American Journal of Infection Control 47 (2019) 13–17

Contents lists available at ScienceDirect

American Journal of Infection Control

journal homepage: www.ajicjournal.org

Major Article

Bacterial burden is associated with increased transmission to health care workers from patients colonized with vancomycin-resistant *Enterococcus*

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Key Words:
Vancomycin-resistant *Enterococcus*
Bacterial burden
Health care workers
Contact precautions

Background: Health care workers (HCWs) are significant vectors for transmission of multidrug-resistant organisms among patients in intensive care units (ICUs). We studied ICU patients on contact precautions, colonized with vancomycin-resistant *Enterococcus* (VRE), to assess whether bacterial burden is associated with transmission to HCWs' gloves or gowns, a surrogate outcome for transmission to subsequent patients.
Methods: From this prospective cohort study, we analyzed 96 VRE-colonized ICU patients and 5 HCWs per patient. We obtained samples from patients' perianal area, skin, and stool to assess bacterial burden and cultured HCWs' gloves and gowns for VRE after patient care.
Results: Seventy-one of 479 (15%) HCW-patient interactions led to contamination of HCWs' gloves or gowns with VRE. HCW contamination was associated with VRE burden on the perianal swab (odds ratio [OR], 1.37; 95% confidence interval [CI], 1.19, 1.57), skin swabs (OR, 2.14; 95% CI, 1.51, 3.02), and in stool (OR, 1.95; 95% CI, 1.39, 2.72). Compared with colonization with *Enterococcus faecalis*, colonization with *Enterococcus faecium* was associated with higher bacterial burden and higher odds of transmission to HCWs.
Conclusions: We show that ICU patients with higher bacterial burden are more likely to transmit VRE to HCWs. These findings have implications for VRE decolonization and other infection control interventions.

BACKGROUND

Enterococcus makes up 14% of all hospital-associated infections (HAIs) reported to the National Healthcare Safety Network at the Centers for Disease Control and Prevention.[1] Roughly 35.5% of all enterococcal HAIs are resistant to vancomycin.[1,2] Vancomycin-resistant *Enterococcus* (VRE) is responsible for bacteremia, surgical site infections, and urinary tract infections[2] and is the cause of approximately 1,300 deaths annually.[3] The 2 predominant species of VRE are *Enterococcus faecium* and *Enterococcus faecalis*, which make up roughly 77% and 9% of isolates, respectively.[3]

Most patient colonization remains unknown in the absence of active surveillance.[4] However, VRE has a demonstrated propensity for skin colonization, which can increase the risk of catheter-related bacteremia and cross-infection.[5] Patients' skin colonization can also lead to transmission of VRE to the hands and clothing of health care workers (HCWs) while providing patient care. This is especially concerning because VRE has been shown to last up to 60 minutes on HCWs' hands in the absence of hand hygiene.[6] This contamination of HCWs can lead to further transmission of VRE to patients' other body sites (cross-contamination), the patient's environment, or other patients in the HCWs' care.[7]

Whether the bacterial burden of VRE increases the risk of transmission is thus far unknown. Although several studies have found

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Presented in part at the Society for Healthcare Epidemiology Annual Meeting, Portland, OR, April 2018 (abstract no. 10453).
Funding/support: Supported by the National Institute of Allergy and Infectious Diseases at the National Institutes of Health (grant no. 5R01AI121146) and the Centers for Disease Control and Prevention (grant no. 1U54CK000450), US Department of Health and Human Services.
Conflicts of interest: S.S.J., L.G.M., and D.P.C. report grants from the National Institutes of Health; A.D.H., L.S.M., and J.K.J. report grants from the National Institutes of Health and the Centers for Disease Control and Prevention; and K.A.S. and K.A.T. report no conflicts of interest relevant to this article.

https://doi.org/10.1016/j.ajic.2018.07.011
0196-6553/

that colonized patients can transmit multidrug-resistant organisms (MDROs), including VRE, to HCWs' gloves and gowns, these studies suffered from small sample sizes,[8-11] and associations between patient bacterial burden or colonizing species and HCW contamination were not estimated. Therefore, we conducted a study of intensive care unit (ICU) patients colonized with VRE to assess the relationship between bacterial burden and transmission to HCWs' gloves or gowns. These results could have major implications for infection prevention practices, such as the use of contact precautions and decontamination policies for patients with high levels of bacterial colonization.

