
NEUROSCIENCE
Lilly

SAFE HARBOR PROVISION

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MARK MINTUN, M.D.

President of Neuroscience Research and Development,
and President of Avid Radiopharmaceuticals

Lilly

2021 **INVESTMENT COMMUNITY MEETING**

NEURODEGENERATION UNMET NEED

THE URGENT PROBLEM OF ALZHEIMER'S AND OTHER DEMENTIA

EVERY 65 SECONDS

NEW CASES: Someone in the United States develops Alzheimer's disease (AD) every 65 seconds. Alzheimer's disease is the sixth-leading cause of death in the United States.

5.8 MILLION AMERICANS IN 2019

HEALTH: Today, 5.8 million Americans are living with Alzheimer's disease, including an estimated 200,000 under the age of 65. By 2050, up to 14 million may have the disease.

$234 BILLION

ECONOMICS: In 2019, 16 million Americans provided 18.5 billion hours of unpaid care for a person living with Alzheimer's disease or other dementia – an economic value of $234 billion.

Non-AD Dementias: There are no current disease modifying treatments for PD, HD, FTD, ALS and other diseases impacting millions of patients. These diseases have similar types of protein misfolding and could respond to the same approaches being developed against AD.

2019 Alzheimer's Disease Facts and Figures – Alzheimer's Association; PD = Parkinson's Disease; HD = Huntington's Disease; FTD = Frontotemporal Dementia; ALS = Amyotrophic lateral sclerosis

NEURODEGENERATION FOCUS AND STRATEGY

Create diagnostics to enable effective treatment

Intervene earlier

Multiple therapies to attack disease pathologies

New platforms for genetic medicine delivery

AMYViD™
Florbetapir F 18 Injection

First radioactive diagnostic agent approved by the FDA for PET imaging of the brain to estimate beta-amyloid neuritic plaque density

TAUVID™
(Flortaucipir F 18 Injection)

Only approved diagnostic to image tau neurofibrillary tangles in the brain

Plasma Phospho-tau217

Blood-based assay being developed as potential Early Diagnostic Biomarker of Alzheimer's Disease

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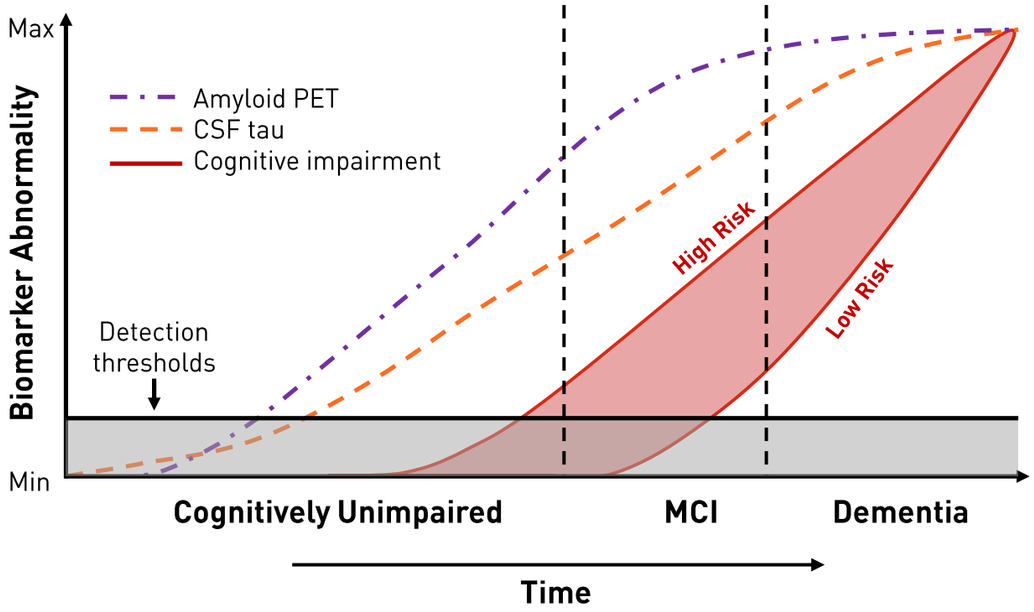
Stages of AD & Biomarker Abnormality Levels



Adapted from Selkoe & Hardy EMBO, 2016

Can we turn back the clock on amyloid accumulation BEFORE brain damage and symptoms?

AD = Alzheimer's disease; CSF = Cerebrospinal Fluid; MCI = Mild Cognitive Impairment

NEURODEGENERATION FOCUS AND STRATEGY

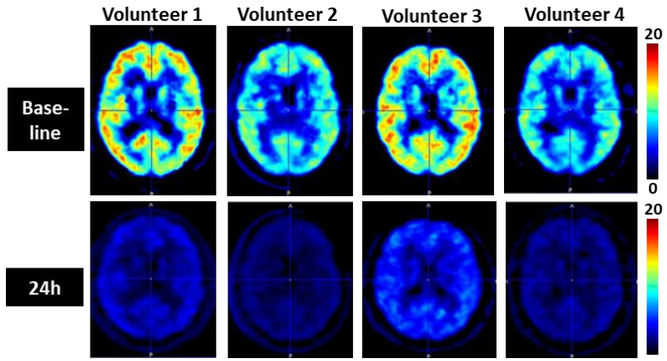
Create diagnostics to enable effective treatment

Intervene earlier

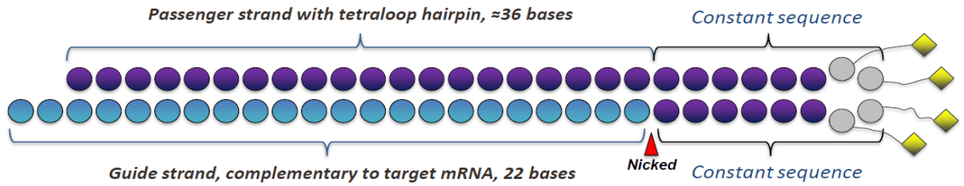
Multiple therapies to attack disease pathologies

New platforms for genetic medicine delivery

Next frontier to target tau pathology beyond anti-amyloid therapy



OGA II



siRNA

NEURODEGENERATION FOCUS AND STRATEGY

Create diagnostics to enable effective treatment

Intervene earlier

Multiple therapies to attack disease pathologies

New platforms for genetic medicine delivery

Prevail THERAPEUTICS

Anima Biotech

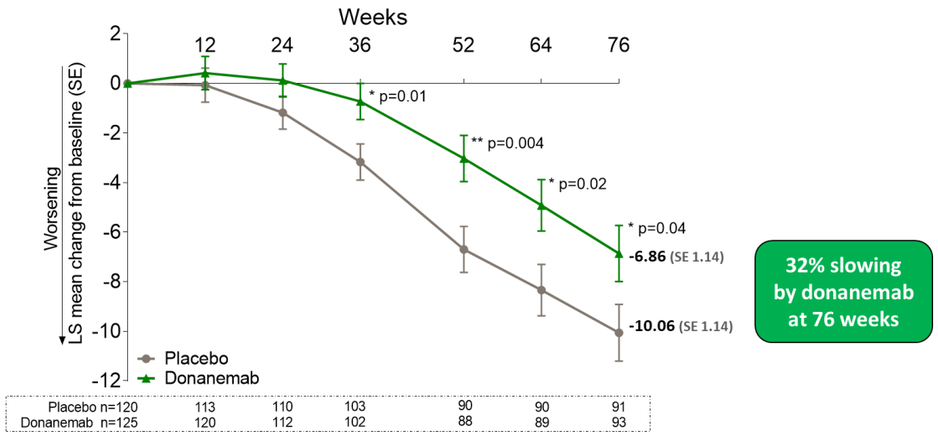
Dicerna pharmaceuticals

evox THERAPEUTICS

AVIDITY BIOSCIENCES

Focus on monogenic diseases and broaden into polygenic diseases

DONANEMAB OVERVIEW



32% slowing by donanemab at 76 weeks

Donanemab is an immunoglobulin G1 antibody **specific for** an N-terminal pyroglutamate amyloid-ß epitope that is present only in **mature brain amyloid plaques**

