Black and White Disparities in Overall and Cause-Specific Infant Mortality in the U.S., 1983-2002*

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Abstract

Relative disparity between black and white infant mortality in the U.S. has been increasing despite substantial declines in the overall infant mortality rate. Objective: To help account for this phenomenon by analyzing variations in racial disparity trajectories for1983-2002 for the five leading specific causes of infant death. Data: NCHS linked birth/infant death cohort files. Method: We estimated annual bivariate and adjusted changes in cause-specific risk of death for blacks and whites using a multilevel random coefficient model with birth cohort as the second-level unit to capture cross-sectional and temporal variations. Findings: Racial disparities, presented in terms of changes in log-odds, increased for the three causes (congenital anomalies, sudden infant death syndrome, and respiratory distress syndrome) regarding which beneficial innovations in perinatal care emerged. For the other two causes for which no such innovations occurred (short gestation and unspecified low birth weight and maternal complications), little change in disparities was evident.

Black and White Disparities in Overall and Cause-Specific Infant Mortality in the U.S., 1983-2002

The disparities between black and white infant mortality rates in the U.S continue to be an issue that is both troubling and challenging. Black and white differentials in infant mortality rates not only persisted but expanded over the past two decades or so, a time period when a range of cause-specific perinatal care technologies emerged or were expanded. The black/white ratio for the infant mortality rate (IMR) stood at 2.0 in 1980 (Guyer et al. 1998). By 2004, the latest year for which official national statistics are available, the IMR for blacks was nearly 2.4 times higher than that for whites (Mathews and MacDorman 2007). In the face of the consistent reduction in infant mortality for both racial groups, relative disparities in black and white infant mortality rates continue to exist, and reducing or eliminating these disparities continues to be a major goal of U.S. health policy (U.S. Department of Health and Human Services [U.S. DHHS] 1991 and 2000).

Analyzing infant mortality trends is essential for gaining additional knowledge as to why changes have occurred and of their consequences in order to guide the development of policies aimed at both improving infant survival in general and reducing racial disparities in particular. Moreover, research needs to take into account changes in the risk factor profiles that characterize groups that are being compared. National statistics show that there have been substantial variations in risk factor profiles over time and by racial groups. In fact, an inspection of changes in risk factors for infant mortality shows that the magnitudes of the changes were such that the gaps between blacks and whites actually narrowed over time.

During the 1980s and 1990s, the five leading causes of infant death were: congenital anomalies (CA), Sudden Infant Death Syndrome (SIDS), Respiratory Distress Syndrome (RDS), disorders relating to short gestation and unspecified low birth weight (SG/LBW), and maternal

complications (MC). Over the period of time encompassed by this analysis (1983-2002), innovations in perinatal care and technology aimed specifically at reducing infant death from CA, SIDS, and RDS emerged (or were expanded). Particular attention will be given to contrasting changes in infant mortality from these three causes with changes in the remaining two causes for which no and/or largely ineffective interventions were available. In a recent review, Phelan and Link (2005: 27) argued that "(A)ny explanation that ignores large improvements in population health and fails to account for the emergence of disparities for specific diseases is an inadequate explanation of current disparities."

OBJECTIVES

Identifying changes in racial disparity trajectories across and within causes of infant death allows us to evaluate the equity (or inequity) impact of various types and characteristics of perinatal care interventions. In regard to CA, SIDS, and RDS, innovations in perinatal care and technology introduced over the past two decades have varied substantially depending on which specific cause was the object of the intervention. For instance, the "Back-to-Sleep" initiative instituted to reduce the risk of infant death from SIDS involves no monetary cost and relies heavily on voluntary behavioral change, while perinatal innovations designed to reduce mortality from CA and RDS are more likely to involve high technical demands and to require extensive and costly treatments. In the case of SG/LBW and MC, no effective innovations occurred.

In the broadest terms, then, our objectives are to analyze the five leading causes of infant death, along with a residual category representing all other causes, by examining temporal changes that occurred for the period 1983-2002 and evaluating the impact of distributional changes in known risk factors for infant mortality. Specific aims include: (1) evaluation of whether, and the extent to which, changes in racial disparities in known contributing factors are

related to changes in racial disparities in the risk of overall and cause-specific infant mortality; (2) net of known risk factors, to determine whether, and the extent to which, racial disparity trajectories are distinct across and within causes of infant mortality for which efficacious interventions were available and those causes for which such interventions were absent; and (3) to more precisely estimate models of the relationships of interest by isolating year-to-year variations via multilevel random effects models.

Most research of this sort has examined overall change in infant mortality or change in one specific cause—typically by contrasting cross-sectional snapshots of data at a given period of time with cross-sectional data at later time periods. By contrast, the present study examines year-by-year changes in black and white infant mortality due to several leading causes. In addition, we go beyond the small amount of previous work on cause-specific infant mortality by covering an extensive time period, by giving more attention to risk factor compositional change, and by developing more precise estimates of the effects of interest through use of a multilevel random coefficient model with birth cohorts as a second-level unit.

CONCEPTUAL FRAMEWORK

Fundamental cause theory posits that health and mortality disparities are due to differentials in access to and effective utilization of social resources including money, knowledge, power, prestige, and social connections (Link and Phelan 1995, 2000, 2002, 2005). The utility of having greater social resources, in turn, varies because (1) health technologies continue to evolve over time, (2) they are more or less designed to influence one particular health condition at a time, and (3) they do not automatically benefit one's health because one must have the resources and take the necessary actions to access innovations in health care. The core proposition of Link and Phelan's theory is that the effects of social status in health and mortality

become stronger when health innovations are newly available. The effect of social status becomes weaker, or remains relatively constant, for health conditions where no beneficial interventions are available—because even resource-rich people cannot redirect their resources to gain health advantages. Thus, there is a persistent effect of social status on health and mortality over time, and the nature of the relationship from one particular health condition to another is transformed.

The ways in which resources affect health interventions across social groups are multifaceted and complex, including differentials in knowledge and information about health in general, and innovations in health care in particular, different financial means to afford the costs associated with devices, drugs, and health care facilities, and different levels of motivation and compliance regarding recommended actions or treatments, alone or in combination. No data set approaches comprehensive coverage of all such mechanisms. But our study allows us to infer which health conditions are influenced by emerging health technologies in the context of continued racial inequality net of measurable risk factors.

In the specific case of infant mortality, Gortmaker and Wise (1997) suggest that the development of effective new interventions, while beneficial for the population as a whole, can also lead to increases in rate disparities across social groups. That is, while new technology or newly-developed information may be "group-neutral" in their effects, access to and use of such technology and information continue to be highly stratified in the context of continuing race/ethnic inequality in the United States.

Several hypotheses may be derived from the foregoing discussion:

1. The black-white relative disparity in infant mortality will increase for CA, SIDS, and RDS—the three causes of infant death for which beneficial advances in perinatal care and technology became available (or were expanded).

1a. With respect to infant mortality from CA and RDS, certain efficacious perinatal interventions were in place at the beginning of the time period covered by our analysis, but new, highly effective innovations emerged in the early 1990s. Hence, one might anticipate that black-white relative disparities in infant mortality will show a gradual increase in the earliest data, but that the gap will increase at an accelerated rate subsequent to the early 1990s, a period in time when these new interventions were implemented.

1b. In the case of SIDS, it is expected that relative racial disparities will remain little changed until the "Back-to-Sleep" initiative was launched, following which racial disparities will show a rapid expansion.

2. Black-white relative racial disparities in relative infant mortality due to SG/LBW and MC will evidence little change over time. However, it is plausible that the SG/LBW gap will narrow somewhat, given evidence that preterm and low birth weight rates have increased slightly among whites and decreased slightly among blacks (Demissie et al. 2001).

3. Given the temporal narrowing in risk factor composition between blacks and whites, we expect that adjustment for risk factor covariates in our longitudinal analysis will lead to an increase in black-white infant mortality disparities. That is, controlling for changes in risk factors may statistically negate the gains made by blacks regarding variables that affect changes in the risk of infant mortality.

THE FIVE LEADING CAUSES OF INFANT MORTALITY

The rationale for our focus on the five causes of infant mortality listed above is obvious—they were the leading causes of deaths of infants in the 1980s and 1990s, and with one remarkable exception, into the 21st Century. From 1970 forward, congenital anomalies have been the number one cause of infant death in the U.S. (Lee et al. 2001). SIDS was the second leading cause into the early 1990s, when it was replaced by SG/LBW (U.S. Bureau of the Census 2001). RDS ranked fourth in the early 1980s, but after the approval of surfactant replacement therapy by the Food and Drug Administration (FDA) in August 1990, infant mortality attributable to this condition showed a marked decline and became the sixth leading cause by the end of the 1990s

(Arias et al. 2003).[1] Infant mortality from MC (e.g., incompetent cervix, premature rupture of membranes, breech or other malpresentations, death of mother, etc) was the fifth leading cause of infant mortality from 1980 to the late 1990s, but it stood fourth in the early 2000s (Mathews et al. 2002). Beyond this, the selection of each of the causes (discussed below) support the utility of a comparative, longitudinal analysis that reveals the responsiveness of rates with and without efficacious perinatal care innovations available, as well as in periods prior to and subsequent to specific perinatal advances. Changes in IMRs for the five conditions appear in Table 1.

