XVIII Convegno

I Centri per i disturbi cognitivi e demenze e la gestione integrata della demenza

VII Sessione

NUOVI FARMACI NELL'ALZHEIMER: DALL'ANALISI PER RESPONDER ALLE STRATEGIE PER LA MINIMIZZAZIONE DEI RISCHI

ISTITUTO SUPERIORE DI SANITÀ

Osservatorio Demenze
ISTITUTO SUPERIORE DI SANITÀ

organizzato da
ISTITUTO SUPERIORE DI SANITÀ
Centro Nazionale Prevenzione delle Malattie e Promozione della Salute (CNaPPS)
Reparto Promozione e Valutazione delle Politiche di Prevenzione delle Malattie Croniche

Le evidenze sul farmaco Lecanemab

S. Sorbi

Università degli Studi di Firenze

PREMESSA

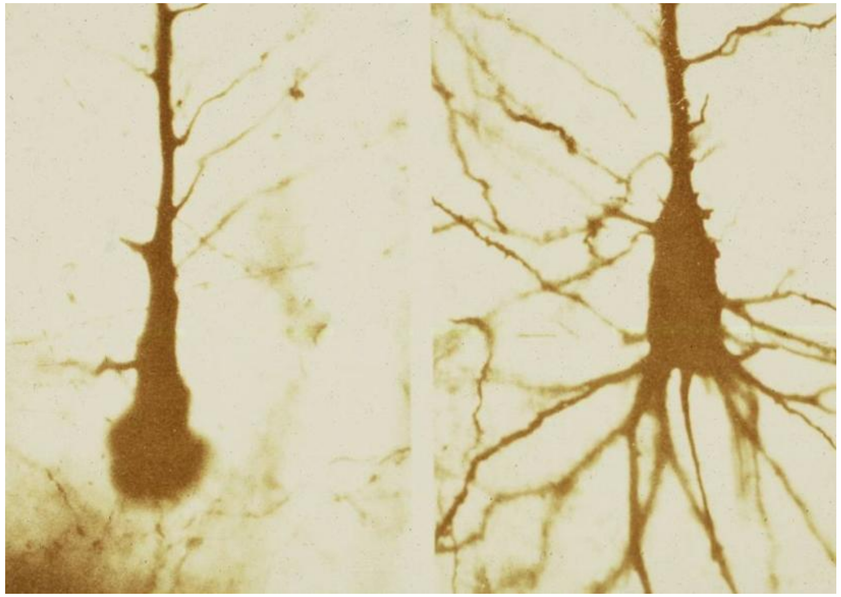
Non ho mai avuto rapporti di consulenza con EISAI.

Non ho partecipato allo studio CLARITY nè allo EXTENSION STUDY open label.

Sono stato invitato da EISAI a presentare Lecanemab in questo convegno.

L’organizzazione della presentazione e la selezione del contenuto è mia; ho richiesto ed ottenuto del materiale ad EISAI.

Left panel shows **loss of dendritic arborization** in Alzheimer's disease. Right panel shows a normal neuron.



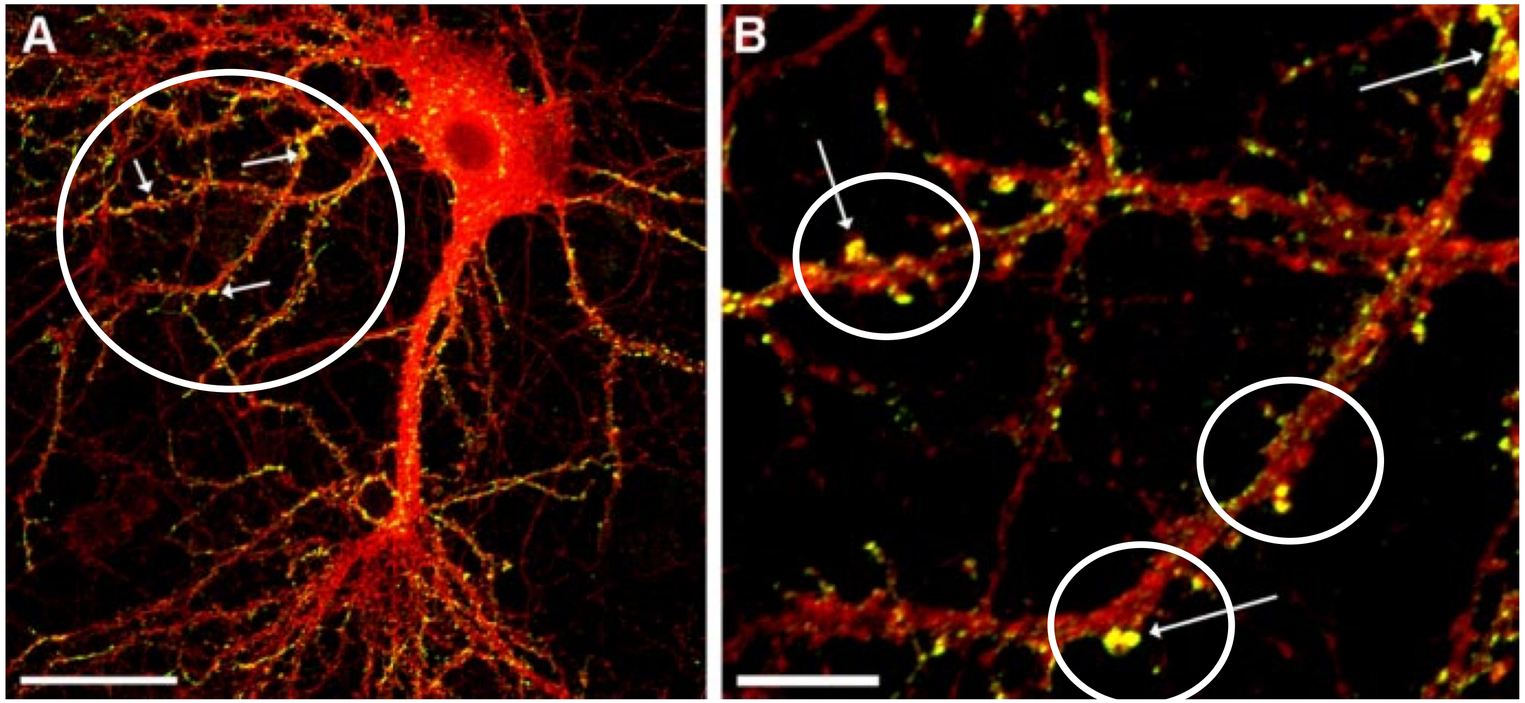
Extensive research supports the hypothesis that **soluble amyloid-beta (Aβ) oligomers in synapses are a primary cause of the initial memory loss and synaptic dysfunction** seen in Alzheimer's disease

The Journal of Neuroscience, November 10, 2004

Synaptic Targeting by Alzheimer's-Related Amyloid β Oligomers

Pascale N. Lacor,[1] Maria C. Buniel,[1] Lei Chang,[1] Sara J. Fernandez,[1] Yuesong Gong,[1] Kirsten L. Viola,[1] Mary P. Lambert,[1] Pauline T. Velasco,[1] Eileen H. Bigio,[2] Caleb E. Finch,[3] Grant A. Krafft,[4] and William L. Klein[1]

[1]Neurobiology and Physiology Department, Northwestern University, Evanston, Illinois 60208, [2]Neuropathology Core, Northwestern Alzheimer's Disease Center, Northwestern Feinberg School of Medicine, Chicago, Illinois 60611, [3]Andrus Gerontology Center, University of Southern California, Los Angeles, California 90089, and [4]Acumen Pharmaceuticals, Glenview, Illinois 60025



Int. J. Mol. Sci. **2021,**

Review

Neurotoxic Soluble Amyloid Oligomers Drive Alzheimer's Pathogenesis and Represent a Clinically Validated Target for Slowing Disease Progression

Martin Tolar *, John Hey, Aidan Power and Susan Abushakra

(1) Only agents that target soluble Aβ oligomers show clinical efficacy in AD patients;
(2) Clearance of amyloid plaque does not correlate with clinical improvements;
(3) Agents that predominantly target amyloid monomers or plaque failed to show clinical effects; and
(4) In positive trials, efficacy is greater in carriers of the ε4 allele of apolipoprotein E (APOE4), who are known to have higher brain concentrations of Aβ oligomers.

International Journal of *Molecular Sciences*

2020

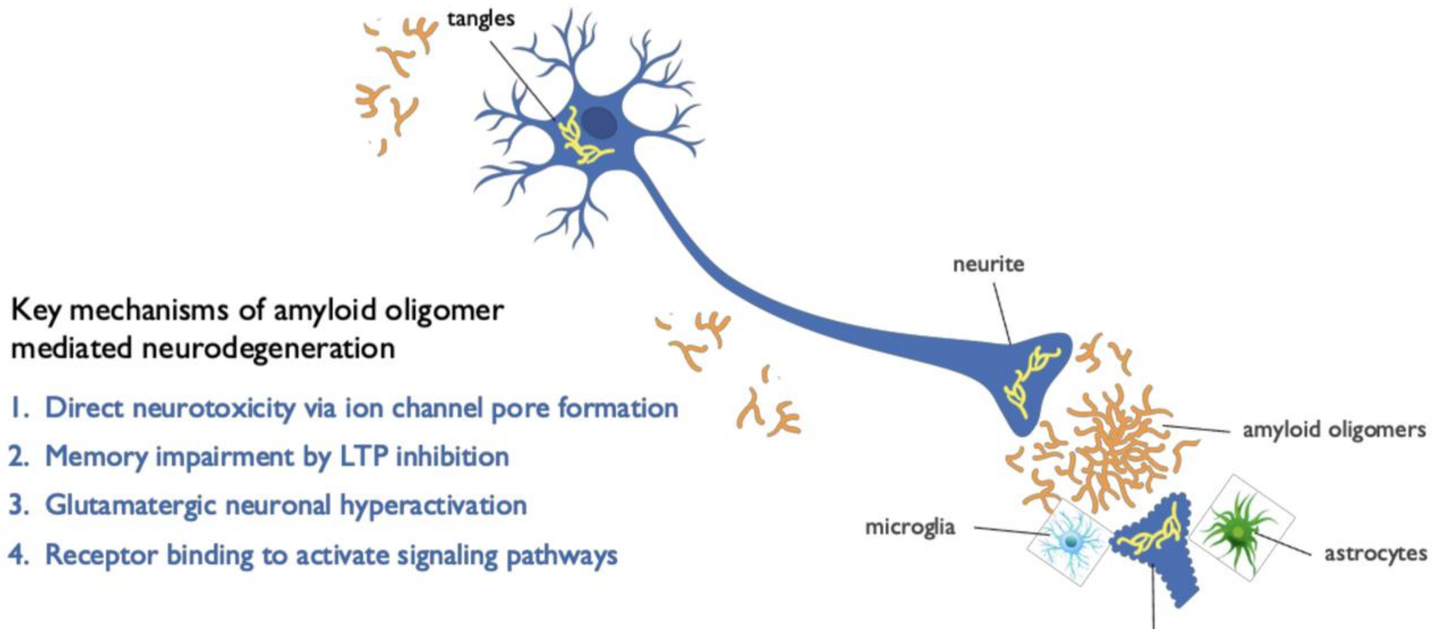
MDPI

Review

Protofibrils of Amyloid-β are Important Targets of a Disease-Modifying Approach for Alzheimer's Disease

Kenjiro Ono [1,*] and Mayumi Tsuji [2]

Arctic transgenic mice, and human samples of AD brain tissues have suggested that the pre-fibrillar forms of Aβ, particularly Aβ protofibrils, may be the most critical species, compared with extracellular fibrillar forms.



