

Early Access to Prenatal Care

Implications for Racial Disparity in Perinatal Mortality

Andrew J. Healy, MD, Fergal D. Malone, MD, Lisa M. Sullivan, PhD, T. Flint Porter, MD, David A. Luthy, MD, Christine H. Comstock, MD, George Saade, MD, Richard Berkowitz, MD, Susan Klugman, MD, Lorraine Dugoff, MD, Sabrina D. Craigo, MD, Ilan Timor-Tritsch, MD, Stephen R. Carr, MD, Honor M. Wolfe, MD, Diana W. Bianchi, MD, and Mary E. D'Alton, MD, for the FASTER Trial Research Consortium*

OBJECTIVE: To investigate racial disparities in perinatal mortality in women with early access to prenatal care.

METHODS: A prospectively collected database from a large, multicenter investigation of singleton pregnancies, the FASTER trial, was queried. Patients were recruited from an unselected obstetric population between 1999 and 2002. A total of 35,529 pregnancies with early access to prenatal care were reviewed for this analysis. The timing of perinatal loss was assessed. The following intervals were evaluated: fetal demise at less than 24 weeks of gestation, fetal demise at 24 or more weeks of gestation, and neonatal demise. Perinatal mortality was defined as the sum of these three intervals.

RESULTS: The study population was 5% black, 22% Hispanic, 68% white, and 5% other. All minority races experienced higher rates of intrauterine growth restriction, preeclampsia, preterm premature rupture of membranes, gestational diabetes, placenta previa, preterm birth, very-preterm birth, cesarean delivery, light vaginal bleeding, and heavy vaginal bleeding compared with the

white population. Overall perinatal mortality was 13 per 1,000 (471/35,529). The adjusted odds ratios (95% confidence intervals) for perinatal mortality (utilizing the white population as the referent race) were: black 3.5 (2.5–4.9), Hispanic 1.5 (1.2–2.1), and other 1.9 (1.3–2.8).

CONCLUSION: Racial disparities in perinatal mortality persist in contemporary obstetric practice despite early access to prenatal care.

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LEVEL OF EVIDENCE: II-2

Racial disparity in perinatal outcomes within the United States has been documented since the early 1920s.¹ Despite improvements in perinatal survival among all races, the disparities between white and nonwhite populations persist. The etiology of these differences is uncertain; nevertheless, among the most commonly cited explanations are racial variations in socioeconomic status, prevalence of specific risk factors, and access to prenatal care.²

The concept of prenatal care was developed in the United States in the early 1900s. The establishment of regularly scheduled medical visits for pregnant women represents one of the most important advances in obstetric care in the past century, and its role in reducing fetal death is well established.^{3–6} Variations in the access and utilization of prenatal care reside along patient demographic, economic, and racial lines.³ The Federal government is cognizant of variations in the utilization of prenatal care. Healthy People 2010, a federal initiative launched with the intent of eliminating racial disparities in health, includes the goal of increasing early (first-trimester) access to prenatal care to 90% of all pregnant women. However, the impact of this laudable goal on this seemingly omnipresent disparity is uncer-

*For a list of FASTER Trial Research Consortium members, see the Appendix.

From Columbia University, New York, New York; DM-STAT, Medford, Massachusetts; University of Utah Health Sciences Center, Salt Lake City, Utah; Swedish Medical Center, Seattle, Washington; William Beaumont Medical Center, Royal Oak, Texas; University of Texas Medical Branch, Galveston, Texas; Mount Sinai Medical Center, New York, New York; Albert Einstein College of Medicine, Bronx, New York; University of Colorado Health Sciences Center, Denver, Colorado; Tufts University, Boston, Massachusetts; New York University, New York, New York; Women and Infants' Hospital, Rhode Island; and University of North Carolina, Chapel Hill, North Carolina.

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Corresponding author: Andrew J. Healy, MD, Columbia University Medical Center, 622 West 168th Street, PH-16, New York, NY 10032; e-mail: ajh2102@columbia.edu.

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tain. Can early access to prenatal care minimize racial disparity in perinatal mortality in contemporary obstetric practice?

