Multiple sclerosis is a disease in which the fatty myelin sheaths around the axons of the brain and spinal cord are damaged, leading to demyelination and scarring as well as a broad spectrum of signs and symptoms. The symptoms of MS can affect all parts of the body. The cause of MS is still unknown, but it most likely is some combination of genetic, environmental, and infectious factors.

Over the last 2 decades the treatments for Multiple Sclerosis have evolved rapidly. The introduction of interferon-β, and glatiramer acetate have demonstrated efficacy in reducing relapse rates and MRI lesion burden, as well as in delaying the accumulation of disability. Depending upon the condition and symptoms of the patients MS will dictate what treatment they shall receive. There appear to be even more appealing treatments for MS on the horizon. Some such treatments are monoclonal antibodies, chimeric molecules, oral therapies, and hematopoietic stem cells.

Monoclonal antibodies are monospecific antibodies that are the same because they are made by one type of immune cell which are all clones of a unique parent cell. There are numerous drugs in use that meet this definition. The greatest success was seen using Alemtuzumab. It reduced the relapse rate by 74% and reduced the risk of sustained disability by 71%. Alemtuzumab also demonstrated superiority on MRI, with a greater reduction lesion load.

A chimeric molecules is a molecule containing sequences derived from two different genes; specifically, from two different species. Abatacept is a chimeric molecule composed of a human CD152 molecule and an IgG tail. The CD152 domain binds to CD80 and CD86 on antigen-presenting cells, blocking their ability to bind CD28 on to T-cells, which would otherwise lead to T-cell activation. They have been unable to properly test this drug due to an imbalance in the test group.

Oral therapy would be the change from self-injection prescribed therapy to an oral medication. Oral fingolimod is currently in phase III evaluation in relapsing–remitting and progressive forms of MS. The relapse rate showed a relative reduction of 53% in the 5.0-mg group and 55% in the 1.25-mg group. However, there were no significant differences between placebo and treatment groups in EDSS score at 12 months. A later publication of 24-month data, showed continued benefit to patients in terms of less MRI activity and lower relapse rate and, additionally, there were no further serious adverse events in the safety evaluation.

There are also even more ideas for what to do with MS on the drawing board. One such idea is to prevent the degeneration of axons and/or promote remyelination. One potential target for promotion of remyelination is the LINGO-1 protein. It has been recently discovered that this molecule is a potent inhibitor of oligodendrocyte progenitor cells and thus an inhibitor of myelination.

Work Cited
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