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Birth Prevalence of Cerebral Palsy: A Population-Based Study

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Abstract

OBJECTIVE—Population-based data in the United States on trends in cerebral palsy (CP) birth prevalence are limited. The objective of this study was to examine trends in the birth prevalence of congenital spastic CP by birth weight, gestational age, and race/ethnicity in a heterogeneous US metropolitan area.

METHODS—Children with CP were identified by a population-based surveillance system for developmental disabilities (DDs). Children with CP were included if they were born in metropolitan Atlanta, Georgia, from 1985 to 2002, resided there at age 8 years, and did not have a postneonatal etiology ($n = 766$). Birth weight, gestational age, and race/ethnicity subanalyses were restricted to children with spastic CP ($n = 640$). Trends were examined by CP subtype, gender, race/ethnicity, co-occurring DDs, birth weight, and gestational age.

RESULTS—Birth prevalence of spastic CP per 1000 1-year survivors was stable from 1985 to 2002 (1.9 in 1985 to 1.8 in 2002; 0.3% annual average prevalence; 95% confidence interval [CI] –1.1 to 1.8). Whereas no significant trends were observed by gender, subtype, birth weight, or gestational age overall, CP prevalence with co-occurring moderate to severe intellectual disability significantly decreased (–2.6% [95% CI –4.3 to –0.8]). Racial disparities persisted over time between non-Hispanic black and non-Hispanic white children (prevalence ratio 1.8 [95% CI 1.5 to 2.1]). Different patterns emerged for non-Hispanic white and non-Hispanic black children by birth weight and gestational age.

CONCLUSIONS—Given improvements in neonatal survival, evidence of stability of CP prevalence is encouraging. Yet lack of overall decreases supports continued monitoring of trends

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and increased research and prevention efforts. Racial/ethnic disparities, in particular, warrant further study.

Cerebral palsy (CP) is a group of permanent disorders of movement and posture causing activity limitations, attributed to nonprogressive disturbances in the brain occurring early in development.¹ Prevalent in ~3 to 4 per 1000 children in the United States, CP is the most common motor disability in childhood.^{2,3} Over the past 5 decades, remarkable improvements have been made in obstetric and neonatal care, resulting in significant declines in infant mortality both in the United States and abroad, particularly for infants born premature and of very low birth weight.^{4–6} Successes in neonatal survival have been met by concerns of resultant increases in adverse neurodevelopmental sequelae, with a major focus on CP.^{7–9}

A meta-analysis reported stability in overall CP prevalence when cohorts of children with CP from the early 1980s to 2004 were CP from the early 1980s to 2004 were incorporated.¹⁰ Trends in birth weight and gestational age-specific CP prevalence from international surveillance programs have shown varied trends, with significant declines from 1980 to 1998 in CP prevalence among children born with very low birth weight (VLBW) (<1500 g), very preterm (VPT) (<32 weeks), and moderately preterm (32 to 36 weeks) and stable prevalence among children born with moderately low birth weight (MLBW) (1500–2499 g) or normal birth weight (NBW) (≥2500 g).^{11–13} Comparable CP prevalence trend data for the US population by birth weight and gestational age are limited.

Although infant mortality in the United States has decreased overall, racial/ethnic disparities are striking, with higher rates among non-Hispanic black (NHB) compared with non-Hispanic white (NHW) infants, particularly among those born small and premature.⁵ Concurrently, several US cross-sectional studies have observed higher CP prevalence among NHB compared with NHW children. A cross-sectional study of California children with CP born between 1991 and 2001 found that this disparity was attributable to the higher frequency of VLBW and MLBW among NHB children.¹⁴ Recently, the Autism and Developmental Disabilities Monitoring (ADDM) Network reported a similar disparity, with higher CP prevalence among NHB compared with NHW children, which remained after adjustment for maternal education.¹⁵ It is unclear whether these racial/ethnic differences in CP prevalence in the United States have persisted over time. Given the race/ethnicity differential in US preterm births over time,¹⁶ an examination of how racial/ethnic differences in CP prevalence are related to racial-ethnic disparities in birth weight and gestational age is needed. Building on earlier work in metropolitan Atlanta among children born in 1975 through 1991,¹⁷ this investigation examines CP birth prevalence trends from 1985 through 2002, with specific focus on differences by birth weight, gestational age, and race/ethnicity.

