Objectives: To identify HLA alleles associated with susceptibility to develop an IMID in Paraguayan patients controlled in a reference centre.

Methods: Paraguayan IMID patients were recruited from the Rheumatology Department of Hospital de Clínicas, Paraguay. IMID HLA II frequencies were compared with a control group of 50 unrelated individuals without disease and from the same geographic origin. Genotyping for HLA was performed using LumineX PCR technology. The association analysis with the IMIDs risk was performed using the chi-square allelic test.

Results: 249 IMID patients (95 lupus, 104 rheumatoid arthritis and 50 systemic sclerosis) were included. Of these 84.4% were women with an average age of 43.4 (± 14). Comparing the haplotypes profiles for the 5 HLA class II genes between the patients and the healthy controls, in the risk association analysis, the association of the known risk allele was corroborated HLA-DRB1*03:01 ($p=2e-06$, OR:14.97). A significant association was identified between the allele HLA-DRB1*08:02 ($p=0.0271$, OR:0.13) and HLA-DRB1*08:07 ($p=0.0133$, OR:0.08). In the gene *HLADQA1*, 1 allele associated with the IMIDs were found, the HLA-DQA1*04:01 ($p=1.4e-05$, OR:0.06). In the *HLADPB1* gene 3 alleles associated with the IMIDs were identified: HLA-DPB1*02:01 ($p=4.2e-05$, OR:82.91), HLA-DPB1*03:01 ($p=2e-06$, OR:14.97), HLA-DPB1*04:01 ($p=1.5e-05$, OR:34.55). Different associations between IMIDs and alleles was identified (table 1).

Abstract AB0005 – Table 1. List of associated alleles stratified by disease

ALLELE (Systemic Lupus Erythematosus Cohort)	P-value	OR
HLADQA1*02:01	0.0253	14
ALLELES (Rheumatoid Arthritis Cohort)	P-value	OR
HLADRB1*08:02	0.0367	0.11
HLADPB1*02:01	0.0003	54.62
HLADPB1*03:01	0.0010	7.92
HLADPB1*04:01	0.0001	29.69
HLADQA1*02:01	0.0467	0.24
HLADQA1*04:01	0.002	0.07
HLADPA1*02:01	0.0022	0.029
ALLELES (Scleroderma Cohort)	P-value	OR
HLADPB1*02:01	0.0003	82.33
HLADPB1*03:01	0.0094	8.44
HLADPB1*04:01	0.0001	47.50
HLADQA1*04:01	0.0013	0.06

Conclusions: In the genetic association analysis, already known associations have been replicated and new ones previously unpublished have been identified in Paraguayan IMIDs patients. This is the first genetic association study in IMID patients Paraguayan origin.

Disclosure of Interest: None declared

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AB0006 MIR-26A POLYMORPHISM IS ASSOCIATED WITH SUSCEPTIBILITY OF RHEUMATOID AND PSORIATIC ARTHRITIS

J. Swierkot¹, R. Sokolik¹, M. Kozłowski², L. Korman¹, P. Wiland¹, K. Bogunia-Kubik². ¹Department of Rheumatology and Internal Medicine, Medical University; ²Laboratory of Clinical Immunogenetics and Pharmacogenetics, Hirsfeld Institute of Immunology and Experimental Therapy, Polish Academy of Sciences, Wrocław, Poland

Background: Serum levels of miR-26a has been reported to act as potential biomarker of rheumatic diseases.

Objectives: The aim of the study was to analyse the genetic variation and expression of miR-26a as potential diagnostic and/or prognostic markers of rheumatoid diseases.

Methods: The miR-26a polymorphism was examined in 111 patients with rheumatoid arthritis (RA), 86 patients with psoriatic arthritis (PsA) and 162 healthy blood donors that served as a control group. Genotyping for miR-26a rs7372209 was performed using a LightSNiP assay. For analysis of the miR-26a expression, RNA was isolated from sera of 15 RA patients (before and 3 months after anti-TNF treatment) and 10 controls (Nucleospin-miRNA Plasma; MACHERY-NAGEL GmbH and Co. KG) followed by cDNA synthesis (TaqMan MicroRNA Reverse Transcription Kit; Applied Biosystems™ by Life Technologies) and Real-time PCR amplifications with hsa-miR-26a TaqMan specific and U6 snRNA control primers for each probe. The results were analysed using the ($\Delta\Delta C_t$) calculations.

Results: It was found that the presence of miR-26a TT genotype (rs7372209) more than 5 times increases the risk of RA (OR=5.28, $p=0.003$) while the presence of CC homozygotes is associated with the risk of PsA (OR=1.77, $p=0.037$).

There was no significant difference in the miR-26a serum levels between patients and controls. Also miR-26a serum levels did not significantly differed between RA patients before, 3 and 6 months after the implementation of biological therapy with TNF-alpha inhibitors.

