case patients, 1 of 58 (1.7%) did not have a urinary catheter present. Contaminated urine cultures (≥3 organisms present) were misclassified as infections in 6 of 58 cases (10.3%), and in 5 of 58 cases (8.6%), no urine culture was obtained. Lastly, in 15 of 58 cases (25.9%), bacteriuria was present (1 or 2 organisms), but the colony count did not reach the NHSN metric threshold of ≥ 100,000 CFU/mL.

The study period comprised 233,921 patient days. The CAUTI rate was 0.24 CAUTIs per 1,000 patient days using the ICD-10-CM metric; this rate was 0.18 when POA cases were eliminated. The CAUTI rate was 0.20 per 1,000 patient days using the NHSN metric.

The NHSN CAUTI metric and the ICD-10-CM CAUTI-like code produce widely discrepant results. Even when ICD-10 cases that were POA were removed to better align with the NHSN criteria, the sensitivity of the ICD-10 metric was only 2.4%. Importantly, no patient safety indicator from AHRQ is available for CAUTI as there is for central venous catheter-related bloodstream infection.[10] This was the primary reason that we used the administrative code (ICD-10-CM) to compare to NHSN surveillance data for detecting CAUTI.

Our results demonstrate that updating ICD-9-CM with more codes to produce ICD-10-CM did not improve the ability of administrative data to identify CAUTIs. The date of the event is an important element used to meet an NHSN site-specific infection criterion, including CAUTI, and that is one reason that administrative data fail to accurately identify cases of HAI.

This study has several limitations. First, it was performed in a single medical center. In addition, we did not review the negative cases via either method, and we assumed that traditional surveillance (NHSN) is the gold standard surveillance method. Therefore, it was not possible to calculate the specificity because our aim was to compare only NHSN and ICD-10-CM CAUTI identified cases. Given that CAUTI is a relatively rare event, we can assume that the specificity of the ICD-10-CM metric is high.

In summary, we found that ICD-10-CM has an extremely low sensitivity for detecting CAUTI cases; it failed to detect 98.3% of the infections at our institution. Almost all cases identified via ICD-10-CM did not fulfill the NHSN criteria. Thus, administrative coding for this HAI is not a useful tool for use as a surveillance method.

ACKNOWLEDGMENTS

Financial support: No financial support was provided relevant to this article.

Potential conflicts of interest: All authors report no conflicts of interest relevant to this article.

Alexandre R. Marra, MD;[1,2]
Mufareh Alkatheri, MD;[1,3]
Michael B. Edmond, MD, MPH, MPA[1]

Affiliations: 1. Office of Clinical Quality, Safety and Performance Improvement, University of Iowa Hospitals and Clinics, Iowa City, Iowa; 2. Division of Medical Practice, Hospital Israelita Albert Einstein, São Paulo, Brazil; 3. Division of Internal Medicine, National Guard Health Affairs, King Saud Bin Abdulaziz University for Health Science, Riyadh, Saudi Arabia.

Address correspondence to Alexandre Rodrigues Marra, MD, University of Iowa Hospitals and Clinics, C51 GH, 200 Hawkins Drive, Iowa City, IA 52242 (alexandre-rodriguesmarra@uiowa.edu).

