

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

weeks later) serum samples were collected. Serum samples were sent to the World Health Organization Collaborative Center for Rickettsial Diseases in Marseille, France. They were tested by immunofluorescence assay (IFA), by using spotted fever group (SFG) rickettsial antigens (*R. conorii conorii*, *R. conorii israelensis*, *R. sibirica mongolitimonae*, *R. aeschlimanii*, *R. massiliae*, *R. helvetica*, *R. slovacica*, and *R. felis*) and *R. typhi* and *R. prowazekii* as previously reported (3). When cross-reactions were noted between several rickettsial antigens, Western blot (WB) assays and cross-adsorption studies were performed as previously described (4). A total of 277 patients were included. We report 2 confirmed cases of *R. typhi* infection in patients from Algeria.

The first patient, a 42-year-old male pharmacist who reported contact with cats and dogs parasitized by ticks, consulted with our clinic for a 10-day history of high fever, sweating, headache, arthralgia, myalgia, cough, and a 6-kg weight loss. He had not received any antimicrobial drugs before admission. No rash, eschar, or specific signs were found. Standard laboratory findings were within normal limits. No acute-phase serum sample was sent for testing. However, IFAs on convalescent-phase serum were negative for SFG antigens (except *R. felis*: immunoglobulin [Ig] G 64, IgM 128), but they showed raised antibodies against *R. typhi* and *R. prowazekii* (IgG 2,048, IgM 1,024).

The second patient, a 25-year-old farmer, was hospitalized for a 5-day history of fever, headache, diarrhea, and lack of response to treatment with amoxicillin and acetaminophen. He reported contact with cats and cattle. A discrete macular rash and pharyngitis were observed. Standard laboratory findings were within normal limits, except neutrophil count was elevated at 11.2/ μ L (normal levels 3–7/ μ L). Acute-phase serum was negative for rickettsial antigens. Convales-

cent-phase serum obtained 2 weeks later was positive for several SFG antigens (IgM only; the highest level was 256 for *R. conorii*), and higher levels of antibodies were obtained against *R. typhi* and *R. prowazekii* (IgG 256, IgM 256). WB and cross-adsorption studies confirmed *R. typhi* infection (Figure). Both patients recovered after a 3-day oral doxycycline regimen and have remained well. (A single 200-mg dose of oral doxycycline usually leads to defervescence within 48–72 hours [1]).

Murine typhus is a mild disease with nonspecific signs. Less than half of patients report exposure to fleas or flea hosts. Diagnosis may be missed because the rash, the hallmark for rickettsial diseases, is present in <50% of patients and is often transient or difficult to observe. Arthralgia, myalgia, or respiratory and gastrointestinal symptoms, as reported here, are frequent; neurologic signs may also occur (5). As a consequence, the clinical picture can mimic other diseases.

A review has reported 22 different diagnoses that were proposed for 80 patients with murine typhus in the United States (6).

Serologic tests are the most frequently used and widely available methods for diagnosis of rickettsioses (7). IFA is the reference method (7). However, *R. typhi* may cross-react with other rickettsial antigens, including SFG rickettsiae, but especially with the other typhus group rickettsia, *R. prowazekii*, the agent of epidemic typhus (8). Epidemic typhus is transmitted by body lice and occurs more frequently in cool areas, where clothes are infrequently changed, and particularly during human conflicts. It is still prevalent in Algeria (9).

This cross-reactivity led to some difficulties in interpreting serologic results (10). However, WB and cross-adsorption studies can be used when cross-reactions occur between rickettsial antigens. They are useful for identifying the infecting rickettsia to the

