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Invasive Cancer Incidence and Survival — United States, 2013

S. Jane Henley, MSPH[1]; Simple D. Singh, MD[1]; Jessica King, MPH[1]; Reda J. Wilson, MPH[1]; Mary Elizabeth O'Neil, MPH[1]; A. Blythe Ryerson, PhD[1]

Although cancer represents many heterogeneous diseases, some cancer types share common risk factors. For example, conclusive evidence links cancer at multiple sites with tobacco use, alcohol use, human papillomavirus (HPV) infection, excess body weight, and physical inactivity (*1*,*2*). To monitor changes in cancer incidence and assess progress toward achieving *Healthy People 2020* objectives,* CDC analyzed data from the U.S. Cancer Statistics (USCS) data set for 2013, the most recent year for which incidence and survival data are available. In 2013, a total of 1,559,130 invasive cancers were reported to cancer registries in the United States (excluding Nevada), for an annual age-adjusted incidence rate of 439 cases per 100,000 persons. Cancer incidence rates were higher among males (479) than females (413), highest among blacks (444), and ranged by state from 364 (New Mexico) to 512 (Kentucky) per 100,000 persons (359 in Puerto Rico). The proportion of persons with cancer who survived ≥5 years after diagnosis was 67%. This proportion was the same for males and females (67%), but lower among blacks (62%) than among whites (67%). Cancer surveillance data are key to cancer epidemiologic and clinical outcomes research, program planning and monitoring, resource allocation, and state and federal appropriations accountability.

The USCS data set is a compilation of data from multiple sources and is used to report official federal cancer statistics through the USCS web-based report. USCS includes high quality incidence data from population-based cancer registries affiliated with CDC's National Program of Cancer Registries (NPCR) and the National Cancer Institute's (NCI's) Surveillance, Epidemiology, and End Results (SEER) program in each state, the District of Columbia (DC), and Puerto Rico; survival data from NPCR; and mortality data from the National Vital Statistics System (*3*,*4*). This report includes data on new cases of invasive cancer diagnosed in 2013 (the most recent year with data available); invasive cancers are all cancers excluding in situ cancers (except in the urinary bladder) and basal and squamous cell skin cancers. Data from DC and all states except Nevada met USCS publication criteria for 2013†;

* As of 2017, *Healthy People 2020* objectives included improving the proportion of persons surviving ≥5 years after cancer diagnosis to 71.7%, reducing colorectal cancer incidence to 39.9 per 100,000 persons, reducing late-stage breast cancer incidence to 42.1 per 100,000 females, and reducing cervical cancer incidence to 7.2 per 100,000 females (https://www.healthypeople.gov/2020/topics-objectives).

† Cancer registries demonstrated that cancer incidence data were of high quality by meeting the six USCS publication criteria: 1) case ascertainment ≥90% complete; 2) ≤5% of cases ascertained solely on the basis of death certificate; 3) ≤3% of cases missing information on sex; 4) ≤3% of cases missing information on age; 5) ≤5% of cases missing information on race; and 6) ≥97% of registry's records passed a set of single-field and interfield computerized edits that test the validity and logic of data components (https://www.cdc.gov/cancer/npcr/uscs/index.htm).

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consequently, incidence data in this report cover 99% of the U.S. population. For comparability with past estimates, data for the United States were restricted to the states and DC, and data for Puerto Rico were analyzed separately. Cases were classified first by anatomic site, using the *International Classification of Diseases for Oncology, Third Edition*.[§] Cases with hematopoietic histologies were classified further, using the *World Health Organization Classification of Tumours of Haematopoietic and Lymphoid Tissues, Fourth Edition*.[¶] Breast cancers were characterized by stage at diagnosis using *SEER Summary Staging Manual 2000*[**]; late-stage cancers include those diagnosed after they had spread regionally or metastasized. To characterize the potential cancer prevalence associated with common risk factors, cancer sites were grouped by association with tobacco use, alcohol use, or HPV infection.[††] Population denominators for incidence rates were annual race-, ethnicity-, and sex-specific county population estimates from the U.S. Census, as modified by NCI and aggregated to the state and national level.[§§] Annual incidence rates per 100,000 population were age-adjusted to the 2000 U.S. standard population.

Survival estimates were based on data from NPCR-funded states that met USCS publication criteria and conducted active case follow-up or linkage with CDC's National Center for Health Statistics National Death Index (*5*). For this report, 29 states met these criteria, covering 66% of the U.S. population. The 5-year relative survival proportion was defined as the proportion of persons surviving ≥5 years after cancer diagnosis compared with the proportion of survivors expected in a comparable group of cancer-free persons. The 5-year relative survival proportion was calculated using the Ederer II actuarial method for cases of cancer diagnosed during 2006–2012 with follow-up through 2012, accounting for shorter follow-up time of cases diagnosed in more recent diagnosis years.

In 2013, a total of 1,559,130 invasive cancers were diagnosed and reported to central cancer registries in the United States (excluding Nevada), including 781,451 among males and 777,679 among females (Table 1). The age-adjusted annual incidence for all cancers was 439 per 100,000 persons (479 in males and 413 in females). Among persons aged <20 years, 14,728 cancers (18 per 100,000 persons <20 years) were diagnosed in 2013 (Table 1). The cancer incidence rate increased

[§] http://codes.iarc.fr/.

[¶] https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3109529/.

[**] https://seer.cancer.gov/tools/ssm.

[††] Tobacco-associated cancers include cancers of the oral cavity and pharynx; esophagus; stomach; colon and rectum; liver; pancreas; larynx; lung, bronchus, and trachea; cervix; kidney and renal pelvis; urinary bladder; and acute myeloid leukemia. Alcohol-associated cancers include cancers of the oral cavity and pharynx; esophagus; colon and rectum; liver; larynx; and female breast. HPV-associated cancers include microscopically confirmed carcinoma of the cervix and squamous cell carcinomas of the vagina, vulva, penis, anus, rectum, and oropharynx.

[§§] Population estimates incorporate bridged single-race estimates derived from the original multiple race categories in the 2010 U.S. Census. https://seer.cancer.gov/popdata/index.html; http://www.census.gov/programs-surveys/popest.html.

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TABLE 1. Number and annual age-adjusted rate* of invasive cancers,† by sex, cancer site, race/ethnicity,§ and age group — National Program of Cancer Registries and Surveillance, Epidemiology, and End Results Program, United States,¶ 2013

	Overall		Male		Female	
Characteristic	**Rate**	**No. (%)**	**Rate**	**No. (%)**	**Rate**	**No. (%)**
All cancer sites	**439.0**	**1,559,130 (100)**	**479.0**	**781,451 (100)**	**412.6**	**777,679 (100)**
Prostate	NA	176,450 (11)	101.6	176,450 (23)	NA	NA
Female breast	NA	230,815 (15)	NA	NA	123.7	230,815 (30)
Late-stage female breast	NA	76,816 (5)	NA	NA	42.1	76,816 (10)
Lung and bronchus	59.4	212,584 (14)	69.8	111,907 (14)	51.5	100,677 (13)
Colon and rectum	38.4	136,119 (9)	44.2	71,099 (9)	33.6	65,020 (8)
Cervix uteri	NA	11,955 (1)	NA	NA	7.2	11,955 (2)
Race/Ethnicity						
White	439.3	1,304,263 (84)	473.9	654,240 (84)	417.4	650,023 (84)
Black	443.6	170,123 (11)	518.6	85,190 (11)	393.6	84,933 (11)
American Indian/Alaska Native	276.4	8,676 (1)	289.3	4,097 (1)	269.6	4,579 (1)
Asian and Pacific Islander	284.0	47,802 (3)	290.5	21,052 (3)	283.3	26,750 (3)
Hispanic	343.7	117,332 (8)	372.0	55,393 (7)	328.7	61,939 (8)
Age group (yrs)						
<20	18.0	14,728 (1)	18.7	7,815 (1)	17.3	6,913 (1)
20–49	154.8	187,560 (12)	111.9	68,160 (9)	197.2	119,400 (15)
50–64	798.3	505,337 (32)	837.0	258,164 (33)	763.2	247,173 (32)
65–74	1,756.0	433,944 (28)	2,075.9	239,204 (31)	1,477.0	194,740 (25)
≥75	2,163.1	417,561 (27)	2,685.9	208,108 (27)	1,813.7	209,453 (27)

Abbreviation: NA = not available.

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

† Excludes basal and squamous cell carcinomas of the skin, except when these occur on the skin of the genital organs, and in situ cancers, except urinary bladder.

§ Racial categories are not mutually exclusive from Hispanic ethnicity. Rates are not presented for cases with unknown or other race. Hispanic category excludes cases from Virginia because a large percentage of cases were missing information on ethnicity.

¶ Compiled from data from cancer registries in 49 states and the District of Columbia that meet the data quality criteria for all invasive cancer sites combined (covering approximately 99% of the U.S. population).

with increasing age group, with highest rates (2,163 per 100,000 persons) among persons aged ≥75 years (Table 1).

By cancer site, incidence rates were highest for cancers of the female breast (124 per 100,000 females); prostate (102 per 100,000 males); lung and bronchus (lung) (59 per 100,000 persons); and colon and rectum (colorectal) (38 per 100,000 persons) (Table 1). These four sites accounted for approximately half of cancers diagnosed in 2013, including 230,815 female breast cancers, 176,450 prostate cancers, 212,584 lung cancers, and 136,119 colorectal cancers. In 2013, cervical cancer incidence was 7.2 per 100,000 females, representing 11,955 reported cancers (Table 1). The incidence (rates per 100,000 persons) for cancers associated with tobacco use, alcohol use, and HPV were 187, 130, and 11, respectively (Table 2).

By state, in 2013, age-adjusted incidence rates for cancers of all sites (all-sites cancer) ranged from 364 per 100,000 persons in New Mexico to 512 per 100,000 persons in Kentucky (Table 2). State site-specific cancer incidence rates for prostate cancer ranged from 69 (Arizona) to 131 (Louisiana) per 100,000 males; for female breast cancer, from 105 (Wyoming) to 148 (New Hampshire) per 100,000 females; for lung cancer, from 26 (Utah) to 93 (Kentucky) per 100,000 persons; for colorectal cancer, from 32 (Arizona, New Mexico, Utah, and Vermont) to 49 (Kentucky and Mississippi) per 100,000 persons; and for cervical cancer, from four (New Hampshire and Montana) to 11 (Arkansas) per 100,000 females (Table 2). The *Healthy People 2020* target for reducing colorectal cancer incidence to ≤39.9 per 100,000 persons was reached in 30 states and the target for reducing cervical cancer incidence to ≤7.2 per 100,000 females was reached in 28 states. For grouped cancers, incidence rates for tobacco-related cancers ranged by state from 128 (Utah) to 245 (Kentucky) per 100,000 persons; for alcohol-related cancers, from 107 (Wyoming) to 146 (Kentucky) per 100,000 persons; and for HPV-related cancers, from seven (Utah) to 14 (Arkansas and Kentucky) per 100,000 persons (Table 2). Compared with the states and DC, cancer incidence rates in Puerto Rico in 2013 were lower for all-sites cancer (359 per 100,000 persons), lung cancer (18 per 100,000 persons), and female breast cancer (96 per 100,000 females), but higher for prostate cancer (145 per 100,000 males), colorectal cancer (42 per 100,000 persons), and cervical cancer (12 per 100,000 females) (Table 2).

