HLA-ANTIGENS AND DISEASE MANIFESTATION IN A COHORT OF 600 SOUTHERN FRENCH PATIENTS WITH PSORIATIC ARTHRITIS

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Introduction Currently, there is no biomarker available to diagnose psoriatic arthritis (PsA). Genome wide association studies showed that the majority of PsA loci are shared with PsC.1 However, PsA has a strong familial predisposition, more so than PsC as was shown in an Icelandic population.2 In the GWAS performed in UK and Germany, the strongest genetic locus is located within the major histocompatibility complex (MHC).3,4 No studies have been performed in southern Europe.

Objectives The primary objective of our study was to focus on the HLA class I and II alleles found in a cohort of 600 French patients with PsA clinically well characterised, compared to a control cohort. The secondary objective was to compare two clinical subsets of PsA, one axial and one peripheral, to test which genotype determines these phenotypes.

Methods 600 patients from the Rheumatology department, St. Marguerite’s Hospital, Marseilles, who fulfilled the CASPAR criteria for PsA, underwent clinical, radiographic and laboratory investigations. HLA Class I and Class II alleles were genotyped. A cohort of 2346 healthy blood donors (HBD) was also tested.

Results
- Comparison between the PsA population and controls showed one set of alleles significantly associated with PsA; HLA-B*27, B*21(B*50), C*06 and with a weak significance HLA-A*01, A*25, B*13.
- Within the PsA population, two genetically different subgroups determine two different clinical subtypes: Peripheral disease was significantly associated with C*06 in disequilibrium linkage with B*13 and DR*07, independently associated with HLA-A*01, B*21 and B*17. Axial disease was significantly associated with HLA-B*27 in disequilibrium linkage with C*01 and C*02.

Conclusions This is the first and the largest study ever realised in southern Europe. It lead to conclude that PsA is genetically heterogenous. We found that PsA is divided in two genetically and phenotypically relevant subgroups, one axial group with HLA-B*27 predominance very close to Ankylosis spondylitis and one peripheral group with HLA-C*06 predominance. Other HLA alleles within the MHC are also implied. The clinical utility of HLA typing in PsA should be further addressed in larger studies.

REFERENCES
canonical NF-κB pathway and downstream DLL4-Notch1/4 may be involved in LEC mediated modulation of T-cell homing and deserves further exploration.

Disclosure of interest None declared