conducted by the African Large Predator Research Unit, University of Bloemfontein. Whole blood samples were drawn aseptically from each lion into EDTA tubes, stored at 4°C before being returned to the laboratory, and then frozen at –70°C in the laboratory. Subsequently, blood samples were thawed, and an aliquot was plated onto 10% sheep blood–enriched agar and incubated at 37°C in a 5% CO_2 atmosphere for a maximum of 45 days. One culture yielded putative bartonellae (small, smooth, white-gray colonies) after 14 days' incubation. A crude DNA extract was prepared from this isolate and used as a template in previously described polymerase chain reaction–based assays to detect and identify *Bartonella* species which targeted fragments of the 16S rRNA encoding gene and 16S/23S intergenic spacer region (6). Amplification products of the expected size were obtained from the DNA extract. The nucleotide base sequence of each product showed that each shared 100% similarity with sequences of other *B. henselae* isolates held in GenBank. The 16S rRNA gene sequence was identical to that of type II variants.

Antisera from 62 of the 65 samples were tested for the presence of anti-*Bartonella* immunoglobulin G antibodies using an enzyme-linked immunosorbent assay previously evaluated to detect antibodies in domestic cats (7). Eighteen of the samples had matrix scores above the upper limit of the normal range of values observed in uninfected cats, thus indicating past exposure to *Bartonella* species. No serum from the *B. henselae* culture-positive animal was available for testing.

Our findings confirm that lions are susceptible to infection by *B. henselae*, but their role as reservoirs for this species remain unclear. The observed prevalence of infection (1.5%) and exposure rate (29%) in our study are lower than those typically observed in domestic cats, particularly in warmer regions of the world. Nonetheless, our serologic data do suggest that a substantial proportion of the lions are exposed to bartonellae. Although limited, our assessment of the lion *B. henselae* isolate suggests that it is within the genetic spectrum of strains associated with domestic cats, and lions may serve as an extension to this reservoir. The extent of contact between domestic cats, or their ectoparasites, and the farmed lions we studied is likely to be minimal, given the remoteness of the enclosures (the infected lion lived on a cat-free ranch). However, the lions may have contact with other wild-living felids such as the African wild cat (*Felis silvestris lybica*), small spotted cat (*Felis nigripes*), and the caracal (*Caracal caracal*) which are endemic to the region.

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Mycobacterium tuberculosis Transmission from Human to Canine

To the Editor: This report is the first known of a case of epidemiologically associated tuberculosis (TB) in a human and a canine caused by the same strain, confirmed by genotyping. In Tennessee, a 71-year-old woman with a 3-week history of a productive, nonbloody cough was evaluated. She lived alone, and standard epidemiologic investigation of family members and other close contacts showed no apparent TB exposure. A TB skin test 20 years earlier had been negative. Chest radiograph showed infiltrates and atelectasis in the upper lobe of the right lung. A TB skin test resulted in a 14-mm area of induration. Sputum stained positive for acid-fast bacilli (AFB) and was positive for *Mycobacterium tuberculosis* by DNA probe and culture. The organism was sensitive to standard antitubercular medications.

Treatment was initiated with isoniazid, rifampin, and pyrazinamide. After 14 days of daily, directly observed therapy, the patient complained of nausea, vomiting, and diarrhea. Treatment adjustments were made, and therapy was completed 11 months later with a complete recovery.

Six months after the patient's TB diagnosis, she took her 3-1/2-year-old male Yorkshire Terrier to a veterinary clinic with cough, weight loss, and vomiting of several months' duration. The dog lived indoors and had been a constant companion to the patient for 3 years. Because of the owner's diagnosis, TB was suspected. The dog's initial sputum sample was negative on AFB staining and *M. tuberculosis* nucleic acid amplification assay.

Eight days after discharge from a referral veterinary teaching hospital with a presumptive diagnosis of TB, the dog was euthanized because of urethral obstruction. Liver and tracheobronchial lymph node specimens collected at necropsy were positive by AFB stain and positive for *M. tuberculosis* complex by polymerase chain reaction. Cultures of liver, lung, and kidney specimens were positive for *M. tuberculosis.* The *M. tuberculosis* isolates from the dog and its owner had an indistinguishable 10-band pattern by IS*6110*-based restriction fragment length polymorphism genotyping (Figure) (1).

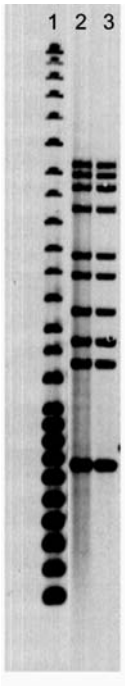

Figure. Ten-band *Mycobacterium tuberculosis* restriction fragment length polymorphism pattern. Lane 1, 25-band Centers for Disease Control and Prevention standard; lane 2, human case; lane 3, canine case.

This report is the first known of epidemiologically associated TB in human and canine cases to be confirmed by *M. tuberculosis* genotyping. The weight of historic data on human-canine TB supports our conclusion that the human owner was the likely source of the canine TB in this instance (2–5). In a review of 48 dogs and cats with known exposure to human TB, 7 (14.6%) were culture-positive for *M. tuberculosis* (2). Another series of eight canine TB infections documented by necropsy showed that seven of the dogs had a close association with humans with active TB disease (5). Other forms of mycobacterial infections, most notably *M. bovis*, have also been epidemiologically linked in humans and dogs (4,6). Cases in which dogs and cats infected with *M. bovis* or *M. tuberculosis* have infected humans have also been reported (4).

