

Quitting Smoking Among Adults — United States, 2000–2015

Stephen Babb, MPH¹; Ann Malarcher, PhD¹; Gillian Schauer, PhD¹; Kat Asman, MSPH¹; Ahmed Jamal, MBBS¹

Quitting cigarette smoking benefits smokers at any age (1). Individual, group, and telephone counseling and seven Food and Drug Administration–approved medications increase quit rates (1–3). To assess progress toward the *Healthy People 2020* objectives of increasing the proportion of U.S. adults who attempt to quit smoking cigarettes to ≥80.0% (TU-4.1), and increasing recent smoking cessation success to ≥8.0% (TU-5.1),* CDC assessed national estimates of cessation behaviors among adults aged ≥18 years using data from the 2000, 2005, 2010, and 2015 National Health Interview Surveys (NHIS). During 2015, 68.0% of adult smokers wanted to stop smoking, 55.4% made a past-year quit attempt, 7.4% recently quit smoking, 57.2% had been advised by a health professional to quit, and 31.2% used cessation counseling and/or medication when trying to quit. During 2000–2015, increases occurred in the proportion of smokers who reported a past-year quit attempt, recently quit smoking, were advised to quit by a health professional, and used cessation counseling and/or medication ($p < 0.05$). Throughout this period, fewer than one third of persons used evidence-based cessation methods when trying to quit smoking. As of 2015, 59.1% of adults who had ever smoked had quit. To further increase cessation, health care providers can consistently identify smokers, advise them to quit, and offer them cessation treatments (2–4). In addition, health insurers can increase cessation by covering and promoting evidence-based cessation treatments and removing barriers to treatment access (2,4–6).

NHIS is an annual, nationally representative, in-person survey of the noninstitutionalized U.S. civilian population. The NHIS Sample Adult core questionnaire is administered to a randomly selected adult (referred to as the sample adult) aged ≥18 years in

* Objectives TU-4.1 and TU-5.1. <https://www.healthypeople.gov/2020/topics-objectives/topic/tobacco-use/objectives>.

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each sampled family. NHIS sample sizes and final response rates for sample adults for 2000, 2005, 2010, and 2015 were 32,374 (response rate = 72.1%), 31,428 (69.0%), 27,157 (60.8%), and 33,672 (55.2%), respectively. Current and former smoking were defined according to *Healthy People 2020* measures.[†] Persons attempting to quit included current smokers who stopped smoking for >1 day during the 12 months before the interview because they were trying to quit and former smokers who had quit during the past year. Former smokers who last smoked 6–12 months ago were considered to have achieved recent cessation success. Every 5 years, a supplemental cancer-control questionnaire is administered to NHIS sample adult respondents; the questionnaire contains questions on interest in quitting smoking, receipt of a health professional's advice to quit, and use of cessation counseling and/or medication. Data were adjusted for differences in the probability of selection and nonresponse, and were weighted to provide nationally representative estimates. Logistic regression was conducted to analyze trends during 2000–2015. Both linear and quadratic terms were initially applied to all models. If the quadratic term was not significant, the linear model was used.

[†]To determine smoking status, respondents were asked, "Have you smoked at least 100 cigarettes in your entire life?" Those who answered "yes" were asked, "Do you now smoke cigarettes every day, some days, or not at all?" Current smokers were those who had smoked at least 100 cigarettes during their lifetime and, at the time of the interview, reported smoking every day or some days. Former smokers were those who reported smoking at least 100 cigarettes during their lifetime but currently did not smoke. <http://www.cdc.gov/nchs/nhis/data-questionnaires-documentation.htm>.

In 2015, 68.0% of all current smokers reported that they wanted to stop smoking completely. Smaller proportions of smokers aged ≥65 years (53.7%) and 18–24 years (62.3%) were interested in quitting than were smokers aged 25–44 years (72.7%) (Table 1). The prevalence of past-year quit attempts increased during 2000–2015 ($p < 0.05$ based on quadratic trend analysis), and was 55.4% in 2015, which was the time point when prevalence was highest (Figure). Past-year quit attempts decreased with increasing age. Higher prevalences of past-year quit attempts were reported by Asians (69.4%) and blacks (63.4%) than by whites (53.3%) (Table 1).

The prevalence of recent cessation increased during 2000–2015 ($p < 0.05$ based on linear trend analysis), and was 7.4% in 2015 (Figure). Recent cessation generally increased with increasing level of educational attainment, and smokers with private health insurance (9.4%) reported a higher prevalence of recent cessation than did smokers who were uninsured (5.2%) or enrolled in Medicaid (including persons with dual Medicaid/Medicare eligibility)[§] (5.9%) (Table 1). As of 2015, among adults who had ever smoked, 59.1% (52.8 million) had quit.

During 2000–2015, increases were reported in receipt of advice from a health professional to quit: prevalence was 57.2% in 2015 ($p < 0.05$ based on quadratic trend analysis); prevalence was highest in 2005 and 2015, with a decrease observed in 2010

[§]A secondary analysis found that the prevalence of reported cessation behaviors for Medicaid enrollees did not change substantially when persons with dual Medicaid/Medicare eligibility were removed from the Medicaid coverage category.

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TABLE 1. Prevalence of interest in quitting smoking,* past-year quit attempt,[†] and recent smoking cessation[§] among adult smokers aged ≥18 years, by selected characteristics — National Health Interview Survey, United States, 2015

Characteristic	Interested in quitting % (95% CI)	Past-year quit attempt % (95% CI)	Recent smoking cessation [¶] % (95% CI)
Overall	68.0 (65.9–70.0)	55.4 (53.5–57.3)	7.4 (6.5–8.3)
Sex			
Men	66.7 (63.8–69.6)	55.3 (52.7–57.9)	7.2 (6.0–8.5)
Women	69.4 (66.7–72.1)	55.6 (53.0–58.1)	7.6 (6.2–8.9)
Age group (yrs)			
18–24	62.3 (55.7–69.0)	66.7 (61.0–72.4)	9.9 (6.1–13.8)
25–44	72.7 (69.7–75.7)	59.8 (57.3–62.3)	8.9 (7.3–10.5)
45–64	68.7 (65.8–71.6)	49.6 (46.8–52.5)	5.7 (4.6–6.7)
≥65	53.7 (48.4–58.9)	47.2 (42.2–52.3)	5.4 (3.4–7.5)
Race/Ethnicity**			
White, non-Hispanic	67.5 (65.0–70.0)	53.3 (50.8–55.7)	7.1 (6.0–8.2)
Black, non-Hispanic	72.8 (68.2–77.4)	63.4 (59.0–67.9)	4.9 (3.2–6.6)
Hispanic	67.4 (61.9–72.8)	56.2 (51.6–60.9)	8.2 (5.5–10.9)
AI/AN, non-Hispanic	55.6 (35.8–75.4)	52.1 (32.1–72.2)	— ^{††}
Asian, non-Hispanic ^{§§}	69.6 (59.5–79.8)	69.4 (62.1–76.7)	17.3 (10.1–24.5)
Multiple race, non-Hispanic	59.8 (45.7–73.9)	57.8 (47.2–68.4)	— ^{††}
Education^{¶¶}			
≤12 yrs (no high school diploma)	68.0 (63.7–72.2)	50.4 (46.2–54.5)	4.4 (2.7–6.1)
GED certificate	65.7 (58.0–73.4)	48.1 (40.1–56.0)	— ^{††}
High school diploma	65.5 (61.9–69.1)	52.2 (48.3–56.2)	6.8 (4.9–8.7)
Some college (no degree)	70.2 (66.1–74.4)	57.8 (53.6–61.9)	7.2 (5.4–9.1)
Associate degree	70.6 (65.3–76.0)	57.4 (52.2–62.7)	9.2 (6.3–12.0)
Undergraduate degree	73.3 (67.7–78.8)	57.6 (51.5–63.8)	11.2 (7.4–15.0)
Graduate degree	74.0 (65.1–82.9)	55.8 (46.0–65.6)	10.8 (4.9–16.7)
Poverty status^{***}			
At or above poverty level	68.2 (65.9–70.4)	55.5 (53.3–57.7)	7.9 (6.8–8.9)
Below poverty level	67.3 (63.4–71.1)	55.2 (51.6–58.8)	5.6 (3.8–7.3)
U.S. Census regions^{†††}			
Northeast	74.5 (69.0–80.1)	58.8 (54.6–63.0)	8.6 (5.9–11.3)
Midwest	67.1 (63.1–71.1)	54.0 (49.7–58.4)	6.4 (4.8–8.0)
South	67.2 (64.0–70.4)	54.3 (51.6–57.0)	7.6 (6.1–9.0)
West	65.5 (60.7–70.2)	56.9 (52.5–61.3)	7.6 (5.7–9.6)
Health insurance coverage^{§§§}			
Private	69.0 (66.1–71.8)	57.2 (54.6–59.9)	9.4 (7.9–10.9)
Medicaid and dual eligibles ^{¶¶¶}	69.2 (65.3–73.2)	56.3 (52.5–60.1)	5.9 (4.1–7.7)
Medicare-Advantage	40.6 (29.9–51.3)	42.6 (32.2–53.0)	— ^{††}
Medicare-only (excluding Advantage)	53.0 (42.5–63.6)	42.0 (32.2–51.8)	— ^{††}
Other coverage	63.6 (57.2–69.9)	50.7 (43.9–57.4)	5.5 (2.4–8.7)
Uninsured	69.5 (65.2–73.9)	53.5 (49.7–57.2)	5.2 (3.3–7.0)
Disability/Limitation^{****}			
Yes	66.4 (61.4–71.3)	55.1 (49.6–60.6)	5.8 (3.8–7.7)
No	66.8 (63.5–70.2)	56.3 (53.6–59.0)	7.9 (6.2–9.5)

See table footnotes on page 1460.

(Figure). Smokers aged 45–64 years (65.7%) and ≥65 years (65.7%) reported a higher prevalence of receiving advice to quit than did smokers aged 18–24 years (44.4%) and 25–44 years (49.8%) (Table 2). Lower prevalences of receiving advice to quit were reported by Asian (34.2%), American Indian/Alaska Native (38.1%), and Hispanic (42.2%) smokers than by white smokers (60.2%); and by uninsured smokers (44.1%) than by smokers with any type of insurance (range = 56.8%–69.2%). Smokers reporting a disability/limitation or serious psychological distress reported a higher prevalence of receiving advice to quit than did smokers without these conditions (71.8% and 70.2%, respectively, vs 53.6% and 55.7%).

Use of cessation counseling and/or medication among smokers who were trying to quit increased during 2000–2005 from 21.9% to 29.1%, with no change in 2010 (31.7%) or 2015 (31.2%) ($p < 0.05$ based on quadratic trend analysis) (Figure). The prevalence of use of counseling and/or medication increased with age through age 64 years (Table 2). Hispanics and Asians reported a lower prevalence of using counseling and/or medication (19.2% and 20.5%, respectively) than did whites (34.3%), as did uninsured smokers (21.4%) compared with smokers with any type of insurance other than Medicare and Medicare Advantage (range = 32.1%–36.0%). The prevalence of using counseling and/or medication was higher

TABLE 1. (Continued) Prevalence of interest in quitting smoking,* past-year quit attempt,[†] and recent smoking cessation[§] among adult smokers aged ≥18 years, by selected characteristics — National Health Interview Survey, United States, 2015

Characteristic	Interested in quitting % (95% CI)	Past-year quit attempt % (95% CI)	Recent smoking cessation [¶] % (95% CI)
Serious Psychological Distress (Kessler Scale)^{†††}			
Yes (Kessler score ≥13)	67.4 (61.3–73.5)	53.0 (46.9–59.1)	— ^{††}
No (Kessler score <13)	68.2 (66.0–70.3)	55.5 (53.5–57.5)	8.1 (7.1–9.1)
Sexual orientation^{¶¶¶¶}			
Straight	68.1 (65.9–70.2)	55.4 (53.5–57.3)	7.6 (6.7–8.6)
Gay/Lesbian/Bisexual	66.7 (56.9–76.6)	48.4 (39.4–57.3)	— ^{††}

Abbreviations: AI/AN = American Indian/Alaska Native; CI = confidence interval; GED = General Educational Development.

* Current smokers who reported that they wanted to stop smoking completely.

[†] Current smokers who reported that they stopped smoking for >1 day during the past 12 months because they were trying to quit smoking and former smokers who quit during the past year.

[§] Former smokers who quit smoking for ≥6 months during the past year.

[¶] Among current smokers who smoked for ≥2 years and former smokers who quit during the past year.

** Excludes 63 respondents of non-Hispanic unknown race. Hispanics can be of any race.

^{††} Data not reported because sample size is <50 or the relative standard error of the estimate is >30%.

^{§§} Does not include Native Hawaiians or Other Pacific Islanders.

^{¶¶} Among persons aged ≥25 years. Excludes 144 persons whose education level was unknown.

^{***} Family income was reported by the family respondent, who might or might not be the same as the sample adult respondent from whom smoking information was collected. Missing values were imputed. Because the weighted Census poverty thresholds for 2014 were not available when the 2015 National Health Interview Survey (NHIS) instrument was created, the poverty thresholds used in the 2015 NHIS were estimated from several sources: weighted average Census poverty thresholds from 2013; the average Consumer Price Index from 2013; actual Consumer Price Index values for January–July 2014; and projected Consumer Price Index values for August–December 2014.

^{†††} *Northeast:* Connecticut, Maine, Massachusetts, New Hampshire, New Jersey, New York, Pennsylvania, Rhode Island, and Vermont. *Midwest:* Illinois, Indiana, Iowa, Kansas, Michigan, Minnesota, Missouri, Nebraska, North Dakota, Ohio, South Dakota, and Wisconsin. *South:* Alabama, Arkansas, Delaware, District of Columbia, Florida, Georgia, Kentucky, Louisiana, Maryland, Mississippi, North Carolina, Oklahoma, South Carolina, Tennessee, Texas, Virginia, and West Virginia. *West:* Alaska, Arizona, California, Colorado, Hawaii, Idaho, Montana, Nevada, New Mexico, Oregon, Utah, Washington, and Wyoming.

^{§§§} Health insurance coverage was from NHIS-recoded data using a hierarchical assignment. Excludes 155 persons whose coverage was unknown.

^{¶¶¶} A secondary analysis found that the prevalence of reported cessation behaviors for Medicaid enrollees did not change substantially when persons with dual Medicaid/Medicare eligibility were removed from the Medicaid coverage category.

^{****} Based on proxy or self-reported presence of selected impairments, including vision, hearing, cognition, and movement and limitations in performing activities of daily living and instrumental activities of daily living. Limitations in performing activities of daily living was defined based on response to the question "Does [person] have difficulty dressing or bathing?" and limitations in performing instrumental activities of daily living was defined based on response to the question, "Because of a physical, mental, or emotional condition, does [person] have difficulty doing errands alone, such as visiting a doctor's office or shopping?" Any disability/limitation was defined as a "yes" response pertaining to at least one of the disabilities/limitations listed (i.e., vision, hearing, cognition, movement, activities of daily living, or instrumental activities of daily living). In 2015, the American Community Survey questions were asked of a random half of the respondents from the 2015 Person File. Excludes four persons whose disability status was unknown.

^{††††} The Kessler Psychological Distress Scale is a series of six questions that asks about feelings of sadness, nervousness, restlessness, worthlessness, hopelessness, and feeling like everything is an effort during the past 30 days. Participants were asked to respond on a Likert Scale ranging between 'None of the Time' (score = 0) and 'All of the time' (score = 4). Responses were summed over the six questions; respondents with a score ≥13 were coded as having serious psychological distress, and respondents with a score <13 were coded as not having serious psychological distress. Excludes 1,416 persons whose psychological distress was unknown. Additional information available at <https://www.cdc.gov/nchs/data/databriefs/db203.pdf>.

among smokers reporting a disability/limitation (39.0%) or serious psychological distress (41.6%) than among smokers without these conditions (28.5% and 30.1%, respectively). Gay, lesbian, or bisexual smokers reported a lower prevalence of counseling and/or medication use (14.5%) than did straight smokers (31.7%).

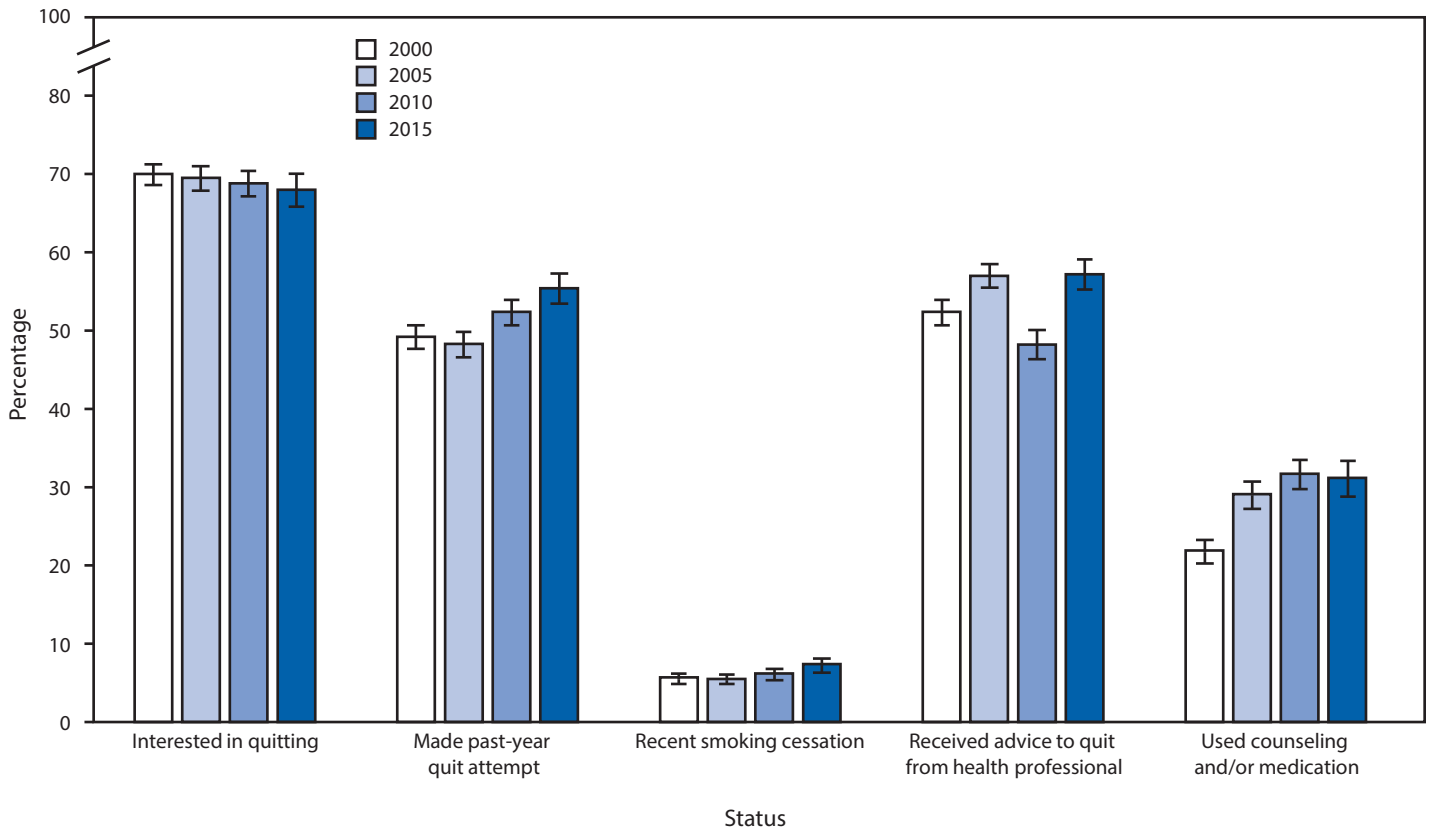
Among smokers who made quit attempts, 6.8% reported using counseling, 29.0% medication, and 4.7% both. Among smokers who used counseling, 4.1% used a telephone quitline, 2.8% used one-on-one counseling, and 2.4% used a stop smoking clinic, class, or support group. Among smokers who used medications, 16.6% used a nicotine patch, 12.5% used nicotine gum or lozenges, 7.9% used varenicline, 2.7% used bupropion, and 2.4% used nicotine spray or inhaler.

Discussion

In 2015, approximately two thirds of cigarette smokers were interested in quitting, and slightly more than half reported receiving advice to quit from a health professional and making a past-year quit attempt. However, fewer than one third of smokers who tried to quit used proven cessation treatments, and fewer than one in 10 smokers overall quit successfully in the past year. Approximately three in five adults who had ever smoked had quit. To enhance cessation rates, it is critical for health care providers to consistently identify smokers, advise them to quit, and offer evidence-based cessation treatments, and for insurers to cover and promote the use of these treatments and remove barriers to accessing them (2–6).

During 2000–2015, modest but statistically significant increases occurred in the prevalence of past-year quit attempts

FIGURE. Prevalence of and change* in interest in quitting,[†] past-year quit attempt,[‡] recent smoking cessation,[§] receiving a health professional's advice to quit smoking,^{**} and use of counseling and/or medication for cessation^{††} among adult smokers aged ≥18 years — National Health Interview Survey, United States 2000–2015



* Based on linear and quadratic trend analyses using logistic regression models controlling for sex, race/ethnicity, and age, $p < 0.05$. There was no change for "interested in quitting," a quadratic trend for "made past-year quit attempt," a linear trend for "recent smoking cessation," a quadratic trend for "received advice to quit from health professional," and a quadratic trend for "used counseling and/or medication."

[†] Current smokers who reported that they wanted to stop smoking completely.

[‡] Current smokers who reported that they stopped smoking for >1 day in the past 12 months because they were trying to quit smoking and former smokers who quit in the past year.

