Centers for Disease Control and Prevention
MMWR
Morbidity and Mortality Weekly Report
Weekly / Vol. 65 / No. 26
July 8, 2016

Human Papillomavirus–Associated Cancers — United States, 2008–2012

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Human papillomavirus (HPV) is a known cause of cervical cancers, as well as some vulvar, vaginal, penile, oropharyngeal, anal, and rectal cancers (*1*,*2*). Although most HPV infections are asymptomatic and clear spontaneously, persistent infections with one of 13 oncogenic HPV types can progress to precancer or cancer. To assess the incidence of HPV-associated cancers, CDC analyzed 2008–2012 high-quality data from the CDC's National Program of Cancer Registries and the National Cancer Institute's Surveillance, Epidemiology, and End Results program. During 2008–2012, an average of 38,793 HPV-associated cancers were diagnosed annually, including 23,000 (59%) among females and 15,793 (41%) among males. By multiplying these counts by the percentages attributable to HPV (*3*), CDC estimated that approximately 30,700 new cancers were attributable to HPV, including 19,200 among females and 11,600 among males. Cervical precancers can be detected through screening, and treatment can prevent progression to cancer; HPV vaccination can prevent infection with HPV types that cause cancer at cervical and other sites (*3*). Vaccines are available for HPV types 16 and 18, which cause 63% of all HPV-associated cancers in the United States, and for HPV types 31, 33, 45, 52, and 58, which cause an additional 10% (*3*). Among the oncogenic HPV types, HPV 16 is the most likely to both persist and to progress to cancer (*3*). The impact of these primary and secondary prevention interventions can be monitored using surveillance data from population-based cancer registries.

CDC analyzed data from population-based cancer registries that participate in the CDC's National Program of Cancer Registries and the National Cancer Institute's Surveillance, Epidemiology, and End Results program and met the criteria for high data quality for all years 2008–2012, covering approximately 99% of the U.S. population.* Cases were classified by anatomic site using the *International Classification of Diseases for Oncology, 3rd Edition*† and were confirmed histologically. HPV-associated cancers were defined as invasive cancers at anatomic sites (i.e., cervix, vulva, vagina, penis, oropharynx, anus, and rectum) with cell types in which HPV DNA frequently is found (all carcinomas of the cervix, including adenocarcinomas and squamous cell cancers [SCC]; SCCs only for the other anatomic sites). Oropharyngeal cancers included cancers of the base of tongue; pharyngeal tonsils, anterior and posterior tonsillar pillars, and glossotonsillar sulci; anterior surface of soft palate and uvula; and lateral and posterior pharyngeal walls.§ Age-adjusted incidence rates were calculated per 100,000 persons and standardized to the 2000 U.S. standard population. Rates were considered significantly different from the referent category at a p-value of <0.05.

* http://www.cdc.gov/uscs.

† http://codes.iarc.fr/.

§ American Joint Committee on Cancer (AJCC). AJCC Cancer Staging Manual. 7th ed. Chicago, IL: Springer; 2010.

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Cancer registries do not routinely collect information on HPV DNA presence in cancer tissues, and HPV-associated cancers defined by anatomic site and cell type include cancers not caused by HPV. Therefore, to calculate HPV-attributable cases, the number of HPV-associated cancers was multiplied by the percentage of each cancer type attributable to HPV based on polymerase chain reaction genotyping studies (*3*). Because rectal squamous cell carcinoma was not included in the genotyping study, the HPV-attributable percentage for anal squamous cell carcinoma, a biologically similar tumor, was used (*2*).

Overall, an average of 38,793 HPV-associated cancers (11.7 per 100,000 persons) were diagnosed annually, including 23,000 (13.5) among females and 15,793 (9.7) among males. The most common of these cancers were 11,771 (7.4 per 100,000 females) cervical carcinomas, and 15,738 (4.5 per 100,000 persons) oropharyngeal SCCs (12,638 among males and 3,100 among females) (Table 1). Rates of oropharyngeal SCC were higher among males (7.6) than females (1.7), whereas rates of anal and rectal SCC were higher among females (1.8 and 0.3) than males (1.1 and 0.2).

Rates of cervical carcinoma were higher among blacks (9.2) than among whites (7.1), and among Hispanics (9.7) than non-Hispanics (7.1); a similar pattern was observed for penile SCCs (Table 1). Rates of vulvar SCC were lower among blacks (1.5) compared with whites (2.1) and among Hispanics (1.3) compared with non-Hispanics (2.1). Among females, rates of anal SCC were lower among blacks (1.4) than whites (1.9), but among males, were higher among blacks (1.5) compared with whites (1.1). The rate of anal SCC among Hispanic males and females (1.1) was lower than among non-Hispanics (1.5). Rates of oropharyngeal SCC in both males and females were higher among whites (8.0 and 1.8) compared with blacks (6.9 and 1.5), and among non-Hispanics (8.0 and 1.8) compared with Hispanics (4.2 and 0.9).

By state, overall rates of all HPV-associated cancers combined ranged from 7.5 per 100,000 persons (Utah) to 14.7 (Kentucky); among females, rates ranged from 9.1 (Utah) to 17.0 (Kentucky and West Virginia), and among males, rates ranged from 6.0 (Utah) to 12.8 (District of Columbia) (Table 2). Most states with overall HPV-associated cancer rates that exceeded the U.S. rate (11.7 per 100,000) were located in the U.S. Census Southern region,[¶] driven by a similar pattern in the distribution of the rates of cervical, anal, and oropharyngeal cancers. The highest rate of cervical cancer was found in Puerto Rico (11.7 per 100,000 females); among the states, the lowest was found in Vermont (4.1) and the highest in West Virginia (9.9).

By multiplying HPV-associated cancer counts by the percent attributable to HPV, 30,700 HPV-associated cancers (79%) were estimated to be attributable to HPV (Table 3). Among these, 24,600 (80%) were attributable to HPV types 16 and 18, which can be prevented by the bivalent, quadrivalent and 9-valent HPV vaccines, and 3,800 (12%) were attributable to

[¶] https://www.census.gov/geo/reference/gtc/gtc_census_divreg.html.

The *MMWR* series of publications is published by the Center for Surveillance, Epidemiology, and Laboratory Services, Centers for Disease Control and Prevention (CDC), U.S. Department of Health and Human Services, Atlanta, GA 30329-4027.

Suggested citation: [Author names; first three, then et al., if more than six.] [Report title]. MMWR Morb Mortal Wkly Rep 2016;65:[inclusive page numbers].

TABLE 1. Rate* and average annual number of human papillomavirus (HPV)-associated cancers,† by anatomic site, sex, age, race, and ethnicity§ — United States, 2008–2012¶

Characteristic	Cervical carcinoma		Vaginal SCC		Vulvar SCC		Penile SCC		Rectal SCC			
	Female		Female		Female		Male		Female		Male	
	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.
Total	**7.4**	**11,771**	**0.4**	**802**	**2.0**	**3,554**	**0.8**	**1,168**	**0.3**	**513**	**0.2**	**237**
Age group (yrs)												
<20	0	12	0	0	—**	—	—**	—	0	0	—	—
20–29	3.0	636	—	—	0.1	23	0	4	—	—	—	—
30–39	11.9	2,350	0.1	19	0.7	134	0.2	30	0	8	0	5
40–49	14.0	3,028	0.4	81	2.0	455	0.5	108	0.3	61	0.2	35
50–59	11.9	2,542	0.7	156	3.3	704	0.9	192	0.7	156	0.3	62
60–69	11.4	1,740	1.2	178	4.6	701	2.1	292	0.9	137	0.4	58
70–79	10.0	919	1.9	178	7.6	694	4.1	306	0.9	82	0.6	42
≥80	7.7	545	2.6	188	11.7	843	5.8	235	1.0	69	0.8	33
Race												
White††	7.1	9,034	0.4	650	2.1	3,170	0.8	989	0.3	455	0.2	196
Black	9.2§§	1,891	0.6§§	117	1.5§§	301	0.9§§	129	0.2§§	43	0.2§§	32
American Indians/Alaska Natives	6.3§§	113	0.3	5	1.1§§	16	0.7	8	—	—	—	—
Asian/Pacific Islander	6.1§§	530	0.2§§	19	0.4§§	31	0.4§§	25	0§§	3	—	—
Ethnicity												
Non-Hispanic††	7.1	9,855	0.4	733	2.1	3,363	0.7	991	0.3	468	0.2	220
Hispanic	9.7§§	1,916	0.5	69	1.3§§	191	1.3§§	177	0.3	44	0.1	17

Characteristic	Oropharyngeal SCC						Anal SCC					
	Female		Male		Female and male		Female		Male		Female and male	
	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.
Total	**1.7**	**3,100**	**7.6**	**12,638**	**4.5**	**15,738**	**1.8**	**3,260**	**1.1**	**1,750**	**1.5**	**5,010**
Age group (yrs)												
<20	—	—	—	—	—**	—	—	—	—	—	—**	—
20–29	0	8	0.1	16	0.1	25	0	4	0.1	11	0	15
30–39	0.3	62	0.8	150	0.6	211	0.3	66	0.4	74	0.4	141
40–49	1.5	338	7.1	1,568	4.2	1,906	2.0	443	1.8	386	1.9	829
50–59	4.2	905	22.5	4,627	13.1	5,532	4.8	1,035	2.6	534	3.8	1,569
60–69	6.0	908	29.1	4,047	17.0	4,955	5.5	843	3.0	413	4.3	1,256
70–79	6.2	570	22.4	1,680	13.5	2,250	5.6	513	2.8	211	4.4	723
≥80	4.4	308	13.3	549	7.7	856	5.0	355	2.9	121	4.3	476
Race												
White††	1.8	2,692	8.0	11,180	4.7	13,871	1.9	2,905	1.1	1,448	1.5	4,353
Black	1.5§§	327	6.9§§	1,152	3.9§§	1,479	1.4§§	279	1.5§§	260	1.4§§	539
American Indians/Alaska Natives	0.9§§	16	4.4§§	66	2.6§§	81	0.9§§	15	0.5§§	7	0.7§§	22
Asian/Pacific Islander	0.6§§	46	2.0§§	136	1.2§§	182	0.4§§	30	0.2§§	15	0.3§§	45
Ethnicity												
Non-Hispanic††	1.8	2,959	8.0	12,025	4.7	14,984	1.9	3,039	1.2	1,634	1.5	4,673
Hispanic	0.9§§	141	4.2§§	612	2.4§§	754	1.4§§	221	0.7§§	116	1.1§§	337

Abbreviation: SCC = squamous cell cancer.

* Per 100,000 persons; age-adjusted to the 2000 U.S. standard population.

† HPV-associated cancers were defined as cancers at specific anatomic sites with specific cell types in which HPV DNA frequently is found. All cancers were confirmed histologically. Cervical cancers (*International Classification of Diseases for Oncology, 3rd Edition* [ICD-0-3] site codes C53.0–C53.9) are limited to carcinomas (ICD-O-3 histology codes 8010–8671, 8940–8941). Vaginal (ICD-O-3 site code C52.9), vulvar (ICD-O-3 site codes C51.0–C51.9), penile (ICD-O-3 site codes C60.0–60.9), anal (ICD-O-3 site code C21.0–C21.9), rectal (ICD-O-3 site code C20.9), and oropharyngeal (ICD-O-3 site codes C01.9, C02.4, C02.8, C05.1, C05.2, C09.0, C09.1, C09.8, C09.9, C10.0, C10.1, C10.2, C10.3, C10.4, C10.8, C10.9, C14.0, C14.2 and C14.8) cancer sites are limited to squamous cell carcinomas (ICD-O-3 histology codes 8050–8084, 8120–8131).

§ Rates are not presented separately for persons with unknown or other race or unknown ethnicity.

¶ Compiled from population-based cancer registries in 49 states and the District of Columbia that participate in the National Program of Cancer Registries, and/or the Surveillance, Epidemiology, and End Results Program and meet criteria for high-quality data for all five years (2008–2012), covering approximately 99% of the U.S. population.

** Data suppressed because the total number of cancers for 2008–2012 was <16.

†† Referent group.

§§ Rate differed significantly from the rate in the referent group ($p<0.05$).

