Eisai Presents New LEQEMBI® (lecanemab-irmb) Investigational Subcutaneous Formulation Interim Study Results and Clinical Improvement Data in Earlier Stages of Early Alzheimer's Disease From Additional Analyses of Clarity AD at The Clinical Trials On Alzhe

October 25, 2023

Investigational Subcutaneous Formulation Clears 14% More Plaque Than IV, Pharmacokinetics (AUC) 11% Higher, And Similar ARIA Rates To IV

76% Of Patients Showed No Decline And 60% Showed Clinical Improvement At 18 Months in Low-Tau Subpopulation in Additional Analysis of Clarity AD

Dual-Acting LEQEMBI Supports Brain Neuron Function by Removing Highly Toxic Proteins (Protofibrils) That Can Continue to Cause Neuronal Injury and Death Even After Plaque Removal, Offering Early AD Patients the Opportunity for Continued Benefit

TOKYO and CAMBRIDGE, England, Oct. 25, 2023 (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 Eisai presented new data for LEQEMBI® (lecanemab-irmb) 100 mg/mL injection for intravenous (IV) use, in the Late Breaking Symposium 4 "Lecanemab for Early Alzheimer's Disease: Long-Term Outcomes, Predictive Biomarkers and Novel Subcutaneous Administration" at the 16 th annual Clinical Trials on Alzheimer's Disease (CTAD) conference held in Boston, Massachusetts, United States and virtually October 24-27, 2023.

1. Subcutaneous Formulation Interim Data; Safety And Effects On Brain Amyloid

1) Weekly subcutaneous (SC) administration showed 14% greater amyloid plaque removal than biweekly IV administration as suggested in a preliminary analysis using amyloid PET at 6 months of treatment.

- The SC substudy, evaluating the SC formulation in an open-label extension (OLE) of the Clarity AD study*, included 72 patients who received LEQEMBI for the first time as the SC formulation, and 322 patients who received intravenous (IV) LEQEMBI in the Clarity AD core study followed by SC administration in this substudy. Reduction from baseline of amyloid in the brain by amyloid PET at 6 months in the newly treated SC patients by centiloid reduction was -40.3 ± 2.27 in SC administration compared to -35.4 ± 1.14 in IV administration.[1]

2) SC Pharmacokinetics (AUC) Higher Than IV By 11%

- Weekly SC administration AUC are 11% higher than the biweekly IV formulation. 90% CI for drug exposure for SC vs. IV is within the bioequivalence limits of 80 to 125%. These data could allow Eisai to select a dose that achieves AUC that are comparable to the IV dose.[1]

3) Lower Systemic Injection Reaction Rates With SC As Compared To IV

- Systemic injection/infusion reactions are uncommon and mild with SC administration, and in particular have not been observed in patients who received LEQEMBI for the first time as the SC formulation. There was a low rate of local injection site reactions (8.1%) in SC treated patients overall. Most were mild and moderate in severity consisting of redness, irritation, or swelling. No skin rash or other hypersensitivity reactions were reported.[1]

4) ARIA Rates Of IV Formulation In Clarity AD Core Study Consistent With Rates In First-Time LEQEMBI Patients Entering The SC Substudy In Clarity AD OLE

- The incidence of ARIA-E with SC was similar to the IV. The incidences of ARIA-E, ARIA-H (cerebral microhemorrhage due to ARIA, cerebral hemorrhage, and brain surface hemosiderin deposition) and ARIA-H alone (ARIA-H without ARIA-E) with IV in the Clarity AD core study (n=898) were 12.6%, 17.3% and 8.9%, respectively. In newly treated patients in the SC substudy of the Clarity AD OLE (n=72), the incidences of ARIA-E, ARIA-H and ARIA-H alone were 16.7%, 22.2% and 8.3%, respectively. However, due to the sample size of newly treated patients in the SC substudy, no exact comparison can be made.[1]
- Based on Phase II and III clinical studies, Cmax (maximum exposure) was the strongest predictor of ARIA-E incidence following IV administration. In the SC substudy, the steady-state exposure (AUCss) appears to be a better predictor of ARIA-E rates in the SC due to a relatively stable exposure profile. [1]

Eisai aims to submit a LEQEMBI SC formulation Biologics License Application (BLA) with the U.S. Food and Drug Administration by March 31, 2024.

