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The role of socio-economic status and perinatal factors in racial disparities in the risk of cerebral palsy

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Abstract

AIM—To determine whether racial disparities in cerebral palsy (CP) risk among US children persist after controlling for socio-economic status (SES) (here indicated by maternal education) and perinatal risk factors.

METHOD—A population-based birth cohort study was conducted using the Autism and Developmental Disabilities Monitoring Network surveillance and birth data for 8-year-old children residing in multi-county areas in Alabama, Georgia, Missouri, and Wisconsin between 2002 and 2008. The birth cohort comparison group included 458 027 children and the case group

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The authors have stated that they had no interests which might be perceived as posing a conflict or bias.

SUPPORTING INFORMATION

The following additional material may be found online:

Appendix S1: Supplementary information on construction of the birth cohort.

Table SI: Analysis restricted to cerebral palsy (CP) cases not attributed to postneonatal causes: unadjusted relative risks, indicating associations between sociodemographic characteristics and CP risk, overall and stratified by spastic versus non-spastic or unspecified CP, with analysis restricted to CP cases with available birth data (i.e. those born in the same state of residence at age 8 years.

Table SII: Analysis restricted to cerebral palsy (CP) cases not attributed to postneonatal causes: results of multivariable logistic regression analysis, restricted to CP cases with available birth records.

included 1570 children with CP, 1202 with available birth records. χ^2 tests were performed to evaluate associations and logistic regression was used to calculate relative risks (RR) and adjusted odds ratios (OR) with 95% confidence intervals (CI).

RESULTS—The risk of spastic CP was more than 50% higher for black versus white children (RR 1.52, 95% CI 1.33–1.73), and this excess risk persisted after adjustment for SES (OR 1.35, 95% CI 1.18–1.55), but not after further adjustment for preterm birth and size for gestational age. The protective effect of maternal education remained after adjustment for race/ethnicity and perinatal factors.

INTERPRETATION—Maternal education appears to independently affect CP risk but does not fully explain existing racial disparities in CP prevalence in the US.

Recent studies in the US and Europe have found an increased risk of cerebral palsy (CP) associated with socio-economic disadvantage.[1–6] Some have found this association to persist, though somewhat attenuated, after controlling for perinatal risk factors such as low birthweight.[1,3–5] Previous studies in the US have also reported higher prevalence of CP among black children relative to white children. [5,7–10] One study found no excess prevalence of CP in black children after adjusting for birthweight. [5] What is unclear from previous studies is the extent to which the excess prevalence of CP in black children in the US is explained by socio-economic disparities.

In the present study, we evaluated available indicators of socio-economic status (SES), including maternal educational attainment when a child is born, and their association with the risk of CP in a large, diverse cohort of US children. We also sought to determine whether racial and ethnic disparities in CP risk persist after controlling for SES. More specifically, we designed the study to test the following three hypotheses: (1) consistent with recent studies, we will find the risk of CP to decline with increasing SES as indicated by maternal education or a census-based indicator of SES; (2) the observed racial and ethnic disparity in CP risk is caused by confounding or is mediated by racial disparities in SES and, therefore, will no longer be present after controlling for SES; and (3) perinatal factors such as preterm birth and small for gestational age mediate the association between race as well as maternal education and CP risk, so that after controlling for these perinatal risk factors, CP risk will not differ by race or maternal education.

METHOD

We implemented a population-based birth cohort study using CP prevalence data for 8-year-old children from the Centers for Disease Control and Prevention's Autism and Developmental Disabilities Monitoring (ADDM) Network[7–11] for the years 2002, 2004, 2006, and 2008, and population information based on birth certificate data for the birth years 1994, 1996, 1998, and 2000. ADDM Network sites conducting CP surveillance included multi-county areas in Alabama, Georgia, and Wisconsin for all four surveillance years and in Missouri for two years (2006 and 2008).

Using de-identified birth records from the National Center for Health Statistics' public use natality and infant death data files, we constructed a birth cohort representing all births

surviving to 1 year who were born in one of the surveillance sites and birth years. Further information on the construction of the birth cohort is provided in Appendix S1 (online supporting information). The resulting birth cohort included 458 027 births from the four sites and four birth years. The characteristics of these births overall, by race and ethnicity, and by maternal education are shown in Table I.

