terms. In the screening for genes regulating maturation of Th1/Th17 cells, CBLB, IRF1, STAT3, SGK1 and TNFSF14 were identified among the genes enriched in RA samples, while EGR3 and ETS1 were enriched only in healthy controls. Additionally, transcription factors Ezh2, Rad21, Ctbp2 and Suz12 were identified as common for both RA and healthy groups genes, associated with significant GO enrichment.
Conclusions: This study confirms the role of survivin as a transcription mediator in CD4 +T cells and is suggested to influence multiple genes involved in RA pathology.

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AB0017 VITAMIN D LEVEL AND MIRNA 22 AND 125B GENE EXPRESSION IN BEHÇET'S DISEASE PATIENTS

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Objectives: To investigate serum levels of vitamin D and its effect on microRNA 22 and 125b gene expression in Behçet's disease (BD) patients and to evaluate their relation to clinical characteristics and disease activity.
Methods: Fifty-one BD patients and 45 matching controls were studied. Disease activity was assessed using BD Current Activity Form (BDCAF). Serum vitamin D3 was measured by ELISA. MicroRNAs were assayed by reverse transcription-quantitative polymerized chain reaction (RT-qPCR).
Results: Patients mean age was 34.3±8.6 years and disease duration 65.3±52.4 months. The BDCAF was 2.4±1.3. Vitamin D level was significantly lower (29.7 ±14.4 ng/ml) in patients than in control (40.1±17.8 ng/ml)($p<0.001$) especially males (25.8±7.5 ng/ml) compared to females (48±23.7 ng/ml)($p<0.001$). There was no relation between 25(OH)D levels and disease activity or with the presence of clinical manifestations. There was a 3.8±1.5 fold increase in miRNA125b while miR-22 showed no significant difference (0.38±0.46) but was significantly reduced in those receiving steroids (0.21±0.27) compared to those not (0.66±0.58) ($p=0.003$). There was no significant difference in the frequency of miR-22 or miR-125 expression according to the presence of clinical manifestations, medications received or disease activity. The fold change in miR-125b significantly correlated with vitamin D ($r=0.54$,$p<0.001$) but not with BDCAF ($p=0.64$).
Conclusions: Vitamin D level is decreased in BD patients and significantly correlated with the fold change of miR-125b especially in males thus representing a possible therapeutic target. The miR-22 expression did not change but was notably downregulated by steroids. Further longitudinal studies on a larger sample are recommended to validate the present results.
Disclosure of Interest: None declared
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AB0018 CLINICAL STATUS AND GENE EXPRESSION IN CLASS IV LUPUS NEPHRITIS

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Background: Lupus nephritis (LN) is one of the most frequent and serious complications in the patients with systemic lupus erythematosus. Several studies have identified risk factors for poor kidney prognosis in patients with SLE, including age, sex, hypertension, decreased estimated GFR (eGFR), proteinuria, and renal pathologic types.[1] Biopsy allows classifying the type of renal involvement, assessing its activity, and thus guiding the therapeutic behaviours. It has been shown that structural changes and inflammatory infiltrate associated with LN contribute to a hypoxic state which induces angiogenesis.[2] Herein, it was hypothesised that differential expression of angiogenic genes could classify Lupus Nephritis Patients (LNP) with the same histological score but different "clinical status".
Objectives: To investigate if there is a differential angiogenic gene expression in biopsies of LNP under the same histological classification but different "clinical status" measured by eGFR.
Methods: Twenty four kidney biopsies samples classified according to ISN/RPS scoring system as Class IV from 24 LNP were divided into eGFR <60 ml/min (n=10, age: 31.00±10.93, range: 17–46) and eGFR >60 ml/min (n=14, age: 32.64 ±11.34, range: 21–64). RNA was isolated using TRIzol-Chloroform technique and then was reverse-transcribed using random primers. Gene expression level of pro-angiogenic factors: VCAM-1, VEGF, TGF-β and ANGPT-1 were evaluated using Quantitative Real Time PCR (QPCR). The threshold cycle (Ct) scores were averaged for calculations of relative expression values. The Ct scores were normalised by subtracting β2Microglobuline (β2M) control, or ΔCt=Ct, gene- Ct,β2M. To test for differential gene expression between groups, a two sample T-test was performed to compare the ΔCt in the two groups.
Results: ΔCt is inversely proportional to the gene expression level. Significant differences between groups was found in VEGF-A gene ($p=0.0326$), where the greatest expression corresponding to eGFR <60 ml/min group. However, there were not statistically significant differences in VCAM-1, ANGPT1 and TGF-β expression (table 1). Particularly TGF-β, a proangiogenic and profibrotic gene showed a uniform expression level in both groups.

