CDC
CENTERS FOR DISEASE CONTROL AND PREVENTION

April 19, 1996 / Vol. 45 / No. SS-2

MMWR™

MORBIDITY AND MORTALITY WEEKLY REPORT

CDC Surveillance Summaries

Prevalence of Selected Developmental Disabilities in Children 3–10 Years of Age: the Metropolitan Atlanta Developmental Disabilities Surveillance Program, 1991

Prevalence of Spina Bifida at Birth — United States, 1983–1990: a Comparison of Two Surveillance Systems

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Centers for Disease Control
and Prevention (CDC)
Atlanta, Georgia 30333

The *MMWR* series of publications is published by the Epidemiology Program Office, Centers for Disease Control and Prevention (CDC), Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA 30333.

SUGGESTED CITATION

General: Centers for Disease Control and Prevention. *CDC Surveillance Summaries*, April 19, 1996. MMWR 1996;45(No. SS-2).

Specific: [Author(s)]. [Title of particular article]. In: *CDC Surveillance Summaries*, April 19, 1996. MMWR 1996;45(No. SS-2):[inclusive page numbers].

Copies can be purchased from Superintendent of Documents, U.S. Government Printing Office, Washington, DC 20402-9325. Telephone: (202) 783-3238.

Contents

Reports Published in *CDC Surveillance Summaries* Since January 1, 1985

Subject	Responsible CIO/Agency*	Most Recent Report
Abortion	NCCDPHP	1995; Vol. 44, No. SS-2
AIDS/HIV		
Distribution by Racial/Ethnic Group	NCID	1988; Vol. 37, No. SS-3
Among Black & Hispanic Children & Women of Childbearing Age	NCEHIC	1990; Vol. 39, No. SS-3
Behavioral Risk Factors	NCCDPHP	1991; Vol. 40, No. SS-4
Birth Defects		
B.D. Monitoring Program (see also Malformations)	NCEH	1993; Vol. 42, No. SS-1
Contribution of B.D. to Infant Mortality Among Minority Groups	NCEHIC	1990; Vol. 39, No. SS-3
Breast & Cervical Cancer	NCCDPHP	1992; Vol. 41, No. SS-2
Campylobacter	NCID	1988; Vol. 37, No. SS-2
Chancroid	NCPS	1992; Vol. 41, No. SS-3
Chlamydia	NCPS	1993; Vol. 42, No. SS-3
Cholera	NCID	1992; Vol. 41, No. SS-1
Congenital Malformations, Minority Groups	NCEHIC	1988; Vol. 37, No. SS-3
Contraception Practices	NCCDPHP	1992; Vol. 41, No. SS-4
Cytomegalovirus Disease, Congenital	NCID	1992; Vol. 41, No. SS-2
Dengue	NCID	1994; Vol. 43, No. SS-2
Dental Caries & Periodontal Disease Among Mexican-American Children	NCPS	1988; Vol. 37, No. SS-3
Developmental Disabilities	NCEH	1996; Vol. 45, No. SS-2
Diabetes Mellitus	NCCDPHP	1993; Vol. 42, No. SS-2
Dracunculiasis	NCID	1992; Vol. 41, No. SS-1
Ectopic Pregnancy	NCCDPHP	1993; Vol. 42, No. SS-6
Elderly, Hospitalizations Among	NCCDPHP	1991; Vol. 40, No. SS-1
Endometrial & Ovarian Cancers	EPO, NCCDPHP	1986; Vol. 35, No. 2SS
Escherichia coli O157	NCID	1991; Vol. 40, No. SS-1
Evacuation Camps	EPO	1992; Vol. 41, No. SS-4
Family Planning Services at Title X Clinics	NCCDPHP	1995; Vol. 44, No. SS-2
Foodborne Disease	NCID	1990; Vol. 39, No. SS-1
Gonorrhea & Syphilis, Teenagers	NCPS	1993; Vol. 42, No. SS-3
Hazardous Substances Emergency Events	ATSDR	1994; Vol. 43, No. SS-2
Health Surveillance Systems	IHPO	1992; Vol. 41, No. SS-4
Hepatitis	NCID	1985; Vol. 34, No. 1SS
Homicide	NCEHIC	1992; Vol. 41, No. SS-3
Homicides, Black Males	NCEHIC	1988; Vol. 37, No. SS-1
Hysterectomy	NCCDPHP	1986; Vol. 35, No. 1SS
Infant Mortality (see also National Infant Mortality; Birth Defects; Postneonatal Mortality)	NCEHIC	1990; Vol. 39, No. SS-3
Influenza	NCID	1993; Vol. 42, No. SS-1
Injury		
Death Rates, Blacks & Whites	NCEHIC	1988; Vol. 37, No. SS-3
Drownings	NCEHIC	1988; Vol. 37, No. SS-1
Falls, Deaths	NCEHIC	1988; Vol. 37, No. SS-1
Firearm-Related Deaths, Unintentional	NCEHIC	1988; Vol. 37, No. SS-1

*Abbreviations

ATSDR	Agency for Toxic Substances and Disease Registry
CIO	Centers/Institute/Offices
EPO	Epidemiology Program Office
IHPO	International Health Program Office
NCCDPHP	National Center for Chronic Disease Prevention and Health Promotion
NCEH	National Center for Environmental Health
NCEHIC	National Center for Environmental Health and Injury Control
NCID	National Center for Infectious Diseases
NCIPC	National Center for Injury Prevention and Control
NCPS	National Center for Prevention Services
NIOSH	National Institute for Occupational Safety and Health
NIP	National Immunization Program

Reports Published in *CDC Surveillance Summaries* Since January 1, 1985 — Continued

Subject	Responsible CIO/Agency*	Most Recent Report
Head & Neck	NCIPC	1993; Vol. 42, No. SS-5
In Developing Countries	NCEHIC	1992; Vol. 41, No. SS-1
In the Home, Persons <15 Years of Age	NCEHIC	1988; Vol. 37, No. SS-1
Motor Vehicle-Related Deaths	NCEHIC	1988; Vol. 37, No. SS-1
Objectives of Injury Control, State & Local	NCEHIC	1988; Vol. 37, No. SS-1
Objectives of Injury Control, National	NCEHIC	1988; Vol. 37, No. SS-1
Residential Fires, Deaths	NCEHIC	1988; Vol. 37, No. SS-1
Tap Water Scalds	NCEHIC	1988; Vol. 37, No. SS-1
Lead Poisoning, Childhood	NCEHIC	1990; Vol. 39, No. SS-4
Low Birth Weight	NCCDPHP	1990; Vol. 39, No. SS-3
Malaria	NCID	1995; Vol. 44, No. SS-5
Maternal Mortality	NCCDPHP	1991; Vol. 40, No. SS-2
Measles	NCPS	1992; Vol. 41, No. SS-6
Meningococcal Disease	NCID	1993; Vol. 42, No. SS-2
Mining	NIOSH	1986; Vol. 35, No. 2SS
Mumps	NIP	1995; Vol. 44, No. SS-3
National Infant Mortality (see also Infant Mortality; Birth Defects)	NCCDPHP	1989; Vol. 38, No. SS-3
Neisseria gonorrhoeae, Antimicrobial Resistance in	NCPS	1993; Vol. 42, No. SS-3
Neural Tube Defects	NCEH	1995; Vol. 44, No. SS-4
Nosocomial Infection	NCID	1986; Vol. 35, No. 1SS
Occupational Injuries/Disease		
Asthma	NIOSH	1994; Vol. 43, No. SS-1
Hazards, Occupational	NIOSH	1985; Vol. 34, No. 2SS
In Meatpacking Industry	NIOSH	1985; Vol. 34, No. 1SS
Silicosis	NIOSH	1993; Vol. 42, No. SS-5
State Activities	NIOSH	1987; Vol. 36, No. SS-2
Parasites, Intestinal	NCID	1991; Vol. 40, No. SS-4
Pediatric Nutrition	NCCDPHP	1992; Vol. 41, No. SS-7
Pertussis	NCPS	1992; Vol. 41, No. SS-8
Plague	NCID	1985; Vol. 34, No. 2SS
Plague, American Indians	NCID	1988; Vol. 37, No. SS-3
Poliomyelitis	NCPS	1992; Vol. 41, No. SS-1
Postneonatal Mortality	NCCDPHP	1991; Vol. 40, No. SS-2
Pregnancy Nutrition	NCCDPHP	1992; Vol. 41, No. SS-7
Pregnancy, Teenage	NCCDPHP	1993; Vol. 42, No. SS-6
Rabies	NCID	1989; Vol. 38, No. SS-1
Racial/Ethnic Minority Groups	Various	1990; Vol. 39, No. SS-3
Respiratory Disease	NCEHIC	1992; Vol. 41, No. SS-4
Rotavirus	NCID	1992; Vol. 41, No. SS-3
Salmonella	NCID	1988; Vol. 37, No. SS-2
Sexually Transmitted Diseases in Italy	NCPS	1992; Vol. 41, No. SS-1
Smoking	NCCDPHP	1990; Vol. 39, No. SS-3
Smoking-Attributable Mortality	NCCDPHP	1994; Vol. 43, No. SS-1
Tobacco Control Laws, State	NCCDPHP	1995; Vol. 44, No. SS-6
Tobacco-Use Behaviors	NCCDPHP	1994; Vol. 43, No. SS-3
Spina Bifida	NCEH	1996; Vol. 45, No. SS-2
Streptococcal Disease (Group B)	NCID	1992; Vol. 41, No. SS-6
Sudden Unexplained Death Syndrome Among Southeast Asian Refugees	NCEHIC, NCPS	1987; Vol. 36, No. 1SS
Suicides, Persons 15–24 Years of Age	NCEHIC	1988; Vol. 37, No. SS-1
Syphilis, Congenital	NCPS	1993; Vol. 42, No. SS-6
Syphilis, Primary & Secondary	NCPS	1993; Vol. 42, No. SS-3
Tetanus	NCPS	1992; Vol. 41, No. SS-8
Trichinosis	NCID	1991; Vol. 40, No. SS-3
Tuberculosis	NCPS	1991; Vol. 40, No. SS-3
Waterborne Disease Outbreaks	NCID	1996; Vol. 45, No. SS-1
Years of Potential Life Lost	EPO	1992; Vol. 41, No. SS-6
Youth Risk Behaviors	NCCDPHP	1995; Vol. 44, No. SS-1

Prevalence of Selected Developmental Disabilities in Children 3–10 Years of Age: the Metropolitan Atlanta Developmental Disabilities Surveillance Program, 1991

Coleen A. Boyle, Ph.D.
Marshalyn Yeargin-Allsopp, M.D.
Nancy S. Doernberg
Patricia Holmgreen, M.S.
Catherine C. Murphy, M.P.H.
Diana E. Schendel, Ph.D.

Division of Birth Defects and Developmental Disabilities
National Center for Environmental Health

Abstract

Problem/Condition: Serious developmental disabilities affect approximately 2% of school-age children and are lifelong conditions that incur substantial financial and societal costs.

Reporting Period: January 1991–December 1991.

Description of System: The Metropolitan Atlanta Developmental Disabilities Surveillance Program (MADDSP) monitors the prevalence of four serious developmental disabilities—mental retardation, cerebral palsy, vision impairment, and hearing impairment—among children 3–10 years of age in the five-county metropolitan-Atlanta area. Children who have at least one of the four developmental disabilities are ascertained through annual review of records at schools, hospitals, and other sources.

