ORIGINAL ARTICLE

Healthcare Antibiotic Resistance Prevalence – DC (HARP-DC): A Regional Prevalence Assessment of Carbapenem-Resistant Enterobacteriaceae (CRE) in Healthcare Facilities in Washington, District of Columbia

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OBJECTIVE. Carbapenem-resistant Enterobacteriaceae (CRE) are a significant clinical and public health concern. Understanding the distribution of CRE colonization and developing a coordinated approach are key components of control efforts. The prevalence of CRE in the District of Columbia is unknown. We sought to determine the CRE colonization prevalence within healthcare facilities (HCFs) in the District of Columbia using a collaborative, regional approach.

DESIGN. Point-prevalence study.

SETTING. This study included 16 HCFs in the District of Columbia: all 8 acute-care hospitals (ACHs), 5 of 19 skilled nursing facilities, 2 (both) long-term acute-care facilities, and 1 (the sole) inpatient rehabilitation facility.

PATIENTS. Inpatients on all units excluding psychiatry and obstetrics-gynecology.

METHODS. CRE identification was performed on perianal swab samples using real-time polymerase chain reaction, culture, and antimicrobial susceptibility testing (AST). Prevalence was calculated by facility and unit type as the number of patients with a positive result divided by the total number tested. Prevalence ratios were compared using the Poisson distribution.

RESULTS. Of 1,022 completed tests, 53 samples tested positive for CRE, yielding a prevalence of 5.2% (95% CI, 3.9%–6.8%). Of 726 tests from ACHs, 36 (5.0%; 95% CI, 3.5%–6.9%) were positive. Of 244 tests from long-term-care facilities, 17 (7.0%; 95% CI, 4.1%–11.2%) were positive. The relative prevalence ratios by facility type were 0.9 (95% CI, 0.5–1.5) and 1.5 (95% CI, 0.9–2.6), respectively. No CRE were identified from the inpatient rehabilitation facility.

CONCLUSION. A baseline CRE prevalence was established, revealing endemicity across healthcare settings in the District of Columbia. Our study establishes a framework for interfacility collaboration to reduce CRE transmission and infection.

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Carbapenem-resistant Enterobacteriaceae (CRE) have increasingly been recognized as multidrug-resistant organisms (MDROs) of significant clinical and public health concern due to their prevalence, colonization potential, high levels of antibiotic resistance, associated morbidity and mortality, and potential for widespread transmission.

Enterobacteriaceae bacteria are common pathogens that represent a major source of infection in healthcare settings.[1–4] Surveillance data indicate that up to 92% of CRE infections occur in patients with sustained healthcare exposures.[5] Active surveillance in outbreak situations has also shown colonization to be common among asymptomatic patients.[6] Colonized patients, both identified and unidentified, can serve as a reservoir for transmission of these organisms to other patients.[2]

Infections caused by CRE have limited or no treatment options. Various resistance mechanisms render these organisms nonsusceptible to several classes of antibiotics, including carbapenems, which are often considered drugs of last resort for CRE infections. Invasive CRE infections have been associated with mortality rates of up to 50%.[7]

CRE also have the potential to spread rapidly, due not only to breaches in infection control but also to the transfer of mobile genetic elements conferring resistance. Enterobacteriaceae resistant to carbapenems, such as *Klebsiella pneumoniae,* often carry genes encoding enzymes like the *K. pneumoniae* carbapenemase (KPC) on mobile plasmids that can confer carbapenem resistance to other gram-negative bacterial species.[8] KPC was first recognized in the United States in 2001, and it has accounted for most of the spread of CRE throughout the United States. In addition to KPC, several additional carbapenemases have been identified since 2009, including New Delhi metallo-β-lactamase (NDM), Verona integron-encoded metallo-β-lactamase (VIM), oxacillinase-48-type carbapenemases (OXA-48), and the imipenemase (IMP) metallo-β-lactamase.[9,10]

The Centers for Disease Control and Prevention (CDC) recommends immediate action to prevent further spread of this "urgent" antibiotic-resistance threat.[11] Due to the movement of patients within healthcare systems, control of MDROs requires widespread engagement and regional coordination across healthcare facilities (HCFs).[12,13] Studies evaluating a regional approach to infection control interventions have shown this approach to be effective in reducing MDRO transmission.[14,15] Mathematical models[16] have estimated that a coordinated approach may result in up to a 74% cumulative reduction in CRE acquisition over a 5-year period compared with independent facility-based interventions.