Among persons with cancer diagnosed during 2006–2012, the 5-year relative survival proportion was 67% (Table 3). This proportion was similar for males and females. The 5-year relative survival proportion was highest among persons who received a diagnosis of cancer before age 45 years (83%) and decreased with increasing age (Table 3). Among the four most common cancer sites, the 5-year relative survival proportion was highest for prostate cancer (99%) and female breast cancer (90%), intermediate for colorectal cancer (66%), and lowest

TABLE 2. Annual age-adjusted rate* of invasive cancers,† by cancer site and state — National Program of Cancer Registries and Surveillance, Epidemiology, and End Results program, United States, 2013

State	Cancer site						Cancers associated with certain risk factors§		
	All sites	Lung and bronchus	Colon and rectum	Prostate	Female breast	Cervix	Tobacco use	Alcohol use	HPV
Alabama	444.0	66.7	44.0	118.5	120.6	8.5	202.5	137.3	12.5
Alaska	410.4	54.7	43.0	76.6	120.5	6.6	187.2	130.0	11.6
Arizona	370.6	48.4	32.1	69.1	110.9	6.3	159.2	112.8	8.7
Arkansas	454.0	78.7	43.1	102.7	118.2	10.6	215.4	133.4	14.4
California	402.8	42.6	35.1	97.5	120.9	7.0	159.9	123.4	10.0
Colorado	396.1	42.2	33.6	101.6	123.6	5.7	152.3	120.5	9.2
Connecticut	474.2	62.3	36.3	104.6	138.4	7.3	196.1	136.4	11.6
Delaware	502.0	69.1	34.9	129.4	144.8	6.7	203.8	142.5	11.9
District of Columbia (DC)	445.2	55.3	41.4	120.1	138.2	8.3	181.4	144.0	13.0
Florida	413.0	58.8	35.8	86.4	114.1	8.4	179.0	123.4	13.4
Georgia	450.3	64.0	40.7	117.7	123.2	6.9	190.3	133.5	12.1
Hawaii	419.8	49.3	41.8	79.2	143.9	6.5	176.1	144.7	8.5
Idaho	419.5	46.9	35.1	101.4	119.4	5.2	169.1	120.8	10.5
Illinois	454.9	63.2	43.0	105.3	130.1	7.2	200.1	138.4	11.6
Indiana	438.8	71.6	42.1	85.7	120.4	7.6	205.2	132.0	12.4
Iowa	456.1	62.0	42.9	96.9	118.4	5.7	195.3	132.2	11.6
Kansas	450.9	61.8	38.6	108.5	115.6	7.1	189.6	125.3	10.8
Kentucky	511.7	93.4	49.0	104.8	123.2	7.9	244.5	146.1	14.1
Louisiana	476.3	68.0	45.0	131.2	124.6	8.2	215.0	142.6	12.7
Maine	463.8	74.8	37.4	80.7	126.0	5.9	203.0	130.1	12.4
Maryland	451.0	56.6	35.8	124.4	134.1	5.9	180.3	133.6	9.8
Massachusetts	457.5	62.6	36.4	97.2	137.2	4.9	192.7	137.7	10.8
Michigan	440.1	62.4	36.7	102.8	124.8	6.7	189.0	128.0	10.8
Minnesota	451.8	56.6	38.7	101.6	127.9	5.3	180.6	129.9	9.4
Mississippi	459.9	75.2	48.7	127.2	112.3	8.1	218.7	139.2	13.0
Missouri	442.6	73.7	41.0	84.7	124.9	7.7	204.9	134.8	12.4
Montana	437.0	58.0	38.8	108.0	109.4	4.1	180.0	119.1	9.0
Nebraska	437.6	60.1	39.9	106.1	118.7	7.4	184.3	125.1	10.1
Nevada	—¶	—¶	—¶	—¶	—¶	—¶	—¶	—¶	—¶
New Hampshire	479.2	64.8	37.0	115.6	148.4	3.6	192.0	140.3	10.2
New Jersey	483.1	57.5	41.9	123.4	135.5	7.5	193.1	139.6	10.9
New Mexico	363.7	39.6	32.1	80.9	112.6	7.2	148.3	115.7	9.0
New York	484.3	59.5	39.3	125.6	130.3	8.0	196.2	135.7	11.2
North Carolina	445.4	68.5	36.3	107.8	126.1	6.6	194.5	131.4	11.7
North Dakota	433.6	56.4	46.0	100.5	125.5	—**	183.8	132.3	8.8
Ohio	452.4	67.4	40.6	101.7	125.8	7.4	200.3	134.3	12.1
Oklahoma	440.3	68.7	42.5	95.0	117.0	9.5	203.0	132.7	13.0
Oregon	431.5	55.6	34.5	88.7	124.8	6.7	179.9	126.8	12.1
Pennsylvania	483.0	64.3	42.3	101.2	130.8	7.1	204.6	139.8	11.8
Rhode Island	479.4	69.9	35.4	91.1	137.8	7.4	206.5	136.3	12.1
South Carolina	436.9	64.4	36.1	105.8	124.5	7.7	191.3	130.7	13.2
South Dakota	450.1	59.4	40.8	102.1	146.1	7.3	183.3	139.1	11.5
Tennessee	450.9	74.1	38.2	106.2	124.6	8.9	203.0	133.4	13.3
Texas	399.4	52.7	37.4	88.6	108.4	8.7	177.5	122.4	10.5
Utah	393.2	26.1	32.0	111.9	111.0	4.8	128.1	107.5	7.2
Vermont	437.1	59.1	31.7	81.7	125.6	5.7	176.7	123.5	10.4
Virginia	418.5	58.2	35.4	101.0	128.3	5.8	175.5	128.3	10.0
Washington	450.3	55.0	35.3	107.3	135.3	6.7	179.9	132.8	10.8
West Virginia	464.0	79.1	47.0	90.3	116.5	8.1	223.0	137.1	12.9
Wisconsin	451.1	59.0	36.4	103.4	128.6	5.5	187.5	129.5	9.7
Wyoming	382.0	38.7	32.5	97.9	105.0	6.0	145.9	106.7	8.3
Puerto Rico (PR)	358.5	18.2	42.0	144.9	95.8	12.3	124.3	117.4	12.3
States + DC + PR	439.0	58.8	38.5	102.1	123.4	7.3	186.2	130.2	11.3
States + DC	438.0	59.4	38.4	101.6	123.7	7.2	187.0	130.4	11.2

See table footnotes on page 73.

TABLE 2. (*Continued*) Annual age-adjusted rate* of invasive cancers,† by cancer site and state — National Program of Cancer Registries and Surveillance, Epidemiology, and End Results program, United States, 2013

Abbreviation: HPV = human papillomavirus.
* Age-adjusted to the 2000 U.S. standard population. Rates are per 100,000 persons except per 100,000 males for prostate cancer and per 100,000 females for female breast and cervical cancers.
† Excludes basal and squamous cell carcinomas of the skin, except when these occur on the skin of the genital organs, and in situ cancers, except urinary bladder.
§ Tobacco-associated cancers include oral cavity and pharynx; esophagus; stomach; colon and rectum; liver; pancreas; larynx; lung, bronchus, and trachea; cervix; kidney and renal pelvis; urinary bladder; and acute myeloid leukemia. Alcohol-associated cancers include oral cavity and pharynx; esophagus; colon and rectum; liver; larynx; and female breast. HPV-associated cancers include microscopically confirmed carcinoma of the cervix and squamous cell carcinomas of the vagina, vulva, penis, anus, rectum, and oropharynx.
¶ Rate not shown because data did not meet publication criteria.
** Rate not shown because <16 cases were reported.

TABLE 3. Percentage of patients with five-year relative survival after cancer diagnosis,* by race, sex, cancer site, and age group — National Program of Cancer Registries, 29 States, 2006–2012†

	Survival (%)								
	All races			Whites			Blacks		
Cancer site/Age group	Overall	Males	Females	Overall	Males	Females	Overall	Males	Females
All sites	**67**	**67**	**67**	**67**	**67**	**68**	**62**	**64**	**59**
Prostate	NA	99	NA	NA	99	NA	NA	97	NA
Female breast	NA	NA	90	NA	NA	91	NA	NA	80
Lung and bronchus	19	17	23	19	17	23	17	14	20
Colon and rectum	66	65	66	66	66	67	59	58	61
Cervix uteri	NA	NA	69	NA	NA	70	NA	NA	59
Age group (yrs)§									
<45	83	78	85	84	80	87	72	67	75
45–54	74	69	78	75	70	80	65	63	66
55–64	71	70	71	71	70	72	64	67	61
65–74	66	69	63	67	69	64	62	67	54
≥75	53	56	50	53	55	51	46	52	42

Abbreviation: NA = not applicable.
* Based on cases of cancer diagnosed during 2006–2012 and follow-up of patients through 2012.
† Compiled from data from 29 cancer registries that met data quality criteria for survival analysis, covering approximately 66% of the U.S. population.
§ Age at cancer diagnosis.

for lung cancer (19%) (Table 3). The 5-year relative survival proportion after any cancer diagnosis was lower among blacks (62%) than among whites (67%), particularly among black females (59%) compared with white females (68%) (Table 3).

Discussion

This report provides estimates of cancer incidence for 2013 in the United States and indicates that national *Healthy People 2020* targets were achieved in 30 states for reduced colorectal cancer incidence and 28 states for reduced cervical cancer incidence. Many cancers could be prevented by implementing evidence-based interventions to reduce cancer risk factors, promote healthy living, and encourage appropriate cancer screening (*6*).

Some cancer risk factors can be addressed through clinical preventive services. As of 2016, the U.S. Preventive Services Task Force recommends that all adults be screened for tobacco use and excessive alcohol use and offered counseling and intervention as needed.¶¶ The U.S. Preventive Services Task Force also recommends the use of low-dose aspirin to prevent colorectal cancer and cardiovascular disease among adults who are considered to be at high risk for cardiovascular disease based on specific criteria (*7*). As of 2016, the Advisory Committee on Immunization Practices recommends vaccination against two cancer-causing viruses, HPV and hepatitis B virus.*** Health care providers play an important role in ensuring that all children, adolescents, and adults receive the preventive services they need at the appropriate time. The Community Preventive Services Task Force offers evidence-based recommendations to increase both patient and provider adherence to guidelines for preventive services as well as community-based approaches to promote physical activity, reduce excessive alcohol use, and reduce tobacco use and tobacco smoke exposure.†††

Cancer incidence and survival data are important for guiding the planning and evaluation of cancer prevention and control programs at the national and local levels. For example, Pennsylvania Cancer Registry data were used to guide community outreach programs in areas with cancer-related health disparities and evaluate the impact of cancer interventions (*8*). These data also assist long-term planning for cancer diagnostic

¶¶ https://www.uspreventiveservicestaskforce.org/Page/Name/recommendations.

*** https://www.cdc.gov/vaccines/hcp/acip-recs/vacc-specific/hpv.html; https://www.cdc.gov/vaccines/hcp/acip-recs/vacc-specific/hepb.html.
††† https://www.thecommunityguide.org/topic/cancer.

and treatment services. By examining prostate cancer treatment data, the New Hampshire State Cancer Registry found promising trends in the management of prostate cancer, with increasing use of surgical procedures for men at high risk for the disease and less aggressive treatment for men at low risk for the disease (*9*). Finally, these data help public health officials set priorities for allocating health resources. The Oregon Health Authority's decision to increase measures to improve HPV vaccination coverage was based in part on data from the Oregon State Cancer Registry that indicated a recent increase in HPV-associated cancer incidence had occurred (*10*).

