What is Driving the Black-White Difference in Low Birthweight in the U.S.?

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Abstract

This is a first effort to quantify the contribution of different factors in explaining racial difference in low birthweight rate (LBW). Mother's health, child characteristics, prenatal care, socioeconomic status (SES), and the socioeconomic and healthcare environment of mother's community are important inputs into the birthweight production function, and a vast literature has delved into obtaining causal estimates of their effect on infant health. What is unknown is how much of the racial gap in LBW is explained by all these inputs together. We apply a nonlinear extension of the Oaxaca-Blinder method proposed by Fairlie to decompose this gap into the portion explained by differences in observed characteristics and the portion that remains unexplained. Data are obtained from several sources in order to capture as many observables as possible, although the primary data source is the Natality Detail Files. Results show that of the 6.8 percentage point racial gap in LBW, only 0.9-1.9 points are explained by white-black differences in endowments across those measures, and of those endowments, most of the gap in LBW is explained by differences in SES. The unexplained difference is attributed to racial differences in the returns to or the marginal product of investments in infant health.

Introduction

In the United States black infants are twice as likely as white infants to be born low birthweight (LBW, less than 2500 g)[1]. LBW has been linked to lower long-term health and economic well-being, including poorer health status, lower educational attainment and more limited labor market outcomes (Behrman, Rosenzweig, and Taubman 1994, Corman 1995, Currie and Hyson 1999, Osmani and Sen 2003, Behrman and Rosenzweig 2004). Furthermore, LBW is a leading cause of black infant mortality, which also exceeds that of white infants by a substantial margin (Geronimus and Bound 1990, Alexander 2000, Abrevaya 2001, Almond, Chay, and Greenstone 2001). In part because of these differences, two of the goals of Healthy People 2010 (DHHS 2000) are to improve the health and well-being of women and infants and to reduce racial disparities in health and health care.

There is clear evidence that white infants are healthier at birth than black infants (Kleinman and Kessel 1987, Cramer 1995, DHHS 2000), and a great many studies have examined the factors responsible for this persistent racial difference. One plausible explanation for the racial gap in birthweight is racial difference in length of gestation. In fact, the higher rate of preterm birth (PTB, gestation $\leq$ 37 weeks) among blacks has been found to explain a quarter of the black-white difference in birthweight (Costa 2004); however, the racial gap in birthweight persists even after controlling for differences in gestation (Rosenzweig and Schultz 1982, 1983).

A number of studies have delved into the black-white difference in infant health, and the findings are that prenatal care use, mother's socioeconomic status, unemployment rate at the time of conception, and the availability of health services are important inputs

[1] Authors' calculations from the 1991 and 2001 Natality Detail Files.

into the birthweight production function and that each of these factors is related to the black-white difference in birthweight (e.g., Emanuel, Hale, and Berg 1989, Frank et. al 1992, Joyce 1994, Cramer 1995, Warner 1995, Geronimus 1996, Abrevaya 2001, Meara 2001, Raus, Andrews, and Garfinkel 2001, Currie and Grogger 2002, Aizer, Lleras-Muney, and Stabile 2004, Dehejia and Lleras-Muney 2004). A similar set of factors, particularly parents' socioeconomic status (education and marital status), number of prenatal care visits, mother's age and stress have also been linked to the probability of preterm birth (Waldron, Hughes, and Brooks 1996, Dole et al. 2003, Raatikainen, Heiskanen, and Heinonen 2005, Conway and Deb 2005). Although the focus of Chandra and Skinner (2004) was not children, they find that the quality of health care in areas that blacks tend to live and seek care in is on average lower than that of whites.

While much of this extensive body of literature has focused on obtaining causal estimates of the effect of prenatal inputs on infant health, this paper is descriptive in nature. It draws upon this literature and contributes to it by asking a hitherto unanswered question: to what extent do differences in observable *characteristics* of black and white mothers explain the overall difference in black-white rates of LBW in the U.S.? This will also allow us to comment on how much of the racial difference in birthweight may be attributed to the black-white difference in the *effect* of these inputs. Results of this analysis will shed light on the fraction of the racial gap in LBW that can be potentially closed by narrowing the gap in observable characteristics, which is arguably the more policy-relevant portion of this racial gap in infant health.

Our primary data source is the 1991 and 2001 Natality Detail Files which is a compilation of birth certificates of the universe of children born in the U.S. in those years. These data are augmented with details about the socioeconomic and health environment in the mothers' metropolitan (MSA) area of residence. We apply the Oaxaca-Blinder method (Blinder 1973; Oaxaca 1973), a tool that is often used in the labor literature but to the best of our knowledge has not been applied in this context. We use this method to decompose the race/ethnic differences in LBW into the part that is attributable to differences in observable characteristics and the part that is due to other factors.[2] Since we are focusing on a binary outcome (LBW), we use a nonlinear variation on the decomposition method, introduced by Fairlie (1999) (described below).

Model

The theoretical basis for our empirical model is the household production function (Becker 1965, 1981) and the idea that parents derive utility from the health and well-being of their children. Individuals, subject to a budget constraint, combine market goods and their own time to produce health (Grossman 1972) and parents' prenatal inputs may be viewed as investments in the production of infant health. A simplified infant health production function would take the following form:

(1) $H = g(X, \varepsilon)$

where H is a measure of infant health such as an indicator for LBW; X is a vector of inputs that produce infant health which includes mother's prenatal care use and other determinants of health such as the child's sex and birth order; and ε is the household's or mother's endowment that she take into account when making input decisions. These endowments, which are not necessarily observed by researchers, include mother's health, her experiences with previous pregnancies, and her expectation regarding her infant's

[2] In health and healthcare research, the Oaxaca-Blinder method has been used to study health insurance coverage, smoking, malnutrition, and health care access and use (Hargraves and Hadley 2003).

health which may influence prenatal choices. This unobserved endowment heterogeneity is said to make prenatal inputs (e.g. timing of prenatal care initiation, doctor visits, tobacco use, and weight gain) endogenous in the health production equation (Rosenzweig and Schultz 1982, 1983, Corman, Joyce and Grossman 1987, Frank et al. 1992, Currie and Grogger 2002). We draw on this literature in selecting the factors to be included in our analysis--mother's health, child characteristics, use of prenatal care, socioeconomic status, and the socioeconomic, health and healthcare environment of the mother's community. We are unable to obtain causal estimates of the potentially endogenous variables here, but we attempt to address this issue by estimating our model both with and without these endogenous variables (discussed later).

