were collected from the Trauma Infectious Disease Outcomes Study (6/09–12/14). Additionally, a previously defined group of colonizing isolates linked to the infecting isolates and a selection of random colonizers were included from groin swabs. DNA extraction and PCR targeting Kv per published methods was performed. Antimicrobial susceptibilities were determined using the BD Phoenix Automated Microbiology System and CLSI criteria. Multidrug resistance was defined as either resistance to ≥2 classes of aminoglycosides, β-lactams, carbapenems and/or fluoroquinolones or production of ESBL or KPC.

Results. Of 237 archived Kp isolates (from 122 patients), 10 (4%) were identified as Kv by PCR (from 8 [7%] patients). The Kv were sources of 4 of blood (40%), 1 intra-abdominal (10%) and 5 from groin (50%). Six (3%) isolates were identified as Kq (4 from groin and 2 from respiratory specimens). The Kv and Kp patients were all males, with a median age of 25 (IQR 21–46) and 23 (IQR 21–28), and length of hospital stay of 24 days (IQR: 5–106) and 53 days (IQR: 36–74), and 30-day mortality was similar in both groups. While Kv was less resistant than Kp, it was more likely to be associated with invasive disease in this group.

Conclusion. Kv represented 4% of the previously identified Kp isolates in this population. Patient characteristics were similar in both groups. While Kv was less resistant than Kp, it was more likely to be associated with invasive disease in this group.

### Table: Antimicrobial susceptibilities

| Antibiotic | C. freundii (n=22) | K. pneumoniae (n=22) | P-value |
|------------|---------------------|----------------------|---------|
| Cefotaxime | 100%                | 100%                 | <0.01   |
| Ceftriaxone| 100%                | 100%                 | <0.01   |
| Cefepime  | 95%                 | 95%                  | <0.01   |
| Levofloxacin| 35%                | 62%                  | 0.09    |
| Piptazocin| 100%                | 50%                  | <0.01   |
| Meropenem | 100%                | 95%                  | 0.02    |
| Amikacin  | 100%                | 89%                  | 0.31    |

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### 504. Epidemiology of End-stage Renal Disease (ESRD) Patients with Carbapenem-Resistant Enterobacteriaceae (CRE) Infections: Atlanta Metropolitan Area, 2012–2017

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**Background.** Patients with end-stage renal disease (ESRD) may have higher risks for resistant organisms including carbapenem-resistant Enterobacteriaceae (CRE). To explore the effect of ESRD on CRE, we compared characteristics of CRE cases with and without ESRD in a population-based cohort.

**Methods.** The Georgia Emerging Infections Program has performed active laboratory- and population-based surveillance for CRE in metropolitan Atlanta (4.1 million in 2017) since 2012. CRE cases are defined by isolation from a sterile body site or urine of a single patient with ESRD, from 6/09–12/17. Each CRE isolate was in CPE with resistance to ≥2 classes of antibiotics and/or the ability to transfer resistance to susceptible strains. CRE were included from infected or colonized patients with ESRD in metropolitan Atlanta from 6/09–12/17. Demographic and clinical characteristics were compared using a multivariate/multivariable logistic regression model with adjustment for patient’s participation in other medical programs. Differences were considered significant at a 2-sided alpha of 0.05.

**Results.** From 6/09 to 12/17, 84% of CRE patients with ESRD were white and 24% were female. Patients with ESRD were more likely to be younger (mean age 52 years vs. 63 years, p<0.001). ESRD patients had more comorbidities as assessed by an estimate of Charlson Comorbidity Index (69 vs. 24, p<0.001). Patients with ESRD were more likely to have been exposed to fluoroquinolones, β-lactams, and third-generation cephalosporins (p<0.001). Patients with ESRD were more likely to have been exposed to KPC (4 vs. 0, p<0.001)

**Conclusion.** ESRD is associated with higher risk for CRE cases than non-ESRD cases, and more common resistance patterns in CRE cases with ESRD. This higher resistance and prevalence may be due to longer duration of care and multidrug exposure.

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### Table: Comparison of Cases with CPE and NCPE, n (%)

| Age* | CPE (n = 25) | NCPE (n = 63) | P-value |
|------|-------------|--------------|---------|
| Nursing home residence | 6 (24) | 4 (6.5) | 0.03 |
| Charlson Comorbidity Index* | 3 (2–5) | 2 (1–3) | 0.12 |
| Dependent functional status | 19 (76) | 24 (38.1) | <0.01 |
| Urinary catheter | 17 (68) | 23 (36.5) | 0.01 |
| Nasogastric tube | 11 (44) | 7 (11.1) | <0.01 |
| Carbenem exposure ≤3 months | 10 (40) | 9 (14.3) | 0.02 |
| Bacteremia | 7 (16) | 3 (4.8) | 0.1 |

### Table: Comparison of Cases with CPE and NCPE, n (%)

| Mortality | CPE | NCPE | P-value (bivariate/PS-adjusted analyses) |
|-----------|-----|------|----------------------------------------|
| In-hospital | 4 (16) | 5 (8.1) | 0.27/0.82 |
| 30-day | 3 (12.5) | 3 (5.1) | 0.35/0.52 |
| LOS after isolation* | 44 (18-71) | 29 (11-43) | 0.11/0.02 |

*Median (IQR).

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System. Georgia vital records data were used to determine 90-day mortality rates. Prevalence estimates were calculated. Comparisons used a χ² test.