A coordinated, regional approach is especially important for the District of Columbia because it is the only metropolitan city that is viewed as a state with respect to assessments of infection control outcomes and epidemiology, which leads to difficulty in interpreting comparative data. All HCFs are clustered in a small region, with patients often receiving care at multiple facilities over a short period of time. These facilities not only serve more than 6 million residents in the metropolitan District of Columbia area but also provide care for patients from Maryland and Virginia, as well as national and international travelers. Therefore, a regional approach with coordination among facilities, partners, and stakeholders is essential.

An initial step in controlling the spread of antibiotic resistance often is to quantify the magnitude and distribution of MDRO infection and colonization. An accurate estimate of the burden of CRE colonization can only be determined through active surveillance of patients at risk for CRE. At the time of this study, because CRE reporting was not mandated in the District of Columbia and because individual facilities do not routinely conduct active surveillance for CRE, the prevalence of colonization was unknown. The objective of this study was to assess CRE colonization prevalence within HCFs in Washington, DC, and to initiate a collaborative approach for further assessment and control efforts.

METHODS

Facility Recruitment

The District of Columbia Department of Health (DOH), the District of Columbia Hospital Association (DCHA), and the District of Columbia Department of Forensic Science-Public Health Lab (DFS-PHL) partnered to design and conduct a multicenter point-prevalence study of CRE colonization in HCFs in the District of Columbia. A total of 16 facilities in the District of Columbia voluntarily agreed to participate: all 8 short-term acute-care hospitals (ACHs), 2 (both) long-term acute-care hospitals (LTACs), 1 (the sole) inpatient rehabilitation facility, and 5 of the 19 skilled-nursing facilities (SNFs). Results for LTACs and SNFs were combined as long-term-care facilities (LTCFs) for analysis. Each HCF conducted bacterial colonization surveillance of all participating units over a 1–3-day interval (depending on facility size) from January 11 to April 14, 2016.

Data Collection

An independent, external ethical review was conducted by the Western Institutional Review Board (WIRB; Pallyup, WA). Facilities could waive oversight of the study protocol to WIRB or evaluate the protocol independently for approval. Written informed consent was waived; verbal consent was obtained. Patient variables collected were age, sex, and zip code. Location variables included facility and unit type, as defined by the National Healthcare Safety Network (NHSN). Types of healthcare units were grouped as critical care units, step-down wards, general wards, inpatient rehabilitation units, and long-term care units (ie, LTAC and SNFs combined).

The Healthcare Antibiotic Resistance Prevalence–DC (HARP-DC) study team, consisting of representatives from the partnering organizations, coordinated with each HCF

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principle investigator to facilitate collection of CRE samples by facility-based volunteers. Sampling teams varied in composition by facility; they included infection preventionists, nurses, residents, and physicians. Adult inpatients on all units (excluding psychiatry and obstetrics-gynecology) who were present in their rooms at the time of study visit were approached for consent. Pediatric patients were only included from the dedicated pediatric facility where parents provided consent. Patients were excluded if they were unable to provide verbal consent due to language barrier, cognitive inability, or if it was a mentally/emotionally or clinically inappropriate time (Figure 1). For patients who agreed to participate, perianal samples were collected using ESwab Collection Kits (Becton Dickinson, Franklin Lakes, NJ). Adult patients capable of swabbing themselves were instructed how to collect the sample properly and were permitted to do so.

CRE Identification

All samples were analyzed for CRE detection at the OpGen Clinical Services Laboratory (Gaithersburg, MD). Using 2015 CDC surveillance definition, CRE were defined as nonsusceptible to any of the carbapenems (ie, imipenem, meropenem, doripenem, or ertapenem) or were identified to possess a carbapenemase gene. Samples were analyzed using the Acuitas CR Elite Test (OpGen), which consists of 2 tests run in parallel, the MDRO gene test and the CRE culture screen.[17] The MDRO gene test is a real-time polymerase chain reaction (qPCR) microfluidic array that detects 10 groups of antibiotic resistance genes, including carbapenemases and β-lactamase genes, that confer resistance to carbapenems in Enterobacteriaceae and other gram-negative pathogens (eg, *Acinetobacter* spp. and *Pseudomonas aeruginosa*). The CRE culture screen selects for phenotypic carbapenem resistance using chromogenic agar medium, with further genus and species identification and antimicrobial susceptibility characterization (ID/AST) using VITEK 2 (bioMérieux, Durham, NC). Thus, CRE were identified genetically by the MDRO gene test, phenotypically by the CRE culture screen, or by both methods. Samples with growth on the CRE culture screen underwent further testing using the Acuitas Resistome Test, a qPCR test that detects 50 antibiotic resistant gene families across several hundred variants associated with MDROs. In brief, 100 µL of total nucleic acid was extracted from each

