OBSTETRICS

A case for not adjusting birthweight customized standards for ethnicity: observations from a unique Australian cohort

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BACKGROUND: Low birthweight is more common in infants of indigenous (Aboriginal and/or Torres Strait Islander) than of White Australian mothers. Controversy exists on whether fetal growth is normally different in different populations.

OBJECTIVE: We sought to determine the relationships of birthweight, birthweight percentiles, and smoking with perinatal outcomes in indigenous vs nonindigenous infants to determine whether the White infant growth charts could be applied to indigenous infants.

STUDY DESIGN: Data were analyzed for indigenous status, maternal age and smoking, and perinatal outcomes in 45,754 singleton liveborn infants of at least 20 weeks gestation or 400 g birthweight delivered in New South Wales, Australia, between June 2010 and July 2015.

RESULTS: Indigenous infants (n=6372; 14%) had a mean birthweight 67 g lower than nonindigenous infants ($P<.0001$; with adjustment for infant sex and maternal body mass index). Indigenous mean birthweight percentile was 4.2 units lower ($P<.0001$). Adjustment for maternal age, smoking, body mass index, and infant sex reduced the difference in birthweight/percentiles to nonsignificance (12 g; $P=.07$).

CONCLUSION: Disparities exist between indigenous and nonindigenous Australian infants for birthweight, birthweight percentile, and adverse outcome rates. Adjustment for smoking and maternal age removed any significant difference in birthweights and birthweight percentiles for indigenous infants. Our data indicate that birthweight percentiles should not be adjusted for indigenous ethnicity because this normalizes disadvantage; because White and indigenous Australians have diverged for approximately 50,000 years, it is likely that the same conclusions apply to other ethnic groups. The disparities in birthweight percentiles that are associated with smoking will likely perpetuate indigenous disadvantage into the future because low birthweight is linked to the development of chronic noncommunicable disease and poorer educational attainment; similar problems may affect other indigenous populations.

Key words: Australian, birthweight, ethnicity, smoking

Optimal birthweight is a complex and vigorously debated subject. The debate is not academic because fetal growth restriction is a key determinant of fetal risk of intrauterine death,[1] perinatal death,[2] and early cardiovascular disease, diabetes mellitus,[3] and renal impairment.[4] A recent supplement of the *American Journal of Obstetricians and Gynecologists*[5] summarized the range of views. In the supplement, 6 different models of fetal growth were advanced by their proponents.[6–11] A particular issue is whether healthy fetal growth is different in different ethnic populations. Studies in Australia may have some unique perspectives on this issue.

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Australia was peopled initially by indigenous Australians, also known as Aboriginal and Torres Strait Islander people, approximately 50 thousand years ago.[12] They had moved out of Africa in the same wave of migration that saw the ancestors of modern Europeans move north into the Middle East and eventually into Europe, although others moved into Asia and eventually, the Americas. Indigenous Australians then remained relatively isolated until Australia was colonized by Europeans in 1788. Indigenous Australians have generally poorer health (http://www.aihw.gov.au)[12–14] and a shorter life expectancy than nonindigenous Australians; increasing evidence indicates that this gap begins at the start of life. Indigenous Australians are known to be born at lower birthweights than other Australians.[15] The majority of adverse neonatal outcomes are known to occur in infants born at <10th percentile.[16–18] However, 2 recent studies have challenged this idea of neonatal risk. In a cohort of >1 million singleton Dutch infants, who were examined between 2002 and 2008, the lowest rates of perinatal morbidity and mortality occurred between the 50th and 97th percentiles.[19] Almost identical results were observed in an Australian setting in the state of Victoria, where >600,000 singleton births between 1999 and 2008 were assessed, and birthweight percentiles were correlated to perinatal outcomes; again, optimal outcomes occurred between the 50th and 90th percentile.[20]

This is a significant change in thinking because it puts more than one-half of all newborn infants at an increased risk of adverse outcomes, albeit with higher risk at the lowest and highest percentiles. Both of these cohort studies reported data from predominantly White populations without reference to ethnicity. Birthweight previously has been shown to predict long-term health[17] and educational outcomes.[21–24] Maternal smoking has been associated with restriction of fetal growth,[25] and indigenous mothers are known to have high rates of cigarette smoking.[26]

AJOG at a Glance

Why was this study conducted?
We compared birthweights of Aboriginal and non-Aboriginal Australian babies.

Key findings
Aboriginal and non-Aboriginal babies have similar birthweights when adjustment is made for maternal smoking status.

What does this add to what is known?
Adjustment for ethnicity normalizes disadvantage.