The goal of the paper is to examine the extent to which black-white differences in observable factors explain the racial difference in LBW. One reason an infant is LBW is because it is born too early (Paneth 1995), thus we estimate our decomposition model for LBW, preterm birth (PTB), and LBW conditional upon being full term. The method we use for decomposing the racial difference in LBW and PTB was introduced in the labor economics literature by Blinder (1973) and Oaxaca (1973), and is traditionally used to explain gender and racial differences in wage rates. We use a nonlinear extension of the Oaxaca-Blinder method due to Fairlie (1999) because our key outcome of interest (LBW) is binary. Fairlie (2005) demonstrates that although the Oaxaca-Blinder method yields a good approximation of the nonlinear decomposition for binary outcomes such as LBW, a nonlinear decomposition that uses a logit or a probit is more appropriate when the racial gaps lie in the tails of the distribution (as is the case for LBW and PTB).

We begin by estimating the health production equations separately for white, non-Hispanic (hereafter white) and black, non-Hispanic (hereafter black) infants. The two estimation equations are expressed as follows:

(2) $H_i^w = F(X_i^w \beta^w + \varepsilon_i^w)$

(3) $H_i^b = F(X_i^b \beta^b + \varepsilon_i^b)$

The Xs that enter equations 2 and 3 are the same (described below) and these equations represent the health production functions of white and black infants, respectively. Since the outcomes of interest are binary, we use probit estimation and F represents the standard normal cumulative density function. The estimated average health outcomes or the estimated average probability of LBW (or PTB) of white and black infants are thus:

(4) $\overline{H}^w = \frac{1}{N^w} \sum_{i=1}^{N^w} F(X_i^w \hat{\beta}^w)$

(5) $\overline{H}^b = \frac{1}{N^b} \sum_{i=1}^{N^b} F(X_i^b \hat{\beta}^b)$

The main object of interest in this analysis is the difference between (4) and (5). By adding and subtracting $\frac{1}{N^b} \sum_{i=1}^{N^b} F(X_i^b \hat{\beta}^w)$ the decomposed difference between two may be written as[3]:

(6) $\overline{H}^w - \overline{H}^b = \left[\sum_{\forall N^w} \frac{F(X_i^w \hat{\beta}^w)}{N^w} - \sum_{\forall N^b} \frac{F(X_i^b \hat{\beta}^w)}{N^b} \right] + \left[\sum_{\forall N^b} \frac{F(X_i^b \hat{\beta}^w)}{N^b} - \sum_{\forall N^b} \frac{F(X_i^b \hat{\beta}^b)}{N^b} \right]$

In case of a continuous dependent variable, the decomposition will take the familiar Oaxaca-Blinder formulation, which is a special case of (6):

[3] Fairlie (2005) shows that in a probit model the equality in (6) is approximate but empirically it is close to equal.

$$(7)\ \overline{H}^w - \overline{H}^b = \left\lfloor (\overline{X}^w - \overline{X}^b)\hat{\beta}^w \right\rfloor + \left\lfloor \overline{X}^b (\hat{\beta}^w - \hat{\beta}^b) \right\rfloor$$

Equation (6) is used because in the nonlinear case $\overline{H}$, is not necessarily the predicted probability estimated at the means of the Xs, $F(\overline{X}\beta)$. However, the intuition of both equations is the same: the first term on the right-hand side represents the portion of the overall racial difference in health due to differences in the distributions of X; X contains characteristics that are likely to be correlated with LBW and the first term measures the extent to which the higher probability of worse health outcomes among black infants may be attributable to racial differences in the distributions of factors like mothers' educational attainment, prenatal doctor visits, and access to prenatal care. It captures the difference between i) the mean predicted probability of LBW (or PTB) for white infants conditional upon the Xs included in the model and ii) what the mean predicted probability of LBW for black infants would be if their returns on health investments were the same as those of white infants. The second term represents the portion of the difference attributable to racial differences in the effect of X. The regression coefficients may also be thought of as the marginal product of inputs that produce infant health, and this term may be interpreted as the black-white difference in the returns on health investments if white infants had black infants' distribution of characteristics. These differences in coefficients may arise due unobserved factors such as quality of prenatal care, attitude, and culture. For example, one additional prenatal care visit may have a different affect on black and white infants' health because the quality of care received or the attitudes of black and white mothers may be different.

An alternative set-up for the decomposition model would be to add and subtract $\frac{1}{N^w}\sum_{i=1}^{N^w} F(X_i^w \hat{\beta}^b)$ to the difference in (4) and (5). As a result, the decomposition equation would be different in two ways: (1) the first term would be weighted by the black coefficients; and (2) the second term would be weighted by the white distribution of Xs. Whilst the resulting equations would be intuitively the same, the choice of weights may change the estimate of the fraction explained by observable differences between white and black mothers. Thus, we estimate the decomposition both ways; we first use white coefficients to weight the black and white distributions of characteristics and then use coefficients from the black equation to weight the same difference.

In our empirical analysis, the probability of LBW is assumed to be a function of correlates of infant and mothers' health that can be roughly compartmentalized into the following six categories[4]:

- Child characteristics such as child's sex and birth order;
- Maternal health which is captured by mothers' age at the time of child's birth and the absence of the following medical risk factors: hemoglobinopathy, incompetent cervix, and rh sensitization. These medical risk factors[5] are randomly assigned by nature and hence unpreventable, and are likely to be correlated with mothers' use of prenatal care and ultimately her infants' health;

[4] Fairlie (2005) shows that unlike the linear case, in the nonlinear case the independent contribution of each X to the overall fraction explained may be sensitive to the ordering of these variables. This is because in the nonlinear case the contribution of one of the Xs depends on the values of the other variables in the model. Thus, we replicate the estimation 150 times, randomize the ordering of variables in each replication, and report the average results over all orderings and replications.