METHODS

Study design and participants

We conducted a prospective cohort study to estimate the contribution of bacterial burden to the transmission of VRE from patients to HCWs' gloves and gowns, a surrogate outcome for possible transmission to other patients. One hundred patients were recruited from the medical and surgical ICUs within the University of Maryland Medical Center between January 1 and November 15, 2017. The medical ICU is a 29-bed unit that provides care to adult patients with acute or potentially life-threatening medical conditions, whereas the surgical ICU is a 24-bed unit designed to care for adult surgical patients. These ICUs screen for VRE on rectal swabs at admission and once weekly as part of the VRE infection prevention active surveillance program. The research staff were notified each day of patients with recent (within 72 hours) VRE-positive rectal surveillance cultures via email alerts associated with hospital microbiology reports. The first 5 HCWs for each patient were approached sequentially for participation in the study before engaging in care activities. The University of Maryland Baltimore institutional review board approved this study.

Data collection

We cultured the patients' perianal area, chest, and antecubital fossa and obtained a stool sample (when available), to measure patient VRE bacterial burden. The perianal area was sampled using aseptic technique with ESwab (Copan Diagnostics, Murrieta, CA). The swab was rubbed gently back and forth 3 times on the skin immediately around the anus, covering an area approximately 4 cm in diameter. The chest and antecubital fossa were chosen because these body sites are likely to be touched by HCWs and have been examined in previous studies.[5,8,10] These skin sites were sampled using a 10- $\times$ 10-cm^2 template, rubbing the swab within the template with a twirling motion to ensure that all sides of the swab came in contact with the skin. Stool samples were collected (when available) in sterile stool specimen containers (Dynarex, Orangeburg, NY).

Five interactions between HCWs and patients were observed shortly after (within a few hours) the patient swabs were obtained. After patient care, but before doffing, the gloves and gowns of each HCW were cultured for the presence of VRE. The BBL dual Culturette swab (BD, Franklin Lakes, NJ) was rubbed gently with a twirling motion along the dorsum of each finger and the palm of both the right and left hands. HCWs' gowns were sampled with a separate swab, using a twirling motion twice on each forearm and then in a *W* pattern along the beltline.

The HCW role and duration of time each HCW spent in the room was observed by the study researchers. Patient characteristics including the presence or absence of an artificial airway (endotracheal or tracheostomy tube), Foley catheter, central venous catheter (central line), chest tube, surgical drain, rectal tube, nasogastric tube, diarrhea, and wound were also collected. ICD-10 codes, age, sex, and race were abstracted from the electronic medical record of each patient. The ICD-10 codes were used to calculate the Elixhauser Index, which is a validated comorbidity score for hospital inpatients.[12]

Laboratory procedures

HCWs' gown and glove swabs were cultured for the presence of VRE. The swabs were placed into tryptic soy broth with 6.5% NaCl and incubated for 24 hours at 35°C $\pm$ 2°C. After incubation, 50 μL from each broth tube were inoculated onto Bile Esculin Azide Agar with 6 μg/mL vancomycin (BEAV; Remel, Lenexa, KS) plate for isolation. The BEAV plates were incubated aerobically at 35°C $\pm$ 2°C for 48 hours. All enterococcal isolates were frozen in tryptic soy broth with 15% glycerol and stored at $-$80°C.

Patient swabs and stool samples were placed into collection tubes, and the swabs from the skin and perianal samples were vortexed separately for 1 minute. One gram of stool was extracted from the stool container, added to 1 mL of 0.9% saline in an Eppendorf tube, and vortexed until well mixed (at least 1 minute). One mL of each patient sample was serially diluted using Butterfield's Buffer. BEAV was inoculated with 100 μL of each serial dilution and distributed evenly onto the agar plate using a cell spreader. Also, 100 μL of the original sample was inoculated into tryptic soy broth with 6.5% NaCl. The plates and broth tubes were incubated for 48 hours aerobically at 35°C $\pm$ 2°C, after which the number of bacterial colonies was counted. If there was no growth on the inoculated plates, 100 μL from the previously inoculated tryptic soy broth with 6.5% NaCl tubes were inoculated onto a BEAV plate. If there was growth on the BEAV agar after 48 hours at 35°C $\pm$ 2°C, a count of 1 colony-forming unit (CFU) was given. After incubation, colonies were subcultured and identified to the species level by the VITEK II (BioMérieux, Marcy-l'Étoile, France). Antimicrobial susceptibility testing was conducted using the Kirby-Bauer disk diffusion method according to the Clinical Laboratory Standards Institute guidelines.

