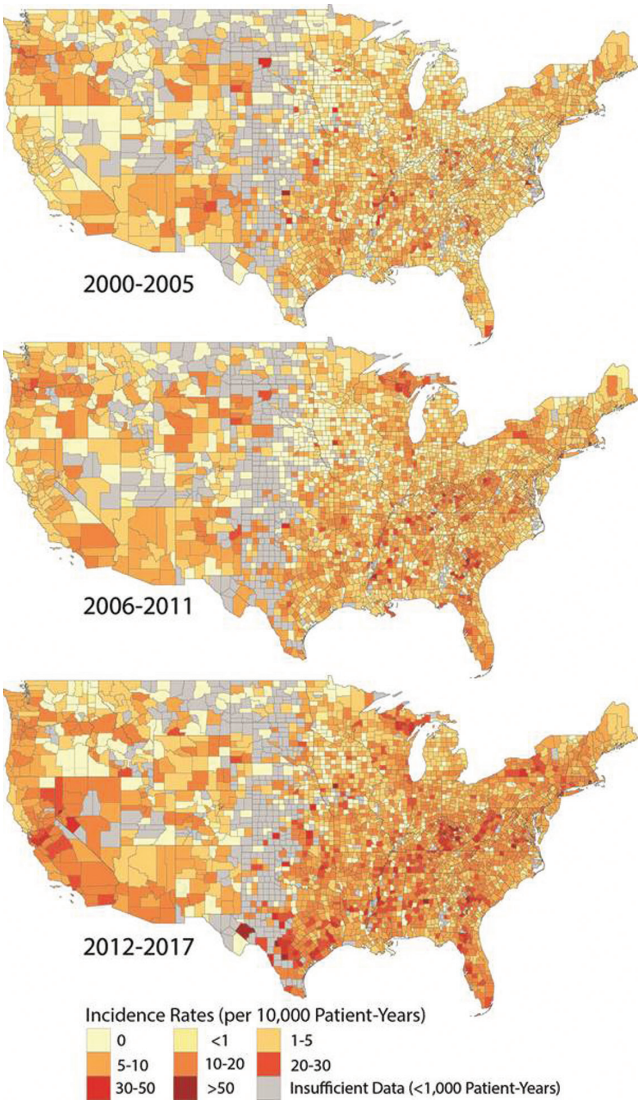
per 10,000 person-years, which increased from 6.3 per 10,000 person-years in 2000 to 14.6 per 10,000 person-years in 2017. County-level incidence rates ranged widely but increased overall (interquartile range [IQR] in 2000–2005: 0–6.7; 2006–2011: 0–9.1; 2012–2017: 3.1–14.3 per 10,000 person-years), with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



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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).

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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.