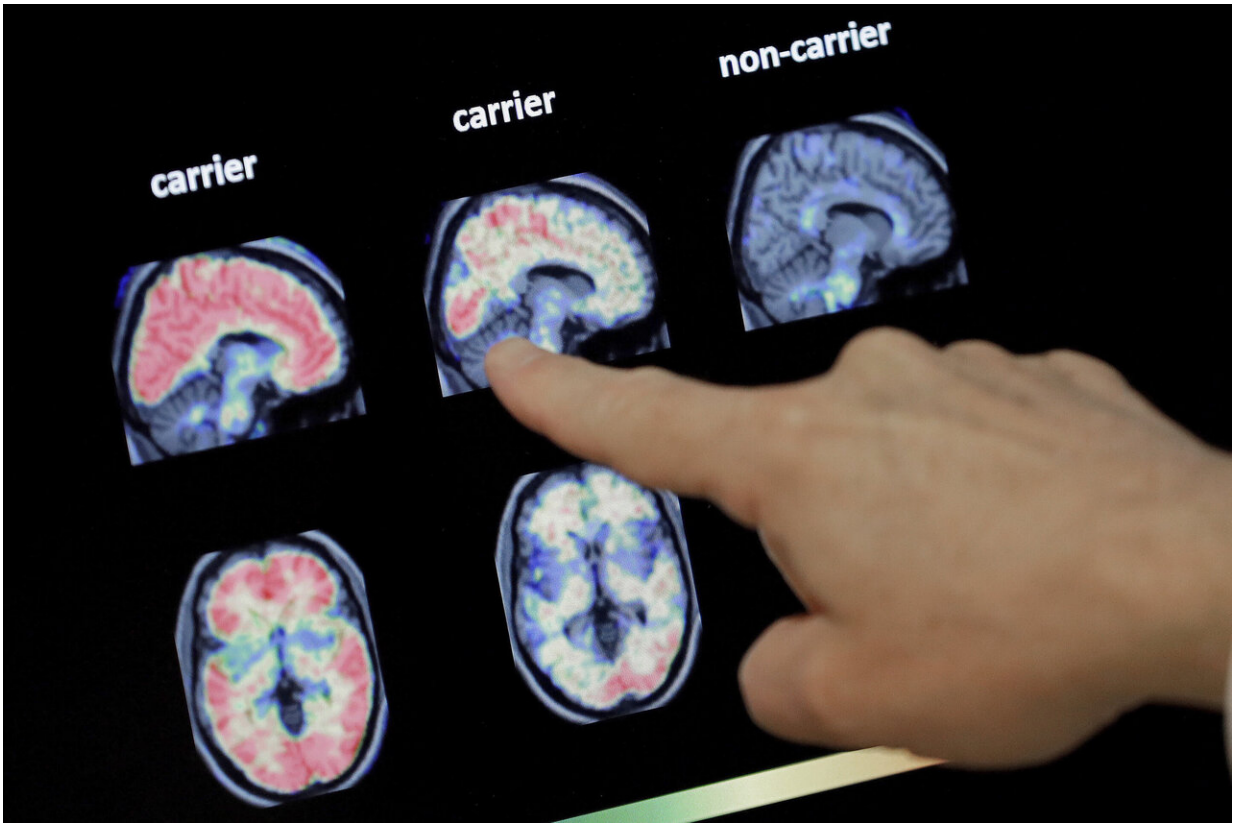
Drugs fail to slow decline in inherited Alzheimer's disease

February 10 2020, by Marilynn Marchione



In this Aug. 14, 2018 file photo, a doctor looks at a PET brain scan at the Banner Alzheimers Institute in Phoenix. Two experimental drugs failed to prevent or slow mental decline in a study of people who are virtually destined to develop Alzheimer's disease at a relatively young age because of rare gene flaws. The results announced Monday, Feb. 10, 2020, are another disappointment for the approach that scientists have focused on for many years—trying to remove a harmful protein that builds up in the brains of people with the disease. (AP Photo/Matt York, File)

Two experimental drugs failed to prevent or slow mental decline in a study of people who are virtually destined to develop Alzheimer's disease at a relatively young age because they inherited rare gene flaws.

The results announced Monday are another disappointment for the approach that scientists have focused on for years—trying to remove a harmful protein that builds up in the brains of people with Alzheimer's, the leading cause of dementia.

"We actually don't even know yet what the drugs did" in term of removing that protein because those results are still being analyzed, said study leader Dr. Randall Bateman at Washington University in St. Louis, Missouri.

But after five years on average, the main goal of the study was not met—people on either of the drugs scored about the same on thinking and memory tests as others given placebo treatments.

More than 5 million people in the United States and millions more worldwide have Alzheimer's. Current drugs only temporarily ease symptoms and do not alter the course of the disease.

The study tested solanezumab by Eli Lilly & Co., and gantenerumab by Swiss drugmaker Roche and its U.S. subsidiary, Genentech. Both drugs gave disappointing results in some earlier studies, but the doses in this one ranged up to four to five times higher and researchers had hoped that would prove more effective.

The study was funded by the U.S. National Institute on Aging, the Alzheimer's Association and some foundations.

It involved about 200 people in the United States, Europe and elsewhere with flaws in one of three genes.

"If you get one of these genetic mutations you're almost guaranteed to get Alzheimer's," typically in your 30s, 40s or 50s, said Dr. Eric McDade, another study leader at Washington University.

People like this account for only about 1% of Alzheimer's cases, but their brain changes and symptoms are similar to those who develop the disease at a later age. That gives a unique chance to test potential treatments.

"We know everyone will get sick and we know about what time that is" in their lives, Bateman said.

Most study participants already had signs of the harmful protein in their brain even if they were showing no symptoms when the study started.

The were given either a gantenerumab shot, an IV of solanezumab or fake versions of these treatments every four weeks. The drugs made no difference in a combination score of four memory and thinking tests compared to placebo treatments.

Side effects were not disclosed, but "there's no evidence of any drug -related deaths in the trial," McDade said.

Details will be given at a medical meeting in April.

Solanezumab is being tested in another study to see if it can slow memory loss in people with Alzheimer's.

Gantenerumab also is being tested in two other large experiments that are expected to give results in two to three years.

It's unclear whether the results will affect views on aducanumab, another experimental drug whose makers say it can remove the harmful protein and slow mental decline. Results on it have been mixed, and the companies have said they will seek federal approval for it soon. Cambridge, Massachusetts-based Biogen is developing it with a Japanese company, Eisai Co. Ltd.

More information: Press release