



# CORPORATE OVERVIEW

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December 2025

# Forward-Looking Statements

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This overview contains forward-looking statements. These statements relate to, among other things, the sufficiency of our cash position to fund advancement of a broad pipeline; the continued advancement of our pipeline, and expected milestones in 2026, 2027, and beyond, including the expected timing of (i) completion of our ongoing Phase 1 clinical trial evaluating PRX019, (ii) completion of the ongoing Phase 2 clinical trial evaluating BMS-986446, (iii) completion of the Phase 3 clinical trial for prasinezumab, and (iv) completion of the Phase 3 clinical trial for coramitug; amounts we might receive under our partnerships and collaborations with Roche, BMS, and Novo Nordisk; our potential to advance, initiate, and complete IND enabling studies for our discovery and preclinical programs; the treatment potential, designs, proposed mechanisms of action, and potential administration of prasinezumab, coramitug, and BMS-986446; potential indications and attributes of epitopes and antibodies we have identified in our programs, including their potential for a best-in-class profile; and plans for ongoing and future clinical trials of prasinezumab, coramitug, BMS-986446, and PRX019. These statements are based on estimates, projections and assumptions that may prove not to be accurate, and actual results could differ materially from those anticipated due to known and unknown risks, uncertainties and other factors, including but not limited to those described in the “Risk Factors” sections of our Quarterly Report on Form 10-Q filed with the Securities and Exchange Commission (SEC) on November 6, 2025, and discussions of potential risks, uncertainties, and other important factors in our subsequent filings with the SEC. This overview is made as of December 1, 2025, and we undertake no obligation to update publicly any forward-looking statements contained in this overview as a result of new information, future events, or changes in our expectations.

# Our Mission Today



## We are Focused on Delivering Life-Saving Therapies...

...for unmet medical needs caused by  
**diseases of protein dysregulation**



# We are Addressing Devastating Proteinopathies Affecting Millions of Patients and Families Worldwide



## NEURODEGENERATIVE DISEASES



### Alzheimer's disease (AD)

**>80 million**

People worldwide living with early symptomatic AD<sup>1</sup>

**>315 million**

People worldwide living with presymptomatic AD<sup>1</sup>

**\$1 trillion**

In annual US healthcare costs by 2050 from AD and other dementias<sup>2</sup>



### Parkinson's disease (PD)

**>10 million**

People living with PD worldwide<sup>3</sup>

**Fastest increasing**

Neurodegenerative disease<sup>3</sup>

**\$52 billion**

In overall economic burden in the US<sup>3</sup>

## RARE PERIPHERAL AMYLOID



### Transthyretin amyloidosis (ATTR)

**450,000**

Estimated number of patients worldwide with wtATTR or ATTRv<sup>4-6</sup>

**2.08 years**

Median overall survival New York Heart Association class III patients with ATTR cardiomyopathy<sup>7,8</sup>

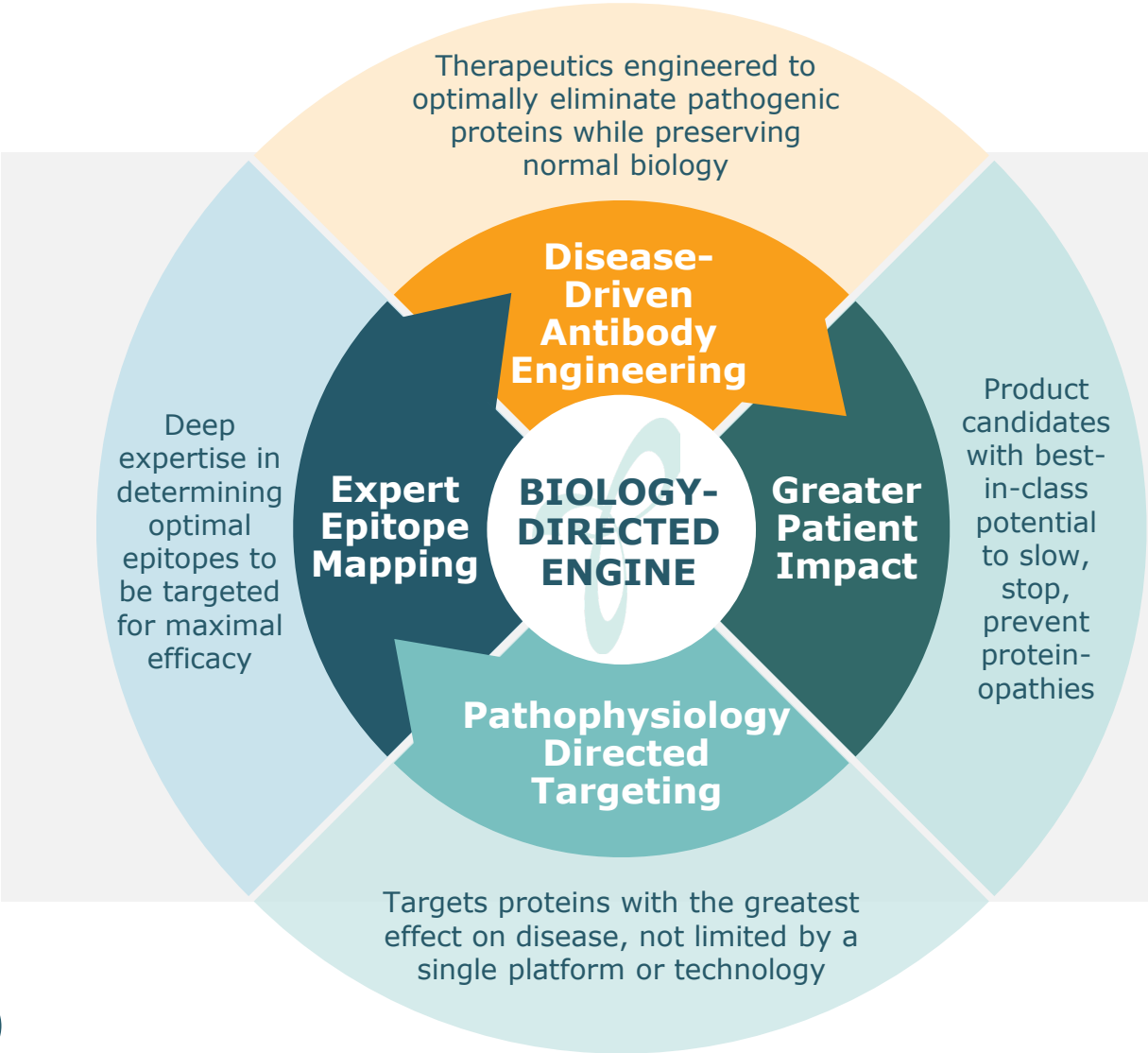
ATTRv: Hereditary amyloid transthyretin; wtATTR: Wild-type ATTR.

<sup>1</sup> Gustavsson, A. et al. "Global estimates on the number of persons across the Alzheimer's disease continuum." *Alzheimer's & Dementia* (2022) 1-13. <sup>2</sup> Long S, Benoit C, Weidner W. World Alzheimer Report 2023: Reducing dementia risk: never too early, never too late. London, England: Alzheimer's Disease International. Accessed July 18, 2024. <https://www.alzint.org/u/World-Alzheimer-Report-2023.pdf>. <sup>3</sup> Parkinson's Foundation. Understanding Parkinson's. Statistics. Accessed July 17, 2024. <https://www.parkinson.org/understanding-parkinsons/statistics>. <sup>4</sup> González-Duarte A, Conceição I, Amass L, Botteman MF, Carter JA, Stewart M. *Neurol Ther*. 2020;9(1):135-149.

<sup>5</sup> González-López E, Gagliardi C, Domínguez F, et al. *Eur Heart J*. 2017;38(24):1895-1904. <sup>6</sup> Tanskanen M, Peuralinna T, Polvikoski T, et al. *Ann Med*. 2008;40(3):232-239. <sup>7</sup> Kumar S, Dispenzieri A, Lacy MQ, et al. *J Clin Oncol*. 2012;30(9):989-995.

<sup>8</sup> Lane T, Fontana M, Martinez-Naharro A, et al. *Circulation*. 2019;140(1):16-26.

