Figure 1: Primary and Secondary Endpoints

Antimicrobial exposure	Cases n = 106 (%)	Controls n = 119 (%)	Odds Ratio (95% CI)
≥ 48 hours of meropenem	21 (19.8)	5 (4.2)	5.63 (2.04 – 15.54)
≥ 7 days of meropenem	15 (14.2)	4 (3.4)	4.74 (1.52 – 14.77)
≥ 7 days of other antipseudomonal antibiotics	37 (34.9)	18 (15.1)	3.01 (1.59 – 5.71)

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484. Metallo-β-Lactamase-Positive Carbapenem-Resistant Enterobacteriaceae and *Pseudomonas aeruginosa* in the Antibiotic Resistance Laboratory Network, 2017–2018

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Background. Infections with metallo-β-lactamase (MBL)-producing organisms are emerging in the United States. Treatment options for these infections are limited. We describe MBL genes among carbapenemase positive carbapenem-resistant Enterobacteriaceae (CP-CRE) and *Pseudomonas aeruginosa* (CP-CRPA) isolates tested during the first two years of the Antibiotic Resistance Laboratory Network (AR Lab Network).

Methods. State and local public health laboratories tested CRE and CRPA isolates for organism identification, antimicrobial susceptibility, and PCR-based detection of bla_{KPC}, bla_{NDM}, $bla_{OXA\text{-}48\text{-}like}$, bla_{VIM}, and bla_{IMP} carbapenemase genes. All testing results were sent to CDC at least monthly.

Results. Since January 2017, the AR Lab Network tested 21,733 CRE and 14,141 CRPA. CP-CRE were detected in 37% of CRE; 2% of CRPA were CP-CRPA. Among CP-CRE, 9% (686/8016) were MBL-producers (NDM, VIM, or IMP). Among MBL-producers, a bla_{NDM} gene was detected most often (81%; 551/686). bla_{NDM} were most common among *Klebsiella* spp. (47%; 261/551), bla_{IMP} were most common among *Providencia* spp. (53%; 40/75), bla_{VIM} was most common among *Enterobacter* spp. (19%; 25/62). Twelve percent (96) of MBL CP-CRE contained more than one carbapenemase gene. Among CP-CRPA, 73% (218/300) were MBL producers and bla_{VIM} was the most common gene (62%; 186). Three (1%) MBL CP-CRPA contained more than one carbapenemase.

Conclusion. Increased testing of CRE and CRPA isolates through the AR Lab Network has facilitated early and rapid detection of hard-to-treat infections caused by MBL-producing organisms across the United States. The widespread distribution of MBL genes highlights the continued need for containment strategies that help prevent transmission between patients and among healthcare facilities. To support therapeutic decisions for severe infections caused by MBL-producing organisms, the AR Lab Network is now offering rapid susceptibility testing against aztreonam/avibactam, using digital dispenser technology. This testing program aims to close the gap between the availability of new drugs or drug combinations and the availability of commercial AST methods, thereby improving patient safety and antimicrobial stewardship.

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485. Clinical and Molecular Epidemiology of Carbapenem Non-susceptible *Citrobacter* sp.

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Background. Carbapenem non-susceptible *Citrobacter* sp. (CNSC) are becoming increasingly recognized as healthcare-associated (HA) pathogens, but data on clinical and molecular epidemiology, species diversity and mechanisms of carbapenem resistance are lacking.

Methods. We reviewed patients at University of Pittsburgh Medical Center with CNSC positive cultures from 2000 to 2018. The diversity of CNSC species among a subset of isolates from all UPMC sites was confirmed by 16S rRNA typing, and the presence of carbapenemase enzymes in the same isolates was determined by PCR amplificon. Minimum inhibitory concentrations (MICs) were determined by broth microdilution. Significance of epidemiological trends over time was determined by linear regression, and correlation with antibiotic consumption was determined by cross-correlation using STATA v15.

