Infection Control Strategies for Methicillin-Resistant \textit{Staphylococcus aureus} and Vancomycin-Resistant Enterococcus: What Is the Evidence?

Laraine L. Washer, MD, and Carol E. Chenoweth, MD

Abstract

- **Objective:** To describe the evidence supporting infection control strategies for methicillin-resistant \textit{Staphylococcus aureus} (MRSA) and vancomycin-resistant enterococci (VRE) in acute care hospitals.
- **Methods:** Review of current guidelines and literature on control of MRSA and VRE.
- **Results:** Contact precautions have been recommended to reduce transmission of MRSA and VRE. Active surveillance for colonized patients along with contact precautions have been effective at decreasing rates of MRSA and VRE in some settings. However, well-designed studies supporting the use of individual strategies alone have not been performed.
- **Conclusion:** More studies are needed to clarify the efficacy of interventions for preventing spread of MRSA and VRE in hospitals.

Hospital-acquired infections due to resistant gram-positive pathogens including methicillin-resistant \textit{Staphylococcus aureus} (MRSA) and vancomycin-resistant enterococci (VRE) species pose increasing risks to patient safety in many hospitals. The recent appearance of vancomycin-intermediate \textit{S. aureus} (VISA) and vancomycin-resistant \textit{S. aureus} (VRSA) is an additional threat. The economic and medical impact of nosocomial infections due to these resistant organisms is substantial. Understanding and implementing effective evidence-based infection control measures is paramount to successful control of emergence and spread of these resistant pathogens.

Scope of the Problem

MRSA and VRE infections are global, but there are important variations in prevalence within hospitals and across countries. Rates of these resistant strains are higher in intensive care units (ICUs) and in hematology/oncology units. Among U.S. National Nosocomial Infection Surveillance (NNIS) system–participating hospitals in 2003, 59.5% of \textit{S. aureus} infections in ICU patients were MRSA, an 11% increase compared with 1998 to 2002 [1]. Rates of MRSA also vary substantially among European countries. A 2001 study of 3051 staphylococcal isolates from 25 European teaching hospitals as part of the SENTRY study found that an average of 25% of \textit{S. aureus} isolates were MRSA, but there was wide variability among European countries. Portugal and Italy had the highest rates (43%–58% MRSA), while the Netherlands had the lowest rate (2% MRSA) [2]. VISA was first reported in clinical isolates in 1997, and now there are multiple reports of VISA throughout the world as well as a smaller number of VRSA isolates in the United States [3].

Prevalence of VRE has been steadily increasing in the United States. In 2003, 28.5% of enterococcal infections in ICUs in the NNIS system were vancomycin-resistant [1]. Prevalence of VRE in other areas of the world is still sporadic. SENTRY data from 1997 to 1999 reported 24% to 27% of U.S. strains of enterococcus were resistant to vancomycin, while less than 3% of enterococcal isolates from Canada, Latin America, Europe, and Asia were vancomycin-resistant [2]. In addition, 2.7% of ICU patients in 1 study were found to be co-colonized with MRSA and VRE, and these patients comprise the substrate for potential VRSA cases [4].

Methicillin resistance has not been associated with increased virulence. However, MRSA infections have been associated with higher mortality than methicillin-susceptible \textit{S. aureus} (MSSA) infections. In a 2003 meta-analysis, MRSA bacteremia was associated with a higher mortality rate than MSSA bacteremia [5]. A recent study of infections in vascular surgery patients found that MRSA infection was an independent risk factor for in-hospital death and was associated with longer length of stay when compared with patients with MSSA infections [6]. The German Nosocomial Infection Surveillance System of 274 ICUs reported that MRSA pneumonias and primary bloodstream infections were associated with higher mortality than pneumonias and bloodstream infections due to MSSA [7].

Similarly, attributable mortality is higher among patients...

From the Department of Internal Medicine, Division of Infectious Diseases, University of Michigan Health System, Ann Arbor, MI.
with VRE bacteremia compared with patients with no bacteremia or vancomycin-susceptible enterococcal bacte-
remia [8–11]. Reasons for the increased mortality related to 
infections with MRSA and VRE are primarily due to lack 
of detection of resistance and delays in appropriate therapy 
[12,13]. However, the inferior outcomes observed in patients 
with resistant strains emphasizes the need for control of 
transmission of these organisms [14,15].