2. Latest Data From Tau Pet Longitudinal Substudy, Including A Post-Hoc Analysis Of The Low And Intermediate + High-Tau Subpopulations In The Clarity AD 18 Month Core Study

1) 76% of patients showed no decline and 60% showed clinical improvement at 18 months in low-tau / earlier stage early AD population.

- The Clarity AD study included an optional Tau PET substudy and used the tau PET probe MK6240** to identify patients

with a low accumulation of tau in the brain, which represents the earlier stage of early AD.

- The low-tau subpopulation, which is in the earlier stages of early AD, is thought to show slow disease progression. In the low-tau subpopulation, 76% of the LEQEMBI group showed no deterioration and 60% showed clinical improvement after 18 months of treatment in the primary endpoint, Clinical Dementia Rating - Sum of Boxes (CDR-SB), compared with 55% and 28% of the placebo group, respectively.[1]
- Importantly, in this low-tau subgroup, LEQEMBI treatment also showed consistent clinical response across multiple endpoints.*** In this population, LEQEMBI treatment favored cognition and function in the earlier stage of early AD.[1]
- The efficacy results of the Tau PET substudy in the Clarity AD study, which observed tau pathology in the brain by tau PET, were consistent with overall results of the Clarity AD study.[1]

2) Tau PET Substudy Showed LEQEMBI Slows Development Of Tau Tangles In Early AD; Tau Spread In The Brain Is A Hallmark Of Disease Progression.

- In the Clarity AD Tau PET substudy, LEQEMBI treatment slowed the buildup of tau proteins in the temporal lobe (early Braak region), where tau accumulation was observed in the earlier stage of early AD. In the Tau PET substudy, LEQEMBI suppressed the accumulation of tau in the medial temporal brain region in low-tau subpopulations, and in a broader range of brain regions in the intermediate and higher accumulation groups**. This suggests that LEQEMBI treatment may have different effects on brain regions indexed by tau depending on the stage of the disease.[1] The spread of tau in the brain is a hallmark of AD progression.[2]

3. Efficacy Results From LEQEMBI Clarity AD Open-Label Extension Study

1) LEQEMBI Patients Continued to Show Benefit at 24 Months of Treatment

- In the 18-month core study of Clarity AD, there was a statistically significant difference in global cognition and function as measured by CDR-SB between the LEQEMBI and placebo groups. The separation in CDR-SB between the group that continued to receive LEQEMBI (early start group) and the group who switched from placebo to LEQEMBI (delayed start group) was maintained during the 6-month OLE following the core study. This indicates that similar disease trajectory for both early and delayed start groups occurred with LEQEMBI administration.[1]
- The blood biomarker results (plasma Aβ42/40 ratio, ptau181, GFAP and NfL) showed improvement even after delayed initiation of treatment with LEQEMBI.[1] These results suggest that LEQEMBI treatment may affect clinical outcomes through improvement of AD pathology.[1]

4. The Mechanism-Based Rationale Of LEQEMBI Treatment In Early AD

1) Dual-Acting LEQEMBI[3] Continues To Support Brain Neuron Function[3,4,5] By Removing Highly Toxic Proteins (Protofibrils**)[2,4] That Can Cause Neuronal Injury And Death Even After Plaque Removal,[5-8] Offering Patients The Opportunity For Continued Benefit.**

- LEQEMBI has a unique dual action[1,3] that binds more selectively to highly toxic protein (protofibrils****) in addition to rapidly clearing plaque,[7] and continues to support neuronal function[3,4] by removing protofibrils**** that can cause neuronal injury and death after plaque has been cleared.[5-8]

Eisai is hosting a live webcast of the scientific session featuring the LEQEMBI presentations, which can be viewed on the investors section of the Eisai Co., Ltd. website. The content will be available on demand afterward.

Eisai serves as the lead of LEQEMBI development and regulatory submissions globally with both Eisai and Biogen co-commercializing and co-promoting the product and Eisai having final decision-making authority.

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.

*Phase III Clarity AD study is a placebo-controlled, double-blind, parallel-group, randomized study to evaluate the efficacy and safety of LEQEMBI 10 mg/kg bi-weekly for 18 months in 1,795 people living with early AD (core study). An OLE is being conducted after the core study. SC dosing is currently being evaluated in the Clarity AD OLE.

