CENTERS FOR DISEASE CONTROL



### January 27, 1984 / Vol. 33 / No. 3

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# Perspectives in Disease Prevention and Health Promotion

# Health-Risk Estimates for 2,3,7,8-Tetrachlorodibenzodioxin in Soil

At the request of the Environmental Protection Agency and the State of Missouri, CDC undertook a risk assessment study of 2,3,7,8-tetrachlorodibenzodioxin (TCDD) levels in soil. Within the last year, CDC advised Missouri that, in two specific residential areas, soil levels above 1 part per billion (ppb) ( $\mu$ g/kg) of TCDD could result in an unreasonable risk to human health. Later, on June 28, 1983, this assessment was reviewed by a group of outside consultants,\* and the assessment was expanded to cover industrial, commercial, farm, and uninhabited areas. The following summarizes these deliberations.

Adequate dose-response data for chronic effects of TCDD are not available from epidemiologic studies of humans. Therefore, extrapolations from animal toxicity experiments (including carcinogenicity and reproduction effects) to possible human health effects have been used to estimate a reasonable level of risk from exposure to this agent. Extrapolations have been derived from a review of published studies; a complex set of assumptions related to human exposure to contaminated soil; and estimates of (1) a dose-response curve, (2) appropriate margins of safety, and/or (3) applicable mechanisms of action.

TCDD is a known carcinogen in animals, and there is considerable discussion about the best way to calculate excess cancer risk in humans exposed to TCDD. General issues of concern include use of appropriate mathematical models for predicting responses at the low end of the dose-response curve, how to use dose-response data from different tissue sites (e.g., liver, lung), and what conversion factors to use in extrapolating from animals to humans to account for species variations (1). Using data from previous toxicologic studies with female rats (2-4), dose-response estimates were derived. The lower boundary of the confidence interval for a dose estimated to increase the risk of developing cancer by one per million was then calculated. For liver cancer (the most sensitive tissue site), a virtually safe dose (VSD)

<sup>\*</sup>Consultants: Conference on Polyhalogenated Aromatic Compunds—Dr. Donald Barnes, Dr. Judy Bellin, Dr. James Falco, U.S. Environmental Protection Agency; Dr. Frank Cordle, U.S. Food and Drug Administration; Dr. George F. Fries, Pesticide Degradation Laboratory, U.S. Dept of Agriculture; Dr. Donald Grant, Health Protection Br, Health and Welfare, Canada; Dr. Robert Harris, Hazardous Waste and Research Div, Center for Energy and Environmental Studies, Princeton University; Dr. David G. Hoel, National Institute of Environmental Health Sciences; Dr. Nancy Kim, Bureau of Toxic Substances Assessment, New York State Dept of Health; Dr. George Lucier, National Institute of Environmental Health Sciences; Dr. Norton Nelson, New York University Medical Center, Institute of Environmental Medicine; Dr. Henry Pitot, McArdle Cancer Research Institute, University of Wisconsin; Dr. Charles F. Reinhardt, DuPont Company, Haskell Lab; Dr. Robert G. Tardiff, Vienna, Virginia; Dr. John Van Ryzin, Div of Biostatistics, School of Public Health, Columbia University.

### Health-Risk Estimates - Continued

was estimated as 28 femtograms (fg)<sup>†</sup> per kg body weight (b.w.) per day. For the risk of inducing tumors in less sensitive tissues, a VSD of 1,428 fg/kg b.w./day was estimated. These doses were then extrapolated directly to humans. The model used was linear; therefore, the levels for an increased risk of one excess cancer per 100,000 are 276 fg to 14.3 picograms (pg)<sup>†</sup>/kg b.w./day.

In addition, using standard toxicologic approaches for assessing reproduction effects of TCDD exposure, a VSD was estimated. Since a "no-observed-effect level" has never been determined, the VSD extrapolated to humans was derived by using a safety factor of 1,000 applied to the lowest level tested in subhuman primates. Using failure to conceive and fetal wasteage as the most sensitive reproduction effects, a VSD of 100 pg/day was derived. However, this VSD is higher than that estimated using carcinogenisis as an end point.

CDC estimated the absorption of TCDD from soil via dermal, gastrointestinal, or respiratory routes for humans having exposure to contaminated soil adjacent to their homes (Figure 1). Estimates of exposure to chemicals in soil are inherently more difficult than estimates of exposure to chemicals in air, water, or food, because soil contamination is less homogeneous, and exposure depends chiefly on individual activities. In this model, the consultants concluded that the greatest contribution of risk came from ingestion of soil, particularly during childhood; the next, from dermal absorption. (Inhalation is a relatively minor contributor in areas with abundant vegetation.) One ppb of TCDD in residential soil was chosen as a level of concern, and at substantially higher levels (e.g., greater than 100 ppb TCDD in soil), calculated risks may increase.

 $^{\dagger}$ Fg = 10<sup>-15</sup>g; pg = 10<sup>-12</sup>g.





\*For an average daily 70kg person over a 70-year lifetime.

<sup>†</sup>The average daily dose of TCDD that would be received if 100% (or 1%) of the accessible soil were contaminated at the given level.

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# Health-Risk Estimates - Continued

The level of concern for 2,3,7,8-TCDD in soil should not be viewed as a universal standard but rather as an operational starting point to analyze each situation. Characteristics unique to each situation—including locations of the contaminated soil, composition of the population exposed, and the likely frequency and duration of future exposures—factor significantly in assessing each case. These characteristics and the potential for limiting or eliminating future exposure in a timely fashion will influence decisions about appropriate actions at specific sites.

In special situations, e.g., horse-riding arenas, with high dust or soil resuspension, inhalation of TCDD may become a more prominent route of exposure, and this would affect risk estimates. For this level of concern, other routes of exposure that may be indirectly related to soil contamination have not been considered, such as food-chain contamination via grazing cattle or bioconcentration in bottom-feeding fish. Soil levels of TCDD in pastures where cattle graze and pigs root might have to be lower because of the potential for bioaccumulation.

In similar residential areas, 1 ppb of TCDD in soil is a reasonable level at which to consider limiting human exposure. Principal attention should be given to children at play who might ingest such soil.

