

Weekly

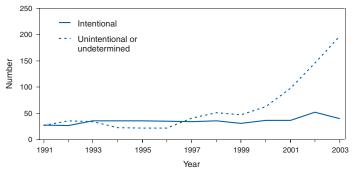
January 21, 2005 / Vol. 54 / No. 2

Increase in Poisoning Deaths Caused by Non-Illicit Drugs — Utah, 1991–2003

Deaths caused by drug poisoning of unintentional and undetermined intent are an increasing problem in Utah and elsewhere in the United States (1). To characterize the trend in drug-poisoning deaths in Utah, CDC and the Utah Department of Health analyzed medical examiner (ME) data for 1991–1998 and 1999–2003. This report summarizes the results of that analysis, which determined that, during 1991-2003, the number of Utah residents dying from all drug poisoning increased nearly fivefold, from 79 deaths in 1991 (rate: 4.4 per 100,000 population) to 391 deaths in 2003 (rate: 16.6). This increase has been largely the result of the tripling of the rate (from 1.5 during 1991-1998 to 4.4 during 1999-2003) in poisoning deaths of unintentional or undetermined intent caused by non-illicit drugs (i.e., medications that can be legally prescribed) (Figure). Further study is needed to understand these trends and to develop strategies to prevent deaths of unintentional or undetermined intent from non-illicit drug poisoning.

Utah has a centralized statewide ME system with statutespecified jurisdiction that includes drug-related deaths. The ME database used for these analyses contains decedent demographics; data on the circumstances, causes, and manner of death; examination results; and laboratory findings (2). A drugpoisoning death was defined as the death of a Utah resident with drug poisoning listed as cause of death. Deaths were identified by searching the ME database for a drug-poisoningrelated keyword (e.g., drug, overdose, poisoning, toxicity, or intoxication). Deaths identified by that search were each reviewed to verify that they met the case definition. Each death was classified as related to illicit drugs only, to non-illicit drugs only, or to both illicit and non-illicit drugs. Each death was also classified as 1) intentional (i.e., suicide or homicide) or 2) unintentional (e.g., nonsuicidal, nonhomicidal, or natural deaths) or undetermined (i.e., cause unknown). Decedent

FIGURE. Number of non-illicit drug-poisoning deaths, by intent and year — Utah, 1991–2003



characteristics, annual numbers and rates of drug-poisoning deaths, and trends in drug-poisoning deaths were analyzed.

Death rates were calculated by using denominators from the Utah Population Estimate Query System (3). To examine a possible association between overweight or obesity and drugpoisoning death, which had been noted anecdotally by Utah MEs, decedents were categorized based on body mass index (BMI) (4). For analysis of this association, population estimates were based on results from the Utah Behavioral Risk Factor Surveillance System (Unpublished data, 2003). To examine the effect of urban versus rural residence, rates were

INSIDE

- 36 Unintentional Non–Fire-Related Carbon Monoxide Exposures — United States, 2001–2003
- 40 Escherichia coli O157:H7 Infections Associated with Ground Beef from a U.S. Military Installation — Okinawa, Japan, February 2004
- 42 Elevated Blood Lead Levels in Refugee Children New Hampshire, 2003–2004
- 46 QuickStats
- 47 Notices to Readers

DEPARTMENT OF HEALTH AND HUMAN SERVICES CENTERS FOR DISEASE CONTROL AND PREVENTION The *MMWR* series of publications is published by the Coordinating Center for Health Information and Service*, Centers for Disease Control and Prevention (CDC), U.S. Department of Health and Human Services, Atlanta, GA 30333.

SUGGESTED CITATION

Centers for Disease Control and Prevention. [Article Title]. MMWR 2005;54:[inclusive page numbers].

Centers for Disease Control and Prevention

Julie L. Gerberding, MD, MPH Director

Dixie E. Snider, MD, MPH Chief of Science

Tanja Popovic, MD, PhD (Acting) Associate Director for Science

Coordinating Center for Health Information and Service*

Blake Caldwell, MD, MPH, and Edward J. Sondik, PhD (Acting) Directors

National Center for Health Marketing*

Steven L. Solomon, MD (Acting) Director

Division of Scientific Communications*

John W. Ward, MD (Acting) Director Editor, MMWR Series

Suzanne M. Hewitt, MPA Managing Editor, MMWR Series

Douglas W. Weatherwax (Acting) Lead Technical Writer-Editor

> Stephanie M. Neitzel Jude C. Rutledge Teresa F. Rutledge *Writers-Editors*

Lynda G. Cupell Malbea A. LaPete Visual Information Specialists

Kim L. Bright, MBA Quang M. Doan, MBA Erica R. Shaver Information Technology Specialists

Notifiable Disease Morbidity and 122 Cities Mortality Data

Patsy A. Hall Deborah A. Adams Felicia J. Connor Rosaline Dhara Donna Edwards Mechelle Hester Tambra McGee Pearl C. Sharp

* Proposed.

calculated separately for four urban counties (Davis, Weber, Salt Lake, and Utah counties) that contain approximately 75% of the Utah population, and for the remaining counties in the state, which were classified as rural (*3*).

During 1991–2003, a total of 2,396 drug-poisoning deaths were identified, of which 947 were caused by illicit drugs only, 1,277 by non-illicit drugs only, and 172 by a combination of illicit and non-illicit drugs. Alcohol was also implicated in 22% of drug-poisoning deaths; however, alcohol was not considered a drug for these analyses. The largest increase in annual drug-poisoning deaths (from 55 in 1991 to 237 in 2003) was attributed to non-illicit drugs. Illicit drug-poisoning deaths increased each year during 1991–1998 and then decreased to 92 deaths in 2003. Deaths resulting from a combination of illicit and non-illicit drugs increased gradually during 1991–2002, then increased substantially, from 15 in 2002 to 35 in 2003.

Among deaths attributed to non-illicit drugs, during 1991– 2003, a total of 733 were classified as of unintentional or undetermined intent; because these deaths had increased substantially since 1999, they were examined for the periods 1991– 1998 and 1999–2003. Further analyses focused on possible associations of selected characteristics of the decedents and the drug types involved in their deaths.

Death rates varied by age group and were highest for adults aged 25-54 years. Comparing cumulative 1991-1998 data with those for 1999-2003, the greatest numeric increase in deaths (from 42 to 142) occurred among adults aged 45-54 years (Table 1). Death rates per 100,000 population were higher for men than women during both periods (men: 1.86 and 4.90; women: 1.08 and 3.90), but the percentage increase in rates from 1991–1998 to 1999–2003 was greater for women than men (261% versus 163%). More deaths occurred in urban areas than rural areas during both periods (186 versus 45, during 1991-1998; 362 versus 140, during 1999–2003); however, the increase in death rate from 1991– 1998 to 1999-2003 was greater in rural areas than urban areas (317% versus 171%). In addition, although substantial increases in death rates occurred from 1991-1998 to 1999-2003 in each BMI category, rates were substantially higher during 1999–2003 among persons who were overweight (5.26 per 100,000 population) or obese (14.25), compared with persons who were not overweight or obese (3.61) (Table 1).

Methadone and other prescription narcotics accounted for most of the increase from 1991–1998 to 1999–2003 in nonillicit drug-poisoning deaths of unintentional or undetermined intent. Comparing these periods, deaths attributable to methadone increased from two to 33 per year, and deaths attributable to other prescription narcotics (principally oxycodone and hydrocodone) increased from 10 to 48 per year (Table 2).

			No. of c	leaths		Death rate				
	1991-1998		1999–	2003				% change in		
Characteristic	No.	(%)	No.	(%)	Difference	1991–1998	1999–2003	death rate		
Total deaths	231		502		271	1.47	4.40	200		
Median deaths per year	30		87		74					
Range of annual deaths	19–41		45–181							
Mean age (yrs)	40.9		40.3							
Age group at death (yrs)										
<25	12	(5)	45	(9)	33	0.16	0.86	438		
25–34	61	(26)	109	(22)	48	2.53	6.35	151		
35–44	86	(37)	159	(32)	73	3.90	10.51	170		
45–54	42	(18)	142	(28)	100	2.91	11.41	292		
55–64	14	(6)	39	(8)	25	1.49	5.21	250		
≥65	16	(7)	7	(1)	-9	1.18	0.73	-38		
Female	85	(37)	222	(44)	137	1.08	3.90	261		
Male	146	(63)	280	(56)	134	1.86	4.90	163		
Urban resident	186	(80)	362	(72)	176	1.53	4.15	171		
Rural resident	45	(19)	140	(28)	95	1.25	5.21	317		
BMI§										
<25.0	65	(31)	130	(27)	65	1.17	3.61	208		
25.0–29.9	65	(31)	143	(30)	78	1.90	5.26	177		
≥30.0	81	(38)	207	(43)	126	6.06	14.25	135		

TABLE 1. Number* and rate[†] of deaths from non-illicit drug poisoning of unintentional or undetermined intent, by selected characteristics --- Utah, 1991-1998 and 1999-2003

* N = 733.

[†]Per 100,000 population, [§]Body mass index (kg/m²).

TABLE 2. Number* and percentage of deaths from non-illicit drug poisoning of unintentional or undetermined intent, by drug
category, drug, and involvement of alcohol — Utah, 1991–1998 and 1999–2003

_	1	991–1998			1999–2003		Difference	% change	
Drug category	No. of deaths No. of deaths No. of deaths No. of deaths No. of deaths $(n = 231)^{\dagger}$ per year (%) [§] $(n = 502)^{\dagger}$ per year (%) [§] 18 2.3 (7.8) 164 32.7 (32.7) 34 4.3 (14.7) 33 6.6 (6.6)	in no. of deaths per year	in no. of deaths per year						
Methadone	18	2.3	(7.8)	164	32.7	(32.7)	31	1,358	
Antidepressants	34	4.3	(14.7)	33	6.6	(6.6)	2	55	
Prescription narcotics other than methadone	79	9.9	(34.2)	239	47.6	(47.6)	38	384	
Propoxyphene	23	2.9	(10.0)	13	2.6	(2.6)	0	-10	
Hydrocodone	31	3.9	(13.4)	83	16.5	(16.6)	13	328	
Oxycodone	10	1.3	(4. <i>3</i>)	111	22.1	(22.2)	21	1,676	
Codeine	15	1.9	(6.5)	21	4.2	(4.2)	2	124	
Fentanyl	2	0.3	(0.9)	27	5.4	(5.4)	5	2,060	
Alcohol involved	76	9.5	(32.9)	100	29.9	(19.9)	11	111	

* N = 733.

More than one drug could be listed as contributing to each death, so the sum of deaths attributed to specific drugs exceeds the total number of deaths. [§]Percentage of deaths attributed to a drug category.

From 1991-1998 to 1999-2003, the proportions of these deaths that involved alcohol or antidepressants decreased from 32.9% and 14.7%, respectively, to 19.9% and 6.6% (Table 2).

Reported by: EM Caravati, MD, Utah Poison Control Center, Salt Lake City; T Grey, MD, B Nangle, PhD, RT Rolfs, MD, Utah Dept of Health. CA Peterson-Porucznik, PhD, EIS Officer, CDC.

Editorial Note: The findings in this report indicate that deaths attributed to drug poisoning have increased in Utah for more than a decade; however, the characteristics of these deaths have changed since 1999, when deaths caused by non-illicit drugs began to increase substantially. In 2003, the typical drugpoisoning decedent in Utah was overweight or obese, aged 25-54 years, had died from the effects of non-illicit drugs, and was less likely than previously to be male and to live in an urban area.

The findings in this report are subject to at least three limitations. First, analysis was limited to deaths investigated by the Utah State Office of the Medical Examiner. Although this office has jurisdiction over all deaths thought to be drugrelated, some drug-poisoning deaths might not have been properly reported and, therefore, might have been excluded from analysis. Second, BMI values for the decedents were based on measurements made by the ME. The measured body weight at postmortem examination might have been less than the decedent's usual body weight when alive. In addition, the denominator used for death rate calculations was based on self-reported data from a telephone survey in which respondents might underreport weight. The combined effects of these two potential biases are uncertain. Finally, whether being overweight or obese is a risk factor for fatal drug poisoning or the result of greater use of these drugs by overweight persons cannot be determined from the data.

The Drug Enforcement Administration collects information regarding the movement of controlled substances from manufacture through commercial distribution channels by using the Automation of Reports and Consolidated Orders System (ARCOS) (5). From 1997 to 2002, the amount of drugs distributed to Utah and the United States (in grams per 100,000 population) increased substantially for several of the prescription drugs described in this report, including methadone (Utah: from 269 g to 1,703 g; United States: 194 g to 954 g), oxycodone (Utah: 1,848 g to 9,804 g; United States: 1,668 g to 8,056 g), and hydrocodone (Utah: 4,754 g to 8,122 g; United States: 3,249 g to 6,777 g). The numbers of drug-poisoning deaths attributed to each of these drugs increased at a greater rate than the supplies of the drugs in Utah. In addition, from 1997 to 2002, the codeine supply declined (Utah: from 7,746 g to 5,179 g; United States: 9,396 g to 8,149 g), possibly suggesting a prescription preference for newer pain-relieving drugs.

The sixfold increase in the methadone supply in Utah and fivefold increase in the United States were not the result of expansion of addiction treatment programs; ARCOS does not track drugs distributed through such programs. Methadone is also used to control pain and can be prescribed by physicians for pain management. Review of ME investigations into methadone deaths during 1996–2000 revealed previous methadone prescriptions for 48% (17 of 35) of decedents. A valid methadone prescription at time of death was found for 40% (14 of 35) of decedents. Of those with a valid prescription, seven (50%) were taking methadone for the first time (range: zero to 17 previous prescriptions) when they died.

Sources of decedents' drugs cannot always be determined from ME data. The narcotics associated with a drugpoisoning death might have been prescribed for pain, acquired illegally, or (in the case of methadone) obtained from an addiction treatment program. Further research is needed to investigate the proportion of deaths that occurred among legitimate users of prescription medications, and to identify risk factors that might increase the likelihood of drugpoisoning deaths for patients using prescription medications. Other state health departments that track drug-poisoning deaths should conduct their own analyses of unintentional or undetermined drug-poisoning deaths caused by non-illicit drugs. Steps should be taken to ensure safe use of non-illicit, pain-relieving medications while more information regarding factors contributing to deaths is collected. Such steps should include increased education for both health-care providers and their patients.