Results. Between 2000 and 2018, 3% (78/2817) of all *Citrobacter* sp. were CNS. CNSC rates increased from 4% in 2000 to 10% in 2018 (R^2 = 0.206, P = 0.05), as did carbapenem consumption (6.5–34.5 DDDs/1000, R^2 = 0.831, P < 0.001) (Figure 1). Twenty-one isolates from 19 patients were available for additional analysis. Patients had multiple comorbidities (84%), frequently acquired CNSC in the healthcare setting (84%), were colonized with other organisms (68%), and had high rates of in-hospital mortality/discharge to hospice (47%) (Table 1). *C. freundii* was the dominant species identified (16/21), followed by *C. farmeri* (2/21), *C. koseri* (2/21), and *C. werkmanii* (1/21). Carbapenemases were identified in 14 isolates, including KPC (n = 12), NDM (n = 2), and OXA-48 (n = 1) (Table 2). Isolates were frequently susceptible to ceftazidime–avibactam (MIC median [IQR]: 2[0.5,8]) 81%) and meropenem-vaborbactam (86%) (MIC median [IQR] 0.12[0.3,0.5]) (Table 2).

Conclusion. CNSC species are diverse, have emerged as an HA pathogen at our center, and cause high rates of mortality. Further studies, including ongoing genome sequencing and analysis, are required to better elucidate CNSC diversity and resistance mechanisms.

Figure 1: Rates of CNSC and Carbapenem Consumption from 2000-2018.

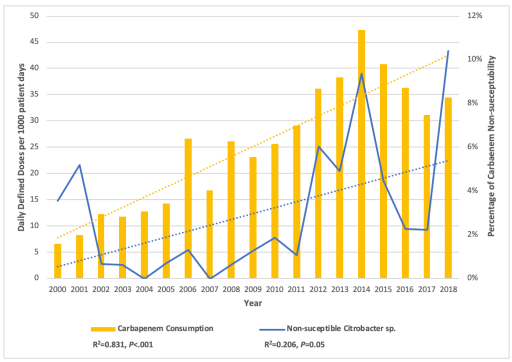

Figure 1 Legend: Carbapenem consumption (daily defined doses) and carbapenem non-susceptibility (non-susceptible isolates/total isolates tested) between 2000-2018. Trends evaluated by linear regression.

Table 1: Clinical characteristics of patients with CNSC.

