murine model. Moreover, Ussing Chamber analysis shows that the intestinal permeability is maintained similar to the one of untreated mice, and colon length results comparable to untreated animals. In addition, both the systemic and local analysis of cytokines shows an increase of the anti-inflammatory cytokine IL-10 and a reduction of inflammatory cytokines.
Conclusions: These results show that SGLT-1 activation may restore the gut barrier function, usually lost during chronic colitis, and contrast with the inflammatory status. Activation of SGLT-1 by D-glucose, orally administered, may be suggested as a new pharmacological approach for the treatment of Crohn's disease.

P277
Clinical features of ulcerative colitis in pANCA-positive patients in Russian Federation

A.G. Kharitonov[1]*, E.A. Kondrashina[1], A.Y. Baranovsky[1], S.V. Lapin[2]. *[1]Saint-Petersburg Medical Academy of Postgraduate Education of Roszdrav, Saint-Petersburg, Russian Federation, [2]Saint-Petersburg State Medical University, Saint-Petersburg, Russian Federation*

Several antibodies have been investigated in the serum of patients with ulcerative colitis (UC). The most commonly described is perinuclear antineutrophil cytoplasm antibodies (pANCA). The aim of our study was to determine whether the presence of pANCA is associated with particular clinical features of UC in Russian patients.
Patients and Methods: Forty nine well-characterized, unrelated patients with UC were prospectively recruited from Caucasian population. Diagnosis was established on clinical findings, endoscopy and histology. ANCA (IgA and IgG) were determined in all patients by means indirect immunofluorescence. Depending on pANCA titre (normal or high) patients were divided in two groups: pANCA-positive patients (APP) with diagnostic titre >1:40 and pANCA-negative patients (ANP). Clinical data, disease activity were gathered at the time of serum sampling as well as clinical course of disease and effect of treatment were investigated in both groups.
Results: Total pANCA (IgA+IgG) were detected in 33 of 49 patients (67%), where 29 were pANCA IgG-positive (59.2%) and 8 pANCA IgA-positive (16.3%). Only 4 from 49 pANCA-positive patients (8%) had both IgA and IgG pANCA. We found that APP had worse well-being ($p = 0.043$), higher body temperature ($p = 0.003$), higher platelet level ($p = 0.01$) in active disease compared with ANP. APP also had greater median disease duration ($p = 0.01$). No significant association was found between pANCA-presence and disease activity and extent.
Conclusion: Patients with diagnostic titre of pANCA had worse well-being, higher body temperature and platelet level in active UC. It is possible depend on greater expressed inflammatory activity and autoimmune process in APP.

P278
Granulocyte macrophage colony stimulating factor improves mucosal repair in a mouse model of acute colitis

E. Bernasconi[1], L. Favre[1], D. Bachmann[1], H. Bouzourene[2], E. Croze[3], S. Velichko[3], J. Parkinson[3], P. Michetti[1], D. Velin[1]*. *[1]Centre Hospitalier Universitaire Vaudois, Lausanne, Switzerland, [2]Institute of Pathology, Lausanne, Switzerland, [3]Berlex Biosciences, Richmond, CA, USA*

Background and Aims: Granulocyte-macrophage colony-stimulating factor (GM-CSF), a cytokine modulating the number and function of innate immune cells, has been shown to provide symptomatic benefit in some patients with Crohn's disease (CD). Since, it becomes widely appreciated that a timely and spatially regulated action of innate immune cells is critical for tissue regeneration, we tested whether GM-CSF therapy may favours intestinal mucosal repair in the acute mouse model of dextran sulfate sodium (DSS)-induced colitis.
Methods: Mice treated with GM-CSF or saline were exposed for 7 days to DSS to induce colitis. On day 5, 7 and 10, mice were subjected to colonoscopy or sacrificed for evaluation of inflammatory reaction and mucosal healing.
Results: GM-CSF therapy prevented body weight loss, diarrhea, dampened inflammatory reactions and ameliorated mucosal damages. Mucosal repair improvement in GM-CSF-treated mice was observed from day 7 on both by colonoscopy (ulceration score 1.2 ± 0.3 (GM-CSF-treated) vs 3.1 ± 0.5 (untreated), $p = 0.01$) and histological analysis (percentage of reepithelialized ulcers $55\% \pm 4\%$ (GM-CSF-treated) vs $18\% \pm 13\%$ (untreated), $p = 0.01$). GM-CSF therapy can still improve the colitis when hematopoietic, but not non-hematopoietic cells, are responsive to GM-CSF, as shown in WT $\rightarrow$ GM-CSFRKO chimeras. Lastly, we observed that GM-CSF-induced promotion of wound healing is associated with a modification of the cellular composition of DSS-induced colonic inflammatory infiltrate, characterized by the reduction of neutrophil numbers and early accumulation of $CD11b^{+}Gr1^{lo}$ myeloid cells.
Conclusion: Our study shows that GM-CSF therapy accelerates the complex program leading to tissue repair during acute colitis and suggests that GM-CSF promotion of mucosal repair might contribute to the symptomatic benefits of GM-CSF therapy observed in some CD patients.

P279
Low dose administration of cytokines and antibodies for the treatment of Crohn's disease

L. Zanobbio, M. Palazzo, S. Gariboldi, G.F. Dusio, D. Cardani, L. Battini, C. Rumio*. *Department of Human Morphology, University of Milan, Milano, Italy*

Aim: Cytokines play an important role in the pathology of Crohn's disease, by determining the nature of the mucosal immune response. In particular, Crohn's disease is characterized by high expression of many inflammatory cytokines, such as IL-1, IL-6 and TNF-α. Today many therapeutic proposals for Crohn's disease aim to re-establish normal levels of cytokines, by using anti-inflammatory cytokines, such as IL-10, or blocking inflammatory mediators, such as TNF-α or IL-1. Though potentially effective, the use of cytokine-based therapies is limited by the possible collateral effects. Therefore, our study aims to determine, in a mouse model of Crohn's-like inflammatory bowel disease, the possible therapeutic activity of low dose IL-10 plus anti-IL-1 antibody solutions, mechanically activated.
Materials and Methods: A chemically-induced mouse model of Crohn's-like inflammatory bowel disease was used. Following oral treatment with activated solutions of IL-10 plus anti-IL-1 antibody (1 fg/die/mouse), transepithelial electrical resistance of isolated mouse colon and colon length were evaluated. Inflammatory cytokines IL-12, TNF-α, IL-17, IFN-γ, KC, and anti-inflammatory IL-10 were evaluated by ELISA in plasma and in organotypic cultures of colon.
Results: Crohn's disease is characterized by a disrupted intestinal barrier function, manifested by an increase in intestinal epithelial permeability. Our data show that oral administration of activated solutions of IL-10 plus anti-IL-1 antibody (1 fg/die/mouse) restores barrier function in our murine model of Crohn's disease: in fact, Ussing Chamber analysis shows that the intestinal permeability is maintained similar to the one of normal mice, and colon length results comparable to normal animals. In addition, both the systemic and local analysis of cytokines shows an increase of the anti-inflammatory cytokine IL-10 and a reduction of inflammatory cytokines.
Conclusions: These preliminary results show that oral administration of activated solutions of IL-10 plus anti-IL-1 antibody may restore the gut barrier function, usually lost during Crohn's disease, and contrast with the inflammatory