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 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 pathology in both, CD and UC.

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LUMINAL ANTIGENS ACCESS NOT ONLY MHC II BUT ALSO MHC I PATHWAYS IN MULTIVESICULAR LATE ENDOSONMES OF INTESTINAL EPITHELIAL CELLS - IN VIVO STUDY IN CROHN’S ILEITIS
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Background and Aims: In contrast to healthy conditions, intestinal epithelial cells (IECs) are thought to stimulate pro-inflammatory CD4+ and CD8- T cells during Crohn’s 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 epitelial traffic 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 tracts 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. Thus, presentation of exogenous antigens by IECs is most likely not restricted to MHC II, but also occurs as “cross presentation” via MHC I. The distinct capacity of IECs in antigen presentation might depend on differential antigen processing and production of immunostimulatory exosomes within the subset of late endosomes identified in our study.

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VISFATIN - A NOVEL MEMBER OF THE ADIPOCYTOKINE FAMILY IS ACTIVATED IN INFLAMMATORY BOWEL DISEASE
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Background: The adipose tissue has emerged as important immunologic or- gan and adipokines have been shown to be potent modulators of inflamma- tion. Several adipokynes such as leptin and adiponectin are involved in intestinal inflammation. In the present study we focused on the role of the recently identified adipokine 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 was determined by RT-qPCR in involved, non-involved, and control colonic biopsy specimens. Circulating visfatin was determined by ELISA (R&D Systems).

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 CD34, CD46, and CD68, moreover, visfatin-stimulated monocytes showed augmented FITC-dextran uptake 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 capacity in vivo and in vitro and thus might be considered as a novel pro-inflammatory adipokine 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
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Rationale: Fish oil-derived n-3 PUFAs have prompted a growing interest human therapeutics as potential inflammatory and immune regulators. Nev- ertheless, until now, when administered orally, their benefit in inflammatory diseases treatment seems not to be of major impact.

Aim: We studied the in vitro modulation of lipopolysaccharide (LPS)- activated PBMCs TNF-alpha and IL-10 production by a 100% n-3 containing lipid emulsion (OmegaVen®), a soybean oil-derived lipid emulsion with a 7.4 n-6/n-3 PUFAs ratio (Endolipide®, Baxter Clincinet).

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 equi) 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) Endolipide® 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% OmegaVen® - 40% OmegaVen® (n-6/n-3: 7.4) may be more efficient in vitro TNF-alpha inhibition (∼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. (ii) Both n-6/n-3 ratios (n-6/n-3: 7.4 and 100% n-3) significantly inhibit IL-10 production in the same experimental conditions. In vitro data, taken together with recent beneficial results reported by administering OmegaVen® intravenously in active rheumatoid arthritis patients [2], need to be considered and to be confirmed before po- tential (nutritional or therapeutic) use of IV n-3 PUFAs in CD patients.