

Several infection risk factors exist simultaneously in this situation. In a CL-endemic area, immigrant populations, who are mostly nonimmune, exert pressure on the environment (deforestation) that directly increases their risk for exposure to infected vectors, in the absence of prophylactic measures. The initial short-term effect of deforestation is the mobilization of aggressive adult sandflies, which have been disturbed while resting. However, the ability of zoophilic vectors to adapt to peridomestic environments has also already greatly influenced the distribution of leishmaniasis in South America (5–7).

Considering the uncertainty of the population estimate, turnover, and immunity status, we assume that incidence rates should be considered cautiously. Nevertheless, we found that gold mining in forested areas constitutes a risk factor for CL, at least in French Guiana and probably in all Amazonian rainforests. This risk could be a public health concern. Larger studies in other gold-mining areas are required to quantify the incidence of CL among workers to effectively focus prophylactic and preventive campaigns.

Acknowledgments

This work was supported by the University of the French West Indies and the French Guiana (Cayenne, French Guiana), by the Contrat Plan État-Région no. 2365, and by the Institut National de la Santé et de la Recherche Médicale (INSERM, Paris, France).

**Brice Rotureau,* Michel Joubert,†
Emmanuel Clyti,† Félix Djossou,†
and Bernard Carme***

*Université des Antilles et de la Guyane, Cayenne, French Guiana; and †Centre Hospitalier Andrée Rosemon, Cayenne, French Guiana

References

1. Rotureau B, Ravel C, Nacher M, Couppie P, Curtet I, Dedet JP, et al. Molecular epidemiology of *Leishmania (Viannia) guyanensis* in French Guiana. *J Clin Microbiol*. 2006;44:468–73.
2. Dedet JP. Cutaneous leishmaniasis in French Guiana: a review. *Am J Trop Med Hyg*. 1990;43:25–8.
3. Carme B, Aznar C, Pradinaud R. Absence of a proven resurgence of Chagas disease or cutaneous leishmaniasis in French Guiana over the last two decades. *Ann Trop Med Parasitol*. 2001;95:623–5.
4. Rotureau B, Ravel C, Couppie P, Pralong F, Nacher M, Dedet JP, et al. Use of PCR-restriction fragment length polymorphism analysis to identify the main New World *Leishmania* species and analyze their taxonomic properties and polymorphism by application of the assay to clinical samples. *J Clin Microbiol*. 2006;44:459–67.
5. Rotureau B. Ecology of the *Leishmania* species in the Guianan ecoregion complex. *Am J Trop Med Hyg*. 2006;74:81–96.
6. Walsh JF, Molyneux DH, Birley MH. Deforestation: effects on vector-borne disease. *Parasitology*. 1993;106(Suppl):S55–75.
7. Lainson R, Shaw JJ, Silveira FT, de Souza AA, Braga RR, Ishikawa EA. The dermal leishmaniasis of Brazil, with special reference to the eco-epidemiology of the disease in Amazonia. *Mem Inst Oswaldo Cruz*. 1994;89:435–43.

Address for correspondence: Bernard Carme, Laboratoire Hospitalo-Universitaire de Parasitologie et Mycologie Médicale, Equipe EA 3593, Unité de Formation et de Recherche en Médecine de l'Université des Antilles et de la Guyane, Campus Saint-Denis, BP 718, 97336 Cayenne, French Guiana; email: ufrmedag2@wanadoo.fr

Mycobacterium tuberculosis Drug Resistance, Ghana

To the Editor: The directly observed treatment strategy (DOTS) for tuberculosis (TB) treatment has been implemented in Ghana since 1994. Before then, TB was treated

without adherence to any concerted guidelines. The 2003 report of the Ghanaian National Tuberculosis Programme (NTP) stated a TB incidence of 281/100,000 (1). NTP ensures treatment of all patients with an 8-month course of streptomycin, isoniazid, rifampin, and pyrazinamide (for 2 months), followed by thiacetazone and isoniazid (6 months). The cure rate for 2003 was >50% (1), and >75% is anticipated for 2005.

To determine the extent of drug resistance and to make suggestions for future Ghanaian NTP strategies, we assessed resistance against anti-TB drugs used in Ghana. A total of 2,064 patients with new cases of pulmonary TB were recruited at Korle Bu Teaching Hospital, Accra; Komfo Anokye Teaching Hospital, Kumasi; 15 periurban hospitals; and hospitals in the Ashanti, Eastern, and Central Regions of Ghana. These patients were consecutively enrolled in a cross-sectional study from September 2001 to December 2004. On all patients' clinical examinations, chest radiographs, sputum smears for staining of acid-fast bacteria, HIV testing, and culturing of *Mycobacterium tuberculosis* complex strains were performed. Samples were taken only after informed consent was given. The study was approved by the appropriate ethics committees.

