Laboratory Diagnosis of Ebola Hemorrhagic Fever during an Outbreak in Yambio, Sudan, 2004

Clayton O. Onyango,1 Martin L. Opoka,2 Thomas G. Ksiazek,3 Pierre Formenty,4 Abdullahi Ahmed,2 Peter M. Tukei,3 Rosemary C. Sang,1 Victor O. Ofula,1 Samson L. Konongoi,1 Rodney L. Coldren,1 Thomas Grein,4 Dominique Legros,4 Mike Bell,5 Kevin M. De Cock,1 William J. Bellini,7 Jonathan S. Towner,7 and Pierre E. Rollin1

1World Health Organization (WHO) Collaborating Center for Arbovirus and Viral Hemorrhagic Fever Reference and Research, 2WHO South Sudan, Warwick Center, 3Kenya Medical Research Institute, and 4Centers for Disease Control and Prevention–Kenya, Nairobi, Kenya; 5US Army Medical Component, Armed Forces Research Institute of Medical Sciences, Thailand; 6Epidemic and Pandemic Alert and Response, WHO, Geneva, Switzerland; and 7Centers for Disease Control and Prevention, Atlanta, Georgia

Between the months of April and June 2004, an Ebola hemorrhagic fever (EHF) outbreak was reported in Yambio county, southern Sudan. Blood samples were collected from a total of 36 patients with suspected EHF and were tested by enzyme-linked immunosorbent assay (ELISA) for immunoglobulin G and M antibodies, antigen ELISA, and reverse-transcription polymerase chain reaction (PCR) of a segment of the Ebola virus (EBOV) polymerase gene. A total of 13 patients were confirmed to be infected with EBOV. In addition, 4 fatal cases were classified as probable cases, because no samples were collected. Another 12 patients were confirmed to have acute measles infection during the same period that EBOV was circulating. Genetic analysis of PCR-positive samples indicated that the virus was similar to but distinct from Sudan EBOV Maleo 1979. In response, case management, social mobilization, and follow-up of contacts were set up as means of surveillance. The outbreak was declared to be over on 7 August 2004.

Ebola virus (EBOV; genus *Ebolavirus*, family *Filoviridae*) is the etiologic agent of Ebola hemorrhagic fever (EHF). This single-stranded, negative-sense RNA virus that can produce high-mortality disease in humans and nonhuman primates has caused sporadic outbreaks in Central Africa and Southeast Asia [1].

The genome organization that encodes 8 viral proteins is arranged in the order 3′–nucleoprotein (NP)–virion structural protein (VP) 35–VP40–glycoprotein (G)–VP30–VP24–polymerase (L)–5′. Currently, there are 4 known species of EBOV, 3 of which are found in Africa (Zaire, Sudan, and Cote d’Ivoire) and a fourth species, Reston, found in Philippines [1]. Historically, outbreaks of filovirus infection have been sporadic and isolated. These viruses persist in nature in a reservoir that is still unidentified despite intensive epidemiological and ecological research. Some unexplained changes in the ecological conditions or demographic factors enable them to resurface, either among nonhuman primates, which then act as intermediate hosts and are the origin of contamination of the human population, or directly in a human index patient. The lack of infection control measures in health centers or hospitals is the root cause of large nosocomial outbreaks.

EBOV was first identified in 1976 [2–4] during 2 simultaneous outbreaks of hemorrhagic fever in Sudan [5] and Zaire [6]. The clinical presentation of EHF includes fever, headache, and malaise at onset, with profuse vomiting and diarrhea developing 2–4 days later [7–10]. Hemorrhagic manifestations are prominent features in patients who die but are observed less commonly in survivors [11, 12]. Several serological techniques have been developed to diagnose EBOV in-
fection. ELISAs for antigen capture and IgG and IgM antibodies are commonly used as diagnostic tools; these show a good measure of sensitivity and specificity and are able to detect all of the EBOV species [13–15]. Antigen-capture diagnostic assays together with nested reverse-transcription (RT)–polymerase chain reaction (PCR) have been developed and used in previous outbreaks [16–18]. These methods proved to be very effective as field diagnostic tools for the detection of EBOV antigen and nucleic acid in patient serum, plasma, and whole blood.

