O166

1-hour Oral Session

Now is the right time to go very deep: recent advances in molecular typing

Global spread of *Clostridium difficile* type 078 and its close relatives between animals and humans

W. Knetsch[1], E. Kuijper[1], T. Lawley[2], -. International studygroup[3]

[1]*Leiden University Medical Center, Leiden, Netherlands*

[2]*Wellcome Trust Sanger Institute, Hinxton, United Kingdom*

[3]*Multiple, -, Netherlands*

Objectives

Clostridium difficile is the main cause of antibiotic associated infection in the developed world. The incidence of *C. difficile* infection (CDI) associated with *C. difficile* PCR ribotype 078 is increasing within the European healthcare system. The causes of the increase in human CDI remain poorly understood. Farm animals are considered as a potential reservoir for human CDI. Here, we applied whole genome phylogenetic single-nucleotide polymorphism (SNP) analysis to compare type 078 isolates and its close relatives (types 033, 045 and 126) collected from diverse sources (human, animal, environment and food) and geographical locations (Europe, North-America, Asia and Australia).

Methods

In total, 325 *C. difficile* isolates were sequenced and SNP typed including type 078/126 isolates (n= 280), type 033 (n= 16) and type 045 (n= 13), supplemented with various other *C. difficile* types (n= 16). Whole genome bacterial re-sequencing was done on extracted genomic DNA using Illumina Hiseq. The short DNA sequence reads were bioinformatically mapped against an reference genome for type 078 (strain M120), after which SNPs were called. Genomes were scanned to identify potential antimicrobial resistance determinants.

Results

The tree topology revealed four clusters for types 033, 045, 066/127 and 078/126 and a more diverse mixed cluster of various C. difficile types. In total 25.641 SNPs were identified, of which 22.223 SNPs belonged to a small group of eight diverse *C. difficile* isolates. Three clusters for types 033, 045 and 078/126 share the same observation that human and animal isolates are mingled, no separate clusters were found with either human or animal isolates solely. Also, we observed in several cases that human and animal isolates had identical SNP genotypes, *i.e.* zero SNP differences. Furthermore, we found identical antimicrobial resistance determinants for tetracycline (tet40, tetO, tet44 en tetM) and streptomycin (Aph3-III, Ant6-Ia, Sat4A and Ant6-Ib) present in human and animal *C. difficile* isolates.

Conclusion

Our results demonstrate that *C. difficile* is capable of spreading between animals and humans although we may not exclude an unidentified common (environmental) source.