A Deadly Disparity: A Unified Assessment of the Black-White Infant Mortality Gap

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Abstract. We provide a unified assessment of a striking disparity in the United States: the differential rate at which white and black infants die. We separate the overall mortality gap into three temporal components – fitness at birth, conditional neonatal mortality, and conditional post-neonatal mortality – and quantify the extent to which each of the components can be predicted using a flexible reweighting method. Almost 90 percent of the overall mortality gap is due to differential fitness at birth, little of which can be predicted by racial differences in background characteristics. The remaining mortality gap stems from conditional post-neonatal mortality differences, nearly all of which can be predicted by background characteristics. The predictability of the mortality gap has declined substantially over the past two decades, largely because the mortality gap among extremely low-fitness infants is increasingly unrelated to background characteristics.

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1. Introduction

The infant mortality rate (IMR) is a fundamental and commonly cited measure of a population's health. In 2005, the U.S. IMR was 6.86 per 1000 live births, placing it 39th among all countries.[1] This overall assessment, however, masks tremendous racial disparities. In 2005, the IMR was 13.63 among non-Hispanic blacks, compared with 5.76 for non-Hispanic whites. The rate at which black infants die is also strikingly high relative to infants in many other countries: considered separately, non-Hispanic blacks would rank 67th internationally, just below Qatar and Uruguay.

The large racial IMR gap in the U.S. has been the subject of numerous studies that have produced three consistent findings. First, although infant mortality has declined substantially for each group over the last two decades, the IMR gap is now larger than it was in the early 1980s in relative terms. Second, most of the IMR gap is due to differential fitness of infants at birth, where fitness is typically defined in terms of birth weight, gestational age, or a combination of the two. Third, much of the gap cannot be predicted by racial differences in background characteristics such as maternal age and educational attainment.

In this paper, we develop a unified approach to studying the racial IMR gap that allows us to assess when the overall IMR gap emerges and what parts of it can and cannot be predicted. This approach includes specifying a decomposition of the IMR gap into three distinct temporal components that are related to concepts frequently examined in previous studies: fitness at birth as measured by birth weight or gestational age, conditional neonatal mortality, and conditional post-neonatal mortality. We then apply a reweighting procedure to examine how each temporal

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[1] UNICEF (2009) provides international rankings of infant mortality rates, and Matthews and MacDorman (2008) provide the race-specific U.S. rates.

component is related to an extensive set of predetermined background characteristics available on birth certificates. These characteristics include several that are routinely used in previous studies, such as the education, age, and marital status of the mother, and two characteristics that are not: the state in which the birth occurred and whether it occurred in a populous county. These latter two characteristics are potentially useful additions because geographic variation in many important inputs for the production of healthy infants, such as employment opportunities and health facilities and social services, may be correlated with geographic variation in race.

Our analysis produces three major findings. First, we confirm that most of the racial IMR gap (almost 90 percent) is already predictable from differential fitness at birth. We further find that, conditional on fitness at birth, differences in neonatal mortality *reduce* the IMR gap slightly and differences in post-neonatal mortality account for the rest of the gap. Second, an extensive list of background characteristics can predict about one-third of the gap, with education and marital status being the most important factors. We find that these background characteristics predict nearly the entire conditional post-neonatal component and very little of the fitness component. Importantly, the measured role of background characteristics is somewhat sensitive to specification. Third, we find that the predictability of the IMR gap has declined over time, largely because the mortality gap among extremely low-fitness infants is increasingly unrelated to background characteristics. This final conclusion is troubling from a policy perspective because it is difficult to design effective policy to reduce the IMR gap when there is little information as to why it exists.

2. Background and Literature Review

Infant mortality declined substantially for both whites and blacks in the U.S. in the latter half of the 20th century. Overall, the U.S. IMR dropped from 26 per 1000 live births in 1960 to 6.9 in

2000 and then remained roughly constant through 2005 (Kung et al., 2008). Although the absolute black-white gap has declined over most of this period, the relative gap has not. The ratio of the non-Hispanic black to non-Hispanic white IMR was 2.1 in 1983, but it has remained above 2.3 since 1990 (National Center for Health Statistics, 2009, Table 18). The size and persistence of this race gap has attracted the attention of numerous economists.[2]

To shed light on the sources of these gaps, researchers have long decomposed differences in IMR, either over time or across groups, into those associated with differences in fitness at birth and those associated with differences in IMR conditional on fitness at birth, where fitness at birth is measured by birth weight, gestational age, or some combination of the two.[3] This distinction has proven useful because extremely small infants and extremely preterm infants, categories with a great deal of overlap, account for disproportionately large shares of infant deaths. For example, infants born at less than 1000 grams accounted for only 0.8 percent of all live births in 2005, but they accounted for 48.2 percent of all infant deaths (Matthews and MacDorman, 2008). In an early study that distinguishes between infant fitness and mortality conditional on fitness, Lee, Paneth et al. (1980) examined the decline in neonatal mortality between 1950 and 1975 and used birth weight to measure infant fitness. They found that the decline in neonatal mortality over this period was entirely due to declines in mortality conditional on birth weight. Several

[2] For example, see Joyce (1987), Chay and Greenstone (2000), Ellen (2000), Fishback, Haines, and Kantor (2001), Meara (2001), Miller (2003), Collins and Thomasson (2004), Costa (2004), and Almond, Chay and Greenstone (2006).

[3] Many experts believe gestational age is a better measure of infant fitness (and therefore a better basis for categorization) than is birth weight, with some experts questioning whether birth weight is causally linked to mortality at all (Wilcox, 2001; Wilcox and Russell, 1986). Birth weight is often the focus of the empirical literature because it is likely to be measured more accurately.

studies have shown that the more recent mortality declines have also been due largely to falling conditional mortality rates rather than improvements in fitness at birth.[4]

Several studies have used decomposition methods to specifically study the black-white IMR gap. For example, Carmichael and Iyasu (1998) find that racial differences in birth weight accounted for 93 percent of the gap in 1983 and 88 percent of the gap in 1991. Schempf, Branum, et al. (2007) studied black-white IMR gaps in 1990 and 2000 using similar methods, focusing instead on gestational age as the measure of infant fitness. They also found that fitness differences could account for most of the IMR gap (roughly 78 percent of it in 2000), but that the role of fitness differences has declined over time.

Another analytic distinction common in the literature involves distinguishing between mortality during the neonatal period (the first 28 days) and the post-neonatal period (the remainder of the first year). Carmichael and Iyasu (1998) and Schempf, Branum, et al. (2007) decompose the racial gaps in neonatal mortality and post-neonatal mortality into their fitness at birth components and mortality conditional on fitness at birth components. Both studies find that the racial gap in neonatal mortality is more than completely predicted by differences in fitness at birth, implying that blacks have a slight advantage in neonatal mortality conditional on fitness. This black advantage, observed mainly at the lowest birth weights and smallest gestational ages, has been declining over time (Alexander et al., 2008). The racial gap in post-neonatal mortality is more evenly split between the fitness and conditional mortality components.

Researchers have also examined whether IMR differences across groups are predicted by differences in the background characteristics of group members. Hummer, Biegler et al. (1999)

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[4] Whether the distributions of birth weight and gestational age have become less favorable or (slightly) more favorable over time depends on the precise time periods and characteristics of the populations studied. See Buehler et al. (1987), Carmichael and Iyasu (1998), Schempf et al. (2007) and Alexander et al. (2008).

use pooled Vital Statistics data for the years 1989-91 and a logistic regression model to examine the role of nativity on IMR differences across races. They find that foreign-born mothers generally experience lower infant mortality but that a substantial black-white IMR gap remains even after controlling for nativity and a host of other background characteristics. Frisbie, Song et al. (2004) applied similar methods to analyze the black-white IMR gap in 1989-90 and 1995-98, focusing on reductions in IMR due to the introduction of pulmonary surfactant therapy. Their results suggest that a large black-white IMR gap remains after controlling for several background characteristics. Similarly, Eberstein, Nam, and Hummer (1990) and Miller (2003) find that large racial IMR gaps exist even after controlling for background characteristics.

The literature on the role of background characteristics has remained distinct from the studies that decompose infant mortality into components related to fitness at birth and mortality conditional on fitness. This separation is likely due to differences in methodology: regression-based approaches to assessing the role of background characteristics are not readily modified to incorporate decompositions. Previous authors have explicitly recognized the need for integrating these two approaches.[5] In the remainder of this paper, we develop and apply methods that allow us to combine these literatures in order to shed additional light on why the racial IMR gap persists.

3. Methods

In the first subsection, we show that the overall IMR gap can be decomposed into three temporal components: fitness at birth measured by birth weight or gestational age, neonatal

[5] Alexander et al. (2008) write, "…it should be emphasized that the [fitness]–specific IMRs provided in this report are based on crude mortality rates for each race group and have not been standardized to control for group variations in maternal risk characteristics. Although beyond the scope of this investigation, similar analyses of trends in racial disparities in mortality by [fitness] categories are needed that take into account sociodemographic, behavior, and other risk factors."

mortality conditional on fitness at birth, and post-neonatal mortality conditional on fitness at birth and surviving the neonatal period. In the second subsection, we develop a reweighting procedure that allows us to examine the extent to which differences in IMR and its components can be predicted by background characteristics.

3.1. Decomposing IMR into its Temporal Components

Let birth weight be the indicator of fitness at birth.[6] For K unique and exhaustive categories of birth weight, define π to be a K×1 vector of infant mortality rates and s to be a K×1 vector of the shares of births that occur in the respective birth weight categories.[7] The actual infant mortality rate for group g can then be written as

(1) $$IMR_g = \pi_g ' s_g \,.$$

This framework allows for the calculation of counterfactual infant mortality rates, where the birth weight distribution for one group is combined with the conditional infant mortality rates for another group. For example, we could compute a counterfactual infant mortality rate by applying the conditional infant mortality rates for blacks (denoted "*b*" hereafter) to the birth weight distribution for whites (denoted "*w*" hereafter):

(2) $$IMR(g_\pi = b, g_s = w) \equiv \pi_b ' s_w \,.$$

Conventionally, the neonatal mortality rate (NMR) and the post-neonatal mortality rate (PMR) are both defined as the number of deaths in the relevant period per 1000 live births. This definition implies that IMR equals the sum of NMR and PMR. Applying (2) to these mortality

[6] We develop our methods in terms of birth weight only for convenience. We consider birth weight and gestational age as alternative indicators of fitness at birth in the empirical results below.
[7] In algebraic expressions for IMR and its components, we treat the π's as probabilities of death. When we show or discuss values of IMR, we multiply the probabilities by 1000 in order to report deaths per 1000 live births.

rates, we can also define a vector of conditional neonatal mortality rates, denoted π_g^n, and a vector of conditional post-neonatal mortality rates, denoted π_g^p, which must also sum to the conditional infant mortality rate vector ($\pi_g = \pi_g^n + \pi_g^p$). To better focus on differences during the post-neonatal period, we also define a vector of post-neonatal mortality rates that is conditional on both birth weight and on surviving the neonatal period, denoted π_g^{pc}. Based on these definitions, it is straightforward to verify that

$$(3) \qquad IMR_g = \pi_g^{n}{}' s_g + \pi_g^{pc}{}'[(1-\pi_g^n) \bullet s_g] \ ,$$

where the dot operator "$\bullet$" denotes element-by-element vector multiplication. The vector $[(1-\pi_g^n) \bullet s_g]$ contains the fractions of births in each birth weight category who survive the neonatal period.

Using the counterfactual framework of (2) and the temporal definitions of (3), the difference in the infant mortality rate between blacks and whites may be written as

$$(4) \qquad IMR_b - IMR_w = \pi_b{}'(s_b - s_w) + (\pi_b^n - \pi_w^n)'[(1-\pi_b^{pc}) \bullet s_w] + (\pi_b^{pc} - \pi_w^{pc})'[(1-\pi_w^n) \bullet s_w] \ .$$

The first component in (4) isolates the role of differences in the birth weight distributions $(s_b - s_w)$, the second component isolates the role of differences in conditional neonatal mortality rates ($\pi_b^n - \pi_w^n$), and the third component isolates the role of differences in conditional post-neonatal mortality rates among those infants who survive the neonatal period ($\pi_b^{pc} - \pi_w^{pc}$). As is typical with these types of decompositions, alternative representations involving similar components exist. We describe one such alternative in the appendix and present select results based on it.

A useful feature of this three-way decomposition is that it clearly separates infant mortality rate gaps into three temporal components that are frequently discussed in the literature: fitness differences apparent at birth, conditional mortality differences during the first 28 days, and conditional mortality differences during days 29 through 365. However, we stress that these components are only suggestive of when the underlying causes of mortality differences arise because birth weight and gestational age are only indicative of the prenatal experience of the fetus and mother, not complete measures of infant fitness. Thus, the conditional neonatal mortality component may still reflect processes that began in utero and the conditional post-neonatal component may reflect processes that began in utero and during the first 28 days of life.