References

- 1. CDC. Unintentional and undetermined poisoning deaths—11 states, 1990–2001. MMWR 2004;53:233–8.
- Utah Code and Constitution, Utah Health Code. Utah Medical Examiner Act. Title 26, Chapter 04; updated 2004. Available at http:// www.le.state.ut.us/-code/title26/26_04.htm.
- 3. Governor's Office of Planning and Budget. Utah Population Estimate Query System; 2004. Available at http://health.utah.gov/ibisq/population/entry.html.
- National Heart, Lung, and Blood Institute. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults: the evidence report. Bethesda, MD: National Institutes of Health; 1998. NIH publication no. 98-4083.
- US Department of Justice, Drug Enforcement Administration. Automation of Reports and Consolidated Orders System. Retail drug summary reports; 1997–2002. Available at http://www.deadiversion. usdoj.gov/arcos/retail_drug_summary/index.html.

Unintentional Non–Fire-Related Carbon Monoxide Exposures — United States, 2001–2003

Carbon monoxide (CO) is a colorless, odorless, poisonous gas that results from incomplete combustion of fuels (e.g., natural or liquefied petroleum gas, oil, wood, coal, or other fuels). CO sources (e.g., furnaces, generators, gas heaters, and motor vehicles) are common in homes or work environments and can put persons at risk for CO exposure and poisoning. Most signs and symptoms of CO exposure are nonspecific (e.g., headache or nausea) and can be mistakenly attributed to other causes, such as viral illnesses. Undetected or unsuspected CO exposure can result in death (1). To examine fatal and nonfatal unintentional, non-fire-related CO exposures, CDC analyzed 2001-2003 data on emergency department (ED) visits from the National Electronic Injury Surveillance System All Injury Program (NEISS-AIP) and 2001-2002 death certificate data from the National Vital Statistics System (NVSS). During 2001-2003, an estimated 15,200 persons with confirmed or possible non-fire-related CO exposure were treated annually in hospital EDs. In addition, during 2001-2002, an average of 480 persons died annually from

non-fire-related CO poisoning. Although males and females were equally likely to visit an ED for CO exposure, males were 2.3 times more likely to die from CO poisoning. Most (64%) of the nonfatal CO exposures occurred in homes. Efforts are needed to educate the public about preventing CO exposure.

NEISS-AIP is operated by the U.S. Consumer Product Safety Commission and collects data regarding initial ED visits for all types and causes of injuries (2). Data are drawn from a nationally representative subsample of 66 of 100 NEISS hospitals that were selected as a stratified probability sample of hospitals in the United States and its territories. NEISS-AIP provides data on approximately 500,000 injury-related and consumer-product-related ED cases each year.

Nonfatal cases were defined as those recorded at an NEISS-AIP hospital as CO exposure or CO poisoning. An incident was identified as a case if 1) the intent of injury was unintentional or undetermined, 2) the principal diagnosis by a physician was "poisoning" or "anoxia," and 3) the consumer product indicated was "CO detector" or "CO poisoning (source unknown)" or a brief narrative abstracted from the medical record indicated either CO exposure or CO poisoning. Firerelated (i.e., burn and smoke inhalation) cases were excluded. In addition, because death data are not captured completely by NEISS-AIP, persons who were dead on arrival or who died in the ED also were excluded. Data for all cases were reviewed independently by two CDC epidemiologists to confirm they met the case criteria. Narratives were also reviewed to determine CO source, exposure status (on the basis of physician diagnosis), and symptoms reported.

Each case was assigned a sample weight on the basis of the inverse of the probability of selection; these weights were summed to provide national estimates of nonfatal CO exposures. Estimates were based on weighted data for 778 patients with confirmed or possible CO exposure treated at NEISS-AIP hospital EDs during 2001–2003. Three years of data were necessary to provide stable rates. Confidence intervals (CIs) were calculated by using a direct variance estimation procedure that accounted for the sample weights and complex sample design. Because CO source and symptoms were undetermined for a high percentage of cases, data on these factors were based on unweighted data for NEISS-AIP cases and thus are not nationally representative.

Death certificate data were obtained from NVSS (3). Using multiple-cause-of-death files from the National Center for Health Statistics (NCHS) (3), CO poisoning deaths were defined as those with any mention on the death certificate of *International Classification of Diseases, Tenth Revision* (ICD-10) code T58 ("Toxic effect of carbon monoxide") as a leading or contributing cause of death and an ICD-10 underlying-cause-of-death code of X47 ("Accidental poisoning by and exposure to other gases or vapors") or Y17 ("Poisoning by and exposure to other gases or vapors, undetermined intent"). NVSS is a complete census of all deaths and therefore is not subject to sampling error; however, CIs were calculated to account for random error (3). The casefatality rate (CFR) was calculated as the number of CO deaths divided by the sum of CO deaths and nonfatal CO exposures multiplied by 100. Rates were calculated by using 2001–2003 U.S. census bridged-race population estimates from NCHS (4).

During 2001-2003, an estimated 15,200 persons were treated annually in EDs for nonfatal, unintentional, non-firerelated CO exposure, and, during 2001-2002, an average of 480 persons died each year from unintentional, non-firerelated CO exposure (Table 1). The nonfatal rate for CO exposure was highest for children aged ≤4 years (8.2 per 100,000 population), whereas the CO death rate was highest for adults aged ≥ 65 years (0.32). Adults aged ≥ 65 years accounted for 23.5% of CO poisoning deaths. The nonfatal rate was similar for males and females; in contrast, the death rate for males was 2.7 times that for females. The CFR increased with age, from 0.6% for children aged ≤ 4 years to 5.5% for adults aged 55-64 years; also, the CFR for males was 2.3 times that for females. The death rate was highest for non-Hispanic whites and blacks (0.17 per 100,000). Eleven percent of those treated in EDs were either hospitalized or transferred to another hospital for specialized care.

The annualized incidence of fatal and nonfatal CO exposures occurred more often during the fall and winter months, with the highest numbers occurring during December (56 fatal and 2,157 nonfatal exposures) and January (69 fatal and 2,511 nonfatal exposures). The annualized incidence was substantially lower during the summer months, with 21 fatal and 510 nonfatal exposures occurring during June and 22 fatal and 524 nonfatal exposures occurring during July.

The majority (64.3%) of nonfatal CO exposures were reported to occur in homes; 21.4% occurred in public facilities and areas. Narratives abstracted from the medical records of NEISS-AIP cases indicated that 18.5% of CO exposure incidents were associated with faulty furnaces (Table 2). An additional 9% were associated with motor vehicles. CO poisonings were diagnosed in approximately half of the NEISS-AIP cases, of which 73% had symptoms noted in the medical record (Table 2). The most common symptoms experienced were headache (37.5%), dizziness (18.0%), and nausea (17.3%). Severer symptoms were reported less often, including loss of consciousness (7.7%), shortness of breath (6.7%), and loss of muscle control (3.5%). According to medical records, 9.3% of patients in the NEISS-AIP sample reported

		Nonfa	tal (200	1–03)*		Fatal	(2001–02))†			
Characteristic	Average no of exposure per year		Rate§	(95% CI¹)	Average no of deaths per year	0. S	Rate [§]	(95% CI)	CFR**		
Age group (yrs)											
0-4	1,596	(10.5)	8.15	(4.47–11.83)	9	(1.9)	0.05	(0.02-0.07)	0.56		
5–14	2,352	(15.5)	5.73	(3.67-7.80)	19	(4.0)	0.05	(0.03-0.06)	0.80		
15–24	2,478	(16.3)	6.11	(4.17-8.04)	58	(12.1)	0.14	(0.12-0.17)	2.29		
25–34	2,750	(18.1)	6.90	(4.69-9.11)	57	(11.9)	0.14	(0.12-0.17)	2.03		
35–44	2,358	(15.5)	5.26	(3.60-6.92)	92	(19.2)	0.20	(0.17–0.23)	3.76		
45–54	1,669	(11.0)	4.17	(2.56-5.78)	79	(16.5)	0.20	(0.17-0.23)	4.52		
55–64	918	(6.0)	3.45	(1.97-4.93)	53	(10.9)	0.20	(0.16-0.24)	5.46		
<u>≥</u> 65	1,079††	(7.1)††	—	_	113	(23.5)	0.32	(0.20-0.36)	_		
Sex											
Male	7,874	(51.8)	5.56	(4.00-7.12)	344	(71.6)	0.24	(0.23–0.26)	4.19		
Female	7,326	(48.2)	5.00	(3.40-6.59)	137	(28.4)	0.09	(0.08-0.10)	1.84		
Race/Ethnicity ^{§§}											
White, non-Hispanic	7,171	(47.2)	_		346	(72.1)	0.17	(0.16–0.19)	—		
Black	3,817	(25.1)	_	—	65	(13.5)	0.17	(0.14–0.20)	—		
Hispanic	690	(4.5)	—	_	51	(10.6)	0.14	(0.11–0.17)	_		
Other, non-Hispanic	135††	(0.9)††	_	_	18	(3.8)	0.12	(0.08-0.16)	—		
Unknown	3,387	(22.3)	_	—	—	_	—	—	—		
Disposition											
Treated and released	13,201	(86.8)	4.58	(3.35–5.81)	—	_	_	—	—		
Hospitalized/Transferred	1,676	(11.0)	0.58	(0.27-0.90)	—	_	_	—	—		
Other/Unknown	324††	(2.1)††	_	_	—	—	—	—	—		
Total	15,200	(100.0)	5.27	(3.83–6.72)	480	(100.0)	0.17	(0.16–0.18)	3.06		

TABLE 1. Estimated annual number, percentage, and rate of persons with nonfatal and fatal unintentional non-fire-related carbon monoxide (CO) exposures, by selected characteristics — United States, 2001–2003

* National estimate of persons with nonfatal CO exposure treated in hospital emergency departments, based on 778 cases reported by the National Electronic Injury Surveillance System All Injury Program (NEISS-AIP).

[†] Based on actual number of persons reported in death certificate data from the National Vital Statistics System.

§ Per 100,000 population.

[¶] Confidence interval.

** Case-fatality rate = annualized CO deaths / (annualized CO deaths + annualized nonfatal CO exposures) x 100.

⁺⁺ Estimates might be unstable because the coefficient of variation is >30% or the number of nonfatal NEISS-AIP cases was <20.

§§ Nonfatal rates and CFR are not presented for racial/ethnic groups because race/ethnicity was unknown for a substantial percentage of persons with nonfatal exposures. "Black" includes Hispanic and non-Hispanic blacks; "Hispanic" excludes black Hispanics.

that they had a CO detector at home, and 100% of those indicated that the detector had alerted them to the presence of CO.

Reported by: *M Vajani, MPH, JL Annest, PhD, Office of Statistics* and Programming; *M Ballesteros, PhD, J Gilchrist, MD, Div of Unintentional Injury Prevention, National Center for Injury Prevention* and Control; A Stock, PhD, Div of Environmental Hazards and Health Effects, National Center for Environmental Health, CDC.

Editorial Note: Data in this report indicate that, each year, approximately 15,000 U.S. residents visit EDs for unintentional, non-fire-related CO exposure and approximately 500 die from unintentional, non-fire-related CO poisoning. Primary CO sources were home appliances, and the majority of exposures occurred during the fall and winter months, when persons are more likely to use gas furnaces and heaters. During warmer months, boating activities might also be a source of exposure (5). This analysis also determined that males are more likely to die from CO poisoning than females, which is consistent with previous findings (6–8). Males might be

exposed to higher CO levels during high-risk activities, such as working indoors or in enclosed garages with combustionengine-driven tools (e.g., generators or power washers) (7). The CO poisoning death rate was highest among persons aged ≥ 65 years, likely attributable to their being at higher risk for undetected CO exposure because symptoms often resemble those associated with other health conditions common among older persons (9).

The findings in this report are subject to at least three limitations. First, data on sources of CO exposure and symptoms of persons with CO poisoning were missing for a substantial percentage of cases. Second, national estimates of nonfatal injuries are based solely on persons treated in EDs and do not include those treated in outpatient settings or not treated at all. Finally, although risks for CO exposure vary by state and locality (e.g., because of differences in winter weather conditions), NEISS-AIP provides only national estimates and not state or local estimates.

TABLE 2. Unweighted number* and percentage of nonfatal, unintentional, non-fire-related carbon monoxide (CO) exposures by source, exposure status, and symptom — United States, 2001–2003

Source/Exposure status/ Symptom	No.	(%)
CO source		
All sources	778	(100.0)
Furnace [†]	144	(18.5)
Motor vehicle [§]	71	(9.1)
Stove/Gas range	38	(4.9)
Gas line leak	38	(4.9)
Gas water heater	33	(4.2)
Generators	22	(2.8)
Space heater	15	(1.9)
Machinery [¶]	12	(1.5)
Other	72	(9.3)
Unknown	333	(42.8)
Exposure status		
All exposures	778	(100.0)
Possible exposure	47	(6.0)
CO exposure	326	(41.9)
CO poisoning	405	(52.1)
Symptom**		
Headache	152	(37.5)
Dizziness	73	(18.0)
Nausea	70	(17.3)
Weakness	39	(9.6)
Vomiting	31	(7.7)
Loss of consciousness	31	(7.7)
Shortness of breath	27	(6.7)
Light-headedness	20	(4.9)
Sleepiness	19	(4.7)
Loss of muscle control	14	(3.5)
Chest tightness	9	(2.2)
Confusion	4	(1.0)
Blurred vision	1	(0.3)
Other	38	(9.4)

* Based on 778 cases reported by the National Electronic Injury Surveillance System All Injury Program (NEISS-AIP).

[†] Includes oil, gas, and unspecified furnaces.

§ Includes cars, vans, sport utility vehicles, and trucks.

[¶] Includes tractors and forklifts.

** Symptoms reported for 297 of the 405 CO poisoning cases. No symptoms were reported for the remaining 108 cases. Multiple symptoms were often reported; therefore, categories are not mutually exclusive.

