Investor Update

NASDAQ: ACIU | Investor Presentation - March 2024

AC Immune

Version: 14.03.2024

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AC Immune at a glance

Pioneering new ways to treat neurodegenerative diseases

Broad, diverse pipeline – 16 programs
1 Phase 3 program and 5 in Phase 2

Key differentiation: Precision Medicine
Integrates therapeutics and diagnostics

Multiple global partnerships
>CHF 2.5 billion in potential milestones

Clinically validated technology platforms
Best-in-class small molecules and biologics

Cash reserves on Balance sheet
Funding into 2026[3]

- Based in Lausanne, Switzerland
- ~150 employees
- Listed September 2016 (NASDAQ: ACIU)
- 98.8 million shares outstanding[1]
- Cash of CHF103 million[2]

  plus CHF15 million milestone (received Feb 1, 2024)

(1) As of December 31, 2023; excluding treasury shares; (2) as of December 31, 2023; (3) assumes second ACI-35-related milestone payment of CHF25 million received in 2025 and no other milestones

Neurodegenerative diseases represent a large and growing market

Prevention the best avenue to long-term preservation of cognition and function.

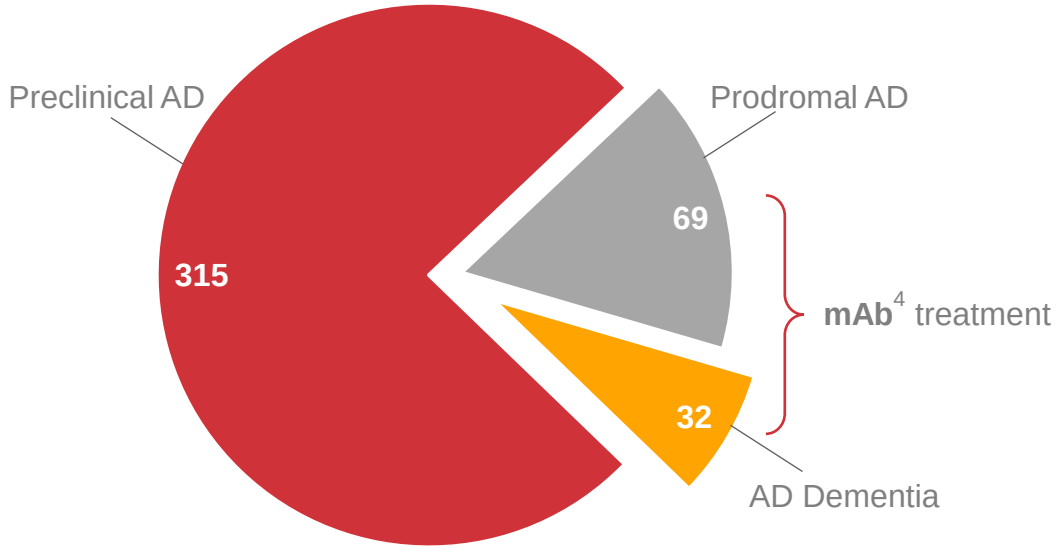
>$1 Trillion global annual cost of dementia[1]

>90 million with Alzheimer's disease globally[2]

>300 million with preclinical AD[3] at risk of disease

>400 million people addressable by active immunotherapy

Global Prevalence of AD Stages[2] (million)



Diagnostics enable earlier intervention

Active immunotherapy for treatment and prevention

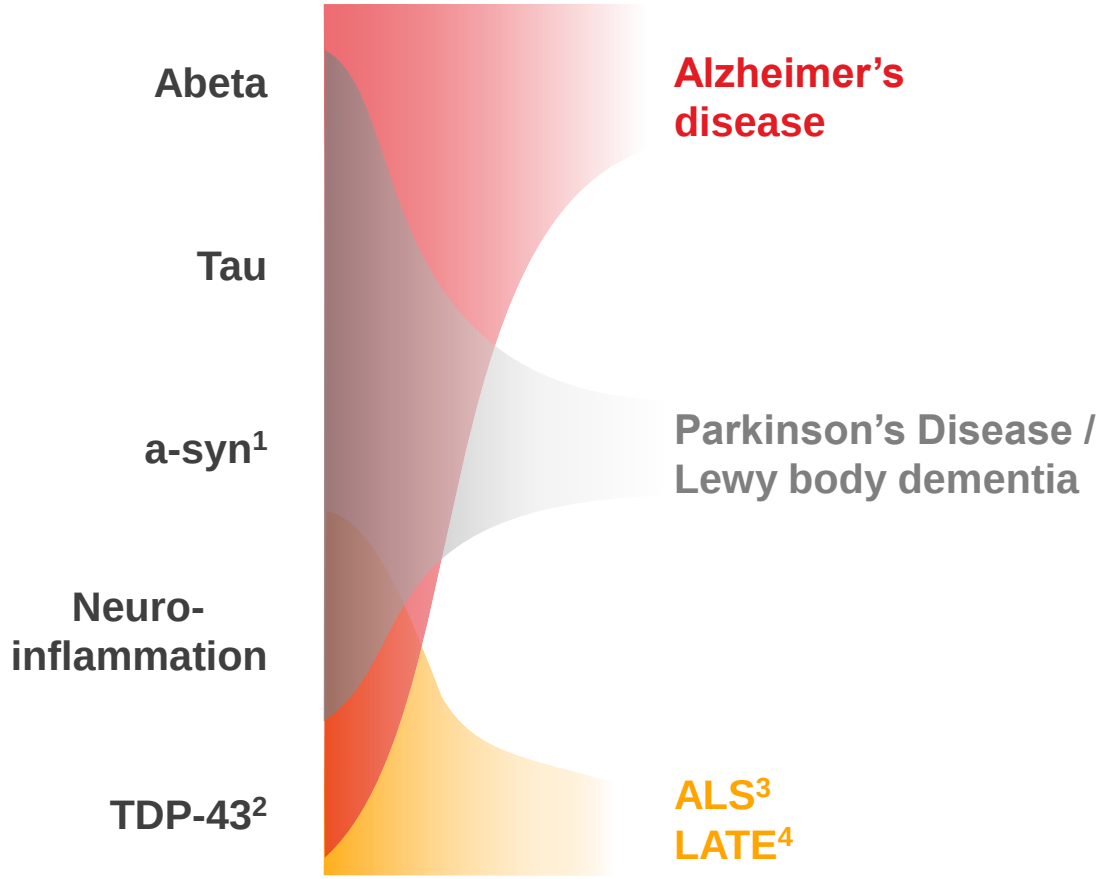
- AD prevention through combination of earlier diagnosis with early active immunotherapy
- Global disease prevention market potentially over 300 million people

(1) Alzheimer's Disease International 2019; (2) Gustavsson et al. Alzheimer's and Dement. 2023 19:658-670. https://doi.org/10.1002/alz.12694; (3) Alzheimer's disease; (4) Monoclonal antibody

Successfully treating neurodegeneration requires precision medicine

From a mono- to a multi-target combination approach

1. **Non-invasive diagnostics** are critical for identifying and monitoring disease
2. **Earlier, more reliable diagnosis** may eventually lead to disease **prevention**
3. Different therapies at different stages
4. Patients selected and treated according to their underlying pathologies
5. **Combination therapy** may be required



- Treating the right proteinopathies, in the right patient, at the right time

(1) alpha-synuclein; (2) TAR DNA-binding protein 43; (3) Amyotrophic lateral sclerosis; (4) Limbic-predominant age-related TDP-43 encephalopathy

