We analyzed laboratory- and population-based surveillance data, Escherichia coli, Pfizer, Shionogi, Inc, and Company. These insights may help inform targeted anticrobials and GN resistance and corroborated the evidence of correlation between **p <.001** ^adjusted for region, teaching, urban, bed size, and season. + p<.10 *p <.05 **p <.01***p <.001 **adjusted for region, teaching, urban, bed size, and season. + p<.10 *p <.05 **p <.01***p <.001

**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 anti-microbial stewardship initiatives during influenza season.

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

**Background.** Influenza infection may affect bacterial transmission dynamics and seasonal antimicrobial resistances (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) (Enterobacterales (ENT), P. aeruginosa (PSA), E. coli (ACB), and S. maltophilia (Sm)) and Gram-positive (GP) pathogens (Staph. 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 (QF-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 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 QF-NS, 498 012 methicillin resistant SA (MRSA), and 44 131 Macr-NS, PCN-NS, and ESC-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 = 0.001] and % PSA-NS [β=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 [β= .066, p=.028].

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

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

**Background.** Carbapenem-resistant Enterobacterales (CRE) are highly antibiotic-resistant bacteria. Whether CRE resistant only to imipenem among carbapenem (ertapenem mono-resistant) represent a unique CRE subset with regards to risk factors, carbapenem 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 pneumoniae, K. oxytoca, K. pneumoniae, or K. variicola resistant to doripenem, 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 genes (determined via polymerase chain reaction at CDC), and mortality of cases with etrapenem "mono-resistant" to "other" CRE (resistant to ≥ 1 carbapenem other than ertapenem). We additionally conducted survival analysis to determine the effect of etrapenem mono-resistant status and isolate source (sterile vs. urine) on survival.

**Results.** Of 2009 cases, 1249 (62.2%) were etrapenem 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 (71.1% vs. 11.7%, p< 0.01) or have a carbapenem 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).

![Flow diagram of carbapenem-resistant Enterobacterales cases included in analysis, 2017-2018. CRE, carbapenem-resistant Enterobacterales; MRC, minimum inhibitory concentration. Ertapenem mono-resistant CRE are only resistant to etrapenem (among carbapenem). Other CRE are resistant to ≥1 carbapenem other than etrapenem. 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.](Image 311x135 to 542x264)

**Figure 1.** Flow diagram of carbapenem-resistant Enterobacterales cases included in analysis, 2017-2018. CRE, carbapenem-resistant Enterobacterales; MRC, minimum inhibitory concentration. Ertapenem mono-resistant CRE are only resistant to etrapenem (among carbapenem). Other CRE are resistant to ≥1 carbapenem other than etrapenem. 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.

![Proportion of etrapenem mono-resistant carbapenem-resistant Enterobacteriales (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 Enterobacterales (CRE) are only resistant to etrapenem (among carbapenem). Other CRE are resistant to ≥1 carbapenem other than etrapenem. Testing via reverse transcriptase polymerase chain reaction.](Image 311x333 to 542x520)

**Figure 2.** Proportion of etrapenem mono-resistant carbapenem-resistant Enterobacterales (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 Enterobacterales (CRE) are only resistant to etrapenem (among carbapenem). Other CRE are resistant to ≥1 carbapenem other than etrapenem. Testing via reverse transcriptase polymerase chain reaction.
Ertapenem mono-resistant isolates were not associated with decreased mortality, and sterile isolate source (i.e., non-urinary isolates) was associated with increased mortality regardless of ertapenem mono-resistance.

Conclusion. Ertapenem mono-resistant CRE rarely have carbapenemase genes and have distinct clinical and microbiologic characteristics compared to other CRE. These findings may inform antibiotic choice particularly when testing for carbapenemases is not readily available.

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178. Endemic Carbapenem Resistance Driven By Clonal and Horizontal Spread of blaIMP-4 Across Diverse Enterobacteriales: Jumping Genes, Promiscuous Plasmids and Killer Clones
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Session: O-35. Trends in Gram-negative Resistance

Background. Carbapenem-resistant Enterobacteriales (CRE) have become endemic and cause significant morbidity and mortality globally. The metallo-beta-lactamase gene blaIMP-4 is a key CRE resistance determinant in Australia and Asia but its genomic context remains unknown. We aimed to determine the genomic epidemiology of blaIMP-4 in clinical and environmental isolates from 2008 – 2020 at our institution.

Methods. We performed whole genome sequencing on 219 blaIMP-4-carrying isolates from 134 patients (219 short-read and 75 long-read). Multi-locus sequence types (MLSTs), resistance determinants and plasmid replications were assessed. High-quality de novo hybrid assemblies were used to identify location of blaIMP-4 gene. We conducted phylogenetic analysis for key MLSTs and plasmids.

Results. blaIMP-4 was noted on a class I integron also harboring aminoglycoside, sulfamethoxazole, chloramphenicol and quaternary ammonium compound resistance genes. Presence of blaIMP-4 on diverse plasmids that varied through the study period was noted. Plasmids were characterised by analysing de novo hybrid assembly data and co-location of blaIMP-4 and plasmid replications on the same contigs.

Conclusion. blaIMP-4 spread on a class I integron was responsible for endemic carbapenem resistance at our institution. This mobile genetic element was able to persist due to both clonal spread and entry into diverse plasmids. Concerningly, we noted a large outbreak driven by IncHI2 plasmids harboring colistin resistance genes with spread to multiple bacterial species.

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179. Identification and Whole Genome Sequencing Analysis of an Oxacillinase (OXA)-48-like-producing Acinetobacter baumannii Outbreak in California
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Session: O-35. Trends in Gram-negative Resistance

Background. In January 2021, a California acute care hospital (ACH A), a sentinel site for Acinetobacter baumannii (AB) surveillance, identified OXA-48-like-carbapenemase producing (CP) AB in a patient admitted from a ventilator-equipped skilled nursing facility (vSNF A). OXA-48-like AB had not been previously reported in the United States.

Methods. Our investigation included onsite infection control (IC) assessments, contact tracing, and point prevalence surveys (PPS) at vSNF A. The Antibiotic Resistance (AR) Laboratory Network performed carbapenemase testing on AB isolates (including those from ACH A) and PPS swabs. A case was defined as a patient with an OXA-48-like AB isolate, or an epidemiologically-linked patient with an OXA-48-like gene detected via screening. We performed whole genome sequencing (WGS) of OXA-48-like AB and other CP organisms on the Illumina MiSeq and Oxford Nanopore MinION for short and long read sequencing, respectively.

Results. Since January 2021, we have identified five OXA-48-like AB cases (including the index), six OXA-48-like cases (no organism recovered), and six patients with other CP organisms at ACH A and vSNF A. Since August 2019, vSNF A has concurrently been experiencing an OXA-109 AB outbreak. A second vSNF A patient, Patient 2, who overlapped with the index patient, had OXA-48-like Klebsiella pneumoniae (KP) (November 2019) and OXA-109 AB (May 2020) isolates. WGS of the index patient's AB and Patient 2's KP isolates identified a rare OXA-48-like gene located on the AB chromosome and a KP plasmid. The OXA-48-like AB was also carrying an OXA-109 gene, and hapSNP analysis indicated it varied by 9-44 single-nucleotide polymorphisms (SNPs) from 14 OXA-109 AB isolates linked to that outbreak, and 0-3 SNPs from the other OXA-48-like AB case isolates.

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