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Interleukin 6 G>G genetic polymorphism (rs1800795) and the response to tocilizumab in rheumatoid arthritis patients

[1]MDC González-Medina, [1]X Díaz-Villamarín, [1]CL Dávila-Fajardo, [1]MJ Soto-Pino, [2]A Gómez- Martín, [2]LJ Martínez-González, [1]I Casas-Hidalgo, [3]E Sánchez-Yáñez, [4]M Domínguez- Cantero, [1]J Cabeza-Barrera.

1Instituto de Investigación Biosanitaria de Granada Hospital Universitario San Cecilio – Granada – Spain, Department of Clinical Pharmacy, Granada, Spain
2Centre for Genomics and Oncological Research GENYO – Pfizer-University of Granada-Andalusian Regional Government, Genomics Unit, Granada, Spain
3Hospital Universitario Virgen de La Victoria, Department of Clinical Pharmacy, Málaga, Spain
4Hospital Puerta Del Mar, Department of Clinical Pharmacy, Cádiz, Spain

BACKGROUND AND OBJECTIVE

Interleukin 6 is involved in the pathogenesis of rheumatoid arthritis via its broad effects on immune and inflammatory responses. Sustained IL6 activity can cause tissue damage in different tissues. Previous studies have shown that G-allele at the -174G>C (rs1800795) polymorphism is related with the high-producing IL6. The aim of our study was to explore the potential role of *IL6* genetic polymorphisms as a predictor of tocilizumab efficacy in rheumatoid arthritis (RA) patients and to compare the results with a previous GWAS.

MATERIAL AND METHODS

The IL6 (G>C) (rs1800795) genetic variant was genotyped using predesigned TaqMan® genotyping assays technology and analyzed on a ViiA7® Real-time PCR system. Clinical response was evaluated at 24 weeks with the use of the 28-joint disease activity score criteria (DAS28) and good response and remission were classified according to EULAR criteria. EULAR good response was defined as a change of DAS28>1.2 and DAS28 ≤3.2. EULAR remission was defined as achieving DAS28 at 14 weeks ≤2.6. The statistical analysis was performed using SPSS v.20

RESULTS

	N=140 n (%) or mean (±sd)
Women	111 (79%)
Age	53.25 (±12.42)
DAS (Baseline)	5.71 (±1.13)

IL-6 G>C polymorphisms	Good EULAR Response		REMISSION	
	O.R (95% C.I.)	P-value	O.R (95% C.I.)	P-value
CC vs no CC	1.07 (0.05-19.7)	0.35	1.11 (0.41-2.98)	0.85
GC vs no GC	1.03 (0.22-4.70)	0.97	1.01 (0.52-1.94)	0.98
GG vs no GG	0.58 (0.12-2.67)	0.5	0.96 (0.48-1.89)	0.88

CONCLUSION

Our results confirm that IL6 G>C rs 1800795 polymorphisms could not be useful as a genetic marker of tocilizumab efficacy in RA patients. More studies are necessary to confirm these results.