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Prevalence of Cerebral Palsy among 8-Year-Old Children in 2010 and Preliminary Evidence of Trends in Its Relationship to Low Birthweight

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Abstract

Background—The public health objective for cerebral palsy (CP) in the United States is to reduce the percentage of children with CP who were born low birthweight (LBW, <2500 g) by 10% between 2006 and 2020. This study reports the prevalence of CP in a constant surveillance area for the years 2006, 2008, and 2010 and describes initial progress towards the CP public health objective.

Methods—Data on children with CP at age 8 years were ascertained by the Autism and Developmental Disabilities Monitoring (ADDM) Network, a population-based surveillance system that monitored CP in four areas of the United States.

Results—CP prevalence in 2010 was 2.9 per 1000 [95% confidence interval (CI) 2.6, 3.2], down from 3.5 (95% CI 3.2, 3.9) in the same surveillance area in 2006. Among CP cases with no documented postneonatal aetiology, 49.1% (95% CI 42.9, 55.2) were born LBW in 2010

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compared with 54.3% (95% CI 48.4, 60.1) in 2006. In 2010, 28.1% (95% CI 22.9, 30.4) were born very low birthweight (VLBW, <1500 g) compared with 35.4% (95% CI 30.0, 41.2) in 2006. The relative risks for associations between CP and both LBW and VLBW also declined, though not significantly, during the study period.

Conclusions—Declines in the associations between CP and LBW categories may have contributed to declines during the study period in both the prevalence of CP and the percentage of children with CP who were born LBW or VLBW. Ongoing monitoring of these trends is warranted.

Keywords

cerebral palsy; birth weight; epidemiology prevalence; public health surveillance

The *Healthy People 2020* (*HP2020*) public health objective for cerebral palsy (CP) in the United States is to reduce by 10%, between 2006 and 2020, the percentage of children with CP who were born low birthweight (LBW, <2500 g).[1] In the baseline year of 2006, this percentage was estimated, based on Autism and Developmental Disabilities Monitoring (ADDM) Network data, to be 50.0 for all 8-year-old children with CP. This percentage was 54.3 in 2006 among CP cases with no documented postneonatal cause of CP in four states that were monitored in 2006 and in two subsequent surveillance years, 2008 and 2010. Using 54.3% as the baseline for the percent born LBW among CP cases not attributed to a post-neonatal cause, and the objective of a 10% reduction in this percentage, we estimate that the target would be to reduce the percentage born LBW in this group to 48.9% by the year 2020.

The background for this public health objective is the experience of the last quarter of the 20th century and first decade of the 21st century, when there was a gradual rise in the frequency of preterm birth and LBW among all livebirths in the United States and among infants surviving to 1 year.[2,3] This trend, attributed to advances in perinatal medicine and neonatal intensive care,[4] led to concerns that improvements in survival of very preterm and LBW infants would be accompanied by increases in the prevalence of CP in the population overall if the risk of CP remained constant within birthweight categories.[5–7] Fortunately, beginning in the 1990s, following the introduction of antenatal corticosteroids and other interventions to improve outcomes of preterm birth, marked decreases in the risk of CP in cohorts born preterm or very low birthweight (<1500 g, VLBW) were observed.[8–11] Given these trends, a reasonable public health goal might be to achieve measurable decreases at the population level in the risk of CP among infants born preterm or LBW. However, our ability to directly measure progress towards such a goal would require a population-based, longitudinal surveillance system, measuring developmental outcomes through early childhood in successive birth cohorts over time. No such system exists in the United States.

An alternative approach, possible with surveillance data from the ADDM Network, is to retrospectively monitor trends in the percentage of children with CP in the population who were born LBW. If ongoing improvements in the survival of LBW infants are accompanied by large enough reductions in the risk of CP associated with LBW, to counter the rising number of infants born LBW, we would expect to see reductions over time in the percentage

of children with CP who were born LBW, making the *HP2020* objective for CP a measurable goal.

The purposes of this article are to provide updated information on the prevalence of CP among 8-year-old children for year 2010 in the four ADDM Network surveillance sites, where CP prevalence has been monitored since 2006, and to assess initial progress towards the *HP2020* public health objective for CP by describing the percentage of children with CP not attributable to postneonatal causes who were born LBW for each of three surveillance years, 2006, 2008, and 2010. In addition to LBW, results are presented for VLBW and stratified by sex, by race and ethnicity, and by two co-occurring conditions, epilepsy and autism spectrum disorder (ASD). Additional aims are to describe the associations between CP and both LBW and VLBW and to evaluate whether these associations declined during the 2006–10 surveillance years.

Methods

The ADDM Network monitored CP among 8-year-old children living in four areas in the United States, including central Alabama; metropolitan Atlanta, Georgia; metropolitan St. Louis, Missouri; and southeastern Wisconsin in 2006, 2008, and 2010. The prevalence of CP for the surveillance years 2006 and 2008 was published previously for a broader study area than the area under surveillance in 2010.[12,13] In one site, the size of the surveillance area changed over time. To allow comparisons over time, this report is restricted to counties in the four states that were under surveillance in all 3 years, 2006, 2008, and 2010 (Table 1).

The methodology for CP surveillance was based on that developed by the CDC's Metropolitan Atlanta Developmental Disabilities Surveillance Program, an ongoing, population-based, multisource surveillance programme that monitors the occurrence of developmental disabilities among 8-year-old children in metropolitan, Atlanta, and that has been described in detail previously.[12–16] For surveillance purposes, we defined CP as a group of permanent disorders of movement and posture that are attributed to nonprogressive disturbances in the developing brain. CP is more than a motor impairment: it is a disorder often accompanied by disturbances of sensation, perception, cognition, communication, and behaviour; by epilepsy; and by secondary musculo-skeletal problems.[17] This surveillance case definition includes cases with a documented postneonatal (>28 days after birth to age 8 years) cause. For analyses monitoring trends in the percent LBW, we restricted the CP cases to those with no documented postneonatal aetiology, which included approximately 93% of the CP cases each year.