Phase 2 primary outcome showed **donanemab significantly slowed clinical progression by 32% on iADRS** at 76 weeks, compared with placebo

Taking on the Challenges Ahead

Advance the Science

Advance scientific understanding of **plaque lowering** and of **ARIA**

Find and Treat Pathology; Develop AD Ecosystem

Advance accurate AD **diagnosis**; Establish diagnostic & infusion **infrastructure**

Demonstrate the Value of Anti-Amyloid Therapy

Confirmatory trial; National Coverage Analysis (NCA) Leadership; Pilot **innovative models**

TRAILBLAZER-ALZ SAFETY PROFILE

AS DEMONSTRATED IN TRAILBLAZER-ALZ, SAFETY REMAINS OF UTMOST IMPORTANCE ACROSS ALL PROGRAMS

SAFETY & TOLERABILITY

Overview of Adverse Events, n (%)			
	Placebo (n=125)	**Donanemab (n=131)**	**p-value**
Deaths	2 (1.6)	1 (0.8)	0.62
SAEs	22 (17.6)	23 (17.6)	>1.00
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
TEAEs	113 (90.4)	119 (90.8)	>1.00
Treatment Emergent Adverse Events ≥5% Incidence, n (%)			
ARIA-E	1 (0.8)	35 (26.7)	<0.001
Fall	19 (15.2)	17 (13.0)	0.72
Dizziness	15 (12.0)	11 (8.4)	0.41
Headache	15 (12.0)	10 (7.6)	0.29
Superficial siderosis of central nervous system	4 (3.2)	18 (13.7)	0.003
Arthralgia	10 (8.0)	10 (7.6)	>1.00
Nausea	4 (3.2)	14 (10.7)	0.03
Upper respiratory tract infection	9 (7.2)	9 (6.9)	>1.00
Urinary tract infection	5 (4.0)	13 (9.9)	0.09
Diarrhea	5 (4.0)	11 (8.4)	0.20
ARIA-H	4 (3.2)	11 (8.4)	0.11
Cerebral microhemorrhage	3 (2.4)	10 (7.6)	0.09
Infusion-Related Reaction	0 (0)	10 (7.6)	0.002
Pneumonia	5 (4.0)	7 (5.3)	0.77
Depression	8 (6.4)	6 (4.6)	0.59
Contusion	10 (8.0)	0 (0)	<0.001
Vomiting	3 (2.4)	7 (5.3)	0.34
Anxiety	2 (1.6)	7 (5.3)	0.17

FINDINGS

- Safety profile in line with Phase 1 results; no new safety signals observed
- Rate of symptomatic ARIA-E was 6% in the treatment arm (with 27% showing any ARIA-E), similar to other plaque-clearing agents
- Treatment discontinuation due to ARIA was driven by strict protocol criteria
- We remain confident in the risk/benefit profile of donanemab, though note an outstanding issue for the field is the development of strategies for avoidance and management of ARIA
- We look forward to contributing to solutions for managing symptomatic ARIA risk and continue to work closely with stakeholders in the Alzheimer's field

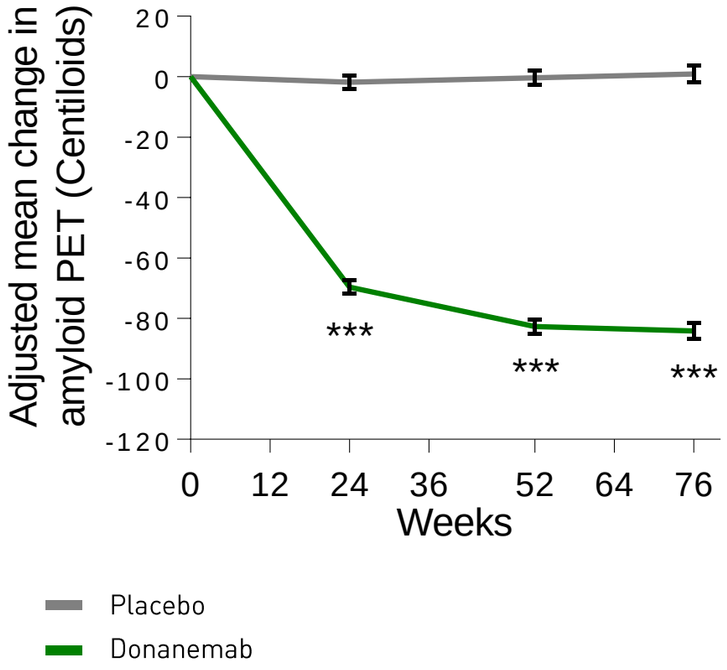
SAE = Serious Adverse Event; TEAE = Treatment-Emergent Adverse Event; ARIA-E = Amyloid-Related Imaging Abnormalities with Edema/Effusions; ARIA-H = Amyloid-Related Imaging Abnormalities with hemosiderin deposits

*Discontinued treatment due to protocol-defined criteria and patient/principal investigator-cited reasons for discontinuation

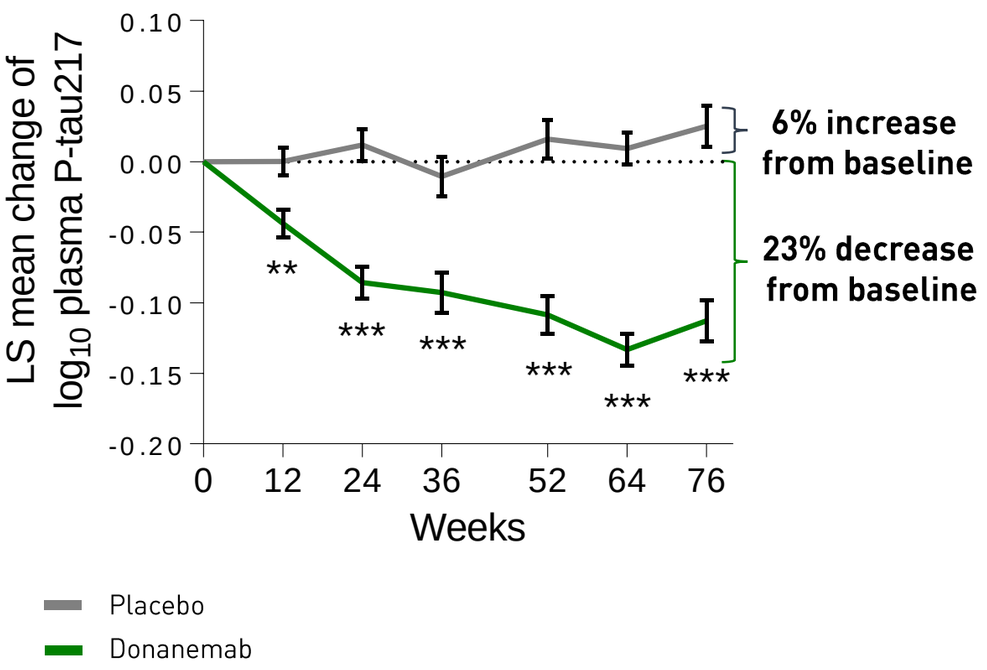
DONANEMAB

BOTH AMYLOID PLAQUE AND PLASMA P-TAU217 WERE SIGNIFICANTLY LOWERED WITH DONANEMAB

Amyloid plaque significantly lowered with donanemab treatment[1]



Plasma P-tau217 significantly lowered with donanemab treatment[2]



Key Findings

- Previously showed evidence for slowing regional tau tangles by PET
- Now with additional evidence that donanemab reduced the tau pathological cascade

