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Explaining Racial Disparities in Infant Health in Brazil

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Abstract

Objective—Racial disparities in infant health are common in Brazil. Explaining these disparities and identifying the pathways through which they arise is essential for identifying risk factors that can be targeted by policy interventions. Our objective is to quantify the extent to which socioeconomic, healthcare, demographic, and geographic effects explain racial disparities in low birth weight (LBW) and preterm birth (PTB) rates in Brazil.

Methods—We employ a sample of 8949 infants born between 1995 and 2009 in 15 cities and 7 provinces in Brazil. We focus on disparities in LBW (< 2500 grams) and PTB (< 37 gestational weeks) prevalence between infants of African ancestry alone or mixed with other ancestries on one side, and infants of European ancestry alone on the other. We quantify the contributions of several conceptually relevant factors to these disparities using a decomposition model.

Findings—The model explains 45-94% and 64-94% of the LBW and PTB disparities, respectively, between the various African ancestry groups and European ancestry. Differences in prenatal care use and geographic location are generally the most important contributors to these disparities, followed by socioeconomic differences. The model explains the majority of the disparities for mixed African ancestry and part of the disparity for African ancestry alone.

Conclusions—Prenatal care and geographic location differences explain a large portion of the health disparities between infants of African and European ancestries. Public policies aiming at improving child health should target these pathways in order to reduce such disparities.

Keywords

Racial disparities; child health; low birth weight; preterm birth; Brazil

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Introduction

Large health disparities exist between black and white infants in Brazil. (1-4) Infant mortality is more than twice as common among black as white infants in Southern Brazil (30.4 versus 13.9 per 1000). (5) Poor birth outcomes including low birth weight (LBW) and preterm birth (PTB) are also more common among black infants. Racial disparities are also reported in prenatal and postnatal care, with white mothers having more and higher quality prenatal visits and greater use of postnatal care. (6, 7)

Documenting the prevalence and magnitude of racial disparities in infant/child health is important. However, of more importance is explaining these disparities and identifying the pathways through which they arise in order to identify contributors that can be targeted by policy interventions. Such effort has life-long implications given the importance of child health for adult health and human capital attainment. (8-12) Since child health may have multiplicative effects on health over life, early health disparities may extend into large health and human capital disparities later in life. (13, 14)

Studies in the United States (US) have shed light on several pathways leading to racial disparities in infant/child health. (15-21) Individual-level factors including socioeconomic status (SES), (22) maternal age, prenatal care use (22-24), and stressful life events before delivery (17) as well as differences in health care access and quality (25-27) and social inequalities due to residential segregation and poverty (15, 28, 29) are thought to be important contributors to racial disparities in infant health in the US.

To our knowledge, there are no studies that simultaneously quantify the contributions of a large number of conceptually relevant factors to racial disparities in infant health in Brazil. In this study, we examine the extent to which socioeconomic, healthcare, demographic, and geographic effects explain disparities in LBW (< 2500 grams) and PTB (< 37 gestational weeks) rates by African ancestry in Brazil. Unlike any previous study for Brazil, we evaluate the contributions of the explanatory factors to the disparities by different degrees of African ancestry. We focus on disparities by African ancestry since they are the most prevalent and affect a large percentage of the Brazilian population. (1-5)

Our study is the first to simultaneously quantify the contributions of several factors both as a group and each on its own (while controlling for the others) to explaining racial disparities in infant health in Brazil. Such a study is needed not only because Brazil is the largest country in South America but because there are many historical, demographic, economic, social, cultural, and healthcare system differences between Brazil and other racially admixed countries such as the US. These differences limit the generalizability of studies of racial infant health disparities in the US to the Brazilian population as these differences may modify the underlying factors and the extent of their contributions to the disparities.

There is a sharp contrast in perception of race between Brazil and the US. (30) Race for individuals of African and/or European ancestry in Brazil has been historically and socially defined on a “continuum” of skin color including black, brown (mixed between black and white), or white, instead of the black or white color line as in the US. This is in part due to the large racial admixing in Brazil. (31) The difference in perceptions of racial identity between Brazil and the US implies potential differences in cultural and socioeconomic factors related to race and how these may affect health and contribute to racial disparities. Brazil also differs significantly in its economic growth and extent of economic disparities by race from the US. (32, 33) Finally, there are major differences in access to and quality of healthcare between Brazil and the US. (34, 35) For all of these reasons, a study that explains the racial disparities in LBW/PTB in Brazil is needed to draw inference that can help to inform policy making and interventions to reduce these disparities in that country.

Methods

Data Sample

We employ a unique sample of 8,949 singleton live births between 1995 and 2009 in 7 provinces, 15 cities and 25 hospitals in Brazil. The sample is identified by the Latin American Collaborative Study of Congenital Malformations (ECLAMC). ECLAMC is an epidemiological research and surveillance program for birth defects in South America. (36, 37) ECLAMC involves a voluntary collaboration with a network of hospitals and health professionals (mostly pediatricians). The health professionals enroll into ECLAMC infants born in their hospitals with and without birth defects before discharge after birth. For each infant with a birth defect, ECLAMC professionals match an unaffected infant by birth date, sex, and hospital of birth. All infants are recruited using the same criteria and data are systematically collected using the same questionnaires across all affiliated hospitals.

ECLAMC professionals obtain data on infant health, prenatal history and several demographic and socioeconomic characteristics by interviewing mothers before discharge and abstraction of hospital records as needed. ECLAMC professionals receive the same standard training before initiating data collection and attend annual group meetings at ECLAMC, which is expected to enhance the quality and consistency of data collection. ECLAMC's data have been used in several previous studies of infant health. (37-40) A detailed description of ECLAMC is available elsewhere. (36)

We only include infants without birth defects who represent the majority of the infant population as birth defects increase LBW and PTB risks (38, 41) and may modify the underlying etiology of racial disparities. Even though our study sample is not randomly selected from the total birth population, there are several factors that suggest that it is representative of a large proportion of the population. Since there are no inclusion criteria into ECLAMC for unaffected infants that are related to infant health (LBW and PTB) and the study explanatory variables, the sample is unlikely to be biased. Even though infants without birth defects enrolled in ECLAMC are matched to the affected infants by sex and birthdate, birth defects are not related to birthdate and only few of them vary slightly by sex. Indeed, the male-to-female ratio in our study sample is close to that of the Brazilian birth population (1.2 versus 1.05). (32) Also, ECLAMC has a high infant-participation rate, with about 95% of infants without birth defects identified to participate enrolling in the program. (42) Furthermore, there are no barriers or inclusion/exclusion criteria for hospitals and pediatricians to join ECLAMC, which is built on a voluntary participation model. Also, ECLAMC hospitals serve geographically and socioeconomically diverse communities as reflected below in the sample's variation of these characteristics, which further enhances the sample's representativeness and generalizability of results.

We limit the sample to infants with birth weights between 500 and 6,000 grams and gestational ages between 19.5 and 46.5 weeks. These restrictions are standard in this literature in order to avoid data recording errors (most babies below the minimum thresholds are stillbirths). This results in 10,777 infants out of 10928 initial observations. The 8,949 infants included in our analysis are those with no missing values for any of the variables used.

Study measures and empirical model

Similar to the US, race is a social construct in Brazil. However, measuring race is complicated particularly in Brazil due to the large admixture of multiple ancestries. (31) The fact that race is perceived in Brazil on a color continuum instead of on the white-black line such as in the US and the lack of clearly defined racial color lines makes racial identification flexible and varying. (30, 43) As a result, race in Brazil is arguably more accurately

measured based on self-report since such a measure will reflect the perceived social identity of the individual. (30, 44, 45) The Brazilian census asks individuals to identify their race under one of the following categories of skin color: black, white, brown, yellow, or indigenous. (33) Since we focus on disparities between black and white infants, the three categories of a skin-color based measure of race that are most related to our analysis are white, brown, and black.

