transduction, regulation of transcription and encode cytokines and surface receptors. Among 76 genes differentially regulated in inactive IBD, only 13 genes were regulated in both CD and UC. In aCD and iCD, 73 genes were significantly differentially expressed. 39 genes were exclusively regulated in aCD, 15 genes were exclusively regulated in iCD.

Conclusions: Treg show a distinct gene expression profile compared to CD+CD25+ T cells. As there are marked differences comparing Treg from HC and IBD as well as from CD to UC, further analysis will help to better understand Treg biology and to define Treg pathobiology in both, CD and UC.

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LUMINAL ANTIGENS ACCESS NOT ONLY MHC II BUT ALSO MHC I PATHWAYS IN MULTIVESICULAR LATE ENDOSOMES OF INTESTINAL EPITHELIAL CELLS - IN VIVO STUDY IN CROHN'S ILEITIS
G. Hunderföhn, S. Strobel, K. P. Zimmer, A. Gebert, D. Ludwig, J. Büning. 1. Department of Gastroenterology, University Clinic of Schleswig-Holstein, Campus Lübeck, Lübeck, Germany; 2. Institute of Anatomy, University of Lübeck, Lübeck, Germany

Background and Aims: In contrast to healthy conditions, intestinal epithelial cells (IECs) are thought to stimulate pro-inflammatory CD4+ and CD8+ T cells during Crohns disease (CD). However, the underlying mechanisms in antigen processing and presentation, particularly with regard to the presentation of exogenous antigens via MHC I, remain unclear. Here we investigated the epithelial expression of MHC I and MHC II and its interference with endocytic pathways of luminal antigens, in vivo.

Methods: During ileoscopy, ovalbumin (OVA) was sprayed onto ileal mucosa of CD patients (active ileitis and ileitis in remission) and controls. The epithelial trafficking of OVA and MHC I/II pathways were subsequently studied in mucosal biopsies using fluorescence and cryo electron microscopy.

Results: Beside its expression at the basolateral membranes, MHC I was detected throughout the endocytic tract of IECs. Of note, MHC I molecules were found to accumulate intracellularly within MHC II-enriched multivesicular late endosomes of IECs. This compartment was efficiently accessed by internalized OVA already 10 minutes after endoscopic application. Vesicles, likewise those enclosed in multivesicular late endosomes, were consistently detected in the intercellular spaces of the epithelium and carried MHC I, MHC II and OVA at later periods. OVA trafficking and the subcellular distribution of MHC I and MHC II in IECs showed no difference between CD patients and controls.

Conclusions: We suggest that multivesicular late endosomes are responsible for MHC I- and MHC II-related processing of exogenous antigens in IECs. The intercellularly detected vesicles might represent immunocompetent exosomes released by IECs and originate from multivesicular late endosomes.

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VISFATIN - A NOVEL MEMBER OF THE ADIPOCYTOKINE FAMILY IS ACTIVATED IN INFLAMMATORY BOWEL DISEASE
A. Maschen, A. Kasner, B. Enrichl, H. Niederregger, H. Tilg. 1. Department of Medicine, Christian Doppler Research Laboratory for Gut Inflammation and Clinical Division of Gastroenterology and Hepatology, Innsbruck Medical University, Innsbruck, Austria; 2. Innsbruck Biocentre, Division of Experimental Pathophysiology and Immunology, Innsbruck Medical University, Innsbruck, Austria

Background: The adipose tissue has emerged as important immunologic or- gan and adipocytokines have been shown to be potent modulators of inflam- mation. Several adipocytokines such as leptin and adiponectin have been shown to be potent modulators of inflammation. Several adipocytokines such as leptin and adiponectin are involved in intestinal inflammation. In the present study we focused on the role of the recently identified adipocytokine visfatin in the immunopathogenesis of inflammatory bowel disease (IBD).

Methods: Serum samples of 56 IBD patients (Crohn’s Disease, n=30; ulcerative colitis, n=26) and 37 healthy controls were assayed for visfatin using a specific enzyme immuno assay. Relative visfatin mRNA expression was determined by qPCR in involved and non-involved intestinal biopsy specimens. Expression of colo- nal sources were determined by confocal microscopy. In vitro, the effect of recombinant visfatin was tested on monocytes, macrophages, and dendritic cells (DCs). In vivo, the effect of recombinant visfatin was tested in Balb/c mice.

