Conclusions. GBS patients with evidence of ZIKV infection were clinically similar to those without evidence of ZIKV infection, but more likely to have facial weakness and paresthesia during acute neurologic illness and report abnormal tear production after treatment with CT. Minimum inhibitory concentrations (MICs) to CT were determined by Etest. Resistance mediated by AmpC hyperproduction was evaluated and after treatment with CT. Of 18.4% (75/408) in the post PCV-10 and PCV13 years (2011–2015). The resistance frequencies to penicillin, cefotaxime, erythromycin, clindamycin, and tetracycline increased significantly after 2000 and were greater than 20% (21/103) in 2006–2008 compared to 8.5% from 3.9% to 21.2% and 21.2% to 46.1% after the introduction of PCV7 years (1986–2005) to 19.6% (88/449) in the post-PCV7 years (2006–2010) and 18.4% (75/408) in the post PCV-10 and PCV13 years (2011–2015). The resistance frequencies to penicillin, cefotaxime, erythromycin, clindamycin, and tetracycline increased significantly after 2000 and were greater than 20% (21/103) in 2006–2008 compared to 8.5% from 3.9% to 21.2% and 21.2% to 46.1% after the introduction of PCV7 years (1986–2005) to 19.6% (88/449) in the post-PCV7 years (2006–2010) and 18.4% (75/408) in the post PCV-10 and PCV13 years (2011–2015). The resistance frequencies to penicillin, cefotaxime, erythromycin, clindamycin, and tetracycline increased significantly after 2000 and were greater than 20% (21/103) in 2006–2008 compared to 8.5% from 3.9% to 21.2% and 21.2% to 46.1% after the introduction of PCV7 years (1986–2005) to 19.6% (88/449) in the post-PCV7 years (2006–2010) and 18.4% (75/408) in the post PCV-10 and PCV13 years (2011–2015). The resistance frequencies to penicillin, cefotaxime, erythromycin, clindamycin, and tetracycline increased significantly after 2000 and were greater than 20% (21/103) in 2006–2008 compared to 8.5% from 3.9% to 21.2% and 21.2% to 46.1% after the introduction of PCV7 years (1986–2005) to 19.6% (88/449) in the post-PCV7 years (2006–2010) and 18.4% (75/408) in the post PCV-10 and PCV13 years (2011–2015). The resistance frequencies to penicillin, cefotaxime, erythromycin, clindamycin, and tetracycline increased significantly after 2000 and were greater than 20% (21/103) in 2006–2008 compared to 8.5% from 3.9% to 21.2% and 21.2% to 46.1% after the introduction of PCV7 years (1986–2005) to 19.6% (88/449) in the post-PCV7 years (2006–2010) and 18.4% (75/408) in the post PCV-10 and PCV13 years (2011–2015).
using ceftazidime (CAZ) and meropenem (MER) with and without clavulanic acid (CLOX) at concentration of 1 mg/mL. In addition, the β-lactamase hydrolysis activity was determined for crude cell lysate of the isolates using a spectrophotometric assay for nitrocefin degradation. Furthermore, whole genome sequencing of the three strains was performed and compared (2365 vs. 2366 and 2367). Reads from each isolate were mapped against the genome of the reference strain F01.01. Variants identified by GATK, SAMTools and CLC Genomics Workbench 8.5 were selected and annotated with Snpeff.

**Results.** Strain 2365 had a CT MIC of 0.75/25 mg/mL while 2366 and 2367 have MICs > 256 mg/mL. AmpC hyperproduction test was positive only for the susceptible isolate (2365). In concordance, the hydrolysis assay showed a weak decrease in hydrolysis by degradation by RT-2 2366 compared with its CT-susceptible isolate 2365. Notably, the three strains (S and R) exhibited a truncated AmpD. Comparison of the resistant derivatives vs. 2365 and 2367 showed a 7 amino acid deletion in the β-loop of the β-lactamase AmpC in both resistant derivatives as well as mutations in genes predicted to encode a hypothetical protein, an ABC transporter ATP-binding protein and a multidrug resistance operon repressor MexR.

**Conclusion.** Our results suggest that the deletion in the β-loop of AmpC in 2366 and 2367 does not contribute to CT in these P. aeruginosa strains. Further characterization of AmpC and other predicted identified proteins by WGS are needed to determine the mechanism of CT-R.

