

Measles — United States, 2011

In 2000, the United States achieved measles elimination (defined as interruption of year-round endemic measles transmission) (1). However, importations of measles into the United States continue to occur, posing risks for measles outbreaks and sustained measles transmission. During 2011, a total of 222 measles cases (incidence rate: 0.7 per 1 million population) and 17 measles outbreaks (defined as three or more cases linked in time or place) were reported to CDC, compared with a median of 60 (range: 37–140) cases and four (range: 2–10) outbreaks reported annually during 2001–2010. This report updates an earlier report on measles in the United States during the first 5 months of 2011 (2). Of the 222 cases, 112 (50%) were associated with 17 outbreaks, and 200 (90%) were associated with importations from other countries, including 52 (26%) cases in U.S. residents returning from abroad and 20 (10%) cases in foreign visitors. Other cases associated with importations included 67 (34%) linked epidemiologically to importations, 39 (20%) with virologic evidence suggesting recent importation, and 22 (11%) linked to cases with virologic evidence of recent importation. Most patients (86%) were unvaccinated or had unknown vaccination status. The increased numbers of outbreaks and measles importations into the United States underscore the ongoing risk for measles among unvaccinated persons and the importance of vaccination against measles (3).

Confirmed measles cases in the United States are reported by state and local health departments to CDC using a standard case definition.* A measles case is considered confirmed if it is laboratory-confirmed or meets the clinical case definition (an illness characterized by a generalized rash lasting ≥ 3 days, a temperature of $\geq 101^\circ\text{F}$ [$\geq 38.3^\circ\text{C}$], and cough, coryza, or conjunctivitis) and is linked epidemiologically to a confirmed case. Laboratory confirmation of measles is made by detection in serum of measles-specific immunoglobulin M (IgM), a significant rise in measles immunoglobulin G (IgG) level, isolation of measles virus, or detection of measles virus by nucleic acid

amplification from a clinical specimen. Cases are considered importations if exposure to measles virus occurred outside the United States 7–21 days before rash onset and rash occurred within 21 days of entry into the United States, with no known exposure to measles in the United States during that time.

For this report, U.S. residents were classified as eligible or ineligible for measles, mumps, and rubella (MMR) vaccination according to the Advisory Committee on Immunization Practices recommendations for measles vaccination (3). Vaccine-eligible patients were defined as U.S. residents who 1) were unvaccinated or had unknown vaccination status, 2) did not have any contraindications for vaccination, and 3) were either born after 1957 and aged ≥ 12 months without previous documentation of presumptive evidence of immunity to measles[†] or aged 6–11 months with recent history of international travel.

[†] Documented receipt of 2 doses of live measles virus-containing vaccine, laboratory evidence of immunity, documentation of physician-diagnosed measles, or birth before 1957.

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* Measles 2010 case definition. Council of State and Territorial Epidemiologists position statement 09-ID-48. Available at http://www.cdc.gov/osels/ph_surveillance/nndss/casedef/measles_2010.htm.



During 2011, a provisional total of 222 measles cases were reported from 31 states (Figure 1). The median age of the patients was 14 years (range: 3 months to 84 years); 27 (14%) were aged <12 months, 51 (26%) were aged 1–4 years, 42 (21%) were aged 5–19 years, and 76 (39%) were aged ≥20 years. Most patients were unvaccinated (65%) or had unknown vaccination status (21%). Of the 222, a total of 196 were U.S. residents. Of those U.S. residents who had measles, 166 were unvaccinated or had unknown vaccination status, 141 (85%) were eligible for MMR vaccination, 18 (11%) were too young for vaccination, six (4%) were born before 1957 and presumed immune, and one (1%) had previous laboratory evidence of presumptive immunity to measles. Among the 141 patients who were unvaccinated and eligible for MMR vaccination, nine (6%) were infants aged 6–11 months and had recent history of international travel; 14 (10%) were aged 12–15 months, the age recommended for receiving the first dose of MMR vaccine; and 66 (47%) were aged 16 months through 19 years. Of those 66 patients, 50 (76%) had not been vaccinated because of a philosophic, religious, or personal objection.

Among the 70 (32%) measles patients who were hospitalized, 17 (24%) had diarrhea, 15 (21%) were dehydrated, and 12 (17%) had pneumonia. No cases of encephalitis and no deaths were reported.

Of the 222 U.S. measles cases, 200 (90%) were associated with importations, of which 72 (36%) were importations from other countries, 67 (30%) were linked epidemiologically to

importations, 39 (20%) had virologic evidence (i.e., isolation of a viral genotype known to circulate in a country with measles) that suggested recent importation, and 22 (11%) were linked to cases with virologic evidence of recent importation. The source of measles acquisition in 22 cases was not determined through contact tracing or viral isolation (i.e., linking the patient to a country with measles or isolation of a viral genotype known to circulate in a country with measles). Importations were reported during 31 of the 52 reporting weeks (Figure 2). Among the 72 cases of measles importation, 52 were linked to U.S. residents who had traveled abroad, and 20 were linked to foreign visitors. Almost half (46%) of the 72 measles importations occurred among persons who acquired the disease in the World Health Organization (WHO) European Region (Table).

Seventeen outbreaks accounted for 112 (50%) of the 222 cases. The median outbreak size was six cases (range: 3–21 cases), and outbreaks lasted a median of 18 days (range: 6–69 days).

Measles was laboratory confirmed in 200 (90%) cases: 94 (47%) by detection of measles-specific IgM and measles virus nucleic acid, 69 (35%) by detection of IgM only, and 37 (19%) by detection of measles virus nucleic acid only. Six genotypes of measles virus were identified among samples collected: D4, D9, D8, B3, G3, and H1 (Table).

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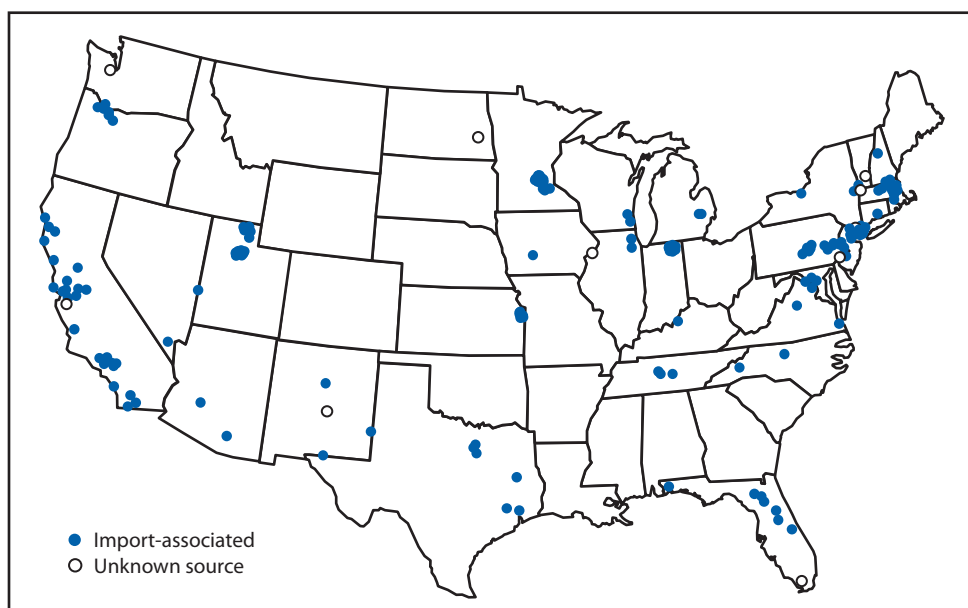
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FIGURE 1. Origin of reported measles cases (N = 222) — United States, 2011



Editorial Note

Measles elimination has been maintained in the United States for more than a decade through high population immunity secondary to high MMR vaccination coverage. Coverage with 1 dose has been >90% among children aged 19–35 months

more than 90% of cases reported to the European Centers for Disease Prevention and Control (4). Although measles has been eliminated in the Region of the Americas since 2002 and considerable progress has been achieved in global measles control, measles is still common in many countries.

since 1996. The increase in measles importations and outbreaks during 2011 serves as a reminder that measles remains endemic in many parts of the world and unvaccinated U.S. residents continue to place themselves and others in their communities at risk for measles and its complications.

The increase in importations reflects recent increases in the incidence of measles in countries visited by U.S. travelers. The source of almost half of the measles importations in 2011 was the WHO European Region, which reported >30,000 cases of measles, including 27 cases of measles encephalitis, a complication that often results in permanent neurologic sequelae, and eight measles-related deaths in 2011. Five countries (France, Italy, Romania, Spain, and Germany) accounted for

FIGURE 2. Number of measles cases, by import status and week of rash onset (N = 222) — United States, 2011

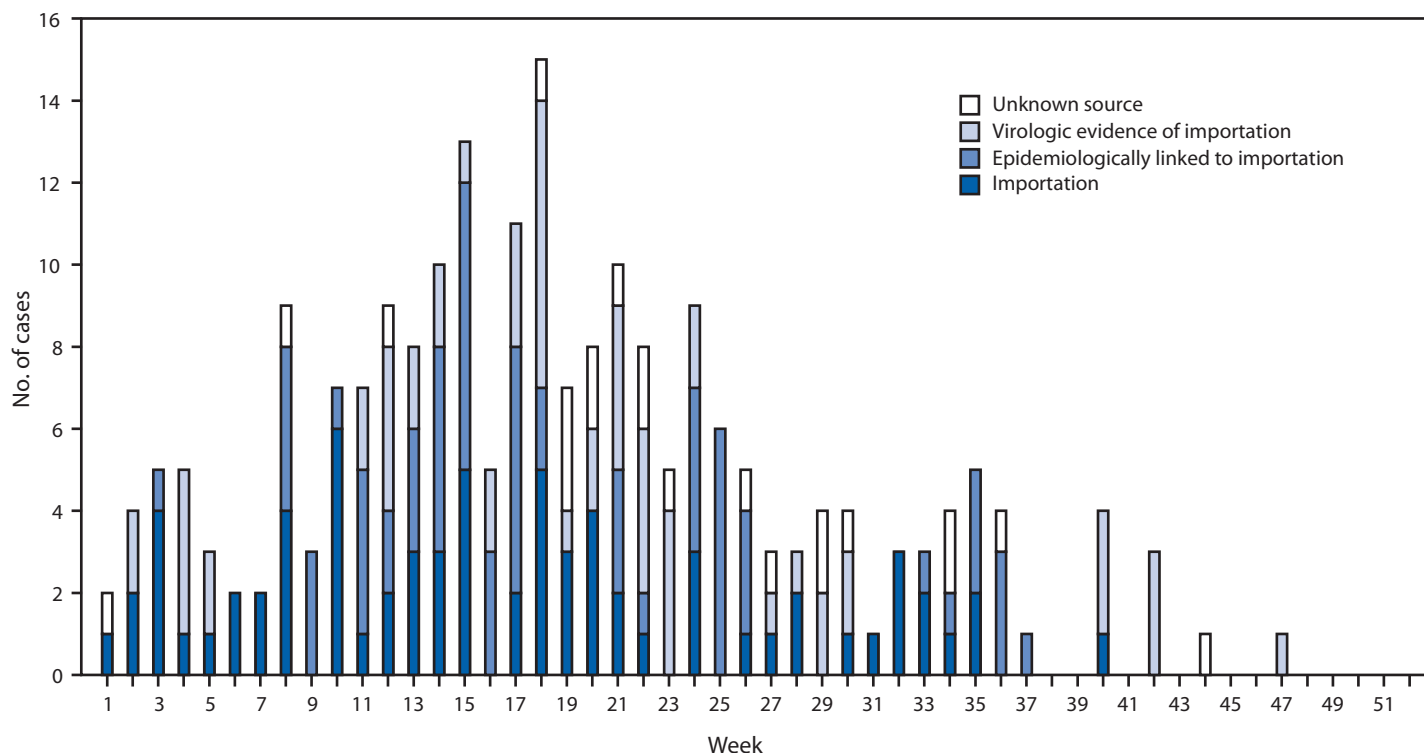


TABLE. Countries where imported measles was acquired, by World Health Organization (WHO) region, number of cases (n = 72), and genotype — United States, 2011

WHO region	No. of cases	Country	No. of cases	Genotype identified*
African	4	Ethiopia	1	B3
		Kenya	2	B3 (2)
		Nigeria	1	B3
Americas	2	Canada	1	
		Dominican Republic [†]	1	D4
Eastern Mediterranean	3	Jordan	1	D4
		Pakistan	2	
European	33	Bulgaria	1	
		France	13	D4 (5), G3
		France/Germany/Spain [§]	1	
		France/Italy [§]	1	D4
		France/Italy/Spain/Germany [§]	1	
		France/Spain/United Kingdom [§]	1	
		France/United Kingdom [§]	1	D4
		Italy	4	D4 (3)
		Poland	1	
		Romania	1	D4
		Romania/Hungary [§]	2	D4 (2)
		Spain	1	
		United Kingdom	5	D4 (3)
South-East Asia	19	Bangladesh	1	
		India	16	D4, D8 (5)
		Indonesia	2	
Western Pacific	11	China	2	H1
		Malaysia	2	D9 (2)
		Philippines	6	D9 (4)
		Philippines/Vietnam/Singapore/Malaysia [§]	1	

* Genotype was determined based on methodology described by the World Health Organization. Measles virus nomenclature update: 2012. *Wkly Epidemiol Rec* 2012;87:73–80.

[†] Although the patient acquired measles in the Dominican Republic, the likely source of infection was a French tourist with measles who stayed in an adjacent room at the same resort at the same time as the patient. The genotype identified in cases epidemiologically linked to this patient was D4, a genotype currently circulating in France.

[§] Patients had visited more than one country in which measles is endemic during the incubation period, and exposure could have occurred in any of the countries listed.

Importations of measles virus into the United States will likely continue and cause outbreaks in communities that have clusters of unvaccinated persons. Maintenance of high MMR vaccination coverage is essential to prevent measles outbreaks and sustain measles elimination in the United States. Despite the relatively small number of reported cases in the United States, the public and the health-care providers must remain vigilant. A drop in MMR vaccination coverage in a community can increase the risk for large, sustained measles outbreaks, as experienced recently in Canada and France (4,5), or reestablishment of endemic transmission, as experienced in the United Kingdom (6).

Occasionally, measles cases are reported without apparent links to importations, but virologic evidence suggests recent importation of an undetected case or chain of cases. Given travel patterns, the highly infectious nature of measles virus,

What is already known on this topic?

Achievement of measles elimination was declared in the United States in 2000, but the disease remains poorly controlled in much of the world. Cases of measles are imported regularly into the United States.

What is added by this report?

During 2011, 222 cases of measles and 17 outbreaks were reported in the United States, an increase compared with cases and outbreaks reported during 2001–2010. Importations accounted for 72 (32%) cases, including 52 (72%) cases among U.S. residents who had traveled abroad recently. Among patients who were U.S. residents, 85% were unvaccinated or had unknown vaccination status and were eligible for measles, mumps, and rubella (MMR) vaccination.

What are the implications for public health practice?

MMR vaccine is highly effective in preventing measles and its complications. Rapid public health response and high 2-dose MMR vaccine coverage are essential in preventing measles outbreaks and sustaining elimination in the United States. One dose of MMR vaccine is recommended routinely for all children at age 12–15 months, with a second dose at age 4–6 years. Adults without evidence of measles immunity should receive 1 MMR vaccine dose, whereas 2 doses are recommended for unvaccinated health-care personnel, international travelers, and students attending post-high school educational institutions.

and limitations of surveillance systems, not every importation of measles virus into the United States is detected. Therefore, collection of samples for virus detection is extremely important. Genetic characterization of viruses can help to confirm or suggest the likely source of imported viruses because measles genotypes are distributed heterogeneously in regions that have not yet eliminated measles (7,8).

