was also used. In order to confirm the specificity of the new genetic variation associated with PsA risk, we analyzed the association with purely cutaneous psoriasis (PsC, n=614) and rheumatoid arthritis (RA, n=1,191). We performed a pharmacogenetic analysis to investigate the new PsA-specific pathways as a source for drug discovery in PsA.
Results: GWAS meta-analysis identified a new association between *B3GNT2* gene and PsA ($P<5e-08$). In the GWAS pathway analysis, we identified and validated a total of 14 genetic pathways associated with PsA risk. From these, the glycosaminoglycan (GAG) metabolism pathway was also found to be significantly associated with PsA risk when directly contrasted to the PsC cohort as well as the RA cohort. At the functional level, we detected a significant differential expression of GAG metabolism pathway genes in blood samples from PsA patients compared to PsC patients. The pharmacogenetic analysis identified several FDA-approved drugs likely to modify the GAG pathway.
Conclusions: The present study represents an important step towards the characterization of the genetic factors specific to PsA risk.
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SAT0459 LOW DOSE IL-2 RESTORES IMBALANCE BETWEEN TH17 AND REGULATORY T CELLS IN PATIENTS WITH PSORIATIC ARTHRITIS

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Background: Psoriasis arthritis is one of chronic, relapsing, inflammatory autoimmune disorders with skin lesions and joint damage. A therapeutic revolution of psoriatic arthritis (PsA) is still a considerable unmet need in the past decades. It has been well known that the imbalance of Th17 cells and regulatory T cells (Tregs) may be a pivotal cause of PsA. Correction of this dysfunction can be a potential therapy of PsA.
Objectives: In this study, we measured and compared both absolute numbers and proportions of CD4+CD17+ Th17 cells and CD4+CD25+Foxp3+ Treg cells in peripheral blood of PsA patients and healthy controls to explore the immunopathogenesis of PsA; on the other hand, the effects of low-dose recombinant human IL-2 (rhIL-2) on Th17 and Treg cells were investigated in patients with PsA.
Methods: Both absolute numbers and proportions of Treg and Th17 cells in peripheral blood, defined as the CD4+CD25+Foxp3+T or CD4+IL-17+ T cell populations, were examined by flow cytometry in 40 healthy controls and 77 patients with PsA, including 39 patients who had never received disease-modifying antirheumatic drugs (DMARDs) and 38 patients who were receiving or had received DMARDs. Among these patients, 20 patients consented at enrollment to receive rhIL-2 treatment. Before and after treatment (50WIU/d for 5 days, IH), Th17 and Treg cells in peripheral blood were analyzed by flow cytometry.
Results: The absolute count of Th17 cells in patients with PsA was very significantly higher than that of healthy controls ($P<0.01$), but the proportions of Th17 cell were not seen difference between PsA and healthy controls ($P>0.05$). In contrast with treated-PsA patients, the absolute count of Th17 cells was significant higher in untreated-PsA patients ($P<0.05$). After the course of rhIL-2 treatment, there was a significant increase in the absolute count of Treg cells ($P<0.05$), but no diference in the absolute count of Th17 cells, Th17/Treg was significantly lower and went back to nomal.
Conclusions: The results suggest that, not the proportion, but the decrease in the absolute count of Th17 cells, defined as the CD4+CD17+ populations, contributes to the pathogenesis of PsA. After the treatment of rhIL-2, there was a more significant increase in the absolute count of Treg cells than that of Th17, and consequently the balance of Th17/Treg was restored to normal, leading to the development of new therapies.
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SAT0460 ASSOCIATION BETWEEN INFLAMMASOME-RELATED POLYMORPHISMS AND PSORIATIC ARTHRITIS

