patient-days increase in carbapenem consumption (95% CI, 1.01–1.03; $P < 0.001$). There were 40 patients with NSEE and 134 patients with NSOCE. In the multivariate analysis, lower carbapenem exposure was significantly associated with the NSEE group (adjusted odds ratio: 0.25; 95% CI, 0.11–0.56). No difference in 14-day and 30-day all-cause mortality between NSEE group and NSOCE group was observed.

Conclusion. The incidence of CRE has risen significantly over a 5-year period at our institution. The important risk factor for nonsusceptibility to other carbapenems compared with nonsusceptibility to ertapenem alone was previous carbapenem use. Our hospital-wide carbapenem use has significantly increased over time, and associated with the increasing incidence of CRE.

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1175. Clinical and Microbiological Features of *Klebsiella pneumoniae* Liver Abscess Caused by Multidrug-Resistant Strains

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Background. The endemic *Klebsiella pneumoniae* liver abscess (KPLA) in East Asian countries are usually caused by hypervirulent strains. These hypervirulent strains are usually susceptible to commonly used antibiotics, aside from their intrinsic resistance to ampicillin. However, hypervirulent *K. pneumonaie* strains with multidrug-resistant (MDR) phenotype has been reported recently. We aim to investigate clinical and microbiological features of KPLA caused by MDR-resistant strains, and the evolution of drug-resistance in the resistant strain causing recurrent KPLA.

Methods. Patients with KPLA were retrospectively identified at Taipei Veterans General Hospital during January 2013 to February 2018. Capsular genotypes were analyzed in all *K. pneumoniae* isolates. Antimicrobial-resistant mechanisms were determined for MDR isolates. Pulse-field gel electrophoresis (PFGE), conjugation experiment, and *in vivo* mice lethality were determined on the strains from a patient with recurrent infection.

Results. During the study period, a total of 211 patients with KPLA, and five patients with recurrence were identified. Most of *K. pneumoniae* isolates ($n = 175$, 83.3%) belonged to capsular type K1/K2/K5/K20/K54/K57. Nineteen MDR strains were identified and 15 of them had virulent capsular types (K1 = 7, K2 = 5, K5 = 2, K54 = 1). The major resistance mechanisms of these MDR strains involved the presence of β-lactamases and the overexpression of efflux pumps. The in-hospital mortality of KPLA caused by MDR strains was not significantly higher than wild-type stains (10.53% vs. 4.69%, $P = 0.275$). In a case with recurrent KPLA, the recurrent capsular type K1 strain (TVGHKP2611) with *bla*SHV-12 was genetically identical to the primary wild-type resistance strain (TVGHKP2329) by PFGE. The SHV-12-carrying plasmid from TVGHKP2611 was successfully conjugated to *Eschericia coli* J53. TVGHKP2611 retained high virulence similar to TVGHKP2329 in mice lethality study (median lethal dose < 500 CFU) despite carrying the resistance determinant.

Conclusion. MDR *K. pneumoniae* strains belonging to virulent capsular types have emerged in KPLA. One SHV-12 producing capsular K1 strain causing recurrent KPLA retained its high virulence, which signals this highly pathogenic and resistant strain could be a major concern in the future.

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1176. Risk Factors for ESBL *Enterobacteriaceae* Colonization Identified by Universal Admission Screening in a London Teaching Hospital

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Background. Individual risk factors such as antibiotic use and foreign travel are typically associated with ESBL-producing Enterobacteriaceae (ESBL-E) carriage. Few studies have evaluated variations in community demographics or social and material deprivation as risk factors for ESBL-E carriage.

Methods. All admissions to a London hospital group were screened for ESBL-E carriage from rectal swabs for four months in 2015. Patients completed a risk factor questionnaire, and those with a residential postcode in the catchment area were linked to a database containing community-based risk factor data. Risk factors for ESBL-E carriage were determined by binary logistic regression. Isolates that were ESBL-E phenotypically were confirmed by microarray (CT103XL Check-MDR, CheckPoint). The Check-MDR array simlutaneously detects common ESBL and carbapenemase genes, including plasmid-mediated AmpC. Genotypic ESBL-E were split into three groups: CTX-M-15 (Group 1), CTX-M-9 (Group 2), and other (Group 3) for further analysis.