Lecanemab has a high affinity for Aβ protofibrils *in vitro*

- Lecanemab is a humanised monoclonal IgG1 antibody with a low affinity for Aβ monomers and **high affinity for aggregated Aβ forms** (>1000x)[1]
- Among aggregated species, **lecanemab shows preferential activity for Aβ protofibrils** over fibrils (>10x)[2]
  - Aβ protofibrils are suggested to be the most critical species to target in AD due to increased toxicity compared with other Aβ species[3–6]

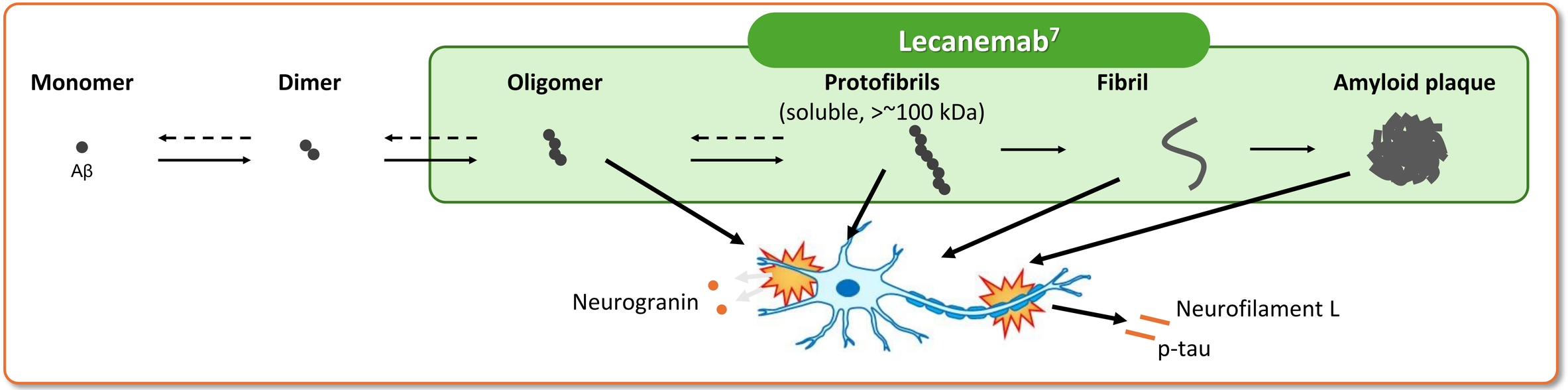


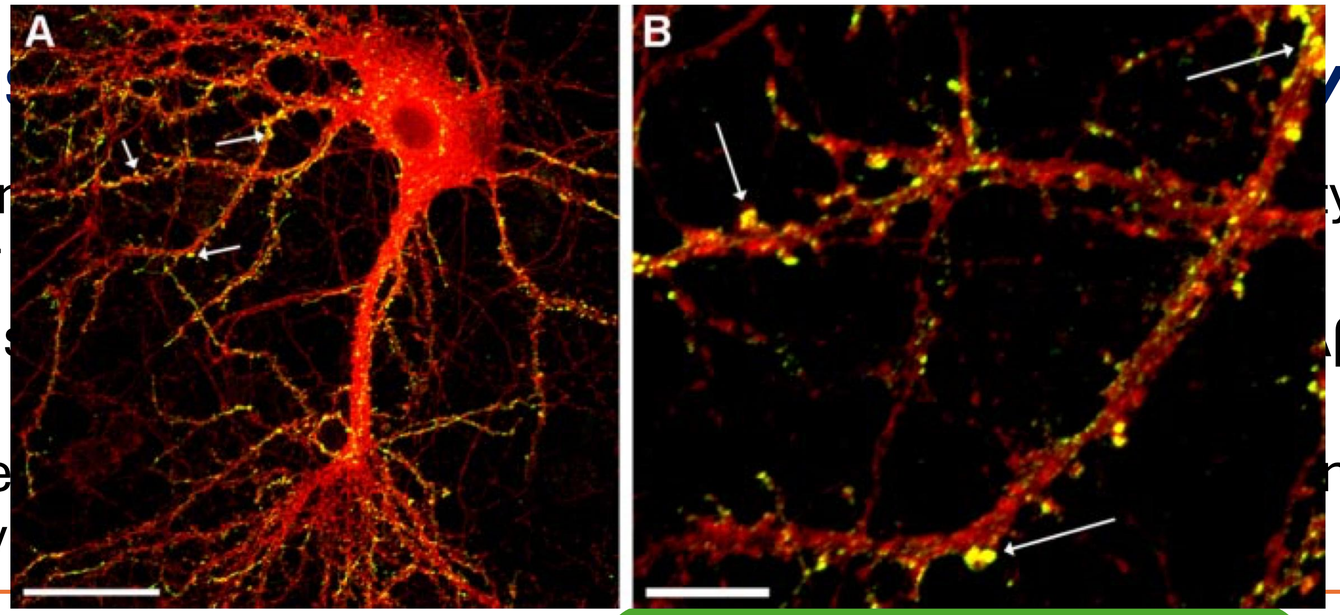
Figure adapted from Swanson CJ, et al. Presented at the Clinical Trials on Alzheimer's Disease Conference 2018; Barcelona, Spain. October 24–27, 2018.

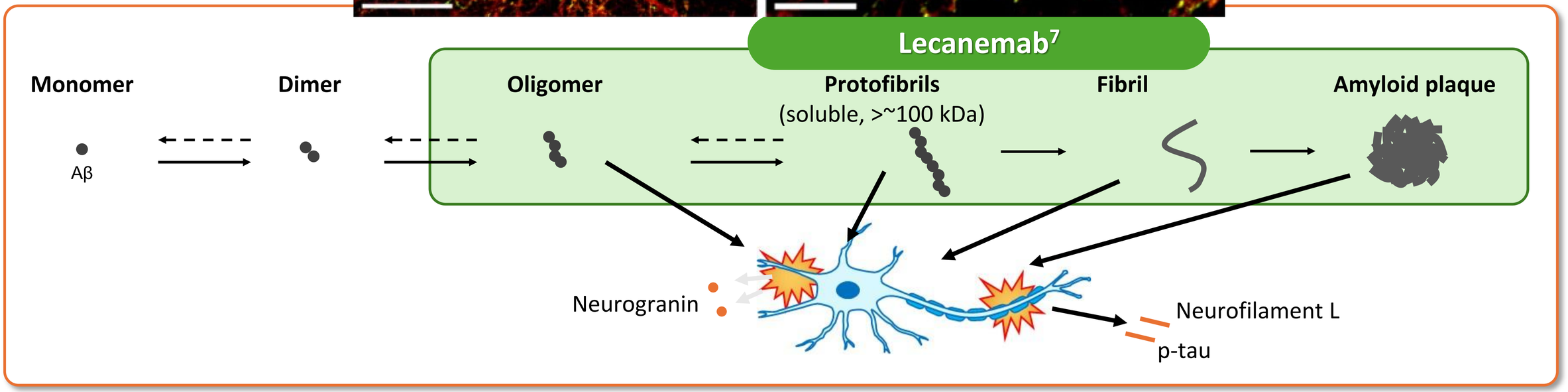
Aβ, amyloid beta; **AD**, Alzheimer's disease; **IgG**, immunoglobulin G; **p-tau**, phosphorylated tau 181.

1. Tucker S, *et al. J Alzheimers Dis*. 2015;43:575–588; **2.** Magnusson K. J. Alzheimers Dis 2013;37:29-40; **3.** Ono K & Tsuji M. *Int J Mol Sci*. 2020;21:952; **4.** Tolar M, *et al. Int J Mol Sci.* 2021;22:6455; **5.** Tolar M, *et al. Alz Dement.* 2019;1–8; **6.** Kayed R & Lasagna-Reeves CA. *JAD.* 2013;33:S67–S78; **7.** Swanson CJ, *et al.* Presented at the Clinical Trials on Alzheimer's Disease Conference 2018; Barcelona, Spain. October 24–27, 2018.

Lecanemab has ...vitro

- Lecanemab is a hu... ...y for Aβ monomers and high affinity for ...
- Among aggregated ... ...Aβ protofibrils over fibrils (>10x)[2]
  - Aβ protofibrils are ... ...n AD due to increased toxicity ...





