factors associated with disease severity could aid in the management and prevention of *S. Aureus* infections.

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624. Prevalence of ST171 in *Enterobacter* Isolates from 2001 to 2013 in 15 Hospitals in NYC

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Background. ST171 was identified as the most prevalent ST in carbapenem-resistant *Enterobacter* in 26/106 typed isolates from New York City by Gomez-Simmonds et al. There is no study of prevalence of this ST over time. To evaluate a large sample of *Enterobacter*, we designed a PCR assay to identify ST171 isolates rapidly.

Methods. Isolates were collected in NYC as part of a cross-sectional Gram-negative antibiotic resistance assessment in the years, 2001, 2003–2004, 2006, 2009, and 2013. Agar dilution MIC were obtained for all isolates as part of these studies. We assayed 284 clinical *Enterobacter* isolates for ST171 using a novel PCR assay, forward primer AGAAGGACGATTTTGGCGCGGT and reverse ACTACGGTGGTAAAGAATGATCGCCA. Following amplification, ST171 positive isolates were identified by gel electrophoresis. *Enterobacter* isolates were also assessed for the presence of bla_{KPC} using a previously described RT-PCR assay.

Results. ST171 was identified in 17/284 (6%) *Enterobacter* isolates. This sample collection was heavily antibiotic-resistant with 83/284 *Enterobacter* isolates harboring bla_{KPC}. Of the 284 isolates, 142 (50%) were resistant to any carbapenem and 113 (40%) were resistant to ceftazidime. Of 17 ST171 positive *Enterobacter* isolates, 14(82%) were also contained bla_{KPC}. These isolates were highly resistant, with 10/17(59%) exhibiting phenotypic resistance to carbapenems and 14/17(82%) were phenotypically resistant to ceftazidime. Twelve of the 17 isolates occurred in clusters of isolates of the same species occurring in a single hospital at the same time. There were 2 clusters of 3 cases and 3 clusters of 2 patients each. The oldest ST171 strain identified was an *Enterobacter cloacae* isolate collected in 2001. Prevalence of ST171 was calculated for each year of data: 1/40(2.5%) in 2001, 0/20 in 2003-04, 7/73(9.6%) in 2006, 6/38(15.8%) in 2009, and 3/113(2.7%) in 2013. Each sample collection year included a different group of participating hospitals.

Conclusion. Here we evaluate a novel PCR assay for ST171 in *Enterobacter spp.* The oldest ST171 identified from 2001 was carbapenem susceptible suggesting that this strain type later acquired plasmid mediated carbapenem resistance. Prevalence of ST171 tracks with the prevalence of carbapenem resistance with a peak observed in 2009 for both and a decrease in 2013

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

625. CRISPR-*Cas* May Prevent Acquisition of Drug Resistance in *Klebsiella pneumoniae*

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Background. *Klebsiella pneumoniae (Kp)* is a Gram-negative bacterium that causes nosocomial UTIs, pneumonia, and sepsis. Carbapenem-resistant *Kp* (CR-*Kp*) is associated with hospital outbreaks, is difficult to treat, and has high mortality rates prompting study of how resistance is obtained. In U.S. strains, resistance to Carbapenems is primarily conferred by genes bla^{KPC-2} and bla^{KPC-3}. Transmission of these genes is via plasmids and to investigate their acquisition, this project analyzed the function of CRISPR-*Cas* in Carbapenem sensitive *Kp* (CS-*Kp*) hospital strains. The CRISPR-*Cas* system has been found to suppress homologous gene transfer and prevent integration of new genes by plasmids or bacteriophages. This study's hypothesis is that *Kp* strains that lack CRISPR-*Cas* can acquire CR plasmids, while those strains that have CRISPR-*Cas*are protected from gaining these plasmids and can maintain sensitivity to Carbapenems.

