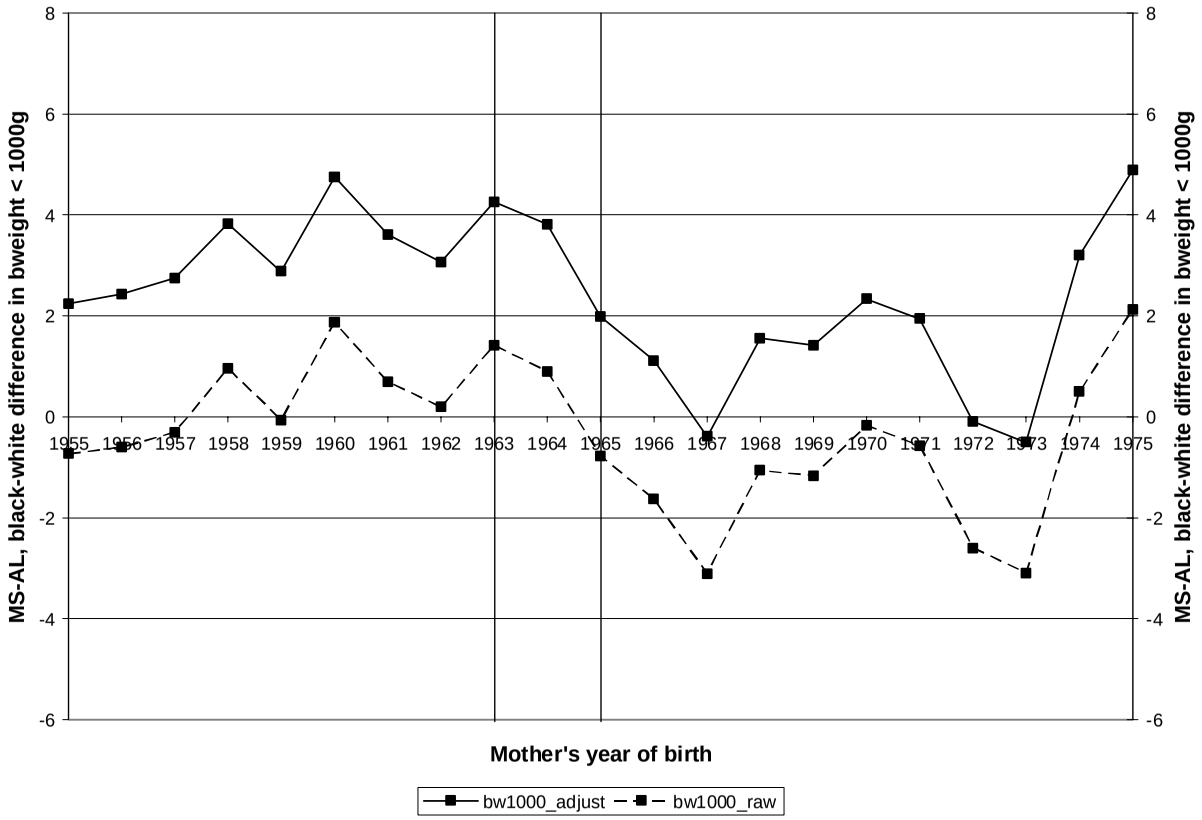
A. Black-White differences in infant mortality rates in Mississippi and Alabama