[§] Former smokers who quit smoking for ≥6 months in the past year, among current smokers who smoked for ≥2 years and former smokers who quit in the past year.

^{**} Received advice from a medical doctor, dentist, or other health professional to quit smoking or to quit using other kinds of tobacco, among current and former cigarette smokers who quit in the past 12 months. The analysis was limited to current and former cigarette smokers who had seen a doctor or other health professional in the past year.

^{††} For 2010 and 2015, used one-on-one counseling, a stop smoking clinic, class, or support group, and/or a telephone help line or quitline; and/or the nicotine patch, nicotine gum or lozenge, nicotine-containing nasal spray or inhaler, varenicline (U.S. trade name Chantix) and/or bupropion (including trade names Zyban and Wellbutrin) in the past year among current smokers who tried to quit in the past year or used when stopped smoking among former smokers who quit in the past 2 years. For 2005, the list included a nicotine tablet and excluded varenicline, as it was not approved by the Food and Drug Administration until 2006. For 2000, the list included a stop smoking program and excluded a stop smoking class or support group, nicotine lozenge (not approved by the Food and Drug Administration until 2002), and varenicline.

(from 49.2% to 55.4%), recent smoking cessation (5.7% to 7.4%), receipt of health professional advice to quit smoking (52.4% to 57.2%), and use of cessation counseling and/or medication (21.9% to 31.2%). However, recent smoking cessation remains low, and little progress has been made since 2005 toward increasing receipt of advice to quit and use of counseling and/or medication. Use of cessation counseling and medication increases quit rates, especially when they are combined (2,3,7): combined behavioral and pharmacotherapy interventions increase cessation by 82%, compared with minimal intervention or usual care (7). Use of cessation

medications is appropriate for most adult smokers, with the exception of pregnant women, light smokers (i.e., persons who smoke < 5-10 cigarettes daily), and persons with specific medical contraindications (2,3). The low prevalence of recent cessation likely is related in part to low use of evidence-based cessation treatments. Because approximately 70% of smokers see a physician annually, and even brief physician advice to quit increases quit rates (2), opportunities exist to increase cessation rates through health care system changes and other population-based strategies (2-4).

TABLE 2. Prevalence of receiving a health professional's advice to quit smoking,* and use of counseling† and medication‡ for cessation among adult smokers aged ≥18 years, by selected characteristics — National Health Interview Survey, United States, 2015

Characteristic	Received health professional's advice to quit % (95% CI)	Used counseling % (95% CI)	Used medication % (95% CI)	Used counseling and/or medication % (95% CI)
Overall	57.2 (55.3–59.1)	6.8 (5.7–7.9)	29.0 (26.8–31.2)	31.2 (28.9–33.5)
Sex				
Men	55.2 (52.5–57.9)	5.8 (4.3–7.4)	27.0 (24.0–30.0)	29.1 (26.0–32.2)
Women	59.3 (56.6–61.9)	7.9 (6.4–9.5)	31.3 (28.2–34.3)	33.6 (30.5–36.6)
Age group (yrs)				
18–24	44.4 (37.1–51.6)	—¶	15.6 (9.5–21.7)	16.8 (10.6–23.0)
25–44	49.8 (46.6–53.0)	6.1 (4.5–7.8)	25.5 (22.2–28.7)	27.4 (24.1–30.8)
45–64	65.7 (62.9–68.4)	8.8 (6.9–11.1)	37.7 (34.0–41.4)	40.2 (36.4–43.9)
≥65	65.7 (61.4–70.0)	9.2 (5.3–13.1)	33.7 (27.7–39.7)	37.0 (31.0–43.1)
Race/Ethnicity**				
White, non-Hispanic	60.2 (58.0–62.4)	6.9 (5.5–8.3)	32.6 (29.8–35.4)	34.3 (31.4–37.2)
Black, non-Hispanic	55.7 (50.2–61.1)	7.6 (4.5–10.8)	25.2 (20.1–30.3)	28.9 (23.5–34.4)
Hispanic	42.2 (37.0–47.5)	5.1 (2.4–7.7)	16.6 (12.4–20.9)	19.2 (14.4–24.0)
AI/AN, non-Hispanic	38.1 (21.4–54.8)	—¶	—¶	—¶
Asian, non-Hispanic††	34.2 (24.2–44.3)	—¶	17.4 (9.4–25.4)	20.5 (12.2–28.8)
Multiple race, non-Hispanic	69.6 (59.2–80.1)	—¶	22.1 (10.5–33.6)	24.6 (12.7–36.4)
Education§§				
≤12 yrs (no high school diploma)	60.8 (56.6–65.1)	5.4 (3.1–7.6)	26.5 (21.8–31.2)	28.7 (23.8–33.6)
GED certificate	61.6 (52.4–70.7)	—¶	30.8 (21.5–40.1)	31.4 (22.0–40.7)
High school diploma	58.1 (53.9–62.3)	7.0 (4.7–9.4)	30.3 (25.5–35.1)	33.1 (28.1–38.1)
Some college (no degree)	59.1 (55.3–63.0)	8.6 (6.0–11.1)	32.5 (28.1–36.9)	34.6 (30.1–39.2)
Associate degree	61.6 (56.4–66.8)	8.6 (5.1–12.2)	33.2 (27.4–39.0)	36.0 (29.8–42.3)
Undergraduate degree	52.6 (46.6–58.5)	7.4 (3.7–11.1)	33.2 (26.5–39.8)	35.1 (28.4–41.7)
Graduate degree	57.7 (48.5–66.8)	—¶	32.8 (22.9–42.6)	35.9 (25.7–46.0)
Poverty status¶¶				
At or above poverty level	57.8 (55.6–60.1)	6.8 (5.6–8.1)	29.5 (27.1–31.8)	31.7 (29.2–34.2)
Below poverty level	54.7 (50.7–58.7)	6.7 (4.6–8.9)	27.0 (21.6–31.6)	29.0 (24.2–33.7)
U.S. Census regions***				
Northeast	65.1 (60.2–70.1)	8.2 (4.9–11.5)	34.7 (27.9–41.5)	37.6 (30.9–44.2)
Midwest	60.0 (56.1–63.9)	4.9 (3.0–6.8)	28.9 (24.9–32.8)	30.2 (26.1–34.4)
South	55.2 (52.2–58.2)	7.2 (5.3–9.0)	27.2 (23.8–30.6)	29.3 (25.7–33.0)
West	50.6 (46.9–54.4)	7.5 (5.1–9.9)	28.0 (23.1–32.8)	30.7 (25.5–35.9)
Health insurance coverage†††				
Private	56.8 (54.0–59.5)	6.8 (5.3–8.3)	29.9 (27.0–32.7)	32.1 (29.1–35.1)
Medicaid and dual eligibles§§§	59.9 (55.7–64.1)	8.0 (5.3–10.7)	32.2 (27.3–37.2)	34.5 (29.3–39.6)
Medicare-Advantage	66.6 (56.5–76.6)	—¶	26.5 (15.5–37.4)	31.6 (19.7–43.4)
Medicare-only (excluding Advantage)	62.0 (51.7–72.3)	—¶	28.5 (15.5–41.5)	35.9 (22.6–49.1)
Other coverage	69.2 (62.8–75.7)	5.2 (2.7–7.7)	34.9 (26.2–43.6)	36.0 (27.3–44.7)
Uninsured	44.1 (38.8–49.3)	4.3 (2.2–6.4)	20.0 (15.6–24.6)	21.4 (17.0–25.8)
Disability/Limitation¶¶¶				
Yes	71.8 (67.4–76.2)	12.6 (8.3–16.9)	35.7 (29.1–42.3)	39.0 (32.1–45.9)
No	53.6 (50.5–56.8)	5.1 (3.8–6.4)	26.3 (22.9–29.6)	28.5 (25.1–31.9)

See table footnotes on page 1463.

Observed disparities were consistent with those reported in previous studies (8). In 2015, smokers who were aged <45 years, Hispanic, Asian, with an Associate's or higher degree, lived in the Northeast, had private health insurance, or had no serious psychological distress met the *Healthy People 2020* target for recent cessation (≥8.0%). Disparities in cessation behaviors by race/ethnicity might be partly explained by differences in tobacco use behaviors, health care utilization, access to cessation treatments, and knowledge about these treatments (1,2,4). Disparities by insurance status in receipt of advice to

quit (44.1% for uninsured smokers versus 56.8% for smokers with private insurance), use of cessation counseling and/or medication (21.4% for uninsured smokers versus 32.1% for smokers with private insurance), and recent cessation (5.2% for uninsured smokers versus 9.4% for smokers with private insurance) are likely attributable, in part, to a lack of access to cessation treatments among the uninsured (2,4,5). Higher prevalence of receiving a health professional's advice to quit and use of counseling and/or medication among smokers with serious psychological distress might be related to greater use

TABLE 2. (Continued) Prevalence of receiving a health professional's advice to quit smoking,* and use of counseling† and medication‡ for cessation among adult smokers aged ≥18 years, by selected characteristics — National Health Interview Survey, United States, 2015

Characteristic	Received health professional's advice to quit % (95% CI)	Used counseling % (95% CI)	Used medication % (95% CI)	Used counseling and/or medication % (95% CI)
Serious Psychological Distress (Kessler Scale)****				
Yes (Kessler score ≥13)	70.2 (64.5–75.8)	12.4 (6.3–18.4)	40.1 (32.5–47.8)	41.6 (33.7–49.5)
No (Kessler score <13)	55.7 (53.7–57.7)	6.3 (5.3–7.4)	27.9 (25.6–30.1)	30.1 (27.8–32.5)
Sexual orientation††††				
Straight	57.1 (55.1–59.1)	6.9 (5.7–8.0)	29.4 (27.2–31.7)	31.7 (29.3–34.1)
Gay/Lesbian/Bisexual	57.7 (48.5–66.9)	—¶	14.4 (7.8–21.0)	14.5 (7.9–21.1)

Abbreviations: AI/AN = American Indian/Alaska Native; CI = confidence interval; GED = General Educational Development.

* Received advice from a medical doctor, dentist, or other health professional to quit smoking or to quit using other kinds of tobacco, among current and former cigarette smokers who quit in the past 12 months. The analysis was limited to current and former cigarette smokers who had seen a doctor or other health professional in the past year.

† Used one-on-one counseling, a stop smoking clinic, class, or support group, and/or a telephone help line or quitline during the past year among current smokers who tried to quit during the past year or used when stopped smoking among former smokers who quit during the past 2 years.

‡ Used nicotine patch, nicotine gum or lozenge, nicotine-containing nasal spray or inhaler, varenicline (U.S. trade name Chantix), and/or bupropion (including trade names Zyban and Wellbutrin) during the past year among current smokers who tried to quit during the past year or used when stopped smoking among former smokers who quit during the past 2 years.

¶ Data not reported because sample size is <50 or the relative standard error of the estimate is >30%.

** Excludes 63 respondents of non-Hispanic unknown race. Hispanics can be of any race.

†† Does not include Native Hawaiians or Other Pacific Islanders.

§§ Among persons aged ≥25 years. Excludes 144 persons whose education level was unknown.

¶¶ Family income was reported by the family respondent, who might or might not be the same as the sample adult respondent from whom smoking information was collected. Missing values were imputed. Because the weighted Census poverty thresholds for 2014 were not available when the 2015 National Health Interview Survey (NHIS) instrument was created, the poverty thresholds used in the 2015 NHIS were estimated from several sources: weighted average Census poverty thresholds from 2013; the average Consumer Price Index from 2013; actual Consumer Price Index values for January–July 2014; and projected Consumer Price Index values for August–December 2014.

*** *Northeast:* Connecticut, Maine, Massachusetts, New Hampshire, New Jersey, New York, Pennsylvania, Rhode Island, and Vermont. *Midwest:* Illinois, Indiana, Iowa, Kansas, Michigan, Minnesota, Missouri, Nebraska, North Dakota, Ohio, South Dakota, and Wisconsin. *South:* Alabama, Arkansas, Delaware, District of Columbia, Florida, Georgia, Kentucky, Louisiana, Maryland, Mississippi, North Carolina, Oklahoma, South Carolina, Tennessee, Texas, Virginia, and West Virginia. *West:* Alaska, Arizona, California, Colorado, Hawaii, Idaho, Montana, Nevada, New Mexico, Oregon, Utah, Washington, and Wyoming.

††† Health insurance coverage was from NHIS-recoded data using a hierarchal assignment. Excludes 155 persons whose coverage was unknown.

§§§ A secondary analysis found that the prevalence of reported cessation behaviors for Medicaid enrollees did not change substantially when persons with dual Medicaid/Medicare eligibility were removed from the Medicaid coverage category.

¶¶¶ Based on proxy or self-reported presence of selected impairments, including vision, hearing, cognition, and movement and limitations in performing activities of daily living and instrumental activities of daily living. Limitations in performing activities of daily living was defined based on response to the question "Does [person] have difficulty dressing or bathing?" and limitations in performing instrumental activities of daily living was defined based on response to the question, "Because of a physical, mental, or emotional condition, does [person] have difficulty doing errands alone, such as visiting a doctor's office or shopping?" Any disability/limitation was defined as a "yes" response pertaining to at least one of the disabilities/limitations listed (i.e., vision, hearing, cognition, movement, activities of daily living, or instrumental activities of daily living). In 2015, the American Community Survey questions were asked of a random half of the respondents from the 2015 Person File. Excludes four persons whose disability status was unknown.

**** The Kessler Psychological Distress Scale is a series of six questions that asks about feelings of sadness, nervousness, restlessness, worthlessness, hopelessness, and feeling like everything is an effort during the past 30 days. Participants were asked to respond on a Likert Scale ranging between 'None of the Time' (score = 0) and 'All of the time' (score = 4). Responses were summed over the six questions; respondents with a score ≥13 were coded as having serious psychological distress, and respondents with a score <13 were coded as not having serious psychological distress. Excludes 1,416 persons whose psychological distress was unknown. Additional information available at <https://www.cdc.gov/nchs/data/databriefs/db203.pdf>.

†††† Response options were "straight, that is, not gay" for men, and "straight, that is, not gay or lesbian" for women. Excludes 1,397 persons whose sexual orientation was unknown.

of health care as well as greater tobacco dependence in this population (1,4).

Changes in the U.S. health care system could have contributed to this report's findings. By increasing the number of adults with health insurance (9) and requiring improved cessation coverage by commercial insurance and Medicaid (5), the Patient Protection and Affordable Care Act[¶] might have contributed to increases in the number of smokers who attempt to quit, use proven cessation treatments, and successfully quit (4,5). Improved cessation insurance coverage,

¶ <http://housedocs.house.gov/energycommerce/ppacacon.pdf>.

together with new health care delivery and payment models and quality measures, might have contributed to increases in health professional advice to quit since 2010 (4,5).

The findings in this report are subject to at least three limitations. First, cigarette smoking and cessation-related measures were self-reported without validation by biochemical testing, and might be subject to social desirability bias. However, self-reported smoking status correlates with serum cotinine levels (10). Second, because NHIS does not include institutionalized populations and persons in the military, results are not generalizable to these groups. Finally, lower NHIS response rates might

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

Quitting cigarette smoking benefits smokers at any age. Cessation counseling and medications each improve smokers' chances of quitting, and have an even greater effect when combined. However, use of counseling and medications remains low.

What is added by this report?

Approximately two thirds of cigarette smokers are interested in quitting, and in 2015, approximately half of smokers reported receiving advice to quit from a health professional and making a quit attempt in the past year. However, fewer than one third of smokers who tried to quit used evidence-based cessation treatments, and fewer than one in 10 smokers overall successfully quit in the past year. As of 2015, approximately three in five adults who had ever smoked had quit.

What are the implications for public health practice?

Health care professionals can help smokers quit by consistently identifying patients who smoke, advising them to quit, and offering them cessation treatments. Health insurers can help smokers quit by covering proven cessation treatments with minimal barriers and promoting their use. States can help smokers quit by implementing population-based policy interventions and anti-tobacco mass media campaigns, and by funding comprehensive state tobacco control programs, including state quitlines, at CDC-recommended levels.

result in nonresponse bias. The extent to which nonresponse might have affected the results reported here is unknown.

Funding state tobacco control programs, including state quitlines, at CDC-recommended levels, increasing tobacco prices, implementing comprehensive smoke-free policies, conducting anti-tobacco mass media campaigns, and enhancing access to quitting assistance can increase tobacco cessation and reduce tobacco-related disease and death (1,4). Opportunities exist for insurers and employers to improve coverage and increase use of cessation treatments and for health systems to integrate cessation interventions into clinical care (1,4,5).

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¹Office on Smoking and Health, National Center for Chronic Disease Prevention and Health Promotion, CDC.

Corresponding author: Stephen Babb, Sbabb@cdc.gov, 770-488-1172.

References

1. US Department of Health and Human Services. The health consequences of smoking—50 years of progress: a report of the Surgeon General. Atlanta, GA: US Department of Health and Human Services, CDC; 2014. <http://www.surgeongeneral.gov/library/reports/50-years-of-progress/full-report.pdf>
2. US Public Health Service. Treating tobacco use and dependence: 2008 update. Clinical practice guideline. Rockville, MD: US Department of Health and Human Services, US Public Health Service; 2008. <http://www.ahrq.gov/professionals/clinicians-providers/guidelines-recommendations/tobacco/index.html>
3. Siu AL; US Preventive Services Task Force. Behavioral and pharmacotherapy interventions for tobacco smoking cessation in adults, including pregnant women: U.S. Preventive Services Task Force recommendation statement. *Ann Intern Med* 2015;163:622–34. <http://dx.doi.org/10.7326/M15-2023>
4. CDC. Best practices for comprehensive tobacco control programs—2014. Atlanta, GA: US Department of Health and Human Services, CDC; 2014. http://www.cdc.gov/tobacco/stateandcommunity/best_practices/index.htm
5. McAfee T, Babb S, McNabb S, Fiore MC. Helping smokers quit—opportunities created by the Affordable Care Act. *N Engl J Med* 2015;372:5–7. <http://dx.doi.org/10.1056/NEJMp1411437>
6. Jamal A, King BA, Neff LJ, Whitmill J, Babb SD, Graffunder CM. Current cigarette smoking among adults—United States, 2005–2015. *MMWR Morb Mortal Wkly Rep* 2016;65:1205–11. <http://dx.doi.org/10.15585/mmwr.mm6544a2>
7. Patnode CD, Henderson JT, Thompson JH, Senger CA, Fortmann SP, Whitlock EP. Behavioral counseling and pharmacotherapy interventions for tobacco cessation in adults, including pregnant women: a review of reviews for the U.S. Preventive Services Task Force. *Ann Intern Med* 2015;163:608–21. <http://dx.doi.org/10.7326/M15-0171>
8. Malarcher A, Dube S, Shaw L, Babb S, Kaufmann R. Quitting smoking among adults—United States, 2001–2010. *MMWR Morb Mortal Wkly Rep* 2011;60:1513–9.
9. Cohen RA, Martinez ME, Zammitti EP. Early release of selected estimates based on data from the National Health Interview Survey, 2015. Hyattsville, MD: US Department of Health and Human Services, CDC, National Center for Health Statistics; 2016. <http://www.cdc.gov/nchs/nhis/releases/released201605.htm>
10. Binnie V, McHugh S, Macpherson L, Borland B, Moir K, Malik K. The validation of self-reported smoking status by analysing cotinine levels in stimulated and unstimulated saliva, serum and urine. *Oral Dis* 2004;10:287–93. <http://dx.doi.org/10.1111/j.1601-0825.2004.01018.x>

Traumatic Brain and Spinal Cord Fatalities Among High School and College Football Players — United States, 2005–2014

Kristen L. Kucera, PhD¹; Rebecca K. Yau, PhD^{1,2}; Johna Register-Mihalik, PhD³; Stephen W. Marshall, PhD²; Leah C. Thomas, MS¹; Susanne Wolf¹; Robert C. Cantu, MD⁴; Frederick O. Mueller, PhD¹; Kevin M. Guskiewicz, PhD³

An estimated 1.1 million high school and 75,000 college athletes participate in tackle football annually in the United States. Football is a collision sport; traumatic injuries are frequent (1,2), and can be fatal (3). This report updates the incidence and characteristics of deaths caused by traumatic brain injury and spinal cord injury (4) in high school and college football and presents illustrative case descriptions. Information was analyzed from the National Center for Catastrophic Sport Injury Research (NCCSIR). During 2005–2014, a total of 28 deaths (2.8 deaths per year) from traumatic brain and spinal cord injuries occurred among high school (24 deaths) and college football players (four deaths) combined. Most deaths occurred during competitions and resulted from tackling or being tackled. All four of the college deaths and 14 (58%) of the 24 high school deaths occurred during the last 5 years (2010–2014) of the 10-year study period. These findings support the need for continued surveillance and safety efforts (particularly during competition) to ensure proper tackling techniques, emergency planning for severe injuries, availability of medical care onsite during competitions, and assessment that it is safe to return to play following a concussion.

NCCSIR has been conducting catastrophic injury surveillance at the national level for high school and collegiate football since 1965 (5) and for all sports since 1982 (6). Deaths were identified through ongoing and systematic monitoring of public media sources (e.g., online search engines and news search engines) and individual reports from national and state-level organizations, clinicians, school authorities, and researchers. Once a death was identified, NCCSIR researchers contacted family members or school staff members to gather additional details. When possible, NCCSIR obtained medical examiner reports. Information was collected about the athlete's age and level of play; player position and activity; and injury type, medical care, and cause of death.

The events included in this study were defined as fatal traumatic brain and spinal cord injuries that occurred during a scheduled team activity (game, practice, or conditioning session) and were directly related to football-specific activities (e.g., tackling or being tackled). Each fatality report was manually reviewed for inclusion and classification. Fatality rates per 1 million players were calculated using National Federation of State High School Associations and National Collegiate Athletic Association participation statistics as the

denominators. The causes and potential strategies to prevent these injuries were described in association with the 10 Haddon energy damage countermeasures (7). All procedures were reviewed and approved by the Institutional Review Board of the University of North Carolina at Chapel Hill.

