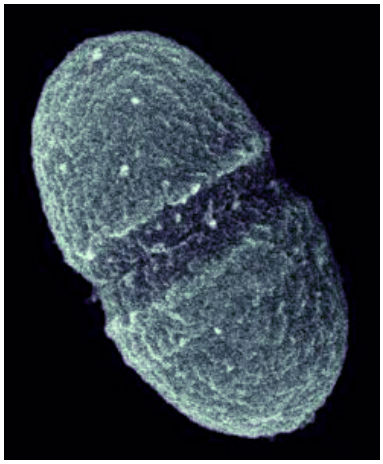

'Tis Better to Give Than to Receive. . .

The VRE Mantra

By *Barbara Fox, MS, MPH, MT(ASCP)*

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Currently she is a consultant and educator in Northern California.

As microbiologists, we need to be aware of , and promptly report the presence of confirmed VRE from hospitalized patient populations.

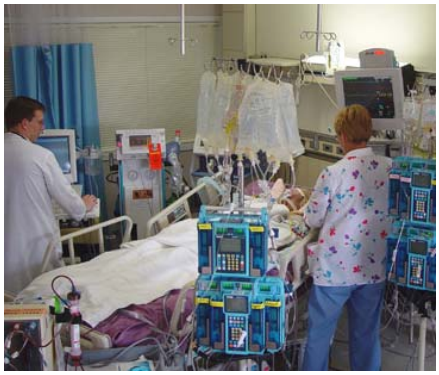
Why? All bacteria share genetic material through various methods that ensure survival of their species. However, Vancomycin Resistant Enterococcus (VRE) stands out as a particularly generous contributor of a not-so-nice gift: resistance to vancomycin, the commonly used glycopeptide antimicrobial.

While generally thought of as less problematic and less media worthy than Methicillin Resistant Staph aureus (MRSA), VRE has been shown to confer Vancomycin resistance to the already highly resistant MRSA and coagulase negative Staphylococci, thus limiting treatment options.

By 2009 the US had nine confirmed cases of VRSA (vancomycin resistant *S. aureus*), at least two of which were shown by molecular testing to have arisen from VRE transferring a key resistance gene (VanA) to MRSA. Other resistance genes of significance include VanB, and VanC. To date, only VanA and VanB are found in clinically relevant isolates, with VanC found in the low level intrinsically resistant enterococci: *E. gallinarum*, *E. casseliflavus*, and *E. flavencens*.

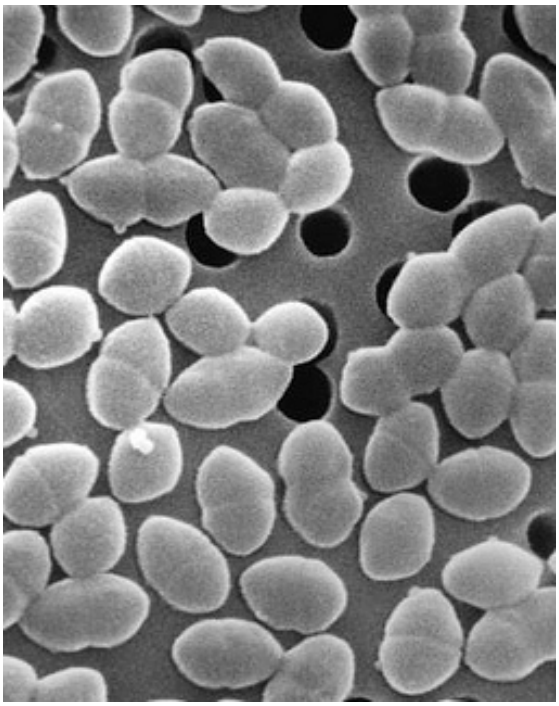
Originally, enterococci were thought to have developed vancomycin resistance due to widespread administration of vancomycin for MRSA and some *Clostridium difficile* infections; yet another example of bacteria in the GI tract sharing genetic information with its neighbors.

Vancomycin resistance was first reported in 1989, 30 years after the introduction of vancomycin. Vancomycin resistant *E. faecium* and *E. faecalis* isolates in the US increased from 0.3% to 7.9% between 1989 and 1993.



Today, one out of every three infections in the ICU is due to VRE, according to CDC data.

Originally, enterococci, facultatively anaerobic gram positive cocci or coccobacilli, were in the genus Streptococcus until 1984. Now deserving their own genus, the Enterococcus group includes a number of species found in both humans and animals. Human pathogens which account for over 95% of clinical isolates in the United States are *E. faecium*, the most common species in hospitalized populations, and *E. faecalis*. Of relatively low virulence, they are normal flora of the GI tract and female genital tract.



Infection with enterococci occur mainly in hospitalized populations undergoing long term treatment with antibiotics including vancomycin, third generation cephalosporins, anti-anaerobic drugs such as clindamycin or the fluoroquinolones.

Other predisposing conditions include GI tract or chest surgery, invasive or long term in-dwelling procedures, immunosuppression, or critical care admissions.

Patient to patient, health care provider to patient, or fomite to patient transmission is common within the health care setting and re-emphasizes the need for prompt reporting of VRE as well as good hand hygiene and proper contact precautions of the patient by healthcare providers.

Most commonly found in catheter associated UTI and bloodstream infections (5-15% of all bacterial endocarditis), enterococci have also been found in GI tract, kidney and meningeal infections, wound infections and intra-abdominal abscesses.

Colonization with VRE can occur with prolonged antibiotic treatment or hospitalizations, and these individuals are at increased risk of developing a true VRE infection. **Mortality with VRE infections are approximately twice that of VSE infections** (36.6% vs.16.4% respectively- current Johns Hopkins data).

In the microbiology laboratory, enterococci have traditionally been detected by use of the catalase reaction, gram stain, growth in 6.5% NaCl, hydrolysis of bile esculin, and a positive PYR reaction.

Figure 1: StrepQuick can be useful for identification of Enterococcus and other gram positive cocci, Cat no. Z122. It performs a rapid PYR, LAP, and ESC test.

Vancomycin MICs can be detected through an automated susceptibility system, with MICs of less than or equal to 4=Vanco sensitive; MIC of 16=Vanco Intermediate; greater than 16 but less than 64 = E test to confirm resistance, and MIC greater than 64=Vanco resistant.

Repeat testing and/or plating of isolates on vancomycin containing agar (BHI with Vancomycin, Cat. no. G14) is prudent to ensure a true result and rule out mixed cultures or contamination. Recent and future developments in detection of VRE include

chromogenic detection agar for VanA VRE and real time PCR.

In summary, while Vancomycin Resistant Enterococci may not garner as much media attention as MRSA, they are a reservoir from which already highly resistant “superbugs” utilize to increase their virulence.

As microbiologists, we must be alert to their appearance in laboratory cultures and help control healthcare associated infections in our workplaces.

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