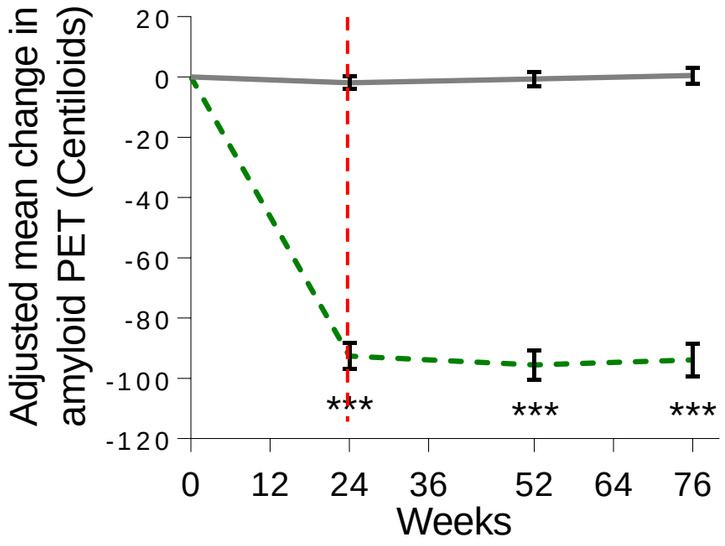
* p<0.05; ** p<0.01; *** p<0.001 vs placebo; [1]secondary endpoint; [2]exploratory objective

TRAILBLAZER-ALZ Mixed Model Repeated Measures (MMRM) analysis; Data points show mean +/- standard error; LS = Least Square; PET = Positron Emission Tomography; p = p-value

DONANEMAB

BOTH AMYLOID PLAQUE AND PLASMA P-TAU217 REDUCTION CONTINUED FOR ONE YEAR AFTER DOSING STOPPED

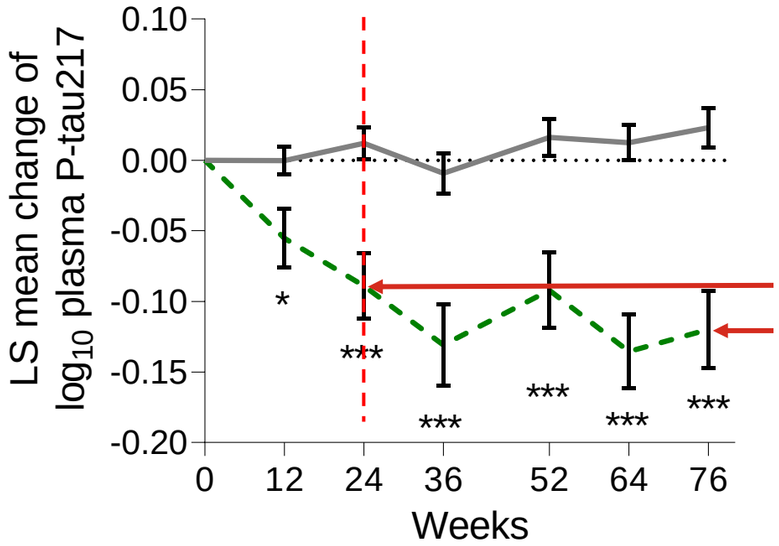
Amyloid plaque significantly lowered with donanemab treatment[1]



Placebo

Donanemab – stopped at 24 weeks due to complete amyloid clearance

Plasma P-tau217 significantly lowered with donanemab treatment[2]



Placebo

Donanemab – stopped at 24 weeks due to complete amyloid clearance

Drop in P-tau217 was still present one year after clearing plaques and stopping donanemab treatment

* p<0.05; ** p<0.01; *** p<0.001 vs placebo; [1]exploratory objective; [2]exploratory objective

TRAILBLAZER-ALZ Mixed Model Repeated Measures (MMRM) analysis; Data points show mean +/- standard error; LS = Least Square; p = p-value

DONANEMAB

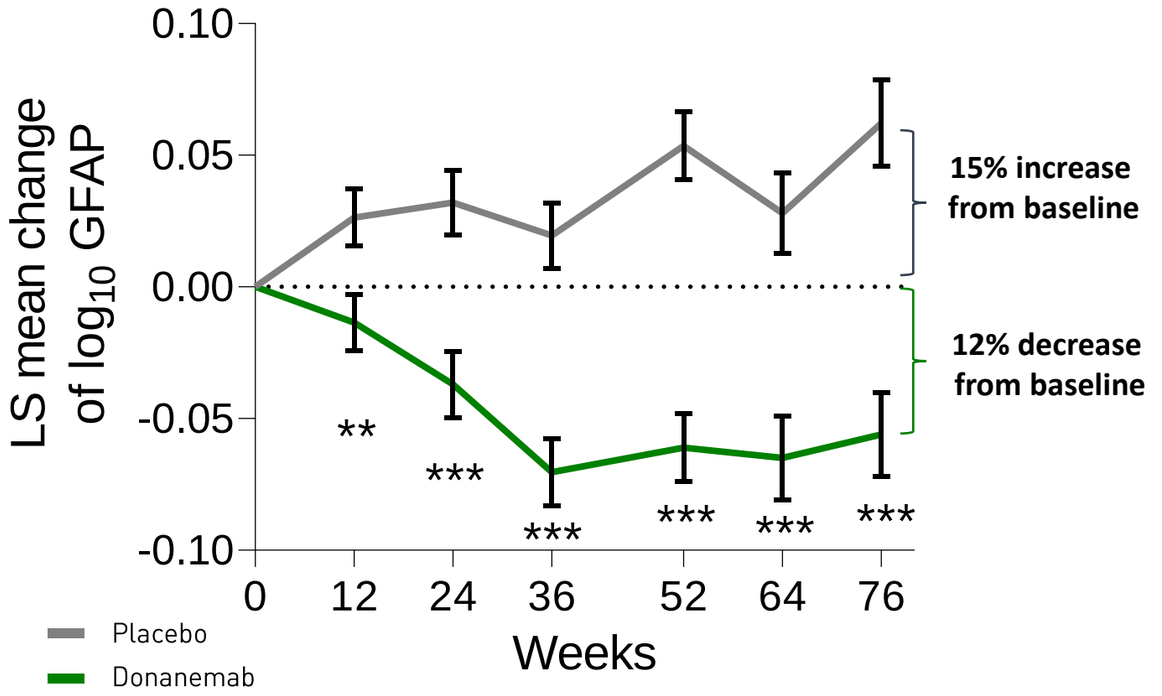
IN TRAILBLAZER-ALZ, AMYLOID PLAQUE, PLASMA P-TAU217 & NOW GFAP WERE SIGNIFICANTLY LOWERED WITH DONANEMAB

GLIAL FIBRILLARY ACIDIC PROTEIN

- Elevated GFAP is a highly specific inflammatory biomarker of astrogliosis in the brain
- Donanemab lowered plasma GFAP similar to the reductions seen in amyloid and P-tau
- Strengthens the evidence that donanemab treatment is associated with reducing multiple pathological processes of Alzheimer's disease

REDUCED INFLAMATORY MARKER

Glial Fibrillary Acidic Protein (GFAP) significantly lowered with donanemab treatment[1]



