Enzyme may be a key to Alzheimer's-related cell death

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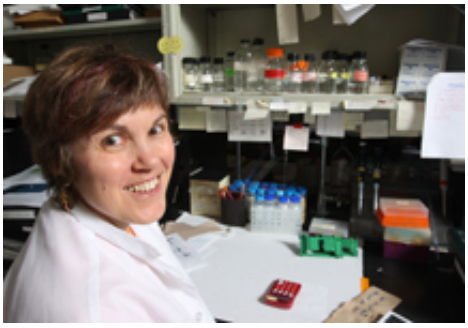

Sandra Rossie found that an enzyme blocks a mechanism that can lead to neural cell death. (Purdue Agricultural Communication photo/Tom Campbell)

(PhysOrg.com) -- A Purdue University researcher has discovered that the amount of an enzyme present in neurons can affect the mechanism thought to cause cell death in Alzheimer's disease patients and may have applications for other diseases such as stroke and heart attack.

Sandra Rossie, a professor of biochemistry, found that increasing the amount of protein phosphatase 5, or PP5, in rat neural cells resulted in less cell death associated with reactive oxygen species, which chemically damage cell molecules. Conversely, decreasing PP5 caused greater cell death. The results of Rossie's study are published in the early online version of *The Journal of Neurochemistry*.

Alzheimer's, a degenerative neurological disease affecting around 5

million people, results in memory loss and dementia. One theory on the cause of Alzheimer's is that overproduction of certain forms of amyloid beta protein by neurons leads to the generation of reactive oxygen species, which activate stress pathways.

"If stress pathways remain active for a prolonged period, the cell will die," Rossie said.

Rossie's lab found that PP5 overexpression prevents neuronal death by amyloid beta and shuts off the stress pathways. When reactive oxygen that wasn't created by amyloid beta was used on the cells, the results were the same. In contrast, neurons with reduced PP5 are more sensitive to death caused by amyloid beta.

"That suggests to us that PP5 protects neurons from cell death induced by reactive oxygen species, not just the presence of amyloid beta," Rossie said. "This means that PP5 may protect against other health problems involving reactive oxygen species as well, such as stroke and heart attacks."

It is possible, Rossie said, that finding a way to increase PP5 activity could help prevent the loss of neurons by amyloid beta.

Rossie said PP5 also could play a role in inhibiting other responses of neurons to amyloid beta. Her lab will work to determine which pathways PP5 affects, and which of those is most responsible for neural protection by PP5.

Provided by Purdue University (news : web)