

Trends in the Prevalence of Excess Dietary Sodium Intake — United States, 2003–2010

Excess sodium intake can lead to hypertension, the primary risk factor for cardiovascular disease, which is the leading cause of U.S. deaths (1). Monitoring the prevalence of excess sodium intake is essential to provide the evidence for public health interventions and to track reductions in sodium intake, yet few reports exist. Reducing population sodium intake is a national priority, and monitoring the amount of sodium consumed adjusted for energy intake (sodium density or sodium in milligrams divided by calories) has been recommended because a higher sodium intake is generally accompanied by a higher calorie intake from food (2). To describe the most recent estimates and trends in excess sodium intake, CDC analyzed 2003–2010 data from the National Health and Nutrition Examination Survey (NHANES) of 34,916 participants aged ≥ 1 year. During 2007–2010, the prevalence of excess sodium intake, defined as intake above the Institute of Medicine tolerable upper intake levels (1,500 mg/day at ages 1–3 years; 1,900 mg at 4–8 years; 2,200 mg at 9–13 years; and 2,300 mg at ≥ 14 years) (3), ranged by age group from 79.1% to 95.4%. Small declines in the prevalence of excess sodium intake occurred during 2003–2010 in children aged 1–13 years, but not in adolescents or adults. Mean sodium intake declined slightly among persons aged ≥ 1 year, whereas sodium density did not. Despite slight declines in some groups, the majority of the U.S. population aged ≥ 1 year consumes excess sodium.

NHANES is a nationally representative, multistage survey of the noninstitutionalized U.S. civilian population. Certain populations are oversampled to allow for reliable estimates within subgroups.* During NHANES 2003–2010, a total of 49,731 participants aged ≥ 1 year (including those currently breastfed) were screened. Participants who completed an initial in-person dietary recall in a mobile examination center were asked to complete a second 24-hour dietary recall by telephone 3–10 days later. After those with missing or incomplete dietary recall data were excluded, the final analytic sample was 34,916,

for a response rate of 70.3% among those screened. The 24-hour dietary recall was collected by trained interviewers using the U.S. Department of Agriculture (USDA) automated multiple-pass method[†] by proxy for those aged 1–5 years, by participants with proxy assistance for those aged 6–11 years, and directly by participants aged ≥ 12 years. The nutrient values of sodium were assigned to foods and beverages using the USDA Food and Nutrient Database for Dietary Studies corresponding with each NHANES 2-year cycle.[§] Sodium intake for each respondent on each recall day was estimated by summing the sodium consumed from each food and beverage during the previous 24 hours (excluding supplements, antacids, and salt added at the table). To evaluate trends, from 2003–2010, estimates of sodium in foods did not include salt adjustments for participants whose household used salt in cooking occasionally or less often.[¶] For children consuming

[†] Additional information available at <http://www.ars.usda.gov/ba/bhnrc/fsrg>.

[§] Additional information available at <http://www.ars.usda.gov/services/docs.htm?docid=12089>.

[¶] Additional information available at <http://www.ncbi.nlm.nih.gov/pubmed/23567248>.

* Additional information available at <http://www.cdc.gov/nchs/nhanes.htm>.

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human milk, the sodium content was estimated and added to sodium from other foods and beverages.**

Up to two 24-hour dietary recalls were used. Data were analyzed with statistical software that fits a measurement error model.^{††} All estimates were based on usual sodium intake, adjusting for within person, day-to-day variability. After adjusting for the day of the week of the recall, age (years), sex, and race/ethnicity, estimates were calculated for mean usual sodium intake, sodium density, and prevalence of excess sodium intake. Jackknife replicate weights based on survey weights were used to estimate standard errors and account for the complex survey design. The differences in the prevalence of excess sodium intake were examined by z test. Using linear regression models with the usual mean intake for each 2-year phase weighted by the inverse of the variance, trends in sodium intake and sodium intake density were examined using a z test. A p-value of <0.05 was considered statistically significant. No adjustment was made for multiple testing.

** The volume of human milk was assumed to be 600 mL per day for children aged 7–11 months fed only human milk; 600 mL per day minus the volume of infant formula plus other milk for other children aged 7–11 months, 89 mL per human milk feeding for children aged 12–18 months, and 59 mL per feeding for children aged 19–36 months. Sodium, potassium, and energy concentrations in human milk were assumed to be 177 mg/L, 531 mg/L, and 75 kcal/L, respectively, based on the USDA National Nutrient Database for Standard Reference values for mature, human milk, 33.8 fluid ounces per liter.

†† PC-SIDE (Software for Intake Distribution Estimation for the Windows operating system), Center for Agriculture and Rural Development, Iowa State University. Additional information available at <http://www.side.stat.iastate.edu/pc-side.php> and <http://www.card.iastate.edu/publications/synopsis.aspx?id=168>.

What is already known on this topic?

Excess sodium intake can lead to hypertension and consequent cardiovascular disease. Sodium consumption in the United States is well above national recommendations. Reports of national data on sodium consumption trends are limited.

What is added by this report?

As of 2010, >90% of U.S. adolescents and adults consume sodium in excess of recommendations, and little has changed since 2003. U.S. children have seen a slight decline in excess sodium consumption during the same period, but 80%–90% of children continue to consume excess sodium. From 2003 to 2010, a slight decrease occurred in average sodium intake, but not sodium intake per calorie.

What are the implications for public health practice?

Small reductions in sodium intake might be related to declines in average energy consumption, rather than changes in the amount of sodium per calorie in foods consumed. Given that average energy and sodium intakes have changed little over time, coupling efforts to reduce obesity with efforts to reduce the sodium content per calorie in foods might accelerate reductions in sodium consumed.

During 2007–2010, the prevalence of excess usual sodium intake ranged from 79.1% for U.S. children aged 1–3 years to 95.4% for U.S. adults aged 19–50 years (Table 1). A statistically significant 2.7–4.9 percentage point decline in excess usual sodium intake occurred from 2003–2006 to 2007–2010

The *MMWR* series of publications is published by the Center for Surveillance, Epidemiology, and Laboratory Services, 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* 2013;62:[inclusive page numbers].

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TABLE 1. Proportion of usual sodium intake exceeding the Institute of Medicine tolerable upper intake level,* by age group, sex, and race/ethnicity† — National Health and Nutrition Examination Survey (NHANES), United States, 2003–2010

Characteristic	Upper limit (mg/day)	2003–2006			2007–2010			Percentage point change	p-value
		No. [§]	Proportion over upper intake level (%)	Standard error	No.	Proportion over upper intake level (%)	Standard error		
Age 1–3 yrs	1,500	1,560	(84.0)	1.4	1,558	(79.1)	1.9	(-4.9)	0.019 [¶]
Male		784	(84.1)	2.0	809	(79.4)	2.7	(-4.7)	0.081
Female		776	(84.3)	2.2	749	(79.7)	2.2	(-4.6)	0.071
White, non-Hispanic		470	(84.0)	2.9	525	(80.3)	3.7	(-3.7)	0.215
Black, non-Hispanic		407	(87.6)	3.3	297	(86.3)	3.0	(-1.3)	0.385
Mexican-American		519	(75.7)	3.2	437	(71.2)	4.9	(-4.5)	0.222
Age 4–8 yrs	1,900	1,682	(97.3)	0.4	1,890	(92.6)	0.8	(-4.6)	<0.001 [¶]
Male		815	(97.7)	0.5	995	(94.3)	1.0	(-3.4)	0.008 [¶]
Female		867	(96.9)	0.8	895	(90.5)	1.4	(-6.3)	<0.001 [¶]
White, non-Hispanic		479	(96.3)	0.8	621	(90.3)	1.5	(-5.9)	<0.001 [¶]
Black, non-Hispanic		519	(98.9)	0.7	402	(95.6)	1.3	(-3.3)	0.012 [¶]
Mexican-American		517	(94.2)	1.4	529	(89.3)	2.6	(-4.9)	0.045 [¶]
Age 9–13 yrs	2,200	2,040	(96.9)	0.7	1,717	(94.2)	0.9	(-2.7)	0.008 [¶]
Male		999	—**	—**	850	(96.8)	0.7	—††	—††
Female		1,041	(91.4)	1.6	867	(90.1)	1.7	(-1.4)	0.279
White, non-Hispanic		516	(97.0)	0.8	544	—**	—**	—††	—††
Black, non-Hispanic		691	—**	—**	406	—**	—**	—††	—††
Mexican-American		669	(95.4)	1.3	456	(84.8)	3.1	(-10.5)	0.001 [¶]
Age 14–18 yrs	2,300	2,673	(94.2)	1.0	1,552	(92.3)	1.5	(-1.9)	0.145
Male		1,353	(97.8)	0.7	818	—**	—**	—††	—††
Female		1,320	(84.2)	2.3	734	(80.2)	3.1	(-4.0)	0.938
White, non-Hispanic		731	(95.7)	1.0	517	(93.4)	1.7	(-2.3)	0.123
Black, non-Hispanic		938	(90.7)	1.8	369	—**	—**	—††	—††
Mexican-American		820	(94.3)	1.3	385	(90.0)	2.2	(-4.3)	0.047 [¶]
Age 19–50 yrs	2,300	5,428	(95.9)	0.4	6,086	(95.4)	0.5	(-0.5)	0.200
Male		2,528	(99.2)	0.1	2,936	(99.1)	0.2	(-0.1)	0.242
Female		2,900	(86.6)	1.2	3,150	(84.8)	1.4	(-1.9)	0.152
White, non-Hispanic		2,384	(97.1)	0.4	2,598	(96.4)	0.6	(-0.7)	0.170
Black, non-Hispanic		1,310	(92.5)	1.4	1,190	(93.4)	0.8	(0.9)	0.709
Mexican-American		1,276	(93.5)	1.0	1,270	(90.8)	1.3	(-2.8)	0.050
Age ≥51 yrs	2,300	4,062	(88.9)	1.0	4,668	(90.1)	0.8	(1.2)	0.839
Male		2,028	(95.9)	0.6	2,341	(96.5)	0.5	(0.6)	0.782
Female		2,034	(77.1)	1.4	2,327	(77.9)	1.4	(0.9)	0.668
White, non-Hispanic		2,416	(91.4)	0.9	2,273	(92.8)	0.8	(1.4)	0.876
Black, non-Hispanic		762	(79.0)	2.4	975	(82.2)	2.0	(3.2)	0.842
Mexican-American		674	(67.7)	3.9	757	(76.3)	3.1	(8.6)	0.959

