**SAT0360**  
**BACTERIAL TRANSGLOTTATION IN THE ADJUVANT INDUCED ARTHRITIS MODEL: PRELIMINARY STUDY AND IMPACT OF NSAIDS**  
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**Background:** In patients with spondyloarthritides, the presence of intestinal inflammation and an increase in digestive permeability responsible for bacterial translocation has been described. No data are available on the effect of non-steroidal anti-inflammatory drugs (NSAIDs) on this bacterial translocation in patients with spondyloarthritides. Zonulin and lipopolysaccharide (LPS) have been described as good biomarkers of intestinal permeability and bacterial translocation, respectively. Adjuvant-induced arthritis (AIA) is a model of recent arthritis characterized by ossification and ankylosis in the post arthritis period. This model can be considered as a model of reactive arthritis in which our previous work has reported a clear efficacy of NSAIDs with differences between molecules at the structural and vascular levels.  

**Objectives:** To test the hypothesis that there is an increase in digestive permeability and bacterial translocation in the AIA model and to show the influence of different NSAIDs on these two parameters.  

**Methods:** Adjuvant-induced arthritis (AIA) was induced in 6-week-old male Lewis rats by an injection at the base of the tail of Mycobacterium butyricum. A group of non-AIA (control) rats received saline. At the first signs of arthritis, the AIA-rats were evaluated (arthritis score 0-6) and treated daily intraperitoneally with naproxen (10 mg/kg/day), diclofenac (5 mg/kg twice daily), celecoxib (3 mg/kg/day) or saline solution (AIA-vehicle group). After 21 days of treatment, the rats were sacrificed and serum levels of zonulin and LPS were evaluated by ELISA and liquid chromatography-mass spectrometry, respectively. Circulating levels of TNF-α and IL-1β and paw radiographic score were measured.  

**Results:** Compared to the control group, there was a significant increase in zonulin concentration (p < 0.001) in the AIA group. There was no significant difference in the concentration of LPS between the two groups. The levels of zonulin were correlated with the TNF-α levels (R= -0.42; p=0.032) and the arthritis score (R=0.45; p=0.013) but not with the level of IL1-β (R= p-0.018; p=0.39). Treatment with NSAIDs significantly and equivalently decreased the arthritis score in each group. Compared to the vehicle group, treatment with naproxen significantly decreased the radiographic score (p<0.001), TNF-α, IL-1β (p < 0.01), zonulin (p<0.001) and LPS (p < 0.05). Celecoxib decreased radiographic score (p < 0.001), TNF-α (p < 0.01), IL-1β (p < 0.01) but increased zonulin levels (p < 0.05) without effect on LPS. Diclofenac also decreased radiographic score (p < 0.001), TNF-α (p < 0.01), and IL-1β (p < 0.01) but increased both zonulin (p < 0.01) and LPS (p < 0.001).  

**Conclusion:** We have demonstrated an increase in serum zonulin levels in the AIA model and a beneficial effect of naproxen on intestinal permeability and bacterial translocation in contrast to celecoxib and diclofenac. Moreover, the plasmatic zonulin levels were correlated with TNF-α supporting a pivotal role of TNF-α on the tight junctions in this model.  

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**Figure 1:** Clustering of study participants into sub phenotypes based on tissue level inflammation by imaging.  

**Figure 2:** Gene expression data by imaging clusters.
ASSOCIATION OF GUT DYSBIOSIS WITH STRUCTURAL DAMAGE AND ACTIVITY IN AXIAL SPONDYLOARTHRITIS PATIENTS. DATA FROM THE COSPAR REGISTRY.

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Methods: Fifteen AxSpA patients and 15 healthy donors (HDs) were included in a cross-sectional study. Disease activity variables such as C-reactive protein and ESR were measured. Structural damage was determined by lateral X-rays of the cervical and lumbar spine to establish the mSASSS index. Axial mobility was evaluated using the BASMI index and the enthesis affectation was evaluated using the BASMI (r=0.512, p=0.015).

