sphingosine-1-phosphate receptor (20,21). The drug induces internalization of the receptor, that is, transfer from the cell surface to the cell's interior, thereby depriving the T cell of a necessary tool for exiting from the lymph node. Only a small subset of T cells, not all T cells, becomes trapped in the lymph nodes.

c. Interferon-beta1 (IFNbeta1)

Interferon-beta1 is a cytokine. When used for treating multiple sclerosis, IFNbeta1 has a number of mechanisms of action. IFNbeta1 acts at a number of points in the immune system (22) and at the blood–brain barrier. The mechanisms of action of interferon in multiple sclerosis treatment are many, and have not been conclusively established (23). The effects of IFNbeta1 in mitigating the disease include action on:

- Cytokine expression. The effects of IFNbeta1 on cytokine expression include increases of anti-inflammatory cytokines, and reductions of pro-inflammatory cytokines (24). IFNbeta1 increases expression of cytokines that are anti-inflammatory, namely interleukin-10 and interleukin-4. IL-10 and IL-4 are Th2-type cytokines (25).
- Reduces various activities of T cells. INFbeta1 reduces the migration of T cells (26) the activation of T cells, and the expression by $CD4^+$ T cells of MHC class II. T cells, which occur as two classes, the $CD4^+$ T cells, and the $CD8^+$ T cells play a major role in the pathology of multiple sclerosis (27).
- Decreases expression of proteases by T cells.
- Inhibition of maturation of dendritic cells (28,29).

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