Understanding Payer Evidentiary Needs for Alzheimer's Disease Monoclonal Antibody Treatments

July 14, 2021

Welcome and Overview

Mark McClellan

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- Marketing and strategic plans, market or competitive evaluations
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- Status of negotiations with present or potential customers, suppliers, payers or healthcare providers
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Agenda

10:00	Welcome and Framing the Issues
10:20	Session I: Current Landscape of mAb Development for AD and Potential Implications for Coverage and Access
12:10	Lunch
12:40	Session II: Opportunities for Generating Clinical Evidence for Promising AD mAb Treatments
2:10	Break
2:30	Session III: Looking Forward
3:45	Closing Remarks

Introductions

Framing the Issues

Mark McClellan

Duke-Margolis Center for Health Policy

Background

- Drugs targeting beta amyloid (e.g. aducanumab), which many scientists believe is the primary cause of AD, have been in development and reported some promising results
- Current clinical trials focus on patients with earlier stages of AD, and labeling for aducanumab notes that treatment should be initiated in patients with mild cognitive impairment or mild dementia stage of disease
- Different stakeholders have differing perspectives on how AD treatments should be valued and priced: ICER's pricing suggestion vs. patient advocates' assessments
- Additional evidence on amyloid-busting therapeutics is critical for coverage, payment, and appropriate use decisions

Remaining Evidentiary Questions

- What is the treatment impact in each stage of the disease and across different types of patients, especially those who have progressed beyond the early stages of AD?
- How can endpoint validation, including surrogate endpoints, potentially be expedited through assessing evidence being developed in the other drug trials or post-market studies?
- How certain is the evidence on how CDR-SB progression translates into additional months of independent living or other key aspects of quality of life?
- Should treatment be continued indefinitely, or is a fixed period adequate (e.g., a year), or should duration be based on resolution of plaques?
  - Are there any long-term safety issues associated with chronic use of the mAb therapies?
- What do appropriate care models for these drugs look like?
  - Will they require monitoring by specialized memory clinics, which have only limited availability today?
  - Will blood tests currently in development reduce the cost and burden of determining treatment eligibility and monitoring disease progression?

Potential Next Steps for CMS

- CMS is a primary payer for the AD mAb population
- CMS's coverage decision is critical for both FFS Medicare and MA, and will also influence decisions by commercial payers and potentially state Medicaid plans
- CMS opened a NCD process for aducanumab and the broader mAb treatment class on July 12
- In making a NCD, CMS could :
  - Elect to deny coverage for the treatment (unprecedented for Part B drugs)
  - Limit coverage to certain populations
  - Make coverage determinations contingent upon the collection of additional data with the goal of developing more evidence on the drug's safety and efficacy for beneficiaries (CED)
- There is a set of issues associated with a CED approach, incl. designing randomized trials and/or obtaining convincing evidence from registries

Potential Next Steps for Other Payers

- Private payer actions will be influenced by CMS actions and will likely use the eligibility criteria from the clinical trials, reflecting current FDA labeling, while monitoring results from the other studies underway to further adjust coverage
- Payers are likely to focus on evidence demonstrating:
  - The relationship between the treatment's surrogate endpoints as shown in the clinical trials to actual cognitive outcome endpoints
  - That these outcomes are associated with the reduction of short and long-term costs of care, a better quality of life and decreased caregiver burden and;
  - More efficient care models
- Payers are also focused on appropriate payment amounts based on assessments of treatment value based on current and emerging evidence

Topics to Consider Today

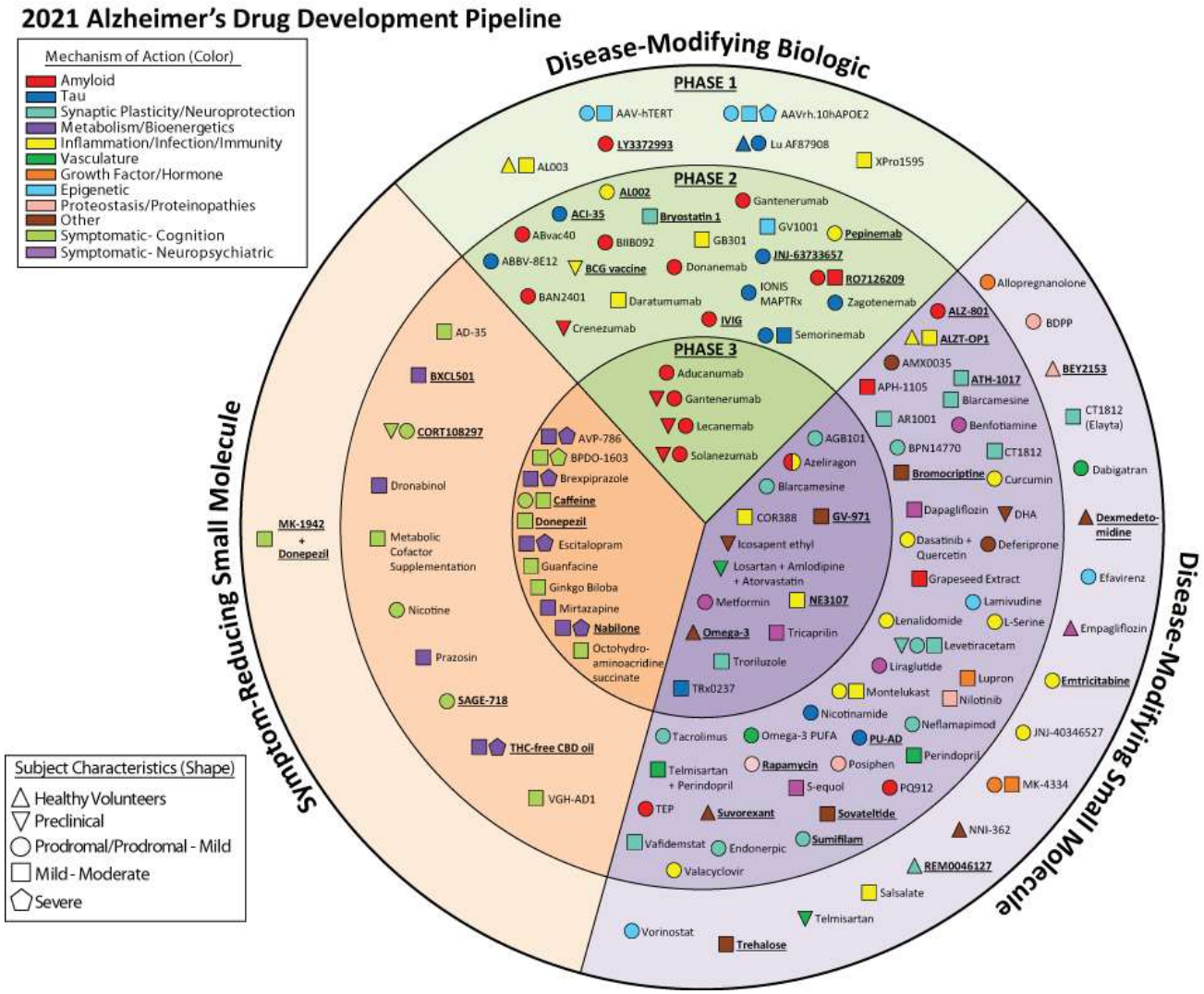
- Key issues that payers are facing in making coverage and payment decisions involving the AD mAb drug class;
- Payers' activities to review the evidence relevant to these decisions, and their initial assessment of the evidence as it is available;
- Top areas where further evidence is needed to address stakeholder needs, and whether studies in progress are likely to be adequate to address these topics; and
- Opportunities for expanding the evidence base on mAb treatments for AD to incorporate payer perspectives.

