Inflammation Bowel Disease II

PTH-025 ★ **VIRULENCE FACTORS, ANTIMICROBIAL RESISTANCE GENES AND PROTEIN STRUCTURE OF CROHN'S DISEASE-DERIVED *E COLI* STRAINS IDENTIFIED USING DNA MICROARRAY AND MALDI-TOF ANALYSIS**

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Introduction Intracellular *Escherichia coli* isolates are found in up to 60% of gut biopsies from patients with Crohn's disease (CD). Although adherent invasive *E coli* (AIEC) are described, other intracellular *E coli* are frequently isolated but specific pathogenic determinants for these have not been identified by conventional techniques. DNA microarray and Matrix-assisted laser desorption/ionisation time of flight (MALDI-TOF) mass spectrometry enable rapid and accurate DNA and proteomic analysis of bacteria.

Aims To investigate for virulence and antimicrobial resistance (AMR) genes in, and analyse the structure of CD-related intracellular *E coli* isolates.

Methods 17 *E coli* strains were isolated using gentamicin protection assay from gut biopsies of 17 CD patients (ileal n=7, colonic n=10). DNA was extracted for analysis by DNA microarray (Prodigy) of 392 virulence and AMR genes. DNA PCR was performed for an additional 18 virulence genes. For Bruker Biotyper MALDI-TOF sample preparation, several colonies of each bacterial strain were spotted onto target plate and prepared as described previously. 10 replicates of the mass/charge spectra for each strain were compared to spectra from a bacterial database.

Results Virulence factors were identified by DNA microarray in a number of *E coli* strains including senB (encodes enterotoxin), Iha (encodes an adhesin) Sat (toxin of uropathogenic *E coli*), iron regulation genes and microcin synthesis genes. DNA PCR revealed additional virulence factors including ibeA (associated with *E coli* invasion in neonatal meningitis) in 2 strains. A minority of strains were positive for AMR genes relating to β-lactams, sulphonamides, tetracycline and streptomycin. MALDI-TOF analysis confirmed the bacteria as *E coli* and clustered the Crohn's disease *E coli* strains together and separate from other *E coli* strains on the Bruker database. There was no clustering within CD strains in relation to disease or biopsy site or immunomodulation.

Conclusion Although CD appears to be at least in part a host immune disorder, pathogenic factors of various organisms may still play a role in pathogenesis. A number of genes encoding for virulence factors have been identified in a proportion of CD-related *E coli* strains which warrant further

investigation (eg, whole gene sequencing and functional assays). Most strains had no antimicrobial resistance genes. Further MALDI-TOF comparison of CD-related bacterial structure with commensal and IBD-related bacteria may be revealing.

Competing interests None.