Health-care providers play an important role in maintaining elimination of measles in the United States. Patients with measles frequently seek medical care; therefore, health-care providers should maintain a high awareness of measles and suspect measles in persons who have a febrile rash illness and clinically compatible symptoms (e.g., cough, coryza, or conjunctivitis) and who recently have traveled abroad or have had contact with travelers. Providers should implement isolation precautions immediately and promptly report suspected measles cases to their local health department to limit spread to other susceptible persons, including those who cannot be vaccinated because of medical contraindications or those too young for vaccination. In several outbreaks during 2011, despite seeking medical care, the source case was not identified until after the first or second generation of cases was reported. Misdiagnosis and delayed reporting resulted in missed opportunities to prevent additional cases because of delayed implementation of control measures. Nevertheless, for most cases, early reporting

by providers and rapid control efforts by state and local public health agencies have prevented measles transmission and limited the size of outbreaks.

Health-care providers should encourage vaccination of all eligible patients, including children and adults. MMR vaccine is recommended routinely for all children at age 12–15 months, with a second dose at age 4–6 years. Two doses of MMR vaccine also are recommended for unvaccinated health-care personnel, international travelers, and students attending post–high school educational institutions. Other adults without evidence of measles immunity should receive 1 dose of MMR vaccine (3). In addition, providers should remind their patients who plan to travel internationally of the increased risk for measles and potential exposures during bus, train, or air travel and at large international events or gatherings (e.g., Euro 2012 and the 2012 Summer Olympics), and of the importance of vaccination. All persons aged ≥ 6 months who will be traveling outside the United States and are eligible to receive MMR vaccine should be vaccinated before travel. Children aged ≥ 12 months should receive 2 doses of MMR vaccine separated by at least 28 days, before travel (3).

References

1. Katz SL, Hinman AR. Summary and conclusions: measles elimination meeting, 16–17 March 2000. *J Infect Dis* 2004;189(Suppl 1):S43–7.
2. CDC. Measles—United States, January–May 20, 2011. *MMWR* 2011;60:666–8.
3. CDC. Measles, mumps, and rubella—vaccine use and strategies for elimination of measles, rubella, and congenital rubella syndrome and control of mumps: recommendations of the Advisory Committee on Immunization Practices (ACIP). *MMWR* 1998;47:(No. RR-8).
4. European Centre for Disease Prevention and Control. Surveillance report: European monthly measles monitoring, 21 February 2012. Stockholm, Sweden: European Centre for Disease Prevention and Control; 2012. Available at http://ecdc.europa.eu/en/publications/publications/sur_emmo_european-monthly-measles-monitoring-february-2012.pdf. Accessed April 6, 2012.
5. Ministère de Santé et Services sociaux Quebec. Measles. Quebec, Canada: Ministère de Santé et Services sociaux Quebec; 2012. Available at http://www.msss.gouv.qc.ca/en/sujets/prob_sante/measles/measles.php. Accessed April 6, 2012.
6. Editorial team. Measles once again endemic in the United Kingdom. *Euro Surveill* 2008;13.
7. Rota PA, Brown K, Mankertz A, et al. Global distribution of measles genotypes and measles molecular epidemiology. *J Infect Dis* 2011;204(Suppl 1):S514–23.
8. World Health Organization. Measles virus nomenclature update: 2012. *Wkly Epidemiol Rec* 2012;87:73–80.

Human Papillomavirus–Associated Cancers — United States, 2004–2008

Oncogenic human papillomavirus (HPV) has a causal role in nearly all cervical cancers and in many vulvar, vaginal, penile, anal, and oropharyngeal cancers (1). Most HPV infections clear within 1–2 years, but those that persist can progress to precancer or cancer. In the United States, public health prevention of cervical cancer includes both secondary prevention through cervical cancer screening and primary prevention through HPV vaccination. Transmission of HPV also can be reduced through condom use and limiting the number of sexual partners. Two vaccines (bivalent and quadrivalent) are available to protect against HPV types 16 and 18, which are responsible for 70% of cervical cancers. HPV 16 also is the most common HPV type found in the other five cancers often associated with HPV (2). To assess the incidence of HPV-associated cancers (i.e., cancers at specific anatomic sites and with specific cell types in which HPV DNA frequently is found), CDC analyzed 2004–2008 data from the National Program of Cancer Registries (NPCR) and the Surveillance, Epidemiology, and End Results (SEER) program. During 2004–2008, an average of 33,369 HPV-associated cancers were diagnosed annually (rate: 10.8 per 100,000 population), including 12,080 among males (8.1 per 100,000) and 21,290 among females (13.2). Multiplying the counts for HPV-associated cancers by percentages attributable to HPV (3), CDC estimated that approximately 26,000 new cancers attributable to HPV occurred each year, including 18,000 among females and 8,000 among males. Population-based cancer registries are important surveillance tools to measure the impact on cancer rates of public health interventions such as vaccination and screening.

CDC analyzed NPCR and SEER data on cancers diagnosed during 2004–2008 in 50 states and the District of Columbia (data covering 100% of the U.S. population are now available through expansion of NPCR) (4). Case definitions based on expert consensus were used to examine the burden of invasive cancers at anatomic sites (cervix, vulva, vagina, penis, anus, and oropharynx [5]) and for cell types (carcinoma of the cervix and squamous cells for the other sites) in which HPV DNA is frequently found. Inclusion of oropharyngeal cancers as HPV-associated was further limited to specific sites where HPV is most likely to be found: base of tongue, tonsils, and “other oropharynx” (5).

Cancer data were analyzed by sex, age, race, Hispanic ethnicity, and state of residence. Race categories included white, black, Asian/Pacific Islander, and American Indian/Alaska Native; “all races” included other and unknown categories.

American Indian/Alaska Native data were enhanced by linkage with Indian Health Service administrative records (4). Hispanic ethnicity included persons of any race who were identified as being Hispanic in the medical record or by use of an algorithm* (4). Age-adjusted incidence rates were calculated per 100,000 persons in SEER*Stat† and were standardized to the 2000 U.S. Standard Population. Significant differences in rates were limited to comparisons at $p < 0.05$. Because HPV-associated cancers defined by cell type and specific anatomic site might include cancers not caused by HPV, and because cancer registries typically do not capture information on HPV infection status, for this analysis, the average annual number of HPV-associated cancers was multiplied by the percentage of each cancer type found attributable to HPV based on genotyping studies (3).

Overall, an average of 33,369 HPV-associated cancers (10.8 per 100,000 population) were diagnosed annually: 21,290 among females (13.2) and 12,080 among males (8.1). Cervical cancer was the most common of these cancers, with an average of 11,967 cases annually; oropharyngeal cancer was the second most common, with an average of 11,726 cases annually (2,370 among females and 9,356 among males) (Tables 1 and 2). The rate of anal cancer among females (1.8 per 100,000) was higher than among males (1.2). The rate of oropharyngeal cancer among males (6.2) was four times that among females (1.4). Rates of cervical and penile cancer were higher among blacks (9.9) and Hispanics (11.3), when compared with whites (7.4) and non-Hispanics (7.4); however, the rate of vulvar cancer was lower among blacks (1.4) and Hispanics (1.2) than among whites (1.9) and non-Hispanics (1.9). Anal cancer in females was highest among whites (2.0), whereas rates in males were highest among blacks (1.6). For both sexes, rates of oropharyngeal cancer were higher among whites (males: 6.4, females: 1.4) and blacks (males: 6.3, females: 1.4) than other races (Table 1).

Rates varied by state, with rates of HPV-associated cancers combined ranging from 8.5 per 100,000 (Utah) to 16.3 (West Virginia) among females, and from 4.9 (Utah) to 11.6 (District of Columbia) among males. Although rates varied by anatomic site, some states had lower or higher rates across cancer sites. Maryland, Colorado, and Utah had cancer rates in the lowest tertile for most or all HPV-associated cancers,

*The North American Association of Central Cancer Registries' Method to Enhance Hispanic/Latino Identification algorithm uses information on ethnicity from the medical record, information reported to the cancer registry, and information on surname (including maiden name, when available) to categorize patients as either Hispanic or non-Hispanic.

† Available at <http://seer.cancer.gov/seerstat>.

TABLE 1. Human papillomavirus (HPV)-associated cancers,* by anatomic site, age group, sex, and race/ethnicity — United States, 2004–2008

Characteristic	Cervical carcinoma			Vulvar SCC			Vaginal SCC			Penile SCC		
	Rate [†]	(95% CI) [§]	Average annual no.	Rate	(95% CI)	Average annual no.	Rate	(95% CI)	Average annual no.	Rate	(95% CI)	Average annual no.
Total	7.7	(7.7–7.8)	11,967	1.8	(1.8–1.9)	3,136	0.4	(0.4–0.4)	729	0.8	(0.8–0.8)	1,046
Age group (yrs)												
0–19	0.0	(0.0–0.1)	15	0.0	(0.0–0.0)	0	— [¶]	—	—	—	—	—
20–29	3.2	(3.1–3.3)	650	0.1	(0.1–0.1)	17	—	—	—	0.0	(0.0–0.0)	5
30–39	12.6	(12.4–12.8)	2,525	0.7	(0.7–0.8)	144	0.1	(0.1–0.1)	21	0.2	(0.1–0.2)	33
40–49	14.2	(14.0–14.4)	3,200	2.0	(1.9–2.1)	461	0.3	(0.3–0.4)	74	0.4	(0.4–0.5)	97
50–59	12.3	(12.1–12.6)	2,411	2.9	(2.8–3.0)	573	0.7	(0.6–0.7)	132	1.0	(0.9–1.0)	182
60–69	12.5	(12.2–12.8)	1,589	4.2	(4.1–4.4)	536	1.2	(1.1–1.3)	147	2.3	(2.2–2.5)	261
70–79	10.8	(10.5–11.1)	975	6.9	(6.6–7.1)	623	1.8	(1.7–2.0)	167	3.7	(3.5–3.9)	262
≥80	8.7	(8.3–9.0)	602	11.1	(10.7–11.4)	781	2.6	(2.5–2.8)	184	5.5	(5.2–5.9)	205
Race												
White (referent)	7.4	(7.3–7.5)	9,278	1.9	(1.9–2.0)	2,788	0.4	(0.4–0.4)	585	0.8	(0.7–0.8)	889
Black	9.9**	(9.7–10.2)	1,867	1.4**	(1.4–1.5)	262	0.7**	(0.6–0.7)	114	0.9**	(0.9–1.0)	112
AI/AN	6.5**	(5.9–7.1)	94	1.1**	(0.9–1.4)	14	0.3	(0.2–0.5)	4	1.0	(0.7–1.4)	9
A/PI	7.1	(6.8–7.4)	510	0.4**	(0.4–0.5)	27	0.3**	(0.2–0.3)	18	0.4**	(0.3–0.5)	21
Ethnicity												
Non-Hispanic (referent)	7.4	(7.3–7.4)	10,099	1.9	(1.9–1.9)	2,989	0.4	(0.4–0.4)	673	0.7	(0.7–0.8)	902
Hispanic	11.3**	(11.1–11.6)	1,867	1.2**	(1.1–1.3)	147	0.4	(0.4–0.5)	56	1.3**	(1.2–1.4)	144

Characteristic	Anal SCC						Oropharyngeal SCC					
	Female			Male			Female			Male		
	Rate	(95% CI)	Average annual no.	Rate	(95% CI)	Average annual no.	Rate	(95% CI)	Average annual no.	Rate	(95% CI)	Average annual no.
Total	1.8	(1.8–1.9)	3,089	1.2	(1.1–1.2)	1,678	1.4	(1.4–1.4)	2,370	6.2	(6.1–6.3)	9,356
Age group (yrs)												
0–19	—	—	—	—	—	—	—	—	—	—	—	—
20–29	0.0	(0.0–0.0)	5	0.0	(0.0–0.1)	7	0.1	(0.0–0.1)	10	0.1	(0.0–0.1)	12
30–39	0.4	(0.4–0.5)	80	0.5	(0.5–0.6)	102	0.3	(0.2–0.3)	53	0.8	(0.7–0.8)	156
40–49	2.3	(2.2–2.4)	527	1.8	(1.7–1.9)	406	1.4	(1.3–1.5)	319	6.6	(6.5–6.8)	1,512
50–59	4.7	(4.6–4.9)	924	2.5	(2.4–2.6)	459	3.4	(3.2–3.5)	660	18.9	(18.7–19.2)	3,549
60–69	5.3	(5.1–5.5)	676	3.0	(2.9–3.2)	342	5.0	(4.8–5.2)	636	22.2	(21.9–22.6)	2,548
70–79	5.6	(5.4–5.8)	504	3.3	(3.1–3.5)	231	5.1	(4.9–5.3)	460	16.9	(16.5–17.4)	1,196
≥80	5.3	(5.1–5.6)	373	3.4	(3.2–3.7)	130	3.3	(3.1–3.5)	231	9.9	(9.5–10.4)	382
Race												
White (referent)	2.0	(1.9–2.0)	2,758	1.1	(1.1–1.2)	1,391	1.4	(1.4–1.5)	2,049	6.4	(6.3–6.4)	8,229
Black	1.4**	(1.3–1.5)	260	1.6**	(1.5–1.7)	243	1.4	(1.3–1.5)	259	6.3	(6.1–6.5)	918
AI/AN	1.0**	(0.8–1.3)	14	0.7**	(0.5–0.9)	9	0.8**	(0.6–1.1)	11	3.2**	(2.7–3.7)	39
A/PI	0.4**	(0.3–0.5)	25	0.2**	(0.2–0.3)	12	0.5**	(0.4–0.6)	32	1.7**	(1.5–1.9)	96
Ethnicity												
Non-Hispanic (referent)	1.9	(1.9–1.9)	2,899	1.2	(1.2–1.2)	1,573	1.5	(1.5–1.5)	2,273	6.5	(6.4–6.6)	8,932
Hispanic	1.4**	(1.3–1.5)	190	0.8**	(0.7–0.9)	105	0.7**	(0.7–0.8)	97	3.5**	(3.3–3.6)	424

Abbreviations: AI/AN = American Indian/Alaska Native; A/PI = Asian/Pacific Islander; CI = confidence interval; SCC = squamous cell carcinoma.
 * HPV-associated cancers are defined as cancers at specific anatomic sites and with specific cellular types in which HPV DNA frequently is found. Data are from population-based cancer registries that participate in the National Program of Cancer Registries and/or the Surveillance, Epidemiology, and End Results Program, and meet criteria for high data quality. Only carcinomas are included for cervical cancer. Only SCCs are included for vulvar, vaginal, penile, anal, and oropharyngeal cancers. Anal cancers include SCCs coded to the rectum. All cell types (histology) were microscopically confirmed. Oropharyngeal sites and other definitions specified in: Watson M, Saraiya M, Ahmed F, et al. Using population-based cancer registry data to assess the burden of HPV-associated cancers in the United States: overview of methods. Cancer. 2008;113:2841–54.
[†] Per 100,000 population.
[§] Based on the gamma distribution using the Tiwari modification (additional information available at <http://seer.cancer.gov/seerstat>).
[¶] Data suppressed because the total number of cancers for 2004–2008 was <16.
 ** Rate differed significantly from the rate in the referent group (p<0.05).

whereas Kentucky, Louisiana, and Tennessee had rates in the highest tertile for most of the cancer sites.[§]

Multiplying the number of HPV-associated cancers by the percentages attributable to HPV (3), CDC estimated that approximately 26,000 new cancers attributable to HPV occurred each year: 18,000 among females and 8,000 among males (Table 2). Cervical and oropharyngeal cancers were the most common of these, with an estimated 11,500 cervical cancers and 7,400 oropharyngeal cancers (5,900 among men and 1,500 among women).

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Editorial Note

The results of this analysis determined that an estimated average of 21,290 HPV-associated cancers occurred among females each year during 2004–2008, making these cancers combined more common than ovarian cancers and nearly as common as melanoma among females.[¶] The combined burden among men was smaller, with an average of 12,080 cases per year, roughly equivalent to the number of invasive brain cancers occurring annually among men. Many HPV-associated cancers likely are preventable through the use of HPV vaccine.