METHODS

Case Ascertainment

Children with CP were identified through the Metropolitan Atlanta Developmental Disabilities Surveillance Program (MADDSP), a population-based surveillance system for 5

developmental disabilities (DDs): CP, intellectual disability (ID), hearing loss (HL), vision impairment (VI), and autism spectrum disorder (ASD). For each surveillance cycle, 8-year-old children residing within the 5-county metropolitan Atlanta area were eligible for inclusion. This study included children born in 1985, 1986, 1988, 1992, 1994, 1996, 1998, 2000, or 2002 identified at age 8 years with 1 of the 5 DDs.

MADDSP is based on review of administrative records from public school special education programs and health care providers who conduct pediatric developmental evaluations. Trained staff screen records for an indication of 1 of the 5 DDs. For CP, records with description of physical or diagnostic findings consistent with possible CP are fully abstracted. Information from multiple education and health records is consolidated into a single composite record for each child, which is systematically reviewed by a team of clinical reviewers for final case determination. CP is defined as a group of permanent disorders of the development of movement and posture that are attributed to nonprogressive disturbances that occurred in the developing brain.¹ Our original case definition (Mutch et al¹⁸) was expanded to reflect the broader considerations of CP defined by Rosenbaum et al,¹ yet surveillance is confirmed by the presence of a documented CP diagnosis or physical findings consistent with CP in an evaluation by a qualified professional at or after age 2 years.

Children with CP are categorized according to their predominant CP subtype: (1) spastic: spastic unilateral (hemiplegia, monoplegia), spastic bilateral (diplegia, quadriplegia, triplegia), mixed spastic subtypes (spastic-ataxic and spastic-dyskinetic), and spastic not otherwise specified; (2) nonspastic: ataxic, dyskinetic, hypotonic, ataxic- dyskinetic; and (3) CP not otherwise specified. Using a similar systematic review process, HL, ID, and VI are objectively defined by qualifying test results,¹⁹ with ASD confirmed by trained clinician reviewers applying a standardized coding scheme based on the Diagnostic and Statistical Manual, Fourth Edition, Text Revision.²⁰ All children are linked to Georgia birth-death vital statistics records to exclude those who died before the surveillance year.

Study Population

For these analyses, children with an event occurring after 28 days of life determined by study staff to be causal in the development of CP (postneonatal CP, $n = 118$) and those born outside of the surveillance area ($n = 442$) were excluded from the analysis, resulting in 766 children who met the inclusion criteria. Birth weight analyses were further restricted to children with congenital spastic CP ($n = 640$) owing to small sample sizes of the other individual CP subtypes and the increased likelihood of children with lower birth weight developing spastic CP.¹¹ Therefore, children with hypotonic CP ($n = 41$), other nonspastic CP ($n = 21$), and CP not otherwise specified ($n = 61$) were excluded. Fifteen children were missing gestational age information.

MADDSP is deemed public health practice by the Centers for Disease Control and Prevention's Institutional Review Board, functions as a public health authority in accordance with its data source agreements, and meets applicable privacy/confidentiality requirements under 45 CFR 46.²¹

Statistical Analyses

Birth Prevalence—Birth prevalence estimates were calculated per 1000 1-year survivors. Numerators included children born in specified years (1985–2002) in the 5-county surveillance area who still resided there at age 8 years and met the surveillance case definition for congenital CP ($n = 766$). One-year survivor denominator data were obtained from linked birth-infant death files for mothers who resided in the surveillance area at the index child's time of birth.