# Our Biology-Directed Engine Propels Prothena's Progress Across our Broad Pipeline



✓ **Multiple Clinical Programs**

- Two partnered Phase 3 programs
- One partnered Phase 2 program
- One partnered Phase 1 program

✓ **Strong Collaborations Established**

- Bristol Myers Squibb
- Novo Nordisk<sup>1</sup>
- Roche

<sup>1</sup> In July 2021 Novo Nordisk acquired coramitug (formerly PRX004) and broader ATTR amyloidosis program and gained full worldwide rights. Prothena is eligible to receive up to \$1.23 billion in total consideration.

# Active Clinical Development Pipeline

| PROGRAM<br>INDICATION (MODALITY)                                                                                                                    | PROTEIN<br>TARGET                   | DISCOVERY                                                     | PRE-CLINICAL | PHASE 1 | PHASE 2 | PHASE 3 | GLOBAL<br>PARTNER                                                                     |
|-----------------------------------------------------------------------------------------------------------------------------------------------------|-------------------------------------|---------------------------------------------------------------|--------------|---------|---------|---------|---------------------------------------------------------------------------------------|
| <b>Prasinezumab</b><br><i>Parkinson's disease</i><br>(mAb)                                                                                          | $\alpha$ -Synuclein<br>(C-terminus) | Phase 3 PARAISO trial to initiate by YE 2025<br>(NCT07174310) |              |         |         |         |    |
| <b>Coramitug (PRX004)</b><br><i>ATTR-CM</i><br>(mAb)               | Transthyretin<br>(misTTR)           | Phase 3 CLEOPATTRA trial initiated<br>(NCT07207811)           |              |         |         |         |    |
| <b>BMS-986446 (PRX005)</b><br><i>Alzheimer's disease</i><br>(mAb)  | Tau<br>(MTBR)                       | Phase 2 TargetTau-1 to complete in 1H 2027<br>(NCT06268886)   |              |         |         |         |    |
| <b>PRX019</b><br><i>Neurodegeneration</i><br>(mAb)                                                                                                  | Undisclosed<br>Target               | Phase 1 to complete in 2026                                   |              |         |         |         |  |

Neurodegenerative

Rare Peripheral

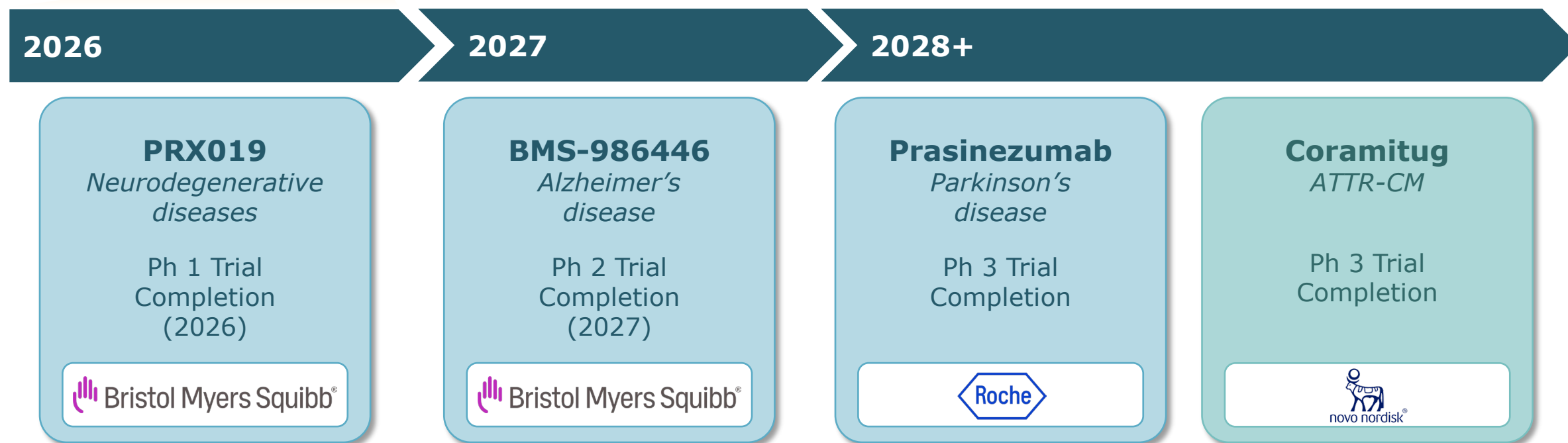
mAb: Monoclonal antibody.

<sup>1</sup> Orphan Drug Designation granted by FDA & EMA; <sup>2</sup> FDA Fast Track designation.

<sup>1</sup> In July 2021 Novo Nordisk acquired coramitug (formerly PRX004) and broader ATTR amyloidosis program and gained full worldwide rights. Prothena is eligible to receive up to \$1.23 billion in total consideration

# Upcoming Partner Milestones

★ **Up to \$105 Million in Clinical Milestones by YE 2026**  
**Related to the Advancement of Coramitug and PRX019<sup>1</sup>**



Neurodegenerative    Rare Peripheral

# Prothena Partnerships Expected to Generate Meaningful Value Across Programs



**Up to \$755 Million in Total Milestones + Royalties**

**Prasinezumab**  
*Parkinson's disease*

- ✓ **\$135 million** paid to date
- ❑ **\$620 million** remaining in regulatory and sales milestones<sup>1</sup>
- ❑ Up to double digit teen royalties
- ❑ US co-promote option



**Up to \$1.23 Billion in Total Milestones**

**Coramitug (PRX004)**  
*ATTR-CM*

- ✓ **\$100 million** paid to date
- ❑ **\$1.13 billion** remaining in clinical, regulatory, and sales milestones



**Up to \$1.55 Billion in Total Milestones + Royalties Across Two Clinical Stage Programs**

✓ Upfront Payment + Equity: \$150 million<sup>2</sup>

**BMS-986446**  
*Alzheimer's disease*

- ✓ **\$135 million** paid to date
- ❑ **\$562.5 million** remaining in regulatory and sales milestones
- ❑ Up to high teens royalties on a weighted average basis

**PRX019**  
*Neurodegenerative diseases*

- ✓ **\$80 million** paid to date
- ❑ **\$617.5 million** remaining in clinical, regulatory, and sales milestones
- ❑ Up to high teens royalties on a weighted average basis

Neurodegenerative

Rare Peripheral

<sup>1</sup> Includes \$5 million clinical milestone payment for an indication outside of Parkinson's Disease.

<sup>2</sup> Bristol Myers Squibb owns approximately 2.2% of Prothena's outstanding shares as of March 3, 2025 ("record date")

# **Prasinezumab**

## **Parkinson's Disease**

### **Phase 3**

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Worldwide Collaboration with Roche

# Prasinezumab: Potential First-in-Class Treatment for Parkinson's Disease



## Prasinezumab *Parkinson's Disease*

Status: Phase 3 initiation by YE 2025

### Anti- $\alpha$ -synuclein Antibody

- Preferentially binds to aggregated  $\alpha$ -synuclein, designed to reduce pathogenic spread and decrease synuclein pathology<sup>1</sup>

### Rapidly Growing Parkinson's Patient Population

- 10 million patients globally<sup>2</sup>
- Fastest increasing neurodegenerative disease<sup>3</sup>

### Worldwide Collaboration with Roche

- ✓ \$135 million paid-to-date
- Up to \$620 million in additional milestones
- Up to double-digit teen royalties
- US co-promote option
- Roche: peaks sales potential >\$3.5B (unadjusted)<sup>4</sup>

### Phase 3 Clinical Program:

- Roche to initiate Phase 3 PARAISO trial (NCT07174310) in ~900 participants with early-stage Parkinson's disease on stable symptomatic levodopa monotherapy by YE 2025
- Primary endpoint: Time to confirmed motor progression event on MDS-UPDRS Part III score up to at least 2 years

### Phase 2 Clinical Program Results:

- First anti- $\alpha$ -synuclein antibody to slow progression on measures of PD
- Consistent positive trends across multiple endpoints observed in both Phase 2b PADOVA (NCT04777331) and Phase 2 PASADENA (NCT03100149) trials
- More pronounced effect observed in population treated with levodopa in Phase 2b PADOVA (~75% of participants)
- PADOVA trial provided the first biomarker evidence of prasinezumab impacting the underlying disease biology
- Open-label extension studies from both PADOVA and PASADENA ongoing to explore the observed effects

