Trends in Tuberculosis — United States, 2013

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World TB Day — March 24, 2014

Each year, World TB Day is observed on March 24. This annual event commemorates the date in 1882 when Dr. Robert Koch announced his discovery of Mycobacterium tuberculosis, the bacillus that causes tuberculosis (TB). World TB Day provides an opportunity to raise awareness about TB-related problems and solutions and to support worldwide TB control efforts. For 2014, CDC selected the theme “Find TB. Treat TB. Working together to eliminate TB.” Health officials in local and state TB programs are encouraged to reach out to their communities to raise awareness about TB and partner with others who are caring for those most at risk for TB. Everyone has a role in ensuring that one day TB will be eliminated.

In 2013, a total of 9,588 new TB cases were reported in the United States, for a rate of 3.0 cases per 100,000 population, a decrease of 4.2% from 2012 (1). This report summarizes provisional TB surveillance data reported to CDC in 2013. Although case counts and incidence rates continue to decline, challenges remain that slow progress toward the goal of TB elimination in the United States. TB still persists at greater incidence rates in specific populations. Foreign-born persons and racial/ethnic minorities continue to be affected disproportionately.

CDC is committed to a world free of TB. Initiatives to improve awareness, testing, and treatment of latent TB infection and TB disease among high-risk groups are critical to reaching the goal of TB elimination in the United States. Additional information about World TB Day and CDC’s TB elimination activities is available at http://www.cdc.gov/tb/events/worldtbday.

Reference


In 2013, a total of 9,588 new tuberculosis (TB) cases were reported in the United States, with an incidence rate of 3.0 cases per 100,000 population, a decrease of 4.2% from 2012 (1). This report summarizes provisional TB surveillance data reported to CDC in 2013. Although case counts and incidence rates continue to decline, certain populations are disproportionately affected. The TB incidence rate among foreign-born persons in 2013 was approximately 13 times greater than the incidence rate among U.S.-born persons, and the proportion of TB cases occurring in foreign-born persons continues to increase, reaching 64.6% in 2013. Racial/ethnic disparities in TB incidence persist, with TB rates among non-Hispanic Asians almost 26 times greater than among non-Hispanic whites. Four states (California, Texas, New York, and Florida), home to approximately one third of the U.S. population,
accounted for approximately half the TB cases reported in 2013. The proportion of TB cases occurring in these four states increased from 49.9% in 2012 to 51.3% in 2013. Continued progress toward TB elimination in the United States will require focused TB control efforts among populations and in geographic areas with disproportionate burdens of TB.

Health departments in the 50 states and the District of Columbia electronically report to CDC verified cases of disease that meet the CDC and Council of State and Territorial Epidemiologists surveillance case definition for TB. Reports include the patient’s country of origin, self-identified race and ethnicity (i.e., Hispanic or non-Hispanic), information on risk factors (e.g., homelessness and incarceration), human immunodeficiency virus (HIV) status, and drug-susceptibility test results. CDC calculates national and state TB incidence rates overall and by racial/ethnic group, using U.S. Census Bureau population estimates. The Current Population Survey provides the population denominators used to calculate TB incidence rates and percentage changes according to national origin.† For TB surveillance, a U.S.-born person is defined as a person born in the United States or its associated jurisdictions,§ or a person born in a foreign country but having at least one U.S.-citizen parent. In 2013, the country of birth was unknown for 0.4% of patients, and race/ethnicity was unknown for 0.4%. In this report, persons of Hispanic ethnicity might be of any race; non–Hispanic persons are categorized as Asian, black, white, American Indian/Alaska Native, Native Hawaiian or other Pacific Islander, or of multiple races.

Compared with the national TB incidence rate of 3.0 cases per 100,000 population, the median incidence rate in reporting areas was 2.2 per 100,000 population, ranging from zero in Wyoming to 9.7 per 100,000 population in Alaska (Figure 1).

FIGURE 1. Rate* of tuberculosis cases, by state/area — United States, 2013†

![Map of the United States showing TB incidence rates by state/area.](image_url)
Thirty-three states had lower rates in 2013 than in 2012. Nine states and the District of Columbia had incidence rates higher than the national rate. In 2013, as in 2012, four states (California, Texas, New York, and Florida) reported more than 500 cases each. Combined, these four states accounted for 4,917 TB cases, 51.3% of all TB cases reported in 2013.

Among U.S.-born persons, the number and rate of TB cases decreased in 2013. The 3,377 TB cases reported among U.S.-born persons (35.4% of all cases with known national origin) were 7.6% fewer than the number reported in 2012 and 61.0% fewer than the number reported in 2000 (Figure 2). The rate of 1.2 per 100,000 population among U.S.-born persons is an 8.4% decrease since 2012 and a 64.7% decrease since 2000.

Among foreign-born persons in the United States, the number and rate of TB cases also decreased in 2013. A total of 6,172 TB cases were reported among foreign-born persons (64.6% of all cases in persons with known national origin), a 1.6% decrease since 2012 and a 19.0% decrease since 2000. The 15.6 cases per 100,000 population TB rate among foreign-born persons is a 2.1% decrease since 2012 and a 41.1% decrease since 2000. In 2013, 54.2% of foreign-born persons with TB and known country of birth originated from five countries: 1,233 (20.0%) from Mexico, 776 (12.6%) from the Philippines, 495 (8.0%) from India, 454 (7.4%) from Vietnam, and 377 (6.1%) from China.

The TB incidence rate among Asians was the highest among all racial/ethnic groups and was 25.9 times higher than the incidence rate among whites (Table). Although incidence rates among all racial/ethnic groups declined in 2013, the decrease was greater among whites (9.2%) and blacks (7.5%) than among Hispanics (5.3%) and Asians (0.3%). Among persons with TB, 95% of Asians, 75% of Hispanics, 40% of blacks, and 23% of whites were foreign-born. Among U.S.-born persons, the incidence rate among blacks was 6.2 times higher than among whites.

HIV status was known for 85% of TB cases reported in 2013, as in 2012. Among TB patients with known HIV status, 6.8% had a positive test result for HIV infection in 2013, compared with 7.4% in 2012.

Among persons aged ≥15 years with TB, 98.5% had known housing status, 5.7% of whom reported being homeless within the past year. Among persons aged ≥15 years with TB, 99.1% had a known incarceration status, 3.9% of whom were confined to a detention or correctional facility at the time of TB diagnosis.

A total of 86 cases of multidrug-resistant TB (MDR TB) were reported in 2012, the most

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**TABLE. Number and rate* of tuberculosis cases and percentage change, by race and ethnicity — United States, 2012 and 2013†**

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<td>43</td>
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<tr>
<td>Total</td>
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* Per 100,000 population.
† Data for 2013 are provisional.
§ Persons included in this category are American Indian/Alaska Native (2013, n = 125, rate = 5.4 per 100,000; 2012, n = 146, rate = 6.3 per 100,000); Native Hawaiian or other Pacific Islander (2013, n = 58, rate = 10.9 per 100,000; 2012, n = 63, rate = 12.1 per 100,000); and multiple race (2013, n = 151, rate = 2.4 per 100,000; 2012, n = 179, rate = 3.8 per 100,000).

recent year for which complete drug-susceptibility results are available. Drug-susceptibility test results for isoniazid and rifampin were reported for 97.9% and 97.6% of cases with culture results positive for *Mycobacterium tuberculosis* in 2011 and 2012, respectively. Among these cases, the percentage of MDR TB for 2012 (1.2% [86 of 7,426 cases]) decreased from the percentage in 2011 (1.6% [129 of 7,906 cases]). The percentage of MDR TB cases among persons without a previous history of TB (1.0%) and the percentage of MDR TB cases among persons with a previous history of TB (3.4%) were lower in 2012 than in 2011. Foreign-born persons accounted for 88.4% of MDR TB cases in 2012. Two cases of extensively drug-resistant TB** have been reported so far for 2013, compared with two cases in 2012 and five cases in 2011.

**Discussion**

Despite the continued decline in U.S. TB cases and rates since 1993, the goal of TB elimination in the United States (i.e., less than one case per 1,000,000) set in 1989 (3) remains unmet. Most states reported fewer cases of TB in 2013. However, elevated rates of TB in specific populations remain a major challenge that impedes progress toward TB elimination.

In 2013, four states (California, Texas, New York, and Florida) reported approximately half of the TB cases in the United States. Their TB burden is disproportionately greater after population adjustment, and their share of the national TB case count has increased, from 49.9% in 2012 to 51.3% in 2013. To continue to make significant progress toward TB elimination, TB control and prevention in the areas with the highest burden will have to continue to be given priority. One contributing factor to the geographic disparity is that these four states have populations at elevated risk for TB. In 2013, 16%–26% of the population in each of these four states was foreign-born (4). In addition, three of these states (California, New York, and Florida) were among the 15 states with the highest rates of homelessness in 2013 (5).

The rate of decline in TB incidence among foreign-born persons (2.1%) lagged behind the rate of decline among the U.S.-born (8.4%) in 2013, causing the proportion of TB cases in foreign-born persons to continue to increase. The majority of TB cases among foreign-born persons have been attributed to reactivation of TB infection acquired previously, with the rate reflecting TB incidence in their countries of origin (6). Further interventions aimed at diagnosing and treating latent TB infection (LTBI) among foreign-born persons are necessary to meet the goal of TB elimination in the United States.