Epidemiology of MRSA and VRE in Acute Care Hospitals

Reservoirs

Patients who are colonized or infected are the primary 
reservoirs of MRSA and VRE in the hospital setting. MRSA 
colonizes the nares, skin, and the perineum of infected and 
healthy persons. Whereas S. aureus nasal colonization is 
found in 29% to 37% of the general population [16–18], MRSA 
nasal colonization is found in only 0.8% [17]. Prevalence of 
MRSA nasal colonization in hospitalized patients is higher 
than in the general population. For example, 2.7% of patients 
admitted to Emory University were colonized with MRSA 
at hospital admission [19], and 6.9% of patients admitted to 
14 French ICUs were colonized with MRSA [20]. Open 
wounds may also act as persistent reservoirs [21].

Enterococcus species are normal commensal inhabitants 
of the gastrointestinal tract, and this is the major reservoir 
for resistant strains in hospitals. Nearly all persons with

VRE bacteremia have gastrointestinal colonization with 
VRE [22]. VRE has also been found colonizing skin, urine, 
and wounds [23,24]. VRE may survive for weeks on hospital 
equipment and the hospital environment, serving as reser-
voirs for transmission [25–28].

Transmission

The major route of transmission of both MRSA and VRE is 
via the hands of hospital personnel. VRE and MRSA have 
been isolated from the hands of health care workers caring 
for infected or colonized patients [27,29]. Contamination of 
health care workers’ clothing may also facilitate transmis-
sion of resistant organisms from patient to patient. Thirty-
seven percent of health care workers’ gowns were found to 
be contaminated with VRE after caring for a patient with 
VRE infection, and 65% of health care workers’ gowns or 
clothes were contaminated with MRSA after caring for a 
patient with MRSA in wounds or urine [26,30]. The role of 
inanimate objects, such as thermometers and other medical 
equipment, in transmission of resistant strains has recently 
been recognized [25–27,31,32].

Risk Factors for Colonization and Infection

In general, risk factors for MRSA and VRE colonization 
and infection overlap. Hospitalized patients with increased 
severity of illness, decreased immune status, and lack of 
integrity of skin also require more hands-on patient care, 
undergo more invasive procedures, and are at increased risk 
for MRSA or VRE colonization or infection. Because many 
of the underlying risk factors are the same, patients are often 
co-colonized with MRSA and VRE [4].

Specific risk factors for MRSA colonization and infection 
include age, ICU care, duration of hospitalization, invasive 
procedures, prior antibiotic exposure, indwelling devices, 
diabetes, wounds, and pressure ulcers. Patients with HIV, 
cystic fibrosis, liver failure, intravenous drug use, rheuma-
tologic diseases, and chronic dermatitis are also at increased 
risk for MRSA colonization and infection (Table 1) [21,33–38].

“Colonization pressure” represents the overall ICU or hospital 
burden of MRSA and has been shown to independently and 
strongly influence acquisition of MRSA in the ICU [39].

Risk factors for VRE acquisition and infection include 
severity of illness, residence in long-term care facilities, 
prolonged hospitalization, proximity to other unisolated 
VRE patients, and care by a nurse who cares for other pa-
tients with VRE. Patients who have undergone abdominal 
surgery or liver transplantation or who have renal failure, 
malignancy, neutropenia, HIV, urethral catheterization, or 
burns are also at increased risk for VRE acquisition and 
infection. Colonization pressure has been found to strongly 
affect acquisition of VRE [40]. In addition, antibiotic ex-
posure, particularly to cephalosporins, vancomycin, and

<table>
<thead>
<tr>
<th>MRSA</th>
<th>VRE</th>
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<tr>
<td>Colonization pressure</td>
<td>Prolonged hospitalization</td>
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<tr>
<td>Duration of hospitalization</td>
<td>Proximity to VRE patients</td>
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<tr>
<td>Residence in nursing home</td>
<td>Nurse caring for VRE patient</td>
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<tr>
<td>Intensive care unit care</td>
<td>Severity of illness</td>
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<tr>
<td>Prior antibiotic use</td>
<td>Malignancy</td>
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<tr>
<td>Indwelling devices</td>
<td>Neutropenia</td>
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<tr>
<td>Neonates, elderly</td>
<td>Corticosteroids</td>
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<tr>
<td>Diabetes</td>
<td>Burns</td>
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<tr>
<td>Trauma</td>
<td>Abdominal surgery</td>
</tr>
<tr>
<td>Burns</td>
<td>Liver transplantation</td>
</tr>
<tr>
<td>Dialysis</td>
<td>HIV</td>
</tr>
<tr>
<td>Wounds or pressure ulcers</td>
<td>Intravenous drug use</td>
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</tbody>
</table>
| HIV | Prior antibiotic use (i.e., cephalo-
| Cystic fibrosis | sporins, vancomycin, anti-
| Liver transplantation | an-aerobic) |
| Intravenous drug use | Renal failure |
| Rheumatologic disease | Urethral catheterization |
| Chronic dermatitides | |