Results: Circulating visfatin was significantly elevated in IBD patients com- pared to healthy controls. Colonic visfatin mRNA expression was up-regulated in involved colonic biopsy specimens of both CD and UC patients compared to control subjects. Determined by confocal microscopy, visfatin expression was detected in macrophages (CD163+) and dendritic cells (DC-SIGN+) of the submucosa. Notably, visfatin was found in colonic epithelial cells (CK18+) and mesenteric adipocytes. In vitro, recombinant visfatin induced the production of IL-1beta, TNFalpha and especially IL-6. It increased the surface expression of CD40, CD46, and CD80. Moreover, visfatin-stimulated monocytes showed augmented MHC-II up-regulation and an enhanced capacity to induce allo- proliferative responses. Notably, in vivo treatment with recombinant visfatin resulted in elevated circulating levels of IL-6 that mainly originated from the small intestine.

Conclusions: Taken together, our data demonstrate that visfatin is up- regulated in human IBD. Recombinant visfatin shows considerably pro- inflammatory activity in vivo and in vitro and thus might be considered as a novel pro-inflammatory adipocytokine in IBD.

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IN VITRO EFFECTS OF TNF-ALPHA AND INTERLEUKIN (IL)-10 (IL-10) PRODUCTION BY ACTIVATED PERIPHERAL BLOOD MONONUCLEAR CELLS (PBMCs) FROM HEALTHY CONTROLS AND CROHN’S DISEASE PATIENTS BY DIFFERENT LIPID EMULSIONS FOR PARENTERAL USE WITH A VARIOUS n-6/n-3 POLY
B. Dupont, S. Allouche, R. Gloro, S. Soli, J.-M. Reimund. 1Hépato-Gastro-Entérologie et Nutrition, CHU de Caen; 2EA 3919, Université de Caen - Base Normandie, UFR de Médecine; 3 Biochimie Hospitalière, CHU de Caen

Rationale: Fish oil-derived n-3 PUFAs have prompted a growing interest in human therapeutics as potential inflammatory and immune regulators. Never- theless, until now, when administered orally, their benefit in inflammatory diseases treatment remains unclear.

Methods: PBMCs have been obtained from peripheral blood from fast- ing Crohn’s disease (CD) patients and healthy controls (HC). PBMCs (1000000/mL/well; activated by 10 microg/mL of LPS from Salmonella abortus suis) were cultured at 37°C [humidified air (95%)/CO2 (5%) atmosphere] in RPMI 1640 containing 10% fetal bovine serum, 2% L-glutamine and 1% antibiotics (streptomycin/penicillin). In the presence or not of 0.01%, 0.1% and 1% of either Omegaven® or Endolipide®. After 24 hours of culture, supernatants were removed and stored at -80°C until cytokine measurement by ELISA (R&D Systems).

Results: (i) Omegaven® did not influence TNF-alpha production by LPS- activated PBMCs from HC and CD patients; noteworthy, Omegaven® at 1% slightly decreased in vitro TNF-alpha concentrations in culture supernatants. (ii) Both Omegaven® and Endolipide® inhibit strongly and dose-dependently IL-10 production by HC and CD patients PBMCs. (iii) Finally, previous studies using HC PBMCs suggest that a more balanced n-6/n-3 ratio (i.e. 60% Endolipide® - 40% Omegaven®) slightly inhibited the inhibition of TNF-alpha production (∼78% inhibition compared to LPS-activated PBMCs TNF-alpha production in the absence of any PUFAs).

Conclusions: (i) By contrast to lipid emulsions for parenteral use with a high n-6/n-3 PUFAs ratio which appear not to be able to modulate LPS-activated PBMCs cytokines production [1], lipid preparations for IV use with a more balanced n-6/n-3 ratio may inhibit in vitro TNF-alpha production from both HC and CD patients LPS-activated PBMCs.