Broad and robust pipeline in neurodegenerative diseases

Driven by validated proprietary technology platforms for sustained growth

Clinical Stage Programs

CLASS	PRODUCT CANDIDATE	INDICATION	Discovery	Preclinical	PHASE 1	PHASE 2	PHASE 3	News	Partner
Active Immunotherapy	**ACI-24.060** *(anti-Abeta)*	AD[1] treatment						data H1 '24[3] data H2 '24	
		AD treatment (*Down syndrome*[2])							
	ACI-7104.056 *(anti-a-syn*[4]*)*	PD[5], a-synucleinopathies						data H2 '24	
	ACI-35.030 *(anti-pTau)*	AD treatment							janssen
Small Molecule Morphomer®	**Tau-PET**[6] **tracer**	AD diagnostic							Life Molecular Imaging
		PSP[7] diagnostic							
	a-syn-PET tracer	a-synucleinopathies (e.g. MSA[8])							
	Tau aggregation inhibitor	Rare Tauopathies treatment							Lilly
		AD treatment							
Monoclonal antibody	**Semorinemab*** *(anti-Tau)*	AD treatment (*mild-to-moderate*)							
	Crenezumab* *(anti-Abeta)*	AD prevention							

(1) Alzheimer's disease; (2) Down syndrome-related Alzheimer's disease; (3) Refers to expected readouts from the ABATE Phase 1b/2 trial of ACI-24.060 in patients with AD; (4) alpha-synuclein; (5) Parkinson's disease; (6) Positron emission tomography; (7) Progressive supranuclear palsy; (8) Multiple system atrophy; * licensed to Genentech (a member of the Roche Group) until April 19, 2024

Broad and robust pipeline in neurodegenerative diseases

Diversification into non-AD[1] and non-CNS[2] diseases

Novel Targets Pipeline

CLASS	PRODUCT CANDIDATE	INDICATION	Discovery	Preclinical	PHASE 1	PHASE 2	News	Partner
Monoclonal antibody	**Anti-TDP-43** [3]	LATE[4], NeuroOrphan						
	Anti-a-syn [5]	PD[6], NeuroOrphan						
	Anti-NLRP3 [7]**-ASC**[8]	CNS, NeuroOrphan						
Small Molecule Morphomer®	**TDP-43-PET**[9] **tracer**	TDP-43-opathies						
	Morphomer® a-syn *(a-syn inhibitor)*	PD, a-synucleinopathies						
	Morphomer® NLRP3-ASC	Non-CNS						
	Morphomer® NLRP3-ASC	CNS, NeuroOrphan						

(1) Alzheimer's disease; (2) Central nervous system; (3) TAR DNA-binding protein 43; (4) Limbic-predominant age-related TDP-43 encephalopathy; (5) alpha-synuclein; (6) Parkinson's disease; (7) (NOD)-like receptor protein 3; (8) Apoptosis-associated speck-like protein containing a CARD, also PYCARD; (9) Positron emission tomography

AC Immune financial position

Value-driven cash management

Year end cash of CHF 103 million[1]
Plus CHF 15 million milestone received Feb 1, 2024

2024 annual cash burn guidance
CHF 65m – 75m

Cash runway[2]
Into Q1 2026, capturing multiple clinical catalysts

Prudent investment strategy focused on major value drivers and near-term catalysts

(1) Cash at December 31, 2023; (2) Assumes second ACI-35.030 milestone payment of CHF 25m received in 2025, no other milestones or deals included.

Key milestones for value creation in 2024

Multiple catalysts across pipeline

● Clinical readouts
○ Other development events

Active immunotherapies		H1	H2	
ACI-24.060	Abeta	●		**ABATE: Initial 6-month amyloid plaque data in AD[1] (PET[2] imaging)**
			●	**ABATE: Interim DS[3] data on safety and immunogenicity**
			●	**ABATE: 12-month amyloid plaque data in AD (PET imaging)**
ACI-35.030 (Janssen)	pTau	○		First Patient In Phase 2b clinical trial (ReTain)
ACI-7104.056	a-syn[4]		●	**Interim safety and immunogenicity Phase 2 VacSYn clinical trial in PD[5]**
Monoclonal antibodies and small molecule drugs				
Monoclonal antibody	TDP-43[6]	○		**Completion of regulatory tox studies**
Morphomer-NLRP3	NLRP3[7]		○	**Clinical candidate declaration**
Morphomer-a-syn	a-syn		○	Lead candidate declaration
Diagnostics				
TDP-43-PET tracer	TDP-43	○		CTA[8] submission
TDP-43-PET tracer	TDP-43		○	Phase 1 initiation
a-syn-PET tracer (ACI-15916)	a-syn		○	**PD candidate, IND[9]-enabling studies completed**

Amyloid-PET Data in H1 and H2 2024

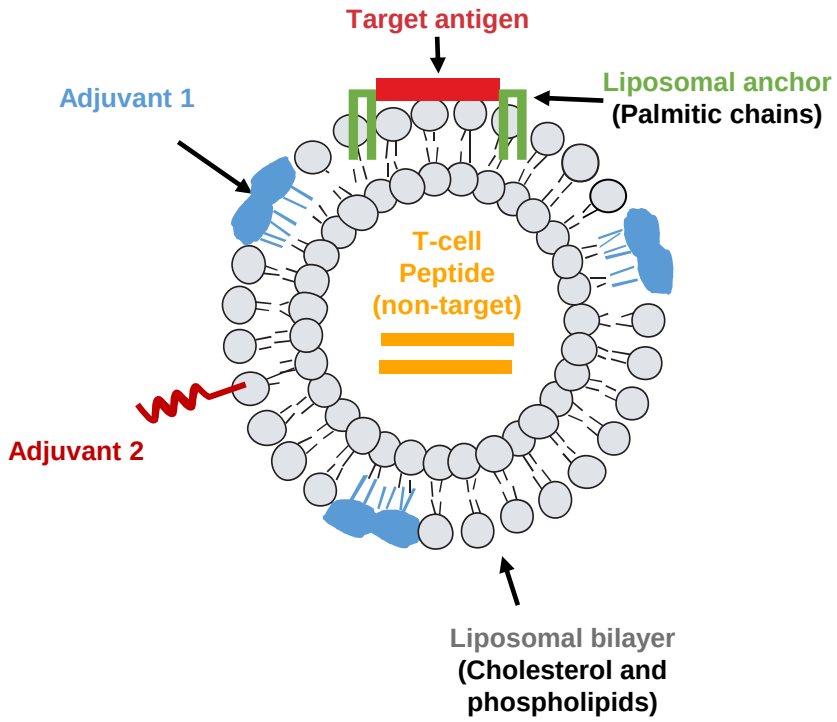
(1) Alzheimer's disease; (2) Positron emission tomography; (3) Down syndrome; (4) alpha-synuclein; (5) Parkinson's disease; (6) TAR DNA-binding protein 43; (7) (NOD)-like receptor protein; (8) Clinical trial application; (9) Investigational new drug

ACTIVE Immune Therapy

targeting neurodegenerative diseases

Disruptive potential of SupraAntigen®

Active immunotherapies delivering superior results in neurodegenerative diseases



Target-specific antibody response

Helper T-cells safely engaged **(target-unrelated)**

Unprecedented Clinical Performance

Immunogenicity	✓
Target & conformation specificity	✓
Avidity increase over time	✓
Sustainable response	✓
Boostable response	✓

- Robust immunogenicity and strong safety demonstrated in humans
- Evidence for lasting immune response supporting a disease prevention approach

For ACI-35.030: (1) 100% response after 1st injection; (2) Increases over time

ACI-24.060: Active immunotherapy to clear Abeta plaques to treat AD[1]

ACI-24.060 targets pyroGlu- and oligomeric Abeta, which are believed to drive AD progression

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AβATE: Biomarker-based Phase 1b/2 study of ACI-24.060 in AD[1] and DS[2]

Placebo-controlled Phase 1b/2 Study Overview

Adaptive Study Design

Both
- Interim analyses of safety/tolerability & immunogenicity
- Biomarker analyses including Abeta PET[3] and others

