

Can We Explain Black-White Disparities in Infant Mortality?

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Abstract. The U.S. infant mortality rate (IMR) improved substantially between 1985 and 2001, falling 35 percent from 10.4 to 6.8 per 1,000 live births. Despite these improvements, large racial disparities persist: in 2001, the IMR was 13.2 for blacks compared with 5.6 for whites. Although it is natural to suspect that the black-white IMR gap arises from socioeconomic differences, such an explanation seems at odds with the fact that the IMR for another socioeconomically disadvantaged group, U.S. Hispanics, was 5.4 in 2001, lower than that of whites. In this paper, we systematically examine the differences in IMRs between blacks and whites, assessing when these differences arise and their potential explanations. Specifically, we consider differences in the birthweight distribution, mortality over the first 28 days, mortality over the remaining part of the first year, the correlates of each of these underlying IMR components, and infant death reporting. The main contributions of this paper are three-fold: we provide a transparent and systematic treatment of the underlying components of infant mortality and their correlates, we pay specific attention to how these components fit together, and we present similar results for other racial/ethnic groups to place the black-white gaps in perspective.

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1. Introduction

The infant mortality rate (IMR) is a commonly cited indicator of a population's health. Between 1985 and 2001, the period examined in this paper, the U.S. IMR fell from 10.4 to 6.8 per 1,000 live births. Despite these improvements, the U.S.'s overall IMR places it 43rd among the countries and territories of the world in 2008.¹ This overall assessment, however, masks tremendous racial differences. In 2001, the IMR was 13.2 for blacks compared with 5.6 for whites, a gap that in proportional terms has not closed over time. Considered separately, blacks would rank 82nd in the world IMR comparisons, just below Mauritius, Brunei, and the United Arab Emirates.² Similar comparisons are cited in *Healthy People 2010*, the official public health objectives for the United States, in which reductions in IMR are a stated goal.³

Given the stated public health goal of reducing overall IMR and its importance as a measure of population health, reducing the exceptionally high rate for blacks is an obvious target. To make progress, it is important to understand why the large disparity between black and white IMR exists. A natural hypothesis is that the IMR disparities arise from well-documented socio-economic disparities between blacks and whites. However, this explanation must confront the striking fact that the infant mortality rate for another socioeconomically disadvantaged group, U.S. Hispanics, was 5.4 in 2001, a rate lower than of whites.

In this paper, we systematically examine the differences in IMR between blacks and whites, describing the differences that exist and assessing their potential explanations. Specifically, we

¹ The CIA's *World Factbook*, <https://www.cia.gov/library/publications/the-world-factbook/rankorder/2091rank.html> (accessed May 15, 2008). The list includes both countries and territories.

² Comparing the 2001 IMR for US blacks to the world list for 2008 likely overstates the ranking for blacks given the general secular improvements in infant mortality worldwide.

³ According to the website, *Healthy People 2010* is "a statement of national health objectives designed to identify the most significant preventable threats to health and to establish national goals to reduce these threats." See <http://www.healthypeople.gov/>.

consider differences in fetal deaths, the birthweight distribution, mortality over the first 28 days, and mortality over the remaining part of the first year, and the correlates of each of these underlying pieces. Although previous research has been devoted to various pieces of the differences, one of the primary contributions of this paper is to pay careful attention to how these underlying pieces fit together. Moreover, even though our focus is on the black-white gap, we present some results for other racial/ethnic differences to place the black-white gaps in perspective.

Our findings are three-fold. First, in setting out the basic facts, we show that about 90 percent of the black-white IMR gap is due to a less favorable birthweight distribution for blacks. Our results suggest that the role of birthweight differences is even greater among neonatal infant deaths. Second, we show that the standard set of observable characteristics can explain a small part of the difference in birthweight distributions, and thus, can explain a small part of the racial IMR gap. Considering several different methods, we find observable characteristics can explain about one third of the racial IMR gap. Third, in part motivated by the unfavorable birthweight distribution of blacks at the very lowest birthweights, we examine whether differences in reporting infant deaths as opposed to fetal deaths can explain any of the gap. Our very preliminary estimates suggest there may be a role for differential reporting.

2. Literature Review

Our research goal is to understand the differences in the IMR across race/ethnicity. Given this broad goal, the relevant literature is vast, with important contributions from many disciplines. We discuss several of the most important strands of the relevant literature here, and then relate some of our key findings to specific studies throughout the rest of the paper. However, a systematic literature review is beyond the scope of this paper.⁴

⁴ For papers that have undertaken systematic literature reviews, see Paneth (1995), Lillie-Blanton et al. (1996), and Wise (2003).

A multitude of studies have established the basic black-white differences in IMR and how it has changed over time. As a recent example, Alexander, Wingate, et al. (2008) report that a live birth to a black mother was 2.04 times more likely to die than a live birth to a white mother in 1980, whereas this ratio increased to 2.46 by 2000. Many of these papers further decompose the extent to which measured IMR differences, both across races and over time, are due to differences in the birthweight distribution and the mortality rate at each point in the birthweight distribution. Three consistent findings emerge from these decompositions. First, the recent declines in IMR are driven by changes in the infant mortality rate conditional on birthweight, not changes in the birthweight distribution (e.g., Alexander, Wingate, et al. 2008). Second, the differences in IMR across race are largely due to differences in the birthweight distribution and are concentrated among causes of death that tend to occur soon after birth, such as congenital malformations and perinatal conditions (e.g., Eberstein, Nam, and Hummer 1990). Third, at very low birthweights, there remains a slight black advantage in survival, although this advantage was much larger a couple of decades ago (Frisbie, Song, et al. 2004). Each of these facts is useful in isolating what black-white differences exist and need to be explained, but in and of themselves, they do not shed light on why the racial disparities exist (Wise 2003).

Numerous studies have directly examined the correlates for IMR disparities between whites and nonwhites. These studies have generally found that, while covariates such as maternal characteristics (e.g., age and education), prenatal behavior (e.g., obtaining care, smoking and drinking), and birth characteristics (e.g., plurality) are important predictors of infant mortality, they can explain only some of the IMR racial gap. For example, using 1989-91 black-white IMR vital statistics data, Hummer, Biegler, et al. (1999) find that such characteristics can explain just over half of the gap as measured by the odds ratios. Chay and Greenstone (2000) find a dramatic narrowing of the gap in the rural south between 1960 and 1970, due at least in part to declining racial disparities in socioeconomic status and access to medical care during this period, but that a large gap remains even conditional on access to medical care. Collins and Thomasson (2004) use state-level panel data over the 1920-1970 period and

a regression approach to explain the IMR differences. They find that, whereas a set of observables that reflect income, education, urbanicity, and physicians per capita can account for more than 80 percent of the IMR gap between 1920 and 1945, the same set can only explain about 33 percent of the gap by 1970. In all years, most of the explanatory power of the observables is due to variation in the income and education variables. Part of the difference in the conclusion of these last two studies rests with examining absolute gaps versus relative gaps, an issue noted by others (Frisbie, Song, et al. 2004). Miller (2003) also examines the role of birthweight and other observable factors using 1989 data and finds that 80 percent of the relative mortality gap appears to be explained by differences in socioeconomic and prenatal care covariates, an amount that is much greater than that found in other studies.

A growing literature has examined the IMR gap for whites versus Hispanics, consistently finding that Hispanics have either a lower or similar infant mortality rate to whites. Frisbie and Song (2003) analyze mortality and indicators for short gestational age and low birth weight, differentiating Hispanics by country of origin and birthplace of the mother. They find that although births to Hispanic mothers are uniformly more likely to be of short gestation and low birthweight than births to non-Hispanic white mothers, most Hispanic groups have lower IMR than whites, with particularly large advantages for foreign-born Mexican mothers. Hummer, Powers, et al. (2007) find a similar pattern, with the IMR among Mexican immigrants being 14 percent lower than that of native non-Hispanic whites, and examine whether the relative advantage of Hispanics can be explained by selective out-migration. They reject this explanation on the grounds that much of the advantage develops within one day of birth, before much out-migration could occur, and instead point to the relative advantage as being additional evidence of the general finding that Hispanics are relatively advantaged along various dimensions of health.

Although the literature on infant mortality has largely ignored fetal mortality, a separate literature has examined several relevant aspects of fetal mortality. An important set of studies have examined the data quality of the fetal death records (Gaudino, Blackmore-Prince, et al. 1998; Lydon-Rochelle, Cardenas, et al. 2005). Concerns about data quality exist because states began requiring reporting of fetal deaths relatively recently, and these requirements are based in part on weeks of gestation, a quantity that is not always accurately known. The studies generally find that reporting is quite accurate by 28 weeks gestation. Other work has established that black-white gaps in fetal death rates are roughly as large as the disparity in infant death rates. Vintzileos, Ananth, et al. (2002) examine the role of prenatal care in explaining black-white fetal death disparities. Although they do not quantify the effects of prenatal care on the racial fetal death gap, they find that prenatal care significantly reduces fetal deaths, with blacks receiving less prenatal care than whites.

3. Data

Our primary data source is the linked birth/infant death data sets compiled by the National Center for Health Statistics (NCHS).⁵ These have been made publicly available for the years 1985-1991 and 1995-2002. The cohort data sets that we use include information from the birth certificates of all live births occurring in the U.S. in the relevant calendar years. Birth information is then linked to information from 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; we ignore the unmatched deaths in our analysis.

As is standard in the literature, we classify births based on information from the birth certificate about race and ethnicity of the mother. Infants are classified as Hispanic if the mother is identified as Hispanic. Non-Hispanic births are then classified into the categories white, black (African American),

⁵ See information at <http://www.cdc.gov/nchs/linked.htm>.

Asian or Pacific Islander, and American Indian (including Eskimos and Aleutian Islanders), based on information about mother's race. Ethnicity information necessary to classify a mother as Hispanic is available only beginning in 1989.

The literature commonly distinguishes between neonatal infant deaths, defined as those that occur within the first 28 days of life, and post-neonatal infant deaths, those that occur during the balance of the first year of life. In 2001, 2/3rds of infant deaths were neonatal. We will also analyze neonatal and post-neonatal mortality differences.

Separate from the birth and death records used to measure infant mortality, each state has requirements regarding the reporting of fetal deaths. These reporting requirements are generally stated in terms of gestational weeks and weight at time of delivery and vary by state of occurrence (see Appendix Table A2). The distinction between a fetal death and an infant death rests with whether the delivery is determined to have resulted in a live birth.⁶ For some of our analysis, we merge onto the birth cohort data files the fetal death records that are distributed as part of the Period Linked Perinatal Mortality File. The fetal death records contain much of the same information as the combined birth-death records we use for our infant mortality analysis, although missing data elements pose a much larger problem.

See the Appendix for more information on all of our data.

4. Setting the Stage: Racial/Ethnic Differences in IMR

In this section, we describe the racial/ethnic difference in the IMR, first laying out the basic facts, then examining the role of birthweight, and finally distinguishing between neonatal and post-neonatal mortality. The results on birthweight and mortality timing are not intended to be explanations for the racial/ethnic differences, but to be suggestive as to where explanations may lay.

⁶ The definition of a live birth used by NCHS states that a "live birth is the complete expulsion or extraction from its mother of a product of conception, irrespective of the duration of pregnancy, which, after such separation, breathes or shows any other evidence of life...." See *Vital Statistics of the United States 2001 Natality, Technical Appendix*, referenced at <http://www.cdc.gov/nchs/data/dvs/linked/Techapp01.pdf> (June 1, 2008).