On 6 May 2004, the medical staff at Yambio County Health Department and the coordinator of the Yambio World Health Organization (WHO) South Sudan Early Warning and Response Network (EWARN; established in southern Sudan in 1999 to monitor disease outbreaks) reported a cluster of 7 suspected cases of hemorrhagic fever, including 2 deaths, to the EWARN team leader in Lokichoggio, Kenya. The patients, 5 from the same family and 2 hospital staff, became ill over a period of 3 weeks. Their symptoms included high fever, bloody diarrhea, and vomiting. The clinical presentation, the family cluster, and the involvement of health care workers led to a suspicion of viral hemorrhagic fever.

Yambio is located in the remote savanna of southern Sudan, near the border with the Central African Republic and the Democratic Republic of the Congo. Approximately 100,000 people live within a 15-km radius of the town of Yambio. The Yambio civil hospital is a small, overcrowded hospital staffed by 2 physicians, some clinical officers, and several nurses. The hospital consists of 4 concrete structures for inpatients and a single grass thatched hut reserved as an isolation room. There is no running water in the patient care areas, and the hospital has a limited supply of masks, caps, and gowns; thus, the staff are unable to routinely practice barrier-nursing techniques.

On 9 May, a joint mission conducted by EWARN and the Kenya Medical Research Institute (KEMRI) traveled to the town of Yambio to make an initial field investigation. The local health authorities, with the assistance of an international team, organized the response activities: creation of an isolation ward for patients with suspected EHF cases, case management and collection of appropriate diagnostic specimens, follow-up of subjects in contact with patients with suspected or confirmed cases, epidemiological surveillance, and social mobilization in the community. A patient with a suspected case of EHF was described as a person who, since April 2004, had a history of severe headache, fever, muscle pains, joint pains, and diarrhea and who had been in close contact with 2 deceased members of the household or with a patient with a confirmed case or a deceased person with a suspected case. The response activities were maintained until 7 August 2004, when the outbreak was declared to be over, 42 days (2 incubation periods) after the death of the last patient with a confirmed case. The management of the outbreak was complicated by the occurrence of severe cases of measles toward the end of the EHF outbreak. We report here the laboratory investigations performed and the identified causative agent.

MATERIALS AND METHODS

Collection of human serum. As of 9 May 2004, we investigated any patient suspected to have EHF living within Yambio payam (district). A total of 17 serum samples were collected: 15 from patients with possible disease presenting with headache, fever, and/or diarrhea and 2 from otherwise healthy presenting contacts. This study is based on an intervention after a suspected disease outbreak; therefore, consent was not required, as stipulated by WHO guidelines. Blood was collected in evacuated clot activator Vacutainer tubes (Becton Dickinson). Serum samples were carefully separated in the Yambio hospital laboratory by technicians using appropriate personal protective equipment. Serum was aliquoted in 3 separate cryotubes, and 6 volumes of viral lysis buffer (containing guanidine isothiocyanide; Qiagen) added in 1 of the 2 cryotubes with serum. The serum samples were then stored in a liquid nitrogen dry shipper for transportation to KEMRI in Nairobi for analysis. An aliquot of each specimen was later shipped to the Centers for Disease Control and Prevention (CDC) (Atlanta, GA). Later during the outbreak investigation, 19 samples were collected from patients suspected to have EHF.

RT-PCR. At KEMRI, all 36 chaotrope-treated serum samples were subjected to RT-PCR using a universal pair of primers for members of the family Filoviridae. Viral RNA was extracted directly from 200-μL samples, using a QIAGEN viral RNA isolation kit (Qiagen). RT-PCR specific for a 419-bp region of the L gene of the filoviruses was performed as described elsewhere [19]. The 419-bp positive amplicons were purified using the Qiagen gel purification kit and subsequently used for genetic sequence analysis. A confirmatory RT-PCR was performed on the filovirus-positive samples to amplify a 428-bp region of the EBOV NP gene with 50 pmol of the oligonucleotide primers EBO-SV (GATGAGGACAAACTTTAA) and EBO-SC (GCC-TCACGGAGTTGCTGATATG). These primers were designed using Primer3 software [20]. All of the positive samples were exposed to a second RNA extraction and RT-PCR to rule out the possibility of contamination. RT-PCR analysis at the CDC was performed as follows: RNA was isolated from 50 μL of sample, using either TriPure as described elsewhere [19] or an Applied Biosystems 6100 nucleic acid prep station as described elsewhere [21]. The nested RT-PCR assay [19] utilized primers capable of amplifying either Sudan or Zaire EBOV NP sequences. PCR products from positive samples were gel purified and exposed to sequencing, using the same primers used for nested PCR.