Persons experiencing homelessness also present a challenge for TB control. During 2006–2010, the TB rate among persons experiencing homelessness was estimated to be 36–47 per 100,000 population, approximately 10 times greater than the overall national TB incidence during that period (7). In addition, recent outbreaks among persons experiencing homelessness have underscored the potential for transmission in homeless shelters (8,9). Effectively addressing TB among persons experiencing homelessness requires partnerships between TB control programs and homeless service providers to diagnose and treat active TB disease and LTBI in this population.

The findings in this report are subject to at least two limitations. First, this analysis is limited to reporting provisional case counts and incidence rates for 2013. Second, incidence rates are calculated based on estimated population denominators from 2013. CDC’s annual TB surveillance report, which is released in September of every year, will provide final TB case counts and incidence rates for 2013. Second, incidence rates are calculated based on estimated population denominators from 2013. CDC’s annual TB surveillance report, which is released in September of every year, will provide final TB case counts and incidence rates for 2013. Second, incidence rates are calculated based on estimated population denominators from 2013. CDC’s annual TB surveillance report, which is released in September of every year, will provide final TB case counts and incidence rates for 2013. Second, incidence rates are calculated based on estimated population denominators from 2013. CDC’s annual TB surveillance report, which is released in September of every year, will provide final TB case counts and incidence rates for 2013.

**What is already known on this topic?**

Tuberculosis (TB) incidence has been declining in the United States since 1993, but an increasing proportion of cases have been among foreign-born persons.

**What is added by this report?**

For 2013, preliminary data show the number of TB cases reported in the United States was 9,588, an incidence of 3.0 cases per 100,000 population, compared with 3.2 cases per 100,000 population in 2012. Four states (California, Texas, New York, and Florida) reported more than half (51.3%) of all TB cases reported in 2013. Although TB cases among foreign-born persons in the United States continued to decline, the rate of decline in TB incidence since 2012 among foreign-born persons (2.1%) lagged behind the rate of decrease among the U.S.-born (8.4%), causing the proportion of TB cases in foreign-born persons to continue to increase.

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

Ongoing surveillance, vigilance, and prevention activities are needed despite the decline. Initiatives to improve awareness, testing, and treatment of TB disease as well as preventing TB by identifying and treating those with asymptomatic latent TB infection are needed to eliminate TB in the United States.

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**Defined by the World Health Organization as a case of TB in a person with an *M. tuberculosis* isolate with resistance to at least isoniazid and rifampin among first-line anti-TB drugs, resistance to any fluoroquinolone (e.g., ciprofloxacin or ofloxacin), and resistance to at least one second-line injectable drug (i.e., amikacin, capreomycin, or kanamycin). Additional information available at http://whqlibdoc.who.int/publications/2010/9789241599191_eng.pdf.**
to translate surveillance data into public health action will be key to achieving TB elimination.

Acknowledgments
State and local TB control officials.

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References
For more than two decades, as the number of tuberculosis (TB) cases overall in the United States has declined, the proportion of cases among foreign-born persons has increased. In 2013, the percentage of TB cases among those born outside the country was 64.6%. (1) To address this trend, CDC has developed strategies to identify and treat TB in U.S.-bound immigrants and refugees overseas. Each year, approximately 450,000 persons are admitted to the United States on an immigrant visa, and 50,000–70,000 are admitted as refugees. Applicants for either an immigrant visa or refugee status are required to undergo a medical examination overseas before being allowed to travel to the United States. CDC is the federal agency with regulatory oversight of the overseas medical examination, and panel physicians appointed by the U.S. Department of State perform the examinations in accordance with Technical Instructions (TI) provided by CDC’s Division of Global Migration and Quarantine (DGMQ). Beginning in 1991, the algorithm for TB TI relied on chest radiographs for applicants aged ≥15 years, followed by sputum smears for those with findings suggestive of TB; no additional diagnostics were used. In 2007, CDC issued enhanced standards for TB diagnosis and treatment, including the addition of sputum cultures (which are more sensitive than smears) as a diagnostic tool and treatment delivered as directly observed therapy (DOT). This report summarizes worldwide implementation of the new screening requirements since 2007. In 2012, the year for which the most recent data are available, 60% of the TB cases diagnosed were in persons with smear-negative, but culture-positive, test results. The results demonstrate that rigorous diagnostic and treatment programs can be implemented in areas with high TB incidence overseas.

2007 Technical Instructions

In 2007, CDC issued Technical Instructions for Tuberculosis Screening and Treatment Using Cultures and Directly Observed Therapy (CDOT TB TI).* Important changes included requiring 1) sputum cultures in addition to sputum smears; 2) tuberculin skin tests or interferon gamma release assays (beginning in 2009) for certain children aged 2–14 years examined in countries where the World Health Organization estimated TB incidence is ≥20 per 100,000 persons; 3) drug-susceptibility testing of positive isolates; and 4) treatment according to guidelines from the American Thoracic Society, CDC, and the Infectious Diseases Society of America (Figure). Treatment is delivered as DOT (a trained health-care professional administers and documents each dose) throughout the entire course.

Implementation of CDOT TB TI

CDC’s DGMQ initially targeted large-volume, high TB-incidence countries to implement CDOT TB TI. Within each screening country, DGMQ worked to develop the infrastructure for implementing sputum culture and DOT. As TB diagnostic and treatment capacities were being developed, DGMQ linked panel physicians with in-country TB programs. A training program for panel physicians was developed that used multiple training modalities, including onsite instruction, webinars, development of a panel physician website,† and regional training summits. To monitor and evaluate the implementation and effectiveness of the algorithm, representatives of the Advisory Council for the Elimination of Tuberculosis and the National Tuberculosis Controllers Association led evaluations at five large-volume screening sites (Thailand, Philippines, Nepal, Vietnam, and Dominican Republic). In addition, to assist these programs with monitoring and evaluating their own progress, the TI require panel physicians to report statistical indicators of their medical screening process.

The first screening programs to implement CDOT TB TI were the refugee screening program in Thailand (for Hmong and Burmese refugees) on April 9, 2007, and the immigrant screening programs in Mexico and Philippines on October 1, 2007. Each year, panel physicians in additional countries conducted the screening according to the new standards. By 2011, panel physicians were using the new instructions in 51 countries, screening 70% of immigrant arrivals to the United States. By August 2012, DGMQ had worked with the Bureau of Consular Affairs at the U.S. Department of State to establish a deadline of October 1, 2013, for panel physicians


† Available at http://www.cdc.gov/panelphysicians/index.html.
worldwide to be screening in accordance with CDOT TB TI. That deadline was met by nearly all countries.

Results of Implementation

During 2007–2013, site visits were conducted in 71 of the 151 jurisdictions that have panel physicians. To fulfill the laboratory culture requirement, new laboratories performing TB cultures were developed in China, India, Kenya, Mexico, Nepal, Thailand, Vietnam, and other countries. In addition, laboratories serving panel physicians in several countries developed the capability to perform drug-susceptibility testing on second-line drugs, which are used to treat multidrug resistant TB (MDR TB). These countries include China, Kenya, Nepal, Thailand, and Vietnam. During 2008–2013, 10 training summits were conducted, attended by panel physicians or U.S. Department of State consular officers, representing a total of 101 countries.

Preliminary analysis of crude indicators reported by panel physicians indicated that approximately 1,100 cases of TB were diagnosed during 2012, the year for which the most recent data were available. Approximately 60% of all cases were smear-negative, but culture-positive. Because the previous system did not require cultures, the smear-negative but culture-positive cases represent a gain in diagnostic yield with the new algorithm.

What is already known about this topic?
The United States is one of the largest immigrant and refugee–receiving countries. Preimmigration screening for tuberculosis (TB) historically has been required before entry and has been demonstrated as effective in preventing importation of active TB. However, the 1991 U.S. screening algorithm was outdated. New TB screening requirements, known as the Culture and Directly Observed Therapy Technical Instructions (CDOT TB TI), were issued in 2007. CDOT TB TI use newer technologies and TB cultures to increase the diagnostic yield, and also require treatment in accordance with U.S. guidelines. Since 2007, CDC has been working to implement CDOT TB TI worldwide.

What is added by this report?
Implementation of CDOT TB TI is effectively complete. During 2007–2014, panel physicians began using the new screening algorithm in 147 of 151 jurisdictions. The diagnostic yield increased twofold to threefold, with approximately 1,100 cases of TB diagnosed worldwide during 2012; approximately 60% of these cases were smear-negative, but culture-positive, representing a gain in diagnostic yield with the new algorithm. Preliminary evidence suggests the percentage of persons with abnormal chest radiographs overseas, but negative sputum smears, who are diagnosed with TB upon arriving in the United States has decreased from approximately 7% to 1%–2%. Implementation also is temporally associated with a decline in reported cases of TB among foreign-born persons in the United States 1 year after their arrival.