Abstract AB0018 – Table 1. Levels of gene expression and laboratory parameters in eGFR <60 ml/min and eGFR >60 mil/min expressed as ΔCt values.

Gene	eGFR<60 ml/min	eGFR>60 ml/min	p value
	n=10	n=14	
VCAM-1	5,021±1768	6,248±3264	0,3165
VEGF-A	3,414±1161	4,651±1409	0,0326
TGF-β	6,168±1011	6,699±1351	0,3421
ANGPT1	10,330±2294	9,891±2113	0,5007

Conclusions: In the present cross-sectional study, increased levels of VEGF-A were observed in biopsies Class IV from LNP with eGFR <60 ml/min. These findings suggest a differential gene expression that may be associated with an impaired renal function, reflected by eGFR.

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AB0019 THE ROLE OF THE IL2-IL21 RS6822844 POLYMORPHISM IN THE PREDISPOSITION TO THE ERYTHROCYTE SEDIMENTATION RATE ELEVATION IN JUVENILE IDIOPATHIC ARTHRITIS

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Background: Juvenile idiopathic arthritis (JIA) is the most common chronic rheumatic disorder in paediatrics.[1] The erythrocyte sedimentation rate (ESR) is one of the key measures of the JIA activity.[2] However, in some patients, even in the active disease period, the ESR is not elevated.[3]
Objectives: The aim of the study was to assess the relationship between the *IL2-IL21* rs6822844 locus polymorphic variants and a predisposition to the ESR elevation in JIA patients.
Methods: The study included 255 JIA patients from the Republic of Bashkortostan, Russia. The ESR was considered elevated if its value exceeded the upper limit of the normal range two or more times. Genotyping was performed by real-time PCR, statistical analysis – using the two-tailed Fisher exact test (p) and the odds ratio (OR) with a 95% confidence interval (CI).
Results: The girls/boys ratio was 65.88%/34.12%. The ESR elevation in the active disease period was seen in 70.98% of patients. When studying the *IL2-IL21* rs6822844 polymorphic locus, a marginal significance level was noted for the rarer occurrence of the GT genotype in patients with the elevated ESR (14.92%

vs. 25.68%, p=0.049, OR=0.508, 95% CI 0.258–0.985, compared with patients with the normal ESR, respectively). After stratification according to the sex, similar differences in the GT genotype frequencies were seen and even intensified only for female JIA patients (15.38% vs. 31.37%, respectively, p=0.022, OR=0.398, 95% CI 0.180–0.897). In addition, a significant increase in the GG genotype proportion and a tendency towards a decrease in the T allele proportion were observed in girls with the elevated ESR (GG: 83.76% vs. 68.63%, p=0.038, OR=2.358, 95% CI 1.058–5.110; T: 8.55% vs. 15.69%, p=0.057, compared with girls with the normal ESR, respectively).
Conclusions: In the present study, the association of the *IL2-IL21* rs6822844 locus polymorphic variants with a predisposition to the ESR elevation in female JIA patients was shown.

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AB0020 DIFFERENTIALLY EXPRESSED GENES IN SJGREN'S SYNDROME MICROARRAY

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Background: Sjogren's syndrome (SS) is a chronic autoimmune disease characterised by a decrease in the secretion of tears and saliva, and is considered to be a common rheumatic immuno disease second only to rheumatoid arthritis. Patients with SS may have different prognosis or different reactions to the same treatment plan. Therefore, a more accurate and more practical predictor is needed to provide a basis for the individualised treatment of SS. Bioinformatics is a new approach. It applies bioinformatics methodology, tools, software and database to molecular mechanisms, new biomarkers and individualised treatment measures.
Objectives: In this study, gene expression data as the basis, combined with bioinformatics tools and literature mining methods, firstly analyses the expression differences between normal people and patients with parotid gland parotid Sjogren syndrome gene, a key pathway and further study the effect of SS involved in the occurrence and development of the application of protein interaction network screening key genes, pathogenesis help to clarify SS, and for the future of this disease and provide a new direction.
Methods: The expression profiles of mRNA in parotid gland of patients with Sjogren syndrome and normal parotid gland were obtained from the GEO database. A total of 24 patients with Sjogren's syndrome, 6 patients with dry mouth and dry eye symptoms and 25 patients without dry mouth and dry eye were enrolled in the study. 65 samples were collected. The GEO2R tool screened the parotid differentially expressed genes in the patients with SS compared with the healthy people, and the DAVID tool enriched the function and pathway of the gene. The STRING database constructs a network that differentially expresses the interaction of gene protein products and screening core genes.
Results: 24 upregulated genes and 147 down regulated genes were screened. KEGG enriched 5 pathways including cell adhesion, intestinal immune network IgA secretion, viral myocarditis pathway, rheumatoid arthritis pathway and leukocyte transendothelial migration. String protein interaction database was used to subcellular localization of differentially expressed gene protein products in parotid gland, and differentially expressed proteins were identified. The interaction network was constructed by Cytoscape software. The protein interaction network was constructed based on the 171 differentially expressed genes of SS, and the isolated and non interacting protein nodes were screened out. 108 upregulated gene encoded proteins were found to interact with each other, forming a complex network containing 390 interactions. The Degree>20 of the node is selected as the standard selection centre node, and 5 Hub genes are obtained. It is found that PTPRC, CD86, STAT1, FYN and LCP2 are key genes and may play an important role in the pathogenesis of Sjogren syndrome.
Conclusions: Above all, we used bioinformatics to find genes and critical pathways related to SS, which is expected to provide new molecular markers for the diagnosis and treatment of SS. It provides a new way of thinking for the treatment and prognosis of Sjogren syndrome.
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AB0021 A GENOME-WIDE SNP LINKAGE ANALYSIS SUGGESTS A NOVEL SUSCEPTIBILITY GENE FOR ANKYLOSING SPONDYLITIS

