Table 1. Genotype and allele frequencies of NCF2, NCF4, CYBA genes polymorphisms in RA patients and controls [n(%)]

SNP	Analyze model		RA (N = 593)	Control (N = 596)	<i>P value</i>
<i>NCF2</i>					
rs10911363	Genotypes	GG	125	144	0.371
		GT	304	289	0.747
		TT	164	163	
	Alleles	G	554	577	0.408
		T	632	615	
<i>NCF4</i>					
rs1883112	Genotypes	GG	57	56	0.972
		GA	248	255	0.754
		AA	288	285	
	Alleles	G	362	367	0.888
		A	824	825	
rs4821544	Genotypes	CC	4	7	0.323
		CT	117	146	0.043
		TT	472	443	
	Alleles	C	125	160	0.031
		T	1061	1032	
	Dominant model	TT	472	443	0.031
		CT+CC	121	153	
rs729749	Genotypes	TT	104	102	0.445
		CT	266	302	0.033
		CC	223	192	
	Alleles	T	474	506	0.219
		C	712	686	
<i>CYBA</i>					
rs3794624	Genotypes	AA	14	15	0.929
		GA	160	147	0.368
		GG	419	43	
	Alleles	A	188	177	0.498
		G	998	1015	
rs4673	Genotypes	AA	1	5	0.140
		GA	85	90	0.673
		GG	507	501	
	Alleles	A	87	100	0.34
		G	1099	1092	

Acknowledgments: no**Disclosure of Interests:** None declared**DOI:** 10.1136/annrheumdis-2020-eular.4278

AB0024

ASSOCIATION OF IL6 RS1800795 BUT NOT IL6R RS2228145, RS4845618 AND STAT4 RS7574865 POLYMORPHISMS WITH CHLAMYDIA-ASSOCIATED RHEUMATOID ARTHRITIS

T. Zybalo¹, V. Yagur¹, R. Goncharova², N. Soroka¹, N. Dostanko¹, V. Apanasovich¹, A. Tushina¹. ¹Belarusian State Medical University, 2-nd Department of Internal Medicine, Minsk, Belarus; ²Institute of Genetics & Cytology NAS Belarus, Laboratory of Molecular Basis of Genomic Stability, Minsk, Belarus

Background: Rheumatoid arthritis (RA), associated with Chlamydial Infection, has some clinical and immunological particulars that interfere with the early diagnosis and require significant changes in treatment strategy [1].

Objectives: To estimate the distribution of some non-HLA genetic markers such as STAT4 rs7574865, IL6 rs1800795, IL6R rs2228145 and rs4845618 in Chlamydia positive and negative RA patients and healthy controls.

Methods: We examined 380 healthy blood donors and 187 RA patients classified according to the ACR/EULAR 2010 criteria for RA [2]. Twenty-three of the RA patients were positive for Chlamydia trachomatis (n=17) or Chlamydia pneumonia (n=6) persistence. DNA from peripheral blood samples was extracted by phenol-chloroform method. SNPs were genotyped by the real-time PCR with fluorescent probes. Statistical significance of SNPs' frequency was estimated by two-way Fisher exact test (F , p_{2-1}) with Bonferroni correction for multiple comparisons (p_{cor}). Moreover, diagnostic odds ratio (dOR), the likelihood ratio of positive (LR^+) and negative (LR^-) tests and corresponding confidence intervals (CI) were calculated.

Results: We revealed statistically significant increase of genotype CC frequency (IL6 rs1800795) in Chlamydia-associated RA (60.9%) vs healthy donors (20.7%): $p_{2-1}=0.000065$; $p_{cor}=0.00026$; dOR=5.95 (CI_{95%} 2.53-13.94); $LR^+=2.94$ (CI_{95%} 1.90-3.29); $LR^-=0.49$ (CI_{95%} 0.28-0.75) as well as in Chlamydia-associated RA (60.9%) vs Chlamydia-negative RA (23.9%): $p_{2-1}=0.00051$; $p_{cor}=0.002$; dOR=4.99 (CI_{95%} 2.04-12.16); $LR^+=2.56$ (CI_{95%} 1.60-3.57); $LR^-=0.51$ (CI_{95%} 0.29-0.78). Significant differences in STAT4 rs7574865, IL6R rs2228145 and IL6R rs4845618 distribution between studied groups were not found.

