**1248. Genomic Sequencing and Clinical Data Integration for Next-Generation Infection Prevention**

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***Background.*** Typical Infection Prevention to detect pathogen transmission in hospitals has relied on observation of (1) uncommon pathogen phenotypes or (2) greater than expected number of pathogen phenotypes in a given timeframe and/or location. Genome sequencing of targeted organisms in conjunction with routine patient geo-temporal information and antibiotic susceptibility data holds promise in identifying transmissions with greater sensitivity and specificity, saving time and effort in reviewing for transmission events.

***Methods.*** In an on-going genomic sequencing surveillance effort in a tertiary care hospital, drug-resistant clinical isolates from the "ESKAPE" pathogens were routinely sequenced in 2017. In parallel, potential clusters were identified for 2017 through conventional Infection Prevention approaches. Groups identified by their genetic distances along with visualizations on antimicrobial susceptibilities, and patient location histories and dates were displayed in an interactive interface, Philips IntelliSpace Epidemiology (PIE), and reviewed by Infection Prevention.

***Results.*** Among 656 patients, 1,239 drug-resistant ESKAPE samples were sequenced. Thirty-eight genetically related groups involving 196 patients were identified. Groups ranged in size from two to 44 patients, primarily consisting of VRE and MRSA. Notably, a review of the 38 groups identified 20 groups where the information at hand suggested a concern for transmission. 16 of the 20 were not previously identified by Infection Prevention. Using PIE to review all 38 groups identified from 1 year's worth of data required 3 hours of time by an Infection Prevention professional, averaging less than 5 minutes per cluster, less than 1 minute per patient, and 11 minutes of review time per actionable opportunity. By conventional means, approximately 23 hours would have been required to review the genomic groups without the aid of the PIE tool.

***Conclusion.*** The use of PIE's genomic-defined groups, along with the integrated clinical data platform, allows for a greater ability, certainty, and speed to detect clusters of organisms representing transmission in the hospital setting. Applied prospectively, PIE can detect transmissions sooner than by conventional means for potential patient safety gains and cost savings.

***Disclosures.*** **D. Chen**, Philips: Scientific Advisor, Consulting fee. **M. Fortunato-Habib**, Philips Healthcare: Collaborator and Employee, Salary. **A. Hoss**, Philips: Employee, Salary. **R. Kolde**, Philips: Employee, Salary. **A. Dhand**, Merck: Speaker's Bureau, Speaker honorarium. Astellas: Scientific Advisor, Consulting fee. **R. Sussner**, Philips: Scientific Advisor, Consulting fee. **J. Carmona**, Philips Healthcare: Employee, Salary. **B. Gross**, Philips Healthcare: Employee, Investigator, Research Contractor, Scientific Advisor and Shareholder, Salary. **J. Fallon**, Philips Healthcare: Investigator, Research support.

**1249. Emergence of Diverse Carbapenem-Resistant Enterobacteriaceae (CRE) in the Dominican Republic**

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***Background.*** Despite the global threat of CRE, data from resource-limited regions such as the Dominican Republic (DR) are limited. A lack of novel antibiotics and molecular diagnostic tools for outbreak detection, coupled with the role of travel in circulating CRE to and from the DR represent significant challenges to limiting their spread. Here, we report the first molecular characterization of DR CRE isolates and compared them to geographically diverse CRE.

***Methods.*** Isolates from DR (one *Citrobacter freundii*, three *Klebsiella pneumoniae),* obtained from patients with bacteremia (one) and pneumonia (three), were compared with CRE from a New York City hospital in a Dominican neighborhood, including isolates (two *Enterobacter cloacae*, one *K. pneumoniae)* from a patient transferred to NYC from another DR institution. Whole genome sequencing was used to determine multi-locus sequence type (MLST) and resistance gene profiles. Phylogenetic analyses of isolates with same ST were performed.

***Results.*** Isolates from the DR and the Dominican patient were of unique genomic backgrounds including pandemic (*K. pneumoniae* ST11) and novel sequence types, and harbored either $bla_{KPC-2}$ or $bla_{KPC-3}$ (Table 1). Replicon typing suggested that these carbapenemase genes were located on distinct plasmids. Phylogenetic analyses using the NYC collection of ~400 sequenced CRE isolates indicated that DR and NYC *K. pneumoniae* ST307 isolates were related (33 SNPs). Further review showed that both patients had recent admissions in Puerto Rico (PR), highlighting the role of regional spread. *K. pneumoniae* ST11 isolates from DR and NYC, on the other hand, were not found to be closely related (1,418–1,440 SNPs).

***Conclusion.*** Genotyping of DR CRE isolates revealed a high genomic diversity, suggesting multiple introductions. Phylogenetics of *K pneumoniae* ST307 place these within a global context, demonstrating links across the Caribbean and North America. International surveillance studies integrating genomics are needed to track and limit the spread of CRE in resource-limited settings such as DR.