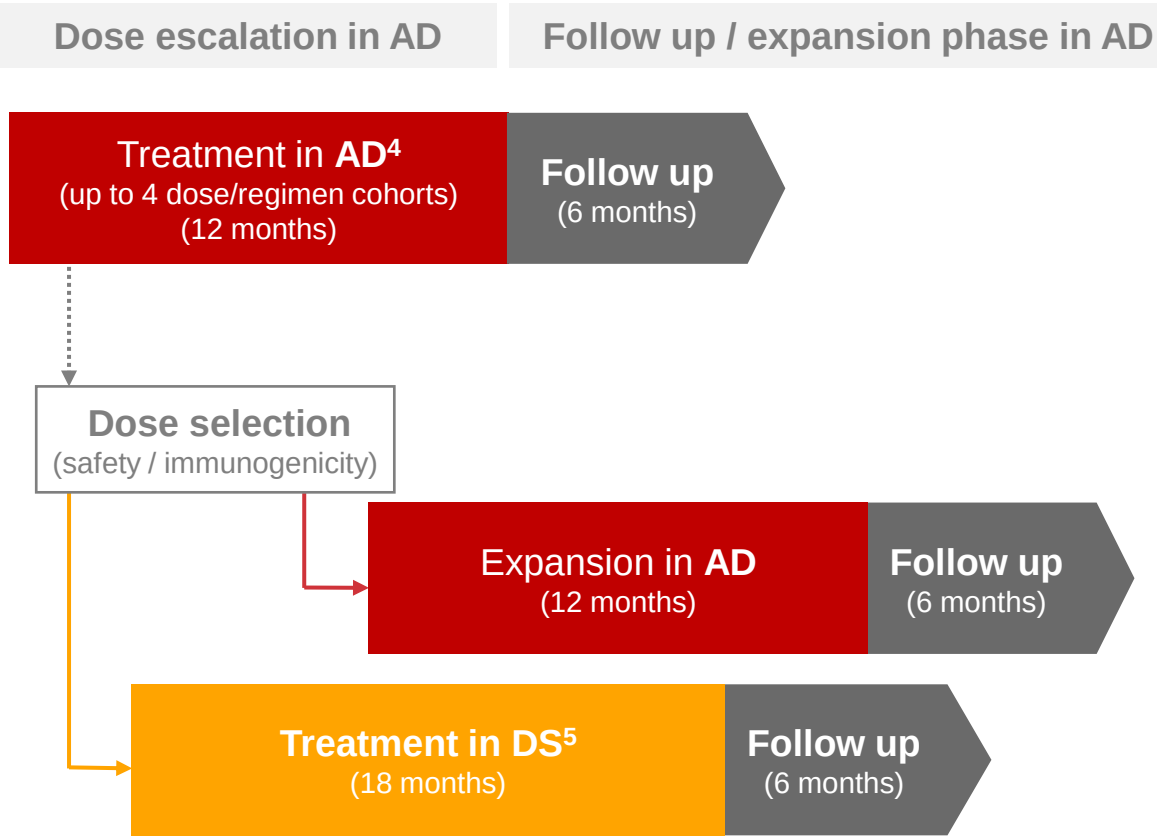
AD
- Up to 4 different doses and/or dose regimens
- Expansion of one cohort to assess effect on Abeta PET

DS
- Initiation using selected dose identified in AD (based on safety/tolerability and immunogenicity)

Outcome measures

Both
- Safety/tolerability
- Pharmacodynamics: Serum anti-Abeta antibody titers
- **Abeta-PET imaging**
- Exploratory biomarkers and clinical endpoints

Trial Schematic

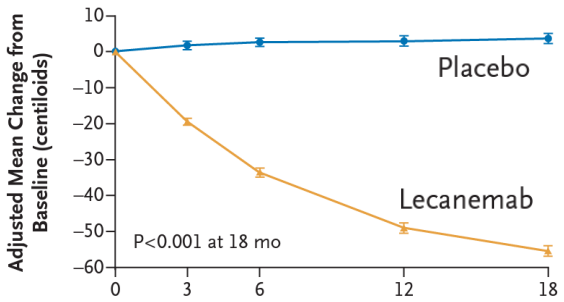


(1) Alzheimer's disease; (2) Down syndrome-related AD; (3) Positron emission tomography; (4) AD participants must between 50 – 85 years of age and have prodromal AD with Clinical Dementia Rating Global Score of 0.5 and Abeta pathology confirmed by PET scan; (5) Cohort comprised of non-demented people living with DS (age 35 – 50 years) and Abeta pathology confirmed by PET scan

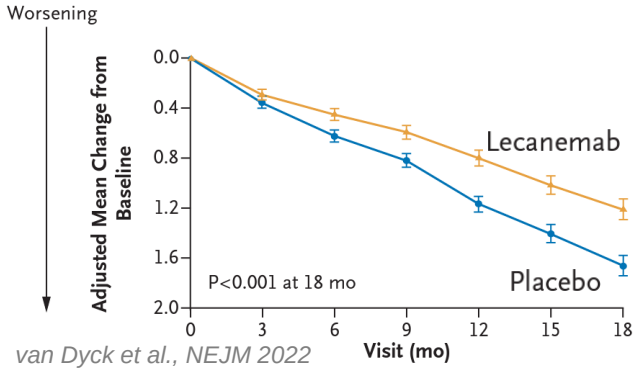
Lowering Amyloid PET[1] is a valid biomarker for clinical effect

Lecanemab & donanemab trials established PET imaging as surrogate for clinical effect

Amyloid Burden on PET

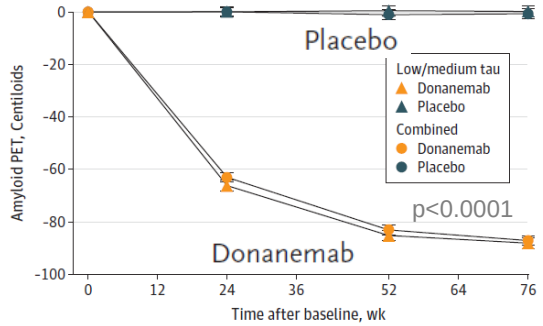


Primary endpoint: CDR-SB[2]

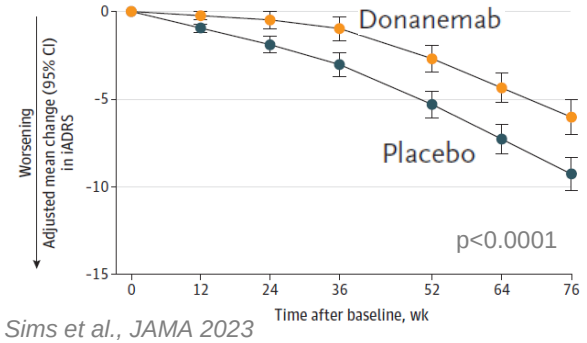


van Dyck et al., NEJM 2022

Amyloid Burden on PET



Primary endpoint: iADRS[3]



Sims et al., JAMA 2023

- Targeting soluble oligomers and pyroglutamate (N3pG) has demonstrated clinical utility
- Reductions in Abeta plaques can be detected as early as 3 months after the start of treatment

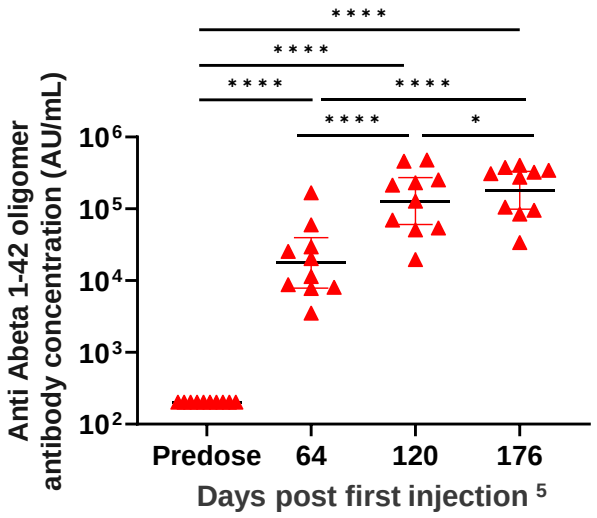
(1) Positron emission tomography; (2) Clinical dementia rating – sum of boxes; (3) Integrated Alzheimer's disease rating scale

