Racial, Ethnic, and Socioeconomic Disparities in the Prevalence of Cerebral Palsy

WHAT'S KNOWN ON THIS SUBJECT: Black infants have an increased risk of cerebral palsy when compared with white infants. The reason for this racial disparity is unclear. Lower socioeconomic status may be associated with an increased risk of cerebral palsy.

WHAT THIS STUDY ADDS: The increase in cerebral palsy among blacks is due to higher rates of low birth weight. Among low birth weight infants, blacks are less likely to have cerebral palsy than whites. Prenatal care and maternal education also impact cerebral palsy risk.

abstract

OBJECTIVE: Racial and ethnic disparities in cerebral palsy have been documented, but the underlying mechanism is poorly understood. We determined whether low birth weight accounts for ethnic disparities in the prevalence of cerebral palsy and whether socioeconomic factors impact cerebral palsy within racial and ethnic groups.

METHODS: In a retrospective cohort of 6.2 million births in California between 1991 and 2001, we compared maternal and infant characteristics among 8397 infants with cerebral palsy who qualified for services from the California Department of Health Services and unaffected infants.

RESULTS: Overall, black infants were 29% more likely to have cerebral palsy than white infants (relative risk: 1.29 [95% confidence interval: 1.19–1.39]). However, black infants who were very low or moderately low birth weight were 21% to 29% less likely to have cerebral palsy than white infants of comparable birth weight. After we adjusted for birth weight, there was no difference in the risk of cerebral palsy between black and white infants. In multivariate analyses, women of all ethnicities who did not receive any prenatal care were twice as likely to have infants with cerebral palsy relative to women with an early onset of prenatal care. Maternal education was associated with cerebral palsy in a dose-response fashion among white and Hispanic women. Hispanic adolescent mothers (aged <18 years) had increased risk of having a child with cerebral palsy.

CONCLUSIONS: The increased risk of cerebral palsy among black infants is primarily related to their higher risk of low birth weight. Understanding how educational attainment and use of prenatal care impact the risk of cerebral palsy may inform new prevention strategies. *Pediatrics* 2011;127:e674–e681

AUTHORS: Yvonne W. Wu, MD, MPH,^{a,b} Guibo Xing, PhD,^c Elena Fuentes-Afflick, MD, MPH,^{b,d} Beate Danielson, PhD,^e Lloyd H. Smith, MD, PhD,^c and William M. Gilbert, MD^{c,f}

^aDepartment of Neurology, ^bPediatrics, and ^dEpidemiology and Biostatistics, University of California San Francisco, San Francisco, California; ^cDepartment of Obstetrics and Gynecology, University of California Davis, Sacramento, California; ^eHealth Information Solutions, Rocklin, California; and ^fSutter Medical Center, Sacramento, California

KEY WORDS

cerebral palsy, racial disparity, epidemiology, sociodemographic

ABBREVIATIONS

DDS—California Department of Developmental Services

RR—relative risk

CI—confidence interval

OR—odds ratio

www.pediatrics.org/cgi/doi/10.1542/peds.2010-1656

doi:10.1542/peds.2010-1656

Accepted for publication Dec 13, 2010

Address correspondence to Yvonne W Wu, MD, MPH, UCSF Department of Child Neurology, Box 0137, 350 Parnassus Ave, Suite 609, San Francisco, CA 94143-0137. E-mail: wuy@neuropeds.ucsf.edu

PEDIATRICS (ISSN Numbers: Print, 0031-4005; Online, 1098-4275).

Copyright © 2011 by the American Academy of Pediatrics

FINANCIAL DISCLOSURE: The authors have indicated that they have no personal financial relationships relevant to this article to disclose.

The rate of cerebral palsy in the United States has remained constant or has increased slightly in recent decades despite improvements in obstetric and perinatal care.^{1,2} Population-based studies^{1,3,4} have reported that black infants have an increased risk of cerebral palsy when compared with white infants. The underlying reason for this racial disparity is unknown. Black people have higher rates of low birth weight and prematurity than whites, which suggests that birth weight or gestational age could contribute to ethnic disparities in cerebral palsy. However, in studies of infants born term or with normal birth weight, blacks demonstrate a 20% to 40% increased risk of cerebral palsy compared with whites, suggesting that gestational age is unlikely to explain disparities in prevalence cerebral palsy.^{5,6} On the other hand, Hispanic infants are at decreased risk of perinatal morbidity and mortality.⁷ Whether infants born to Hispanic women experience a decreased risk of cerebral palsy has not been well studied.