3.2. Predicting IMR and its Temporal Components

The decomposition of an IMR gap into its temporal components can provide important information as to when the gap arises, but it cannot tell us why the gap arises. For example, we know that birth weight differences play an important role, but why do blacks and whites have different birth weight distributions? In this section, we develop methods to examine how background characteristics affect infant mortality in order to begin to explore why racial differences exist.

Previous research has examined the role of background characteristics by estimating regression-based models of infant mortality, including indicator variables for racial/ethnic groups and a variety of individual characteristics (and sometimes also measures of birth weight or gestational age). Some studies also explore the underlying elements of infant mortality by adopting different dependent variables, such as an indicator for low birth weight or an indicator for neonatal death. These approaches take a step toward our goal of describing precisely when

the IMR gap emerges and what can be predicted by background characteristics, but they do not provide a framework to fit the underlying pieces together.

We develop an approach that is based on a reweighting procedure pioneered by DiNardo, Fortin, and Lemieux (1996; hereafter DFL).[8] The intuition for the method is straightforward: one constructs weights so that, for example, the reweighted white population has the same distribution of observable characteristics as the black population, and then one analyzes outcomes in the reweighted white population. Importantly, DFL reweighting procedures can be used to examine how background characteristics affect the entire distribution of an outcome variable, not just a particular feature of a distribution such as its mean. Additionally, we can use this method to assess the extent that background characteristics predict the various temporal components of IMR gaps developed in the previous section. All of the appropriate algebraic relationships are maintained because all of the quantities are tabulated from the same reweighted data.

More formally, let $f(y \mid g)$ be the probability density of an outcome *y* for group *g* and let $F(x \mid g)$ be the cumulative distribution of background characteristics *x* for group *g*. We can then write the group-specific density of *y* as

(5) $$f(y \mid g) = \int_x f(y \mid g, x) dF(x \mid g) \equiv f(y; g_{y|x}, g_x),$$

which is the density of *y* for group *g* conditional on characteristics *x*, integrated over the distribution of characteristics of individuals who are in group *g*. In the shorthand notation given by the last term in (5), $g_{y|x}$ indexes the group whose conditional density $f(y \mid g, x)$ is used,

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[8] Several studies have assessed the asymptotic and finite-sample properties of reweighting methods, including Hirano, Imbens, and Ridder (2003), Wooldridge (2007), and Busso, DiNardo, and McCrary (2009), concluding that their performance in most circumstances is comparable to or better than regression-based and matching methods.

while g_x indexes the group whose distribution of characteristics $F(x \mid g)$ is used. Counterfactual densities can then be defined by choosing the conditional outcome density we observe for one group ($g_{y|x}$) and the characteristics distribution we observe for another group (g_x). For example, we can define the distribution of *y* that would result if whites retained their own conditional outcome density but had the distribution of characteristics observed for blacks,

$$(6) \qquad f(y; g_{y|x} = w, g_x = b) \equiv \int_x f(y \mid g = w, x)\, dF(x \mid g = b)\,.$$

The key insight of DFL is that (6) can be estimated as an integral over a weighted function of the actual density of white individuals, with weights that are simple to construct. Specifically,

$$(7) \qquad f(y; g_{y|x} = w, g_x = b) = \int_x f(y \mid g = w, x)\, \psi_{W \to B}(x)\, dF(x \mid g = w)\,,$$

where $\psi_{W \to B}(x)$ is a weighting function defined as

$$(8) \qquad \psi_{W \to B}(x) \equiv \frac{dF(x \mid g = b)}{dF(x \mid g = w)} = \frac{\Pr(g = b \mid x)}{\Pr(g = w \mid x)} \times \frac{\Pr(g = w)}{\Pr(g = b)}\,.$$

The last equality in (8) follows from Bayes' Rule. The first fraction of the last term can be estimated using a binary model (such as logit) of racial group membership as a function of covariates *x*, and the second fraction involves only the sample proportions of individuals in each group.

Using (8) to reweight the data for whites, we can generate a counterfactual IMR,

$$(9) \qquad IMR_{w^*} \equiv IMR(g_{y|x} = w, g_x = b)\,,$$

where the subscript "*w**" denotes quantities that are calculated from the data of whites that are reweighted to have the background characteristics of blacks. This counterfactual answers the

question, “what would the white IMR have been if whites had the black distribution of background characteristics?” One measure of the black-white IMR gap that can be predicted by differences in background characteristics is therefore IMR_{w*} - IMR_w, where we substitute the weighted white IMR for the actual black IMR.

As in the decomposition approach developed by Oaxaca (1973) and Blinder (1973), an alternative counterfactual exists and typically generates a different answer for how much of the IMR gap can be predicted by background characteristics. Specifically, we could have computed the counterfactual infant mortality rate IMR_{b*}, the mortality rate among the black population reweighted to have the same background characteristics as whites, and then computed the predicted IMR gap as IMR_b - IMR_{b*}, substituting the weighted black IMR for the actual white IMR. We report our main results using both approaches.

Combining DFL reweighting methods with the decomposition of (4) provides a flexible and powerful method for understanding the nature of IMR gaps. Just as the IMR gap can be decomposed into its three temporal components, the predicted IMR gaps (IMR_{w*} - IMR_w) and (IMR_b - IMR_{b*}) each can be decomposed into three temporal components. Comparing the predicted temporal components to the actual temporal components provides a measure of what parts of the overall IMR gap can be predicted by background characteristics.

In addition to assessing the overall contribution of differential background characteristics to racial IMR gaps, we are also interested in isolating the contributions of specific characteristics such as educational attainment. We adopt a simple approach here: we repeat the reweighting procedure using only one of the characteristics at a time to calculate the weights in (8). This

approach is transparent and can be given a formal interpretation based on conditional distributions.[9]

4. Data

Our primary data source is the linked birth/infant death cohort data compiled by the National Center for Health Statistics (NCHS).[10] These datasets have been made publicly available for the years 1985-1991 and 1995-2004. Each cohort data set includes information from the birth certificates of all live births occurring in the U.S. in the relevant calendar year, which are then linked to death certificates for all infants who die within their first year of life. We limit our analysis to births that occur in the fifty U.S. states or the District of Columbia, excluding U.S. territories. NCHS is unable to match a small fraction of death certificates to birth certificates (about 1 percent in 2001); we ignore the unmatched deaths for our analysis. We initially focus on 2001 cohort data because the later years exclude key data elements, as we describe below.

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[9] For example, suppose one is interested in the density of an outcome that would result if whites had (a) the black distribution of educational attainment, (b) their own distribution of all other covariates, conditional on the distribution of educational attainment, and (c) their own mapping from all characteristics to the outcome. To calculate this density, partition the vector of background characteristics *x* into two components, x_1 and x_2, letting x_1 denote educational attainment and x_2 denote all other background characteristics. Consider the counterfactual density

$$f(y; g_{y|x_1,x_2} = w, g_{x_1} = b, g_{x_2|x_1} = w) \equiv$$
$$\int_{x_1}\int_{x_2|x_1} f(y \mid g_{y|x_1,x_2} = w, x_1, x_2)\, dF(x_2 \mid x_1, g = w)\, dF(x_1 \mid g = b)$$
$$= \int_{x_1}\int_{x_2|x_1} f(y \mid g_{y|x_1,x_2} = w, x_1, x_2)\, dF(x_2 \mid x_1, g = w) \frac{dF(x_1 \mid g = b)}{dF(x_1 \mid g = w)} dF(x_1 \mid g = w).$$

This counterfactual density can be computed by reweighting the white data by

$$\frac{dF(x_1 \mid g = b)}{dF(x_1 \mid g = w)} = \frac{\Pr(g = b \mid x_1)}{\Pr(g = w \mid x_1)} \times \frac{\Pr(g = w)}{\Pr(g = b)}.$$

Drawing on the discussion of (8), these weights can be constructed using an estimated model of group membership as a function of educational attainment only. For a discussion of other approaches for assessing the roles of individual covariates, see Fortin, Lemieux, and Firpo (2010).

[10] See information at http://www.cdc.gov/nchs/linked.htm.

As is standard in the literature, we classify births based on the race and ethnicity of the mother. In our main analyses, we exclude those identified as being of Hispanic ethnicity or for whom ethnicity information is missing. Thus, “whites” and “blacks” refer to non-Hispanic whites and blacks unless otherwise indicated.

Whenever we disaggregate by birth weight, we divide births into cells by individual ounce, leading to 173 cells in 2001.[11] For comparability with other research, we express birth weight in grams when displaying or discussing our results. We express gestational age in integer values of completed weeks using the NCHS-edited gestational age variable provided in the public data files. This measure is derived from the mother’s reported last menstrual period when it is available and consistent with birth weight; otherwise, it is based on a clinical estimate. The small shares of infants for whom birth weight or gestational age is not reported are omitted from the relevant figures but included as separate categories in the analyses reported in tables.

We compute standard errors for all estimated quantities using bootstrap resampling techniques, based on 200 replicates using random sampling with replacement.

Background characteristics. Conceptually, we classify as predetermined background characteristics those observable attributes that are determined prior to information the mother might have received about the fitness of the fetus. Such predetermined characteristics can provide important insight into the factors that cause IMR gaps. In contrast, characteristics that are not predetermined may reflect differences in fetal/infant health that have already emerged. For example, information that a pregnancy is at high risk may lead to a greater number of

[11] While the research literature typically reports birth weight in grams (as do the cohort data sets), birth weight is commonly measured in pounds and ounces in the U.S. A close examination of the data reveals a pronounced heaping of observations at gram values that correspond to ounce values, so we round gram values to the nearest ounce when grouping by birth weight. We then consider 173 ounce-based cells: 9 or fewer ounces, 10 ounces, 11 ounces, …, 179 ounces, 180 or more ounces, and missing.

prenatal visits, inducing a negative association between prenatal care and outcomes and obscuring any causal effect of prenatal care on fitness. Similarly, birth weight and gestational age do not satisfy our conceptual requirement for predetermined background characteristics because they reflect the fitness of the infant at birth, not the factors that determine fitness.

It is important to recognize that associations between predetermined background characteristics and outcomes are only a starting point for understanding the causal mechanisms at work. For example, educational attainment may be associated with lower infant mortality because education imparts knowledge and income that aid the production of a healthy infant, but the association might also reflect that individuals who already possess the skills to produce a healthy infant tend to obtain more education.

Implementing our conceptual definition of predetermined characteristics is not always straightforward due to data limitations and our desire to connect to the previous literature. We include variables that are commonly used in previous studies and are clearly predetermined to information on infant fitness: maternal education, maternal age, previous pregnancy loss (either elective or spontaneous), infant sex, live birth order, and plurality.[12] Two others that we examine, prenatal care and marital status, are less clearly predetermined but are often included in previous studies. In light of this concern about prenatal care, we use only an indicator variable for whether it is begun in the first trimester. Similarly, marital status is measured at the time of

[12] We specify education with five indicator variables (<12 years, 12 years, 13-15 years, 16 years, >16 years), maternal age with six indicator variables (<20, 20-24, 25-29, 30-34, 35-39, and >39), and live birth order with five indicator variables (1st, 2nd, 3rd, 4th, >4th). We use single indicators for whether the infant is male, whether the birth was plural, and whether the mother experienced a previous pregnancy loss (either elective or spontaneous).

birth in birth certificate data, so it might be affected by information on the health of the fetus.[13] We include marital status in our baseline analysis but also consider the effect of its exclusion.[14]

Our baseline analysis departs in two important ways from most previous studies of the black-white IMR gap. First, we include indicators for the state in which a birth occurs (50 states and the District of Columbia) and an indicator for whether the county of occurrence had a population greater than 250,000 people. Many important inputs for the production of healthy infants vary by geography, such as employment opportunities, social services, pollution, and health care access and quality. If blacks tend to live in places that are less conducive to the production of healthy infants than do whites, then these geographic indicators would predict some of the IMR gap. Second, we do not include information on smoking and drinking during pregnancy. These measures are missing much more frequently than are other variables and, more importantly, are not necessarily predetermined to information on fetal fitness. For comparability to previous studies, we provide results that include smoking and drinking in an unpublished appendix.

5. Results

Table 1 provides a detailed look at infant mortality outcomes for all births in 2001. Whites and blacks accounted for 2.9 million of the 4.0 million live births in 2001, with an overall mortality rate per 1000 births of 5.64 among whites and 13.21 among blacks. Roughly two-thirds of the infant mortality rates for whites (3.77 of 5.64) and for blacks (8.91 of 13.21) occurs during the neonatal period. The black IMR is substantially above that of the other race and

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[13] For example, Bachu (1999) reports that among first-time mothers unmarried at conception, 30.5 percent of non-Hispanic whites and 10.2 percent of non-Hispanic blacks were married at the time of birth in the 1990-94 period. It is unknown whether or to what extent the probability that an unmarried woman marries during pregnancy is influenced by information on the health of the fetus. See Cooksey (1990) and Akerlof, Yellen, and Katz (1996) for analyses of the decision of pregnant women to marry before they give birth.

