rate is 0.04–0.1 per 100,000 (*10*). In this study, the detection rates of *V. cholerae* O1 in stools from patients with severe diarrhea were 23% (49/213 cases) in 2012 and 14% (35/250 cases) in 2013, respectively. Although our investigation is merely the tip of the iceberg for studies of cholera in Myanmar, our data provide crucial initial insights into the genetic backgrounds of recent Yangon isolates of *V. cholerae* O1. Epidemiologic surveillance linked to laboratory investigations is need to minimize the risk for *V. cholerae* infection in children.

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Role of Race/Ethnicity in Pulmonary Nontuberculous Mycobacterial Disease

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To the Editor: We read with interest the study of gender and age in nontuberculous mycobacterial (NTM) lung disease case-patients in Taiwan (*1*). NTM lung disease is relatively uncommon; however, the exact prevalence of NTM lung disease and causative organisms are largely unknown in many regions of the United States because the disease is not reportable. A recent study using Medicare claims data in the United States showed that the annual prevalence of NTM lung disease increased from 20 cases/100,000 persons in 1997 to 47 cases/100,000 persons in 2007 (*2*). The study also showed that Hawaii had the highest period prevalence of cases (396 cases/100,000 persons), which was at least partially attributed to the large Asian/Pacific Islander population (*2*). During June–December 2011, we conducted a cross-sectional study to evaluate the epidemiologic and clinical significance of NTM isolated from patients in Honolulu, Hawaii; the patients had suspected pulmonary tuberculosis (TB) and were in airborne isolation at a university-affiliated, tertiary-care hospital.

NTM cases were defined according to the 2007 criteria of the American Thoracic Society/Infectious Diseases Society of America (*3*). The process required to establish a diagnosis of NTM lung disease is sometimes lengthy; thus, patients who did not initially meet the disease criteria but who had cultures positive for NTM were reviewed again 1 year after the original data were collected to see if follow-up microbiological and radiographic studies would confirm the presence of NTM lung disease. Descriptive statistics were used to describe categorical and continuous variables. During June–December 2011, a total of 113 patients with suspected pulmonary TB were placed into isolation at the tertiary-care hospital. Of these patients, 85 (75.2%) were men and 28 (24.8%) were women; the median age was 59.8 ± 17 years. Eighteen (15.9%) patients were white, 92 (81.4%) were Asian/Pacific Islander, and 1 (0.9%) was African American; for 2 (1.8%) patients, race/ethnicity was classified as not specified/other.

Of the 113 isolated patients, 21 (18.6%) were positive for mycobacteria. Of these 21 patients, 14 (66.7%) were men and 7 (33.3%) were women; the median age

was 64.3 ± 17.3 years. Three (14.3%) of these patients were white, and 18 (85.7%) were Asian/Pacific Islander. *Mycobacterium tuberculosis* and NTM were identified in samples from 3 (14.3%) and 18 (85.7%) of the 21 patients, respectively. Of the 18 patients with NTM-positive samples, 4 (22.2%) had definite NTM lung disease (all of these patients were Asian/Pacific Islander); 2 (11.1%) had probable NTM lung disease; and 12 (66.7%) had possible NTM lung disease. *M. chelonae* (identified by DNA sequencing) was the causative agent for most of the definite cases (n = 3, 75%), and the largest proportion of possible cases was caused by *M. avian* complex bacteria (n = 5, 41.7%).

Our finding that 22.2% (4/18) of the patients in Honolulu with NTM-positive clinical samples during June–December 2011 received a definite diagnosis of NTM lung disease is slightly higher than but consistent with reports from other regions, which show that 9.8%–17.0% of such patients receive a definite NTM disease diagnosis (*4*,*5*). For unclear reasons, the number of NTM disease cases appears to be highest in Asian/Pacific Islander populations. Determining the reason(s) for this discrepancy should be the subject of future research efforts.

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Rickettsial Infections in Monkeys, Malaysia

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To the Editor: The cynomolgus monkey (*Macaca fascicularis*), also known as the long-tailed macaque or crab-eating monkey, is commonly found in the Southeast Asia region (*1*). The macaque has been associated with several bacterial infections, such as those caused by hemotropic *Mycoplasma* and *Bartonella quintana* (*2*). As a result of rapid deforestation and changes in land use patterns, cynomolgus monkeys live in close proximity to human-populated areas (*1*). Human–macaque conflict may increase the risk for zoonoses.

Little is known about rickettsial and anaplasma infections in cynomolgus monkeys in Malaysia. Although *Rickettsia* spp. RF2125 and Rf31 have been identified from cat fleas in Malaysia (*3*), the presence of *Anaplasma bovis* in monkeys is not known.

Rickettsia felis, a member of the spotted fever group rickettsiae, is an emergent fleaborne human pathogen distributed worldwide (*4*). The obligate intracellular bacterium has been identified from cats, dogs, opossums, and the ectoparasites of various mammalian hosts. Several uncultured rickettsiae genetically closely related to the *R. felis*–type strain URRWXCal2 (referred to as *R. felis*–like organisms and including *Rickettsia* spp. RF2125, Rf31, *Candidatus* Rickettsia asemboensis, and others) have also been identified from various arthropods and fecal samples of primates (*5*). *A. bovis* is a gram-negative, pleomorphic, tickborne intracellular bacterium that infects a wide range of mammal species in many geographic regions (*6*).

To learn more about these infections in monkeys, we examined blood samples from 50 cynomolgus monkeys caught by the Department of Wildlife and National Parks at 12 residential areas in Peninsular Malaysia during a population management and wildlife disease surveillance program (January 2012–December 2013). Most monkeys (14 male, 36 female) were adults and were active and healthy. DNA was extracted from 200 μL of each blood sample by using a QIAamp DNA Mini Kit (QIAGEN, Hilden, Germany). We performed PCRs selective for the rickettsial citrate synthase gene (*gltA*) by using primers CS-78 and CS-323 and for the 135-kDa outer membrane protein B gene (*ompB*) by using primers 120-M59 and 120-807 (*7*). As positive controls, we