A total of 2,064 *Mycobacterium* isolates were cultured at the Kumasi Centre for Collaborative Research. After decontamination of sputum samples (N-acetyl-L-cysteine/NaOH) and centrifugation, sediments were transferred onto Lowenstein-Jensen (LJ) media, incubated (37°C), and read weekly for 10 weeks for mycobacterial growth. Subsequently, cultures were sent to the German National Reference Centre for Mycobacteria in Borstel, Germany, a reference laboratory of the World Health Organization, for drug sensitivity testing (DST; proportion method on LJ media). Sensitivity to isoniazid, rifampin, pyrazinamide,

ethambutol, and streptomycin was determined for 2,064 isolates and to thiacetazone for 1,288 isolates. For ambiguous results and DST of thiacetazone, the modified proportion method (Bactec 460TB; Becton Dickinson, Cockeysville, MD, USA) was performed. Data were analyzed with EpiInfo (Centers for Disease Control and Prevention, Atlanta, GA, USA) and Fourth Dimension (ACI Group, San Jose, CA, USA) software programs.

Of the isolates, 32.8% were from female patients, and 67.8% were from male patients. The mean age of participants (33 years, range 10–60) did not differ by sex. HIV prevalence was 14.3% (males, $n = 179$, females, $n = 117$).

A total of 1,578 (76.5%) isolates were susceptible to all drugs tested, whereas 304 (14.7%) were monodrug resistant, and 177 (8.7%) were multi- or polydrug resistant to combinations (multidrug resistance meant resistance to at least isoniazid and rifampin (2.2%); polydrug resistance meant resistance to several drugs, excluding combined resistance to isoniazid and rifampin (6.5%). The overall prevalence of any drug resistance was 23.5% (486 isolates) (Table). No differences were observed between HIV-negative and HIV-positive patients. The highest level of resistance was against streptomycin, followed by isoniazid. Resistance to rifampin, pyrazinamide, and thiacetazone was lower. Monoresistance to ethambutol was not observed; resistance to ethambutol combined with other drugs occurred in 0.9% of isolates.

In all, 6.5% of isolates were polydrug resistant and virtually always included resistance to isoniazid. Among isolates with double- and triple drug resistance, combinations of resistance to isoniazid and streptomycin and to isoniazid-thiacetazone-streptomycin occurred most frequently. Other combinations were relatively rare.

Table. Resistance to first-line antituberculosis drugs, Ghana*

	Isolates from HIV-negative patients, n (%)	Isolates from HIV-positive patients, n (%)
Resistance	1,768† (85.7)	296† (14.3)
Any resistance	415 (23.4)	71 (24.0)
Monoresistance	255 (14.3)	49 (16.6)
H only	74 (4.2)	15 (5.1)
R only	12 (0.7)	4 (1.4)
S only	160 (9.0)	25 (8.4)
Z only	7 (<0.5)	5 (1.7)
T only	2 (<0.5)	–
E only	–	–
HR resistance (MDR)	39 (2.2)	4 (1.4)
HR	3 (<0.5)	1 (<0.5)
HRE	1 (<0.5)	–
HRS	11 (0.6)	2 (0.7)
HRZ	1 (<0.5)	–
HRES	6 (<0.5)	–
HREZ	1 (<0.5)	–
HRTS	3 (<0.5)	–
HRZS	4 (<0.5)	1 (<0.5)
HRETS	4 (<0.5)	–
HRESZ	5 (<0.5)	–
H + other resistance	116 (6.6)	15 (0.5)
HE	1 (<0.5)	–
HT	5 (<0.5)	–
HS	88 (5.0)	11 (3.7)
HES	–	1 (<0.5)
HTS	15 (0.8)	3 (1.0)
HSZ	6 (<0.5)	1 (<0.5)
HTSZ	1 (<0.5)	–
R + other resistance	1 (<0.5)	1 (<0.5)
RS only	1 (<0.5)	1 (<0.5)
Any drug resistance		
Any H	232 (13.1)	36 (12.2)
Any R	54 (3.1)	10 (3.4)
Any S	306 (17.3)	45 (15.2)
Any Z	26 (1.5)	7 (2.4)
Any T	33 (3.0)	4 (2.2)
Any E	18 (1.0)	1 (<0.5)

*H, isoniazid; R, rifampin; S, streptomycin; Z, pyrazinamide; T, thiacetazone; E, ethambutol; MDR, multidrug-resistance.

†Resistance to T tested in only 1,108 isolates and 180 isolates from HIV-negative and HIV-positive persons, respectively.

In 1989, an initial drug resistance rate of 54.5% in pulmonary TB was observed in Ghana (2); 27% were resistant to isoniazid, 23% to streptomycin, 29% to thiacetazone, 16% to streptomycin-isoniazid, and 5% to thiacetazone-streptomycin-isoniazid. A later study reported a high prevalence of primary drug resistance to isoniazid (23%), while sensitivity to rifampicin, pyrazinamide, ethambutol, streptomycin, and ciprofloxacin was maintained (3). However, the number of isolates tested was fewer in both stud-

ies ($n = 99$ and 25 , respectively) than in ours. This report supplements data from patients in Ghana whose conditions were newly diagnosed as HIV-negative and HIV-positive. Samples were collected in 2 large regions of Ghana, the Greater Accra and the Ashanti Regions, and were supplemented by samples from additional regions. Thus, these results are likely representative of the entire country.

