

(defined as non-susceptible to at least 1 of the following drugs: cefepime, ceftriaxone, ceftazidime, ceftolozane/tazobactam, ceftazidime/avibactam); CR = carbapenem resistance (defined as non-susceptible to at least 1 carbapenem); FR = fluoroquinolone resistance (defined as non-susceptible to at least 1 fluoroquinolone); AAPC = annual average percentage change; CI = confidence interval.

**Conclusion.** Overall, MDR, ESB, CR, and FR in *Enterobacteriales* and *P. aeruginosa* decreased from 2011 to 2020 in the VA. These results may be related to the robust infection control and antimicrobial stewardship programs instituted among VA Medical Centers nationally.

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**176. Antibiotic Resistance Patterns, Seasonality, and Correlation with the Influenza Season in the United States: A Multicenter Evaluation Reveals Surprising Association Between Influenza Season and Gram Negative Pathogens**  
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**Session:** O-35. Trends in Gram-negative Resistance

**Background.** Influenza infection may affect bacterial transmission dynamics and seasonality of antimicrobial resistance (AMR). There is a paucity of data on the association of influenza season and AMR rates. We aimed to describe trends of AMR and their correlation with the influenza season in ambulatory and inpatient settings in the United States (US).

**Methods.** We used the *BD Insights Research Database* (Franklin Lakes, NJ USA) to identify 30 day non-duplicate isolates collected from patients >17 years old with susceptibility profile of Gram-negative (GN) (Enterobacteriales (ENT), *P. aeruginosa* (PSA), *A. baumannii* spp. (ACB), and *S. maltophilia* (Sm)) and Gram-positive (GP) pathogens (*S. aureus* (SA), and *S. pneumoniae* (Sp)) in up to 257 US healthcare institutions from 2011-19. We defined the outcomes as rates per 100 admissions and % of non-susceptibility (NS), stratified by community and inpatient settings, resistance type (resistance to carbapenem (Carb-NS), quinolone (FQ-NS), macrolide (Macr NS), penicillin (PCN NS), and extended spectrum cephalosporin (ESC NS)) and isolate origin (respiratory and non-respiratory). Influenza data were presented as the % of positive laboratory tests. We used descriptive statistics and generalized estimating equations models to evaluate the monthly trends of AMR outcomes and correlation with the influenza season.

**Results.** We identified 16 576 274 confirmed non-duplicate pathogens, of which 154 841 were GN Carb-NS, 1 502 796 GN FQ-NS, 498 012 methicillin resistant SA (MRSA), and 44 131 Macr-NS, PCN-NS, and ESC-NS Sp. Among the Carb-NS pathogens, Influenza rate was correlated with % ACB-NS [ $\beta=0.205, p<.001$ ]. In the FQ-NS group, influenza was associated with overall % ENT-NS [ $\beta=0.041, p<.001$ ] and % PSA-NS [ $\beta=0.039, p=.015$ ]. For the GP pathogens, all Sp. rates were correlated with increased influenza positivity % (See Table). Only MRSA rates of respiratory source were associated with influenza [ $\beta=.066, p=.028$ ].

Summary of Multivariate regressions of AMR and % Flu by Source and Setting (controlling for hospital level factors): 2011-2019

	Overall	Respiratory Source	Non-Respiratory Source	Outpatient	Inpatient
% Carb-NS ACB	.205 (<.001)***	.379 (<.001)***	.134 (.040)*	.123 (.077)+	.255 (<.001)***
% FQ-NS ENT	.041 (<.001)***	.130 (<.001)***	.031 (.030)*	.018 (.043)+	.048 (<.001)***
% FQ-NS PSA	.039 (.015)*	.022 (.036)*	.020 (.087)+	.032 (.172)	.044 (.015)*
MRSA Rate/ 100 Adm	.060 (.615)	.066 (.028)*	-.087 (.065)+		
Macr NS S pneumo Rate/ 100 Adm	.464 (<.001)***	.253 (<.001)***	.068 (.376)		
PCN NS S pneumo Rate/100 Adm	.062 (.011)*	.056 (.046)*	.044 (.103)		
ESC NS S pneumo Rate/100 Adm	.033 (.036)*	.032 (.012)*	.018 (.073)+		

Data in each cell is presented as the coefficient ( $\beta$ ) and p-value is in parentheses. ^adjusted for region, teaching, urban, bed size, and season. + p<.10 \*p<.05 \*\*p<.01 \*\*\*p<.001

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**Conclusion.** Our study revealed surprising association between influenza epidemics and GN resistance and corroborated the evidence of correlation between respiratory GP and influenza infections. These insights may help inform targeted antimicrobial stewardship initiatives during influenza season.

**Disclosures.** Amine Amiche, PhD, Sanofi (Employee, Shareholder) Heidi Kabler, MD, Sanofi Pasteur (Employee) Janet Weeks, PhD, Becton, Dickinson and Company (Employee) Kalvin Yu, MD, BD (Employee) Vikas Gupta, PharmD, BCPS, Becton, Dickinson and Company (Employee, Shareholder)

**177. Distinctive Features of Ertapenem Mono-Resistant Carbapenem-Resistant Enterobacteriales in the United States: A Cohort Study**

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**Session:** O-35. Trends in Gram-negative Resistance

**Background.** Carbapenem-resistant Enterobacteriales (CRE) are highly antibiotic-resistant bacteria. Whether CRE resistant only to ertapenem among carbapenems (ertapenem mono-resistant) represent a unique CRE subset with regards to risk factors, carbapenemase genes, and outcomes is unknown.

