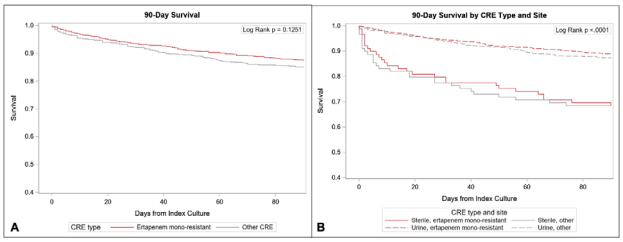
Figure 3. Survival analysis comparing patients with carbapenem-resistant Enterobacterales (CRE) that are ertapenem mono-resistant to other CRE (i.e., resistant to ≥1 carbapenem other than ertapenem), either total (A) or stratified by isolate site (B).



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 Carbapenem Resistance Driven By Clonal and Horizontal Spread of *bla*$_{IMP-4}$ Across Diverse Enterobacterales: Jumping Genes, Promiscuous Plasmids and Killer Clones

Nenad Macesic, MBBS MA PhD[1]; Luke Blakeway, PhD[2]; Adam W. Jenney, MBBS, FRACP, FRCPA, PhD[3]; Anton Peleg, MBBS MPH PhD[1]; [1]Monash University / Alfred Hospital, Melbourne, Victoria, Australia; [2]Alfred Hospital / Monash University, Melbourne, Victoria, Australia; [3]Alfred Health, Melbourne, Victoria, Australia

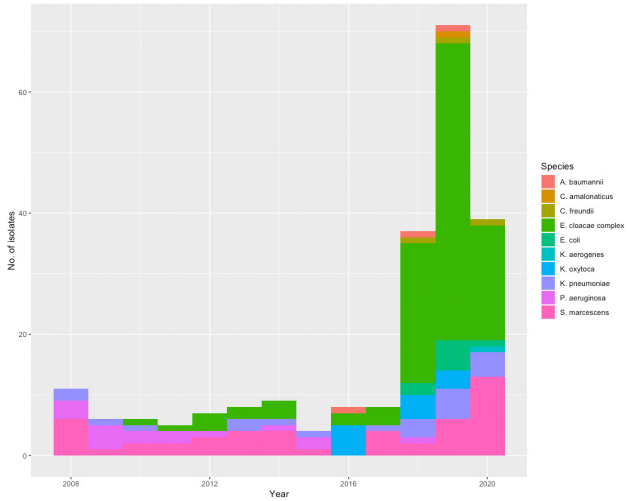
Session: O-35. Trends in Gram-negative Resistance

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

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

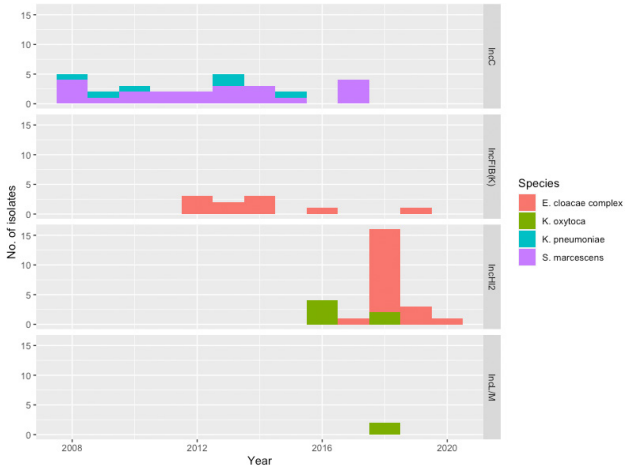
Results. *Bla*$_{IMP-4}$ was noted on a class I integron also harboring aminoglycoside, sulfamethoxazole, chloramphenicol and quaternary ammonium compound resistance genes. This integron was able to migrate over time to 10 bacterial species (42 STs) and 6 different plasmid types (Figure 1 and Figure 2). From 2008-2020, *bla*$_{IMP-4}$ was present on IncC plasmids in *Serratia marcescens* and *Klebsiella pneumoniae*. We noted small outbreaks of *Pseudomonas aeruginosa* ST111 with chromosomal integration of *bla*$_{IMP-4}$ from 2008-2018 (16 isolates) and *Enterobacter cloacae* complex ST114 with *bla*$_{IMP-4}$ on IncFIB(K)/IncFIA(HI1) plasmids from 2011-2020 (19 isolates). From 2016-2020, there was an explosion of diverse IncHI2 plasmids carrying *bla*$_{IMP-4}$. This was driven by clonal expansion of *E. cloacae* complex ST93/ST190 (79 isolates), with spillover of IncHI2 plasmids to *Klebsiella* spp (13 isolates), *Citrobacter* spp (2 isolates), *S. marcescens* (1 isolate), *Escherichia coli* (4 isolates). In addition to *bla*$_{IMP-4}$, these plasmids carried *mcr-9.1*, a colistin resistance gene, and resistance determinants to nearly all key classes of Gram-negative antimicrobials.

