# Article information:

Lecanemab in Early Alzheimer’s Disease | NEJM  
<https://www-nejm-org.sh527.top/doi/full/10.1056/NEJMoa2212948>

# Article summary:

1. Lecanemab is a humanized IgG1 monoclonal antibody that binds to Aβ soluble protofibrils and is being tested in persons with early Alzheimer’s disease.

2. An 18-month, double-blind, phase 3 trial involving 1795 participants 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.

3. Lecanemab was associated with infusion-related reactions and amyloid-related imaging abnormalities with edema or effusions.

# Article rating:

May be slightly imbalanced: The article presents the information in a generally reliable way, but there are minor points of consideration that could be explored further or claims that are not fully backed by appropriate evidence. Some perspectives may also be omitted, and you are encouraged to use the research topics section to explore the topic further.

# Article analysis:

The article provides an overview of the results from a phase 3 clinical trial of lecanemab, a humanized IgG1 monoclonal antibody that binds to Aβ soluble protofibrils, for the treatment of early Alzheimer’s disease. The article is generally well written and provides clear information about the study design, results, and conclusions. However, there are some potential biases that should be noted.

First, the authors do not provide any information about potential conflicts of interest or funding sources for the study. This could lead to bias in favor of the drug being tested as well as potential promotional content for the drug manufacturer (Eisai and Biogen). Additionally, while the authors note that there were no significant differences between lecanemab and placebo in a Bayesian analysis of 12-month change in a composite score (primary end point), they do not provide any further details about this analysis or why it did not reach significance.

Second, while the authors note that there were greater reductions in brain amyloid burden with lecanemab than with placebo at 18 months, they do not provide any information about possible long-term effects or risks associated with taking lecanemab over an extended period of time. Furthermore, they do not discuss any potential side effects or adverse events associated with taking lecanemab beyond those mentioned briefly at the end of the article (infusion-related reactions and amyloid-related imaging abnormalities).

Finally, while the authors note that longer trials are warranted to determine efficacy and safety of lecanemab in early Alzheimer’s disease, they do not discuss any other possible treatments or interventions for early Alzheimer’s disease beyond lecanemab nor do they explore any counterarguments to their findings or conclusions.

In conclusion, while this article provides useful information about a phase 3 clinical trial testing lecanemab for early Alzheimer’s disease, there are some potential biases that should be noted including lack of disclosure regarding conflicts of interest/funding sources as well as lack of discussion regarding long-term effects/risks associated with taking lecanemab as well as other treatments/interventions for early Alzheimer’s disease beyond lecanemab.

# Topics for further research:

* Conflicts of interest in clinical trials
* Long-term effects of lecanemab
* Adverse events associated with lecanemab
* Bayesian analysis of clinical trials
* Alternative treatments for early Alzheimer's disease
* Counterarguments to lecanemab efficacy

# Report location:

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