In assessing the implications of this level of concern for any particular site, one should use additional information and recognize a complex set of underlying assumptions, such as the amount of TCDD people might receive, how often they are exposed, and whether humans have the same response to TCDD as animals. To err on the side of public safety, these assumptions should be conservative and should address factors related to: uniformity of TCDD concentration in soil; uniformity of human access (particularly children's access) to and activity on the soil; intensity, frequency, and duration of exposure; and the bioavailability of TCDD in different soils and through different types of exposure. Furthermore, when soil is measured for TCDD concentration, the adequacy of the sampling plan, the degree of laboratory extraction of TCDD from soil, and the accuracy of its subsequent measurement must be considered. *Reported by Office of the Director, Special Studies Br, Chronic Diseases Div, Center for Environmental Health, CDC.* 

Editorial Note: For many environmental (and occupational) toxins, adequate dose-response data from epidemiologic studies of humans are not available for predicting risk, particularly at low levels of exposure. Often, toxicologic data on animals are the best available predictors of risk. Cumulative human intake (exposure, dose) can also not be measured directly in many instances (e.g., TCDD, asbestos, formaldehyde); exposure assessments reflect the likely human intake on the basis of chemical levels in the environment and of human activity patterns. Many environmental standards and regulatory decisions—from safe drinking water and air standards to individual decisions at Superfund<sup>§</sup> sites or other settings—are based on exposure and risk assessment.

### Reference

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- Kociba RJ, Keyes DG, Beyer JE, et al. Results of a two year chronic toxicity and oncogenicity study of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in rats. Toxicol Appl Pharmacol 1978; 46:279-303.
- National Toxicology Program. Carcinogenisis bioassay 2,3,7,8-tetrachlorodibenzo-p-dioxin (CAS #1 174-6-01-6) in Swiss-Webster mice (dermal study). National Toxicology Program Tech Rep Ser 1982; 201:113.
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<sup>&</sup>lt;sup>§</sup>Known officially as the Comprehensive Environmental Response Compensation and Liability Act of 1980. This act provides for liability, compensation, cleanup, and emergency response for hazardous substances released into the environment and the cleanup of inactive hazardous waste disposal sites.

Epidemiologic Notes and Reports

# **Update: Sporadic Hemorrhagic Colitis**

In 1983, CDC reported on investigations in Michigan and Oregon of two 1982 outbreaks of a gastrointestinal illness designated hemorrhagic colitis (1). The illness was caused by a previously unrecognized pathogen, *Escherichia coli* O157:H7. Since August 1982, sporadic cases of this illness have been reported to CDC, and stool specimens have been examined from patients meeting the following case definition: a person with bloody diarrhea, abdominal cramps, and low-grade or no fever, whose stool culture is negative for recognized pathogens including *Salmonella*, *Shigella*, *Campylobacter*, and *Yersinia* and for ova and parasites.

During 1983, stool specimens were examined from 35 ill persons in 16 states. *E. coli* O157:H7 was identified in 10 specimens collected a mean of 4.7 days after onset of illness. The culture-negative specimens were collected a mean of 7.6 days after onset. Culture-positive specimens were received from Wisconsin (three), California (two), Alabama, Florida, Illinois, Massachusetts, and Minnesota (one each). Patients ranged in age from 2 to 80 years (median 15 years), and both sexes were equally affected. The average duration of illness was 10 days, and nine of the 10 patients were hospitalized. Barium enemas of two patients revealed spasm in one and "thumbprinting" in the ascending colon in the other. Sigmoidoscopy performed in two other patients revealed erythema, edema, and friable mucosa. None of the patients required transfusions, and four were treated with antibiotics.

Reported by WE Birch, DVM, State Epidemiologist, Alabama Dept of Public Health, JJ Sacks, MD, Acting State Epidemiologist, Florida Dept of Health & Rehabilitative Svcs; J Chin, MD, State Epidemiologist, California State Dept of Health Svcs; BJ Francis, MD, State Epidemiologist, Illinois Dept of Public Health; NJ Fiumara, MD, State Epidemiologist, Massachusetts Dept of Public Health; AG Dean, MD, Minnesota State Dept of Health; JP Davis, MD, State Epidemiologist, Wisconsin State Dept of Health and Social Svcs; Enteric Diseases Br, Div of Bacterial Diseases, Center for Infectious Diseases, CDC.

**Editorial Note:** The frequency with which *E. coli* O157:H7 causes hemorrhagic colitis in the United States is unknown. Isolation of this pathogen from 29% of submitted specimens suggests that it is an important cause of bloody diarrhea in patients in whom no other pathogens are detected. The organism is cleared rapidly from the stool (1); since the stool specimens were collected earlier for culture-positive cases than for culture-negative cases, *E. coli* O157:H7 may also have been the responsible pathogen in some of the culture-negative cases.

Disease caused by *E. coli* O157:H7 has not been limited to the United States nor to gastrointestinal manifestations. Sporadic cases of hemorrhagic colitis were also identified in Canada during 1983 (2). Three of these patients subsequently developed hemolytic-uremic syndrome. *E. coli* O157:H7 was isolated from stools of two of these patients and from the stools of two ill siblings of the third patient, who had typical symptoms of hemorrhagic colitis before developing hemolytic-uremic syndrome.

Since early stool collection is important for identifying this organism, physicians encountering typical cases should obtain the specimen as quickly as possible and then hold a portion frozen while their laboratories perform examinations for other recognized pathogens. If these test results are negative, arrangements can be made through state epidemiologists and state laboratory directors to examine the frozen portion of the specimen for *E. coli* O157:H7.

Although the outbreak cases were caused by eating hamburger products, no common exposures have yet been identified among sporadic cases. The sources of *E. coli* O157:H7 for sporadic cases are currently under investigation through an ongoing case-control study.

# Hemorrhagic Colitis - Continued

# References

- 1. Riley LW, Remis RS, Helgerson SD, et al. Hemorrhagic colitis associated with a rare *Escherichia coli* serotype. N Engl J Med 1983;308:681-5.
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# Respiratory Virus Surveillance — United States, 1983-1984

Reports of noninfluenza respiratory virus isolations from certain state and university laboratories received by CDC through January 18 show: (1) large numbers of respiratory syncytial virus (RSV) isolates were reported beginning in December and continuing into January from the New England, Mid-Atlantic, and South Atlantic regions. Large numbers of RSV were also reported from the Mountain region through December (no January data available). The South Atlantic region reported the largest number of RSV isolates: 70 of 114 respiratory specimens tested in December and January were positive for RSV. Fewer RSV isolates were reported for the same period in the East South Central, West South Central, and Pacific regions; (2) parainfluenza type 1 isolates peaked in October, with very few isolates reported in December and January; (3) smaller numbers of parainfluenza types 2 and 3 and rhinovirus isolates were reported throughout this period in some regions.

