

*ompA*: JF803906). All samples were cultured in human embryonic lung fibroblasts as described (6). After 4 weeks, no bacteria were isolated.

We report a human case of *R. aeschlimannii* infection in Crete, Greece. Our finding was confirmed by molecular methods. However, we were not able to cultivate *R. aeschlimannii* from samples collected. This result suggests that living microorganisms may have died before testing or that only DNA, but no living organism, was present in the samples. *R. aeschlimannii* was first isolated from *Hyalomma marginatum* ticks from Morocco (7). In Europe, *R. aeschlimannii* has also been found in ticks from Germany, Russia, Italy, France, Croatia, Portugal, and Spain (8). In Greece, *R. aeschlimannii* has been detected in *H. anatolicum excavatum* ticks collected from sheep (1). The tick removed from this patient was *Rh. turanicus*, a species that has been reported in Spain to be infected with *R. aeschlimannii* (9).

The first human case of *R. aeschlimannii* infection was identified in a patient who had fever, rash, and an eschar after travel in Morocco (10). *R. aeschlimannii* infections in humans have been previously confirmed in South Africa, in Algeria, and in Tunisia (8). To our knowledge, human cases of *R. aeschlimannii* infection have not been reported in Europe. Our results emphasize that ticks should be considered as potential vectors for rickettsial infections in humans. We recommend that when one species or serotype of tick-transmitted *Rickettsia* is identified in an area, physicians be informed through established clinical or public health channels of the potential pathogen, its manifestations, and recommended treatments for humans.

**Antonios Germanakis,  
Dimosthenis Chochlakis,  
Emmanouil Angelakis,  
Yannis Tselentis,  
and Anna Psaroulaki**

Author affiliations: General Hospital of Agios Nikolaos, Crete, Greece (A. Germanakis); Regional Laboratory of Public Health, Heraklion-Crete, Greece (D. Chochlakis, Y. Tselentis); University of Crete Medical School, Heraklion, Greece (Emmanouil Angelakis, Anna Psaroulaki); and Aix-Marseille University School of Medicine, Marseille, France (E. Angelakis)

DOI: <http://dx.doi.org/10.3201/eid1907.130232>

## References

1. Psaroulaki A, Ragiadaku D, Kouris G, Papadopoulos B, Chaniotis B, Tselentis Y. Ticks, tick-borne rickettsiae, and *Coxiella burnetii* in the Greek Island of Cephalonia. *Ann N Y Acad Sci*. 2006;1078:389–99. <http://dx.doi.org/10.1196/annals.1374.077>
2. Psaroulaki A, Germanakis A, Gikas A, Scoulica E, Tselentis Y. First isolation and genotypic identification of *Rickettsia conorii* Malish 7 from a patient in Greece. *Eur J Clin Microbiol Infect Dis*. 2005;24:297–8. <http://dx.doi.org/10.1007/s10096-005-1304-z>
3. Psaroulaki A, Germanakis A, Gikas A, Scoulica E, Tselentis Y. Simultaneous detection of “*Rickettsia mongolotimonae*” in a patient and in a tick in Greece. *J Clin Microbiol*. 2005;43:3558–9. <http://dx.doi.org/10.1128/JCM.43.7.3558-3559.2005>
4. Ioannou I, Sandalakis V, Kassinis N, Chochlakis D, Papadopoulos B, Loukaides F, et al. Tick-borne bacteria in mouflons and their ectoparasites in Cyprus. *J Wildl Dis*. 2011;47:300–6.
5. Chochlakis D, Ioannou I, Sandalakis V, Dimitriou T, Kassinis N, Papadopoulos B, et al. Spotted fever group *Rickettsiae* in ticks in Cyprus. *Microb Ecol*. 2012;63:314–23. <http://dx.doi.org/10.1007/s00248-011-9926-4>
6. Angelakis E, Richet H, Rolain JM, La Scola B, Raoult D. Comparison of real-time quantitative PCR and culture for the diagnosis of emerging Rickettsioses. *PLoS Negl Trop Dis*. 2012[cited 2012 Jul 12];6:e1540. <http://www.plosntds.org/article/info%3Adoi%2F10.1371%2Fjournal.pntd.0001540>
7. Beati L, Meskini M, Thiers B, Raoult D. *Rickettsia aeschlimannii* sp. nov., a new spotted fever group rickettsia associated with *Hyalomma marginatum* ticks. *Int J Syst Bacteriol*. 1997;47:548–54. <http://dx.doi.org/10.1099/00207713-47-2-548>
8. Demoncheaux JP, Socolovschi C, Davoust B, Haddad S, Raoult D, Parola P. First detection of *Rickettsia aeschlimannii* in *Hyalomma dromedarii* ticks from Tunisia. *Ticks Tick Borne Dis*. 2012;3:398–402. <http://dx.doi.org/10.1016/j.ttbdis.2012.10.003>
9. Fernández-Soto P, Encinas-Grandes A, Perez-Sanchez R. *Rickettsia aeschlimannii* in Spain: molecular evidence in *Hyalomma marginatum* and five other tick species that feed on humans. *Emerg Infect Dis*. 2003;9:889–90.
10. Raoult D, Fournier PE, Abboud P, Caron F. First documented human *Rickettsia aeschlimannii* infection. *Emerg Infect Dis*. 2002;8:748–9. <http://dx.doi.org/10.3201/eid0807.010480>

