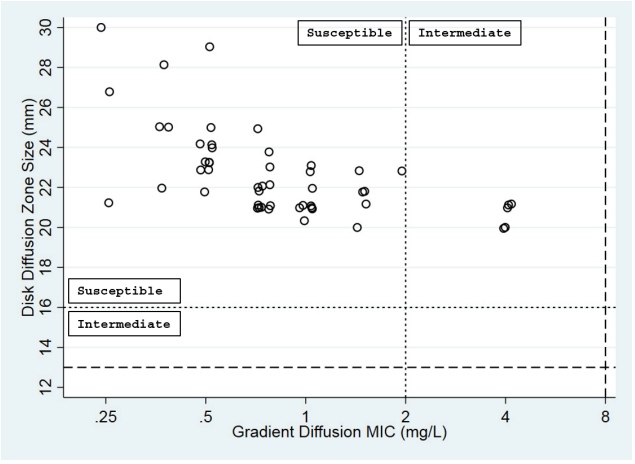
mL) methods according to manufacturer instructions and using FDA clinical breakpoints for interpretation.

Results. 51 isolates were tested: 21 *E. coli*, 9 *P. mirabilis*, 7 *E. cloacae*, 6 *K. pneumoniae*, 3 *K. oxytoca*, 3 *S. marcescens*, 1 *K. aerogenes*, and 1 *C. freundii*. Specimen sites included: 29 blood, 8 urine, 8 soft tissue or bone, 5 intra-abdominal, and 1 sputum. Previous phenotypic AST results demonstrated 19 (37%) were CRE, of which 5 were also gentamicin and tobramycin resistant, and 32 (63%) were tobramycin and gentamicin resistant but carbapenem susceptible. Plazomicin zone diameters and minimal inhibitory concentrations (MIC) for all isolates are shown in the figure (data jittered to show frequency). There was a significant correlation between increased MIC and smaller zone diameters (Pearson coefficient −0.443, $P = 0.001$). However, while all 51 isolates were susceptible by DD breakpoints, only 46 (92%) were susceptible by GD breakpoints. All 5 discordant results were *P. mirabilis* which had an MIC of 4 µg/mL (intermediate) but zone diameters of 20–21 mm (susceptible).

Conclusion. Concordance between plazomicin DD and GD susceptibility was only 92%. All 5 discordant results were *P. mirabilis*. Surveillance studies demonstrate >80% of *P. mirabilis* have MIC of 2–4 mg/L. Given the DD breakpoint is 16 mm, our data suggest DD was overly active in our sample set. Comparison of DD and GD to reference broth microdilution against a larger set of isolates is warranted to determine which method is optimal; however, our data suggest DD may result in categorical errors for *P. mirabilis*.



Disclosures. Alexander Lepak, MD, Paratek Pharmaceuticals: Research Grant; Tetraphase Pharmaceuticals: Research Grant.

2163. Clinical Impact of Inter-site Blood Culture Transport in a Canadian Tertiary Care Center

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Session: 243. Bacterial Diagnostics
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Background. The potential delays caused by transport of blood cultures to server laboratories might result in delayed issuance of results for patients with positive blood cultures. In this study, we aimed to determine the clinical impacts of inter-site transport of blood cultures.

Methods. We performed a retrospective cohort study involving cases with positive blood cultures (1 positive blood culture/species/patient/7 days; not deemed as a contaminant) at two sites of a Canadian tertiary care center between January 1, 2018 and December 31, 2018. Blood cultures from the affiliated site were transported to the laboratory of the primary server site. These two sites are located 8 km apart. The following outcomes were studied: the duration between blood culture sampling and issuance of the first report and the duration between blood culture sampling and administration of the first effective antibiotic.

Results. We observed 349 episodes of bacteremia, including 161 in the affiliated site (45.5%) and 193 in the primary server center (54.5%). *Enterobacteriaceae* (n = 151, 43%) and *Staphylococcus aureus* (n = 77, 22%) were the most commonly observed causative bacteria. Median duration for issuance of the first positive report was significantly shorter in the primary server hospital (32.4 h, interquartile range [IQR] 19.8–44.3) than in the affiliated center (37.9 h, IQR 24.1–46.5; $P = 0.004$). The median duration between blood culture sampling and administration of the first effective antibiotic was 2.7 h in the server site (IQR 0.75–15.2) and 2.3 h in the affiliated site (IQR 1–8.45) ($P = 1.0$). Receiving the first effective antibiotic after blood culture sampling required > 60 min in 8/189 patients (4.2%) in the affiliated site and 9/158 patients (5.7%) in the primary server site ($P = 0.3$). The 30-day mortality was 13.8% (26/189) and 8.9% (14/158) at the primary server site and affiliated site, respectively ($P = 0.16$).