Nucleotide sequencing. Gel-purified PCR products generated using Filoviridae universal primers were quantified, and
nucleotide sequences of amplicons with a minimum of 60 ng of DNA per microliter were determined using the Big Dye Terminator sequencing ready reaction kits with AmpliTaq DNA polymerase FS (Applied Biosystems). A cycle sequencing reaction was performed with each of the primers Filo A and Filo B in a final volume of 20 μL with 30 ng of PCR product, 3.5 pmol of primer, and 4 μL of BigDye Terminators premix, in accordance with the manufacturer’s protocol. Briefly, the tubes were heated to 96°C for 2 min, and the reaction mixture underwent 25 cycles of 30 s at 96°C, 30 s at 50°C, and 4 min at 60°C. Excess BigDye Terminators were removed by precipitation with absolute ethanol. The partial nucleotide sequence was obtained with an Applied Biosystems 3100 sequencer, in accordance with the manufacturer’s instructions. A search was performed using Basic Local Alignment Search Tool (BLAST; National Center for Biotechnology Information [http://www.ncbi.nlm.nih.gov/blast/]).

**Phylogenetic analysis of Yambio 2004 samples.** The L gene partial nucleotide sequences obtained from Yambio 2004 samples were aligned with other EBOV sequences from GenBank (table 1) using BioEdit’s ClustalW multiple nucleotide sequence analysis software [22]. The phylogenetic analysis was performed using MEGA3 software [23] to determine the range of geographic relationship among the Yambio viruses and the other isolates.

**Virus isolation, antigen detection, and IgM and IgG ELISAs.** In the CDC Special Pathogens branch, the specimens were initially processed under biosafety level 4 (BSL4) conditions. An aliquot of each sample was inactivated by γ-irradiation and used for further antigen and antibody testing for hemorrhagic fever viruses and measles testing. Virus isolation was attempted on all specimens in the BSL4 laboratory by use of Vero E6 cells. The flasks were observed daily for cytopathic effect (CPE). At days 7 and 14 or if CPE was obvious, the cultures were harvested and tested by use of EBOV- and Marburg virus–specific immunofluorescence assays.

The EBOV antigen-detection ELISAs were performed as described elsewhere [13, 16]. Specimens were tested at 4 dilutions (1:4, 1:16, 1:64, and 1:256). Titers and the cumulative sum (4 dilutions) of the optical density (OD sum) were recorded. The EBOV IgM and IgG ELISAs were also performed as described elsewhere [14, 16]. Specimens were tested at 4 dilutions (1:100, 1:400, 1:1600, and 1:6400). Titers and the cumulative sum (4 dilutions) of the OD sum were recorded.

**Differential diagnosis.** In addition to the EBOV-specific tests described above, antigen detection and IgM and IgG ELISAs were performed for Marburg and Crimean-Congo hemorrhagic fever viruses on the first set of 17 samples and then discontinued when EBOV was recognized as the causative agent of the outbreak. RT-PCR analysis was performed to exclude the following pathogens: Crimean-Congo hemorrhagic fever virus, Marburg virus, Rift Valley fever virus, Rickettsia, Leptospira, Brucella, members of the genus Alphavirus, and Bunyavirus, as described elsewhere [24]. All serum samples were assayed for the presence of measles virus IgG and IgM antibodies by use of the CDC ELISA tests [25].

**RESULTS**

On 16 May 2004, KEMRI identified filovirus infection in 3 of 12 samples tested by RT-PCR and in 2 of 12 samples tested by antigen-detection ELISA. On 19 May 2004, sequence data for the partial filovirus L gene from 3 patients identified Sudan EBOV as the causative agent. The results obtained are sum-

---

**Table 1. Virus isolates used in the phylogenetic comparison of Yambio 2004 isolates, based on a portion of polymerase (L) gene and their accession nos. in GenBank**