[14] We use the NCHS-imputed marriage variable throughout our analysis. All states except for MI and NY ask directly about marital status in 2001, although other information exists for MI and NY that allows the determination of marital status with reasonable accuracy. Outside of these two states, less than .03 percent of the population is missing marital status; the NCHS assigns these mothers to be married.

ethnic groups. Only 0.6 percent of mothers had unreported ethnicity in 2001, but this population has the highest infant mortality. This last finding is important because it implies that missing data, while not prevalent, are much more common among infants who die.

Figure 1 shows the time trends in infant mortality for the non-Hispanic blacks and whites based on the linked birth/infant death data sets for all available years between 1989 and 2004. We also show the infant mortality time trend for all blacks and whites, so that we can extend the series back to 1985 (only 25 states collected information on ethnicity before 1989). The non-Hispanic white line and "all white" line essentially coincide because Hispanics have very similar IMRs compared to non-Hispanic whites, and the non-Hispanic black line and "all black" line coincide because there are relatively few Hispanic blacks. The figure shows similar IMR declines for all groups between 1985 and 1997, with little or no systematic changes in the following years.

For the analyses that follow, we exclude births with missing values for any of the background characteristics. We further exclude births with missing birth weight or gestational age for graphical analyses related to birth weight and gestational age. Table 2 shows the sample sizes and infant mortality rates for our analysis sample. Again, it is readily apparent that missing data are not a serious problem (the white sample size declines by 2.6 percent and the black sample size declines by 4.8 percent when compared to Table 1), but the infant mortality rates decline, particularly for blacks, implying that missing data are more common among infants who die.[15]

[15] The ratio of the black IMR to the white IMR changes only modestly, from 2.34 to 2.31, when births with missing data are excluded. Appendix Table A1 provides more details on the extent of missing data for each variable.

5.1. IMR and its components: the basic facts

Figure 2 is the starting point for our analysis of the black-white IMR gap in 2001, using birth weight as our indicator of fitness at birth. Panel A shows kernel density estimates of the black and white densities of birth weight, which are smoothed graphs of the *k* elements of s_b and s_w. The black density is clearly shifted to the left of the white density and is noticeably thicker in the left tail. Panel B shows the smoothed IMR conditional on birth weight curves for blacks and whites (smoothed graphs of the *k* elements of π_b and π_w), expressed per 1000 births.[16] The curves look very similar, with the most apparent difference being slightly *higher* white mortality rates among infants born at less than 1500 grams. Taken together, these figures suggest that much of the black-white IMR gap is due to differences in birth weight, not due to differences in mortality conditional on birth weight.

An important drawback of Panels A and B is that it is difficult to assess where the empirically important racial differences exist. For example, although conditional mortality rates are high at birth weights less than 1000 grams, there are relatively few births at these weights. Panel C of Figure 2 addresses this weakness by graphing the number of infant deaths per 1000 live births across birth weight groups (smoothed graphs of the *k* elements of $\pi_b \bullet s_b$ and $\pi_w \bullet s_w$). Panel C clearly shows that the largest black-white gap is at the very low end of the birth weight distribution. The peak of the black curve is roughly 0.015 at 600 grams, meaning that for every 1000 live black babies born, there were about 0.015 infant deaths among infants born between 600 and 601 grams, nearly four times the analogous number for whites. While the most dramatic

[16] We use the kernel density estimator to examine the birth weight density in panel A and a lowess smoother (a locally weighted regression procedure) to non-parametrically examine the relationship between IMR and birth weight, both based on procedures available in Stata, version 9.2. We chose Stata's default bandwidth, which is optimal if the underlying data are Gaussian, for kernel density estimation and a bandwidth of 0.1 for lowess smoothing.

racial disparities arise at the lowest birth weights, the black curve is distinctly above the white curve at birth weights up to about 3750 grams. Thus, even though Panel B showed the conditional IMRs to be very low for blacks and whites in the normal range of birth weights, black mortality rates are higher at these birth weights to an extent that is empirically relevant to the overall IMR gap.

To better demonstrate where the IMR gap emerges, Figure 3 shows the cumulative excess mortality rate of blacks relative to whites. For example, the curve labeled "Actual (b-w)" in Panel A of Figure 3 is the cumulative version of the difference between the black and white mortality rates in Panel C of Figure 2. Thus, the end point of the "Actual (b-w)" curve in Panel A of Figure 3 reflects the full mortality gap between blacks and whites, roughly 7 deaths per 1000 live births. The cumulative IMR gap at 1000 grams is roughly 5, implying that about 70 percent of the total IMR gap occurs among infants weighing less than 1000 grams at birth. Panel B of Figure 3 shows the analogous figure for gestational age, and the conclusion is very similar: excess IMR is most apparent among low gestational age infants, with about 70 percent of the total IMR gap occurring among infants with gestational ages of fewer than 29 weeks.

In Table 3, we move to the three-way decomposition of the IMR gap based on (4), which delineates the roles of fitness at birth, conditional neonatal mortality and conditional post-neonatal mortality. As the first two columns of Panel A show, differences in the birth weight distributions of blacks and whites account for 6.09 excess black deaths per 1000 live births, or 86.7 percent of the total IMR gap. The conditional neonatal mortality component accounts for 0.24 fewer black deaths per 1000 live births (corresponding to negative 3.4 percent of the overall IMR gap). The conditional post-neonatal mortality component accounts for 1.17 excess black deaths per 1000 births, 16.7 percent of the overall IMR gap. The standard errors reported in both

the "Level" and "Fraction" columns are small, indicating that the components of (4) are estimated precisely.

The three-way decomposition could also be based on gestational age as the measure of fitness at birth or could have been computed using an alternative decomposition (see (A1) in the appendix). Panel B of Table 3 shows the results based on (4) and gestational age, and Figure 4 compares the results from Table 3 to decomposition results based on (A1). As is readily apparent from the figure, our basic conclusions are the same across all specifications: roughly 90 percent of the IMR gap is present at birth and the remaining IMR gap emerges during the post-neonatal period.

5.2. Can background characteristics predict the IMR gaps?

Although the results of the previous section are informative as to when the IMR gaps emerge, they provide little information as to why they emerge. To begin to understand the sources of IMR gaps, we next examine the extent to which differences in background characteristics can predict differences in IMR and its components.

To show the potential importance of background characteristics, Table 2 provides summary statistics for mortality rates and background characteristics for whites in column 1 and for blacks in column 2 (with standard errors of the mortality rates in parentheses). Along with higher mortality rates, blacks have substantially different background characteristics: black mothers are more than twice as likely not to have completed high school (.247 versus .119) or be less than 20 years old (.189 versus .082), only 40 percent as likely to be married at the time of birth (.316 versus .776), and 60 percent more likely to give birth in the South (.566 versus .350).

We assess whether these differences in background characteristics can predict the differences in infant mortality, using the reweighting methods described in Section 3.2. Again, the intuition for the method is straightforward: we weight each group's population in order to match the distribution of background characteristics of the other group, and then we examine infant mortality and related outcomes in the reweighted populations. To demonstrate how well the reweighting procedure works, we show summary statistics for the reweighted white population in column 3 and for the reweighted black population in column 4. As is readily apparent, the reweighted white population has background characteristics that are very similar to blacks, and the reweighted black population has background characteristics that are very similar to whites. For example, reweighted whites' marriage rate is similar to that of blacks (.319 versus .316) and reweighted blacks' marriage rate is similar to that of whites (.802 versus .776). These similarities are apparent across all background characteristics.

Comparing the reweighted mortality rates to the actual mortality rates provides direct estimates of the extent to which the background characteristics can predict the mortality gaps. For example, among whites reweighted to have the background characteristics of blacks, the estimated infant mortality rate is 8.28, implying that differential background characteristics account for 41.5 percent ((8.28 – 5.36) / (12.39 – 5.36)) of the overall IMR gap. Similarly, the estimated IMR among reweighted blacks is 10.56, implying that differential background characteristics account for only 26.1 percent ((12.39 – 10.56) / (12.39 – 5.36)) of the overall IMR gap. We return below to the important question of why these predicted roles of background characteristics differ.

A principal advantage of the reweighting method lies in its flexibility in allowing for analyses of detailed outcomes. Returning to Table 3, we use the three-way decomposition of (4)

to assess the extent to which background characteristics can predict each of the temporal components. Specifically, columns 3 and 4 of the table show results from applying (4) to the predicted gap based on the reweighted white data ($IMR_{w*} - IMR_{w}$). Consider the results based on birth weight as the measure of infant fitness at birth. If we partition the 2.92 (= 8.28 – 5.36) predicted gap in deaths per 1000 births among the three temporal components, 1.58 deaths (22.5 percent of the overall IMR gap of 7.03) are due to the birth weight component, 0.23 deaths (3.2 percent of the overall IMR gap) are due to the conditional neonatal mortality component, and 1.11 deaths (15.8 percent of the overall IMR gap) are due to the conditional post-neonatal mortality component. Thus, racial differences in background characteristics can predict nearly all of the racial gap in conditional post-neonatal mortality (1.11 of the 1.17 total component is predicted), but predict relatively little of the birth weight component (1.58 of the 6.09 total component is predicted). Both the actual and predicted conditional neonatal mortality components are small. These overall conclusions are quite stable across both measures of infant fitness and are based on precise estimates.

The final two columns of Table 3 present analogous results based on a comparison of reweighted and unweighted black data ($IMR_b - IMR_{b*}$).[17] As shown above, the predicted gap of 1.83 is 26.1 percent of the overall IMR gap. Whether we use birth weight or gestational age to measure fitness at birth, racial differences in background characteristics again predict little of the birth weight component but predict most of the post-neonatal mortality component. Thus, regardless of whether birth weight or gestational age is used to measure infant fitness, and regardless of whether the predicted gaps are based on reweighted white data or reweighted black

[17] As is apparent in the table, standard errors are roughly 1.5 to 3 times larger for the estimates based on reweighted black data than for the estimates based on reweighted white data. We suspect that this is because there are only about one-fourth as many black births as white births.

data, two overall conclusions emerge from Table 3: (1) most of the overall IMR gap is due to differential fitness, with post-neonatal mortality accounting for the rest of the gap, and (2) a small share of the fitness gap can be predicted by background characteristics, while nearly all of the conditional post-neonatal mortality gap can be.

Figure 3 shows in greater detail how infant mortality is related to infant fitness. Along with the cumulative actual excess deaths, each panel shows the cumulative predicted excess deaths based on the reweighted white data (w*–w) and the reweighted black data (b–b*). Panel A shows the results based on birth weight, and Panel B shows the results based on gestational age. As we saw previously, the actual excess death curves demonstrate that much of the IMR gap emerges in the left tail of the fitness distribution, among births at less than 1000 grams or 29 weeks gestation. In contrast, the predicted excess death curves rise relatively little up to this point, particularly for the reweighted black data curves. Among births at more than 1000 grams or 28 completed weeks, the actual and predicted curves move in a more parallel fashion, implying that the excess mortality is more predictable by background characteristics at higher birth weights and longer gestational ages.

We now consider individually the role of the background characteristics using methods discussed in section 3.2. For example, to isolate the effect of education, we compare the actual white IMR to the IMR among whites reweighted to have the black educational distribution, but their own distribution of all other characteristics conditional on education. This hypothetical difference in IMR is divided by the total IMR gap to obtain a measure of the share of the gap that can be predicted by differences in education. Similar calculations are performed based on reweighting blacks. These results are summarized for each characteristic in Figure 5, where the

"w*–w" bars denote computations based on reweighted white data and the "b–b*" bars are based on reweighted black data.

Figure 5 shows that differences in mother's education and mother's marital status are relatively important in predicting the IMR gap for both reweighting methods. Whites have more education and are more likely to be married (see Table 2), and both of these characteristics are associated with lower infant mortality (see Appendix Table A2). Mother's age differences are also relatively important for the *w*–w* case, and differences in states in which births occur are relatively important for the *b–b** case.[18]

The differences between the *w*–w* and *b–b** bars in Figure 5 also suggest why we can predict more of the IMR gap by reweighting whites than by reweighting blacks. For instance, differences in the share of mothers who are married account for about 17 percent of the IMR gap based on reweighted white data, but only about 8 percent based on reweighted blacks. Appendix Table A2, which shows logit models of infant mortality as a function of background characteristics separately by race, provides similar evidence: the marginal effect of being married on infant mortality is about twice as large for whites as it is for blacks. A similar phenomenon holds for education. Put another way, the central difference between the *w*–w* and *b–b** bars in Figure 5 reflects the fact that marital status and education predict infant deaths much more strongly among whites than among blacks.[19]

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[18] We have also explored the role of individual characteristics by directly estimating logit models of infant mortality. An advantage of such a method is that it allows one to include all characteristics simultaneously, thereby isolating the effects of characteristics that are correlated with each other. The results are broadly similar to those shown in Figure 5, with the primary exception being that the logit results suggest a relatively important role for geography based on the white as well as the black mapping from background characteristics to infant mortality. See the unpublished appendix for these results.

