MAJOR ARTICLE

Prior Environmental Contamination Increases the Risk of Acquisition of Vancomycin-Resistant Enterococci

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(See the editorial commentary by Harris on pages 686–8)

Background. Patients colonized with vancomycin-resistant enterococci (VRE) frequently contaminate their environment, but the environmental role of VRE transmission remains controversial.

Methods. During a 14-month study in 2 intensive care units, weekly environmental and twice-weekly patient surveillance cultures were obtained. VRE acquisition was defined as a positive culture result >48 h after admission. To determine risk factors for VRE acquisition, Cox proportional hazards models using time-dependent covariates for colonization pressure and antibiotic exposure were examined.

Results. Of 1330 intensive care unit admissions, 638 patients were at risk for acquisition, and 50 patients (8%) acquired VRE. Factors associated with VRE acquisition included average colonization pressure (hazard ratio [HR], 1.4 per 10% increase; 95% confidence interval [CI], 1.2–1.8), mean number of antibiotics (HR, 1.7 per additional antibiotic; 95% CI, 1.2–2.5), leukemia (HR, 3.1; 95% CI, 1.2–7.8), a VRE-colonized prior room occupant (HR, 3.1; 95% CI, 1.6–5.8), any VRE-colonized room occupants within the previous 2 weeks (HR, 2.5; 95% CI, 1.3–4.8), and previous positive room culture results (HR, 3.4; 95% CI, 1.2–9.6). In separate multivariable analyses, a VRE-colonized prior room occupant (HR, 3.8; 95% CI, 2.0–7.4), any VRE-colonized room occupants within the previous 2 weeks (HR, 2.7; 95% CI, 1.4–5.3), and previous positive room culture results (HR, 4.4; 95% CI, 1.5–12.8) remained independent predictors of VRE acquisition, adjusted for colonization pressure and antibiotic exposure.

Conclusions. We found that prior room contamination, whether measured via environmental cultures or prior room occupancy by VRE-colonized patients, was highly predictive of VRE acquisition. Increased attention to environmental disinfection is warranted.

Since first described in 1988, vancomycin-resistant enterococci (VRE) have become an important nosocomial pathogen in the United States and worldwide [1, 2]. By 2003, the percentage of enterococcal isolates in US intensive care units (ICUs) resistant to vancomycin had reached nearly 30% [3], and VRE are now the third-leading cause of hospital-acquired infection [4]. Although asymptomatic colonization exceeds infection by 10-fold [5], colonized patients frequently develop subsequent VRE infection [6]. VRE infections have been linked to increased morbidity, mortality, and health care expenditures [7–13], making prevention of VRE colonization of utmost importance. Extensive research has identified multiple risk factors for VRE acquisition, including severity of illness, length of hospital stay, antibiotic exposure, and colonization pressure (the proportion of VRE-colonized patients in a given unit) [14–19].

Vancomycin resistance does not arise de novo in vancomycin-susceptible enterococci via spontaneous mutation [20, 21]; instead, susceptible patients acquire VRE exogenously in the context of antibiotic selective pressure. Because hand hygiene among health care workers is frequently suboptimal [21], it is thought that

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spread of VRE between patients occurs primarily via health care workers [22, 23]. However, it is also well known that VRE colonizes environmental surfaces and equipment and may persist for prolonged periods despite routine cleaning [24, 25]. VRE transmission has been attributed to fomites, such as fluidized beds [26] and thermometers [27, 28], but in settings of endemicity, it is frequently difficult to differentiate environmental acquisition from that associated with health care workers.

Although many studies have suggested that environmental contamination plays an important role in VRE transmission, all have been subject to various limitations. These studies have been experimental in nature rather than assessing room or hand contamination that occurs during usual patient care [29, 30], have selected only certain rooms or time periods for environmental sampling [6, 15, 31, 32], or have been conducted in outbreak situations [22], when both levels of contamination and health care workers' and housekeeping behavior may differ from those in settings of endemicity. Several other studies have investigated risk of VRE acquisition due to admission to rooms that previously housed VRE-positive patients, as a proxy for environmental contamination [19, 33]. However, these studies have been limited by retrospective design and subsequent inability to control for other known risk factors for VRE acquisition, such as antibiotic exposure, severity of illness, or colonization pressure, and they have lacked environmental cultures as direct proof of room contamination. To overcome these limitations, we performed weekly environmental and twice-weekly patient surveillance cultures during a 14-month prospective study conducted at our academic tertiary care medical center. Our study objectives were to determine the extent of VRE environmental contamination and subsequent risk of acquisition.