[5] See Kutinova and Conway (2008) for a careful examination of mothers' health outcomes at birth (anemia, pregnancy-related hypertension, and placental abruption). The authors examine whether Medicaid expansions benefit mothers' health; in doing so they distinguish between medical risk factors that are preventable vs. those that are not preventable.

- Maternal socioeconomic status such as educational attainment, income, and marital status;
- Prenatal and perinatal characteristics such as alcohol and tobacco use, number of prenatal care visits, trimester of prenatal care initiation, and having a trained attendant at the birth;
- Health and healthcare environment, i.e., the availability of healthcare and the general health of women in the mother's community, including the prevalence of overweight/obesity, smoking, and mortality rate in the county; and
- Social and economic environment such as racial homogeneity, unemployment rate, and factors that may affect the cost of obtaining healthcare within the community of residence, i.e., population of the city of residence and geographic region.

The implementation of the Fairlie (1999, 2005) decomposition consists of several steps. We use probit to estimate the LBW and PTB equations for white and black infants separately. The white coefficients are applied to every observation in the black and white samples to obtain mean predicted probabilities in the black and white samples, respectively. The difference in these means yields an estimate of the extent to which differences in the distribution of endowments explain the racial gap in LBW and PTB rates in the U.S. We repeat this process using black coefficients because the results may be sensitive to the choice of coefficients. We report the nonlinear Fairlie (2005) estimates but we also test (results not reported) the model using linear probability models to produce standard Oaxaca-Blinder estimates and check for the sensitivity of the results to alternate estimation methods; the exception is the model that includes county fixed effects which is estimated using Ordinary Least Squares to make the estimation with our large sample size tractable   Further, as stated before, prenatal inputs are potentially endogenous in the health production equation; however, obtaining causal estimates of the effect of each of the prenatal inputs is a monumental task as it requires the use of various instrumental variables and estimation strategies**.** In order to understand how these potentially endogenous variables affect our estimation, we estimate our decomposition models both with and without them. The advantage of ignoring this bias is that it makes the problem tractable, but the disadvantage is that our results may only be interpreted as suggestive and cannot be used to make unequivocal policy recommendations.

Data

We use the 1991 and 2001 Natality Detail Files as the primary data source for this analysis. These files are a compilation of birth certificates for the universe of live births in the U.S. in 1991 and 2001. We use two years of data ten years apart for two reasons: first, this allows us to study whether the drivers of the racial gap in infant health have changed over time; and second, we can test whether the inclusion of county fixed effectsto account for time-invariant unobserved county characteristics changes the results of the decomposition. Admittedly, ten years is an ad hoc choice, but a time period shorter than that may be too narrow a time frame for county characteristics to change.

There are approximately 4 million children born in the U.S. every year which leads to a large sample for our analysis. For computational ease we use a random sample of the data, a practice also used elsewhere (see for example Currie and Moretti 2003; Mayer and Sarin 2005; Lhila 2009). In order to obtain white and black samples that are approximately equal in size, we draw 10 and 30 percent random samples of non-Hispanic white and black infants, respectively. The analytical sample is limited to singleton births to mothers who live in a metropolitan statistical area (MSA). We focus on singleton births because observations from multiple births are likely to be correlated and multiple births are disproportionately more likely to be LBW and PTB. Further, what is considered LBW or PTB for singletons is normal for multiple births; for example, 40 weeks is considered to be full term for a singleton birth, but 37 weeks is normal for a twin birth. We limit our analysis to children whose mothers live in an MSA because it provides a more homogenous healthcare environment for the mothers in our sample. We cannot include children whose mothers reside in counties with populations less than 100,000 people because the Natality Files do not provide geographic identifiers for those counties; these identifiers are needed to merge healthcare availability information from the Area Resource File (ARF, described below). Dropping observations that are missing information on key variables yields a sample of 290,615 and 257,131 white and black infant observations, respectively, for our analysis.

A key variable for this analysis is family income. Unfortunately, the Natality files do not provide income information. The lack of income information may not be problematic per se because we include in our model maternal characteristics – education and marital status that are highly correlated with mothers' income (Kaestner, Dubay, and Kenney 2002). Nonetheless, we attempt to overcome this limitation by using race-specific county-level income information as a proxy for mothers' household income. We have for each county and race the fractions of the population with income less than $25,000, $25-50,000, and greater than $50,000. Merging this to each observation by county and race, is akin to assigning to each mother the probability of belonging to each income category depending upon the race-specific income distribution in the county. We acknowledge that there may be other data sources that contain household income and prenatal care and maternal information similar to the Natality files. However, we choose to use the Natality files for two reasons. First, the large sample size lends statistical precision to our analysis. Second, the Natality files provide geographic identifiers at a level that is seldom available in public-use nationally-representative datasets; this allows us to merge county-level health supply variables and thus capture the healthcare environment in which the mothers reside.[6]

We supplement that Natality files with data from two sources -- the Area Resource File (ARF) and the 1991 and 2001 Behavioral Risk Factor Surveillance System (BRFSS). From ARF we obtain two sets of county-level variables: first, race-specific female mortality rate and number of obstetric-gynecologists per 1000 people corresponding with the NDF sample years[7] which capture the health and supply of healthcare in the mother's county of residence; second, we use the 1991 and 2001 16+ unemployment rate and fraction of the county that identifies with the same race as the mother which serve to measure the socioeconomic environment in the mother's community. BRFSS is a household survey which yields data on health risk behaviors and healthcare access in the United States. Approximately 350,000 adults are interviewed each year and BRFSS is considered to be the largest survey of its type in the world. We aggregate tobacco use and overweight/obesity among 18-45 year old female respondents to obtain county-level estimates of the prevalence of tobacco use and overweight/obesity

[6] While the ability to include information on the county of residence is a substantial improvement over prior work, it provides only an approximation of the individual's local community.
[7] For the 1991 Natality File, we use 1990 data on county obstetric-gynecologists supply and 1992 data on female mortality rate because 1991 data were not available.

among black and white reproductive-age females. These control for the general attitude towards health and health status in the mother's county of residence.