Socioeconomic factors also may influence the risk of cerebral palsy.^{8,9} In West Sussex, England, low socioeconomic status was associated with elevated risk of cerebral palsy, and adjustment for birth weight and gestational age did not account for this increased risk.⁸ The mechanism by which socioeconomic status and race interact to impact an individual's risk of cerebral palsy has not been adequately studied. To assess the relationship between ethnicity and low birth weight with respect to the prevalence of cerebral palsy, and to determine whether socioeconomic status impacts the risk of cerebral palsy within racial/ethnic groups, we studied a large, multiethnic population in California.

METHODS

We conducted a retrospective cohort study of birth records for all infants who were born in California during an 11-year period (January 1, 1991, to December 31, 2001). Data were retrieved from 3 sources: (1) the California Office of Statewide Health Planning and Development Patient Discharge Diagnoses; (2) the California Department of Health Services Linked Vital Statistics Birth and Infant Death file; and (3) the California Department of Developmental Services (DDS), a statewide program that provides services to children with cerebral palsy. All study procedures were approved by the California Protection of Human Subjects Committee and by the institutional review boards at the California Office of Statewide Health Planning and Development; the University of California, San Francisco; and the University of California, Davis.

The California Office of Statewide Health Planning and Development maintains a database of discharge abstracts for all admissions to nonfederal hospitals, which represent 96% (571 of 594) of all hospitals in the state. According to the 1991–1998 California natality figures from the Centers for Disease Control and Prevention,¹⁰ 96.7% of all live births in California were recorded in the California Office of Statewide Health Planning and Development hospital discharge data set. All maternal and infant predictor variables were extracted from the linked California Office of Statewide Health Planning and Development–California Vital Statistics Birth files: maternal age; race and ethnicity; education; timing of onset of prenatal care; infant gender; birth weight; gestational age; and source of payment for the birth hospitalization, which was analyzed as an indicator of socioeconomic status. Women who were publicly insured or uninsured were categorized as having

low insurance status, whereas women with private or managed care insurance were categorized as having high insurance status. We defined 3 birth weight categories: normal (≥ 2500 g), moderately low (1500–2499 g), and very low (< 1500 g).

The DDS runs a statewide program that provides occupational and physical therapy, case management, and social services for all residents who have a substantive disability related to cerebral palsy.¹¹ Cerebral palsy is defined as follows: (1) a nonprogressive lesion or disorder in the brain occurring during intrauterine life or the perinatal period and characterized by paralysis, spasticity, or abnormal control of movement or posture that is manifest before 2 to 3 years of age; and (2) other significant motor dysfunction appearing before age 18 years. Each year, individuals who receive services from the DDS receive a comprehensive evaluation by a staff physician who records data regarding medical diagnoses including cerebral palsy.

From the DDS data set, we identified all children who were born between 1991 and 2001 and who had been diagnosed with cerebral palsy before November 30, 2006. Thus, children were at least 5 years old at the time of cerebral palsy ascertainment. Because we were interested in prenatal and perinatal risk factors for cerebral palsy, we excluded children with certain postnatal causes of cerebral palsy, including child abuse ($n = 272$), motor vehicle and other vehicle injuries ($n = 213$), and near drowning ($n = 72$). For patients with cerebral palsy who did not meet exclusion criteria, we extracted the following clinical information from the DDS: type of cerebral palsy (spastic, dyskinetic [dystonia or choreoathetosis], hypotonic, ataxic, and other) and distribution of limb involvement (quadriplegia, diplegia, hemiplegia, monoplegia, and other). Cerebral

palsy severity was determined by the degree of limitation of daily activities and functions: severe cerebral palsy is defined as significant limitations or precludes daily activities and functions; moderate is defined as having some limitations; mild is defined as having no limitations. Suspected cerebral palsy refers to patients with a suspected disability from cerebral palsy, but the level of impairment has not been determined by the DDS.

To assess ethnic disparities in the risk of cerebral palsy, we compared with white children the risk of cerebral palsy among black Asian, Hispanic, and other children. We calculated univariate relative risk (RRs) and 95% confidence intervals (CIs) and stratified the results by birth weight and gestational age to determine whether low birth weight or prematurity explain observed differences in the risk of cerebral palsy. We then determined the independent contribution of socioeconomic factors to the risk of cerebral palsy within each racial and ethnic group by performing stratified multivariable logistic regression analyses to estimate odds ratios (ORs). The logistic regression models included potentially modifiable sociodemographic factors (maternal age, education, onset of prenatal care, and insurance status) as well as infant gender, a consistently strong risk factor for cerebral palsy. Note that birth weight and low gestational age were not included in these multivariate logistic regression models because these may represent downstream effects of low socioeconomic status, and controlling for these intermediary variables would reduce our ability to observe the effect of socioeconomic factors on cerebral palsy. All ORs closely approximate the RR given the low prevalence of cerebral palsy. Analyses were performed using Stata statistical package (version 9.0; Stata Corpora-

