

**P1450 Population genomics of *Escherichia coli* bacteraemia in the Netherlands (2014 - 2016)**

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**Background:** Previous studies have suggested evolutionary specialization of a subset of commensal *E. coli* strains, as reflected by the identification of clones that are frequently found in invasive infections. Possibly, there are molecular patterns of sub specialization within invasive strains as well. We aimed to analyse the current population genomics of *E. coli* bacteremia (ECB) in the Netherlands, with special attention to genomic differences between strains with different phenotypic and epidemiological characteristics.

**Materials/methods:** Patients with a blood culture growing *E. coli* between January 2014 and December 2016 were retrospectively identified in two participating hospitals. For each year, a random selection of approximately one-fourth of all first available isolates was drawn. In addition to the random sample, all ESBL-producing ECB isolates from 2014 to 2016 were included. Whole genome sequencing with Illumina HiSeq was performed on all isolates.

**Results:** The random selection resulted in 235 ECB isolates, of which 23 (9.8%) were ESBL-positive. The median age among selected patients was 69 (IQR 59-76), 112 (47.7%) patients were female, 188 (80%) ECB were of community onset and the most frequent primary focus was the urinary tract (115, 48.9%). The predominant sequence types were ST131 (n=34, 14.5%), ST73 (n=26, 11.1%) and ST69 (n=19, 8.1%). In addition to the random selection, another 47 ESBL-producing ECB isolates were included. Among the total group of 70 ESBL-producing *E. coli* isolates, 31 (44.3%) belonged to ST131, as opposed to 22 (10.4%) of all ESBL-negative (n=212) isolates (p-value <0.001). MLST distribution differed between ESBL-groups, but within each group there was no difference between community and nosocomial onset bacteremia. There was no distinct clustering of isolates with different primary foci based on MLST and core genome phylogeny.

**Conclusions:** The majority of ECB were of community onset and 9.8% was caused by ESBL-producing isolates. Within the group of ESBL-producers, ST131 was more prevalent than within ESBL-negative strains. We did not observe distinct phylogenetic clustering of ECB isolates with nosocomial as compared to community onset, suggesting that they share an evolutionary history. The same is true when comparing ECB isolates with different primary foci of infection.