and 44.3 hours by the conventional method. Patients with BF-FA-BCIP received the optimal therapy after a median of 25.5 hours (95%CI 21.0 - 31.2) as compared to 45.7 hours (95%CI 37.7 - 51.2) in the control group (Figure 1). We found no effect of the identification method on secondary outcomes.

Kaplan-Meier curve representing the probability of implementing the optimal therapy at any given time according to the identification method (Standard vs. BF-FA-BCIP).

Results. From 103 pts grew 114 bacterial sp: E (n=54; 16 ESBL, 1 KPC-producer), S. aureus (n=29, 22 MRSA), Enterococcus (n=21, 16 VRE), P. aeruginosa and others (n=10), 12 ESBL-E produced CTX-M 14/15. T2R sensitivity and specificity was 78% and 99%, respectively, compared to sequencing of resistance markers. Sensitivity was excellent for vanA/B, KPC (100% each), and CTX-M14/15 (92%); specificity was 58% for mecA/C. T2R detected resistance determinants in 3-7h. Median time to appropriate Ab was 16.8h, which was significantly longer for VRE (25.6h) and ESBL- or KPC-E (50.9h) BSIs than for T2R marker-negative bacteria (6.7h; p=0.04). Pts with VRE or ESBL/KPC-E BSIs were less likely to receive appropriate empiric Ab (18% and 30%, respectively) than pts with T2R marker-negative BSI (63%; p=0.02; Fig.1). Median times to achieve ≥80% appropriate Ab therapy of marker-negative VRE and CTX-M/KPC-E BSIs were 15.8h (after Gram stain), 43.9h (after MALDI) and 63.5h (after sensi), respectively.

Conclusion. There was a significant delay in appropriate Ab therapy of BSIs, especially in pts infected with VRE and ESBL/KPC-E. T2R rapidly and accurately detected BSI caused by VRE and ESBL/KPC-E, and has the potential to significantly shorten time to appropriate Ab.

Disclosures. Corneliuss J. Clancy, MD, Merck (Grant/Research Support) Ryan K. Shields, PharmD, MS, Shionogi (Consultant, Research Grant or Support) Minh-Hong Nguyen, MD, Merck (Grant/Research Support)
Total number of tests and proportion of IGRA:TST obtained by month, from October 2015-January 2021.

Conclusion. While most TB infection tests in this age group were TSTs, the monthly proportion of tests that were IGRA increased over time between 2015-2021. IGRA were obtained in varied clinical settings. In this low burden setting, rates of invalid/ineterminate IGRA were low among children < 2 years old, which suggests that IGRA are reasonable TB testing options for patients < 2 years old, and may be preferred given limitations of TSTs.

Disclosures. Gabriella S. Lamb, MD, MPH, Nothing to disclose

656. Sulbactam-Durlobactam MIC Determination: Comparative Evaluation of the New ETEST SUD to the CLSI 2021 Broth Microdilution Method

Background. Species belonging to the 

Klebsiella pneumoniae species complex were excluded.

Methods. We analyzed 112 isolates, including 19 hvKp, 67 cKp, and 26 colonizers, by whole-genome sequencing. Population genomics revealed that the K1 sequence type (ST) 82 clade was distinct from that of K1-ST23 clones (Figure 1). The virulence gene profiles also differed between K1-ST82 (aerobactin and rmpA) and K1-ST23 (aerobactin, yersiniabactin, salmochelin, colibactin, and rmpA/rmpA2). The K2 genotype was more diverse than that of K1. A neighboring subclade of K1-ST23 (comprising ST29, ST412, ST36, and ST268) showed multidrug-resistance and hypervirulence potentials. Logistic-regression analysis revealed that diabetes mellitus was associated with K. pneumoniae infection (odds ratio [OR]: 4.11; 95% confidence interval [CI]: 1.14–14.8). No significant association was found between hvKp diagnosis and clinical characteristics, such as diabetes mellitus or community acquisition (Table 1). The K1 genotypes (OR: 9.02; 95% CI: 2.49–32.7; positive-likelihood ratio [LR]: 4.08), rmpA (OR: 8.26; 95% CI: 1.77–38.5; positive LR: 5.83), and aerobactin (OR: 4.59; 95% CI: 1.22–17.2; positive LR: 3.49) were substantial diagnostic predictors of hvKp (Table 2).

Figure 1. Phylogenetic distribution of genetic virulence factors in 112 K. pneumoniae isolates

The highlighted strains are clinically pathogenic (orange, hypervirulent K. pneumoniae; yellow, classical K. pneumoniae; sky blue, colonization). The non-highlighted strain (NTUH-K2044) is a reference K. pneumoniae strain.