Case inclusion criteria were as follows: residence in one of the respective surveillance areas during years 2006, 2008, or 2010 at any time during the age of 8 years; and documentation of a CP diagnosis or physical findings consistent with CP in an evaluation by a qualified professional at or after 2 years of age. Children suspected of having CP were identified by screening comprehensive evaluations at multiple sources, including hospitals, clinics, diagnostic centres, health care providers, and state public health and rehabilitation agencies. In Georgia, potential cases also were identified through public school special education

programmes. Records containing one or more evaluations with a confirmed or suspected CP diagnosis or descriptions of physical findings consistent with CP were abstracted.

Demographic data, diagnostic summaries, descriptions of physical findings and motor functioning, and information about co-occurring disabilities were collected. 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, other, or undetermined. Data were combined into one composite record per child and subsequently reviewed by clinicians using a specified protocol to determine case status. Clinician reviewers included developmental paediatricians, a paediatric 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.

Two methods for classifying gross motor function were incorporated into clinician review. When sufficient information on gross motor function at or after 4 years of age was abstracted, clinician reviewers used the Gross Motor Function Classification System (GMFCS)[18] to assign a functional level. GMFCS is based on the gross motor skills needed for self-initiated movement, including sitting, transferring, and mobility. Due to reliance on the documented findings in the source files, we were unable to assign a GMFCS level for all cases.[19]

Motor function was also classified based on walking ability as ‘unaided walking’, ‘walking with aids’, or ‘unable to walk’.[20] In determining walking ability, children classified as GMFCS Levels I or II were categorised as ‘walks independently’; Level III as ‘walks with handheld mobility device’; and Levels IV or V as ‘limited or no walking ability’. For children with insufficient information to GMFCS level, walking ability was independently assigned.[21]

Definitions of co-occurring developmental disabilities

Epilepsy—Children older than 1 month were identified as having epilepsy if evaluations by qualified professionals documented diagnoses of epilepsy or an epilepsy syndrome, or included descriptions of two or more unprovoked, nonfebrile seizures, >24 h apart.[22]

Autism spectrum disorder—Children were classified as having an autism spectrum disorder (ASD) if evaluation records documented behaviours consistent with the *Diagnostic and Statistical Manual, Fourth Edition, Text Revision* (DSM-IV-TR)[23] criteria for autistic disorder; pervasive developmental disorder, not otherwise specified; or Asperger disorder.

Data on birthweight

For children with CP whose place of birth was in the same state as their residence at age 8 years (N = 916 or 76.3% of the cases) and for all births in the respective surveillance sites and birth years (1998, 2000, and 2002), information on birthweight, gestational age, sex, and race and ethnicity was obtained from birth certificates. For CP cases born in state, individual-level birth certificate information was linked to case data and then de-identified

before inclusion in the ADDM Network analytic files. For the birth cohort overall, we used de-identified birth records from the National Center for Health Statistics' (NCHS) public use Natality and infant death data files.[24] From these files, we constructed a birth cohort representing all infants surviving to 1 year born in one of the surveillance sites and birth years. The resulting birth cohort included 354 054 infants from the four sites and three birth years. As mentioned, birthweight <2500 g was classified as LBW, and birthweight <1500 g was classified as VLBW. We classified infants as preterm if gestational age at birth was <37 weeks and as very preterm if gestational age was <32 weeks.

Data quality

Before independent review of abstracted records for case determination, initial inter-rater reliability was established among the reviewers to minimum standards of 90% agreement on case status. Ongoing quality control of both abstractors and clinician reviewers was conducted throughout the case identification and determination processes.[25] Reliability was evaluated in a blinded, random 10% sample of abstracted records scored independently by two reviewers. Average percentage inter-rater agreement on final case status was 96% (linear weighted K = 0.91). Project staff performed no clinical examinations of children.

Data analysis

We calculated period prevalence using as the denominator the number of 8-year-old children residing in the surveillance areas according to the NCHS bridged-race 2010 decennial census population estimates.[26]

For CP cases with available birth certificate information and no documented postneonatal aetiology of CP, we calculated the percentages, with 95% CIs, of those born LBW and VLBW. Appendix 1 provides information on the percentages born LBW and VLBW among all CP cases with available birthweight information (i.e. including those with and without a documented postneonatal aetiology), and Appendix 2 provides a summary of the demographic and clinical characteristics of all CP cases in each surveillance year, stratified by the availability of birth certificate data.

We performed Cochran–Armitage chi-square tests for trend[27] to evaluate the significance of trends over time in CP prevalence overall and in the frequency of LBW and VLBW among children with CP not attributed to postneonatal causes. Using the overall birth cohort as the comparison group, we calculated relative risks with 95% CIs[28] to evaluate associations between CP risk and categories of LBW relative to birthweight ≥2500 g. The term relative risk is used here as a generic term for measure of association. For LBW, the relative risks were computed by dividing the number of birth cohort members born LBW who were identified as having CP with no documented postneonatal aetiology by the total number of cohort members born LBW, and then dividing this by the number of birth cohort members not born LBW who were identified as having CP with no documented postneonatal aetiology divided by the total number of birth cohort members who were not born LBW. The same approach was used to calculate relative risks for VLBW. These relative risks are similar to prevalence ratios, as the CP cases are 'prevalent' rather than 'incident' cases. However, the CP case group does not include cases from the birth cohort who moved out of

the surveillance area before the age of 8 years. For this reason, we were unable to compute prevalence and prevalence ratios in the birth cohort overall.

We tested the significance of differences in the relative risks from 2006 to 2010 using the approach described by Altman and Bland.[29] *P*-values < 0.05 were considered statistically significant. We performed stratified analyses to evaluate the consistency of associations across subgroups and calculated population attributable risks to convey the proportion of CP cases in the population each year that may be attributed to LBW and VLBW, respectively.[30] We also evaluated the impact of adjusting for site effects on the associations between CP and both LBW and VLBW by computing adjusted odds ratios using logistic regression. Because the adjusted odds ratios were similar to the relative risks, we have not included them in this report. Statistical analyses were performed using SAS version 9.2 (SAS Institute Inc., Cary, NC, USA) and SPSS version 22 (IBM Corp., Armonk, NY, USA).