The ADDM Network surveillance system identified 1570 CP cases residing in the surveillance area at age 8 years, including 1202 cases from the birth cohort (i.e. born in one of the four states). An additional 368 children with CP living in the surveillance area at age 8 years were born out of state. Birth certificate information was available to the surveillance system only for in state births. An unknown number of children with CP were born into the cohort and moved out of the surveillance area before the age of 8 years and thus are included in the cohort (denominator) but could not be identified as CP cases by the surveillance system.[12]

Case definition

The ADDM Network implemented surveillance based on methods developed by the Centers for Disease Control and Prevention's Metropolitan Atlanta Developmental Disabilities Surveillance Program, an ongoing, population-based, multisource surveillance program that monitors the occurrence of developmental disabilities among 8-year-old children in metropolitan Atlanta.[7–13] These methods have been described in detail previously.[7] For surveillance purposes, we defined CP as a group of permanent disorders of movement and posture that are attributed to non-progressive disturbances in the developing brain.[14] All cases of CP were included, including those with a documented postneonatal (>28d after birth to 8y) cause.

Case inclusion criteria

The case inclusion criteria included birth in 1994, 1996, 1998, or 2000; residence in the surveillance area at any time while the child was aged 8 years; and documentation in an evaluation, conducted by a qualified professional when the child was aged 2 years or older, describing a CP diagnosis or physical findings consistent with CP. Children suspected of having CP were identified by screening comprehensive evaluations at multiple sources, including hospitals, clinics, diagnostic centers, health care providers, and state public health and rehabilitation agencies. Potential CP cases in Georgia also were identified through public school special education programs. Indicators for abstraction included confirmed or suspected CP diagnosis or descriptions of physical findings associated with CP.

Data collection

Diagnostic summaries and descriptions of physical findings from evaluations and demographics were collected as part of the surveillance program. Data were abstracted into one composite record per child, de-identified, and subsequently reviewed by trained clinicians using a specified protocol to determine case status. CP clinician reviewers from the four sites included developmental pediatricians, a pediatric neurologist, physical therapists, and occupational therapists. In the absence of excludable conditions, such as progressive disorders and neuromuscular diseases, children were classified as confirmed CP

cases based on diagnostic information and physical finding descriptions consistent with CP in the abstracted records. No direct clinical examinations of children were performed by project staff.

Socio-economic status classification and birth information

For children with CP whose place of birth was in the same state as their residence at age 8 years (*n*=1202; 76.5% of cases) and for the birth cohort overall, information on maternal education, birthweight, gestational age, prenatal care, and other birth characteristics was obtained from their birth certificates. The level of maternal education attained was classified into three SES categories: low (<high school graduate); middle (high school graduate with or without some college); and high (≥bachelor's degree). Size for gestational age was calculated using sex-specific intrauterine growth curves,[15] and birthweights below the tenth centile for gestational age were classified as small for gestational age. Race and ethnicity of each child was determined from information in clinical or education records or from birth certificate information, and classified as white non-Hispanic, black non-Hispanic, Hispanic, and other or undetermined.

Separate analysis based on census data

Because birth certificate information on maternal education was unavailable for children with CP born outside of their state of residence at age 8 years, we performed a separate analysis using census data to evaluate associations between CP and race and SES in the full cohort of CP cases. For this analysis we were unable to control for perinatal factors because data on these were unavailable for the census-based denominator or the CP cases born out of state. For this analysis, we used an area-based indicator of SES based on census block group of residence using an approach similar to that described by Krieger and colleagues.[16] In short, residents of census block groups where 10% or fewer adults had a bachelor's degree were classified as low SES, those in block groups where 11% to 35% of adults had a bachelor's were classified as middle SES, and those in block groups where more than 35% of adults had a bachelor's degree were classified as high SES.

Children with CP who migrated into the surveillance area after birth appeared to be of higher SES than those born in state; based on the census SES indicator, 14.7% of CP cases born out of state were classified as low SES, compared to 29.6% of those born in state ($p<0.001$). Thus, restricting the analysis to CP cases born in state could lead to selection bias by differentially excluding cases born to higher SES families, and create the appearance of an SES gradient even if one did not exist. The separate analysis using census data allowed us to evaluate whether findings on the effects of race and SES on CP risk that were inferred from the birth cohort analysis, which was necessarily restricted to CP cases born in state, could be generalized to the full cohort.

Data analysis

We calculated relative risks (RR) with 95% confidence intervals (CI) to evaluate associations between CP and maternal education, and other risk factors. We performed stratified analyses to evaluate whether these associations differed by CP type (spastic vs other and undetermined). We used χ^2 analysis to evaluate differences in proportions, and

multivariable logistic regression to compute adjusted odds ratios (OR), CIs, and *p* values for associations between CP risk and multiple risk factors. Variables included in the multivariable models were those shown to be associated with both race/ethnicity and CP risk. The adjusted ORs allowed evaluation of whether the racial disparity in CP risk persisted after adjustment for SES and for perinatal factors. The statistical analyses were performed using IBM-SPSS Version 22 (SPSS Inc. Chicago, IL, USA).