* The upper intake level is the age-specific, tolerable upper intake level, as defined by the Institute of Medicine (2005). The proportion of usual sodium intake over the upper intake level was estimated using PC-SIDE software (Department of Statistics, Iowa State University) with jackknife replicate weights and adjusted for the day of the week of the recall, age (years), sex, and race/ethnicity. Persons missing data on incomplete first-day recall were excluded from the analysis.

† Other racial/ethnic groups were not included. The sum of the sample size of non-Hispanic white, non-Hispanic black, and Mexican-American is not equal to the total sample size.

§ Sample sizes unweighted.

¶ $p < 0.05$, when trends of proportion of usual sodium intake over the upper intake level were examined using the z test.

** Data statistically unreliable; relative standard error ≥ 0.3 .

†† Not applicable.

among children aged 1–3, 4–8, and 9–13 years, but not among adolescents or adults. Among children aged 4–8 years, statistically significant declines occurred across all sex and race/ethnicity subgroups.

Mean usual sodium intake among the U.S. population aged ≥ 1 year decreased slightly from 2003–2004 to 2009–2010 (3,518 mg versus 3,424 mg; p -value for trend = 0.037). The U.S. population aged ≥ 1 year consumed, on average, approximately 1,700 mg sodium per 1,000 kcal during 2009–2010,

with no significant trend over time compared with previous investigation years (Table 2). Across age groups, mean usual sodium density did not change significantly over time, with the exception of youths aged 14–18 years, for whom sodium density increased slightly. Within age groups, mean usual sodium density slightly increased among males aged 4–8 years and females aged 14–18 years and slightly declined among non-Hispanic whites aged ≥ 51 years.

TABLE 2. Mean usual sodium density* (mg/1,000 kcal), by age group, sex, and race/ethnicity† — National Health and Nutrition Examination Survey (NHANES), United States, 2003–2010

Characteristic	2003–2004			2005–2006			2007–2008			2009–2010			Changes per cycle [¶]	p-value for trend
	No. [§]	Mean	Standard error	No.	Mean	Standard error	No.	Mean	Standard error	No.	Mean	Standard error		
Overall	8,579	1,661	10	8,866	1,693	14	8,473	1,697	12	8,998	1,689	10	9	0.248
Male	4,192	1,653	9	4,315	1,666	14	4,266	1,695	15	4,483	1,690	14	14	0.054
Female	4,387	1,669	17	4,551	1,719	17	4,207	1,698	16	4,515	1,688	13	2	0.879
White, non-Hispanic	3,541	1,679	10	3,455	1,710	14	3,367	1,698	11	3,711	1,692	10	4	0.560
Black, non-Hispanic	2,284	1,617	26	2,343	1,637	14	1,939	1,664	21	1,700	1,632	17	5	0.652
Mexican-American	2,123	1,548	15	2,352	1,569	16	1,773	1,582	16	2,061	1,581	28	13	0.063
Age 1–3 yrs	740	1,431	21	820	1,458	34	765	1,429	23	793	1,427	15	-3	0.589
Male	363	1,404	32	421	1,472	46	399	1,392	34	410	1,419	25	0	0.993
Female	377	1,457	31	399	1,433	20	366	1,463	27	383	1,433	22	-3	0.727
White, non-Hispanic	226	1,435	25	244	1,472	36	246	1,399	36	279	1,434	34	-5	0.729
Black, non-Hispanic	218	1,500	30	189	1,464	34	163	1,497	29	134	1,479	75	-3	0.840
Mexican-American	228	1,364	49	291	1,343	31	207	1,368	46	230	1,360	47	3	0.695
Age 4–8 yrs	783	1,541	19	899	1,550	19	934	1,530	20	956	1,556	23	2	0.822
Male	382	1,491	20	433	1,531	21	500	1,544	31	495	1,573	41	27	0.028**
Female	401	1,594	29	466	1,567	28	434	1,518	24	461	1,541	21	-18	0.252
White, non-Hispanic	220	1,545	31	259	1,522	28	300	1,480	26	321	1,546	37	-7	0.747
Black, non-Hispanic	261	1,574	42	258	1,614	40	230	1,620	32	172	1,568	32	-3	0.840
Mexican-American	224	1,434	34	293	1,491	23	250	1,524	31	279	1,487	31	3	0.695
Age 9–13 yrs	995	1,601	23	1,045	1,633	16	832	1,637	32	885	1,636	19	9	0.292
Male	482	1,580	35	517	1,640	29	411	1,647	40	439	1,665	30	25	0.102
Female	513	1,622	34	528	1,627	39	421	1,625	45	446	1,613	27	-3	0.269
White, non-Hispanic	266	1,568	28	250	1,648	25	252	1,638	45	292	1,635	23	17	0.370
Black, non-Hispanic	350	1,750	58	341	1,685	39	224	1,722	48	182	1,599	30	-44	0.140
Mexican-American	301	1,520	45	368	1,613	27	206	1,514	64	250	1,598	38	12	0.700
Age 14–18 yrs	1,343	1,567	26	1,330	1,636	39	738	1,683	36	814	1,689	30	43	0.036**
Male	697	1,594	33	656	1,638	50	385	1,721	38	433	1,678	37	35	0.143
Female	646	1,535	31	674	1,625	36	353	1,644	36	381	1,698	37	54	0.036**
White, non-Hispanic	360	1,586	33	371	1,639	48	247	1,717	47	270	1,675	38	34	0.137
Black, non-Hispanic	488	1,542	42	450	1,531	27	195	1,594	50	174	1,609	25	27	0.137
Mexican-American	411	1,551	31	409	1,607	28	165	1,656	70	220	1,631	58	36	0.104
Age 19–50 yrs	2,583	1,657	17	2,845	1,717	20	2,865	1,718	14	3,221	1,708	11	12	0.345
Male	1,226	1,651	21	1,302	1,687	22	1,404	1,712	15	1,532	1,703	20	18	0.163
Female	1,357	1,660	25	1,543	1,742	29	1,461	1,723	22	1,689	1,712	17	10	0.527
White, non-Hispanic	1,189	1,663	21	1,195	1,729	25	1,188	1,720	18	1,410	1,709	15	11	0.432
Black, non-Hispanic	633	1,603	50	677	1,641	30	623	1,664	31	567	1,636	22	5	0.697
Mexican-American	560	1,578	17	716	1,598	25	598	1,602	15	672	1,601	30	9	0.113
Age ≥51 yrs	2,135	1,778	17	1,927	1,759	16	2,339	1,768	23	2,329	1,748	20	-8	0.159
Male	1,042	1,784	25	986	1,712	24	1,167	1,768	25	1,174	1,760	36	-3	0.904
Female	1,093	1,775	20	941	1,799	19	1,172	1,767	27	1,155	1,736	24	-15	0.290
White, non-Hispanic	1,280	1,799	18	1,136	1,771	17	1,134	1,752	17	1,139	1,738	25	-21	0.012**
Black, non-Hispanic	334	1,671	29	428	1,689	30	504	1,726	32	471	1,697	34	12	0.354
Mexican-American	399	1,637	45	275	1,567	47	347	1,657	42	410	1,631	31	4	0.809

* Sodium intake density was calculated as sodium intake divided by daily calories. Mean usual sodium intake density was estimated using PC-SIDE software (Department of Statistics, Iowa State University) with jackknife replicate weights and adjusted for the day of the week of the recall, age (years), sex, and race/ethnicity. Persons missing first-day recall data were excluded.

† Other racial/ethnic groups were not included. The sum of the sample size of non-Hispanic white, non-Hispanic black, and Mexican-American is not equal to the total sample size.

§ Sample sizes are unweighted.

¶ Mean change in sodium density per 2-year cycle (mg/1,000 kcal) estimated from a linear regression model with the usual mean sodium density for each 2-year phase weighted by the inverse of the variance.