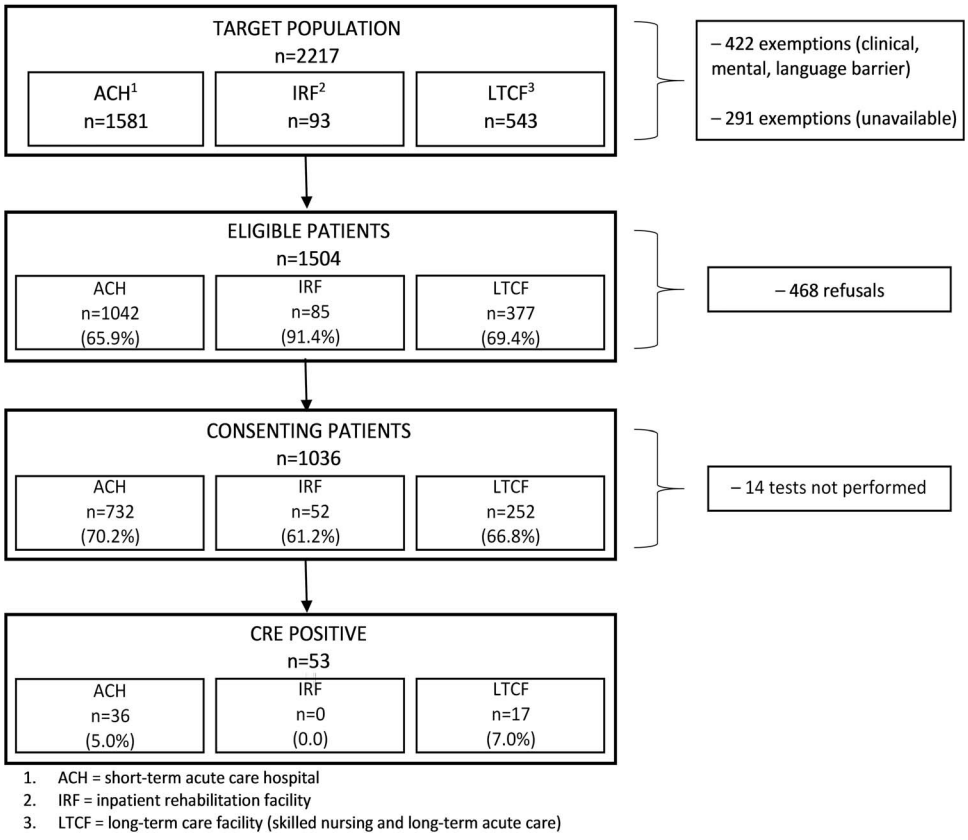

FIGURE 1. HARP-DC study enrollment process. Display of patient recruitment for point prevalence study of CRE colonization by facility type. Inpatients on all wards (excluding psychiatry and ob-gyn) present in their room at the time of study were approached. Exclusion criteria included inability to provide verbal consent due to a language barrier, or inappropriate timing (given the patient's clinical/mental/emotional status).

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culture isolate. qPCR amplifications specific to the Acuitas Resistome Test targets were performed using the template from each sample and primers and fluorescent reporter probes for each target. Results were analyzed on a BioMark HD System (Fluidigm, San Francisco, CA).

Determining Strain Relatedness

Phenotypic and genetic results of recovered isolates were integrated to construct Lighthouse profiles for typing, cluster identification, and tracking. Each Lighthouse profile comprises codes that include (1) organism genus and species identification, (2) phenotype code determined by AST results, (3) listing of up to 3 gene codes determined by the Acuitas Resistome Test, (4) a unique numerical code representing the pattern of all positive and negative assays from the Acuitas Resistome Test results, and (5) the AST profile code representing the unique pattern of nonsusceptible AST results (Supplementary Figure S1). Genetically related types were determined by combining the organism name and the code for the Acuitas Resistome Test results. These types were further divided into subtypes using the Lighthouse profile's AST code.