B. Mississippi-Alabama, black-white difference in infant mortality and low birth weight incidence



C. Raw and regression-adjusted, Mississippi-Alabama, black-white difference in birth weight below 1000 grams



D. Raw and regression-adjusted, Mississippi-Alabama, black-white difference in APGAR of 8 or below

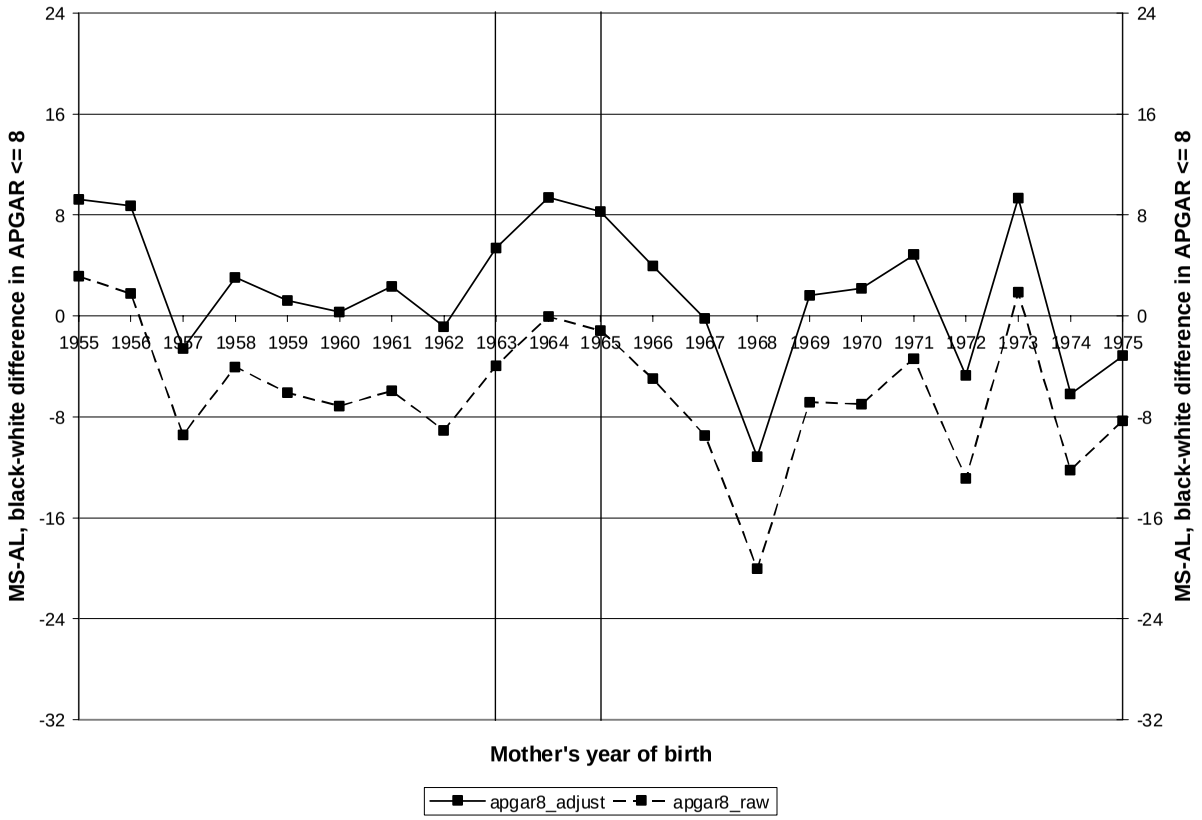
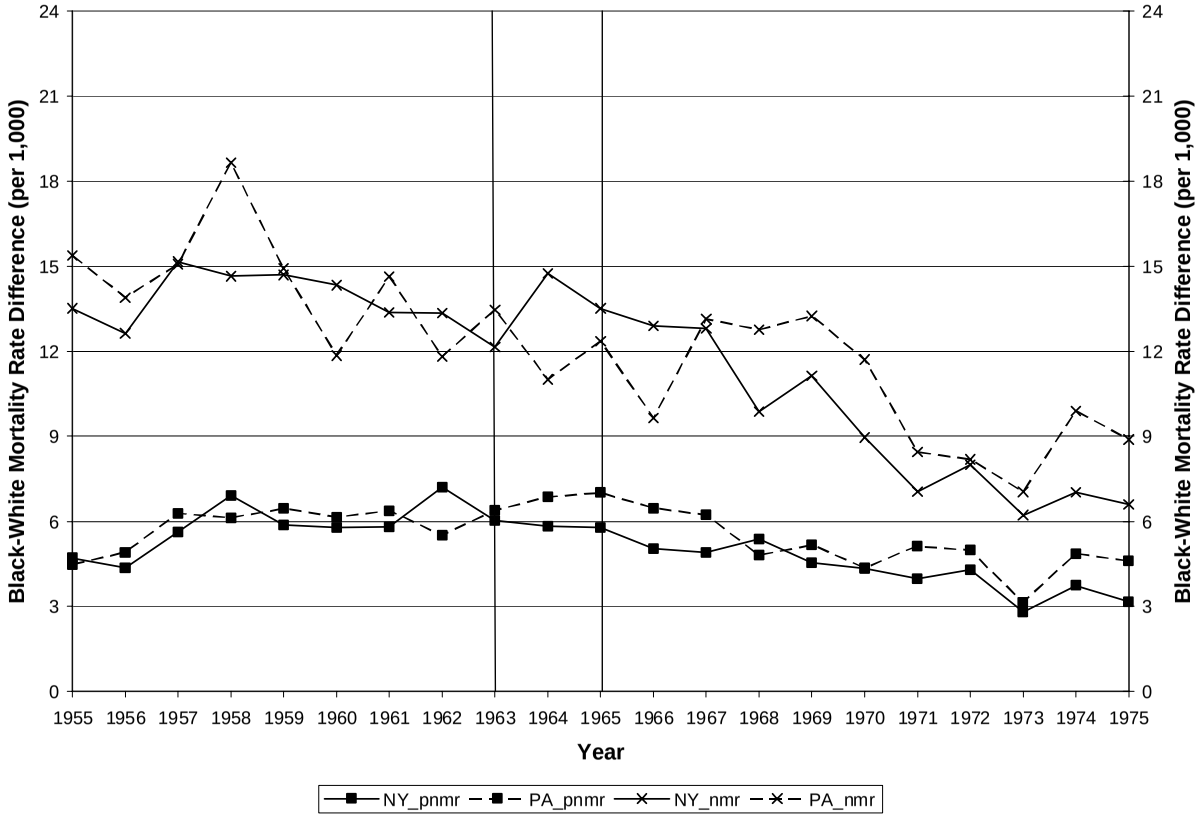
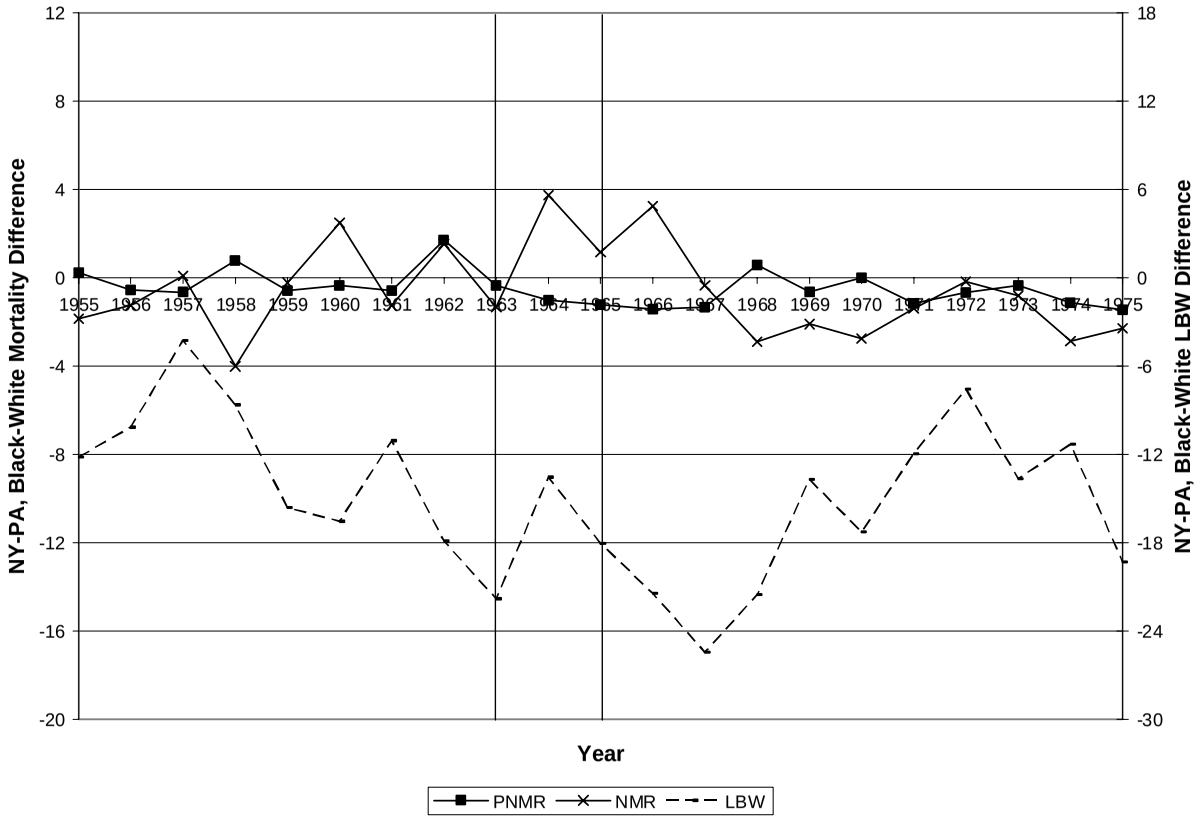