Results. Three hundred sixty (9.0%) of 4,006 patients carried ESBL-E; of which, 1,633 had a residential postcode within the catchment area. In multivariable analysis, risk factors for phenotypic ESBL-E carriage included travel to Asia (OR 5.0, CI 2.5–10.0) or Africa (OR 2.9, CI 1.2–7.0) in the past 12 months, and two or more courses of antibiotics in the past 6 months (OR 2.2, CI 1.5–3.4). Residence in an area with a high proportion of Arab residents and residence in an area with a low proportion of houses with two or more bedrooms were associated with ESBL-E carriage in univariable but not multivariable analysis. Risk factors for the three genotypic ESBL groups were broadly similar to the analysis of phenotypic ESBL carriage, although the number of days traveling abroad in the past 12 months was more associated with Group 1 (CTX-M-15) and Group 3 (other), and older age was associated with Group 3 (other) ESBLs.

Conclusion. We linked individual risk factor information with community-based risk factor information, concluding that individual risk factors (including antibiotic use and overseas travel) were more important than community-based risk factors for predicting colonization with ESBL-E at the time of hospital admission. This information is useful when identifying risk groups for targeted screening.

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1177. Carbapenemase-Producing Carbapenem-Resistant Organism Colonization Screening Surveys, Maryland, April 2017–April 2018

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Background. In April 2017, the Maryland Department of Health (MDH) began screening healthcare contacts of confirmed cases of carbapenemase-producing carbapenem-resistant organisms (CP-CROs) to identify potential transmission, per guidance published by the Centers for Disease Control and Prevention. The results of MDH's CP-CRO colonization screening surveys (CSSs) conducted as of April 1, 2018, are summarized.

Methods. Rectal swabs were collected on epidemiologically linked CP-CRO contacts and sent to the MDH Laboratories Administration, where the Cepheid Xpert® Carba-R assay was used to detect five carbapenemases: *Klebsiella pneumoniae* carbapenemase (KPC), New Delhi metallo-β-lactamase (NDM), Verona integron encoded metallo-β-lactamase (VIM), imipenemase (IMP), and oxacillinase-48-like carbapenemase (OXA-48). Identification of CP-CROs in contacts sometimes resulted in additional CSSs to ensure complete case detection. Non-KPC cases were combined for analysis.

Results. During April 1, 2017–April 1, 2018, MDH received reports of 278 incident cases of confirmed CP-CROs. Of these, 16 (6%) expressed non-KPC carbapenemases. The 7 (3%) cases with healthcare contacts prompting CSSs led to screening of 132 first-round contacts, with additional CP-CROs identified in 13 (10%), all of which had KPC. Of these, 12 (92%) resided in ventilator units of skilled nursing facilities (vSNFs). In the first-round CSS at one vSNF, 64% of screened contacts were positive for KPC, which had not been identified in the index case. Weekly follow-up CP-CRO admission screenings and serial follow-up CSSs at the vSNF resulted in screening of a total of 72 unique patients; 38 (53%) were KPC-positive. Of these 38 cases, 32 (89%) were previously unidentified and were placed on contact precautions if not already on them. Staff were re-trained in infection prevention (IP) techniques, and staff and KPC-positive patients were cohorted.

Conclusion. Detection of CP-CROs that express non-KPC carbapenemases in Maryland is rare, and transmission of these carbapenemases has not been identified. However, CSSs identified previously unknown cases of KPC, most commonly in vSNFs, demonstrating the utility of CSSs to detect CP-CROs, and resulting in important IP interventions.

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1178. A Multicenter Prospective Study of Clinical and Molecular Epidemiological Analysis of Carbapenem-Resistant *Enterobacteriaceae* (CRE) and Carbapenemase-Producing *Enterobacteriaceae* (CPE) in Japan

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Background. Data of a multicenter study on CRE from Japan are limited. Comparative analyses of carbapenemase-producing *Enterobacteriaceae* (CPE) and non-carbapenemase-producing CRE (NCP-CRE) have not yet been conducted.

Methods. Cases with CPE or CRE (defined as (1) meropenem [MPM] MIC, ≥2 mg/L or (2) imipenem [IPM] MIC, ≥2 mg/L and cefmetazole MIC, ≥64 mg/L [CLSI criteria]) were included from August 2016 to May 2017. PCR was used to detect carbapenemase.