4.1. The Basic Facts

Table 1 presents sample sizes and tabulations from our data for 2001, along with a comparison to NCHS published data about infant mortality rates by race/ethnicity. The 0.6 percent of births for which no information about ethnicity is available are excluded from our analyses (information about Hispanic ethnicity is less complete for some earlier years, especially prior to 1989). The IMRs by race/ethnicity in the data we use are quite similar to the published rates, though slightly lower for all groups. Much of the difference is due to the fact that we do not include an adjustment for unlinked deaths, which account for 1.1 percent of deaths in 2001. Another source of slight differences is that the published IMRs are based on period data, which calculates rates based on births and deaths occurring in the calendar year.

Table 1 also highlights the large disparity between the white and black IMRs, as well as the fact that the Hispanic rate is slightly lower than the white rate. The Asian and Pacific Islander group, which for convenience we will usually refer to as Asian, had an even lower IMR in 2001. The IMR for American Indians is relatively high, though a good deal lower than that of blacks. Because of the relatively small number of births in the American Indian group, we do not include it in further analyses.

The long term trend in infant mortality is downward for all racial/ethnic groups, but the pattern is not uniform over the time period we are considering. Figure 1 displays the trend for the groups we are considering based on cohort data sets for all years of our data.⁷ The figure shows a rather steady downward trend up to 1996, and only slight declines after 1996. Tabulations from period data files (Mathews and MacDorman, 2007) show IMRs as rather flat for all groups from 2001 through 2004.

⁷ Ethnicity is identified for 94.9 percent of births in 1989 and at least 98.5 percent in the later years. The calculations for Hispanics and the groups labeled as nonhispanic in those years exclude those for whom ethnicity is not identified. In 1985 ethnicity is not identified for 40.3 percent of births, so we do not identify Hispanics separately in that year, and we use all births by racial group. IMRs for whites, blacks and Asians are very similar in 1989 and later years whether or not only nonhispanics are included.

Infant mortality for blacks is substantially above that for other groups in every year, whereas the Asian IMR is consistently lowest. There is no evidence of convergence over time.

4.1. The Role of Birthweight

Methods. Birthweight is an important indicator of infant fitness at birth and therefore provides some insight as to when disparities begin to arise. To examine the role of birthweight differences for the IMR gaps, we decompose the IMR into two components, the infant mortality rate conditional on birthweight and the distribution of birthweight. Specifically, suppose we consider K unique and exhaustive categories of birthweight.⁸ Then, define π to be a $K \times 1$ vector of the infant mortality rate for the birthweight categories and b to be a $K \times 1$ vector of the share of births that occur in the respective birthweight categories. The infant mortality rate for a particular group j can then be written as $IMR_j = \pi_j' b_j$.

We can use this decomposition to calculate counterfactual infant mortality rates. For example, we could compute a counterfactual infant mortality rate where we apply the birthweight distribution of group 2 to the infant mortality rate conditional on birthweight for group 1, $IMR_{cf} = \pi_1' b_2$. We can use such counterfactual infant mortality rates to examine the source of differences between two groups. In particular, it is straightforward to verify that the difference in infant mortality rate between groups 2 and 1 is

$$(1a) \quad IMR_2 - IMR_1 = (\pi_2' b_2 - \pi_2' b_1) + (\pi_2' b_1 - \pi_1' b_1).$$

We interpret the first term on the right-hand side of (1a) as the amount of the IMR difference due to differential birthweight distributions and the second term to be the amount of the IMR difference

⁸ For the decompositions and graphical analysis, our approach to dividing births into birthweight categories is as follows. While the research literature and NCHS data sets usually report birthweight in grams, in the U.S. birthweight is commonly measured in pounds and ounces. A close examination of the NCHS data reveals a pronounced heaping of observations at gram values that correspond to ounce values. We therefore round gram values to the nearest ounce and use ounce values as our birthweight categories, resulting in 173 categories. For comparability with other research, we convert all birthweight reports back to grams when displaying or discussing our results.

due to differential infant mortality rates conditional on birthweight. As is typical in such decompositions, this calculation is not unique. The decomposition could have also been written as

$$(1b) \text{IMR}_2 - \text{IMR}_1 = (\pi_1' b_2 - \pi_1' b_1) + (\pi_2' b_2 - \pi_1' b_2)$$

Although the first term is still interpreted as the IMR difference due to differential birthweight distributions, this alternative calculation generally will not yield the same values as (1a). Because there is no reason to prefer the decomposition in (1a) to (1b), we will examine both decompositions.

A small fraction of births, about 0.1 percent in 2001, do not have birthweight recorded. We keep these as a separate category for the tabular counterfactual comparisons using (1a) and (1b), but exclude them from the graphical comparisons.

IMR in 2001. We begin with some graphs for 2001. Figure 2 presents four graphs related to infant mortality of whites, blacks and Hispanics. Panel A shows kernel density estimates of the race-specific distribution of birthweights. The black distribution is clearly shifted to the left as compared to that of whites and Hispanics and is noticeably thicker in the left tail. Panel B shows the smoothed IMR conditional on birthweight (IMR|BW) curves for the three groups. Panel B suggests that the only (and small) difference between the IMR|BW curves by race is in the extreme left tail. However, given the large number of births in the middle of the distribution (see Panel A), even small differences in IMR|BW in the middle could matter. The next two graphs present weighted versions of Panel B that make this point clear.

Panel C graphs infant deaths per 10,000 live births by race/ethnicity and is intended to reflect the contribution of various birthweight deaths to the overall IMR. This graph is constructed so that the height of the curve equals the number of deaths per 10,000 live births for the birthweight. For example, the black peak of roughly 0.18 near 500 grams means that, for every 10,000 live black babies born, 0.18 black babies were born at 500 grams and died before their first birthday. Integrating over the

distribution of birthweights (i.e., summing the total area under the curve) gives the overall black IMR of 132.1 per 10,000 live births.

Panel C shows clearly that most of the excess infant deaths for blacks are at the low end of the birthweight distribution, but the black curve is also distinctly above the others over most of the range of birthweights. Comparing Panels B and C of the figure also illustrates that the distribution of deaths can be quite flat in a range where the IMR|BW is sharply decreasing (e.g., around 1,250 grams for whites and Hispanics), and the Panel C curves even increase at higher birth rates. These phenomena reflect the much larger numbers of births at higher birthweights.

Although Panel C is useful to highlight the important birthweight ranges of IMR differences, Panel C combines the effects of a group's birthweight distribution and its distribution of IMR|BW. For example, the much higher peak of the black curve at 500 grams as compared to whites and Hispanics must be due to some combination of two factors: 1) proportionally more black babies are born around 500 grams, and 2) among babies born at these low weights, the probability of dying is much higher for blacks than for whites and Hispanics.

Panel D is similar to Panel C, except that the group-specific IMR|BW relationships are combined with a standardized distribution of birthweights: the simple average of the distributions of the three groups.⁹ The graph depicts how the distribution of infant deaths would compare across groups if each kept its own conditional IMRs but shared a common birthweight distribution. This graph isolates the role of IMR|BW differences in explaining differences in mortality while still focusing our attention on the range of birthweights that are important to the overall IMR. The differences with Panel C are striking. The black curve is now essentially identical to the others in the left tail, suggesting that all of the excess infant deaths for infants under 1,000 grams are accounted for by larger shares of births in that range of birthweight. On the other hand, black IMR|BW is higher than for the other groups

⁹ The curves change very little if we instead use the overall distribution of birthweight (which is a weighted average across races), rather than the simple average across races.

through much of the normal birthweight range. Although mortality rates in this range are quite low, the differences across races become quantitatively important because far more births occur in this range than at low birthweights.

The relative contributions of birthweight distributions and conditional mortality rates to IMR disparities across groups can be quantified using the decompositions in (1a) and (1b). In Table 2, each cell represents an actual or counterfactual IMR, calculated by combining the IMR|BW for one group with the birthweight distribution of another group ($IMR = \pi_i 'b_j$). The columns of Table 2 indicate which conditional mortality rate (π) is used, while the rows indicate which birthweight distribution (b) is used. Thus, the diagonal elements (shaded) are the actual IMRs and the off-diagonals are counterfactual IMRs.

Table 2 confirms what we saw in Panels A through D of Figure 2: the IMR gap between blacks and whites overwhelmingly is due to an unfavorable distribution of birthweight for blacks. Specifically, the actual white IMR is 5.64, whereas the actual black IMR is 13.21 (the diagonal elements); thus, the black/white IMR gap is 7.57 (=13.21–5.64). If blacks were to keep their own IMR|BW (looking down the “Black” column) but achieve the white birthweight distribution (looking across the “White” row), then the black IMR would fall by 6.58 (from 13.21 to 6.63). Based on this comparison, we would infer that differences in the birthweight distribution account for 87 percent of the black/white gap ($6.58/7.57 \cdot 100$) and differences in IMR|BW account for 13 percent of the black/white gap. As mentioned previously, this decomposition could have been done in another way. In particular, if blacks were to keep their own birthweight distribution (looking across the “Black” row) but achieve the white IMR|BW (looking down the “White” column), then black IMR would fall by 0.37 (from 13.21 to 12.84). In this case, differences in the IMR|BW account for 5 percent of the black/white gap ($0.37/7.57 \cdot 100$), and differences in the birthweight distribution would account for 95 percent of the gap. Because there is no reason to prefer one method over the other, we conclude that differences in the birthweight distribution account for roughly 90 percent of the black/white IMR gap.

Another lesson that can be drawn from Table 2 is that the relatively small disparity between Hispanic and white IMRs (which favors Hispanics) is due primarily to differences in $IMR|BW$. The IMR numbers change little as we swap birthweight distributions across rows 1 and 3 but more substantially as we interchange the distributions of $IMR|BW$ across the columns. The favorable Asian American IMR relative to whites is also primarily accounted for by lower conditional mortalities, although it is clear from Table 2 (by comparing the white and Asian rows) that Asians also have a more favorable distribution of birthweights.

Changes over Time. Figure 3 is the direct analogue to Panel D of Figure 2, but for 1989. The white and Hispanic distributions of deaths look quite similar to each other, as they do in Figure 1. What is most notable about Figure 3 is that, with a standardized birthweight distribution, black deaths would be noticeably lower than those of the other groups over the range of birthweights from about 750-2,000 grams. This survival advantage of blacks at low birthweights was almost gone by 2001, a phenomenon that has been noted by others (e.g., Alexander et al., 2008).

Figure 4 focuses on whites and blacks only (not excluding Hispanics) and puts 1985 and 2001 on the same graph. The 1985 curves show what the distribution of deaths would be for each group if they shared the average of the 1985 birthweight distributions, but each had its own conditional mortalities. The 2001 curves are created similarly. This figure illustrates several findings: (a) the general decline in infant deaths for both groups across the birthweight distribution, as the 2001 curves lie almost entirely below the 1985 curves for the respective groups; (b) a survival advantage for blacks at low birthweights in 1985 that is nearly gone by 2001; and (c) for both groups, a distribution of deaths that has shifted over time to lower birthweights. In the normal birthweight range, the conditional mortality rates have fallen for both groups, but they appear to have stayed in about the same proportion.

The distribution of birthweights has shifted over time in a generally unfavorable direction, especially for whites. Thus declining mortality rates have been the result of declining conditional mortality rates, which more than offset shifts in the distribution toward lower birthweights. Another

striking feature of Figure 4 is that the peak that occurs around 500 grams is roughly the same height for both groups in both years. This happens because (a) the year-specific conditional mortality rates did not vary greatly across race, and (b) declining conditional mortality rates at this birthweight were just offset by increases in the share of births occurring there.

Table 3 provides the specific decompositions that correspond to the changes over time. In the first two rows, we show the actual IMRs by group for 1989 and counterfactual IMRs for what would have occurred had each group maintained its 1989 conditional mortalities but experienced its 2001 birthweight distribution. For each group the 2001 birthweight distribution would have led to a higher IMR. The largest effect, proportionately and absolutely, is for whites. In the remaining two rows, we perform a similar counterfactual exercise, starting with the 2001 IMRs and asking what they would have been with the 1989 distributions. Here the results are more mixed. For whites and Asians, the 1989 distributions again look more favorable, but for black and Hispanics the 2001 distributions give slightly lower IMRs.