Table 1 provides the means and standard deviations of the variables included in the estimation model. The top panel of Table 1 confirms that black infants tend to be in worse health than white infants, both in terms of LBW rates and PTB rates. It shows that the black-white difference of 6.8 percentage points in LBW rates is statistically significant, and that this difference is accompanied by a substantial black-white difference in the probability of preterm birth (25.2% vs. 14.7%).

The remainder of Table 1 contains descriptive statistics separately for black and white populations and sheds light on the racial differences in observable characteristics. As shown in the table, the black-white differences in characteristics are significantly different from zero in all cases except one. This is not surprising given the large samples used in this analysis; however, the table also reveals several substantive differences between the two populations. Whilst black and white mothers are similar (in magnitude) in terms of the probability of having daughters, comparing the birth order percentages for black and white infants suggests that black mothers are likely to have more children, on average, than white mothers. Although statistically significant, the difference in probability of having no risk factors (hemoglobinopathy, incompetent cervix, and rh sensitization) is similar for black and white mothers. The maternal age distribution of black and white mothers are noticeably different: black mothers tend to be younger at the time of child birth with a greater risk of teenage motherhood (13.7% vs. 4.2%) and a 15 percentage point greater likelihood of giving birth between 19 and 24 years of age relative to white mothers.

In terms of prenatal/perinatal characteristics the story is mixed: black and white mothers are similar in the likelihood of delivering in a hospital and the probability of having a doctor or trained midwife attend child birth. However, black mothers are less likely to initiate prenatal care in the first trimester and have 1.6 fewer doctor visits, on average, compared to white mothers. Further, whereas 12.4% of black mothers use tobacco while pregnant, 15.6% of white mothers report prenatal smoking.

Black and white mothers differ quite strikingly in terms of their socioeconomic status: whereas 81.4% of white mothers are married at the time of child birth, black mothers are half as likely (32.4%) to be married at the time of child birth; black mothers tend to have lower income (based on our proxy for income)[8] and are less educated than white mothers; 39.5% of black mothers did not report fathers' information on the birth certificate compared to 7.7% of white mothers.

The social and economic environments in the county of residence are different for black and white mothers as well. Black mothers are more likely to reside in the south, tend to live in bigger cities (which is likely to be correlated with the cost of acquiring healthcare), and tend to live in counties where, on average, they are a racial minority, compared to white mothers. Finally, whereas black women tend to have higher overweight/obesity rate, black women are slightly less likely to be smokers than white women. Overall the descriptive statistics in Table 1 suggest noticeable white and black differences in the characteristics of the children, maternal health indicators, pregnancy characteristics, socioeconomic status, and the characteristics of the communites within which they reside.

[8] Although based on a proxy measure, this finding is consistent with national data showing black women tend to have significantly lower income than white women (authors' tabulations on the American Community Survey).

Results

Table 2 contains the first set of decomposition results; the first three columns contain the results using white coefficients and the last three contain results using black coefficients. The top panel of Table 2 shows that the overall racial gaps in LBW and PTB are 6.8 and 10.5 percentage points, respectively; as expected, the probability of LBW is considerably lower among full-term infants and the racial gap is 2.6 percentage points. The decomposition results in Table 2 are based on the Fairlie extension; however, we also estimate the standard Oaxaca-Blinder decomposition using a linear probability model for the black and white regressions. Results (available upon request) of the linear and nonlinear decompositions are similar.

As shown in columns 1 and 2, black-white differences in observed characteristics explain virtually the same fraction of the racial gap in LBW and PTB, at 27.2% and 27.5%, respectively. Much of that difference is driven by parents' SES, which is measured as mother's educational attainment, marital status, father's age, and our proxy for household income, and to a somewhat lesser extent their prenatal care decisions (timing of prenatal care initiation, number of doctor visits, tobacco use, etc.). By itself, black-white differences in SES explain 21.4% of the racial gap in LBW, and 19% of the difference in the probability of PTB; and differences in prenatal care use explains 13.4% and 12.4% of the LBW and PTB difference. Although maternal health, measured as mother's age and absence of medical risk factors, explains only 7.4% and 4.8% of the difference in LBW and PTB, these results suggest that differences in maternal health favor black infants. This result makes sense because black mothers tend to give birth at younger ages when the risk of LBW (Raus, Andrews and Garfinkel 2001) and PTB

(Conway and Deb 2005) is inherently lower. Column 3 shows that when we limit our sample to full-term infants, differences in observed factors explain one-third of the difference in LBW. The racial difference in SES explains the lion's share (31.1%) of the overall difference and relative to columns (1) and (2), the difference in prenatal/perinatal care explains little of the overall difference in LBW.

That prenatal/perinatal care explains a small proportion of the overall difference in infant health is not surprising since prenatal care is often found to be ineffective in lowering PTB (Alexander and Korenbrot 1995). However, that prenatal/perinatal care explains so little of the overall difference in LBW among full-term births is unexpected as the same study found prenatal care to be most effective in lowering LBW among full-term infants. We speculate that the latter may be because our study does not account for the potential endogeneity of the prenatal/perinatal care variables. While we control for a wide range of child and mother characteristics, it is possible that unobserved differences between black and white mothers (e.g., health attitudes and behaviors related to exercise and dietary intake), may affect their prenatal/perinatal care use and birth outcomes. In this analysis, we cannot separate the effects of who receives prenatal/perinatal care from the effects of receiving that care.

Columns 4, 5, and 6 show that these results are sensitive to which set of coefficients we choose as weights. When coefficients obtained from the black sample are used, the overall percent explained is lower; this shows that on average, the marginal product of inputs into the infant health production function is lower for black infants than for white infants, which may arise due to unobserved factors such as quality of prenatal care, preferences and attitudes, cultural factors, and the local environment.[9] Another way to think about the difference in results when using black coefficients is: if white infants' return on health investment were the same as black infants' return on investments, then observable differences would explain a smaller portion of the overall racial gap in infant health. According to Table 2, columns 4, 5, and 6, 13.3% of the difference in LBW, 21.1% of the difference in PTB, and 13.9% of the difference in LBW conditional upon being born full term is explained by differences in observable characteristics. Differences in SES and prenatal/perinatal characteristics each explain approximately 19% of the difference in LBW and approximately 15% of the difference in PTB. Finally, column (6) shows that conditional upon full term, SES once again explains the bulk (30.1%) of the overall difference in LBW and differences in prenatal/perinatal characteristics explain a smaller fraction of the LBW gap[10].

