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Evaluation of a Neighborhood Rat-Management Program — New York City, December 2007–August 2009

The Norway rat (*Rattus norvegicus*) is a pervasive urban rodent that can carry a variety of pathogens transmissible to humans, bring stress to residents of infested neighborhoods, damage property, and cause financial loss (*1–4*). Several areas of New York City have experienced persistent rat infestation despite a longstanding rat control program that employed property-level inspection and control measures triggered by individual citizen complaints, a common approach in urban areas (*3*). Recognizing the need to address conditions conducive to rat infestation at the community level, in 2007 the New York City Department of Health and Mental Hygiene launched a proactive "rat indexing" (active surveillance) program, using rapid inspections of properties in several Bronx neighborhoods with persistent rat infestation (*5*). The program included repeated, neighborhood-wide inspections; education and enforcement actions to promote rat control measures by property owners; and community outreach. Signs of rat infestation were noted and recorded electronically by inspectors, and records were analyzed to evaluate program effectiveness. After three rounds of indexing over a 21-month period, the percentage of properties with active rat signs (ARS) had declined 54%, and the percentage with severe rat infestation had declined 58%. The indexing approach to rat control subsequently was expanded to other parts of the city. Indexing can be an effective control strategy in urban neighborhoods with persistent rat infestation.

Within the Bronx, a zone consisting of approximately 36,000 privately owned properties was selected for the pilot rat indexing program based on 1) historically high rates of rat infestation found by complaint-based inspections, 2) support from local community boards and elected officials, and 3) expected displacement of rats by a major construction project under way in one section of the zone. The indexing zone included all or part of 11 neighborhoods, as defined by community district boundaries,* with an estimated population of 777,000 residents in 12 square miles (31 square kilometers), a density of 64,750 persons per square mile (24,775 persons per square kilometer). Approximately 37% of indexing zone residents lived below the federal poverty threshold.†

During each indexing round, inspectors walked every block of each neighborhood, using handheld computers loaded with maps of the properties to record inspection findings. Inspectors were instructed to inspect as much of the exterior of the property as could be accessed at the time of inspection, including front, side, and rear yards or garbage areas, looking for any of six different ARS: 1) fresh tracks, 2) fresh droppings, 3) active burrows, 4) active runways and rub marks, 5) fresh gnawing marks, and 6) live rats (*6,7*). Inspectors recorded a severity score for each sign, ranging from zero (sign not present) to three; total ARS scores, including all six signs, could range from zero to 18. For this report, any property with a total ARS score ≥3 was considered to have a severe rat infestation. Inspectors also noted and recorded conditions conducive to rats including accessible "garbage" (poor containerization of food waste resulting in the feeding of rats) or "harborage" (clutter and dense vegetation promoting the nesting of rats).

* Information available at http://www.nyc.gov/html/dcp/html/lucds/cdstart.shtml.

† Information available at http://www.census.gov/prod/cen2000.

INSIDE

Continuing Education examination available at http://www.cdc.gov/mmwr/cme/conted_info.html#weekly.

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What is already known on this topic?

The Norway rat is a pervasive urban pest that is known to carry disease and cause extensive property damage. Active surveillance for rats can help public health professionals assess levels of infestation and track the success of interventions over time.

What is added by this report?

During December 2007–August 2009, New York City conducted three rounds of proactive indexing inspections for rat activity in an area of the Bronx. Results indicated that, among 29,996 properties, the percentage with active rat signs declined 54%, from 9.75% to 4.51%, and the percentage with severe rat infestation decreased 58%, from 0.48% to 0.20%.

What are the implications for public health practice?

Active surveillance for rats, publication of findings, enforcement of abatement orders including charges and fines, and education and outreach to neighborhood groups and property owners all are key components of a health department's rodent control strategy. In addition, by inspecting entire neighborhoods for rat activity, rather than single properties alone, action can be timed to have an impact at the neighborhood level and avoid displacing rats from one property to another.

An official order to abate was mailed to the owner of every private property that failed inspection, along with detailed inspection findings, a rodent control educational guide,[§] and advice tailored to the problems identified by the inspection. Property owners were allowed 5–10 business days to comply with the order, after which a compliance inspection was conducted. An Internet-based rat information portal[¶] was launched in October 2008 to make rat indexing data publicly available for all properties and to promote collective responses to neighborhood-scale infestations. Agencies responsible for public properties were mailed a referral letter and expected to take action and report back in weekly rodent task force meetings. Results of both private and public property inspections were reported on the rat information portal.

When private owners failed to comply with the health department's order, inspectors issued a violation to the owner and deployed staff members licensed as pest control professionals to reinspect and, if needed, apply rodenticide bait. Certain properties with severe garbage or rat harborage conditions were cleaned by health department personnel. The costs of these services were charged to the property owner. Automated processes helped to ensure timely issuance of orders and fines, scheduling of inspections and reinspections, and tracking of progress.

Education and outreach were conducted throughout the Bronx. A coordinator met with local elected officials and community boards and built relationships with community-based organizations. A local Bronx weatherizing (sealing and insulating) company was engaged to assist in rodent prevention outreach efforts and to launch a "Rodent Academy" for building owners, managers, and superintendents.

[§] Available at http://www.nyc.gov/html/doh/downloads/pdf/pest/rodent_control.pdf.

[¶] Available at http://www.nyc.gov/rats.

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From December 3, 2007, to August 21, 2009, three rounds of indexing were completed. In round 1, a total of 35,691 properties were indexed. Because indexing inspections were simpler and more efficient than the typical complaint-based inspections,** the six inspectors were able to index an average of 88 properties per inspector per work day, compared with an average of 10 properties per inspector per work day for typical complaint-based inspections. Accuracy of the inspections was evaluated by performing the more thorough complaint-based inspection methods on 49 properties that were randomly selected from the 35,691 properties in the indexing zone. Fourteen of the 49 properties were found to have ARS after the more thorough inspection. Of those 14 properties, 12 (86%) had been correctly identified as having ARS by the indexing program; two properties that had passed the indexing inspection failed the complaint-based inspection.

Because of changes in the indexing zone and properties lost to follow-up, 5,695 (16%) of the properties inspected in round 1 (including 1.98% found to have ARS) were not reinspected in round 2 and round 3. The remaining 29,996 (84%) properties, including 2,926 (9.75%) with ARS in round 1, comprised the study cohort for this report (Table). In round 2, the number of properties with ARS declined to 1,748 (5.83%), and in round 3, the number declined to 1,354 (4.51%). From round 1 to round 3, the number of properties with ARS declined by 1,572 or 54% ($p<0.001$). The percentage of properties with severe infestation (total severity score ≥3) declined 58%, from 0.48% in round 1 to 0.20% in round 3 ($p<0.001$).

Among the 2,926 properties with ARS in round 1, a total of 371 did not receive a compliance inspection because of various factors.†† Of the remaining 2,555 properties, a total of 1,153 (45%) failed their immediate compliance inspection. Of these, 361 (31%) still had ARS in round 2. In contrast, among the 1,402 (55%) properties with ARS in round 1 that passed their compliance inspection, only 224 (16%) still had ARS during round 2 indexing ($p<0.001$).

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** During indexing, inspectors looked for the same six ARS as during complaint-based inspections. However, indexing inspectors spent less time because they did not inspect the interior of buildings or individual units inside a multifamily building.

†† Including the following: not eligible because city-owned, no owner found, no access to the property, dangerous conditions, and employee error.

TABLE. Properties with active rat signs (ARS)* and severe infestations,† by neighborhood-wide indexing round — Bronx, New York, December 3, 2007–August 21, 2009

Indexing round	Period	Active rat signs %	Severe infestations %
Round 1	December 3, 2007–June 23, 2008	9.75	0.48
Round 2	July 14, 2008–December 30, 2008	5.83	0.27
Round 3	January 20, 2009–August 21, 2009	4.51	0.20

* Inspectors looked for any of six signs: 1) fresh tracks, 2) fresh droppings, 3) active burrows, 4) active runways and rub marks, 5) fresh gnawing marks, and 6) live rats.

