Editorial

Role of Environmental Contamination in the Transmission of Vancomycin-Resistant Enterococci

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The acquisition and spread of bacterial resistance to commonly used antibiotics is an ongoing problem in the 1990s.[1] Important nosocomial pathogens with an increasing incidence or newly acquired resistance include oxacillin-resistant *Staphylococcus aureus*, enteric gram-negative bacilli producing extended-spectrum β-lactamase, and vancomycin-resistant *Enterococcus* species (VRE). Reports of VRE began to appear in the mid-1980s in Europe and are now an important problem in an ever-growing number of hospitals in the United States. Data accumulated via the National Nosocomial Infection Surveillance (NNIS) system of the Centers for Disease Control and Prevention (CDC) revealed that VRE increased 35-fold among all nosocomial isolates of enterococci (0.3% to 10.4%) between 1989 and 1995.[2] By 1994 and 1995, 41% of all NNIS hospitals reported at least one nosocomial enterococcal infection. A recent report notes that attributable mortality is approximately 40%.[3] Because of the importance of VRE as a nosocomial pathogen, the Hospital Infection Control Practices Advisory Committee (HICPAC) of the CDC has published guidelines for preventing nosocomial transmission.[4]

Among the important scientific questions regarding nosocomially acquired VRE are the following. First, do patients colonized or infected with VRE contaminate their environment? Second, what is the role of surface contamination in the transmission of VRE? Third, is surface contamination linked to the transmission of other nosocomial pathogens? Finally, what scientifically based policies can infection control professionals adopt to prevent or reduce nosocomial transmission of these pathogens?

Several investigators have studied the frequency of environmental contamination found in the rooms of patients with VRE.[5-11] Cultures of the surface environment yielded VRE in 7% to 37% of samples (Table). These investigations also produced several other important findings. Boyce et al reported that environmental contamination was more widespread in the rooms of patients with diarrhea[6] (Table). In a later study, Boyce et al reported that the disposable gowns of nurses who cared for a patient with copious diarrhea also were contaminated with VRE.[8] Montecalvo et al reported that 8% of cultures taken after terminal cleaning still yielded VRE.[7]

Molecular analysis of VRE strains has demonstrated both multiple circulating strains[9,11,12] and outbreaks due to a single strain.[6,8,12,13] In some cases, isolates obtained from the environment were identical to the epidemic strain causing infection.[6,8] However, in these outbreaks, it often has been difficult to determine whether cross-transmission occurred due to contaminated common equipment (eg, stethoscopes), acquisition of transient hand car-

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TABLE
ENVIRONMENTAL CONTAMINATION IN ROOMS OF PATIENTS COLONIZED OR INFECTED WITH VANCOMYCIN-RESISTANT ENTEROCOCCI

Reference	Study Subset	Frequency of Contamination	Sites Contaminated
5	ICU housing VRE patients	12% (2/17)	EKG pressure monitor dials, doorknob to isolation room bed
6	Patients without diarrhea	15% (8/53)	Patient gowns, bed linens, bed siderails
	Patients with diarrhea	46% (18/39)	As above plus intravenous pumps, EKG monitors, overbed tables, floors, blood pressure cuff, pulse-oximeter coupling, stethoscope, bathroom door
7	Rooms housing VRE patients	29% (48/167)	—
	Post-terminal cleaning	8% (13/162)	—
8	—	37% (15/41)	Patient gowns, siderails, overbed tables, bed linen, a door handle, the floor, a blood pressure cuff, an intravenous fluid pump, an EKG monitor, a cabinet, a computer table
9	—	13% (4/30)	Electrocardiograph wires, ventilator tubing, a bedside stand, an automated medication dispenser serving the entire surgical intensive-care unit
10	—	7% (5/67)	Blood pressure cuffs in three rooms, a blood glucose monitor, a toilet surface
11	—	7% (22/306)	Sheets, bedrails, bedside tables, blood pressure cuffs

Abbreviations: ICU, intensive-care unit; VRE, vancomycin-resistant *Enterococcus;* EKG, electrocardiogram.

riage by healthcare personnel due to direct contact with a colonized or infected patient, or acquisition of transient hand carriage by healthcare personnel due to contact with a contaminated surface.

Cross-transmission of VRE occasionally has been linked to contaminated medical devices, including an electronic thermometer[13] and a fluidized bed.[14] Disinfection or removal of the contaminated equipment terminated the outbreaks.

The survival of enterococci experimentally inoculated onto environmental surfaces has been studied by Noskin and coworkers.[15] They reported that *Enterococcus faecalis* survived for 5 days and *Enterococcus faecium* for 7 days on countertops. Both enterococcal species survived on bedrails for 24 hours without significant die-off, on telephone handpieces for 60 minutes, on the diaphragmatic surface of stethoscopes for 30 minutes, and on gloved and ungloved fingers for at least 60 minutes. Other investigators also have demonstrated prolonged survival (>3 days) of VRE on either experimentally inoculated surfaces[16] or equipment contaminated by colonized or infected patients.[6] Survival of 18 hours on pieces of sterile cotton sheets (a surrogate for clothes) also has been demonstrated.[17] Frequent surface contamination also has been demonstrated for other nosocomial pathogens, especially oxacillin-resistant *Staphylococcus aureus*[18-21] and *Clostridium difficile.*[22-24] For both pathogens, environmental contamination has been felt to play a role in nosocomial transmission. There also is an extensive literature on the experimental transmission via inanimate surfaces of viruses, including influenza, respiratory syncytial virus, rhinovirus, and rotavirus. Human volunteer studies with rhinovirus and rotavirus also have established that self-inoculation and infection may occur via fingers contaminated with infectious virus present on inanimate surfaces.[25] Further, disinfection of inanimate surfaces has interrupted experimental transmission of infectious rotavirus from inanimate surfaces to humans.[26]

