No.26-39

July 14, 2026
Eisai Co., Ltd.

Eisai Presents Latest Findings Showed Etalanetug Reduced Alzheimer's Disease Tau Tangle-Specific Plasma Biomarker MTBR-tau243 at Alzheimer's Association International Conference® (AAIC®) 2026

Measuring the plasma eMTBR-tau243 biomarker may serve as a practical, less invasive method of capturing Alzheimer's disease-specific pathological changes and may play an important role in etalanetug's future clinical development

TOKYO, July 14, 2026 — Eisai Co., Ltd. today announced the latest findings on etalanetug (development code: E2814) showed that this investigational anti-MTBR antibody reduced levels of plasma eMTBR-tau243, a key biomarker of Alzheimer's disease (AD) tau tangle pathology. The investigational compound etalanetug may have the potential to bind to the microtubule-binding region (MTBR) of tau protein and prevent the seeding and propagation of tau pathology in the brain. Tau tangles are a hallmark of AD and are believed to be strongly associated with memory loss, cognitive decline, and disease progression.[1] The findings were presented during the Featured Research Session, "Mechanisms Beyond Amyloid: Etalanetug Reduces Tau Tangle-Specific Plasma Biomarker eMTBR-tau243 in Dominantly Inherited Alzheimer's Disease (DIAD)," at the Alzheimer's Association International Conference® (AAIC®) 2026 in London and online.

The highly sensitive blood biomarker assay measuring eMTBR-tau243 was developed as a potential alternative for cerebrospinal fluid (CSF) and Positron Emission Tomography (PET) testing. This analysis evaluated changes in plasma tau biomarkers following etalanetug administration and compared them with changes observed in CSF biomarkers in individuals with DIAD enrolled in the Phase Ib/II Study 103 (NCT04971733).

Key Findings

Plasma eMTBR-tau243 captures disease-specific pathological changes originating from tau pathology in the brain

- Etalanetug reduced CSF eMTBR-tau243 by 62% at three months and by 89% at nine months.
- Etalanetug reduced plasma eMTBR-tau243 by 78% at 3 months and by more than 90% at 9 months, indicating that plasma eMTBR-tau243 closely mirrored disease-related changes captured in CSF.
- Plasma phosphorylated tau (p-tau217, p-tau181, and p-tau231) and t-tau increased after etalanetug administration in both individuals with DIAD and healthy adults. This change is considered to be attributable to non-CNS tau species derived from peripheral tissues being stabilized by binding to etalanetug in plasma, thereby inhibiting their degradation.
- Plasma eMTBR-tau243 was largely absent in healthy adults and detected only in patients with DIAD, suggesting it reflects disease-related tau pathology. Administration of etalanetug reduced levels of this biomarker.
- These results suggest that plasma eMTBR-tau243, unlike plasma phosphorylated tau species and t-tau, is a biomarker that captures disease-specific pathological changes originating from tau pathology in the brain.

Etalanetug acts on tau pathology in the brain, one of the underlying pathologies of AD

- Etalanetug reduced multiple CSF phosphorylated tau species and t-tau in patients with DIAD. Among these, p-tau205 is a marker reflecting late-stage tau pathology (T2 biomarker) in the Alzheimer's Association guidelines. [2]
- These findings represent the first report of an anti-tau therapy reducing CSF p-tau205 in patients with DIAD.

These results highlight that etalanetug acts on tau pathology in the brain, one of the underlying pathologies of AD. Plasma eMTBR-tau243 may serve as a practical, less invasive biomarker that captures AD-specific pathological changes and may play an important role in the future clinical development of etalanetug.

* eMTBR-tau243 is a novel fluid biomarker consisting of tau fragments that include tau protein amino acid residue 243 and MTBR, with endogenous cleavage at the C-terminal side of residue 256. It is thought to arise during the formation of neurofibrillary tangles, a key pathological feature of AD, and a strong correlation has been shown between tau PET and eMTBR-tau243 in both plasma and CSF.[3]

** DIAD: Dominantly Inherited Alzheimer's disease (DIAD) is a rare form of AD that causes memory loss and dementia in individuals — typically while they are in their 30s to 50s. The disease affects less than 1% of the total population of people with Alzheimer's disease.

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[Notes to editors]

1. **About etalanetug (E2814)**
   Etalanetug is an anti-MTBR (microtubule-binding region) tau antibody discovered through collaborative research between Eisai and University College London. It is designed to inhibit the propagation of tau seeds within the brain. Etalanetug is being developed as a potential disease-modifying therapy for tauopathies, including sporadic Alzheimer's disease (AD).

   Currently, etalanetug is being evaluated in two ongoing clinical studies: the Tau NexGen Phase II/III trial in dominantly inherited Alzheimer's disease (DIAD), conducted under the Dominantly Inherited Alzheimer Network Trials Unit (DIAN-TU) and led by Washington University School of Medicine in St. Louis, added to the standard-of-care anti-Aβ protofibril antibody lecanemab (brand name: LEQEMBI), and the Phase II Study 202, a global randomized trial in individuals with early sporadic AD, also assessing etalanetug added to lecanemab. In September 2025, etalanetug received Fast Track designation from the U.S. Food and Drug Administration (FDA).

2. **Phase 1b/2 Study 103 (NCT04971733)**
   Phase 1b/2 Study 103 is an open-label study evaluating the safety, tolerability, and effects of etalanetug on tau-related biomarkers in cerebrospinal fluid (CSF) in participants with dominantly inherited Alzheimer's disease (DIAD).

References

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