Background: The etiopathogenesis of axial spondyloarthritis (AxSpA) is multi-factorial. The possible role of alteration in gut microbiome (dysbiosis) has been recently suggested. However, the association of dysbiosis with structural damage is still unknown and further studies are needed to assess its association with disease activity.

Objectives: To determine the alterations in the gut microbiota in AxSpA patients. To evaluate whether changes in the gut microbiome in AxSpA patients are associated with structural damage (mSASSS) and enthesis affectation (BASMI). Methods: Fifteen AxSpA patients and 15 healthy donors (HDs) were included in a cross-sectional study. Disease activity variables such as C-reactive protein and ESR were measured. Structural damage was determined by lateral X-rays of the cervical and lumbar spine to establish the mSASSS index. Axial mobility was evaluated using the BASMI index and the enthesis affectation was evaluated using the BASMI (r=0.512, p=0.015).

Results: AxSpA patients had a significant alteration of the gut microbiota. These alterations are associated with inflammatory and erosive damage.

Conclusion: The rational of our study was to assess whether delphinidin can in vitro suppress IL-17 and IFN-γ production in peripheral blood mononuclear cell (PBMC) subsets from patients with psoriatic arthritis (PsA).

Background: Delphinidin, a dietary anthocyanin and powerful anti-oxidant from pigmented fruits and vegetables, has broad anti-inflammatory properties. In a human skin model of psoriasis, delphinidin reduced expression of proliferative and inflammatory markers (1).

Objectives: The rationale of our study was to assess whether delphinidin can in vitro suppress IL-17 and IFN-γ production in peripheral blood mononuclear cell (PBMC) subsets from patients with psoriatic arthritis (PsA).

Methods: PBMCs were obtained from 24 patients with PsA attending the outpatient clinic of the Department of Rheumatology/clinical Immunology at the University General Hospital of Larissa, Greece. 16 age- and sex-matched healthy volunteers were also included in the study. Delphinidin was supplemented at a concentration ranging from 1 to 50μg/ml, one hour prior to cell stimulation. Cell viability (Annexin V staining) and innate/adaptive lymphocyte subpopulations were assessed by flow cytometry with a panel of fluorochrome-conjugated antibodies against CD56, CD3, CD4 and CD8. Intrauterine expression of IL-17 and IFN-γ was measured following PMA/ionomycin stimulation for 5 hours using standard cell permeabilization protocols and monoclonal antibodies against IL-17 and IFN-γ.

Results: Delphinidin at concentration ≥10 μg/ml sharply diminished IL-17 production by CD4+ T cells (Th17) and CD56+(CD3+) (NKT) cells from patients with psoriatic arthritis and normal controls (p<0.05). IFN-γ producing T (CD4 and CD8) cells, as well as NK and NKT cells were also dose-dependently suppressed following delphinidin pre-incubation in both patients and healthy controls (UHC). The concentration of IFN-γ+ cells ranged from 2.7 to 50 μg/ml. The inhibitory effect of delphinidin on IL-17 and IFN-γ producing lymphocytes was not due to compromised cell viability, as assessed by annexin V binding.

Conclusion: Delphinidin exerts, in a dose-dependent manner, a profound in vitro inhibitory effect on T cell and NKT cell IL-17 and IFN-γ production in PsA, and therefore, it may be used as a dietary immunosuppressant, complementary to standard treatment.

References:

Disclosure of Interests: A. Mavropoulos1, S. Tsigkas2, D. Skyvalidas3, C. Liakos3, A. Roussaki-Schulze1, E. Zafiriou1, D. Bogdanos1, L. Sakas2, University of Thessaly, Rheumatology and clinical Immunology, Faculty of Medicine, Larissa, Greece; 2University of Thessaly; Dermatology, Faculty of Medicine, Larissa, Greece

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DELPHINIDIN DOSE-DEPENDENTLY DIMINISHES PERIPHERAL IL-17 AND IFN-γ PRODUCING LYMPHOCYTES IN PSORIATIC ARTHRITIS

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ASSOCIATION OF GUT DYSBIOSIS WITH STRUCTURAL DAMAGE AND ACTIVITY IN AXIAL SPONDYLOARTHRITIS PATIENTS. DATA FROM THE COSPAR REGISTRY.