Neurocentria Demonstrates that Elevation of Brain Magnesium Reverses Memory Deficits in Alzheimer Mice

HAYWARD, Calif., September 13, 2014 /PRNewswire via COMTEX/ -- Scientists at the Center for Learning and Memory, Tsinghua University, Beijing, and the California-based biopharmaceutical company Neurocentria, Inc. (www.neurocentria.com) have demonstrated a novel therapy for reversing memory decline in mice with Alzheimer's Disease. By increasing brain magnesium levels, they find significant cognitive improvement in advanced stage AD mice. The study is the first to demonstrate a mechanism for reversing cognitive decline for advanced stage AD mice and is also the first to show an effective long-term treatment for early stage AD mice.

"We found that the elevation of brain magnesium can prevent cognitive decline in Alzheimer's Disease model mice," explained Magceutics founder Guosong Liu, a professor at Tsinghua University and senior investigator on the study. The report, which appeared in the September 13th issue of Molecular Brain, shows that a newly developed magnesium compound known as L-Threonic acid Magnesium salt (L-TAMS, aka Magnesium-L Threonate) can prevent cognitive impairment when administered to mice with early stage AD. The treatment was shown to remain effective for at least 16 months. Additionally, L-AMS significantly improved memory and cognition when given to advanced stage AD mice.

Liu and colleagues believe that because the loss of neuronal connections in brain regions critical for memory function is major hallmark of Alzheimer's Disease, preventing those losses can lead to new treatment options. In earlier studies, they determined the principle for controlling the density and plasticity of connections (synapses) between neurons in the hippocampus, the brain region that processes memory. Those studies revealed that the elevation of brain magnesium can selectively reduce background calcium within synapses and thereby enhance synaptic plasticity and density. Further work by Liu and colleagues revealed that elevating magnesium can reverse memory decline in aging rats.

The team's new study builds on those findings and sheds light on the mechanism by which increased magnesium levels may act to protect the brain from neurodegeneration. To explore the protective mechanism, they investigated major signaling pathways critical for synapse function and memory formation. They found that elevated ABeta leads to widespread activation of calcium-dependent signaling molecules that contribute to neuronal degeneration. The activation of these molecules dampens the activity of proteins that are critical for synapse remodeling and memory function - effects that the researchers discovered can be overcome with L-TAMS therapy.

Neurocentria plans to launch a clinical trial in conjunction with Stanford University later this year to determine whether L-TAMS can reverse memory decline for human patients with Alzheimer's Disease.