ECLAMC does not ask about skin color, but obtains a related measure which is ethnic ancestry. Mothers were asked to report all the ethnic ancestries of the child including mainly African, European, Native, and other minority groups. Therefore, multiple ancestries are reported for children who have admixed race/ethnicity. This measure allows for creating an ethnic ancestry continuum to represent different racial/ethnic groups.

We study three groups of African ancestries – African only (AO), African-European (AE), and African-non-European (ANE) – and a group of European only (EO) ancestry. AO indicates that the mother reported that the child has African ancestry without any other ancestry. While we do not observe a skin-color based measure of race, the AO group is expected to mostly include individuals who would self-identify as black. AE indicates that both African and European ancestries were reported but no other ancestries. ANE indicates that both African and other non-European ancestries were reported, but no European ancestry. Both the AE and ANE groups are expected to mostly include individuals who would self-identify as brown based on the skin-color race measure used in the Brazilian census. The EO group includes children for whom only European ancestry is reported and is expected to mostly include individuals who would self-identify as white. Therefore, our measure of ethnic ancestry is consistent with perceptions of race in Brazil and accommodates the flexibility of its racial identity continuum. This measure has been used in several previous studies of infant and maternal health in Brazil including studies of racial disparities. (34, 41, 46, 47) We separately compare each of the three African-ancestry groups to the EO group because the contributions of the evaluated explanatory variables to racial disparities may vary between these groups.

We employ a multivariate model for infant health that includes several theoretically relevant demographic, socioeconomic, healthcare, and geographic effects. The underlying pathways for disparities are complex and multi-level including both individual- and geographic-level effects. (16, 48-50) Recognizing this complexity is essential for explaining disparities. Given that our goal is to simultaneously quantify the extent to which several variables explain the observed racial disparities and to explain as much of these disparities as possible, we do not limit our conceptual framework to a single theory for health determinants or disparities. Relying on a single existing theory to specify our model significantly hinders our study goal. Instead, our selection of explanatory variables is motivated by several theories for determinants of health and racial disparities and by results from previous studies that have highlighted an important role for these variables. We appeal to general microeconomic and psychosocial theories that highlight the importance of education, prenatal care, geographic location/residential segregation, and other factors for child health and racial disparities (16, 22, 48, 49, 51-55) and to previous studies (22, 37, 38, 41, 56-62) when possible for selecting conceptually relevant explanatory variables. We choose the following model:

$$H_{i,y=1,2}=\alpha_{0y}+\beta_y Ancestry_i+\beta_y PNC_i+\beta_y Dem_i+\beta_y Health_i+\beta_y Fertility_i+\beta_y SES_i+\beta_y Area_i+u_{yi} \quad (1)$$

where for child i, H is health measured by either LBW (y=1) or PTB (y=2) and is a function of child ancestry (*Ancestry*), prenatal care (*PNC*), demographic characteristics (*Dem*),

maternal health (*Health*), maternal fertility (*Fertility*), socioeconomic status (*SES*) and geographic effects (*Area*); u is the error term.

Prenatal care is measured by the number of prenatal visits. Several studies highlight the importance of prenatal care for LBW and PTB. (37, 41, 57, 58, 63) Notable disparities in the number and quality of prenatal care visits exist between white and black/mixed race women in Brazil. (5, 6, 64, 65) Demographic characteristics include a binary indicator for infant sex, continuous maternal and paternal age variables, and age squared. In addition to their direct effects on infant health (especially in the case of infant's sex and maternal age), these characteristics also influence parental health preferences and behaviors. (12, 66) Since we do not have a direct measure of marital status, which is relevant for LBW and PTB, (67) we include length of parents' cohabitation before child's birth as a proxy similar to previous studies. (68) A number of studies have found that cohabitation status (60, 62) and cohabitation length (61) are associated with a decrease in LBW and fetal death in Brazil. Maternal health and fertility history are relevant for LBW and PTB and affect maternal health behaviors as shown in several previous studies. (35, 39, 41, 47, 69) These are measured by indicators for acute and chronic illnesses during pregnancy, history of conception difficulty, and numbers of previous live births and miscarriages/stillbirths.

We also include family *SES* measured by mother's and father's education and employment/occupational status. Parental education and socioeconomic status may affect infant health in several ways including by increasing the health benefit that the mother obtains from prenatal care through enhanced information processing and greater compliance with treatment plans, improving psychosocial status and social networking, improving maternal health and health behavior, and increasing access to health care. (12, 55) Many studies report a positive association between maternal education and infant health in Brazil. (41, 51, 70, 71) Maternal occupation may also influence infant health through several indirect pathways such as by affecting income and maternal time for health investments, but also through occupational/environmental exposures. (72-75)

We also include geographic location represented by binary indicators for the city of child's birth in order to evaluate the contribution of geographic effects to racial disparities in infant health, which may result from differences in residential distribution by race and geographic variation in healthcare availability and quality, economic growth, and social infrastructure. Residential segregation may affect health by reducing access to social, economic, healthcare, and environmental resources needed for maternal and infant health. (76, 77) Several studies have highlighted adverse consequences for infant health from reductions in the quality of the physical and socioeconomic environments in Brazil including from pollution, (78) poverty concentration, (79) and residence in the Northeast region. (80-82)

We estimate equation (1) using logistic regression separately for each of the three African ancestry groups described above compared to EO infants. We adjust the standard errors for non-independence across the city of birth using a Huber-type robust variance estimator. (83) We also estimate a nested-specification of equation (1) that only includes the ancestry indicator in order to evaluate the total racial disparity in LBW/PTB.

Disparity decomposition

While comparing the ancestry effects on infant health between the full and nested specifications for equation (1) allows for evaluating the extent to which all model variables as a group explain the racial disparities, this comparison does not quantify the individual variable contributions. Such decomposition is needed to identify the factors that are most relevant for explaining these disparities. We employ the Fairlie decomposition model (84) in order to quantify the contributions of the model explanatory variables to the racial disparities

in LBW/PTB. This model is an extension of the Oaxaca-Blinder decomposition model to non-linear models for binary outcomes and has been successfully applied in previous studies. (56, 85-87) The model identifies the extent to which differences in a particular characteristic between two groups explains the difference in their outcomes, and has been previously used to explain racial health disparities in other contexts. (34, 56)

For each racial comparison (e.g., AO versus EO), the model first estimates equation (1). Since the sample sizes for the two ancestry groups are different, the model randomly selects a subsample from the majority group equal in size to the minority group. From equation (1), outcome (e.g. LBW) probabilities are predicted for each observation in the minority sample and majority subsample. Within each group, the observations are ranked by their probability, and the observations are then matched one-to-one between the two groups by their rank. One at a time for each explanatory variable in equation (1), the model substitutes the variable value of each observation in the minority group by that of the matched observation from the majority subsample. Using equation (2) below, the model then estimates the contribution (C) of variable k to the outcome difference between the two groups as follows:

$$C_k = \frac{1}{N^M} \sum_{i=1}^{N^M} F\left(a_0 + \sum_{j=1}^{k-1} \beta_j X_{ij}^M + \beta_k X_{ik}^O + \sum_{j=k+1}^{K} \beta_j X_{ij}^O\right) - F\left(a_0 + \sum_{j=1}^{k-1} \beta_j X_{ij}^M + \beta_k X_{ik}^M + \sum_{j=k+1}^{K} \beta_j X_{ij}^O\right) \quad (2)$$

where M and O indicate minority and majority groups, respectively, j indicates the variable order (1 to K), N^M is the number of individuals in the minority group, and F is the cumulative density function. The model is repeated for all variables in the model (last evaluated variable is of order K).