Reported by LL Minnich, MS, CG Ray, MD, Arizona Health Science Center, Tucson; B Lauer, MD, M Levin, MD, University of Colorado Health Sciences Center, Denver; C Brandt, PhD, HW Kim, MD, Children's Hospital National Medical Center, District of Columbia; L Pierik, K McIntosh, MD, The Children's Hospital, Boston, Massachusetts; P Swenson, PhD, North Shore University Hospital, Manhasset, CB Hall, MD, University of Rochester Medical Center, Rochester, New York; H Friedman, MD, S Plotkin, MD, The Children's Hospital of Philadelphia, Pennsylvania; M Kervina, MS, E Sannella, MS, PF Wright, MD, Vanderbilt University School of Medicine, Nashville, Tennessee; L Corey, MD, Children's Orthopedic Hospital, Seattle, Washington; Respective State Virus Laboratory Directors; Div of Viral Diseases, Center for Infectious Diseases, CDC.

Editorial Note: RSV is the major lower respiratory tract pathogen in infants and children under 2 years old (1). In this age group, it is the principal etiologic agent of bronchiolitis and

	New England	Mid Atlantic	South Atlantic	East South Central	West South Central	Mountain	Pacific
November 1983	3	0	14	3	6	7	2
December 1983	10	20	55	4	12	52	9
January 1984 <sup>†</sup>	26	21	15	§	§	§	§
Total	39	41	84	7	18	59	11

# TABLE 1. Respiratory syncytial virus (RSV) isolates, by region — United States, November 1983-January 1984\*

\*No RSV isolates reported from the East North Central and West North Central regions during this time. †Includes isolates identified through January 18, 1984.

<sup>§</sup>January 1984 data pending.

### Respiratory Virus – Continued

pneumonia and can be a serious nosocomial pathogen, especially in patients with compromised cardiac and respiratory systems (2,3). Hospitalized infants and young children with proven or suspected RSV infections should be placed in contact isolation during their illnesses (4). RSV infections recur throughout life, with illness in adults usually an upper respiratory infection, though there are reports of outbreaks of RSV with lower respiratory tract illness and death in the elderly. Outbreaks of RSV occur yearly throughout the United States beginning sometime between late fall and spring. They usually last from 2 to 5 months.

### References

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- 2. Hall CB. Nosocomial viral respiratory infections: perennial weeds on pediatric wards. Am J Med 1981;70:670-6.
- MacDonald NE, Hall CB, Suffin SC, Alexson C, Harris PJ, Manning JA. Respiratory syncytial viral infection in infants with congenital heart disease. N Engl J Med 1982;307:397-400.
- 4. Garner JS, Simmons BP. Guideline for isolation precautions in hospitals. Infect Control 1983; 4(suppl):245-325.

		3rd Week Endir	ng	Cumula	tive, 3rd Week	Ending
Disease	January 21, 1984	January 22, 1983	Median 1979-1983	January 21, 1984	January 22, 1983	Median 1979-1983
Acquired Immunodeficiency Syndrome (AIDS)	32	N	N	113	N	N
Aseptic meningitis	91	103	54	264	268	209
Encephalitis: Primary (arthropod-borne						
& unspec.)	1 11	21	18	24	60	44
Post-infectious	1 1	3	. 2	-;	4	Å
Gonorrhea: Civilian	14 803	18.954	19 589	43 445	55 090	55 090
Military	288	458	520	1 100	1 4 7 5	1 571
Hepatitis: Type A	464	513	513	993	1 2 1 0	1 2 1 0
Type B	398	443	327	1 0 1 8	1 129	912
Non A, Non B	59	58	Ň	149	131	Ň
Unspecified	124	146	172	249	370	447
Legionellosis	8	7	Ň	16	30	Ň
Leprosy	3	8	2	12	19	7
Malaria	9	14	16	28	30	37
Measles: Total*	5	11	24	28	26	86
Indigenous	3	10	N	24	20	Ň
Imported	2	1	Ň	4	6	Ň
Meningococcal infections: Total	40	60	62	122	145	149
Civilian	40	55	62	122	138	147
Military		5			7	1
Mumps	53	88	129	166	192	272
Pertussis	18	13	13	36	40	40
Rubella (German measles)	8	3	42	22	28	110
Syphilis (Primary & Secondary): Civilian	553	663	581	1.338	1,992	1.718
Military	6	11	7	9	34	25
Toxic Shock syndrome	5	4	N	17	21	Ň
Tuberculosis	307	350	429	818	915	1.025
Tularemia	-	4	2	3	8	5
Typhoid fever	6	3	4	12	18	18
Typhus fever, tick-borne (RMSF)		3	-	3	4	4
Rabies, animal	33	77	83	120	242	237

### TABLE I. Summary-cases specified notifiable diseases, United States

### TABLE II. Notifiable diseases of low frequency, United States

	Cum. 1984		Cum. 1984
Anthrax	-	Plague	-
Botulism: Foodborne	- 1	Poliomyelitis: Total	-
Infant (Ark. 1)	2	Paralytic	-
Other (Mass. 1)	1	Psittacosis (Mass. 1, Ga. 1)	3
Brucellosis	3	Rabies, human	-
Cholera		Tetanus	-
Congenital rubella syndrome	- 1	Trichinosis	2
Diphtheria	-	Typhus fever, flea-borne (endemic, murine)	2
Leptospirosis	- 1		

\*Two of the 5 reported cases for this week were imported from a foreign country or can be directly traceable to a known internationally imported case within two generations.