Statistical analysis

Frequencies with proportions and means with SD were calculated to describe the demographics, clinical characteristics, and VRE species of the sample. Bacterial counts were logged transformed ($\log_{10}$ [VRE bacterial burden +1]) so that those without recovered VRE were included as 0, and counts were modeled in $\log_{10}$ CFU/mL for the perianal and stool samples and in $\log_{10}$ CFU/cm^2 for the skin samples. The chest and antecubital fossa skin swabs were combined into 1 variable by taking the higher of the 2 measurements. The association between patient bacterial burden and VRE species with HCWs' glove or gown contamination was estimated by use of logistic regression fit with generalized estimating equations with an exchangeable correlation matrix used to account for within-patient correlation. These associations were expressed in odds ratios (ORs) and 95% confidence intervals (CIs). The final model was selected by testing all covariates in the model, and the model with the smallest quasi-Akaike information criterion was chosen. Separate models were constructed for VRE burden recovered from each of the cultured patient sites. The following covariates were selected a priori as potential confounders: patient age, race, comorbidities, presence of invasive devices, diarrhea, type of HCW (physician/nurse practitioner, nurse, patient care technician, physical/occupational therapist, respiratory technician, or other), and duration of time the HCW spent in the room. We estimated the mean bacterial burden and 95% CI found in each of the samples by species. Pearson correlations were calculated to compare the bacterial burden between each sample. All analyses were conducted using SAS version 9.4 (SAS Institute, Cary, NC).

Table 1
Demographics and clinical characteristics of VRE-colonized ICU patients enrolled between January 1, 2017, and November 15, 2017

Characteristic	N = 96
Age in years, mean (SD)	60.8 (13)
White race	51 (53)
Male sex, n (%)	50 (52)
ICU location, n (%)	
Medical ICU	58 (60)
Surgical ICU	38 (40)
Elixhauser Index, median (range)	6 (0-14)
Diarrhea, n (%)	29 (30)
Wound, n (%)	56 (58)
Endotracheal tube, n (%)	50 (52)
Central line, n (%)	68 (71)
Foley catheter, n (%)	55 (57)
Chest tube, n (%)	7 (7)
Surgical drain, n (%)	25 (26)
Rectal tube, n (%)	32 (33)
Nasogastric tube, n (%)	53 (55)
Number of devices, mean (SD)	3 (1.6)

ICU, intensive care unit; *VRE*, vancomycin-resistant *Enterococcus*.

Table 2
Adjusted associations between VRE bacterial burden and HCWs' glove or gown contamination by patient sample type

Patient sample type	OR (95% CI)
Perianal (log_{10} CFU/mL)	1.37 (1.19, 1.57)*
Skin (log_{10} CFU/cm^2)	2.14 (1.51, 3.02)*
Stool (log_{10} CFU/mL)	1.95 (1.39, 2.72)†

CFU, colony-forming unit; *CI*, confidence interval; *HCW*, health care worker; *OR*, odds ratio; *VRE*, vancomycin-resistant *Enterococcus*.
*Adjusted for nasogastric tube, diarrhea, age, and time the HCW spent in the room.
†Adjusted for nasogastric tube, age, and time the HCW spent in the room.

RESULTS

Of the 100 patients enrolled, chest and antecubital fossa skin samples were available from 96 patients, perianal swabs from 94, and stool samples from 43. The demographics and clinical characteristics of the patients are presented in Table 1. The mean patient age was 61 years (SD, 13), 51 of 97 (53%) were white, 50 of 97 (52%) were men, 58 of 97 (60%) were from the medical ICU, and the median number of comorbidities was 6 (range, 0-14) as measured by the Elixhauser Index. Most patients had at least 1 invasive device (93%), with a mean of 3 devices (SD, 1.6).