Table 3 presents the results of the decomposition when potentially endogenous prenatal/perinatal variables are omitted from the estimation. It shows that omitting these variables from the estimation leaves the story virtually unchanged for the racial gap in LBW and PTB. Here too, using white coefficients explains roughly a quarter of the overall difference in LBW and PTB, and using coefficients from the black regression explains a lower fraction of the overall difference in LBW and PTB – 13.3% and 18.6%, respectively.

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[9] We speculate that unobserved differences in the local environment may be a particularly important issue here as we are only able to measure the local environment at the county level. This likely masks significant black-white differences given the significant racial segregation in many communities in the United States. .

[10] Given that difference in SES plays a key role in explaining the racial gap in infant health, we estimated our decomposition model without our proxy for mother's income. This allows us to check if the proxy variable is driving our results. Results (not reported) are similar and the conclusions drawn from these results would be identical to those in Table 2. The key difference is that SES plays a smaller role (as expected) and healthcare environment plays a somewhat bigger role in explaining the LBW and PTB gaps.

Comparing Tables 2 and 3 gives the following insights: first, in the absence of controls for prenatal/perinatal characteristics, differences in SES play a larger role in explaining the overall difference in LBW and PTB. This is probably because SES is likely to be highly correlated with prenatal/perinatal characteristics so that the SES coefficients pick up part of the association between prenatal/perinatal characteristics and infant health. Second, unlike R-squared from an Ordinary Least Squares regression which increases whenever another explanatory variable is added to the right-hand side, the total percent explained by differences in observed characteristics does not necessarily increase when the number of explanatory variables increase. This is because the total percent explained sums the percent explained by the various inputs in the health production function, some of which are negative and some positive. The positive differences explain why black infants are worse off and the negative differences show that the health of black infants is protected by some of these characteristics, e.g. maternal age at child birth.

Table 4 contains the results of the decomposition conducted separately by year. The top panel shows that between 1991 and 2001, the racial gap in infant health has narrowed: the black-white gap in LBW (PTB) decreased from 7.5 (12.4) percentage points in 1991 to 6.1 (8.6) percentage points in 2001. The gaps have narrowed because the health status of black infants has improved while that of white infants has worsened; black LBW (PTB) rate declined from 11.9 to 10.8 (25.7 to 24.7) and white LBW (PTB) rate increased from 4.4 to 4.7 (13.3 to 16.1). Decomposing these differences show the following: (i) differences in SES and prenatal/perinatal characteristics are the largest drivers of racial difference in LBW and PTB in both years; (ii) differences in Xs explain a bigger percentage of LBW and PTB in 2001 than in 1991, partly because the amount of the overall gap explained by differences in Xs has increased over time (as in the case of LBW) and partly because the gap itself has decreased (as in the case of both LBW and PTB); and (iii) conditional upon full-term gestation, differences in endowments explain 22.8% of the racial gap in LBW in 1991 compared to 65.2% in 2001, but in both years, differences in SES continue to account for the bulk of the total percent explained.

Table 5 presents the decomposition results when county fixed effects are included in the model instead of county-level socioeconomic and healthcare environment variables[11]. Like the models without county fixed effects, results shows that differences in prenatal/perinatal characteristics and SES carry the most explanatory power and that the overall fraction of the LBW and PTB gaps explained is approximately 21% when county fixed effects are included. Column 3 shows that racial difference in SES and prenatal/perinatal characteristics respectively explain 23.3% and 4.7% of the racial gap in LBW controlling for full-term birth.

Discussion

This paper presents a first attempt at a comprehensive analysis of the factors that explain racial differences in LBW. In addition, we study PTB and LBW conditional upon full-term birth as an outcome because PTB is one reason why babies are born LBW. We control for a wide range of characteristics of the child, his or her mother's health, her prenatal/perinatal characteristics, her socioeconomic circumstances, and the local community. Results show that differences in the endowments of white and black women across those measures explain 13-27% of the differences in LBW and 21-27% of the

[11] We also exclude our proxy for mother's income in the model with county fixed effects since it is based on income in the county.

difference in PTB between white and black infants, depending upon which set of coefficients are used to weight the distribution of endowments. This means that of the 6.8 (10.5) percentage point gap between white and black LBW (PTB) rates, racial differences in the observable characteristics of these babies and their mothers only explain about 0.9-1.9 (2.2-2.9) percentage points. Most of the racial difference is explained by differences in SES and to a lesser extent differences in prenatal/perinatal characteristics. Difference in SES continues to be salient and prenatal/perinatal characteristics play a smaller role in explaining the racial gap in LBW when the analysis is restricted to full-term infant. Differences in mother's own health and her socioeconomic and health environment together explain a small portion of the overall difference in PTB and LBW.   This pattern of results holds when the analysis is conducted separately by year, when county fixed effects are included, and regardless of whether we use the coefficients based on the models for black or white mothers. There were, however, some differences across models that call for additional analyses, including exploring changes in black-white differences over time and differences in the role of the local community.

Dropping the potentially endogenous prenatal/perinatal variables from the model does not change the overall percent of the racial gap explained, but in the absence of these controls, the vast majority of the racial gap in LBW and PTB is attributed to the racial difference in SES. Thus, it is the racial differences in our proxy for income, father's age, mother's educational attainment, and marital status that explain the biggest portion of the racial gap in LBW. This result echoes the findings in the literature that income is a powerful determinant of child health, a relationship that is not completely explained by parents' own health, access to healthcare, and the environment in which the child lives (Case, Lubotsky, and Paxson 2002). Further, marital status may be linked to better pregnancy outcomes because marriage offers social support and greater financial security to the parents (Waldron, Hughes, and Brooks 1996, Raatikainen, Heiskanen, and Heinonen 2005).

This study provides the insight that SES is perhaps the single most important driver of the racial gap in LBW. Reducing the racial gaps in SES and prenatal/perinatal characteristics may be effective in narrowing the racial gap in PTB, but difference in SES remains the most important explanation for the LBW after conditioning upon full-term birth. The take-home message of this study is that policies that improve the socioeconomic status of pregnant women may be effective in reducing LBW directly and indirectly, by reducing the probability of preterm births.