Results and Interpretation: During 1991, rates for mental retardation varied by age, race, and sex; rates ranged from 5.2 per 1,000 children to 16.6 per 1,000 children. Regardless of the absolute rate of mental retardation in each of the age-, race-, and sex-specific categories, severe mental retardation (i.e., an intelligence quotient of <50) accounted for one third of all cases. The overall crude rate of cerebral palsy was 2.4 per 1,000 children; however, the rate was higher among black children (3.1 per 1,000 children) than among white children (2.0 per 1,000 children). The rate of moderate to severe hearing impairment was 1.1 per 1,000 children, and the rate of vision impairment was 0.8 per 1,000 children. Rates of hearing impairment were higher among black males than among children in the other race and sex groups, whereas rates for vision impairment varied only slightly between these groups. The rates of the developmental disabilities were not adjusted for possible confounding factors (e.g., maternal education, family income, and various medical conditions). Consequently, the variation in rates may reflect social or other characteristics unique to the study population.

Actions Taken: MADDSP data will be used to direct early childhood intervention efforts to reduce the prevalence of these four developmental disabilities. MADDSP data also are being used to measure progress toward the year 2000 national objectives for the prevention of serious mental retardation.

INTRODUCTION

Since 1968, CDC has conducted surveillance for birth defects (i.e., structural malformations and genetic diseases) in the five-county metropolitan-Atlanta area (*1*). However, those birth defects that generally are evident at birth (e.g., cleft palate and spina bifida) represent only part of a spectrum of developmental problems that appear during childhood. Other conditions (e.g., mental retardation, autism, and cerebral palsy) usually are manifested after infancy and may be sufficiently severe to require specialized medical and educational services for many years (*2*). Such conditions are referred to as developmental disabilities.

To address the problem of developmental disabilities among children, CDC and the Georgia Department of Human Resources initiated the Metropolitan Atlanta Developmental Disabilities Study in 1984. For this study, which was conducted in Atlanta during 1985–1987, investigators devised methods for determining the prevalence of mental retardation, epilepsy, cerebral palsy, blindness, and hearing impairment among children 10 years of age (*3*). Children who had these conditions were identified by searching record systems of sources that were likely to contain information relating to the evaluation or treatment of children who have developmental disabilities (e.g., schools, hospitals, and state programs for persons who have developmental disabilites). The majority of the children were identified through special-education departments within the Atlanta-area public school systems. The success of this study prompted CDC to establish the Metropolitan Atlanta Developmental Disabilities Surveillance Program (MADDSP) in 1991, an ongoing system for monitoring the occurrence of selected developmental disabilities.

The two principal objectives of MADDSP are to a) provide regular and systematic monitoring of prevalence rates for selected developmental disabilities according to various demographic characteristics of children and their mothers (which is the focus of this report) and b) provide a framework for initiating special studies of children who have the selected developmental disabilities by establishing a population-based case series of such children. In the future, MADDSP data will be used to measure progress toward the year 2000 national objectives (*4*) for the prevention of serious mental retardation.

METHODS

MADDSP was established to ascertain all children who have one or more of four developmental disabilities—mental retardation, cerebral palsy, hearing impairment, and vision impairment—in the five-county (i.e., Clayton, Cobb, DeKalb, Fulton, and Gwinnett) metropolitan-Atlanta area. In 1990, this area had an estimated population of 2.2 million persons, 39,000 births, and 249,500 children 3–10 years of age. This area has an active birth defects surveillance program—the Metropolitan Atlanta Congenital Defects Program, which is operated by CDC. Consequently, additional medical data can be obtained by linking the children identified through MADDSP to the birth defects registry.

As a consequence of the Individuals with Disabilities Education Act* (*5*), most children eligible for MADDSP are either enrolled in special education programs at nine public school systems serving the study area or enrolled in other Georgia Department of Education programs for children who have developmental disabilities

*Public Law 94-142.

(e.g., state schools for children who are hearing or vision impaired and regional psychoeducational centers) (*5*). Additional sources used to identify children who are eligible for inclusion in MADDSP include a) Georgia Department of Human Resources facilities that provide services for children who have developmental disabilities and b) two metropolitan-Atlanta–area pediatric-care hospitals, one public hospital, and the clinics associated with these facilities.

Source records (e.g., medical and school records) are reviewed annually for children who are potentially eligible for inclusion in the surveillance program. Because most cases of these developmental disabilities are considered lifelong conditions (*3*), children who have been included in MADDSP in a previous year are included in current year prevalence rates if they still meet age and residence requirements. A child's record is re-examined (on the basis of a time schedule that considers the child's age and underlying diagnosis) to verify and update the child's diagnostic information.

Case Definition

For the purposes of MADDSP, a case is defined as a child a) who is 3–10 years of age at any time during the study year of interest; b) whose parent(s) or legal guardian(s) reside in the five-county metropolitan-Atlanta area during the study year of interest; and c) who has one or more of the four developmental disabilities. The age range of 3–10 years was chosen because the lower boundary (i.e., 3 years of age) corresponds to the beginning of the age span covered by Part B of the Individuals with Disabilities Education Act (*5*), and the upper boundary (i.e., 10 years of age) is the age at which most children served under the Act should have entered special education programs.

Developmental Disability Definitions

- *Mental retardation* is (for the purposes of this surveillance program) a condition marked by an intelligence quotient (IQ) of ≤70 on the most recently administered standardized psychometric test. In the absence of an IQ-test score, a written statement by a psychometrist that a child's intellectual functioning falls within the range for mental retardation is acceptable. The severity of mental retardation is defined according to the following International Classification of Disease, Ninth Edition, Clinical Modification (ICD-9–CM) categories: mild (an IQ of 50–70), moderate (an IQ of 35–49), severe (an IQ of 20–34), and profound (an IQ of <20) (*6*).
- *Cerebral palsy* is a group of nonprogressive, but often changing, motor impairment syndromes secondary to lesions or anomalies of the brain arising at any time during brain development (*7*). For this surveillance system, the definition includes postnatally acquired cerebral palsy diagnosed before 11 years of age. Children are included in the surveillance system if they have been a) diagnosed as having cerebral palsy by a qualified physician or b) identified by other qualified professionals (e.g., physical and occupational therapists) as having this disability on the basis of physical findings noted in source records. Cerebral palsy is categorized as "disabling" or "nondisabling" on the basis of the degree of the affected child's ambulation and use of assistive devices (*8*). Children who have nondisabling cerebral palsy are fully ambulatory and do not require the use of assistive devices, whereas children who have disabling cerebral palsy require the use of assistive devices for ambulation (either intermittently or at all times).

- *Vision impairment* is a measured visual acuity of 20/70 or worse, with correction, in the better eye. In the absence of a measured visual acuity, a child is considered visually impaired if a source record includes a) a functional description, by an eye specialist, of visual acuity of 20/70 or worse or b) a statement by an eye specialist that the child has low vision or blindness. Severity of visual impairment is defined using the following ICD-9–CM categories: moderate visual impairment (corrected visual acuity of 20/70–20/160), severe visual impairment (corrected visual acuity of 20/200–20/400), and profound, near total, and total visual impairment, which were grouped together (corrected visual acuity of 20/500 or worse) (*6*).
- *Hearing impairment* is a measured, bilateral, pure-tone hearing loss at frequencies of 500, 1,000, and 2,000 hertz averaging 40 decibels (dBs) or more, unaided, in the better ear. In the absence of a measured, bilateral hearing loss, children meet the case definition if their source records include a description, by a licensed or certified audiologist or qualified physician, of a hearing loss of 40 dBs or more in the better ear. For this program, severity was defined on the basis of the following hearing impairment levels (measured in the better ear): moderate (a hearing loss of 40–64 dBs), severe (a hearing loss of 65–84 dBs), and profound (a hearing loss of ≥85 dBs) (*9*).

Data Collection

In addition to a standard list of demographic variables and identifying information, MADDSP collects both the earliest and most recent evaluation data relevant to the child's specific disabilities. For example, for children whose disability meets the case definition for mental retardation, scores on both the earliest and most recent tests of cognitive functioning are recorded, whereas for those whose disability meets the definition for hearing impairment, both the earliest and most recent hearing level in each ear and the type of hearing loss are recorded. Information concerning associated medical conditions (e.g., chromosomal defects or unintentional injuries) that may be associated with the etiology of the developmental disability is also collected. Data are collected for race because of previously reported race-specific differences in the prevalences of these disabilities (*10*). Race is obtained from source records.

Analysis

The 1990 U.S. Census data were used to calculate point-prevalence rates for 1991 for children 2–9 years of age (who were 3–10 years of age in 1991) in the five-county metropolitan-Atlanta area. Both the overall rate of each developmental disability and rates by level of severity were calculated. Rates were calculated by race (i.e., white and black), sex, and age. Other race groups were not included in this report because of the limited number of reported cases among other races. Age represents the age the child reached in 1991.

Point-prevalence rates were calculated rather than birth-cohort prevalence rates (or ratios) because they represent the burden of disease in the population at one particular time (*11*). Moreover, these rates are the more appropriate measure for the data collected because they reflect all cases rather than the subset involving children who

were born in the study area. Children were counted in more than one rate if they had more than one of the four disabilities.

RESULTS

In metropolitan Atlanta during 1991, a total of 2,692 children were identified as having one or more of the four developmental disabilities. Eighteen percent of these children had more than one developmental disability, representing a total of 3,295 disabilities.

Source of Ascertainment

With the exception of children who had cerebral palsy, >90% of children who had developmental disabilities were identified through one of the Georgia Department of Education sources. Among children identified as having cerebral palsy, 17% were identified from hospital records, and 7% were identified from a Department of Human Resources' source.

Mental Retardation

The overall prevalence of mental retardation was 8.7 per 1,000 children 3–10 years of age, and approximately two thirds of cases were of mild severity (Table 1). The prevalence of mental retardation varied with age, increasing from 5.2 per 1,000 children 3–4 years of age to 12.3 per 1,000 children 9–10 years of age. This increase in reported prevalence with advancing age occurred among children who had mild and moderate mental retardation but not among those who had severe or profound mental retardation; for these categories, the rate was relatively constant with advancing age.

Rates of mental retardation for black males were 3.1, 2.4, and 1.7 times higher than rates for white females, white males, and black females, respectively (Table 2). This pattern was found within each level of mental retardation, with the exception of profound mental retardation. In each of the sex-race–specific groups, regardless of the absolute rate of mental retardation, two thirds of the children were in the mild range.

Cerebral Palsy

The overall rate of cerebral palsy was 2.4 per 1,000 children 3–10 years of age (Table 3). An age-specific pattern was evident—the youngest children had the lowest prevalence rates, and children in the middle age range (i.e., 5–6 years of age) had the highest rates; this pattern was found for disabling, but not for nondisabling, cerebral palsy.

Rates of nondisabling cerebral palsy were higher for black children than for white children (Table 4). Although a similar disparity between races occurred among children with disabling cerebral palsy, the magnitude of the difference was considerably less. Black females and black males had similar rates of both disabling and nondisabling cerebral palsy. Compared with white males, white females had lower rates of nondisabling cerebral palsy but similar rates of disabling cerebral palsy.