We observed 479 HCW-patient interactions, of which 71 of 479 (15%) led to HCWs' glove or gown contamination with VRE. Figure 1 shows that patients who transmit VRE to HCWs have higher bacterial distributions in their perianal, skin, and stool samples. Table 2 presents the adjusted ORs and 95% CIs for HCWs' glove and gown contamination for each patient sample type, controlling for nasogastric tube, diarrhea, age, and amount of time the HCW spent in the room. In each patient sample, there is an association between increasing bacterial burden and HCWs' glove or gown contamination: adjusted OR (aOR) of 1.37 (95% CI, 1.19, 1.57) for the perianal samples, aOR of 1.95 (95% CI, 1.39, 2.72) for the stool samples, and aOR of 2.14 (95% CI, 1.51, 3.02) for the skin samples. This association between skin colonization and HCWs' glove or gown contamination did not change after adjusting for the stool bacterial burden (data not shown).

The frequency and mean bacterial burden by VRE species and patient sample type is presented in Table 3. VRE was identified in 60 of 94 perianal samples collected, ranging from 1 to 1.30×10^6 CFU/mL. Patients who were colonized with *E faecium* had an average of 2.4 log_{10} (95% CI, 0.89, 3.91) colony counts higher than those with *E faecalis*. VRE was identified on 42 of 43 collected stool samples,

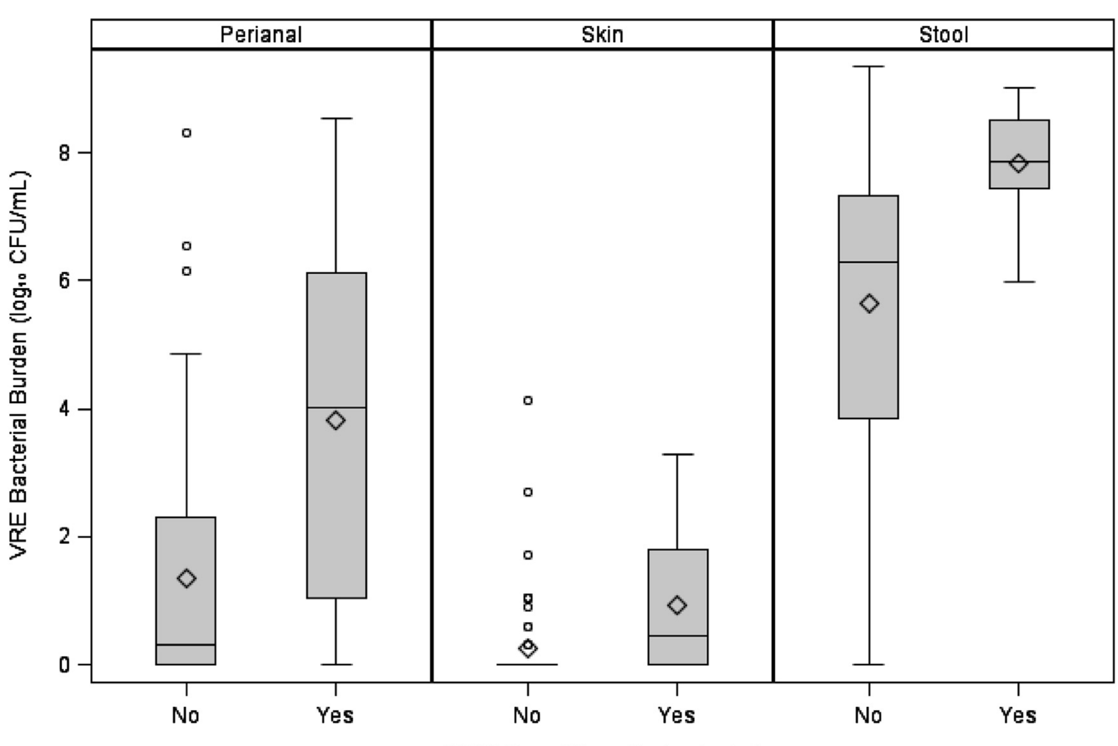

Fig 1. Bacterial distributions of each sample by transmission to HCWs' gloves or gowns.
CFU, colony-forming units; *HCW*, health care worker; *VRE*, vancomycin-resistant *Enterococcus*.