**Using the MK6240 tau PET probe, tau accumulation in the brain was defined as low tau accumulation group (MK6240 cutoff value <1.06, 141 subjects), intermediate accumulation group (MK6240 cutoff value between 1.06 and 2.91, 191 subjects), and high accumulation group (MK6240 cutoff value >2.91, 10 subjects).

***Multiple endpoints: CDR-SB, a numeric scale used to quantify the severity of symptoms of dementia; ADAS-Cog14, common cognitive assessment instrument used in AD clinical trials all over the world; and ADCS MCI-ADL, a scale to assess the parties' activities of daily living.

****Protofibrils:

- One of the AD pathological features is the accumulation of clusters (plaques) of amyloid beta (Aβ) in the brain. The formation of these plaques is the result of a continuous process by which individual Aβ proteins join together, latching onto each other, one at a time, like adding links to a chain.[9] In the early part of this process these small chains of Aβ are soluble and are toxic to the nerves within the brain.[10,11]
- The most toxic of the soluble chains is called a protofibril. 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.[4,11]

- 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.[12]

INDICATION

LEQEMBI is indicated for the treatment of Alzheimer's disease. Treatment with LEQEMBI should be initiated in patients with mild cognitive impairment or mild dementia stage of disease, the population in which treatment was initiated in clinical trials.

WARNING: AMYLOID RELATED IMAGING ABNORMALITIES (ARIA)

- **Monoclonal antibodies directed against aggregated forms of amyloid beta, including LEQEMBI, can cause amyloid related imaging abnormalities (ARIA), characterized as ARIA with edema (ARIA-E) and ARIA with hemosiderin deposition (ARIA-H). Incidence and timing of ARIA vary among treatments. ARIA usually occurs early in treatment and is usually asymptomatic, although serious and life-threatening events rarely can occur. Serious intracerebral hemorrhages >1 cm, some of which have been fatal, have been observed in patients treated with this class of medications.**
  - **Apolipoprotein E ε4 (ApoE ε4) Homozygotes: Patients who are ApoE ε4 homozygotes (approximately 15% of Alzheimer's disease patients) treated with this class of medications, including LEQEMBI, have a higher incidence of ARIA, including symptomatic, serious, and severe radiographic ARIA, compared to heterozygotes and noncarriers. Testing for ApoE ε4 status should be performed prior to initiation of treatment to inform the risk of developing ARIA. Prior to testing, prescribers should discuss with patients the risk of ARIA across genotypes and the implications of genetic testing results. Prescribers should inform patients that if genotype testing is not performed, they can still be treated with LEQEMBI; however, it cannot be determined if they are ApoE ε4 homozygotes and at higher risk for ARIA.**
- **Consider the benefit of LEQEMBI for the treatment of Alzheimer's disease and potential risk of serious adverse events associated with ARIA when deciding to initiate treatment with LEQEMBI**

CONTRAINDICATION

LEQEMBI is contraindicated in patients with serious hypersensitivity to lecanemab-irmb or to any of the excipients of LEQEMBI. Reactions have included angioedema and anaphylaxis.

WARNINGS AND PRECAUTIONS

AMYLOID RELATED IMAGING ABNORMALITIES

- LEQEMBI can cause ARIA-E and ARIA-H. ARIA-E can be observed on MRI as brain edema or sulcal effusions, and ARIA-H as microhemorrhage and superficial siderosis. ARIA can occur spontaneously in patients with Alzheimer's disease. ARIA-H associated with monoclonal antibodies directed against aggregated forms of beta amyloid generally occurs in association with an occurrence of ARIA-E. ARIA-H and ARIA-E can occur together. ARIA usually occurs early in treatment and is usually asymptomatic, although serious and life-threatening events, including seizure and status epilepticus, rarely can occur. Reported symptoms associated with ARIA may include headache, confusion, visual changes, dizziness, nausea, and gait difficulty. Focal neurologic deficits may also occur. Symptoms associated with ARIA usually resolve over time.