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

### 318. Reduced Ertapenem Susceptibility Due to OXA-2 Production in Klebsiella pneumoniae ST410

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**Session:** 51. Emerging Resistance - Epidemiology and Mechanisms

**Background.** OXA-2 is a class D β-lactamase which is primarily found in E. coli (Ec) and P. aeruginosa and confers resistance to penicillins as well as narrow-spectrum cephalosporins. However, recent reports suggest that OXA-2 also possesses carbapenemase activity. We report a case of K. pneumoniae that confers reduced susceptibility to ceftazidime and ertapenem due to production of OXA-2.

**Methods.** K. pneumoniae (Kp) strain YD7C87 was identified from the BAL culture of an inpatient in December 2016. The strain was initially reported as intermediate to ertapenem and the pmrA2 gene encoding quinolone resistance. The pmrA2 gene was located on an FIA/FIB plasmid of ~100 Kb, in association with ISΔ44125, and upstream of a putative blomycin resistance gene, a conserved arrangement among NDM expressing Gram-negative organisms. Cell lysate assays showed decreased to carbapenemase activity with increasing concentrations of EDTA and an increase in activity with the addition of zinc, suggesting the NDM-5 metallo-β-lactamase is largely responsible for the observed carbapenemase activity. Comparison with plasmid sequences available suggested convergence of resistance determinants captured from a wide geographic area.

**Conclusion.** Plasmid-mediated spread of β-lactamases among Enterobacteriaceae is a rapidly evolving threat, with the introduction of NDM-5 and OXA-181 in the United States being a particularly disturbing development. Introduction of multidrug-resistant carbapenemase resistance may change the landscape of antimicrobial resistance in the United States.

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

### 320. Characterization of Enterobacter and Citrobacter spp. Isolates from United States Hospitals by Whole-Genome Sequencing Analysis and Activity of Ceftazidime-Avibactam and Comparator Agents

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**Session:** 51. Emerging Resistance - Epidemiology and Mechanisms

**Background.** Enterobacter spp. and Citrobacter spp. are common pathogens in a variety of clinical infections. These organisms can overexpress the chromosomal AmpC that impairs resistance to several β-lactams. Additionally, these isolates may carry acquired BL genes. We evaluated the presence of BL and the activity of ceftazidime-avibactam (CAZ-AVI) among 410 isolates collected in US hospitals during 2016.

**Methods.** In total, 258 E. cloaceae (ECL), 81 E. aerogenes, 70 C. freundii, and 1 C. koseri displaying MIC values ≤16 µg/mL for CAZ and/or ≤2 µg/mL for ceftazidime were submitted to WGS, de novo assembly and screening for BL genes using an in-house-developed pipeline.

**Results.** The most common acquired BL gene was blaoxa-23 (25 isolates, 20 ECL) and included blaoxa-23-(19 isolates) and six other variants. EBL blaoxa-23 (six variants) was noted among 39 isolates and blaoxa-23-(20 isolates) and blaoxa-23-(3) were also noted. BlaOXA-23 carrying ECL were detected among 8 isolates (5 blaoxa-23, 1 blaoxa-23, and 1 blaoxa-23), in ECL. Carbapenemase-encoding genes detected 19 isolates included five blaoxa-23-11, blaoxa-23-1 and one each of blaoxa-23-10, blaoxa-23-11, and blaoxa-23-11 isolates carrying these genes were five C. freundii, one E. aerogenes, and 13 ECL. The blaoxa-23-carrying ECL exhibited meropenem, doripenem, and imipenem MIC values of 0.06, 0.12, and 0.5 µg/mL, respectively. The majority of the E. aerogenes isolates did not carry acquired BL. In total, 70 C. koseri carried carbapenemase activity, of which 41 isolates were resistant to CAZ-AVI while 29 isolates were susceptible (MIC ≤1 µg/mL). Comparison of the isolates, and two isolates were resistant to CAZ-AVI: one ECL carrying acquired BL genes. We evaluated the presence of BL and the activity of ceftazidime-avibactam (CAZ-AVI) among 410 isolates collected in US hospitals during 2016.

**Conclusion.** Altered MBLs were more frequent in ECL and were mostly blaoxa-23-1 or blaoxa-23-1. Carbapenemases were also detected. Cephalosporin resistance is likely due to over-expression of AmpC among E. aerogenes. CAZ-AVI was very active against these isolates, including most (17/19) carbapenemase producers.

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### 321. Frequency and Mechanisms of Spontaneous Fosfomycin Non-susceptibility Observed upon Disk Diffusion Testing of Escherichia coli

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**Session:** 51. Emerging Resistance - Epidemiology and Mechanisms

**Background.** Escherichia coli, the most common cause of urinary tract infections has become increasingly resistant to commonly used oral antibiotics. Fosfomycin maintains excellent activity against most E. coli clinical isolates. The growth of E. coli