Neuronal Specific Targeting of Caveolin Expression

Tech ID: 21446 / UC Case 2010-117-0

BACKGROUND
Understanding the basic mechanisms of cognitive decline and how the subcellular organization of signaling molecules is altered with cognitive decline could potentially yield novel therapeutic targets for neuronal aging and neurodegeneration.

Cholesterol is a major lipid component of synapses and a limiting factor in synapse activity. Age-related impairments in the biosynthesis, transport, or uptake of cholesterol by neurons in the CNS may adversely affect synaptic circuitry. Moreover, caveolin-1 (Cav-1), a cholesterol binding and resident protein of membrane lipid rafts (MLR; discrete regions of the plasma membrane enriched in cholesterol), organizes and targets synaptic components of the neurotransmitter and neurotrophic receptor signaling pathways to MLR.

TECHNOLOGY DESCRIPTION
UC San Diego scientists have discovered a role for Cav-1 and MLR in organizing synaptic pro-survival signaling components that are essential for neuroprotection against ischemic injury and neuronal regeneration and for maintaining synapse stabilization and formation. This invention comprises a new method of treating neurodegenerative diseases by targeting increased expression of caveolin-1 protein to brain neurons. For example, up-regulation of caveolin-1 in stem cells transplanted into the brain or spinal cord may enhance new synaptic connections, thereby significantly accelerating regeneration. This represents a novel means by which function of the aged brain, diseased brain, or injured brain can be improved.

INTELLECTUAL PROPERTY INFO
This technology is available for licensing.

RELATED MATERIALS

PATENT STATUS

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Additional Patent Pending