<table>
<thead>
<tr>
<th>Country</th>
<th>Year</th>
<th>Designation</th>
<th>Strain</th>
<th>Source of isolate</th>
<th>GenBank accession no.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sudan</td>
<td>2004</td>
<td>Yambio 0401</td>
<td>Maleo</td>
<td>Human</td>
<td>EF183506</td>
</tr>
<tr>
<td>Sudan</td>
<td>2004</td>
<td>Yambio 0402</td>
<td>Maleo</td>
<td>Human</td>
<td>EF183507</td>
</tr>
<tr>
<td>Sudan</td>
<td>2004</td>
<td>Yambio 0403</td>
<td>Maleo</td>
<td>Human</td>
<td>EF183508</td>
</tr>
<tr>
<td>Sudan</td>
<td>1979</td>
<td>Maleo 1979</td>
<td>Maleo</td>
<td>Human</td>
<td>U23458</td>
</tr>
<tr>
<td>Uganda</td>
<td>2000</td>
<td>Gulu 2000</td>
<td>Gulu</td>
<td>Human</td>
<td>AY729654</td>
</tr>
<tr>
<td>Zaire</td>
<td>1995</td>
<td>Zaire 1995</td>
<td>Zaire</td>
<td>Human</td>
<td>AY354458</td>
</tr>
<tr>
<td>Zaire</td>
<td>1976</td>
<td>Mayinga</td>
<td>Mayinga</td>
<td>Human</td>
<td>X67110</td>
</tr>
<tr>
<td>Gabon</td>
<td>1996</td>
<td>Chiro MB03J</td>
<td>Zaire</td>
<td>Bat</td>
<td>DQ206415</td>
</tr>
<tr>
<td>Gabon</td>
<td>1996</td>
<td>Chiro EK02</td>
<td>Zaire</td>
<td>Bat</td>
<td>DQ206410</td>
</tr>
<tr>
<td>Gabon</td>
<td>1996</td>
<td>MendembaB</td>
<td>Zaire</td>
<td>Human</td>
<td>DQ206417</td>
</tr>
<tr>
<td>Democratic Republic of the Congo</td>
<td>1999</td>
<td>Marburg DRC99</td>
<td>O5DRC99</td>
<td>Human</td>
<td>DQ447851</td>
</tr>
<tr>
<td>Philippines</td>
<td>1996</td>
<td>Reston Phil.</td>
<td>Reston</td>
<td>...</td>
<td>AB050936</td>
</tr>
<tr>
<td>Gabon</td>
<td>1996</td>
<td>Mayibout</td>
<td>Zaire</td>
<td>Human</td>
<td>DQ205418</td>
</tr>
</tbody>
</table>
marized in table 2. On 21 May, the CDC confirmed the presence of Sudan EBOV or antibody in 10 of 17 samples tested by IgM, IgG, and antigen capture ELISAs as well as PCR and gene sequencing (table 2).

**RT-PCR and nucleotide sequencing.** In total, samples from 6 patients were positive for filovirus, as demonstrated by the amplification of a fragment of the L gene. These were subsequently found to be positive for Sudan EBOV by serotyping targeting the nucleoprotein gene, using primers specific for Sudan EBOV. Analysis of the nucleotide sequences obtained with filovirus (L gene) primers confirmed that the amplicons were partial sequences from the EBOV L gene. The nested NP assay also identified 6 patients as positive for Sudan EBOV.

**EBOV antigen, IgM and IgG antibodies, and virus isolation.** EBOV infection was confirmed in a total of 13 patients by use of classical serological and virological techniques: EBOV antigen was detected in serum samples from only 2 patients; 12 patients had IgM antibodies against EBOV, and 8 patients had EBOV IgG antibodies at the time of collection; all virus isolation attempts were negative at the beginning of the outbreak, but EBOV was isolated from patients 33, 34, and 35 (strains SPB200407830, SPB200407831, and SPB200407832, respectively) at the end of the outbreak (table 2). All of these patients were recovering, which was demonstrated by the presence of IgG and the lack of detectable antigen (the threshold of detection of the test is ~100 pfu of virus [Special Pathogens Branch, CDC, unpublished data]).

**Phylogenetic analysis of EBOV based on the L gene.** Derived nucleotide sequences from Yambio samples aligned with other EBOV strains were used to construct a phylogenetic tree. By use of the maximum parsimony method, we determined 3 monophyletic groups based on portions of the EBOV L gene, with a Marburg virus partial L gene sequence as the root (figure 1). Similar results were obtained on the basis of analysis of the NP gene fragment sequences (data not shown).