Table 1. Risk Factors for Methicillin-Resistant Staphylococcus aureus (MRSA) and Vancomycin-
Resistant Enterococci (VRE) Colonization or Infection
antianaerobic antibiotics, is associated with increased risk of VRE colonization and infection (Table 1) [8,11,24,41].

**Infection Control Strategies**

The Centers for Disease Control’s Hospital Infection Control Practices Advisory Committee (HICPAC) and the Society for Healthcare Epidemiology of America (SHEA) have published guidelines recommending optimal approaches to infection control of resistant bacteria including MRSA and VRE [14,15]. These guidelines recommend appropriate antibiotic use, education of medical staff, and screening of high-risk patients followed by infection control practices including use of gloves and gowns when caring for colonized or infected patients with MRSA and VRE (Table 2).

Use of these infection control strategies has been shown to reduce transmission of MRSA and VRE in endemic and epidemic settings [42–46]. However, determining the effectiveness of individual strategies can be challenging because studies are often observational in nature and employ multiple simultaneous interventions. Infection control trials frequently evaluate colonization outcomes rather than morbidity and mortality data. In addition, many infection control trials are hampered by the historically poor adherence to infection control practices [47].

### Hand Hygiene

Hand hygiene is the cornerstone of infection control measures for preventing transmission of microorganisms from patient to patient. While handwashing is recommended after all patient contacts, hand hygiene compliance is universally poor, averaging around 40%, and worst among physicians, with compliance at 30% or less [14,48]. Any effort at control of MRSA and VRE transmission should emphasize increased compliance with hand hygiene. Gloving decreases VRE and MRSA acquisition on hands but does not completely prevent contamination of hands; therefore, handwashing is recommended after removal of gloves [49]. Antiseptic soap and alcohol-based washes are more effective than bland soap in removing resistant microorganisms from the hands of hospital personnel and are recommended in hospital settings to promote proper hand hygiene [50,51].

### Contact Precautions

In addition to standard hand hygiene, contact precautions have been used to prevent spread of VRE and MRSA in the hospital setting. Contact precautions include use of gloves, gowns, and masks while caring for colonized or infected patients. Cohorting of colonized patients or health care workers has been applied in some settings [28,52]. When implemented after a program of active surveillance, there is evidence that the combined effect of contact precautions as a whole can reduce MRSA transmission. However, the independent effects of individual components of contact precautions (gowns, gloves, masks, cohorting) without use of active surveillance have been less well studied. A 1982 study by Thompson et al [53] found that when contact precautions were initiated only when MRSA was found passively by clinical culture results, MRSA prevalence increased over time.

Some studies support the use of contact precautions to decrease MRSA transmission once MRSA is identified by active surveillance. Jernigan and colleagues [54] demonstrated that the risk of MRSA transmission was reduced 16-fold when MRSA patients were cared for in contact isolation as compared with use of standard precautions. A Dutch ICU showed a 38-fold higher frequency of transmission from patients with MRSA colonization who were not cared for using contact precautions compared with MRSA-colonized patients cared for with contact precautions [55]. Denmark and Finland [56] have kept MRSA to less than 2% of *S. aureus* isolates using rigorous infection control policies that include strict application of contact precautions, active surveillance, and decolonization.

There are multiple studies documenting control of VRE outbreaks using contact precautions as part of infection control strategy [57]. Montecalvo and colleagues [52] found that institution of contact precautions in an oncology ward resulted in significant decreases in VRE bloodstream infections when compared with historical use of standard precautions. However, there are conflicting data regarding whether the use of gowns in addition to gloves is necessary.