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LECANEMAB NEL MONDO

CANADA

Lecanemab è stato autorizzato da Health il 27 ottobre 2025

U.S.A

FDA ha concesso l'approvazione accelerata il 6 Gennaio 2023 e l'approvazione standard in data 6 Luglio 2023

In data 26 gennaio 2025, FDA ha approvato la **dose di mantenimento** di lecanemab (1 volta ogni 4 settimane)

In data 30 agosto 2025, FDA ha approvato BLA di lecanemab (1 volta ogni settimana)

Mexico

Lecanemab è stato approvato dal COFEPRIS il 4 dicembre 2024

United Kingdom

Lecanemab è stato autorizzato dal MHRA il 22 agosto 2024

Europe

Eisai ha ricevuto approvazione EMA il 15 aprile

LECANEMAB

Austria	25 agosto 2025
Germania	1 settembre 2025

Israel

Eisai ha ricevuto l'approvazione il 12 luglio 2024

UAE

Eisai ha ricevuto l'approvazione il 14 Agosto 2024

China

Eisai ha ricevuto l'approvazione il 9 Gennaio 2024

South Korea

Eisai ha ricevuto l'approvazione dal MFDS il 27 Maggio 2024

Japan

Eisai ha ricevuto l'approvazione dal MHLW il 25 Settembre 2023

Hong Kong

Eisai ha ricevuto l'approvazione dal Dipartimento della Salute l'11 luglio 2024

Australia

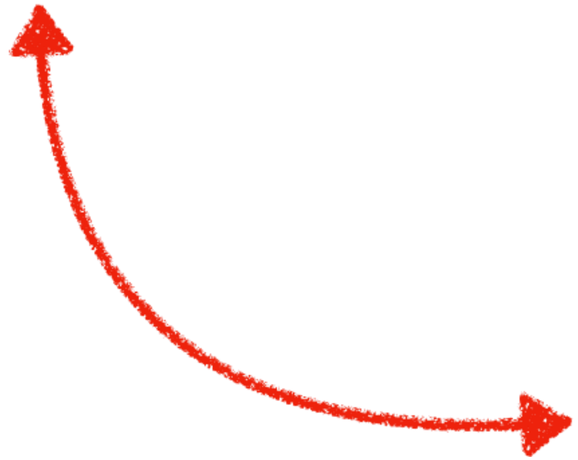
Eisai ha ricevuto l'approvazione il 23 settembre 2025

- ❑ Macau
- ❑ Oman
- ❑ Taiwan
- ❑ Qatar
- ❑ Singapore

Eisai ha inoltre presentato domande di approvazione per lecanemab in altre nazioni e regioni

News Release: Eisai Co., Ltd.

- Clarity randomised
- Clarity extension open-label
- Real life data



- Efficacia
- Effetti collaterali
- Effetto sospensione

Clarity AD Study Design

Clarity AD Core and Open-Label Extension Includes 4 Years of Lecanemab Treatment

Clarity AD = 1795 patients with early AD

Clarity AD is a global, placebo-controlled, double-blind, parallel-group, randomized study

Study Population

- 1,795 participants with Early AD
- MCI due to AD or mild Alzheimer's dementia
- Amyloid pathology confirmed
- MMSE score between 22 and 30 at screening and baseline
- WMS-IV LMSII ≥1 SD below age-adjusted mean at screening

Randomised phase (18 months)

R 1:1

- **Lecanemab** 10 mg/kg biweekly (IV infusion)
- **Placebo** Biweekly (IV infusion)

Extension phase (open-label)

- **Lecanemab** 10 mg/kg biweekly (IV infusion)
- **Lecanemab** 10 mg/kg monthly (IV infusion)
- **Lecanemab** (subcutaneous)

Optional longitudinal sub-studies

- Amyloid burden (amyloid PET; n=716)
- Brain tau pathology (tau PET; n=342)
- CSF biomarkers of neurodegeneration (n=281)
- Subcutaneous formulation (OLE)

Randomization Phase
Primary Outcome Measure:

CDR: Change from Baseline at 18 months

Key Secondary Outcome Measures:

Change from Baseline at 18 months:
Amyloid PET
ADAS-Cog14
ADCOMS
ADCS MCI-ADL

Extension Phase
Primary Outcome Measures

Number of Participants with TEAEs
Change from Core Study Baseline in CDR-SB

Additional Outcome Measures:

Chang from Baseline for:
ADAS-Cog14
ADCS MCI-ADL

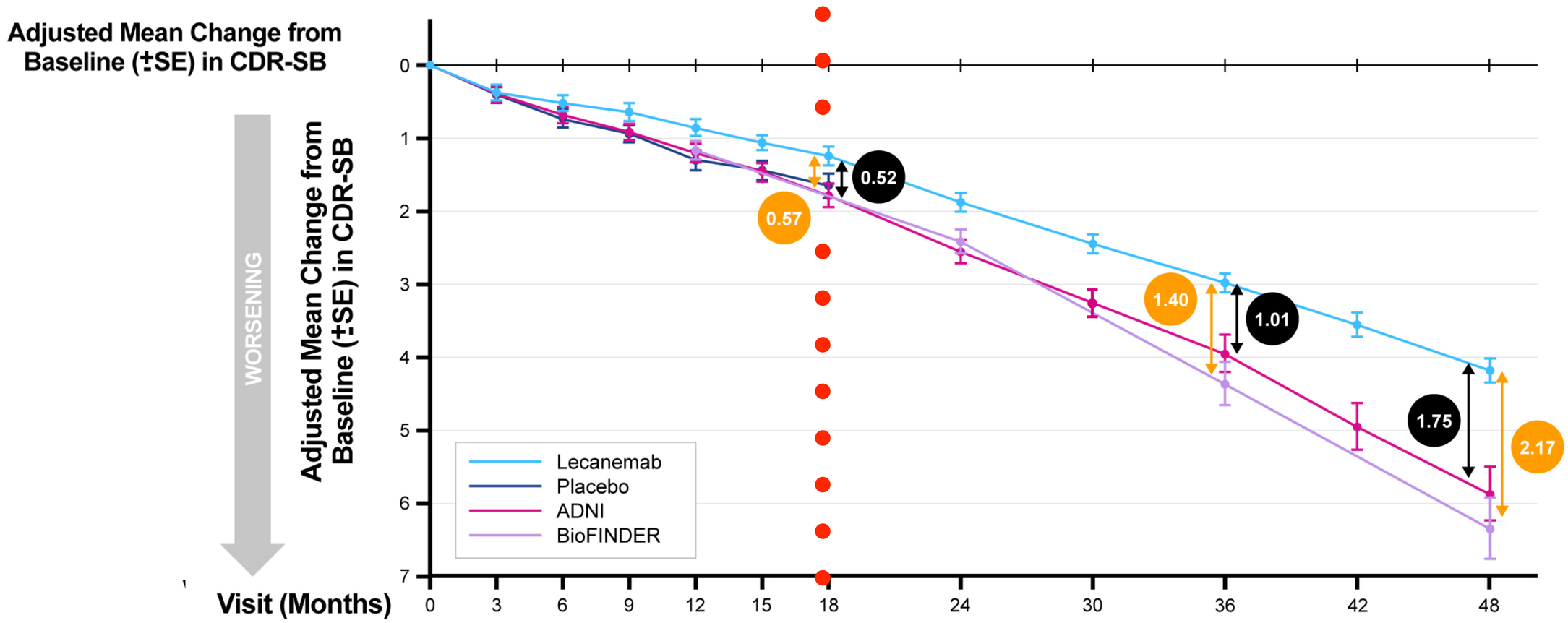
Biomarkers

AD, Alzheimer's disease. ADAS-Cog14, Alzheimer's Disease Assessment Scale-Cognitive Subscale. ADCOMS, Alzheimer's Disease Composite Score. ADCS MCI-ADL, Alzheimer's Disease Cooperative Study-Activities of Daily Living Scale for Mild Cognitive Impairment. ApoE4, apolipoprotein E4. CDR-SB, Clinical Dementia Rating-sum of boxes. CSF, cerebrospinal fluid; IV, intravenous. MCI, mild cognitive impairment; MMSE, Mini-Mental State Exam. OLE, open-label extension. PET, positron emission tomography. SD, standard deviation. TEAEs, treatment emergent adverse events. WMS-IV LMSII, Wechsler Memory Scale IV-Logical Memory (subscale) II. van Dyck CH, et al., N Engl J Med. 2023;388:9-21.

Clarity AD OLE: CDR-SB Efficacy Through 48 Months

Lecanemab-Treated Patients Continue to Accrue Benefit Over Time

CDR-SB



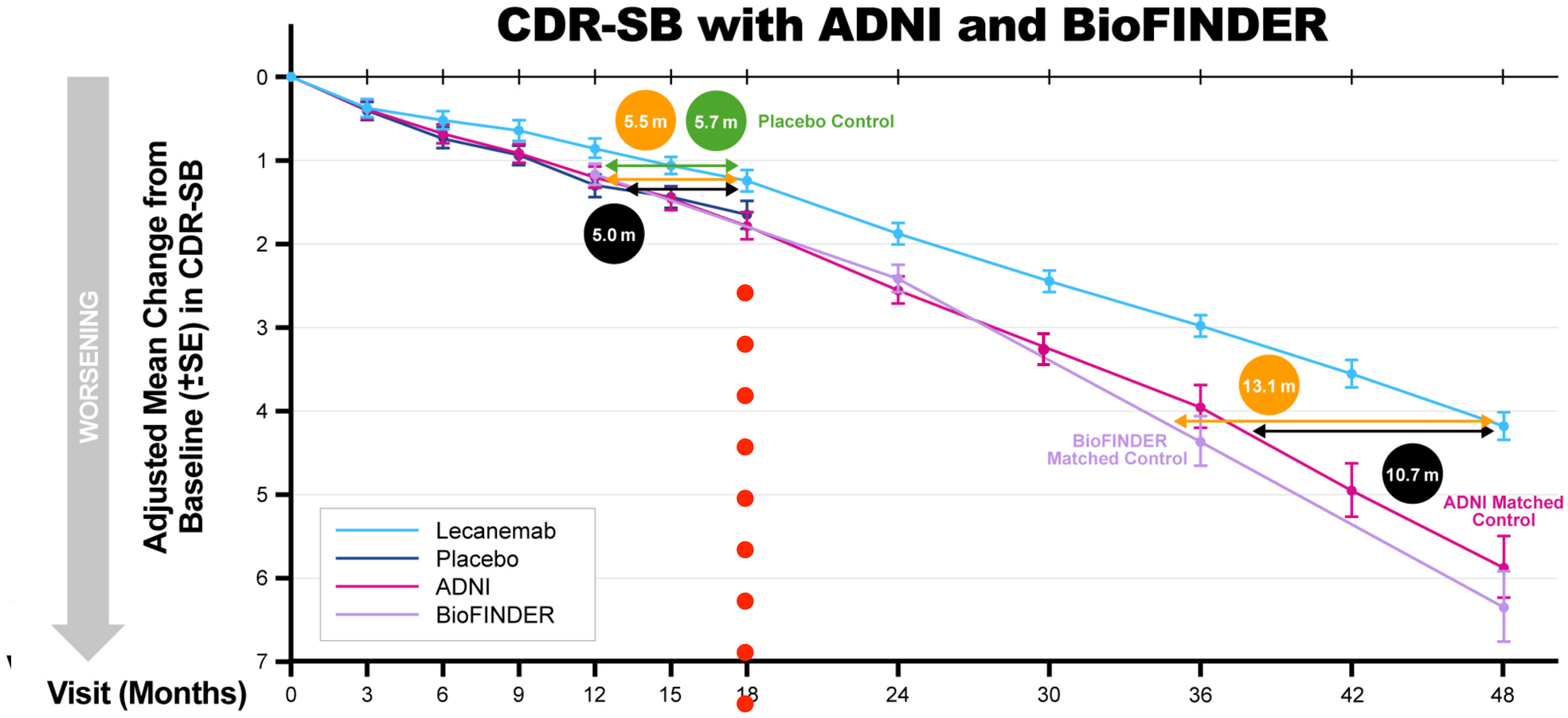
N (Placebo)	875	849	828	813	779	767	757					
N (Lecanemab)	859	824	798	779	765	738	714	659	613	573	527	478
N (ADNI)	436		410		401		121	301	173	173	98	98
N (BioFINDER)	147				139			137		117		112

