Press release

Leqembi® (lecanemab) launched in the EU today

Stockholm, August 25, 2025 – BioArctic AB's (publ) (Nasdaq Stockholm: BIOA B) partner Eisai announced today that the launch of Leqembi in the EU started in Austria on August 25, 2025, and will be launched in Germany on September 1, 2025. Leqembi received the European Commission (EC) approval in April 2025 as the first therapy that targets an underlying cause of Alzheimer's disease (AD). It is indicated for the treatment of adult patients with a clinical diagnosis of mild cognitive impairment (MCI) and mild dementia due to AD (early AD) who are apolipoprotein E ε4 (ApoE ε4[1]) non-carriers or heterozygotes with confirmed amyloid pathology.[i] Germany and Austria will mark the first launches in the EU.

Following the EC approval, Eisai has been collaborating with the regional and local healthcare authorities to implement the mandatory authorisation requirements ahead of launch. The required controlled access program[2] is now in place in Austria and Germany, enabling the launch in these first two EU countries.

Alzheimer's disease is a progressive, relentless disease with Aβ and tau as hallmarks. It progresses in stages that increase in severity over time, and each stage of the disease presents different challenges for those living with the disease and their care partners. There is a significant unmet need for new treatment options that slow the progression of Alzheimer's disease from its early stage and reduce the overall burden on people affected by Alzheimer's disease and society. Only Leqembi fights Alzheimer's disease in two ways - targeting both amyloid plaque and protofibrils[3], which can impact tau accumulation downstream.

In the Clarity AD clinical trial, the primary endpoint was the global cognitive and functional scale, Clinical Dementia Rating – Sum of Boxes (CDR-SB).[i] Treatment with lecanemab (n=757), in the EU

[1] Apolipoprotein E is a protein involved in the metabolism of lipid in humans. It is implicated in AD. People with only one (heterozygous) or no copy (non-carriers) of the ApoE ε4 gene are less likely to experience ARIA than people with two ApoE ε4 copies (homozygous).[ii] ARIA is a recognized important side effect with lecanemab that involves swelling and potential bleeding in the brain.[i, ii]

[2] Controlled access program is a system that restricts the use and distribution of certain medicines. It is designed to promote the appropriate use of medicines while ensuring patient safety. In line with the EC approval requirements, initiation of lecanemab treatment should be through a central registration system implemented as part of CAP.

[3] 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.[v] 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. It is believed the reduction of protofibrils may prevent the progression of AD by reducing damage to neurons in the brain and cognitive dysfunction. [v, vi]

indicated population (ApoE ε4 non-carriers or heterozygotes, measured by controlled-based multiple imputation[4]), reduced clinical decline on CDR-SB by 31% at 18 months compared to placebo (n=764).[i]

In the EU indicated population (ApoE ε4 non-carriers or heterozygotes) (n=757), the most common adverse reactions were infusion-related reaction (26%), ARIA-H (13%), headache (11%) and ARIA-E (9%). Symptomatic ARIA-E occurred in 2% of participants. Symptomatic ARIA-H occurred in 0.8% of patients.[i]

Leqembi is the result of a long-standing collaboration between BioArctic and Eisai, and the antibody was originally developed by BioArctic based on the work of Professor Lars Lannfelt and his discovery of the Arctic mutation in Alzheimer's disease. Eisai is responsible for the clinical development, applications for market approval and commercialization of Leqembi for Alzheimer's disease. BioArctic has the right to commercialize Leqembi in the Nordic region together with Eisai and the two companies are preparing for a joint commercialization in the region.

The information was released for public disclosure, through the agency of the contact person below, on August 25, 2025, at 09:00 a.m. CET.

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About lecanemab (Leqembi®)
Lecanemab is the result of a strategic research alliance between BioArctic and Eisai. It is a humanized immunoglobulin gamma 1 (IgG1) monoclonal antibody directed against aggregated soluble (protofibril) and insoluble forms of amyloid-beta (Aβ).[i,ii]

Lecanemab is approved in the U.S., Japan, EU, China, Great Britain, and several other markets for the treatment of mild cognitive impairment (MCI) due to Alzheimer's disease (AD) and mild AD dementia. Lecanemab's approvals in these countries, as well as the EC's market authorization, were primarily based on Phase 3 data from Eisai's global Clarity AD clinical trial, in which it met its primary endpoint and all key secondary endpoints with statistically significant results.[i,ii] Clarity AD was a Phase 3 global, placebo-controlled, double-blind, parallel-group, randomized study in 1,795 patients with early AD (MCI or mild dementia due to AD, with confirmed presence of amyloid pathology), of which 1,521 were in the recommended indicated population in the label in the European Union (ApoE ε4 heterozygotes or non-carriers).[ii] The treatment group was administered lecanemab 10 mg/kg bi-weekly, with participants allocated in a 1:1 ratio to receive either placebo or lecanemab for 18 months.[ii]

[4] As requested by the regulatory authority, efficacy analyses were conducted for ApoE ε4 non-carriers or heterozygotes participants using control-based multiple imputation method, in which all missing values were imputed with copy-increments (change between visits) using the actual value in placebo group.[vii] This methodology differs from that used in the Clarity AD primary analysis which used mixed-model repeat measures (MMRM) with missing at random assumption.

