A 43-year-old woman of Mayan origin from Quintana Roo, Mexico, was diagnosed with diffuse lepromatous leprosy. The etiologic bacillus was determined to be *Mycobacterium lepromatosis* instead of *Mycobacterium leprae*. This case likely represents the first report of this leprosy form and its agent in the southeastern tip of Mexico.
matched completely with *M. lepromatosis*. Therefore, this species was the etiologic agent of this infection.

How the patient contracted the infection was uncertain. She had no known contacts with other leprosy patients. She had lived all her life, with no travel more than 50 km away from home, in Quintana Roo, one of the three states in the Yucatan Peninsula, the southeastern tip of Mexico, where DLL had been unknown previously. Although she related that her villagers had armadillo in their diet, she had never eaten or cooked this animal. It is also noteworthy that there are no published or known studies of armadillo in the Yucatan Peninsula to show that this animal carries a leprosy agent. One study noted leprosy-like infection in an armadillo caught near Mexico City (5).

Leprosy (Hansen’s disease) is likely the oldest human infection that can be traced to its African origin with humans (6–8) and possibly much earlier to the hominid era millions of years ago (9). *M. leprae* has been known to be the sole leprosy agent since its

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**FIG 1** Histopathology of diffuse lepromatous leprosy in a 43-year-old woman. Shown are dense histiocytic infiltration in the dermis at low and high magnifications (A, 20×; B, 400×) and neuritis (C, 400×). All panels, hematoxylin and eosin.

**FIG 2** Acid-fast bacilli in the nerve (A) and endothelia (B). Fite stain, 1,000×.
The study confirmed and differentiated the mycobacteria in analysis of 120 Mexican leprosy cases using archived biopsy tissue lepromatosis cases came from nine western and central Mexico also 42 cases of other clinical forms of leprosy. In the study, the M. lepromatosis alone caused 18 cases, and both species together caused 14 cases. 87 ulceration in the late stage, known as Lucio’s phenomenon (20–22). On histopathology, DLL shows the usual acid-fast bacillary infiltration of the skin and nerves, panniculitis, vasculitis, and in the late stage, unique endothelial proliferation and vascular occlusion (20, 21, 23). DLL has been endemic in western and central Mexico (20, 22) and Costa Rica (24) but rarely reported elsewhere. The present case also echoes a similar recent report (26), earlier clinical experience of Rea and Jerskey with DLL (21), and the steady decline in leprosy incidence in Mexico in recent decades (25). Thus, the standard multidrug regimen for multibacillary leprosy likely works for M. lepromatosis infection.

Recently, leprosy-like dermatitides of animals have been described in cats in Australia (29), red squirrels in Scotland (30), and cows in France (31). Thus far, the etiologic acid-fast bacilli have not been cultivated, similar to the difficulty in cultivation of M. leprae and M. lepromatosis. Limited genetic studies of the organisms in cats and squirrels have indicated similarities to the leprosy bacilli (29, 30). The study of the cow agent analyzed portions of 6 genes totaling 3,231 nucleotides (31). Judged from the GenBank deposits (KJ095004 to KJ095009), the five protein-coding genes matched 88% to 93% those of M. leprae and/or M. lepromatosis, and the 16S rRNA gene—the most conserved bacterial gene—matched best with M. lepromatosis (98.4% [361 of 367 bp]). These results thus raise the likelihood of a new Mycobacterium species. Whether the cow agent contains pseudogenes—the hallmark of the leprosy bacilli—is yet to be seen.

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FIG 3 Detection of the 16S rRNA gene of Mycobacterium lepromatosis by heminested differential PCRs. Lane 1, 100-bp DNA size marker; lane 2, 159-bp amplicon from the common leprosy primers; lane 3, 142-bp specific amplicon for M. lepromatosis; lane 4, lack of the 135-bp amplicon for M. leprae."

initial discovery in 1873 (10). In 2008, a novel Mycobacterium species named M. lepromatosis was recognized as the cause of death of two Mexican patients with DLL (11). Further phylogegetic studies of 20 genes and pseudogenes revealed a 9.1% genetic difference between the two leprosy bacilli (11). This large difference contrasts with the clonal nature of worldwide M. leprae strains (6, 8, 12). It also hints at the ancient divergence of the two bacilli, ~10 million years ago, from their last common ancestor (9, 13). Most recently, genomes of two M. lepromatosis strains were sequenced, revealing an ~13% genome-wide difference from M. leprae but with similar genome sizes and organizations between the species (14, 15). Analysis of the one of the draft genomes also refined the divergence time to 13.9 million years (14).

