

attribute to increased risk of cancers can be genomic instability and impaired DNA repair.

Objectives: The aim of the study was to assess the processes of endogenous and exogenous DNA damage and its repair in patients with IIM as compared to healthy controls.

Methods: The study included 10 patients (9 men and 1 woman, mean age 46.6 ± 16.5) with idiopathic inflammatory myopathies (dermatomyositis or polymyositis) as well as 7 healthy control individuals (4 men and 3 women, mean age 33.8 ± 9.8). DNA damage and repair were investigated by the comet assay. To perform the comet assay human peripheral blood mononuclear cells (PBMCs) were isolated and incubated with tert-butyl hydroperoxide (t-BOOH) or bleomycin. Both compounds are common DNA damaging agents – t-BOOH induces oxidative DNA lesions whereas bleomycin induces also DNA double strand breaks (DSBs). To test the DNA repair capability, PBMCs were allowed to recover for 2 hour. The level of endogenous DNA lesions was also investigated.

Results: The levels of endogenous DNA damage were not statistically significantly different between tested groups (IIM-3.3±3.6% vs 3.2±3.8% in control; $p=0.648$). The extent of the DNA damage induced by bleomycin (IIM-23.3 ± 19.5% vs 9.8±5.9% in control) as well as oxidative stress (IIM-14.7±16.2% vs 10.4±7.4% in control) was significantly higher in PBMCs derived from IIM patients than in healthy counterparts ($p<0.001$). Kinetic curves of DNA repair are different but the background mechanism underlying observed differences in the repair curve between healthy subjects and patients need to be evaluated further.

Conclusions: Understanding the etiology of this phenomena in these diseases may provide insight into disease pathogenesis and explain the increased susceptibility of patients to malignancies. Finding the patients with increased DNA instability could potentially serve as a biomarker and indicate the group of patients who should be carefully screened for neoplasms disorder.

Disclosure of Interest: None declared

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AB0011 EVALUATION OF SALIVARY MIRNAS IN PATIENTS AFFECTED BY SJÖGREN'S SYNDROME AND CORRELATION WITH CLINICAL AND ULTRASONOGRAPHIC OUTCOMES

R. Talotta¹, M. Biasin², S. Bongiovanni¹, V. Mercurio², C. Vittori², L. Boccassini¹, F. Rigamonti¹, A. Batticciotto¹, F. Atzeni³, D. Trabatonfi², P. Sarzi-Puttini¹.
¹Rheumatology Unit, ²Biomedical and Clinical Sciences, University Hospital "Luigi Sacco", Milan; ³Rheumatology Unit, AOU "G.MARTINO", Messina, Italy

Background: It has been demonstrated that miRNAs expressed in PBMCs and salivary glands of patients affected by Sjögren's Syndrome (SS) could be involved in the epigenetic control of the disease.

Objectives: We aimed to compare the concentration of miRNA-146a/b, 16, 17–92 cluster and 181a in salivary and plasmatic samples collected from SS patients and healthy controls and to evaluate the associations with clinical, laboratory and ultrasound findings.

Methods: We collected plasmatic and salivary samples from 28 patients (27 females, mean age 64.4±10.1 years, mean disease duration 10.7±6.9 years) affected by primitive SS according to ^{ACR} 2012 and/or 2016 criteria and 23 matched healthy controls. In the group of patients, the following data were recorded: ESS-DAI and ESSPRI scores, anti-SSA and anti-SSB status and laboratory data, Schirmer's test, ultrasound scores of the four major salivary glands according to Cornec et al. and concomitant treatments. The following miRNA were extracted, retro-transcribed and quantified: miR16–5 p, miR17–5 p, miR18a–5p, miR19a–5p, miR19b–1–5 p, miR20a, miR92–5 p, miR146a–5p, miR146b–5p, miR181a–5p.

Results: The concentration of miRNAs evaluated in plasma and saliva did not significantly match both in patients and in controls, underlining a different modulation in their expression according to the corporeal district. In patients and controls miRNAs16, 17 and 20 were hyper-expressed and miRNA146b and 181 hypo-expressed in salivary samples compared to plasmatic ones. Salivary miRNAs 18a and 146a were hyper-expressed in patients and hypo-expressed in controls, when compared to plasmatic concentrations.

Comparing salivary and plasmatic patients' miRNAs concentrations to those of healthy subjects, plasmatic miRNA16 and 181a were significantly more expressed in patients than in controls (two-tailed Wilcoxon test and Student's T test for unpaired samples).

In SS patients, salivary miRNA 181 and 146a were significantly increased in older subjects ($p=0.01$ and $p=0.04$ respectively). Spearman's correlation revealed that salivary miRNA146b was significantly hyper-expressed in patients with worse ESSPRI scores ($p=0.02$); on the contrary, salivary miRNA17, 146b and plasmatic miRNA17 were reduced in patients with higher scores at ultrasound evaluation ($p=0.01$, $p=0.01$ and $p=0.04$ respectively). Plasmatic concentration of miRNA18a was increased in patients with less lachrymal production at Schirmer's test ($p=0.01$) and plasmatic concentration of miRNA17 was reduced in patients with

higher ESR values ($p=0.01$). Salivary miRNA18a was significantly increased in patients with anti-La/SSB ($p=0.04$; Mann-Whitney U test for unpaired samples). Salivary and plasmatic miRNAs did not correlate with disease duration and concomitant therapies.

