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Effects of Maternal Age and Age-Specific Preterm Birth Rates on Overall Preterm Birth Rates — United States, 2007 and 2014

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Reductions in births to teens and preterm birth rates are two recent public health successes in the United States (*1*,*2*). From 2007 to 2014, the birth rate for females aged 15–19 years declined 42%, from 41.5 to 24.2 per 1,000 females. The preterm birth rate decreased 8.4%, from 10.41% to 9.54% of live births (*1*). Rates of preterm births vary by maternal age, being higher among the youngest and oldest mothers. It is unknown how changes in the maternal age distribution in the United States have affected preterm birth rates. CDC used birth data to assess the relative contributions of changes in the maternal age distribution and in age-specific preterm birth rates to the overall decrease in preterm birth rates. The preterm birth rate declined in all age groups. The effects of age distribution changes on the preterm birth rate decrease were different in younger and older mothers. The decrease in the proportion of births to mothers aged ≤19 and 20–24 years and reductions in age-specific preterm rates in all age groups contributed to the overall decline in the preterm birth rate. The increase in births to mothers aged ≥30 years had no effect on the overall preterm birth rate decrease. The decline in preterm births from 2007 to 2014 is related, in part, to teen pregnancy prevention and the changing maternal age distribution. Effective public health strategies for further reducing preterm birth rates need to be tailored to different age groups.

National Vital Statistics System data for all live births to U.S. residents in 2007 and 2014 were analyzed for the effects of maternal age on the decline in preterm birth rates. The analysis was limited to births with gestational age ≥20 weeks, as determined by the obstetric estimate. The year 2007 was the first year the obstetric estimate was available nationally (*1*,*3*). The year 2014 was the most recent year with final birth data available at the time of analysis. Preterm birth rates were defined as <37 completed weeks of gestation and expressed as a percentage of live births. Maternal age was categorized as ≤19, 20–24, 25–29, 30–34, and ≥35 years. Using rate decomposition methods, the change in preterm birth rates from 2007 to 2014 was divided into two components: 1) changes in the maternal age distribution, and 2) changes in the age-specific preterm birth rates (*4*). The two components were calculated relative to each other; one was held constant (by using the average value for the 2 years) as the observed variation in the other component was assessed. The sum of the two components across the age groups equaled the total preterm birth rate difference (*4*).

From 2007 to 2014, maternal age increased from a mean of 27.4 years to 28.3 years (Figure). A decrease in the percentage of births to mothers aged ≤24 years was observed, which included a 39.5% decrease in births to teens and an increase in births to women aged ≥25 years (Table).

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FIGURE. Percentages of live births (LBs) and preterm births (PTBs), by maternal age — United States, 2007 and 2014

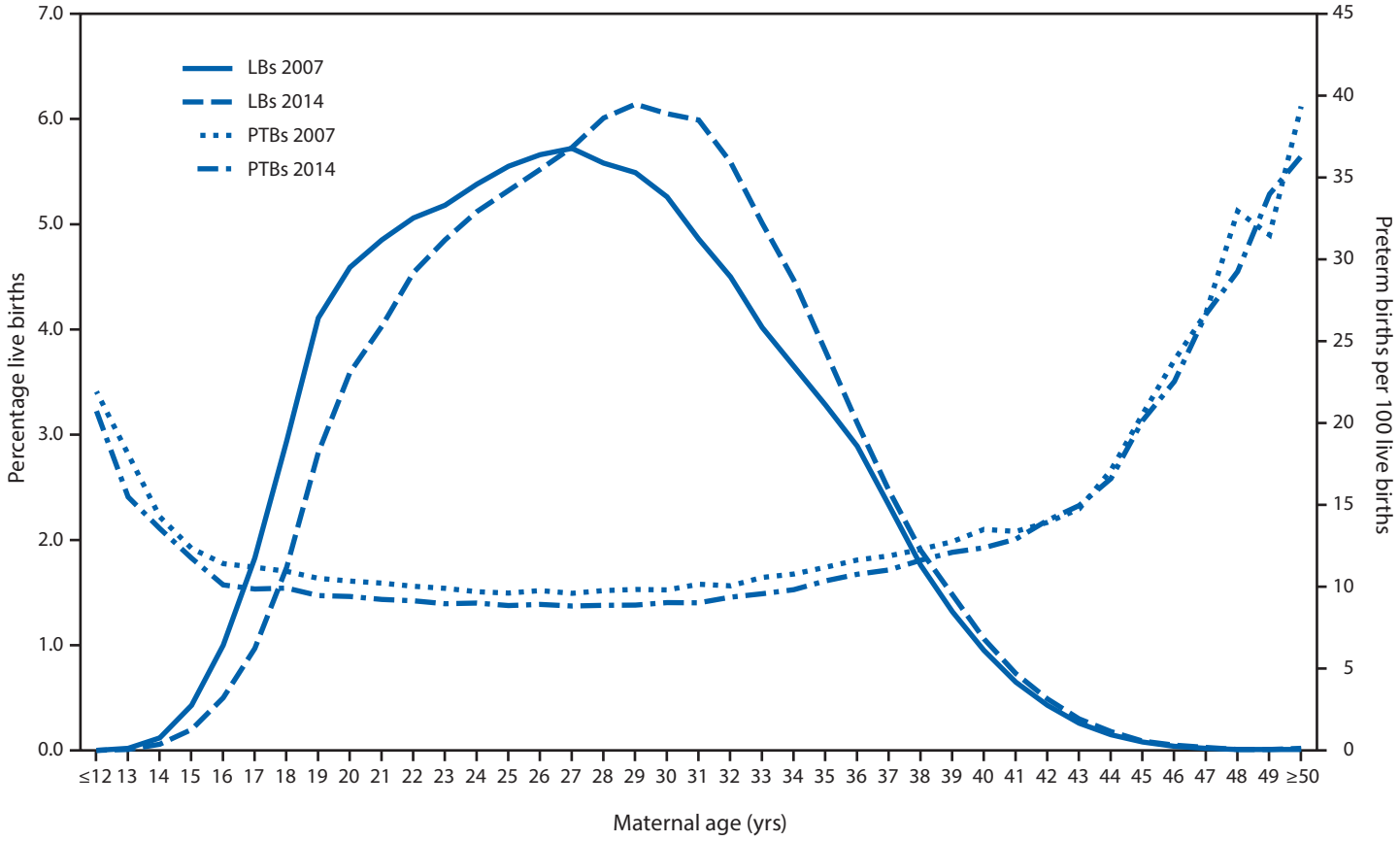

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TABLE. Number and percentage of all births and preterm births and the components of the preterm rate change, by maternal age — United States, 2007 and 2014

Maternal age (yrs)	All births			Preterm births			Rate change components*		
	2007 No. (%)	2014 No. (%)	% Change	2007 No. (%)	2014 No. (%)	Rate difference	Age distribution	Age-specific rate	Total effect
<20	448,461 (10.4)	251,467 (6.3)	-39.5	49,222 (10.98)	24,707 (9.82)	-1.15	-0.43	-0.10	**-0.53**
20–24	1,076,492 (25.1)	881,395 (22.1)	-11.7	107,989 (10.03)	80,477 (9.13)	-0.90	-0.28	-0.21	**-0.49**
25–29	1,202,608 (28.0)	1,144,008 (28.7)	2.6	116,846 (9.72)	101,450 (8.87)	-0.85	0.07	-0.24	**-0.17**
30–34	957,551 (22.3)	1,080,027 (27.1)	21.6	97,982 (10.23)	100,750 (9.33)	-0.90	0.47	-0.22	**0.25**
≥35	609,370 (14.2)	626,513 (15.7)	10.8	74,977 (12.30)	72,523 (11.58)	-0.73	0.18	-0.11	**0.08**
Total	**4,294,482 (100)**	**3,983,410 (100)**	**NA**	**447,016 (10.41)**	**379,901 (9.54)**	**-0.87**	**0.01**	**-0.88**	**-0.87**

Abbreviation: NA = not applicable.

* Per 100 live births. For each age stratum, the decomposition components are calculated as follows: Let P_{2007} = the proportion of the age distribution in 2007 and P_{2014} = the proportion of the age distribution in 2014. Let R_{2007} = the preterm birth rate in 2007 and R_{2014} = the preterm birth rate in 2014. The age distribution component = $(P_{2014} - P_{2007}) \times [(R_{2007} + R_{2014})/2]$. The age-specific rate component = $(R_{2014} - R_{2007}) \times [(P_{2007} + P_{2014})/2]$.

From 2007 to 2014, the preterm birth rate decreased from 10.41% to 9.54%, an absolute rate difference of -0.87% (Table). A U-shaped relationship between maternal age and preterm birth was present in both years with the lowest preterm birth rate occurring among women aged 25–29 years (Table) (Figure). The decrease in preterm birth rates from 2007 to 2014 was observed for mothers at all ages <42 years. The absolute rate difference was highest among teens and lowest among women aged ≥35 years (Table).

The decomposition analysis partitioned the overall observed rate difference of -0.87% into two parts, age distribution and age-specific rate components (Table). The change in age distribution contributed to the preterm birth rate decrease (as indicated by the negative values) only among mothers aged ≤24 years. In contrast, the age distribution component for mothers aged ≥25 years, and especially for mothers aged ≥30 years, offset this decline. When the age distribution components were summed across the age groups, a negligible effect (0.01) was observed on the overall change in preterm birth rates. The change in age-specific preterm birth rates contributed to the decline in preterm birth rate across all age groups.

Examining the total effect of both components on the preterm birth rate decline by age group, the largest total effects were observed among mothers aged ≤19 and mothers aged 20–24 years (Table). In these two groups, the change in age distribution had a larger effect than the change in the age-specific preterm birth rate. For mothers aged 25–29 years, the total effect also contributed to the overall preterm birth rate decline because the age-specific rate component was greater than the age distribution component. For mothers aged ≥30 years, the total effect of both components did not contribute to the overall preterm birth rate decrease; the rate increases from the age distribution components were greater than the rate decreases from the age-specific rate components.

These analyses included all births; however, sensitivity analyses restricting to singleton births produced similar results. The overall absolute rate difference for singletons during this period was -0.85%, compared with the -0.87% for all births.

