Abstract: Glatiramer acetate is a synthetic, random copolymer widely used as a first-line agent for the treatment of relapsing-remitting multiple sclerosis (MS). While earlier studies primarily attributed its clinical effect to a shift in the cytokine secretion of CD4+ T helper (T(h)) cells, growing evidence in MS and its animal model, experimental autoimmune encephalomyelitis (EAE), suggests that glatiramer acetate treatment is associated with a broader immunomodulatory effect on cells of both the innate and adaptive immune system. To date, glatiramer acetate-mediated modulation of antigen-presenting cells (APC) such as monocytes and dendritic cells, CD4+ T(h) cells, CD8+ T cells, Foxp3+ regulatory T cells and antibody production by plasma cells have been reported; in addition, most recent investigations indicate that glatiramer acetate treatment may also promote regulatory B-cell properties. Experimental evidence suggests that, among these diverse effects, a fostering interplay between anti-inflammatory T-cell populations and regulatory type II APC may be the central axis in glatiramer acetate-mediated immune modulation of CNS autoimmune disease. Besides altering inflammatory processes, glatiramer acetate could exert direct neuroprotective and/or neuroregenerative properties, which could be of relevance for the treatment of MS, but even more so for primarily
neurodegenerative disorders, such as Alzheimer's or Parkinson's disease. In this review, we provide a comprehensive and critical overview of established and recent findings aiming to elucidate the complex mechanism of action of glatiramer acetate.

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