New research dashes notions of benign brain plaque

May 22 2012

(Medical Xpress) -- The time may have come to scrub the idea that brain plaque — deposits of protein that clog passages between brain cells — might not be all that bad.

University of Florida researchers have discovered that people with no signs of dementia during their lives, even though their brains contained the debris typical of Alzheimer's disease, probably would have experienced health problems had they lived longer, according to a study to appear this week in the open access journal *Alzheimer's Research & Therapy*.

Scientists suspect patients who experience relatively few cognitive problems even with a substance called amyloid beta protein accumulating in their brains — the hallmark of Alzheimer's disease — might collect a less toxic form of the so-called brain plaque.

But UF College of Medicine scientists with colleagues from the Mayo Clinic in Jacksonville found few differences when they compared the postmortem brain tissue of Alzheimer's patients with that from people who accumulated plaque without symptoms, a condition known as pathological aging.

"Pathological aging may be early Alzheimer's disease rather than a benign form of amyloid protein deposition, or it may be patients with PA are resistant to the toxic effects of the amyloid plaques," said Dr. Todd Golde, director of the UF's Center for Translational Research in

Neurodegenerative Disease. “It will be important to understand the differences between these two neurodegenerative pathologies in treatment and prevention efforts.”

Alzheimer’s disease is characterized by severe loss of neurons in brain regions important for learning and memory because of overproduction of amyloid beta protein. In a healthy brain, these protein fragments are broken down and eliminated. But when they accumulate, scientists believe amyloid plaque interferes with the brain’s ability to generate new cells and contributes to tangles — twisted masses of protein fibers within the brain cell.

The researchers found similar amounts of insoluble amyloid in Alzheimer’s and pathologically aged brain tissue, with elevated levels in both types of abnormal tissue compared with healthy brain tissue. Researchers also found a great deal of similarity and overlap in the subtypes of amyloid protein, according to Golde, who is also affiliated with UF’s McKnight Brain Institute.

Experimental models suggest that therapies that target these proteins may be effective in preventing or delaying disease development. Without treatment or prevention breakthroughs, a projected 7.7 million patients in the U.S. will have Alzheimer’s by 2030, according to the Alzheimer’s Association. That number will grow to between 11 million and 16 million by 2050.

Provided by University of Florida