## 636. Genome Epidemiology of Carbapenem-Resistant *Acinetobacter baumannii* (CRAb) in the United States

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**Background.** CRAb is a major cause of healthcare-associated infections and is associated with high mortality due to the lack of reliable treatment options. We aimed to elucidate the contemporary population structure of CRAb isolates circulating in US hospitals using whole-genome sequencing (WGS).

**Methods.** A total of 131 CRAb isolates were identified at four tertiary care medical centers located in Ohio, Pennsylvania, Texas and North Carolina between 2017 and 2018. The genomes were sequenced with Illumina NextSeq and *de novo* assembled. Sequence types (STs) were identified using the Pasteur Institute MLST scheme.  $\beta$ -Lactamase genes were identified by ResFinder and manually curated.

**Results.** The 131 isolates belonged to 10 different ST types, including 8 known and 2 novel ones. In this collection, 101 isolates (77.1%) belonged to ST2, the dominant drug-resistant clone in the United States and Europe; 20 isolates belonged to ST499, a less common, but also globally distributed clone. Two isolates each belonged to ST496 and ST79, both common in South America. For the chromosomally encoded  $bla_{0XA-95}$  being predominant. For the chromosomally encoded  $bla_{0XA-95}$  being the most common. The most frequent acquired carbapenemase genes were identified much less frequently and included  $bla_{0XA-29}$   $bla_{0XA-29}$ ,  $bla_{0XA-$ 

**Conclusion.** ST2 is the most prevalent ST type among contemporary CRAb isolates identified in US hospitals, however, new STs are emerging, most notably ST499. Significant diversity was seen among chromosomal  $bla_{OXA-SI}$  group carbapenemase, intrinsic  $bla_{ADC}$ -group cephalosporinase and plasmid-mediated  $bla_{OXA}$ -group carbapenemase genes, which likely represented diversification within the STs. Correlations between clinical presentation and outcomes and the genomic features of the infecting isolates are being investigated

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### 637. Whole-Genome Sequencing (WGS) of Isolates from Resolving (RB) and Persistent (PB) Methicillin-Resistant *Staphylococcus aureus* Bacteremia Batu K. Sharma-Kuinkel, PhD<sup>1</sup>; Felix Medie, PhD<sup>2</sup>; Felicia Ruffin, MSN<sup>1</sup>; Rebecca Wattam, PhD<sup>3</sup>; Maulik Shukla, PhD<sup>4</sup>; James Davis, PhD<sup>5</sup>; Michael Yeaman, PhD<sup>6</sup>; Vance G. Fowler, Jr., MD, MHS<sup>1</sup>; <sup>1</sup>Duke University Medical Center, Durham, North Carolina; <sup>2</sup>Duke University, Durham, North Carolina; <sup>3</sup>Virginia Tech, Blacksburg, Virginia; <sup>4</sup>Argonne Lab, Lemont, Illinois; <sup>5</sup>Argonne National Lab, Lemont, Illinois; <sup>6</sup>University of California at Los Angeles, Torrance, California

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**Background.** The basis for PB in MRSA is poorly understood. We tested the hypothesis that PB isolates will exhibit distinct genetic variation vs. resolving bacteremia (RB).

**Methods.** MRSA isolates from an existing cohort of prospectively enrolled patients with PB (n = 81) and RB (n = 81) were matched by sex, age, dialysis, diabetes mellitus, and presence of any implantable medical device. WGS was performed via MiSeq 2x150 runs (Illumina<sup>\*</sup>) with a targeted depth of 150-fold at Duke Genome Sequencing Core (DUGSIM). The genome sequence was analyzed, assembled, and annotated in PATRIC.

**Results.** No genetic signature was associated with PB or RB isolates at the genomewide significance level. Phylogenetic analysis separated the genomes into two distinct clades [Clade 1 (C1): 77 genomes; predominantly CC5; Clade 2 (C2): 74 genomes; predominantly CC8; Figure 1]. Most isolates in C1 genome carried SCC Mec type II (2A) vs. the SCC Mec type IV (2B) in C2. C1 and C2 had a total of 3602 (407 unique only to C1) and 3563 (368 unique only to C2) protein families, respectively, with 3195 families shared across the two groups. Out of the 407 unique C1 protein families, majority were not shared across all the genomes and only 28 were found in more than 40 of the genomes. A detailed look of 28 protein families showed that majority were found to be in 3 distinct genomic regions (R1—5.6 kb, 7 genes, also contains mecl; R2—18.2 kb; 16 genes; R3—6.3 kb, 6 genes, 2 predicted erythromycin resistance gene). Furthermore, 12 of C1 genomes lack these unique regions. Most of the genomes in C1 (Other than 12 genomes) carry genes responsible for erythromycin and clindamycin resistance. Out of the 368 protein families unique to C2, only 3 are widely shared (2 hypothetical proteins and 1 two-component transcriptional regulator. Patient characteristics were not associated with clade type.