ARIA Monitoring and Dose Management Guidelines

- Obtain recent baseline brain magnetic resonance imaging (MRI) prior to initiating treatment with LEQEMBI. Obtain an MRI prior to the 5th, 7th and 14th infusions.
- Recommendations for dosing in patients with ARIA-E and ARIA-H depend on clinical symptoms and radiographic severity. Depending on ARIA severity, use clinical judgment in considering whether to continue dosing, temporarily discontinue treatment, or permanently discontinue LEQEMBI.
- Enhanced clinical vigilance for ARIA is recommended during the first 14 weeks of treatment with LEQEMBI. If a patient experiences symptoms suggestive of ARIA, clinical evaluation should be performed, including MRI if indicated. If ARIA is observed on MRI, careful clinical evaluation should be performed prior to continuing treatment.
- There is no experience in patients who continued dosing through symptomatic ARIA-E or through asymptomatic, but radiographically severe, ARIA-E. There is limited experience in patients who continued dosing through asymptomatic but radiographically mild to moderate ARIA-E. There are limited data in dosing patients who experienced recurrent ARIA-E.

Incidence of ARIA

- In Study 2, symptomatic ARIA occurred in 3% (29/898) of LEQEMBI-treated patients. Serious symptoms associated with ARIA were reported in 0.7% (6/898) of patients treated with LEQEMBI. Clinical symptoms associated with ARIA resolved in 79% (23/29) of patients during the period of observation.
- Including asymptomatic radiographic events, ARIA was observed in LEQEMBI: 21% (191/898); placebo: 9% (84/897). ARIA-E was observed in LEQEMBI: 13% (113/898); placebo: 2% (15/897). ARIA-H was observed in LEQEMBI: 17% (152/898); placebo: 9% (80/897). There was no increase in isolated ARIA-H for LEQEMBI vs placebo.

ApoE ε4 Carrier Status and Risk of ARIA

- In Study 2, 16% (141/898) of patients in the LEQEMBI arm were ApoE ε4 homozygotes, 53% (479/898) were heterozygotes, and 31% (278/898) were noncarriers.
- The incidence of ARIA was higher in ApoE ε4 homozygotes (LEQEMBI: 45%; placebo: 22%) than in heterozygotes (LEQEMBI: 19%; placebo: 9%) and noncarriers (LEQEMBI: 13%; placebo: 4%). Among patients treated with LEQEMBI,

symptomatic ARIA-E occurred in 9% of ApoE ε4 homozygotes compared with 2% of heterozygotes and 1% noncarriers. Serious events of ARIA occurred in 3% of ApoE ε4 homozygotes, and approximately 1% of heterozygotes and noncarriers.
- The recommendations on management of ARIA do not differ between ApoE ε4 carriers and noncarriers.

Radiographic Findings

- The majority of ARIA-E radiographic events occurred early in treatment (within the first 7 doses), although ARIA can occur at any time and patients can have more than 1 episode. The maximum radiographic severity of ARIA-E in patients treated with LEQEMBI was mild in 4% (37/898), moderate in 7% (66/898), and severe in 1% (9/898). Resolution on MRI occurred in 52% of ARIA-E patients by 12 weeks, 81% by 17 weeks, and 100% overall after detection. The maximum radiographic severity of ARIA-H microhemorrhage in LEQEMBI-treated patients was mild in 9% (79/898), moderate in 2% (19/898), and severe in 3% (28/898) of patients; superficial siderosis was mild in 4% (38/898), moderate in 1% (8/898) , and severe in 0.4% (4/898). Among LEQEMBI-treated patients, the rate of severe radiographic ARIA-E was highest in ApoE ε4 homozygotes 5% (7/141), compared to heterozygotes 0.4% (2/479) or noncarriers 0% (0/278). Among LEQEMBI-treated patients, the rate of severe radiographic ARIA-H was highest in ApoE ε4 homozygotes 13.5% (19/141), compared to heterozygotes 2.1% (10/479) or noncarriers 1.1% (3/278).

Intracerebral Hemorrhage

- Intracerebral hemorrhage >1 cm in diameter was reported in 0.7% (6/898) of patients in Study 2 after treatment with LEQEMBI compared to 0.1% (1/897) on placebo. Fatal events of intracerebral hemorrhage in patients taking LEQEMBI have been reported.