Address for correspondence: Anna Psaroulaki, Laboratory of Clinical Bacteriology, Parasitology, Zoonoses and Geographical Medicine, Faculty of Medicine, University of Crete, PO Box 1393, Heraklion, Crete 71110, Greece; email: [annapsa@med.uoc.gr](mailto:annapsa@med.uoc.gr)

## Antibodies against Rift Valley Fever Virus in Cattle, Mozambique

**To the Editor:** During the past 2 decades, several countries in Africa and the Arabian Peninsula, to which Rift Valley fever virus (RVFV) is endemic, have reported outbreaks of Rift Valley fever in humans and livestock. The first evidence of RVFV in Mozambique was documented as early as the 1960s (1). Endemicity was subsequently confirmed in the 1980s by a prevalence study that identified virus-specific antibodies in 2% of pregnant women (2) and in the 1990s by serosurveillance in Zambezia Province, which showed that cattle had been infected with RVFV (3).

Apart from those observations, the RVFV situation in Mozambique is still poorly understood. We recently found an unexpectedly high level of RVFV activity in cattle in Namaacha District in Maputo Province (4), a region where there had been no recorded evidence of the virus since 1969 (1). We conducted a cross-sectional study in which serum samples were collected throughout Maputo Province

during 2010–2011 to ascertain whether any RVFV circulation had remained undetected among bovids.

The study was approved by the Mozambican Board of Agriculture. Animals investigated were of mixed breed, had been present in their respective localities since birth, were >6 months of age, and had not been vaccinated against RVFV. Samples were analyzed by using a plaque-reduction neutralization test (4), and RVFV seropositivity was defined as 80% reduction of virus infectivity at a serum dilution of 1:40.

A total of 404 serum samples were analyzed, and 149 were positive for RVFV-neutralizing antibodies. This finding represents an overall seroprevalence of 36.9% (95% CI 32.2%–41.6%) in Maputo Province, which is a high level for an area in which no RVFV disease activity has been reported during the past 4 decades.

Although the study was designed to determine the overall prevalence in the province, our data also provided an indication of the distribution of RVFV at a district level. Maputo Province is subdivided into 7 districts, and samples from 6 of these districts were available for analysis: Boane (n = 28), Magude (n = 34), Manhiça (n = 65), Marracuene (n = 82), Matutuine (n = 131), and Moamba (n = 64). The livestock populations in Magude, Manhiça, Matutuine, and Moamba Districts range in size from 20,000 to 70,000 animals, and Boane and Marracuene Districts have smaller populations of 6,000 and 9,000, respectively.

We found the highest seroprevalence to be 61.5% (95% CI 49.7%–73.4%) in Manhiça District and 62.2% (95% CI 51.7%–72.7%) in Marracuene District. Some of the animals affected by RVFV during outbreaks in 1969 were raised on farms near or in these 2 regions (1). Our data indicate that the RVFV activity is still high in those districts, possibly because breeding of mosquito vectors is promoted by environmental factors,

including irrigation ditches on sugar cane farms, extensive flood plains, wetlands, and ponds (5).