We therefore sought to determine the relationships between maternal smoking, birthweight, birthweight percentile distribution, and perinatal outcomes for indigenous and non-indigenous singleton births in the Hunter and New England Local Health District (HNELHD) of New South Wales, Australia. This region is appropriate because 25% of all indigenous births in New South Wales occur in the HNELHD region, and New South Wales has more indigenous people than any other state.[13] The HNELHD includes urban, rural, and remote areas that allowed us to interrogate our data for geographic effects. It is not clear whether infants born to Australian indigenous mothers should be assessed with the same birthweight charts as used for non-indigenous infants, because customizing charts for ethnicity may normalize the abnormal and minimize indigenous disadvantage. We therefore have used a birthweight percentile calculator without adjusting for ethnicity.[27]

Methods

Perinatal data

We analyzed data on singleton live infants who were born to women attending hospitals across the HNELHD that were collected between June 2010 and July 2015. Institutional review board approval was provided by both the Hunter New England Area Health Authority Ethics Committee and the Aboriginal Health and Medical Research Committees (DOC160715-16072015153511 AU201808-08).

De-identified data were obtained from an electronic database (Obstetrix; http://www.meridianhi.com/obstetrix/) that comprises computerized birth notification forms that were completed for all births of at least 20 weeks of gestation or 400 g birthweight, usually by midwives but sometimes by medical practitioners (N=45,754). We used the term "indigenous" to refer to infants whose mothers identified as of Aboriginal or Torres Strait Islander descent or who identified their infant as being of Aboriginal or Torres Strait Islander descent. Gestational age was reported in completed weeks of gestation, calculated from either the first day of the last menstrual period for which the cycle length was $\geq$21 and $\leq$35 days or by first trimester ultrasound scanning. Birthweight was reported to the nearest 5 g. We used the birthweight percentile calculator that was developed by the Perinatal Institute for Maternal and Child Health (www.perinatal.org.uk).[27] The percentile calculator produces customized birthweight percentiles with the use of the principles of the Gestation Related Optimal Weight method. Birth percentile was calculated and standardized by maternal height, infant sex, and length of gestation, but not for ethnicity. We used the Australian Bureau of Statistics website to interpret geographic location based on residential postcodes.[28] Smoking status of mothers was recorded prospectively.

Outcomes of interest comprised the continuous measures birthweight (grams) and birth percentile (0−100%) and a binary measure of combined adverse outcomes (stillbirth [fetal death before delivery of $\geq$20 completed weeks of gestation or of $\geq$400 g birthweight], neonatal death [within 28 days of delivery], or admission to neonatal intensive care unit/special care nursery).

Statistical analyses

Descriptive statistics were generated; for continuous variables, data were summarized with the use of means and standard deviations and median with first and third quartiles, although categoric variables were summarized as frequencies and percentages. Differences in means and frequencies between groups were assessed with *t*-tests and chi-square tests for continuous and categoric variables, respectively. To assess distributions visually in indigenous and non-indigenous infants, kernel densities for birthweight and birth percentile were estimated with the use of the Epanechnikov kernel function.

Directed acyclic graphs were constructed (Supplementary Data) to establish which maternal and infant characteristics confound or mediate the relationships under investigation.[29] The direct effect of ethnicity (the effect attributable to ethnicity after adjustment for confounders and mediators) and the total effect of ethnicity (adjusts only for confounders) were modelled for each outcome of interest.

The relationships among indigenous status and birthweight and birth percentile were assessed by linear regression with Huber-White standard errors. Total effects of ethnicity were obtained from the association of ethnicity with birthweight/percentile that included infant sex and maternal body mass index (BMI). The direct effects were obtained after adjustment for maternal age, maternal geographic location, and maternal smoking as mediators; maternal BMI and infant sex were also included to account for residual variance. Regressions for birthweight were adjusted for gestational age; however, the regression for percentile was not because gestational age was used for the calculation of standardized percentiles. Complete-case analyses were conducted.

The hypotheses that maternal geographic location and maternal smoking were mediators of the effect of ethnicity on birthweight and percentile

TABLE 1
Characteristics of indigenous and non-indigenous infants

Variable	Non-indigenous (n=39,382)	Indigenous (n=6372)	*P* value
Gestational length, d			
Mean±standard deviation	273±17	272±17	<.0001
Median (quartile 1, quartile 3)	276 (269, 283)	275 (267, 282)	<.0001
Birthweight, g			
Mean±standard deviation	3375±642	3264±659	<.0001
Median (quartile 1, quartile 3)	3435 (3070, 3770)	3330 (2940, 3670)	<.0001
Birthweight percentile			
Mean±standard deviation	44.4±29.6	39.9±29.9	<.0001
Median (quartile 1, quartile 3)	41.4 (18.2, 69.0)	34.3 (13.4, 63.6)	<.0001
Maternal age, y			
Mean±standard deviation	29±6	26±6	<.0001
Median (quartile 1, quartile 3)	29 (25, 33)	25 (21, 30)	<.0001
Body mass index, kg/m^2			
Mean±standard deviation	26.66±6.49	26.82±7.01	.0956
Median (quartile 1, quartile 3)	25.20 (22.02, 30.07)	25.33 (21.63, 30.68)	.9828
Infant sex, n (%)			
Male	20256 (51)	3304 (52)	.5220
Female	19125 (49)	3066 (48)	
Premature, n (%)			
No	35823 (91)	5631 (89)	<.0001
Yes	3527 (9.0)	729 (11)	
Apgar score at 5 minute, n (%)			
<7	1405 (3.6)	230 (3.6)	.8672
≥7	37977 (96)	6142 (96)	
Nursery admission, n (%)			
No	34476 (90)	5397 (87)	<.0001
Yes	3838 (10)	816 (13)	
Neonatal outcome, n (%)			
Discharged or transferred	38923 (99)	6289 (99)	.4991
Neonatal death	140 (0.4)	22 (0.3)	
Stillbirth	275 (0.7)	53 (0.8)	
Any adverse birth outcome, n (%)[a]			
No	35138 (89)	5481 (86)	<.0001
Yes	4244 (11)	891 (14)	
Maternal smoking, n (%)			
No	32836 (83)	3788 (60)	<.0001
Yes/not known	6511 (17)	2568 (40)	