We first decompose the LBW/PTB disparities over categories of conceptually related variables as defined in equation (1) using the same steps listed above. The categories of variables (instead of individual variables) are ordered and the values of all variables within the same category are switched simultaneously between the majority and minority observations. Then, we repeat the decomposition over each variable (instead of over variable categories) to identify variables within categories that are most relevant for these disparities.

Since results may change with the particular selected majority subsample, we perform 2,000 random subsample selections and average the results across these replications. (84) Also, since the variable (or category) order j in the model could affect results, we randomly select this order at the time of majority subsample selection, which provides an approximation of all possible orders.

Results

Table 1 shows the variable distributions in the study sample. About 9%, 51%, and 17% are AO, AE and ANE, respectively. The LBW and PTB rates are 12.4% and 18.9%, respectively, for infants of any African ancestry compared to 8.1% and 15% for EO infants. LBW and PTB rates are overall comparable between the three African ancestry groups. There are several differences in the explanatory variables between the ancestry groups. The average number of prenatal visits is 5.9, 6.5, 7.0, and 6.8 among AO, AE, ANE and EO ancestries, respectively. Conception difficulty and number of previous live births are highest among AO ancestry, while the rate of chronic illness is highest among ANE ancestry. Educational attainment is highest among EO ancestry.

Table 2 reports the unadjusted and adjusted odds ratios (OR) for ancestry effects on LBW and PTB from equation (1). When unadjusted, African ancestry (alone or mixed) significantly increases LBW and PTB risks by about 1.6-1.7 and 1.3-1.4 times, respectively.

When adjusted for all explanatory variables in Table 1, the effects of African ancestry become small and insignificant (OR=1.0-1.3).

Tables 3 and 4 show the results from decomposing the LBW and PTB disparities, respectively, in relation to the explanatory variable categories. Panel A reports the difference in LBW or PTB rate by ancestry and the difference jointly explained by all model variables. Panel B reports the difference in LBW or PTB rate by ancestry that is independently explained by each category of the study variables. Figures 1 and 2 show the percentages of the LBW and PTB disparities that are significantly explained by the study variable categories. Variable categories that do not explain these disparities are not shown in these figures.

The study variables explain a large percentage of the LBW/PTB disparities between African and EO ancestries, ranging from 44.6% of the LBW gap for AO to 93.9% of the PTB gap for AE ancestry. Geographic effects are most relevant for explaining disparities for the mixed African ancestries, explaining 70-80% of the gaps. Prenatal care is the only relevant variable for explaining disparities for AO ancestry, explaining 37-63% of the gaps and second most relevant for AE ancestry. We further describe these results below and highlight the most relevant variables within each category (detailed results for individual variable contributions available from the authors).

AO versus EO

As shown in Tables 3 and 4, the model variables explain 44.6% and 64.2% of the LBW and PTB gaps, respectively, between AO and EO ancestries. Most of the explained gaps (about 37.1% and 63.1% of the LBW and PTB gaps, respectively) are accounted for by the lower number of prenatal visits for AO ancestry. None of the other variable categories has significant effects on these disparities.

Mixed African ancestries versus EO

The model variables explain 93.6% and 72.6% of the LBW gap for AE and ANE versus EO ancestry, respectively (see Tables 3 & 4). Differences in geographic location explain about 84.0% and 70.1% of these disparities, respectively. Differences in the number of prenatal visits explain 13.2% of the LBW gap for AE ancestry. SES differences explain 6.8% and 9.2% of the LBW disparities for AE and ANE ancestries, respectively, with parental occupation accounting for most of these effects. Differences in household demographics, mainly parental age, explain 8.5% of the LBW disparity for ANE ancestry. In the ANE group, the average number of visits is larger than in the EO group, suggesting that the LBW disparity would have been larger if prenatal visits were lower. Similarly, the significantly lower rates of acute illnesses during pregnancy in the AE group compared to the EO group reduced the AE LBW disparity, which would have been otherwise larger.

The model variables also explain most of the PTB gaps – 93.9% and 74.8% for AE and ANE ancestries, respectively. Geographic effects are also the most relevant, explaining 79.5% and 76.1% of these disparities for AE and ANE ancestries, respectively. Differences in prenatal visits explain 27.4% of the PTB gap for the AE ancestry. Differences in household demographics (mainly maternal age) explain 4.9% of the PTB gap for ANE ancestry. Similar to the LBW disparities, the ANE and AE PTB disparities would have been larger if the ANE group had a similar or lower average of prenatal visits and the AE group had similar or higher rates of acute illnesses compared to the EO group.

Discussion

In Brazil, LBW and PTB rates are significantly higher among infants of African ancestry alone or mixed with other ancestries than those of EO ancestry. The disparities we find are consistent with those from other studies in Brazil. (35, 65) For example, Barros et al. report a 14-24% increased likelihood of LBW and PTB among non-white infants compared to white infants in Southern Brazil. (35) However, our study is the first to formally decompose these disparities in Brazil and quantify how they relate to demographic, socioeconomic, healthcare, and geographic differences. We find that the model variables explain a significant portion of LBW (about 45%) and PTB (64%) disparities for infants with AO and most of the disparities for infants with mixed African ancestries. These findings suggest that racial disparities in infant health in Brazil are mainly socially and economically driven and are amenable to policy interventions that address these pathways. Our model notably explains more of these gaps than a recent study of LBW and PTB disparities between black and white infants in the US using a similar approach which only explained close to a third of the LBW (27.2%) and PTB (27.5%) gaps. (22) This highlights the importance of population-specific studies and that US-based studies of racial disparities may not generalize to the Brazilian population.

Geographic, prenatal care and socioeconomic differences are the most relevant factors for explaining these disparities. Improving access to prenatal care especially for AO mothers may significantly reduce racial disparities in infant health in Brazil. In our sample, mothers of AO ancestry use one less prenatal visit on average than those of EO ancestry. One reason why SES differences do not significantly explain racial disparities between AO and EO ancestries is that they are strongly predictive of prenatal care use differences between these groups and may be influencing disparities through prenatal care. In an additional model, we decompose the disparities between AO and EO ancestries excluding prenatal visits from the model and find SES to significantly explain 53% and 26% of the disparities in LBW and PTB respectively. This suggests that the disparities explained by prenatal care differences are in part driven by SES differences. In the recent US-based study mentioned above, SES explained 21.4% and 19% of the LBW and PTB gaps, respectively, between black and white infants, and prenatal care explained 13.4% and 12.4% of the LBW and PTB disparities, respectively. In contrast, prenatal care explains a much larger part of the gaps in our study especially for infants of AO ancestry (37.1% and 63.1% of the LBW and PTB gaps, respectively), and SES explains a smaller part of the gap than that study. Again, these results highlight the need for population-specific studies of racial disparities.

While the university graduation rate is low in the study sample, this rate is significantly lower among individuals of African ancestry especially AO (less than 1 percent). Further, unemployment and low-skill occupations are significantly more common among mothers of African ancestry (about 20% compared to 13% among EO ancestry). These sample-based differences are consistent with population-level differences. (33) Therefore, economic and educational policies that improve the human capital and socioeconomic status for the whole Brazilian population may reduce the observed LBW and PTB disparities.

