

## National and State Estimates of the Numbers of Adults and Children with Active Epilepsy — United States, 2015

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Epilepsy, a brain disorder leading to recurring seizures, has garnered increased public health focus because persons with epilepsy experience pronounced and persistent health and socioeconomic disparities despite treatment advances, public awareness programs, and expanded rights for persons with disabilities (1,2). For almost all states, epilepsy prevalence estimates do not exist. CDC used national data sources including the 2015 National Health Interview Survey (NHIS) for adults (aged ≥18 years), the 2011–2012 National Survey of Children's Health (NSCH), and the 2015 Current Population Survey data, describing 2014 income levels, to estimate prevalent cases of active epilepsy, overall and by state, to provide information for state public health planning. In 2015, 1.2% of the U.S. population (3.4 million persons: 3 million adults and 470,000 children) reported active epilepsy (self-reported doctor-diagnosed epilepsy and under treatment or with recent seizures within 12 months of interview) or current epilepsy (parent-reported doctor-diagnosed epilepsy and current epilepsy). Estimated numbers of persons with active epilepsy, after accounting for income and age differences by state, ranged from 5,900 in Wyoming to 427,700 in California. NHIS data from 2010–2015 indicate increases in the number of persons with active epilepsy, probably because of population growth. This study provides updated national and modeled state-specific numbers of active epilepsy cases. Public health practitioners, health care providers, policy makers, epilepsy researchers, and other epilepsy stakeholders, including family members and people with epilepsy, can use these findings to ensure that evidence-based programs meet the complex needs of adults and children with epilepsy and reduce the disparities resulting from it.

Epilepsy has been assessed only intermittently in population surveys (1,2). Before 2010, the last U.S. national estimate of epilepsy prevalence was based on 1986–1990 data using one question assessing the occurrence of epilepsy or repeated seizures, convulsions, or blackouts in any household family members (3). Other recent estimates based on limited U.S. and international geographic regions, clinical samples, and decades-old data are not representative of the current U.S. population (2,4). Data from the 2010 and 2013 NHIS using a validated case definition indicate approximately 1% of the U.S. population had active epilepsy (5). A study using 2005 Behavioral Risk Factor Surveillance System data employing

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similar epilepsy case-ascertainment questions\* provided state-level estimates of a history of epilepsy for 19 states (1.65%) and active epilepsy for 13 states (0.84%) (6). No substantial differences among states in the prevalence of a history of epilepsy or active epilepsy were detected (6). A third study, which extrapolated 2007–2011 administrative claims data from multiple states to the overall U.S. population found an epilepsy prevalence estimate of 0.84% (4). For almost all states, epilepsy prevalence estimates do not exist. Groups interested in reducing epilepsy prevalence need updated estimates of the numbers of persons living with epilepsy nationally and within their states. This study aims to provide updated national and modeled state-specific estimates of active epilepsy prevalence

based on the latest data available to provide information for public health action to reduce epilepsy burden.

To estimate the number of prevalent cases of active epilepsy among adults aged  $\geq 18$  years, CDC analyzed three questions on epilepsy from the 2015 Sample Adult component of NHIS, an annual, cross-sectional household survey of the civilian, noninstitutionalized U.S. population. Adults classified as having “active epilepsy” reported a history of doctor-diagnosed epilepsy and were taking medication to control it, had had one or more seizures in the past year, or both (Table 1) (5,6). Validation of survey questions for surveillance of active epilepsy yielded sensitivity and specificity exceeding 80% and 99%, respectively, with a positive predictive value of 74% similar to validation estimates seen in surveillance of other chronic disorders (5). Only 0.07% of adults in 2015 refused to answer or did not know if they had doctor-diagnosed active epilepsy. To estimate prevalent cases of active epilepsy among children aged 0–17 years, CDC analyzed data from the 2011–2012 NSCH,<sup>†</sup> a cross-sectional telephone survey of households with at least one resident child aged 0–17 years at interview. NSCH asks parents or guardians if a doctor or health care provider ever told them that their child had epilepsy or seizure disorder, and if so, if their child currently has epilepsy or seizure disorder (current epilepsy) (Table 1). Only 0.03% of parents or guardians refused to answer or did not know if a

\*In 2005, the Behavioral Risk Factor Surveillance System included the following five epilepsy questions: 1) “Have you ever been told by a doctor that you have a seizure disorder or epilepsy?” (response options: “yes,” “no,” “don’t know,” and “refused”). Participants who answered “yes” to this question were asked some or all of the following questions: 2) “Are you currently taking any medicine to control your seizure disorder or epilepsy?” (response options: “yes,” “no,” “don’t know,” and “refused”); 3) “How many seizures have you had in the last 3 months?” (response options: “none,” “one,” “more than one,” “no longer have epilepsy or seizure disorder,” “don’t know,” and “refused”); 4) “In the past year, have you seen a neurologist or epilepsy specialist for your epilepsy or seizure disorder?” (response options: “yes,” “no,” “don’t know,” and “refused”); and 5) “During the past 30 days, to what extent has epilepsy or its treatment interfered with your normal activities like working, school, or socializing with family or friends?” (response options: “not at all,” “slightly,” “moderately,” “quite a bit,” “extremely,” “don’t know,” and “refused”). The only change between the earlier BRFSS and the later NHIS case-ascertainment is that BRFSS includes a 3-month recall for any seizure occurrence and NHIS, a 12-month recall for any seizure occurrence. This change reflected updated consensus guidance on case-ascertainment for community-based epilepsy surveillance.

<sup>†</sup> National Survey of Children’s Health, 2011–2012. <https://www.cdc.gov/nchs/slraits/nsch.htm>.

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TABLE 1. Epilepsy surveillance case ascertainment questions, by survey

Survey	Questions	Possible responses
National Health Interview Survey (2015)	1. Have you ever been told by a doctor or other health professional that you have a seizure disorder or epilepsy?	1) Yes, 2) No, 7) Refused, 8) Not ascertained, 9) Don't know
	2. Are you currently taking any medicine to control your seizure disorder or epilepsy?	1) Yes, 2) No, 7) Refused, 8) Not ascertained, 9) Don't know
	3. Today is <date>. Think back to last year about the same time. About how many seizures of any type have you had in the past year?	0) None, 1) One, 2) Two or three, 3) Between four and ten, 4) More than 10, 7) Refused, 8) Not ascertained, 9) Don't know
National Survey of Children's Health (2011–2012)	1. Has a doctor or health care provider ever told you that your child has epilepsy or a seizure disorder?	1) Yes, 2) No, 7) Refused, 8) Not ascertained, 9) Don't know
	2. Does your child currently have epilepsy or a seizure disorder?	1) Yes, 2) No, 7) Refused, 8) Not ascertained, 9) Don't know

doctor had ever told them their child had epilepsy or a seizure disorder. Prevalence of current epilepsy among children based on NSCH data was estimated to be 6.3 per 1,000, similar to estimates from administrative data (7,8).

Obtaining state-level estimates required using the best available data to confirm that epilepsy prevalence did not differ significantly across states (6). Epilepsy prevalence and state populations do differ by age and income distribution. NHIS and NSCH data was used to calculate the prevalence (proportion) of active epilepsy for three age groups (0–17 years, 18–64 years, and ≥65 years) stratified by three family income groups (0%–99%, 100%–199%, ≥200% of poverty thresholds). Data for 2014 was obtained for the three age groups and three family income groups among civilian and military noninstitutionalized populations for each state from the U.S. Census's Current Population Survey 2015 Annual Social and Economic Supplement.<sup>§</sup> Multiplying the age- and income-specific active epilepsy prevalence estimates by the population estimates for each of the three age and income groups yielded state-level estimates of active epilepsy, indirectly standardized for age and income.<sup>¶</sup> Adding these standardized estimates for both groups from each data set produced total estimated numbers of cases with active epilepsy. Combining the variance estimates of both adults and children with epilepsy from each survey and of these age- and income-specific population estimates as the variance of the product of these two random variables yielded 95% confidence intervals for these total estimates.\*\*

<sup>§</sup> Current Population Survey Annual and Social Economic Supplement, 2014 Poverty Status by State and Age Groups. <https://www.census.gov/data/tables/time-series/demo/income-poverty/cps-pov/pov-46.2014.html>.