What are the implications for public health practice?
Successful implementation of CDOT TB TI demonstrates that rigorous diagnostic and treatment programs meeting international standards can be implemented in areas with high incidence of TB overseas. To further reduce the number of cases of TB among foreign-born persons, consideration might be given to extending screening to long-term visitors, developing strategies to address latent TB infection in the foreign-born, and strengthening U.S. follow-up for arriving persons identified overseas as being at risk for TB.

Discussion

Overseas implementation of CDOT TB TI during April 2007–February 2014 was a successful worldwide TB intervention that directly benefitted U.S. TB control. In addition to increasing the yield of diagnoses overseas, implementation of CDOT TB TI was temporally associated with a decline in TB cases among foreign-born persons in the United States.
since 2007 (2). Although many factors could have produced the decline in TB rates, an increase in diagnosis and treatment of active TB overseas and the timing of the decline suggests implementation of CDOT TB TI was a major determinant (DGMQ, CDC, unpublished data, 2014).

Screening applicants for U.S. immigration status has been demonstrated to be an effective tool for identifying persons with TB disease before they enter the United States (3). However, given the nature of TB, vigilance after arrival also is needed, because persons with latent TB infection can convert to an active state after arrival. During the period in which the 1991 TB TI was in use, 7% of immigrants and refugees who had abnormal chest radiographs suggestive of TB, but negative sputum smears, were diagnosed with TB disease after their arrival in the United States (3). Under CDOT TB TI, early data suggest that percentage has declined to 1%–2% (4). Although formal economic analyses have not been completed, the gains in overseas diagnosis and the decrease in cases suggest that successful implementation of this screening program could result in crude savings in excess of $15 million yearly.

A previous analysis determined that investments in TB control in countries where the disease is endemic can yield a greater return on investment than only improving preentry screening algorithms (5). For this reason, a key component of DGMQ’s implementation plan has been to link panel physicians with their country’s national TB programs. Such successful linkages have included panel physicians in the Dominican Republic, who entered into a public-private partnership with that country’s National Tuberculosis Program (6), and the International Organization for Migration, which manages refugee resettlements and serves as the panel physician for applicants in Nairobi, Kenya, providing laboratory testing and DOT for certain nonresettling or immigrating populations (International Organization for Migration, unpublished data, 2011). To maximize the opportunity for the laboratory and treatment infrastructure to benefit more than U.S.-bound populations, efforts should continue to seek ways in which the screening program can collaborate with in-country TB programs.

Although the 1991 algorithm was shown to help prevent importation of TB (2), it did not incorporate newer, more sensitive technologies for diagnosing TB or include a treatment component. To help determine what changes could be made to the TI, DGMQ and CDC’s Division of Tuberculosis Elimination collaborated on research activities. A key outcome of that effort was the 2006 publication of a study demonstrating that, compared with the gold standard of mycobacterial cultures, the 1991 algorithm relying on sputum smears was only 34% sensitive in diagnosing TB (7).

Implementation of CDOT TB TI is one part of a broader strategy to address TB among foreign-born persons in the United States. Resources should be devoted toward rigorous monitoring of the program to maintain what has been developed and increase linkages with in-country efforts. Moreover, additional strategies to further decrease TB among foreign-born persons might be explored, such as extending screening to long-term visitors (8), developing innovative strategies to address the reservoir of latent TB infection in the foreign-born population (9), and strengthening U.S. follow-up for arriving persons identified overseas as being at risk for TB (10).

1Division of Global Migration and Quarantine, National Center for Emerging and Zoonotic Infectious Diseases, CDC. (Corresponding author: Drew L. Posey, dposey@cdc.gov, 404-498-1600)

References

2. CDC. Decrease in reported tuberculosis cases—United States, 2009. MMWR 2010;59:289–94.
Combined Use of Inactivated and Oral Poliovirus Vaccines in Refugee Camps and Surrounding Communities — Kenya, December 2013

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Since the launch of the Global Polio Eradication Initiative (GPEI) in 1988, circulation of indigenous wild poliovirus (WPV) has continued without interruption in only three countries: Afghanistan, Nigeria, and Pakistan (1). During April–December 2013, a polio outbreak caused by WPV type 1 (WPV1) of Nigerian origin resulted in 217 cases in or near the Horn of Africa, including 194 cases in Somalia, 14 cases in Kenya, and nine cases in Ethiopia (all cases were reported as of March 10, 2014) (2,3). During December 14–18, 2013, Kenya conducted the first-ever campaign providing inactivated poliovirus vaccine (IPV) together with oral poliovirus vaccine (OPV) as part of its outbreak response. The campaign targeted 126,000 children aged ≤59 months who resided in Somali refugee camps and surrounding communities near the Kenya-Somalia border, where most WPV1 cases had been reported, with the aim of increasing population immunity levels to ensure interruption of any residual WPV transmission and prevent spread from potential new importations. A campaign evaluation and vaccination coverage survey demonstrated that combined administration of IPV and OPV in a mass campaign is feasible and can achieve coverage >90%, although combined IPV and OPV campaigns come at a higher cost than OPV-only campaigns and require particular attention to vaccinator training and supervision. Future operational studies could assess the impact on population immunity and the cost-effectiveness of combined IPV and OPV campaigns to accelerate interruption of poliovirus transmission during polio outbreaks and in certain areas in which WPV circulation is endemic.

During April–July 2013, a total of 14 paralytic polio cases caused by WPV1, genetically linked to a virus originating in Nigeria and also circulating in Somalia, were reported in Kenya; seven cases occurred in residents of refugee camps, six in surrounding communities, and one in a noncontiguous district but also near the Kenya-Somalia border (3) (Figure). In response to the outbreak, the Kenyan Ministry of Health conducted one national and five subnational OPV campaigns during May–November 2013. In December, the Ministry of Health administered IPV and OPV combined in a campaign directed at approximately 126,000 children aged ≤59 months including those who lived in five refugee camps (Dagahaley, Ifo 1, Ifo 2, Hagadera, and Kambioos: 98,365 children), and those in communities within five divisions that surround the camps (Dadaab, Dertu, Jarajila, Sabuli, and Liboi: approximately 27,000 children) near the border with Somalia. GPEI partners1 provided funding and technical support for campaign planning and evaluation, staff training, vaccine procurement, and social mobilization. The Kenya Ministry of Health planned and implemented immunization activities with refugee camp coordinating agencies.2

IPV/OPV Campaign Implementation

The campaign was implemented by 299 teams (173 in camps and 126 in surrounding communities) assigned to fixed (i.e., permanent) sites in health facilities and to “temporary fixed” sites in each block (in camps) or surrounding communities; mobile teams were used to reach scattered settlements of nomads. Each team included one health-care worker and two volunteers (in communities) or three volunteers (in camps). The health-care worker administered IPV (and OPV on some teams). One to two volunteers administered OPV and tallied children or marked fingers after vaccination, and one volunteer conducted door-to-door mobilization of caregivers, encouraging them to take their children to the vaccination sites. Children aged <6 weeks received OPV alone; children aged 6 weeks–59 months received OPV followed immediately by IPV.

Focus group interviews conducted before the campaign suggested ready acceptance of an injectable polio vaccine. Participants thought injections were very effective but would only accept injection by health-care workers. Caregivers also had no concerns about simultaneous administration of IPV and OPV because they viewed the two vaccines as working differently (i.e., IPV provides protection in the bloodstream, and OPV provides protection in the gut). Based on these responses, communication materials and volunteers emphasized the concept that receiving OPV is essential, but IPV can enhance immunity against polio.

1 The GPEI initiative was founded in 1988 with the following spearheading agencies: the World Health Organization, Rotary International, CDC, and UNICEF; the Bill and Melinda Gates Foundation subsequently became a major supporter of GPEI.

2 The organizations coordinating health services in refugee camps are Medecins Sans Frontieres in Dagahaley, Islamic Relief Kenya in Ifo 1, Kenya Red Cross in Ifo 2, and the International Rescue Committee in Hagadera and Kambioos. The United Nations High Commissioner for Refugees oversaw implementation in all camps.
Vaccination activities of 47 randomly selected teams were assessed by trained campaign monitors using a standardized checklist. Of the 47 teams, 43 (91%) had sufficient staff, vaccine, and supplies to vaccinate the estimated target population, and in 39 (83%), a team member conducted door-to-door mobilization of caregivers. Of 47 vaccinators observed, five (11%) made an error in IPV administration (injection site or dosage), two (4%) prefilled syringes before the session began, and eight (17%) recapped needles during injection preparation. Errors in finger-marking or tallying of children who had received vaccine were observed in two (4%) and seven (15%) teams, respectively. Vaccines were kept in vaccine carriers with at least two ice packs at 44 (94%) of the sites. No vaccine vial monitor§ on OPV

Abbreviations: IPV = inactivated poliovirus vaccine; OPV = oral poliovirus vaccine; WPV1 = wild poliovirus type 1.