4.3. The Role of Timing: Neonatal versus Post-neonatal Mortality

Conventionally, neonatal and post-neonatal mortality rates (NMR and PMR) are both defined as deaths per 1,000 live births in the relevant period: the first 28 days for NMR and the rest of the first year for PMR. In particular, letting the superscript on π denote the mortality rate in the relevant time period, then it is straightforward to see that

$$\begin{aligned}
 (2) \quad \text{IMR}_j &= \pi_j' b_j \\
 &= (\pi_j^n + \pi_j^p)' b_j \\
 &= \pi_j^n' b_j + \pi_j^p' b_j \\
 &= \text{NMR}_j + \text{PMR}_j.
 \end{aligned}$$

Thus, a group's IMR is simply the sum of its NMR and PMR, and these mortality rates can be decomposed just as the IMR was decomposed.

Figure 5 makes use of these decompositions and repeats Panels C and D of Figure 2 separately for neonatal and post-neonatal deaths. Panels A and B of Figure 5 are for neonatal deaths. In Panel A we see that black neonatal deaths are much more frequent in the left tail of birthweight than for the other groups, as was true for all deaths. We also see that neonatal deaths are heavily concentrated among births under 1,000 grams for all groups. When the birthweight distribution is standardized across groups in Panel B, the distributions of neonatal deaths are almost indistinguishable across groups. This strongly suggests that higher neonatal deaths for blacks are accounted for by a less favorable birthweight distribution.

The post-neonatal graphs of deaths (for deaths occurring in the remainder of the first year), Panels C and D, look very different than the neonatal graphs. They are bimodal, with the highest peaks for whites and Hispanics in the normal range where most births occur. Note the scaling of the vertical axis relative to Panels A and B—among blacks, the left-tail mode in Panel C is only about 1/7 as high as for neonatal deaths. With the birthweight distribution standardized in Panel D, we can see that while the white and Hispanic distributions of deaths look largely similar, black deaths are notably higher through most of the birthweight distribution.

One drawback of this conventional definition of the PMR is that it does not clearly separate between neonatal and post-neonatal mortality. In particular, any live birth that results in a death during the neonatal period cannot also result in a death in the post-neonatal period. Thus, a group that has a high neonatal mortality rate could end up with low post-neonatal mortality rate, even if the relative risk of death during the post-neonatal period is also high. To circumvent this problem, we also define a PMR that is conditional on one surviving into the post-neonatal period, denoted PMR^c ,

$$(3) \quad \begin{aligned} PMR_j^c &= \pi_j^{p*} ' b_j^p \\ &= \pi_j^{p*} ' [(ones - \pi_j^n) \# b_j], \end{aligned}$$

where *ones* is a vector of 1's and # represents element-by-element multiplication.

Table 4 presents a series of counterfactuals that makes use of the decompositions in (2) and (3). Panels A and B of Table 4 are identical to Table 2, but instead decompose NMR (Panel A) and the PMR (Panel B) into its birthweight component and the mortality conditional on birthweight component. Panel C decomposes the PMR^c into a birthweight component and the mortality conditional on birthweight component, both only including those births that survive into the post-neonatal period.

First comparing the actual NMR (Panel A) and PMR (Panel B) to IMR (see Table 2) for each group, we see that about 2/3rds of the overall IMR emerges within the first 28 days. Looking at whites, for example, the actual NMR is 3.73, whereas the actual IMR is 5.64. In addition, the two black-white mortality ratios are nearly identical at about 230 percent.

Focusing on the NMR in Panel A, we see that the other groups would actually have higher neonatal mortality than blacks if they had the black birthweight distribution (comparing across the columns). A small black NMR|BW advantage among low birthweight infants accounts for this difference. Within each of the columns, we see that the large increase in the NMR arises when the black birthweight distribution is used. Thus the differences in birthweight distributions account for more than 100 percent of the black neonatal mortality disparities with other groups.¹⁰

The role of PMR|BW is quite different than the role of NMR|BW. Looking across the black row in Panel B, we see that post-neonatal mortality would be much lower with any of the other sets of conditional mortalities. At the same time, looking down the black column, we see that black post-neonatal mortality would also be lower with any of the other birthweight distributions. Comparing blacks to whites, for example, of the 2.42 (4.29-1.87) excess post-neonatal deaths per 1,000 births, 1.21 (3.08-1.87) can be accounted for by conditional mortality differences if the white birthweight shares are used and 1.49 (4.29-2.80) if the black birthweight shares are used.

¹⁰ When comparing blacks to Asians in neonatal mortality (not shown in the table), whether birthweight distribution differences explain more than 100 percent of the disparity depends on which form of the decomposition is used. In either case, birthweight distribution differences are far more important than conditional mortality differences.

The decomposition for PMR^c in Panel C does not look appreciably different than the decomposition for PMR . The reason for this similarity is that the $NMR|BW$ is so similar between the three groups. The slight difference that does exist is as expected. The slight black advantage in $NMR|BW$ tends to understate the relative role of the birthweight distribution during the post-neonatal period.

Despite the worsening of the $MR|BW$ for blacks when comparing the $PMR|BW$ to the $NMR|BW$, the bulk of the difference between blacks and whites still rests with the differential birthweight distribution. This conclusion can be inferred by the relative stability of the black-white mortality gaps discussed above, despite the fact that conditional mortality rates are relatively worse for blacks in the post-neonatal period: this worsening does not cause an increase of the relative black-white gap over the course of the year.

4.4. Summary

In this section, we set out to describe the basic facts regarding IMR differences across race, paying particular attention as to when these differences arise. We summarize these conclusions before we try to “explain” them in the next section.

- The black-white IMR gap is 7.6 per 1,000 live births in 2001, a gap that implies that black IMR is nearly 240 percent that of whites. The 2001 IMR is slightly lower for Hispanics when compared to whites.
- In relative terms, the racial/ethnic IMR gaps have remained fairly stable over time.
- In 2001, about 90 percent of the black-white IMR gap is due to differential birthweight distributions; in particular, there are far more low-birthweight black babies than there are low-birthweight white babies. There is very little difference in the birthweight distribution between whites and Hispanics. Asian Americans have a somewhat more favorable birthweight distribution.

- Differences in IMR|BW across race and ethnicity are for the most part small. In 2001, Hispanics and Asian Americans had slightly lower IMR|BW than whites overall. Blacks had higher IMR|BW than the other groups in the normal birthweight range. A black advantage in IMR|BW for those at low birthweight has eroded over time.
- If we further distinguish between neonatal and post-neonatal mortality rates (NMR and PMR), about 2/3rds of the IMR for each group arises in the first 28 days. If we consider NMR in 2001, the entire black-white difference is due to differential birthweight distributions, although blacks enjoy a slight advantage in the NMR|BW at very low birthweights. However, blacks experience a higher PMR|BW. The black-white NMR and IMR ratios are virtually identical.

5. Can Differences in Observable Characteristics Explain the Black/white Gap?

The above analyses examined racial and ethnic gaps in infant mortality, highlighting the relative roles of birthweight, infant mortality conditional on birthweight, and the timing of infant deaths. Now we examine the extent to which the IMR and its underlying components can be explained by differences in observable characteristics, such as the mother's education, marital status and state of residence. At this stage, our analysis remains a descriptive exercise in that we only try to identify the correlates of the differences.

Methods. Our methods are based on calculating counterfactual quantities, just as in section 4.1, but now we first adjust the underlying components for differences in observables. These adjustments are made through a weighting procedure developed in DiNardo, Fortin, and Lemieux (1996; hereafter DFL).

Suppose we wish to understand the source of differences in the density of an outcome variable y across races j , where $f_j(y)$ is the group-specific density function and j indexes the group (e.g., blacks or whites). For example, the outcome variable could be birthweight, gestational age or infant death.

Further, suppose that observable attributes x in a particular group are distributed as $F(x | j)$. We can then write the group-specific density function to be

$$(4) \quad f_j(y) = \int_x f(y | j, x) dF(x | j) \equiv f(y; j_{y|x}, j_x).$$

Equation (4) simply states that the group j -specific distribution of y can be expressed as a group- and attribute-specific distribution, integrated over the distribution of attributes of individuals who are in group j . In the shorthand notation of the last term in (4), $j_{y|x}$ indexes the group whose conditional distribution $f(y/x)$ is used, while j_x indexes the group whose distribution of attributes $F(x)$ is used.

Counterfactual densities can then be defined by choosing the conditional outcome distribution we observe for one group (the $j_{y|x}$) and the attribute distribution we observe for another group (the j_x).

For example, we can compute the birthweight distribution that would result if blacks retained their birthweight conditional-on-attributes distribution, but had the distribution of attributes observed for whites,

$$(5) \quad f(y; j_{y|x} = \text{black}, j_x = \text{white}) \equiv \int_x f(y | j_{y|x} = \text{black}, x) dF(x | j = \text{white}).$$

The key insight of DFL is that (5) can be computed as a weighted function of the actual density of black individuals, with weights that are simple to construct. Specifically,

$$(6) \quad f(y; j_{y|x} = \text{black}, j_x = \text{white}) = \int_x f(y | j = \text{black}, x) \psi_{B \rightarrow W}(x) dF(x | j = \text{black}),$$

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

$$(7) \quad \psi_{B \rightarrow W}(x) = \frac{dF(x | j = \text{white})}{dF(x | j = \text{black})} = \frac{\Pr(j = \text{white} | x)}{\Pr(j = \text{black} | x)} \times \frac{\Pr(j = \text{black})}{\Pr(j = \text{white})}$$

The first fraction in the last term of (6) can be estimated using a simple binary model (such as a probit or logit) of membership in a particular racial group as a function of covariates x , while the second fraction involves only the sample proportions of individuals in each group.

The appealing aspect of this method is that we can use the weights to examine flexibly many different counterfactual objects. For example, based on equations (5) through (7), it is straightforward to estimate the counterfactual density of birthweight that would result if blacks had the observable attributes of whites but retained their own mapping between observable attributes and birthweight. We estimate the counterfactual density by using the actual micro data for blacks, but applying the weights given by equation (7). Similarly, we can compute the mean birthweight for blacks that would result if blacks had the observable attributes of whites, but retained their own mapping between observable attributes and birthweight, by estimating the weighted mean birthweight of blacks as,

$$(8) \quad \overline{BW}_{cf}(j_{y|x} = black, j_x = white) = \frac{\sum_{i \in black} BW_i \psi_{B \rightarrow W}(x_i)}{\sum_{i \in black} \psi_{B \rightarrow W}(x_i)},$$

where i indexes observations in the micro data (the denominator on the right side is necessary because the full set of weights need not sum to 1).

Combining the re-weighting methods here with the counterfactual calculations in Section 4, the actual IMR for group j can be written as

$$(9) \quad IMR_j = \pi_j' b_j = \pi(j_{y|x}, j_x)' b(j_{y|x}, j_x).$$

Consider the two counterfactuals

$$(10a) \quad IMR_{cf} = \pi(j_{y|x} = black, j_x = black)' b(j_{y|x} = black, j_x = white)$$

$$(10b) \quad IMR_{cf} = \pi(j_{y|x} = black, j_x = white)' b(j_{y|x} = black, j_x = black)$$

Expression (10a), for example, represents the counterfactual IMR for black infants if they maintained their own mapping from attributes to birthweight and IMR conditional on birthweight, but their birthweight distribution was generated from the white distribution of x . Combining such counterfactuals with those already described allows us to decompose the amount that attributes explain of the black-white IMR gap into two components: one due to IMR conditional on birthweight and one due to the distribution of birthweight.

