Alzheimer's protein kills nerve cells in nose

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A protein linked to Alzheimer's disease kills nerve cells that detect odors, according to an animal study in the September 28 issue of *The Journal of Neuroscience*. The findings shed light on why people with Alzheimer's disease often lose their sense of smell early on in the course of the disease.

"Deficits in odor detection and discrimination are among the earliest symptoms of Alzheimer's disease, suggesting that the sense of smell can potentially serve as a 'canary in the coal mine' for early diagnosis of the disease," said Leonardo Belluscio, PhD, of the National Institute of Neurological Disorders and Stroke, who led the study. "The changes taking place in the olfactory system as a result of Alzheimer's disease may be similar to those in other regions of the brain but appear more rapidly" he added.

Researchers once thought that protein plaques commonly seen in the brains of people with Alzheimer's disease were responsible for killing off nerve cells, causing disruptions in memory - a hallmark of the disease. The plaques are primarily derived from a protein called amyloid precursor protein (APP). The new study suggests that APP alone - in the absence of the plaques - may be to blame for the death of nerve cells.

In the new study, Belluscio and his colleagues genetically manipulated mice to produce high levels of a mutated version of human APP in olfactory nerve cells. The mutated form of the protein is seen in some people with early-onset Alzheimer's disease, a rare form that runs in families and strikes before age 65.

The researchers found that mice making mutant APP had four times as much olfactory nerve cell death by three weeks of age compared with normal mice. Although the cells that produced mutant APP died, the neighboring cells - that did not have mutant APP - survived. The cell death also occurred in the absence of amyloid plaques. Together, this showed that the cell death was initiated from within the cells making the mutant APP, not from plaques outside the cells. When the researchers blocked the olfactory nerve cells from producing high levels of the mutant precursor protein, more cells lived.

"Reducing APP production suppressed the widespread loss of nerve cells, suggesting that such disease-related death of nerve cells could potentially be stopped," Belluscio said.

"Together, these results support the hypothesis that amyloid proteins are involved in the degeneration of the brain that occurs with Alzheimer's disease," said Donald Wilson, PhD, of New York University School of Medicine and the Nathan Kline Institute for Psychiatric Research, an olfactory system expert who was unaffiliated with the study. "Further, they provide an exciting opportunity to explore how to prevent or reverse the events that lead to cell death and, ultimately, dementia."

More information: www.jneurosci.org/

Provided by Society for Neuroscience

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