### Campaign Monitoring

Vaccination activities of 47 randomly selected teams were assessed by trained campaign monitors using a standardized checklist. Of the 47 teams, 43 (91%) had sufficient staff, vaccine, and supplies to vaccinate the estimated target population, and in 39 (83%), a team member conducted door-to-door mobilization of caregivers. Of 47 vaccinators observed, five (11%) made an error in IPV administration (injection site or dosage), two (4%) prefilled syringes before the session began, and eight (17%) recapped needles during injection preparation. Errors in finger-marking or tallying of children who had received vaccine were observed in two (4%) and seven (15%) teams, respectively. Vaccines were kept in vaccine carriers with at least two ice packs at 44 (94%) of the sites. No vaccine vial monitor§ on OPV

§OPV can be frozen without having impact but potency is affected by high temperatures, which can be detected by vaccine vial monitors on the label. The vaccine vial monitor changes color when a vial has been exposed to excessive temperature over time that has likely damaged the vaccine. Vaccine can be used if the inner square is lighter than the outer circle but must be discarded if the inner square is the same color or darker than the outer circle. IPV is sensitive to freezing and heat and recommended to be stored and transported at 36.6°F–46.4°F (2°C–8°C); IPV vials used in this campaign had no vaccine vial monitors.
vials had a color change indicating substantial heat exposure. No vaccine vial monitors were used on IPV vials. One team was found to have frozen IPV vials; follow-up investigation revealed that these vials had been stored in a freezer before distribution to the site. Electronic temperature monitors placed inside 42 vaccine carriers during vaccination activities recorded periods of ≥60 minutes below 36.6°F (2°C) in 12 (42%) carriers and above 46.4°F (8°C) in eight (19%) carriers.

Vaccine cost was $2.09 per IPV dose† and $0.14 per OPV dose; the operational cost per child vaccinated during the IPV/OPV December campaign was $1.04, compared with $0.36 in the November OPV-only campaign. Estimated total cost per child vaccinated was $3.27 and $0.50 for the December and November campaigns, respectively.

No serious illnesses, hospitalizations, or deaths were reported through the passive surveillance system implemented for detecting adverse events during the week following vaccination. One child who received OPV via intramuscular injection caused by vaccinator error experienced pain and local inflammation at the injection site, and it resolved within a few days as this child was monitored.

Coverage Survey

During December 19–23, 2013, vaccination coverage surveys were conducted using cluster survey methodology. The sampling frame was derived for camps from information provided by the United Nations High Commissioner for Refugees registry office and adapted to include areas with “unregistered” populations; campaign coordinators provided the estimated number of children aged ≤59 months for surrounding communities.** Because of the absence of a sampling frame for nomads, a convenience sample of nomadic families settled near villages participating in the survey was selected. Receipt of OPV with or without IPV in the December campaign, reasons for nonvaccination, and receipt of OPV in the November campaign were recorded for all children aged ≤59 months in each household.

Of 1,286 houses surveyed, caregiver recall information on receipt of IPV or OPV was available for 2,161 children in 1,016 households. Coverage with OPV and IPV in the December campaign was 92.8% in the refugee camps and 95.8% in surrounding communities. Receipt of OPV in the November campaign was 97.2% in the refugee camps and 97.3% in surrounding communities (Table 1).

Among 107 (5%) children aged ≥6 weeks who did not receive IPV, caregivers for 49 (46%) reported not knowing where to go for vaccination; 16 (15%) cited potential refusals (ill child, five; fear of pain, eight; and fear of adverse effects, three), and 10 (9%) children were absent during the campaign. Twelve of the 107 children who missed IPV received OPV (Table 2). Among 1,009 (99%) caregivers who were aware of the campaign, the most common sources of information were public address system or megaphone announcements (76%), a visit by a social mobilizer (47%) or health-care worker (43%), and radio (36%).

In 65 nomadic households surveyed, 40 (34%) of 118 eligible children had received IPV and OPV in the December campaign, and 37 (31%) had received OPV in the November campaign. Among children in the nomadic households, reported reasons for missing vaccine in the December campaign were lack of awareness of the campaign (70 of 76 [92%]) and not knowing where to get vaccine (six of 76 [9%]). Sources of information about the December campaign among 24 caregivers who knew about the campaign included a neighbor (54%), megaphone announcements (33%), and radio (29%).

Discussion

Clinical trials have demonstrated that administration of IPV to children who had received OPV increases humoral and mucosal immunity to the three poliovirus serotypes more effectively than a supplementary dose of OPV (4,5). In December 2013, after a WPV1 outbreak, the Kenya Ministry of Health implemented a mass campaign with combined IPV/OPV administration in Somali refugee camps and surrounding communities in Kenya to boost population immunity levels to ensure interruption of any residual WPV transmission and prevent spread from potential new importations. This population was considered at greater risk because of the high number of cases reported in the outbreak, prior importations of WPV and vaccine-derived polioviruses (3), and frequent population movement between Somalia and major Kenyan cities in the area.

Several challenges to the use of IPV in campaign settings have been noted previously, including 1) increased cost and operational complexity; 2) potentially reduced coverage, because injectable vaccines cannot be delivered house-to-house; and 3) concerns about caregiver mistrust of IPV or their rejection of OPV-only campaigns in the future. Factors that contributed to the success in overcoming these challenges for this campaign in Kenya included 1) strong commitment from the Ministry of Health and coordination among implementing partners in developing comprehensive operational plans and allocating resources quickly, 2) flexibility to move “temporary fixed” sites frequently in response to caregiver demands to bring vaccine closer to their homes, and 3) high acceptance of IPV by caregivers, as shown by the high coverage and the small proportion of

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× The IPV cost included vaccine, syringes, needles, and shipment. The OPV cost included vaccine and shipment.

** Eligible households were those in which children aged ≤59 months resided. Liboi Division was excluded from the survey because of insecurity.
unvaccinated children (15%) who missed vaccine because of potential refusals. Vaccination coverage in a subsequent OPV-only campaign conducted in February 2014 in the area was similar to coverage in previous campaigns, showing that IPV use in one campaign did not negatively impact a subsequent OPV campaign.

Challenges in field implementation of the IPV/OPV campaign can provide lessons for future campaigns. Observation of vaccinators revealed errors in injection technique and in IPV use; similar findings have been identified with other injectable vaccines used in campaign settings (6,7), stressing the need for appropriate training of vaccinators and supervisors. Both vaccines are recommended to be stored and transported at 36.6°F–46.4°F (2°C–8°C), but this study found that temperatures inside vaccine carriers might be above or below the recommended range. Whereas OPV would not be affected by low temperatures, and vaccine vial monitors would indicate when vaccine has been damaged by heat, IPV vials used in this campaign did not have vaccine vial monitors, and staff members were unaware that IPV can be damaged by freezing (8). Comprehensive precampaign planning of cold chain requirements, consideration of vaccine vial monitor inclusion on IPV vials, and appropriate staff training on existing guidelines for prevention of vaccine damage from heat or freezing (8) will be important to prevent loss of vaccine effectiveness in future campaigns. Additionally, a survey in nomadic settlements found low campaign awareness and a high proportion of children who did not receive vaccine during either the November or December campaigns, suggesting that certain settlements are missed repeatedly. Additional strategies, including improved communications, are needed to track and access nomadic populations during all vaccination campaigns and reflect seasonality of nomadic movements.

As part of the Polio Eradication and Endgame Strategic Plan 2013–2018,†† which aims to discontinue all use of OPV after

<table>
<thead>
<tr>
<th>Study area</th>
<th>Target population size</th>
<th>Percentage who received both IPV and OPV in December campaign</th>
<th>Percentage who received OPV only in November campaign</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. surveyed</td>
<td>(%)</td>
<td>(95% CI)</td>
</tr>
<tr>
<td>Dagahaley camp</td>
<td>23,815</td>
<td>299</td>
<td>(83.3)</td>
</tr>
<tr>
<td>Ifo 1 camp</td>
<td>22,350</td>
<td>270</td>
<td>(94.1)</td>
</tr>
<tr>
<td>Ifo 2 camp</td>
<td>21,560</td>
<td>331</td>
<td>(98.2)</td>
</tr>
<tr>
<td>Hagadera camp</td>
<td>24,660</td>
<td>340</td>
<td>(95.3)</td>
</tr>
<tr>
<td>Kambioos camp</td>
<td>5,980</td>
<td>328</td>
<td>(96.3)</td>
</tr>
<tr>
<td>Total camps</td>
<td>98,365</td>
<td>1,568</td>
<td>(93.3)</td>
</tr>
<tr>
<td>Surrounding communities</td>
<td>21,831</td>
<td>593</td>
<td>(95.8)</td>
</tr>
<tr>
<td>Overall</td>
<td>120,196</td>
<td>2,161</td>
<td>93.3</td>
</tr>
</tbody>
</table>

Abbreviation: CI = confidence interval.

*Infants aged <6 weeks received OPV only. Children aged 6 weeks–59 months received OPV followed by IPV. Receipt of vaccination was documented by caregiver.
†Resident of communities in the following divisions: Dadaab, Dertu, Jarajila, and Sabuli; Liboi Division was excluded from the survey for security reasons.