We estimate the weighting function $\psi_{B \rightarrow W}(x)$ using logit with the linked birth/infant death data, using the population of births to black and white mothers in 2001 (2.9 million total births). We flexibly control for a broad set of attributes that are available on birth certificates and that others have found to be associated with birth outcomes. Specifically, we include dummy variables for seven categories of mother's reported education levels, 51 state of delivery indicators, 9 categories of mother's age by 5-year intervals, 1 for plurality, 3 each for prenatal tobacco and alcohol consumption by the mother, one for marital status, 1 for whether the birth occurred in a metropolitan area of 250,000 or more, and 7 categories indicating the gestational month that prenatal care began (including one indicating that the mother did not undertake any prenatal care measures). Appendix Table A3 lists summary statistics for these attributes separately for blacks, whites and weighted blacks in the 2001 NCHS data.¹¹

Results. Figure 6 illustrates the effects of the DFL-based weighting on the distribution of birthweight among blacks. These are the same as the densities shown in Figure 1, Panel A, but without the curve for Hispanics and with the addition of a curve for "weighted blacks." The figure shows that this counterfactual density is much closer to the actual density of blacks than the actual density of whites, even though it is based on the white distribution of attributes. Apparently, observable differences between blacks and whites (at least those reported in birth certificate data) can explain very little of the difference in birthweight distributions between the two groups. Most importantly, although the weighting modestly shifted the mean of the black distribution, it appears to have had very little effect on the lower tail, especially the "very-low birthweight" babies under 1500 grams that contribute disproportionately to infant mortality. This graphical evidence is suggestive that differences in observable characteristics across races fail to explain much of the difference in the birthweight

¹¹ While our logit specification is quite flexible (all covariates are entered as sets of categorical variables, with in most cases the most disaggregated set of categories possible) it is not a "fully saturated" model, because we do not include interaction terms among the covariates. Therefore the weighted black distribution of attributes does not exactly match the white distribution in every dimension. However Appendix Table A3 shows that the weighted black distribution of attributes is quite close to the white distribution, even when the black distribution of attributes is very different.

distribution. Given that the overall black-white IMR gap is associated with differential birthweight distributions, this finding also suggest that differences in observable attributes cannot explain much of the black-white IMR gap.

We present visual evidence of the effect on black IMR of switching to the distribution of observable characteristics of whites in Figure 7. The top panel graphs infant deaths per 10,000 live births for blacks, whites, and weighted blacks (the “Black” and “White” curves are the same as in Panel C of Figure 2). It captures differences across groups both in birthweight distributions and in IMR conditional on birthweight. The weighted black curve lies below the unweighted black curve throughout much of the distribution, although it lies much closer to the actual curve for blacks than the actual curve for whites. This implies that attributes apparently explain very little of the dramatic difference across races in the left tail of the distribution of birthweight.

The bottom panel of Figure 7 isolates variation across the three groups in IMR conditional on birthweight by using a common distribution of birthweights for all three curves (the average of the black and white birthweight distributions). The differences across races in the left tail apparent in the top panel have disappeared, confirming that the excess infant deaths in the lower tail among blacks, both weighted and unweighted, are due entirely to greater proportion of births in these weight ranges. Differences in attributes can apparently explain a substantial amount of the between-race variation in $IMR|BW$ in the “normal” birthweight range, though, since the “weighted blacks” curve lies roughly equidistant to the curves for blacks and whites in the 2500-4000 gram range. This finding suggests that attributes are much more important in explaining variation across races in $IMR|BW$ than variation in birthweight.

Table 5 quantifies these visual findings with tabular decomposition. Each cell in the table represents an actual or counterfactual 2001 IMR. Note that the upper left four cells of the table are identical to those in Table 2, while the rightmost column and bottom row refer to a third group: blacks weighted to have the same attribute distribution as whites (so that all entries in this row and column are

counterfactual). Each column uses the conditional mortality rates for the indicated group, while the row indicates which birthweight distribution is used. As noted above, the actual infant mortality rate for whites is 5.64 per thousand live births in 2001, while it is 13.21 for blacks. After weighting, the implied counterfactual black IMR is 11.30, a reduction of 1.91 or roughly 25 percent ($1.91 / (13.21 - 5.64)$) of the racial IMR gap can be explained by differences in attributes.

The advantage of using DFL-based methods, however, lies in the fact that it is straightforward to decompose this counterfactual into a component due to birthweight and another due to IMR conditional on birthweight. In other words, we can decompose this 1.91 reduction in IMR into components due to a shift in the birthweight distribution and a shift in $IMR|BW$, holding the observable attributes constant. For example, if blacks maintained their own $IMR|BW$, but had a birthweight distribution generated by the white distribution of attributes, the black IMR would fall by 1.33, from 13.21 to 11.88; the remaining 0.58 of the overall 1.91 reduction is then attributed to a change in blacks' $IMR|BW$. In an alternative calculation, if blacks maintained their own birthweight distribution, but had a $IMR|BW$ generated by the white distribution of attributes, the black IMR would fall by 0.61, from 13.21 to 12.60. As before, the order of decomposition could matter in this type of calculation, but in this case it does not – regardless of order, if blacks and whites had identical distributions of characteristics as reported on birth certificates, the black-white IMR gap would decline by roughly 0.60 (about 8 percentage points of the overall black-white IMR gap of 7.57) due to a convergence in $IMR|BW$ and 1.31 (17 percentage points) due to convergence in the birthweight distribution.

Robustness. In Table 6, we examine the robustness of our key result that about 25 percent of the black-white IMR gap can be explained by a set of commonly used observables. First focusing on the top set of results, we repeat in column 1 the calculation from above, which used DFL-based methods and weighted the black distribution to look like the white distribution. Column 2 also uses DFL-based

methods, but instead weights the white distribution to look like the black distribution. This calculation method indicates that 35 percent of the black-white IMR gap can be explained by the observables.

When focusing on IMR differences as in Table 6, we can use regression-based methods to assess the amount explained as do previous studies. In column 3, we use OLS, regressing the death indicator variable on the regressor set used for the DFL-based methods and a binary indicator for race. The percent explained is measured by the attenuation in the coefficient on the race indicator relative to a regression model with no other covariates. Column 4 reports analogous results to column 3, but instead uses a logit model, recognizing the binary nature of our dependent variable.¹² The OLS model returns an amount explained of 29 percent, whereas the logit model returns an amount explained of 40 percent.

In columns 5 and 6, we use an Oaxaca-Blinder decomposition to assess the amount explained, with column 5 using the black regression coefficients and column 4 using the white regression coefficients. The results are very similar.¹³ Column 5 indicates that about 30 percent of the gap can be explained, whereas Column 6 indicates about 33 percent of the gap explained.

Table 6 shows two additional sets of results to allow us to examine changes over time. The middle set shows results for 2001, but only uses the regressors that are available consistently for both 2001 and 1989. The last set shows results for 1989. Comparing the top and middle sets of results, we see that the amount explained increases a little when we move from the first to the second regressor set, dropping information on pre-natal smoking and drinking. These results suggest that the drinking and smoking behavior for whites was, on average, more harmful to infant mortality than was the behavior for blacks; this result is reflected in the descriptive statistics for smoking in that whites are more likely

¹² The amount of explained for the logit model is evaluated by evaluating the marginal effect for each individual and then averaging these marginal effects.

¹³ In results not presented here, we show that the DFL-based methods and Oaxaca-Blinder decompositions return identical results whenever the regressor sets include a complete set of dummy variables and their interactions (i.e., a fully saturated model).

to report smoking than are blacks (see Appendix Table A3). Comparing the middle and bottom sets of results, we see that somewhat more of the racial IMR gap can be explained by observables in 1989, a trend that has been reported in other studies as well (see Collins and Thomasson 2004).

See Appendix Table A4 for the coefficients from the OLS results.

6. Can Live-Birth Reporting Differences Explain the Black-white Gap?

[The results in this subsection should be treated as very preliminary—they are based on analyses that are currently in progress.]

Although we carefully examined the timing of infant deaths in Section 4 (prenatal versus post-neonatal), another potentially important timing issue exists that has received very little attention in the racial IMR gap literature: fetal deaths versus infant deaths. A given pregnancy can end in an infant death only if the infant is first determined to be a live birth, and thus a birth certificate is issued. Even though the United States Vital Statistics system lists a clear definition for a live birth, such a definition may be difficult in practice to apply consistently across time and jurisdiction. Indeed, MacDorman et al. (2005) consider reporting changes at the margin between fetal deaths and neonatal deaths as a possible factor in the rising share of live births reported at less than 750 grams, concluding that reporting changes “cannot be discounted as a factor.” In this section, we present some preliminary results to shed light on whether reporting differences may influence the measured racial IMR gap.

We saw in Section 4.3 that about 2/3 of infant deaths are classified as neonatal deaths in 2001, which amounts to 17,709 neonatal deaths. If we further break down neonatal deaths by timing, 10,447 of these neonatal deaths (59.0 percent) took place within the first 24 hours. During the same year, 24,817 fetal deaths were reported for fetuses that were classified to be over 20 weeks of gestation. Thus, even if a small fraction of fetal deaths were reported as infant deaths or vice versa, such differential reporting could have a large effect on measured infant mortality. As an example, the rate of fetal deaths of 20 weeks or more gestation was 11.6 for blacks in 2003 (MacDorman et al. 2005). A

10 percent shift in reporting between fetal deaths and infant deaths would thus change the reported black infant mortality rate by about 1.2, or nearly 15 percent of the gap between black and white infant mortality in that year.

Figure 8 shows the birthweight (Panel A) and gestational age (Panel B) distributions for three groups: fetal deaths after 20 weeks, neonatal deaths in the first 24 hours, and the balance of neonatal deaths. Whereas the densities for neonatal deaths in the first 24 hours are clearly shifted to the left of the densities for neonatal deaths during the rest of the first month, the fetal death densities are located between the other two. As measured by birthweight and gestational age, the fetal death population is not less mature than the population of infants who die in the first 24 hours. The possibility for ambiguity in reporting of fetal deaths versus infant deaths is even greater once one considers that there are 2.5 times more fetal deaths than infant deaths in the first 24 hours and 3 times more fetal deaths than neonatal deaths after the first 24 hours.

Table 7 provides an initial analysis regarding the extent to which the inclusion of fetal deaths can close the measured black-white IMR gap. The regressions should be viewed as only suggestive because missing data are much more of an issue for the fetal death data than they are for the linked cohort data, so our previous practice of simply including a dummy variable for missing is no longer feasible (see Appendix Table A5). As an extreme example, because there is only missing marital status in the fetal death file, including a missing marital status indicator would predict death with great accuracy. For our analysis in Table 7, we drop marital status as an indicator variable in the analysis, but similar issues arise to a less extreme extent for several other variables. With that caveat in mind, Table 7 shows some evidence that the inclusion of fetal deaths reduces the unexplained mortality gap.

7. Summary and Discussion

In this paper we explore the persistent disparity in IMR between blacks, whites, and other racial and ethnic groups in the U.S., paying special attention to the relative roles of birthweight distributions

and infant mortality conditional on birthweight. Consistent with other research, we find that in 2001 most of the disparity in IMR between blacks and whites (about 90 percent) is accounted for by differences in the distributions of birthweight, although our methods highlight where in the distribution differences arise. We also find that blacks have higher conditional mortality post-neonatally, and when this post-neonatal difference in $IMR|BW$ is isolated (Table 4, Panel C), it can by itself account for 16-19 percent of the IMR disparity.