Figure 11: Comparisons of New York and Pennsylvania

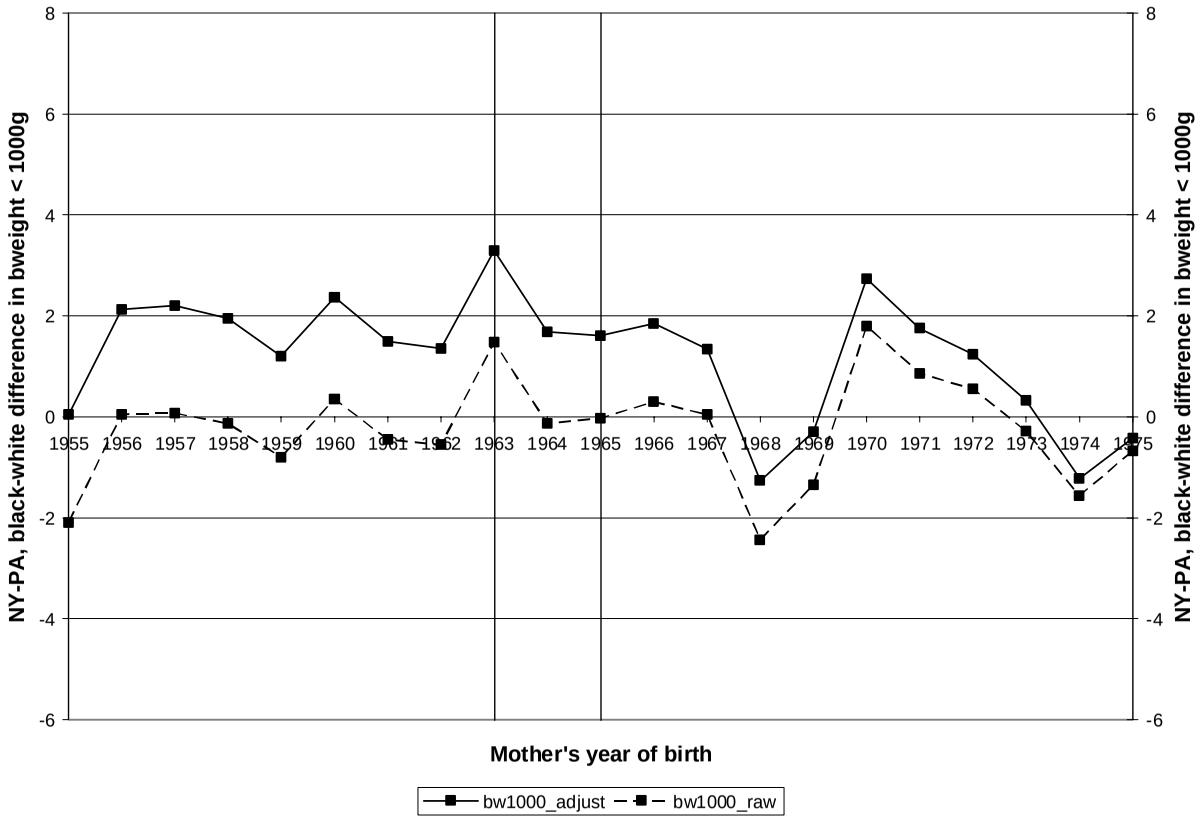
A. Black-White differences in infant mortality rates in New York and Pennsylvania