<sup>¶</sup> The estimated numbers of active epilepsy cases are calculated in the same way as the expected numbers in indirect standardization are calculated to account for confounding (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3406211>).

\*\* The formula to calculate the variances for the 95% confidence intervals of the expected numbers of active epilepsy cases is found in <http://www.jstor.org/stable/2281592>.

In 2015, 1.2% (95% confidence interval = 1.1–1.4) of the U.S. population was classified as having active epilepsy (3.4 million; 3 million adults and 470,000 children). Among adults, the estimated number of cases of active epilepsy ranged from 5,100 in Wyoming to 367,900 in California (Table 2). Among children, the estimated number of cases of current epilepsy ranged from 800 in Wyoming to 59,800 in California. The number of persons estimated to have active epilepsy was <14,000 in nine states and the District of Columbia, 14,000–32,799 in 11 states, 32,800–56,799 in nine states, 56,800–92,699 in 10 states, and ≥92,700 persons in 11 states. (Table 2).

## Discussion

This study provides updated national and estimated state-specific numbers of the active epilepsy cases. Affecting 3.4 million U.S. residents, epilepsy is not a rare condition. Epilepsy poses substantial individual and societal burdens that require heightened public health action (1,2). As a complex condition varying in severity and impact, it affects persons of all ages and racial and ethnic groups, especially those with the lowest incomes (2,5,9). Persons with epilepsy often have multiple co-occurring conditions (e.g., stroke, heart disease, depression, or developmental delay) that complicate their epilepsy management, impair life goals, and contribute to early mortality (1,2). Among five chronic conditions in children and adolescents selected because of their adverse impact on academic and health outcomes, epilepsy is the costliest and the second most common (8). Children with seizures are more likely to live in poverty, and their parents more frequently report food insecurity (9). Direct yearly health care costs per person with epilepsy ranged from \$10,192 to \$47,862 (2013 U.S. dollars) and were higher for persons with uncontrolled seizures (10).

Medicaid recipients have a higher prevalence of epilepsy, especially among adults aged 20–64 years (3.4%) (4); this study adjusted for income to account for this confounder.

TABLE 2. Estimated numbers of active epilepsy cases, by state and age group — United States, 2015

Geographic area	Age group (yrs)		
	All ages	<18*	≥18†
	No. (95% CI <sup>‡</sup> )	No. (95% CI)	No. (95% CI)
<b>United States</b>	<b>3,439,600 (3,009,100–3,870,100)</b>	<b>471,900 (392,600–551,200)</b>	<b>2,967,700 (2,544,500–3,390,800)</b>
Alabama	54,100 (46,400–61,900)	7,500 (5,900–9,200)	46,600 (39,000–54,200)
Alaska	7,200 (6,100–8,300)	1,100 (800–1,400)	6,100 (5,000–7,200)
Arizona	77,000 (66,400–87,500)	11,200 (8,900–13,600)	65,700 (55,400–76,000)
Arkansas	32,800 (28,000–37,600)	4,900 (3,700–6,100)	28,000 (23,300–32,600)
California	427,700 (372,600–482,900)	59,800 (49,000–70,600)	367,900 (313,800–422,000)
Colorado	56,800 (48,300–65,300)	7,800 (6,000–9,600)	49,000 (40,700–57,300)
Connecticut	35,900 (30,400–41,400)	4,500 (3,400–5,700)	31,400 (26,000–36,800)
Delaware	9,700 (8,200–11,100)	1,300 (900–1,600)	8,400 (7,000–9,900)
District of Columbia	7,500 (6,300–8,800)	800 (600–1,100)	6,700 (5,500–7,900)
Florida	223,900 (194,100–253,800)	27,300 (21,900–32,800)	196,600 (167,200–225,900)
Georgia	110,200 (94,900–125,500)	16,700 (13,200–20,100)	93,500 (78,600–108,500)
Hawaii	14,000 (11,900–16,100)	2,000 (1,500–2,400)	12,000 (10,000–14,100)
Idaho	16,800 (14,200–19,300)	2,600 (2,000–3,200)	14,200 (11,700–16,600)
Illinois	136,600 (117,900–155,400)	18,600 (14,900–22,400)	118,000 (99,700–136,400)
Indiana	69,500 (59,600–79,400)	10,600 (8,300–13,000)	58,900 (49,200–68,500)
Iowa	31,400 (26,800–36,100)	4,400 (3,400–5,400)	27,000 (22,500–31,600)
Kansas	29,900 (25,500–34,300)	4,400 (3,400–5,400)	25,500 (21,200–29,900)
Kentucky	49,500 (42,000–57,000)	6,800 (4,900–8,700)	42,700 (35,500–50,000)
Louisiana	54,900 (46,600–63,200)	7,900 (6,200–9,700)	47,000 (38,900–55,100)
Maine	14,100 (11,900–16,300)	1,700 (1,200–2,200)	12,400 (10,300–14,600)
Maryland	59,900 (50,700–69,100)	7,900 (6,200–9,700)	52,000 (42,900–61,000)
Massachusetts	71,600 (60,900–82,300)	8,400 (6,500–10,300)	63,200 (52,600–73,700)
Michigan	108,900 (93,300–124,500)	13,600 (10,800–16,400)	95,300 (79,900–110,600)
Minnesota	53,700 (45,700–61,700)	7,400 (5,900–9,000)	46,300 (38,400–54,100)
Mississippi	35,700 (30,600–40,700)	5,100 (3,900–6,300)	30,600 (25,700–35,500)
Missouri	61,200 (52,400–70,000)	8,300 (6,500–10,100)	52,900 (44,200–61,600)
Montana	10,800 (9,100–12,600)	1,400 (1,000–1,800)	9,400 (7,700–11,100)
Nebraska	19,600 (16,600–22,500)	2,800 (2,200–3,500)	16,700 (13,800–19,600)
Nevada	31,600 (26,800–36,400)	4,400 (3,300–5,400)	27,200 (22,500–31,900)
New Hampshire	13,100 (11,100–15,200)	1,500 (1,100–1,900)	11,600 (9,600–13,700)
New Jersey	92,700 (79,100–106,200)	12,000 (9,500–14,500)	80,600 (67,300–93,900)
New Mexico	23,200 (19,800–26,500)	3,400 (2,600–4,200)	19,800 (16,400–23,100)
New York	215,200 (186,300–244,000)	26,600 (21,600–31,500)	188,600 (160,200–217,100)
North Carolina	110,100 (94,700–125,500)	15,200 (11,800–18,500)	94,900 (79,900–110,000)
North Dakota	7,300 (6,200–8,500)	1,000 (700–1,200)	6,400 (5,300–7,500)
Ohio	126,400 (109,300–143,400)	16,900 (13,600–20,300)	109,400 (92,700–126,200)
Oklahoma	41,100 (34,900–47,300)	6,400 (5,000–7,900)	34,700 (28,700–40,700)
Oregon	42,900 (36,300–49,400)	5,400 (4,100–6,800)	37,400 (31,000–43,900)
Pennsylvania	133,000 (114,600–151,400)	16,900 (13,500–20,200)	116,100 (98,000–134,200)
Rhode Island	11,100 (9,300–12,900)	1,300 (900–1,700)	9,800 (8,100–11,500)
South Carolina	53,400 (45,500–61,300)	7,100 (5,500–8,700)	46,300 (38,500–54,000)
South Dakota	8,900 (7,400–10,400)	1,300 (900–1,600)	7,600 (6,200–9,100)
Tennessee	73,900 (62,900–84,800)	10,000 (7,800–12,300)	63,800 (53,100–74,600)
Texas	292,900 (255,400–330,300)	47,200 (38,500–56,000)	245,600 (209,200–282,000)
Utah	29,300 (24,900–33,600)	5,300 (4,100–6,500)	24,000 (19,800–28,200)
Vermont	6,300 (5,300–7,300)	700 (500–900)	5,600 (4,700–6,600)
Virginia	84,800 (72,600–97,000)	11,000 (8,800–13,200)	73,800 (61,800–85,800)
Washington	74,600 (64,000–85,200)	10,200 (8,100–12,300)	64,400 (54,000–74,800)
West Virginia	21,500 (18,100–25,000)	2,500 (1,900–3,100)	19,000 (15,600–22,500)
Wisconsin	59,600 (50,800–68,300)	7,900 (6,300–9,500)	51,700 (43,100–60,300)
Wyoming	5,900 (5,000–6,800)	800 (600–1,000)	5,100 (4,200–6,000)