TABLE 2. State incidence* of human papillomavirus (HPV)-associated cancers,† by cancer site and sex — National Program of Cancer Registries and Surveillance, Epidemiology, and End Results program, United States, 2008–2012§

	Cervical	Vaginal	Vulvar	Penile	Anal			Rectal			Oropharyngeal			HPV-associated cancers combined		
State/Territory	Female	Female	Female	Male	Male and female	Male	Female	Male and female	Male	Female	Male and female	Male	Female	Male and female	Male	Female
Alabama	8.1	0.6	2.1	0.8	1.4	1.0	1.7	0.4	0.2	0.5	4.9	8.3	2.0	12.7	10.4	15.0
Alaska	6.7	—¶	2.0	—¶	1.1	—¶	1.6	—¶	—¶	—¶	4.1	6.4	1.7	10.6	8.1	13.1
Arizona	6.2	0.3	1.2	0.6	1.1	0.7	1.4	0.2	0.2	0.3	3.5	6.0	1.3	9.1	7.5	10.6
Arkansas	9.6	0.4	2.2	0.8	1.3	0.9	1.7	0.2	—¶	0.3	5.2	8.9	1.8	13.5	10.9	16.0
California	7.4	0.4	1.4	0.8	1.5	1.2	1.7	0.2	0.2	0.3	3.9	6.7	1.3	10.8	8.9	12.6
Colorado	5.6	0.3	1.6	0.6	1.4	1.0	1.8	0.1	—¶	0.2	4.1	6.9	1.5	9.8	8.6	11.1
Connecticut	6.1	0.5	2.1	0.9	1.3	1.0	1.5	0.2	—¶	0.2	4.4	7.6	1.6	10.9	9.7	12.0
Delaware	8.3	—¶	2.5	—¶	1.6	1.4	1.9	—¶	—¶	—¶	4.7	7.8	2.0	12.8	10.1	15.3
District of Columbia	9.2	—¶	1.7	—¶	2.4	2.7	2.0	—¶	—¶	—¶	5.2	8.9	2.2	14.3	12.8	15.9
Florida	8.5	0.4	1.7	0.8	1.9	1.4	2.3	0.3	0.2	0.4	5.7	9.7	2.2	13.7	12.1	15.4
Georgia	7.7	0.5	2.1	0.9	1.6	1.3	1.8	0.3	0.2	0.4	4.8	8.3	1.8	12.6	10.7	14.3
Hawaii	7.5	0.4	1.0	0.5	0.9	0.7	1.1	—¶	—¶	—¶	3.5	6.2	1.0	9.4	7.6	11.2
Idaho	6.1	0.4	1.9	0.5	1.8	1.0	2.6	0.2	—¶	—¶	4.4	7.5	1.5	10.9	9.0	12.8
Illinois	7.9	0.5	2.1	0.8	1.4	1.1	1.6	0.2	0.1	0.2	4.6	7.9	1.8	12.1	9.9	14.1
Indiana	7.3	0.5	2.4	0.8	1.6	1.1	2.0	0.2	0.1	0.2	5.0	8.5	1.8	12.3	10.5	14.2
Iowa	6.8	0.4	2.8	1.0	1.5	0.9	2.0	0.2	—¶	0.2	4.3	7.2	1.5	11.5	9.2	13.7
Kansas	6.9	0.4	2.2	0.8	1.3	0.7	1.7	0.3	—¶	0.4	4.3	7.5	1.4	11.1	9.1	13.0
Kentucky	8.5	0.7	3.0	1.2	1.7	1.1	2.3	0.3	0.2	0.3	5.8	9.7	2.2	14.7	12.2	17.0
Louisiana	9.1	0.6	2.2	0.9	1.6	1.3	1.8	0.3	0.2	0.4	5.1	8.6	1.9	13.5	11.0	15.9
Maine	5.8	0.4	2.6	1.0	1.7	1.5	1.8	—¶	—¶	—¶	5.4	8.8	2.3	12.3	11.4	13.2
Maryland	6.3	0.4	1.9	0.6	1.4	1.2	1.6	0.2	—¶	0.2	4.1	7.3	1.4	10.5	9.1	11.8
Massachusetts	5.1	0.4	2.3	0.9	1.4	1.1	1.7	0.2	0.1	0.2	5.0	8.2	2.1	11.2	10.3	11.9
Michigan	6.6	0.5	2.4	0.8	1.4	1.0	1.7	0.2	0.2	0.3	4.5	7.5	1.8	11.5	9.5	13.4
Minnesota	5.8	0.4	2.0	1.0	1.2	0.9	1.6	0.1	—¶	0.2	4.2	6.9	1.6	10.2	8.8	11.6
Mississippi	9.3	0.7	2.4	1.2	1.7	1.3	2.0	0.2	—¶	0.3	5.3	9.0	2.0	14.3	11.5	16.7
Missouri	8.1	0.5	2.3	0.6	1.7	1.3	2.0	0.2	—¶	0.2	4.8	8.2	1.8	12.6	10.2	14.9
Montana	6.2	—¶	1.8	1.0	1.4	0.8	1.8	—¶	—¶	—¶	4.5	7.1	1.8	10.7	9.1	12.2
Nebraska	6.8	0.4	2.5	0.7	1.2	0.8	1.5	0.2	—¶	—¶	3.6	5.8	1.5	10.2	7.4	12.9
Nevada¶	—**	—**	—**	—**	—**	—**	—**	—**	—**	—**	—**	—**	—**	—**	—**	—**
New Hampshire	5.0	0.4	2.1	0.8	1.5	0.9	2.1	—¶	—¶	—¶	5.0	7.8	2.3	10.8	9.5	12.0
New Jersey	7.6	0.5	1.9	0.7	1.4	1.0	1.8	0.2	0.1	0.3	3.8	6.6	1.5	11.1	8.5	13.5
New Mexico	7.2	0.5	1.2	1.0	1.2	0.8	1.6	0.2	—¶	0.4	3.3	5.5	1.3	9.9	7.5	12.2
New York	7.7	0.4	1.8	0.9	1.6	1.4	1.8	0.3	0.2	0.3	3.9	6.6	1.6	11.4	9.1	13.6
North Carolina	6.8	0.6	2.3	0.8	1.5	1.2	1.9	0.2	0.1	0.2	5.4	9.3	2.0	12.6	11.3	13.8
North Dakota	6.5	—¶	2.3	—¶	0.6	—¶	0.9	—¶	0	—¶	4.0	6.7	1.3	9.5	7.8	11.3
Ohio	7.1	0.4	2.2	0.8	1.5	1.0	1.8	0.2	0.1	0.3	4.6	7.7	1.7	11.8	9.7	13.7
Oklahoma	8.9	0.4	2.2	0.7	1.6	1.2	2.1	0.3	0.2	0.4	4.8	8.1	1.8	13.0	10.2	15.8
Oregon	6.6	0.4	2.1	0.6	2.0	1.3	2.6	0.1	—¶	0.2	4.7	7.9	1.7	11.7	9.9	13.5
Pennsylvania	7.5	0.4	2.5	0.6	1.4	1.1	1.8	0.2	0.1	0.3	4.5	7.6	1.8	11.9	9.4	14.2
Rhode Island	6.1	0.4	3.4	0.7	1.5	1.0	1.9	—¶	—¶	—¶	4.2	7.3	1.4	11.3	9.1	13.3
South Carolina	7.4	0.4	2.4	0.7	1.5	1.1	1.7	0.2	0.1	0.3	5.3	9.0	2.0	12.7	10.9	14.3
South Dakota	6.6	—¶	2.2	—¶	1.4	0.9	1.9	—¶	—¶	—¶	3.9	6.3	1.6	10.4	8.1	12.8
Tennessee	8.4	0.6	2.5	0.9	1.8	1.3	2.2	0.3	0.2	0.3	4.9	8.3	1.9	13.4	10.6	15.9
Texas	8.4	0.4	1.5	1.0	1.2	0.9	1.4	0.2	0.2	0.3	4.1	6.9	1.5	11.2	9.0	13.5
Utah	5.1	0.3	1.3	0.7	1.0	0.7	1.3	—¶	—¶	—¶	2.7	4.5	0.9	7.5	6.0	9.1
Vermont	4.1	—¶	2.4	1.1	1.4	1.0	1.9	—¶	—¶	—¶	4.9	8.5	1.5	10.7	10.9	10.5
Virginia	6.0	0.4	1.9	0.7	1.4	1.0	1.8	0.2	0.1	0.3	4.3	7.5	1.5	10.6	9.4	11.8
Washington	6.6	0.4	1.8	0.7	1.7	1.1	2.1	0.2	0.2	0.3	4.5	7.5	1.7	11.2	9.5	12.9
West Virginia	9.9	0.5	2.5	1.1	1.5	0.8	2.1	0.2	—¶	—¶	5.1	8.6	1.7	13.9	10.7	17.0
Wisconsin	5.8	0.3	2.0	0.7	1.3	0.9	1.6	0.1	—¶	0.2	4.3	6.8	1.9	10.2	8.5	11.8
Wyoming	8.2	—¶	2.3	—¶	0.8	—¶	1.1	—¶	—¶	—¶	4.3	6.9	1.7	11.0	8.0	14.1
Puerto Rico	11.7	0.7	1.3	2.4	1.1	0.7	1.3	0.4	0.2	0.6	3.2	6.0	0.8	13.1	9.3	16.5

* Rate per 100,000 persons; age-adjusted to the 2000 U.S. standard population.

† HPV-associated cancers were defined as cancers at specific anatomic sites with specific cell types in which HPV DNA frequently is found. All cancers were confirmed histologically. Cervical cancers (*International Classification of Diseases for Oncology, 3rd Edition* [ICD-0-3] site codes C53.0–C53.9) are limited to carcinomas (ICD-O-3 histology codes 8010–8671, 8940–8941). Vaginal (ICD-O-3 site code C52.9), vulvar (ICD-O-3 site codes C51.0–C51.9), penile (ICD-O-3 site codes C60.0–60.9), anal (ICD-O-3 site code C21.0–C21.9), rectal (ICD-O-3 site code C20.9), and oropharyngeal (ICD-O-3 site codes C01.9, C02.4, C02.8, C05.1, C05.2, C09.0, C09.1, C09.8, C09.9, C10.0, C10.1, C10.2, C10.3, C10.4, C10.8, C10.9, C14.0, C14.2 and C14.8) cancer sites are limited to squamous cell carcinomas (ICD-O-3 histology codes 8050–8084, 8120–8131).

§ Compiled from population-based cancer registries in 49 states and the District of Columbia that participate in the National Program of Cancer Registries, and/or the Surveillance, Epidemiology, and End Results Program and meet criteria for high-quality data for all five years (2008–2012), covering approximately 99% of the U.S. population.

¶ Rate suppressed because fewer than 16 cases were reported.

** Data from Nevada did not meet United States Cancer Statistics publication criteria, which assess completeness and the quality of the source of the data. http://www.cdc.gov/cancer/npcr/uscs/technical_notes/criteria.htm.

the five additional HPV types (31, 33, 45, 52, 58), which can be prevented by the 9-valent HPV vaccine. Among cervical carcinoma cases, 7,800 cases were attributable to HPV types 16 and 18 and 1,700 were attributable to the additional HPV types. Among oropharyngeal SCC cases, 9,500 cases were attributable to HPV types 16 and 18, and another 900 cases were attributable to the additional types.

Discussion

Each year during 2008–2012, an average of 38,793 HPV-associated cancers were diagnosed, including 23,000 among females and 15,793 among males; 79% of these were attributable to HPV. Compared with a previous analysis, which reported 33,369 HPV-associated cancer cases diagnosed each year during 2004–2008, the results of this analysis demonstrate an overall increase in HPV-associated cancer incidence, from 10.8 per 100,000 persons during 2004–2008 to 11.7 per 100,000 persons during 2008–2012, despite a slight decrease in the rate of cervical carcinoma (*4*). Part of this increase is because of the inclusion of additional subsites for oropharyngeal cancer; however, the increase persisted when these subsites were excluded from analysis.

