Tennessee. We defined an incident 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. A random sample of isolates was screened at CDC for carbapenemases using the modified carbapenem inactivation method (mCIM) and real-time PCR.

Results. During the 12-month period, we identified 3,042 incident cases among 2,154 patients. The crude incidence rate was 21.2 (95% CI, 20.4–21.9) per 100,000 persons and varied by site (range: 7.7 in Oregon to 31.1 in Maryland). The median age of patients was 64 years (range: <1–101) and 41.2% were female. Nearly all (97.1%) had at least one underlying condition and 10.2% had cystic fibrosis (CF); 17.8% of cases were from CF patients. For most cases, isolates were from the lower respiratory tract (49.2%) or urine (35.3%) and occurred in patients with recent hospitalization (87.2%) or indwelling devices (70.3%); 8.7% died. At the clinical laboratory, 84.7% of isolates were susceptible to an aminoglycoside and 66.4% to ceftazidime or cefepime. Among the 391 isolates tested, nine (2.3%) were mCIM-positive; one had a carbapenemase detected by PCR (bla_{VIM-4}).

Conclusion. The burden of CRPA varied by EIP site. Most cases occurred in persons with healthcare exposures and underlying conditions. The majority of isolates were susceptible to at least one first-line antimicrobial. Carbapenemase producers were rare; a more specific phenotypic definition would greatly facilitate surveillance for these isolates.

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

1163. Impact of Difficult-to-Treat Resistance on Survival in Gram-Negative Bacteremia: A Risk-Adjusted Analysis Using Electronic Health Record-based Clinical Data From 140 US Hospitals

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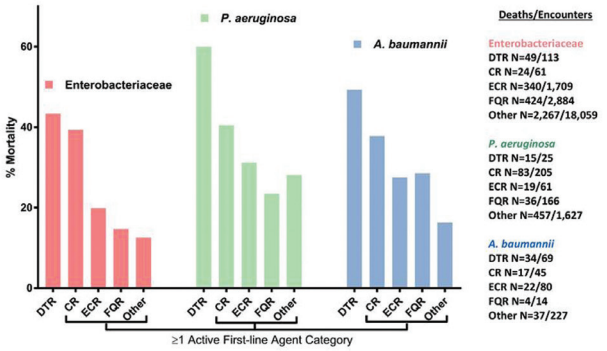
Background. In Gram-negative bacteremia (GNB), administrative data suggest that "difficult-to-treat resistance" (DTR; i.e., co-resistance to all first-line antibiotics) increases mortality. However, adequate risk-adjustment for severity of illness (SOI) may require granular laboratory and physiologic data.

Methods. Adult inpatients with GNB were identified from electronic health records (EHRs) of 140 hospitals in the *Cerner Healthfacts* database between 2009 and 2015. Mortality from DTR (intermediate/resistant *in vitro* to β-lactams including carbapenems and fluoroquinolones) was compared with GNB phenotypes susceptible to at least one first-line agent, but otherwise resistant to carbapenems (CR), extended-spectrum cephalosporins (ECR), or fluoroquinolones (FQR) per US Centers for Disease Control and Prevention surveillance definitions. Relative risk of mortality was adjusted (aRR) for age, sex, baseline Sequential Organ Failure Assessment (SOFA) score, Elixhauser comorbidity index, GNB source, taxon, hospital vs. community onset, year, and hospital region, bed capacity, and urban and teaching status using Poisson regression.

Results. Of 25,448 unique GNB encounters, 207 (1%) met DTR criteria. DTR patients were 2-fold more likely to receive intravenous colistin and 5-fold more likely to receive tigecycline compared with CR cases susceptible to ≥1 first-line agent. Crude mortality varied considerably by taxon and resistance phenotype, but resistance *per se* was associated with only a minority of overall deaths (DTR = 3% of deaths; any of the four resistance phenotypes = 28% of deaths; Figure 1). Inclusion of EHR-derived, baseline SOFA scores in SOI adjustments decreased aRR effect estimates; nonetheless, all resistance phenotypes still significantly increased mortality (Figure 2A). Among resistance phenotypes, aRR of mortality was similar for DTR vs. CR (aRR = 1.18; 95% CI, 0.91–1.54; $P = 0.2$), but higher for DTR vs. ECR (aRR = 1.26 [1.01–1.58]; $P = 0.04$), and DTR vs. FQR (aRR = 1.36 [1.08–1.70]; $P = 0.008$), respectively (Figure 2B).