Infect Control Hosp Epidemiol 2017;38:506–507

 0899-823X/2017/3804-0027. DOI: 10.1017/ice.2016.335

REFERENCES

1. Klevens RM, Edwards JR, Richards CL Jr, et al. Estimating healthcare-associated infections and deaths in US hospitals, 2002. *Public Health Rep* 2007;122:160.
2. Horan TC, Andrus M, Dudeck MA. CDC/NHSN surveillance definition of health care-associated infection and criteria for specific types of infections in the acute care setting. *Am J Infect Control* 2008;36:309–332.
3. Cass AL, Kelly JW, Probst JC, Addy CL, Mckeown RE. Identification of device-associated infections utilizing administrative data. *Am J Infect Control* 2013;41:1195–1199.
4. Stevenson KB, Khan Y, Dickman J, et al. Administrative coding data, compared with CDC/NHSN criteria, are poor indicator of healthcare-associated infections. *Am J Infect Control* 2008;36:155–164.
5. Stamm AM, Bettacchi CJ. A comparison of 3 metrics to identify healthcare associated infections. *Am J Infect Control* 2012:688–691.
6. Goto M, Ohl ME, Schweizer ML, Perencevich EN. Accuracy of administrative code data for the surveillance of healthcare-associated infections: a systematic review and meta-analysis. *Clin Infect Dis* 2014;58:688–696.
7. van Mourik MSM, van Duijn PJ, Moons KGM, Bonten MJM, Lee GM. Accuracy of administrative data for surveillance of healthcare-associated infections: a systematic review. *BMJ Open* 2015;5:e008424.
8. 2016/17 ICD-10-CM Diagnosis Code T83.51. Infection and inflammatory reaction due to urinary catheter. International Classification of Diseases, 10th Revision, Data website. http://www.icd10data.com/ICD10CM/Codes/S00-T88/T80-T88/T83-/T83.51. Published 2016. Accessed September 8, 2016.
9. Identifying healthcare-associated infections (HAI) for NHSN surveillance. Centers for Disease Control and Prevention website. https://www.cdc.gov/nhsn/PDFs/pscManual/2PSC_IdentifyingHAIs_NHSNcurrent.pdf. Accessed November 17, 2016.
10. Marra AR, Jansen DB, Edmond MB. Utility of the central venous catheter-related bloodstream infection patient safety indicator. *Infect Control Hosp Epidemiol* 2016;37:1389–1391.

Measures to Prevent and Control Vancomycin-Resistant Enterococci: Do They Really Matter?

To the Editor—The study by Lemieux et al[1] from Ontario, Canada, and other recent studies in this journal highlight the continuing importance of vancomycin-resistant enterococci

(VRE) but also the varying approaches depending on circumstances.[2,3] Complete cessation of control measures for VRE in 4 large academic hospitals in Ontario had no significant impact on VRE bloodstream infection (BSI) rates, or carriage rates.[1] A single-center study from Buffalo, New York, in a hematology-oncology unit found no increased incidence of VRE BSI after discontinuation of contact precautions although all patients were admitted to a single room.[2] However, a systematic review of the incidence of VRE infection in the United States found that VRE continues to be an important cause of healthcare-associated infection and that, in Atlanta and Detroit, there have been increases in recent years associated with mortality, longer length of stay, and higher healthcare costs.[3]

The literature does confirm the success of interventions, either in single-center studies or in other studies, even if many of these studies are less than ideal. A bundle that included active surveillance and better environmental hygiene in Singapore resulted in a significant decrease in cases of VRE infection and colonization; the authors felt that VRE screening and isolation were an in important component of that bundle.[4] While there is considerable variation regarding which patients should be screened for VRE and how, in some centers screening itself often seems to have a beneficial effect.[5] This benefit may arise from increased awareness leading to better compliance with standard precautions.

Many horizontal infection prevention and control measures applied to all patients can have a beneficial impact on the VRE load. For example, chlorhexidine bathing in a stem-cell transplantation unit over a 9-year period reduced rates of VRE colonization and infection, but the potential for increased resistance to chlorhexidine was suggested.[6] In addition, the results of this before-and-after study may have been influenced by other factors. The inconsistency of research studies on VRE prevention and control have been well illustrated in a recent systematic review of measures carried out by de Angelis et al.[7] Of 549 initial studies, only 9 met the eligibility criteria for scientific rigor, and in most cases these studies described multiple interventions.[7]

Measures to prevent VRE need to be tailored to specific patient groups, the local epidemiology, the clinical setting, and other measures being undertaken to prevent infection. For example, in a well-designed interrupted time-series study and cluster-randomized trial involving 13 intensive care units in the Netherlands, improved hand hygiene and chlorhexidine body washing were the only significant factors in reducing the acquisition of antimicrobial-resistant bacteria, including VRE.[8] This study also evaluated the impact of rapid detection using molecular methods, but it did not have a significant impact in a critical care setting. Like methicillin-resistant *Staphylococcus aureus* and *Clostridium difficile,* VRE can persist for prolonged periods in significant numbers in the environment. In a 30-month, prospective, intervention study on 6 high-risk units, the additional use of hydrogen peroxide to decontamination regimens significantly reduced the chances of acquiring a multidrug-resistant organism (MDRO), but much of the explanation for the overall effect on MDROs arose from the beneficial impact on VRE acquisition.[9] Even where hydrogen peroxide is not used or is not feasible, improved environmental decontamination is important in preventing VRE and other healthcare-associated infections. This, together with enhanced hand hygiene remain essential as efforts to decolonize patients are only partially successful and probably only for limited periods of time, given the normal habitat of VRE and the complex microbiome in the gastrointestinal tract.[10]