† Inspectors recorded a severity score for each ARS ranging from zero (sign not present) to three; total ARS scores ranged from zero to 18. Any property with a total ARS score ≥3 was considered to have a severe infestation.

Editorial Note

This report describes how New York City found that rounds of inspections conducted in neighborhoods, combined with prompt communication with owners, publication of findings, and fines for noncompliance, reduced the prevalence and severity of rat infestations in a large area with a history of severe rat problems. Urban rat control programs should include a comprehensive survey assessing ARS and environmental conditions conducive to rats and conduct inspections by neighborhood rather than by individual property alone (*3,5–10*). Directly comparable data on properties with ARS were not collected from other New York City neighborhoods during the same period as the Bronx intervention. However, compared with the rat indexing data in the Bronx, much lower percentage declines in the number of rat sighting complaints under the complaint-based system were observed from 2007 to 2009 in Brooklyn (16%) and Queens (10%). Given the results from rat indexing in the Bronx, New York City has expanded the program to include all of Manhattan and limited neighborhoods in Queens and Brooklyn, in addition to the Bronx.

Previous large-scale rat control initiatives were conducted in the 1990s in Baltimore, Maryland, and Boston, Massachusetts. Baltimore found that a decrease in the prevalence of rat infestation could be achieved, but the results were largely attributable to a government-subsidized baiting program (*8*). Boston tracked sanitation deficiencies and signs of rodent activity on properties within the area of the "Big Dig" highway construction corridor and in surrounding neighborhoods in periodic surveys and achieved an 87% reduction in referrals for rat activity and sanitation violations. Boston's program also included extensive baiting services in and around the construction area and sewer lines (*3,9*).

New York City's rat indexing program is similar to the rat control programs in Baltimore and Boston in that a neighborhood-wide survey was used to assess a baseline prevalence of infestation (round 1) before the intervention. The New York City program differed from those of the other two cities in

its focus on rapid indexing inspections and the expectation that property owners would respond to rat infestations rather than rely mainly on extensive baiting by the city government.

The findings in this report are subject to at least two limitations. First, although owners of properties with ARS were advised to inspect, clean, remove garbage and harborage, and hire a licensed pest professional, the specific remediation actions of property owners were not recorded. Second, the rat indexing program did not include assessment or treatment of rat populations in sewers, subways, or other subsurface infrastructures. Some properties might have passed inspection while harboring subsurface rats that would later emerge to forage in the indexing zone. In such cases, the reduction in ARS might have been temporary, and neighborhoods might have continued to experience infestations even if rat activity had been abated at the surrounding properties (*3,9*).

All municipal rat control programs recognize the importance of outreach and education to neighborhoods (*3,5,8,9*), but the actual impact of the New York City outreach on rat remediation is unknown. Outreach has the potential to increase knowledge of best practices in rat management, increase confidence in the community in the potential to control rats, and promote simultaneous actions that increase the likelihood of sustainable community success. Rat management at both the property and neighborhood level is best achieved through removal of food sources and harborage conditions, combined with the judicious use of rodenticides applied by trained pest professionals.

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Chikungunya Outbreak — Cambodia, February–March 2012

Chikungunya virus (CHIKV) is an alphavirus transmitted to humans through the bite of infected *Aedes* mosquitoes (*1*). CHIKV causes fever and usually is not fatal, but can cause debilitating joint pains or, in rare instances, severe illness. The East/Central/South African strain of chikungunya has been emerging in Asia since 2006, first in the Indian subcontinent, then Thailand. This report describes the characteristics of a local outbreak linked with chikungunya reemergence in a rural Asian setting. Sporadic cases of chikungunya were identified in Cambodia in 2011 (*2*). Antibodies to CHIKV have been detected in serum collected in Cambodia in 2007, but the strain could not be identified for those cases (U.S. Naval Medical Research Unit 2, unpublished data, 2012). On March 7, 2012, several cases of rash with fever were reported among village residents of Trapeang Roka in Kampong Speu Province, Cambodia. Subsequent field investigation revealed that four of six blood samples from affected persons were positive for CHIKV by polymerase chain reaction (PCR) at U.S. Naval Medical Research Unit 2 in Phnom Penh. Investigators from the Cambodian Communicable Disease Control Department, National Malaria Center, Institut Pasteur du Cambodge (IPC), local health centers, and village authorities conducted a seroprevalence study of village residents on March 26 to gather information for response planning and control efforts. The outbreak affected families throughout the village, and 44.7% of the population tested had evidence of infection by CHIKV, which affected all age groups. Public health agencies and policymakers in affected and nearby unaffected areas of Asia and elsewhere should be alert to the potential spread and reemergence of CHIKV.

Trapeang Roka has a population of approximately 695 persons in 134 houses; most adults are farmers or factory workers. For the survey, the village was divided into six sectors created by the roads crossing through it. One team of investigators was assigned to each sector, and a central blood-sampling station was established. Teams worked outward from the center of the village, going from house-to-house. In each house, all occupants present were interviewed. Those who were in the field or factory rather than at home were interviewed when they returned to the village in the evening. Informed consent was obtained in the Khmer language from all adult residents and parents or guardians of children; none refused to participate. A standardized questionnaire was used to gather information on demographics and recent (since the February 14–15 rains) or current self-reported symptoms including joint pains, fever >38°C (axillary or subjective), and rash. A clinical case was defined as one or more of these symptoms in a person from this village with onset after February 14 through March 26, 2012. Blood specimens were obtained for serologic testing; dried blood spots were collected from all persons surveyed and venous samples were collected from febrile patients. Confirmed cases had a positive laboratory test.

At IPC's Virology Unit, immunoglobulin M (IgM) antibody capture enzyme-linked immunosorbent assay (MAC-ELISA) testing was performed to detect anti-CHIKV IgM (*3*). Serologic testing also was performed by MAC-ELISA for recent infection by dengue (DENV) and Japanese encephalitis B (JEV) viruses (*4*). Positive serology for DENV and JEV pointed to recent flavivirus infection, which might present similar symptoms. Patients who were febrile at investigation also were tested by real-time reverse transcriptase PCR for CHIKV and also DENV and JEV. The 91 persons with recent or current symptoms who were negative for CHIKV IgM were screened for malaria by PCR.

The survey included 98 (73.1%) households distributed throughout the village. The team interviewed 425 persons (61.1% of village population; male:female ratio = 0.82); mean age was 26.4 years (range: 1 month–87 years; interquartile range: 10–39 years). Among the 425 persons interviewed were 96 (22.6%) farmers, 89 (20.9%) students, 91 (21.4%) factory workers, and 73 (17.2%) persons who stayed at home; the remaining 76 persons were preschool-aged children, construction workers, vendors, or pig farmers.

Of the 425 persons interviewed, 312 (73.4%) reported that they had at least one of the three defining symptoms since the rains of February 14–15, 2012 (Table). Among those 312 persons, 173 (55.4%) took leave from school or work. One death during that period was identified retrospectively in a woman aged 33 years with no known underlying disease. The woman had fever and intense joint pain, developed neurologic signs, and died within 2 hours of hospital admission. No samples were available for testing. Her two children were symptomatic, CHIKV IgM-positive, and seronegative for dengue and JEV.

MAC-ELISA tests identified CHIKV IgM in 188 (44.2%) of the 425 persons. Of the six febrile persons, venous blood samples could be analyzed from four: two had positive PCR results for CHIKV, bringing the total that were laboratory confirmed to 190 (44.7%) CHIKV-positives (Table). One person with fever and joint pains was positive for DENV-4. PCR results found traces of *Plasmodium* spp. nucleic acid indicative of incubating or recent malaria in one afebrile person. Of the 190 CHIKV-positive persons identified, 10 (5.3%) reported having none of the three defining symptoms.