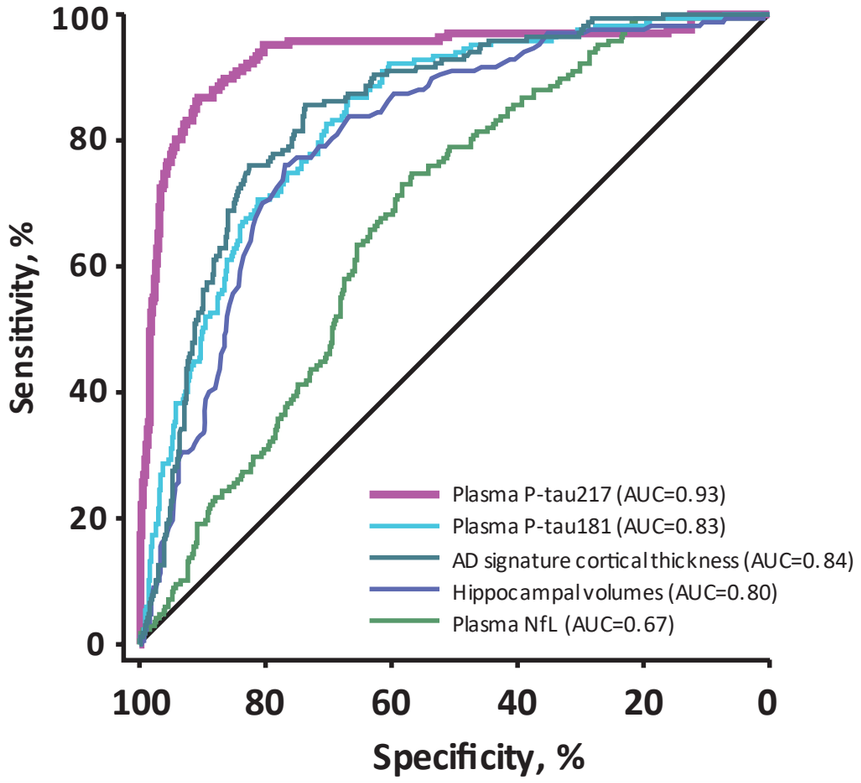
* p<0.05; ** p<0.01; *** p<0.001 vs placebo; [1]exploratory objective

TRAILBLAZER-ALZ Mixed Model Repeated Measures (MMRM) analysis; Data points show mean +/- standard error; LS = Least Square; p = p-value

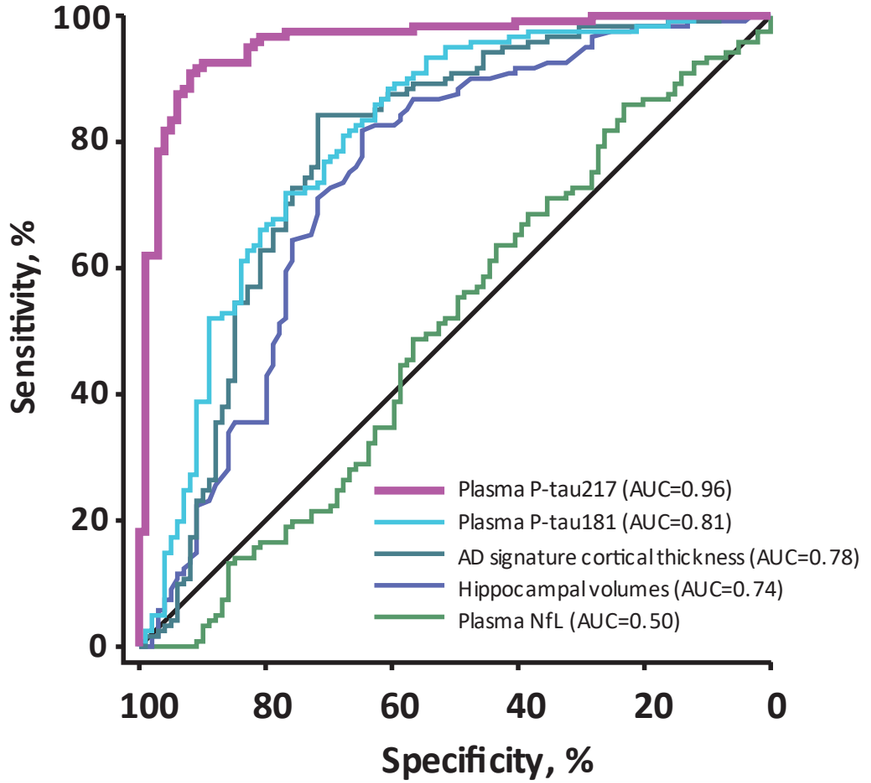
P-TAU 217 PLASMA ASSAY MAY TRANSFORM AD DIAGNOSIS

P-TAU217 PREDICTED AD PATHOLOGY IN A RESEARCH SETTING

Predicted tau-PET with an AUC of 0.93



Discriminated AD from other neurodegenerative diseases with an AUC of 0.96



P-tau217 also predicted amyloid-PET with an AUC of 0.87

Data from Palmqvist et al, JAMA 2020; AD = Alzheimer's disease; PET = Positron Emission Tomography; p = p-value; AUC = Area Under the Curve; NfL = Neurofilament Light Chain

P-TAU 217 PLASMA ASSAY MAY TRANSFORM AD DIAGNOSIS

LEVERAGING PLASMA P-TAU217 FOR FUTURE CLINICAL CARE

Potential to speed up diagnosis, decrease biomarker testing cost and provide more convenient access to patients

- Represents evolution towards less invasive diagnostics for patients
- Accessibility should lower the barrier for AD biomarker testing
- Investigating how to best transition our research experience into clinical practice
- Will look to combine Lilly's expertise with established diagnostic partners

AD = Alzheimer's disease

Not for promotional use

TRAILBLAZER ALZ-3
PREVENTION STUDY IN COGNITIVELY UNIMPAIRED

TRIAL DESIGN

- Preclinical AD event-driven prevention study
  - 3300 participant randomized trial measuring the Global Scale for Clinical Dementia Rating as primary endpoint
  - Target individuals 55-80 years of age who are cognitively unimpaired but with evidence of AD disease pathology
- P-tau217 assay used as part of inclusion criteria
  - New paradigm for clinical trial recruitment
  - Increased flexibility for screening activities, e.g., mobile units
- Decentralized trial with central services
  - Decentralized sites, including those in under-represented communities, to enable recruitment of diverse populations
  - Central safety monitoring including MRIs for detecting ARIA
- Informed by learnings from solanezumab A4 study

GOALS

- To assess whether a short course of donanemab treatment (9 infusions) at the start of the trial can slow or prevent progression to the clinical stages of AD
- Plan to complete enrollment by the end of 2022
- Anticipate 3-4 years after last patient enrolled for events to accrue for primary endpoint

AD = Alzheimer's disease

TRAILBLAZER ALZ-4

HEAD-TO-HEAD PHASE 3 STUDY COMPARING DONANEMAB TO ADUCANUMAB

TRIAL DESIGN & TIMELINE

- 200 patient, 18-month treatment study measuring change in amyloid at three time points (6, 12 & 18 months)
- Enrollment to be completed 1Q 2022
- Primary readout at 6-month time point; expected in the second half of 2022

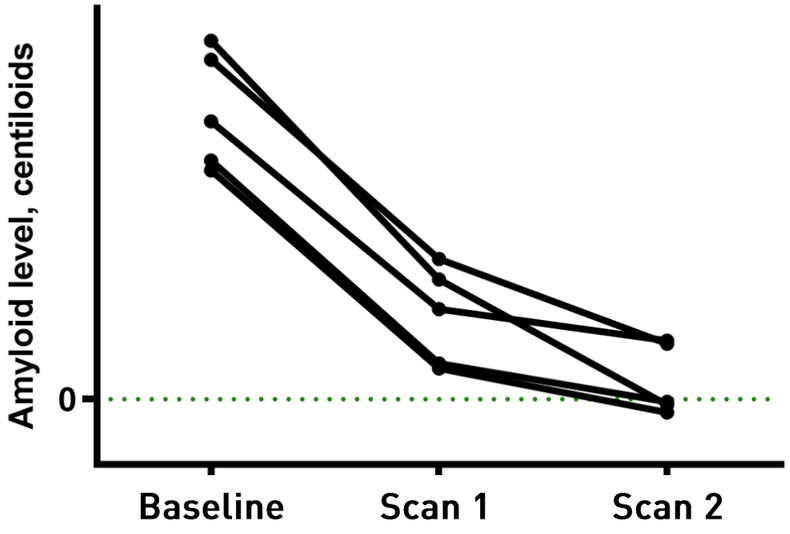
GOALS & RATIONALE

- To assess superiority of brain amyloid plaque clearance in early symptomatic population
- First trial to provide direct comparison between amyloid-lowering agents for biomarker efficacy
- Given plaque lowering is FDA-approved surrogate biomarker for efficacy, the magnitude and speed of plaque lowering should be critical basis of comparison

