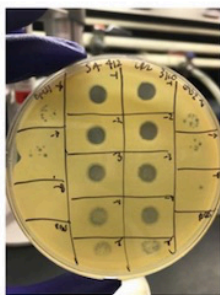


1a

		Agar overlay, PFU/ml	
		OMS1	OMS2
MRSA	SA408	7.5x10 ⁷	5x10 ⁸
	SA409	5x10 ⁷	1.5x10 ⁸
	SA412	2.0x10 ¹⁰	2.5x10 ¹⁰
	SA413	1.5x10 ⁷	1.75x10 ⁷
	SA419	2.5x10 ⁷	2.5x10 ⁷
	DVT1125	5x10 ⁵	1.5x10 ⁶
	DVT1126	5x10 ⁸	2.5x10 ⁹
	DVT1127	5x10 ⁵	5x10 ⁵
	DVT1128	5x10 ⁵	1.5x10 ⁶
	MSSA	SALAB	7.5x10 ⁸
VAZ#1		2.5x10 ⁸	5x10 ¹⁰
VAZ#2		2.5x10 ⁸	1.5x10 ⁹
GRIM		1x10 ⁹	5x10 ⁹
JON		7.5x10 ⁴	1x10 ⁴
PAR		1x10 ⁴	1x10 ⁴

1b



1c

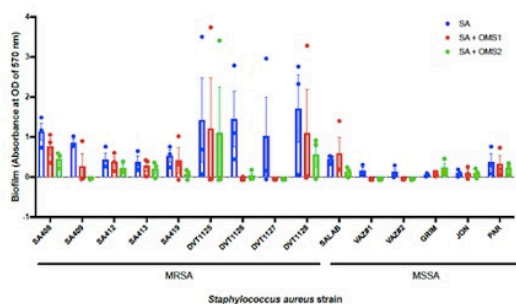


Figure 1a) Phage titers in PFU/ml obtained from each bacterial isolate via agar overlay method. 1b) Representative agar plate demonstrating lytic plaques from OMS1 and OMS2 on a *Staphylococcus aureus* LVAD isolate. 1c) Biofilm biomass of *Staphylococcus aureus* isolates alone (SA) or with individual phage (SA+OMSA1 and SA+OMS2) assessed by optical density readings at 570 nm; error bars represent standard error of the mean.

Conclusion. We demonstrated *in vitro* lytic and anti-biofilm activity of 2 *S. aureus* phages against clinical *S. aureus* isolates from patients with LVAD infection. Our data suggests that phage susceptibility measured with agar overlay does not always correlate with phage susceptibility of *S. aureus* biofilms, suggesting that more than one method should be used to assess *in vitro* activity. We plan to assess for synergistic activity with the phage combination.

Disclosures. Ryan K. Shields, PharmD, MS, Shionogi (Consultant, Research Grant or Support) Ran Nir-Paz, MD, BiomX (Consultant) Technophage (Scientific Research Study Investigator, Advisor or Review Panel member) Saima Aslam, MD, MS, BioMx (Consultant) Cystic Fibrosis Foundation (Grant/Research Support) Gilead (Consultant) Johnson and Johnson (Consultant) Merck (Consultant)

1277. Colonization Rates for Antimicrobial-resistant Bacteria in Kenya: An

Antibiotic Resistance in Communities and Hospitals (ARCH) Study Sylvia Omulo, PhD¹; Ulzii-Orshikh Luvsansharav, PhD²; Teresa Ita, BS³; Robert Mugoh, BSc⁴; Mark Caudell, PhD⁴; Brooke M. Ramay, Pharm D⁵; Guy H. Palmer, DVM, PhD¹; Linus Ndegwa, PhD⁶; Jennifer Verani, MD MPH⁷; Susan Bollinger, MPH, MT(ASCP)⁸; Aditya Sharma, MD⁹; Douglas Call, PhD¹; Rachel Smith, MD, MPH¹; ¹Washington State University, Pullman, Washington; ²CDC, Decatur, Georgia; ³Washington State University Global Health - Kenya, Nairobi, Nairobi Area, Kenya; ⁴Food and Agriculture Organization of the United Nations, Nairobi, Nairobi Area, Kenya; ⁵Universidad del Valle de Guatemala, Center for Health Studies, Paul G. Allen School for Global Health, Washington State University, Pullman, USA, Guatemala City, Sacatepequez, Guatemala; ⁶US CDC Kenya Office, Nairobi, Nairobi Area, Kenya; ⁷Centers for Disease Control and Prevention, Atlanta, Georgia; ⁸U.S. Centers for Disease Control and Prevention, Atlanta, Georgia

Session: P-72. Resistance Mechanisms

Background. Characterization of antimicrobial-resistant organism (ARO) colonization is critical to understand transmission dynamics and infection risk, however data in resource-limited settings are scarce. We estimated the prevalence of Enterobacteriales colonization with extended-spectrum cephalosporin-resistance (ESCrE), carbapenem-resistance (CRE) and methicillin-resistant *Staphylococcus aureus* (MRSA) among community residents and hospitalized patients in rural (Siaya County) and urban (Kibera) Kenya.

Methods. Community-dwelling adults and children were enrolled via cluster randomized sampling. Inpatients of all ages were enrolled by simple random sampling. Stool/rectal and nasal swabs were collected and screened for ESCrE, CRE and MRSA, respectively, using HardyChrom™ media. Vitek2™ was used for isolate confirmation and antibiotic susceptibility testing. Fisher's exact tests were used to compare prevalence of AROs.

