SDSU researcher explores novel protein as potential target in Alzheimer's treatment

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(PhysOrg.com) -- A South Dakota State University researcher and his colleagues elsewhere have discovered a previously unreported mitochondrial protein that interacts with a protein known to play a role in Alzheimer's disease.

The discovery adds to what is known about the disease as researchers continue to search for ways to treat it.

The research is reported in June 2010 in the European Journal of Neuroscience, Vol. 31. Assistant professor Hemachand Tummala in SDSU's Department of Pharmaceutical Sciences is the lead author of the journal article, which builds on work he carried out while at the University of Memphis. His co-authors are Xiaofan Li of the Mayo School of Graduate Medical Education and the University of Tennessee Health Science Center; and Ramin Homayouni of the University of Memphis and the University of Tennessee Health Science Center.

Alzheimer's disease affected 5.5 million Americans in 2009. With a rapidly aging baby boomer population, this number is predicted to go up to 16 million by 2050. Besides patient distress, Alzheimer's disease also inflicts health care costs to society of $172 billion annually. Currently, Alzheimer's disease has no cure.

"In Alzheimer's disease, there is a protein involved called APP, Amyloid precursor protein. If this protein is mutated, it causes early onset Alzheimer's disease. We don't know exactly what this protein does,"

Tummala said. “One thing is very well documented in Alzheimer’s disease, mitochondria in a cell are damaged. They lose their function. This happens long before the appearance of symptoms. So there is a theory that mitochondria play a big role in the disease progression.”

Mitochondria are membrane-enclosed structures in cells that generate the cell’s supply of energy and are involved in processes such as signaling, cell death, cell growth, and possibly aging.

Neurons are special cells in the brain that help the body to carry out functions associated with hearing, seeing, moving, remembering information, and learning new things. In Alzheimer’s disease neurons that function in learning and memory die. That’s why Alzheimer’s disease symptoms include memory loss and the other behavioral cognitive problems.

Tummala said he and his colleagues’ important finding is that APP binds to the mitochondrial protein, NIPSNAP1. NIPSNAP1 is specifically seen in neurons and it may have a role in neuronal death in Alzheimer’s disease. “Taken together, our data suggest that APP directly interacts with the neuron-specific mitochondrial protein NIPSNAP1, and may thereby regulate mitochondrial function in neurons,” the scientists note in their journal article.

Tummala said his next step, working with his University of Memphis colleague, Dr. Ramin Homayouni, will be a closer examination of NIPSNAP1 using mouse models. Tummala already has won a grant of $96,926 through South Dakota’s 2010 Competitive Research Grant Program, accompanied by an SDSU match of $98,978. The grant will help fund his work learning more about the protein as a potential drug target for treating Alzheimer’s disease.

“This is still at the preliminary stages. If everything goes well, if we

establish the link, this may in the future become a new therapeutic target," Tummala said. "Our hypothesis is that mitochondria are damaged long before the appearance of symptoms. If you could stop that mitochondrial damage, it may slow down neuronal death and halt the disease progression. But that would be far in the future, not now."

Provided by South Dakota State University