Session I: Current Landscape of mAb Development for AD and Potential Implications for Coverage and Access

Jeff Cummings

University of Nevada Las Vegas

2021 Alzheimer's Pipeline



126 agents in Clinical Trials

- Phase 3 – 28
- Phase 2 – 74
- Phase 1 – 24
- DMTs – 104 (83% of agents)
- Biologics – 31
- Small molecule DMTs – 73
- Repurposed – 50 (40% of agents)
- Cog enhancer – 13
- NPS treatment – 9

Cummings J et al. Alz&Dem: TRCI 2021; 7(1):e12179. doi: 10.1002/trc2.12179.

Biomarker role in trial[a]	N of trials (%)	
	Phase 3 DMTs	Phase 2 DMTs
Biomarker as an outcome measure[a]		
CSF amyloid	15 (25%)	10 (48%)
CSF tau	17 (28%)	9 (43%)
FDG-PET	7 (11%)	1 (5%)
vMRI	8 (13%)	8 (38%)
Plasma amyloid	7 (11%)	2 (10%)
Plasma tau	2 (3%)	1 (5%)
Amyloid PET	5 (8%)	7 (33%)
Tau PET	4 (7%)	3 (14%)
Biomarker as an entry criterion[a]		
Amyloid PET	4 (17%)	11 (14%)
CSF amyloid	1 (4%)	9 (12%)
Amyloid PET or CSF amyloid	6 (25%)	11 (14%)
Tau PET	0	2 (3%)
CSF amyloid or CSF tau	0	2 (3%)
Amyloid PET or CSF tau	0	1 (1%)

Biomarkers used as outcome measures in current Phase 2 and Phase 3 DMT trials

Cummings J et al. Alz&Dem: TRCI 2021; 7(1):e12179. doi: 10.1002/trc2.12179.

Mark Mintun

Eli Lilly and Company

Adapted from presentation at ADPD March 2021

Donanemab slows progression of early symptomatic Alzheimer's disease in Phase 2 proof of concept trial

Mark A. Mintun, Albert C. Lo, Cynthia Duggan Evans, Paul A. Ardayfio, Scott W. Andersen, Sergey Shcherbinin, Jeffrey L. Dage, Ming Lu, Emily C. Collins, John R. Sims, Miroslaw Brys, Daniel M. Skovronsky

Mark A. Mintun
Presenter Disclosure Information

Vice-President of Alzheimer's Disease Development, Eli Lilly and Company, Indianapolis, IN, USA
President, Avid Radiopharmaceuticals, Inc. Philadelphia, PA, USA

Eli Lilly and Company, Indianapolis, IN, USA

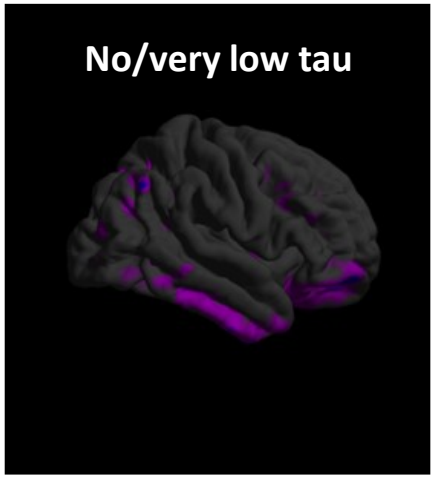
TRAILBLAZER-ALZ*

- Phase 2 registration quality trial to evaluate safety, tolerability and efficacy of donanemab
- Multi-center (56 sites across the United States and Canada), randomized, double-blind, placebo-controlled
- Study population
  - Women and men, 60-85 years of age, with early symptomatic AD (combination of prodromal AD [mild cognitive impairment-AD] and mild AD dementia)
  - Screening procedures included Mini–Mental State Examination (MMSE), flortaucipir F18 Positron Emission Tomography (PET) scan, florbetapir F18 PET scan, and magnetic resonance imaging
- Pre-specified statistical analysis plan and independent data-monitoring committee
- Unique features
  - Tau threshold screening
  - Combination arm with donanemab and BACE inhibitor (discontinued with 15 patients enrolled)
  - Short titration phase to full dose aiming to achieve rapid amyloid plaque removal

*NCT03367403

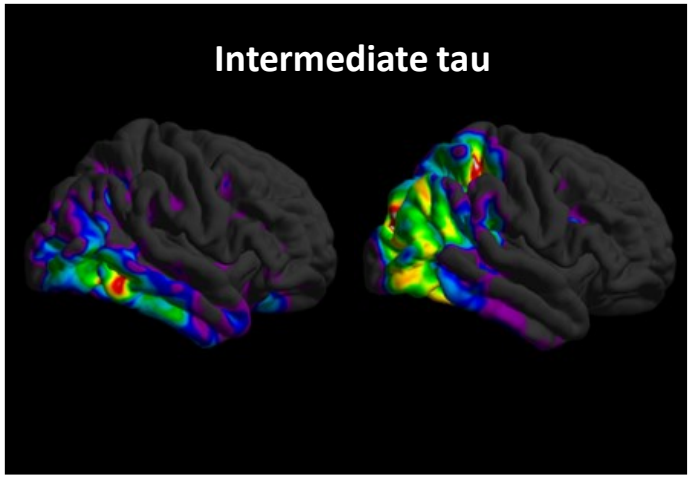
First study to screen and enroll patients based on their tau pathology