**Abbreviation:** CI = confidence interval.

\* Active epilepsy cases in children are estimated from the current epilepsy prevalence in children (2011–2012 National Survey of Children's Health) and the population of children, accounting for the ratios of family income to poverty thresholds.

† Active epilepsy cases in adults are estimated from the prevalence of active epilepsy (taking medication, having had a seizure in the past year, or both) in adults (2015 National Health Interview Survey) and the population of adults, accounting for the ratios of family income to poverty thresholds. The total population estimates come from the 2014 weighted person counts of the Current Population Survey, 2015 Annual Social and Economic Supplement of the civilian noninstitutionalized population living in houses and military population living in houses.

‡ Confidence interval represents only sampling uncertainty in the prevalence estimates and in the state-specific and age-specific ratios of family income to poverty thresholds.

**Summary****What is already known about this topic?**

Epilepsy is a common neurologic disorder resulting in substantial health, social, and mortality disparities.

**What is added by this report?**

In 2015, approximately 3 million U.S. adults and 470,000 children had active epilepsy. For almost all states, epilepsy prevalence estimates do not exist. Estimated numbers of active epilepsy ranged from 5,900 persons with epilepsy in Wyoming to more than 427,000 in California. The number of persons with active epilepsy increased compared with earlier years, likely because of population growth.

**What are the implications for public health practice?**

This study provides updated national estimates and the first modeled estimates of active epilepsy cases for all States. Public health practitioners, health care providers, policy makers, epilepsy researchers, and other epilepsy stakeholders including family members and people with epilepsy, can use these findings to ensure that evidence-based programs meet the complex needs of adults and children with epilepsy and reduce the disparities resulting from it.

The estimated 3 million U.S. adults with active epilepsy and 470,000 U.S. children with current epilepsy in 2015 exceed the estimated 2.3 million U.S. adults in 2010 (5) and the 450,000 U.S. children with current epilepsy in 2007 (7). The estimated increase in numbers of persons with epilepsy is not explained by age or income, because this study controlled for these known confounders. The increase is likely because of population growth over the past decade, or other unknown factors (e.g., an increased willingness to disclose one has epilepsy). The number of prevalent cases of active epilepsy by state generally mirrors the states' population distributions. The 2015 NHIS epilepsy prevalence estimate (1.2%) in this study is roughly consistent with the BRFSS estimate from 13 states (0.84% [95% confidence interval = 0.74–0.96]) that used a slightly more conservative approach assessing a 3-month seizure recall period versus 12 months (6).

The findings in this report are subject to at least four limitations. First, because these estimates depend on self-report, they might be subject to reporting bias. Second, these state estimates do not account for possible differences in seizure type, severity, or etiology. Third, underreporting associated with perceived repercussions in disclosing epilepsy (e.g., stigma or driver's license restrictions) (2) and the exclusion of institutionalized adults from the NHIS and the Census might underestimate epilepsy prevalence. Fourth, the assumption of applying national estimates to states is based on findings from

13 geographically disparate states indicating no differences in epilepsy prevalence, after accounting for multiple comparisons and sample size limitations (6). Although adjusting for age and income might account for some of the variation in prevalence across all states in this study, without available direct surveillance data on epilepsy, these estimates of active epilepsy cases in states need empirical confirmation.

Public health practitioners, health care providers, policy makers, epilepsy researchers, and other epilepsy stakeholders, including family members and people with epilepsy, can use these findings to ensure that evidence-based programs meet the complex needs of adults and children with epilepsy and reduce the disparities resulting from it.

**Conflict of Interest**

No conflicts of interest were reported.

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## Acute Flaccid Myelitis Among Children — Washington, September–November 2016

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In October 2016, Seattle Children's Hospital notified the Washington State Department of Health (DOH) and CDC of a cluster of acute onset of limb weakness in children aged ≤14 years. All patients had distinctive spinal lesions largely restricted to gray matter detected by magnetic resonance imaging (MRI), consistent with acute flaccid myelitis (AFM). On November 3, DOH issued a health advisory to local health jurisdictions requesting that health care providers report similar cases. By January 24, 2017, DOH and CDC had confirmed 10 cases of AFM and excluded two suspected cases among residents of Washington during September–November 2016. Upper respiratory tract, stool, rectal, serum, buccal, and cerebrospinal fluid (CSF) specimens were tested for multiple pathogens. Hypothesis-generating interviews were conducted with patients or their parents to determine commonalities between cases. No common etiology or source of exposure was identified. Polymerase chain reaction (PCR) testing detected enterovirus D68 (EV-D68) in nasopharyngeal swabs of two patients, one of whom also tested positive for adenovirus by PCR, and detected enterovirus A71 (EV-A71) in the stool of a third patient. *Mycoplasma* spp. immunoglobulin M (IgM) titer was elevated in two patients, but both had upper respiratory swabs that tested negative for *Mycoplasma* spp. by PCR. Clinicians should maintain vigilance for AFM and report cases as soon as possible to state or local health departments.

On October 3, 2016, DOH and CDC were notified of a boy aged 7 years who was evaluated for acute onset of limb weakness at Seattle Children's Hospital. Eight additional patients with limb weakness were reported by the same hospital during that month, including one retrospectively identified patient with onset of weakness on September 14. MRI studies indicated distinctive lesions of the spinal cord largely restricted to gray matter in all nine patients. The clinical presentation and MRI findings among patients were similar to those reported among clusters of cases in other states during 2014 (1,2). This led to ongoing routine surveillance by DOH in Washington since 2014 and the implementation of a standard case definition for AFM\* in 2015. On November 3, DOH issued a health advisory reiterating that local health jurisdictions should report suspected AFM cases.

\* <http://c.ymcdn.com/sites/www.cste.org/resource/resmgr/2015PS/2015PSFinal/15-ID-01.pdf>.

An AFM case was defined as acute onset of weakness in any limb in persons of any age and either an MRI indicating spinal cord lesions largely restricted to gray matter and spanning ≥1 vertebral segments (confirmed case) or CSF pleocytosis with a white blood cell count >5 cells/mm<sup>3</sup> (probable case). By January 24, DOH had received patient summary forms<sup>†</sup> for 12 suspected AFM cases from three health care facilities in Washington. DOH and CDC classified cases on the basis of patient summary forms, MRI reports, and MRI images. During September 2016–January 2017, among 12 suspected AFM cases, 10 were confirmed; two did not meet confirmed or probable case criteria.

Among 10 patients with confirmed AFM, date of onset of neurologic symptoms ranged from September 14 to November 9 (Figure). All patients were hospitalized for treatment of their neurologic illnesses. The median patient age was 6 years (range = 3–14 years); seven patients were male, five were white, one was American Indian/Alaska Native, one was black, and the race of three patients was unknown.