**Differential diagnosis.** The serum samples were all found to be negative by RT-PCR for Marburg virus, Crimean-Congo hemorrhagic fever virus, West Nile virus, dengue virus, chi-

---

### Table 2.

**History and results obtained from patients during the Ebola hemorrhagic fever (EHF) outbreak, Yambio, Sudan, 2004.**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sex</th>
<th>Age, years</th>
<th>EBOV IgM</th>
<th>EBOV IgG</th>
<th>EBOV antigen</th>
<th>EBOV PCR and sequencing</th>
<th>EBOV isolation (cell culture)</th>
<th>Measles virus IgM</th>
<th>Measles virus IgG</th>
</tr>
</thead>
<tbody>
<tr>
<td>1*</td>
<td>M</td>
<td>33</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>−</td>
<td>−</td>
<td>−</td>
</tr>
<tr>
<td>2*</td>
<td>F</td>
<td>33</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>+</td>
<td>−</td>
<td>−</td>
<td>−</td>
</tr>
<tr>
<td>3</td>
<td>M</td>
<td>42</td>
<td>+</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>+</td>
</tr>
<tr>
<td>4</td>
<td>F</td>
<td>40</td>
<td>+</td>
<td>+</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>+</td>
</tr>
<tr>
<td>5*</td>
<td>F</td>
<td>34</td>
<td>+</td>
<td>−</td>
<td>+</td>
<td>+</td>
<td>−</td>
<td>−</td>
<td>+</td>
</tr>
<tr>
<td>6</td>
<td>M</td>
<td>28</td>
<td>+</td>
<td>+</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>+</td>
</tr>
<tr>
<td>7</td>
<td>M</td>
<td>29</td>
<td>+</td>
<td>+</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>+</td>
</tr>
<tr>
<td>10</td>
<td>M</td>
<td>25</td>
<td>+</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>13</td>
<td>F</td>
<td>60</td>
<td>+</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>+</td>
</tr>
<tr>
<td>14</td>
<td>M</td>
<td>35</td>
<td>+</td>
<td>+</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>+</td>
</tr>
<tr>
<td>33</td>
<td>F</td>
<td>23</td>
<td>+</td>
<td>+</td>
<td>−</td>
<td>+</td>
<td>+</td>
<td>I</td>
<td>+</td>
</tr>
<tr>
<td>34*</td>
<td>F</td>
<td>60</td>
<td>+</td>
<td>+</td>
<td>−</td>
<td>+</td>
<td>+</td>
<td>I</td>
<td>+</td>
</tr>
<tr>
<td>35*</td>
<td>F</td>
<td>26</td>
<td>+</td>
<td>+</td>
<td>−</td>
<td>+</td>
<td>+</td>
<td>I</td>
<td>+</td>
</tr>
</tbody>
</table>

**NOTE.** +, positive; −, negative; EBOV, Ebola virus; I, indeterminate; PCR, polymerase chain reaction.

*a* Nucleotide sequence used to determine the phylogenetic relationship.

*b* Patient had a fatal EHF case.
kungunya virus, Sindbis virus, Rift Valley fever virus, Rickettsia, Leptospira, Brucella, and members of the genus Bunyavirus. IgM ELISA results remained negative for Marburg virus, yellow fever virus, West Nile virus, dengue virus, chikungunya virus, and Sindbis virus. Antibodies (IgM, IgG, or both) against measles virus were detected in 33 of 36 patients tested during the EHF outbreak. The IgM ELISA test for measles virus demonstrated 12 positive patient specimens, whereas 25 patients were found to be positive for measles by IgG ELISA. Six patient samples remained indeterminate by IgM ELISA but were IgG positive, and 8 patient specimens remained indeterminate by IgG ELISA against measles virus. All the EBOV-positive patients tested positive for measles IgG, indicating past infection.

**DISCUSSION**

Samples from 36 patients with possible EBOV infection were collected within Yambio payam. A total of 13 patients had EBOV activity demonstrated by either IgM and/or IgG antibody ELISA, antigen capture ELISA, RT-PCR, or subsequent virus isolation. Another 4 patients, whose blood samples could not be obtained, died and were classified as having probable EBOV infection, according to the case definition developed by the epidemiology team [26]. Of the 36 patients, 8 had both IgG and IgM antibodies against EBOV; another 4 patients had IgM antibodies only (table 2). All 12 of these patients recovered from the disease.

