and no reports of disease transmission were received from any other recipients of organs from this donor. In addition, the patient had no history of receiving infusions of holistic or alternative medicines.

The organism was initially detected in the aerobic Bact/Alert blood culture system (bioMérieux, Inc., Durham, NC) after 72 h incubation at 35°C. Presumptive identification of the pleomorphic gram-positive bacillus as *Streptomyces* sp. was based on phenotypic characterization by using standard conventional tests and cellular fatty acid analysis. Species identification was determined by DNA sequencing of the 16S rRNA gene. DNA sequencing reactions were performed with the Tag Dye Deoxy Terminator Cycle Sequencing Kit (Applied Biosystems, Inc., Foster City, CA), and data were generated with an ABI 377 automated instrument. The sequence data were assembled, edited, and compared with published sequences for the 16S rRNA gene of *S. bikiniensis* (2).

The genus *Streptomyces* belongs to the order *Actinomycetales*, which includes *Mycobacterium*, *Nocardia*, and *Actinomyces*. *Streptomyces* are gram-positive, extensively branched, filamentous bacteria that form aerial hyphae with chains of spores. Their natural habitat is soil, and each species has a defined geographic distribution. None are common in the United States. With the exception of specimens from actinomycotic mycetoma, the isolation of *Streptomyces* from clinical specimens frequently is considered laboratory contamination (3). Rare cases of clinical disease attributed to *Streptomyces* have been published, including bloodstream infection (1,4) and focal invasive infections (5–9). *Streptomyces* was not the only potential pathogen isolated from some of the clinical specimens in these studies.

Scant data are available on effective treatment of *Streptomyces* infection. Mycetoma caused by *Streptomyces* is often treated with penicillin, sulfonamides, or tetracycline; however, the cure rate is low. The recommended duration of therapy is lengthy (up to 10 months). Isolates of *S. griseus* referred to the Centers for Disease Control and Prevention were frequently resistant to ampicillin (80%), sulfamethoxazole (43%), cotrimoxazole (29%), and ciprofloxacin (57%) (10). Resistance to doxycycline (19%) and minocycline (10%) was lower. Vancomycin susceptibility was not tested. Resistance patterns must be interpreted cautiously because *Streptomyces* can synthesize antibiotics, potentially confounding results of in-vitro susceptibility testing.

The patient described in this report had no signs or symptoms of infection. The transient fever that prompted the first blood culture was probably due to the methotrexate infusion and not infection with *S. bikiniensis*. That the fever was of short duration despite persistently positive blood cultures supports this conclusion. The potential for causing minimal symptoms may contribute to assignment of *Streptomyces* as a contaminant. Clinical correlation is difficult if the infection is silent. *Streptomyces* isolated from blood cultures should not be dismissed as contaminants without careful consideration of the clinical situation; the isolation of *Streptomyces* from repeat blood cultures strongly suggests a pathogenic role.

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Drug-Resistant *Mycobacterium tuberculosis* among New Tuberculosis Patients, Yangon, Myanmar

To the Editor: Spread of drug-resistant tuberculosis (TB) and disastrous rates of HIV-TB co-infection pose serious threats to TB-control programs around the world (1). The World Health Organization/International Union Against Tuberculosis and Lung Diseases urges all national TB programs to practice the Directly Observed Treatment-Short Course (DOTS) strategy as well as to closely monitor the patterns and trends of anti-TB drug resistance (2). Such data allow an assessment of the quality of TB control, help forecast future trends

of drug-resistance, and serve as guidelines for suitable therapy.

