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Yaks living in captivity in a Belgian animal park showed clinical signs of BT. A clinical examination performed on 1 yak showed loss of weight associated with a progressive weakness linked to anorexia, ulcerative and necrotic lesions on the muzzle with some crusts and mucopurulent nasal discharge, and udder erythema with papules and crusts. The tongue was severely swollen and cyanotic and protruded from the mouth (Figure). The animal was reluctant to move and was recumbent (possibly as a consequence of podal lesions linked to BT); it died 7 days after examination. Necropsies were performed on carcasses of this and another yak. The main lesions found were severe diffuse congestion of the lungs with edema and emphysema, acute hemorrhagic enteritis restricted to the ileum and jejunum, and petechial hemorrhages on the abomasums. No lesions characteristic of coronitis were noted.

Samples of spleen and bone marrow were taken and prepared according to the method of Parsonson and McColl (4). A real-time reverse transcription quantitative-PCR (RT-qPCR) targeting BTV segment 5 (RT-

qPCR_S5) was used to detect BTV RNA in tissues samples. Each test was performed in parallel with a RT-qPCR that amplifies β -actin mRNA as an internal control (RT-qPCR_ACT). Both assays were conducted according to Toussaint and others (5), with slight modifications. Briefly, total RNA was purified from 25 mg of tissue by Trizol extraction (Invitrogen, Carlsbad, CA, USA) and denatured by heating for 3 min at 95°C with 10% dimethylsulfoxide (Sigma-Aldrich, St. Louis, MO, USA). Reverse transcription reactions were conducted by using the Taqman reverse transcription reagents according to the manufacturer instructions (Applied Biosystems, Foster City, CA, USA). RT-qPCR reactions consisted of 1 \times concentrated Taqman fast universal PCR master mix (Applied Biosystems), 375 nM (β actin) or 500 nM (BTV) of each primer, 250 nM Taqman probe, and 5 μ L cDNA. Cycling conditions were as follows: 1 cycle at 95°C for 20 s, followed by 45 cycles of 1 s at 95°C, and 20 s at 60°C. The specificity of the RT-qPCR used had been previously tested against prototype strains of genetically related viruses (9 strains of epizootic hemorrhagic disease virus

Bluetongue in Captive Yaks

To the Editor: In August 2006, several northern European countries including Belgium reported cases of bluetongue (BT) (1). This noncontagious, arthropod-borne animal disease is caused by *Bluetongue virus* (BTV), genus *Orbivirus*, family *Reoviridae*. The genome of BTV consists of 10 segments of double-stranded RNA; 24 serotypes have been reported (2). Serotype 8 (BTV-8) was implicated in the emergence in Belgium (3). All ruminant species are thought to be susceptible to BT (2). We report laboratory-confirmed clinical cases of BT in yaks (*Bos grunniens grunniens*).



Figure. A captive yak infected with bluetongue virus. Tongue is swollen, cyanotic, and protruding from the mouth.

and 9 strains of African horse sickness virus) (5). The RT-qPCR tests confirmed BTV viremia.

The yak species in its natural biotope is usually rarely exposed to competent *Culicoides* vectors. Antibodies against BTV have been found in many wild ruminants (6), and our results extend the number of ruminant species susceptible to BTV. In the northern European BT outbreak, lesions in cattle and sheep were mainly localized to the regions of the muzzle, mouth, and eye; clinical signs were not always obvious (7,8). As in cattle and sheep, clinical signs in yaks were observed on the muzzle, in the periorbital region, and around and inside the mouth. These signs clearly reflected viral-induced endothelial damage triggering disseminated intravascular coagulation and a hemorrhagic diathesis commonly described in sheep and cattle (2). In our case, lesions depicted pronounced microvascular damage. According to the severity of the lesions and rates of illness and death observed, the yak, like sheep, appears to be highly susceptible to BTV.

In the epidemiology of BT in African countries, cattle and wild ruminant species such as antelopes play a role as asymptomatic reservoir hosts of the virus (2). Some wild ruminant species in captivity could also play this role in European countries affected by the recent BT outbreak. These cases could be of particular concern for all parks and zoos that gather numerous wild ruminants. Illness, reproductive failure, and deaths usually reported with BT (9) could generate substantial losses on these premises. Moreover, the source of BTV-8 in the northern European outbreak remains unclear, and the role of wild ruminant species has to be taken into account. In the future, European authorities should consider vaccination to prevent the spread of the disease in European member states (10). All premises with wild ruminants need to be involved in BT control and prophylaxis.

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Murine Typhus, Algeria

To the Editor: Rickettsioses, or typhoid diseases, are caused by obligate intracellular bacteria of the order Rickettsiales. The ubiquitous murine typhus is caused by *Rickettsia typhi*. Although cat fleas and opossums have been suggested as vectors in some places in the United States, the main vector of murine typhus is the rat flea (*Xenopsylla cheopis*), which maintains *R. typhi* in rodent reservoirs (1). Most persons become infected when flea feces containing *R. typhi* contaminate broken skin or are inhaled, although infections may also result from flea bites (1). Murine typhus is often unrecognized in Africa; however, from northern Africa, 7 cases in Tunisia were documented in 2005 (2).

We conducted a prospective study in Algeria which included all patients who had clinical signs leading to suspicion of rickettsioses (high fever, skin rash, headache, myalgia, arthralgia, eschar, or reported contact with ticks, fleas, or lice) who visited the Oran Teaching Hospital in 2004–2005 for an infectious diseases consultation. Clinical and epidemiologic data as well as acute-phase (day of admission) and convalescent-phase (2–4