

**Disclosures.** R. Patel, CD Diagnostics, BioFire, Curetis, Merck, Hutchison Biofilm Medical Solutions, Accelerate Diagnostics, Allergan, and The Medicines Company: Grant Investigator, Research grant – monies paid to Mayo Clinic. Curetis, Specific Technologies, Selux Dx, GenMark Diagnostics, PathoQuest and Genentech: Consultant and Scientific Advisor, Consulting fee – monies paid to Mayo Clinic. ASM and IDSA: Travel reimbursement and editor's stipends. NBME, Up-to-Date and the Infectious Diseases Board Review Course: Varies, Honoraria. Mayo Clinic: Employee, Salary. R. Banerjee, Accelerate Diagnostics, Biomerieux, BioFire: Grant Investigator, Research grant and Research support.

## 1053. Biofilm Production and Clinical Characteristics of *S. maltophilia* Causing Persistent or Relapsing Bacteremia

Seung Ji Kang, MD<sup>1</sup>; Tae Hoon Oh, MD<sup>1</sup>; Younggon Jung, MD<sup>2</sup>; Seong Eun Kim, MD<sup>1</sup>; Uh Jin Kim, MD<sup>1</sup>; Hee-Chang Jang, MD<sup>1</sup>; Kyung-Hwa Park, MD<sup>1</sup> and Sook-In Jung, MD<sup>1</sup>; <sup>1</sup>Chonnam National University Medical School and Hospital, Gwangju, Korea, Republic of (South), <sup>2</sup>Chonnam National University Hospital, Gwang ju, Korea, Republic of (South)

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**Background.** This study aimed to identify clinical or microbiological factors related to persistence or recurrence of *Stenotrophomonas maltophilia* bacteremia in adult patients.

**Methods.** S. maltophilia isolated from blood in two tertiary hospitals between 2011 and 2017 were investigated. Persistent bacteremia was defined as the consecutive blood culture positive for  $\geq 5$  days after initiation of appropriate antibiotics therapy. Relapse was defined as isolation of S. maltophilia from blood after completion of antibiotics treatment for the first episode of bacteremia. Biofilm formation was assessed in 96-well polystyrene plate with Trypticase Soy Broth using 0.5% crystal violet staining. The presence of smf-1 gene was detected by polymerase chain reaction.

Results. Of total 100 patients with S. maltophilia bacteremia, 10 of persistent, 8 of relapsing, and 46 of nonpersistent, nonrelapsing cases were investigated. The presence of indwelling urinary catheter (P = 0.011), nasogastric tube (P = 0.003), mechanical ventilator treatment (P = 0.001), and previous colonization of S. maltophilia (P = 0.016) were more frequently observed in patients with persistent bacteremia compared with nonpersistent, nonrelapsing bacteremia cases. In patients with relapsing bacteremia, hematologic malignancy (P = 0.022), neutropenia (P = 0.001), and concomitant isolation of S. maltophilia in clinical samples other than blood (P = 0.041) were more common than nonpersistent, nonrelapsing bacteremia patients. Catheter-related infection (37.0%) followed by pneumonia (28.3%) was the most common primary focus of nonpersistent, nonrelapsing bacteremia whereas pneumonia was the most frequent cause of bacteremia in both of persistent and relapsing cases (40.0% and 50.0%). Most of isolates (63 of 64) were susceptible to cotrimoxazole. The resistance to levofloxacin were comparable among isolates from persistent, relapsing and nonpersistent, nonrelapsing cases (10.0% vs. 12.5% vs. 15.2%, P = 0.988). Biofilm formation ability was not significantly different between three groups (optical density at 595, mean  $\pm$  SD, 0.69  $\pm$  0.34 vs.  $0.78 \pm 0.33$  vs.  $0.70 \pm 0.33$ , P = 0.529). The smf-1 gene was found in all isolates.

**Conclusion.** More careful treatment approaches to patients with risk factors for *S.maltophilia* treatment failure should be warranted.

Disclosures. All authors: No reported disclosures.

