No.25-80

November 19, 2025
Eisai Co., Ltd.

Eisai to Present Data on Lecanemab Continued Treatment, Subcutaneous Initiation Dosing, and Real-World Experience at the 18th Clinical Trials on Alzheimer's Disease Conference

Latest findings from Eisai's robust Alzheimer's disease pipeline explore long-term treatment with lecanemab and estimated time savings over 10 years, safety and potential benefits of subcutaneous administration of lecanemab for initiation dosing

Oral and poster presentations also include data on the effects of lecanemab on soluble amyloid-β protofibrils and insights from real-world clinical practice studies, including the ALZ-NET Registry

Eisai Co., Ltd. (Headquarters: Tokyo, CEO: Haruo Naito, "Eisai") announced today that the company will present the latest findings from its robust Alzheimer's disease (AD) pipeline and research, including our anti-amyloid beta (Aβ) protofibril* antibody for the treatment of AD, lecanemab (generic name, U.S. brand name: LEQEMBI®) at the 18th Clinical Trials on Alzheimer's Disease (CTAD) Conference, being held in San Diego and virtually from December 1-4. Eisai will present eight oral presentations, 19 posters and one symposium.

Key Oral Presentations

- **Continued Treatment**: On Tuesday, Dec. 2, at 5:05 PM PT and Wednesday, Dec. 3, at 2:40 PM PT, new analyses will be presented on benefits of continued therapy and estimated time savings over 10 years of lecanemab treatment based on Phase III clinical data. (LB12, LB21)
- **Subcutaneous Initiation Dosing:** On Wednesday, Dec. 3, the late-breaking symposium, "Lecanemab Subcutaneous Formulation for Treatment Initiation in Early Alzheimer's Disease: Optimizing Patient Care with a Potential New Option (3:10 – 3:50 PM PT), will explore potential benefits of subcutaneous lecanemab initiation dosing as well as pharmacokinetic and safety findings. (LB Symposium2)
- **Real-World Experience:** An oral presentation on Thursday, Dec. 4 at 11:40 AM PT will share findings from an interim analysis of a post-marketing observational study of lecanemab in Japan. (OC30)
- **Etalanetug (E2814):** Two late-breaking oral presentations on Monday, Dec. 1, at 4:35 PM PT and Wednesday, Dec. 3, at 2:25 PM PT will highlight the potential of etalanetug in AD, including its impact on a novel plasma tau biomarker in Dominantly Inherited Alzheimer's Disease (DIAD) and use in a blood-based screening algorithm. (LB3, LB20)
- **Mechanism-Related:** An oral presentation scheduled for Tuesday, Dec. 2 at 1:40 PM PT, will review the effects of lecanemab treatment on soluble Aβ protofibrils in the Clarity AD clinical trial. (OC5)

Key Lecanemab Poster Presentation:

- **Real-World Experience:** During the poster session on Monday, Dec. 1, at 3:00 PM PT and Tuesday, Dec. 2, at 5:30 PM PT, Poster 055 presents an overview of baseline characteristics and preliminary safety findings from a study of lecanemab in AD using the ALZ-NET registry.

"As lecanemab long-term data and real-world experience grows, we are seeing the impact of continued treatment on slowing disease progression. Because lecanemab fights Alzheimer's disease by targeting both protofibrils and amyloid plaque, it continuously addresses the underlying disease process," said Lynn D. Kramer, M.D., FAAN, Chief Clinical Officer, Deep Human Biology Learning (DHBL), Eisai. "Additionally, our ongoing research evaluating new modes of treatment administration, a preclinical patient population and different therapeutic targets, demonstrate our unwavering commitment to advancing our understanding of Alzheimer's disease and bringing new options to patients and their loved ones to better meet their needs."

Additional Oral Presentations

Biomarkers and Imaging

Oral Session Date and Time	Title, Abstract Number
Dec. 3 (Weds.) 9:55 AM PT	Combining Endogenous MTBR-tau243 and Plasma pTau217 Enhances Prediction of Continuous Regional Tau PET Burden in Amyloid-Positive Early Alzheimer's Disease (OC11)

Preclinical AD

Oral Session Date and Time	Title, Abstract Number
Dec. 4 (Thurs.)11:00 AM PT	Building a Scalable and Efficient Recruitment Funnel for Preclinical Alzheimer's Disease: The AHEAD 3-45 Model (Symposium 5)