In this issue, Saurina and colleagues provide important data on the activity of disinfectants against VRE.[27] Phenolics, quaternary ammonium compounds, and alcohol all are used for low-level disinfection of noncritical surfaces such as walls, floors, bedrails, blood pressure cuffs, and stethoscopes. The data presented by Saurina et al demonstrated that all of these agents were very effective disinfectants at the tested exposure times of 3 and 10 minutes. It was sur-

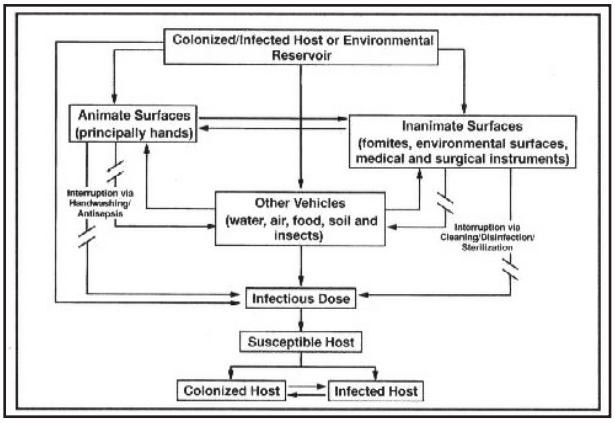

FIGURE. Transmission of infectious agents via animate and inanimate surfaces (modified from reference 25).

prising that 3% hydrogen peroxide did not demonstrate bactericidal activity. While registered hospital disinfectants must demonstrate efficacy against the Association of Official Analytical Chemists (AOAC) test organisms with a 10-minute exposure time, during actual hospital use, exposure times typically are substantially lower. This study demonstrated that a 3-minute exposure time provided greater than a 5-log reduction in VRE for all agents except hydrogen peroxide. However, the practical value of this paper would have been enhanced by studying short exposure times (eg, 30 seconds). In several respects, this study assessed antimicrobial activity under conditions more favorable to inactivation, including the use of a suspension test, sterile water, and no proteinaceous load. Additional studies should expand these initial findings by using a carrier test or surface test to evaluate whether the efficacy of these products is altered by the hardness of the water and the amount and type of proteinaceous material. It should be stressed that current data suggest that antibiotic-resistant strains such as VRE and oxacillin-resistant *S aureus* do not exhibit altered susceptibility to germicides.[28] Even when laboratory strains relatively resistant to germicides have been isolated, the concentrations of resistance are far below the levels of germicides used in clinical practice.

There are multiple transmission routes by which patients may acquire VRE or other infectious agents that are capable of surviving in the environment (Figure). Multiple risk factors have been associated with VRE colonization or infection,[29] including management in an intensive-care unit, prolonged hospitalization, serious underlying diseases, prior antibiotic therapy (especially vancomycin), exposure to contaminated medical equipment, and proximity to known VRE cases. Based on environmental sampling and analysis of risk factors for VRE infection, HICPAC has published guidelines for minimizing the risk of VRE transmission and infection. Investigators have stressed the importance of regulating vancomycin use[5,30]; implementing barrier precautions, including both gloves and gowns[6,31]; surveillance and cohorting of colonized patients[7]; labeling records of patients with VRE to aid in prompt isolation at the time of readmission[8]; and surveillance cultures of persons in close proximity to patients known to be colonized or infected with VRE.[31] However, few of these control measures have been subject to rigorous scientific tests of efficacy.

As stated above, it will be extremely difficult to disentangle the contributions of the animate and inanimate reservoirs of VRE in leading to transient hand carriage of VRE by medical personnel. Clearly, proper hand washing with an antimicrobial agent before and after each contact with patients or their immediate environment is crucial in preventing person-to-person transmission of nosocomial pathogens. Unfortunately, compliance with CDC handwashing guidelines has been noted in less than one half of the instances in which it is indicated.[32] For this reason, additional contact precautions have been recommended, including wearing gloves when entering the rooms of patients with VRE. A recent controlled trial failed to demonstrate an added utility for universal use of gowns plus gloves compared with universal use of gloves only.[11] These findings may have been biased, because widespread environmental contamination did not occur during the study period.[29]

We believe that there is sufficient evidence to state that inanimate surfaces likely play a role in the transmission of VRE. Supportive evidence includes environmental cultures demonstrating widespread surface contamination in rooms of many patients colonized or infected with VRE and experimental evidence that VRE can survive on environmental surfaces for hours and that hands can become colonized with VRE via patient or environmental surfaces. Further, diarrhea in source patients, which would increase environmental contamination, has been reported to be a risk factor for VRE acquisition.

Even though surface contamination may play a role in disease transmission, changes in routine disinfection only are unlikely to reduce disease transmission because recontamination of the patient environment likely is rapid. Preliminary studies suggest that current protocols for terminal cleaning may not eliminate VRE from environmental surfaces.[33] These observations need to be studied using a rigor-

ous scientific protocol. If current terminal cleaning practices are inadequate, modifications may be required.

In conclusion, we believe that widespread environmental contamination with VRE is likely in the rooms of colonized or infected patients. Good handwashing and use of recommended barrier precautions are indicated to prevent cross-transmission of VRE. There is no evidence that changing routine cleaning protocols is likely to alter the level of surface contamination. However, terminal cleaning protocols may need to be altered. Research efforts should focus on improving compliance by healthcare providers with currently recommended handwashing and barrier precautions. The efficacy of control measures should be evaluated in appropriately designed studies.

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