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Surveillance for and Comparison of Birth Defect Prevalences in Two Geographic Areas — United States, 1983–88

Influenza — United States, 1988–89

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Trichinosis	NCID	1991; Vol. 40, No. SS-3
Tubal Sterilization Among Women	NCCDPHP	1983; Vol. 32, No. 3SS
Tuberculosis	NCPS	1991; Vol. 40, No. SS-3
Water-Related Disease	NCID	1991; Vol. 40, No. SS-3
Years of Potential Life Lost	EPO	1992; Vol. 41, No. SS-6

Abbreviations

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
CIO	Centers/Institute/Offices
NCPS	National Center for Prevention Services
IHPO	International Health Program Office
EPO	Epidemiology Program Office
NIOSH	National Institute for Occupational Safety and Health

Surveillance for and Comparison of Birth Defect Prevalences in Two Geographic Areas — United States, 1983–88

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Abstract

Problem/Condition: CDC and a number of states have developed surveillance systems to monitor the birth prevalence of major defects.

Reporting Period Covered: This report covers birth defects surveillance in Metropolitan Atlanta, Georgia and selected jurisdictions in California for the years 1983-1988.

Description of System: The California Birth Defects Monitoring Program and the Metropolitan Atlanta Congenital Defects Program are two population based surveillance systems that employ similar data collection methods. The prevalence estimates for 44 diagnostic categories are based on data from 1983 to 1988 for 639,837 births in California and 152,970 births in metropolitan Atlanta. The prevalences in the two areas are compared adjusting for race, sex and maternal age using Poisson regression.

Results: Regional differences in the prevalence of aortic stenosis, fetal alcohol syndrome, hip dislocation/dysplasia, microcephalus, obstruction of the kidney/ureter, and scoliosis/lordosis may be attributable to general diagnostic variability. However, differences in the prevalences of arm/hand limb reduction, encephalocele, spina bifida, or trisomy 21 (Down Syndrome) are probably not attributable to differences in ascertainment because these defects are relatively easy to diagnose.

Interpretation: Regional differences in prenatal diagnosis and pregnancy termination may affect prevalences of trisomy 21 and spina bifida. However, the reason for differences in arm/hand limb reduction is unknown, but may be related to variability in environmental exposure, heterogeneity in gene pool, or random variation.

Actions Taken: Because of the similarities of these data bases, several collaborative studies are being implemented. In particular, the differences in the birth prevalence of spina bifida and Down Syndrome will focus attention on the impact of prenatal diagnosis.

*Dr. Schulman is currently with Battelle Memorial Institute, Arlington, VA.

INTRODUCTION

Many states have begun birth defects monitoring programs in response to growing public concern about the potential effects of environmental hazards (*1*). The data in this surveillance summary are from one of the largest (California Birth Defects Monitoring Program [CBDMP]) and one of the oldest (Metropolitan Atlanta Congenital Defects Program [MACDP]) birth defects registries in the United States. This study was possible because of the existence of these two population-based registries that employ similar data collection methods. The prevalence of specific birth defects in two geographic areas was compared because studying the geographic distribution of disease may be important in the search for possible etiologic clues.

METHODS

The CBDMP — instituted in five San Francisco Bay Area counties in 1983 — initially monitored about 75,000 births annually. Currently, more than 300,000 births per year are monitored. Data collection specialists routinely visit all hospitals and genetic centers to identify children <1 year of age who are diagnosed with major structural malformations. The medical charts for these children are reviewed and detailed demographic and diagnostic information is abstracted (*2*).

The MACDP, which is directed by CDC and sponsored by CDC, the Emory University School of Medicine, and the Georgia Mental Health Institute, has maintained a population-based registry of malformed children since October 1967. Approximately 40,000 births occur each year in the MACDP surveillance area. Like the CBDMP, the MACDP includes an active surveillance component; data collection specialists visit hospitals to identify records of children with birth defects (*3*). Only slight differences exist between the CBDMP and the MACDP in how children's records are identified for chart review and in the criteria for diagnostic inclusion.

Diagnostic information for this report is grouped into 44 categories on the basis of a classification system developed at the MACDP. The categories represent a variety of anatomic abnormalities and reflect the malformations most frequently addressed in scientific literature.

Prevalence estimates were based on data from 1983 to 1988 for 639,897 births in California (561,737 whites and 78,160 blacks), and 152,970 births in metropolitan Atlanta (96,380 whites and 56,590 blacks). Race was defined using maternal race as designated on the birth certificate. Because few Asians and Hispanics reside in Atlanta, data for these groups were excluded in this analysis. Only data for live births were analyzed.

Because the racial composition of the two areas differs and the rates of some birth defects vary by race, the prevalence of each defect by race and by region was estimated separately. Exact 95% confidence intervals (CIs) for the estimates are presented (*4*).

The ratio of the prevalence for the MACDP compared to the CBDMP in each defect category was also estimated. Values >1.0 indicate that the prevalence is higher for the MACDP than for the CBDMP. These ratios have been adjusted for sex, maternal age, and race using Poisson regression (*5*). Approximate 95% CIs for the ratios are also presented.

TABLE 1. A comparison of the California Birth Defects Monitoring Program and Metropolitan Atlanta Congenital Defects Program, 1983–1988

Condition	Prevalence* (No. of cases, prevalence, and CI)				Prevalence ratio† and CI§ for MACDP vs. CBDMP
	White		Black		
	CBDMP	MACDP	CBDMP	MACDP	
Central Nervous System					
Anencephalus	91	27	10	5	1.49
	0.16	0.28	0.13	0.09	0.99,2.25
	0.13,0.20	0.18,0.41	0.06,0.24	0.03,0.21	
Spina bifida	250	79	21	31	1.87§
	0.45	0.82	0.27	0.55	1.49,2.35
	0.39,0.50	0.65,1.02	0.17,0.41	0.37,0.78	
Encephalocele	59	15	11	16	1.63§
	0.11	0.16	0.14	0.28	1.04,2.55
	0.08,0.14	0.09,0.26	0.07,0.25	0.16,0.46	
Microcephalus	601	48	289	67	0.37§
	1.07	0.50	3.70	1.18	0.31,0.46
	0.99,1.16	0.37,0.66	3.28,4.15	0.92,1.50	
Hydrocephalus	477	70	57	61	1.01
	0.85	0.73	0.73	1.08	0.83,1.22
	0.72,0.93	0.57,0.92	0.55,0.94	0.82,1.38	
Eye					
Anophthalmia	25	8	4	2	1.51
	0.04	0.08	0.05	0.04	0.72,3.19
	0.03,0.07	0.04,0.16	0.01,0.13	0.00,0.13	
Cataract	102	21	20	11	1.04
	0.18	0.22	0.26	0.19	0.70,1.56
	0.15,0.22	0.13,0.33	0.16,0.40	0.10,0.35	
Microphthalmia	221	32	24	29	1.05
	0.39	0.33	0.31	0.51	0.78,1.41
	0.35,0.45	0.23,0.47	0.20,0.46	0.34,0.74	
Glaucoma	38	5	7	5	0.86
	0.07	0.05	0.09	0.09	0.42,1.75
	0.05,0.09	0.02,0.12	0.04,0.18	0.03,0.21	
Cardiovascular					
Aortic stenosis	126	42	7	7	1.89§
	0.22	0.44	0.09	0.12	1.35,2.64
	0.19,0.27	0.31,0.59	0.04,0.18	0.05,0.25	
Hypoplastic left heart	121	27	15	17	1.36
	0.22	0.28	0.19	0.30	0.96,1.94
	0.18,0.26	0.18,0.41	0.11,0.32	0.18,0.48	
Single ventricle	67	8	12	2	0.52
	0.12	0.08	0.15	0.04	0.27,1.02
	0.09,0.15	0.04,0.16	0.08,0.27	0.00,0.13	
Tetralogy of Fallot	193	41	27	29	1.32
	0.34	0.43	0.35	0.51	0.99,1.74
	0.30,0.40	0.31,0.58	0.23,0.50	0.34,0.74	
Transposition of the great vessels	280	63	39	24	1.19
	0.50	0.65	0.50	0.42	0.93,1.52
	0.44,0.56	0.50,0.84	0.35,0.68	0.27,0.63	
Truncus arteriosus	63	6	3	3	0.66
	0.11	0.06	0.04	0.05	0.33,1.35
	0.09,0.14	0.02,0.14	0.01,0.11	0.01,0.15	