We also adapt the methods of DiNardo, Fortin and Lemieux (1996) to examine the extent differences in a broad set of attributes, including education, place of residence, mother's age and marital status, smoking and drinking during pregnancy, and use of prenatal care, can explain black-white IMR differences. In other words, we examine how black IMR would be different if black infants in 2001 had their actual mortality experience, but the population of black infants was weighted in such a way that its distribution of attributes closely matched the white population. To the extent that weighting does matter for IMR, the DFL approach allows us to decompose the effect into an effect on birthweight distribution and an effect on $IMR|BW$.

We find that weighting the population of black infants to match white attributes has a limited effect on the distribution of birthweights, and therefore not surprisingly, a limited effect on the black-white IMR disparity. Nonetheless, weighting using our broad set of attributes reduces the black IMR by 1.90, about a 25 percent reduction in the actual gap of 7.56. Of that reduction, about two thirds comes from an improved distribution of birthweight. We then use several alternative methods to assess the overall amount of the racial IMR gap explained by observables. Our results suggest that about one-third of the racial IMR gap can be explained.

Motivated in part by the importance of the difference in the birthweight distribution between blacks and whites, we then examine whether the racial IMR gap can be explained by reporting differences in

fetal deaths and infant deaths. Our preliminary results provide some evidence that reporting differences could be important.

Appendix Tables

Table A1 provides descriptive statistics for the various data files used in the paper.

Table A2 provides information about fetal death reporting requirements state by state for 2001.

Table A3 provides descriptive information for the weighted and unweighted black sample.

Table A4 provides the OLS results for Table 6.

Table A5 provides descriptive information for the observable characteristics by timing of death.

Appendix Table A1. Summary Statistics for NCHS Linked Cohort Files and Fetal Death Files

	Cohort File 1985	Cohort File 1989	Cohort File 2001	Cohort File 2002
Sample size		4,045,880	4,031,646	
IMR (per 1,000)		9.54	6.74	
Race				
White		.790	.790	
Black		.166	.150	
Native Amer.		.010	.010	
Asian/Pacific Is.		.033	.050	
Missing		.001	0	
Hispanic ethnicity				
Yes		.132	.212	
No		.816	.782	
Missing		.052	.006	
Maternal edu.				
Missing		.089	.014	
<12		.211	.214	
12		.356	.311	
13-15		.185	.213	
16		.104	.152	
17		.054	.096	
Maternal age				
<20		.128	.113	
20-24		.267	.254	
25-29		.313	.263	
30-34		.208	.234	
35-39		.073	.112	
>39		.011	.024	
Multiple birth		.023	.032	
Smoked cigarettes				
Yes		.131	.103	
No		.542	.760	
Missing		.327	.137	
Drank alcohol				
Yes		.029	.007	
No		.660	.854	
Missing		.311	.139	

Married		
Yes	.729	.665
No	.271	.335
Missing	0	0
First prenatal care		
1 st	.163	.217
2 nd	.370	.413
3 rd	.205	.184
4 th	.090	.067
5 th – 9 th	.151	.096
Missing	.021	.024
Urban county	.630	.650
Birthweight		
Mean	3342	3305
Median	3374	3345
Missing	.001	<.001
Gestational age		
Mean	39.2	38.7
Median	39	39
Missing	.014	.010

Notes. This table presents tabulations from the underlying linked birth certificate/death certificate cohort files.

Appendix Table A2. 1997 Fetal Death Reporting Requirements by State

All products of human conception:

AR, CO, GA, HI, NY, RI, VA

20 weeks gestation or more:

AL, AK, CA, CT, FL, IL, IN, IA, ME, MD, MN, NE, NV, NJ, NC, ND, OH, OK, OR, TX, UT, VT, WA, WV, WY

20 weeks gestation or more OR weight of at least 350 grams:

AZ, DE, ID, KY, LA, MA, MS, MO, MT, NH, SC, WI

Gestation of 20 weeks or more OR weight of at least 400 grams:

MI

Gestation of 20 weeks or more OR weight of at least 500 grams:

DC

Weight of at least 350 grams:

KS

Weight of at least 500 grams:

NM, SD, TN

Gestation of 16 weeks or more:

PA

Notes: This information comes from NCHS's "State Definitions and Reporting Requirements for Live Births, Fetal Deaths, and Induced Terminations of Pregnancy," available at <http://www.cdc.gov/nchs/data/misc/itop97.pdf>. This information is accurate as of 1997 and presumed accurate as of 2001.

Appendix Table A3. Summary Statistics by Race for DFL Analysis

	Whites	Blacks	Weighted Blacks
Sample size	2,327,114	590,105	590,105
Maternal education			
Missing	.008	.017	.009
<12	.119	.244	.124
12	.303	.392	.301
13-15	.240	.228	.239
16	.204	.077	.193
17	.126	.043	.133
Maternal age			
<20	.082	.189	.077
20-24	.225	.329	.236
25-29	.268	.226	.260
30-34	.269	.156	.261
35-39	.129	.081	.135
>39	.028	.019	.032
Multiple birth	.036	.035	.032
Smoked cigarettes while preg.			
Yes	.143	.086	.175
No	.779	.854	.739
Missing	.078	.060	.086
Drank alcohol while preg.			
Yes	.009	.009	.009
No	.911	.929	.902
Missing	.080	.062	.088
Married	.775	.314	.795
Month first sought prenatal care			
1 st	.229	.191	.232
2 nd	.461	.347	.455
3 rd	.179	.183	.177
4 th	.052	.091	.051
5 th – 9 th	.061	.156	.065
Missing	.018	.032	.019
Urban county of occurrence			

Notes. The other regressors used to calculate weights are a complete set of indicator variables for states and the District of Columbia.

Appendix Table A4. OLS Results

	2001 Infant Death	2001 Infant Death
Black indicator	.0076 (.0001)	.0054 (.0001)
Maternal education		
Missing		--
<12		-.0182 (.0005)
12		-.0196 (.0005)
13-15		-.0204 (.0005)
16		-.0214 (.0005)
17		-.0217 (.0005)
Maternal age		
<20		--
20-24		-.0010 (.0002)
25-29		-.0011 (.0002)
30-34		-.0014 (.0002)
35-39		-.0007 (.0002)
>39		.0003 (.0004)
Multiple birth		.0267 (.0003)
Smoked cigarettes while pregnant		
Yes		-.0054 (.0011)
No		-.0083 (.0011)
Missing		--
Drank alcohol while pregnant		
Yes		.0010 (.0011)
No		-.0017

		(.0010)
Missing		--
Married		-.0016 (.0001)
Month first sought prenatal care		
1 st		--
2 nd		-.0007 (.0001)
3 rd		-.0012 (.0002)
4 th		-.0012 (.0002)
5 th – 9 th		.0012 (.0002)
Missing		.0098 (.0004)
Urban county of occurrence		.0017 (.0001)
State/DC indicator variables	Yes	Yes
R-squared	.0013	.0067
N	2,917,219	2,917,219

Notes. . .

Appendix Table A5. Descriptive Statistics for Fetal Death File

	Fetal Deaths	Neo, <24 hrs	Neo, rest	Post-neo
Sample size	24,817	10,447	7,262	8,745
Race/Ethnicity				
White	.488	.482	.516	.497
Black	.280	.322	.261	.290
Hispanic	.194	.163	.189	.180
Asian/Pacific Islander	.038	.033	.035	.033
Married				
Yes	.356	.543	.567	.535
No	.300	.457	.433	.465
Missing	.344	0	0	0
Maternal education				
Missing	.119	.074	.030	.016
<12	.206	.224	.249	.334
12	.326	.333	.329	.358
13-15	.179	.196	.204	.178
16	.114	.113	.115	.071
17	.056	.061	.726	.043
Maternal age				
<20	.131	.146	.149	.199
20-24	.244	.255	.256	.328
25-29	.230	.247	.235	.219
30-34	.214	.205	.201	.145
35-39	.136	.119	.120	.082
>39	.045	.028	.038	.027
Multiple birth	.090	.201	.174	.079
Smoked cigarettes while preg.				
Yes	.119	.123	.139	.217
No	.660	.756	.740	.671
Missing	.220	.121	.122	.112
Drank alcohol while preg.				
Yes	.014	.011	.012	.013
No	.781	.867	.864	.873
Missing	.205	.123	.124	.114
Month first sought prenatal care				
1 st	.210	.235	.241	.190
2 nd	.330	.362	.371	.347
3 rd	.148	.144	.155	.181

4 th	.061	.055	.062	.084
5 th – 9 th	.121	.117	.120	.162
Missing	.129	.086	.050	.036
Urban county	.673	.699	.713	.637

Notes.

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Table 1. Tabulated and Published 2001 IMR by Race/Ethnicity

	Births	Linked Deaths	IMR*	Published IMR**
White	2,327,114	13,128	5.64	5.72
Black	590,105	7,793	13.21	13.46
Hispanic	856,632	4,650	5.43	5.44
Asian or Pacific Islander	195,210	883	4.52	4.73
American Indian***	38,023	354	9.31	9.65
Unclassified Ethnicity	24,562	372	15.15	na
All	4,031,646	27,180	6.74	
Including Unlinked Deaths	4,031,646	27,472	6.81	6.84

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.” The published IMRs are based on the 2001 linked period files.

* Infant deaths in the first year of life per 1,000 live births.

** Mathews and MacDorman (2007).

*** Includes Eskimos and Aleutian Islanders.

Table 2. 2001 Actual and Counterfactual IMRs by Race/Ethnicity

BW Distribution	IMR BW			
	White	Black	Hispanic	Asian
White	5.64	6.63	5.45	4.87
Black	12.84	13.21	12.38	11.67
Hispanic	5.68	6.58	5.43	4.83
Asian	5.53	6.43	5.30	4.52

Notes. Counterfactual IMR calculations are based on equations (1a) and (1b) in text. The diagonal elements (shaded) are the actual IMRS. The off-diagonal elements are the counterfactual IMRs.

Table 3. The Effect of Changing Birthweight Distributions by Race/Ethnicity and Year

	White	Black	Hispanic	Asian
Actual IMR for 1989	7.77	17.97	8.09	7.26
CF IMR: 1989 IMR BW, 2001 BW	8.75	18.68	8.37	7.89
Actual IMR for 2001	5.64	13.21	5.43	4.52
CF IMR: 2001 IMR BW, 1989 BW	5.07	13.04	5.53	4.18

Notes. Counterfactual IMR calculations are based on equations (1a) and (1b) in text. All counterfactual calculations use quantities for ones own race.

Table 4. 2001 Actual and Counterfactual NMR, PMR, and PMR^c by Race/Ethnicity

Panel A: Actual and Counterfactual NMR			
BW Distribution	NMR BW		
	White	Black	Hispanics
White	3.77	3.55	3.63
Black	10.04	8.91	9.57
Hispanic	3.78	3.49	3.59

Panel B: Actual and Counterfactual PMR			
BW Distribution	PMR BW		
	White	Black	Hispanics
White	1.87	3.08	1.82
Black	2.80	4.29	2.81
Hispanic	1.90	3.10	1.84

Panel C: Actual and Counterfactual PMR ^c			
Surviving BW Distribution	PMR ^c BW		
	White	Black	Hispanics
White	1.87	3.05	1.81
Black	2.89	4.29	2.85
Hispanic	1.92	3.08	1.84

Notes. Counterfactual IMR calculations are based on equations (2) and (3) in text.

Table 5. 2001 Actual and Counterfactual IMRs by Race/Ethnicity

BW Distribution	IMR BW		
	White	Black	Weighted Black
White	5.64	6.63	6.00
Black	12.84	13.21	12.60
Weighted Black	11.39	11.88	11.30

Notes. Counterfactual IMR calculations are based on equations (10a) and (10b) in text, where the weighted black results use DFL weights to make the black population to have observable characteristics similar to the white population.

