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Overview:

Multiple sclerosis is a tragic disease where individuals, typically in their third or fourth decade of life, begin experiencing degradation of their nervous system. As a result, symptoms range from impacting the physical capabilities of the individual, such as abnormal sensations, vision problems, motor dysfunction, and paralysis, to the cognitive abilities of the individual, such as slower processing speed and memory deficiencies. Multiple sclerosis comes in many stages but is characterized by the body attacking the nervous system, invoking damage and inflammation, specifically the myelin sheath that insulates the axon. This breakdown not only takes out the myelin, but also begins degrading the axon after the layer of protection is removed. Once the demyelination of the neuron has occurred, the body attempts to correct for the deficiency and redistribute sodium channels along the axon and even tries to remyelinate the axon of still functional neurons. This model explores this attempt by Schwann cells to remyelinate the axon to restore the insulation created by the myelin sheath and the efficient nature of the neuron's signal propagation abilities. The model also looks at the effects of the sodium channels throughout the cell. There is the ability to analyze the needed sodium channel density needed in order to allow for an action potential propagation and whether the redistribution of sodium channels along the axon can restore conduction within the neuron. This model will allow us to alter the geometric parameters of the neuron, the channel densities and distributions, the myelin coverage, and the properties of the membrane to better understand how multiple sclerosis destroys the biological capabilities of the neuron.

References:

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