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References

1. Martini GA. Marburg agent disease: in man. Trans R Soc Trop Med Hyg. 1969;63:295–302. DOI: 10.1016/0035-9203(69)90001-7
2. Hopp M. Marburg hemorrhagic fever—Uganda. ProMed. August 3, 2007 [cited 2009 Oct 28]. http://www.promedmail.org, archive no. 20070803.2514.
3. Swanepoel R, Smit SB, Rollin PE, Formenty P, Leman PA, Kemp A, et al.; International Scientific and Technical Committee for Marburg Hemorrhagic Fever Control in the Democratic Republic of Congo. Studies of reservoir hosts for Marburg virus. Emerg Infect Dis. 2007;13:1847–51.
4. Timen A, Koopmans M. Marburg hemorrhagic fever—the Netherlands ex Uganda. ProMed. July 11, 2008 [cited 2009 Oct 28]. http://www.promedmail.org, archive no. 20080711.2115.
5. Towner JS, Pourrut X, Albariño CG, Nkogue CN, Bird BH, Grard G, et al. Marburg virus infection detected in a common African bat. PLoS One. 2007;2:e764. DOI: 10.1371/journal.pone.0000764
6. Towner JS, Amman BR, Sealy TK, Reeder Carroll SA, Comer JA, Kemp A, et al. Isolation of genetically diverse Marburg viruses from Egyptian fruit bats. PLoS Pathog. 2009;5:e1000536. DOI: 10.1371/journal.ppat.1000536
7. Johnson ED, Johnson BK, Silverstein D, Tukei P, Geisbert TW, Sanchez AN, et al. Characterization of a new Marburg virus isolated from a 1987 fatal case in Kenya. Arch Virol Suppl. 1996;11:101–14.
8. Smith DH, Johnson BK, Isaacson M, Swanepoel R, Johnson KM, Killey M, et al. Marburg-virus disease in Kenya. Lancet. 1982;1:816–20. DOI: 10.1016/S0140-6736(82)91871-2
9. Kuzmin IV, Niezgoda M, Franka R, Agwanda B, Markotter W, Beagley JC, et al. Lagos bat virus in Kenya. J Clin Microbiol. 2008;46:1451–61. DOI: 10.1128/JCM.00016-08
10. Centers for Disease Control and Prevention. 2008: Marburg hemorrhagic fever, imported case—United States [cited 2009 Feb 3]. http://www.cdc.gov/ncidod/dvrd/spb/outbreaks/index.htm#USA

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Human African Trypanosomiasis in Areas without Surveillance

To the Editor: Human African trypanosomiasis (HAT), sleeping sickness, is a systemic protozoan disease transmitted by the bite of a tsetse fly; untreated infection is fatal (*1*). Control of HAT caused by *Trypanosoma brucei gambiense*, which caused 97% of all cases reported from 1997 through 2006 (*2*), is based on active screening of the population at risk by mobile teams and treatment of all infected persons, with or without vector control.

The epidemiologic curve of reported new cases varies considerably; incidence peaks were high in the 1920s and 1990s but low in the 1960s and in the past decade (2000–2009) (*2–4*). The recent reduction of reported cases (69% decrease from 1997 through 2006) was made possible by 1) cessation of large-scale civil wars (e.g., in Angola); 2) increased commitment of donors, national control programs, the World Health Organization (WHO), and nongovernment organizations; and 3) free production and supply of antitrypanosomal drugs. In May 2007, after a WHO informal consultation on sustainable sleeping sickness control, representatives from countries to which HAT is endemic concluded that HAT elimination is possible (*5*). Médecins sans Frontières (MSF), an international nongovernment organization, wishes to challenge this conclusion.

Because of insufficient coverage by surveillance systems, only a fraction of HAT cases are reported. In 2004, for example, although WHO received reports of only 17,500 new cases, they estimated that the actual incidence was 50,000–70,000 cases (*6*). Recent MSF HAT projects in remote and politically unstable areas of the Central African Republic and the Democratic Republic of Congo are finding new information about the location and nature of some of these blind spots (areas without surveillance) (Table).

In the zones de santé (administrative districts) of Doruma, Ango, and Bili, in northeastern Democratic Republic of Congo, no HAT control activities have taken place over the past 3 decades, mainly because of extreme remoteness of these areas. In July 2007, MSF launched a HAT control program and found high (3.4%) disease prevalence and a large proportion of patients in the first stage of the disease (60%), indicating intense transmission. In March 2009, the MSF team was attacked by rebels from the Lord's Resistance Army, leading to total suspension of the project for an indefinite period. The lack of trained staff in existing health structures and the complexity of HAT management prevented emergency handover of the project to local partners.

