

8. Lutumba P, Robays J, Miaka mia Bilenge C, Mesu VK, Molisho D, Declercq J, et al. Trypanosomiasis control, Democratic Republic of Congo, 1993–2003. *Emerg Infect Dis.* 2005;11:1382–8.

Address for correspondence: François Chappuis, Médecins sans Frontières, rue de Lausanne 78, 1202 Geneva, Switzerland; email: francois.chappuis@hcuge.ch

Using Museum Collections to Detect Pathogens

To the Editor: Natural history museum collections have evolved in recent years to meet the challenges of current and future interdisciplinary scientific studies. Many natural history museums have built tissue collections and made digital information (e.g., photographs, publications, geographic coordinates) freely available on the Internet. These collections provide endless opportunities to conduct studies, including temporal and spatial surveys of emerging and reemerging pathogens (1). We report an example of a museum collection being useful in detecting *Trypanosoma cruzi*, the etiologic agent of Chagas disease, in the southern plains woodrat (*Neotoma micropus*) in southern Texas. This finding is of interest in the epidemiology of Chagas disease because the climatic characteristics and demographics of the region are similar to areas in Latin America where Chagas disease is an important zoonotic agent that infects ≈20 million persons (2).

Tissue samples from *N. micropus* woodrats archived in the Natural Science Research Laboratory at the Museum of Texas Tech University were evaluated for *T. cruzi* DNA by PCR methods. All samples were originally collected during March 2001–June

2003 from the Chaparral Wildlife Management Area in southern Texas (28°18'N, 99°24'W), 86 km west of the Mexico–US border; some samples had been used previously in other research projects (3). Individual rodents were captured with live traps (n = 13) or by excavating middens in which all the nest occupants were collected by hand (n = 146). Animals were later euthanized and tissue samples (heart, kidney, liver, lung, muscle, spleen) were obtained. Tissues were immediately frozen in liquid nitrogen and permanently stored in ultralow-temperature freezers. We extracted 1 DNA sample from each animal's liver for use in this survey. DNA amplification was performed by using primers specific to *T. cruzi* (TCZ1 and TCZ2) (4) under previously standardized conditions and positive controls (5). *T. cruzi* DNA was detected in 42 (26.4%) of 159 woodrat samples tested. Males were infected significantly more often (31/82) than females (11/73); sex was not determined for 4 individuals (Score test for a binomial proportion, $z = -4.0$, $p < 0.01$). Adults had a nonsignificant higher prevalence (24/92) than all other individuals in the remaining age categories combined (14/54) (age was not determined for 13 individuals) (Score test for a binomial proportion, $z = -0.02$, $p = 0.98$). Middens that harbored infected individuals (n = 28, mean = 1.8) were not significantly ($t = 0.79$, $df = 84$, $p = 0.43$) more populated than middens that harbored uninfected individuals (n = 58, mean = 1.6).

Woodrats had been shown by using microscopy to be infected by *T. cruzi* and *T. cruzi*-like organisms (6); however, no definitive DNA-based confirmation had been performed (6,7). The results of this research confirm the infection of *N. micropus* woodrats with *T. cruzi* and show a higher prevalence than that reported in previous studies that used other diagnostic methods. These results also point to woodrats as a potentially important reservoir of *T. cruzi* in North America. We hy-

pothesize that the high prevalence is a consequence of the nest-building habits of these rodents. These nests are complexes of dry branches, grasses, and leaves, with a mean diameter of 84 cm, and offer easy access and permanent refuge to triatomine bugs. Woodrats have been found in association with at least 5 triatomine species: *Triatoma gerstaeckeri*, *T. lecticularia*, *T. neotomae*, *T. protracta*, and *T. sanguisuga* (8). Another factor for consideration is woodrats' multigenerational midden use, which may enable the permanent occurrence of triatomine colonies and therefore maintain long-term circulation of *T. cruzi*. Whereas recent characterizations of North American strains have included isolates from other mammalian reservoir hosts (9), the genotyping of parasites from *N. micropus* woodrats and other woodrats is still to be done.

Despite successful results from tracking pathogens by using material deposited in natural history museum collections (10), this practice is not common. We suggest that natural history museum collections be used more frequently, especially for surveying and genotyping *T. cruzi* in mammals, because of the importance of such information in clarifying the epidemiology and the evolutionary history of this pathogen.

Acknowledgments

Nisha Garg, Jian-Jun Wen, and Carlos Machado graciously supplied positive controls. Robert Baker, Heath Garner, and Kathy MacDonald provided access to DNA samples. Special thanks to Lisa Longhofer, Brian Amman, Darin Carroll, Michelle Haynie, Nevin Durish, Mary Lou Milazzo, and Ciro Milazzo for collecting specimens. Thanks to David Synatske and Donald Ruthven for access to the study area.

Specimens were collected by using funds provided by a National Institutes of Health grant (AI-41435, "Ecology of emerging arenaviruses in the southwest-

ern United States”) to C.F.F. and R.D.B.; funding for laboratory work was provided by Texas Tech University Association of Biologists to C.M.P.