CDC annually provides cancer surveillance data via several products such as USCS, CDC WONDER, State Cancer Facts, and the CDC Chronic Disease Indicators web tool, and through the CDC's National Center for Health Statistics Research Data Center.[§§§] Although cancer mortality data sets formatted for use with NCI SEER*Stat software have been available since 2003, for the first time, CDC is releasing a public use NPCR cancer incidence data set that can be analyzed using the NCI SEER*Stat software; information about this data set, including variable formats for cancer groups related to tobacco use, alcohol use, HPV, obesity, and physical activity, is available online.[¶¶¶]

The findings in this report are subject to at least four limitations. First, analyses based on race/ethnicity might be biased if race/ethnicity was systematically misclassified; ongoing procedures are used to ensure that this information is as accurate as possible (*4*). Second, delays in cancer reporting might result in an underestimation of certain cancers; reporting delays are more common for cancers such as melanoma and prostate cancer that are diagnosed and treated in nonhospital settings such as physicians' offices. Third, relative survival could only be calculated for white and black racial groups because accurate life tables were not available for other racial/ethnic groups. Finally, because information about risk factors is not routinely collected by cancer registries, estimates for risk factor–associated cancers depict the number potentially associated, not the number definitively attributable.

Public health officials use population-based cancer incidence, mortality, and survival surveillance data to plan and monitor programs, conduct clinical outcomes research, help make decisions about allocating resources, and hold recipients of state and federal appropriations accountable. To achieve the national cancer objectives set forth in *Healthy People 2020*, initiatives to promote healthy living, reduce exposure to cancer risk factors, improve adherence to cancer screening recommendations, and assure timely and appropriate clinical preventive services for all persons should be maximized. The effects of these initiatives can be monitored using cancer surveillance data.

Summary

What is already known about this topic?

Some risk factors, such as tobacco use, alcohol use, excess body weight, physical inactivity, and human papillomavirus (HPV) infection increase the risk for more than one type of cancer.

What is added by this report?

Based on the U.S. Cancer Statistics dataset, in 2013 (the most recent year for which incidence and survival data are available), a total of 1,559,130 new invasive cancers were diagnosed in the United States (excluding Nevada), for an annual incidence of 479 per 100,000 males and 413 per 100,000 females. All-sites cancer incidence rates ranged, by state, from 364 to 512 per 100,000 persons, and the rate was 359 per 100,000 persons in Puerto Rico. *Healthy People 2020* targets for reducing incidence rates were reached in 30 states for colorectal cancer and 28 states for cervical cancer. Approximately two of three persons survived ≥5 years after cancer diagnosis.

What are the implications for public health practice?

Cancer surveillance data are key to cancer epidemiologic and clinical outcomes research, program planning and monitoring, resource allocation, and state and federal appropriations accountability. To achieve the national cancer objectives set forth in *Healthy People 2020,* initiatives to promote healthy living, reduce exposure to cancer risk factors, improve adherence to cancer screening recommendations, and assure timely and appropriate clinical preventive services and treatment for all persons must be maximized. The impact of these initiatives can be monitored using publicly available cancer surveillance data.

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[1]Division of Cancer Prevention and Control, National Center for Chronic Disease Prevention and Health Promotion, CDC.

Corresponding author: S. Jane Henley, shenley@cdc.gov, 770-488-4157.

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Cluster of an Unusual Amnestic Syndrome — Massachusetts, 2012–2016

Jed A. Barash, MD[1]; Nick Somerville, MD[2]; Alfred DeMaria, Jr., MD[3]

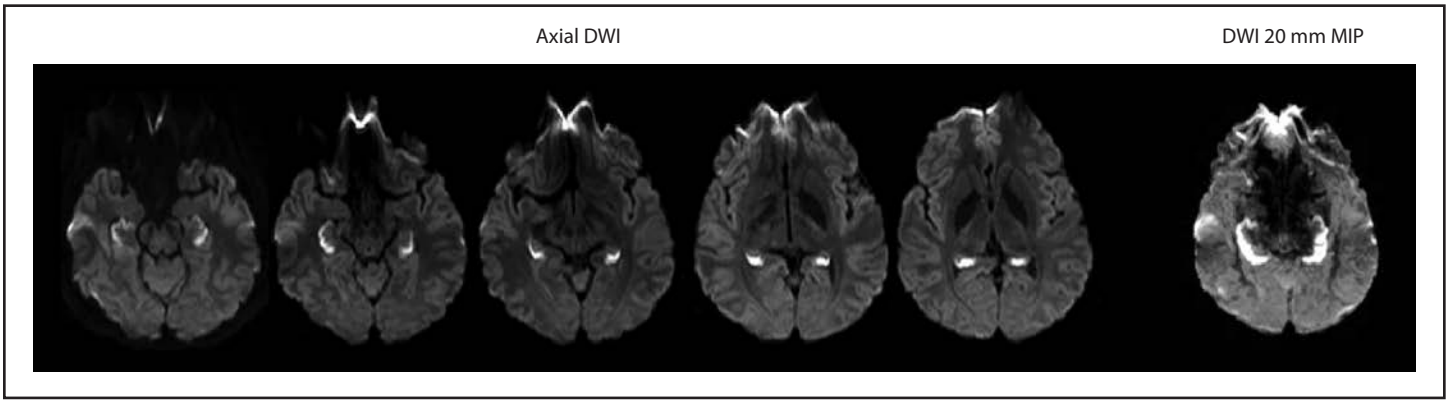
In November 2015, a neurologist in the Boston, Massachusetts, area reported four cases of an uncommon amnestic syndrome involving acute and complete ischemia of both hippocampi, as identified by magnetic resonance imaging (MRI), to the Massachusetts Department of Public Health (MDPH) (*1*). A subsequent e-mail alert, generated by the Massachusetts Board of Registration in Medicine and sent to relevant medical specialists (including neurologists, neuroradiologists, and emergency physicians), resulted in the identification of 10 additional cases that had occurred during 2012–2016. All 14 patients (mean and median age = 35 years) had been evaluated at hospitals in eastern Massachusetts. Thirteen of the 14 patients underwent routine clinical toxicology screening at the time of initial evaluation; eight tested positive for opioids, two for cocaine, and two for benzodiazepines. Apart from sporadic cases (*2–6*), this combination of clinical and imaging findings has been reported rarely. The apparent temporospatial clustering, relatively young age at onset (19–52 years), and associated substance use among these patients should stimulate further case identification to determine whether these observations represent an emerging syndrome related to substance use or other causes (e.g., a toxic exposure).

The four patients reported in November 2015 had been evaluated at a single Boston-area medical center during the preceding 3 years (*1*). MRI of the head revealed changes consistent with acute and complete ischemia of both hippocampi (Figure) in all four patients at the time of initial evaluation. Three of the four patients tested positive for opiates on initial toxicology screening, and the fourth, who was not tested, had a reported history of heroin use. No readily apparent evidence for another established etiology of hippocampal amnesia (*7*,*8*) existed for any of the patients. Several previous isolated case reports were associated with cocaine use only (*2–4*), and one case of complete unilateral hippocampal infarction involving heroin inhalation was reported in France in 2013 (*9*).

In February 2016, MDPH requested that neurologists, radiologists (including neuroradiologists), and emergency department physicians report any similar cases for medical record review. For the purposes of the review, a case was defined as a patient evaluated in Massachusetts since January 1, 2012 with 1) new onset amnesia in the absence of evidence to support a readily apparent cause, and 2) changes consistent with acute and complete ischemia of both hippocampi on MRI at initial assessment. To investigate each report, the authors (including a board-certified neurologist) reviewed available clinical documentation and diagnostic testing. After preliminary case review, demographic, behavioral, and clinical data, including information related to substance use, were abstracted for analysis from records of patients who met the case definition.

Including the four initial cases, medical records of 25 patients, dating back to 2008, were reviewed after the February 2016 request by MDPH for case reporting. Medical testing was not uniform among all patients, because each patient underwent variable and extensive testing based on clinical context and the assessment of their health care provider. Fourteen

FIGURE. Diffusion-weighted imaging (DWI) findings at initial brain magnetic resonance imaging in a patient with unusual amnestic syndrome* — Massachusetts, 2012



Source: Adapted from: Small JE, Butler PM, Zabar Y, Barash JA. Complete, bilateral hippocampal ischemia: a case series. Neurocase 2016;22:411–5. Reprinted with permission of the publisher, Abingdon, (OX), United Kingdom: Taylor & Francis, Ltd. http://www.tandfonline.com.
Abbreviations: MIP = maximum intensity projection; mm = millimeter.
* Axial DWI demonstrates bright signal consistent with complete bilateral hippocampal ischemia. The complete extent of hippocampal ischemia is best evident on thick 20 mm MIP images constructed from the axial DWI data.

(56%) patients met the case definition (Table 1). Among these 14, a total of 11 were identified retrospectively, including two in 2012, five in 2014, and four in 2015. Three were identified prospectively in 2016 after the MDPH request, the most recent in late July 2016. None of the reports of patients with onset before 2012 met the clinical case definition.

All 14 patients had been evaluated at hospitals in eastern Massachusetts. One was a resident of southeastern New Hampshire and another was visiting from the state of Washington. Patient age ranged from 19 to 52 years (mean and median = 35 years). Nine patients were unconscious at the time they came to medical attention, five of whom required endotracheal intubation. After regaining consciousness, all nine were noted to be amnestic. Among the other five patients, family members, friends, or acquaintances observed the emergence of severe memory loss after limited time apart and brought them to the emergency department for further assessment. In addition to striking anterograde amnesia, deficits of orientation, attention, and executive function were variously noted. These deficits were reported to have improved over time, with resolution of memory loss in one patient at 5 months, but persisting in two patients with follow-up of more than 1 year (Table 2). For 13 patients, MRI of the head was performed within 5 days of initial evaluation, and at 8 days in the 14th patient. In addition to bilateral hippocampal ischemia (Figure), nine patients also exhibited ischemic changes in one or more, often asymmetric extra-hippocampal regions, primarily in the subcortical and posterior areas (Table 2). Follow-up MRI in one patient, at 5 weeks, demonstrated complete resolution of the initial abnormalities; in two other patients, at 13 and 22 months after onset, MRI revealed residual, bilateral hippocampal volume loss.

A history of substance use disorder was documented in 13 of 14 patients; the remaining patient tested positive for opiates and cocaine at the time of initial evaluation. The other patient, who tested positive for cocaine, also tested positive for opiates, amphetamines, and benzodiazepines, none of which were being prescribed at the time. Overall, 12 of 14 patients had a history of opioid use, and eight tested positive for opiates on routine toxicology screening, including one whose medication list included oxycodone-acetaminophen and another who had not reportedly filled a prescription for buprenorphine/naloxone in approximately 2 months. Among the six patients with a history of benzodiazepine use, four had lorazepam or clonazepam on their medication list, and two tested positive for benzodiazepines. Tobacco and alcohol histories were incompletely documented for multiple patients, although no patient tested positive for alcohol on routine screening. One of the two patients with negative toxicology results upon routine testing had reported heroin use in the days preceding the event, and the other had a history of opioid use, but further details were unavailable. Marijuana, lysergic acid diethylamide (LSD), 3,4-methylenedioxymethamphetamine (MDMA, commonly known as Ecstasy), mushrooms, and phencyclidine (PCP) were among other substances reported to have been used (Table 2). Neither of the patients with a history of dextroamphetamine/amphetamine use had amphetamines listed as a prescribed medication. Among four patients with gabapentin on their active medication list, one reportedly had evidence of gabapentin overdose at the time of evaluation. Routine clinical toxicology screening in that patient also revealed the presence of opiates, cannabinoids, and salicylates.