Each participating site met applicable local institutional review board and privacy/confidentiality requirements under 45 CFR 46.

Results

The prevalence of CP per 1000 8-year-old children in the combined surveillance area declined from 3.5 (95% CI 3.2, 3.9) in 2006 to 2.9 (95% CI 2.6, 3.2) in 2010 (Table 2, test for trend $P = 0.036$). Prevalence was lower in 2010 than in 2006 for both boys and girls, in each of the four surveillance sites, and among white non-Hispanic and Hispanic children, though the trends within most subgroups were not statistically significant. In contrast to other subgroups, no decline in the prevalence of CP was evident among black non-Hispanic children (Table 2).

In the birth cohort serving as the comparison group for the CP cases, the percent born LBW increased slightly, from 7.8 (95% CI 7.6, 7.9) in birth year 1998 to 8.1 (95% CI 8.0, 8.3) in 2002, while the percentage born VLBW was constant during the study period at 1.3% (Table 3).

In contrast, among children with CP not attributed to postneonatal causes and with available birthweight information, the percentage born LBW declined by 9.6% during the study period, from 54.3% (95% CI 48.4, 60.1) in 2006 to 49.1% (95% CI 42.9, 55.2) in 2010. This trend was not statistically significant (test for trend $P = 0.213$, Table 3). The percentage of these cases born VLBW declined by >20%, from 35.4% (95% CI 30.0, 41.2) in 2006 to 28.1% (95% CI 22.9, 34.0) in 2010 ($P = 0.066$, Table 3). The direction of these trends was fairly consistent across demographic subgroups (Table 3). Analogous results are provided in Appendix 3 for the categories of preterm and very preterm birth.

Comparing CP cases not attributed to postneonatal causes to the birth cohort overall, and using births ≥2500 g as the reference group, the relative risks indicating associations between CP and both LBW and VLBW appeared to decline during the study period, from 13.9 (95% CI 11.0, 17.4) in 2006 to 10.8 (95% CI 8.5, 13.7) in 2010 for LBW and from 39.1 (95% CI 30.9, 49.5) to 28.6 (95% CI 22.0, 37.2) for VLBW (Table 4). The differences

between the relative risks in 2006 and 2010, however, were not significantly different from zero for either LBW ($P = 0.136$) or VLBW ($P = 0.082$).

The population attributable risks for LBW were 50.2% (95% CI 41.2, 56.1) in 2006 and 44.3% (95% CI 37.9, 50.7) in 2010. For VLBW, they were 33.1% (95% CI 16.3, 38.7) in 2006 and 26.4% (95% CI 21.5, 32.0) in 2010.

Comments

This is the first population-based study in the United States to show a significant decline in the population prevalence of CP. It also showed preliminary evidence, though not statistically significant, of declining trends in the association between CP and both LBW and VLBW. The findings are encouraging given concern raised in earlier decades that increases in the survival of infants at greatest risk of CP (i.e. those born preterm and VLBW) may have given rise to increases in the population prevalence of CP.[5–7] This study was only able to monitor CP prevalence in a common surveillance area for the years 2006, 2008, and 2010. Although the results are encouraging, it is not possible to know from the data available whether the significant decline in CP prevalence observed during the study period is due to advances in health care for infants born VLBW. Nor is it possible to predict from the initial results reported here whether declines in CP prevalence and its association with LBW will continue after 2010.

Less hopeful were our findings of no decline in CP prevalence for black non-Hispanic children during the study period, and evidence that the racial disparity in CP prevalence appeared to increase over time. In 2010, the prevalence of CP was 68% higher in black children relative to white children (Table 2). These findings are difficult to reconcile with our observation that the contribution of LBW and VLBW to the risk of CP appeared to decline for both black and white children, and with previous research showing that the excess prevalence of CP among black children in the United States is largely explained by the excess frequency of LBW among black infants.[31,32] Further research is needed to identify other risk factors contributing to the excess prevalence of CP among black children and to identify and monitor strategies to reduce the risk of CP and to reduce racial disparities in CP risk in the United States.

The results of this study suggest that initial progress was made by 2010 towards the *HP2020* objective of reducing by 10% between 2006 and 2020 the percentage of children with CP who were born LBW. By 2010, this percentage among CP cases with no documented postneonatal aetiology had declined by 9.6%. Even more impressive was the 20% decline during the study period in the percentage of CP cases born VLBW. Although encouraging, these trends are limited to three surveillance years and were not statistically significant due to the limited size of the population covered by the ADDM Network CP surveillance system. Nor are the results based on a probability sample selected to be representative of the nation. We reiterate that ongoing monitoring is necessary to evaluate future trends and progress towards the *HP2020* CP objective. If improvements in neurodevelopmental outcomes of VLBW have occurred due to advances in perinatal and neonatal medicine in the 1990s and early 2000s, it is unclear that the pace of those advances will continue. Future trends in the

percentage of CP cases born LBW might also be affected by overall trends in the frequency of LBW and preterm birth among all livebirths. After decades of gradually increasing, the percentages of births in the United States that were LBW or preterm peaked in 2006 and have declined slightly over time since then.[33]

Additional limitations of this study are that birth-weight information was available only for the approximately 75% of CP cases who were born in the state in which they resided at age 8 years and that the occurrence of CP could not be ascertained for an unknown number of birth cohort members no longer residing in the surveillance area at that age. Because of these limitations, we reported the prevalence of CP in the population of 8-year-olds using census counts for the denominator, rather than the prevalence or cumulative incidence in the birth cohort. For evaluating birth-weight trends, it was necessary to rely on the birth cohort rather than census counts and the subset of CP cases with available birth certificate information. A limitation of this approach is that the relative risks could be affected by selection bias if the birthweight distributions of CP cases residing in the surveillance area at age 8 were different from those of an unknown number of CP cases from the birth cohort who could not be identified by the surveillance system, because they were no longer living in the surveillance area at age 8 years. However, we have no evidence that the birthweight distribution of CP cases from the birth cohort who could not be ascertained by the surveillance system because they did not reside in the surveillance area at age 8 differed from the distribution of CP cases from the birth cohort who were represented in the surveillance system. Moreover, even if these distributions did differ, we have no reason to think that the differences would have changed during the surveillance period to bias the relative risks or trends in the percentage of CP cases that were born LBW.

