Eisai Presents New Data on the Continued and Expanding Benefit of LEQEMBI® (lecanemab-irmb) Maintenance Treatment in Early Alzheimer's Disease at the Clinical Trials on Alzheimer's Disease (CTAD) Conference 2025

December 3, 2025

Long-term LEQEMBI treatment suggests potential to delay disease progression from Mild Cognitive Impairment (MCI) to moderate Alzheimer's disease by up to 8.3 years in low-amyloid group who started treatment at an early stage

New safety and efficacy data presented at scientific symposium on subcutaneous formulation for LEQEMBI initiation treatment, which is under regulatory review in the United States

TOKYO and CAMBRIDGE, Mass., Dec. 03, 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 latest findings on time savings with continued treatment with humanized anti-soluble aggregated amyloid-beta (Aβ) monoclonal antibody lecanemab (generic name, U.S. brand name LEQEMBI®) were presented at the 18th Clinical Trials on Alzheimer's Disease (CTAD) Conference. Additionally, a scientific symposium was held on the subcutaneous formulation (SC-AI), which was approved for maintenance treatment in the United States in August 2025, and the rolling supplemental Biologics License Application (sBLA) for initiation treatment was completed in November 2025. The application for a subcutaneous injectable formulation in Japan was submitted in November 2025.

Alzheimer's disease (AD) is a progressive, relentless disease with Aβ and tau as hallmarks, that is caused by a continuous underlying neurotoxic process driven by protofibrils* (PF) that begins before amyloid plaque removal and continues afterward.[1,2,3] Only LEQEMBI fights AD in two ways – targeting both PF and amyloid plaque, which can impact tau downstream.

Estimating the 10-Year Time-Savings Benefits of Lecanemab Treatment (Presentation: December 3, 2:40 PM PT)
This analysis used data from the Clarity AD open-label extension (OLE) and 16 clinical studies of monoclonal antibodies for AD to estimate long-term AD progression over 10 years and the slowing effect of continued lecanemab treatment. The analysis evaluated estimated "time savings" (slowing of disease progression) compared to natural decline based on ADNI** (Alzheimer's Disease Neuroimaging Initiative) data (untreated group), using Clinical Dementia Rating - Sum of Boxes (CDR-SB). These results suggest that early initiation and long-term lecanemab treatment may continue to slow AD progression and help maintain cognitive function over a longer period.

Findings from each group:

- Time Savings from Mild Cognitive Impairment (MCI) Due to AD to Mild AD
  - The time to progression from MCI due to AD to mild AD was 7.2 years in the untreated group, whereas with continued LEQEMBI treatment to the onset of moderate AD, progression to mild AD took 9.7 years, indicating a time savings of 2.5 years.
  - In the low-amyloid group (patients who started treatment at an early stage: amyloid PET <60 centiloids), the time to progression from MCI to mild AD was 13.2 years with continued LEQEMBI treatment to the onset of moderate AD, suggesting a time savings of 6.0 years.

- Time Savings from MCI due to AD to Moderate AD

  - The time to progression from MCI due to AD to moderate AD was 10.1 years in the untreated group, whereas with continued LEQEMBI treatment to the onset of moderate AD, progression to moderate AD took 13.6 years, indicating a time savings of 3.5 years.
  - In the low-amyloid group, the time to progression with continued LEQEMBI treatment to the onset of moderate AD was 18.4 years, suggesting a time savings of 8.3 years.

These findings indicate that earlier initiation of LEQEMBI treatment may provide a greater delay in disease progression. Furthermore, each additional year on LEQEMBI could further delay disease progression compared to stopping treatment, even long after plaque is expected to have been cleared.

Lecanemab Subcutaneous Formulation for Treatment Initiation in Early Alzheimer's Disease: Optimizing Patient Care with a Potential New Option (Symposium Presentation: December 3, 3:10 PM PT)
In this symposium, the latest data from the lecanemab subcutaneous clinical development program were presented focusing on treatment initiation, including results from the subcutaneous (SC) formulation subcohort (n=273) in the Clarity AD trial OLE. It was shown that weekly administration of lecanemab SC-AI at 500 mg (two 250 mg injections) demonstrated bioequivalence in drug exposure compared to intravenous (IV) dosing of 10 mg/kg every two weeks (exposure ratio: 104%, 90% CI: 99.1%–109%).