** p<0.05, when mean usual sodium intake density was examined by using linear regression model.

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Editorial Note

The findings in this report indicate that during 2007–2010, approximately eight out of 10 U.S. children aged 1–3 years and nine out of 10 U.S. residents aged ≥4 years were at potential risk for high blood pressure attributable to excess sodium intake. Although a slight decrease in the prevalence of excess usual sodium intake occurred after 2003–2006 among children aged 1–13 years, excess intake did not decrease among adolescents and adults. During 2003–2010, a slight decrease occurred in average population sodium intake, but not sodium intake per calorie. Although some variation in trends occurred among population subgroups in usual mean sodium intake and sodium density, the lack of a change in sodium consumed per calorie (approximately 1,700 mg/1,000 kcal) suggests that the small reduction in usual sodium intake might be related to declines in calorie consumption, rather than to changes in sodium density of foods.

Previous reports (4,5) included data on trends in U.S. sodium intake from the 1970s to 2003. The findings in this report update these trends, and include new data on usual excess sodium intake and sodium density. The slight declines in excess usual sodium intake among children aged 1–13 years might be partially explained by declines in energy intake among children over the same period.^{§§} Given an average sodium consumption of 1,700 mg/1,000 kcal/day, reducing 100 calories per day could result in a mean reduction of 170 mg of sodium per day, slightly shifting the distribution of sodium intake and lowering the percentage of those with excess intake. Among adults, the pattern of trends in sodium intake also might be explained by changes in energy intake over time. Although average energy intake declined slightly during 1999–2010 among adults aged 20–39 years, it did not change among older adults (6).

The findings in this report are subject to at least four limitations. First, NHANES data exclude military personnel and institutionalized populations such as persons who reside in long-term care or correctional facilities. Second, the response rate was 70.3%; lower response rates can result in response bias. Third, the 24-hour dietary recall underestimates mean caloric intake by an estimated 11% and sodium intake by 9%, and sodium intake excluded use of salt at the table, which accounts for nearly 5% of U.S. sodium intake (7). Finally, no adjustments for multiple comparisons were performed to determine whether differences between any pair of estimates were statistically significant.

^{§§} Additional information available at <http://www.cdc.gov/nchs/data/databriefs/db113.htm>.

Despite slight declines in sodium intake among some population groups, most U.S. residents aged ≥1 year consume excess sodium. Given consumption of approximately 1,700 mg of sodium per 1,000 kilocalories/day, a mean energy reduction of approximately 600 kcal/day would be required to reduce mean sodium intake by approximately 1,000 mg, to approximately 2,300 mg/day. A sodium density target of 1,000 mg/1,000 kcal was recently proposed to lower sodium intake to <2,300 mg per day (2). Given that average energy and sodium intakes have changed little over time, coupling efforts to reduce obesity with efforts to reduce the sodium content per calorie in foods might accelerate progress. Considering that 8.1% of sodium intake among U.S. children comes from school meals (8), new school food guidelines might promote progress toward achieving goals for reducing sodium consumption among children who obtain meals at school.^{¶¶} Other ongoing public health efforts include working with industry to gradually reduce sodium in commercially processed packaged and restaurant foods.^{***} Even a 400 mg reduction in mean U.S. sodium intake might save billions of health-care dollars (9).

^{¶¶} Additional information available at <http://www.gpo.gov/fdsys/pkg/FR-2012-01-26/html/2012-1010.htm>.

^{***} Additional information available at http://www.scielo.org/scielo.php?script=sci_arttext&pid=S1020-49892012001000009&lng=en&nrm=iso&tlng=en.

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Outbreak of Staphylococcal Food Poisoning from a Military Unit Lunch Party — United States, July 2012

On July 30, 2012, the emergency department at a military hospital was visited by 13 persons seeking care for gastrointestinal illness with onset 2–3 hours after a work lunch party. The hospital responded by opening up temporary evaluation and treatment capacity in primary-care clinics and a progressive-care unit and by diverting one patient to a local civilian hospital. An immediate outbreak investigation was conducted by local military public health personnel with assistance from CDC. Initial epidemiologic analysis implicated “perlo” (a chicken, sausage, and rice dish) and bacterial intoxication as the outbreak mechanism. This enabled public health personnel to 1) recommend no further consumption of perlo and 2) reassure appropriate authorities that no additional ill persons likely would be seeking care and advise that nothing more than supportive care of ill persons likely would be required. After interviewing party attendees, investigators found nine additional persons who met their case definition. Subsequent CDC laboratory analysis of a sample of perlo detected staphylococcal enterotoxin A, supporting the epidemiologic findings. Improper food handling and preparation measures were identified and addressed by the appropriate authorities, who provided additional detailed education on food preparation safety for the persons who prepared the meal.

Epidemiologic and Environmental Investigation

Immediate steps were taken to interview the 12 patients who received care at the military hospital (one of the 13 who sought care at the emergency department was diverted to a local civilian hospital) using a questionnaire to define the problem, make public health and clinical recommendations as necessary, and assess future patient, hospital, and military installation impact. A case initially was defined as gastrointestinal illness (i.e., nausea, vomiting, abdominal pain, or diarrhea) in a person who had attended the lunch party. Stool specimens were obtained from three persons who met this case definition, and leftover food was collected from all prepared dishes and sent to CDC for analysis. A party attendee list was obtained, and 35 (88%) of 40 attendees were interviewed with the same questionnaire; the other five attendees were not available for interview. The case definition was later modified to include any of the 35 attendees who experienced gastrointestinal illness within 24 hours of the party.

Of the 35 persons interviewed, 22 (62%) met the modified case definition. Among the 22 patients, 19 (86%) had nausea, 15 (69%) vomiting, 17 (77%) diarrhea, 17 (77%) abdominal

pain, and 13 (59%) headache. Thirteen (59%) reported feeling chills without fever, four (18%) reported fever with chills. None of the 12 persons who received care at the military hospital had a documented temperature >100.5°F (>38.1°C). Other than the 13 persons who sought care at the military hospital (one was diverted to a civilian hospital), no other person who was interviewed and met the case definition sought medical care. Mean self-reported period to illness onset was 2.1 hours from the time of consumption (Figure), and mean duration of illness was 10.7 hours (range: 1–32 hours).

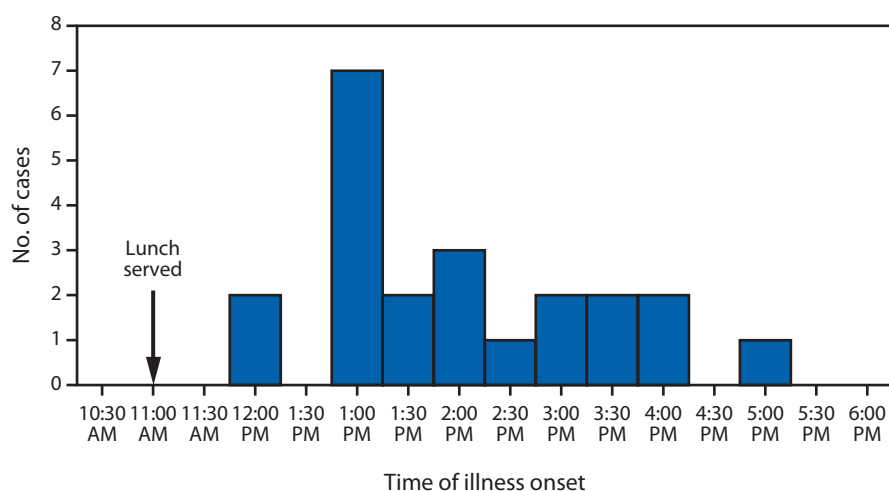
Illness was associated with eating perlo (risk ratio = 5.7); however, the association did not reach statistical significance (95% confidence interval = 0.9–35.0) (Table). The stool specimens from three patients and samples from the four main dishes (i.e., perlo, chicken wings, pulled pork, and green beans with potatoes) were sent to CDC for laboratory testing for the most likely bacterial organisms based on the epidemiologic investigation (*Staphylococcus aureus*, *Bacillus cereus*, and *Clostridium perfringens*). Using commercially available rapid detection test kits, laboratory workers tested all of the four main dishes for the presence of staphylococcal enterotoxins (A through E) and *B. cereus* diarrheal toxins (HBL and NHE). The three stool specimens were tested for *C. perfringens* enterotoxin (CPE).

Laboratory testing detected staphylococcal enterotoxin A in the perlo dish, confirming that this outbreak resulted from a staphylococcal intoxication. Evidence of enterotoxin was not found in the other foods. Perlo was cultured for *S. aureus*, and 7.2×10^6 colony forming units/g were isolated. Nucleic acid amplification testing of coagulase-positive isolates detected the enterotoxin gene *sea*, but not *seb*, *sec*, *sed*, *see*, or *seh* (1). Testing for *B. cereus* and *C. perfringens* was negative.

Food Preparation Findings

Food for the July 30 lunch party was purchased during July 27–28, with the exception of the pork, which was purchased approximately 1 week earlier. All perishable products (chicken thighs, chicken wings, breakfast sausage, and pork) were stored in a freezer.

Perlo. On July 29, the raw chicken thighs and sausage were defrosted in the microwave. The defrosted chicken thighs were cooked in a stock pot of boiling water. After cooling, the chicken was removed from the thigh bones by hand and placed back into the stock pot. Sausage was cooked in a skillet and added to the pot. Onions and other seasonings were sautéed in the sausage oil and added to the pot. To complete the perlo,

FIGURE. Number of cases (N = 22) of gastrointestinal illness resulting from a military unit lunch party, by time of illness onset — United States, July 2012

rice was added to the stock pot and cooked until all remaining water was absorbed. The pot of cooked perlo then was placed in an unheated oven for approximately 8 hours overnight. On the morning of July 30, the perlo was found to be warm. It was transferred to a slow cooker for reheating for approximately 1 hour on a high setting before transport and consumption.