Each of the participating sites met applicable local Institutional Review Board and privacy/confidentiality requirements under 45 CFR 46.

RESULTS

Among the 1570 CP cases included in the study, 79.8% were classified as spastic (excluding some with mixed spastic and other forms of CP), and the remaining as hypotonic, ataxic or dyskinetic, mixed spastic and other, or other or unspecified forms of CP (Table II). Clinical characteristics did not differ for cases born in state versus those who migrated into the surveillance area after birth (Table II), but there were important differences between cases born in versus out of state in terms of racial and ethnic composition and SES (Table II).

In examining the associations between CP risk and sociodemographic factors (race, ethnicity, SES, sex of the child), we found some associations differed between CP classified as spastic versus non-spastic or unspecified. Therefore, we present the associations between CP risk and sociodemographic factors overall and stratified by spastic versus other. In the analysis restricted to those born in state, compared to white non-Hispanic children, the risk of spastic CP was 52% higher in black non-Hispanic children (RR 1.52, 95% CI 1.33–1.73), while there was no significant difference between black and white children in the risk of non-spastic/unspecified CP (RR 0.89, 95% CI 0.67–1.20). The risk of CP overall and of spastic and non-spastic CP did not differ between Hispanic and white non-Hispanic children (Table IIIa).

Low versus high SES was associated with a 67% increased risk of CP overall (relative risk 1.67, 95% CI 1.41–1.98) and 93% increased risk of spastic CP (RR 1.93, 95% CI 1.60–2.33). For non-spastic and unspecified CP, there was no evidence of increased risk associated with low SES (Table III). The risk of CP was 32% higher in males than females (RR 1.32, 95% CI 1.18–1.48), and this association was similar for spastic and non-spastic or unspecified CP (Table III).

In the separate analyses based on census data and inclusive of all cases of CP, the findings were similar in direction to those restricted to children born in state, though somewhat attenuated. For spastic but not for other and unclassified CP, the risk was higher for black versus white children (RR 1.44, 95% CI 1.28–1.62), and for children of low versus high SES (RR 1.40, 95% CI 1.20–1.63) (Table IV).

In the birth cohort analysis of those born in state, the increased risk of spastic CP in black versus white children persisted after adjustment for SES (OR 1.35, 95% CI 1.18–1.55). Similarly, after adjustment for race and ethnicity, the risk of spastic CP remained higher for children of low versus high SES (OR 1.85, 95% CI 1.52–2.26) (Table IIIb).

Table IIIa includes unadjusted RRs for several perinatal risk factors for CP, including maternal age, low birthweight, preterm birth, small for gestational age, lack of documented prenatal care, and multiple birth. In contrast to the findings for race and SES, the associations between these perinatal risk factors and CP were similar for spastic and non-spastic or unspecified CP.

After adjusting for perinatal risk factors, we found the risk of CP overall was significantly lower in black and Hispanic children relative to white children (OR 0.87, 95% CI 0.77–0.99 for black vs white children; OR 0.75, 95% CI 0.58–0.99 for Hispanic vs white children; Table V). Adjustment for perinatal risk factors had little effect on the association between maternal educational attainment and CP risk (Table V), suggesting that the protective effect of maternal education on the risk of spastic CP appeared to be independent of measured perinatal risk factors. We repeated these analyses after excluding CP cases attributed to postneonatal causes and found similar results (Tables SI and SII, online supporting information).

DISCUSSION

In support of our first hypothesis and consistent with several recent epidemiologic studies from the US and Europe[1–6] and some older studies,[17] we found CP risk to decline with increasing socio-economic advantage, here indicated primarily by maternal education. However, contrary to our second hypothesis, we did not find the effects of SES on CP risk to fully explain the excess prevalence of CP in black versus white children that is documented in this paper and in previously published findings from the ADDM Network.[7–10] Thus, even after controlling for racial differences in SES, the risk of spastic CP was significantly higher in black relative to white children. This finding is consistent with the study from California by Wu et al. showing that adjustment for maternal education did not eliminate the excess prevalence of CP among black children.[5]