Birth Weight and Gestational Age Analyses—Birth weight data were categorized as (1) VLBW (<1500 g), (2) MLBW (1500–2499 g), and (3) NBW (≥2500 g). Gestational age was defined based on last menstrual period or clinical estimate if last menstrual period data were missing and was categorized as (1) VPT (<32 weeks), (2) moderately preterm (32–36 weeks), and (3) term (≥37 weeks).

Negative binomial and Poisson regression (with a parameter to correct for overdispersion) were used to model linear trends in observed single-year prevalence.²² We used χ^2 goodness-of-fit tests to examine the fit of each trend ($P > .05$).²³ To evaluate trends in congenital spastic CP birth prevalence by birth weight, gestational age, and race/ethnicity, we combined data into 3 time periods indicated by birth years: (1) 1985 to 1988, (2) 1992 to 1996, and (3) 1998 to 2002. Because of small sample sizes, birth weight- and gestational age-specific analyses did not include Hispanic children. Cochran–Armitage trend tests were used to assess the significance of trends within the subgroups across the 3 time periods.²⁴ Prevalence ratios were calculated using Poisson regression, and χ^2 statistics were used to evaluate changes in subgroup distributions over time.

RESULTS

The overall birth prevalence of congenital CP increased modestly from 1.9 per 1000 1-year survivors in 1985 to 2.2 in 2002, representing an average annual increase of 1.2% (95% confidence interval [CI] 0.1 to 2.3) (Fig 1). However, when restricted to children with spastic CP there were no significant trends in birth prevalence (average annual change in birth prevalence of 0.3% [95% CI –1.1 to 1.8]) (Table 1). No changes in prevalence were observed by spastic laterality. A significant increase was observed for children with hypotonic CP, driven primarily by higher prevalence in 2008 and 2010, which contributed to the small overall increase (Fig 1).

Congenital Spastic CP

Trends in congenital spastic CP prevalence were stable for males and females, with comparable average annual prevalence estimates of 1.9 and 1.7 per 1000 1-year survivors, respectively (Table 1). Whereas there was no change in CP prevalence for either NHW or NHB children from 1985 to 2002, CP prevalence among Hispanic children decreased from 1994 (2.1) to 2002 (1.3) (–4.6% annually [95% CI –7.3 to –1.8]). Combining all surveillance years, CP prevalence among NHB children was ~80% higher than among NHW children (prevalence ratio [PR] 1.8 [95% CI 1.5 to 2.1]) and 50% higher than among Hispanic children (PR 1.5 [95% CI 1.1 to 2.0]). CP prevalence with co-occurring ASD, mild

ID, HL, and VI did not change from 1985 to 2002. A decrease was observed among children with CP and co-occurring moderate to profound ID, from 0.7 to 0.4 per 1000 -2.6% annually [95% CI -4.3 to -0.8]. On average, ID (52%) was the most common co-occurring DD, followed by VI (17%), ASD (6%), and HL (5%).

Birth Weight and Gestational Age

Infant mortality in metropolitan Atlanta decreased significantly from 1985 to 2002, most notably from 1985 to 1996 among infants born VLBW and/or preterm (Supplemental Figure 3). The categorical distributions of birth weight and gestational age among 1-year survivors changed significantly across the 3 time periods ($P < .0001$ and $P < .003$, respectively), with increases among children born VLBW (1.1% in 1985–1988 to 1.4% in 1998–2002) or VPT (1.4%–1.6%, respectively) (data not shown). Whereas we observed an increase in the proportion of children with CP born VLBW, from 35% in 1985 to 1988 to 42% in 1998 to 2002 (Fig 2A), along with slight decreases within MLBW (22%–18%) and NBW (43%–41%) categories, these changes were not significant ($P = .67$) (Fig 2A). The categorical distribution of gestational age among children with CP was stable as well ($P = .32$) (Fig 2B).

