Etymologia: Buruli Ulcer

Tony M. Korman, Paul D.R. Johnson, John Hayman

Author affiliations: Monash University, Melbourne, Victoria, Australia (T.M. Korman); Austin Health, Melbourne (P.D.R. Johnson); University of Melbourne, Melbourne (P.D.R. Johnson, J. Hayman)

DOI: https://doi.org/10.3201/eid2612.200744

To the Editor: The recent etymologia by Henry in the March 2020 issue of Emerging Infectious Diseases recounts the fascinating origin of the name Buruli ulcer (*1*). Further to the history, in 1948, pathologist Peter MacCallum first described the clinical features for 6 patients from Victoria, Australia, each with an ulcer with undermined edges on an arm or a leg, and the characteristic histopathologic findings, including extensive necrosis and abundant acid-fast bacilli without granuloma formation (*2*). Five of the patients were identified by general practitioners D.G. Alsop, L.E. Clay, and J.R. Searls from the city of Bairnsdale (thus, another eponym "Bairnsdale ulcer") (*3*). Glen Buckle and Jean Tolhurst at the Alfred Hospital in Melbourne established experimental animal infections, and eventually isolated the causative organism (*2*), which they later named *Mycobacterium ulcerans* (*4*). The growth of *M. ulcerans* required prolonged incubation at a temperature of 30°C–33°C (*2*), which was only realized after the inadvertent use of a faulty incubator.

In 1964, Clancey described a "new" mycobacterium causing chronic skin ulcers in Uganda that "resembled" *M. ulcerans* which he named "*Mycobacterium buruli*" (*5*). However, the causative organism of Buruli ulcer was subsequently recognized as *Mycobacterium ulcerans,* which had been originally described in Australia.

About the Author

Dr. Korman is an adjunct clinical professor at Monash University; Director, Monash Infectious Diseases; and Director of Microbiology, Monash Health, Clayton, Australia. He has a wide range of clinical, laboratory, and research interests.

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Address for correspondence: Tony M. Korman, Monash Infectious Diseases, Monash Health Centre, 246 Clayton Rd, Clayton, VIC 3168, Australia; email: tony.korman@monash.edu

Arthritis Caused by MRSA CC398 in Patient without Animal Contact, Japan

Anders R. Larsen, Jesper Larsen

Author affiliation: Statens Serum Institut, Copenhagen, Denmark

DOI: https://doi.org/10.3201/eid2612.202780

To the Editor: In their recent article, Nakaminami et al. describe a case of human infection caused by Panton-Valentine leucocidin (PVL)–positive livestock-associated methicillin-resistant *Staphylococcus aureus* clonal complex 398 (MRSA CC398) in Japan (*1*). *S. aureus* CC398 includes 2 major MRSA variants with distinct genetic and epidemiologic properties, a highly transmissible and virulent human variant comprising both PVL-positive and PVL-negative strains and a more benign PVL-negative livestock-associated variant (*2*). We have previously shown that, in Denmark, nearly all case-patients colonized or infected with PVL-positive MRSA CC398 strains of the human variant have links to countries in mainland Asia, where the strain is endemic in the community (*3*). Our analysis revealed the existence of 2 phylogenetically distinct lineages (L1 and L2) with unique sequence types (STs), ST398 linked to China and ST1232 linked to Vietnam, Thailand, and Cambodia. Besides being PVL-positive and belonging to ST1232, the isolate described by Nakaminami et al. (*1*) also shared other genetic and phenotypic characteristics with the L2 strains: it carried *spa* type t034 and SCC*mec* type V and was resistant to aminoglycosides (gentamicin), lincosamides (clindamycin),

macrolides (clarithromycin), and tetracyclines (tetracycline). We therefore suspect that the isolate belongs to the human variant of MRSA CC398.

In recent years, Denmark has witnessed increased importation of PVL-positive MRSA CC398 from mainland Asia because of international travel, in 1 case leading to a large hospital outbreak among mothers and infants in a maternity ward (*3*), and it seems possible that Japan and other countries might face a similar risk in the near future. Strain identification, source attribution, and knowledge about the transmission dynamics are essential for maintaining an effective MRSA infection control and prevention program. We therefore advocate using genotypic methods (e.g., as described by Stegger et al. [*4*]) that can accurately distinguish the human variant of MRSA CC398 from the livestock-associated variant.

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Address for correspondence: Jesper Larsen, Department of Bacteria, Parasites, and Fungi, Statens Serum Institut, Artillerivej 5, DK-2300 Copenhagen S, Denmark; e-mail: jrl@ssi.dk

Hidemasa Nakaminami

Author affiliation: Tokyo University of Pharmacy and Life Sciences, Tokyo, Japan

DOI: https://doi.org/10.3201/eid2612.203738

In Response: In our article (*1*), we hypothesized that the transmission route of the Panton-Valentine leukocidin (PVL)–positive sequence type (ST) 1232 (CC398) MRSA strain is not only from humans but also from imported edible meat for humans. However, in their letter, Larsen and Larsen (2) indicated that *S. aureus* CC398 includes 2 major MRSA variants with distinct genetic and epidemiologic properties; 1 being a highly transmissible and virulent human variant comprising both PVL-positive and PVL-negative strains, and the other being a more benign PVL-negative livestock-associated variant (3). The presence of PVL genes and immune evasion cluster (IEC) genes in CC398 strain provides supportive evidence for the association of human colonization or infections. Furthermore, they showed that most case-patients in Denmark who were colonized or infected with PVL-positive MRSA CC398 strains of the human variant have links to countries in mainland Asia (4).

Actually, we confirmed *scn*, *chp*, and *sak* of the IEC genes in the PVL-positive ST1232 strain. Hence, as Larsen and Larsen suggested, the ST1232 strain might be a human variant of CC398. We recently reported a second case of the ST1232 strain with characteristics similar to the previous patient in Japan (*5*). The data strongly suggest that the incidence of human variant of CC398 has been increasing in Japan. Therefore, I agree with their opinion that accurate discrimination of the human variant of MRSA CC398 from the livestock-associated variant is essential for maintaining effective MRSA infection control. I presume that detection of PVL and IEC genes might be a useful simplified marker for classification of the human variant of CC398.

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Address for correspondence: Hidemasa Nakaminami, Department of Microbiology, School of Pharmacy, Tokyo University of Pharmacy and Life Sciences, 1432-1 Horinouchi, Hachioji, Tokyo 192-0392, Japan; email: nakami@toyaku.ac.jp