There are several limitations of this analysis. Although we control for a great many correlates of infant health, we are unable to control for some important factors like family income and insurance coverage. Including more of these relevant correlates of LBW and PTB may improve the fraction of the overall difference that is explained by the model. Second, this paper does not account for the potential endogeneity of several prenatal/perinatal inputs. Results show that omitting these variables does not change the results, nonetheless extensions of this work will attempt to estimate a causal model and verify these results. This is a first attempt at quantifying the extent to which observable differences account for the racial gap in infant health and although our model includes a vast array of correlates of infant health, more than three-quarters of the racial difference in LBW and PTB remains unexplained.

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Table 1: Black-White Difference in Means, 1991 and 2001 Natality Detail Files
Means and (standard deviations in parentheses)

	White, non-Hispanic		Black, non-Hispanic		Difference
Sample size	290,615		257,131		
Dependent variables					
Birthweight (grams)	3426.50	553.29	3141.72	628.09	284.78 ***
Probability of low birthweight (<2500 g)	4.52	20.78	11.36	31.73	-6.83 ***
Probability of preterm birth (gestation < 37 weeks)	14.66	35.37	25.20	43.41	-10.54 ***
Child characteristics					
Pct girl births	48.86	49.99	49.16	49.99	-0.30 **
Pct birth order = 1	42.90	49.49	37.98	48.53	4.92 ***
Pct birth order = 2	33.92	47.34	29.37	45.55	4.55 ***
Pct birth order = 3	15.35	36.05	17.62	38.10	-2.27 ***
Pct birth order = 4	5.10	21.99	8.24	27.50	-3.15 ***
Pct birth order = 5	1.63	12.66	3.67	18.81	-2.04 ***
Pct birth order = 6	0.59	7.64	1.63	12.64	-1.04 ***
Pct birth order = 7	0.24	4.94	0.76	8.70	-0.52 ***
Pct birth order >= 8	0.27	5.20	0.72	8.47	-0.45 ***
Maternal health					
Pct mothers with no medical risk factors	98.84	10.70	99.11	9.40	-0.27 ***
Pct mothers' age 13-18 years	4.23	20.13	13.74	34.43	-9.51 ***
Pct mothers' age 19-24 years	23.14	42.17	38.13	48.57	-14.99 ***
Pct mothers' age 25-34 years	58.04	49.35	39.61	48.91	18.43 ***
Pct mothers' age >= 35 years	14.59	35.30	8.50	27.89	6.09 ***
Prenatal/perinatal characteristics					
Pct children delivered in a hospital	98.95	10.21	99.36	7.96	-0.42 ***
Pct births attended by doctor/trained midwife	99.12	9.32	99.13	9.28	-0.01
Pct prenatal care initated in 1st trimester	87.62	32.94	68.73	46.36	18.89 ***
Pct who used tobacco	15.54	32.93	12.41	29.96	3.13 ***
Pct mothers who consumed alcohol	2.13	13.04	2.25	13.71	-0.12 ***
Mean number of prenatal care visits	11.91	3.67	10.31	4.64	1.60 ***

*(**)(***) represents statistical significance at the 0.1(0.05)(0.01) level, respectively.

Table 1, continued: Black-White Difference in Means, 1991 and 2001 Natality Detail Files
Means and (standard deviations in parentheses)

	White, non-Hispanic		Black, non-Hispanic		Difference	
Socioeconomic status						
Pct mothers married	81.38	38.93	32.35	46.78	49.03	***
Pct mothers with income < $25k [2]	28.57	9.71	48.96	12.93	-20.39	***
Pct mothers with income $25k-50k [2]	31.61	5.18	29.11	3.77	2.49	***
Pct mothers with income > $50k [2]	39.82	13.78	21.93	11.06	17.90	***
Pct mothers w/ education = less than HS	1.52	12.23	2.72	16.27	-1.20	***
Pct mothers w/ education = some HS	9.51	29.33	23.72	42.54	-14.21	***
Pct mothers w/ education = HS grad	31.87	46.60	40.39	49.07	-8.52	***
Pct mothers w/ education = some college	23.95	42.68	22.39	41.69	1.56	***
Pct mothers w/ education = coll. grad or more	33.16	47.08	10.77	31.00	22.38	***
Pct mothers who did not report father info	7.74	26.73	39.48	48.88	-31.74	***
Pct fathers' age < 19 years	0.98	9.84	2.32	15.04	-1.34	***
Pct fathers' age 19-24 years	13.02	33.66	16.21	36.86	-3.19	***
Pct fathers' age 25-34 years	53.26	49.89	28.45	45.12	24.81	***
Pct fathers' age > 35 years	24.99	43.30	13.54	34.21	11.46	***
Social and Economic Environment						
Pct residing in city w/ pop < 100k	72.39	44.71	36.74	48.21	35.65	***
Pct residing in city w/ pop 100-250k	10.82	31.06	15.85	36.52	-5.03	***
Pct residing in city w/ pop 250-500k	7.20	25.85	13.51	34.18	-6.31	***
Pct residing in city w/ pop 500k-1mil.	4.66	21.08	11.52	31.92	-6.86	***
Pct residing in city w/ pop > 1million	4.93	21.65	22.38	41.68	-17.45	***
Pct residing in Northeast	24.13	42.78	20.80	40.59	3.32	***
Pct residing in Midwest	25.87	43.79	24.35	42.92	1.52	***
Pct residing in South	27.96	44.88	44.94	49.74	-16.98	***
Pct residing in West	22.05	41.46	9.91	29.88	12.14	***
Pct population of same race in county [2]	82.76	11.84	26.00	15.58	56.76	***
Age 16+ unemployment rate in county [2]	5.44	2.03	5.82	1.84	-0.38	***
Health and Healthcare Environment						
Mean no. of ob-gyns/1000 in county [2]	0.14	0.06	0.17	0.07	-0.03	***
Female mortality per 100 females in county [2]	0.86	0.20	0.72	0.17	0.14	***
Female overweight/obesity per 100 females in cou	30.27	9.52	51.37	16.84	-21.10	***
Female smokers per 100 females in county [3]	26.40	7.80	22.79	14.52	3.61	***

Notes:

1. Analysis restricted to 10% and 30% random samples of non-Hispanic, White and non-Hispanic Black singleton births, respectively, in the 1991 and 2001 Natality Detail Files.
2. Year and county specific data are obtained from the Area Resource Files.
3. Authors' calculations from the 1991 and 2001 Behavioral Risk Factor Surveillance System.
4. Medical risk factors include hemoglobinopathy, incompetent cervix, and rh sensitization.
5. *(**)(***) represents statistical significance at the 0.1(0.05)(0.01) level, respectively.