**C. Miguel Pinto,
B. Dnate' Baxter,
J. Delton Hanson,
Francisca M. Méndez-
Harclerode, John R. Suchecki,
Mario J. Grijalva,
Charles F. Fulhorst,
and Robert D. Bradley**

Author affiliations: American Museum of Natural History and City University of New York, New York, New York, USA (C.M. Pinto); Texas Tech University, Lubbock, Texas, USA (C.M. Pinto, B.D. Baxter, J.D. Hanson, F.M. Méndez-Harclerode, J.R. Suchecki, R.D. Bradley); Pontificia Universidad Católica del Ecuador, Quito, Ecuador (C.M. Pinto, M.J. Grijalva); Ohio University, Athens, Ohio, USA (M.J. Grijalva); and University of Texas Medical Branch, Galveston, Texas, USA (C.F. Fulhorst).

DOI: 10.3201/eid1602.090998

References

1. Suarez AV, Tsutsui ND. The value of museum collections for research and society. *Bioscience*. 2004;54:66–74. DOI: 10.1641/0006-3568(2004)054[0066:TVOMCF]2.0.CO;2
2. Hanford EJ, Zhan FB, Lu Y, Giordano A. Chagas disease in Texas: recognizing the significance and implications of evidence in the literature. *Soc Sci Med*. 2007;65:60–79. DOI: 10.1016/j.socscimed.2007.02.041
3. Baxter BD, Méndez-Harclerode F, Fulhorst CF, Bradley RD. Genetics of social behavior: a molecular examination of relatedness, multiple maternity, and cohabitation of the southern plains woodrat (*Neotoma micropus*). *Journal of Mammalogy*. 2009. 90:819–831.
4. Moser DR, Kirchhoff LV, Donelson JE. Detection of *Trypanosoma cruzi* by DNA amplification using the polymerase chain reaction. *J Clin Microbiol*. 1989;27:1477–82.
5. Virreira M, Torrico F, Truyens C, Alonso-Vega C, Solano M, Carlier Y, et al. Comparison of polymerase chain reaction methods for reliable and easy detection of congenital *Trypanosoma cruzi* infections. *Am J Trop Med Hyg*. 2003;68:574–82.
6. Eads RB, Hightower BG. Blood parasites of southwest Texas rodents. *J Parasitol*. 1952;38:89–90. DOI: 10.2307/3274189
7. Burkholder JE, Allison TC, Kelly VP. *Trypanosoma cruzi* (Chagas) (Protozoa: Kinetoplastida) in invertebrate, reservoir, and human hosts of the lower Rio Grande valley of Texas. *J Parasitol*. 1980;66:305–11. DOI: 10.2307/3280824
8. Lent H, Wygodzinsky P. Revision of the Triatominae (Hemiptera, Reduviidae), and their significance as vectors of Chagas' disease. *Bulletin of the American Museum of Natural History*. 1979;163:123–520.
9. Roellig DM, Brown EL, Barnabé C, Tibayrenc M, Steurer FJ, Yabsley MJ. Molecular typing of *Trypanosoma cruzi* isolates, United States. *Emerg Infect Dis*. 2008;14:1123–5. DOI: 10.3201/eid1407.080175
10. Yates TL, Mills JN, Parmenter CA, Ksiazek TG, Parmenter RR, Vande Castle JR, et al. The ecology and evolutionary history of an emergent disease: Hantavirus pulmonary syndrome. *Bioscience*. 2002;52:989–98. DOI: 10.1641/0006-3568(2002)052[0989:TEAHO]2.0.CO;2

Address for correspondence: C. Miguel Pinto, Department of Mammalogy and Sackler Institute for Comparative Genomics, American Museum of Natural History, New York, NY 10024, USA; email: mpinto@amnh.org

Aggression and Rabid Coyotes, Massachusetts, USA

To the Editor: In 1959, coyotes (*Canis latrans*) were found in only 3 Massachusetts towns, but by 2007, their population was estimated at 10,000 and they were present throughout the state, except on the islands of Martha's Vineyard and Nantucket (1). The coyote is highly adaptable and readily tolerates living near humans (2). Because the raccoon rabies virus (RRV) variant is endemic to Massachusetts and spillover into the coyote population occurs (3), coyotes are a

potential source of rabies exposure for humans. Rabies in coyotes has emerged in Massachusetts at the same time that coyote and human populations have increased. From 1985 through 2008, the Massachusetts Department of Public Health tested coyotes by following the standard direct fluorescent antibody testing protocol published by the Centers for Disease Control and Prevention (4).

Of the 111 coyotes submitted for rabies testing, 4 (3.6%) were unsatisfactory because of decomposed brain tissue. Of the remaining 107 coyotes, 10 (9.0%) were found to be rabid; strain typing confirmed all 10 to have had spillover RRV. Within each county, the time between the first identification of RRV in an animal and finding a rabid coyote within that county ranged from 558 to 4,857 days; median was 2,799 days. The long time before spillover from raccoon to coyote was detected suggests that coyotes might avoid rabid reservoir animals. The time lag may also be the result of the distinct ecologic niches of these animals; coyotes are the top predators in ecosystems, and raccoons are only 1 of several mesocarnivores.

The public health rabies surveillance system in the United States is passive and relies on interaction of humans or domestic animals with rabies vector species (5). Because a rabid wild animal would go untested if a human or domestic animal had not had potentially infectious contact with it, the 10 coyotes with confirmed rabies likely represented only some portion of all rabid coyotes in Massachusetts during the study period.

Among 97 nonrabid coyotes, 7 had reportedly been in contact with humans and domestic animals. Among the 10 rabid coyotes, 4 were reported to have been in contact with humans and domestic animals. The coyotes in contact with both were 8.6× more likely to be rabid than were those in contact with only 1 or the other ($p < 0.05$).