TABLE 1. A comparison of the California Birth Defects Monitoring Program and Metropolitan Atlanta Congenital Defects Program, 1983–1988 — Continued

Condition	Prevalence* (No. of cases, prevalence, and CI)				Prevalence ratio† and CI§ for MACDP vs. CBDMP
	White		Black		
	CBDMP	MACDP	CBDMP	MACDP	
Respiratory					
Agenesis of lung	425	68	56	49	1.01
	0.76	0.71	0.72	0.87	0.82,1.24
	0.69,0.83	0.55,0.89	0.54,0.93	0.64,1.14	
Orofacial					
Cleft lip w/wo cleft palate	647	116	65	47	1.03
	1.15	1.2	0.83	0.83	0.87,1.23
	1.06,1.24	0.99,1.44	0.64,1.06	0.61,1.10	
Cleft palate	399	54	41	37	0.90
	0.71	0.56	0.52	0.65	0.71,1.13
	0.64,0.78	0.42,0.73	0.38,0.71	0.46,0.90	
Gastrointestinal					
Gastroschisis	134	26	9	8	1.10
	0.24	0.27	0.12	0.14	0.75,1.61
	0.20,0.28	0.18,0.40	0.05,0.22	0.06,0.28	
Hirschsprung disease	85	21	18	7	1.10
	0.15	0.22	0.23	0.12	0.71,1.70
	0.12,0.19	0.13,0.33	0.14,0.36	0.05,0.25	
Malrotation of intestine	221	27	27	19	0.78
	0.39	0.28	0.35	0.34	0.56,1.08
	0.34,0.45	0.18,0.41	0.23,0.50	0.20,0.52	
Stenosis or atresia of duodenum	121	19	22	6	0.72
	0.22	0.20	0.28	0.11	0.47,1.12
	0.18,0.26	0.12,0.31	0.18,0.43	0.04,0.23	
Stenosis or atresia of rectum or anus	241	49	27	18	1.12
	0.43	0.51	0.35	0.32	0.85,1.48
	0.38,0.49	0.38,0.67	0.23,0.50	0.19,0.50	
Stenosis or atresia of small intestine	82	13	23	18	1.00
	0.15	0.13	0.29	0.32	0.66,1.52
	0.12,0.18	0.07,0.23	0.19,0.44	0.19,0.50	
Tracheo-esophageal fistula/esophageal atresia	193	27	19	8	0.77
	0.34	0.28	0.25	0.14	0.53,1.11
	0.30,0.40	0.18,0.41	0.14,0.38	0.06,0.28	
Biliary atresia	38	10	10	5	1.17
	0.07	0.10	0.13	0.09	0.64,2.15
	0.05,0.09	0.05,0.19	0.06,0.24	0.03,0.21	
Pyloric stenosis	1,346	240	56	41	1.02
	2.40	2.49	0.72	0.72	0.90,1.17
	2.27,2.53	2.19,2.83	0.54,0.93	0.52,0.98	
Genitourinary					
Bladder or urethra obstruction	111	22	19	22	1.30
	0.20	0.23	0.24	0.39	0.91,1.86
	0.16,0.24	0.14,0.35	0.15,0.38	0.24,0.59	

TABLE 1. A comparison of the California Birth Defects Monitoring Program and Metropolitan Atlanta Congenital Defects Program, 1983–1988 — Continued

Condition	Prevalence* (No. of cases, prevalence, and CI)				Prevalence ratio† and CI§ for MACDP vs. CBDMP
	White		Black		
	CBDMP	MACDP	CBDMP	MACDP	
Hypospadias, 2nd and 3rd degree	264 0.47 0.41,0.53	58 0.60 0.46,0.78	30 0.38 0.26,0.55	19 0.34 0.20,0.52	1.19 0.92,1.54
Obstruction of kidney or ureter	631 1.12 1.04,1.21	77 0.80 0.63,1.00	68 0.87 0.68,1.10	41 0.72 0.52,0.98	0.74§ 0.60,0.90
Renal agenesis	269 0.48 0.42,0.54	45 0.47 0.34,0.62	27 0.35 0.23,0.50	11 0.19 0.10,0.35	0.88 0.65,1.17
Musculoskeletal					
Arm/hand limb reduction	264 0.47 0.41,0.53	36 0.37 0.26,0.52	39 0.50 0.35,0.68	16 0.28 0.16,0.46	0.72 0.53,0.98
Arthrogryposis multiplex congenita	179 0.32 0.27,0.37	23 0.24 0.15,0.36	27 0.35 0.23,0.50	22 0.39 0.24,0.59	0.87 0.62,1.21
Hip dislocation/ dysplasia	956 1.70 1.60,1.81	101 1.05 0.85,1.27	42 0.54 0.39,0.73	17 0.30 0.18,0.48	0.61§ 0.50,0.74
Diaphragmatic hernia	166 0.30 0.25,0.34	28 0.29 0.19,0.42	14 0.18 0.10,0.30	15 0.27 0.15,0.44	1.08 0.77,1.52
Leg/foot limb reduction	125 0.22 0.19,0.27	16 0.17 0.09,0.27	18 0.23 0.14,0.36	10 0.18 0.08,0.32	0.75 0.48,1.15
Scoliosis/lordosis	190 0.34 0.29,0.39	11 0.11 0.06,0.20	22 0.28 0.17,0.43	9 0.16 0.07,0.30	0.40§ 0.25,0.64
Chromosomal					
Trisomy 13	71 0.13 0.10,0.16	8 0.08 0.04,0.16	10 0.13 0.06,0.24	9 0.16 0.07,0.30	0.84 0.49,1.45
Trisomy 18	94 0.14 0.13,0.20	17 0.18 0.10,0.28	7 0.22 0.13,0.35	18 0.32 0.19,0.50	1.20 0.81,1.79
Trisomy 21 (Down Syndrome)	555 0.99 0.91,1.07	109 1.13 0.93,1.36	48 0.61 0.45,0.81	61 1.08 0.82,1.38	1.29§ 1.08,1.53
Other					
Amniotic bands	98 0.17 0.14,0.21	14 0.15 0.08,0.24	12 0.15 0.08,0.27	7 0.12 0.05,0.25	0.81 0.50,1.31