TABLE 1 Clinical Characteristics of 8397 Infants With Cerebral Palsy Among 6.2 Million Births in California, 1991–2001

	Percentage
Type of cerebral palsy	
Spastic	60.5
Hypotonic	14.4
Ataxic	5.6
Dyskinetic	2.7
Other	16.9
Type of limb involvement	
Quadriplegia	52.2
Paraplegia	20.1
Hemiplegia	14.4
Monoplegia	2.0
Other	11.3
Severity of motor impairment	
Severe	34.7
Moderate	43.9
Mild	17.7
Suspected	3.7

tion, College Station, TX) and with SAS statistical package (version 9.2; SAS Institute Inc, Cary, NC).

RESULTS

Among 6 221 001 live births in California from 1991 to 2001, 8397 children with cerebral palsy provided an overall prevalence of 1.4 per 1000 live births. Over the 10-year study period, the prevalence of cerebral palsy did not vary significantly by birth year (data not shown). Almost 63% of cerebral palsy cases were of the spastic or dyskinetic subtypes (Table 1). Quadriplegia was the most common distribution of limb involvement, followed by paraparesis and hemiparesis. Three-quarters of cases were categorized as moderately severe or severe.

In crude assessments of ethnic disparities in cerebral palsy, black children were 29% more likely to have cerebral palsy than white children (Table 2). Asian children were 20% less likely to have cerebral palsy than white children, and there was no difference between Hispanic and white children. Women who were younger (aged <18 years), older (aged ≥35 years), had no

prenatal care, or had low insurance status had an increased risk of having a child with cerebral palsy. Compared with women with a college diploma, women with lower educational attainment were more likely to have a child with cerebral palsy. The effect of maternal education on the risk of cerebral palsy demonstrated an inverse dose-response relationship; compared with college graduates, mothers with primary school or no education (RR: 1.33) demonstrated the highest risk, whereas mothers with a high school diploma (RR: 1.21) or some college (RR: 1.14) exhibited a more modest increased risk of cerebral palsy (Table 2).

Thirty-seven percent of children with cerebral palsy were born at moderately low (16%) or very low birth weight (21%). Very low birth weight infants were 24 times more likely to have cerebral palsy than normal birth weight infants, and very premature infants (<28 weeks' gestation) were 26 times more likely to have cerebral palsy than infants born term (Table 2).

Black children were more likely than white children to have been born at very low (3.5% vs 1.3%, $P = .0001$) or moderately low (9.5% vs 4.6%, $P = .0001$) birth weight. Similarly, black children were more likely to have been born premature (<37 weeks' gestation) than white infants (20.4% vs 11.9%, $P = .0001$). Asian children were less likely to be born at very low birth weights than white children (1.2% vs 1.3%, $P = .02$) but more likely to have been born with moderately low birth weight (6.5% vs 5.1%, $P = .0001$) when compared with white children.

To assess whether ethnic differences in birth weight or gestational age explain ethnic disparities in the risk of cerebral palsy, we performed stratified analyses (Table 3). Among normal birth weight infants, there was no difference in the risk of cerebral palsy

TABLE 2 Univariate Risks for Cerebral Palsy Among 6.2 Million Live Births in California, 1991–2001

	Cerebral Palsy Cases		Incidence per 1000 Live Births	RR		95% CI		<i>P</i>
	<i>n</i>	%						
Maternal ethnicity								
Hispanic	3963	47.2	1.42	1.04	0.99	—	1.09	.11
White	2878	34.3	1.36	1.00		Reference		Reference
Black	758	9.0	1.75	1.29	1.19	—	1.39	<.0001
Asian	656	7.8	1.09	0.80	0.74	—	0.87	<.0001
Other	105	1.3	1.37	1.01	0.83	—	1.22	.96
Maternal age, y								
<18	439	5.2	1.62	1.24	1.12	—	1.36	<.0001
18–34	6504	77.5	1.31	1.00		Reference		Reference
≥35	1451	17.3	1.75	1.33	1.26	—	1.41	<.0001
Maternal education, y								
Primary school or none (0–5 y)	438	5.2	1.57	1.33	1.20	—	1.49	.0001
Secondary school (6–11 yy)	2514	29.9	1.48	1.26	1.18	—	1.35	.0001
High school graduate (12 y)	2514	29.9	1.43	1.21	1.13	—	1.30	.0001
Some college (13–15 y)	1568	18.7	1.34	1.14	1.06	—	1.23	.0006
College graduate (≥16 y)	1273	15.2	1.18	1.00		Reference		Reference
Low insurance status	4393	52.3	1.46	1.12	1.07	—	1.16	<.0001
Onset of prenatal care								
No prenatal care	205	2.4	3.18	2.35	2.05	—	2.70	<.0001
Third trimester	288	3.4	1.48	1.09	0.97	—	1.23	.16
Second trimester	1332	15.9	1.39	1.03	0.97	—	1.09	.35
First trimester	6454	76.9	1.36	1.00		Reference		Reference
Birth weight, g								
Very low (<1500)	1766	21.0	22.0	23.60	22.3	—	24.9	<.0001
Moderately low (1500–2499)	1318	15.7	4.70	5.06	4.76	—	5.37	<.0001
Normal (≥2500)	5309	63.2	0.93	1.00		Reference		Reference
Gestational age, wk								
<28	924	11.0	25.94	26.67	25.48	—	29.36	<.0001
28–31	672	8.0	14.92	15.34	14.35	—	16.87	<.0001
32–36	1137	13.5	2.40	2.47	2.32	—	2.63	<.0001
≥37	5029	59.9	0.97	1.00		Reference		Reference
Infant gender, male	4871	58.0	1.57	1.32	1.26	—	1.38	<.0001