Removes those hypothesized as unlikely to have significant decline in 18 months



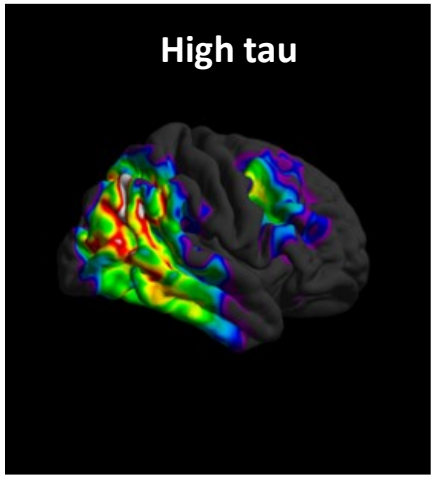
EXCLUDED

Whole brain Tau SUVr < 1.10*



TAU PET INCLUSION WINDOW

Removes those hypothesized as too advanced to be slowed by anti-amyloid therapy

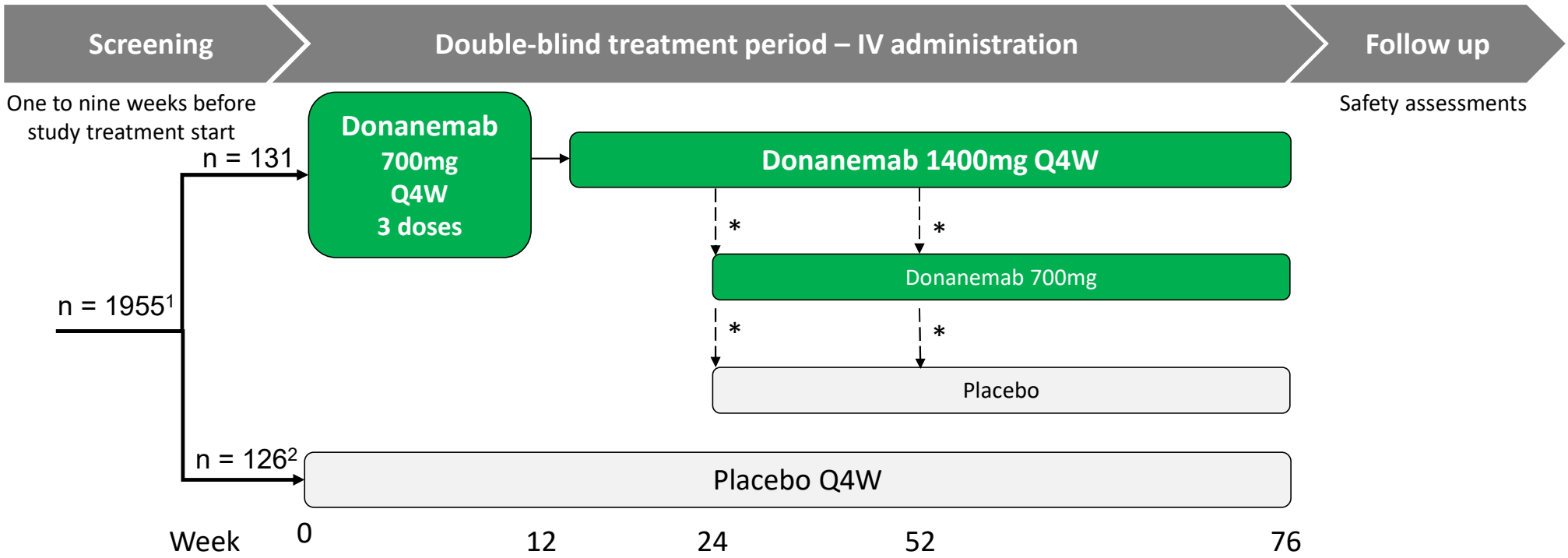


EXCLUDED

Whole brain Tau SUVr > 1.46

SUVr = Standardized Uptake Value ratio. *Visual interpretation also done and took precedent when highly discordant.

Study designed to achieve amyloid clearance and then stop dosing



*At 6-month and 12-month florbetapir PET scans, dosing decision to continue 1400mg Q4W or reduce to 700mg Q4W if amyloid was 11≤ CL < 25 or switched to placebo if it was <11 CL at any one measure or 11≤ CL< 25 for two consecutive scans

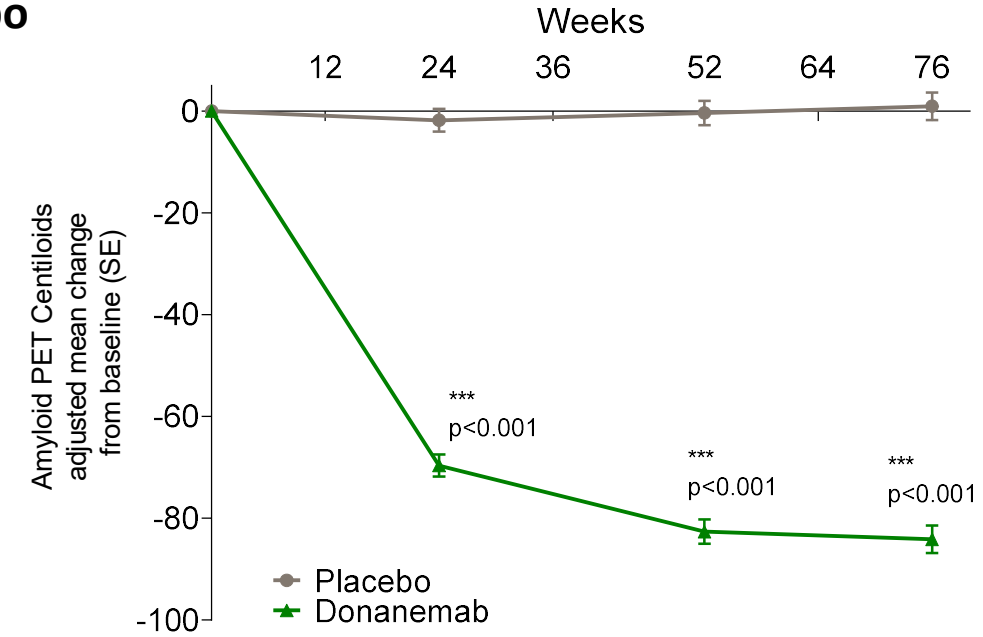
[1]1683 patients excluded due to: screen fail (1563), withdrawal by patient (96), caregiver circumstance (6), and other (18); 15 patients were randomized to discontinued combo.

[2]One patient was randomized to placebo but discontinued the study before receiving an infusion

CL = Centiloids; IV = intravenous; n = number of patients; PET = Positron Emission Tomography; Q4W = every 4 weeks

Secondary outcomes: amyloid lowering

Treatment with donanemab reduced amyloid plaque by 85 Centiloids at 76 weeks compared with placebo



Placebo n=112 Donanemab n=121	111 115	91 92	91 90
Donanemab 'amyloid negative' <24.1 CL, n (%)	46 (40%)	55 (60%)	61 (68%)

	LS Mean Change Δ (SE) Donanemab vs. Placebo
W24	-67.83 (3.16)
W52	-82.30 (3.41)
W76	-85.06 (3.87)

40% of donanemab-treated participants reached amyloid negative levels by 24 weeks

CI = Confidence Interval ; CL = Centiloids; LS = Least Squares; n = number of patients; SE = Standard Error; W = weeks
Amyloid negative defined as < 24.1 CL

Safety profile

ARIA-E the most common treatment emergent adverse event

Adverse events (AE)