South Africa keeps continuous records of RVFV outbreaks, and several outbreaks in cattle were reported in KwaZulu-Natal and Mpumalanga Provinces during 2008–2010 (6). Matutuine, the southernmost district in Maputo Province, shares a border with KwaZulu-Natal Province, whereas Magude and Moamba Districts border Mpumalanga Province. Accordingly, the high seropositivity rates of 19.8% (95% CI 13.0%–26.7%), 26.5% (95% CI 11.6%–41.3%), and 29.7% (95% CI 18.5%–40.9%) in Matutuine, Magude, and Moamba Districts, respectively, are not remarkable. In comparison, a seroprevalence of 14.3% (95% CI 1.3%–27.2%) was noted in Boane District, which has a smaller livestock population.

Because of long-term persistence of IgG against RVFV, the actual time of infection could not be determined from the data obtained in our study. However, information was available regarding the age of the cattle in Moamba District. From this information, we deduced that the most recent RVFV infections in this district occurred at some point during 2009–2010.

Our results strongly suggest that RVFV is widely distributed among bovids in Maputo Province, although the modality of this circulation is unknown. RVFV infection can remain undetected in adult livestock but can cause abortions in pregnant animals and neonatal death in small ruminants (7), which has major economic consequences. Transmission to humans is common during epizootics, and the proximity to and density of cattle in an area have been shown to be major factors for RVFV seroconversion in human populations (8). Human infections are often manifested as a febrile illness and can easily be mistaken for other diseases. Consequently, unidentified or underdiagnosed RVFV infections among livestock in Maputo

Province warrant further research, and implementation of surveillance and livestock vaccination programs in the studied area should be discussed.

#### Acknowledgment

We thank Z. Massicame and A. Malumana for providing bovine serum samples.

This study was supported by the Swedish International Development Cooperation Agency.

**Nina Lagerqvist,  
Belisário Moiane,  
Lourenço Mapaco,  
José Fafetine, Sirkka Vene,  
and Kerstin I. Falk**

Author affiliations: Karolinska Institutet, Stockholm, Sweden (N. Lagerqvist, B. Moiane, K.I. Falk); Swedish Institute for Communicable Disease Control, Solna, Sweden (N. Lagerqvist, S. Vene, K.I. Falk); Universidade Eduardo Mondlane, Maputo, Mozambique (B. Moiane, J. Fafetine); and Direção de Ciências Animais, Maputo (L. Mapaco)

DOI: <http://dx.doi.org/eid1907.130332>

#### References

1. Valadão FG. Notes about the occurrence of a new disease in Mozambique: Rift Valley fever [in Portuguese]. *Veterinaria Mocambicana*. 1969;2:13–20.
2. Niklasson B, Liljestrand J, Bergström S, Peters CJ. Rift Valley fever: a sero-epidemiological survey among pregnant women in Mozambique. *Epidemiol Infect*. 1987;99:517–22. <http://dx.doi.org/10.1017/S0950268800068011>
3. Annual report 2001 [in Portuguese]. Maputo (Mozambique): National Directorate of Livestock; 2002. p. 32–3.
4. Lagerqvist N, Moiane B, Bucht G, Fafetine J, Paweska JT, Lundkvist A, et al. Stability of a formalin-inactivated Rift Valley fever vaccine: evaluation of a vaccination campaign for cattle in Mozambique. *Vaccine*. 2012;30:6534–40. <http://dx.doi.org/10.1016/j.vaccine.2012.08.052>
5. Asante K, Brito R, Brundrit G, Epstein P, Fernandes A, Marques MR, et al., editors. Climate change report: study on the impact of climate change on disaster risk in Mozambique. Maputo (Mozambique): National Institute for Disaster Management (INGC); 2009.

6. World Organization of Animal Health (OIE). World animal health information database 2008, 2009 and 2010 [cited 2013 Apr 26]. [http://www.oie.int/wahis\\_2/public/wahid.php/Wahidhome/Home](http://www.oie.int/wahis_2/public/wahid.php/Wahidhome/Home)
7. Ikegami T, Makino S. The pathogenesis of Rift Valley fever. *Viruses*. 2011;3:493–519. <http://dx.doi.org/10.3390/v3050493>
8. Heinrich N, Saathoff E, Weller N, Clowes P, Kroidl I, Ntinginya E, et al. High seroprevalence of Rift Valley fever and evidence for endemic circulation in Mbeya region, Tanzania, in a cross-sectional study. *PLoS Negl Trop Dis*. 2012;6:e1557.