The primary endpoint was the global cognitive and functional scale, CDR-SB [ii] In the Clarity AD clinical trial, treatment with lecanemab (n=757), in the EU indicated population (ApoE ε4 non-carriers or heterozygotes, measured by control-based multiple imputation), reduced clinical decline on CDR-SB by 31% at 18 months compared to placebo (n=764).[i] The mean CDR-SB score at baseline was approximately 3.2 in both groups.[i] The adjusted least-squares mean change from baseline at 18 months was 1.217 with lecanemab and 1.752 with placebo (difference, −0.535; 95% confidence interval [CI], −0.778 to −0.293).[1] CDR-SB is a global cognitive and functional scale that measures six domains of functioning, including memory, orientation, judgement and problem solving, community affairs, home and hobbies, and personal care.[iii]

In addition, the secondary endpoint from the AD Cooperative Study-Activities of Daily Living Scale for Mild Cognitive Impairment (ADCS MCI-ADL), which measures information provided by people caring for patients with AD, noted 33% less decline compared to placebo at 18 months.[i] The adjusted mean change from baseline at 18 months in the ADCS MCI-ADL score was −3.873 in the lecanemab group and −5.809 in the placebo group (difference, 1.936; 95% CI, 1.029 to 2.844).[i] The ADCS MCI-ADL assesses the ability of patients to function independently, including being able to dress, feed themselves and participate in community activities.[iv] Amyloid Positron Emission Tomography (PET) using Centiloids and ADAS-Cog14 also showed highly statistically significant results compared with placebo (P<0.001).[i]

In the EU indicated population (ApoE ε4 heterozygotes or non-carriers), the most common adverse reactions were infusion-related reaction (26%), ARIA-H (13%), headache (11%) and ARIA-E (9%). Symptomatic ARIA-E occurred in 2% of participants. Symptomatic ARIA-H occurred in 0.8% of patients.[i]

Lecanemab has been approved in 48 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., and application have been filed in 9 countries and regions. 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, Eisai's Phase 3 clinical study (AHEAD 3-45) with lecanemab in individuals with preclinical AD, meaning they are clinically normal and have intermediate or elevated levels of amyloid in their brains, is ongoing. The study was fully recruited in October 2024. AHEAD 3-45 is a four-year study conducted as a public-private partnership between Eisai, Biogen and 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. 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.

About the collaboration between BioArctic and Eisai
Since 2005, BioArctic has a long-term collaboration with Eisai regarding the development and commercialization of drugs for the treatment of Alzheimer's disease. The most important agreements are the Development and Commercialization Agreement for the lecanemab antibody, which was signed 2007, and the Development and Commercialization agreement for the antibody Leqembi back-up for Alzheimer's disease, which was signed 2015. In 2014, Eisai and Biogen entered into a joint development and commercialization agreement for lecanemab. Eisai is responsible for the clinical development, application for market approval and commercialization of the products for Alzheimer's disease. BioArctic has the right to commercialize lecanemab in the Nordic region and is currently preparing for commercialization in the Nordics together with Eisai.

BioArctic has no development costs for lecanemab in Alzheimer's disease and is entitled to payments in connection with regulatory approvals, and sales milestones as well as royalties on global sales.

About BioArctic AB

BioArctic AB (publ) is a Swedish research-based biopharma company focusing on innovative treatments that can delay or stop the progression of neurodegenerative diseases. The company invented Leqembi® (lecanemab) – the world's first drug proven to slow the progression of the disease and reduce cognitive impairment in early Alzheimer's disease. Leqembi has been developed together with BioArctic's partner Eisai, who are responsible for regulatory interactions and commercialization globally. In addition to Leqembi, BioArctic has a broad research portfolio with antibodies against Parkinson's disease and ALS as well as additional projects against Alzheimer's disease. Several of the projects utilize the company's proprietary BrainTransporter™ technology, which has the potential to actively transport antibodies across the blood-brain barrier to enhance the efficacy of the treatment. BioArctic's B share (BIOA B) is listed on Nasdaq Stockholm Large Cap. For further information, please visit www.bioarctic.com.

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[i] European Medicines Agency Summary of Product Characteristics (SmPC）

[ii] van Dyck, C.H., et al. Lecanemab in Early Alzheimer's Disease. New England Journal of Medicine. 2023;388:9-21. https://www.nejm.org/doi/full/10.1056/NEJMoa2212948.

[iii] Morris, J.C. The Clinical Dementia Rating (CDR): current version and scoring rules. Neurology. 1993;43:2412-2414.

[iv] Pedrosa, H., et al. Functional evaluation distinguishes MCI patients from healthy elderly people–the ADCS/MCI/ADL scale. The Journal of Nutrition, Health and Aging. 2010;14(8):703–9.

[v] Amin, L., Harris, D.A. Aβ receptors specifically recognize molecular features displayed by fibril ends and neurotoxic oligomers. Nature Communications. 2021;12:3451. doi:10.1038/s41467-021-23507-z.

[vi] 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.

[vii] Froelich L., et al. Lecanemab for treatment of individuals with early Alzheimer's disease (AD) who are apolipoprotein E E4 (ApoE e4) non-carriers of heterozygotes. Poster presented at German Association for Psychiatry, Psychotherapy and Psychosomatics (DGPPN) conference, November 2024