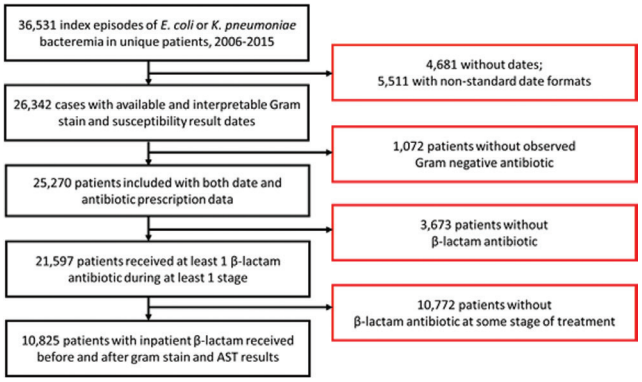

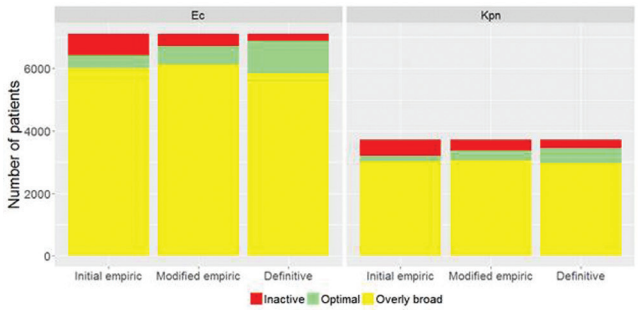

Disclosures. **R. Patel**, CD Diagnostics, BioFire, Curetis, Merck, Hutchison Biofilm Medical Solutions, Accelerate Diagnostics, Allergan, and The Medicines Company: Grant Investigator, Research grant – monies paid to Mayo Clinic. Curetis, Specific Technologies, Selux Dx, GenMark Diagnostics, PathoQuest and Genentech: Consultant and Scientific Advisor, Consulting fee – monies paid to Mayo Clinic. ASM and IDSA: Travel reimbursement and editor's stipends, 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

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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 ≥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 ± SD, 0.69 ± 0.34 vs. 0.78 ± 0.33 vs. 0.70 ± 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

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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 $OD_{540} = 0.017$) and ST393 (average $OD_{540} = 0.016$) had higher average biofilm formation while ST69 (average $OD_{540} = 0.007$) and ST405 (average $OD_{540} = 0.002$) had lower biofilm formation. *E. coli* isolates with non-multidrug-resistant (non-MDR) phenotype were associated with increased biofilm formation (MDR: average $OD_{540} = 0.006$; average non-MDR: $OD_{540} = 0.01$; $P = 0.003$). 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

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Background. Carbapenems are the treatment of choice for bacteremia caused by extended spectrum β-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 (83%) were Carba-NP negative. All six recurrent CR isolates occurred in patients with leukemia. 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.