Note: OLE includes those participants on subcutaneous and intravenous formulations. BioFINDER data are from BioFINDER 1.

Based on modified intention-to-treat analysis population. Adjusted mean change from baseline, SE and p-value are derived using mixed model repeat measures (MMRM) with treatment group, visit, treatment group by visit interaction, clinical subgroup, use of Alzheimer's disease symptomatic medication at baseline, ApoE4 carrier status, region, baseline value by visit interaction as fixed effects, and baseline value as covariate.

ADNI, Alzheimer's Disease Neuroimaging Initiative. CDR-SB, Clinical Dementia Rating-sum of boxes. OLE, open-label extension. SE, standard error.

Lecanemab Extends Time Spent in Early Disease With Increasing Magnitude of Treatment Effect Over Time



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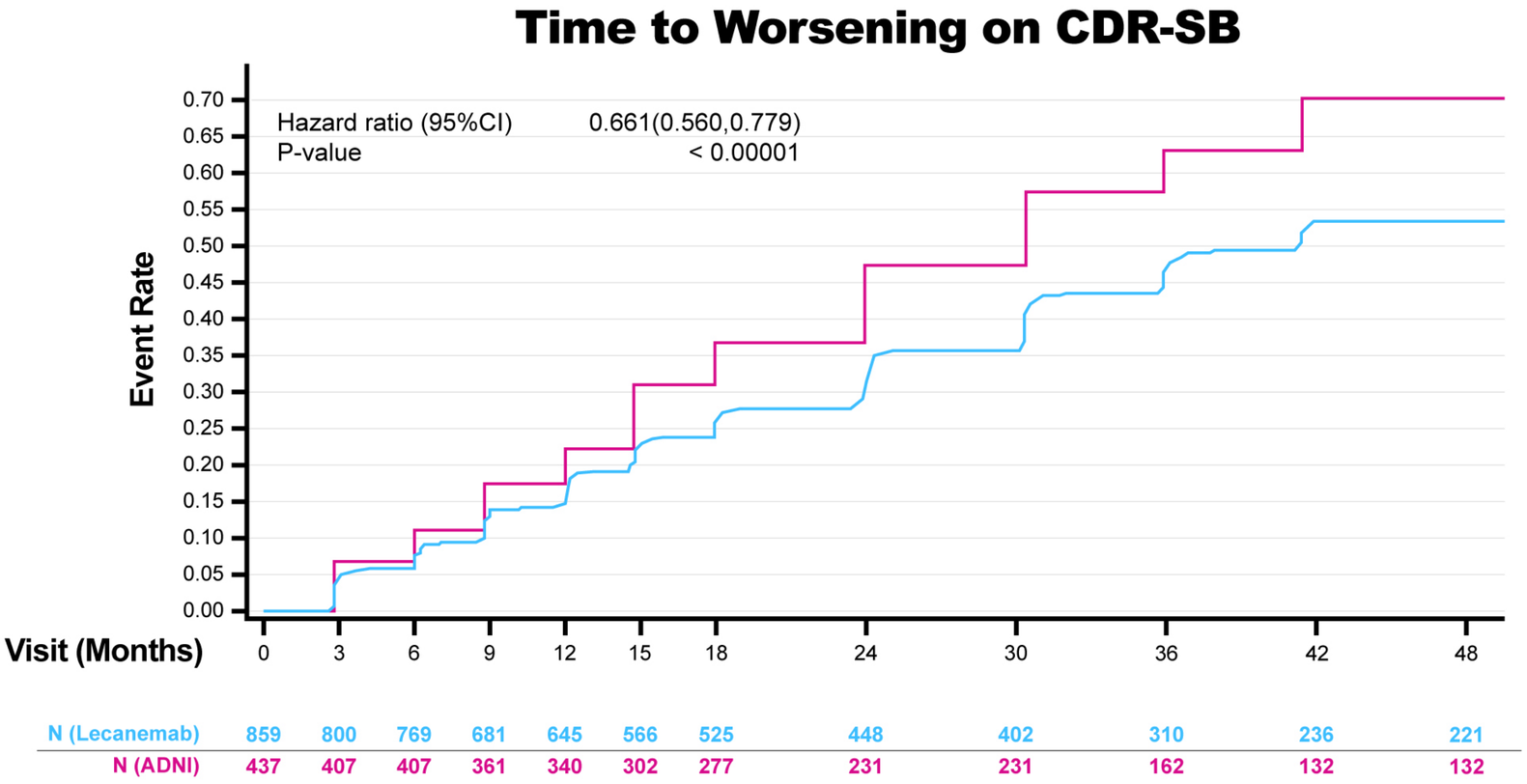
ADNI, Alzheimer's Disease Neuroimaging Initiative. CDR-SB, Clinical Dementia Rating-sum of boxes. SE, standard error.

Lecanemab Meaningfully Delayed Progression to Next Disease Stage Through 48 Months

- **Lecanemab reduces relative risk of progression to next stage of disease by 34% (vs ADNI)**



- **Proportion of patients that progress to next disease stage:**
  - **ADNI: 70.1%**
  - **Lecanemab: 53.3%**

CDR-SB Range	Staging Category
0	Normal
0.5 – 4.0	Questionable Cognitive Impairment
0.5 – 2.5	Questionable Impairment
3.0 – 4.0	Very Mild Dementia
4.5 – 9.0	Mild Dementia
9.5 – 15.5	Moderate Dementia
16.0 – 18.0	Severe Dementia

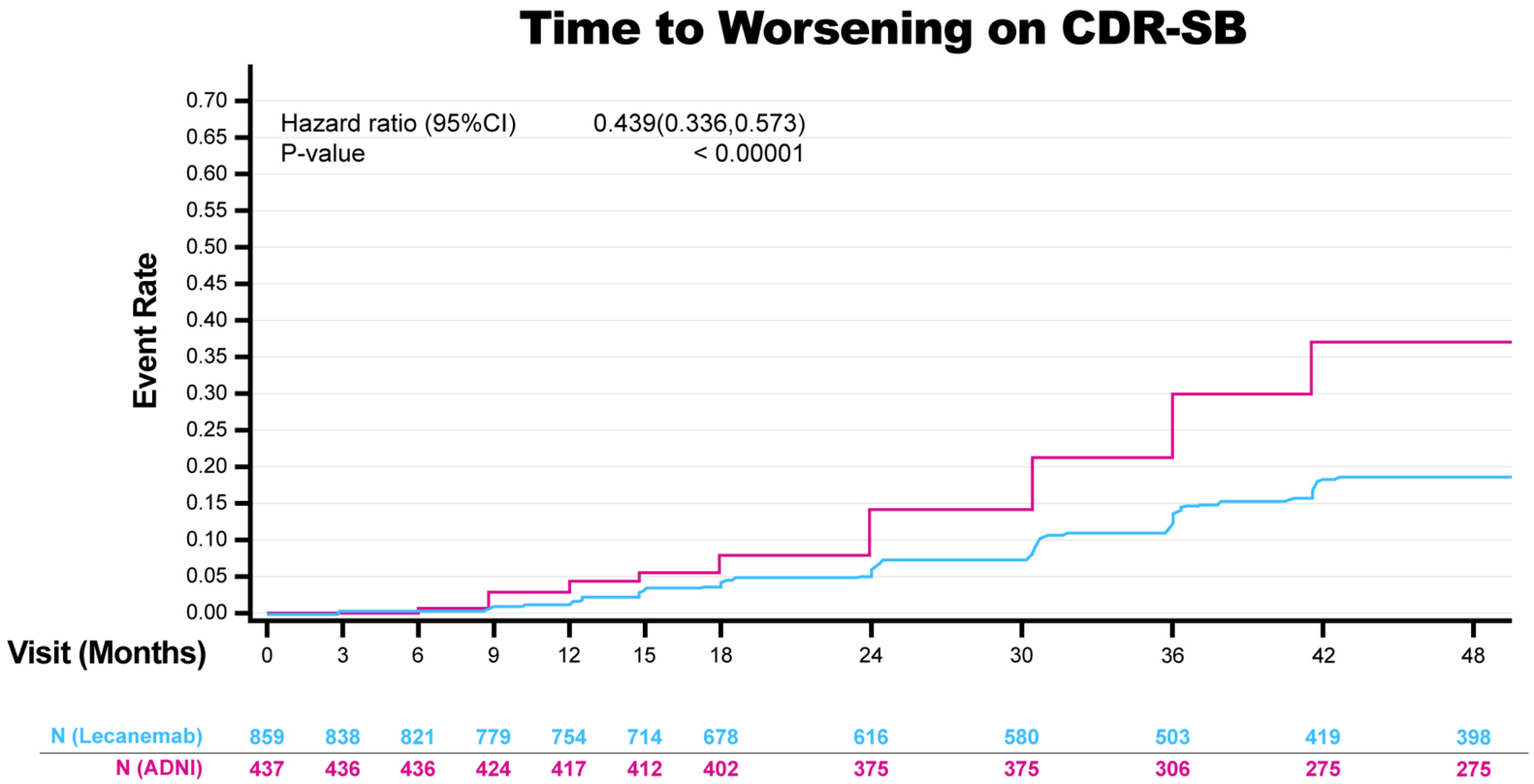
- Progression was defined as CDR-SB Score progressing from MCI (0.5-4) to mild AD dementia (4.5-9) or mild dementia (9.5-15.5) based on dementia staging on CDR-SB (O'Bryant et al., Arch Neurol 2008)
- Given less frequent assessment, since controlled-based imputation was used for missing data in this analysis, CDR-SB (which has greater range) was used rather than global CDR for disease staging

Note: OLE includes those participants on subcutaneous and intravenous formulations.

