have been shown to play a key role, these observations suggest a distinct contribution of Nod1 and Nod2 in mucosal homeostasis following the cellular uptake of muramyl peptides by HepT1.

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THE PRESENCE OF ESCHERICHIA COLI BACTERIOPHAGES IN HUMAN STOOL OF PATIENTS WITH INFLAMMATORY BOWEL DISEASE
M. Lusiak-Szelachowska1, B. Weber-Dabrowska1, A. Gorski1, A. Annabhani2, K. Blachut2, L. Paradowski1, Institute of Immunology and Experimental Therapy, Polish Academy of Sciences, Center of Excellence, Wroclaw; 2Department of Gastroenterology and Hepatology, Wroclaw Medical University

Introduction: Bacteriophages are bacteria-specific viruses that attack bacteria and, after multiplication, lyse the host cell. Escherichia coli bacteriophages are widely distributed throughout the environment as well as in many human individuals and animals. Endogenous bacteriophages may play a valuable role in protecting against microbes.

Aim: The aim of our study was to investigate the presence of coliphages in the human intestine and to compare their frequency in healthy individuals and patients with inflammatory bowel disease (IBD) (Crohn’s disease and ulcerative colitis) in a Polish population.

Material and method: Stool samples were taken from 70 volunteers and 165 patients with IBD (48 with Crohn’s disease and 117 with ulcerative colitis). Coliphages were detected in human feces using the indicator bacteria E. coli B and E. coli 1962 from the Institute’s collection. E. coli-specific phages were examined by the double-agar-layer method after 24 hours of incubation at 37 °C of the stool sample with the bacterial host culture. The activity of phages was determined in immediate materials.

Results: Coliphages for both E. coli B and E. coli 1962 strains were detected in 30% of the stools of volunteers (mean concentration: 2.7 × 10^5 PFU/g) and 34.1% of the patients with Crohn’s disease (mean concentration: 2.9 × 10^5 PFU/g) and 28.7% of samples from patients with ulcerative colitis (mean concentration: 3.6 × 10^5 PFU/g).

Conclusion: Our results suggest that the frequency of samples positive for E. coli bacteriophages is higher in those from volunteers than in those from patients with IBD. The concentrations of coliphages in patients’ feces were higher compared with healthy individuals. The results suggest that there may be associations between phage presence in the human gastrointestinal tract and its disorders.

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CYTOMEGALOVIRUS BLOCKS APOPTOSIS IN PATIENTS WITH ACTIVE ULCERATIVE COLITIS AND COLONIC VIRAL INFECTION
R. Vega1, H. Kesheh1, E. Domenech2, I. Ojanguren2, M.A. Gassull2, A. Forbes1, R. Day1. 1University College Hospital; 2Hospital Universitari Germans Trias i Pujol

Introduction: The immunological response of the colonic mucosa to cytomegalovirus (CMV) infection in patients with active ulcerative colitis (UC) remains unknown. TNF-related apoptosis-inducing ligand (TRAIL) mediated apoptosis is increased in patients with inflammatory bowel disease. Human CMV is known to block apoptosis in different types of cells despite an up-regulation of TRAIL and TRAIL membrane receptors.

Aim: To examine the apoptosis rates in active UC patients with and without CMV infection; and to investigate the expression of TRAIL and TRAIL R1 and R2 in the colonic tissue of these patients.

Materials and methods: Paraffin embedded colonic tissue specimens were obtained from 8 non-steroid resistant active UC patients, 8 steroid-resistant active UC, and 6 steroid-resistant active UC with colonic infection by CMV, which were included in a clinical prospective study to investigate the prevalence of CMV in UC. Apoptosis of epithelial and lamina propria cells was assessed by immunohistochemical staining for cleaved caspase-3. Immunohistochemical staining was also performed for TRAIL, TRAIL-R1 and R2.

Results: There was no significant difference in disease activity index, CRP and ESR among the patient groups when the biopsies were obtained. Histology assessment showed moderate to severe colonic inflammation. Colonic CMV infection in the samples was confirmed by immunohistochemical staining for CMV. TRAIL was strongly expressed in the epithelial cells in all samples, especially in the upper 1/3 area of the crypts. The expression of TRAIL-R2 in lamina propria was higher in infected patients compared to the patients without viral infection regardless to steroid response. There were no differences in TRAIL-R1 expression among the different groups. Apoptotic indices (proportion of casapse-3 positive cells from the total of cells counted) in intestinal epithelial cells were significantly lower (0.27±0.15) in patients with CMV infection compared to those with steroid-resistant patients (0.53±0.13) and non-steroid-resistant patients (0.66±0.22) p<0.05. No significant differences in the apoptosis indices were seen in lamina propria cells among the different groups.