Eleven percent of the children who had cerebral palsy (N=58) had a postnatal cause for their disability, resulting in a prevalence of 0.2 per 1,000 children for acquired

TABLE 1. Age-specific prevalence of mental retardation among children 3–10 years of age, by level of mental retardation — Metropolitan Atlanta Developmental Disabilities Surveillance Program, 1991

	Level of mental retardation*									
	Mild		Moderate		Severe		Profound		Total†	
Age (yrs)	No.	Rate§	No.	Rate§	No.	Rate§	No.	Rate§	No.	Rate§
3–4	244	3.7	36	0.5	21	0.3	30	0.5	343	5.2
5–6	295	4.7	89	1.4	65	1.0	31	0.5	483	7.6
7–8	409	6.6	110	1.8	71	1.1	28	0.5	620	10.0
9–10	499	8.2	166	2.7	59	1.0	23	0.4	747	12.3
Total	**1,447**	**5.7**	**401**	**1.6**	**216**	**0.9**	**112**	**0.4**	**2,193**	**8.7**

* Mild=intelligence quotient (IQ) of 50–70; moderate=IQ of 35–49; severe=IQ of 20–34; and profound=IQ of <20.
† Includes 17 children whose level of mental retardation was unknown.
§ Rate per 1,000 children.

TABLE 2. Prevalence of mental retardation among children 3–10 years of age, by level of mental retardation, sex, and race — Metropolitan Atlanta Developmental Disabilities Surveillance Program, 1991

		Level of mental retardation*									
		Mild		Moderate		Severe		Profound		Total†	
Sex	Race	No.	Rate§	No.	Rate§	No.	Rate§	No.	Rate§	No.	Rate§
Male	White	330	4.4	94	1.3	51	0.7	22	0.3	503	6.8
	Black	538	11.3	149	3.1	78	1.6	24	0.5	791	16.6
	Total	**892**	**6.9**	**255**	**2.0**	**133**	**1.0**	**48**	**0.4**	**1,335**	**10.4**
Female	White	255	3.6	58	0.8	33	0.5	32	0.5	384	5.4
	Black	292	6.3	81	1.7	48	1.0	31	0.7	454	9.8
	Total	**557**	**4.5**	**146**	**1.2**	**83**	**0.7**	**64**	**0.5**	**858**	**6.9**
Total	White	586	4.0	152	1.0	84	0.6	54	0.4	887	6.1
	Black	829	8.8	230	2.4	126	1.3	55	0.6	1,245	13.2
	Total¶	**1,447**	**5.7**	**401**	**1.6**	**216**	**0.9**	**112**	**0.4**	**2,193**	**8.7**

* Mild=intelligence quotient (IQ) of 50–70; moderate=IQ of 35–49; severe=IQ of 20–34; and profound=IQ of <20.
† Includes 17 children whose level of mental retardation was unknown.
§ Rate per 1,000 children.
¶ Includes 61 children of races other than white or black.

TABLE 3. Age-specific prevalence of cerebral palsy (CP) among children 3–10 years of age, by severity — Metropolitan Atlanta Developmental Disabilities Surveillance Program, 1991

	Severity of CP					
	Nondisabling CP		Disabling CP*		Total CP†	
Age (yrs)	No.	Rate§	No.	Rate§	No.	Rate§
3–4	48	0.7	63	1.0	135	2.0
5–6	61	1.0	98	1.6	177	2.8
7–8	58	0.9	94	1.5	159	2.6
9–10	53	0.9	78	1.3	137	2.3
Total	**220**	**0.9**	**333**	**1.3**	**599**	**2.4**

*Children in this category require the use of assisted devices for ambulation either intermittently or at all times.
†Includes 46 children who had CP of unknown severity.
§Rate per 1,000 children.

TABLE 4. Prevalence of cerebral palsy (CP) among children 3–10 years of age, by severity, sex, and race — Metropolitan Atlanta Developmental Disabilities Surveillance Program, 1991

		Severity of CP					
		Nondisabling CP		Disabling CP*		Total CP†	
Sex	Race	No.	Rate§	No.	Rate§	No.	Rate§
Male	White	56	0.8	92	1.2	157	2.1
	Black	66	1.4	71	1.5	154	3.2
	Total	**129**	**1.0**	**169**	**1.3**	**322**	**2.5**
Female	White	32	0.5	89	1.3	130	1.8
	Black	55	1.2	70	1.5	138	3.0
	Total	**91**	**0.7**	**164**	**1.3**	**277**	**2.2**
Total	White	90	0.6	181	1.3	287	2.0
	Black	121	1.3	141	1.5	292	3.1
	Total¶	**220**	**0.9**	**333**	**1.3**	**599**	**2.4**

*Children in this category require the use of assisted devices for ambulation either intermittently or at all times.
†Includes 46 children who had CP of unknown severity.
§Rate per 1,000 children.
¶Includes 21 children of races other than white or black.

cerebral palsy and 2.2 per 1,000 children for congenital cerebral palsy. Eighty-three percent of all children who had cerebral palsy had spastic cerebral palsy, 2% had dyskinetic cerebral palsy, 1% had ataxic cerebral palsy, and 7% had other types of cerebral palsy. The type of cerebral palsy could not be determined from the available records for 7% of affected children.

Hearing Impairment

The overall prevalence of hearing impairment was 1.1 per 1,000 children 3–10 years of age, and the rate increased steadily with age (Table 5)—a trend that generally was found for all severity levels. Overall rates of hearing impairment were higher for black males than for other race and sex groups (Table 6).

The predominant type of hearing impairment was sensorineural (79%), followed by conductive hearing impairment (6%), and both sensorineural and conductive combined (3%). The type of hearing loss was unknown for 12% of children.

Vision Impairment

The overall prevalence rate for vision impairment was 0.8 per 1,000 children 3–10 years of age. The prevalence of vision impairment increased with age up to the age of 7 years and then leveled off (Table 7). This pattern was observed for all severity levels of vision impairment. The prevalence of vision impairment was similar among the various race- and sex-specific subgroups of children (Table 8).

Coexisting Developmental Disabilities

Seventy-three percent of children who had vision impairment and 66% of those who had cerebral palsy met the case definition for one of the other developmental disabilities included in MADDSP (primarily mental retardation and vision impairment for children who had cerebral palsy and mental retardation and cerebral palsy for children who had vision impairment) (Table 9). In contrast, only 22% of children who had mental retardation and 23% of children who had hearing impairment had coexisting

TABLE 5. Age-specific prevalence of hearing impairment among children 3–10 years of age, by level of impairment — Metropolitan Atlanta Developmental Disabilities Surveillance Program, 1991

	Level of hearing impairment*							
	Moderate		Severe		Profound		Total	
Age (yrs)	No.	Rate†	No.	Rate†	No.	Rate†	No.	Rate†
3–4	23	0.4	11	0.2	25	0.4	59	0.9
5–6	26	0.4	15	0.2	19	0.3	60	1.0
7–8	31	0.5	17	0.3	30	0.5	78	1.3
9–10	39	0.6	20	0.3	27	0.4	86	1.4
Total	**119**	**0.5**	**63**	**0.3**	**101**	**0.4**	**283**	**1.1**

* Moderate=children who had a hearing loss of 40–64 decibles (dBs); severe=children who had a hearing loss of 65–84 dBs; and profound=children who had a hearing loss of ≥85 dBs.
† Rate per 1,000 children.

TABLE 6. Prevalence of hearing impairment among children 3–10 years of age, by level of impairment, sex, and race — Metropolitan Atlanta Developmental Disabilities Surveillance Program, 1991

		Level of hearing impairment*							
		Moderate		Severe		Profound		Total	
Sex	Race	No.	Rate†	No.	Rate†	No.	Rate†	No.	Rate†
Male	White	32	0.4	20	0.3	27	3.6	79	1.1
	Black	32	0.7	16	0.3	26	5.5	74	1.6
	Total	**70**	**0.5**	**39**	**0.3**	**57**	**4.4**	**166**	**1.3**
Female	White	32	0.5	13	0.2	19	2.7	64	0.9
	Black	14	0.3	11	0.2	22	4.7	47	1.0
	Total	**49**	**0.4**	**24**	**0.2**	**44**	**3.6**	**117**	**0.9**
Total	White	64	0.4	33	0.2	46	3.2	143	1.0
	Black	46	0.5	27	0.3	48	5.1	121	1.3
	Total§	**119**	**0.5**	**63**	**0.3**	**101**	**4.0**	**283**	**1.1**

* Moderate=children who had a hearing loss of 40–64 decibles (dBs); severe=children who had a hearing loss of 65–84 dBs; and profound=children who had a hearing loss of ≥85 dBs.
† Rate per 1,000 children.
§ Includes 19 children in racial groups other than white or black.

TABLE 7. Age-specific prevalence of vision impairment among children 3–10 years of age, by level of impairment — Metropolitan Atlanta Developmental Disabilities Surveillance Program, 1991

	Level of vision impairment*							
	Moderate		Severe		Profound		Total†	
Age (yrs)	No.	Rate§	No.	Rate§	No.	Rate§	No.	Rate§
3–4	2	<0.1	8	0.1	11	0.2	29	0.4
5–6	5	0.1	22	0.4	14	0.2	52	0.8
7–8	20	0.3	16	0.3	23	0.4	69	1.1
9–10	13	0.2	22	0.4	16	0.3	59	1.0
Total	**40**	**0.2**	**68**	**0.3**	**64**	**0.3**	**209**	**0.8**

* Moderate=children who had visual acuity (VA) of 20/70–20/160; severe=children who had VA of 20/200–20/400; and profound=children who had VA of ≥20/500.
† Includes 37 children whose level of vision impairment was unknown.
§ Rate per 1,000 children.

TABLE 8. Prevalence of vision impairment among children 3–10 years of age, by level of impairment, sex, and race — Metropolitan Atlanta Developmental Disabilities Surveillance Program, 1991

		Level of vision impairment*							
		Moderate		Severe		Profound		Total†	
Sex	Race	No.	Rate§	No.	Rate§	No.	Rate§	No.	Rate§
Male	White	16	0.2	26	0.4	19	0.3	70	0.9
	Black	10	0.2	13	0.3	12	0.3	42	0.9
	Total	**26**	**0.2**	**42**	**0.3**	**32**	**0.3**	**117**	**0.9**
Female	White	7	0.1	16	0.2	17	0.2	51	0.7
	Black	5	0.1	10	0.2	15	0.3	39	0.8
	Total	**14**	**0.1**	**26**	**0.2**	**32**	**0.3**	**92**	**0.7**
Total	White	23	0.2	42	0.3	36	0.3	121	0.8
	Black	15	0.2	23	0.2	27	0.3	81	0.9
	Total¶	**40**	**0.2**	**68**	**0.3**	**64**	**0.3**	**209**	**0.8**

*Moderate=children who had visual acuity (VA) of 20/70–20/160; severe=children who had VA of 20/200–20/400; and profound=children who had VA of ≥20/500.
†Includes 37 children whose level of vision impairment was unknown.
§Rate per 1,000 children.
¶Includes seven vision-impaired children in racial groups other than white or black.

TABLE 9. Number and percentage of disabled children 3–10 years of age who had coexisting developmental disabilities, by type of disability — Metropolitan Atlanta Developmental Disabilities Surveillance Program, 1991

		Type of coexisting disability									
		Any coexisting developmental disability*		Mental retardation		Cerebral palsy		Hearing impairment		Vision impairment	
Type of disability	No. of children	No.	(%)	No.	(%)	No.	(%)	No.	(%)	No.	(%)
Mental retardation	2,193	473	(22)	—	—	381	(17)	58	(3)	145	(7)
Cerebral palsy	599	396	(66)	381	(64)	—	—	22	(4)	101	(17)
Hearing impairment	283	66	(23)	58	(20)	22	(8)	—	—	6	(2)
Vision impairment	209	152	(73)	145	(69)	101	(48)	6	(3)	—	—

*The numbers in this column are less than the sum of the four types of disabilities because some children had more than one additional disability.

conditions (primarily cerebral palsy for children who had mental retardation and mental retardation for children who had hearing impairment).