The observed geographic effects suggest: 1) significant racial differences in geographic location and 2) large geographic differences in LBW and PTB prevalence. Differences in geographic location by race can be clearly seen by the sample's ancestry distributions across the study provinces as shown in supplementary Figure S1 and are supported by previous studies documenting large racial residential segregation in Brazil. (31) Also, supplementary Figure S2 shows significant variation in the sample LBW and PTB rates across the study provinces. Racial residential segregation correlates with poverty concentration in certain geographic locations in Brazil. (88, 89) Geographic differences in LBW/PTB may arise

from differences in access to healthcare and social and economic resources (social support, safety, healthy food outlets) that are important for maternal and infant health. (76, 77) There are many ways through which racial differences in residential location can lead to racial disparities in infant health including by restricting access to such important resources as previously shown in several studies in the US. (15, 90-92) We cannot identify the specific factors that contribute to geographic differences in LBW/PTB in this study. However, the results suggest that policies that aim at eliminating the underlying causes for racial residential segregation may reduce racial disparities in infant health in Brazil.

The model explains less of the disparity for AO than for mixed African ancestries. This suggests potential differences in the underlying pathways for disparities between these groups. Since geographic location is more similar between AO and EO ancestries than between AE or ANE and EO as shown in supplementary Figure 1, geographic effects are important for explaining the disparities for mixed African ancestries but not for AO ancestry. Also, the lower SES for AO compared to mixed African ancestry may increase the relative influence of individual- versus geographic-level factors on AO disparities. The results highlight the importance of further research to evaluate the role of other factors not included in our model that may be contributing to the disparities for AO infants. Furthermore, our model explains a larger portion of the disparities for AE than ANE. This may suggest greater similarity in unmeasured relevant characteristics for infant health between AE and EO ancestries such as cultural factors, which increases the explanatory power of the model variables.

Our study highlights the importance of studying racial disparities in health using population-specific data. As mentioned above, there are important social, economic, and healthcare system differences between Brazil and the US. Among these is the difference in perception of racial identity between the two countries as discussed above. The perception of race along a color continuum in Brazil is a social phenomenon that has historically existed for over five hundred years during the colonial period and before the abolishment of slavery and is widely recognized by all Brazilians. (30, 31, 43, 93) Furthermore, Brazil has one of the most racially admixed populations worldwide, (31) and the percentage of the population mixed between white and black has increased from 21.2% in 1940 to 38.5% in 2000. (32) Also, there are distinct historical/political differences between Brazil and the US related to race. For example, after the abolition of slavery in Brazil there were no laws that instituted racial segregation as in the US. (30) In addition, there are major economic differences between the two countries. Average income per capita in Brazil in 2010 was only 22.7% of that in the US ($10710 versus $47153). (35) Furthermore, income disparity by race is larger in Brazil than the US. For example, average family income of the black and brown population in Brazil in 2006 was 44% of that of whites. (33) In contrast, average household income of blacks in the US in 2006 was 63% of that of whites. (94) Also, large racial disparities exist in private health insurance in Brazil, (34) which unlike the US, does not have a public health insurance system aimed at covering less affluent mothers and children. Additionally, the capacity of the healthcare system in Brazil is markedly lower than that of the US. (95) All these differences suggest that there may be major limitations to generalizing findings from studies on racial disparities across countries

There are some limitations to this study. We are unable to evaluate the effects of other conceptually relevant variables for LBW and PTB such as insurance status, diet, exercise, stress, smoking, substance use, maternal weight gain, quality of prenatal care and cultural factors. (66, 96-100) This is especially important for further explaining the disparities between AO and EO ancestries and for understanding the socioeconomic and behavioral pathways leading to disparities. In a separate model, we evaluated the effect of medication use but found overall that it does little in explaining any of the disparities (detailed results

available from the authors). A related limitation is that we do not have intergenerational data that enables exploring the root causes of some of the maternal and prenatal factors relevant to infant health disparities such as the importance of the mother's own growing environment for her educational attainment and health behavior later in life. Also, we are unable to explain the pathways through which geographic location explains the LBW disparities for mixed African ancestries due to the lack of data on geographic-level characteristics. As in any self-reported measure, our measure of ethnic ancestry may involve some errors such as in cases when the mother or father does not know their complete family ancestry. However, we expect such errors to be minimal, especially since race in Brazil is strongly linked to skin-color, which in turn is related to ethnic ancestry. Also, the number of prenatal visits was capped at 9 (for visits greater than 9) during data entry in certain years. This is not expected to bias the contribution of prenatal visits to explaining the LBW or PTB gap but to inflate its variance, which is of minimal consequence since the contribution is significant. Finally, even though our sample is socioeconomically and geographic diverse, it is not randomly selected and may not be fully representative of the entire birth population.

Yet our study has several strengths including a large diverse sample, a measure of ethnic ancestry that reflects the perception of race along a continuum in Brazil and accommodates the large ancestry admixture, detailed and consistently collected data across multiple sites in Brazil, and an approach that quantifies the contribution of multiple variables both as a group and individually while controlling for the other variables to racial disparities. Our study offers insights for several future studies. First, studies using intergenerational data are needed to identify earlier causes of infant health disparities and to explain the effects of proximal factors such as maternal education, health behavior, and geographic location. Similarly, studies that evaluate the contributions of additional maternal healthcare and behavioral characteristics not measured in our study such as insurance status, smoking, alcohol use, and diet and specific area-level characteristics such as number of healthcare providers, quality of prenatal care, and neighborhood wealth and safety indicators are needed to evaluate their contributions to infant health disparities. Also, examining more detailed measures of race and ethnic ancestry is important in order to more fully capture the subtleties of racial perceptions in Brazil. Finally, it is important to replicate our study using a population-based nationally representative sample from Brazil.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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References

1. Wood CH, Lovell PA. Racial Inequality and Child Mortality in Brazil. Social Forces. 1992; 70(3): 703–724.
2. Victora CG, Vaughan JP, Barros FC, Silva AC, Tomasi E. Explaining trends in inequities: evidence from Brazilian child health studies. The Lancet. 2000; 356(9235):1093–1098.
3. Cardoso AM, Santos RV, Coimbra CE Jr. Infant mortality according to race/color in Brazil: what do the national databases say? Cad Saude Publica. 2005; 21(5):1602–8. [PubMed: 16158168]
4. Antunes JLF, Peres MA, Mello TRdC, Waldman EA. Multilevel assessment of determinants of dental caries experience in Brazil. Community Dentistry and Oral Epidemiology. 2006; 34(2):146–152. [PubMed: 16515679]