[19] Black-white differences in the relationship between mother's age and adverse birth outcomes are also important, as shown in Appendix Table A2 and previously noted by Geronimus (1996). Specifically, giving birth as a teenager is associated with higher infant mortality for whites when compared to white non-teenage mothers, but lower infant mortality for blacks when compared to black non-teenage mothers. Moreover, a higher proportion of blacks give

In an unpublished appendix, we explore the sensitivity of our results to a variety of analytic decisions we have made. First, we show that a logit model gives similar results for how much of the mortality gap can be predicted by background characteristics. Second, we examine the effects of several sample specification changes: including Hispanics, focusing just on first births, including observations with missing background characteristics, including information on smoking and drinking, and dropping the marriage variable. Generally, these changes lead to an increase in the size of the residual gap, buttressing our overall conclusion that much of the racial IMR is not predictable by background characteristics.

6. Changes over time in the temporal components and predictability of IMR gaps

Thus far, we have only analyzed data from the 2001 birth cohort due to cross-year data comparability issues. We now present evidence on how the roles of the temporal components of IMR gaps and the roles of background characteristics in predicting these components have changed over time.

Figure 6 shows how the predictive power of background characteristics has changed over time. To present smoothed trends, an issue particularly relevant for the results based on the reweighted black data, all data points combine two years of data, with each point graphed at the second of the two years. Series 1 uses our baseline specification from Section 5.2 for the years 1996 through 2001. To extend the series before 1996 or beyond 2001, we must exclude characteristics or states because of missing data issues. Series 2, which can be graphed from 1991 to 2004, excludes New Hampshire, Oklahoma, and Washington and the populous county indicator. Series 3, which can be graphed from 1986 to 2001, excludes California, New York,

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birth as teenagers than do whites. Thus, aligning the black and white distributions of maternal age reduces the IMR gap when the white age-mortality relationship is used, but not when the black age-mortality relationship is used.

Texas, and Washington but includes Hispanics.[20] Panels A and B show results based on reweighted whites ($IMR_{w*} - IMR_w$) and reweighted blacks ($IMR_b - IMR_{b*}$), respectively.

The two panels of Figure 6 tell a consistent story: the predicted share of the racial IMR gap declined by about one-third from the mid 1980s to the early 2000s. For example, based on Series 3 in Panel A, the predicted share declined from .49 in 1986 to .33 in 2001. The sample exclusions shift the Series 2 curves downward compared to Series 1 and including Hispanics in Series 3 shifts the curve down in Panel A (see the unpublished appendix for more details on the inclusion of Hispanics). Both longer series show clear downward trends. Comparing the two panels, the curves fluctuate more from year to year in Panel B than in Panel A, which is expected given the much smaller numbers of black births.

In order to investigate why the predictability of the IMR gap has fallen over time, Figure 7 shows how the relative roles of the temporal components of the black-white IMR gap have changed and how much of these roles can be predicted by background characteristics. For these figures, we focus on the years 1991, 1996, 2001, and 2004, excluding New Hampshire, Oklahoma, and Washington to obtain a consistent time series. For each year, we show the actual shares attributable to the three temporal components (which sum to 1) and the predicted share for each component (which to sum to total share of the yearly IMR gap that is predicted).

First examining the actual components, birth weight is the most important component of the IMR gap in all years, with its overall importance increasing slightly over time (from 85 percent

[20] For Series 2, births in New Hampshire and Oklahoma are excluded because they are missing information on ethnicity for some years before 1995, and births in Washington are excluded because education is missing in some years; the populous county variable is excluded because it is not reported for any state in the 2002 public-use data. For Series 3, Hispanics are included because 25 states did not collect ethnicity prior to 1989. California, New York, Texas, and Washington are excluded because they do not include education in some of the relevant years.

in 1991 to 88 percent in 2004).[21] The roles of the other two components do not exhibit any clear trends. To compute the predicted amounts, we use the reweighted white data. The results suggest that the decline in predictive power shown in Figure 6 stems solely from a reduction in the ability to predict the birth weight component. In 1991, the predicted birth weight component was 28.2 percent of the full IMR gap, and that amount declined to 21.1 percent in 2004. In contrast, the predictability of the other temporal components did not significantly vary across years. Taken together, Figures 6 and 7 show that the IMR gap is increasingly unrelated to racial differences in observable background characteristics, principally because these observable differences are predicting less of the racial disparity in fitness at birth.

Given that residual differences in birth weight became increasingly important from 1991 to 2004, we turn next to assessing where in the birth weight distribution these residual differences arise. Panel A of Figure 8 sets the stage by plotting the cumulative share of excess black deaths per 1000 live births across the birth weight distribution in 1991, 1996, 2001, and 2004, using the same sample selection criteria as Figure 7. These curves are constructed similarly to the "Actual (b-w)" curve in Panel A of Figure 3, except that each is scaled so that its height approaches 1 as birth weight increases to its maximum value. As the figure shows, infants weighing less than 1000 grams at birth accounted for roughly 61 percent of the black-white IMR gap in 1991. This contribution rose to 71 percent in 2004, implying that racial IMR disparities are increasingly due to differences in the extreme left tail of the distribution of birth weight.

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[21] Carmichael and Iyasu (1998) found a small decline between 1983 and 1991 in the share of the black-white IMR gap accounted for by differences in birth weight. Similarly, Schempf, Branum et al. (2007) found a small decline between 1990 and 2000 in the share accounted for by differences in gestational age. In analysis not reported here, our findings are consistent with theirs for those specific years and measures of infant fitness, but we found that these trends were not robust to the inclusion of additional years of data. Based on all available years of data from 1986 to 2004, the roles of both birth weight and gestational age have shown relatively little trend.

Panel B of Figure 8 plots the cumulative share of excess black deaths that can be predicted by differences in background characteristics, based again on the comparison of whites and reweighted whites. Consider the curve labeled “1991”. The height of the rightmost point of the curve is 0.468, implying that 46.8 percent of the black-white IMR gap was predictable in 1991. At 1000 grams, the height of the curve is roughly 0.20, reflecting that 20 percent of the black-white IMR gap in 1991 can be accounted for by the role of differences in background characteristics in producing excess black mortality at birth weights of less than 1000 grams (as shown above, this excess mortality is primarily due to the relatively large number of black infants born at low birth weights). The rightmost endpoint of the 2004 curve is very similar to 2001 and substantially lower than 1991 and 1996, reflecting the overall decline in the predictability of IMR. This analysis clearly shows what a regression-based approach cannot – the decline over time in the predictive power of background characteristics is confined to birth weights below 1000 grams. The curves are roughly parallel at birth weights greater than 1000 grams, implying that the predictive power of background characteristics at these higher birth weights has not diminished.

Taken together, the two panels of Figure 8 show that the black-white IMR gap became increasingly driven by disparities in deaths among extremely low birth weight infants during the 1990s. At the same time, the role of background characteristics in predicting racial differences in deaths at these low birth weights diminished. Both of these phenomena caused the black-white IMR gap to become less predictable between 1991 and 2004. We obtain similar results when we instead measure fitness by gestational age.

7. Summary and Discussion

We have provided a unified assessment of a fundamental and strikingly large disparity in the United States: the differential rate at which white and black infants die. Our primary findings are threefold. First, almost 90 percent of the racial IMR gap reflected differences in fitness at birth in 2001, with the rest of the gap emerging during the post-neonatal period. Second, we find that an extensive list of background characteristics can predict about one third of the infant mortality gap in 2001, with education and marital status being the most important factors. Importantly, most of the mortality differences that emerge during the post-neonatal period can be predicted by background characteristics, but little of the differential fitness at birth can be predicted. Third, we find that the role of racial differences in background characteristics for the IMR gap has declined over the last two decades, reflecting a decline in the predictability of the large racial discrepancy in the number of infant deaths at birth weights of less than 1000 grams.

We develop and apply a methodology that provides a unified picture of how the IMR gap emerges and how the various pieces are related to background characteristics. This analysis includes the development of a decomposition that clearly distinguishes between the temporal components of the infant mortality rate and the application of a reweighting method to assess the role of background characteristics. Importantly, this reweighting method is flexible enough to examine objects like birth weight densities and decomposition components. Moreover, because the reweighting method preserves the underlying algebraic relationships in the data, it shows how background characteristics affect the various components of infant mortality in a framework in which the underlying pieces fit together. Such a unified method could be applied to the study of dynamic processes in other contexts, such as the completion of high school or the formation of families.

Another contribution of our study is its treatment of geography. Many inputs for the production of healthy infants vary by geography, including employment opportunities, social services, pollution, and access to health care, and the populations of blacks and whites are distributed quite differently across states and urban areas. Rather than try to quantify each of the possible avenues through which geographic areas systematically differ, we take the simpler approach of including the state in which a birth occurs as a background characteristic. Our results suggest, on the one hand, that the state and populous county indicators are relatively important contributors to the predicted gap, especially based on the black mapping from characteristics to outcomes.[22] On the other hand, the overall predictive power of all regressors is still small, and large residual gaps remain despite our flexible specification for allowing for geographic differences. Thus, the black-white infant mortality gap is not primarily a result of blacks giving birth in states where infant mortality is high for both races. Because all specifications include state indicators, we would not gain any predictive power by adding other background variables that vary only at the state level, such as Medicaid policy variables or state-level social program variables.

Overall, the paper sheds light on why the racial IMR gap exists and where to direct future research and policy efforts. For example, because there is effectively no racial disparity in conditional neonatal mortality rates, differences in neonatal medical care or parental behaviors immediately after birth are unlikely to be important contributors to the overall gap. Most of the fitness gap at birth cannot be predicted by the expansive set of background characteristics we consider, so there is little evidence why this fitness gap exists; unfortunately, such a null finding provides little direct basis for policy formulation and should continue to be a focus of research.

[22] The estimated role of the state and populous county variables is also relatively important based on the white mapping when we use a logit-based approach; see the unpublished appendix for further details.

In contrast, we find evidence of important disparities in the post-neonatal experience of blacks and whites, with these disparities largely predicted by differences in observable characteristics such as marital status, education, and geography. A potential explanation for this finding is that married women with more education are able to invest more time and resources into the health of their infants when compared to their unmarried, less-educated counterparts. Understanding the specific causal mechanisms that underlie these associations provides an important agenda for future research.

Appendix

An alternative three-way decomposition to (4) is:

(A1) $$IMR_b - IMR_w = \pi_w'(s_b - s_w) + (\pi_b^n - \pi_w^n)'[(1-\pi_w^{pc}) \bullet s_b] + (\pi_b^{pc} - \pi_w^{pc})'[(1-\pi_b^n) \bullet s_b]\ .$$

In both cases, the first term is the birth weight component, the second term is the conditional neonatal mortality component, and the third term is the conditional post-neonatal mortality component. The decomposition in (4) can be thought of as moving sequentially from the black parameters to the white parameters and (A1) can be thought of as sequentially moving from white parameters to black parameters, where ordering is determined by the temporal aspect of the measures. Kitigawa (1955), in a similar setting, proposed averaging the two decompositions. We prefer to present two estimates to give a sense of the range of possible values, and then put forward conclusions that are consistent with both.

When we apply these formulas to the weighted quantities (IMR_{w*} - IMR_w) and (IMR_b – IMR_{b*}), we substitute the weighted quantities of one group for the actual quantities of the other group, and then apply the ordering of (4) and (A1). For example, when we analyze (IMR_{w*} - IMR_w), we substitute “w*” for “b”.

Table A1 provides descriptive statistics for select years of the linked cohort data files used in the paper. Table A2 shows results from logit models of infant mortality as a function of background characteristics, separately by race.