Discussion

The overall decline in the preterm birth rate from 2007 to 2014 was related to declines in age-specific preterm birth rates and a decrease in prevalence of births to teens and women aged 20–24 years. The contribution from mothers aged ≥24 years to the age-distribution component was offset by an increased prevalence of births to older mothers who have high rates of preterm birth. Thus, the total age distribution component masked divergent influences of younger and older mothers on the overall preterm birth rate decline. Because of this, the influence of younger mothers on the overall preterm birth rate decline is more appropriately indicated by examining the age-specific total effects in the decomposition analysis. Considering relative effects of both age distribution and age-specific preterm birth rate components, only mothers aged ≤29 years contributed to the overall rate decline, with the largest contributions from teens and women aged 20–24 years.

Other studies have documented increased preterm birth rates among teen and older mothers compared with mothers in their mid-twenties to early thirties (*5*). Although teen and older mothers might share some common preterm birth risk factors, such as low socioeconomic status, extremes of body mass index, and smoking, physiologic immaturity is a risk factor for teen mothers and the prevalence of preexisting chronic disease is greater among older mothers (*5*,*6*). This heterogeneity of risk for preterm births according to maternal age and the variation in changes in age-specific preterm birth rates, combined with the changes in maternal age distribution over time, suggest the need for varying preterm birth prevention strategies across the reproductive life span.

The findings in this report are subject to at least one limitation: the relationship of preterm birth with maternal age is associative, not causal. The analysis did not assess the impact

Summary

What is already known about this topic?

Rates of births to teens and of preterm births declined in the United States from 2007 to 2014. Preterm births are more common among the youngest and oldest mothers.

What is added by this report?

Preterm birth rates declined for all age groups and overall from 10.41% to 9.54% of live births. Mean maternal age increased from 27.4 years to 28.3 years. The contribution of fewer births to teens and to women aged 20–24 years to the overall decline in preterm births was offset by increases in births to older mothers.

What are the implications for public health practice?

The changing distribution of maternal age might indicate success of programs to prevent teen and unintended pregnancies. Effective public health strategies for further reducing preterm birth rates need to be tailored to different age groups.

of other pregnancy outcomes, such as elective termination or fetal death, or of potential confounders, such as maternal race/ethnicity, obstetric history, smoking, socioeconomic status, body mass index, chronic or pregnancy-related conditions, prenatal care, and delivery method (*5*–*7*). The effects on preterm birth rates of maternal 17-hydroxyprogesterone use, a preterm birth prevention strategy that increased during this period (*6*), were not examined and the effects of maternal age on spontaneous, medically indicated, early, or late preterm births were not assessed.

The overall decline in the preterm birth rate from 2007 to 2014 is related in part to the changing maternal age distribution associated with the success of teen pregnancy prevention and declines in unintended pregnancy (*8*). Teen pregnancy prevention is one of CDC's Winnable Battles (*9*). Although teen pregnancy prevention and family planning have many positive health and societal effects, the results of this analysis suggest these programs might also have direct effects on reducing preterm birth rates. Based on recent data, 75% of pregnancies to teens aged 15–19 years and 59% of pregnancies to women aged 20–24 years are unintended (*8*). The need for prevention of first and repeat teen pregnancies (*10*) continues. Prevention of unintended pregnancy and encouragement of optimal birth spacing is one part of a five-part strategy for preterm birth prevention (*7*). Other strategies include improved access to preconception care, preterm birth risk identification and treatment, reduction of elective delivery before 39 weeks gestation, and single embryo transfer in assisted reproductive technology (*7*). These strategies need to be implemented throughout the reproductive life span to reduce preterm births for all maternal ages.

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Ocular Syphilis — Eight Jurisdictions, United States, 2014–2015

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Ocular syphilis, a manifestation of *Treponema pallidum* infection, can cause a variety of ocular signs and symptoms, including eye redness, blurry vision, and vision loss. Although syphilis is nationally notifiable, ocular manifestations are not reportable to CDC. Syphilis rates have increased in the United States since 2000. After ocular syphilis clusters were reported in early 2015, CDC issued a clinical advisory (*1*) in April 2015 and published a description of the cases in October 2015 (*2*). Because of concerns about an increase in ocular syphilis, eight jurisdictions (California, excluding Los Angeles and San Francisco, Florida, Indiana, Maryland, New York City, North Carolina, Texas, and Washington) reviewed syphilis surveillance and case investigation data from 2014, 2015, or both to ascertain syphilis cases with ocular manifestations. A total of 388 suspected ocular syphilis cases were identified, 157 in 2014 and 231 in 2015. Overall, among total syphilis surveillance cases in the jurisdictions evaluated, 0.53% in 2014 and 0.65% in 2015 indicated ocular symptoms. Five jurisdictions described an increase in suspected ocular syphilis cases in 2014 and 2015. The predominance of cases in men (93%), proportion of those who are men who have sex with men (MSM), and percentage who are HIV-positive (51%) are consistent with the epidemiology of syphilis in the United States. It is important for clinicians to be aware of potential visual complications related to syphilis infections. Prompt identification of potential ocular syphilis, ophthalmologic evaluation, and appropriate treatment are critical to prevent or manage visual symptoms and sequelae of ocular syphilis.

In early 2015, clusters of ocular syphilis cases were reported in Washington and California. CDC issued a clinical advisory, notifying clinical providers and health departments of a potential increase in suspected ocular syphilis cases. After this advisory, eight jurisdictions performed a review of syphilis surveillance and case investigation data to identify syphilis cases with ocular manifestations. Seven jurisdictions reviewed data from January 1, 2014 to December 31, 2015; Indiana reviewed data from 2015 only. A patient whose illness met the surveillance case definition of syphilis (*3*) was considered to have a suspected case of ocular syphilis if the patient had concurrent ocular signs or symptoms noted in the surveillance database from a local syphilis case investigation or reported by a local health care provider. A standard form was used to abstract de-identified information on each case, including demographic information, syphilis stage and treatment, and any information on extent of ocular involvement. Each jurisdiction also provided a total number of syphilis surveillance cases, including numbers from all stages of syphilis, as defined by the surveillance case definitions (*3*).

A total of 388 suspected ocular syphilis cases were identified, 157 cases in 2014 and 231 cases in 2015 (Table 1). Overall, 0.60% of total syphilis cases were identified as suspected ocular syphilis cases, 0.53% in 2014 and 0.65% in 2015. The percentage of total syphilis cases with ocular manifestation varied by jurisdiction, ranging from 0.17% to 3.9%. Five jurisdictions described an increase in suspected ocular syphilis cases in 2014 and 2015.

TABLE 1. Suspected ocular syphilis and total syphilis cases — eight jurisdictions, United States, 2014–2015

	Suspected ocular syphilis		Total surveillance syphilis cases		% surveillance syphilis cases with suspected ocular syphilis	
Jurisdiction	**2014**	**2015**	**2014**	**2015**	**2014**	**2015**
California*	48	60	6,238	7,824	0.77	0.77
Florida	10	32	6,030	7,154	0.17	0.45
Indiana†	—	8	—	714	—	1.10
Maryland	10	17	1,524	1,779	0.66	0.96
New York City	14	12	5,798	6,116	0.24	0.20
North Carolina	21	42	1,799	2,435	1.20	1.70
Texas	27	16	7,337	8,400	0.37	0.19
Washington	27	44	857	1,125	3.20	3.90
Total	**157**	**231**	**29,583**	**35,547**	**0.53**	**0.65**

* California does not include syphilis reports from San Francisco or Los Angeles.
† Indiana reviewed data from 2015 only.

Most patients with suspected ocular syphilis were male (93%), and 249 (69%) of those with information on sex partners were MSM (Table 2). The mean age of patients was 44 years (range = 17–79 years). Approximately one half of the cases met surveillance criteria for early syphilis (primary, secondary, and early latent syphilis) (Table 3); stage of syphilis was not associated with any specific symptom, diagnosis, or extent of eye involvement. Overall, patients with suspected ocular syphilis had high rapid plasma reagin (RPR) titers, with a median titer of 128 (range = 1–16,384). Approximately 22% of patients reported additional symptoms of neurosyphilis, including headache, neck pain, altered mental status, or changes in hearing.

Specific symptoms were reported by 326 (84%) persons suspected of having ocular syphilis; 54% of patients reported blurry vision, and 28% of patients reported at least some vision loss. Specific ocular diagnoses were available for 158 (41%) patients, and uveitis (n = 72) was the most common diagnosis. More serious diagnoses were also recorded, including retinitis (n = 20), optic neuritis (n = 18), and retinal detachment (n = 6). Of 136 (35%) patients with available information on which eye was affected, one eye was involved in 64 (47%) patients, and both eyes were affected in 72 (53%) patients.

Among 174 patients with cerebrospinal fluid (CSF) test results, 122 (70%) had a reactive CSF Venereal Disease Research Laboratory (VDRL) test. Patients with a reactive CSF VDRL test were not more likely than patients with a nonreactive CSF VDRL to report additional neurologic symptoms, have vision loss or bilateral eye involvement, or be diagnosed with severe disease, including retinitis, optic neuritis, or retinal detachment.

Recommended treatment for neurosyphilis and ocular syphilis is 18–24 million units intravenous (IV) aqueous penicillin G, administered daily as a continuous infusion, or divided into every 4-hour dosing, for 10–14 days (*4*). Approximately 60% of patients with suspected ocular syphilis received IV penicillin, and the other 38% received varied treatments, most commonly benzathine penicillin given as an intramuscular injection (Table 3).

One half of patients with suspected ocular syphilis were HIV-positive (n = 198). Of those persons, 62 (32%) were first diagnosed with HIV at the time of their ocular syphilis diagnosis. Compared with HIV-negative patients, HIV-positive patients had a higher median RPR titer (256 versus 128, p<0.001), more often received a lumbar puncture (57.1% versus 40.8%, p = 0.005), and were more often treated with IV penicillin (66.7% versus 44.2%, p<0.001). Patients with HIV infection did not differ significantly from HIV-negative patients in proportion having a reactive CSF VDRL, both eyes involved, additional symptoms of neurosyphilis, or an ophthalmologic exam. CD4 count was available for 126 patients; 84 (67%) had a CD4 count <500 cells/μL. Compared with patients with higher CD4 counts, those with a CD4 count <500 cells/μL more often received a lumbar puncture (65.5% versus 45.2%, p = 0.03), had a reactive CSF VDRL (84.0% versus 50.0%, p = 0.006), and had both eyes involved (35.7% versus 7.1%, p = 0.003), but did not differ in proportion with blurry vision or vision loss.