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**Table 2. Summary of Current Recommendations for Control of Methicillin-Resistant *Staphylococcus aureus* and Vancomycin-Resistant Enterococci in Hospitals**

<table>
<thead>
<tr>
<th>Hand hygiene</th>
<th>Antiseptic soap or waterless antiseptic</th>
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<tbody>
<tr>
<td></td>
<td>Before and after patient contact</td>
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<tr>
<td>Contact precautions</td>
<td>Private room or patient cohorting</td>
</tr>
<tr>
<td></td>
<td>Gloves</td>
</tr>
<tr>
<td></td>
<td>Gowns</td>
</tr>
<tr>
<td></td>
<td>Masks*</td>
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<tr>
<td>Single patient equipment</td>
<td>Blood pressure cuffs</td>
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<tr>
<td>Stethoscopes</td>
<td></td>
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<tr>
<td>Active surveillance for high-risk patients/units</td>
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<tr>
<td>Antibiotic stewardship</td>
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</tbody>
</table>

Data from references 14 and 15.

*The Centers for Disease Control and Prevention recommends masks to be used for vancomycin-intermediate *S. aureus* and vancomycin-resistant *S. aureus* only.*
for the control of VRE transmission. Slaughter et al [58] found that the universal use of gowns and gloves was no better than the universal use of gloves alone in preventing rectal colonization in a medical ICU where VRE was endemic. However, 4 other studies have shown lower rates of VRE acquisition when gowns were used in addition to gloves [58–62]. For example, Puzniak et al [61] found that gowns were protective in reducing VRE acquisition in a medical ICU with a high VRE colonization pressure (Table 3).

The role of masks in contact precautions for MRSA has not been studied adequately. Masks have been associated with decreased MRSA nasal colonization in health care workers during MRSA outbreaks, but no studies have shown an independent effect of masks on MRSA infection rates in hospitals [14,54]. Masks are recommended when caring for patients with VISA or VRSA to prevent nasal colonization of health care workers with these highly resistant organisms but are not uniformly recommended when caring for patients with MRSA. Because VRE is not associated with nasal carriage, masks are not recommended while caring for patients colonized with VRE.

The use of single patient rooms or patient cohorting for MRSA and VRE is recommended by existing guidelines and is supported by data demonstrating contamination of patient environment and air as discussed above [14,15]. The German KISS study found that isolation in private rooms or cohorts was protective at preventing transmission of MRSA [63]. Cohorting hemodialysis patients with MRSA colonization has been shown to be modestly effective at decreasing MRSA infection rates [28]. Only 1 study from the United Kingdom found that there was no change in transmission of MRSA using isolation/cohorting compared with a form of universal barrier precautions [64]. For VRE infection or colonization, cohorting on a single ward with dedicated nursing staff and patient-care equipment decreased 1 hospital’s VRE prevalence from 8.1% to 4.7% [65].

The decision to remove patients with prior MRSA or VRE infection from isolation is difficult. Criteria for discontinuation of contact precautions should be rigorous, as colonization often persists throughout hospitalization, especially with re-exposure to antibiotics. The Centers for Disease Control and Prevention recommendation for 3 sequential negative cultures for VRE from multiple body sites at least 1 week apart before removing patients from VRE contact precautions [15] is supported by data showing that after 3 sequential negative cultures, 95% of patients remain culture-negative for VRE [66]. However, some authorities suggest that precautions be continued for patients with VRE during their entire hospitalization given a high risk of recolonization and imperfect sensitivity of culture techniques [67,68]. Hospital information systems may be used to identify colonized patients who are readmitted and require rescreening and contact isolation [69].

Despite the probable benefits of contact precautions for the prevention of transmission of resistant organisms, there may be negative effects of isolation of patients. Health care workers are less likely to enter the rooms of patients in contact isolation and attending physicians are less likely to examine patients in contact isolation compared with patients not in contact isolation [70,71]. Stelfox and colleagues [72] found that patients isolated for MRSA colonization or infection were generally less satisfied with their treatment, had more preventable adverse events (pressure ulcers, falls, electrolyte disorders), and had less documentation of care. There were no significant differences in mortality or adverse events involving medical procedures or medications.

### Active Surveillance for Patients Colonized with MRSA or VRE

The use of contact precautions only for patients with clinically recognized MRSA or VRE infections fails to address unrecognized colonized patients who may serve as reservoirs of transmission. Active surveillance by culturing for MRSA or VRE in those patients at high risk for colonization but without clinical evidence of infection seeks to define the reservoir for transmission. Multiple studies of infection control interventions support the use of screening or active surveillance for colonization followed by contact isolation for both MRSA and VRE [23,42–46,52]. Intensive infection control programs that include active surveillance have been successful at consistently keeping MRSA rates less than 2% of *S. aureus* isolates [56]. Conclusions drawn