RESEARCH DESIGN AND METHODS

Study design and setting. Data derive from a prospective, interventional, crossover study in the medical and surgical ICUs at Tufts–New England Medical Center, Boston, conducted during the period February 2002 through March 2003. Each unit comprises 10 private rooms. The intervention consisted of discontinuing the gown use requirement from the contact precautions protocol for patients infected or colonized with VRE or methicillin-resistant *Staphylococcus aureus* and has been described previously [34]. The study consisted of 3 phases, during which gown use was required in both units, the medical ICU but not the surgical ICU, and then the opposite. Standard contact precautions were used for VRE-colonized patients in both units throughout the study period. Standard housekeeping procedures included damp disinfection of all surfaces within reach, including walls and furniture, using a quaternary ammonia solution. Curtains were changed only if visibly soiled. No additional methods were used for the rooms of VRE-colonized patients, and housekeeping procedures did not change during the study period. No specific measurement of the compliance with these housekeeping procedures was performed. The institutional review board approved the study protocol.

Data collection. Prospectively collected patient data included demographic information; comorbidities; baseline vital signs and laboratory results; date and time of ICU admission and discharge; and need for short-term hemodialysis, mechanical ventilation, or surgery. APACHE-II scores were calculated using these baseline data. Antibiotic use was retrieved via the pharmacy database and recorded as days of exposure to each antibiotic class. Hand hygiene compliance was monitored during randomly selected periods and was found not to differ significantly between periods during which gown use was or was not required [34].

Microbiological methods. Rectal swab or stool specimens were obtained from each patient within 48 h after ICU admission, twice weekly while the patient remained in the ICU, and at the time of ICU discharge. Environmental cultures were taken weekly from 2 specific locations in each room (bed rails and the intravenous pump). Culture specimens were obtained by rubbing premoistened swabs repeatedly over each designated site and placing the swabs in liquid transport media. Compliance was ~80% for patient surveillance cultures and 98% for environmental surveillance cultures. Specimens were inoculated onto BBL Campy CVA agar (BD), and plates were examined after 48 h of incubation. Colonies suspected of being VRE were confirmed as VRE using Gram staining, catalase, pyruvate, and glucose fermentation testing.

Definitions. We defined initial VRE colonization as isolation of VRE within the first 48 h after ICU admission and VRE acquisition as isolation of VRE after the first 48 h after ICU admission, with a negative initial culture result and no history of VRE colonization or infection. We assumed that patients remained colonized for the duration of their current and any subsequent ICU stays. We censored data for patients who acquired VRE after acquisition and excluded patients colonized at admission from analysis, but both groups were used to calculate colonization pressure. For patients with multiple ICU admissions or transfer between ICUs, the ICU stay was considered to be continuous if $\leq$7 days had elapsed between stays but was considered to be a new ICU admission if >7 days had elapsed.

We defined daily colonization pressure as the proportion of patients in the ICU who were colonized on each day, and we calculated daily cumulative averages of each patient's colonization pressure until either acquisition of VRE or ICU discharge occurred. We adjusted each calculation for the fractional days patients spent in the ICU on days of admission and discharge.

Table 1. Characteristics of patients who did and did not acquire vancomycin-resistant enterococci (VRE).

Characteristic	Patients who acquired VRE (n = 50)	Patients who did not acquire VRE (n = 588)	P
Demographic characteristics and comorbidities			
Age, mean years ± SD	56.8 ± 14.5	58.8 ± 16.3	.39
Male sex	52.0	57.3	.47
Chronic renal failure	12.5	12.2	.94
Long-term hemodialysis	4.2	3.7	.87
Gastrointestinal disease	18.8	16.2	.64
Chronic liver disease	20.1	17.9	.61
Wounds	22.9	24.2	.84
Diabetes	20.8	21.8	.88
Transplantation	14.6	10.6	.40
Leukemia	10.4	3.8	.03
Other cancer	16.7	23.1	.31
Any immunodeficiency[a]	36.0	36.2	.97
Severity of illness indicators			
Mean APACHE-II score ± SD	21.5 ± 7.3	18.9 ± 8.0	.03
Mechanical ventilation	75.0	58.6	.03
Short-term hemodialysis	18.8	9.3	.04
Any surgery	39.6	42.4	.71
Abdominal surgery	20.8	19.3	.79
Length of stay			
Time to VRE acquisition or ICU discharge, median days (IQR)	7.0 (4.0–10.0)	4.0 (3.0–9.0)	.01
Total length of stay in the ICU, median days (IQR)	17.8 (9.1–33.4)	4.0 (3.0–9.0)	<.001
Total length of hospital stay, median days (IQR)	32.0 (20.0–49.0)	12.0 (7.0–21.0)	<.001
Location/colonization pressure			
Medical ICU (vs. surgical ICU)	62.0	47.8	.05
Colonization pressure, mean % ± SD	22.6 ± 9.2	17.0 ± 9.9	<.001