TABLE 2. Demographic characteristics of patients with suspected ocular syphilis — eight jurisdictions, United States, 2014–2015

Characteristic	No.	(%)
Total	**388**	**(100.0)**
Male	362	(93.3)
Known MSM (among 362 males)	249	(68.8)
Race		
White	217	(55.9)
Black	81	(20.9)
Hispanic	48	(12.4)
Asian	13	(3.4)
Native Hawaiian/Pacific Islander	1	(0.3)
Other/Unknown	28	(7.2)
HIV-positive	198	(51.0)

Abbreviations: HIV = human immunodeficiency virus; MSM = men who have sex with men.

Discussion

Ocular syphilis is a serious manifestation of syphilis. This report is the first evaluating suspected ocular syphilis across multiple jurisdictions in the United States. Although there is no national reporting of ocular manifestations, eight jurisdictions reviewed their syphilis surveillance data to identify cases with ocular manifestations in 2014 and 2015. Ocular manifestations were present in 0.60% of all reported syphilis cases, ranging by jurisdiction from 0.17% to 3.9%. In most jurisdictions, the percentage was similar to data from a study in England that estimated ocular syphilis affected approximately 0.6% of early syphilis cases from 2009 to 2010 (*5*). Five of the seven jurisdictions that reviewed cases in both years described an increase in suspected ocular syphilis cases in 2014 and 2015. In addition, after the clinical advisory, CDC was notified of suspected ocular syphilis cases from 20 states (*2*). The number of cases with ocular syphilis detected in 2014 and 2015 in the United States could be attributable to increased recognition of ocular manifestations in the setting of increased syphilis rates, or an actual increase in the proportion of syphilis cases with ocular disease. The predominance of cases in men and MSM, as well as the proportion also diagnosed with HIV, is consistent with the epidemiology of syphilis in the United States (*6*)

Although two of the cases from Washington reported in the October 2015 *MMWR* (*2*) were in sex partners, no suspected

TABLE 3. Clinical characteristics, laboratory results and diagnoses for syphilis and suspected ocular syphilis — eight jurisdictions, United States, 2014–2015

Characteristic	No.	(%)
Total	**388**	**(100.0)**
Stage of syphilis		
Primary	8	(2.1)
Secondary	101	(26.0)
Early latent	79	(20.4)
Late or latent of unknown duration	193	(49.7)
Unknown	7	(1.8)
Additional symptoms of neurosyphilis	87	(22.4)
Reported ocular symptoms (among 326 with symptoms)		
Blurry vision	210	(64.4)
Vision loss	107	(32.8)
Eye pain or red eye	46	(14.1)
Eye exam	158	(40.7)
Diagnosis (among 158 with documented eye exam)*		
Uveitis	72	(45.6)
Retinitis	20	(12.7)
Optic neuritis	18	(11.4)
Retinal detachment	6	(3.8)
CSF analysis performed	188	(48.5)
CSF VDRL (among 174 with a documented result)		
Reactive	122	(70.1)
Nonreactive	52	(29.9)
Treatment		
Aqueous penicillin G IV	230	(59.3)
Other treatment	146	(37.6)
No/Unknown treatment	12	(3.1)

Abbreviations: CSF = cerebrospinal fluid; IV = intravenous; VDRL = Venereal Disease Research Laboratory test.
* Can be included in multiple categories.

ocular syphilis cases in North Carolina, Indiana, New York City, or Florida named a person with ocular syphilis as a sex partner; data from other jurisdictions were unavailable. In addition, in a preliminary study, no specific strain was identified in patients with ocular syphilis, suggesting a single oculotropic strain of *T. pallidum* is not responsible for the apparent increase in ocular syphilis (*7*). The absence of both a specific strain and epidemiologic links supports a hypothesis that manifestations of ocular syphilis occur in a subset of patients with syphilis infection, possibly influenced by undetermined risk factors.

The findings in this report are subject to at least five limitations. First, each jurisdiction had slightly different methods for identifying patients with possible ocular manifestations of syphilis; capacity and infrastructure to investigate syphilis vary by state as well. Second, patients with ocular syphilis would have been missed if ocular symptoms were not documented in the case investigation, or if a case investigation was not completed per local investigation protocols. Therefore, the numbers described here could either underrepresent or overrepresent the burden of ocular syphilis. Third, a majority of jurisdictions did not request medical charts to confirm that ocular symptoms were syphilis-related; therefore, some symptoms could have causes unrelated to syphilis infection. Fourth, many cases had incomplete information provided as specific ocular diagnoses are not routinely collected as part of syphilis case investigations. Finally, trends over time cannot be assessed because information about suspected ocular syphilis was not available for years before 2014.

Public health interventions aimed at both providers and persons at risk are necessary to prevent ocular syphilis, and to ensure prompt diagnosis and treatment. All patients diagnosed with syphilis that exhibit ocular manifestations, such as eye pain, blurry vision, or vision loss, should immediately be treated for neurosyphilis, and be referred for expert ophthalmologic examination. Severe outcomes, including blindness, occur in both HIV-positive and HIV-negative patients. Further investigation is currently underway to identify additional risk factors specific to ocular syphilis. Because the prevalence of syphilis is increasing in the United States, education of both patients and providers is critical to identify ocular manifestations of syphilis as early as possible and manage disease sequelae.

Summary

What is already known about this topic?

Ocular syphilis, an infrequent manifestation of syphilis infection, can cause a variety of eye symptoms, including vision loss. Clusters of ocular syphilis were reported from late 2014 to 2015. In the United States, syphilis rates have increased since 2000, but little is known about ocular syphilis cases.

What is added by this report?

Eight jurisdictions that reviewed syphilis surveillance and case investigation data from 2014, 2015, or both found that in 0.6% of syphilis cases, the patient had symptoms consistent with ocular syphilis. Most suspected cases were in males, and half were in HIV-positive persons. Severe outcomes, including blindness, occurred in both HIV-positive and HIV-negative patients.

What are the implications for public health practice?

All patients diagnosed with syphilis that exhibit ocular manifestations should immediately be treated for neurosyphilis and be referred for formal ophthalmologic examination. Education of both patients and providers is critical to identify ocular manifestations of syphilis and manage disease sequelae.

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Recommendations for Use of Meningococcal Conjugate Vaccines in HIV-Infected Persons — Advisory Committee on Immunization Practices, 2016

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At its June 2016 meeting, the Advisory Committee on Immunization Practices (ACIP) recommended routine use of meningococcal conjugate vaccine (serogroups A, C, W, and Y; including MenACWY-D [Menactra, Sanofi Pasteur] or MenACWY-CRM [Menveo, GlaxoSmithKline]) for persons aged ≥2 months with human immunodeficiency virus (HIV) infection. ACIP has previously recommended routine vaccination of persons aged ≥2 months who have certain medical conditions that increase risk for meningococcal disease (*1*), including persons who have persistent (e.g., genetic) deficiencies in the complement pathway (e.g., C3, properdin, Factor D, Factor H, or C5–C9); persons receiving eculizumab (Soliris, Alexion Pharmaceuticals) for treatment of atypical hemolytic uremic syndrome or paroxysmal nocturnal hemoglobinuria (because the drug binds C5 and inhibits the terminal complement pathway); and persons with functional or anatomic asplenia (including persons with sickle cell disease). Routine vaccination with meningococcal conjugate vaccine is also recommended for all healthy adolescents in the United States (*1*). This report summarizes the evidence considered by ACIP in recommending vaccination for HIV-infected persons, and provides recommendations and guidance for use of meningococcal conjugate vaccines (serogroups A, C, W, and Y) among HIV-infected persons aged ≥2 months; the majority of meningococcal disease among HIV-infected persons is caused by these four serogroups.

Recommendations for routine use of vaccines in children, adolescents, and adults are developed by the Advisory Committee on Immunization Practices (ACIP). ACIP is chartered as a federal advisory committee to provide expert external advice and guidance to the Director of the Centers for Disease Control and Prevention (CDC) on use of vaccines and related agents for the control of vaccine-preventable diseases in the civilian population of the United States. Recommendations for routine use of vaccines in children and adolescents are harmonized to the greatest extent possible with recommendations made by the American Academy of Pediatrics (AAP), the American Academy of Family Physicians (AAFP), and the American College of Obstetricians and Gynecologists (ACOG). Recommendations for routine use of vaccines in adults are harmonized with recommendations of AAFP, ACOG, and the American College of Physicians (ACP). ACIP recommendations approved by the CDC Director become agency guidelines on the date published in the Morbidity and Mortality Weekly Report (MMWR). Additional information is available at http://www.cdc.gov/vaccines/acip.

Methods

The ACIP Meningococcal Vaccines Work Group reviewed the immunogenicity and safety data from two studies of MenACWY-D in HIV-infected persons (*2–4*) during monthly teleconferences. No studies of immunogenicity or safety of MenACWY-CRM in HIV-infected persons are available. According to a nonsystematic literature search of PubMed using the search terms "meningococcal conjugate vaccine," "quadrivalent," and "HIV," and consultation with the manufacturers, these two studies represent all known evidence for the immunogenicity and safety of these vaccines in HIV-infected persons. The work group also evaluated the evidence and unpublished surveillance data regarding meningococcal disease epidemiology among HIV-infected persons in the United States and a cost-effectiveness analysis of routine vaccination of HIV-infected persons. The cost-effectiveness analysis of routine vaccination versus no vaccination of HIV-infected persons was conducted assuming an initial vaccine efficacy of 75% (range = 37%–91%) for persons with a high CD4 count and 37% (range = 24%–60%) for persons with a low CD4 count based on the immunogenicity data reported in the literature for MenACWY-D (*2–4*). A summary of the data reviewed and work group discussions was presented to ACIP; recommendations for use of meningococcal conjugate vaccines among HIV-infected persons aged ≥2 months were approved by ACIP at its June 22, 2016 meeting (detailed meeting minutes are available at http://www.cdc.gov/vaccines/acip/meetings/meetings-info.html).

CDC vaccine recommendations are developed using the Grading of Recommendations, Assessment, Development, and Evaluation (GRADE) framework (http://www.cdc.gov/vaccines/acip/recs/grade/index.html). The type and quality of available evidence supporting the use of meningococcal conjugate vaccines among HIV-infected persons aged ≥2 months were evaluated using GRADE. There is no available evidence for safety or effectiveness of these vaccines in HIV-infected persons aged <2 years or ≥25 years.