Our third hypothesis was only partially supported by the findings. We did find, as hypothesized and demonstrated previously by Wu, et al,[5] that after controlling for perinatal risk factors, the excess risk of CP in black children was no longer present, and the risk of CP was paradoxically lower for black and Hispanic children than white children after adjusting for perinatal risk factors. This suggests that the excess risk of CP in black children is mediated by preterm birth and associated perinatal factors, and that elimination of the excess risk of CP in black children in the US will require elimination of the excess risk of preterm birth and associated perinatal risk factors experienced by black infants.[5] The ‘low birth weight paradox’ with respect to neonatal mortality refers to the finding that US black infants who are low birthweight experience lower neonatal mortality than white infants of comparable weight, even though neonatal mortality overall, when birthweight is not controlled, is higher among black infants than white infants.[18] This paradox has been attributed to an artifact from the use of fixed cut-offs for low birthweight categories despite differences in the Gaussian distributions of birthweight between black and white infants in the US.[19] Our data suggest that the paradox may apply to both spastic and non-spastic CP, though the adjusted OR of 0.92 for spastic CP among black versus white children was not significantly less than 1.0 (Table V). Notwithstanding the low birthweight paradox, our

overall findings support the conclusion that the racial disparity in CP risk is mediated by perinatal risk factors.

With respect to maternal education, our findings did not support the hypothesis that its protective effect on CP risk operates entirely through preterm birth or other perinatal risk factors for which we had measures, since adjustment for perinatal risk factors attenuated but did not eliminate the association between maternal education and CP risk.

Our finding that the increased risk of CP for black children and those born to mothers with lower levels of education or of low SES was limited to spastic CP is somewhat consistent with the findings of Dolk, et al.[2] They found that cases of CP classified as spastic bilateral had a stronger association with socio-economic disadvantage than did other CP types. Within the broad category of 'spastic CP', different subtypes and causes could be associated with SES differently. Further research is needed into the causal mechanisms underlying the associations between low SES and spastic CP.

Previous publications have noted the relatively wide variation in CP prevalence across populations.[10,12,20,21] Some of this variation could be caused by the effects of sociodemographic factors on CP risk that have been demonstrated in this study.

A strength of this study is the large, diverse population from which the estimates are based, making it possible to investigate separate and interrelated effects of sociodemographic factors on CP prevalence and to show that these associations differ for CP cases classified as spastic versus other. Another strength is the use of a birth cohort design and incorporation of information on perinatal risk factors for CP for the population-based cohort as a whole and for most of the CP cases. Our study's definition of postneonatal causes as having an onset up to age 8 years and inclusion of CP cases attributed to postneonatal causes could limit comparability of the findings to other studies, though in a separate analysis we found that excluding the 4% of cases attributed to postneonatal causes had little effect on our results. A limitation of our birth cohort approach is that we were unable to determine CP status of an unknown number of children with CP born into the cohort who were not residing in the surveillance area at the age of 8 years, though all children born in the surveillance area and surviving to age 1 year were included in the birth cohort serving as the comparison group. To help compensate for this limitation, we included in a separate analysis all cases, including those residing in the surveillance area at age 8 who were born out of state, and found the effects of race and SES on spastic CP risk were similar to analyses restricted to cases born in state. This provided some assurance that the SES gradient seen in the subsample born in state was not entirely caused by selection bias. It is possible that the SES gradient found in this study is underestimated if the surveillance system was unable to identify some children because of lack of health insurance or access to healthcare.

Another limitation of the study is the small number of CP cases and controls of other specified racial or ethnic groups, which limited our ability to evaluate disparities in CP risk among children not classified as non-Hispanic white or black or Hispanic. In addition, Hispanic children in these four states are not necessarily representative of all Hispanic children in the US. An additional limitation is our reliance on maternal education as the

primary indicator of SES. Further research is warranted on the effects of other components of SES, such as income, occupation, and insurance coverage, and on longitudinal trajectories allowing evaluation of the direction of the association between CP and SES.

Taken together, our findings suggest that maternal education may influence the risk of spastic CP independently of its association with race and with established perinatal risk factors for CP. A reasonable public health goal would be to reduce the risk of CP in the population overall to the level of risk experienced by offspring of college-educated mothers. Further research should be directed at finding ways to achieve this.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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ABBREVIATIONS

ADDM	Autism and Developmental Disabilities Monitoring
SES	Socio-economic status

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What this paper adds

- The risk of spastic CP was higher in black relative to white children.
- Disparities in maternal education, an indicator of SES, did not fully explain the excess CP risk among black children.
- Factors associated with higher maternal education may help protect against the risk of spastic CP.