The Advisory Committee on Immunization Practices recommends routine vaccination with any of the available HPV vaccines (bivalent, quadrivalent, or 9-valent) for females and quadrivalent or 9-valent for males (*5*). Vaccination is recommended at ages 11–12 years and through age 26 years for females and age 21 years for males, if they were not previously vaccinated (*5*). High-income countries have observed a population-level impact of HPV vaccination programs, including reductions in vaccine type prevalence and rates of anogenital warts, most of which are caused by HPV types 6 and 11, two types targeted by the quadrivalent and 9-valent HPV vaccines (*6*). Among U.S. adolescent females aged 13–17 years in 2014, 60.0% received ≥1 dose, 50.3% received ≥2 doses, and 39.7% received ≥3 doses; male coverage with ≥1, ≥2, and ≥3 doses was 41.7%, 31.4%, and 21.6%, respectively (*7*). Series initiation was higher among blacks and Hispanics compared with whites, and among persons below the poverty level, in both male and female U.S. populations. Increasing vaccination coverage could decrease the cancer incidence and disparities in the United States.

Most cervical cancers are preventable with regular screening for precancerous lesions among women aged 21–65 years linked with follow-up for abnormal test results (*8*); there are currently no effective population-based screening strategies for the other HPV-associated cancers. The *Healthy People 2020* target for cervical cancer screening is 93%**; however in 2013, only 80.7% of women reported up-to-date cervical cancer screening, with even lower rates noted among Asians, Hispanics, women aged 51–65 years, foreign-born, uninsured, and publicly insured women (*9*).

The findings in this report are subject to at least two limitations. First, although population-based cancer registries provide a reliable system for counting invasive cancers, no registry routinely collects or reports information on HPV DNA status in cancer tissue, so the HPV-attributable cancers are only estimates. Second, reporting of race and ethnicity uses data from medical records, which might be inaccurate in a small proportion of cases.

Of the 38,793 cancers that occurred each year in the United States at anatomic sites associated with HPV, approximately 30,700 can be attributed to HPV. Of these, 24,600 cancers are attributable to HPV types 16 and 18, which are included in all current HPV vaccines, and 28,500 are attributable to high-risk HPV types included in the 9-valent HPV vaccine. Ongoing surveillance for 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 and changes in cervical cancer screening practices.

Summary

What is already known about this topic?

Persistent infections with human papillomavirus (HPV) can cause carcinomas of the cervix, and squamous cell cancers of the vulva, vagina, penis, anus, rectum, and oropharynx. Many of these cancers are preventable with currently available vaccines; effective screening programs can identify cervical precancers for treatment before they can progress to cancer.

What is added by this report?

An average of 38,793 HPV-associated cancers (11.7 per 100,000 persons) were diagnosed annually in the United States during 2008–2012, including 23,000 (13.5) among females and 15,793 (9.7) among males. Among these cancers, CDC estimates that 30,700 (79%) can be attributed to HPV, and 28,500 of these are attributable to HPV types that are preventable with the 9-valent HPV vaccine.

What are the implications for public health practice?

Full vaccination coverage of the U.S. population could prevent future HPV-attributable cancers and potentially reduce racial and ethnic disparities in HPV-associated cancer incidence. Ongoing surveillance for 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 and changes in cervical cancer screening practices.

** http://www.healthypeople.gov/2020/topicsobjectives2020/default.aspx.

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TABLE 3. Estimated average annual percentage and estimated number of cancers attributable to human papillomavirus (HPV),* by anatomic site and sex — United States, 2008–2012†

Cancer	Average annual no.	Attributable to any HPV type§	Attributable to HPV 16/18§	Attributable to HPV 31/33/45/52/58§	Attributable to HPV 16/18/31/33/45/52/58§
		No. (%)	No. (%)	No. (%)	No. (%)
Cervical	11,771	10,700 (90.6)	7,800 (66.2)	1,700 (14.7)	9,500 (80.9)
Vaginal	802	600 (75.0)	400 (55.1)	100 (18.3)	600 (73.4)
Vulvar	3,554	2,400 (68.8)	1,700 (48.6)	500 (14.2)	2,200 (62.8)
Penile	1,168	700 (63.3)	600 (47.9)	100 (9.0)	700 (56.9)
All anal cancers	5,010	4,600 (91.1)	4,000 (79.4)	400 (8.2)	4,400 (87.6)
Female	3,260	3,000 (92.5)	2,600 (79.5)	400 (10.8)	2,900 (90.3)
Male	1,750	1,600 (88.7)	1,400 (79.1)	100 (3.8)	1,500 (82.9)
All rectal cancers	750	700 (91.1)	600 (79.4)	100 (8.2)	700 (87.6)
Female	513	500 (92.5)	400 (79.5)	100 (10.8)	500 (90.3)
Male	237	200 (88.7)	200 (79.1)	— (3.8)	200 (82.9)
All oropharyngeal cancers	15,738	11,000 (70.1)	9,500 (60.2)	900 (5.7)	10,400 (65.9)
Female	3,100	2,000 (63.3)	1,600 (50.8)	300 (9.5)	1,900 (60.3)
Male	12,638	9,100 (72.4)	8,000 (63.4)	600 (4.4)	8,600 (67.8)
Total	**38,793**	**30,700 (—)**	**24,600 (—)**	**3,800 (—)**	**28,500 (—)**

* HPV-associated cancers were defined as cancers at specific anatomic sites with specific cell types in which HPV DNA frequently is found. All cancers were confirmed histologically. Cervical cancers (International Classification of Diseases for Oncology, 3rd Edition [ICD-0-3] site codes C53.0–C53.9) are limited to carcinomas (ICD-O-3 histology codes 8010–8671, 8940–8941). Vaginal (ICD-O-3 site code C52.9), vulvar (ICD-O-3 site codes C51.0–C51.9), penile (ICD-O-3 site codes C60.0–60.9), anal (ICD-O-3 site code C21.0–C21.9), rectal (ICD-O-3 site code C20.9), and oropharyngeal (ICD-O-3 site codes C01.9, C02.4, C02.8, C05.1, C05.2, C09.0, C09.1, C09.8, C09.9, C10.0, C10.1, C10.2, C10.3, C10.4, C10.8, C10.9, C14.0, C14.2 and C14.8) cancer sites are limited to squamous cell carcinomas (ICD-O-3 histology codes 8050–8084, 8120–8131).

† Compiled from population-based cancer registries in 49 states and the District of Columbia that participate in the National Program of Cancer Registries, and/or the Surveillance, Epidemiology, and End Results Program and meet criteria for high-quality data for all five years (2008–2012), covering approximately 99% of the U.S. population.

§ Estimates for attributable fraction were based on studies that used population-based data from cancer tissue to estimate the percentage of those cancers probably caused by HPV. The attributable fraction for rectal squamous cell carcinoma was based on the attributable fraction for anal squamous cell carcinoma. The estimated number of HPV-attributable cancers was calculated by multiplying the HPV-associated cancer counts by the percentage of each cancer attributable to HPV. Estimates were rounded to the nearest 100. Estimates less than 100 are not presented. Individual counts may not sum to the total count because of rounding.

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Trends in Methadone Distribution for Pain Treatment, Methadone Diversion, and Overdose Deaths — United States, 2002–2014

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Use of the prescription opioid methadone for treatment of pain, as opposed to treatment of opioid use disorder (e.g., addiction), has been identified as a contributor to the U.S. opioid overdose epidemic. Although methadone accounted for only 2% of opioid prescriptions in 2009 (*1*), it was involved in approximately 30% of overdose deaths. Beginning with 2006 warnings from the Food and Drug Administration (FDA), efforts to reduce methadone use for pain have accelerated (*2*,*3*). The Office of the Assistant Secretary for Planning and Evaluation of the U.S. Department of Health and Human Services and CDC analyzed methadone distribution, reports of diversion (the transfer of legally manufactured methadone into illegal markets), and overdose deaths during 2002–2014. On average, the rate of grams of methadone distributed increased 25.1% per year during 2002–2006 and declined 3.2% per year during 2006–2013. Methadone-involved overdose deaths increased 22.1% per year during 2002–2006 and then declined 6.5% per year during 2006–2014. During 2002–2006, rates of methadone diversion increased 24.3% per year; during 2006–2009, the rate increased at a slower rate, and after 2009, the rate declined 12.8% per year through 2014. Across sex, most age groups, racial/ethnic populations, and U.S. Census regions, the methadone overdose death rate peaked during 2005–2007 and declined in subsequent years. There was no change among persons aged ≥65 years, and among persons aged 55–64 years the methadone overdose death rate continued to increase through 2014. Additional clinical and public health policy changes are needed to reduce harm associated with methadone use for pain, especially among persons aged ≥55 years.

To identify methadone-related deaths, information was obtained from the 2002–2014 National Vital Statistics System multiple cause of death mortality data (*4*). Methadone-related deaths were defined as those with an underlying cause of death classified by the *International Classification of Diseases, 10th Revision* (ICD-10) external cause of injury codes as X40–X44, X60–X64, X85, or Y10–Y14 and ICD-10 code T40.3 for methadone poisoning. Methadone could be listed alone or in combination with other drugs. Age-adjusted death rates were calculated by applying age-specific death rates to the 2000 U.S. standard population age distribution.

Methadone distribution in grams for 2002–2013 was obtained from the Drug Enforcement Administration (DEA) Automation of Reports and Consolidated Orders System.* To limit the analysis to methadone used for pain treatment, methadone distributed to opioid treatment programs was excluded. Data on 2002–2014 reports of methadone diversion, determined through forensic laboratory testing of substances associated with drug cases obtained in federal, state, and local law enforcement operations, were obtained from DEA's National Forensic Laboratory Information System.† Annual counts of methadone diversion reports, and rates per 100,000 population were calculated nationally and by U.S. Census region for 2002–2014. Counts and rates per 100,000 population for methadone overdose deaths were calculated annually, by sex, age group, race/ethnicity, and U.S. Census region for the period 2002–2014. Grams of methadone distributed each year, and rates per 100 population were calculated nationally and by U.S. Census region for 2002–2013.

Joinpoint regression was used to examine changes in trends in rates over time.§ Joinpoint models annual trend data by fitting an exponential curve (i.e., zero joinpoints or no annual percentage change); then adding joinpoints, one at a time, and using a Monte Carlo permutation test to determine the optimal number of joinpoints. In the final model, each joinpoint indicates a statistically significant increase or decrease in trend, and each of these trends is described by an annual percentage change, which represents the average percentage change per year between each joinpoint. For all analyses, a p-value of <0.05 was considered to be statistically significant. The Pearson correlation coefficient (r) was used to assess correlation between the methadone distribution rate and rates of methadone diversion and overdose deaths.

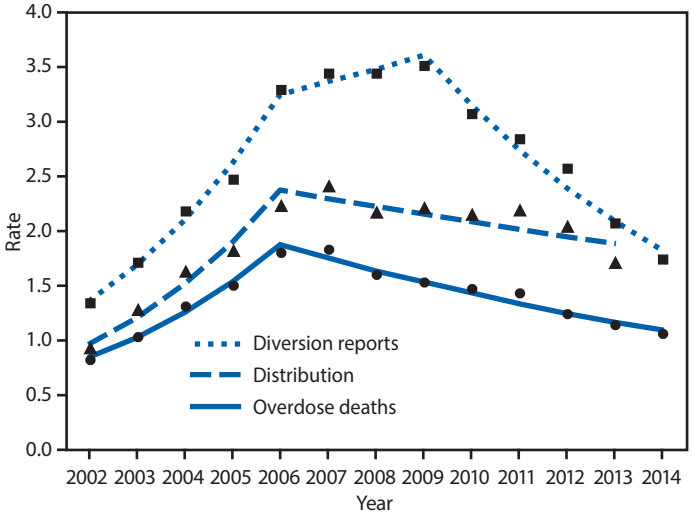
During 2002–2006, the national methadone distribution rate increased, on average, 25.1% per year, and during 2006–2013, declined, on average, 3.2% per year (Figure 1). The methadone overdose death rate increased, on average, 22.1% per year through 2006. After 2006, the overdose death rate declined, on average, 6.5% in each subsequent year. Rates of methadone diversion reports increased, on average, 24.3% per year through 2006 and during 2006–2009 continued to increase, but substantially more slowly (an average of 3.5% per year); after 2009, methadone diversion rates declined, on average, 12.8% per year. There was a strong positive correlation between the rate of methadone distribution and the rates of overdose death ($r = 0.89$, $p<0.05$) and methadone diversion ($r = 0.95$, $p<0.05$).

* http://www.deadiversion.usdoj.gov/arcos/index.html.