The overall primary drug resistance rate of 23.5% in Ghanaian TB patients ranks Ghana among those

African countries with a high prevalence of drug-resistant TB. The high degree of mono-, multi- and polyresistance to streptomycin may be the result of selective pressure exerted by treatment of other infections with streptomycin and to incomplete treatment courses. Drug resistance to streptomycin and isoniazid are of concern, since these drugs are core components of the NTP. The relative ineffectiveness of streptomycin and the low level of resistance to ethambutol justify the most recent replacement of streptomycin by ethambutol by the Ghanaian NTP.

Low rates of initial drug resistance have been reported in countries in which the DOTS strategy has been successfully implemented. Adequate use of standardized treatment regimens under DOTS will limit further emergence of drug resistance but not substantially reduce the current degree of resistance (4). Although the levels of drug resistance in Africa are lower than in several other countries (5), measures to provide controlled application of second-line drugs, supervision of drug distribution and compliance, enforcement of DOTS protocols, and sustained training of all personnel involved in TB management are crucial.

The authors received a grant from the German Ministry of Education and Research within the frame of the National Genome Research Network.

**Ellis Owusu-Dabo,* Ohene Adjei,*
Christian G. Meyer,†
Rolf D. Horstmann,†
Anthony Enimil,‡
Thomas F. Kruppa,§ Frank Bonsu,¶
Edmund N.L. Browne,*
Margaret Amanua Chinbuah,#
Ivy Osei,# John Gyapong,#
Christof Berberich,*
Tanja Kubica,** Stefan Niemann,**
and Sabine Ruesch-Gerdes****

*Kwame Nkrumah University of Science and Technology, Kumasi, Ghana; †Bernhard-Nocht-Institute for Tropical Medicine, Hamburg, Germany; ‡Komfo Anokye Teaching Hospital, Kumasi, Ghana; §Kumasi Centre for Collaborative Research in Tropical Medicine, Kumasi, Ghana; ¶National Tuberculosis Programme, Accra, Ghana; #Ministry of Health, Accra, Ghana; and **National Reference Centre for Mycobacteria, Borstel, Germany

References

1. National Tuberculosis Control Programme. Annual report 2003. Accra (Ghana): The Programme; 2003.
2. van der Werf TS, Groothuis DG, van Klingeren B. High initial drug resistance in pulmonary tuberculosis in Ghana. *Tubercle*. 1989;70:249–55.
3. Lawn SD, Frimpong EH, Al-Ghusein H, Acheampong JW, Uttley AH, Butcher PD, et al. Pulmonary tuberculosis in Kumasi, Ghana: presentation, drug resistance, molecular epidemiology and outcome of treatment. *West Afr J Med*. 2001;20:92–7.
4. Farmer P, Bayona J, Becerra M, Furin J, Henry C, Hiatt H, et al. The dilemma of MDR-TB in the global era. *Int J Tuberc Lung Dis*. 1998;2:869–76.
5. World Health Organization. Anti-tuberculosis drug resistance in the world. Third global report. WHO/HTM/TB/2004.323. Geneva: The Organization; 2004.

Address for correspondence: Christian G. Meyer, Department of Molecular Medicine, Bernhard-Nocht-Institute for Tropical Medicine, Bernhard, Nocht Str 74, 20359 Hamburg, Germany; email: c.g.meyer@bni.uni-hamburg.de

Avian Influenza Risk Communication, Thailand

To the Editor: Twenty-two human cases of H5N1 highly pathogenic avian influenza (HPAI) have been reported in Thailand since 2003, with 14 deaths (1). From July to December 2005, I investigated Thai consumers' food safety practices by conducting an oral survey prepared in the Thai language. Interviews were conducted in 3 areas that have not had cases of H5N1 avian influenza, Bangkok (urban, n = 126), Rangsit (suburban, n = 125), and Phetchabun (rural, n = 50). Of the 301 Thai consumers surveyed, 92% thought that Thailand has ≥ 1 food safety problems, such as pesticide residues (62%), poor personal hygiene of food vendors (39%), and microbiologic/viral contamination of food (26%). Although the Thai Ministry of Public Health has conducted an aggressive public education campaign regarding HPAI (2), only 6% named bird flu as their primary concern. Most participants had some knowledge of avian influenza; 88% of participants knew the name of the disease, and of those, all knew that infections can be deadly, and 97% knew that interacting with and slaughtering infected birds are the most risky activities.

In the rural area, 72% of participants had backyard chickens (almost no one had them in urban and suburban areas). Of those, only 6% were aware of the symptoms of HPAI in poultry. Most villagers knew that minimizing contact with birds could reduce their risk for infection; however, they were not sure how they could minimize contact. None of the owners of backyard chickens had tested them for HPAI. The reporting system for HPAI was not easily accessible for home poultry producers.

The findings of this study are similar to those of Olsen et al., who

