**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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T Elliott,1,2,* B Hudspith,2 N Rayment,2 L Randall,3 G Wu,3 A Boussioutas,4 J Hermon-Taylor,2 J Brostoff,2 L Petrovska,5 J D Sanderson1,2 1Gastroenterology, Guy’s and St Thomas’ Hospital, London, UK; 2Nutritional Sciences Division, King’s College London, London, UK; 3Veterinary Laboratories Agency, Surrey, UK; 4Department of Medicine, University of Melbourne, Melbourne, Victoria, Australia; 5Veterinary Laboratories Agency, London, UK

**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 \( \beta \)-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.