cobas®LIAT® PCR system. Performing saliva swabs will result in less discomfort and distress to children who are tested for GAS. Further study is needed to determine the sensitivity and specificity of saliva swabs for the detection of GAS in children presenting with acute pharyngitis.

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

### 2180. Novel Method for Determining Rapid E.coli Antibiotic Susceptibility (AST) Results for Urinary Tract Infections

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**Session:** 243. Bacterial Diagnostics

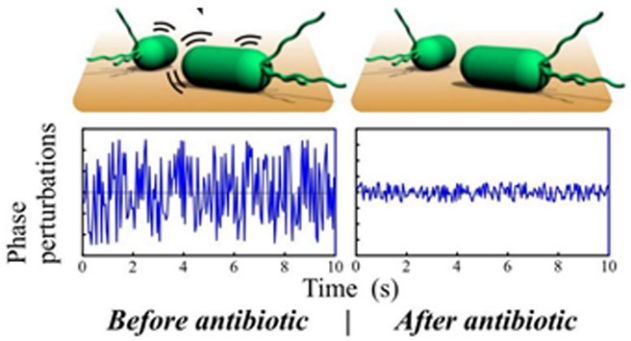
*Saturday, October 5, 2019: 12:15 PM*

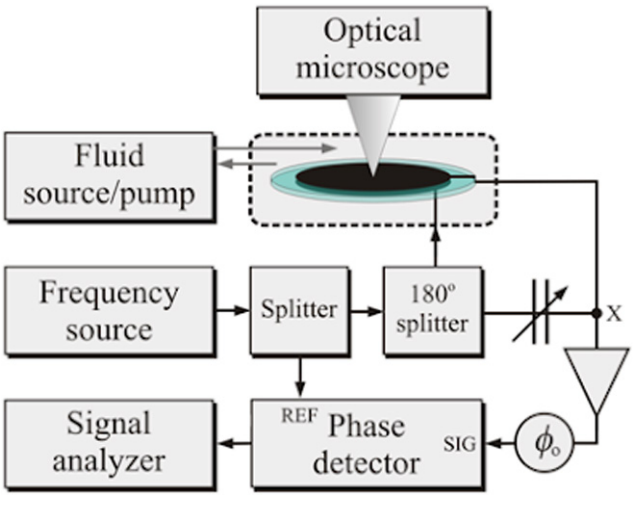
***Background.*** Measuring changes in phase noise from bacteria on a quartz crystal resonator has been shown to effectively distinguish viable from non-viable *E. coli*. We report using this method to rapidly perform AST for *E. coli* isolated from a leftover clinical urinary tract infection (UTI) specimen.

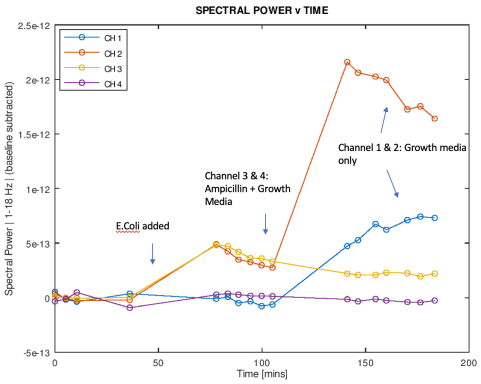
***Methods.*** An experimental system was designed to sense changes in bacterial mechanics through changes in phase noise generated by bacterial cells (Figure 1). The system includes a quartz-crystal resonator with thin-film gold electrodes on opposite surfaces housed within a module. The module provides electrical contact to the crystal's electrodes, and incorporates channels through which fluids can be pumped (Figure 2). *E. coli* was isolated from a leftover positive urine culture specimen, cultured overnight and resuspended in phosphate-buffered saline (PBS). The suspension was run through the experimental system. E.coli cells were adhered to the surface of the quartz resonant crystal coated with a cationic polymer. After a growth phase, the cells were exposed to antibiotic (ampicillin). Phase noise was monitored throughout the test. The power spectral density of the noise was averaged each 5 minutes. E.coli was classified as ampicillin susceptible if the spectral power of the added phase noise was at least 50% lower compared with controls. Controls were in growth media only (Figure 3). Automated microscopy was utilized to monitor cell growth.