All results were posted on the secure Acuitas Lighthouse MDRO Management System (OpGen) website portal for real-time access by the study coordinators and principle investigators.

Statistical Analysis

Descriptive statistics were performed to summarize patient-level and facility characteristics. Proportions were reported for categorical variables, and differences were tested using 2-tailed χ^2 tests. Prevalence and 95% confidence intervals were calculated using the Poisson distribution. The prevalence of CRE was calculated as the number of patients with a positive result (by qPCR or by ID/AST) divided by the total number tested. Differences in prevalence were assessed using the prevalence ratio. Covariates of interest measured at time of sample collection were assessed using Poisson regression with robust variance estimates to determine association with CRE colonization. All statistical analyses were performed using Stata statistical software version 13 (StataCorp, College Station, TX).

RESULTS

Of 1,022 completed tests, 53 samples tested positive for CRE, yielding a prevalence of 5.2% (95% CI, 3.9%–6.8%). Of 726 tests from ACHs, 36 (5.0%; 95% CI, 3.5%–6.9%) were positive; of 244 tests from LTACs and SNFs, 17 (7.0%; 95% CI, 4.1%–11.2%) were positive. The relative prevalence ratio was 0.9 (95% CI, 0.5–1.5) and 1.5 (95% CI, 0.9–2.6), respectively. Of 52 samples from the inpatient rehabilitation facility, none were positive.

We observed wide variability in the prevalence of CRE by facility and facility type (Table 1). The median percentage of positive samples by facility was 2.7, with a range of 0.0 to 29.4. Within ACHs, 6 of 90 critical care patients (6.7%; range, 0.0%–11.6%), 1 of 61 step-down patients (1.6%; range, 0.0%–3.7%), and 29 of 575 ward patients (5.0%; range, 0.0%–9.5%) tested positive. Males had a significantly higher prevalence (7.1%; 95% CI, 5.3%–10.6%) than females (3.7%; 95% CI: 2.1%–5.6%; $P < .02$). The prevalence by age group was 1.8% (95% CI, 0.0%–10.1%) for those under 20 years of age, 8.0% (95% CI, 3.2%–16.4%) for those aged 20–39, 5.6% (95% CI, 3.2%–9.1%) for those aged 40–59, 5.9% (95% CI, 3.8%–8.6%) for those aged 50–79, and 2.2% (95% CI: 0.4%–6.4%) for those over 79 years of age (Table 2). Zip code data were collected but were not interpretable for long-term-care patients because most listed the facility as their home zip code. Of 697 patients with zip code recorded from ACHs, no more than 3 patients tested positive from any of the 200 zip codes recorded.

The CDC definition of CRE includes Enterobacteriaceae with carbapenemase production (CP-CRE) and those with carbapenem resistance without an identified resistance gene (non–CP-CRE).[13] In this study, CP-CRE were detected by qPCR with parallel testing by ID/AST for non–CP-CRE. Therefore, CRE positivity could be detected by either method or by both methods. qPCR detected CP-CRE in 45 samples: 44 positive for bla_{KPC}, 1 for bla_{NDM}, and 1 with both bla_{KPC}

TABLE 1. Prevalence of Carbapenem-Resistant Enterobacteriaceae (CRE) by Facility and Unit Type

Patient Care Type	No. Facilities	No. Sampled	No. CRE Positive	% CRE Positive (95% CI)	Median (Range)	Prevalence Ratio (95% CI)[a]
Inpatient rehabilitation	1	52	0	0.0		0.0
Long-term care	7	244	17	7.0 (4.0–11.1)	3.0 (0.0–29.4)	1.5 (0.9–2.6)
Short-term acute care	8	726	36	5.0 (3.5–6.9)	2.6 (0.0–7.7)	0.9 (0.5–1.5)
Critical care	8	90	6	6.7 (2.4–14.5)	0.0 (0.0–11.6)	1.3 (0.6–3.0)
Step down	4	61	1	1.6 (0.0–9.1)	0.0 (0.0–3.7)	0.3 (0.0–2.2)
Ward	8	575	29	5.0 (3.5–7.2)	2.5 (0.0–9.5)	0.9 (0.5–1.5)
Total	16	1,022	53	5.2 (3.9–6.8)	2.7 (0.0–29.4)	…

NOTE. CI, confidence interval.

[a]Prevalence ratio = % CRE in patient care type / % CRE for all other patient care types.