Conclusion. Inter-site transport of blood cultures is associated with a significant delay in the issuance of positive blood culture reports. However, this delay does not cause any delay in administration of effective antibiotic therapy because of rapid recognition of sepsis in bacteremia patients. These results are reassuring in the context of increasing microbiology service centralization.

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

2164. Activity of Fosfomycin (FOF) and Frequency of Nonsusceptible Inner Colonies During Susceptibility Testing of an International Collection of Clinical *Pseudomonas aeruginosa* (PA) Isolates

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Background. FOF has been used clinically for the treatment of PA infections in the absence of established interpretive criteria. A recent study identified a low frequency of nonsusceptible inner colony mutants during disk diffusion (DD) testing of *Escherichia coli*; however, the frequency of this phenomenon in PA isolates is not well characterized. We sought to determine FOF activity against an international collection of PA isolates and the frequency of inner colony mutants observed during Etest and DD testing.

Methods. Minimal inhibitory concentration (MIC) values were determined for a convenience collection of 109 PA ([70/94] 64.2% MDR) isolates from 4 institutions in the United States and Australia. MIC testing was conducted in duplicate on separate days utilizing agar dilution (AD), broth microdilution (BMD), DD, and Etest as recommended per Clinical and Laboratory Standards Institute (CLSI). CLSI *E.coli* interpretive criteria (≤ 64 mg/L susceptible) were used for MIC interpretations. The proportion of isolates containing inner colonies was determined using DD and Etest. Inner colony mutants were subcultured and retested using BMD with comparison to the parent isolate MICs.

Results. FOF MICs varied widely and ranged from 1024 mg/L with MIC_{50}/MIC_{90} values of 64/256 (AD), 64/512 (Etest), and 64/256 (BMD) mg/L. Using *E. coli* criteria, susceptible/resistant rates were: 60.5/17.4% for AD; 60.5/22.0% for Etest; 86.2/7.3% for DD; and 53.2/17.4% for BMD. Inner colonies were frequently observed in 38.5% and 35.8% of DD and Etest inhibition zones, respectively. After repeat testing, mutant MIC values ranged from 64 to > 1024 mg/L and a majority (85.9%) had MIC values ≥ 512 mg/L.

Conclusion. Observed MIC values of this (64% MDR) collection varied widely with $MIC_{50/90}$ values commonly at or above the *E. coli* susceptibility breakpoint. Inner colony mutants were frequently observed and highly resistant. Whole-genome sequencing is currently underway for a subset of parent/mutant pairs to determine whether specific genetic alterations are attributed to the increased MICs. Based on these results, caution should be warranted in extrapolating *E. coli* breakpoints to other organisms, and treatment of PA with FOF should be further evaluated.

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2165. *Helicobacter pylori* Infections in the Bronx, New York: Whole-Genome Sequencing for Rapid Genotypic Susceptibility Testing

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Background. Susceptibility-guided treatment of *H. pylori* is superior to empiric therapy. We determined the accuracy of whole-genome sequencing (WGS) compared with phenotypic testing using CLSI/EUCAST breakpoints.

Methods. Thirty-three clinical isolates of *H. pylori* cultured from gastric biopsies were sequenced with a coverage range between 40x and 80x using Illumina Miseq platform and the reads were assembled and annotated with PATRIC. Phenotypic susceptibility tests were performed using E-test strips under microaerophilic conditions for 72 hours. Mutations associated with amoxicillin, tetracycline, clarithromycin, levofloxacin, metronidazole and rifampin resistance were examined.

Results. Of the 33 isolates, two were phenotypically resistant to amoxicillin: one carried a β-lactamase gene ($bla_{TEM-116}$) and the other exhibited a point mutation *pbp2* (A541T). All isolates were tetracycline susceptible phenotypically, but three isolates had point mutations in *16S rRNA* that are associated with resistance (A926G). Clarithromycin results showed a good correlation between methods. Nine clarithromycin-resistant isolates demonstrated point mutations in 23S *rRNA* (A2142G/A2143G). Fifteen isolates were phenotypically resistant to levofloxacin, but resistance mutations were found in only 14 strains (N87I/N87K/D91Y/D91N/D91G/D99N in *gyrA*). We analyzed our strains for the presence of intact genes *rdxA* and *frxA,* either of which convert the prodrug form of metronidazole into the active form. Twenty-four