Two vaccines (bivalent and quadrivalent) are available to protect against HPV 16 and 18, the types that cause most cervical and other anogenital cancers as well as some oropharyngeal cancers. Data from clinical trials have shown that both vaccines prevent cervical precancers; quadrivalent vaccine also has been shown to prevent vaginal, vulvar, and anal precancers. Because HPV 16 is responsible for the majority of noncervical cancers caused by HPV, the vaccines also might protect against other HPV-associated cancers. The Advisory Committee on Immunization Practices recommends routine vaccination of females aged 11 or 12 years with 3 doses of either vaccine and routine vaccination of males aged 11 or 12 years with 3 doses of quadrivalent vaccine** (6). Catch-up vaccination is recommended for females through age 26 years and for males

What is already known on this topic?

Persistent human papillomavirus (HPV) infection causes almost all cervical cancers and many vulvar, vaginal, penile, anal, and oropharyngeal cancers. The incidence of these cancers is influenced by sexual behaviors that lead to transmission of HPV, programs that screen for precancerous lesions, and the use of a recently introduced HPV vaccine.

What is added by this report?

An average of 33,369 HPV-associated cancers were diagnosed annually in the United States during 2004–2008 (10.8 per 100,000): 12,080 among males (8.1 per 100,000) and 21,290 among females (13.2). Of these, CDC estimates that 26,000 can be attributed to HPV: 18,000 among females and 8,000 among males.

What are the implications for public health practice?

Ongoing surveillance of HPV-associated cancers using high-quality population-based cancer registry data and consistent methodology is needed to monitor the impact of HPV vaccines, changes in cervical cancer screening practices, and changes in risk behaviors. Cervical cancer rates have decreased in the United States, largely as a result of the success of screening, but disparities still remain. HPV vaccine likely will help decrease cervical cancer rates further and reduce the disparities. Other HPV-associated cancers do not have approved screening programs; therefore, HPV vaccines are important prevention tools to reduce the incidence of noncervical cancers.

through age 21 years. In 2010, 32% of females aged 13–17 years had received 3 doses of HPV vaccine^{††}(2).

Most cases of invasive cervical cancer are preventable with regular screening for precancerous lesions (e.g., by Papanicolaou test) and follow-up of abnormal results. A recent analysis of data from the National Health Interview Survey found an overall cervical cancer screening rate of 83%, with lower rates among Asian, American Indian/Alaska Native, Hispanic, and foreign-born women (7). Higher rates of cervical cancer among black and Hispanic women might be the result, in part, of reduced access to screening and/or follow-up care (8). If smaller percentages of adolescent girls in the same demographic groups receive HPV vaccine, disparities in cervical cancers might increase (2). Cervical cancer screening guidelines recently changed in the United States, with guidelines now recommending screening intervals of 3 years, if screening with a Papanicolaou (Pap) test alone for women aged ≥21 years, or 5 years if screening with a Pap test and an HPV DNA test, which is an option for women aged ≥30 years (9).

Reasons for variations in rates of noncervical HPV-associated cancers by race/ethnicity and state are not clear but might be attributable, in part, to demographics, screening practices,

^{††} Only 1.4% of males aged 13–17 years received HPV vaccine in 2010; the recommendation for routine vaccination was published in December 2011.

[§] Maps available at <http://www.cdc.gov/cancer/hpv/statistics/state/index.htm>.

[¶] Data available at <http://www.wonder.cdc.gov/cancer>.

** Only the quadrivalent HPV vaccine is licensed for use in males.

TABLE 2. Estimated average annual percentage and number of cancers attributable to human papillomavirus (HPV), by anatomic site and sex — United States, 2004–2008

Site	Average annual no.*	% attributable to HPV [†]		No. attributable to HPV [‡]	
		%	Range	No.	Range
Cervix	11,967	96	(95–97)	11,500	(11,400–11,600)
Vulva	3,136	51	(37–65)	1,600	(1,200–2,000)
Vagina	729	64	(43–82)	500	(300–600)
Penis	1,046	36	(26–47)	400	(300–500)
Anus					
Female	3,089	93	(86–97)	2,900	(2,700–3,000)
Male	1,678	93	(86–97)	1,600	(1,400–1,600)
Oropharynx					
Female	2,370	63	(50–75)	1,500	(1,200–1,800)
Male	9,356	63	(50–75)	5,900	(4,700–7,000)

* Data are from population-based cancer registries that participate in the National Program of Cancer Registries and/or the Surveillance, Epidemiology, and End Results Program, and meet criteria for high data quality.

[†] Source: Gillison ML, Chaturvedi AK, Lowy DR. HPV prophylactic vaccines and the potential prevention of noncervical cancers in both men and women. *Cancer* 2008;113(10 Suppl):3036–46.

[‡] The estimated number of HPV-attributable cancers was calculated by multiplying the HPV-associated cancer counts (Table 1) by the percentage of each cancer attributable to HPV. Estimates rounded to the nearest 100. Female and male anal cancers do not equal the total number of anal cancers because of rounding.

tobacco use, or other factors related to HPV infection or persistence. Although analysis of oropharyngeal cancers was limited to cancer at specific anatomic sites most likely to be HPV-associated, variations in incidence of these cancers might be attributable to variations in smoking and alcohol use rather than, or in combination with, HPV infection. Studies on whether oral HPV infection interacts with these exposures to further increase the risk for oropharyngeal cancer are inconclusive (10). Population-based screening for noncervical HPV-associated cancers generally is not recommended.

The findings in this report are subject to at least three limitations. First, although population-based cancer registries provide a reliable system for counting invasive cancers, they typically do not capture information on HPV status or risk factors such as smoking. Not all cancers termed “HPV-associated” reflect actual HPV infections, and the numbers judged to be HPV-attributable are only estimates. Second, reporting of race and ethnicity uses data from medical records, which might be inaccurate in a small proportion of cases. Finally, current requirements for reporting cancer registry data are rigorous and require multiple steps; therefore, the most recent data are several years old.

Of the 33,369 cancers that occur each year in the United States at anatomic sites associated with HPV, approximately 26,000 can be attributed to HPV and might be preventable through the use of HPV vaccine. Ongoing surveillance of HPV-associated cancers using high-quality population-based registries is needed to monitor trends in cancer incidence that might result from increasing use of HPV vaccines, changes in cervical cancer screening practices, and changes in behaviors that increase risk for HPV infection, persistence, or progression.

References

1. International Agency for Research on Cancer. Monographs on the evaluation of carcinogenic risks to humans. Volume 90: human papillomaviruses. Lyon, France: World Health Organization, International Agency for Research on Cancer; 2007.
2. CDC. National and state vaccination coverage among adolescents aged 13 through 17 years—United States, 2010. *MMWR* 2011;60:1117–23.
3. Gillison ML, Chaturvedi AK, Lowy DR. HPV prophylactic vaccines and the potential prevention of noncervical cancers in both men and women. *Cancer* 2008;113(10 Suppl):3036–46.
4. CDC. United States Cancer Statistics (USCS) 1999–2007 cancer incidence and mortality data. Atlanta, GA: US Department of Health and Human Services, CDC; 2012. Available at <http://www.cdc.gov/uscs>. Accessed April 25, 2012.
5. Watson M, Saraiya M, Ahmed F, et al. Using population-based cancer registry data to assess the burden of human papillomavirus-associated cancers in the United States: overview of methods. *Cancer* 2008;113:2841–54.
6. CDC. Recommendations on the use of quadrivalent human papillomavirus vaccine in males—Advisory Committee on Immunization Practices (ACIP), 2011. *MMWR* 2011;60:1705–8.
7. CDC. Cancer screening—United States, 2010. *MMWR* 2012;61:41–5.
8. Benard VB, Lawson HW, Ehemann CR, Anderson C, Helsel W. Adherence to guidelines for follow-up of low-grade cytologic abnormalities among medically underserved women. *Obstet Gynecol* 2005;105(6):1323–8.
9. US Preventive Services Task Force. Screening for cervical cancer. Rockville, MD: US Preventive Services Task Force; 2012. Available at <http://www.uspreventiveservicestaskforce.org/uspstf/uspscerv.htm>. Accessed April 16, 2012.
10. Smith EM, Rubenstein LM, Haugen TH, Hamsikova E, Turek LP. Tobacco and alcohol use increases the risk of both HPV-associated and HPV-independent head and neck cancers. *Cancer Causes Control* 2010;21:1369–78.

***Pseudomonas aeruginosa* Respiratory Tract Infections Associated with Contaminated Ultrasound Gel Used for Transesophageal Echocardiography — Michigan, December 2011–January 2012**

In late December 2011, the Department of Epidemiology at Beaumont Health System (BHS) in Royal Oak, Michigan, noted an increase in the number of positive respiratory cultures in one surgical intensive-care unit (ICU), prompting further investigation. The increase in positive cultures was attributed entirely to *Pseudomonas aeruginosa*. Investigation by BHS staff members found that all of these positive cultures were related to use of ultrasound transmission gel from a single manufacturer during transesophageal echocardiography. Seven patients were infected with *P. aeruginosa* based on National Healthcare Safety Network (NHSN) criteria (1), and nine were colonized. Cultures from one open and one unopened bottle of the gel grew *P. aeruginosa* closely related to the outbreak strain based on molecular typing via repetitive extragenic palindromic polymerase chain reaction (rep-PCR). The Oakland County Health Department, the Michigan Department of Community Health, and the Food and Drug Administration (FDA) were notified of the findings. On January 23, all implicated ultrasound gel in multiuse bottles was removed from BHS facilities and replaced with a single-use, sterile ultrasound gel for all potentially invasive procedures. On April 18, FDA issued a Safety Communication* advising health-care professionals and facilities not to use certain lot numbers of the ultrasound transmission gel and further advising that the only ultrasound gel that is sterile is unopened gel in containers labeled as sterile. To date, no further respiratory cultures have been positive for *P. aeruginosa*.

Surveillance for nosocomial infection at BHS is driven by results of clinical microbiology cultures. Positive cultures are reviewed using a combination of microbiology reports and paper or electronic medical records to determine infections and colonizations. Initial review found *P. aeruginosa* in respiratory specimens taken from endotracheal tubes in 10 patients in a single surgical ICU in December. No cultures of these patients' surgical sites or blood grew *P. aeruginosa*. The same unit had averaged less than three respiratory tract cultures positive for *P. aeruginosa* monthly during the preceding 11 months and had only one infection by NHSN criteria during that period.

Review of the 10 *P. aeruginosa* cultures revealed that all patients had undergone cardiovascular surgery. No clustering by operating room, surgeon, operating room staff member, ICU room number, or nursing staff was observed. Because

all isolates were from the respiratory tract, the initial focus included a review of postoperative nursing and respiratory-care practices, respiratory therapy equipment management, and anesthesia practice and equipment management. No clustering by respiratory medications administered was observed. Discussion with operating room staff members revealed that a unique aspect of these patients' surgeries included the use of an intraoperative transesophageal echocardiogram (TEE). TEEs involve the insertion of a probe with an ultrasound conducting tip into a patient's esophagus and are used during cardiovascular surgery to aid in visualization of the posterior of the heart. The TEE probe is coated with a coupling gel and then inserted by an anesthesiologist before surgical incision. Their duration of placement depends on the specific procedure being performed. Environmental cultures of TEE probes, storage tubes, and work surfaces were performed, and all TEE probes were inspected. All cultures were negative, and only one probe had a mechanical defect; this probe was removed from use.

Intensified surveillance (performing respiratory tract cultures on all mechanically ventilated patients in this surgical ICU) during January 6–20 identified six additional patients colonized with *P. aeruginosa* (of the 20 patients tested). All six of these patients also had undergone cardiovascular surgery. Surveillance respiratory cultures from another surgical ICU identified only one patient colonized (of the 11 patients tested) with *P. aeruginosa*; this isolate had a different antibiotic susceptibility pattern from those of the 16 isolates found earlier. Of the 16 patients identified during the outbreak, two had pneumonia, five had tracheobronchitis, and nine had respiratory tract colonization only. Time from surgery to identification of a positive culture from a respiratory tract specimen ranged from 2 to 14 days (median: 5 days). The patients had undergone various surgical procedures. Those who had undergone valvular surgery alone (n = 13) were at significantly higher risk (relative risk = 5.7, 95% confidence interval = 1.75–18.86) for *Pseudomonas* infection or colonization than those who had undergone coronary artery bypass grafting alone (n = 32) (Table). The investigation noted that patients undergoing valvular surgery have TEE probes in place during nearly the entire procedure, whereas those undergoing coronary artery bypass grafting had shorter durations of TEE use. A review of operative times found that procedures lasting ≥ 5 hours (n = 70) were more frequently associated with *P. aeruginosa* infection

* Available at <http://www.fda.gov/medicaldevices/safety/alertsandnotices/ucm299409.htm>.

TABLE. Number of cultures positive and total number of surgical procedures among patients with respiratory tract cultures growing *Pseudomonas aeruginosa*, and patients with respiratory tract cultures growing *P. aeruginosa* per 100 surgical procedures performed, by type of surgical procedure — Beaumont Health System, Michigan, December 9, 2011–January 20, 2012

Type of surgical procedure	No. of cultures positive	Total no. of procedures	%
Coronary artery bypass (CABG)	3	32	9
Valvular surgery	7	13	54
CABG + valvular surgery	3	15	20
Minimally invasive valve repair	2	27	7
Other	1	13	8

or colonization (relative risk = 6.4, 95% confidence interval = 0.89–46.45) than procedures lasting <5 hours (n = 30).

The investigation focused further on manipulations of the respiratory and gastrointestinal tract. An ultrasound transmission gel, Other-Sonic (Pharmaceutical Innovations, Inc., Newark, New Jersey), which was not labeled or sold as a sterile product, was used with TEE probes. The multidose containers of gel were collected and replaced with a single-use, sterile product on January 23. After this change, no additional respiratory cultures with *P. aeruginosa* were observed.

Molecular typing was performed on the 10 isolates on January 26, and all were determined to be >99% similar by rep-PCR. Cultures of the four previously opened Other-Sonic ultrasound transmission gel bottles removed from the operating room were performed; one of four samples grew *P. aeruginosa*, and molecular typing revealed it to be highly related (>99%) to the outbreak strain. Five other strains of *P. aeruginosa* isolated throughout the hospital during the outbreak period also were analyzed and determined to be unrelated to each other or the outbreak strain. Two bottles of sealed, unopened Other-Sonic ultrasound transmission gel subsequently were cultured, one of which grew *P. aeruginosa*. At this point a health-care system-wide recall of all bottles of Other-Sonic ultrasound transmission gel was initiated, local and state health departments were contacted, and FDA was notified. Additional molecular typing studies (using rep-PCR) showed that the *Pseudomonas* isolate from the sealed bottle also was >99% similar to the outbreak strain, strongly suggesting contamination of the product during manufacturing, packaging, storage, or shipping.

Reported by

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What is already known on this topic?

Medical gels have been linked to outbreaks of infection in several reports, including reports of gels contaminated at the site of packaging. As a result, Health Canada in 2004 issued recommendations for minimizing the risk for infection from medical gels. No such guidelines exist in the United States.

What is added by this report?

An outbreak of seven cases of *Pseudomonas aeruginosa* respiratory tract infection and nine instances of respiratory tract colonization was linked to contaminated ultrasound gel. *P. aeruginosa* isolates found in 10 patients, one of four opened gel bottles in use in the operating room, and one of two unopened, sealed gel bottles were found to be more than 99% similar by molecular typing.

What are the implications for public health practice?

Because of the risk that an ultrasound gel might be contaminated with *P. aeruginosa* or other bacteria, single-use, sterile products should be used for invasive procedures and procedures involving contact with nonintact skin or mucous membranes.