### Table 3. Studies on Effect of Gowns on Vancomycin-Resistant Enterococci (VRE) Transmission

<table>
<thead>
<tr>
<th>Study</th>
<th>Interventions</th>
<th>VRE Acquisition</th>
<th>Comments</th>
</tr>
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<tbody>
<tr>
<td>Slaughter et al [58]</td>
<td>Universal gloves vs. gown/gloves in intensive care unit</td>
<td>23%–25% acquired VRE in both groups</td>
<td>Compliance: 79% gowns; 62% no gowns</td>
</tr>
<tr>
<td>Puzniak et al [61]</td>
<td>Gowns vs. no gowns before and after patient contact</td>
<td>9.1/1000 patient-days vs. 19.6/1000 patient-days</td>
<td>VRE risk factors more prevalent in no-gown group</td>
</tr>
<tr>
<td>Srinivasan et al [62]</td>
<td>Gown/gloves vs. gloves before and after patient contact</td>
<td>1.8/100 patient-days vs. 3.78/100 patient-days</td>
<td>Universal on admit until VRE screen negative</td>
</tr>
</tbody>
</table>

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from this “search and isolate” approach are limited, as they are based on observational studies and not randomized controlled interventions. Girou and colleagues [43] found that selective screening for MRSA nasal carriage at time of admission to high-risk areas may contribute to identification of a substantial proportion of cases of MRSA and result in earlier implementation of infection control measures. A high endemic level of MRSA in an Italian hospital was decreased significantly from 46% to 17% of blood isolates with an intervention of active surveillance cultures to identify MRSA-colonized patients followed by isolation using contact precautions [42]. Passive surveillance using clinical culture results alone followed by contact precautions has not been shown to be effective in controlling MRSA transmission [47, 53]. Thompson and colleagues [53] demonstrated that the addition of active surveillance to contact precautions was able to decrease endemic levels of MRSA in a U.S. hospital when passive surveillance followed by contact precautions had previously failed to control MRSA rates.

Multiple studies evaluating the role of active surveillance for VRE with perirectal swabs or stool cultures in high-risk patients have shown reduced VRE bacteremia rates, more monoclonal populations, and decreased hospital-wide VRE incidence rates [46, 52, 73-74]. Hachem and colleagues [75] found that surveillance of VRE with stool cultures in high-risk patients with hematologic malignancy followed by contact isolation resulted in an eightfold decrease in VRE bacteremias despite a high level of vancomycin use. Mathematical models assessing the effects of active surveillance on VRE provide additional data in the absence of randomized trials and suggest that active surveillance followed by contact precautions may yield a 39% to 65% reduction in VRE colonization [76, 77].

As is the case for other contagious pathogens, there have been no completed randomized controlled trials to date regarding the efficacy of active surveillance for MRSA and VRE. The National Institutes of Health's Bacterial and Mycology Study Group is currently conducting a multicenter randomized trial comparing active surveillance followed by enhanced standard precautions versus enhanced hand hygiene compliance to reduce colonization and infection caused by VRE and MRSA in ICUs. This study should provide additional information on the efficacy of screening and contact precautions for the prevention of MRSA and VRE transmission.

Control of Environment
MRSA and especially VRE may heavily contaminate the environment of colonized or infected patients. Between 69% and 73% of hospital rooms housing patients colonized or infected with MRSA have some environmental contamination [30]. VRE and MRSA have frequently been isolated from environmental surfaces including fabric-covered furniture, bed rails, and privacy drapes in patient’s rooms and have the ability to survive on dry surfaces for up to several months [27, 31, 32]. Appropriate cleaning of the environment is important to eliminate environmental reservoirs. In addition, several studies have shown that stethoscopes, blood pressure cuffs, and thermometers can become contaminated and act as a vehicle of transmission for VRE or MRSA [25, 26]. Therefore, it is recommended that patients in contact isolation have designated equipment including stethoscopes and blood pressure cuffs [14]. Routinely disinfecting equipment with 70% isopropyl alcohol has been shown to decrease bacterial counts on equipment that cannot be limited to single patient use. Routinely used disinfectants are active against both VRE and MRSA [78].

Eradication of Reservoirs
Mupirocin is up to 97% effective in transiently reducing S. aureus nasal carriage. Targeted prophylaxis of S. aureus nasal carriers before cardiac and orthopaedic procedures has shown reductions in S. aureus infections and is a promising approach [79, 80]. Randomized trials and a meta-analysis of intranasal mupirocin in hemodialysis patients colonized with MRSA have shown reductions in S. aureus infections [81, 82]. Chlorhexidine body washes have been used in combination with mupirocin to eradicate S. aureus colonization and has shown added benefit [43]. Concerns regarding mupirocin resistance, superinfection with yeast, and recurrence of MRSA colonization leave the role of decontamination unclear in endemic situations. Treatment of MRSA-colonized patients in endemic situations is not recommended but may be helpful in outbreak situations and in certain populations [83, 84].