Results. From five tertiary hospitals, 24 isolates (14 CPE and 10 NCP-CRE) were collected from 22 patients. Of the 10 NCP-CRE, seven were *Enterobacter aerogenes* and three were *Enterobacter cloacae*; of the 14 CPE, five were *Klebsiella pneumoniae*; 3, *E. cloacae*; 3, *E. coli*; 2, *Citrobacter freundii*; and 1, *E. aerogenes.* CPE were frequently isolated from the urine (5 [42%]) and sputum (3 [25%]) and NCP-CRE from sputum (4 [40%]), bile (3 [30%]), and urine (2 [20%]). Cases with CPE were older with more frequent use of urinary catheter and/or NG tube than

NCP-CRE (table). The 30-day mortality or length of hospital stay (LOS) did not differ between the two groups. Majority (n = 12) of CPE were identified to carry bla_{IMP} (MPM MIC, ≥2 mg/L), and two CPE were positive for $bla_{OXA-181}$ and $bla_{OXA-232}$ (MPM MIC, ≤1 mg/L). All NCP-CRE had IPM MIC of ≥2 mg/L; 7 (70%) had MPM of ≤1 mg/L. Resistance to amikacin (AMK) and levofloxacin (LFX) was noted in one and five CPE, respectively, whereas all NCP-CRE were sensitive, and nine bla_{IMP} and 1 $bla_{oxa-232}$ were transferable by conjugation.

Conclusion. CPE and NCP-CRE had different clinical characteristics. Non-β-lactam treatment options were more available for NCP-CRE than CPE. CPE and NCP-CRE might require different control strategies.

Table: Comparison of CPE and NCP-CRE, *n* (%)

	CPE (n = 12)	NCP-CRE (n = 10)	P-value
Age[a]	80 (74–93)	68 (63–73)	0.04
Male	5 (42)	8 (80)	0.1
Nursing home residence	4 (33)	0	0.1
Charlson Comorbidity Index[a]	3 (1–5)	2 (2–5)	0.92
Dependent functional status	9 (75)	3 (30)	0.08
Urinary catheter	9 (75)	2 (20)	0.03
NG tube	8 (67)	0	<0.01
Infection (not colonization)	3 (27)	3 (30)	>0.99
Polymicrobial isolation	7 (58)	9 (90)	0.16
Carbapenem exposure[b]	3 (25)	2 (20)	>0.99
Any antimicrobial exposure[b]	10 (83)	8 (80)	>0.99
30-day mortality	1 (10)	0	>0.99
LOS after isolation[a], days	31 (10–59)	22 (8–45)	0.39

[a]Median (IQR) and [b] ≤ 1 month.

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1179. Incidence of Bacteremia and Bacteriuria With Antibiotic-Resistant *Enterobacteriaceae* After Transrectal Ultrasound-Guided Biopsy of the Prostate (TRUSBP)

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Background. Infection with *Escherichia coli* after TRUSBP is common, but other *Enterobacteriaceae* also occur. In the absence of microbiological data, prophylaxis with co-trimoxazole (TMP-SMX) or fluoroquinolones (FQ) is usually prescribed. We estimated the incidence of bacteremia and bacteriuria after TRUSBP with distinct species of *Enterobacteriaceae* and their rate of resistance to common antibiotics.

Methods. Using Veterans Healthcare Administration (VHA) databases, we identified patients undergoing TRUSBP between January 1, 2013 and December 31, 2017. We determined the incidence of *Enterobacteriaceae* isolated from urine and blood cultures obtained within 30 days of TRUSBP. Using microbiology data from VHA, we determined rates of resistance to TMP-SMX, FQ (ciprofloxacin as marker), ESC (ceftriaxone as marker), and carbapenems (Carb) (ertapenem as marker).

Results. Overall, 377 (0.3%) and 1,739 (1.4%) of 126,761 TRUSBPs were complicated by bacteremia or bacteriuria with *Enterobacteriaceae*, respectively. *E. coli* was predominant (91% of blood and 81% in urine). Rates of FQ resistance were low in *Klebsiella*and *Enterobacter* but exceeded 60% in *E. coli*. In general, TMP-SMX resistance exceeded 30%. Of note, 16.6% of blood and 11% of urine *Enterobacteriacaea* were resistant to ESC, while Carb-resistance was rare.

Conclusion. FQ and ESC-resistant *Enterobacteriaceae* are prevalent in bacteremia and bacteriuria after TRUSBP. Antibiotics used for prophylaxis and empirical treatment are likely to be ineffective. The prevention and management of TRUSBP-related infections should include microbiology-guided approaches.