Based on clinical data and modeling analysis, the effect on amyloid removal in the brain and safety (ARIA-E incidence) was shown to be independent of the route of administration and explained by exposure, suggesting that weekly 500 mg SC dosing provides similar efficacy and safety to biweekly 10 mg/kg IV dosing. Additionally, ARIA-E incidence was also predicted to be comparable between SC and IV administration (12.4% overall, 30.9% in ApoE4 homozygotes).

In this sub-cohort which had prior exposure to lecanemab, safety evaluation showed systemic infusion reactions occurred in 0% of patients receiving 500 mg SC, all of whom had previously received IV lecanemab, and 1.4% of patients who initiated on 720 mg SC by vial, which is favorable compared to systemic infusion reactions in the IV group (26.4%). Immunogenicity assessment indicated a low incidence of anti-drug antibodies (ADA) at 1.4%.

These results indicate that the subcutaneous formulations of lecanemab, designed with consideration for the convenience of patients and their care partners, maintains efficacy with a low incidence of systemic infusion reactions, and is otherwise equivalent to conventional IV administration.

Eisai serves as the lead for lecanemab's development and regulatory submissions globally with Eisai and Biogen 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.[3] 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.[4]

** ADNI is a clinical research project launched in 2005 to develop methods to predict the onset and progression of AD and to confirm the effectiveness of treatments. The project involves a multi-year longitudinal observation targeting healthy elderly individuals as well as patients with mild cognitive impairment (MCI) and early stages of AD.

INDICATION

LEQEMBI® is indicated for the treatment of Alzheimer's disease (AD). 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.

IMPORTANT SAFETY INFORMATION

WARNING: AMYLOID-RELATED IMAGING ABNORMALITIES (ARIA)

- **Monoclonal antibodies directed against aggregated forms of beta amyloid, including LEQEMBI, can cause 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, including seizure and status epilepticus, can occur. ARIA can be fatal. Serious intracerebral hemorrhages (ICH) >1 cm, some of which have been fatal, have been observed with this class of medications. Because ARIA-E can cause focal neurologic deficits that can mimic an ischemic stroke, consider whether such symptoms could be due to ARIA-E before giving thrombolytic therapy to a patient being treated with LEQEMBI.**
  - **Apolipoprotein E ε4 (ApoE ε4) Homozygotes: Patients who are ApoE ε4 homozygotes (~15% of patients with AD) treated with this class of medications 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 AD and the potential risk of serious ARIA events when deciding to initiate treatment with LEQEMBI.**

CONTRAINDICATION

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

WARNINGS AND PRECAUTIONS

Amyloid-Related Imaging Abnormalities

Medications in this class, including LEQEMBI, can cause ARIA-E, which can be observed on MRI as brain edema or sulcal effusions, and ARIA-H, which includes microhemorrhage and superficial siderosis. ARIA can occur spontaneously in patients with AD, particularly in patients with MRI findings suggestive of cerebral amyloid angiopathy (CAA), such as pretreatment microhemorrhage or superficial siderosis. ARIA-H generally occurs with ARIA-E. Reported ARIA symptoms may include headache, confusion, visual changes, dizziness, nausea, and gait difficulty. Focal neurologic deficits may also occur. Symptoms usually resolve over time.

Incidence of ARIA

Symptomatic ARIA occurred in 3% and serious ARIA symptoms in 0.7% with LEQEMBI. Clinical ARIA symptoms resolved in 79% of patients during the period of observation. ARIA, including asymptomatic radiographic events, was observed: LEQEMBI, 21%; placebo, 9%. ARIA-E was observed: LEQEMBI, 13%; placebo, 2%. ARIA-H was observed: LEQEMBI, 17%; placebo, 9%. No increase in isolated ARIA-H was observed for LEQEMBI vs placebo.

Incidence of ICH

ICH >1 cm in diameter was reported in 0.7% with LEQEMBI vs 0.1% with placebo. Fatal events of ICH in patients taking LEQEMBI have been observed.