Table I

a: Characteristics of the study birth cohort, by categories of race and ethnicity, and maternal education - by race and ethnicity

	White non-Hispanic (*n*=276 324)	Black non-Hispanic (*n*=135 016)	Hispanic (*n*=30 556)	Other or undetermined (*n*=16 131)	Total (*n*=458 027)
Maternal education					
% < High school	11.9	25.2	50.5	15.2	18.5
% High school grad	49.8	59.8	34.6	40.2	51.4
% ≥Bachelor's	37.8	14.1	9.2	40.8	29.0
% Missing	0.5	0.9	5.7	3.8	1.1
p<0.001					
% Male sex	51.2	50.6	50.4	51.2	51.2
p<0.001					
Maternal age, y					
% <20	8.2	20.7	16.0	6.1	12.3
% 20–34	76.6	70.5	76.6	78.0	74.9
% ≥35	15.2	8.8	7.4	15.9	12.8
p<0.001					
Birthweight, g					
% <1500	1.0	2.7	1.0	0.9	1.5
% 1500–2499	5.3	9.8	4.9	5.9	6.6
% ≥2500	93.7	87.4	94.0	92.9	91.8
% missing	<0.1	0.1	0.1	0.3	0.1
p<0.001					
Gestational age at birth, wk					
% <28	0.4	1.4	0.5	0.4	0.7
% 28–36	9.6	15.2	9.2	9.6	11.3
% ≥37	89.9	83.0	90.0	89.5	87.8
% missing	0.1	0.3	0.3	0.5	0.2
p<0.001					

a: Characteristics of the study birth cohort, by categories of race and ethnicity, and maternal education - by race and ethnicity

	White non-Hispanic (*n*=276 324)	Black non-Hispanic (*n*=135 016)	Hispanic (*n*=30 556)	Other or undetermined (*n*=16 131)	Total (*n*=458 027)
% Small for gestational age	8.6	16.6	10.4	13.9	11.4
% missing	1.0	1.2	1.1	1.1	1.0
p<0.001					
% No documented prenatal care	1.2	4.0	6.3	3.9	2.5
p<0.001					
% Multiple birth	3.3	3.2	1.7	2.3	3.1
p<0.001					

b: Characteristics of the study birth cohort, by categories of race and ethnicity, and maternal education - by maternal educational attainment

	High school graduate (*n*=84 762)	High school graduate with or without some college (*n*=235 309)	Bachelor's degree or higher (*n*=133 018)	Unknown (*n*=4918)	Total (*n*=458 027)
% Male sex	50.6	51.0	51.3	51.2	51.0
p=0.016					
Maternal age, y					
<20	43.3	8.1	0	14.7	12.3
20–34	53.0	81.3	77.5	72.2	74.9
≥35	3.7	10.6	22.5	13.1	12.8
p<0.001[a]					
Birthweight, g					
<1500	1.8	1.6	1.0	2.0	1.5
1500–2499	8.9	6.7	5.0	6.7	6.6
≥2500	89.2	91.6	93.9	90.9	91.8
% missing	0.1	0.1	0.1	0.4	0.1
p<0.001					

b: Characteristics of the study birth cohort, by categories of race and ethnicity, and maternal education - by maternal educational attainment

	High school graduate (*n*=84 762)	High school graduate with or without some college (*n*=235 309)	Bachelor's degree or higher (*n*=133 018)	Unknown (*n*=4918)	Total (*n*=458 027)
Gestational age at birth, wk					
% <28	1.0	0.8	0.4	1.3	0.7
% 28–36	13.9	11.6	9.1	10.3	11.3
% ≥37	84.9	87.4	90.4	87.0	87.8
% missing	0.3	0.2	0.1	1.4	0.2
p<0.001					
% Small for gestational age	15.7	11.6	7.9	12.7	11.4
% missing	1.6	1.1	0.6	2.1	1.0
p<0.001					
% No documented prenatal care	4.9	2.2	0.7	20.0	2.5
p<0.001					
% Multiple birth					
p<0.001	2.2	3.0	3.8	2.4	3.1

The *p* values are based on chi-squared analyses with missing values included as a category when relevant, and indicate the significance of differences in characteristics by categories of race and ethnicity in A, and categories of maternal educational attainment in B.

[a]We note that one of the expected cell sizes for evaluating the association between maternal age and maternal education was <5, making the χ^2 test for this comparison uninterpretable. To overcome this limitation, we performed a separate analysis comparing the percent of births to mothers aged ≥35 years by maternal education. For this test, the minimum expected cell count was 629.49 and the χ^2 p-value was <0.001.