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(continued)

TABLE 1
Characteristics of indigenous and non-indigenous infants *(continued)*

Variable	Non-indigenous (n=39,382)	Indigenous (n=6372)	*P* value
Location, n (%)			
Urban	20447 (52)	2156 (34)	<.0001
Regional	18353 (47)	4029 (64)	
Remote	212 (0.5)	112 (1.8)	

[a] Included stillbirth, neonatal death, and nursery admission.

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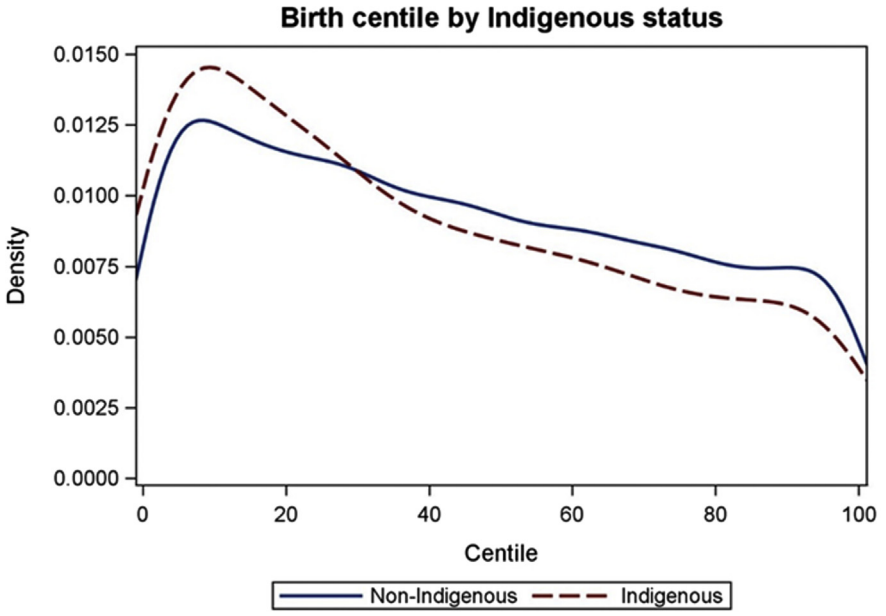
were tested. Although the effect of the proposed mediator on birthweight/percentile was not significant, sensitivity analysis of the birthweight/percentile on ethnicity regression was conducted by removing the nonsignificant variable and assessing the change in the ethnicity effect size. For all analyses, maternal smoking status categories were for "no" or for "yes and unknown" combined.

The distribution of combined adverse outcomes (stillbirth, neonatal death, or nursery admission) for indigenous and non-indigenous infants was assessed with the use of histograms of the raw proportions within birth ventiles (5% bands). Logistic regression with Huber-White standard errors was then used to assess the relationship between birth ventile and the combined adverse outcomes, which included ethnicity as a predictor, and adjustment for confounders (maternal age, maternal geographic location, maternal smoking, and infant sex). Using this model, the adjusted probabilities of adverse outcome within each ventile, by ethnicity and infant sex, were plotted (at the reference category of other confounders in the model). This method was repeated for infant death (stillbirth or neonatal death) with fewer percentile categories because of the rarity of the outcome and omission of geographic location because of low cell counts. Complete-case analyses were conducted.

For all analyses, results with a probability value of <.05 were considered significant. Statistical analyses were conducted using SAS software (version 9.4; SAS Institute, Cary, NC).

FIGURE 1
Kernel densities



Kernel densities for birth percentile by indigenous status that were estimated with the use of the Epanechnikov kernel function.

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Results

Descriptive statistics of relevant variables and birth outcomes by indigenous status are shown in Table 1. There were 6372 indigenous infants (14%) in the total sample of 45,754 singleton births; 0.2% of subjects of both ethnicities had smoking status unrecorded.

Crude analyses indicated that indigenous infants tended to have lower birthweights and percentiles and more likely to be born premature or to be admitted to special care nurseries. Mothers of indigenous infants, compared with non-indigenous mothers, tended to be younger, were more often from regional or remote geographic locations, and were more likely to have smoking status recorded as "yes" or "not known" (indigenous 40%; non-indigenous 17%). The BMIs of indigenous mothers were not different to those of non-indigenous mothers.