Meningococcal Disease in HIV-Infected Persons

Surveillance data for cases of meningococcal disease among HIV-infected persons in the United States are limited. The HIV status of patients with meningococcal disease is routinely captured in Active Bacterial Core surveillance (ABCs), an active population-based and laboratory-based surveillance system that operates in 10 sites, representing a population of approximately 43 million persons, or 14% of the U.S. population (*5*). However, the HIV status of patients with meningococcal disease is not reported through the National Notifiable Diseases Surveillance System (NNDSS), a passive surveillance system that operates in all U.S. states and territories. During 1995–2014, a total of 62 cases of meningococcal disease among HIV-infected persons were reported to ABCs; these cases represent 2% of the 3,951 meningococcal disease cases reported to ABCs during that period (CDC, unpublished data, 2016). Thirteen (21%) cases were serogroup B, 23 (37%) were serogroup C, three (5%) were serogroup W, 17 (27%) were serogroup Y, and six (10%) were other/unknown serogroups (CDC, unpublished data, 2016). The majority (92%) of cases of meningococcal disease among HIV-infected persons occurred in adults aged 20 through 59 years.

Although surveillance data for cases of meningococcal disease among HIV-infected persons are limited in the United States, a growing body of evidence demonstrates an increased risk for meningococcal disease among HIV-infected persons. In studies from South Africa, the United States, and the United Kingdom, the incidence of meningococcal disease in HIV-infected persons ranged from 3.4 to 6.6 per 100,000 (relative risk = 5–13 compared with HIV-uninfected persons) (Table 1) (*6*–*9*). Among HIV-infected persons, a low CD4 count or high viral load were associated with an increased risk (*7*). Similar increased risk for meningococcal disease was observed for both males and females with HIV infection (*7*,*9*).

Data on the case-fatality ratio of meningococcal disease in HIV-infected persons vary: in the South Africa study, the case-fatality ratio among HIV-infected patients was 20%, compared with 11% among patients who did not have HIV infection (*6*). However, in the most recent studies from New York City and the United Kingdom, the meningococcal disease case fatality ratio observed among HIV-infected patients was lower than that among HIV-uninfected patients (*7*,*8*).

MenACWY-D Immunogenicity and Safety in HIV-Infected Persons

The immunogenicity and safety of MenACWY-D in 324 HIV-infected adolescents and young adults aged 11 through 24 years were evaluated in an open-label trial with a randomized second dose component (*2*,*4*). At study entry, participants received 1 dose of MenACWY-D. At 24 weeks, participants with CD4 percentage (the percentage of total lymphocytes that are CD4 cells) (CD4%) ≥15% were randomized to receive or not receive a second dose of MenACWY-D; all participants with CD4% <15% received a second dose. Vaccine effectiveness was inferred from serum bactericidal antibodies, measured using a serum bactericidal assay with a rabbit complement

TABLE 1. Evidence of increased risk for meningococcal disease among HIV-infected persons compared with HIV-uninfected persons — seven study populations, 1996–2013

Period	Study site	Age group	No. of cases*	Increase in meningococcal disease rate among HIV-infected compared with HIV-uninfected persons	Serogroups
1996–1999	Australia†	All ages	60	5-fold	B, C
1990–2000	London§	All ages	2,900	14-fold	B, C
1988–1993	Atlanta, Georgia¶	18–45 years	132	24-fold	B, C, Y
2003–2007	South Africa**	All ages	504	11-fold	A, B, C, W, Y
2000–2008	United States ABCs††	25–64 years	491	13-fold	B, C, W, Y
2000–2011	New York City§§	15–64 years	265	10-fold	C, Y
2011–2013	United Kingdom¶¶	All ages	2,353	5-fold	A, B, C, W, Y

Abbreviations: ABCs = Active Bacterial Core surveillance; HIV = human immunodeficiency virus.

* Total number of meningococcal disease cases reported during the study period regardless of HIV infection status

† Couldwell DL. Invasive meningococcal disease and HIV coinfection. Commun Dis Intell Q Rep 2001;25:279–80.

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¶ Stephens DS, Hajjeh RA, Baughman WS, Harvey RC, Wenger JD, Farley MM. Sporadic meningococcal disease in adults: results of a 5-year population-based study. Ann Intern Med 1995;123:937–40.

** Cohen C, Singh E, Wu HM, et al. Increased incidence of meningococcal disease in HIV-infected individuals associated with higher case-fatality ratios in South Africa. AIDS 2010;24:1351–60.

†† Harris CM, Wu HM, Li J, et al. Meningococcal disease in patients with HIV infection — a review of cases reported through active surveillance in the United States, 2000–2008. Open Forum Infect Dis 2016. Epub October 24, 2016. http://ofid.oxfordjournals.org/content/early/2016/10/24/ofid.ofw226.full.pdf+html.

§§ Miller L, Arakaki L, Ramautar A, et al. Elevated risk for invasive meningococcal disease among persons with HIV. Ann Intern Med 2014;160:30–7.

¶¶ Simmons RD, Kirwan P, Beebeejaun K, et al. Risk of invasive meningococcal disease in children and adults with HIV in England: a population-based cohort study. BMC Med 2015;13:297.

source (rSBA). Rates of seroresponse (proportion of subjects with a ≥fourfold rise in rSBA titer compared with the baseline titer) against each meningococcal serogroup (A, C, W, Y), geometric mean titers (GMT), and the percentage of subjects with rSBA at or above a predefined titer (≥1:128) were determined from sera obtained at study entry and at weeks 4, 24, 28, and 72. Adverse events (AEs) were assessed for 6 weeks after each MenACWY-D dose through active follow-up (*2*,*4*).

Among participants with CD4% ≥15% who received 1 vaccine dose, the proportions of participants with rSBA titers ≥1:128 at 4, 28, and 72 weeks were 65%, 31%, and 21%, respectively, against serogroup C, and 83%, 75%, 63%, respectively, against serogroup Y (*2*). Among participants with CD4% ≥15% who received 2 doses (at 0, 24 weeks), the proportions of participants with rSBA titers ≥1:128 at 4, 28, and 72 weeks were 59%, 64%, and 35%, respectively, against serogroup C; and 73%, 83%, 71%, respectively, against serogroup Y (*2*). Among participants with CD4% <15%, all of whom received 2 doses (at 0, 24 weeks), the proportions of participants with rSBA titers ≥1:128 at 4, 28, and 72 weeks were 22%, 22%, and 6%, respectively, against serogroup C, and 30%, 30%, 28%, respectively, against serogroup Y (*2*). A serious AE was experienced by 2.2%–6.5% of participants through 6 weeks post-vaccination*; one serious AE (ocular pain) was judged to be related to MenACWY-D. Serious AE rates were inversely related to entry CD4%. Two deaths were reported, but both were determined to be unrelated to the vaccine (*2*,*4*).

The immunogenicity and safety of MenACWY-D in 59 HIV-infected children aged 2 through 10 years with CD4% ≥25% was evaluated in an open label trial (*3*). Participants received MenACWY-D at study entry and at week 24. Vaccine effectiveness was inferred from serum bactericidal antibodies, measured using a serum bactericidal assay with rSBA. Rates of seroresponse (proportion of subjects with a ≥fourfold rise in post-vaccination rSBA titer compared with the baseline titer) against each meningococcal serogroup (A, C, W, Y), GMTs, and the percentage of subjects with rSBA at or above a predefined titer (≥1:128) were determined from sera obtained at entry and weeks 4, 24, 28, and 72. Study participants were assessed for AEs 6 weeks after each MenACWY-D dose (*3*).

The proportion of participants with rSBA titers ≥1:128 after 1 dose (week 4) and 2 doses (week 28) of MenACWY-D were 96% and 96%, respectively, for serogroup A, 49% and 80%, respectively, for serogroup C, 98% and 100%, respectively, for serogroup W, and 90% and 98%, respectively, for serogroup Y (*3*). At week 72 the proportions of participants with rSBA titers ≥1:128 were 80% for serogroup A, 45% for serogroup C, 95% for serogroup W, and 91% for serogroup Y (*3*). Overall, 5% of participants reported a serious AE; no AE was judged to be related to MenACWY-D (*3*).

Evidence supporting the use of meningococcal conjugate vaccines in HIV-infected persons was evaluated using the GRADE framework and was determined to be type 3 (low level of evidence) (Table 2). The recommendation was designated as Category A (recommended for all persons in an age-based or risk factor–based group) because of the epidemiologic data supporting an increase in risk for meningococcal disease among HIV-infected persons.

From a lifetime perspective, it is estimated that, compared with no vaccination, approximately 122 (95% confidence interval [CI] = 116–129) cases and 23 (CI = 18–29) deaths could be prevented, and 385 (CI = 230–458) quality-adjusted life years (QALYs) could be saved, at a mean cost per QALY of $732,000 (CI = $337,000–$1,218,000) with a meningococcal conjugate vaccination program that includes a primary vaccination series followed by lifelong booster doses until age 70 years, targeting all currently HIV-infected persons aged ≥2 months in the United States (CDC, unpublished data, 2016).†

Recommendations

HIV-infected persons aged ≥2 months should routinely receive meningococcal conjugate vaccine (Table 3). HIV-infected children aged <2 years should receive the vaccine in accordance with the age-appropriate, licensed, multidose schedule (*1*,*10*). Persons aged ≥2 years with HIV infection who have not been previously vaccinated should receive a 2-dose primary series of MenACWY conjugate vaccine. Persons aged ≥2 years with HIV infection who have been previously

* Serious adverse events (AEs) defined as Guillain-Barré syndrome, death, and new grade 3 or higher AE according to the December 2004 Division of AIDS AE Grading Table.

† Unpublished data, ACIP meeting June 2016. Key model assumptions were presented at the June 2016 ACIP meeting. Methods described in Shepard CW, Ortega-Sanchez IR, Scott RD 2nd, Rosenstein NE. Cost-effectiveness of conjugate meningococcal vaccination strategies in the United States. Pediatrics 2005;115:1220–32.