Age at First Diagnosis

Only 10% of children who had mental retardation, 35% of children who had cerebral palsy, 17% of those who had hearing impairment, and 17% of those who had vision impairment were first diagnosed before 2 years of age. Children who had more severe forms of a developmental disability generally were diagnosed at an earlier age. For example, 36% of children who had profound mental retardation were identified before the age of 2 years compared with only 7% of children who had mild mental retardation. However, by 5 years of age, 67% of children who had mental retardation, 87% of children who had cerebral palsy, 83% of children who had hearing impairment, and 70% of children who had vision impairment had been identified.

DISCUSSION

Developmental disabilities are lifelong conditions that result in substantial emotional, psychological, and financial costs to affected persons, their families, and society. The direct and indirect lifetime costs for cerebral palsy were recently estimated to be $445,000 (in 1988 dollars) per affected person (*12*). MADDSP data indicate that 1% of children 3–10 years of age in metropolitan Atlanta have a serious developmental disability and that 16% of these affected children have more than one developmental disability. Furthermore, although MADDSP includes most of the major developmental disabilities, some disabilities (e.g., autism and epilepsy) have not yet been incorporated into MADDSP. Other disabilities (e.g., learning and behavioral disabilities) are more difficult to identify and include in a surveillance system; the inclusion of such disabilities likely would substantially increase the prevalence of developmental disabilities.

The validity of the ascertainment methodology of MADDSP relies on the consequences of the Individuals with Disabilities Education Act (*5*). Because this law requires public schools to provide educational services to children who have developmental problems, public schools must identify and maintain information concerning such children. MADDSP has employed this requirement to assist in and enhance surveillance; however, nearly 20% of children who had cerebral palsy were identified from sources other than educational services.

Although MADDSP is an active surveillance system, its function is dependent on data collected and used for purposes other than public health surveillance. Consequently, some records do not contain the information necessary to determine a child's eligibility for MADDSP. Although few children were found to be ineligible for inclusion because of insufficient information, some eligible children may have been excluded from surveillance because they were not identified as having the disability or, even if they were identified previously, did not need continuing special care or services. These children are likely those with milder forms of the disabilities. School-age children are screened periodically for vision and hearing impairments, whereas children who have mental retardation or cerebral palsy are identified by the medical and educational systems because of their need for special care or because they cannot adapt or function successfully in their environment. Because not all school-age children are

systematically evaluated for cognitive and motor deficits, some biases may increase the likelihood of identifying children who have certain demographic or socioeconomic characteristics.

The specific findings from MADDSP are consistent with findings from previous studies and expand the knowledge of the epidemiology of developmental disabilities in a large U.S. population group. Specifically, MADDSP rates for each of the disabilities among children 10 years of age concur with those of a previous CDC study of children 10 years of age that involved a more detailed ascertainment procedure (Table 10) (*4*) and are within the ranges reported in other studies (*13–20*).

With the exception of vision impairment, the prevalence of developmental disabilities among children living in the metropolitan-Atlanta area varied substantially by age, race, and sex. The prevalence of each of the disabilities increased with age because many children are not identified as having disabilities until they attend school—a pattern that has been noted in previous studies (*13,14,17,18*). Rates for children 3–4 years of age were probably lower because 1991 was the first year in which public schools provided educational services to this age group. The unusual age pattern among children who had disabling cerebral palsy (i.e., an increase in prevalence up to 5 or 6 years of age and a decrease thereafter [which did not occur among children who had nondisabling cerebral palsy]) suggests an increase in mortality or out-migration among this group. Children are followed longitudinally in this surveillance program; therefore, future analyses may be able to address this observation directly.

The higher rates of mental retardation, cerebral palsy (especially nondisabling cerebral palsy), and hearing impairment for black children were also consistent with rates of previous studies (*10,21–25*). A substantial proportion of the difference in the reported mental retardation rates between black and white children likely reflects socioeconomic disparities (*26,27*); cerebral palsy and hearing impairment may also be associated with such disparities. Another factor contributing to higher rates of mental retardation among black children may be the use of standardized intelligence tests designed to test skills considered relevant to intelligence by the predominant culture (*28*).

These race-related differences suggest an important strategy for preventing mental retardation—ameliorating those aspects of the socioeconomic environment that negatively influence the cognitive ability of children. Future epidemiologic studies should be designed to further the understanding of specific aspects of the social

TABLE 10. Prevalence of selected developmental disabilities obtained from the Metropolitan Atlanta Developmental Disabilities Surveillance Program (MADDSP) and other previous studies, by type of disability

	MADDSP*†	Previous CDC study (*3*)	Other previous studies (*11–18*)
Type of disability	**Rate§**	**Rate§**	**Rate§**
Mental retardation	11.6	12.0	3.1–43.6
Cerebral palsy	2.3	2.3	2.0– 3.0
Hearing impairment	1.5	1.1	0.8– 2.0
Vision impairment	0.6	0.7	0.3– 0.6

*Limited to children 10 years of age.
†For comparability, only children who had corrected visual acuity of ≥20/200 were included in rate calculations.
§Rate per 1,000 children.

environment that may impair cognitive ability; such knowledge could then be used to target interventions to those children at greatest risk.

The slightly elevated rates of mental retardation and hearing impairment among boys—particularly among black children—have been reported previously (*17,10,25*). These discrepancies may result, in part, from both sex-linked genetic disorders and more frequent referral and testing of boys because of behavioral problems in school.

Although genetic, metabolic, and infectious factors are the cause of some cases of these developmental disabilities, the etiologies of most cases are undetermined. MADDSP enables ongoing monitoring of the occurrence of four common developmental disabilities in a community setting, which may lead to the identification of new risk factors for such disabilities. The demographic patterns described in this report may reflect social or other characteristics unique to the study population.

References

1. Public Health Service. Healthy people 2000: national health promotion and disease prevention objectives—full report, with commentary. Washington, DC: US Department of Health and Human Services, Public Health Service, 1991;DHHS publication no. (PHS)91-50212.
2. Crocker AC. The spectrum of medical care for developmental disabilities. In: Rubin IL, Crocker AC, eds. Developmental disabilities: delivery of medical care for children and adults. Philadelphia, PA: Lea & Febiger, 1989.
3. Yeargin-Allsopp M, Murphy CC, Oakley GP, et al. A multiple source method for studying the prevalence of developmental disabilities in children: the Metropolitan Atlanta Developmental Disabilities Study. Pediatrics 1992;89:624–30.
4. Edmonds LD, Layde PM, James LM, et al. Congenital malformations surveillance: two American systems. Int J Epidemiol 1981;10:247–52.
5. Individuals with Disabilities Education Act Admendments of 1991, PL 102-119. 20 USC 1400 et seq. Federal Register 1991;137(1901).
6. International classification of diseases, ninth edition. Manual of the international statistical classification of diseases, injuries, and causes of death. Vol. 1. Geneva: World Health Organization, 1977.
7. Mutch L, Alberman E, Hagberg B, Kodama K, Perat M. Cerebral palsy epidemiology: where are we now and where are we going? Dev Med Child Neurol 1992;34:547–55.
8. Pinto-Martin JA, Riolo S, Cnaan A, et al. Cranial ultrasound prediction of disabling and nondisabling cerebral palsy at age two in a low birth weight population. Pediatrics 1995;95:249–54.
9. Rabin I. Children with hearing impairment. In: Swaiman KF, ed. Pediatric neurology. St Louis: CV Mosby, 1989:895–908.
10. Murphy CC, Yeargin-Allsopp M, Decoufle P, Drews CD. Adminstrative prevalence of mental retardation in 10-year-old children, metropolitan Atlanta, 1985 through 1987. Am J Public Health 1995;85:319–23.
11. Kleinbaum DG, Kupper LL, Morgenstern H. Epidemiologic research: principles and quantitative methods. Belmont, CA: Lifetime Learning Publications, 1982.
12. Waitzman NJ, Romano PS, Scheffler RM. Estimates of the economic costs of birth defects. Inquiry 1994;31:188–205.
13. Kiely M. The prevalence of mental retardation. Epidemiol Rev 1987;9:194–218.
14. Kiely M, Lubin RA, Kiely JL. Descriptive epidemiology of cerebral palsy. Public Health Rev 1984;12:79–101.
15. Elliott LL. Epidemiology of hearing impairment and other communicative disorders. Adv Neurol 1978;19:399–420.
16. Martin JA. Aetiological factors relating to childhood deafness in the European community. Audiology 1982;21:149–58.
17. Schein JD. Childhood hearing loss: epidemiology and implications. Birth Defects 1980;16:3–7.
18. Goldstein H. The reported demography and causes of blindness throughout the world. Adv Ophthalmol 1980;40:1–99.
19. Halldorsson S, Bjornsson G. Childhood blindness in Iceland: a study of legally blind and partially seeing children in Iceland 1978. Acta Ophthalmol 1980;58:237–42.

20. Stewart-Brown SL, Haslum MN. Partial sight and blindness in children of the 1970 birth cohort at 10-years-of-age. J Epidemiol Community Health 1988;42:17–23.
21. Reschly DJ, Jipson FJ. Ethnicity, geographic locale, age, sex, and urban-rural residence as variables in the prevalence of mild retardation. Am J Ment Defic 1976;81:154–61.
22. Murphy CC, Yeargin-Allsopp M, Decoufle P, Drews CD. Prevalence of cerebral palsy among ten-year-old children in metropolitan Atlanta, 1985 through 1987. J Pediatr 1993;123(suppl): S13–S20.
23. Torfs CP, van den Berg BJ, Oechsli FW, Cummins S. Prenatal and perinatal factors in the etiology of cerebral palsy. J Pediatr 1990;116:615–9.
24. Haerer A, Anderson D, Schoenberg B. Prevalence of cerebral palsy in the biracial population of Copiah County, Mississippi. Dev Med Child Neurol 1984;26:195–9.
25. Drew CD, Yeargin-Allsopp M, Murphy CC, Decoufle P. Hearing impairment among 10-year-old children: metropolitan Atlanta, 1985 through 1987. Am J Public Health 1994;84:1164–6.
26. Yeargin-Allsopp M, Drews CD, Decoufle P, Murphy CC. Mild mental retardation in black and white children in metropolitan Atlanta: a case-control study. Am J Public Health 1995;85:324–8.
27. Brooks-Gunn J, Klebanov P, Duncan GJ. Ethnic differences in children's intelligence tests: role of economic deprivation, home environment, and maternal characteristics. Child Dev (in press).
28. Mercer JR. Labeling the mentally retarded. Berkeley, CA: University of California Press, 1973.

Prevalence of Spina Bifida at Birth — United States, 1983–1990: a Comparison of Two Surveillance Systems

Joseph M. Lary, Ph.D.
Larry D. Edmonds, M.S.P.H.

Division of Birth Defects and Developmental Disabilities
National Center for Environmental Health

*Contributors from state programs**

Abstract

Problem/Condition: Spina bifida is a birth defect of the spinal column that is a substantial contributor to serious developmental disabilities in the United States. The risk for spina bifida and other neural tube defects (NTDs) can be reduced if women consume 0.4 mg of folic acid before and during the first trimester of pregnancy. Public health programs are being developed to prevent many NTDs by increasing the consumption of folic acid by women of childbearing age. To assess the national impact of these programs on the prevalence of NTDs at birth, multistate surveillance is needed to monitor secular trends in birth-prevalence rates. This report summarizes a collaborative effort by CDC and state birth defect surveillance programs in 16 states to a) obtain multistate, population-based data concerning the birth prevalence and descriptive epidemiology of spina bifida and b) determine the usefulness of combining state surveillance data to monitor national trends in the birth prevalence of NTDs.