B. New York-Pennsylvania, black-white difference in infant mortality and low birth weight incidence



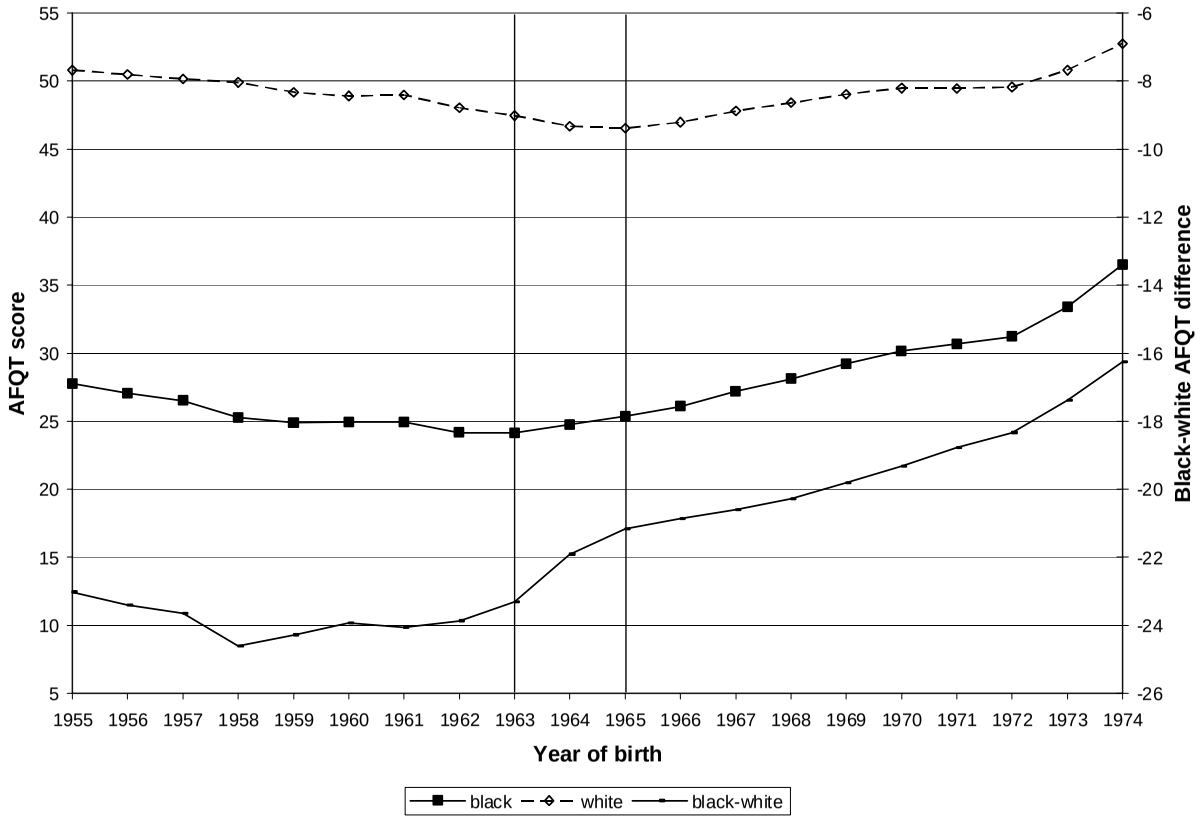
C. Raw and regression-adjusted, New York-Pennsylvania, black-white difference in birth weight below 1000 grams