Conclusions: These results imply that miR-26a rs7372209 allelic variants differentially affect the risk of rheumatoid and psoriatic arthritis while anti-TNF biological treatment seems not to affect the miR-26a expression in RA patients.

Disclosure of Interest: None declared

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AB0007 ASSOCIATION AT SYSTEMIC LEVELS OF CYTOKINE MRNAS AND PROTEIN QUANTITIES IN RHEUMATOID ARTHRITIS

I. Manolova¹, M.G. Ivanova², G. Vasilev³, R. Stoilov², L. Miteva¹, S. Stanilova¹.

¹Department of Molecular Biology, Immunology and Medical Genetics, Medical Faculty, Trakia University, Stara Zagora; ²Clinic of Rheumatology, University Hospital "St. Iv. Rilski", Medical Faculty, Medical University; ³Laboratory of Clinical Immunology, University Hospital "St. Ivan Rilski", Medical Faculty, Medical University, Sofia, Bulgaria

Background: Rheumatoid arthritis (RA) is an autoimmune inflammatory disease, characterised by chronic synovitis, bone and cartilage destruction, as well as systemic manifestation. In accordance with the pivotal role of cytokines in autoimmunity and their impact as biomarkers, we analysed gene expression at both mRNA and protein levels of several cytokines in peripheral blood of RA patients.

Objectives: The aim of the present study was to investigate the gene expressions at mRNA and protein levels of main pro-inflammatory (TNF- α , IL18, IL-12p40; IL-23, IL-6) and immunosuppressive (TGF- β 1 and IL10) cytokines and transcription factor Foxp3 in peripheral blood of RA patients.

Methods: Total RNA from peripheral blood was isolated from 32 patients matching the ACR/EULAR 2010 criteria for RA and 27 healthy controls. Quantitative real-time polymerase chain reaction was performed for the 8 genes of interests, using the TaqMan detection system. Relative quantitative evaluation of mRNAs was performed by the comparative $\Delta\Delta C_t$ method and results were presented as n-fold mean difference (RQ-relative quantity) of target genes relative to calibrator (healthy controls) after normalisation to the reference genes (GAPDH and 18srRNA). Serum quantities of cytokines were measured by ELISA.

Results: From studied pro-inflammatory cytokine genes, we found down-regulation in the following order: IL6 > TNFA > IL12B; up regulation of IL23A and no change in IL18 gene expression in RA patients group compared to healthy controls. For anti-inflammatory genes we detected significantly increased quantity for IL10 mRNA and no change for TGF β 1 mRNA. The most profound down-regulation (more than 7-fold) was observed for IL6 gene ($p<0.001$), while the serum level of the same cytokine was significantly increased as compared to the same controls. The similar tendency was observed in the expression of TNFA, which gene expression was approximately 2-fold down-regulated, whereas serum levels were increased. IL12B mRNA were slightly but not significantly decreased (RQ=0.709; $p=0.169$) in RA patients. An upregulation of IL-23 was detected for IL23A gene (RQ=2.422, $p=0.002$) and serum level of IL-23 as well.

TGF β 1 mRNA levels were approximately equal in patients and controls in contrast to IL-10, which was upregulated in both mRNA (RQ=1.6; $p=0.034$) and serum levels (over 6.6 fold; $p<0.05$). In addition, mRNA expression of FoxP3, a master transcription factor for Treg subset was also down-regulated over 4-fold in RA patients ($p<0.001$). A positive correlation was found between gene expression of IL6 with Foxp3, TNFA and TGF β 1 in RA ($r=0.744$, $p=0.004$; $r=0.6$, $p=0.03$; $r=0.556$, $p=0.048$, respectively).

Conclusions: Our results demonstrated significant differences in the expression of mRNA encoded cytokines and their protein quantities at systemic level of RA patients, mostly on IL-23, IL-6 and TNF-alpha.

Disclosure of Interest: None declared

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AB0008 CROSS-TALK BETWEEN BONE TURNOVER AND CARDIOVASCULAR DISEASE. ASSOCIATION OF MICRORNAS EXPRESSION, FRACTURE AND ABDOMINAL AORTIC CALCIFICATIONS

M.-E. Pickering^{1,2}, M. Croset^{1,2}, M. Millet^{1,2}, E. Somay-Rendu^{1,2}, J.-C. Rousseau^{1,2}, O. Borel^{1,2}, P. Szulc^{1,2}, R. Chapurlat^{1,2}. ¹Hospices Civils de Lyon; ²INSERM UMR 1033, Lyon, France

Background: MicroRNAs (miRs) have emerged as pivotal epigenetic key actors of gene regulation and several miRs have been shown to be at the crossroads of angiogenesis and of bone turnover, taking part in the calcification process by acting on osteoblasts and osteoclasts.¹ Calcification of the aortic media is highly