Chicken wings. On July 28, the raw chicken wings were defrosted in the refrigerator overnight. On July 29, they were seasoned and placed back in the refrigerator. On the morning of July 30, they were fried and placed in a foil-covered pan for transport and consumption on July 30.

Pulled pork. The raw pork was defrosted in the microwave on July 29 and cooked overnight in a slow cooker for approximately 8 hours on a low setting before transport and consumption on July 30.

Green beans with potatoes. On the morning of July 30, the green bean and potato dish was prepared by combining canned green beans and chopped potatoes in a slow cooker on a high setting.

All precooked food was maintained in slow cookers for transport on July 30 and reheated at the venue in separate slow cookers with the exception of the chicken wings, which were placed in a foil container after preparation and never reheated. The food was served buffet style at approximately 11:00 a.m., using utensils owned by the preparer. Throughout the food preparation and food service process, food preparers did not wear gloves. The preparers did not have any open wounds on their hands, and their hands were not tested for any organisms.

Reported by

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Editorial Note

Staphylococcal food poisoning, one of the most common foodborne illnesses in the United States, is caused by ingestion of one or more preformed staphylococcal enterotoxins. *S. aureus* is able to grow and express enterotoxins in a wide variety of foods (e.g., milk, meat, and egg products; mixed foods; cakes; and ice cream) (2). Intoxication is characterized by rapid onset of nausea, violent and copious vomiting, and abdominal cramping (with or without diarrhea). Fever usually is absent. The incubation period ranges from 30 minutes to 8 hours (3 hours on average), depending on individual susceptibility and the amount of toxin ingested (3). Illness usually is self-limited, resolving in 24–48 hours, and rarely is severe enough to warrant hospitalization (2,4).

TABLE. Attack rates and risk ratios comparing cases of gastrointestinal illness (N = 22) associated with a military unit lunch party, by food type — United States, July 2012

Food type	Ate the food			Did not eat the food			Risk ratio	(95% CI)
	No. ill	No. well	Attack rate (%)	No. ill	No. well	Attack rate (%)		
Perlo*	21	5	(81)	1	6	(14)	5.7	(0.9–35.0)
Chicken wings	17	11	(61)	5	0	(100)	0.6	(0.5–0.8)
Green beans with potatoes	11	3	(79)	11	8	(58)	1.4	(0.9–2.2)
Pulled pork	17	8	(68)	5	3	(63)	1.1	(0.6–2.0)
Chocolate-covered strawberries	4	4	(50)	18	7	(72)	0.7	(0.3–1.5)
Cake	7	6	(54)	15	5	(75)	0.7	(0.4–1.3)

Abbreviation: CI = confidence interval.

* Perlo is a chicken, sausage, and rice dish.

What is already known on this topic?

Staphylococcus aureus intoxication is a common foodborne illness that usually is not detected or reported because outbreaks are sudden and short-lived, have a low mortality rate, and laboratory confirmation is not obtained. Staphylococcal enterotoxin A is the most common cause of staphylococcal food poisoning.

What is added by this report?

This report describes 22 cases of staphylococcal intoxication associated with a lunch party at a military base. Epidemiologic analysis suggested a preformed enterotoxin in a chicken, sausage, and rice dish. Isolation of *S. aureus* along with identification of staphylococcal enterotoxin A in food confirmed the cause of illness. Rapid detection methods, which are widely available commercially, were used to detect the enterotoxin in food samples, establishing the likely cause of the outbreak before culture results were available.

What are the implications for public health practice?

This report highlights the importance of immediate public health outbreak response, adds to the understanding of food poisoning caused by *S. aureus*, and confirms the need to communicate better food safety practices to both food workers and the general public. Laboratory use of toxin detection kits can provide rapid identification of staphylococcal enterotoxins directly from food and help guide the response of public health authorities.

S. aureus produces numerous serologically distinct enterotoxins, (A through V, excluding F) (2). Staphylococcal enterotoxin A is considered the main cause of staphylococcal food poisoning (4). Illness can be caused by ingestion of as little as 20 ng of enterotoxin (2). Although staphylococci commonly are found on environmental surfaces and in various food products, food handlers carrying enterotoxin-producing *S. aureus* in their noses or on their hands are regarded as the main source of contamination (2,5).

Temperature limits for *S. aureus* replication and enterotoxin production are 43°F–118°F (6°C–48°C) and 50°F–115°F (10°C–46°C), respectively. Optimal growth temperatures for *S. aureus* replication and enterotoxin production are 95°F–106°F (35°C–41°C) and 93°F–104°F (34°C–40°C), respectively (2). Staphylococcal enterotoxins are resistant to heat treatment, low pH, and proteolytic enzymes (all of which easily destroy *S. aureus*). Once toxins are produced, they are retained through subsequent food preparation and storage processes and digestive tract ingestion (2). Measures to prevent the proliferation of the *S. aureus* organism therefore are critical.

In this outbreak, the initial source of contamination of the perlo is unknown but might have occurred while the preparer was handling the chicken after it was initially cooked. The overnight storage of the precooked perlo in an unrefrigerated environment was the probable cause of organism proliferation

and enterotoxin production. Subsequent rewarming of the perlo for approximately 1 hour the following day did not destroy the heat-stable toxin and might have further increased toxin load.

Toxin detection kits are commercially available and can detect staphylococcal enterotoxins A through E directly from food. The commercial testing platforms usually are passive agglutination or enzyme-linked immunosorbent assays (6). The *B. cereus* and *S. aureus* enterotoxin tests used in this outbreak are only approved for food samples; the test for the *C. perfringens* enterotoxin is approved only for stool specimens. The commercial kits were helpful in the identification of *S. aureus* toxin as the cause of this outbreak and might be useful for state and local public health agencies.

In this outbreak, as in many others, poor food handling practices and inadequate refrigeration of foods were identified as the main contributing factors (2,7). Staphylococcal contamination and subsequent intoxication can be prevented by ensuring clean food preparation, storage, and equipment surfaces, and by immediately cooling and storing prepared “potentially hazardous” foods at temperatures below 41°F (5°C). To permit rapid cooling after preparation, food should be stored in small portions in containers that are shallow and loosely covered to facilitate adequate air flow and rapid transfer of heat from the food to the container (8). Consuming food immediately after preparation or removal from refrigeration also is advisable. Finally, handwashing for 20 seconds using soap and water before handling food and food preparation and storage materials is a simple but effective preventive measure to avoid staphylococcal contamination.

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Outbreak of *Escherichia coli* O104:H4 Infections Associated with Sprout Consumption — Europe and North America, May–July 2011

In May 2011, public health authorities in Europe began investigating an outbreak of Shiga toxin–producing *Escherichia coli* (STEC) O104:H4 infections that ultimately involved more than 4,000 persons in 16 countries. Early in the outbreak, it became evident that international surveillance would be necessary to determine the scope of the outbreak, characterize the disease, and identify the source. This report describes surveillance conducted in the United States, which involved active case-finding, use of laboratory testing protocols specific to non-O157 STEC, interviews to identify potential exposures of interest, and documentation of clinical courses. Six cases in the United States were associated with the outbreak. Although European epidemiologic studies, including analyses of restaurant cohorts and traceback investigations, ultimately implicated raw fenugreek sprouts as the food vehicle, none of the patients in the United States definitively recalled sprout consumption. These events highlight challenges in investigating outbreaks, particularly those caused by rare pathogens or associated with food vehicles that are consumed in small quantities as part of other dishes. Clinical laboratories should adhere to STEC testing recommendations because they are critical for identification of rare or novel STEC pathogens. Robust public health infrastructure is necessary to effectively manage and resolve foodborne outbreaks.

On May 19, 2011, the Robert Koch Institute in Germany was notified of a cluster of three patients with hemolytic uremic syndrome (HUS) admitted to a single hospital in Hamburg (1). Enteroaggregative STEC O104:H4, a rare pathogen to which few human illnesses had been previously attributed (2), was isolated from patient specimens. Illness onsets began in early May, and cases initially were concentrated in northern Germany. Over the next few weeks, case counts mounted rapidly in Germany, and by June 1, approximately 1,534 cases were identified, including 470 (31%) cases complicated by HUS (3). New cases were quickly identified throughout Europe and elsewhere in persons who had recently traveled in Europe. On May 25, German public health authorities made an International Health Regulations notification to the World Health Organization regarding the substantial rise in STEC O104:H4 cases, and the U.S. Department of State notified CDC. Surveillance was conducted in the United States to assist in determining the extent of the outbreak and to identify case clusters that could be investigated for common food or environmental exposures.

Restaurant cohort studies and traceback investigations conducted in Germany implicated mixed raw sprouts from one farm in Germany, and the product was recalled on June 10 (4). On June 24, France reported a cluster of diarrheal illnesses and HUS cases among attendees at an event near Bordeaux. Ten persons had culture-confirmed STEC O104:H4 infection, and seven developed HUS (5). Ill persons reported consuming locally grown raw sprouts, and subsequent European Food Safety Authority traceback investigations identified one lot of fenugreek seeds imported from Egypt as the source of the sprouts responsible for the outbreaks in Germany and France (6).