Table. Human African trypanosomiasis control programs run by Médecins sans Frontières in central Africa, January 2007–May 2009*

Location (program dates)	No. persons screened			No. (%) new diagnoses			Prevalence, %†
	Total	Passive	Active	Total	First stage	Second stage	
Zones de santé of Doruma, Ango and Bili, DRC (2007 Jul–2009 Mar)	46,601	18,559	28,042	1,570	947 (60)	623 (40)	3.4
Maitikoulou and neighboring villages, CAR and Chad (2009 Jan–2009 May)	4,633	286	4,347	665	450 (68)	215 (32)	14.4

*DRC, Democratic Republic of Congo; CAR, Central African Republic.
†Total no. patients with parasitologically confirmed human African trypanosomiasis (total no. new diagnoses) divided by total no. persons screened during the period of first-round active screening (total no. persons screened).

Batangafo and Maitikoulou, in northwestern Central African Republic, are also historical foci of HAT that have been neglected over the past years, mainly because of political insecurity and logistic constraints. From January 2007 through April 2009, HAT was diagnosed for 1,074 patients in Batangafo (prevalence 3.1%). From January through May 2009, MSF screening of 4,633 persons from 23 villages around Maitikoulou in Central African Republic and Chad found high disease prevalence and a high proportion of first-stage illness.

Because the case-finding activities in the Central African Republic and Democratic Republic of Congo were restricted for security reasons, the MSF programs may only be seeing a small part of the problem. The above-listed examples illustrate that many HAT patients are still found in historical foci that have been devoid of active surveillance for years or decades because of their remoteness, insecurity, neglect, or a combination of these factors. Thus, many more patients probably continue to have no access to care and therefore remain invisible.

We emphasize the crucial need to continue research and development efforts toward simpler diagnostic methods and treatment. The effect of inadequate tools is particularly obvious in remote or unstable areas of high disease prevalence, where health facilities are often poorly functioning and severely understaffed. The recent completion of a study showing excellent safety and efficacy of a simpler treatment for second-stage patients is an encouraging first step (*7*).

The remoteness of many HAT-endemic areas, persistence of forgotten conflicts, and insufficient resources continue to restrict the possibility of eliminating HAT, except in countries or regions where the disease is already well controlled or where control programs cover all disease-endemic foci. As long as most HAT patients continue to have no access to care and are therefore not reported, HAT cannot be eliminated. Moreover, countrywide statistics should be interpreted with caution. The decline of new cases observed in the Democratic Republic of Congo from 1998 through 2003 is largely the result of efforts of 1 nongovernment organization in 1 province (Equateur-Nord), while the incidence trends remained stable in other provinces (*8*).

Donors must be aware that HAT epidemiology is heterogeneous. The allocation of funds should not be restricted to maintaining surveillance and control efforts in areas of low disease prevalence (to prevent future flare-ups). Adequate funding must also be provided to allow control programs to reach remote disease-endemic foci that have been left without active surveillance for years.

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References

1. Brun R, Blum J, Chappuis F, Burri C. Human African trypanosomiasis. Lancet. 2009 Oct 13; [Epub ahead of print].
2. Simarro PP, Jannin J, Cattand P. Eliminating human African trypanosomiasis: where do we stand and what comes next? PLoS Med. 2008;5:e55. DOI: 10.1371/journal.pmed.0050055
3. Barrett MP. The fall and rise of sleeping sickness. Lancet. 1999;353:1113–4. DOI: 10.1016/S0140-6736(98)00416-4
4. Barrett MP. The rise and fall of sleeping sickness. Lancet. 2006;367:1377–8. DOI: 10.1016/S0140-6736(06)68591-7
5. World Health Organization. Report of a WHO informal consultation on sustainable control of human African trypanosomiasis. Geneva: The Organization; 2007.
6. World Health Organization. Human African trypanosomiasis (sleeping sickness): epidemiological update. Wkly Epidemiol Rec. 2006;81:71–80.
7. Priotto G, Kasparian S, Mutombo W, Ngouama D, Ghorashian S, Arnold U, et al. Nifurtimox–eflornithine combination therapy for second-stage African *Trypanosoma brucei gambiense* trypanosomiasis: a multicenter, randomized, phase III, noninferiority trial. Lancet. 2009;374:56–64. DOI: 10.1016/S0140-6736(09)61117-X

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.

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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 hypothesize 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.

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