--Table 1 About Here--

Congenital Anomalies

The category of congenital anomalies includes a wide variety of conditions, but the greatest amount of attention appears to have focused on some of the most life-threatening conditions, especially cardiovascular malformations or neural tube defects (Lee et al. 2001; Texas Department of Health 1995). Consumption of folic acid prior to conception and in the early stages of pregnancy apparently reduces the risk of neural tube defects. Hence, in 1998, the FDA mandated "the fortification of enriched grain products with folic acid" (Williams et al. 2005: 580). In 1994, and again in 1999, Congress provided the Centers for Disease Control and Prevention (CDC) with appropriations "to establish or improve their birth defect surveillance systems" (Erickson 2000: 2) because earlier and more accurate detection of CA allows both antenatal and postnatal interventions to occur in a much more timely manner. Other interventions have been directed at reducing CA. These include enhancement of the ability to detect congenital malformations in the course of prenatal care, educational campaigns warning against use of harmful substances, antenatal surgical procedures to correct malformations detected in the fetus,

[1] It was displaced by cord and placental complications by the year 2000, but the latter was not among the five most lethal conditions for the vast majority of years covered by our analysis (Mathews, Menacker, and MacDorman 2002).

and post-partum interventions designed to preserve the life of infants born with CA (Boneva et al. 2001; Boyle and Cordero 2005; Nsiah-Jefferson 1993). Most of these interventions are extremely costly (Mahoward et al. 2006; Russo and Elixhauser 2007). Barriers limiting access to these interventions are much higher for "low-income and women of color" (Nsiah-Jefferson 1993: 308). One of the exceptional cases which require less financial means is the folic acid supplementation and mandatory fortification and in turn, one would expect that these new innovations may have been a countervailing force diminishing the racial gap in infant mortality attributable to CA. The available evidence, however, shows that black and poor women are much less likely to take folic acid supplements and eat food high in folic acid and natural folate (CDC 2002; Dowd and Aiello 2008).

Sudden Infant Death Syndrome

The etiology of SIDS is not well established. However, research suggests that "airway protection is compromised in the prone sleeping position" (and improved in the supine position) because when infants sleep on their stomachs, "the swallowing rate is reduced significantly" (with) "no compensatory increase in arousal" (Jeffrey, Megevand, and Page 1999: 263). Shortly after early studies demonstrated the relationship between sleeping position and SIDS, the American Academy of Pediatrics (AAP) recommended (in 1992) that infants <u>not</u> be placed in the prone position for sleep, followed by the "Back-to-Sleep" nationwide educational campaign in 1994 (Gibson et al. 2000; Pollack and Frohna 2001, 2002). The "Back to Sleep" program appears to be the single most significant intervention in that it had a rapid and positive impact on survival chances (Adams et al. 1998; Malloy and Freeman 2000; Pollack and Frohna 2001). Willinger et al. (1998) show that, prior to 1992, the SIDS mortality rate changed little, even though infant death rates from many other causes were on the decline. Unfortunately, blacks seem to be less

apt to be advised to put infants to sleep on their backs and to adhere to the "Back-to-Sleep" recommendation (Colson et al. 2006).

Respiratory Distress Syndrome

RDS results from a deficiency of naturally occurring surfactant in the lungs of the fetus such that the functioning of the aveoli may be compromised and gas exchange may fail. It is a largely (but not entirely) a problem of preterm (or low birth weight) infants in that, prior to 26 weeks gestation, there is usually little or no natural secretion of surfactant (British Columbia Reproductive Care Program 1993; Halliday 1997; Malloy and Freeman 2000). There is compelling evidence that surfactant therapy is "the single most important advance in neonatal medicine of the past 20 years" (Cummings 1999). After FDA approval of surfactant therapy for general use in August 1990, clinical studies documented both the efficacy of surfactant replacement as well as a differential effect by race. For example, Hamvas et al. (1996) using clinical data from three St. Louis hospitals demonstrated that, after administration of surfactant, what had been a black survival advantage at low birth weight from RDS changed to a black disadvantage, compared to their white counterparts, in the post-surfactant period. In addition, several population-based studies demonstrated a substantial drop in infant mortality from RDS after the introduction of surfactant replacement therapy, along with a widening of the black RDS survival disadvantage between the early and mid-1990s (Frisbie et al. 2004; Malloy and Freeman 2000; Ranganathan et al. 2000). However, the risk of RDS deaths began to drop for whites in the U.S. before surfactant therapy was introduced in 1990 (Lee et al. 1999; Malloy et al. 1987). The latter finding represents yet another reason for beginning our analysis at the earliest possible date (1983) at which the necessary data are available. Surfactant therapy is complementary to existing interventions that were already in place. Indeed, "adequate management of RDS includes

prenatal referral to a tertiary perinatal unit, prophylactic prenatal corticosteroids, early rescue exogenous surfactant, and ventilatory support" (Moriette et al. 2001). The cost of such a course of treatment in neonatal intensive care units (NICUs) is extremely high and leads to the expectation of a widening of the white RDS survival advantage inasmuch as blacks are, on average, much more economically disadvantaged compared to whites.

Short Gestation/Low Birth Weight

In 1980, the SG/LBW rate ranked fourth in lethality behind CA, SIDS, and RDS rates in descending order (U.S. Census Bureau 2001), but by the mid-1990s, SG/LBW was second only to CA as a leading cause of infant death. More disturbingly, the IMR for SG/LBW has actually risen in recent years, probably due to an increase in rates of adverse birth outcomes and ineffective perinatal care technologies for reducing spontaneous preterm delivery (Blondel et al. 2002; Demissie et al. 2001; Pool 1998). Creasy and Merkatz noted that spontaneous preterm delivery was "the most significant problem facing clinicians as we enter the 1990s" (1990:25).

Spontaneous preterm birth occurs for reasons not completely understood, and interventions designed to prevent preterm labor have, in the past, not been particularly successful (Cockey 2005; Moore and Freda 1998). Pharmacological intervention, e.g., the administration of tocolytic agents to arrest uterine contractions, may delay, but does not prevent, preterm labor (Viamontes 1996). Also, while "intravenous hydration is a commonly used first clinical effort to reduce preterm labor contractions," as late as the mid-1990s, there was "no published evidence that pregnancies have been prolonged through use of hydration" (Freda and DeVore 1996: 385). Results from recent clinical trials allow for optimism in that a form of progesterone (commonly referred to as 17P) may be effective in preventing preterm labor (da Fonseca et al. 2003; Petrini

et al. 2005). However, given the timeframe for which data are available for this study, it is unlikely that results from the latter intervention will be reflected in this analysis.

Maternal Complications

The MC category includes a wide range of conditions such as incompetent cervix, premature rupture of membranes, ectopic pregnancies, breech or other malpresentations, death of mother, etc. Few publications that are comparative by race/ethnicity deal with this heterogeneous category. We do know, however, that maternal complications were the fifth leading cause of death from 1980 to the late 1990s (U.S. Census Bureau 2001: Table 103). We also know that the black infant mortality rate from MC has been about two and one-half times that of whites (Mathews et al. 2002; Muhuri, MacDorman, and Ezzati-Rice 2004).

DATA AND METHODS

Data

The data source is the 1983-1991 and 1995-2002 linked birth/infant death cohort files released by the NCHS. The linked cohort data first became available in 1983, but no linked files were generated by NCHS for the period 1992-1994. The data for 2002 were the most recent available for public use at the time of this research. A data set with a very large number of cases is required to ensure estimates with high precision, especially when conducting detailed analyses by cause-of-death. This means that recourse much be made to vital statistics that contain all recorded births and deaths of infants in the U.S.—approximately 4 million births per year. The match rate of the linked birth/infant death cohort files is exceptionally high. For all years included, over 97% of the infant deaths were successfully matched with live births (U.S. Department of Health and Human Services 1995). Infants whose deaths were not linked to their respective birth certificates are removed from the analyses.