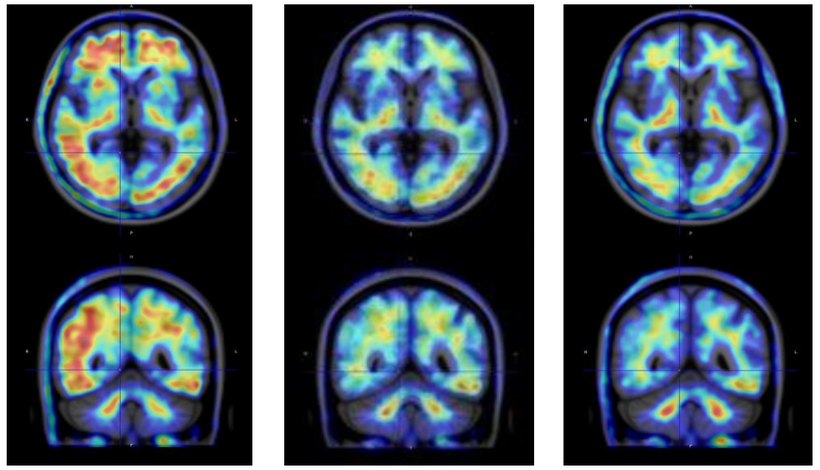
N3PG4 (LY3372993)

ONGOING PHASE 1B SHOWS DEEP PLAQUE CLEARANCE AND SAFETY PROFILE CONSISTENT WITH AMYLOID-LOWERING CLASS

Phase 1 Data



Amyloid PET Scans for a Patient



- Aiming for next generation N3pG **amyloid lowering agent** with flexible dosing regimens, including subcutaneous, to address the different needs of the Alzheimer's disease population
- **Prioritizing** safety and more-convenient patient experience

Note: Figures in parenthesis indicate standard deviation; CL level of zero indicates average amyloid level of a cognitively normal young control subject

Based on Phase 1 results, potential start of pivotal studies are expected in 2022

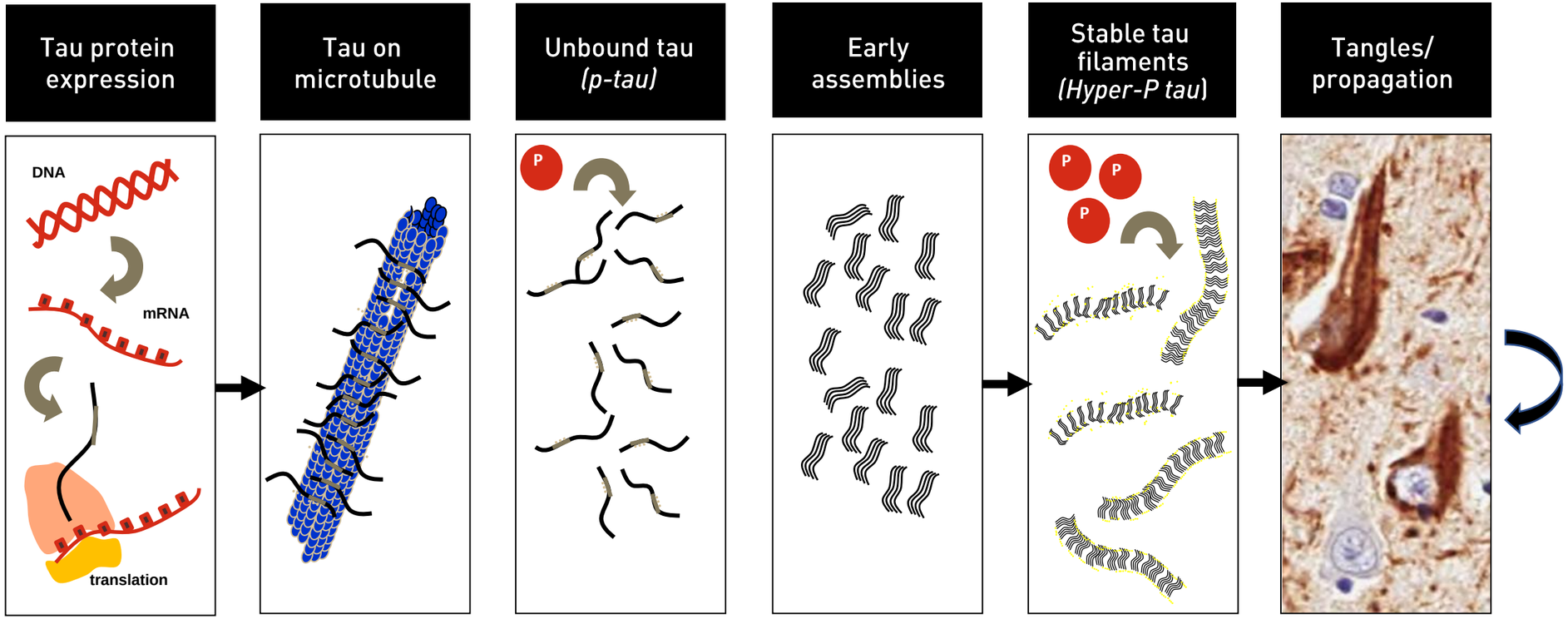
TARGETING TAU FOR ALZHEIMER'S DISEASE

Addressing various phases of tau pathology

Suppress Tau expression using multiple approaches

Block Tau aggregation with small molecule approaches, such as OGA inhibitor

Stop Transneuronal Tau pathology spreading



Note: zagotenemab development was discontinued

O-GlcNAcase (OGA) INHIBITOR (LY3372689)

AN ORAL ANTI-TAU SMALL MOLECULE WITH THE POTENTIAL TO MODIFY ALZHEIMER'S DISEASE

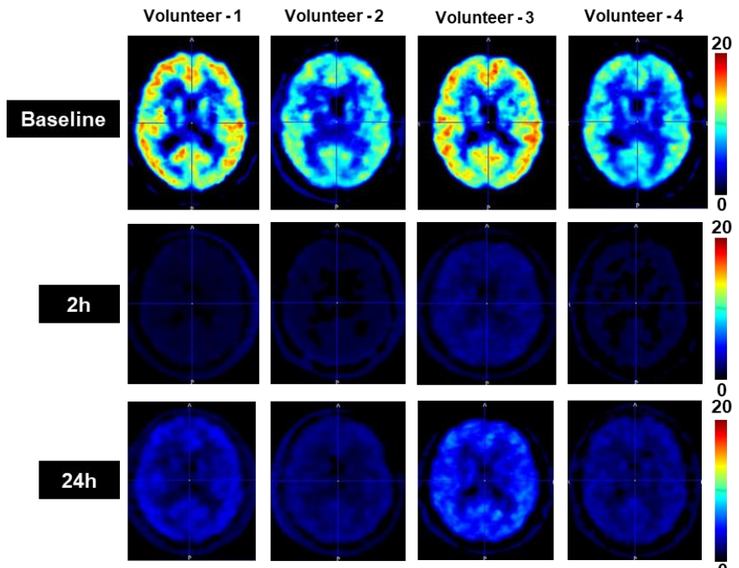
PRE-CLINICAL DATA

- Hit the target
  - ✓ Demonstrated >80% enzyme occupancy (EO)
- Reduced AD pathology
  - ↓ 50% reduction of tau pathology
- Slowed neurodegeneration
  - ↓ 40% reduction of brain atrophy
- Improved memory decline in mouse models