ACI-24.060: Potent immune response against toxic Abeta species

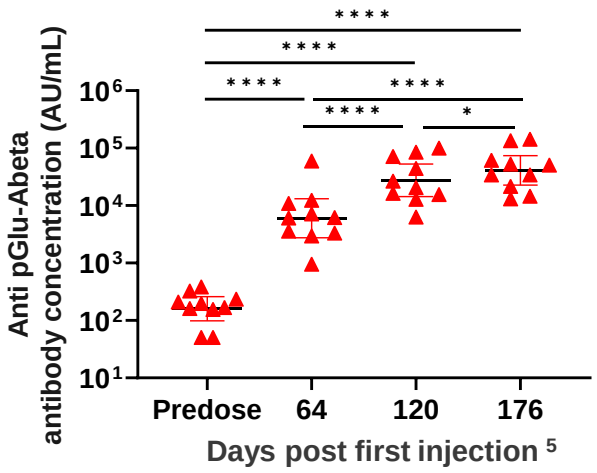
Strong antibody response against targets of lecanemab and donanemab (NHP[1])

ACI-24.060 in NHPs

Anti Abeta1-42 oligomer IgG

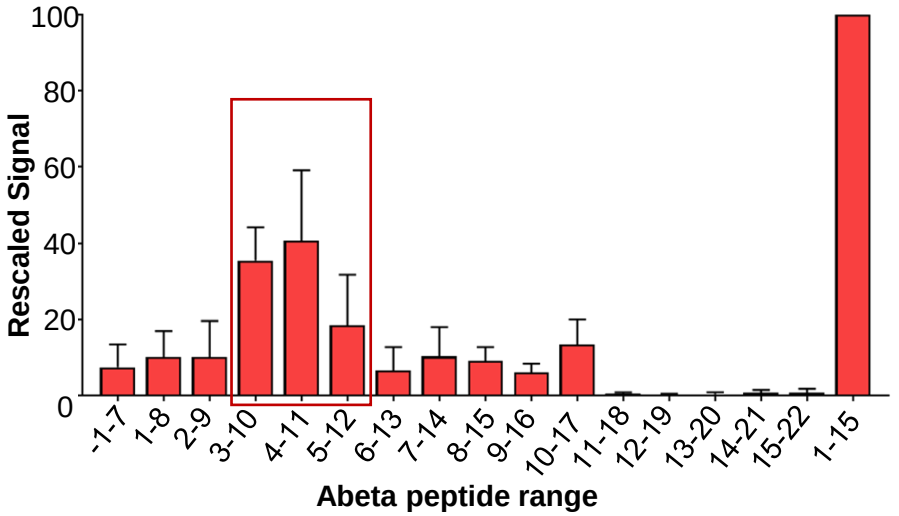


Anti-pyroGlu[2]-Abeta IgG



* p<0.05 **** p<0.0001

Epitope mapping in NHP (120 days)



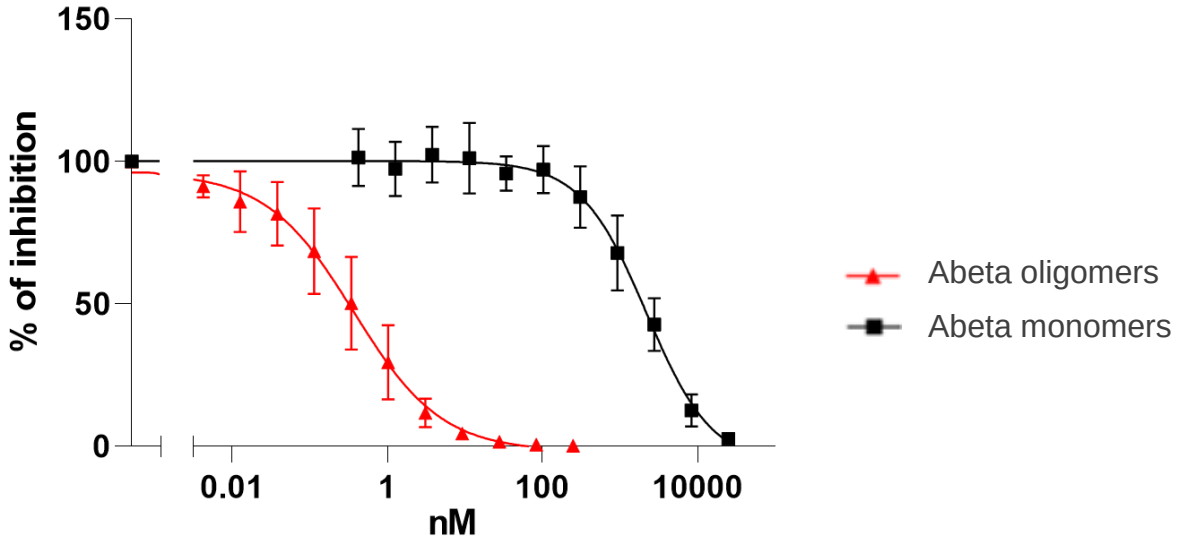
Ref.: Global Down Syndrome Forum 2021; M. Vukicevic, et al., Brain Comm, 2022

- Sustained, boostable IgG response against Abeta oligomers[3] and pyroglutamate[4] Abeta
- ACI-24.060 represents a potential breakthrough compared to previous anti-Abeta therapeutics

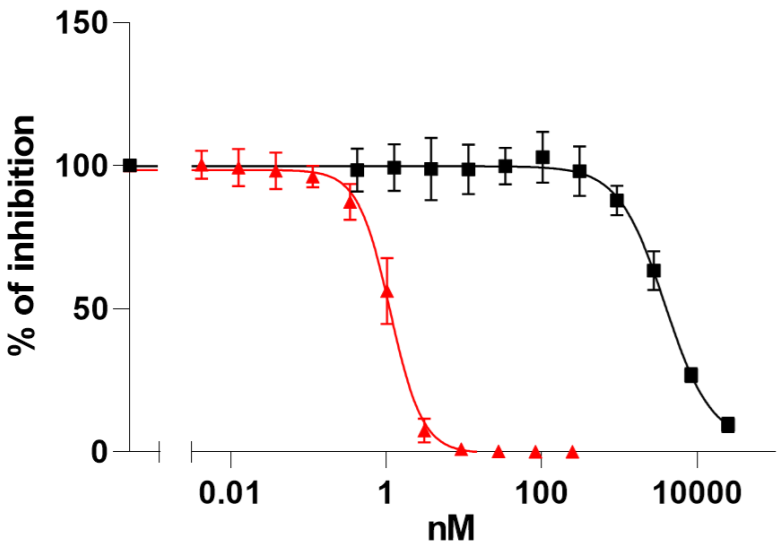
(1) Non-human primates; (2) Pyroglutamate; (3) Target of lecanemab; (4) Target of donanemab (5) Injections on days 0, 29, 57, 85, 113, 141, 169

ACI-24.060: antibodies highly specific for pathologic oligomeric Abeta

Antibodies in NHP[1] immune sera have >1000-fold preference for oligomers over monomers