ADNI, Alzheimer's Disease Neuroimaging Initiative. CDR-SB, Clinical Dementia Rating-sum of boxes.

Lecanemab Meaningfully Delayed Progression to Dementia Stage (moderate or severe) Through 48 Months

- **Lecanemab reduces relative risk of progression to dementia stage of disease by 56% (vs ADNI)**



- **Proportion of patients that progress to dementia stage:**
  - **ADNI: 37.4%**
  - **Lecanemab: 18.6%**

CDR-SB Range	Staging Category
0	Normal
0.5 – 4.0	Questionable Cognitive Impairment
0.5 – 2.5	Questionable Impairment
3.0 – 4.0	Very Mild Dementia
4.5 – 9.0	Mild Dementia
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- Given less frequent assessment, since controlled-based imputation was used for missing data in this analysis, CDR-SB (which has greater range) was used rather than global CDR for disease staging

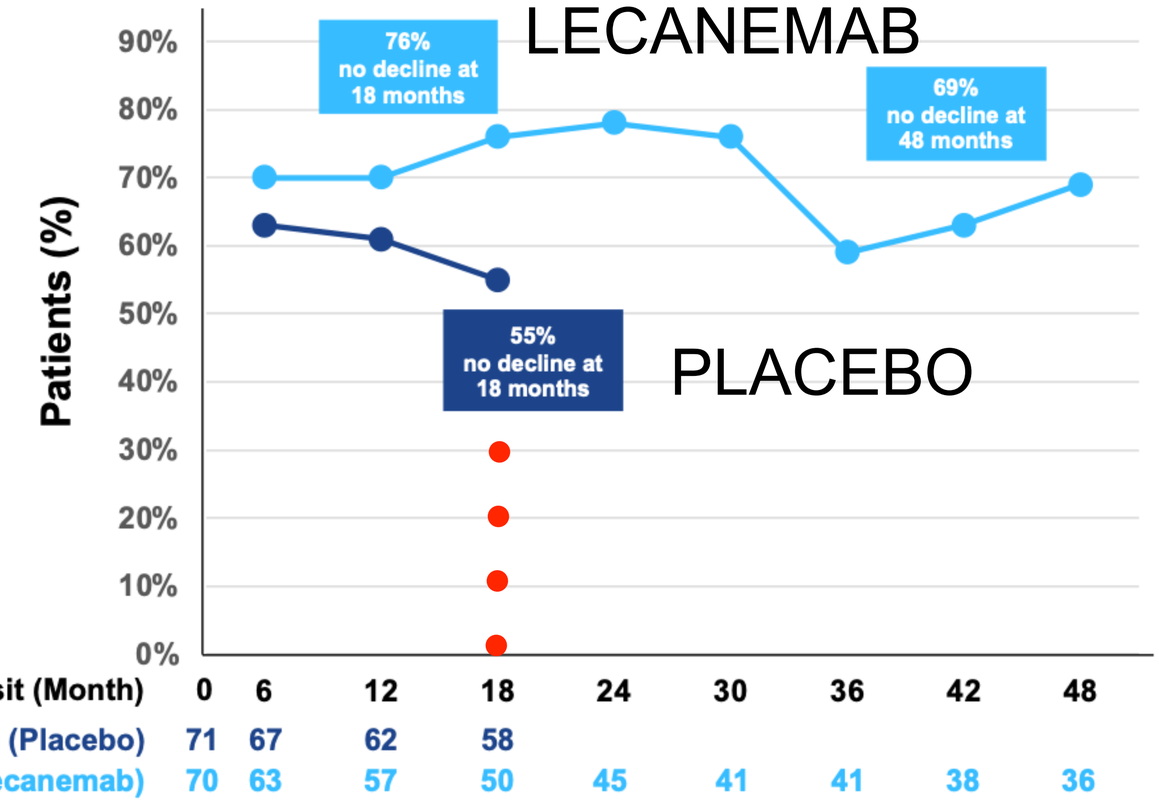
Note: OLE includes those participants on subcutaneous and intravenous formulations.

ADNI, Alzheimer's Disease Neuroimaging Initiative. CDR-SB, Clinical Dementia Rating-sum of boxes.

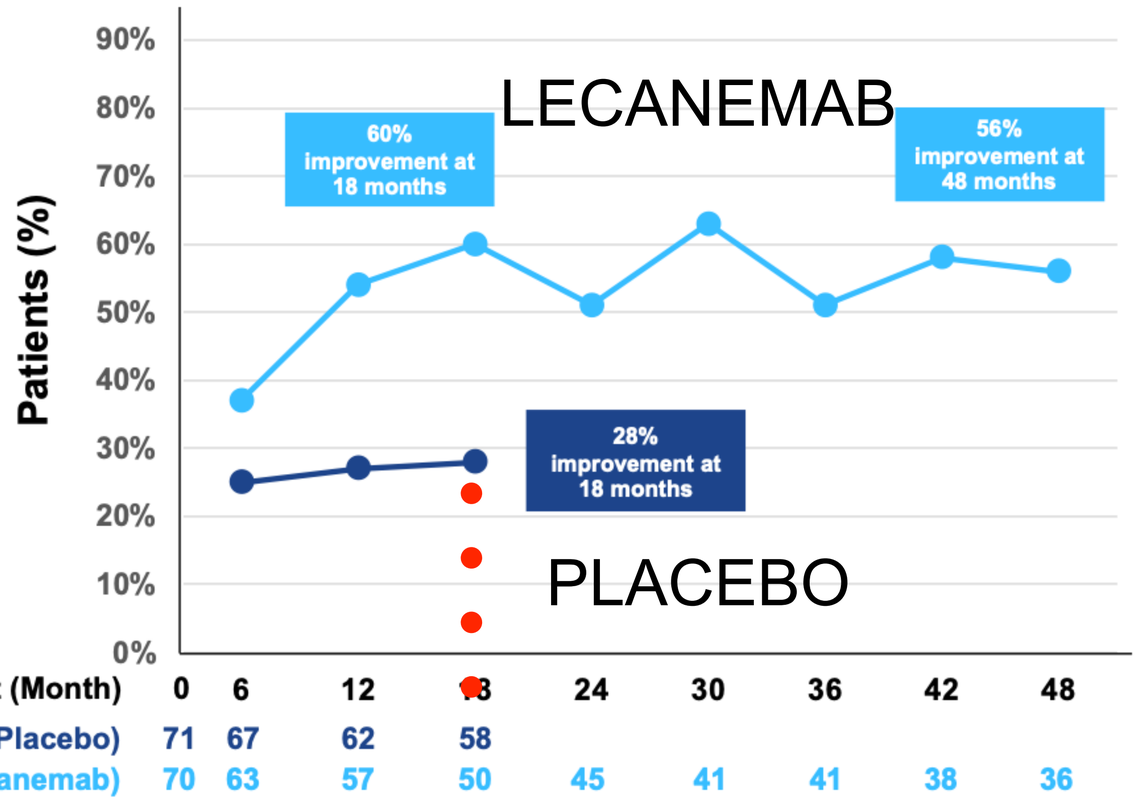
Observed ‘No Decline’ and ‘Improvement’ Rates in Low Tau

Early-Stage Participants Continue to Benefit from Lecanemab Through 48 Months

CDR-SB No Decline - Low Tau Population



CDR-SB Improvement - Low Tau Population



PET TAU IN 342 PAZIENTI

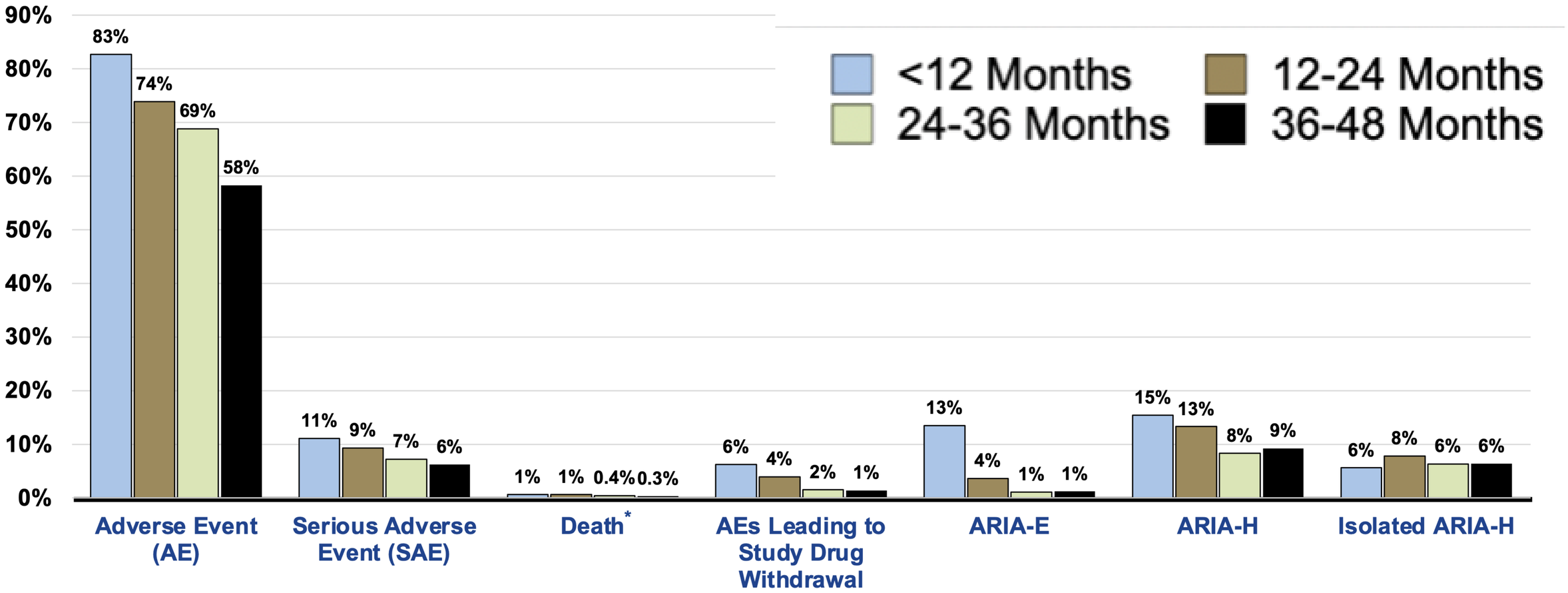
Observed rates for ‘No Decline’ and ‘Improvement’ at 48 months

- ADAS-Cog14: 51% and 51% for lecanemab
- ADCS MCI-ADL: 64% and 58% for lecanemab

Note: OLE includes those participants on subcutaneous and intravenous formulations.

