

MRSA in Dermatology

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Methicillin-resistant *Staphylococcus aureus* (MRSA), first described in hospital settings (hospital-acquired or health care-associated MRSA; HA-MRSA), has garnered a tremendous amount of interest from both health-care workers and the general public since its discovery in 1961 (Barber, 1961). This interest stems from a number of factors, including the magnitude of the infections, the broader concern of antibiotic resistance, and media sensationalism of selected cases. Genetic resistance patterns as well as the initial locations of infection have recently shifted, with a trend toward affecting the nonhospitalized population. Community-associated MRSA (CA-MRSA) (Saravolatz *et al.*, 1982) is even more intriguing to lay and professional groups. CA-MRSA is distinguished from HA-MRSA by its more limited antibiotic-resistance profile, differences in the toxins it produces, the population susceptible to it, and its propensity for outbreaks (Naimi *et al.*, 2003). Of interest to dermatologists, CA-MRSA has a stronger predilection for skin involvement compared with HA-MRSA.

The emergence of MRSA in the community has led many physicians to wonder how prevalent these infections are and how they should be treated in the outpatient setting. These questions have been at least partly answered by the EMERGENCY ID Net Study Group, which reported that 59% of adult patients with acute purulent skin and soft-tissue infections who presented to their emergency rooms were infected with MRSA (Moran *et al.*, 2006). This study also demonstrated that the most effective treatment in this population was incision and drainage of the infection. Such studies provide tremendous insight into MRSA, yet they have also raised critical questions for practitioners in dermatology. In this issue, Jappe *et al.* analyze the frequency, antibiotic resistance, and molecular profiles of cutaneous infections from *S. aureus* in the outpatient dermatology clinic at the University of Heidelberg in Germany.

Over a 6-year period, the authors isolated *S. aureus* in 52% of their infected study population; 14% of these isolates were MRSA. Thus, only 7% of their total study population had MRSA. Additionally, via strict definitions as well as extensive genotyping and resistance profiling, Jappe *et al.* (2008) concluded that 22% of the total MRSA isolates were caused by CA-MRSA and the remainder due to HA-MRSA. These observations are intriguing because, contrary to many clinicians' prior impressions, MRSA infections were relatively uncommon in this study population, with CA-MRSA accounting for only 1.4% of the total isolates.

Through the following questions, we examine this paper in greater detail. For brief answers, please refer to <http://network.nature.com/group/jidclub>.

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QUESTIONS

1. What are the differences between CA-MRSA and HA-MRSA?
2. How does the study sample influence the study results?
3. How are *S. aureus* infections categorized?
4. What are the major findings of this study?
5. What may be the clinical implications of this article?
6. What further studies could be performed?

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