Two-Year Real-World Study of LEQEMBI® in the United States Presented at Alzheimer's Association International Conference (AAIC) 2025

July 30, 2025

TOKYO and CAMBRIDGE, Mass., July 30, 2025 (GLOBE NEWSWIRE) -- Eisai Co., Ltd. (Headquarters: Tokyo, CEO: Haruo Naito, “Eisai”) and Biogen Inc. (Nasdaq: BIIB, Corporate headquarters: Cambridge, Massachusetts, CEO: Christopher A. Viehbacher, “Biogen”) announced today that the two-year real-world study in the U.S of lecanemab (generic name, product name: LEQEBMI®), an anti-Aβ protofibril* antibody, was presented at the Alzheimer's Association International Conference (AAIC) 2025, held in Toronto, Canada and virtually. Only lecanemab fights AD in two ways – targeting both amyloid plaque and protofibrils*, which can impact tau downstream.

Lecanemab received traditional approval in the U.S. in July 2023 for the treatment of early Alzheimer's disease (AD). This retrospective study was conducted to investigate the actual state of real-world clinical treatment with lecanemab at 15 medical centers in the U.S., with a final report scheduled for late in the third quarter of Eisai’s fiscal year ending March 31, 2026. This presentation serves as an interim report as of July 1, 2025.

Patient Baseline and Actual Treatment Situation
The interim study collected information on 178 people living with early AD from nine US medical centers using a standardized case report format. The disease stage of the patients at baseline was mild cognitive impairment (MCI) due to AD in 57.6% and mild AD in 42.4%. The average age of the patients was 74.2 (±6.6) years, and the ratio of men to women was 44.6 to 55.4.

The mean duration of lecanemab treatment was 375.4 days (± 182.8 days). The mean time from diagnosis to first treatment was 224.2 days (± 295.4 days) and the mean number of lecanemab treatments was 24.8 (± 11.5). At the time of case reporting, 87.4% of patients (152 patients) were continuing treatment with lecanemab. Adverse events leading to discontinuation of treatment included ARIA-E (ARIA-edema/exudation) in two patients (1.1%), ARIA-H (ARIA-cerebral microbleeds, cerebral hemorrhage and superficial hemosiderin deposition) in two patients (1.1%), and concomitant ARIA-E and ARIA-H in one patient (0.6%). Three patients (1.7%) discontinued due to adverse events other than ARIA. In addition, 11 patients (6.3%) reported discontinuing the treatment for personal reasons or at the discretion of their doctor or the individual.

In this study, 83.6% of patients either remained at the same clinical stage or improved from mild dementia to MCI (stable: 76.9%, improvement: 6.7%). Additionally, at time of interim data cut, 86.7% of patients who had received 40 or more doses over 18 months remained stable or showed clinical improvement (stable: 66.7%, improvement: 20.0%:).

Of the 178 patients, ARIA was observed in 23 (12.9%). 14 (7.9%) were observed to have ARIA-E, of which 12 (6.7%) were asymptomatic. ARIA-H was present in 11 patients (6.2%), all of whom were asymptomatic. Infusion reactions were observed in four patients (2.2%). Additionally, no serious bleeding events or deaths were reported.

Impact of APOE4 Status
Of the 178 patients in this study, 12 were excluded with unknown status. Among the remaining 166 patients, 30 (18.1%) were APOE ε4 homozygotes, 84 (49.4%) were heterozygotes, 54 (32.5%) were non-carriers. Generally, the proportion of homozygotes among people with AD is thought to be 15% or more.

The incidence of ARIA was 20.0%, 9.8% and 14.8% in homozygous carriers, heterozygous carriers and non-carriers, respectively (45.0%, 19.0% and 13.0% respectively in the Phase 3 Clarity AD 18-month core study). The incidence rate of ARIA-E and ARIA-H were 13.3% and 10.0%, respectively (32.6% and 39.0% in the Clarity AD core study), which is within the FDA-approved label range. The majority of ARIA cases (0.13%) were asymptomatic. The incidence of adverse events leading to discontinuation was 16.7% in homozygous carriers, 2.4% in heterozygous carriers and 5.6% in non-carriers.