In 1997 the national TB programs of Myanmar introduced DOTS in the capital city, Yangon, which has approximately 5 million inhabitants. All new case-patients in national TB program clinics are routinely treated with isoniazid, rifampicin, ethambutol, and pyrazinamide without drug susceptibility testing of *Mycobacterium tuberculosis.* However, isolates from previously treated patients are frequently tested for drug susceptibility, and treatment is guided by the results. Myanmar is one of the 22 countries that account for 80% of the world's new TB cases (3), yet little is known about drug-resistant TB in that country. We report on the pattern of drug resistance to first-line anti-TB drugs among *M. tuberculosis* complex isolates from Zone 1 TB center in Yangon, which receives approximately 70% of the national TB programs' TB cases in Yangon. Of the 864 patients who attended this center in July 2000, a total of 202 were diagnosed as having pulmonary TB on the basis of medical history, clinical signs, two smear-positive sputum samples, and chest x-ray, if necessary. Approximately half of these cases were new pulmonary TB patients, i.e. smear-positive patients who had never been treated previously. Sputum specimens from 72 consecutive, new pulmonary TB case-patients were injected on Ogawa medium according to standard procedure (2); samples from 68 patients (94%) were culture-positive. Isolates from 17 patients were lost for further study because of bacterial contamination and failure to grow on subculture. Thus, isolates from 51 patients were available for the current investigation. By using the AccuProbe *Mycobacterium tuberculosis* complex test (Gen-Probe, San Diego, CA), all isolates were found to belong to the *M. tuberculosis* complex. Testing of isolates for susceptibility to isoniazid, rifampicin, ethambutol, and streptomycin was performed by using the standard Mycobacteria Growth Indicator Tube manual system, as recommended by the manufacturer (Becton Dickinson, Sparks, MD). The Wayne assay (4), which measures the activity of pyrazinamidase, was used for pyrazinamide susceptibility testing. This assay was performed according to World Health Organization guidelines for speciation within the *M. tuberculosis* complex (5). Eighteen isolates (35%) were resistant to any one of the five anti-TB drugs. Thirteen isolates (26%) were resistant to isoniazid, nine isolates (18%) to streptomycin, four isolates (8%) to ethambutol, one isolate (2%) to rifampicin, and one isolate (2%) to pyrazinamide. Only one isolate (2.0%) was multidrug resistant (MDR)-*M. tuberculosis*, i.e., resistant to both isoniazid and rifampicin.

The World Health Organization/ International Union Against Tuberculosis and Lung Diseases global survey in the year 2000 (6) showed that the prevalence of resistance to at least one anti-TB drug (isoniazid, rifampicin, ethambutol, and streptomycin) among new cases ranged from 1.7% to 36.9%. In our study, 33.3% of the isolates from new pulmonary TB patients were resistant to at least one of these drugs. The finding shows that a relatively high frequency of drug resistance exists among our patients. If pyrazinamide is included in the calculation, the proportion of drug resistance among our patients is 35.3%. In 1994, Ti et al. reported that MDR-TB represented 1.25% of the isolates from 400 patients with newly diagnosed pulmonary TB who attended the Zone 1 TB center (7). When one considers the corresponding figure of 2.0% in the current material, frequency of MDR-TB in Yangon does not seem to have changed dramatically during the period 1994–2000. MDR-TB among new patients appears to be less common in Yangon than in big cities in Thailand (4.2%) (8) and in China (4.5%) (6). However, a substantial number of our isolates (15.7%) were resistant to two or more anti-TB drugs, in most cases to both isoniazid and streptomycin (9.8%). In the 1994 report by Ti et al., mono-resistance to streptomycin (6.5%) or isoniazid (5.8%) predominated, and 2.0% of the isolates were resistant to both isoniazid and streptomycin (7). Our present results, therefore, indicate that drug resistance is an imminent threat to TB-control efforts in Yangon, although MDR-TB still seems to be relatively rare.

