CLSI clinical breakpoints for Gram-positive bacteria. *Int J Antimicrob Agents*. 2012;40(4):313-322.

19. Stoakes L, Reyes R, Daniel J, et al. Prospective comparison of a new chromogenic medium, MRSASelect, to CHROMagar MRSA and mannitol-salt medium supplemented with oxacillin or cefoxitin for detection of methicillin-resistant *Staphylococcus aureus*. *J Clin Microbiol*. 2006;44(2):637-639.

20. Telang NV, Satpute MG, Niphadkar KB, Joshi SG. An increased incidence of biofilm-producing multidrug-resistant methicillin-resistant *Staphylococcus aureus* in a tertiary care hospital from India: a 2-year study. *Am J Infect Control*. 2010;38(2):165-166.

21. Joshi SG, Paff M, Friedman G, Fridman G, Fridman A, Brooks AD. Control of methicillinresistant *Staphylococcus aureus* in planktonic form and biofilms: a biocidal efficacy study of nonthermal dielectric-barrier discharge plasma. *Am J Infect Control*. 2010;38(4):293-301.

22. Chaieb K, Zmantar T, Souiden Y, Mahdouani K, Bakhrouf A. XTT assay for evaluating the effect of alcohols, hydrogen peroxide and benzalkonium chloride on biofilm formation of *Staphylococcus epidermidis. Microb Pathog.* 2011;50(1):1-5.

23. Los R, Sawicki R, Juda M, et al. A comparative analysis of phenotypic and genotypic methods for the determination of the biofilm-forming abilities of *Staphylococcus epidermidis. FEMS Microbiol Lett.* 2010;310(2):97-103.

24. Cue D, Lei MG, Lee CY. Genetic regulation of the intercellular adhesion locus in staphylococci. *Front Cell Infect Microbiol*. 2012;2:38-51.

25. Gutiérrez D, Delgado S, Vázquez-Sánchez D, et al. Incidence of *Staphylococcus aureus* and analysis of bacterial-associated communities on food industry surfaces. *Appl Environ Microbiol*. 2012;78(24):8547-8554.

26. Ammendolia MG, Di Rosa R, Montanaro L, Arciola CR, Baldassarri L. Slime production and expression of the slime-associated antigen by staphylococcal clinical isolates. *J Clin Microbiol*. 1999;37(10):3235-3238.

27. Piessens V, De Vliegher S, Verbist B, et al. Characterization of coagulase-negative *Staphylococcus* species from cows' milk and environment based on *bap*, *icaA*, and *mecA* genes and phenotypic susceptibility to antimicrobials and teat dips. *J Dairy Sci.* 2012;95(12):7027-7038.

28. Sulzberger MB, Herrmann F, Zak FG. Studies of sweating; preliminary report with particular emphasis of a sweat retention syndrome. *J Invest Dermatol.* 1947;9(5):221-242.

29. Hölzle E, Kligman AM. The pathogenesis of miliaria rubra: role of the resident microflora. *Br J Dermatol*. 1978;99(2):117-137.

30. Knobloch JK, Bartscht K, Sabottke A, Rohde H, Feucht HH, Mack D. Biofilm formation by *Staphylococcus epidermidis* depends on functional RsbU, an activator of the sigB operon: differential activation mechanisms due to ethanol and salt stress. *J Bacteriol*. 2001;183(8):2624-2633.

31. Choi C, Hailu T, Cusack CA, Allen HB, Lodha S, Hailu T. The earliest immunologic finding in atopic dermatitis: periductal Toll-like receptor 2 expression in response to ductal occlusion by Staphylococcus epidermidis biofilm. J Am Acad Dermatol. 2012;66:AB71.

32. Kerstan A, Bröcker E-B, Trautmann A. Decisive role of tumor necrosis factor-a for spongiosis formation in acute eczematous dermatitis. *Arch Dermatol Res.* 2011;303(9):651-658.

33. Bibel DJ, Greenberg JH, Cook JL. Staphylococcus aureus and the microbial ecology of atopic dermatitis. *Can J Microbiol*. 1977;23(8):1062-1068.

34. Abramson JS, Dahl MV, Walsh G, Blumenthal MN, Douglas SD, Quie PG. Antistaphylococcal IgE in patients with atopic dermatitis. *J Am Acad Dermatol.* 1982;7(1):105-110.

