Objectives: Our aim was to analyze the association of HLA class II with HS in a Caucasian population from Cantabria (northern Spain).

Methods: In this study we analyzed the HLA-A, -B, -C, DRB1, -DQA1 and -DQB1 allele distribution in 106 HS patients and 262 age- and sex-matched controls from a Caucasian population of Cantabria (northern Spain).

Results: HLA-A*29 and B*50 were significantly more frequent in HS patients and A*30 and B*37 in controls, but these associations disappeared after correction. On the other hand, DRB1*07, DQA1*02 and DQB1*02 were significantly more frequent in controls (p = 0.026, p = 0.0012 and p = 0.0005 respectively), and the HLA allele DQB1*0301 was significantly more frequent in HS patients (p = 0.00007) all of them after Bonferroni correction. Furthermore, the DRB1*07; DQA1*02; DQB1*02 haplotype was significantly more frequent in controls (p = 0.0005).

Conclusion: This is the first study showing an association of HLA-class II with HS. Our results suggest that HLA-II alleles (DRB1*07, DQA1*02, DQB1*02 and DQB1*0301) and the DRB1*07–DQA1*02–DQB1*02 haplotype could influence on resistance or susceptibility to HS.

References:

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HYPOMETHYLATION OF THE PROMOTER REGION OF TL4 GENE AT A SYSTEMIC LEVEL IN PATIENTS WITH RHEUMATOID ARTHRITIS AND PERIODONTITIS

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Background: Periodontitis (PD) has long been linked with Rheumatoid arthritis (RA) [1]. Epigenetic modifications are being recently explored to explain such associations, DNA methylation being one such important mechanism.

Objectives: To study the effect chronic generalized periodontitis on systemic methylation of TL4 genes in comparison to only RA and RA with PD patients.

Methods: Twenty-three RA patients, among which 11 patients had chronic generalized PD, 20 patients with only PD and 15 healthy individuals recruited. DNA was isolated from PBMCs of the patients blood, then were first bisulphite converted and then methylation specific PCR were performed using primers for methylated and unmethylated promoters of TL4. The DNA amplifications were checked in horizontal gel electrophoresis. The methylation signatures were verified by DNA sequencing (Sanger) of the amplified products.

Results: The anti-CCP, DAS-28 and HAQ DI were higher in patients with both RA and PD (220±40, 5.7±0.2, 15.0±1 respectively, p<0.05). Control samples had shown amplification bands for methylated primers of TL4 but not for unmethylated primers of TL4. However, only RA, only PD and RA with PD samples, had shown amplification for unmethylated primers and not for methylated primers. These results together with DNA sequencing indicated that 4 CpG sites in the promoter of TL4 genes were hypo-methylated in the PBMCs of patients whereas those remain methylated in healthy individuals.

Conclusion: The observations indicated that though PD is a localised disease of the gingiva there is a systemic involvement of TL4 mediated pathways in them which is similar to those in RA. However, further validation in larger cohort and down-stream signalling molecules needs to be studied.

References:

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VALUE OF ULTRASOUND IN ASSESSMENT OF ACTIVE SACROILIAC IN PATIENTS WITH AXIAL SPONDYLOARTHRITIS

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Background: The inflammatory of the sacroiliac joints (SJJs) called sacroiliitis, is a characteristic of axial Spondyloarthritis (axSpA). The detection of sacroiliitis is meaningful to prevent irreversible changes. The tool of assessment of sacroiliitis including radiographs, computed tomography (CT) and magnetic resonance imaging (MRI). Ultrasound (US) has also been used in the evaluation of sacroiliitis in recent years.

Objectives: We aimed to evaluate the value of US in the assessment of active sacroiliitis in axSpA patients.

Methods: Fifty-one patients fulfilling Assessment of SpondyloArthritis International Society (ASAS) 2009 criteria for the classification of axSpA were recruited. The patient underwent MRI and US evaluation of bilateral SJJs. MRI was performed using the sequences of T1WI, T2WI and fat suppression T2WI (FS-T2WI). MRI sacroiliitis was defined according to ASAS criteria of active sacroiliitis. The Spondyloarthritis research Consortium of Canada (SPARC) scoring was used to evaluate the inflammatory lesions in SJJs. US were performed by an ultrasonographer with 10 years of experience in musculoskeletal ultrasound, and resistive index (RI) value was recorded. The US sacroiliitis was defined as the presence of more flow signals at SJ with an RI ≤ 0.75. The HLA-B27, erythrocyte sedimentation rate (ESR) and hypersensitive C-reactive protein (hsCRP) were also evaluated. Consistency rate, sensitivity, specificity, positive predictive values (PPV) and negative predictive values (NPV) for the diagnosis of sacroiliitis by US were calculated, using MRI as the gold standard.

Results: Of the 51 patients, 24 were female and 27 were male. The HLA-B27 positive rate was 90.2% (46/51). The consistency rate of US and MRI sacroiliitis was 55.88 (57/102). The sensitivity and specificity of US for the diagnosis of sacroiliitis were 55.93 (33/59) and 55.81 (24/43) respectively. The PPV and NPV were 63.46 (33/52) and 48 (24/50) respectively. There was no significant difference in ESR and hsCRP between the US positive sacroiliitis and the others (P = 0.7477 and 0.2268, respectively). The SPARC scores have no significant difference between the US positive sacroiliitis and the others (P = 0.2206). The RI was not significantly associated with the MRI SPARC score (P = 0.4236).

Conclusion: US may be an optional method for preliminary screening sacroiliitis. But its reliability as a diagnostic method needs further verification.

References:

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THE IMPACT OF STAT4 RS7574865, IL6 RS1800795, IL6R RS2228145 AND RS4845618 ON RHEUMATOID ARTHRITIS SUSCEPTIBILITY IN BELARUSIAN POPULATION

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Background: Rheumatoid arthritis (RA) is a chronic systemic disorder of the connective tissue of still unknown aetiology and complex autoimmune pathogenesis that primarily affects small joints. HLA alleles provide for 11-37% of the RA heritability, suggesting the substantial role of the non-HLA loci in genetic predisposition to RA. Among non-HLA loci, IL6, IL6R and STAT4 genes attract attention, however, the data concerning their influence on RA risk are somewhat contradictory.

Objectives: The aim of the study was to analyze the involvement of four SNPs (STAT4 rs7574865, IL6 rs1800795, IL6R rs2228145 and rs4845618) in RA susceptibility.

Methods: 187 patients diagnosed with RA (mean age 58.2 ± 11.9), and 380 healthy blood donors (mean age 37.18 ± 10.69 years) were included into the