† http://www.deadiversion.usdoj.gov/nflis/index.html.

§ http://surveillance.cancer.gov/joinpoint/.

FIGURE 1. Rates* of methadone-involved overdose deaths, methadone distribution, and methadone diversion† reports — United States, 2002–2014§



* The rates shown are for the number of methadone-involved overdose deaths per 100,000 population, number of methadone diversion reports per 100,000 population, and number of grams of methadone distributed per 100 population.
† The transfer of legally manufactured methadone into illegal markets.
§ Each joinpoint represents a statistically significant change in trend, p<0.05.

In the Northeast, Midwest, and South census regions, the methadone distribution rate peaked in 2006, followed by average annual declines of 2.3%, 0.2%, and 5.7%, respectively (Figure 2). The rate of methadone distribution in the West region stabilized during 2006–2011 and then declined, on average, 10.8% annually during 2011–2013. The rates of methadone diversion and overdose death within each region followed a similar pattern as methadone distribution.

Males consistently experienced higher overdose death rates than females during 2002–2014 (Table). The overdose death rate in males increased an average of 23.1% per year during 2002–2006 and then declined an average of 6.5% per year during 2006–2011, followed by a steeper average annual decline (11.0% per year) during 2011–2014. Among females, the overdose death rate increased an average of 20.2% per year during 2002–2006, followed by a more gradual average annual decline (5.6% per year) compared with that among males.

The methadone overdose death rate peaked during 2005–2007 among all groups aged <55 years. Persons aged 25–54 years had the highest overdose death rates during the study period, and all experienced significant declines after 2006. The largest average annual decline (17.3%) occurred among persons aged 15–24 years. Among persons aged 55–64 years, the methadone overdose death rate continued to increase during the study period. There was no statistically significant change in the trend among persons aged ≥65 years.

Summary

What is already known about this topic?

Use of the prescription opioid methadone for treatment of pain, rather than for treatment of opioid use disorder, has been identified as an important contributor to the rise in opioid-related overdose deaths. In recent years, a number of actions to reduce the use of methadone for pain treatment have been taken.

What is added by this report?

During 2002–2006, the national distribution rate of methadone increased, on average, 25.1% per year, methadone-involved drug overdose deaths increased 22.1% per year, and methadone diversion increased 24.3% per year. After 2006, methadone distribution declined 3.2% per year, and methadone-involved overdose deaths declined 6.5% per year. Rates of methadone diversion continued to increase during 2006–2009, but substantially more slowly, and then declined an average of 12.8% per year beginning in 2010. By sex, most age groups, race/ethnicity, and U.S. Census region, the methadone overdose death rate peaked during 2005–2007 and declined in subsequent years. Persons aged 25–54 years had the highest overdose death rates during the study period. There was no significant change in the overdose death rate trend among persons aged ≥65 years, who also had the lowest overdose death rate. Among persons aged 55–64 years, the rate of methadone overdose deaths continued to increase through 2014.

What are the implications for public health practice?

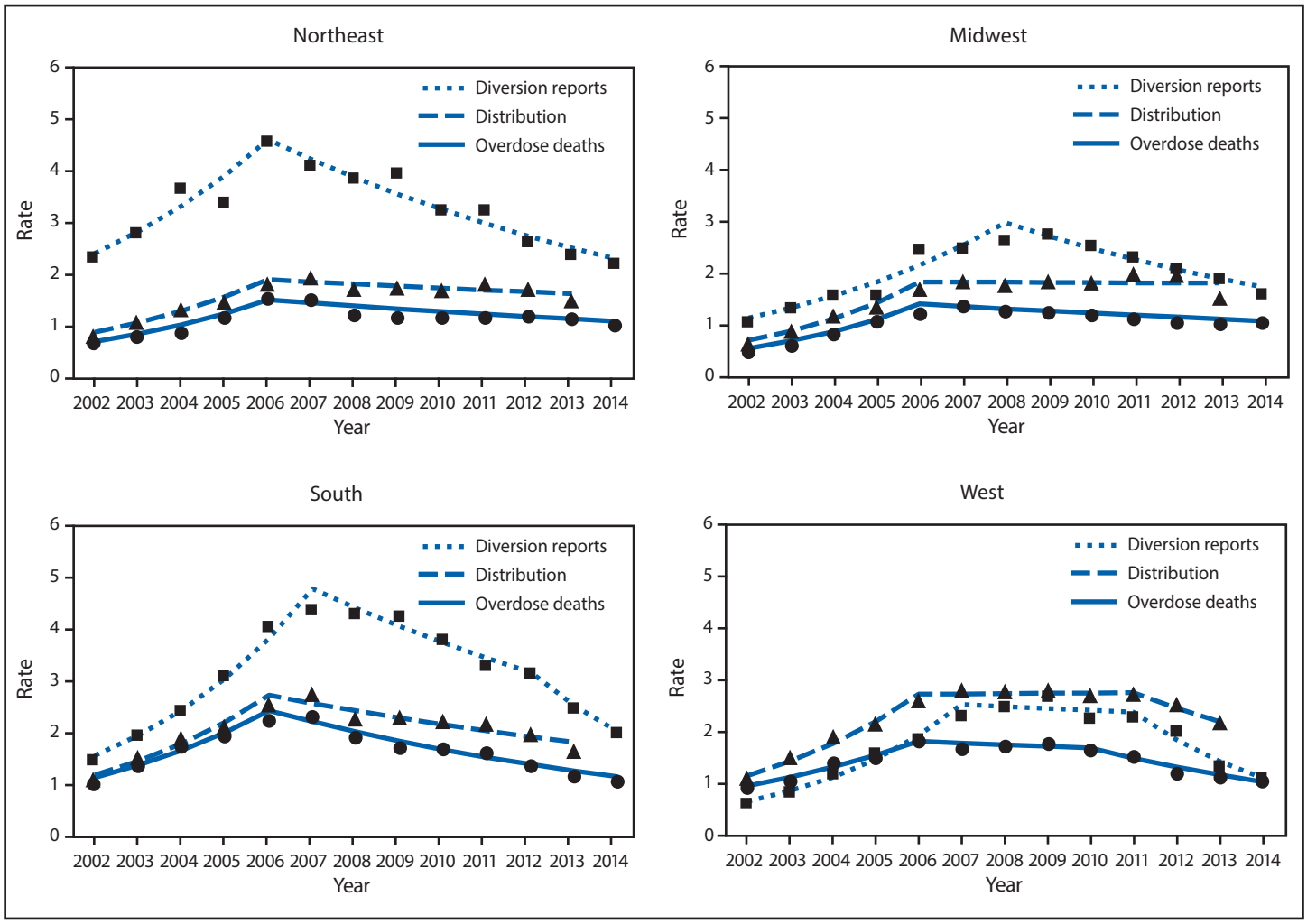
Additional clinical and public health policy changes are needed to further reduce methadone-related harm, especially among persons aged ≥55 years.

The methadone overdose death rate peaked in 2006 among non-Hispanic whites, non-Hispanic blacks, and Hispanics, with average annual declines of 6.6%, 4.3%, and 3.9%, respectively, after 2006. Throughout the study period, non-Hispanic whites experienced higher methadone overdose death rates than other racial/ethnic groups.

Discussion

During 2002–2014, there was a strong positive association between rates of methadone distribution for use in pain treatment and methadone diversion and overdose deaths. The 3,400 reported methadone overdose deaths in 2014 is the lowest number since 2003. With few exceptions, the decline in methadone overdose deaths was seen by sex and across age groups and racial/ethnic populations. Importantly, these declines occurred in the context of more than 100,000 additional persons receiving methadone for the treatment of opioid use disorder during 2002–2013, suggesting that policies targeting methadone use for pain are not affecting access to methadone for treatment of opioid use disorder.

FIGURE 2. Rates* of methadone-involved overdose deaths, methadone distribution, and methadone diversion† reports, by U.S. Census region — United States, 2002–2014§



* The rates shown are for the number of methadone-involved overdose deaths per 100,000 population, number of methadone diversion reports per 100,000 population, and number of grams of methadone distributed per 100 population.
† The transfer of legally manufactured methadone into illegal markets.
§ Each joinpoint represents a statistically significant change in trend, p<0.05.

The declines identified in this study coincide with actions aimed at reducing methadone use for pain. In 2006, FDA issued warnings about the risks of prescribing methadone for pain. Because methadone has a long and variable half-life, FDA also revised the dosing interval from every 3–4 hours to every 8–12 hours (*2*), and in January 2008, DEA and methadone manufacturers agreed to limit the distribution of the largest (40 mg) formulation of methadone to opioid use disorder treatment programs and hospitals.¶ Since that time, professional practice guidelines, medical societies, and government recommendations have targeted reducing the use of methadone for pain (*1*,*5*,*6*). In addition, at least 16 states have removed methadone from their Medicaid preferred drug list, a step that can substantially reduce the routine use of methadone for pain (*3*).

Although the findings in this study are encouraging, the persistent methadone-involved overdose death rates found among persons aged ≥55 years, and the more moderate decline among women, as well as continued increases in opioid overdose deaths not involving methadone, which rose from 11,430 deaths in 2002 to 27,488 deaths in 2014 suggest additional opportunities for intervention. In addition, the variation among U.S. Census regions, which reflects the variation in overall opioid prescribing among states (*7*), underscores the need for interventions that facilitate evidence-based pain treatment and reduce inappropriate opioid prescribing. The Centers for Medicare & Medicaid Services recently issued recommendations on best practices for addressing prescription opioid

¶ http://www.deadiversion.usdoj.gov/pubs/advisories/methadone_advisory.htm.

TABLE. Number and rate* of methadone overdose deaths, by selected characteristics — United States, 2002–2014

Characteristic	2002 No. (rate)	2003 No. (rate)	2004 No. (rate)	2005 No. (rate)	2006 No. (rate)	2007 No. (rate)	2008 No. (rate)	2009 No. (rate)	2010 No. (rate)	2011 No. (rate)	2012 No. (rate)	2013 No. (rate)	2014 No. (rate)	Year of joinpoint†
Overall	**2,358 (0.82)**	**2,972 (1.03)**	**3,845 (1.31)**	**4,460 (1.50)**	**5,406 (1.80)**	**5,518 (1.83)**	**4,924 (1.60)**	**4,696 (1.53)**	**4,577 (1.47)**	**4,418 (1.43)**	**3,932 (1.24)**	**3,591 (1.14)**	**3,400 (1.06)**	**2006**
Sex														
Female	832 (0.57)	1,040 (0.71)	1,343 (0.91)	1,548 (1.03)	1,789 (1.17)	1,921 (1.26)	1,723 (1.10)	1,662 (1.06)	1,646 (1.05)	1,675 (1.05)	1,480 (0.93)	1,457 (0.91)	1,391 (0.87)	2006
Male	1,526 (1.05)	1,932 (1.32)	2,502 (1.71)	2,912 (1.97)	3,617 (2.42)	3,597 (2.40)	3,201 (2.12)	3,034 (1.99)	2,931 (1.91)	2,743 (1.77)	2,452 (1.57)	2,134 (1.35)	2,009 (1.27)	2006, 2011
Age group (yrs)														
15–24	298 (0.73)	442 (1.07)	597 (1.42)	603 (1.42)	811 (1.89)	861 (2.00)	699 (1.61)	624 (1.43)	564 (1.29)	477 (1.09)	341 (0.78)	274 (0.62)	241 (0.55)	2007
25–34	461 (1.17)	540 (1.38)	814 (2.07)	979 (2.49)	1,238 (3.14)	1,305 (3.29)	1,145 (2.85)	1,096 (2.69)	1,150 (2.80)	1,094 (2.62)	989 (2.34)	855 (2.00)	796 (1.83)	2006, 2011
35–44	794 (1.78)	965 (2.19)	1,142 (2.61)	1,217 (2.80)	1,394 (3.22)	1,305 (3.05)	1,102 (2.61)	1,036 (2.50)	982 (2.39)	1,005 (2.47)	906 (2.24)	842 (2.08)	768 (1.90)	2006
45–54	662 (1.66)	841 (2.06)	1,024 (2.46)	1,290 (3.04)	1,521 (3.51)	1,525 (3.47)	1,472 (3.31)	1,332 (2.97)	1,245 (2.77)	1,194 (2.67)	1,017 (2.30)	898 (2.05)	854 (1.97)	2006
55–64	101 (0.38)	151 (0.54)	209 (0.71)	300 (0.98)	351 (1.10)	450 (1.36)	413 (1.21)	523 (1.48)	558 (1.53)	564 (1.48)	590 (1.53)	594 (1.51)	629 (1.57)	2007
≥65	26 (0.07)	—	36 (1.10)	50 (0.14)	63 (0.17)	45 (0.12)	59 (0.15)	69 (0.17)	51 (0.13)	70 (0.17)	75 (0.17)	114 (0.26)	98 (0.21)	NA
Race/Ethnicity														
White, non–Hispanic	2,058 (1.05)	2,623 (1.33)	3,377 (1.73)	3,895 (2.00)	4,745 (2.45)	4,840 (2.46)	4,301 (2.20)	4,062 (2.08)	3,943 (1.98)	3,769 (1.93)	3,332 (1.70)	3,012 (1.54)	2,845 (1.43)	2006
Black, non-Hispanic	141 (0.42)	149 (0.43)	216 (0.60)	257 (0.70)	315 (0.87)	312 (0.81)	245 (0.63)	266 (0.68)	254 (0.62)	278 (0.68)	239 (0.58)	239 (0.59)	256 (0.63)	2006
Other, non-Hispanic	33 (0.21)	39 (0.23)	59 (0.34)	74 (0.42)	81 (0.46)	82 (0.43)	90 (0.45)	97 (0.49)	88 (0.43)	91 (0.44)	86 (0.40)	71 (0.33)	50 (0.24)	2005, 2011
Hispanic	109 (0.32)	151 (0.43)	175 (0.46)	215 (0.55)	246 (0.62)	269 (0.61)	268 (0.60)	243 (0.52)	274 (0.56)	250 (0.51)	241 (0.47)	248 (0.50)	228 (0.46)	2006