A dramatic inverse relationship was observed comparing children with CP born VLBW versus NBW (PR 68.0 [95% CI 57.4 to 80.6]) and MLBW versus NBW (PR 6.6 [5.3 to 8.1]) when all years were combined. Yet, across the 3 time periods, no significant trends in CP prevalence by birth weight or gestational age were observed (Table 2). These overall birth weight and gestational age patterns were observed among children with spastic bilateral CP as well.

Birth Weight, Gestational Age, and Race/Ethnicity

Across all time periods, a greater percentage of NHB than NHW children with CP were born VLBW. The percentage of NHB children born VLBW increased over time (39% in 1985–1988 to 50% in 1998–2002), whereas the percentage of NHW children born VLBW decreased (32%–28%, respectively). Nevertheless, the birth weight and gestational age distributions among NHW and NHB children with CP did not change significantly across the 3 time periods.

CP birth prevalence was stable across all 3 birth weight and gestational age groups for NHW and NHB children with CP, with the exception of a decrease in prevalence among NHW children who were born VLBW (69.0 in 1985–1988 to 39.3 in 1998–2002, $P < .05$) (Table 3). No changes in prevalence were observed for NHB children born at <1500 g or for NHB or NHW children born at <32 weeks. Higher proportions of NHB children with CP were born VLBW, but when data from all years were combined, the prevalence of CP among VLBW births was not significantly higher among NHB than NHW children. Conversely, CP prevalence among children born NBW and/or term was higher among NHB than NHW children (NHB to NHW PR for NBW 1.4 [95% CI 1.1 to 1.9] and for term 1.6 [95% CI 1.2 to 2.0]).

DISCUSSION

This report provides the most recent population-based data in the United States examining trends in CP birth prevalence by demographic characteristics, co-occurring DDs, birth weight, and gestational age. In general, our findings are consistent with those from other CP surveillance programs in Europe and Australia that have reported increases in CP birth prevalence over the 1970s and 1980s, with stable or declining estimates from the early to mid-1990s to early 2000s.^{25–29} With continued monitoring in the same metropolitan Atlanta surveillance area, our findings also support those reported by Winter et al¹⁷ of a modest increase in CP birth prevalence among 1-year survivors, from 1.7 (1975–1977) to 2.0 (1986–1991) per 1000 ($P < .02$). When we restricted our sample to children with spastic CP, we did not see changes in overall prevalence or by laterality. This stability was also present in the Winter et al cohorts¹⁷ when similarly restricted to children with spastic CP (1.3 in 1975–1977 to 1.6 in 1981–1985 and 1.5 in 1986–1991, $P = .37$).

Whereas empirical evidence across the 3 time periods under study demonstrates increased neonatal survival, most apparently among infants born VPT and VLBW, this coincides with significant changes in the management of preterm birth and the care of premature infants. These include initiation of more aggressive use of assisted ventilation (1985–1988); introduction of surfactant and ante- and postnatal steroid therapies (1992–1996); and decreases in postnatal steroid use coupled with increases in use of other less invasive measures for respiratory support and prevention of related conditions (eg, sepsis) in the early 2000s onward.^{30–36} Our observation of stable prevalence of congenital spastic CP over this time period suggests that improved survival among children born at lower birth weights and gestational ages has not resulted in an increased frequency of spastic CP in the population. Conversely, the noted improvements in neonatal care over this same time period pose an expectation of improved neurodevelopmental outcomes among VLBW and VPT births, a decline in CP prevalence not supported by our results. Nonetheless, we did observe a decrease in the prevalence of CP and co-occurring moderate to profound ID, a finding similar to that reported by Sellier et al among NBW births in Europe.¹³ These findings are encouraging and suggest that improvements in neonatal care may be resulting in prevention of more severe forms of CP. Although our frequencies of CP and co-occurring mild ID, HL, VI, and ASD are comparable to those from other surveillance programs, the absence of declines emphasizes the need to address functional limitations involving multiple systems and develop screening tools for early identification of co-occurring DDs among children with CP.^{37–39} In addition, the frequencies of CP and co-occurring ID, VI, ASD, and HL are higher than the population prevalence of each DD (15 per 1000 for ASD, 13 per 1000 for ID, and 1 per 1000 for HL and VI) suggesting potentially shared etiologies.³⁹

