Rheumatology 2017, 7:1(Suppl)
http://dx.doi.org/10.4172/2161-1149.C1.016

Association of IL17A and *IL17F* genes with rheumatoid arthritis disease and the impact of genetic polymorphisms on response to treatment

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Background & Aim: Previous studies have reported an association between polymorphisms in Il17A and *IL17F* genes and the prevalence of rheumatoid arthritis (RA) in Caucasian populations. The aim of the current study was to investigate that polymorphisms in both genes may affect RA susceptibility in the Tunisian population and to study the relation between serum IL-17 levels and synovial fluid (SF) levels and risk in RA patients. We suggested also that these polymorphisms may influence response to treatment in Tunisian RA patients.

Methods: We studied IL17A -152 G/A (rs2275913), *IL17F* 7488 A/G (rs763780) and *IL17F* 7383 A/G polymorphisms in a Tunisian population. The genotypic and allelic distributions of IL-17A and IL-17F genes polymorphisms were analyzed by polymerase chain-reaction (PCR) and restriction fragment length polymorphism (RFLP) for 108 patients and 202 healthy controls. IL-17 levels were measured in synovial fluid (SF) and in serum of both 108 patients and 47 controls (pg/ml) using enzyme-linked immunosorbent assay (ELISA) technique.

Results: Our results indicated that *IL17F* 7488 A/G and *IL17F* 7383 A/G polymorphisms were significantly associated with RA risk in the whole population. However, IL17A-152 G/A polymorphism did not show any significant association with RA prevalence in the Tunisian population. Stratification according to demographic and clinical features revealed differential significant associations of IL17A -152 G/A, *IL17F* 7488 A/G and *IL17F* 7383 A/G polymorphisms within different subgroups and subtypes of clinical-pathologic features in RA patients. IL17A -152 G/A polymorphism was associated with an enhanced response to biologic and MTX treatment. *IL17F* 7383 A/G polymorphism was associated with an enhanced response to biologic treatment. The *IL17F* 7488 A/G polymorphism decreased significantly good response to biologic treatment, but enhanced response to MTX treatment. The expression of IL-17 in serum and synovial fluid (SF) in RA patients was significantly higher than that observed in healthy controls ($P<0.0001$) and their levels depend on RA severity. The mean IL-17 levels in the anti-TNF α treated patients decreased significantly at 0 week, 14 weeks and 30 weeks ($P=0.0032$ and $P<0.0001$, respectively).

Conclusions: Our results confirmed IL17A and *IL17F* as potential candidate genes involved in RA. They play pivoting roles in the susceptibility and in clinical features of RA disease. Responses to RA treatments are differently conditioned by polymorphisms in IL17A and *IL17F* genes.

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