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Table 1. Infant Mortality by Detailed Race/Ethnicity and Timing, 2001 Birth Cohort

			Deaths per 1000 Births		
	Births	Deaths	IMR	NMR	PMR
White (non-Hispanic)	2,327,114	13,128	5.64	3.77	1.87
			(.05)	(.04)	(.03)
Black (non-Hispanic)	590,105	7,793	13.21	8.91	4.29
			(.15)	(.12)	(.09)
Native American*	38,023	354	9.31	4.13	5.18
			(.49)	(.33)	(.37)
Asian or Pacific Islander	195,210	883	4.52	3.05	1.48
			(.15)	(.12)	(.09)
Hispanic	856,632	4,650	5.43	3.59	1.84
			(.08)	(.06)	(.05)
Unclassified Ethnicity	24,562	372	15.15	12.21	2.93
			(.78)	(.70)	(.34)
All	4,031,646	27,180**	6.74	4.51	2.23
			(.04)	(.03)	(.02)

Notes: This table lists the full population from the 2001 linked birth cohort file. Births with listed race but unlisted ethnicity are listed as “Unclassified Ethnicity.” Standard errors are in parentheses.

* Includes American Indians, Eskimos and Aleutian Islanders.

** There were 292 deaths in 2001 that could not be linked back to a birth record; these deaths are dropped for all of our analysis.

Table 2. Average Mortality and Background Characteristics by Race, 2001 Birth Cohort

	Whites (w)	Blacks (b)	Reweighted Whites (w*)	Reweighted Blacks (b*)
Sample size (unweighted)	2,265,332	562,039	2,265,332	562,039
<u>Mortality characteristics</u>				
Infant mortality rate	5.36 (0.05)	12.39 (0.13)	8.28 (0.14)	10.56 (0.32)
Neonatal mortality rate	3.52 (0.04)	8.16 (0.10)	4.98 (0.11)	7.82 (0.28)
Post-neonatal mortality rate	1.85 (0.03)	4.23 (0.08)	3.30 (0.10)	2.74 (0.14)
<u>Background characteristics</u>				
Maternal education (years)				
<12	.119	.247	.249	.120
12	.304	.398	.398	.303
13-15	.243	.233	.234	.239
16	.207	.079	.077	.199
>16	.127	.044	.043	.139
Maternal age				
<20	.082	.189	.187	.073
20-24	.224	.331	.329	.227
25-29	.268	.227	.228	.259
30-34	.269	.155	.155	.268
35-39	.129	.080	.082	.140
>39	.027	.019	.020	.032
Mother married	.776	.316	.319	.802
First trimester prenatal care	.886	.746	.742	.882
Previous loss	.256	.286	.293	.263
Male	.512	.508	.508	.514
Plural birth	.036	.035	.034	.033
Live birth order				
1^{st}	.409	.374	.381	.436
2^{nd}	.342	.296	.292	.328
3^{rd}	.162	.179	.178	.155
4^{th}	.056	.083	.083	.052
5^{th} – 9^{th}	.031	.067	.066	.029
Populous birth county	.561	.704	.714	.560
Census region†				
Northeast	.182	.159	.150	.170
Midwest	.281	.197	.180	.241
South	.350	.566	.589	.373
West	.187	.078	.081	.216

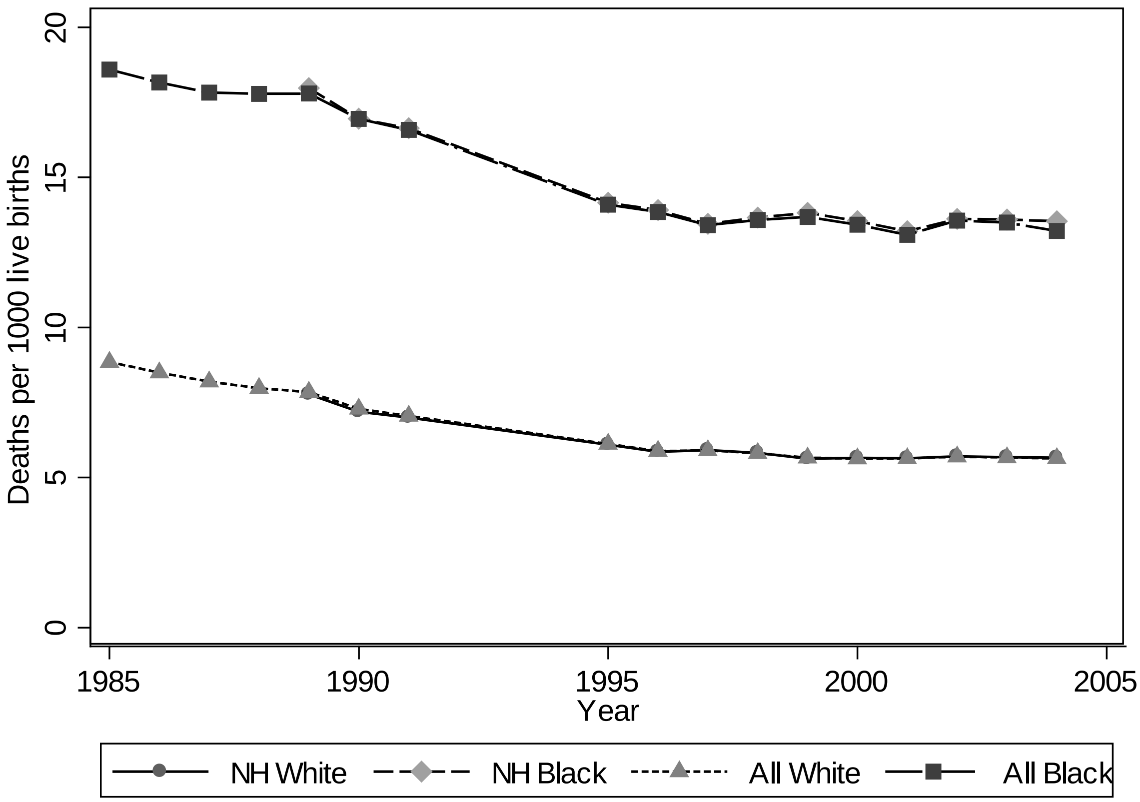
Notes: "White*" ("Black*) refers to white (black) births weighted to have the black (white) distribution of background characteristics. †The logit used to calculate weights includes a complete set of indicator variables for states and the District of Columbia; we report census region results for parsimony.

Table 3. Decomposing IMR Gaps and the Predictive Power of Background Characteristics, 2001 Birth Cohort

	Actual gap (b – w)		Predicted gap (w* – w)		Predicted gap (b – b*)	
	Level	Fraction	Level	Fraction	Level	Fraction
Full Gap	7.03	1	2.92	.415	1.83	.261
	(0.14)		(0.13)	(.024)	(0.34)	(.048)
A: Measuring fitness by birth weight						
Fitness component	6.09	.867	1.58	.225	0.62	.088
	(0.10)	(.012)	(0.08)	(.012)	(0.25)	(.035)
Neonatal mortality component	-0.24	-.034	0.23	.032	0.04	.006
	(0.06)	(.008)	(0.08)	(.010)	(0.17)	(.024)
Post-neonatal mortality	1.17	.167	1.11	.158	1.18	.168
component	(0.08)	(.010)	(0.08)	(.012)	(0.13)	(.018)
B: Measuring fitness by gestational age						
Fitness component	5.61	.798	1.52	.216	1.10	.157
	(0.09)	(.011)	(0.07)	(.011)	(0.19)	(.028)
Neonatal mortality component	-0.03	-.004	0.22	.032	-0.42	-.059
	(0.06)	(.009)	(0.07)	(.010)	(0.18)	(.025)
Post-neonatal mortality	1.44	.205	1.18	.167	1.15	.163
component	(0.08)	(.009)	(0.08)	(.012)	(0.13)	(.018)

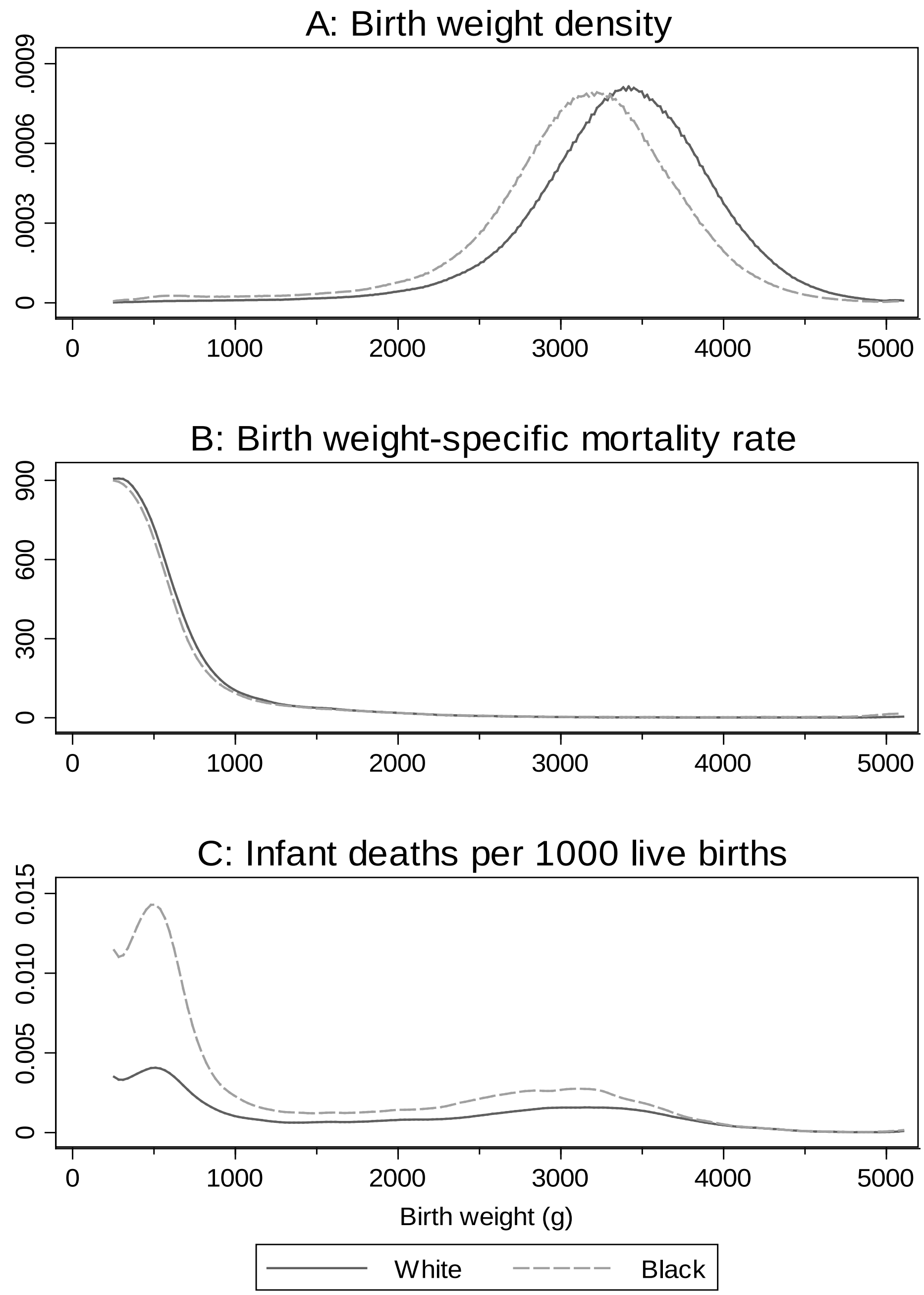
Notes: These decompositions are based on (4) in the text.

Figure 1. Infant Mortality Rate among Whites and Blacks, Multiple Birth Cohorts



Notes: "NH White" refers to non-Hispanic whites, and "All White" refers to whites, regardless of Hispanic ethnicity designation; the black labels are defined similarly.

Figure 2: Decomposing IMR by Birth Weight, 2001 Birth Cohort



Notes: Panel A is a smoothed graph of the birth weight shares (the *k* elements of s_g from (1)). Panel B is a smoothed graph of the birth weight-specific IMR (π_g from (1)), scaled by 1000 live births per birth weight bin. Panel C is a smoothed graph of the number of infant deaths in each birth weight bin per 1000 live births (($\pi_g \bullet s_g$) from (1)).