Table 1. Variables analyzed for predicting hvKp infection

| Variables               | OR (95% CI) | p value |
|-------------------------|-------------|---------|
| Diabetes mellitus       | 1.49 (0.52–4.23) | 0.46 |
| Liver cirrhosis         | 12.4 (2.11–127)  | 0.034 |
| Community-acquired      | 1.34 (0.48–3.73)  | 0.58 |
| Positive string test    | 4.07 (1.08–15.3) | 0.038 |
| K1                      | 9.02 (2.49–32.7)  | 0.001 |
| K2                      | 0.94 (0.32–2.82)  | 0.92 |
| Aerobactin              | 4.59 (1.22–17.2)  | 0.024 |
| Yersiniabactin          | 2.11 (0.74–6.04)  | 0.16 |
| Salmochelin             | 2.56 (0.83–7.91)  | 0.11 |
| Colibactin              | 1.86 (0.63–5.52)  | 0.26 |
| rmpA                    | 8.20 (1.77–38.5)  | 0.007 |
| rmpA2                   | 1.26 (0.44–3.37)  | 0.71 |

hvKp, hypervirulent K. pneumoniae; OR, odds ratio; CI, confidence interval

Table 2. Microbiological diagnostic predictive values for hvKp

| Characteristics | Sensitivity (%) | Specificity (%) | PPV (%) | NPV (%) | LR+ | LR- |
|-----------------|-----------------|-----------------|---------|---------|-----|-----|
| Positive string test | 84.2 | 43.3 | 29.6 | 90.6 | 1.49 | 0.37 |
| K1 genotype     | 61.5 | 84.9 | 42.1 | 92.5 | 4.08 | 0.45 |
| Aerobactin      | 30.8 | 91.2 | 84.2 | 46.3 | 3.49 | 0.76 |
| rmpA            | 33.3 | 94.3 | 89.5 | 49.3 | 5.83 | 0.71 |

hvKp, hypervirulent K. pneumoniae; PPV, positive predictive value; NPV, negative predictive value; LR, likelihood ratio

The highlighted strains are clinically pathogenic (orange, hypervirulent K. pneumoniae; yellow, classical K. pneumoniae; sky blue, colonization). The non-highlighted strain (NTUH-K2044) is a reference K. pneumoniae strain.

Session: P-29. Diagnostics: Bacteriology/mycrobacteriology

Background. Japan is one of the hypervirulent Klebsiella pneumoniae (hvKp) endemic areas, resulting in an alarming issue in actual clinical settings. However, little is known regarding key virulence factors responsible for hvKp infection.

Methods. We analyzed K. pneumoniae isolates collected between 2017 and 2019, and defined hvKp as a pyrogenic infection. Classical K. pneumoniae (cKp) involved a non-invasive infection or uncomplicated bacteremia. Isolates belonging to the K. pneumoniae species complex were excluded.

Results. We analyzed 112 isolates, including 19 hvKp, 67 cKp, and 26 colonizers, by whole-genome sequencing. Population genomics revealed that the K1 sequence type (ST) 82 clade was distinct from that of K1-ST23 clones (Figure 1). The virulence gene profiles also differed between K1-ST82 (aerobactin and rmpA) and K1-ST23 (aerobactin, yersiniabactin, salmochelin, colibactin, and rmpA/rmpA2). The K2 genotype was more diverse than that of K1. A neighboring subclade of K1-ST23 (comprising ST29, ST412, ST36, and ST268) showed multidrug-resistance and hypervirulence potentials. Logistic-regression analysis revealed that diabetes mellitus was associated with K. pneumoniae infection (odds ratio [OR]: 4.11; 95% confidence interval [CI]: 1.14–14.8). No significant association was found between hvKp diagnosis and clinical characteristics, such as diabetes mellitus or community acquisition (Table 1). The K1 genotypes (OR: 9.02; 95% CI: 2.49–32.7; positive-likelihood ratio [LR]: 4.08), rmpA (OR: 8.26; 95% CI: 1.77–38.5; positive LR: 5.83), and aerobactin (OR: 4.59; 95% CI: 1.22–17.2; positive LR: 3.49) were substantial diagnostic predictors of hvKp (Table 2).

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hvKp, hypervirulent K. pneumoniae; OR, odds ratio; CI, confidence interval

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hvKp, hypervirulent K. pneumoniae; PPV, positive predictive value; NPV, negative predictive value; LR, likelihood ratio