TABLE 3 Race As a Risk Factor for Cerebral Palsy, Stratified by Infant Birth Weight and Gestational Age

	Very Low Birth Weight (<1500 g), <i>n</i> = 1765 Cerebral Palsy			Moderately Low Birth Weight (1500– 2400 g), <i>n</i> = 1318 Cerebral Palsy			Normal Birth Weight (≥2500 g), <i>n</i> = 5309 Cerebral Palsy		
	RR	95% CI	<i>P</i>	RR	95% CI	<i>P</i>	RR	95% CI	<i>P</i>
Maternal ethnicity									
Hispanic	1.06	0.95–1.19	.27	0.90	0.80–1.02	.11	1.03	0.97–1.09	.32
White	1.00	Reference	Reference	1.00	Reference	Reference	1.00	Reference	Reference
Black	0.79	0.68–0.91	.002	0.71	0.59–0.85	.0002	1.00	0.89–1.12	.98
Asian	0.72	0.59–0.88	.002	0.63	0.51–0.77	.001	0.82	0.74–0.91	.0002
	<32 weeks' gestation, <i>n</i> = 1596			32–36 weeks' gestation, <i>n</i> = 1137			≥37 weeks' gestation, <i>n</i> = 5029		
Maternal ethnicity									
Hispanic	0.99	0.89–1.12	.91	0.78	0.69–0.89	.00	1.05	0.99–1.12	.10
Non-Hispanic white	1.00	Reference	Reference	1.00	Reference	Reference	1.00	Reference	Reference
Black	0.81	0.69–0.95	.01	0.95	0.8–1.16	.58	1.04	0.9–1.16	.56
Asian	0.66	0.53–0.83	.00	0.76	0.6–0.94	.01	0.84	0.8–0.93	.00

between blacks and whites (RR: 1.00 [95% CI: 0.89–1.12]). In contrast, black infants who were born of moderately low (RR: 0.71 [95% CI: 0.59–0.85]) or very low (RR: 0.79 [95% CI: 0.68–0.91]) birth weight were less likely to be diag-

nosed with cerebral palsy than comparable white infants. Similar results were obtained when we stratified by gestational age. That is, although there was no difference in the risk of cerebral palsy between black and white in-

fants born term (RR: 1.04 [95% CI: 0.9–1.16]), very premature (<31 weeks' gestation) black infants were less likely to have cerebral palsy than comparable white infants (RR: 0.81 [95% CI: 0.69–0.95]).

TABLE 4 Independent Effects of Sociodemographic Factors on Risk of Cerebral Palsy in a Multivariate Model, Stratified by Racial and Ethnic Groups^a