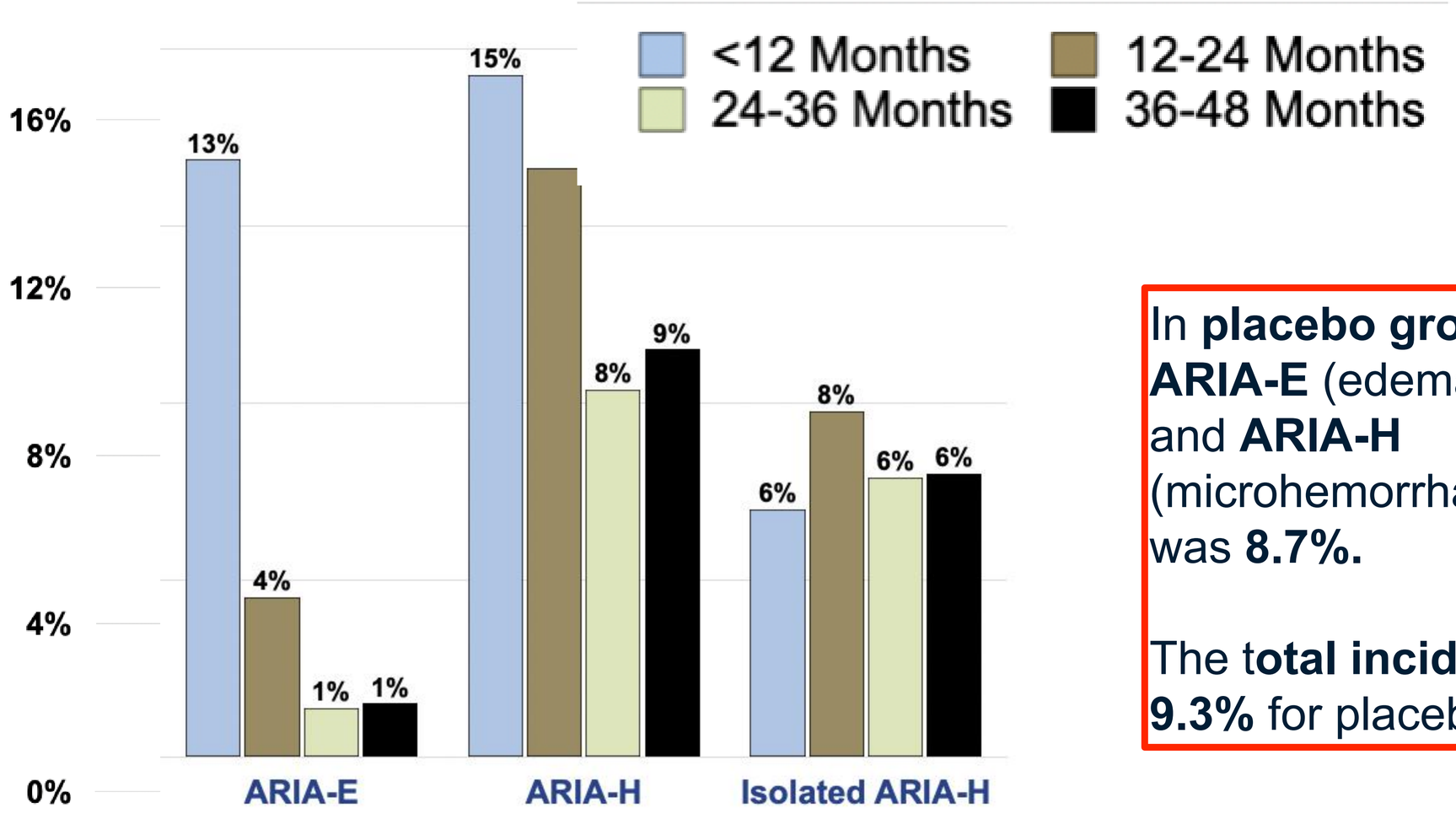
ADAS-Cog14, Alzheimer’s Disease Assessment Scale-Cognitive Subscale. ADCS MCI-ADL, Alzheimer's Disease Cooperative Study-Activities of Daily Living Scale for Mild Cognitive Impairment. CDR-SB, Clinical Dementia Rating-sum of boxes.

Summary of Adverse Events and ARIA by 12 Months Interval



OLE is based on IV datasets (as of 31 Mar 2025)
*Includes all post-treatment events.

Summary of ARIA by 12 Months Interval



In **placebo group** the incidence of **ARIA-E** (edema/effusion) was **1.7%** and **ARIA-H** (microhemorrhages/hemorrhages) was **8.7%.**

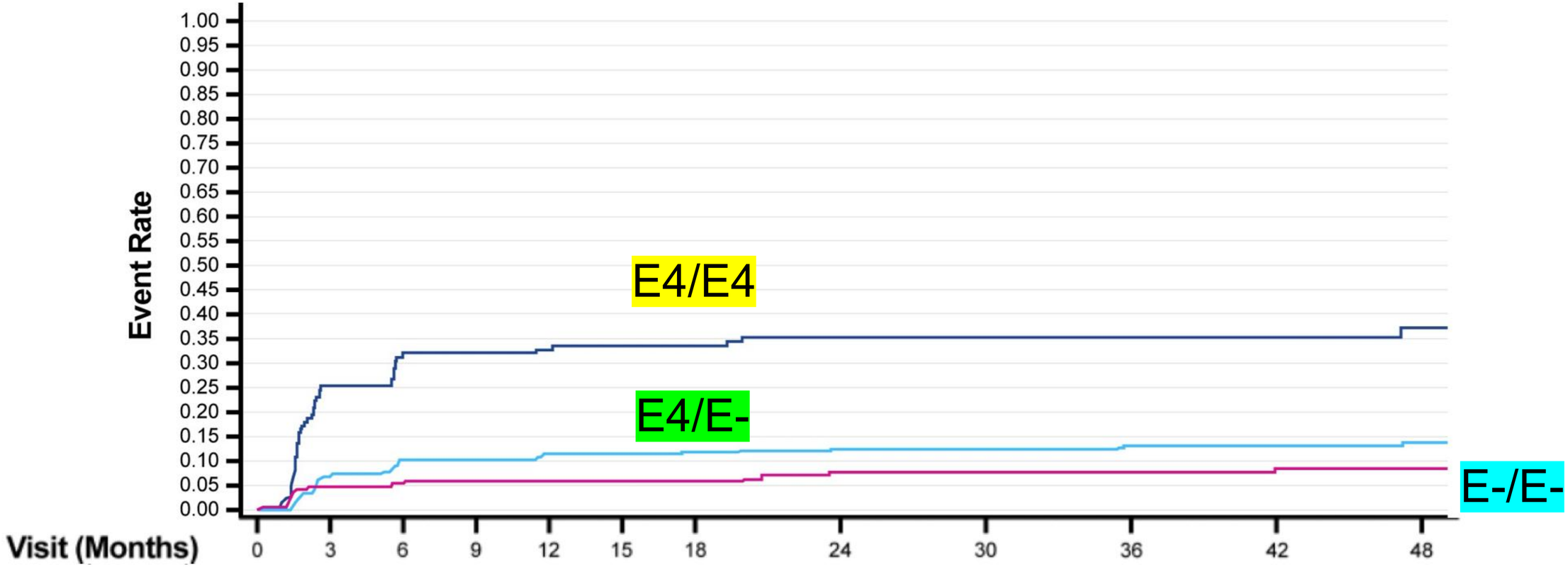
The t**otal incidence** of ARIA was **9.3%** for placebo.

OLE is based on IV datasets (as of 31 Mar 2025)

*Includes all post-treatment events.

Clarity AD OLE: Time to ARIA-E Events and Long-Term Treatment

- Risk of ARIA-E is greatest in first 3 months and generally occurs before 6 months
- Risk period for ARIA-E is first 6 months based on vascular amyloid clearance and increased permeability



N (Homozygous)	141	116	101	100	89	89	89	86	86	85	81	74	73	73	67	66	65	61	58	57	52	48	41	38	37	35	34
N (Heterozygous)	479	446	415	404	390	386	381	369	365	360	348	332	329	320	298	282	270	253	240	228	206	197	181	173	167	163	158
N (Noncarrier)	278	259	250	245	235	225	220	214	208	205	203	192	188	184	170	168	160	155	151	147	135	131	118	110	106	101	93

OLE is based on IV datasets (as of 31 Mar 2025)

AD, Alzheimer's disease. ARIA-E, amyloid related imaging abnormalities with edema. IV, intravenous. OLE, open-label extension.