**Methods.** We analyzed laboratory- and population-based surveillance data from nine sites participating in CDC's Emerging Infections Program (EIP). We defined an incident case as the first isolation of *Enterobacter cloacae* complex, *Escherichia coli*, *Klebsiella aerogenes*, *K. oxytoca*, *K. pneumoniae*, or *K. variicola* resistant to doripenem, ertapenem, imipenem, or meropenem (determined at clinical laboratory) from a normally sterile site or urine identified from a resident of the EIP catchment area in 2016-2017. We compared risk factors, carbapenemase genes (determined via polymerase chain reaction at CDC), and mortality of cases with ertapenem "mono-resistant" to "other" CRE (resistant to  $\geq 1$  carbapenem other than ertapenem). We additionally conducted survival analysis to determine the effect of ertapenem mono-resistant status and isolate source (sterile vs. urine) on survival.

**Results.** Of 2009 cases, 1249 (62.2%) were ertapenem mono-resistant and 760 (37.8%) were other CRE (Figure 1). Ertapenem mono-resistant CRE cases were more frequently  $\geq 80$  years old (29.1% vs. 19.5%,  $p<0.0001$ ), female (67.9% vs. 59.0%,  $p<0.0001$ ), and white (62.6% vs. 45.1%,  $p<0.0001$ ). Ertapenem mono-resistant isolates were more likely than other CRE to be *Enterobacter cloacae* complex (48.4% vs. 15.4%,  $p<0.0001$ ) but less likely to be isolated from a normally sterile site (7.1% vs. 11.7%,  $p<0.01$ ) or have a carbapenemase gene (2.4% vs. 47.4%,  $p<0.0001$ ) (Figure 2). Ertapenem mono-resistance was not associated with difference in 90-day mortality (unadjusted odds ratio [OR] 0.82, 95% confidence interval [CI] 0.63-1.06) in logistic models or survival analysis (Figure 3).

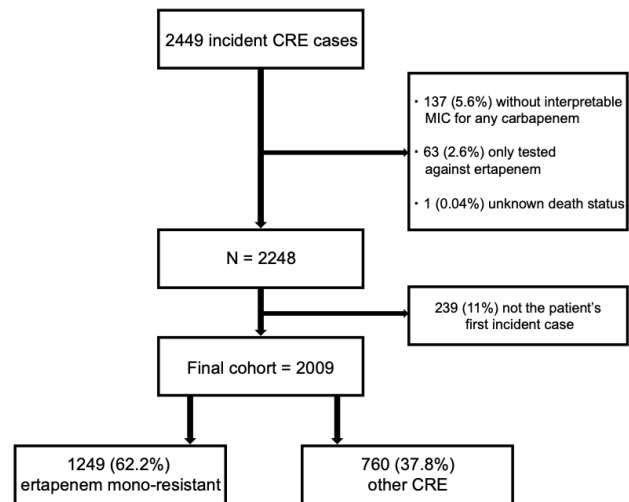


Figure 1. Flow diagram of carbapenem-resistant Enterobacteriales cases included in analysis, 2017-2018. CRE, carbapenem-resistant Enterobacteriales; MIC, minimum inhibitory concentration. Ertapenem mono-resistant CRE are only resistant to ertapenem (among carbapenems). Other CRE are resistant to  $\geq 1$  carbapenem other than ertapenem. We excluded isolates that (1) had no interpretable MICs for any carbapenem, (2) were only tested against ertapenem, (3) had unknown death status, or (4) were not associated with patient's first incident case.

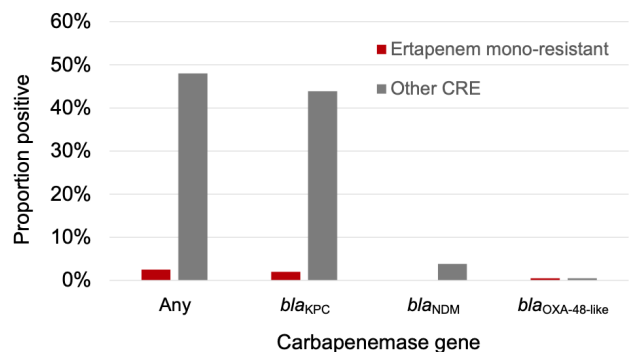


Figure 2. Proportion of ertapenem mono-resistant carbapenem-resistant Enterobacteriales (CRE) vs. other CRE isolates with specific carbapenemase genes. KPC, Klebsiella pneumoniae carbapenemase; NDM, New Delhi metallo- $\beta$ -lactamase; OXA, oxacillinase. Ertapenem mono-resistant carbapenem-resistant Enterobacteriales (CRE) are only resistant to ertapenem (among carbapenems). Other CRE are resistant to  $\geq 1$  carbapenem other than ertapenem. Testing via reverse transcriptase polymerase chain reaction.