<table>
<thead>
<tr>
<th>Reasons</th>
<th>Children in refugee camps (n = 90)</th>
<th>Children in surrounding communities (n = 17)</th>
<th>Overall (N = 107)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Communication /Social mobilization</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unaware of campaign</td>
<td>7 (8)</td>
<td>0</td>
<td>7 (7)</td>
</tr>
<tr>
<td>Didn’t know where to get vaccine</td>
<td>47 (52)</td>
<td>2 (12)</td>
<td>49 (46)</td>
</tr>
<tr>
<td><strong>Delivery issues</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vaccination site too far</td>
<td>3 (3)</td>
<td>0</td>
<td>3 (3)</td>
</tr>
<tr>
<td>Vaccination time inconvenient</td>
<td>4 (4)</td>
<td>0</td>
<td>4 (4)</td>
</tr>
<tr>
<td><strong>Individual reasons</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ill child</td>
<td>5 (6)</td>
<td>0</td>
<td>5 (5)</td>
</tr>
<tr>
<td>Fear of pain from injection</td>
<td>3 (3)</td>
<td>5 (29)</td>
<td>8 (7)</td>
</tr>
<tr>
<td>Fear of adverse effects from vaccine</td>
<td>2 (2)</td>
<td>1 (6)</td>
<td>3 (3)</td>
</tr>
<tr>
<td>Child absent during vaccination activities</td>
<td>6 (7)</td>
<td>4 (24)</td>
<td>10 (9)</td>
</tr>
<tr>
<td>Reason not recorded</td>
<td>13 (14)</td>
<td>5 (29)</td>
<td>18 (17)</td>
</tr>
</tbody>
</table>

*Twelve children received oral poliovirus vaccine only; 95 did not receive either vaccine.


TABLE 1. Vaccination coverage with inactivated poliovirus vaccine (IPV) and oral poliovirus vaccine (OPV) during a December campaign and with OPV only during a November campaign,* by refugee camp and surrounding communities† — Kenya, 2013

TABLE 2. Reasons reported by caregivers for children aged ≥6 weeks not receiving inactivated poliovirus vaccine during a December vaccination campaign in refugee camps and surrounding communities*— Kenya, 2013
eradication of WPV, IPV is to be introduced by the end of 2015 into the routine immunization schedules of 126 countries that use only OPV (9,10). The Kenya experience has shown that IPV can also be provided in campaigns with high coverage and community acceptance, although at a higher cost than OPV-only campaigns and requiring particular attention to training and supervision. IPV/OPV campaigns could be considered to improve population immunity and accelerate interruption of poliovirus transmission in other polio outbreaks and in certain areas where WPV transmission is endemic. Operational studies during future campaigns should assess the impact on population immunity and the cost-effectiveness of this strategy in different settings.

Acknowledgments


References


What is already known on this topic?

Results from clinical trials have suggested that administration of inactivated poliovirus vaccine (IPV) in combination with oral poliovirus vaccine (OPV) through mass campaigns in certain settings could achieve the high population immunity levels required to interrupt poliovirus transmission with fewer campaigns. IPV, administered by intramuscular injection, has not been used in campaigns because of concerns about the increased cost and operational complexity, potential reduction in coverage, and potentially lower caregiver acceptance.

What is added by this report?

The first community-based IPV/OPV campaign was conducted during December 14–18, 2013, in Kenya in response to a wild poliovirus type 1 outbreak. The campaign targeted an estimated 126,000 children aged ≤59 months who lived in five refugee camps and in communities surrounding the camps in five divisions near the Kenya-Somalia border. A survey estimated coverage with both vaccines at 92.8% in refugee camps and 95.8% in surrounding communities.
Update on Vaccine-Derived Polioviruses — Worldwide, July 2012–December 2013

Ousmane M. Diop, PhD1, Cara C. Burns, PhD2, Steven G. Wassilak, MD3, Olen M. Kew, PhD2 (Author affiliations at end of text)

In 1988, the World Health Assembly resolved to eradicate poliomyelitis worldwide (1). One of the main tools used in polio eradication efforts has been live, attenuated oral poliovirus vaccine (OPV), an inexpensive vaccine easily administered by trained volunteers. OPV might require several doses to induce immunity, but then it provides long-term protection against paralytic disease through durable humoral immunity. Rare cases of vaccine-associated paralytic poliomyelitis can occur among immunologically normal OPV recipients, their contacts, and persons who are immunodeficient. In addition, vaccine-derived polioviruses (VDPVs) can emerge in areas with low OPV coverage to cause polio outbreaks and can replicate for years in persons who have primary, B-cell immunodeficiencies. This report updates previous surveillance summaries (2) and describes VDPVs detected worldwide during July 2012–December 2013. Those include a new circulating VDPV (cVDPV) outbreak identified in Pakistan in 2012, with spread to Afghanistan; an outbreak in Afghanistan previously identified in 2009 that continued into 2013; a new outbreak in Chad that spread to Cameroon, Niger, and northeastern Nigeria; and an outbreak that began in Somalia in 2008 that continued and spread to Kenya in 2013. A large outbreak in Nigeria that was identified in 2005 was nearly stopped by the end of 2013. Additionally, 10 newly identified persons in eight countries were found to excrete immunodeficiency-associated VDPVs (iVDPVs), and VDPVs were found among immunocompetent persons and environmental samples in 13 countries. Because the majority of VDPV isolates are type 2, the World Health Organization has developed a plan for coordinated worldwide replacement of trivalent OPV (tOPV) with bivalent OPV (bOPV; types 1 and 3) by 2016, preceded by introduction of at least 1 dose of inactivated poliovirus vaccine (IPV) containing all three poliovirus serotypes into routine immunization schedules worldwide to ensure high population immunity to all polioviruses (3).

Properties of VDPVs

Three poliovirus serotypes (PV1, PV2, and PV3) have been identified. Poliovirus isolates are grouped into three categories: 1) WPVs (current WPVs are genetically unrelated to any vaccine strain), 2) vaccine-related polioviruses (VRPVs; <1% divergent [PV1 and PV3] or <0.6% divergent [PV2] from the corresponding OPV strain), and 3) VDPVs (VRPVs >1% divergent [PV1 and PV3] or >0.6% divergent [PV2] from the corresponding OPV strain) (2). VDPVs are further categorized as 1) cVDPVs when evidence of person-to-person transmission in the community exists; 2) iVDPVs, which are isolated from persons with primary, B-cell immunodeficiencies (defects in antibody production); and 3) ambiguous VDPVs (aVDPVs), which are either clinical isolates from persons with no known immunodeficiency and no evidence of transmission or sewage isolates whose source is unknown (2).

VDPVs can cause paralytic polio in humans and have the potential for sustained circulation. VDPVs resemble WPVs biologically (2) and differ from VRPV isolates by having genetic properties consistent with prolonged replication or transmission. Because poliovirus genomes evolve at an overall rate of approximately 1% per year, VRPVs that differ from the corresponding OPV strain by >1% of nucleotide positions (determined by sequencing the genomic region that encodes the major viral surface protein [VP1]) are presumed to have replicated for ≥1 year in one or more persons after administration of an OPV dose and are VDPVs. The typical period of vaccine virus replication is 4–6 weeks in an OPV recipient.

Virologic Testing for VDPVs

All poliovirus isolates are characterized by laboratories of the Global Polio Laboratory Network (4). The original protocol to screen for VDPVs, using a combination of molecular and antigenic methods, has largely been replaced by a real-time reverse transcription–polymerase chain reaction (rRT-PCR) nucleic acid amplification targeted to nucleotide substitutions that typically revert to the WPV sequence during replication of OPV in the human intestine (5). The rRT-PCR methods have been transferred to 88 of 146 Global Polio Laboratory Network laboratories (4). Candidate VDPVs identified by rRT-PCR screening are sequenced in the VP1 region for definitive analysis; the complete genome is sequenced if required for higher-resolution analysis.

cVDPVs

The number of countries with indigenous cVDPV circulation increased from six to seven since the April 2011–June 2012 reporting period (2). Outbreaks in the Democratic Republic of the Congo (6), Madagascar, Mozambique, and Yemen (type 2 cVDPV [cVDPV2]) appeared to have been interrupted (2);
outbreaks identified during the previous period in Afghanistan and Somalia continued; a large outbreak in Nigeria has reached very low incidence (2, 7); and new outbreaks were detected in Chad, China, and Yemen. Circulating VDPVs were exported from Chad to Cameroon, Niger, and Nigeria; from Pakistan to Afghanistan; and from Somalia to Kenya. In all countries but Yemen (cVDPV3 outbreak), the cVDPVs detected during this reporting period were type 2 (Table, Figure 1).

Afghanistan. During July 2009–February 2013, cVDPV2s were isolated from 15 acute flaccid paralysis (AFP) patients and eight contacts from insecure areas of Helmand Province. Two 2012 cVDPV2 isolates from contacts of a child with AFP represented a second emergence in Helmand. Four cVDPV2 isolates from Kandahr Province during October 2012–March 2013 revealed frequent cross-border transmission from Pakistan.

Cameroon. Among four 2013 cVDPV2 isolates in the Extrême Nord region, two were closely related to isolates from Chad, and two were most closely related to virus circulating in Borno state, Nigeria, and ultimately related to cVDPV2 circulating in Chad.