Table II

Frequency and characteristics of 8-year-old children with cerebral palsy (CP) in the surveillance area, including multi-county areas in Alabama, Georgia, Missouri and Wisconsin, birth years 1994, 1996, 1998, and 2000

	% of those born in state (*n*=1202)	% of those born out of state (*n*=368)	% of total (*n*=1570)	*p*[a]
Type of CP				0.243
Spastic	80.5	77.4	79.8	
Hypotonic	3.9	4.3	4.0	
Ataxic/dyskinetic	3.1	2.7	3.0	
Mixed spastic and other	5.5	4.5	5.4	
Other or unspecified	7.0	10.6	7.8	
Extent of limb involvement				0.635
Quadriplegia/triplegia	22.2	21.5	22.0	
Diplegia	30.0	29.6	29.9	
Hemiplegia/monoplegia	26.1	24.2	25.7	
Other/unspecified	21.6	24.7	22.4	
Documented postneonatal CP Etiology	4.2	3.3	4.0	0.401
Male sex	57.9	60.9	58.6	0.342
Race/ethnicity				<0.001
White non-Hispanic	54.6	41.3	51.5	
Black non-Hispanic	36.7	32.1	35.6	
Hispanic	5.2	12.0	6.8	
Other	3.6	5.2	3.9	
Undetermined	0	9.5	2.2	
SES Based on maternal education[b]				-
Low (< high school graduate)	23.1	N/A	N/A	
Middle (high school graduate)	53.7	N/A	N/A	
High (≥bachelor's degree)	21.7	N/A	N/A	
Missing	1.5	N/A	N/A	
SES based on census indicator[c]				<0.001
Low	29.6	14.7	26.1	
Medium	46.8	47.0	46.9	
High	23.5	38.3	27.0	

[a] Comparing respective percentages for those born in and out of state, based on χ^2 analysis.

[b] Maternal educational attainment at birth of index child, available only for those with available birth certificate information.

[c] Based on aggregate educational attainment of adults in census block group of child's residence at age 8.

Table III

a: Unadjusted relative risks (RR), indicating associations between sociodemographic and birth characteristics and cerebral palsy (CP) risk, overall and stratified by spastic versus non-spastic or unspecified CP, with analyses restricted to CP cases with available birth data (i.e. those born in the same state of residence at age 8y)

	Non-cases (*n*=456 457), *n* (%)	All CP (*n*=1202)		Spastic CP (*n*=968)	Non-spastic and unspecified CP (*n*=234)
		CP cases, *n* (%)	RR (95% CI)	RR (95% CI)	RR (95% CI)
Race/ethnic category					
White non-Hispanic	275 516 (60.4)	656 (54.6)	Reference	Reference	Reference
Black non-Hispanic	134 457 (29.5)	441 (36.7)	1.38 (1.22–1.55)[a]	1.52 (1.33–1.73)[a]	0.89 (0.67–1.20)
Hispanic	30 450 (6.7)	62 (5.2)	0.86 (.66–1.11)	0.89 (.67–1.19)	0.74 (0.41–1.33)
Other/undetermined	16 034 (3.5)	43 (3.6)	1.13 (0.83–1.53)	1.08 (0.76–1.54)	1.29 (0.70–2.37)
SES[b]					
Low	84 442 (18.5)	278 (23.1)	1.67 (1.41–1.98)[a]	1.93 (1.60–2.33)[a]	0.90 (0.61–1.35)
Middle	234 478 (51.3)	645 (53.7)	1.40 (1.21–1.61)[a]	1.51 (1.28–1.78)[a]	1.06 (0.79–1.43)
High	132 637 (29.1)	261 (21.7)	Reference	Reference	Reference
Missing	4900 (1.1)	18 (1.5)			
Sex					
Male	232 565 (51.0)	696 (57.9)	1.32 (1.18–1.48)[a]	1.31 (1.15–1.49)[a]	1.38 (1.07–1.80)[c]
Female	223 892 (49.0)	506 (42.1)	Reference	Reference	Reference
Maternal Age, y					
<20	56 206 (12.3)	188 (15.6)	1.38 (1.18–1.62)[a]	1.53 (1.29–1.82)[a]	0.81 (0.53–1.26)
20–34	341 858 (74.9)	827 (68.8)	Reference	Reference	Reference
≥35	58 393 (12.8)	186 (15.5	1.32 (1.13–1.54)[a]	1.31 (1.10–1.57)[d]	1.33 (0.94–1.88)
Birthweight, g					
<1500	6435 (1.4)	370 (30.8)	37.4 (32.9–42.4)[a]	42.9 (37.3–49.3)[a]	21.5 (15.5–29.7)[a]
1500–2499	29 939 (6.6)	219 (18.2)	5.00 (4.28–5.86)[a]	5.32 (4.48–6.32)[a]	3.96 (2.80–5.60)[a]
≥2500	419 940 (92.0)	612 (50.9)	Reference	Reference	Reference

a: Unadjusted relative risks (RR), indicating associations between sociodemographic and birth characteristics and cerebral palsy (CP) risk, overall and stratified by spastic versus non-spastic or unspecified CP, with analyses restricted to CP cases with available birth data (i.e. those born in the same state of residence at age 8y)