Methods. *Kp* strains from the urine of patients from Montefiore Medical Center and Stony Brook University Hospital were collected and sensitivity to Carbapenems was determined by chart review. Next, hospital strains were screened for CRISPR-*Cas* using PCR. 4 CS-*Kp* strains (strains 1 and 2 without CRISPR-*Cas*, and strains 3 and 20 with CRISPR-*Cas*) were studied. bla^{KPC-2}, bla^{KPC-3}, and control plasmid pPROBEKT-GFP (Km^R) were then transformed into *Kp*by standard electroporation. Meropenem agar plates for CR-containing plasmids, Kanamycin for control plasmids, and PCR were used to evaluate transformation success.

Results. Successful transformation of bla^{KPC-2}, bla^{KPC-3}, and the control plasmid was achieved in strains 1 and 2, which lacked CRISPR-*Cas*. Successful transformation of the control plasmid was achieved in strains 3 and 20. However, neither bla^{KPC} plasmid could be transformed into strain 3, and while very low success was seen with bla^{KPC-3} in strain 20, bla^{KPC-2}could not be transformed into that strain either.

Conclusion. This study supports the hypothesis that CRISPR-*Cas* prevents acquisition of drug resistance plasmids. It is notable that a CS-*Kp* strain with CRISPR-*Cas* was protected against one KPC gene and not fully against the other. This may indicate a difference in the CRISPR sequences in individual *Klebsiella* strains.

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626. Differences in Gene Expression Levels of Methicillin-Resistant *Staphylococcus aureus* Genes Between Persistent and Resolving Bacteremia

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Background. Persistent MRSA bacteremia (PB) is associated with higher mortality than resolving MRSA bacteremia (RB). We previously described that PB and RB isolates had no significant differences in genotypes and microbiologic characteristics. In other small studies, the presence of specific genes or phenotype characteristics was associated with PB, but the results were inconsistent. Aim of this study was to determine whether differences in the expression of major genes contribute to the development of PB.

Methods. We analyzed expression levels of major regulatory genes (agr, sarA, sigB, graRS, walKR, saeRS, and vraRS) and virulence factors (htrA, prsA, murZ, spa, clfA, clfB, sdrC, sdrD, sdrE, ebps, fib, hla, psma, icaA, seg, and sen) in 40 MRSA strains isolated from 20 patients with PB (>7 days) and 20 patients with RB (< 3 days) who were matched for clinical and epidemiologic characteristics and bacterial genotypes. Relative gene expression level to gyrB was determined using real-time RT-PCR. In the same way, differential expression of the genes between the first and last isolates from 20 patients with PB was analyzed to evaluate changes of gene expression during PB. In addition, RNA-seq was performed on selected MRSA strains to evaluate the overall differential expression of genes.

Results. There was no difference in the expression level of *agr* between PB and RB isolates. However, significant differences in gene expression levels between PB and RB isolates were observed in the following genes. Gene with increased expression levels in PB was *sigB*, global regulator. Genes with decreased expression levels in PB included *sarA*, global regulator; *graRS, walKR*, and *saeRS*, two-component regulatory systems; *htrA*, *clfA*, *sdrD, sdrE, ebps*, and *fib*, surface protein genes; *seg* and *sen*, secreted protein genes (Figure 1). A similar trend was found in RNA-seq. There were no significant differences in expression of specific genes between the first and last isolates of PB.

Conclusion. Our results suggest that PB may develop due to infection caused by MRSA strain with altered gene expression rather than changes in specific gene expression levels during bacteremia.

