

## LETTERS TO THE EDITOR

## Failure of Decolonization in Patients With Infections Due to Mupirocin-Resistant Strains of Community-Associated Methicillin-Resistant *Staphylococcus aureus*

*To the Editor*—We read the recent article by Rahimian et al.<sup>1</sup> with great interest, and we applaud the authors for their efforts in addressing the role of treatment with mupirocin for recurrent methicillin-resistant *Staphylococcus aureus* (MRSA) skin and skin structure infections. We hypothesize that one potential reason for the high rate of recurrence of skin and skin structure infections observed by Rahimian et al.<sup>1</sup> in patients with MRSA nasal colonization treated with mupirocin (6 [32%] of 19) may be plasmid-mediated resistance to mupirocin. In a prior publication, Shastry et al.<sup>2</sup> demonstrated that there was a very high level of clindamycin resistance in their population of men who have sex with men (63 [63%] of 100).

We have demonstrated that clindamycin resistance and mupirocin resistance are both encoded on a single plasmid, pUSA03, that is frequently identified in multidrug-resistant strains of community-associated MRSA genotype USA300.<sup>3</sup> We have noted that the pUSA03-positive USA300 subclone is particularly prevalent as a cause of skin and skin structure infections in the population of men who have sex with men in San Francisco and Boston.<sup>4</sup> Most notably, this subclone was the pathogen in skin and skin structure infections in men who have sex with men who had no history of prior clindamycin or mupirocin use, suggesting person-to-person transmission of the multidrug-resistant USA300 clone.

With respect to the study by Rahimian et al.,<sup>1</sup> it would be of great interest to know (1) how many of the 19 patients treated with mupirocin had initial infecting and nasal colonizing strains resistant to both clindamycin and mupirocin (thus suggesting the presence of pUSA03) and (2) how many of these patients were men who have sex with men. Although the findings of Rahimian et al.<sup>1</sup> indicate that an attempt at decolonization with mupirocin may not be beneficial in preventing recurrent disease due to community-associated MRSA in their patient population, the effect of decolonization with mupirocin in a population with lower rates of clindamycin and mupirocin resistance in colonizing and/or infecting MRSA strains remains undetermined.

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## Reply to Graber and Schwartz

*To the Editor*—We appreciate the insightful comments by Graber and Schwartz<sup>1</sup> regarding our article on mupirocin treatment for recurrence of community-associated methicillin-resistant *Staphylococcus aureus* (CA-MRSA) skin and skin structure infections.<sup>2</sup> The high number of recurrences of colonization that we found in patients treated with mupirocin may indeed be unique to our study population. Among a subset of our study population (ie, 19 patients who had nasal MRSA colonization that was treated with mupirocin), 17 were men who have sex with men, a population that we previously found to have a high rate of colonization with clindamycin-resistant CA-MRSA strains.<sup>3</sup> Of the 19 colonized patients treated with mupirocin, 15 carried MRSA strains in their nares that were resistant to clindamycin (of note, all 15 of these patients were men who have sex with men).

Unfortunately, our laboratory did not test strains for mupirocin susceptibility. However, given that mupirocin resistance and clindamycin resistance are both encoded on the pUSA03 plasmid,<sup>4</sup> it is likely that a significant number of