

not verify a role for the environment in transmission of VRE, but merely point out that survival can be prolonged. Future studies should address this aspect of the epidemiology of VRE.

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The authors reply

It is known that vancomycin-resistant enterococci (VRE) can survive for prolonged periods in what many other microbes would consider a hostile environment, including environmental surfaces frequently encountered in the healthcare setting.¹ This fact raises concern as to whether these surfaces serve as a reservoir for spread of nosocomial infections. Although this remains speculative, transmission of VRE among patients by a contaminated rectal thermometer has been well documented.² Understanding the potential risk of persistent environmental contamination due to prolonged bacterial survival is one important aspect to control resistant pathogens, because it may have an impact on spread in both institutional and office practice, where infected patients and new hosts at risk are examined and treated.

The report by Bonilla and coworkers in this issue of *Infection Control and Hospital Epidemiology* extends previous investigations examining the duration that VRE can survive on environmental surfaces.³ They documented persistent recovery of VRE for 58 days following inoculation onto a laboratory countertop. The fact that VRE can be recovered for months following inoculation is an important observation for individuals developing infection control practices intended to manage this organism. Their data suggest that transmission to a susceptible host may occur long after the "donor" individual has left the contaminated area. This confirms the necessity of complying with, and perhaps expanding, the Centers for Disease Control and Prevention (CDC) recommendations that individuals entering the room of a patient with VRE wear gloves and that a gown be worn if contact with the patient is anticipated.⁴ At our institution, we have taken a further precaution of requiring both gowns and gloves for everyone entering the room of a patient known to be infected or colonized with VRE and have found that, when carefully followed, this approach can halt the spread of clonal VRE.⁵ Furthermore, periodically obtaining cultures of environmental surfaces in institutions where VRE is endemic appears reasonable. Such environmental surveillance is one way to assess the effectiveness of cleaning procedures that may need periodic re-evaluation in light of the data reported by Bonilla and coworkers.

Our experience suggests that an active, ongoing educational program is needed to maintain awareness of how to manage resistant organisms such as VRE appropriately. Furthermore, it is reasonable that infection control practitioners emphasize that VRE is capable of prolonged survival on inanimate objects frequently encountered in hospitals, because this persistence may play a role in nosocomial transmission. The degree to which this occurs is unknown, but must not be underestimated in developing policies to control VRE. An important question that remains unknown is whether a healthcare worker who touches a surface colonized with VRE can transmit the organism to a patient. While we may never know the precise answer to this question, the fact that this is even possible should persuade healthcare workers to consider carefully the

CDC-approved guidelines for preventing the spread of VRE and consistently apply them to the healthcare setting in which they practice.

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As pointed out by Bonilla et al,¹ a number of investigators have reported that surfaces in the immediate vicinity of patients with vancomycin-resistant enterococci (VRE) frequently become contaminated with the organism. The extent of environmental contamination reported is variable and may depend on whether or not affected patients have diarrhea, the types of patient-care practices that are used to minimize fecal contamination of objects near the patient, the presence or absence of VRE colonization at other body sites, and the adequacy of housekeeping measures.

Contaminated surfaces have the potential to serve as a reservoir for VRE, because the organism can remain viable for days on dry surfaces.^{2,3} Bonilla et al¹ have provided additional data regarding the ability of VRE to survive on contaminated surfaces. The fact that a surface artificially contaminated with stool from a colonized patient yielded viable organisms

after 1 week lends credibility to the belief that contaminated surfaces may serve as potential reservoirs of VRE in hospitals. Their finding that a surface contaminated with a pure culture of VRE yielded viable organisms after nearly 2 months is supported by other studies dealing with the survival of enterococci on dry surfaces.⁴

How important contaminated objects are in transmission of VRE has not been established. Most reports describing environmental contamination by VRE have not provided epidemiologic evidence that patients acquired the organism from the environment. A report implicating contaminated electronic thermometers in the spread of VRE provides the strongest evidence to date that contaminated medical equipment may transmit VRE among patients.⁵ Presumably, transmission from environmental reservoirs also could occur by other means. If "terminal" cleaning of patient rooms between discharge of one patient and admission of a new patient to the room did not remove VRE from contaminated surfaces, the new patient could acquire the organism by touching contaminated items. If this occurred commonly, one would expect to see clustering of VRE cases in certain hospital rooms. To date, this phenomenon has not been reported by hospitals experiencing problems with VRE, and analysis of room locations of VRE cases at the Miriam Hospital seldom has uncovered examples of clustering of cases in certain rooms. These findings suggest that routine terminal cleaning procedures may be adequate in many facilities. Perhaps a more likely scenario is that *daily* cleaning routines in the rooms of colonized patients do not remove VRE from contaminated items and that healthcare workers may contaminate their hands or gloves by touching such objects. If hands are not washed appropriately (or gloves are not removed) upon leaving the patient's room, the organism could be transmitted to other susceptible patients. As suggested by Bonilla et al,¹ additional studies are needed to determine the levels of environmental contamination that are epidemiologically important and the means by which the organism is spread from surfaces to patients.

Transmission of VRE probably occurs most frequently by healthcare workers who have contaminated their hands while caring for an affected patient. Accordingly, control efforts should place a high priority on improv-

ing compliance with recommended barrier precautions and handwashing between patients. Early detection of colonized patients by the microbiology laboratory also has been an important component of effective control programs. Careful cleaning of patient rooms and medical equipment is also important, but should not be the major focus of a VRE control program.

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Bloodstream Infection From a Port-A-Cath: Successful Treatment With the Antibiotic Lock Technique

To the Editor:

A 39-year-old woman with leiomyosarcoma of the stomach had a Port-a-Cath implanted into the arteria hepatica for chemotherapy of liver metastasis during gastrectomy in 1994. In July 1994, complete remission was achieved with six courses of chemotherapy. The arterial port was flushed weekly thereafter to avoid clotting of the catheter. In February 1995, 3 hours after the port had been flushed, the patient complained of myalgia, nausea, chills, and fever up to 40°C and was readmitted to the hospital.

On admission, the patient had no clinical signs of catheter-site infection other than a fever of 38.9°C. The leukocyte count was $2.59 \times 10^9/L$ with 80.5% neutrophils. A penicillinase-positive *Staphylococcus chromogenes*, which was oxacillin susceptible, grew from blood cultures within 16 hours.

Empiric intravenous (IV) therapy with amoxicillin-clavulanic acid was started on admission. The patient became afebrile within 24 hours. Antimicrobial treatment was changed to oral rifampin 450 mg bid and ciprofloxacin 750 mg bid after identification of the microorganism from blood cultures. In addition, the Port-a-Cath system was refilled thrice weekly with 1.6 mL teicoplanin (100 mg) after appropriate aspiration. Rifampin, ciprofloxacin, and teicoplanin were discontinued after 2 weeks; no relapse was observed during follow-up of more than 1 year.

Implanted IV ports and catheters are used widely today for chemotherapy in patients with malignancies. Approximately 1.37 infections per 1,000 catheter-days are observed with implanted IV ports. However, there are few reports on arterial ports and, to our knowledge, none about incidence and treatment of such infections. Given the limited experience, we treated the patient systemically with ciprofloxacin plus rifampin, because of its known efficacy in the treatment of staphylococcal foreign body infections.^{1,2} We additionally administered teicoplanin locally with the antibiotic lock technique.³ This case report raises evidence that arterial port infections can be treated successfully with antimicrobials only.

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