Mechanism to Enhance Anterograde Microtubule-Based Transport in Neurodegenerative Diseases

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ABSTRACT

Researchers at the University of California, Davis have developed polypeptide compositions of the peripheral nervous system tau isoform ("big tau") that appear to selectively enhance kinesin-3 motor activity and bias intracellular transport toward anterograde direction to treat diseases linked to impaired axonal transport.

FULL DESCRIPTION

The invention provides novel polypeptides derived from the peripheral nervous system isoform of tau protein, known as big tau, and engineered sequence variants that modulate microtubule-based motor protein activity. These compositions can enhance kinesin-3 (KIF1A) activity while inhibiting dynein-mediated retrograde transport, effectively biasing intracellular cargo movement toward the synapse, i.e., the anterograde direction. Such modulation addresses the critical challenge of maintaining neuronal function by improving intracellular cargo transport along microtubules. Big tau and its variants act on neurons to enhance forward motor activity, reduce retrograde interference, and potentially counteract transport defects implicated in a variety of neurodegenerative and neurodevelopmental diseases.

APPLICATIONS

- Potential therapeutics for neurodegenerative diseases including amyotrophic lateral sclerosis (ALS) and Alzheimer’s disease.
- Possible treatment of inherited peripheral neuropathies like Charcot-Marie-Tooth disease and hereditary spastic paraplegia.
- Targeted intervention for rare kinesin-associated neurological disorders (KAND) and other neurodevelopmental disorders.
- Research tools for studying intracellular transport modulation and neuron function.
- Drug development platforms leveraging polypeptide and genetic vector delivery to affected neurons.
- Potential applications in regenerative medicine addressing neuronal maintenance and repair through improved intracellular trafficking.

FEATURES/BENEFITS

- Accelerates kinesin-3 (KIF1A) motor velocity and increases microtubule landing rate.
- Suppresses dynein-driven retrograde transport to shift cargo movement toward anterograde directionality.
- Leverages native peripheral nervous system “big tau” and optimizes engineered variants to maximize functional modulation of axonal transport.

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OTHER INFORMATION

KEYWORDS

alzheimer's disease, amyotrophic lateral sclerosis, axonal transport, charcot-marie-tooth disease, kinesin-3, microtubule motor proteins, neurodegenerative disease, neurodevelopmental disorder, tau protein, therapeutic peptides

CATEGORIZED AS

- **Biotechnology**
  - Health
- **Medical**

▶ Enables flexible development and delivery by supporting multiple nucleic acid formats, vectors, and host-cell platforms.

▶ Improves function of mutant KIF1A motors implicated in kinesin-related neurological disorders (KAND).

▶ Restores disrupted axonal transport that drives neurodegenerative and neurodevelopmental disease mechanisms.

▶ Mitigates progressive motor, sensory, cognitive, and autonomic decline by improving intracellular cargo movement.

▶ Targets the core motor-driven transport defect in conditions where existing treatments largely fail to address underlying pathology (e.g., ALS, Charcot–Marie–Tooth disease, hereditary spastic paraplegia, Alzheimer's disease, and KAND).

▶ Provides a way to selectively boost anterograde transport while minimizing unintended interference with essential retrograde functions.

PATENT STATUS

Patent Pending

▶ Disease: Central Nervous System

▶ Research Tools

▶ Therapeutics

▶ **Research Tools**

▶ Nucleic Acids/DNA/RNA

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