Concomitant Antithrombotic Medication:

- In Study 2, baseline use of antithrombotic medication (aspirin, other antiplatelets, or anticoagulants) was allowed if the patient was on a stable dose. The majority of exposures to antithrombotic medications were to aspirin. Antithrombotic medications did not increase the risk of ARIA with LEQEMBI. The incidence of intracerebral hemorrhage was 0.9% (3/328 patients) in patients taking LEQEMBI with a concomitant antithrombotic medication at the time of the event compared to 0.6% (3/545 patients) in those who did not receive an antithrombotic. Patients taking LEQEMBI with an anticoagulant alone or combined with an antiplatelet medication or aspirin had an incidence of intracerebral hemorrhage of 2.5% (2/79 patients) compared to none in patients who received placebo.
- Because intracerebral hemorrhages >1 cm in diameter have been observed in patients taking LEQEMBI, additional caution should be exercised when considering the administration of anticoagulants or a thrombolytic agent (e.g., tissue plasminogen activator) to a patient already being treated with LEQEMBI.

Other Risk Factors for Intracerebral Hemorrhage:

- Patients were excluded from enrollment in Study 2 for findings on neuroimaging that indicated an increased risk for intracerebral hemorrhage. These included findings suggestive of cerebral amyloid angiopathy (prior cerebral hemorrhage >1 cm in greatest diameter, >4 microhemorrhages, superficial siderosis, vasogenic edema) or other lesions (aneurysm, vascular malformation) that could potentially increase the risk of intracerebral hemorrhage. The presence of an ApoE ε4 allele is also associated with cerebral amyloid angiopathy, which has an increased risk for intracerebral hemorrhage. Caution should be exercised when considering the use of LEQEMBI in patients with factors that indicate an increased risk for intracerebral hemorrhage and in particular for patients who need to be on anticoagulant therapy.

HYPERSENSITIVITY REACTIONS

Hypersensitivity reactions, including angioedema, bronchospasm, and anaphylaxis, have occurred in LEQEMBI-treated patients. Promptly discontinue the infusion upon the first observation of any signs or symptoms consistent with a hypersensitivity reaction, and initiate appropriate therapy.

INFUSION-RELATED REACTIONS

- In Study 2, infusion-related reactions were observed in LEQEMBI: 26% (237/898); placebo: 7% (66/897), and the majority of cases in LEQEMBI-treated patients (75%, 178/237) occurred with the first infusion. Infusion-related reactions were mostly mild (69%) or moderate (28%) in severity. Infusion-related reactions resulted in discontinuations in 1% (12/898) of LEQEMBI-treated patients. Symptoms of infusion-related reactions included fever and flu-like symptoms (chills, generalized aches, feeling shaky, and joint pain), nausea, vomiting, hypotension, hypertension, and oxygen desaturation.
- In the event of an infusion-related reaction, the infusion rate may be reduced, or the infusion may be discontinued, and appropriate therapy initiated as clinically indicated. Prophylactic treatment with antihistamines, acetaminophen, nonsteroidal anti-inflammatory drugs, or corticosteroids prior to future infusions may be considered.

ADVERSE REACTIONS

- In Study 2, the most common adverse reactions leading to discontinuation of LEQEMBI was ARIA-H microhemorrhages that led to discontinuation in 2% (15/898) of patients treated with LEQEMBI compared to <1% (1/897) of patients on placebo.
- In Study 2, the most common adverse reactions reported in ≥5% of patients treated with LEQEMBI (N=898) and ≥2% higher than placebo (N=897) were infusion-related reactions (LEQEMBI: 26%; placebo: 7%), ARIA-H (LEQEMBI: 14%; placebo: 8%), ARIA-E (LEQEMBI: 13%; placebo: 2%), headache (LEQEMBI: 11%; placebo: 8%), superficial siderosis of central nervous system (LEQEMBI: 6%; placebo: 3%), rash (LEQEMBI: 6%; placebo: 4%), and nausea/vomiting

(LEQEMBI: 6%; placebo: 4%).

Please see full Prescribing Information for LEQEMBI, including Boxed WARNING.