Table 3
Frequency, mean bacterial burden, and ORs for transmission to HCWs' gloves and gowns by VRE species and patient sample type

Patient sample type	VRE species*	n/Total (%)	Mean burden† (95% CI)	OR (95% CI) for transmission to HCWs‡
Perianal	*E faecium*	49/59 (83)	3.92 (3.30, 4.55)	1.98 (0.53, 7.41)
	E faecalis	10/59 (17)	1.52 (0.16, 2.89)	Reference
Stool	*E faecium*	36/40 (90)	7.09 (6.49, 7.69)	1.61 (0.37, 6.89)
	E faecalis	4/40 (10)	5.53 (3.72, 7.34)	Reference
Skin	*E faecium*	26/31 (84)	1.68 (1.31, 2.06)	9.32 (1.32, 66.03)
	E faecalis	5/31 (16)	0.72 (-0.13, 1.57)	Reference

CFU, colony-forming units; *CI*, confidence interval; *E faecalis*, *Enterococcus* faecalis; *E faecium*, *Enterococcus faecium*; *HCW*, health care worker; *OR*, odds ratio; *VRE*, vancomycin-resistant *Enterococcus*.
*Other species not listed were identified as *Enterococcus avium* and *Enterococcus casseliflavus.*
†Perianal and stool bacterial loads are expressed in $\log_{10}$ CFU/mL. Skin bacterial load is expressed in $\log_{10}$ CFU/cm^2.
‡Adjusted for bacterial burden found in that sample.

ranging from 0 to 2.25×10^9 CFU/mL. Those colonized with *E faecium* in the stool had 1.6 $\log_{10}$ (95% CI, −0.39, 3.52) higher bacterial burden than those with *E faecalis*. VRE was identified in 18 of 96 samples collected from the patients' chest, with bacterial burden ranging from 1 to 1.91×10^3 CFU/cm^2. VRE was identified on 23 of the antecubital fossa swabs obtained from 95 patients, ranging from 1 to 1.33×10^4 CFU/cm^2. Patients colonized with *E faecium* on the skin had 0.97 $\log_{10}$ (95% CI, 0.04, 1.89) higher than those with *E faecalis*. The amount of bacteria found in the stool was moderately correlated with the amount found in the perianal sample ($r = 0.56$; $P < .001$) and mildly correlated with the amount found on the skin ($r = 0.31$; $P < .001$).

VRE species were also associated with increased HCWs' glove and gown contamination, controlling for bacterial burden (Table 3). Patients colonized with *E faecium* on the skin were 9.32 (95% CI, 1.32, 66.03) times as likely to contaminate HCWs' gowns or gloves as those colonized with *E faecalis*. The odds of contamination were increased for those colonized with *E faecium* compared with *E faecalis* in the perianal and stool samples as well, but these associations did not reach statistical significance.

DISCUSSION

This study is the first of its size to quantify VRE bacterial burden and examine its role in the transmission of VRE from colonized patients to HCWs' gloves or gowns. As bacterial burden in all patient samples increases, so does the likelihood of contamination of HCWs' gloves and gowns, a potential source of transmission to other patients in the ICU. Previous studies have not examined the association between VRE species and transmission potential. Our results indicate the main driver of this transmission is likely attributable to *E faecium*, which is associated with higher colony counts than *E faecalis*. Patients colonized with *E faecium* on their skin were 9 times as likely to transmit the bacteria to HCWs as those colonized with *E faecalis* after adjusting for bacterial burden.

Although we were only able to recover VRE from a small proportion of skin samples (27% from the antecubital fossa and 19% from the chest), there was a 114% increase in HCWs' glove and gown contamination for each $\log_{10}$ increase in skin bacterial burden. This strong association remained even after adjustment for stool bacterial burden. These results indicate that skin may be an efficient means of VRE transfer. Duckro et al[10] found that the antecubital fossa was the most efficient body site for VRE transmission. In that study, HCWs contaminated their gloves 100% of the time after touching the patient's antecubital fossa compared with 60% after contact with the patient's chest.[10] Our study also found higher bacterial burden on the patients' arms than on their chests. The patient's antecubital fossa is touched often by HCWs during clinical care (eg, for blood draws and blood pressure measurement), resulting in increased bacterial burden and transmission efficiency. As has been suggested previously, this area may also be a habitable environment for VRE.[5]