TABLE. Chikungunya virus (CHIKV) serologic or polymerase chain reaction status and self-reported symptoms, recent or current, since February 14, 2012 — Trapeang Roka, Cambodia, March 2012

	CHIKV-positive residents (n = 190)		CHIKV-negative residents (n = 235)		Total (N = 425)		Relative risk*	
Symptoms	No.	(%)	No.	(%)	No.	(%)	RR	95% CI
Fever	163	(85.8)	97	(41.3)	260	(61.2)	2.1	(1.8–2.4)
Skin rash	139	(73.2)	52	(22.1)	191	(44.9)	3.3	(2.6–4.3)
Joint pain	159	(83.7)	100	(42.5)	259	(60.9)	2.0	(1.7–2.3)
Any two of the symptoms	178	(93.7)	134	(57.0)	312	(73.4)	1.6	(1.5–1.9)
All three symptoms	126	(66.3)	37	(15.7)	163	(38.3)	4.2	(3.1–5.8)

Abbreviations: RR = relative risk; CI = confidence interval.
* Relative risk for self-reported symptom in CHIKV-positive residents and signs in CHIKV-negative residents.

The onset of the epidemic was protracted. Among 140 persons with self-reported fever who recalled an onset date and who had positive results for CHIKV (IgM or PCR), most reported onset occurring approximately 3–5 weeks after a 2-day period of rain (Figure 1). The epidemic curve suggested that the CHIKV outbreak began 3 weeks after the rains, lasted about 3 weeks, and was on the verge of ending when this serosurvey was conducted on March 26, 2012.

Laboratory results were analyzed by age group (Figure 2). The analysis revealed a 40% IgM seroprevalence among persons aged ≤5 years; for persons aged 6–45 years, IgM seroprevalence was approximately 50%, declining sharply for each age group after that. Circulation of at least two other viruses was identified, including 11 cases of DENV, seven cases of JEV, and 15 cases of undistinguishable flaviviruses (among these, 10, four, and eight cases, respectively, occurred among CHIKV-positive cases).

During a small-scale entomologic assessment conducted during March 29–30, 2012, 123 mosquitoes were collected in Trapeang Roka and 651 mosquitoes were collected from a wider area, including two nearby villages that also reported cases, based on syndromic data. *Aedes aegypti* comprised 41.4% and 53% of mosquitoes collected in the two areas, respectively, and *Culex* species comprised 21.1% and 13.8%, respectively; the rest were *Anopheles* species, and no *Aedes albopictus* were identified.

What is already known on this topic?

The East/Central/South African strain of chikungunya has been emerging in Asia since 2006, first in the Indian Subcontinent, then Thailand. Little is known of the transmission dynamics of this chikungunya reemergence in Asia.

What is added by this report?

This report describes the epidemiology of a chikungunya outbreak in a Cambodian village. The outbreak occurred 3 weeks after rains and lasted 3 weeks. The outbreak affected the entire village and all age groups; 44.7% of the population tested had evidence of infection by chikungunya virus (CHIKV). The investigation found an end-of-outbreak clinical attack rate of 73.4%, and a 5.3% proportion of asymptomatic infections.

What are the implications for public health practice?

Awareness of the location and extent of CHIKV infection can help guide health planning efforts and prioritization of resources for control, both in Cambodia and nearby, currently unaffected countries such as Laos or Vietnam.

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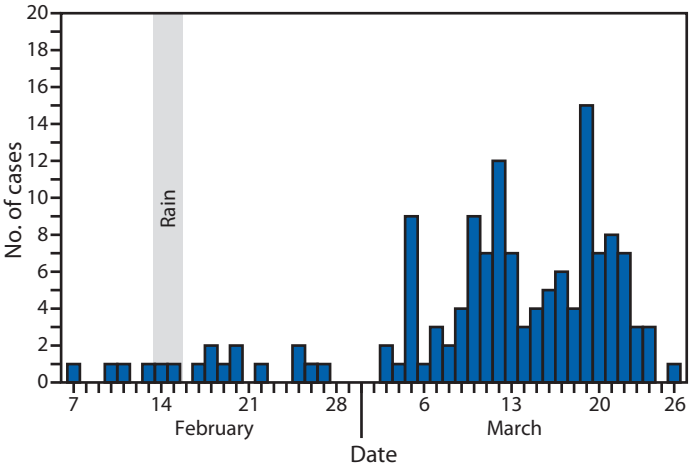
Editorial Note

The serosurvey in Trapeang Roka on March 26, 2012, documented a CHIKV outbreak in a medium-size rural Cambodian community of 695 residents. It is the first CHIKV serosurvey in Cambodia since the initial isolation of CHIKV in 1961, and the first known serosurvey conducted during a chikungunya outbreak in Southeast Asia. The experience of at least one symptom since the rains was reported by 73.4% of interviewees. CHIKV prevalence was 44.7%, and the prevalence of asymptomatic CHIKV infection was 5.3%. A temporal association was noted between an unusually large rainfall on February 13–14 and the subsequent outbreak of cases of CHIKV beginning 3 weeks later and lasting for 3 weeks.

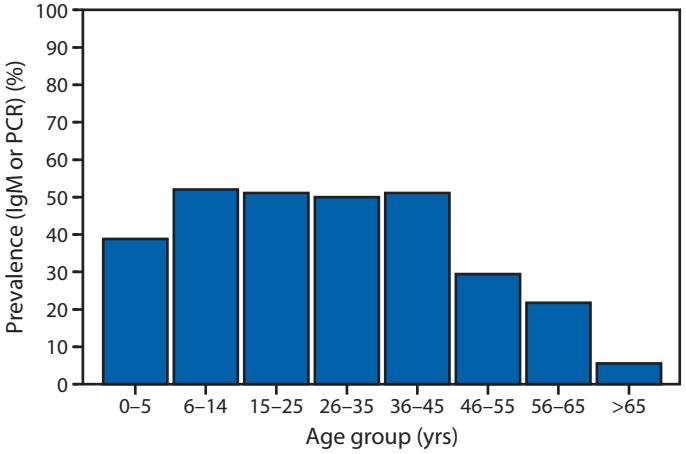
Most persons who were CHIKV-positive cases had clinical signs and symptoms consistent with chikungunya. The level of seroprevalence in this outbreak is consistent with levels from CHIKV serosurveys conducted during outbreaks

FIGURE 1. Onset of fever among village residents who recalled an onset date and had laboratory confirmed cases of chikungunya virus (CHIKV) infection* — Trapeang Roka, Cambodia, February and March 2012



* Among these 140 persons with self-reported fever who recalled an onset date and who had positive laboratory results for CHIKV, most reported onset occurring approximately 3–5 weeks after a 2-day period of rain.

FIGURE 2. Prevalence of chikungunya immunoglobulin M (IgM) or polymerase chain reaction (PCR) positivity, by age group — Trapeang Roka, Cambodia, March 2012



on Reunion Island (35%) (*5,6*) and Mayotte (38%–45%) (*7*), but lower than that of Lamu Island, Kenya (75%) (*8*). Seroprevalence was lower in persons aged ≥45 years. This might be the result of immunity acquired when the CHIKV Asian strain circulated in Cambodia in the 1960s. The only other published CHIKV serologic study from the Mekong region was conducted in the Bangkok area in 1998–1999 (*9*), before the emergence of the East/Central/South African genotype. In that study, the prevalence in mothers (using cord blood) was 31.6% and 45% in women aged >35 years. This new genotype, which emerged in Africa in 2004 and is emerging in Cambodia, is well known to have an E226V mutation that is more efficiently transmitted by *Ae. albopictus.*

The findings in this report are subject to at least four limitations. First, cocirculating arboviruses (i.e., dengue and JEV) share the same vector and cause similar symptoms. Some symptoms might have been caused by dengue or JEV infection. Some of the clinical cases, therefore, could have been caused by undetected viruses other than CHIKV, and this could explain why clinical cases outnumbered laboratory-confirmed cases. Second, CHIKV IgM seroprevalence might have been underestimated because of suboptimal sensitivity of tests performed on dried blood spots. Third, IgM screening might miss infection still in the incubation period. Finally, the presence of a large team of medical investigators in a rural village might have stimulated over-reporting of symptoms. These also might account for the difference between the clinical attack rate and CHIKV IgM-positive prevalence (approximately 30%).