Primary prevention of residential CO exposure can be accomplished through simple precautions (Box). Although residential CO detectors are important for early detection of CO, they should be considered a secondary prevention method. High oil and gas prices and power outages during winter months can contribute to consumer use of improperly vented heating sources. Public education campaigns, especially during winter months, combined with provision of battery-operated CO detectors for low-income persons, might reduce CO poisonings (10). Previous studies also suggest a need for multilingual educational campaigns to reach non– English-speaking populations (10).

BOX. Guidelines to prevent carbon monoxide (CO) exposure

- Have your heating system, water heater, and any other gas-, oil-, or coal-burning appliances serviced by a quali-fied technician every year.
- Install a battery-operated CO detector in your home and check or replace the battery when you change the time on your clocks each spring and fall.
- If your CO detector sounds, evacuate your home immediately and telephone 911.
- Seek prompt medical attention if you suspect CO poisoning and are feeling dizzy, light-headed, or nauseated.
- Do not use a generator, charcoal grill, camp stove, or other gasoline- or charcoal-burning device inside your home, basement, or garage or near a window.
- Do not run a car or truck inside a garage attached to your house, even if you leave the door open.
- Do not burn anything in a stove or fireplace that is not vented.
- Do not heat your house with a gas oven.

Acknowledgments

This report is based on data contributed by T Schroeder, MS, C Irish, and other staff members, Div of Hazard and Injury Data Systems, US Consumer Product Safety Commission.

References

- Carlson SA. Non-fire carbon monoxide deaths associated with the use of consumer products. 2001 estimates. Bethesda, MD: US Consumer Product Safety Commission; 2004.
- 2. CDC. National estimates of nonfatal injuries treated in hospital emergency departments—United States, 2000. MMWR 2001;50:340–6.
- Kochanek KD, Murphy SL, Anderson RN, Scott C. Deaths: final data for 2002. Natl Vital Stat Rep 2004;53(5):1–115.
- National Center for Health Statistics. U.S. census populations with bridged-race categories. Hyattsville, MD: US Department of Health and Human Services, CDC, National Center for Health Statistics; 2004. Available at http://www.cdc.gov/nchs/about/major/dvs/ popbridge/popbridge.htm.
- 5. CDC. Carbon-monoxide poisoning resulting from exposure to skiboat exhaust—Georgia, June 2002. MMWR 2002;51:829–30.
- Mott JA, Wolfe MI, Alverson CJ, et al. National vehicle emissions policies and practices and declining US carbon monoxide-related mortality. JAMA 2002;288:988–95.
- US Consumer Product Safety Commission. Incident, deaths, and indepth investigations associated with carbon monoxide and enginedriven tools, 1990–2003. Memorandum. Bethesda, MD: US Consumer Product Safety Commission; March 8, 2004. Available at http:// www.cpsc.gov/LIBRARY/FOIA/FOIA04/os/epiedt.pdf.
- Cobb N, Etzel RA. Unintentional carbon monoxide-related deaths in the United States, 1979 through 1988. JAMA 1991;266:659–63.
- 9. Harper A, Croft-Baker J. Carbon monoxide poisoning: undetected by both patients and their doctors. Age Ageing 2004;33:105–9.
- CDC. Use of carbon monoxide alarms to prevent poisonings during a power outage—North Carolina, December 2002. MMWR 2004;53:189–92.

Escherichia coli O157:H7 Infections Associated with Ground Beef from a U.S. Military Installation — Okinawa, Japan, February 2004

In February 2004, the Okinawa Prefectural Chubu Health Center (OCHC) and the Okinawa Prefectural Institute of Health and Environment (OIHE), Japan, investigated three cases of Escherichia coli O157:H7 infection in a Japanese family associated with eating ground beef. Public health officials from multiple agencies in Japan and the United States collaborated on this investigation, which resulted in a voluntary recall of approximately 90,000 pounds of frozen ground beef in the United States and at U.S. military bases in the Far East. This was the first reported instance in which Japanese public health officials identified contaminated, commercially distributed ground beef that was produced in the United States. This report summarizes epidemiologic and laboratory investigations conducted by OCHC and OIHE. The results underscore the importance of using standardized molecular subtyping methods throughout the world to facilitate international public health communication and intervention.

Cases were ascertained through surveillance for laboratoryconfirmed *E. coli* O157:H7 infection. Laboratory investigation of implicated food items was conducted using methods recommended by the Japanese Ministry of Health, including culture of food samples, immunomagnetic separation, and polymerase chain reaction to characterize isolates. Pulsed-field gel electrophoresis (PFGE) of the genomic DNA fragments of *E. coli* O157:H7 isolates was performed after restriction with *Xba*I enzyme in accordance with the PulseNet protocol by the National Institute of Infectious Diseases, Japan. PFGE patterns were analyzed and transmitted electronically to PulseNet USA* at CDC for comparison with U.S. isolates.

On February 17, 2004, OCHC was notified of laboratoryconfirmed *E. coli* O157:H7 infection in a hospitalized child in Okinawa. The child had been hospitalized with bloody diarrhea and, 6 days previous, had other symptoms, including abdominal pain and fever. Interviews with the child's family revealed that a sibling appeared to have some of the same symptoms. Family members were also questioned about food history; all family members had eaten hamburgers on February 6. In addition to the hospitalized child, *E. coli* O157:H7 was isolated from the symptomatic sibling and one asymptomatic family member.

The frozen ground beef patties eaten by the family were purchased from a U.S. military commissary in Okinawa. OCHC obtained the remaining frozen ground beef patties from the family and sent a sample to OIHE for laboratory evaluation; E. coli O157:H7 was isolated from the ground beef patties. Epidemiologic and laboratory findings were reported by the Okinawa Prefecture to the U.S. Naval Hospital in Okinawa. To exclude the possibility that the patties were contaminated after opening, the U.S. Naval Hospital obtained unopened frozen ground beef patties with the same lot number from the base commissary for microbiologic analysis; E. coli O157:H7 was isolated from these previously unopened ground beef patties. Isolates from the unopened package, leftover ground beef patties, and the three human isolates had indistinguishable PFGE patterns. The pattern had not been previously observed in Japan or in the PulseNet USA database.

Results of the investigations indicated that the source of infections was contaminated ground beef patties obtained from the U.S. military base in Okinawa. Traceback of the lot number indicated that the frozen patties were produced on August 11, 2003, by a U.S. company. Fresh and frozen ground beef products produced on that day were distributed to U.S. military installations in the Far East and to institutional and retail outlets in California, Idaho, Oregon, and Washington.

As a result of this investigation, the Food Safety Inspection Service of the U.S. Department of Agriculture announced a voluntary recall by the company of approximately 90,000 pounds of frozen ground beef and other ground beef products (1). Identification of the contaminated lot and the subsequent recall likely prevented additional infections.

Reported by: J Kudaka, R Asato, K Itokazu, M Nakamura, DVM, K Taira, DVM, Okinawa Prefectural Institute of Health and Environment; H Kuniyosi, MD, Y Kinjo, Okinawa Prefectural Chubu Health Center, Okinawa; J Terajima, DVM, H Watanabe, MD, J Kobayashi, MD, Field Epidemiology Training Program, National Institute of Infectious Diseases, Tokyo, Japan. B Swaminathan, PhD, CR Braden, MD, Div of Bacterial and Mycotic Diseases, National Center for Infectious Diseases; JR Dunn, DVM, EIS Officer, CDC.

Editorial Note: *E. coli* O157:H7 infection is a major cause of foodborne illness in many countries, including the United States and Japan (2). In 1996, Japanese public health officials investigated the largest outbreak of *E. coli* O157:H7 infection, which was associated with consumption of radish sprouts, with approximately 6,000 persons becoming ill (3). The outbreak described in this report demonstrates the need to eliminate *E. coli* O157:H7 contamination of ground beef and the need for consumers to follow guidelines for safe food preparation (4). Moreover, this outbreak demonstrates the potential

^{*} The national molecular subtyping network for foodborne surveillance, available at http://www.cdc.gov/pulsenet.

for multinational foodborne outbreaks and the benefits of international public health communication and use of standardized methods of molecular subtyping for detection and prevention of foodborne diseases.

During the weeks after this investigation, three additional *E. coli* O157:H7 infections were identified as potentially associated with this outbreak, one in Japan and two in the United States. On February 27, a child aged 11 years of a U.S. military family in Okinawa was hospitalized with *E. coli* O157:H7 infection; the PFGE pattern was indistinguishable from that of the three infected persons described in this report. The family had purchased the same brand of frozen ground beef patties from the U.S. military commissary in Okinawa. The hamburgers were prepared and eaten on February 22, 2 days before the recall notice. Although the company name was the same, the lot number could not be confirmed because the family discarded the package after learning of the recall.

In the United States, two clinical E. coli O157:H7 isolates with the outbreak PFGE pattern were identified in a woman aged 40 years and a child aged 10 years in Orange County, California; both patients were hospitalized. Both patients had eaten beef during the week preceding their illness. Specimen collection dates were August 26, 2003, and September 8, 2003. No association with the recalled product was made, although the PFGE pattern was unique to California, and the cases were temporally related with respect to distribution of the recalled products to institutional and retail establishments in California. The 6-month lag between production in the United States and sale in Japan, with intervening cases in the United States, demonstrates the long life of products such as frozen ground meat and the prolonged survival of foodborne pathogens in frozen foods. This investigation also highlights the ability of PulseNet USA to identify small clusters of indistinguishable isolates and the potential for prevention, particularly if epidemiologic links can be made between ill persons and food items in a timely and coordinated manner.

The use of standardized protocols for molecular subtyping during international outbreaks of foodborne disease and the ability to communicate with international public health authorities have been important in previous outbreaks (5,6). The development of PulseNet USA has had an important impact on the investigation of foodborne outbreaks and public health in the United States. PFGE was used to characterize food and clinical isolates after a large outbreak of *E. coli* O157:H7 infections in 1993 (7). Subsequently, CDC standardized PFGE protocols, disseminated them to state and local public health partners, and began building the PulseNet USA network (8).

MMWR

(MMWR on line)

cdc.gov/mmwr



Use of the PulseNet USA protocols during the public health investigation by Japan led to an international recall of contaminated ground beef and enabled international comparison of isolates facilitating detection of presumptively associated *E. coli* O157:H7 infections in the United States. In collaboration with many partners, CDC has facilitated establishment of PulseNet International, which has launched networks in several regions of the world (9). The continued development of PulseNet International will enhance international collaboration in the investigation of foodborne diseases and outbreaks.

Acknowledgments

The findings in this report are based, in part, on contributions from M Irei, MD, Okinawa Prefectural Chubu Health Center; M Iwanaga, MD, Dept of Microbiology, Graduate School of Medicine, Univ of the Ryukyus; D Baker, M Sekine, U.S. Naval Hospital, Okinawa; Japan District Veterinary Office, Camp Zama, Japan. Food Safety Inspection Svc, U.S. Dept of Agriculture.

References

- US Department of Agriculture, Food Safety Inspection Service. California firm recalls ground beef products for possible *E. coli* O157:H7. Available at http://www.fsis.usda.gov/frame/frameredirect.asp?main=http:// www.fsis.usda.gov/oa/recalls/prelease/pr007-2004.htm.
- O'Brien AD, Kaper JB. *Escherichia coli* O157:H7 and other Shiga toxinproducing *E. coli* strains. Washington, DC: ASM Press; 1998.
- Michino H, Araki K, Minami S, et al. Massive outbreak of *Escherichia coli* O157:H7 infection in schoolchildren in Sakai City, Japan, associated with consumption of white radish sprouts. Am J Epidemiol 1999;150:787–96.
- 4. US Department of Agriculture, Food Safety Inspection Service. FSIS issues alert on the importance of cooking and handling ground beef. Available at http://www.fsis.usda.gov/oa/news/2004/alert012904.htm.
- Lindsay EA, Lawson AJ, Walker RA, et al. Role of electronic data exchange in an international outbreak caused by *Salmonella enterica* serotype Typhimurium DT204b. Emerg Infect Dis 2002;8:732–4.
- Mahon BE, Ponka A, Hall WN, et al. An international outbreak of Salmonella infections caused by alfalfa sprouts grown from contaminated seeds. J Infect Dis 1997;175:876–82.
- Barrett TJ, Lior H, Green JH, et al. Laboratory investigation of a multistate food-borne outbreak of *Escherichia coli* O157:H7 by using pulsedfield gel electrophoresis and phage typing. J Clin Microbiol 1994;32:3013–7.
- Swaminathan B, Barrett TJ, Hunter SB, Tauxe RV, CDC PulseNet Task Force. PulseNet: the molecular subtyping network for foodborne bacterial disease surveillance, United States. Emerg Infect Dis 2001;7:382–9.
- CDC. PulseNet International. Available at http://www.cdc.gov/pulsenet/ pulsenet_international.htm.

Elevated Blood Lead Levels in Refugee Children — New Hampshire, 2003–2004

As a result of reductions in lead hazards and improved screening practices, blood lead levels (BLLs) in children aged 1-5 years are decreasing in the United States. However, the risk for elevated BLLs ($\geq 10 \mu g/dL$) remains high for certain populations, including refugees (1,2). After the death of a Sudanese refugee child from lead poisoning in New Hampshire in 2000, the New Hampshire Department of Health and Human Services (NHDHHS) developed lead testing guidelines to screen and monitor refugee children (3). These guidelines recommend 1) capillary blood lead testing for refugee children aged 6 months-15 years within 3 months after arrival in New Hampshire, 2) follow-up venous testing of children aged <6 years within 3-6 months after initial screening, and 3) notation of refugee status on laboratory slips for first tests. In 2004, routine laboratory telephone reports of elevated BLLs to the New Hampshire Childhood Lead Poisoning Prevention Program (NHCLPPP) called attention to a pattern of elevated BLLs among refugee children. To develop prevention strategies, NHDHHS analyzed NHCLPPP and Manchester Health Department (MHD) data, focusing on the 37 African refugee children with elevated BLLs on follow-up for whom complete data were available. This report describes the results of that analysis, which indicated that 1) follow-up blood lead testing is useful to identify lead exposure that occurs after resettlement and 2) refugee children in New Hampshire older than those routinely tested might have elevated BLLs. Refugee children in all states should be tested for lead poisoning on arrival and several months after initial screening to assess exposure after resettlement.