TABLE 1. Selected characteristics of 14 patients with sudden-onset amnesia and complete hippocampal ischemia of unclear etiology — Massachusetts, June 2012–July 2016

Characteristic	No. (%)
Age group (yrs)	
19–30	6 (43)
31–40	2 (14)
41–52	6 (43)
Male sex	**10 (71)**
Reported history of substance use	**13 (93)**
Opioids*	12 (86)
Benzodiazepines†	6 (43)
Marijuana	6 (43)
Cocaine	5 (36)
Amphetamines (dextroamphetamine/amphetamine)	2 (14)
Lysergic acid diethylamide (LSD)	2 (14)
3,4-methylenedioxymethamphetamine (MDMA)	1 (7)
Mushrooms	1 (7)
Phencyclidine (PCP)	1 (7)
Toxicology screening (blood and/or urine) done	**13 (93)**
Any positive results	11 (85)
Opiates	8 (62)
Marijuana/Cannabinoids	4 (31)
Cocaine	2 (15)
Benzodiazepines	2 (15)
Amphetamines	1 (8)
Barbiturates§	1 (8)
Salicylates	1 (8)
Multiple substances	5 (38)

* One patient had oxycodone/acetaminophen prescribed.
† Four patients had a benzodiazepine prescribed.
§ Patient had butalbital/acetaminophen/caffeine prescribed.

One patient had a history of seizures on two occasions in the past, possibly related to alcohol withdrawal, but no evidence of seizure at the time of assessment. Another patient had witnessed seizure activity during transport to the emergency department, but had no history of seizures. A third patient developed a seizure disorder after evaluation for the amnestic episode. No epileptiform abnormalities were noted on electroencephalography (EEG) at the time of initial evaluation in these three patients or in eight others who underwent EEG.

Six patients had history of at least one vascular disease risk, including hypertension, dyslipidemia, diabetes, and sleep apnea. Echocardiogram performed in six patients, and vessel imaging of

TABLE 2. Selected characteristics of 14 patients with sudden-onset amnesia and complete hippocampal ischemia of unclear etiology, by onset year — Massachusetts, June 2012–July 2016

Onset year	Age (yrs)	Sex	Substance abuse disorder history	Positive toxicology results	Locations of extra-hippocampal signal abnormalities on MRI	Clinical follow-up
2012	27	M	Opioids, marijuana	Opiates	None	Not available
2012	22	M	Opioids, marijuana, LSD, MDMA, cocaine	Opiates	None	At 22 months, residual mildly impaired attention and storage, variable processing speed
2014	49	M	None reported	Opiates, cocaine	Occipital lobe	Not available
2014	21	M	Opioids	Marijuana	Basal ganglia, fornix, midbrain, cerebellum, temporal lobe	Not available
2014	51	F	Opioids, marijuana, cocaine	Opiates,* cannabinoids, salicylates	Cerebellum, occipital lobe	Not available
2014	33	F	Opioids (benzodiazepine prescribed)	Opiates, benzodiazepine	Basal ganglia	At 13 months: moderate short-term memory loss, mild inattention and executive dysfunction (for visuospatial and language tasks)
2014	41	M	Opioids	Not performed	None	At 8 weeks: severe short-term memory loss, mildly diminished working memory; at 9 months: died from cardiac arrest
2015	46	M	Opioids (benzodiazepine prescribed)	Negative	None	Not available
2015	19	M	Marijuana, LSD, mushrooms, amphetamine/dextroamphetamine	Cannabinoids	Cerebellum	At 5 months: short-term memory loss resolved; persistent seizure disorder
2015	52	F	Opioids, cocaine (benzodiazepine prescribed)	Opiates, barbiturates†	Basal ganglia	Not available
2015	36	M	Opioids	Negative	Basal ganglia, corpus callosum, centrum semiovale, occipital lobe, cerebellum	Not available
2016	21	F	Opioids, cocaine, benzodiazepine, marijuana	Opiates	Basal ganglia	Not available
2016	22	M	Opioids, benzodiazepine, marijuana (benzodiazepine prescribed)	Marijuana	None	Not available
2016	50	M	Opioids, benzodiazepine, PCP, cocaine, amphetamine/ dextroamphetamine	Amphetamines, benzodiazepine, cocaine, opiates	Parietal lobe	Not available

Abbreviations: F = female; LSD = lysergic acid diethylamide; M = male; MDMA = 3,4-methylenedioxymethamphetamine; MRI = magnetic resonance imaging; PCP = phencyclidine.
* Patient had oxycodone/acetaminophen prescribed.
† Patient had butalbital/acetaminophen/caffeine prescribed.

the head and neck performed in seven patients, did not reveal a source of thromboembolism. Electrocardiogram revealed a new diagnosis of atrial fibrillation in the two oldest patients (aged 50 and 52 years). One patient aged 36 years demonstrated pulseless electrical activity and respiratory arrest (after a documented brief response to naloxone), with resolution on prehospital resuscitation. Cerebrospinal fluid findings in five patients who underwent lumbar puncture were unremarkable. Carboxyhemoglobin and methemoglobin levels were measured in two patients and were unremarkable. Initial aspartate and alanine aminotransferase were elevated in all 13 patients tested, with both levels in one patient exceeding 500 units/liter (approximately 10 times the upper limit of normal). Otherwise, extensive work-up was unremarkable. Investigation of the 14 cases is ongoing.

Summary

What is already known about this topic?

Acute, complete, and bilateral ischemia of the hippocampus is a rare cause of memory loss (associated with toxic exposure, among other etiologies) that has been reported rarely and in isolation. A single 2013 case of complete unilateral hippocampal ischemia has been linked to heroin inhalation.

What is added by this report?

A unique cluster of 14 cases of sudden onset amnesia with acute, complete, and bilateral ischemia of the hippocampus was identified in Massachusetts during 2012–2016. No clear etiology exists, but at time of initial evaluation, 13 of 14 tested positive for opioids or had opioid use recorded in their medical history.

What are the implications for public health practice?

The apparent temporospatial clustering, relatively young age at onset (19–52 years), and extensive substance use associated with this group of patients suggests broader surveillance is needed to determine whether this represents an emerging syndrome related to substance use or other causes, including introduction of a toxic substance.

Discussion

The combination of clinical findings described in this report has previously been reported rarely and in isolation, associated with isolated cocaine use, influenza, and carbon monoxide poisoning (*2*–*6*). This cluster of amnestic syndrome associated with bilateral complete hippocampal ischemia is unusual given the absence of a readily identifiable etiology, the temporospatial clustering, relatively young patient age, and extensive substance use among affected persons.

Cardiopulmonary, cerebrovascular, or other mechanisms might serve as plausible explanations underlying certain findings. Hypoxemic injury to the relatively vulnerable hippocampal regions, for example, has been raised as one possibility (*10*). However, further case identification and reporting are needed to determine whether these combined observations represent an emerging syndrome related to substance use or other causes (e.g., a toxic exposure).

The findings in this report are subject to at least three limitations. First, information was obtained from medical records from several different facilities, and differences in documentation and medical assessment across patients limited the consistent characterization of variables. Second, this investigation was intended to establish the existence of the case cluster and generate hypotheses about possible associated exposures. A case-control study could more rigorously test potential associations. Finally, the identification of cases required that MRI of the head had been performed during patient work-up, which might not be consistently performed by medical providers for various reasons.

MRI of the head, toxicology screening, and neurologic consultation should be considered in all adults aged ≥18 years with sudden-onset amnesia, particularly in patients with altered consciousness. Advanced laboratory testing, including testing for synthetic opioids (e.g., fentanyl) and their analogues, as well as extraneous substances not assessed in these reported cases, might further clarify an association with substance use.

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[1]Lahey Hospital and Medical Center, Burlington, Massachusetts and Soldiers' Home, Chelsea, Massachusetts; [2]Epidemic Intelligence Service, CDC; [3]Massachusetts Department of Public Health, Jamaica Plain, Massachusetts.

Corresponding author: Alfred DeMaria, Jr., alfred.demaria@state.ma.us, 617-983-6550.

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Prevalence of Pelvic Inflammatory Disease in Sexually Experienced Women of Reproductive Age — United States, 2013–2014

Kristen Kreisel, PhD[1]; Elizabeth Torrone, PhD[1]; Kyle Bernstein, PhD[1]; Jaeyoung Hong, PhD[1]; Rachel Gorwitz, MD[1]

Pelvic inflammatory disease (PID) is a clinical syndrome of the female reproductive tract characterized by inflammation of the endometrium, fallopian tubes, or peritoneum (*1*). PID occurs when microorganisms ascend from the vagina or cervix to the fallopian tubes and other upper genital tract structures (*1*). PID can result from untreated bacterial infections, including chlamydia and gonorrhea, and can lead to infertility, ectopic pregnancy, and chronic pelvic pain (*1*). Because there is no single diagnostic test for PID, clinicians rely on nonspecific signs and symptoms for diagnosis. The purpose of these analyses was to assess the burden of self-reported PID in a nationally representative sample using data from the National Health and Nutrition Examination Survey (NHANES) 2013–2014 cycle. Starting in 2013, NHANES female participants aged 18–44 years were asked about a lifetime history of PID diagnosis. Based on these data, the estimated prevalence of self-reported lifetime PID was 4.4% in sexually experienced women of reproductive age (18–44 years). The prevalence of self-reported lifetime PID was highest in women at increased risk, such as women reporting a previous sexually transmitted infection (STI) diagnosis. Stratified by race/ethnicity and having a previous STI diagnosis, non-Hispanic black (black) and non-Hispanic white (white) women reporting a previous STI diagnosis had nearly equal self-reported lifetime PID prevalence (10.0% versus 10.3%). However, the lifetime prevalence of PID among black women was 2.2 times that among white women if no previous STI was diagnosed (6.0% versus 2.7%). These findings suggest that PID is prevalent and associated with previous STI diagnoses; therefore, it is important for clinicians to screen female patients for chlamydia and gonorrhea to reduce the incidence of PID.

NHANES is a cross-sectional, complex, multistage survey designed to be nationally representative of the noninstitutionalized U.S. civilian population (https://www.cdc.gov/nchs/nhanes.htm). Participants undergo a medical examination and are interviewed in person, during which time questions regarding sexual and reproductive health are asked. In NHANES 2013–2014, a total of 1,444 women aged 18–44 years were interviewed and had a medical exam; the response rate was 71.1%. The 1,171 (81%) reproductive-aged female participants who responded "Yes" to the question, "Have you ever had vaginal, anal, or oral sex?" were defined as sexually experienced and were the focus of these analyses. Participants who responded "Yes" to the question, "Have you ever been treated for an infection in your fallopian tubes, uterus or ovaries, also called a pelvic infection, pelvic inflammatory disease, or PID?" met the case definition of a lifetime PID diagnosis. Having received a diagnosis of a previous STI was defined as having had a chlamydia or gonorrhea infection during the past 12 months or ever having had herpes, human papillomavirus, or genital warts. The prevalence of self-reported lifetime PID, prevalence ratios (PRs), and 95% confidence intervals (CIs) were estimated overall and by various characteristics. Associations were measured by use of the Rao-Scott chi-square test. All analyses were conducted using SAS statistical software (version 9.3) and accounted for the complex survey design and sampling weights. As such, these results are nationally representative. The mobile examination center exam sampling weights were used to weight the data. Population counts were estimated by multiplying weighted prevalence estimates by the average of the American Community Survey estimates during 2013–2014.

Among 1,171 sexually experienced reproductive-aged women in NHANES 2013–2014, the prevalence of self-reported lifetime PID was 4.4% (Table), indicating that approximately 2.5 million women aged 18–44 nationwide have received a diagnosis of PID in their lifetime (95% CI = 1.8–3.2 million). No significant differences existed in prevalence of a lifetime PID diagnosis by age, race/ethnicity, or socioeconomic factors, such as income-poverty ratio, current health insurance coverage, or having a current usual place for health care.

Significant differences in the prevalence of lifetime PID were observed by the sexual behaviors and sexual health histories of respondents. The prevalence of self-reported lifetime PID among women whose age of sexual debut was <12 years was approximately eight times that of women whose age of sexual debut was ≥18 years (PR = 8.6). Similarly, the lifetime PID prevalence among women with ≥10 lifetime male vaginal sex partners was approximately three times that of women with a single partner (PR = 3.6). The prevalence of lifetime PID was approximately double in women reporting lesbian/bisexual versus heterosexual orientation (PR = 2.1), and the prevalence among women reporting a previous STI diagnosis was approximately three times that of women without a previous STI diagnosis (PR = 3.3).