Reporting Period: This report presents data from birth defects surveillance systems in 16 states for the period 1983–1990 (specific periods covered varied by state). These findings are compared with CDC's Birth Defects Monitoring Program (BDMP) for the same period.

*The following persons also contributed to this report: T Flood, MD, Office of Chronic Disease Epidemiology, Arizona Dept of Health Svcs; M Brewster, PhD, Arkansas Reproductive Health Monitoring System; J Harris, MD, California Birth Defects Monitoring Program, California State Dept of Health Svcs; S Keefer, MS, Colorado Dept of Health; R Merz, MS, Hawaii Birth Defects Monitoring Program, Hawaii Dept of Health; H Howe, PhD, Div of Epidemiologic Studies, Illinois Dept of Public Health; D Krishnamurti, MS, Dept of Pediatrics, Univ of Iowa Hospitals and Clinics, Iowa City; S Panny, MD, Div of Hereditary Disorders, Maryland Dept of Health and Mental Hygiene; J Bakewell, Bur of Health Data Analysis, Missouri Dept of Health; M Seeland, Health Data Support Div, Nebraska State Dept of Health; P Costa, MA, Birth Defects Surveillance Program, Special Child Health Svcs, New Jersey State Dept of Health; C Olsen, PhD, Bur of Environmental Epidemiology and Occupational Health, New York State Dept of Health; R Meyer, PhD, State Center for Health and Environmental Statistics, North Carolina Dept of Environment, Health, and Natural Resources; L Ploughman, PhD, Dept of Human Genetics, Medical College of Virginia, Richmond; C Hill, Birth Defects Registry, Washington Dept of Health.

Description of Systems: Population-based data about live-born and stillborn infants who have spina bifida were analyzed from 16 state programs.* These 16 programs differed in size and racial/ethnic composition of the populations, surveillance methods, and completeness of case ascertainment. Hospital-based data about live-born and stillborn infants who have spina bifida also were analyzed from BDMP, a passive case ascertainment surveillance system that obtains data from participating hospitals in 50 states.

Results and Interpretation: From 1983 through 1990, the birth-prevalence rate for spina bifida for the 16 states was 4.6 cases per 10,000 births; the BDMP rate was nearly identical (4.4 cases). State-specific rates varied substantially, ranging from 3.0 (Washington) to 7.8 (Arkansas). Both state-based and BDMP rates varied among racial/ethnic groups; in both systems, the rates were highest for Hispanics and lowest for Asians/Pacific Islanders. In both the state-based surveillance systems and BDMP, the annual rate of spina bifida for the total population declined during the period 1983–1990. Much of this decline can be attributed to increased prenatal diagnosis in the 1980s. However, because the decline in the rates of spina bifida and other NTDs in the United States began before the widespread availability of prenatal diagnostic services, an environmental component may have contributed substantially to the etiologies of these defects. The birth-prevalence rate of spina bifida was slightly higher among females than males. The ratio of female-to-male prevalence rates was 1.2 for both the state-based surveillance systems and BDMP. This ratio varied considerably among racial/ethnic groups and among states. The similarities of rates and trends in the birth prevalence of spina bifida between the state-based surveillance data and the BDMP data indicate that both types of surveillance systems can provide reliable information concerning national trends in the birth prevalence of spina bifida.

Actions Taken: CDC and state birth defects surveillance programs will use results from this analysis to monitor national trends in the birth prevalence of spina bifida in the United States. Aggregated state-based surveillance data about spina bifida, anencephaly, and other NTDs will facilitate the monitoring of changes in NTDs after implementation of programs to increase folic acid consumption by women of childbearing age.

INTRODUCTION

Spina bifida, a birth defect of the spinal column that causes varying degrees of paralysis, is a major contributor to serious developmental disabilities in the United States. The public health impact of this disability is substantial. Each year, approximately 1,500 infants are born with spina bifida (*1*). The annual medical and surgical costs (based on 1985 dollars) for persons who have spina bifida exceed $200 million (*1*), and the lifetime cost to society per person who has spina bifida is estimated to be

*These programs included the Arizona Birth Defects Monitoring Program; the Arkansas Reproductive Health Monitoring System; the California Birth Defects Monitoring Program; the Colorado Registry for Children with Special Needs; the Hawaii Birth Defects Monitoring Program; the Illinois Adverse Pregnancy Outcome Reporting System; the Iowa Birth Defects Registry; the Maryland Birth Defects Reporting and Information System; the Missouri Multi-Source Birth Defects Registry; the Nebraska Birth Defects Registry; the New Jersey Birth Defects Registry; the New York State Congenital Malformations Registry; the North Carolina Birth Defects Registry; the Virginia Congenital Anomalies Reporting and Education System; the Washington State Birth Defects Registry; and CDC's Metropolitan Atlanta Congenital Defects Program.

$258,000 greater than the cost for persons who are unaffected by this disability (in 1988 dollars) (*2*). Dietary supplementation with folic acid (a B vitamin) reduces the risk for spina bifida and other neural tube defects (NTDs) (*3*). In 1992, the Public Health Service (PHS) published a recommendation that all women of childbearing age who are capable of becoming pregnant should consume 0.4 mg of folic acid per day to reduce the risk for having a pregnancy affected by spina bifida or other NTDs (*3*). PHS estimates that if all women in the United States followed this recommendation, the annual number of cases of spina bifida and other NTDs would decrease by 50% (*3*).

Multistate surveillance of birth-prevalence rates is required to assess the national impact of public health programs to reduce the prevalence of NTDs by dietary supplementation with folic acid. Until recently, the only source of ongoing information about the national birth prevalence of spina bifida and other NTDs was CDC's Birth Defects Monitoring Program (BDMP), a hospital-based surveillance system that obtains information about birth defects among newborns from discharge abstracts submitted by participating hospitals (*4*). However, in the past decade, several states have established population-based birth defect surveillance systems that collect data regarding spina bifida and other serious birth defects. In this report, CDC has analyzed data from 16 states that maintain birth defects surveillance systems to obtain multistate, population-based data regarding the birth prevalence and descriptive epidemiology of spina bifida. This report expands the analysis of previously published state-based data for 1983–1990 (*5*) and compares the results with BDMP data for the same period to assess trends.

METHODS

The participating states provided surveillance data regarding spina bifida for the period 1983–1990; however, not all states had data for the entire period. All live-born and stillborn infants who had spina bifida* were included in the analysis; however, in three states, data were not available for cases involving stillborn infants. Nine state surveillance systems (i.e., Colorado, Illinois, Maryland, Missouri, Nebraska, New Jersey, New York, North Carolina, and Virginia) identified cases of spina bifida from reports submitted by physicians and the staff of hospitals, clinics, and other health-care facilities (i.e., passive case ascertainment). Seven states (i.e., Arizona, Arkansas, California, Georgia, Hawaii, Iowa, and Washington) used trained surveillance staff to identify cases of spina bifida by systematic review of medical and other records from hospitals, clinics, and other health-care facilities (i.e., active case ascertainment). Birth-prevalence rates for spina bifida were determined for each state during the surveillance period. However, the specific surveillance periods covered from 1983 through 1990 varied by state (Table 1). BDMP rates for spina bifida in the United States for this period also were determined for comparison with state data.

Because prevalence rates have differed previously between certain racial and ethnic groups, data were collected by race/ethnicity (i.e., white, black, Asian,† American Indian,§ and Hispanic) in both the state-based surveillance systems and BDMP. State-based surveillance systems primarily determined race/ethnicity from birth certificates, whereas BDMP determined race/ethnicity from hospital discharge summaries.

*As defined by *International Classification of Diseases, 9th Revision*, Code 741.
†Includes Pacific Islanders.
§Includes Alaskan Natives.

TABLE 1. Birth prevalence* of spina bifida reported from 16 state-based birth defects surveillance systems and the Birth Defects Monitoring Program (BDMP), by race/ethnicity — United States, 1983–1990

Surveillance system	Years covered	Total live births†	Race/Ethnicity											
			White		Black		Hispanic		Asian§		American Indian¶		Total†	
			Cases	Rate	Cases	Rate	Cases	Rate	Cases	Rate	Cases	Rate	Cases	Rate
Northeast														
New Jersey	1985–1990	688,991	179	4.1	38	3.0	40	4.6	3	1.7	0	0.0	295	4.3
New York	1983–1990	2,160,230	550	4.2	182	4.3	211	6.1	—	—	—	—	983	4.6
Total		**2,849,221**	**729**	**4.2**	**220**	**4.0**	**251**	**5.8**	**3**	**1.7**	**0**	**0.0**	**1,278**	**4.5**
North Central														
Illinois	1988–1989	374,955	79	2.8	19	2.3	—	—	—	—	—	—	118	3.1
Iowa	1983–1990	319,714	159	5.2	2	2.2	—	—	0	0.0	1	7.1	166	5.2
Missouri	1983–1986	303,647	147	5.8	8	1.8	—	—	2	7.8	0	0.0	157	5.2
Nebraska	1983–1990	198,601	95	5.3	6	5.9	3	5.9	—	—	0	0.0	104	5.2
Total		**1,196,917**	**480**	**4.7**	**35**	**2.4**	**3**	**5.9**	**2**	**3.7**	**1**	**2.2**	**545**	**4.6**
South														
Arkansas	1983–1989	107,184	64	8.1	17	6.1	—	—	—	—	—	—	84	7.8
Georgia	1983–1990	269,472	106	6.5	50	4.9	—	—	2	3.5	1	54.6	163	6.0
Maryland	1984–1990	437,645	119	4.0	45	3.5	—	—	—	—	—	—	172	3.9
North Carolina	1984–1988	456,631	190	5.8	46	3.6	—	—	3	7.4	4	6.0	243	5.3
Virginia	1987–1989	264,593	78	4.0	21	3.3	—	—	0	0.0	0	0.0	99	3.7
Total		**1,535,525**	**557**	**5.3**	**179**	**4.0**	**—**	**—**	**5**	**3.0**	**5**	**7.0**	**761**	**5.0**
West														
Arizona	1986–1988	189,686	43	4.0	5	6.5	32	6.0	0	0.0	11	6.2	91	4.8
California	1983–1988	1,029,765	245	4.4	21	2.7	171	6.8	26	2.2	4	5.9	486	4.7
Colorado	1989–1990	106,188	36	4.5	0	0.0	9	5.1	0	0.0	1	11.7	53	5.0
Hawaii**	1989–1990	39,773	6	6.3	0	0.0	—	—	7	2.5	0	0.0	13	3.3
Washington	1987–1990	297,305	77	3.0	1	0.7	3	1.4	5	3.1	1	1.3	88	3.0
Total		**1,662,717**	**407**	**4.0**	**27**	**2.6**	**215**	**6.3**	**38**	**2.2**	**17**	**5.1**	**731**	**4.4**
Total		**7,244,380**	**2,173**	**4.5**	**461**	**3.7**	**469**	**6.0**	**48**	**2.3**	**23**	**5.0**	**3,315**	**4.6**
BDMP	**1983–1990**	**4,965,030**	**1,634**	**4.7**	**220**	**3.5**	**130**	**4.7**	**7**	**1.0**	**5**	**3.1**	**2,192**	**4.4**

*Rate per 10,000 live births.
†Includes persons from all racial/ethnic groups for whom data were available and persons for whom race was unknown.
§Includes Pacific Islanders.
¶Includes Alaskan Natives.
**Rates for Hawaii were estimated from the proportion of births by race in 1988. For 1989 and 1990, numbers were available only for total state births and for spina bifida cases by race.

