Amyloid beta protein gets bum rap

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While too much amyloid beta protein in the brain is linked to the development of Alzheimer's disease, not enough of the protein in healthy brains can cause learning problems and forgetfulness, Saint Louis University scientists have found.

The finding could lead to better medications to treat Alzheimer's disease, said John Morley, M.D., director of the division of geriatrics at Saint Louis University and the lead researcher on the study.

"This research is very exciting because it causes us to look at amyloid beta protein in a different way," Morley said.

"After 20 years of research, what we found goes totally against long-standing beliefs about amyloid beta protein. Our results indicate that amyloid beta protein itself isn't the bad guy. The right amount of amyloid beta protein happens to be very important for memory and learning in those who are healthy."

Researchers found that young, healthy mice that received low doses of amyloid beta protein showed improvement in recognizing objects and successfully navigating through a maze. Conversely, mice that received a drug that blocked amyloid beta protein had learning impairment.

"You can't totally wipe out amyloid beta protein. If you do this, you are going to create dementia," Morley said. "In treating Alzheimer's disease, we have to be careful not to lower amyloid beta too much because it will cause as many problems as if you had an excess of amyloid beta

protein."

In short, Alzheimer's disease is connected to too much of a good thing. The right amount of amyloid beta in healthy brains actually enhances learning and memory rather than impairs it.

"Excess production of amyloid beta results in memory deficits," Morley said. "Overall, we believe these studies strongly suggest that the physiological role of amyloid beta is to enhance learning and memory.

These findings are important in understanding the optimal design of drugs to treat Alzheimer's disease."

The research, conducted in an animal model, is published in electronic edition of the Sept. 11 edition of the *Journal of Alzheimer's Disease.*

Source: Saint Louis University