During 2005–2014, a total of 24 high school and four college football–related traumatic brain and spinal cord injury fatalities were identified, for a combined average of 2.8 fatalities per year. Among the 24 high school fatalities (Table 1), 22 (92%) involved head/brain injuries. All four college fatalities involved a brain injury. Subdural hematoma was the most common diagnosis for both high school and college fatalities (46% overall). Four (18%) of the 22 high school players who died from brain injuries had sustained a concussion within 4 weeks of the event, and second impact syndrome (in which a second concussion occurs before a first concussion has properly healed, causing rapid and severe brain swelling) was implicated in three of these four events.

Among the 24 high school fatalities (Table 1), 20 (83%) occurred during a game and during the regular season; 17 (71%) involved tackling or being tackled. Among the four college fatalities, two occurred during a regular season game, and two occurred during spring football. The most common player positions among those fatally injured were running back (32% of players overall) and linebacker (21%). Of the 28 deaths, head first/head down contact was identified in eight deaths. Six illustrative cases provide associations with the Haddon energy damage countermeasures and extensions to football recommendations for preventing traumatic brain and spinal cord fatalities (Table 2).

The average number of high school deaths per year was 2.4 (standard deviation [SD] = 2.2) and ranged from zero to seven deaths annually. The average number of deaths among college players per year was 0.4 (SD = 0.7) and ranged from zero to two deaths annually. For 2 years (2007 and 2012) of the 10-year study period, no traumatic brain or spinal cord injury deaths were reported among either high school or college football players. Fatality rates over the study period were 5.96 fatalities per 1 million college football players (95% confidence interval [CI] = 0.12–11.81) and 2.18 fatalities per 1 million high school football players (CI = 1.31–3.06) (Figure). All four of the college deaths and 14 (58%) of the 24 high school deaths occurred during the last 5 years (2010–2014) of the 10-year study period.

Discussion

The finding of an annual average of 2.8 brain and spinal cord injury deaths for high school and college football combined is consistent with a previous report of 3.1 brain injury fatalities annually during 1990–2010 (4). Also consistent with previous studies (3,4), most brain and spinal cord injury deaths occurred during competition, among players at running back and linebacker positions, and as a result of tackling or being tackled.

Head first/head down contact was identified as contributing to eight of the 28 deaths. This emphasizes the importance of instruction in proper tackling techniques (both delivery and receipt of tackles) for all players, but particularly for running backs, linebackers, and defensive backs. A previous evaluation of football tackling programs among youth league football players indicated a reduction in concussions in practice and games when education of coaches was combined with practice contact restrictions (8), providing evidence that these programs might have a positive impact on reducing nonfatal head injuries among youth league players. However, it is unclear whether older players who learned high risk methods can be retrained in new techniques. Football is a collision sport played at high velocity, and players must act and react quickly. In such situations, new techniques might be difficult to deploy, resulting in players possibly reverting to past behaviors and reactions unless coaches routinely intervene to correct their technique.

The finding that 18% of high school players with fatal traumatic brain injuries had a concussion <4 weeks earlier is consistent with a previous study that found 16% of football players who died from traumatic brain injuries over a 20-year period had a previous concussion within 30 days of death (4). This finding supports the importance of recognition, reporting, management, and adherence to recommended return-to-play protocols after a concussion. All 50 states and the District of Columbia currently have concussion education and safety laws in place that include appropriate medical evaluation by a trained medical professional, no same-day return to play, and return to play only after medical clearance. All laws include education for various stakeholders about concussion symptoms and management. However, for the laws to be effective, athletes must report their concussion symptoms, and medical professionals must be able to accurately assess symptom resolution and full recovery from the concussion before allowing an athlete to resume contact. The implementation and impact of these laws are an important area for future inquiry.

The cases described in this report illustrate the importance of emergency preparedness, recognition, and access to medical services. All schools should have written emergency action plans specific to their school and venue that are rehearsed annually by coaches and staff (9). The availability of medical

TABLE 1. Traumatic brain and spinal cord injury fatalities among high school and college football players, by selected characteristics — United States, 2005–2014

Characteristic	College No. (%)	High school No. (%)	Total No. (%)
Grade level			
Freshman	3 (75)	1 (4)	4 (14)
Sophomore	0 (0)	3 (13)	3 (11)
Junior	0 (0)	11 (46)	11 (39)
Senior	1 (25)	7 (29)	8 (29)
Unknown	0 (0)	2 (8)	2 (7)
Activity			
Game	2 (50)	20* (83)	22 (79)
Practice	2 (50)	4 (17)	6 (21)
Season			
Spring football	2 (50)	0 (0)	2 (7)
Preseason	1 (25)	3 (13)	4 (14)
Regular season	1 (25)	20 (83)	21 (75)
Postseason	0 (0)	1 (4)	1 (4)
Player action			
Tackling	1 (25)	9 (38)	10 (36)
Being tackled	0 (0)	7 (29)	7 (25)
Being blocked	1 (25)	4 (17)	5 (18)
Blocking	0 (0)	1 (4)	1 (4)
General play	2 (50)	2 (8)	4 (14)
Conditioning	0 (0)	1 (4)	1 (4)
Suspected cause			
Arteriovenous malformation	0 (0)	1 (4)	1 (4)
Bleed	0 (0)	1 (4)	1 (4)
Blood clot	0 (0)	1 (4)	1 (4)
Cerebral swelling	0 (0)	1 (4)	1 (4)
Fracture	0 (0)	2 (8)	2 (7)
Hemorrhage	0 (0)	2 (8)	2 (7)
Subdural hematoma	2 (50)	11 (46)	13 (46)
Traumatic brain injury	2 (50)	5 (21)	7 (25)
Body part injured			
Head/Brain	4 (100)	22 (92)	26 (93)
Spinal cord	0 (0)	2 (8)	2 (7)
Position			
Cornerback	0 (0)	2 (8)	2 (7)
Defensive back	1 (25)	2 (8)	3 (11)
Running back	2 (50)	7 (29)	9 (32)
Running back/Linebacker	0 (0)	1 (4)	1 (4)
Linebacker	0 (0)	6 (25)	6 (21)
Defensive lineman	1 (25)	1 (4)	2 (7)
Lineman	0 (0)	1 (4)	1 (4)
Offensive lineman	0 (0)	1 (4)	1 (4)
Safety	0 (0)	2 (8)	2 (7)
Kickoff coverage	0 (0)	1 (4)	1 (4)
Total	4 (100)	24 (100)	28 (100)

* One activity was a junior varsity scrimmage against a visiting team and was classified as a game.

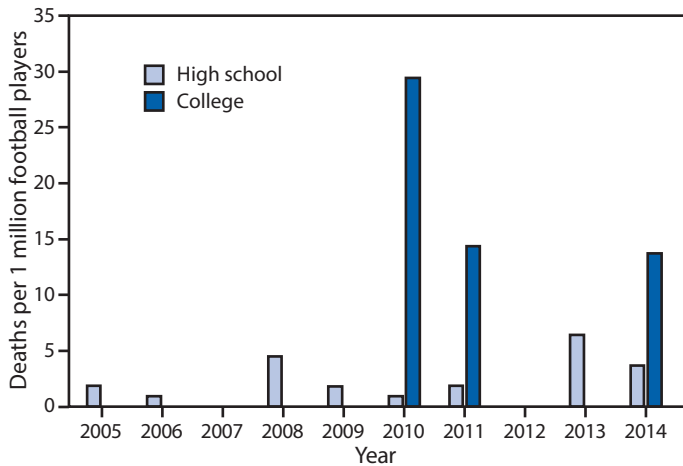
professionals onsite who are trained to recognize and act in emergency situations is critical in catastrophic football injury events. Many schools employ certified athletic trainers, and for competitions, have emergency medical services onsite. However, nationally, 30% of public high schools do not have access to an athletic trainer, and 50% do not have athletic trainers present at practices (10). One of the deaths in this report occurred during a junior varsity football scrimmage

TABLE 2. Descriptions of six illustrative cases of deaths from traumatic brain and spinal cord injury among high school and college football players and associated Haddon energy damage countermeasures* — United States, 2005–2014

Year	Player action	Position	Activity	Narrative	Associated Haddon countermeasures
2005	Blocking	Kickoff coverage	Regular season game	A high school player aged 16 years was injured at the end of the second quarter of the game while blocking on a kickoff return. The athlete lowered his helmet into the chest of an opponent and appeared unconscious when he hit the ground. He was immediately attended to by emergency medical services and transported by ambulance to a hospital. Surgery was performed to relieve pressure on the brain, but the player never recovered consciousness. He died 1 week following the injury. Cause of death was a traumatic brain injury.	1. Prevent the creation of the hazard: Ban head first contact, regardless of intention, enforce ban. 4. Modify the rate or spatial distribution of the hazard from its source: Decrease the closing distance on kickoffs. 8. Make what is to be protected more resistant to damage from the hazard: Provide universal education about proper technique for blocking, strengthen neck muscles.
2008	Being tackled	Running back	Regular season game	A high school player aged 16 years was injured during the second quarter of the game. He collapsed on the sideline after being tackled while carrying the ball. He was transported to a hospital and died the next day. He had received a concussion in practice 2 days before the game, and it is unclear whether he had clearance from a physician to return to play. Cause of death was a traumatic brain injury resulting from second impact syndrome.	5. Separate by time or space the hazard from that which can be protected: Return concussed athletes to play only when symptom free, following a graduated return to play progression, and when fully healed (prevent second impact syndrome).
2010	Tackling	Defensive back	Spring football	A college player aged 21 years was injured during a spring season game. He was injured on the last play of the game while making a tackle and taking a blow to the head. He suffered an acute subdural hematoma. He walked off the field, but began vomiting on the sideline. He was immediately attended to by the athletic trainer, and emergency medical services were summoned. He was taken to a regional hospital and flown by helicopter to a trauma center, where he later died.	1. (see above) 2. Reduce the amount of the hazard: Reduce the number and magnitude of head impacts in spring season events. 8. (see above)
2011	General play	Running back	Preseason practice	A college player aged 22 years was participating in football drills during practice when he collapsed. He was taken to a regional medical center and flown by helicopter to a trauma center, where a diagnosis was made of severe head trauma and swelling of the brain. He died several days later after multiple surgeries. Reports indicated that his forehead had been bleeding for 2 days prior as a result of a previously sustained head injury. He returned to practice despite complaints of a headache and dizziness.	2. (see above) 5. (see above) 10. Stabilize, repair and rehabilitate the damage or injured person: Provide advanced trauma care.
2014	Tackling	Lineman	Regular season game	A high school football player who was a junior sustained an injury during a game with the possibility of head-on-head contact during a kick return. He walked off the field at halftime and shortly afterward collapsed and lost consciousness. Police onsite called emergency medical services; there was no ambulance at the game. Cardiopulmonary resuscitation was administered upon the arrival of the emergency medical services unit, and he was transported by ambulance to a local hospital 18 miles away when he was unable to be stabilized for helicopter transport. He died shortly afterward at the hospital. Preliminary cause of death was blunt force trauma to the head.	1. (see above) 8. (see above) 9. Move rapidly to detect and evaluate the damage that has occurred and counter its continuation and extension: Implement an emergency action plan and provide emergency medical services onsite for games. 10. (see above)
2013	Tackling	Cornerback	Regular season game	A high school player aged 16 years collapsed after making a "routine" tackle during a junior varsity scrimmage against a visiting team. Witnesses to the event reported that his head was up when he made the tackle and that his head hit his opponent's chest. Coaches and adults responded and found he was not breathing. They telephoned emergency medical services, which took 15 minutes to arrive at the suburban school. Emergency medical services began cardiopulmonary resuscitation and transported the player to the hospital, where he died shortly afterward. Autopsy confirmed C-3 cervical fracture from blunt force head and neck trauma.	8. (see above) 9. (see above) 10. (see above)

* Haddon W Jr. Energy damage and the ten countermeasure strategies. *J Trauma* 1973;13:321–31.

FIGURE. Fatality rates from traumatic brain and spinal cord injuries among high school and college football players — United States, 2005–2014



when emergency medical services were not onsite and arrival of emergency medical services took 15 minutes because of traffic. Current best practices include access to athletic trainers for practices and competition and maintaining emergency medical services onsite during competitions (9).

The findings in this report are subject to at least four limitations. First, most events were captured through publicly available media sources, and it is possible that some football deaths were missed. Second, football participation numbers are representative of National Federation of State High School Associations and National Collegiate Athletic Association-affiliated schools and likely underestimate the actual number of football participants. Third, whenever possible, medical diagnoses and medical examiner report causes of death were used, however, the exact diagnosis was unknown for seven of the traumatic brain injury deaths. Information availability might be hampered by the sensitivity surrounding a fatal event, potential litigation, and inability to talk with persons involved. Finally, public interest and media attention about sport-related deaths and traumatic brain injuries increased during the study period, and it is unknown how this might have affected the identification of fatal injuries over time.

These findings support continued surveillance and safety efforts to ensure proper tackling techniques, emergency planning, and medical care, particularly during competition, and adherence to protocols for safe return-to-play after a concussion. These measures will also reduce the risk for concussion and improve treatment and management after a concussion is sustained. CDC provides emergency action plan templates and guidance (<https://www.cdc.gov/niosh/>

Summary

What is already known about this topic?

Fatalities resulting from catastrophic brain and spinal cord injuries occur infrequently among high school and college football players.

What is added by this report?

During 2005–2014, a total of 28 traumatic brain and spinal cord injury deaths in high school and college football were identified (2.8 deaths per year). The most common playing positions of those fatally injured were running back and linebacker. Approximately 18% of identified high school brain injury deaths were preceded by an earlier concussion, which might have led to second impact syndrome.

What are the implications for public health practice?

Implementing enhanced safety measures to prevent fatalities from catastrophic brain and spinal cord injuries among high school and college football players has the potential to reduce the number of these fatalities. Continued surveillance is important to monitor the circumstances of these deaths and develop risk scenarios to improve prevention measures.

[docs/2004-101/emrgact/emrgact1.html](https://www.cdc.gov/docs/2004-101/emrgact/emrgact1.html)) and information about concussions through the CDC HEADS UP program (<https://www.cdc.gov/headsup/>). Information on state laws related to concussions is available at <https://www.ncsl.org/research/health/traumatic-brain-injury-legislation.aspx>. Catastrophic sport injuries can be reported to the National Center for Catastrophic Sport Injury Research at <http://nccsir.unc.edu/>.

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¹National Center for Catastrophic Sport Injury Research, Department of Exercise & Sport Science, University of North Carolina at Chapel Hill; ²Injury Prevention Research Center, University of North Carolina at Chapel Hill; ³Matthew Gfeller Sport-Related Traumatic Brain Injury Research Center, Department of Exercise & Sport Science, University of North Carolina at Chapel Hill; ⁴Emerson Hospital and Boston University, Massachusetts.

Corresponding author: Kristen Kucera, kkucera@email.unc.edu, 919-962-6228.

References

1. Dompier TP, Kerr ZY, Marshall SW, et al. Incidence of concussion during practice and games in youth, high school, and collegiate American football players. *JAMA Pediatr* 2015;169:659–65. <http://dx.doi.org/10.1001/jamapediatrics.2015.0210>
2. Kerr ZY, Collins CL, Fields SK, Comstock RD. Epidemiology of player—player contact injuries among US high school athletes, 2005–2009. *Clin Pediatr (Phila)* 2011;50:594–603. <http://dx.doi.org/10.1177/0009922810390513>
3. Cantu RC, Mueller FO. Brain injury-related fatalities in American football, 1945–1999. *Neurosurgery* 2003;52:846–53. <http://dx.doi.org/10.1227/01.NEU.0000053210.76063.E4>
4. Boden BP, Breit I, Beachler JA, Williams A, Mueller FO. Fatalities in high school and college football players. *Am J Sports Med* 2013;41:1108–16. <http://dx.doi.org/10.1177/0363546513478572>
5. Mueller FO, Blyth CS. Fatalities from head and cervical spine injuries occurring in tackle football: 40 years' experience. *Clin Sports Med* 1987;6:185–96.
6. Cantu RC, Mueller FO. The prevention of catastrophic head and spine injuries in high school and college sports. *Br J Sports Med* 2009;43:981–6. <http://dx.doi.org/10.1136/bjsm.2009.067728>
7. Haddon W Jr. Energy damage and the ten countermeasure strategies. *J Trauma* 1973;13:321–31. <http://dx.doi.org/10.1097/00005373-197304000-00011>
8. Kerr ZY, Yeargin S, Valovich McLeod TC, et al. Comprehensive coach education and practice contact restriction guidelines result in lower injury rates in youth American football. *Orthop J Sports Med* 2015;3:2325967115594578. <http://dx.doi.org/10.1177/2325967115594578>
9. Casa DJ, Guskiewicz KM, Anderson SA, et al. National athletic trainers' association position statement: preventing sudden death in sports. *J Athl Train* 2012;47:96–118.
10. Pryor RR, Casa DJ, Vandermark LW, et al. Athletic training services in public secondary schools: a benchmark study. *J Athl Train* 2015;50:156–62. <http://dx.doi.org/10.4085/1062-6050-50.2.03>

Adverse Health Effects Associated with Living in a Former Methamphetamine Drug Laboratory — Victoria, Australia, 2015

Jackie Wright, PhD¹; Michaela E. Kenneally²; John W. Edwards, PhD¹; G. Stewart Walker, PhD³

The manufacture of methamphetamine in clandestine drug laboratories occurs in various locations, including residential houses and apartments. Unlike the controlled manufacture of chemicals and drugs, clandestine manufacture results in the uncontrolled storage, use, generation, and disposal of a wide range of chemicals and the deposit of methamphetamine drug residues on indoor surfaces (1). These residues have been found at high levels on porous and nonporous surfaces and have been shown to persist for months to years (1). Persons exposed to these environments often have poorly defined exposures and health effects. It is commonly assumed that these levels of exposure are low compared with those related to illicit drug use or therapeutic use of amphetamine-based drugs for managing behavioral issues such as attention deficit hyperactivity disorder (2). In 2015, a family that was unknowingly exposed to methamphetamine residues in a house in Australia was found to have adverse health effects and elevated methamphetamine levels in hair samples, highlighting the potential for public health risks for persons who might live in methamphetamine-contaminated dwellings. This case study highlights the importance of the identification and effective decontamination of former clandestine drug laboratories.

In May 2013, police seized chemicals and manufacturing equipment from a property in rural Victoria, Australia, and arrested the owner. After completing investigation of the property, police issued a notice to the local council indicating that the property was a former clandestine drug laboratory and posed a potential health risk. The council issued a notice to clean up the property; however, the cleanup was not performed. The property was sold in August 2013, and normal prepurchase checks did not identify the property as a former methamphetamine laboratory or reveal that a notice to clean up the house had been issued. In October 2013, the new owners, a family of five, moved into the house. Seven months later, in May 2014, the local council contacted the owners to advise them that their home was a former drug laboratory. Environmental testing of the home was carried out during late May–October 2014 and revealed elevated levels of methamphetamine on surfaces inside the home, ranging from 11.7–26.0 $\mu\text{g}/100\text{ cm}^2$, well above the Australian limit of 0.5 $\mu\text{g}/100\text{ cm}^2$ (3) for safe levels in a residential home. The family was required to vacate the property in March 2015.

The family included two adults and three children aged 7, 8, and 11 years. None of the family members had ever used methamphetamine or had been taking any prescribed

amphetamine-based medications. Data concerning health effects were obtained from documentation of medical assessments from the family doctor, school medical information, self-reported observations by the family members in response to a questionnaire, and evaluation of behavioral issues, based on the completion of the *Behavior Assessment System for Children, Second Edition* (BASC-2)* Parent Rating Scales (PRS).

Assessment of drug exposure levels was based on the collection and analysis of hair samples from all members of the family 1 week after leaving the home, and from the children, approximately 3 months later. Because none of the family members used amphetamines, through either prescription or illicit drug use, the testing of hair to determine environmental exposure was considered suitable. Hair samples measuring 3–4 cm in length (from the scalp) were collected, which was considered representative of exposure over the 3–4 months preceding sampling. Testing of the hair was conducted at Forensic Science, South Australia by methanolic extraction and analysis of the extract by liquid chromatography with tandem mass spectrometry using electrospray ionization. The hair was subjected to a methanol wash before extraction to remove any recent external contamination. The washes also were analyzed for the presence of amphetamines. The lower limit of detection for this analysis was 5 pg/mg for methamphetamine and amphetamine.

All family members experienced adverse health effects while living in the home. The most serious health effects were reported in the youngest child (a boy aged 7 years) and included development of asthma-like symptoms, trouble sleeping, and behavior changes (Table 1). Of note, a parent-requested cognitive behavioral assessment to evaluate potential gifted traits, undertaken for this child 3 months before moving into the home identified no at-risk or clinically significant behavioral issues. From the BASC-2 PRS evaluation conducted 1 week after moving out of the home, anxiety, attention issues, and somatization were scored as at-risk or clinically significant and were consistent with observations provided by the mother and school personnel. Most of the health problems identified in all family members, including the youngest child, were observed to resolve over time (6–12 months) after they were moved from the contaminated premises (Table 1).

Methamphetamine was detected in the hair of all family members at concentrations ranging from 5 to 460 pg/mg

* <http://www.pearsonclinical.com/education/products/100000658/behavior-assessment-system-for-children-second-edition-basc-2.html>.