TABLE 1. A comparison of the California Birth Defects Monitoring Program and Metropolitan Atlanta Congenital Defects Program, 1983–1988 — Continued

Condition	Prevalence* (No. of cases, prevalence, and CI)				Prevalence ratio† and CI§ for MACDP vs. CBDMP
	White		Black		
	CBDMP	MACDP	CBDMP	MACDP	
Choanal atresia	103	18	16	3	0.78
	0.18	0.19	0.20	0.05	0.48,1.26
	0.15,0.22	0.11,0.30	0.12,0.33	0.01,0.15	
Fetal alcohol syndrome	57	2	45	21	0.53§
	0.10	0.02	0.58	0.37	0.33,0.85
	0.08,0.13	0.00,0.07	0.42,0.77	0.23,0.57	
Omphalocele	127	25	14	17	1.26
	0.23	0.26	0.18	0.30	0.88,1.80
	0.19,0.27	0.17,0.38	0.10,0.30	0.18,0.48	

*Per 1,000 live births.
†Adjusted for race, sex, maternal age.
§95% CI does not include 1.00

RESULTS

Prevalence estimates are presented separately for whites and blacks in each geographic area (Table 1). Only those categories that have at least a two-fold difference in the prevalence of disease between the two areas are mentioned in the table, and at least four cases of disease for each race in each area. Only one of the 44 diagnostic categories, microcephalus, met these criteria for both races simultaneously; the prevalence for microcephalus was higher in the CBDMP than in the MACDP. The prevalence for stenosis/atresia of the duodenum among blacks and for scoliosis/lordosis among whites was higher in the CBDMP when compared with the MACDP. Conversely, the prevalence for spina bifida and encephalocele among blacks was higher in the MACDP than in the CBDMP.

When the potential confounding effects of maternal age, sex, and race were removed, the prevalence of disease in the two geographic areas was similar for most birth defect categories examined. However, the CBDMP has a higher prevalence of arm/hand limb reduction, microcephalus, obstruction of the kidney/ureter, scoliosis/lordosis, hip dislocation/dysplasia and fetal alcohol syndrome. The MACDP has a higher prevalence of spina bifida, aortic stenosis, encephalocele, and trisomy 21 (Down's Syndrome).

Data for stillbirths are not included in this analysis because the enumeration of fetal deaths (in California) is incomplete and the diagnosis of birth defects among fetal deaths is not consistent. Although estimates for both areas are higher for certain malformations (e.g., anencephalus) when stillbirths are included in the calculations, the patterns observed do not change.

DISCUSSION

The prevalences of spina bifida, encephalocele, and arm/hand limb reduction vary distinctively in the two populations described in this surveillance report. In addition, these two populations differ in the rates of anencephalus, hypoplastic left heart, and single ventricle. Because all of these defects are easy to identify and diagnose, it is unlikely that these differences are due to differences in ascertainment. Anencephalus, spina bifida, and encephalocele are all defects of the neural tube; the Metropolitan Atlanta rates were notably elevated compared to the California rates for all three of these defects. Other reports have noted a higher prevalence of spina bifida and anencephalus in the eastern states compared with the western states (*6*). In addition, regional differences in prenatal diagnosis and early termination of pregnancy may account for some of the differences in the rates for trisomy 21, but information is not available to assess the potential impact of these practices.

The reasons for the observed differences between these populations for arm/hand limb reduction, hypoplastic left heart, and single ventricle are unknown. The differences in these defects are possibly due to regional factors, environmental exposures, the genetic makeup of the two populations, or random variations. Regional differences in prenatal diagnosis and early termination of pregnancy may account for some of the differences in the rates for anencephalus, spina bifida, and encephalocele.

Statistically significant differences exist between the two populations in the prevalences of several conditions (including microcephalus, aortic stenosis, obstruction of the kidney or ureter, hip dislocation/dysplasia, scoliosis/lordosis, fetal alcohol syndrome, and a marginally significant difference for tetralogy of Fallot); however, these conditions are more difficult to diagnose. Therefore, the regional differences in these defects are possibly due to variability in case ascertainment. However, regional differences in environmental exposures or differences in genetic makeup of the populations cannot be discounted.

Although some differences are noted, the two systems are very similar. Similarities have led to a number of collaborative studies. This analysis has also pointed out the need to pay attention to regional differences in prenatal diagnosis when analyzing data.

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Influenza — United States, 1988–89

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Abstract

Problem/Condition: CDC monitors the emergence and spread of new influenza virus variants and the impact of influenza on morbidity and mortality annually from October through May.

Reporting Period Covered: This report covers United States influenza surveillance conducted from October 1988 through May 1989.

Description of System: Weekly reports from the vital statistics offices of 121 cities provided an index of influenza's impact on mortality; 58 WHO collaborating laboratories reported weekly identification of influenza viruses; weekly morbidity reports were received both from the state and territorial epidemiologists and from 153 sentinel family practice physicians. Nonsystematic reports of outbreaks and unusual illnesses were received throughout the year.

Results: During the 1988–89 influenza season, influenza A (H1N1) and B viruses were identified in the United States with essentially equal frequency overall, although both regional and temporal patterns of predominance shifted over the course of the season. Throughout the season increases in the indices of influenza morbidity in regions where influenza A (H1N1) predominated were similar to increases in regions where influenza B predominated. Only 7% of identified viruses were influenza A (H3N2), but isolations of this subtype increased as the season waned and it subsequently predominated during the 1989–90 season. During the 1988-89 season outbreaks in nursing homes were reported in association with influenza B and A (H3N2), but not influenza A (H1N1).

Interpretation: The alternating temporal and geographic predominance of influenza strains A (H1N1) and B during the 1988–89 season emphasizes the importance of con-

tinual attention to regional viral strain surveillance, since amantadine is effective only for treatment and prophylaxis of influenza A.

Actions Taken: Weekly interim analyses of surveillance data produced throughout the season allow physicians and public health officials to make informed choices regarding appropriate use of amantadine. CDC's annual surveillance allows the observed viral variants to be assessed as candidates for inclusion as components in vaccines used in subsequent influenza seasons.

INTRODUCTION

Influenza virus infections remain an important cause of morbidity and mortality in the United States (*1,2*). During major epidemics, hospitalizations among the elderly and persons with chronic health problems may increase two- to five-fold compared with nonepidemic periods (*3*); mortality from pneumonia and influenza, as well as from cardiopulmonary and other chronic diseases that can be exacerbated by influenza infection, may also increase substantially (*4*).