NHP immunized with ACI-24.060



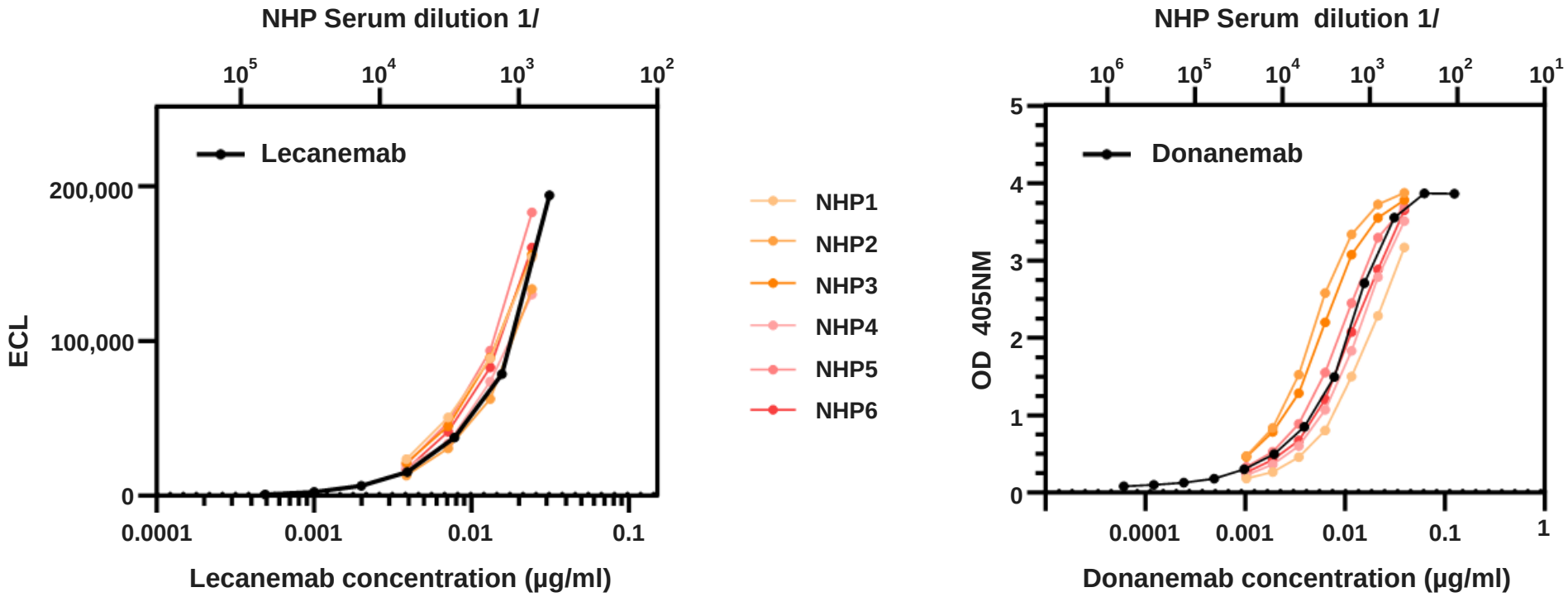
Lecanemab

- ACI-24.060 induced antibodies in NHPs: >1000-fold stronger recognition of Abeta oligomers than monomers, similar to lecanemab

(1) Non-human primates

ACI-24.060 generates antibodies highly specific for pathologic Abeta species

Antibodies in NHP[1] immune sera similar specific activity to lecanemab and donanemab



Immunization of NHPs with ACI-24.060 generates IgGs in quantities which recognize:

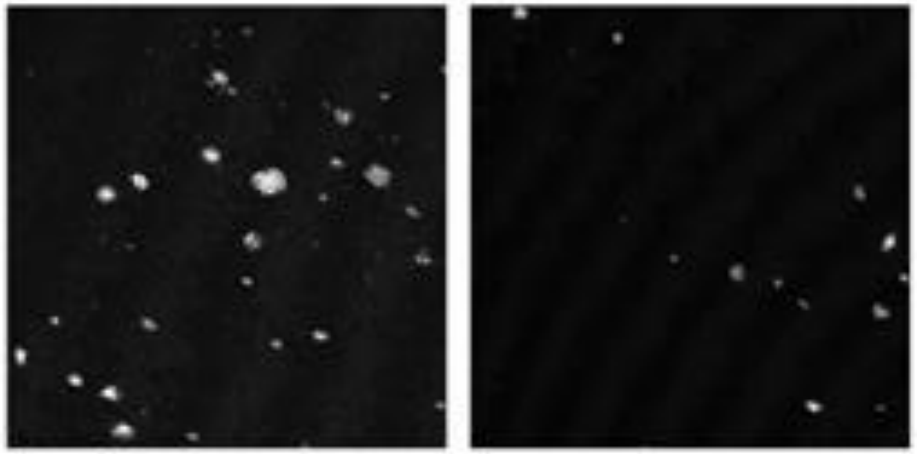
- Abeta oligomers equivalent to the range of 18 - 26 µg/mL of lecanemab
- pyroglu-Abeta equivalent to in the range of 11 - 48 µg/mL of donanemab

(1) Non-human primates

ACI-24 active immunotherapy reduces Abeta plaque burden

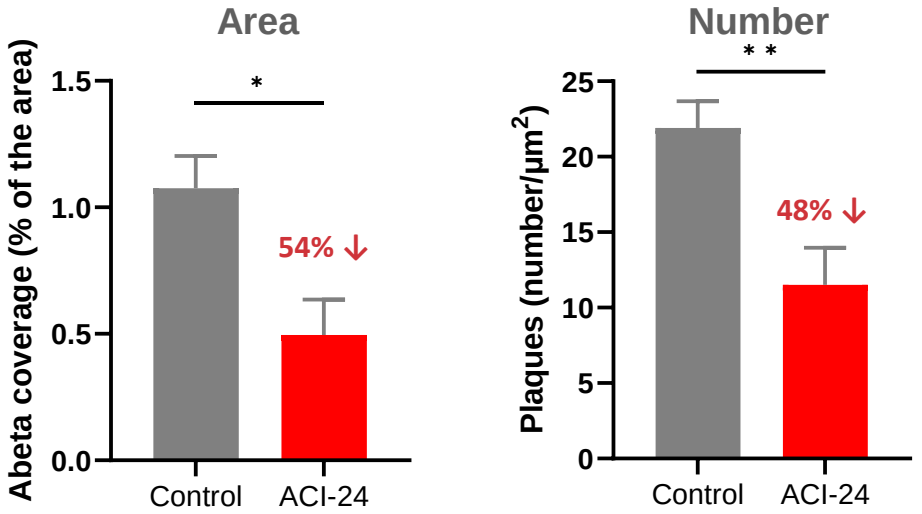
Significant Abeta plaque reduction *in vivo* in preclinical APPxPS1 model[1]