(Table 2). Amphetamine was also detected in the hair of the two youngest children, ranging from 16 to 20 pg/mg (Table 2). The highest methamphetamine levels in the hair samples collected were found in the two youngest children (boys aged 7 and 8 years), with a lower level (50 pg/mg) found in the hair of the older child, a girl aged 11 years.

Lower levels of methamphetamine were reported in the hair of the children's mother (17 pg/mg) and father (5 pg/mg). The only detection of methamphetamine in the hair wash was from the mother (8 pg/mg).

Retesting for drugs in hair samples 3 months after moving out of the home (June 2015), indicated clearance of the drug, with no detection of amphetamines in the hair for most family

members. Methamphetamine (60 pg/mg) was reported in the hair sample of the boy aged 8 years and was thought to be related to differences in hair growth rate and hair sample collection.

Discussion

The data from this case study of a single family present evidence of adverse health effects and reflect exposure to methamphetamine that occurred while living in a home with environmental methamphetamine surface contamination levels in the range of 11.7–26.0 $\mu\text{g}/100\text{ cm}^2$.

The highest levels of methamphetamine detected in the hair samples analyzed were from the two younger children. These children had the lowest body weights in the family

TABLE 1. Adverse health effects reported by family members while living in and after vacating a methamphetamine-contaminated house — Victoria, Australia, 2015

Age (yrs)	Sex	Respiratory	Cognitive/Behavioral	Other adverse health effects
7	Male	Persistent cough, asthma-like symptoms	Trouble sleeping, fearfulness, vivid/scary dreams, irritability, aloof, easily distracted. BASC-2 testing: anxiety, somatization, ADHD (at-risk or clinically significant; not present before living in home)	Skin rashes; sore, watery eyes
8	Male	Asthma	BASC-2 testing: no issues identified 1 week after vacating home; anxiety and somatization reported 3 months after vacating home, while living in rented accommodation without access to personal possessions	Sore, watery eyes
11	Female	Persistent cough	Trouble sleeping, irritability. BASC-2 testing: no significant issues identified	Sore, watery eyes
40	Female	Persistent cough	Excess energy	Sore, watery eyes; weight loss; improved distance vision
38	Male	none	Trouble sleeping, decreased memory function (self-reported)	Sore, watery eyes; dizziness and blurry vision while cleaning contaminated areas

Abbreviations: BASC-2 = *Behavior Assessment System for Children, Second Edition*; ADHD = attention deficit hyperactivity disorder.

TABLE 2. Concentrations of methamphetamine and amphetamine in hair samples collected from members of a family exposed to a methamphetamine-contaminated residence 1 week and 3 months after vacating the home — Victoria, Australia, 2015

Age (yrs)	Sex	Concentrations at 1 week after vacating the home (pg/mg)		Concentrations at 3 months after vacating the home (pg/mg)		Factors that might increase or decrease exposure in the home
		Methamphetamine	Amphetamine	Methamphetamine	Amphetamine	
7	Male	460	20	Not detected	Not detected	Regularly played games that involved rolling on the floor, touching all surfaces and running hands along the walls. Infrequent washing of hands.
8	Male	330	16	60	Not detected	
11	Female	50	Not detected	Not detected	Not detected	Spent a lot of time on electronic media and limited active play with brothers.
40	Female	17*	Not detected	Not tested	Not tested	Regularly cleaned the home, including just before hair samples were collected. Has colored hair.
38	Male	5	Not detected	Not tested	Not tested	Works out of the home most of the day, including weekends.

* Methamphetamine (8 pg/mg) also was detected in the methanol wash of this hair sample.

and, according to their parents, had multiple opportunities for regular close contact with indoor surfaces, including engaging in activities that involved rolling on the floor, running around the house, rubbing their hands on walls, and touching items throughout the home. In addition, these children were reported by their parents to wash their hands less frequently than other members of the family. The oldest child reportedly spent more time using electronic media and was involved in limited physical play; her hair methamphetamine level was substantially lower than those of her two brothers.

The lower levels of methamphetamine reported in the hair of the mother potentially reflect lower levels of methamphetamine exposure in the home. In addition, the mother reported that she colored her hair, which has been reported to result in some loss of amphetamine in tested hair samples (4,5). The children's mother reported regularly cleaning the house, including just before hair sampling after moving out of the home. This might account for a measured level of methamphetamine identified in the extract from the external hair wash. The children's father worked away from the home most of the day; his hair had the lowest level of methamphetamine contamination.

Methamphetamine levels in the hair samples of the two younger children (460 pg/mg and 330 pg/mg) are consistent with the lower end of the range reported in children removed from clandestine drug laboratories (range = 100 pg/mg–131,000 pg/mg) (6,7) and chronic adult drug users (range = 100 pg/mg–128,000 pg/mg) (6,8). The levels reported were similar to those reported in studies conducted on low-level methamphetamine use by adults smoking doses of approximately 500–1,000 mg/day (8). The detection of amphetamine in the hair of the younger children might reflect environmental exposures in the home, where low levels of amphetamine also were detected in the surface samples analyzed (but not quantified by the laboratory), or the presence of metabolites, supporting the systemic absorption of methamphetamine. The ratio of methamphetamine to amphetamine in the hair was found to be consistent with the mean reported in hair samples from children removed from clandestine drug laboratories and from drug-exposed children (6,7).

The most substantial health effects were in the youngest child, who also had the highest measured levels of methamphetamine in hair. The health effects reported in this study also have been reported in children removed from active methamphetamine drug laboratories (9). Follow-up with the family has identified that the respiratory effects, trouble sleeping, and behavioral changes mostly resolved during the 12 months after the family vacated the contaminated home.

The findings in this report are subject to at least two limitations. First, there is no established quantitative relationship between the dose of methamphetamine to which a person is exposed and the measured methamphetamine level in hair. In addition, there

Summary

What is already known about this topic?

The clandestine manufacture of methamphetamine is known to result in various levels of contamination of all surfaces in homes. Information is available on drug exposures and health effects for drug users as well as persons exposed during manufacture.

What is added by this report?

A family of five, including three children aged 7–11 years, lived in a home in rural Victoria, Australia, that was previously a clandestine methamphetamine drug laboratory with documented environmental contamination. The family members developed adverse health effects, and there was evidence of systemic absorption of methamphetamine from the environment, based on hair samples collected after they had vacated the premises. Health effects were most pronounced in the youngest child, who also had the highest methamphetamine levels in hair, possibly related to a combination of repeated contact with surfaces during play activities and less frequent hand washing.

What are the implications for public health practice?

If properties formerly used for the clandestine manufacture of methamphetamine are not properly cleaned the public might be unknowingly exposed to drug residues. Appropriate identification and management of these properties, including measures by authorities to prevent the sale of unremediated homes, are important to prevent exposures and adverse health effects.

is significant variation among persons who might have had the same level of exposure and the measured levels in hair, although for an individual, correlation between dose and the concentration of amphetamines in hair has been reported (8,10). However, the data reported for the family members support the association of higher levels of methamphetamine and amphetamine in hair with higher levels of exposure (based on reported activities, body weight, and time in the contaminated home). The fact that hair concentrations declined precipitously after the exposure was removed is consistent with an association between exposure and elevated hair levels. Second, the presence of methamphetamine and amphetamine in the hair samples collected reflect exposures that might have occurred during the preceding 3–4 months (10) with the environmental data (surface residue sampling) reflecting contamination levels at the time of sampling only (approximately 3–6 months before the collection of hair samples). Levels of environmental contamination and exposures that might have occurred before this time are not known and might have been higher.

Residual environmental methamphetamine contamination can result in adverse health effects in exposed persons, particularly in young children. Appropriate identification and management of former clandestine drug laboratories, including appropriate remediation and measures to prevent the sale of contaminated homes, is important to prevent exposures and adverse health effects.

¹Health and Environment, School of the Environment, Flinders University, Adelaide, South Australia, Australia; ²Toxicology, Forensic Science SA, Government of South Australia, Adelaide; ³School of Chemical & Physical Sciences, Flinders University, Adelaide, South Australia, Australia.

Corresponding author: Jackie Wright, jackie@enrisks.com.au.

References

- Martyny JW, Arbuckle SL, McCammon CS Jr, Esswein EJ, Erb N, Van Dyke M. Chemical concentrations and contamination associated with clandestine methamphetamine laboratories. *J Chem Health Saf* 2007;14:40–52. <http://dx.doi.org/10.1016/j.jchas.2007.01.012>
- Weisheit R. Making methamphetamine. *South Rural Sociol* 2008;23:78–107.
- Australian Government. Clandestine drug laboratory remediation guidelines. Canberra, Australia: Commonwealth of Australia; 2011. <https://www.ag.gov.au/CrimeAndCorruption/Drugs/Documents/Clandestinedruglaboratoryremediationguidelines.pdf>
- Baeck S, Han E, Chung H, Pyo M. Effects of repeated hair washing and a single hair dyeing on concentrations of methamphetamine and amphetamine in human hairs. *Forensic Sci Int* 2011;206:77–80. <http://dx.doi.org/10.1016/j.forsciint.2010.06.023>
- Tsanaclis L, Wicks JF. Patterns in drug use in the United Kingdom as revealed through analysis of hair in a large population sample. *Forensic Sci Int* 2007;170:121–8. <http://dx.doi.org/10.1016/j.forsciint.2007.03.033>
- Bassindale T. Quantitative analysis of methamphetamine in hair of children removed from clandestine laboratories—evidence of passive exposure? *Forensic Sci Int* 2012;219:179–82. <http://dx.doi.org/10.1016/j.forsciint.2012.01.003>
- Castaneto MS, Barnes AJ, Scheidweiler KB, et al. Identifying methamphetamine exposure in children. *Ther Drug Monit* 2013;35:823–30. <http://dx.doi.org/10.1097/FTD.0b013e31829685b2>
- Han E, Paulus MP, Wittmann M, Chung H, Song JM. Hair analysis and self-report of methamphetamine use by methamphetamine dependent individuals. *J Chromatogr B Analyt Technol Biomed Life Sci* 2011;879:541–7. <http://dx.doi.org/10.1016/j.jchromb.2011.01.002>
- Wright J, Edwards J, Walker S. Exposures associated with clandestine methamphetamine drug laboratories in Australia. *Rev Environ Health* 2016;31:329–52. <http://dx.doi.org/10.1515/reveh-2016-0017>
- Polettini A, Cone EJ, Gorelick DA, Huestis MA. Incorporation of methamphetamine and amphetamine in human hair following controlled oral methamphetamine administration. *Anal Chim Acta* 2012;726:35–43. <http://dx.doi.org/10.1016/j.aca.2012.01.042>

Human Rabies — Puerto Rico, 2015

Ashley Styczynski, MD^{1,2}; Cuc Tran, PhD^{1,2}; Emilio Dirlikov, PhD^{1,3}; María Ramos Zapata, MPH³; Kyle Ryff, MPH³; Brett Petersen, MD²; Anibal Cruz Sanchez, MPH³; Marrielle Mayshack^{3,4}; Laura Castro Martinez, MPH³; Rene Condori, MS²; James Ellison, PhD²; Lillian Orciari, MS²; Pamela Yager²; Rafael González Peña³; Dario Sanabria, MD⁵; Julio Cádiz Velázquez, MD³; Dana Thomas, MD^{3,6}; Brenda Rivera García, DVM³

On December 1, 2015, the Puerto Rico Department of Health (PRDH) was notified by a local hospital of a suspected human rabies case. The previous evening, a Puerto Rican man aged 54 years arrived at the emergency department with fever, difficulty swallowing, hand paresthesia, cough, and chest tightness. The next morning the patient left against medical advice but returned to the emergency department in the afternoon with worsening symptoms. The patient's wife reported that he had been bitten by a mongoose during the first week of October, but had not sought care for the bite. While being transferred to the intensive care unit, the patient went into cardiac arrest and died. On December 3, rabies was confirmed from specimens collected during autopsy. PRDH conducted an initial rapid risk assessment, and five family members were started on rabies postexposure prophylaxis (PEP).

Given potential additional exposures, PRDH and CDC undertook contact investigations among additional community and family members (N = 32) and hospital personnel (N = 39) to identify persons who required PEP. After the contact investigations, two additional family members and two hospital staff members received PEP. PRDH recommends that persons with a history of a mongoose bite should seek medical care and receive PEP. Health care providers should maintain a high index of clinical suspicion for rabies, including taking a history of animal exposure and adhering to recommended infection control practices when examining and treating anyone with suspected rabies or with acute, progressive encephalitis. Despite a high prevalence of rabies among mongoose populations in Puerto Rico, this is the first rabies-associated death directly related to a mongoose bite in Puerto Rico. In 2003, a case of human rabies occurred in a person infected with a mongoose variant of the virus after a dog bite (1). This case represents only the third documented rabies death in Puerto Rico during the past century. In Puerto Rico, public health outreach activities should continue to educate members of the community on mongoose-associated rabies and PEP.

Case Report

The patient, a man aged 54 years, was a resident of south-eastern Puerto Rico. On the evening of November 30, he came to a local emergency department with fever, difficulty swallowing, hand paresthesia, cough, and chest tightness. He had refused most food and drink for the preceding 5 days and had

difficulty managing his oral secretions. No history of animal exposure was elicited. He was given a preliminary diagnosis of lower respiratory tract infection and started empirically on antibiotics and antiemetics. A subsequent chest radiograph and computed tomography scan of his head were performed, which were both normal. The next morning, the patient left the hospital against medical advice.

The patient returned to the emergency department in the afternoon with worsening symptoms. The patient's wife, who accompanied him on this visit, reported that during the first week of October he had been bitten by a mongoose while tending to a chicken coop located on their property; he had not sought care after the encounter. Clinical suspicion of rabies triggered notification to PRDH. Shortly thereafter, the patient experienced cardiac arrest while being transferred to the intensive care unit and could not be resuscitated.

An autopsy was performed on December 2 at the Puerto Rico Institute for Forensic Sciences, and specimens were submitted to the PRDH Public Health Laboratory for rabies testing. On December 3, results of direct fluorescent antibody testing were positive for rabies infection. Additional specimens sent to CDC for confirmatory testing were positive by direct fluorescent antibody and reverse transcription–polymerase chain reaction, and antigenic typing and sequence analysis were consistent with Caribbean mongoose rabies virus variant.

Public Health Investigation

Upon notification of the suspected case on December 1, PRDH collaborated with CDC's Poxvirus and Rabies Branch. An initial rapid risk assessment conducted by PRDH identified five family members who had potential exposures through close contact with the deceased patient, and all five family members were started on PEP. These persons included the patient's immediate family and household members.

Beginning on December 9, PRDH and CDC initiated contact investigations among additional family and community members as well as hospital personnel to determine other persons with potential exposure who required PEP. Based on the Advisory Committee on Immunization Practices guidelines for rabies virus exposures, PEP is recommended for persons with contact with the patient's saliva, tears, or cerebrospinal fluid to open wounds or mucous membranes during the infectious period (2 weeks before symptom onset) (2). A total of 76 contacts were

evaluated for their risk for exposure, including two additional family members who required PEP because of exposure to the patient while he was hospitalized (Table). Among the 37 family and community contacts, median age was 33 (range = 1–78), and 20 (54%) were female. Municipality of residence was the same as that of the deceased patient for the 34 community members who reported place of residence. In total, seven (19%) of the 37 family and community members received PEP.

Thirty-nine hospital personnel were reported to have had contact with the patient. These staff members worked in various positions, including intensive care, respiratory therapy, and patient transport. Median age was 35 years (range = 23–65), and 18 (46%) were female. Through the contact investigation, two (5%) of the hospital staff members who had contact with the patient received PEP because they had exposures to the patient's saliva onto open wounds or mucous membranes. These exposures resulted from not wearing gloves or masks in the situations indicated in standard precautions (3), namely intubation and management of oral secretions.

After the contact investigations, education and outreach were conducted to inform community members and hospital personnel about rabies. PRDH designed and distributed educational materials to address the most frequently asked questions and held an informational session with the community to promote open discussion. In addition, hospital personnel participated in a debriefing that highlighted the need for appropriate use of standard precautions with all patients, regardless of suspected diagnosis.

Discussion

This is the first reported case of human rabies associated with a mongoose bite in North America. Mongooses were introduced from India to the Caribbean, including Puerto Rico, during the 19th century to control rat populations in

sugarcane fields (4) and have become the principal reservoir of rabies in Puerto Rico, accounting for nearly 75% of all animal rabies cases (5,6). In Puerto Rico, mongoose-associated rabies virus is phylogenetically linked to the North Central skunk and cosmopolitan dog variants (7). Seroprevalence of rabies virus-neutralizing antibodies in the mongoose population is estimated at 40% (8). Seventy-five mongoose bites were reported in 2014 (1.9 bites per 100,000 persons); during 2005–2008, 97% of 151 submitted animal specimens after mongoose bites were positive for rabies virus.* PRDH recommends rabies PEP after all mongoose bites if the animal is not available for testing, and an estimated 95% of persons reporting mongoose bites receive PEP.

This case highlights the need for increased public awareness for the potential for mongoose-related rabies in Puerto Rico. The standardized risk assessment tool used in the contact investigations ensured that contacts with exposures promptly received PEP, thus mitigating costs from indiscriminate use of PEP. This tool could be adapted for use in other rabies exposure risk assessments in Puerto Rico or elsewhere. Health care providers should routinely assess for animal exposures in the medical history and maintain a high index of suspicion for rabies when animal exposure has occurred or is suspected. More generally, universal use and monitoring of standard precautions are necessary to minimize risk for exposures to infectious diseases in health care settings. Occupational exposures to rabies in health care settings frequently occur as a breach of standard precautions (9). Among hospital personnel interviewed for this investigation, only two (5%) had an exposure, and both received PEP.

* Rivera-García B. Profile of mongoose inflicted bite injuries and PEP referral in Puerto Rico during fiscal years 2005–2008. Presented at Rabies in the Americas XX Conference, October 20, 2009, Québec, Canada.

TABLE. Characteristics of hospital and community contacts of human rabies case — Puerto Rico, 2015

Characteristic	Hospital (N = 39)			Community (N = 37)		
	Unexposed	Exposed*	Total	Unexposed	Exposed*	Total
Median age (range) (yrs)	35 (23–65)	46 (34–58)	35 (23–65)	33 (6–78)	33 (1–56)	33 (1–78)
Male, no. (%)	20 (51)	1 (3)	21 (54)	13 (76)	4 (24)	17 (46)
Relationship to patient, no. (%)						
Physician	8 (22)	1 (50)	9 (23)	NA	NA	NA
Nurse	11 (30)	1 (50)	12 (31)	NA	NA	NA
Medical student	5 (14)	0	5 (13)	NA	NA	NA
Respiratory therapist	5 (14)	0	5 (13)	NA	NA	NA
Radiology technician/Patient escort	4 (11)	0	4 (10)	NA	NA	NA
Janitorial staff	4 (11)	0	4 (10)	NA	NA	NA
Immediate family	NA	NA	NA	1 (3)	5 (71)	6 (16)
Relatives and friends	NA	NA	NA	29 (97)	2 (29)	31 (84)
Total	37 (100)	2 (100)	39	30 (100)	7 (100)	37

Abbreviation: NA = not applicable.

* Based on Advisory Committee on Immunization Practices recommendations for postexposure prophylaxis.

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

Human rabies associated with a mongoose encounter has never been reported in the United States or U.S. territories; however, studies by the U.S. Department of Agriculture indicate rabies seropositivity of approximately 40% among the Puerto Rican mongoose population. Because of the public health risk, Puerto Rico provides rabies postexposure prophylaxis (PEP) to any patient who experiences a mongoose bite.

What is added by this report?

A man aged 54 years who was bitten by a mongoose in October 2016 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. Limited awareness of rabies prevention and symptoms of the disease by the general public and health care personnel was likely a contributing factor in the exposures to the patient that required PEP.

What are the implications for public health practice?

This case highlights the importance of public and health care provider awareness of rabies to prevent adverse outcomes after exposures and reduce unnecessary exposures. This awareness includes maintaining a higher suspicion for zoonotic diseases by including animal exposures in the medical history. Universal use and monitoring of standard precautions in health care settings are necessary to minimize risk for occupational exposure to infectious diseases such as rabies when the nature of the illness is unknown.

Public health measures to reduce the risk for human rabies should include increased resources for primary prevention, including routine pet vaccination (canine rabies in Puerto Rico results from transmission from mongooses) and preexposure prophylaxis for persons at highest risk. Community education should highlight measures to avoid bites from pets and wildlife. Effective oral rabies vaccine baits targeting mongooses might also be considered as they become commercially available (10). Interventions should focus on areas with known human-mongoose contacts, as determined by overlaying bite surveillance data and population density. Secondary prevention measures should be aimed at increasing awareness of the need for medical evaluation and PEP after any mongoose bite.

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¹Epidemic Intelligence Service, Division of Scientific Education and Professional Development, CDC; ²Poxvirus and Rabies Branch, National Center for Emerging and Zoonotic Infectious Diseases, CDC; ³Puerto Rico Department of Health; ⁴Office for State, Tribal, Local, and Territorial Support, CDC; ⁵Puerto Rico Institute for Forensic Sciences; ⁶Division of State and Local Readiness, Office of Public Health Preparedness and Response, CDC.

Corresponding author: Ashley Styczynski, astyczynski@cdc.gov, 404-661-6663.

