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.

Disclosures. All Authors: No reported disclosures

178. Endemic Carbenapenem Resistance Driven By Clonal and Horizontal Spread of \textit{bla}\textsubscript{IMP-4} Across Diverse Enterobacteriales: Jumping Genes, Promiscuous Plasmids and Killer Clones

Nenad Macesic, MBBS MA PhD\textsuperscript{1}; Luke Blakeway, PhD\textsuperscript{2}; Adam W. Jenney, MBBS, FRACP, FRCPA, PhD\textsuperscript{3}; Anton Peleg, MBBS MPH PhD\textsuperscript{4}; Monash University / Alfred Hospital, Melbourne, Victoria, Australia; \textsuperscript{5}Alfred Health, Melbourne, Victoria, Australia

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

Background. Carbenapenem-resistant Enterobacteriales (CRE) have become endemic and cause significant morbidity and mortality globally. The metallo-beta-lactamase gene \textit{bla}\textsubscript{IMP-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 \textit{bla}\textsubscript{IMP-4} in clinical and environmental isolates from 2008 – 2020 at our institution.

Methods. We performed whole genome sequencing on 219 \textit{bla}\textsubscript{IMP-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 \textit{de novo} hybrid assemblies were used to identify location of \textit{bla}\textsubscript{IMP-4}. We conducted phylogenetic analysis for key MLSTs and plasmids.

Results. \textit{bla}\textsubscript{IMP-4} was noted on a class I integron also harboring aminoglycoside, sulfa, methoxazole, chloramphenicol and quaternary ammonium compound resistance genes. This integron was able to migrate over time to 10 bacterial species (42 STs) and 6 different plasmid types (Figure 1 and Figure 2). From 2008-2020, 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 \textit{IncHII} 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 \textit{Acinetobacter baumannii} Outbreak in California, January-May 2021

Diana Holden, MPH\textsuperscript{1}; Matthew Sylvestre, PhD\textsuperscript{2}; John Crandall, n/a\textsuperscript{3}; Fengfeng Xu, n/a\textsuperscript{3}; Emily C. Schneider, MPH\textsuperscript{4}; Hillary Berman Watson, MPH, MPH, PHD\textsuperscript{5}; Peng Zhang, PhD\textsuperscript{6}; Jaclyn Bucad, n/a\textsuperscript{2}; Rafael Mejia, n/a\textsuperscript{7}; Erinn Epson, MD\textsuperscript{1}; Zenda Berrada, PhD\textsuperscript{1}; Tisha Mutsuga, DrPH, ScM\textsuperscript{1}; Rituparna Mukhopadhyay, PhD\textsuperscript{1}; California Department of Public Health, Richmond, CA; \textsuperscript{8}Washington State Department of Health, Shoreline, Washington

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

Background. In January 2021, a California acute care hospital (ACH A), a sentinel site for \textit{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 \textit{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 hqSNP 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.

Figure 1. Phylogenetic Tree Comparison of OXA-109 AB and OXA-48-like AB Isolates

Presence of \textit{bla}\textsubscript{IMP-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 \textit{bla}\textsubscript{IMP-4} and plasmid replications on the same contigs.

Conclusion. \textit{bla}\textsubscript{IMP-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 \textit{IncHII} plasmids harboring colistin resistance genes with spread to multiple bacterial species.

Disclosures. AB Authors: No reported disclosures

Figure 3. Survival analysis comparing patients with carbapenem-resistant Enterobacteriales (CRE) that are ertapenem mono-resistant to other CRE (i.e., resistant to \( \geq 1 \) carbapenem other than ertapenem), either total (A) or stratified by isolates site (B).

Blamp-4 was noted in diverse bacterial species over the study period. Outbreaks of \textit{Enterobacter cloacae} complex ST114, ST190 and ST93 and \textit{Pseudomonas aeruginosa} ST111 were noted.