The kernel densities for birth percentile estimated with the use of the Epanechnikov kernel function are shown in Figure 1. Indigenous infants

TABLE 2
Estimates of the effect of ethnicity on birthweight or birth percentile from linear regression models

	Total effect of ethnicity (indigenous vs non indigenous)[a]			Direct effect of ethnicity (indigenous vs non indigenous)[b]		
Outcome	Beta	95% Confidence interval	*P* value	Beta	95% Confidence interval	*P* value
Birthweight, g	−67	−80 to −55	<.0001	−12	−24 to 1	.0709
Birth percentile	−4.2	−5.1 to −3.4	<.0001	−0.6	−1.4 to 0.3	.1943

[a] Obtained from the association of ethnicity with birthweight/percentile, with infant sex, and maternal body mass index included to account for residual variance; [b] Obtained after adjustment for maternal age, geographic location, smoking, body mass index, and infant sex (N=42,736).

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tended to be lighter; a greater proportion of them was born <50th percentile.

Table 2 summarizes the results of the linear regressions of birthweight and percentile on ethnicity (N=42736 complete cases). The total effects were statistically significant, with indigenous infants being 67 g lighter and birth percentile 4.2% lower, on average, than non-indigenous infants after adjustment for infant sex and maternal BMI ($P<.0001$). After maternal age, smoking and geographic location were accounted for, direct effects of ethnicity were no longer statistically significant (birthweight, $P=.08$; birth percentile, $P=.19$). A sensitivity analysis was performed that removed maternal geographic location from the regressions because location was not statistically associated with the outcomes; this resulted in effect sizes and confidence intervals relatively unchanged from those presented. Geographic location was retained in the models for direct effects because content knowledge provides a plausible basis for its role as a mediator.[18] These analyses demonstrate that the effect of ethnicity on birthweight/percentile is mediated through demographic and lifestyle variables (mainly maternal age and smoking), which account for the majority of the observable variation between indigenous and non-indigenous infants.

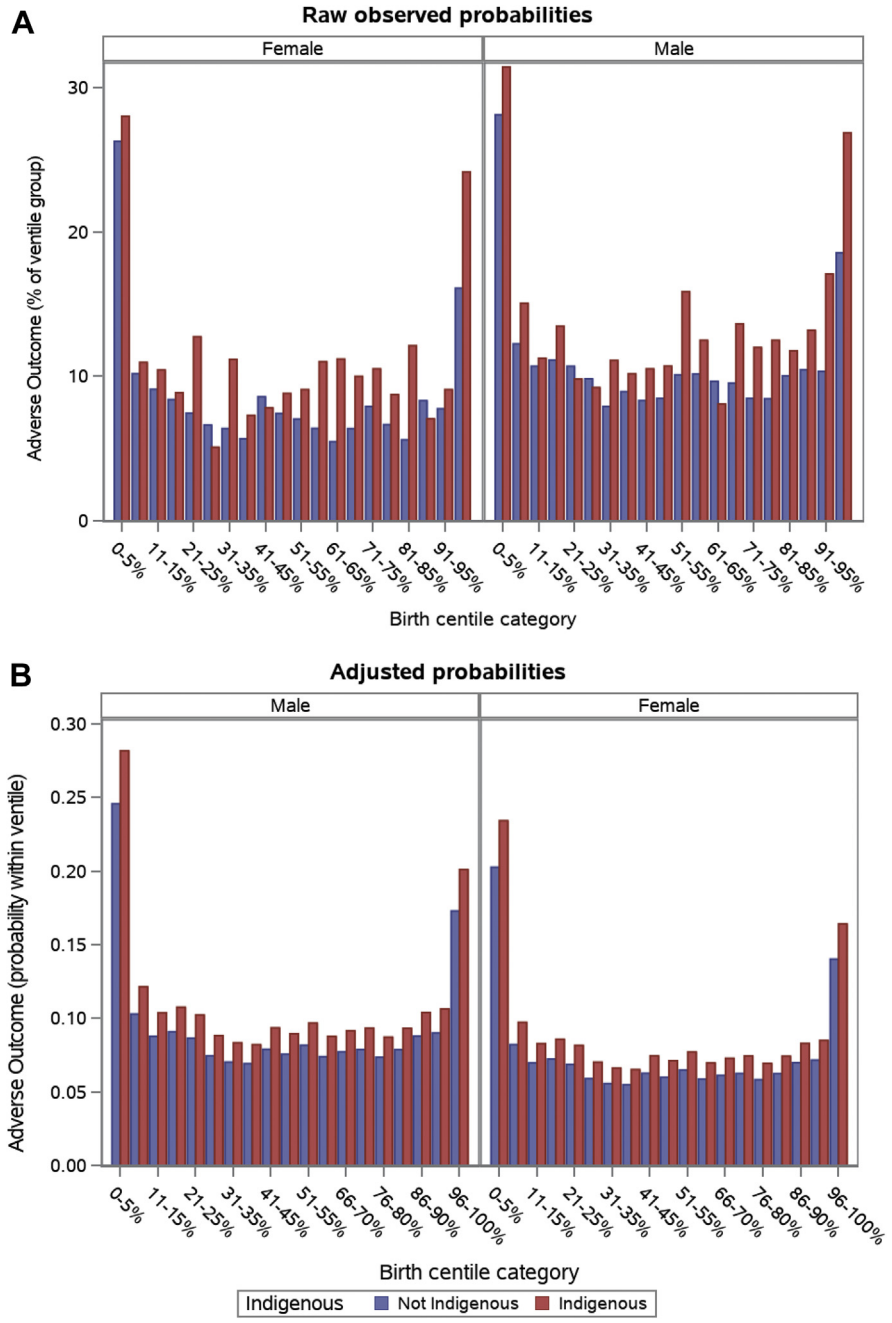
The relationship between birth ventile and having any of the adverse perinatal outcomes (stillbirth, neonatal death, or neonatal intensive care or special care nursery admission) was investigated by ethnicity and sex. Histograms of the raw observed percentages are presented in Figure 2, A. Across ventiles, the raw percentage of adverse outcomes was consistently higher for indigenous infants than for non-indigenous infants. The ventiles with the highest percentages of adverse outcomes for all infants corresponded to the 0-5th and the 96-100th ventiles. Through the center of the distributions, raw probabilities were substantially lower than at the bounds and reasonably uniformly distributed.

