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B cells are supposed to play an important role in rheumatoid arthritis (RA). Accordingly, rituximab, a chimeric monoclonal antibody against CD20, effectively depletes B cells ameliorates disease although a subset of patients don't respond. We aimed to identify biomarkers and biological processes underlying non-responsiveness. Therefore, the effect of rituximab on gene-expression levels in peripheral blood of responders and non-responders was studied.

RNA was isolated from 15 rituximab-treated RA patients before, 3 and 6 months after start of treatment. Gene-expression profiling was performed using Illumina HumanHT 12-vs bead chips. Clinical responder status was determined after 6 months by change in disease activity score (Δ DAS).

Pharmacogenomics revealed 16 B cell related genes that were significantly downregulated after 3 months. Subsequent analysis of expression profiles of patients ranked by increasing Δ DAS learnt that a good response (Δ DAS28 >1.2) is observed for patients with a low level of expression of a cluster of type-I interferon response genes (IRG) at baseline ($p=0.065$). We observed that after 3 months regulation of IRG-activity varied between patients. An increase in IRG-expression was observed for good responders (Δ DAS28 >1.2), whereas no induction or decrease was found for non-responders.

The IRG signature is associated with responder status upon B cell depletion therapy in RA.

A30 IFN SIGNATURE DETERMINES RESPONDER STATUS ON B CELL DEPLETION IN RHEUMATOID ARTHRITIS PATIENTS

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