Rift Valley Fever in Goats, Cameroon

To the Editor: Rift Valley fever (RVF) is caused by an RNA virus (*Phlebovirus*, *Bunyaviridae*), which is carried by mosquito vectors (*1*). In nature, it is only known in Africa and the Arabian Peninsula. In the central African region, RVF has been reported in humans and livestock in the savanna of northern Cameroon and Chad (*2*,*3*) and from forests in the Central African Republic (*4*). Human epidemics are sometimes preceded by an increase in RVF virus (RVFV) prevalence in domestic ruminants, which manifests as increased abortions and high neonatal deaths (*3*). Human epidemics can have serious health implications, as demonstrated by the most recent outbreaks in Kenya in 1997 and 1998 (*5*) and Saudi Arabia in 2001 (*6*).

In June 2003, 14 goats from an urban market in Yaoundé (3.9°N, 11.5ºE), Cameroon, and 36 goats from 3 adjacent villages in tropical lowland rainforest ≈80 km south of Mvangan (2.4ºN, 11.8ºE), Cameroon, were sampled. The goats in the rural villages were bred locally and allowed to roam freely throughout the villages. No vaccinations had been given to goats in the rural sites. Goats in the urban market in Yaoundé generally originated in northern Cameroon and had been transported by train to Yaoundé. Owners did not report high levels of abortion or high neonatal deaths.

Jugular blood was collected in a 5-mL dry Vacutainer tube and centrifuged within 48 hours of collection. The frozen sera were shipped on ice to the Onderstepoort Veterinary Institute, the United Nations Food and Agriculture Organization reference laboratory for RVFV. An indirect enzyme-linked immunosorbent assay (I-ELISA) was used to detect RVFV immunoglobulin G (IgG) antibodies in 26 samples. In this assay, optical density readings were converted to a percentage of high-positive control serum (PP) and positive samples were those with PP ≥10. These samples were further tested with RVFV hemagglutination inhibition (HI), and samples with titers ≥20 were considered seropositive (*7*). Positive I-ELISA and HI samples were confirmed by using a serum neutralization (SN) assay (*7*,*8*). A sample was considered seropositive when it had an SN titer of ≥4, determined during experiments on laboratory-injected sheep (*7*–*9*) and testing of wild and domestic African ruminants (*7*). The SN assay has been shown to be highly specific; cross-reactivity with other viruses from the family *Bunyaviridae* is not likely (*9*).

Of the 26 goat samples tested for RVFV, 6 tested positive to RVFV (Table). Samples from 5 goats from the rural villages and 1 from the urban market had RVFV IgG PP ≥10. Samples from 2 goats from the rural villages had high RVFV HI titers (320 and 5,120). Three of the 6 samples from the rural villages with high IgG PP and HI titers had elevated neutralization titers (≥4).

The results indicate for the first time that RVFV is present in forests of southern Cameroon. Given the ages of the seropositive goats (2, 3, and 4.5 years), transmission of the virus occurred recently.

In savanna goat herds in northern Cameroon, RVFV IgG prevalence has been reported at 9% to 20% (*2*). To determine prevalence of RVFV in goats in southern Cameroon, more animals need to be sampled; the small sample size and isolation of the few rural villages are unrepresentative.

The presence of RVFV antibodies in domestic animals suggests that this virus may also be circulating in human populations, despite the absence of reports. A study of 21 persons in Ngoïla in southern Cameroon found no RVFV antibodies during a bloody diarrhea epidemic in 1998 (*10*,*4*); however, testing facilities for RVFV are not available in Cameroon, and the general population and healthcare providers have limited awareness of the virus and associated disease.

Central African forests have a high diversity of forest ungulates (>10 species), and RVFV has been reported from a number of wild African ungulates (*7*). The potential for exchange of RVFV and other pathogens between goats and wild ungulates could have substantial effects on animal production and on the conservation of wild species, some of which are considered near-threatened. Livestock disease surveillance can play a role not only in assessing the distribution of livestock pathogens but also in monitoring disease emergence.

Table. Site, age, and sex of goat samples positive for Rift Valley fever virus*

Site	Age, y	Sex	I-ELISA PP	HI titer	SN titer
Market	1.5	F	10	–	/
Rural	3	M	14	–	/
Rural	3.5	F	15	–	/
Rural	4	F	16	–	/
Rural	4.5	M	35	–	4
Rural	3	F	/	320	8
Rural	2	F	106	5,120	12

*By indirect enzyme-linked immunosorbent assay (I-ELISA), hemagglutination inhibition (HI), and serum neutralization (SN); PP, optical density expressed as a percentage of high-positive control serum. /, not tested; –, negative.