MATERIALS AND METHODS

A prospectively collected database from a large, multicenter investigation of singleton pregnancies, the FASTER (First- and Second-Trimester Evaluation of Risk) trial, was studied. The primary objective of this trial, funded by the National Institute of Child and Human Development, was to evaluate first-trimester nuchal translucency along with first- and second-trimester maternal serum markers as screening modalities for Down syndrome. Patients were recruited from an unselected obstetric population between 1999 and 2002. Patients were enrolled between 10^{3/7} to 13^{6/7} weeks of gestation therefore ensuring early access to prenatal care. Enrollment occurred at 15 centers distributed across 9 states. Institutional review board approval was obtained at all sites. Upon initial intake, baseline data were collected by self-administered questionnaires. Patients were asked to identify themselves as American Indian or Alaskan Native, Asian or Pacific Islander, black (not of Hispanic origin), white (not of Hispanic origin), Hispanic, or other. Race was assigned based on patient response to this question. Because of the relatively small numbers who self-identified as American Indian or Alaskan Natives and Asian or Pacific Islanders, these two categories were combined with the other category for the purposes of this analysis. When a patient indicated more than one race, the patient was then assigned to a single race by the data management group based on a preestablished algorithm. Patients were recoded to a single race with the following hierarchy: black, Hispanic, Asian, American Indian, and white.

Elective terminations and patients with incomplete demographic information were excluded. Pregnancies complicated by major structural anomalies or aneuploidy were also excluded, leaving 35,529 pregnancies for analysis. The database contains detailed antenatal, birth, and pediatric outcomes on a large, racially diverse, obstetric population.

The following demographic characteristics were assessed: age, education, marital status, body mass index (BMI), tobacco use during pregnancy, illicit drug use during pregnancy, alcohol consumption during pregnancy, medication use in pregnancy, pregestational diabetes, obstetric history (previous live birth, miscarriage, and preterm delivery), utilization of assisted reproductive technologies and antihypertensive medication use before pregnancy. In addition, 12 pregnancy complications, defined in a previous publication⁷ were recorded.

These included intrauterine growth restriction (IUGR), gestational hypertension, preeclampsia, preterm labor, preterm premature rupture of the membranes (PROM), gestational diabetes mellitus (diabetes with onset during pregnancy and/or only symptomatic during pregnancy), placental abruption, placenta previa, preterm birth, very-preterm birth (less than 32 weeks of gestation), cesarean delivery, light (vaginal) bleeding, and heavy (vaginal) bleeding. Gestational age was assigned by first-trimester crown rump length.

The timing of perinatal loss was assessed. The following time intervals for perinatal loss were evaluated: fetal demise at less than 24 weeks, fetal demise at 24 or more weeks of gestation, and neonatal demise. Perinatal mortality was defined as the sum of these three categories.

Differences in background characteristics and pregnancy complications were compared by race using analysis of variance for continuous characteristics and χ^2 tests for categorical characteristics. Means and standard deviations of continuous variables and percentages of categorical variables were estimated for each race. Four distinct outcomes were considered: fetal demise at less than 24 weeks of gestation, fetal demise at 24 or more weeks of gestation, neonatal demise, and perinatal mortality. Estimates and 95% confidence intervals of the incidence of each outcome were calculated. Unadjusted and adjusted odds ratios with 95% confidence intervals were estimated for each race utilizing the white race as the referent group. Odds ratios were then adjusted for racial variations in demographics (Table 1), pregnancy complications (Table 2), and site of enrollment. In exploratory analyses the effect of race on perinatal mortality was investigated in specific subgroups.

RESULTS

A total of 35,529 pregnancies with early access to prenatal care were reviewed for this analysis. Race was distributed in the population in the following manner: 5% black, 22% Hispanic, 68% white, and 5% other. A total of fifteen patients indicated more than one race and were assigned to a single minority population. None of these patients experienced a perinatal loss. The demographic differences between the study populations are depicted in Table 1. Demographic characteristics were significantly different between the racial groups. In more general terms, members of all the minority races had higher BMIs, reported increased use of antihypertensive medication before pregnancy, and were more likely to have had pregestational diabetes than the white (referent) race. The black and Hispanic populations were