Risk Factors of ARIA and ICH

ApoE ε4 Carrier Status

Of the patients taking LEQEMBI, 16% were ApoE ε4 homozygotes, 53% were heterozygotes, and 31% were noncarriers. With LEQEMBI, ARIA was higher in ApoE ε4 homozygotes (LEQEMBI: 45%; placebo: 22%) than in heterozygotes (LEQEMBI: 19%; placebo: 9%) and noncarriers (LEQEMBI: 13%; placebo: 4%). Symptomatic ARIA-E occurred in 9% of ApoE ε4 homozygotes vs 2% of heterozygotes and 1% of noncarriers. Serious ARIA events occurred in 3% of ApoE ε4 homozygotes and in ~1% of heterozygotes and noncarriers. The recommendations on management of ARIA do not differ between ApoE ε4 carriers and noncarriers.

Radiographic Findings of CAA

Neuroimaging findings that may indicate CAA include evidence of prior ICH, cerebral microhemorrhage, and cortical superficial siderosis. CAA has an increased risk for ICH. The presence of an ApoE ε4 allele is also associated with CAA.

The baseline presence of at least 2 microhemorrhages or the presence of at least 1 area of superficial siderosis on MRI, which may be suggestive of CAA, have been identified as risk factors for ARIA. Patients were excluded from Clarity AD for the presence of >4 microhemorrhages and additional findings suggestive of CAA (prior cerebral hemorrhage >1 cm in greatest diameter, superficial siderosis, vasogenic edema) or other lesions

(aneurysm, vascular malformation) that could potentially increase the risk of ICH.

Concomitant Antithrombotic or Thrombolytic Medication
In Clarity AD, baseline use of antithrombotic medication (aspirin, other antiplatelets, or anticoagulants) was allowed if the patient was on a stable dose. Most exposures were to aspirin. Antithrombotic medications did not increase the risk of ARIA with LEQEMBI. The incidence of ICH: 0.9% in patients taking LEQEMBI with a concomitant antithrombotic medication vs 0.6% with no antithrombotic and 2.5% in patients taking LEQEMBI with an anticoagulant alone or with antiplatelet medication such as aspirin vs none in patients receiving placebo.

Fatal cerebral hemorrhage has occurred in 1 patient taking an anti-amyloid monoclonal antibody in the setting of focal neurologic symptoms of ARIA and the use of a thrombolytic agent.

Additional caution should be exercised when considering the administration of antithrombotics or a thrombolytic agent (e.g., tissue plasminogen activator) to a patient already being treated with LEQEMBI. Because ARIA-E can cause focal neurologic deficits that can mimic an ischemic stroke, treating clinicians should consider whether such symptoms could be due to ARIA-E before giving thrombolytic therapy in a patient being treated with LEQEMBI.

Caution should be exercised when considering the use of LEQEMBI in patients with factors that indicate an increased risk for ICH and, in particular, patients who need to be on anticoagulant therapy or patients with findings on MRI that are suggestive of CAA.

Radiographic Severity With LEQEMBI
Most ARIA-E radiographic events occurred within the first 7 doses, although ARIA can occur at any time, and patients can have >1 episode. Maximum radiographic severity of ARIA-E with LEQEMBI was mild in 4%, moderate in 7%, and severe in 1% of patients. Resolution on MRI occurred in 52% of ARIA-E patients by 12 weeks, 81% by 17 weeks, and 100% overall after detection. Maximum radiographic severity of ARIA-H microhemorrhage with LEQEMBI was mild in 9%, moderate in 2%, and severe in 3% of patients; superficial siderosis was mild in 4%, moderate in 1%, and severe in 0.4% of patients. With LEQEMBI, the rate of severe radiographic ARIA-E was highest in ApoE ε4 homozygotes (5%) vs heterozygotes (0.4%) or noncarriers (0%). With LEQEMBI, the rate of severe radiographic ARIA-H was highest in ApoE ε4 homozygotes (13.5%) vs heterozygotes (2.1%) or noncarriers (1.1%).

Monitoring and Dose Management Guidelines
Baseline brain MRI and periodic monitoring with MRI are recommended. Enhanced clinical vigilance for ARIA is recommended during the first 14 weeks of treatment. Depending on ARIA-E and ARIA-H clinical symptoms and radiographic severity, use clinical judgment when considering whether to continue dosing or to temporarily or permanently discontinue LEQEMBI. If a patient experiences ARIA symptoms, 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.