The distributions of adverse outcome probabilities that were estimated from a logistic regression model that adjusted for hypothesized confounders are presented in Figure 2, B. The model parameters for ventile, ethnicity, and sex were associated significantly with the outcome ($P<.0001$). After adjustment for potential confounders (maternal age, geographic location, smoking, and infant sex), the probabilities of adverse outcomes remained higher for indigenous infants compared with non-indigenous infants, and the distributions displayed patterns similar to the raw observed percentages. Figure 2, B, ventile probabilities are at the reference level of all variables in the logistic regression model (urban, nonsmoking, 28-year-old mothers) and therefore represent the probabilities of adverse outcomes in the absence of hypothesized risk factors. This analysis indicates that, although adverse outcomes associate with low and high birth percentile, indigenous infants experience a higher probability of adverse outcomes than do non-indigenous infants, regardless of birth percentile, even after mediators and confounders are accounted for; this is due to neonatal intensive care unit admission, with no increase in stillbirth or neonatal mortality rate.

The relationship between birth percentile and death was also investigated. Figure 3 depicts histograms of the raw observed mortality percentages by percentile group and the adjusted probabilities that were estimated from a logistic regression model that adjusted for hypothesized confounders. The percentile categories were collapsed in larger bands because of the rarity of the outcome. The model parameter for percentile category was associated significantly with death ($P<.0001$); however, ethnicity was not ($P=.21$), and infant sex was associated only very weakly with death ($P=.05$). The probability of death was highest for infants born <5th percentile. There was no statistical evidence of differences in mortality rates because of ethnicity after we controlled for possible risk factors (maternal age, geographic location, smoking status, and infant sex).

The profound impact of maternal smoking on birth percentiles of both indigenous and non-indigenous infants is illustrated in Figure 4. The data (Figure 4, A) indicate that the distribution of percentiles for infants of nonsmoking indigenous mothers is not distinguishable from those of non-indigenous infants of nonsmoking mothers. Equally, Figure 4, B, indicates that the effect of smoking is enough to generate bimodal Kernel density plots for both indigenous and non-indigenous infants.

FIGURE 2
Percentages and probabilities



A, Raw percentages and **B,** model-based adjusted probabilities of combined adverse outcomes by ventile group, ethnicity and sex.