Table 6. Robustness Analysis for Amount of Gap Explained

	DFL-w	DFL-b	OLS	Logit	OLS-w	OLS-b
2001 (gap=7.56)						
Regressor set 1						
Unexplained gap	5.66	4.93	5.39	4.52	5.29	5.04
% Explained	25.2	34.8	28.8	40.3	30.0	33.4
Regressor set 2						
Unexplained gap	5.20	4.53	4.95	3.96	4.89	4.74
% Explained	31.3	40.2	34.5	47.6	35.3	37.3
1989 (gap=10.20)						
Regressor set 2						
Unexplained gap	4.99	5.46	5.87	4.62	5.86	5.44
% Explained	51.1	46.5	42.4	54.7	42.6	46.6

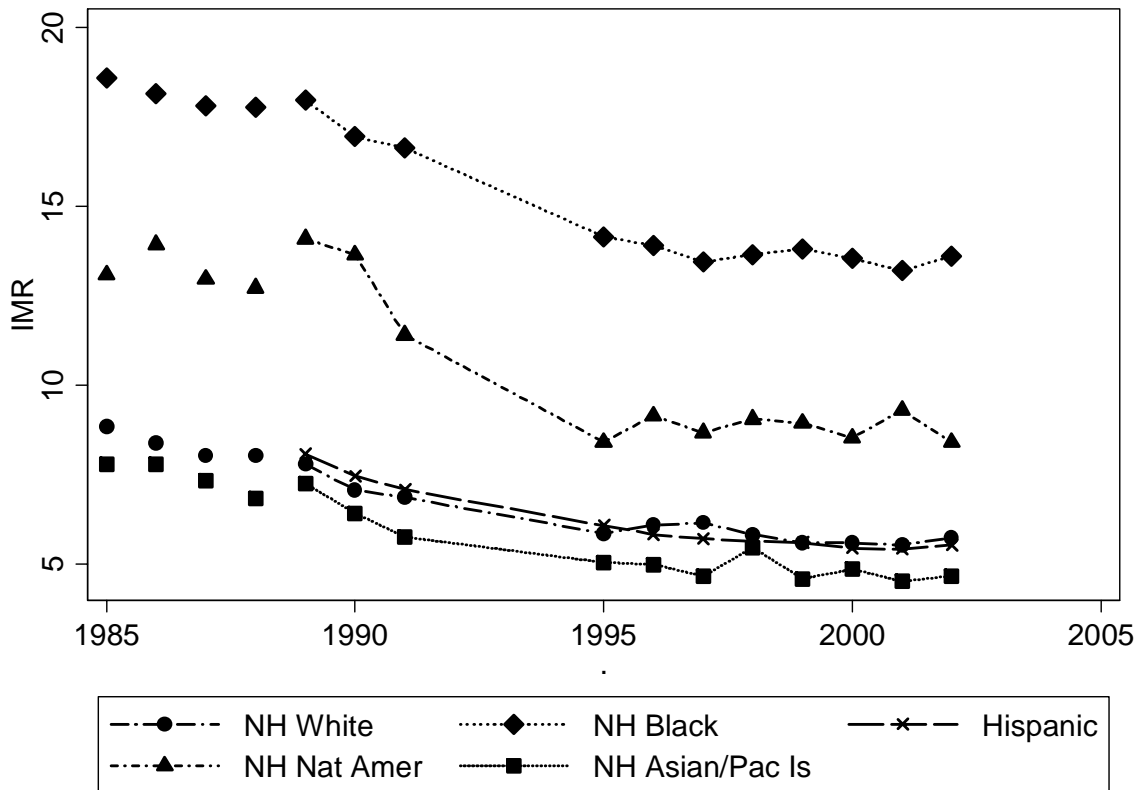
Notes. Regressor set 1 includes all of the variables listed in Table B1 and state of occurrence dummies. Regressor set 2 includes only the variables in regressor set 1 that are consistently defined in 1989, implying that we drop the smoking and alcohol consumption variables.

Table 7. Amount Explained of Fetal and Infant Deaths

	DFL-w	DFL-b	OLS	Logit	OLS-w	OLS-b
Fetal or 24 hr. deaths among all 20 wk. fetuses						
Unadjusted gap			9.94		9.94	9.94
Unexplained gap			7.80		7.88	6.43
% Explained			21.5		20.8	35.3
Fetal deaths among all 20 wk. fetuses						
Unadjusted gap			6.45		6.45	6.45
Unexplained gap			4.89		5.02	3.47
% Explained			24.2		22.1	46.2
24 hr. deaths among all live births						
Unadjusted gap			3.54		3.54	3.54
Unexplained gap			2.97		2.91	2.96
% Explained			16.2		17.9	16.4

Notes. The regressor set includes all variables in regressor set 1 of Table 6, except it excludes the marital status variable.

Figure 1. Trends in IMR by Race/Ethnicity



Notes: Infant deaths per 1,000 live births. Table is based on cohort data from 1985 through 2002, except for the years 1992-1994. Unlinked deaths are excluded. Ethnicity is not available before 1989, so ethnicity is not broken out separately in these years. The years for which ethnicity is not broken out are denoted by not having the line extend through the markers.

Figure 2. Decomposing 2001 IMR by Race/Ethnicity

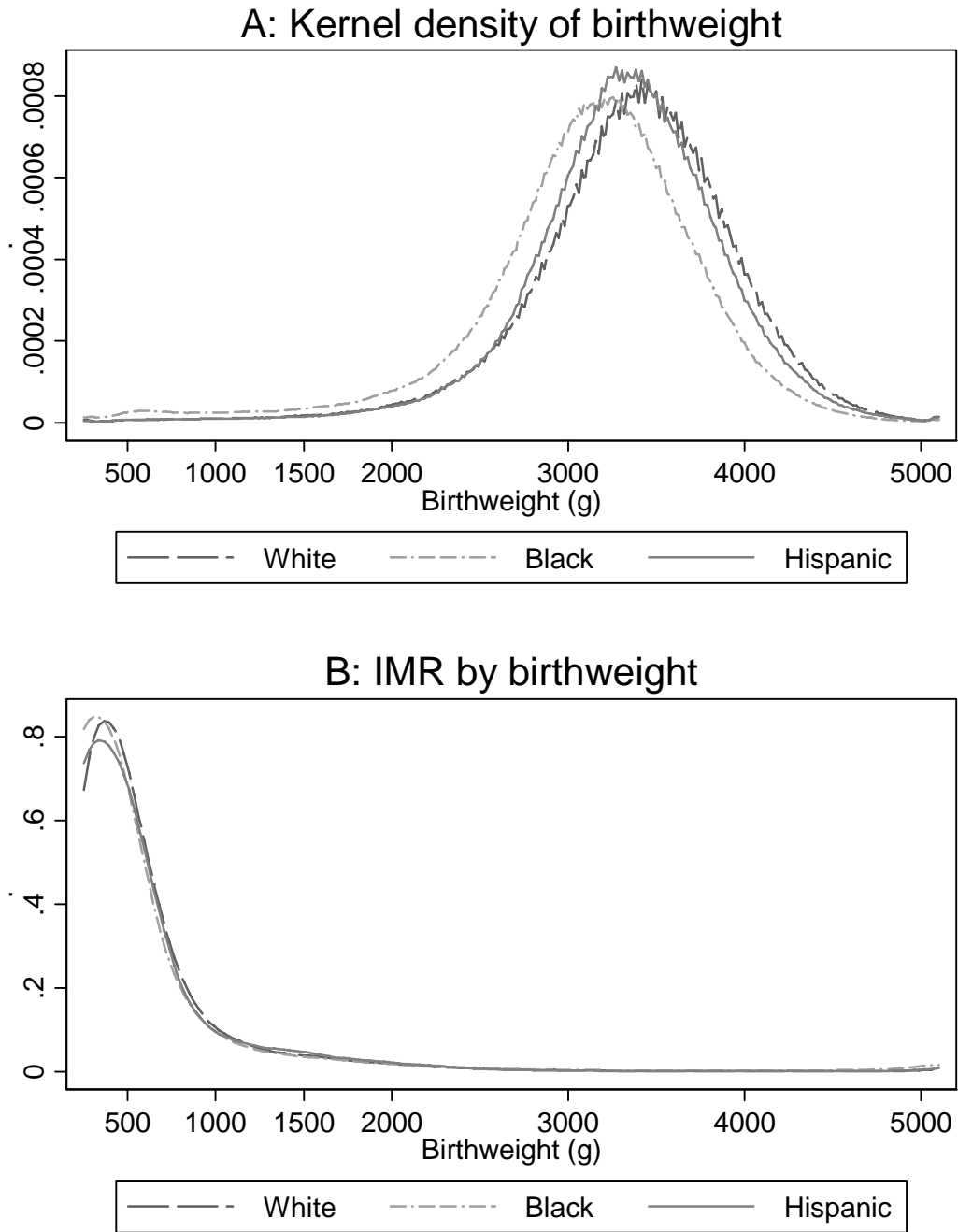


Figure 2 (cont'd). Decomposing 2001 IMR by Race/Ethnicity

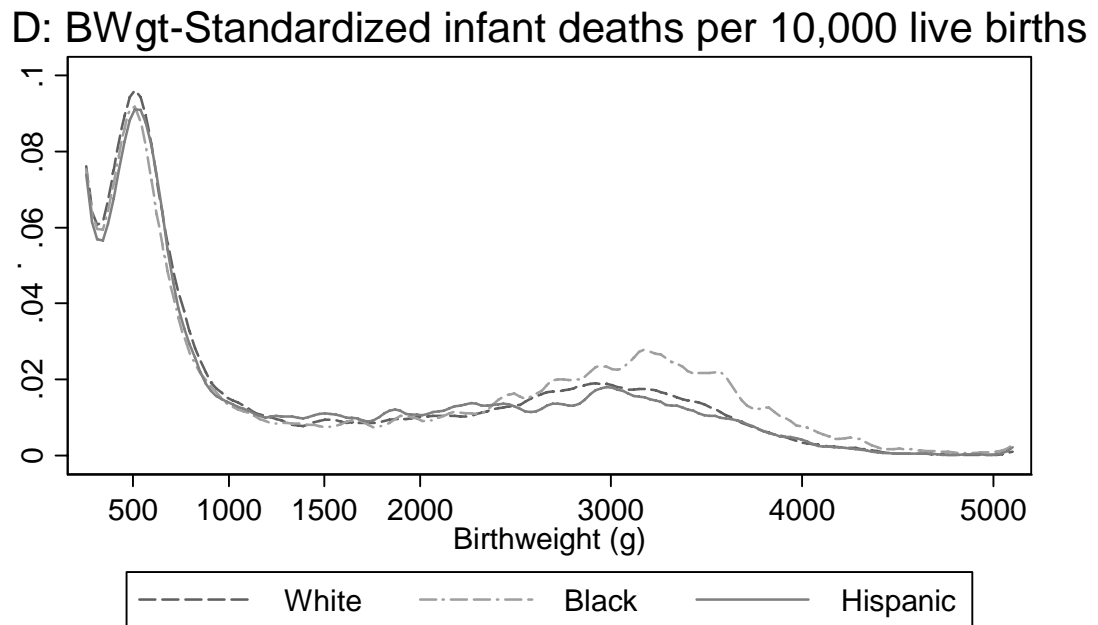
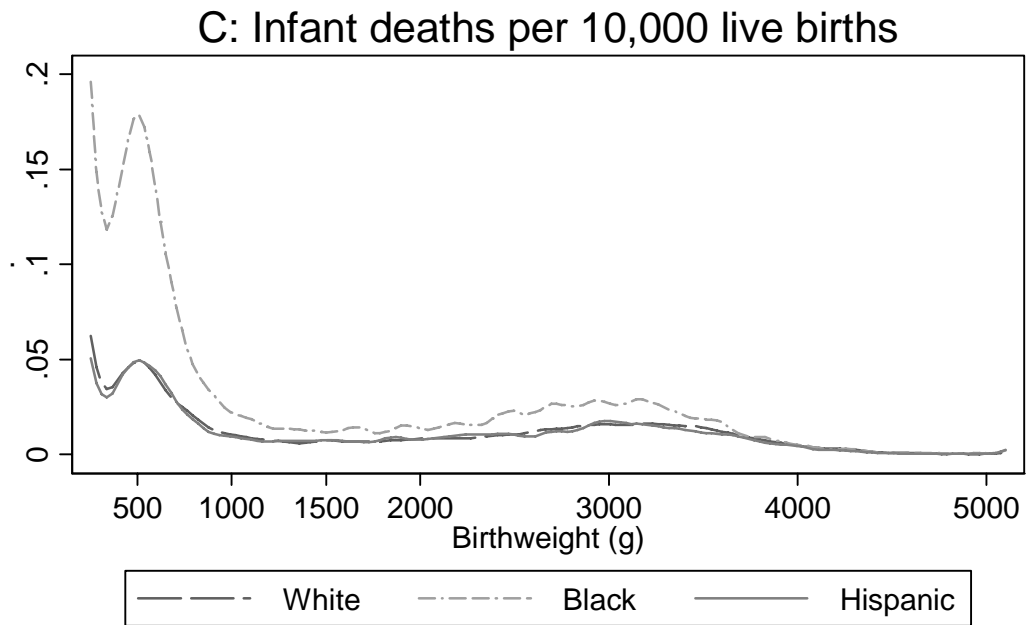