Abeta Plaque Staining in Control and ACI-24-treated Mice



Control ACI-24

Quantification of Abeta Plaques



* p<0.05 ** p<0.01

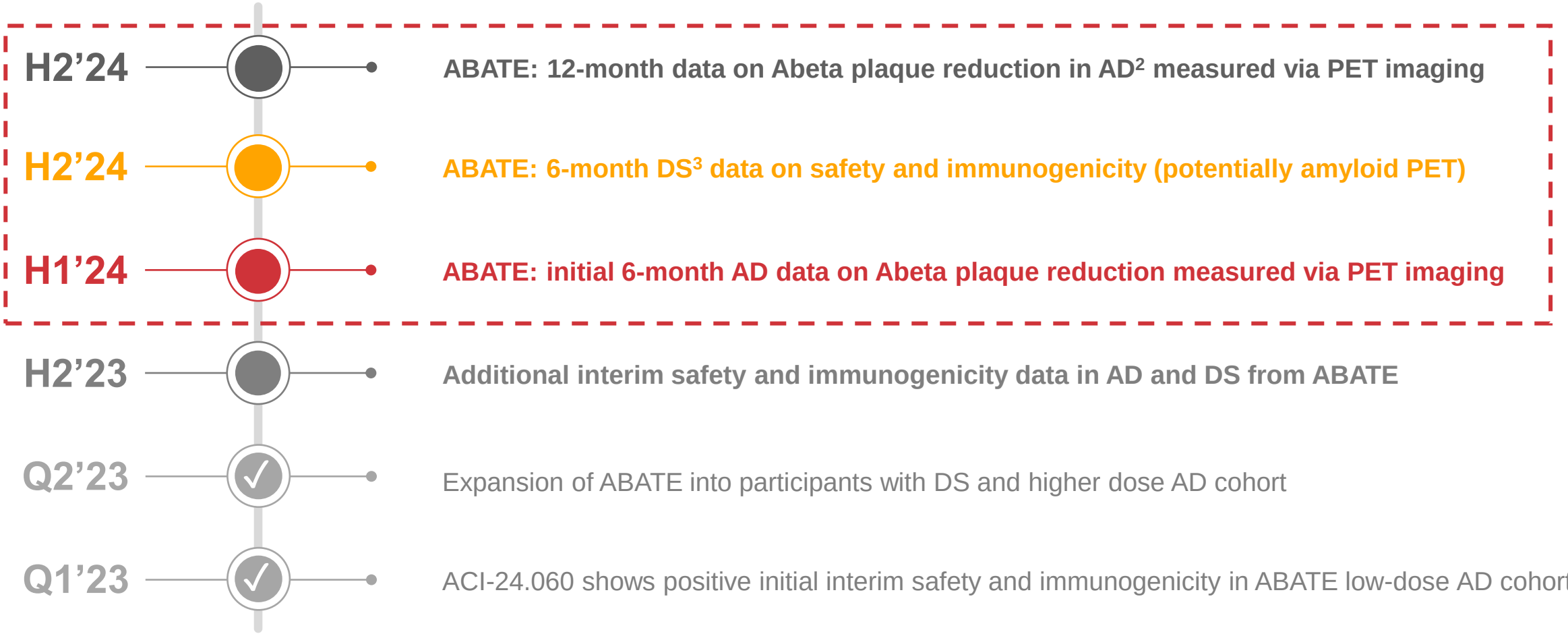
Ref: Njavro, *et al.*, Cells 2023

- ACI-24 treatment significantly reduces Abeta plaque burden in aggressive APPxPS1 model
- Similar plaque reductions seen with lecanemab and donanemab in less aggressive APP models

(1) Alzheimer's disease mouse model: APPxPS-1 double transgenic mice; (2) Alzheimer's disease; (3) Antibodies

ACI-24.060 program: Anticipated milestones

ABATE Ph 1b/2 trial: Abeta plaque reduction measured via PET[1] imaging expected in H1 2024



(1) Positron emission tomography; (2) Alzheimer's disease; (3) Down syndrome

ACI-35.030: Anti-pTau active immunotherapy developed for preclinical AD[1]

Phase 2b ReTain trial in preclinical AD

Clinical Stage Programs

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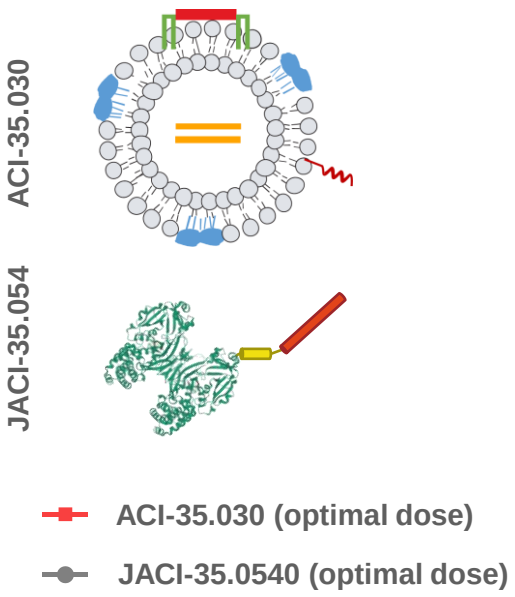
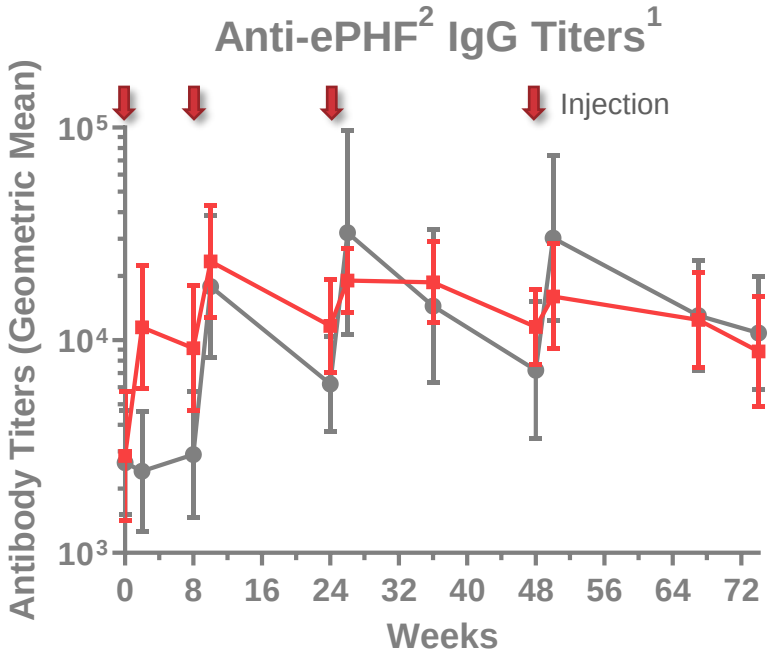
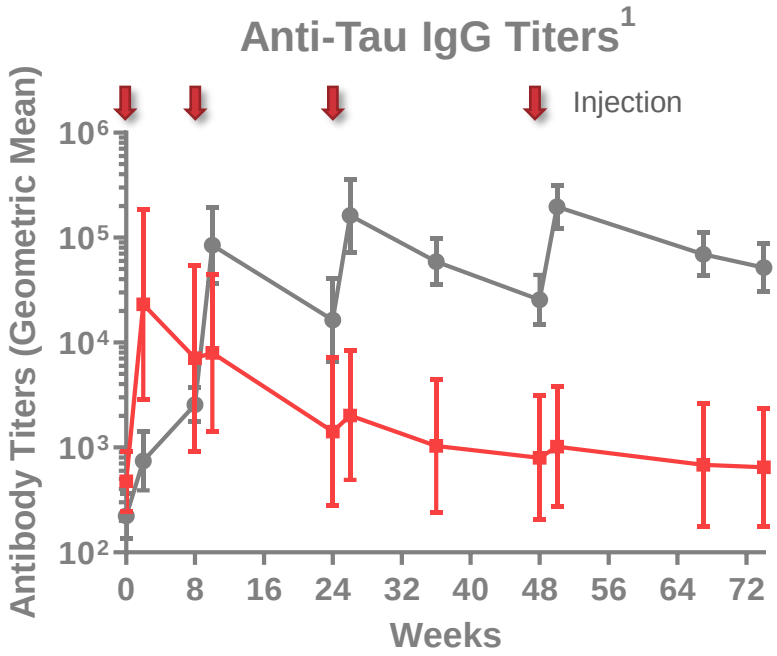
ACI-35.030 selected for further development by partner Janssen

Follows data showing ACI-35.030's superior specificity for pathological Tau vs. JACI-35.054







1 JACI-35.054 is a protein conjugate active immuno-therapy utilizing the same pTau[3] epitope as ACI-35.030

2 ACI-35.050 and JACI-35.054 were evaluated in parallel in the Phase 1b/2a trial in AD[4] patients

3 ACI-35.030 induced Ab[5] responses in 100% of patients after 1st injection compared to 50% with JACI-35.054

4 ACI-35.030-induced anti-ePHF Abs: longer apparent half-lives, less variability, lower peak-to-trough ratios

(1) ACI-35.030 original sub-cohort 1.2 data; (2) Enriched paired helical filaments; (3) Phosphorylated Tau; (4) Alzheimer's disease; (5) Antibody

ReTain: a Phase 2b study of ACI-35.030 in preclinical AD[1]