Chad. Circulating VDPV2s were isolated from 16 AFP patients during August 2012–May 2013, derived from at least two separate emergences. One (associated with 14 reported patients during August 2012–May 2013, derived from at least two separate introductions from the Somalia outbreak.

China. During October 2011–February 2012, cVDPV2s were isolated from three patients with AFP who had received no prior doses of OPV and one contact in Sichuan Province in a country that had gaps in rOPV coverage.

Kenya. During July 2012–September 2012, three distinct cVDPV2s were isolated from one AFP patient and two contacts in the Dadaab refugee camp near the border with Somalia, representing two separate introductions from the Somalia outbreak.

Niger. One cVDPV2 was isolated from a patient in Diffa, southeastern Niger, along the border with Nigeria, with onset of AFP in July 2013. The isolate was most closely related to cVDPV2s circulating in Borno state, Nigeria, but appears to have originated in Chad. As with previous cVDPV2 importations (2), no secondary cases were found in Niger.

Nigeria. The large indigenous cVDPV2 outbreak (383 AFP cases) in northern Nigeria (7), first detected in 2005, appears to have reached very low incidence. During July–December 2012, the indigenous cVDPV2s were isolated from five AFP patients and three contacts. In addition, virus closely related to the indigenous cVDPV2 was isolated from 46 environmental samples. Although the last isolate was from an AFP patient with paralysis onset in December 2012, the indigenous cVDPV2 was detected in 11 environmental samples, most recently in

TABLE. Vaccine-derived polioviruses (VDPVs) detected worldwide — July 2012–December 2013

<table>
<thead>
<tr>
<th>Category</th>
<th>Country</th>
<th>Year(s) detected†</th>
<th>Source (total cases or specimens)†</th>
<th>Sero-type</th>
<th>No. of isolates§ July 2012–December 2013</th>
<th>VP1 divergence from Sabin OPV strain (%)</th>
<th>Routine coverage with 3 doses of polio vaccine (%)*</th>
<th>Estimated duration of VDPV replication (yrs)**</th>
<th>Current status (date of last outbreak case, last patient isolate, or last environmental sample)</th>
</tr>
</thead>
<tbody>
<tr>
<td>cVDPV††</td>
<td>Afghanistan</td>
<td>2009–2013</td>
<td>Outbreak (15 cases)</td>
<td>2</td>
<td>8</td>
<td>(0.9–5.5)</td>
<td>(71)</td>
<td>5 February 13, 2013</td>
<td></td>
</tr>
<tr>
<td>Afghanistan</td>
<td>2012–2013</td>
<td>Importation†§</td>
<td>4</td>
<td>4</td>
<td>8</td>
<td>(2.0–2.3)</td>
<td>(71)</td>
<td>— March 13, 2013</td>
<td></td>
</tr>
<tr>
<td>Cameroon</td>
<td>2013</td>
<td>Importation††</td>
<td>2</td>
<td>4</td>
<td>8</td>
<td>(1.2–2.0)</td>
<td>(85)</td>
<td>— August 12, 2013</td>
<td></td>
</tr>
<tr>
<td>Chad</td>
<td>2012–2013</td>
<td>Importation††</td>
<td>2</td>
<td>16</td>
<td>8</td>
<td>(0.7–1.8)</td>
<td>(56)</td>
<td>1.5 May 12, 2013</td>
<td></td>
</tr>
<tr>
<td>China</td>
<td>2011–2012</td>
<td>Outbreak (3 cases)</td>
<td>2</td>
<td>3</td>
<td>1</td>
<td>(0.7–1.8)</td>
<td>(99)</td>
<td>1.5 February 8, 2012</td>
<td></td>
</tr>
<tr>
<td>Kenya</td>
<td>2012</td>
<td>Importation††</td>
<td>2</td>
<td>1</td>
<td>2</td>
<td>(4.3–4.9)</td>
<td>(82)</td>
<td>— August 29, 2012</td>
<td></td>
</tr>
<tr>
<td>Niger</td>
<td>2013</td>
<td>Importation†††</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>(2.1)</td>
<td>(78)</td>
<td>July 14, 2013</td>
<td></td>
</tr>
<tr>
<td>Nigeria§§§</td>
<td>2005–2013</td>
<td>Outbreak (383 cases)†††</td>
<td>2</td>
<td>12</td>
<td>1</td>
<td>(0.7–7.3)</td>
<td>(59)</td>
<td>September 18, 2013</td>
<td></td>
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<tr>
<td>Nigeria</td>
<td>2013</td>
<td>Importation†††</td>
<td>2</td>
<td>4</td>
<td>12</td>
<td>(1.2–2.4)</td>
<td>(59)</td>
<td>— November 20, 2013</td>
<td></td>
</tr>
<tr>
<td>Pakistan</td>
<td>2012–2013</td>
<td>Outbreak (61 cases)‡</td>
<td>2</td>
<td>61</td>
<td>6</td>
<td>(0.7–3.3)</td>
<td>(75)</td>
<td>3 December 30, 2013</td>
<td></td>
</tr>
<tr>
<td>Somalia</td>
<td>2008–2013</td>
<td>Outbreak (19 cases)‡</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>(3.3–4.0)</td>
<td>(47)</td>
<td>5 January 9, 2013</td>
<td></td>
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<tr>
<td>Yemen</td>
<td>2011–2013</td>
<td>Outbreak (4 cases)</td>
<td>3</td>
<td>4</td>
<td>2</td>
<td>(2.0–3.0)</td>
<td>(89)</td>
<td>2.5 July 25, 2013</td>
<td></td>
</tr>
<tr>
<td>iVDPV</td>
<td>Afghanistan</td>
<td>2013</td>
<td>AFP patient PID</td>
<td>2</td>
<td>1</td>
<td>(0.9)</td>
<td>(71)</td>
<td>&lt;1 October 22, 2013</td>
<td></td>
</tr>
<tr>
<td>China</td>
<td>2013</td>
<td>AFP patient PID</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td>(1.3)</td>
<td>(99)</td>
<td>&lt;1 May 19, 2013</td>
<td></td>
</tr>
<tr>
<td>Egypt</td>
<td>2012</td>
<td>Non-AFP patient PID</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>(1.0)</td>
<td>(99)</td>
<td>~1 July 4, 2012</td>
<td></td>
</tr>
<tr>
<td>India</td>
<td>2013</td>
<td>AFP patient AGG</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>(0.9)</td>
<td>(70)</td>
<td>&lt;1 November 11, 2012</td>
<td></td>
</tr>
<tr>
<td>Iran</td>
<td>2012</td>
<td>AFP patient PID</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>(1.0)</td>
<td>(70)</td>
<td>&lt;1 November 11, 2012</td>
<td></td>
</tr>
<tr>
<td>Iraq</td>
<td>2012</td>
<td>AFP patient PID</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>(1.0)</td>
<td>(70)</td>
<td>&lt;1 November 11, 2012</td>
<td></td>
</tr>
<tr>
<td>Saudi Arabia</td>
<td>2013</td>
<td>Non-AFP patient SCID</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>(1.3)</td>
<td>(93)</td>
<td>&lt;1 July 7, 2013</td>
<td></td>
</tr>
<tr>
<td>United States</td>
<td>2013</td>
<td>AFP patient SCID</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>(1.3)</td>
<td>(93)</td>
<td>&lt;1 July 7, 2013</td>
<td></td>
</tr>
</tbody>
</table>

See table footnotes on page 244.
November 2013. During March–November 2013, cVDPV2s were also isolated from five AFP patients and 12 environmental samples in the northeastern states of Borno and Adamawa following importation from Chad. **Pakistan.** During August 2012–December 2013, cVDPV2s were isolated from 61 AFP patients, six contacts, and three environmental samples. The outbreak was associated with an emergence that was first observed in Killa Abdullah, Balochistan Province, with spread in 2013 to the insecure northwestern governorates of Sa‘adah, Hajjah, and Al Hudaydah. The cVDPV3 outbreak followed a cVDPV2 outbreak (11 reported cases, two independent contacts) during April 2011–February 2012. **Somalia.** Since 2004, VDPV2s have been isolated from 15 AFP patients and 21 contacts in the southern regions; most were derived from a single emergence. Circulating VDPV2s were isolated from an AFP patient in Lower Juba in July 2012 and from a contact of an AFP case in Lower Shebelle in January 2013. **Yemen.** During April 2012–July 2013, cVDPV3s were isolated from four AFP patients and two contacts in the insecure northwestern governorates of Sa‘adah, Hajjah, and Al Hudaydah. The cVDPV3 outbreak followed a cVDPV2 outbreak (11 reported cases, two independent contacts) during April 2011–February 2012. **iVDPVs** Since the introduction of OPV in 1961, more than 70 persons with primary immunodeficiencies have been found worldwide to be excreting iVDPVs (indicating prolonged infections); the majority of these immunodeficiencies