On May 26, CDC initiated active surveillance for cases in the United States associated with this outbreak. Objectives included 1) identification of any travel and food consumption commonalities among patients, 2) ascertainment of information about clinical courses of patients, and 3) microbiologic characterization of isolates from patient specimens. CDC asked states to report all cases of STEC diarrheal illness or HUS associated with recent travel to Germany. Announcements were sent electronically via CDC's Foodborne Outbreak Listserv and Epidemic Information Exchange (Epi-X) web communication network. Additionally, a health advisory for clinicians was distributed via CDC's Health Alert Network. A suspected primary case was defined as HUS or Shiga toxin–positive diarrheal illness in a person who had traveled to Germany after April 1, 2011, and illness onset either during travel in Germany or within 3 weeks after returning from Germany. A suspected secondary case was defined as HUS or diarrheal illness in a person who had not traveled to Germany within 3 weeks of illness, but who had contact with a person with a confirmed case. Cases were confirmed when STEC O104:H4 with a pulsed-field gel electrophoresis (PFGE) pattern matching the outbreak strain was isolated from a clinical specimen.

STEC O104:H4 was isolated at state public health laboratories or at CDC from Shiga toxin–positive stool enrichment broths submitted by clinical laboratories. Isolates were subtyped by PFGE in PulseNet, the national subtyping network for foodborne disease surveillance, and characterized at CDC with serotyping, antimicrobial susceptibility testing, and virulence profiling. Patients were contacted to complete food exposure questionnaires designed and provided by the Robert Koch Institute and to undergo open-ended interviews regarding food consumption and environmental exposures during the 3 weeks before illness onset. In instances where the patient was

What is already known on this topic?

Although Shiga toxin–producing *Escherichia coli* (STEC) O157 is an often identified pathogen, illnesses involving non-O157 serogroups are increasingly recognized. During May–July 2011, a large outbreak of STEC O104:H4 occurred in Europe and North America that was associated with consumption of raw fenugreek sprouts. In addition to producing Shiga toxin, the strain had the characteristics of the enteroaggregative *E. coli* pathotype. This combination of virulence characteristics seems to have increased the pathogenicity of the strain.

What is added by this report?

Surveillance conducted in the United States during the outbreak identified six cases associated with the outbreak; four were complicated by hemolytic uremic syndrome. These are the first documented cases of STEC O104:H4 in the United States. None of the U.S. patients recalled consumption of sprouts, the outbreak vehicle.

What are the implications for public health practice?

Adherence to recommended STEC diagnostic testing is critical in detecting illnesses and outbreaks attributable to non-O157 STEC. High-quality public health infrastructure allows for comprehensive responses and rapid control of outbreaks that might involve similarly rare pathogens.

too ill to undergo interview, travel companions were identified and questioned as proxies. To detect any unusual food items or other exposures in common, a single-interviewer strategy was employed.

During May 26–June 16, six confirmed cases were identified in five states: Arizona (one), Massachusetts (one), Michigan (two), North Carolina (one), and Wisconsin (one). Ages of patients ranged from 38 to 72 years (median: 52 years); two patients were female. Five reported travel to or from Germany in the 3 weeks before their illness; the additional secondary case was in a close relative of a patient who had traveled. Patients reported consumption of various fresh produce items while in Germany, such as tomatoes, lettuce, and cucumbers. However, none recalled consumption of sprouts, the food vehicle ultimately implicated in the outbreak. All patients had diarrhea, including four (66%) with bloody diarrhea. Four (66%) patients were reported by physicians to have developed HUS, requiring dialysis and ventilator support. One patient died.

Microbiologic characterization of the pathogens isolated from clinical specimens demonstrated STEC O104:H4 within all specimens, with a PFGE pattern indistinguishable from the outbreak strain. This strain combines features of the STEC and enteroaggregative (EAEC) pathotypes of *Escherichia coli*. All isolates were positive for the *stx2a* gene, which encodes a Shiga toxin variant often associated with severe *E. coli* illness, such as bloody diarrhea or HUS (7). Additionally, all isolates

were positive for the *aggR* gene, which encodes a regulator of virulence plasmid and chromosomal genes and is characteristic of EAEC (7). The strains were negative for the *eae* gene, which encodes a mucosal adherence protein in most STEC (7). Isolates from the six patients had almost identical antimicrobial resistance profiles; all were resistant to ampicillin, ceftriaxone, streptomycin, sulfisoxazole, tetracycline, and trimethoprim/sulfamethoxazole.

On July 5, 2011, the European Union banned importation of fenugreek seeds and various other seeds, beans, and sprouts from Egypt. Worldwide surveillance continued for an additional 3 weeks, but no new cases were identified. On July 26, public health authorities in Germany declared the outbreak to be over. The final case count was 4,075 cases (including 908 cases complicated by HUS) and 50 deaths in 16 countries (3).

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Editorial Note

This report summarizes the second-largest STEC outbreak worldwide and includes the first documented STEC O104:H4 illnesses in the United States (1). Although only six cases were identified in the United States, the clinical severity of the illnesses associated with the strain warranted aggressive surveillance. Identifying and investigating these cases allowed for more complete understanding of this uncommon pathogen. Previous surveillance indicates that HUS complicates approximately 6% of STEC O157:H7 infections (1, 2); however, HUS occurred in 66% of U.S. cases and 22% of worldwide cases (including the U.S. cases) during this outbreak, indicating that the outbreak strain might have been especially virulent. Surveillance provided important information regarding disease transmission; one instance of secondary transmission was documented among U.S. cases. Additionally, investigation of these cases reinforced the utility of the recipe-based restaurant analytic studies employed by German investigators. Inability to recall sprout consumption was apparent in U.S. and German

patients. In early interviews, only 25% of German HUS patients reported eating sprouts in the 2 weeks before their illness (4). Limited recall for food items consumed as ingredients within dishes has proved challenging in other foodborne outbreak investigations, particularly outbreaks involving raw produce (9). Restaurant-based investigations allow for the identification of a cohort with exposure to a limited menu of dishes and ingredients that can be exhaustively scrutinized for commonalities, addressing the problem of incomplete recall of so-called “stealth vehicles” (9).

This outbreak also highlights the importance of adherence to laboratory testing recommendations in the identification of outbreak pathogens, particularly when the outbreak involves rare or novel pathogens. CDC’s recommended protocols for routine testing of acute community-acquired diarrhea specimens call for assays to detect Shiga toxin, simultaneous culture on selective and differential agar to distinguish STEC O157, which accounts for approximately half of all laboratory-confirmed STEC infections in the United States (10), and additional testing of Shiga toxin–positive specimens at public health laboratories to identify non-O157 STEC (10). Recent evaluations of laboratories in the United States involved in processing diarrhea specimens indicated that 22% of surveyed laboratories adhered to recommendations.* More complete adherence would allow for better detection and identification of both O157 and non-O157 illness-causing STEC. Prompt and accurate pathogen identification and diagnosis is critical for both patient and outbreak management.

Effectively responding to foodborne disease outbreaks, such as the outbreak described in this report, relies on having robust public health infrastructure in place. Such infrastructure, including systematic disease surveillance, laboratory capacity, and the ability to conduct epidemiologic and traceback investigations, is essential for maintaining a safe food

supply. Sustaining and enhancing capacity to conduct these activities, both internationally and domestically, will be critical in confronting future challenges related to known and novel pathogens.

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*Additional information available at <https://idsa.confex.com/idsa/2012/webprogram/Paper37586.html>.

Update: Influenza Activity — United States, September 29–December 7, 2013

CDC collects, compiles, and analyzes data on influenza activity year-round in the United States (<http://www.cdc.gov/flu/weekly/fluactivitysurv.htm>). The influenza season generally begins in the fall and continues through the winter and spring months; however, the timing and severity of circulating influenza viruses can vary by geographic location and season. Influenza activity in the United States continued to increase from mid-November through the beginning of December. This report summarizes U.S. influenza activity* during September 29–December 7, 2013.†

Viral Surveillance

During September 29–December 7, 2013, approximately 140 World Health Organization (WHO) and National Respiratory and Enteric Virus Surveillance System collaborating laboratories in the United States tested 61,261 respiratory specimens for influenza viruses; 4,183 (6.8%) were positive (Figure 1). Of these, 3,819 (91.3%) were influenza A viruses, and 364 (8.7%) were influenza B viruses. Of the 3,819 influenza A viruses, 1,998 (52.3%) were subtyped; 198 (10%) of these were influenza A (H3) viruses, and 1,800 (90%) were influenza A (H1N1)pdm09 (pH1N1) viruses. Since September 29, 2013, influenza-positive tests have been reported from 48 states, the District of Columbia, and Puerto Rico, representing all 10 U.S. Department of Health and Human Services (HHS) regions.§ Thus far, influenza A viruses have predominated nationally and in all 10 HHS regions.

