Rift Valley fever virus (RVFV) is a vector-borne zoonotic disease causing severe mortality in young ruminants and abortion in pregnant females. The virus is transmitted by mosquitoes or direct contact with body fluids or tissues from viremic animals.1

In Madagascar, RVFV was first isolated in 1979 from pools of mosquitoes captured in the highlands without any reported human or animal cases.2 First epizootics occurred in 1990–1991 in livestock of the highlands during two consecutive rainy seasons.3 In 2008–2009, a large RVFV outbreak occurred, affecting livestock and humans, in most of the country districts.4 Carroll et al.5 showed that RVFV strains isolated in 1979, 1990–1991, and 2008–2009 were genetically different and probably introduced from the African mainland by cattle trade.

Because of a temperate climate, the highlands were not considered at risk for RVFV transmission. However, this area was heavily infected during the 2008–2009 outbreak.4 In a pilot area of these highlands (200 km²; the so-called Anjorozoro District), a serological survey was conducted on ear-tagged cattle in 2009 and 2010. In 2009, the estimated seroprevalence rate was 28% (95% confidence interval = 25–31%; N = 894). Between 2009 and 2010, a seroconversion rate of 7% was observed (95% confidence interval = 5–10%; N = 516).5,7 A large serological and virological study performed in wild rodents of this area suggested that these animals were not involved in the RVFV maintenance.8 In January of 2011, 179 bovines were present in these four villages, among which 24 bovines had tested negative in 2010 and were still seronegative in January of 2011. In December of 2011, 173 bovines were present in the studied villages, of which 149 bovines could be sampled. In each month of 2011, between 88 and 127 bovines were sampled in each village (1,353 blood samples as a whole). Blood samples were tested for anti-RVFV immunoglobulin M (IgM) and IgG using ID Screen RVF IgM Capture (IDvet, Grabels, France) and ID Screen RVF Competition Multi-species (IDvet), respectively. Molecular detection using real-time quantitative reverse transcription polymerase chain reaction was also been performed as previously described.10 Cattle movements—death, slaughtering, birth events, and trade—were monitored.

Estimated birth and mortality rates as well as the number of exchanged animals were consistent with previous observations.7 Newly introduced animals were systematically sampled. All of these animals were seronegative (N = 25). Four animals of the cohort that tested negative in January were found IgG-positive several months later (two animals in May and two animals in August). In the following months, each of them showed an increase of the IgG level. The specificity of these IgG-positive results was verified by seroneutralization. Although clinical signs and IgM were not observed or detected, these four animals seemed to be newly infected, because they were seronegative at the beginning of the study, were IgG-positive after a few months, and showed a subsequent increase of IgG titer in the next months). Furthermore, they were infected during the dry and cold season, when RVF vectors are rare or absent.9

Viral RNA was detected in two of these animals with sequences specific of RVFV. Viral genome was detected in four consecutive samples of a 10-year-old female (September to December) and one sample of a 7-year-old male (December) from the four animals. These animals were located in two villages—Anorana for the male and Antanifotsy for the female—that were strongly affected by the outbreak of 2008–2009, and human cases had been reported at that time. Both villages were surrounded by different landscapes (i.e., savannah and edge of the primary rainforest, respectively). In 2009, seroprevalence levels of 14% for Anorana (N = 7) and 26% for Antanifotsy (N = 39) had been estimated. In 2010,
no seroconversion (N = 3) had been detected in Anorana, and a seroconversion rate of 12% (N = 17) had been estimated in Antanifotsy. No clinical case was reported in human or cattle throughout the pilot area since the 2007–2008 outbreak.

Serological and virological analyses suggest that seroconversions occurred in 2011 in the Madagascar highlands. These seroconversions were observed in animals that did not move from their village during the whole serological survey. Three years after the last clinical cases, these seroconversions evidence an RVFV local circulation when mosquitoes are rare or inactive. Direct transmission mechanisms, virus overwintering in vectors (residual active mosquito population during the dry and cold season and ticks11,12), or the existence of a wild reservoir other than wild terrestrial small mammals8 may explain these seroconversions.

Received September 18, 2013. Accepted for publication November 20, 2013.

Acknowledgments: We acknowledge the personnel of the Veterinary Services and breeders for their involvement.

Financial support: This study was granted by the Regional Centre of Monitoring of the Indian Ocean (CRVOI) through the project entitled “Rift Valley fever in the Indian Ocean Islands” (RIFT-OI), International Centre for Research in Agriculture and Development, and French Agency for Food, Environmental and Occupational Health and Safety.

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