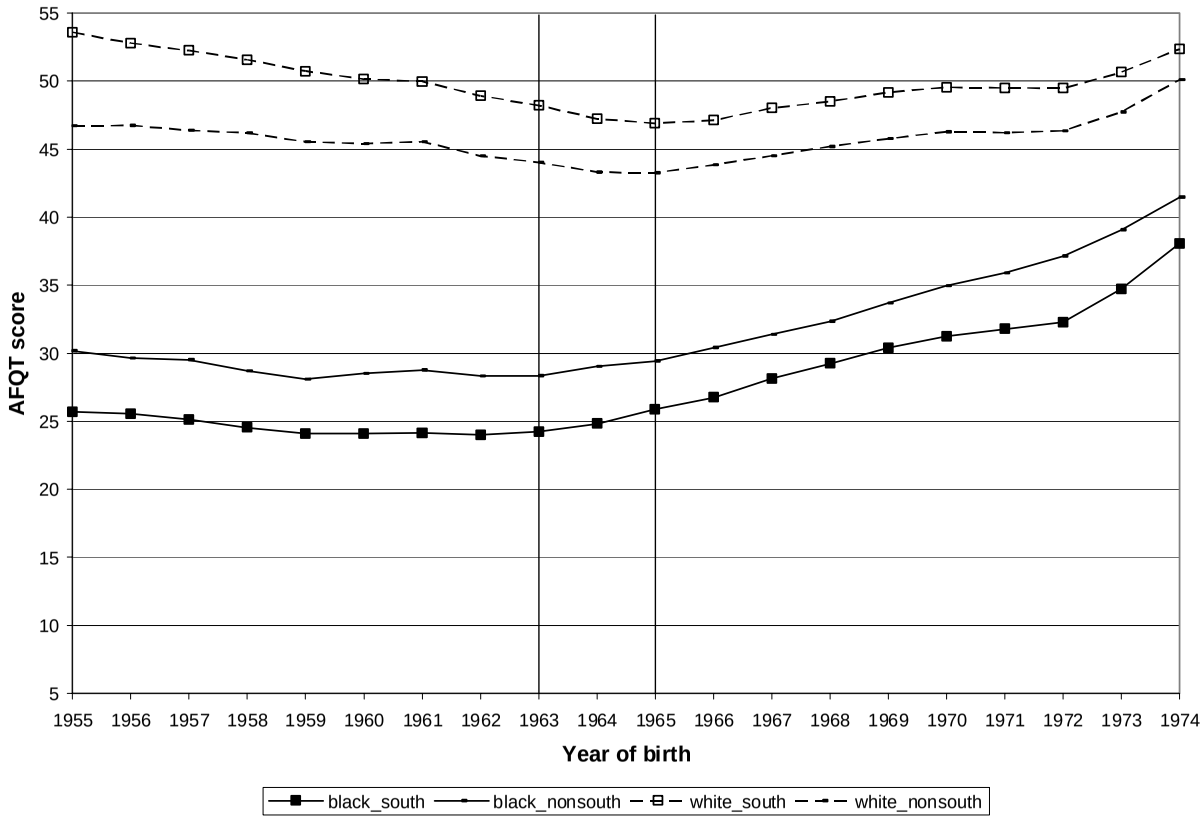
D. Raw and regression-adjusted, New York-Pennsylvania, black-white difference in APGAR of 8 or below

Appendix Figure 1: AFQT scores for men born between 1955-1974 who took exam between 1976-1991

A. United States AFQT scores by race and the black-white difference



B. AFQT scores by race and region – South, Non-South (Mid-Atlantic, East North Central)



C. AFQT scores by race and year in South, unadjusted and adjusted for birth cohort effects