References

- Petersen BW, Rupprecht CE. Human rabies epidemiology and diagnosis [Chapter 10]. In: Tkachev S, ed. Non-flavivirus encephalitis. Rijeka, Croatia: InTech Open Science; 2011.
- Manning SE, Rupprecht CE, Fishbein D, et al.; Advisory Committee on Immunization Practices Centers for Disease Control and Prevention (CDC). Human rabies prevention—United States, 2008: recommendations of the Advisory Committee on Immunization Practices. *MMWR Recomm Rep* 2008;57(No. RR-3).
- Garner JS; The Hospital Infection Control Practices Advisory Committee. Guideline for isolation precautions in hospitals. *Infect Control Hosp Epidemiol* 1996;17:53–80. <http://dx.doi.org/10.2307/30142367>
- Tierkel ES, Arbona G, Rivera A, de Juan A. Mongoose rabies in Puerto Rico. *Public Health Rep* 1952;67:274–8. <http://dx.doi.org/10.2307/4588059>
- Dyer JL, Yager P, Orciari L, et al. Rabies surveillance in the United States during 2013. *J Am Vet Med Assoc* 2014;245:1111–23. <http://dx.doi.org/10.2460/javma.245.10.1111>
- Krebs JW, Smith JS, Rupprecht CE, Childs JE. Rabies surveillance in the United States during 1998. *J Am Vet Med Assoc* 1999;215:1786–98.
- Nadin-Davis SA, Velez J, Malaga C, Wandeler AI. A molecular epidemiological study of rabies in Puerto Rico. *Virus Res* 2008;131:8–15. <http://dx.doi.org/10.1016/j.virusres.2007.08.002>
- Berentsen AR, Johnson SR, Gilbert AT, VerCauteren KC. Exposure to rabies in small Indian mongooses (*Herpestes auro-punctatus*) from two regions in Puerto Rico. *J Wildl Dis* 2015;51:896–900. <http://dx.doi.org/10.7589/2015-01-016>
- Kan VL, Joyce P, Benator D, et al. Risk assessment for healthcare workers after a sentinel case of rabies and review of the literature. *Clin Infect Dis* 2015;60:341–8. <http://dx.doi.org/10.1093/cid/ciu850>
- Blanton JD, Meadows A, Murphy SM, et al. Vaccination of small Asian mongoose (*Herpestes javanicus*) against rabies. *J Wildl Dis* 2006;42:663–6. <http://dx.doi.org/10.7589/0090-3558-42.3.663>

***Escherichia coli* O157:H7 Infections Associated with Contaminated Pork Products — Alberta, Canada, July–October 2014**

Lance Honish, MSc^{1,2}; Nisha Punja^{1,2}; Sarah Nunn^{1,2}; Dale Nelson^{1,2}; Nyal Hislop, MSc^{1,2}; Garth Gosselin^{1,2}; Natisha Stashko, PhD^{2,3}; Darlene Dittrich^{2,3}

During July–October 2014, an outbreak of 119 *Escherichia coli* O157:H7 infections in Alberta, Canada was identified through notifiable disease surveillance and investigated by local, provincial, and federal public health and food regulatory agencies. Twenty-three (19%) patients were hospitalized, six of whom developed hemolytic uremic syndrome; no deaths were reported. Informed by case interviews, seven potential food sources were identified and investigated. The majority of patients reported having consumed meals containing pork at Asian-style restaurants in multiple geographically diverse Alberta cities during their exposure period. Traceback investigations revealed a complex pork production and distribution chain entirely within Alberta. *E. coli* O157:H7–contaminated pork and pork production environments and mishandling of pork products were identified at all key points in the chain, including slaughter, processor, retail, and restaurant facilities. An outbreak-specific pulsed-field gel electrophoresis (PFGE) cluster pattern was found in clinical and pork *E. coli* O157:H7 isolates. Measures to mitigate the risk for exposure and illness included pork product recalls, destruction of pork products, temporary food facility closures, targeted interventions to mitigate improper pork-handling practices identified at implicated food facilities, and prosecution of a food facility operator. Pork should be considered a potential source in *E. coli* O157:H7 investigations and prevention messaging, and pork handling and cooking practices should be carefully assessed during regulatory food facility inspections.

Epidemiologic Investigation

For this outbreak, a case was defined as a laboratory culture-confirmed *E. coli* O157:H7 infection with one of 16 PFGE cluster patterns identified in a resident of or visitor to Canada during July–October 2014. Cases were identified through notifiable disease surveillance.

A total of 119 outbreak cases were identified, including four (3%) in patients who were classified as having secondary infections (i.e., acquired through household contact with an outbreak-associated patient). All patients were in Alberta during all or part of the incubation period. Dates of illness onsets for the 119 patients ranged from July 20 to October 6 (Figure 1). Cases occurred among persons in a wide geographic distribution across Alberta. Twenty-three (19%) patients were hospitalized, six of whom developed hemolytic uremic syndrome; no

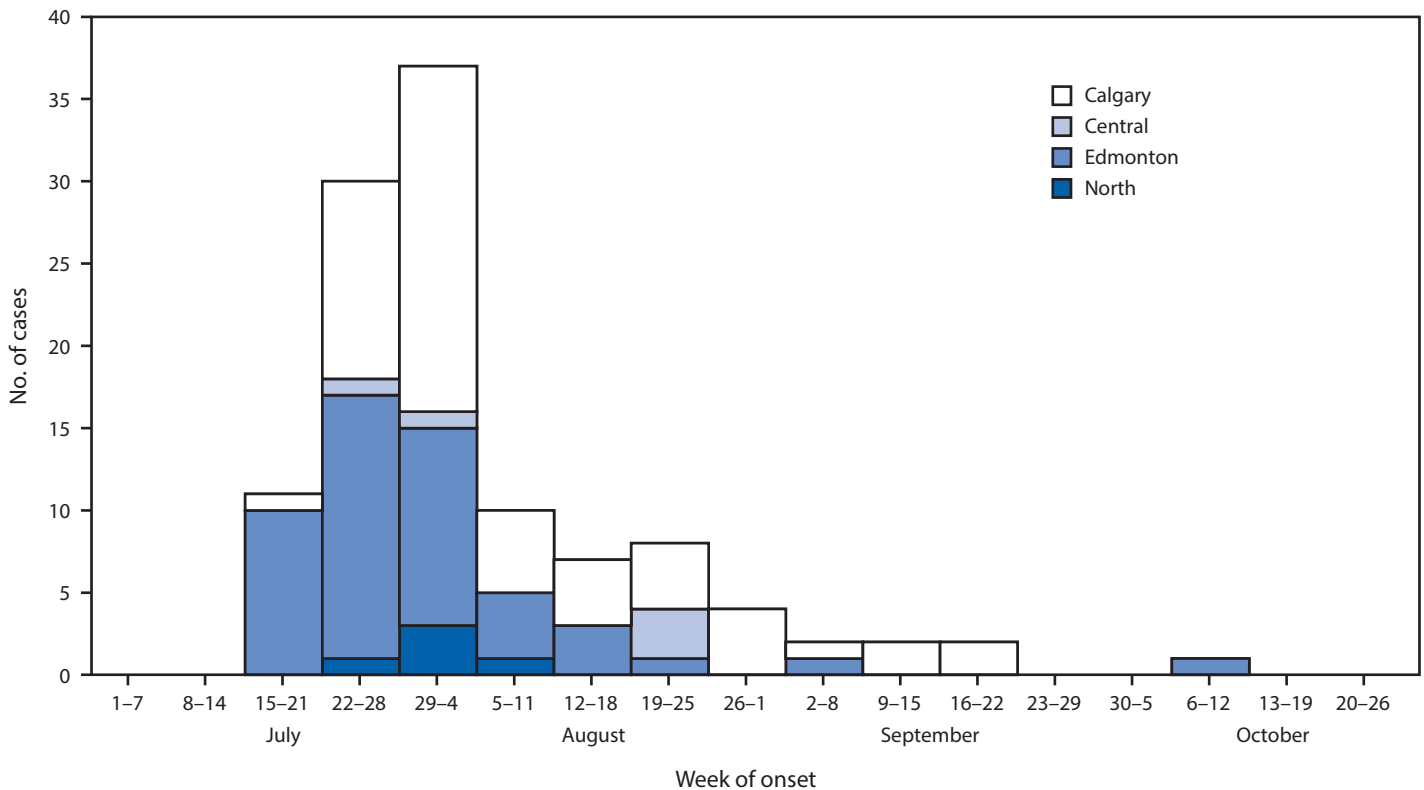
deaths were reported. The median age of patients was 23 years (range = 1–82 years), and 76 patients (64%) were female.

Exposure to food at Alberta Asian-style restaurants (36 facilities widely distributed across the province) was reported by 85 (74%) of the 115 primary outbreak patients. Routine public health follow-up interviews failed to identify the source. Enhanced interviews with patients and follow-up at restaurants revealed that the exposure-specific frequency for each of seven ingredients (mung bean sprouts, beef, carrots, cucumbers, green onions, lettuce, and pork) exceeded 35%.

Environmental Investigation

Regulatory agencies conducted inspections at 201 restaurant and food processing facilities to inform the investigation and control the outbreak. Extensive investigation of Alberta mung bean sprout supplier/distributor facilities ruled out this product as a source. A traceback investigation was initiated that focused on suppliers of the six remaining high exposure-frequency foods. No single common restaurant supplier was identified for these foods. Pork was identified as the only ingredient with a supplier network entirely within Alberta, and thus emerged as the leading hypothesized source of the outbreak. Confirmation of the complex intra-Alberta pork supplier network (Figure 2) revealed that exposure to food from a facility within the network was the most common identified exposure (Table) among primary outbreak patients (96/115, 83%). Most of these exposures occurred at restaurants (81, 84%). Consumption of pork was identified among 65% of outbreak patients. A total of 295 samples, including environmental surface swabs (n = 157), food (116), food surface swabs (13), and water (9), were collected and analyzed for the presence of *E. coli* O157:H7. Although a range of sample types were collected during hypothesis generation, sample collection later focused on pork and pork-production environments, as informed by the investigation. *E. coli* O157:H7 was identified in 18 samples,* all of which were from pork or pork products or surface swabs in pork production facilities. Apart from two isolates from a slaughter facility, PFGE cluster patterns identified

* Eighteen *E. coli* O157:H7–positive samples were obtained from the pork production environment (n = 1); pork production equipment (5); pork carcass (1); raw fresh pork (4); raw frozen pork (1); raw marinated pork (3); spring rolls containing raw pork (1); chicken sausage containing raw pork (1); and a delivery vehicle (1) among one slaughter facility (facility F), two processing/distribution facilities (facilities B and C), one restaurant, and two private dwellings.

FIGURE 1. Cases of pork-associated *Escherichia coli* O157:H7 infection by week of onset and region — Alberta, Canada, July–October 2014*

* Excludes five outbreak cases in persons who were not Alberta residents.

in patient isolates matched those in food and environmental sample isolates. Four outbreak cases were associated with exposure to chicken sausage products from one facility; laboratory analysis of the products identified *E. coli* O157:H7, detected pork, and did not detect poultry. Investigation revealed that the chicken product producer had purchased pork fraudulently labeled as chicken by an illegal distributor linked to a facility in the Alberta pork-supplier network.

Public Health Response

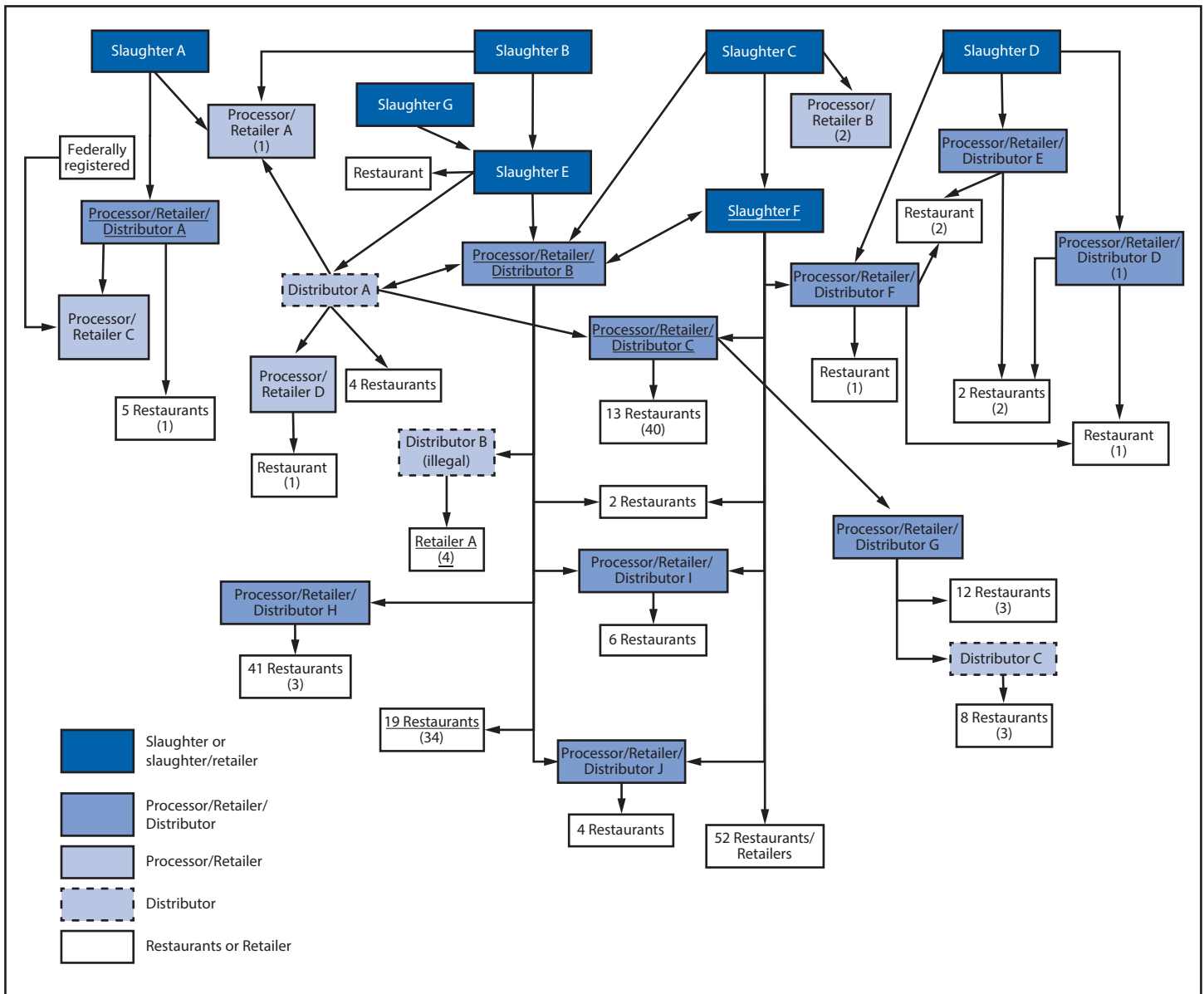
The local health department ordered four facilities, including one slaughter/retail facility, two processor/distributor/retail facilities, and one restaurant facility, to temporarily close because of the numbers of cases associated with exposure to food distributed by the facility, critical food handling violations identified, or *E. coli* O157:H7–positive surface swabs. The illegal pork distributor fraudulently selling pork as chicken was issued court orders to close the business and to appear for questioning. The operator failed to appear, and an arrest warrant was issued. The Canadian Food Inspection Agency issued recall notices for pork products (and chicken products containing pork) distributed by six facilities. Multiple news

releases issued to local media outlets informed the public of the outbreak investigation.

Root cause analyses were conducted by food regulatory agencies at four slaughter facilities implicated in the pork supplier network. All facilities slaughtered multiple species, including cattle. Common observations included opportunities for cross-contamination related to sharing of animal pens, inadequate cleaning and sanitation of knives or equipment between carcasses, and close proximity of carcasses during slaughter activities. At the slaughter facility that was temporarily closed, inconsistent personnel hygiene practices and poor knowledge of food safety were also identified. Corrective actions related to sanitary dressing procedures, process flow, hygiene, hand-washing, cleaning, and sanitation were initiated and monitored through routine inspections. Products suspected of being contaminated were removed from one facility.

Environmental Health Officers (EHOs) with the local health department conducted comprehensive assessments of pork-handling practices and other potential contributing factors at 111 restaurants (those at which patients were thought to have acquired their infection and additional, selected similar restaurants in Alberta). EHOs observed practices used by operators at baseline, surveyed them about their procedures using

FIGURE 2. Alberta pork supplier network, pork-associated *Escherichia coli* O157:H7 outbreak — Alberta, Canada, July–October 2014*†,‡,§,¶



* Underlined facility = *E. coli* O157:H7–positive sample collected from the facility directly or indirectly (i.e., at home of outbreak case).

† Numbers in brackets = number of outbreak cases with exposure to facility.

‡ Some cases had multiple facility exposures.

§ Four secondary cases are excluded.

a standardized questionnaire, and used this information to inform intervention strategies. Only 32% of operators used validated or standardized procedures for cooking pork products; 77% used visual indicators to ascertain whether pork products were adequately cooked. Cross-contamination concerns that might have contributed to infection were identified in several restaurants; for example, 74% of facilities did not use a cleaning schedule for food equipment, and food handlers did not wash their hands between tasks in 54% of facilities. At facilities that met food safety training requirements (82%), trained

personnel often did not have direct oversight of day-to-day food handling activities. Interventions and ongoing monitoring programs with short, intermediate, and long-term objectives were implemented at the facilities to mitigate identified problems. This phased approach included delivery of onsite food safety training by EHOs, development and distribution of educational resources in the first language of employees (printed and online), and assistance with the creation of food safety plans for properly cooking pork products. Mitigation strategies included the distribution of digital thermometers and

TABLE. Exposure characteristics of 115* primary cases of pork-associated *Escherichia coli* O157:H7 — Alberta, Canada, July–November, 2014

Potential exposure sites	No. of patients with exposure to site	No. of patients with exposure to pork (%)
Asian-style restaurant(s) [†]	81	48 (59)
Asian-style market [†]	3	1 (33)
Sausage producer/retailer [†]	4	4 (100)
Festival temporary food facility [†]	7	7 (100)
Meat processor/retailer [†]	1	1 (100)
Asian-style restaurant(s) [§]	4	4 (100)
No suspect source facility [¶]	12	10 (83)
Poor historian	3	NA
Total	115	75 (65)

Abbreviation: NA = not applicable.

* Four secondary cases excluded.

[†] Facility within implicated pork supplier chain (96/115 primary cases had this exposure).

[§] Facility outside implicated pork supplier chain.

[¶] After complete exposure assessment.

digital timers by EHOs. During onsite training sessions, EHOs demonstrated proper handwashing and environmental surface sanitation procedures and identified other strategies operators could use to reduce the likelihood of cross-contamination. Compliance with these food safety elements was measured before and after mitigation strategies were carried out to help evaluate selected intervention measures.

Discussion

This outbreak represents the second largest foodborne and third largest overall *E. coli* O157:H7 outbreak in Canadian history, after a foodborne outbreak associated with salami produced in British Columbia in 1999 with 143 laboratory-confirmed cases (1) and a waterborne outbreak in Walkerton, Ontario in 2000 with 167 laboratory-confirmed cases (2). Strong epidemiologic evidence exists indicating that the cause of this outbreak was exposure to contaminated pork products produced and distributed in Alberta. The molecular epidemiology of the clinical and pork *E. coli* O157:H7 outbreak isolates is described elsewhere (3). Pork is a known, although infrequent, source of human *E. coli* O157 infection (4–8). Most documented outbreaks have been associated with sausage products containing pork and other meats, and the species-specific source of contamination was not confirmed. It has been reported that *E. coli* O157:H7 is prevalent globally at varying rates in swine, that infected swine might shed the bacteria for 2 months,

Summary

What is already known about this topic?

Pork is a known, although infrequent, source of human *Escherichia coli* O157:H7 infection. *E. coli* O157:H7 infections often result in clinically severe illness with serious complications in humans.

What is added by this report?

During July–October 2014, an outbreak of 119 cases of *E. coli* O157:H7 infections associated with exposure to contaminated pork products occurred in Alberta, Canada. *E. coli* O157:H7–contaminated pork and pork production environments and mishandling of pork products were identified at all key points in the implicated pork distribution chain. Measures to control the outbreak included product recalls, destruction of pork products, temporary food facility closures, targeted interventions to mitigate improper pork-handling practices, and prosecution of a food facility operator.

What are the implications for public health practice?

Pork should be considered in public health *E. coli* O157:H7 investigations and prevention messaging, and pork handling and cooking practices should be carefully assessed during regulatory food facility inspections.

and that horizontal transmission between swine and other livestock species might occur (9).

E. coli O157:H7–contaminated pork and pork production environments and mishandling of pork products were identified at all key points in the implicated Alberta pork distribution chain, including slaughter, processor, retail, and restaurant facilities. However, the originating source or sources of the contamination were not identified. Cross-contamination appears to be an important contributing factor in this outbreak, as evidenced by absence of known pork exposure in 35% of outbreak cases. On the basis of the findings of this investigation, pork should be considered a potential source in public health *E. coli* O157:H7 investigations and prevention messaging, and pork handling and cooking practices should be carefully assessed during regulatory food facility inspections.

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¹Alberta Health Services, Canada; ²Investigative Team from Alberta Health Services, Alberta Provincial Laboratory for Public Health, Alberta Health, Alberta Agriculture and Forestry, Public Health Agency of Canada, Canadian Food Inspection Agency, Health Canada, First Nations and Inuit Health Branch; ³Alberta Agriculture and Forestry, Canada.

Corresponding author: Lance Honish, lance.honish@ahs.ca.