We will focus on infants born to the black and white populations in the 50 states and the District of Columbia. It might be preferable to distinguish whites and blacks on the basis of Hispanic origin – given that the mortality experience of Hispanic infants is similar to that of non-Hispanic white infants. However, most states prior to 1989 did not include information on Hispanic identity in their vital records. The failure to separate race from Hispanic ethnicity should result in little or no distortion of our results since 94 % of Hispanics identify their race as white (Albrecht et al. 1996).

Many studies of infant mortality are limited to births weighing 500 grams or more because of concerns about misclassification of stillbirths as live births and misreporting of birth weight. Consistent with NCHS reports, however, this study will include these extremely low weight infants because, although the proportion of these compromised births is small, this strategy leaves out a large number of infant deaths. Preliminary analysis shows that infants born weighing less than 500 grams account for only 0.15 % of all live births, but the exclusion of these births from the analysis lowers the infant mortality rate by about 15 % compared to that provided by NCHS, which generally include births weighing less than 500 grams in its official tabulations.

Method

The amount of missing data is generally minimal, except in the case of education. Information on education was not routinely compiled until the early 1990s for three states – California, Texas and Washington. Rather than omit these states from the analysis, which would have required deletion of more than 10 % of all births, we will adopt the conventional strategy of assigning a dummy category for cases where information is missing for education. This procedure has been widely utilized and proven successful in previous research (Frisbie et al.

1998; Singh and Yu 196). Following deletion of missing cases (except that for the education variable) from the data set, the percent missing on covariates ranges from 0.00 % to 2.29 %. The cumulative percent missing for all covariates combined is 4.39 %. Excluding records with missing data does not lead to any serious distortion of results. It is logical to assume that whatever bias exists would result in a conservative estimation of true differentials because information is more apt to be missing for women with a high risk of infant death who are more likely to be black than white. The resulting data set contains more than 60 million births.

Causes of death are classified according to the International Classification of Diseases (ICD-9) for the years1983-1991 and 1995-1998. As of January 1999, the Ninth Revision was replaced by the ICD-10 for coding of causes of death. A special study (Anderson et al. 2001) based on 1996 cause of death data coded according to both the ICD-9 and ICD-10 produces the comparability ratios shown in Table 2.

--Table 2 About Here--

As can be seen in Table 2, the codes for SIDS, RDS, and MC translate on close to a one-to-one basis. A lesser, but still fairly high, degree of comparability has been achieved for CA and SG/LBW. Although concerns have been raised about comparability between the two revisions, the results should be minimally affected by the revision of ICD codes because there is no reason to believe any distortion has occurred differently across sub-populations. Great caution, however, will be used in identifying any unexpected disturbances which may result from the code shift with regard to disease classification.

The outcome variable consists of seven categories including: (1) CA, (2) SIDS, (3) RDS, (4) SG/LBW, (5) MC, (6) a residual category representing all other causes, with (7) survival as the referent. Race is comprised of the black and white populations. Whites serve as the reference

category. Cases are defined by the mother's race as recommended in the past by NCHS. This operationalization renders our study comparable with previous research as the vast majority of earlier studies adopted the NCHS recommendation. Intervening factors are those for which data were available throughout 1983-2002 and are measured in conventional fashion [2] as shown in Table 3, which displays the risk factor distributions in the Result Section.

Following the descriptive analyses, which present annual rates of overall and cause-specific infant mortality for blacks and whites and annual black/white rate ratios, we estimate year-to-year changes (contrasting blacks with whites) using a multilevel random coefficient model that treats birth cohorts as the second-level unit. Under this specification, each birth cohort has its unique intercept and coefficient for race that allows simultaneous exploration of cross-sectional and temporal variations in the risk of infant death for whites and for blacks relative to whites.[3]

The baseline model includes only the race variable and the full model adds the complete set of risk factors listed in Table 3. For all covariates, the reference category is the one associated with the least risk of infant mortality. All covariates except the race variable are grand-mean-centered to facilitate the interpretation of the intercept. The intercept represents the average log-odds of the risk of infant death for whites that is at the mean of all the predictors included in the model. It is important to note that grand-mean centering does not affect the estimation of the log-

[2] However, the parity variable is operationalized via the Kleinman-Kessel (1987) index that takes into account the well-known curvilinear relationship between maternal age and birth order. It has been shown that there is no collinearity problem between parity and maternal age (Frisbie et al. 1998).

[3] The conventional model employs estimates for race, dummies for years, and the interactions between race and year dummies as covariates. According to Yang and Land (2006), there is little difference in the estimates between these two strategies, but the conventional model tends to underestimate standard errors of estimation because it ignores the potential dependence of individual outcomes within a contextual unit such as birth cohorts. Multilevel modeling is specifically designed to deal with such a complex error structure, thereby generating more efficient estimates than conventional modeling (see Agresti et al. 2000; DiPrete and Forristal 1994).

odds of race. The baseline and full models estimate the risk of overall infant death, followed by each cause, as compared to survival as the reference category.

The baseline model specification is as follows:

The level-1 model

$$\text{Log}\,(P_{ij}/(1-P_{ij}) = \beta_{0j} + \beta_{1j}\text{RACE}_{ij}$$

The level-2 model

$$\beta_{0j} = \gamma_{00} + U_{0j}\,, \qquad U_{0j} \sim \text{N}\,(0, \tau_{00}).$$

$$\beta_{1j} = \gamma_{10} + U_{1j}\,, \qquad U_{0j} \sim \text{N}\,(0, \tau_{11}).$$

For i= 1, 2, …, nj infants within a birth cohort j,

j=1983, 1984,…. 1991, 1995,… 2002 (for 17 birth cohorts)

The Level 1 model estimates, within each birth cohort j, infant i's mortality risk, as a function of mother's race. Notice that there is a subscript j for the intercept and slope coefficient for race that allow each birth cohort to have a unique intercept and coefficient for race.

The Level 2 equation specifies the inter-birth cohort differences in the intercept and slope coefficient of race. β_{0j} and β_{1j} have means of γ_{00} and γ_{10}, representing the values with respect to the average intercept and the average slope coefficients of race for all birth cohorts. It follows that the variance of β_{0j} and β_{1j}, τ_{00} and τ_{11}, are measures of the variability of the intercept and slope coefficient of race between birth cohorts, respectively. U_{0j} and U_{1j}, in turn, represent the residual random effect, indicative of deviations in the intercept and the slope coefficient of race for each birth cohort j from the average intercept (γ_{00}) and average slope coefficient of race (γ_{10}) for all birth cohorts. These values are obtained from the residual files.

The final baseline model is as follows:

The combined model of equations 1 and 2

$$\text{Log}\,(P_{ij}/(1-P_{ij}) = \gamma_{00} + \gamma_{10}\text{RACE}_{ij} + U_{0j} + U_{1j}\,\text{RACE}_{ij}$$

When the Level 2 equation is combined with the Level 1 equation, the final baseline model includes individual fixed effects (γ_{00} for the average log-odds of infant death for whites and γ_{10} for the average log-odds for blacks relative to whites) and the variance components that can be decomposed to birth-cohort level U_{0j} and U_{1j} for the random intercept and for the random slope of race, respectively. To this baseline model, we add the intervening factors.

The full model is as follows:

$$\text{Log}\,(P_{ij}/(1-P_{ij}) = \gamma_{00} + \gamma_{10}\text{RACE}_{ij} + \gamma_{20}X_{1ij} + \gamma_{30}X_{2ij} + \gamma_{40}X_{3ij} \ldots + U_{0j} + U_{1j}\,\text{RACE}_{ij}$$

$X_{1ij,}\ X_{2ij},\ X_{3ij,\ldots\ldots}$ = Intervening factors.

In the full model, the intercept (γ_{00}) and slope coefficient of race (γ_{10}) reflect the average log-odds of the risk of infant death for whites and for blacks relative to whites, net of the distribution of intervening factors. In terms of the variance components, U_{0j} and U_{1j} are the remaining variations in the intercept and race coefficient for each birth cohort j from the average intercept and average coefficient of race over all birth cohorts after taking into account a set of intervening factors.

Of analytic interest will be the residual random effect of birth cohort j with respect to the intercept and the race coefficient (U_{0j} and U_{1j}), their changes from the baseline model to the full model, and their residuals in the full model. As clarified above, U_{0j} ($=\beta_{0j} - \gamma_{00}$) and U_{1j} ($=\beta_{1j} - \gamma_{10}$) are equivalent to the amount of unique increment in the intercept for whites and the slope of blacks relative to whites associated with each birth cohort j (β_{0j} and β_{1j}) deviated from the average intercept and the average coefficient of race for all birth cohorts (γ_{00} and γ_{10}). In other words, U_{0j} and U_{1j} quantify the year-to-year change in the risk of infant death for whites and the year-to-year change in the risk of infant death for blacks relative to whites on a log-odds scale.