Editorial Note

This report describes an outbreak of *Pseudomonas aeruginosa* respiratory tract colonization and infection related to the use of contaminated ultrasound transmission gel. Sixteen cardiovascular surgery patients were affected during the outbreak, seven with infection per NHSN criteria, and nine with colonization. Initial investigation suggested the possibility that the TEE probes were the source of the outbreak, given that contaminated TEE probes have been linked to pulmonary infection outbreaks of *Legionella* previously (2). However, surveillance cultures of the probes were negative, and there was no evidence that all case patients were linked to the use of a particular probe. The ultrasound transmission gel, however, was contaminated with *P. aeruginosa*. Although contamination during use was initially suspected, the fact that one of two tested bottles of sealed, unopened product was contaminated with *P. aeruginosa* suggests that the contamination might have occurred before the product reached BHS.

Contaminated ultrasound gels have been associated with outbreaks of infection in various settings and with various organisms, including *Klebsiella* (3), *Burkholderia* (4,5), *Achromobacter* (6), and *Staphylococcus aureus* (7). Although most of these outbreaks were believed to have occurred from inappropriate use of products, in one circumstance it was determined that the gel had been contaminated at the site of production (4). Although these gels contain parabens or methyl benzoate, which are thought to render them bacteriostatic, some Gram-negative bacteria can degrade these components (4), and investigations of several reported outbreaks suggest

extrinsic contamination of gels might easily occur (3,5–7). One study demonstrated that an ultrasound gel had no intrinsic antimicrobial properties (8), and interestingly, results of another in vitro study suggested *Pseudomonas* spp. might actually survive for shorter periods in ultrasound gel compared with *S. aureus* or *Escherichia coli* (9). No ingredient information is available publicly for Other-Sonic ultrasound gel.

Numerous products are available to be used as ultrasound transmission gels. No national guidelines exist in the United States recommending specific types of gel for specific procedures. However, in 2004, Health Canada issued recommendations for minimizing the health risks of using gels (10). These recommendations suggested use of single-use, sterile gels for invasive procedures that pass through a tissue, for all studies involving neonates, for all procedures involving sterile equipment or non-intact skin, and for procedures on intact mucous membranes. The results of this report further support a recommendation for the use of only sterile gels for invasive procedures and procedures involving contact with nonintact skin or mucous membranes. Moreover, because only unopened ultrasound gel containers labeled as sterile should be considered sterile and extrinsic contamination might easily occur, only single-use sterile products should be used for such purposes.

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References

1. Horan TC, Andrus M, Dudeck MA. CDC/NHSN surveillance definition of health care-associated infection and criteria for specific types of infection in the acute care setting. *Am J Infect Control* 2008; 36:309–32.
2. Levy PY, Teysseire N, Etienne J, Raoult D. A nosocomial outbreak of *Legionella pneumophila* caused by contaminated transesophageal echocardiography probes. *Infect Control Hosp Epidemiol* 2003; 24:619–22.
3. Gaillot O, Maruejols C, Abachin E, et al. Nosocomial outbreak of *Klebsiella pneumoniae* producing SHV-5 extended-spectrum beta-lactamase, originating from contaminated ultrasonography coupling gel. *J Clin Microbiol* 1998;36:1357–60.
4. Hutchinson J, Runge W, Mulvey M, Norris et al. *Burkholderia cepacia* infections associated with intrinsically contaminated ultrasound gel: the role of microbial degradation of parabens. *Infect Control Hosp Epidemiol* 2004;25:291–6.
5. Jacobson M, Wray R, Kovach D, Henry D, Speert D, Matlow A. Sustained endemicity of *Burkholderia cepacia* complex in a pediatric institution, associated with contaminated ultrasound gel. *Infect Control Hosp Epidemiol* 2006;27:362–6.
6. Olshtain-Pops K, Block C, Temper V, et al. An outbreak of *Achromobacter xylosoxidans* associated with ultrasound gel used during transrectal ultrasound guided prostate biopsy. *J Urol* 2011;185:144–7.
7. Weist K, Wendt C, Petersen LR, Versmold H, Ruden H. An outbreak of pyoderma among neonates caused by ultrasound gel contaminated with methicillin-susceptible *Staphylococcus aureus*. *Infect Control Hosp Epidemiol* 2000;21:761–4.
8. Muradali D, Gold WL, Phillips A, Wilson S. Can ultrasound probes and coupling gel be a source of nosocomial infection in patients undergoing sonography? An in vivo and in vitro study. *Am J Roentgenol* 1995;164:1521–4.
9. Ohara T, Itoh Y, Itoh K. Ultrasound instruments as possible vectors of staphylococcal infection. *J Hosp Infect* 1998;40:73–7.
10. Health Canada. Notice to hospitals: important safety information on ultrasound and medical gels. Ottawa, Canada: Health Canada; 2004. Available at http://www.hc-sc.gc.ca/dhp-mps/medeff/advisories-avis/prof/_2004/ultrasound_2_nth-ah-eng.php. Accessed April 11, 2012.

Tracking Progress Toward Global Polio Eradication, 2010–2011

In January 2012, polio eradication was declared a “programmatic emergency for global public health” by the Executive Board of the World Health Organization (WHO) (1). Since the Global Polio Eradication Initiative (GPEI) began in 1988 (2), progress has been tracked by surveillance of acute flaccid paralysis (AFP) cases and testing of linked stool specimens for polioviruses (PVs) in WHO-accredited Global Polio Laboratory Network (GPLN) laboratories, complemented by sewage testing (environmental surveillance) in selected areas. Monitoring AFP surveillance quality at national and subnational administrative levels using standard performance indicators identifies potential gaps where PV circulation might go undetected; monitoring specimen transport and laboratory reporting timeliness identifies areas where reporting delays could lead to late response, permitting ongoing transmission. This report provides an assessment of 2010–2011 performance indicators for AFP surveillance at national and subnational levels in polio-affected countries and laboratory reporting at the regional level, updated from 2009–2010 (3). Overall, 16 (62%) of 26 countries with circulating wild PV (WPV) met national AFP surveillance indicator targets during both 2010 and 2011. All three countries with reestablished WPV transmission and 16 of 19 countries with WPV outbreaks had substantial proportions (>20%) of their respective populations living in areas with underperforming surveillance during 2010 or 2011. Targets for timely reporting of PV isolation and type characterization results were met in three of six WHO regions in 2010 and five regions in 2011. To achieve polio eradication, efforts are needed to improve AFP surveillance and laboratory performance.

AFP Surveillance

AFP surveillance detects paralytic illness of many causes, including paralytic poliomyelitis caused by WPV. The objectives of AFP surveillance are to identify areas with PV transmission and areas with low AFP reporting where polio cases might go undetected. AFP surveillance sensitivity is measured by the annual proportion of AFP cases that are negative for WPV among children aged <15 years (nonpolio AFP [NPAFP] rate). Adequacy of stool specimen collection is measured by the proportion of AFP cases from which two adequate stool specimens (collected ≥ 24 hours apart and within 14 days after paralysis onset) arrived in good condition at a GPLN laboratory.

The WHO Region of the Americas, the Western Pacific Region, and European Region are certified as polio-free. However, four countries in the European Region (Kazakhstan, Tajikistan, Turkmenistan, and the Russian Federation) experienced WPV outbreaks in 2010, and China in the Western Pacific Region experienced an outbreak in 2011 (Table 1). Collectively, the polio-free

regions maintained overall AFP surveillance sensitivity at the WHO-specified target NPAFP rate of ≥ 1 case per 100,000 children aged <15 years, and all but the Region of the Americas met the WHO-specified target of $\geq 80\%$ AFP cases with adequate stool specimens at the regional level during 2010 and 2011 (Table 1).

During 2010–2011, a total of 21 countries in the polio-endemic African, Eastern Mediterranean, and South-East Asia regions experienced WPV transmission. Endemic WPV transmission continued in Afghanistan, Nigeria, and Pakistan. As of February 2012, endemic WPV transmission had ceased in India, with 42 WPV cases reported in 2010, one case reported in 2011, and no cases reported since (4,5). Reestablished transmission continued in the previously polio-free countries of Angola, Chad, and Democratic Republic of Congo (DRC) (6), and WPV outbreaks occurred in 13 African countries and Nepal during 2010–2011 (Table 1). A target NPAFP rate of ≥ 2 cases per 100,000 children aged <15 years has been established in the polio-endemic regions, all countries reporting WPV cases, and in neighboring countries at risk for WPV outbreaks (7). The three regions that had polio-endemic countries met the ≥ 2 NPAFP rate target and the $\geq 80\%$ specimen adequacy regional target during 2010 and 2011 (Table 1).

Twenty-four (92%) of 26 polio-affected countries met the ≥ 2 NPAFP rate target in both 2010 and 2011 (Table 1). Eighteen (69%) of 26 of polio-affected countries met the $\geq 80\%$ target for the proportion of AFP cases with adequate stool specimens in both 2010 and 2011 (Table 1). Among polio-affected countries, surveillance quality varied substantially at the subnational level (states and provinces). A NPAFP rate of ≥ 2 cases per 100,000 children aged <15 years in $\geq 80\%$ of subnational areas was achieved in 19 (73%) countries in 2010, 16 (62%) countries in 2011, and 14 (54%) countries in both years (Table 1, Figure). Meeting the standard of $\geq 80\%$ specimen adequacy in $\geq 80\%$ of subnational areas was achieved in 14 (54%) countries in 2010, 12 (46%) countries in 2011, and 11 (42%) countries in both years (Table 1). The countries where $\geq 80\%$ of the population lived in areas with AFP surveillance meeting targets during 2010 and 2011 included all four countries with endemic WPV transmission, none of the three countries with reestablished WPV transmission, and three of 19 countries with WPV outbreaks.

GPLN

The GPLN, established in 1990 by WHO, consists of 146 laboratories in 97 countries. Accredited members in the network follow standardized protocols and procedures appropriate for each laboratory tier: 1) isolation and identification of PVs; 2) intratypic differentiation (ITD) of the three (1, 2, and 3) WPV serotypes, Sabin vaccine-related PVs, and vaccine-derived PVs

TABLE 1. National and subnational acute flaccid paralysis (AFP) surveillance indicators and number of confirmed wild poliovirus (WPV) cases in persons with AFP, by World Health Organization (WHO) region and polio-affected country, 2010 and 2011*

WHO region†/ country	2010							2011						
	No. of AFP cases	NPAFP rate [§]	% subnational areas with NPAFP rate ≥2 [¶]	National % AFP cases with adequate specimens**	% subnational areas with ≥80% adequate specimens	% population in areas meeting both indicators	No. of confirmed WPV cases	No. of AFP cases	NPAFP rate [§]	% subnational areas with NPAFP rate ≥2 [¶]	National % AFP cases with adequate specimens**	% subnational areas with ≥80% adequate specimens	% population in areas meeting both indicators	No. of confirmed WPV cases
Americas	2,006	1.2	—	79	—	—	—	1,728	1.0	—	78	—	—	—
African	16,500	4.3	—	87	—	—	657	16,635	4.4	—	88	—	—	350
Angola ^{††}	387	3.1	94	87	83	76	33	257	2.3	56	91	89	43	5
CAR ^{§§}	137	7.2	100	89	86	87	—	142	6.0	100	81	71	68	4
Chad ^{††}	302	4.5	94	67	17	12	26	468	5.7	100	75	39	33	132
Congo ^{§§}	582	5.1	100	23	27	7	441	93	3.1	60	74	55	20	1
Côte d'Ivoire ^{§§}	309	3.3	95	79	53	47	—	511	5.1	95	64	0	0	36
DRC ^{††}	2,194	5.5	100	73	18	24	100	2,216	4.9	100	79	27	34	93
Gabon ^{§§}	24	2.7	50	46	30	19	—	30	2.9	0	60	33	10	1
Guinea ^{§§}	215	4.0	100	67	13	9	—	203	3.7	100	68	0	0	3
Kenya ^{§§}	403	2.2	88	87	88	66	—	563	3.1	88	82	75	49	1
Liberia ^{§§}	50	2.8	60	88	79	36	2	55	3.3	40	85	80	34	—
Mali ^{§§}	171	2.2	50	93	100	53	4	210	2.7	100	84	67	64	7
Mauritania ^{§§}	65	4.4	86	97	100	90	5	53	3.8	100	92	92	90	—
Niger ^{§§}	358	4.4	100	73	25	32	2	319	3.9	88	72	13	3	5
Nigeria ^{¶¶}	5,996	7.9	100	93	100	100	21	6,096	8.1	100	93	100	100	62
Senegal ^{§§}	312	5.3	100	59	9	6	18	115	2.1	55	79	55	38	—
Sierra Leone ^{§§}	168	6.3	100	86	75	77	1	173	6.6	100	83	50	57	—
Uganda ^{§§}	424	2.9	61	87	75	45	4	466	3.2	53	87	71	41	—
Eastern Mediterranean	11,338	5.6	—	91	—	—	169	11,669	5.7	—	90	—	—	278
Afghanistan^{¶¶}	1,572	8.8	100	93	97	95	25	1,827	10.0	100	92	91	91	80
Pakistan^{¶¶}	5,392	6.7	88	88	100	99	144	5,696	7.0	100	88	88	95	198
European	2,087	1.2	—	86	—	—	478	1,542	1.3	—	86	—	—	—
Kazakhstan ^{§§}	112	3.5	73	99	100	80	1	100	3.2	93	97	94	88	—
Russian Federation ^{§§}	387	1.7	28	95	93	26	14	354	1.6	28	93	88	18	—
Tajikistan ^{§§}	583	5.3	80	87	80	53	460	52	2.1	60	96	100	69	—
Turkmenistan ^{§§}	50	3.2	83	100	100	91	3	33	2.3	50	100	100	32	—
South-East Asia	60,456	11.2	—	83	—	—	48	65,604	12.2	—	85	—	—	1
India ^{¶¶}	55,785	12.4	94	83	76	87	42	59,683	13.6	91	84	79	87	1
Nepal ^{§§}	604	5.4	100	88	80	90	6	566	5.1	100	88	80	90	—
Western Pacific	6,401	1.8	—	89	—	—	—	7,247	2.0	—	90	—	—	21
China ^{§§}	5,285	1.9	27	91	100	37	—	6,136	2.8	77	94	100	89	21
Total	98,788	5.6	—	85	—	—	1,352	104,425	5.9	—	86	—	—	650

Abbreviations: NPAFP = nonpolio AFP, CAR = Central African Republic, DRC = Democratic Republic of Congo.

* Data as of February 21, 2012.

† Regional NPAFP rates use United Nations Development Program populations as denominators, and thus tend to be higher than country rates, which use their own subnational populations as denominators. Regional data available at http://apps.who.int/immunization_monitoring/en/diseases/poliomyelitis/case_count.cfm.

§ Per 100,000 persons aged <15 years.

¶ For subnational areas (states and provinces) with populations >100,000.

** Standard WHO target is adequate stool specimen collection from ≥80% of AFP cases, in which two specimens are collected ≥24 hours apart and within 14 days of paralysis onset, shipped on ice or frozen ice packs, and arriving in good condition in a WHO-accredited laboratory. Stool adequacy proportions from regions and countries in the European Region and China do not include the criteria of good stool specimen condition. For the Americas, an adequate stool specimen is one specimen collected within 14 days of paralysis onset.

†† Countries with reestablished WPV transmission.

§§ Countries with WPV outbreaks.

