# Article information:

Lecanemab in Early Alzheimer’s Disease | NEJM
<https://www.nejm.org/doi/full/10.1056/NEJMoa2212948?casa_token=ANnJZWwIy1MAAAAA%3AFKHW23QdJZrEWBxfVV4gtSduRApU9cTrEYSc0yiqavGDceuJ8eet_UJfwoJhyOuXOhLA2PUDUcpyHaXt>

# Article summary:

1. Lecanemab, a humanized monoclonal antibody that binds to soluble amyloid-beta (Aβ) protofibrils, was tested in an 18-month phase 3 trial involving persons with early Alzheimer’s disease.

2. The study found that Lecanemab reduced markers of amyloid in early Alzheimer’s disease and resulted in moderately less decline on measures of cognition and function than placebo at 18 months but was associated with adverse events.

3. Longer trials are warranted to determine the efficacy and safety of Lecanemab in early Alzheimer’s disease.

# Article rating:

Appears moderately imbalanced: The article provides some useful information, but is missing several important points or pieces of evidence that would be required to present the discussed topics in a balanced and reliable way. You are encouraged to seek a more balanced perspective on the presented issues by exploring the provided research topics and looking at different information sources.

# Article analysis:

The article titled "Lecanemab in Early Alzheimer's Disease" published in the New England Journal of Medicine reports on a phase 3 trial involving persons with early Alzheimer's disease. The study aimed to determine the safety and efficacy of lecanemab, a humanized monoclonal antibody that binds with high affinity to soluble amyloid-beta (Aβ) protofibrils.

The article provides a detailed description of the trial design, eligibility criteria, end points, and results. The primary end point was the change from baseline at 18 months in the score on the Clinical Dementia Rating–Sum of Boxes (CDR-SB), with key secondary end points being changes in amyloid burden on PET, cognitive subscale scores, and activities of daily living scale scores.

The results showed that lecanemab reduced markers of amyloid in early Alzheimer's disease and resulted in moderately less decline on measures of cognition and function than placebo at 18 months but was associated with adverse events. Longer trials are warranted to determine the efficacy and safety of lecanemab in early Alzheimer's disease.

While the article provides valuable information about the trial and its outcomes, it is important to note some potential biases and limitations. Firstly, the study was funded by Eisai and Biogen, which may have influenced its design, analysis, interpretation, and reporting. Secondly, there is no mention of any conflicts of interest among the authors or site investigators.

Moreover, while the article acknowledges that current therapeutic agents for Alzheimer's disease-related dementia temporarily improve symptoms but do not alter the underlying disease course, it does not explore other potential treatments or interventions that may complement or replace lecanemab. Additionally, there is no discussion about possible long-term effects or risks associated with lecanemab beyond 18 months.

Furthermore, while the article notes that lecanemab resulted in infusion-related reactions in 26.4% of participants and amyloid-related imaging abnormalities with edema or effusions in 12.6%, it does not provide further details about these adverse events or their severity.

In conclusion, while this article provides valuable insights into a phase 3 trial involving persons with early Alzheimer's disease treated with lecanemab, it is important to consider its potential biases and limitations when interpreting its findings. Further research is needed to determine the long-term efficacy and safety of lecanemab as well as other potential treatments for Alzheimer's disease-related dementia.

# Topics for further research:

* Long-term effects of lecanemab in Alzheimer's disease
* Alternative treatments for Alzheimer's disease-related dementia
* Conflicts of interest in Alzheimer's disease research
* Severity of infusion-related reactions in lecanemab treatment
* Risks associated with amyloid-related imaging abnormalities with edema or effusions
* Mechanism of action of lecanemab in Alzheimer's disease

# Report location:

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