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References

1. Fontenille D, Traore-Lamizana M, Diallo M, Thonnon J, Digoutte JP, Zeller HG. New vectors of Rift Valley fever in West Africa. Emerg Infect Dis. 1998;4:289–93.
2. Zeller HG, Bessin R, Thiongane Y, Bapetel I, Teou K, Ala MG, et al. 1995 Rift Valley fever antibody prevalence in domestic ungulates in Cameroon and several West African countries (1989–1992) following the 1987 Mauritanian outbreak. Res Virol. 1995;146:81–5.
3. Ringot D, Durand J-P, Tolou H, Boutin J-P Davoust B. Rift Valley fever in Chad. Emerg Infect Dis. 2004;10:945–7.
4. Nakounné E, Selekon B, Morvan J. Veille microbiologique : les fièvres hémorragiques virales en République Centrafricaine ; données sérologiques actualisées chez l'homme. Bull Soc Pathol Exot. 2000;93:340–7.
5. Woods CW, Karpati AM, Grein T, McCarthy N, Gaturuku P, Muchiri E, et al. An outbreak of Rift Valley fever in northeastern Kenya, 1997–98. Emerg Infect Dis. 2002;8:138–44.
6. Balkhy HH, Memish ZA. Rift Valley fever: an uninvited zoonosis in the Arabian peninsula. Int J Antimicrob Agents. 2003;21:153–7.
7. Paweska JT, Smith SJ, Wright IM, Williams R, Cohen AS, Van Dijk AA. Indirect enzyme-linked immunosorbent assay for the detection of antibody against Rift Valley fever virus in domestic and wild ruminant sera. Onderstepoort J Vet Res. 2003;70:49–64.
8. Swanepoel R, Struthers JK, Erasmus MJ, Shepherd SP, McGillivry GM, Erasmus BJ, et al. Comparison of techniques for demonstrating antibodies to Rift Valley fever virus. J Hyg (Lond). 1986;97:317–29.
9. Swanepoel R, Struther JK, Erasmus MJ, Shepherd SP, McGillivray GM, Shepherd AJ, et al. Comparative pathogenicity and antigenic cross-reactivity of Rift Valley fever and other African phleboviruses in sheep. J Hyg (Lond). 1986;97:331–46.
10. Germani Y, Cunin P, Tedjouka E, Ncharre CB, Morvan J, Martin P. Enterohaemorrhagic *Escherichia coli* in Ngoila (Cameroon) during an outbreak of bloody diarrhoea. Lancet. 1998;352:625–6.

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HIV-1 CRF07_BC Infections, Injecting Drug Users, Taiwan

To the Editor: To date, Taiwan's human immunodeficiency virus type 1 (HIV-1) epidemic has primarily spread via sexual contact. The subtype B and circulating recombinant form (CRF) 01_AE account for >95% of all infections (*1*). However, since 2003 Taiwan has experienced a major outbreak of CRF07_BC among injecting drug users (IDUs).

The first wave of HIV-1 infections in Taiwan can be traced to the early 1980s, when a group of hemophilia patients received imported HIV-1-contaminated antihemophilia medications. By the time these medications had been replaced by heat-treated factor VIII concentrates, at least 53 patients had contracted HIV-1 infections (*2*). According to Taiwan's Center for Disease Control (CDC), HIV infections have been diagnosed in 9,229 persons (including 523 foreigners) as of July 31, 2005 (*3*). The number of persons living with HIV-1/AIDS has increased rapidly in the past few years, with a 77% increase in 2004, compared to 11% in 2003 (online Table, available at http://www.cdc.gov/ncidod/EID/vol12no04/05-0762.htm#table). According to the results of a risk factor analysis of people living with HIV-1/AIDS reported to the Taiwan CDC, the proportion of IDUs increased from 1.7% (13/773) in 2002 to 8.1% (70/861) in 2003 to 30.3% (462/1,521) in 2004 (online Table). The Taiwan CDC received reports of 1,241 IDUs diagnosed with HIV-1 infections from January 1 to July 31, 2005; these account for >75% of all reported HIV-1 infections in 2005 (*3*). The evidence points to an explosive epidemic of HIV-1 infections among IDUs in Taiwan since 2003, with no indication of a slowdown.

Taiwan has ≈60,000 IDUs (*1*). According to the Republic of China Ministry of Justice, the number of incarcerated drug offenders increased from 5,988 in 2003 to 9,303 in 2004; the rate of HIV-1 seropositive inmates increased from 13.3/100,000 in 2002 to 56.8/100,000 in 2004 (Y-M. Wu, Ministry of Justice, pers. comm.). Since all inmates are routinely tested for HIV-1 in detention centers, and all infected inmates are separated from HIV-1–seronegative inmates, the