TABLE 2. Summary of evidence for meningococcal conjugate vaccination of HIV-infected persons aged ≥2 months using the Grading of Recommendations, Assessment, Development, and Evaluation (GRADE)* framework — United States

Outcome	Evidence type†
Benefits	
Short-term immunogenicity 4 weeks after 1 dose (week 4)	3
Short-term immunogenicity 4 weeks after 2 doses (week 28)	3
Persistence of immunogenicity 48 weeks after 2 doses (week 72)	3
Harms	
Serious adverse events (after any dose)	4

* http://www.cdc.gov/vaccines/acip/recs/grade/index.html.
† Evidence type: 1 = highest level of evidence; 2 = high level of evidence; 3 = low level of evidence; 4 = lowest level of evidence.

TABLE 3. Recommended meningococcal conjugate vaccination schedule and intervals for HIV-infected persons — Advisory Committee on Immunization Practices, United States, 2016

Age group	Recommended schedule and intervals
Primary vaccination	
<2 years	4 doses of MenACWY-CRM (Menveo)* at ages 2, 4, 6, and 12–15 months 2 doses of MenACWY-D (Menactra) at age 9–23 months, 12 weeks apart†,§,¶
≥2 years	2 doses of MenACWY-D or MenACWY-CRM, 8–12 weeks apart†,¶
Booster dose	
<7 years at previous dose	Additional dose of MenACWY-D or MenACWY-CRM 3 years after primary series; boosters should be repeated every 5 years thereafter**
≥7 years at previous dose	Additional dose of MenACWY-D or MenACWY-CRM 5 years after primary series; boosters should be repeated every 5 years thereafter

* MenACWY-CRM is licensed for use in persons aged 2 months through 55 years. Children aged 7 through 23 months who initiate vaccination with MenACWY-CRM should receive 2 doses 12 weeks apart, with the second dose administered after the first birthday. Source: Food and Drug Administration. Menveo U.S. package insert. http://www.fda.gov/downloads/BiologicsBloodVaccines/Vaccines/ApprovedProducts/UCM201349.pdf.

† MenACWY-D is licensed for use in persons aged 9 months through 55 years. Source: Food and Drug Administration (FDA). Menactra U.S. package insert. http://www.fda.gov/downloads/BiologicBloodVaccines/Vaccines/ApprovedProducts/UCM131170.pdf.

§ If MenACWY-D is used, it should be administered at least 4 weeks after completion of all pneumococcal conjugate vaccine doses.

¶ If MenACWY-D is to be administered to a child at increased risk for meningococcal disease, including children with HIV infection, it is recommended that MenACWY-D be given either before DTaP or concomitantly with DTaP.

** If the most recent dose was received before age 7 years, a booster dose should be administered 3 years later.

vaccinated with 1 dose of meningococcal conjugate vaccine should receive a booster dose at the earliest opportunity, provided at least 8 weeks have elapsed since the previous dose, and then continue to receive boosters at the appropriate interval throughout life.§ The recommendations for children aged 2 months through 2 years and persons aged ≥25 years are based on expert opinion; the vaccine was not studied in HIV-infected persons in these age groups. On the basis of available data and expert opinion, either MenACWY-CRM or MenACWY-D may be used in HIV-infected persons.

The same vaccine product should be used for all doses. However, if the product used for previous doses is unknown or unavailable, the vaccination series may be completed with any age- and formulation-appropriate meningococcal conjugate vaccine. Although no data on interchangeability of meningococcal conjugate vaccines in HIV-infected persons are available, limited data from a postlicensure study in healthy adolescents suggests safety and immunogenicity of MenACWY-CRM are not adversely affected by prior immunization with MenACWY-D (*1,11*).

ACIP recommends that HIV-infected infants aged 2 through 23 months receive MenACWY-CRM. HIV-infected children should not receive MenACWY-D before age 2 years, similar to the recommendation for children with functional or anatomic asplenia. Previously, children with functional or anatomic asplenia were recommended to receive 13-valent pneumococcal conjugate vaccine (PCV13) according to the normal schedule but to delay MenACWY-D vaccination until age 2 years because of immune interference (*1*,*12*). Because MenACWY-CRM does not demonstrate immune interference with 7-valent pneumococcal conjugate vaccine (PCV7) after the 12-month dose (*13*–*15*), MenACWY-CRM can be administered concomitantly with PCV13.

In addition, new data suggest the potential for immunologic interference in the meningococcal human complement serum bactericidal assay (hSBA) responses when MenACWY-D is administered 30 days after Daptacel (diphtheria and tetanus toxoids and acellular pertussis vaccine [DTaP], Sanofi Pasteur) (*16*). In one study among children aged 4 through 6 years, the hSBA responses to all four meningococcal serogroups failed to meet noninferiority criteria when MenACWY-D was administered 30 days after Daptacel. In contrast, co-administration of MenACWY-D and Daptacel was not associated with reduced hSBA responses to all four meningococcal serogroups. The study objectives did not include evaluation of the potential for interference that other DTaP containing vaccines might have on meningococcal seroresponse rates (*16*). If MenACWY-D is to be administered to a child at increased risk for meningococcal disease, including children who are HIV-infected, it is recommended that MenACWY-D be given either before or concomitantly with DTaP.

MenACWY is recommended for HIV-infected persons aged ≥56 years because of the need for revaccination (i.e., booster doses). Meningococcal polysaccharide vaccine (MPSV4, Menomune, Sanofi Pasteur) is the only licensed meningococcal vaccine for adults aged ≥56 years; however, no data are available on use of MPSV4 in HIV-infected adults. For healthy adults who have received MenACWY previously, limited data demonstrate a higher antibody response after a subsequent dose of MenACWY compared with a subsequent dose of MPSV4 (*1*).

To date, no randomized, controlled clinical trials have been conducted to evaluate use of MenACWY vaccines in pregnant or lactating women. Pregnancy should not preclude indicated vaccination with MenACWY.

§ If the most recent dose was received before age 7 years, a booster dose should be administered 3 years later. If the most recent dose was received at age ≥7 years, a booster dose should be administered 5 years later.

Summary

What is currently recommended?

The Advisory Committee on Immunization Practices (ACIP) currently recommends routine vaccination with meningococcal conjugate vaccine for all adolescents and for certain groups of persons at increased risk for meningococcal disease: persons who have persistent complement component deficiencies; persons who have anatomic or functional asplenia; microbiologists who routinely are exposed to isolates of Neisseria meningitidis; persons identified to be at increased risk because of a meningococcal disease outbreak attributable to serogroup A, C, W, or Y; military recruits; first-year college students living in residence halls; and persons who travel to or reside in areas in which meningococcal disease is hyperendemic or epidemic. In addition, ACIP recommends routine vaccination with serogroup B meningococcal (MenB) vaccine for persons who have persistent complement component deficiencies; persons who have anatomic or functional asplenia; microbiologists who routinely are exposed to isolates of N. meningitidis; and persons identified to be at increased risk because of a serogroup B meningococcal disease outbreak.

Why are the recommendations being modified now?

A growing body of evidence supports an increased risk for meningococcal disease in human immunodeficiency virus (HIV)–infected persons. The evidence supporting the use of meningococcal conjugate vaccines in HIV-infected persons was evaluated using the Grading of Recommendations, Assessment, Development, and Evaluation (GRADE) framework.

What are the new recommendations?

All HIV-infected persons aged ≥2 months should routinely receive meningococcal conjugate vaccine; children aged <2 years should be vaccinated using a multidose schedule. Persons aged ≥2 years with HIV who have not been previously vaccinated should receive a 2-dose primary series of meningococcal conjugate vaccine. Persons with HIV who have been previously vaccinated with meningococcal conjugate vaccine should receive a booster dose at the earliest opportunity (at least 8 weeks after the previous dose) and then continue to receive boosters at the appropriate intervals. If the most recent dose was received before age 7 years, a booster dose should be administered 3 years later. If the most recent dose was received at age ≥7 years, a booster should be administered 5 years later and every 5 years thereafter throughout life.

Precautions and Contraindications

Before administering meningococcal conjugate vaccines, health care providers should consult the package insert for precautions, warnings, and contraindications (*13*,*16*). Adverse events occurring after administration of any vaccine should be reported to the Vaccine Adverse Event Reporting System (VAERS). Reports can be submitted to VAERS online, by fax, or by mail. Additional information about VAERS is available by telephone (1-800-822-7967) or online (https://vaers.hhs.gov).

Acknowledgments

ACIP members (the membership roster for July 2015–June 2016 is available at http://www.cdc.gov/vaccines/acip/committee/members.html); ACIP Meningococcal Vaccine Work Group.

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Progress Toward Poliomyelitis Eradication — Afghanistan, January 2015–August 2016

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Only 74 cases of wild poliovirus (WPV) were reported globally in 2015, the lowest number of cases ever reported worldwide (*1*,*2*). All of the reported cases were WPV type 1 (WPV1), the only known WPV type still circulating; WPV type 2 has been eradicated, and WPV type 3 has not been detected since November 2012 (*1*). In 2015 in Afghanistan, WPV detection also declined from 2014, and trends observed in 2016 suggest that circulation of the virus is limited to a few localized areas. Despite the progress, there are concerns about the ability of the country's Polio Eradication Initiative (PEI) to meet the goal of interrupting endemic WPV transmission by the end of 2016 (*3*). The deteriorating security situation in the Eastern and Northeastern regions of the country considerably limits the ability to reach and vaccinate children in these regions. Furthermore, because of frequent population movements to and from Pakistan, cross-border transmission of WPV1 continues (*4*). Although the national PEI has taken steps to improve the quality of supplementary immunization activities (SIAs),* significant numbers of children living in accessible areas are still being missed during SIAs, and routine immunization services remain suboptimal in many parts of the country. This report describes polio eradication activities and progress in Afghanistan during January 2015–August 2016 and updates previous reports (*5*,*6*). During 2015, a total of 20 WPV1 cases were reported in Afghanistan, compared with 28 cases in 2014; eight cases were reported during January–August 2016, compared with nine cases reported during the same period in 2015. To achieve interruption of poliovirus transmission in Afghanistan, it is important that the 2016–2017 National Emergency Action Plan† for polio eradication be systematically implemented, including 1) improving the quality of SIAs and routine immunization services, 2) ensuring ongoing dialogue between PEI leaders and local authorities, 3) adopting innovative strategies for reaching children in security-compromised and inaccessible areas, and 4) strengthening cross-border coordination of polio vaccination and surveillance activities with Pakistan.