Chi-square tests were used to compare differences between racial and ethnic groups in spina bifida rates and female-to-male rate ratios. To determine temporal trends in spina bifida rates and female-to-male rate ratios, a linear regression analysis was performed on the logarithms of the annual rates and rate ratios.

RESULTS

The participating states were grouped into the four U.S. census regions: Northeast, North Central, South, and West (Table 1). From 1983 through 1990, the birth-prevalence rate of spina bifida for these 16 states was 4.6 cases per 10,000 births. The rate of spina bifida determined by BDMP was nearly identical (4.4 cases). Although rates were similar by region, state-specific rates varied substantially, ranging from 3.0 (Washington) to 7.8 (Arkansas).

State-based rates also varied among racial/ethnic groups. The rate was highest for Hispanics (6.0) and lowest for Asians (2.3). Rates for whites, blacks, Hispanics, and Asians were all significantly different ($p<0.01$); the rate for American Indians differed significantly only from the rate for Asians ($p<0.01$). Rates determined by BDMP also varied among racial/ethnic groups. The rates were highest for Hispanics and whites (both 4.7) and lowest for Asians (1.0). Most of the rates for racial/ethnic groups were significantly different ($p<0.01$); however, the rate for American Indians did not differ significantly from rates for any other group.

The relative risk for spina bifida by race/ethnicity was determined for state-based data (Table 2). To evaluate potential confounding by state, the data were stratified by state, and a summary Mantel-Haenszel estimate for relative risk was calculated. The crude and adjusted relative risks were similar, indicating that minimal confounding occurred. Similar state-adjusted analyses were not made for BDMP data because of the limited number of cases and total births available from each state.

For state-based surveillance systems, the annual rate of spina bifida for the total population declined from a peak of 5.9 cases per 10,000 births in 1984 to 3.2 cases per 10,000 births in 1990 (Figure 1). The rate for the total population decreased by 7.8% annually during the period 1983–1990 ($p<0.01$). The rate for whites declined 9.2% annually during this period ($p<0.01$), and the rate for Hispanics declined 10.6% annually ($p<0.01$). The rate for blacks decreased 4.7% annually ($p=0.07$). Most of the BDMP rates for spina bifida also declined during the period 1983–1990 (Figure 2), although the declines were less than those in the state-based rates. The annual rate of spina bifida for the total population declined from a peak of 5.0 cases per 10,000 births in 1984 to 4.1 cases per 10,000 births in 1990. The rate for the total population decreased by 3.5% annually during the period 1983–1990 ($p<0.05$). The rate for whites decreased 2.2% annually; however, the decrease was not significant ($p=0.11$). The rate for Hispanics decreased 9.3% annually ($p=0.06$). The rate for blacks was variable and showed no significant trend over time.

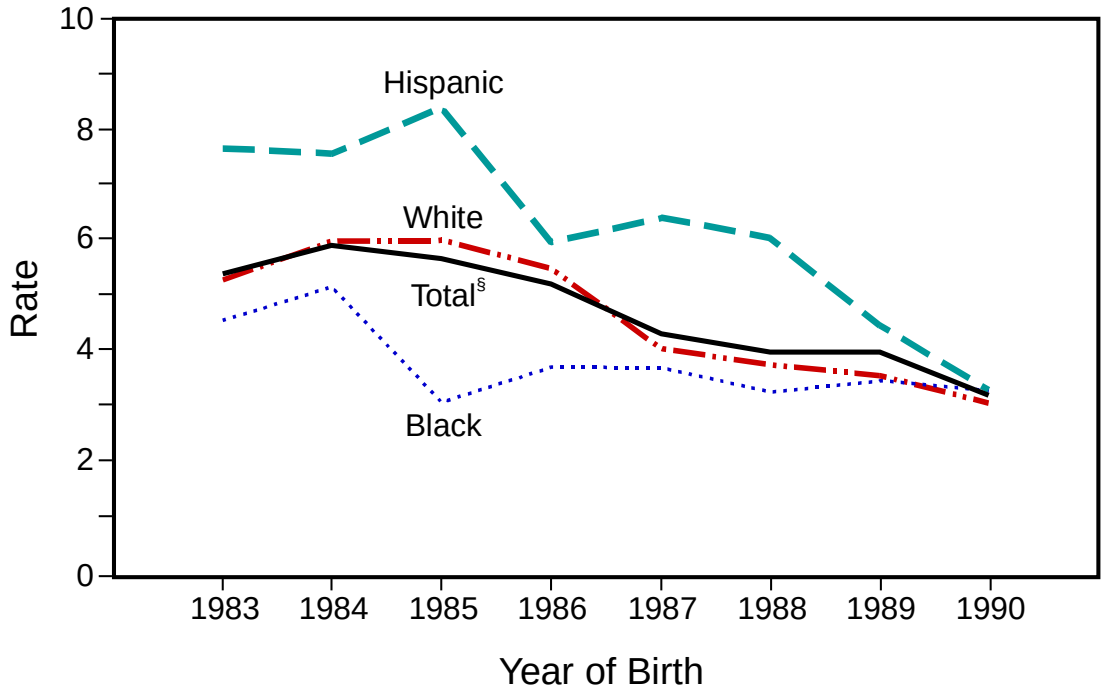
The birth-prevalence rate of spina bifida was slightly higher for females than for males (Table 3). The ratio of female-to-male prevalence rates was 1.2 for both the state-based surveillance systems and BDMP. This ratio varied considerably among racial/ethnic groups, although it was significantly different from 1.0 only among whites ($p<0.01$ for both the state-based systems and BDMP). The female-to-male rate ratio also differed considerably among states, ranging from 1.7 (North Carolina) to 0.9 (Illinois, Georgia, and New Jersey) (Table 3). Of the 16 states, 11 had ratios

TABLE 2. Relative risk for spina bifida reported from 16 state-based birth defects surveillance systems, by race/ethnicity — United States, 1983–1990

Race/Ethnicity	Crude relative risk (95% CI)*	Adjusted relative risk† (95% CI)
White	1.00§	1.00§
Black	0.82 (0.70–0.90)	0.80 (0.72–0.88)
Hispanic	1.43 (1.29–1.59)	1.41 (1.26–1.58)
Asian/Pacific Islander	0.49 (0.36–0.65)	0.51 (0.38–0.70)
American Indian/ Alaskan Native	1.05 (0.69–1.58)	1.13 (0.74–1.74)

*Confidence interval.
†Adjusted across states.
§Referent group.

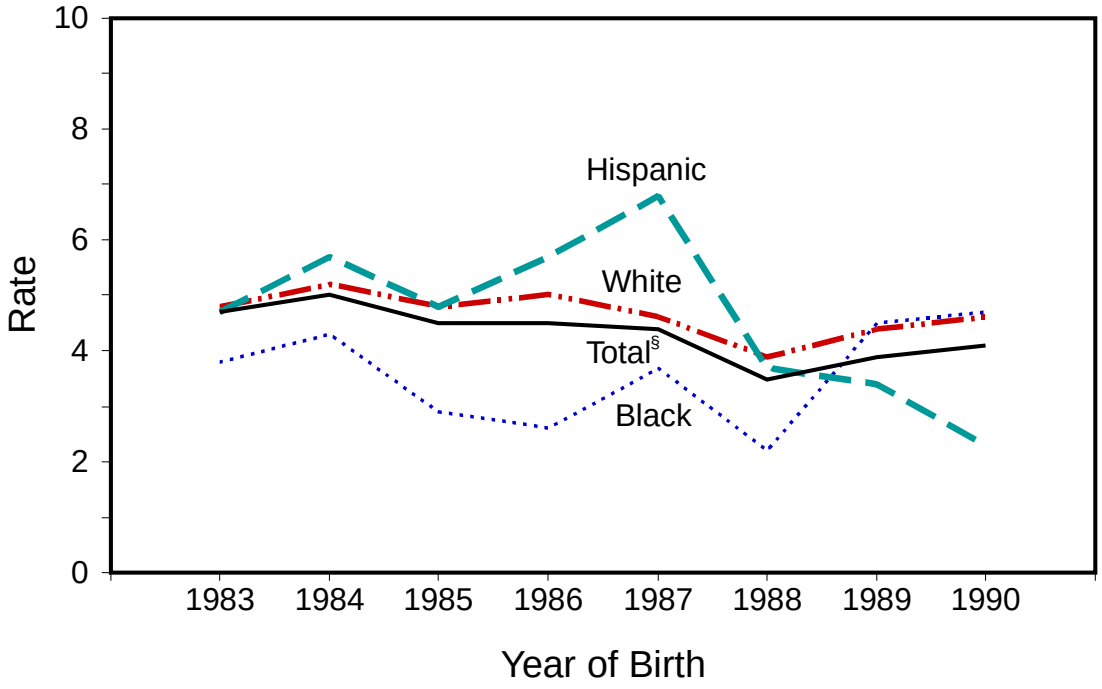
FIGURE 1. Rates* of spina bifida reported from 16 state-based birth defects surveillance systems, by race/ethnicity† and year of birth — United States, 1983–1990



*Per 10,000 live births.
†Annual rates for Asians/Pacific Islanders and American Indians/Alaskan Natives were not included because of the limited number of cases among these groups per year.
§Includes persons from all racial/ethnic groups for whom data were available and persons for whom race was unknown.

>1.0 and five had ratios <1.0. Although state-based spina bifida rates for the total population and for whites and Hispanics declined substantially from 1983 through 1990 (Figure 1), no significant decline occurred in the female-to-male rate ratio for the total population or for any racial/ethnic group (Figure 3). Among blacks, the female-to-male rate ratio was high in 1983 and 1984 compared with subsequent years, but declined substantially in 1985; the rate ratio remained relatively constant

FIGURE 2. Rates* of spina bifida reported from the Birth Defects Monitoring Program, by race/ethnicity† and year of birth — United States, 1983–1990



*Per 10,000 live births.
†Annual rates for Asians/Pacific Islanders and American Indians/Alaskan Natives were not included because of the limited number of cases among these groups per year.
§Includes persons from all racial/ethnic groups for whom data were available and persons for whom race was unknown.

thereafter. The relatively high rate of spina bifida for blacks in 1983 and 1984 (Figure 1) resulted from excess cases among females during those years. Within the BDMP population (Figure 4), the ratio of female-to-male prevalence rates decreased slightly but significantly from 1983 to 1990 for the total population (p=0.01). No significant temporal trends in rate ratios among racial or ethnic groups were noted, although the ratio for whites decreased slightly (p=0.06).