In stratified analyses (Figure), the prevalence of self-reported lifetime PID among women reporting a previous STI diagnosis was similar in whites and blacks (10.0% [95% CI = 4.4–15.6] versus 10.3% [95% CI = 1.3–19.4], p = 0.97).

TABLE. Prevalence of self-reported lifetime pelvic inflammatory disease* among sexually experienced women† aged 18–44 years (n = 1,171), by selected characteristics — National Health and Nutrition Examination Survey, United States, 2013–2014.

Characteristic	Sample size no.	Prevalence (%)§ (95% CI)	Prevalence ratio¶ (95% CI)
Total	**1,171**	**4.4 (3.1–5.7)**	**— (—)**
Age group (yrs) (p = 0.28)**			
18–24	327	2.9†† (0.8–5.0)	Ref (—)
25–29	185	4.6†† (1.4–7.9)	1.6 (0.6–4.1)
30–34	212	5.0†† (1.8–8.2)	1.7 (0.7–4.3)
35–39	209	3.5 (1.5–5.4)	1.2 (0.5–2.7)
40–44	238	6.7 (2.6–10.8)	2.3 (0.8–6.6)
Race/Ethnicity (p = NC)**			
White, non-Hispanic	436	4.4 (2.8–6.0)	Ref (—)
Black, non-Hispanic	245	6.8 (4.0–9.5)	1.5 (0.9–2.5)
Asian, non-Hispanic	130	0.0 (—)	— (—)
Mexican American	195	—§§ (—§§)	—§§ (—§§)
Education level (p = 0.21)**			
Less than high school	212	4.3†† (1.0–7.6)	Ref (—)
High school graduate/GED	243	3.1†† (1.1–5.2)	0.7 (0.3–1.8)
Some college/Associates degree	430	6.2 (3.2–9.2)	1.5 (0.6–3.7)
College graduate or above	286	3.0¶¶ (0.4–5.7)	0.7 (0.2–2.9)
Marital status (p = 0.56)**			
Married/Living with partner	618	5.0 (2.5–7.6)	Ref (—)
Divorced/Separated/Widowed	120	5.3¶¶ (0.6–9.9)	1.0 (0.4–3.0)
Never married	309	3.5 (1.3–5.8)	0.7 (0.3–1.9)
Income-poverty ratio* (p = 0.50)****			
<150% FPL	500	5.1 (2.9–7.3)	Ref (—)
150%–299% FPL	244	4.7†† (1.0–8.4)	0.9 (0.3–2.5)
≥300% FPL	357	3.2†† (0.9–5.4)	0.6 (0.3–1.5)
Health insurance coverage (p = 0.20)**			
Covered	860	4.0 (2.5–5.5)	Ref (—)
Not covered	303	6.1 (2.6–9.6)	1.5 (0.7–3.2)
Has a usual place for health care (p = 0.87)**			
Yes	952	4.4 (2.6–6.1)	Ref (—)
No	219	4.7†† (1.2–8.3)	1.1 (0.4–2.9)
Type of place for usual health care (p = NC)**			
Doctor's office/HMO	655	3.8 (1.8–5.9)	Ref (—)
Clinic/health center	219	6.0†† (1.2–10.9)	1.6 (0.6–4.2)
Hospital outpatient department/ED	63	—§§ (—§§)	—§§ (—§§)
Age at sexual debut, in years (p = 0.0002)**			
≥18	475	2.7 (1.2–4.2)	Ref (—)
16–17	371	4.6 (1.7–7.5)	1.7 (0.7–4.3)
14–15	228	4.9 (2.5–7.4)	1.8 (0.9–3.6)
12–13	79	8.9 (3.3–14.5)	3.4 (1.4–8.5)
<12	18	23.6¶¶ (0.9–46.2)	8.6 (2.7–27.9)
Sexual orientation (p = NC)**			
Heterosexual	1,042	4.1 (2.8–5.4)	Ref (—)
Lesbian/Bisexual	102	8.7†† (2.3–15.1)	2.1 (0.9–4.8)
No. male lifetime vaginal sex partners (p = 0.0005)**			
1	292	2.5¶¶ (0.1–4.8)	Ref (—)
2–3	230	2.0†† (0.3–3.7)	0.8 (0.3–2.4)
4–9	400	4.1 (2.1–6.0)	1.7 (0.7–4.2)
≥10	249	8.7 (5.0–12.4)	3.6 (1.2–10.7)
Previous STI diagnosis††† (p<0.0001)**			
No	978	3.1 (1.9–4.2)	Ref (—)
Yes	193	10.2 (6.0–14.3)	3.3 (1.9–5.7)

Abbreviations: CI = confidence interval; ED = emergency department; FPL = Federal Poverty Level; GED = General Educational Development certification; HMO = health maintenance organization; NC = not calculated; STI = sexually transmitted infection.

* Prevalence estimates based on response to the question "Have you ever been treated for an infection in your fallopian tubes, uterus or ovaries, also called a pelvic infection, pelvic inflammatory disease, or PID?"

† Based on a response of "Yes" to the question "Have you ever had vaginal, anal, or oral sex?"

§ Estimates were weighted to be nationally representative of the U.S. population, accounting for unequal probabilities of selection and nonresponse.

¶ Respondents with missing or unknown values were excluded from prevalence ratio calculations.

** Calculated via use of the Rao-Scott chi-square test. The overall p-values could not be calculated (p = NC) for some characteristics with zero prevalence in categories not shown (e.g., "other" category for sexual orientation).

†† Relative standard error >30% but <40%.

§§ Relative standard error >50%; estimates are suppressed.

¶¶ Relative standard error >40% but <50%.

*** Ratio of family income to poverty level as defined by the U.S. Census Bureau.

††† Participants who were told by a doctor or other health care professional in the last 12 months that they had chlamydia or gonorrhea or were ever told they have herpes, human papilloma virus, or genital warts.

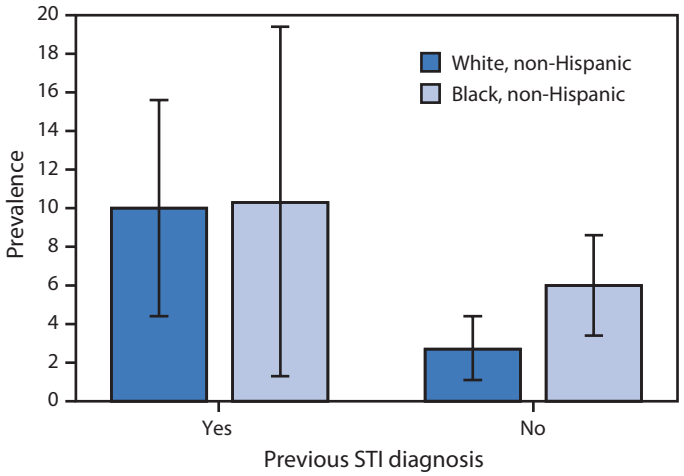
However, among women with no previous STI diagnosis, the prevalence of self-reported lifetime PID in black women was 2.2 times the prevalence in white women (black: 6.0% [95% CI: 3.4–8.6] versus white: 2.7% [95% CI: 1.1–4.4], p = 0.01).

Discussion

Based on NHANES 2013–2014 data, an estimated 2.5 million women aged 18–44 years in the United States reported a lifetime history of PID diagnosis. The increased prevalence among women reporting a previous STI diagnosis and other behaviors that increase risk for acquiring an STI underscores the need for STI prevention and control activities. The higher prevalence among black versus white women without a previous STI diagnosis suggests that black women might be more likely to have had an undiagnosed, asymptomatic STI or less likely to have received or reported a diagnosis for a symptomatic infection, possibly because of decreased access to care (*2*).

PID is not nationally notifiable but is reportable in some states. Few studies have assessed the incidence of PID using nationally representative data. Estimates from the National Survey of Family Growth found a similar prevalence of self-reported lifetime PID treatment among reproductive-aged women (5.7% during 2006–2010) and variations in self-reported lifetime PID treatment by sexual behaviors (*3*); women with a younger age of sexual debut and a higher number of lifetime vaginal sex partners were more likely to

FIGURE. Prevalence of self-reported lifetime pelvic inflammatory disease* among sexually experienced women† aged 18–44 years (n = 1,171), by race/ethnicity and previous STI diagnosis§,¶ — National Health and Nutrition Examination Survey, United States, 2013–2014



Abbreviation: STI = sexually transmitted infection.

* Prevalence estimates based on response to the question, "Have you ever been treated for an infection in your fallopian tubes, uterus or ovaries, also called a pelvic infection, pelvic inflammatory disease, or PID?" Estimates were weighted to be nationally representative of the U.S. population, accounting for unequal probabilities of selection and nonresponse.

† Based on a response of "Yes" to the question, "Have you ever had vaginal, anal, or oral sex?"

§ Participants who were told by a doctor or other health care professional in the last 12 months that they had chlamydia or gonorrhea or were ever told they have herpes, human papilloma virus, or genital warts.

¶ Bars indicate 95% confidence interval. Prevalence estimates among non-Hispanic black women with a previous STI diagnosis have a relative standard error >40% but <50%.

Summary

What is already known about this topic?

Pelvic inflammatory disease (PID) has various etiologies, including untreated chlamydia and gonorrhea infections, and is a potential sequela of these infections, with serious and costly outcomes. Chlamydia and gonorrhea infections are largely asymptomatic among women, and as such, most infections are undiagnosed and untreated.

What is added by this report?

In the National Health and Nutrition Examination Survey 2013–2014 cycle, the prevalence of a self-reported lifetime PID diagnosis was 4.4% among sexually experienced reproductive-aged women, equating to 2.5 million prevalent PID cases in women aged 18–44 years nationwide. Prevalence of a self-reported lifetime PID diagnosis varied by sexual behaviors and sexual health history and differed by race/ethnicity in women without a prior STI diagnosis.

What are the implications for public health practice?

These findings highlight differences in reproductive health by sexual behaviors and sexual health history. Given the potential of asymptomatic infection to lead to PID and the substantial costs associated with treatment, it is important that clinicians follow chlamydia and gonorrhea screening recommendations for women to decrease the incidence of PID.

have received treatment for PID. The results of that study indicated that black race, having less than a high school education, and an income <150% of the federal poverty level were associated with receipt of PID treatment.

The findings in this study are subject to at least four limitations. First, small sample sizes led to unstable estimates and wide CIs. Hence, these results should be interpreted cautiously. Second, NHANES PID data are based on self-report, an inherent problem of which is social-desirability bias. Third, given that PID is often asymptomatic and difficult to diagnose because of the lack of a diagnostic test and the low sensitivity and specificity associated with the use of a clinical case definition, estimates in this report might underestimate the actual prevalence of PID. Finally, temporality could not be established for all factors, and as such, there is no way to know whether the occurrence of certain factors (i.e., health insurance, access to health care, previous STI diagnoses) occurred before the PID diagnosis.

PID can result from untreated bacterial infections, including chlamydia and gonorrhea, both of which are treatable and preventable. Each case of PID results in an estimated average cost of $3,202 (*4*). Chlamydia and gonorrhea are the most commonly reported STIs in the United States, with approximately 1.5 million chlamydia and approximately 400,000 gonorrhea infections reported in 2015 (*5*). Most chlamydia and gonorrhea infections are asymptomatic in women and many go undiagnosed and untreated (*6*). Results from randomized controlled trials suggest that chlamydia screening is associated with a decreased incidence of PID (*7*,*8*). The U.S. Preventive Services Task Force recommends that all sexually active women aged <25 years and older women at increased risk for infection be screened for chlamydia and gonorrhea (*9*).