Figure 1. Bacterial species harboring blaIMP-4 2008-2020



BlaIMP-4 was noted in diverse bacterial species over the study period. Serratia marcescens and Klebsiella pneumoniae were present throughout. Outbreaks of Enterobacter cloacae complex ST114, ST190 and ST93 and Pseudomonas aeruginosa ST111 were noted.

Figure 2. Diverse plasmids associated with blaIMP-4 carriage determined by de novo hybrid assembly



Presence of blaIMP-4 on diverse plasmids that varied through the study period was noted. Plasmids were charaterised by analysing de novo hybrid assembly data and co-location of blaIMP-4 and plasmid replicons on the same contigs.

Conclusion. *Bla*$_{IMP-4}$ 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 IncHI2 plasmids harboring colistin resistance genes with spread to multiple bacterial species.

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

179. Identification and Whole Genome Sequencing Analysis of an Oxacillinase (OXA)-48-like-producing *Acinetobacter baumannii* Outbreak in California, January-May 2021

Diana Holden, MPH[1]; Matthew Sylvester, PhD[1]; John Crandall, n/a[1]; Fengfeng Xu, n/a[1]; Emily C. Schneider, MPH[2]; Hillary Berman Watson, PhD, MPH, PHM[1]; Peng Zhang, PhD[1]; Jaclyn Bacud, n/a[1]; Rafael Mejia, n/a[1]; Erin Epson, MD[1]; Zenda Berrada, PhD[1]; Tisha Mitsunaga, DrPH, ScM[1]; Rituparna Mukhopadhyay, PhD[1]; [1]California Department of Public Health, Richmond, CA; [2]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 *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 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

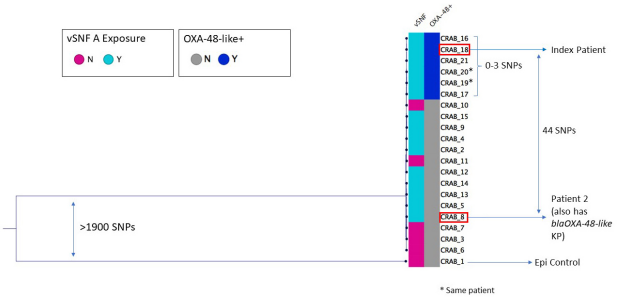
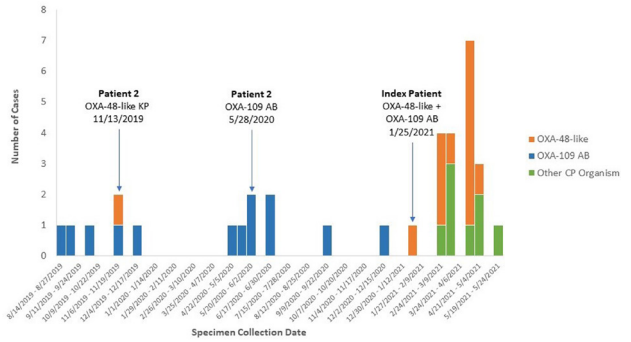


Figure 2. Epidemic Curve of OXA-109 AB, OXA-48-like AB, and Other CP Organism Cases, 2019-2021



Conclusion. The first reported case of OXA-48-like AB in the US was identified through public health sentinel laboratory surveillance, allowing prompt response to contain spread of a novel multidrug-resistant organism (MDRO). WGS detected a rare OXA-48-like gene in AB and KP and provides evidence for possible interspecies transfer of this gene from KP to AB through plasmid transfer followed by chromosomal integration.