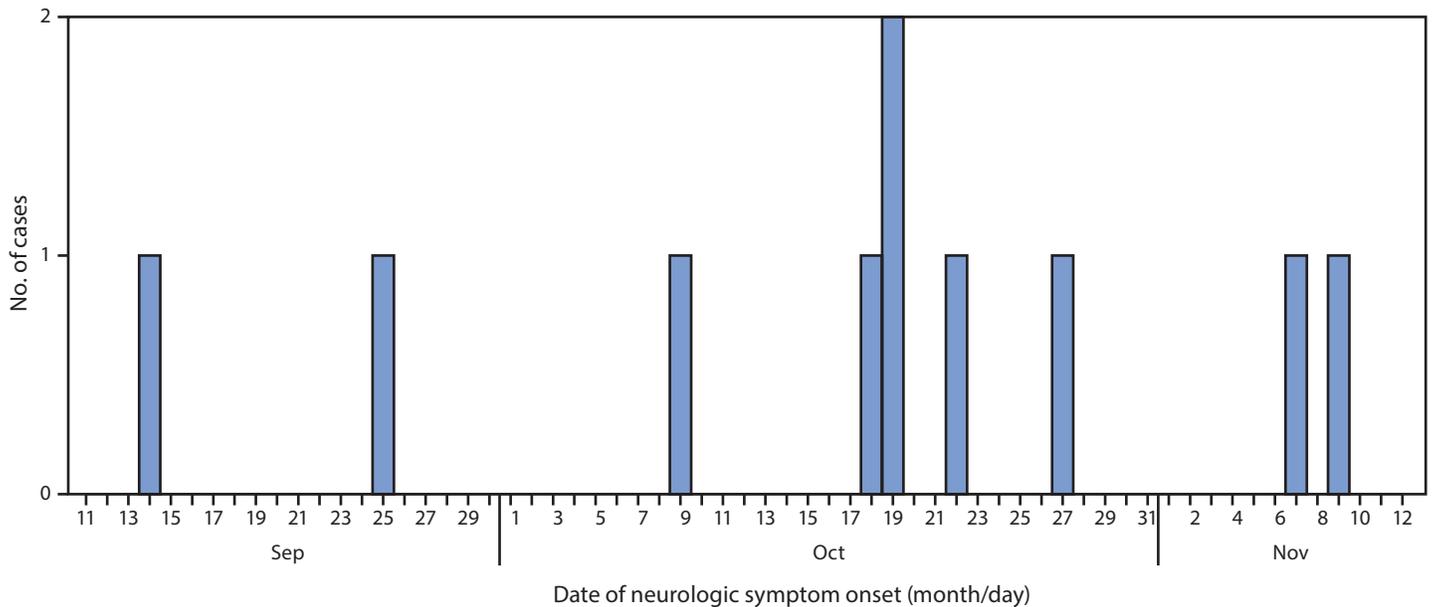
Prodromal respiratory symptoms, gastrointestinal symptoms, or both were reported for eight patients. The median interval from onset of respiratory symptoms to onset of neurologic symptoms was 7 days (range = 1–12 days), and from onset of gastrointestinal symptoms to onset of neurologic symptoms was 2 days (range = 0–12 days). For nine patients, fever was reported in the 4 weeks before the onset of limb weakness. All patients were reported to have been previously healthy, although one patient had an asymptomatic Chiari I malformation. No patient had a reported rash or had previously received immunosuppressing agents. According to patients' vaccination records, all but one had been vaccinated according to Advisory Committee on Immunization Practices recommendations.<sup>§</sup> The median interval between receipt of the last vaccination and onset of neurologic symptoms was 1.9 years (range = approximately 2 months–7 years).

All patients initially had acute onset of weakness in one or more limbs. At the peak of neurologic symptoms, eight patients had more than one involved limb, two had three involved limbs, and four patients had all limbs involved. Other neurologic signs included acute neck weakness (one patient),

<sup>†</sup> <https://www.cdc.gov/acute-flaccid-myelitis/hcp/data.html>.

<sup>§</sup> <https://www.cdc.gov/vaccines/schedules/hcp/child-adolescent.html>.

**FIGURE. Number of confirmed cases of acute flaccid myelitis (N = 10), by date of onset of neurologic symptoms — Washington, September 14–November 9, 2016**



bladder or bowel incontinence (five patients), and cranial nerve dysfunction, including facial weakness or diplopia (three patients). The severity of symptoms in one patient limited parts of the neurologic examination that require patient participation; this patient also required ventilator support. The median duration of hospitalization was 7 days (range = 4–35 days). Four patients received intravenous immunoglobulin and eight patients received intravenous steroids with an oral taper. Seven patients underwent rehabilitation therapy, four as inpatients and three as outpatients. Among nine patients for whom follow-up information was available 1.5–3 months after discharge, five had mild to no residual deficits, three had moderate improvement with residual limb weakness, and one had moderate improvement, but was not ambulatory without assistance. No deaths among confirmed cases occurred.

CSF collection was attempted in all patients; however, contamination with blood rendered one patient's sample uninterpretable. Seven of nine patients had pleocytosis (median = 163 cells/mm<sup>3</sup>; range = 13–395 cells/mm<sup>3</sup>; reference = 0–5 cells/mm<sup>3</sup>). CSF protein range was 19–99 mg/dL (median = 57.5 mg/dL; reference <40 mg/dL). Nine patients received an MRI of the full spinal cord, and one patient received an MRI of the cervical and upper thoracic region. All patients received a brain MRI. All patients had lesions at the cervical cord level; nine patients had thoracic cord lesions and five patients had lesions at the conus medullaris level (termination of the spinal cord at approximately lumbar [L1/L2]). No MRI reports noted enhancement of the cauda equina (nerve roots descending below the end of the termination of

the spinal cord) in any patient. Two patients had supratentorial (the region of the brain containing the cerebrum) lesions, one had subcortical lesions, and three had brainstem lesions. Lesions were predominantly confined to the gray matter in nine patients and to the gray and white matter in one patient. Lesions in two patients displayed enhancement with contrast media, which was used in all patients.

Hypothesis-generating interviews were conducted with seven patients or their parents; three patients or their families declined to be interviewed. Questions covered all activities undertaken 2 months before onset of prodromal symptoms, including contact with sick persons, travel within and outside the United States, and exposures to environmental sources. Household members of four patients reported upper respiratory symptoms during the patient's prodromal illness. No other household members developed AFM. No patient had traveled outside four states (California, Idaho, Oregon, and Washington). Six patients had participated in open freshwater activities (lakes and rivers), but these took place at separate locations. The patients were residents of six counties in Washington; two patients residing outside of the Seattle metropolitan area lived within 11 miles of each other, but otherwise no spatial clustering was observed. No common environmental exposures were identified.

Specimens were available for all patients, including upper respiratory tract (eight patients), CSF (10), stool or rectal swab (eight), buccal swab (one), and serum (10). Specimens were tested at hospital laboratories in Washington and at CDC's Picornavirus Laboratory for multiple pathogens, including

adenovirus, cytomegalovirus, enteroviruses (including poliovirus), Epstein-Barr virus, herpes simplex virus, human herpes virus 6, influenza, parechoviruses, varicella zoster virus, West Nile virus, and fecal and respiratory bacteria.

EV-D68 was detected by PCR testing in nasopharyngeal swabs of two patients, one of whom also tested positive for adenovirus by PCR. EV-A71 was detected by PCR testing in the stool of a third patient. PCR testing of the CSF from the patient with EV-A71 was also indeterminate for Epstein-Barr virus and human herpes virus 6. Two patients had elevated *Mycoplasma* spp. IgM titers; their IgG titers were within normal range and results of testing upper respiratory swabs were negative for *Mycoplasma* spp. by PCR. Test results for all specimens were negative for poliovirus.