Table 1. Differences in Background Characteristics by Race

Characteristic	Black (n = 1,803)	Hispanic (n = 7,762)	White (n = 24,221)	Other (n = 1,743)	P
Mean (SD) maternal age (y)	28.7 (6.3)	27.6 (5.9)	31.7 (5.2)	30.9 (5.4)	< .001
Mean (SD) education (y)	13.5 (2.3)	12.1 (2.9)	15.1 (2.2)	15.1 (1.9)	< .001
Married (%)	694 (38.5)	3625 (46.7)	22138 (91.4)	1558 (89.4)	< .001
Mean (SD) body mass index	28.5 (7.2)	26.1 (5.4)	23.6 (4.6)	24.5 (4.9)	< .001
Tobacco use during pregnancy (%)	146 (8.1)	342 (4.4)	1138 (4.7)	52 (3.0)	< .001
Illicit drug* use during pregnancy (%)	49 (2.7)	78 (1.0)	218 (0.9)	14 (0.8)	< .001
Alcohol use during pregnancy (%)	20 (1.1)	85 (1.1)	630 (2.6)	37 (2.1)	< .001
Medication use during pregnancy (%)	600 (33.3)	1964 (25.3)	10149 (41.9)	640 (36.7)	< .001
Pregestational diabetes (%)	47 (2.6)	109 (1.4)	194 (0.8)	23 (1.3)	< .001
Previous live birth (%)	986 (54.7)	4657 (60.0)	12982 (53.6)	819 (47.0)	< .001
Previous miscarriage (%)	552 (30.6)	2034 (26.2)	6346 (26.2)	443 (25.4)	< .001
Previous preterm birth (%)	184 (10.2)	551 (7.1)	1550 (6.4)	106 (6.1)	< .001
Current pregnancy result of assisted reproductive technology (%)	36 (2.0)	85 (1.1)	1550 (6.4)	99 (5.7)	< .001
Antihypertensive medication use prior to pregnancy (%)	44 (2.4)	51 (0.7)	136 (0.6)	13 (0.8)	< .001

Data are presented as mean (standard deviation [SD]) or n (percentage).

All P values for analysis of variance tests for differences in means and χ^2 tests for differences in proportions across racial groups.

* Includes cocaine, marijuana, and heroin.

younger, had fewer years of formal education, and were less likely to be married than the white and other populations. The black population reported nearly twice the rate of smoking and four times the rate of antihypertensive medication use before pregnancy compared with the white population. The black population was also most likely to have used illicit drugs while members of the white population were most likely to have consumed alcohol during the pregnancy. The distribution of pregnancy complications

among the various races is depicted in Table 2. All minority races experienced higher rates of IUGR, preeclampsia, preterm PROM, gestational diabetes, placenta previa, preterm birth, very preterm birth, cesarean delivery, light vaginal bleeding, and heavy vaginal bleeding compared with the white population.

The frequency of perinatal mortality for the entire study population was 13 per 1,000 (471/35,529) and race specific perinatal mortality varied from 10 to 42 per 1,000 (Table 3). The incidence and timing of the

Table 2. Frequency of Pregnancy Complications by Race

Characteristic	Black (n = 1,803)	Hispanic (n = 7,762)	White (n = 24,221)	Other (n = 1,743)	P*
IUGR	31 (1.7)	101 (1.3)	242 (1.0)	21 (1.2)	< .009
Gestational hypertension	96 (5.3)	225 (2.9)	1211 (5.0)	61 (3.5)	< .001
Preeclampsia	67 (3.7)	210 (2.7)	484 (2.0)	38 (2.2)	< .001
Preterm labor	99 (5.5)	272 (3.5)	1356 (5.6)	91 (5.2)	< .001
Preterm PROM	49 (2.7)	140 (1.8)	363 (1.5)	31 (1.8)	< .002
Gestational diabetes	83 (4.6)	349 (4.5)	678 (2.8)	120 (6.9)	< .001
Placental abruption	13 (0.7)	31 (0.4)	194 (0.8)	14 (0.8)	< .003
Placenta previa	13 (0.7)	47 (0.6)	121 (0.5)	17 (1.0)	< .090
Preterm birth [†]	188 (10.4)	567 (7.3)	1574 (6.5)	131 (7.5)	< .001
Very preterm birth [‡]	97 (5.4)	186 (2.4)	315 (1.3)	35 (2.0)	< .001
Cesarean delivery	526 (29.2)	2065 (26.6)	5353 (22.1)	450 (25.8)	< .001
Light bleeding [§]	274 (15.2)	1025 (13.2)	3003 (12.4)	230 (13.2)	< .001
Heavy bleeding	40 (2.2)	186 (2.4)	315 (1.3)	31 (1.8)	< .001

IUGR, intrauterine growth restriction; PROM, premature rupture of membranes.