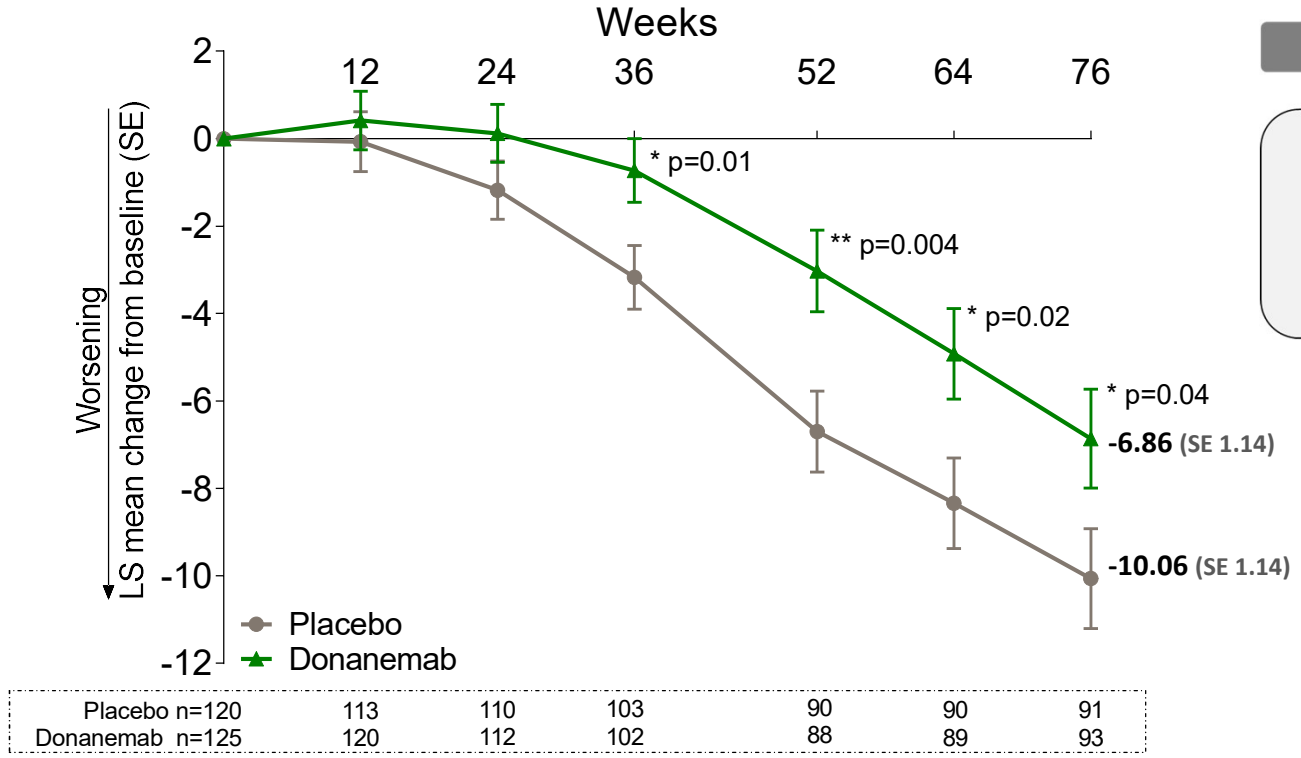
Participants, n (%)	Placebo (n=125)	Donanemab (n=131)	p-value
Deaths	2 (1.6%)	1 (0.8%)	0.615
Serious AE	22 (17.6%)	23 (17.6%)	>0.999
Treatment discontinuations due to AE*	**9 (7.2%)**	**40 (30.5%)**	**<0.001**
Study discontinuations due to AE*	**6 (4.8%)**	**20 (15.3%)**	**0.007**
Treatment-Emergent AE	113 (90.4%)	119 (90.8%)	>0.999

ARIA-E = Amyloid-Related Imaging Abnormalities-Edema/Effusions
*Discontinued treatment due to protocol-defined criteria and patient/principal investigator-cited reasons for discontinuation.

Treatment emergent AE ≥5%

Preferred Term, n (%)	Placebo (n=125)	Donanemab (n=131)	p-value
ARIA-E	**1 (0.8%)**	**35 (26.7%)**	**<0.001**
ARIA-E Symptomatic (subset)	1 (0.8%)	8 (6.1%)	
Fall	19 (15.2%)	17 (13.0%)	0.720
Dizziness	15 (12.0%)	11 (8.4%)	0.410
Headache	15 (12.0%)	10 (7.6%)	0.294
Superficial siderosis of central nervous system	**4 (3.2%)**	**18 (13.7%)**	**0.003**
Arthralgia	10 (8.0%)	10 (7.6%)	>0.999
Nausea	**4 (3.2%)**	**14 (10.7%)**	**0.026**
Upper respiratory tract infection	9 (7.2%)	9 (6.9%)	>0.999
Urinary tract infection	5 (4.0%)	13 (9.9%)	0.086
Diarrhea	5 (4.0%)	11 (8.4%)	0.198
ARIA-H	4 (3.2%)	11 (8.4%)	0.109
Cerebral microhaemorrhage	3 (2.4%)	10 (7.6%)	0.085
Infusion Related Reaction	**0**	**10 (7.6%)**	**0.002**
Pneumonia	5 (4.0%)	7 (5.3%)	0.770
Depression	8 (6.4%)	6 (4.6%)	0.590
Contusion	**10 (8.0%)**	**0**	**<0.001**
Vomiting	3 (2.4%)	7 (5.3%)	0.335
Anxiety	2 (1.6%)	7 (5.3%)	0.173

Primary outcome showed treatment with donanemab significantly slowed disease progression by 32% on iADRS at 76 weeks, compared with placebo



Placebo n=120	113	110	103	90	90	91
Donanemab n=125	120	112	102	88	89	93

Primary Outcome: iADRS

Composite measure combining the ADAS-Cog$_{13}$ and the ADCS-iADL, to assess cognition and function, respectively

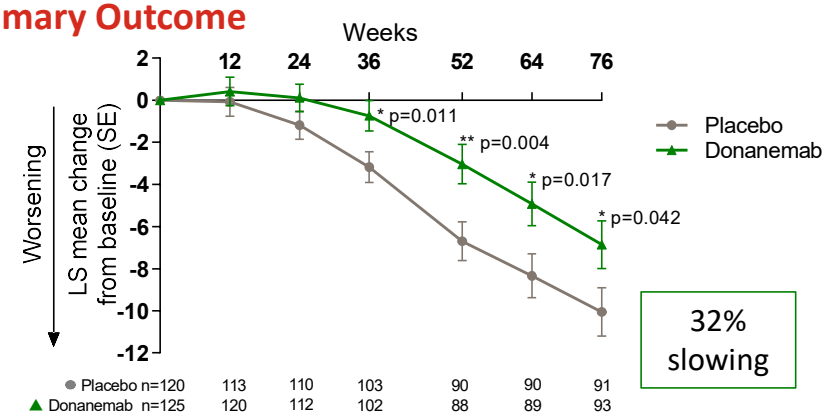
32% slowing by donanemab at 76 weeks

LS mean change from baseline, SE, 95% CI and p-value are derived using MMRM with factors for treatment, visit, treatment-by-visit interaction, pooled investigator, AChEI and/or memantine use at baseline, and covariates for baseline score, age at baseline, and baseline score-by-visit interaction.

MMRM statistical analysis used. iADRS = Integrated Alzheimer's Disease Rating Scale; LS = Least Squares; MMRM = mixed model for repeated measures; n = number of patients; SE = Standard Error; AChEI = acetylcholinesterase inhibitor

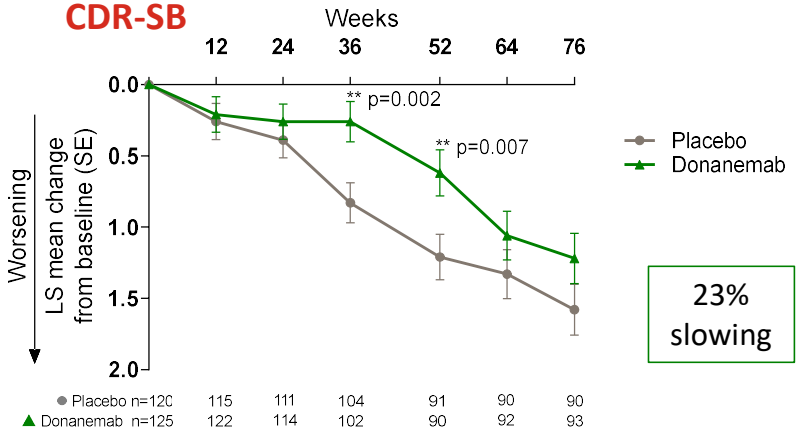
Donanemab consistently slowed cognitive and functional decline on all secondary clinical endpoints at multiple timepoints compared with placebo

