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Role of *TRAILR1* and *TNFR1A* polymorphisms in the susceptibility and pharmacogenetics of rheumatoid arthritis and ankylosing spondylitis patients treated with infliximab

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Introduction

Polymorphisms in genes involved in antibody-dependent cellular cytotoxicity and apoptosis have been associated with inter-individual differences in the response to anti-TNF agent infliximab in arthritides. *TRAILR1* and *TNFR1A* are two genes related to the extrinsic pathway of apoptosis.

Aim

To evaluate the role of two polymorphisms (rs20575/G36A and rs767455/C626G) in these genes in the susceptibility and pharmacogenetics of rheumatoid arthritis (RA) and ankylosing spondylitis (AS) patients treated with infliximab. Patients and Methods. *TRAILR1* and *TNFR1* genotyping was performed in 138 patients (86 RA and 52 AS), and in two groups of controls (158 for RA and 182 for AS). A subset of 47 RA and 40 AS patients was also studied for the association of this polymorphism with the response to infliximab treatment assessed by EULAR and BASDAI criteria, respectively.

Results

No significant differences were observed between cases and controls in the genotype and allele distribution of *TRAILR1* or *TNFR1A* polymorphisms for both diseases, though a tendency for a RA protective role of *TNFR1A* AA genotype was observed (29.8% vs. 38.2%; p=0.1172).

TRAILR1 G allele was associated with poor response after 3 month of infliximab treatment (G: 32.4% vs. C 14%; p=0.044). Similar results were observed for AS, as the prevalence of non-responders was significantly lower in C carriers both, at 3 months (21.4% vs. 83.3%; p=0.003) and 6 months (11.5% vs. 50%; p=0.029) of infliximab treatment. No significant differences were observed for *TNFR1* polymorphism in terms of response.

Conclusions

This work provides the first evidence that rs20575 polymorphism in *TRAILR1* seems to influence the response to infliximab treatment both in RA and AS patients.

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