Glatiramer acetate antibodies, gene expression and disease activity in multiple sclerosis

**Background:** Glatiramer acetate (GA) treatment suppresses disease activity in multiple sclerosis (MS). The immunological response to treatment may differ in patients who are stable on GA therapy and patients with breakthrough disease activity, but the results of previous studies are inconsistent.

**Objectives:** We studied the immunological response to GA and its relationship with disease activity.

**Methods:** Anti-GA antibodies in plasma and the expression of genes encoding cytokines and T-cell-polarizing transcription factors in blood cells were analysed by flow cytometric bead array and polymerase chain reaction (PCR) analysis in 39 untreated and 29 GA-treated relapsing–remitting MS patients. Definition of breakthrough disease was based on the occurrence of relapses, disability progression, or gadolinium (Gd)-enhanced MRI.

**Results:** The expression of T helper type 1 (Th1) and Th17 cytokines and transcription factors was reduced during long-term treatment, but there was no relationship between the expression of cytokines and transcription factors and anti-GA antibodies. High expression of mRNA encoding GATA3 and lymphotoxin-β (LT-β) was associated with low disease activity in Gd-enhanced MRI studies. None of the variables studied were associated with clinical disease activity. GA treatment resulted in the development of IgG and IgG4 anti-GA antibodies during the first months of treatment, persisting during long-term treatment.

**Conclusions:** The observed relationship between the expression of mRNA encoding GATA3 and LT-β expression and MRI disease activity deserves further analysis in future studies. The development of anti-GA antibodies was observed in all patients treated with GA, but this was not related with measures of cellular immunity, clinical or MRI disease activity.

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