**Table 1:** Comparison of DR Isolates

| Organism | MLST | KPC Gene | Origin |
|---|---|---|---|
| *K. pneumoniae* | ST11 | $bla_{KPC-2}$ | DR |
| | ST1040 | $bla_{KPC-3}$ | NYC, DR patient |
| | ST307 | $bla_{KPC-2}$ | DR, travel to PR |
| | Novel ST | $bla_{KPC-3}$ | DR |
| *C. freundii* | ST95 | $bla_{KPC-2}$ | DR |
| *E. cloacae* | ST456 | $bla_{KPC-3}$ | NYC, DR patient |

***Disclosures.*** **A. C. Uhlemann**, Merck: Investigator, Grant recipient.

**1250. Prevalence and Risk Factors for Acquiring Carbapenem-Resistant *Enterobacteriaceae* in an Intensive Care Unit at a Tertiary Hospital**

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***Background.*** Active surveillance testing of carbapenem-resistant *Enterobacteriaceae* (AST-CRE) is recommended in high-risk settings, such as intensive care units (ICUs), to prevent CRE outbreaks or invasive infections. This study aimed to investigate the effects of AST-CRE by analyzing the prevalence and risk factors for acquiring CRE during the ICU care.

***Methods.*** We conducted AST-CRE on rectal swabs of patients admitted to the ICU in the emergency room at a tertiary hospital in South Korea for 12.5 months. AST-CRE was performed upon admission and weekly thereafter. To assess the risk factors of acquiring AST-CRE during the admission period in adult patients, those colonized with CRE upon admission and aged <18 years were excluded. AST-CRE was performed using Centers for Disease Control and Prevention methods. A polymerase chain reaction assay was performed to detect five carbapenemase genes (NDM, KPC, VIM, IMP, and OXA).

***Results.*** A total of 810 patients were admitted during the study period. The acquisition rate and carbapenemase-producing CRE were 2.6% (21/810) and 42.9% (9/21), respectively. No invasive infection due to CRE was found. The most common species were *Klebsiella pneumoniae* (71.4%, 15/21), and eight KPC and one NDM genes were detected. In CRE-positive patients, in-hospital mortality and length of hospitalization were higher ($P = 0.003$) and longer ($P < 0.001$), respectively. Multivariate analyses showed that male gender (adjusted odds ratio [aOR] 8.0; 95% confidence interval [CI] 1.7–36.8), previous hospitalization in the last year (aOR 5.1; 95% CI 1.6–16.4), co-colonization with multidrug-resistant *Acinetobacter* species (aOR 18.3; 95% CI, 4.2–79.2) and extended-spectrum β-lactamase-producing bacteria (aOR 3.4; 95% CI, 1.1–10.9), and length of ICU admission until CRE detection for ≥10 days (aOR 6.5; 95% CI 2.2–19.2) were independently associated with CRE acquisition.

***Conclusion.*** To prevent CRE outbreak or invasive infections, patients admitted in the ICU should be screened using AST-CRE.

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**1251. Contaminated Sinks May be an Environmental Source for Serial Transmission of Carbapenem-Resistant *Enterobacteriaceae* (CRE) to ICU Patients**

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***Background.*** We performed an investigation after noting an increase in hospital-onset (HO) KPC-producing *Enterobacteriaceae* (KPC-E) infections in patients admitted to a tertiary referral hospital in North Carolina.

***Methods.*** We defined pre-outbreak (January 1, 2017–June 30, 2017), outbreak (July 1, 2017–October 31, 2017), and post-outbreak (November 1, 2017–March 31, 2018) phases. A clinical case was defined as any positive clinical culture for KPC-E. HO was defined as a positive clinical or surveillance culture collected on hospital day ≥3. Patients were mapped in space and time to inform targeted environmental sampling. Whole-genome sequencing (WGS) was performed on selected KPC *K. pneumoniae* environmental and patient isolates to determine relatedness.

In October 2017, a CRE prevention bundle was implemented that included daily communication of CRE patient movement, increased audits/feedback of HCW compliance with hand hygiene, enhanced cleaning and disinfection in CRE rooms and high-risk units with bleach and UVC disinfection, and weekly rectal surveillance screens in four adult ICUs.

***Results.*** 0.67 clinical cases of KPC-E per month were observed during the pre-outbreak period compared with 3.75 clinical cases of KPC-E per month during the outbreak period. *K. pneumoniae* was the most common species (Figure 1). Mapping of patients revealed probable direct and indirect transmission between patients in multiple hospital units (Figure 2). three patients who were non-sequentially admitted to the same ICU room over a 12-week span acquired KPC *K. pneumoniae* (Figure 2). Environmental cultures from the in-room sink drain and P-trap grew KPC *K. pneumoniae* that was related to the patient isolates by WGS; the sink was removed. Although no additional clinical cases of KPC-E occurred after full implementation of the bundle and sink removal, we continued to observe acquisition of KPC-E rectal colonization in all four ICUs (Figure 3).

***Conclusion.*** We describe a multispecies outbreak of KPC-E that was mitigated through evidence-based CRE control measures and removal of a colonized sink. However, ongoing low-level presumed transmission of KPC points to persistent environmental sources. Additional study is needed to understand the prevalence of CRE in hospital sinks, factors that drive drain colonization, and contribution of CRE in a sink to nosocomial transmission.