5. Matijasevich A, Victora CG, Barros AJ, Santos IS, Marco PL, Albernaz EP, et al. Widening ethnic disparities in infant mortality in southern Brazil: comparison of 3 birth cohorts. Am J Public Health. 2008; 98(4):692–68. [PubMed: 17761568]
6. Victora CG, Matijasevich A, Silveira M, Santos I, Barros AJ, Barros FC. Socio-economic and ethnic group inequities in antenatal care quality in the public and private sector in Brazil. Health Policy Plan. 2010; 25(4):253–61. [PubMed: 20123940]
7. Matijasevich A, Santos IS, Silveira MF, Domingues MR, Barros AJ, Marco PL, et al. Inequities in maternal postnatal visits among public and private patients: 2004 Pelotas cohort study. BMC Public Health. 2009; 9:335. [PubMed: 19751521]
8. Frankel S, Elwood P, Sweetnam P, Yarnell J, Smith GD. Birthweight, body-mass index in middle age, and incident coronary heart disease. Lancet. 1996; 348(9040):1478–80. [PubMed: 8942776]
9. Jarvelin MR, Sovio U, King V, Lauren L, Xu B, McCarthy MI, et al. Early life factors and blood pressure at age 31 years in the 1966 northern Finland birth cohort. Hypertension. 2004; 44(6):838–46. [PubMed: 15520301]
10. Gluckman PD, Hanson MA, Cooper C, Thornburg KL. Effect of in utero and early-life conditions on adult health and disease. N Engl J Med. 2008; 359(1):61–73. [PubMed: 18596274]
11. Victora CG, Adair L, Fall C, Hallal PC, Martorell R, Richter L, et al. Maternal and child undernutrition: consequences for adult health and human capital. Lancet. 2008; 371(9609):340–57. [PubMed: 18206223]
12. Currie J. Healthy, Wealthy, and Wise: Socioeconomic Status, Poor Health in Childhood, and Human Capital Development. Journal of Economic Literature. 2009; 47(1):87–122.
13. Cunha F, Heckman J, Schennach S. Estimating the Technology of Cognitive and Noncognitive Skill Formation. Econometrica. 2010; 78(3):883–931. [PubMed: 20563300]
14. Doyle O, Harmon CP, Heckman JJ, Tremblay RE. Investing in early human development: timing and economic efficiency. Econ Hum Biol. 2009; 7(1):1–6. [PubMed: 19213617]
15. Acevedo-Garcia D, Osypuk TL, McArdle N, Williams DR. Toward a policy-relevant analysis of geographic and racial/ethnic disparities in child health. Health Aff (Millwood). 2008; 27(2):321–33. [PubMed: 18332486]
16. Chen E, Martin AD, Matthews KA. Understanding health disparities: the role of race and socioeconomic status in children's health. Am J Public Health. 2006; 96(4):702–8. [PubMed: 16507739]
17. Lu MC, Halfon N. Racial and ethnic disparities in birth outcomes: a life-course perspective. Matern Child Health J. 2003; 7(1):13–30. [PubMed: 12710797]
18. Miller, D. What underlies the black-white infant mortality gap? The importance of birthweight, behavior, environment, and health care. University of California at Berkeley; 2003. Working paper
19. Pitts, M.; Walker, M.; Armour, B. A Decomposition of the Black-White Differential in Birth Outcomes. Federal Reserve Bank of Atlanta; 2011. Working Paper Series 2011-1
20. Cramer J. Racial and Ethnic Differences in Birthweight: The Role of Income and Financial Assistance. Demography. 1995; 32(2):231–247. [PubMed: 7664962]
21. Lin W. Why has the health inequality among infants in the US declined? Accounting for the shrinking gap. Health Economics. 2009; 18(7):823–841. [PubMed: 18816582]
22. Lhila A, Long S. What is driving the black-white difference in low birthweight in the US? Health Econ. 2012; 21(3):301–315. [PubMed: 21294220]
23. Collins JW Jr, David RJ. Racial Disparity in Low Birth Weight and Infant Mortality. Clinics in Perinatology. 2009; 36(1):63–73. [PubMed: 19161865]
24. Raus V, Andrews H, Garfinkel R. The contribution of maternal age to racial disparities in birthweight: a multilevel perspective. American Journal of Public Health. 2001; 91(11):1815–1824. [PubMed: 11684610]
25. Shone LP, Dick AW, Klein JD, Zwanziger J, Szilagyi PG. Reduction in Racial and Ethnic Disparities After Enrollment in the State Children's Health Insurance Program. Pediatrics. 2005; 115(6):e697–e705. [PubMed: 15930198]
26. Liu J, Probst JC, Martin AB, Wang J-Y, Salinas CF. Disparities in Dental Insurance Coverage and Dental Care Among US Children: The National Survey of Children's Health. Pediatrics. 2007; 119(Supplement 1):S12–S21. [PubMed: 17272579]

27. Chin MH, Alexander-Young M, Burnet DL. Health Care Quality-Improvement Approaches to Reducing Child Health Disparities. Pediatrics. 2009; 124(Supplement 3):S224–S236. [PubMed: 19861474]
28. Fiscella K, Williams DR. Health Disparities Based on Socioeconomic Inequities: Implications for Urban Health Care. Academic Medicine. 2004; 79(12):1139–1147. [PubMed: 15563647]
29. Nyarko KA, Wehby GL. Residential Segregation and the Health of African-American Infants: Does the Effect Vary by Prevalence? Matern Child Health J. 2011
30. Telles EE. Residential Segregation by Skin Color in Brazil. American Sociological Review. 1992; 57(2):186–197.
31. Telles, EE. Race in another America : the significance of skin color in Brazil. Princeton, N.J.: Princeton University Press; 2004.
32. CIA CIA. The World Factbook. South America: Brazil: 2012.
33. IBGE IBdGeE. Race and Color. 2006
34. Wehby GL, Murray JC, McCarthy AM, Castilla EE. Racial Gaps in Child Health Insurance Coverage in Four South American Countries: The Role of Wealth, Human Capital, and Other Household Characteristics. Health Services Research. 2011; 46(6pt2):2119–2138. [PubMed: 21210797]
35. Barros FC, Victora CG, Horta BL. Ethnicity and infant health in Southern Brazil. A birth cohort study. Int J Epidemiol. 2001; 30(5):1001–8. [PubMed: 11689511]
36. Castilla EE, Orioli IM. ECLAMC: the Latin-American collaborative study of congenital malformations. Community Genet. 2004; 7(2-3):76–94. [PubMed: 15539822]
37. Wehby GL, Murray JC, Castilla EE, Lopez-Camelo JS, Ohsfeldt RL. Quantile effects of prenatal care utilization on birth weight in Argentina. Health Economics. 2009; 18(11):1307–1321. [PubMed: 19142894]
38. Wehby GL, Murray JC, Castilla EE, Lopez-Camelo JS, Ohsfeldt RL. Prenatal care demand and its effects on birth outcomes by birth defect status in Argentina. Economics & Human Biology. 2009; 7(1):84–95. [PubMed: 19059012]
39. Wehby GL, Castilla EE, Lopez-Camelo J. The impact of altitude on infant health in South America. Economics & Human Biology. 2010; 8(2):197–211. [PubMed: 20594925]
40. Lopez Camelo JS, Campana H, Santos R, Poletta FA. Effect of the interaction between high altitude and socioeconomic factors on birth weight in a large sample from South America. Am J Phys Anthropol. 2006; 129(2):305–10. [PubMed: 16323195]
41. Wehby GL, Murray JC, Castilla EE, Lopez-Camelo JS, Ohsfeldt RL. Prenatal care effectiveness and utilization in Brazil. Health Policy Plan. 2009; 24(3):175–88. [PubMed: 19282483]
42. Castilla E. Participation in ECLAMC. ECLAMC Coordinator. 2009
43. Degler, CN. Neither Black nor white : slavery and race relations in Brazil and the United States. Madison, Wis.: University of Wisconsin Press; 1986.
44. Chor D, Lima CRdA. Aspectos epidemiológicos das desigualdades raciais em saúde no Brasil. Cadernos de Saúde Pública. 2005; 21:1586–1594. [PubMed: 16158166]
45. Chaturvedi N. Ethnicity as an epidemiological determinant—crudely racist or crucially important? International Journal of Epidemiology. 2001; 30(5):925–927. [PubMed: 11689494]
46. Wehby G, McCarthy AM, Castilla EE, Murray JC. The Impact of Household Investments on Early Child Neurodevelopment and on Racial and Socioeconomic Developmental Gaps in South America. Forum for Health Economics & Policy. 2011; 14(2)
47. Wehby GL, Castilla EE, Lopez-Camelo JS, Murray JC. Predictors of multivitamin use during pregnancy in Brazil. Int J Public Health. 2009; 54(2):78–87. [PubMed: 19296054]
48. Aber JL, Bennett NG, Conley DC, Li J. The effects of poverty on child health and development. Annu Rev Public Health. 1997; 18:463–83. [PubMed: 9143727]
49. Adler NE, Newman K. Socioeconomic Disparities In Health: Pathways And Policies. Health Affairs. 2002; 21(2):60–76. [PubMed: 11900187]
50. Currie J, Neidell M, Schmieder JF. Air Pollution and Infant Health: Lessons from New Jersey. Journal of Health Economics. 2009; 28(3):688–703. [PubMed: 19328569]