*The CDC influenza surveillance system collects five categories of information from eight data sources: 1) viral surveillance (World Health Organization collaborating laboratories, the National Respiratory and Enteric Virus Surveillance System, and novel influenza A virus case reporting); 2) outpatient illness surveillance (U.S. Outpatient Influenza-Like Illness Surveillance Network); 3) mortality (122 Cities Mortality Reporting System and influenza-associated pediatric mortality reports); 4) hospitalizations (Influenza Hospitalization Surveillance Network [FluSurv-NET], which includes the Emerging Infections Program and surveillance in three additional states); and 5) summary of the geographic spread of influenza (state and territorial epidemiologist reports).

† Data reported as of December 13, 2013.

§ *Region 1:* Connecticut, Maine, Massachusetts, New Hampshire, Rhode Island, and Vermont. *Region 2:* New Jersey, New York, Puerto Rico, and the U.S. Virgin Islands. *Region 3:* Delaware, District of Columbia, Maryland, Pennsylvania, Virginia, and West Virginia. *Region 4:* Alabama, Florida, Georgia, Kentucky, Mississippi, North Carolina, South Carolina, and Tennessee. *Region 5:* Illinois, Indiana, Michigan, Minnesota, Ohio, and Wisconsin. *Region 6:* Arkansas, Louisiana, New Mexico, Oklahoma, and Texas. *Region 7:* Iowa, Kansas, Missouri, and Nebraska. *Region 8:* Colorado, Montana, North Dakota, South Dakota, Utah, and Wyoming. *Region 9:* Arizona, California, Hawaii, Nevada, American Samoa, Commonwealth of the Northern Mariana Islands, Federated States of Micronesia, Guam, Marshall Islands, and Republic of Palau. *Region 10:* Alaska, Idaho, Oregon, and Washington.

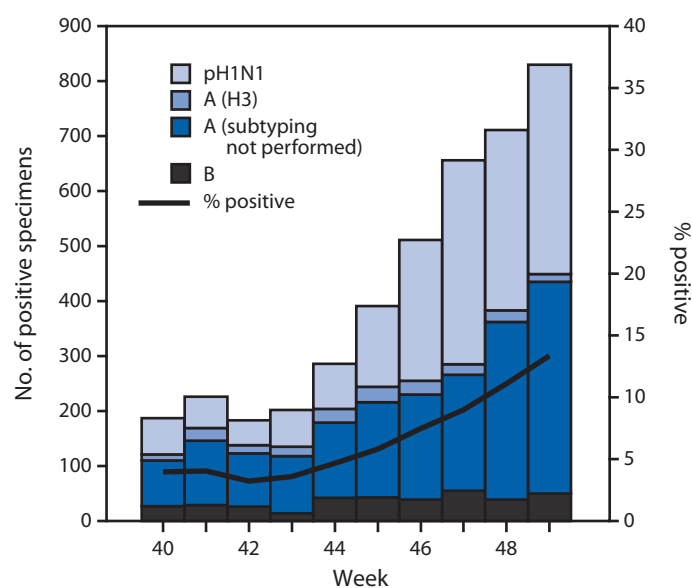
Novel Influenza A Viruses

One infection with an influenza A (H3N2) variant virus (H3N2v) was reported to CDC from Iowa during the week ending October 12, 2013 (week 41). Contact between the patient and swine in the week preceding illness was reported. The patient was mildly ill and fully recovered; no further cases have been identified among contacts of the patient. This is the first H3N2v infection reported for the 2013–14 influenza season.

Antigenic Characterization

WHO collaborating laboratories in the United States are requested to submit a subset of their influenza-positive respiratory specimens to CDC for further antigenic characterization (*I*). CDC has antigenically characterized 221 influenza viruses collected by U.S. laboratories during the 2013–14 season, including 184 pH1N1 viruses, 31 influenza A (H3N2) viruses, and six influenza B viruses. All pH1N1 and influenza A (H3N2) viruses were antigenically like the 2013–14 Northern Hemisphere influenza A vaccine components (A/California/7/2009-like [H1N1] and A/Texas/50/2012-like [H3N2]). Two (33%) of the influenza B viruses tested belong to the B/Yamagata lineage and were characterized as B/Massachusetts/2/2012-like, which

FIGURE 1. Number* and percentage of respiratory specimens testing positive for influenza reported by World Health Organization and National Respiratory and Enteric Virus Surveillance System collaborating laboratories in the United States, by type, subtype, and week — United States, September 29–December 7, 2013†



* N = 4,183.

† Data reported as of December 13, 2013.

is included as an influenza B component in the 2013–14 Northern Hemisphere trivalent and quadrivalent influenza vaccines. The remaining four (67%) influenza B viruses tested belong to the B/Victoria lineage and were characterized as B/Brisbane/60/2008-like, which is included as an influenza B component in the 2013–14 Northern Hemisphere quadrivalent influenza vaccine.

Antiviral Resistance of Influenza Viruses

Testing of pH1N1, influenza A (H3N2), and influenza B virus isolates for resistance to neuraminidase inhibitors (oseltamivir and zanamivir) is performed at CDC using a functional assay. Additional pH1N1 and influenza A (H3N2) clinical samples are tested for mutations of the virus known to confer oseltamivir resistance. Since October 1, 2013, a total of 463 influenza viruses have been tested for antiviral resistance, including 395 pH1N1 viruses, 55 influenza A (H3N2) viruses, and 13 influenza B viruses. Of the 395 pH1N1 viruses tested, seven (1.8%) were resistant to oseltamivir. Of the 273 pH1N1 viruses tested, all (including the seven oseltamivir-resistant viruses) were sensitive to zanamivir. Of the 55 influenza A (H3N2) viruses and 13 influenza B viruses tested, all were sensitive to both oseltamivir and zanamivir.

Outpatient Illness Surveillance

Since September 29, 2013, the weekly percentage of outpatient visits for influenza-like illness (ILI)[‡] reported by approximately 1,800 U.S. Outpatient ILI Surveillance Network (ILINet) providers in 50 states, New York City, Chicago, the U.S. Virgin Islands, Puerto Rico, and the District of Columbia, which comprise ILINet, has ranged from 1.2% to 2.1% and was at or above the national baseline** of 2.0% during the weeks ending November 30, 2013, and December 7, 2013 (weeks 48 and 49) (Figure 2). Peak weekly percentages of outpatient visits for ILI ranged from 2.4% to 7.6% from the 1997–98 through 2012–13 seasons, excluding the 2009 pandemic. For the week ending December 7, 2013 (week 49), three regions (HHS regions 4, 6, and 8) reported ILI activity above their region-specific baseline levels. This is the fourth week this season during which one or more region-specific baselines were exceeded. Data collected in ILINet are used to produce a

measure of ILI activity^{††} by jurisdiction. During week 49, four states (Alabama, Louisiana, Mississippi, and Texas) experienced high ILI activity, no jurisdictions experienced moderate ILI activity, and five states (Arkansas, Colorado, Oklahoma, South Carolina, and Utah) and New York City experienced low ILI activity. Forty-one states experienced minimal ILI activity, and data were insufficient to calculate an ILI activity level for the District of Columbia.

Geographic Spread of Influenza Activity

For the week ending December 7, 2013 (week 49), no jurisdictions reported the geographic spread of influenza^{§§} as widespread, 14 states (Alabama, Arkansas, Colorado, Florida, Kentucky, Louisiana, Maine, Maryland, Mississippi, North Carolina, Oklahoma, South Carolina, Texas, and Utah) reported regional spread, and 18 states (Alaska, Arizona, Connecticut, Georgia, Illinois, Kansas, Massachusetts, Michigan, Minnesota, Nebraska, Nevada, New Jersey, New York, Ohio, Oregon, Tennessee, Virginia, and Wyoming) reported local spread. Sporadic influenza activity was reported by the District of Columbia, Guam, Puerto Rico, and 16 states. No influenza activity was reported by the U.S. Virgin Islands and two states (New Hampshire and Vermont).

Influenza-Associated Hospitalizations

CDC monitors hospitalizations associated with laboratory-confirmed influenza in adults and children through the Influenza