Table 2: Explained Difference in Racial Disparity in Low Birthweight and Preterm Birth Rates, Using alternate coefficients, Natality Detail Files 1991 and 2001

	Using non-Hispanic White Coefficients			Using non-Hispanic Black Coefficients		
	Prob. Of LBW	**Prob. Of PTB**	**Prob. Of LBW, Full-Term Birth Infants**	**Prob. Of LBW**	**Prob. Of PTB**	**Prob. Of LBW, Full-Term Birth Infants**
Non-Hispanic Black	0.114	0.252	0.040	0.114	0.252	0.040
Non-Hispanic White	0.045	0.147	0.015	0.045	0.147	0.015
Difference (actual gap)	0.068 ***	0.105 ***	0.026 ***	0.068 ***	0.105 ***	0.026 ***
n	547,746	547,746	440,352	547,746	547,746	440,352
Difference Explained						
Child characteristics	-0.003 ***	-0.00001	-0.0009 ***	-0.002 ***	0.002 ***	-0.0004 ***
	(0.0002)	(0.0002)	(0.0002)	(0.0002)	(0.0002)	(0.0002)
	-4.5%	-0.01%	-3.5%	-2.7%	2.3%	-1.7%
Maternal health	-0.005 ***	-0.005 ***	-0.001 ***	-0.007 ***	-0.004 ***	-0.002 ***
	(0.0005)	(0.0006)	(0.0003)	(0.0005)	(0.0006)	(0.0003)
	-7.4%	-4.8%	-5.7%	-10.9%	-4.2%	-7.2%
Prenatal/perinatal characteristics	0.009 ***	0.013 ***	0.001 ***	0.013 ***	0.016 ***	0.001 ***
	(0.0005)	(0.0006)	(0.0003)	(0.0004)	(0.0005)	(0.0003)
	13.4%	12.4%	4.5%	19.1%	15.3%	5.9%
Socioeconomic status	0.015 ***	0.020 ***	0.008 ***	0.013 ***	0.016 ***	0.008 ***
	(0.002)	(0.003)	(0.002)	(0.002)	(0.002)	(0.001)
	21.4%	19.0%	31.1%	19.3%	15.1%	30.1%
Social & Economic Environment	0.003	-0.001	0.003	-0.006	-0.004	-0.002
	(0.003)	(0.004)	(0.002)	(0.004)	(0.005)	(0.003)
	3.9%	-1.3%	9.9%	-8.1%	-3.7%	-7.1%
Health & Healthcare Environment	0.0002	0.002	-0.0004	-0.002 *	-0.004 **	-0.002 *
	(0.001)	(0.002)	(0.001)	(0.001)	(0.002)	(0.001)
	0.2%	2.2%	-1.8%	-3.3%	-3.7%	-6.1%
Total	0.019	0.029	0.009	0.009	0.022	0.004
	27.2%	27.5%	34.5%	13.3%	21.1%	13.9%

Notes:

1. Analysis restricted to 10% and 30% random samples of non-Hispanic, White and non-Hispanic Black singleton births, respectively, in the 1991 and 2001 Natality Detail Files.

2. LBW: birthweight < 2500 grams; PTB: gestation <= 37 weeks

3. Black and white coefficients obtained from Probit estimation of LBW and PTB equations for black and white infants, respectively.

4. Total percent explained sums the percent explained by each set of explanatory variables.

5. *(**)(***) represents statistical significance at the 0.1(0.05)(0.01) level, respectively.

Table 3: Explained Difference in Racial Disparity in Low Birthweight and Preterm Birth Rates when Endogenous Variables are Omitted, Using Alternate Coefficients, Natality Detail Files 1991 and 2001

	Using Non-Hispanic White Coefficients			Using Non-Hispanic Black Coefficients		
	Prob. Of LBW	Prob. Of PTB	Prob. Of LBW, Full-Term Birth Infants	Prob. Of LBW	Prob. Of PTB	Prob. Of LBW, Full-Term Birth Infants
Sample means						
Non-Hispanic Black	0.114	0.252	0.040	0.114	0.252	0.040
Non-Hispanic White	0.045	0.147	0.015	0.045	0.147	0.015
Difference (actual gap)	0.068 ***	0.105 ***	0.026 ***	0.068 ***	0.105 ***	0.026 ***
n	547,746	547,746	440,352	547,746	547,746	440,352
Difference Explained						
Child characteristics	-0.002 ***	0.0007 ***	-0.0005 ***	0.001 ***	0.005 ***	0.0003 **
	(0.0002)	(0.0002)	(0.0002)	(0.0002)	(0.0002)	(0.0001)
	-2.5%	0.7%	-1.8%	1.0%	4.3%	1.1%
Maternal health	-0.005 ***	-0.005 ***	-0.002 ***	-0.008 ***	-0.004 ***	-0.003 ***
	(0.0005)	(0.0006)	(0.0004)	(0.0005)	(0.0006)	(0.0003)
	-7.7%	-4.4%	-7.5%	-12.0%	-3.7%	-10.3%
Prenatal/perinatal characteristics	-	-	-	-	-	-
Socioeconomic status	0.026 ***	0.031 ***	0.013 ***	0.026 ***	0.029 ***	0.012 ***
	(0.003)	(0.003)	(0.002)	(0.002)	(0.002)	(0.001)
	37.4%	29.0%	50.0%	37.6%	27.1%	46.5%
Social & economic environment	0.0003	-0.003	0.002	-0.004	-0.003	-0.001
	(0.003)	(0.004)	(0.002)	(0.004)	(0.005)	(0.003)
	0.4%	-3.1%	6.2%	-6.4%	-3.0%	-5.5%
Health & healthcare environment	0.001	0.004 **	-0.0004	-0.005 ***	-0.006 ***	-0.002 ***
	(0.001)	(0.002)	(0.001)	(0.001)	(0.002)	(0.001)
	1.9%	4.1%	-1.6%	-7.0%	-6.0%	-9.4%
Total	0.020	0.028	0.012	0.009	0.020	0.006
	29.4%	26.3%	45.4%	13.3%	18.6%	22.4%