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Background: In recent years, research on the interleukin 1β(IL1 β)–regulating protein complex, called the inflammasome, has shown interesting associations with various inflammatory diseases. E.g. for Rheumatoid Arthritis (RA) (1) and psoriasis (Pso) (2, 3) associations with genetic polymorphisms in genes related to the inflammasome has been discovered. So far, no studies investigating genetic polymorphisms in inflammasome genes in Psoriatic Arthritis (PsA) patients have been published.
Objectives: To examine whether polymorphisms in genes related to inflammasomes confer increased risk for psoriatic arthritis.
Methods: DNA from 771 PsA patients and 793 healthy controls from Sweden were analyzed for different single nucleotide polymorphisms (SNPs) in *NLRP3* (rs35829419, rs10733113, rs4353135), *CARD8* (rs 2043211) and *NLRP1* (rs8079034, rs878329).
Results: Significant associations with PsA were found between carriage of allele, C, of rs878329 in *NLRP1* (Chi-2=6.5, OR (95% CI); 0.75 (0.60–0.94), P=0.011) and allele G in rs4353135 in *NLRP3* (Chi-2=4.8, OR (95% CI); 1.25 (1.02–1.53), p=0.028). Genotype distribution were also significantly different between patients and controls and for rs878329 in *NLRP1* there was a significant difference in allele frequency (G/C) between patients and controls (Chi-2=5.8, OR (95% CI); 1.20 (1.03–1.38), p=0.016). No significant associations with PsA were found for the other SNPs analyzed.
In genotype analysis, a significant higher frequency of genotype GG in rs878329 in PsA was detected (32.9% vs 26.9%, Chi-2=6.49, OR (95% CI); 1.34 (1.07–1.67), p=0.011), whilst no significant differences were detected for genotypes GC or CC. For rs4353135, a significantly higher frequency of genotype TG (43% vs 37.6%, Chi-2=4.66, OR (95% CI); 1.25 (1.02–1.53), P=0.033) and a significantly lower frequency of genotype TT (50.5% vs 56.1%, Chi-2=4.85, OR (95% CI); 0.80 (0.65–0.98), P=0.028) was seen in PsA, no significan difference was detected for genotype GG.
Conclusions: Carriage of rs878329C in *NLRP1* was less frequent in patients with PsA compared with controls indicating a protecting effect, but when different genotypes were analysed the difference likely results from an increased risk of PsA with genotype GG. The results are in contrast with the study of Ekman *et al*, where an increased transmission of rs878329C to family members with psoriasis was seen (3), indicating an increased risk of developing skin psoriasis for carriers of C, but in agreement with the study of Sui *et al* on patients with RA, where an association was detected for carriage of C (OR 0.82, p=0.02), with the risk genotype for RA being GG (4). Thus, the genotype GG possibly confers risk of arthritic disease whilst the C-allele seems associated with skin disease. Carriage of rs4353135G in *NLRP3* was more frequent in PsA patients compared with controls indicating an increased risk of disease, but only genotype GT was significantly increased in PsA. The study is, to our knowledge, the first to study possible associations between genes related to the inflammasome and PsA. In the study associations were found between one SNP in *NLRP3* and one SNP in *NLRP1*, indicating a possible involvement in pathogenesis of PsA disease.
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SAT0461 CHARACTERISATION OF DIFFERENT LOW DISEASE ACTIVITY MEASUREMENTS IN PATIENTS WITH PSORIATIC ARTHRITIS

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Background: Selection of the correct target to guide treatment is crucial for effective disease management in patients with psoriatic arthritis (PsA).
Objectives: To evaluate the prognostic value of several low disease activity (LDA) measurements in patients with PsA and psoriasis to assist physicians choose a valid target that facilitates assessment in clinical practice.
Methods: This was a post-hoc analysis from the PRESTA[1] clinical study. LDA targets analyzed were: Disease Activity in PsA (DAPSA) LDA ≤14 (tender joint count [TJC], swollen joint count [SJC], patient global visual analog scale [Pt VAS], pain VAS, C-reactive protein [CRP]; clinical (c)DAPSA LDA ≤13 (DAPSA without CRP); and minimal disease activity (MDA) measurement defined as 5/7 cut-offs (TJC ≤1, SJC ≤1, psoriasis activity and severity index [PASI] ≤1, Pt pain ≤15mm, Pt VAS ≤20mm, health assessment questionnaire ≤0.5, tender entheseal points ≤1). Additional MDA measurements were investigated where 5/7