Another limitation of this study is that detailed information on prenatal, perinatal, and neonatal care was not available for the case group or cohort, precluding our ability to examine potential impacts at the population level of specific health care interventions that have been shown to improve neurodevelopmental outcomes of preterm birth and VLBW. Our observation that the frequency of LBW increased and the frequency of VLBW did not change over the study period in the overall cohort, while the relative risk for CP and both LBW and VLBW declined, though not significantly, is consistent with the possibility that advances in the perinatal and neonatal care of preterm infants might have contributed to declines in the risk of CP associated with LBW and the prevalence of CP in the population. Further research is needed to confirm this possibility and monitor future progress.

This study has a number of implications for public health monitoring related to CP. One is that to establish whether progress has been made towards the *HP2020* objective of reducing by 10% the percentage of CP cases born at LBW, it is necessary to monitor this percentage in a population large enough to be able to find a 10% reduction to be statistically significant. Based on our baseline of 54.3%, and the CP prevalence estimates and percent with available birthweight information estimated from this study, it would be necessary to perform ongoing surveillance of CP in a population of at least 512 000 8-year-olds in order to have 80% power to detect a 10% reduction as statistically significantly different from no change. For the study year 2010, the size of the ADDM CP Network surveillance population was limited to 131 352, and the power to detect a 10% reduction in the percentage of CP cases born

LBW in this population was only 28%. With the sample size available, the minimum reduction in the *HP2020* CP indicator that would be detectable given a power of 80% would be about 20%. If the initial trend from 2006 to 2010 were to continue beyond 2010, >a 20% reduction in the indicator could be achieved by the year 2020. Given the fact that VLBW is a much stronger risk factor for CP than the broader category of LBW, future public health objectives for CP would benefit from including reductions in the percentage of children with CP who were born VLBW.

In summary, our preliminary findings of progress towards the *HP2020* objective for CP and the significant decline during the study period in the population prevalence of CP are encouraging trends. Future monitoring of progress will require ongoing surveillance in relatively large surveillance areas that are consistent over time. These results also point to the need for further research to investigate the long-term developmental impacts at the population level of improvements in perinatal and neonatal care and to identify strategies to eliminate racial disparities in the United States in the risk of both LBW and CP.

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Appendix 1

Frequency of birthweight categories among all cerebral palsy (CP) cases (including those with documented post-neonatal aetiology) at age 8 years, by surveillance year, by race and ethnicity, sex, co-occurring epilepsy, and co-occurring autism spectrum disorder (ASD).

All CP cases with available birth certificate data, including those with documented postneonatal aetiology, age 8 years	Surveillance year 2006 (birth year 1998)	Surveillance year 2008 (birth year 2000)	Surveillance year 2010 (birth year 2002)
Total number of CP cases	316	314	286
% LBW	51.6	47.1	47.2
% VLBW	33.5	30.9	26.6
Number of white non-Hispanic cases	175	156	120
% LBW	43.4	40.4	40.8
% VLBW	20.6	21.8	19.2
Number of black non-Hispanic cases	114	121	126
% LBW	62.3	54.5	54.0
% VLBW	50.9	40.5	34.9
Number of Hispanic CP cases	13	26	26
% LBW	76.9	42.3	42.3
% VLBW	61.5	26.9	19.2
Number of boys	173	194	158
% LBW (95% CI)	49.1	48.5	44.3
% VLBW (95% CI)	32.4	30.4	27.2
Number of girls	143	120	128
% LBW (95% CI)	54.5	45.0	50.8
% VLBW (95% CI)	35.0	30.4	27.2
Number with co-occurring epilepsy	111	127	125
% LBW (95% CI)	43.2	45.7	44.0
% VLBW (95% CI)	26.1	26.0	25.6
Number with no co-occurring epilepsy	205	187	161
% LBW (95% CI)	56.1	48.1	49.7
% VLBW (95% CI)	37.6	33.8	27.3
Number with co-occurring ASD	20	21	23
% LBW (95% CI)	70.0	42.9	56.5
% VLBW (95% CI)	30.0	28.6	34.8

All CP cases with available birth certificate data, including those with documented postneonatal aetiology, age 8 years	Surveillance year 2006 (birth year 1998)	Surveillance year 2008 (birth year 2000)	Surveillance year 2010 (birth year 2002)
Number with no co-occurring ASD	296	293	263
% LBW (95% CI)	50.3	47.4	46.4
% VLBW (95% CI)	33.8	31.1	25.9

CI, confidence interval; LBW, low birthweight (<2500 g); VLBW, very low birthweight (<1500 g).