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TABLE 2. Prevalence of Carbapenem-Resistant Enterobacteriaceae (CRE) by Age and Gender

Age, y	No. in Age Group	No. CRE Positive	% CRE Positive (95% CI)	% Male with CRE (95% CI)	% Female with CRE (95% CI)
<20	55	1	1.8 (0.0–10.1)	4.2 (0.1–23.2)	0.0
20–39	88	7	8.0 (3.2–16.4)	12.0 (4.4–26.1)	2.6 (0.1–14.6)
40–59	285	16	5.6 (3.2–9.1)	9.0 (4.8–15.3)	2.1 (0.4–6.3)
60–79	442	26	5.9 (3.8–8.6)	6.3 (3.5–10.6)	5.4 (2.8–9.5)
>79	137[a]	3	2.2 (0.4–6.4)	1.8 (0.0–10.1)	2.4 (0.3–8.8)
Total	1,007[b]	53	5.3	7.1 (4.9–9.8)	3.6 (2.1–5.6)[a]

NOTE, CI, confidence interval.
[a]$P < .05$.
[b]15 samples lacked age data.

TABLE 3. Carbapenem-Resistant Enterobacteriaceae (CRE) Identification by Resistance Gene Testing and Microbiologic Culture Methods

	Carbapenase Genes				
Organisms Identified by ID/AST	*bla*$_{KPC}$ (CP-CRE)	*bla*$_{NDM}$ (CP-CRE)	*bla*$_{OXA\ 48}$ (CP-CRE)	No Carbapenemase Detected (non–CP-CRE)	Total (% of total by ID/AST)
Klebsiella pneumoniae	16			3	19 (35.8)
Enterobacter cloacae	6			1	7 (13.2)
Escherichia coli		1		3	4 (7.5)
Serriata marcescens				1	1 (1.9)
Citrobacter amalonaticus	1				1 (1.9)
C. koseri	1				1 (1.9)
Indeterminant	1				1 (1.9)
No growth	19		1		19 (35.8)[a]
Total (% of total by qPCR)	44 (83.0)	1 (1.9)	1 (1.9)	8 (15.1)	53[a]

NOTE. CP-CRE, Enterobacteriaceae with carbapenemase production; ID/AST, genus and species identification and antimicrobial susceptibility characterization; qPCR, quantitative polymerase chain reaction.
[a]1 sample without growth was positive for both KPC and OXA-48. The total column corrects for the double count.

and *bla*$_{OXA48}$. Of these, 19 samples were identified only by qPCR. ID/AST detected CRE in 33 samples: 19 *Klebsiella pneumoniae*, 7 *Enterobacter cloacae*, 4 *Escherichia coli*, and 1 each of *Serratia marcescens*, *Citrobacter amalonaticus*, and *Citrobacter koseri*. Of these, 8 were non–CP-CRE, with identification only by ID-AST but not by qPCR (Table 3).

Lighthouse profiles were used to assess strain relatedness (Supplementary Table S1). Of the 53 samples positive for CRE, 18 were determined to share genetic similarity with at least 1 other sample. Among these 18 samples, 5 genetic types containing a unique combination of resistance genes were identified. AST results were incorporated to further divide these 5 types into subtypes using the code for each unique antibiogram within the type. Within each type, there were no AST differences for aztreonam, cefazolin, cefepime, ceftazidime, ceftriaxone, ciprofloxacin, imipenem, levofloxacin, meropenem, piperacillin/tazobactam, or ticarcillin. Within-type AST differences were only observed for aminoglycocides and trimethoprim-sulfa. In addition, 5 patients from HCF A were positive for CRE *E. cloacae* with an identical genetic type and AST subtype (Type 1). Type 2 was detected in 5 samples in 4 facilities; type 3 was detected in 3 samples in 2 facilities; type 4 was detected in 3 samples in 3 facilities; and type 5 was detected in 2 sample in 2 facilities (Figure 2).

DISCUSSION

HARP-DC is one of the first studies to our knowledge to measure CRE colonization prevalence in a region aligning with the CDC's recommended "collaborative approach." Active surveillance data collected over a short period from all participating HCF types and units were analyzed using a single laboratory with a comprehensive testing methodology. This design (1) provides estimates of CRE colonization in inpatient HCFs across all age groups and the continuum of care in Washington, DC, (2) establishes a baseline prevalence to monitor trends over time, and (3) enables comparison of rates across facilities. The overall prevalence of 5.2% confirms that CRE has become endemic in HCFs; the prevalence of 29.4% in 1 facility demonstrates the potential for hyperendemicity, which may have grave implications for patient care. The study also provides a model for other regions to conduct CRE prevalence measurements.