NOTE. Data are percentage of patients, unless otherwise indicated. ICU, intensive care unit; IQR, interquartile range.

[a] Diabetes, leukemia, chronic renal failure, or transplantation.

We defined antibiotic exposure as the proportion of ICU-days, prior to either VRE acquisition or ICU discharge, during which the patient received each antibiotic or antibiotic class. We calculated these variables by dividing the total number of days of antibiotic use by the length of ICU stay. Only antibiotics that were given in the ICU were available for analysis. We grouped antibiotics into the following classes: vancomycin (oral or parenteral), metronidazole (only parenteral was available in the database), all cephalosporins, third- or fourth-generation cephalosporins, carbapenems, extended-spectrum penicillins (β-lactam/β-lactamase–inhibitor combinations), and anti-anaerobic agents (clindamycin, metronidazole, carbapenems, and extended-spectrum penicillins). Fluoroquinolones were not reliably recorded in the database and were therefore not analyzed further.

We investigated the role of environmental contamination in several ways. We determined whether the patient was admitted to a room in which the immediate prior occupant was colonized with VRE and whether any VRE-colonized patient had stayed in the same room within the past 2 weeks. We also examined whether there were any positive results of environmental cultures in the patient's room within 1 week before the patient's admission or during the patient's admission but prior to VRE acquisition.

Statistical analysis. We compared characteristics of patients who did or did not acquire VRE using Mantel-Haenszel χ^2 and t tests for categorical and continuous variables, respectively. We performed Wilcoxon rank sum tests for continuous variables that were not normally distributed. Variables included baseline characteristics (demographics, comorbidities), severity of illness indicators (APACHE-II score, mechanical ventilation, hemodialysis, or surgery), and other previously described risk factors for VRE acquisition (antibiotic exposure, length of stay, and colonization pressure). We used Cox proportional hazards models with time-dependent variables to account for the cumulating changes in exposure to antibiotics and colonization pressure. Because of significant collinearity of the antibiotic exposure variables and to avoid overfitting the multivariate models, we created composite antibiotic variables and used clinical judgment to determine which variables should be in-

Table 2. Antibiotic and environmental exposures in patients who did and did not acquire vancomycin-resistant enterococci (VRE).

Exposure	Patients who acquired VRE (n = 50)	Patients who did not acquire VRE (n = 588)	P
Median no. of antibiotics per day (IQR)	0.85 (0.14–1.40)	0.17 (0.00–0.75)	<.001
Antibiotic use, mean % of ICU days ± SD[a]			
Vancomycin	24.0 ± 27.6	9.3 ± 19.5	<.001
All cephalosporins	14.6 ± 28.3	6.7 ± 17.9	.09
Third- and/or fourth-generation cephalosporins	13.1 ± 26.6	2.5 ± 12.3	<.001
Intravenous metronidazole	18.0 ± 28.9	5.5 ± 17.6	<.001
Carbapenems	6.1 ± 19.0	2.2 ± 11.0	.002
Extended-spectrum penicillins	12.0 ± 21.1	8.0 ± 21.0	.02
Anti-anaerobic agents	36.9 ± 34.7	16.2 ± 28.7	<.001
Environmental variables			
Prior room occupant colonized with VRE, %	38.0	20.2	.003
Any room occupant in prior 2 weeks colonized with VRE, %	60.0	41.8	.01
Positive room culture result before admission or VRE acquisition, %	8.0	4.8	.31
Any positive room culture result during patient's ICU stay, %	28.0	1.7	<.001
Any positive room culture result within 1 week after patient's ICU stay, %	6.0	1.0	.004

NOTE. ICU, intensive care unit; IQR, interquartile range.