Address for correspondence: Nina Lagerqvist, Swedish Institute for Communicable Disease Control, Nobelsväg 18, SE-171 82 Solna, Sweden; email: [nina.lagerqvist@smi.se](mailto:nina.lagerqvist@smi.se)

## Avian Influenza A(H7N9) Virus Infections, Shanghai, China

**To the Editor:** On March 31, 2013, the National Health and Family Planning Commission of China notified the World Health Organization of 3 cases of human infections with avian influenza A(H7N9) virus. These cases were caused by a novel virus that was identified by laboratory testing at the China Centers for Disease Control and Prevention (CDC) on March 29 (1).

As of April 19, 2013, a total of 91 laboratory-confirmed human cases (17 deaths) of infection with avian influenza A(H7N9) virus were reported in 4 provinces in China (2). We report clinical features of 2 infected adults who died, 2 critically ill infected adults who recovered, and 1 infected child who had a mild case during this outbreak in Shanghai, China.

A 3.5-year-old boy had fever (39.5°C) for 3 days and mild rhinorrhea starting on March 31. He was admitted

to a district pediatric outpatient clinic on April 1. At admission, the child was given oseltamivir for 5 days, even though signs and symptoms had resolved. Nasopharyngeal swab samples were positive by real-time PCR for avian influenza A(H7N9) virus. All symptoms resolved uneventfully by April 3, and CDC was notified that avian influenza A(H7N9) virus was identified in his respiratory sample. The patient was discharged on day 11 after illness onset.

The 4 adult patients were given diagnoses of severe pneumonia with shortness of breath, dyspnea, and marked hypoxia (Table). Duration from disease onset to severe illness was 5–7 days. At admission, the 4 patients with severe cases had decreased peripheral blood leukocyte counts and increased levels of aspartate aminotransferase; 3 had increased levels of lactate dehydrogenase (Table).

All 4 adult patients had radiologically confirmed pneumonia and bilateral patchy alveolar opacities or diffused lobar consolidation with or without pleural effusion (Figure, Appendix, [wwwnc.cdc.gov/EID/article/19/7/13-0523-F1.htm](http://wwwnc.cdc.gov/EID/article/19/7/13-0523-F1.htm)). Findings on chest radiographs for severe cases requiring mechanical ventilation were consistent with those for acute respiratory distress syndrome.

Among the 4 severe cases in adults, a 52-year-old woman (patient 1) and a 49-year-old man (patient 2) died from acute respiratory distress syndrome and multiple organ failure on days 14 and 10, respectively, after disease onset and 1–2 days after progression to respiratory failure. Two other patients showed improvement and were virus negative 6 and 4 days after antiviral treatment. After 23–24 days of treatment in an intensive care unit, the 2 patients with severe cases recovered and were discharged (Table).

The 2 patients who died were given methylprednisolone. Of the 2 patients who recovered, 1 was given

a low dose of methylprednisolone for 1 week and the other was not given methylprednisolone. Although it is difficult to assess the role of glucocorticoids in treatment because of limited number of cases, caution is advised because of possible serious adverse events, including death, as reported for human infection with influenza A(H1N1) virus (4).

One of the adult patients reported exposure to poultry. The family of the child patient raised chickens and ducks, but these animals had no apparent disease, and cloacal swab specimens were negative for avian influenza A(H7N9) virus. One patient who died (patient 2) had frequent occupational exposure to poultry. Sixteen contacts of the child and 45 contacts of the 4 adult patients were monitored, and routine virologic sampling was performed. One contact (husband of patient 1) of a patient who died (Table) became febrile and was positive for avian influenza A(H7N9) virus on April 12 (day 24 after disease onset for patient 1); as of the date of this report, he was receiving treatment in an intensive care unit. However, it is difficult to tell if this is a case of human-to-human transmission or if both persons were exposed to infectious poultry. All remaining contacts had no symptoms and were negative for virus by PCR.

Several features of this avian influenza A(H7N9) outbreak are distinct from those of previous avian influenza outbreaks. Human infection with this virus showed a case-fatality rate of 18.7% (17/91), but this rate is not as high as that for avian influenza A(H5N1) virus (case-fatality rate 59%) (5).

Avian influenza A(H7N9) virus infection seems to cause more severe human illness than do other subgroups of H7 influenza A viruses (subtypes H7N2, H7N3, and H7N7), which are usually associated with poultry outbreaks but cause mild disease in humans. However, infection with avian