Results. The prevalence of ESCrE was higher for the urban hospital (69.8%, 263/377) compared to rural hospitals (62.7%, 298/475, $P=0.04$); a similar pattern was

evident for CRE (16.7%, 63/377 and 6.5%, 31/475, respectively, $P<0.01$). The prevalence of MRSA was 3.2% for both urban and rural hospitals ($P=0.99$). For adults, the prevalence of ESCrE was higher in Kibera households (51.4%, 346/673) compared to Siaya (44.6%, 283/634, $P=0.02$) while the prevalence of both CRE and MRSA was $<3\%$ for both areas and did not differ significantly (CRE, $P=0.13$, MRSA, $P=0.14$). There was no significant difference between urban and rural children for ESCrE (47.7%, 74/155 and 53.4%, 135/253, $P=0.31$); both CRE and MRSA were rarely detected ($<2\%$) with no difference across settings (CRE, $P=1.0$, MRSA, $P=0.42$). Among Enterobacteriaceae recovered, *Escherichia coli* and *Klebsiella* spp. predominated.

Conclusion. Colonization with AROs were widespread in households and hospitals in urban and rural areas. Hospitals with elevated prevalence of highly transmissible AROs should consider whether implementation of colonization screening can be incorporated as part of their infection prevention and control programs. Risk factors for ARO colonization should be elucidated to identify novel prevention strategies.

Disclosures. All Authors: No reported disclosures

1278. Testing the Synergistic Effect of Ceftazidime-Avibactam Plus Aztreonam on Metallobeta-lactamase-Producing Non-Fermenting Gram-negative Bacteria

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Session: P-72. Resistance Mechanisms

Background. Metallo-beta-lactamases (MBL) are rapidly becoming a more widespread form of antimicrobial resistance. MBL are class B beta-lactamases that use zinc rather than serine in their active site and are only inactivated by monobactams, such as aztreonam. Unfortunately, most MBL-producing organisms also produce aztreonam-inactivating beta-lactamases. Synergy between ceftazidime-avibactam and aztreonam is well documented for MBL-producing Enterobacteriaceae but has not been tested extensively in non-fermenting Gram-negative bacteria. This study evaluates the susceptibilities of non-fermenting Gram-negative bacteria via E-test to this combination *in vitro*, in order to provide support for use to treat infections from these organisms.

Methods. The antibiotic combination ceftazidime-avibactam+aztreonam was tested against a total of 33 isolates, including MBL-producing *Pseudomonas aeruginosa*, *Pseudomonas putida*, and the intrinsically aztreonam resistant *Acinetobacter baumannii* using the E-test method. MBL-producing Enterobacteriaceae were included as positive controls. All isolates were also tested against ceftazidime alone, aztreonam alone, and ceftazidime-avibactam. Bacterial isolates were procured from the Multidrug-resistant organism Repository & Surveillance Network at the Walter Reed Army Institute of Research. Antimicrobial resistance genes were previously identified by whole genome sequencing

Results. Of 13 *Pseudomonas* spp. isolates tested, 9 were resistant, 3 were intermediate, and 1 was susceptible to aztreonam. Synergistic testing of ceftazidime-avibactam+aztreonam reduced the MIC of 4 *Pseudomonas* isolates by 1-2 doubling dilutions. While *Acinetobacter* spp. are usually considered intrinsically resistant to aztreonam, synergistic testing of ceftazidime-avibactam+aztreonam reduced the MIC of all 12 isolates tested by 1 to 3 doubling dilutions.

Conclusion. The ability of ceftazidime-avibactam+aztreonam to reduce the MICs of *Acinetobacter baumannii* and MBL-producing *Pseudomonas aeruginosa* is a potentially promising therapeutic option when faced with growing antimicrobial resistance.

Disclosures. All Authors: No reported disclosures

1279. Risk Factors for Colistin Resistance Among Carbapenem-Resistant *Klebsiella pneumoniae* (CRKP) in a Network of Long-term Acute Care Hospitals (LTACHs)

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Session: P-72. Resistance Mechanisms

Background. Colistin resistance in CRKP presents a serious clinical challenge for patients of LTACHs. However, risk factors for colistin-resistant CRKP have not been previously characterized in this population. Here, we determined risk factors for colistin resistance among CRKP isolates from a network of LTACHs.

Methods. CRKP clinical cultures were collected from 21 Kindred Healthcare LTACHs in 4 US states (California, Texas, Florida, Kentucky) from 8/1/14-7/25/15. Cultures collected within 30 days of a prior CRKP culture from the same patient were excluded. Colistin resistance (minimum inhibitory concentration ≥ 4) was determined using a custom Sensititre™ broth microdilution assay (ThermoFisher Scientific, Waltham, MA). Multivariate logistic regression was performed to evaluate candidate risk factors of age, sex, cirrhosis, chronic kidney disease, culture source, length of stay, indwelling line or tracheostomy, and antibiotic exposure (colistin, fluoroquinolones, 3rd-5th generation cephalosporins, piperacillin-tazobactam, carbapenems, and aminoglycosides) for ≥ 48 hours in the prior 30 days.

Results. Among 430 CRKP cultures (237 respiratory, 145 urine, 38 blood, 10 wound) from 375 patients, 144 (33.5%) were colistin-resistant. In multivariate analysis,