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Background: Ankylosing Spondylitis (AS) is a chronic, progressive and inflammatory disease, which is considered to be hereditary. However, the responsible molecular genetic determinants remain unidentified.
Objectives: To detect susceptibility gene(s) for AS by using an affected-only linkage analysis and high density single nucleotide polymorphism (SNP) in genome-wide manner.
Methods: All AS patients in three families of Cantonese were recruited. Their clinical material were collected by questionnaires. Genomic DNA derived from individual peripheral blood leukocytes was genotyped using Illumina HuamHap 610-Quad SNP Chip. Genotype data were generated using the Illumina BeadStudio 3.2 software. An affected-only linkage analysis was carried out using non-parametric and parametric linkage analysis. The customised allele frequencies were based on the 980 Cantonese healthy controls. SNP genetic map positions were interpolated as their physical positions in megabyte.
Results: *1. Clinical data:* The mean age was 42.3±14.9 years (ranging from 18~62 years), mean age of onset was 23.8±7.4 years (ranging from 10~30 years), mean duration of affection was 17.0±13.0 years (ranging from 0.2~50.0 years), and the sex ratio of male to female was 2.5: 1. There was no Iritis and dactylitis, hip involvement (4, 19.05%), peripheral arthritis (4, 19.05%), inflammatory back pain (21, 100%) and HLA-B27 positive (20, 95.24%). *2. Results of non-parameter linkage analysis:* The highest LOD value was found in chromosome 16, which reached 2.362. Although chromosome 6 was considered to be relative to the pathogenesis of AS, its LOD value was 1.499 and the range of the peak was located in 6 p21, where 96 SNPs (such as rs6930977) were included. *3. Results of parameter linkage analysis:* The LOD value of chromosome 16 was 4.6807 and higher than that of other chromosomes which were less than 3 by the same analysis. A susceptibility locus was found in 16q12, spanning 88.5 Kb with LOD value above 3 (ranging: 51030764~51915940). *4. Susceptibility genes:* According to the result of parameter linkage analysis in chromosome 16, seven genes (TOX high mobility group box family member 3 (TOX3), LOC643714, LOC146253, LOC100132440, LOC390730, LOC100128523 and chromodomain helicase DNA binding protein 9 (CHD9)) could be detected in the position where the LOD value exceeded 3. Interestingly, six SNPs could be found in CHD9 gene. Likewise, they were also found in another association analysis, which included 400 AS patients and 977 healthy controls. P value for SNP rs10153130 was 0.005879 (adjust p≤0.05/6=0.00833).
Conclusions: Genome-wide SNP linkage analysis in three AS families supports that a susceptibility locus for AS was found in 16q12, spanning 88.5 Kb with LOD value above 3 (ranging: 51030764~51915940).
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AB0022 FAMILY-BASED WHOLE-EXOME SEQUENCING REVEALS THE GENETIC BASIS OF RELAPSING POLYCHONDRITIS

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Background: Relapsing polychondritis (RP) is a rare systemic disease, characterised by recurrent episodes of inflammation of cartilaginous tissues and other proteoglycan rich structures involving the cartilage of the ears, nose, larynx, tracheobronchial tree and cardiovascular system.[1 2] The susceptibility to RP has been reported to be significantly related to genetic factors.[3 4] However, family occurrence has yet to be reported and the responsible molecular genetic determinants hasn't been clearly elucidated.