

Fingolimod and its Treatment in Multiple Sclerosis

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Fingolimod (FTY720) is a sphingosine-1-phosphate-receptor modulator that is administered orally, which is currently being evaluated for the multiple sclerosis treatment.

Fingolimod (previously FTY720) is an immunomodulator that's utilized in combination therapy with other immunosuppressant drugs within the prophylaxis of acute rejection after solid organ transplantation and for the treatment of multiple sclerosis. It is phosphorylated intracellularly to fingolimod phosphate, which binds to the sphingosine-1-phosphate receptor, and reduces the recirculation of lymphocytes from lymph nodes to blood and peripheral tissue, including the graft site. This results in reduced infiltration of probably autoaggressive lymphocytes into the central systema nervosum. Preclinical findings also suggest that fingolimod may promote neuroprotective and repair processes within the central systema nervosum by modulating sphingosine-1-phosphate receptors expressed on neural cells.

Mechanism of Action

Fingolimod is a small molecule. This molecule springs from a natural product made by the fungus, *Isaria sinclairii*. The natural product is myriocin. Fingolimod acts on T cells that reside in lymph nodes, and prevents these T cells from exiting the lymph nodes, where the end-effect is preventing them from migrating to the CNS. In detail, fingolimod acts on a membrane-bound protein of the T cell, namely, the sphingosine-1-phosphate receptor (48,49).

reasoning with evidence-based practice in addressing the individualized symptoms of the person and assists in meeting their specific goals [7]. So how do clinicians address these physical impairments while overcoming a loss of physical contact with our patient cohort?

Telerehabilitation is an emerging cost-effective method of bridging the gap of standard face-to-face clinician-patient care by enabling service delivery in their home environment. In the case of neurological conditions, the best rehabilitative strategies aim to stimulate the brain through interactions with the environment. Telerehabilitation grants allied health professionals the opportunity to deliver tailored interventions based on the individual's needs and interactions with their native environment. This has been shown. 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 little subset of T cells, not all T cells, becomes trapped within the lymph nodes.

Multiple Sclerosis

Fingolimod's pharmacologic activity is targeted towards lymphocyte migration out of lymph nodes. This action is highly dependent on the engagement of a G-protein-coupled receptor, S1P1, present on the surface of the lymphocytes. Fingolimod is structurally almost like S1P and may function as an agonist by engaging four of the five known S1P receptors (S1P1, S1P3, S1P4, S1P5). This results in a discount in activated T cells that are ready to exit the lymph gland and subsequently cross the blood-brain barrier to exert their potential pathogenic effects on perivascular tissue. Studies have indicated the potential for S1P receptors to be present on other cells, including neurons, microglial cells, oligodendrocytes, and astrocytes, suggesting a putative role for fingolimod in influencing myelin repair, modulating survival of oligodendrocyte progenitor cells, and directing astrocyte migration and proliferation.

Pediatric Multiple Sclerosis

Fingolimod, or FTY720, was the primary oral immunosuppressant approved by the FDA for MS. Fingolimod affects the sphingosine-1-phosphate receptor and sequesters lymphocytes in lymph nodes. The TRANSFORMS double-blind, double-dummy randomized controlled trial in adults demonstrated the superior effect of fingolimod as compared to IFN- β . In patients who received fingolimod, adverse events included cardiac arrhythmias, macular edema, increased liver-enzymes, carcinoma, and herpes viral infections. One published retrospective review of pediatric MS patients treated with fingolimod demonstrated reduced relapse rate. No adverse side effects were reported but follow-up time was on average 8.6 months. A randomized double-dummy active comparator study of fingolimod to IFN- β is currently underway.

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