The most effective available preventive measure is annual immunoprophylaxis with inactivated (killed-virus) trivalent vaccine, which contains a virus from each of the three major categories of influenza viruses that have co-circulated in human populations since 1977 (B, A [H1N1], and A [H3N2]). However, long-term control of influenza by vaccination is complicated by the propensity of the virus for antigenic variation (*5*). Antigenic drift, which occurs in viruses in all three groups, necessitates annual reformulation of the vaccine (*6–8*).

Influenza-associated mortality and morbidity can also be reduced by the appropriate use of chemoprophylaxis or therapy with an influenza-specific antiviral drug (i.e., amantadine or rimantadine) (*9*). Only amantadine is currently licensed for use in the United States. The efficacy of both drugs is limited to influenza A (*10*).

CDC conducts national influenza surveillance every season from October through May to monitor influenza-associated morbidity and mortality, to detect the emergence and spread of new viral variants, and to assess the appropriateness of these variants as vaccine component candidates. This report summarizes final influenza surveillance data collected by CDC during the 1988–89 season in the United States. It updates preliminary 1988–89 data included in the previously published influenza surveillance summaries and interim reports (*11–13*) and allows comparison with surveillance trends during the seasons that both preceded and followed 1988–89.

METHODS

Sources for influenza surveillance information during the 1988–89 season were similar to those used in both previous and subsequent years. In addition to receiving occasional telephone reports of outbreaks and unusual illnesses, CDC systematically collects data through the following four surveillance systems:

1. *State and territorial epidemiologists' reports*. Influenza activity, as assessed by the state and territorial epidemiologists, is reported to CDC on a weekly basis as widespread, regional, sporadic, or no activity.*

2. *Sentinel physician surveillance network*. One hundred fifty-three physicians participated in a surveillance network in which the number of patients with influenza-like illness (ILI) per total number of patient visits by age group and the number of hospitalizations for ILI were reported weekly. In addition, a subgroup of 103 physicians collected nasopharyngeal specimens from selected patients and submitted the specimens to a contract laboratory for virus identification.

3. *World Health Organization (WHO) collaborating laboratories in the United States*. During 1988–89, 58 WHO collaborating laboratories (the majority from state or local health departments, with some university or hospital laboratories also participating) reported weekly the total number of specimens received for respiratory virus testing, as well as the number and type of influenza viruses identified.

4. *CDC 121 Cities Surveillance System*. Each week, the vital statistics offices of 121 cities reported the total number of death certificates filed that week due to all causes and the number of those for which pneumonia was identified as either the immediate or underlying cause of death or for which influenza was mentioned in any position on the certificate. A seasonal baseline was calculated by using a robust regression procedure in which a periodic regression model is applied to observed percentages of deaths due to pneumonia or influenza since 1983. An "epidemic threshold" for each season is arbitrarily set at 1.645 standard deviations above the seasonal baseline. Reported data are graphed against this seasonal baseline and epidemic threshold, providing an index for the impact of influenza on mortality (*4*).

RESULTS

State and territorial epidemiologists

Although sporadic ILI was reported by five state epidemiologists during the first surveillance week of the 1988–89 influenza season (CDC surveillance week 40, October 2–8, 1988), a sustained† regional level of influenza activity was first reported by Nebraska during the week ending December 17 (week 50) (Figure 1). Sustained widespread activity was first reported by Oklahoma the week ending January 14 (week 2 of 1989), by which time 10 states—in all regions except the Pacific region—were reporting regional or widespread levels of influenza activity (Figure 2). The number of states reporting either regional or widespread activity increased rapidly to a sustained peak of 32–36 states per week (primarily in the West South Central and South Atlantic regions) during each of 4 weeks beginning January 29 and ending February 25 (weeks 5–8) and then declined rapidly. The last reports of sustained widespread activity were received from Nebraska and Virginia the week ending March 11 (week 10). For the week ending March 25 (week 12), no states reported widespread

* *Widespread:* outbreaks of influenza-like illness (ILI) or culture-confirmed influenza in counties having a combined population of ≥50% of the state's total population. *Regional:* Outbreaks of ILI or culture-confirmed influenza in counties having a combined population of <50% of the state's total population. *Sporadic:* sporadically occurring cases of ILI or culture-confirmed influenza, with no outbreaks detected.

† *Sustained reporting:* same level reported for ≥2 consecutive weeks.

FIGURE 1. State and territorial health departments' reports of influenza-like illness, by level of activity — United States, 1988–89

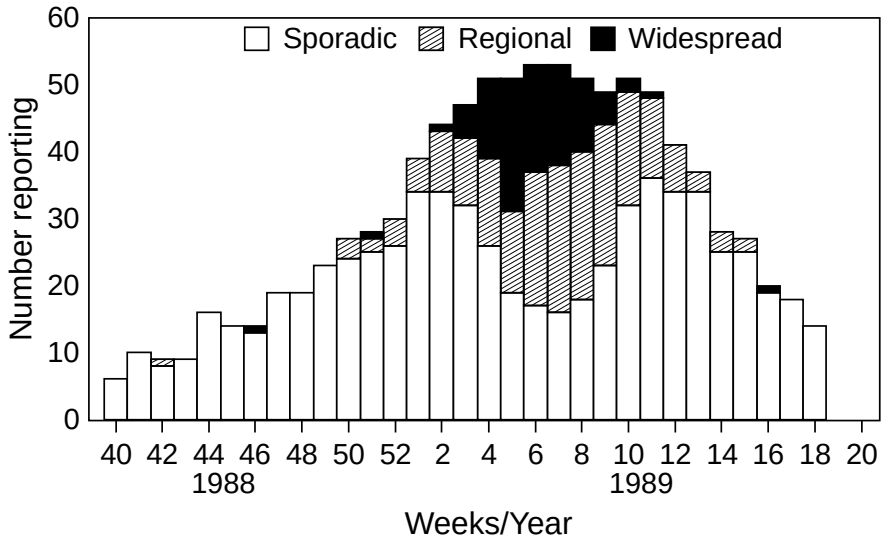
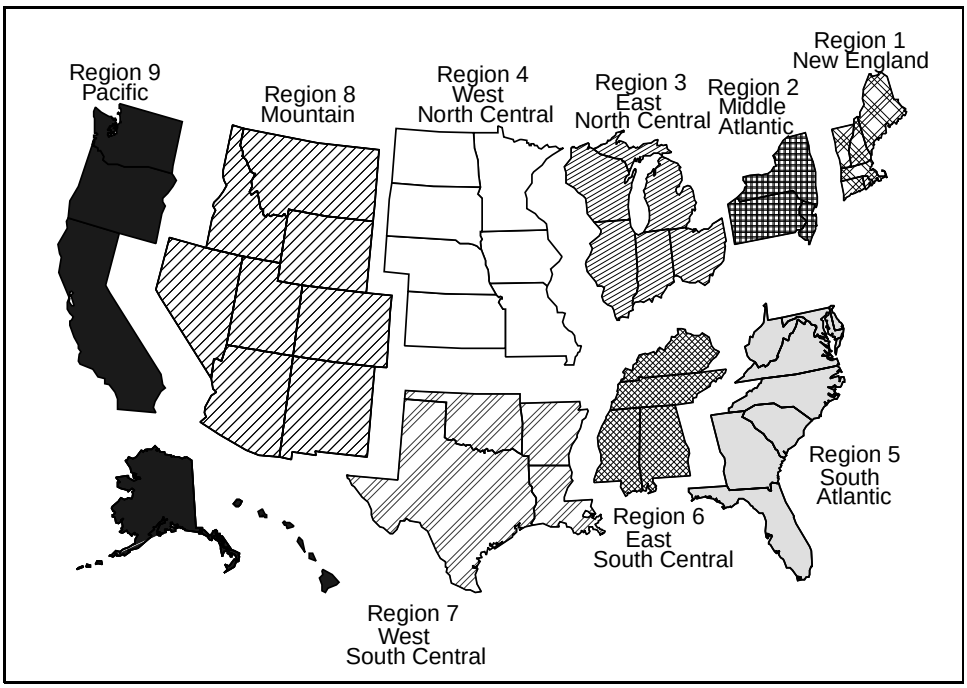


