weeks. After the 2nd dose she began with abdominal pain and increased depositions. Acute phase reactants were increased major PCR 194 mg/dl ESR 87 mg/dl. In colonoscopy, biopsies objective and inflammatory lesions with extensive areas of mucosal ulceration compatible with Crohn's disease were observed.

Case 2 A 59-year-old man with a diagnosis of ankylosing spondylitis of 30 years of evolution without a clinical response to NSAIDs and sulphasalazine, was treated with SEK 150 mg. After the second dose during induction, He presented fever 38.9°C, abdominal pain, bloody diarrhea, and nocturnal tenesmo. A colonoscopy showed typical ulcerative colitis lesions with severe activity.

Conclusions SEK is a very potent treatment for psoriasis and psoriatic arthritis, with positive results in Spondylitis, but not effective for IBD. IL-17 is a cytokine with a role in the normal intestinal homeostasis.

Psoriasis and Spondyloarthritis patients have an association with IBD and uveitis.

We present 2 patients who were diagnosed of IBD shortly after start SEK, anti-IL17. We think they could have a previous undiagnosed IBD; SEK could have trigger a flare but We can't rule out a role of IL-17 inhibition. During clinical trials for SEK, only 0.7 cases per 100 patients-year of IBD were reported. Caution must be taken before start SEK for patients to detect possible symptoms of IBD.

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Disclosure of Interest None declared.

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THE ROLE OF THE IL-23/TH17 AXIS AS MODULATOR OF B CELL-MEDIATED (AUTO)IMMUNE RESPONSES

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Career situation of first and presenting author Post-doctoral fellow.

Introduction Rheumatoid arthritis (RA) is characterized by chronic synovitis and joint destruction. Autoantibodies and autoreactive B cells are a hallmark of RA. Furthermore, Th17 cells have been demonstrated to be crucial for disease development. However, checkpoints and mechanisms regulating the onset of rheumatoid arthritis (RA) remain largely unclear.

Objectives Recently, we have demonstrated, that Th17 cells suppress the enzyme ST6 a-galactoside b-2,6-sialyltransferase in developing plasma cells. Thereby, Th17 cells are able to increase the inflammatory activity of autoantibodies in an IL-23 dependent manner by regulating the degree of Fc-glycosylation. However, the molecular mechanisms that mediate this IL-23/Th17-mediated proinflammatory reprogramming of B cells and the relevance for arthritis remains to be determined in detail.

Methods K/BxN mice were treated twice a week with neutralizing antibodies against IL-17A and IL-22 from week 1 until week 9. Both, clinical, histological and immunological parameters of arthritis were assessed. Serum was collected weekly and autoantibody titers were determined using ELISA. Collected Sera were used for K/BxN serum transfer to evaluated IgG activity. Additionally, the expression levels of the corresponding interleukin receptors (IL17RA and IL22Ra1) were analyzed during collagen-induced arthritis.

Results Here we show, that in contrast the upregulation of IL-22Ra1 on plasmablasts in the spleen, lymph node and bone-marrow during collagen-induced arthritis, its blockade during K/BxN arthritis did not prevent the onset of arthritis. Also, the IgG from K/BxN mice remained its inflammatory activity. We also show, that while IL17RA is basically absent on B cells during arthritis, the neutralization of IL-17A during K/BxN arthritis led to a strongly delayed development of autoimmune arthritis. Furthermore, depletion of IL17A led to a decreased titer of autoantibodies during early phases of K/BxN arthritis. However, in the K/BxN serum transfer mice which received serum that was produced in the absence of IL-17 were not protected against inflammatory arthritis.

Conclusions Our data indicate that IL-22 is not directly involved in the regulation of IgG activity during K/BxN arthritis. Furthermore, our data indicate that IL-17A is indeed a crucial cytokine during inflammatory processes inside the joint and may be involved in the germinal center reaction or plasma cell survival. However, neither IL-17 nor IL-22 is involved in the IL-23/Th17-mediated proinflammatory reprogramming of B cells to produce highly pathogenic autoantibodies. Therefore, other cytokines produced by Th17 cells and/or mechanisms depending on cell-cell contact proteins might orchestrate the Th17/B-cell crosstalk leading to the downregulation of St6gal1 in developing plasma cells.

Disclosure of Interest None declared.

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INTRACELLULAR INTERLEUKIN-1 RECEPTOR ANTAGONIST RELEASED UPON CELL DEATH ACTS AS AN ALARMIN INHIBITOR IN ALDARA CREAM-INDUCED PSORIASIS-LIKE SKIN INFLAMMATION

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Career situation of first and presenting author Post-doctoral fellow.

Introduction The inflammatory effects of interleukin (IL)-1 are tightly controlled by IL-1 receptor antagonist (IL-1Ra), which blocks the binding of both IL-1 α and IL-1 β to IL-1R1. Four IL-1Ra isoforms are produced from the same gene by the use of different first exons, alternative mRNA splicing and translation initiation sites. One IL-1Ra isoform is secreted (sIL-1Ra), whereas the three others are intracellular (icIL-1Ra1, 2, 3) due to the absence of a signal peptide. In contrast to the well-characterized function of the secreted isoform, the biological role of the intracellular isoforms remains largely unclear. The icIL-1Ra1 isoform is constitutively expressed and represents the major isoform in keratinocytes.