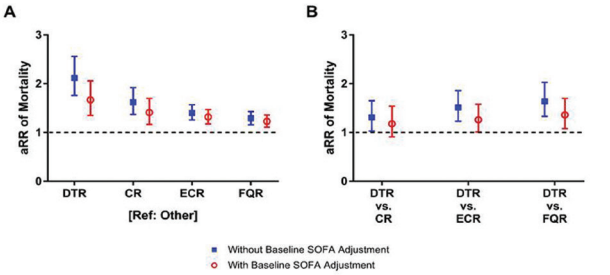
Conclusion. DTR is associated with nonsurvival and greater use of reserve antibiotics in GNB, but adds little to the risk of death associated with CR. The impact of resistance on survival is attenuated but still present even after risk adjustment using granular clinical data.

Figure 1. Crude mortality across patients with Gram-negative bacteremia by taxon and resistance phenotype



Crude mortality varied considerably by taxon and phenotype as seen in the figure. 97% of deaths in patients with GNB occurred in those in whom at least 1 first line agent was active, and 72% of deaths in those with GNB without any of the 4 resistance phenotypes. "Other" category refers to GNB encounters not classified as either DTR, ECR or FQR. CR= Carbapenem resistant; DTR=Difficult to treat resistance; ECR= Extended-spectrum cephalosporin-resistant; FQR= Fluoroquinolone resistant

Figure 2: Adjusted relative risk of mortality in Gram-negative bacteremia by resistance phenotype with and without adjustment for baseline Sequential Organ Failure Assessment (SOFA) score



In all Poisson regression models, aRR was adjusted using age, gender, Elixhauser comorbidity index, taxon, infection source, hospital vs. community onset, year, as well as hospital region, bed capacity, and urban and teaching status. The impact of adjustment by severity of acute illness using laboratory and physiologic data in the form of baseline SOFA score are presented by estimates in solid blue and hollow red. "Other" category refers to GNB encounters not classified as either DTR, ECR or FQR. CR= Carbapenem resistant; DTR=Difficult to treat resistance; ECR= Extended-spectrum cephalosporin-resistant; FQR= Fluoroquinolone resistant.

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1164. County-Level Geographic Distribution of Extended-Spectrum Cephalosporin-Resistant Enterobacteriaceae Across Outpatient Settings of the Veterans Health Administration, 2000–2017

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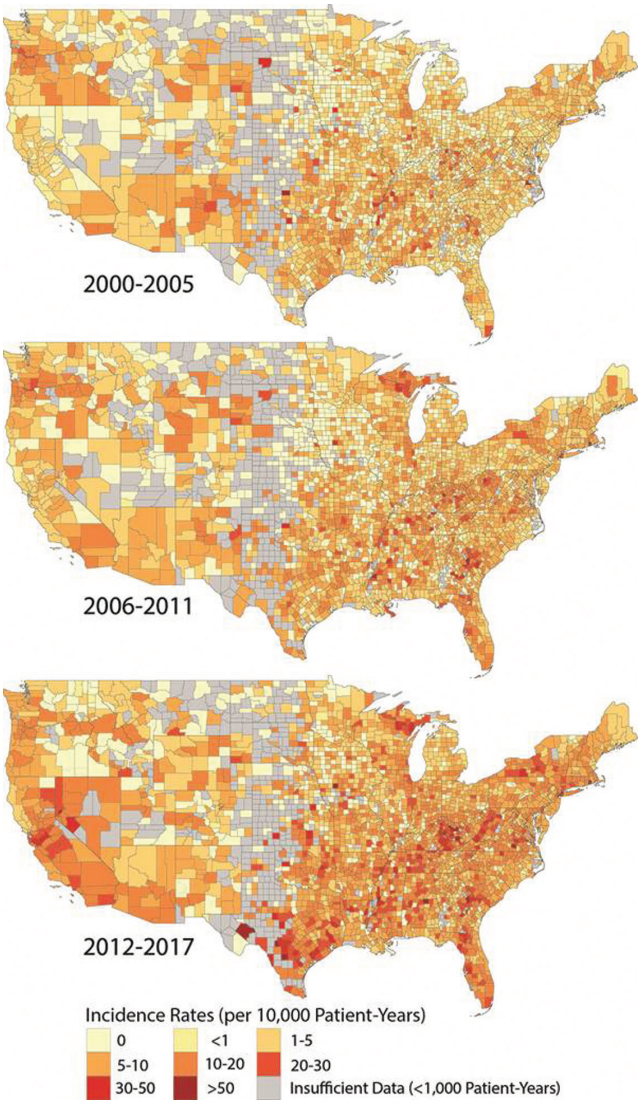
Background. Extended-spectrum cephalosporin resistance (ESCR) among Enterobacteriaceae has emerged globally over the last two decades, with increased prevalence in the community. Data from European countries and healthcare-associated isolates in the United States have demonstrated substantial geographic variability in the prevalence of ESCR, but community-onset isolates in the United States have been less studied. We aimed to describe geographic distribution and spread of ESCR among outpatient settings across the Veterans Health Administration (VHA) over 18 years.