Isolate ID	Age	Gender	Major Comorbid Conditions	Year	Source	Organism	Culture site	Clinical Infection	Polymicrobial culture (Y/N)	Other orgs	Outcome of hospitalization
RS-102	74	F	ESLD, liver transplant	2018	HA	*C. freundii*	Rectal swab	Colonization	N	N/A	In-hospital death
RS-189	66	F	Diverticulosis	2017	HA	*C. freundii*	BAL	Pneumonia	N	N/A	Discharge to facility
RS226	56	F	Opiate dependency, CHF, HTN	2018	HA	*C. freundii*	Urine	Colonization	Y	*E. coli*	In-hospital death
RS236	70	M	Pancreatic cancer, COPD, DM, HTN	2018	HA	*C. freundii*	Peritoneal fluid	Intra-Abdominal	Y	*C. freundii* (ESBL), *E. faecium* (VRE) *C. tropicalis, C. parapsilosis*	Transfer to hospice
RS237	80	F	Sinus OM	2018	Community	*C. freundii*	Urine	Colonization	N	N/A	Discharge to home
RS240	29	F	Nephrolithiasis obstructive uropathy	2017	Community	*C. freundii*	Urine	Colonization	Y	*S. maltophilia, S. hemolyticus E. faecalis E. cloacae*	Discharge to home
RS259	61	F	Metastatic lung cancer, COPD, DM	2018	HA	*C. freundii*	Peritoneal fluid	Intra-Abdominal	Y	*E. coli, P. aeruginosa*	In-hospital death
RS289	92	M	Bladder cancer, dementia, CKD, COPD, CHF	2018	Community	*C. koseri*	Urine	UTI	Y	*E. faecalis*	Discharge to home
RS77	49	M	ESLD, liver transplant	2018	HA	*C. freundii*	Rectal swab	Colonization	N	N/A	Discharge to home
YDCS82	54	M	ESLD, HCC	2012	HA	*C. freundii*	Abdominal drain	Intra-Abdominal	Y	*E. aerogenes*	In-hospital death
YDC608	57	M	HLD	2013	HA	*C. freundii*	BAL	Colonization	N	N/A	Transfer to hospice
YDC638-2	27	M	ESLD, Crohn's diseases, PSC, liver transplant	2013	HA	*C. freundii*	Biliary drainage	Intra-Abdominal	Y	*E. faecium* (VRE)	Discharge to home
YDC645	67	F	CAD, CHF, DM, ESRD, dementia	2013	HA	*C. freundii*	Blood	SSTI/ Bacteremia/ Endocarditis	Y	*Bacteroides, E. rafflosis*	Transfer to hospice
YDC661	64	M	Heart transplant	2014	HA	*C. freundii*	BAL	Pneumonia	Y	*S. maltophilia*	Discharge to facility
YDC667-1	73	M	CHF, DM, CAD, CKD	2014	HA	*C. freundii*	BAL	Pneumonia	Y	*K. pruarmoniae* (ESBL)	In-hospital death
YDC689-2	61	M	ESLD, liver transplant	2015	HA	*C. koseri*	BAL	Pneumonia	N	N/A	Discharge to facility
YDC693*	65	M	SBT, adrenal insufficiency	2015	HA	*C. freundii*	BAL	Pneumonia	Y	ESBL *E. coli*	In-hospital death
YDC693-2*	65	M	SBT, adrenal insufficiency	2015	HA	*C. freundii*	BAL	Pneumonia	Y	ESBL *E. coli*	In-hospital death
YDC697-2*	65	M	SBT, adrenal insufficiency	2015	HA	*C. farmeri*	Tracheostomy site drainage	SSTI	Y	*K. oxytoca* (KPC)	In-hospital death
YDC730	71	M	Multiple myeloma	2015	HA	*C. werkmanii*	Pelvic abscess fluid	Intra-Abdominal	Y	*E. faecium* (VRE)	Discharge home
YDC849-1	26	F	CVID	2018	HA	*C. freundii*	Urine	UTI	Y	*C. freundii* (NDM)	In-hospital death

Abbreviations: BAL: Bronchoalveolar lavage CAD: coronary artery disease, CHF: congestive heart failure , CKD: chronic kidney disease, COPD: chronic pulmonary disease, CVID: common variable immunodeficiency, ESBL: Extend spectrum ß-lactamase, ESLD: end stage liver disease, ESRD: end stage renal disease DM: diabetes mellitus, F: female, HAP: hospital acquired pneumonia, HCC: hepatocellular carcinoma, HTN: hypertension, KPC: *Klebsiella pnuemoniae* carbapenemase, OM: osteomyelitis, M: male, NDM: New Delhi metallo-beta-lactamase, PSC: primary sclerosing cholangitis, SBT: small bowel transplant, SSTI: skin and soft tissue infection, VRE: Vancomycin-resistant *Enterococcus*, UTI: urinary tract infection

*Same patient

Table 2: Carbapenemase Enzymes and Antimicrobial Susceptibilities of CNSC to Novel Beta-Lactam-Beta-Lactamase Agents

Isolate	Organism	Carbapenemase*	Ceftazidime-Avibactam MIC	Meropenem-Vabrobactam MIC
RS102	*C. freundii*	-	0.5	0.06
RS189	*C. freundii*	NDM	0.5	0.015
RS226	*C. freundii*	KPC	<0.25	0.015
RS236	*C. freundii*	-	2	0.5
RS237	*C. freundii*	-	64 (R)	0.5
RS240	*C. freundii*	-	8	0.5
RS259	*C. freundii*	KPC	>256 (R)	>8 (R)
RS289	*C. koseri*	-	2	0.12
RS77	*C. freundii*	KPC	0.5	0.015
YDC582	*C. freundii*	KPC	>256 (R)	>8 (R)
YDC608	*C. freundii*	KPC	4	0.06
YDC638-3	*C. freundii*	KPC	2	0.06
YDC645	*C. freundii*	KPC	<0.25	0.03
YDC661	*C. freundii*	KPC	4	0.06
YDC667-1	*C. freundii*	KPC	0.5	0.03
YDC689-2	*C. koseri*	-	4	0.12
YDC693	*C. freundii*	KPC	1	0.03
YDC693-2	*C. farmeri*	KPC	4	0.12
YDC697-2	*C. farmeri*	KPC/OXA-48	4	0.12
YDC730	*C. werkmanii*	-	0.5	0.12
YDC849-1	*C. freundii*	NDM	>256 (R)	16 (R)