***Results.*** The method correctly classified the E.coli as ampicillin susceptible. Power spectral density increased in untreated cells and dropped or stayed steady in cells treated with Ampicillin. Corresponding loss of E. coli viability was confirmed microscopically. Results were compared with standard of care antibiotic susceptibility testing.

***Conclusion.*** The phase noise measurement method correctly identified ampicillin susceptible E.coli isolated from a leftover patient urine sample in three and one half hours. It shows promise for providing rapid AST results to treat UTIs.




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

### 2181. Yield and Impact of Molecular Diagnostics for Pathogen Detection in Pediatric Patients: 16/18S rRNA PCR and Noninvasive Assays

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**Session:** 243. Bacterial Diagnostics

*Saturday, October 5, 2019: 12:15 PM*

***Background.*** Molecular diagnostic tests can identify bacterial and fungal pathogens from clinical samples. Nucleic acid detection tests include 16S and 18S rRNA gene PCR (16/18S PCR) and plasma next-generation sequencing (NGS). Other assays (fungal galactomannan and 1,3-β-D-glucan) detect structural factors. Our objective was to assess the utilization, yield, and impact of molecular diagnostics in pediatric patients who had samples sent for 16/18S PCR.

***Methods.*** Sterile site fluid or tissue specimens were collected as part of standard care at Lurie Children's Hospital, cultured, and sent to Northwestern Memorial Hospital for 16/18S PCR as clinically indicated. Medical records were reviewed for diagnostics, antibiotics, and clinical course.

***Results.*** From 1/2016–8/2018, 236 samples were sent for 16 and/or 18S PCR from 183 patients. 83% had a concurrent ID consult. 16S PCR was done on 215 samples, 42 (20%) were positive, and 36 yielded species identification (Table 1). Antibacterial agents were administered prior to specimen collection in 73% and did not affect likelihood of positive 16S PCR. 18S PCR was sent on 163 samples; 12 (7.4%) were positive (Table 2) of which 10 were from immunocompromised hosts. 40% of patients were on antifungals prior to sample acquisition. 16/18S PCR impacted antimicrobial decision-making in 70 cases (30%). A pathogenic fungus was detected by PCR but *not* culture in 2 cases. Time to positivity of fungal culture was 1–15 days. Fungal culture was positive in 5 cases with-negative 18S PCR. Seventeen patients had positive serum 1,3-ß-D-glucan and/or galactomannan: 3 of which had positive 18S PCR, 5 with fungal growth, 5 presumed infection based on imaging, 1 *Nocardia*, and 3 noninfectious etiology. Plasma NGS was sent on 45 cases, was positive in 34, and affected clinical management in 10.

***Conclusion.*** 16S PCR can identify bacterial pathogens in the setting of negative culture and impact clinical care. Abscess, bronchial/pleural fluid, and brain/organ tissue were high yield specimens. 18S PCR can provide expeditious fungal identification in cases of suspected invasive disease, but fungal culture and serum molecular testing increase diagnostic yield. No single fungal test is comprehensive. Plasma NGS had relatively high yield and clinical impact in selected patients.