These results show that skin contamination increases the odds of VRE transmission and highlight the need for ICUs to invest in decontamination protocols, such as bathing with chlorhexidine gluconate (CHG), for their VRE-colonized patients. CHG bathing has been shown to be associated with reductions in the incidence of VRE acquisition and gram-positive bacteremias.[7,13,14] In 2006, Vernon et al[7] performed a single-center clinical trial comparing the effect of 3 types of bathing routines on VRE acquisition. Compared with soap and water, daily bathing of patients with 2% CHG-saturated cloths resulted in a 60% decrease in VRE acquisition, a 40% reduction of HCWs' glove contamination, and a 70% reduction in environmental contamination.[7] Furthermore, the use of CHG bathing resulted in a decrease of inguinal bacterial burden by 2.5 $\log_{10}$ colony counts.[7] The multicenter, cluster-randomized trial conducted by Climo et al[13] in 2013 similarly found that daily CHG bathing reduced overall MDRO acquisition by 23% and bloodstream infections by 28% compared with cleansing with nonantimicrobial washcloths. Bleasdale et al[14] also found that daily CHG bathing compared with soap-and-water baths leads to a reduction in hospital-acquired bloodstream infections. However, this trial and one by Noto et al[15] did not see a reduction in other HAIs such as urinary tract infection, ventilator-associated pneumonia, or *Clostridium difficile infection*. There was no reduction in incidence of bloodstream infections either, although duration of the CHG intervention was only 10 weeks compared with the 24+ weeks in other trials.[15] The longer duration of the above trials[7,13,14] may have reduced colonization pressure in the ICU by reducing the amount of bacteria in the environment over time. Patients in the medical and surgical ICUs in this study were bathed with CHG once every 24 hours per hospital policy. The results of the preceding trials imply that the low levels of patient skin contamination in our study may be owing to frequent CHG bathing.

Our results indicate that the association with perianal bacterial burden and transmission to HCWs is not as strong as the association seen in the stool or skin samples. The odds of HCWs' glove or gown contamination increase by 37% for each $\log_{10}$ increase in VRE isolated from the perianal swab. Even though a greater number of colonies were isolated from the perianal sample than from the skin, this area is likely not often accessed by HCWs for routine procedures in the ICU. We found a nearly a 2-fold increase in the odds of HCWs' contamination for each $\log_{10}$ increase in stool bacterial burden. Large concentrations of VRE in the stool previously have been found to correlate with skin and environmental contamination.[16]

Potential limitations of this study include the fact that we sampled only a portion of the gloves and gowns instead of using alternative methods to culture the entire surface area. The transmission rate of VRE may be much higher than we were able to detect. However, we did culture from areas most likely to come in contact with the patient (glove fingers and gown arms), which are most likely involved in transmission. In addition, we did not quantify the bacterial burden recovered from the HCWs' gloves and gowns. As such, we do not know how much VRE was transferred to HCWs or what amount of VRE is needed for transmission to future patients. But had these HCWs not been wearing personal protective equipment (ie, gowns and gloves), they would have had VRE on their hands and clothing, which could lead to subsequent transmission to other patients. This study was conducted at a single site and in 2 ICUs. Transmission of VRE between patients and HCWs may vary in other acute care settings owing to differences in patient care practices. However, these findings will likely be generalizable to other large academic hospitals and ICUs with a similar patient case mix. Furthermore, the findings of this study may not be generalizable to other organisms considering that transmission mechanisms may differ among pathogens.

As far as we know, this study is the first of its size to study the role of VRE bacterial load in transmission to HCWs. We only sampled from ICUs in our hospital that conducted active surveillance to minimize selection bias. Clinical cultures are often ordered when a patient shows clinical signs and symptoms of an infection. Bias may be introduced if patients identified from clinical cultures are included, because these patients may have different risk factors for transmission than patients identified by surveillance cultures. The former patients may be sicker, may have greater number of comorbidities and devices, and may be higher transmitters than patients identified through surveillance cultures.

This study demonstrates the role that bacterial burden plays in transmission. In the absence of active surveillance, most VRE-colonized patients are unknown to HCWs. Those with higher bacterial burden are more likely to transmit VRE to HCWs in the absence of personal protective equipment. The role of *E faecium* in transmission should be explored further. These results also have major implications for infection prevention practices that aim to lower VRE levels and decrease transmission. Examples include increased CHG bathing for VRE-colonized patients in the ICU or for future work on alteration of the human microbiome that could lower levels of VRE colonization.

Acknowledgments

We thank the study participants, without whom this research would not be possible; Lisa Pineles for her help with the study logistics; Shirley Goodman and Alyssa Sbarro for their help with data collection; and Nicole Karikari, Licheng Zhao, and Gwen Paszkiewicz for specimen processing.