Appendix 2

Frequency and characteristics of cerebral palsy (CP) cases (aged 8 years) by availability of birth certificate data, by surveillance year (2006, 2008, and 2010)[a]

	Surveillance year 2006 (birth year 1998)			Surveillance year 2008 (birth year 2000)			Surveillance year 2010 (birth year 2002)		
	% Among CP cases with available birth data (*N* = 316)	% Among CP cases with no available birth data (*N* = 107)	% Among all CP (*N* = 423)	% Among CP cases with available birth data (*N* = 314)	% Among CP cases with no available birth data (*N* = 89)	% Among all CP (*N* = 403)	% Among CP cases with available birth data (*N* = 286)	% Among CP cases with no available birth data (*N* = 89)	% Among all CP (*N* = 375)
Male sex	54.7	60.7	56.3	61.8	56.2	60.5	55.2	53.9	54.9
Race/ethnicity									
White, non-Hispanic	55.4	45.8	53.0	49.7	34.8	49.7	42.0	38.2	41.1
Black, non-Hispanic	36.1	26.2	33.6	38.5	34.8	38.5	44.1	27.0	40.0
Hispanic	4.1	12.1	6.1	8.3	10.1	8.7	9.1	11.2	9.6
Other	4.1	9.7	5.4	3.5	3.4	3.5	4.5	11.2	6.2
Unknown	0	7.5	1.9	0	16.9	3.7	0.3	12.4	3.2
CP subtype									
Spastic	80.0	82.2	81.3	76.4	73.0	75.7	82.9	83.1	82.9
Hypotonic	4.4	5.6	4.7	5.3	6.5	5.5	6.3	1.1	5.1
Ataxic/dyskinetic	0.9	0.9	0.9	2.5	5.6	3.2	1.0	3.4	1.6
Mixed spastic	6.6	5.6	6.4	9.2	7.9	8.9	4.2	5.6	4.5
Other/unspecified	7.0	5.6	6.6	6.1	6.7	6.2	5.6	6.7	5.9
Extent of limb involvement									
Quadriplegia/triplegia	21.8	17.8	21.8	16.2	20.2	17.1	25.5	16.9	23.5
Diplegia	35.8	39.3	36.6	29.9	33.7	30.8	32.9	39.3	34.4
Hemiplegia/monoplegia	23.1	24.3	23.4	30.3	19.1	27.8	24.5	27.0	25.1
Unspecified	18.7	19.3	19.1	27.0	23.6	24.3	17.1	16.9	17.1
Documented postneonatal aetiology	7.9	6.5	7.6	6.4	5.6	6.2	6.6	10.1	7.5
GMFC available	74.4	76.6	74.9	73.2	83.1	75.4	73.8	75.3	74.1
GMFC among cases with available information									
I	35.1	47.9	38.2	40.8	39.1	40.5	45.0	56.7	47.8
II	16.9	15.1	16.4	12.9	23.2	15.2	7.1	9.0	7.6

	Surveillance year 2006 (birth year 1998)			Surveillance year 2008 (birth year 2000)			Surveillance year 2010 (birth year 2002)		
	% Among CP cases with available birth data (*N* = 316)	% Among CP cases with no available birth data (*N* = 107)	% Among all CP (*N* = 423)	% Among CP cases with available birth data (*N* = 314)	% Among CP cases with no available birth data (*N* = 89)	% Among all CP (*N* = 403)	% Among CP cases with available birth data (*N* = 286)	% Among CP cases with no available birth data (*N* = 89)	% Among all CP (*N* = 375)
III	13.4	12.3	13.2	15.0	2.9	12.3	9.0	7.5	8.6
IV	15.6	13.7	15.1	15.8	20.3	16.8	16.6	13.4	15.8
V	19.0	11.0	17.1	15.4	14.5	15.2	22.3	13.4	20.1
Walking ability available	74.9	77.3	75.4	72.6	77.2	74.7	81.1	86.5	82.4
Walking classification among cases with available information									
Walks independently	54.0	65.9	57.1	58.6	62.2	59.5	55.6	68.8	58.9
With assistive device	11.5	9.8	11.0	12.8	1.4	10.0	8.2	6.5	7.8
Limited or no walking	34.5	24.4	31.9	28.6	36.5	30.6	36.2	24.7	33.3
Co-occurring epilepsy	35.1	31.8	34.3	40.4	37.1	39.7	43.7	38.2	42.4
Co-occurring ASD	6.3	15.0	8.5	6.7	7.9	6.9	8.0	5.6	7.5

GMFC, Gross Motor Functional Classification; ASD, autism spectrum disorder.

[a]Results for surveillance years 2006 and 2008 have been reported previously based on a larger surveillance area.[11,12] The results here are restricted to a surveillance area composed of counties that were included in all three surveillance years.

Appendix 3

Frequency of preterm (<32 weeks' gestational age) and very preterm (<32 weeks' gestational age) birth among all livebirths surviving beyond 1 year and among cerebral palsy (CP) cases at age 8 years, by surveillance year, by race and ethnicity, sex, co-occurring epilepsy, and co-occurring autism spectrum disorder (ASD).

	Surveillance year 2006 (birth year 1998)	Surveillance year 2008 (birth year 2000)	Surveillance year 2010 (birth year 2002)	Test for trend *P*-value
Total number of births surviving to age 1 year	125 166	130 884	128 722	
Number with gestational age information at birth	125 025	130 803	128 676	
% Preterm (95% CI)	11.9 (11.7, 12.0)	11.9 (11.7, 12.0)	12.5 (12.3, 12.6)	<0.001
% Very preterm (95% CI)	1.8 (1.7, 1.9)	1.8 (1.8, 1.9)	1.8 (1.2, 1.4)	0.381
Total number of white, non-Hispanic births surviving to age 1 year (% of total white non-Hispanic births)	75 141	74 437	70 688	
% Preterm (95% CI)	9.9 (9.7, 10.1)	10.3 (10.0, 10.5)	10.8 (10.6, 11.1)	<0.001
% Very preterm (95% CI)	1.2 (1.1, 1.3)	1.3 (1.2, 1.3)	1.3 (1.2, 1.4)	0.161
Total number of black, non-Hispanic births surviving to age 1 year (% of total black non-Hispanic births)	37 587	38 898	37 005	