### Discussion

Among a cluster of 10 cases of AFM among children in Washington during September–November 2016, no common etiology or source of exposure was identified. During the preceding year (August 2015–August 2016), no confirmed cases and only one probable case were reported in Washington, consistent with the limited number of AFM cases reported nationally during that period (3). In 2014, only two AFM cases were reported in Washington and 120 cases nationally (4). The demographic characteristics and clinical presentation of patients in this cluster are similar to those of previously reported cases (1,2,4,5). However, among patients in previous clusters for whom follow-up information was available (2,4,5), a larger proportion required ventilator support, reported persistent motor deficits, or were transferred to a rehabilitation facility.

EV-A71 and EV-D68, which were identified in three patients in this cluster, have been associated with outbreaks of neurologic disease (6,7). However, as in previously reported clusters of AFM, no pathogen was consistently isolated from all specimens tested (1,2,8). The prodrome of fever and respiratory or gastrointestinal symptoms, combined with clinical outcomes consistent with reported cases of neurologic disease from enterovirus and other neurotropic virus infection, suggest that the etiology of these AFM cases is infectious (4,9). Another possible etiology might be a postinfectious phenomenon, in which the viral infection leads to a delayed immune response and for which laboratory evidence of the involved pathogen might be lacking at the time of weakness onset (9). This underscores the importance of timely reporting of cases and expanded AFM testing to include both infectious and noninfectious causes.

The findings in this report are subject to at least two limitations. First, because reporting of AFM is voluntary, incidence in the United States is unknown. Second, because of the clinical

### Summary

#### What is known about this topic?

Acute flaccid myelitis (AFM) is a neurologic condition with newly standardized clinical criteria that aid in its recognition. AFM is characterized by acute onset of flaccid limb weakness and lesions in the gray matter of the spinal cord evident on magnetic resonance imaging. Investigation of previously reported clusters did not identify a specific etiology, although during 2014, a temporal association between clusters of AFM and increased incidence of enterovirus-D68 (EV-D68) infections was reported. Because reporting is voluntary, the range of clinical signs, severity, and incidence in the United States is difficult to determine.

#### What is added by the report?

During September–November 2016, 10 confirmed cases of AFM were reported in Washington. No common etiology or source of exposure was identified. Enterovirus-A71 was detected in one patient and EV-D68 in two patients, one of whom also tested positive for adenovirus. *Mycoplasma* spp. immunoglobulin M titer was detected in two patients, but polymerase chain reaction testing of an upper respiratory swab was negative in both.

#### What are the implications for public health practice?

Clinicians should remain vigilant for AFM and report cases to state or local health departments as soon as possible. Timely collection of specimens for laboratory testing and expansion of testing to include infectious and noninfectious causes might help uncover a common etiology within a cluster.

similarity between AFM and other neurologic conditions such as idiopathic transverse myelitis and acute inflammatory demyelinating polyneuropathy subtype of Guillain-Barré syndrome, cases might be misdiagnosed and not reported to state and local health departments. AFM typically leads to chronically depressed reflexes, and sensory findings are not typically as discrete as in transverse myelitis, or progressively ascending as in acute inflammatory demyelinating polyneuropathy. AFM lesions indicated on an MRI are more often confined to the gray matter than lesions associated with transverse myelitis, and can also include nerve root enhancement and cranial nerve involvement (5,10).

Clinicians, specifically pediatric neurologists, should maintain vigilance for AFM. They are encouraged to report cases as soon as possible to state or local health departments to add to information regarding clinical signs, severity, and illness prognosis.

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### Conflict of Interest

No conflicts of interest were reported.

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# Recommendations for HIV Screening of Gay, Bisexual, and Other Men Who Have Sex with Men — United States, 2017

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CDC's 2006 recommendations for human immunodeficiency virus (HIV) testing state that all persons aged 13–64 years should be screened for HIV at least once, and that persons at higher risk for HIV infection, including sexually active gay, bisexual, and other men who have sex with men (MSM), should be rescreened at least annually (1). Authors of reports published since 2006, including CDC (2), suggested that MSM, a group that is at highest risk for HIV infection, might benefit from being screened more frequently than once each year. In 2013, the U.S. Preventive Services Task Force (USPSTF) found insufficient evidence to specify an HIV rescreening interval but recommended annual screening for MSM as a reasonable approach (3). However, some HIV providers have begun to offer more frequent screening, such as once every 3 or 6 months, to some MSM. A CDC work group conducted a systematic literature review and held four expert consultations to review programmatic experience to determine whether there was sufficient evidence to change the 2006 CDC recommendation (i.e., at least annual HIV screening of MSM in clinical settings). The CDC work group concluded that the evidence remains insufficient to recommend screening more frequently than at least once each year. CDC continues to recommend that clinicians screen asymptomatic sexually active MSM at least annually. Each clinician can consider the benefits of offering more frequent screening (e.g., once every 3 or 6 months) to individual MSM at increased risk for acquiring HIV infection, weighing their patients' individual risk factors, local HIV epidemiology, and local testing policies.

HIV testing is the critical first step in making HIV-infected persons aware of their status, so that they can obtain treatment and prevent transmission of HIV. In 2014, CDC estimated that 15% of all persons living with HIV in the United States had undiagnosed infections (4). Early HIV care and adherence to antiretroviral therapy (ART) prolong life and decrease the chances of HIV transmission (5). The increasing availability of antigen-antibody HIV tests means that a greater number of infections can be detected in the highly infectious, acute stage of infection (6). The potential benefits of early detection and treatment of HIV were the driving force behind CDC's initiative to assess the benefits and harms associated with more frequent screening of MSM. This policy note describes the results of that initiative.

## Systematic Review

A CDC work group of federal employees comprising a diverse group of epidemiologists, clinicians, behavioral scientists, health policy experts, and health economists was convened. To identify studies comparing annual versus more frequent screening among MSM, the CDC work group conducted a systematic literature review, using methods adapted from the Guide for Community Preventive Services (7,8), and convened four consultations with 24 external experts to obtain their individual input on the programmatic and scientific evidence. During 2013–2014, and updated in January 2015, the CDC work group conducted a systematic review of published studies indexed in MEDLINE, EMBASE, PsycINFO, and CINAHL. The search was restricted to articles that 1) were published during 2005–2014 (last search conducted in January 2015); 2) described analyses conducted in the United States, Canada, Australia, New Zealand, and Western Europe; and 3) contained the following search terms: HIV seropositivity, HIV infection, AIDS serodiagnosis, sexually transmitted diseases/infections, men who have sex with men (MSM), high risk, test, screen. Included articles provided information on one of four outcomes of interest: 1) health benefits to individual MSM being screened or to the community (e.g., averted secondary HIV infections); 2) harms to individual MSM (stigma or out-of-pocket costs); 3) acceptability (MSM attitudes toward more frequent screening); or 4) feasibility (barriers to or facilitators of state or local screening). Included studies were restricted to those conducted in clinical settings. A manual search of gray literature was also conducted.

The CDC work group reviewed 6,479 abstracts resulting from the automated search, 111 of which met the initial screening inclusion criteria and were reviewed in full. Three members of the CDC work group, working in overlapping pairs, applied inclusion criteria to these studies, rating each study for outcome (benefits, harms, acceptability, or feasibility). They used a quantitative study assessment tool to note key findings. Discrepancies were resolved by a third reviewer who was not a member of the original pair (7,8).

Thirteen studies met the inclusion criteria and were evaluated on quality of evidence (9). For each of the four study outcomes, CDC HIV testing experts then evaluated the quality of evidence to determine design suitability (high, moderate, or

low), execution (good, fair, or poor), and consistency of study results, with one exception: the eight mathematical modeling studies were not rated on quality of execution because of the lack of a grading system appropriate for the different mathematical model types included.

