The Sphingosine-1-Phosphate Receptor is involved with the immune system's central memory T-cells (TCM) (Vermersch 2018). Fingolimod, a Multiple Sclerosis drug, binds to the S1P receptor, causing the recruitment of β-arrestin, which induces receptor internalization. This internalization prevents TCM migration into the CNS, a crucial part of autoimmune reactions (Park & Im, 2017). The objective of this project is to model the mechanism of how Fingolimod acts as a receptor modulator when it interacts with the sphingosine-1-phosphate receptor. After initial research with academic journals and prototyping with Craft Kits, TinkerCAD, and repeated 3D print tests, the final model illustrates the inward movement and internalization of the receptor-drug complex within the TCM once the drug binds. The internalization of the S1P receptor and Fingolimod drug complex in the TCM blocks further signaling and, thus, prevents circulating lymphocytes from entering the CNS. This reduces the autoimmune reaction that typically leads to myelin sheath degradation in Multiple Sclerosis. Our model can facilitate understanding the mechanisms between the Fingolimod drug, the S1P receptor, and the TCM to guide future research toward treating other T-cell-mediated autoimmune diseases.