Using nationally representative data, this study found a substantial prevalence of PID in the United States. Lifetime prevalence of PID was highest in women with sexual behaviors and a sexual health history putting them at increased risk for STIs, including having had a prior STI diagnosis, and differed by race/ethnicity in those without a prior STI diagnosis. Given the potential for asymptomatic infections to lead to PID and the costs associated with treatment, it is important for clinicians to adhere to U.S. Preventive Services Task Force guidelines for chlamydia and gonorrhea screening in an effort to decrease the PID burden in sexually experienced women of reproductive age nationwide (*9*).

[1]Division of STD Prevention, National Center for HIV/AIDS, Viral Hepatitis, STD, and TB Prevention, CDC.

Corresponding author: Kristen Kreisel, ltq1@cdc.gov, 404-718-5148.

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Association Between Infant Mortality Attributable to Birth Defects and Payment Source for Delivery — United States, 2011–2013

Lynn M. Almli, PhD[1]; Caroline C. Alter, MS[2]; Rebecca B. Russell, MSPH[2]; Sarah C. Tinker, PhD[1]; Penelope P. Howards, PhD[3]; Janet Cragan, MD[1]; Emily Petersen, MD[1]; Gerard E. Carrino, PhD[4]; Jennita Reefhuis, PhD[1]

Birth defects are a leading cause of infant mortality in the United States (*1*), accounting for approximately 20% of infant deaths. The rate of infant mortality attributable to birth defects (IMBD) in the United States in 2014 was 11.9 per 10,000 live births (*1*). Rates of IMBD differ by race/ethnicity (*2*), age group at death (*2*), and gestational age at birth (*3*). Insurance type is associated with survival among infants with congenital heart defects (CHD) (*4*). In 2003, a checkbox indicating principal payment source for delivery was added to the U.S. standard birth certificate (*5*). To assess IMBD by payment source for delivery, CDC analyzed linked U.S. birth/infant death data for 2011–2013 from states that adopted the 2003 revision of the birth certificate. The results indicated that IMBD rates for preterm (<37 weeks of gestation) and term (≥37 weeks) infants whose deliveries were covered by Medicaid were higher during the neonatal (<28 days) and postneonatal (≥28 days to <1 year) periods compared with infants whose deliveries were covered by private insurance. Similar differences in postneonatal mortality were observed for the three most common categories of birth defects listed as a cause of death: central nervous system (CNS) defects, CHD, and chromosomal abnormalities. Strategies to ensure quality of care and access to care might reduce the difference between deliveries covered by Medicaid and those covered by private insurance.

This analysis used 2011–2013 Linked Birth and Infant Death Data from the National Vital Statistics System* for infants aged <1 year born to U.S. residents. The 2003 revision of the birth certificate included information on the payment source for delivery for the first time; thus, analysis of this variable was limited to states that adopted the 2003 revision. In 2011, 33 states and the District of Columbia (DC) (representing 76% of U.S. births) used the 2003 revision; in 2012, 36 states and DC (83%) used the revision, and in 2013, 38 states and DC used the revision (86%). Approximately 1.0%–1.2% of infant death records could not be linked to their corresponding birth certificates. The linkage completion by state ranged from 95.5%–100%, with approximately 50% of states linking all of their records each year. To accommodate nonlinked death records, estimates of the number of infant deaths for each state were weighted according to the percentage of records linked to a birth certificate by state and age group at death (*6*). Gestational age was based on last menstrual period (LMP). Birth and death records with unknown gestational age, gestational age of <20 weeks or >44 weeks, and implausible combinations of gestational age and birthweight (*7*) were excluded (7.9% of infant deaths and 1.2% of live births). Births not covered by Medicaid or private insurance (18.8% of infant deaths and 17.1% of live births) were included in the totals used for rate calculations. Deaths attributable to major birth defects were defined as those for which the underlying cause of death on the death certificate was classified as a birth defect according to the *International Classification of Diseases, 10th Revision*, codes Q00.0–Q99.9. Exceptions included the following: undescended testicles (Q53.1, Q53.2, and Q53.9) or cardiovascular conditions that were not considered structural heart defects (Q27.0–Q28.9); preterm births with an underlying cause of death considered to be a complication of prematurity (i.e., lung hypoplasia [Q33.6], persistent foramen ovale [PFO, Q21.1], and patent ductus arteriosus [PDA, Q25.0]); and all neonatal deaths among term infants with PFO or PDA as the underlying cause.

Estimates for IMBD rates and 95% confidence intervals by payment source for delivery were calculated for each stratum of the key variables: maternal race/ethnicity (non-Hispanic white, non-Hispanic black, Hispanic, and other race), maternal age (<20, 20–34, and >34 years), infant gestational age (<37 and ≥37 weeks), and infant age group at death (<28 days and ≥28 days to <1 year). IMBD rates by payment source were estimated separately for the three largest defect categories listed as a cause of death (CNS defects [Q00.0–Q07.9]; CHD [Q20.0–Q26.9]; and chromosomal abnormalities [Q90.0–Q99.9]). To assess the association between payment source for delivery and IMBD, Poisson regression was used to estimate the rate ratio, adjusted for maternal age group and race/ethnicity, comparing neonatal and postneonatal IMBD among infants whose deliveries were covered by Medicaid compared with those covered by private insurance, stratified by gestational age at birth.

The linked birth/infant death records from states that adopted the 2003 revision of the birth certificate during 2011–2013 included data from 9,542,603 live births (80.6% of the U.S. total) and 53,002 infant deaths (74.5% of the total). For 11,111 (21.0%) infant deaths, a birth defect was noted as the underlying cause of death, an overall IMBD rate of 11.6 per 10,000 live births. The rate of IMBD varied by maternal race/ethnicity (non-Hispanic white, 10.9; non-Hispanic

* https://www.cdc.gov/nchs/nvss/linked-birth.htm.

black, 13.6; and Hispanic, 12.6 per 10,000 live births) and by gestational age (preterm, 50.2; and term, 6.8 per 10,000 live births) (Table 1). Approximately 15% of all infant deaths and 70% of IMBD occurred in the neonatal period. CNS defects, CHD, and chromosomal abnormalities accounted for 57% of neonatal and 76% of postneonatal IMBD.

The IMBD rate overall was higher for deliveries covered by Medicaid (13.4 per 10,000 live births) than for deliveries covered by private insurance (9.6) (Table 1). The IMBD rate also was higher for deliveries covered by Medicaid when stratified by each of the key variables, except for maternal age <20 years (Table 1).

Payment source for delivery was associated with IMBD rates (Table 2). Among preterm births, neonatal and postneonatal IMBD rates were 12% (adjusted rate ratio [aRR] = 1.12; 95% confidence interval [CI] = 1.04–1.20) and 49% (aRR = 1.49; 95% CI = 1.29–1.72) higher, respectively, for infants whose delivery was covered by Medicaid than for those covered by private insurance. Among term births, neonatal and postneonatal IMBD rates were 44% (aRR = 1.44; CI = 1.33–1.56) and 45% (aRR = 1.45; CI = 1.31–1.60) higher, respectively, for infants whose delivery was covered by Medicaid, compared with infants whose delivery was covered by private insurance.

Among preterm births, postneonatal mortality for deliveries covered by Medicaid was 40% higher for CHD and 81% higher for chromosomal abnormalities, compared with those covered by private insurance. Among term births, neonatal mortality for deliveries covered by Medicaid was 37% higher for CNS defects, 46% higher for CHD, and 22% higher for chromosomal abnormalities, compared with those covered by private insurance. Postneonatal mortality was 39% higher for CNS defects, 43% higher for CHD, and 48% higher for chromosomal abnormalities among term deliveries covered by Medicaid than among deliveries covered by private insurance.

Discussion

The analysis of differences in IMBD according to source of payment was possible because of the addition of a checkbox for payment source for delivery on the 2003 revision of the U.S. standard birth certificate. This variable has been assessed for data quality with moderate to substantial validity (*5*). Differences in IMBD between deliveries covered by Medicaid and those covered by private insurance were observed across categories of gestational age at birth and age group at death. Postneonatal IMBD for preterm infants, and both neonatal and postneonatal IMBD for term infants were approximately 45% higher for deliveries covered by Medicaid than those covered by private insurance. Postneonatal mortality was higher for deliveries covered by Medicaid than those covered by private insurance for the three most common categories of birth defects listed as a cause of death: CNS, CHD, and chromosomal abnormalities.

Although IMBD differed by payment source for delivery, the factors underlying this difference are not known. Differences

TABLE 1. Maternal and infant characteristics among cases of infant mortality attributable to birth defects (IMBD), by payment source for delivery* — United States, 2011–2013

Characteristic	Total†			Private insurance			Medicaid		
	No. of IMBD cases	No. of live births	Rate§ (95% CI)	No. of IMBD cases	No. of live births	Rate§ (95% CI)	No. of IMBD cases	No. of live births	Rate§ (95% CI)
Total	**11,111**	**9,542,603**	**11.6 (11.4–11.9)**	**4,227**	**4,391,048**	**9.6 (9.3–9.9)**	**5,580**	**4,163,142**	**13.4 (13.1–13.8)**
Infant age group at death¶									
Neonatal	7,767	9,542,603	8.1 (8.0–8.3)	3,020	4,391,048	6.9 (6.6–7.1)	3,787	4,163,142	9.1 (8.8–9.4)
Postneonatal	3,344	9,542,603	3.5 (3.4–3.6)	1,207	4,391,048	2.8 (2.6–2.9)	1,793	4,163,142	4.3 (4.1–4.5)
Gestational age at birth**									
Preterm	5,375	1,069,836	50.2 (48.9–51.6)	2,068	442,736	46.7 (44.7–48.7)	2,679	517,340	51.8 (49.8–53.7)
Term	5,736	8,472,767	6.8 (6.6–6.9)	2,159	3,948,312	5.5 (5.2–5.7)	2,901	3,645,802	8.0 (7.7–8.2)
Maternal race/Ethnicity									
White, non-Hispanic	5,548	5,090,511	10.9 (10.6–11.2)	2,901	3,039,303	9.5 (9.2–9.9)	2,082	1,617,825	12.9 (12.3–13.4)
Black, non-Hispanic	1,861	1,365,718	13.6 (13.0–14.2)	426	349,951	12.2 (11.0–13.3)	1,246	889,764	14.0 (13.2–14.8)
Hispanic	2,953	2,338,059	12.6 (12.2–13.1)	565	577,763	9.8 (9.0–10.6)	1,921	1,412,422	13.6 (13.0–14.2)
Other	582	672,661	8.7 (7.9–9.4)	269	385,008	7.0 (6.2–7.8)	256	214,677	11.9 (10.5–13.4)
Maternal age (yrs)									
<20	1,033	742,117	13.9 (13.1–14.8)	158	116,418	13.6 (11.5–15.7)	760	551,857	13.8 (12.8–14.8)
20–34	7,923	7,374,464	10.7 (10.5–11.0)	2,984	3,361,206	8.9 (8.6–9.2)	4,002	3,237,908	12.4 (12.0–12.7)
>34	2,155	1,426,022	15.1 (14.5–15.8)	1,085	913,424	11.9 (11.2–12.6)	818	373,377	21.9 (20.4–23.4)

* Includes residents during 2011–2013 of states that used the 2003 revised U.S. standard birth certificate, which added a checkbox indicating principal payment source for delivery: 33 states in 2011, 36 in 2012, and 38 in 2013.
† Includes Medicaid, private insurance, self-pay, other, and unknown categories of payment source for delivery.
§ Number of IMBD cases per 10,000 live births.
¶ Neonatal mortality is death of an infant with birth defects at <28 days of age. Postneonatal mortality is death of an infant with birth defects at ≥28 days to <1 year.
** Preterm birth is classified as <37 completed weeks of gestation. Term birth is classified as ≥37 completed weeks of gestation.