	Surveillance year 2006 (birth year 1998)	Surveillance year 2008 (birth year 2000)	Surveillance year 2010 (birth year 2002)	Test for trend *P*-value
% Preterm (95% CI)	16.6 (16.2, 16.9)	16.1 (15.7, 16.5)	17.1 (16.7, 17.5)	0.057
% Very preterm (95% CI)	3.3 (3.1, 3.5)	3.2 (3.1, 3.4)	3.1 (2.9, 3.2)	0.075
Total number of Hispanic births surviving to age 1 year (% of total Hispanic births)	6156	9348	11 831	
% Preterm (95% CI)	8.5 (7.8, 9.2)	9.1 (8.5, 9.7)	9.4 (8.9, 10.0)	0.038
% Very preterm (95% CI)	1.0 (0.8, 1.3)	0.9 (0.7, 1.1)	1.1 (0.9, 1.3)	0.367
CP cases with no documented postneonatal aetiology (age 8 years) with available birth certificate data				
Total number of CP cases	291	294	267	
Number with gestational age information at birth	290	294	267	
% Preterm (95% CI)	52.1 (46.2, 57.9)	52.0 (46.2, 57.9)	48.7 (42.6, 54.9)	0.213
% Very preterm (95% CI)	35.9 (30.3, 41.7)	35.4(30.0, 41.2)	31.1 (25.7, 37.1)	0.241
Number of white non-Hispanic cases	161	147	118	
% Preterm (95% CI)	44.7 (37.0, 52.7)	45.6 (37.4, 54.0)	43.2 (34.2, 52.7)	0.825
% Very preterm (95% CI)	22.2 (17.9, 31.7)	27.9 (21.0, 36.0)	23.7 (16.6, 32.6)	0.989
Number of black non-Hispanic cases	103	111	110	
% Preterm (95% CI)	61.2 (51.0, 70.5)	58.6 (48.8, 67.7)	55.5 (45.7, 64.8)	0.398
% Very preterm (95% CI)	54.2 (42.4, 62.3)	44.1 (34.8, 53.9)	39.1 (30.1, 48.9)	0.052
Number of Hispanic CP cases	13	25	25	
% Preterm (95% CI)	76.9 (46.0, 93.8)	52.0 (31.8, 71.7)	44.0 (25.0, 64.7)	0.068[a]
% Very preterm (95% CI)	53.9 (26.1, 79.6)	28.0 (12.9, 49.6)	24.0 (10.2, 45.5)	
Number of boys	158	181	143	
% Preterm (95% CI)	50.6 (42.6, 58.6)	54.7 (47.2, 62.1)	49.0 (40.1, 57.4)	0.795
% Very preterm (95% CI)	34.2 (27.0, 42.2)	35.4 (28.4, 42.9)	33.6 (26.0, 42.0)	0.919
Number of girls	132	113	124	
% Preterm (95% CI)	53.8 (44.9, 62.4)	47.8 (38.4, 57.4)	48.4 (39.4, 57.5)	0.383
% Very preterm (95% CI)	37.9 (29.7, 46.8)	35.4 (26.8, 45.0)	28.2 (20.7, 37.1)	0.105
Number with co-occurring epilepsy	99	122	114	
% Preterm (95% CI)	43.4 (33.6, 53.8)	46.7 (37.7, 55.9)	46.5 (37.2, 56.0)	0.665
% Very preterm (95% CI)	29.3 (20.8, 39.4)	24.6 (17.5, 33.4)	28.1 (20.3, 37.4)	0.869
Number with no co-occurring epilepsy	191	172	153	
% Preterm (95% CI)	56.5 (49.2, 63.6)	55.8 (48.1, 63.3)	50.3 (42.8, 59.1)	0.263
% Very preterm (95% CI)	39.3 (32.4, 46.6)	43.0 (35.6, 50.8)	33.3 (26.1, 41.5)	0.303
Number with co-occurring ASD	20	20	23	
% Preterm (95% CI)	55.0 (32.1, 76.2)	50.0 (27.9, 72.2)	52.2 (31.2, 72.6)	0.862
% Very preterm (95% CI)	30.0 (12.8, 54.3)	30.0 (12.8, 54.3)	34.8 (17.2, 57.2)	0.733
Number with no co-occurring ASD	270	274	244	
% Preterm (95% CI)	51.9 (45.7, 57.9)	52.2 (46.1, 58.2)	48.4 (42.0, 54.8)	0.439

	Surveillance year 2006 (birth year 1998)	Surveillance year 2008 (birth year 2000)	Surveillance year 2010 (birth year 2002)	Test for trend *P*-value
% Very preterm (95% CI)	36.3 (30.6, 42.4)	35.8 (30.2, 41.8)	30.7 (25.1, 37.0)	0.192

ASD, autism spectrum disorder; CI, confidence interval; CP, cerebral palsy.

[a] *P*-value not calculated due to small expected cell sizes.

Table 1

Population characteristics for 8-year-old children by site, Autism and Developmental Disabilities Monitoring Network, surveillance years 2006, 2008, and 2010[a]

	Surveillance year		
	2006 *n* (%)	**2008 *n* (%)**	**2010 *n* (%)**
Total 8-year-old children	121 843 (100)	124 632 (100)	131 352 (100)
Male	62 062 (50.9)	63 786 (51.2)	66 604 (50.7)
Race and ethnicity			
White non-Hispanic	65 189 (53.5)	65 094 (52.2)	67 801 (51.6)
Black non-Hispanic	40 053 (32.9)	40 119 (32.2)	40 420 (30.8)
Hispanic	11 090 (9.1)	13 899 (11.2)	16 956 (12.9)
Other	5511 (4.5)	5520 (4.4)	6175 (4.7)
Surveillance site			
Alabama[b]	21 101 (17.3)	21 473 (17.2)	21 833 (16.6)
Georgia[c]	43 241 (35.5)	45 248 (36.3)	48 529 (37.0)
Missouri[d]	26 138 (21.5)	25 301 (20.3)	25 367 (19.3)
Wisconsin[e]	31 363 (25.7)	32 610 (26.2)	35 623 (27.1)

[a]Data were obtained from the National Center on Health Statistics' bridged-race census population estimates for each surveillance year. For 2006 and 2008, they were from intercensal population estimates.

[b]The Alabama site included nine counties in central Alabama.