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······································	<b>I</b>	Asentic	Encer	halitis			н	epatitis (V	'iral), by tv	ре		
Reporting Area	AIDS	Menin- gitis	Primary	Post-in- fectious	Gond (Civ	orrhea ilian)	A	В	NA,NB	Unspeci- fied	Legionel- losis	Leprosy
	Cum. 1984	1984	Cum. 1984	Cum. 1984	Cum. 1984	Cum. 1983	1984	1984	1984	1984	1984	Cum. 1984
UNITED STATES	113	91	24	2	43,445	55,090	464	398	59	124	8	12
NEW ENGLAND	4	1	-	-	1,628	1,417	8	14	1	10	-	1
N.H.	-	-	-	-	30	36	2	i	1	-	-	-
Mass.	1	-	-	-	568	656	6	12	-	10	-	1
Conn.	3	ī	-	-	73 876	555	-	-	-	-	-	-
MID ATLANTIC	11	6	1	-	4,111	5,801	121	87	4	24	1	-
N.Y. City	-	5	-	-	2,230	2,607	99	39	-	14	1	-
N.J. Pa.	11	Ū	1	:	565 918	866 1,744	13 U	14 U	1 U	6 U	Ū	-
E.N. CENTRAL	5	10	5	-	6,068	7,933	12	24	4	5	3	1
Ohio Ind.	4	2	1	-	2,094 347	2,268 1,156	2	7	1	1	3	-
HI. Minh	-	3	1	-	807	1,738	7	3	-	-	-	-
Wis.	-	4	-	-	640	645	-	-	-	-	-	-
W.N. CENTRAL	1	3	1		2,135	2,696	19	8	1	2	-	-
lowa	1	-	1	-	268	306	-	3	-	1	-	-
Mo. N. Dak.	-	1	2	-	863 20	1,207 25	3	2	-	1	-	-
S. Dak.	-	1	-	-	67	56	12	-	-	-	-	-
Kans.	-	-	-	-	380	518	-	-	-	-	-	-
S. ATLANTIC	9	15	4	2	9,327	13,226	11	74	11	11	1	-
Md.	4	i	2	-	1,752	1,985	1	15	3	2	-	-
D.C. Va.	2	1	-	2	617 1 186	942 1.194	3	1 9	3	1	-	-
W. Va.	-	-	-	-	110	161		-		-	-	-
S.C.	-	-	-	-	1,086	1,480	-	11	-		-	-
Ga. Fla.	1	3 4	1	:	2,318	2,371 3,235	2 4	22 8	2	4	1	-
E.S. CENTRAL	-	7	1	-	3,909	5,052	17	21	3	4	1	-
Ky. Tenn	-	1	1	-	505 1.623	630 1.762	8	2 9	2	3	-	:
Ala. Miss	-	6	-	-	1,211	1,674	5	8	1	1	1	-
WS CENTRAL	_	2	1		6992	300	3 02	2	5	35		-
Ark.	-	-	-		600	624	2	2	-	1	-	-
La. Okla.	-	-	-	-	1,886 784	955 912	- 6	1	2	-	-	-
Tex.	-	2	1	-	3,713	5,297	85	30	3	34	-	•
MOUNTAIN Mont	3	3	-	-	1,376	1,577	47	19	4	11	1	-
Idaho	-	-	-	-	75	69	-	-	-	-	-	-
Wyo. Colo.	-	2	-	-	31 383	62 367	17	- 6	-	4		-
N. Mex.	3	-	-	-	160	217	1	1	1	-	-	-
Utah	-	-	-	-	76	453	4	-	-	-	-	-
Nev.	-	1	-	-	250	256	-	-	-	1	-	-
PACIFIC Wash.	80	44 1	11	-	7,908 117	9,600 560	136 12	115 8	26 5	22	1	10
Oreg. Calif.	80	36	11	-	388 7,135	367 8,302	28	6 98	4 17	22	1	-
Alaska	-		-	-	160	179	-	-	-			-
Guam	-	,	-	-	108	192	1	3	-	-	-	1
P.R.	-	1	-	-	178	14 186	U -	7	U -	U 1	U -	:
v.i. Pac. Trust Terr.	-	Ū	-	-	28	26	u U	2 U	Ū	Ū	Ū	:

TABLE III. Cases of specified notifiable diseases, United States, weeks ending

N: Not notifiable

U: Unavailable

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I		r		/0.4				T							
	Malaria	India	Mea	sles (Rut	oeola)	Total	Menin- gococcal	Mur	nps	, I	Pertussis	•		Rubella	
Reporting Area	Cum. 1984	1984	Cum. 1984	1984	Cum. 1984	Cum. 1983	Infections Cum. 1984	1984	Cum. 1984	1984	Cum. 1984	Cum.	1984	Cum.	Cum.
UNITED STATES	3 28	3	24	2	4	26	122	53	166	18	36	40	8	22	28
NEW ENGLAND	2	-	-	-	-	-	4	1	3		1	1	1		20
Maine	-	-	-	-	-	-	-	-	1	-	-	-	-	-	-
Vt.	-	-	-			-	-	2	1	-	-	1	-		-
Mass. R I	2	-	-	-	-	-	1	1	1	-	-	-	1	1	-
Conn.	-	-	-	-		-	1	-	-	-	1	-	2	-	-
MID ATLANTIC	-	-	-	-	-	-	9	10	34	1	2	9	_	_	1
Upstate N.Y.	-	-	-	-	-	-	4	4	6	1	2	6	-	-	i
N.J.		-	-	-	-		1	-	26	-		3	-	-	-
Pa.	-	υ	-	U	-	-	ĩ	Ŭ	1	U	-	-	Ū		-
E.N. CENTRAL	-	-	9	-	-	14	25	11	41	-	1	13	-	2	5
Ind.	-	-	-	-	-	-	2	7	12	-	-	9	-	-	1
III. Adiata	-	-	9	-	-	14	5	3	15	-	1	1	-	1	-
Wis.	-	-	-	-	:	-	5 2	1	10 4	-	2	- 3	-	1	1
W.N. CENTRAL	2					_	11	1	6	1	4	2			
Minn.	-	-	-	-	-	-	-	-	-	-	2	-	-		2
lowa Mo.	2	-	-	-	-	-	6	1	1	1	2	1	-	-	-
N. Dak.	-	-	-	-	-	-	-	-	-	-	-		-	-	-
S. Dak. Nebr	-	-	-	-	-	-	1	-	-	-	-	-	-	-	-
Kans.	-	-	-	-	-	-	1	-	2	1	-	1	-		1
S. ATLANTIC	4		-	-			38	5	13	2	4				
Del.	2	-	-	-	-	-	1	-	1	-	-	-	-	-	-
Md. D.C.	2	-	-	-	-	-	3	1	4	-	-	-	-	-	-
Va.	-	-	-	-	-	-	6	-	1	1	1	1	-		-
W.Va. N.C.	:	-	-	-	-	-	-	1	2	-	1	1	-	-	-
S.C.	-	-	-	-	-	-	5	-	-	-	-	-	-	2	-
Ga. Fla	-	-	-	-	-	-	11	1	1	-	-	2	-	-	1
		-			•	-	10	N	N	2	2	-	1	1	-
E.S. CENTRAL	-	-	-	-	-	-	3	1	4	2	2	-	-	-	!
Tenn.	-	-	-	-	-	-	2	-	-	i	i		-	-	1
Ala. Miss	-	-	-	-	-	-	1	1	1	-	-	-	-	-	-
		-	-	•	•	-	-	-	-	-	-	-	-	-	-
W.S. CENTRAL Ark	-	-	-	-	-	-	7	1	1	3	3	5	3	3	2
La.	-	-	-	-	-	-	2	-	-	-	3		-	-	-
Okla. Tex.	-	-	-	-	-	-	1	N	N	-	-	2	-	-	-
				-	•	-	4			-	-	5	3	3	2
Mont.	1	-	12	-	-	-	4	12	30	4	11	3	2	2	1
Idaho	-	-	-	-	-	-		-	i	-	-	-	2	-	-
VVyo. Colo.	-	-	-	-	-	-	-	-	-	-	Ā	-	-	-	-
N. Mex.	-	-	-	-	-	-	-	Ň	Ň	1	1	- 2	-	:	-
Ariz. Utah	1	-	12	-	-	-	-	11	28	-	-	-	-	-	-
Nev.	-	-	- 12	-	-	-	-	2	-	-	-	1	2	2	1
PACIFIC	19	3	3	2	4	12	21	11	34	4	R	2		10	
Wash. Oreg	1	-	-	-	-		2	1	4	2	5	-	-	-	- 14
Calif.	17	3	3	1+	3	11	6 13	N R	N 28	2	-	-	-		.:
Alaska	-	-	-		-	-	-	ž	2	-	-	-	-	- 13	14
- awaii	1	-	-	1 †	1	1	-	-	-	-	-	-	-	-	-
Guam P.R.	- 2	U	-	U	-	-	-	U	÷	U	-	-	U		-
V.I.	-	-	-	:	-	2	:	3	-	-	-	1	-	-	÷
Pac. Trust Terr.	-	U	-	U	-	-	-	U	-	U	-	-	Ū	-	-