51. Desai S, Alva S. Maternal education and child health: is there a strong causal relationship? Demography. 1998; 35(1):71–81. [PubMed: 9512911]
52. Kogan MD. Social causes of low birth weight. J R Soc Med. 1995; 88(11):611–5. [PubMed: 8544143]
53. Bryant AS, Worjoloh A, Caughey AB, Washington AE. Racial/ethnic disparities in obstetric outcomes and care: prevalence and determinants. American Journal of Obstetrics and Gynecology. 2010; 202(4):335–343. [PubMed: 20060513]
54. Kramer MR, Cooper HL, Drews-Botsch CD, Waller LA, Hogue CR. Metropolitan isolation segregation and Black–White disparities in very preterm birth: A test of mediating pathways and variance explained. Social Science & Medicine. 2010; 71(12):2108–2116. [PubMed: 20947234]
55. Grossman M. On the Concept of Health Capital and the Demand for Health. Journal of Political Economy. 1972; 80(2):223–255.
56. Guarnizo-Herreno CC, Wehby GL. Explaining Racial/Ethnic Disparities in Children's Dental Health: A Decomposition Analysis. Am J Public Health. 2012; 102(5):859–66. [PubMed: 22420801]
57. Rosenzweig MR, Schultz TP. Estimating a Household Production Function: Heterogeneity, the Demand for Health Inputs, and Their Effects on Birth Weight. The Journal of Political Economy. 1983; 91(5):723–746.
58. Aragão, VMdF; Silva, AAMd; Aragão, LFd; Barbieri, MA.; Bettiol, H.; Coimbra, LC., et al. Risk factors for preterm births in São Luís, Maranhão, Brazil. Cadernos de Saúde Pública. 2004; 20:57–63. [PubMed: 15029304]
59. Nyarko K, Lopez-Camelo JS, Castilla E, Wehby G. Does the relationship between prenatal care and birth weight vary by oral clefts? Evidence using South American and US samples. Journal of Pediatrics. 2012 Under review.
60. Barbieri MA, Silva AA, Bettiol H, Gomes UA. Risk factors for the increasing trend in low birth weight among live births born by vaginal delivery, Brazil. Rev Saude Publica. 2000; 34(6):596–602. [PubMed: 11175604]
61. Almeida, MFd; Alencar, GP.; Novaes, HMD.; França, I., Jr; Siqueira, AAFd; Campbell, OMR., et al. Risk-factors for antepartum fetal deaths in the city of São Paulo, Brazil. Rev Saude Publica. 2007; 41:35–43. [PubMed: 17273632]
62. Silva AA, Barbieri MA, Gomes UA, Bettiol H. Trends in low birth weight: a comparison of two birth cohorts separated by a 15-year interval in Ribeirao Preto, Brazil. Bull World Health Organ. 1998; 76(1):73–84. [PubMed: 9615499]
63. Rosenzweig MR, Schultz TP. The Stability of Household Production Technology - a Replication. Journal of Human Resources. 1988; 23(4):535–549.
64. Burgard S. Race and pregnancy-related care in Brazil and South Africa. Social Science & Medicine. 2004; 59(6):1127–1146. [PubMed: 15210086]
65. Silva LM, Silva RA, Silva AA, Bettiol H, Barbieri MA. Racial inequalities and perinatal health in the southeast region of Brazil. Braz J Med Biol Res. 2007; 40(9):1187–94. [PubMed: 17713668]
66. Kramer MS. Determinants of low birth weight: methodological assessment and meta-analysis. Bull World Health Organ. 1987; 65(5):663–737. [PubMed: 3322602]
67. Shah PS, Zao J, Ali S. Maternal Marital Status and Birth Outcomes: A Systematic Review and Meta-Analyses. Matern Child Health J. 2010
68. Rittler M, Castilla EE, Chambers C, Lopez-Camelo JS. Risk for gastroschisis in primigravidity, length of sexual cohabitation, and change in paternity. Birth Defects Res A Clin Mol Teratol. 2007; 79(6):483–7. [PubMed: 17358037]
69. Bettiol H, Rona RJ, Chinn S, Goldani M, Barbieri MA. Factors associated with preterm births in Southeast Brazil: a comparison of two birth cohorts born 15 years apart. Paediatric and Perinatal Epidemiology. 2000; 14(1):30–38. [PubMed: 10703032]
70. Victoria CG, Huttly SRA, Barros FC, Lombardi C, Vaughan JP. Maternal education in relation to early and late child health outcomes: Findings from a Brazilian cohort study. Social Science & Medicine. 1992; 34(8):899–905. [PubMed: 1604379]
71. Alves D, Belluzzo W. Infant mortality and child health in Brazil. Economics & Human Biology. 2004; 2(3):391–410. [PubMed: 15576245]