PHASE 1 DATA

- No dose limiting Adverse Events
- >80% EO achieved at 1mg dose
- Robust EO and manageable safety profile confidently inform Phase 2 dosing

OGA Inhibitor Demonstrates Strong Enzyme Occupancy After a Single Dose of 1mg



Distribution volume (V_T) images generated using Logan graphical analysis and arterial input function

PHASE 2 DESIGN

- Target population
  - Early symptomatic Alzheimer's disease
  - Evidence of tau pathology by PET scan
- Trial Design
  - Three arms (placebo, high dose, low dose)
  - Randomized, 110 patients/arm
  - Estimated enrollment completion: 2H 2022
- Primary endpoint
  - Integrated Alzheimer's Disease Rating Scale (iADRS) at 76-124 weeks with common close design in those with intermediate levels of brain tau
- Independent Data Safety Monitoring reviews

AD = Alzheimer's disease

Not for promotional use

ANTI-TAU siRNA DEVELOPMENT

Tau therapeutic hypothesis…

Pathogenesis of tauopathies (including AD/PSP)

MAPT (tau) transcription

↓

siRNA to degrade mRNA ⊣ *MAPT* mRNA expression

↓

Tau protein expression

↓

Tau aggregation (NFTs) and propagation (spread)

↓

Neurodegeneration

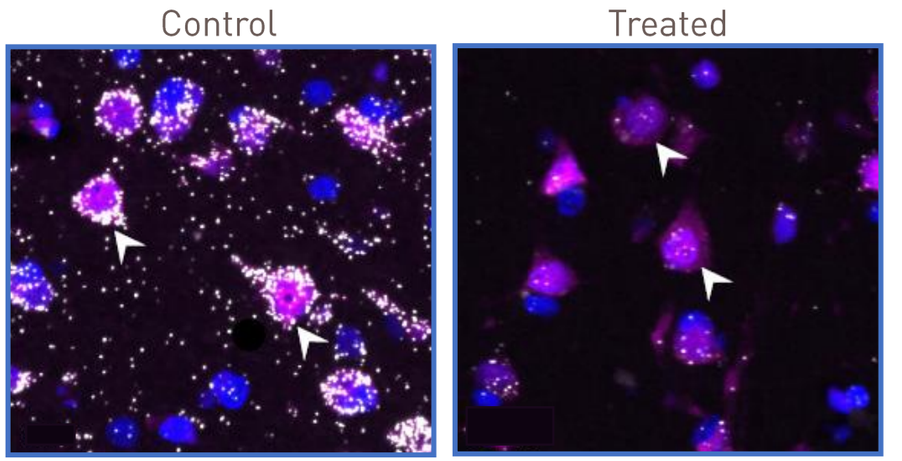
↓

Clinical symptoms and progression

Reducing tau protein expression will prevent down-stream effects

MAPT (Tau) mRNA Reduction…

MAPT mRNA Reduction in NHP Cortical Neurons following CSF Injection: MAPT SiRNA



- Presence of MAPT mRNA – white staining
- DAPI (nuclei)
- NeuN (neuronal marker)

Tau siRNA assets developed in collaboration with Dicerna; NHP = Non-Human Primates; MAPT = Microtubule-Associated Protein Tau; NFT = Neurofibrillary Tangles; siRNA = small interfering RNA; mRNA = messenger RNA; CSF = Cerebrospinal fluid

PREVAIL THERAPEUTICS: FOCUS ON GENE THERAPY

THREE ON-GOING PHASE 1/2 CLINICAL TRIALS (OPEN-LABEL, DOSE-ESCALATING)

REPLACE GBA1 ENZYME

PD-GBA Patients

PR001

propel

- Moderate to severe Parkinson's disease
- Single or biallelic GBA1 mutations
- Anticipated LPE: Mid-2022

Type 2 Gaucher Patients

PR001

provide

- Neurological signs & symptoms consistent with Type 2 Gaucher disease
- Biallelic GBA1 mutations
- Anticipated LPE: 2023

REPLACE GRN PROTEIN

FTD-GRN Patients

PR006

proclaim

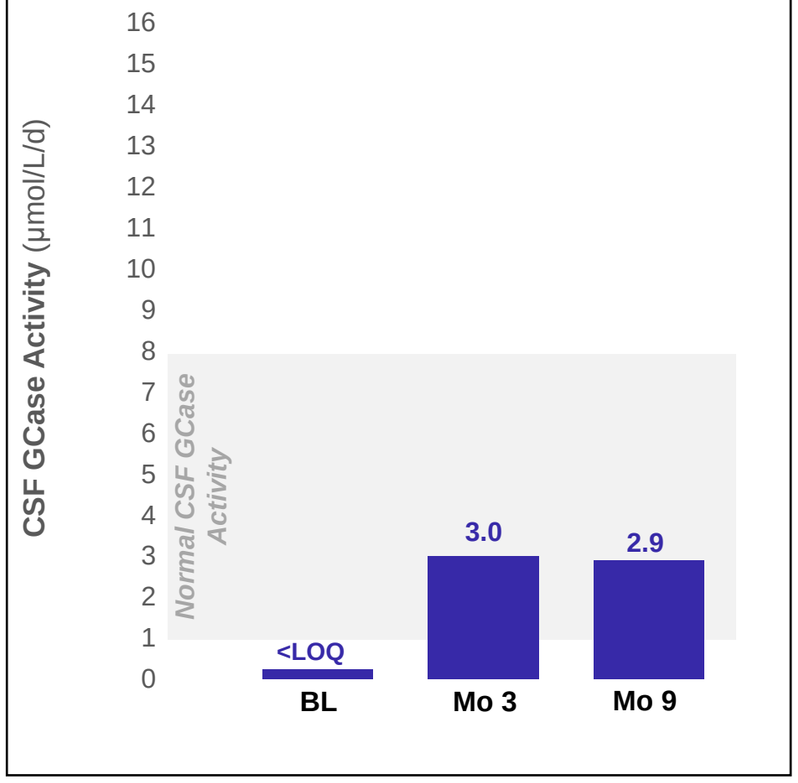
- Single pathogenic GRN mutation
- 30-80 years old
- Symptomatic disease stage
- Anticipated LPE: 2023

PD-GBA = Parkinson's disease glucosylceramidase beta; FTD GRN = Frontotemporal dementia progranulin; LPE = Last Patient Enrolled

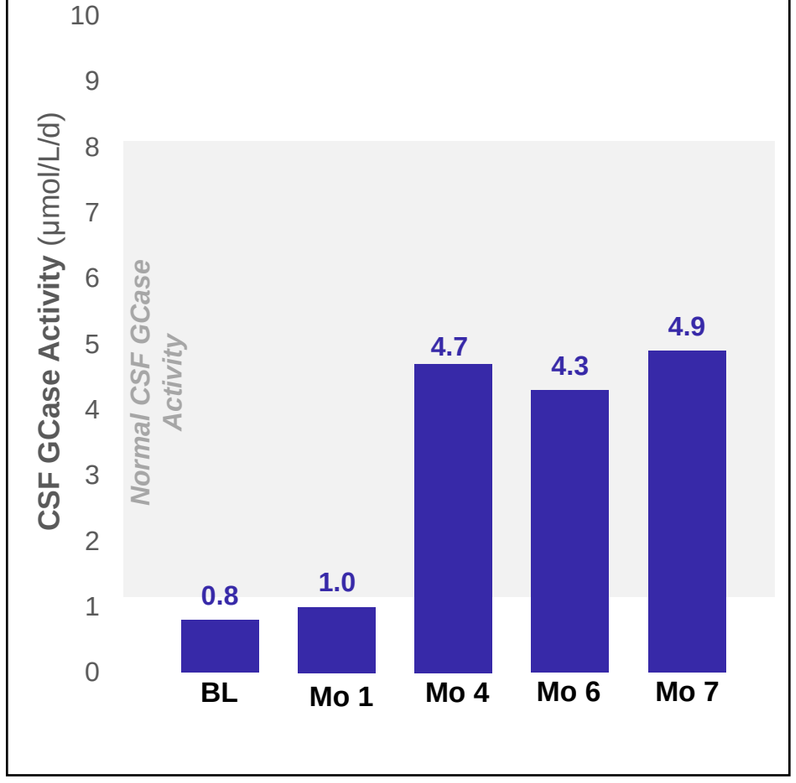
PREVAIL THERAPEUTICS: PRELIMINARY BIOMARKER DATA