iADRS – Primary Outcome



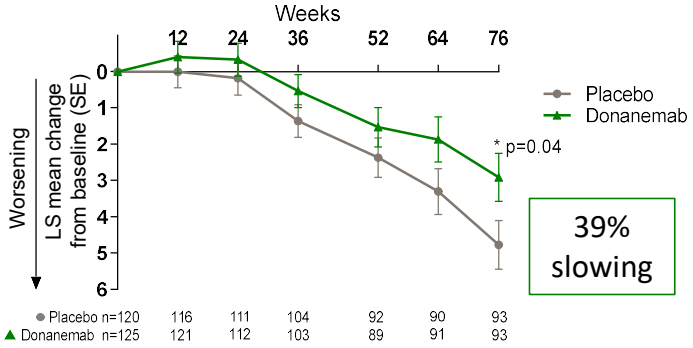
32% slowing

CDR-SB



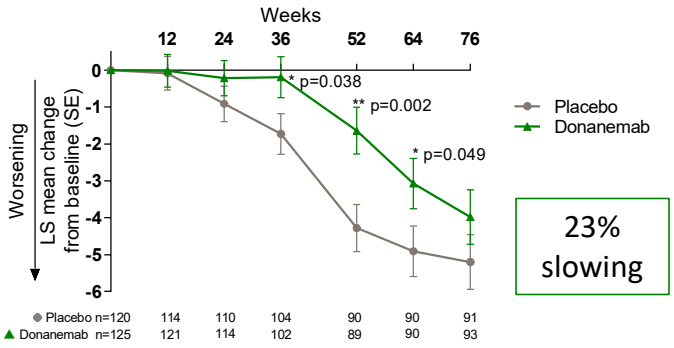
23% slowing

ADAS-Cog13



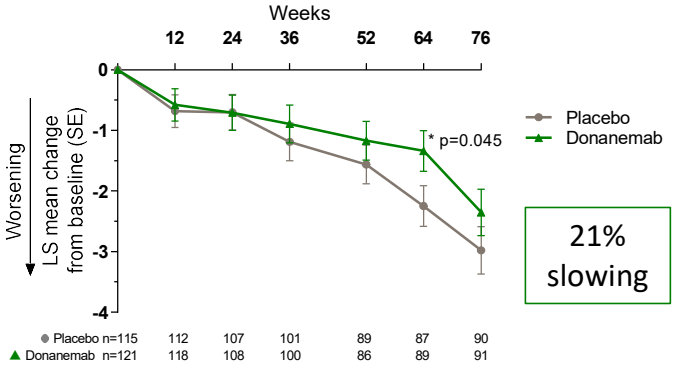
39% slowing

ADCS-iADL



23% slowing

MMSE

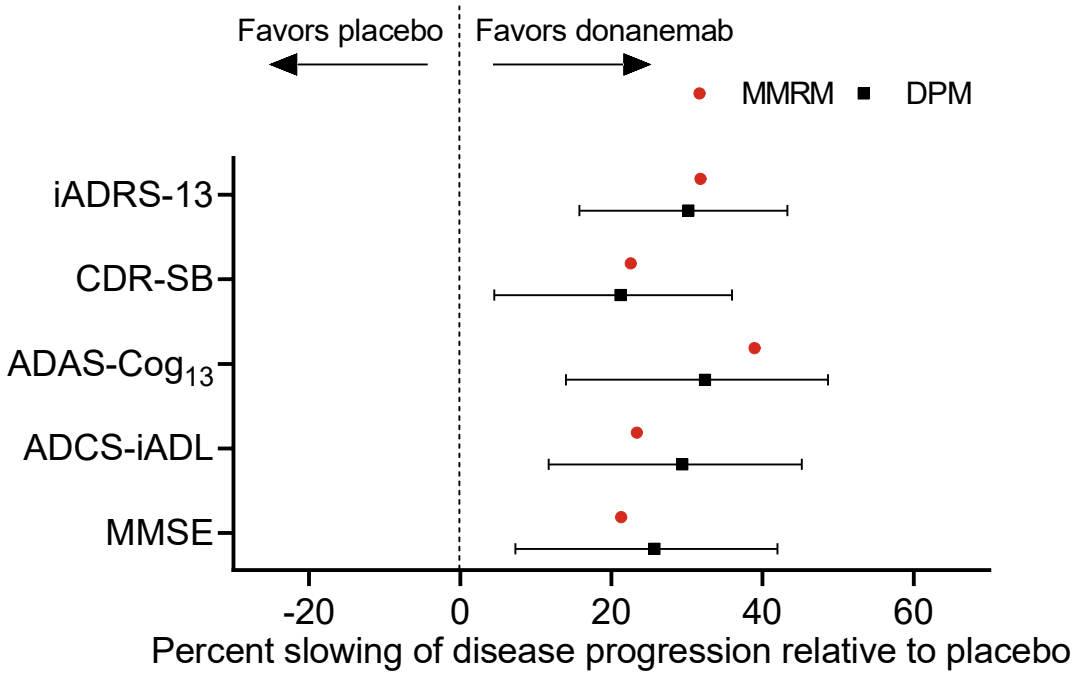


21% slowing

ADAS-Cog$_{13}$ = Alzheimer's Disease Assessment Scale - Cognitive subscale; ADCS-iADL = Alzheimer's Disease Cooperative Study - Activities of Daily Living Inventory; CDR-SB = Clinical Dementia Rating Scale; iADRS = Integrated Alzheimer's Disease Rating Scale; LS = Least Squares; n = number of patients; MMSE = Mini–Mental State Examination; SE = Standard Error

Secondary outcomes: DPM analysis

DPM analysis showed slowing in all clinical endpoints relative to placebo and was similar in magnitude with MMRM



The Disease Progression Model (DPM) assumes a proportional treatment effect relative to placebo, includes diffuse priors and generated a posterior probability distribution of the disease progression ratio.

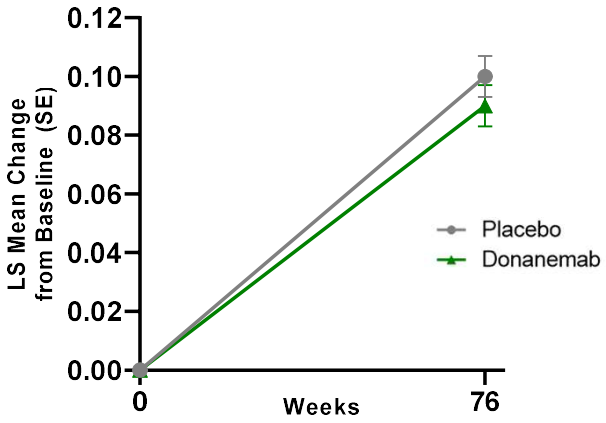
- MMRM model: at the 18-month endpoint
- Bayesian DPM: over the entire 18 months (95% credible intervals)

ADAS-Cog$_{13}$ = Alzheimer's Disease Assessment Scale - Cognitive subscale; ADCS-iADL = Alzheimer's Disease Cooperative Study - Activities of Daily Living Inventory; CDR-SB = Clinical Dementia Rating Scale; iADRS = Integrated Alzheimer's Disease Rating Scale; MMRM = Mixed-Model Repeated-Measures; MMSE = Mini–Mental State Examination