Data are presented as n (percentage).

* P values for χ^2 tests for differences in proportions across racial groups.

[†] Birth prior to 37 weeks of gestation.

[‡] Birth prior to 32 weeks of gestation.

[§] Vaginal spotting within the 4 weeks prior to study enrollment.

^{||} Vaginal bleeding similar to menses within the 4 weeks prior to study enrollment.



Table 3. Outcomes by Race

Outcome	Black (n = 1,803)	Hispanic (n = 7,762)	White (n = 24,221)	Other (n = 1,743)	P
Fetal demise at < 24 wk of gestation					
Rate (per 1,000)	29.4	11.1	6.9	10.3	–
Crude OR (95% CI)	4.4 (3.2–5.9)	1.6 (1.2–2.1)	–	1.5 (0.9–2.4)	< .001
Adjusted* OR (95% CI)	3.2 (2.2–4.8)	1.4 (1.0–2.1)	–	1.7 (1.0–2.7)	< .001
Fetal demise at ≥ 24 wk of gestation					
Rate (per 1,000)	8.3	3.9	2.4	4.0	–
Crude OR (95% CI)	3.5 (2.0–6.2)	1.6 (1.0–2.5)	–	1.7 (0.8–3.7)	< .001
Adjusted* OR (95% CI)	3.1 (1.5–6.2)	1.6 (0.8–2.9)	–	1.7 (0.8–3.8)	< .013
Neonatal demise [†]					
Rate (per 1,000)	4.4	0.9	0.7	2.3	–
Crude OR (95% CI)	6.3 (2.7–14.7)	1.3 (0.5–3.1)	–	3.3 (1.1–9.7)	< .001
Adjusted* OR (95% CI)	9.9 (3.2–30.5)	2.2 (0.7–6.4)	–	4.2 (1.4–13.0)	< .001
Perinatal mortality [‡]					
Rate (per 1,000)	42.1	15.9	10.0	16.6	–
Crude OR (95% CI)	4.3 (3.3–5.6)	1.6 (1.3–2.0)	–	1.7 (1.1–2.5)	< .001
Adjusted* OR (95% CI)	3.6 (2.6–5.0)	1.5 (1.1–2.1)	–	1.8 (1.2–2.7)	< .001
Perinatal mortality 2 [§]					
Rate (per 1,000)	12.7	4.8	3.1	6.3	–
Crude OR (95% CI)	4.2 (2.6–6.7)	1.5 (1.0–2.3)	–	2.0 (1.1–3.9)	< .001
Ajusted* OR (95% CI)	4.2 (2.3–7.5)	1.7 (1.0–2.9)	–	2.2 (1.2–4.2)	< .001

OR, odds ratio; 95% CI, 95% confidence interval.

P values for crude odds ratios based on χ^2 tests for differences in proportions across racial groups. P values for adjusted ORs based on multiple logistic regression analysis testing significance of ORs for each race as compared with white race.

* Odds ratios generated for each race as compared with white race, adjusting for maternal age, education, marital status, body mass index, tobacco use during pregnancy, illicit drug use during pregnancy, alcohol use during pregnancy, medication use during pregnancy, pregestational diabetes, obstetric history (history of previous live birth, miscarriage, and preterm delivery), use of assisted reproductive technologies, antihypertensive medication use prior to pregnancy, and site of enrollment.

[†] Due to the small numbers of neonatal demise, effects should be interpreted with caution.

[‡] Defined as fetal demise at less than 24 weeks of gestation plus fetal demise 24 or more weeks of gestation plus neonatal demise

[§] Defined as fetal demise 24 or more weeks of gestation plus neonatal demise.

perinatal loss sustained by each race are depicted in Table 3.