Hypersensitivity Reactions
Hypersensitivity reactions, including angioedema, bronchospasm, and anaphylaxis, have occurred with LEQEMBI. 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 (IRRs)
IRRs were observed—LEQEMBI: 26%; placebo: 7%—and most cases with LEQEMBI (75%) occurred with the first infusion. IRRs were mostly mild (69%) or moderate (28%). Symptoms included fever and flu-like symptoms (chills, generalized aches, feeling shaky, and joint pain), nausea, vomiting, hypotension, hypertension, and oxygen desaturation.

IRRs can occur during or after the completion of infusion. In the event of an IRR during the infusion, the infusion rate may be reduced or discontinued, and appropriate therapy initiated as clinically indicated. Consider prophylactic treatment prior to future infusions with antihistamines, acetaminophen, nonsteroidal anti-inflammatory drugs, or corticosteroids.

ADVERSE REACTIONS

- The most common adverse reactions reported in ≥5% with LEQEMBI infusion every 2 weeks and ≥2% higher than placebo were IRRs (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%)
- Safety profile of LEQEMBI IQLIK for maintenance treatment was similar to LEQEMBI infusion. Patients who received LEQEMBI IQLIK experienced localized and systemic (less frequent) injection-related reactions (mild to moderate in severity)

LEQEMBI (lecanemab-irmb) is available:

- Intravenous infusion: 100 mg/mL
- Subcutaneous injection: 200 mg/mL

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

MEDIA CONTACTS

Eisai Co., Ltd.
Public Relations Department
TEL: +81 (0)3-3817-5120

Eisai Europe, Ltd.
EMEA Communications Department
+44 (0) 797 487 9419
Emea-comms@eisai.net

Eisai Inc. (U.S.)
Libby Holman
+1-201-753-1945
Libby_Holman@Eisai.com

Biogen Inc.
Madeleine Shin
+1-781-464-3260
public.affairs@biogen.com

INVESTOR CONTACTS

Eisai Co., Ltd.
Investor Relations Department
TEL: +81 (0) 3-3817-5122

Biogen Inc.
Tim Power
+ 1-781-464-2442
IR@biogen.com

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).

   Lecanemab has been approved in 51 countries and regions including Japan, the United States, Europe, China, South Korea, Taiwan, and Saudi Arabia, and is under regulatory review in 9 countries. LEQEMBI received manufacturing and marketing approval in Japan in September, 2023 for the indication of slowing progression of mild cognitive impairment (MCI) and mild dementia due to Alzheimer's disease (AD). 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, the United Kingdom, and others, 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. A rolling Supplemental Biologics License Application (sBLA) for initiation treatment was initiated under Fast Track status in September 2025, and completed in November 2025. In November 2025, an application for a subcutaneous injectable formulation in Japan was submitted.

   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 (LEQEMBI); the potential benefits, safety and efficacy of lecanemab; 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 lecanemab; and risks and uncertainties associated with drug development and commercialization. These forward-looking statements may be accompanied by such words as “aim,” “anticipate,” “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 realised 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 our long-term success in developing, licensing, or acquiring other product candidates or additional indications for existing products; expectations, plans, prospects and timing of actions relating to product approvals, approvals of additional indications for our existing products, sales, pricing, growth, reimbursement and launch of our marketed and pipeline products; the potential impact of increased product competition in the biopharmaceutical and healthcare industry, as well as any other markets in which we compete, including increased competition from new originator therapies, generics, prodrugs and biosimilars of existing products and products approved under abbreviated regulatory pathways; our ability to effectively implement our corporate strategy; difficulties in obtaining and maintaining adequate coverage, pricing, and reimbursement for our products; the drivers for growing our business, including our dependence on collaborators and other third parties for the development, regulatory approval, and commercialization of products and other aspects of our business, which are outside of our full control; risks related to commercialization of biosimilars, which is subject to such risks related to our reliance on third-parties, intellectual property, competitive and market challenges and regulatory compliance; 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; and 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, which are available on the SEC’s website at www.sec.gov.

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. 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.

Digital Media Disclosures
From time to time, we have used, or expect in the future to use, our investor relations website (investors.biogen.com), the Biogen LinkedIn account (linkedin.com/company/biogen-) and the Biogen X account (https://x.com/biogen) as a means of disclosing information to the public in a broad, non-exclusionary manner, including for purposes of the SEC’s Regulation Fair Disclosure (Reg FD). Accordingly, investors should monitor our investor relations website and these social media channels in addition to our press releases, SEC filings, public conference calls and websites, as the information posted on them could be material to investors.

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