	White, <i>n</i> = 2878 CP			Black, <i>n</i> = 758 CP			Hispanic, <i>n</i> = 3963 CP			Asian, <i>n</i> = 656 CP		
	OR	95% CI	<i>P</i>	OR	95% CI	<i>P</i>	OR	95% CI	<i>P</i>	OR	95% CI	<i>P</i>
Maternal age, y												
<18	1.0	0.8–1.3	.89	1.3	0.97–1.8	.08	1.2	1.02–1.3	.03	1.2	0.65–2.0	0.61
18–34		Reference	Reference	1.0	Reference	Reference	1.0	Reference	Reference	1.0	Ref	Ref
≥35	1.4	1.3–1.6	<.0001	1.1	0.9–1.4	.28	1.5	1.3–1.6	<.0001	1.3	1.1–1.6	0.003
Maternal education												
Primary school or none	1.7	0.96–2.9	.07	0.6	0.1–2.4	.45	1.3	1.04–1.5	.02	1.4	0.97–1.9	0.07
Secondary school	1.4	1.2–1.6	<.0001	1.1	0.8–1.5	.58	1.2	0.99–1.4	.07	1.2	0.93–1.7	0.15
High school graduate	1.3	1.1–1.4	<.0001	1.2	0.9–1.6	.11	1.1	0.96–1.3	.15	1.1	0.84–1.3	0.66
Some college	1.1	1.00–1.2	.05	1.1	0.9–1.5	.33	1.1	0.9–1.3	.27	1.3	1.05–1.6	0.01
College graduate		Reference	Reference	1.0	Reference	Reference	1.0	Reference	Reference	1.0	Ref	Ref
Low insurance status	1.1	1.0–1.2	.001	1.0	0.9–1.2	.97	1.0	0.9–1.1	.95	1.0	0.83–1.2	0.95
Onset of prenatal care												
No prenatal care	2.2	1.7–3	<.0001	2.0	1.3–3	.001	2.3	1.9–2.8	<.0001	2.0	0.84–4.9	0.12
Third trimester	1.0	0.8–1.3	.81	1.0	0.7–1.5	.96	1.0	0.9–1.2	.69	1.0	0.59–1.7	0.96
Second trimester	0.9	0.8–1	.21	1.0	0.8–1.2	.87	1.0	0.9–1.1	.76	0.9	0.73–1.2	0.53
First trimester		Reference	Reference	1.0	Reference	Reference	1.0	Reference	Reference	1.0	Ref	Ref
Infant gender, male	1.3	1.2–1.4	<.0001	1.4	1.2–1.6	<.0001	1.3	1.2–1.4	<.0001	1.5	1.27–1.7	<.0001

^a Each logistic regression model includes all the variables listed in this table.

In a logistic regression model, black race was no longer a risk factor for cerebral palsy after controlling for birth weight and, in fact, was associated with decreased risk (OR: 0.87 [95% CI: 0.80–0.94]). Similarly, controlling for gestational age eliminated the apparent adverse effect of black race on cerebral palsy (OR: 0.95 [95% CI: 0.87–1.03]). Thus, both stratified and logistic regression analyses suggest that the increased rate of low birth weight and prematurity alone accounts for the increased risk of cerebral palsy observed in black infants.

In contrast, controlling for maternal education and insurance status in a logistic regression model did not eliminate the increased risk of cerebral palsy observed in blacks (OR: 1.3 [95% CI: 1.2–1.4]), suggesting that low socioeconomic status does not fully explain the racial disparity between blacks and whites. Yet socioeconomic markers were associated with cerebral palsy in crude analyses (Table 2). Furthermore, maternal race was highly correlated with maternal educational attainment; for instance, 84% of mothers with the lowest educational level (0–5 years) were Hispanic. Maternal race also was correlated with insur-

ance status. Hispanics had the highest rate of government or no insurance (62%) and Asians had the lowest rate (6%). Therefore, to determine the independent contributions of sociodemographic risk factors for cerebral palsy within each racial and ethnic subgroup, we performed stratified multivariate analyses.

Among whites, decreasing maternal educational attainment was independently associated with increasing risk of cerebral palsy in a dose-response fashion (Table 4). In multivariate analysis, Hispanic women with 0 to 5 years of education also were more likely to have a child with cerebral palsy than their counterparts who were college graduates (OR: 1.3 [95% CI: 1.04–1.5]). In contrast, maternal education was not associated with cerebral palsy risk among blacks.

Black Hispanic, and white women who delivered without receiving prenatal care had a significant twofold increased risk of cerebral palsy, compared with their counterparts who received early prenatal care (Table 4). For these 3 racial and ethnic groups, lack of prenatal care was associated with an increased risk of cerebral

palsy even after adjusting for birth weight gestational age (data not shown). Asian women who did not receive prenatal care also demonstrated a twofold increased risk of cerebral palsy, although this was not a statistically significant finding.

Older Asian, Hispanic, and white mothers were 32% to 45% more likely to have a child with cerebral palsy than women who were aged 18 to 34 years (Table 4). Adolescent childbearing was significantly associated with cerebral palsy only among Hispanic women (OR: 1.2 [95% CI: 1.02–1.3]), although a similar trend was seen in blacks (OR: 1.3 [95% CI: 0.97–1.8]).