Our birth weight-specific findings of stable trends among 1-year survivors were consistent with earlier work from metropolitan Atlanta for children born VLBW and MLBW. In contrast to the modest increase CP in CP prevalence among children born NBW found by Winter et al,¹⁷ our more recent cohorts indicated stability among NBW births in the 1990s and early 2000s. This stability is comparable to that reported among term births in California from 1991 to 2003 and children born NBW in Western Australia from 1985 to 1999.^{26,40} Contrary to findings from Surveillance of Cerebral Palsy in Europe, we did not find

opposing trends between spastic bilateral (decreasing) and unilateral (increasing) CP among NBW births.²⁸

Racial/Ethnic Disparities

Our findings of consistently higher prevalence among NHB compared with NBW and Hispanic children support earlier work by Winter et al,¹⁷ data from the ADDM Network, and a study of California births from 1991 to 2001.^{14,15} The ADDM Network found that the higher CP prevalence among NHB versus NHW children persisted after controlling for socioeconomic status but was no longer significantly different after controlling for prematurity. Wu et al¹⁴ described a lower risk of CP among NHB than NHW children born VLBW and MLBW and no disparity for those born NBW. In contrast, our data support a potentially higher risk for NHB versus NHW children born NBW. Interestingly, although NHB children were more likely to have an adverse perinatal outcome and, as a consequence, be at higher risk for CP, NHB children born VLBW and MLBW did not have higher estimates of CP prevalence than their NHW counterparts in the same birth weight or gestational age categories. Because of small sample sizes, it is challenging to interpret the decrease in CP prevalence among NHW children born at ≥ 1500 g. Ongoing surveillance will yield additional data to evaluate if this decreasing trend, as well as that among Hispanic children, continues.

Strengths and Limitations

MADDSP's multiple source record review methodology is not subject to biases inherent in single-source ascertainment. We used comparable surveillance methods since the inception of the program, which strengthens our internal validity and ability to examine trends. In addition, the racial/ethnic diversity in metropolitan Atlanta enabled us to examine NHW and NHB disparities in CP prevalence. Of note, MADDSP's birth prevalence (2.2 per 1000 in 2010) is comparable to that of other surveillance programs, but lower than MADDSP's traditionally reported period prevalence (3.4 per 1000 in 2010) owing to the choice of denominator (children versus 1-year survivors). For MADDSP, birth prevalence is likely an underestimate of CP population prevalence, as mortality between ages 1 and 8 and outmigration are not accounted for in the 1-year survivor birth denominator.⁴¹

We exercise some caution with interpretation of our birth weight– specific results, as small changes may have gone undetected. In addition, because of small sample sizes, we were limited in our ability to adequately test for trends within refined birth weight and gestational age and CP subtype subgroups, specifically <1000 g and <28 weeks' gestation. Finally, gestational age data were missing for 15 children with CP, 14 of whom were in the first time period. These missing data were more frequent for NHB children born at <1500 g ($n = 6/9$) compared with NHW children ($n = 2/5$) which may limit some of the gestational age trend findings.

CONCLUSIONS

It is encouraging that the prevalence of congenital spastic CP did not increase over the 17-year time period, yet the absence of decline underscores the continued need for resources

and support of children with CP and their families, as well as accelerated focus on understanding risk factors, targeting prevention strategies, and reducing disparities. The persistence of higher CP prevalence among NHB compared with NHW children over time warrants further investigation.