Abbreviations: R: Resistant

*Carbapenemase gene presence was evaluated by multiplex PCR. Genomic DNA from isolates was used in 50 µL reactions containing BlueTaq DNA Polymerase (Denville). Each multiplex PCR reaction contained KPC primers (forward TCGCCGTCTAGTTCTGCTGTCTTG and reverse ACAGCTCCGCCACCGTCAT), NDM primers (forward ACTTGGCCTTGCTGTCCTT and reverse CATTAGCCGCTGCATTGAT), and OXA-48 primers (forward ATGCGTGTATTAGCCTTATCG and reverse CATCCTTAACCACGCCCAAATC) at 0.5 µm concentration. Cycle parameters were as follows: 1 cycle at 94 °C for 15 min, then 30 cycles of 94 °C for 60 s, 66 °C for 90 s, and 72 °C for 90 s, followed by 1 cycle at 72 °C for 10 min

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486. Epidemiology of Carbapenem-Resistant *Pseudomonas aeruginosa* Identified through the Emerging Infections Program (EIP), United States, 2016–2018

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Background. *Pseudomonas aeruginosa* is intrinsically resistant to many commonly used antimicrobials, and carbapenems are often required to treat infections. We describe the crude incidence, epidemiology, and molecular characteristics of carbapenem-resistant *P. aeruginosa* (CRPA) in the EIP catchment area.

Methods. From August 1, 2016 through July 31, 2018, we conducted laboratory- and population-based surveillance for CRPA in selected areas in eight sites. We defined a case as the first isolate of *P. aeruginosa* resistant to imipenem, meropenem, or doripenem from the lower respiratory tract, urine, wounds, or normally sterile sites identified from a resident of the EIP catchment area in a 30-day period. Patient charts were reviewed. Analysis excluded cystic fibrosis patients. A random sample of isolates was collected. Real-time PCR to detect carbapenemase genes and whole-genome sequencing are in progress.

Results. We identified 4,209 cases in 3373 patients. The annual incidence was 14.50 (95% CI, 14.07–14.94) per 100,000 persons and varied among sites from 4.89 in OR to 25.21 in NY. The median age of patients was 66 years (range: < 1–101), 42.1% were female, and nearly all (97.5%) had an underlying condition. Most cases were identified from urine (42.8%) and lower respiratory tract (35.7%) cultures. Nearly all (93.3%) occurred in patients with inpatient healthcare facility stay, surgery, chronic dialysis, or indwelling devices in the prior year; death occurred in 7.2%. Among 937 isolates tested, 847 (90.4%) underwent PCR; six (0.7%) harbored a carbapenemase, from four sites (CO, MD, NY, and OR): bla_{VIM} (3), bla_{KPC} (2), and bla_{IMP} (1). Of 612 (65.3%) isolates sequenced, the most common ST types were ST235 (9.2%) and ST298 (4.9%).

Conclusion. Carbapenemases were rarely the cause of carbapenem resistance but were found at EIP sites with high and low CRPA incidence. The emergence of mobile carbapenemases in *P. aeruginosa* has the potential to increase the incidence of CRPA. Increased detection and early response to carbapenemase-producing CRPA is key to prevent further emergence.