FIGURE 2. Influenza surveillance regions — United States, 1988–89



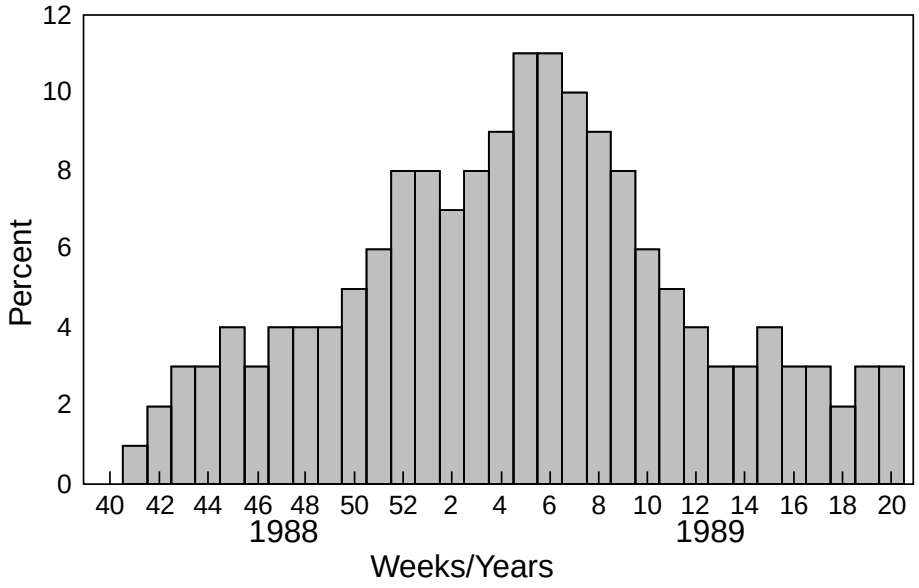
activity and only seven states continued to report regional activity, a number that declined progressively through the week ending April 22 (week 15). Reports of sporadic ILI were received from <20 states per week from April 23 through May 6 (weeks 16–18).

Sentinel physicians

Nationally, ≤2% of patient visits reported by sentinel physicians before October 16 (week 41) were attributed to ILI. From October 16 through December 11 (weeks 42–49), 3%–4% of reported patient visits were attributed to ILI. This proportion increased steadily to a plateau of 7%–8% from December 25, 1988, through January 21, 1989, peaked at 11% during the 2-week period ending February 11 (weeks 5–6), and then dropped steadily to 3% by the week ending April 1 (week 13), where it remained essentially stable through the end of the influenza surveillance season (week 20, ending May 20, 1989) (Figure 3). Nationally, ≥4% of sentinel physician office visits were attributable to ILI for the 19 weeks from November 27, 1988, through March 25, 1989 (weeks 47–12). In individual surveillance regions, the peak proportion of sentinel physician office visits attributable to ILI ranged from 9% to 22%. These regional peaks temporally clustered during the first 2 weeks in February, but individually occurred as early as January and as late as mid-March.

Overall, 3% of visits to sentinel physicians for ILI resulted in hospitalizations. The highest proportion of total office visits per week that resulted in hospitalizations for ILI (0.3% of all office visits) was reported from January 15 to February 4, 1989 (weeks 3–5). This 3-week period immediately preceded the peak of both reported influenza virus isolations by WHO laboratories (an average of 610 isolates per week during weeks 5–7) and influenza-associated mortality (7.3% of reported deaths during

FIGURE 3. Percentage of office visits attributed to influenza-like illness, as reported by sentinel physicians — United States, 1988–89



week 8). Patients >64 years old accounted for only 9% of ILI visits to sentinel physicians but 35% of ILI-associated hospitalizations.

Four hundred eighty-three specimens submitted by sentinel physicians for testing yielded 140 influenza B and 69 influenza A viruses.

WHO collaborating laboratories

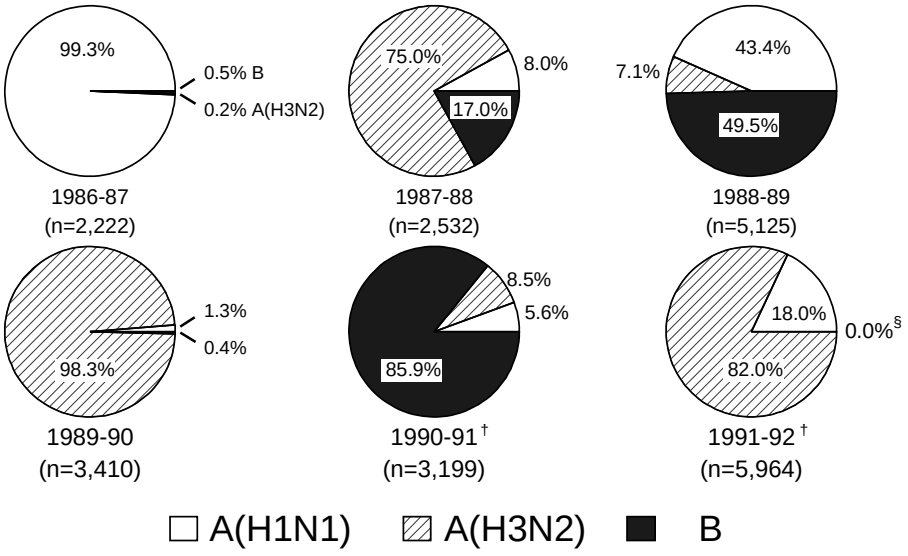
During the 1988–89 influenza season, the 58 WHO collaborating laboratories tested 29,537 specimens for respiratory viruses and identified 5,125 influenza viruses; 2,530 (49%) were influenza B and 2,595 (51%) were influenza A.

Of all influenza A isolates subtyped this season, 86% were influenza A(H1N1) viruses (Figure 4).