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[Notes to editors]

1. **About Lecanemab (generic name, U.S. brand name: LEQEMBI®),**
Lecanemab is the result of a strategic research alliance between Eisai and BioArctic. Lecanemab is a humanized immunoglobulin gamma 1 (IgG1) monoclonal antibody directed against aggregated soluble (protofibril) and insoluble forms of amyloid-beta (Aβ). In the U.S., LEQEMBI was granted traditional approval by the U.S. Food and Drug Administration (FDA) on July 6, 2023. LEQEMBI is an amyloid beta-directed antibody indicated as a disease-modifying treatment for Alzheimer's disease (AD) in the U.S. Treatment with LEQEMBI should be initiated in patients with mild cognitive impairment (MCI) or mild dementia stage of disease, the population in which treatment was initiated in clinical trials. There are no safety or effectiveness data on initiating treatment at earlier or later stages of the disease than were studied. In Japan, Eisai received approval from the Ministry of Health, Labour and Welfare (MHLW) on September 25, 2023 to manufacture and market of lecanemab as a treatment for slowing progression of MCI and mild dementia due to AD.

Please see full U.S. Prescribing Information for LEQEMBI, including Boxed WARNING.

Eisai has also submitted applications for approval of lecanemab in EU, China, Canada, Great Britain, Australia, Switzerland, South Korea and Israel. In China and Israel, the applications have been designated for priority review, and in Great Britain, lecanemab has been designated for the Innovative Licensing and Access Pathway (ILAP), which aims to reduce the time to market for innovative medicines.

Eisai has completed a lecanemab subcutaneous bioavailability study, and subcutaneous dosing is still being evaluated in the Clarity AD (Study 301) open-label extension (OLE). A maintenance dosing regimen has been evaluated as part of Study 201.

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 LEQEMBI 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 LEQEMBI for the treatment of AD pursuant to an agreement with BioArctic in December 2007. The development and commercialization agreement on the antibody LEQEMBI 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.

5. **About Biogen**
Founded in 1978, Biogen is a leading global biotechnology company that has pioneered multiple breakthrough innovations including a broad portfolio of medicines to treat multiple sclerosis, the first approved treatment for spinal muscular atrophy, and two co-developed treatments to address a

defining pathology of Alzheimer's disease. Biogen is advancing a pipeline of potential novel therapies across neurology, neuropsychiatry, specialized immunology and rare diseases and remains acutely focused on its purpose of serving humanity through science while advancing a healthier, more sustainable and equitable world.

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

Biogen Safe Harbor

This news release contains forward-looking statements about the potential clinical effects of LEQEMBI; the potential benefits, safety and efficacy of LEQEMBI; potential regulatory discussions, submissions and approvals and the timing thereof; the treatment of Alzheimer's disease; the anticipated benefits and potential of Biogen's collaboration arrangements with Eisai; the potential of Biogen's commercial business and pipeline programs, including LEQEMBI; and risks and uncertainties associated with drug development and commercialization. These statements may be identified by words such as "aim," "anticipate," "believe," "could," "estimate," "expect," "forecast," "intend," "may," "plan," "possible," "potential," "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 studies may not be indicative of full results or results from later stage or larger scale clinical studies and do not ensure regulatory approval. You should not place undue reliance on these statements.

These statements involve risks and uncertainties that could cause actual results to differ materially from those reflected in such statements, including without limitation unexpected concerns that may arise from additional data, analysis or results obtained during clinical studies, including the Clarity AD clinical trial, AHEAD 3-45 study and SC substudy; the occurrence of adverse safety events; risks of unexpected costs or delays; the risk of other unexpected hurdles; regulatory submissions may take longer or be more difficult to complete than expected; regulatory authorities may require additional information or further studies, or may fail or refuse to approve or may delay approval of Biogen's drug candidates, including LEQEMBI; actual timing and content of submissions to and decisions made by the regulatory authorities regarding LEQEMBI; uncertainty of success in the development and potential commercialization of LEQEMBI; failure to protect and enforce Biogen's data, intellectual property and other proprietary rights and uncertainties relating to intellectual property claims and challenges; product liability claims; third party collaboration risks; and the direct and indirect impacts of the ongoing COVID-19 pandemic on Biogen's business, results of operations and financial condition. The foregoing sets forth many, but not all, of the factors that could cause actual results to differ from Biogen's expectations in any forward-looking statement. Investors should consider this cautionary statement as well as the risk factors identified in Biogen's most recent annual or quarterly report and in other reports Biogen has filed with the U.S. Securities and Exchange Commission. These statements speak only as of the date of this news release. Biogen does not undertake any obligation to publicly update any forward-looking statements.

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