Community-Acquired Methicillin-Resistant *Staphylococcus aureus* in Nebraska

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Methicillin-resistant *Staphylococcus aureus* (MRSA) is an ever-increasing hospital infection-control problem. Currently in the United States, approximately 50% of all *S. aureus* isolates from intensive care units are resistant to methicillin (or oxacillin). MRSA isolates are also typically resistant to multiple non-*B*-lactam anti-staphylococcal antibiotics. Therefore, vancomycin is typically administered to patients with MRSA infections. Even though the prevalence of MRSA in hospitals is high, staphylococcal infections that are acquired in the community setting are typically methicillin-susceptible. Recently, however, community-acquired MRSA (CA-MRSA) have been associated with several pediatric fatalities in the northern great plains region of the United States (1). Two recent epidemiological studies have demonstrated that the majority of CA-MRSA are generally susceptible to non-*B*-lactam antibiotics, found typically in skin and soft tissue infections, and appear highly-related as assessed by pulsed-field gel electrophoresis (PFGE) (2,3).

One of the questions surrounding the evolution of CA-MRSA is whether they are actually "escapees" from the hospital environment or whether the gene (*mecA*) that mediates methicillin-resistance has been recently acquired by a previously methicillin-susceptible *S. aureus* strain that is endemic in certain communities. The NPHL has begun to address this question by studying CA-MRSA isolated from Thurston County, Nebraska in 1998-1999. CA-MRSA isolates (33) were compared with 32 hospital-associated MRSA (HAMRSA) using antibiotic susceptibility testing, PFGE, superantigen production, and *spa* typing. As shown in **Figure 1** and by others, CA-MRSA are more susceptible to non-*B*-lactam antibiotics compared to HAMRSA.

Of note was the small percentage of resistance to erythromycin, clindamycin, and ciprofloxacin in CA-MRSA compared to HA-MRSA. Superantigen typing demonstrated that 32/33 CA -MRSA isolates produced either SEB or SEC toxin. Five isolates (15%) produced SEB while 26 (81%) produced SEC. No isolates produced Toxic Shock Syndrom Toxin (TSST-1). In contrast, no HA-MRSA isolates produced SEB, SEC, or TSST-1.

Both SEB and SEC have been implicated as the causative agents of non-menstrual TSST (nmTSST). As a further distinction, both PFGE and *spa* typing suggested that all CA-MRSA strains were highly-related yet distinct from HA-MRSA. The CAMRSA from Nebraska were also highlyrelated or identical to those strains from the northern great plains that caused pediatric fatalities. These data suggest that CAMRSA strains are a unique strain of MRSA, distinct from HA-MRSA, that are circulating in certain communities capable of causing serious disease. Recent work by Ma, etal. demonstrated that the *mec* element itself is distinct in CA-MRSA giving further credence to the notion that CAMRSA are actually previously methicillinsusceptible *S. aureus* strains that have recently acquired the *mecA* gene; and not "escapees" from the hospital environment (4).

Dr. Tom Safranek, State Epidemiologist, has asked for your help in determining the prevalence of CA-MRSA in Nebraska through the submission of erythromycin-susceptible MRSA isolates to the NPHL for further study. Currently, approximately 20% of these erythromycinsusceptible MRSA isolates have the "CAMRSA signature" as described. The NPHL appreciates your collaboration in this effort and would like to continue to receive erythromycin-susceptible MRSA isolates collected in your laboratory.

1. Centers for Disease Control and Prevention. 1999. Four pediatric deaths from community-acquired methicillinresistant *Staphylococcus aureus*--- Minnesota and North Dakota, 1997-1999. Morb. Mortal. Wkly. Rep. 48:707-710.

2. Groom AV, Wolsey DH, Naimi, TS, et al. 2001. Community-acquired methicillin -resistant *Staphylococcus aureus* in a rural American Indian Community. JAMA;286:1201-1205.

3. Naimi TS, LdDell KH, Boxrud DJ,

etal. 2001. Epidemiology and clonality of community-acquired methicillinresistant *Staphylococcus aureus* Minnesota, 1996-1998. Clin. Infect. Dis.;33:990-996.
4. Ma, X.X., Ito, T., Tiensasitorn, C. etal. 2002. Novel type of staphylococcal cassette chromosome *mec* identified in community-acquired methicillin - resistant *Staphylococcus aureus* strains. Antimicrob. Agents Chemother. 46:1147-1152.

Figure 1.