Because U_{0j} and U_{1j} are scaled to have means of 0, they facilitate comparisons of the direction and magnitude of changes across causes of infant death.

As noted earlier, all intervening variables included in the full model are grand-mean-centered so that the magnitude of changes in U_{0j} and U_{1j} from the baseline model to the full model captures the contribution of the compositional changes in the intervening factors to the temporal changes in the log-odds of the risk of infant death for whites and for blacks relative to whites.

In the full model, the values of U_{0j} and U_{1j} represent the temporal differences that remain when compositional differences in the known intervening factors are equalized across birth cohorts. The equity (or inequity) impact of perinatal care interventions on racial disparities, in turn, is gauged by comparing and contrasting the direction and magnitude of changes in U_{0j} and U_{1j} between and within cause of infant death. As a simple illustration, for the three causes of infant death for which effective perinatal care technologies were available, change in U_{0j} has a negatively sloping curve over time, while change in U_{1j} has a positively sloping curve over time. For the other remaining causes with no and/or largely ineffective innovations, there are no substantial variations in U_{0j} and U_{1j} over the years.

DESCRIPTIVE RESULTS

Changes in Risk Profiles

Table 3 shows the distribution of risk factors separately for the white (Table 3a) and black (Table 3b) populations, for every cohort. Consonant with all prior studies, blacks are more disadvantaged than whites in the risk profiles across all birth cohorts. For example, black mothers are more likely than their white counterparts to be in the poorly educated, teenage, and unmarried categories. They are slightly more apt than whites to have a history of previous

pregnancy loss and much less likely to initiate prenatal care in the first trimester. And of course, the incidence of preterm and low weight births is considerably greater among blacks.

--Table 3 about here—

However, several notable changes in risk factor distributions occurred over the years. For instance, black women continued to lag behind white women in the level of education, and the amount of missing data throughout the 1980s makes comparisons difficult. But if we draw the comparison beginning in 1990, we see that the percentage of white mothers with less than a high school education remained virtually constant at about 21%, while the percentage for low educated black mothers dropped from 28.7% in 1983 to about 24.0 % in the year 2000. The growth in the proportion that went on to college is close to equal for blacks and whites over the same time period, and if we compare recent educational changes (say, 1995 to 2002), the improvement for blacks entering college slightly surpassed the white percentage. It is also true that timely prenatal care increased for both racial groups, but with a more substantial improvement for blacks as compared to whites.

The other risk factors changed in a direction that was less favorable for infant survival. But again the black-white gap narrowed because the unfavorable changes were greater in magnitude among whites. To illustrate, during the past two decades, the rates of births born to unmarried have increased by more than 50 % among white women, as compared to only a 15 % increase for their black counterparts during the past two decades so that the racial gap in marital status actually narrowed. The same is true for the infant morbid conditions in that there were considerable increases in the rates of preterm and low weight births as well as multiple births. Such a worrisome trend was observed for both racial groups, but it was much more pronounced for their white populations than for black counterparts. In summary, whites fare best in regard to

their risk profiles across all years. However, over-time changes in the risk profiles were, in every case, more favorable for black mothers than white mothers.

Infant Mortality Rates and Rate Ratios

Table 4 is divided into two sections. The first (Table 4a) presents the empirical IMRs (i.e., those observed in the raw data) for each cohort and every specific cause of infant death, and the second (Table 4b) displays rate ratios obtained by dividing the black IMRs by the white IMRs. As was known before entering into this analysis, with the exception of CA, black infant mortality rates are much higher than the corresponding white rates (Table 4a).

More interesting for present purposes are the rate ratios. The overall IMR (and some of the cause-specific IMRs) among blacks are two to four times higher than the white IMRs (Table 4b). Importantly, and as expected, the rate ratios for the three causes of infant death for which advances in perinatal care and technology occurred show notable increases in black-white disparities following the introduction of cause-specific beneficial interventions. For example, the ratio for SIDS jumped from 1.98 in 1991 (before the "Back-to-Sleep" intervention) to 2.35 in 1995 (after the sleeping position recommendations of 1992 and 1994). Considering infant mortality from CA, the ratios began at a value of 1.10 in 1983 and then followed an increasing trend—hardly surprising since effective interventions emerged and were expanded to reduce infant death from that cause. The same is true for the case of RDS in that the black and white disparities showed increases over the years, with evidence of a somewhat steeper increase after surfactant therapy received FDA approval in 1990. Table 4b shows that the RDS rate ratios increased from 2.28 in 1989 to 2.59 in 1990 and to 2.65 in 1991, following which the ratio was never lower than 2.65 and stood at 2.96 in 2002. Different from these three leading causes of infant death for which effective innovations were available, the rate ratios for SG/LBW and MC,

along with other causes combined, were reduced for the latter time period, despite an increasing trend up to the early 1990s. Note that infant mortality from SG/LBW and MC for both racial groups remained fairly stable from 1983 to 1988 and then began to gradually increase over the following years.

--Table 4 About Here--

MULTIVARIATE RESULTS

Figure 1 includes six separate graphs, one for each of the five leading causes of infant death and one for the other causes residual category. Figures 1a -1f delineate the inter-cohort variation in the risk of infant death for whites (U_{0j}) and for blacks relative to whites (U_{1j}) on a log-odds scale, indicating deviations from the average log-odds of the risk of infant death for whites (γ_{00}) and for blacks relative to whites (γ_{10}) for all birth cohorts. The Figures contain results from the bivariate and full models. In order to improve readability, we do not include the inter-cohort variation in the risk of infant death for blacks in the Figures. However, these values can be obtained simply by adding two random effects, $U_{0j} + U_{1j}$. Appendix provides full information regarding parameter estimates and cohort-variance components, along with random cohort effects on which the Figures are based.

There was a gradual decline in the CA log-odds for white populations across all years, but the rate of improvement appears to be larger for years between 1998 and 1999, i.e., immediately after mandatory fortification of grain products took place. Conditional on risk factors, the negatively sloping curve observed in the bivariate model becomes steeper. This is consonant with distributional changes in the risk profile for whites that have worsened over time. In particular, rates of preterm and low weight births to white women rose from 1983 to 2002. Figure 1a shows a positively sloping curve for the black-white bivariate comparison, which

reflects a modest increase in black-white disparities in infant mortality due to CA. It is important to note that this does not imply that there was no reduction in the risk of CA death for black infants. Rather, it indicates that the rate of improvement was smaller for blacks as compared to whites. Adjustment for risk factors leads to an even wider black-white differential as evidenced by the steeper upward slope in the full model. This was anticipated in that changes in the risk profiles were generally less favorable for white mothers than for black mothers. A somewhat steeper, gradual increase in the disparity from the mid 1990s onward - following the introduction of folic acid interventions - was seen in the full model.

Figure 1b confirms that the "Back-to-Sleep" intervention was associated with a large reduction in infant mortality caused by SIDS. Prior to the early 1990s, there was little change in the SIDS risk in the U.S. As expected, there was a sharp downward inflection in the white log-odds immediately following the "Back-to-Sleep" initiative in the mid-1990s. Interestingly, adjusting for risk factors had virtually no effect on changes in the inter-cohort variation in the SIDS rate for the white population. There was also an upward inflection in the black-white SIDS disparity following the educational campaign recommending that infants be put to sleep in the supine position. This upturn in the differential was greater in the full model than in the bivariate model. The steepness of the slopes depicting racial disparity in regard to SIDS deaths increased in the mid-1990s. Two explanations might be offered. It is certainly plausible that the information disseminated during the "Back-to Sleep" campaign failed to reach many blacks. Another possible interpretation is that blacks were simply less compliant.

The risk of RDS death among white infants showed a marked decline throughout the 1983-2002 period (Figure 1c). Controls for risk factors steepened the negatively sloping curve over time and especially after surfactant therapy came to be widely used. This is not surprising

because of unfavorable distributional changes in risk factors, especially associated with low weight and preterm births, for the white populations. An observed reinforcement for the downturn in the RDS rate from 1990 onward seems to evidence the effectiveness of surfactant therapy. Figure 1c also shows that there was a marked increase in RDS survival advantage for whites than for blacks over the years. As was true for CA and SIDS causes of infant death, adjusting for risk factors results in a steeper log-odds curve for the disparity, especially for the period after surfactant therapy was introduced in 1990. This implies that white infants benefited from surfactant therapy to a greater extent than black infants.

--Figure 1 About Here--

Consistent with Hypothesis 2, the nearly flat log-odds slopes for SG/LBW (Figure 1d) and MC (Figure 1e) indicate little change in the risk of infant death from these conditions among white infants. That is, in contrast to the three leading causes (CA, SIDS, and RDS) for which effective perinatal interventions were available, the inter-cohort variations for both the trend for whites and the trend for black-white disparities were quite small, and this pattern holds for both the baseline and full models.