**Table 1: Yield of 16/18S PCR from Sterile Site Samples**

| Sample source | Total (n=236) | 16S PCR positive: # +/# sent (%) | 16S PCR organisms identified | 18S PCR positive: # +/# sent (%) | 18S PCR organisms identified |
|---|---|---|---|---|---|
| Osteoarticular (fluid or tissue sample) | 60 | 12/58 (21%) | *Acinetobacter spp., Bartonella henselae, CONS, Enterobacter cloacae, E coli, Fusobacterium nucleatum, Kingella kingae, S agalactiae, S pneumoniae* | 0/32 | |
| Cerebrospinal fluid | 38 | 5/36 (14%) | *S pneumoniae, Ureaplasma parvum* | 1/21 (5%) | *Rhodotorula spp.* |
| Pleural fluid | 34 | 9/32 (28%) | *Fusobacterium spp., Porphyromonas spp., Prevotella spp., S aureus, S pneumoniae* | 1/20 (5%) | *Rhizomucor spp.* |
| Lung tissue | 23 | 0/21 | | 2/22 (9%) | *Aspergillus fumigatus + Cunninghamella, Blastomyces dermatitidis* |
| Other organ/tissue† | 14 | 4/14 (29%) | *Pseudomonas aeruginosa, Prevotella spp, Serratia marcescens* | 1/12 (8%) | *Blastomyces dermatitidis* |
| Abscess | 13 | 6/12 (50%) | *B fragilis, Fusobacterium nucleatum, Pseudomonas aeruginosa, S aureus, S anginosis, S mitis* | 2/7 (29%) | *Candida albicans, Lichtheimia ramosa* |
| Lymph node biopsy | 9 | 1/8 (13%) | *S aureus* | 0/8 | |
| Respiratory/bronchial | 9 | 3/7 (43%) | *Gronulicatella spp., Haemophilus influenzae, Porphyromonas spp.* | 0/9 | |
| Bone marrow | 8 | 0/8 | | 0/8 | |
| Peritoneal/biliary fluid | 8 | 0/6 | | 0/5 | |
| Skin biopsy | 8 | 0/4 | | 4/8 (50%) | *Aureobasidium pullulans, Candida lucitaniae, Rhizopus spp.* |
| Brain biopsy | 7 | 3/6 (50%) | *S intermedius, S pneumoniae* | 0/6 | |
| Sinus aspirate | 3 | 0/1 | | 1/3 (33%) | *Aspergillus spp.* |
| Pericardial fluid | 2 | 0/2 | | 0/2 | |

† Organ tissue biopsies: arytenoid/larynx (1), chest mass (3), kidney (1), liver (3), mediastinum (1), parotid (1), spleen (4)

**Table 2: 18S PCR or Fungal Culture Positive Samples**

| Sample Source | 18S PCR result | Culture result | 1,3-ß-D-glucan | Galactomannan | Plasma NGS |
|---|---|---|---|---|---|
| Abdominal abscess | *Candida albicans* | *Negative* | Positive (>500) | Negative | Not done |
| CSF | *Rhodotorula spp.* | Negative | Positive (249) | Positive (0.6) | *Candida albicans* |
| Fibula bone/tissue | Negative | *Blastomyces dermatitidis* | Negative | Negative | Not done |
| Lung | *Aspergillus fumigatus, Cunninghamella* | *Aspergillus fumigatus,* few *Bacillus cereus* | Indeterminate (67) | Negative | Not done |
| Lung | Negative | *Aspergillus fumigatus* | Positive (>500) | Positive | Not done |
| Lung | Negative | *Aspergillus fumigatus* | Positive (>500) | Negative | *Pneumocystis jirovecii,* Torque tenovirus 10 |
| Lung | *Blastomyces dermatitidis* | *Blastomyces dermatitidis* | Not done | Not done | Not done |
| Lung | Negative | *Phellinus spp.* | Positive (80) | Not done | Not done |
| Pleural fluid | *Rhizomucor spp.* | *Rhizomucor spp.* | Not done | Not done | *Staphylococcus epidermidis, Granulicatella adiacens* |
| Sinus aspirate | *Aspergillus* | *Aspergillus fumigatus* | Negative | Negative | Not done |
| Sinus aspirate | Negative | *Exserohilum rostratum* | Positive (142) | Negative | *Eikenella corrodens, Abiotrophia defectiva, Aspergillus flavus, Aspergillus oryzae,* CMV, *Enterococcus faecalis, Haemophilus parainfluenzae, Staphylococcus aureus* |
| Skin biopsy | *Aureobasidium pullulans* | Negative | Not done | Not done | Not done |
| Skin biopsy | *Lichtheimia ramosa* | Negative | Negative | Negative | *Absidia idahoensis*, Pseudomonas aeruginosa, Argobacterium tumefaciens* |
| Skin biopsy | *Rhizopus oryzae* | *Rhizopus oryzae* | Negative | Negative | Not done |
| Skin biopsy | *Rhizopus spp.* | *Rhizopus spp.* | Not done | Not done | Not done |
| Tissue, chest wall | *Blastomyces dermatitidis* | *Blastomyces dermatitidis* | Not done | Not done | Not done |
| Tissue, leg | *Candida lusitaniae* | *Candida lusitaniae, Candida parapsilosis* | Positive (448) | Negative | *Aspergillus fumigatus* †, *Escherichia coli,* HSV-1, *Rhizopus oryzae* † |