Reported influenza virus identifications peaked during a 5-week period from January 29 through March 3, 1989 (weeks 5–9), when 59% of all isolations were reported (range in individual regions, 41%–75%) (Figure 5).

The pattern of influenza virus isolations varied both from region to region and temporally within regions. During the early part of the season, influenza A viruses predominated among isolates reported from the New England, Middle Atlantic, East North Central, and Pacific regions. Influenza A isolations continued to predominate throughout the season in the New England region. From early February through early March, the marked predominance of influenza A viruses in each of the remaining three regions shifted to a 1- to 5-week period of co-dominance, during which time essen-

FIGURE 4. Influenza isolates* reported by the World Health Organization collaborating laboratories — United States, 1986–1992



*Not all influenza A isolates were subtyped. The percentages shown are based on those subtyped.
†Includes reports from Health Care Financing Administration Influenza Vaccine Demonstration Project Surveillance Laboratories
§0.01% influenza B

tially equal proportions of influenza A and B viruses were isolated (February 12 through 18 in the East North Central region, February 26 through March 11 in the Middle Atlantic region, and January 29 through February 18 in the Pacific region). After this co-dominant period, a clear predominance of influenza B isolations emerged in all three regions (Figure 6). Influenza B isolations remained predominant throughout the rest of the season in the Pacific region. Influenza A viruses reemerged in essentially equal proportions during the last 3 weeks that isolations were reported from the Middle Atlantic region (week 16, beginning April 16, through week 18, ending May 6). Only one isolate of influenza B was reported from the East North Central region after week 16, when influenza A virus again accounted for the majority of the relatively few influenza isolates (Table 1).

In contrast, influenza B virus clearly predominated the influenza isolates early in the season through late January in the South Atlantic, East South Central, West South Central, and Mountain regions. In the East South Central region, influenza B viruses continued to predominate throughout the season. In the remaining three regions, a 1- to 6-week period of essentially equal proportions of influenza A and B isolation began some time after January (February 12 through 18 in the West South Central region, February 5 through March 4 in the Mountain region, and January 29 through March 11 in the South Atlantic region), then shifted into a predominance of influenza A isolations for the rest of the surveillance season (Figure 6, Table 1).

FIGURE 5. Influenza virus isolates reported by World Health Organization collaborating laboratories — United States, 1988–89

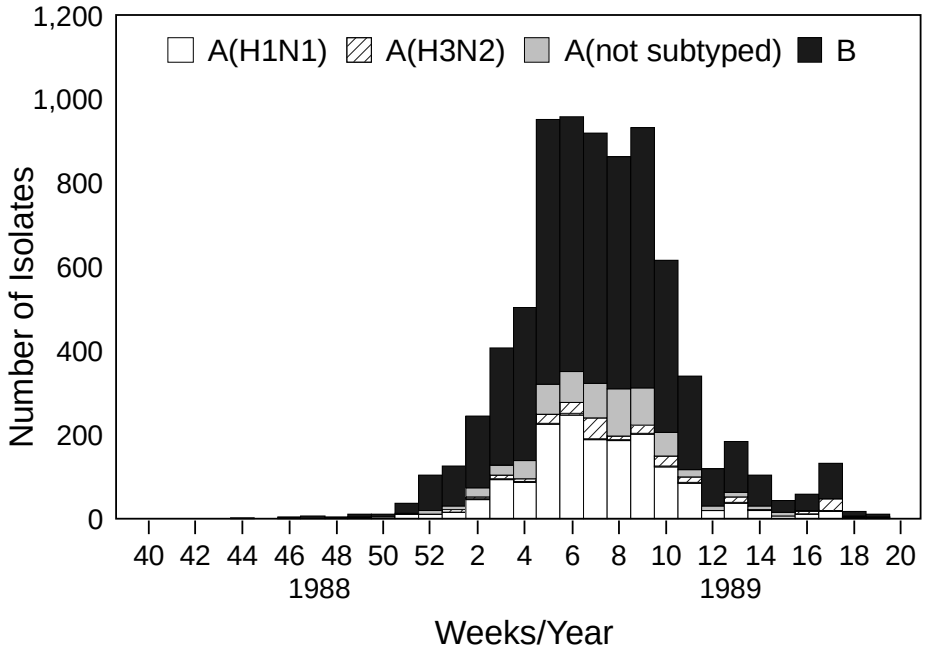
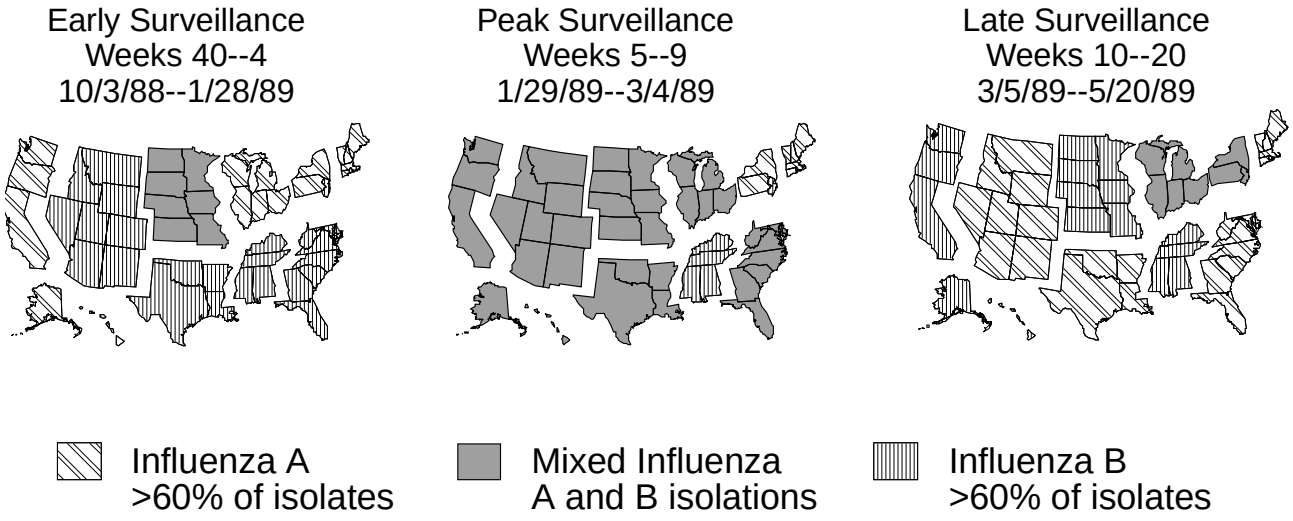


FIGURE 6. Predominant influenza type isolated in nine surveillance regions — United States, 1988–89



Influenza A and B viruses co-circulated in the West North Central region during the early part of the surveillance season. Beginning in mid-February and continuing throughout the season, influenza B isolations clearly predominated (Figure 6, Table 1).