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Comment

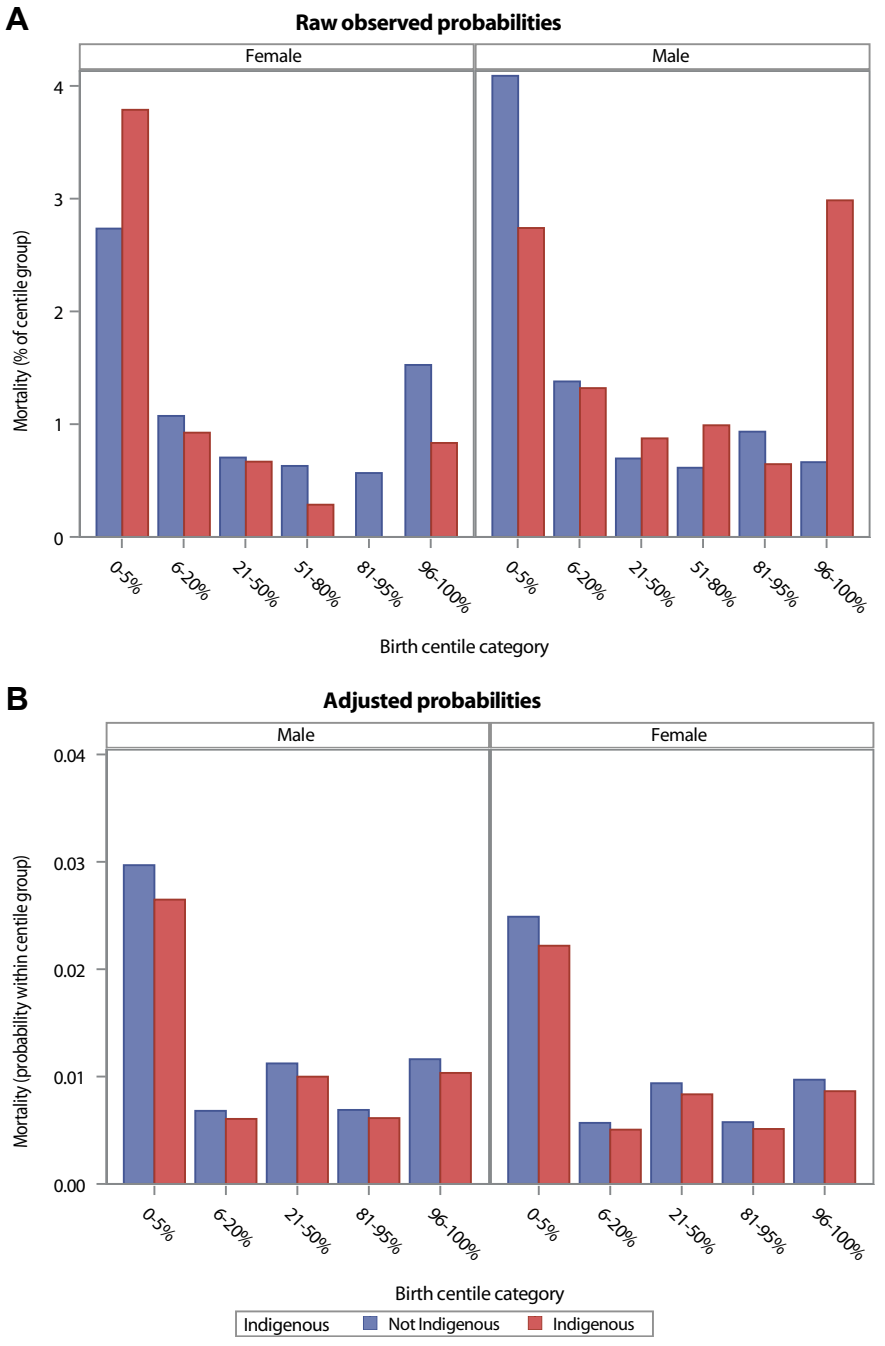
Our key findings are that (1) for Australian indigenous infants, the relationship between birthweight percentile and perinatal outcomes follows a similar pattern to that in non-indigenous infants, (2) at all birthweight percentiles, indigenous perinatal outcomes are worse predominantly because of admission to neonatal care nursery, (3) indigenous and non-indigenous pregnancies were associated with very similar risk of stillbirth and neonatal death, (4) indigenous infants are born lighter, with a greater proportion born at <50th percentile. However, adjustment for maternal age and smoking is associated with normalization of indigenous birthweights; consequently, it is unnecessary to adjust infant birthweight percentiles for Australian indigenous infants.

Our crude summary statistics for Australian pregnancy and childbirth characteristics and outcomes are similar to those reported by the Australian Institute of Health and Welfare (AIHW).[14] The AIHW report in 2013 indicated that indigenous infants were 1.7 times as likely as non-indigenous infants to be born preterm (14% vs 8%), compared with our 11% and 9%. The AIHW reported that the average indigenous liveborn baby was 161 g lighter (3200 vs 3361 g) than non-indigenous babies; the difference for our cohort was 111 g (Table 1). Indigenous Australian infants in the 2013 AIHW report and those in our sample were more likely than non-indigenous infants to be admitted to special care nursery/neonatal intensive care units (AIHW, 17% vs 14%; $P<.001$; Obstetrix, 13% vs 10%).

Effect of ethnicity on birthweight

A key finding in our study was that infants of nonsmoking indigenous mothers had birthweights that were indistinguishable from the non-indigenous nonsmoking population, which suggests that the birthweight charts that we used correctly represent normal indigenous fetal growth in the absence of maternal risk factors and that charts should not be adjusted for indigenous ethnicity. The data indicate that human fetal growth is constant across ethnic groups in the absence of constraining factors, such as malnutrition or smoking. White and Aboriginal humans have been separated for at least 50,000 years, and mitochondrial and genomic DNA studies show that they diverge as much as any non-African ethnic groups.[30–32] Thus, birthweight charts that adjust for ethnicity seem likely to normalize disadvantage and should not be used. Despite the similarity of indigenous and non-indigenous birthweights

FIGURE 3
Histograms



A, The raw observed mortality percentages by percentile group and **B,** the adjusted probabilities estimated from a logistic regression model that adjusted for hypothesized confounders.