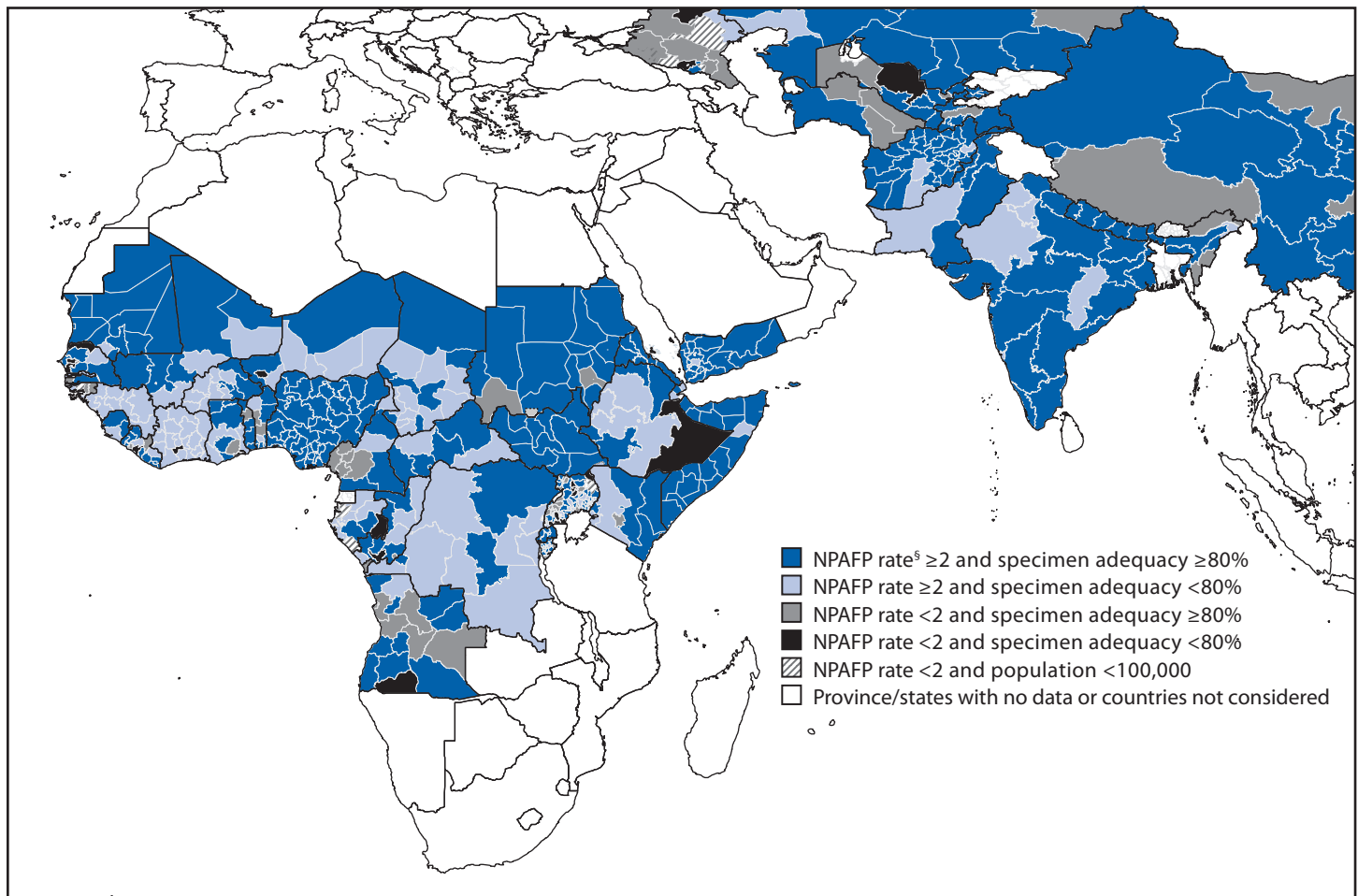
¶¶ Countries with endemic WPV transmission.

(VDPVs) (8); and 3) genomic sequencing of the VP1 region of WPVs and VDPVs to investigate transmission links among isolates. The laboratory performance standard for five of the six WHO regions is to report PV isolation results for ≥80% of specimens within 14 days of receipt; in 2011, the European Region and China (Western Pacific Region) maintained a standard of PV isolation and serotyping within 28 days. Although the introduction of real-time polymerase chain reaction assays

into laboratories allows ITD testing within 7 days (9), the official standard in five WHO regions for timely reporting of ITD results is within 60 days of paralysis onset, allowing time for case detection, investigation, and transport of specimens; however, the Eastern Mediterranean Region introduced an ITD reporting standard of 45 days.

Targets for timely reporting of PV detection were met in five WHO regions in 2011, compared with four WHO regions

FIGURE. Combined performance indicators for the quality of acute flaccid paralysis (AFP) surveillance* in subnational areas (states and provinces) of 26 current or recently polio-affected countries and neighboring countries, 2011[†]



Abbreviation: NPAFP = nonpolio AFP.

* The Global Polio Eradication Initiative 2010–2012 strategic plan sets the following targets for countries with current or recent wild poliovirus (WPV) transmission and their states/provinces: 1) an NPAFP detection rate of ≥ 2 cases per 100,000 persons aged <15 years, and 2) adequate stool specimen collection from $\geq 80\%$ of AFP cases, with specimen adequacy defined as two specimens collected ≥ 24 hours apart, both within 14 days of paralysis onset, shipped on ice or frozen ice packs, and arriving in good condition (without leakage or desiccation) at a World Health Organization–accredited laboratory.

[†] Data are for AFP cases with onset during 2011, reported as of February 21, 2012.

[§] Per 100,000 persons aged <15 years.

in 2010, when delays occurred during the European outbreak because of batching of samples (Table 2). Five WHO regions met the standard of reporting ITD results within 60 days of paralysis onset for $\geq 80\%$ of specimens for both 2010 and 2011 (Table 2). The Region of the Americas was the only region meeting neither timeliness standard in 2011.

GPLN tested 206,899 stool specimens from AFP case investigations in 2011, a 6% increase from the 194,374 stool specimens tested in 2010. A total of 10,235 PV isolates (including 1,570 WPVs and 86 VDPVs) were detected in 2011 from AFP case specimens, a 13% increase from 9,090 PV isolates (including 1,679 WPVs and 188 VDPVs) in 2010 because of an increase in Sabin-related PV isolation. During 2010–2011, a total of

274 (1.7%) of the 16,076 vaccine-related viruses screened were determined by genomic sequencing to be VDPVs (Table 2). Genomic sequencing identified three genotypes in the African Region during 2011: WPV1 West Africa-B (WEAF-B) in Chad, Central African Republic, Kenya, Niger, and Nigeria; WPV3 WEAF-B in Chad, Côte d'Ivoire, Guinea, Mali, and Nigeria; and WPV1 South Asia (SOAS) in Angola, Congo, DRC, and Gabon. In the Eastern Mediterranean Region, WPV3 SOAS isolates were detected in Pakistan, and WPV1 SOAS isolates were detected in Pakistan and Afghanistan. Sequence analysis provided evidence that AFP surveillance of varying qualities had missed chains of WPV transmission during 2011 in Afghanistan, Angola, Chad, Côte d'Ivoire, Kenya, Mali, Nigeria, and Pakistan.

TABLE 2. Number of poliovirus (PV) isolates from stool specimens of persons with acute flaccid paralysis, and timing of results, by World Health Organization (WHO) region, 2010 and 2011*

WHO region	2010						2011					
	No. of specimens	No. of PV isolates			% PV isolation results on time [¶]	% ITD results within 60 days ^{**}	No. of specimens	No. of PV isolates			% PV isolation results on time [¶]	% ITD results within 60 days ^{**}
		Wild	Sabin [†]	VDPV [§]				Wild	Sabin [†]	VDPV [§]		
African	34,689	798	2,535	166	95	72	36,942	1,035	2,476	46	91	86
Americas	1,459	0	30	0	79	100	1,762	0	36	1	5	— ^{††}
Eastern Mediterranean	26,325	326	981	9	92	92	23,011	512	807	17	98	97
European	3,091	508	93	0	75	100	3,188	0	87	0	96	99
South-East Asia	116,041	47	3,329	12	94	99	127,543	2	4,907	15	97	98
Western Pacific	12,769	0	255	1	95	80	14,453	21	266	7	97	86
Total	194,374	1,679	7,223	188	94	92	206,899	1,570	8,579	86	95	94

Abbreviations: VDPV = vaccine-derived poliovirus, ITD = intratypic differentiation.

* Data as of March 26, 2012.

[†] Either concordant Sabin-like results in ITD tests and VDPV screening, or <1% sequence difference compared with Sabin vaccine virus.

[§] For PV types 1 and 3, 10 or more VP1 nucleotides difference from the respective Sabin PV; for PV type 2, six or more VP1 nucleotides difference from Sabin type 2 PV.

[¶] Results reported within 14 days for laboratories in the following WHO regions: African, Americas, Eastern Mediterranean, and South-East Asia, and Western Pacific (not including China). Results reported within 28 days for the European Region and China (Western Pacific Region).

^{**} Results reported within 60 days of paralysis onset for all WHO regions except Eastern Mediterranean region, which reported within 45 days of paralysis onset.

^{††} ITD reporting results from the Region of the Americas were unavailable as of March 26, 2012.

Environmental Surveillance

During 2010–2011, environmental surveillance of WPV transmission was accomplished through testing of sewage samples (10) in 21 countries without active PV transmission (20 countries in the European Region and Egypt in the Eastern Mediterranean Region) and three countries with WPV transmission (India, Nigeria, and Pakistan). In India, sewage sampling expanded from 10 sites in two states in 2010 to 15 sites in four states in 2011. The last detected WPV from sewage testing in India was in November 2010 (4). In Pakistan (with 18 sites in four states), widespread WPV1 circulation was detected in sewage during 2010–2011, although WPV3 has not been detected in sewage since October 2010. Sewage testing in Nigeria began in 2011 (with three sites in Kano state) and detected only circulating VDPVs (and no WPVs) during 2011, despite the continued occurrence of WPV cases (3).

Reported by

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Editorial Note

The GPEI relies on sensitive AFP surveillance (complemented by environmental surveillance) and virologic testing and sequencing to track the transmission of PVs globally. Standardized case definitions, protocols for case investigation and laboratory testing, and surveillance performance

indicators, allow comparability across countries and WHO regions. Assessing subnational surveillance indicators is important because national indicators can mask substantial gaps in surveillance quality.

AFP surveillance performance among polio-affected countries during 2010–2011 was varied, with some key countries not meeting indicator targets. Genomic sequence analysis of WPV isolates provided evidence that several chains of transmission were missed by deficiencies in AFP surveillance during 2010–2011, including some countries where subnational performance standards were met. Among countries with reestablished transmission, Angola had subnational gaps in NPAFP detection that worsened from 2010 to 2011; although Chad and DRC struggled to attain subnational stool specimen adequacy standards in both years, both showed slight improvement from 2010 to 2011. Countries with WPV outbreaks also had surveillance gaps, with 12 (63%) of 19 countries having majorities of their populations residing in subnational areas with poor AFP surveillance in either 2010 or 2011. The clustering of states and provinces with substandard surveillance indicators within countries and across borders (as observed in Central and West Africa) also is concerning. Detailed AFP surveillance reviews are being conducted or planned in countries with suboptimal surveillance indicators or with evidence of missed WPV transmission from genomic sequence analysis to assess reasons for poor performance in AFP case detection, specimen collection, and reporting, and to make recommendations for improving surveillance quality.

Combined with sensitive AFP surveillance, environmental surveillance has provided additional evidence to monitor the

absence of WPV transmission in India and the extent of WPV transmission in Pakistan. However, a lack of WPV detection in sewage does not exclude the possibility of ongoing transmission if areas and populations at highest risk for WPV transmission are not included. Environmental surveillance could be expanded in countries where undetected transmission is a concern or where the risk for importation is high. Feasibility assessments in Luanda, Angola, and Nairobi, Kenya, are planned.

GPLN overall performance during 2010–2011 remains high and actually improved in 2011, despite continued annual increases in workload (9% in 2010 [3] and 6% in 2011). Over 90% of PV isolation and ITD results were reported within the target periods. The prompt provision of laboratory test results allows a more rapid and well-targeted response. Timely ITD of PVs in the African Region has been complicated by difficulties in international specimen transport (3), which is a challenge that needs to be addressed continually.

To achieve polio eradication, national immunization programs and partners should make greater efforts to strengthen polio surveillance and to improve monitoring at all administrative levels, especially in polio-affected countries and neighboring countries at risk for outbreaks. Evidence also suggests that maintenance of high-quality AFP surveillance in polio-free regions is critical. These efforts require strengthened commitments on all levels and the provision of sufficient human and financial resources to meet target indicators at both national and subnational levels.

References

1. World Health Organization Executive Board. Poliomyelitis: intensification of the global eradication initiative. Geneva, Switzerland: World Health Organization; 2012. Available at http://apps.who.int/gb/ebwha/pdf_files/eb130/b130_r10-en.pdf. Accessed April 10, 2012.
2. CDC. Progress toward interruption of wild poliovirus transmission—worldwide, January 2010–March 2011. *MMWR* 2011;60:582–6.
3. CDC. Tracking progress toward global polio eradication—worldwide, 2009–2010. *MMWR* 2011;60:441–5.
4. CDC. Progress toward poliomyelitis eradication—India, January 2010–September 2011. *MMWR* 2011;60:1482–6.
5. World Health Organization. Global Polio Eradication Initiative: three to go. Geneva, Switzerland: World Health Organization; 2012. Available at <http://www.polioeradication.org/tabid/461/iid/201/default.aspx>. Accessed April 10, 2012.
6. CDC. Progress toward global polio eradication—Africa, 2011. *MMWR* 2012;61:190–4.
7. World Health Organization. Conclusions and recommendations of the Advisory Committee on Poliomyelitis Eradication, Geneva, 11–12 October 2005. *Wkly Epidemiol Rec* 2010;80:410–6.
8. CDC. Update on vaccine-derived polioviruses—worldwide, July 2009–March 2011. *MMWR* 2011;60:846–50.
9. CDC. Laboratory surveillance for wild and vaccine-derived polioviruses—worldwide, January 2008–June 2009. *MMWR* 2009;58:950–4.
10. Deshpande JM, Shetty SJ, Siddiqui ZA. Environmental surveillance system to track wild poliovirus transmission. *Appl Environ Microbiol* 2003;69:2919–27.

What is already known on this topic?

The Global Polio Eradication Initiative (GPEI) tracks progress towards polio eradication through surveillance of acute flaccid paralysis (AFP) cases, testing of linked stool specimens for polioviruses (PVs), typing and sequencing of PVs to track transmission, as well as environmental sewage testing. Standardized case definitions, case investigation and laboratory protocols, and performance indicator targets allow comparability of surveillance performance across countries over time.

What is added by this report?

AFP surveillance is suboptimal in some key countries and unimproved overall. During 2010–2011, only 62% of countries with circulating wild PV (WPV) met national AFP surveillance indicator targets. Surveillance at the subnational level varied, and sequencing of WPV isolates provided evidence that several chains of transmission were missed by AFP surveillance deficiencies. All three countries with reestablished WPV transmission and 16 of 19 countries with WPV outbreaks had >20% of their respective populations living in areas with underperforming surveillance during 2010 or 2011.

What are the implications for public health practice?

Assessing subnational surveillance quality indicators is important in identifying areas where WPV transmission might go undetected and would otherwise be masked by national indicators. Given the current elevated status of polio eradication as a “programmatically emergency for global public health,” efforts are needed to improve or maintain sufficient AFP surveillance quality at all administrative levels to track PV transmission and provide timely response to new PV importations.

Vital Signs: Unintentional Injury Deaths Among Persons Aged 0–19 Years — United States, 2000–2009

On April 16, 2012, this report was posted as an MMWR Early Release on the MMWR website (<http://www.cdc.gov/mmwr>).

Abstract

Background: Unintentional injuries are the leading cause of death in the United States for persons aged 1–19 years and the fifth leading cause of death for newborns and infants aged <1 year. This report describes 10-year trends in unintentional injury deaths among persons aged 0–19 years.

Methods: CDC analyzed 2000–2009 mortality data from the National Vital Statistics System by age group, sex, race/ethnicity, injury mechanism, and state.

Results: From 2000 to 2009, the overall annual unintentional injury death rate decreased 29%, from 15.5 to 11.0 per 100,000 population, accounting for 9,143 deaths in 2009. The rate decreased among all age groups except newborns and infants aged <1 year; in this age group, rates increased from 23.1 to 27.7 per 100,000 primarily as a result of an increase in reported suffocations. The poisoning death rate among teens aged 15–19 years nearly doubled, from 1.7 to 3.3 per 100,000, in part because of an increase in prescription drug overdoses (e.g., opioid pain relievers). Childhood motor vehicle traffic–related death rates declined 41%; however, these deaths remain the leading cause of unintentional injury death. Among states, unintentional injury death rates varied widely, from 4.0 to 25.1 per 100,000 in 2009.