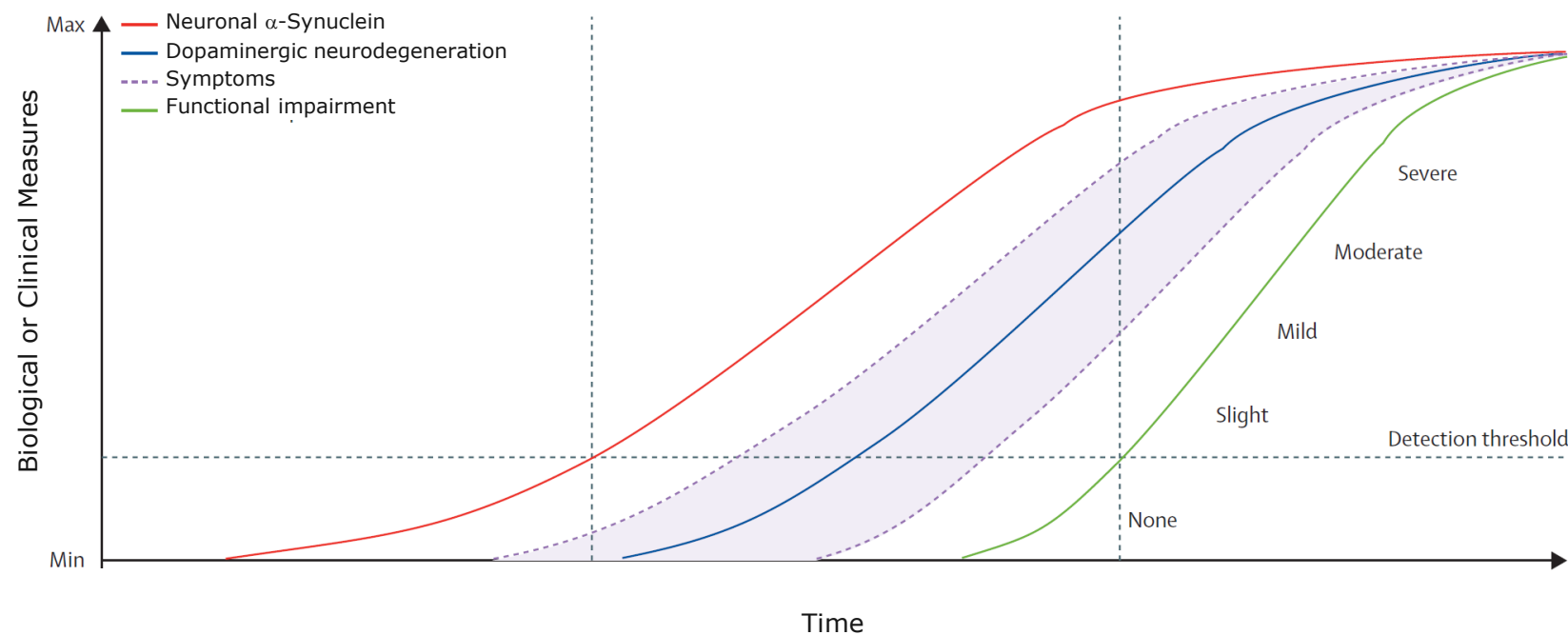
MDS-UPDRS: Movement Disorder Society – Unified Parkinson's Disease Rating Scale.

<sup>1</sup> Jankovic J et al. *JAMA Neurol.* 2018; 75:1206–1214; <sup>2</sup> Parkinson's Foundation. Understanding Parkinson's. Statistics; <sup>3</sup> GBD 2015 Neurological Disorders Collaborator Group (2017) Global, regional, and national burden of neurological disorders during 1990–2015: a systematic analysis for the Global Burden of Disease Study 2015. *Lancet Neurol* 16, 877–897. <sup>4</sup> Roche's Pharma Day 2025 Presentation on September 22, 2025; not a milestone or royalty projection. Roche states >3B CHF in their 9/22/2025 Pharma Day presentation; conversion rate of 1 CHF = 1.26 USD (as of October 22, 2025) equates to ~3.77B USD.

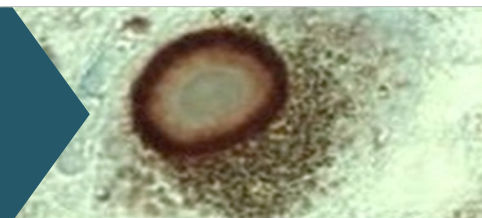
# $\alpha$ -Synuclein Pathology is Strongly Implicated in Parkinson's Disease

Accumulation of  $\alpha$ -Synuclein is a predominant neuropathological feature and follows the topological progression of disease

Genetically validated target with evidence favoring a prominent role for  $\alpha$ -Synuclein in early PD: missense mutations and duplication/triplication



$\alpha$ -Synuclein is the predominant component of Lewy bodies found in Parkinson's disease and other synucleinopathies