Conclusions: Our data show that the expression of salivary miRNAs 17, 18a and 146b may be altered in SS patients and associated with worse ultrasound and ESSPRI scores and anti-La/SSB positivity.

Disclosure of Interest: None declared

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AB0012 ALLELE AND GENOTYPE FREQUENCY OF SOME GENE POLYMORPHISMS DIFFERS IN VARIOUS JUVENILE IDIOPATHIC ARTHRITIS SUBTYPES

H.A. Yatskiu¹, N.V. Nikitchenko¹, N.V. Savina¹, T.D. Kuzhir¹, A.M. Tchitchko², I. D. Chyzhetskaya³, N.L. Bilskaaya⁴, M.G. Myslivets⁵, A.V. Sukalo², R. I. Goncharova¹. ¹Institute of Genetics and Cytology of NAS of Belarus; ²Belarusian State Medical University; ³4th City Children's Clinical Hospital, Minsk; ⁴Gomel Regional Children's Clinical Hospital, Gomel; ⁵Grodno Regional Children's Clinical Hospital, Grodno, Belarus

Background: In spite of the fact that chronic arthritis has been the core of paediatric rheumatology, etiology and pathogenesis of juvenile idiopathic arthritis (JIA) are still unclear. It is important to ascertain genetic nature of such a heterogeneous disease.

Objectives: The aim of the study was to assess the role of some SNPs of six genes implicated in immune and inflammatory responses: *TNFA* (rs1800629, rs361525), *PTPN22* (rs2476601), *MIF* (rs755622, rs5844572), *TRAF/C5* (rs3761847), *CTLA4* (rs5742909, rs231775), *STAT4* (rs7574865); as well as homozygous deletions of xenobiotic biotransformation genes *GSTT1* and *GSTM1*.

Methods: 206 patients diagnosed with JIA (mean age 8.87±4.92), and 218 hospital controls with no signs of autoimmune or inflammatory diseases (mean age 13.88±2.72) were recruited for the study. The JIA patients were divided into subgroups according to IAR classification criteria; the majority of them (125 patients) had oligoarthritis, 42 children developed RF-negative polyarthritis and 27 patients were diagnosed with systemic arthritis. Genomic DNA was extracted from peripheral blood samples by means of the phenol-chloroform method. SNPs were genotyped using PCR-RFLP, Real-Time PCR or fragmental analysis.

Results: The allele frequencies for all SNPs in the hospital control group were similar to those in European populations. The allele and genotype frequency distribution for all SNPs was identical in patients and controls. However, when comparing distinct subtypes of JIA, *STAT4* polymorphism demonstrated higher frequencies of minor T allele (30.5% vs. 17.8%, $p=0.01$, OR=2.03, 95% CI [1.14–3.6] and G/T genotype (46.3% vs. 25.6%, $p=0.03$, OR=2.51, 95% [1.2–5.24]) in RF-negative polyarthritis than in oligoarthritis. On the contrary, Δ *GSTT1* appeared to prevail in oligoarthritis (23.7% vs. 7.9% in RF-negative polyarthritis, $p=0.03$, OR=3.6, 95% CI [1.03–12.7]). As to systemic arthritis, it was shown, that minor G allele of *TRAF/C5* was more frequent in comparison with oligoarthritis ($p=0.037$, OR=2.43, 95% CI [1.05–5.6]).

Conclusions: The results obtained can be considered as evidence for distinct genetic nature of the different JIA subtypes.

Disclosure of Interest: None declared

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AB0013 EXPRESSION LEVELS OF MIR-124 IN THE PLASMA OF RHEUMATOID ARTHRITIS PATIENTS

R. Shumnalieva¹, D. Kachakova², V. Shumnalieva-Ivanova³, P. Miteva⁴, R. Kaneva², Z. Kolarov¹, S. Monov¹. ¹Department of Internal Medicine, Clinic of rheumatology, ²Molecular Medicine Center, Department of Medical Chemistry and Biochemistry, Medical University – Sofia, ³University Hospital "Alexandrovska", Medical University – Sofia, Bulgaria, ⁴University Hospital "Prof. Ivan Mitev", Medical University – Sofia, Bulgaria, Sofia, Bulgaria

Background: Micro-ribonucleic acids (microRNAs) comprise a class of small non-coding RNAs that regulate gene expression on post transcriptional level. Levels of miR-124 have been found to be decreased in rheumatoid arthritis (RA) synoviocytes and *in vivo* studies have shown that treatment with pre-miR-124 suppresses the progression of joint damage¹.

Objectives: To evaluate the expression levels of miR-124–3 p in plasma of RA patients and to determine its possible role as biomarker for diagnosis and disease activity.

Methods: 34 RA patients according to the 1987 ACR criteria were included in the study. Expression levels of miR-124–3 p in the plasma were determined by PCR