When we restricted the analyses in Tables 3 and 4 to children with moderate or severe cerebral palsy, the findings remained essentially unchanged, except the effect sizes became slightly larger (data not shown). In additional sensitivity analyses, we limited the stratified analyses to children with spastic or dyskinetic cerebral palsy, because this group is likely to be etiologically distinct from infants with hypotonic and ataxic cerebral palsy. The significant findings in Tables 3 and 4 remained unchanged when the analy-

ses were restricted to spastic or dyskinetic cerebral palsy. However, the risk of spastic or dyskinetic cerebral palsy was elevated in blacks compared with whites both among normal birth weight (RR: 1.3, $P = .0004$) and among infants of term gestational age (RR: 1.3, $P = .0004$). Hispanics were similarly at increased risk of spastic or dyskinetic cerebral palsy when compared with whites, in both normal birth weight (RR: 1.1, $P = .02$) and term gestational age (RR: 1.5, $P = .001$) strata. In contrast, black infants were less likely than white infants to have ataxic cerebral palsy (RR: 0.6 [95% CI: 0.3–0.97]) or hypotonic cerebral palsy (RR: 0.4 [95% CI: 0.2–0.5]).

DISCUSSION

This population-based study of California children confirms previous findings of an elevated risk of cerebral palsy among black infants^{1,3,12} and suggests that this increased risk is explained by increased rates of low birth weight and prematurity. Relative to white children, black children have a two- to threefold increased risk of being born with low birth weight, and the dramatically increased risk of cerebral palsy in very low and moderately low birth weight infants accounts for the increased rate of cerebral palsy in blacks. Within the 2 subgroups of low birth weight infants that we studied, we found that black infants have a paradoxically reduced risk of cerebral palsy when compared with white infants. However, because black infants have higher rates of low birth weight infants, and low birth weight is so strongly associated with cerebral palsy, the relatively lower rate of cerebral palsy among low birth weight black infants does not fully remove the overall ethnic disparity that is observed on a population level.

The birth weight paradox^{13,14} refers to the fact that black premature infants

experience lower neonatal mortality rates than white premature infants, although black infants generally have a higher neonatal mortality rate than white infants.^{13,14} We found a similar paradoxical relationship between low birth weight and cerebral palsy among black infants. These findings are consistent with the results of a previous study⁶ that reported that black low birth weight infants had a lower cerebral palsy risk when compared with white low birth weight infants, although the study sample was smaller and the ethnic difference did not achieve statistical significance.

Low birth weight infants are about twice as common in blacks as in whites,¹⁵ and despite improvements in the 1990s, the preterm birth rate among black women began increasing in 2000.¹⁶ Prematurity accounts for most of the disparity in infant mortality between black and white infants.¹⁷ In our study of California births, we found that preterm birth and low birth weight completely accounted for the increased risk of cerebral palsy among black infants. Thus, to address the racial disparity in cerebral palsy rates, it is crucial to focus efforts to reduce the incidence of both prematurity and low birth weight.

The most commonly cited explanations for racial disparities in prematurity include suboptimal prenatal care,^{18,19} maternal education,²⁰ neighborhood poverty and disparity,^{21,22} social stressors,²³ and genetic differences.²⁴ Nativity is an important factor because the birth weight distribution of African-born black women more closely resemble American-born whites than American-born blacks,²⁵ suggesting that environmental factors play a key role in determining risk of prematurity and low birth weight.

Maternal age and low socioeconomic status are both known risk factors for cerebral palsy and neonatal encephalopathy.^{1,8,9,26,27}

In our population, advanced maternal age and low maternal educational attainment increased the risk of cerebral palsy in whites, Hispanics and Asians but not blacks. In contrast, young maternal age was a significant risk factor for cerebral palsy among Hispanics, with a similar trend seen in blacks. Because the number of black children with cerebral palsy was 5 times lower than the number of Hispanic children with cerebral palsy, we had less power to detect statistically significant risk factors among blacks.

There is an increased risk of cerebral palsy among male children across all gestational ages for unclear reasons.^{1,26,28,29} Possible explanations include hormonal factors and gender differences in cell-death pathways in the setting of newborn brain injury.³⁰ How prenatal care might impact cerebral palsy risk also is unclear, and in our population, the increased risk of cerebral palsy associated with poor prenatal care persisted even after adjusting for low birth weight and prematurity. Epidemiologic studies of prenatal care have not consistently demonstrated a beneficial effect on low birth weight and other perinatal outcomes.³¹ Our findings suggest that receipt of prenatal care may be associated with lower cerebral palsy risk. However, randomized controlled trials of differing degrees and types of prenatal care would be necessary to establish a causal link between prenatal care and cerebral palsy.³¹