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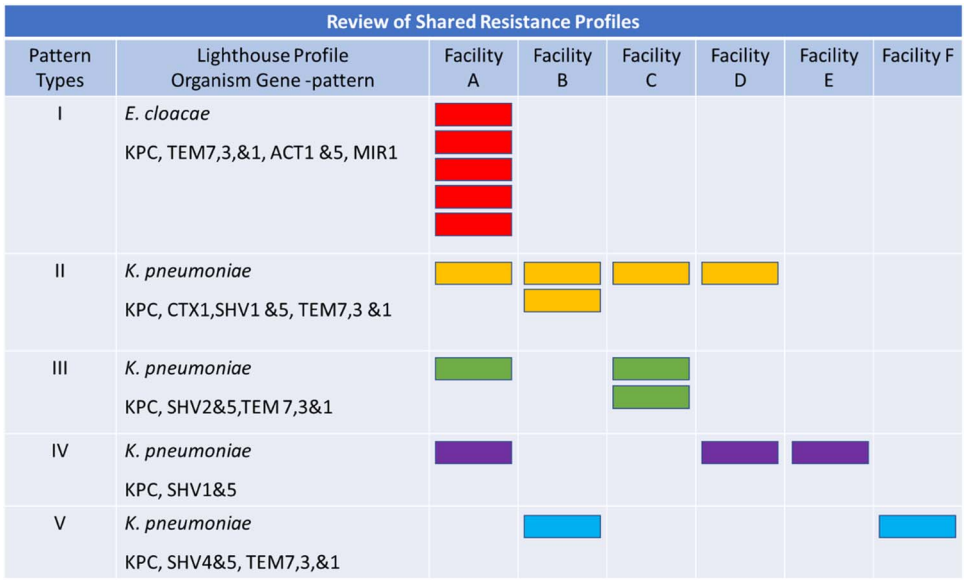

FIGURE 2. Review of shared resistance profiles. Illustration of how 18 nonunique samples that were identified by Lighthouse profiles were distributed among participating facilities. The Lighthouse profile was composed of organism, PCR gene identification, and antibiogram. Patterns 1, 2, and 3 show possible transmission within facilities and Patterns 2, 3, 4, and 5 show possible transmission between facilities.

Since CRE were first identified, the CDC has advised HCFs to measure infection incidence and to aggressively respond to any rate increase or evidence of transmission within an individual facility.[13] The CDC has also advocated for public health organizations to measure incidence or prevalence in a region, as organisms can spread between facilities by patients, staff, or shared environmental sources. The 4 main methods of CRE surveillance are (1) individual facility surveillance using clinical cultures to estimate incidence,[18] (2) multifacility surveillance using clinical cultures to measure regional incidence,[19–23] (3) individual facility using active surveillance to detect CRE colonization following identification of a cluster,[24] and (4) multifacility active surveillance for colonization to determine regional prevalence.[25–28]

Traditionally, facilities have relied on clinical cultures to conduct CRE surveillance, with active surveillance occurring only after the identification of an outbreak or cluster. Clinical cultures provide a measurement of CRE infection and are easier to obtain because samples are more readily available. Some researchers have found that patients with clinical cultures are more likely to transmit the organism to others.[29] However, because colonization plays a role in transmission, is a predictor for possible later infection, and is often under-recognized,[21] conducting active surveillance provides an accurate measure of overall CRE burden rather than infection, and it establishes a baseline prevalence.

Although the CDC has advocated for a regional approach to MDRO control, few studies to date have undertaken regional CRE surveillance through either clinical culture or active surveillance because such studies are often too expensive or labor intensive.[22,24,26] In addition, variation in laboratory methods, changes in CRE definition, discord between automatic testing instruments and broth microdilution methods for CRE identification, unavailability of molecular testing in many clinical laboratories,[23] and changing Clinical Laboratory Standards Institute break points can make assessment of patterns over time or comparison between facilities difficult. HARP-DC controlled for these variations by using the CDC CRE surveillance definition and by limiting testing to a single laboratory.

