

Astrocytes as a novel target in Alzheimer's disease

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Alzheimer's disease is a severe neurodegenerative disease that affects 45% of people over 85 years of age. The research teams of Prof. Jin-Moo Lee at Washington University in Saint Louis, USA, and Prof. Milos Pekny at Sahlgrenska Academy in Gothenburg, Sweden, have identified astrocytes as a novel target for the development of future treatment strategies. The results have just been published in the *FASEB Journal*.

Astrocytes are known as cells that control many functions of the healthy as well as diseased brain, including the control of regenerative responses.

In patients suffering from Alzheimer's disease, astrocytes in the vicinity of <u>amyloid plaques</u> and degenerating neurons become hyperactive.

Until now, many researchers considered this astrocyte hyperactivity in the brains of Alzheimer's disease patients as negative and contributing to the progression of this devastating disease.

The current study generated groundbreaking data with important implications. The US and Swedish research teams used a mouse model of Alzheimer's disease in which they genetically reduced astrocyte hyperactivity. They found that such mice developed more amyloid deposits and showed more pronounced signs of neurodegeneration than mice with normal response of astrocytes.

This suggests that astrocyte response to the disease process slows down the disease progression.



"We are truly exited about these findings. Now we need to understand the mechanism underlying the beneficial role of hyperactive astrocytes in Alzheimer's disease progression. Understanding this process on a molecular level should help us to design strategies for optimization of the astrocyte response," says Prof. Milos Pekny.

"We see that astrocyte hyperactivity in Alzheimer's disease brains is tightly connected to activation of microglia, the brain's own <u>immune</u> <u>cells</u>. This implies that the two cell types communicate to mediate a coordinated response to disease states," says Prof. Jin-Moo Lee.

This international collaborative team of neuroscientists is pursuing further studies to understand molecular mechanisms by which astrocytes prevent the deposition of amyloid plaques in Alzheimer's disease.

More information: Andrew W. Kraft et al. Attenuating astrocyte activation accelerates plaque pathogenesis in APP/PS1 mice, *FASEB J* fj.12-208660; published ahead of print October 4, 2012, doi:10.1096/fj.12-208660

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