In recent years, greater emphasis has been placed on measures to prevent *Clostridium difficile* infection and infections due to carbapenamase-producing bacteria, but a significant healthcare burden is associated with VRE, with the added concern of resistance to agents used in treatment, such as linezolid and daptomycin. Some centers would be wise to continue with active screening to monitor trends and to target additional measures, even if many of the measures to prevent and control VRE are similar to those for other nosocomial pathogens, including improved hand-hygiene, better environmental decontamination, and antibiotic stewardship. Consequently, we should be reluctant to discontinue even selective screening and contact precautions until larger studies in more diverse clinical settings are carried out.

ACKNOWLEDGMENTS

Financial support: The topic of this correspondence was not funded but arose from routine clinical and academic activity.

Potential conflicts of interest: The author receives research funding from Pfizer (Ireland) and Astellas. In recent years, he has also received lecturer or consultancy fees from Pfizer, Cepheid Novartis, Astellas, and Astra Zeneca.

Hilary Humphreys, MD, FRCPI, FRCPath[1,2]

Affiliations: 1. Department of Clinical Microbiology, the Royal College of Surgeons in Ireland, Dublin, Ireland; 2. Department of Microbiology, Beaumont Hospital, Dublin, Ireland.

Address correspondence to Hilary Humphreys, Department of Clinical Microbiology, RCSI, Education and Research Centre, Beaumont Hospital, Dublin D09 YD60, Ireland (hhumphreys@rcsi.ie).

Infect Control Hosp Epidemiol 2017;38:507–509

 0899-823X/2017/3804-0028. DOI: 10.1017/ice.2016.329

REFERENCES

1. Lemieux C, Gardam M, Evans G, et al. Longitudinal multicenter analysis of outcomes after cessation of control measures for vancomycin-resistant enterococci. *Infect Control Hosp Epidemiol* 2016:1–7.
2. Almyroudis NG, Osawa R, Samonis G, et al. Discontinuation of systematic surveillance and contact precautions for vancomycin-resistant *Enterococcus* (VRE) and its impact on the incidence of VRE *faecium* bacteremia in patients with hematologic malignancies. *Infect Control Hosp Epidemiol* 2016;37:398–403.

3. Chiang H-Y, Perencevich EN, Nair R, et al. Incidence and outcomes associated with infections caused by vancomycin-resistant enterococci in the United States: systematic literature review and meta-analysis. *Infect Control Hosp Epidemiol* 2016:1–13.
4. Fisher D, Pang L, Salmon S, et al. A successful vancomycin-resistant enterococci reduction bundle at a Singapore hospital. *Infect Control Hosp Epidemiol* 2016;37:107–109.
5. Humphreys H. Controlling the spread of vancomycin-resistant enterococci. Is active screening worthwhile? *J Hosp Infect* 2014; 88:191–198.
6. Mendes ET, Ranzani OT, Marchi AP, et al. Chlorhexidine bathing for the prevention of colonisation and infection with multidrug-resistant microorganisms in a hematopoietic stem cell transplantation over a 9-year period. Impact of chlorhexidine susceptibility. *Medicine* 2016;95:46 (e5271).
7. DeAngelis G, Cataldo MA, DeWaure C, et al. Infection control and prevention measures to reduce the spread of vancomycin-resistant enterococci in hospitalized patients: a systematic review and meta-analysis. *J Antimicrob Chemother* 2014;69:1185–1192.
8. Derde LPG, Cooper BS, Goossens H, et al. Interventions to reduce colonization and transmission of antimicrobial-resistant bacteria in intensive care units: an interrupted time series study and cluster randomized trial. *Lancet Infect Dis* 2014;14:31–39.
9. Passaretti CL, Otter JA, Reich NG, et al. An evaluation of an environmental decontamination with hydrogen peroxide vapor for reducing the risk of patient acquisition of multidrug-resistant organisms. *Clin Infect Dis* 2013;56:27–35.
10. Cheng VCC, Chen JHK, Tai JWM, et al. Decolonisation of gastrointestinal carriage of vancomycin-resistant *Enterococcus faecium*: cases series and review of literature. *BMC Infect Dis* 2014;14:514.

