following years the number of cases decreased. Ninety-one and 38 human cases were confirmed, respectively, in 1990 and during the first six months of 1991. Since the first diagnosis, HFRS cases were observed only in the northern and eastern part of France except for 2 imported cases from Finland and Romania (laboratory infection). Recently, viruses were isolated twice: one from a early serum sample of a human case. By monoclonal antibodies, both viruses belongs to the Puumala serogroup. Patients are between 11 and 79 years old, mainly male (81.8%), lived in the country and noticed rodent in the vicinity in 68% of cases. Clustering of cases in the same family occurred several times. The symptoms observed are classical of the Scandinavian nephropathia epidemica form with a nonspecific, pseudogrippal early phase followed by thrombopenia and acute renal failure with high levels of serum creatinine, proteinuria and hematuria. Only 5% of the patients were dialysed. Neither death nor sequellae (except mild hypertension in early convalescent phase of 2 cases) were observed. High titer (1024-2048) of specific antibodies against Puumala-related viruses were detected by IFA as soon as 5-7 days after the onset of the disease and remained so for several years. The incidence of HFRS is significant in France and appears to be higher than elsewhere in western Europe except for the Scandinavian countries. Physicians in the endemic areas should be aware of this renal disease.

143 REPLICATION AND PERSISTENCE OF CRIMEAN-CONGO HEMORRHAGIC FEVER VIRUS IN TWO SPECIES OF WEST AFRICAN TICKS. Gonzalez JP*, Camicas JL, Zeller HG, Cornet JP, Some J, and Wilson ML. Institut Francais de Recherche scientifique pour le Developpement en Cooperation, Dakar, Senegal; Institut Pasteur, Dakar, Senegal; Ministere de l'Agriculture et l'Elevage, Bobo-Dioulasso, Burkina Faso; and Yale University School of Medicine, New Haven, CT.

Crimean-Congo hemorrhagic fever (CCHF) virus is a tick-borne *Nairovirus* producing severe zoonotic disease throughout much of Eurasia and Africa. Many of the >30 tick species found naturally infected may be capable of maintaining CCHF virus transmission; studies of vector competence are needed to understand this, as well as the risk of human infection. We studied CCHF virus survival and replication in *Hyalomma truncatum* and *Amblyomma variegatum*, two ticks that are abundant and widespread in Africa. Adult male and female ticks were infected by intracoelomic inoculation. Ticks were later tested for virus by suckling mouse inoculation, antigen capture ELISA, and immunoflorescent antibody. Both species became infected, although in *H. truncatum* the titer of CCHF virus was greater and was detected longer (up to 10 mo. post infection). In another experiment, hypostomectomized male *H. truncatum* that had been inoculated with CCHF virus were allowed to feed and mate with uninfected females on laboratory rabbits. Female ticks became infected, apparently during spermatophore transfer as males were unable to feed. Subsequent transovarial transmission was observed. Results are discussed in the context of CCHF epidemiology in West Africa.

144 RIFT VALLEY FEVER VIRUS ANTIBODY IN HUMAN SERA COLLECTED AFTER AN OUTBREAK IN DOMESTIC ANIMALS IN KENYA. Logan TM*, Davies FG, Linthicum KJ, and Ksiazek TG. Virology Division, U.S. Army Medical Research Institute of Infectious Diseases, Ft. Detrick, MD; Veterinary Research Laboratory, Kabete, Kenya; and Disease Assessment Division, U.S. Army Medical Research Institute of Infectious Diseases, Ft. Detrick, MD.

An outbreak of Rift Valley fever (RVF) occurred among herds of domestic animals during June and July 1989, on farms along the margin of Lake Naivasha, Kenya. During October 1989, finger-prick blood samples were collected from herdsmen that worked with affected herds on the 3 farms that had RVF virus-infected herds. Rift Valley fever virus antibodies were measured using an ELISA test. Blood samples that were RVF IgG antibody positive were tested for RVF IgM antibodies. Twelve of the 30 (40%) herdsmen tested in this study had detectable RVF IgG antibody. Five of these twelve IgG-positive samples also contained RVF IgM antibodies. No human disease was seen during this outbreak and none of the herdsmen could recall being sick during the outbreak period despite an association with the RVF affected herds. Thus the 5 RVF IgM-positive herdsmen may be the only humans that encountered RVF

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