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Paper Poster Session
Diagnosing gastrointestinal tract infection

Toxigenicity of Clostridium difficile strains isolated from GDH-positive faecal samples and epidemiological relevance for the management of CDI patients

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Background: Recent studies suggest that *C.difficile* carriers may be an important reservoir in both healthcare settings and in the community. Since the introduction of multi-step testing algorithms, increasing attention has been given to patients testing positive for the presence of *C.difficile* antigen GDH but negative for toxin EIA (TOX), so-called potential *C. difficile* 'excretors',as an important source of transmission of *Clostridium difficile* infection (CDI). We aimed to investigate the proportion of these individuals in relation to confirmed CDI cases, the molecular profile of the strains and match with epidemiological information from a consecutive group of inpatients.

Material/Methods: For a two year period between May 2012 and April 2014 over 4,000 faecal samples were verified with a combined GDH and toxin EIA (TOX) test (Allere/Techlab) as part of a two-step algorithm for CDI testing. All GDH+ samples were cultured, regardless of the TOX result. Isolates were subject to tests for the presence of toxin genes by multiplex PCR and profiling by PCR-ribotyping.

Results: A total of 357 GDH+/TOX- and 240 GDH+/TOX+ samples were cultured, yielding 298 and 233 isolates, respectively. Of the 298 GDH+/TOX- isolates, 193 (65%) were found to carry genes for toxin A and/or B, whilst 105 (35%) were deemed non-toxigenic. Among the 193 toxigenic isolates, PCR-ribotype 014 (18%) was the most frequently identified, followed by RT002 (12%), RT015 (11%), RT020 (8%), RT005 (7%) and RT078 (5%). This distribution was similar to that of isolates cultured from CDI patients who were found to be TOX+, except for an increased incidence of RT027 in the latter.

Conclusions: While 35% of faecal samples testing GDH+/TOX- in our setting yielded a non-toxigenic isolate and were considered true carriers, the majority of samples contained an isolate possessing toxin genes that could potentially cause CDI. Whilst the GDH test is believed to display higher sensitive than the toxin test, the differentiation of dysbiotic patients carrying the organism from emerging CDI cases remains an area of intense debate. The potential contribution of GDH+/TOX- individuals as a reservoir for nosocomial transmission of CDI cannot be neglected and our results support the incorporation of a confirmatory nucleic acid amplification test (NAAT) for real-time detection of toxin genes into current diagnostic algorithms. GDH+/TOX- patients are believed to display spontaneous recovery from diarrhoea and fewer complications, and to confirm this additional work is currently being conducted in relation to primary clinical outcomes, such as Recurrence and Death.