	Non-cases (*n*=456 457), *n* (%)	All CP (*n*=1202)		Spastic CP (*n*=968)	Non-spastic and unspecified CP (*n*=234)
		CP cases, *n* (%)	RR (95% CI)	RR (95% CI)	RR (95% CI)
Missing	143 (<0.1)	1 (0.1)			
Gestational age at birth, wk					
<28	3086 (0.7)	242 (20.1)	47.6 (41.2–55.0)[a]	55.6 (47.4–65.2)[a]	25.8 (17.5–38.1)[a]
28–36	51 223 (11.2)	343 (28.5)	4.35 (3.81–4.96)[a]	4.85 (4.19–5.62)[a]	2.82 (2.07–3.85)[a]
≥37	401 671 (88.0)	615 (51.2)	Reference	Reference	Reference
Missing	477 (0.1)	2 (0.2)			
Small for gestational age					
No	403 886 (88.5)	974 (81.0)	Reference	Reference	Reference
Yes	52 094 (11.4)	226 (18.8)	1.80 (1.55–2.07)[a]	1.86 (1.58–2.18)[a]	1.55 (1.10–2.19)[c]
Missing	477 (0.1)	2 (0.2)			
Documented receipt of prenatal care					
No	11 154 (2.4)	70 (5.8)	2.46 (1.93–3.13)[a]	2.54 (1.95–3.31)[a]	2.16 (1.21–3.85)[d]
Yes	445 303 (97.6)	1132 (94.2)	Reference	Reference	Reference
Multiple birth					
No	442 474 (96.9)	1080 (89.9)	Reference	Reference	Reference
Yes	13 983 (3.1)	122 (10.1)	3.55 (2.95–4.28)[a]	3.67 (2.99–4.50)[a]	3.12 (1.99–4.85)[a]

b: Adjusted odds ratios (OR), indicating associations between sociodemographic and birth characteristics and cerebral palsy (CP) risk, overall and stratified by spastic versus non-spastic or unspecified CP, with analyses restricted to CP cases with available birth data (i.e. those born in the same state of residence at age 8y).

	All CP (*n*=1202)	Spastic CP (*n*=968)	Non-spastic and Unspecified CP (*n*=234)
	OR (95% CI)	OR (95% CI)	OR (95% CI)
Race/ethnic category			
White non-Hispanic	Reference	Reference	Reference

b: Adjusted odds ratios (OR), indicating associations between sociodemographic and birth characteristics and cerebral palsy (CP) risk, overall and stratified by spastic versus non-spastic or unspecified CP, with analyses restricted to CP cases with available birth data (i.e. those born in the same state of residence at age 8y).

	All CP (*n*=1202)	**Spastic CP (*n*=968)**	**Non-spastic and Unspecified CP (*n*=234)**
	OR (95% CI)	**OR (95% CI)**	**OR (95% CI)**
Black non-Hispanic	1.25 (1.11–1.42)[a]	1.35 (1.18–1.55)[a]	0.88 (0.65–1.19)
Hispanic	0.71 (0.55–0.93)[c]	0.72 (0.54–0.98)[c]	0.69 (0.38–1.28)
Other/undetermined	1.11 (0.81–1.51)	1.07 (0.75, 1.53)	1.23 (0.66–2.28)
SES[b]			
Low	1.65 (1.38–1.97)[a]	1.85 (1.52–2.26)[a]	0.99 (0.65–1.50)
Middle	1.34 (1.16–1.56)[a]	1.43 (1.21–1.69)[a]	1.10 (0.81–1.50)
High	Reference	Reference	Reference

[a] $p<0.001$.

[b] SES in this analysis is based on maternal educational attainment. Maternal education less than high school graduate was classified as low SES; maternal education of high school graduate with or without some college was classified as middle SES, and maternal education of bachelor's degree or higher was classified as high SES.

[c] $p<0.05$.

[d] $p<0.01$.

