order to investigate the impact of CS on the clinical course of IBD, its effect on the concentrations of these molecules in serum and urine was assessed.

**Methods:** Prospective observational 12-month (m) follow-up study in patients with IBD in remission for at least 6 m, starting CS (Condrozan®). Biobérica S.A., Barcelona, Spain) treatment for osteoarthritis (OA). Visits were as follows: Baseline, 3rd, 6th, 9th and 12th m. CDAI and modified Truelove–Witts clinical indexes were calculated for Crohn's disease (CD) and ulcerative colitis (UC) respectively. Orosomucoid, C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) were also determined. Levels of VEGFA, VEGFC, FGF2, HGF, Ang1, Ang2, TGFβ, TNFα, IL1β, -6, -12, -17, -23, ICAM1, VCAM1, MMP3 and PGE2 were determined by ELISA. OA joint pain was evaluated by a visual analogue scale. The study is still ongoing.

**Results:** At present, 29 patients with IBD (16 UC, 13 CD) have been included. Mean age was 62 years, and 72% were women. The mean disease duration was 14 years. 69% of patients were studied pro-inflammatory markers were not found. At 12th m visit (236 pg/mL) (P < 0.05). Further differences regarding the other studied pro-inflammatory markers were not found. At 12th m, the OA joint pain had improved in all patients (P < 0.01). 30% of patients suffered adverse events, but only 6% were related to the drug.

**Conclusions:** The incidence of IBD relapse in patients under CS treatment was lower than the generally reported. CS decreases pain related to OA in patients with IBD.

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**ADAMDEC1: a novel molecule linked to Crohn's disease, is associated with an increased susceptibility to Citrobacter rodentium colitis in the knock out mouse**

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**Background:** Innate immunity is attenuated in patients with Crohn’s disease (CD), with impaired neutrophil recruitment, delayed clearance of E. coli, and defective secretion of pro-inflammatory cytokines from macrophages [1,2]. This primary macrophage defect may result in failure to eradicate bacterial flora entering the tissues and lead to the chronic granulomatous inflammation characteristic of CD. To discover the molecules responsible, transcriptomic profiles were obtained from cultured human macrophages from CD patients and controls. ADAMDEC1 a Disintegrin and Metalloprotease was under-expressed in -10% of CD patients. This protein is almost exclusively found in macrophages and dendritic cells in the small and large bowel lamina propria. Here we describe the response of Adamdcl+/− mice to an enteric bacterial infection with Citrobacter rodentium.

**Methods:** Adamdcl+/− and wild type mice were administered ~10⁸ or 10⁹ C. rodentium by oral gavage and body weight monitored for three weeks. At intervals mice were sacrificed and samples of serum, stool, colon and spleen were collected. Serum cytokine levels were measured and bacteria counted in stool and spleen. Bowel inflammation was assessed histologically. Neutrophil and immune cell recruitment to the colon were measured by MPO assay and qPCR respectively.

**Results:** During infection, control mice experienced a mild self-limiting colitis, with minimal weight loss. Expression of Adamdcl was up-regulated in the colon and this normalised with resolution. Adamdcl−/− mice were more susceptible to C. rodentium infection: they demonstrated dramatic weight loss (p < 0.001), a more severe colitis and a reduced survival at the higher dose (67% vs 0%, p < 0.009). Serum levels of TNF, IL-1 β and IL-6 were significantly lower in the knock-out mice (p < 0.05). Impaired survival was associated with positive cultures of the organisms from the spleen (p = 0.02).

**Conclusions:** By analysing the transcriptome of macrophages from CD patients we have identified a novel molecule involved in mucosal immunity. Further work is underway to elucidate the precise role of ADAMDEC1 in the immune response. Individuals with grossly attenuated expression levels may be