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in the absence of risk factors, indigenous infants still experienced higher rates of adverse outcomes, predominantly neonatal nursery admission, which suggests that factors other than birth percentile are contributing to the increased risk of neonatal nursery admission in indigenous infants.

Optimal birthweight percentile

Our results regarding perinatal mortality rates in non-indigenous infants are broadly consistent with the results previously reported for the Dutch[33] and Victorian[20] cohorts, with the lowest mortality rate above the 20th percentile. Accounting for other risk factors, we found no statistical evidence that the probability of death by birth percentile differed between indigenous and non-indigenous infants.

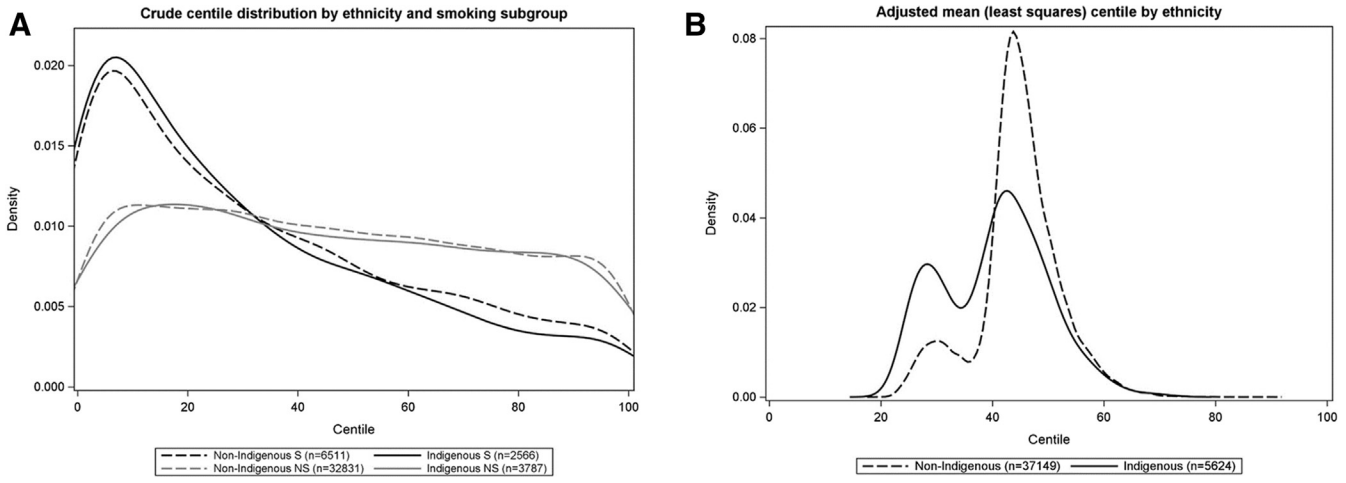
Smoking rates

Data on smoking status were not recorded for some of our cohort (0.2% indigenous and 0.2% non-indigenous); the reasons are unclear, but the group with unrecorded smoking status had similar birth outcomes to the smoking group (median birthweight, 2715 g, compared with 3170 g in known smokers and 3480 g in known nonsmokers). Eighty-three percent of non-indigenous mothers in our 2010–2015 cohort reported being nonsmokers; 60% of indigenous mothers reported not smoking. Data from the Darwin Health Region from 1987–1990 also reported a 54% maternal smoking rate as a key association with low birthweight and intrauterine growth retardation.[34] Thus, smoking has remained an important association with Australian indigenous low birthweight over the last 20 years, with little sign of improvement. It is likely that the relationship between smoking and low birthweight is causative because an inverse relationship exists between indigenous maternal plasma cotinine and infant birthweight.[26]

Gestational programming

The "fetal origins of disease" or "gestational programming" hypothesis proposes that adverse influences during pregnancy can induce permanent changes in the developing fetus, resulting in low birthweight. It is well-established that low birthweight and preterm deliveries predispose infants to adverse neonatal outcomes.[20,33,35] Those who survive tend to have impaired immune function[36] and an increased risk of poor school performance.[21–23] Guthridge et al[37] conducted a study of 7601 children (61% indigenous) born in the Northern Territory between 1999 and 2004 in which information was linked

FIGURE 4
Kernel density plots of crude birth percentile distributions and the adjusted mean birth percentile



A, Densities of crude birthweight percentiles by ethnicity and smoking status. **B**, Densities of the conditional mean percentile estimated from the adjusted model by ethnicity. The bimodal distribution (in particular, the lower range peak) is representative of the reduction in birthweight of infants because of smoking. *Broken lines* represent distributions for non-indigenous infants; *solid lines* represent distributions for indigenous infants.

NS, mother was nonsmoker or not recorded; *S,* mother was a smoker.