# TABLE III. (Cont.'d). Cases of specified notifiable diseases, United States, weeks ending January 21, 1984 and January 22, 1983 (Third Week)

\*For measles only, imported cases includes both out-of-state and international importations.

N: Not notifiable U: Unavailable <sup>†</sup>International <sup>§</sup>Out-of-state

Reporting Area	Syphilis (Primary &	Syphilis (Civilian) (Primary & Secondary)		Tuber	rculosis	Tula- remia	Typhoid Fever	Typhus Fever (Tick-borne) (RMSF)	Rabies, Animal
	Cum. 1984	Cum. 1983	1984	1984	Cum. 1984	Cum. 1984	Cum. 1984	Cum. 1984	Cum. 1984
UNITED STATES	1,338	1,992	5	307	818	3	12	3	120
NEW ENGLAND	32	56	-	4	23	-	-	-	1
Maine	-	-	-	1	2	-	-	-	1
N.H. Vt	-	1	-	1	1	-	-	-	-
Mass.	23	36	-	-	11	-		-	
R.I.	-1	1	-	-		-	-	-	-
Conn.	8	18	-	2	8	-	-	-	-
MID ATLANTIC	178	218	-	50	164	-	1	-	12
Upstate N.Y.	8	12	-	8	25	-	1	-	-
N.Y. City	110	133	-	26	67	-	-	-	-
N.J. Pa.	39	39	ū	16 U	40	-	-	-	12
							•		
E.N. CENTRAL	54	110	2	56	99	-	2	-	10
Ind	24	40	-	3	10	-		-	1
III.	-	33	1	17	39	-	-	-	3
Mich.	9	13	1	18	18	-	-	-	-
Wis.	6	9	-	8	10	-	1	-	5
W.N. CENTRAL	26	25	-	12	21	-	-	-	23
Minn.	6	12	-	2	2	-	-	-	3
lowa	3	2	-	2	4	-	-	-	8
MO. N. Dak	16	9	-	0	9	-	-	-	37
S Dak	-	-	-	-	1	-	-	-	,
Nebr.	-	-		-	ż	-	-	-	-
Kans.	1	2	-	2	3	-	-	-	2
S. ATLANTIC	372	487	-	78	204	-	-	-	20
Del.		3	-	-	2	-	-	-	-
Md.	25	29	-	10	34	-	-	-	-
D.C.	12	19	-	!	6	-	-	-	-
Va.	20	3/	-	4	5	-	-	-	10
N C	34	50	-	13	32		-	-	
S.C.	40	41	-	5	29	-	-	-	-
Ga.	-	86	-	17	17	-	-	-	9
Fla.	238	221	-	26	71	-	-	-	-
E.S. CENTRAL	99	155	-	29	57	-	-	-	7
Ky.	4	8	-	5	10	-	-	-	2
Tenn.	27	50	-	6	8	-	-	-	2
Miss	38	25	-	18	39	-	-	-	3
W.S. CENTRAL	303	483	2	17	29	-	-	1	27
Ark.	83	89	1	-	10	-	-	1	ь
Okla.	6	11	1	4	10	-	-	-	3
Tex.	204	377	-	13	19	-	-	-	18
MOUNTAIN	35	40	-	6	15	3	2	2	2
Mont.	-	2	-		-	-	ī	2	-
Idaho		1	-	-	-	-	-	-	-
Wyo.	1	1	-	-	-	-	-	-	-
LOID.	4	10	-	-	-	-	-	-	-
Ariz	12	13		2	5	-		-	2
Utah	2	1	-	-	1	2	-	-	-
Nev.	14	4	-	1	1	-	-	-	-
PACIFIC	239	418	1	55	206	-	7		18
Wash.	-	18	-	-	4	-	-	-	-
Oreg.	10	3	1	4	9	-	-	-	
Calif.	221	394	-	39	174	-	7	-	17
Alaska Hawaii	- 2	- 2	-	- 12		-	-	-	ı
	0	3	-	12	19	-	-	-	-
Guam	- 26	-	υ	U	-	-	-	-	
r.n. VI	1	- 1	-	-	12	-	-	-	1
Pac. Trust Terr	· ·	-	Ū	ū	-	-	-	-	-

# TABLE III. (Cont.'d). Cases of specified notifiable diseases, United States, weeks ending January 21, 1984 and January 22, 1983 (Third Week)

