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Alzheimer's drug hopes dashed in trials

Chris Smyth Health Editor

The fight against dementia was dealt a blow yesterday when a drug many hoped would become the first to slow the disease fell at the final hurdle.

Solanezumab had no significant effect on the thinking abilities of people with mild Alzheimer's in the final stage of tests, leaving patient groups disappointed.

The drug was the most advanced of the clutch of medicines intended to target the plaques of amyloid protein in the brain that are the hallmarks of Alzheimer's. Scientists hope that other methods of clearing amyloid will fare better. Jeremy Hughes, chief executive of the Alzheimer's Society, said: "We had high hopes for this drug to become the first to slow down Alzheimer's disease. It's extremely disappointing to learn that it hasn't delivered a meaningful change for people living with dementia, when the need is clearly so great."

There are no drugs to treat Britain's 850,000 dementia patients, two thirds of whom have Alzheimer's disease.

The pharmaceutical company Eli Lilly announced that it would not be seeking a licence for solanezumab after the final-stage trial involving 2,100 people failed. The drug had previously failed in people with more advanced disease. The company is now considering whether it should be tested in people who do not yet have Alzheimer's.

"We are disappointed for the millions of people waiting for a potential disease-modifying treatment for Alzheimer's," John C. Lechleiter, the company's chief executive officer, said. "We will evaluate the impact of these results on the development plans for solanezumab."

David Reynolds, chief scientific officer at Alzheimer's Research UK, said: "Our 15-year wait for a new Alzheimer's drug does not end today but we shouldn't view this setback as the end of the line — the journey towards a new treatment can and will continue.

"It's very disappointing that solanezumab has not shown benefit for people with mild Alzheimer's and will no doubt trigger important debate within the research community. We only have headline information today, so we'll need to see the full data to understand why solanezumab didn't show benefits and what researchers must learn from the findings."

James Warner, consultant old age psychiatrist at Imperial College London, said: "It was a potentially ground-breaking approach to treatment. I think this does represent a failure."

Some were encouraged by signs in the data that solanezumab had some effect on patients' brains. Elizabeth Coulthard, senior lecturer in dementia neurology at the University of Bristol, said: "Again there seems to be a hint that Alzheimer's is modifiable, but solanezumab does not appear to work well enough to recommend as a treatment.

"Fortunately ... this is probably not the end for amyloid approaches because there are other types of amyloid that other drugs can target."

Peter Roberts, emeritus professor of pharmacology at the University of Bristol, warned that the problem was a fundamental one.

"There is still no convincing evidence that shows a clear relationship between amyloid deposition and deficits in cognition in humans," he said. "All we really know is that evidence of amyloid deposition begins up to maybe 20 years before the onset of Alzheimer's disease. This might be a good indicator, but does not prove causality."