TABLE 2. Rates* of infant mortality attributable to birth defects (IMBD), by gestational age at birth,† payment source for delivery,§ birth defect category, and infant age group at death¶ — United States, 2011–2013

	Preterm			Term		
Birth defect category	Private insurance	Medicaid	Adjusted rate ratio** (95% CI)	Private insurance	Medicaid	Adjusted rate ratio** (95% CI)
Total††						
Neonatal	38.2	39.6	1.12 (1.04–1.20)	3.3	4.7	1.44 (1.33–1.56)
Postneonatal	8.1	11.7	1.49 (1.29–1.72)	2.2	3.2	1.45 (1.31–1.60)
Central nervous system defects						
Neonatal	5.6	6.6	1.17 (0.98–1.40)	0.6	0.8	1.37 (1.13–1.66)
Postneonatal	0.7	1.4	1.54 (0.98–2.43)	0.2	0.4	1.39 (1.03–1.89)
Congenital heart defects						
Neonatal	5.4	5.6	1.11 (0.92–1.34)	0.8	1.2	1.46 (1.24–1.70)
Postneonatal	3.5	4.9	1.40 (1.12–1.74)	1.1	1.6	1.43 (1.24–1.64)
Chromosomal abnormalities						
Neonatal	9.2	7.4	1.00 (0.85–1.16)	0.8	0.9	1.22 (1.03–1.44)
Postneonatal	1.5	2.1	1.81 (1.30–2.53)	0.4	0.6	1.48 (1.18–1.87)

* Number of IMBD cases per 10,000 live births among residents during 2011–2013 of states that used the 2003 revised birth certificate: 33 states in 2011, 36 in 2012, and 38 in 2013.
† Preterm birth is classified as <37 completed weeks of gestation. Term birth is classified as ≥37 completed weeks of gestation.
§ Includes residents during 2011–2013 of states that used the 2003 revised U.S. standard birth certificate, which added a checkbox indicating principal payment source for delivery: 33 states in 2011, 36 in 2012, and 38 in 2013.
¶ Neonatal mortality is death of an infant with birth defects at <28 days of age. Postneonatal mortality is death of an infant with birth defects at ≥28 days to <1 year.
** Infant births covered by Medicaid compared with births covered by private insurance, adjusted for maternal age group and race/ethnicity.
†† Includes infants born to Hispanic, non-Hispanic black, non-Hispanic white women, and women of other race/ethnicity.

in health status, access to and utilization of preconception health care, prenatal care, prenatal screening, and termination of pregnancies for fetal anomalies, as well as changes in insurance policies and Medicaid coverage over time could have influenced these findings (*8*,*9*).

The findings in this report are subject to at least four limitations. First, although 76%–86% of all live births were captured in this analysis, data from 12 to 17 states were not included; thus, these results are not generalizable to all states. Second, because of small numbers, it was not possible to examine other payment sources for delivery, including those deliveries that were not covered by any insurance. Third, deaths for which birth defects were listed as a contributing cause of death but not the underlying cause were not included, which likely resulted in an underestimation of IMBD. Finally, misclassification of the underlying cause of death, gestational age based on LMP (*10*), and payment source for delivery might differ by factors considered in these analyses. It is possible that mothers with high-risk pregnancies switched their payment source during pregnancy, which could have increased IMBD rates among Medicaid-covered births.

Birth defects are serious conditions that affect about one in 33 births, and in 2011–2013, one in five infant deaths had a birth defect listed as the underlying cause of death. Although IMBD rates are declining because of improvements in treatment and early detection, strategies need to be implemented to reduce IMBD for all deliveries, regardless of payment source.

Summary

What is already known about this topic?

Infant mortality attributable to birth defects (IMBD) differs by race/ethnicity, gestational age, and age group at death.

What is added by this report?

The rates of IMBD among preterm infants whose deliveries were covered by Medicaid were 12% and 49% higher during the neonatal and postneonatal periods, respectively, than IMBD rates among preterm infants whose deliveries were covered by private insurance. IMBD rates among term infants whose deliveries were covered by Medicaid were 44% and 45% higher during the neonatal and postneonatal periods, respectively, than rates among term infants whose deliveries were covered by private insurance.

What are the implications for public health practice?

Rates of infant mortality attributable to birth defects are higher for births covered by Medicaid than for those covered by private insurance. Strategies to ensure quality of care and access to care might reduce the differences between deliveries covered by Medicaid and those covered by private insurance.

[1]Division of Congenital and Developmental Disorders, National Center on Birth Defects and Developmental Disabilities, CDC; [2]March of Dimes Foundation, White Plains, New York; [3]Department of Epidemiology, Rollins School of Public Health, Emory University, Atlanta, Georgia; [4]Texas A&M School of Public Health, College Station, Texas.

Corresponding author: Lynn Almli, imv0@cdc.gov, 404-498-3821.

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Notes from the Field

Multistate Outbreak of *Escherichia coli* O157:H7 Infections Linked to Dough Mix — United States, 2016

Laura Gieraltowski, PhD[1]; Colin Schwensohn, MPH[1]; Stephanie Meyer, MPH[2]; Dana Eikmeier, MPH[2]; Carlota Medus, PhD[2]; Alida Sorenson, MPH[3]; Matthew Forstner[3]; Asma Madad, MS, MPH[4]; Joseph Blankenship, MPH[4]; Peter Feng, PhD[4]; Ian Williams, PhD[1]

On January 4, 2016, CDC PulseNet, the molecular subtyping network for foodborne disease surveillance, identified a cluster of 10 Shiga toxin-producing *Escherichia coli* (STEC) O157:H7 infections with indistinguishable pulsed-field gel electrophoresis (PFGE) pattern combinations. STEC infections with the identified outbreak PFGE pattern are commonly reported to PulseNet, with an average of 40–50 illnesses reported annually. Because this was a relatively common strain of STEC, multiple locus variable-number tandem repeat analysis (MLVA), another subtyping technique used to characterize the genetic relatedness of bacteria, was used to help define cases in the cluster. CDC collaborated with state and local health and agricultural agencies and the Food and Drug Administration (FDA) to investigate the outbreak. A case was defined as STEC O157:H7 infection with an isolate having PFGE and MLVA patterns indistinguishable from the outbreak strain in a person with diarrheal illness onset during December 6, 2015–February 9, 2016.

Thirteen STEC O157:H7 outbreak-associated cases were identified in nine states: Iowa (one case), Illinois (one), Kansas (one), Minnesota (five), North Carolina (one), Nebraska (one), New Jersey (one), South Dakota (one), and Wisconsin (one). The median age of patients was 17 years (range = 7–71 years); 53% were female. Among 12 patients with available information, eight were hospitalized, including two who developed hemolytic uremic syndrome; no deaths were reported.

Among the 12 interviewed patients, nine reported eating at one of nine locations of restaurant A, a national restaurant chain, during the week preceding illness onset, including eight who ate a specific dessert pizza made with a proprietary dough mix provided by manufacturer A. The ninth patient consumed bread sticks made from the same dough mix. At one Minnesota location, six of 21 (28%) non-ill patrons reported eating the implicated dessert pizza. Assuming this was representative of patrons of restaurant A, the proportion of cases who consumed dessert pizza was significantly higher than what would be expected by chance using the binomial distribution model ($p<0.001$). As an intervention in this outbreak, restaurant A locations stopped using dough mix from manufacturer A on February 4, 2016.

Eighty-eight samples of dry dough mix from five restaurant A locations where patients reported eating were collected by public health officials in five states (Illinois, Iowa, Minnesota, Nebraska, and Wisconsin). The Minnesota Department of Agriculture identified non-O157 STEC in seven of 17 collected samples, including one Shiga toxin-1–producing non-O157 STEC isolate and six Shiga toxin-2 (stx2)–producing non-O157 STEC isolates. FDA collected six samples of dry dough mix from manufacturer A. All six samples tested negative for STEC O157:H7, but one yielded an stx2-producing STEC O8:H28. All identified strains lacked known adherence factors and were therefore considered to present a low health risk.

Flour is a raw agricultural product and does not undergo processing to kill bacteria and other pathogens, so it is not sterile. Generic *E. coli* and coliforms have been found previously in flour (*1*,*2*). Flour and flour-based mixes have been suspected or implicated as the source of other foodborne *Salmonella* and STEC O157 outbreaks (*1*,*3*–*6*). Of note, this PFGE pattern was previously isolated from a sample of bulk flour collected during a 2009 outbreak investigation (*5*). Although no laboratory evidence identified contaminated flour as the ultimate source of this STEC O157:H7 outbreak, the identification of other enteric pathogens in multiple samples of dry dough mix consumed by patients associated with the outbreak implicates contaminated flour as the possible source of pathogen introduction for this outbreak. The small number of cases and the lack of additional restaurant clusters suggest that this was a low level contamination event or that contamination only affected a limited amount of product. Evidence obtained at one restaurant A location showed that dessert pizzas were made with the same dough mix used in traditional pizzas, but used thicker dough and might have been undercooked at some locations. Flour is usually not thought to be a food safety risk, but flour-based mixes are ubiquitous in restaurants and are often used for dusting of surfaces for transfer of pizzas. This outbreak serves as a reminder that consumers, industry, and government should consider that flour, a raw agricultural product, might be contaminated with pathogens and, when consumed raw or undercooked, might pose a risk to human health.

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[1]National Center for Emerging and Zoonotic Infectious Diseases, CDC; [2]Minnesota Department of Health; [3]Minnesota Department of Agriculture; [4]Food and Drug Administration.

Corresponding author: Laura Gieraltowski, LGieraltowski@cdc.gov, 404-639-3868.

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Notes from the Field

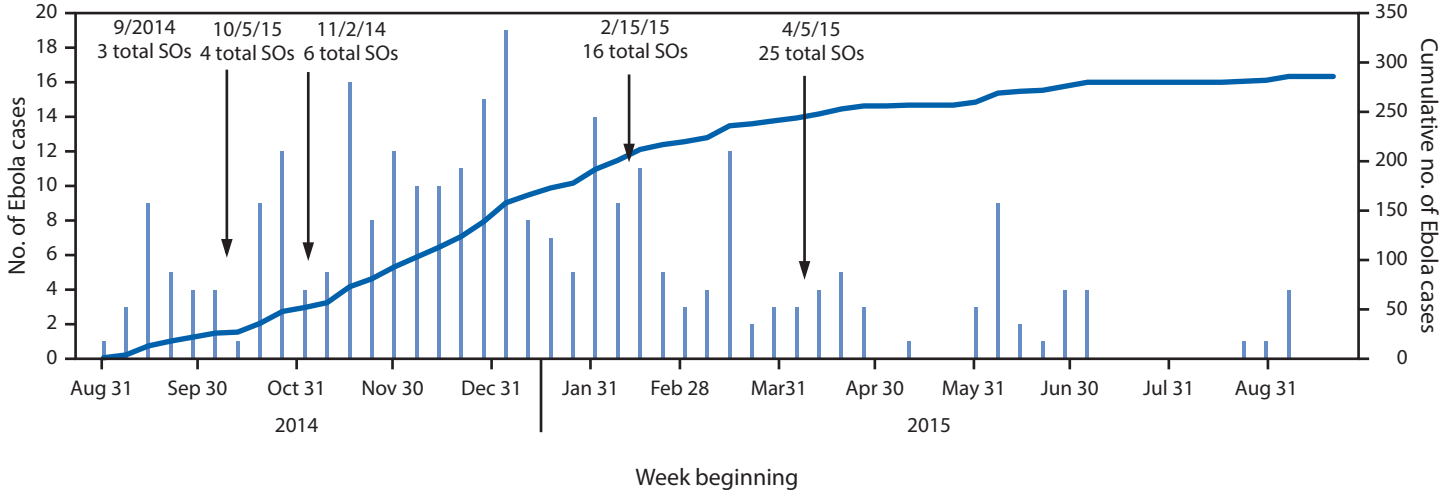
Impact of Increasing the Number of Ebola Surveillance Officers — Kambia District, Sierra Leone, September 2014–September 2015