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

180. Alterations to the Gut Microbiomes and Acquisition of Bacteria Resistance Elements among US International Travelers

Sushmita Sridhar, PhD[1]; Colin Worby, Ph.D.[2]; Ryan Bronson, B.S.[2]; Sarah Turbett[1]; Jason Harris, MD MPH[1]; Edward T. Ryan, MD[1]; Ashlee Earl, Ph.D.[2]; Regina C. LaRocque, MD, MPH[1]; [1]Massachusetts General Hospital, Boston, MA; [2]Broad Institute of MIT and Harvard, Cambridge, Massachusetts

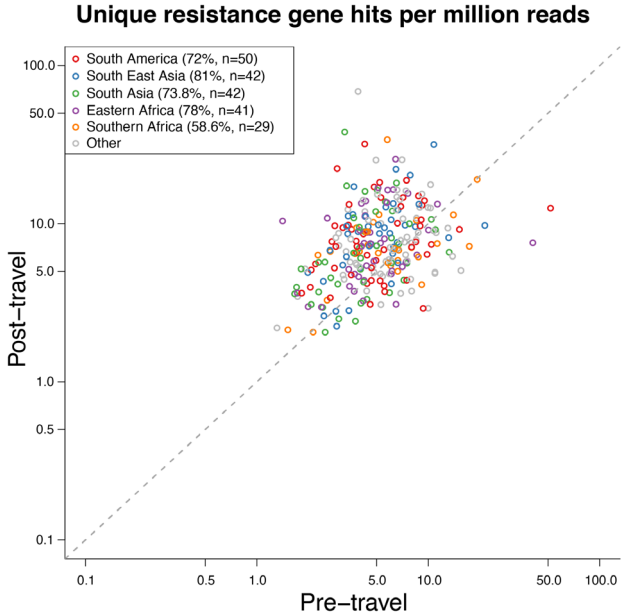
Session: O-35. Trends in Gram-negative Resistance

Background. This study investigated the impact of international travel on the acquisition and carriage of antimicrobial resistance (AMR). We prospectively assessed U.S. international travelers for the acquisition of resistant *Enterobacterales* species and evaluated changes in travelers' gut microbiomes.

Methods. Metagenomic sequencing was performed on DNA extracted from pre- and post-travel stool samples of 273 U.S. international travelers. We used Kraken2 to assess microbial gut composition and analyzed antibiotic resistance gene (ARG) content using the Resistance Gene Identifier (RGI) and ResFinder, and read mapping to ARG databases. We assessed the change in gut profile and resistome associated with (i) all international travel; (ii) travel to specific geographic regions; and (iii) traveler's diarrhea.

Results. International travel resulted in a perturbation of the gut microbiome, which was greater in travelers receiving treatment for diarrhea during travel (p = 4E-5). There was an overall loss in microbial diversity following travel, regardless of health outcome (p = 0.011); this was most consistently observed in travelers to South East Asia (SEA) (loss of gut diversity in 81% of SEA travelers). 78% of all travelers had a higher relative abundance of *E. coli* after travel, including 85% of travelers who acquired AMR bacteria during travel. Travel to South Asia was also associated with a significantly greater increase of *E. coli* relative to other destinations (p = 0.04). Additionally, the relative abundance of *Pasteurellales* was higher in the pre-travel samples of those who subsequently acquired AMR bacteria (FDR = 0.08). Furthermore, there was a significant increase in ARG content among the post-travel samples, with regional differences in the magnitude of acquisition (**Figure 1**). 72% of all travelers had a greater resistance burden post-travel. SEA was associated with the greatest increase in resistome diversity, while South America was associated with the greatest increase in overall ARG content.

Resistance genes present in the gut microbiome.