### TABLE. (Continued) Vaccine-derived polioviruses (VDPVs) detected worldwide — July 2012–December 2013

<table>
<thead>
<tr>
<th>Category</th>
<th>Country</th>
<th>Year(s)</th>
<th>Source (total cases or specimens)*</th>
<th>Serotype</th>
<th>No. of isolates§ July 2012–December 2013</th>
<th>Non-AFP Source</th>
<th>Routine coverage with 3 doses of polio vaccine (%)</th>
<th>Estimated duration of VDPV replication (yrs)**</th>
<th>Current status (date of last outbreak case, last patient isolate, or last environmental sample)</th>
</tr>
</thead>
<tbody>
<tr>
<td>aVDPV</td>
<td>Angola</td>
<td>2013</td>
<td>AFP patient 2</td>
<td></td>
<td>1</td>
<td></td>
<td>(0.8)</td>
<td>&lt;1</td>
<td>October 6, 2013</td>
</tr>
<tr>
<td></td>
<td>China</td>
<td>2012****</td>
<td>AFP patient 1</td>
<td></td>
<td>1</td>
<td></td>
<td>(2.3)</td>
<td>~1</td>
<td>May 29, 2012</td>
</tr>
<tr>
<td></td>
<td>China</td>
<td>2013</td>
<td>AFP patient 3</td>
<td></td>
<td>1</td>
<td></td>
<td>(1.3)</td>
<td>~1</td>
<td>May 19, 2013</td>
</tr>
<tr>
<td></td>
<td>Egypt</td>
<td>2012–2013</td>
<td>Environment 2</td>
<td></td>
<td>1</td>
<td></td>
<td>(0.7–0.8)</td>
<td>~1</td>
<td>June 7, 2013</td>
</tr>
<tr>
<td></td>
<td>Egypt</td>
<td>2012–2013</td>
<td>Environment 1</td>
<td></td>
<td>1</td>
<td></td>
<td>(1.1)</td>
<td>~1</td>
<td>June 7, 2013</td>
</tr>
<tr>
<td></td>
<td>Estonia</td>
<td>2008–2012</td>
<td>Environment 2</td>
<td></td>
<td>2</td>
<td></td>
<td>(0.7–1.8)</td>
<td>April 14, 2013</td>
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<td></td>
<td>Ethiopia</td>
<td>2012</td>
<td>AFP patient 2</td>
<td></td>
<td>1</td>
<td></td>
<td>(16.2)</td>
<td>&gt;15</td>
<td>December 27, 2012</td>
</tr>
<tr>
<td></td>
<td>Finland</td>
<td>2008–2013</td>
<td>Environment 1</td>
<td></td>
<td>6</td>
<td></td>
<td>(99)</td>
<td>&lt;1</td>
<td>July 20, 2012</td>
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<tr>
<td></td>
<td>India</td>
<td>2013</td>
<td>AFP patient 2</td>
<td></td>
<td>1</td>
<td></td>
<td>(0.9)</td>
<td>~1</td>
<td>May 13, 2013</td>
</tr>
<tr>
<td></td>
<td>India</td>
<td>2012–2013</td>
<td>Environment 1</td>
<td></td>
<td>1</td>
<td></td>
<td>(1.0)</td>
<td>~1</td>
<td>2012</td>
</tr>
<tr>
<td></td>
<td>Iraq</td>
<td>2012</td>
<td>AFP patient 2</td>
<td></td>
<td>0</td>
<td></td>
<td>(0.7–0.8)</td>
<td>~1</td>
<td>November 10, 2012</td>
</tr>
<tr>
<td></td>
<td>Israel</td>
<td>2009–2012</td>
<td>Environment 1</td>
<td></td>
<td>1</td>
<td></td>
<td>(13.8)</td>
<td>&gt;15</td>
<td>November 28, 2013</td>
</tr>
<tr>
<td></td>
<td>Nigeria</td>
<td>2012–2013</td>
<td>AFP patients 2</td>
<td></td>
<td>4</td>
<td></td>
<td>(0.7–0.8)</td>
<td>~1</td>
<td>October 28, 2013</td>
</tr>
<tr>
<td></td>
<td>Sudan</td>
<td>2012</td>
<td>AFP patient 1</td>
<td></td>
<td>2</td>
<td></td>
<td>(0.7–1.4)</td>
<td>~1</td>
<td>January 21, 2013</td>
</tr>
<tr>
<td></td>
<td>Syria</td>
<td>2012</td>
<td>AFP patient 2</td>
<td></td>
<td>1</td>
<td></td>
<td>(1.0)</td>
<td>~1</td>
<td>February 24, 2013</td>
</tr>
<tr>
<td></td>
<td>Turkey</td>
<td>2012</td>
<td>AFP contact 3</td>
<td></td>
<td>1</td>
<td></td>
<td>(1.0)</td>
<td>~1</td>
<td>September 23, 2012</td>
</tr>
</tbody>
</table>

**Abbreviations:** cVDPV = circulating VDPV; iVDPV = immunodeficiency-associated VDPV; aVDPV = ambiguous VDPV; OPV = oral poliovirus vaccine; IPV = inactivated poliovirus vaccine; AFP = acute flaccid paralysis; AGG = agammaglobulinemia; PID = primary immunodeficiency; SCID = severe combined immunodeficiency.

* Total years detected and cumulative totals for previously reported cVDPV outbreaks (Nigeria and Somalia).

1 Outbreaks list total cVDPV cases. Some VDPV case isolates from outbreak periods might be listed as aVDPVs.

2 Total cases for VDPV-positive specimens from AFP cases and total VDPV-positive samples for environmental (sewage) samples.


4 Duration of cVDPV circulation was estimated from extent of VP1 nucleotide divergence from the corresponding Sabin OPV strain; duration of immunodeficiency-associated VDPV replication was estimated from clinical record by assuming that exposure was from initial receipt of OPV; duration of ambiguous VDPV replication was estimated from sequence data.

5 All VDPV isolates except those from China were vaccine/nonvaccine recombinants.

6 Importation from Pakistan.

7 Importation from Chad.

8 Importation from Somalia.

9 Importation from Nigeria of cVDPV2 originating in Chad.

10 All Nigerian cVDPV2 isolates in 2012 from AFP patients, contacts, and the environment were indigenous.

11 Count does not include 29 cases with <10 substitutions in VP1 detected before 2010.

12 **Patient from Myanmar.**
were detected only after onset of AFP. After implementation of intensified surveillance for VDPVs and special studies of iVDPV excretion among persons with primary immunodeficiencies in developing and middle-income countries (8), detection of iVDPV infections increased from two during January 2008–June 2009, to nine during July 2009–June 2011, to 12 during April 2011–June 2012 (2), and declined to 10 during July 2012–December 2013 (Table). Type 2 iVDPVs are the most prevalent (64%), followed by type 1 (21%) and type 3 (15%).

Afghanistan. A boy aged 36 months with primary immunodeficiency infected with iVDPV2 developed AFP in October 2013.

China. A boy aged 7 months with primary immunodeficiency was infected with iVDPV3 after receiving three tOPV doses and developed AFP in May 2013.

Egypt. Two infants, aged 6 months and 5 months, with severe combined immunodeficiency who did not have AFP were found to be infected with iVDPV2s in July 2012 and November 2013, respectively.

India. A girl aged 4 months with hypogammaglobulinemia and infected with iVDPV2 developed AFP in 2013, and a child with agammaglobulinemia infected with iVDPV2 developed AFP and died in 2013.

Iran. Iran has maintained sensitive clinical and laboratory surveillance to screen persons with primary immunodeficiencies for poliovirus infections. During this reporting period, two AFP patients were found to be excreting iVDPVs. A boy aged 11 months with primary immunodeficiency and infected with iVDPV2 developed AFP in August 2012, and a boy aged 13 months with primary immunodeficiency infected with iVDPV2 developed AFP in January 2013.

Iraq. A boy aged 24 months with primary immunodeficiency infected with iVDPV2 developed AFP in July 2012 and died in December 2013.
**Saudi Arabia.** A girl aged 2 years was taken to Germany for treatment for severe combined immunodeficiency. She did not have AFP, but was found to be infected with iVDPV2.

**United States.** A boy aged 7 months with severe combined immunodeficiency who had received 2 doses of OPV in India was infected with iVDPV1 and developed AFP in July 2013, 2 weeks after arrival in the United States; he died 3 weeks after symptom onset.

**aVDPVs**

During June 2012–December 2012, aVDPVs were isolated in 13 countries (Table). The most divergent aVDPVs were continuations of lineages previously detected in sewage samples in Estonia, Finland, and Israel, countries with >90% polio vaccination coverage. The persons infected with the corresponding aVDPVs have not been identified. Detection of aVDPVs in settings (including local pockets) with <60% polio vaccination coverage might signal cVDPV emergence and potential gaps in surveillance. Some aVDPVs, especially those with limited divergence in areas with high vaccination coverage and in patients with no known immunodeficiency, might represent limited spread of OPV virus or the upper limit of OPV sequence divergence in a single normal vaccine recipient or contact.

**Angola.** An AFP patient with no known immunodeficiency was infected with aVDPV2 in October 2013.

**China.** A boy aged 18 months with no known immunodeficiency who received 2 OPV doses in Myanmar traveled to Yunnan, China, after AFP onset and was found to be infected with aVDPV1. Environmental aVDPV2 isolates were detected in Shandong and Guangdong provinces.