Abbreviation: NA = not applicable.
* Rates (deaths per 100,000 population) are presented as age-adjusted rates, with the exception of rates by age group.
† Each joinpoint represents a statistically significant change in trend, p<0.05.

overdose, focusing on steps insurers can take to reduce harms associated with methadone use for pain (*8*). In addition, CDC recommends that methadone not be the first choice for a long-acting opioid and that only clinicians who are familiar with methadone's unique risk profile and are prepared to educate and closely monitor their patients consider prescribing methadone for pain (*9*). Finally, health systems should consider the use of coordinated care plans to optimize evidence-based care. Coordinated care plans structure and coordinate care by increasing precautions for patients taking high dosages, codifying treatment agreements with patients, monitoring patients with urine drug tests and prescription drug monitoring program checks, and avoiding prescribing opioids in conjunction with benzodiazepines; coordinated care plans have been shown to reduce risk factors for opioid-related harm (*10*), and might be especially effective with older patients.

The findings in this report are subject to at least four limitations. First, vital statistics underestimate the number of overdose deaths from specific drugs because the type of drug is not specified on approximately 20%–25% of death certificates. Second, some deaths might have resulted from methadone provided in take-home doses by opioid use disorder treatment programs. Third, although methadone distributed to opioid treatment programs was excluded from the analysis, it is possible that some of the methadone distributed to hospitals, which was included in the analysis, might have been used in the short-term treatment of opioid use disorder. Finally, National Forensic Laboratory Information System estimates of methadone diversion might be subject to variation associated with sample estimates, including nonresponse bias. However, the National Forensic Laboratory Information System estimation methodology was consistent across the study period.

Significant declines in methadone overdose deaths and diversion reports strongly correlate with reduced amounts of methadone used for pain. Insurer strategies and clinical practice guidelines that place parameters and structured monitoring on the use of methadone for pain are promising approaches and should be studied further. Importantly, the declines found in this study appear to be appropriately linked to the use of methadone for pain and not impeding access to methadone for the treatment of opioid use disorder.

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Vital Signs: Motor Vehicle Injury Prevention — United States and 19 Comparison Countries

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On July 6, 2016, this report was posted as an MMWR *Early Release on the* MMWR *website (http://www.cdc.gov/mmwr).*

Abstract

Background: Each year >32,000 deaths and 2 million nonfatal injuries occur on U.S. roads.

Methods: CDC analyzed 2000 and 2013 data compiled by the World Health Organization and the Organisation for Economic Co-operation and Development (OECD) to determine the number and rate of motor vehicle crash deaths in the United States and 19 other high-income OECD countries and analyzed estimated seat belt use and the percentage of deaths that involved alcohol-impaired driving or speeding, by country.

Results: In 2013, the United States motor vehicle crash death rate of 10.3 per 100,000 population had decreased 31% from the rate in 2000; among the 19 comparison countries, the rate had declined an average of 56% during this time. Among all 20 countries, the United States had the highest rate of crash deaths per 100,000 population (10.3); the highest rate of crash deaths per 10,000 registered vehicles (1.24), and the fifth highest rate of motor vehicle crash deaths per 100 million vehicle miles traveled (1.10). Among countries for which information on national seat belt use was available, the United States ranked 18th out of 20 for front seat use, and 13th out of 18 for rear seat use. Among 19 countries, the United States reported the second highest percentage of motor vehicle crash deaths involving alcohol-impaired driving (31%), and among 15, had the eighth highest percentage of crash deaths that involved speeding (29%).

Conclusions and Comments: Motor vehicle injuries are predictable and preventable. Lower death rates in other high-income countries, as well as a high prevalence of risk factors in the United States, suggest that the United States can make more progress in reducing crash deaths. With a projected increase in U.S. crash deaths in 2015, the time is right to reassess U.S. progress and set new goals. By implementing effective strategies, including those that increase seat belt use and reduce alcohol-impaired driving and speeding, the United States can prevent thousands of motor vehicle crash-related injuries and deaths and hundreds of millions of dollars in direct medical costs every year.

Introduction

In the United States, reducing motor vehicle crash deaths has been reported as one of the great public health achievements of the 20th century (*1*). Despite this success, motor vehicle crashes remain a leading cause of death for Americans aged 1–54 years (*2*). Each year >32,000 deaths and 2 million nonfatal injuries occur on U.S. roads (*2*). The purposes of this study were to describe motor vehicle death data for the United States and 19 other high-income countries and to report seat belt use by seating location and the percentage of deaths that involved alcohol-impaired driving or speeding.

Methods

The number of country-specific motor vehicle crash deaths was provided by the World Health Organization's (WHO) most recent Global Status Report on Road Safety (*3*). Data representing 97.3% of the world's population were collected and validated by trained National Data Coordinators. To be included in the study, a country was required to have membership in the Organisation of Economic Co-operation and Development (OECD; http://www.oecd.org), meet the World Bank's definition for high income (gross national income per capita ≥$12,736), have a population >1 million persons, and report the annual number of motor vehicle deaths and vehicle miles traveled. In addition, the difference between the country-reported motor vehicle crash death rate and the WHO-estimated rate could not exceed 1 death per 100,000 population. The United States and 19 of the 34 OECD member countries met these inclusion criteria, including 14 countries in Europe, two in Asia, two in the Americas, and two in Oceania.*

* *Asia:* Israel and Japan. *Europe:* Austria, Belgium, Denmark, Finland, France, Germany, Ireland, the Netherlands, Norway, Slovenia, Spain, Sweden, Switzerland, and the United Kingdom. *The Americas:* Canada and the United States *Oceania:* Australia and New Zealand.

Data from 2000 and 2013 were used for analyses. Motor vehicle crash death rates were calculated per 100,000 population, per 100 million vehicle miles traveled, and per 10,000 registered vehicles. The percentages of crash deaths that involved alcohol-impaired driving and speeding were calculated. National data on seat belt use by seating location (front and rear) and country, were compared, when available.

Data on the number of deaths related to alcohol-impaired driving, reported seat belt use, and the number of registered vehicles were obtained from the Global Status Report on Road Safety (*3*). The U.S. estimates for seat belt use, based on observation of occupants in noncommercial vehicles at controlled intersections, and the number of deaths in 2013 were obtained from the National Highway Traffic Safety Administration (*4*,*5*), and data for Canada were obtained from Transport Canada's National Collision Database (*6*). Data on the number of vehicle miles traveled and deaths related to speeding were obtained from the International Road Traffic and Accident Database,[†] which is maintained by the International Transport Forum, an intergovernmental organization within the OECD (*7*). Original data presented in kilometers were converted to miles.

Results

All 20 countries reported the number of deaths and front seat belt use; 19 countries reported the percentage of deaths related to alcohol-impaired driving; 18 countries reported rear seat belt use; and 15 countries reported the percentage of deaths related to speeding. From 2000 to 2013, the U.S. motor vehicle death rate decreased 31%, from 14.9 to 10.3 deaths per 100,000 population (Figure). The average death rate among all 19 comparison countries declined 56% between 2000 and 2013, from 10.0 deaths per 100,000 to 4.4 deaths per 100,000. Each of the 19 comparison countries had a higher percentage reduction in their motor vehicle crash death rate than did the United States, ranging from 38.3% (Finland) to 75.1% (Spain) (Figure).

During 2013, motor vehicle crash death rates from the 19 comparison countries ranged from 2.7 per 100,000 (Sweden) to 6.5 (Belgium) (Table 1) with mean and median rates of 4.4 and 4.1, respectively. The rate of motor vehicle crash deaths in the United States during 2013 (10.3 per 100,000 [32,894 deaths]) was approximately twice the average rate of the comparison countries. In the United States, these deaths represented 1.10 motor vehicle crash deaths per 100 million vehicle miles traveled; in the comparison countries, this rate ranged from 0.54 (Sweden) to 1.22 (Japan and Spain), with a mean of 0.85 and median of 0.80 (Table 1). Among all 20 countries, the rate in the United States (1.10) was the fifth highest, after Belgium (1.14), Slovenia (1.16), Japan (1.22) and Spain (1.22). The United States also had the highest rate of deaths per 10,000 registered vehicles (1.24); the rate in the comparison countries ranged from 0.44 (Finland) to 1.04 (Belgium), with a mean of 0.68 and median of 0.66 (Table 1). The United States had the second highest rate of registered vehicles per 1,000 population in 2013 (828). In the comparison countries, this rate per 1,000 population ranged from 369 (Israel) to 1,080 (Finland), with a mean and median of 670 (data not shown).

Alcohol-impaired driving was involved in 31% of U.S. motor vehicle crash deaths. Percentages of crash deaths that involved alcohol-impaired driving across 18 countries reporting these data ranged from 3.2% (Israel) to 33.6% (Canada) (mean = 19.1%; median = 18.0%) (Table 2). Speeding was involved in 29% of U.S. motor vehicle crash deaths. In 15 comparison countries reporting these data, the mean (28.8%) and median (29.0%) percentages were similar to the U.S., but ranged from 15% (United Kingdom and Ireland) to 42% (Finland). The United States tied with New Zealand for the second highest percentage of motor vehicle crash deaths related to alcohol impairment, and had the eighth highest percentage of speeding involved deaths (Table 2).

During 2013, 87% front seat belt use and 78% rear seat belt use were reported nationally in the United States (Table 2). Among comparison countries, front seat belt use ranged from 86% (Austria) to 99% (France) with a mean of 94.1% and a median of 95.0%. The United States ranked 18th out of 20 countries for front seat belt use. Among comparison countries, rear seat belt use ranged from 65% (Austria) to 97% (Germany) with a mean of 82.1% and a median of 84%. The United States ranked 13th in rear seat belt use among 18 countries reporting.

Conclusions and Comments

Although substantial progress has been made in reducing the number of motor vehicle crash deaths in the United States, motor vehicle crashes remain a serious public health problem resulting in >32,000 deaths and 2 million nonfatal injuries each year. Compared with 19 other high-income countries, the United States had the most motor vehicle crash deaths per 100,000 population and per 10,000 registered vehicles; the second highest percentage of deaths related to alcohol impairment; the third lowest national front seat belt use; and the lowest percentage decline in the rate of motor vehicle crash deaths between 2000 and 2013. If the United States had the same motor vehicle crash death rate as Belgium (the country with the second highest death rate), 12,000 fewer lives would have been lost in 2013 and an estimated $140 million in direct

[†] International Road Traffic and Accident Database. http://www.itf-oecd.org/IRTAD.

