Study suggests anti-inflammatory drugs as promising treatments for Alzheimer's disease

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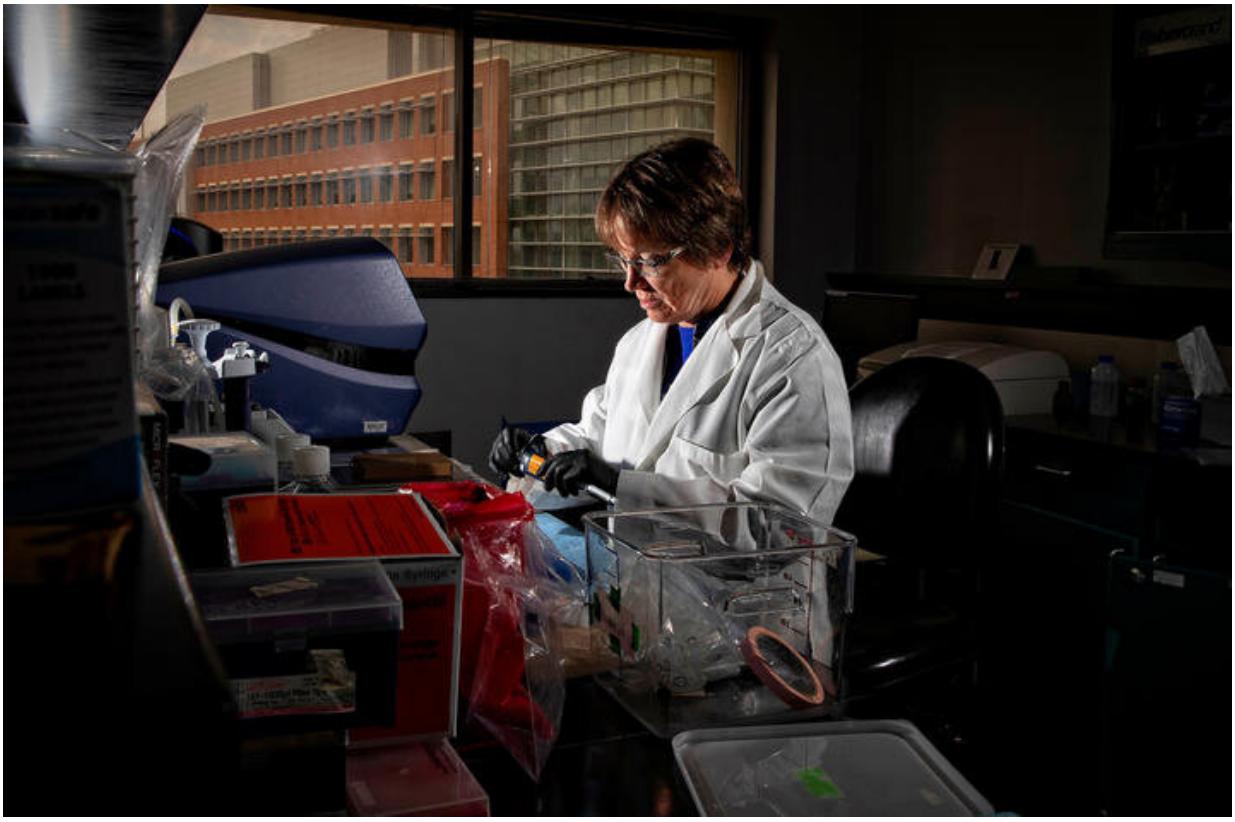

SBCoA director Linda J. Van Eldik, Ph.D., on March 14, 2019. Credit: Mark Cornelison

A recent study from the lab of the University of Kentucky's Sanders-Brown Center on Aging Director Linda Van Eldik, Ph.D., centers around the idea that various anti-inflammatory drugs could be effective

treatments for Alzheimer's disease (AD).

The study focuses on a protein known as p38. Many labs have been working with this protein as a potential target for drug development to treat Alzheimer's disease and other conditions with neuroinflammatory dysfunction.

Van Eldik and her team used genetic techniques to stop the production of p38 in the major immune cell type within the brain, the microglia. They tested the effects of this in an early stage mouse model of AD to determine whether it would alter the trajectory of amyloid plaque formation, a major component of AD pathology.

While the plaques themselves were not affected, the amount of microglia in proximity to these plaques was decreased, suggesting that suppression of microglial p38 may affect their interactions with aspects of AD pathology. The results were published in the journal *PLOS ONE*.

Some classes of anti-inflammatory drugs include p38 inhibitors, which are currently under clinical development and have shown encouraging results during recent human clinical trials. However, it is still not clear when during the disease process these p38 inhibitors should be administered and whether long-term suppression of p38 is harmful.

The findings reported by the Van Eldik lab indicate that early inhibition of p38 may be able to alter interactions between brain immune cells and AD pathology, and they suggest that long-term suppression of p38 does not cause noticeable adverse effects.

More information: David J. Braun et al, Early chronic suppression of microglial p38α in a model of Alzheimer's disease does not significantly alter amyloid-associated neuropathology, *PLOS ONE* (2023). DOI: 10.1371/journal.pone.0286495

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