Conclusions: Apoptosis in colonic epithelial cells decreases in CMV infected patients despite the presence of TRAIL and the expression of TRAIL-R2. This may be explained by the capacity of the human CMV proteins, such as viral inhibitor of caspases-8-induced apoptosis (vICA) and viral mitochondrial inhibitor of apoptosis (vMIA), which directly interfere with the apoptotic signalling pathway in infected tissues. CMV may contribute to steroid resistance in UC infected patients.

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IS ESCHERICHIA COLI MORE THAN A COMMENSAL BACTERIUM IN THE INTESTINAL MUCOSA OF CROHN’S DISEASE PATIENTS?
M. Martinez-Medina1, X. Aldegue2, F. Gonzalez-Huix2, D. Acero2, M. Lopez-Siles1, L.J. Garcia-Gil1, University of Girona; 2Hospital Josep Trueta

The role of bacteria in Crohn’s disease (CD) has received special attention in the last few years. In particular, Escherichia coli (EC) has been suggested to play a role in the onset of the inflammatory response [1-4] and has recently been shown to be more prevalent in CD patients [5] in this work EC strains isolated from CD patients have been analyzed and compared with those from healthy controls in order to identify and characterize the pathogenic strains.

A total of 1600 presumptive EC strains from 8 CD patients, 10 healthy controls and 2 patients suffering from ulcerative colitis, were isolated. Transient and loosely attached bacteria were discarded by mild sonication. A mild osmotic shock was performed to release intracellular bacteria. EC was isolated on TBX agar medium and further confirmed by indole assay. The identity of isolates was checked by PFGE using two enzymes (XbaI and SpeI). The presence of up to 18 virulence genes (cnf1, cnf2, hly, lapG, sfa/foc, afa, bfp, iuc, sfa/foc, et alia and est) was tested by PCR. A total of 1600 presumptive EC from 8 CD patients, 10 healthy controls and 2 patients suffering from ulcerative colitis, were isolated. After analyzing the clonality of the isolates, a total of 31 different EC subtypes were obtained. On average, 1.93 ± 0.96 different strains per individual were found, with no significant differences between CD patients and controls. Except for 5 subtypes, which were found in two patients each, every EC clone were unique to each human carrier, which supports the idea that the host-microbe relationship in the gut ecosystem is controlled by complex factors. We have not found genetic relatedness among EC from CD patients in terms of presence of certain known virulence genes, neither by their PFGE profile. Our results so far indicate that there is not a common strain inhabiting the colonic mucosa of CD patients, as previously suggested. Nevertheless, the pathogenic features of the isolates are currently under study, since the possibility of certain common features of EC from CD patients in terms of pathogenicity cannot be ruled out.


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GALECIN-4 INDUCES T CELL APOPTOSIS AND AMELIORATES INTESTINAL INFLAMMATION IN AN EXPERIMENTAL MODEL OF MURINE COLITIS
D. Pacick1, C. Guzy1, U. Berndt1, B. Wiedermann1, A. Dignass2, H.-J. Gabius1, A. Sturm1. 1Charite, Campus-Virchow Clinic; 2Markus-Krankenhaus, Frankfurt; 3Institut für Physiologische Chemie, Tierärztliche Fakultät, LMU, München

Background: Galectins, a growing family of animal lectins, has recently attracted the interest of cell biologists and immunologists as master regulators of immune cell homeostasis. In this family, Galectin-4 (Gal-4) is of particular interest with regard to the mucosal immune system since it is expressed in intestinal epithelial cells and the colonic mucosa. Thus, we aimed to explore the effect of Gal-4 in the human immune system and in an experimental model of colitis.

Methods: PBT were stimulated with cross-linked anti-CD3/28 mAb and cultured in the presence or absence of Gal-4. Flow cytometry analysis was used to determine apoptosis (Annexin-V), necrosis (PI), cell activation (CD25), cell cycle progression (Cyclin A, B1) and expansion (CFDA). Cytokine secretion was determined by CBA analysis. Acute colitis was induced in BALB/c mice by feeding 3% DSS to the drinking water for 7 days. PBT were treated i.p. with either NaCl as control or Gal-4 (1 mg/kg BW) three times a day until they were sacrificed on day 8. The disease activity (DAI) and histological inflammation index were determined. The TUNEL and Bcl-2 staining were performed to determine mucosal apoptosis and proliferation.

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