**Results.** Of 1,511 CRE cases, 136 (9%) were on current chronic dialysis, 128 (94%) of which were on hemodialysis (HD) and 5 (4%) were on peritoneal dialysis. Among CRE cases with HD, 94 (73%) had a catheter and 30 (23%) had an arteriovenous fistula or graft. Sixty-four (48%) of these cases with HD or graft had CRE. The remaining CRE cases with HD or graft were more likely to have been hospitalized >3 days before the culture compared with CRE cases with ESRD and positive cultures from other body sites (52% vs. 24%). The 90-day mortality rate per 100,000 population was higher among CRE cases with ESRD (100.9 cases) than without ESRD (1.0 cases). Twenty-five patients (5%) progressed from CRE bacteriuria to an invasive CRE infection within one year (median 34 days). Predictors of progression in univariable analyses included the presence of a urinary catheter (OR 6.4 [95% CI: 1.9–21.6]), decubitus ulcer, CVC or other indwelling device, Klebsiella pneumoniae, black race, CGI >3, and ICU stay after urine culture was obtained (Table 2). In a multivariable analysis, urinary catheter (OR 4.6 [95% CI: 1.3–16.1]) predicted progression as well as K. pneumoniae, CGI >3 and CVC.

**Conclusion.** Progression from CRE bacteriuria to an invasive CRE infection is rare but clinically significant and is associated with urinary catheters. Future interventions should target bacterial removal, where possible, in patients with CRE bacteriuria.

### Table 1: Demographics of patients with CRE bacteria in metropolitan Atlanta stratified by the presence of a urinary catheter

| Age (mean years, SD) | No urinary catheter (n = 227) | Urinary catheter (n = 280) | P-value |
|----------------------|------------------------------|---------------------------|---------|
| Male                  |                             |                           |         |
| Female               |                             |                           |         |
| Race                  |                             |                           |         |
| White                 |                             |                           |         |
| Black                 |                             |                           |         |
| Charlson comorbidity index >3 (n = 504) | 191 (38) | 87 (96) | 0.038 |

### Table 2: Risk factors for progression to an invasive CRE infection

| Risk Factor                        | No progression (n = 482) | Progression Univariable (OR 95% CI) | Multivariable OR (95% CI) |
|------------------------------------|--------------------------|-------------------------------------|--------------------------|
| Age (mean years, SD)               | 63.6 (17.6)              | 3.5 (1.8–6.5)                       | 3.0 (1.6–5.2)            |
| Female                             | 279 (56)                 | 0.0 (0.4–1.9)                       | 0.3 (0.1–1.7)            |
| Black                              | 280 (63)                 | 3.0 (1.0–9.8)                       | 3.3 (1.1–9.8)            |
| Charlson comorbidity index >3 (n = 504) | 178 (36) | 11 (60) | 2.6 (1.4–4.6) | 3.0 (1.3–7.1) |
| Diabetes mellitus                   | 105 (14)                 | 2.6 (1.2–5.6)                       | 3.0 (1.3–7.1)            |
| Urinary catheter                    | 239 (50)                 | 4.6 (2.3–9.1)                       | 4.6 (2.3–9.1)            |
| Other indwelling device             | 161 (33)                 | 3.6 (1.5–8.2)                       | 3.6 (1.5–8.2)            |
| Organism K. pneumonia               | 301 (64)                 | 13.3 (8.9–21.7)                     | 9.7 (3.2–37.3)           |
| E. cloacae                          | 43 (9)                   | 0.0 (0.0–1.3)                       | 0.0 (0.0–1.3)            |
| E. aerogenes                        | 17 (4)                   | 0.0 (0.0–1.1)                       | 0.0 (0.0–1.1)            |
| E. asuka                           | 11 (2)                   | 0.0 (0.0–1.2)                       | 0.0 (0.0–1.2)            |

### Background.** Carbenapenem-producing Enterobacteriaceae can form a reservoir in hospital wastewater biofilms. Klebsiella quasipneumoniae is increasingly recognized as an emerging nosocomial threat, frequently carrying antimicrobial resistance (AMR) genes on plasmids. The dynamics of AMR gene and plasmid gain/loss over time in this species remain unclear.

### Methods.** Klebsiella pneumoniae carbenapenem producing KPC-Kq isolates from patients and wastewater sites from drains and toilets were sequenced (Illumina). Sequence assemblies (SPAdes) were probed in silico for AMR genes and plasmid Incompatibility types (using AMRFinder and PlasmidFinder databases, respectively). For related isolates (<100 SNV) cultured from the same sites longitudinally, we compared the accumulation of AMR genes in patients and environmental reservoirs over time.

### Results.** From 2009 to 2016 there were a total of 15 KPC-Kq isolates from 8 patients and 17 environmental isolates from 11 rooms. The mean number of resistance genes identified in patients and environmental isolates were 15 and 14, respectively (P = NS), with five resistance genes carried by all isolates including blac via. There was an average of 4.4 unique incompatibility types from patients and 4.0 from the environment (P = NS). For the longitudinal subset, there were 17 related isolates from two patients and two sink drains. One hospitalized patient with repeated antimicrobial exposure had a KPC-Kq initial isolate with 3 plasmid types and 13 AMR genes and died one year later with a KPC-Kq isolated from blood with 11 plasmid types and 25 AMR genes. The other patient was primarily an outpatient with little antimicrobial exposure. His KPC-Kq lost 1 plasmid and 3 AMR genes over 15 months. One KPC-Kq strain in the environment lost 3 plasmid types and 8 AMR genes over 4 months; the other was unchanged over 5 months.

### Conclusion.** KPC-Kq has been seen in both patients and the environment for several years at our institution. Sequencing of longitudinal isolates revealed that under antimicrobial pressure a patient KPC-Kq accumulated multiple plasmids and AMR genes. This same accumulation was not witnessed environmental isolates although the numbers are small and will require confirmatory work.

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