[a] Only antibiotic use prior to VRE acquisition was included. Means ± SDs are reported because the median values for most of these variables were 0 for both groups. Because of the skewed distribution of these variables, Wilcoxon rank sum tests were used to calculate P values.

cluded in the final models. We checked proportional hazards assumptions using Schoenfeld residuals and explored interactions of predictor variables with time. All analyses were performed using SAS software, version 9.1 (SAS Institute).

RESULTS

During the study period, 1330 patients were admitted to the 2 study ICUs. Of these patients, 118 (9%) were colonized with VRE at the time of admission, and 574 (43%) were excluded from analysis (because of an ICU stay <48 h in duration or a prior history of VRE but negative culture result at admission). Therefore, 638 patients were at risk for new VRE acquisition, of whom 50 (8%) acquired VRE. Of the 638 at-risk patients, approximately one-half were admitted to each unit. During the study period, there were ~8200 total patient-days, of which ~1900 were VRE-positive patient–days. The medical ICU had higher endemic rates of VRE than did the surgical ICU throughout the study period, with overall average colonization pressures of 26% (range, 0%–70%) and 15% (range, 0%–40%), respectively. No clinically apparent outbreaks of VRE infection occurred during the study period.

Patients who acquired VRE were more likely than those who did not acquire to have leukemia, but no other demographic characteristics or comorbidities were significantly different (table 1). Patients who acquired VRE had slightly higher APACHE-II scores and more frequently required mechanical ventilation or short-term hemodialysis. Total lengths of stay in the hospital and in the ICU were longer for patients who acquired VRE; the durations of both pre- and postacquisition stays were increased, compared with those for patients who did not acquire VRE.

Patients who acquired VRE were more likely to stay in the medical ICU and were exposed to a higher average colonization pressure than were those who did not acquire VRE (table 2). Patients who acquired VRE took more antibiotics than did those who did not acquire VRE, regardless of whether antibiotics were calculated as average number of all antibiotics per day or proportion of ICU days exposed to specific groups of antibiotics. Use of vancomycin, third- or fourth-generation cephalosporins, metronidazole, and anti-anaerobic agents was significantly more common among patients who acquired VRE. Vancomycin use was significantly correlated with the use of cephalosporins, metronidazole, carbapenems, and extended-spectrum penicillins (data not shown); its independent contribution to VRE acquisition could not be elucidated.

During the study period, ~1220 environmental samples for culture were obtained from patient rooms in each unit, of which 47 (4%) yielded positive results in the medical ICU and 20 (2%) yielded positive results in the surgical ICU. Of 61 instances when a room had any positive culture results, 7 (11%) yielded positive culture results for both sites on the same day. Approximately one-quarter of patients who acquired VRE had a positive environmental room culture result during their stay, and 6% had a positive environmental culture result for their room within 1 week after they had been discharged. Patients who acquired VRE were more likely to stay in a room that previously housed a VRE-colonized patient, whether defined as only the immediate prior patient or any patient within the

Table 3. Univariate predictors of acquisition of vancomycin-resistant enterococci (VRE) using Cox proportional hazards.

Predictor	Hazard ratio (95% CI)	*P*
Leukemia	3.09 (1.22–7.83)	.02
APACHE-II score (per point)	1.00 (0.96–1.04)	.90
Mechanical ventilation	0.90 (0.46–1.75)	.76
Short-term hemodialysis	1.40 (0.68–2.90)	.37
Medical ICU location	1.82 (1.03–3.24)	.04
Average colonization pressure[a]	1.43 (1.12–1.84)	.005
Length of ICU stay prior to VRE acquisition or discharge (per day)	0.94 (0.86–1.02)	.14
Mean no. of antibiotics per day (per antibiotic)	1.72 (1.16–2.54)	.007
Antibiotics, % of ICU days[b]		
Vancomycin	1.01 (1.00–1.02)	.02
Third- or fourth-generation cephalosporins	1.03 (1.02–1.04)	<.001
Metronidazole	1.01 (1.00–1.02)	.02
Carbapenems	1.01 (0.99 –1.02)	.43
Extended-spectrum penicillins	1.00 (0.99–1.01)	.70
Anti-anaerobic agents	1.01 (1.00–1.01)	.09
Environmental variables		
Prior room occupant colonized with VRE	3.07 (1.63–5.80)	<.001
Any room occupant in prior 2 weeks colonized with VRE	2.49 (1.30–4.80)	.006
Positive room culture result prior to admission or VRE acquisition	3.39 (1.20–9.58)	.02
Either positive room culture result or prior room occupant colonized with VRE	2.52 (1.43–4.45)	.001

NOTE. ICU, intensive care unit.