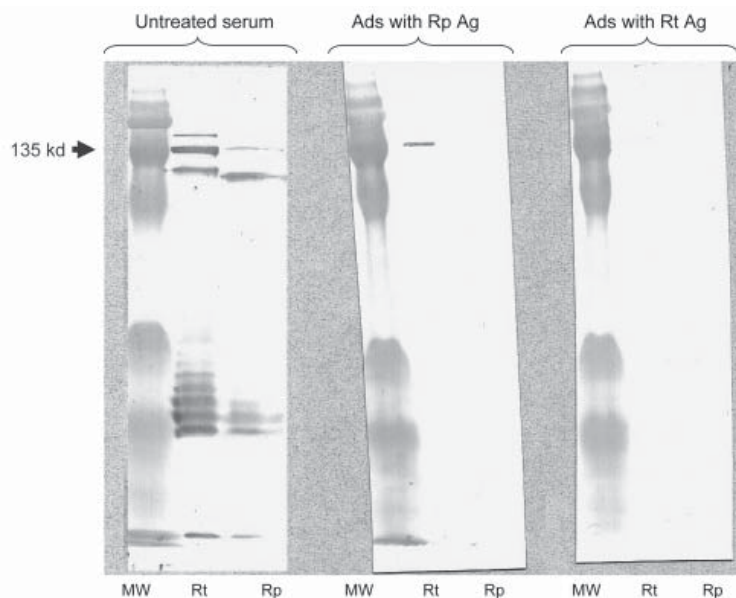


Figure. Western blot assay and cross-adsorption studies of an immunofluorescence assay-positive serum sample from a patient with rickettsiosis in Algeria. Antibodies were detected at the highest titer (immunoglobulin [Ig] G 256, IgM 256) for both *Rickettsia typhi* and *R. prowazekii* antigens. Columns Rp and Rt, Western blots using *R. prowazekii* and *R. typhi* antigens, respectively. MW, molecular weight, indicated on the left. When adsorption is performed with *R. typhi* antigens (column Ads with Rt Ag), it results in the disappearance of the signal from homologous and heterologous antibodies, but when it is performed with *R. prowazekii* antigens (column Ads with Rp Ag), only homologous antibody signals disappear, indicating that the antibodies are specific for *R. typhi*.

species level and for providing new data about the emergence or reemergence of rickettsioses, as reported here. These assays are, however, time-consuming and only available in specialized reference laboratories.

Clinicians need to be aware of the presence murine typhus in Algeria, especially among patients with unspecific signs and fever of unknown origin. Tetracyclines remain the treatment of choice.

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Natural Co-infection with 2 Parvovirus Variants in Dog

To the Editor: Canine parvovirus (CPV) emerged in the late 1970s, presumably by mutations in feline panleukopenia virus, and became a major viral pathogen of dogs worldwide (1). Between 1979 and 1981, the original type 2 virus (CPV-2) was replaced by a new genetic and antigenic variant, type 2a (CPV-2a). Between 1983 and 1984, CPV-2a was replaced by type 2b (CPV-2b), which differs from type 2a by only 1 epitope located at residue 426 of the VP2 capsid protein (2). CPV-2 does not replicate in cats, but the new variants replicate in dogs and cats (3). Recently, an antigenic change has been observed in a new strain, CPV-2c, isolated from domestic dogs in Italy (4). This variant was also detected in Vietnam (5), other countries in Europe (6), and the United States (7). CPV-2c was recently detected in cats (8) and is characterized by a replacement of aspartic acid with glutamic acid at residue 426 of the VP2 capsid protein.

To identify CPV types 2, 2a, and 2b, PCR methods were developed (9). However, these methods could not distinguish type 2c from type 2b (4). Consequently, we used a PCR–restriction fragment-length polymorphism (RFLP) assay with endonuclease *MboII*. This enzyme can distinguish type 2c from other CPVs (4). Recently, a real-time PCR assay based on minor groove binder (MGB) probe technology was developed for rapid identification and characterization of the antigenic variants. This assay is based on 1 nucleotide polymorphism in the VP2 gene (10).

In June 2006, a 10-week-old female dog (PT-32/07) was brought to the veterinary clinic in Figueira da Foz, Portugal, with clinical signs of parvovirus infection, after an episode of gastrointestinal disease in her litter. Three littermates, also brought to the clinic, showed no signs of infection. None of the dogs were vaccinated against CPV. Clinical signs in dog PT-32/07 were lethargy, anorexia, vomiting, diarrhea, and a temperature of 39.3°C. Identical signs were observed in 1 littermate 3 days later; the 2 other dogs did not show any signs other than lethargy and loose stools.

Rectal swab samples from all dogs were screened for CPV by using an immunomigration rapid test (Synbiotics Corporation, Lyon, France). Two of the dogs showed negative results, and 2 showed positive results. Feces, serum, and lingual swab samples were positive for parvovirus DNA. DNA was quantified by using a real-time PCR with TaqMan technology performed in an i-Cycler iQ (BioRad Laboratories, Milan, Italy).

CPV variants were characterized by using MGB probe technology. This technology uses type-specific probes labeled with different fluorophores (FAM and VIC) that can detect single nucleotide polymorphisms between CPV types 2a/2b and 2b/2c (10). MGB probes specific for type 2b were labeled with FAM in both type 2a/2b