Disease-modifying treatments include the following:

A. Selective immunomodulation
   1. Glatiramer acetate (Copaxone)
   2. Dimethyl fumarate (Tecfidera)

B. Nonspecific immunomodulation
   1. IFN-β-1a (Avonex, Plegridy, Rebif)
   2. IFN-β-1b (Betaseron, Extavia)

C. Selective adhesion molecule inhibitor
   1. Natalizumab (Tysabri)

D. Sphingosine 1-phosphate receptor modulator
   1. Fingolimod (Gilenya)

E. Immunosuppression
   1. Mitoxantrone (Novantrone)
   2. Alemtuzumab (Lemtrada)

F. Immunomodulatory agent
   1. Teriflunomide (Aubagio)

Immunomodulating and immunosuppressive agents have an effect on some aspect of the immune response; with some agents, the effect is general, with others more specific.

A. IFN-β thought to:
   1. Inhibit T cell proliferation
   2. Inhibit synthesis of inflammatory cytokines (IFN-γ, TNF-α)
   3. Downregulate expression of MHC class II molecules induced by IFN-γ
   4. Reduce antigen presentation to T cells
   5. Downregulate adhesion molecules and promote BBB integrity
   6. Stimulate production of IL-10, a regulatory cytokine

B. Glatiramer acetate thought to:
   1. Induce suppressor T cells
   2. Be structurally similar to myelin basic protein. When glatiramer acetate (GA) induced T cells are presented to myelin basic protein (MBP) in the CNS, it is felt that they are stimulated to proliferate and release cytokines (TGF-β, IL-4, IL-10).

C. Mitoxantrone thought to:
   1. Produce a broad spectrum of immunosuppression with some B-cell suppression.