CHIKV affects all age groups. While conducting the survey, investigators also provided education to help control the outbreak, which had nearly ended. The main vector identified in the historic Reunion Island outbreak was *Ae. albopictus*, the Asian tiger mosquito, but none were found in the current study area. The fact that *Ae. aegypti* were found, but not *Ae. albopictus*, is important for researchers working on biomolecular aspects of transmission of the new variant chikungunya. Awareness of the location and extent of chikungunya virus infection can help guide health planning efforts and prioritization of resources for control, both in Cambodia and nearby, currently unaffected countries such as Laos or Vietnam.

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Update on Vaccine-Derived Polioviruses — Worldwide, April 2011–June 2012

In 1988, the World Health Assembly resolved to eradicate poliomyelitis worldwide (*1*). One of the main tools used in polio eradication efforts has been the live, attenuated oral poliovirus vaccine (OPV). This inexpensive vaccine is administered easily by mouth, makes recent recipients resistant to infection by wild polioviruses (WPVs), and provides long-term protection against paralytic disease through durable humoral immunity. Nonetheless, rare cases of vaccine-associated paralytic poliomyelitis can occur both among immunologically normal OPV recipients and their contacts and among persons who are immunodeficient. In addition, vaccine-derived polioviruses (VDPVs) can emerge to cause polio outbreaks in areas with low OPV coverage and can replicate for years in persons who are immunodeficient. This report updates previous surveillance summaries (*2,3*) and describes VDPVs detected worldwide during April 2011–June 2012. In 2011, a new outbreak of circulating VDPVs (cVDPVs) was identified in Yemen; a second VDPV isolate, related to a previously reported VDPV isolate (*2*), signaled an outbreak in Mozambique; and VDPV circulation reemerged in Madagascar. An outbreak that began in Somalia in 2008 continued until December 2011. Outbreaks in Nigeria and the Democratic Republic of the Congo (DRC) identified in 2005 and 2008, respectively, continued in 2012. Niger experienced a new cVDPV importation from Nigeria in 2011. Twelve newly identified persons in six middle-income countries were found to excrete immunodeficiency-associated VDPVs (iVDPVs), and VDPVs were found among healthy persons and environmental samples in 13 countries. To prevent VDPV emergence and spread, all countries should maintain high vaccination coverage against all three poliovirus serotypes; OPV use will be discontinued worldwide once all WPV transmission is interrupted (*4*).

Properties of VDPVs

VDPVs can cause paralytic polio in humans and have the potential for sustained circulation. VDPVs resemble WPVs biologically (*2*) and differ from most vaccine-related poliovirus (VRPV) isolates by having genetic properties consistent with prolonged replication or transmission. VDPVs were first identified by sequence analyses of poliovirus isolates. Because poliovirus genomes evolve at a 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 at least 1 year in one or more persons after administration of an OPV dose and are VDPVs. One year is substantially longer than the normal period of vaccine virus replication of 4–6 weeks in an OPV recipient.

Three poliovirus serotypes have been identified: types 1, 2, and 3 (PV1, PV2, and PV3). Poliovirus isolates are grouped into three categories, based on the extent of divergence compared with the corresponding OPV strain: 1) VRPVs (<1% divergent [PV1 and PV3] or <0.6% divergent [PV2]); 2) VDPVs (VRPVs >1% divergent [PV1 and PV3] or >0.6% divergent [PV2]); and 3) WPVs (WPV1, WPV2, and WPV3, no genetic evidence of derivation from any vaccine 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) who have prolonged VDPV infections; and 3) ambiguous VDPVs (aVDPVs), which are either clinical isolates from persons with no known immunodeficiency or sewage isolates whose source is unknown (*2*).

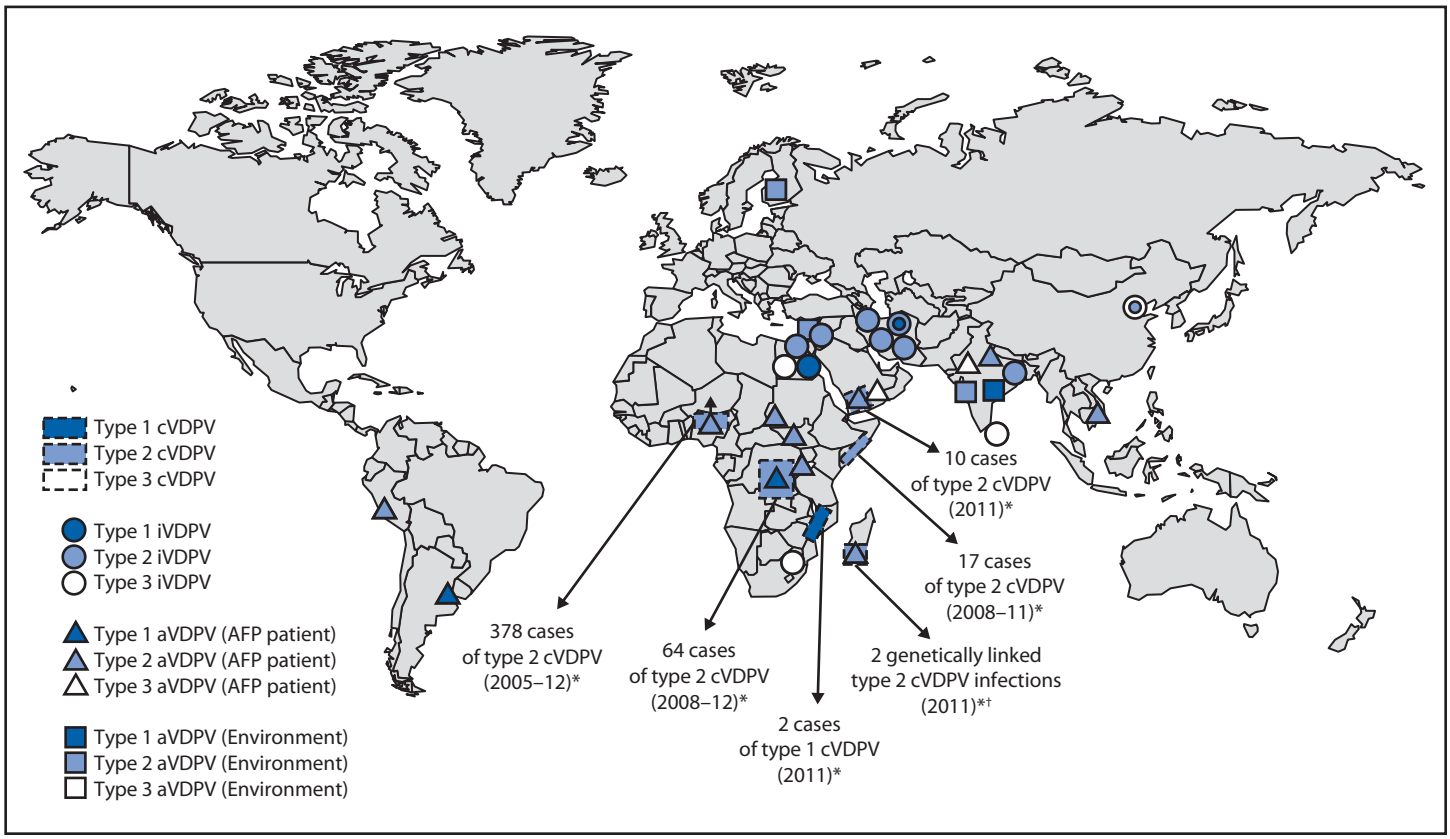
Virologic Testing for VDPVs

All poliovirus isolates are characterized by laboratories of the Global Polio Laboratory Network (GPLN) (*5*). 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 occur early in VDPV emergence (*2*). The original rRT-PCR procedure specifically amplified sequences typical of OPV strains; newer methods amplify sequences of potential VDPVs by targeting sequences that typically revert during replication of OPV in the human intestine. The rRT-PCR methods have been transferred to 60 of 146 GPLN laboratories (*5*). Candidate VDPVs following rRT-PCR screening are sequenced routinely in the VP1 region; the complete genome is sequenced if required for higher-resolution analysis.

cVDPVs

The number of countries with indigenous cVDPV circulation remained unchanged at six since the July 2009–June 2011 reporting period (*2*). Outbreaks in Afghanistan, Ethiopia, and India appeared to have stopped; outbreaks in DRC and Somalia continued; a large outbreak in Nigeria abated; new outbreaks were detected in Mozambique and Yemen, and genetic evidence indicated renewed VDPV circulation in Madagascar. Apart from a cVDPV exported from Nigeria into neighboring Niger, no cVDPVs were exported from the countries of emergence. In all but Mozambique, the emerging cVDPVs were PV2 (Figure).