KEY CSF BIOMARKER GOAL ACHIEVED ACROSS ALL THREE TRIALS

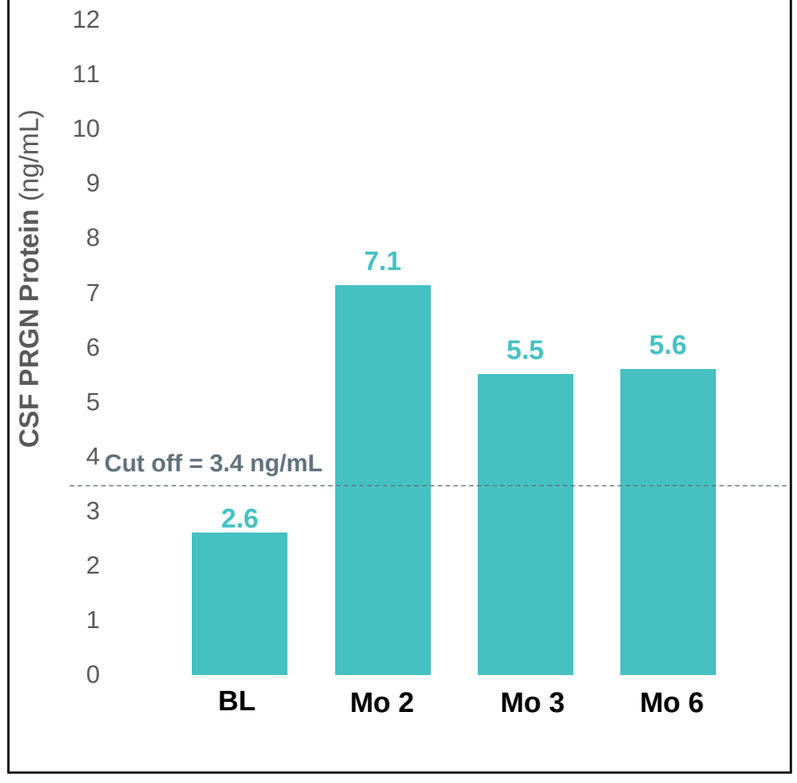
PD-GBA (GD1) patient treated with PR001



GD2 patient treated with PR001



FTD-GRN patient treated with PR006



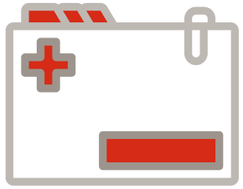
CSF = Cerebrospinal fluid; GBA1 = mutation associated with Parkinson's disease subtype; T2 = type 2; GRN = mutation associated with frontotemporal dementia subtype; LOQ = Limit of Quantification; BL = Baseline; Mo = Month; GCase = Glucocerebrosidase; PRGN = Progranulin

CHRONIC PAIN IS A PUBLIC HEALTH CRISIS

1.7B

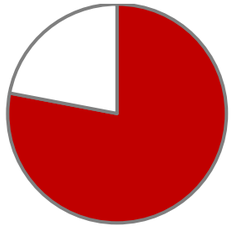
1 in 5 people suffer from chronic pain globally [1]

#1



Most common cause of long-term disability[2, 3]

78%



Of chronic pain patients are unsatisfied with treatment[4]

> $1T

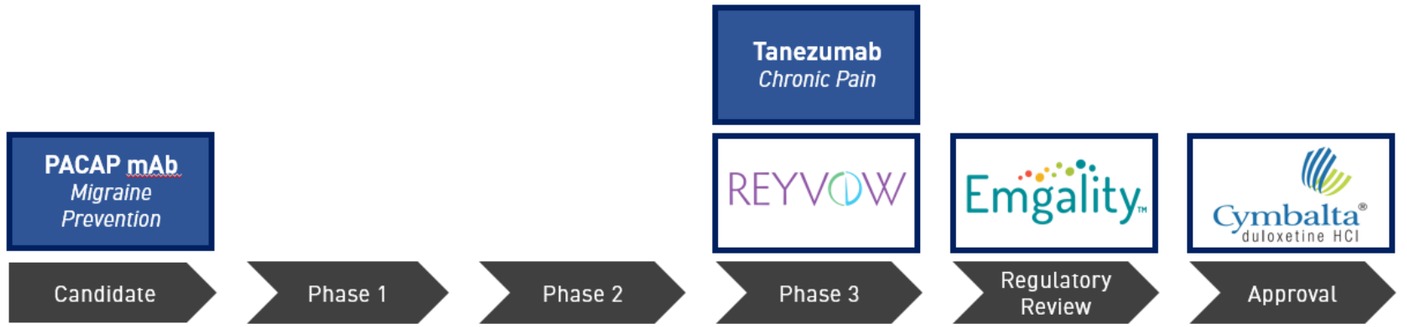
Annual cost of chronic pain[5,6] – more than cancer, heart disease and diabetes combined[7]

1 https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3201926/
2 https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6676152/#bib5
3 https://www.painresearchforum.org/news/114659-worldwide-scientific-and-policy-response-problem-chronic-pain
4 https://pae-eu.eu/wp-content/uploads/2017/07/PAE-Survey-2017-poster-for-web.pdf
5 Combined estimates from US, EU, JP
6 https://www.ncbi.nlm.nih.gov/books/NBK92521/; https://www.businesswire.com/news/home/20170519005141/en/Societal-Impact-of-Pain-Costs-the-EU-up-to-441-Billion-Euros-Annually
7 https://www.sciencedirect.com/science/article/pii/S1526590012005597

WHERE WE WERE...

PAIN PIPELINE (DECEMBER 2018)

Gap existed between late phase and discovery pipeline



DEVELOPING INNOVATIVE SOLUTIONS FOR PAIN

STRATEGIC APPROACH

Challenges for Pain Research

- Animal models do not identify responsive populations
- Many credible targets but few with POC in patients

Rapid Clinical POCs

- Lilly launched the first Pain Master Protocol (PMP)
- Three assets entered PMP to date.
- Expected to have 13 new POC readouts by 2023-end (including 12 from PMP)