Case Series

During October 1, 2003–September 30, 2004, a total of 242 refugee children, 238 (98%) of whom were African, resettled in New Hampshire; of these, 216 (89%) resettled in Manchester*. Of the 242 children, 32 had no lead test, 113 had a first but no follow-up test (17 overdue and 96 either not yet due or too old for follow-up), five had only elevated first tests (i.e., delayed because too young or extenuating circumstances), and 92 had two tests. A refugee child identified with

^{*} Most refugee families were resettled in Manchester because of the availability of affordable housing units that can accommodate larger families.

Vol. 54 / No. 2

MMWR

an elevated BLL received the same follow-up care as any other child with the same BLL. Different BLLs trigger different actions. A BLL \geq 15 µg/dL triggers a home visit during which a MHD staff administers parents questionnaires about their children's habits, diet, and potential sources of lead exposure, both inside and outside of the home. For children with BLLs \geq 20 µg/dL, NHCLPPP routinely performs environmental investigations to identify lead hazards in or around the child's home. Lead hazards are defined as surfaces with lead paint present and with at least one of the following properties: chipping or peeling paint, a chewable surface, or a surface that creates friction on impact (e.g., windows and doors), increasing the likelihood that dust is generated. Intact lead paint is not considered a lead hazard.

After noting a pattern of elevated BLLs among refugee children, NHDHHS and NHCLPPP tabulated existing home visit and environmental data on refugee children with elevated BLLs. In addition, MHD abstracted height and weight measurements recorded up to 1 year before immigration on International Office of Migration medical examination forms. A computerized anthroprometry module was used to calculate percentages of children falling below two standard deviations (-2 Z-scores) using growth reference curves for height-for-age (HAZ) and weight-for-height (WHZ). Concern for malnutrition in a population occurs when the prevalence of low HAZ, indicating growth retardation or stunting from chronic malnutrition or chronic illness, or low WHZ, indicating acute malnutrition or wasting, is substantially greater than the expected 2.3% of a population (4).

A total of 92 (38.0%) of the 242 refugee children had both initial and follow-up blood lead testing; of these, 13 (14.1%) had elevated BLLs at both initial screening and follow-up, 10 (10.9%) had elevated BLLs at initial screening but not at follow-up, 27 (29.3%) were not elevated at screening but were elevated at follow-up, and 42 (45.7%) were not elevated at either screening or follow-up. Forty children had elevated BLLs at follow-up. Three children, for whom data were incomplete, were excluded from this analysis; therefore, this report describes the 37 (40.2%) of the 92 children who had elevated BLLs on follow-up testing (Table).

All 37 children (from 19 families) were born in Africa and resettled in Manchester. Seventeen (46.0%) were Somalis; 21 (56.8%) were female. The prevalence for low HAZ was 35.1% (13 of 37) and for low WHZ was 21.6% (eight of 37), indicating chronic and acute malnutrition. No other data from before immigration were available to assess micronutrient sufficiency.

Median age at the time of follow-up testing was 4.9 years (range: 14 months–13 years). Median initial screening BLL was 8.1 μ g/dL (range: 2–28 μ g/dL), performed 7–77 days (median: 22 days) after arrival. Median follow-up BLL was 18.6 μ g/dL (range: 10–63 μ g/dL), performed 35–188 days (median: 89 days) after arrival. Follow-up BLLs increased for 35 of 37 children; the average increase was 11 μ g/dL (range: 1–59 μ g/dL), and 26 (70.2%) became elevated after the initial testing. Three children received chelation therapy for BLLs >45 μ g/dL.

Of the nine families who received home visits, eight had been placed in multi-unit rental properties constructed before 1978. Paint used in housing before 1978 can contain high levels of lead (5). Six families (66.7%) reported that their children exhibited one or more behaviors that could increase the chance of lead ingestion: frequently putting nonfood items in the mouth (five); picking at loose paint, plaster, or putty (five); and chewing on painted surfaces or items (four). Of eight apartments in which environmental investigations were performed, lead hazards were identified in seven.

Blood lead testing identified five additional refugee children with elevated BLLs, but data for these children were not included in this study because the children did not have both an initial and a follow-up blood lead test. For these five children, median age at time of blood lead test was 2.4 years (range: 11 months–4 years), and tests were performed 117–190 days after arrival in New Hampshire. Median BLL was 33.8 μ g/dL (range: 17–72 μ g/dL). One child, who had a BLL of 72 μ g/dL, received chelation therapy immediately.

Reported by: J Kellenberg, MPH, R DiPentima, MPH, M Maruyama, R Caron, PhD, C Campbell, MD, P Alexakos, MPH, S Gagnon, A Krycki, Manchester Health Dept; M Dembiec, MEd, L Speikers, M Tehan, MPH, Childhood Lead Poisoning Prevention Program; EA Talbot, MD, J Greenblatt, MD, L Bujno, MSN, W Kassler, MD, New Hampshire Dept of Health and Human Svcs. MJ Brown, ScD, T Dignam, MPH, C Thomas, MPA, Emergency and Environmental Health Svcs, National Center for Environmental Health; R Plotinsky, MD, EIS Officer, CDC.

Editorial Note: The findings in this report indicate that BLLs became elevated after resettlement for nearly 30% of refugee children with two tests, suggesting that lead exposure for these children occurred in the United States. Investigations revealed several risk factors for lead poisoning: living in old homes, the presence of lead hazards, behaviors that could increase the chance of ingesting lead, a lack of awareness of the dangers of lead, and evidence of chronic and acute malnutrition. Malnutrition is common in refugee populations (*6*); a December 2003 nutritional survey conducted in a refugee camp in Kenya,

			Initial capillary	Follow-up venous	Environ	Environmental investigation results [§]			
Family	Age(s)*	Country of emigration (Country of origin) [†]	screening BLL in μg/dL (No. of weeks after arrival)	BLL in µg/dL (No. of weeks after initial screening)	Lead hazards identified inside apartment	Lead hazards identified outside building	Elevated lead levels in dust samples ¹		
1	2 yrs 10 yrs	Côte d'Ivoire (Liberia)	20 (1) 16 (1)	21 (9) 17 (9)	Yes	No	Yes		
2	3 yrs 6 yrs	Tanzania (Burundi)	28 (4) 8 (4)	55 (9) 12 (9)	Yes	Yes	Yes		
3	20 mos 4 yrs	Côte d'Ivoire (Liberia)	3 (1) 9 (1)	20 (8) 11 (8)	Yes	No	Yes		
4	2 yrs	Côte d'Ivoire (Liberia)	9 (2)	48 (9)	No	Yes**	None taken		
5	5 yrs 7 yrs 6 yrs 16 mos 11 yrs	Kenya (Somalia)	3 (5) 3 (5) 4 (5) 5 (5) 3 (5)	26 (12) 25 (12) 63 (13) 25 (16) 11 (11)	Yes	Yes ^{††}	Yes		
6	3 yrs 6 yrs 10 yrs	Kenya (Somalia)	10 (4) 4 (4) 3 (4)	27 (13) 12 (13) 15 (13)	Yes	Yes ^{††}	Yes		
7	14 mos 14 mos	Côte d'Ivoire (Liberia)	5 (11) 10 (11)	24 (10) 29 (10)	Yes	Yes	None taken		
8	5 yrs	Kenya (Somalia)	2 (1)	11 ^{§§} (26)	Yes	Yes	Yes		
9	8 yrs	Kenya (Somalia)	4 (2)	11 (9)	NP ^{¶¶}	NP	NP		
0	14 mos	Kenya (Somalia)	9 (8)	10 (4)	NP	NP	NP		
1	13 yrs 10 yrs	Sierra Leone (Sierra Leone)	19 (1) 10 (1)	17 (7) 14 (7)	NP	NP	NP		
2	4 yrs 6 yrs 7 yrs	Côte d'Ivoire (Liberia)	11 (3) 6 (3) 10 (3)	11 (10) 12 (10) 12 (10)	NP	NP	NP		
3	5 yrs	Côte d'Ivoire (Liberia)	12 (1)	14 (5)	NP	NP	NP		
4	3 yrs	Kenya (Somalia)	9 (6)	11 (3)	NP	NP	NP		
5	4 yrs	Côte d'Ivoire (Liberia)	5 (3)	11 (2)	NP	NP	NP		
6	3 yrs 7 yrs 15 mos	Côte d'Ivoire (Liberia)	8 (1) 8 (1) 7 (1)	13 (12) 12 (12) 10 (10)	NP	NP	NP		
7	19 mos 4 yrs 5 yrs 7 yrs	Kenya (Somalia)	5 (2) 4 (2) 5 (2) 5 (2)	15 (10) 15 (10) 12 (10) 11 (10)	NP	NP	NP		
18	5 yrs	Côte d'Ivoire (Liberia)	14 (10)	14 (6)	NP	NP	NP		
19	2 yrs	Kenya (Somalia)	4 (1)	10 (4)	NP	NP	NP		

TABLE. Characteristics of refugee children with follow-up blood lead levels (BLLs) \geq 10 μ g/dL — New Hampshire, 2003–2004

* Age at time of follow-up BLL test.

[†] All children came to New Hampshire from refugee camps, which were not necessarily located in the country of origin.

§ According to state guidelines, environmental investigation is performed if child has BLL ≥20 µg/dL. Lead hazards were defined as surface with lead paint present with at least one of the following qualities: chipping or peeling paint, a chewable surface, or surface that creates friction on impact.

[¶] Samples taken from areas suspected for lead dust. Reference levels vary depending on a surface sampled. Elevated levels were defined as follows: for floors, >40 μg/ft²; for window sills, >250 μg/ft²; for window wells, >400 μg/ft².

** Lead hazards identified in neighborhood park.

^{††} These families are in the same apartment building and share a common courtyard.

§§ Capillary sample.

^{¶¶} Not performed (BLLs <20 μ g/dL).

which was inhabited predominantly by Somalis, indicated that 95% of children aged <6 years were anemic (7). Anemia can enhance lead absorption and thus can increase risk for elevated BLLs, even in housing with minimal lead exposure hazards.

The findings in this report are subject to at least two limitations. First, not all refugee children were tested. Second, not all of those children tested had two tests as recommended by the state guidelines for refugee children. Despite these limitations, these findings demonstrate that lead toxicity can be a substantial risk for refugee children. This investigation highlights the importance of lead testing of this population so children with elevated BLLs can be appropriately identified and managed. To control and prevent lead poisoning, NHDHHS is proposing state adoption of expanded medical and environmental protocols and has implemented active case finding of refugee children who have not had blood lead testing. In addition, CDC and NHDHHS are planning a study to obtain more information about risk factors for elevated BLLs among refugee children, which will help guide lead poisoning prevention strategies for refugee children.

Federal standards stipulate that refugees receive a medical screening within 90 days of arrival in the United States. Federal law does not require that refugee children have a blood lead test; however, some states, including New Hampshire, screen refugee children for lead toxicity. In 2004, a total of 9,333 children aged <7 years, 58.3% of whom were from Africa, were resettled in 49 states. Other states should review their lead testing and care practices for refugee children to help identify problems in this vulnerable population. CDC is working with other federal agencies involved in refugee health to include blood lead testing for refugee children. A blood lead test is the only way to know if a child has been exposed to lead. Other interventions include:

- pediatric multivitamins with iron for refugee children aged <59 months immediately on arrival in the United States;
- blood lead tests, hemoglobin or hematocrit tests, and nutritional assessments for children aged <6 years within 90 days of arrival, and another blood lead test 3–6 months after placement in a permanent residence; and
- consideration of blood lead screening for children aged ≥6 years if lead hazards are evident.

States should ensure that refugee families receive nutritional counseling and referral to the Supplemental Nutrition Program for Women, Infants, and Children (WIC). Increased lead-hazard training for refugee and resettlement case workers, health-care providers, and other agencies serving this population can help prevent lead poisoning among refugee children who enter the United States.

e xperience.

For over 50 years, MMWR has been the key provider of up-to-date public health reports and news. All of our publications-the Weekly, Recommendations and Reports, and Surveillance Summaries-are available online, free of charge.

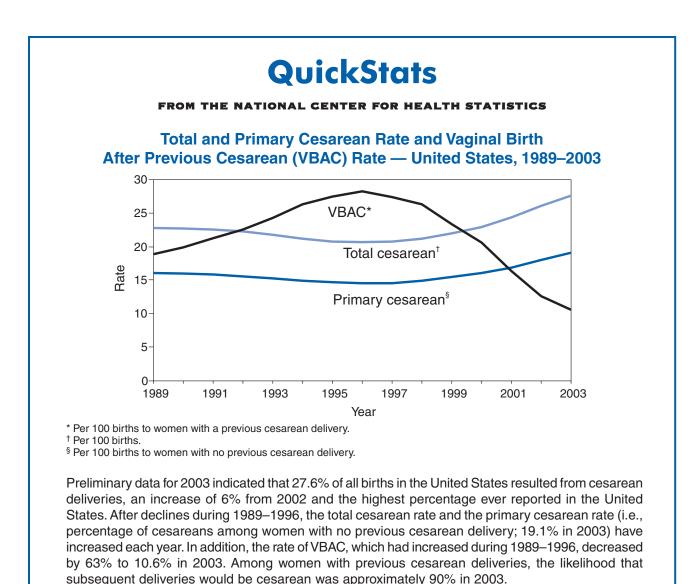
Visit **cdc.gov/mmwr** and experience timely public health information from a trusted source.

know what matters.