FIGURE. Vaccine-derived polioviruses (VDPVs) detected worldwide, April 2011–June 2012



Abbreviations: cVDPV = circulating VDPV; iVDPV = immunodeficiency-associated VDPV; aVDPV = ambiguous VDPV; AFP = Acute flaccid paralysis.
* Spread of cVDPVs followed the elimination of the corresponding serotype of indigenous wild poliovirus, but with continued introduction of oral poliovirus vaccine into communities with growing immunity gaps. All of the cVDPV outbreaks were detected first by the laboratory, using sequence data and evolutionary analyses.
† Circulation was inferred because two isolates from healthy children shared a common VDPV2 ancestor but were genetically divergent.

DRC. Circulation of cVDPV2 in DRC continued in 2012, with a total of 64 cases detected since 2008. Since April 2011, 28 cVDPV2 isolates (0.7%–3.5% divergent) from persons with acute flaccid paralysis (AFP) have been detected, all in Katanga Province, where reported OPV coverage was low (*6*). An additional aVDPV2 isolate (0.7% divergent) from an AFP patient was detected in Katanga. As in Nigeria during 2005–2011, multiple independent VDPV2 emergences were identified in DRC.

Madagascar. Serial and concurrent emergences of VDPVs were first reported in Madagascar during 2002 (*7*). VDPV surveillance has been enhanced by virologic testing of stools collected from healthy children in southern Madagascar. Two recent genetically related VDPV isolates provide evidence of circulation.

Mozambique. A second VDPV isolate (4.3% divergent) related to a previously reported aVDPV isolate (*2*) was identified in a child with AFP, providing evidence of circulation during this reporting period.

Niger. One cVDPV2 (5.2% divergent) was isolated from a patient in southern Niger, near the border with Nigeria, with onset of AFP in November 2011. The isolate was closely related to cVDPVs circulating in nearby Kano and Jigawa states, Nigeria. As with the five previous cVDPV2 importations from Nigeria detected since May 2006 (*2*), no secondary cases were found in Niger.

Nigeria. Since 2005, a total of 378 AFP cases associated with a cVDPV2 outbreak (0.7%–6.5% divergent) were reported in 11 northern and three north-central states of Nigeria where routine vaccination with trivalent OPV (tOPV) coverage was low (<60%) (*2*). The outbreak peaked (153 cases) in 2009; 27 cases were detected in 2010, and 35 cases were detected in 2011. Only two cases had been detected as of June 2012, but 30 additional genetically distinct cVDPV2 isolates were obtained from environmental samples in the northern states of Kano and Sokoto. The outbreak is associated with approximately 25 independent VDPV2 emergences, at least seven of which led to cVDPV2 transmission (*2*).

Somalia. VDPV2 has been detected in Somalia since 2005. During April–December 2011, cVDPV2 (1.0%–3.5% divergent) were isolated from three patients with AFP and 14 contacts in the regions surrounding Mogadishu; all were derived from a single emergence.

What is already known on this topic?

Genetically divergent vaccine-derived polioviruses (VDPVs) are detected by poliovirus surveillance and have biological properties indistinguishable from wild polioviruses. High coverage with poliovirus vaccine can eradicate all WPVs and prevent circulating VDPV (cVDPV) outbreaks, but prolonged immunodeficiency-associated VDPV (iVDPV) infections will occur as long as live, attenuated oral poliovirus vaccine (OPV) is used.

What is added by this report?

During April 2011–June 2012, although cVDPV outbreaks in three countries appear to have stopped, and the large outbreak in Nigeria has been reduced sharply, outbreaks continued in the Democratic Republic of the Congo and Somalia, and new outbreaks were detected in three countries. Twelve new prolonged iVDPV infections were detected, with increasing numbers found in developing and middle-income countries. Approximately 85% of VDPVs have been type 2.

What are the implications for public health practice?

Circulation of VDPVs does not present an insurmountable obstacle to polio eradication but instead is a symptom of low poliovirus vaccine coverage. Circulating VDPV outbreaks can be prevented and controlled by high OPV coverage. By contrast, only cessation of OPV use will prevent prolonged iVDPV infections. The World Health Organization has responded to the global VDPV risk by 1) increasing the frequency of trivalent OPV (tOPV) supplemental immunization activities (SIAs) in countries with low routine tOPV coverage, 2) considering a shift to bivalent OPV (type 1 plus type 3) as soon as possible, 3) encouraging the ongoing shift to inactivated poliovirus vaccine, 4) enhancing VDPV surveillance, and 5) encouraging development of antiviral drugs to clear prolonged iVDPV infections.

Yemen. During April–October 2011 cVDPV2 (0.6%–1.6% divergent) were isolated from nine patients with AFP and one contact. The outbreak was derived from at least two independent emergences.

iVDPVs

Since the introduction of OPV in 1961, approximately 65 persons with primary immunodeficiencies have been found worldwide to be excreting iVDPVs (indicating prolonged infections); the majority of these immunodeficiencies 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, detection of iVDPV infections increased from two during January 2008–June 2009 (*3*), to nine during July 2009–June 2011 (*2*), and to 12 during April 2011–June 2012.

China. A girl aged 11 months with common variable immunodeficiency developed AFP in February 2012, following 3 OPV doses. The patient was coinfected with iVDPV2 and iVDPV3.

Egypt. Surveillance for VDPVs in Egypt was enhanced during the reporting period by screening of persons with primary immunodeficiencies. An iVDPV1 was isolated from a boy aged 18 months with agammaglobulinemia after onset of AFP in May 2011, an iVDPV3 was isolated from a child aged 21 months with primary immunodeficiency after onset of AFP in April 2011, and a girl aged 3 months with agammaglobulinemia was found to be infected with iVDPV2 in 2011 but did not develop AFP. All three patients died.

India. A girl aged 6 months with primary immunodeficiency in West Bengal was infected with iVDPV2.

Iran. Iran has maintained sensitive clinical and laboratory surveillance to screen persons with primary immunodeficiencies for poliovirus infections. During April 2011–June 2012, four AFP patients were found to be excreting iVDPVs. A boy aged 6 years with primary immunodeficiency infected with an iVDPV2 developed AFP in May 2011. A boy aged 15 months with primary immunodeficiency and infected with an iVDPV2 developed AFP in June 2011, a boy aged 25 months with primary immunodeficiency who was coinfected with an iVDPV1 and iVDPV2 developed AFP in December 2011, and a boy aged 6 months with primary immunodeficiency and infected with an iVDPV2 developed AFP in March 2012. The last two boys died.

South Africa. A boy aged 10 months with agammaglobulinemia and infected with an iVDPV3 developed AFP in September 2011 and subsequently died.

Sri Lanka. A girl aged 8 years with common variable immunodeficiency and infected with an iVDPV3 developed AFP in 2011; she remained alive through mid-2012, with a last VDPV-positive specimen collected in March 2012.