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487. Prevalence of Antimicrobial Resistance in Gram-Negative Bacilli Bloodstream Infections at a Tertiary Teaching Hospital in the Dominican Republic

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Background. Bloodstream infections (BSI) with gram-negative bacilli (GNB) are a major cause of morbidity and mortality worldwide. Sepsis due to BSI can carry a mortality rate as high as 40%, with higher mortality in developing nations. Early and appropriate empiric therapeutic selection plays an important role in survival. The rising incidence of antimicrobial resistance (AMR) limits empiric treatment options. Local susceptibility patterns can vary per region, institution or setting. Understanding local AMR may help guide empiric treatment choices. We seek to describe resistance rates for GNB BSI in the Dominican Republic (DR).

Methods. This is a retrospective review of antimicrobial susceptibility patterns from bloodstream infections in a tertiary hospital in the DR. Susceptibility data from all adult inpatient blood cultures were collected from January 1 to December 31, 2017.

Results. A total of 124 blood cultures were reported. The most common organisms were *Escherichia coli* (43%) and *Klebsiella pneumoniae* (23%). Fluoroquinolone resistance was present in 70% of *E. coli*. Phenotypic susceptibility patterns consistent with extended-spectrum β-lactamase (ESBL) producing GNB were present in 46% of isolates. Carbapenem resistance was found in 4 samples and was most common in *P. aeruginosa*. Susceptibility profile is described on Table 1.

Conclusion. AMR was high in GNB BSIs in the DR. High rates of ESBL render common cephalosporins sub-optimal for empiric treatment. PTZ retains *in vitro* susceptibilities despite cefepime resistance but clinical efficacy is controversial. CTX-M ESBLs may cause these resistance pattern *in vitro*. Further studies are needed to determine genetic mechanisms of resistance. Establishing antimicrobial stewardship programs with rapid diagnostic testing that identify mechanisms of resistance may promote judicious use of carbapenems and reduce further the risk of further development of AMR.

Table 1. Susceptibility patterns for GNB BSI (%)

Organisms	Ampicillin-Sulbactam	Cefazolin	Cefepime	Ceftazidime	Ceftriaxone	Ciprofloxacin (FQ)	Ertapenem	Gentamicin	Imipenem	Piperacillin-tazobactam (PTZ)
E. coli	47	47	48	48	48	30	100	59	100	97
K. pneumoniae	29	37	37	37	37	52	97	50	97	82
P. aeruginosa	-	-	92	97		87	-	92	79	75
E. cloacae	-	-	90	81	81	81	100	100	100	86
A. baumannii	86	-	86	86		86	-	86	100	100

Disclosures. **All authors:** No reported disclosures.

488. Epidemiology and Outcomes for *Stenotrophomonas maltophilia* Infections at a Tertiary Care Center in Detroit, MI

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Background. *Stenotrophomonas maltophilia* is a gram-negative, biofilm-forming bacterium. The increasing use of antibiotics has allowed this bacterium to become a predominant nosocomial pathogen with inherent resistance to several antibiotics. In this study, we describe the epidemiology and outcomes for patients treated for *S. maltophilia* infections who were admitted to Detroit Medical Center from January 1, 2010 to August 31, 2018.

Methods. This was a retrospective cohort study that included *S. maltophilia* cultures isolated from sterile body sites from January 1, 2010 to August 31, 2018. Nonsterile body sites and tissue cultures were excluded, as well as cultures that were deemed to be colonization based upon clinical evaluation. Appropriate empiric antibiotic therapy was defined as a regimen administered three days prior to or four days following the *S. maltophilia* culture date. Appropriate definitive therapy was defined as antibiotic treatment administered five to fourteen days following the culture date. Patient data were extracted from the electronic medical record which included demographic information, length of stay and outcome data. Bivariate analysis was performed using SAS database.

Results. 126 patients with *S. maltophilia* infections were analyzed: 89 had bacteremia, 22 had lung infections, and 15 had other infections. The median length of stay was 16 days (IQR 6–30 days). Sixty-one patients (48%) admitted to the ICU had a median length of stay of 10 days (Table 2). Among the patients that were followed after discharge, 21 were readmitted within 30 days. Table 1 highlights the bivariate analysis of patients who died within 30 days vs. survived. Patients who received definitive antibiotic therapy had lower 30-day mortality (Table 1; CI 95%, OR=0.37, $P = 0.03$). In