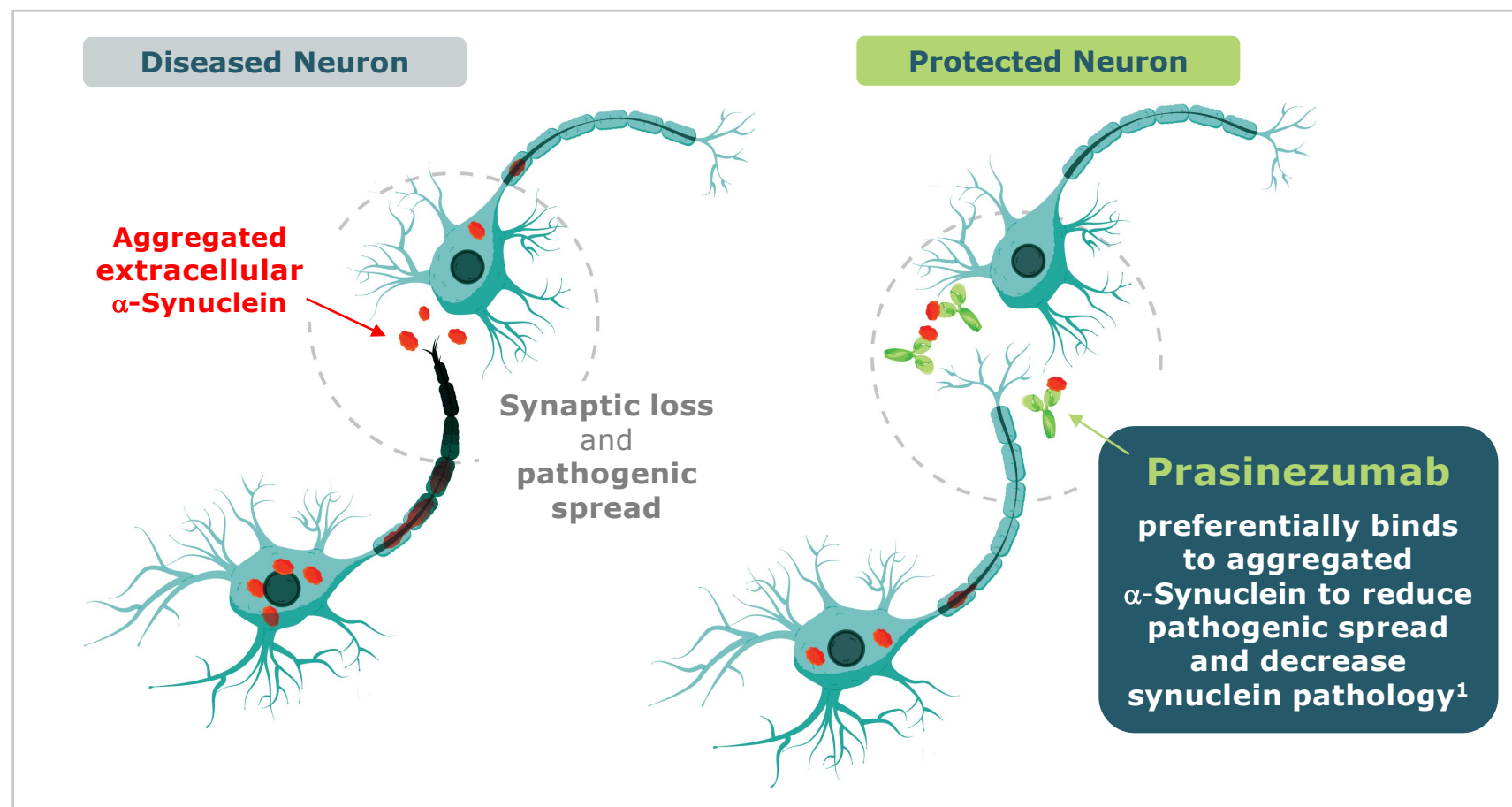


# Prasinezumab: $\alpha$ -Synuclein Immunotherapy

REDUCE NEURONAL TOXICITY AND PREVENT CELL-TO-CELL TRANSMISSION<sup>1</sup>

## $\alpha$ -Synuclein as an extracellular target during pathogenesis

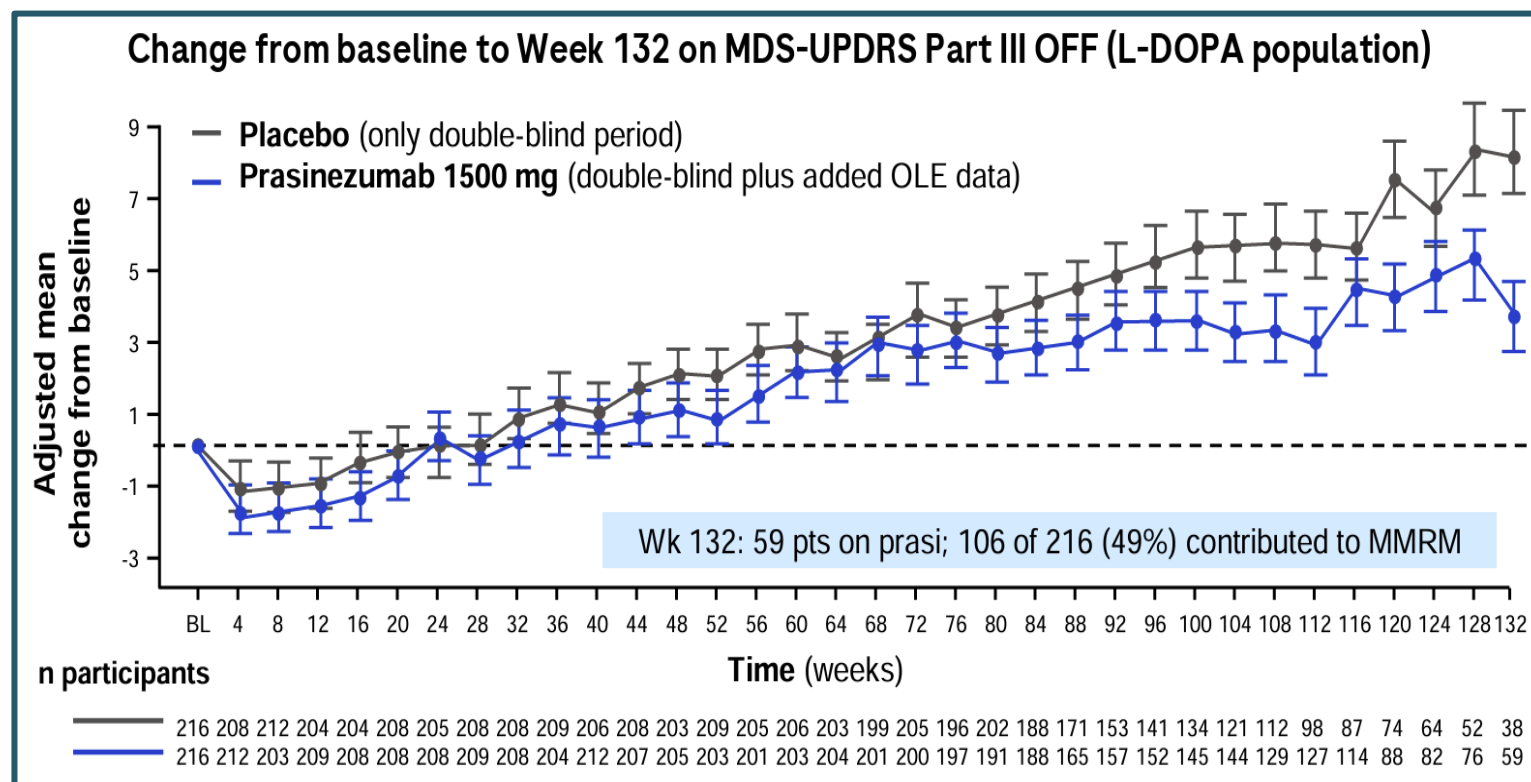
- Caudal-rostral staging, host-to-graft transfer, various propagation models



# Moving into Phase 3 in Parkinson's Disease

PHASE 2B (PADOVA) AND LONGER FOLLOW-UP DATA SUGGEST CLINICAL BENEFIT IN DELAY OF CONFIRMED MOTOR PROGRESSION

## Phase 2b (PADOVA) 2.5 years results<sup>1</sup>



- Multiple endpoints from the PADOVA and OLE study suggest potential clinical benefit of prasinezumab; more pronounced effect in L-DOPA treated pts (~75% of population)
- Positive trends towards reduced motor progression sustained at 2.5 years (incl. OLE data)
- PASADENA and PADOVA OLE studies continuing with high retention / rollover
- Phase 3 to initiate by YE 2025

# Roche's Phase 3 Go Decision Based on Meeting the Bar Criteria

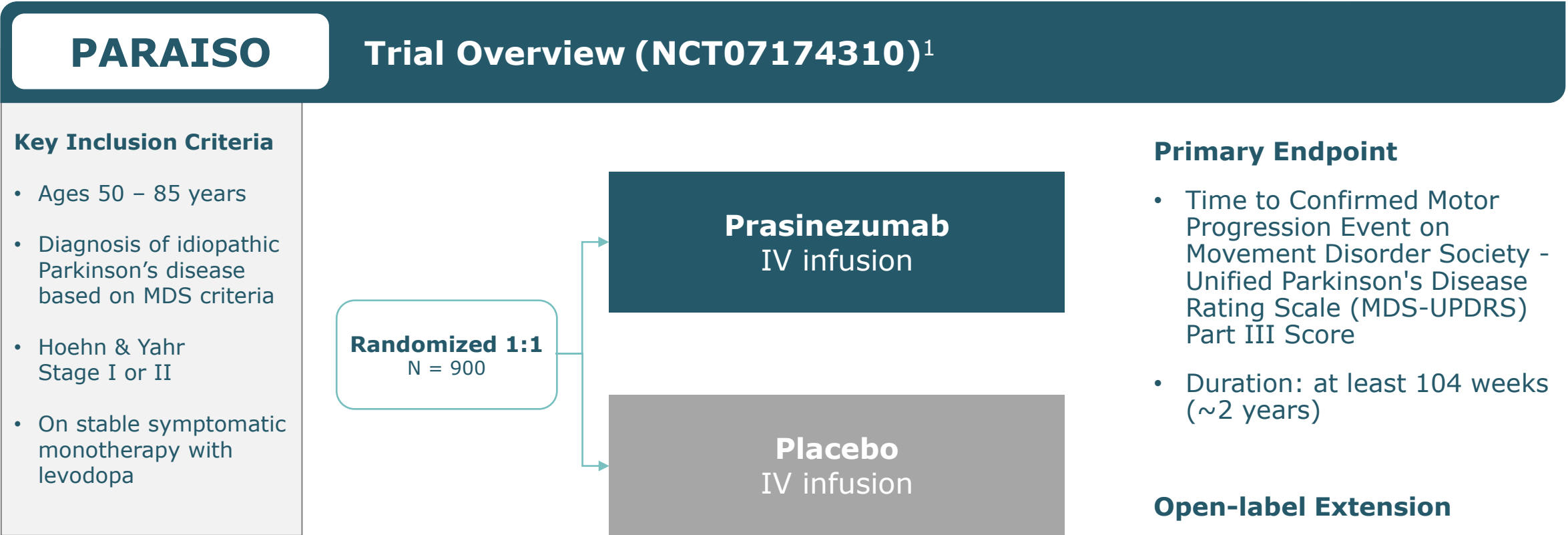


MULTIPLE ENDPOINTS FROM PHASE 2 STUDIES (PASADENA & PADOVA) AND OLE SUGGEST POTENTIAL TO DELAY MOTOR PROGRESSION

| The Bar                                                                     | Prasinezumab                                                                                                                                                                                                            |
|--------------------------------------------------------------------------------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
|  <b>Answers a clear &amp; addressable unmet need</b>                        | <ul style="list-style-type: none"> <li>&gt;10m Parkinson's disease patients globally; no approved disease modifying therapy to slow/stop progression</li> </ul>                                                         |
|  <b>Engages a 'foundational target'</b>                                     | <ul style="list-style-type: none"> <li><math>\alpha</math>-synuclein is a known biological driver of PD progression, as supported by preclinical data and Ph II clinical studies (e.g., PADOVA and PASADENA)</li> </ul> |
|  <b>Possesses worthy pharmacologic &amp; developability characteristics</b> | <ul style="list-style-type: none"> <li>Potentially first in class anti-<math>\alpha</math>-synuclein antibody</li> <li>Favorable safety and tolerability profile (PADOVA and PASADENA)</li> </ul>                       |
|  <b>Achieves meaningful therapeutic differentiation</b>                    | <ul style="list-style-type: none"> <li>Evidence of delayed motor progression</li> <li>Effect on top of effective symptomatics, i.e. L-DOPA (PADOVA)</li> </ul>                                                          |
|  <b>Unlocks a path to value</b>                                           | <ul style="list-style-type: none"> <li>Peak sales potential &gt;\$3.5B (unadjusted)*</li> </ul>                                                                                                                         |