FIGURE. Motor vehicle crash deaths per 100,000 population — 20 high-income countries, 2000 and 2013

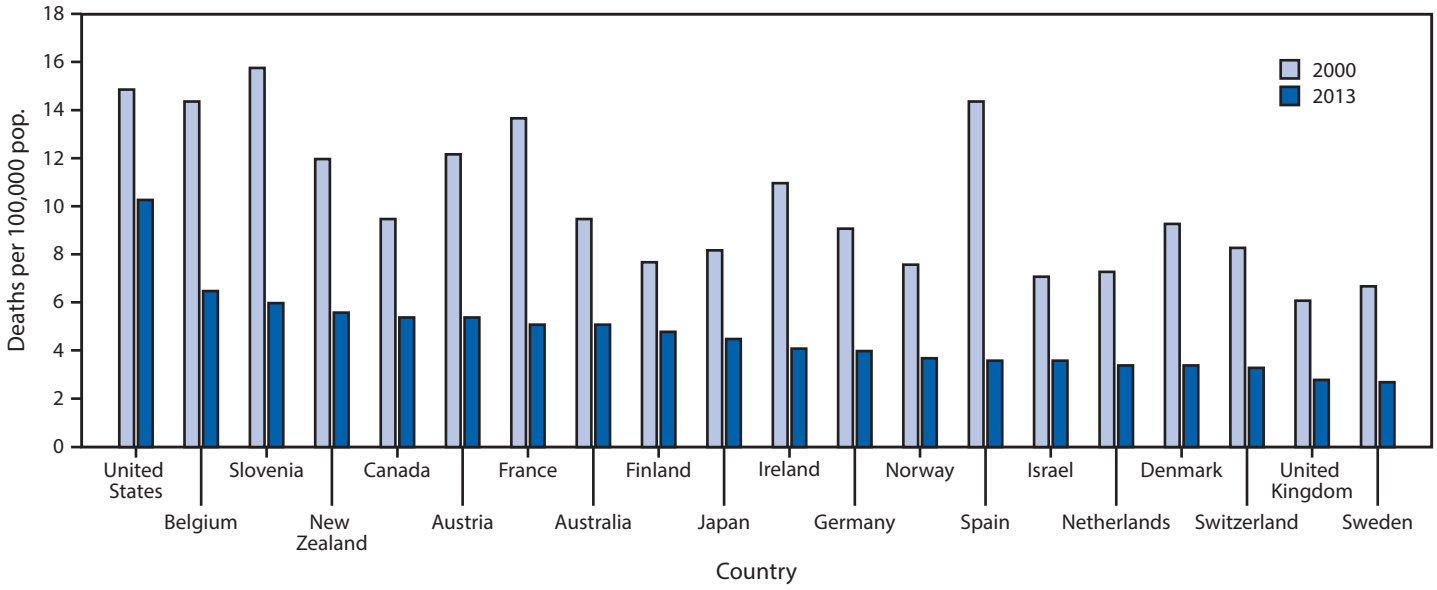

TABLE 1. Motor vehicle crash deaths per 100,000 population, per 100 million vehicle miles traveled, and per 10,000 registered vehicles, and percentage decreases from 2000 to 2013 — selected high-income countries, 2013*

Country†	Reported no. motor vehicle crash deaths	Motor vehicle crash deaths per 100,000 population	Decrease in motor vehicle crash deaths per 100,000 population 2000–2013 (%)	Vehicle miles traveled (billions)	Motor vehicle crash deaths per 100 million vehicle miles traveled	Decrease in motor vehicle crash deaths per 100 million vehicle miles traveled 2000–2013 (%)	Total no. registered vehicles	Motor vehicle crash deaths per 10,000 registered vehicles
United States	32,894	10.3	31.0	2,988.3	1.10	28.0	265,043,362	1.24
Belgium	724	6.5	54.7	63.6	1.14	56.6	6,993,767	1.04
Slovenia	125	6.0	61.8	10.7	1.16	72.9	1,395,704	0.90
New Zealand	253	5.6	53.2	25.1	1.01	54.0	3,250,066	0.78
Canada	1,908	5.4	42.9	211.7	0.90	39.8	22,366,270	0.85
Austria	455	5.4	56.1	48.5	0.94	61.2	6,384,971	0.71
France	3,268	5.1	62.9	352.8	0.93	63.6	42,792,103	0.76
Australia	1,192	5.1	46.2	148.9	0.80	45.3	17,180,596	0.69
Finland	258	4.8	38.3	33.7	0.77	44.0	5,862,216	0.44
Japan	5,679	4.5	45.5	463.7	1.22	43.2	91,377,312	0.62
Ireland	188	4.1	63.1	29.8	0.63	66.0	2,482,557	0.76
Germany	3,339	4.0	55.6	450.9	0.74	59.3	52,391,000	0.64
Norway	187	3.7	51.2	27.3	0.69	59.4	3,671,885	0.51
Spain	1,680	3.6	75.1	137.7§	1.22§	68.4§	32,616,105	0.52
Israel	277	3.6	49.5	31.8	0.87	56.4	2,850,513	0.97
Netherlands	570	3.4	53.4	79.1	0.72	55.2	9,612,273	0.59
Denmark	191	3.4	63.5	30.7	0.62	63.9	2,911,147	0.66
Switzerland	269	3.3	59.9	38.9	0.69	59.5	5,693,642	0.47
United Kingdom	1,770	2.8	54.0	316.0	0.56	53.0	35,582,650	0.50
Sweden	260	2.7	59.5	48.0	0.54	60.8	5,755,952	0.45
Overall mean	2,774.4	4.7	53.9	276.9	0.86	55.5	30,810,704	0.70
Comparison country statistics (n = 19; United States excluded)								
Mean	*1,189.1*	*4.4*	*55.1*	*134.2*	*0.85*	*57.0*	*18,482,670*	*0.68*
Median	*455.0*	*4.1*	*54.7*	*48.5*	*0.80*	*59.3*	*6,384,971*	*0.66*
Range	*125–5,679*	*2.7–6.5*	*38.3–75.1*	*10.7–463.7*	*0.54–1.22*	*39.8–72.9*	*1,395,704–91,377,312*	*0.44–1.04*

* The number of deaths in 2013, total population, and the number of registered vehicles are from the Global Status Report on Road Safety 2015. United States and Canada estimates for the number of deaths in 2013 were obtained from the National Highway Traffic Safety Administration and from Transport Canada's National Collision Database, respectively. The number of deaths in 2000, and vehicle miles travelled are from the Organisation for Economic Co-operation and Development/International Transport Forum (OECD/ITF) Road Safety Annual Report 2015. Data were the most recently available data at the time the Global Status Report 2015 and the OECD/ITF Road Safety Annual Report 2015 were published.

† Countries are listed in descending order by the number of deaths per 100,000 population.

§ Vehicle miles traveled for Spain is for nonurban areas only.

TABLE 2. Percentages of motor vehicle crash deaths, by alcohol impairment and speeding, and national seat belt use for front and rear seat occupants — 20 selected high-income countries, 2013*

	Deaths with specified risk factors (%)		National seat belt use (%)	
Country†	Alcohol-impaired driving	Speeding	Front seat	Rear seat
United States	31.0	29.0	87.0	78.0
Belgium	25.0	—	86.4	—
Slovenia	30.0	39.0	94.5	66.2
New Zealand	31.0	33.0	96.0	90.0
Canada	33.6	20.0	95.5	89.2
Austria	6.8	28.0	86.0	65.0
France	29.0	25.0	99.0	87.0
Australia	30.0	33.0	97.0	96.0
Finland	22.0	42.0	89.0	86.0
Japan	6.2	—	97.9§	68.2§
Ireland	15.6	15.0	94.0	89.0
Germany	9.4	35.0	98.0	97.0
Norway	17.0	—	94.0	—
Spain	14.0	22.0	90.5	80.6
Israel	3.2	—	95.0	74.0
Netherlands	18.9	30.0	96.6	82.0
Denmark	—	40.0	94.0¶	81.0
Switzerland	16.4	26.0	91.0	72.0
United Kingdom**	16.0	15.0	95.0	88.0
Sweden	19.0	—	98.0	84.0
Overall mean	19.7	28.8	93.7	81.8
Comparison country statistics (n = 19; United States excluded)				
Mean	*19.1*	*28.8*	*94.1*	*82.1*
Median	*18.0*	*29.0*	*95.0*	*84.0*
Range	*3.2–33.6*	*15.0–42.0*	*86.0–99.0*	*65.0–97.0*

* Alcohol-impaired driving data are from the Global Status Report on Road Safety 2015. National seat belt estimates are also from the Global Status Report on Road Safety 2015, except for US data. United States estimates for seat belt use were reported from the National Highway Traffic Safety Administration 2013 data. Speeding estimates were reported in the Organisation for Economic Co-operation and Development/International Transport Forum (OECD/ITF) Road Safety Annual Report 2015. Data were the most recently available data at the time the Global Status Report 2015 and the OECD/ITF Road Safety Annual Report 2015 were published.

† Countries are listed in descending order by the number of deaths per 100,000 population.

§ Seatbelt use for Japan was reported for expressways only.

¶ Estimated seat belt use for Denmark was available for drivers of personal vehicles only; other front seat passengers are not included.

** The United Kingdom estimate was for Great Britain only. Great Britain makes up 97% of the population of the United Kingdom.

medical costs would have been averted.§ Similarly, if the United States' motor vehicle crash death rate was equivalent to the average in the 19 comparison countries, at least 18,000 fewer lives would have been lost and an estimated $210 million in direct medical costs would have been averted.§ And, if the United States' motor vehicle crash death rate was equivalent to that in Sweden (the best performing country), at least 24,000 fewer lives would have been lost and an estimated $281 million in direct medical costs would have been averted§ in the United States in 2013.

§ https://wisqars.cdc.gov:8443/costT/.

When accounting for factors that differ across countries, including population size, vehicle miles traveled, and number of registered vehicles, the United States consistently ranked poorly among OECD comparison countries. This low ranking is consistent with other cross-national motor vehicle injury research findings (*8–10*). Although it is difficult to identify and quantify the reasons for differences between the United States and the comparison countries, differences in policies and their enforcement, use of advanced engineering and technology, and differences in public acceptance and use of effective strategies have all contributed to reducing death rates in the best performing countries. The United States is highly dependent on transportation by personal vehicle. In 2014, there were 1.2 vehicles per licensed driver and 2.1 vehicles per household in the United States, and the US share of world car registrations was 15.1% (*11*). Given this reliance on personal vehicles, and need to address safety issues without delay, bringing policies in line with best practices (e.g., related to child passenger safety, seat belt use, and alcohol-impaired driving), enforcement, infrastructure, vehicles, and technologies such as ignition interlocks and automated enforcement (cameras) could help narrow the gap between the United States and higher performing countries (*3*,*12*).

The complexity of improving road safety requires a broad view and more universal implementation and enforcement of existing effective strategies in the United States (*12–14*), as well as system-level changes in vehicle safety and transportation infrastructure (*13*). To maximize lives saved and injuries prevented in the United States, increasing restraint use and reducing alcohol-impaired driving could have the most, as well as an immediate, impact. Each year approximately half the passenger vehicle occupants who die in crashes in the United States are unrestrained (N = 9,777 in 2013)(*15*). Implementing primary enforcement seat belt laws that cover occupants in all seating positions, and requiring the use of car seats and booster seats for motor vehicle passengers through at least age 8 years could increase restraint use and prevent injuries and deaths in the United States. During 2013, seat belts saved approximately 12,500 lives in the United States (*15*). If restraint use was at 100% in the United States, an additional 3,000 lives would be saved in a single year (*15–17*).

Each year in the United States, approximately 10,000 persons die in alcohol-impaired–driving crashes (*18*). Several proven prevention strategies could accelerate progress in the United States (*19*,*20*), including publicized sobriety checkpoints (*21*), ignition interlocks (a breath-test device connected to a vehicle's ignition that prevents the vehicle from starting unless a blood alcohol concentration below a preset low limit is detected) for all convicted offenders (*22*), having lower blood

alcohol concentration limits, and maintaining and enforcing the minimum legal U.S. drinking age of 21 years (*23*).

