infected with EBV. Furthermore, ELS-containing RA synovia transplanted into SCID mice supported production of ACPA and anti-VCP1/VCP2 antibodies cross-recognised by ACPA. Analysis of CD4+ and CD8+ T-cell localisation and granzyme B expression suggests that EBV persistence in ELS-containing synovia is favoured by exclusion of CD8+ T cells from B-cell follicles and impaired CD8-mediated cytotoxicity.

Conclusions We demonstrated active EBV infection within ELS in the RA synovium that appears to contribute to local growth and differentiation of ACPA-reactive B cells.

A6.6 THE ROLE OF SYNDECAN-4 IN EXPERIMENTAL COLITIS

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Background and Objectives The transmembrane heparan sulphate proteoglycan syndecan-4 (Scd4) has been implicated in cell-matrix adhesion, cell migration, differentiation, proliferation and plays an important role during inflammation in rheumatoid arthritis. Scd4 is a mediator and modulator of inflammatory signals, upon its binding of cytokines Scd4 acts either as a decoy receptor or through the initiation of Scd-dependend signalling, followed by the formation of a Scd4 complex. Cartilage damage is decreased in sdc4-deficient mice, but osteopontin-mediated liver damage is increased. Because of these dual effects we investigate the impact of sdc4 in murine experimental colitis.

Materials and Methods We performed DSS-induced colitis in Scd4$^{-/-}$ and C57BL/6 WT mice. We used weight loss, colon length and histological scoring of colonic modifications to measure the course of colitis. Scd4$^{-/-}$ and WT mice were orally gavaged with 5×10^8 colony-forming units (CFU) of invasive bacterium *Citrobacter rhodentium* (*C. rhodentium*). The changes of body weight and faecal excretion of *C. rhodentium* were monitored for 21 days followed by evaluation of histological changes after infection. The permeability of the colon was examined in vitro by infection of colon samples from Scd4$^{-/-}$ and C57BL/6 WT mice with *C. rhodentium*. The migration behaviour of endothelial human cells (T-84) and scd4-siRNA T-84 knockdown cells was analysed by scratch assay.

Results DSS-treated Scd4$^{-/-}$ mice lost dramatically more body weight compared to the WT mice and the histological damage according to the Dieleman-Score was markedly increased. At day 19 of post infection the clearance of *C. rhodentium* in Scd4$^{-/-}$ mice was markedly prolonged. In vitro infection of colon samples from Scd4$^{-/-}$ mice with *C. rhodentium* revealed a higher permeability for the bacterium compared to WT colon samples. The knockdown of Scd4 in human endothelial T-84 cells leads to delayed cell migration.

Conclusions Like in inflammatory liver damage, Scd4 appears to play an important role in colitis and exerts protective effects in intestinal inflammation. The Scd4 deficiency leads to a higher permeability of the colon to *C. rhodentium* and a delayed cell migration. Further analysis are needed to explore the mechanisms of Sdc4-signalling in colitis.

A6.7 ALTERATIONS IN NAILFOLD VIDEOCAPILLAROSCOPY IN PATIENTS WITH GRANULOMATOSIS WITH POLYANGIITIS (WEGENER'S): AN OBSERVATIONAL STUDY

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Background Nailfold videocapillaroscopy (NFC), allows for the detection of changes in microcirculation. In the granulomatosis with polyangiitis (GPA) the existence of a defined pattern has not been found.

Objectives The main objective of our study was to detect the possible existence of a defined pattern in the microcirculation of the nailfold capillaries of patients with GPA. The second objective was to investigate the possible correlation between abnormalities found and systemic involvement.

Methods We identified 10 patients with a current mean age of 55.7 ± 16.5 years and predominantly female (60%). The mean age at diagnosis was 49.4 years. 70% had upper respiratory tract involvement, the same percentage had pulmonary involvement (cavitated nodules or alveolar haemorrhage), the cutaneous manifestations such as purpura or necrotic ulcers were present in 70%. About 40% had renal involvement (renal failure, proliferative glomerulonephritis), and 40% had peripheral neurological involvement. NFC was carried out by the same rheumatologist, on fingers 3 through to 5 of both hands using a ZUZI videocapillaroscopy, trinocular, dual illumination and zoom of 1 X 4 X.

Results Abnormalities of the microcirculation of nailfold capillaries were found in 8 of the 10 patients. Among the patients with this pathological microcirculation, 62.5% had structural alterations (tortuous capillaries), 50% presented with micro-haemorrhage (single or multiple), avascular areas were found in 37.5% and 75% showed lower capillary density. Neither capillary dilation nor the formation of new vessels were detected within the sample of patients.

Abstract A6.7 Table 1 Correlation between capilaroscopic finding with organ involvement

Organ involvement	Pathological capillaroscopy	Abnormal morphology	Bleeding	Avascular areas	Reduced capillary density	Expansion
Respiratory (7)	5	3	3	2	5	0
Renal (4)	3	3	1	1	3	0
Neurological (4)	3	1	1	0	3	0
Skin (7)	6	3	3	2	4	0

Conclusions We have observed, more frequent bleeding, avascular areas and reduced capillary density and these findings were not related with any specific organ involvement. There is one only study in GPA which communicates a high percentage of avascular areas. [1]

Reference

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A6.8 BIOLOGICAL THERAPIES IN JUVENILE IDIOPATHIC ARTHRITIS

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Background Biological therapies have dramatically changed the prognosis for children with juvenile idiopathic arthritis (JIA). There are doubts about the possibility of discontinuing treatment once remission is achieved. We focus in this question in our series.

Objective To assess the efficacy and safety of these drugs in our series of patients with JIA.

Matherials and Methods We identified 9 children with JIA treated with biologic therapies, and we made a description of our experience.

Results The mean age was 14.55 ± 5.85, with a female predominance (66.7%). At diagnosis, mean age was 4.94 ± 2.9, and at the beginning of biological treatment of 8.77 ± 2.63. The median time