U: Unavailable

### TABLE IV. Deaths in 121 U.S. cities,\* week ending

### January 21, 1984 (Third Week)

		All Caus	es, By A	ge (Year	s)					All Cause	es, By Aç	je (Years	.)		
Reporting Area	All Ages	≥65	45-64	25-44	1-24	<1	P&I** Total	Reporting Area	All Ages	≥65	45-64	25-44	1-24	<1	P&I** Total
NEW ENGLAND Boston, Mass. Bridgeport, Conn.	760 209 56	536 135 46	159 53 7	41 11 3	9 4	14 6	70 26 4	S. ATLANTIC Atlanta, Ga. Baltimore, Md.	1,374 166 231	939 107 142	270 37 59	89 12 16	37 5 7	35 5 7	59 4 6
Cambridge, Mass.	12	10	1	1	-	-	2	Charlotte, N.C.	63	47	9	5	-	2	4
Hartford Conn	38	32	5	1	-	-	1	Jacksonville, Fla. Miami, Ela	119	72	33	9	5	-	6
Lowell, Mass.	30	22	4	2	1	1	4	Norfolk, Va.	40	20	14	3	2	1	1
Lynn, Mass.	18	11	5	2	-	-	-	Richmond, Va.	96	62	20	6	4	4	9
New Bedford, Mas	27	24	3	-	-	-	2	Savannah, Ga.	66	35	21	7	1	2	7
Providence, R.I.	85	59	18	3	3	1	11	Tampa, Fla.	54	35	11	2	1	5	5
Somerville, Mass.	12	9	3	-	-	-	-	Washington, D.C.	§ 180	158	1	5	6	ő	4
Springfield, Mass.	57	44	.9	2	1	1	3	Wilmington, Del.	74	49	18	4	2	1	6
Worcester, Mass.	52	38	11	2		1	4	E S. CENTRAL	1.058	665	274	73	23	23	67
			••	-		•	3	Birmingham, Ala.	186	120	44	9	6	7	6
MID. ATLANTIC	2,672	1,815	552	184	49	72	139	Chattanooga, Ten	n. 79	60	12	7	-	-	10
Allentown, Pa.	24	18	6	4	2	6	5	Knoxville, lenn.	105	63	29	10	3	3	4
Buffalo, N.Y.	122	84	32	2	1	3	16	Memphis, Tenn.	295	199	69	21	4	2	20
Camden, N.J.	46	27	12	6	1	-	2	Mobile, Ala.	53	35	9	7	-	2	1
Elizabeth, N.J. Frie Pat	30	25	2	1	2	-	3	Montgomery, Ala.	70	44	17	4	3	2	6
Jersey City, N.J.	50	34	11	4	1	-	-	Nashville, Tehn.	140	70	54	0	5	5	9
N.Y. City, N.Y.	1,542	1,045	302	118	33	44	67	W.S. CENTRAL	1,944	1,187	470	139	76	72	92
Newark, N.J.	49	24	12	10	2	1	1	Austin, Tex.	47	33	10	4	-	-	6
Philadelphia, Pa.t	208	133	53	15	2	7	3	Corpus Christi Te	v 93	32	15	6	1	3	6
Pittsburgh, Pa.†	91	55	26	5	1	4	2	Dallas, Tex.	246	149	65	13	9	10	7
Reading, Pa.	36	30	5	1		-	4	El Paso, Tex.	71	41	19	4	6	1	3
Schenectady NY	28	85	21	5	2	4	13	Fort Worth, Tex.	135	88	28	9	2	8	12
Scranton, Pa.†	27	18	7	1	1	2	3	Little Bock Ark	100	205	25	45	2/	22	23
Syracuse, N.Y.	95	73	17	4	1	-	1	New Orleans, La.	260	140	80	24	11	5	-
Trenton, N.J.	44	30	9	3	1	1	1	San Antonio, Tex.	245	166	46	14	9	10	15
Yonkers, N.Y.	27	18	ż	1	-	1	2	Shreveport, La. Tulsa, Okla.	93 127	60 89	27 19	2 12	2 4	2	1
E.N. CENTRAL	2,503	1,615	565	154	77	92	95	MOUNTAIN	697	464	144	39	16	33	34
Akron, Ohio	88	63	18	3	1	3	-	Albuquerque, N.M	ex. 89	60	18	5	2	4	4
Canton, Ohio	41	34	6	-	1	-	4	Colo. Springs, Col	o. 37	23	8	3	1	2	2
Cincigo, III Cincinnati Ohio	176	121	40	50	21	35	21	Las Vegas Nev	123	85	20	8	5	4	8
Cleveland, Ohio	134	79	39	12	-	4	2	Ogden, Utah	15	14	19	4	3	3	
Columbus, Ohio	131	83	29	9	7	3	4	Phoenix, Ariz.	187	121	38	15	3	10	7
Dayton, Ohio	107	66	35	5	-	1	4	Pueblo, Colo.	27	19	6	1	-	1	5
Evansville Ind	61	48	12	27	9	1/		Tucson Ariz	1h 56	33	13	1	1	8	-
Fort Wayne, Ind.	63	40	19	-	-	4	6			05	21	2	'		5
Gary, Ind.	22	8	3	2	9	-	-	PACIFIC	1,901	1,278	383	133	53	53	134
Grand Hapids, Mit	49	29	15	3	1	1	-	Berkeley, Calif.	11	9	1	-	1	-	
Madison, Wis.	33	20	9	1	3	<u>'</u>	3	Glendale, Calif.	26	17	28	2		2	1
Milwaukee, Wis	152	116	25	5	1	5	7	Honolulu, Hawaii	72	48	18	i	1	4	2
Peoria, III.	43	29	5	3	3	3	3	Long Beach, Calif.	102	70	22	6		4	5
South Bend, Ind.	66	43 53	8	4	i	1	1	Oakland Calif	. 483	310	111	42	12	8	25
Toledo, Ohio	150	103	32	7	5	3	ż	Pasadena, Calif.	36	26	6	1	1	2	4
Youngstown, Ohi	62	37	16	4	4	1	1	Portland, Oreg. Sacramento, Calif	145	117	13	8	3	4	16
W.N. CENTRAL	796	558	162	32	15	29	38	San Diego, Calif.	143	98	29	3	3	5	4 19
Des Moines, lowa	80	49	26	4	-	1	5	San Francisco, Cal	lit. 180	122	25	23	6	4	1
Kansas City, Kans	45	34	7	2	1	1	4	Seattle, Wash	1/8	122	39	16	4 5	2	21
Kansas City, Mo.	123	78	26	8	ż	9	9	Spokane, Wash	63	41	18	2	1	1	3
Lincoln, Nebr.	33	25	6	1	-	1	1	Tacoma, Wash.	38	27	8	1	1	1	3
Minneapolis, Mini	78	55 76	15	3	3	2	1	τοται	13 70E tt	9.057	2 0 7 0	004	255	400	700
St. Louis. Mo.	147	105	29	4	5	4	5 6		13,705	9,057	∡,979	884	355	423	/28
St. Paul, Minn.	69	51	11	4	ĩ	2	-								
Wichita, Kans.	76	56	11	3	2	4	7								