Figure 3: Cumulative Excess Black Infant Deaths per 1000 Live Births, 2001 Birth Cohort

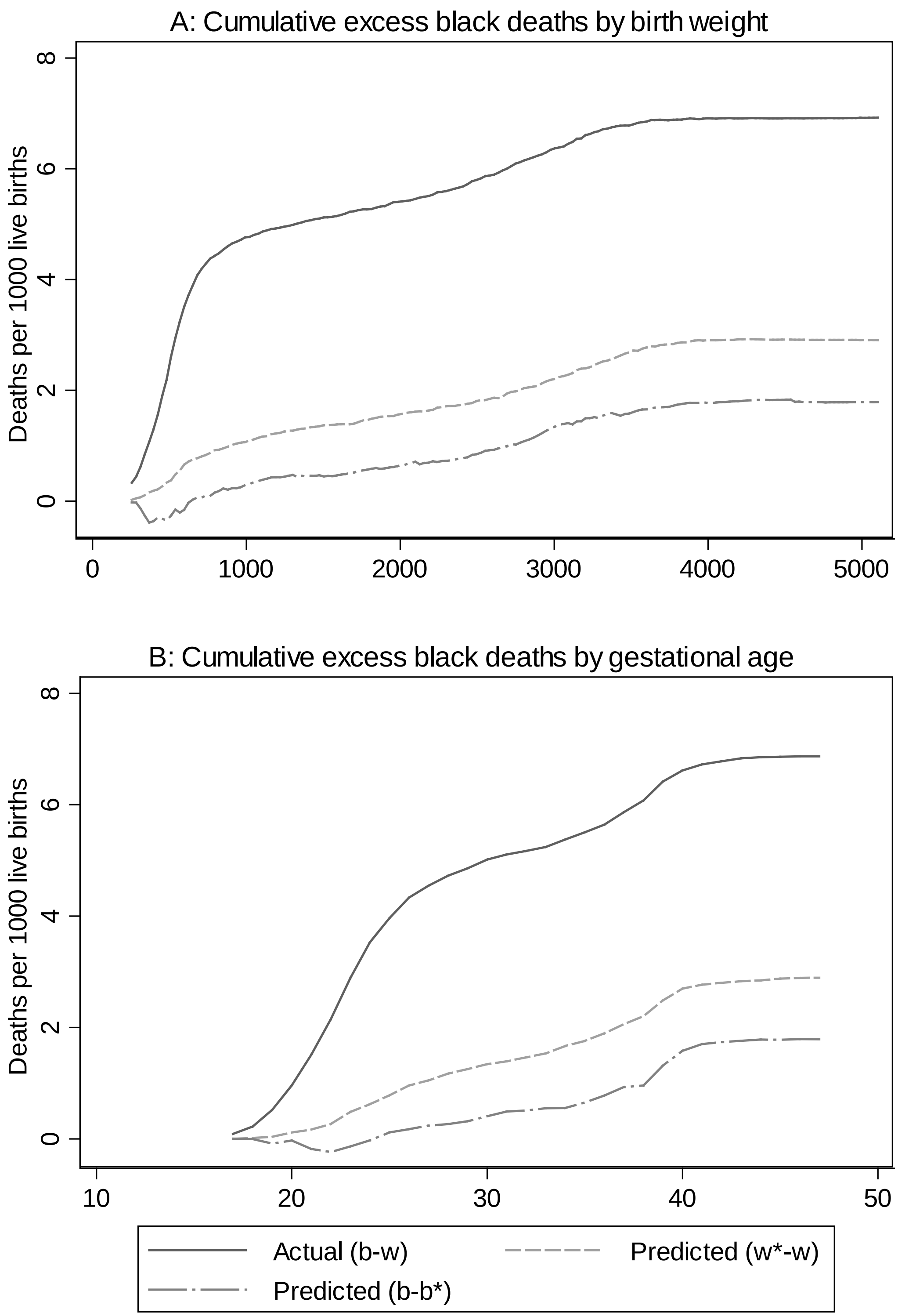
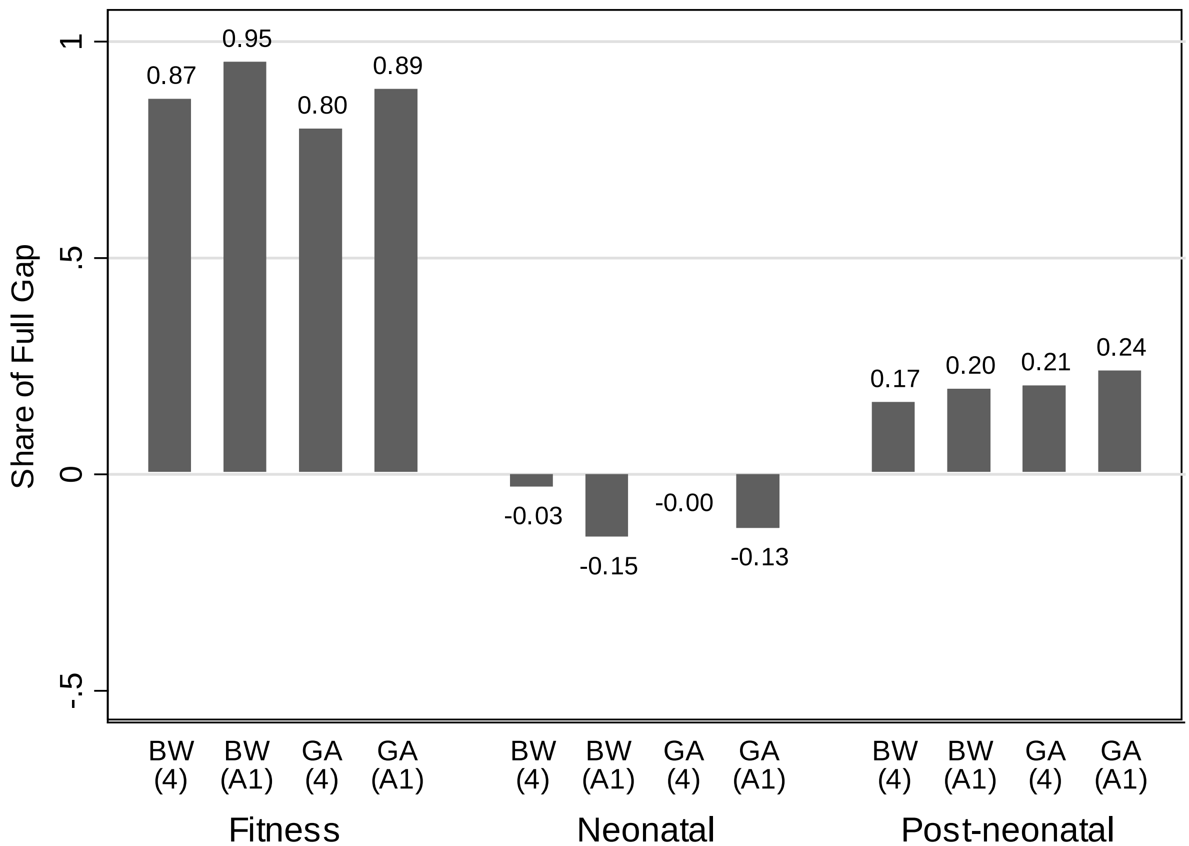


Figure 4: 3-Way Decomposition for Various Methods, 2001 Birth Cohort



Notes: The figure presents the share of the overall IMR gap that is attributable to racial differences in fitness, conditional neonatal mortality, and conditional post-neonatal mortality, based on four different 3-way decompositions: the combination of two different measures of infant fitness at birth (birth weight (BW) and gestational age (GA)) and two different decomposition orders (based on equations (4) and (A1)).

Figure 5: Contribution of Background Characteristics to IMR, 2001 Birth Cohort

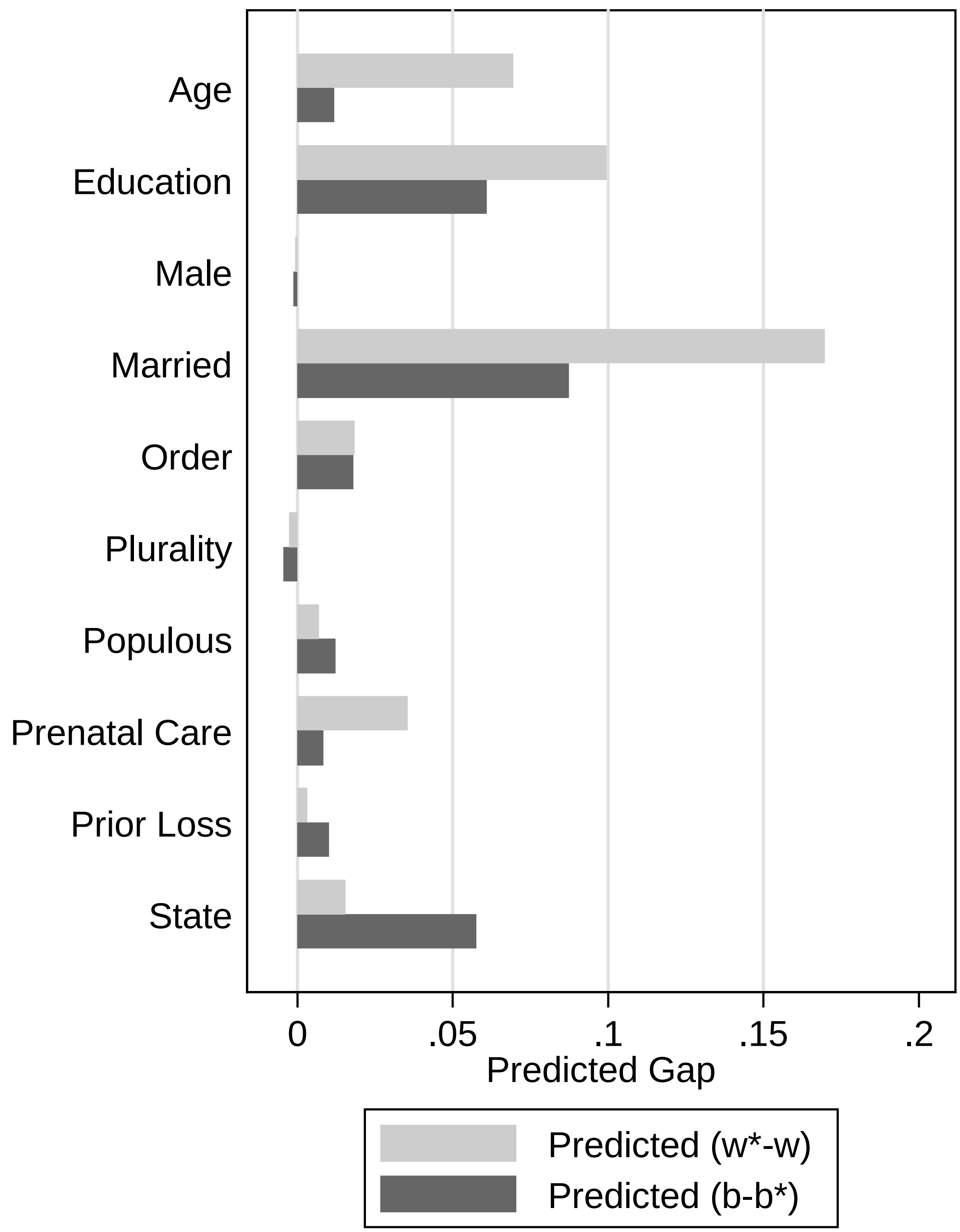


Figure 6: Share of Gap Predicted by Observables Characteristics, Multiple Birth Cohorts

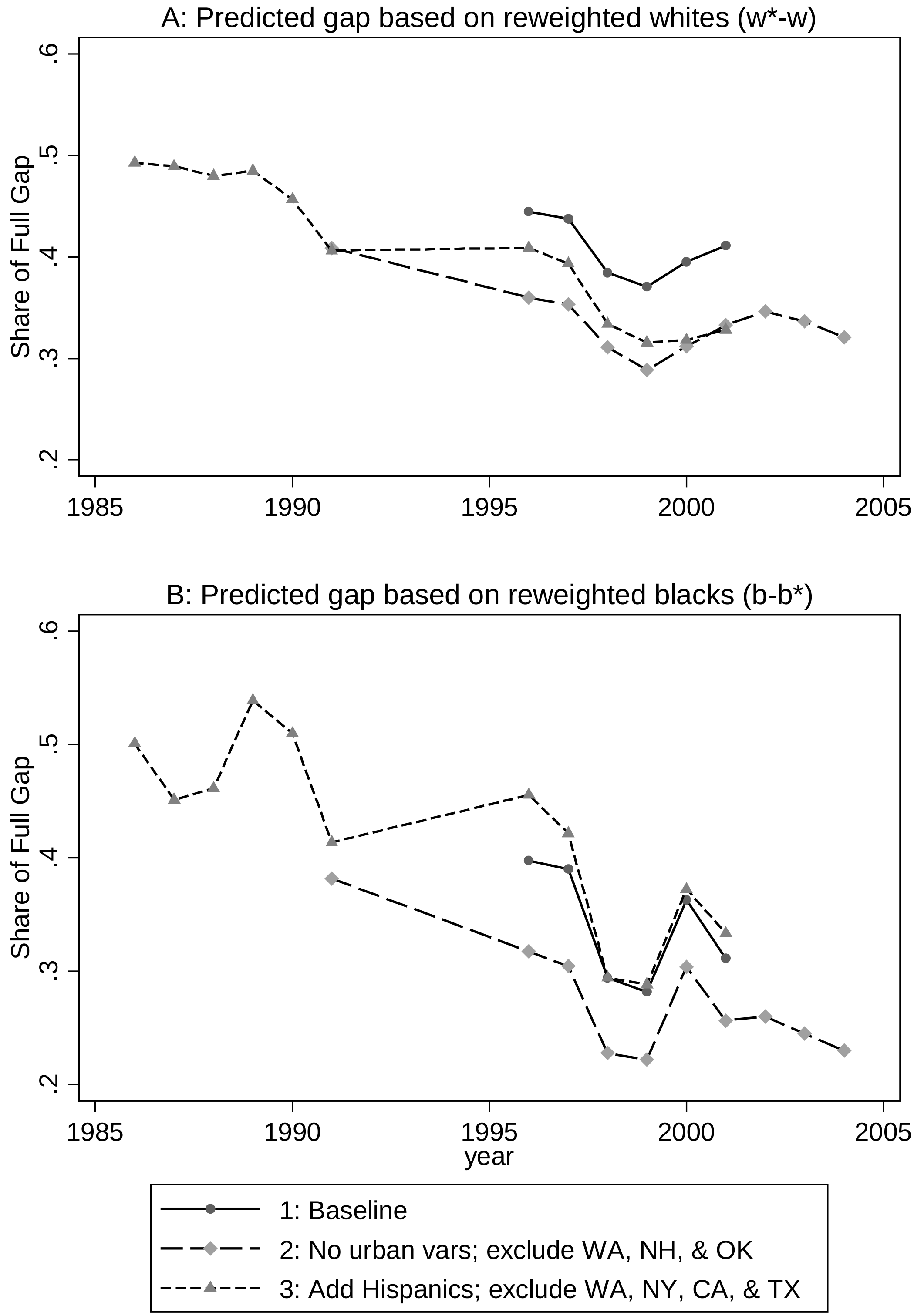


Figure 7: Relative Roles of Birth Weight, Neonatal Mortality, and Post-neonatal Mortality for IMR gaps, Multiple Birth Cohorts