It is challenging to interpret the decrease in CP prevalence among NHW children born at 1500 g, as the numbers are small and examination of more refined birth weight and gestational age groups was not feasible. More updated information will allow us to determine if this decreasing trend in CP prevalence continues.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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ABBREVIATIONS

ADDM	Autism and Developmental Disabilities Monitoring
ASD	autism spectrum disorder
CI	confidence interval
CP	cerebral palsy
DD	developmental disability
HL	hearing loss
ID	intellectual disability
MADDSP	Metropolitan Atlanta Developmental Disabilities Surveillance Program
MLBW	moderately low birth weight
NBW	normal birth weight
NHB	non-Hispanic black
NHW	non-Hispanic white
PR	prevalence ratio
VI	vision impairment
VLBW	very low birth weight
VPT	very preterm

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WHAT'S KNOWN ON THIS SUBJECT: Outside the United States, decreases in neonatal mortality have not resulted in increases in cerebral palsy prevalence overall, yet varied trends by birth characteristics have been observed. Comparable population-based US data are limited, particularly to examine trends by race/ethnicity.

WHAT THIS STUDY ADDS: Birth prevalence of spastic cerebral palsy was stable from 1985 to 2002. Whereas no significant trends were observed by birth weight or gestational age overall, different patterns emerged within racial/ethnic subgroups. Racial disparities persisted over time, particularly among children born at term or with normal birth weight.

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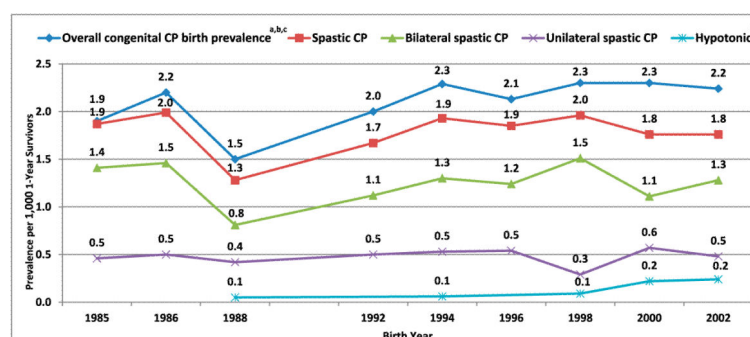


FIGURE 1. Trends in birth prevalence of congenital CP per 1000 1-year survivors by subtype, birth years 1985 to 2002. ^a $P < .05$ for trend. ^bIncludes CP cases with ataxic, dyskinetic, mixed, and CP not otherwise specified. ^cTrend not significant when hypotonic CP cases are excluded.