35. Boguniewicz M. Atopic dermatitis: beyond the itch that rashes. *Immunol Allergy Clin North Am.* 2005;25(2):333-351, vii.

36. Ogawa T, Katsuoka K, Kawano K, Nishiyama S. Comparative study of staphylococcal flora on the skin surface of atopic dermatitis patients and healthy subjects. *J Dermatol.* 1994;21(7):453-460.

37. Nakata K, Inoue Y, Harada J, et al. A high incidence of *Staphylococcus aureus* colonization in the external eyes of patients with atopic dermatitis. *Ophthalmology*. 2000;107(12):2167-2171.

38. Katsuyama M, Ichikawa H, Ogawa S, Ikezawa Z. A novel method to control the balance of skin microflora, part 1: attack on biofilm of *Staphylococcus aureus* without antibiotics. *J Dermatol Sci.* 2005;38(3):197-205.

NOTABLE NOTES

Mór Cohen, Better Known as Moriz Kaposi

Filippo Pesapane, MD; Gianluca Nazzaro, MD; Antonella Coggi, MD; Raffaele Gianotti, MD

Today, Moriz Kaposi is remembered for the first description, in 1872, of the entity that bears his name,¹ but he was also one of the founders of the Viennese School of Dermatology.

Kaposi was born in 1837 in a poor Jewish family. The story of his name is curious: his first name is written "Moritz" in the records of the Jewish Community, but he almost always used "Moriz," and in some of his Hungarian publications he used "Moricz" and "Mór." The many versions of his first name simply reflected the multiple languages spoken by the educated classes in the Hapsburg monarchy. Originally his surname was Cohen, but, after his conversion to the Catholic faith, he changed it in 1871 to Kaposi, in reference to his birth town Kaposvár, in the Austro-Hungarian Empire. It is still debated why he changed his surname; it is unlikely to have been due to the pressures of anti-Semitism because Kaposi was not an opportunist, and at that time he was well established in his career. According to his own words, Mór Cohen changed his surname to avoid confusion with 5 other physicians named similarly in the Vienna School of Medicine.²

In 1886 Kaposi married Martha Hebra, daughter of Ferdinand Ritter von Hebra who was his mentor and with whom he authored the book *Textbook of Skin Diseases* in 1878. Kaposi's main work, however, was *Pathology and Therapy of the Skin Diseases in Lectures for Practical Physicians and Students*, which became one of the most significant books in the history of dermatology and was translated into many languages. Kaposi's remarkable skill with languages stood him in good stead; he was fluent in Hungarian, German, and French. In addition, he was versed in English and of course Latin, the official language of the Empire. 3

While Hebra is considered the "father of dermatology," Kaposi was one of the first to establish dermatology on its anatomical pathology scientific basis. In his field, Kaposi concerned himself chiefly with syphilis, its clinical presentation, its etiology, and treatment. He wrote with Hebra some of the early descriptions of cutaneous lupus erythematosus and noted the systemic involvement in 1872, and in 1875 he described the rash as "butterfly." Kaposi used his skills of observation and description to first report and delineate many other entities, such as xeroderma pigmentosum, diabetic and leukemic skin changes, syringoma, gangrenous zoster or eczema herpeticum, and pustulosis varioliformis acuta, which later became known as *Kaposi varicelliform eruption* or *eczema herpeticatum*.

Kaposi died peacefully in his sleep in Vienna at only 65 years of age.

Author Affiliations: Fondazione IRCCS Ca' Granda, Ospedale Maggiore Policlinico, University of Milan, Milan, Italy.

Corresponding Author: Filippo Pesapane, MD, Department of Medical-Surgical and Transplantation Physiopathology, "Fondazione IRCCS Ca' Granda, Ospedale Maggiore Policlinico," University of Milan, Milan, Italy (filippopesapane@gmail.com).

1. Kaposi M. Idiopathisches multiples Pigmentsarkomen der Hault. Arch Dermatol Syph. 1872;4:265-273.

- 2. Ingber A. Why Kaposi and not Kohn? Am J Dermatopathol. 1983;5:103.
- 3. Oriel JD. Moritz Kaposi (1837-1902). Int J STD AIDS. 1997;8(11):715-717.