\* Mortality data in this table are voluntarily reported from 121 cities in the United States, most of which have populations of 100,000 or more. A death is reported by the place of its occurrence and by the week that the death certificate was filed. Fetal deaths are not included.

\*\* Pneumonia and influenza

Figure 1 Because of changes in reporting methods in these 4 Pennsylvania cities, these numbers are partial counts for the current week. Complete counts will be available in 4 to 6 weeks.

tt Total includes unknown ages.

§ Data not available. Figures are estimates based on average of past 4 weeks.

# Ampicillin and Chloramphenicol Resistance in Systemic *Haemophilus influenzae* Disease

In late August 1983, a 19-month-old girl was transferred from the Dominican Republic to a hospital in Houston, Texas, with a diagnosis of relapsing *Haemophilus influenzae* type b (Hib) meningitis.

Her initial cerebrospinal fluid (CSF) examination in the Dominican Republic contained 800 white blood cells (WBC), predominantly polymorphonuclear leukocytes, and a glucose concentration of 7 mg/dl. She was treated with ampicillin and chloramphenicol for 72 hours and then changed to chloramphenicol alone for 9 more days after the initial Hib isolate was demonstrated to be  $\beta$ -lactamase positive.

At the end of a 12-day course of antibiotic treatment, the patient was reported well and afebrile. Lumbar puncture after completion of treatment was sterile, showed five lymphocytes, and reportedly had normal glucose and protein values.

Three days later, she developed vomiting and fever up to 40.5 C (105 F). She was again started on chloramphenicol and was given three doses of ceftazidime before arrival in Houston. Her CFS at this time had 300 WBC/mm<sup>3</sup>, with 39% polys, a glucose of 50 mg/dl, and a protein concentration of 52 mg/dl. CSF culture was sterile, but counterimmunoelectrophoresis (CIE) was positive for Hib polyribosylribitol phosphate (PRP) antigen. She received a 12-day course of moxalactam (200 mg/kg/day), remained afebrile from the second day, and had normal neurologic examinations throughout hospitalization. At completion of therapy, the CSF was sterile and CIE-negative and contained 99 WBC, 2% polys, 98% monocytes, protein 22 mg/dl, and glucose 39 mg/dl. The original CSF isolate from the Dominican Republic was confirmed as *H. influenzae* type b,  $\beta$ -lactamase positive. On testing in Houston, the organism had a minimum inhibitory concentration (MIC) of 12.5  $\mu$ g/ml and a minimum bactericidal concentration (MBC) of 25  $\mu$ g/ml to chloramphenicol and produced chloramphenicol acetyltransferase. The MIC and MBC to moxalactam were both 0.016  $\mu$ g/ml.

Reported by JN Walterspiel, MD, SL Kaplan, MD, Baylor College of Medicine, MJ Kessler, MD, Houston, Texas; LF Reid, MD, Clinica Gomez Patiño, Santo Domingo, Dominican Republic; Respiratory and Special Pathogens Epidemiology Br, Div of Bacterial Diseases, Center for Infectious Diseases, CDC.

**Editorial Note:** Resistance of *H. influenzae* strains to ampicillin or chloramphenicol, conventional antimicrobial therapy for systemic (bacteremic) *H. influenzae* disease, is of growing concern among medical practitioners. An estimated 15,000-20,000 cases of systemic disease caused by *H. influenzae*, including meningitis, sepsis, pneumonia, epiglottitis, cellulitis, septic arthritis, and osteomyelitis, occur annually in the United States. Most such systemic infections occur among children under 5 years of age and are caused by serotype b organisms.

Since ampicillin-resistant isolates of Hib were first recognized in 1974, resistant strains have become increasingly prevalent. In 1975-1976, a national survey of pediatric centers in the United States found the prevalence of ampicillin-resistant *H. influenzae* isolates from cultures of blood and CSF was 4.5% (1). Since then, several reports have documented a steady trend of increasing prevalence of ampicillin resistance (2-3). By 1980-1981, ampicillin resistance rates of 17% to 28% were reported (2-5). Nationwide data on this trend are available for 1978-1982 from CDC's passive surveillance system for bacterial meningitis maintained in collaboration with the Conference of State and Territorial Epidemiologists (Table 2). These data are based on hospital reporting of resistance from 20 states that (1) participated in all years from 1978 to 1982, (2) reported ampicillin-testing results on at least 50 isolates, and (3) had over 75% of all isolates from reported cases tested for ampicillin susceptibility. Ampicillin resistance among HI isolates from reported cases of bacterial meningitis varied from

H. influenzae - Continued

19% in 1978 to 24% in 1981-1982. Geographic differences emerged when the frequency of resistance was analyzed by individual reporting states (Figure 2).

The primary mechanism of ampicillin resistance is by production of the TEM  $\beta$ -lactamase enzyme, mediated by a plasmid that contains a gene coding for this enzyme. Similar plasmids mediating resistance to penicillin and ampicillin have been found in Enterobacteriaceae and in *Neisseria gonorrhoeae* strains, and such genes are transferable between species.

Resistance of Hib strains to chloramphenicol has remained at a low prevalence rate of under 1% since the first report appeared in 1972 (6). Most chloramphenicol-resistant (MIC

Year	Total number tested	Ampicillin resistant (%)
1978	1,166	19.2%
1979	1,171	19.1%
1980	1,416	19.6%
1981	1,418	24.0%
1982	1,552	23.3%
Total	6,723	21.2%

 
 TABLE 2. Ampicilin-resistant Haemophilus influenzae isolates from patients with bacterial meningitis — United States, 1978-1982

FIGURE 2. Percentage of *Haemophilus influenzae* strains from cerebrospinal fluid reported as resistant to ampicillin, by participating states — United States, 1978-1982



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### H. influenzae – *Continued*

greater than 2  $\mu$ g/ml) strains produce chloramphenicol acetyltransferase, an enzyme capable of inactivating chloramphenicol, and resistance is often plasmid-mediated (7).