Immunization Activities

Estimated national routine vaccination coverage of infants with 3 doses of oral poliovirus vaccine (OPV3) in Afghanistan increased from 75% in 2014 to 77% in 2015 (*7*). The proportion of nonpolio acute flaccid paralysis (NPAFP)§ cases among children aged 6–23 months who were reported to have received ≥3 OPV doses through routine immunization services (a proxy indicator for routine OPV3 coverage) was 65% nationally in 2015, with percentages ranging from 40% in the Southern Region to 88% in the northern province of Badakhshan. The proportion of children aged 6–23 months with NPAFP who had never received OPV either through routine immunization services or SIAs (i.e., "zero-dose" children) was approximately 1% nationally in 2015.

During January 2015–August 2016, house-to-house SIAs in Afghanistan targeted children aged <5 years, using trivalent (types 1, 2, and 3), bivalent (types 1 and 3), and monovalent (type 1) OPV. During this period, 28 SIAs were conducted using OPV, including seven national immunization days (NIDs), six subnational immunization days (SNIDs), and 15 short-interval, additional dose, case-response vaccination campaigns.¶ In addition, SIAs using injectable inactivated poliovirus vaccine (IPV) were conducted in selected health districts in the Southern and Eastern regions where children were at high risk for poliovirus transmission. Children aged <10 years entering the country from Pakistan were vaccinated at major transit points and border crossings, and SIAs were conducted in camps for displaced persons.

The worsening security situation in the Eastern and Northeastern regions of the country has imposed considerable limitations on the ability to reach and vaccinate children in these areas. Estimates of children living in inaccessible areas** in the Eastern Region ranged from 22,938 to 131,781 during February–August 2016. An estimated 165,333 children could

* Mass campaigns conducted for a brief period (days to weeks) in which 1 dose of oral poliovirus vaccine is administered to all children aged <5 years, regardless of vaccination history. Campaigns can be conducted nationally or subnationally (in portions of the country).

† http://polioeradication.org/wp-content/uploads/2016/07/4.2_14IMB.pdf.

§ Vaccination histories of children aged 6–23 months with acute flaccid paralysis who do not test WPV-positive are used to estimate OPV coverage of the overall target population and to corroborate national reported routine vaccination coverage estimates.

¶ Short-interval, additional dose campaigns are used for case-response vaccination after detection of a WPV case or during negotiated periods of nonviolence in otherwise inaccessible areas, to provide 2 doses of monovalent or bivalent OPV within 1–2 weeks.

** Areas where vaccination teams are temporarily unable to operate because of security concerns or local bans on vaccination.

not be reached in the Northeastern Region during the May 2016 NIDs. The majority of these children live in the province of Kunduz, where conflict has intensified over the past year. Taken together, the Eastern and Northeastern regions account for >350,000 children who cannot consistently be reached, representing almost 3% of the national target population of approximately 9.5 million children aged <5 years. Intermittent bans on polio SIAs in the Southern Region have also hindered access to children. Despite the constraints of inaccessibility, data indicate that the majority of missed children live in areas that are accessible for vaccination activities. Postcampaign monitoring data from the May and August 2016 NIDs suggest that up to 55% of missed children lived in areas that were accessible during the campaigns.

Lot quality assurance sampling (LQAS),[††] which is used to assess the quality of SIAs (*8*), indicates that there were improvements in the quality of SIAs in 2016, compared with SIAs conducted during 2015. For example, the number of lots (health districts) rejected at the pass threshold of ≥80% in the 47 very high-risk districts[§§] decreased from 40% in November 2015 to 17% in May 2016. Considering all assessed districts without regard to risk status, LQAS performance at the threshold of ≥80% improved from 68% in January 2016 to 78% in April 2016.

Poliovirus Surveillance

Acute flaccid paralysis (AFP) surveillance.[¶¶] In 2015, the annual national NPAFP rate was 13.8 per 100,000 children aged <15 years (regional range = 9.8–19.2) (Table). The percentage of AFP cases for which adequate stool specimens were collected was 93% (regional range = 84%–97%). Three AFP cases were classified as polio-compatible, including two cases from Farah Province in the Western Region and one case from Nimroz Province in the Southern Region.

Environmental surveillance. Supplemental surveillance for polioviruses through sewage sampling began in Afghanistan in September 2013. Environmental surveillance is being conducted at 14 sites in five provinces (Kandahar and Helmand in the Southern Region, Nangarhar and Kunar in the Eastern Region, and Kabul City in the Central Region). WPV1 was first isolated from sewage samples in July 2014. Since then, 37 specimens from 11 sites were positive for WPV1. Nineteen (13%) of 148 sewage specimens tested positive for WPV1 in 2015. WPV1 was most recently detected in sewage samples taken from Nangarhar Province in December 2015. To date, none of the 112 specimens collected in 2016 have tested positive.

[††] A rapid survey method used to assess the quality of vaccination activities after SIAs in predefined areas, such as health districts (referred to as "lots"), using a small sample size. Lot quality assurance sampling involves dividing the population into "lots" and randomly selecting persons in each lot. If the number of unvaccinated persons in the sample exceeds a predetermined value, then the lot is classified as having an unsatisfactory level of vaccination coverage, and mop-up activities are recommended. If the threshold of ≥80% is met, the area/district is classified as having "passed," although mop-up activities might still be indicated in certain areas

[§§] Defined in November 2012, districts with confirmed polio cases in the previous 2 years, or confirmed polio cases in 1 of the previous 2 years, plus one of the following: reported "zero-dose" NPAFP cases in the previous 2 years; <90% estimated OPV coverage in the previous two SIAs; failed LQAS in more than one round of vaccination campaigns; average level of community awareness of SIAs <50% in previous two SIAs; and inaccessibility.

[¶¶] The quality of AFP surveillance is monitored by performance indicators that include 1) the detection rate of NPAFP cases and 2) the proportion of AFP cases with adequate stool specimens. World Health Organization (WHO) operational targets for countries with endemic poliovirus transmission are an NPAFP detection rate of ≥2 cases per 100,000 population aged <15 years and adequate stool specimen collection from ≥80% of AFP cases, in which two specimens are collected ≥24 hours apart, both within 14 days of paralysis onset, and shipped on ice or frozen packs to a WHO-accredited laboratory, arriving in good condition (without leakage or desiccation).

TABLE. Acute flaccid paralysis (AFP) surveillance indicators and reported cases of wild poliovirus (WPV), by region and period — Afghanistan, January 2015–August 2016*

	AFP surveillance indicators (2015)			No. of WPV cases reported		
Region of Afghanistan	No. of AFP cases	Rate of nonpolio AFP[†]	% of AFP cases with adequate specimens[§]	Jan–Jun 2015	Jul–Dec 2015	Jan–Aug 2016
All regions	**2,718**	**13.8**	**93**	**6**	**14**	**8**
Badakhshan	57	10.2	97	0	0	0
Northeastern	281	12.8	94	0	0	0
Northern	343	13.9	91	0	1	0
Central	439	9.8	96	0	0	0
Eastern	374	19.2	95	0	10	4
Southeastern	209	10.8	97	0	0	2
Southern	567	16.4	84	1	3	2
Western	448	17.2	95	5	0	0

* Data as of August 31, 2016.
† Per 100,000 children aged <15 years.
§ Two specimens collected ≥24 hours apart, both within 14 days of paralysis onset, and shipped on dry ice or frozen packs to a World Health Organization–accredited laboratory, arriving in good condition (without leakage or desiccation).

Epidemiology of WPV and Vaccine-Derived Poliovirus (VDPV)

Twenty WPV1 cases were reported in Afghanistan in 2015, compared with 28 cases in 2014. Eight WPV1 cases were reported during January–August 2016, compared with nine during January–August 2015 (Figure 1) (Figure 2). WPV1 cases were reported from 16 (4%) of the 399 districts in Afghanistan in 2015 and from four (1%) districts as of August 31 in 2016. The Eastern Region accounted for half of the 20 WPV cases reported in 2015, and 25% of the cases were reported from the Western Region, including four cases from Farah Province and one from Hirat Province. Four of the remaining five WPV cases reported in 2015 were reported from the Southern Region (two cases from Helmand Province and one each from Kandahar and Nimroz provinces); Faryab Province in the Northern Region reported a single polio case. Among the eight WPV cases reported in 2016 as of August 31, four were from the Eastern Region, all from the district of Shigal Wa Sheltan in Kunar Province. The Southern Region has accounted for two cases (one each from Helmand and Kandahar provinces), and the remaining two cases were reported from Paktika Province in the Southeastern Region. Among the 28 WPV1 cases reported during January 2015–August 2016, children aged <36 months accounted for 20 (71%) cases. Among these 20 children, 11 (55%) had never received OPV, two (10%) had received only 2 doses, one (5%) had received 3 doses, and six (30%) had received ≥4 doses. All eight WPV1 cases reported in 2016 were in children who had never received OPV through routine immunization services, with ages ranging from 12 to 59 months.

Genetic patterns of WPV1 isolates identified during January 2015–August 2016 indicate localized circulation within areas with endemic transmission, including Kunar, Kandahar, and Helmand, and evidence of cross-border transmission between districts in the Eastern Region of Afghanistan and northwest Pakistan. No polio cases attributable to WPV type 3 or circulating vaccine-derived poliovirus (cVDPV)*** have been detected in Afghanistan since April 2010 and March 2013, respectively.

Discussion

Signs of progress toward polio eradication in Afghanistan during 2015–2016 include a decline in overall WPV1 incidence, a narrowing of the geographic distribution of cases, and decreased diversity of WPV1 isolates. However, persistent poliovirus circulation in the country's core poliovirus reservoirs in the Eastern and Southern regions and the emergence of sporadic cases elsewhere highlight the need for urgent action by the country's PEI to address program vulnerabilities.

*** VDPVs can cause paralytic polio in humans and have the potential for sustained circulation. VDPVs resemble WPVs biologically and differ from the majority of Sabin vaccine–related poliovirus isolates by having genetic properties consistent with prolonged replication or transmission.