[c]The Georgia site included five counties of metropolitan Atlanta.

[d]The Missouri site included five counties in metropolitan St. Louis.

[e]The Wisconsin site included 10 counties in southeastern Wisconsin.

Table 2

Prevalence per 1000 (95% CI) of cerebral palsy (CP) among 8-year-old children by site, Autism and Developmental Disabilities Monitoring Network, surveillance years 2006, 2008, and 2010[a]

	Surveillance year			
	2006 (*N* = 423 CP cases) prevalence per 1000 (95% CI)	**2008 (*N* = 403 CP cases) prevalence per 1000 (95% CI)**	**2010 (*N* = 375 CP cases) prevalence per 1000 (95% CI)**	**Test for trend *P*-value**
Overall	3.5 (3.2, 3.9)	3.2 (2.9, 3.5)	2.9 (2.6, 3.2)	0.036
Sex				
Male	3.8 (3.3, 4.3)	3.8 (3.4, 4.3)	3.1 (2.7, 3.5)	0.083
Female	3.1 (2.7, 3.6)	2.6 (2.2, 3.0)	2.6 (2.2, 3.0)	0.236
Race and ethnicity				
White non-Hispanic	3.4 (3.0, 3.9)	2.9 (2.5, 3.3)	2.2 (1.9, 2.6)	0.002
Black non-Hispanic	3.5 (3.0, 4.1)	3.8 (3.2, 4.5)	3.7 (3.2, 4.3)	0.536
Hispanic	2.3 (1.6, 3.4)	2.5 (1.8, 3.5)	2.1 (1.5, 2.9)	0.191
Surveillance site				
Alabama[b]	3.0 (2.3, 3.8)	3.3 (2.6, 4.2)	2.4 (1.8, 3.2)	0.392
Georgia[c]	4.1 (3.5, 4.7)	4.0 (3.5, 4.6)	3.4 (2.9, 4.0)	0.094
Missouri[d]	3.2 (2.6, 4.0)	2.5 (2.0, 3.2)	2.7 (2.1, 3.4)	0.311
Wisconsin[e]	3.1 (2.5, 3.8)	2.7 (2.2, 3.3)	2.5 (2.0, 3.0)	0.481
Documented postneonatal cause				
No	3.2 (2.9, 3.5)	3.0 (2.7, 3.3)	2.6 (2.3, 2.9)	0.046[f]
Yes	0.3 (0.2, 0.4)	0.2 (0.1, 0.3)	0.2 (0.1, 0.3)	

CI, confidence interval.

[a] The prevalence results for surveillance years 2006 and 2008 have been reported previously based on a larger surveillance area.[11,12] The results here are restricted to a surveillance area composed of counties that were included in all three surveillance years.

[b] The Alabama site included nine counties in central Alabama.

[c] The Georgia site included five counties of metropolitan Atlanta.

[d] The Missouri site included five counties in metropolitan St. Louis.

[e] The Wisconsin site included 10 counties in southeastern Wisconsin.

[f] *P*-value not calculated due to small expected cell sizes.

Table 3

Frequency of birthweight categories among all livebirths surviving beyond 1 year, and among cerebral palsy (CP) cases at age 8 years, by surveillance year, by race and ethnicity, sex, co-occurring epilepsy, and co-occurring autism spectrum disorder (ASD)

	Surveillance year 2006 (birth year 1998)	Surveillance year 2008 (birth year 2000)	Surveillance year 2010 (birth year 2002)	Test for trend *P*-value
Total number of births surviving to age 1 year (% of total births)	125 166	130 884	128 722	
% LBW (95% CI)	7.8 (7.6, 7.9)	7.9 (7.8, 8.1)	8.1 (8.0, 8.3)	0.002
% VLBW (95% CI)	1.3 (1.2, 1.4)	1.3 (1.3, 1.4)	1.3 (1.2, 1.4)	0.787
Total number of white, non-Hispanic births surviving to age 1 year (% of total white non-Hispanic births)	75 172	74 459	70 705	
% LBW (95% CI)	5.9 (5.7, 6.1)	6.2 (6.0, 6.4)	6.3 (6.1, 6.5)	0.003
% VLBW (95% CI)	0.8 (0.7, 0.9)	0.9 (0.8, 1.0)	0.9 (0.8, 1.0)	0.183
Total number of black, non-Hispanic births surviving to age 1 year (% of total black non-Hispanic births)	37 689	38 936	37 025	
% LBW (95% CI)	12.1 (11.8, 12.4)	12.0 (11.8, 12.5)	12.6 (12.3, 12.9)	0.049
% VLBW (95% CI)	2.4 (2.3, 2.6)	2.4 (2.3, 2.6)	2.3 (2.2, 2.5)	0.379
Total number of Hispanic births surviving to age 1 year (% of total Hispanic births)	8510	12 604	15 464	
% LBW (95% CI)	5.4 (4.9, 5.9)	5.7 (5.3, 6.1)	5.7 (5.3, 6.1)	0.088
% VLBW (95% CI)	0.8 (0.6, 1.0)	0.8 (0.7, 1.0)	0.8 (0.7, 1.0)	0.701
CP cases with no documented postneonatal aetiology (age 8 years) with available birth certificate data				
Total number of CP cases	291	294	267	
% LBW (95% CI)	54.3 (48.4, 60.1)	50.0 (44.2, 55.9)	49.1 (42.9, 55.2)	0.213
% VLBW (95% CI)	35.4 (30.0, 41.2)	32.7 (27.4, 38.4)	28.1 (22.9, 34.0)	0.066
Number of white non-Hispanic cases	161	147	118	
% LBW (95% CI)	46.0 (38.2, 54.0)	42.9 (34.8, 51.3)	40.7 (31.0, 50.1)	0.373
% VLBW (95% CI)	22.4 (16.3, 29.7)	23.1 (16.8, 30.9)	19.5 (13.0, 28.0)	0.598
Number of black non-Hispanic cases	104	111	110	
% LBW (95% CI)	65.4 (55.3, 74.3)	58.6 (48.9, 67.7)	60.0 (50.2, 69.1)	0.427
% VLBW (95% CI)	52.9 (42.9, 62.7)	43.2 (34.0, 53.0)	40.0 (30.9, 49.8)	0.060
Number of Hispanic CP cases	13	25	25	
% LBW (95% CI)	76.9 (46.0, 93.8)	44.0 (25.0, 64.7)	40.0 (21.8, 61.1)	0.050[a]
% VLBW (95% CI)	61.5 (32.3, 84.9)	28.0 (12.9, 49.6)	16.0 (5.3, 36.9)	
Number of boys	158	181	143	
% LBW (95% CI)	51.9 (43.9, 59.9)	51.4 (43.9, 58.8)	53.1 (38.5, 55.4)	0.389
% VLBW (95% CI)	34.2 (27.0, 42.2)	32.0 (25.4, 39.4)	29.4 (22.2, 37.7)	0.373
Number of girls	133	113	124	
% LBW (95% CI)	57.1 (48.3, 65.6)	47.8 (38.4, 57.4)	51.6 (42.5, 60.6)	0.364
% VLBW (95% CI)	36.8 (28.8, 45.7)	33.6 (25.2, 43.2)	26.6 (19.3, 35.4)	0.082
Number with co-occurring epilepsy	100	122	114	
% LBW (95% CI)	47.0 (37.0, 57.2)	47.5 (38.5, 56.7)	46.5 (37.2, 56.0)	0.936

