Tumour necrosis factor (TNF)? -308 G/G promoter polymorphism and TNF? levels correlate with a better response to adalimumab in patients with rheumatoid arthritis

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Objective: To investigate the influence of -308 tumour necrosis factor-? (TNF?) promoter polymorphism and circulating TNF? levels in the clinical response to adalimumab treatment in patients with rheumatoid arthritis (RA). Methods: Eighty-one patients with active RA were genotyped for the -308 TNF? polymorphism by polymerase chain reaction-restriction fragment length polymorphism (PCR-RFLP) analysis and subdivided into two groups for each polymorphism (G/A and G/G genotype). All received 40 mg of adalimumab subcutaneously every other week. We

compared the groups' clinical responses to adalimumab at 8, 16, and 24 weeks using the Disease Activity Score in 28 joints (DAS28). Results: Both groups showed a significant improvement from baseline. A significant difference between groups was found at week 24. We found that 88.2% of G/G versus 68.4% of G/A for the -308 polymorphism were DAS28 responders ($p = 0.05$). The score improvement at week 24 was 2.5±1.3 in the G/G group and 1.8±1.3 in the