Figure 1. Unique Cases of Hospital-acquired KPC by Month

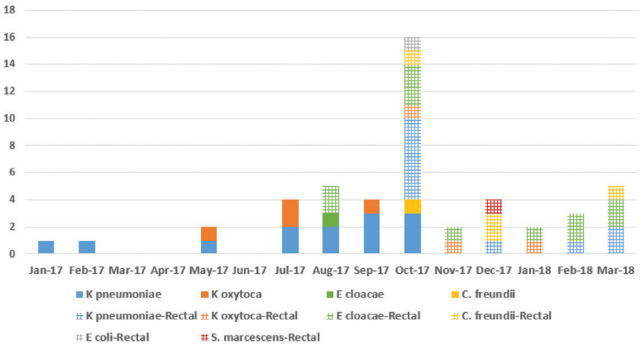
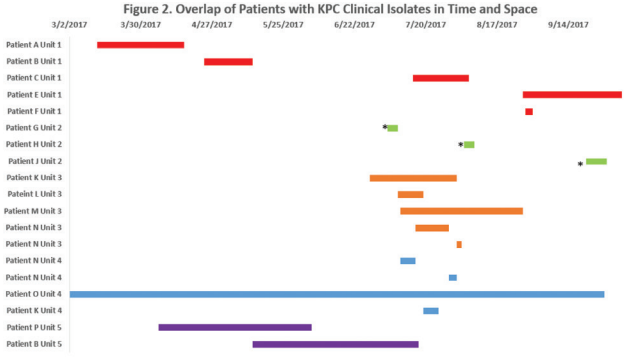



Figure 2. Overlap of Patients with KPC Clinical Isolates in Time and Space




* 3 patients admitted to the same ICU room with hospital-onset KPC *K. pneumoniae* clinical cultures

Figure 3. Sub-Clinical Transmission of KPC in Four Adult ICUs Following Implementation of a Prevention Bundle in October 2017


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

## 1252. A Challenging *Burkholderia* Outbreak Investigation Across Multiple Units at an Academic Medical Center From June 2017 to February 2018

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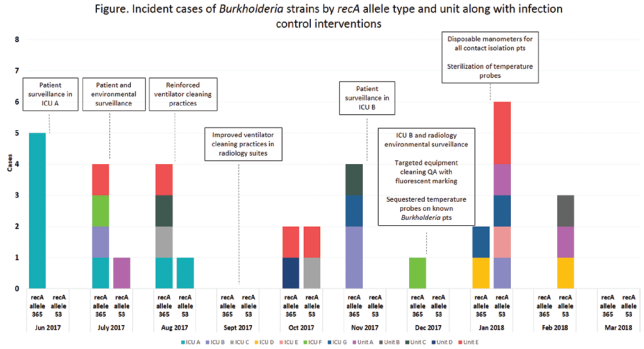
***Background.*** Most outbreak investigations involve short-term, geographically localized clusters. However, some organisms can form environmental reservoirs leading to more prolonged, widespread outbreaks. We describe a prolonged outbreak of *Burkholderia* at our institution.

***Methods.*** An epidemiological investigation was conducted. *Burkholderia* isolates were genotyped using pulsed-field gel electrophoresis (PFGE) and *recA* gene sequencing. Initial isolates were sent to a national reference laboratory for multilocus sequence typing (MLST).

***Results.*** 32 patients on 12 units (see figure) had ≥1 positive culture for *Burkholderia* from June 2017 to February 2018. 21 had *B. cenocepacia* (PFGE pattern A, *recA* allele 365) and 11 had *B. cepacia* (PFGE pattern C, *recA* allele 53). MLST revealed that isolates with *recA* allele 365 were unique compared with previously identified *B. cenocepacia* strains. Of 32 patients, 28 (88%) had positive respiratory cultures. Of 32 patients, 3 (9%) had bacteremia. Thirty-day mortality was 4/29 (14%). A case–control study did not reveal a common point source. All surveillance cultures from asymptomatic patients were negative ($n$ = 53). Two of nine sink drains in rooms of cases were positive for an unrelated strain of *B. cepacia*. Other environmental cultures were negative for *Burkholderia* ($n$ = 49). Cases continued despite routine interventions (see figure), with some incident cases detected long after potential exposures. Ventilator/respiratory equipment (V/RE) cleaning was investigated. Multiple V/RE interventions were implemented: (1) ensuring a sterilization process for ventilator temperature probes (used in heated humidification) was occurring; (2) using disposable manometers on contact isolation patients; (3) reinforcing ventilator cleaning, including those in radiology suites after use.

***Conclusion.*** No definitive source of the outbreak was found. New cases continued after reinforcement of basic infection control practices, but subsided after focused attention on V/RE cleaning practices. Control of this outbreak was challenging due to the complexity of a prolonged "latency period" for *Burkholderia*, difficulty identifying reservoirs, and multiple possible modes of transmission, especially for organisms like *Burkholderia* that can persist on environmental surfaces and equipment.

Figure. Incident cases of *Burkholderia* strains by *recA* allele type and unit along with infection control interventions




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