West Bank and Gaza. A boy aged 1 year with severe combined immunodeficiency who had not developed AFP was found to be infected with an iVDPV2 in 2011. The patient died of immunodeficiency complications in January 2012.

aVDPVs

During April 2011–June 2012, aVDPVs were isolated in 12 countries (Table). The most divergent aVDPVs were continuations of lineages previously detected in sewage samples in Finland and Israel, two countries with high (>90%) polio vaccination coverage. The persons infected with the corresponding aVDPVs have not been identified. Detection of aVDPVs in settings (including local pockets) with low (<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.

TABLE. Vaccine-derived polioviruses (VDPVs) detected worldwide, April 2011–June 2012

Category	Country	Year(s) detected*	Source (total cases or specimens)†	Serotype	No. of isolates§ April 2011–June 2012			VP1 divergence from Sabin OPV strain (%)	Routine coverage with 3 doses of poliovirus vaccine (%)¶	Estimated duration of VDPV replication**	Current status (date of last outbreak case, last patient isolate, or last environmental sample)
					Cases	Contacts	Non-AFP Source				
cVDPV††	DRC§§	2008–2012	Outbreak (64 cases)	2	28	—	—	0.7–3.5	78	4 yrs	April 4, 2012
	Madagascar¶¶	2011	Circulation (2 specimens)	2	—	—	2	3.3–3.7	88	3 yrs	May 20, 2011
	Mozambique***	2011	Outbreak (2 cases)	1	1	—	—	3.0–4.3	73	4 yrs	June 2, 2011
	Niger†††	2011	Importation	2	1	—	—	5.2	44	—	November 11, 2011
	Nigeria§§§	2005–2012	Outbreak (378 cases)¶¶¶	2	27	1	23	0.7–6.5	73	7.5 yrs	April 8, 2012
	Somalia****	2008–2011	Outbreak (17 cases)	2	3	5	—	0.7–3.5	49	3.5 yrs	December 10, 2011
	Yemen	2011	Outbreak (9 cases)	2	9	2	—	0.7–1.6	81	1.5 yrs	October 5, 2011
iVDPV††††	China	2012	AFP patient CVID	2 3	1	—	—	1.3 1.6	96	1 yr 1 yr	April 9, 2012
	Egypt	2011	Non-AFP patient AGG	2	—	—	1	1.4	96	0.6 yrs	June 3, 2011
	Egypt	2011	AFP patient AGG	1	1	—	—	2.1	96	1.5 yrs	May 2, 2011
	Egypt	2012	AFP patient PID	3	1	—	—	4.2	96	2 yrs	July 16, 2012
	India	2012	AFP	2	1	—	—	1.2–2.2	70	~2 yrs	June 2012
	Iran	2011	AFP patient PID	2	1	—	—	1.4	99	1.5 yrs	May 14, 2011
	Iran	2011	AFP patient PID	2	1	—	—	2.7	99	2.5 yrs	June 3, 2011
	Iran	2011	AFP patient PID	1 2	1	—	—	2.7 3.3	99	3 yrs	December 14, 2011
	Iran	2012	AFP patient PID	2	1	—	—	1.4	99	1.5 yrs	March 3, 2012
	South Africa	2011	AFP patient AGG	3	1	—	—	1.9	73	2 yrs	September 17, 2011
	Sri Lanka	2011	AFP patient CVID	3	1	—	1	1.9	99	2 yrs	March 12, 2012
	West Bank and Gaza	2011	Non-AFP patient SCID	2	—	—	1	1.2	—	1 yr	November 9, 2011

See table footnotes on page 745.

Argentina. An aVDPV1 was isolated in May 2011 from a paralyzed girl aged 15 months who had received 2 doses of OPV. Final diagnosis was botulism, and the child was no longer excreting VDPV.

DRC. An aVDPV1 (0.7% divergent) was isolated from an AFP patient with no known immunodeficiency in December 2011.

Finland. A highly divergent aVDPV2 (15.4%) was isolated from sewage samples collected in July 2011. This aVDPV2 isolate was related to aVDPV2 isolates detected during 2008–2011 in sewage samples, and nearly equivalent in divergence to aVDPV1 isolates and aVDPV3 isolates likely derived from a single tOPV dose (*2*).

India. In 2011, genetically distinct aVDPV2s (0.7%–1.1% divergent) were isolated from five AFP patients with no known immunodeficiency, and an aVDPV3 (1.5% divergent) was isolated from another AFP patient. In addition, both aVDPV1 (1.2% divergent) and aVDPV2 (0.7%–1.1% divergent) were isolated from sewage samples.

Israel. Two genetically distinct groups of highly divergent aVDPV2s had been detected in sewage samples in 1998 (group 1, 15.6%–16.2% divergent) and 2006 (group 2, 10.7%–11.2% divergent) (*2,8*). Group 1 virus was detected in sewage samples in September 2011. Group 2 virus has been not been detected in sewage samples since March 2011.

Madagascar. An aVDPV2 (0.7% divergent) genetically distinct from the cVDPV2s described in Madagascar was isolated from an unvaccinated healthy child in southern Madagascar.

Nigeria. Two aVDPV2s (0.7%–1.1% divergent) were isolated in November 2011 and May 2012 from two unvaccinated AFP patients in Niger and Edo states, providing evidence of

TABLE. (Continued) Vaccine-derived polioviruses (VDPVs) detected worldwide, April 2011–June 2012

Category	Country	Year(s) detected*	Source (total cases or specimens)†	Serotype	No. of isolates§ April 2011–June 2012			VP1 divergence from Sabin OPV strain (%)	Routine coverage with 3 doses of poliovirus vaccine (%)¶	Estimated duration of VDPV replication**	Current status (date of last outbreak case, last patient isolate, or last environmental sample)
					Cases	Contacts	Non-AFP Source				
aVDPV	Argentina	2011	AFP patient	1	1	—	—	1.1	95	<1 yr	May 15, 2011
	Burundi	2011	AFP patient	2	1	—	—	0.7	94	<1 yr	December 15, 2011
	DRC	2011	AFP patient	1	1	—	—	0.7	78	<1 yr	December 20, 2011
	Finland§§§§	2008–2011	Environment	2	—	—	1	>11	99 (IPV)	>15 yrs	July 25, 2011
	India	2011	AFP patients	2	4	—	—	0.7–1.1	70	0.5–1 yr	November 25, 2011
		2011	AFP patient	3	1	—	—	1.3–1.5		1–1.5 yr	October 7, 2011
		2011	Environment	2	—	—	6	0.7–1.1		0.5–1 yr	April 2011
		2011	Environment	1	—	—	1	1.2		~1 yr	April 2011
		2012	Environment	2	—	—	4	0.7–1.1		0.5–1 yr	May 2012
	Israel¶¶¶¶	1998–2011	Environment	2	—	—	8	15.6–16.2	94 (IPV)	<15 yrs	September 20, 2011
	Madagascar	2011	Healthy child	2	—	—	1	0.7	88	<1 yr	May 20, 2011
	Nigeria	2011–2012	AFP patient	2	2	—	—	0.7–1.1	73	0.5–1 yr	May 22, 2012
			Environment	2	—	—	2	0.7–0.8		<1 yr	May 28, 2012
	Peru	2011	AFP patient	2	1	—	—	2.2	91	2 yrs	April 11, 2011
	South Sudan	2012	AFP patient	2	1	—	—	1.1	46	~1 yr	February 24, 2012
	Sudan	2012	AFP patient	2	1	—	—	0.7	93	<1 yr	April 1, 2012
	Viet Nam	2012	AFP patient	2	1	—	—	0.7	96	<1 yr	February 14, 2012
	Yemen	2011	AFP patients	2	2	—	—	1.0–1.1	81	~1 yr	February 2, 2012
		2012	AFP patient	3	1	—	—	2.3		2 yrs	April 27, 2012

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; CVID = common variable immunodeficiency; DRC = Democratic Republic of the Congo; PID = primary immunodeficiency; SCID = severe combined immunodeficiency; VP1 = viral protein 1 (the major viral surface protein).