Primary measure of Tau^{IQ} Global Tau Load showed no significant change

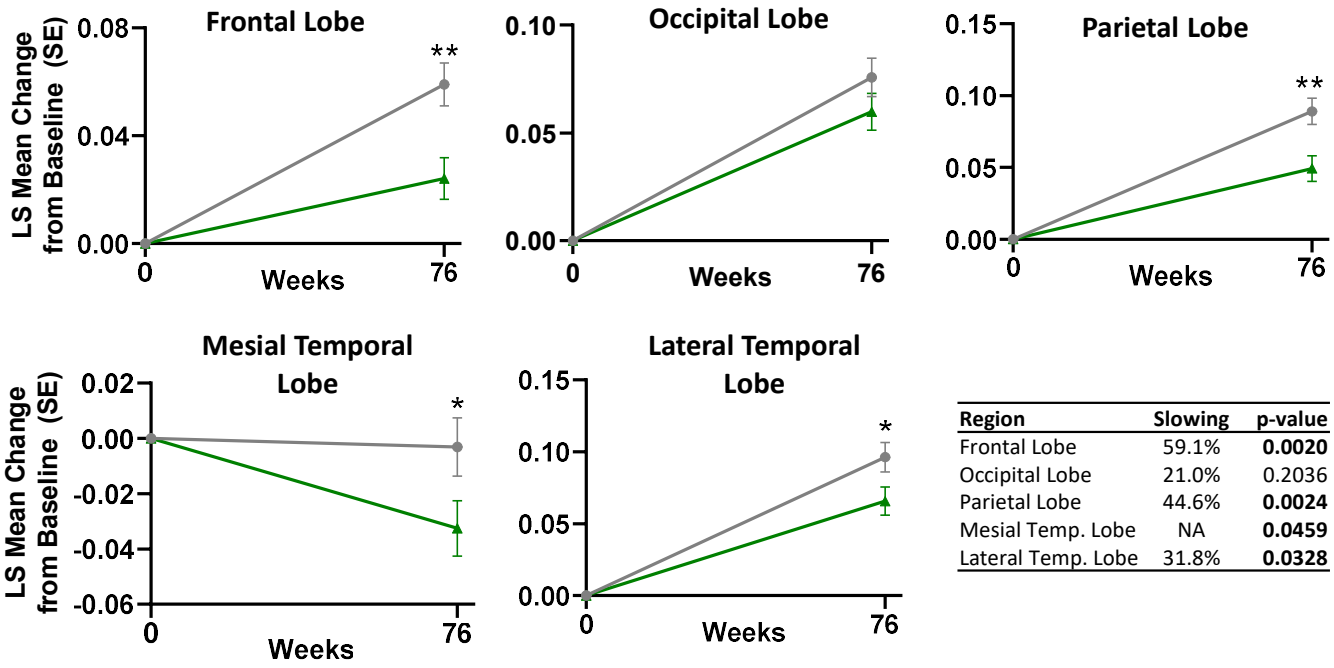
Exploratory Regional Analysis shows SIGNIFICANT DECREASE IN TAU LOAD

Global Tau Load (Tau^{IQ})



Treatment Arm	Baseline Mean (SD)	LS Mean Change from Baseline (SE)	p-value (treatment difference)
Donanemab	0.44 (0.163)	0.09 (0.007)	0.56
Placebo	0.46 (0.152)	0.10 (0.007)	

Regional SUVR with Cerebellar Gray Reference[#]

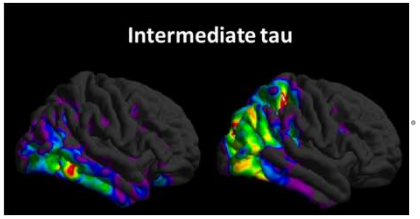


Region	Slowing	p-value
Frontal Lobe	59.1%	**0.0020**
Occipital Lobe	21.0%	0.2036
Parietal Lobe	44.6%	**0.0024**
Mesial Temp. Lobe	NA	**0.0459**
Lateral Temp. Lobe	31.8%	**0.0328**

LS = Least Squares; SD = standard deviation; SE = Standard Error; SUVr=Standardized Uptake Value ratio

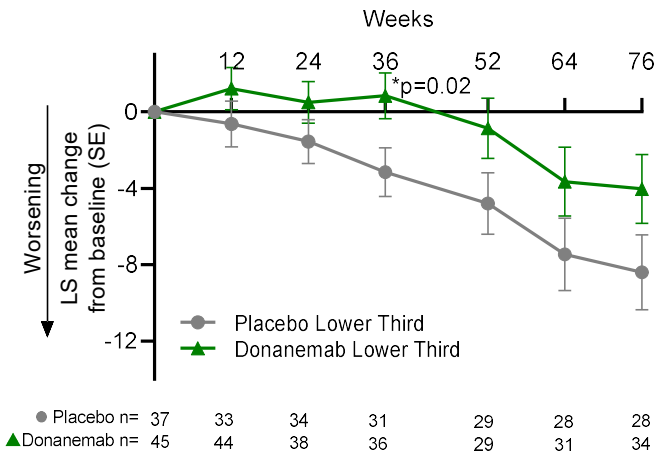
[#] AAL Regions using posterior cerebellum gray matter reference region

Exploratory analysis of iADRS of enrolled patients by baseline tau PET levels

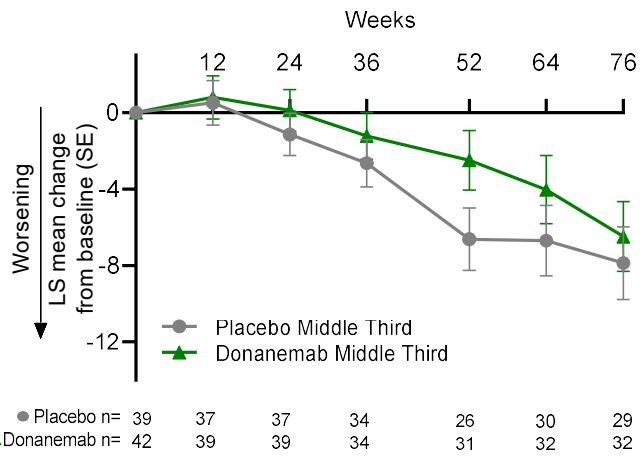


Intermediate tau enrolled patients were further stratified into terciles by baseline tau PET

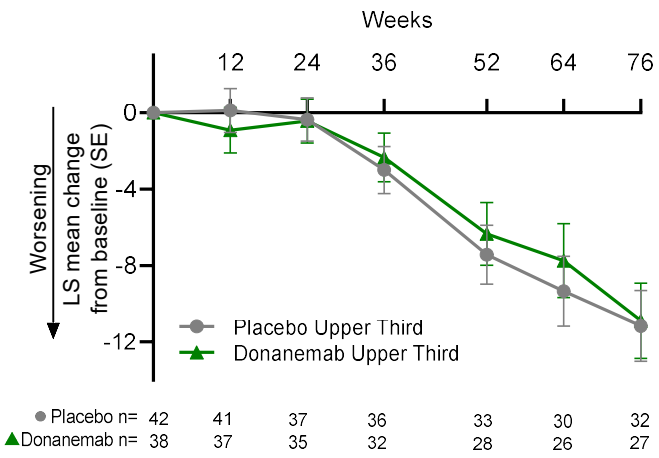
Lower baseline tau



Middle baseline tau



Upper baseline tau



Stratified by baseline flortaucipir SUVr; lower third cut point is 1.144; upper third cut point 1.274

iADRS = Integrated Alzheimer's Disease Rating Scale; LS = Least Squares; n = number of patients; PET = positron emission tomography; SE = Standard Error