Note that infant mortality from SG/LBW showed an increase for whites from the late 1980s forward (Figure 1e). Different from the three leading causes of infant deaths for which effective perinatal interventions were available, the inter-cohort variation was smaller in the full model than in the bivariate model.

The log-odds for the Other Causes category showed a rather remarkable decline in infant mortality for whites in both the baseline and full models during the 1980s into the early 1990s, with little improvement since the mid-1990s (Figure 1f). In terms of the temporal pattern of black and white disparities, the baseline (bivariate) and full models show small increases up to

about 1991, at which point there was a plateau in the trend. The trend lines for the two disparity models are very nearly identical.

CONCLUSIONS

All hypotheses receive fairly strong support in our analyses. The disparities in the likelihood of infant death between blacks and whites did increase for the three causes of death (CA, SIDS, and RDS) for which advances in perinatal care and technology occurred during the time period encompassed by our data (Hypothesis 1). Further, as predicted the black-white gap (as seen in the log-odds trends in Figure 1) showed at least a moderate upward inflection for RDS in the first half of the1990s—following the introduction of pulmonary surfactant therapy and for CA in the late 1990s—following the fortification of grain products with folic acid (Hypothesis 1a). Further, as predicted by Hypothesis 1b, what had been a gradual downward trend in SIDS deaths took a sharp downward inflection after the "Back-to-Sleep" initiative, and the racial disparity curve showed a notable upturn. Hypothesis 2, which anticipated little or no change in either the risk of death [4] or in racial disparities from the two specific causes (SG/LBW and MC) for which no efficacious interventions emerged, was fully supported. Finally, given the fact that changes in risk factors typically favored blacks, Hypothesis 3 predicted that adjustment for risk factors make over-time increases in black and white infant mortality disparities more pronounced. In every case, this expectation was borne out.

A limitation of our study is that the number of control variables available to us was far from optimal because several potentially influential risk factors were not available in our data set before 1989. For example, the only direct indicator of socioeconomic status (SES) in the NCHS linked cohort files is maternal education. It would clearly have been preferable to be able to

[4] In fact, the risk of death from SG/LBW increased a bit over time.

include measures of income, wealth, and health insurance status. However, this limitation is offset by the fact that, to our knowledge, we were able to construct complex models of racial disparities longitudinally over a longer period of time than any previous research.

In evaluating these results in the context of the conceptual framework guiding our analysis, it must be acknowledged that our data set contains no information on which infants received beneficial interventions and which did not. This, in turn, means that we have conducted only an indirect test of the fundamental cause theory through use of a "before and after natural experiment." Nevertheless, we believe that our findings allow drawing strong inferences that support the validity of that theory.

Table 1. Selected Causes of Infant Mortality Rates*: United States, 1980, 1990, and 2000

Cause of Death	1980	1990	2000
Congenital Anomalies	2.6	2.0	1.42
Sudden infant death syndrome	1.5	1.3	0.62
Respiratory distress syndrome	1.4	0.7	0.25
Short Gestational/Low Birth Weight	1.0	1.0	1.08
Maternal Complication	0.4	0.4	0.35
All-Cause	12.6	9.2	6.91

Sources: Statistical Abstract of the U.S. 2001, Table 103; Arias et al. 2003.

* Infant Mortality Rates per 1,000 Live Births.

Table 2. ICD - 9 and ICD - 10 Codes for the Selected Causes of Death and Comparability Ratios

Cause of Death	ICD-9 Codes	ICD-10 Codes	Estimated Comparability Ratios
Congenital Anomalies	Q00-Q99	740-759	0.91
Sudden infant death syndrome	R95	798	1.04
Respiratory distress syndrome	P22	769	1.03
SG/LBW	P07	765	1.11
Maternal Complication	P01	761	1.04

Table 3-a. Percentage Distributions of Risk Factors for Whites by Birth Cohorts: United States, 1983-2002

	WHITE																
	1983	**1984**	**1985**	**1986**	**1987**	**1988**	**1989**	**1990**	**1991**	**1995**	**1996**	**1997**	**1998**	**1999**	**2000**	**2001**	**2002**
Education																	
< 12 years	14.15	13.63	13.37	13.20	12.93	12.18	19.35	20.56	21.61	21.12	21.12	20.89	20.75	20.78	20.89	21.12	21.16
12 years	33.53	33.18	32.68	31.97	31.35	29.05	35.11	34.90	35.60	32.82	32.19	31.63	31.24	30.67	30.25	29.98	29.56
13+ years	29.28	30.08	30.34	30.81	31.52	29.92	36.51	37.36	39.84	45.01	45.73	46.52	46.93	47.41	47.75	47.84	48.27
Missing	23.03	23.11	23.62	24.01	24.19	28.85	9.03	7.17	2.95	1.05	0.95	0.96	1.08	1.14	1.11	1.06	1.00
Maternal Age																	
10-17 Yrs.	3.80	3.64	3.60	3.59	3.61	3.57	3.60	3.60	3.79	4.25	4.13	4.03	3.84	3.67	3.43	3.20	3.11
18-34 Yrs.	90.42	90.17	89.82	89.30	88.72	88.21	87.64	87.22	86.49	83.76	83.28	82.92	82.77	82.68	82.63	82.68	82.69
35+ Yrs.	5.77	6.18	6.58	7.11	7.67	8.22	8.75	9.18	9.72	12.00	12.59	13.04	13.39	13.65	13.94	14.12	14.21
Marital Status																	
Married	87.45	86.82	85.76	84.55	83.60	82.59	81.12	79.97	78.45	74.98	74.57	74.51	73.99	73.55	73.19	72.60	71.80
Not Married	12.55	13.18	14.24	15.45	16.40	17.41	18.88	20.03	21.55	25.02	25.43	25.49	26.01	26.45	26.81	27.40	28.20
Parity																	
First	43.15	42.66	42.33	42.28	42.01	41.76	41.71	41.65	41.55	41.93	41.40	41.07	40.52	40.56	40.39	39.85	39.82
Low	44.45	45.09	45.48	45.48	45.71	45.81	45.51	45.35	45.20	45.31	45.68	45.88	46.28	46.17	46.13	46.45	46.41
High	12.41	12.25	12.20	12.24	12.28	12.43	12.78	13.00	13.26	12.76	12.92	13.05	13.20	13.27	13.48	13.70	13.77
Previous Loss																	
No	80.69	80.05	79.69	79.24	78.79	77.99	76.75	75.93	75.58	75.33	75.30	75.35	75.62	76.07	76.28	76.31	76.39
Yes	19.31	19.95	20.31	20.76	21.21	22.01	23.25	24.07	24.42	24.67	24.70	24.65	24.38	23.93	23.72	23.69	23.61
Prenatal Care Initiation																	
First Trimester	79.82	80.09	79.75	79.49	79.72	79.81	79.14	79.33	79.60	83.77	84.14	84.79	84.94	85.17	85.10	85.29	85.53
Second Trimester	15.88	15.58	15.79	15.84	15.59	15.56	15.78	15.83	15.82	12.82	12.62	12.05	11.87	11.67	11.68	11.59	11.44
Third Trimester and None	4.31	4.34	4.46	4.67	4.70	4.64	5.07	4.84	4.58	3.41	3.25	3.16	3.19	3.16	3.23	3.12	3.03
Plural Birth																	
No	98.05	98.04	97.98	97.91	97.86	97.81	97.76	97.73	97.67	97.41	97.27	97.16	97.02	96.94	96.90	96.81	96.71
Yes	1.95	1.96	2.02	2.09	2.14	2.19	2.24	2.27	2.33	2.59	2.73	2.84	2.98	3.06	3.10	3.19	3.29
Gestational Age																	
< 37 Weeks	7.95	7.81	8.12	8.28	8.46	8.41	8.71	8.81	9.01	9.61	9.68	10.10	10.39	10.59	10.51	10.88	11.06
≥ 37 Weeks	92.05	92.19	91.88	91.72	91.54	91.59	91.29	91.19	90.99	90.39	90.32	89.90	89.61	89.41	89.49	89.12	88.94
Birth Weight																	
< 2500 Grams	5.47	5.38	5.42	5.42	5.47	5.42	5.58	5.58	5.67	6.10	6.23	6.35	6.40	6.44	6.44	6.59	6.71
≥ 2500 Grams	94.53	94.62	94.58	94.58	94.53	94.58	94.42	94.42	94.33	93.90	93.77	93.65	93.60	93.56	93.56	93.41	93.29

Source: NCHS Linked Birth/Infant Death Cohort Files, 1983-1991 and 1995-2002.