Utilizing the white population as the referent population, odds ratios with 95% confidence intervals were generated to explore the crude effect of race on the incidence of perinatal mortality: black 4.3 (3.3–5.6), Hispanic 1.6 (1.3–2.0), and other 1.7 (1.1–2.5). All minorities experienced significantly more perinatal mortality with the black population having the highest number, followed by the other and Hispanic populations, respectively (Table 3). The black population, which comprised only 5% of the study population, had 16% of the perinatal mortality. In addition, the black population sustained significantly more loss at each and every interval throughout the pregnancy and the neonatal period. After controlling for the 14 demographic characteristics listed in Table 1 and site of enrollment, race remained a significant predictor of perinatal mortality in all three groups (Table 3). To assess possible confounding effects of patient demographics, enrollment site, and pregnancy complications on the observed racial disparities, a fully adjusted model was developed. This model

incorporated all 14 variables in Table 1 and site of enrollment along with the following pregnancy complications: gestational hypertension, preeclampsia, IUGR, gestational diabetes, and preterm PROM. After adjustment for these variables, the following odds ratios (with 95% confidence intervals) for perinatal mortality were generated: black 3.5 (2.5–4.9), Hispanic 1.5 (1.2–2.1), and other 1.9 (1.3–2.8). Secondary exploratory analyses revealed the single most important predictor of perinatal mortality in the study population was preterm PROM. This complication occurred in 67 (14%) of the 471 perinatal losses. When the sample was stratified by the occurrence of preterm PROM (subgroups of women with and without preterm PROM) the effect of black race persisted in both subgroups. The odds ratios (95% confidence interval) for perinatal mortality in women with and without preterm PROM were 5.5 (2.3–13.0) and 2.8 (2.0–3.8), respectively, for black as compared with white women.

When the definition of perinatal mortality was restricted to a fetal demise at 24 or more weeks of gestation and a neonatal demise, the overall perinatal



mortality rate for the entire study population decreased to 4 per 1,000 (146/35,529). However, racial disparities in both the crude and adjusted models remain and are presented in Table 3.

DISCUSSION

Despite first-trimester initiation of prenatal care, minority pregnancies experienced more perinatal mortality when compared with pregnancies in the white population. The persistence of excess perinatal mortality in the minority populations after controlling for differences in patient demographics, pregnancy complications, and site of enrollment implicates race as an independent etiologic factor. However, the knowledge that genetic diversity exists as a continuum, with no clear genetic breaks delineating these categories,⁸ renders this conclusion untenable. Once an immutable genetic explanation is removed from consideration, optimism is fostered by the knowledge that all other causative factors should be responsive to the appropriate intervention(s).

Perhaps the etiology can be found not in race but in the components that are inherent to race. Cultural differences often parallel racial and ethnic lines and manifest as variations in nutrition, stress, and compliance with accepted medical standards as well as countless other variables. The ability to identify and capture the individual contributions of the innumerable components of a population's culture represents a formidable task. The difficulty of this task is only increased when the search is limited to the finite period of time encompassing a pregnancy. The integration of pediatric, obstetric, and geriatric databases enabling the study of generations of individual families has been proposed as a model to uncover and pursue the implications of such variables.⁹

Low birth weight is a significant contributor to neonatal and infant morbidity and mortality.¹⁰ Review of national data reveals a nearly 2-fold incidence of low birth weight in the black population compared with the white population.¹¹ However, birth weight patterns of infants of African-born blacks have been found to more closely resemble American-born whites than American-born blacks.¹² Similar negative effects on birth weight have been observed in the Hispanic community when stratifying by maternal natality.¹³ These observations suggest a specific etiologic factor or factors endemic to American-born minorities. The effect of birth weight was not assessed in the above analysis and may have contributed to the neonatal component of perinatal mortality.

The majority of the fetal losses in the study population occurred before 24 weeks of gestation.

The low perinatal mortality rate observed after the omission of this first interval was likely achieved by the exclusion of multiple gestations, major structural anomalies, and fetuses with aneuploidy. Preterm PROM occurred more often in minority races and may have functioned as a surrogate for intrauterine infection.

Among the strengths of this study is the prospective nature in which the data were collected and the inclusion of a large, unselected obstetric population. These patients represent those cared for by physicians in private offices in the various communities, resident clinics at numerous teaching centers, and the private offices of academicians in tertiary care medical centers.

