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## USA300 MRSA: An Overview

A single clone of community-acquired MRSA appeared in the U.S. metropolitan areas about a decade ago and was subsequently designated as USA300 (3,6). Its subsequent appearance in metropolitan areas in Colombia has been linked to illicit drug use (1,2). The infected patients generally presented with complicated skin and soft tissue infections; disseminated infection with bacteremia was common.

The USA300 was found to be a single pulsed-field type (multilocus sequence type 8) that typically harbored Panton-Valentine leukocidin (PVL) toxin genes, the arginine catabolic mobile element (ACME), and Staphylococcal cassette chromosome *mec* (SCC*mec*) type IVa (6).

USA300 isolates have now emerged as a cause of health-care associated infections (5). Whereas traditional hospital-associated MRSA strains are usually resistant to multiple antimicrobial classes, USA300 isolates are typically susceptible to most classes of antimicrobial agents and are resistant only to oxacillin and erythromycin (4). Treatment options for MRSA skin and soft-tissue infections include trimethoprim-sulfamethoxazole (TMP-SMZ), clindamycin, and tetracyclines. Resistance to these other antistaphylococcal antibiotics has been sporadically reported in USA300 isolates (including fluoroquinolones, tetracycline, clindamycin, and mupirocin) (4,6).

Antimicrobial resistance in staphylococci typically emerges by acquisition of resistance determinants residing on plasmids and is often associated with transposons or insertion sequences. Fluoroquinolone resistance in *S. aureus* arises via chromosomal mutations in the DNA gyrase gene *gyrA*. Initially, reports of USA300 isolates with plasmid-mediated-resistance have been sporadic or restricted to specific geographical areas. However, USA300 isolates with expanded antimicrobial resistance patterns have been documented, including resistance to clindamycin, mupirocin (high level), gentamicin, trimethoprim (high level), and/or doxycycline. The resistance is plasmid-mediated and the infections are usually hospital-acquired (4).

In summary, USA300 appears inherently more virulent than other MRSA, has spread from the community into the hospital, and resistance to multiple antistaphylococcal agents is emerging within this clone.

## References

1. Arias CA, Rincon S, Chowdhury S, Martínez E, Coronell W, Reyes J, Nallapareddy SR, Murray BEMRSA USA300 clone and VREF--a U.S.-Colombian connection? *N Engl J Med.* 2008;359(20):2177-9.
2. Alvarez CA, Barrientes OJ, Leal AL, Contreras GA, Barrero L, Rincon S, et al. Community-associated methicillin-resistant *Staphylococcus aureus*, Colombia. *Emerg Infect Dis.* 2006; 12(12):2000-1.
3. Limbago B, Fosheim GE, Schoonover V, Crane CE, Nadle J, Petit S, et al. Characterization of methicillin-resistant *Staphylococcus aureus* isolates collected in 2005 and 2006 from patients with invasive disease: a population-based analysis. *J Clin Microbiol.* 2009;47(5):1344-51.
4. McDougal LK, Fosheim GE, Nicholson A, Bulens SN, Limbago BM, Shearer JE, et al. Emergence of resistance among USA300 methicillin-resistant *Staphylococcus aureus* isolates causing invasive disease in the United States. *Antimicrob Agents Chemother.* 2010;54(9):3804-11.
5. Seybold U, Kourbatova EV, Johnson JG, Halvosa SJ, Wang YF, King MD, et al. Emergence of community-associated methicillin-resistant *Staphylococcus aureus* USA300 genotype as a major cause of health care-associated blood stream infections. *Clin Infect Dis.* 2006;42(5):647-56.
6. Tenover FC, Goering RV. Methicillin-resistant *Staphylococcus aureus* strain USA300: origin and epidemiology. *J Antimicrob Chemother.* 2009;44:1-6. Epub 2009 Jul 16.