Figure 3. 1989 BWgt-Standardized Infant Deaths per 10,000 Live Births

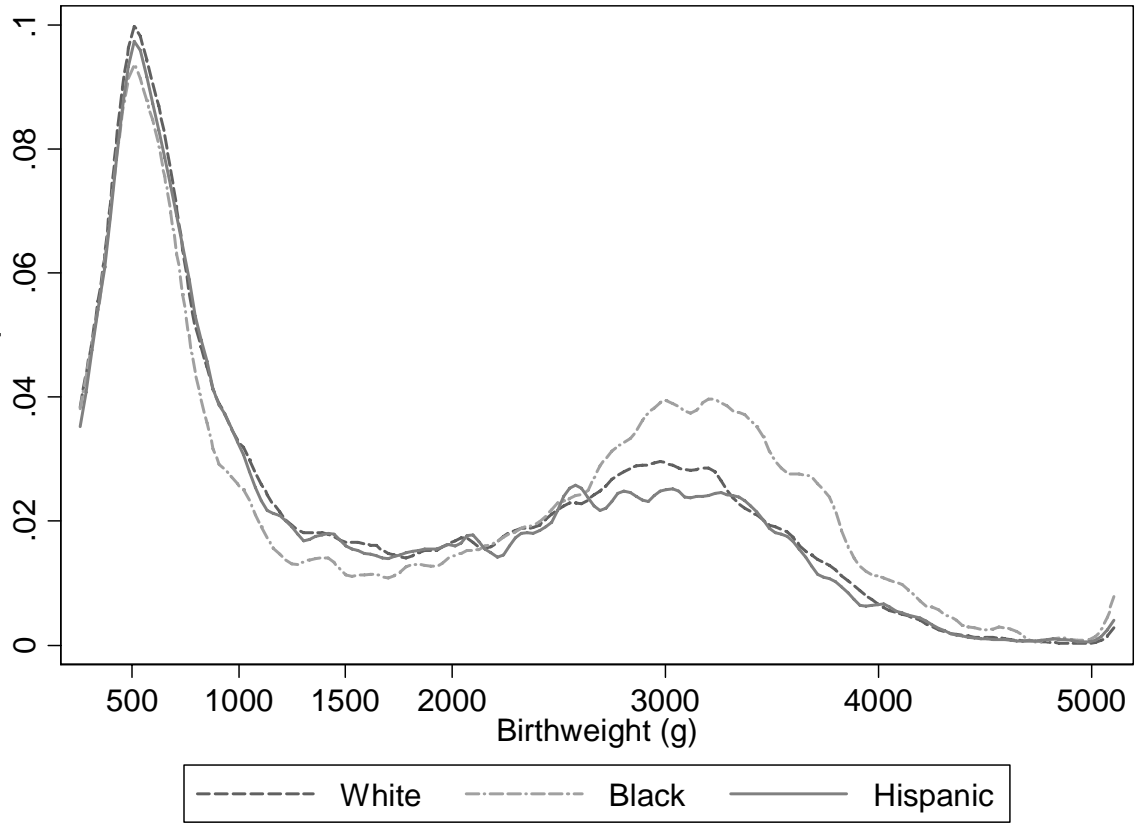


Figure 4. 1985 and 2001 BWgt-Standardized Infant Deaths per 10,000 Live Births

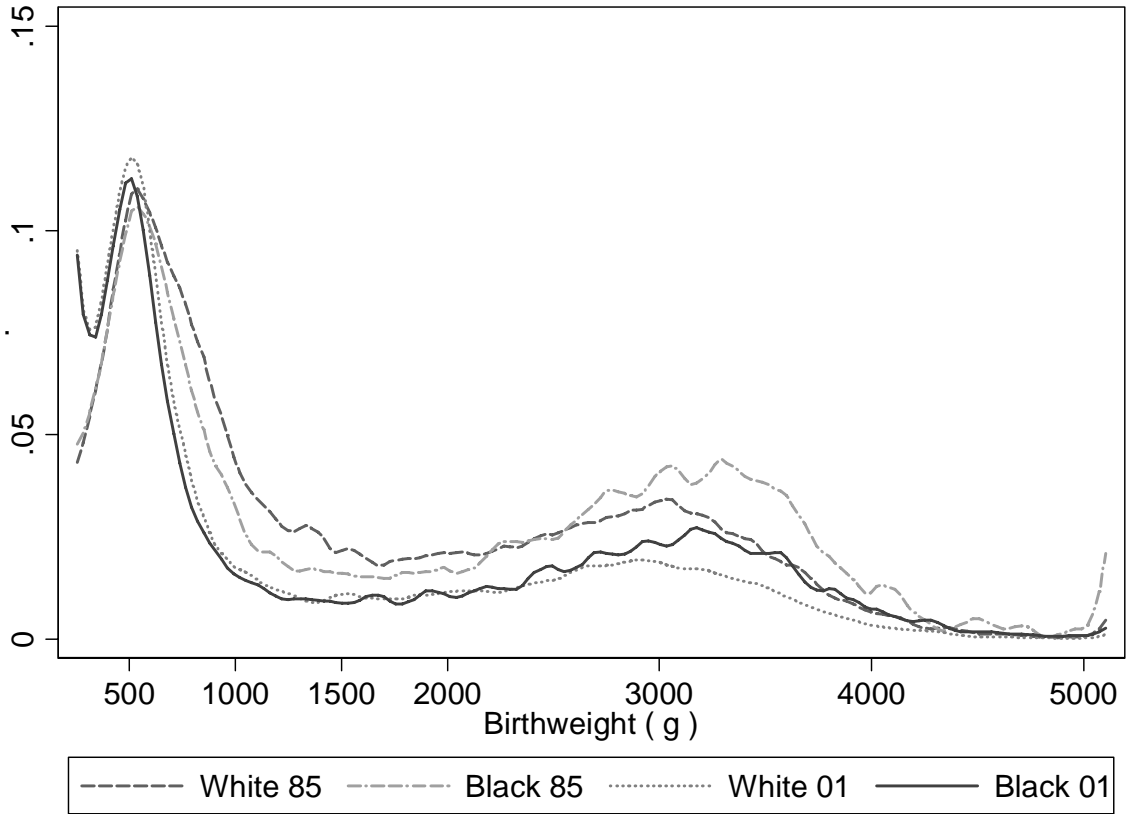
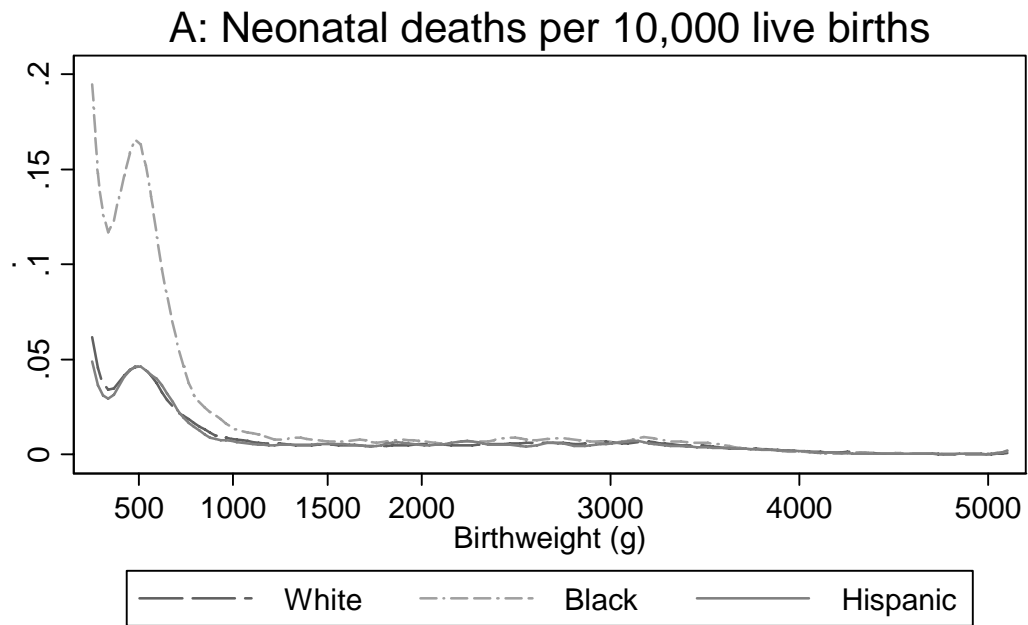


Figure 5. 2001 Neonatal and Post-neonatal Mortality per 10,000 Live Births by Race/Ethnicity