Overall, the quality of studies was low. Eleven studies addressed health or economic benefits of more frequent screening compared with annual screening. Eight of these were mathematical models that the CDC work group classified as having low suitability because of uncertainty about the validity of the parameter estimates and questions about the models' generalizability. Two studies addressed intervals between HIV screening or diagnostic tests in clinical settings, but did not directly address the acceptability of more frequent than annual HIV screening among asymptomatic MSM. No studies addressed harms associated with, or the feasibility of, conducting more frequent HIV screening in clinical settings in the United States. Additional details about these studies can be found elsewhere (9).

After deliberations that involved discussion, consensus building, and voting, the CDC work group concluded that insufficient evidence exists in the published and unpublished literature to warrant changing CDC's current recommendation to offer HIV screening at least annually to all sexually active MSM.

### Expert Consultation Series Results

During August–December 2014, the CDC work group convened a series of consultations with external subject matter experts, including clinicians, epidemiologists, academic researchers, health department policy and program staff members, and members of the MSM community, to 1) obtain their individual input on the results of the systematic review and preliminary conclusions; 2) obtain the opinions and experiences of experts from three public-sector HIV screening programs that provided more frequent than annual HIV screening to MSM; and 3) identify studies missed in the literature review or data that could be analyzed in the future to inform recommendations about HIV screening frequency.

Postconsultation analysis of the individual feedback from experts revealed that most believed the literature was insufficient to conclude that more frequent screening had demonstrated benefits over annual screening but that the scientific and programmatic evidence suggested that some MSM would be willing to be screened more frequently. Experts from health departments already implementing more frequent than annual screening described benefits of their programs, including decreases in the proportion of MSM with undiagnosed HIV infection. Experts also individually agreed that the estimates from the mathematical models suggest a benefit to

more frequent screening, particularly in jurisdictions providing prompt, high-quality access to HIV medical care, where early treatment with ART decreases infectiousness and would likely decrease the number of new HIV infections in sex or drug-using partners. In addition, individual experts stressed the importance of the cost-effectiveness modeling studies, which estimated that more frequent screening, compared with annual screening, would be more cost-effective by averting new HIV infections (incremental cost-effectiveness ratio, range = cost-saving – \$138,200/quality-adjusted life year) (9). Finally, most experts stated that mathematical models do not provide sufficient evidence to warrant by themselves a change in the guideline, because of limitations in their study design, and that additional studies are needed to update the current recommendation.

### Recommendations

CDC concludes that the evidence, programmatic experience, and expert opinions are insufficient to warrant changing the current recommendation (annual screening for MSM) to more frequent screening (every 3 or 6 months). Therefore, CDC's 2006 recommendation for HIV screening of MSM is unchanged; providers in clinical settings should offer HIV screening at least annually to all sexually active MSM. Clinicians can also consider the potential benefits of more frequent HIV screening (e.g., every 3 or 6 months) for some asymptomatic sexually active MSM based on their individual risk factors, local HIV epidemiology, and local policies (9). Additional research is needed to establish the individual- or community-level factors that might increase the risk for HIV acquisition for MSM and merit more frequent HIV screening. For MSM who are prescribed preexposure prophylaxis, HIV testing every 3 months and immediate testing whenever signs and symptoms of acute HIV infection are reported (10) is indicated. MSM who experience a specific high-risk sexual exposure or have symptoms of recent HIV infection should seek immediate HIV testing, and clinicians should be alert for the symptoms of acute HIV infection and provide appropriate diagnostic testing.

CDC encourages researchers to conduct studies to evaluate the benefits and harms of more frequent screening for MSM. Findings from these studies will inform future assessment of recommendations. CDC will continue to monitor the evidence on the effectiveness of various HIV screening intervals and consider the need to revise current recommendations in light of new evidence.

### Conflict of Interest

No conflicts of interest were reported.

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## Notes from the Field

### Increase in Coccidioidomycosis — California, 2016

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Coccidioidomycosis, or Valley Fever, is an infectious disease caused by inhalation of *Coccidioides* spp. spores (1). This soil-dwelling fungus is endemic in the southwestern United States, with most (97%) U.S. cases reported from Arizona and California (1,2). Following an incubation period of 1–3 weeks, symptomatic patients most often experience self-limited, influenza-like symptoms, but coccidioidomycosis also can lead to severe pulmonary disease and to rare cases of disseminated disease, including meningitis (1). Those at increased risk for severe disease include persons of African or Filipino descent, pregnant women, adults in older age groups, and persons with weakened immune systems (1). In 2016, a large increase in coccidioidomycosis incidence was observed in California compared with previous years (3). Using data reported by health care providers and laboratories via local health departments to the California Department of Public Health as of May 9, 2017, incidence rates were calculated by estimated year of illness onset as the number of confirmed coccidioidomycosis cases per 100,000 population (3). Estimated year of illness onset was extracted from the closest date to the time when symptoms first appeared for each patient. From 1995, when coccidioidomycosis became an individually reportable disease in California, to 2009, annual incidence rates ranged from 1.9 to 8.4 per 100,000, followed by a substantial increase to 11.9 per 100,000 in 2010 and a peak of 13.8 per 100,000 in 2011 (Figure). Annual rates decreased during 2012–2014, but increased in 2016 to 13.7 per 100,000, with 5,372 reported cases, the highest annual number of cases in California recorded to date.

Coccidioidomycosis incidence rates increased disproportionately in counties considered to have endemic disease. Most cases in 2016 were in residents of the Central Valley and Central Coast regions, with 42% (2,238 cases, rate 251.7 per 100,000) reported from Kern County and 28% (1,515 cases, rate 54.5 per 100,000) from six other counties (Fresno, Kings, Madera, San Joaquin, San Luis Obispo, and Tulare) (3). From 2015 to 2016, the combined incidence from these seven counties increased 109%, from 48.9 per 100,000 (2015) to 102.3

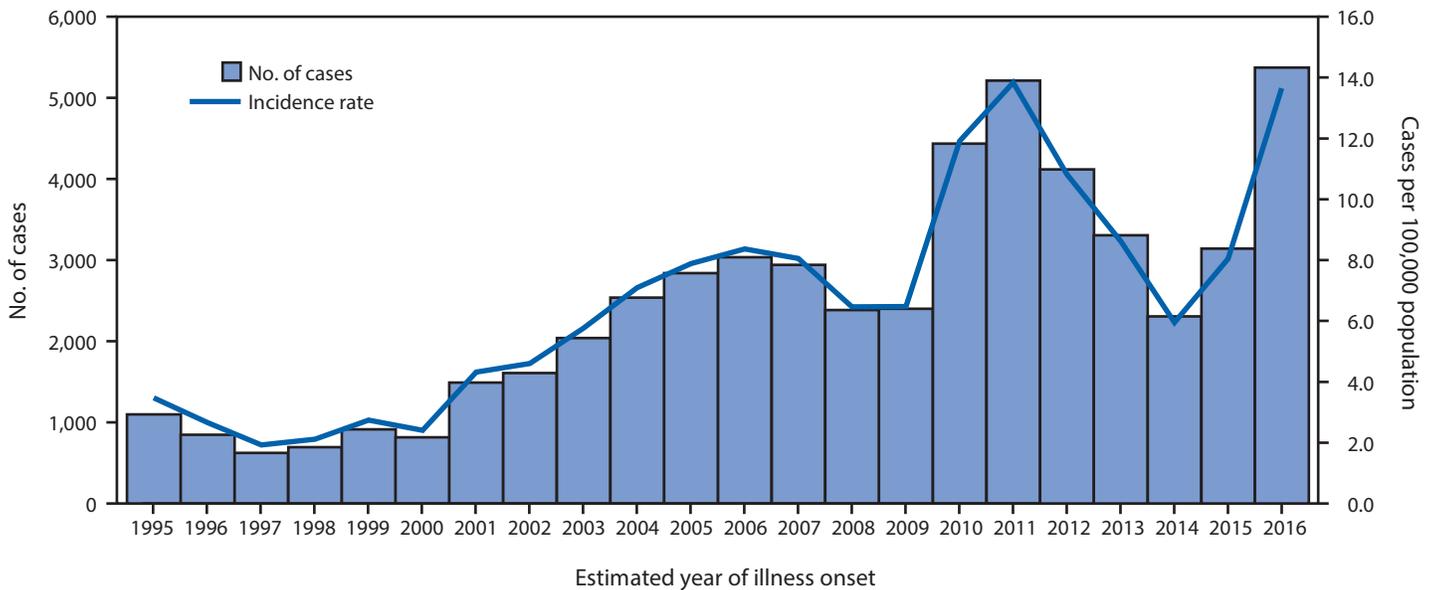
(2016), while the rate in the remaining counties in California increased by 18% (from 3.8 to 4.5 per 100,000).