Table 3-b Percentage Distributions of Risk Factors for Blacks by Birth Cohorts: United States, 1983-2002

	BLACK																
	1983	**1984**	**1985**	**1986**	**1987**	**1988**	**1989**	**1990**	**1991**	**1995**	**1996**	**1997**	**1998**	**1999**	**2000**	**2001**	**2002**
Education																	
< 12 years	28.96	27.98	27.32	26.74	26.41	25.53	28.44	28.68	29.72	28.05	27.63	27.11	26.39	25.45	25.00	24.46	23.99
12 years	35.98	36.35	36.63	36.78	36.71	35.65	40.83	41.59	42.10	39.01	38.81	38.39	38.50	38.80	39.18	39.18	39.03
13+ years	19.96	20.44	20.98	21.32	21.63	21.25	25.01	25.63	26.44	31.44	32.21	33.16	33.66	34.17	34.43	34.96	35.65
Missing	15.10	15.24	15.07	15.15	15.25	17.56	5.73	4.10	1.74	1.50	1.35	1.34	1.45	1.58	1.39	1.41	1.33
Maternal Age																	
10-17 Yrs.	10.91	10.67	10.33	10.40	10.48	10.53	10.46	10.13	10.25	10.80	10.32	9.72	8.92	8.22	7.75	7.27	6.90
18-34 Yrs.	84.59	84.68	84.79	84.46	84.18	83.95	83.88	83.86	83.41	80.93	81.04	81.31	81.96	82.45	82.61	82.80	83.00
35+ Yrs.	4.49	4.65	4.89	5.15	5.34	5.52	5.65	6.02	6.34	8.27	8.64	8.97	9.12	9.33	9.64	9.93	10.10
Marital Status																	
Married	41.28	40.14	39.39	38.19	37.18	35.94	34.55	33.75	32.24	30.39	30.42	31.07	31.10	31.32	31.65	31.74	31.96
Not Married	58.72	59.86	60.61	61.81	62.82	64.06	65.45	66.25	67.76	69.61	69.58	68.93	68.90	68.68	68.35	68.26	68.04
Parity																	
First	39.53	39.59	39.41	39.37	39.07	38.73	38.29	37.43	37.08	39.91	39.21	38.85	38.25	37.89	37.52	37.57	37.69
Low	37.48	37.88	38.22	38.41	38.54	38.51	38.31	38.32	37.97	37.51	38.09	38.57	39.27	39.66	39.95	39.95	39.92
High	22.99	22.53	22.37	22.23	22.39	22.76	23.40	24.25	24.95	22.58	22.70	22.58	22.48	22.45	22.52	22.49	22.39
Previous Loss																	
No	78.07	77.70	77.59	77.10	76.64	75.47	73.88	72.97	72.57	70.86	70.74	70.90	71.23	71.19	71.27	71.22	71.09
Yes	21.93	22.30	22.41	22.90	23.36	24.53	26.12	27.03	27.43	29.14	29.26	29.10	28.77	28.81	28.73	28.78	28.91
Prenatal Care Initiation																	
First Trimester	61.68	62.34	62.02	61.78	61.42	61.42	60.24	60.81	62.00	70.52	71.52	72.36	73.39	74.19	74.34	74.53	75.28
Second Trimester	29.01	28.43	28.42	28.17	28.07	28.36	28.14	28.10	27.50	22.02	21.26	20.50	19.75	19.29	19.10	19.03	18.62
Third Trimester and None	9.31	9.22	9.56	10.05	10.51	10.21	11.62	11.08	10.49	7.46	7.22	7.14	6.87	6.51	6.56	6.43	6.10
Plural Birth																	
No	97.55	97.60	97.47	97.53	97.47	97.43	97.33	97.32	97.25	97.14	97.05	96.95	96.83	96.74	96.63	96.56	96.48
Yes	2.45	2.40	2.53	2.47	2.53	2.57	2.67	2.68	2.75	2.86	2.95	3.05	3.17	3.26	3.37	3.44	3.52
Gestational Age																	
< 37 Weeks	17.51	16.93	17.62	17.87	18.21	18.46	18.76	18.61	18.70	17.41	17.18	17.32	17.30	17.31	17.10	17.36	17.42
≥ 37 Weeks	82.49	83.07	82.38	82.13	81.79	81.54	81.24	81.39	81.30	82.59	82.82	82.68	82.70	82.69	82.90	82.64	82.58
Birth Weight																	
< 2500 Grams	12.38	12.12	12.16	12.29	12.41	12.62	13.22	12.98	13.26	12.84	12.76	12.74	12.82	12.86	12.79	12.77	13.13
≥ 2500 Grams	87.62	87.88	87.84	87.71	87.59	87.38	86.78	87.02	86.74	87.16	87.24	87.26	87.18	87.14	87.21	87.23	86.87

Source: NCHS Linked Birth/Infant Death Cohort Files, 1983-1991 and 1995-2002.

Table 4-a. Overall and Cause-Specific Infant Mortality Rates* for White and Black Populations: United States, 1983-1991 and 1995-2002

	1983	1984	1985	1986	1987	1988	1989	1990	1991	1995	1996	1997	1998	1999	2000	2001	2002
									WHITE								
IMR	8.57	8.19	8.02	7.68	7.41	7.20	7.29	6.76	6.53	5.75	5.59	5.57	5.44	5.30	5.29	5.34	5.39
CA	2.17	2.11	2.05	1.95	1.86	1.85	1.85	1.78	1.66	1.55	1.48	1.48	1.41	1.25	1.31	1.27	1.29
SIDS	1.20	1.15	1.19	1.18	1.13	1.15	1.17	1.07	1.07	0.67	0.63	0.60	0.57	0.53	0.50	0.46	0.46
RDS	0.80	0.76	0.75	0.68	0.63	0.59	0.67	0.49	0.45	0.27	0.25	0.23	0.24	0.19	0.16	0.17	0.16
SG/LBW	0.51	0.49	0.46	0.44	0.44	0.42	0.48	0.48	0.51	0.55	0.60	0.58	0.60	0.64	0.64	0.69	0.71
MC	0.27	0.27	0.24	0.25	0.24	0.22	0.25	0.26	0.24	0.23	0.22	0.22	0.23	0.23	0.22	0.26	0.30
OTHER	3.62	3.40	3.32	3.18	3.10	2.97	2.86	2.67	2.60	2.48	2.40	2.46	2.39	2.46	2.45	2.50	2.48
									BLACK								
IMR	17.28	16.25	16.43	15.97	15.57	15.43	16.37	15.47	15.24	13.02	12.94	12.42	12.59	12.65	12.46	12.28	12.66
CA	2.39	2.18	2.32	2.16	2.10	2.06	2.00	2.08	1.98	1.78	1.82	1.71	1.67	1.60	1.55	1.53	1.65
SIDS	2.46	2.24	2.17	2.27	2.15	2.14	2.26	2.17	2.13	1.57	1.48	1.38	1.28	1.24	1.21	1.07	1.08
RDS	1.43	1.35	1.35	1.25	1.29	1.28	1.52	1.27	1.19	0.71	0.72	0.67	0.65	0.53	0.48	0.48	0.46
SG/LBW	1.84	1.74	1.71	1.75	1.78	1.65	2.27	2.19	2.24	2.49	2.36	2.44	2.44	2.60	2.57	2.60	2.74
MC	0.59	0.53	0.54	0.49	0.53	0.56	0.60	0.66	0.62	0.66	0.62	0.56	0.60	0.65	0.70	0.73	0.81
OTHER	8.57	8.22	8.35	8.05	7.71	7.73	7.72	7.10	7.08	5.81	5.94	5.65	5.94	6.03	5.95	5.88	5.92

Source: NCHS Linked Birth/Infant Death Cohort Files, 1983-1995 and 1995 and 2002.
* Infant Mortality Rates per 1,000 Live Births

Table 4-b. Rate Ratios of Overall and Cause-Specific Infant Mortality Rates for Blacks in comparsion to Whites: United States, 1983-1991 and 1995-2002

	1983	1984	1985	1986	1987	1988	1989	1990	1991	1995	1996	1997	1998	1999	2000	2001	2002
IMR	2.02	1.98	2.05	2.08	2.10	2.14	2.25	2.29	2.33	2.26	2.32	2.23	2.31	2.39	2.36	2.30	2.35
CA	1.10	1.03	1.13	1.11	1.13	1.12	1.08	1.17	1.19	1.15	1.23	1.16	1.18	1.28	1.18	1.21	1.28
SIDS	2.06	1.94	1.81	1.93	1.90	1.85	1.93	2.02	1.98	2.35	2.34	2.29	2.23	2.36	2.44	2.30	2.37
RDS	1.79	1.78	1.79	1.84	2.04	2.16	2.28	2.59	2.65	2.65	2.87	2.97	2.72	2.81	2.98	2.88	2.96
SG/LBW	3.57	3.52	3.69	3.96	4.04	3.96	4.71	4.54	4.40	4.50	3.93	4.21	4.09	4.06	4.00	3.79	3.86
MC	2.24	1.98	2.21	1.99	2.22	2.52	2.37	2.52	2.57	2.91	2.82	2.52	2.60	2.78	3.12	2.80	2.70
OTHER	2.36	2.41	2.52	2.53	2.49	2.61	2.69	2.66	2.73	2.34	2.47	2.30	2.49	2.45	2.43	2.36	2.39

Source: NCHS Linked Birth/Infant Death Cohort Files, 1983-1995 and 1995 and 2002.