Methods. We analyzed a retrospective cohort of all patients who had any positive clinical culture specimen for ESCR Enterobacteriaceae collected in an outpatient setting; ESCR was defined by phenotypic nonsusceptibility to at least one extended-spectrum cephalosporin agent or detection of an extended-spectrum β-lactamase. Patient-level data were grouped by county of residence, and the total number of unique patients who received care within VHA for each county was used as a denominator. We aggregated data by time terciles (2000–2005, 2006–2011, and 2012–2017), and overall and county-level incidence rates were calculated as the number of unique patients in each year with ESCR Enterobacteriaceae per person-year.

Results. During the study period, there were 1,980,095 positive cultures for Enterobacteriaceae from 870,797 unique patients across outpatient settings of VHA, from a total of 107,404,504 person-years. Among those, 136,185 cultures (6.9%) from 75,500 unique patients (8.7%) were ESCR. The overall incidence rate was 9.0 cases

per 10,000 person-years, which increased from 6.3 per 10,000 person-years in 2000 to 14.6 per 10,000 person-years in 2017. County-level incidence rates ranged widely but increased overall (interquartile range [IQR] in 2000–2005: 0–6.7; 2006–2011: 0–9.1; 2012–2017: 3.1–14.3 per 10,000 person-years), with some geographic clustering (figure).

Conclusion. This study demonstrates that there has been geographic variation both in incidence rates and trends of ESCR Enterobacteriaceae in outpatient settings of VHA, which suggests the importance of tailoring local antibiotic-prescribing guidelines incorporating geographic variability in epidemiology.



Disclosures. **M. Ohl**, Gilead Sciences, Inc.: Grant Investigator, Research grant.

1165. Comparing Patient Risk Factors, Sequence Type, and Resistance Loci Identification Approaches for Predicting Antibiotic Resistance in *Escherichia coli* Bloodstream Infections

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Background. To improve the adequacy of empiric antibiotic therapy, an important predictor of clinical outcome, rapid diagnostic tests of antibiotic resistance are increasingly being developed that identify the presence or absence of antibiotic resistance genes/Loci. Few approaches have utilized other sources of predictive information, which could be identified in shorter time periods, including patient epidemiologic risk factors for antibiotic resistance and markers of lineage (e.g., sequence type).

Methods. Using a dataset of 414 *Escherichia coli* isolated from separate episodes of bacteremia at a single academic institution in Toronto, Canada between 2010 and 2015, we compared the potential predictive ability of three approaches (epidemiologic, sequence type, and gene identification) for classifying antibiotic resistance to three commonly used classes of broad-spectrum antibiotic therapy (third-generation cephalosporins, fluoroquinolones, and aminoglycosides). We used logistic regression models with binary predictor variables to generate model receiver operating characteristic curves. Predictive discrimination was measured using apparent and corrected (bootstrapped) area under the curves (AUCs).

Results. Using two simple epidemiologic risk factors (prior antibiotic exposure and recent prior Gram-negative susceptibility), modest predictive discrimination was achieved (AUCs 0.65–0.74). Sequence type demonstrated strong discrimination (AUCs 0.84–0.94) across all three antibiotic classes. Epidemiologic risk factors significantly improved sequence-type prediction for cephalosporins and aminoglycosides ($P < 0.05$). Gene identification approaches provided the highest degree of discrimination (AUCs 0.73–0.99), with no statistically significant benefit of adding epidemiologic predictors.

Conclusion. Rapid identification of sequence type, or other lineage-based classification, could produce excellent discrimination of antibiotic resistance, and may be improved by incorporating readily available epidemiologic predictors.