Christopher Sandi[1]; Osman Barrie[1]; Hassan Kanu[1]; Foday Sesay[1]

Kambia is one of 14 districts in Sierra Leone. Located in the northwest part of the country, Kambia comprises seven rural chiefdoms. The total population is approximately 344,000. The first case of Ebola virus disease (Ebola) in Kambia occurred on September 4, 2014. Three disease surveillance officers within the District Health Management Team (DHMT) of the Ministry of Health and Sanitation (MoHS) responded to suspected Ebola case alerts. The role of the surveillance officers was to investigate all suspected Ebola cases, and in the event of a confirmed case, initiate the isolation and contact tracing processes. With only one surveillance officer for every 115,000 persons, staffing available for investigation of and response to alerts was inadequate. Without sufficient resources to identify and contain new cases, the Ebola case count continued to increase. To help contain the outbreak, the number of surveillance officers in Kambia was augmented four times. The first addition of a surveillance officer (increasing the number of officers from three to four) occurred in October 2014. By April 2015, the number of surveillance officers had been increased to 25, with two associated rounds of training. MoHS reviewed the number of Ebola cases recorded during September 2014–September 2015 and assessed the impact of the addition of more surveillance officers on the number of Ebola cases recorded in Kambia District.

During September 2014, there were only three surveillance officers in Kambia District, an area approximately 1,200 square miles, and the number of Ebola cases was steadily increasing, with a recorded mean each week of four new confirmed and probable Ebola cases (*1*) (hereafter "confirmed and probable cases" are referred to as "cases") (Figure). On October 5, 2014, an additional DHMT officer was assigned to surveillance in Kambia, bringing the number of surveillance officers to four; however, the number of cases continued to increase (mean number of cases per week was seven). On November 2, after a total of 48 cases had been recorded, two additional DHMT officers were assigned to surveillance, bringing the total number of surveillance officers in Kambia to six; during the 15 weeks from November 2014 to mid-February 2015, the Ebola epidemic in Kambia reached its peak, with a mean of 10 new cases recorded each week. On February 15, 2015, after 153 additional Ebola cases had been recorded since November 2 (212 cumulative cases), 10 new surveillance officers were trained as chiefdom surveillance officers, permanently based in each of the chiefdoms, bringing the total number of surveillance officers to 16. Three of seven rural chiefdoms were assigned two officers; others were assigned one chiefdom surveillance officer. The goal with adding these 10 surveillance officers was to reduce transportation time between chiefdoms in responding to alerts, and for the officers to act as first responders by ascertaining whether a suspected

FIGURE. Number of incidents and cumulative Ebola virus disease cases* and the number of additional surveillance officers (SOs) — Kambia District, Sierra Leone, September 2014–August 2015



* Case numbers refer to the sum of confirmed and probable cases.

case met the case definition before the alert was escalated to a headquarters surveillance officer.

The number of new Ebola cases in Kambia District began to decline during mid-February–early April, (mean number of cases per week was six). On April 5, 2015, after 244 cumulative Ebola cases had been recorded (almost 2 months since the previous addition of surveillance officers), nine new surveillance officers were trained to operate from headquarters, bringing the total number of surveillance officers in Kambia District to 25. From early April through the first week of September 2015, with 25 surveillance officers in the district (eight times the number at the beginning of the outbreak, and proportional to the number in larger districts, such as Port Loko, which had approximately 40 surveillance officers for a population of 614,000), the number of Ebola cases continued to decline (mean number of cases per week was two). The last Ebola case in Kambia was recorded on September 9, 2015, after a total of 286 cases had been recorded in the district. The two major increases in the number of district surveillance officers coincided with the initial decrease in Ebola cases after the epidemic's peak, and the second gradual decline to zero cases.

The addition of disease surveillance officers in Kambia enabled public health officials to provide a more timely response to alerts as well as conduct active case searching throughout the district, which was associated with earlier detection and a decline in number of new cases recorded. Active surveillance was combined with outreach and community education from surveillance officers regarding the importance of reporting deaths and raising alerts. The faster response to alerts resulted in early isolation of patients and initiation of quarantine, which limited community spread. Increasing the number of district surveillance officers made early detection and containment possible and led to an eventual end of the Ebola outbreak in Kambia District.

Acknowledgments

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[1]District Health Management Team, Ministry of Health and Sanitation, Kambia District, Sierra Leone.

Corresponding author: Christopher Sandi, kambiadso@gmail.com, 232-99981766.

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Announcements

Community Preventive Services Task Force Recommends Interventions Engaging Community Health Workers for Diabetes Prevention

The Community Preventive Services Task Force recently posted new information on its website: "Diabetes: Interventions Engaging Community Health Workers." The information is available at https://www.thecommunityguide.org/findings/diabetes-interventions-engaging-community-health-workers.

Established in 1996 by the U.S. Department of Health and Human Services, the task force is an independent, nonfederal, uncompensated panel of public health and prevention experts whose members are appointed by the director of CDC. The task force provides information for a wide range of decision makers on programs, services, and policies aimed at improving population health. Although CDC provides administrative, research, and technical support for the task force, the recommendations developed are those of the task force and do not undergo review or approval by CDC.

25th Anniversary of National Program of Cancer Registries, 1992–2017

This year marks the 25th anniversary of the Cancer Registries Amendment Act (Public Law 102–515), which established the National Program of Cancer Registries (NPCR) administered by CDC (*1*). NPCR collects high quality data on cancer occurrence (including the type, extent, and location of the cancer), type of initial treatment, and outcomes. NPCR supports cancer registries in 45 states, the District of Columbia, Puerto Rico, and U.S. Pacific Island jurisdictions, covering 96% of all cancers diagnosed in the United States.

This week, *MMWR* features a Surveillance Summary and a weekly report that use NPCR data. The Surveillance Summary describes national cancer incidence and death rates for 68 cancer types among men and 72 among women, and state-specific rates for common cancers and trends for all cancer sites combined (*2*). The weekly report summarizes incidence rates for common cancers tracked in *Healthy People 2020* and cancers that can be prevented through limiting risk factors (tobacco use, alcohol use) or increasing vaccination (against human papilloma virus) (*3*), and highlights states' use of registry data to advance public health. Together, these reports demonstrate how public health planners in states, territories, and tribes use NPCR data to measure progress and target action for cancer prevention and control.

CDC annually provides cancer surveillance data via several products (*3*). This month, for the first time, CDC released a public use research NPCR data set, available at https://www.cdc.gov/cancer/npcr/public-use. Detailed, de-identified information on several million cancer cases from 1999 to 2013 is now available, providing researchers and the interested public the opportunity to analyze these data to better understand cancer, inform coordinated efforts to address cancer through prevention, and evaluate progress in cancer control.

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Errata

Vol. 65, No. 52

In the report "Human Rabies — Puerto Rico, 2015," on page 1476, the first sentence under the Summary heading "What is added by this report?" should have read "A man aged 54 years who was bitten by a mongoose in October **2015** was the first person to acquire rabies from a mongoose in the United States or U.S. territories, confirming mongoose rabies as a public health threat."

Vol. 66, Nos. SS-1 and SS-2

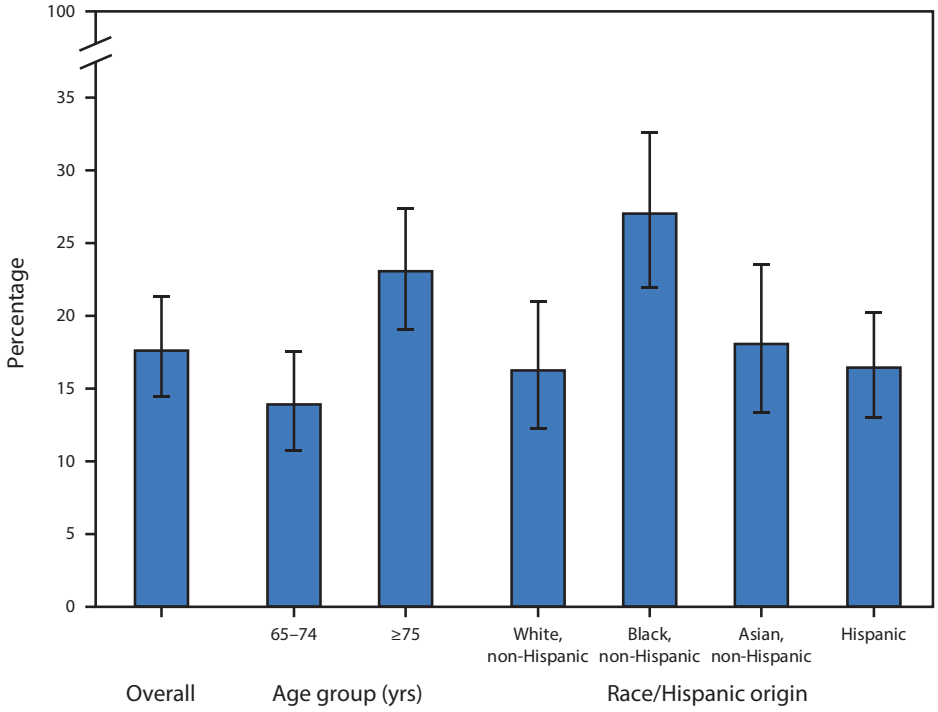
In the Surveillance Summaries "Leading Causes of Death in Nonmetropolitan and Metropolitan Areas — United States, 1999–2014" and "Reducing Potentially Excess Deaths from the Five Leading Causes of Death in the Rural United States," an error occurred in Figure 5 and Figure 3, respectively. In the last panel of bar charts (stroke), the colors for the left-most set of bars (public health region 1) **should be reversed**.

In addition, the following person should have been listed as the Guest Editor in the masthead and the Acknowledgments: **Robin M. Wagner, PhD, MS, *Guest Editor*.**

QuickStats

FROM THE NATIONAL CENTER FOR HEALTH STATISTICS

Prevalence* of Edentualism† in Adults Aged ≥65 Years, by Age Group and Race/Hispanic Origin — National Health and Nutrition Examination Survey, 2011–2014



* With 95% confidence intervals indicated with error bars.
† Edentualism is the loss of all natural, permanent teeth. Data were collected by dentists in the mobile examination center as part of the oral health component of the National Health and Nutrition Examination Survey.

During 2011–2014, 17.6% of adults aged ≥65 years were edentulous or had lost all their natural, permanent teeth. Adults aged ≥75 years (23.0%) were more likely to be edentulous compared with adults aged 65–74 years (13.9%). Non-Hispanic black adults aged ≥65 years were more likely to be edentulous (27.0%) compared with non-Hispanic white (16.2%), non-Hispanic Asian (18.0%), and Hispanic adults (16.4%) aged ≥65 years.

Source: CDC/NCHS. National Health and Nutrition Examination Survey Data. Hyattsville, MD: US Department of Health and Human Services, CDC, National Center for Health Statistics; 2011–2014. https://www.cdc.gov/nchs/nhanes.htm.

Reported by: Eleanor Fleming, PhD, DDS, efleming@cdc.gov, 301-458-4062; Joseph Afful, MS; Steven M. Frenk, PhD.

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