References

- 1. Binns HJ, Kim D, Campbell C. Targeted screening for elevated blood lead levels: populations at high risk. Pediatrics 2001;108:1364–6.
- 2. Geltman PL, Brown MJ, Cochran J. Lead poisoning among refugee children resettled in Massachusetts, 1995 to 1999. Pediatrics 2001;108:158-62.
- 3. CDC. Fatal pediatric lead poisoning—New Hampshire, 2000. MMWR 2001;50:457–9.
- 4. Gorstein J, Sullivan K, Yip R, et al. Issues in the assessment of nutritional status using anthropometry. Bull World Health Organ 1994;72:273-83.
- US Consumer Product Safety Commission. Ban of lead-containing paint and certain consumer products bearing lead-containing paint. 16 CFR 1303. Fed Reg 1977;42:44199.
- United Nations System/Standing Committee on Nutrition. Nutrition information in crisis situations. Geneva, Switzerland: Secretariat of the UNS/SCN; May 2004. Report no. 2. Available at http:// www.unsystem.org/scn/Publications/RNIS/NICSv2.pdf.
- 7. Prinzo AW, de Benoist B. Meeting the challenges of micronutrient deficiencies in emergency-affected populations. Proc Nutr Soc 2002;61:251-7.



SOURCE: National Vital Statistics System, annual files, 1989–2003. Available at http://www.cdc.gov/nchs/ births.htm.

Notice to Readers

New CDC Program for Rapid Genotyping of Mycobacterium tuberculosis Isolates

In January 2004, the CDC Tuberculosis Genotyping Program was initiated to enable rapid genotyping of isolates from every patient in the United States with culture-positive tuberculosis (TB). The Federal Tuberculosis Task Force recommended nationwide TB genotyping in response to the Institute of Medicine report, *Ending Neglect: The Elimination of Tuberculosis in the United States* (1,2). Subsequently, TB control programs in 50 states and two large cities (New York and San Diego) were approved to participate in the TB Genotyping Program, which was developed in collaboration with the National TB Controllers Association (NTCA).

The TB Genotyping Program contracts with laboratories in California and Michigan, which provide results within 10 working days from two polymerase chain reaction (PCR)-based genotyping tests: mycobacterial interspersed repetitive units (MIRU) typing (*3*) and spoligotyping (*4*). In combination, these two tests provide a highly discriminatory method to identify strains. An additional genotyping method, IS*6110*–based restriction fragment length polymorphism fingerprinting (*5*), is available to provide further discrimination between strains for isolates with identical PCR results. The mycobacteriology laboratory branch at CDC also participates in the TB Genotyping Program by performing genotyping testing for quality-control purposes.

In 2004, NTCA and CDC published the *Guide to the Application of Genotyping to Tuberculosis Prevention and Control* (6). TB genotyping will help TB-control programs identify recent transmission of TB, detect outbreaks sooner, identify false-positive *M. tuberculosis* cultures, evaluate completeness of routine contact investigations, and monitor progress toward TB elimination (6,7).

References

- CDC. Federal Tuberculosis Task Force plan in response to the Institute of Medicine Report, *Ending Neglect: The Elimination of Tuberculosis in the United States*. Atlanta, GA: US Department of Health and Human Services, CDC; 2003. Available at http://www.cdc.gov/nchstp/tb/pubs/ TaskForcePlan/TOC.htm.
- Institute of Medicine. Ending neglect: the elimination of tuberculosis in the United States. Washington, DC: The National Academies Press; 2000.
- Mazars E, Lesjean S, Banuls AL, et al. High-resolution minisatellitebased typing as a portable approach to global analysis of *Mycobacterium tuberculosis* molecular epidemiology. Proc Natl Acad Sci U S A 2001;98:1901–6.
- Kamerbeek J, Schouls L, Kolk A, et al. Simultaneous detection and strain differentiation of *Mycobacterium tuberculosis* for diagnosis and epidemiology. J Clin Microbiol 1997;35:907–14.

- van Embden JD, Cave MD, Crawford JT, et al. Strain identification of *Mycobacterium tuberculosis* by DNA fingerprinting: recommendations for standardized methodology. J Clin Microbiol 1993;31:406–9.
- 6. National TB Controllers Association/CDC Advisory Group on Tuberculosis Genotyping. Guide to the application of genotyping to tuberculosis prevention and control. Atlanta, GA: US Department of Health and Human Services, CDC; 2004. Available at http://www.cdc.gov/ nchstp/tb/genotyping/toc.htm.
- McNabb SJN, Braden CR, Navin TR. DNA fingerprinting of *Mycobac*terium tuberculosis: lessons learned and implications for the future. Emerg Infect Dis 2002;8:1314–9.

Notice to Readers

Satellite Broadcast on Epidemiology and Prevention of Vaccine-Preventable Diseases

CDC's National Immunization Program and the Public Health Training Network (PHTN) will present a live, fourpart, satellite broadcast series entitled "Epidemiology and Prevention of Vaccine-Preventable Diseases" on February 17 and 24 and March 3 and 10, 2005, from 12:00 noon to 3:30 p.m. Eastern Time. The series is intended for physicians, nurses, nurse practitioners, physician assistants, pharmacists, residents, medical and nursing students, and colleagues who either administer vaccinations or set policy in their workplaces.

Session 1 will cover principles of vaccination, general recommendations on immunization, vaccine administration, storage and handling, and vaccine safety. Session 2 will cover pertussis, pneumococcal disease (childhood), polio, and *Haemophilus influenzae* type b disease. Session 3 will cover measles, rubella, varicella, smallpox, and meningococcal disease. Session 4 will cover hepatitis B, hepatitis A, influenza, and pneumococcal disease (adult). Participants will be able to interact with instructors through toll-free telephone, fax, and TTY lines.

Continuing education credit will be offered for various professions based on 3 hours of instruction for each of the four broadcast sessions, providing a maximum of 12 hours of credit for all four sessions. Course participants should obtain their own copy of the primary course text, *Epidemiology and Prevention of Vaccine-Preventable Diseases*, 8th edition (2004). The text is available from the Public Health Foundation for \$29, by telephone at 877-252-1200, or at http://bookstore.phf.org/ prod111.htm.

The programs can be viewed via live webcast and will also be available for viewing after each live broadcast at http:// www.phppo.cdc.gov/PHTN/webcast/epv05/default.asp. Information about the satellite broadcasts or about continuing education credits is available at http://www.phppo.cdc.gov/ PHTN/epv05/default.asp. A list of state distance learning coordinators can be found at http://www.cdc.gov/nip/ed/ coordinators.htm.

Erratum: Vol. 54, No. 1

In the report, "Update: Influenza Activity — United States, 2004–05 Season," an error occurred on page 16 in the footnote linked to the bullet point, "• Out-of-home caregivers and household contacts of persons with high-risk conditions^{†††}." The footnote text should read, "Persons at high risk include adults aged ≥ 65 years, children aged 0-23 months, persons aged 2-64 years with underlying chronic medical conditions, women who will be pregnant during the influenza season, residents of nursing homes and long-term-care facilities, and children aged 2-18 years on chronic aspirin therapy."

CASES CURRENT DECREASE INCREASE DISEASE 4 WEEKS Hepatitis A, acute 144 Hepatitis B, acute 225 Hepatitis C, acute 26 70 Legionellosis 2 Measles Meningococcal disease 44 10 Mumps 855 Pertussis 0 Rubella 0.03125 0.0625 0.125 0.25 0.5 2 1 4 Ratio (Log scale)[†]

FIGURE I. Selected notifiable disease reports, United States, comparison of provisional 4-week totals January 15, 2005, with historical data

* No rubella cases were reported for the current 4-week period yielding a ratio for week 2 of zero (0). † Ratio of current 4-week total to mean of 15 4-week totals (from previous, comparable, and subsequent 4-week periods for the past 5 years). The point where the hatched area begins is based on the mean and two standard deviations of these 4-week totals.

Beyond historical limits

TABLE I. Summary of	f provisional cases	of selected notifiable	diseases, United States	, cumulative, week endin	g January [•]	15, 2005 (2nd Week)*	k

Disease	Cum. 2005	Cum. 2004	Disease	Cum. 2005	Cum. 2004
Anthrax	—	—	Hemolytic uremic syndrome, postdiarrheal [†]	2	
Botulism:			HIV infection, pediatric [†]	—	_
foodborne			Influenza-associated pediatric mortality**	2	_
infant	_	3	Measles	1 ⁺⁺	1 ^{§§}
other (wound & unspecified))	l —	_	Mumps	6	7
Brucellosis	2	1	Plague	—	_
Chancroid	2	3	Poliomyelitis, paralytic	_	_
Cholera	_	1	Psittacosis [†]	_	_
Cyclosporiasis [†]	_	1	Q fever [†]	3	1
Diphtheria	l —	_	Rabies, human	_	_
Domestic arboviral diseases			Rubella	_	1
(neuroinvasive & non-neuroinvasive):	_	—	Rubella, congenital syndrome	—	_
California serogroup ^{†§}	l —	_	SARS [†] **	_	_
eastern equine ^{†§}	l —	_	Smallpox [†]	_	_
Powassan ^{†§}	_		Staphylococcus aureus:		
St. Louis [†] §	l —	_	Vancomycin-intermediate (VISA) [†]	_	_
western equine ^{†§}	l —	_	Vancomycin-resistant (VRSA) [†]	_	_
Ehrlichiosis:	_		Streptococcal toxic-shock syndrome [†]	1	11
human granulocytic (HGE)†	_	3	Tetanus	_	1
human monocytic (HME) [†]		2	Toxic-shock syndrome	5	3
human, other and unspecified [†]	_	_	Trichinellosis	_	_
Hansen disease [†]	1	4	Tularemia [†]	_	_
Hantavirus pulmonary syndrome [†]	-	—	Yellow fever	—	—

-: No reported cases.

* Incidence data for reporting years 2004 and 2005 are provisional and cumulative (year-to-date).

Not notifiable in all states. Ş

Updated weekly from reports to the Division of Vector-Borne Infectious Diseases, National Center for Infectious Diseases (ArboNet Surveillance).

¹ Updated monthly from reports to the Division of HIV/AIDS Prevention, National Center for HIV, STD, and TB Prevention. Last update November 28, 2004. ** Updated weekly from reports to the Division of Viral and Rickettsial Diseases, National Center for Infectious Diseases.

†† The one case reported was indigenous.

\$5 The one case reported was imported from another country.

^{¶¶} Formerly Trichinosis.

(2nd Week)*					•		-		
	A	IDS	Chla	mydia [†]	Coccidioio	domycosis	Cryptosp	oridiosis	
Reporting area	Cum. 2005§	Cum. 2004	Cum. 2005	Cum. 2004	Cum. 2005	Cum. 2004	Cum. 2005	Cum. 2004	
UNITED STATES		22	18,204	30,981	89	60	38	101	
NEW ENGLAND	_	_	1,312	1,006	_	_	_	7	
Maine	—	_	109	55	N	N	_	2	
N.H.	_	_	72	55	_	—	_	1	
Vt. [¶] Mass.	—	—	22 583	29 495	_	_	—	2	
R.I.	_	_	154	495 206	_	_	_	2	
Conn.	_	_	372	166	Ν	Ν	_	_	
MID. ATLANTIC		14	2,001	3,239	_	_	5	12	
Upstate N.Y.	_	_	164	270	Ν	Ν	2	3	
N.Y. City	_	14	942	1,134	_	—	1	5	
N.J.	—	—	442	799			_	1	
Pa.	—	—	453	1,036	N	N	2	3	
E.N. CENTRAL Ohio	—	5	1,136 1	5,063 1,229	N	N	9 9	22	
Ind.	_	_	796	597	N	N	9	6	
III.	_	_	155	1,488	_	_	_	6	
Mich.	_	5	78	1,149	_	_	_	5	
Wis.	—	—	106	600	—	—	—	5	
W.N. CENTRAL	—	—	451	2,104			4	8	
Minn.	_	—	—	496	N	N	—	1	
Iowa Mo.	_	_	_	251 736	N	<u>N</u>	2	1 2	
N. Dak.	_	_	21	46	N	N		<u> </u>	
S. Dak.	_	_	116	88	_	_	1	2	
Nebr. ¹	—	_		145			_	_	
Kans.		—	314	342	N	N	1	2	
S. ATLANTIC	—	—	6,133	5,793			12	23	
Del. Md.	_	_	136 578	104 665	N	<u>N</u>	3	1	
D.C.	_	_	139	132	_	_			
Va.	_	_	1,212	968	_	_	_	_	
W. Va.	—	_	87	99	N	N	_	_	
N.C. S.C. ¹			1,727 764	1,114 296	N	N	2	8 1	
Ga.	_	_	277	1,418	_	_	5	5	
Fla.	_	_	1,213	997	N	N	2	8	
E.S. CENTRAL	_	_	962	2,136	_	_	1	5	
Ky.		_	414	267	N	N	—	_	
Tenn. ¹	—	—	396	863	N	N		1	
Ala. ¹ Miss.	_	_	3 149	553 453	_	_	1	3 1	
		_			_	_	_		
W.S. CENTRAL Ark.	_	_	2,081 118	4,633 269	_	_	_	4 1	
La.	_	_	404	1,516	_	_	_	_	
Okla.	_	_	392	395	N	N	—	_	
Tex. ¹	—	_	1,167	2,453	N	N	_	3	
MOUNTAIN	_	_	1,406	1,801	54	5	1	6	
Mont.	—	—	7		N	N	—	—	
Idaho Wyo.	_	_	1 40	97 34	N	<u>N</u>			
Colo.	_	_	265	448	Ν	Ν	1	4	
N. Mex.		_	21	238	_	1	—	_	
Ariz.	—	_	824	630	51	_	_	—	
Utah Nev. ¹	_	_	18 230	108 246	3	4	_	1	
	—						6		
PACIFIC Wash.	_	3	2,722 647	5,206 468	35 N	55 N	6	14	
Oreg. ¹	_	_	234	227		_	1	1	
Calif.	—	3	1,788	4,159	35	55	5	13	
Alaska	—	—	53	92	_	—	—	_	
Hawaii	_	_	—	260	_	_	_	_	
Guam	—	—		44					
P.R. V.I.	_	_	20	54 24	N	<u>N</u>	<u>N</u>	<u>N</u>	
Amer. Samoa	U	U	U	24 U	U	U	U	U	
C.N.M.I.	_	Ŭ	_	Ŭ	_	Ŭ	_	Ŭ	

TABLE II. Provisional cases of selected notifiable diseases, United States, weeks ending January 15, 2005, and January 17, 2004 (2nd Week)*

 N: Not notifiable.
 U: Unavailable.
 —: No reported cases.
 C.N.M.I.: Commonwealth of Northern Mariana Islands.

