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Short Communication

Treatment of Charcot-Marie-Tooth Disease by Stem Cell Therapy

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DESCRIPTION

Charcot-Marie-Tooth illness has no known treatment. Neither have scientists developed any approved medicines to address the inherited neurological disorder's underlying causes. CMT is caused by a gene mutation, and several mutations are linked to it. A defective gene results in the production of an aberrant protein or none at all. Extra copies of a gene are sometimes involved in mutations, resulting in protein overproduction. This causes a disturbance in nerve signals between the brain and muscles, resulting in numbness and weakness, which usually begins in the feet.

Gene therapy and stem cell therapy are two treatments being developed by researchers for CMT. Gene therapy involves giving a fully functional copy of a disease-causing gene. Healthy cells are delivered to stimulate nerve regeneration in stem cell treatment [1,2].

In a mouse model with Charcot-Marie-Tooth type 1A, Mesenchymal Stem Cells (MSCs) can improve myelin-producing cells and the function of peripheral nerves, which supply mobility and feeling to the arms and legs. MSCs' therapeutic advantages are achieved through the release of specific cytokines, which are chemicals that mediate and regulate immunological and inflammatory processes.

CMT1A, the most frequent subtype of CMT type 1, is caused by mutations that create a duplication in the PMP22 gene's DNA sequence. This gene produces the PMP22 protein, which is a crucial component of the myelin sheath that protects nerve cells and allows them to communicate properly. Schwann cells, a type of cell found in peripheral nerves, produce myelin, a fatty coating [3].

The analysis showed that, indeed, the messenger RNA levels the molecule generated from DNA that serves as a template for protein production of both genes were significantly increased in PMP22-overexpressing cells grown together with MSCs.

According to the researchers, mesenchymal stem cells may guard against the negative consequences of PMP22 overexpression and boost myelin gene production, which may relieve the symptoms of demyelinating CMT [4].

Further research revealed that MSCs' therapeutic advantages are likely linked to their anti-inflammatory capabilities. They discovered that MSCs cultured with PMP22-overexpressing Schwann cells produced a lot of Growth Differentiation Factor-15 (GDF-15) and Amphiregulin (AREG). When compared to untreated Schwann cells, GDF-15 significantly increased the number of viable PMP22-overexpressing Schwann cells.

MSCs are gaining popularity as a potential treatment option for a variety of ailments, including neurological disorders. MSCs are anti-inflammatory, neuroprotective, and regenerative cells found in a variety of organs including the umbilical cord, bone marrow, and adipose tissue [5].

Furthermore, GDF-15 reduced the number of cells that died from 10.5% to 6.3%. GDF-15 also boosted the activity of the genes coding for Oct6 and MPZ in PMP22-overproducing Schwann cells. Because all of these data came from laboratory (*in vitro*) experiments, the researchers decided to look into the therapeutic effects of AREG and GDF-15 in a CMT1A animal model.

CONCLUSION

Overall, these data indicate MSCs' therapeutic effects in CMT1A, specifically via AREG and GDF-15 release, in reducing myelin-producing cell loss and increasing myelin formation in peripheral nerves. The researchers stated, "Further understanding of the underlying processes of GDF-15 and AREG in myelination might provide a firm basis for the development of viable therapeutics against demyelinating CMT.

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