A randomized, multicenter, double-blind, placebo-controlled Phase 2b study

Study population

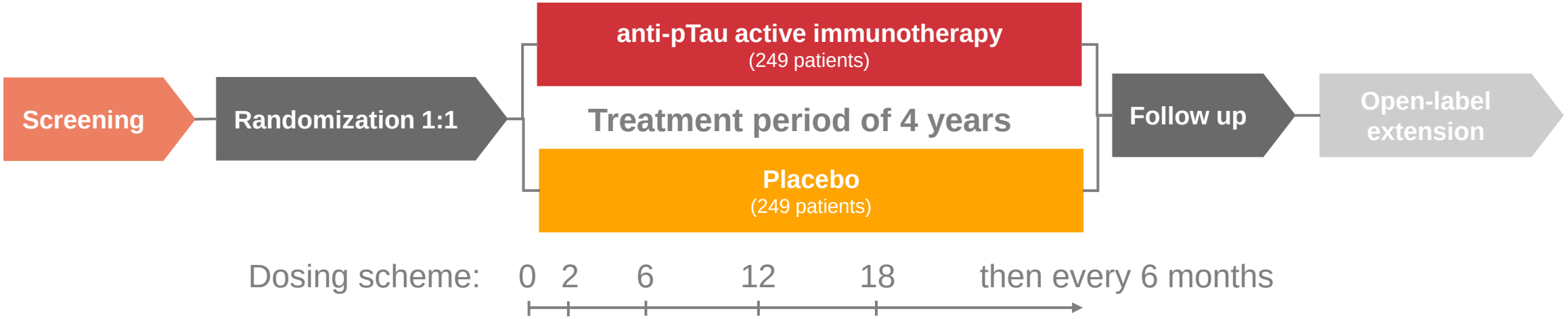
- ~500 participants with **preclinical AD**:
  - Cognitively normal
  - Tau PET positive
  - Amyloid positive[2]
- Prior to appearance of clinical symptoms

Biomarker readouts

- Tau pathology compared with placebo:
  - Tau-PET imaging[3]
  - Baseline and annually for 4 years
- Potential **BLA filing** and **accelerated approval**

Primary cognitive endpoint

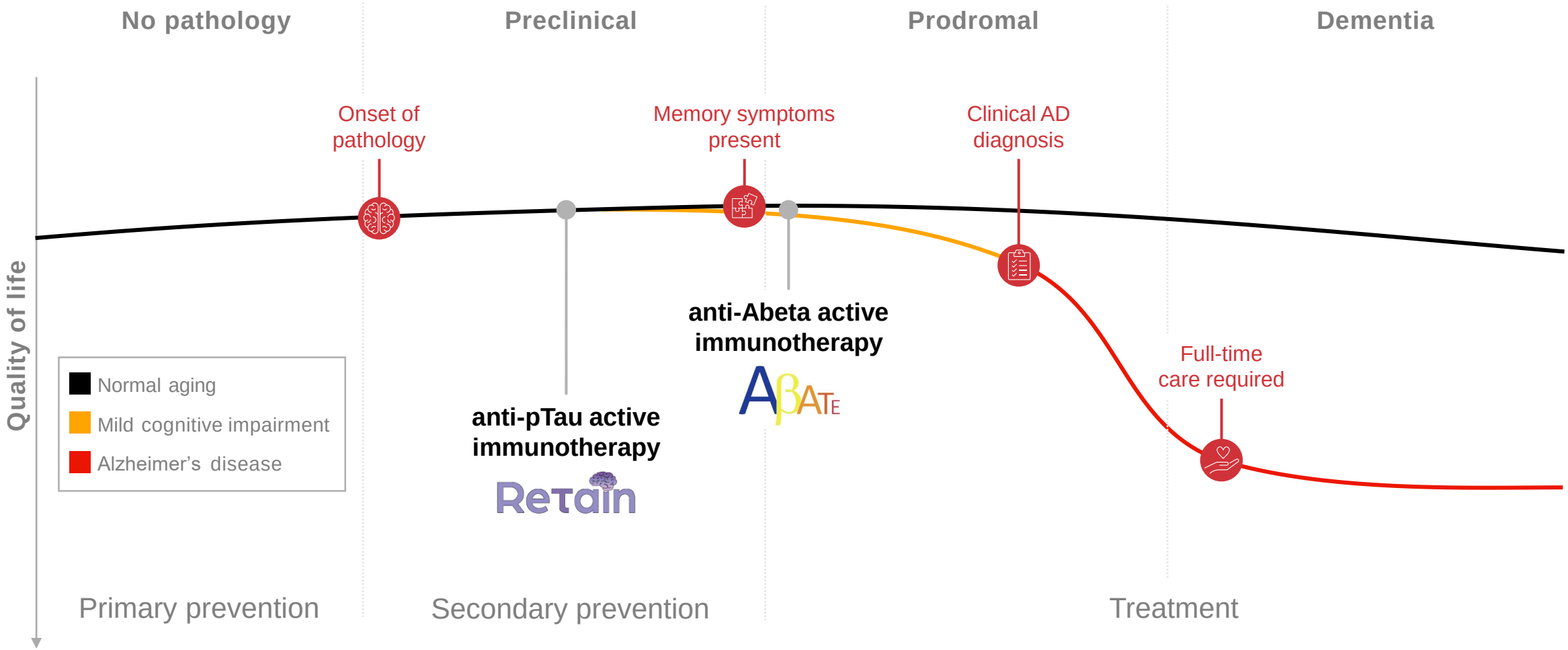
- Preclinical AD Cognitive Composite 5[4]:
  - Episodic memory
  - Timed executive function
  - Global cognition
- Potential **traditional approval**



(1) Alzheimer's disease; (2) Implied Abeta positivity (A+) because of Tau positivity (T+), but not part of the inclusion criteria; (3) Tau-PET measured in the Tau-naïve composite region; (4) PACC-5

Modifying the course of Alzheimer's disease…

…from slowing progression to prevention



ACI-7104: Anti-a-syn[1] active immunotherapy for Parkinson's disease

Update on Phase 2 VacSYn trial in H2

Clinical Stage Programs

CLASS	PRODUCT CANDIDATE	INDICATION	News	Partner
Active Immunotherapy	ACI-24.060 *(anti-Abeta)*	AD[1] treatment	data H1 '24[3] data H2 '24	
		AD treatment (*Down syndrome[2]*)		
	ACI-7104.056 *(anti-a-syn[4])*	PD[5], a-synucleinopathies	**data H2 '24**	
	ACI-35.030 *(anti-pTau)*	AD treatment		janssen
Small Molecule Morphomer®	Tau-PET[6] tracer	AD diagnostic		Life Molecular Imaging
		PSP[7] diagnostic		
	a-syn-PET tracer	a-synucleinopathies (e.g. MSA[8])		
	Tau aggregation inhibitor	Rare Tauopathies treatment		Lilly
		AD treatment		
Monoclonal antibody	Semorinemab *(anti-Tau)*	AD treatment (*mild-to-moderate*)		
	Crenezumab *(anti-Abeta)*	AD prevention		

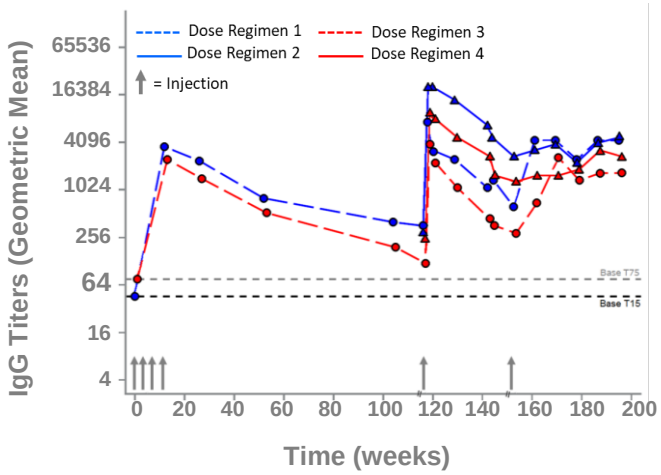
(1) Alzheimer's disease; (2) Down syndrome-related Alzheimer's disease; (3) Refers to expected readouts from the ABATE Phase 1b/2 trial of ACI-24.060 in patients with AD; (4) alpha-synuclein; (5) Parkinson's disease; (6) Positron emission tomography; (7) Progressive supranuclear palsy; (8) Multiple system atrophy