 * Incidence data for reporting years 2004 and 2005 are provisional and cumulative (year-to-date).
 *

 † Chlamydia refers to genital infections caused by *C. trachomatis.* §

 9 Updated monthly from reports to the Division of HIV/AIDS Prevention, National Center for HIV, STD, and TB Prevention. Last update November 28, 2004.

 1 Contains data reported through National Electronic Disease Surveillance System (NEDSS).

50

(2nd Week)*	-									
		Escher	<i>ichia coli</i> , Ente							
			-	n positive,	Shiga toxi				-	
		7:H7		o non-O157	not sero	<u> </u>	Giardi			rrhea
Reporting area	Cum. 2005	Cum. 2004	Cum. 2005	Cum. 2004	Cum. 2005	Cum. 2004	Cum. 2005	Cum. 2004	Cum. 2005	Cum. 2004
UNITED STATES	20	37	2	4	5	2	265	507	6,559	12,001
NEW ENGLAND	1	_	_	1	_	_	20	43	266	250
Maine	—	—	—	—	—	—	1	7	2	10
N.H. Vt.	_	_	_	_	_	_	3	2 2	6 2	
Mass.	1	—	—	1	—	—	16	32	92	126
R.I. Conn.	_	_	_	_	_	_	_	_	15 149	44 66
MID. ATLANTIC	_	6	_	_	_	_	28	111	641	1,132
Upstate N.Y.	_	3	_	_	_	_	9 5	13	108	90
N.Y. City N.J.	_	3	_	_	_	_	5 12	48 16	248 162	435 253
Pa.	—	3	—	—	—	—	2	34	123	354
E.N. CENTRAL Ohio	7	10 5	—	1	2	1	21	94 37	460	2,180
Ind.	6	_	_	_	_2	1	18	_	1 328	785 244
III. Mish		2	—	—	—	—		26	55	598
Mich. Wis.	1	3	_	1	_	_	3	23 8	31 45	387 166
W.N. CENTRAL	3	3	_	2	_	_	20	37	134	797
Minn. Iowa	2	_	_	_	_	_	3	8 8	_	210 52
Mo.	1	2	_	2	_	_	8	14	_	332
N. Dak.	_	_	_	_	_	_	_		1	4
S. Dak. Nebr.	_	_	_	_	_	_	2	1	13	11 61
Kans.	—	1	_	—	—	—	2 7	5	120	127
S. ATLANTIC	4	3			3	1	64	79	2,680	2,870
Del. Md.	2	_	N	N	N	<u>N</u>	6	4	33 285	46 353
D.C.	—	—	_	—	—	—	—	—	91	104
Va. W.Va.	_	_	_	_	_	_	1	_	380 27	366 31
N.C.	_	—	_	_	3	1	N	N	808	661
S.C. Ga.	1	1	_	_	_	_	1 34	40	382 128	142 680
Fla.	1	2	_	_	—	—	22	35	546	487
E.S. CENTRAL	—	—	_	_	—	—	4	12	407	1,099
Ky. Tenn.	_	_	_	_	_	_	N 1	N 2	164 176	121 393
Ala.	_	—	_	—	—	—	3	10	3	344
Miss.	—	_	—	_	—	_	_	_	64	241
W.S. CENTRAL Ark.	_	_2	_	_	_	_	2	7 3	935 78	1,910 116
La.	_	—	_	_	—	—	_	4	216	732
Okla. Tex.	_	2	_	_	_	_	2 N	N	140 501	179 883
MOUNTAIN	1	4	2	_	_	_	28	38	395	513
Mont.	_	_	—	—	—	—		1	1	3
ldaho Wyo.	1	1	1	_	_	_	5 1	5	1	3
Colo.	_	—	1	_	—	_	15	23	127	152
N. Mex. Ariz.	_	_	N	N	N	N	1	1	2 167	22 203
Utah	_	1	_	—	_	_	3	5	1	12
Nev.	_	2	_	_	—	_	2	3	96	119
PACIFIC Wash.	4	9	_	_	_	_	78	86	641 84	1,250 74
Oreg.	_	1	_	_	—	_	5	22	39	30
Calif. Alaska	3	5	_	_	_	_	70 2	62 1	509 9	1,069 14
Hawaii	1	3	—	_	_	—	1	1	_	63
Guam	N	Ν	—	_	_	—	—	—	_	12
P.R. V.I.	_	_		_	_	_	_	_	8	2 9
Amer. Samoa	U	U	U	U	U	U	U	U	U	U
C.N.M.I.	_	U	_	U	—	U	—	U	_	U

TABLE II. (*Continued*) Provisional cases of selected notifiable diseases, United States, weeks ending January 15, 2005, and January 17, 2004 (2nd Week)*

N: Not notifiable. U: Unavailable. —: No reported cases. C.N.M.I.: Commonwealth of Northern Mariana Islands. * Incidence data for reporting years 2004 and 2005 are provisional and cumulative (year-to-date).

(2nd Week)*		10						
·	All a	aes		Haemophilus inf		<5 years		
	All sero		Serot	type b		erotype b	Unknown	serotype
Reporting area	Cum. 2005	Cum. 2004	Cum. 2005	Cum. 2004	Cum. 2005	Cum. 2004	Cum. 2005	Cum. 2004
UNITED STATES	50	98	2005		2005	2004	4	12
NEW ENGLAND	4	8	_	_	_	_	2	_
Maine	—	_	—	—	—	—	_	—
N.H. Vt.	4	1 1	_	_	_	_	2	_
Mass.		6	_	_	_	_	_	_
R.I. Conn.		_	_		_	_	_	_
MID. ATLANTIC	13	27	_	_	_	_	_	3
Upstate N.Y.	4	4	—	—	—	_	—	—
N.Y. City N.J.	1 4	3 8	_	_	_	_	_	1 1
Pa.	4	12	_	_	_	_	_	1
E.N. CENTRAL	6	21	_	_	_	_	1	5
Ohio Ind.	5 1	6	—	_	—	—	1	1
III.		9	_	_	_	_	_	2
Mich.	—	3	—	—	—	_	—	1
Wis.	—	3	—	—	—	—	—	1
W.N. CENTRAL Minn.	4	4	_	_	_	_	_	_
lowa	_	_	_	_	_	_	_	_
Mo.	4	—	—	—	—	_	—	—
N. Dak. S. Dak.	_	_	_	_	_	_	_	_
Nebr.	_	4	_	_	—	_	—	—
Kans.			—	—		—	_	_
S. ATLANTIC Del.	17	17	_	_	1	_	1	_2
Md.	5	7	_	_	1	_	1	_
D.C. Va.	_	_	_	_	_	_	_	_
W. Va.	_	_	_	_	_	_	_	_
N.C. S.C.	2 1	_	_	_	_	_	_	_
Ga.	2	6	_	_	_	_	_	2
Fla.	7	4	—	—	—	—	—	—
E.S. CENTRAL	—	6	—	—	—	_	—	1
Ky. Tenn.		1	_	_	_	_	_	_
Ala.	—	5	_	—	—	_	_	1
Miss.			—	—	—	—	—	—
W.S. CENTRAL Ark.	1	4	_	_	_	_	_	_
La.	_	3	_	_	_	_	_	_
Okla. Tex.	1	1	_		_	_	_	_
MOUNTAIN	3	10	_	_	1	_	_	1
Mont.		10	_	_	_	_	_	—
Idaho	_	—	—	—	—	_	—	—
Wyo. Colo.	1	5	_	_	_	_	_	_
N. Mex.	—	4	_	_	_	—	_	1
Ariz. Utah	1	_	_	_	_	_	_	_
Nev.	1	1	—	—	1	_	—	_
PACIFIC	2	1	_	_	_	_	_	_
Wash. Oreg.	1	1	—	—	—	—	_	—
Calif.	_	<u> </u>	_	_	_	_	_	_
Alaska Hawaii	1	_	_	_	_	_	_	_
Guam P.R.	_	_	_	_	_	_	_	_
V.I.		_			_		_	_
Amer. Samoa C.N.M.I.	U 	U U	U 	U U	U 	U U	U 	U U

 TABLE II. (Continued) Provisional cases of selected notifiable diseases, United States, weeks ending January 15, 2005, and January 17, 2004

 (2nd Week)*

N: Not notifiable. U: Unavailable. —: No reported cases. C.N.M.I.: Commonwealth of Northern Mariana Islands. * Incidence data for reporting years 2004 and 2005 are provisional and cumulative (year-to-date).

52

Vol. 54 / No. 2	
-----------------	--

(2nd Week)*					, 10, 2000, and ou	laal y 11, 2001
		Α	Hepatitis (vir	al, acute), by type B		С
	Cum.	Cum.	Cum.	Cum.	Cum.	Cum.
Reporting area	2005	2004	2005	2004	2005	2004
UNITED STATES	72	218	122	173	12	29
NEW ENGLAND	17	29	3	8	—	_
Maine N.H.	_	_	1	_	_	_
Vt.	—	_		1	—	_
Mass. R.I.	14	25	2	5	_	_
Conn.	3	4	_	2	_	_
MID. ATLANTIC	1	38	20	25	1	4
Upstate N.Y. N.Y. City	1	10	1	4	—	—
N.J.	_	11	 18	11	_	_
Pa.	_	17	1	10	1	4
E.N. CENTRAL	4	17	5	13	1	2
Ohio Ind.	2	1	5	6	_	_
III.	_	9 5	_	—		
Mich. Wis.	2	5 2	_	5 2	1	2
W.N. CENTRAL	3	6	7	9	3	5
Minn.	—	_	<u> </u>		—	—
lowa	1	_	4			
Mo. N. Dak.	1	2	4	8	3	5
S. Dak.	_	_	_	_	—	_
Nebr. Kans.	1	2	2 1	1	_	_
S. ATLANTIC	17	41	68	62	5	4
Del.	—	_	—	_	_	_
Md. D.C.	_	4	5	7	3	2
Va.	_	_	_	_	_	_
W.Va. N.C.	1	_	12	1	1	_
S.C.	_	_	_	1	—	_
Ga. Fla.	9 7	25 12	30 21	31 22	1	1 1
E.S. CENTRAL	<i>'</i>	7	2	12	1	1
Ky.	_	_			—	
Tenn. Ala.	—	5 2	1	3	1	—
Miss.	_		1	9	—	1
W.S. CENTRAL	_	36	_	7	_	9
Ark.	_	1	_	—	—	_
La. Okla.	_	1	_	7	_	7
Tex.	_	34	_	—	—	2
MOUNTAIN	12	1	5	7	1	1
Mont. Idaho	2	_	_	1	_	_
Wyo.	_	_		1	—	—
Colo. N. Mex.	2	1	1	_	_	_
Ariz.	7	—	_	—	<u> </u>	—
Utah Nev.	1	_	3 1	5	1	1
PACIFIC	18	43	12	30	_	3
Wash.	_	—	—	_	—	—
Oreg. Calif.	2 16	4 38	1 11	10 20	_	1 1
Alaska		—	—		_	—
Hawaii	—	1	_	_	_	1
Guam P.R.		_	_	_	_	_
V.I.	_	_	_	_	_	_
Amer. Samoa C.N.M.I.	U	U U	U	U U	<u> </u>	U U
U.IN.IVI.I.		U	_	U		0

TABLE II. (Continued) Provisional cases of selected notifiable diseases, United States, weeks ending January 15, 2005, and January 17, 2004

N: Not notifiable. U: Unavailable. —: No reported cases. C.N.M.I.: Commonwealth of Northern Mariana Islands. * Incidence data for reporting years 2004 and 2005 are provisional and cumulative (year-to-date).

(2nd Week)*										
		Legionellosis		eriosis	Lyme d		Mala	ria		
Reporting area	Cum. 2005	Cum. 2004	Cum. 2005	Cum. 2004	Cum. 2005	Cum. 2004	Cum. 2005	Cum. 2004		
UNITED STATES	27	57	14	23	69	309	23	54		
NEW ENGLAND	_	_	_	_	_	24	1	3		
Maine	—	—	—	—	—	—	—	_		
N.H. Vt.	—	—	—	—	—	_	_	_		
Mass.	_	_	_	_	_	24	1	3		
R.I.	_	_	_	_	_	—	_	_		
Conn.	—	—	—	—	_	—	—	—		
MID. ATLANTIC	5	16	1	4 1	48	251	2	12		
Upstate N.Y. N.Y. City	2	1	_		4	63	1	6		
N.J.	2	6	_	2	34	62	1	2		
Pa.	1	9	1	1	10	126	—	4		
E.N. CENTRAL Ohio	6 4	22 11	2 1	3 2	6 5	5 1	1 1	3		
Ind.	4	—	_		5		—	_		
III.	<u> </u>	5	_	_		—	—	1		
Mich. Wis.	1	5 1	1	1	1 U	4	_	1		
W.N. CENTRAL		1	1	·	0	2		2		
Minn.	_	_	_	_	_		_			
Iowa	_	_	1	_	_	1	—	_		
Mo. N. Dak.	_	_	_	_	_	1	_	2		
S. Dak.	_	1	_	_	_	_	_	_		
Nebr. Kans.	—	—	—	—	—	—	—	_		
			_	_			_			
S. ATLANTIC Del.	8	8	4 N	6 N	13	21 2	3	16		
Md.	3	2	1	2	8	17	1	5		
D.C. Va.			_	_	_	_	_	_		
W. Va.	_	_	_	_	_	_	_	_		
N.C.	1	3	2	2	—	—	—			
S.C. Ga.	1	1	_	1	_	_	2	1 4		
Fla.	3	2	1	1	5	2	—	6		
E.S. CENTRAL	—	2	—	2	1	—	2	1		
Ky. Tenn.	_	_		1	1		2	_		
Ala.	_	2	_	<u> </u>		_		_		
Miss.	—	—	—	—	—	—	—	1		
W.S. CENTRAL	—	3	_	—	—	3	—	10		
Ark. La.	_	_	_	_	_	_	_	2		
Okla.	—		—	—	—		—	_		
Tex.	—	3	—	—	—	3	—	8		
MOUNTAIN Mont.	—	2	—	2	—	—	2	2		
Idaho	_	_	_	_	_	_	_	_		
Wyo.	—	1	_	_	—	_	—	_		
Colo. N. Mex.	_	1	_	1	_		_	1		
Ariz.	—	—	—	—	—	_	1	_		
Utah Nev.	_	_	_	1	_	_	1	1		
PACIFIC	8	3				3	12			
Wash.	<u> </u>	_	6	6	1	<u> </u>	12	5		
Oreg.	N	N	_	2		_	1			
Calif. Alaska	8	3	6	4	1	3	11	5		
Hawaii	_	_	_	_	N	Ν	_	_		
Guam	_	_	_	_	_	_	_	_		
P.R.	—	—	—	—	N	Ν	—	—		
V.I. Amer. Samoa	 U	 U	 U	U	U	 U	 U	U		
C.N.M.I.		Ŭ	_	Ŭ	_	Ū	_	Ŭ		
NI: Nist a stiff shis				ON MILL OF						

TABLE II. (*Continued*) Provisional cases of selected notifiable diseases, United States, weeks ending January 15, 2005, and January 17, 2004 (2nd Week)*

N: Not notifiable. U: Unavailable. —: No reported cases. C.N.M.I.: Commonwealth of Northern Mariana Islands. * Incidence data for reporting years 2004 and 2005 are provisional and cumulative (year-to-date).