Conclusions and Implications for Public Health Practice: Although the annual rate is declining, unintentional injury remains the leading cause of death among children and adolescents in the United States, led by motor vehicle traffic–related deaths. Death rates from infant suffocation and teen poisoning are increasing. The 2012 National Action Plan for Child Injury Prevention provides actions in surveillance, research, communication, education, health care, and public policy to guide efforts in saving lives by reducing injuries.

Introduction

Unintentional injuries are the leading cause of death in the United States among persons aged 1–19 years, accounting for 37% of all deaths in this age group in 2009, and the fifth leading cause of death among newborns and infants aged <1 year (1). Unintentional injury deaths are responsible for more years of potential life lost before age 65 years than cancer, heart disease, or any other cause of death, in part because children and adolescents die from unintentional injuries much more commonly than other causes (1). For every childhood injury death, more than 1,000 are treated or receive medical consultation for a nonfatal injury (2). In 2009, child and adolescent unintentional injuries resulted in approximately 9,000 deaths, 225,000 hospitalizations, and 8.4 million patients treated and released from emergency departments (1). Unintentional injuries occurring in 2005 that resulted in death, hospitalization, or an emergency department visit cost nearly \$11.5 billion in medical expenses (1). These injuries are preventable (3,4), and effective interventions for reducing childhood injuries are less costly than

the medical expenses and productivity losses associated with those injuries (5).

The high incidence and preventability of child and adolescent unintentional injuries highlight the need for public health action. Although unintentional injury death rates have decreased in recent decades (6), rates remain high in some population subgroups and states (7). This report summarizes trends in unintentional injury deaths among persons aged 0–19 years, from 2000 to 2009, by age group, sex, race/ethnicity, injury mechanism, and state, using data from the National Vital Statistics System.

Methods

CDC's National Vital Statistics System collects death certificate data from 50 states and the District of Columbia.* Annual mortality files were analyzed for deaths among persons aged 0–19 years. Unintentional injury deaths were defined as those with an underlying cause of death classified by *International*

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

Classification of Diseases, 10th Revision (ICD-10) external cause of injury codes as V01–X59 or Y85–Y86. Deaths were categorized by mechanism as drowning, fall, fire/burn, motor vehicle traffic–related, other transportation-related, poisoning, suffocation, and all other, using the external cause-of-injury mortality matrix.[†] Motor vehicle traffic-related deaths were divided further into occupant, pedestrian, pedal cyclist, unspecified, and all other motor vehicle traffic–related deaths. Race/ethnicity was coded into five mutually exclusive categories: Hispanic (of any race), and four non-Hispanic racial groups (white, black, American Indian/Alaska Native, and Asian/Pacific Islander). Annual death rates were calculated using population totals from the U.S. Census.[§] Weighted least squares regression was used to test for linear trends in death rates over time, using all years of data. A p value of <0.05 indicated statistical significance.

Results

From 2000 to 2009, the overall annual unintentional injury death rate declined 29% among persons aged 0–19 years, from 15.5 to 11.0 per 100,000 (Table 1). The rate for males was higher than that for females in each age group. Among both males and females, the death rate declined 29%: from 19.9 to 14.1 per 100,000 for males and from 10.8 to 7.7 per 100,000 for females. A significant linear decline across all racial/ethnic groups was observed, with declines ranging from 21% among blacks to 38% among Asian/Pacific Islanders (Table 1). American Indian/Alaska Natives had the highest death rate throughout the study period, at 30.4 per 100,000 in 2000 and 23.8 in 2009, nearly double that of blacks (16.2 and 12.8), the population with the next highest rates in 2009.

By injury mechanism, motor vehicle traffic–related death rates decreased 41%, from 9.3 to 5.5 per 100,000, yet that category recorded the most deaths in 2000 (7,497) and 2009 (4,564) to remain the leading cause of unintentional injury death among persons aged 0–19 years. Drowning, other transportation, fire/burn, fall, and all other unintentional injuries also showed significant linear declines, whereas both suffocation and poisoning showed significant linear increases (30% and 80%, respectively) (Table 1).

Death rates varied substantially by age group and mechanism, with the highest rates in the youngest (aged <1 year) and oldest (15–19 years) age groups (Figure). The overall rate decreased among all age groups except children aged <1 year, whose death rate increased from 23.1 to 27.7 per 100,000,

surpassing rates among persons aged 15–19 years (Table 2). This increase can be attributed largely to a rise in suffocation death rates,[¶] which increased from 13.8 to 21.3 per 100,000, claiming the lives of 907 newborns and infants in 2009. The death rate for those aged 15–19 years declined 33%, from 33.4 to 22.3 per 100,000, most notably as a result of a 41% decline in motor vehicle traffic–related death rates from 25.3 to 15.1 per 100,000. However, poisoning death rates in the 15–19 year age group increased by 91%, from 1.7 to 3.3 per 100,000 over the same period (Table 2).

Wide variations in death rates were found among states, with 2009 rates ranging from 4.0 per 100,000 in Massachusetts and 4.5 in New Jersey to 23.6 per 100,000 in South Dakota and 25.1 in Mississippi (Table 3). In 11 states, death rates were significantly lower than the overall national rate of 11.0 per 100,000, and 21 states had rates that were significantly higher than 11.0. No states had significant linear increases in child and adolescent unintentional injury death rates; however, 31 states showed significant linear decreases. The largest decreases occurred in Delaware, Oregon, Iowa, and Virginia, where rates declined by at least 45%.

Conclusions and Comments

This report is the first from CDC to describe trends over time in child and adolescent unintentional injury deaths by mechanism and state. Population subgroup results generally are consistent with previous research. For example, males had higher death rates than females in each age group, racial/ethnic differences were observed with the highest rates among American Indians/Alaska Natives, and motor vehicle traffic–related injuries were the leading cause of unintentional injury death among the three oldest age groups (7,8). The wide variations in death rates among states suggest that environment, exposure to hazards (e.g., vehicle miles traveled, exposure to water settings, urban or rural environment), and differences in public policy might play a role. In 2009, if the overall national rate had been equal to the lowest state unintentional injury death rate, 5,785 lives would have been saved.

The high incidence of infant suffocation underscores the importance of a safe sleeping environment as recommended by the American Academy of Pediatrics, which includes supine positioning, a firm sleep surface, room-sharing without bed-sharing, and avoiding loose bedding (9). CDC has developed the Sudden Unexpected Infant Death (SUID) Case Registry, aimed at better understanding and ultimately preventing SUID deaths, which include suffocation in bed.**

[†] Additional information available at http://www.cdc.gov/nchs/injury/injury_tools.htm.

[§] Additional information available at http://www.cdc.gov/nchs/nvss/bridged_race.htm.

[¶] “Suffocation” refers to ICD-10 codes W75–W84. In 2009 73% of infant and newborn suffocation deaths were coded W75: accidental suffocation or strangulation in bed.

** Additional information available at <http://www.cdc.gov/sids/suidabout.htm>.

TABLE 1. Number of unintentional injury deaths and annual death rates* among persons aged ≤19 years, by sex, race/ethnicity, and mechanism — National Vital Statistics System, United States, 2000–2009

Sex, Race/Ethnicity, and Mechanism	No. of deaths		Death rate										% change from 2000 to 2009 [†]	p value [§]
	2000	2009	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009		
United States overall	12,441	9,143	15.5	15.0	15.3	14.8	14.9	14.4	14.2	14.0	12.2	11.0	-29	<0.001
Sex														
Male	8,217	6,016	19.9	19.5	19.9	19.1	19.0	18.4	18.2	17.9	15.9	14.1	-29	<0.001
Female	4,224	3,127	10.8	10.3	10.5	10.3	10.6	10.1	9.9	9.8	8.4	7.7	-29	0.002
Race/Ethnicity														
American Indian/Alaska Native	265	200	30.4	30.5	28.6	30.1	26.4	28.1	28.4	27.3	24.2	23.8	-22	<0.001
Black	2,004	1,615	16.2	16.0	16.2	14.3	15.0	15.0	15.2	15.2	13.4	12.8	-21	0.003
White	8,183	5,467	16.3	15.8	16.1	15.7	16.2	15.2	14.8	15.1	13.4	11.5	-29	0.002
Hispanic [¶]	1,691	1,625	12.4	12.1	12.5	12.5	11.6	12.2	12.1	10.6	9.1	8.8	-28	0.002
Asian/Pacific Islander	252	194	7.8	8.0	8.6	8.0	7.4	6.8	7.1	6.9	5.3	4.8	-38	<0.001
Missing data	46	42	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Mechanism**														
Motor vehicle traffic ^{††}	7,497	4,564	9.3	9.1	9.4	9.0	8.9	8.3	8.1	7.6	6.1	5.5	-41	<0.001
Occupant	3,571	1,953	4.4	4.5	5.1	4.7	4.6	4.2	3.9	3.5	2.5	2.3	-47	<0.001
Unspecified	2,794	1,866	3.5	3.2	3.0	2.9	3.0	2.8	2.9	2.9	2.5	2.2	-36	<0.001
Pedestrian	767	504	1.0	1.0	0.9	0.9	0.8	0.8	0.8	0.8	0.7	0.6	-37	<0.001
Other	185	152	0.2	0.2	0.2	0.3	0.3	0.3	0.3	0.3	0.3	0.2	-21	0.999
Pedal cyclist	180	89	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.1	0.1	0.1	-52	<0.001
Suffocation	864	1,160	1.1	1.1	1.2	1.1	1.3	1.3	1.4	1.5	1.6	1.4	30	<0.001
Drowning	1,314	983	1.6	1.5	1.4	1.3	1.3	1.4	1.3	1.3	1.2	1.2	-28	<0.001
Poisoning	442	824	0.5	0.6	0.7	0.8	0.9	0.9	1.0	1.2	1.1	1.0	80	<0.001
Other transportation	743	541	0.9	0.8	0.7	0.8	0.8	0.8	0.8	0.7	0.7	0.6	-30	0.010
Fire/Burn	682	391	0.8	0.8	0.7	0.7	0.7	0.6	0.6	0.7	0.5	0.5	-45	<0.001
Fall	180	151	0.2	0.3	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	-19	0.018
All other	719	529	0.9	0.9	0.9	0.8	0.8	0.9	0.8	0.8	0.7	0.6	-29	0.001

Abbreviation: NA = not applicable.

* Per 100,000 population.

[†] Percentage change might not match calculations because of rounding.

[§] P value from weighted least squares regression to assess linear trend significance during 2000–2009.

[¶] Hispanics, who might be of any race, were not included in any of the racial categories.

** Underlying cause of death mechanism classified by the *International Classification of Diseases, 10th Revision* (ICD-10) external cause of injury codes. Motor vehicle traffic: Occupant ([V30–V79](.4–.9), [V83–V86](.0–.3)), Unspecified, (V87(.0–.8), V89.2), Pedestrian ([V02–V04](.1, .9), V09.2), Other (including motorcyclist) ([V20–V28](.3–.9), V29(.4–.9), V80(.3–.5), V81.1, V82.1), and Pedal cyclist ([V12–V14](.3–.9), V19(.4–.6)). Suffocation (W75–W84); Drowning (W65–W74); Poisoning (X40–X49); Other transportation (V01, [V02–V04](.0), V05, V06, V09(.0–.1, .3, .9), V10–V11, [V12–V14](.0–.2), V15–V18, V19(.0–.3, .8, .9), [V20–V28](.0–.2), [V29–V79](.0–.3), V80(.0–.2, .6–.9), [V81–V82](.0, .2–.9), [V83–V86](.4–.9), V87.9, V88(.0–.9), V89(.0, .1, .3, .9), V90–V99)); Fire/Burn (X00–X19); Fall (W00–W19). All other (mechanisms aggregated in table): cut or pierced (W25–W29, W45, W46), unintentional firearm (W32–W34), machinery (W24, W30–W31), natural and environmental (W42–W43, W53–W64, W92–W99, X20–X39, X51–X57), overexertion (X50), struck by or against (W20–W22, W50–W52), other specified (W23, W35–W41, W44, W49, W85–W91, Y85, X58, Y86), and unspecified (X59).

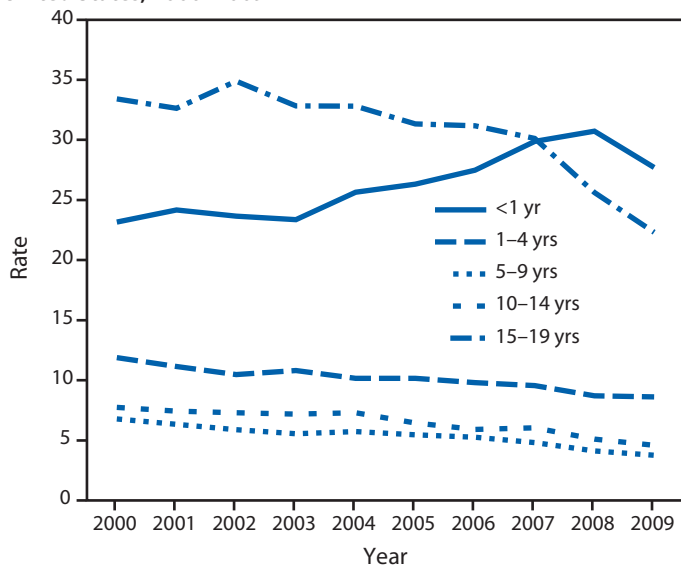
^{††} Categorized by injured person.

The increasing trend in poisoning deaths among those aged 15–19 years is consistent with the reported increases in drug poisoning deaths seen in the U.S. population overall during this period (10). Poisoning deaths from prescription drug misuse is a growing concern (11); during 2002–2004 an estimated 13.5% of those aged 12–17 years reported ever having misused prescription drugs (12). The percentage of poisoning deaths among those aged 15–19 years with prescription drugs as a contributing cause increased from 30% in 2000 to 57% in 2009 (13). Strategies to reduce the misuse of prescription drugs include appropriate prescribing, proper storage and disposal, discouraging medication sharing, and state-based prescription drug monitoring programs (11).

Improvements in seat belt use, child safety seat and booster seat use, licensing requirements, vehicle design, the road

environment, and reductions in alcohol-impaired driving likely contributed to the decline in motor vehicle traffic-related deaths (4,14). Despite this success, traffic crashes remain the leading cause of death for persons in age groups 5–19 years, accounting for 67% of unintentional injury deaths and 28% of deaths from all causes among those aged 15–19 years in 2009 (1). Increasing seat belt use and implementing components of graduated driver licensing practices, such as limiting nighttime driving and limiting teen passengers, likely can lead to further declines. Parents can learn how to reduce their teens' risks of motor vehicle-related injury through the CDC's teen driving initiative, Parents are the Key (<http://www.cdc.gov/parentsarethekey/index.html>). For communities, the Guide to Community Preventive Services provides evidenced-based strategies to reduce motor vehicle traffic-related injuries (e.g.,

FIGURE. Annual unintentional injury death rates* among persons aged ≤19 years, by age group — National Vital Statistics System, United States, 2000–2009



* Per 100,000 population.

laws mandating child safety seat use and primary enforcement of seat belt use, and multiple measures to reduce alcohol-impaired driving) (<http://www.thecommunityguide.org/mvoi/index.html>).

Even with the reported declines, the U.S. unintentional injury death rate among persons aged 0–19 years does not compare favorably with other developed countries. Among the 34 Organization for Economic Cooperation and Development countries, the U.S. unintentional injury death rate for persons aged 0–14 years ranked 30th in 2008, with a rate four times higher than the top performing nations (15). Among persons aged 0–19 years, unintentional injury death rates in 2004 in the United States were almost twice the combined rates of high-income countries in the World Health Organization's European and Western Pacific Regions (4).