[a] Reported hazard ratio is calculated per 10% increase in average colonization pressure (i.e., per 1 additional VRE-colonized patient in a 10-bed unit).

[b] Reported hazard ratios are calculated per 1% increase in ICU days during treatment with each antibiotic.

previous 2 weeks. Patients who acquired VRE were also more likely to have a room culture test positive for VRE within 1 week before their admission, although this finding did not achieve statistical significance.

In univariate analysis, each additional VRE-colonized patient in the ICU per day (i.e., 10% increase in average colonization pressure) resulted in an ~40% increased risk of VRE acquisition (table 3). Each additional antibiotic, regardless of type, increased risk of acquisition by 72%. Among specific classes of antibiotics, vancomycin, third- or fourth-generation cephalosporins, metronidazole, and anti-anaerobic agents were significantly associated with VRE acquisition. These hazard ratios appear small but are cumulative; a hazard ratio of 1.02 for third- or fourth-generation cephalosporins indicates a 2% increase in risk for each 1% increase in cephalosporin use. A positive environmental culture result for a patient's room before admission increased risk of acquisition by >3-fold. VRE acquisition was also more than twice as likely when patients were admitted to rooms that had previously housed VRE-colonized patients (either the immediate prior patient or any patients within the previous 2 weeks).

In multivariable analysis, a positive room environmental culture remained a significant predictor of VRE acquisition, after colonization pressure and antibiotic exposure were adjusted for (see model 1 in table 4). Identical models substituting the other environmental variables (VRE-colonized prior room occupant or any VRE-colonized room occupants within the past 2 weeks) for the positive room culture variable also showed a significant but lessened effect (models 2 and 3, respectively). Leukemia did not remain significant in any of these models. Model diagnostics determined that the risk of acquisition due to a previously contaminated room was nonproportional (i.e., risk decreased over time). To correct for this phenomenon, the environmental variables were allowed to remain positive only for the first 2 weeks that the subsequent patient remained in the same room. The revised models (table 4) demonstrated slightly higher hazard ratios and lower *P* values but were otherwise unchanged. No significant interactions were detected.

DISCUSSION

Patients acquire VRE under 2 conditions: exposure to exogenous VRE organisms and susceptibility to the establishment of VRE colonization, usually via antibiotic exposure [35]. Our study found that colonization pressure, a measure of exposure to VRE in the unit, and multiple antibiotic classes increased risk of VRE acquisition. However, adjusting for these factors, we found that the strongest predictors of VRE acquisition were

Table 4. Multivariate predictors of acquisition of vancomycin-resistant enterococci (VRE) using Cox proportional hazards.

Model, predictor	Adjusted hazard ratio (95% CI)	*P*
Model 1		
Positive room culture result prior to admission or VRE acquisition	4.35 (1.49–12.75)	.007
Average colonization pressure[a]	1.36 (1.06–1.76)	.02
Mean no. of antibiotics per day[b]	1.88 (1.25–2.84)	.003
Model 2		
Previous patient in room colonized with VRE	3.82 (1.99–7.35)	<.001
Average colonization pressure[a]	1.39 (1.07–1.81)	.01
Mean no. of antibiotics per day[b]	1.89 (1.27–2.82)	.002
Model 3		
Any VRE-positive patient in room within previous 2 weeks	2.69 (1.37–5.29)	.004
Average colonization pressure[a]	1.39 (1.08–1.80)	.01
Mean no. of antibiotics per day[b]	1.78 (1.19–2.66)	.005

[a] Reported hazard ratio is calculated per 10% increase in average colonization pressure (i.e., per one additional VRE-colonized patient in a 10-bed unit).