Asian infants had a reduced risk of cerebral palsy across all birth weight categories, and this effect persisted in all birth weight and gestational age groups. This finding is consistent with data suggesting that Asian Americans experience improved neonatal outcomes when compared with whites^{32,33} and confirms the results of a previous study⁵ that reported that Asians have a

lower cerebral palsy rate than whites. However, perinatal outcomes vary widely between different Asian American subgroups,^{34,35} and additional study is needed to determine whether the risk of cerebral palsy varies by Asian subgroup. Among normal birth weight and term infants, blacks and Hispanics had a significantly increased risk of spastic or dyskinetic cerebral palsy compared with whites. Spastic or dyskinetic cerebral palsy is the most common form of cerebral palsy that is attributable to birth asphyxia.³⁶ Thus, our results suggest that the disparity in spastic or dyskinetic cerebral palsy rates observed between black and white infants may be attributed in part to differing rates of birth asphyxia among term or normal birth weight infants. Although beyond the scope of this study, future epidemiologic investigations of race, birth complications, and long-term neurologic outcomes may further clarify the impact of race/ethnicity on cerebral palsy.

REFERENCES

1. Yeargin-Allsopp M, Van Naarden Braun K, Doernberg NS, Benedict RE, Kirby RS, Durkin MS. Prevalence of cerebral palsy in 8-year-old children in three areas of the United States in 2002: a multisite collaboration. *Pediatrics*. 2008;121(3):547–554
2. Clark SL, Hankins GD. Temporal and demographic trends in cerebral palsy: fact and fiction. *Am J Obstet Gynecol*. 2003;188(3):628–633
3. Murphy CC, Yeargin-Allsopp M, Decoufle P, Drews CD. Prevalence of cerebral palsy among ten-year-old children in metropolitan Atlanta, 1985 through 1987. *J Pediatr*. 1993;123(5):S13–S20
4. Wu YW, Escobar GJ, Grether JK, Croen LA, Greene JD, Newman TB. Chorioamnionitis and cerebral palsy in term and near-term infants. *JAMA*. 2003;290(20):2677–2684
5. Wu YW, Croen LA, Shah SJ, Newman TB, Najjar DV. Cerebral palsy in a term population: risk factors and neuroimaging findings. *Pediatrics*. 2006;118(2):690–697
6. Winter S, Autry A, Boyle C, Yeargin-Allsopp M. Trends in the prevalence of cerebral palsy in a population-based study. *Pediatrics*. 2002;110(6):1220–1225
7. Hessel NA, Fuentes-Afflick E. The perinatal advantage of Mexican-origin Latina women. *Ann Epidemiol*. 2000;10(8):516–523
8. Sundrum R, Logan S, Wallace A, Spencer N. Cerebral palsy and socioeconomic status: a retrospective cohort study. *Arch Dis Child*. 2005;90(1):15–18
9. Dolk H, Pattenden S, Johnson A. Cerebral palsy, low birthweight and socio-economic deprivation: inequalities in a major cause of childhood disability. *Paediatr Perinat Epidemiol*. 2001;15(4):359–363
10. CDC Wonder [Web site], 2006. Available at: <http://wonder.cdc.gov>. Accessed May 7, 2006
11. State of California Department of Developmental Services. Information about developmental disabilities [article online], 2007. Available at: www.dds.ca.gov/general/info_about_dd.cfm. Accessed May 20, 2010
12. Wu YW, Colford JM. Chorioamnionitis as a risk factor for cerebral palsy: a meta-analysis. *JAMA*. 2000;284(11):1417–1424
13. Bruckner TA, Saxton KB, Anderson E, Goldman S, Gould JB. From paradox to disparity: need, the racial and ethnic make-up of DDS clients in 2007 is not identical to our study population (39% vs 34% whites, 33% vs 45% Hispanics, 10% vs 7% blacks, and 8% vs 10% Asians).

CONCLUSIONS

Sociodemographic factors interact in complex ways to influence the risk of cerebral palsy. Our results suggest that reducing ethnic disparities in prematurity and low birth weight would be expected to reduce disparities in cerebral palsy. Understanding how educational attainment and use of prenatal care impact the risk of cerebral palsy within specific ethnic groups may inform new strategies to prevent cerebral palsy.

ACKNOWLEDGMENTS

This study was funded by the Cerebral Palsy International Research Foundation and by National Institutes of Health Grant R03-HD050575.

We thank Tom Newman for his invaluable insights regarding data analysis.

