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LECTURE

Effect of Glatiramer Acetate on Peripheral Blood Brain-Derived Neurotrophic Factor and Phosphorylated TrkB Levels in Relapsing-Remitting Multiple Sclerosis

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Abstract

Glatiramer acetate (GA) is one of the most widely used disease-modifying drugs for the treatment of relapsing-remitting multiple sclerosis; is assumed to have inductor effects on neurotrophic factor expression. One of these neurotrophic factor systems is the brain-derived neurotrophic factor (BDNF)/receptor tyrosine kinase B (TrkB) pathway. Peripheral blood is thought to contain soluble BDNF, and some blood cells express TrkB. We attempted to determine whether GA treatment leads to changes in plasma BDNF levels and TrkB activation. Such a phenomenon are relapsing-remitting multiple sclerosis patients is significantly reduced; GA treatment is not influencing peripheral BDNF levels, after one year of sustained therapy, not from the point of view of total free BDNF nor the phosphorylated TrkB.

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