OLE: Open label extension; PD: Parkinson's disease.  
 Slide adapted from Roche's Pharma Day 2025 Presentation on September 22, 2025.  
 \* Roche states >3B CHF in their 9/22/2025 Pharma Day presentation; conversion rate of 1 CHF = 1.26 USD (as of October 22, 2025) equates to ~3.77B USD

# Prasinezumab: PARAISO Phase 3 Trial Design



# **Coramitug (formerly PRX004)**

## **ATTR Amyloidosis with Cardiomyopathy**

### **Phase 3**

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ATTR Business Acquired by Novo Nordisk

# Coramitug (formerly PRX004): Potential First-in-Class Treatment for ATTR-CM



## Coramitug

*ATTR amyloidosis with  
cardiomyopathy (ATTR-CM)*

Status: Phase 3

### Differentiated Depletor Mechanism of Action<sup>1</sup>

- Designed to inhibit fibril formation and specifically bind to pathogenic TTR
- Uniquely designed for patients at high risk of early mortality due to amyloid deposition

### Worldwide Collaboration with Novo Nordisk

- ✓ \$100 million paid-to-date
- \$1.13 billion in potential additional milestones
- Potential blockbuster therapy

### Phase 3 Clinical Program:

- Novo Nordisk initiated the Phase 3 CLEOPATTRA trial (NCT07207811) in ~1280 participants with ATTR-CM
- NYHA Class I-IV patients randomized 1:1 to coramitug with continued SOC vs. placebo with continued SOC
- Primary endpoint: Number of occurrences of composite endpoint of CV deaths and recurrent CV events (CV hospitalizations and urgent heart failure visits) up to ~4 years

### Phase 2 Signal Detection Trial:

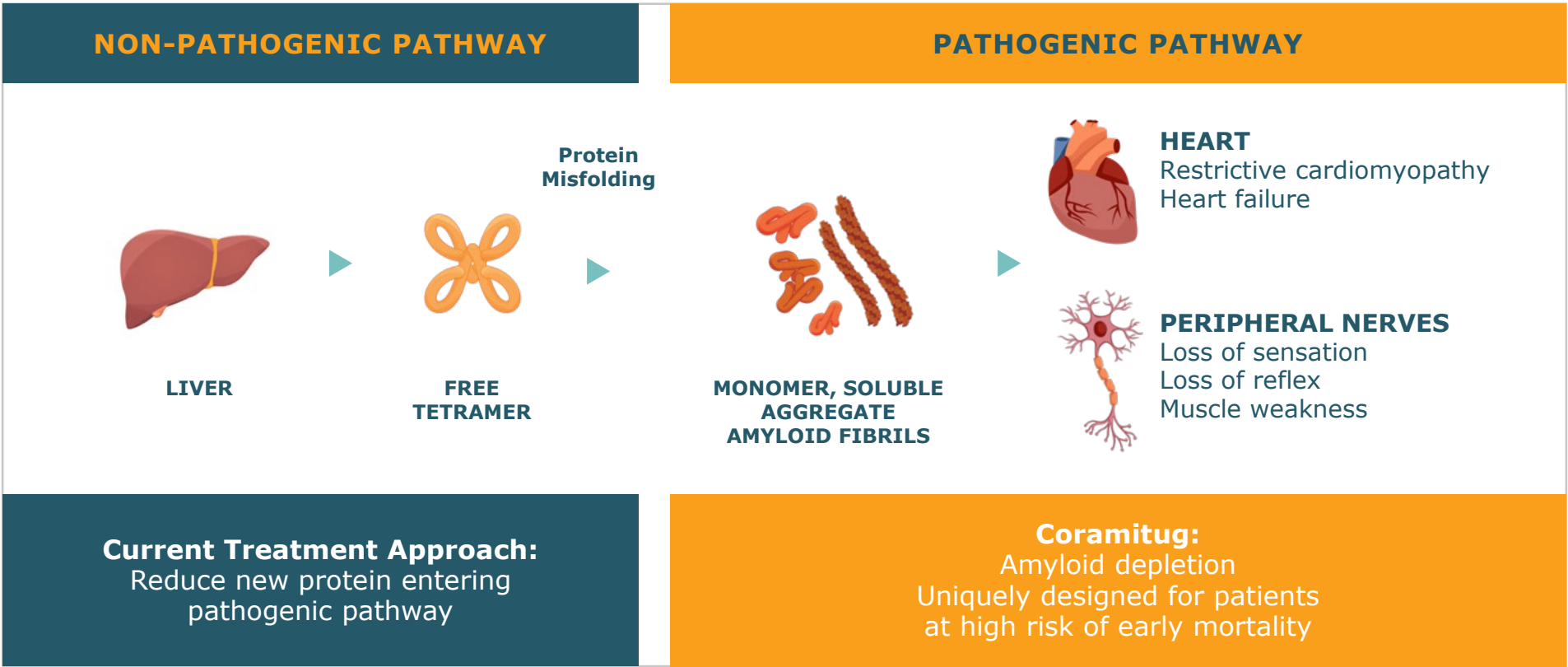
- Phase 2 trial in 105 ATTR-CM patients (NCT05442047)
- Participants received IV infusion Q4W of 10 mg/kg or 60 mg/kg of coramitug or placebo added to SOC
- Co-primary Endpoints: Change from baseline in NT-proBNP levels AND 6MWT at 52 weeks
- Results support Phase 3 development and were shared in a late-breaker presentation at the AHA Scientific Sessions and published in *Circulation*<sup>2</sup>

CV: Cardiovascular; SOC: Standard of care.

<sup>1</sup> Preclinical studies of mPRX004, the murine form of PRX004; Higaki JN et al. *Amyloid*, 2016; Suhr OB et al., *Amyloid*, 2025

<sup>2</sup> Fontana M et al. *Circulation*, 2025; <https://doi.org/10.1161/CIRCULATIONAHA.125.077304>

# Differentiated Mechanism for ATTR-CM



**Coramitug: Depleter MoA May Provide a New Treatment Paradigm for Patients at High-risk of Early Mortality Due to Amyloid Deposition**

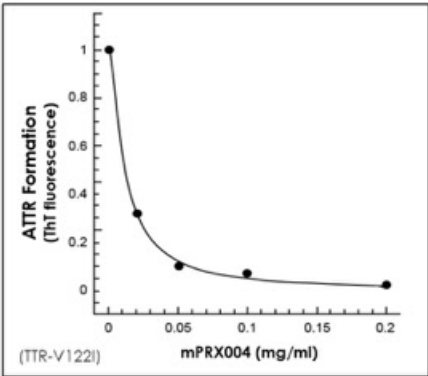
# Designed to Deplete Amyloid

## SUMMARY OF PRECLINICAL EFFECTS OF mPRX004

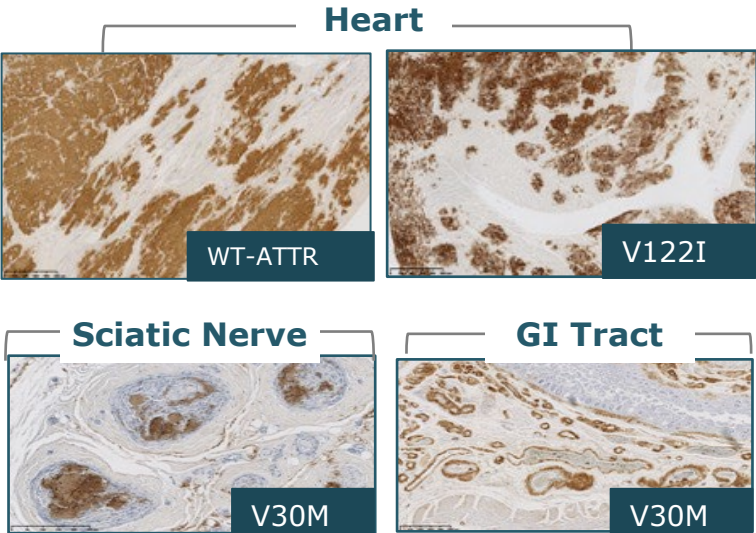
**mPRX004 (murine form of PRX004) preclinical results:**<sup>1</sup>