[b] Reported hazard ratio is calculated per one additional antibiotic.

a prior positive room culture for VRE and prior room occupancy by VRE-colonized patients (a presumed proxy for room contamination). This effect persisted for as long as 2 weeks, when at least one and possibly multiple "terminal cleanings" had occurred. Indeed, VRE has been shown to persist through an average of 2.8 standard room cleanings [24], and even when the more effective "bucket" method [24] is used, the results may be suboptimal without additional housekeeper training and close monitoring [32]. In experimental settings, VRE has been found to persist up to 58 days on counter tops [36] and can also persist in fabrics for extended periods [37, 38]. Of note, multiple antibiotic resistance does not reduce susceptibility of enterococci to routinely used disinfectants [21, 38], so no special compounds, such as bleach, are required. We also discovered that the risk due to room contamination declines over time, as would be expected because of natural decay of the environmental reservoir as well as removal via daily room cleaning.

Differentiating VRE transmission via health care workers' hands from direct transmission from the environment is difficult. Organisms found in the environment may result from rather than cause patients' colonization [39]. In settings of endemicity, multiple strains of VRE frequently circulate, and it cannot be proven that a patient's VRE isolate came from the environment and not from a concurrent patient [20]. However, even if all VRE transmission does occur via health care workers, interventions aimed at the environment are still likely to reduce transmission, given the frequency of environmental contamination [30, 40, 41] that can subsequently contaminate health care workers' hands, even without direct patient contact [29, 30]. Health care workers are clearly capable of carrying VRE from a contaminated site to a noncontaminated site [31] and frequently touch room sites after hand hygiene upon entry into a room and before touching patients. Although we found positive environmental cultures to be a significant risk factor for VRE acquisition, our overall yield from environmental cultures was quite low (<5% positivity rate in both units). Environmental cultures may be a useful tool for health care facilities to monitor the effectiveness of room disinfection procedures or in specific circumstances, such as outbreak control. Our data do not support routine environmental cultures as an infection control intervention.

The strengths of this study include prospective design with subsequent ability to control for other known risk factors for VRE acquisition. We conducted the study over a 14-month period in both a medical and a surgical ICU and performed weekly environmental surveillance for VRE in every room throughout the duration of the study. Therefore, we were able to document the temporal relationship between room contamination and VRE acquisition. Cox proportional hazard models were used to fully utilize the time-dependent nature of several of the predictive variables and demonstrated the decreasing risk of environmental contamination over time. We were able to find a significant effect of colonization pressure, antibiotic exposure, and room contamination despite relatively low levels of endemicity and few VRE acquisitions, and our findings are generalizable to other tertiary care academic centers with low to moderate levels of VRE endemicity and other nonoutbreak situations.

This study also has certain limitations. Surveillance cultures, whether of patients or the environment, are not 100% sensitive and depend on the quantity of VRE in the stool [42] and the sites of environmental sampling. Therefore, some acquisitions may have been preexisting colonization not detected by baseline

surveillance, and some environmental contamination was likely not detected. We did not perform enrichment cultures, which further lowers the study's sensitivity, but the culture methods used were taken to maximize feasibility given that we performed nearly 50 environmental and 100 patient cultures weekly for >1 year. Antibiotic exposure, colonization pressure, and environmental exposure that occurred outside of the ICU were not available, nor were VRE acquisitions after patients left the ICU (unless a clinical culture yielded VRE). That leukemia was the only comorbidity significantly associated with VRE acquisition (in univariate but not multivariate analysis) may indicate that leukemia is a marker for higher antibiotic exposure or residence in the hematology-oncology ward, which tended to have higher VRE rates than other non-ICU settings, rather than a direct cause of VRE acquisition itself. Last, the relatively few VRE acquisitions and positive environmental cultures, although a positive finding from a patient care and infection control standpoint, limited our statistical ability to evaluate multiple risk factors and required the use of a composite antibiotic variable in our final models.

VRE has been described as a "triple threat" for its ability to colonize patients' gastrointestinal tracts, skin, and the environment [20]. When patients become colonized or infected, not only can gastrointestinal colonization persist for months or years [43], but patients also frequently contaminate their skin and immediate environment. Our study has demonstrated that prior environmental contamination, whether measured directly with environmental cultures or via prior room occupation by VRE-positive patients, places patients at risk for VRE acquisition, and that this risk persists after adjusting for other important risk factors, such as colonization pressure and antibiotic exposure. Universal hand hygiene is critical for patient safety and must continue to be emphasized. However, interventions aimed at optimizing environmental disinfection will likely reduce not only direct acquisition from the environment but also the frequency of contamination of health care workers and subsequent transmission of VRE and other resistant pathogens.

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