* Total years detected and cumulative totals for previously reported cVDPV outbreaks (Democratic Republic of the Congo, Ethiopia, Nigeria).

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

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

¶ Based on 2011 data from the World Health Organization (WHO) Vaccine Preventable Diseases Monitoring System (2012 global summary) and WHO-UNICEF coverage estimates, available at http://www.who.int/immunization_monitoring/en/globalsummary/countryprofileselect.cfm. National data might not reflect weaknesses at subnational levels.

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

†† All cVDPV isolates from DRC, Madagascar, Mozambique, Niger, Nigeria, Somalia, and Yemen were vaccine/nonvaccine recombinants.

§§ Previously reported outbreak. Additional information available at http://www.cdc.gov/mmwr/preview/mmwrhtml/mm6025a3.htm.

¶¶ Circulation was inferred because two isolates from healthy children shared a common VDPV2 ancestor but were genetically divergent.

*** The first isolate was initially categorized as an aVDPV1. Additional information available at http://www.cdc.gov/mmwr/preview/mmwrhtml/mm6025a3.htm?s_cid=mm6025a3_w. Isolation of a related isolate from a second patient confirmed VDPV1 circulation.

††† Importation from Nigeria.

§§§ Previously reported outbreak. Additional information available at http://www.cdc.gov/mmwr/preview/mmwrhtml/mm6025a3.htm.

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

**** Previously reported outbreak. Additional information available at http://www.cdc.gov/mmwr/preview/mmwrhtml/mm6025a3.htm.

†††† None of the iVDPV isolates appeared to be vaccine/nonvaccine recombinants.

§§§§ Previously reported. Additional information available at http://www.cdc.gov/mmwr/preview/mmwrhtml/mm6025a3.htm.

¶¶¶¶ Previously reported. Additional information available at http://www.cdc.gov/mmwr/preview/mmwrhtml/mm6025a3.htm.

continued cVDPV2 emergence. Two aVDPV2s unrelated to the known cVDPV2s were detected in Sokoto sewage samples in May and June 2012.

Peru. An aVDPV2 (2.2% divergent) was isolated from an AFP patient with no known immunodeficiency in April 2011.

South Sudan. An aVDPV2 (1.1% divergent) was isolated from an AFP patient in February 2012.

Sudan. An aVDPV2 (0.7% divergent) was isolated from an AFP patient in April 2012.

Yemen. Two genetically distinct aVDPV2s (1.0%–1.1% divergent) were isolated from AFP patients in separate communities in Yemen in September 2011 and February 2012, providing evidence of new cVDPV2 emergences. An aVDPV3 (2.3% divergent) was isolated from an AFP patient in April 2012.

Reported by

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Editorial Note

The World Health Organization convened a meeting in May 2012 to review the current understanding of VDPV emergence and transmission. Several key points that were reaffirmed are as follows: 1) the clinical signs and severity of paralysis associated with VDPV and WPV infections are indistinguishable; 2) cVDPVs pose the same public health threat as WPVs and require the same control measures; 3) persons with prolonged iVDPV infections can transmit poliovirus to others, raising the risk for VDPV circulation in settings of low population immunity to the corresponding poliovirus serotype; 4) existing surveillance suggests that prolonged iVDPV excretion is uncommon among persons with primary immunodeficiencies exposed to OPV; 5) the prevalence of long-term iVDPV excretors might be higher than detected by existing surveillance of persons with primary immunodeficiencies, as suggested by the detection of aVDPVs that closely resemble iVDPVs in sewage in Finland, Israel, and other countries with high poliovirus vaccine coverage; and 6) the development of treatment for prolonged iVDPV infections might facilitate detection of and access to those with infections.

Detection of genetically related VDPVs from different persons who are not close contacts, even if none of the infected persons had AFP, is evidence of VDPV circulation and should prompt the same vaccination response as detection of VDPVs in AFP patients or WPV in persons or the environment. Key risk factors for cVDPV emergence and spread are 1) development of immunity gaps arising from low poliovirus vaccine coverage; 2) prior elimination of the corresponding WPV serotype, which also eliminates immunity from natural infection; 3) emphasis on use in supplementary immunization activities (SIAs)* of monovalent OPV (mOPV) and bivalent OPV (bOPV) types 1 and 3 vaccine formulations, which can lead to immunity gaps to PV2 (*2,9*); and 4) insensitive AFP surveillance. Many of these factors exist in areas of insecurity, such as in parts of Somalia and Yemen. In this context, VDPV2s present the greatest threat for emergence (*2*), and it was emphasized at the meeting that routine immunization should be strengthened and, for the immediate future, regular SIAs using tOPV (which efficiently closes population immunity gaps when used at high coverage rates) should be conducted.

Since 1999, when the last WPV2 case was identified, all cases of poliomyelitis involving PV2 have been associated with the use of tOPV primarily in the context of low poliovirus vaccine coverage. To prevent emergence and transmission of VDPV2 as progress is made toward WPV eradication, the Strategic Advisory Group of Experts advising the World Health Organization has recommended simultaneous global cessation of tOPV use in both routine vaccination services and SIAs and switching to bOPV as soon as it is safe to do so (*10*). Prerequisites for a switch from tOPV to bOPV include 1) strong evidence of cessation of all cVDPV2 transmission based on certification-standard global poliovirus surveillance, including environmental surveillance, to detect VDPV and WPV infections as a supplement to AFP surveillance in appropriate settings; 2) maintenance of high poliovirus vaccination coverage against all three poliovirus serotypes in all countries; 3) increased use of inactivated poliovirus vaccine to maintain immunity to all three serotypes; and 4) strategic deployment of OPV stockpiles in the event of outbreaks.

* 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 nationally or in portions of a country.

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Notes from the Field

Histoplasmosis Outbreak Among Day Camp Attendees — Nebraska, June 2012

On June 21, 2012, the Douglas County Health Department (DCHD) in Omaha, Nebraska, was notified of an acute respiratory illness cluster among 32 counselors at city-sponsored day camps. Laboratory-confirmed histoplasmosis was diagnosed in one camp counselor. DCHD and the Nebraska Department of Health and Human Services (NDHHS) investigated the extent and source of the outbreak to prevent further infections.

Histoplasmosis is a common fungal infection in the United States (*1*) and is a cause of respiratory illness outbreaks in endemic areas, which include areas in the midwestern states, and particularly the Mississippi and Ohio River valleys (*2*). Illness usually is acquired from inhalation of soil contaminated with bird or bat droppings (*2*); human-to-human transmission does not occur. Symptoms include fever, headache, and respiratory symptoms, although infected persons can remain asymptomatic (*2*). Most patients will recover regardless of treatment, but severe disease can lead to respiratory failure and should be treated; immunocompromised patients are at high risk for developing histoplasmosis that spreads throughout the body (*2*).

All camp counselors and camp attendees' parents were informed of the outbreak. Counselors were requested to complete a questionnaire to report their demographic information, activities, campsite assignments, and symptoms. All camp attendees' parents were administered a separate Internet-based questionnaire regarding their child's week of attendance and symptoms. Campsite assignments were obtained from camp administrators.

Serum and urine samples from all counselors were tested by enzyme immunoassay for *Histoplasma capsulatum* antigen to detect active infection. Parents of all attendees were mailed a letter explaining the symptoms of histoplasmosis, treatment and testing indications, and that testing could be performed free of charge if they desired.

A confirmed case of histoplasmosis was defined as a serum or urine test positive for *H. capsulatum,* regardless of the person's symptoms, at any time after that person's arrival at camp. A suspected case was defined as illness comprising self-reported fever and at least one additional symptom (headache, chest pain, shortness of breath, or cough) in a camp counselor or attendee, beginning ≥3 days after camp arrival during May 21–June 27, 2012, regardless of that person's test results.