Resistance of Hib strains to both ampicillin and chloramphenicol, first reported in 1980 (8-10) and seen in the case described above, is rare. Presently, there are no indications that any change in the initial therapy for suspected systemic disease caused by *H. influenzae*— ampicillin and chloramphenicol—is warranted. However, the substantial rise in prevalence of ampicillin resistance in the past decade, and similarities in mechanisms mediating ampicillin and chloramphenicol resistance, suggest the possibility that chloramphenicol resistance alone or combined with ampicillin resistance could emerge as a more prevalent problem. Through the existing meningitis surveillance system, CDC plans to begin monitoring reported cases of *H. influenzae* meningitis for the prevalence of chloramphenicol resistance. *References* 

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# Contact Spread of Vaccinia from a Recently Vaccinated Marine — Louisiana

On December 27, 1983, a 20-year-old woman was admitted to a suburban New Orleans hospital with vesicular lesions on her face, thighs, buttocks, and labia. The patient had noted labial and inguinal pain and swelling beginning on December 24, and on December 25, clear vesicles on her face, thighs, and buttocks.

On admission, the patient had a fever of 38.0 C (100.4 F) and seven lesions ranging from clear vesicles to pustular-umbilicated lesions on her nose and chin and along her left cheek and malar areas. There was tender, firm submental, submandibular, and anterior cervical lymphadenopathy on the left. Additional lesions were noted on the right thigh and left buttock. A pelvic examination showed similar lesions on her labia majora and minora and

### Vaccinia — Continued

posterior fourchette area and along the right lateral vaginal wall and anterior cervix. Marked inguinal and femoral lymphadenopathy was present. On December 28, pox virus was confirmed by electron microscope, and the patient received 0.3 ml/kg of vaccinia immune globulin (VIG).

On December 29, the patient developed a new clear vesicular lesion on the right wrist and a satellite lesion adjacent to a vesicle at the angle of the mandible. A second dose of VIG was given; by December 30, these lesions had not developed further. The size and pain of the mandibular nodes decreased, and the patient noted a marked decrease in pain. Her remaining hospital course was uneventful.

The patient's fiancé, an officer in the U.S. Marine Corps, had received his first smallpox vaccination while completing a training session on December 16, 1983. He went on leave on December 17, and he and the patient spent December 17-25 together. The patient had never received a smallpox vaccination.

Cultures for herpes simplex 2 and serologic studies for chlamydia and herpes 2 were negative. Vaccinia virus was cultured from the patient's vaginal and facial lesions. No other cases among her large extended family occurred.

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**Editorial Note:** This episode resembles a 1981 vaccinia outbreak in Canada (1) and demonstrates the potential for contact spread of vaccinia from recently vaccinated persons. It is unusual because of the culture-proven intravaginal lesions.

The Department of Defense routinely vaccinates all active duty, National Guard, and Reserve personnel against smallpox on entry into service and at 5-year intervals. In general, these vaccinations are performed at the start of basic training, summer camps, or other settings that minimize contact between recently vaccinated military personnel and the general public. The present case illustrates the potential for serious complications by accidental spread of vaccinia to unvaccinated individuals. Accidental spread to individuals at high risk, such as children under 1 year of age and individuals with eczema or immunodeficiencies, could be more serious.

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### Current Trends

# Influenza Outbreaks — District of Columbia, Iowa, Louisiana, New York City, North Carolina

Influenza type A(H1N1) has been isolated from patients in outbreaks that began in January in the District of Columbia, Iowa, Louisiana, New York City, and North Carolina. In the District of Columbia, 50 of the 400 students at a private high school reported influenza-like illnesses between January 3 and January 17. In Iowa, 100 of the 400 children in a Cedar Rapids grade school were absent with influenza-like illnesses when an outbreak there peaked in mid-January. The outbreak confirmed in Louisiana (the second in January) occurred in one unit of a state institution for the mentally retarded. Nineteen of the 22 residents of the unit (total institutional population about 500) had influenza-like illnesses between January 11 and January 14. Two other laboratory-confirmed outbreaks occurred in penal institutions in New York City

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# Influenza Outbreaks - Continued

and North Carolina. In New York City, 120 of 2,500 inmates had influenza-like illnesses beginning the second week of January; in North Carolina, 12 of the 120 young adult inmates developed influenza-like illness between January 3 and January 6.

Nationwide, reports continue to be received of school and college influenza outbreaks, many beginning since January 9, with high rates of absenteeism and clinic visits. Laboratory diagnoses are pending on many of these.

Three states, Hawaii, Maine, and Arizona, reported their first influenza isolations for the season. In Hawaii, three isolations of type A(H1N1) virus were obtained from children who had influenza in December, and two isolations of influenza type B virus were obtained from a child and a young adult who had influenza early in January. In Maine, an isolation of type A(H1N1) influenza virus was obtained from a young woman who developed influenza on January 7. In Arizona, an isolation of type A(H3N2) influenza virus was obtained from a 16-year-old who developed influenza on January 15. From Colorado and Nevada, where influenza type A(H1N1) viruses have been isolated earlier this season, sporadic isolations of influenza virus type B in January have been reported.

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The Morbidity and Mortality Week/y Report is prepared by the Centers for Disease Control, Atlanta, Georgia, and available on a paid subscription basis from the Superintendent of Documents, U.S. Government Printing Office, Washington, D.C. 20402, (202) 783-3238.

The data in this report 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 succeeding Friday.

The editor welcomes accounts of interesting cases, outbreaks, environmental hazards, or other public health problems of current interest to health officials. Such reports and any other matters pertaining to editorial or other textual considerations should be addressed to: ATTN: Editor, *Morbidity and Mortality Week/y Report*, Centers for Disease Control, Atlanta, Georgia 30333.

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\*U.S. Government Printing Office: 1984-746-149/2017B Region IV

DEPARTMENT OF HEALTH & HUMAN SERVICES Public Health Service Centers for Disease Control Atlanta GA 30333

Official Business Penalty for Private Use \$300



Postage and Fees Paid U.S. Dept. of H.H.S. HHS 396

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