study, DNA extraction from peripheral blood samples was performed using the phenol-chloroform method. SNPs were genotyped using the real-time PCR with fluorescent probes. The allele and genotype frequencies were compared using the y2 test. Odds ratios (ORs) and 95% confidence intervals (95% CI) were calculated using the VassarStats online tool.

Results: Utilizing recessive genetic model we found an association between TT genotype of ST74 rs7574865 (OR = 2.362; 95% CI [1.037 – 5.376], p = 0.038) and RA. For IL6 rs1800795, it was found that CC genotype had significantly higher frequency among patients with rheumatoid arthritis as compared to that in controls (OR = 1.52; 95% CI [1.02 – 2.27], p = 0.0456). No associations of IL6R and IL6 with RA. For STAT4 results were calculated using the VassarStats online tool.

Conclusion: We identify an average age of 38.5±12. Cases and controls remained in Hardy-Weinberg equilibrium. An association study was performed using Finetti, a p value ≤ 0.05 indicated association.

Results: We identify an average age of 38.5±12. Cases and controls remained in Hardy-Weinberg equilibrium. An association with susceptibility to systemic lupus erythematosus (SLE) in Mexican population.

Objectives: To determine whether the BANK1 R61H (rs1051487G/A) and A383T (rs3733197G/A) SNVs are associated with clinical and immunological manifestations in SLE.

Methods: Our study included 123 Mexican women with SLE (SLICC 2012 criteria). Genotyping of the two BANK1 SNVs were obtained by TaqMan probes and real-time PCR. An association analysis was performed between the alleles and genotypes of BANK1 R61H and A383T with the clinical and immunological manifestations included in the SLE SLICC classification criteria. Hardy-Weinberg equilibrium and an association study was performed using Finetti, a p value ≤ 0.05 indicated association.

Results: We identify an average age of 38.5±12. Cases and controls remained in Hardy-Weinberg equilibrium. An association with susceptibility to SLE was found between the alleles and genotypes of the two BANK1 SNVs and joint manifestations rs1051487G/A; AA + GA vs GG, OR 4.45, p = 0.004, rs3733197G/A; AA + GA vs GG, OR 2.68, p = 0.032, respectively, as well as with protection for neurological and renal involvement (rs1051487G/A, OR 0.16, p = 0.02, rs3733197G/A; OR 0.40, p = 0.02, respectively) (Table 1a and b). No association was found with other clinical manifestations.

Conclusion: Our data in the Mexican population show that both BANK1 R61H and A383T SNVs are risk factors for synovitis. On the other hand, these BANK1 R61H and A383T variants are protective factors for neurological and renal damage, respectively.

References:

Disclosure of Interests: None declared

AB0009
ASSOCIATION BETWEEN POLYMORPHISMS OF BANK1 AND MANIFESTATIONS OF SLE


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Background: BANK1 encodes an adaptor/ scaffold protein primarily expressed in B cells, which is involved in cell signaling and activation. Genome-wide association studies (GWAS) have identified different BANK1 single nucleotide variants (SNVs) associated with SLE primarily in European or Asian-derived populations. Interestingly, we recently have documented an association between this gene and susceptibility to systemic lupus erythematosus (SLE) in the Belarusian population.

Objectives: To determine the association between the BANK1 R61H (rs1051487G/A) and A383T (rs3733197G/A) SNVs with risk of RA and their impact on disease subtypes in the Belarusian population, some features of interplay being revealed between gene polymorphisms analyzed and RA antibody titers. Above-mentioned SNPs may contribute to RA genetic susceptibility in the Belarusian population.

Disclosure of Interests: None declared
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Results: No statistically significant differences between patients with IgAV and healthy controls were observed when each IL17A genetic variant was analyzed independently. Similarly, no statistically significant differences between patients with IgAV and healthy controls were found when the five IL17A polymorphisms were evaluated combined conforming haplotypes. In addition, there were no statistically significant differences in genotype, allele and haplotype frequencies of IL17A when patients with IgAV were stratified according to the age at disease onset or to the presence/absence of gastrointestinal or renal manifestations.

Conclusion: Our results do not support an influence of IL17A on the pathogenesis of IgAV.

References:

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INFLUENCE OF IL17A GENE ON THE PATHOGENESIS OF IMMUNOGLOBULIN-A VASCULITIS


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Background: Cytokines signaling pathway genes represent a key component of the genetic network implicated in the pathogenesis of Immunoglobulin-A vasculitis (iGAV) [1], an inflammatory vascular pathology. Interleukin (IL)17A is a genetic risk locus for autoimmune diseases, such as giant cell arthritis [2] and spondyloarthritis [3].

Objectives: To determine the potential influence of IL17A on IgAV.

Methods: Five IL17A tag polymorphisms (rs4711998, rs8193036, rs3819024, rs2275913 and rs7747909) were genotyped in 360 Caucasian patients with IgAV and 1,003 sex and ethnically matched healthy controls.

Results: No statistically significant differences between patients with IgAV and healthy controls were observed when each IL17A genetic variant was analyzed independently. Similarly, no statistically significant differences between patients with IgAV and healthy controls were found when the five IL17A polymorphisms were evaluated combined conforming haplotypes. In addition, there were no statistically significant differences in genotype, allele and haplotype frequencies of IL17A when patients with IgAV were stratified according to the age at disease onset or to the presence/absence of gastrointestinal or renal manifestations.

Conclusion: Our results do not support an influence of IL17A on the pathogenesis of IgAV.

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ROLE OF IRF5 GENE ON THE PATHOGENESIS OF IMMUNOGLOBULIN-A VASCULITIS


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Background: Interferon signaling pathway genes plays a relevant role in autoimmunity. Genetic variants in the interferon regulatory factor (IRF) 5 gene, that encodes the major regulator of the type I interferon induction [1], have been related to the development of several inflammatory diseases [2].