The findings in this report are subject to at least two limitations. First, fatalities are based on death certificate data and are subject to misclassification errors if a mechanism is not specified correctly on the death certificate or if classification standards have changed over time. For example, challenges in distinguishing between sudden infant death syndrome and suffocation combined with reporting differences among those completing death certificates might be contributing factors in the increase in reported suffocation deaths among newborns and infants (9). Second, this report is limited to unintentional injury deaths; excluding nonfatal injuries substantially under-reports the total burden from injury on society and the medical care system (1).

Key Points

- Unintentional injuries are the leading cause of death among persons aged 1–19 years and the fifth leading cause of death for newborns and infants aged <1 year. Nearly two in five deaths among persons aged 1–19 years are caused by unintentional injuries.
- From 2000 to 2009, the overall unintentional injury death rate among persons aged 0–19 years decreased 29%, from 15.5 to 11.0 per 100,000. Motor vehicle traffic-related deaths declined, but remain the leading cause of injury deaths.
- Wide variations in death rates were found among states with the rate for Mississippi more than six times the rate for Massachusetts.
- Unintentional infant suffocation death rates increased 54% during 2000–2009, driving the overall increase in newborn and infant unintentional injury death rates.
- Poisoning death rates increased 91% among persons aged 15–19 years.
- Unintentional injury deaths are preventable, and efforts to increase child and adolescent safety through evidence-based prevention initiatives can reduce death rates even further.
- The National Action Plan for Child Injury Prevention provides a framework to address child unintentional injury prevention with specific actions in surveillance, research, communication, education, health care, and public policy (<http://www.cdc.gov/safechild/nap>). Taking steps to implement the National Action Plan could result in substantial reductions in needless deaths, injuries, and costs associated with injuries among children and adolescents in the United States.

The frequency and cost of child and adolescent unintentional injury deaths, along with the effectiveness of existing public health interventions, make injury prevention a priority for improving the health of children and adolescents. Efforts to prevent these deaths likely will result in fewer nonfatal injuries as well. Although unintentional injury death rates are declining, findings reported here demonstrate the need to take further action. CDC has developed the Protect the Ones You Love Initiative to help parents reduce unintentional injuries from burns, drowning, falls, poisonings, motor vehicle crashes, suffocation, and sports (<http://www.cdc.gov/safechild>). Health-care providers, educators, community members and others also can take steps to reduce child injury. CDC and its partners have released the National Action Plan for Child Injury Prevention,

TABLE 2. Number of unintentional injury deaths and annual death rates* among persons aged ≤19 years, by age group and mechanism — National Vital Statistics System, United States, 2000–2009

Age group/Mechanism	No. of deaths			Death rate										% change from 2000 to 2009 [†]	p value [§]
	2000	2009	2009 (%)	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009		
Total 0–19 yrs	12,441	9,143	(100.0)	15.5	15.0	15.3	14.8	14.9	14.4	14.2	14.0	12.2	11.0	-29	<0.001
<1 yr															
Total	881	1,181	(100.0)	23.1	24.2	23.7	23.4	25.6	26.3	27.5	29.9	30.7	27.7	20	<0.001
Suffocation	526	907	(76.8)	13.8	15.2	15.9	15.3	17.7	18.2	20.2	22.3	24.7	21.3	54	<0.001
Motor vehicle traffic	162	91	(7.7)	4.3	3.4	3.0	3.6	3.4	3.4	3.3	2.8	2.3	2.1	-50	0.002
Drowning	75	45	(3.8)	2.0	1.7	1.6	1.4	1.5	1.6	1.2	1.3	1.0	1.1	-46	<0.001
Fire/Burn	39	25	(2.1)	1.0	1.2	1.0	0.8	0.7	0.9	0.7	0.9	0.5	0.6	-43	0.006
Poisoning	14	22	(1.9)	— [¶]	—	0.7	0.5	—	0.5	—	—	—	0.5	NA	NA
Fall	8	19	(1.6)	—	0.6	—	—	0.6	—	0.6	0.6	—	—	NA	NA
Other transportation	12	6	(0.5)	—	—	—	—	—	—	—	—	—	—	NA	NA
All other	45	66	(5.6)	1.2	1.4	1.0	1.3	1.4	1.3	1.1	1.4	1.5	1.5	31	0.120
1–4 yrs															
Total	1,826	1,466	(100.0)	11.9	11.1	10.5	10.8	10.2	10.2	9.8	9.6	8.7	8.6	-28	<0.001
Drowning	493	450	(30.7)	3.2	3.0	2.9	2.9	2.7	3.0	2.8	2.8	2.6	2.6	-18	0.006
Motor vehicle traffic	563	362	(24.7)	3.7	3.6	3.4	3.2	3.2	3.0	2.9	2.6	2.1	2.1	-42	<0.001
Fire/Burn	297	169	(11.5)	1.9	1.5	1.4	1.4	1.4	1.3	1.2	1.2	1.0	1.0	-49	<0.001
Other transportation	127	147	(10.0)	0.8	0.7	0.6	0.9	0.8	1.0	0.8	0.9	0.8	0.9	4	0.161
Suffocation	151	125	(8.5)	1.0	0.9	0.9	1.0	0.8	0.8	0.8	0.9	0.9	0.7	-25	0.061
Fall	36	46	(3.1)	0.2	0.2	0.2	0.3	0.3	0.2	0.2	0.2	0.2	0.3	15	0.995
Poisoning	32	37	(2.5)	0.2	0.2	0.2	0.3	—	0.1	0.2	0.2	0.2	0.2	4	0.937
All other	127	130	(8.9)	0.8	1.0	0.8	0.8	0.8	0.8	0.8	0.8	0.9	0.8	-8	0.512
5–9 yrs															
Total	1,391	773	(100.0)	6.8	6.3	5.9	5.5	5.7	5.5	5.3	4.8	4.1	3.8	-45	<0.001
Motor vehicle traffic	731	378	(48.9)	3.6	3.3	3.1	3.0	3.0	2.9	2.6	2.3	1.9	1.8	-48	<0.001
Drowning	201	119	(15.4)	1.0	0.8	0.8	0.6	0.7	0.6	0.7	0.6	0.7	0.6	-41	0.008
Fire/Burn	183	88	(11.4)	0.9	0.8	0.8	0.7	0.9	0.7	0.6	0.7	0.5	0.4	-52	<0.001
Other transportation	106	68	(8.8)	0.5	0.5	0.4	0.5	0.5	0.4	0.5	0.4	0.3	0.3	-36	0.023
Suffocation	45	26	(3.4)	0.2	0.2	0.2	0.2	0.2	0.2	0.3	0.2	0.2	0.1	-42	0.124
Poisoning	17	13	(1.7)	—	—	—	—	—	—	—	—	—	—	NA	NA
Fall	16	12	(1.6)	—	0.2	—	—	—	—	—	—	0.1	—	NA	NA
All other	92	69	(8.9)	0.4	0.4	0.5	0.4	0.4	0.5	0.4	0.5	0.3	0.3	-25	0.070
10–14 yrs															
Total	1,588	916	(100.0)	7.7	7.4	7.3	7.2	7.3	6.4	5.9	6.0	5.1	4.6	-41	<0.001
Motor vehicle traffic	916	491	(53.6)	4.5	4.2	4.1	4.3	4.4	3.7	3.4	3.4	2.6	2.5	-45	<0.001
Other transportation	161	117	(12.8)	0.8	0.8	0.7	0.7	0.7	0.6	0.6	0.6	0.5	0.6	-25	<0.001
Drowning	174	90	(9.8)	0.8	0.8	0.8	0.7	0.7	0.6	0.6	0.5	0.6	0.5	-47	<0.001
Fire/Burn	84	53	(5.8)	0.4	0.4	0.5	0.4	0.4	0.4	0.3	0.4	0.3	0.3	-35	0.006
Suffocation	72	41	(4.5)	0.4	0.3	0.3	0.2	0.3	0.3	0.3	0.3	0.2	0.2	-41	0.062
Poisoning	28	37	(4.0)	0.1	0.2	0.1	0.2	0.2	0.2	0.2	0.3	0.2	0.2	36	0.116
Fall	21	16	(1.7)	0.1	0.2	0.1	0.1	0.1	—	0.1	0.1	—	—	NA	NA
All other	132	71	(7.8)	0.6	0.6	0.6	0.6	0.5	0.6	0.4	0.4	0.5	0.4	-45	0.002
15–19 yrs															
Total	6,755	4,807	(100.0)	33.4	32.6	34.9	32.8	32.8	31.3	31.2	30.1	25.6	22.3	-33	0.001
Motor vehicle traffic	5,125	3,242	(67.4)	25.3	25.1	27.0	25.1	24.6	22.9	22.5	21.3	17.2	15.1	-41	<0.001
Poisoning	351	715	(14.9)	1.7	2.0	2.4	2.5	3.1	3.0	3.5	3.9	3.9	3.3	91	<0.001
Drowning	371	279	(5.8)	1.8	1.6	1.6	1.4	1.5	1.5	1.5	1.5	1.3	1.3	-29	0.006
Other transportation	337	203	(4.2)	1.7	1.3	1.2	1.4	1.3	1.3	1.2	1.2	1.3	0.9	-43	0.019
Suffocation	70	61	(1.3)	0.3	0.3	0.3	0.2	0.3	0.3	0.4	0.2	0.3	0.3	-18	0.160
Fall	99	58	(1.2)	0.5	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.3	-45	0.001
Fire/Burn	79	56	(1.2)	0.4	0.4	0.4	0.4	0.3	0.3	0.4	0.4	0.3	0.3	-33	0.040
All other	323	193	(4.0)	1.6	1.6	1.6	1.4	1.4	1.6	1.4	1.2	1.0	0.9	-44	0.001

Abbreviation: NA = not applicable.

* Per 100,000 population.

† Percentage change might not match calculations because of rounding.

§ P value from weighted least squares regression to assess linear trend significance 2000–2009.

¶ Death rates based on fewer than 20 deaths suppressed for unreliability.

TABLE 3. Number of unintentional injury deaths and annual death rates* among persons aged ≤19 years, by state — National Vital Statistics System, United States, 2000–2009

State	No. of deaths		Death rate										% change from 2000 to 2009 [†]	p value [§]
	2000	2009	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009		
United States overall [¶]	12,441	9,143	15.5	15.0	15.3	14.8	14.9	14.4	14.2	14.0	12.2	11.0	-29	<0.001
States with death rates significantly higher than overall U.S. rate of 11.0 in 2009**														
Mississippi	268	216	30.7	28.9	29.8	25.9	27.4	29.7	26.8	30.7	22.2	25.1	-18	0.061
South Dakota	68	53	29.9	24.3	26.8	25.6	28.8	28.4	22.6	17.0	18.4	23.6	-21	0.030
Montana	81	51	31.5	17.2	25.3	26.3	27.6	17.7	24.6	19.7	20.9	20.5	-35	0.346
Wyoming	38	30	26.1	21.8	28.3	26.5	21.6	23.2	28.0	21.8	24.2	20.2	-23	0.236
Louisiana	304	253	22.2	24.1	25.6	21.5	25.9	24.0	21.1	21.0	23.4	20.1	-9	0.184
Oklahoma	229	200	22.8	23.3	18.5	19.0	24.2	22.4	23.1	22.1	20.0	19.5	-15	0.582
Alaska	69	39	33.2	28.4	19.7	25.9	25.0	25.7	24.3	24.1	23.3	19.0	-43	0.072
South Carolina	252	219	22.2	24.2	23.9	18.4	21.3	21.8	21.2	22.7	19.1	18.0	-19	0.071
New Mexico	118	100	20.9	18.1	21.1	22.5	20.3	20.8	18.6	18.7	14.7	17.6	-16	0.048
Arkansas	201	139	26.4	28.4	24.9	23.8	27.4	26.6	24.1	23.4	22.0	17.6	-34	0.006
Alabama	301	220	24.0	25.3	25.4	21.9	23.9	23.1	24.8	22.0	19.0	17.4	-27	0.006
North Dakota	24	29	13.1	15.5	17.0	21.3	17.4	18.8	21.4	16.7	13.2	17.3	32	0.635
Kentucky	267	190	24.0	22.2	21.9	21.4	25.5	23.0	20.1	18.3	16.3	16.8	-30	0.004
Missouri	372	266	23.3	19.3	21.8	20.9	21.1	19.6	20.1	21.1	19.6	16.6	-29	0.031
Kansas	168	119	21.0	22.5	19.9	19.7	16.3	17.0	16.0	16.1	14.0	15.1	-28	<0.001
Nevada	96	104	17.1	13.7	16.5	18.2	16.6	14.2	16.8	15.2	15.3	14.0	-18	0.317
Florida	744	621	18.4	17.6	17.4	19.1	18.9	20.3	18.5	18.6	15.2	13.7	-25	0.086
Tennessee	362	227	23.2	20.9	22.8	19.9	22.4	19.4	21.7	21.4	16.7	13.7	-41	0.009
North Carolina	376	328	17.1	17.9	18.1	18.5	19.9	17.2	16.4	16.6	15.4	12.8	-25	0.014
Indiana	324	228	18.4	16.9	16.5	15.6	18.0	16.5	17.1	16.7	16.6	12.8	-30	0.059
Texas	1,198	945	18.3	17.3	17.2	16.5	15.5	14.7	15.4	13.9	12.9	12.4	-32	<0.001
States with death rates not significantly different from overall U.S. rate of 11.0 in 2009														
Idaho	78	65	18.8	23.6	19.3	17.7	18.2	17.2	20.2	18.9	15.3	13.9	-26	0.021
West Virginia	98	54	21.6	19.3	23.5	22.7	23.5	17.8	20.9	20.2	20.8	12.4	-43	0.055
Michigan	465	319	16.1	14.3	15.7	14.8	14.2	13.3	11.8	13.6	11.0	12.0	-25	<0.001
Delaware	53	28	24.3	15.5	14.5	12.1	17.4	11.5	14.4	10.8	10.3	12.0	-51	0.027
Nebraska	83	60	16.5	14.9	19.3	17.1	16.5	15.6	18.4	16.4	14.5	11.8	-28	0.104
Wisconsin	245	175	16.0	13.0	15.1	15.9	13.5	15.5	13.9	15.7	13.2	11.8	-26	0.160
Maine	50	36	14.9	16.1	15.0	12.7	15.2	15.7	15.6	12.9	12.5	11.7	-22	0.042
Arizona	279	213	18.4	20.6	17.3	17.8	18.3	17.0	17.9	16.4	12.2	11.2	-39	0.002
Utah	113	106	13.9	13.9	15.6	13.6	11.6	13.2	12.3	14.0	10.8	11.0	-21	0.022
Hawaii	33	35	10.1	10.2	14.3	13.9	9.8	8.7	11.5	10.3	8.4	10.8	8	0.390
Georgia	446	297	18.5	19.3	17.6	17.7	16.5	16.5	15.1	15.2	13.6	10.3	-44	<0.001
Iowa	155	82	18.7	13.8	14.1	16.3	13.5	14.2	11.3	15.2	12.8	10.2	-46	0.019
Pennsylvania	435	319	13.3	13.6	13.8	14.3	13.3	13.0	12.1	13.8	11.8	10.1	-24	0.015
Washington	249	174	14.8	13.6	13.8	12.0	11.9	11.2	13.3	10.5	9.7	10.0	-33	<0.001
Colorado	167	136	13.6	15.6	15.6	15.5	15.0	11.7	12.1	10.9	11.4	10.0	-27	0.002
New Hampshire	39	29	11.3	13.8	6.3	9.2	13.0	11.0	9.1	9.2	9.9	8.8	-22	0.663
Rhode Island	27	20	9.6	8.5	8.5	11.3	9.9	7.6	8.1	8.5	— ^{§§}	7.6	-21	0.079
Vermont ^{††}	28	17	16.8	—	13.4	12.3	—	22.9	—	—	16.7	—	NA	NA
States with death rates significantly lower than overall U.S. rate of 11.0 in 2009														
Ohio	450	278	14.0	13.3	13.9	13.1	15.1	13.9	12.9	12.5	12.3	9.1	-35	0.018
Minnesota	205	122	14.3	13.2	15.7	14.1	12.9	10.7	11.9	10.5	9.5	8.6	-40	<0.001
Oregon	154	84	16.3	14.3	14.6	17.0	16.7	13.1	13.7	11.6	14.2	8.6	-47	0.010
Virginia	280	164	14.5	12.0	13.7	12.9	13.3	12.6	12.6	12.3	11.7	7.9	-45	0.013
Illinois	476	280	13.2	12.6	13.2	11.6	10.6	11.1	11.7	12.3	9.5	7.9	-40	0.004
Maryland	147	117	9.8	12.2	10.8	11.5	11.2	9.2	10.1	10.4	8.7	7.7	-22	0.020
California	1,038	785	10.1	9.7	10.9	10.9	10.3	11.0	10.6	9.7	7.6	7.5	-26	0.026
New York	399	334	7.7	9.0	9.0	7.6	7.8	7.3	7.5	7.7	6.6	6.7	-13	0.011
Connecticut	77	59	8.3	9.3	9.5	7.9	9.8	8.3	8.4	9.2	7.6	6.4	-22	0.076
New Jersey	180	102	7.9	9.2	9.1	7.4	8.1	7.5	7.7	7.9	7.2	4.5	-43	0.013
Massachusetts	120	66	7.2	7.8	6.6	7.4	7.0	6.9	6.7	7.6	5.6	4.0	-44	0.017

Abbreviation: NA = not applicable.

