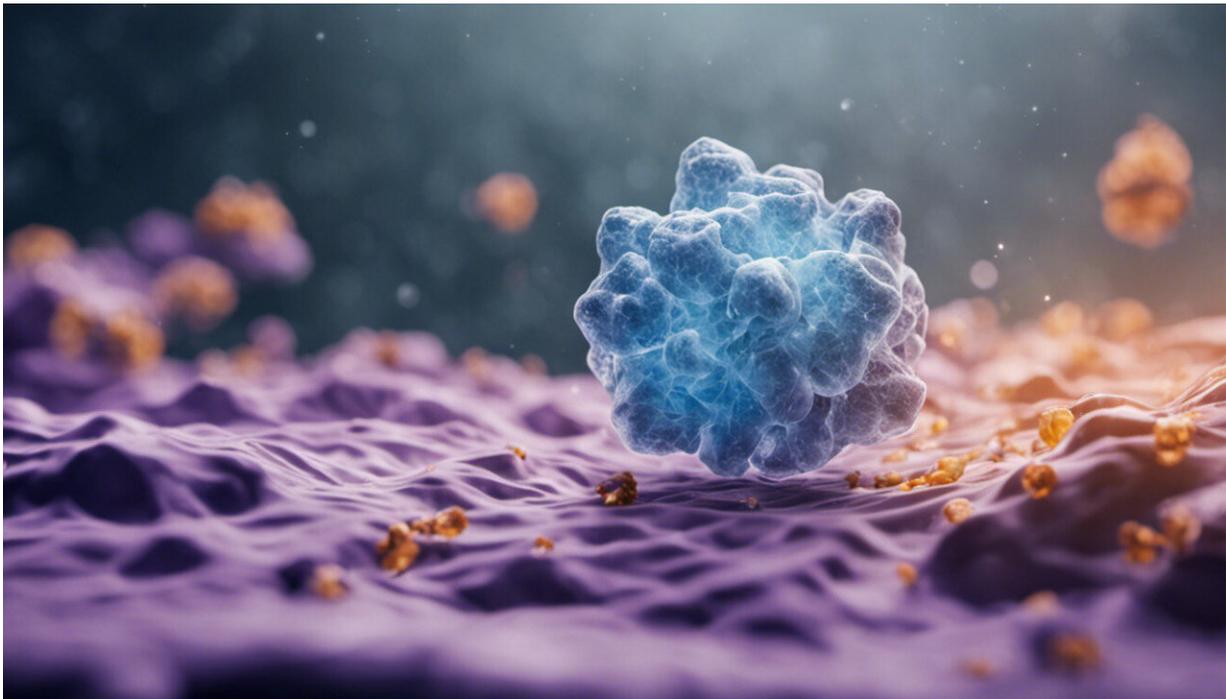


Lecanemab: Experimental drug is a ray of hope for Alzheimer's disease

December 22 2022, by Frederic Calon and Étienne Aumont



Credit: AI-generated image ([disclaimer](#))

On Nov. 30, Eisai and Biogen announced the [results of their latest phase 3 clinical trial in Alzheimer's disease](#). The verdict: an 18-month treatment with lecanemab slows functional and cognitive loss by 27 percent in people with mild cognitive impairment or mild dementia due to Alzheimer's disease. [The study results were also published in the *New*](#)

[England Journal of Medicine.](#)

The pharmaceutical company Eisai claims that unlike the drugs already approved for Alzheimer's treatment, this one is able to slow down the disease rather than just reduce its symptoms.

This good news, however, is only the first step towards finding a real cure for Alzheimer's.

As neuroscience experts specializing in the study of Alzheimer's, we are following the search for treatments for this disease very closely.

Promising but modest results

Lecanemab is an antibody that attaches to beta-amyloid proteins accumulated in the brain and allows the [immune system](#) to get rid of them. In Alzheimer's, this protein forms aggregates and is thought to contribute to the [initial progression of the disease](#).

Eisai therefore selected people with the greatest chance of benefitting from the treatment: those in the early stages of the disease or with [mild cognitive impairment](#) who have large accumulations of beta-amyloid. This is the case for about [seven out of eight people with a diagnosis of Alzheimer's and half of those living with mild cognitive impairment](#). After 18 months of treatment one-third of those treated were at normal levels of beta-amyloid.

This is [not the first time a product has gotten rid of beta-amyloid](#), but it is the first time a treatment has led to statistically clear cognitive and functional benefits. However, the effect is small: on [the 18-point clinical dementia rating scale](#), treated people lost half a point. The actual impact on the life of a treated person is therefore modest. As Alzheimer's symptoms progress slowly in the early stages of the disease, it will be

important to determine whether the effect continues after more than 18 months.

Worrisome side effects

The treatment is also not all good news. It led to a 25 percent faster shrinkage of the brain. Researchers have attributed this atrophy to the removal of beta-amyloid. However, this is not a universally accepted idea, as the brain levels of [beta-amyloid are too small to explain such shrinkage](#). The consequences of such shrinkage are unknown.

One-sixth of those treated developed cerebral edema—a build-up of water indicating inflammation. The treatment also led to twice as many brain hemorrhages— about one in six—compared to those who received the placebo. However, only one in 30 people actually experienced symptoms related to these two abnormalities. Although mild microhemorrhages are quite common in the elderly, these could reduce the brain's ability to adapt. This could therefore [increase the brain's vulnerability to diseases such as Alzheimer's](#).

Fortunately, subgroups of patients may benefit more from this treatment. Men and people aged 75 and over had a more than 40 percent reduction in cognitive decline. People without the e4 variant of the APOE gene, the [main risk factor for Alzheimer's](#), experienced fewer side effects while having a greater slowdown in the progression of the disease.

Conversely, people carrying two copies of APOE e4—from both parents—were six times more likely to develop symptoms from brain hemorrhage or edema. In addition, these people did not, on average, experience any positive effects from lecanemab. Having a single copy of APOE e4 seems to allow people to benefit from the treatment while slightly increasing the risk of side effects.

These data offer hope that health-care professionals will be able to select patients who are most likely to benefit from the treatment.

A resource-intensive treatment

All indications are that lecanemab will be resource intensive. First, before giving lecanemab to a patient, we need to ensure that his/her brain contains high levels of beta-amyloid. This will require expensive imaging equipment in addition to a team of well-trained professionals.

The antibody must also be injected once every two weeks, requiring more involvement from patients, their relatives and health professionals. To manage the risk of side effects, follow-up imaging will also be essential. Added to this is the cost of the drug itself, which has not yet been announced. According to analysts, this could be [nearly US\\$20,000 per year](#).

In short, the health-care system and the [research community](#) will have to dedicate significant resources to offer this new treatment equitably to the greatest number of people. More medical and neuropsychological follow-ups will be needed, new brain imaging infrastructures will have to be built and specialized personnel will have to be trained.

We must hope that this new treatment will be worth the effort.

Let's also hope that future clinical trial results will report greater efficacy in women and in people with APOE e4.

After all, lecanemab is just the beginning, and much more will be needed to truly cure Alzheimer's disease.

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