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for each child on perinatal health, student enrolment, and the Australian National Assessment Program-Literacy and Numeracy Year 3 (aged 8) results. Indigenous children had much higher odds, compared with non-indigenous children, of a result below the national minimum standard for both reading (odds ratio, 8.58; 95% confidence interval, 7.55—9.74) and numeracy (odds ratio, 11.52; 95% confidence interval, 9.94—13.35); poor educational performance in indigenous children was associated with maternal smoking and low birthweight.[38] The evidence we present in this study further demonstrates that disparities between indigenous and non-indigenous children in birthweight and educational outcomes are likely to remain until the rates of adverse influences, such as smoking during pregnancy for indigenous mothers are reduced. Similar challenges likely exist in other indigenous populations, such as First Nations in Canada[39] and the United States.[40]

Study strengths and weaknesses

Our study strengths include the use of the electronic Obstetrix data base, which comprehensively prospectively records birth characteristic of the infants across the Hunter New England Local Health District and a large sample size over a recent period of 5 years. A study weakness is the missing data on actual smoking rates in a significant proportion of the indigenous mothers; however, our rate is very similar to the reported Australian national smoking rate for indigenous Australians of 47%.[13]

Conclusion

In this study, we demonstrate that differences in birthweight between indigenous and non-indigenous infants may be accounted for substantially by smoking and that factors other than birthweight are contributing to the increased risk of neonatal admission in indigenous infants. The association between low birthweight, future health, and school performance means that indigenous children will remain disadvantaged into the future. Our data strongly indicate that maternal risk factors, particularly smoking, account for the majority of the difference in birthweights among Australian indigenous infants and that the use of birthweight charts that adjust for ethnicity normalizes indigenous disadvantage, which is likely to apply to other indigenous populations. ■

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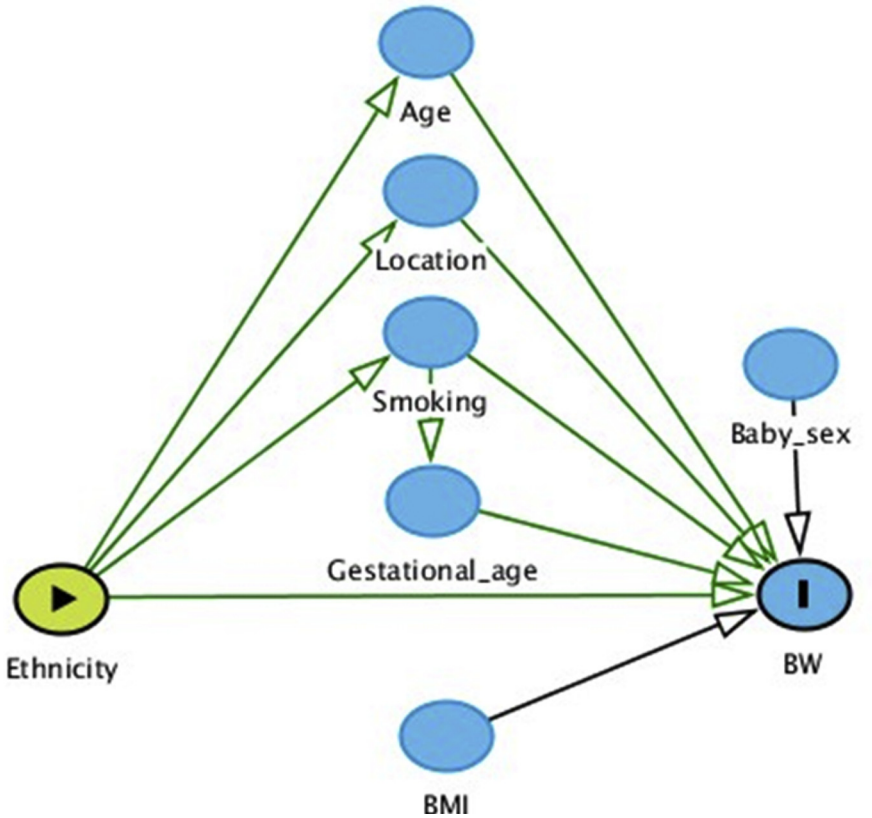
Supplemental Material

Glossary of Terms

Beta: a coefficient from a regression equation that denotes the estimated change in the outcome per unit increase in the exposure.
Confounders: variables that influence both the outcome and exposure and may distort the estimated association
Directed acyclic graphs (DAG): a diagram that depicts the hypothesized causal pathways among a set of variables, where arrowheads indicate the direction of influence
Epanechnikov kernel density function: a nonparametric estimator of the distribution function
Exposure variable: an independent variable that is hypothesized to affect an outcome
Kernel densities: the results of the application of the Epanechnikov kernel density function
Mediators: variables that lie on the causal pathway between exposure and outcome

Supplemental Data

FIGURE
Directed acyclic graph



Directed acyclic graph describes interactions between indigenous status and birthweight.

BMI, body mass index; *BW*, birthweight.

Smith et al. Maternal smoking mediates low birthweight in indigenous Australians. Am J Obstet Gynecol 2019.