Short-term effect of IFN-β therapy on the expression of IL23A, FOXP3 and IL10 in CD4+ and CD25+ T cells of MS patients

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Abstract

Multiple sclerosis (MS) is an autoimmune disorder causing demyelination in axons. Available therapies target different molecules, but not all have therapeutic effects on disease progression, and this effect can only be seen after a long-time administration. By the time, the disease progresses, and its outcomes become unbearable for the patient. IFN-β has been used in MS therapy for many years. It slows down the disease progression, also reduces disease symptoms by targeting T cells. Yet, a considerable portion of the patient has experienced no therapeutic response to IFN-β. Therefore, it is necessary to determine disease-specific biomarkers which allow early diagnosis or treatment of MS. Here, it was aimed to determine the effects of IL10, IL23A and FOXP3 genes on the therapeutic response to MS after IFN-β administration. PBMCs were extracted from blood samples to isolate CD4+ and CD25+ T cells. Cytotoxicity assays were performed on each cell type for determining optimum drug concentration. Then cells were cultured again and determined drug concentration was administered to the cells to measure gene expressions with RT-PCR. At the end of the study, it was found that the cytotoxic effect of IFN-β was more efficient as the exposure time was expanded regardless of drug concentration. Moreover, CD25+ T lymphocytes were more resistant to IFN-β. IL23A was down-regulated, whereas FOXP3 was up-regulated at 48h in CD4+ T cells. For CD25+ T cells, the graded increase of FOXP3 was obtained while IL10 expression was gradually decreased throughout the drug intake, which both were statistically significant.

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