Notes:

1. Analysis restricted to 10% and 30% random samples of non-Hispanic, White and non-Hispanic Black singleton births, respectively, in the 1991 and 2001 Natality Detail Files.
2. LBW: birthweight < 2500 grams; PTB: gestation <= 37 weeks
3. Black and white coefficients obtained from Probit estimation of LBW and PTB equations for black and white infants, respectively.
4. Total percent explained sums the percent explained by each set of explanatory variables.
5. *(**)(***) represents statistical significance at the 0.1(0.05)(0.01) level, respectively.

Table 4: Explained Difference in Racial Disparity in Low Birthweight and Preterm Birth Rates, by Year, Natality Detail Files 1991 and 2001

	1991			2001		
	Prob. Of LBW	Prob. Of PTB	Prob. Of LBW, Full-Term Birth Infants	Prob. Of LBW	Prob. Of PTB	Prob. Of LBW, Full-Term Birth Infants
Non-Hispanic Black	0.119	0.257	0.044	0.108	0.247	0.036
Non-Hispanic White	0.044	0.133	0.015	0.047	0.161	0.014
Difference (actual gap)	0.075 ***	0.124 ***	0.029 ***	0.061 ***	0.086 ***	0.022 ***
n	281,758	281,758	228,062	265,988	265,988	212,290
Difference Explained						
Child characteristics	-0.004 ***	-0.0009 **	-0.0010 ***	-0.002 ***	0.001 *	-0.0005 *
	(0.0004)	(0.0004)	(0.0002)	(0.0003)	(0.0002)	(0.0003)
	-5.1%	-0.7%	-3.6%	-3.7%	0.6%	-2.3%
Maternal health	-0.005 ***	-0.004 ***	-0.002 ***	-0.005 ***	-0.007 ***	-0.001 **
	(0.0007)	(0.0009)	(0.0003)	(0.0007)	(0.0006)	(0.0005)
	-7.0%	-3.1%	-5.5%	-8.3%	-8.1%	-5.9%
Prenatal/perinatal characteristics	0.013 ***	0.018 ***	0.002 ***	0.005 ***	0.009 ***	0.000
	(0.0009)	(0.0009)	(0.0003)	(0.0005)	(0.0005)	(0.0003)
	17.0%	14.8%	7.3%	8.8%	10.1%	0.3%
Socioeconomic status	0.016 ***	0.028 ***	0.008 ***	0.021 ***	0.026 ***	0.010 ***
	(0.004)	(0.005)	(0.002)	(0.004)	(0.002)	(0.003)
	21.4%	22.7%	29.0%	34.8%	30.7%	46.7%
Social & economic environment	0.002	0.004	0.001	0.004	-0.003	0.004
	(0.004)	(0.006)	(0.002)	(0.004)	(0.005)	(0.003)
	3.2%	2.9%	2.7%	5.9%	-3.4%	19.5%
Health & healthcare environment	-0.0021	-0.003	-0.0021 *	0.002	0.008 **	0.002
	(0.002)	(0.003)	(0.001)	(0.002)	(0.002)	(0.002)
	-2.8%	-2.5%	-7.2%	4.0%	9.1%	6.9%
Total	0.020	0.042	0.007	0.026	0.033	0.014
	26.7%	34.0%	22.8%	41.5%	39.0%	65.2%

Notes:

1. Analysis restricted to 10% and 30% random samples of non-Hispanic, White and non-Hispanic Black singleton births, respectively, in the 1991 and 2001 Natality Detail Files.

2. LBW: birthweight < 2500 grams; PTB: gestation <= 37 weeks

3. Decomposition results based on regression coefficients for non-Hispanic white mothers obtained from Probit estimation

4. Total percent explained sums the percent explained by each set of explanatory variables.

5. *(**)(***) represents statistical significance at the 0.1(0.05)(0.01) level, respectively.

Table 5: Explained Difference in Racial Disparity in Low Birthweight and Preterm Birth Rates, Including County Fixed Effects, Natality Detail Files 1991

	Prob. Of Low Birthweight		Prob. Of Pre-term Birth		Prob. Of Low Birthweight, Full-Term Birth Infants	
Non-Hispanic Black	0.114		0.252		0.040	
Non-Hispanic White	0.045		0.147		0.015	
Difference (actual gap)	0.068	***	0.105	***	0.026	***
n	547,746		547,746		440,352	
Difference Explained						
Child characteristics	-0.001	***	0.000		0.000	**
	(0.0001)		(0.0002)		(0.0001)	
	-2.2%		0.3%		-1.2%	
Maternal health	-0.005	***	-0.005	***	-0.001	***
	(0.0004)		(0.0006)		(0.0002)	
	-7.3%		-4.8%		-5.8%	
Prenatal/perinatal characteristics	0.007	***	0.012	***	0.001	***
	(0.0003)		(0.0005)		(0.0002)	
	10.2%		11.5%		4.7%	
Socioeconomic status	0.014	***	0.015	***	0.006	***
	(0.001)		(0.001)		(0.000)	
	20.4%		14.4%		23.3%	
Total	0.014		0.023		0.005	
	21.1%		21.4%		20.9%	

Notes:

1. Analysis restricted to 10% and 30% random samples of non-Hispanic, White and non-Hispanic Black singleton births, respectively, in the 1991 and 2001 Natality Detail Files.
2. LBW: birthweight < 2500 grams; PTB: gestation <= 37 weeks
3. Decomposition results based on regression coefficients for non-Hispanic white mothers obtained from OLS estimation
4. Total percent explained sums the percent explained by each set of explanatory variables.
5. *(**)(***) represents statistical significance at the 0.1(0.05)(0.01) level,