## 1054. Biofilm Formation Among *Escherichia coli* Bloodstream Infection Isolates Is Associated With Source of Bacteremia and Bacterial Sequence Type

Carolyn Chang, BS<sup>1</sup>; Felicia Ruffin, MSN, RN<sup>2</sup>; Vance G. Fowler Jr, MD<sup>3</sup> and Joshua T. Thaden, MD, PhD<sup>2</sup>; <sup>1</sup>Division of Infectious Diseases, Duke University, Durham, North Carolina, <sup>2</sup>Division of Infectious Diseases, Duke University Medical Center, Durham, North Carolina, <sup>3</sup>Duke University Medical Center, Durham, North Carolina,

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Background. The clinical impact of Escherichia coli biofilm formation is unknown.

**Methods.** Adults with *E. coli* bloodstream infections (BSI) were prospectively enrolled from 2002 to 2015. All *E. coli* isolates were genotyped using Multilocus sequence typing (MLST) and underwent crystal violet biofilm formation assay quantified by absorbance at 540 nm (OD540) in triplicate. Associations between biofilm formation and patient/bacterial characteristics were characterized by *t*-tests and ANOVA tests.

**Results.** Ninety-eight percent (186) of the 189 isolates formed detectable biofilms. Bacterial sequence type (ST) was associated with biofilm formation (P < 0.001), as ST73 (average OD<sub>540</sub> = 0.017) and ST393 (average OD<sub>540</sub> = 0.016) had higher average biofilm formation while ST69 (average OD<sub>540</sub> = 0.002) had lower biofilm formation. *E. coli* isolates with non-multidrug-resistant (non-MDR) phenotype were associated with increased biofilm formation (MDR: average OD<sub>540</sub> = 0.006; average non-MDR: OD<sub>540</sub> = 0.01; P = 0.003). BSI isolates arising from pneumonia or urine/pyelonephritis were associated with the highest biofilm production (P = 0.04). No associations were identified between biofilm formation and route of infection, APACHE-II score, mortality, or complications of BSI.

**Conclusion.** In this prospective study of *E. coli* BSI isolates, biofilm formation was associated with ST, non-MDR phenotype, and BSI source.

Disclosures. All authors: No reported disclosures.

**1055.** Epidemiology and Mechanisms of Carbapenem Resistance in Recurrent Extended-Spectrum β-Lactamase- Producing *Enterobacteriaceae* Bacteremia Samuel L. Aitken, PharmD<sup>1,2</sup>; Micah Bhatti, MD, PhD<sup>3</sup>; Pranti Sahasrabhojane, MS<sup>4</sup>; Jessica Galloway-Pena, PhD<sup>4</sup>; Xiqi Li, MS<sup>5</sup>; Frank P. Tverdek, PharmD<sup>2</sup>; Cagney Reeves, PharmD<sup>2</sup>; Patrick McDaneld, PharmD<sup>2</sup>; David Greenberg, MD, FIDSA<sup>6</sup> and Samuel Shelburne, MD, PhD, FIDSA<sup>7</sup>; <sup>1</sup>Center for Antimicrobial Resistance and Microbial Genomics (CARMiG), UTHealth McGovern Medical School, Houston, Texas, <sup>2</sup>Division of Pharmacy, The University of Texas MD Anderson Cancer Center, Houston, Texas, <sup>3</sup>Department of Laboratory Medicine, The University of Texas MD Anderson Cancer Center, Houston, Texas, <sup>4</sup>The University of Texas MD Anderson Cancer Center, Houston, Texas, <sup>6</sup>Infectious Disease, University of Texas Southwestern Medical Center, Dallas, Texas, <sup>7</sup>Infectious Diseases, MD Anderson Cancer Center, Houston, Texas

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**Background.** Carbapenems are the treatment of choice for bacteremia caused by extended spectrum  $\beta$ -lactamase producing *Enterobacteriaceae* (ESBL-E). The emergence carbapenem resistance (CR) in ESBL-E isolates has been described, however, the rate of such resistance in clinical settings is unknown. We describe the frequency and mechanisms of CR in recurrent ESBL-E bacteremia at an NCI-designated cancer center.

