

# Study suggests personalized medicine may be the future of Alzheimer's disease treatment

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A recently released paper from the Department of Physiology and Sanders-Brown Center on Aging (SBCoA) at the University of Kentucky College of Medicine suggests that your genetics can influence your response to Alzheimer's disease pathology.

The laboratory of Donna Wilcock, Ph.D., professor in the Department of Physiology and SBCoA associate director, investigated inflammation

in [human brain tissue](#) from UK's Alzheimer's Disease Research Center. Brain tissue was analyzed from individuals with different forms of the genetic risk factor, apolipoprotein E (ApoE).

ApoE comes in various forms including ApoE2, ApoE3 and ApoE4. ApoE2 is typically thought of as "protective" and reduces the risk of developing Alzheimer's disease. ApoE3 is the most common form of the gene, while ApoE4 increases the risk and severity of Alzheimer's disease.

This work, led by graduate student Courtney Kloske, found that individuals with ApoE4 had a reduced [inflammatory response](#) to Alzheimer's disease pathology compared to individuals with ApoE3.

"This finding contradicts data found from mouse work, highlighting the need to always confirm studies in both mouse and then [human tissue](#)," Wilcock said.

"Because of the differing response depending on genotype, targeting inflammation in ApoE4 patients may not be the best approach according to our research," said Kloske. "This work shows that your [genetic makeup](#) may influence your response to certain types of treatment for Alzheimer's disease."

The Wilcock lab hopes this work will help contribute to moving treatments closer toward precision medicine.

The study was published in *Journal of Neuropathology & Experimental Neurology*.

**More information:** Courtney M Kloske et al, Inflammatory Pathways Are Impaired in Alzheimer Disease and Differentially Associated With Apolipoprotein E Status, *Journal of Neuropathology & Experimental*

*Neurology* (2021). [DOI: 10.1093/jnen/nlab085](https://doi.org/10.1093/jnen/nlab085)

Provided by University of Kentucky

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