## **Community-Acquired MRSA (CA-MRSA)**

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Since we last discussed this topic in the NPHL newsletter (Spring of 2002), there have been many significant developments in the epidemiology, genetic background, and prevalence of CA-MRSA. At that time, CA-MRSA was mainly detected in select populations in the United States and was generally susceptible to non-b-lactam antibiotics. In addition, the main CA-MRSA genetic background or "strain" in 2002 was called USA400. However, since that time, many changes have occurred. First, the prevalence of USA400 has decreased significantly and a new CA-MRSA clone has emerged, which is called USA300. The USA300 and USA400 clones are named according to their pulsed-field gel electrophoresis pattern (**Figure 1**). The genome of USA300 has been recently



sequenced and it was found to be highly related to some common laboratory strains (e.g. COL-one of the first MRSA isolated in 1961 in London). However, USA300 also carries a gene that codes for Panton-Valentine Leukocidin (PVL; encoded by a bacteriophage) and a pathogenicity island (called the ACME island) that encodes several unique genes. It is currently unclear what role some of these proteins may have in virulence of CA-MRSA, however, PVL has recently been postulated to have a significant role in necrotizing pneumonia caused by USA300. In addition, USA300 is not only detected in the community but also is commonly isolated in hospital environments and is no longer universally susceptible to erythromycin and the fluoroquinolones.

Secondly, the prevalence of USA300 and CA-MRSA has increased significantly over the last few years. In a recent study from the New England Journal of Medicine (NEJM, 2006, 355:666-674. G. J. Moran et al.), 57% of all skin and soft tissue infections presenting to 11 university-affiliated emergency departments were CA-MRSA USA300. These data suggest that Emergency Room physicians should treat empirically for CA-MRSA with all skin and soft tissue infections. Concerns have been raised that this recent "emergence" of USA300 is an epidemic.

Lastly, based on the data reflecting the increased prevalence of USA300 and other MRSA, many hospitals are screening all new patients for colonization with MRSA. This decision directly impacts the clinical microbiology laboratories as new methodologies (DNA based) may need to be implemented to process the number of specimens that are received. In addition to the increased workload, some hospitals may ask their laboratories to decrease the turn around time on MRSA detection to less than a day (which rules out culture methodologies). Next year (summer of 2008), educational programs by the NPHL and the Nebraska Department of Health and Human Services will focus on issues surrounding MRSA; including those issues involving the clinical microbiology laboratory and infection control. For more information about CA-MRSA, please contact Dr. Fey at 402-559-2122 or pfey@unmc.edu.