FIGURE 1. Number of cases of wild poliovirus type 1 (WPV1) and circulating vaccine-derived poliovirus type 2 (cVDPV2), by month and year of paralysis onset — Afghanistan, 2013–2016

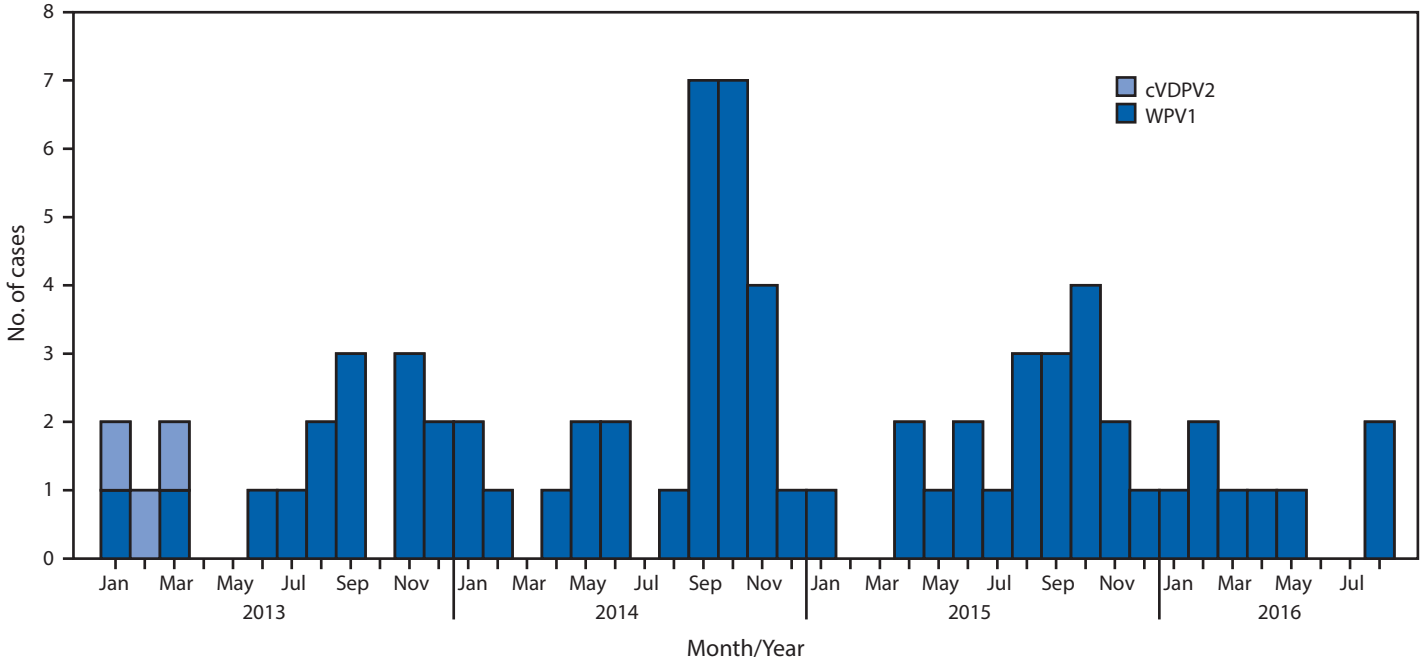
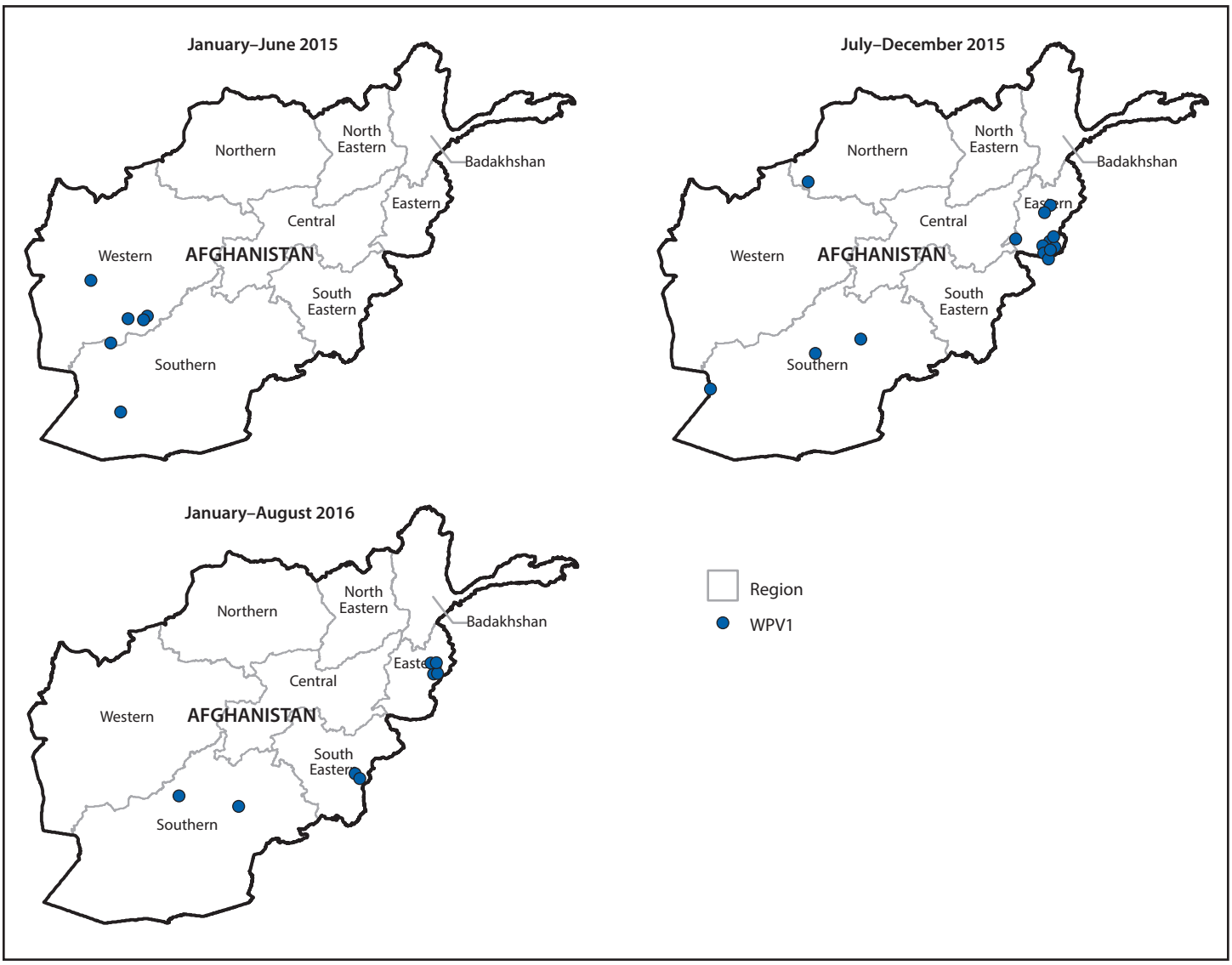


FIGURE 2. Cases of wild poliovirus type 1 (WPV1), by region — Afghanistan, January 2015–August 2016*



* Each dot represents one case. Dots are randomly placed within regions.

In the Eastern Region, a surge in WPV1 cases in Nangarhar Province during July–October 2015 was followed by sustained transmission in Kunar Province. Genetic sequencing linked some cases in the Eastern Region to active cross-border transmission, but it also indicated sustained local transmission. The Southern Region continues to show encouraging signs of reduced virus circulation, with only a few cases reported in 2015 and just a single case each reported from Helmand and Kandahar provinces to date in 2016; however, inadequate vaccination campaign quality persists. Sporadic polio cases in previously polio-free areas, such as Paktika in the Southeastern Region and Faryab Province in the Northern Region, represent importations from other parts of the country, and raise concerns about gaps in population immunity.

The establishment of emergency operations centers (EOCs) at the national level and in the Eastern, Southern, and Western regions has strengthened the management and coordination of polio eradication activities. A key focus of the EOCs has been rapid improvement in the quality of SIAs. To achieve this, several steps were taken by the country's PEI, notably updating the list of high-risk districts, and reprioritizing 47 of these districts as very high-risk districts. Microplans in these districts were revised and are being updated before each SIA to analyze local immunization data, prepare an operational map to reach and vaccinate children, and identify special activities for hard-to-reach areas. In addition, frontline polio workers in all districts were trained using a revised training package,

Summary

What is already known about this topic?

Afghanistan is one of three countries where indigenous wild poliovirus (WPV) transmission has never been interrupted. The Eastern and Southern regions have been the main areas in Afghanistan with endemic WPV transmission. The last case of WPV type 3 was reported in November 2012, and only WPV type 1 has been detected globally since then. WPV type 2 has been eradicated, with the last case occurring in 1999.

What is added by this report?

The number of WPV type 1 cases reported in Afghanistan in 2015 declined by 29% from levels reported in 2014, indicating progress toward the eradication of polio in the past year. The establishment of national and regional emergency operations centers has led to some improvements in the quality of immunization activities; however, WPV continues to circulate in the Eastern and Southern regions, and sporadic cases are being reported from previously polio-free areas. Worsening conflict in the Eastern and Northeastern regions is imposing significant constraints on immunization activities in both regions, and cross-border transmission of polio to and from Pakistan remains unresolved.

What are the implications for public health practice?

Afghanistan faces considerable challenges in its quest to eliminate indigenous poliovirus transmission by the end of the year. To address these challenges, it is important that leaders of the national Polio Eradication Initiative act with a sense of urgency to implement the revised National Emergency Action Plan, leveraging the assets of the emergency operations centers to improve the quality of polio immunization activities. In addition, the worsening security situation in parts of the country calls for renewed negotiations with local authorities and innovative approaches to gain access to vaccinate children in such areas.

and the scope of the fifth-day revisit strategy for vaccinating children missed during earlier days of polio campaigns was expanded. Postcampaign monitoring and LQAS results indicate that these initiatives have yielded improvements in the quality of vaccination campaigns. However, substantial numbers of children are still being missed in accessible areas, indicating the need for further improvement in the quality of supervision and monitoring during SIAs.

There is an urgent need to improve vaccination coverage among children living in areas with security and access limitations. Key strategies already employed to achieve this, such as continued dialogue between the PEI and local authorities to gain access, the use of permanent transit teams to target and vaccinate children at all transit points close to inaccessible areas and cross-border points, and the implementation of several IPV-OPV rounds in newly accessible areas in short succession, need to be continued and scaled up wherever needed. Cross-border coordination of immunization activities and surveillance with neighboring Pakistan must remain a top priority, and immunization activities of the two countries should be synchronized whenever feasible.

