median age 25.5 years (16.7–69); 45% male) patients across three Belgian IBD centres (University Hospitals Brussels, Ghent and Leuven). New diagnosis was defined as within 3 months after diag-nosis, naive for biologicals and immunosuppressives, and no previous IBD-related surgery. A panel of 91 inflammatory proteins (Olink) was quantified in the serum sample taken at diagnosis, and of age- and gender-matched healthy controls (n = 80, ± 3.6 years). Wilcoxon rank-sum and t-tests were used as appropriate, and multiple testing correction applied (Benjamini-Hochberg method, R 3.4.2). An adjusted p-value of < 0.05 was considered significant.

**Results:** Comparison of protein levels in CD patients with matched controls identified 44 significantly different proteins, with OSM (fold change (FC) = 4.0, p = 1.7E–12) and IL-6 (FC = 3.7, p = 3.9E–12) as the most dysregulated proteins. When comparing UC with controls, 39 significantly different proteins were found, 29 of which were also different in CD, incl. IL-6 and OSM (ranked 9th and 10th). The most differentially expressed protein in UC was CXCL1 (FC = 1.7, p = 4.5E–07). We then stratified CD and UC into quartiles based on the age at diagnosis (≤21.5, 21.5–25.5, 25.5–33.5 and >33.5 years for CD; ≤20.6, 20.6–26.0, 26.0–33.7 and >33.7 years for UC), and compared each subgroup with its matched control group. A comparable number of differentially expressed proteins was observed for quartiles 1 to 3 in CD (n = 25–30 proteins), of which 12 overlapped, incl. OSM and IL-6. Only one protein, FGF-19, was dysregulated in the oldest CD group. For UC patients, quartiles 1 and 2 showed comparable results (13 and 8 different proteins, overlap of 7), while no differences were observed for the two oldest UC groups.

**Conclusions:** We identified panels of inflammatory markers defining newly diagnosed CD and UC, with some common for both (OSM, IL-6), while others appear to be specific for either CD or UC. We found a decreased inflammatory burden with increasing age, providing further evidence for the less severe clinical symptoms in late-onset compared with early-onset IBD.

**P119**

**The immunohistochemical assessment of CD30+ lymphocytes in the intestinal mucosa facilitates diagnosis of paediatric ulcerative colitis**

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**Background:** Diagnosis of paediatric inflammatory bowel diseases (IBD) remains challenging. We aimed at evaluated the value of immunohistochemical assessment of CD30+ lymphocytes in the intestinal mucosa in differential diagnosis between paediatric Crohn’s disease (CD) and ulcerative colitis (UC) and its utility as a predictor of future differentiation in patients with IBD unclassified (IBDU).

**Methods:** 74 treatment naive paediatric patients with IBD (33 CD, 30 UC, and 11 IBDU) were enrolled into the study. Biopsy samples from six different regions (terminal ileum, caecum, ascending colon, transverse colon, descending colon, and rectum) were immunohistochemically stained with anti-CD30 antibody and number of positive cells per one high power field was quantified.

**Results:** Significant differences between CD and UC were found when compared with all counts of CD30+ cells in median numbers, mean values, and maximal numbers and also for separate counts in terminal ileum, transverse colon, descending colon and rectum. The most profound difference between CD and UC was shown for total median values of CD30+ cells and for the values in rectal localisation. The difference was independent on the intensity of inflammation. A cut-off value of 2.5 CD30+ cells with sensitivity 83% and specificity 90% was found for the rectum. There was no difference between patients with CD and IBDU, but a marked difference between UC and IBDU patients was revealed.

**Conclusions:** Histopathological assessment of biopsy with rectal CD30+ count is reliable and simple method that could help in differential diagnosis among IBD subtypes in children with IBD.

**P120**

**Vitamin D deficiency in Crohn’s disease**

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**Background:** Vitamin D deficiency is prevalent in Crohn’s disease (CD) and has been associated with decreased disease activity and reduced need for corticosteroids. However, the value of this relationship is not clearly understood.

**Methods:** A cross-sectional study of 454 consecutive CD patients attending gastroenterology clinics at a single tertiary centre. Patients were age and sex matched with a control group of 454 healthy subjects. The body mass index (BMI) was calculated, and the 25(OH)D level measured. The study included 294 treatment naïve patients (152 females, median age 26.3 years, IQR 19.2–35.2). Median BMI was 24.1 (IQR 22.7–26.6). A total of 202 patients (69%) were vitamin D deficient with serum 25(OH)D levels < 30 nmol/L. The median vitamin D level was 18.8 (IQR 11.9–30.6) nmol/L.