*likely same organism as *Lichtheimia*

† Additional sites (lung, sacral wound) with pathology and cultures consistent with multiple foci of opportunistic infection

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

## 2182. Harnessing Direct-from-Blood MALDI-TOF (DBM) and Local Blood Stream Infection (BSI) Antibiogram Data to Direct Optimal Therapy for Gram-Negative Rod (GNR) Bacteremia

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**Session:** 243. Bacterial Diagnostics

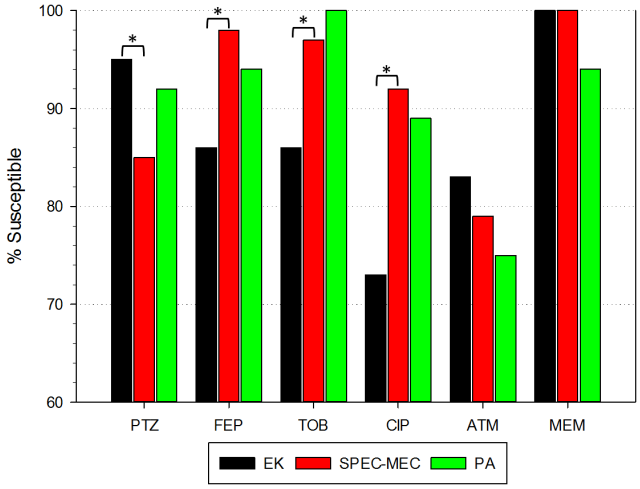
*Saturday, October 5, 2019: 12:15 PM*

***Background.*** GNR bacteremia is common in the hospital environment. Drug-resistance challenges thoughtful use of, and timely de-escalation to, regimens that limit carbapenem use. DBM accurately provides rapid identification of bacteremia pathogens, but lacks antibiotic susceptibility testing (AST) results to aid clinicians in refining therapy. We investigated whether integrating DBM identification and local BSI antibiogram data could be used to identify carbapenem-sparing regimens with predicted efficacy of ≥ 95% prior to traditional AST results.

***Methods.*** We identified GNR blood culture results in adults from January 1, 2018 to December 31, 2018. The first isolate per bacteremia episode per patient was analyzed. AST was performed by MicroScan. We categorized isolates into 1 of 3 groups based on the most common resistance mechanisms in our population (i.e. CTX-M ESBL, AmpC): 1] *Escherichia* spp. and *Klebsiella* spp. (EK), 2] *S. marscecens, P. mirabilis, E. (Klebsiella) aerogenes, C. freundii, M. morganii, E. cloacae* complex, and *C.* non-*freundii* (SPEC-MEC), and 3] *P. aeruginosa* (PA). We compiled susceptibility rates to common antibiotics for GNR sepsis and compared results using Chi-square.

***Results.*** 381 GNR isolates were recovered in blood cultures from adult patients in 2018: EK 264 (69%), SPEC-MEC 66 (17%), PA 36 (9%), other 15 (4%). Susceptibility results in common GNR sepsis agents are shown in Figure 1 (*$P$ < 0.05 between groups). Within each organism group, susceptibility was highest in the following scenarios: 1] EK: PTZ and MEM ($P$ < 0.05 vs. FEP, TOB, CIP, and ATM), 2] SPEC-MEC: FEP, TOB, and MEM ($P$ < 0.05 vs. PTZ and ATM), and 3] PA: comparably similar (> 90%) for PTZ, FEP, TOB, and MEM.