*Conclusion.* The basis for the PB phenotype in patients with MRSA bacteremia is not due to sequence-based variation in the bacterial genome.

Figure 1: Phylogenetic tree of 162 MRSA genomes



Figure 1: Phylogenetic analysis of 162 MRSA genomes from PB and RB patients separated the genomes into two distinct clades [Clade 1 (C1): 77 genomes; predominantly Clonal Complex 8 (CC8); Clade 2 (C2): 74 genomes; predominantly Clonal Complex 8 (CC8)].

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# 638. Comparative Genomics of $Mycoplasma\ pneumoniae$ Isolated From Children with Pneumonia: South Korea, 2010–2016

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**Background.** This study applied high-throughput whole-genome sequencing (WGS) technologies to investigate the comparative genomics of 30 M. pneumoniae strains isolated from children with pneumonia in South Korea during two epidemics from 2010 to 2016 in comparison with a global collection of 48 *Mycoplasma pneumoniae* strains which includes seven countries ranging from 1944 to 2017.

Methods. A total number of 30 *M. pneumoniae* strains were selected for whole-genome sequence analysis from two epidemics, 2010–2012 and 2014–2016. Next-generation sequencing (NGS) of all *M. pneumoniae* strains was performed using the Illumina MiSeq desktop sequencer. Comparative genomic analysis was performed using BLAST Ring Image Generator (BRIG), MAUVE, MAFFT, CLC Phylogeny Module, SnpEff, and Pathosystems Resource Integration Center (PATRIC).

**Results.** The 30 Korean strains had approximately 40% GC content and ranged from 815,686 to 818,669 base pairs, coding for a total of 809 to 828 genes. Overall, BRIG revealed 99% to >99% similarity among strains. The genomic similarity dropped to approximately 95% in the P1 type 2 strains when aligned to the reference M129 genome, which corresponded to the region of the p1 gene. MAUVE detected four subtype-specific of which were all hypothetical proteins except for one tRNA insertion in all P1 type 1 strains. eBURST analysis demonstrated two clonal complexes which are accordant with the known P1 typing, with higher diversity among P1 type 2 strains. The phylogenetic tree constructed with 78 genomes including 48 genomes outside Korea, formed three clusters, in which the sequence type 3 strains from Korea were divided into two P1 type 1 clusters.

**Conclusion.** The comparative genomics of the 78 *M. pneumoniae* strains including 30 strains from Korea by WGS reveals structural diversity and phylogenetic associations, even though the similarity across the strains was very high.

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### 639. Performance Characteristics of an FDA Cleared Multiplex Panel for the Rapid Identification of Fungal Pathogens From Positive Blood Culture Sean Zhang, MD, PhD<sup>1</sup>; Linoj Samuel, PhD<sup>2</sup>; Frederick Nolte, PhD<sup>3</sup>; Esther Babady, PhD<sup>4</sup>; <sup>1</sup>The Johns Hopkins Hospital, Baltimore, Maryland; <sup>2</sup>Henry Ford Health System, Detroit, Michigan; <sup>3</sup>Medical University of South Carolina, Charleston, South Carolina; <sup>4</sup>Memorial Sloan Kettering Cancer Center, New York, New York.

### Session: 67. New Diagnostics

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**Background.** Routine identification of fungal pathogens from positive blood cultures by conventional culture-based methods can be time-consuming, delaying effective treatment of appropriate antifungal agents. The GenMark Dx ePlex Investigational Use Only Blood Culture Identification Fungal Pathogen Panel (BCID-FP) was assessed in a multicenter study to determine the performance of all 15 fungal targets on the BCID-FP sample-to-answer cartridge, which incorporates extraction, amplification, digestion, hybridization and detection using GenMark eSensor technology, in approximately 100 minutes.