Figure 1. Log-Odds Showing Changes in the Risk of Cause-Specific Infant Mortality of Whites and of Black-White Log-Odds Differences, 1983-2002

Figure 1a. Log-Odds of Whites and of Blacks Relative to Whites - CA

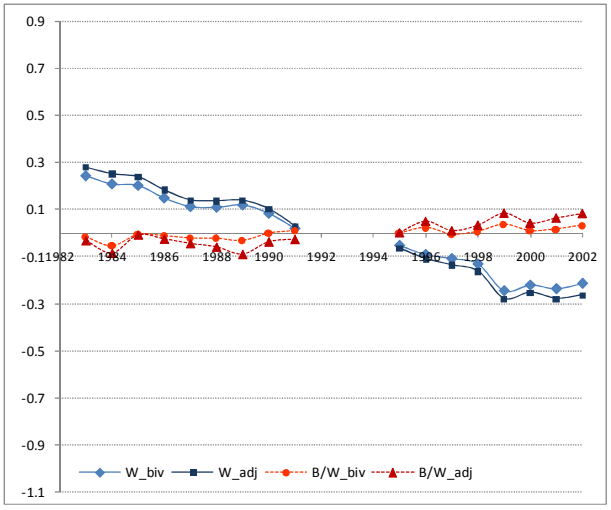


Figure 1b. Log-Odds of Whites and of Blacks Relative to Whites - SIDS

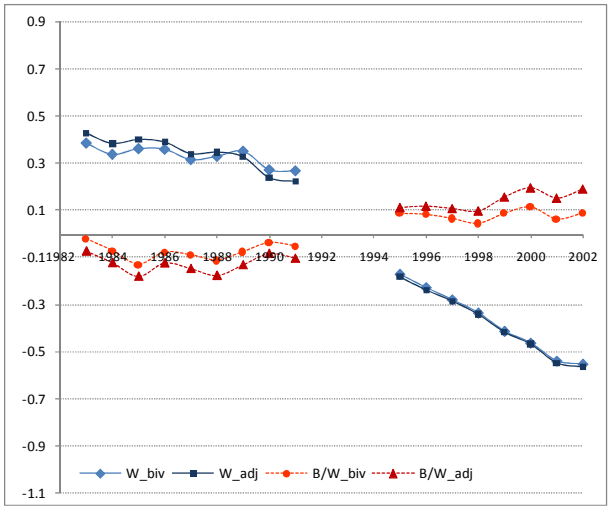


Figure 1c. Log-Odds of Whites and of Blacks Relative to Whites - RDS

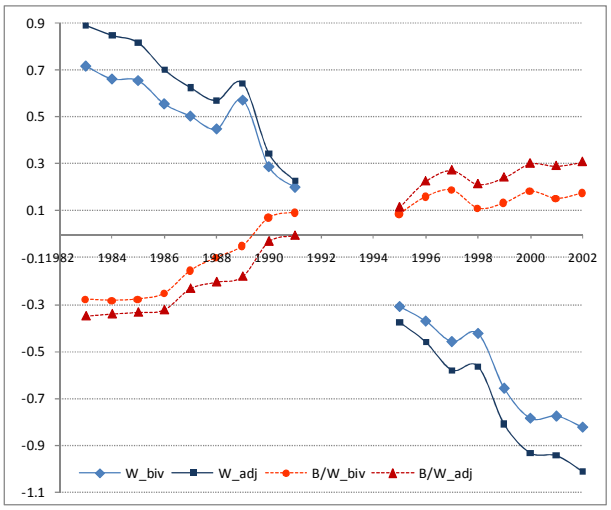


Figure 1d. Log-Odds of Whites and of Blacks Relative to Whites – SGLBW

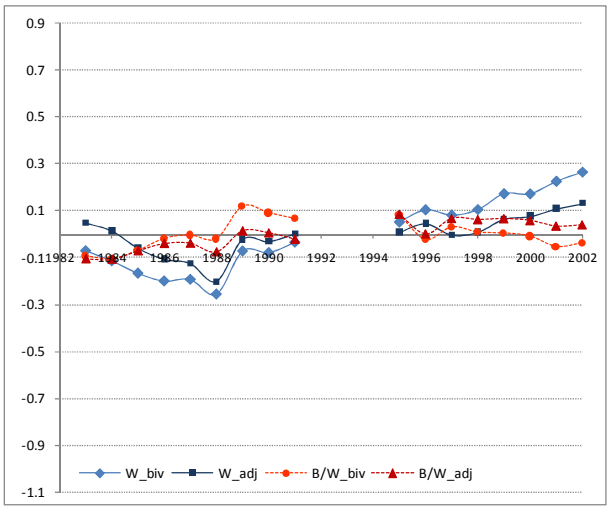


Figure 1e. Log-Odds of Whites and of Blacks Relative to Whites - MC

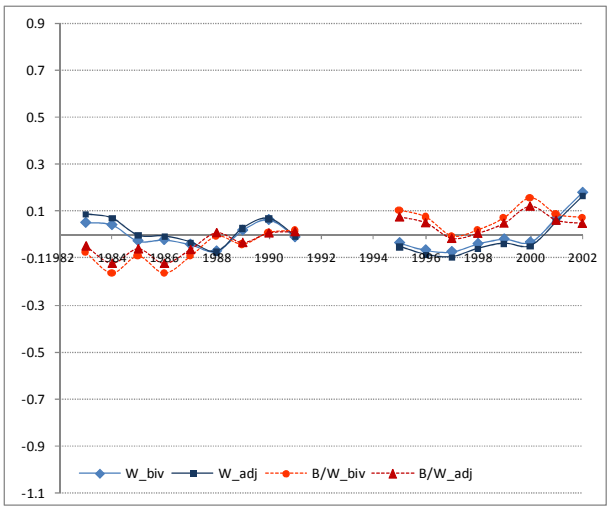
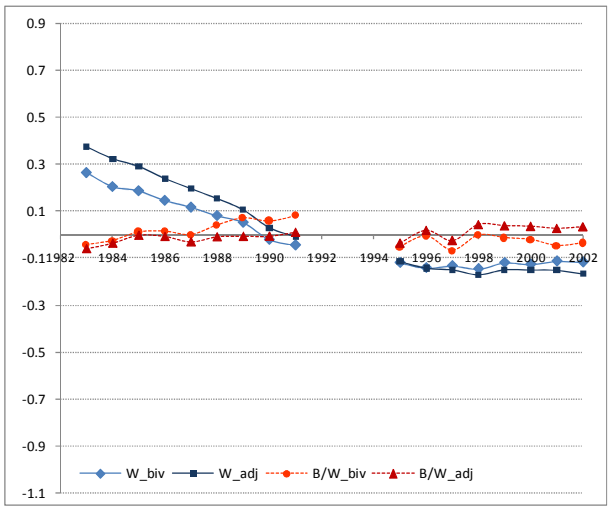