Obstetric-related epidemiological questions have traditionally been addressed utilizing large state or national datasets based on birth certificate data. The validity of these datasets has been challenged in multiple publications.¹⁴⁻¹⁷ This study entailed a secondary analysis of the FASTER trial database which was compiled by trained research nurses through direct patient contact and chart review. However, the original study design was not powered to detect racial or ethnic disparities in perinatal mortality. Some caution is warranted in the interpretation of our results, especially in those outcomes with small numbers and wide confidence intervals. Nevertheless, these findings are among the largest prospective validations, in a contemporary population, of previously reported findings based on national data.^{1,18}

The factors commonly used in the determination of adequacy of care include the timing of initiation, total number of visits, and the gestational age of delivery.¹⁹ Although the timing of initiation of care may be stipulated by enrollment in the FASTER trial, the frequency of visits and its relationship to the timing of delivery were not evaluated. In addition, the content of prenatal visits was not assessed. Differences in content, specifically the degree in which ultrasonography, amniocentesis, and tocolytics are used, has been shown to vary between racial groups.²⁰

Membership in a particular race was self-reported by all patients included in the study. This technique may be limited by multiple factors, including the failure to identify patients and pregnancies of mixed racial heritage. The utilization of the recoding algorithm for women who indicated more than one race served only to decrease the rates of perinatal mortality in minority populations as none of these women had a perinatal loss. In addition, the extent of participation in activities which have known detrimental effects on obstetric performance, such as smoking and illicit



drug use during pregnancy, may have been underreported by study participants.

Racial disparities in health outcomes are prevalent in the United States. Review of national data from 1990 to 2002 demonstrates no change in the non-Hispanic black/non-Hispanic white ratio of perinatal mortality and a 5% increase in the fetal mortality ratio of these two groups.²¹ Differences in perinatal mortality comprise only a small piece of this complex puzzle. However, as the differences observed in the perinatal period and infancy are believed to play a role in the acquisition of pathology diagnosed later in life,²² the elimination of these racial disparities must be considered of paramount importance. There is no doubt that the future will bring increased perinatal survival and the improvement in survival will benefit all races. However, the disparities between white and nonwhite populations will continue unless specific etiologies are identified and interventions are developed. Prenatal care, although unequivocally helpful and necessary, remains insufficient in its present form for minority women. Therefore, increasing early access to current prenatal care systems in the effort to minimize racial and ethnic disparities in perinatal mortality is insufficient.