* Per 100,000 population.

† Percentage change might not match calculations because of rounding.

§ P value from weighted least squares regression to assess linear trend significance during 2000–2009.

¶ The District of Columbia was excluded because of low death counts in all years; however District of Columbia deaths are included in the overall U.S. death rate.

** Significance determined using Z-test for states with ≥100 deaths and 95% confidence intervals from a gamma distribution for states with <100 deaths in 2009.

†† With 17 deaths, rate for Vermont in 2009 was unstable and thus cannot be statistically compared with the overall U.S. death rate.

§§ Death rates based on fewer than 20 deaths have been suppressed for unreliability.

providing actions in surveillance, research, communication, education, health care, and public policy (<http://www.cdc.gov/safechild/nap>). Implementing the National Action Plan could result in significant reductions in needless deaths, injuries, and costs associated with injuries among children and adolescents in the United States.

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References

1. CDC. Web-based Injury Statistics Query and Reporting System (WISQARS). Atlanta, GA: US Department of Health and Human Services, CDC; 2009. Available at <http://www.cdc.gov/injury/wisqars/index.html>. Accessed March 13, 2012.
2. National Center for Health Statistics. National Health Interview Survey 2009 data release. Hyattsville, MD: CDC, National Center for Health Statistics; 2010. Available at http://www.cdc.gov/nchs/nhis/nhis_2009_data_release.htm. Accessed March 13, 2012.
3. Doll LS, Bonzo SE, Mercy JA, Sleet DA, eds. Handbook of injury and violence prevention. New York, NY: Springer; 2007.
4. Peden M, Oyegbite K, Ozanne-Smith J, Hyder AA, Branche C, Rahman AKM, et al., eds. World report on child injury prevention. Geneva, Switzerland: World Health Organization; 2008. Available at http://www.who.int/violence_injury_prevention/child/injury/world_report/report/en/index.html. Accessed March 13, 2012.
5. Miller TR, Finkelstein AE, Zaloshnja E, Hendrie D. The cost of child and adolescent injuries and the savings from prevention. In: Liller KD, ed. Injury prevention for children and adolescents: research, practice, and advocacy. 2nd ed. Washington, DC: American Public Health Association 2012.
6. Singh G, Kogan M. Widening socioeconomic disparities in U.S. childhood mortality, 1969–2000. *Am J Public Health* 2007;97:1658–65.
7. Borse N, Gilchrist J, Dellinger A, Rudd R, Ballesteros M, Sleet D. CDC childhood injury report: patterns of unintentional injuries among 0–19 year olds in the United States, 2000–2006. Atlanta, GA: US Department of Health and Human Services, CDC; 2008. Available at <http://www.cdc.gov/safechild/childhoodinjuryreport/index.html>. Accessed March 13, 2012.
8. Ballesteros MF, Sleet DA. Epidemiology of injuries among children and adolescents: focus on unintentional injuries. In: Liller KD, ed. Injury prevention for children and adolescents: research, practice, and advocacy. 2nd ed. Washington, DC: American Public Health Association 2012.
9. Task force on Sudden Infant Death Syndrome. Moon RY. SIDS and other sleep-related infant deaths: expansion of recommendations for a safe infant sleeping environment. *Pediatrics* 2011;128:1030–9.
10. Warner M, Chen LH, Makuc DM, Anderson RN, Miniño AM. Drug poisoning deaths in the United States, 1980–2008. NCHS data brief no. 81. Hyattsville, MD: CDC, National Center for Health Statistics; 2011. Available at <http://www.cdc.gov/nchs/data/databriefs/db81.htm>. Accessed March 13, 2012.
11. CDC. Vital signs: overdoses of prescription opioid pain relievers — United States, 1999–2008. *MMWR* 2011;60:1487–92.
12. Colliver JD, Kroutil LA, Dai L, Gfroerer JC. Office of Applied Studies. Misuse of prescription drugs: Data from: 2002–2004 national surveys on drug use and health. Table 5. Rockville, MD: Substance Abuse and Mental Health Services Administration, Office of Applied Studies; 2006. Available at <http://www.oas.samhsa.gov/prescription/appd.htm#tab5-2b>. Accessed March 13, 2012.
13. CDC. WONDER [Database]. Atlanta, GA: US Department of Health and Human Services, CDC; 2011. Available at <http://wonder.cdc.gov/mcd-icd10.html>. Accessed April 9, 2012.
14. Longthorne A, Subramanian R, Chen CL. An analysis of the significant decline in motor vehicle traffic crashes in 2008. DOT HS 811 346. Washington, DC: National Highway Traffic Safety Administration; 2010. Available at <http://www-nrd.nhtsa.dot.gov/pubs/811346.pdf>. Accessed March 13, 2012.
15. Global Health Observatory Data Repository. Mortality and burden of disease: disease and injury country estimates, 2008, by sex and age. Available at <http://apps.who.int/ghodata>. Accessed February 16, 2010.

Notes from the Field

Infections with *Salmonella* I 4,[5],12:i:- Linked to Exposure to Feeder Rodents — United States, August 2011–February 2012

CDC is collaborating with 22 state health departments in an ongoing investigation of an outbreak of human *Salmonella* I 4,[5],12:i:- infections associated with exposure to rodents sold as food for pet reptiles and amphibians (i.e., feeder rodents). This outbreak strain also was implicated in a 2009 outbreak in the United Kingdom and a 2010 outbreak in the United States, both linked to frozen feeder rodents from a single U.S. supplier, resulting in recalls (1,2).

During August 29, 2011–February 2, 2012, a total of 46 cases of human *Salmonella* I 4,[5],12:i:- infection were reported in 22 states. The median patient age was 11 years (range: <1–69 years); 37% were aged ≤5 years, and 52% were male. Of the 27 patients interviewed, six (22%) reported hospitalization, 20 (74%) reported reptile or amphibian exposures, and 15 (56%) reported feeder rodent exposure. For 12 patients who recalled the types of rodent contacted, five (42%) reported exposure to live rodents, four (33%) to frozen rodents, and three (25%) to both live and frozen rodents. Seven (58%) patients reported exposure to mice, two (17%) to rats, two (17%) to both mice and rats, and one (8%) was unsure. No patients reported exposure to rodents purchased from the same pet store.

Frozen mice specimens from two North Carolina pet stores where two patients purchased feeder rodents yielded the outbreak strain. Tracing the source of these mice has been difficult for investigators because of complex breeder and supplier networks and inadequate records. However, two breeders supplying pet stores from which patients had purchased rodents had received mice from the company implicated in the 2009

and 2010 outbreaks. Given the wide distribution of illnesses, rodent suppliers, and pet stores, the outbreak strain might now be endemic in feeder rodents.

The fact that 37% of patients were aged ≤5 years supports recommendations that young children avoid exposure to reptiles or amphibians, including in the home (3). Owners of reptiles, amphibians, or other animals that are fed rodents should be aware of the risk for salmonellosis from the animals and live and frozen feeder rodents. Safe handling instructions for all of these animals should be provided at the point of sale.

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References

1. Harker K, Lane C, De Pinna E, Adak G. An outbreak of *Salmonella* Typhimurium DT191a associated with reptile feeder mice. *Epidemiol Infect* 2011;139:1254–61.
2. CDC. Investigation announcement: multistate outbreak of human *Salmonella* I 4,[5],12:i:- infections associated with frozen rodents. Atlanta, GA: US Department of Health and Human Services, CDC; 2010. Available at <http://www.cdc.gov/salmonella/frozenrodents/index.html>. Accessed April 13, 2012.
3. CDC. Reptiles, amphibians, and *Salmonella*. Atlanta, GA: US Department of Health and Human Services, CDC; 2012. Available at <http://www.cdc.gov/features/salmonellafrogturtle>. Accessed April 16, 2012.

Announcements

World Malaria Day — April 25, 2012

World Malaria Day is commemorated on April 25, the date in 2000 when 44 African leaders met in Abuja, Nigeria, and committed their countries to cutting malaria-related deaths in half by 2010. In the decade since, increased funding and control efforts have led to a scale-up of effective malaria interventions, resulting in decreased malaria morbidity and mortality in many countries. In 2010, an estimated 216 million cases of malaria and 655,000 deaths were reported worldwide, a 17% decrease in malaria incidence and 25% reduction in global malaria mortality since 2000 (1). World Malaria Day 2012's theme, Sustain Gains, Save Lives: Invest in Malaria, underscores the need to consolidate these gains and continue scaling up malaria interventions.

CDC supports these efforts through the President's Malaria Initiative, a U.S. government interagency initiative to reduce malaria incidence and mortality in 19 countries in sub-Saharan Africa and in the Mekong subregion in Asia. In addition, CDC conducts multidisciplinary strategic and applied research globally to increase knowledge about malaria and develop safe, effective interventions that can lead to the elimination and eventual eradication of malaria.

As a World Health Organization (WHO) Collaborating Center for Prevention and Control of Malaria, CDC works closely with WHO, which has just released new malaria surveillance manuals and launched the T3: Test, Treat, and Track initiative, urging increased investment in national capacity for diagnostic testing, diagnosis-based treatment, and surveillance. Additional information is available from WHO at <http://www.who.int/malaria/en>. Additional information regarding CDC's malaria activities is available at <http://www.cdc.gov/malaria>.

Reference

1. World Health Organization. World malaria report 2011. Geneva, Switzerland: World Health Organization; 2011. Available at http://www.who.int/malaria/world_malaria_report_2011/en/index.html. Accessed April 10, 2012.

National Infant Immunization Week — April 21–28, 2012

The 18th annual National Infant Immunization Week (NIIW) will be observed April 21–28, 2012. Local and state health departments, national immunization partners, health-care

professionals, and community leaders across the United States will collaborate to highlight the achievements and benefits of immunization through community activities and events, including grand rounds and educational training for health-care professionals and parents, media briefings, and immunization clinics.

This year, NIIW will be observed simultaneously with World Immunization Week (WIW), the first ever global immunization observance, an initiative of the World Health Organization to promote immunization and advance equity in the use of vaccines and universal access to vaccination services. This year also includes the inaugural presentation of the CDC Childhood Immunization Champion Award, given jointly by CDC and the CDC Foundation to recognize persons for the important contributions they have made to public health through their work in childhood immunizations. In the United States, CDC currently recommends that children aged ≤ 2 years receive vaccines to protect against diseases caused by 14 different pathogens (1).

Immunization rates for vaccines routinely recommended for children remain at or near record highs (2). Nevertheless, recent cases of measles (3) and pertussis (4) in the United States highlight the importance of maintaining high immunization rates. High vaccination coverage in children requires parental acceptance of routine childhood immunization. A recent survey of U.S. parents with children aged < 6 years indicated that most parents are confident or very confident in the safety (72.2%), effectiveness (77.8%), and benefit of vaccines (79.6%) (5). To remind parents of the value of their children being vaccinated in accordance with the recommended immunization schedule, CDC is introducing print advertisements, posters, and radio and television public service announcements. Additional information about NIIW is available at <http://www.cdc.gov/vaccines/events/niiw>.

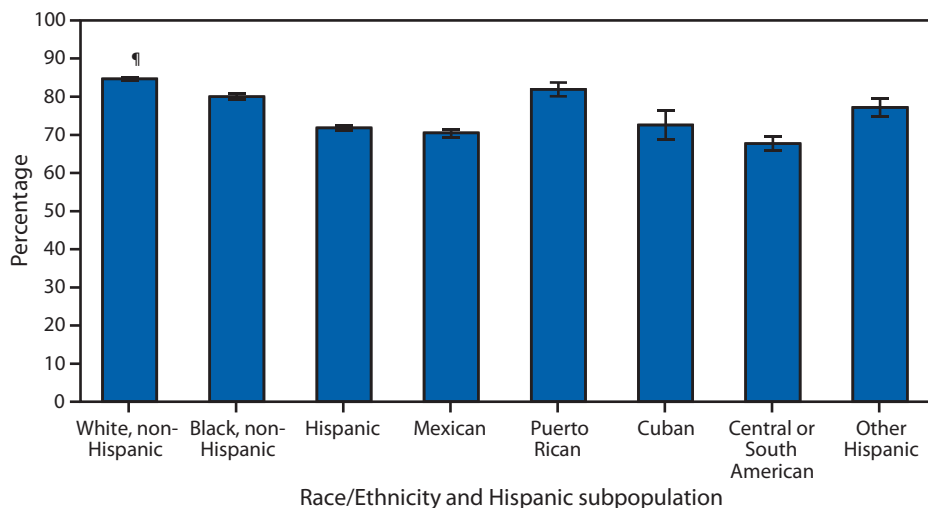
References

1. CDC. Recommended immunization schedules for persons aged 0 through 18 years—United States, 2012. MMWR 2012;61(5).
2. CDC. National, state, and local area vaccination coverage among children aged 19–35 months—United States, 2009. MMWR 2010;59:1171–7.
3. CDC. Measles—United States, 2011. MMWR 2012;61:253–7.
4. CDC. Notice to readers: final 2010 reports of nationally notifiable infectious diseases. MMWR 2011;60:1088–101.
5. Kennedy A, LaVail K, Basket M, Nowak G. Assessing the current state of immunization attitudes in the United States: results from the 2011 ConsumerStyles Survey [Poster]. Presented at the 1st Annual Immunization Conference Online, March 26–28, 2012. Available at <http://cdc.confex.com/cdc/nic2012/webprogram/Paper30098.html>.

QuickStats

FROM THE NATIONAL CENTER FOR HEALTH STATISTICS

Percentage of Adults Aged ≥ 18 Years Who Have a Usual Place for Health Care,* by Race and Hispanic Subpopulation[†] — National Health Interview Survey, United States, 2010[§]



* Based on "Yes" and "There is more than one place" responses to the following question: "Is there a place that you usually go to when you are sick or need advice about your health?" The usual place to go for health care does not include a hospital emergency department. Unknowns were not included in the denominators when calculating percentages.

[†] Persons of Hispanic ethnicity might be of any race or combination of races.

[§] Estimates are based on household interviews of a sample of the U.S. civilian, noninstitutionalized population. Estimates are age adjusted using the projected 2000 U.S. population as the standard population and using four age groups: 18–44 years, 45–64 years, 65–74 years, and ≥ 75 years.

[¶] 95% confidence interval.

Hispanic adults (71.8%) were less likely to have a usual place for health care than non-Hispanic white adults (84.7%) and non-Hispanic black adults (80.0%). Among the five Hispanic subpopulations, Puerto Rican adults (81.9%) were more likely to have a usual place for health care compared with Mexican adults (70.5%), Cuban adults (72.6%), and Central or South American adults (67.7%).

Source: National Health Interview Survey, 2010 data. Available at <http://www.cdc.gov/nchs/nhis.htm>.

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