Figure 1f. Log-Odds of Whites and of Blacks Relative to Whites – OTHER



Appendix. Log-Odds based on the Multilevel Random Coefficient Model

		CA		SIDS		RDS		SG/LBW		MC		OTH.	
		Bivariate	Full	Bivariate	Full	Bivariate	Full	Bivariate	Full	Bivariate	Full	Bivariate	Full
Intercept	White	-6.525 ***	-7.005 ***	-6.989 ***	-7.236 ***	-7.746 ***	-11.167 ***	-7.295 ***	-12.066 ***	-8.161 ***	-12.517 ***	-5.738 ***	-6.526 ***
Race	Black	0.175 ***	-0.251 ***	0.751 ***	0.178 ***	0.881 ***	0.079	1.404 ***	0.555 ***	0.926 ***	0.290 ***	0.917 ***	0.245 ***
Education	No High		0.049 ***		0.690 ***		0.065 ***		0.015		-0.131 ***		0.227 ***
	High		0.075 ***		0.369 ***		0.107 ***		0.114 ***		0.043 *		0.148 ***
	Missing		0.098 ***		0.351 ***		0.138 ***		0.482 ***		0.148 ***		0.244 ***
Maternal Age	10-17 yrs.		0.006		0.233 ***		0.223 ***		0.124 ***		0.101 **		0.087 ***
	35+ yrs.		0.192 ***		-0.700 ***		-0.191 ***		-0.137 ***		-0.148 ***		-0.099 ***
Marital Status	Unmarried		-0.096 ***		0.499 ***		0.041 **		0.114 ***		0.059 **		0.171 ***
Parity	First		-0.177 ***		-0.449 ***		0.051 ***		0.228 ***		0.230 ***		-0.006
	High		0.086 ***		0.412 ***		0.044 **		-0.104 ***		-0.051 *		0.138 ***
Previous Loss	Yes		-0.008		0.071 ***		0.193 ***		0.476 ***		0.505 ***		0.179 ***
Prenatal Trimester	Second		-0.017		0.300 ***		-0.172 ***		-0.421 ***		-0.415 ***		-0.033 ***
	Third or None		-0.025		0.442 ***		0.252 ***		0.420 ***		0.148 ***		0.300 ***
Plurality	Plural		-0.901 ***		-0.004		0.223 ***		-0.342 ***		1.154 ***		-0.097 ***
Prematurity	Preterm		0.575 ***		0.310 ***		2.870 ***		3.827 ***		3.413 ***		1.405 ***
Low Birth Weight	Low		2.493 ***		0.864 ***		4.246 ***		4.877 ***		4.371 ***		2.215 ***
Random Effect	**Birth Cohort**												
Intercept (White)	**1983**	0.247 ***	0.283 ***	0.386 ***	0.427 ***	0.718 ***	0.890 ***	-0.069	0.050	0.052	0.090 **	0.265 ***	0.376 ***
	1984	0.212 ***	0.255 ***	0.338 ***	0.384 ***	0.664 ***	0.848 ***	-0.112 **	0.017	0.043	0.072 *	0.206 ***	0.325 ***
	1985	0.206 ***	0.242 ***	0.362 ***	0.400 ***	0.657 ***	0.817 ***	-0.164 ***	-0.058	-0.026	-0.002	0.188 ***	0.292 ***
	1986	0.152 ***	0.186 ***	0.360 ***	0.390 ***	0.557 ***	0.702 ***	-0.197 ***	-0.105 ***	-0.021	-0.005	0.147 ***	0.240 ***
	1987	0.116 **	0.144 **	0.316 ***	0.340 ***	0.505 ***	0.626 ***	-0.191 ***	-0.123 ***	-0.041	-0.030	0.118 ***	0.197 ***
	1988	0.113 **	0.140 **	0.329 ***	0.347 ***	0.450 ***	0.571 ***	-0.253 ***	-0.199 ***	-0.070 *	-0.071 *	0.082 *	0.155 **
	1989	0.123 **	0.142 **	0.351 ***	0.327 ***	0.573 ***	0.644 ***	-0.070	-0.020	0.019	0.029	0.054	0.107 *
	1990	0.087 *	0.105 *	0.273 **	0.238 **	0.289 *	0.346 *	-0.077	-0.029	0.065 *	0.071 *	-0.019	0.031
	1991	0.023	0.033	0.268 **	0.224 *	0.201	0.229	-0.033	0.004	-0.009	-0.006	-0.043	-0.006
	1995	-0.050	-0.063	-0.171	-0.182	-0.308 *	-0.373 *	0.052	0.010	-0.034	-0.050	-0.117 ***	-0.114 *
	1996	-0.088 *	-0.107 *	-0.228 *	-0.236 *	-0.370 **	-0.457 **	0.104 *	0.047	-0.065	-0.084 *	-0.139 ***	-0.141 **
	1997	-0.106 *	-0.134 **	-0.279 **	-0.283 **	-0.458 **	-0.576 ***	0.082	-0.002	-0.072 *	-0.094 **	-0.131 ***	-0.149 **
	1998	-0.129 **	-0.161 **	-0.335 ***	-0.340 ***	-0.423 **	-0.560 **	0.105 *	0.011	-0.038	-0.058	-0.144 ***	-0.169 ***
	1999	-0.242 ***	-0.277 ***	-0.413 ***	-0.416 ***	-0.657 ***	-0.807 ***	0.172 ***	0.066 *	-0.018	-0.036	-0.117 ***	-0.148 **
	2000	-0.218 ***	-0.251 ***	-0.463 ***	-0.466 ***	-0.785 ***	-0.928 ***	0.172 ***	0.078 **	-0.031	-0.047	-0.124 ***	-0.149 **
	2001	-0.234 ***	-0.277 ***	-0.539 ***	-0.546 ***	-0.776 ***	-0.940 ***	0.225 ***	0.110 ***	0.072 *	0.057	-0.111 **	-0.151 **
	2002	-0.211 ***	-0.262 ***	-0.552 ***	-0.562 ***	-0.823 ***	-1.007 ***	0.264 ***	0.132 ***	0.182 ***	0.167 ***	-0.115 ***	-0.167 ***
Race Slope (Black)	**1983**	-0.014	-0.030	-0.019	-0.071	-0.276 ***	-0.346 ***	-0.090 *	-0.104 *	-0.074	-0.047	-0.041 *	-0.059 **
	1984	-0.052	-0.083 *	-0.070	-0.120 *	-0.281 ***	-0.337 ***	-0.102 *	-0.106 **	-0.161 *	-0.122 *	-0.023	-0.035
	1985	-0.004	-0.005	-0.128 ***	-0.179 **	-0.275 ***	-0.330 ***	-0.067	-0.070	-0.090	-0.060	0.013	-0.002
	1986	-0.010	-0.022	-0.076 *	-0.122 **	-0.251 ***	-0.320 ***	-0.017	-0.039	-0.162 *	-0.122 *	0.016	-0.007
	1987	-0.019	-0.042	-0.088 *	-0.146 **	-0.153 *	-0.229 **	-0.003	-0.037	-0.090	-0.063	0.002	-0.030
	1988	-0.020	-0.057	-0.110 **	-0.175 ***	-0.097	-0.201 **	-0.017	-0.074	-0.005	0.010	0.042 *	-0.008
	1989	-0.030	-0.088 **	-0.075 *	-0.130 **	-0.047	-0.178 *	0.119 ***	0.015	-0.038	-0.032	0.072 ***	-0.006
	1990	0.001	-0.035	-0.035	-0.082	0.074	-0.029	0.091 *	0.007	0.011	0.009	0.059 **	-0.006
	1991	0.012	-0.024	-0.050	-0.102 *	0.096	-0.004	0.067	-0.020	0.020	0.014	0.082 ***	0.011
	1995	0.001	0.004	0.090 *	0.114 *	0.090	0.117	0.085 *	0.086 *	0.105	0.078	-0.049 *	-0.035
	1996	0.025	0.053	0.086 *	0.120 *	0.163 *	0.227 **	-0.021	0.002	0.077	0.053	-0.004	0.019
	1997	-0.003	0.013	0.068	0.110 *	0.191 **	0.274 **	0.032	0.068	-0.003	-0.014	-0.064 **	-0.024
	1998	0.009	0.036	0.047	0.099	0.114	0.215 *	0.010	0.064	0.022	0.008	0.001	0.045 *
	1999	0.039	0.088 *	0.091 *	0.159 **	0.136	0.243 **	0.006	0.067	0.074	0.051	-0.011	0.039 *
	2000	0.012	0.045	0.117 *	0.198 ***	0.186 *	0.302 **	-0.007	0.060	0.158 **	0.124 *	-0.019	0.037
	2001	0.020	0.067	0.066	0.155 **	0.156	0.292 **	-0.050	0.035	0.090	0.064	-0.044 *	0.028
	2002	0.035	0.086 *	0.091	0.193 ***	0.179 *	0.309 **	-0.035	0.041	0.075	0.050	-0.033 *	0.035 *
Cohort Variance													
Intercept	**White**	0.028 **	0.039 **	0.133 **	0.143 **	0.328 **	0.493 **	0.024 **	0.007 **	0.005 *	0.040 **	0.019 **	0.038 **
Race	**Black**	0.001	0.004 *	0.008 *	0.008 *	0.034 **	0.067 **	0.005 *	0.005 *	0.011 *	0.036 **	0.002 *	0.001 *

Source: NCHS Linked Birth/Infant Death Cohort Files, 1983-1991 and 1995-2002.

Note: * at $p \leq 0.05$; ** at $p \leq 0.01$; *** at $p \leq 0.001$ (two-tailed tests).

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