73.3% of homozygote patients’ clinical stage remained stable or improved (stable: 66.6%, improved: 6.7%), 88.0% of heterozygotes patients’ clinical stage remained stable or improved (stable: 83.0%, improved: 4.9%) and 85.2% of non-carrier patients’ clinical stage remained stable or improved (stable: 75.9%, improved: 9.3%).

Utilization of Blood-Based Biomarkers (BBMs)
BBMs are being developed in AD to identify brain Aβ pathology and are intended for use in prescreening (triage) and confirmatory diagnosis. Of the 178 patients in this study, 49 patients (27.5%) were diagnosed using BBMs. In some of these cases (11 patients, 6.1%), it was also used for confirmatory diagnosis. Data collected from clinical practices showed the volume of tests doubling every 4 to 8 months, with BBMs using p-tau217 growing most rapidly.

Satisfaction with Lecanemab Treatment
The results of a physician, patient and care partner lecanemab satisfaction survey was presented. The survey was based on questionnaires and interviews with nine U.S. physicians and evaluated treatment from multiple perspectives, including efficacy, safety and quality of life (QOL).

In the physicians’ evaluation, the average satisfaction level for the treatment efficacy and safety (out of 10) was 8.7. The scoring criteria included: cognition 8.1, daily function 8.1, behavioral/neuropsychiatric symptoms 7.9 and QOL 8.0. The satisfaction level of the patients as assessed by physicians was 8.8, and that of the care partners was 8.2. These results highlight the favorable evaluation of lecanemab’s efficacy and safety in real-world clinical practice and reinforce its therapeutic value.

Note:
Retrospective real-world studies can be very valuable in providing additional information to complement clinical trial data. However, there may have several limitations to keep in mind:

Potential for Biases
Data Completeness and Consistency
Data may be collected inconsistently since data collection is completed by different people at independent sites.
* *Mitigation*: Data inconsistency is pursued by providing site access to standardized electronic case-report forms.

Lack of Control Group
Interpretation of data may be limited since real-world studies do not utilize placebo-controlled arms

Confounding Variables
Confounding variables are not controllable, which may impact the relationship between the exposure and outcome.

Eisai serves as the lead for lecanemab's development and regulatory submissions globally with both companies co-commercializing and co-promoting the product and Eisai having final decision-making authority.

* Protofibrils are thought to be the most toxic Aβ species that contribute to brain damage in AD and play a major role in the cognitive decline of this progressive and devastating disease. Protofibrils can cause neuronal and synaptic damage in the brain, which can subsequently adversely affect cognitive function through multiple mechanisms.[1] The mechanism by which this occurs has been reported not only by increasing the formation of insoluble Aβ plaques, but also by directly damaging signaling between neurons and other cells. It is believed that reducing protofibrils may reduce neuronal damage and cognitive impairment, potentially preventing the progression of AD. [2]

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Notes to Editors

1. **About lecanemab (generic name, brand name: Leqembi®)**

   Lecanemab is the result of a strategic research alliance between Eisai and BioArctic. It is a humanized immunoglobulin gamma (IgG1) monoclonal antibody directed against aggregated soluble (protofibril) and insoluble forms of amyloid-beta (Aβ). Protofibrils are believed to contribute to the brain injury that occurs with AD and are considered to be the most toxic form of Aβ, having a primary role in the cognitive decline associated with this progressive, debilitating condition.[1] Protofibrils cause injury to neurons in the brain, which in turn, can negatively impact cognitive function via multiple mechanisms, not only increasing the development of insoluble Aβ plaques but also increasing direct damage to brain cell membranes and the connections that transmit signals between nerve cells or nerve cells and other cells.[2] It is believed the reduction of protofibrils may prevent the progression of AD by reducing damage to neurons in the brain and cognitive dysfunction.