References

1. MacDonald DM, Fyfe M, Paccagnella A, Trinidad A, Louie K, Patrick D. *Escherichia coli* O157:H7 outbreak linked to salami, British Columbia, Canada, 1999. *Epidemiol Infect* 2004;132:283–9. <http://dx.doi.org/10.1017/S0950268803001651>
2. Bruce-Grey-Owen Sound Health Unit. Waterborne outbreak of gastroenteritis associated with a contaminated municipal water supply, Walkerton, Ontario, May–June 2000. *Can Commun Dis Rep* 2000;26:170–3.
3. Berenger BM, Berry C, Peterson T, et al. The utility of multiple molecular methods including whole genome sequencing as tools to differentiate *Escherichia coli* O157:H7 outbreaks. *Euro Surveill* 2015;20:30073. <http://dx.doi.org/10.2807/1560-7917.ES.2015.20.47.30073>
4. Trotz-Williams LA, Mercer NJ, Walters JM, Maki AM, Johnson RP. Pork implicated in a Shiga toxin-producing *Escherichia coli* O157:H7 outbreak in Ontario, Canada. *Can J Public Health* 2012;103:e322–6.
5. CDC. *Escherichia coli* O157:H7 outbreak linked to commercially distributed dry-cured salami—Washington and California, 1994. *MMWR Morb Mortal Wkly Rep* 1995;44:157–60.
6. Paton AW, Ratcliff RM, Doyle RM, et al. Molecular microbiological investigation of an outbreak of hemolytic-uremic syndrome caused by dry fermented sausage contaminated with Shiga-like toxin-producing *Escherichia coli*. *J Clin Microbiol* 1996;34:1622–7.
7. Williams RC, Isaacs S, Decou ML, et al. Illness outbreak associated with *Escherichia coli* O157:H7 in Genoa salami. *E. coli* O157:H7 Working Group. *CMAJ* 2000;162:1409–13.
8. Conedera G, Mattiazzi E, Russo F, et al. A family outbreak of *Escherichia coli* O157 haemorrhagic colitis caused by pork meat salami. *Epidemiol Infect* 2007;135:311–4. <http://dx.doi.org/10.1017/S0950268806006807>
9. Tseng M, Fratamico PM, Manning SD, Funk JA. Shiga toxin-producing *Escherichia coli* in swine: the public health perspective. *Anim Health Res Rev* 2014;15:63–75. <http://dx.doi.org/10.1017/S1466252313000170>

Zika Virus — 10 Public Health Achievements in 2016 and Future Priorities

Nadia L. Oussayef, JD¹; Satish K. Pillai, MD¹; Margaret A. Honein, PhD¹; C. Ben Beard, PhD¹; Beth Bell, MD¹; Coleen A. Boyle, PhD¹; Lars M. Eisen, PhD¹; Katrin Kohl, MD, PhD¹; Matthew J. Kuehnert, MD¹; Eva Lathrop, MD¹; Stacey W. Martin, MSc¹; Rebecca Martin, PhD¹; Janet C. McAllister, PhD¹; Elizabeth Pantino McClune, MSW, MPA¹; Paul Mead, MD¹; Dana Meaney-Delman, MD¹; Brett Petersen, MD¹; Lyle R. Petersen, MD¹; Kara N.D. Polen, MPH¹; Ann M. Powers, PhD¹; Stephen C. Redd, MD¹; James J. Sejvar, MD¹; Tyler Sharp, PhD¹; Julie Villanueva, PhD¹; Denise J. Jamieson, MD¹

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The introduction of Zika virus into the Region of the Americas (Americas) and the subsequent increase in cases of congenital microcephaly resulted in activation of CDC's Emergency Operations Center on January 22, 2016, to ensure a coordinated response and timely dissemination of information, and led the World Health Organization to declare a Public Health Emergency of International Concern on February 1, 2016. During the past year, public health agencies and researchers worldwide have collaborated to protect pregnant women, inform clinicians and the public, and advance knowledge about Zika virus (Figure 1). This report summarizes 10 important contributions toward addressing the threat posed by Zika virus in 2016. To protect pregnant women and their fetuses and infants from the effects of Zika virus infection during pregnancy, public health activities must focus on preventing mosquito-borne transmission through vector control and personal protective practices, preventing sexual transmission by advising abstinence from sex or consistent and correct use of condoms, and preventing unintended pregnancies by reducing barriers to access to highly effective reversible contraception.

1. Issuing Travel Guidance to Warn Pregnant Women Not to Travel to Areas with Ongoing Zika Virus Transmission

On January 15, 2016, CDC issued a travel notice to alert travelers about the risk of Zika virus transmission in 14 countries and territories in Central and South America and the Caribbean, including Puerto Rico. As of December 15, 2016, a total of 60 international Zika travel notices have been issued, including 49 in the Americas. These notices advise pregnant women to avoid travel to areas of active Zika virus transmission, provide recommendations for travelers to avoid exposure to Zika virus, and inform returning travelers about transmission prevention and testing. On August 1, 2016, after the first instance of confirmed mosquito-borne Zika virus transmission in the continental United States, CDC issued domestic travel and diagnostic testing guidance for pregnant women and women of reproductive age living in or traveling to an area of Miami-Dade County, Florida (1). On November 28, 2016, local mosquito-borne transmission of Zika virus was

reported in Brownsville (Cameron County), Texas, and on December 14, 2016, CDC issued guidance for travel and testing of pregnant women and women of reproductive age living in or traveling to Brownsville (2). CDC has continued to collaborate closely with state and local jurisdictions to determine when to issue, revise, or lift domestic travel guidance on the basis of epidemiologic evidence (3–5).

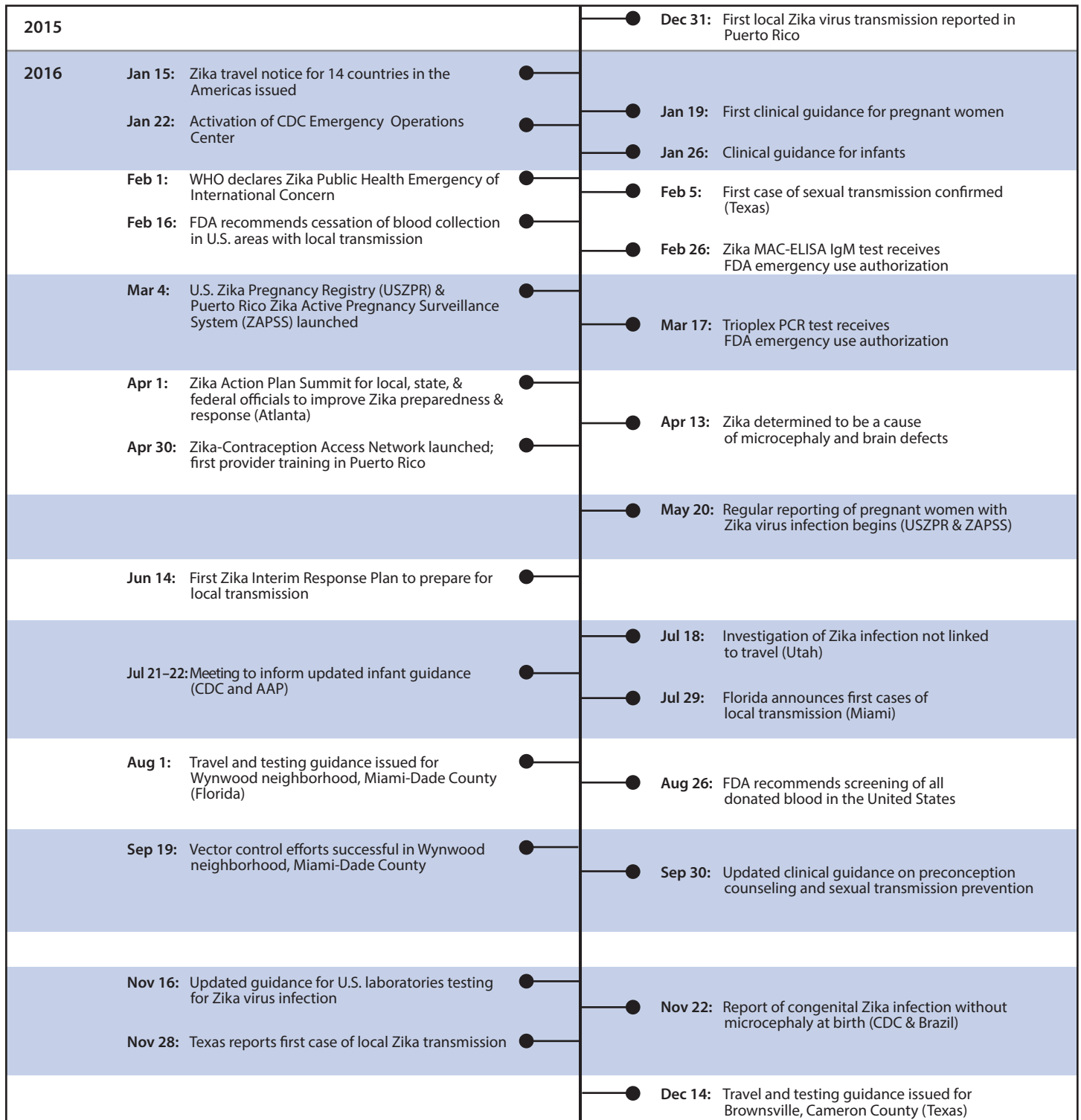
2. Publishing Clinical Guidance for the Care of Pregnant Women and Infants

As a newly recognized congenital infection, Zika virus presents unique challenges for obstetric and pediatric health care providers. CDC's first Zika-related clinical guidance outlining evaluation, testing, and clinical management of Zika virus in pregnant women was released on January 19, 2016 (6), and on January 26, 2016, guidance for the evaluation and testing of infants with possible congenital Zika virus infection was released (7). As new evidence emerged, CDC updated pregnancy and infant guidance and developed guidance for reproductive-aged women (8–13). These evidence-based recommendations have been disseminated to health care providers through partnerships with professional organizations, including the American College of Obstetricians and Gynecologists and the American Academy of Pediatrics, and have provided clear guidance for providers monitoring and caring for pregnant women and fetuses and infants affected by Zika virus infection.

3. Identifying Sexual Transmission of Zika Virus Infection

In late January, CDC, in collaboration with Texas health officials, confirmed sexual contact as the source of Zika virus infection in a Dallas man whose partner had traveled to Brazil (14) and issued guidance for the prevention of sexual transmission of Zika virus in February (15). To date, 38 cases of sexually transmitted Zika virus infection have been confirmed in the United States (16). Most cases have involved transmission from symptomatic men to women (17); however, cases of male-to-male (14), female-to-male (18), and asymptomatic male-to-female (19) sexual transmission have also been documented. In April and May, CDC initiated two studies to determine the frequency and duration of Zika virus shedding

FIGURE 1. Timeline of Zika virus response events, by month — worldwide, January–December 2016



Abbreviations: AAP = American Academy of Pediatrics; FDA = Food and Drug Administration; MAC-ELISA = immunoglobulin M-capture enzyme-linked immunosorbent assay; PCR = polymerase chain reaction; WHO = World Health Organization.

in the semen and urine of infected men. CDC continues to work closely with state, local, and territorial health officials to identify and investigate possible cases of sexual transmission of Zika virus. As new information regarding sexual transmission emerges, one recommendation remains consistent: men who live in or have traveled to an area with active Zika virus transmission should prevent sexual transmission to their pregnant partners by abstaining from sex or consistently and correctly using condoms for the duration of their partner's pregnancy (10,12,13,15).

4. Monitoring Blood Safety and Availability

Because of known transfusion-transmission risks associated with other flaviviruses, Zika virus was recognized as a potential threat to blood safety. The Food and Drug Administration (FDA) and CDC collaborated to recommend travel and risk factor–related deferrals for all U.S. blood donors; in February 2016, FDA issued guidance recommending that, until laboratory screening of blood donations or pathogen-reduction technology could be implemented, blood centers in areas with active mosquito-borne Zika virus transmission cease local blood collection and import blood from U.S. areas without active transmission (20). Because of ongoing local transmission of Zika virus in Puerto Rico and unavailability of either screening or pathogen-reduction technology for all blood products, CDC, in collaboration with the Puerto Rico Department of Health, conducted a rapid assessment of blood collection and use on the island to help guide blood importation measures (21). Blood importation, supported by the Biomedical Advanced Research and Development Authority, continued for Puerto Rico until April 2016, when Zika virus screening of blood donations was implemented under an FDA-approved investigational new drug application (21–23). Based on increasing reports of persons infected through travel as well as local transmission, FDA expanded its blood screening recommendations in August 2016 to include all areas of the United States (24). As of December 10, 2016, products from 78 donations in the continental United States and Hawaii and 353 donations in Puerto Rico have been prevented from entering the blood supply as a result of screening.

5. Developing and Distributing Laboratory Test Kits and Reagents

Working with FDA, CDC obtained the first two emergency use authorizations for CDC-developed in vitro tests to diagnose Zika virus infection: the Zika immunoglobulin M capture enzyme-linked immunosorbent assay (MAC-ELISA) on February 26, 2016 and the Triplex real-time reverse transcription–polymerase chain reaction assay for the detection

and differentiation of RNA from dengue, chikungunya, and Zika viruses on March 17, 2016. CDC manufactured and conducted quality control testing of reagents required for both tests and distributed them domestically and to approximately 100 countries (Figure 2). CDC continues to provide guidance to laboratories on all aspects of testing and interpretation of test results for all Zika virus emergency use authorization tests (25). CDC has continued to work to expand Zika immunoglobulin M testing capacity by entering into material transfer agreements and biologic material licensing agreements with commercial laboratories.

6. Establishing a Causal Link Between Zika Virus Infection During Pregnancy and Serious Brain Abnormalities, Including Microcephaly

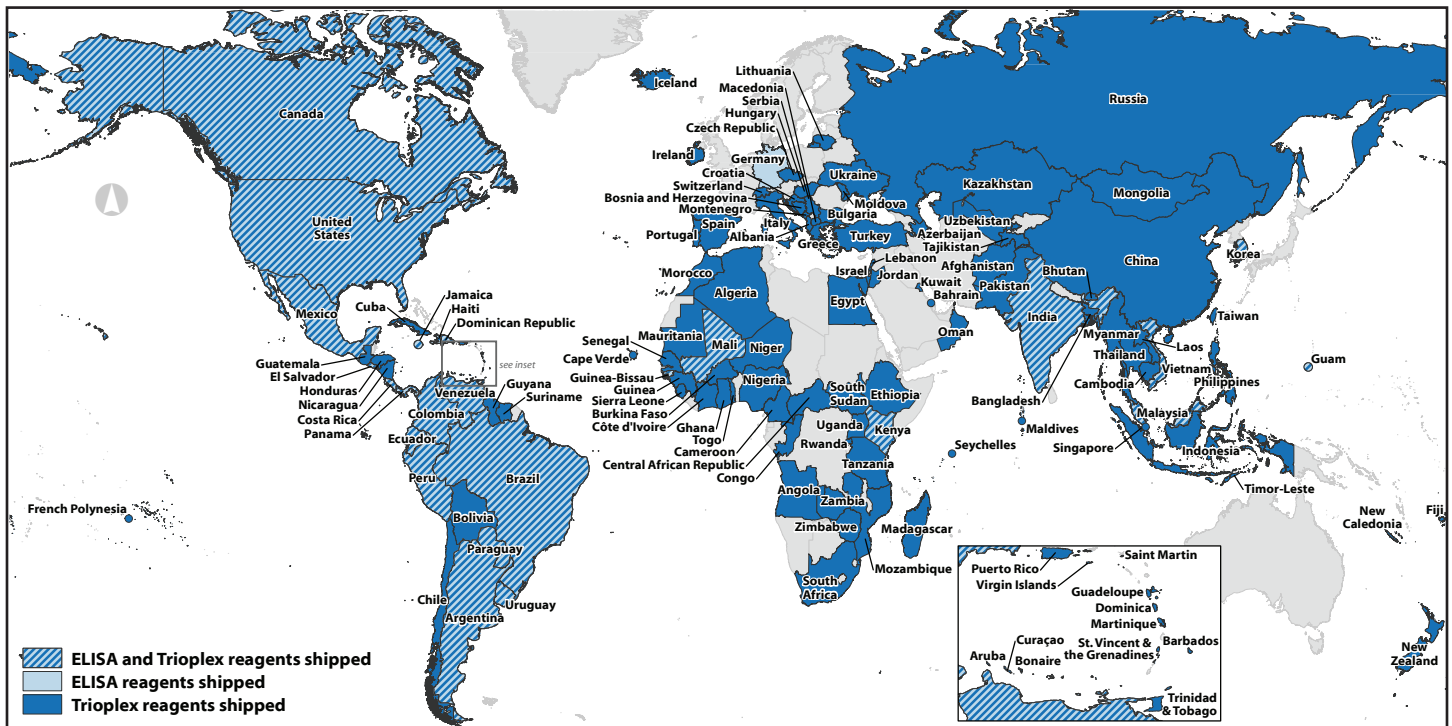
In collaboration with colleagues from Brazil, CDC pathology experts identified the first evidence of Zika virus infection in the fetal brain and in placental tissues, providing evidence of the possible role of Zika virus infection during pregnancy in adverse fetal and infant outcomes (26,27). In April 2016, CDC authors published a comprehensive analysis of the data, concluding that sufficient evidence existed to support a causal relationship between Zika virus infection during pregnancy and microcephaly and other brain abnormalities (28). As of December 15, 2016, 29 countries and territories have reported potential cases of congenital Zika syndrome.*

7. Gathering and Analyzing Zika Pregnancy Surveillance Data to Understand the Magnitude of the Risk and the Full Range of Fetal and Infant Outcomes

To monitor the effect of Zika virus infection during pregnancy, pregnancy and infant surveillance was put in place in U.S. states and territories (29). The U.S. Zika Pregnancy Registry was established in coordination with state, local, tribal, and territorial health departments to monitor all states and territories except Puerto Rico. In Puerto Rico, the Zika Active Pregnancy Surveillance System was established to address specific needs resulting from the anticipated large outbreak (30). CDC also collaborated with the *Instituto Nacional de Salud* (National Institute of Health) in Colombia to conduct enhanced surveillance of pregnant women with symptomatic Zika virus disease in three cities (31). These surveillance systems continue to provide information about the magnitude of risk, the gestational timing of highest risk, and the spectrum of congenital Zika syndrome. Data reported to the U.S. Zika Pregnancy Registry from the continental United States and

* <http://apps.who.int/iris/bitstream/10665/252533/1/zikasitrep15Dec2016-eng.pdf?ua=1>.

FIGURE 2. Distribution of reagents for CDC Zika diagnostic tests* for use under an emergency use authorization as of December 6, 2016



* Enzyme-linked immunosorbent assay (ELISA) and Triplex real-time reverse transcription–polymerase chain reaction (Triplex).

Hawaii suggest that among pregnant women with laboratory evidence of possible Zika virus infection, approximately 6% of fetuses or infants have a birth defect potentially related to Zika virus, and among women with first-trimester Zika infection, 11% of fetuses or infants have evidence of Zika-associated birth defects (32). The proportion of completed pregnancies affected by birth defects was similar following either symptomatic or asymptomatic infection during pregnancy (32). This estimate is consistent with models based on the outbreak in Bahia, Brazil, which estimated a 1%–13% risk for microcephaly after a Zika virus infection during the first trimester (33).

8. Improving Access to the Full Range of Voluntary, Reversible Contraception Methods to Decrease Unintended Pregnancies as a Strategy to Reduce the Impact of Zika Virus Infection

Prevention of unintended pregnancies is a primary strategy to reduce births of infants with Zika-related birth defects. A review of contraception use in Puerto Rico demonstrated limited supply, few trained providers, a cumbersome referral process, and limited provider reimbursement (34). The CDC Foundation, in collaboration with local partners and CDC, established the Zika Contraceptive Access Network (Z-CAN), with the aim of building a network of providers trained in contraception counseling and provision, securing sufficient contraceptive products to meet the needs of women in Puerto Rico, and

raising awareness about the role of contraception in the context of Zika. By the end of 2016, among the 150 physicians actively providing obstetrical services in Puerto Rico, 105 (70%) had been trained and mentored on provision of all reversible methods of contraception. After approximately 3,000 initial Z-CAN visits, 96% of patients have received same-day contraceptive services, and 64% have chosen a long acting reversible contraceptive method. On August 2, 2016, CDC published a review of contraception use among women of reproductive age at risk for unintended pregnancy in states at potential risk for local Zika transmission; the review identified barriers to the use of highly effective contraception and described key strategies states can implement to increase access to contraception during periods of local Zika virus transmission (35).

9. Implementing Vector Control Strategies and Building the Evidence Base for Best Practices

Successful control of *Aedes aegypti*, the primary mosquito vector of Zika virus, has proven extremely difficult using existing control methods. CDC's technical assistance during the Zika response has therefore included support for improved mosquito control infrastructure, novel mosquito control techniques, and integrated vector management that uses existing control methods. During the Zika virus outbreak in the Wynwood neighborhood of Miami-Dade County, Florida, aggressive ground-based integrated vector management was

supplemented by sequential aerial applications of adulticide and larvicide, which rapidly reduced adult mosquito counts in surveillance traps by approximately 90% and helped to end this local outbreak (36). A similar approach in Miami Beach, Florida, using aerial applications of adulticide and ground-based applications of larvicide, also substantially reduced adult mosquito counts. Recent advances in aerial insecticide application methods, and the fact that, in the continental United States, *Aedes aegypti* lives primarily outdoors, likely contributed to the success of the aerial approach in Miami-Dade County. Public opposition to aerial pesticide application in Puerto Rico precluded a similar approach there; instead, lethal mosquito traps have been deployed as part of large community trials that aim to evaluate this method of preventing future outbreaks of mosquito-borne disease on the island (37).

10. Improving Understanding of the Link Between Guillain-Barré Syndrome and Zika Virus Infection

Many countries have reported increases in the occurrence of severe neurologic illness, particularly Guillain-Barré syndrome (GBS), after Zika virus outbreaks, with reported rates two to 10 times higher than those reported before Zika virus disease outbreaks (38–40). During the past year, the Puerto Rico Department of Health and CDC established an enhanced surveillance system for GBS in Puerto Rico. Initial analyses have demonstrated that among 56 patients with suspected GBS during January 1–July 31, 2016, a total of 26 (47%) had confirmed (n = 10, 18%) or presumptive (16, 29%) Zika virus infection (41). Other work related to GBS includes retrospective case-control investigations in Puerto Rico, Brazil, and Colombia, which will help improve understanding of the association between Zika virus infection and GBS.

