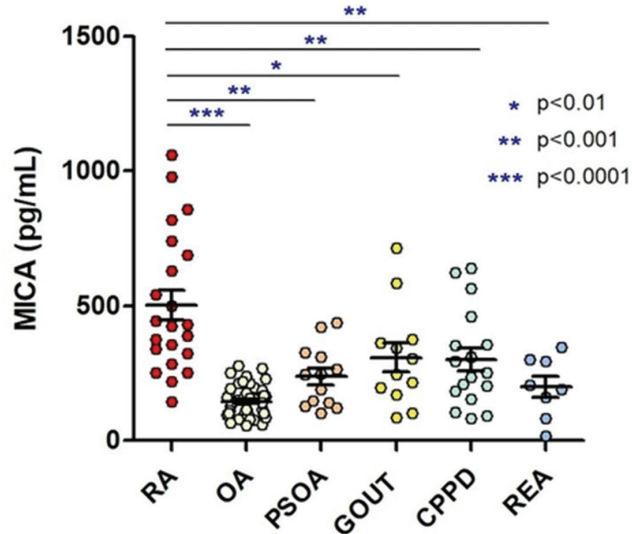


Pyrophosphate Deposition Disease (CPPD), 8 Reactive Arthritis (REA) and 45 Osteoarthritis patients. Gout and CPPD diseases were confirmed by the presence of crystals in SF. Clinical data were collected. The concentration of soluble MIC-A (sMIC-A), interleukin (IL)-1, IL-6 and IL-8 was measured by ELISA. Murine Rae-1, H60 and Mult1 transcripts were quantified by real-time quantitative PCR (RT-qPCR) in 3 models of joint inflammation: Serum Transfer Arthritis (STA), Collagen-induced arthritis (CIA) and Collagenase-Induced Osteoarthritis (CIOA).

Results: Significantly higher levels of sMIC-A were found in the synovial fluid of RA patients in comparison with all others diseases ($p < 0.001$, figure n.1). sMIC-A levels were correlated to white blood cell counts and levels of inflammatory cytokines IL-1, IL-6 and IL-8. Similarly, higher expression levels of Rae-1, H60 and Mult1 were found in chronic arthritis mouse models in comparison with osteoarthritic mice.



Abstract AB0069 – Figure 1

Conclusions: Our data identifies synovial sMIC-A as an important player in rheumatoid arthritis compared to other rheumatic diseases and osteoarthritis. Investigations in mouse models are in agreement with this finding

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AB0070 ADIPOKINES AND CYTOKINES IN THE PATHOGENESIS OF PSORIASIS AND METABOLIC DISORDERS

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Background: Psoriasis is a systemic immune-associated disease with a specific comorbidity. The manifestations of the metabolic syndrome in this category of

patients are changes that develop on systemic immune-associated inflammatory psoriatic process background and contribute to the progression of chronic inflammation.

Objectives: The aim of the study was determination of the main adipokines and cytokines content in the serum of peripheral blood, severity and activity of the disease in patients with psoriasis; clinical and laboratory evaluation of metabolic disorders. The interconnection between the production of adipokines and cytokines in psoriasis was analysed depending on the severity and activity of the psoriatic process and the nature of metabolic disorders.

Methods: Serum levels of adipokines (C-peptide, ghrelin, insulin, glucagon, leptin, visfatin, resistin, GIP, GLP-1 and PAI-1) and cytokines (IL-1 β , IL-1ra, IL-2, IL-4, IL-5, IL-6, IL-7, IL-8, IL-9, IL-10, IL-12 (p70), IL-13, IL-15, IL-17, eotaxin, FGF-2, G-CSF, GM-CSF, IFN- γ , IP-10, MCP-1, MIP-1 α , MIP-1 β , PDGF-BB, RANTES, TNF- α and VEGF) were measured in 36 patients with moderate and severe psoriasis, psoriatic arthritis. There was control group of 15 basically healthy persons. Clinical and laboratory evaluation of metabolic disorders (BMI, dyslipidemia, carbohydrate metabolism disorders) and cardiovascular diseases was performed for all examined patients. The duration, severity and the amount of body surface area involved in psoriasis were evaluated in all patients using recommended indices (BSA, PASI).

Results: Patients with psoriasis showed an increase in the production of glucagon, leptin, visfatin, GLP-1 ($p < 1.0E-03$) and a decrease in the level of C-peptide, insulin, GIP, PAI-1, resistin ($p < 1.0E-06$) compared with the control group. The difference of ghrelin concentrations in both groups was not statistically significant. Patients with psoriasis showed an increase in the level of IL-1 β , IL-1ra, IL-2, IL-4, IL-5, IL-6, IL-8, IL-9, IL-10, IL-12 (p70), IL-13, IL-15, IL-17, eotaxin, FGF-2, GM-CSF, IFN- γ , IP-10, MCP-1, MIP-1 β and TNF- α compared to control group ($p < 0.01$). Positive correlations between the level of adipokines and cytokines were revealed.

Conclusions: The obtained data allow us to define adipokines as mediators between immune and endocrine systems. The imbalance between the proinflammatory and anti-inflammatory effects of adipokines observed in psoriasis demonstrates lipid metabolism dysfunction as one of the possible provoking factors of chronic inflammation determining the severity of the underlying disease. According to the results of the study, the following biological markers of severity of the psoriatic immune-associated inflammatory process, with all its comorbid risks: GLP-1, glucagon, leptin, visfatin, IL-1ra, IL-2, IL-4, IL-5, IL-6, IL-7, IL-8, IL-12, IL-13, IL-17, eotaxin, FGF-2, GM-CSF, IFN- γ , IP-10, MCP-1, MIP-1 β , TNF- α . The levels of adipokines and cytokines are probably the earliest biological markers in patients with metabolic syndrome and psoriasis, the control of adipokines and cytokines level can be used to optimise therapy.

Disclosure of Interest: None declared

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AB0071 EFFECTS OF CHONDROITIN SULPHATE AND GLUCOSAMINE ON INFLAMMATORY CYTOKINES IN MACROPHAGES

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Background: The combination of pathogen-associated molecular patterns (PAMPs), such as lipopolysaccharide (LPS), and endogenous danger-associated molecular patterns (DAMPs), such as hyaluronan (HA) fragments, are known to be able to induce an inflammatory response from macrophages characterised by the release of pro-inflammatory cytokines¹. We have previously shown that pharmaceutical grade chondroitin sulphate (CS), commonly used in the symptomatic treatment of osteoarthritis (OA), can attenuate the inflammatory response.

Objectives: To evaluate the synergistic effects of CS and glucosamine (GLU) in combination on LPS/HA mediated inflammatory responses of an immortalised human macrophage cell line (THP-1) and primary synovial fluid cells.

Methods: THP-1 monocyte cells were grown and differentiated into mature macrophages by the addition of 200 nM of phorbol 12-myristate 13-acetate (PMA) as previously described¹. Cells were cultured with a physiologically achievable range of concentrations of CS and GLU (0, 10, 50, 200 μ g/ml of each, Bioibérica, S.A. U.) for 6 hours, then primed with physiologically relevant concentrations of LPS (10 ng/ml) (n=12/group). After 24 hours, cell culture media were replaced with serum free Opti-MEM supplemented with the previously mentioned concentrations of CS and GLU, LPS, and 10 μ g/ml HA fragments (ultra-low molecular weight, Lifecore). After a further 24 hours, supernatants were harvested for NF- κ B activity and pro-inflammatory cytokine (IL-1 β , IL-6, IFN- γ , and TNF- α) assessment. Cell viability was measured using PrestoBlue reagent. The human knee