Antioxidant has potential in the Alzheimer's fight, researchers find

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(Medical Xpress) -- When you cut an apple and leave it out, it turns brown. Squeeze the apple with lemon juice, an antioxidant, and the process slows down.

Simply put, that same "browning" process-known as oxidative stress-happens in the brain as Alzheimer's disease sets in. The underlying cause is believed to be improper processing of a protein associated with the creation of free radicals that cause oxidative stress.

Now, a study by researchers in the University of Georgia College of Pharmacy has shown that an antioxidant can delay the onset of all the indicators of Alzheimer's disease, including cognitive decline. The researchers administered an antioxidant compound called MitoQ to mice genetically engineered to develop Alzheimer's. The results of their study were published in the Nov. 2 issue of the Journal of Neuroscience.

According to the Alzheimer's Society, more than 5 million Americans currently suffer from the neurodegenerative disease. Without successful prevention, almost 14 million Americans will have Alzheimer's by 2050, accounting for healthcare costs of more than $1 trillion a year.

Oxidative stress is believed to cause neurons in the brain to die, resulting in Alzheimer's. Study author James Franklin, an associate professor of pharmaceutical and biomedical sciences, has studied neuronal cell death and oxidative stress at UGA since 2004.

"The brain consumes 20 percent of the oxygen in the body even though it only makes up 5 percent of the volume, so it's particularly susceptible to oxidative stress," said Franklin, coauthor of the study along with Meagan McManus, who received her Ph.D. in neuroscience from UGA in 2010.

The UGA researchers hypothesized that antioxidants administered unsuccessfully by other researchers to treat Alzheimer's were not concentrated enough in the mitochondria of cells. Mitochondria are structures within cells that have many functions, including producing oxidative molecules that damage the brain and cause cell death.

"MitoQ selectively accumulates in the mitochondria," said McManus, who is now studying mitochondrial genetics and dysfunction as a postdoctoral researcher at Children's Hospital of Philadelphia.

"It is more effective for the treatment to go straight to the mitochondria, rather than being present in the cell in general," she said.

Although he had not previously conducted research on Alzheimer's disease, Franklin was moved to approve McManus' research proposal to take his laboratory research in a more clinical direction in part because of her family's history with the disease.

"Two of my grandparents had Alzheimer's disease, but they presented with it very differently. While my granddad often couldn't remember who we were, he was still the same soulful funnyman I'd always loved. But the disease changed my grandmother's mind in a different way, and turned her into someone we'd never known," said McManus.

"So the complexity of the disease was most intriguing to me. I wanted to know how and why it was happening, and more importantly, how to stop it from happening to other people," she said.

In their study, mice engineered to carry three genes associated with familial Alzheimer's were tested for cognitive impairment using the Morris Water Maze, a common test for memory retention. The mice that had received MitoQ in their drinking water performed significantly better than those that didn't. Additionally, the treated mice tested negative for the oxidative stress, amyloid burden, neural death and synaptic loss associated with Alzheimer's.

More information: The full paper is available online at www.jneurosci.org/content/31/44/15703.full

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