					Meningoco	ccal disease					
	All sero	aroups	Serog A, C, Y, a		Serog	roun B	Other se	rogroup	Serogroup unknowr		
	Cum.	Cum.	Cum.	Cum.	Cum.	Cum.	Cum.	Cum.	Cum.	Cum.	
Reporting area	2005 30	2004 101	2005 1	2004 6	2005	2004	2005	2004	2005 14	2004 21	
NEW ENGLAND	4	3		_	_	_		_	3	2	
Maine	—	_	_	_	_	_	_	_	_	_	
N.H. Vt.	3	_	_	—	—	—	_	_	3	_	
Mass.	1	3	_	_	_	_	_	_		2	
R.I.	—	—	—	_	—	—	—		—	—	
Conn.	_	—	_	_	_	—	_	_	_	_	
MID. ATLANTIC Upstate N.Y.	1 1	13 5	_	2 2	_	_	_	_	1	3 3	
N.Y. City	_	1	—	_	—	—	_	—	—	—	
N.J. Pa.	_	3 4	_	_	_	_	_	_	_	_	
E.N. CENTRAL	4	17	_	3	_	_	_	_	2	4	
Ohio	1	9	_	3	_	_	_	_	1	4	
Ind.	1		—	_	—	—	—		1	_	
III. Mich.	2	1 7	_	_	_	_	_	_	_	_	
Wis.	_	—	_	_	—	—	_	_	—	—	
W.N. CENTRAL	5	5	—	—	—	—	—		2	2	
Minn. Iowa		_	_	_	_	_	_	_	_	_	
Mo.	4	1	_	_	_	_	_	_	2	_	
N. Dak.	—	_	—	—	—	—	—	_	—	_	
S. Dak. Nebr.	_	1 1	_	_	_	_	_	_	_	1 1	
Kans.	1	2	_	_	_	_	_	_	_	_	
S. ATLANTIC	6	14	1	_	_	_	_	_	2	2	
Del. Md.	1	2	_	_	_	_	_	_	1	2	
D.C.	_		_	_	_	_	_	_	_		
Va.	—	—	—	—	—	—	—	_	—	—	
W.Va. N.C.	1	_	1	_	_	_	_	_	_	_	
S.C.	1	_	_	_	—	—	_	—	1	—	
Ga. Fla.	1 2	3 9	_	_	_	_	_	_	_	_	
E.S. CENTRAL	_	4	_	_	_	_	_	_	_	1	
Ky.	_	_	_	_	_	_	_	_	_	_	
Tenn.		3	—	_	_	—	_	_	_	1	
Ala. Miss.	_	1	_	_	_	_	_	_	_	_	
W.S. CENTRAL	_	13	_	1	_	_	_	_	_	6	
Ark.	—	—	—	_	—	—	_	—	—	—	
La. Okla.	_	8	_	1	_	_	_	_	_	6	
Tex.	_	5	_	_	_	_	_	_	_	_	
MOUNTAIN	2	3	_	_	_	_	_		1	1	
Mont.	_	_	—	—	—	—	—	—	—	—	
ldaho Wyo.	_	1 1	_	_	_	_	_	_	_	1	
Colo.	1	1	_	_	—	—	_	_	—	_	
N. Mex. Ariz.		_	_	_	_	_	_	_	1	_	
Utah	_	_	_	_	_	_	_	_	—	_	
Nev.	—	_	—	_	—	—	—	—	—	_	
PACIFIC	8	29	—	—	—	—	—	—	3	—	
Wash. Oreg.	3	6	_	_	_	_	_	_	3	_	
Calif.	5	22	—	—	—	—	—	—	_	—	
Alaska Hawaii		1	_	_	_	_	_	_	_	_	
			_	_	—	_	_		—	_	
Guam P.R.	_	_	_	_	_	_	_	_	_	_	
V.I.	<u></u>	_	_	—	—	—	_	—	—	—	
Amer. Samoa C.N.M.I.	U	U U	_	_	_	_	_	_	_	_	
					ML: Common						

TABLE II. (*Continued*) Provisional cases of selected notifiable diseases, United States, weeks ending January 15, 2005, and January 17, 2004 (2nd Week)*

N: Not notifiable. U: Unavailable. —: No reported cases. C.N.M.I.: Commonwealth of Northern Mariana Islands. * Incidence data for reporting years 2004 and 2005 are provisional and cumulative (year-to-date).

	Pert	ussis	Rabies,	animal	Rocky N spotte		Salmor	nellosis	Shigellosis		
Reporting area	Cum. 2005	Cum. 2004	Cum. 2005	Cum. 2004	Cum. 2005	Cum. 2004	Cum. 2005	Cum. 2004	Cum. 2005	Cum. 2004	
UNITED STATES	309	275	92	385	14	15	562	978	165	399	
NEW ENGLAND	9	96	23	5	_	3	24	55	4	15	
Maine N.H.	_	_	1	_	_	_	1	2 1	_	_	
Vt.	6	_	_	_	_	_	5	2	_	_	
Mass. R.I.	3	93	17	5	_	3	12	43	4	12	
Conn.	—	3	5	_	_	_	5	7	_	3	
MID. ATLANTIC	26	59	6	20	—	3	30	118	3	41	
Upstate N.Y. N.Y. City	8	18 6	5 1	5 1	_	1	5 10	9 43	1	14 11	
N.J. Pa.	 18	13 22	_	 14	_	2	5 10	34 32	2	10 6	
Fa. E.N. CENTRAL	10	48	1	14	1		36	158	8	48	
Ohio	110	19	1	1	1	_	29	39	o 4	40	
Ind. III.	_	1	_	_	_	_	_	 66	_	28	
Mich.	4	3	—	—	_	—	7	21	4	6	
Wis.	—	25	—	—	_	—	—	32	_	5	
W.N. CENTRAL Minn.	30	26	2	14 3	2	_	40 1	49 7	15 1	19 1	
Iowa	_	4	_	2	_	—	12	4	3	2	
Mo. N. Dak.	9 6	19	2	1	2	_	16 2	17 1	10	6 1	
S. Dak.	1	—	—	3	—	_	1	3	_	_	
Nebr. Kans.	7 7	3	_	5	_	_	4 4	3 14	1	9	
S. ATLANTIC	17	7	33	269	11	4	242	210	41	106	
Del. Md.	7	5	5	 13	1	_	17	 16	5	1 5	
D.C.		_	_	—	—	_	_	_			
Va. W. Va.	_	2	6	6 3	_	_	_2	1	_	_	
N.C.	_	—	16	26	5	2	65	33	—	14	
S.C. Ga.	5	_	_	 14	3	2	9 60	1 51	21	2 33	
Fla.	5	_	6	207	2	—	89	108	15	51	
E.S. CENTRAL	3	6	1	40	—	5	17	60	6	11	
Ky. Tenn.	_	4	_	1 36	_	2	2 5	2 13	4	2	
Ala. Miss.	3	1 1	1	3	_	1 2	10	25 20	2	7 2	
W.S. CENTRAL	2	3	20	30	_		13	117	13	89	
Ark.	1	2	5		_	_	3	4	2	—	
La. Okla.	_	1	2	2	_	_	2 3	12 14	3 6	8 8	
Tex.	1	—	13	28	_	_	5	87	2	73	
MOUNTAIN	102	16	4	4	—	_	44	60	19	17	
Mont. Idaho	6 2	3 2	_	_	_	_	1 2	3 12	_	_	
Wyo. Colo.	1 84	1 5	—	—	—	_	3	1 21	5	1 5	
N. Mex.	_	5 4	_	_	_	_	16	10	_	5 11	
Ariz. Utah	7 2	1	4	4	—	_	17 2	4	10	—	
Nev.		_	_	—	_	_	3	9	4	_	
PACIFIC	6	14	2	2	_	_	116	151	56	53	
Wash. Oreg.	5	14	_	_	_	_	1	23	- 1	6	
Calif.	_	—	2	2	—	_	109	113	54	41	
Alaska Hawaii	1	_	_	_	_	_	3 3	9 6	1	6	
Guam	_	_	_	_	_	_	_	_	_	1	
P.R.	_	_	2	1	Ν	Ν	_	3	_	_	
V.I. Amer. Samoa	U	U	U	U	U	U	U	U	U	U	
C.N.M.I.	_	Ŭ		Ŭ		Ŭ		Ŭ		Ŭ	

N: Not notifiable. U: Unavailable. —: No reported cases. C.N.M.I.: Commonwealth of Northern Mariana Islands. * Incidence data for reporting years 2004 and 2005 are provisional and cumulative (year-to-date).

(2nd Week)*				,		,		-,	. , ,			
				coccus pneum	oniae, invasiv	/e disease	Syphilis					
		cal disease, , group A	Drug res all ag		Age <5	vears	Primary & se		Conge	nital		
	Cum.	Cum.	Cum.	Cum.	Cum.	Cum.	Cum.	Cum.	Cum.	Cum.		
Reporting area	2005	2004	2005	2004	2005	2004	2005	2004	2005	2004		
UNITED STATES	122	242	60	138	20	25	106	251	2	18		
NEW ENGLAND Maine	5	9	_	_	1	3	9	4	_	_		
N.H. Vt.	_	1	_	—	—	_	—	_	—	—		
Mass.	5	8	N	N	1	3	9	1	_	_		
R.I. Conn.		_	_	_	 U	 U	_	3	_	_		
MID. ATLANTIC	17	35	2	11	2	2	5	27	_	3		
Upstate N.Y. N.Y. City	10 1	8 11	1 U	2 U	1 U	1 U	_	 17	_	1		
N.J.	3	7		_	1		4	7	_	1		
Pa.	3	9	1	9		1	1	3	—	1		
E.N. CENTRAL Ohio	5 2	65 16	8 8	32 30	6 6	10 8	1	30 6	_	1		
Ind.	_	—	_	2	_	_	1	4	—	—		
III. Mich.	3	20 23	N	N	_	_	_	11 7	_	1		
Wis.	—	6	N	N	_	2	—	2	—	_		
W.N. CENTRAL Minn.	10	13	3	1	3	1	_	5	_	_		
Iowa	N	N	N	N	_	_	—	1	_	_		
Mo. N. Dak.	4 1	4	3	1	1	_	_	4	_	_		
S. Dak. Nebr.	3 2	1 2	_	_	1	1	_	_	_	_		
Kans.	<u> </u>	6	N	N	1	_	_	_	_	_		
S. ATLANTIC	43	38	40	71	4	2	52	60	—	5		
Del. Md.	22	6	_	_	4	2	 10	1 13	_	1		
D.C. Va.	_	_	N	N	_	_	5 1	1 1	—	1		
W.Va.	_	_	_	_	_	_	_	_	_	_		
N.C. S.C.	5	2 1	N	N	U	U	11	5 4	_	1		
Ga.	7 9	16 13	13 27	29 42	_	—	 25	4 31	—	_		
Fla. E.S. CENTRAL	9 2	13	27	42 6	_	_	25 10	11	1	2 1		
Ky.		1	_	1	_	_	_	4	_	_		
Tenn. Ala.	2	13	2	5	_	_	7 3	5 1	1	1		
Miss.	_	_	—	—	_	_	_	1	_	_		
W.S. CENTRAL Ark.	2	27	3 1	5 1	1	2	18	46 1	_	6		
La.	_	1	2	4	_	1	4	7	_	_		
Okla. Tex.	2	1 25	N N	N N	1	- 1	2 12	1 37	_	1 5		
MOUNTAIN	24	17	1	3	3	5	4	12	1	_		
Mont. Idaho	_	1	N	N	_	_	_	3	—	_		
Wyo.	_	2		1	_	_	_		_	_		
Colo. N. Mex.	12	6 8	_	2	3	5	_	2 4	_	_		
Ariz.	12	_	N	N	—	—	2	2	1	—		
Utah Nev.	_	_	1	_	_	_	2	1	_	_		
PACIFIC	14	24	1	9	_	_	7	56	_	2		
Wash. Oreg.	N	N	N	N	_	_	2	_	_	_		
Calif.	10	19	N	N	_	—	5	56	_	2		
Alaska Hawaii	4	5	1	9	_	_	_	_	_	_		
Guam					_	_	_	_	_	_		
P.R. V.I.	<u>N</u>	<u>N</u>	<u>N</u>	N	_	_	_	2 1	_	_		
Amer. Samoa C.N.M.I.	U	U U	U	U U	U	U U	U	U U	U	U U		
U.IN.IVI.I.		0		0		0		U	_	0		

TABLE II. (Continued) Provisional cases of selected notifiable diseases, United States, weeks ending January 15, 2005, and January 17, 2004 (2nd Week)*

N: Not notifiable. U: Unavailable. —: No reported cases. C.N.M.I.: Commonwealth of Northern Mariana Islands. * Incidence data for reporting years 2004 and 2005 are provisional and cumulative (year-to-date).