72. Zuckerman BS, Frank DA, Hingson R, Morelock S, Kayne HL. Impact of Maternal Work Outside the Home During Pregnancy on Neonatal Outcome. Pediatrics. 1986; 77(4):459–464. [PubMed: 3960614]
73. Bonzini M, Coggon D, Godfrey K, Inskip H, Crozier S, Palmer KT. Occupational physical activities, working hours and outcome of pregnancy: findings from the Southampton Women's Survey. Occupational and Environmental Medicine. 2009; 66(10):685–690. [PubMed: 19770355]
74. Visness CM, Kennedy KI. Maternal employment and breast-feeding: findings from the 1988 National Maternal and Infant Health Survey. American Journal of Public Health. 1997; 87(6):945–950. [PubMed: 9224174]
75. Berger LM, Hill J, Waldfogel J. Maternity leave, early maternal employment and child health and development in the US*. The Economic Journal. 2005; 115(501):F29–F47.
76. Lane SD, Keefe RH, Rubinstein R, Levandowski BA, Webster N, Cibula DA, et al. Structural violence, urban retail food markets, and low birth weight. Health & Place. 2008; 14(3):415–423. [PubMed: 17928255]
77. Landrine H, Corral I. Separate and unequal: residential segregation and black health disparities. Ethn Dis. 2009; 19(2):179–84. [PubMed: 19537230]
78. Gouveia N, Bremner SA, Novaes HMD. Association between ambient air pollution and birth weight in São Paulo, Brazil. Journal of Epidemiology and Community Health. 2004; 58(1):11–17. [PubMed: 14684720]
79. Szwarcwald CL, Andrade CLTd, Bastos FI. Income inequality, residential poverty clustering and infant mortality: a study in Rio de Janeiro, Brazil. Social Science & Medicine. 2002; 55(12):2083–2092. [PubMed: 12409122]
80. Carvalho, JAMd; Wood, CH. Mortality, Income Distribution, and Rural-Urban Residence in Brazil. Population and Development Review. 1978; 4(3):405–420.
81. Sastry N. What explains rural-urban differentials in child mortality in Brazil? Social Science & Medicine. 1997; 44(7):989–1002. [PubMed: 9089920]
82. Victora CG, Barros FC. Infant mortality due to perinatal causes in Brazil: trends, regional patterns and possible interventions. Sao Paulo Medical Journal. 2001; 119:33–42. [PubMed: 11175624]
83. Wooldridge, JM. Econometric analysis of cross section and panel data. Cambridge and London: MIT Press; 2002.
84. Fairlie RW. An extension of the Blinder-Oaxaca decomposition technique to logit and probit models. Journal of Economic and Social Measurement. 2005; 30(4):305–316.
85. Oaxaca R. Male-Female wage differentials in urban labor markets. International Economic Review. 1973; 14(3):693–709.
86. Blinder AS. Wage discrimination: Reduced form and structural estimates. Journal of Human Resources. 1973; 8(4):436–455.
87. Wehby GL, Murray JC, McCarthy AM, Castilla EE. Racial Gaps in Child Health Insurance Coverage in Four South American Countries: The Role of Wealth, Human Capital, and Other Household Characteristics. Health Services Research. 2011:no–no.
88. Telles EE. Race, Class and Space in Brazilian Cities. International Journal of Urban and Regional Research. 1995; 19(3):395–406.
89. Skidmore TE. Toward a Comparative Analysis of Race Relations Since Abolition in Brazil and the United States. Journal of Latin American Studies. 1972; 4(01):1–28.
90. Colen CG, Geronimus AT, Bound J, James SA. Maternal upward socioeconomic mobility and black-white disparities in infant birthweight. Am J Public Health. 2006; 96(11):2032–9. [PubMed: 17018818]
91. Ahmed AT, Mohammed SA, Williams DR. Racial discrimination & health: pathways & evidence. Indian J Med Res. 2007; 126(4):318–27. [PubMed: 18032807]
92. Walton E. Residential segregation and birth weight among racial and ethnic minorities in the United States. J Health Soc Behav. 2009; 50(4):427–42. [PubMed: 20099449]
93. Telles EE. Racial Distance and Region in Brazil: Intermarriage in Brazilian Urban Areas. Latin American Research Review. 1993; 28(2):141–162.

94. DeNavas-Walt, C.; Bernadette, B.; Smith, J. Income, Poverty, and Health Insurance Coverage in the United States: 2006. Washington, DC: U.S. Census Bureau; 2007.

95. Paim J, Travassos C, Almeida C, Bahia L, Macinko J. The Brazilian health system: history, advances, and challenges. The Lancet. 2011; 377(9779):1778–1797.

96. Valero de Bernabé J, Soriano T, Albaladejo R, Juarranz M, Calle MaE, Martínez D, et al. Risk factors for low birth weight: a review. European Journal of Obstetrics & Gynecology and Reproductive Biology. 2004; 116(1):3–15. [PubMed: 15294360]

97. Merialdi, M.; de Onis, M. LOW BIRTHWEIGHT AND PRETERM INFANTS | Causes, Prevalence and Prevention. In: Benjamin, C., editor. Encyclopedia of Human Nutrition. Second Edition. Oxford: Elsevier; 2005. p. 161-167.Editor-in-Chief

98. Acevedo-Garcia D, Soobader M-J, Berkman LF. The Differential Effect of Foreign-Born Status on Low Birth Weight by Race/Ethnicity and Education. Pediatrics. 2005; 115(1):e20–e30. [PubMed: 15629963]

99. Chomitz VR, Cheung LWY, Lieberman E. The Role of Lifestyle in Preventing Low Birth Weight. The Future of Children. 1995; 5(1):121–138. [PubMed: 7633859]

100. Goldenberg RL, Culhane JF, Iams JD, Romero R. Epidemiology and causes of preterm birth. The Lancet. 371(9606):75–84.

Table 1

Distribution of Study Variables

		Only African Ancestry (N=827)	Non-European African mixed Ancestry (N=1514)	European African mixed Ancestry (N=4583)	Only European Ancestry (N=2025)
Variable	**Description**				
Prenatal visits	Number of prenatal care visits	5.9 (2.6) [<0.001]	7.0 (2.6) [0.008]	6.5 (2.4) [<0.001]	6.8 (2.2)
Infant characteristics					
Low birth weight	Low birth weight (< 2500 grams) (%)	12.1 [0.001]	12.2 [<0.001]	12.8 [<0.001]	8.10
Preterm birth	Less than 37 weeks of gestation (%)	18.6 [0.016]	19.6 [0.001]	18.4 [0.001]	15.0
Female	Female infants (%)	46.1 [0.239]	48.0 [0.011]	44.3 [0.653]	43.7
Maternal demographics					
Acute	Acute illnesses (%)	45.7 [0.001]	48.8 [0.005]	43.9 [<0.001]	53.5
Chronic	Chronic illnesses (%)	14.9 [0.756]	18.0 [0.004]	15.6 [0.227]	14.4
Conception	Conception difficulty (%)	15.2 [0.007]	10.6 [0.389]	7.64 [<0.001]	11.6
Live births	Number of Live births (mean)	1.67 (2.11) [<0.001]	1.33 (1.92) [0.028]	1.30 (2.04) [0.047]	1.20 (1.78)
Still births	Number of spontaneous still births (mean)	0.296 (0.819) [0.966]	0.302 (0.803) [0.796]	0.298 (0.855) [0.877]	0.295 (0.798)
Maternal age	Maternal age 13 to 49 years (mean)	25.40 (6.68) [0.169]	26.19 (6.90) [0.072]	25.28 (6.64) [0.006]	25.77 (6.58)
Maternal age squared	Maternal age 13 to 49 years squared (mean)	689.69 (366.51) [0.232]	733.32 (384.62) [0.043]	683.34 (362.66) [0.012]	707.67 (363.95)
Cohabitation	Cohabitation length (mean)	1.85 (3.69) [0.002]	1.74 (3.65) [0.006]	1.61 (3.41) [0.034]	1.42 (3.37)
Maternal education[a]					
No schooling	Maternal no schooling and cannot read (%)	1.69 [0.001]	1.39 [0.003]	1.11 [0.008]	0.44
Primary school	Maternal primary school complete (%)	12.8 [0.004]	13.5 [0.003]	14.7 [0.008]	17.2
Incomplete secondary	Maternal secondary school incomplete (%)	16.2 [0.444]	16.1 [0.391]	15.7 [0.531]	15.1
Complete secondary	Maternal secondary school complete (%)	18.7 [0.001]	20.9 [0.009]	21.6 [0.006]	24.7
Incomplete university	Maternal university incomplete (%)	0.85 [0.001]	1.98 [0.007]	1.99 [0.001]	3.51
Complete university	Maternal university complete (%)	0.73 [<0.001]	2.97 [0.006]	3.08 [0.001]	4.79
Maternal occupation[b]					
Unemployed	Maternal Unemployed (%)	5.80 [<0.001]	7.0 [<0.001]	4.06 [0.005]	2.67
Unskilled	Maternal blue collar (%)	13.1 [0.081]	15.8 [<0.001]	16.3 [<0.001]	10.8
Skilled	Maternal blue collar (%)	4.84 [0.001]	8.78 [0.609]	5.26 [<0.001]	9.28