- ✓ Inhibits fibril formation
- ✓ Specifically binds to pathogenic TTR
- ✓ Reacts to amyloid deposits in multiple organs in both wtATTR and ATTRv patients
- ✓ Promotes in vivo ATTR amyloid clearance

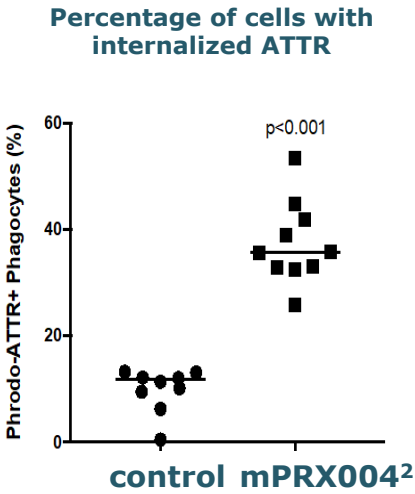
### Inhibition of amyloid formation



### Specific binding to amyloid



### Clearance of amyloid



# Phase 2 Results Support Advancement to Phase 3

## PH 2 DATA PRESENTED AT LATE-BREAKING SESSION AT AHA 2025 AND PUBLISHED IN *CIRCULATION*

- ✓ Treatment with coramitug 60 mg/kg resulted in a statistically significant reduction in NT-proBNP levels compared with placebo (**-48%; p=0.0017**) over 52 weeks with a reduction from baseline
  - Notably, favorable reduction was achieved on top of background use of standard-of-care TTR stabilizers in most patients (80% in the coramitug 60 mg/kg group and 85.7% in the placebo group)
- ✓ Coramitug 60 mg/kg showed an estimated treatment difference of 13.45 m in 6MWT vs. placebo
  - Statistical significance not achieved, potentially due to the small sample size and relatively short study duration
- ✓ Across a wide range of echocardiographic parameters - including measures of LV and RV systolic function, diastolic function, and estimated pulmonary arterial pressures - coramitug 60 mg/kg was associated with improvements versus placebo, suggestive of favorable cardiac remodeling
- ✓ Coramitug demonstrated an acceptable safety profile and was generally well tolerated

**Coramitug for ATTR-CM represents a potential multi-billion dollar global market opportunity**

LV: Left Ventricle; RV: Right Ventricle; TTR: Transthyretin.  
Phase 2 Results Presented During Late-Breaking Session of The American Heart Association Scientific Sessions 2025; November 10, 2025; New Orleans, LA  
Phase 2 Results Published in November 10, 2025 Edition of *Circulation*: <https://doi.org/10.1161/CIRCULATIONAHA.125.077304>

# Coramitug: CLEOPATTRA Phase 3 Trial Design

## CLEOPATTRA

## Trial Overview (NCT07207811)<sup>1</sup>

### Key Inclusion Criteria

- 18 years and older
- Diagnosis of ATTR-CM<sup>2</sup>
- Demonstrated cardiac amyloid infiltration
- Increased LV wall thickness  $\geq 12$  mm
- Chronic heart failure (NYHA Class I – IV) requiring ongoing treatment with a loop diuretic

**Randomized 1:1**  
N = 1280

**Coramitug**  
IV infusion

**Placebo**  
IV infusion

### Primary Endpoint

- Number of occurrences of composite endpoint of CV deaths and recurrent CV events (CV hospitalizations and urgent heart failure visits) from baseline to end of study
- Duration: up to ~4 years

Everyone in the study will continue receiving their usual heart treatments as recommended by their doctor

CV: Cardiovascular; NYHA: New York Heart Association.  
<sup>1</sup> Trial design slide adapted from information available on posting on clinicaltrials.gov.  
<sup>2</sup> ATTR-CM includes wild-type ATTR (ATTRwt) or variant ATTR (ATTRv); target ATTRv resuitment is ~15% of the study population.

# **BMS-986446 (formerly PRX005)**

## **Alzheimer's Disease**

### **Phase 2**

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Global Neuroscience Collaboration  
with Bristol Myers Squibb

Bristol Myers Squibb®

**BMS-986446**  
*Alzheimer’s disease*

Status: Phase 2, completion 2027

Anti-Tau Mechanism of Action

- Designed to specifically bind with high affinity to a key epitope within the microtubule binding region (MTBR) of tau, a protein implicated in the causal pathophysiology of Alzheimer’s disease

Global Rights Deal for BMS-986446<sup>1</sup>

- ✓ \$135 million paid-to-date for global rights
- BMS funds all development and commercialization
- Up to \$562.5 million in regulatory/sales milestones
- Up to high teens royalties on a weighted average basis
- Potential blockbuster therapy

Phase 2 Trial (NCT06268886): Ongoing

- Global, double-blind, placebo-controlled
- 310 participants with early AD
- Randomized, 3 arms (two doses and placebo)
- Primary Endpoint: Change from baseline in brain tau deposition as measured by PET at week 76
- Secondary endpoints include change from baseline in CDR-SB score at week 76
- Primary completion expected in 2027

Phase 1 Trial (NCT06955741): Ongoing

- Open-label single-dose study to assess PK, tolerability, and bioavailability of subcutaneous administration
- Primary completion expected in 2H 2025

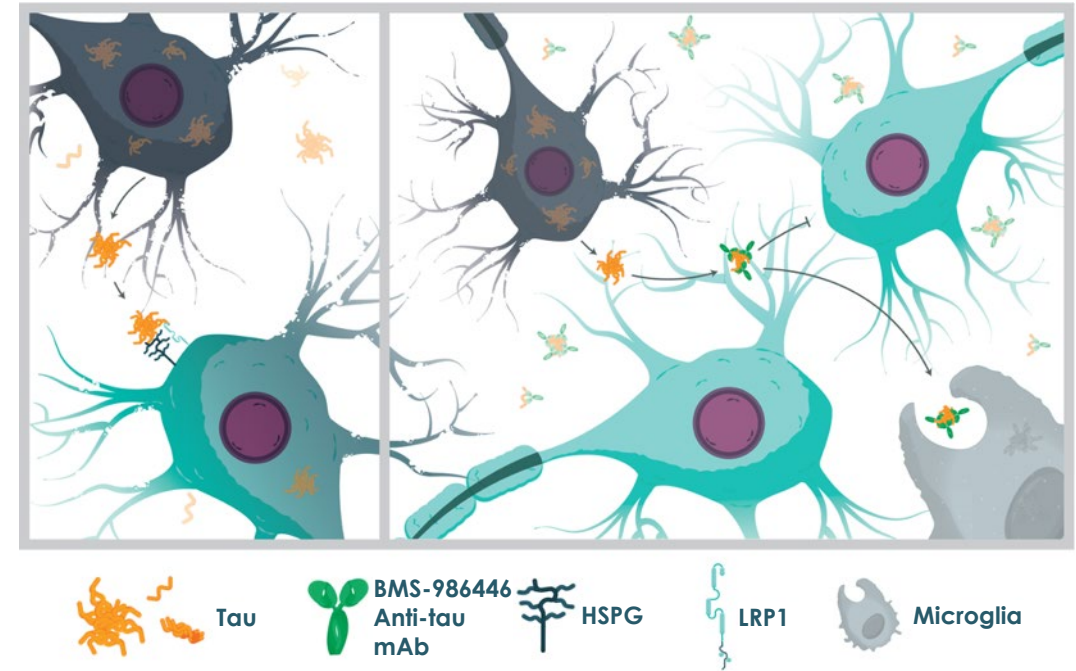
# BMS-986446: MTBR-Specific Anti-Tau Antibody