(2nd Week)*							1					
	Tuba		osis Typhoid fever			cella		West Nile virus disease [†] Neuroinvasive Non-neuroinvasive [§]				
	Cum.	culosis Cum.	Cum.	Cum.	Cum.	enpox) Cum.	Cum.	Cum.	Cum.			
Reporting area	2005	2004	2005	2004	2005	2004	2005	2004	2005			
UNITED STATES	62	305	1	6	448	585	_	_	—			
NEW ENGLAND	_	7	_	_	17	42	_	_	—			
Maine N.H.		_	_	_	16	_	_	_	_			
Vt.	_	_	_	_	1	42	_	_	_			
Mass. R.I.		2 2	_	_	_	_	_	_	_			
Conn.	_	3	_	_	_	_	_	_	_			
MID. ATLANTIC	29	46	_	1	1	3	_	_	_			
Upstate N.Y. N.Y. City	29	1 44	_	_	_	_	_	_	_			
N.J.	29	_	_	1	_	_	_	_	_			
Pa.	—	1	—	—	1	3	—	—	—			
E.N. CENTRAL	7	5	_	1	188	270	—	—	—			
Ohio Ind.	5 2	1 2		1	51	60	_	_	_			
III.	—	_	—	_	_		—	—	—			
Mich. Wis.	_	2	_	_	137	197 13	_	_	_			
WIS. W.N. CENTRAL	8	2			3	7	_	_	_			
Minn.	8	_	_	_	3	_	_	_	_			
lowa	_	_	—	_	N	Ν	_	_	_			
Mo. N. Dak.	8	_	_	_	_	6	_	_	_			
S. Dak.	—	_	—	—	3	1	—	—	—			
Nebr. Kans.		_	_	_	_	_	_	_	_			
S. ATLANTIC	2	41	1	_	56	92	_	_	_			
Del.	_	1	_	_			_	_	_			
Md. D.C.	_	1	_	—	_	_	_	_	_			
Va.	_	_	_	_	_	_	_	_	_			
W.Va. N.C.		1	1	_	55	89	_	_	_			
S.C.	_	1	_	_	1	3	_	_	_			
Ga.	—	37	—	—	_	—	—	—	—			
Fla.	—	_	—	—	_	—	—	—	—			
E.S. CENTRAL Ky.		4	_	_	_	_	_	_	_			
Tenn.	—		—	—	—	—	—	—	—			
Ala. Miss.		4		_	_	_	_	_	_			
W.S. CENTRAL	1	86	_	3	41	137	_	_	_			
Ark.	1	1	_	_			_	_	_			
La. Okla.	—	4	—	_	_	_	_	—	—			
Tex.	_	81	_	3	41	137	_	_	_			
MOUNTAIN	_	3	_	_	142	34	_	_	_			
Mont.	—	—	—	—	_	—	—	—	—			
Idaho Wyo.	_	_	_	_	2	7	_	_	_			
Colo.	—	1	—	—	118	—	—	—	—			
N. Mex. Ariz.		1	_	_	_	2	_	_	_			
Utah	_	1	_	_	22	25	_	_	_			
Nev.	_	—	—	—	—	_	—	—	_			
PACIFIC Wash.	15 10	113 6	—	1	—	_	_	—	_			
Oreg.	1	2	_	_	_	_	_	_	_			
Calif.	—	101	—	1	—	—	—	—	—			
Alaska Hawaii	4	4	_	_	_	_	_	_	_			
Guam	_	4	_	_	_	9	_	_	_			
P.R.	—	_	—	—	—	14	—	—	—			
V.I. Amer. Samoa	U	 U	 U	 U	 U	 U	 U	 U	_			
C.N.M.I.	_	U	_	Ŭ	_	Ŭ	_	Ŭ	_			
N: Not notifiable	LI: I Inavailable	: No r	enorted cases	C NU		ern Mariana Islands						

TABLE II. (Continued) Provisional cases of selected notifiable diseases, United States, weeks ending January 15, 2005, and January 17, 2004 (2nd Week)*

N: Not notifiable. U: Unavailable. —: No reported cases. C.N.M.I.: Commonwealth of Northern Mariana Islands. * Incidence data for reporting years 2004 and 2005 are provisional and cumulative (year-to-date). [†] Updated weekly from reports to the Division of Vector-Borne Infectious Diseases, National Center for Infectious Diseases (ArboNet Surveillance). [§] Not previously notifiable.

TABLE III. Deaths in 122 U.S. cities,* week ending January 15, 2005 (2nd Week)

TABLE III. Dealins	All causes, by age (years)		2005 (21			All causes, by age (years)									
Reporting Area	All Ages	<u>≥</u> 65	45-64	25–44	1–24	<1	P&I [†] Total	Reporting Area	All Ages	<u>≥</u> 65	45-64	25–44	1-24	<1	P&I [†] Total
NEW ENGLAND	635	468	112	41	6	8	80	S. ATLANTIC	1,308	800	309	114	46	39	72
Boston, Mass.	189	124	39	17	3	6	28	Atlanta, Ga.	212	136	47	19	7	3	11
Bridgeport, Conn.	42	32	8	1	_	1	5	Baltimore, Md.	191	103	53	22	4	9	14
Cambridge, Mass.	18	14	4	—		—	2	Charlotte, N.C.	131	90	27	11	1	2	17
Fall River, Mass.	32	27	4		1	_	4	Jacksonville, Fla.	136	88	30	10	3	5	5
Hartford, Conn. Lowell, Mass.	61 23	43 18	13 4	4 1	_	1	8 1	Miami, Fla. Norfolk, Va.	81 61	46 39	16 13	7 4	6 4	6 1	4 2
Lynn, Mass.	13	12	4	1	_	_	_	Richmond, Va.	75	43	13	7	4	3	2 5
New Bedford, Mass.	26	15	9	2	_	_	5	Savannah, Ga.	63	38	16	5	1	3	2
New Haven, Conn.	26	19	5	2	_	_	4	St. Petersburg, Fla.	65	48	13	4	_	_	1
Providence, R.I.	66	55	10	1	_	—	6	Tampa, Fla.	165	104	38	11	9	3	6
Somerville, Mass.	3	3	_	_		—	_	Washington, D.C.	101	48	30	11	8	4	2
Springfield, Mass.	38	29	5	3	1	_	5	Wilmington, Del.	27	17	7	3	_	_	3
Waterbury, Conn. Worcester, Mass.	34 64	29 48	3 8	2 7	1	_	3 9	E.S. CENTRAL	1,043	722	216	66	18	21	77
								Birmingham, Ala.	232	160	47	14	7	4	19
MID. ATLANTIC	2,525	1,772	526	130	53	43	194	Chattanooga, Tenn.	100	74	21	2	2	1	8
Albany, N.Y. Allentown, Pa.	70 27	51 24	15 3	_	2	2	12 3	Knoxville, Tenn.	83 90	63	10 22	8 5	1	2 1	7 6
Buffalo, N.Y.	60	24 42	14	4	_	_	4	Lexington, Ky. Memphis, Tenn.	215	61 147	45	12	3	8	10
Camden, N.J.	26	16	7	_	_	3	2	Mobile, Ala.	85	55	18	8	3	1	4
Elizabeth, N.J.	33	20	6	4	_	3	2	Montgomery, Ala.	81	56	18	5	1	1	7
Erie, Pa.	51	40	9	1	1	—	6	Nashville, Tenn.	157	106	35	12	1	3	16
Jersey City, N.J.	48	33	14			1		W.S. CENTRAL	1,622	1,070	366	108	39	39	133
New York City, N.Y.	1,402	1,014	271	76	21	19	91	Austin, Tex.	86	61	18	5	1	1	4
Newark, N.J. Paterson, N.J.	70 25	39 15	20 6	9 2	2 2	_	5 1	Baton Rouge, La.	50	33	8	4	2	3	2
Philadelphia, Pa.	25 249	121	80	22	2 17	9	9	Corpus Christi, Tex.	65	42	14	6	2	1	3
Pittsburgh, Pa.§	29	16	11	2		_	_	Dallas, Tex.	173	113	31	17	3	9	16
Reading, Pa.	20	15	3	1	_	1	3	El Paso, Tex.	88	69 110	14 37	2 7	3 4	7	6 12
Rochester, N.Y.	152	122	25	2	2	1	20	Ft. Worth, Tex. Houston. Tex.	165 428	280	37 99	27	4 11	11	35
Schenectady, N.Y.	23	20	2			1	6	Little Rock, Ark.	420	200 54	24	5	2	1	7
Scranton, Pa.	40	32	5	2	1	_	2	New Orleans, La.	45	23	13	7	2		_
Syracuse, N.Y. Trenton, N.J.	134 22	100 18	26 3	2	3 1	3	23 2	San Antonio, Tex.	273	175	67	20	6	5	32
Utica, N.Y.	15	12	2	1	_	_	1	Shreveport, La.	32	25	5	1	1		3
Yonkers, N.Y.	29	22	4	2	1	_	2	Tulsa, Okla.	131	85	36	7	2	1	13
E.N. CENTRAL	2,506	1,679	563	152	44	68	198	MOUNTAIN Albuquerque, N.M.	1,054 136	708 87	233 37	67 4	24 6	21 1	69 7
Akron, Ohio	56	41	12	2	_	1	5	Boise, Idaho	49	31	10	6	2	_	4
Canton, Ohio	40 331	31 201	8 85	1 28	7	10	9 19	Colo. Springs, Colo.	65	49	11	3	_	2	2
Chicago, III. Cincinnati, Ohio	132	83	32	20 8	3	6	10	Denver, Colo.	102	61	24	9	3	5	9
Cleveland, Ohio	303	225	56	11	4	7	21	Las Vegas, Nev.	271	176	69	15	6	5	13
Columbus, Ohio	226	144	56	15	4	7	25	Ogden, Utah Phoenix, Ariz.	38 80	29 53	5 17	3 6	1 1	3	6 3
Dayton, Ohio	146	102	30	7	—	7	15	Pueblo, Colo.	38	23	10	3	1	1	3
Detroit, Mich.	218	127	61	20	6	4	18	Salt Lake City, Utah	134	94	22	12	4	2	11
Evansville, Ind. Fort Wayne, Ind.	69 62	49 45	15 13	5	3	1	6 4	Tucson, Ariz.	141	105	28	6	_	2	11
Gary, Ind.	22	43	7	5		1	-	PACIFIC	2,118	1,443	486	117	42	30	207
Grand Rapids, Mich.	76	55	11	4	3	3	4	Berkeley, Calif.	14	7	5	1		1	2
Indianapolis, Ind.	254	156	68	18	6	6	15	Fresno, Calif.	226	158	52	7	7	2	22
Lansing, Mich.	63	49	11	3		_	2	Glendale, Calif.	24	21	1		2	_	4
Milwaukee, Wis.	125	81	27	9	4	4	17	Honolulu, Hawaii	100	66	25	6	2	1	10
Peoria, III. Rockford, III.	53 67	37 52	10 11	2 4	_	4	3 9	Long Beach, Calif. Los Angeles, Calif.	75 338	53 213	19 78	1 33	1 9	1 5	10 36
South Bend. Ind.	52	32	11	2	1	1	3	Pasadena, Calif.	338 U	213 U	/8 U	33 U	U	U	30 U
Toledo. Ohio	121	79	30	7	2	3	9	Portland, Oreg.	186	133	41	8	3	1	17
Youngstown, Ohio	90	76	9	1	1	3	4	Sacramento, Calif.	215	138	55	12	4	6	30
W.N. CENTRAL	562	399	105	33	14	11	48	San Diego, Calif.	191	134	43	5	5	4	17
Des Moines, Iowa	54	46	6	1	1	_	14	San Francisco, Calif.	170	119	34	9	3	5	22
Duluth, Minn.	37	31	4	1	1	_	2	San Jose, Calif.	204	142	40	17	2	3	13
Kansas City, Kans.	19	14	4	_	1	_	1	Santa Cruz, Calif. Seattle, Wash.	25 147	17 90	7 42	1 11	3	1	4 11
Kansas City, Mo.	133	88	32	7	3	3	8	Spokane, Wash.	72	90 55	42 14	2	1		8
Lincoln, Nebr.	22	18	4	_		_	3	Tacoma, Wash.	131	97	30	4	_	_	1
Minneapolis, Minn.	89	59	16	7 U	1 U	6 U	12	TOTAL					206	200	
Omaha, Nebr. St. Louis, Mo.	U 69	U 39	U 17	6	6	1	U 1		13,373 ¹	9,061	2,916	828	286	200	1,078
St. Paul, Minn.	63	45	13	4	1	_	3								
Wichita, Kans.	76	59	9	7		1	4								

U: Unavailable. —: No reported cases. * Mortality data in this table are voluntarily reported from 122 cities in the United States, most of which have populations of ≥100,000. 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.

[§] Because of changes in reporting methods in this Pennsylvania city, these numbers are partial counts for the current week. Complete counts will be available in 4 to 6 weeks.

¹ Total includes unknown ages.

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 each week, send an e-mail message to *listserv@listserv.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/mmwr* or from CDC's file transfer protocol server at *ftp://ftp.cdc.gov/pub/publications/mmwr*. 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 E-96, CDC, 1600 Clifton Rd., N.E., Atlanta, GA 30333; telephone 888-232-3228.

All material in the MMWR Series is in the public domain and may be used and reprinted without permission; citation as to source, however, is appreciated.

All MMWR references are available on the Internet at http://www.cdc.gov/mmwr. Use the search function to find specific articles.

Use of trade names and commercial sources is for identification only and does not imply endorsement by the U.S. Department of Health and Human Services.

References to non-CDC sites on the Internet are provided as a service to *MMWR* readers and do not constitute or imply endorsement of these organizations or their programs by CDC or the U.S. Department of Health and Human Services. CDC is not responsible for the content of these sites. URL addresses listed in *MMWR* were current as of the date of publication.

☆U.S. Government Printing Office: 2005-733-116/00067 Region IV ISSN: 0149-2195