REFERENCES

1. National Center for Health Statistics. Infant, Fetal, and Maternal Mortality, United States 1963. Series 20, No. 3. Washington, DC: Public Health Services; Sept. 1966.
2. Vital and Health Statistics. Perinatal Mortality in the United States 1985–91. Series 20, No. 26. Washington, DC: Public Health Services; August 1995.
3. Ryan GM Jr, Sweeney PJ, Solola AS. Prenatal care and pregnancy outcome. *Am J Obstet Gynecol* 1980;137:876–81.
4. Foster DC, Guzick DS, Pulliam RP. The impact of prenatal care on fetal and neonatal death rates for uninsured patients: a “natural experiment” in West Virginia. *Obstet Gynecol* 1992; 79:40–5.
5. Vintzileos AM, Ananth CV, Smulian JC, Scorza WE. The impact of prenatal care on preterm births among twin gestations in the United States, 1989–2000. *Am J Obstet Gynecol* 2003;189:818–23.
6. Keeping JD, Chang AM, Morrison J, Esler EJ. Poor antenatal attendance and obstetric performance. *Aust N Z J Obstet Gynaecol* 1980;20:139–43.
7. Weiss JL, Malone FD, Vidaver J, Ball RH, Nyberg DA, Comstock CH, et al. Threatened abortion: a risk factor for poor pregnancy outcome, a population-based screening study. *Am J Obstet Gynecol* 2004;190:745–50.
8. Marshall E. DNA studies challenge the meaning of race. *Science* 1998;282:654–5.
9. Lu MC, Halfon N. Racial and ethnic disparities in birth outcomes: a life-course perspective. *Matern Child Health J* 2003;7:13–30.
10. Shapiro S, McCormick MC, Strafield BH, Krischer JP, Bross D. Relevance of correlates of infant deaths for significant morbidity at 1 year of age. *Am J Obstet Gynecol* 1980;136: 363–73.
11. National Center for Health Statistics. Birth: Preliminary Data for 2003. Volume 53, No. 9. Washington, DC: Public Health Services; August 2004.
12. David RJ, Collins JW Jr. Differing birth weight among infants of U.S.-born blacks, African-born blacks, and U.S.-born whites. *N Engl J Med* 1997;337:1209–14.
13. Fuentes-Afflick E, Hessol NA, Perez-Stable EJ. Maternal birth-place, ethnicity, and low birth weight in California. *Arch Pediatr Adolesc Med* 1998;152:1105–12.
14. Piper JM, Mitchel EF Jr, Snowden M, Hall C, Adams M, Taylor P. Validation of 1989 Tennessee birth certificates using maternal and newborn hospital records. *Am J Epidemiol* 1993;137:758–68.
15. Buescher PA, Taylor KP, Davis MH, Bowling JM. The quality of the new birth certificate data: a validation study in North Carolina. *Am J Public Health* 1993;83:1163–5.
16. Dobie SA, Baldwin LM, Rosenblatt RA, Fordyce MA, Andrilla CH, Hart LG. How well do birth certificates describe the pregnancies they report? The Washington State experience with low-risk pregnancies. *Matern Child Health J* 1998;2:145–54.
17. Lydon-Rochelle MT, Holt VL, Cárdenas V, Nelson JC, Easterling TR, Gardella C, et al. The reporting of pre-existing maternal medical conditions and complications of pregnancy on birth certificates and in hospital discharge data. *Am J Obstet Gynecol* 2005;193:125–34.
18. Vintzileos AM, Ananth CV, Smulian JC, Scorza WE, Knuppel RA. Prenatal care and black-white fetal death disparity in the United States: heterogeneity by high-risk conditions. *Obstet Gynecol* 2002;99:483–89.
19. Alexander GR, Kotelchuck M. Quantifying the adequacy of prenatal care: a comparison of indices. *Public Health Reports* 1996;111:408–18.
20. Brett KM, Schoendorf KC, Kiely JL. Differences between black and white women in the use of prenatal care technologies. *Am J Obstet Gynecol* 1994;170:41–6.
21. Martin JA, Kochanek KD, Strobino DM, Guyer B, MacDorman MF. Annual summary of vital statistics: 2003. *Pediatrics* 2005;115:619–34.
22. Barker DJ. Fetal origins of coronary heart disease. *BMJ* 1995; 311:171–4.

APPENDIX

Members of the FASTER Research Consortium: K. Welch, MS, R. Denchy, MS (Columbia University, NY, NY); R. Ball, MD, M. Belfort, MD, B. Oshiro, MD, L. Cannon, BS, K. Nelson, BSN, C. Loucks, RNC, A. Yoshimura (University of Utah, and IHC Perinatal Centers, Salt Lake City, Provo, and Ogden, UT); D. Nyberg, MD, S. Coe, MS (Swedish Medical Center, Seattle, WA); D. Schmidt, MS, J. Esler, BS (William Beaumont Medical Center, Royal Oak, MI); R. Bukowski, MD, G. Hankins, MD, J. Lee, MS, (UTMB Galveston, TX); K. Eddleman, MD, Y. Kharbutli, MS (Mount Sinai Medical Center, NY, NY); I. Merkatz, MD, S. Gross, MD, S. Carter, MS (Montefiore Medical Center, Bronx, NY); J. Hobbins, MD,



L. Schultz, RN (University of Colorado Health Science Center, Denver, CO); M. Paidas, MD, J. Borsuk, MS (NYU Medical Center, NY, NY); B. Isquith, MS, B. Berlin, MS (Tufts University, Boston, MA); J. Canick, PhD, G. Lambert-Messerlian, PhD, C. Duquette, RDMS (Brown University, Providence, RI);

R. Baughman, MS (University of North Carolina, Chapel Hill, NC); J. Hanson, MD, F. de la Cruz, MD (National Institute of Child Health and Human Development); J. Vidaver, MA, T. Tripp, MA, D. Emig, MPH, K. Dukes, PhD (DM-STAT, Inc, Medford, MA).



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