		Only African Ancestry (N=827)	Non-European African mixed Ancestry (N=1514)	European African mixed Ancestry (N=4583)	Only European Ancestry (N=2025)
Variable	**Description**				
Independent	Maternal independent (%)	1.45 [<0.001]	1.85 [<0.001]	3.14 [0.001]	4.79
Clerk	Maternal clerk (%)	12.9 [0.915]	10.4 [0.032]	12.7 [0.88]	12.8
Executive	Maternal executive (%)	0.85 [0.001]	1.25 [0.001]	1.94 [0.001]	3.26
Paternal demographics					
Paternal age	Paternal age 13 to 69 years (mean)	28.72 (8.18) [0.045]	29.09 (7.77) [0.288]	28.64 (7.69) [0.001]	29.37 (7.73)
Paternal age squared	Paternal age 13 to 69 squared (mean)	891.55 (536.26) [0.155]	906.43 (506.56) [0.366]	879.37 (502.76) [0.002]	922.13 (513.82)
Paternal education[a]					
No schooling	Paternal no schooling and cannot read (%)	1.33 [0.046]	2.05 [0.001]	1.55 [0.001]	0.59
Primary school	Paternal primary school complete (%)	17.3 [0.688]	16.5 [0.272]	18.7 [0.468]	17.9
Incomplete secondary	Paternal secondary school incomplete (%)	14.9 [0.234]	14.7 [0.208]	13.0 [0.822]	13.2
Complete secondary	Paternal secondary school complete (%)	17.9 [<0.001]	19.5 [<0.001]	19.8 [<0.001]	25.3
Incomplete university	Paternal university incomplete (%)	1.09 [0.001]	2.11 [0.002]	1.78 [<0.001]	4.00
Complete university	Paternal university complete (%)	0.73 [<0.001]	2.05 [<0.001]	2.20 [<0.001]	5.33
Paternal occupation[c]					
Unskilled	Paternal unskilled blue collar (%)	38.2 [<0.001]	38.4 [<0.001]	42.5 [<0.001]	27.2
Skilled	Paternal skilled blue collar (%)	19.1 [0.054]	24.5 [<0.137]	17.5 [<0.001]	22.4
Independent	Paternal independent (%)	4.96 [<0.001]	7.13 [<0.001]	7.16 [<0.001]	11.2
Clerk	Paternal clerk (%)	26.7 [<0.001]	17.9 [<0.001]	19.4 [<0.001]	24.4
Executive	Paternal executive (%)	2.17 [<0.001]	2.97 [<0.001]	4.62 [<0.001]	9.43

Notes: The table reports the descriptive statistics including frequencies for categorical variables and means with standard deviations (SD) in parentheses for continuous variables based on observations with complete data on all study variables. P-values for differences between the three African ancestry groups and the European only group are in brackets (p values rounded to <0.001 where applicable).

[a]Reference category is incomplete primary education and literate without formal schooling.

[b]Reference category is stay home mothers.

[c]Reference category is unemployed or stay home fathers.

Table 2

Overall Effects (Odd Ratios) of African Ancestry Indicators on low birth weight and preterm birth

	Total model sample (N)	Unadjusted		Adjusted	
		Low birth weight	Preterm birth	Low birth weight	Preterm birth
AO [a]	2,852	1.56*** [1.20,2.03]	1.30** [1.05,1.61]	1.28 [0.99,1.65]	1.10 [0.97,1.58]
ANE [a]	3,539	1.66*** [1.39,2.00]	1.39*** [1.16,1.65]	1.10 [0.76,1.42]	1.09 [0.92,1.32]
AE [a]	6,608	1.58*** [1.27,1.97]	1.28*** [1.11,1.48]	1.02 [0.75,1.22]	1.01 [0.89,1.30]

Notes: The table shows odds ratios for the effects of ethnic ancestry on LBW and PTB with 95% confidence intervals in brackets. A separate model is estimated for each African ancestry group relative to EO ancestry. The sample size for each ancestry group is the same as that Table 1. The model sample size is the sum of each African ancestry group sample and that of the EO group and is the same for all adjusted and unadjusted models for LBW and PTB for a certain ancestry comparison. The adjusted model includes as covariates all the explanatory variables listed in Table 1.

[a]Reference is EO ancestry.

** $p < 0.05$,

*** $p < 0.01$

Table 3

Decomposition of racial disparities in LBW

	AO versus EO	ANE versus EO	AE versus EO
Panel A: Total difference in LBW rate and difference jointly explained by variables			
Difference in LBW rate (0-1)	0.0399	0.0412	0.0469
Explained difference	0.0178	0.0299	0.0439
% Explained	44.6	72.6	93.6
% Unexplained	55.4	27.4	6.4
Panel B: Difference in LBW rate independently explained by variable categories			
Prenatal visits	0.0148 *** (0.0034)	-0.0049 ** (0.0023)	0.0062 *** (0.0015)
Maternal fertility history	-0.0019 * (0.0011)	-0.0009 (0.0007)	-0.0004 (0.0008)
Maternal health	-0.0009 (0.0013)	-0.0006 (0.0013)	-0.0046 *** (0.0012)
Household demographics	0.0011 (0.0013)	0.0035 ** (0.0015)	0.0002 (0.0011)
Socio-economic status	0.0030 (0.0040)	0.0038 * (0.0023)	0.0032 ** (0.0013)
Geographic location	0.0017 (0.0041)	0.0289 *** (0.0067)	0.0394 *** (0.0049)
Total model sample (N)	2,852	3,539	6,608

Notes: The table reports the differences in LBW rate (on a scale between 0 and 1) by ancestry and the contributions of the model variables to these differences. The standard errors of the variable contributions are in parentheses.

* $p < 0.1$,

** $p < 0.05$,

*** $p < 0.01$. For example, the number of prenatal care visits explains 0.0148 points of 0.0399-point difference (or 1.48 percentage-points of the 3.99 percentage-point difference) in LBW rate between infants of AO and EO ancestries). The sample size for each ancestry group is the same as that in Table 1. The model sample size is the sum of each African ancestry group sample and that of the EO group.

Table 4

Decomposition of racial disparities in PTB

	AO versus EO	ANE versus EO	AE versus EO
Panel A: Total difference in PTB rate and difference jointly explained by variables			
Difference in PTB rate (0-1)	0.0366	0.0465	0.0347
Explained difference	0.0235	0.0348	0.0326
% Explained	64.2	74.8	93.9
% Unexplained	35.8	25.2	6.1
Panel B: Difference in PTB rate independently explained by variable categories			
Prenatal visits	0.0231*** (0.0041)	-0.0060*** (0.0016)	0.0095*** (0.0024)
Maternal fertility history	0.0022 (0.0018)	-0.0004 (0.0007)	0.0007* (0.0004)
Maternal health	-0.0018 (0.0015)	-0.0011 (0.0012)	-0.0028** (0.0014)
Household demographics	0.0009 (0.0014)	0.0023* (0.0013)	0.0004 (0.0006)
Socio-economic status	0.0001 (0.0065)	0.0046 (0.0041)	-0.0027 (0.0025)
Geographic location	-0.0009 (0.0069)	0.0354*** (0.0072)	0.0276*** (0.0070)
Total model sample (N)	2852	3539	6608

Notes: The table reports the differences in PTB rate (on a scale between 0 and 1) by ancestry and the contributions of the model variables to these differences. The standard errors of the variable contributions are in parentheses.

* $p < 0.1$,

** $p < 0.05$,

*** $p < 0.01$. For example, the number of prenatal care visits explains 0.0231 points of 0.0366-point difference (or 2.31 percentage-points of the 3.66 percentage-point difference) in PTB rate between infants of AO and EO ancestries. The sample size for each ancestry group is the same as that in Table 1. The model sample size is the sum of each African ancestry group sample and that of the EO group.