Reported 2016 incidence was highest among persons aged 40–59 years (18.8 per 100,000), compared with rates in persons aged <20 years (5.6), 20–39 years (14.9), 60–79 years (16.4) and ≥80 years (13.1). However, the sharpest increases in incidence from 2015 to 2016 occurred in persons aged <20 years (134%) and 20–39 years (90%); increases were less pronounced in persons aged 40–59 years (64%), 60–79 years (40%) and ≥80 years (35%). Rates were higher among males (17.3 per 100,000) than among females (10.0). Incidence rates by race and ethnicity were not calculated because these data were missing for approximately one third (32.7%) of reports.

Although annual coccidioidomycosis incidence rates in California and Arizona typically follow similar trends, Arizona reported a decrease in the rate from 2015 to 2016 (from 112.8 to 89.3 per 100,000) (2,4,5). In the remaining states where coccidioidomycosis was reportable in both 2015 and 2016, preliminary data show that incidence remained stable at 0.5 per 100,000 in both years.

The reasons for the increased incidence of coccidioidomycosis in California in 2016, particularly in the Central Valley and Central Coast regions, are not known, but climatic and environmental factors favorable to *Coccidioides* proliferation and airborne release might have contributed, including rainfall after several years of drought and soil disturbance resulting from construction (2). To decrease the risk for infection, persons living, working, or traveling in areas where *Coccidioides* is endemic, especially those at increased risk for severe disease, should limit exposure to outdoor dust as much as possible, including staying inside and keeping windows and doors closed during windy weather and dusty conditions (3). Previous outbreaks of coccidioidomycosis have occurred among persons working outdoors in areas where *Coccidioides* is endemic, including construction workers; recommendations for reducing the risk for infection on construction worksites include using personal protective respiratory equipment, dust suppression, and worker education (6,7). Health care providers should be alert for coccidioidomycosis among patients who live in or have traveled to areas where the disease is endemic, especially those who work or participate in activities where dust is generated.

FIGURE. Number of coccidioidomycosis cases and incidence rate, by estimated year of illness onset\* — California, 1995–2016



\* Estimated year of illness onset was extracted from the closest date to the time when symptoms first appeared for each patient.

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### Conflict of Interest

No conflicts of interest were reported.

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## Notes from the Field

### Zika Virus-Associated Neonatal Birth Defects Surveillance — Texas, January 2016–July 2017

Noemi Borsay Hall, PhD<sup>1,2</sup>; Kelly Broussard, MPH<sup>2</sup>; Nicole Evert, MS<sup>2</sup>; Mark Canfield, PhD<sup>2</sup>

On November 28, 2016, the Texas Department of State Health Services (Texas DSHS) reported its first confirmed case of local mosquito-borne Zika virus transmission in the city of Brownsville, located in south Texas along the U.S.-Mexico border. Zika virus infection during pregnancy has been linked to adverse congenital outcomes including microcephaly, neural tube defects, early brain malformations, structural eye abnormalities, congenital deafness, and limb contractures (1). On January 1, 2016, Texas DSHS established enhanced surveillance to identify women with laboratory evidence of possible Zika virus infection during pregnancy and suspected cases of Zika virus-associated birth defects among completed pregnancies.

Relevant epidemiologic information, comprising arboviral disease case investigation findings (including pregnancy status and likely location of exposure) and laboratory test results is collected and reviewed by the Texas DSHS Zoonosis Control Branch as a part of routine arboviral disease surveillance. Each week, the Zoonosis Control Branch shares a line list of pregnant women with laboratory evidence of possible Zika virus infection and their reported pregnancy outcomes with the Texas DSHS Birth Defects Epidemiology and Surveillance Branch. Among possible cases with no reported pregnancy outcome, recent birth certificate data are searched for the reported pregnant woman's name and birth date to determine whether a live birth has occurred. Birth Defects Epidemiology and Surveillance Branch staff members review neonatal medical records of all babies born to women with possible Zika virus infection during pregnancy to identify all documented birth defects.

During January 1, 2016–July 31, 2017, a total of 219 pregnant women with laboratory evidence of possible recent Zika virus infection were identified in Texas, including 49 (22%) with laboratory-confirmed Zika virus infection (Table). One woman was infected in Texas; all others were exposed outside the United States and its territories. Among the 219 pregnancies, outcomes were recorded for 185 (84%), including 182 live-born infants and three pregnancy losses that occurred at any time during gestation. Among the remaining 34 pregnant women, 20 have an estimated due date which has not yet passed, four have an estimated due date which has passed but no pregnancy outcomes have been reported, and for 10, there was no reported estimated due date. All recorded completed

pregnancies were reviewed by the Zoonosis Control and Birth Defects Epidemiology and Surveillance Branches to ascertain Zika virus testing status and to identify any birth defects. Zika virus testing was completed for 80 (43%) of the 185 infants or fetuses, and Zika virus-associated birth defects were documented in 15 (8%) pregnancies (14 live-born infants and one fetal loss), including six (17%) of the 36 infants or fetal losses delivered by women with laboratory-confirmed Zika virus infection. Ten infants or fetuses had microcephaly; five of those with microcephaly had additional birth defects, including holoprosencephaly, hydranencephaly, craniosynostosis, and clubfeet. Zika virus-associated birth defects identified in the remaining three infants included holoprosencephaly, cataracts, and ventral pons hypoplasia.

Zika virus testing was not completed for 105 (57%) infants or fetuses; including three pregnancy losses and 10 live-born infants for whom only a placental or cord blood specimen was tested. In the absence of other evidence, testing of cord blood is insufficient to determine an infant's infection status (2). Placental testing only provides information regarding possible maternal Zika virus infection and cannot confirm or exclude congenital Zika virus infection (3). Specimens from 13 infants were unsatisfactory for testing (specimens arrived at an incorrect temperature) or were of insufficient quantity to conduct testing. For the remaining 79 infants, no reason was reported for not conducting Zika virus testing.

The occurrence of travel-related Zika virus infections, combined with the threat of local transmission in Texas, indicates a need for continued surveillance for birth defects associated with Zika virus infection. This analysis found that only 43% of identified infants or fetuses for whom testing was indicated received testing. Efforts to increase the frequency of collecting and testing of specimens from infants born to mothers with laboratory evidence of possible recent Zika virus infection are needed. Physicians caring for newborn infants need to be aware of the Zika testing status of the mother, particularly in geographic locations with high potential for local mosquito-borne transmission. Serum specimens are strongly preferred to placenta or cord blood specimens for infant testing, and should be collected soon after birth (2). Neuroimaging before hospital discharge is also recommended for infants born to mothers with evidence of Zika virus infection during pregnancy to detect subtle findings (e.g., calcifications) that indicate congenital Zika infection (2). Affected infants should be referred for appropriate clinical and intervention services (2).