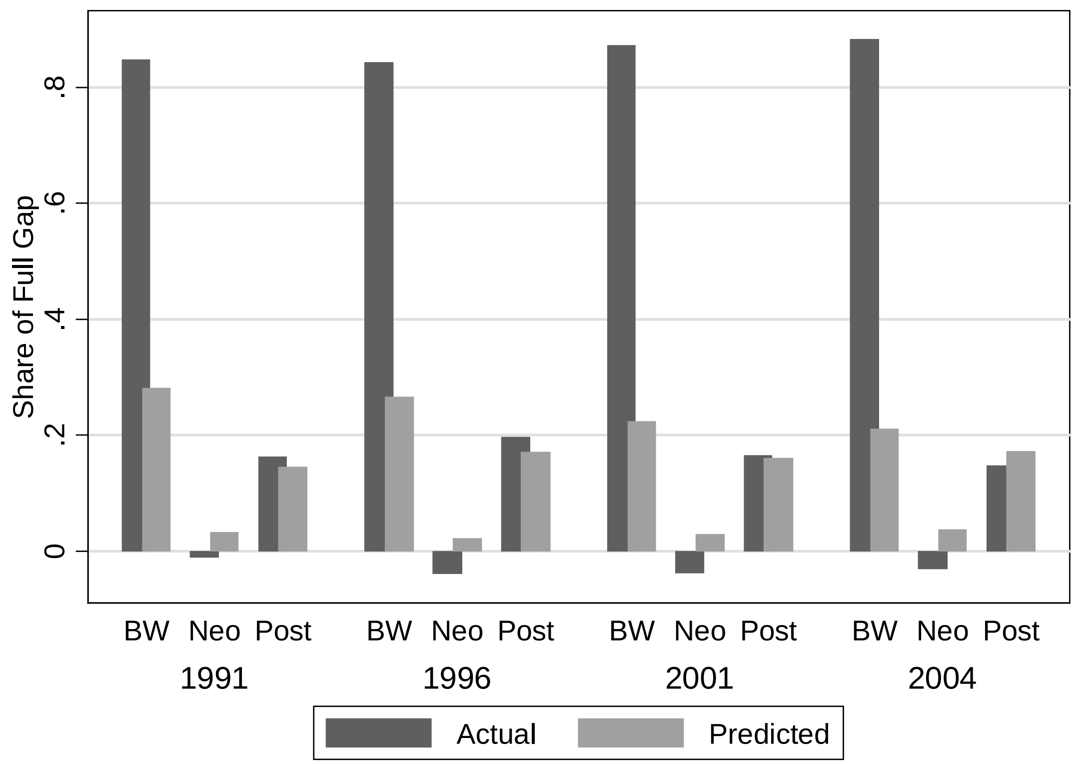
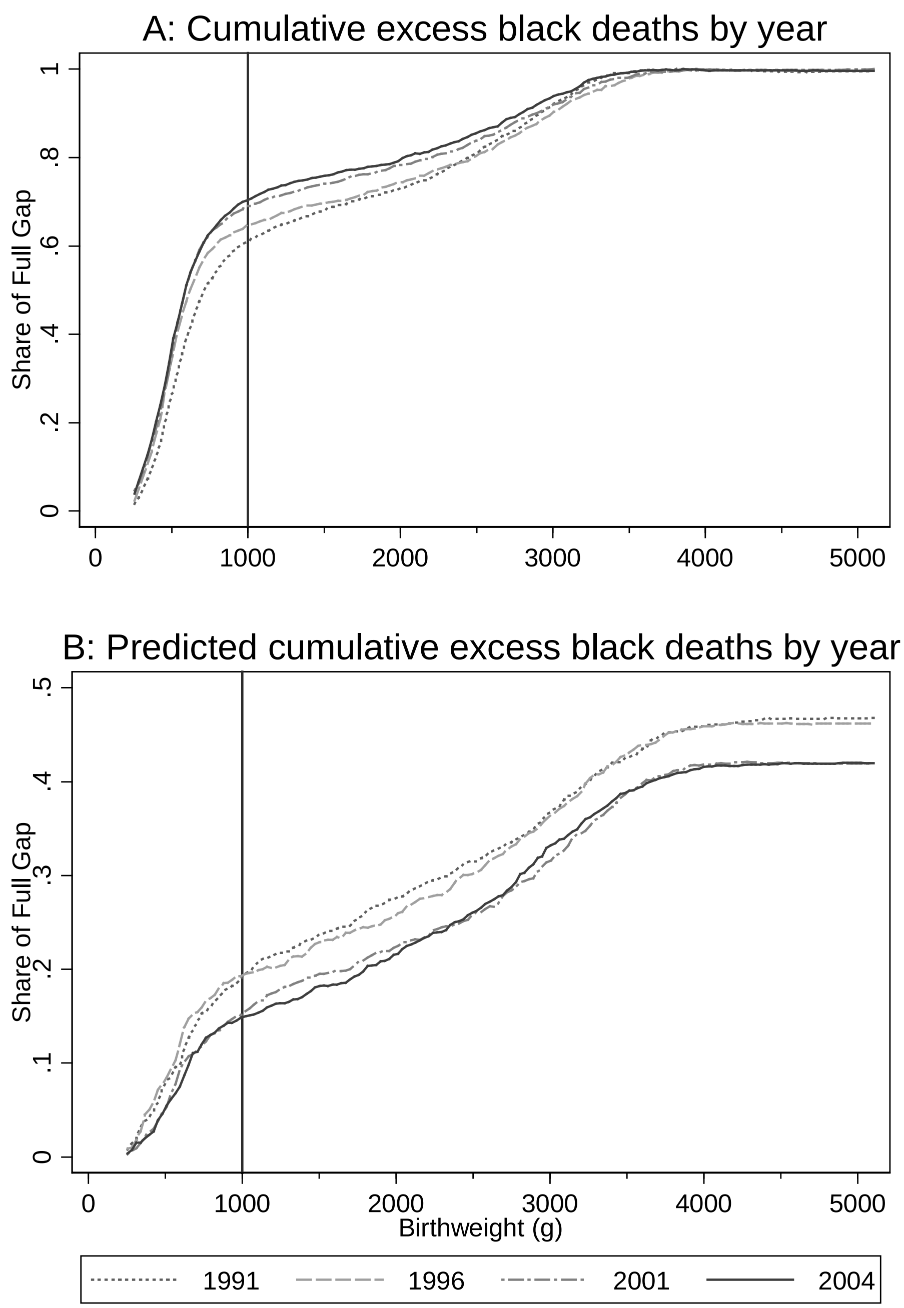


Figure 8: Actual and Predicted Cumulative Shares of Excess Black Deaths across Birth Weight, Multiple Birth Cohorts



Notes: Excess deaths are infant deaths per 1000 births minus white infant deaths per 1000 births. Contribution of background characteristics is based on the ratio of reweighted white data (w*-w). See Section 6 for more details.

Appendix Table A1. Summary Statistics for NCHS Linked Cohort Files, Select Years

	1986	1991	1996	2001	2004
Number of births	3,760,997	4,115,494	3,894,874	4,031,646	4,118,956
IMR (per 1,000)	10.09	8.63	7.09	6.74	6.74
Race					
White	.802	.789	.795	.790	.784
Black	.157	.166	.153	.150	.150
Native Amer.	.009	.009	.010	.010	.011
Asian/Pacific Is./Other	.029	.035	.043	.050	.056
Missing	.003	.001	0	0	0
Hispanic ethnicity					
Yes	.105	.152	.181	.213	.231
No	.498	.835	.805	.781	.769
Missing	.397	.013	.015	.006	.008
Maternal education					
<12	.155	.231	.221	.214	.212
12	.319	.363	.329	.311	.291
13-15	.155	.199	.218	.213	.219
16	.089	.116	.141	.152	.159
17+	.041	.059	.077	.096	.105
Missing	.241	.032	.014	.014	.013
Maternal age					
<20	.126	.129	.129	.113	.103
20-24	.293	.265	.243	.254	.251
25-29	.319	.297	.275	.263	.269
30-34	.192	.215	.231	.234	.235
35-39	.061	.081	.103	.112	.116
>39	.008	.013	.019	.024	.027
Married*	.766	.705	.676	.665	.643
First trimester prenatal care					
Yes	.742	.746	.797	.814	.797
No	.235	.233	.177	.163	.174
Missing	.022	.021	.026	.024	.028
Previous loss					
Yes	.207	.245	.249	.241	.239
No	.787	.749	.742	.755	.755
Missing	.006	.006	.008	.004	.006
Male	.512	.511	.511	.511	.512
Plural birth	.022	.024	.027	.032	.034
Live birth order					
First	.413	.405	.408	.396	.397
Second	.328	.320	.322	.325	.321
Third	.157	.163	.161	.168	.169

Fourth	.058	.064	.062	.065	.067
Fifth or later	.038	.043	.042	.042	.043
Missing	.005	.005	.006	.003	.005
Populous birth county	.612	.634	.653	.650	.680
Region					
Northeast	.190	.188	.179	.170	.165
Midwest	.236	.227	.223	.219	.216
South	.346	.340	.353	.368	.372
West	.227	.245	.244	.242	.247
Smoked cigarettes					
Yes	--	.129	.108	.103	.085
No	--	.601	.684	.759	.749
Missing	--	.270	.208	.137	.166
Drank alcohol					
Yes	--	.022	.012	.007	.006
No	--	.726	.834	.854	.827
Missing	--	.252	.154	.139	.167
Birth weight (g)					
Mean	3348	3335	3325	3305	3281
Median	3374	3374	3372	3345	3317
Missing	.001	.001	.001	<.001	<.001
Gestational age (wks)					
Mean	39.3	39.1	38.9	38.7	38.6
Median	40	39	39	39	39
Missing	.041	.011	.010	.010	.010

Notes: This table presents tabulations from the underlying linked birth certificate/death certificate cohort files. *NCHS-provided imputations are used for the marriage indicator.