HARP-DC is one of the few studies (Table 4) to measure regional CRE colonization prevalence without an instigating outbreak or a focus on units with anticipated higher rates.[25–28] In this study, the prevalence in general wards was 2.5 per 100 patients compared to 2.7 for the entire study, including intensive care units (ICUs) and LTACs (prevalence ratio = 0.9). Until recently, researchers expected a relatively low CRE prevalence on general wards compared with ICUs or LTACs.[21]

Rapid MDRO identification by qPCR for resistance profile characterization is a helpful method either for active surveillance for colonized patients or for surveillance using clinical samples. While HARP-DC's goal was to provide an estimate of CRE prevalence, the rapid organism-profile identification of a previously unrecognized cluster within a hospital also provided an example of how surveillance programs using such techniques may identify outbreaks at an early stage.

As described in the literature,[30–32] molecular nucleic amplification technologies such as qPCR are more sensitive than culture. In this study, 19 samples were identified as CP-CRE by qPCR without growth by culture. qPCR has the additional ability to identify the presence of nonviable cells, which could also explain these results. However, in several

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TABLE 4. Published Reports of Regional Carbapenem-Resistant Enterobacteriaceae (CRE) Prevalence Studies Using Active Surveillance

Study	Time Period	Population	No. of Specimens	Percent Positive	Anatomic Source
HARP-DC	Jan 2016–Apr 2016	Inpatient from ACH, LTCF, and IRF	1,022	5.2	Perianal
State of Maryland[25]	July–Aug 2010 (12 days)	Patients on ventilators	358	6.0	Perianal and sputum
University of Virginia Health System[26]	Sept 2009–Jan 2010 and Jan 2012–Dec 2012	Selected ICU and LTAC units where CRE had been prevalent	7,608	2.6	Perianal
Chicago[27]	ICU, Jul 2010–Jul 2011 and LTAC, Jan 2011–May 2011	ICU and LTAC	ICU, 910 LTAC, 391	ICU, 3.3 LTAC, 30.4	Inguinal and urine (if catheter in place)
New York[28]	Feb 2009–Jan 2010	ICU, medical-surgical units, acute rehabilitation	5,676	5.4	Perianal

NOTE. HARP-DC, Healthcare Antibiotic Resistance Prevalence–District of Columbia; ACH, acute-care hospital; LTCF, long-term-care facility; IRF, inpatient rehabilitation facility; ICU, intensive care unit; LTAC, long-term acute-care hospital.

cases, CRE was recovered by culture but not detected by qPCR. This discrepancy may be because qPCR only detects targets in its panel. The 8 non–CP-CRE identified in this study may be due to novel resistance mechanisms other than carbapenemase production, such as β-lactamases, and/or to mutations in the bacterial cell membrane.[33]

We faced several challenges and limitations in this study. We were only able to enroll ~50% of the target population. As participation required verbal consent from the patient or a legally authorized representative, enrollment of the cognitively impaired or ventilated patients was difficult. In addition, use of the perianal site for sample collection may have decreased the acceptance rate. Instead of using the perianal site for sampling, alternative sites such as the inguinal site[34–36] might have improved the sample strategy. Because the collectors could determine patient selection appropriateness, some patients were not enrolled because of modesty or concern that repositioning would be too uncomfortable. These limitations may have biased the study to disproportionately sample those who were more mobile, movable, alert, and coherent, and therefore, the CRE prevalence estimate may be underestimated.

To ensure maximum HCF participation, the only patient-based variables collected were age, gender, zip code, and unit type. Without epidemiologic risk-factor data collection, the investigators were unable to assess the role of comorbidities, device days, surgeries, use of mechanical ventilation, hemodialysis, antimicrobial therapy, and preceding HCF exposure. Therefore, we were able to determine whether a particular sample possessed a carbapenemase-producing gene but not whether or how that organism had been transmitted. Similarly, because results were only analyzed at the unit level, HCFs were unable to use the data to isolate colonized patients or to make clinical decisions. In addition, we did not collect variables to allow classification of likely acquisition as defined by the NHSN to represent community onset or healthcare onset. Without a clearer understanding of colonization duration, it is difficult to establish onset.[21,28]

In conclusion, this study provides an important baseline measurement of CRE burden in Washington, DC, HCFs and confirms likely transmission within and between facilities. Testing performed by a single laboratory over a short period of time can provide a useful measure of CRE burden in a region and establish patterns of transmission within and between facilities.

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SUPPLEMENTARY MATERIAL

To view supplementary material for this article, please visit https://doi.org/10.1017/ice.2017.110

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