In addition to effective interventions, there is an approach to road safety that began in Sweden and is gaining traction in the United States called Vision Zero (*24*). This is an aspirational vision that, in the long-term, seeks to eliminate death and serious injury on the road. Vision Zero starts with the premise that traffic injuries are not "accidents", no loss of life on the road is acceptable, all humans make mistakes, and traffic injuries are preventable. In the Vision Zero program, responsibility for crashes and injuries are shared between the users of the road, who are expected to follow basic rules, and the so-called "system providers," which include developers of road infrastructure, the automobile industry, and the police, who are responsible for the functioning of the system. Eighteen U.S. cities have adopted this approach and many more are considering implementing it. Additionally, several U.S. states and the Federal Highway Administration have embraced "Towards Zero Deaths," which is based on the Vision Zero philosophy.¶

The findings in this report are subject to at least three limitations. First, definitions and reporting of motor vehicle deaths vary by country. To limit these differences, countries with motor vehicle death rates that differed substantially from WHO's estimated rates were excluded from the analysis. Second, legal definitions and reporting of alcohol-impaired driving, speeding, and seat belt use also vary among countries. For example, in the United States, the United Kingdom, and Canada, drivers are considered to be alcohol impaired when their blood alcohol concentration is ≥0.08 grams per deciliter (g/dL); whereas, in the other comparison countries, drivers have lower blood alcohol concentrations limits (0.02–0.05 g/dL). Also, in Canada, all provinces except Quebec have administrative laws penalizing drivers (e.g., 3-day license suspension, fine, or 3-day vehicle impoundment) with blood alcohol concentrations of 0.05–0.08 g/dL (0.04–0.08 in Saskatchewan) (*25*). Finally, the United States is larger and more populous than the comparison countries and has a lower population density (rural roads have higher death rates) than most. Travel behaviors, transportation modes, and infrastructure also vary widely among countries. These differences might account for some of the differences in motor vehicle death rates; however, by reporting rates per 100,000 population, per 100 million miles traveled, and per 10,000 registered vehicles, it was possible to partially adjust for these differences.

Motor vehicle injuries are predictable and preventable, and yet, in 2013, 90 persons died every day on U.S. roads. Lower rates in other high-income countries, as well as a high prevalence of risk factors in the United States, suggest that the United States can make more progress toward reducing motor vehicle crash deaths. With a projected increase in U.S. crash deaths in 2015 (*26*), the time is right to reassess progress and set new goals. By implementing proven effective strategies, the United States can save thousands of persons and hundreds of millions of dollars in direct medical costs from motor vehicle crash injuries and deaths every year.

Key Points

- In 2013, the United States motor vehicle crash death rate of 10.3 per 100,000 population had decreased 31% from the rate in 2000; the average rate among 19 high income comparison countries had declined 56% during this time, nearly twice as much. The United States had the lowest percentage decline among the comparison countries from 2000 to 2013.
- Motor vehicle crash deaths are responsible for >32,000 deaths and 2 million nonfatal injuries per year.
- Compared with 19 other high-income countries, the United States had the most motor vehicle crash deaths per 100,000 population and per 10,000 registered vehicles; second highest percentage of deaths involving alcohol-impaired driving; and third lowest national front seat belt use.
- Despite proven measures in motor vehicle injury prevention, in 2013, 90 persons died every day on U.S. roads. Lower rates in other high-income countries suggest that the United States can make more progress in reducing motor vehicle crash deaths.
  - If the United States' motor vehicle crash death rate was equivalent to the rate in Belgium (the country with the second highest death rate), 12,000 fewer lives would have been lost and $140 million in direct medical costs would have been averted in 2013.
  - If the United States' motor vehicle crash death rate was equivalent to the average of the 19 comparison countries, at least 18,000 fewer lives would have been lost and $210 million in direct medical costs would have been averted in 2013.
  - If the United States' motor vehicle crash death rate was equivalent to the rate in Sweden (the best performing country), at least 24,000 fewer lives would have been lost and $281 million in direct medical costs would have been averted in 2013.
- Additional information is available at http://www.cdc.gov/vitalsigns.

¶ http://www.towardzerodeaths.org.

Acknowledgment

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Notes from the Field

Outbreak of Hand, Foot, and Mouth Disease Caused by Coxsackievirus A6 Among Basic Military Trainees — Texas, 2015

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On July 7, 2015, a man aged 22 years reported to sick call during basic military training at Lackland Air Force Base (AFB), Texas. He had erythematous, crusted papulovesicular lesions on the extensor surfaces of the upper and lower extremities. The patient was afebrile and otherwise well, and was evaluated later that day by the dermatology service. A viral infection was considered most likely because of the patient's age, absence of fever or constitutional symptoms, and the distribution and morphology of the lesions. The initial differential diagnosis included Henoch-Schönlein purpura, parvovirus B19, and Rocky Mountain spotted fever. However, the clinical signs, including the unique morphology and distribution of grouped vesicles and papules was suggestive of hand, foot, and mouth disease (HFMD), although the patient did not have oral lesions and reported no contact with another person with HFMD. A viral culture and punch biopsy of one of the lesions were obtained.

On July 8, another patient with similar complaints was evaluated at the clinic, and by September 18, a total of 53 patients had been evaluated (Figure 1). Ten patients, who had extensive involvement of the face, forearms, and lower extremities (Figure 2), were evaluated in the dermatology clinic. Prodromal symptoms of fever and malaise were reported by 11% and 96% of patients, respectively; these symptoms were typically followed by erosive stomatitis and a rash that began on the palms and soles. Patients ranged in age from 18 to 33 years (mean = 21 years [the overall average age of basic trainee population]). Forty-eight (91%) patients were male (overall, approximately 77% of all trainees are male).

A suspected case of HFMD was defined as the occurrence of multiple erythematous papulovesicular lesions on the legs, arms, face, or oral mucosa in a person involved in basic military training activities at Lackland AFB during July 6–September 18. A confirmed case was defined as an illness meeting the clinical case definition with laboratory identification of an enterovirus.

FIGURE 1. Confirmed and suspected cases of hand, foot, and mouth disease, by date of symptom onset and patients' military classification — Lackland Air Force Base, Texas, July 6–September 18, 2015

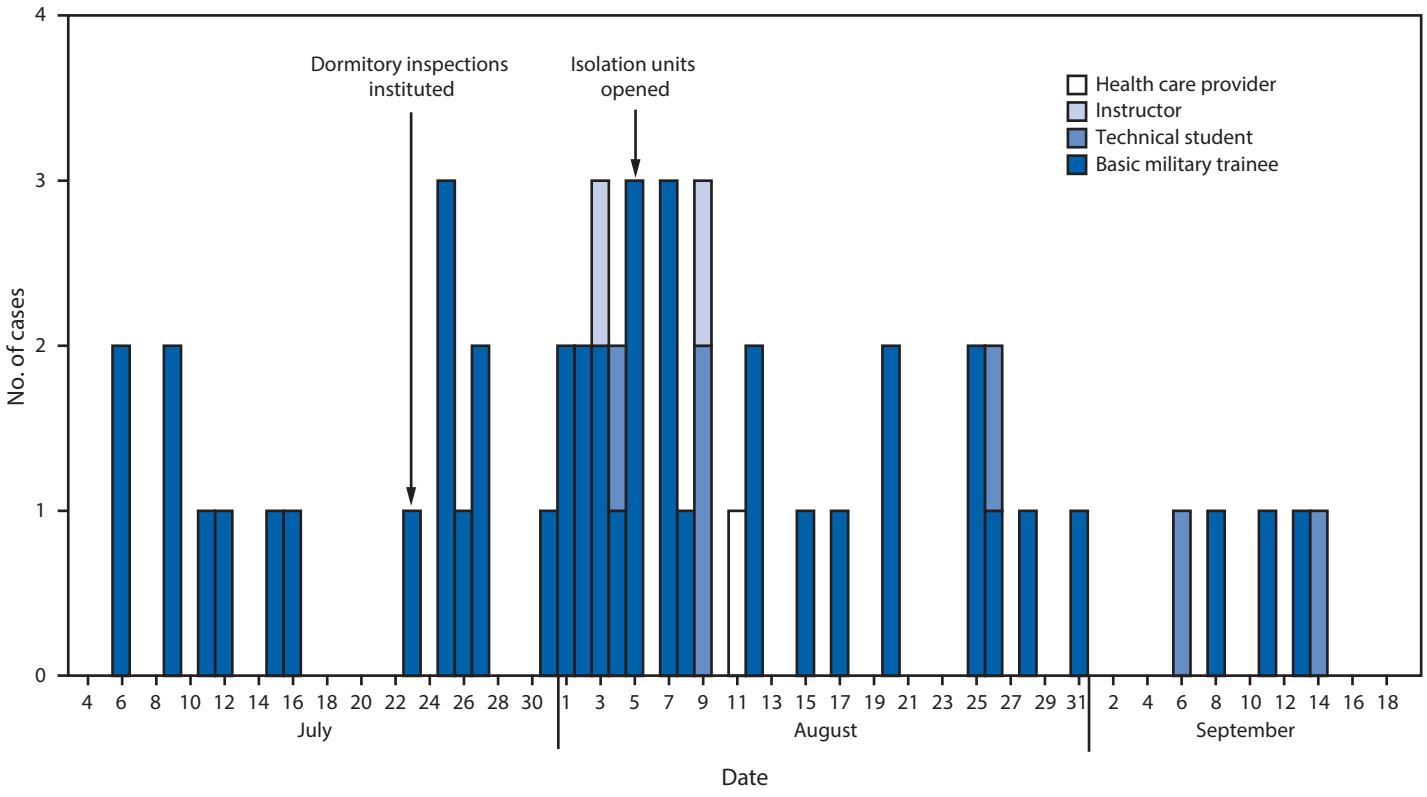
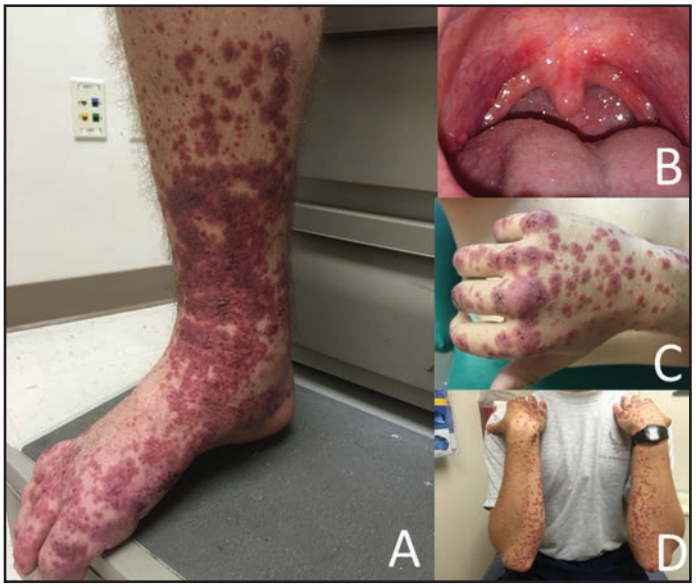


FIGURE 2. Dermatologic and mucosal manifestations of hand, foot, and mouth disease among military personnel, demonstrating (A) extensive and confluent purpuric and hemorrhagic crusted papules and plaques on the foot and anterior shin; (B) erythematous papules and erosions on the palate; (C) grouped purpuric papules on the hand; and (D) similar lesions with extensive involvement of the extensor aspects of the upper extremities — Lackland Air Force Base, Texas, July 6–September 18, 2015



Fifty-three cases (eight confirmed, 45 suspected) were identified in 44 basic military trainees, six recent graduates, two instructors, and one health care provider (Figure 1). Patients were initially identified through provider reporting. However, once the outbreak was recognized, some patients did not receive a referral for a dermatology or infectious diseases consultation; these patients were later identified through a retrospective record review of the electronic health record for all patients on the installation. This method most likely captured all cases of HFMD occurring on the installation during the defined period: basic trainees are in a closed environment under strict supervision, and all basic training instructors were directed to have any trainee displaying symptoms be seen by a provider on base. Eight of 12 nasopharyngeal specimens tested locally by reverse transcription–polymerase chain reaction (RT-PCR) were positive for enterovirus. Five enterovirus-positive specimens were tested by pan-enterovirus viral protein 1 (VP1) RT-PCR, followed by Sanger sequencing of four of five positive specimens at CDC. Bioinformatic analysis of the sequences identified coxsackievirus A6 (CVA6) in all four nasopharyngeal specimens. The rate of infection was 0.4% (50 of 12,270 persons) in the basic training population and 0.3% (two of 602 persons) among instructors. After accounting for clustering (i.e., restricting the population at risk for HFMD to persons in training subunits that experienced cases), the rate of infection among trainees was 4.7% (50 of 1,054 persons).