REAL WORLD

Received: 14 May 2025 | Revised: 2 July 2025 | Accepted: 23 July 2025
DOI: 10.1002/alz.70652

Alzheimer's & Dementia®
THE JOURNAL OF THE ALZHEIMER'S ASSOCIATION

SHORT REPORT

Real-world lecanemab adoption in Japan 1 year after launch: Insights from 311 specialists on infrastructure and reimbursement barriers

Giappone

J Prev Alz Dis 2024;6(11):1549-1562
Published online September 3, 2024, http://dx.doi.org/10.14283/jpad.2024.159

Original Research

Initial Experience with Lecanemab and Lessons Learned in 71 Patients in a Regional Medical Center

L.B.E. Shields[1], H. Hust[1], S.D. Cooley[1], G.E. Cooper[1], R.N. Hart[1], B.C. Dennis[1,2], S.W. Freeman[1], J.F. Cain[1], W.Y. Shang[1], K.M. Wasz[1], A.T. Orr[1], C.B. Shields[1], S.S. Barve[1,3], K.G. Pugh[1]

Drugs & Aging
https://doi.org/10.1007/s40266-025-01261-x

ORIGINAL RESEARCH ARTICLE

Check for updates

Initial Real-World Evidence for Lecanemab in the United States

Diana I. Brixner[1] · Chenyue Zhao[2] · Hideki Toyosaki[2] · Feride H. Frech[2] · Michael H. Rosenbloom[3]

Received: 27 September 2025 / Accepted: 6 October 2025

Stati Uniti

Chin Med J (Engl). 2025 Nov 20;138(22):2907-2916. doi: 10.1097/CM9.0000000000003888. Epub 2025 Oct 27.

Safety and effectiveness of lecanemab in Chinese patients with early Alzheimer's disease: Evidence from a multidimensional real-world study

Brain. 2025 Nov 11:awaf427. doi: 10.1093/brain/awaf427. Online ahead of print.

Safety and short-term outcomes of lecanemab for Alzheimer's disease in China: a multicentre study

CINA

Real-World Use of Lecanemab in Patients With Early Alzheimer's Disease in the United States: A Case Series Review

ORAL PRESENTATION – AAIC 2025 TORONTO

By David Weisman, MD
Abington Neurological Associates, Abington, PA, US

Study Summary

Real-World Use of Lecanemab in Patients With Early Alzheimer's Disease in the United States: A Case Series Review

SLIDE ADAPTED FROM THE ORAL PRESENTATION – AAIC 2025 TORONTO

By David Weisman, MD

Abington Neurological Associates, Abington, PA, US

Retrospective, multicenter, real-world study in the US

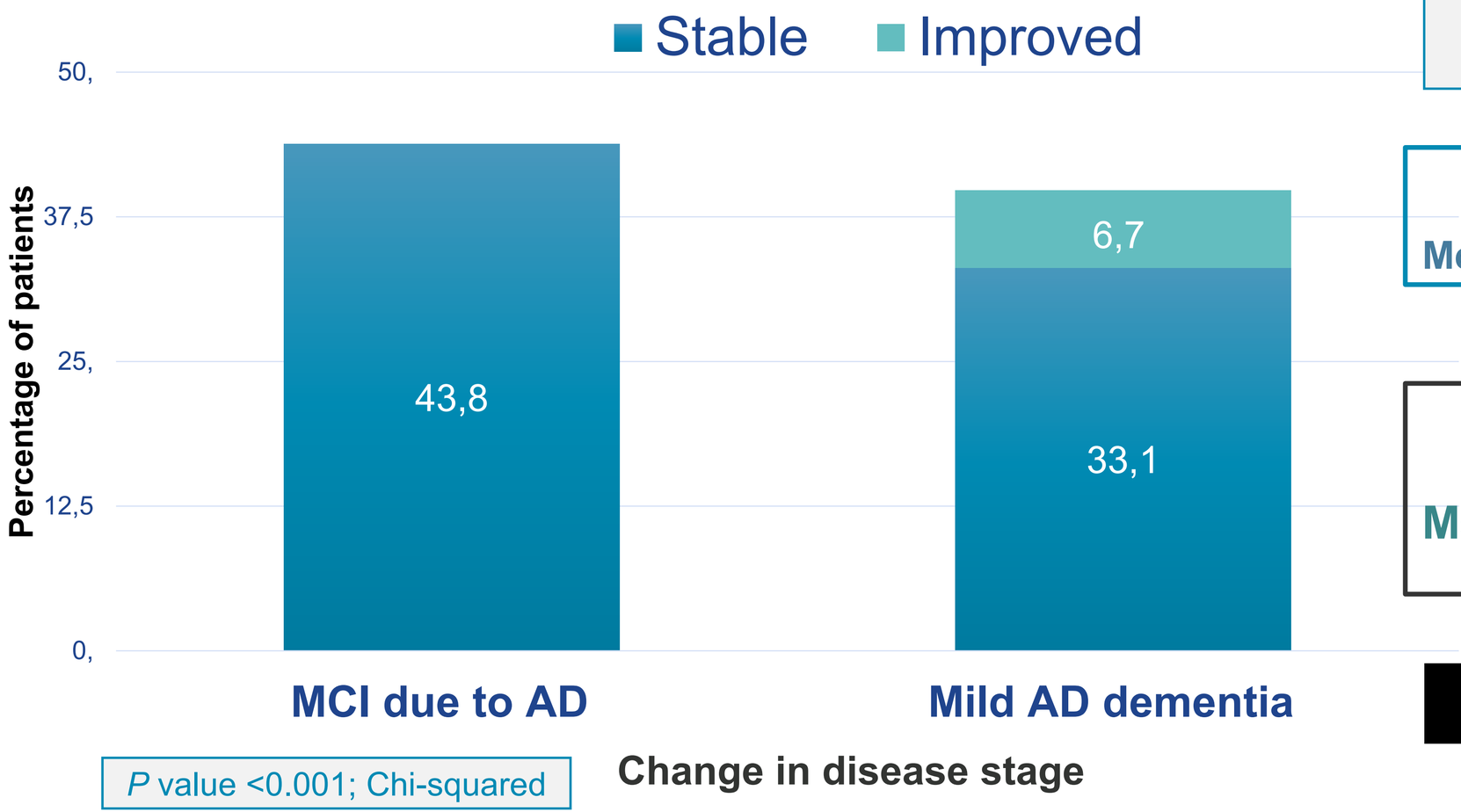
Primary endpoint	To describe real-world utilization patterns of lecanemab in the US
Secondary endpoint	To describe lecanemab real-world implementation learnings
Exploratory endpoint	To describe clinical outcomes of patients treated with 7 or more lecanemab infusions

15 HCPs

March to August 2025

HCP, healthcare provider.

~84% of Patients on Lecanemab Either Remained Stable* or Clinically Improved† Based on Disease Stage at the time of Chart Extraction



P value <0.001; Chi-squared

The **mean ± SD age** of patients was **74.2 ± 6.6 years**

76.9% were stable*
Mean (SD) number of doses: 19.8 (11.4)

6.7% improved† from mild AD dementia to MCI due to AD
Mean (SD) number of doses: 20.2 (11.7)

n=177

* Stable is defined as a patient staying in the same disease stage (MCI due to AD or mild AD dementia) from baseline throughout the course of lecanemab treatment. Disease stage is based on clinical judgment; † Improvement is defined as a patient going from mild AD dementia at baseline to MCI due to AD over the course of lecanemab treatment. Disease stage is based on clinical judgment.
AD, Alzheimer's disease; MCI, mild cognitive impairment; SD, standard deviation.

Majority of Patients Did Not Progress to the Next Disease Stage Regardless of Sex

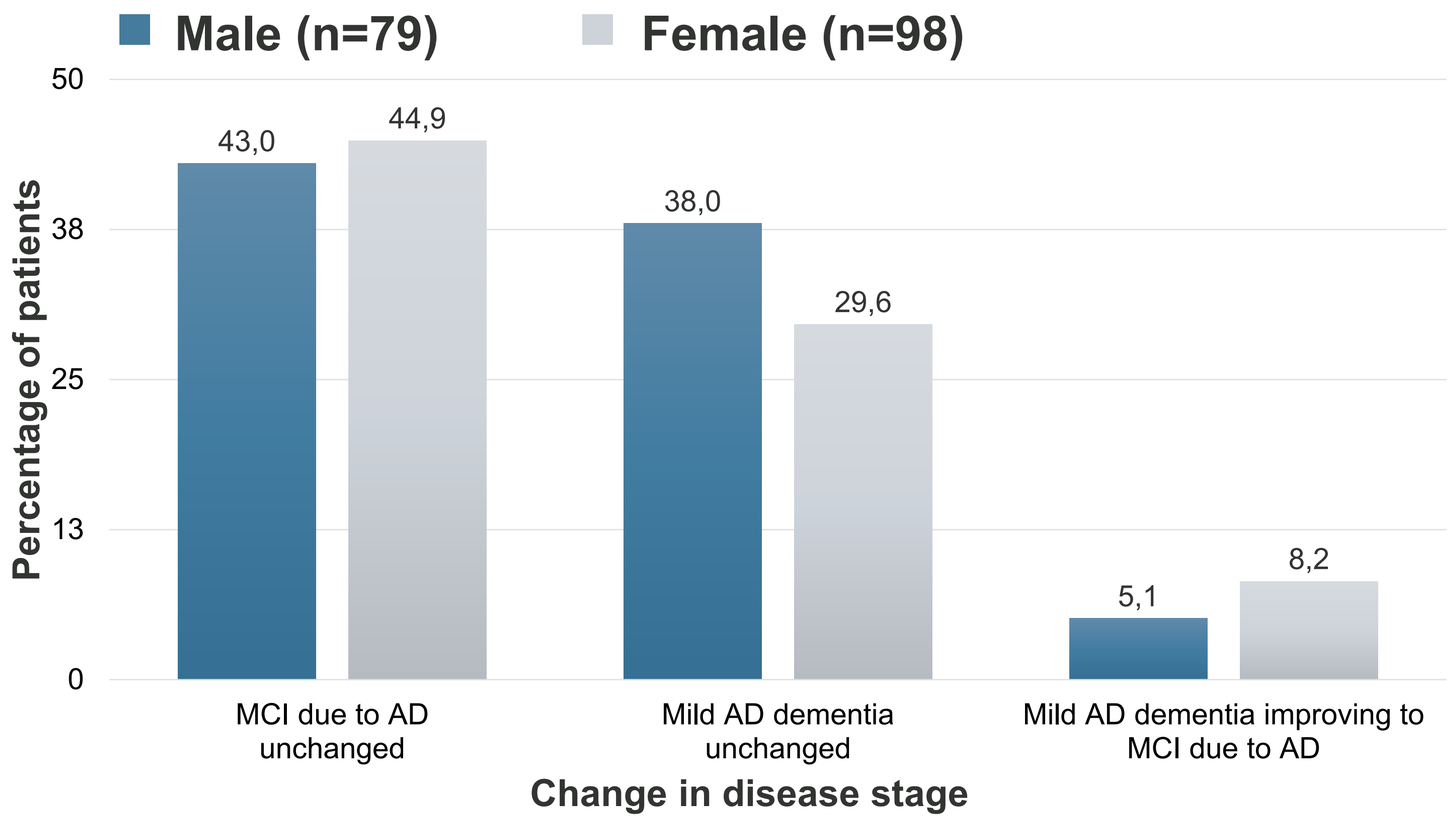
Mean (SD) time on lecanemab

Male: 296.4 (177.9) days

Female: 328.9 (174.2) days

Sex	% Stable* (Mean [SD] number of doses)	% Improved† (Mean [SD] number of doses)
Male	81.0%	5.1%
	17.7 (10.6)	16.2 (6.7)
Female	74.5%	8.2%
	21.7 (11.9)	22.8 (14.1)