   Lecanemab has been approved in 46 countries and is under regulatory review in 10 countries. In January 2025, the supplemental Biologics License Application (sBLA) for intravenous (IV) maintenance dosing of the treatment was approved in the U.S. After an 18 months initiation phase with once every two weeks of dosing, a transition to the maintenance dosing regimen of 10 mg/kg once every four weeks or continuing 10 mg/kg once every two weeks may be considered. Additionally, the U.S. Food and Drug Administration (FDA) accepted Eisai's Biologics License Application (BLA) for the LEQEMBI subcutaneous autoinjector for weekly maintenance dosing in January 2025 and set a PDUFA action date for August 31, 2025.

   Since July 2020, the Phase 3 clinical study (AHEAD 3-45) for individuals with preclinical AD, meaning they are clinically normal and have intermediate or elevated levels of amyloid in their brains, is ongoing. AHEAD 3-45 is conducted as a public-private partnership between the Alzheimer's Clinical Trial Consortium that provides the infrastructure for academic clinical trials in AD and related dementias in the U.S, funded by the National Institute on Aging, part of the National Institutes of Health, Eisai and Biogen. Since January 2022, the Tau NexGen clinical study for Dominantly Inherited AD (DIAD), that is conducted by Dominantly Inherited Alzheimer Network Trials Unit (DIAN-TU), led by Washington University School of Medicine in St. Louis, is ongoing and includes lecanemab as the backbone anti-amyloid therapy.

2. **About the Collaboration between Eisai and Biogen for AD**

   Eisai and Biogen have been collaborating on the joint development and commercialization of AD treatments since 2014. Eisai serves as the lead of lecanemab development and regulatory submissions globally, with both companies co-commercializing and co-promoting the product and Eisai having final decision-making authority.

3. **About the Collaboration between Eisai and BioArctic for AD**

   Since 2005, Eisai and BioArctic have had a long-term collaboration regarding the development and commercialization of AD treatments. Eisai obtained the global rights to study, develop, manufacture and market lecanemab for the treatment of AD pursuant to an agreement with BioArctic in December 2007. The development and commercialization agreement on the antibody lecanemab back-up was signed in May 2015.

4. **About Eisai Co., Ltd.**

   Eisai's Corporate Concept is "to give first thought to patients and people in the daily living domain, and to increase the benefits that health care provides." Under this Concept (also known as *human health care* (*hhc*) Concept), we aim to effectively achieve social good in the form of relieving anxiety over health and reducing health disparities. With a global network of R&D facilities, manufacturing sites and marketing subsidiaries, we strive to create and deliver innovative products to target diseases with high unmet medical needs, with a particular focus in our strategic areas of Neurology and Oncology.

   In addition, we demonstrate our commitment to the elimination of neglected tropical diseases (NTDs), which is a target (3.3) of the United Nations Sustainable Development Goals (SDGs), by working on various activities together with global partners.

   For more information about Eisai, please visit www.eisai.com (for global headquarters: Eisai Co., Ltd.), and connect with us on X, LinkedIn and Facebook. The website and social media channels are intended for audiences outside of the UK and Europe. For audiences based in the UK and Europe, please visit www.eisai.eu and Eisai EMEA LinkedIn.

5. **About Biogen**

   Founded in 1978, Biogen is a leading biotechnology company that pioneers innovative science to deliver new medicines to transform patient's lives and to create value for shareholders and our communities. We apply deep understanding of human biology and leverage different modalities to advance first-in-class treatments or therapies that deliver superior outcomes. Our approach is to take bold risks, balanced with return on investment to deliver long-term growth.

   The company routinely posts information that may be important to investors on its website at www.biogen.com. Follow Biogen on social media – Facebook, LinkedIn, X, YouTube.