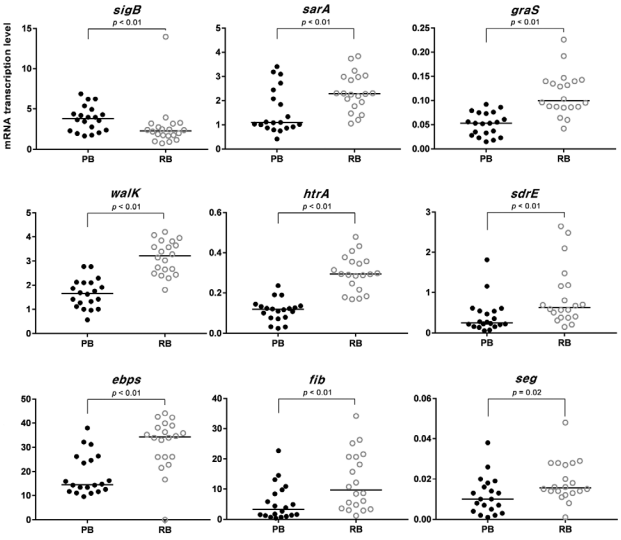

Figure 1. Differences in specific gene expression between PB and RB isolates. mRNA transcription level is presented as relative expression ratio of target genes to *gyrB*. PB, persistent MRSA bacteremia; RB, resolving MRSA baceremia.

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627. Virulence Factors of Healthcare Associated Infection by Uropathogenic *E. coli* Strains Isolated in Korea

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Background. *Escherichia coli* (*E.coli*) is most predominant organism of urinary tract infection. There are only a few studies about the difference of virulent factors of healthcare associated urinary tract infection.

Methods. The study was performed on 133 *E. coli*isolates from patients with urinary tract infection who were diagnosed at Keimyung university of Dongsan medical center from February 2015 through May 2016. Phylogenetic group and 29 virulence factors were identified by multiplex PCR. We compared antimicrobial susceptibility and virulence factors according to community acquired and healthcare associated infection.

Results. The phylogenetic group analysis reveals that most of uropathogenic *E.coli* are group B2 and D. B2 was more frequently observed and D2 was lesser in healthcare associated infection: B2(95.5% vs. 72.1%), D(4.5% vs. 23.4%). Among the virulence factors, *PAI, iutA* were more related with healthcare associated infection. *kpsMT II* and K1 serotype was less observed in healthcare associated infection. Relapse within 3 months was more frequently observed in healthcare associated infection. Healthcare associated infection was more related with antimicrobial resistance and extended spectrum β-lactamase producing *E.coli*.

Conclusion. In our study, community acquired and healthcare associated infection showed some differences of virulence factor in uropathogenic *E. coli*. Healthcare associated infection showed high presentation of iron metabolism-related virulence factor. Relapse was more frequently observed in healthcare associated infection.

	Virulence factor	Community(*n* = 111)	Healthcare(*n* = 22)	*p*		Virulence factor	Community(*n* = 111)	Healthcare(*n* = 22)	*p*
Adhesion	*papA*	74(66.7%)	14(63.6%)	0.784	Iron metabolism	*fyuA*	107(96.4%)	22(100%)	1.000
	firnH	109(98.2%)	22(100%)	1.000		*iutA*	74(66.7%)	21(95.5%)	0.006
	papEF	8(7.2%)	0	0.352	Toxin	*hlyA*	30(27.0%)	10(45.5%)	0.085
	sfa/focED	17(15.3%)	1(4.5%)			*cvaC*	8(7.2%)	2(9.1%)	0.671
	nfaE	2(1.8%)	0	1.000		*cnf1*	30(27.0%)	10(45.5%)	0.085
	afa/draBC	9(8.1%)	4(18.2%)	0.229	Protection	*kpsMTIII*	3(2.7%)	0	1.000
	focG	15(13.5%)	0	0.131		*kpsMTII*	70(63.1%)	7(31.8%)	0.007
	papC	78(70.3%)	14(63.6%)	0.538		*traT*	82(73.9%)	20(90.9%)	0.084
	papG	77(69.4%)	14(63.6%)	0.597	Others	*PAI*	78(70.3%)	21(95.5%)	0.013
	papG allele II	74(66.7%)	14(63.6%)	0.597		*ibeA*	7(6.3%)	1(4.5%)	1.000

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628. The Gut Microbiome: An Advanced Understanding of Microbial Health Across Countries and Cultures

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Background. Recent research has shown that the microbiome of the human gut plays a significant role in an individual's health. Gut microflora composition can be correlated with health or disease, such as Crohn's disease and colon polyps. The gut microbiome is known to vary among individuals and across geo-locations. Therefore, measuring the extent of such variation is critical in determining the baseline microbiome.

