A report from the department of staph affairs

Evolution marches on, at least in the microbial world. On page 177 in this issue of the *Journal,* Dr. Susan Rehm reviews some of the clinical manifestations and therapeutic dilemmas associated with methicillin-resistant *Staphylococcus aureus* (MRSA) infection.

Once, we worried about penicillin-resistant staph, but we had methicillin and several other effective antibiotics. Then, in the 1960s, MRSA started to appear. Intravenous drug abusers and then the chronically ill were favored hosts. Some hospitals became nests for MRSA. It began to acquire a reputation as a particularly nasty invader, associated with necrotizing pneumonia and resistant endocarditis. By 2004 more than 60% of staph isolates from critical care units were resistant to methicillin. But we had vancomycin.

Now, the hospital bugs are being reinforced by their community brethren—new strains from the suburbs that carry toxins that can damage tissue via stimulation of apoptosis of host cells. Most community-associated MRSA species are still sensitive to vancomycin (as well as to trimethoprim-sulfamethoxazole and clindamycin). But not all are. A growing community of bugs is relatively resistant to vancomycin and carries toxins—microbial suicide bombers with body armor.

The clinical presentation of necrotizing cellulitis, first appearing as a "spider bite," is now seen in emergency wards around the country. In some cities, the overwhelming majority of deep skin infections evaluated in emergency rooms are due to MRSA. And patients with these infections, as well as asymptomatic nasal carriers of MRSA, are bringing these bugs into our hospitals.

The trends of emerging antibiotic resistance, discovery (and then perhaps overprescription) of new antibiotics, and changing patterns of staph infections are intimately intertwined. The evolutionary pressure that we humans are putting on *S aureus* with our antibiotics is taking this bug to a whole new place.

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