***Conclusion.*** Integration of DBM and antibiogram data identified species-specific carbapenem-sparing regimens that would predict ≥ 95% of patients with GNR bacteremia are on effective therapy prior to traditional AST results. In appropriate patients, this could lead to a significant reduction in carbapenem duration and avoid broad-scale carbapenem use while also optimizing time to effective therapy. This is a readily adaptable process for other centers to improve care of patients with GNR bacteremia. Our data suggest using β-lactam alternatives for GNR bacteremia, especially EK, should be done with caution.




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

## 2183. Survival Impact and Clinical Predictors of Anaerobic Bloodstream Infection

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**Session:** 243. Bacterial Diagnostics

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***Background.*** Few controlled studies are available for the outcome and risk factor analysis of anaerobic bloodstream infection (BSI). We conducted a cohort study to identify the clinical predictors and survival impact of anaerobic BSI as compared with aerobic BSI.

***Methods.*** Consecutive emergency department patients in a tertiary medical center with laboratory confirmed BSI between 2015 and 2016 were prospectively enrolled. We compared demographics, comorbidity, and sources of infection between anaerobic and aerobic BSI. We then constructed a multivariable logistic regression model to identify independent risk factors for anaerobic BSI. The survival impact of anaerobic BSI was evaluated by propensity score-matched analysis.

***Results.*** We identified 1,166 episodes of BSIs during the 2-year study period, of which 61 (5.2%) were anaerobic BSI. Clinical variables predicted anaerobic BSI with moderate discrimination (optimism corrected C statistic = 0.75). Significant predictors included metastatic cancer (OR 6.03, 95% CI 2.78–13.09), intra-abdomen infection (OR 3.92, 95% CI 1.47–10.45), liver abscess (OR 2.65, 95% CI 1.26–5.62), skin and soft-tissue infection (OR 2.40, 95% CI 1.13–5.08) as the positive predictors. Urinary tract infection (OR 0.15, 95% CI 0.04–0.62), diabetes mellitus (OR 0.38, 95% CI 0.18–0.78), or thrombocytopenia (OR 0.33, 95% CI 0.18–0.60) were identified as the negative predictors of anaerobic BSI. Anaerobic BSI were not associated with worse prognosis in either adjusted (HR 1.08, 95% CI 0.68–1.72) or PS-matched analysis (HR 1.50, 95% CI 0.61–3.67).

***Conclusion.*** Anaerobic BSI accounted for a significant proportion (approximately 1 in 20) of community-onset BSI. Clinical predictors identified in this study may help guide the prescription of empiric anti-anaerobe antibiotics. The apparent adverse outcome associated with anaerobic BSI may be explained by the underline comorbidity, high-risk infection site, and inadequate initial antibiotics.

Table 1. Comparison of the source of bacteremia

| Source of bacteremia | Total | Anaerobic BSI (n=61) | Non-anaerobic BSI (n=1105) | P value |
|---|---|---|---|---|
| Intra-abdomen infection | 49 (4.2%) | 12 (19.7%) | 37 (3.3%) | <0.001* |
| Pneumonia | 159 (13.6%) | 12 (19.7%) | 147(13.3%) | 0.158 |
| Skin and soft tissue infection | 110 (9.4%) | 11 (18.0%) | 99 (9.0%) | 0.018* |
| Biliary tract infection | 165 (13.4%) | 10 (16.4%) | 146 (13.2%) | 0.477 |
| Primary bacteremia | 191 (16.4%) | 7(11.5%) | 184 (16.7%) | 0.288 |
| Liver abscess | 55 (4.7%) | 6 (9.8%) | 49 (4.4%) | 0.053 |
| Urinary tract infection | 297 (25.5%) | 2 (3.3%) | 295 (26.7%) | <0.001* |
| Spontaneous bacterial peritonitis | 40 (3.4%) | 2(3.3%) | 38(3.4%) | 0.947 |
| Infective endocarditis | 47 (4.2%) | 1 (1.6%) | 46 (4.2%) | 0.329 |
| Catheter related infection | 46 (3.9%) | 0(0%) | 46 (4.2%) | 0.104 |