A patient who had a blood sample drawn ~2 days after the onset of illness was confirmed to be positive by RT-PCR. At this early stage, the patient had not mounted an antibody response against the virus. The patient died 10 days after the onset of illness, before more blood samples could be collected.

At the beginning of the outbreak, all virus isolation attempts were negative, but EBOV was isolated from 3 patients toward the end of the outbreak (table 2). A likely explanation for the failure to isolate the virus could be the suboptimal shipment conditions of the first 17 samples received at CDC, as evidenced by the complete failure of virus isolation from any of the antigen-positive specimens.

The Yambio 0401 and Yambio 0403 strains had 100% nucleotide sequence identity in the L gene, and Yambio 0402 had 99% nucleotide sequence identity compared with the other 2 isolates (data not shown). Yambio 0402 had a minor amino acid residue change, S629T. Despite the residue change in Yambio 0402, all 3 Yambio isolates formed a well-supported monophyletic group with Sudan EBOV Maleo, which was quite distinct from the Zaire EBOV lineages (figure 1). Both the nucleotide and the amino acid sequence analysis of Yambio viruses suggests that a single-genetic-lineage virus was associated with the 2004 outbreak. However, we recommend full genome sequencing of these isolates for additional genetic information.

In Yambio, the occurrence of the concomitant measles outbreak complicated the task of selecting and classifying cases for isolation and management. It was evident that both the measles virus and EBOV spread within families and within groups of contacts, making it difficult to clinically differentiate severe measles from EHF, because both diseases clustered and caused similar clinical symptoms during the early stages of infection. A total of 12 patients suspected to have EHF on the basis of clinical and epidemiological findings (but whose laboratory test results were negative for EBOV) tested positive for IgM antibodies against measles virus.

The laboratory confirmation of the clinical suspicion of hemorrhagic fever helped to reduce the size, duration, and management of the outbreak (i.e., adequate isolation policy and restriction of the follow-up surveillance to only the contacts of the confirmed cases). However, the absence of laboratory diagnostic facilities in the field to quickly confirm the clinical diagnosis hampered control activities, leading to the misclassification and isolation of patients with measles in the Ebola reserved ward. In the future, the deployment of field-based laboratory diagnostic facilities should be considered to be an integral part of a filovirus infection control team.

After previous EHF outbreaks in Sudan and elsewhere in Africa, the Yambio 2004 outbreak is one of the smallest outbreaks ever recorded. The early detection of this outbreak by EWARN, on the basis of surveillance of hemorrhagic fever case definitions and subsequent laboratory confirmation, coupled with a rapid response may have contributed to the small number of cases. The swift response by the WHO, CDC, Médecins Sans Frontières, KEMRI, and other partners further enhanced an efficient surveillance system and put infection control measures in place during the outbreak, potentially saving many lives. The WHO officially declared the end of the EHF epidemic on 7 August 2004, 42 days after the death of the patient with the last registered case and ~5 months after the index patient was infected.

**Acknowledgments**

We thank colleagues at the Kenya Medical Research Institute (KEMRI) and the Centers for Disease Control and Prevention (especially Tara Sealy), for their technical support during the outbreak; the international team responsible for controlling the outbreak that collaborated with the Sudan People’s Liberation Movement Health Secretariat and the Early Warning and Response Network team, which included partners from the Global Outbreak Alert and Response Network and brought together Médecins Sans Frontières–France, the United Nations Children’s Fund, and teams from the following countries: Austria (Institute for the History of Medicine, Department of Ethnomedicine, University of Vienna, Vienna), Kenya (KEMRI, Nairobi), the Netherlands (Catholic Organisation for Relief and Development, The Hague), Tunisia (World Health Organization Mediterranean Centre for Vulnerability Reduction, Tunis), the United Kingdom (Health Protection Agency and the European Programme for Intervention Epidemiology Training, London, England), and the United States (Centers for Disease Control and Prevention, Atlanta, GA).
Supplement sponsorship. This article was published as part of a supplement entitled “Filoviruses: Recent Advances and Future Challenges,” sponsored by the Public Health Agency of Canada, the National Institutes of Health, the Canadian Institutes of Health Research, Gangene, CUH2A, Smith Carter, Hemisphere Engineering, Crucell, and the International Centre for Infectious Diseases.

References