**TABLE. Zika virus-associated neonatal birth defects among live-born infants and fetal losses delivered by pregnant women with evidence of Zika virus infection during pregnancy — Birth Defects Epidemiology and Surveillance Branch, Texas Department of State Health Services, January 2016–July 2017**

Characteristic	No. (%)		
	Total	Laboratory evidence of possible recent maternal Zika virus infection*	Laboratory-confirmed maternal Zika virus infection†
Pregnant women	219 (100)	170 (78)	49 (22)
Completed pregnancies	185 (84)	149 (81)	36 (19)
Liveborn infants <sup>§</sup>	182 (98)	147 (81)	35 (19)
Pregnancy loss <sup>§</sup>	3 (2)	2 (67)	1 (33)
Zika-associated birth defects <sup>§</sup>	15 (8)	9 (60)	6 (40)
Microcephaly	10 <sup>¶</sup> (67)	6 (60)	4 (40)
Other Zika-associated birth defects	5** (33)	3 (60)	2 (40)
Infant/Fetus received testing for Zika	80 <sup>††</sup> (43)	57 (71)	23 (29)

\* Recent Zika virus infection detected by a positive Zika virus RNA Nucleic Acid Test (NAT) (e.g., reverse transcription-polymerase chain reaction [RT-PCR]) on any maternal, placental, or fetal/infant specimen or detection of recent Zika virus infection or recent unspecified flavivirus infection by serologic tests on a maternal or infant specimen (i.e., either positive or equivocal Zika virus immunoglobulin M [IgM] and Zika virus plaque reduction neutralization test [PRNT] titer  $\geq 10$ , regardless of dengue virus PRNT value or negative Zika virus IgM, and positive or equivocal dengue virus IgM, and Zika virus PRNT titer  $\geq 10$ , regardless of dengue virus PRNT titer). Those persons who meet lab-confirmed criteria are not represented among those who have laboratory evidence of possible recent maternal Zika virus infection.

† Zika virus RNA documented by a positive NAT in a maternal, placental, or fetal/infant specimen or detection of recent Zika virus infection by serologic tests on a maternal or infant specimen (i.e., Zika virus IgM was positive or equivocal and Zika virus PRNT titer was  $\geq 10$  and dengue virus PRNT was  $< 10$ ).

§ Among completed pregnancies, including live-born infants and fetal losses at any time during gestation.

¶ Five of these infants had additional birth defects including holoprosencephaly, hydranencephaly, craniosynostosis, and clubfeet.

\*\* Includes holoprosencephaly, ventriculomegaly, cataracts, choroid plexus cysts, and ventral pons hypoplasia.

†† Testing not completed for 105 (57%) infants or fetuses, including three pregnancy losses, and 10 live-born infants for whom only a placental or cord blood specimen was tested; 13 specimens could not be tested because the specimens were unsatisfactory, and for the remaining 79 infants, the reason for not testing was not provided.

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### Conflict of Interest

No conflicts of interest were reported.

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## Announcement

### Fungal Disease Awareness Week — August 14–18, 2017

In 2017, CDC initiated a national observance, Fungal Disease Awareness Week, to increase awareness about fungal diseases, which can cause severe illness but frequently go undiagnosed. Awareness is one of the most important means to reduce delays in diagnosis and treatment, which can lead to better health outcomes and save lives.

The theme of this year's observance is "Think Fungus," and aims to encourage the public and clinicians to consider the possibility of a fungal infection if a patient's symptoms are not improving with treatment. There are many types of fungal diseases. Immunocompromised persons are more likely to acquire serious fungal diseases, but some types of fungal infections occur in otherwise healthy persons.

Fungal diseases are an increasing problem worldwide, although the exact prevalence is difficult to quantify (1). In the United States, coccidioidomycosis (often called "Valley fever") is particularly concerning; although approximately 10,000 cases are reported each year, it is likely that many more cases go undiagnosed, with an estimated 150,000 infections annually (2). This issue of *MMWR* includes a report on a substantial increase in coccidioidomycosis cases in California in 2016 (3). *Candida*, a common cause of mucosal and skin infections, is an important cause of bloodstream infections in hospitalized patients (4). Antifungal resistance is a growing public health problem, particularly in *Candida* and *Aspergillus* infections,\*

compounded by the recent emergence of *Candida auris*, a multidrug-resistant yeast that spreads in health care facilities (5). Resistant infections lead to longer hospital stays, higher medical costs, and more deaths. Globally, cryptococcal meningitis, histoplasmosis, and *Pneumocystis* pneumonia remain important causes of death in patients with human immunodeficiency virus infections and acquired immunodeficiency syndrome. Additional information about Fungal Disease Awareness Week is available at <https://www.cdc.gov/fungal/awareness-week.html>.

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\* <https://www.cdc.gov/drugresistance/threat-report-2013/index.html>.

## Erratum

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### Vol. 66, No. 25

In the report “Multistate Outbreak of *Salmonella* Anatum Infections Linked to Imported Hot Peppers — United States, May–July 2016,” on page 663, the footnote (¶) at the bottom of the page should have read “Louisiana (**two**).”

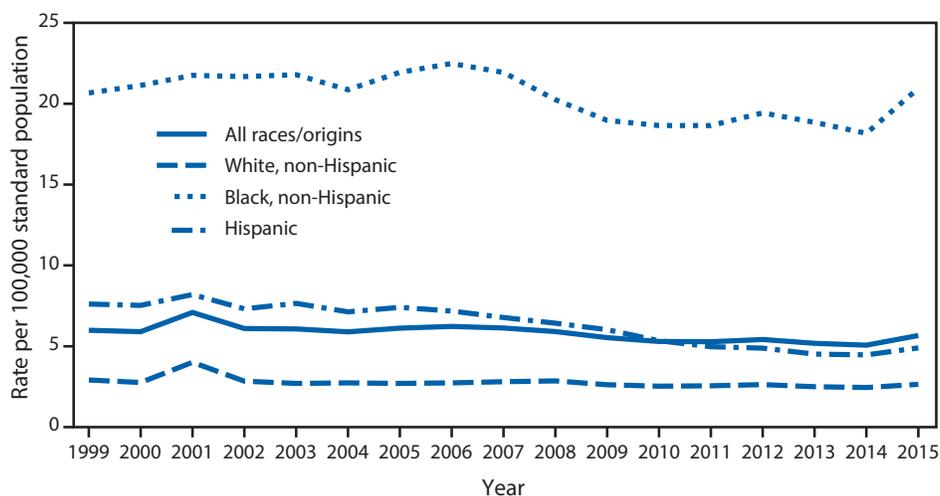
### Vol. 66, No. 29

In the announcement on World Hepatitis Day on page 794, the title should have read “World Hepatitis Day — **July 28**, 2017.”

## QuickStats

FROM THE NATIONAL CENTER FOR HEALTH STATISTICS

### Age-Adjusted Rates for Homicides,\* by Race/Ethnicity† — United States, 1999–2015



\* Deaths from homicide were identified using the *International Classification of Diseases, Tenth Revision* underlying cause of death codes \*U01–\*U02,X85–Y09,Y87.1.

† Of the persons who died as a result of the homicide.

During 1999–2014, a general decline in homicide trends for non-Hispanic white, non-Hispanic black, and Hispanic populations occurred, followed by a significant increase in the rates for all three groups between 2014 and 2015. In 2015, homicide rates were 5.7 deaths per 100,000 for the total population, 20.9 for non-Hispanic blacks, 4.9 for Hispanics, and 2.6 for non-Hispanic whites. During 1999–2015, rates of deaths from homicide were highest for non-Hispanic blacks and lowest for non-Hispanic whites and declined the most for Hispanics.

**Source:** CDC/National Center for Health Statistics, National Vital Statistics System, 1999–2015, Mortality. CDC Wonder online database. <https://wonder.cdc.gov/ucd-icd10.html>.

**Reported by:** Arialdi Miniño, MPH, [aminino@cdc.gov](mailto:aminino@cdc.gov), 301-458-4376.

For more information on this topic, CDC recommends the following link: <https://www.cdc.gov/violenceprevention/index.html>.

## Morbidity and Mortality Weekly Report

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