Methods. We performed a prospective whole genome sequencing (WGS) study and retrospective cohort review of adult (age ≥18 years) patients with ESBL-E bacteremia between January 2015 and July 2016. Recurrent bacteremia was defined as identification of the same organism in blood culture at any time following initial successful treatment. CR was defined as resistance to meropenem. Carbapenemase production was assessed in the microbiology laboratory using Carba-NP. Available paired isolates underwent WGS via Illumina HiSeq for assessment of clonality and identification of CR mechanisms.

**Results.** One hundred and sixteen patients with ESBL-E bacteremia were identified. *E. coli* was the most common organism (86%), followed by *K. pneumoniae* (12%), and *K. oxytoca* (2%). Recurrent bacteremia was identified in 17 (15%) patients (*E. coli* [n = 15], *K. pneumoniae* [n = 2]). Of these, 6 (35%) were CR and 5/6 (8%) were Carba-NP negative. All six recurrent CR isolates occurred in patients with letwemia. Five isolate pairs were available for WGS. In four of five pairs (three *E. coli*, one *K. pneumoniae*), CR emerged from the same strain causing the original infection; one recurrence was caused by a distinct *E. coli* with a OXA-48-like gene. Compared with parental strains, CR *E. coli* contained deletions in porin-encoding genes and had increased mapping depth for genes encoding CTX-M ESBLs. The *K. pneumoniae* was Carba-NP negative with no identifiable CR mechanism.

**Conclusion.** Emergence of CR following treatment for ESBL-E bacteremia was seen only in leukemia patients and was primarily due to porin loss and amplification of ESBL genes, rather than acquisition of exogenous carbapenemases. These are the first clinical data describing the molecular mechanism of ESBL-E transformation to CR. These data serve as the basis for future studies of antimicrobial stewardship interventions to limit the emergence of CR in ESBL-E.

Disclosures. S. L. Aitken, Shionogi: Scientific Advisor, Consulting fee. Medicines Company: Scientific Advisor, Consulting fee. Merck & Co.: Scientific Advisor, Consulting fee. Chung-Jong Kim, MD, PhD<sup>3</sup>; Kyoung-Ho Song, MD, PhD<sup>2</sup>; Chang Kyung Kang, MD<sup>5</sup>; Pyeong Gyun Choe, MD<sup>4</sup>; Ji Yun Bae, MD<sup>5</sup>; Hee Jung Choi, MD<sup>6</sup>; Younghee Jung, MD<sup>7</sup>; Seung Soon Lee, MD<sup>8</sup>; Wan Beom Park, MD, PhD<sup>4</sup>; Ji Hwan Bang, MD<sup>4</sup>; Eu Suk Kim, PhD<sup>9</sup>; Sang Won Park, MD, PhD<sup>4</sup>; Nam Joong Kim, MD, PhD<sup>4</sup>; Myoung-Don Oh, MD, PhD<sup>4</sup>; Hong Bin Kim, MD, PhD<sup>2</sup> and KIND Study Group; <sup>1</sup>Internal Medicine, Ewha Womans University, Seoul, Korea, Republic of (South), <sup>5</sup>Department of Internal Medicine, Seoul National University Bundang Hospital, Seongnam, Korea, Republic of (South), <sup>4</sup>Department of Internal Medicine, Seoul National University College of Medicine, Seoul, Korea, Republic of (South), <sup>6</sup>Department of Internal Medicine, Seoul, Korea, Republic of (South), <sup>6</sup>Department of Internal Medicine, Seoul, Korea, Republic of (South), <sup>7</sup>Division of Infectious Diseases, Hallym University Sacred Heart Hospital, Hallym University College of Medicine, Sacred Heart Hospital, Hallym University College of Medicine, Korea, Republic of (South), <sup>6</sup>Division of Infectious Diseases, Hallym University Sacred Heart Hospital, Hallym University College of Medicine, Anyang-si, Korea, Republic of (South), <sup>8</sup>Division of Infectious Diseases, Hallym University Sacred Hospital, Hallym University College of Medicine, Anyang-si, Korea, Republic of (South), <sup>8</sup>Seoul National University Sacred Hospital, Hallym University College of Medicine, Anyang-si, Korea, Republic of (South), <sup>8</sup>Division of Infectious Diseases, Republic of (South), <sup>9</sup>Division of Infectious Diseases, Republic of (South), <sup>9</sup>Division of Infectious Diseases, Hallym University Sacred Heart Hospital, Hallym University College of Medicine, Anyang-si, Korea, Republic of (South), <sup>9</sup>Division of Infectious Diseases, Republic of (South), <sup>9</sup>Seoul National University Bundang Hospital, Seoul, Korea, Republic of (South)

