

Two Forms of MRSA Attack Different Populations

BY BETSY BATES

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SANTA BARBARA, CALIF. — Community-acquired methicillin-resistant *Staphylococcus aureus* is unlike its hospital-acquired cousin epidemiologically, clinically, and genetically, an infectious disease specialist explained at a dermatology meeting.

"It is a different organism," Dr. Paul Holtom said at the annual meeting of the California Society of Dermatology and Dermatologic Surgery.

For starters, the two forms of MRSA attack different populations.

Risk factors for hospital-acquired (HA) MRSA have not changed appreciably in the roughly 20 years since its emergence. The first risk factor, quite obviously, is hospitalization. Infections are most commonly seen in the intensive care unit, although they are known to spread rapidly throughout health care institutions.

Within the hospitalized population, risk factors include use of fluoroquinolones, enteral feeding, surgery, previous hospitalization, and extended lengths of stay.

For community-acquired (CA) MRSA, early lists of potential risk factors included intravenous drug use, homosexuality (men having sex with men), homelessness or marginal housing, and being in a correctional institution. Athletes with skin-to-skin contact were then added to the risk pool, but it soon became clear that the risk was widespread in the healthy population, extending to children as well as adults.

"The problem has moved far outside these original risk groups to almost everyone," said Dr. Holtom, director of the infectious disease clinic fellowship program and hospital epidemiologist at the Los Angeles County Hospital/University of Southern California Medical Center (LAC+USC Medical Center).

For example, the LAC+USC Medical Center and the University of California, Los Angeles, Medical Center—institutions that draw from profoundly disparate socioeconomic populations—both have CA-MRSA rates of greater than 70% in patients presenting to the emergency departments with skin and soft tissue infections.

Genetic testing reveals that CA-MRSA is indeed a distinct entity, rather than an HA-MRSA that has spread to the nonhospital world, Dr. Holtom said.

Hospital-acquired MRSA contains a special *mecA* gene that codes for penicillin-binding protein (PBP) 2a, which is an important binding site for penicillins and related antibiotic classes. Specifically, the staphylococcus cassette chromosome (SCC) *mec* types found in HA-MRSA are types I, II, and III, with type II dominating.

It's a different story for CA-MRSA, which is characterized by SCC*mec* types IV and sometimes V, which carry genes for a variety of toxins not seen in HA-MRSA, including the Panton-Valentine leukocidin toxin known for generating a prolific inflammatory response and skin necrosis.

"The one thing that has been very interesting about HA-MRSA for all of these years is that it does not spread in the community setting," he noted.

Dr. Holtom theorized that other or-

ganisms may have a competitive advantage in non-health care settings, or that HA-MRSA may spread too slowly in the immunocompetent population.

"Maybe it's because—unlike in the hospital where the patient is awash in an antibiotic-saturated environment—when [the patient goes] home there are no antibiotics that drive the growth of MRSA, or at least kill other organisms and allow [MRSA] to have a better environmental niche," he speculated. ■

MRSA Infections by Presenting Site

Characteristics	Hospital-Acquired MRSA (n = 937)	Community-Acquired MRSA (n = 131)
Skin/soft tissue	37%	75%
Respiratory tract	22%	6%
Urinary tract	20%	1%
Bloodstream	9%	4%
Otitis media/externa	1%	7%
Other	12%	8%

Source: JAMA 2003;290:2976-84

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