

## etymologia

### *Cochliomyia hominivorax* [kok"le-o-mi'yə]

From the Greek *kochlias* (“snail with a spiral shell”) + *myia* (“fly”) and the Latin *hominis* (“man”) + *vorax* (“consuming”), *Cochliomyia hominivorax*, or the New World screwworm fly (formerly *Callitroga* [Greek *kallos*, “beautiful,” + *trogein*, “to gnaw”] *americana*), was first described by French entomologist Charles Coquerel in 1858. *C. hominivorax* larvae enter wounds and feed on living tissue, and if untreated, infestations can be fatal. *C. hominivorax* was eliminated in the United States in 1982 and in much of Central America in the 1990s, although outbreaks associated with reimportations in infected humans and animals continue to occur.



Figure. Dorsal view of the “Primary screwworm” fly, *Cochliomyia hominivorax*, a member of the family Calliphoridae. Image: Public Health Image Library.

#### Source

1. Dear JP. A revision of the New World chrysomyini (Diptera: Calliphoridae). *Revista Brasileira de Zoologica*. 1985;3:109–69. <http://dx.doi.org/10.1590/S0101-81751985000300001>

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## LETTER

### *Mycobacterium lepromatosis* Lepromatous Leprosy in US Citizen Who Traveled to Disease-Endemic Areas

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**To the Editor:** Virk et al. (1) reported a *Mycobacterium lepromatosis* infection in a US citizen with a history of multiple international travels and concluded that *M. lepromatosis* lepromatous leprosy is a travel-related hazard for travelers to endemic areas. The conclusions drawn, however, need extensive support of thoroughly

conducted case studies before generalizing *M. lepromatosis* as a travel-related hazard.

In the case report, the exact source of *M. lepromatosis* infection was unclear. Moreover, experimental evidence used in this work are not enough to prove that *M. lepromatosis* is a travel-related hazard. Confirming a source of infection by DNA fingerprinting of *M. lepromatosis* can be ideal to rule out infection from unreported native patients or environmental reservoirs (2).

It is possible that the patient in this report may have contracted *M. lepromatosis* infection as a result of his host-susceptible genetic factors. Host genetic susceptibility to leprosy is complicated because of the genetics of *M. lepromatosis*, interaction between genetic and environmental factors, gene–gene interactions, and ethnicity (3). Host genetics plays a major role in determining a person’s risk of developing clinical leprosy. Thus, even a short trip to a leprosy-endemic country is sufficient for a host susceptible to *M. lepromatosis* to acquire an infection. The host

immune response influences the course of leprosy infection; it is challenging to understand the genetics of disease susceptibility and immunopathogenesis of leprosy (4,5).

With an inference of only a single case study, it is hard to say that *M. lepromatosis* lepromatous leprosy is a travel-related hazard for all US citizens. More surveillance data, such as patients' immunity toward the disease, their genetic susceptibility, and travel history, are needed to explore the travel-related hazard. In addition, evolutionary knowledge and how widely the disease is circulating in nonendemic regions will help in understanding the nature of the disease.

### References

1. Virk A, Pritt B, Patel R, Uhl JR, Bezalel SA, Gibson LE, et al. *Mycobacterium lepromatosis* lepromatous leprosy in US citizen who traveled to disease-endemic areas. *Emerg Infect Dis*. 2017;23:1864–6. <http://dx.doi.org/10.3201/eid2311.171104>

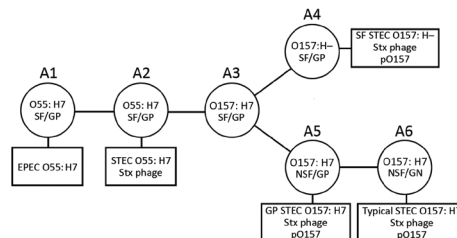
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4. Araújo S, Lobato J, Reis EM, Souza DOB, Gonçalves MA, Costa AV, et al. Unveiling healthy carriers and subclinical infections among household contacts of leprosy patients who play potential roles in the disease chain of transmission. *Mem Inst Oswaldo Cruz*. 2012;107(Suppl 1):55–9. <http://dx.doi.org/10.1590/S0074-02762012000900010>
5. Blake LA, West BC, Lary CH, Todd JR IV. Environmental nonhuman sources of leprosy. *Rev Infect Dis*. 1987;9:562–77. <http://dx.doi.org/10.1093/clindis/9.3.562>

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## Corrections

### Vol. 24, No. 12

In Figure 1 of the article Genomic Characterization of  $\beta$ -Glucuronidase-Positive *Escherichia coli* O157:H7 Producing Stx2a (Y. Ogura et al.), Shiga toxin-producing *Escherichia coli* O157 was mislabeled several times, and the term STEC was incompletely defined. The corrected figure is shown, and the article has been corrected online ([https://wwwnc.cdc.gov/eid/article/24/12/18-0404\\_article](https://wwwnc.cdc.gov/eid/article/24/12/18-0404_article)).



### Vol. 25, No. 1

Table 3 misstated the number of animals tested during 2001–2004 and the first author's biographical sketch was incorrect in Multiple Introductions of Domestic Cat Feline Leukemia Virus in Endangered Florida Panthers (E.S. Chiu et al.). The article has been corrected online ([https://wwwnc.cdc.gov/eid/article/25/1/18-1347\\_article](https://wwwnc.cdc.gov/eid/article/25/1/18-1347_article)).

Author Sang-Ho Choi's name was listed incorrectly and author affiliations were unclear in Clinical and Radiologic Characteristics of Human Metapneumovirus Infections in Adults, South Korea (H.J. Koo et al.). The article has been corrected online ([https://wwwnc.cdc.gov/eid/article/25/1/18-1131\\_article](https://wwwnc.cdc.gov/eid/article/25/1/18-1131_article)).

Two locations in Figure 1 were shown incorrectly in Risk Factors for *Elizabethkingia* Acquisition and Clinical Characteristics of Patients, South Korea (M.H. Choi et al.), and the article has been corrected online ([https://wwwnc.cdc.gov/eid/article/25/1/17-1985\\_article](https://wwwnc.cdc.gov/eid/article/25/1/17-1985_article)).