	Surveillance year 2006 (birth year 1998)	Surveillance year 2008 (birth year 2000)	Surveillance year 2010 (birth year 2002)	Test for trend *P*-value
% VLBW (95% CI)	29.0 (20.6, 39.1)	27.0 (19.6, 36.0)	27.2 (19.5, 36.5)	0.775
Number with no co-occurring epilepsy	191	172	153	
% LBW (95% CI)	58.1 (50.8, 65.1)	51.7 (44.3, 59.1)	51.0 (42.8, 59.1)	0.174
% VLBW (95% CI)	38.7 (31.9, 46.1)	36.6 (29.5, 44.4)	28.8 (21.9, 36.7)	0.059
Number with co-occurring ASD	20	20	23	
% LBW (95% CI)	70.0 (45.7, 87.2)	45.0 (23.8, 68.0)	56.5 (34.9, 76.1)	0.408
% VLBW (95% CI)	30.0 (12.8, 54.3)	30.0 (12.8, 54.3)	34.8 (17.2, 57.2)	0.733
Number with no co-occurring ASD	271	274	244	
% LBW (95% CI)	53.1 (47.0, 59.2)	50.4 (44.3, 56.4)	48.4 (42.0, 54.8)	0.278
% VLBW (95% CI)	35.8 (30.1, 42.0)	32.8 (27.4, 38.8)	27.5 (22.1, 33.6)	0.044

CI, confidence interval; LBW, low birthweight (<2500 g); VLBW, very low birthweight (<1500 g).

[a] *P*-value not calculated due to small expected cell sizes.

Table 4

Relative risks (95% CI) for CP[a] by low birthweight categories (reference category = birthweight ≥2500 g) by surveillance year, overall and by race/ethnicity

	Surveillance year 2006 (birth year 1998) Relative risk (95% CI)	Surveillance year 2008 (birth year 2000) Relative risk (95% CI)	Surveillance year 2010 (birth year 2002) Relative risk (95% CI)
Overall			
≥2500 g	1.0 (reference)	1.0 (reference)	1.0 (reference)
LBW[b]	13.9 (11.0, 17.4)	11.5 (9.2, 14.4)	10.8 (8.5, 13.7)
VLBW[c]	39.1 (30.9, 49.5)	34.2 (26.9, 43.4)	28.6 (22.0, 37.2)
Boys			
≥2500 g	1.0 (reference)	1.0 (reference)	1.0 (reference)
LBW	13.9 (10.2, 19.0)	13.3 (9.9, 17.7)	11.0 (7.9, 15.2)
VLBW	39.5 (28.7, 54.5)	33.4 (24.6, 45.3)	32.7 (23.0, 46.6)
Girls			
≥2500 g	1.0 (reference)	1.0 (reference)	1.0 (reference)
LBW	14.1 (10.0, 19.9)	10.0 (6.7, 14.0)	10.8 (7.6, 15.3)
VLBW	39.1 (27.7, 55.4)	35.6 (24.2, 52.3)	24.8 (16.8, 36.8)
White non-Hispanic			
≥2500 g	1.0 (reference)	1.0 (reference)	1.0 (reference)
LBW	13.3 (9.8, 18.1)	11.2 (8.1, 15.5)	10.1 (7.0, 14.6)
VLBW	32.1 (22.3, 46.1)	31.0 (21.3, 45.1)	25.5 (16.3, 40.0)
Black non-Hispanic			
≥2500 g	1.0 (reference)	1.0 (reference)	1.0 (reference)
LBW	13.4 (9.0, 20.2)	10.2 (7.0, 14.9)	10.3 (7.0, 15.0)
VLBW	43.3 (29.7, 63.4)	29.9 (20.7, 43.3)	27.1 (18.6, 39.5)
Hispanic			
≥2500 g	1.0 (reference)	1.0 (reference)	1.0 (reference)
LBW	59.6 (16.5, 215.5)	13.5 (6.2, 29.6)	10.8 (4.9, 24.0)
VLBW	188.4 (63.8, 556.6)	54.1 (23.4, 125.2)	24.5 (8.5, 69.6)

CI, confidence interval; LBW, low birthweight (<2500 g); VLBW, very low birthweight (<1500 g).

[a] CP cases restricted to those not attributed to a postneonatal cause.

[b] *P*-value = 0.136 for the difference between the relative risk in 2006 and 2010.

[c] *P*-value = 0.082 for the difference between the relative risk in 2006 and 2010.