Gene Transfer of Prohibitin for the Treatment of Alzheimer’s Disease and Other Neurodegenerative Diseases

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BACKGROUND

1. Alzheimer’s disease (AD) is associated with mitochondrial dysfunction that may contribute to cognitive impairment;
2. Transgenic mice overexpressing the Swedish mutation of amyloid precursor protein (APP) (Tg2576) exhibit mitochondrial oxidative stress, as well as dysfunction in hippocampal synaptic plasticity and spatial memory;
3. Prohibitin (PHB) is an endogenous protective protein localized to mitochondria that counteracts hypoxic-ischemic neuronal injury by preventing mitochondrial dysfunction and oxidative stress;

INVENTION SUMMARY

This invention discloses prohibitin as a new composition for the treatment of Alzheimer’s disease and other neurodegenerative diseases and also provides gene transfer methods for the treatment of the diseases.

SUMMARY

1. Prohibitin protein levels are reduced in APP mice;
2. PHB viral gene transfer increases PHB expression in hippocampal neurons in APP mice;
3. Hippocampal PHB upregulation is associated with restoration in LTP, improvement of spatial memory, and normalization of locomotor activity in a mouse model of APP overexpression;
4. PHB may have therapeutic potential in AD and other cognitive disorders associated with mitochondrial dysfunction.

COMMERCIAL APPLICATIONS

- Treatment for Alzheimer’s disease and other neurodegenerative diseases and conditions

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