B: BWgt-Standardized neonatal deaths per 10,000 live births

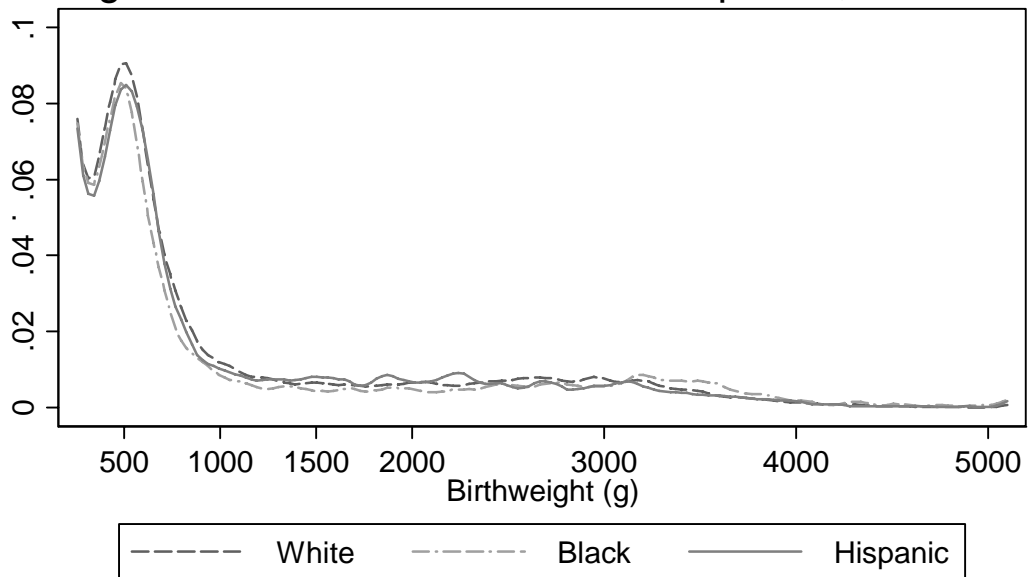
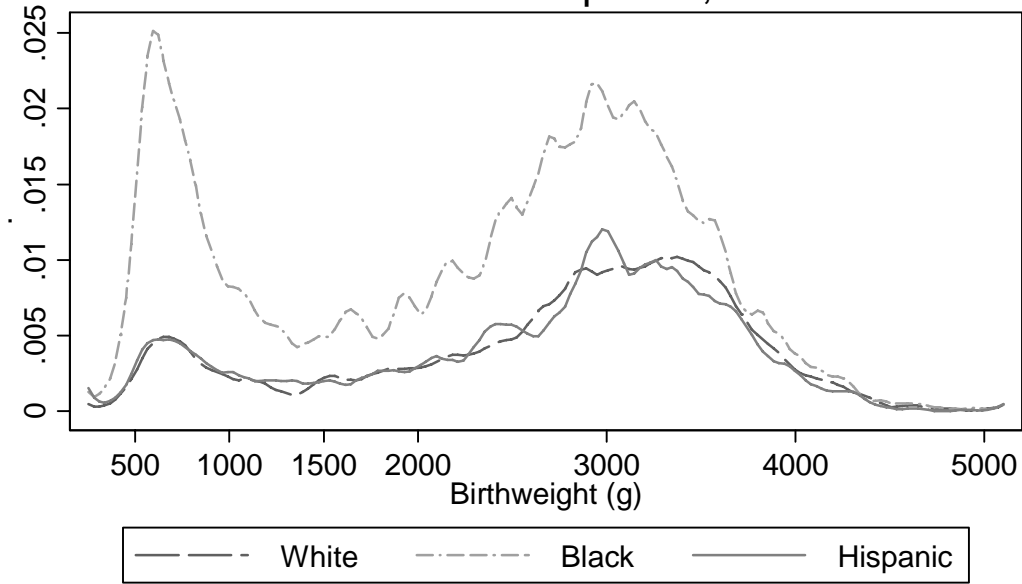


Figure 5 (cont'd). 2001 Neonatal and Post-neonatal Mortality per 10,000 Live Births by Race/Ethnicity

C: Post-neonatal deaths per 10,000 live births



D: BWgt-Standardized post-neonatal deaths per 10,000 live births

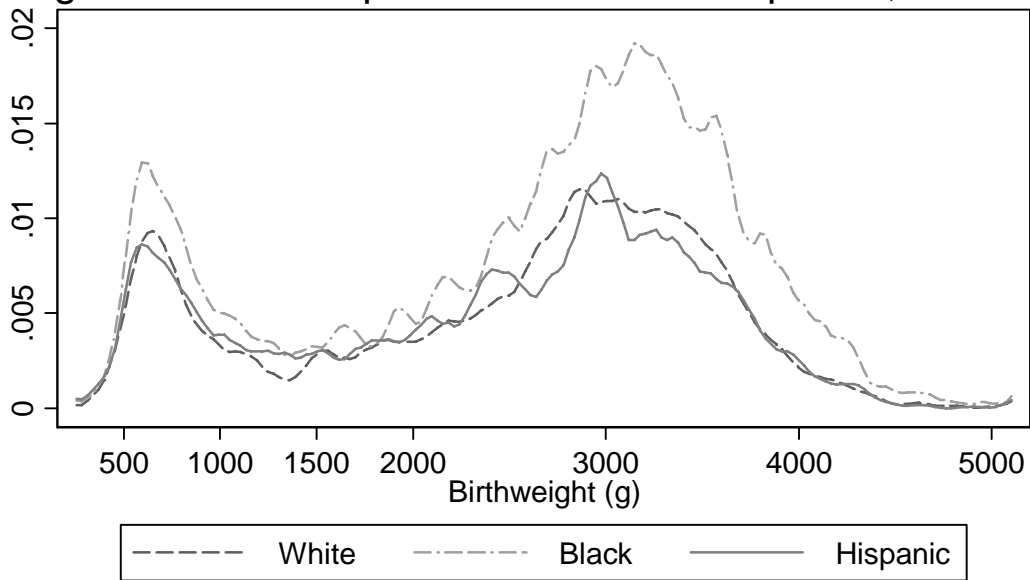


Figure 6. Kernel Density Estimates of 2001 Birthweight by Race, Weighted and Unweighted

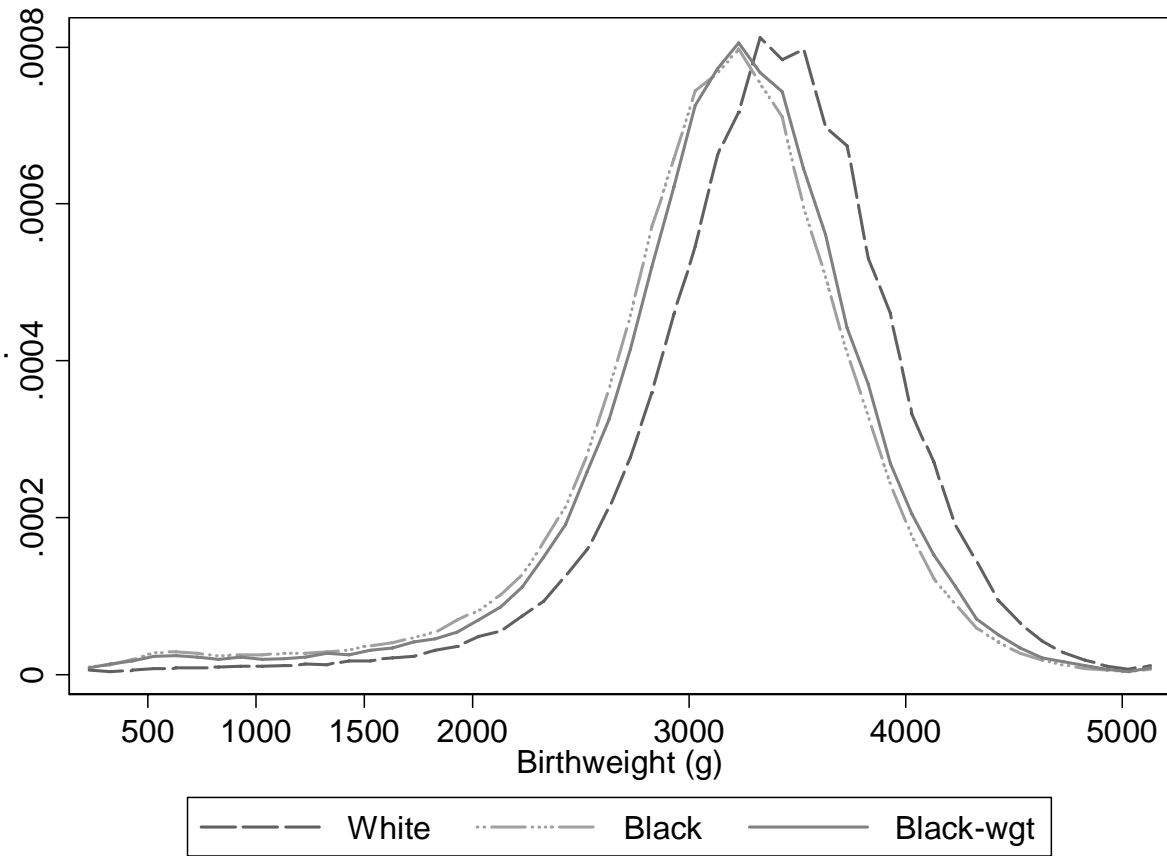


Figure 7. Decomposing Unweighted and Weighted IMR by Race

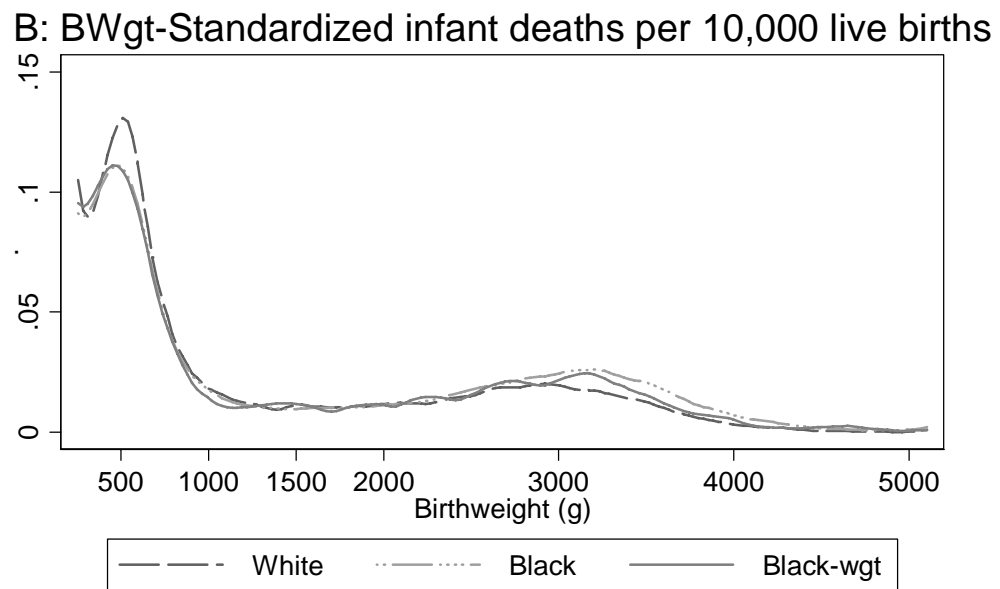
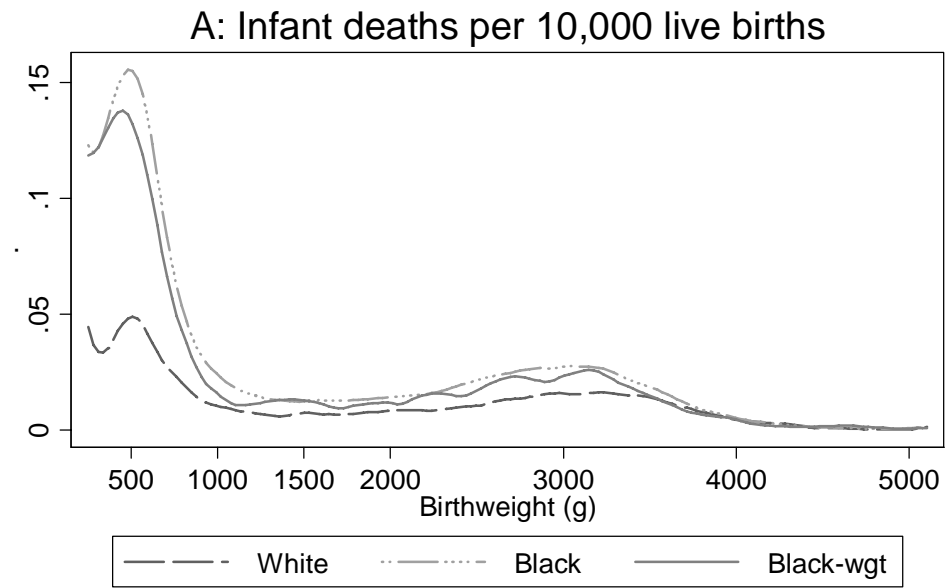


Figure 8. Comparing birthweight and gestational age densities for fetal and infant deaths

