Pharmacological fortification and/or restoration of protective nerve coverings via a novel therapeutic target

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OTHER INFORMATION

CATEGORIZED AS
- Medical
  - Disease: Central Nervous System
  - New Chemical Entities, Drug Leads
  - Therapeutics

RELATED CASES
2022-731-0
BRIEF DESCRIPTION

Researchers at UC Irvine have discovered a novel mechanism by which restoration of protective nerve coverings fails in degenerative disease like multiple sclerosis. While therapeutics to slow disease progression exist, there are currently none aimed at preventing or restoring damage to nerve coverings.

SUGGESTED USES

- Treatment of diseases in which damage to nerve coverings (myelin) occurs.
- Developmental fortification of nerve coverings by supplementation in baby formula.

FEATURES/BENEFITS

- Potential cure: Disease-slowing therapies exist, but there are currently no cures. This treatment holds potential to prevent and/or cure diseases in which nerve coverings are damaged.
- Novel mechanism of action: This treatment targets a newly identified molecular pathway.
- Safe: The therapeutic has previously been shown to be highly tolerable in preclinical animal models and in humans.

TECHNOLOGY DESCRIPTION

There are currently no cures for diseases involving damage to protective nerve coverings, a process called demyelination. One of the most famous of these diseases is multiple sclerosis (MS). After disease onset, immune cells create an inhibitory environment for remyelination by a different cell type known as OPCs. Specifically, these immune cells are thought to prevent expression of sugars on the cell surface of OPCs, a process essential for their normal cell function. Therefore, therapeutics aimed at promoting expression of these sugars on the surface of OPCs, and thereby restoring their myelinating functions, represent a potential cure for these diseases.

Researchers at UC Irvine have shown that promoting expression of these sugars can developmentally fortify newborn rodent nerve coverings or restore the myelinating functions of OPCs in preclinical rodent models of demyelination disease. They also show that in MS patients, serum levels of these sugars inversely correlated with demyelination and damage (i.e. less sugar, more damage). Importantly, this sugar is known to be highly safe in humans, with large intravenous doses (20g and 100g) in humans demonstrating no toxicity issues or alterations in blood glucose or insulin. The therapeutic is currently being tested in Phase I clinical trials and holds potential to be the first true cure for demyelinating disease like MS.

STATE OF DEVELOPMENT

Currently in Phase I, partially complete clinical trials

RELATED MATERIALS

- N-acetylglucosamine drives myelination by triggering oligodendrocyte precursor cell differentiation. - 09/25/2020

PATENT STATUS

Patent Pending