## BMS-986446, a differentiated tau antibody that targets an optimal tau region within the MTBR

- Recent publications strongly suggest that tau appears to spread throughout the brain via synaptically-connected pathways<sup>1</sup>
- This propagation of pathology is thought to be mediated by tau "seeds" containing the MTBR of tau<sup>2</sup>

## Potential for best-in-class efficacy

- Preclinical evaluation of our antibodies in our AD models demonstrated that MTBR-specific antibodies are superior to non-MTBR tau antibodies in blocking tau uptake and neurotoxicity
- Demonstrated significant inhibition of cell-to-cell transmission and neuronal internalization in vitro and in vivo and slowed pathological progression in a tau transgenic mouse model



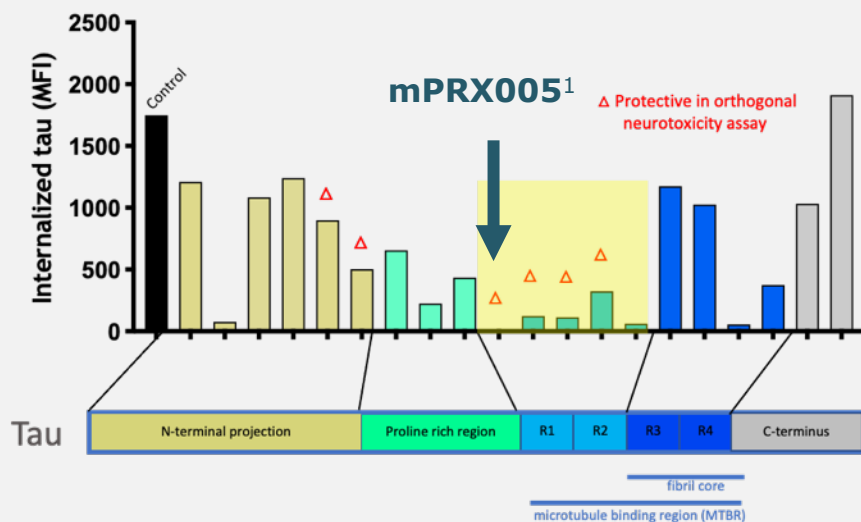
**BMS-986446: Potential Best-in-Class MTBR-Specific Anti-Tau Antibody to Reduce Pathogenic Tau Spread**

<sup>1</sup> Ahmed, Z., Cooper, J., et al. A novel in vivo model of tau propagation with rapid and progressive neurofibrillary tangle pathology: the pattern of spread is determined by connectivity, not proximity. 2014 *Acta Neuropathol*;

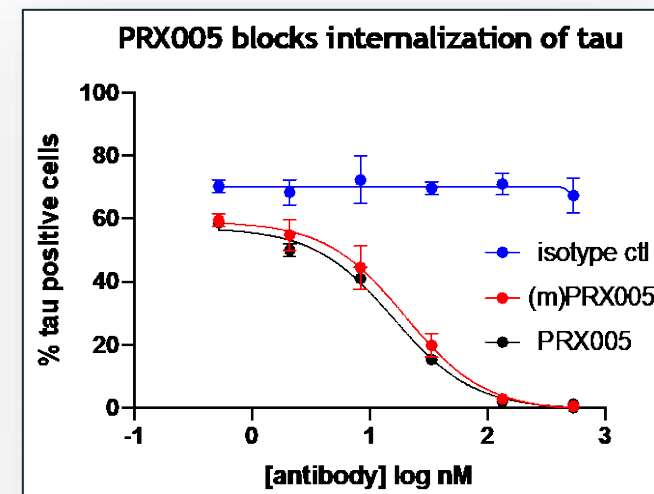
<sup>2</sup> Horie, K., Barthelemy, H., Bateman, R.J. CSF tau microtubule binding region identifies tau tangle and clinical stages of Alzheimer's disease. 2021 *Brain*

# BMS-986446 (PRX005): Superior in Blocking Cellular Internalization of Tau and Downstream Neurotoxicity

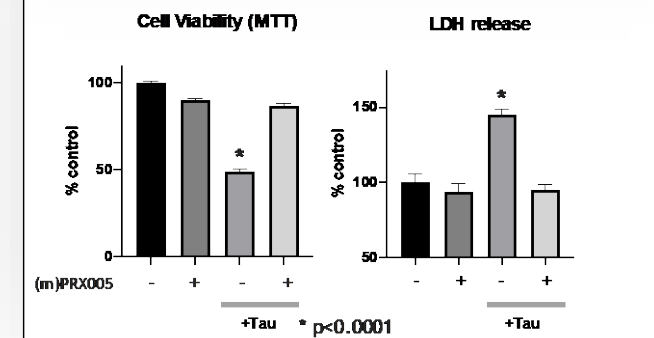
Repeats 1 and 2 defined as the strongest inhibitory regions in screening with cellular internalization and neurotoxicity assays



- Panel of Prothena antibodies targeted throughout the tau molecule were screened for optimal affinity and epitope
- These were tested *in vitro* for their ability to block internalization and toxicity



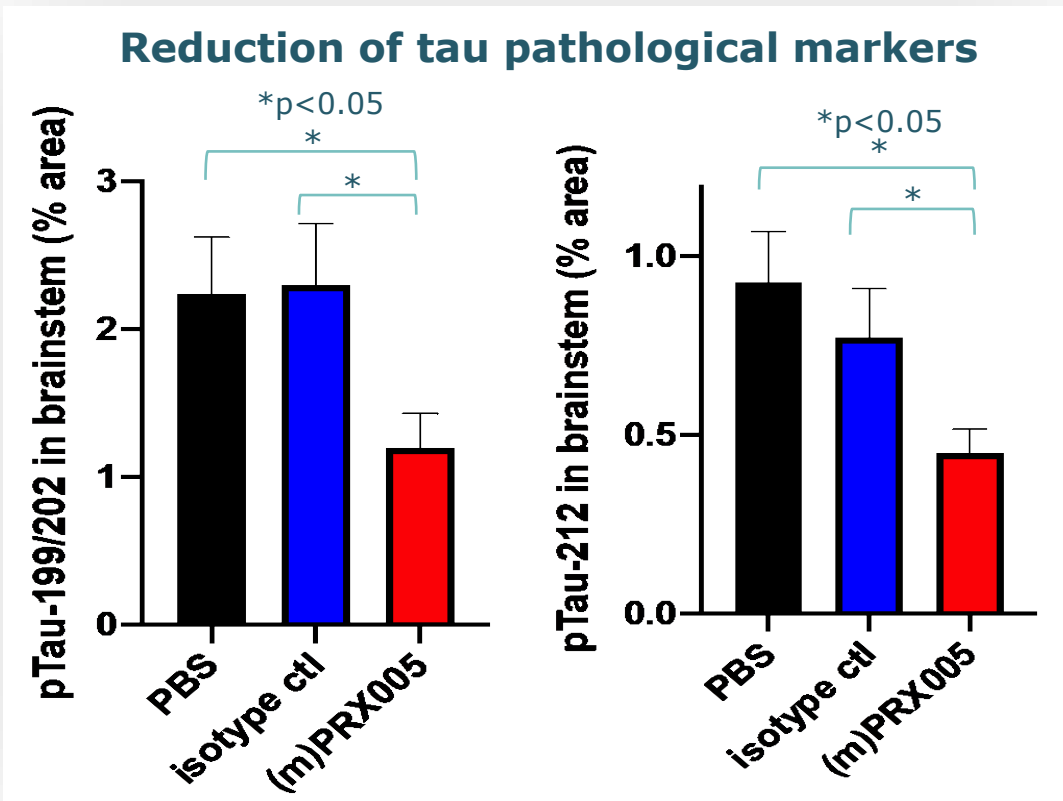
(m)PRX005 protects rodent primary cortical neurons from tau-induced toxicity



