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ANTAGONIZING THE ENDOTHELIN B RECEPTOR TO IMPROVE AGING-DEPENDENT NEURONAL REGENERATIVE DECLINE

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Value proposition: *A cost-effective, drug-based therapy targeting the endothelin B receptor (ETBR) to promote peripheral nerve regeneration, offering improved recovery and compatibility with existing treatments.*

Technology Description

Researchers at Washington University in St. Louis have developed a method to promote axonal regeneration following injury to portions of the peripheral nerve by antagonizing the activities of the endothelin B receptor (ETBR). Currently, there is no effective drug-based treatment of nerve injuries on the market. Additionally, functional recovery from nerve injury in humans remains limited and the ability of injured neurons to regenerate their axons declines with age. Existing treatments include surgery and electrical stimulation, while ongoing research of nerve regeneration involves scaffolding and biomaterial-based conduits.

This new drug-based approach for nerve regeneration is more cost effective than current treatments, ensures patient compliance, and can be combined with existing non-drug-based approaches.

Stage of Research

Conducted studies on mice that show endothelin signaling in Satellite glial cells (SGCs), which completely envelop the sensory neuron soma and limits axon regenerative capacity of sensory neurons. In addition, the researchers have found that blocking endothelin signaling with the FDA-approved compound Bosentan reverts the age-dependent axon regenerative decline along peripheral nerves in mice.

Applications

- Axonal Regeneration.

Key Advantages:

- Enhances axonal regrowth following injury.
- Counteracts the age-dependent decline of neuro-regeneration.

Patents:

Application filed

Related Web Links - Valeria Cavalli Profile; Cavalli Lab