   Biogen Safe Harbor

   This news release contains forward-looking statements, including about the potential clinical effects of lecanemab; the potential benefits, safety and efficacy of lecanemab and continued treatment with lecanemab; potential regulatory discussions, submissions and approvals and the timing thereof; the treatment of Alzheimer's disease; the anticipated benefits, risks and potential of Biogen's collaboration arrangements with Eisai; the potential of Biogen's commercial business and pipeline programs, including lecanemab; and risks and uncertainties associated with drug development and commercialization. These forward-looking statements may be accompanied by such words as "aim," "anticipate," "assume," "believe," "contemplate," "continue," "could," "estimate," "expect," "forecast," "goal," "guidance," "hope," "intend," "may," "objective," "plan," "possible," "potential," "predict," "project," "prospect," "should," "target," "will," "would," and other words and terms of similar meaning. Drug development and commercialization involve a high degree of risk, and only a small number of research and development programs result in commercialization of a product. Results in early-stage clinical trials may not be indicative of full results or results from later stage or larger scale clinical trials and do not ensure regulatory approval. You should not place undue reliance on these statements. Given their forward-looking nature, these statements involve substantial risks and uncertainties that may be based on inaccurate assumptions and could cause actual results to differ materially from those reflected in such statements.

   These forward-looking statements are based on management's current beliefs and assumptions and on information currently available to management. Given their nature, we cannot assure that any outcome expressed in these forward-looking statements will be realized in whole or in part. We caution that these statements are subject to risks and uncertainties, many of which are outside of our control and could cause future events or results to be materially different from those stated or implied in this document, including, among others, uncertainty of long-term success in developing, licensing, or acquiring other product candidates or additional indications for existing products; expectations, plans and prospects relating to product approvals, approvals of additional indications for our existing products, sales, pricing, growth, reimbursement and launch of our marketed and pipeline products; our ability to effectively implement our corporate strategy; the successful execution of our strategic and growth initiatives, including acquisitions; the risks associated with third party collaborations; the risk that positive results in a clinical trial may not be replicated in subsequent or confirmatory trials or success in early stage clinical trials may not be predictive of results in later stage or large scale clinical trials or trials in other potential indications; risks associated with clinical trials, including our ability to adequately manage clinical activities, unexpected concerns that may arise from additional data or analysis obtained during clinical trials, regulatory authorities may require additional information or further studies, or may fail to approve or may delay approval of our drug candidates; the occurrence of adverse safety events, restrictions on use with our products, or product liability claims; and

any other risks and uncertainties that are described in other reports we have filed with the U.S. Securities and Exchange Commission.

These statements speak only as of the date of this press release and are based on information and estimates available to us at this time. Should known or unknown risks or uncertainties materialize or should underlying assumptions prove inaccurate, actual results could vary materially from past results and those anticipated, estimated or projected. Investors are cautioned not to put undue reliance on forward-looking statements. A further list and description of risks, uncertainties and other matters can be found in our Annual Report on Form 10-K for the fiscal year ended December 31, 2024 and in our subsequent reports on Form 10-Q and Form 10-K, in each case including in the sections thereof captioned “Note Regarding Forward-Looking Statements” and “Item 1A. Risk Factors,” and in our subsequent reports on Form 8-K. Except as required by law, we do not undertake any obligation to publicly update any forward-looking statements whether as a result of any new information, future events, changed circumstances or otherwise.

References

1. Amin L, Harris DA. Aβ receptors specifically recognize molecular features displayed by fibril ends and neurotoxic oligomers. *Nat Commun*. 2021;12:3451. doi:10.1038/s41467-021-23507-z
2. Ono K, Tsuji M. Protofibrils of Amyloid-β are Important Targets of a Disease-Modifying Approach for Alzheimer's Disease. *Int J Mol Sci.* 2020;21(3):952. doi: 10.3390/ijms21030952. PMID: 32023927; PMCID: PMC7037706.