		Total	ESC-Resistant		Carb-Resistant		FQ Resistant		TMP-SMX Resistant	
Enterobacteriaceae	Blood	377	61	16.4%	2	0.5%	235	62.3%	129	34.2%
	Urine	1739	192	11.0%	20	1.2%	986	56.7%	607	34.9%
Citrobacter	Blood	3	1	%	0	–	1	–	1	–
	Urine	22	5	22.7%	2	9.1%	8	36.4%	7	31.8%
Enterobacter	Blood	4	0		0		0		0	
	Urine	46	8	17.4%	2	4.3%	3	6.5%	1	2.2%
Escherichia coli	Blood	344	57	16.6%	0	0.0%	232	67.4%	123	35.8%
	Urine	1415	157	11.1%	2	0.1%	934	66.0%	556	39.3%
Klebsiella	Blood	24	3	12.5%	1	4.2%	2	8.3%	4	16.7%
	Urine	196	14	7.1%	6	3.1%	15	7.7%	22	11.2%
Morganella	Blood	2	1		1		0		1	
	Urine	13	2	15.4%	5	38.5%	5	38.5%	8	61.5%
Proteus		33	6	18.2%	3	9.1%	17	51.5%	11	33.3%
Providencia		2	0		0	–	1	–	0	–
Serratia		11	0		0		2		1	
Shigella		1	0		0		1		1	

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1180. Addition of Chronic Kidney Disease Status to Pitt Bacteremia Score Improves Prediction of Mortality in Patients With Carbapenem-Resistant Enterobacteriaceae Infections

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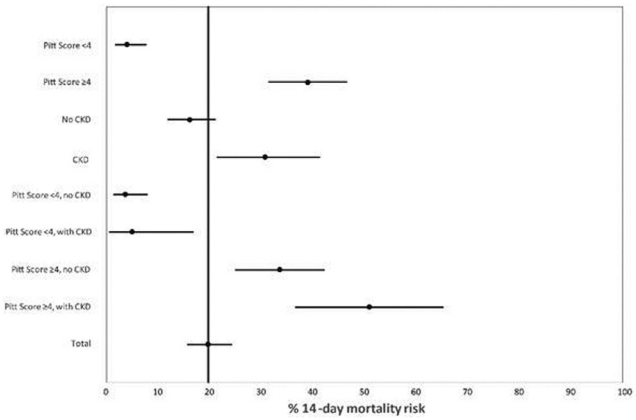
Background. Carbapenem-resistant Enterobacteriaceae (CRE) infections are associated with high mortality. The Pitt Bacteremia Score (PBS) was developed and validated to predict mortality in bloodstream infections (BSI). The first goal of this analysis is to evaluate whether PBS also predicts mortality in non-BSI infections. Second, we determine whether adding chronic kidney disease (CKD) as a parameter to PBS improves prediction of mortality.

Methods. The Consortium on resistance against carbapenems in *Klebsiella* and other Enterobacteriaceae (CRACKLE-1) is a prospective multicenter consortium of hospitals. Each patient with CRE infection was included once at the time of the last positive culture episode. Infections were distinguished from colonization using established definitions. Relative risk regression was used to evaluate the association of PBS ≥4 and CKD with 14-day all-cause hospital mortality.

Results. From December 2011 to June 2016, 364 unique patients were included with the following infections: bloodstream (34%), respiratory (20%), urinary (30%), and wound (16%). Median PBS was 3 (IQR: 2–4); 45% of patients had PBS ≥4. CKD was present in 31% of patients with PBS ≥4 and 20% of patients with PBS <4. All-cause mortality within 14 days of the last positive culture episode was 20%. In multivariable analysis, PBS ≥4 was strongly associated with mortality in patients with bacteremia (PBS ≥4 adjusted RR = 6.1, 95% CI 2.5–14.6, CKD aRR = 1.5, 95% CI 0.9–2.3) and in patients with other infections (PBS ≥4 aRR = 14.0, 95% CI 4.3–44.6, CKD aRR = 1.6, 95% CI 1.0–2.7). Adding CKD as a parameter to the PBS improved mortality prediction, specifically in patients with PBS ≥4 (figure).

Conclusion. As expected, PBS ≥4 was predictive of the 14-day risk of hospital mortality in this cohort of CRE bacteremic patients. In patients with other CRE infections, PBS ≥4 was also predictive of mortality. In this cohort, adding CKD to the PBS improved prediction of mortality patients with PBS ≥4.

Figure: Risks and 95% confidence intervals for 14-day all-cause hospital mortality, by Pitt bacteremia score and chronic kidney disease (CKD) status.



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