Pediatric Rheumatology



Poster presentation



# FOXP3 Polymorphism and gene expression in Italian patients with Kawasaki Syndrome

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## Background

The transcription factor FOXP3 is a key regulator of immune homeostasis of natural regulatory T lymphocytes (Treg). Aim of our study was to investigate the link between the FOXP3 gene expression and polymorphism, and Kawasaki Syndrome (KS).

## Materials and methods

The single nucleotide polymorphism (SNP) at position 543 of exon 5 of FOXP3 was investigated in 58 caucasian children patients with KS (F:30, M:28), recruited at our Paediatric Reumatology Unit. In 6 patients we could evaluate the percentage of circulating CD4+ T cells co-expressing CD25 and FOXP3 by flow cytometry, mRNA transcripts of FOXP3 in PBMCs by Real Time PCR (with Rnase P as internal control gene), and the levels of IL-17 in PBMCs supernatants (5 × 104 cells) by a quantitative immunoassay Kit.

## Results

In KS subject, 2 females (3.4%) showed the 543 SNP in heterozygosis (C>T), as actually reported in healthy european females (4.3%). FOXP3 mRNA expression was significantly lower (3-fold) than in healthy controls, while FOXP3 protein expression determined by flow cytometry was not lower than in controls. IL-17 levels were 8-fold higher in KS than in controls (mean 340 *vs* 40 pg/mL)

## Conclusion

Compared to healthy controls, in our patients, FOXP3 gene 543 SNP does not differ, FOXP3 mRNA expression was lower, without a correspondent lower protein expression, and IL-17 levels were much higher in patients. Due to the small number of patients, these results warrant further studies to evaluate FOXP3 expression in relationship with IL-17 in KS.