Despite recent progress, Afghanistan faces significant constraints in its quest to eliminate WPV circulation by the end of 2016, notably inaccessibility and attendant gaps in population immunity. To address these challenges, it is important that priority be given to ensuring timely implementation of all elements of the updated National Emergency Action Plan.

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Becky Maholland, Office of Public Health Preparedness and Response, CDC; World Health Organization Global Polio Laboratory Network.

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Notes from the Field

Rift Valley Fever Response — Kabale District, Uganda, March 2016

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On March 9, 2016, a male butcher from Kabale District, Uganda, aged 45 years, reported to the Kabale Regional Referral Hospital with fever, fatigue, and headache associated with black tarry stools and bleeding from the nose. One day later, a student aged 16 years from a different sub-county in Kabale District developed similar symptoms and was admitted to the same hospital. The student also had a history of contact with livestock. Blood specimens collected from both patients were sent for testing for Marburg virus disease, Ebola virus disease, Rift Valley fever (RVF), and Crimean Congo Hemorrhagic fever at the Uganda Virus Research Institute, as part of the viral hemorrhagic fevers surveillance program. The Uganda Virus Research Institute serves as the national viral hemorrhagic fever reference laboratory and hosts the national surveillance program for viral hemorrhagic fevers, in collaboration with the CDC Viral Special Pathogens Branch and the Uganda Ministry of Health.

On March 11, the blood specimens from both patients were found to be positive for RVF by reverse transcription–polymerase chain reaction and RVF immunoglobulin M testing. These two cases were the first confirmed cases of RVF detected in Uganda since 1968 (*1*). Concurrently, probable cases of RVF were identified in Kabale District; both of the patients in these cases died. Both patients had RVF symptoms and a history of contact with animals through animal care and butchering, but specimens were not obtained from these patients for testing before they died.

RVF virus belongs to the Bunyaviridae family, genus *Phlebovirus* (*2*). RVF outbreaks in animals are characterized by a large number of spontaneous abortions in pregnant animals (*3*). Human cases typically occur after RVF disease in animals. In humans, RVF symptoms can range from an asymptomatic or mild influenza-like illness to a severe disease with hepatitis, retinitis, or encephalitis (*4*). Approximately 1% of RVF cases progress to hemorrhagic disease. RVF transmission to humans can occur through direct contact with an animal's infected tissue or body fluid, particularly during spontaneous abortion of an infected animal, as well as via fomites. Mosquitoes also transmit the virus to humans and animals (*3*). Populations at high risk for infection include herdsmen, butchers, and abattoir workers (*5*). Previous large RVF outbreaks have occurred throughout Africa and the Arabian Peninsula, recently including South Africa, Kenya, Sudan, Saudi Arabia, Tanzania and Yemen (*3*).

Prevention of RVF includes measures such as recognition of sick animals to avoid spread to other animals or humans, use of personal protective equipment, and thorough cooking of meat and milk before consumption. Use of mosquito nets and wearing long sleeves and pants can help prevent transmission by mosquitoes. After this RVF outbreak in March 2016, a multidisciplinary investigation was initiated in coordination with the Uganda Ministry of Health, Uganda Ministry of Agriculture Animal Industry and Fisheries, the Uganda Virus Research Institute, and CDC. Surveillance for RVF in mosquito and animal populations in Kabale and neighboring districts, as well as a knowledge, attitudes, and practices survey for persons living in the region, are underway. In addition, a multisectoral national task force was organized to train health care workers on recognition of RVF signs and symptoms in humans and animals. The task force also began a social mobilization and health information campaign in Kabale and surrounding districts to increase RVF awareness through community discussions, radio messaging, and informational pamphlets developed by the World Health Organization, as well as distribution of informational posters targeting community members, health care providers, veterinarians, farmers/herdsmen, and abattoir workers.

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Announcements

National Diabetes Month — November 2016

November is National Diabetes Month. In the United States, 29 million persons have diabetes and 86 million adults have prediabetes, putting them at risk for developing type 2 diabetes, heart disease, and stroke (*1*). Approximately 28% of those with diabetes are undiagnosed (*1*), and 89% of those with prediabetes do not know they have it (*2*). Type 2 diabetes, which accounts for 90%–95% of all cases of diagnosed diabetes in the United States, can be prevented through lifestyle changes, such as weight loss, healthy eating, and increased physical activity (*1*,*3*). Persons with diabetes can take steps to control the disease and prevent complications (*1*,*4*).

CDC and partners play a crucial role in delaying or preventing type 2 diabetes, preventing diabetes complications, and improving the health and quality of life for all persons with diabetes. Good Health and Wellness in Indian Country is a CDC partnership (http://www.cdc.gov/chronicdisease/pdf/ghwic-aag.pdf) that supports a coordinated and holistic approach to healthy living and chronic disease prevention for American Indians and Alaska Natives (AIAN). AIAN are twice as likely as non-Hispanic whites to have diagnosed diabetes (*1*); AIAN also experience higher death rates from diabetes and other chronic diseases (*5*).

The U.S. Diabetes Surveillance System provides a Diabetes Atlas, (http://gis.cdc.gov/grasp/diabetes/DiabetesAtlas.html), which allows users to view the latest state-level data and trends on any mobile device. The new Diabetes At A Glance fact sheet (http://www.cdc.gov/chronicdisease/resources/publications/aag/pdf/2016/diabetes-aag.pdf) provides comprehensive information about diabetes, including risk factors, complications, and the financial costs of living with diabetes. More information about diabetes prevention and control is available at http://www.cdc.gov/diabetes. Additional information about preventing diabetes complications is available at http://www.cdc.gov/features/preventing-diabetes-complications/index.html.

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Drowsy Driving Prevention Week — November 6–13, 2016

Drowsy Driving Prevention Week, the National Sleep Foundation's annual campaign to educate the public about the hazards of driving while sleepy, will be observed November 6–13, 2016. A report released by the AAA Foundation for Traffic Safety in 2014 concluded that drowsy drivers were involved in an estimated 21% of fatal crashes, based on a nationally representative sample of motor vehicle crashes during 2009–2013 (*1*).

Drivers who work the night shift, work multiple jobs, or have irregular work schedules are at increased risk for motor vehicle crashes caused by drowsy driving (*2*). These groups are also more likely to report sleeping <7 hours per day (*3*): 70% of night shift workers in the transportation and warehousing industry reported less than 7 hours of sleep per night (*3*), compared with 30% of all adult U.S. workers.

General information about drowsy driving is available from the National Sleep Foundation (http://drowsydriving.org/). Information for shift workers regarding how to improve their sleep and reduce their risk for drowsy driving also is available online (http://www.cdc.gov/niosh/topics/workschedules/).

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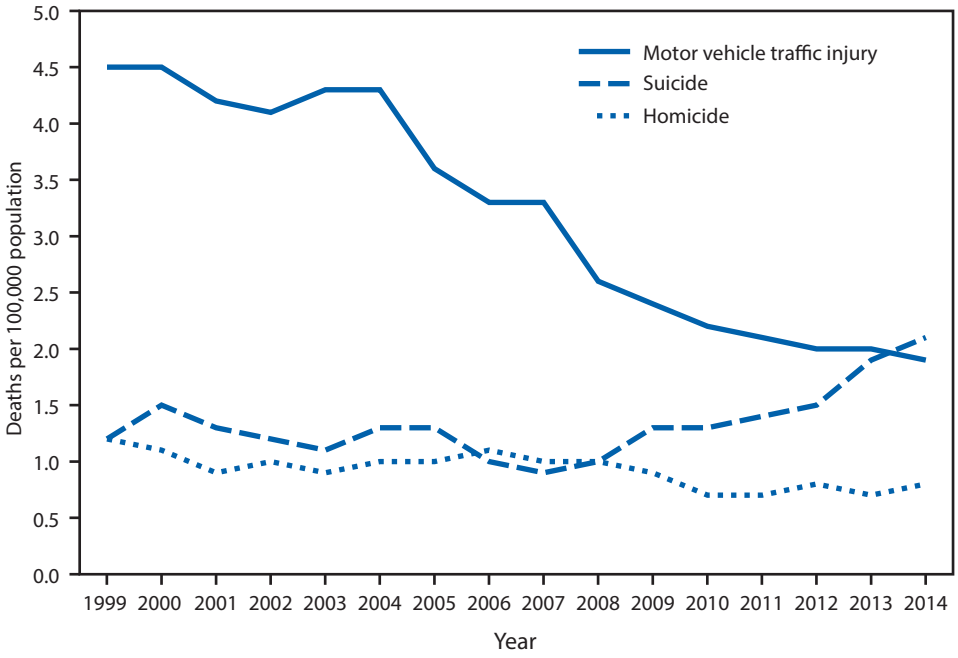
QuickStats

FROM THE NATIONAL CENTER FOR HEALTH STATISTICS

Death Rates for Motor Vehicle Traffic Injury,* Suicide,† and Homicide§ Among Children and Adolescents aged 10–14 Years — United States, 1999–2014



* Motor vehicle traffic injuries are identified with *International Classification of Disease, 10th Revision (ICD-10)* codes V02-V04[.1,.9],V09.2,V12-V14[.3-.9],V19[.4-.6],V20-V28[.3-.9],V29-V79[.4-.9],V80[.3-.5],V81.1,V82.1,V83-V86[.0-.3],V87[.0-.8],V89.2). All motor vehicle traffic injuries are unintended.
† Suicides are identified with *ICD-10* codes U03, X60-X84, and Y87.0.
§ Homicides are identified with *ICD-10* codes U01-U02, X85-Y09, and Y87.1.

In 1999, the mortality rate for children and adolescents aged 10–14 years for deaths from motor vehicle traffic injury (4.5 per 100,000) was about four times higher than the rate for deaths for suicide and homicide (both at 1.2). From 1999 to 2014, the death rate for motor vehicle traffic injury declined 58%, to 1.9 in 2014 (384 deaths). From 1999 to 2007, the death rate for suicide fluctuated and then doubled from 2007 (0.9) to 2014 (2.1, 425 deaths). The death rate for homicide gradually declined to 0.8 in 2014. In 2013 and 2014, the differences between death rates for motor vehicle traffic injury and suicide were not statistically significant.

Source: CDC/NCHS, National Vital Statistics System, Mortality Data; http://www.cdc.gov/nchs/nvss/deaths.htm.

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For more information on this topic, CDC recommends the following link: https://www.cdc.gov/injury/.

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