(defined as non-susceptible to at least 1 of the following drugs: cefepime, ceftriaxone, cefotaxime, cefoxolone/tazoabactam, 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, ESBL, CR, and FR in Enterobacteriaceae and B. 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.

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

**Background.** Influenza infection may affect bacterial transmission dynamics and subsequently influence 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) Enterobacteriaceae (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 2011-2019. 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 source (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 of positive laboratory tests. We used logistic regression to determine the effect of influenza season and setting on the outcome of interest. The effect of ertapenem mono-resistant status and Flu source (respiratory vs. urine) on survival was achieved using Cox proportional hazards regression.

**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 [β= 0.205, p<.001]. In the FQ-NS group, influenza was associated with overall % ENT-NS [β= 0.041, p<.001] and % PSA-NS [β= 0.039, p<.015]. For the GP pathogens, all Sp rates were correlated with influenza [β= -0.066, 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  | 225.6 (1.001)*** | 379.0 (1.001)*** | 134.0 (1.001)*** | 123.0 (1.077)*** | 25.0 (1.001)*** |
| % FQ-NS ENT    | 0.016 (1.001)*** | 0.131 (1.001)*** | 0.011 (1.001)*** | 0.018 (1.048)*** | 0.048 (1.013)*** |
| % FQ-NS PSA    | 0.09 (1.015)*** | 0.232 (1.060)*** | 0.230 (1.075)*** | 0.127 (1.177)*** | 0.041 (1.015)*** |
| MRSA Rate/100 Adm | 0.005 (1.015) | 0.006 (1.025) | 0.007 (1.033) | 0.012 (1.035) | 0.016 (1.034) |
| Macr NS pneumo Rate/100 Adm | 0.005 (1.011) | 0.025 (1.013) | 0.010 (1.102) | 0.016 (1.053) | 0.009 (1.064) |
| PCN NS pneumo Rate/300 Adm | 0.006 (1.013) | 0.046 (1.046) | 0.046 (1.046) | 0.052 (1.053) | 0.041 (1.050) |
| ESC NS pneumo Rate/100 Adm | 0.002 (1.008) | 0.002 (1.007) | 0.002 (1.008) | 0.002 (1.007) | 0.002 (1.008) |

Data in each cell is presented as the coefficient (β) and p-value is in parentheses; adjusted for region, teaching, urban, bed size, and season. + p <0.05 ** p <0.01 *** p <0.001.

**Conclusion.** Our study revealed surprising association between influenza epidermis 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.

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

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

**Background.** Carbapenem-resistant Enterobacteriaceae (CRE) are highly antibiotic-resistant bacteria. Whether CRE resistant only to ertapenem among carbapenem-resistant (ertapenem mono-resistant) represent a unique CRE subset with regards to risk factors, carbapenem resistance, 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 pneumoniae, 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, carbapenem resistance, and outcomes (determined via polymerase chain reaction at CDC), and mortality of cases with ertapenem “mono-resistant” to “other” CRE (resistant to ≥ 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 ≥ 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 carbapenem gene (2.4% vs. 4.7%, 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).

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**Figure 1. Flow diagram of carbapenem-resistant Enterobacteriaceae cases included in analysis, 2017-2018. CRE, carbapenem-resistant Enterobacteriaceae; MIC, minimum inhibitory concentration. Ertapenem mono-resistant CRE are only resistant to eratapenem (among carbapenems). Other CRE are resistant to ≥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 Enterobacteriaceae (CRE) vs. other CRE isolates with specific carbapenemase genes. KPC, Klebsiella pneumoniae carbapenemase; NDM, New Delhi metallo-ß-lactamase; OXA, oxacillinase. Ertapenem mono-resistant carbapenem-resistant Enterobacteriaceae (CRE) are only resistant to ertapenem (among carbapenems). Other CRE are resistant to ≥1 carbapenem other than ertapenem.**

**Note:** All figures are presented in color in the online version of OFID.