# RESEARCHERS FIND A HINT AT HOW TO DELAY ALZHEIMER'S SYMPTOMS. NOW THEY HAVE TO PROVE IT - ASSOCIATED PRESS

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*Jake Heinrichs hugs his wife, Rachel Chavkin, after speaking about early-stage Alzheimer's disease while inside their home in New York, on Wednesday, March 12, 2025.*

An experimental treatment appears to delay Alzheimer's symptoms in some people genetically destined to get the disease in their 40s or 50s, according to new findings from ongoing research now caught up in Trump administration funding delays.

The early results — a scientific first — were published Wednesday even as study participants worried that politics could cut their access to a possible lifeline.

"It's still a study but it has given me an extension to my life that I never banked on having," said Jake Heinrichs of New York City.

Now 50, Heinrichs has been treated in that study for more than a decade and remains symptom-free despite inheriting an Alzheimer's-causing gene that killed his father and brother around the same age.

If blocked funding stops Heinrichs' doses, "how much time do we have?" asked his wife, Rachel Chavkin. "This trial is life."

Two drugs sold in the U.S. can modestly slow worsening of early-stage Alzheimer's by clearing the brain of one of its hallmarks, a sticky gunk called amyloid. But until now, there haven't been hints that removing amyloid far earlier – many years before the first symptoms appear – just might postpone the disease.

The research led by Washington University in St. Louis involves families that pass down rare gene mutations almost guaranteeing they'll develop symptoms at the same age their affected relatives did – information that helps scientists tell if treatments are having any effect.

The new findings center on a subset of 22 participants who received amyloid-removing drugs the longest, on average eight years. Long-term amyloid removal cut in half their risk of symptom onset, researchers reported Wednesday in the journal Lancet Neurology.

Despite the study's small size, "it's incredibly important," said Northwestern University neuroscientist David Gate, who wasn't involved with the research.

Now participants have been switched from an earlier experimental drug to Leqembi, an IV treatment approved in the U.S., to try to answer the obvious next question.

“What we want to determine over the next five years is how strong is the protection,” said Washington University’s Dr. Randall Bateman, who directs the Dominantly Inherited Alzheimer’s Network of studies involving families with these rare genes. “Will they ever get the symptoms of Alzheimer’s disease if we keep treating them?”

Here’s the worry: Bateman raised money to start that confirmatory study while seeking National Institutes of Health funding for the full project but his grant has been delayed as required reviews were canceled. It's one example of how millions of dollars in research have been stalled as NIH grapples with funding restrictions and mass firings.

At the same time researchers wonder if NIH will shift focus away from amyloid research after comments by Dr. Jay Bhattacharya, nominated as the agency's new director.

“One of the reasons I think that we have not made progress in Alzheimer’s, as much as we ought to have, is because the NIH has not supported a sufficiently wide range of hypotheses,” Bhattacharya told senators, responding to one who brought up an example of earlier science misconduct unrelated to current research.

Scientists don’t know exactly what causes Alzheimer’s, a mind-destroying disease that affects nearly 7 million Americans, mostly late in life. What’s clear is that silent changes occur in the brain at least two decades before the first symptoms -- and that sticky amyloid is a major contributor. At some point amyloid buildup appears to trigger a protein named tau to begin killing neurons, which drives cognitive decline.

Tau-fighting drugs now are being tested. Researchers also are studying other factors including inflammation, the brain’s immune cells and certain viruses.

NIH’s focus expanded as researchers found more potential culprits. In 2013, NIH’s National Institute on Aging funded 14 trials of possible Alzheimer’s drugs, over a third targeting amyloid. By last fall, there were 68 drug trials and about 18% targeted amyloid.

Northwestern’s Gate counts himself among scientists who “think amyloid isn’t everything,” but said nothing has invalidated the amyloid hypothesis. He recently used brain tissue preserved from an old amyloid study to learn how immune cells called microglia can clear those plaques and then switch to helping the brain heal, possible clues for improving today's modest therapies.

For now, amyloid clearly is implicated somehow and families with Alzheimer's-causing genes are helping answer a critical question for anyone at risk: Can blocking amyloid buildup really stave off symptoms? Without NIH funding, Bateman said, that opportunity will be lost.

“It’s absolutely insane,” said longtime study participant June Ward, who lives near Asheville, North Carolina, and plans to ask friends to complain to lawmakers.

Ward turns 64 in June and is healthy, two years older than when her mother's symptoms appeared. “It is exciting to think about the possibility that Alzheimer’s disease might not be what gets me,” she said.

In New York, Heinrichs said he has hope that his 3-year-old son won’t “experience the stress and sorrow that I lived through as a young man to watch my father fade away.”

“We need the NIH to be not politicized,” added Chavkin, his wife. “It’s just about keeping people alive or helping them live better. And in this case, it’s helping my husband survive.”

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