Appendix Table A2. Results from Logit Models of Infant Death

	2001 Full Sample	2001 White Sample	2001 Black Sample
Black indicator	4.65	--	--
	(0.14)		
Maternal education (<12 excluded)			
12	-1.85	-2.01	-1.70
	(0.17)	(0.18)	(0.40)
13-15	-2.78	-3.06	-2.27
	(0.19)	(0.20)	(0.48)
16	-3.88	-3.99	-3.48
	(0.21)	(0.21)	(0.69)
17	-4.08	-4.05	-4.89
	(0.23)	(0.23)	(0.85)
Maternal age (<20 excluded)			
20-24	-0.71	-1.00	-0.19
	(0.19)	(0.21)	(0.48)
25-29	-1.04	-1.54	0.29
	(0.22)	(0.23)	(0.57)
30-34	-1.51	-2.07	0.45
	(0.23)	(0.25)	(0.64)
35-39	-1.51	-1.59	1.12
	(0.23)	(0.27)	(0.75)
>39	-0.21	-0.62	1.69
	(0.38)	(0.38)	(1.21)
Married	-1.79	-1.92	-1.19
	(0.13)	(0.14)	(0.37)
First trimester prenatal care	-0.79	-1.05	-0.46
	(0.15)	(0.16)	(0.35)
Previous loss	1.76	1.07	4.37
	(0.11)	(0.11)	(0.34)
Male	1.56	1.23	2.88
	(0.10)	(0.10)	(0.29)
Plural birth	25.60	21.63	42.35
	(0.26)	(0.26)	(0.81)
Live birth order (1^{st} excluded)			
2^{nd}	-0.82	-0.41	-2.67
	(0.12)	(0.12)	(0.38)
3^{rd}	-0.50	-0.02	-2.67
	(0.15)	(0.15)	(0.46)
4^{th}	0.04	0.81	-3.11
	(0.22)	(0.23)	(0.61)
5^{th} – 9^{th}	1.73	1.83	-0.24
	(0.28)	(0.30)	(0.69)

Populous birth county	1.71	1.68	2.32
	(0.11)	(0.11)	(0.39)
N	2,827,371	2,265,332	562,039

Notes: We report marginal effects and their standard errors in the table, computed as a unit change for each variable at the sample mean of the other variables.

Not for Publication

Unpublished Appendix

The main text of the paper references several results withheld for brevity and readability. We present those results here.

Table UA1 includes predicted gaps for the three-way decomposition of (A1), analogous to those presented in Table 3 of the paper based on (4). As is readily apparent, the results are very similar across both decompositions.

Table UA2 presents a number of sensitivity analyses for estimating the share of the overall IMR gap that can be predicted by the background characteristics. The results in the main text, given in the top row of Table 3, are repeated in Row A of Table UA2 as the “Base model”. Row B shows the results that are obtained using a logit-based approach. Specifically, we estimated a logit model of mortality as a function of background characteristics among whites and used the estimates to construct a predicted probability of death for each infant in the black population. This resulting predicted IMR is comparable to IMR_{w*}, and an analogous procedure can be used to construct a predicted IMR that is comparable to IMR_{b*}. For both gaps, the DFL-based results using reweighted white data are very similar to the logit results using white coefficients, and the DFL-based results using reweighted black data are very similar to the logit results using black coefficients.

Row C considers the effects of excluding Hispanics and those with missing ethnicity on our main DFL results. Including Hispanics reduces the total black-white IMR gap slightly, from 7.03 to 6.94. This small change is not surprising because infant mortality among Hispanics is very similar to that of non-Hispanic whites, and over 98 percent of the Hispanics are classified as white. The predicted share based on reweighting whites decreases substantially, from .415 to

.311, because the distribution of socioeconomic characteristics among Hispanics is more similar to blacks than to whites, implying that there is less scope for observables to predict outcome differences. Interestingly, the predicted share slightly increases based on reweighting blacks.

Row D uses only first births. We chose to focus on all births in the main text because our primary interest lies in understanding the overall IMR gap. The total gap to be predicted is smaller than in the baseline sample (6.30 versus 7.03), suggesting that differences in fertility preferences may contribute to the unadjusted gap. The fraction that can be predicted is also somewhat smaller (.396 versus .415 based on reweighted white data and .194 versus .261 based on reweighted black data). To the extent that focusing on first births allows us to better abstract from fertility preferences, these results suggest that the contribution of background characteristics to the racial IMR is even smaller than the main results above imply.

Row E explores the role of missing data. Unlike in the base model, we include observations with missing values for education, prenatal care, live birth order and previous loss and include a missing data indicator for each. The three percent increase in sample size increases the IMR gap to 7.56, and it increases the predictive power of background characteristics modestly, indicating data are missing differentially and systematically across race.

Row F and its associated rows explore the role of smoking and drinking. We did not include these variables in our baseline specification for three reasons: (1) smoking and drinking are missing much more often than other variables, (2) mothers may underreport alcohol and tobacco usage during pregnancy in order to avoid revealing stigmatized behavior, and (3) alcohol and tobacco usage during pregnancy are not necessarily predetermined to information on fetal fitness. We show three different sets of results. The first is identical to the base model but excludes California, which does not collect information on alcohol or tobacco usage. These

results differ little from the baseline results in Row A. The second set of results includes information on smoking and drinking and missing data indicators so that the sample size remains constant compared to the first set. The predictive power of background characteristics declines by about 20 percent for both sets of reweighted results because whites report higher smoking and drinking rates than blacks, which would lead whites to have higher IMRs than blacks. The third set of results excludes the observations with missing smoking and drinking information. These are similar to the second set of results, demonstrating that the decline in predictive power does not merely stem differential patterns in missing information on smoking and drinking. Overall, to the extent that smoking and drinking are appropriate background characteristics, even less of the overall white-black IMR gap is predictable than in the base model.

In Row G we exclude marital status. As discussed above in Section 4, this variable might not be predetermined to information on fetal fitness and would therefore be an inappropriate background characteristic. Compared to the base model, the predictive power falls for both sets of reweighted results, but the drop is much greater for the reweighted white results (from .415 to .310 based on reweighted white data and from .261 to .241 based on reweighted black data).

In Figure UA1, we compare our estimates of the role of the individual background characteristics in predicting the overall gap to results based on the group-specific logit models. Specifically, we adopt Fairlie's (2003) method for assessing the role of individual coefficients to outcome differences in logit models.[23] There are advantages to a logit-based approach: it allows for parsing out the partial effects of regressors that are correlated, while the proposed DFL method does not. Despite this difference, most of the results for the two methods are similar.

[23] Fairlie, R.W. (2005) "An Extension of the Blinder-Oaxaca Decomposition Technique to Logit and Probit Models," Journal of Economic and Social Measurement, 30(4): 305-16. The paper proposes a method that isolates the effect of one regressor in a logit framework that directly takes into account that the entire distribution of a regressor matters and that the effects of regressors are nonlinear.

For example, educational attainment and marital status have the strongest effects; gender, birth order, and plurality have little effect; and the effect of age differs substantially between the black results and white results. Perhaps the biggest difference between the two sets of results is related to the geographic variables. The logit results suggest that these variables have somewhat larger predictive power in both cases than does the DFL method. On one hand, the logit results indicate that the role that geography plays is similar to that of education and marriage and should not be dismissed. On the other hand, the overall predictive power of geography is still small, implying that the black-white disparity is *not* primarily a result of blacks living in states where infant mortality is high for both races.

Figure UA2 repeats the analysis presented in Figure 8 of the paper, but instead classifies births by gestational age.

Table UA1 – Three-way Temporal Decompositions, Equation 4 vs. Equation A1

	Actual, b-w		Predicted, w*-w		Predicted, b-b*	
	Level	Fraction	Level	Fraction	Level	Fraction
Full Gap	7.03	1	2.92	.415	1.83	.261
Birth weight, equation (4)						
Fitness component	6.09	.867	1.58	.225	0.62	.088
Neonatal mortality component	-0.24	-.034	0.23	.032	0.04	.006
Post-neonatal mortality comp.	1.17	.167	1.11	.158	1.18	.168
Birth weight, equation (A1)						
Fitness component	6.69	.953	1.47	.210	0.60	.085
Neonatal mortality component	-1.05	-.150	0.24	.035	0.02	.003
Post-neonatal mortality comp.	1.39	.197	1.20	.171	1.22	.173
Gestational age, equation (4)						
Fitness component	5.61	.798	1.52	.216	1.10	.157
Neonatal mortality component	-0.03	-.004	0.22	.032	-0.42	-.059
Post-neonatal mortality comp.	1.44	.205	1.18	.167	1.15	.163
Gestational age, equation (A1)						
Fitness component	6.25	.890	1.45	.207	1.23	.175
Neonatal mortality component	-0.91	-.130	0.21	.030	-0.55	-.079
Post-neonatal mortality comp.	1.68	.239	1.25	.178	1.15	.164

Table UA2. Sensitivity Analysis for Amount Predicted, 2001 Birth Cohort

	N	Actual (b – w)	Predicted (w* – w)	Predicted (b – b*)
A: Base model	2,827,371	7.03	.415	.261
B: Base model sample, logit results	2,827,371	7.03	.405 (.018)	.267 (.033)
C: Including Hispanics and missing ethnicity	3,650,266	6.94	.311	.287
D: First births only	1,136,127	6.30	.396	.194
E: Role of missing data	2,917,219	7.56	.428	.298
F: Role of smoking and drinking				
Base model without CA	2,631,677	7.08	.422	.279
Base model without CA, but including smoking, drinking, and indicators for missing values	2,631,677	7.08	.347	.222
Base model without CA and observations missing smoking and drinking	2,612,532	7.07	.345	.231
G: Dropping marriage variable	2,827,371	7.03	.310	.241

Note: All results except for Row B are based on DFL methods.

Figure UA1: Contribution of Background Characteristics to IMR, 2001 Birth Cohort

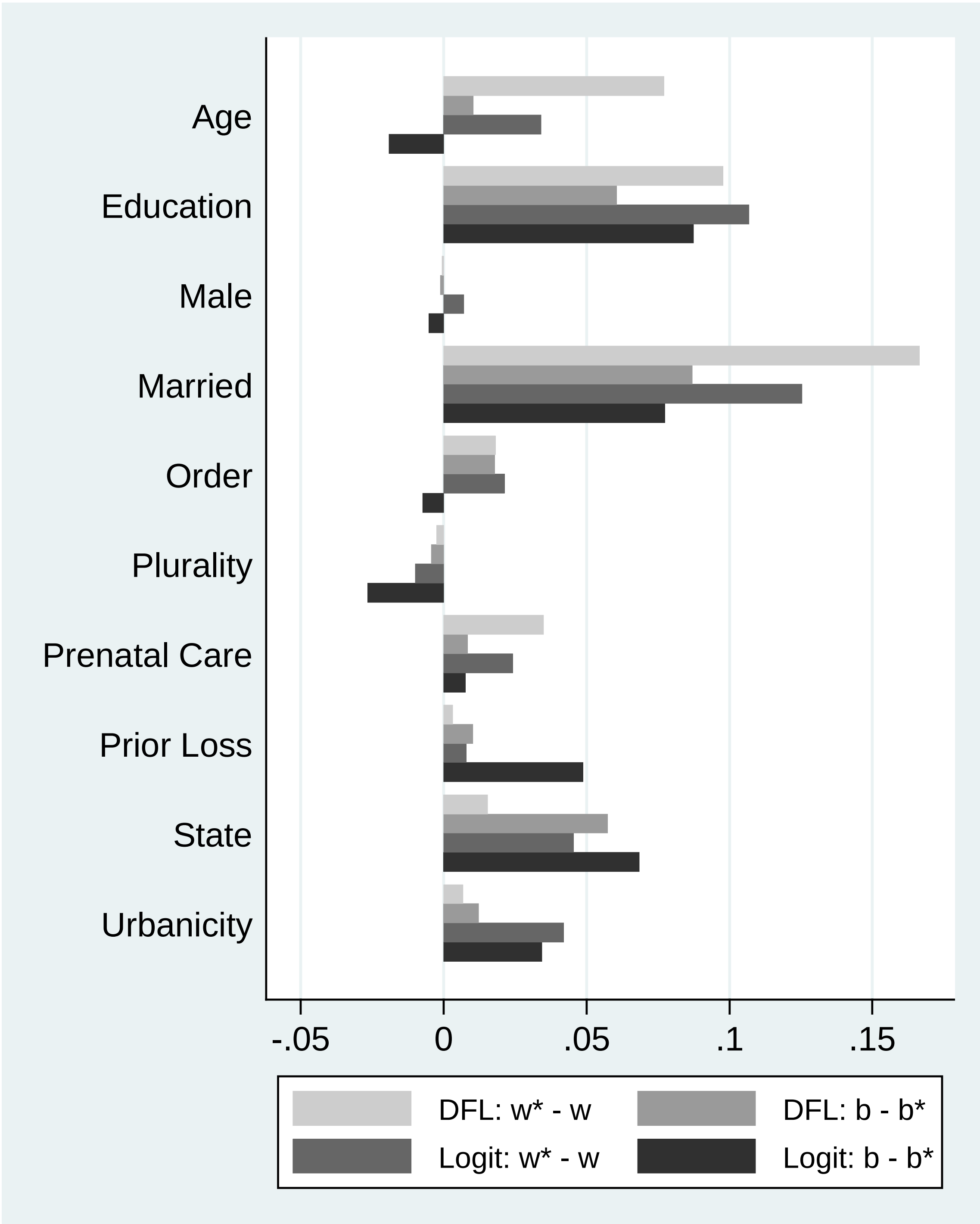


Figure UA2: Actual and Predicted Cumulative Shares of Excess Black Deaths across Gestational Age, Multiple Birth Cohorts