Ongoing/Future Studies

- The pivotal TRAILBLAZER-ALZ2 (NCT04437511) study is ongoing and will continue to test donanemab in a larger study and broader geographic footprint

- TRAILBLAZER-EXT, a follow-on study for those who participated in TRAILBLAZER-ALZ, is currently enrolling participants (NCT04640077)

Sheila Seleri

Roche/Genetech

Genentech
A Member of the Roche Group

Roche

GENENTECH AND ROCHE IN ALZHEIMER'S DISEASE

Sheila Seleri, MD, PhD

Group Medical Director, US Medical Affairs

M-US-00011641

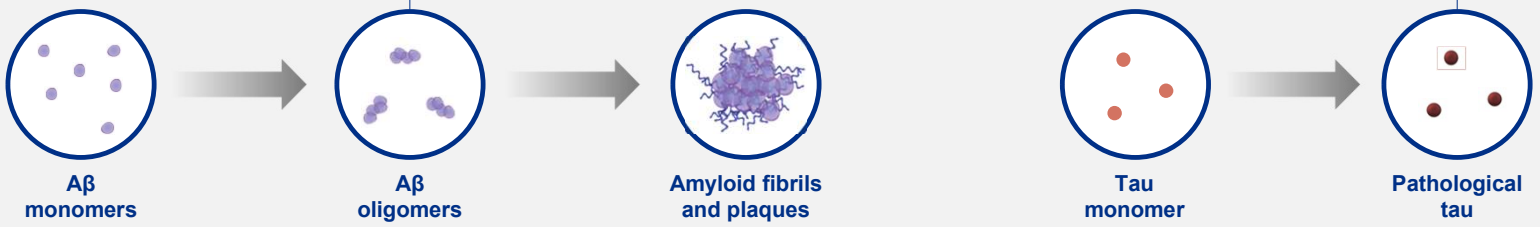
OUR INVESTIGATIONAL THERAPEUTICS SPAN DIFFERENT TARGETS, TYPES AND STAGES OF ALZHEIMER'S DISEASE

Crenezumab[1–3]

- Humanized anti-Aβ IgG4 mAb
- Designed to target multiple Aβ forms, with a preference for oligomers
- Ongoing study in ADAD only

Semorinemab[3,4–6]

- Anti-tau mAb
- Designed to target extracellular tau, including toxic soluble tau
- LAURIET ongoing in moderate AD



Gantenerumab[3,7–9]

- Fully human anti-Aβ IgG1 mAb
- Designed to target aggregated Aβ forms – binds oligomers and plaques
- Phase 3 GRADUATE program ongoing

Brain shuttle gantenerumab[10-11]

Brain shuttle technology is designed to optimize the penetration of large molecules into the brain

The interaction between Aβ and tau pathology in AD imply that both anti-Aβ and anti-tau therapies are important investigational treatment strategies for people with AD

Gantenerumab, crenezumab, semorinemab and brain shuttle technology are investigational and have not been approved by the FDA. Efficacy and safety have not been established.

Aβ, amyloid beta; AD, Alzheimer's disease; ADAD, autosomal dominant Alzheimer's disease; mAb, monoclonal antibody

1. Adolfsson O et al. *J Neurosci.* 2012;32:9677–9689; 2. ClinicalTrails.gov (NCT01998841). https://clinicaltrials.gov/ct2/show/NCT01998841. Accessed 27 September 2020; 3. Roche LTD. Product Development Portfolio. Updated on 23 July 2020. Accessed 27 September 2020. https://www.roche.com/research_and_development/who_we_are_how_we_work/pipeline.htm; 4. Clavaguera F et al. *Nat Cell Biol.* 2009;11:909–913. 5. ClinicalTrials.gov (NCT03289143). Available at: https://clinicaltrials.gov/ct2/show/NCT03289143. Accessed 27 September 2020; 6. ClinicalTrials.gov (NCT03828747). Available at: https://clinicaltrials.gov/ct2/show/NCT03828747. Accessed 27 September 2020; 7. Ostrowitzki S et al. *Arch Neurol* 2012;69:198–207; 8. Bohrmann B et al. *J Alzheimers Dis.* 2012;28:49–69. 9. ClinicalTrials.gov (NCT03443973). Available at https://clinicaltrials.gov/ct2/show/NCT03443973. Accessed 27 September 2020; 10.Weber F et al. *Cell Reports*. 2018;22:149–162; 11. Brain Shuttle. https://www.roche.com/research_and_development/what_we_are_working_on/research_technologies/protein-related_technologies/brain_shuttle.htm. Accessed on 13 October 2020.

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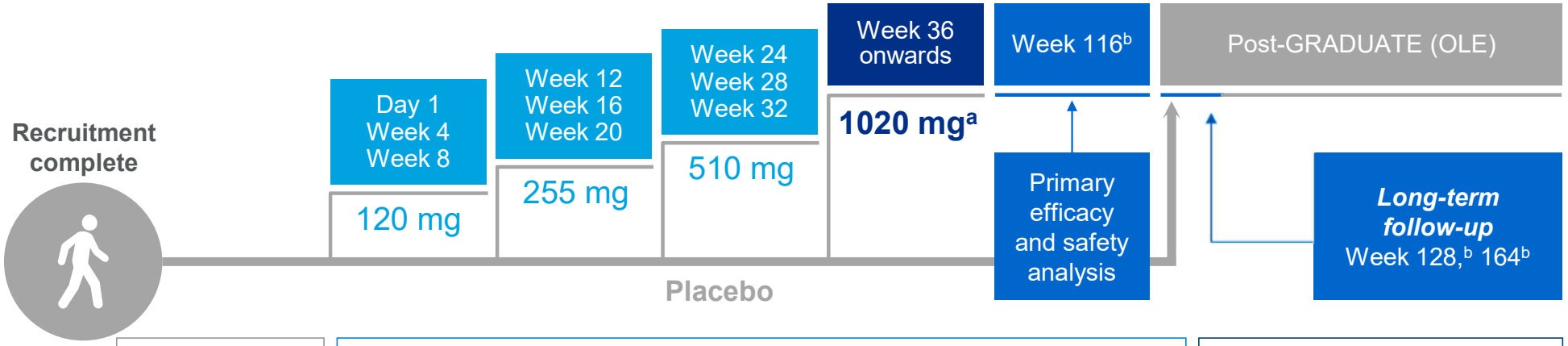
Veeva Document Number # M-US-00011641

GRADUATE I AND II (NCT03444870, NCT03443973) STUDY DESIGN

GRADUATE I GRADUATE II



Screening
up to
12 weeks

Main GRADUATE double-blind study

- SC gantenerumab dose escalation for 9 months followed by gantenerumab at target dose until primary analysis at Week 116[b]
- SC placebo as scheduled until primary analysis at Week 116[b]

Universal titration
Option for home administration
Optimised manufacturing process for 1020 mg dose

Post–double-blind period
1-year follow-up ***or*** optional enrolment in open-label extension study

Gantenerumab is investigational and has not been approved by the FDA. Efficacy and safety have not been established.

[a]1020 mg new drug formulation is similarly bioavailable as 1200 mg of the previous drug formulation. [b]GRADUATE studies could be extended to 30 months in total, in the event that COVID-19-related interruptions in dosing and other study procedures worsen significantly.
OLE, open-label extension; SC, subcutaneous.
1. Pross N et al. Presented at AD/PD 2019, March 26–31, Lisbon, Portugal; 2. Data on file.