Nationally, 74% of influenza A viruses reported were subtyped. Within each region except the New England, Pacific, and Middle Atlantic, 91%–98% of all subtyped influenza A viruses were H1N1. In these other three regions, influenza A(H1N1) viruses accounted for 83% (95/115, New England), 81% (105/130, Pacific), and 59% (256/432, Middle Atlantic) of subtyped isolates.

Within the Middle Atlantic region, 89% of all influenza A isolates were subtyped; 70% of the 157 influenza A isolates subtyped before February 11 (week 6) were influenza A(H1N1) and 30% were influenza A(H3N2). However, influenza A(H1N1) and A(H3N2) viruses were isolated with almost equal frequency (53% and 47%, respectively) from February 12 through May 20 (weeks 7–20).

Through all sources, 43 states identified and reported influenza B viruses; 35 reported influenza A(H1N1) viruses; 23 reported influenza A(H3N2) viruses; and 38 states, including nine that reported neither influenza A(H1N1) nor (H3N2), reported influenza A viruses that were not subtyped.

Antigenic drift and changes in vaccine composition, 1987–89.

During the preceding 1987–88 season, influenza A(H3N2) had been the predominant strain. Many 1987–88 season H3N2 isolates resembled two antigenically distinguishable strains first identified in China in 1987 (A/Sichuan/2/87 and A/Shanghai/11/87),* both of which were associated with outbreak activity affecting all age

*The name given to an influenza reference virus indicates both when and where it was first isolated. For example, A/Sichuan/2/87 (H3N2) was the second influenza A (H3N2) virus isolated in Sichuan, China, in 1987 and A/Shanghai/11/87 (H3N2) was the eleventh influenza A (H3N2) virus isloated in Shanghai, China, in 1987.

TABLE 1. Temporal patterns of viral isolations by World Health Organization collaborating laboratories in the United States — 1988–89

	Early Season 10/3/88–1/28/89 weeks 40–4			Peak Season 1/29/89–3/4/89 weeks 5–9			Late Season 3/5/89–5/20/89 weeks 10–20		
Region	Total isolates	(%A)	(%B)	Total isolates	(%A)	(%B)	Total isolates	(%A)	(%B)
1. New England	17	(88)	(12)	110	(94)	(6)	32	(75)	(25)
2. Middle Atlantic	84	(82)	(18)	444	(67)	(33)	265	(45)	(55)
3. East North Central	153	(65)	(35)	380	(52)	(48)	111	(55)	(45)
4. West North Central	128	(51)	(49)	551	(46)	(54)	155	(32)	(68)
5. South Atlantic	67	(21)	(79)	364	(54)	(46)	98	(62)	(38)
6. East South Central	14	(14)	(86)	29	(28)	(72)	27	(30)	(67)
7. West South Central	326	(10)	(90)	606	(51)	(49)	120	(87)	(13)
8. Mountain	159	(31)	(69)	207	(45)	(55)	96	(73)	(27)
9. Pacific	90	(86)	(14)	315	(51)	(49)	177	(31)	(69)
National totals:	**1038**	**(41)**	**(59)**	**3006**	**(54)**	**(46)**	**1081**	**(51)**	**(49)**

groups (*14*). Antigenic drift from the 1987–88 vaccine strain A/Leningrad/360/86 (H3N2) can be inferred from the reactions of each of these variants with ferret antisera (Table 2). On the basis of these and other findings, A/Sichuan/2/87 was chosen for inclusion in the 1988–89 vaccine (*15*) (Table 3).

During the 1988–89 season, influenza A/Shanghai/11/87-like strains predominated among the influenza A(H3N2) isolates antigenically characterized by the WHO reference laboratory at CDC, although influenza A(H3N2) isolates were a minor component of that season. This strain replaced A/Sichuan/2/87 as the A(H3N2) vaccine component for the 1989–90 season (*11*) (Table 3).

Toward the end of the previous season (1987–88), isolations of both influenza B and A(H1N1) had increased. Antigenic variants of influenza B virus had circulated during the 1987–88 season; most outbreak-associated variants had resembled B/Victoria/2/87 (*14*), a new variant first identified in 1987 that was incorporated into the influenza vaccine for the 1988–89 season (Table 3).

During the 1988–89 season, influenza B viruses isolated outside Asia and characterized at CDC were predominantly B/Victoria/2/87-like. However, an antigenic variant, B/Yamagata/16/88, was first identified in Japan in the spring of 1988. Viruses isolated inside Asia were either B/Victoria/2/87-like or B/Yamagata/16/88-like, and the latter was chosen for inclusion in the 1989–90 vaccine (*11*) (Table 3). The antigenic relationships between B/Victoria/2/87, B/Yamagata/16/88, and B/Ann Arbor/1/86 (which had been the B component of the 1987–88 vaccine) can be inferred from the ferret antisera data (Table 2).

TABLE 2. Hemagglutination titers of influenza viruses with serum specimens from infected ferrets*

Influenza B		**Ferret antisera**	
Reference antigen	**B/Ann Arbor/1/86**	**B/Victoria/2/87**	**B/Yamagata/16/88**
B/Ann Arbor/1/86	**80**	40	5
B/Victoria/02/87	20	**40**	5
B/Yamagata/16/88	10	10	**320**
Influenza A (H3N2)		**Ferret antisera**	
Reference antigen	**A/Leningrad/360/86**	**A/Sichuan/2/87**	**A/Shanghai/11/87**
A/Leningrad/360/86	**2560**	80	160
A/Sichuan/2/87	80	**640**	320
A/Shanghai/11/87	640	160	**1280**

* Titers are used to infer antigenic relationships between viruses. Differences of fourfold in titer of a serum with two viruses are normally indicative of an experimentally significant variation between the viruses. In some cases, only asymmetric differences are seen when several variants are tested simultaneously.

TABLE 3. Components of the United States influenza vaccines, 1987–88 through 1992–93 seasons

Season	**Influenza A (H1N1)**	**Influenza A (H3N2)**	**Influenza B**
1987–88	A/Taiwan/1/86	A/Leningrad/360/86	B/Ann Arbor/1/86
1988–89	A/Taiwan/1/86	A/Sichuan/2/87	B/Victoria/2/87
1989–90	A/Taiwan/1/86	A/Shanghai/11/87	B/Yamagata/16/88
1990–91	A/Taiwan/1/86	A/Shanghai/16/89	B/Yamagata/16/88
1991–92	A/Taiwan/1/86	A/Beijing/353/89	B/Panama/45/90
1992–93	A/Texas/36/91	A/Beijing/353/89	B/Panama/45/90

Most influenza A(H1N1) viruses characterized during both 1988–89 and the two preceding seasons (1986–87 and 1987–88) were A/Taiwan/1/86-like. A/Taiwan/1/86 (H1N1) had been a stable trivalent vaccine component for two seasons and was retained for the 1989–90 vaccine (*10,16,17*) (Table 3).