Building Neuronal Health Platform

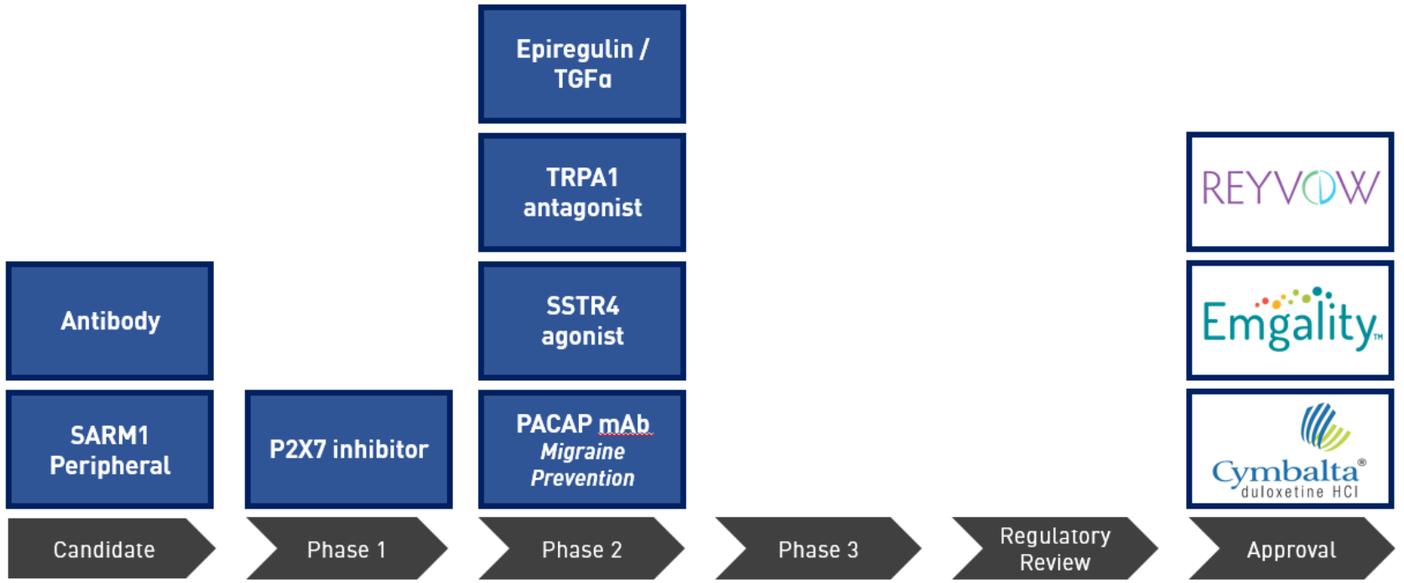
- IND-ready programs for neuronal degeneration
- Evaluating targets to repair damage

Business Development

- Four Pain deals in 3 years (SSTR4 agonist, TRPA1 antagonist, P2X7 inhibitor, and SARM1 inhibitor)

PAIN PIPELINE (DECEMBER 2021)

Lilly has significantly grown early pipeline (seven molecules between CS and Ph2 today)



POC = Proof of Concept; IND = Investigational New Drug

Not for promotional use

LILLY NEUROSCIENCE PIPELINE

SELECT NME AND NILEX PIPELINE AS OF OCTOBER 22, 2021

Pre-Clinical

- TAU siRNA ★ — Alzheimer's
- SARM1 INHIBITOR — Pain
- PI3Ka SELECTIVE — Cancer
- FGFR3 INHIBITOR — Cancer
- 2nd GENERATION RET INHIBITOR — Cancer
- BCL2 INHIBITOR — Cancer
- DACRA — Obesity / Diabetes
- LAARA — Obesity / Diabetes
- Glucose Sensing Insulin — Diabetes

PHASE 1

- RIPK1 INHIBITOR — Immunology
- P2X7 INHIBITOR — Pain
- NRG4 AGONIST — Heart Failure
- LP(a) siRNA — CVD
- KHK INHIBITOR II — Diabetes / NASH
- GIPR AGONIST LA II — Diabetes
- CD200R MAB AGONIST — Immunology
- ANGPTL3 siRNA — CVD
- PYY ANALOG — Diabetes
- O-GLCNACASE INH ★ — Alzheimer's
- N3PG Aβ MAB ★ — Alzheimer's
- KRAS G12C II — Cancer
- IDH1/2 INHIBITOR — Cancer
- GIP/GLP COAGONIST PEPTIDE — Diabetes
- AUR A KINASE INHIBITOR — Cancer
- RELAXIN-LA — Heart Failure
- OXYNTOMODULIN — Diabetes
- NOT DISCLOSED — Diabetes
- LP(a) INHIBITOR — CVD
- IL-17A SMALL MOL INHIBITOR — Immunology
- GIPR AGONIST LA — Diabetes
- BTLA MAB AGONIST — Immunology

PHASE 2

- TIRZEPATIDE — NASH
- IL-2 CONJUGATE — Ulcerative Colitis
- PIRTOBRUTINIB — B-Cell Malignancies
- GGG TRI-AGONIST — Obesity
- GBA1 GENE THERAPY ★ — Gaucher Disease Type 2
- TRPA1 ANTAGONIST — Pain
- GLP-1R NPA — Obesity
- SSTR4 AGONIST — Pain
- GLP-1R NPA — Diabetes
- PACAP38 MAB — Migraine
- PD-1 MAB AGONIST — Rheumatoid Arthritis
- IL-2 CONJUGATE — Systemic Lupus Erythematosus
- MEVIDALEN — Symptomatic LBD
- GBA1 GENE THERAPY ★ — Parkinson's Disease
- GRN GENE THERAPY ★ — Frontotemporal Dementia
- EPIREG/TGFα MAB — Chronic Pain
- GGG TRI-AGONIST — Diabetes
- BEBTELOVIMAB (LY-CoV1404 MAB) — COVID-19
- CXCR1/2L MAB — Hidradenitis Suppurativa
- AUTOMATED INSULIN DELIVERY SYS — Diabetes
- BASAL INSULIN-FC — Diabetes

PHASE 3

- PIRTOBRUTINIB — R/R CLL Combination
- TIRZEPATIDE — CV Outcomes
- TIRZEPATIDE — Obesity
- SELPERCATINIB — 1L NSCLC
- TIRZEPATIDE — Heart Failure pEF
- PIRTOBRUTINIB — R/R MCL Monotherapy
- SELPERCATINIB — 1L Med Thyroid Cancer
- EMPAGLIFLOZIN* — Post MI
- MIRIKIZUMAB — Crohn's Disease
- BARICITINIB — Systemic Lupus Erythematosus
- EMPAGLIFLOZIN* — Chronic Kidney Disease
- ABEMACICLIB — HER2+ Early BC
- ABEMACICLIB — Prostate Cancer
- SOLANEZUMAB ★ — Preclinical AD
- IMLUNESTRANT — ER+ HER2- mBC
- MIRIKIZUMAB — Ulcerative Colitis
- PIRTOBRUTINIB — R/R CLL Monotherapy
- DONANEMAB^ ★ — Early Alzheimer's
- LEBRIKIZUMAB — Atopic Dermatitis

REG REVIEW

- EMPAGLIFLOZIN* — Heart Failure pEF
- CONNECTED CARE PREFILLED INSULIN PEN — Diabetes
- BARICITINIB — Alopecia Areata
- TIRZEPATIDE — Diabetes
- SINTILIMAB (US)* — NonSquam NSCLC 1L

LEGEND

- NME
- NILEX
- * *Commercial Collaboration*
- ^ *Rolling submission in the U.S. initiated*
- ☆ *Discussed in Dec 15 Investment Community Meeting*

Note: select pre-clinical assets listed, most of which were discussed at the Lilly Investment Community meeting on December 15, 2021; NME = new molecular entity; NILEX = new indication or line extension

NEUROSCIENCE SUMMARY

- Lilly is an established leader with a deep commitment to advance AD research
  - Our work on amyloid imaging transformed the field and enabled the current generation of plaque-clearing antibodies
  - Our work on tau imaging raised the bar on staging AD pathology and supported our successful results in TRAILBLAZER-ALZ
  - Our ongoing work on plasma biomarkers, particularly pTau217, may enable new advances in diagnosis and disease monitoring for next generation of drug development
- Donanemab is well positioned to be a differentiated solution for patients, if approved
  - Deep and rapid amyloid removal associated with statistically significant slowing in primary endpoint
  - Multiple pathology-related biomarkers support limited dosing regimen
- Lilly is rapidly moving beyond anti-amyloid medicines for AD with novel targets using next generation platform modalities, both for neurodegeneration and pain

MARK MINTUN, M.D.
President of Neuroscience Research and Development, and President of Avid Radiopharmaceuticals

DAN SKOVRONSKY, M.D., PH.D.
Chief Scientific and Medical Officer, and President of Lilly Research Laboratories

ANNE WHITE
President, Lilly Neuroscience

Lilly

2021 **INVESTMENT COMMUNITY MEETING**