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**Background.** Central nervous system (CNS) complications occurring in patients with *Staphylococcus aureus* bacteremia (SAB) are the most severe complications. In this study, we compared clinical data of SAB patients between cases with and without CNS complication and analyzed the risk factor of CNS complications.

**Methods.** Data from cases with SAB occurred during 5 years at four hospitals were collected. The presence of CNS complications was confirmed by brain MRI, CT, or lumbar puncture. We excluded the cases who already had CNS lesions such as trauma, brain tumor, or cerebrovascular accident. We also excluded the cases who were died or transfer out <7 days of bacteremia onset. Cases were divided into complication group or noncomplication group according to the presence of CNS complication. We compared the clinical profiles between the groups, and analyzed the risk factor of CNS complications by multivariate logistic regression analysis.

**Results.** A total of 1,085 cases of SAB patients were included. Among these, 43 (4%) cases were complication group (embolic infarct [n = 23], brain hemorrhage [n = 8], infarct with hemorrhage [n = 8], and brain abscess or meningitis [n = 4]), while 810 (74%) cases were noncomplication group. Two hundred and forty-one cases were excluded. The results of multivariate analysis were shown in table. When selecting by having less than three factors among SOFA > 5, methicillin-susceptible, endovascular infection (weight 2), presence of metastatic infection and community onset, it helps to exclude CNS complications (AUC of ROC curve = 0.77, P < 0.01, sensitivity; 67.5%, specificity: 75.5%, positive predictive value: 12.9%, negative predictive value 97.7%).

Conclusion. CNS complication could be excluded by using clinical variables

| Variables                                | Complication<br>Group<br>(n = 43) | Non-<br>Complication<br>Group<br>(n = 801) | <i>P</i> -Value | Adjusted Odds<br>Ratio | <i>P</i> -Value |
|--|-----------------------------------|--|-----------------|------------------------|-----------------|
| Sex (m)                                  | 23 (54%)                          | 499 (62%)                                  | 0.25            | 1.4 (0.8-2.8)          | 0.28            |
| Age (mean)                               | 66 (j¾17)                         | 64 (j¾16)                                  | 0.38            |                        |                 |
| Community onset                          | 30 (70%)                          | 450 (56%)                                  | 0.08            | 1.3 (0.7-2.7)          | 0.44            |
| Methicillin-susceptible isolates         | 27 (63%)                          | 380 (47%)                                  | 0.05            | 1.9 (0.9–3.9)          | 0.07            |
| SOFA score (median)                      | 6 (3–9)                           | 3.5 (1-6)                                  | 0.01            | 1.1 (1.1-1.2)          | 0.01            |
| Duration of bacteremia (median)          | 4 (1-6)                           | 2 (0-5)                                    | 0.01            |                        |                 |
| Endovascular involvement of<br>infection | 18 (42%)                          | 106 (13%)                                  | <0.01           | 3.1 (1.5–6.3)          | 0.01            |
| Presence of any metastatic<br>infection  | 20 (47%)                          | 155 (19%)                                  | <0.01           | 2.3 (1.2–4.7)          | 0.02            |
| 90 day-mortality                         | 11 (34%)                          | 121 (19%)                                  | 0.03            |                        |                 |

Disclosures. All authors: No reported disclosures.