Future Priorities

Zika virus remains a serious threat to world health that is likely to continue until a safe and effective vaccine becomes available and is widely implemented. Threats from mosquito-borne infection are likely to continue until better vector control interventions are developed. The severe consequences of Zika virus infection require a long-term approach with dedicated resources (42). Important future priorities include continuing to protect pregnant women and fetuses and infants from Zika virus infection; developing improved diagnostics, including the ability to distinguish among flaviviruses serologically; collaborating among government and private partners to accelerate vaccine development; developing and implementing improved vector surveillance and control strategies and capacities; improving contraceptive access to reduce unintended

Summary

What is already known about this topic?

The introduction of Zika virus into the Region of the Americas and the subsequent increase in cases of congenital microcephaly resulted in activation of CDC's Emergency Operations Center and the declaration of a Public Health Emergency of International Concern by the World Health Organization. As of December 15, 2016, 61 countries and territories have reported local Zika virus transmission as part of the current outbreak; 29 countries and territories have reported potential cases of congenital Zika syndrome.

What is added by this report?

CDC's emergency response to Zika virus rapidly addressed many acute public health needs associated with the outbreak and developed new public health surveillance and infection control tools, including issuing travel and clinical guidance; identifying sexual transmission; monitoring blood safety; developing and distributing laboratory test kits; establishing the causal link between in utero infection and congenital Zika syndrome and assessing the range of outcomes and the magnitude of risk; improving access to contraception to prevent unintended pregnancies; implementing vector control strategies; and improving the understanding of the link between and Zika virus infection and other neurological illnesses.

What are the implications for public health practice?

To protect pregnant women and their fetuses and infants from the effects of Zika virus infection during pregnancy, public health activities must focus on preventing mosquito-borne transmission through vector control and personal protective practices, preventing sexual transmission by advising abstinence from sex or consistent and correct use of condoms, and preventing unintended pregnancies by reducing barriers to access to highly effective reversible contraception. Collectively, these critical strategies can reduce the effect of the virus on infants, families, and communities.

pregnancies; and improving understanding of the long-term outcomes for infants exposed to Zika virus in utero. Much remains to be done to protect pregnant women and fetuses and infants from Zika virus infection; the rapid action, dedication, and collaboration demonstrated by the global public health community during the past year provide a solid foundation for future work.

¹CDC.

Corresponding author: Corresponding author: Satish K. Pillai, eoevent236@cdc.gov.

References

1. CDC. CDC guidance for travel and testing of pregnant women and women of reproductive age for Zika virus infection related to the investigation for local mosquito-borne Zika virus transmission in Miami-Dade and Broward Counties, Florida. Atlanta, GA: US Department of Health and Human Services, CDC; 2016. <https://emergency.cdc.gov/han/han00393.asp>

2. CDC. CDC guidance for travel and testing of pregnant women and women of reproductive age for Zika virus infection related to the investigation for local mosquito-borne Zika virus transmission in Brownsville, Cameron County, Texas. Atlanta, GA: US Department of Health and Human Services, CDC; 2016. <https://emergency.cdc.gov/han/han00399.asp>
3. CDC. CDC expands guidance for travel and testing of pregnant women, women of reproductive age, and their partners for Zika virus infection related to mosquito-borne Zika virus transmission in Miami-Dade, Florida. Atlanta, GA: US Department of Health and Human Services, CDC; 2016. <https://emergency.cdc.gov/han/han00394.asp>
4. CDC. CDC updates guidance for travel and testing of pregnant women and women of reproductive age for Zika virus infection related to the ongoing investigation of local mosquito-borne Zika virus transmission in Miami-Dade County, Florida. Atlanta, GA: US Department of Health and Human Services, CDC; 2016. <https://emergency.cdc.gov/han/han00396.asp>
5. CDC. CDC updates guidance for pregnant women and women and men of reproductive age for Zika virus infection related to the ongoing investigation of local mosquito-borne Zika virus transmission in Miami-Dade County, Florida. Atlanta, GA: US Department of Health and Human Services, CDC; 2016. <https://emergency.cdc.gov/han/han00398.asp>
6. Petersen EE, Staples JE, Meaney-Delman D, et al. Interim guidelines for pregnant women during a Zika virus outbreak—United States, 2016. *MMWR Morb Mortal Wkly Rep* 2016;65:30–3. <http://dx.doi.org/10.15585/mmwr.mm6502e1>
7. Staples JE, Dziuban EJ, Fischer M, et al. Interim guidelines for the evaluation and testing of infants with possible congenital Zika virus infection—United States, 2016. *MMWR Morb Mortal Wkly Rep* 2016;65:63–7. <http://dx.doi.org/10.15585/mmwr.mm6503e3>
8. Oduyebo T, Petersen EE, Rasmussen SA, et al. Update: interim guidelines for health care providers caring for pregnant women and women of reproductive age with possible Zika virus exposure—United States, 2016. *MMWR Morb Mortal Wkly Rep* 2016;65:122–7. <http://dx.doi.org/10.15585/mmwr.mm6505e2>
9. Petersen EE, Polen KN, Meaney-Delman D, et al. Update: interim guidance for health care providers caring for women of reproductive age with possible Zika virus exposure—United States, 2016. *MMWR Morb Mortal Wkly Rep* 2016;65:315–22. <http://dx.doi.org/10.15585/mmwr.mm6512e2>
10. Oster AM, Russell K, Stryker JE, et al. Update: interim guidance for prevention of sexual transmission of Zika virus—United States, 2016. *MMWR Morb Mortal Wkly Rep* 2016;65:323–5. <http://dx.doi.org/10.15585/mmwr.mm6512e3>
11. Oduyebo T, Igbinoza I, Petersen EE, et al. Update: interim guidance for health care providers caring for pregnant women with possible Zika virus exposure—United States, July 2016. *MMWR Morb Mortal Wkly Rep* 2016;65:739–44. <http://dx.doi.org/10.15585/mmwr.mm6529e1>
12. Brooks JT, Friedman A, Kachur RE, LaFlam M, Peters PJ, Jamieson DJ. Update: interim guidance for prevention of sexual transmission of Zika virus—United States, July 2016. *MMWR Morb Mortal Wkly Rep* 2016;65:745–7. <http://dx.doi.org/10.15585/mmwr.mm6529e2>
13. Petersen EE, Meaney-Delman D, Neblett-Fanfair R, et al. Update: interim guidance for preconception counseling and prevention of sexual transmission of Zika virus for persons with possible Zika virus exposure—United States, September 2016. *MMWR Morb Mortal Wkly Rep* 2016;65:1077–81. <http://dx.doi.org/10.15585/mmwr.mm6539e1>
14. Deckard DT, Chung WM, Brooks JT, et al. Male-to-male sexual transmission of Zika virus—Texas, January 2016. *MMWR Morb Mortal Wkly Rep* 2016;65:372–4. <http://dx.doi.org/10.15585/mmwr.mm6514a3>
15. Oster AM, Brooks JT, Stryker JE, et al. Interim guidelines for prevention of sexual transmission of Zika virus—United States, 2016. *MMWR Morb Mortal Wkly Rep* 2016;65:120–1. <http://dx.doi.org/10.15585/mmwr.mm6505e1>
16. CDC. Zika virus: case counts in the US. Atlanta, GA: US Department of Health and Human Services, CDC; 2016. <https://www.cdc.gov/zika/geo/united-states.html>
17. Hills SL, Russell K, Hennessey M, et al. Transmission of Zika virus through sexual contact with travelers to areas of ongoing transmission—continental United States, 2016. *MMWR Morb Mortal Wkly Rep* 2016;65:215–6. <http://dx.doi.org/10.15585/mmwr.mm6508e2>
18. Davidson A, Slavinski S, Komoto K, Rakeman J, Weiss D. Suspected female-to-male sexual transmission of Zika virus—New York City, 2016. *MMWR Morb Mortal Wkly Rep* 2016;65:716–7. <http://dx.doi.org/10.15585/mmwr.mm6528e2>
19. Brooks RB, Carlos MP, Myers RA, et al. Likely sexual transmission of Zika virus from a man with no symptoms of infection—Maryland, 2016. *MMWR Morb Mortal Wkly Rep* 2016;65:915–6. <http://dx.doi.org/10.15585/mmwr.mm6534e2>
20. Food and Drug Administration. Recommendations for donor screening, deferral, and product management to reduce the risk of transfusion-transmission of Zika virus. Silver Spring, MD: US Department of Health and Human Services, Food and Drug Administration; 2016. <http://www.fda.gov/downloads/BiologicsBloodVaccines/GuidanceComplianceRegulatoryInformation/Guidances/Blood/UCM486360.pdf>
21. Vasquez AM, Sapiano MR, Basavaraju SV, Kuehnert MJ, Rivera-Garcia B. Survey of blood collection centers and implementation of guidance for prevention of transfusion-transmitted Zika virus infection—Puerto Rico, 2016. *MMWR Morb Mortal Wkly Rep* 2016;65:375–8. <http://dx.doi.org/10.15585/mmwr.mm6514e1>
22. Food and Drug Administration. FDA allows use of investigational test to screen blood donations for Zika virus. Silver Spring, MD: US Department of Health and Human Services, Food and Drug Administration; 2016. <http://www.fda.gov/NewsEvents/Newsroom/PressAnnouncements/ucm493081.htm>
23. Kuehnert MJ, Basavaraju SV, Moseley RR, et al. Screening of blood donations for Zika virus infection—Puerto Rico, April 3–June 11, 2016. *MMWR Morb Mortal Wkly Rep* 2016;65:627–8. <http://dx.doi.org/10.15585/mmwr.mm6524e2>
24. Food and Drug Administration. Revised recommendations for reducing the risk of Zika virus transmission by blood and blood components. Silver Spring, MD: US Department of Health and Human Services, Food and Drug Administration; 2016. <http://www.fda.gov/downloads/BiologicsBloodVaccines/GuidanceComplianceRegulatoryInformation/Guidances/Blood/UCM518213.pdf>
25. CDC. Guidance for U.S. laboratories testing for Zika virus infection. Atlanta, GA: US Department of Health and Human Services, CDC; 2016. <https://www.cdc.gov/zika/pdfs/laboratory-guidance-zika.pdf>
26. Martines RB, Bhatnagar J, de Oliveira Ramos AM, et al. Pathology of congenital Zika syndrome in Brazil: a case series. *Lancet* 2016;388:898–904. [http://dx.doi.org/10.1016/S0140-6736\(16\)30883-2](http://dx.doi.org/10.1016/S0140-6736(16)30883-2)
27. Martines RB, Bhatnagar J, Keating MK, et al. Notes from the field: evidence of Zika virus infection in brain and placental tissues from two congenitally infected newborns and two fetal losses—Brazil, 2015. *MMWR Morb Mortal Wkly Rep* 2016;65:159–60. <http://dx.doi.org/10.15585/mmwr.mm6506e1>
28. Rasmussen SA, Jamieson DJ, Honein MA, Petersen LR. Zika virus and birth defects—reviewing the evidence for causality. *N Engl J Med* 2016;374:1981–7. <http://dx.doi.org/10.1056/NEJMs1604338>
29. Honein MA, Jamieson DJ. Monitoring and preventing congenital Zika syndrome. *N Engl J Med* 2016;375:2393–4. <http://dx.doi.org/10.1056/NEJMe1613368>

30. Ellington SR, Devine O, Bertolli J, et al. Estimating the number of pregnant women infected with Zika virus and expected infants with microcephaly following the Zika virus outbreak in Puerto Rico, 2016. *JAMA Pediatr* 2016;170:940–5. <http://dx.doi.org/10.1001/jamapediatrics.2016.2974>
31. Cuevas EL, Tong VT, Rozo N, et al. Preliminary report of microcephaly potentially associated with Zika virus infection during pregnancy—Colombia, February–November 2016. *MMWR Morb Mortal Wkly Rep* 2016;65:1409–13. <http://dx.doi.org/10.15585/mmwr.mm6549e1>
32. Honein MA, Dawson A, Petersen EE, et al. Birth defects among fetuses and infants of US women with evidence of possible Zika virus infection during pregnancy. *JAMA Pediatr* 2016. E-pub December 13, 2016.
33. Johansson MA, Mier-y-Teran-Romero L, Reefhuis J, Gilboa SM, Hills SL. Zika and the risk of microcephaly. *N Engl J Med* 2016;375:1–4. <http://dx.doi.org/10.1056/NEJMp1605367>
34. Tepper NK, Goldberg HI, Bernal MI, et al. Estimating contraceptive needs and increasing access to contraception in response to the Zika virus disease outbreak—Puerto Rico, 2016. *MMWR Morb Mortal Wkly Rep* 2016;65:311–4. <http://dx.doi.org/10.15585/mmwr.mm6512e1>
35. Boulet SL, D’Angelo DV, Morrow B, et al. Contraceptive use among nonpregnant and postpartum women at risk for unintended pregnancy, and female high school students, in the context of Zika preparedness—United States, 2011–2013 and 2015. *MMWR Morb Mortal Wkly Rep* 2016;65:780–7. <http://dx.doi.org/10.15585/mmwr.mm6530e2>
36. Likos A, Griffin I, Bingham AM, et al. Local mosquito-borne transmission of Zika virus—Miami-Dade and Broward counties, Florida, June–August 2016. *MMWR Morb Mortal Wkly Rep* 2016;65:1032–8. <http://dx.doi.org/10.15585/mmwr.mm6538e1>
37. Lorenzi OD, Major C, Acevedo V, et al. Reduced Incidence of chikungunya virus infection in communities with ongoing *Aedes Aegypti* mosquito trap intervention studies—Salinas and Guayama, Puerto Rico, November 2015–February 2016. *MMWR Morb Mortal Wkly Rep* 2016;65:479–80. <http://dx.doi.org/10.15585/mmwr.mm6518e3>
38. Cao-Lormeau VM, Blake A, Mons S, et al. Guillain-Barré Syndrome outbreak associated with Zika virus infection in French Polynesia: a case-control study. *Lancet* 2016;387:1531–9. [http://dx.doi.org/10.1016/S0140-6736\(16\)00562-6](http://dx.doi.org/10.1016/S0140-6736(16)00562-6)
39. dos Santos T, Rodriguez A, Almiron M, et al. Zika virus and the Guillain-Barré syndrome—case series from seven countries. *N Engl J Med* 2016;375:1598–601. <http://dx.doi.org/10.1056/NEJMc1609015>
40. Petersen LR, Jamieson DJ, Powers AM, Honein MA. Zika Virus. *N Engl J Med* 2016;374:1552–63. <http://dx.doi.org/10.1056/NEJMra1602113>
41. Dirlikov E, Major CG, Mayshack M, et al. Guillain-Barré syndrome during ongoing Zika virus transmission—Puerto Rico, January 1–July 31, 2016. *MMWR Morb Mortal Wkly Rep* 2016;65:910–4. <http://dx.doi.org/10.15585/mmwr.mm6534e1>
42. World Health Organization. Statement on the 5th Meeting of the Emergency Committee under the International Health Regulations (2005) regarding microcephaly, other neurological disorders and Zika virus. Geneva, Switzerland: World Health Organization; 2016. <http://www.who.int/mediacentre/news/statements/2016/zika-fifth-ec/en/>

Notes from the Field

Compliance with Postexposure Prophylaxis for Exposure to *Bacillus anthracis* Among U.S. Military Personnel — South Korea, May 2015

Koya C. Allen, PhD^{1,2}; Katherine Hendricks, MD³;
Eric Sergienko, MD⁴; Raul Mirza, DO⁵; Rohit A. Chitale, PhD^{1,6}

In the United States, *Bacillus anthracis* is a select agent and is subject to select agent requirements under the U.S. Code of Federal Regulations.* On April 20, 2015, samples of *B. anthracis* spores considered inactivated were shipped from a U.S. Department of Defense (DoD) laboratory at Dugway Proving Ground, Utah, to various laboratories for routine collaborative diagnostics research. On May 22, 2015, CDC was notified of live *B. anthracis* in one sample received by a private company and initiated a response. On May 29, 2015, DoD began reviewing safety practices for generating and handling inactivated *B. anthracis* spores. By June 1, 2015, the Office of the Assistant Secretary of Defense for Nuclear, Chemical, and Biological Defense Programs had established a task force to coordinate the DoD response (1).

The DoD Comprehensive Anthrax Laboratory Review (2) was completed within 30 days and addressed five main objectives: 1) conduct root cause analysis for incomplete inactivation of *B. anthracis*; 2) investigate the lack of effective postinactivation sterility testing for detection of live *B. anthracis*; 3) review DoD laboratory biohazard safety procedures/protocols; 4) determine laboratory adherence to established procedures/protocols; and 5) identify systemic problems and corresponding solutions. The DoD investigation identified 194 commercial companies, academic institutions, and federal laboratories that had received potentially live *B. anthracis* samples across 50 states, the District of Columbia, three U.S. territories, and nine foreign countries.

In South Korea, the Joint U.S. Forces Korea Portal and Integrated Threat Recognition program works on detection of biologic agents in the environment. A sample of *B. anthracis* was sent to Osan Air Base from the Dugway Proving Ground shipment for research, and 22 DoD personnel were exposed to the sample. Immediately after the event was discovered, these personnel were assessed for the need for emergency postexposure prophylaxis (PEP). On May 27, 2015, all 22 potentially exposed personnel began a PEP regimen tailored to their

individual vaccination history. Persons lacking prior anthrax vaccination or with expired vaccination history received the standard emergency use protocol for PEP: 3 anthrax vaccine doses over 4 weeks plus 60 days of oral ciprofloxacin (500 mg twice a day) or doxycycline (100 mg twice a day) (3,4). Persons current for *B. anthracis* vaccination received emergency PEP: 30 days of oral ciprofloxacin or doxycycline (3,4) (Table).

The cohort of exposed personnel was monitored by the Armed Forces Health Surveillance Center, in collaboration with CDC and Army Public Health Center. Cases were monitored for adherence with PEP regimens and onset of symptoms consistent with exposure. No clinical anthrax cases were associated with this incident. Of the 22 persons exposed in South Korea, 14 (63.6%) who lacked prior anthrax vaccination or had expired vaccination received anthrax vaccine and a 60-day schedule of ciprofloxacin or doxycycline; all 14 completed antibiotics, and 13 of the 14 completed all anthrax vaccine doses. Eight persons who were current for *B. anthracis* vaccination had 30-day antibiotic schedules, with 100% completing their PEP. No adverse events to vaccination or antibiotics were reported; one pregnant woman was medically advised to transition from ciprofloxacin to amoxicillin.

This unintentional incident that resulted in no clinical cases highlights the importance of vigilance in preparedness and response capabilities for biologic events. Surveillance of potentially exposed military personnel demonstrated near 100% adherence to required PEP. Although challenging because of the mobility of this unique population, the swift DoD response ensured control of the population at risk, minimized risk for disease, and demonstrated that a high rate of compliance is achievable in closely monitored otherwise healthy persons.

TABLE. Completion of postexposure prophylaxis (PEP) by U.S. military personnel potentially exposed to anthrax (N=22), by anthrax vaccination history and military service branch — South Korea, May 2015

Service	Total no.	PEP among those current for anthrax vaccination No. (%)	PEP among those not current for anthrax vaccination No. (%)	Total completing PEP No. (%)
Army	16	3 (19)	13 (81)	15* (94)
Navy	1	0 (0)	1 (100)	1 (100)
Air Force	5	5 (100)	0 (0)	5 (100)
Total	22	8 (100)	14 (100)	21* (95)

* One person completed the antibiotic series but did not complete all anthrax vaccine doses.

* http://www.ecfr.gov/cgi-bin/text-idx?SID=f4edcf593150dda1ca3154c98de05e9e&mc=true&node=se42.1.73_13&rtn=div8.

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¹Integrated Biosurveillance Section, Armed Forces Health Surveillance Branch, Public Health Division, Defense Health Agency, Falls Church, Virginia; ²Counter Biothreats Cell, Force Health Protection, J4 Logistics Directorate, U.S. European Command Headquarters, U.S. Department of Defense; ³Division of High-Consequence Pathogens and Pathology, National Center for Emerging and Zoonotic Infectious Diseases, CDC; ⁴Liaison to CDC, U.S. Department of Defense; ⁵Occupational Medicine, Army Public Health Center, U.S. Department of Defense; ⁶Division of Global HIV and Tuberculosis, CDC.

Corresponding author: Koya C. Allen, koya.c.allen.ctr@mail.mil, 49-0711-680-5648.

References

1. Committee for Comprehensive Review of DoD Laboratory Procedures, Processes, and Protocols Associated with Inactivating *Bacillus anthracis* Spores. Review Committee report: inadvertent shipment of live *Bacillus anthracis* spores by DoD. Washington, DC: U.S. Department of Defense; 2015. https://www.defense.gov/Portals/1/features/2015/0615_lab-stats/Review-Committee-Report-Final.pdf
2. US Department of Defense. Laboratory review. Washington, DC: US Department of Defense; 2015. <https://www.defense.gov/News/Special-Reports/DoD-Laboratory-Review>
3. Wright JG, Quinn CP, Shadomy S, Messonnier N; Centers for Disease Control and Prevention (CDC). Use of anthrax vaccine in the United States: recommendations of the Advisory Committee on Immunization Practices (ACIP), 2009. *MMWR Recomm Rep* 2010;59(No. RR-06).
4. US Army Medical Research Institute of Infectious Diseases (USAMRIID). Medical management of biological casualties handbook. 8th ed. Frederick, Maryland: US Army Medical Research Institute of Infectious Diseases; 2014.