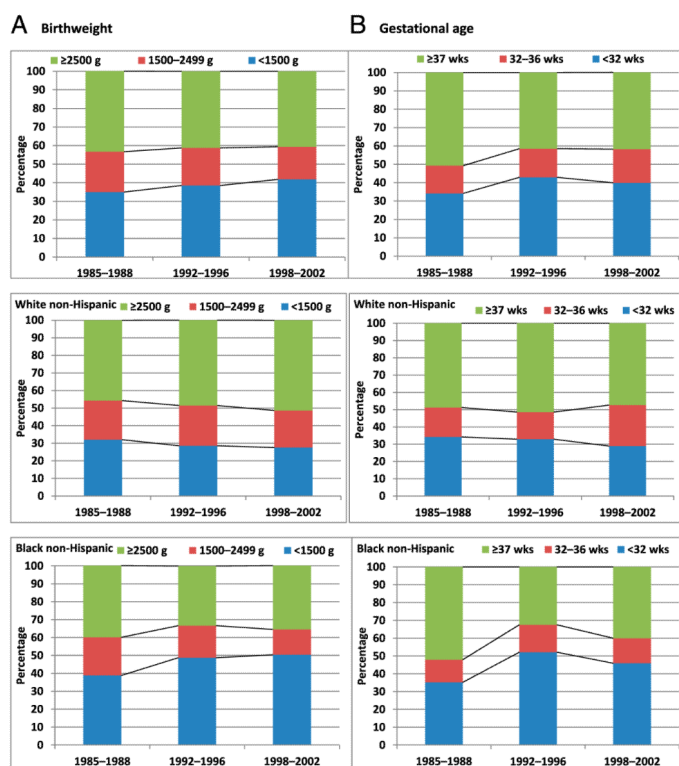


FIGURE 2. Distribution of birth weight and gestational age among children with congenital spastic CP overall and by race/ethnicity, birth years 1985 to 2002. A, Birth weight. B, Gestational age.

Trends in Birth Prevalence Per 1000 1-y Survivors and Characteristics of Children With Congenital Spastic CP ($n = 643$), Birth Years 1985 to 2002^a

TABLE 1

Characteristic	1985	1986	1988	1992	1994	1996	1998	2000	2002	1985 to 2002 % Prevalence	Average annual change, 1985 to 2002, % (95% CI)
Overall	1.86	1.98	1.27	1.66	1.92	1.85	1.96	1.75	1.76	100	1.78
Male	2.17	1.97	1.19	1.97	1.99	1.94	2.07	1.95	1.68	53.8	1.88
Female	1.53	1.98	1.36	1.35	1.86	1.75	1.84	1.55	1.83	46.2	1.68
NHW	1.65	1.49	1.02	1.25	1.17	1.21	1.32	1.28	1.34	35.3	1.30
NHB	2.29	2.89	1.58	2.21	2.71	2.44	2.58	2.22	2.34	51.9	2.36
Hispanic	NR	NR	NR	NR	2.11	2.18	1.55	1.59	1.27	7.6	1.62
Co-occurring developmental disabilities											
ID	0.88	1.11	0.75	0.95	1.09	1.06	0.83	0.83	0.82	51.5	0.92
Mild (IQ 50 to 70)	0.16	0.31	0.19	0.26	0.33	0.32	0.22	0.20	0.36	29.0	0.27
Moderate to profound (IQ <50)	0.72	0.74	0.50	0.58	0.63	0.64	0.49	0.52	0.36	61.3	0.56
VI	0.26	0.28	0.30	0.24	0.33	0.30	0.31	0.36	0.26	16.6	0.30
HL	NR	NR	NR	NR	NR	NR	0.16	NR	0.10	4.5	0.08
ASD	b	b	NR	0.18	0.13	0.20	0.18	NR	0.16	6.2	0.13
											-0.02 (-0.85 to 5.39) ^c

^a Because of small sample sizes, data for non-Hispanic American Indian/Alaska Native and non-Hispanic Asian/Pacific Islander are not shown. NR, not reported due to small sample sizes.

^b ASD surveillance for children born in 1985 and 1986 was not conducted.

^c Overall prevalence estimates (1985-2002) and trend analyses for surveillance years with $n < 5$ children include all years' data and reflect weighted prevalence with the subsequent surveillance year(s).

TABLE 2

Trends in Birth Prevalence (Prev) of Congenital Spastic CP Per 1000 1-y Survivors by Birth Weight and Gestational Age, Birth Years 1985 to 2002^a