References

1. Sievert DM, Ricks P, Edwards JR, et al. Antimicrobial-resistant pathogens associated with healthcare-associated infections: summary of data reported to the National Healthcare Safety Network at the Centers for Disease Control and Prevention, 2009-2010. Infect Control Hosp Epidemiol 2013;34:1-14.
2. Weiner LM, Webb AK, Limbago B, Dudeck MA, Patel J, Kallen AJ, et al. Antimicrobial-resistant pathogens associated with healthcare-associated infections: Summary of data reported to the national healthcare safety network at the centers for disease control and prevention, 2011-2014. Infect Control Hosp Epidemiol 2016;37:1288–1301.
3. Centers for Disease Control and Prevention. Antibiotic resistance threats in the United States. Available from: https://www.cdc.gov/drugresistance/pdf/ar-threats-2013-508.pdf . Accessed April 12, 2018.
4. Shadel BN, Puzniak LA, Gillespie KN, Lawrence SJ, Kollef M, Mundy LM. Surveillance for vancomycin-resistant enterococci: type, rates, costs, and implications. Infect Control Hosp Epidemiol 2006;27:1068-75.
5. Beezhold DW, Slaughter S, Hayden MK, Matushek M, Nathan C, Trenholme GM, et al. Skin colonization with vancomycin-resistant enterococci among hospitalized patients with bacteremia. Clin Infect Dis 1997;24:704-6.
6. Noskin GA, Stosor V, Cooper I, Peterson LR. Recovery of vancomycin-resistant enterococci on fingertips and environmental surfaces. Infect Control Hosp Epidemiol 1995;16:577-81.
7. Vernon MO, Hayden MK, Trick WE, Hayes RA, Blom DW, Weinstein RA, et al. Chlorhexidine gluconate to cleanse patients in a medical intensive care unit: the effectiveness of source control to reduce the bioburden of vancomycin-resistant enterococci. Arch Intern Med 2006;166:306-12.
8. Hayden MK, Blom DW, Lyle EA, Moore CG, Weinstein RA. Risk of hand or glove contamination after contact with patients colonized with vancomycin-resistant enterococcus or the colonized patients' environment. Infect Control Hosp Epidemiol 2008;29:149-54.
9. Morgan DJ, Rogawski E, Thom KA, Johnson JK, Perencevich EN, Shardell M, et al. Transfer of multidrug-resistant bacteria to healthcare workers' gloves and gowns after patient contact increases with environmental contamination. Crit Care Med 2012;40:1045-51.
10. Duckro AN, Blom DW, Lyle EA, Weinstein RA, Hayden MK. Transfer of vancomycin-resistant enterococci via health care worker hands. Arch Intern Med 2005;165:302-7.
11. Snyder GM, Thom KA, Furuno JP, Perencevich EN, Roghmann MC, Strauss SM, et al. Detection of methicillin-resistant Staphylococcus aureus and vancomycin-resistant enterococci on the gowns and gloves of healthcare workers. Infect Control Hosp Epidemiol 2008;29:583-9.
12. Quan H, Sundararajan V, Halfon P, Fong A, Burnand B, Luthi JC, et al. Coding algorithms for defining comorbidities in ICD-9-CM and ICD-10 administrative data. Med Care 2005;43:1130-9.
13. Climo MW, Yokoe DS, Warren DK, Perl TM, Bolon M, Herwaldt LA, et al. Effect of daily chlorhexidine bathing on hospital-acquired infection. N Engl J Med 2013;368:533-42.
14. Bleasdale SC, Trick WE, Gonzalez IM, Lyles RD, Hayden MK, Weinstein RA. Effectiveness of chlorhexidine bathing to reduce catheter-associated bloodstream infections in medical intensive care unit patients. Arch Intern Med 2007;167:2073-9.
15. Noto MJ, Domenico HJ, Byrne DW, Talbot T, Rice TW, Bernard GR, et al. Chlorhexidine bathing and health care-associated infections: a randomized clinical trial. JAMA 2015;313:369-78.
16. Sethi AK, Al-Nassir WN, Nerandzic MM, Donskey CJ. Skin and environmental contamination with vancomycin-resistant enterococci in patients receiving oral metronidazole or oral vancomycin treatment for Clostridium difficile-associated disease. Infect Control Hosp Epidemiol 2009;30:13-7.