Notes from the Field

Botulism Outbreak from Drinking Prison-Made Illicit Alcohol in a Federal Correctional Facility — Mississippi, June 2016

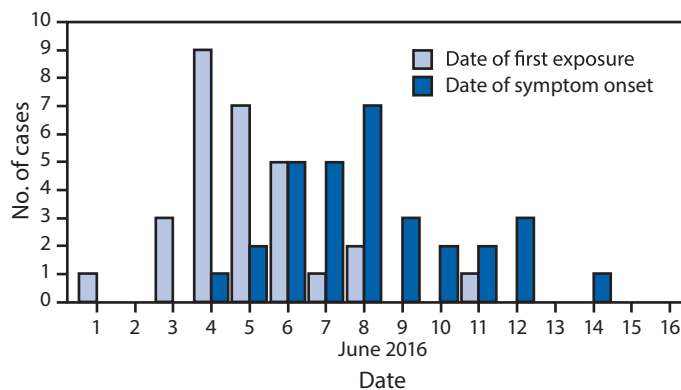
Lindsey McCrickard, DVM^{1,2}; Mariel Marlow, PhD^{1,2}; Julie L. Self, PhD^{1,2}; Louise Francois-Watkins, MD²; Kevin Chatham-Stephens, MD²; Jannifer Anderson³; Sheryl Hand³; Kathryn Taylor, MD³; Jennifer Hanson³; Keiundria Patrick³; Carolina Luquez, PhD²; Janet Dykes, MS²; Suzanne R. Kalb, PhD⁴; Kaitlin Hoyt⁴; John R. Barr, PhD⁴; Todd Crawford, MSN²; Anthony Chambers, MD²; Brian Douthit⁵; Robert Cox, MD⁶; Matt Craig, MD⁶; John Spurzem, MD⁶; Joseph Doherty, MD⁶; Michael Allswede, DO⁷; Paul Byers, MD³; Thomas Dobbs, MD³

On June 9, 2016, the Mississippi Poison Control Center and the Mississippi State Department of Health (MSDH) notified CDC of five suspected cases of botulism, a potentially fatal neuroparalytic illness (*I*), in inmates at a medium-security federal correctional institution (prison A). By June 10, a total of 13 inmates were hospitalized, including 12 in Mississippi and one in Oklahoma (the inmate in Oklahoma had been transferred there after his exposure for reasons unrelated to his illness). MSDH, Oklahoma State Department of Health, Bureau of Prisons, and CDC conducted an investigation to identify the source and scope of the outbreak, and to develop recommendations.

Prison A staff members suspected that an alcoholic beverage, illicitly made by inmates and known as “hooch” or “pruno,” was the source of the outbreak. Among 33 inmates who reported consuming hooch during June 1–19, 2016, a total of 31 (94%) had signs or symptoms suggesting botulism. The median interval from first exposure to symptom onset was 3 days (range = 0–11 days) (Figure). Cases were categorized using modified Council of State and Territorial Epidemiologists definitions. A confirmed case was defined as an illness in an inmate consistent with botulism that began on or after June 1, with botulinum toxin type A detected in a serum or stool specimen or *Clostridium botulinum* cultured from a stool specimen; a probable case was defined as an illness in an inmate with signs or symptoms of any cranial nerve palsy and extremity weakness that began on or after June 1; and a suspected case was an illness in an inmate with signs or symptoms of any cranial nerve palsy without extremity weakness that began on or after June 1.

Thirty-one cases were identified, all in men, including 19 confirmed cases (18 in Mississippi and one in Oklahoma), 10 probable cases (nine in Mississippi and one in Texas), and two suspected cases in Mississippi. Patients from Texas and Oklahoma were transferred from prison A to other prisons before their illness began as part of routine inmate transfers.

FIGURE. Botulism cases (n = 31) in a federal correctional facility, by reported date of hooch exposure* and symptom onset — Mississippi, June 1–19, 2016



* Hooch is defined as an illicitly made alcoholic beverage. First exposure is defined as the first exposure to hooch occurring during June 1–19, 2016. Date of exposure was unknown for two inmates.

Twenty-four (77%) patients were non-Hispanic black, six (19%) were Hispanic white, and one identified as other non-Hispanic (3%). The median age was 36 years (range = 23–47 years). By the end of the outbreak, 24 inmates were hospitalized, including 15 (63%) who were admitted to an intensive care unit and nine (38%) who required intubation and mechanical ventilation; none died. Twenty (83%) patients received botulinum antitoxin; 11 patients with mild illness did not receive antitoxin.

Medical chart abstractions (30) and interviews (30) were conducted for patients in Mississippi (29), Oklahoma (1) and Texas (1) to obtain information on hooch exposure, clinical signs and symptoms, medical management, and patients' understanding of botulism. One patient in Oklahoma was not able to be interviewed, and one patient in Texas did not have a medical chart for abstraction. Among 30 patients interviewed, 27 (90%) had never heard of botulism, and 23 (77%) did not know that drinking hooch could make them sick. Eleven (42%) of 26 patients who responded to questions regarding the frequency of hooch consumption reported drinking hooch at least once a month. Some hooch exposure dates associated with this outbreak coincided with a farewell party for one inmate and the National Basketball Association finals. Although prison A staff members confiscated >20 gallons of hooch during the investigation, the number of circulating batches of hooch immediately before the outbreak, and the ingredients and preparation method of the batch responsible for the outbreak, are unknown. One patient reported that

honey, potatoes, apples, and tomato paste from a bulging can were combined, hidden, and fermented in a sealed plastic bag at room temperature for 3–5 days. Possible sources of *Clostridium botulinum* or toxin include tomato paste, potatoes, other ingredients, or contamination from the environment. Potatoes have been hypothesized as the source of other pruno-associated outbreaks (2).

This botulism outbreak, the largest in the United States since 1978 (3), highlights the clinical spectrum of illness, ranging from total paralysis requiring intensive care and mechanical ventilation to cranial nerve complaints not requiring hospitalization (4). Facility staff members should consider the potential for increased hooch consumption during celebratory events. Educating correctional facility staff members and inmates about the risks of consuming hooch and good communication channels between facility staff members and inmates can help to identify and treat persons with botulism quickly and prevent deaths.

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¹Epidemic Intelligence Service, CDC; ²Division of Foodborne, Waterborne, and Environmental Diseases, National Center for Emerging and Zoonotic Infectious Diseases, CDC; ³Mississippi State Department of Health; ⁴Division of Laboratory Sciences, National Center for Environmental Health, CDC; ⁵Federal Bureau of Prisons, Washington, D.C.; ⁶University of Mississippi Medical Center, Jackson, Mississippi; ⁷Integris Southwest Medical Center, Oklahoma City, Oklahoma.

Corresponding Author: Lindsey McCrickard, Vjf4@cdc.gov 404-718-1607.

References

1. Sobel J. Botulism. *Clin Infect Dis* 2005;41:1167–73. <http://dx.doi.org/10.1086/444507>
2. CDC. Botulism from drinking prison-made illicit alcohol—Utah 2011. *MMWR Morb Mortal Wkly Rep* 2012;61:782–4.
3. CDC. US Public Health Service: botulism—New Mexico. *MMWR Morb Mortal Wkly Rep* 1978;27:138.
4. McCarty CL, Angelo K, Beer KD, et al. Notes from the field: large outbreak of botulism associated with a church potluck meal—Ohio, 2015. *MMWR Morb Mortal Wkly Rep* 2015;64:802–3. <http://dx.doi.org/10.15585/mmwr.mm6429a6>

Notes from the Field

Detection of Sabin-Like Type 2 Poliovirus from Sewage After Global Cessation of Trivalent Oral Poliovirus Vaccine — Hyderabad and Ahmedabad, India, August–September 2016

Sunil Bahl, MD¹; Lee M. Hampton, MD²; Pankaj Bhatnagar, MD³; Gadala Srinivasa Rao, MD⁴; Pradeep Haldar, MBBS⁵; Lucky Sangal, MD³; Puttaraju AK Jetty, MBBS³; Uma P Nalavade, MSc⁶

During September 2–October 4, 2016, four sewage samples collected during August 3–September 19 (Hyderabad, Telangana State, India) and one sewage sample collected on August 30 (Ahmedabad, Gujarat State, India) tested positive for Sabin-like type 2 polioviruses. These polioviruses were detected approximately 4 months after April 25, 2016, when India officially ceased use of trivalent oral poliovirus vaccine (tOPV), containing Sabin attenuated types 1, 2, and 3 polioviruses, and switched to bivalent OPV (bOPV), containing Sabin attenuated types 1 and 3 polioviruses (1).

Detection of Sabin-like type 2 poliovirus approximately 4 months after the switch from tOPV to bOPV suggested that tOPV use might have continued after it was supposed to stop globally, creating a risk for emergence of new type 2 vaccine-derived polioviruses (VDPV2s), which can cause paralysis. Genetic sequencing of the 903-nucleotide VP1 region of the isolated viruses showed zero, one, two, and four nucleotide changes in the four Hyderabad isolates and one nucleotide change in the Ahmedabad isolate, compared with the type 2 polioviruses in tOPV. These findings indicated that the isolated polioviruses had not replicated sufficiently to accumulate more than a few mutations on a potential pathway to becoming VDPV2s, and that the tOPV they originated from had likely been used during the preceding 4 months.

In accordance with global guidelines for responding to poliovirus events (2), detailed investigations were initiated within 48 hours of detection of the type 2 poliovirus in Hyderabad and the neighboring Rangareddy district, and in Ahmedabad (Box). As part of global poliovirus containment efforts (3), laboratories in those areas potentially storing type 2 polioviruses had previously been found to not have such polioviruses, so they were not searched. Telangana and Gujarat state officials met with immunization program stakeholders in the affected districts and other districts in their states regarding the need to reconfirm withdrawal of all tOPV.

In Hyderabad and Rangareddy districts, the two main district vaccine cold stores, along with 13 private vaccine retailers and distributors and 4,498 public and private health facilities,

were searched during September 4–October 5. Thirty-seven tOPV vials from four manufacturers were found in 17 private clinics; the majority were small clinics not affiliated with an organized medical association. Twenty-two of the tOPV vials were unopened; however, 15 had been partially used. Six vials were beyond their expiration date, and 31 had expiration dates from December 2016 to November 2017. No tOPV vials or bulk type 2 polio vaccine were found at the only vaccine manufacturer in Hyderabad.

In Ahmedabad District, the main district vaccine cold store, 572 other cold chain storage points and public and private health facilities, and 12 private vaccine retailers and distributors were searched during September 14–October 17. Two tOPV vials were found at a private vaccine retailer, and another 11 tOPV vials were found at eight private clinics; the majority were small clinics not affiliated with an organized medical association. All tOPV vials had expiration dates ranging from December 2016 to November 2017.

BOX. Components of investigations to find trivalent oral poliovirus vaccine (tOPV) still in use after its use was officially ceased — Hyderabad and Ahmedabad, India

- Conduct immediate search of all known vaccine cold chain storage points.
- Visit all health facilities regularly reporting acute flaccid paralysis cases to inquire about tOPV use, with extensive search for tOPV vials at medical colleges and other large health facilities.
- Conduct a street-by-street physical check of all public and private health facilities that do not regularly report acute flaccid paralysis cases.
- Map and search all private vaccine retailers and distributors in coordination with the state drug regulator.
- Upon identification of any tOPV vials at any location, trace back the source and timing of the supply. Ensure safe disposal of recovered vaccine.
- Visit any OPV manufacturers to check for tOPV and bulk type 2 polio vaccine.
- Meet with immunization program stakeholders (e.g., professional health associations, public and private hospitals, vaccine retailers and distributors, immunization program officers, cold chain officers, and World Health Organization and United Nations Children's Fund staff members) regarding the need to reconfirm tOPV withdrawal.

All tOPV vials found during the investigations had been purchased and delivered before the switch from tOPV to bOPV. All tOPV vials found were removed, labeled for destruction, and placed in the responsible immunization officer's custody.

These investigations for tOPV possibly in use after the global switch from tOPV to bOPV are the first triggered by detection of Sabin-like type 2 polioviruses in either environmental surveillance sewage samples or stool specimens from persons with paralysis. The finding of tOPV vials in health facilities and at a vaccine retailer indicates that some tOPV might still be in use and that future detections of Sabin-like type 2 virus anywhere should prompt checks for tOPV vials, according to the guidelines for responding to type 2 polioviruses (2). The risk that Sabin-like type 2 virus could spread and evolve into a circulating VDPV2 increases over time with the progressive decrease in population immunity to type 2 poliovirus infection following the switch from tOPV to bOPV (4).

The finding that all tOPV discovered was at a private vaccine retailer or private clinics indicates that future investigations to identify tOPV still in use should carefully assess the private sector. This investigation underscores the importance of maintaining robust surveillance for polioviruses and of immunization workers being alert for tOPV vials in cold chain storage and reporting any tOPV vials that they find. Additional efforts are needed to ensure that the private sector is aware of the need for cessation of tOPV use.

¹World Health Organization, South-East Asia Regional Office, New Delhi, India; ²Global Immunization Division, CDC; ³World Health Organization, Country Office for India, New Delhi, India; ⁴Government of Telangana, India; ⁵Ministry of Health and Family Welfare, Government of India, New Delhi, India; ⁶Enterovirus Research Centre, Mumbai, India.

Corresponding author: Lee M. Hampton, lhampton@cdc.gov, 404-639-4722.

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References

1. Hampton LM, Farrell M, Ramirez-Gonzalez A, et al.; Immunization Systems Management Group of the Global Polio Eradication Initiative. Cessation of trivalent oral polio vaccine and introduction of inactivated poliovirus vaccine—worldwide, 2016. *MMWR Morb Mortal Wkly Rep* 2016;65:934–8. <http://dx.doi.org/10.15585/mmwr.mm6535a3>
2. Global Polio Eradication Initiative. Responding to a poliovirus event and outbreak—part 2: protocol for poliovirus type 2. Geneva, Switzerland: World Health Organization, Global Polio Eradication Initiative; 2016. http://polioeradication.org/wp-content/uploads/2016/09/Responding-to-a-poliovirus-event-and-outbreak-SOPs-Part-2-Protocol-for-PV-Type-2_EN.pdf
3. Previsani N, Tangermann RH, Tallis G, Jafari HS. World Health Organization guidelines for containment of poliovirus following type-specific polio eradication—worldwide, 2015. *MMWR Morb Mortal Wkly Rep* 2015;64:913–7. <http://dx.doi.org/10.15585/mmwr.mm6433a5>
4. Duintjer Tebbens RJ, Hampton LM, Thompson KM. Implementation of coordinated global serotype 2 oral poliovirus vaccine cessation: risks of inadvertent trivalent oral poliovirus vaccine use. *BMC Infect Dis* 2016;16:237. <http://dx.doi.org/10.1186/s12879-016-1537-8>

Announcement

National Birth Defects Prevention Month and Folic Acid Awareness Week — January 2017

The Zika virus disease outbreak has led to renewed focus on how some birth defects are caused by infection during pregnancy. “Prevent Infections for Baby’s Protection” is the theme of January 2017’s National Birth Defects Prevention Month. Birth defects are common, costly, and critical, and they affect one in 33 U.S. babies annually (1). Not all birth defects can be prevented, but women can increase their chances of having a healthy baby by reducing their risk for getting an infection during pregnancy.

Women can take the following steps to prevent infections: talk to their health care provider about how they can reduce their risk for infections with viruses such as Zika and congenital syphilis, if they are pregnant or currently planning a pregnancy (2); properly prepare food to avoid illnesses, such as listeriosis (3); protect themselves from insects and animals known to carry diseases, such as Zika and toxoplasmosis (4); and maintain good hygiene to prevent infections, such as cytomegalovirus (5). CDC encourages everyone to join this nationwide effort to raise awareness of birth defects, their causes, and their impact. Additional information is available at <https://www.cdc.gov/ncbddd/birthdefects/prevention-month.html>.

January 8–14, 2017, is National Folic Acid Awareness Week. CDC urges all women who can become pregnant to get 400 μg of folic acid every day to help reduce the risk for serious birth defects of the brain and spine (spina bifida and other neural tube defects) (6). Women can get folic acid from fortified foods or supplements, or both. Additional information about folic acid is available at <http://www.cdc.gov/folicacid>.

References

1. CDC. Update on overall prevalence of major birth defects—Atlanta, Georgia, 1978–2005. *MMWR Morb Mortal Wkly Rep* 2008;57:1–5.
2. CDC. For women in areas with Zika: plan your pregnancy. Atlanta, GA: US Department of Health and Human Services, CDC; 2016. <http://www.cdc.gov/zika/pdfs/zika-plan-your-pregnancy.pdf>
3. US Department of Agriculture. Food safety for pregnant women. Silver Spring, MD: US Department of Agriculture, Food and Drug Administration; 2011. <http://www.fda.gov/downloads/Food/FoodborneIllnessContaminants/UCM312787.pdf>
4. CDC. Pregnant? Protect yourself from mosquito bites. Atlanta, GA: US Department of Health and Human Services, CDC; 2016. <http://www.cdc.gov/zika/pdfs/zika-pregnancy.pdf>
5. Cannon MJ, Davis KF. Washing our hands of the congenital cytomegalovirus disease epidemic. *BMC Public Health* 2005;5:70. <http://dx.doi.org/10.1186/1471-2458-5-70>
6. Honein MA, Paulozzi LJ, Mathews TJ, Erickson JD, Wong LY. Impact of folic acid fortification of the US food supply on the occurrence of neural tube defects. *JAMA* 2001;285:2981–6. <http://dx.doi.org/10.1001/jama.285.23.2981>

Notice to Readers

Final *MMWR* Issue Including Table III Data

This January 6, 2017, issue of *MMWR* (Vol. 65, No. 52) will be the last to include data from the National Center for Health Statistics (NCHS) Mortality Surveillance System in Notifiable Disease and Mortality Tables, Table III (“Number of

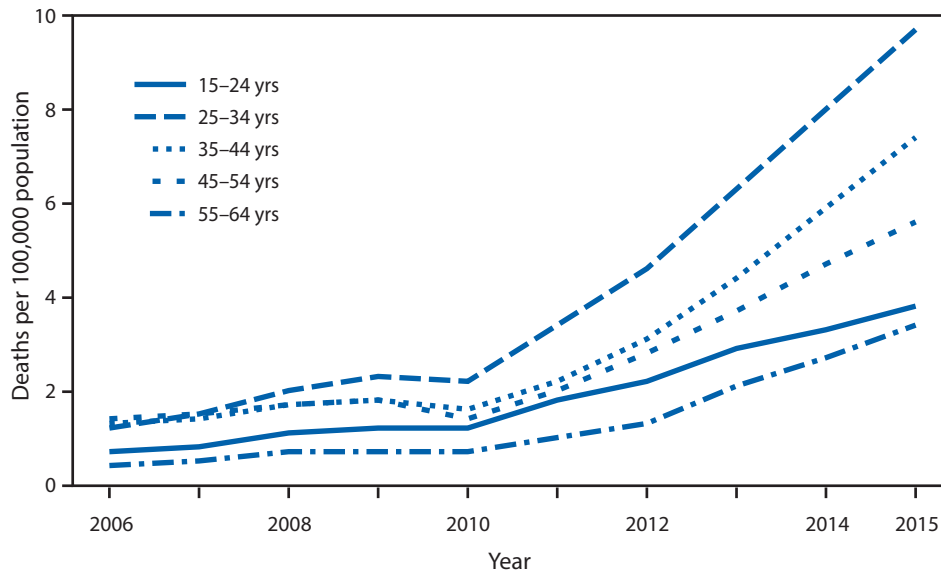
deaths from pneumonia and influenza and all deaths, by U.S. Department of Health and Human Services region and state”).

Beginning January 13, 2017, the most recent data from the NCHS Mortality Surveillance System can be found at <https://data.cdc.gov> and <https://gis.cdc.gov/grasp/fluview/mortality.html> (FluView Interactive).

QuickStats

FROM THE NATIONAL CENTER FOR HEALTH STATISTICS

Rates of Drug Overdose Deaths Involving Heroin,* by Selected Age Groups — United States, 2006–2015



* Drug overdose deaths involving heroin are identified using the *International Classification of Diseases, 10th Revision* underlying cause of death codes X40–X44, X60–X64, X85, and Y10–Y14, with a multiple cause of death code of T40.1. During 2006, there were 2,088 drug overdose deaths involving heroin (age-adjusted rate of 0.7 per 100,000 population); during 2015, there were 12,989 deaths (age-adjusted rate of 4.1).

The rate of drug overdose deaths involving heroin increased slightly during 2006–2010 but more than tripled during 2010–2015 for all age groups shown. During 2010–2015, the rates increased from 1.2 to 3.8 per 100,000 for persons aged 15–24 years, from 2.2 to 9.7 for persons aged 25–34 years, from 1.6 to 7.4 for persons aged 35–44 years, from 1.4 to 5.6 for persons aged 45–54 years, and from 0.7 to 3.4 for persons aged 55–64 years. In 2015, the rate of drug overdose deaths involving heroin was highest for persons aged 25–34.

Source: National Vital Statistics System mortality data. <http://www.cdc.gov/nchs/deaths.htm>.

Reported by: Holly Hedegaard, MD, hdh6@cdc.gov, 301-458-4460; Margaret Warner, PhD; Arialdi M. Miniño, MPH.

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

Morbidity and Mortality Weekly Report

The *Morbidity and Mortality Weekly Report (MMWR)* Series is prepared by the Centers for Disease Control and Prevention (CDC) and is available free of charge in electronic format. To receive an electronic copy each week, visit *MMWR*'s free subscription page at <http://www.cdc.gov/mmwr/mmwrsubscribe.html>. Paper copy subscriptions are available through the Superintendent of Documents, U.S. Government Printing Office, Washington, DC 20402; telephone 202-512-1800.

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