DISCUSSION

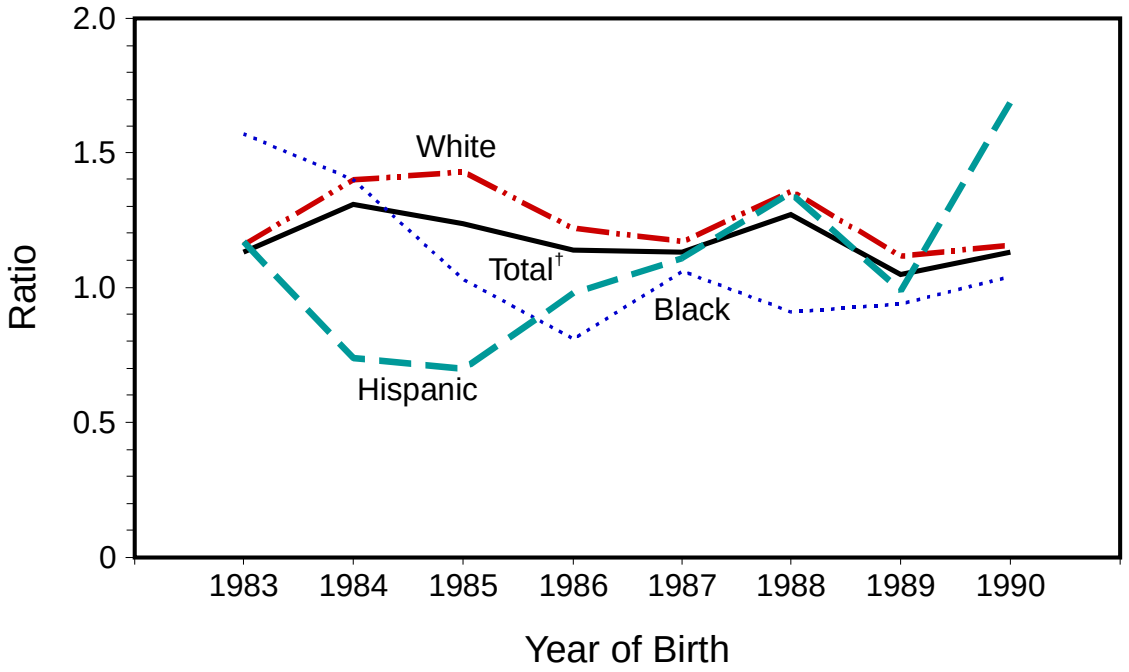
Data from both the state-based surveillance systems and BDMP indicated a decline in spina bifida birth-prevalence rates in the U.S. population from 1983 through 1990, which is consistent with the decline in spina bifida rates in previous decades in the United States (*6*). Increasing utilization of prenatal diagnosis in the 1980s likely contributed to the decline in rates during that period (*6*). In a study of the impact of prenatal diagnosis on NTD rates during the period 1985–1994, selective abortion of fetuses prenatally diagnosed with spina bifida reduced the expected birth-prevalence rate of spina bifida by 20%–30% in five of six states (*7*). However, because the decline in the rates of spina bifida and other NTDs in the United States began before the widespread availability of prenatal diagnostic services, a substantial environmental component in the etiology of these defects may also exist (e.g., improved nutrition

TABLE 3. Ratio of female-to-male prevalence rates of spina bifida reported from 16 state-based birth defects surveillance systems and the Birth Defects Monitoring Program (BDMP), by race/ethnicity — United States, 1983–1990

Surveillance system	Female-to-male rate ratio					
	White	Black	Hispanic	Asian*	American Indian†	Total
Northeast						
New Jersey	1.08	1.09	0.58	0.54	E§	0.92
New York	1.41	1.32	1.18	—	—	1.33
Total	**1.32**	**1.28**	**1.05**	**0.54**	**E**	**1.22**
North Central						
Illinois	0.98	0.75	—	—	—	0.88
Iowa	1.01	0.00	—	E	E	0.98
Missouri	1.17	0.15	—	0.00	E	1.04
Nebraska	1.28	E	E	—	E	1.50
Total	**1.10**	**0.77**	**E**	**0.00**	**E**	**1.06**
South						
Arkansas	1.26	1.89	—	—	—	1.38
Georgia	1.11	0.58	—	1.06	E	0.90
Maryland	1.04	1.08	—	—	—	1.05
North Carolina	1.82	1.59	—	0.54	3.22	1.68
Virginia	1.22	0.63	—	E	E	1.07
Total	**1.33**	**0.99**	**—**	**0.71**	**4.26**	**1.22**
West						
Arizona	0.76	0.70	1.03	E	1.21	0.90
California	1.45	0.41	0.98	1.45	0.99	1.20
Colorado	1.05	E	2.06	E	0.00	1.09
Hawaii	0.52	E	—	2.57	E	1.21
Washington	1.40	0.00	2.03	0.71	0.00	1.32
Total	**1.29**	**0.43**	**1.03**	**1.46**	**0.90**	**1.16**
Total	**1.26**	**1.05**	**1.05**	**1.15**	**1.33**	**1.18**
BDMP	**1.24**	**1.03**	**1.21**	**2.69**	**1.55**	**1.22**

*Includes Pacific Islanders.
†Includes Alaskan Natives.
§E designates a ratio that could not be calculated because of division by zero.

FIGURE 3. Ratio of female-to-male prevalence rates of spina bifida reported from 16 state-based birth defects surveillance systems, by race/ethnicity* and year of birth — United States, 1983–1990



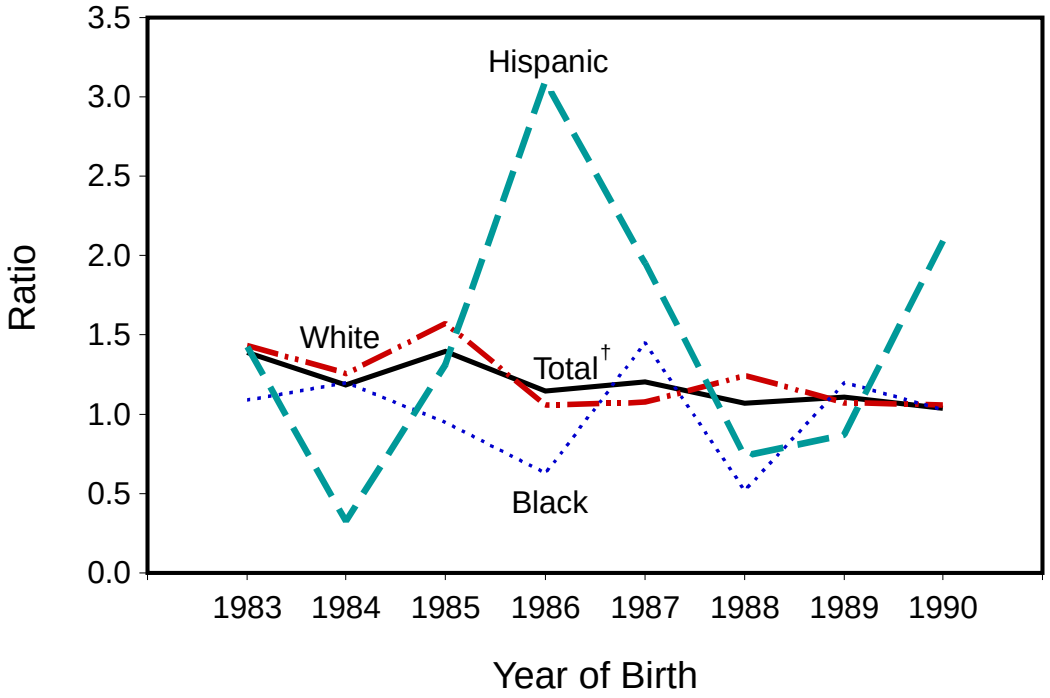
*Annual rates for Asians/Pacific Islanders and American Indians/Alaskan Natives were not included because of the limited number of cases among these groups per year.
†Includes persons from all racial/ethnic groups for whom data were available and persons for whom race was unknown.

among pregnant women) (*6*). From 1970 through 1982, the average annual percent decline in the BDMP spina bifida rate was 4.0% (*8*); from 1983 through 1990, the decline was only 3.5%. Thus, environmental factors may have played a lesser role in the rate of decline from 1983 through 1990 than in earlier years, when prenatal diagnostic services were less often available.

Studies published since 1981 indicate that dietary supplementation with folic acid can reduce the risk for spina bifida and other NTDs (*3*). In 1991, CDC recommended folic acid supplementation for women who previously had had an infant or fetus affected by an NTD and who planned to have more children (*9*). CDC recommended that these women take a 4-mg daily dose of folic acid (under a physician's supervision) beginning at least 1 month before conception and continuing throughout the first trimester of pregnancy. Subsequently, PHS issued a recommendation that all women of childbearing age who are capable of becoming pregnant should consume 0.4 mg of folic acid per day to reduce their risk for having an NTD-affected pregnancy (*3*).

The differences in spina bifida rates among the states included in this report (Table 1) can be attributed to several factors, including differences in completeness of case ascertainment; however, the rates of states that have active case ascertainment overlapped considerably with the rates of states that have passive case ascertainment. In addition, genetic and dietary differences and differences in the rate of utilization of prenatal diagnostic services also may have contributed to differences in rates by state.

FIGURE 4. Ratio of female-to-male prevalence rates of spina bifida reported by the Birth Defects Monitoring Program, by race/ethnicity* and year of birth — United States, 1983–1990



*Annual rates for Asians/Pacific Islanders and American Indians/Alaskan Natives were not included because of the limited number of cases among these groups per year.
†Includes persons from all racial/ethnic groups for whom data were available and persons for whom race was unknown.

Rates of spina bifida were highest for Hispanics, followed by whites, blacks, and Asians. The prevalence rates for American Indians were between those of Hispanics and Asians. The relative differences in spina bifida rates by race/ethnicity corroborate reports of previous racial/ethnic differences in rates for spina bifida and other NTDs (i.e., anencephaly and encephalocele) (*10–16*). In metropolitan Atlanta, rates of anencephaly and spina bifida were 3.1 and 2.5 times higher for whites than blacks, respectively (*10*). In North Carolina, the rate of anencephaly (calculated from fetal and infant death certificate data) was 3.6 times higher for whites than blacks (*11*). In Los Angeles County, rates for anencephaly, spina bifida, and encephalocele were 1.8, 1.4, and 8.0 times higher for whites than blacks, respectively (*12*). The prevalence rate of spina bifida for Asians (i.e., Chinese and Japanese) in Los Angeles County was even lower than for blacks, although the population size was relatively small (i.e., 15,000 births). A low prevalence rate of spina bifida has been reported for Asians in California compared with that for whites (*13*). Higher prevalence rates of spina bifida for Hispanics compared with non-Hispanic whites and blacks have been noted in California (*13*), New York (*14*), Los Angeles (*15*), and in the National Collaborative Perinatal Project (*16*). Although most Hispanics living in California have different origins than those living in New York (i.e., Mexico and Puerto Rico, respectively), rates for Hispanics were similar in both states (*17*). The etiology of spina bifida might have

a substantial genetic component, based on the significant racial/ethnic differences in the rates of spina bifida, both locally and nationwide (*12,18*).

For the total U.S. population, the prevalence rate of spina bifida for females was higher than the rate for males (Table 3), although the female-to-male rate ratio was less than ratios that have been reported for anencephaly (*6,19*). The rate was higher for females than for males in all racial/ethnic groups, both for state-based surveillance data and BDMP data, but the difference was significant only for whites. In most populations, spina bifida occurs more often among females than males (*19*). Differential rates of spontaneous abortion for male and female fetuses may account for the differences in sex-specific prevalence rates for spina bifida and other NTDs (*19*). Combined data from studies of spontaneously aborted fetuses with NTDs indicated that 32 (56%) of 57 abortuses were male (*19*), suggesting that the overall excess of NTD-affected females among stillbirths and live-births may partially result from a higher spontaneous abortion rate for NTD-affected males than for NTD-affected females. Other factors that may account for sex-specific differences in prevalence rates are differences in a) the rate of development of female and male embryos and b) susceptibility to teratogenic insult (*19*).