Additional Poster Presentations

Lecanemab

Poster Session Date	Title, Abstract Number
Dec. 1 (Mon.) – Dec. 2 (Tues.)	Characterizing Enrollment Patterns in a Preclinical Alzheimer's Disease Trial (P006)
Dec. 1 (Mon.) - Dec. 2 (Tues.)	Stability and Improvement in Early Alzheimer's Disease with Lecanemab: Sub-analysis from a United States Multicenter, Retrospective Real-World Study (P049)
Dec. 1 (Mon.) – Dec. 2 (Tues.)	Long-Term Benefit of Lecanemab in Patients with Low Baseline Amyloid: Estimation of Time Saved (P052)
Dec. 1 (Mon.) – Dec. 2 (Tues.)	Patient, Care Partner, and Health Care Professional Acceptability of the Autoinjector for the Subcutaneous Delivery of Lecanemab in Patients with Early Alzheimer's Disease in the US (P053)
Dec. 1 (Mon.) – Dec. 2 (Tues.)	Real-World Clinical, Safety and Patient-Reported Outcomes of Treatment with Lecanemab in a New England Alzheimer's Disease Center (P072)
Dec. 1 (Mon.) – Dec. 2 (Tues.)	Comparison of Amyloid-related Imaging Abnormalities Risk for Lecanemab versus Donanemab and the Potential Implications (P096)
Dec. 3 (Weds.)	C2N Eligibility, APOE Genotype Identification, Amyloid Confirmation Results from the AHEAD 3-45 Programme at Neuroclin Glasgow (P256)
Dec. 3 (Weds.)	A Simulation of Long-Term Lecanemab Treatment Effect on the Alzheimer's Disease Progression in ApoE4 Non-Carriers and Heterozygous Carriers (P278)
Dec. 3 (Weds.)	Neuro-Dynamic Quantitative Systems Pharmacology (QSP) Model Predicts Increasing Benefits of Continued Lecanemab Treatment with Clarity AD 48-Month Data (P279)
Dec. 4 (Thurs.)	Clinical Outcomes and Patient Experience of Subcutaneous Lecanemab Administration from an Alzheimer's Disease Treatment Center (P342)
Dec. 4 (Thurs.)	Societal Cost and Efficiency Comparison of Subcutaneous vs Intravenous Lecanemab for Early Alzheimer's Disease in the United States (P361)

Etalanetug (E2814)

Poster Session Date	Title, Abstract Number
Dec. 4 (Thurs.)	Antibody Effector Function of the Murine Analogue of Etalanetug Drives Efficacy in an Animal Model of Pathological Tau Seeding and Spreading (P390)

Biomarkers and Imaging

Poster Session Date	Title, Abstract Number
Dec. 1 (Mon.) – Dec. 2 (Tues.)	Analytical and Clinical Validation of the APOE Genotyping PCR Test in Patients from the Phase III Clarity AD Trial (P092) Collaboration with Sysmex
Dec. 3 (Weds.)	Cross-Platform CSF Proteomics Reveals Reproducible Biomarkers and Novel Mechanisms Beyond Amyloid and Tau in AD (P276)

Risk Factors, Diagnosis and Treatment in Early AD

Poster Session Date	Title, Abstract Number
Dec. 1 (Mon.) – Dec. 2 (Tues.)	A Delphi Panel Exploring the Burden of ARIA Management from the Provider's Perspective (P103)
Dec. 3 (Weds.)	Functional Decline Associated with All-Cause Mortality in Patients with Mild Cognitive Impairment and Alzheimer's Dementia in Real-World Clinical Practice (P304)
Dec. 4 (Thurs.)	Caregiving and Economic Burden of Alzheimer's Disease in Japan (P377)
Dec. 4 (Thurs.)	Physician Perception of Treatment Landscape and Readiness in Care Delivery with Anti-Amyloid Therapy for Alzheimer's Disease in China (P379)

Poster viewing time begins at 3:00 PM PT on Monday, Dec. 1, 5:30 PM PT on Tuesday, Dec. 2 and is set from 7:15 AM – 5:30 PM PT on Wednesday, Dec. 3 and 7:15 AM– 5:00 PM PT on Thursday, Dec. 4.

This release discusses investigational uses of agents in development and is not intended to convey conclusions about efficacy or safety. There is no guarantee that such investigational agents will successfully complete clinical development or gain health authority approval.

* 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 slowing the progression of AD.[2]

[Notes to editors]

1. About lecanemab (LEQEMBI®)

Lecanemab is the result of a strategic research alliance between Eisai and BioArctic. It is a humanized immunoglobulin gamma 1 (IgG1) monoclonal antibody directed against aggregated soluble (protofibril) and insoluble forms of amyloid-beta (Aβ). Lecanemab has been approved in 51 countries and regions and is under regulatory review in 9 countries. Following the initial phase with treatment every two weeks for 18 months, intravenous (IV) maintenance dosing with treatment every four weeks was approved in the U.S., China, and other countries, and applications have been filed in 4 countries and regions. The U.S. FDA approved Eisai's Biologics License Application (BLA) for subcutaneous maintenance dosing with LEQEMBI IQLIK in August 2025. In September 2025, the rolling sBLA application to the U.S. FDA for the subcutaneous initiation dosing with LEQEMBI IQLIK was also initiated.

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.

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.