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Ivana Rubino

Biogen

Lynn Kramer

Eisai

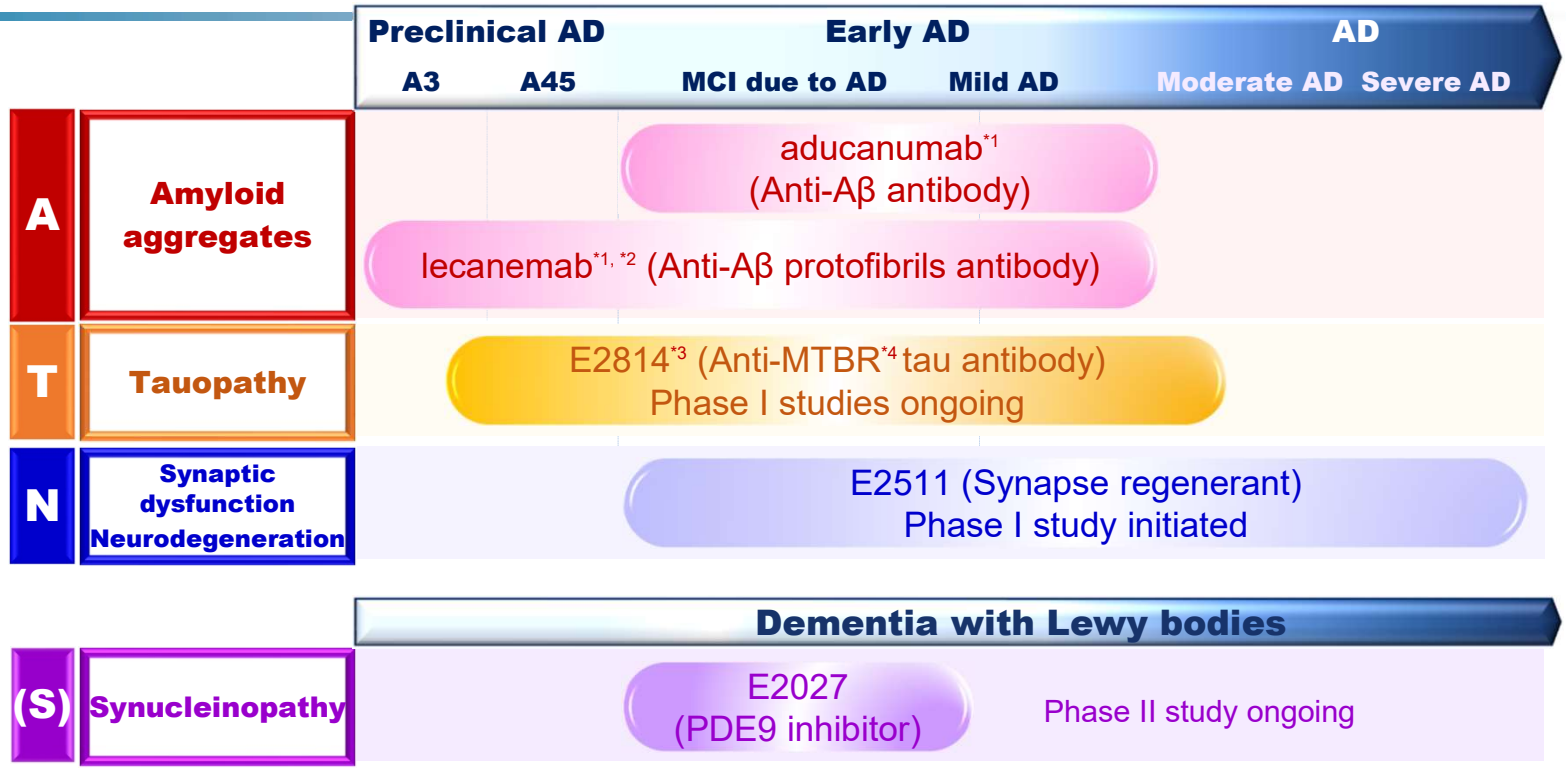
Neurodegenerative Disease Pipeline

Eisai Co., Ltd.

July 14, 2021

human health care

Clinical Stage Pipeline Based on AD Continuum and ATN+



Implement drug creation based on hypothesis of novel pathophysiology
Conduct clinical research making full use of biomarkers

All projects are investigational. *1: Co-development with Biogen *2: Generic name for BAN2401, an investigational antibody for Alzheimer's disease produced as the result of a strategic research alliance between Eisai and BioArctic *3: Co-research with University College London (UCL), UK *4: microtubule binding region

Open Discussion

Discussion Questions

- What are potentially achievable outcomes that payers will look for in AD mAb treatments? Based on currently available information about the drug class, as well as your own organizations' early review of this information, what are the highest priority questions where more evidence would be needed to inform coverage decisions? Specific questions to consider include:
  - For which populations might these treatments offer the greatest impact or value?
  - What are the most effective ways to improve understanding of the relationship between the treatment's surrogate endpoints and cognitive outcome endpoints? The relationship between the cognitive outcomes to other measures of value, such as quality of life, independence and reduced supportive care costs and caregiver burden?
  - What are the expectations associated with differing durations of treatment?
  - What are the most effective and efficient care models for mAb treatment, including diagnosis, monitoring, and management of treatment complications?

Discussion Questions

- What coverage and payment decisions might be appropriate for these treatments?
  - How will payers treat populations included in clinical trials versus less-studied populations when making coverage determinations? Would coverage differ by population group?
  - What additional evidence described previously would affect payers' willingness to cover these drugs?
- What are payers' pricing and payment expectations with regard to the AD mAb drug class? What is the expected pricing dynamic when there are several similar drugs in this space?
  - How might past experiences in the context of pricing and competition help inform this discussion, for example, the approval of multiple PCSK9 drugs for lowering cholesterol and the competition that impacted hepatitis C treatment prices?

Session II: Opportunities for Generating Clinical Evidence for Promising AD mAb Treatments

Maria Carrillo

The Alzheimer's Association

George Vradenburg

UsAgainstAlzheimer's

Open Discussion

Joseph Johnston

Eli Lilly and Company

Jennifer Whiteley

Roche/Genentech

Chris Leibman

Biogen

Amir Tahami

Eisai

Open Discussion

Discussion Questions

- Will the pivotal trials underway now or the planned Phase 4 (postmarket) studies provide substantial insights into the key evidence questions?
- What are near-term ways to augment these studies to fill key evidence gaps?
- Can these key evidence questions be addressed through observational real-world studies (e.g., registries), or are randomized studies needed?
- What potentially feasible further postmarket studies are most important to consider now – for both early-stage and later-stage patients?
- Are there any learnings to leverage from our collective experience with registries, practical platform trials, and other postmarket evidence initiatives?
- What should CMS consider in terms of an evidence development (e.g., CED) approach for these therapies? How might these studies be designed and executed given the existing coverage issues and challenges associated with creating a randomized controlled trial for the treatment?

Session III: Looking Forward

Steve Miller

Cigna

Kate Goodrich

Humana

Open Discussion

Discussion Questions

- How can stakeholders work together to address these questions in both premarket and postmarket studies?
- Would additional public-private collaboration help address these questions?
- What potential payment models could address the health care spending and evidence concerns associated with these therapies?

Closing Remarks

Mark McClellan

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Thank You!

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