The low number of MDR cases in our study could partly be explained by demographic features of the studied population, which is composed predominately of people residing in satellite townships of Yangon. These townships usually attract young people who immigrate to Yangon from village areas. These immigrants are less likely to have previous exposure to TB than the permanent population since the prevalence of TB infection is lower in rural than in urban areas (9). Moreover, population densities of the satellite townships are 2- to 10-fold lower than in inner Yangon city (Myanmar Central Statistical Organization). The high number of drug-resistance cases among our patients with newly detected TB could be explained by an undisclosed past exposure to anti-TB drugs. The case detection rate reported by the Myanmar national TB programs is 48% (3), suggesting that many TB patients receive their treatment elsewhere. A World Health Organization report (10) indicates that >80% of the health-care expenditure in Myanmar and other Asian countries such as India, Vietnam, and Cambodia is spent in the private sector. In such countries, poor treatment practices in the private sector may lessen the impact of the DOTS implemented by national TB programs and contribute to a growing incidence of drug-resistant TB. This problem will undoubtedly be escalated by the availability of anti-TB drugs without prescription. HIV-TB coinfection often results in increased frequency of adverse drug effects, which may reduce compliance and increase induction of drug resistance. Although

the prevalence of HIV positivity among our patients is unknown, a preliminary study from Yangon shows that the prevalence of drug-resistant TB among HIV-seropositive and -seronegative patients is the same (pers. comm., Myanmar national TB programs]. To our knowledge, this report is the first to describe drug-resistant patterns in *M. tuberculosis* isolates from Myanmar.

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Pneumocystis carinii vs. *Pneumocystis jiroveci*: Another Misnomer (Response to Stringer et al.)

To the Editor: The proposal by Stringer et al. to change the name of *Pneumocystis carinii* found in humans to *Pneumocystis jiroveci* requires critical consideration (1). First, their rationale for the choice of Jírovec is not compelling. Principle III of the International Code of Botanical Nomenclature (ICBN) states: "the nomenclature of a taxonomic group is based upon priority of publication" (2). Jírovec's publication in 1952 was not the first to report *P. carinii* infection in human lungs. In 1942, two Dutch investigators, van der Meer and Brug, described *P. carinii* as the infecting organism in a 3-month-old infant with congenital heart disease and in 2 of 104 autopsy cases (a 4-month-old infant and a 21-year-old adult) (3). Their description, photomicrographs, and drawings of *P. carinii* are unequivocal. They also described the typical "honeycomb" patterns in alveoli. In 1951, Dr. Josef Vanek at Karls-Universität in Praha, Czechoslovakia, reported his study of lung sections from 16 children with interstitial pneumonia and demonstrated that the disease was caused by *P. carinii* (4). Vanek notes in his report, "In man the parasite was for the first time established as a cause of pneumonia in a child by G. Meer and S. L. Brug (1942)." In 1952, Jírovec reported *P. carinii* as the cause of interstitial plasmacellular pneumonia in neonates (5). A year later, in a coauthored publication, Vanek, Jírovec, and J. Lukes acknowledged and referenced the earlier reports of van der Meer and Brug and Vanek (6). If principle III is to be followed, as well as fairness to the investigators, both van der Meer and Brug and Vanek hold priority over Jírovec, assuming the designation of the species name should be based on the name of the first person to discover *P. carinii* in humans.

The nomenclature of *P. carinii* has actually been fraught with errors from the beginning. In the earliest publications, Carlos Chagas and Antonio Carini mistook the organism for stages in the life cycle of trypanosomes. Chagas placed it in a new genus, *Schizotrypanum* (7,8). In 1912, Delanoë and Delanoë at the Pasteur Institute in Paris published the first description of the organism as a new entity unrelated to trypanosomes (9). They proposed the name *"Pneumocystis carinii"* as a tribute to Carini. The Delanoë paper has remained unchallenged as the original description of *P. carinii*. Both Chagas and Carini later acknowledged their errors and the validity of the Delanoës' conclusion. By current ICBN principles, *P. carinii* is acceptable nomenclature because the authors of the first publication proposed the name of Carini, rather than their own.

In addition, changing the name to *P. jiroveci* will create confusion in clinical medicine where the name *P. carinii* has served physicians and microbiologists well for over half a