Press release

First patient treated with Leqembi® (lecanemab) in the Nordics

Stockholm, October 14, 2025 – BioArctic AB (publ) (Nasdaq Stockholm: BIOA B) announced today that Leqembi has now been made available at a private clinic in Finland and that the first patient has started treatment. BioArctic copromotes Leqembi with its partner Eisai in the Nordic countries, and this marks an important strategic step for BioArctic on the company's journey towards building Sweden's next major pharmaceutical company.

"I am very happy that we are now starting to help people diagnosed with early Alzheimer's disease also in the Nordic countries. There is a significant unmet need for new treatment options that slow the progression of Alzheimer's disease from its early stage, and to reduce the overall burden on people affected by Alzheimer's disease, as well as care partners and society," said Gunilla Osswald, CEO at BioArctic. "It is great to see that this innovation, based on Professor Lars Lannfelts research at Uppsala University here in Sweden, is finally, after more than 20 years of research and development, beginning to help patients close to where it all started."

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.[2]

Following the EC approval, BioArctic and Eisai have been collaborating with the Nordic healthcare authorities to implement the mandatory authorisation requirements. The required controlled access program[3] is now in place in Finland, enabling private clinics such as Terveystalo Ruoholahti, where the first patient was treated, to help patients with early Alzheimer´s disease. In the meantime, Leqembi is under assessment for inclusion in the publicly funded health care system.

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. The data show that amyloid-beta protofibrils

[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.

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

[3] 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.

and tau tangles play roles in the neurodegeneration process,[4,5,6] and Leqembi is the only approved treatment that fights Alzheimer's disease in two ways – targeting both protofibrils[i] and amyloid plaque, which can impact tau downstream.

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 October 14, 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).

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. 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).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.[7]

The primary endpoint was the global cognitive and functional scale, CDR-SB 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

[4] 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.
[5] 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.
[6] Morris JC. Neurology. 1993;43(11):2412-4.
[7] 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

compared to placebo (n=764). The mean CDR-SB score at baseline was approximately 3.2 in both groups. 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). 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.[2]

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. 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). The ADCS MCI-ADL assesses the ability of patients to function independently, including being able to dress, feed themselves and participate in community activities. Amyloid Positron Emission Tomography (PET) using Centiloids and ADAS-Cog14 also showed highly statistically significant results compared with placebo ($P<0.001$).[2,7]

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.[2]

Lecanemab is approved in 50 countries including the U.S., Japan, China, and the European Union for the treatment of Alzheimer's disease (AD) in patients with Mild Cognitive Impairment (MCI) or mild dementia stage of disease (collectively referred to as early AD) and is under regulatory review in 10 countries. Following the initial phase with treatment every two weeks for 18 months, intravenous (IV) maintenance dosing with treatment every four weeks is approved in China, the U.S. and others, and applications have been filed in 9 countries and regions. Leqembi Iqlik™ is approved for subcutaneous injection for maintenance dosing for the treatment of early Alzheimer's disease in the US.

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] 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. It is believed the reduction of protofibrils may prevent the progression of AD by reducing damage to neurons in the brain and cognitive dysfunction.[2]