Food Indwelling *Clostridium difficile* in Naturally Contaminated Household Meals: Data for Expanded Risk Mathematical Predictions

To the Editor—We read with interest the study "An Evaluation of Food as a Potential Source for *Clostridium difficile* Acquisition in Hospitalized Patients," in which Kwon et al[1] made mathematical predictions on the risk of *C. difficile* infection (CDI) acquisition due to consumption of *C. difficile* (CD)–contaminated foods in one hospital setting (presumably from a single kitchen source). Although the authors tested many small-sized, mixed-meal samples (n = 910) consumed by 149 patients (median length of hospital stay, 4 days), their mathematical predictions were based on prevalence data obtained using, apparently, nonenrichment culture methods (previously tested for fecal swabs), which are suboptimal for culturing CD and foodborne pathogens from food. Furthermore, the study used a nonstandard heat-shock treatment (80°C, 10 minutes) prior to culture, which introduces a negative bias because this heat treatment has been shown recently to kill up to 75% of CD isolates in liquid media.[2] Hence, not surprisingly, Kwon et al reported a low prevalence of CD in the tested meals (0.22%). This observed prevalence was then used for mathematical modeling. With low-prevalence data, their prediction regarding causal connections between food contamination and the incidence of CDI was, reasonably, that food is an unlikely risk for CDI (<1 colonization per 1,000 admissions) in their study.

Although the study makes an important contribution to the controversial topic of whether CDIs are foodborne, their conclusion seems biased due to suboptimal CD culture methodology. Distinctive methodological imperfections, without critical interpretation, may set us back to the first studies in the 1980s, when CD was not found in hospital meals using nonenrichment methods. After decades of believing CDI was strictly nosocomial,[3] there is now solid evidence based on whole-genome sequencing of CD isolates in hospitals that less than one-third of CDIs are nosocomial, whereas most sources of exposure that result in CDIs remain unknown.[4] With such genomic hospital discoveries, and with the persistence of CDI despite immense efforts to prevent nosocomial transmission, it is not advisable to discard the most plausible source of toxigenenic CD spores (ie, food), even if some studies report negative results. Many more unbiased reports have shown that food can be a real source of CD spores of virulent or multidrug-resistant CD strains,[5] including studies of hospital meals showing 17% and 27% prevalence on cooked and uncooked meats, respectively.[6,7] Even Kwon et al reported important CD strains in food: specifically, CD spores of toxigenic PCR-ribotypes 001 in gelatin dessert and 027 in 'vegetable/bread/grain.'

Food-dwelling CD became evident as a natural source of exposure to humans in 2005 when emerging hypervirulent CD strains causing severe disease in humans in Canada and United Kingdom were unexpectedly found in food animals[8] and retail foods.[3,9] To date, no studies have addressed kitchens as complex food environments where cross contamination and cooking practices may influence the prevalence of CD at the consumer level. Here, we would like to contribute to the external validity of the Kwon study on hospital-cooked meals by reporting, for the first time, CD data for household-cooked meals. Although we did not study colonization in humans, we blindly quantified CD in household meals, and we investigated the potential for environment–food cross contamination after visiting 35 rural and urban households in Ohio (2.3 ± 1.2 visits/each; over four months). In total, 467 samples of food (collected from 188 kitchen pots or refrigerators) and 279 samples from the household environment were processed using validated food-enrichment protocols.[9] Meals, cooked, uncooked, or processed, were sampled, homogenized, centrifuged, and stored as sediments at −80°C until processing.[9] Environmental swabs (8 cm × 4 cm × 1 cm) from kitchen countertops (n = 32), sinks (n = 56), refrigerator shelves (n = 59), gloves (n = 23), shoes (n = 56), and washing machines (n = 52) were taken using sponges premoistened with buffered peptone water (5 mL, Hydrasponge, Biotrace, London, UK).[10] Thawed samples were enriched anaerobically in CD broth for 15 days

https://doi.org/10.1017/ice.2016.329 Published online by Cambridge University Press