Efforts using combinations of oral zinc, bacitracin, and doxycycline to eradicate VRE carriage in the intestinal tract have been largely ineffective [85]. Ramoplanin, a nonabsorbable agent with bactericidal activity against gram-positive organisms, is effective in temporarily decreasing VRE colonization of the intestinal tract and studies are in progress on its effects on decreasing VRE bacteremia [86].

Antibiotic Stewardship
Prior exposure to antibiotics is a consistent risk factor for acquisition of MRSA and VRE, yet antibiotics are often inappropriately used for prophylactic or empiric reasons and for inappropriate durations. A Veterans Affairs Medical Center study found that 70% of vancomycin use was inappropriate according to HICPAC guidelines [87]. Several studies have shown decreases in MRSA prevalence following antibiotic restriction programs with simultaneous changes in infection control policies. Landman and colleagues [88] found that changes in a hospital formulary to decrease use of cephalosporins, imipenem, clindamycin, and vancomycin resulted...
in a reduced number of patients with MRSA infections. Changing from a third-generation cephalosporin to a first-generation cephalosporin for perioperative prophylaxis resulted in decreased MRSA rates in a Japanese hospital [89].

Many hospitals have instituted pharmacy formulary changes aimed to decrease prevalence of VRE colonization with conflicting results. One hospital’s formulary switch from ceftazidime to piperacillin/tazobactam resulted in a two thirds decrease in VRE colonization [60]. Another hospital’s efforts to decrease vancomycin and cephalosporin use was found to decrease VRE fecal colonization from 47% to 15% [90]. In contrast, Stiefel’s [91] retrospective evaluation of 4 academic medical centers did not find a predictable relationship between antienterococcal β-lactam use and VRE colonization rates. Lautenbach and colleagues [92] found that limiting vancomycin and cephalosporin use did not prevent an overall increase in VRE colonization in their hospital; however, they did note a significant association between clindamycin use and VRE colonization, suggesting that avoiding unnecessary use of antianaerobic antibiotics may be helpful in controlling VRE. In the future, specific antibiotic selection may be guided by the VRE colonization status of the patient.

Costs
There are obvious additional costs associated with nosocomial infections from resistant organisms including MRSA and VRE. One author found an attributable cost of $27,083 for MRSA blood stream infections compared with $9661 for MSSA bloodstream infections [93]. A meta-analysis of attributable costs of health care-associated infections found that there was a $35,367 attributable cost to MRSA infections [94]. VRE bloodstream infection was associated with excess hospital costs of $27,000 to $79,589 when compared with vancomycin-sensitive enterococcal bloodstream infection [11,95].

Given the significant excess costs attributable to nosocomial infections with MRSA and VRE, infection control approaches can be cost-effective overall. Muto et al [74] found perirectal surveillance cultures for controlling VRE to be cost-effective given the excess costs of VRE bacteremia. Montecalvo et al [96] implemented a 15-component VRE infection control program for a net 1-year savings of almost $190,000. Papia et al [97] calculated that early identification of MRSA-colonized patients that limited nosocomial transmission of MRSA to as few as 6 patients would be cost-effective.

Conclusions
MRSA and VRE infections acquired in hospitals are significant public health and patient safety concerns. Infection control measures as currently implemented have not halted the spread of MRSA and VRE in most hospitals. Current barriers to implementation of effective infection control strategies include practical issues of costs and hospital resources including private rooms. Concern regarding decreased quality of patient care associated with contact isolation also limits implementation. The challenge is to design rigorous and evidence-based infection control strategies that are simultaneously practical and cost-effective.

Data behind use of active surveillance in screening and contact precautions are substantial and compelling but lack support by randomized controlled trials. The use of contact precautions alone without identifying the reservoir of transmission has been disappointing in terms of control. Randomized controlled trials are needed to clarify the role of active surveillance to prevent spread of MRSA and VRE.

Corresponding author: Carol E. Chenoweth, MD, University of Michigan Health System, 3116 Taubman Ctr., 1500 E. Medical Center Dr., Ann Arbor, MI 48109, cchenow@umich.edu.

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