Possibility of Leishmaniasis Transmission in Jura, France

To the Editor: The report of a human cutaneous leishmaniasis case acquired in Clairvaux-les-lacs (*1*) led us to carry out an investigation with the veterinary clinics in Jura Department, France. Clairvaux-les-lacs is a lakeside resort located in Jura, one of the areas in France with the coldest average temperatures, and is clearly located outside the usual leishmaniasis-endemic area. At least 31 cases of canine leishmaniasis were diagnosed by veterinary clinics in Jura during 2007–2011. Because these dogs were native of or traveled in the leishmaniasis-endemic area along the Mediterranean Sea, all veterinarians considered the infections as acquired outside Jura.

Although phlebotomine sand flies have not been reported in Jura to date, *Phlebotomus perniciosus* sand flies, proven vectors of leishmaniasis, have been found in 2 areas neighboring Jura: Côte-d'Or and Saône-et-Loire (*2*,*3*). We have also recently caught *P. mascittii* sand flies, a species with an unknown vectorial competence, in the Swiss region of Jura, Alsace, Champagne-Ardennes, and Belgium. Therefore, the presence of sand flies in Jura, particularly in wet and milder microclimatic areas (as Clairvaux-les-lacs), is likely, and canine infections could have been acquired locally.

A recent model predicted that new at-risk areas are mostly located in western France along the Atlantic coast (*4*). In accordance with this model, we report new foci of autochthonous canine leishmaniasis in Deux Sèvres, Loire-Atlantique, and Loiret. Canine leishmaniasis cases contracted in the Rhine Valley in Germany (*5*) and the canine cases in Jura argue for a northeastern spread of the disease-endemic area along the Rhone-Rhine axis and mild microclimatic niches. Entomologic and serologic surveys will be carried out in summer 2012 in Jura to look for evidence of possible indigenous transmission of leishmaniasis. These data should supplement the current model of northern spread of leishmaniasis-endemic areas.

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Etymologia: Prion

To the Editor: The January 2012 Etymologia section might confuse readers because it incorrectly reports that "prion" describes a noninfectious agent (*1*). In fact, prion—pronounced *pree'-on*—is a term coined in 1982 by Nobel laureate Stanley Prusiner to describe the novel infectious agent responsible for scrapie, a transmissble neurodegenerative disorder of sheep and goats. He proposed his new term to underscore that the agents are "small ***pro***teinaceous ***in***fectious particles" resistant to procedures that attack nucleic acids (*2*). In his seminal article, he summarized experimental data indicating that the molecular properties of this infectious agent differed from those of other infectious agents, including viruses, viroids, and plasmids; he proposed the word prion to replace other terms then in circulation, such as "unconventional virus" or "unusual slow virus–like agent."

Although Dr. Prusiner acknowledged that he could not exclude the possibility of a small nucleic acid contained within the interior of the prion particle, now 3 decades later, no nucleic acid in the agent has yet been identified. Increasingly accepted in the scientific community, prions are now considered to be a class of misfolded proteinaceous, infectious agents responsible for several types of human and animal transmissible spongiform encephalopathies. Their evolving defining characteristics classically include at least partial protease resistance, insolubility, and transmissibility. The term, prions, usually refers to the complete transmissible proteinaceous particles in nature or to their classically present, transmissible, protease-resistant oligomer cores, composed of protein fragments with molecular masses of ≈27–30 kDa.

Adding confusion to the terminology, it has become customary

for prion researchers to refer to the normal nonpathogenic conformation of prions as "cellular prion proteins" (*3*). When these normal cellular prion precursors convert to pathogenic prion proteins, the transmissible conformations are characterized by β-pleated sheets rather than the normal α-helix structure, and they do not elicit an immune response (*4*).

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Hepatitis E Virus Infection in Sheltered Homeless Persons, France

To the Editor: Kaba et al. (*1*) reported a seroprevalence of 11.6% for hepatitis E virus (HEV) among homeless persons in the city of Marseille, located in southern France, and a multivariate analysis suggested that injection drug use (IDU) was an independent risk factor for HEV transmission. We disagree with this reported finding.

We conducted a retrospective subanalysis of results from a multicenter therapeutic trial assessing HEV seroprevalence among HIV/hepatitis C co-infected patients in France (*2*). Serum samples from 84 IDU patients, enrolled during 2000–2002 were stored at −80°C. The mean ± SD age of the patients was 39 ± 4 years; 53 (63%) were men, 19 (23%) were born outside France, and 38 (45%) were living in southern France. HEV antibodies were tested with the same assay as that used by Kaba et al. (*1*), and HEV RNA was detected by using a real-time reverse transcription PCR amplifying open reading frame 3 (*3*). None of the patients had detectable IgM against HEV or HEV RNA. Test results for 3 (3.6%) patients were positive for HEV IgG. Two of them lived in southern France, resulting in a 5.3% (2/38) HEV prevalence for IDU patients living in this region, where HEV IgG prevalence for healthy blood donors has reportedly ranged from 9% to 16.6% (*4*).

The difference between our study, which demonstrated low HEV IgG prevalence in IDU patients, even in southern France, and the results from Kaba et al. (*1*) must be interpreted with caution because there were several epidemiologic differences between the 2 populations. Moreover, there is a risk for false-negative serologic results for HIV patients because of impaired immunity, and the predictive value of serologic testing is probably low because of the artificially low HEV prevalence reported for this population. Despite these limitations, our study suggests that the high prevalence of HEV infection among homeless persons in southern France was not influenced by IDU, but reflected the general epidemiology of HEV in this region.

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