The proportion of females among the total cases of anencephaly and the birth-prevalence rate of this defect are positively associated (*20*). However, this association has not been documented for spina bifida (*19,20*). In this report, no statistically significant change in the female-to-male rate ratio occurred over time for state-based surveillance data (Figure 3), despite the steadily declining prevalence rate (Figure 1). For the BDMP data, a slight but statistically significant decrease occurred in the female-to-male rate ratio for the total population (Figure 4) that corresponded to the overall decrease in the prevalence rate over time (Figure 2). However, no consistent correlation was found in either system between annual fluctuations in the birth-prevalence rate and the female-to-male rate ratio. These findings are consistent with previous studies regarding the lack of a relationship between birth-prevalence rate and the proportion of females among the total cases of spina bifida.

CONCLUSIONS

Both the state-based surveillance systems and BDMP have provided useful data concerning national trends in the birth prevalence of spina bifida. Such data are needed to monitor changes in the prevalence of spina bifida and other NTDs following implementation of programs to increase folic acid consumption by women of childbearing age. However, whereas these surveillance systems enable monitoring of overall changes in the prevalence rates of NTDs, they cannot determine the relative contributions of folic acid supplementation and termination of NTD-affected pregnancies to changes in NTD rates. To assess the impact of these factors on the rates of NTDs in the United States, data must be collected concerning a) the use of folic acid supplements by women of childbearing age and b) prenatal diagnosis and selective abortion of fetuses that have NTDs.

References

1. CDC. Economic burden of spina bifida—United States, 1980–1990. MMWR 1989;38:264–7.
2. Waitzman NJ, Romano PS, Scheffler RM. Estimates of the economic costs of birth defects. Inquiry 1994;31:188–205.
3. CDC. Recommendations for the use of folic acid to reduce the number of cases of spina bifida and other neural tube defects. MMWR 1992;41(No. RR-14):1–7.
4. Edmonds LD, Layde PM, James LM, Flynt JW, Erickson JD, Oakley GP Jr. Congenital malformations surveillance: two American systems. Int J Epidemiol 1981;10:247–52.
5. CDC. Spina bifida incidence at birth—United States, 1983–1990. MMWR 1992;41:497–500.
6. Yen IH, Khoury MJ, Erickson JD, et al. The changing epidemiology of neural tube defects, United States, 1968–1989. Am J Dis Child 1992;146:857–61.
7. Cragan JD, Roberts HE, Edmonds LD, et al. Surveillance for anencephaly and spina bifida and the impact of prenatal diagnosis—United States, 1985–1994. In: CDC Surveillance Summaries, August 25, 1995. MMWR 1995;44(No. SS-4):1–13.
8. Edmonds LD, Windham G. Trends in the incidence of neural tube defects in the United States. In: Mizejewski GJ, Porter IH. Alpha-fetoprotein and congenital disorders. Orlando, FL: Academic Press, 1985.
9. CDC. Use of folic acid for prevention of spina bifida and other neural tube defects—1983–1991. MMWR 1991;40:513–6.
10. Erickson JD. Racial variations in the incidence of congenital malformations. Ann Hum Genet (London) 1976;39:315–50.
11. Turnbull CD, Scurletis TD, Danielson AH. Anencephalus in North Carolina: a 25-year experience. N C Med J 1977;38:713–8.
12. Sever LE. An epidemiologic study of neural tube defects in Los Angeles County II: Etiologic factors in an area with low prevalence at birth. Teratology 1982;25:323–34.
13. Shaw GM, Jensvold NG, Wasserman CR, Lammer EJ. Epidemiologic characteristics of phenotypically distinct neural tube defects among 0.7 million California births, 1983–1987. Teratology 1994;49:143–9.
14. Feldman JG, Stein SC, Klein RJ, Kohl S, Casey G. The prevalence of neural defects among ethnic groups in Brooklyn, New York. J Chronic Dis 1982;35:53–60.
15. Strassburg MA, Greenland S, Portigal L, Sever LE. A population-based case-control study of anencephalus and spina bifida in a low-risk area. Dev Med Child Neurol 1983;25:632–41.
16. Myrianthopoulos NC, Melnick M. Studies in neural tube defects I. Epidemiologic and etiologic aspects. Am J Med Genet 1987;26:783–96.
17. National Center for Health Statistics. Vital statistics of the United States, 1986, Vol 1. Natality. Washington, DC: US Department of Health and Human Services, Public Health Service, National Center for Health Statistics; DHHS publication no. (PHS)88-1123.
18. Janerich DT. Endocrine dysfunction and anencephaly and spina bifida: an epidemiologic hypothesis. Am J Epidemiol 1978;99:1–6.
19. Little J, Elwood JM. Epidemiology of neural tube defects. In: Kiely, M, ed. Reproductive and perinatal epidemiology. Boca Raton, FL: CRC Press, 1991.
20. Elwood JM, Little J, Elwood JH. Epidemiology and control of neural tube defects. Oxford, England: Oxford University Press, 1992.

State and Territorial Epidemiologists and Laboratory Directors

State and Territorial Epidemiologists and Laboratory Directors are acknowledged for their contributions to *CDC Surveillance Summaries*. The epidemiologists and laboratory directors listed below were in the positions shown as of February 1996.

State/Territory	Epidemiologist	Laboratory Director
Alabama	John P. Lofgren, MD	William J. Callan, PhD
Alaska	John P. Middaugh, MD	Gregory V. Hayes, DrPH
Arizona	Norman J. Petersen, SM (Acting),	Barbara J. Erickson, PhD
Arkansas	Thomas C. McChesney, DVM	Michael G. Foreman
California	Stephen H. Waterman, MD, MPH	Michael G. Volz, PhD
Colorado	Richard E. Hoffman, MD, MPH	Ronald L. Cada, DrPH
Connecticut	James L. Hadler, MD, MPH	Sanders F. Hawkins, PhD
Delaware	A. LeRoy Hathcock, PhD	Mahadeo P. Verma, PhD
District of Columbia	Martin E. Levy, MD, MPH	James B. Thomas, ScD
Florida	Richard S. Hopkins, MD, MSPH	E. Charles Hartwig, ScD
Georgia	Kathleen E. Toomey, MD, MPH	Elizabeth A. Franko, DrPH
Hawaii	Richard L. Vogt, MD, MPH	Vernon K. Miyamoto, PhD
Idaho	Jesse F. Greenblatt, MD, MPH	Richard H. Hudson, PhD
Illinois	Byron J. Francis, MD, MPH	David F. Carpenter, PhD
Indiana	Gregory K. Steele, DrPH, MPH	David E. Nauth (Acting)
Iowa	M. Patricia Quinlisk, MD, MPH	Mary J. R. Gilchrist, PhD
Kansas	Gianfranco Pezzino, MD, MPH	Roger H. Carlson, PhD
Kentucky	Reginald Finger, MD, MPH	Thomas E. Maxson, DrPH
Louisiana	Louise McFarland, DrPH	Henry B. Bradford, Jr, PhD
Maine	Kathleen F. Gensheimer, MD, MPH	John A. Krueger (Acting)
Maryland	Diane M. Dwyer, MD, MPH	J. Mehsen Joseph, PhD
Massachusetts	Alfred DeMaria, Jr, MD	Ralph J. Timperi, MPH
Michigan	Kenneth R. Wilcox, Jr, MD, DrPH	Robert Martin, DrPH
Minnesota	Michael T. Osterholm, PhD, MPH	Pauline Bouchard, JD, MPH
Mississippi	Mary Currier, MD, MPH	Joe O. Graves, PhD
Missouri	H. Denny Donnell, Jr, MD, MPH	Eric C. Blank, DrPH
Montana	Todd A. Damrow, PhD, MPH	Douglas O. Abbott, PhD
Nebraska	Thomas J. Safranek, MD	John D. Blosser
Nevada	Randall L. Todd, DrPH	Arthur F. DiSalvo, MD
New Hampshire	M. Geoffrey Smith, MD, MPH	Veronica C. Malmberg, MSN
New Jersey	Lyn Finelli, DrPh (Acting)	Marion Pierce
New Mexico	C. Mack Sewell, DrPH, MS	Loris W. Hughes, PhD
New York City	Benjamin A. Mojica, MD, MPH	Stanley Reimer
New York State	Dale L. Morse, MD, MS	Ann Wiley, PhD
North Carolina	J. Michael Moser, MD, MPH	Lou F. Turner, DrPH
North Dakota	Larry A. Shireley, MS, MPH	James D. Anders, MPH
Ohio	Thomas J. Halpin, MD, MPH	Kathleen L. Meckstroth, DrPH
Oklahoma	J. Michael Crutcher, MD, MPH (Acting)	Garry L. McKee, PhD
Oregon	David W. Fleming, MD	Michael R. Skeels, PhD, MPH
Pennsylvania	James T. Rankin, Jr, DVM, PhD, MPH	Bruce Kieger, DrPH
Rhode Island	Utpala Bandy, MD, MPH	Walter S. Combs, PhD
South Carolina	James J. Gibson, MD, MPH	Harold Dowda, PhD
South Dakota	Susan E. Lance, DVM, PhD, MPH	Richard S. Steece, PhD
Tennessee	William L. Moore, MD	Michael W. Kimberly, DrPH
Texas	Diane M. Simpson, MD, PhD	David L. Maserang, PhD
Utah	Craig R. Nichols, MPA	Charles D. Brokopp, DrPH
Vermont	Vacant	Burton W. Wilke, Jr, PhD
Virginia	Grayson B. Miller, Jr, MD, MPH	James L. Pearson, DrPH
Washington	Paul Stehr-Green, DrPH, MPH	Jon M. Counts, DrPH
West Virginia	Loretta E. Haddy, MA, MS	Frank W. Lambert, Jr, DrPH
Wisconsin	Jeffrey P. Davis, MD	Ronald H. Laessig, PhD
Wyoming	Gayle L. Miller, DVM, MPH	Roy J. Almeida, DrPH
American Samoa	Edgar C. Reid, MO, DSM, MPH	—
Federated States of Micronesia	Vacant	—
Guam	Robert L. Haddock, DVM, MPH	Florencia Nocon (Acting)
Marshall Islands	Tom D. Kijner	—
Northern Mariana Islands	Jose L. Chong, MD	Isamu J. Abraham, DrPH
Palau	Jill McCready, MS, MPH	—
Puerto Rico	Carmen C. Deseda, MD, MPH	Jose Luis Miranda Arroyo, MD
Virgin Islands	Donna M. Green, MD	Norbert Mantor, PhD

The *Morbidity and Mortality Weekly Report* (*MMWR*) Series is prepared by the Centers for Disease Control and Prevention (CDC) and is available free of charge in electronic format and on a paid subscription basis for paper copy. To receive an electronic copy on Friday of each week, send an e-mail message to *lists@list.cdc.gov*. The body content should read *subscribe mmwr-toc*. Electronic copy also is available from CDC's World-Wide Web server at *http://www.cdc.gov/* or from CDC's file transfer protocol server at *ftp.cdc.gov*. To subscribe for paper copy, contact Superintendent of Documents, U.S. Government Printing Office, Washington, DC 20402; telephone (202) 512-1800.

Data in the weekly *MMWR* are provisional, based on weekly reports to CDC by state health departments. The reporting week concludes at close of business on Friday; compiled data on a national basis are officially released to the public on the following Friday. Address inquiries about the *MMWR* Series, including material to be considered for publication, to: Editor, *MMWR* Series, Mailstop C-08, CDC, 1600 Clifton Rd., N.E., Atlanta, GA 30333; telephone (404) 332-4555.

☆**U.S. Government Printing Office: 1996-733-175/27051— Region IV**