**Results:** The median BMI of the vitamin D deficient patients was 24.5 (IQR 22.9–26.4) compared to 24.0 (IQR 21.9–26.3) in the controls (p = 0.004). Vitamin D deficiency was associated with a lower BMI (p = 0.002), but not with age or gender (p > 0.05). Vitamin D deficiency was associated with a lower BMI (p = 0.002), but not with age or gender (p > 0.05). The relationship between vitamin D deficiency and BMI was independent of age and gender (p > 0.05).

**Conclusions:** Vitamin D deficiency is prevalent in Crohn’s disease and is associated with a lower BMI. This suggests that vitamin D deficiency may contribute to the pathogenesis of Crohn’s disease and its associated features, such as decreased weight gain and increased weight loss.
Background: Vitamin D deficiency is common among patients with Crohn’s disease (CD) and has been a proposed risk factor for the development and flare of CD. It remains unclear, however, if this association is a result of the inflammatory process, or a cause. Furthermore, most studies have relied on radioimmunoassays to measure 25-hydroxyvitamin D (25(OH)D), which may be less accurate than the accepted gold standard of liquid chromatography tandem mass spectrometry (LC/MS/MS). Circulating 25(OH)D is metabolised to the metabolically active 1,25(OH)2D. The alternative pathway involves the production of the inactive 24,25(OH)D via 24-hydroxylase prior to elimination. The ratio of 25(OH)D:24,25(OH)D may be a more accurate measure of vitamin D status than 25(OH)D alone. We aimed to characterise vitamin D metabolism in patients with active and inactive CD using LC/MS/MS.

Methods: We report the baseline cross-sectional results of a prospective cohort study. Patients were included if they had active CD defined as ulceration at endoscopy; or a Crohn’s disease Activity Index (CDAI) >220 and a C-reactive protein >10 mg/l or faecal calprotectin >250 mg/kg. Remission was defined as a CDAI <150 and normal inflammatory biomarkers. Patients were excluded if they received corticosteroids or vitamin D supplementation in the preceding 4 weeks. Serum was tested for 25(OH)D, epi-25(OH)D, 1,25(OH)2D and 24,25(OH)2D using an LC/MS/MS assay. Validated questionnaires were used to estimate vitamin D exposure from diet and sunlight. Spearman’s correlation coefficient was used to test correlations and unpaired t-tests to test differences between active and remission CD groups.

Results: Forty-seven consecutive patients with CD (20 active and 27 remission) were recruited; 55% were male. Median age was 37 years (range 23 to 76yr). Fewer patients in the active group were on immunomodulators (30% vs. 61% p = 0.03) or TNF inhibitors (25% vs. 89% p < 0.001). There was no difference in serum 25(OH)D, epi-25(OH)D or 1,25(OH)2D levels between the groups. Serum 24,25(OH)2D levels were significantly lower in the active group (mean 1.3 vs. 2.5ng/ml p < 0.001) and thus the 25(OH)D:24,25(OH)2D ratio was higher (49.4 vs. 26.1 p < 0.001). There was an inverse correlation between CDAI and 24,25(OH)2D levels (r2 = 0.33; p = 0.01). Dietary vitamin D intake and sunlight exposure were not different between the groups.

Conclusions: In the setting of active inflammation, levels of vitamin D should be measured to assess the degree of vitamin D deficiency. The 25(OH)D:24,25(OH)2D ratio may provide a more sensitive marker of vitamin D status in patients with CD.

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Vitamin D activates human intestinal fibroblasts

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Background: Vitamin D signals through the vitamin D receptor (VDR) which is a member of the nuclear receptor family of transcription factors. VDR selectively activates genes involved in mineral homeostasis, cell growth, differentiation and apoptosis. In addition, the VDR is also involved in other biologic processes such as immune system and metabolism. The aim of this work is to determine the effect of 1,25(OH)2D3 on human intestinal fibroblasts (HIF) via VDR.

Methods: Isolation of human intestinal fibroblasts from healthy subjects. Viability and proliferation were assessed by Trypan blue dye exclusion and CCK-8 assay. Western blot analysis for VDR, and collagen I and III were performed. Realtime PCR for VDR, collagen I and III, and TGF-β1,

Results: In the presence of 1,25(OH)2D3, the viability and proliferation of intestinal fibroblasts were increased and the expression of VDR, collagen I, and III. Moreover, the expression of TGF-β1 was decreased in the presence of 1,25(OH)2D3.

Conclusions: Our results show that 1,25(OH)2D3 is able to activate human intestinal fibroblasts via VDR. This effect could be useful for the treatment of inflammatory bowel disease.