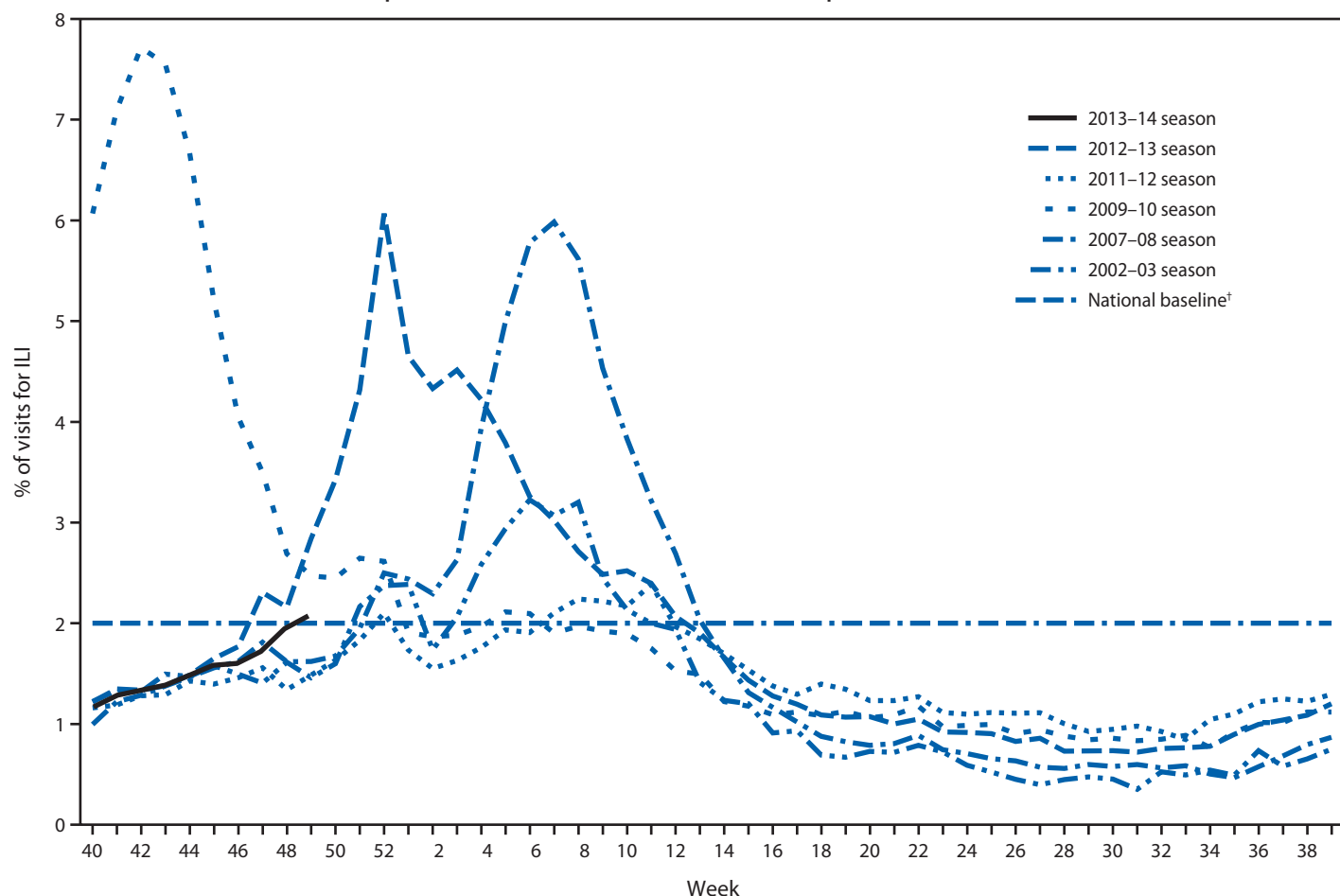
^{††} Activity levels are based on the percentage of outpatient visits in a jurisdiction attributed to ILI and are compared with the average percentage of ILI visits that occur during weeks with little or no influenza virus circulation. Activity levels range from minimal, which would correspond to ILI activity from outpatient clinics being at or below the average, to high, which would correspond to ILI activity from outpatient clinics being much higher than the average. Because the clinical definition of ILI is very nonspecific, not all ILI is caused by influenza; however, when combined with laboratory data, the information on ILI activity provides a clearer picture of influenza activity in the United States.

^{§§} Levels of activity are 1) no activity; 2) sporadic: isolated laboratory-confirmed influenza case(s) or a laboratory-confirmed outbreak in one institution, with no increase in activity; 3) local: increased ILI, or at least two institutional outbreaks (ILI or laboratory-confirmed influenza) in one region of the state, with recent laboratory evidence of influenza in that region and virus activity no greater than sporadic in other regions; 4) regional: increased ILI activity or institutional outbreaks (ILI or laboratory-confirmed influenza) in at least two but less than half of the regions in the state with recent laboratory evidence of influenza in those regions; and 5) widespread: increased ILI activity or institutional outbreaks (ILI or laboratory-confirmed influenza) in at least half the regions in the state, with recent laboratory evidence of influenza in the state.

[‡] Defined as a temperature $\geq 100^{\circ}\text{F}$ ($\geq 37.8^{\circ}\text{C}$), oral or equivalent, and cough and/or sore throat, without a known cause other than influenza.

** The national and regional baselines are the mean percentage of visits for ILI during noninfluenza weeks for the previous three seasons plus two standard deviations. A noninfluenza week is defined as periods of ≥ 2 consecutive weeks in which each week accounted for $< 2\%$ of the season's total number of specimens that tested positive for influenza. National and regional percentages of patient visits for ILI are weighted on the basis of state population. Use of the national baseline for regional data is not appropriate.

FIGURE 2. Percentage of visits for influenza-like illness (ILI)* reported to CDC, by surveillance week — Outpatient Influenza-Like Illness Surveillance Network, United States, September 29–December 7, 2013, and selected previous influenza seasons



* Defined as a temperature of $\geq 100^{\circ}\text{F}$ ($\geq 37.8^{\circ}\text{C}$), oral or equivalent, and cough and/or sore throat, without a known cause other than influenza.

† The national baseline is the mean percentage of visits for ILI during noninfluenza weeks for the previous three seasons plus two standard deviations. Noninfluenza weeks are defined as periods of ≥ 2 consecutive weeks in which each week accounted for $< 2\%$ of the season's total number of specimens that tested positive for influenza. National and regional percentages of patient visits for ILI are weighted on the basis of state population. Use of the national baseline for regional data is not appropriate.

Hospitalization Surveillance Network (FluSurv-Net),⁴⁴ which covers approximately 27 million persons, 8.5% of the U.S. population. From October 1 through December 7, 2013 (week 49), 531 laboratory-confirmed influenza-associated hospitalizations were reported. This yields a rate of 2.0 per 100,000 population. Among cases, 470 (88.5%) were influenza A, 52 (9.8%) were influenza B, four (0.8%) were influenza A and influenza B coinfections, and five (0.9%) had no virus type information. Among those with influenza A subtype information, six (4.1%) were influenza A (H3), and 141 (95.9%) were pH1N1. The most commonly reported chronic underlying medical conditions among adults were obesity, metabolic disorders, chronic lung disease (excluding asthma), and cardiovascular disease. Approximately 42.9% of hospitalized children had no identified chronic underlying medical conditions. The most commonly reported chronic underlying medical conditions

⁴⁴ FluSurv-NET conducts population-based surveillance for laboratory-confirmed influenza-associated hospitalizations among children aged < 18 years (since the 2003–04 influenza season) and adults aged ≥ 18 years (since the 2005–06 influenza season). FluSurv-NET covers approximately 70 counties in the 10 Emerging Infections Program states (California, Colorado, Connecticut, Georgia, Maryland, Minnesota, New Mexico, New York, Oregon, and Tennessee) and additional Influenza Hospitalization Surveillance Project (IHSP) states. IHSP began during the 2009–10 season to enhance surveillance during the 2009 H1N1 pandemic. IHSP sites included Iowa, Idaho, Michigan, Oklahoma, and South Dakota during the 2009–10 season; Idaho, Michigan, Ohio, Oklahoma, Rhode Island, and Utah during the 2010–11 season; Michigan, Ohio, Rhode Island, and Utah during the 2011–12 season; Iowa, Michigan, Ohio, Rhode Island, and Utah during the 2012–13 season; and Michigan, Ohio, and Utah during the 2013–14 season. Incidence rates are calculated using CDC's National Center for Health Statistics population estimates for the counties included in the surveillance catchment area. Laboratory confirmation is dependent on clinician-ordered influenza testing, and testing for influenza often is underutilized because of the poor reliability of rapid test results and greater reliance on clinical diagnosis for influenza. As a consequence, the number of cases identified as part of influenza hospitalization surveillance likely is an underestimation of the actual number of persons hospitalized with influenza.

in children (those aged <18 years) were asthma, obesity, and metabolic disorders. Among 21 hospitalized women of child-bearing age (15–44 years), three were pregnant.

Pneumonia and Influenza-Associated Mortality

During the week ending December 7, 2013 (week 49), pneumonia and influenza (P&I) was reported as an underlying or contributing cause of 6.2% (791 of 12,758) of all deaths reported to the 122 Cities Mortality Reporting System. This percentage is below the epidemic threshold of 6.8% for that week.^{***} Since September 29, 2013, the weekly percentage of deaths attributed to P&I ranged from 5.3% to 6.2% and has not exceeded the epidemic threshold so far this season. Peak weekly percentages of deaths attributable to P&I in the previous five seasons ranged from 7.9% during the 2008–09 and 2011–12 seasons to 9.9% during the 2012–13 season.

Influenza-Associated Pediatric Mortality

As of December 7, 2013 (week 49), three influenza-associated pediatric deaths that occurred in the 2013–14 season were reported to CDC: one death was associated with an influenza A and influenza B virus coinfection, one was associated with an influenza A virus for which no subtyping was performed, and one was associated with a pH1N1 virus. The number of influenza-associated pediatric deaths reported to CDC in the previous three influenza seasons has ranged from 35 during the 2011–12 season to 169 for the 2012–13 season. During the 2009 pandemic, 348 pediatric deaths were reported from April 15, 2009, through October 2, 2010 (traditional influenza seasons include data from October (week 40) through September (week 39) of the following year). In the two seasons before the 2009 pandemic, influenza-associated pediatric deaths reported to CDC ranged from 67 during the 2008–09 season (through April 14, 2009) to 88 during the 2007–08 season.

^{***} The seasonal baseline proportion of P&I deaths is projected using a robust regression procedure, in which a periodic regression model is applied to the observed percentage of deaths from P&I that were reported by the 122 Cities Mortality Reporting System during the preceding 5 years. The epidemic threshold is set at 1.645 standard deviations above the seasonal baseline.