Table IV

Unadjusted relative risks (RR), indicating associations between race/ethnicity, SES, sex and cerebral palsy (CP) risk, overall and stratified by spastic versus non-spastic or unspecified CP, based on census data for SES and denominator information (using CP prevalence reports for denominator totals, and percent distributions from weighted census file for number in each SES categories and numbers of males and females)

	Total population of 8-year-old children, based on census data (*n*=472 619), *n* (%)	**All CP* (*n*=1570)**		**Spastic CP (*n*=1253)**	**Non-spastic and unspecified CP (*n*=317)**
		CP cases, *n* (%)	**RR (95% CI)**	**RR (95% CI)**	**RR (95% CI)**
Race/ethnic category					
White non-Hispanic	267 258 (56.6)	808 (51.5)	Reference	Reference	Reference
Black non-Hispanic	144 026 (30.5)	559 (35.6)	1.28 (1.15–1.43)[a]	1.44 (1.28–1.62)[a]	0.78 (0.60–1.01)
Hispanic	42 723 (9.0)	106 (6.8)	0.82 (0.67–1.01)	0.88 (0.71–1.10)	0.62 (0.39–1.00)[b]
Other	18 435 (3.9)	62 (3.9)	1.11 (0.86–1.44)	1.01 (0.74–1.38)	1.42 (0.90–2.31)
Undetermined	0	35 (2.2)	–	–	–
SES[c]					
Low	106 812 (22.6)	410 (26.1)	1.23 (1.07–1.41)[d]	1.40 (1.20–1.63)[a]	0.73 (0.53–0.99)[b]
Middle	230 165 (48.7)	736 (46.9)	1.02 (0.91–1.15)	1.11 (0.97–1.27)	0.77 (0.60–0.99)[b]
High	135 642 (28.7)	424 (27.0)	Reference	Reference	Reference
Sex					
Male	240 563 (50.9)	920 (58.6)	1.36 (1.23–1.51)[a]	1.38 (1.23–1.54)[a]	1.32 (1.05–1.65)[b]
Female	232 056 (49.1)	650 (41.4)	Reference	Reference	Reference

[a] $p<0.001$.

[b] $p<0.05$.

[c] SES categories based on aggregate educational attainment of adults in census block group of child's residence at age 8.

[d] $p<0.01$.

* The overall prevalence of CP based on these data is 3.3 per 1000 8-year-old children.

Table V

Results of multivariable logistic regression analysis, restricted to cerebral palsy (CP) cases with available birth records

	All CP	Spastic CP	Non-spastic or unspecified CP
	Adjusted OR (95% CI)	Adjusted OR (95% CI)	Adjusted OR (95% CI)
Race/ethnic category			
White non-Hispanic	Reference	Reference	Reference
Black non-Hispanic	0.87 (0.77–0.99)[a]	0.92 (0.80–1.06)	0.68 (0.50–0.93)[a]
Hispanic	0.75 (0.58–0.99)[a]	0.77 (0.57–1.04)	0.71 (0.38–1.31)
Other/undetermined	1.05 (0.76–1.44)	1.00 (0.70–1.46)	1.20 (0.64–2.22)
SES[b]			
Low	1.42 (1.16–1.73)[c]	1.55 (1.24–1.94)[c]	0.99 (0.63–1.58)
Middle	1.26 (1.08–1.46)[d]	1.32 (1.11–1.57)[d]	1.08 (0.79–1.47)
High	Reference	Reference	Reference
Sex			
Male	1.31 (1.17–1.47)[c]	1.29 (1.14–1.47)[c]	1.37 (1.05–1.77)[a]
Female	Reference	Reference	Reference
Maternal age, y			
<20	1.05 (0.87–1.24)	1.10 (0.91–1.34)	0.78 (0.48–1.27)
20–34	Reference	Reference	Reference
≥35	1.34 (1.14–1.58)[c]	1.36 (1.13–1.63)[d]	1.28 (0.90–1.82)
Gestational age at birth, wk			
<28	50.06 (42.43–59.06)[c]	57.05 (47.66–68.28)[c]	27.21 (17.82–41.53)[c]
28–36	4.17 (3.63–4.78)[c]	4.61 (3.95–5.36)[c]	2.77 (2.00–3.84)[c]
≥37	Reference	Reference	Reference
Small for gestational age			
No	Reference	Reference	Reference
Yes	1.87 (1.61–2.17)[c]	1.93 (1.64–2.28)[c]	1.63 (1.15–2.31)[d]
Documented receipt of prenatal care			
No	1.25 (0.97–1.62)	1.21 (0.91–1.60)	1.44 (0.78–2.64)
Yes	Reference	Reference	Reference
Multiple birth			
No	Reference	Reference	Reference
Yes	1.15 (0.94–1.41)	1.13 (0.91–1.41)	1.26 (0.78–2.04)

ORs for each CP category are adjusted for all variables included in the table.

[a] $p<0.05$.

[b] SES in this analysis is based on maternal educational attainment. Maternal education less than high school graduate was classified as low SES; maternal education of high school graduate with or without some college was classified as middle SES, and maternal education of bachelor's degree or higher was classified as high SES.

[c] $p<0.001$.

[d] $p<0.01$.