# 1057. Treatment Efficacy of Ceftriaxone vs. Cefazolin for Methicillin-Susceptible Staphylococcus aureus Infections

Kavitá Bhavan, MD<sup>1</sup>; Anisha Ganguly, BS, BA<sup>2</sup>; Helen King, MD<sup>2</sup>; Aurelia Schmalstieg, MD<sup>2</sup>; Norman Mang, PharmD<sup>3</sup> and Ryan Collins, PA<sup>4</sup>; <sup>1</sup>Infectious Disease, UT Southwestern Medical Center, Dallas, Texas, <sup>2</sup>UT Southwestern Medical Center, Dallas, Texas, <sup>3</sup>Parkland Health And Hospital System, Dallas, Texas, <sup>4</sup>Parkland Health and Hospital System, Dallas, Texas

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**Background.** Methicillin-susceptible *Staphylococcus aureus* (MSSA) infections are traditionally treated with intravenous (IV) nafcillin, oxacillin, or cefazolin, all antibiotics that require multiple doses per day. Despite theoretical limitations of using ceftriaxone in MSSA infections, some clinical studies suggest noninferiority of ceftriaxone compared with standard of care. At Parkland Memorial Hospital, many patients diagnosed with MSSA infections receive self-administered Outpatient Parenteral Antimicrobial Therapy (S-OPAT). Daily-dosed ceftriaxone is often used for convenience and feasibility of medication adherence.

*Methods.* We conducted a retrospective cohort study among S-OPAT patients receiving cefazolin and ceftriaxone for treatment of MSSA infections. We compared infection type and planned duration of therapy as baseline differences between the

treatment cohorts. Our clinical outcomes of interest were 30-day readmission rates and treatment failure as defined by repeat positive blood culture within 6 months.

**Results.** We identified 184 patients treated with cefazolin and 74 patients treated with ceftriaxone. Characteristics of treatment plan are shown in Table 1. There were no statistically significant differences in infection type or mean duration of therapy between the two treatment cohorts. Outcomes are shown in Table 2. There were no statistically significant differences in readmission rates and rate of treatment failure.

**Conclusion.** Our retrospective review suggests patients treated with ceftriaxone for MSSA bacteremia had similar clinical outcomes as those treated with cefazolin. While this study is limited in its retrospective nature, the findings suggest that ceftriaxone may be a safe and more convenient antibiotic option in certain MSSA infections.

|                                | Cefazolin ( $n = 184$ ) | Ceftriaxone ( $n = 74$ ) | P-Value |
|--------------------------------|-------------------------|--------------------------|---------|
| Infection type                 |                         |                          | 0.87    |
| Bacteremia                     | 106                     | 29                       |         |
| Osteomyelitis                  | 23                      | 30                       |         |
| Skin and soft-tissue infection | 14                      | 6                        |         |
| Endocarditis                   | 14                      | 2                        |         |
| Line-related                   | 11                      | 1                        |         |
| Pulmonary                      | 9                       | 2                        |         |
| GU                             | 5                       | 2                        |         |
| Other                          | 2                       | 2                        |         |
| Mean duration of therapy       | 30 days                 | 32 days                  | 0.26    |

Disclosures. All authors: No reported disclosures.

#### 1058. Prognostic Biomarkers for Persistent Bacteremia and Mortality in Complicated *S. aureus* Bloodstream Infection