TABLE 1: Antibiotic resistance prediction performance, as measured by area under the curve (AUC), for gene and sequence typing based approaches with or without the addition of epidemiologic risk factors for resistance.

	With Epidemiologic Predictors			Without Epidemiologic Predictors		
ANTIBIOTIC MODEL (N=414)	**Apparent AUC**	**AUC 95% CI**	**Corrected AUC**	**Apparent AUC**	**AUC 95% CI**	**Corrected AUC**
CEFTRIAXONE (Tested=396)						
Baseline (Epi predictors Alone)	0.74	0.66-0.82	0.72	-	-	-
Sequence Type**	0.88	0.83-0.93	0.85	0.83	0.78-0.88	0.81
Beta-lactamases						
TEM	0.73	0.64-0.81	0.7	0.49	0.41-0.56	0.47
TEM+CTX	0.94	0.88-0.99	0.91	0.91	0.85-0.96	0.9
TEM+CTX+OXA	0.94	0.88-0.99	0.91	0.91	0.85-0.97	0.9
TEM+CTX+OXA+CMY	0.98	0.96-1	0.97	*	*	*
TEM+CTX+OXA+CMY+SHV	*	*	*	*	*	*
FLUOROQUINOLONE (Tested=414)						
Baseline (Epi predictors alone)	0.68	0.63-0.73	0.67	-	-	-
Sequence Type	0.95	0.94-0.97	0.94	0.94	0.91-0.96	0.93
GyrA + ParC Mutations	0.99	0.98-1	0.99	0.99	0.98-1	0.99
GENTAMICIN (Tested=414)						
Baseline (Epi predictors Alone)	0.65	0.58-0.72	0.64	-	-	-
Sequence Type**	0.87	0.82-0.92	0.83	0.84	0.79-0.89	0.8
Aminoglycoside Acyl-transferases						
aac3	0.96	0.93-1	0.96	*	*	*
aac3 + aac6	0.96	0.93-1	0.96	0.94	0.90-0.99	0.94
aac3 + aac6 + aadB	0.98	0.95-1	0.97	0.97	0.94-1	0.97

*Model instability given highly predictive explanatory variable.
**Significant difference between With Epidemiologic Predictors and Without Epidemiologic Predictors AUC (p<0.05).

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

1166. Development of a Bedside Tool to Predict the Probability of Drug-Resistant Pathogens Among an Adult Population With Gram-Negative Infections

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Background. Identification of infections caused by antimicrobial-resistant microorganisms is critical to administration of early appropriate antibiotic therapy. We developed a clinical bedside tool to estimate the probability of carbapenem-resistant Enterobacteriaceae (CRE), extended spectrum β-lactamase-producing Enterobacteriaceae (ESBL), and multidrug-resistant *Pseudomonas aeruginosa* (MDRP) among hospitalized adult patients with Gram-negative infections.

Methods. A retrospective observational study of the Premier Hospital Database (PHD) was conducted. The study included adult hospitalized patients with complicated urinary tract infection (cUTI), complicated intraabdominal infection (cIAI), bloodstream infections (BSI), or hospital-acquired/ventilator-associated pneumonia (HAP/VAP) with a culture-confirmed Gram-negative infection in PHD from 2011 to 2015. Model development steps are shown in Figure 1. The study population was split into training and test cohorts. Prediction models were developed using logistic regression in the training cohort (Figure 1). For each resistant phenotype (CRE, ESBL, and MDRP), a separate model was developed for community-acquired (index culture ≤3 days of admission) and hospital-acquired (index culture >3 days of admission) infections (six models in total). The predictive performance of the models was assessed in the training and test cohorts. Models were converted to a singular user-friendly interface for use at the bedside.

Results. The most important predictors of antibiotic-resistant Gram-negative bacterial infection were prior number of antibiotics, infection site, prior infection in the last 3 months, hospital prevalence of each resistant pathogen (CRE, ESBL, and MDRP), and age (Figure 2). The predictive performance was highly acceptable for all six models (Figure 3).

Conclusion. We developed a clinical prediction tool to estimate the probability of CRE, ESBL, and MDRP among hospitalized adult patients with community- and hospital-acquired Gram-negative infections. Our predictive model has been implemented as a user-friendly bedside tool for use by clinicians to predict the probability of resistant infections in individual patients, to guide early appropriate therapy.