pathogens intrusion. Blackleg (1970, 1995) and the contagious ecthyma (1999) were probably introduced into the country by live ruminants imported from Madagascar (9). Since 2002, importation of live animals from Tanzania has been common, increasing the risk of introducing continental pathogens or vectors as illustrated with outbreaks of East Coast fever in 2003 and 2004 in Grande Comore (10). RVFV circulation presented in this study is another example of the exposure of the Republic of Comoros to emerging pathogens and potentially bears major consequences for the local economy and for public health. The improvement of the Comorian veterinary services and the setting up of surveillance programs are essential to limit the risk of introducing devastating diseases in the area.

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Yersinia pestis in Small Rodents, Mongolia

To the Editor: Plague is known to be endemic in several areas of Mongolia, but transmission to humans seems to play only a minor role because the number of recognized cases is relatively low (Figure 1). The first human cases in Mongolia were reported to the World Health Organization in 1980, and <20 human cases have occurred each year since then (2). However, human plague was first reported in 1897 (3), such infections have been documented since the 1940s, and Yersinia pestis can be found in many provinces of Mongolia (Figure; T. Damindorj, pers. comm.) (3,4)

The most common source of
human plague in Mongolia is contact with and consumption of the marmot (*Marmota sibirica*) (1). Moreover, the great gerbil (*Rhombomys opimus*) and the Mongolian gerbil (*Meriones unguiculatus*) are suspected of being enzootic reservoirs. Although small rodents are also assumed to be reservoirs of *Y. pestis*, the interaction of individual mammals or fleas of particular species in the infectious cycle and the dynamics of an epizootic are not yet clear (5). In a retrospective study, we screened tissue samples from small rodents for *Y. pestis* DNA to investigate the prevalence of *Y. pestis* in a potential enzootic reservoir.

During the course of zoologic investigations in Mongolia during 2002, 2005, and 2006, 133 rodents (gerbils, jerboas, and squirrels) were trapped by standard methods (5), dissected, and cataloged (Figure). Documentation included species, sex, date and location of trapping, animal size (weight, length) and organ dimensions, as well as all pathologic findings. Although the trapped animals showed a high degree of parasitic infestation, signs of a severe infectious disease were not observed. After the dissection of animals, samples were conserved in 70% ethanol.

Subsequently, total DNA was extracted from alcohol-conserved spleen and liver tissue of 133 animals by using QIAamp DNA Mini Kit (QIAGEN, Hamburg, Germany), according to the manufacturer’s instructions. Screening was performed by using a real-time PCR targeting the *pla* gene of *Y. pestis* pPCP1, including a PCR inhibition control, as described (6). As positive control, the *Y. pestis* vaccine strain EV76 was used. As negative controls, we included tissues of 53 laboratory rodents, which were processed analogs, beginning with DNA extraction.

In the real-time PCR targeting the *pla* gene, 7 (5.3%) of 133 spleen tissue samples were positive for *Y. pestis*. In contrast, all liver samples and samples of laboratory rodents tested negative. Identification of several host species was supported by partial sequencing of the cytochrome b gene (7). The animals tested positive for plague were gerbils (*Meriones* sp., 1; *M. unguiculatus*, 2; *Rhombomys opimus*, 2) and jerboas (*Allactaga sibirica*, 1; *Cardiocranius paradoxus*, 1).

The identity of the 230-bp *pla* PCR fragment was confirmed by DNA sequencing, showing 100% similarity to the *pla* gene sequences deposited in the European Molecular Biology Laboratory nucleotide database. Molecular subtyping of the 7 *pla*-positive DNA samples was attempted by clustered regularly interspaced short palindromic repeats analysis, targeting the 3 loci YPa, YPb, and YPc, respectively. Also included was DNA originating from the above-mentioned negative control tissues. However, only 1 sample from the spleen of a *M. unguiculatus* gerbil found the YPb locus, which then was sequenced, and resulted in the spacer signature b1-b2-b3-b4-b5*. This signature is known from a *Y. pestis* biovar, Orientalis, that has been isolated from *Rattus flaviventer* rats in the plague focus of the Yunnan–Guangdong–Fujian provinces in the People’s Republic of China (8).

Detection of *Y. pestis*–specific DNA in wild rodents has been described. For instance, a wild rodent community in the eastern Sierra Nevada mountains in the United States was screened for plague by *pla*-specific real-time PCR; of 89 rodents, 1 chipmunk (1.1%) had positive results (9).

The permanent presence of *Y. pestis* in rodent communities in North America has led to smaller and more distant-living colonies of prairie dogs (10). Strikingly, in the present study, >5% of the screened rodents were found to carry *Y. pestis* DNA. This high number was unexpected for the investigated areas, which have had a low level of plague activity (Figure). To our knowledge, *Y. pestis* has also not yet been reported in Manlai Sum (district) in the Umnugovi Aimag (subdivision) (Figure) (2–4) nor has the presence of *Y. pestis* DNA in a *Cardiocranius paradoxus* jerboa.

Our findings emphasize that rodents play a role as zoonotic reservoirs of *Y. pestis* in Mongolia and...
that the actual prevalence of plague seems to be underestimated. The low population density in Mongolia explains the low amount of illness in humans. Further investigations should include the screening of rodent populations near the plague-positive loci. In addition, fleas and other parasites (and also predators of small mammals) should be studied. Mongolia is a key area of plague genesis and therefore is an ideal location for more detailed study of the role of rodents as epizootic and enzootic reservoirs of \textit{Y. pestis}.

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Typhoon-related Leptospirosis and Melioidosis, Taiwan, 2009

To the Editor: Global climatic changes have resulted in more natural disasters worldwide. These natural disasters can then cause outbreaks of emerging infectious diseases, including leptospirosis and melioidosis (1–7). In 2009, the moderate-strength Typhoon Morakot, with a maximum cumulative rainfall amount up to 3,059.5 mm, damaged Taiwan. After this natural disaster, unusual epidemics of leptospirosis and melioidosis occurred. The main objective of this study was to clarify whether these epidemics have resulted from this natural disaster.

Information about past typhoons that affected Taiwan was collected from the website of the Taiwan Meteorological Bureau (http://photino.cwb.gov.tw/tyweb/mainpage.htm; www.cwb.gov.tw) during January–August 2009. The influential period of Morakot was in the 32nd week (August 5–August 10) in 2009. To evaluate the effects of this specific natural disaster, we divided the period into 2 intervals for analysis. The early period (before the typhoon) was from the 28th through the 32nd weeks, and the latter period (after the typhoon) was from the 33rd through the 37th weeks in 2009. Information regarding 16 typhoons from 2000 through 2009 was further collected to evaluate effects of typhoon level, rainfall level, and maximum cumulative rainfall amounts on case numbers of leptospirosis and melioidosis after a typhoon.

The historical records of numbers of leptospirosis and melioidosis cases for analysis were obtained from the database collected weekly by the Centers for Disease Control, Taiwan. The information was referred to the website of the Taiwan Center for