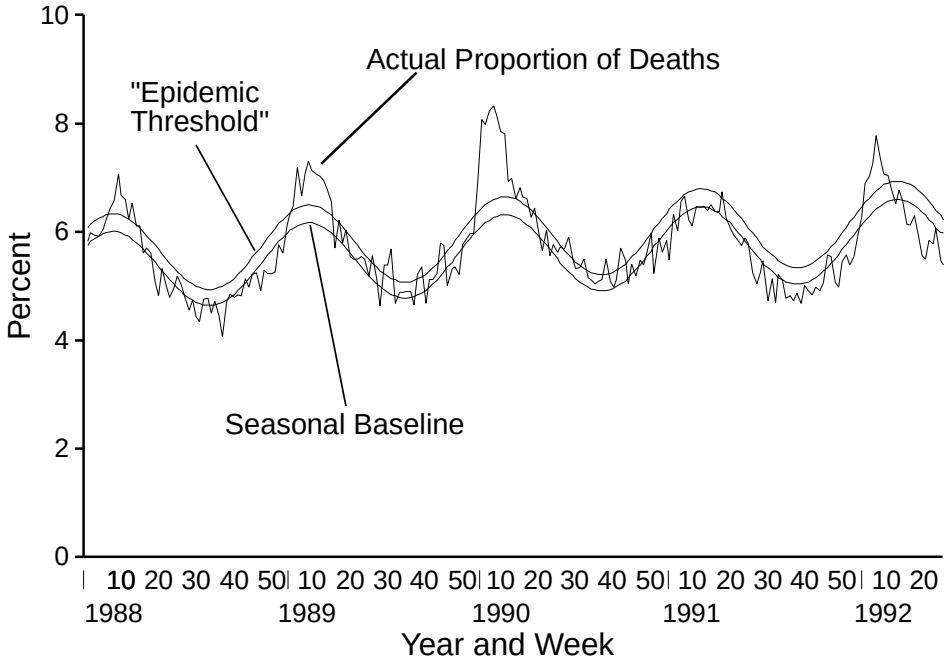
CDC 121 cities surveillance system

The proportion of all deaths reported through the 121 Cities Surveillance System that were attributed to pneumonia and influenza exceeded the epidemic threshold for 11 weeks, from January 21 through April 8, 1989 (weeks 4–14), and peaked at 7.3% of deaths reported the week ending February 25 (week 8) (Figure 7).

Outbreak reports

From mid-November through the end of January (weeks 45–5), the South Atlantic, West South Central, East North Central, and Pacific regions reported influenza B outbreaks in middle or elementary schools, with high absentee rates and occasional school closures. Influenza B was the most frequently isolated virus in only two (South Atlantic and West South Central) of these four regions during this period. Outbreaks of influenza A (not subtyped) were reported from schools in the Pacific, New England, and South Atlantic regions. An outbreak of influenza A(H3N2) was reported from a nursing home in the Middle Atlantic region, which had the highest proportion (41%) of influenza A isolates subtyped as H3N2. An outbreak of influenza B in a nursing home

FIGURE 7. Percentage of deaths due to pneumonia or influenza, CDC 121 Cities Surveillance System — United States, January 1, 1988–May 15, 1992



was reported from the West South Central region. No reports of influenza A (H1N1) outbreaks in nursing homes were received, despite the relative predominance of this influenza virus among circulating subtypes.

DISCUSSION

Analysis of influenza surveillance data reveals distinct patterns of interplay of circulating influenza subtypes that vary among seasons. The co-dominance of two influenza types (influenzas B and A[H1N1]) during the 1988–89 season was very unusual. During most seasons one influenza type predominates, but others circulate in substantial numbers. Infrequently, such as during the 1989–90 season, circulating influenza strains are almost entirely (>98%) one subtype.

The rare co-dominance of influenza B and influenza A (H1N1) viruses during the 1988–89 season and the shifting pattern of regional predominance over time make its mixed nature clearer than in other recent mixed influenza seasons such as 1991–92 (*13*). The variable spatial and temporal predominance of influenza A and B viruses during the 1988–89 season demonstrates the importance for influenza surveillance of continued virus culturing throughout the season. This alternating predominance also highlights the importance for practicing clinicians of continued attention to local influenza surveillance patterns, since amantadine is effective only against influenza A.

The temporal and geographic changes in predominance of influenza B or A(H1N1) illustrated clearly by strain surveillance were not reflected in distinguishable changes in patterns of visits to physicians' offices or outbreak activity. Reports of outbreaks in schools associated with both influenza B and A were received in essentially equal numbers, though reports of influenza A-associated outbreaks tended to be among older age groups. Although 86% of all 1988–89 influenza A isolates subtyped were H1N1, outbreaks in nursing homes were reported only in association with influenza B, A (not subtyped), and A (H3N2).

The "herald wave" phenomenon, in which a relatively small cluster of influenza virus infections occurring during the latter half of one influenza season is predictive of the predominant virus for the following season, was first described by Glezen and colleagues (*18,19*). Subtle herald waves both preceded and closed the 1988–89 influenza season. The co-dominant nature of the 1988–89 season had been heralded by a late season increase in isolation of both B and A(H1N1) influenza viruses during the 1987–88 season, during which influenza A(H3N2) had been predominant among circulating viruses (*14*). A herald wave of influenza A(H3N2) isolations that began late in the 1988–89 season presaged the predominance of this viral subtype during the subsequent 1989–90 season (*12*).

Although the influenza A (H3N2) component of the trivalent influenza vaccine had been stable for three seasons, from 1983 to 1986 (*20–22*), sufficient antigenic diversity was identified among circulating A (H3N2) viruses during each season from 1986 through 1991 (including 1988–89) to warrant an annual change in this vaccine component (*11,14–16,23–25*).

B/Victoria/2/87 was first identified in 1987, and B/Yamagata/16/88 was first recognized in 1988. The vaccine component was changed from B/Victoria/2/87 to B/Yamagata/16/88 between the 1988–89 and 1989–90 seasons (*11*). However, influenza B viruses closely related to both these reference strains continued to co-circulate

through 1991 (*11–15*), although the identification of B/Victoria lineage viruses was increasingly infrequent after 1990 (*12,13,16,24*).

A/Taiwan/1/86 had initially been isolated during the spring of 1986, incorporated into a supplemental monovalent vaccine for the 1986–87 season (*26*), and subsequently into the trivalent vaccine for the 1987–88 season. In the absence of substantial identified antigenic drift among circulating influenza A (H1N1) viruses, A/Taiwan/1/86 remained the only constant vaccine component through five seasons (including 1988–89) until 1992–93 (*11,14,15,23–25*) (Table 3).

Analysis of influenza surveillance in the United States during the 1988-89 season revealed an unusual co-dominance of influenza B and Influenza A (H1N1) viruses. The spatial and temporal predominance of these virus types varied throughout the season, complicating clinical decision-making regarding the appropriate use of antiviral agents.

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State and Territorial Epidemiologists and Laboratory Directors

State and Territorial Epidemiologists and Laboratory Directors are gratefully acknowledged for their contributions to this report. The epidemiologists listed below were in the positions shown as of February 12, 1993, and the laboratory directors listed below were in the positions shown as of January 1993.

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Connecticut	James L. Hadler, MD, MPH	Sanders F. Hawkins, PhD (Acting)
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