Genotyping has become a powerful tool for confirming epidemiologically linked transmission of *M. tuberculosis*. Two previous reports showed genotype matches between human and elephant TB cases and between human and monkey TB cases (7,8).

In our case, signs and symptoms that likely represented onset of TB appeared first in the woman and followed several weeks to months later in her pet dog. The owner often kept the dog in her lap, and the dog was allowed to lick the owner's face. A thorough, standard epidemiologic investigation did not identify any other infected contacts of the dog or owner; however, because the patient had limited social contacts, nonstandard investigation such as social network analysis was not formally conducted.

Cross-contamination of specimens was unlikely to have occurred at the laboratory at which cultures for *M. tuberculosis* were confirmed; several months passed between the times the two isolates were identified and subsequently sent to the Centers for Disease Control and Prevention for genotyping. This pattern has never been identified in a national database of >10,000 unique patterns, so this match is not likely to be due to anything other than transmission between the dog and its owner. Because systematic genotyping is not performed routinely in Tennessee, we are unable to determine more definitively whether this pattern has ever occurred in the state.

Although the true risk for TB transmission from humans to dogs, and vice versa, is not known, pet owners, physicians, and veterinarians should be aware of this potential. While standard tests, such as culture for *M. tuberculosis*, may be helpful in understanding the dynamics of TB between humans and other animals, genotyping has become the standard for confirming the association.

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Taura Syndrome Virus and Mammalian Cell Lines

To the Editor: Audelo-del-Valle et al. concluded that human and monkey cell lines (rhabdomyosarcoma [RD], human larynx carcinoma [Hep-2C], and Buffalo green monkey kidney [BGM]) could be infected by a penaeid shrimp virus, Taura syndrome virus (TSV) (1). They also concluded that *Penaeus* spp. could likely be a reservoir of a virus that might become pathogenic to humans and other mammals (1).

Though researchers have tried to develop continuous marine crustacean cell lines for >30 years, their efforts have not been successful. The lack of continuous marine crustacean cell lines has become an obstacle to conducting research on viral disease in shrimp (2,3). During the last 20 years, many researchers searched for substitute cell lines on which to study shrimp viruses (4,5). Audelo-del-Valle et al. likely chose RD, Hep-2C, and BGM cell lines because TSV was "recently reported to be genomically related to the cricket paralysis virus of the *Cripavirus* genus, family *Dicistrovirdae* of the 'picornavirus superfamily'" (1), and these cell lines were susceptible to some picornaviruses. If their findings are correct, they may have found substitute cell lines for isolating and studying TSV. To confirm their findings, we selected two mammalian cell lines, Hep-2 and Vero, which are highly sensitive to some picornaviruses (6,7), and tested them to determine their susceptibility to TSV.

The TSV extract was prepared from frozen cephalothoraxes of shrimp, *Litopenaeus vananmei*, that were infected with TSV (confirmed by standard reverse transcriptase–polymerase chain reaction [RT-PCR]) (8). To verify the TSV extract's validity, 50 µL of diluted TSV extract (approximately 0.8% volume of shrimp body weight) was injected into each of eight healthy shrimp, *L. vannamei.* Another eight healthy shrimp (control group) were injected with a diluted extract prepared from frozen cephalothoraxes of healthy shrimp. All of the TSV-injected shrimp died within 6 days and were TSV-positive; control shrimp did not die and were TSV-negative, which showed that our TSV extract was active and viable. The TSV extract was transferred into cell culture flasks according to a method previously reported (9). The cell monolayers were exposed to 100 µL of diluted and filtered TSV extracts for 1 hour; the extracts were then removed from the flasks, 2 mL of maintenance medium was added to each flask, and the flasks were incubated in three separate rooms at 37°C, 35°C, and 33°C, respectively. If a cytopathic effect (CPE) was not evident within 7 days, cell monolayers were washed with Hank's balanced salt solution (HBSS) six times to eliminate viral particles from the primary extract or from infected cells. Then cells were lysed in 2 mL HBSS, the lysate was clarified, and a portion of it was used for the first passage. This procedure was repeated three times. The control cell lines were injected with diluted extract from healthy shrimp, and passage was conducted as described earlier. RNA samples were extracted and purified from 150-µL lysates of primary cells and four passage cells and used as templates for RT-PCR analysis to determine the presence of TSV.

No CPE was observed in either the Hep-2 or Vero cell line that had been injected with TSV after 7 days of culture at any of the three temperatures tested, and CPE was not found after the fourth passage. The RT-PCR analysis resulted in weak amplification (positive) from the first lysate, but no amplification was found in lysates of four passage cells. Had TSV replicated (productive infection) in either of the two cell lines, RT-PCR would have shown a strong amplification from each lysate. Such a weak amplification may have been the result of residual extracellular viruses that remained in the cell culture flask after washing. However, after first passage and repeated washing with HBSS, any remnants of the original medium were not likely to have been present. Therefore, our result showed that TSV was incapable of infecting Hep-2 and Vero cell lines.

Generally, aquatic viruses replicate in cells of aquatic animals at 20°C–35°C, their natural environmental temperature. We incubated cultures as noted earlier, as we did not know which temperature was most conducive for viral replication; all attempts were unsuccessful. Hep-2