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O0356 Shiga toxin-producing Escherichia coli (STEC) from Swedish patients: prevalence and molecular characteristics in correlation to clinical symptoms and duration of STEC shedding

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Background: Shiga toxin−producing *Escherichia coli* (STEC) are causative pathogens of diseases ranging from asymptomatic carriage to bloody diarrhea (BD), hemorrhagic colitis (HC), and hemolytic uremic syndrome (HUS). STEC are widely spread and associated with gastrointestinal symptoms and HUS in Europe. Sweden showed high STEC prevalence rate with the largest outbreak occurred in 2005. Here a 12-years period STEC investigation in patients with diarrhea was undertaken in Region Jönköping County, Sweden to reveal the prevalence and molecular characteristics of STEC in patients, and demonstrate the correlation of molecular factors to clinical symptoms and duration of STEC shedding.

Materials/methods: Diarrheal stool samples were screened for *stx* by real-time PCR. PCR positive specimens were inoculated onto selective plates for STEC isolation. Serogenotypes, *stx* subtypes, virulence spectrums, and antimicrobial resistance genes contents of each isolate were characterized by DNA microarray and whole genome sequencing analysis. Comparative genomic analysis was undertaken to assess phylogenetic relationships and find unique genetic markers to predict high pathogenicity and long duration of *stx* shedding.

Results: Specimens from 26382 patients were analyzed. *stx* was positive in 391 patients (1.5%), including 13 HUS cases. 171 isolates were recovered, among which 8 were from HUS cases. The most prevalent serotypes were O157:H7, O26:H11, O121:H19 and O103:H2. Isolates from HUS cases were assigned as O157:H7, O121:H19, O104:H4, and O98: H21 serotypes, most of HUS isolates harbored *stx2a* subtype, while one isolate carried *stx1a* subtype. Notably, two O104:H4 isolates from Swedish HUS cases which were part of the 2011 German outbreak, exhibited same virulence markers and sequences type with the outbreak strain. The correlation of virulence factors with clinical symptoms and *stx* shedding were observed, including genes encoding toxins, adhesions, and secrection factors. Phylogenetic analysis reveals that our strains were highly diverse, and a number of strains show close relatedness to HUS-associated EHEC collection.

Conclusions: Here, we systematically investigated STEC prevalence and molecular features in Swedish patients on a large scale. Our study reveals molecular traits of high virulent human STECs, and find genetic markers that can be used for risk assessment and prediction for severe clinical symptoms and long duration of *stx* shedding.