Genes mapping to the Comprehensive Antibiotic Resistance Database were measured pre- (x-axis) and post-travel (y-axis) to assess the acquisition of resistance genes in association with travel, distinguished by geographic region. Colors indicate geographic regions visited by travelers: South America (red), South East Asia (blue), South Asia (green), Eastern Africa (purple), Southern Africa (orange), Other (grey).

Conclusion. International travel is associated with a perturbation in the gut microbial community, with the acquisition of AMR bacteria and genes, and an increase in the relative abundance of *E. coli*. These perturbations following travel may be important factors in the global spread of AMR.

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

181. Potential Benefit of Masking and other COVID-19 Infection Prevention Measures on Late-Onset Infections in the NICU

Charlene Bultmann, MD[1]; Jaclyn Wiggins, MD[2]; Sagori Mukhopadhyay, MD[3]; Dustin Flannery, MD[4]; Mark Conaway, MD[2]; Miren Dhudasia, md[5]; Sam Garber, MD[4]; Joseph B. Cantey, MD[6]; Robert Schelonka, MD[7]; H Weitkamp, MD[8]; Kristin Weimer, MD[5]; Dipen Vyas, MD[9]; Margaret Gilfillan, MD[10]; Alison Carey, MD[9]; Julie Wohrley, MD[11]; Andrew Berenz, MD[11]; Sarah Khan, MD, MSc, FRCPC[12]; M Favara, MD[13]; D Tuttle, MD[13]; K Ziegler, MD[14]; E Chang, MD[14]; J Gaulton, MD[14]; Pablo J. Sanchez, MD[15]; David Kaufman, MD[1]; [1]University of Virginia, Charlottesville, Virginia; [2]UVA, Charlottesville, Virginia; [3]U PENN, Philadelphia, Pennsylvania; [4]UPENN, Phila, Pennsylvania; [5]DUKE, durham, North Carolina; [6]University of Texas Health San Antonio, San Antonio, TX; [7]Oregon, Oregan, Oregon; [8]Vanderbilt, Nashville, Tennessee; [9]St Chris, PHila, Pennsylvania; [10]St Christopher's, Phila, Pennsylvania; [11]Rush, Chicago, Illinois; [12]McMaster University, Hamilton, Ontario, Canada, Hamilton, Ontario, Canada; [13]Christiana, Wilmington, Delaware; [14]Abington, Abington, Pennsylvania; [15]Nationwide Children's Hospital - The Ohio State University, Columbus, Ohio

MASKING STUDY GROUP

Session: O-36. Trends in Pediatric Bacterial Disease

Background. Incidence of blood stream infections (BSI) among NICU admissions remains high, with associated mortality and morbidity. Due to COVID-19, there are increased infection prevention (IP) measures in NICUs including universal masking for all healthcare workers and families, social distancing, visitation restrictions, and increased attention to hand hygiene. These measures may also affect late-onset infection rates and offer understanding of novel interventions for prevention.

Methods. We examined infection rates during the 24 months prior to implementation of COVID-19 IP measures (PRE-period) compared to the months after implementation from April 2020 (POST-period). Late-onset infections were defined as culture-confirmed infection of the blood, urine, or identification of respiratory viral pathogens. An interrupted time series analysis of infection per 1000 patient days was performed based on a change-point Poisson regression with a lagged dependent variable and the number of patient days used as offsets. Each month was treated as independent with additional analysis using an observation-driven model to account for serial dependence.

Results. Multicenter analysis to date included all infants cared for at three centers (Level 3 and 4) from 2018-2020. Monthly BSI rates decreased in the POST-period at the three centers (**Figure 1).** At all centers actual BSI rate was lower than the expected rate in the POST-period (**Figure 2).** The combined BSI rate per 1000 patient days was 41% lower compared to the rate prior to implementation (95% CI, 0.42 to 0.84, P=0.004) (**Table 1**). In subgroup analysis by birthweight, infants< 1000g had a 39% reduction in BSI (P=0.023), for1000-1500g patients there was a 44% reduction (P=0.292) and in those > 1500g there was a 53% reduction (0.083).

Figure 1. PRE and POST MASKING and other COVID Infection Prevention Measures and Monthly BSI Rates.