P=0.801; Chi-squared test



* Stable is defined as a patient staying in the same disease stage (MCI due to AD or mild AD dementia) from baseline throughout the course of lecanemab treatment. Disease stage is based on clinical judgment; † Improvement is defined as a patient going from mild AD dementia at baseline to MCI due to AD over the course of lecanemab treatment. Disease stage is based on clinical judgment.
AD, Alzheimer's disease; MCI, mild cognitive impairment; SD, standard deviation.

Safety Data: Incidence of ARIA and Adverse Events Leading to Discontinuation.

87% of Patients Remained on Lecanemab

	RWE study N=178	Clarity AD[1,2] Lecanemab treatment N=898
	n (%)	n (%)
ARIA (all)*	**23 (12.9)**	**191 (21.3)**
ARIA-E*	14 (7.9)	113 (12.6)
ARIA-H*	11 (6.2)	152 (17.3)
Infusion-related reactions	5 (2.8)	237 (26.4)
Patients discontinuing due to ARIA	5 (2.8)	-

	RWE study N=178	Clarity AD[1,2] Lecanemab treatment N=898
	n (%)	n (%)
ARIA (all)*	23 (12.9)	191 (21.3)
ARIA-E*	14 (7.9)	113 (12.6)
Mild	11 (6.2)	37 (4.1)
Moderate	3 (1.7)	66 (7.3)
Severe	0 (0)	9 (1.0)
ARIA-H*	11 (6.2)	152 (17.3)
Mild	10 (5.6)	117 (13.0)
Moderate	1 (0.6)	27 (3.0)
Severe	0 (0)	32 (3.6)

- **No macrohemorrhages/ intracerebral hemorrhages were reported**
- **No deaths were reported**

* Concurrent ARIA-E and ARIA-H: n=2; † All infusion-related reactions occurred during the first lecanemab dose; ‡ One patient discontinued due to concurrent ARIA-E and ARIA-H; this patient is included in the counts for both ARIA-E and ARIA-H; § Other adverse events include lacunar infarct (n=1), acute confusion event (n=1), PMR (n=1).

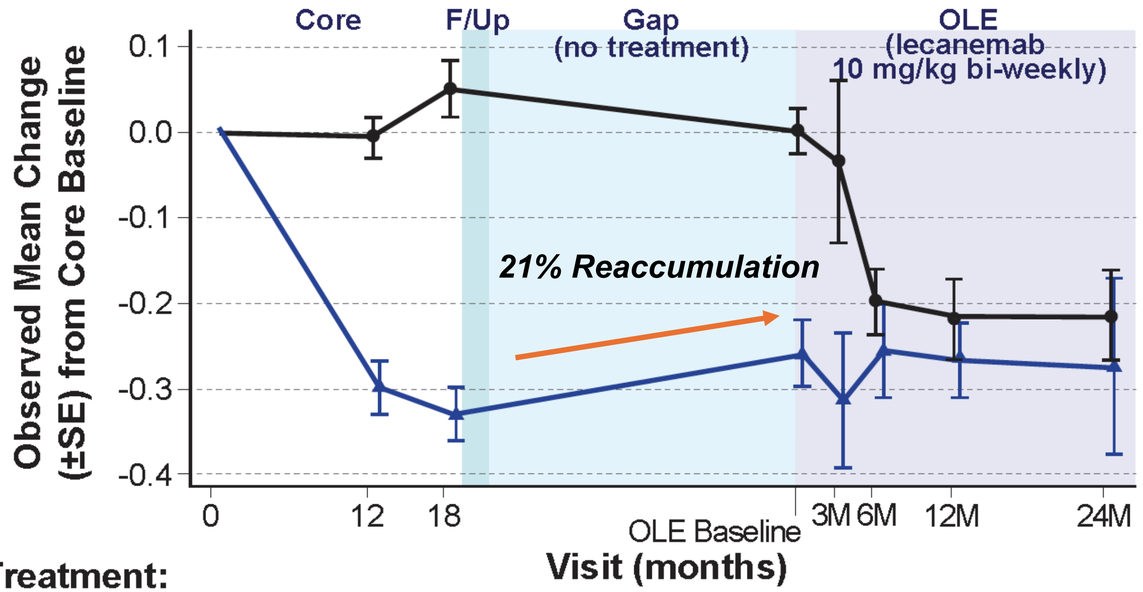
AD, Alzheimer's disease; ARIA, amyloid-related imaging abnormalities; ARIA-E, amyloid-related imaging abnormalities—edema/effusion;
ARIA-H, amyloid-related imaging abnormalities—hemosiderin/microhemorrhage; FDA, Food and Drug Administration; PMR, polymyalgia rheumatica; RWE, real-world evidence.

1. van Dyck CH, et al. N Engl J Med. 2023;388(1):9–21; 2. LEQEMBI (lecanemab-irmb). US Prescribing Information. Eisai Inc. https://www.leqembi.com/en/-/media/Files/Leqembi/Prescribing-Information.pdf (Accessed July 2025).

Interrompere il trattamento ?

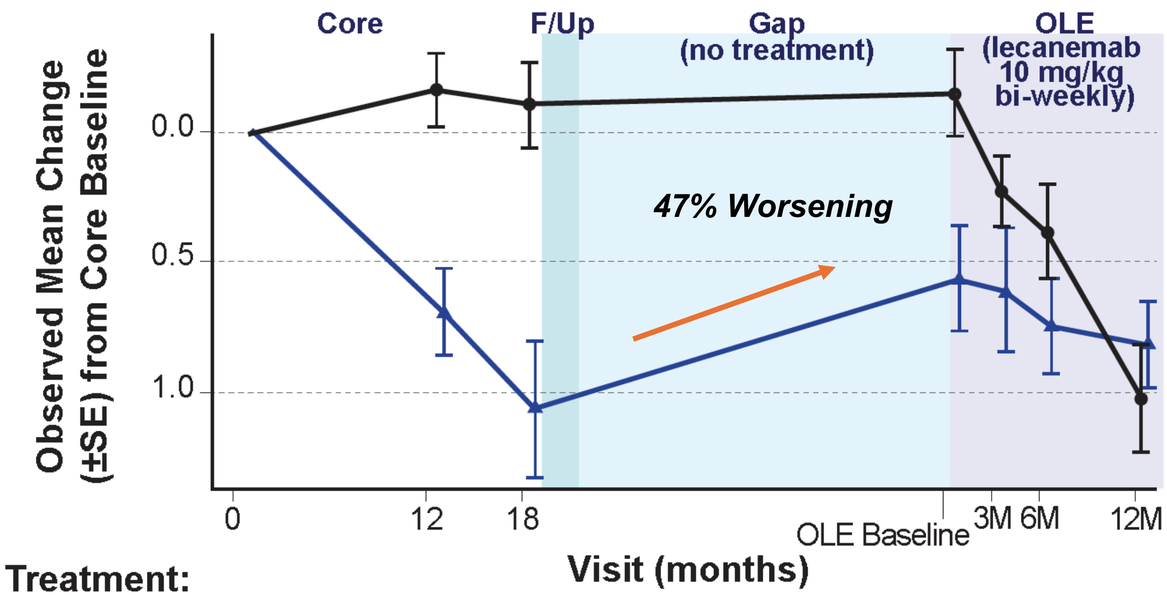
Interrupting Treatment is Associated with Variable Rates of Reaccumulation of AD Biomarkers: Amyloid PET & Ab42/40 Ratio

Amyloid PET



Core Treatment:							
Placebo:	18	14	13	16	3	1	9	6
10 bi-Weekly:	17	13	12	12	9	1	12	3

Aβ42/40 Ratio



Core Treatment:							
Placebo:	32	32	30	32	31	31	31
10 bi-Weekly:	25	23	24	25	23	24	25

Long-term therapy continues to improve biomarkers further, and avoid the reaccumulation of biomarkers when anti-amyloid treatment is stopped

McDade et al. Alzheimers Res Ther. 2022;14:191.

Aβ42/40 = ratio of Aβ42 to Aβ40. OLE = open-label extension. PET= positron emission tomography.

Conclusioni

- La Malattia di Alzheimer è una malattia cronico/subacuta letale e la sua fisiopatologia persiste anche dopo la rimozione delle placche amiloidi.
- Lecanemab riduce sia le protofibrille Aβ solubili che le placche Aβ insolubili con un doppio meccanismo d'azione.
- I pazienti trattati con lecanemab hanno continuato a mostrare benefici clinici fino a 48 mesi.
- Non sono emersi nuovi eventi avversi clinicamente significativi con il trattamento a lungo termine.
- I risultati nella fase iniziale (ad es. tau bassa) supportano la stabilità clinica o il miglioramento con l'inizio precoce del trattamento con lecanemab.
- Durante la sospensione di lecanemab, è stato osservato un riaccumulo dei biomarcatori.
- Lecanemab prolunga il tempo trascorso nella fase iniziale della malattia con un aumento dell'efficacia del trattamento nel tempo.