What is already known on this topic?

CDC collects, compiles, and analyzes data on influenza activity year-round in the United States. The influenza season generally begins in the fall and continues through the winter and spring months; however, the timing and severity of circulating influenza viruses can vary by geographic location and season.

What is added by this report?

During September 29–December 7, 2013, influenza activity overall in the United States has been increasing. The vast majority of the influenza viruses characterized thus far this season have been antigenically like the components included in the 2013–14 Northern Hemisphere trivalent influenza vaccine: A/California/7/2009-like (H1N1), A/Texas/50/2012-like (H3N2), and B/Massachusetts/2/2012-like. The majority of influenza viruses tested to date have been sensitive to the antiviral drug oseltamivir; all are sensitive to the antiviral drug zanamivir.

What are the implications for public health practice?

Vaccination remains the most effective method to prevent influenza and its complications. Health-care providers should offer vaccine to all unvaccinated persons aged ≥6 months now and throughout the influenza season. Treatment with influenza antiviral medications can reduce severe outcomes of influenza, especially when initiated as early as possible, in patients with confirmed or suspected influenza.

Reported by

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Editorial Note

Influenza activity so far this season has increased during the most recent weeks and is expected to continue to increase in the coming weeks. During September 29–December 7, 2013, pH1N1 viruses were identified most frequently in the United States, but influenza A (H3N2) and influenza B viruses also were reported. Antigenic characterization of influenza-positive respiratory specimens submitted to CDC indicates that 1) the majority of these isolates tested were antigenically like the components of the 2013–14 Northern Hemisphere trivalent influenza vaccine viruses and 2) all of these isolates tested were antigenically like the components of the 2013–14 Northern Hemisphere quadrivalent influenza vaccine viruses. Although the timing of influenza activity varies from year to year, peak activity in the United States most commonly occurs in February, but there can be substantial influenza activity as early as November and December, and activity can occur as late as May (2). Vaccination remains the most effective method to prevent influenza and its complications. Health-care providers should offer vaccine to all unvaccinated persons aged ≥ 6 months now and throughout the influenza season.

Antiviral medications continue to be an important adjunct to vaccination for reducing the health impact of influenza. On January 21, 2011, Advisory Committee on Immunization Practices recommendations on the use of antiviral agents for treatment and chemoprophylaxis of influenza were released (3). This guidance remains in effect for the 2013–14 season. Treatment with antivirals is recommended as soon as possible for patients with confirmed or suspected influenza who have severe, complicated, or progressive illness; who require hospitalization; or who are at higher risk for influenza complications,^{†††} without waiting for confirmatory testing (3). Antiviral treatment also may be considered for outpatients with

confirmed or suspected influenza who do not have known risk factors for severe illness, and is most effective when treatment is initiated within 48 hours of illness onset. Recommended antiviral medications include oseltamivir (Tamiflu) and zanamivir (Relenza). The majority of influenza viruses tested for the 2013–14 season since October 1, 2013, have been susceptible to oseltamivir. The seven oseltamivir-resistant pH1N1 viruses identified in late October through November were from three different states in the South and West. Sporadic oseltamivir-resistant pH1N1 virus infections, including small geographic clusters, have occurred previously, but the public health impact has been limited (4,5). This situation is being closely monitored. All influenza viruses, including those from the oseltamivir-resistant cluster, tested since October 1, 2013, have been susceptible to zanamivir. Amantadine and rimantadine are not recommended because of high levels of resistance to these drugs among circulating influenza A viruses (3). In addition, influenza B viruses are not susceptible to amantadine or rimantadine.

Influenza surveillance reports for the United States are posted online weekly and are available at <http://www.cdc.gov/flu/weekly>. Additional information regarding influenza viruses, influenza surveillance, influenza vaccine, influenza antiviral medications, and novel influenza A virus infections in humans is available at <http://www.cdc.gov/flu>.

Acknowledgments

State, local, and territorial health departments and public health laboratories. US World Health Organization collaborating laboratories. National Respiratory and Enteric Virus Surveillance System laboratories. US Outpatient Influenza-Like Illness Surveillance Network. Influenza Hospitalization Surveillance Network. Influenza-Associated Pediatric Mortality Surveillance System. 122 Cities Mortality Reporting System.

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^{†††} Persons at higher risk include 1) children aged <5 years (especially those aged <2 years); 2) adults aged ≥ 65 years; 3) persons with chronic pulmonary conditions (including asthma); cardiovascular disease (except hypertension alone); renal, hepatic, or hematologic (including sickle cell disease); metabolic disorders (including diabetes mellitus); or neurologic and neurodevelopment conditions (including disorders of the brain, spinal cord, peripheral nerve, and muscle, such as cerebral palsy, epilepsy [seizure disorders], stroke, intellectual disability [mental retardation], moderate to severe developmental delay, muscular dystrophy, or spinal cord injury); 4) persons with immunosuppression, including that caused by medications or by human immunodeficiency virus infection; 5) women who are pregnant or postpartum (within 2 weeks after delivery); 6) persons aged ≤ 18 years who are receiving long-term aspirin therapy; 7) American Indians/Alaska Natives; 8) persons who are morbidly obese (i.e., body mass index ≥ 40); and 9) residents of nursing homes and other chronic-care facilities.

Announcement

Smartphone Application Available for Extension Ladder Safety

Falls from ladders are a common cause of preventable injuries. Misjudging the ladder angle is a significant risk factor for a fall. A free smartphone application is available from CDC to improve the safety of extension ladder users.

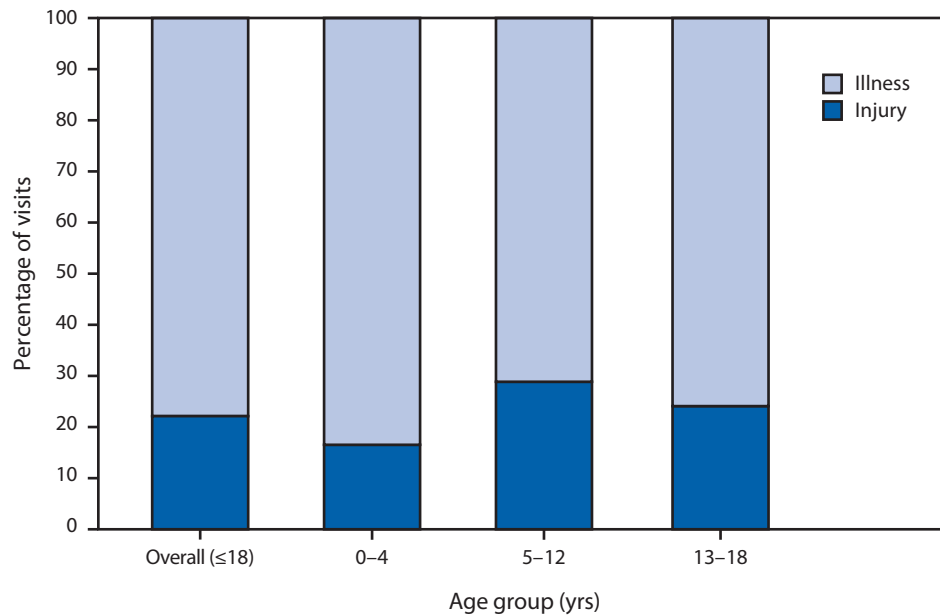
The ladder safety application features an indicator that provides visual, sound, and vibration signals to assist users in quickly positioning an extension ladder at an optimal angle. The application also provides graphic-oriented interactive reference materials, safety guidelines, and checklists for extension ladder selection, inspection, accessorizing, and use, and can serve as a convenient training tool.

CDC's National Institute for Occupational Safety and Health developed the ladder safety application using innovative research, existing information from safety regulations and consensus standards, and input from industry. The application is available for Apple iPhone/iPad and Google Android devices, and is also available in Spanish. The ladder safety application and additional information are available at <http://www.cdc.gov/niosh/topics/falls>.

QuickStats

FROM THE NATIONAL CENTER FOR HEALTH STATISTICS

Percentage of Hospitalizations After Emergency Department Visits Resulting from Illness and Injury Among Persons Aged ≤ 18 Years, by Age Group — National Hospital Ambulatory Medical Care Survey, United States, 2007–2010*



* Percentages are 4-year annual averages.

During 2007–2010, emergency department visits by children resulting in hospital admission were more likely to be related to illness (78%) than injury (22%). This pattern applied to persons aged 0–4 years, 5–12 years, and 13–18 years, with the greatest difference observed among children aged 0–4 years, for whom 84% of visits resulting in hospital admission were related to illness, compared with only 16% related to injury.

Source: National Hospital Ambulatory Medical Care Survey, 2007–2010. Available at <http://www.cdc.gov/nchs/ahcd.htm>.

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Morbidity and Mortality Weekly Report

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. To receive an electronic copy each week, visit *MMWR*'s free subscription page at <http://www.cdc.gov/mmwr/mmwrsubscribe.html>. Paper copy subscriptions are available through the Superintendent of Documents, U.S. Government Printing Office, Washington, DC 20402; telephone 202-512-1800.

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U.S. Government Printing Office: 2014-723-032/01037 Region IV ISSN: 0149-2195