Independent studies have corroborated this new cause of leprosy. Vera-Cabrera et al. (16, 17) reported several cases of infection from Nuevo Leon, Mexico. Jessamine and colleagues (18) reported infection of a native Canadian man who manifested polynuropathy and skin rashes but had no significant history of contact or travel to areas of endemicity. DLL is a unique, severe form of leprosy initially recognized by Lucio and Alvarado in 1852 (19) and further described by Latapi and Chevez-Zamora in 1948 (20), both in Mexico. It is thus also called Lucio’s leprosy. This form shows a diffuse cutaneous infiltration, with no nodule or plaque formation, and frequent skin ulceration in the late stage, known as Lucio’s phenomenon (20–22). On histopathology, DLL shows the usual acid-fast bacillary infiltration of the skin and nerves, panniculitis, vasculitis, and in the late stage, unique endothelial proliferation and vascular occlusion (20, 21, 23). DLL has been endemic in western and central Mexico (20, 22) and Costa Rica (24) but rarely reported elsewhere.

Realization of two leprosy bacilli led us to conduct an etiologic analysis of 120 Mexican leprosy cases using archived biopsy tissue (1, 2). The study confirmed and differentiated the mycobacteria in 87 cases. Of these, M. lepromatosis alone caused 55 cases, M. leprae alone caused 18 cases, and both species together caused 14 cases. M. lepromatosis caused not only all 13 DLL cases specifically but also 42 cases of other clinical forms of leprosy. In the study, the M. lepromatosis cases came from nine western and central Mexico states, which matched the historical areas where areas of DLL is endemic (20, 25). Among other states in Mexico, studies (16, 17, 26) have noted M. lepromatosis infection in Tamaulipas, Nuevo Leon, and Coahuila in the northeast, which border Texas in the United States. The present case from a native of Quintana Roo further adds the far southeastern tip of Mexico to the list. Thus far, 13 of the 30 Mexico States were known to have M. lepromatosis leprosy. Recently, a likely family transmission of this agent was noted that involved a pair of Mexican siblings (A. F. Marsh and C. Hill, 2015 unpublished data).

In addition to Mexico and Canada, M. lepromatosis has been identified in Brazil (4), Singapore (3), and Myanmar (4). In these studies, the organism caused fatal DLL and other forms of leprosy, and dual infections with M. leprae were also seen. Thus, M. lepromatosis is a long-elusive second cause of leprosy with a wide trans-Pacific distribution. The long record of DLL and the likely dominance of M. lepromatosis in Mexico have led us to propose that the disease came with the first American settlers from Asia over 13,000 years ago (1). Finding M. lepromatosis in Myanmar (4) and in Singapore (3) supports this Asian origin. Finding it in Brazil (4) accords with further American spread from the North America to Central America, such as Costa Rica, where DLL has been endemic (24), and to South America, such as the Amazon region of Brazil. In the Brazilian Amazon, leprosy has been known for at least a century (27) and is still hyperendemic (28).

The present case also adds to our growing experience on the presentation, diagnosis, treatment, and follow-up of M. lepromatosis infection. The normal laboratory findings and the lack of skin ulcers—a usual feature of late-stage DLL—suggested mild infection. During treatment, the patient experienced mild erythema nodosum leprosum, a common reaction of leprosy that usually occurs during the early course of multidrug treatment. In two other featured cases of M. lepromatosis infection (26), erythema nodosum leprosum was a presenting sign along with high fever, lymphadenopathy, and skin rashes. The treatment success of the present case also echoes a similar recent report (26), earlier clinical experience of Rea and Jerskey with DLL (21), and the steady decline in leprosy incidence in Mexico in recent decades (25). Thus, the standard multidrug regimen for multibacillary leprosy likely works for M. lepromatosis infection.
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