Characteristic	1985		1986		1988		1992		1994		1996		1998		2000		2002		PR (95% CI) ^a	Average Annual Change, % (95% CI)
	n	Prev	n	Prev	n	Prev	n	Prev	n	Prev	n	Prev	n	Prev	n	Prev	n	Prev		
Birth weight overall, g	57	1.87	64	1.99	45	1.25	62	1.6	76	1.9	75	1.9	87	2.0	87	1.8	87	1.7		0.28 (−1.24 to 1.82)
<1500	21	65.63	23	65.71	14	37.74	22	44.4	32	66.4	28	54.8	44	66.4	37	53.7	28	40.8	68.0 (57.4 to 80.6)	−0.73 (−3.2 to 1.82)
1500 to 2499	15	8.16	14	7.09	7	3.10	18	7.1	14	5.5	11	4.1	13	4.4	11	3.3	22	6.2	6.6 (5.3 to 8.1)	−2.02 (−5.56 to 1.66)
2500	21	0.74	27	0.91	24	0.72	22	0.6	30	0.8	36	1.0	30	0.7	39	0.9	37	0.8	Referent	0.46 (−1.03 to 1.98)
Gestational age overall, wks	55	1.88	57	1.84	40	1.23	62	1.65	76	1.94	74	1.83	87	1.96	87	1.76	87	1.74		0.46 (−1.01 to 1.95)
<32	21	47.95	18	40.91	13	29.68	26	39.8	35	56.3	30	47.7	41	54.7	37	46.1	26	34.6	51.1 (43.1 to 60.6)	0.34 (−1.03 to 1.98)
32 to 36	12	4.33	9	3.03	2	0.58	12	3.1	13	3.5	8	2.1	15	3.5	14	3.0	19	3.8	3.4 (2.8 to 4.3)	1.44 (−3.26 to 6.37)
37	22	0.85	30	1.13	25	0.87	24	0.7	28	0.8	36	1.0	31	0.8	36	0.8	42	0.9	Referent	−0.34 (−1.93 to 1.28)

^aNormal birth weight (≥2500 g) and term gestational age (≥37 wks) served as referent groups for their respective analyses.

TABLE 3

Trends and Characteristics in Birth Prevalence (Prev) of Congenital Spastic CP Per 1000 1-y Survivors by Birth Weight, Gestational Age, and Race/ Ethnicity, Birth Years 1985 to 2002

Characteristic	1985 to 1988		1992 to 1996		1998 to 2002		Average Annual Prevalence, 1985 to 2002	NHB: NHW PR (95% CI) ^a	P, Cochran– Armitage Trend Test
	n	Prev	n	Prev	n	Prev			
Birthweight overall									
NHW	81	1.4	70	1.2	76	1.3	1.3	—	0.742
NHB	80	2.2	117	2.4	135	2.4	2.4	1.8 (1.5 to 2.1)	0.664
<1500 g									
NHW	26	69.0	20	48.7	21	39.3	50.7	—	0.048 ^b
NHB	31	48.5	57	57.6	68	53.2	53.7	1.06 (0.8 to 1.4)	0.776
1500 to 2499 g									
NHW	18	6.9	16	6.0	16	5.2	6.0	—	0.422
NHB	17	5.2	21	4.7	19	3.7	4.4	0.7 (0.5 to 1.1)	0.280
2500 g									
NHW	37	0.7	34	0.6	39	0.7	0.7	—	0.737
NHB	32	1.0	39	0.9	48	1.0	1.0	1.4 (1.1 to 1.9)	0.889
Gestational age overall									
NHW	76	1.4	70	1.3	76	1.4	1.4	—	0.922
NHB	71	2.1	117	2.8	135	2.7	2.6	1.9 (1.6 to 2.3)	0.156
<32 wks									
NHW	26	55.7	23	44.7	22	36.1	44.6	—	0.125
NHB	25	30.5	61	47.5	62	42.8	41.7	0.9 (0.7 to 1.2)	0.245
32 to 36 wks									
NHW	13	3.1	11	2.5	18	3.4	3.1	—	0.762
NHB	9	1.9	18	3.0	19	2.9	2.7	0.9 (0.6 to 1.3)	0.346
37 wks									
NHW	37	0.7	36	0.7	36	0.7	0.7	—	0.812
NHB	37	1.3	38	0.9	54	1.1	1.1	1.6 (1.2 to 2.0)	0.493

^a NHW children served as the referent group.

^b Significant at $P < .05$.