Conclusion: Our data suggest the association between CC genotype of IL6 rs1800795 and Chlamydia-associated RA.

References:

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Disclosure of Interests: Tatiana Zybalo: None declared, Viktor Yagur: None declared, Roza Goncharova: None declared, Nikolaj Soroka Grant/research support from: JSC BIOCAD, Natalia Dostanko: None declared, Valery Apanasovich: None declared, Anastasiya Tushina: None declared

DOI: 10.1136/annrheumdis-2020-eular.5739

Adaptive immunity (T cells and B cells) in rheumatic diseases

AB0025

B-CELL SUBSETS AS ADDITIONAL DIAGNOSTIC TOOL FOR PRIMARY SJÖGREN'S SYNDROME AND SYSTEMIC LUPUS ERYTHEMATOSUS

S. Benevolenskaya¹, I. Kudryavtsev², M. Serebriakova³, I. Grigor'eva⁴, A. Budkova⁵, D. Zammoeva⁴, D. Motorin¹, A. Zaritsky⁴, S. Lapin⁵, A. Maslyanskiy⁴. ¹Almazov National Medical Research Centre, Saint-Petersburg, Russian Federation; ²Institute of Experimental Medicine, Saint-Petersburg, Russian Federation; ³Institute of Experimental Medicine, Saint-Petersburg, Russian Federation; ⁴Almazov National Medical Research Centre, Saint-Petersburg, Russian Federation; ⁵Pavlov First Saint Petersburg State Medical University, Saint-Petersburg, Russian Federation

Background: Systemic lupus erythematosus (SLE) and primary Sjögren's syndrome (pSS) are chronic complex disorders with an autoimmune background, multifactorial etiology, multiple circulating antinuclear antibodies and damage of various organs. SLE and pSS have several similar clinical and serological aspects; likewise, SLE and Sjögren's syndrome may coexist (so-called secondary Sjögren's syndrome). However, applied classification criteria do not differentiate SLE and pSS. It is known that humoral immunity plays significant part in pathogenesis of those diseases; hereby, we can expect imbalances in B cell subset frequencies during SLE and pSS.

Objectives: To investigate clinical utility of B cell subsets in distinguish SLE and pSS during diagnosis.

Methods: A total of 25 SLE patients, 25 SS patients and 49 healthy volunteers (HV) were included in the study. The diagnosis of SLE was performed according to the 2019 EULAR – ACR classification criteria, the diagnosis of pSS – according to the 2016 EULAR – ACR criteria. Phenotyping of blood B cell subsets was done using flow cytometry. Total peripheral blood B cells were identified using CD19 expression, distinct B cell subsets were characterized by IgD, CD38 and CD27 expression. All of the statistical analysis of data was performed with STATISTICA Version 12.0 Inc. (USA).

Results: We evaluated the percentages of circulating B-cell subsets using three major classification schemes based on the relative co-expression of either IgD/CD38 (so-called "Bm1-Bm5" classification), IgD/CD27 and CD38/CD27. A discriminant analysis was performed for all B cell classifications. Analysis of CD38 and CD27 co-expression demonstrated most significant separation between patients with SLE and pSS (fig. 1). Moreover, discriminant analysis carried out by using a forward stepwise model demonstrated that the top significance was documented while assessing the percentage of plasmoblasts (CD27hiCD38hi), resting memory B-cells (CD27dimCD38low), mature active B-cells (CD27dimCD38dim), naive mature B-cells (CD27dimCD38low), as well as counting the absolute numbers of transitional B-cells (CD27lowCD38hi), model percent correct was 78,6% ($p < 0,05$, tab.1).

Figure 1. Graphic distribution of SLE and pSS patients as well as HV analyzed by discriminant analysis.