<u>Yi Cao</u>, PhD<sup>1</sup>; Alessander Guimaraes, PhD<sup>1</sup>; Kyu Hong, PhD<sup>1</sup>; Oleg Mayba, phD<sup>1</sup>; Melicent Peck, MD, PhD<sup>1</sup>; Johnny Gutierrez, PhD<sup>1</sup>; Felicia Ruffin, MSN, RN<sup>2</sup>; Montserrat Carrasco-Triguero, PhD<sup>1</sup>; Jason Dinoso, PhD<sup>1</sup>; A. Asa Clemenzi-Allen, MD<sup>3</sup>; Catherine Koss, PhD<sup>4</sup>; Stacey A. Maskarinec, MD, PhD<sup>5</sup>; Henry F. Chambers, MD<sup>6</sup>; Vance G. Fowler Jr., MD<sup>7</sup>; Amos Baruch, PhD<sup>1</sup> and Carrie Rosenberger, PhD<sup>1</sup>; <sup>1</sup>Genentech Inc., South San Francisco, California, <sup>2</sup>Division of Infectious Diseases, Duke University Medical Center, Durham, North Carolina, <sup>3</sup>Division of HIV/AIDS, Infectious Diseases and Global Medicine, University of California San Francisco, San Francisco, California, <sup>4</sup>UCSF, San Francisco, California, <sup>6</sup>Infectious Diseases, Duke University Medical Center, Durham, North Carolina, <sup>6</sup>Clinical Research Services, University Medical Center, Durham, North Carolina, Clinical Research Services, San Francisco, California, San Francisco, Clinical and Translational Sciences Institute, San Francisco, California, <sup>7</sup>Duke University, Durham, North Carolina

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**Background.** Staphylococcus aureus is a leading cause of bacteremia, yet there remains a significant knowledge gap in the identification of relevant biomarkers that predict clinical outcomes in patients with *S. aureus* bacteremia. Heterogeneity in the host response to invasive *S. aureus* infection suggests that specific biomarker signatures could be utilized to differentiate patients prone to severe disease, thereby facilitating earlier implementation of more aggressive therapies. To further elucidate the inflammatory correlates of poor clinical outcomes in patients with *S. aureus* bacteremia, we evaluated the association between a panel of blood proteins at initial presentation of bacteremia and disease severity outcomes.

**Methods.** We conducted an observational study (n = 32) to evaluate the prognostic value of circulating protein biomarkers for mortality and persistent bacteremia in patients with *S. aureus* bloodstream infections. A case–control study of 124 patients with complicated confirmed *S. aureus* bloodstream infections was used to validate our findings in the observational study.

**Results.** We identified 13 candidate proteins that were correlated with mortality and persistent bacteremia by multiple comparisons. Further statistical modeling identified IL-8 and CCL2 as the strongest individual predictors of mortality, with the combination of these biomarkers having the best power to classify fatal outcome. Baseline IL-17A levels were elevated in patients with persistent bacteremia, endovascular and metastatic tissue infections.

**Conclusion.** The results demonstrate the potential utility of selected biomarkers to distinguish patients with the highest risk for treatment failure and bacteremia-related complications, providing a valuable tool for clinicians in the management of *S. aureus* bacteremia. Additionally, these biomarkers could identify patients with the greatest potential to benefit from novel therapies in clinical trials.

Disclosures. All authors: No reported disclosures.

#### 1059. *Staphylococcus aureus* Bacteremia Treatment: Results From Pilot Surveillance in Four US States

Sarah Kabbani, MD MSc<sup>1</sup>; Kelly Jackson, MPH<sup>1</sup>; Lauren Epstein, MD, MSc<sup>1</sup>; Anita Gellert, RN<sup>2</sup>; Carmen Bernu, BS<sup>3</sup>; Rahsaan Overton, MDFH<sup>4</sup>; Joelle Nadle, MPH<sup>5</sup>; Ghinwa Dumyati, MD, FSHEA<sup>6</sup>; Ruth Lynfield, MD, FIDSA<sup>3</sup>; Susan M. Ray, MD<sup>7</sup>; Erin Epson, MD<sup>8</sup> and Isaac See, MD<sup>1</sup>; <sup>1</sup>Division of Healthcare Quality Promotion, Centers for Disease Control and Prevention, Atlanta, Georgia, <sup>3</sup>NY Emerging Infections Program, Center for Community Health and Prevention, University of Rochester Medical Center, Rochester, New York, <sup>3</sup>Minnesota Department of Health, St. Paul, Minnesota, <sup>4</sup>Georgia Emerging Infections Program, Atlanta, Georgia, <sup>5</sup>California Emerging Infections Program, Oakland, California, <sup>6</sup>University of Rochester Medical Center, Rochester, New York, <sup>7</sup>Emory University