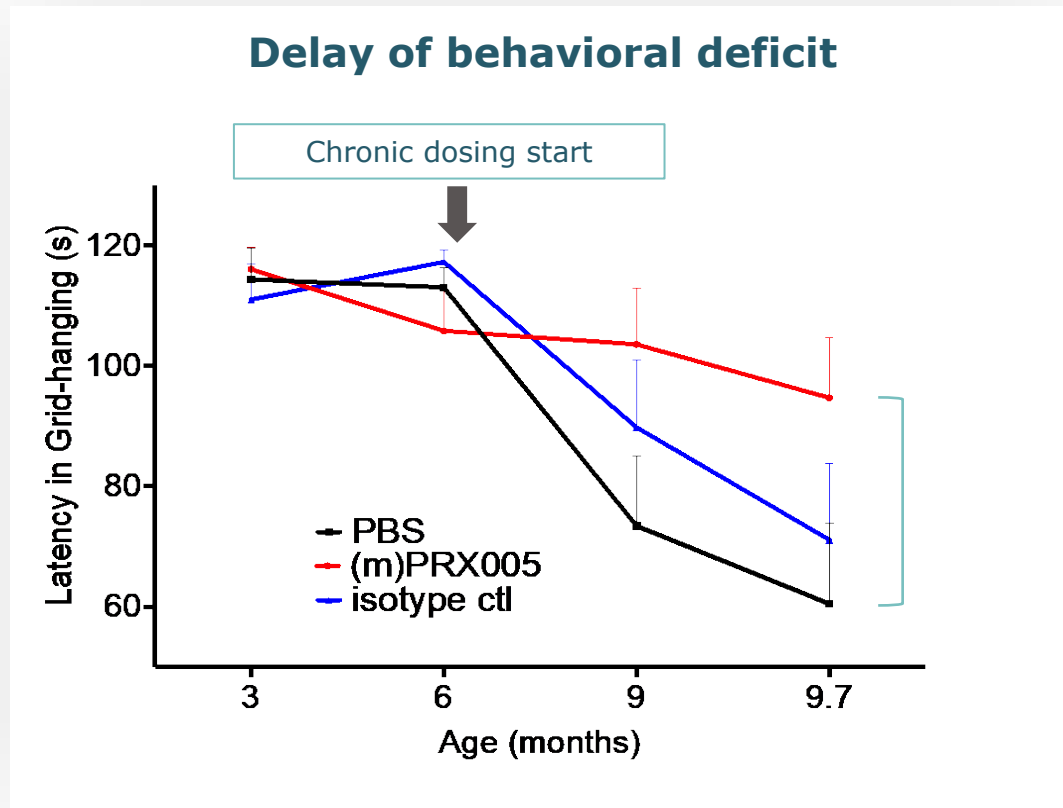
<sup>1</sup> (m)PRX005: murine form of PRX005 (BMS-986446)

# (m)PRX005 Reduces Pathological Tau and Ameliorates Behavioral Deficit in Transgenic Tau Mouse Model

All values are mean ± SE (n=15-20)

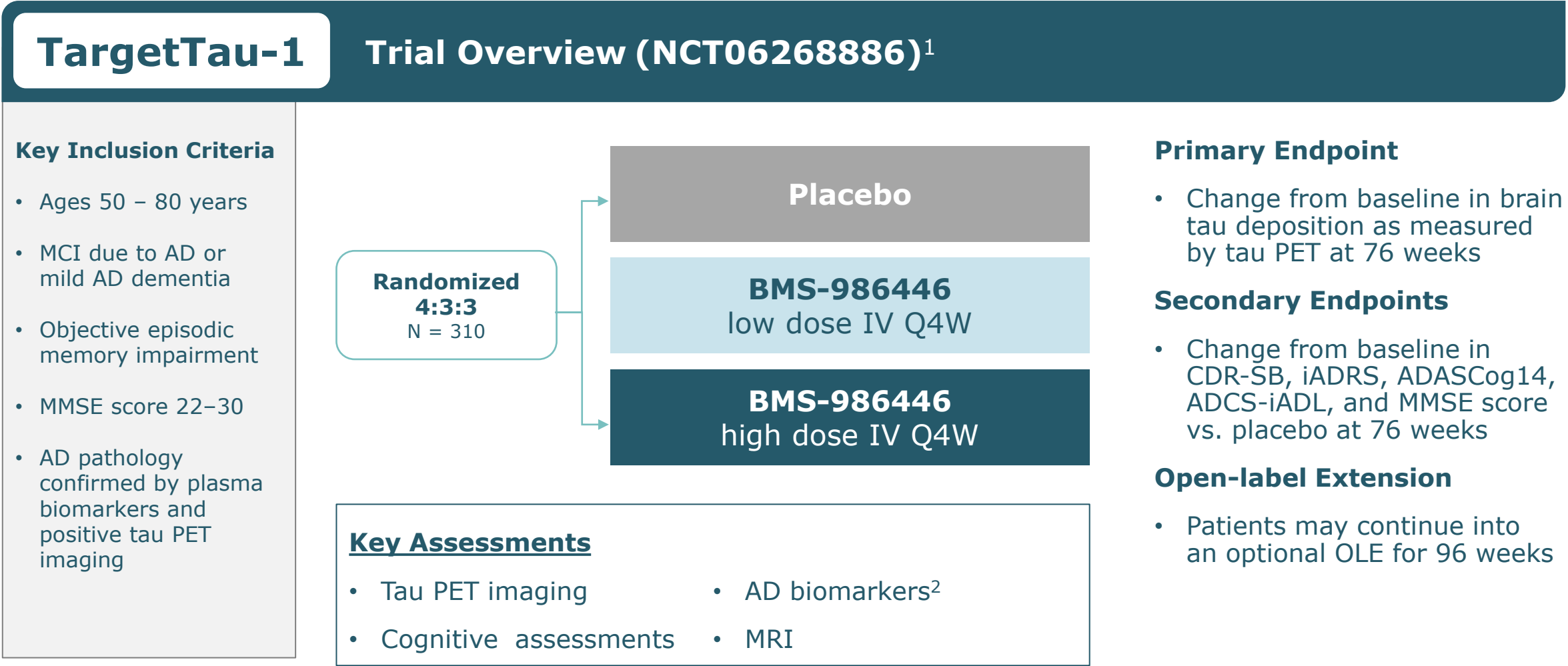


- PS19 transgenic mice overexpressing tau mutation (P301S) cause high levels of neuronal tau pathology and resultant behavioral deficits



- Initiation of treatment (weekly i.p.) at the onset of pathological development (treatment mode) with (m)PRX005 delays brainstem tau pathology and consequent behavioral deficits

# BMS-986446: TargetTau-1 Phase 2 Trial Design



AD: Alzheimer’s disease; ADAS-Cog14: 14-item Alzheimer’s Disease Assessment Scale-Cognitive Subscale; ADCS-iADL: Alzheimer’s Disease Cooperative Study-Instrumental Activities of Daily Living Scale; CDR-SB: Clinical Dementia Rating Scale Sum of Boxes; iADRS: Integrated Alzheimer’s Disease Rating Scale; IV: Intravenous; MMSE: Mini-Mental State Examination; MRI: Magnetic resonance imaging; OLE: Open label extension; PET: Positron Emission Tomography; Q4W: Every 4 weeks.  
<sup>1</sup> Presented at: The 2024 AAIC Annual Meeting; July 28, 2024; Philadelphia, PA. Clinicaltrials.gov.  
<sup>2</sup> Standard bFluid-based biomarkers include total tau, p181tau, p217tau, Aβ [1-42], Aβ [1-40], neurofilament light chain, and glial fibrillary acidic protein.

# **PRX019**

## **Neurodegenerative Diseases**

### **Phase 1**

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Global Neuroscience Collaboration  
with Bristol Myers Squibb

# PRX019: Potential Treatment of Neurodegenerative Diseases



## PRX019

### *Neurodegenerative diseases*

Status: Phase 1, completion 2026

#### Global Rights Deal for PRX019<sup>1</sup>

- ✓ \$80 million paid-to-date for global rights
- Up to \$617.5 million remaining in clinical, regulatory, and sales milestones
- Up to high teens royalties on a weighted average basis

#### Phase 1 Trial: Ongoing

- Phase 1 clinical trial being conducted by Prothena
- Single ascending dose and multiple ascending dose in healthy adults
- Phase 1 trial to evaluate:
  - Safety
  - Tolerability
  - Immunogenicity
  - Pharmacokinetics
- Trial expected to complete in 2026

**Potential Clinical Milestone Could be Achieved at Time of BMS Decision to Further Develop PRX019; Expected by YE 2026**