Among the 32 counselors, 19 (17 confirmed, two suspected) (59%) had illness meeting the case definition, 11 (34%) were symptomatic with fever and at least one additional symptom, and 10 (31%) sought medical care for their symptoms. No hospitalizations or deaths occurred. Median age of the counselors was 20 years (range: 18–23 years). No specific activities or campsite assignments were associated with illness when confirmed and suspected cases were combined; however, when suspected cases were excluded, digging fire pits was associated with increased risk for illness among persons with confirmed illness (risk ratio [RR] = 2.7; Fisher's exact test p-value = 0.01).

Camp activities had occurred in a wooded park with 12 campsites, nine of which were open, dirt-floor shelters with roofs supported by posts. During May 21–May 25, counselors participated in a precamp clean-up week. Activities included raking leaves, cleaning picnic tables, digging fire pits, and moving firewood; counselors did not wear personal protective equipment while cleaning. They reported observing bat guano on picnic tables and dirt floors in two of the shelters. Day camps began on June 4, 2012, each lasting from Monday through Friday. Campers were aged 6–14 years (median: 9 years); each was assigned to one campsite, where activities included cooking on wood-fired grills and eating at picnic tables. Camp activities included nature walks, outdoor games, wilderness skill training, archery, and arts and crafts; all campers participated in these activities, but none participated in high-risk activities (e.g., digging in dirt, digging fire pits, raking leaves, or cleaning campsites).

Of 797 children attending camps, questionnaires were completed on 142 (18%), and laboratory testing was performed on 21 (3%). Laboratory or questionnaire data were obtained for 153 (19%) children, of whom 17 (11.1%) had illness meeting the case definition for histoplasmosis (five confirmed, 12 suspected). A multilevel logistic regression model with a random effect for campsite was used to compare illnesses among 18 children assigned to the two campsites where guano was identified, 32 children assigned to two campsites ≤20 yards from campsites with guano, and 92 children assigned to eight campsites ≥21 yards from campsites with guano (referent group). Compared with the referent group, children assigned to campsites with guano had 2.4 times the odds of illness (95% confidence interval [CI] = 0.5–11.4), and children assigned to campsites ≤20 yards from campsites with guano had 2.2 times the odds of illness (95% CI = 0.5–8.2). A decreasing trend in illness occurred with increasing distance from campsites with guano (Cochran-Armitage test p-value = 0.04).

During a visit by DCHD and NDHHS personnel on June 26, 2012, bat guano was noted on picnic tables and dirt floors at two campsites. At that time, DCHD and NDHHS

recommended closing these campsites, and the areas were fenced off. Soil samples of all campsites, and other areas of the park were obtained for *Histoplasma* testing; results are pending.

The probable infection source in this outbreak was campsite contamination of soil and picnic tables by bat guano, which likely became aerosolized during camp activities or clean-up before camper arrival. No other potential sources of infection were identified. Subsequent to this investigation, the city parks and recreation division relocated the day camp to a different park. The health department provided recommendations to the city's parks and recreation division regarding prevention of bat roosting, procedures for inspecting and identifying potentially contaminated areas, and procedures to mitigate biohazardous sites contaminated with *Histoplasma* (*3*). Persons living in endemic areas should be aware that exposure to aerosolized soil or guano in sites with bird or bat droppings can lead to histoplasmosis, should avoid such exposures, and should seek professional assistance for cleanup efforts.

Reported by

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Announcement

National Gay Men's HIV/AIDS Awareness Day–September 27, 2012

National Gay Men's HIV/AIDS Awareness Day is observed each year on September 27 to focus on the continuing effects of the human immunodeficiency virus infection (HIV) and acquired immune deficiency syndrome (AIDS) on gay, bisexual, and other men who have sex with men (MSM) in the United States. By the end of 2009, more than 592,000 MSM were living with HIV infection, 52% of persons living with HIV infection in the United States (*1*).

Although MSM represent approximately 2% of the U.S. population (*2*), in 2009, they accounted for 64% of all new HIV infections (including MSM who also were injection drug users [3% of new infections]). During 2006–2009, the estimated number of new HIV infections among MSM was stable overall, but increased approximately 34% among MSM aged 13–29 years, and approximately 48% in black or African American MSM in that age group. In 2009, approximately 22% of new infections among MSM were among young black or African American MSM, the highest number of new infections among any age or race/ethnicity group of MSM (*3*).

CDC supports a range of efforts to reduce HIV infection among MSM. These include HIV prevention services to reduce the risk for acquiring and transmitting HIV, diagnosis of HIV infection, and linkage of MSM with HIV infection to treatment, including programs designed specifically for young black or African American MSM. Additional information about these efforts is available at http://www.cdc.gov/hiv/topics/msm. Additional information about National Gay Men's HIV/AIDS Awareness Day is available at http://www.cdc.gov/features/ngmhaad.

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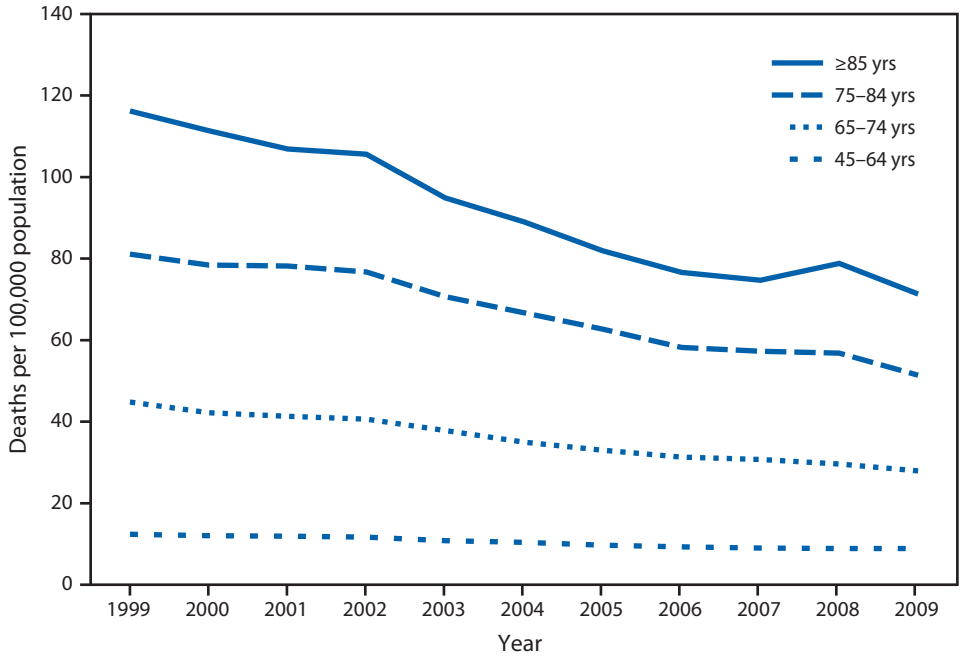
QuickStats

FROM THE NATIONAL CENTER FOR HEALTH STATISTICS

Death Rate* From Complications of Medical and Surgical Care Among Adults Aged ≥45 Years, by Age Group — United States, 1999–2009



* Deaths from complications of medical and surgical care are those coded Y40–Y84 or Y88 in the *International Classification of Diseases, 10th Revision*. Death rates are those with any mention of complications of medical and surgical care on the death certificate.

During 1999–2009, rates of death from complications of medical and surgical care declined among all age groups for persons aged ≥45 years. Deaths per 100,000 population declined 39%, to 71.3 deaths for adults aged ≥85 years; 37%, to 51.4 deaths for those aged 75–84 years; 38%, to 27.9 deaths for adults aged 65–74 years; and 28%, to 8.9 deaths for adults aged 45–64 years rates. The rate of decline among adults aged 45–64 years was lower compared with the rates of decline for all older age groups.

Source: CDC. National Vital Statistics System. Available at http://www.cdc.gov/nchs/nvss/mortality_public_use_data.htm.

CDC. Health Data Interactive. Available at http://www.cdc.gov/nchs/hdi.htm.

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

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