**Egypt.** An aVDPV1 was isolated from Alexandria sewage collected in 2012, and unrelated aVDPV2s were isolated from sewage collected in nine sites in eight cities during 2012–2013.

**Estonia.** Two aVDPV2s, closely related to isolates detected in 2008–2010 in Estonia, were detected in Tallinn sewage collected in December 2012. Shared noncapsid sequences with a similarly divergent aVDPV3 strongly suggest origination from a chronic iVDPV excretor (9).

**Ethiopia.** An aVDPV2 was isolated in July 2012 from an AFP patient with no known immunodeficiency.

**Finland.** Highly divergent aVDPV1s and aVDPV2s were isolated from sewage samples collected in Tampere in 2013. The aVDPV isolates were unrelated to aVDPVs found in Estonia, but were closely related to sewage isolates detected during 2008-2012 in Finland, and were likely derived from a single tOPV dose (2).

**India.** In 2013, an aVDPV2 was isolated from an AFP patient with no known immunodeficiency. In addition, one aVDPV1, nine aVDPV2s, and one aVDPV3 were isolated from sewage samples during the reporting period.

**Iraq.** A boy aged 24 months with no known immunodeficiency infected with VDPV2 developed AFP in November 2012.

**Israel.** Highly divergent aVDPV2s had been detected in sewage samples in the Tel Aviv area since 1998 (2), and highly divergent aVDPV1s had been detected in sewage samples in Haifa since 2009. The aVDPVs appear to be derived from different chronic excretors.

**Nigeria.** Environmental isolates closely related to cVDPVs known to be circulating in Nigeria were classified as cVDPVs. Three aVDPV2s were isolated from AFP patients during March–October 2013, and two aVDPV2s were isolated from sewage samples collected at two sites in Sokoto state in June 2012 and January 2013.

**Sudan.** An aVDPV2 was isolated from a contact of an AFP patient in February 2013.

**Syria.** An aVDPV2 was isolated from an AFP patient with no known immunodeficiency in November 2012.

**Turkey.** An aVDPV3 was isolated from a contact of an AFP patient in September 2012.

**Discussion**

Circulating VDPV outbreaks continue to emerge in settings of conflict and insecurity, poor infrastructure, and widening immunity gaps. Outbreaks in Afghanistan, Nigeria, Pakistan, and Somalia have occurred in areas with recent WPV circulation and where conflict and insecurity has limited access of immunization teams to children. As with WPVs, cVDPVs can spread to neighboring countries, causing sporadic cases and outbreaks. In Afghanistan and Nigeria, polio cases were associated with indigenous and imported cVDPVs. When children are accessible, cVDPV outbreaks have been stopped by supplementary immunization activities (SIAs). The large and prolonged indigenous cVDPV2 outbreak in northern Nigeria appears to have reached very low incidence by successive tOPV SIA rounds of increasing quality, but a new outbreak from imported cVDPV2 occurred in insecure areas of the northeast.

Since eradication of WPV2 in 1999, all poliomyelitis cases associated with PV2 have resulted from the continued use of tOPV.

*SIAs are mass vaccination campaigns conducted in a short period (days to weeks) during which a dose of OPV is administered to all children aged <5 years, regardless of previous vaccination history. Campaigns can be conducted nationwide or in portions of a country.*
coordinated worldwide withdrawal of tOPV and replacement with bOPV into its new strategic plan, ultimately leading to withdrawal of all OPV use (3). The switch from tOPV to bOPV, planned for April 2016, is predicated on the complete cessation of cVDPV2 transmission and will require intensification of AFP and poliovirus surveillance. Routine immunization will be strengthened, and in countries using OPV, 1 dose of IPV will be given with the third dose of diphtheria-pertussis-tetanus vaccine. Large stockpiles of monovalent OPV will be maintained, and a robust surveillance and response capacity will be established (3).

Replacement of tOPV with bOPV will greatly reduce the risk for cVDPV2 outbreaks, and global cessation of OPV use (3) will prevent virtually all cVDPV outbreaks and all new iVDPV infections. However, a small number of persons with chronic iVDPV infections are likely to continue to excrete poliovirus for at least a decade after the administration of the last OPV dose. Therefore, maintenance of high levels of population immunity by comprehensive coverage with IPV will be essential to protect against possible iVDPV spread in the community. Detection of chronic iVDPV excretors in all countries (8) and clearing their infections also will be important (10).

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References

A Cluster of Lymphocytic Choriomeningitis Virus Infections Transmitted Through Organ Transplantation — Iowa, 2013

Ilana J. Schafer, DVM1, Rachel Miller, MD2, Ute Ströher, PhD3, Barbara Knust, DVM3, Stuart T. Nichol, PhD3, Pierre E. Rollin, MD3 (Author affiliations at end of text)

On April 26, 2013, the United Network for Organ Sharing reported to CDC a cluster of ill organ transplant recipients in Iowa with a common organ donor. Infection with lymphocytic choriomeningitis virus (LCMV) was suspected. LCMV is a rodent-borne virus that most commonly causes nonfatal, influenza-like illness and occasional aseptic meningitis, but when transmitted through organ transplantation or in utero can cause severe, life-threatening disease.

The organ donor, a man aged 49 years, had experienced a headache and vomiting and was then found unresponsive in his home on March 23, 2013. Imaging revealed a large intracerebral hemorrhage, and he was declared brain dead on March 24. Four patients received donated organs or tissues on March 26, and three were hospitalized between April 12 and 16 with symptoms including fever, abdominal pain, diarrhea, altered mental status, and respiratory compromise. At the time of CDC’s notification, patient A (liver recipient) and patient C (left kidney recipient) were hospitalized in critical condition and patient B (right kidney recipient) had been discharged with resolving symptoms. Patient D (cornea recipient) was asymptomatic.

Diagnostic testing at CDC confirmed LCMV as the causative agent. LCMV was detected in liver and/or blood samples from patients A and B and in donor aortic endothelial cells by reverse transcription–polymerase chain reaction. All three ill recipients developed virus-specific immunoglobulin M. Patient D tested negative for LCMV.

Immunosuppression was reduced in all three ill recipients. Treatment with oral ribavirin was commenced for patient B on May 2. Patients A and C were started on intravenous ribavirin treatment on May 3, with patient C having received oral ribavirin for 2 days prior. Patients A and C were also treated with intravenous immunoglobulin.

All nontransplanted donor organs and tissues were traced and destroyed or sent to CDC for testing, including plasma that was donated 2 days before death. No definitive evidence of rodent exposure was discovered for the donor, although he had spent much time outside along the Mississippi River.

Patient A died on May 11. As of February 20, 2014, patient B had recovered except for mild memory deficits. Patient C was in a nursing facility in fair condition, with ongoing memory deficits and with allograft failure necessitating return to dialysis.

This reported cluster is the fifth LCMV organ transplant-associated cluster documented in the United States, with 14 LCMV-infected organ recipients, including 11 deaths, previously described (1–3). All five clusters have occurred in the past decade. The three previous cornea recipients also did not develop LCMV infections (1,2). Physicians and public health practitioners should be aware that organ donors with suspected central nervous system infection, and some with intracranial hemorrhage without evidence of infection, could be infected with LCMV, especially when rodent exposure has occurred. Testing for LCMV should be considered in organ recipients who develop febrile illness, neurologic changes, or multiorgan dysfunction in the early posttransplant period, especially if multiple recipients from the same donor become ill.

The recommended treatment for LCMV infections obtained through organ transplantation includes reduced immunosuppression and ribavirin. The efficacy of ribavirin in these cases has not been determined; however, early detection of LCMV and prompt treatment initiation might improve outcome.


References
Announcement

World Water Day — March 22, 2014

World Water Day, sponsored by the United Nations, is observed each year on March 22. This year, World Water Day focuses on water and energy. Water and energy are closely linked. Worldwide, 1.3 billion persons currently live without electricity (1), 780 million lack access to safe drinking water, and 2.5 billion are without sanitation (2).

To some degree, water is crucial to produce, transport, and use all forms of energy, and these activities vary significantly with water resources. Demand for freshwater and energy will continue to increase significantly over the coming decades, presenting major challenges and straining resources in nearly all regions. This is especially true in developing and emerging economies. Better understanding of the linkages between the water and energy sectors can lead to improved coordination among policymakers, planners, and others in providing and obtaining more efficient and cost-effective water and energy services.

Additional information about World Water Day and ideas on how to get involved are available at http://www.unwater.org/worldwaterday. Information on CDC’s efforts to ensure global access to improved water, sanitation, and hygiene resources is available at http://www.cdc.gov/healthywater/global.

References
QuickStats

FROM THE NATIONAL CENTER FOR HEALTH STATISTICS


From 2007 to 2012, the birth rate for females aged 15–19 years in the United States overall declined by 29%, from 41.5 to 29.4 births per 1,000 females in that age group. Among racial/ethnic populations, declines ranged from 25% for non-Hispanic white females to 39% for Hispanics. Rates decreased 29% for non-Hispanic black females and American Indian/Alaska Natives and 34% for Asian/Pacific Islanders.


Reported by: Brady E. Hamilton, PhD, bhamilton@cdc.gov, 301-458-4653.