Methods. CosmosID has developed an innovative analysis platform to detect, identify, and characterize microorganisms in a metagenomic sample, using its ultra-fast bioinformatics tool and curated databases containing more than 65,000 genomes.

Over 500 whole genome shotgun (WGS) metagenomic sequenced samples of human stool of healthy and diseased subjects were downloaded from public databases, including the Human Microbiome Project (HMP). Samples from geographic locations, including Spain, Denmark, Tanzania, India, Japan, and the United States, comprised the dataset. The samples were analyzed using CosmosID's automated cloud-based metagenomics software to investigate correlation of geographical location with the microbiome and to analyze differences between healthy and diseased microflora.

Results. Diverse gut microflora, including bacteria, viruses, fungi, and protists, were identified in samples collected from different geographic regions. Samples from the same geographic locations, such as Denmark and Spain, showed differences in microbiome composition, but more limited compared with samples from Tanzania. Additionally, the gut microbiome of healthy individuals from all locations revealed the presence of pathogens with genomic potential for causing disease. These results will be used to assess differences in gut microbial composition of individuals from different geographic locations and to characterize baseline compositions for future analyses.

Conclusion. Subspecies and strain level data describing the microbiome of healthy and diseased individuals provide valuable in-depth information for the clinician, including identifying the community resistome and virulence.

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629. The Gut Microbiome in Pediatric Allogeneic Hematopoietic Stem Cell Transplant Patients

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Session: 72. Microbiome

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Background. Studies in adult allogeneic hematopoietic stem cell transplant (allo-HSCT) recipients demonstrate 1) domination by certain taxa correlates with subsequent bacteremia and 2) diversity at engraftment is an independent predictor of mortality. In limited pediatric literature, one study of 10 patients showed higher relative abundance of Bacteroidetes in pre-HSCT samples in patients without acute graft vs. host disease (aGVHD). In this study we explore the post-HSCT gut microbiome in patients receiving myeloablative (MA) vs. reduced intensity (RIC) pre-transplant conditioning regimens.

Methods. 119 fecal specimens were collected from 17 patients at start of conditioning, 2 weeks post-transplant, monthly and with fever or infection episodes until 6 mo. Bacterial communities were characterized by sequencing the V4 region of the bacterial 16S rRNA gene using Illumina Miseq. Bacterial density was estimated by 16S rRNA gene copy number measured by qPCR.

Results. Patient characteristics included median age 9.7 years (0.3 to 20.5), females 53%, first time transplant 70%, matched unrelated donors 70% and bone marrow stem cells 70%. 10 patients (8 with malignancy) received MA regimen and 7 with non-malignant diagnoses received RIC. Outcomes included aGVHD in First year in 6 (35%), chronic GVHD in 4 (28%) survivors past 6 mo and 12 (70%) patients surviving to one year. Fever or infection episodes in First 6 mo occurred at median 2 episodes (1 to 7) including 8 bacteremia episodes, with intestinal domination preceding two (*E cloacae* and *E fecalis*). There was a significant decrease in bacterial density in engraftment samples compared with pre-transplant samples (median 9.9 E+7 copies (cp)/mg of stool vs. 7.6 E+8 cp/mg, $P = 0.008$). Bacterial density was significantly lower (1.1E+5 cp/mg vs. 2.6E+8 cp/mg, $P = 0.007$) and Bacteroidetes domination (>30% of taxa in a sample) less common in engraftment samples of patients with aGVHD compared with those without aGVHD.

Conclusion. Allo-HSCT is associated with disruption of intestinal flora in pediatric patients. Dominance of the gut microbiota by a specific taxon may be associated with bacteremia with that organism. In preliminary analysis, bacterial density was lower and Bacteroidetes domination was less frequent in patients with aGVHD.

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630. SYN-006, a Novel Carbapenemase, Intended to Protect the Gut Microbiome from Antibiotic-Mediated Damage May Also Reduce Propagation of Carbapenem-Resistant Pathogens

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Background. B-lactam antibiotics that are excreted in bile can damage the gut microbiota, leading to serious adventitious infections and propagation of antibiotic