HFMD typically occurs in children in the United States during summer and autumn. Clinical manifestations usually begin with fever and malaise and can progress to painful oral lesions and a rash involving the hands and feet; infected adults often are asymptomatic. Although coxsackievirus A16 has historically been the primary etiologic agent of HFMD in the United States, CVA6 has emerged as the cause in many recently reported outbreaks (*1*–*5*). Young adults appear to be more susceptible to infection with CVA6, which causes atypical perioral eruptions and skin manifestations that extend beyond the palmar and plantar surfaces (*1*). Military trainees are particularly vulnerable to communicable diseases, because of close living and sleeping arrangements, physical and mental stressors, and substandard hygiene (*6*). Transmission of CVA6 occurs through contact with a patient's respiratory secretions, scabs, vesicle fluid, and feces, and through contact with fomites (*7*).

After the first five cases were identified, steps were taken to limit spread and minimize lost training days by providing education about the signs and symptoms of HFMD and the importance of seeking care if symptoms developed. Surgical masks were issued to trainees with suspected HFMD (*8*), who also slept in separate bedrooms and dined apart from other trainees. Public health personnel inspected all affected living quarters and training sites (Figure 1) and made recommendations regarding hygiene (*9*); the clinical response was coordinated among all providers who might evaluate new cases. Clinics were advised to issue face masks to all patients suspected to be infected with HFMD. Cases continued to be reported for weeks after these interventions (Figure 1), but the disease was confined to 20 (17%) of the 117 basic military training units on the installation during the outbreak period. All 53 cases resolved spontaneously with supportive therapy; none required hospitalization. HFMD infection is primarily determined by clinical diagnosis because CVA6 is a fastidious enterovirus and typically does not easily grow in culture; VP1 RT-PCR followed by sequencing and sequence analysis can be used to confirm the diagnosis (*10*). Measures to prevent transmission should be implemented as soon as clinical suspicion of the disease occurs.

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Notes from The Field

Ebola Virus Disease Cluster — Northern Sierra Leone, January 2016

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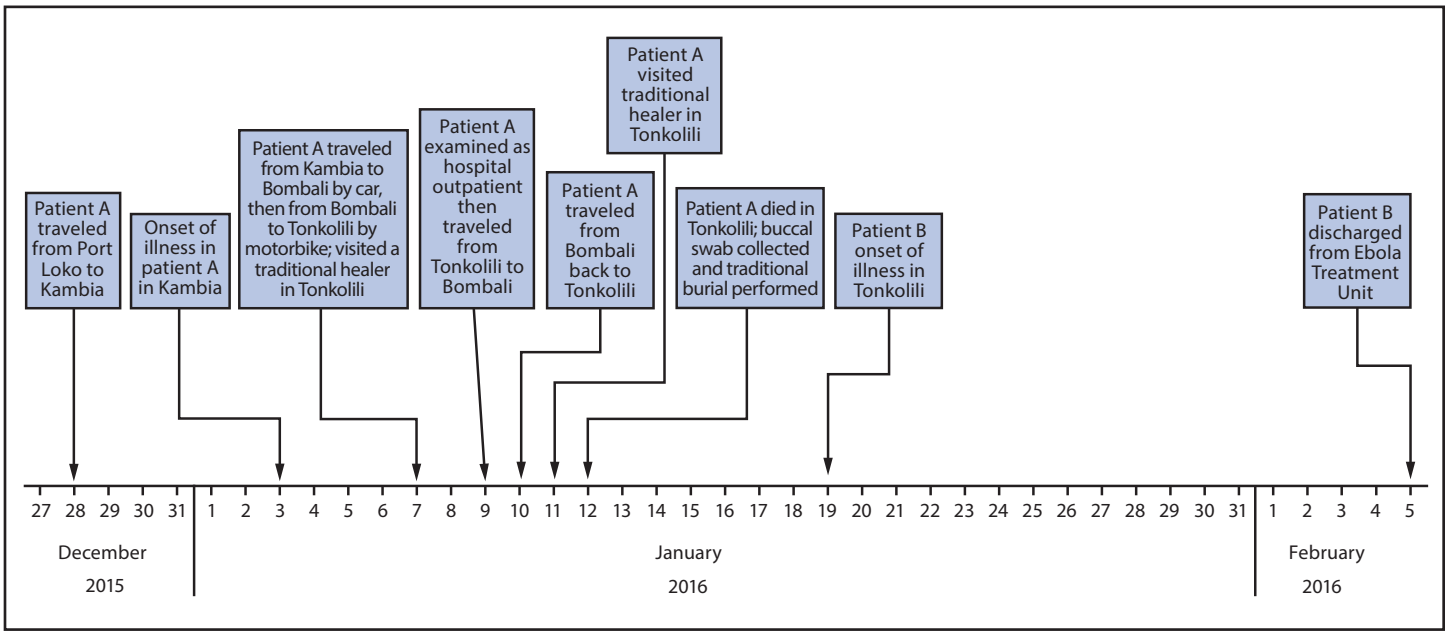
On January 14, 2016, the Sierra Leone Ministry of Health and Sanitation was notified that a buccal swab collected on January 12 from a deceased female aged 22 years (patient A) in Tonkolili District had tested positive for Ebola virus by reverse transcription–polymerase chain reaction (RT-PCR). The most recent case of Ebola virus disease (Ebola) in Sierra Leone had been reported 4 months earlier on September 13, 2015 (*1*), and the World Health Organization had declared the end of Ebola virus transmission in Sierra Leone on November 7, 2015 (*2*). The Government of Sierra Leone launched a response to prevent further transmission of Ebola virus by identifying contacts of the decedent and monitoring them for Ebola signs and symptoms, ensuring timely treatment for anyone with Ebola, and conducting an epidemiologic investigation to identify the source of infection.

Patient A lived in Port Loko District and traveled to Kambia District on December 28, 2015, where she stayed with family and became ill on January 3, 2016 (Figure). Her initial complaints were severe weakness, constipation, and an episode of self-induced vomiting. On January 7 she left Kambia by car, stopping briefly in Bombali District to change to a motorbike before proceeding to Tonkolili, where she was cared for by relatives and saw a traditional healer. She was seen as an outpatient at a government hospital on January 9, but was not tested for Ebola virus. After this visit, she continued to Bombali to see another traditional healer and spent the night there, returning to Tonkolili on January 10. On January 11, she sought care from the traditional healer in Tonkolili a second time. She died in Tonkolili on January 12. As per national policy for all deaths at that time, a routine postmortem buccal swab was collected for Ebola virus RT-PCR by a person trained in swab collection. Her family and community members performed a traditional burial during which they washed the decedent's body and her clothes, prior to RT-PCR results being available.

Investigations identified 131 contacts across four districts, with the majority in Tonkolili (46 persons [35%]) and Kambia (45 [34%]). Where possible, contacts were monitored for 21 days after their last possible exposure to patient A; however, 12 contacts potentially at high risk and 36 persons of interest from Kambia were not located. Because some contacts were not located, Kambia implemented enhanced community surveillance for 2 months after the end of contact monitoring.

FIGURE. Timeline of events for patients A and B in the Tonkolili cluster of Ebola virus disease (Ebola) — Sierra Leone, December 2015–February 2016



Interviews with contacts of patient A failed to identify a source of infection. The viral genome obtained from her buccal swab (1601_C12_KT014149b, GenBank KX121193) indicated a high similarity (one and two nucleotide differences) to two viral genomes from Western Area, Sierra Leone, from November 2014 (KP759709, KP759704). The minimal genetic change in the viral genome during the interval from November 2014 until patient A's illness onset suggests viral persistence in a survivor as the source of infection (*3*), although no survivors were identified who could conclusively be linked to patient A.

On the night of January 19, a high-risk female contact of patient A who was in quarantine (patient B) complained of weakness, chest pain, nausea, and a single episode of self-induced vomiting. Patient B was isolated on the morning of January 20, and her blood tested positive for Ebola virus by RT-PCR that day. Patient B's viral genome (2001_C11_KTO14515b, GenBank KX121194) was identical to that of patient A. Patient B was transferred to an Ebola Treatment Unit in Freetown, Sierra Leone, where she was successfully treated; she was discharged on February 5.

After the declaration of the end of Ebola virus transmission in Sierra Leone, the nation's policy of performing buccal swabs for Ebola virus RNA on all decedents continued. Without this policy, patient A's infection would not have been detected.

The success of this response, the first led by the Sierra Leone Ministry of Health and Sanitation after transition from the National and District Ebola Response Centers on January 1, 2016, can be measured by the case's detection from a routine swab, genetic sequencing performed by locally trained scientists, and the limitation of transmission of Ebola virus from the index case to a single, identified high-risk contact.

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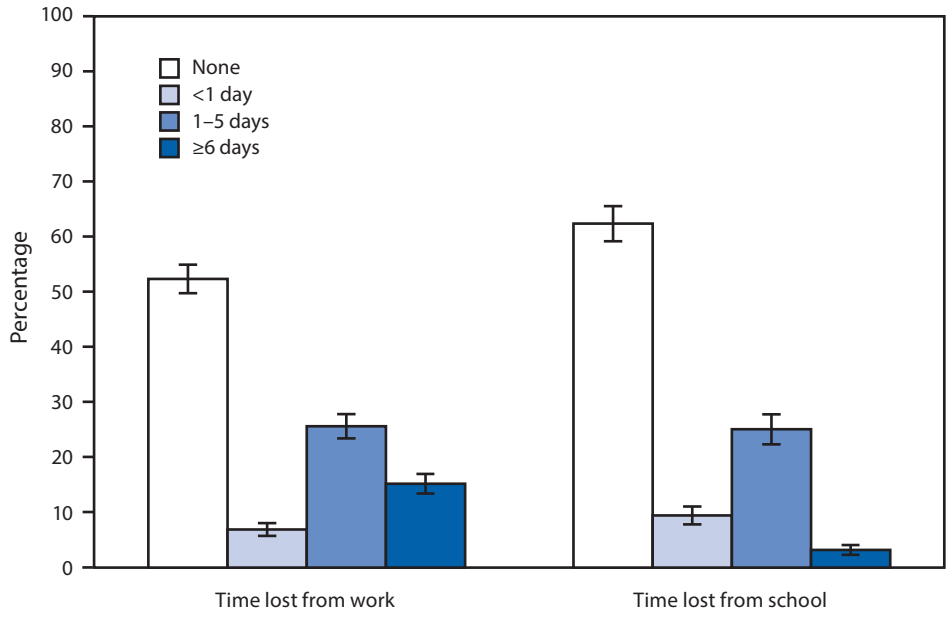
QuickStats

FROM THE NATIONAL CENTER FOR HEALTH STATISTICS

Percentage* of Medically Attended Injury Episodes† That Resulted in Time Lost from Work§ or School,¶ by Number of Days Lost — National Health Interview Survey, United States, 2011–2014



* With 95% confidence intervals.
† An injury episode refers to a trauma event resulting in damage to the body from an external cause. Estimates are for nonfatal, medically attended injuries occurring during the 5 weeks preceding the interview.
§ Time lost from work among persons aged ≥13 years who were employed at the time of injury.
¶ Time lost from school among students aged ≥5 years who attended school at the time of injury.

During 2011–2014, an average of 15.6 million medically attended injury episodes were reported annually among employed persons aged ≥13 years. Nearly half of these injury episodes resulted in time lost from work: 7% for <1 day, 26% for 1–5 days, and 15% for ≥6 days. An average of 9.4 million medically attended injury episodes were reported annually among persons aged ≥5 years who attended school. More than one third of these injury episodes resulted in time lost from school: 9% for <1 day, 25% for 1–5 days, and 3% for ≥6 days.

Source: National Health Interview Survey, 2011–2014 data. http://www.cdc.gov/nchs/nhis.htm.

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ISSN: 0149-2195 (Print)