- trends in neonatal death in very low birth weight non-Hispanic black and white infants, 1989–2004. *J Pediatr*. Oct 2009; 155(4):482–487
14. North AF Jr, MacDonald HM. Why are neonatal mortality rates lower in small black infants than in white infants in similar birth weight? *J Pediatr*. 1977;90(5):809–810
15. Martin JA. United States vital statistics and the measurement of gestational age. *Paediatr Perinat Epidemiol*. 2007;21(Suppl 2):13–21
16. Martin JA, Hamilton BE, Sutton PD, Ventura SJ, Menacker F, Kirmeyer S. Births: final data for 2004. *Natl Vital Stat Rep*. 29 2006; 55(1):1–101
17. Iyasu S, Becerra JE, Rowley DL, Hougue CJ. Impact of very low birthweight on the black-white infant mortality gap. *Am J Prev Med*. 1992;8(5):271–277
18. Frisbie WP, Echevarria S, Hummer RA. Prenatal care utilization among non-Hispanic Whites, blacks, and Mexican Americans. *Matern Child Health J*. 2001;5(1):21–33
19. Healy AJ, Malone FD, Sullivan LM, et al. Early access to prenatal care: implications for ra-

- cial disparity in perinatal mortality. *Obstet Gynecol.* 2006;107(3):625–631
20. Nanyonjo RD, Montgomery SB, Modeste N, Fujimoto E. A secondary analysis of race/ethnicity and other maternal factors affecting adverse birth outcomes in San Bernardino County. *Matern Child Health J.* Jul 2008;12(4):435–441
 21. Collins JW Jr, David RJ, Rankin KM, Desireddi JR. Transgenerational effect of neighborhood poverty on low birth weight among blacks in Cook County, Illinois. *Am J Epidemiol.* 2009;169(6):712–717
 22. Messer LC, Kaufman JS, Mendola P, Laraia BA. Black-white preterm birth disparity: a marker of inequality. *Ann Epidemiol.* 2008;18(11):851–858
 23. Dailey DE. Social stressors and strengths as predictors of infant birth weight in low-income black women. *Nurs Res.* 2009;58(5):340–347
 24. Menon R, Velez DR, Thorsen P, et al. Ethnic differences in key candidate genes for spontaneous preterm birth: TNF-alpha and its receptors. *Hum Hered.* 2006;62(2):107–118
 25. 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.* Oct 23 1997;337(17):1209–1214
 26. Wu YW, Croen LA, Torres AR, Van de Water J, Grether JK, Hsu NN. Interleukin-6 genotype and risk of cerebral palsy in term and near-term infants. *Ann Neurol.* 2009;66(5):663–670
 27. Badawi N, Kurinczuk JJ, Keogh JM, et al. Antepartum risk factors for newborn encephalopathy: the Western Australian case-control study. *BMJ.* 1998;317(7172):1549–1553
 28. Ancel PY, Livinec F, Larroque B, et al. Cerebral palsy among very preterm children in relation to gestational age and neonatal ultrasound abnormalities: the EPIPAGE cohort study. *Pediatrics.* 2006;117(3):828–835
 29. Platt MJ, Cans C, Johnson A, et al. Trends in cerebral palsy among infants of very low birthweight (<1500 g) or born prematurely (<32 weeks) in 16 European centres: a database study. *Lancet.* 2007;369(9555):43–50
 30. Johnston MV, Hagberg H. Sex and the pathogenesis of cerebral palsy. *Dev Med Child Neurol.* 2007;49(1):74–78
 31. Fiscella K. Does prenatal care improve birth outcomes? A critical review. *Obstet Gynecol.* 1995;85(3):468–479
 32. Baker LC, Afendulis CC, Chandra A, McConville S, Phibbs CS, Fuentes-Afflick E. Differences in neonatal mortality among whites and Asian American subgroups: evidence from California. *Arch Pediatr Adolesc Med.* 2007;161(1):69–76
 33. Le LT, Kiely JL, Schoendorf KC. Birthweight outcomes among Asian American and Pacific Islander subgroups in the United States. *Int J Epidemiol.* 1996;25(5):973–979
 34. Fuentes-Afflick E, Hessol NA. Impact of Asian ethnicity and national origin on infant birth weight. *Am J Epidemiol.* 1997;145(2):148–155
 35. Wong LF, Caughey AB, Nakagawa S, Kaimal AJ, Tran SH, Cheng YW. Perinatal outcomes among different Asian-American subgroups. *Am J Obstet Gynecol.* 2008;199(4):381–e386
 36. ACOG Task Force on Neonatal Encephalopathy and Cerebral Palsy. *Neonatal Encephalopathy and Cerebral Palsy.* Washington, DC: American College of Obstetricians and Gynecologists; 2003
 37. Nelson KB. The epidemiology of cerebral palsy in term infants. *Ment Retard Dev Disabil Res Rev.* 2002;8(3):146–150
 38. Wu YW, Swaiman KF. Cerebral palsy. In: Swaiman A, Ferriero, DM, Eds. *Pediatric Neurology: Principles and Practice.* Vol 1. Philadelphia, PA: Mosby Elsevier; 2006, p 491
 39. ACPR G. Report of the Australian Cerebral Palsy Register, birth years 1993–2003. *Australian Cerebral Palsy Register Report* [brochure]. Sydney, Australia; 2009.