Clinically validated[1] anti-a-syn[2] active immunotherapy in PD[3]

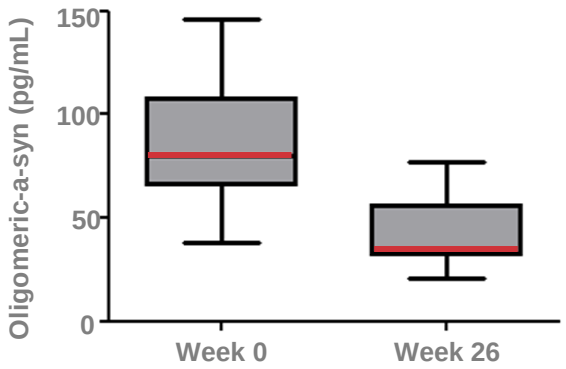
Phase 1 results in *The Lancet Neurology* support best-in-class profile

THE LANCET Neurology

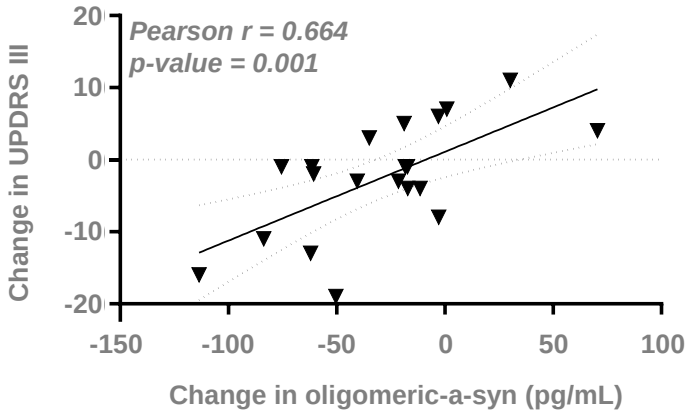
Strong and boostable antibody response



50% reduction[4] of pathological a-syn in CSF[5]



Changes[6] in oligo-a-syn and UPDRS III correlate



1 Safe and well tolerated with no safety concerns noted in patients followed for more than 3.5 years

2 Strong and boostable antibody responses

3 Target engagement evidence: 50% reduction in pathological (oligomeric) a-syn in the CSF

4 Signal of clinical efficacy: stabilization of UPDRS[7] III scores correlated with reductions in oligomeric a-syn

(1) Volc *et al.*, Lancet Neurol. 2020; (2) alpha-synuclein; (3) Parkinson's disease; (4) Data from 75 µg dose group; (5) Cerebrospinal fluid; (6) Change in oligomeric a-syn calculated at week 26, change in UPDRS III calculated at week 100; (7) Unified Parkinson's Disease Rating Scale

AC Immune

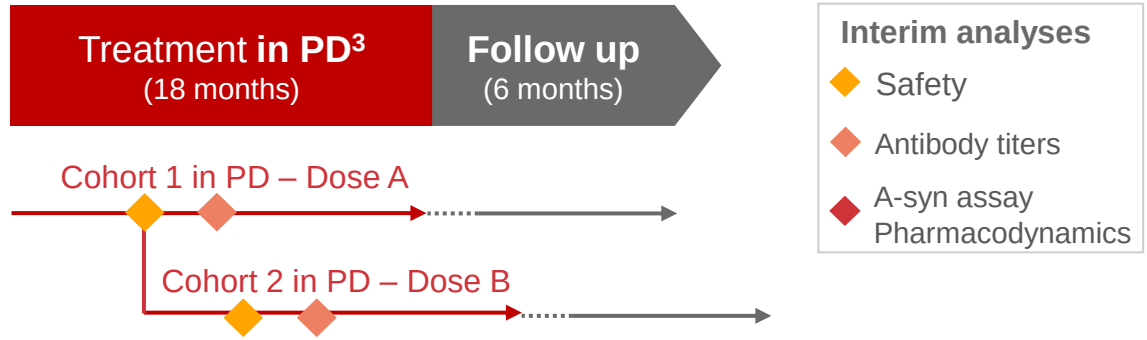
VacSYn: an adaptive biomarker-based Phase 2 study of ACI-7104 in early PD[1]

Placebo-controlled Phase 2 Study Overview

- Seamless transition
  - All participants from Part 1 will contribute to final analysis
- Biomarker based interim analyses
  - Early immunogenicity to tailor dose and/or dose regimen
  - Apply disease-relevant biomarkers for early transition to filing

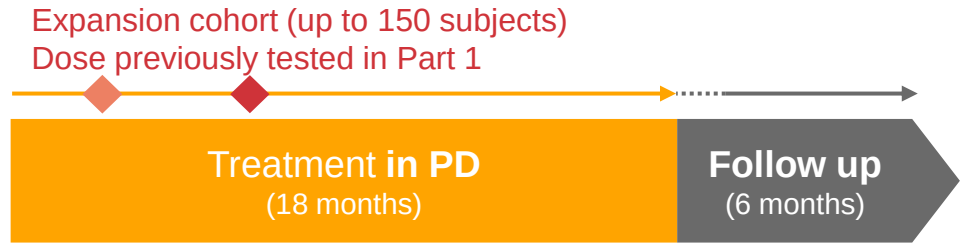
Part 1: Safety & PK/PD[2]

- Key immunogenicity measures
- Measures of pathological a-syn[4] (a-syn oligomers and aggregates)



Part 2: PoC[5] in early PD

- Motor and Non-Motor Functioning (UPDRS[6] based)
- Degeneration of dopaminergic terminals (DaT SPECT[7] imaging)
- Advanced MRI (including ASL[8] and DTI[9])
- Digital biomarkers of motor and non-motor function
- Functional and patient reported outcomes



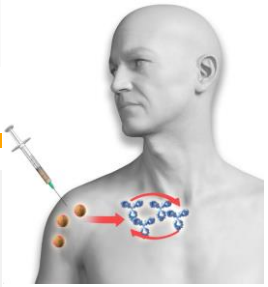
(1) Parkinson's disease; (2) Pharmacokinetics and Pharmacodynamics; (3) Participants must have idiopathic PD and be stable on up to 300 mg of L-Dopa treatment and dopaminergic deficit determined by Dopamine Transporter Single Photon Emission Computed Tomography; (4) alpha-synuclein; (5) Proof-of-concept; (6) Unified Parkinson's disease rating scale; (7) Dopamine Transporter Single Photon Emission Computed Tomography; (8) Arterial spin labeling; (9) Diffusion tensor imaging

Active immunotherapy: clear advantages for long-term use

Provides opportunity to prevent AND treat neurodegenerative diseases globally

ACTIVE Immune Therapy

- Long-lasting specific immunity for pathological target, consistent, boostable, durable
- Limited annual dosing (once or twice) after priming year
- No observed ARIA-E[1] to date (safety profile well suited to long-term use)
- Cost-effective (attractive healthcare economics across global populations)
- Improved access (ease of administration, simple logistics)

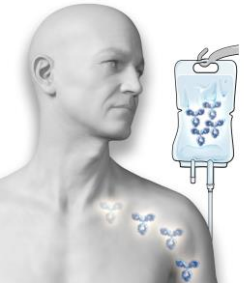


Active immunotherapy

Vaccines stimulate the patient's immune system to produce antibodies

Passive immunotherapy

Externally generated monoclonal antibodies require administration every two to four weeks

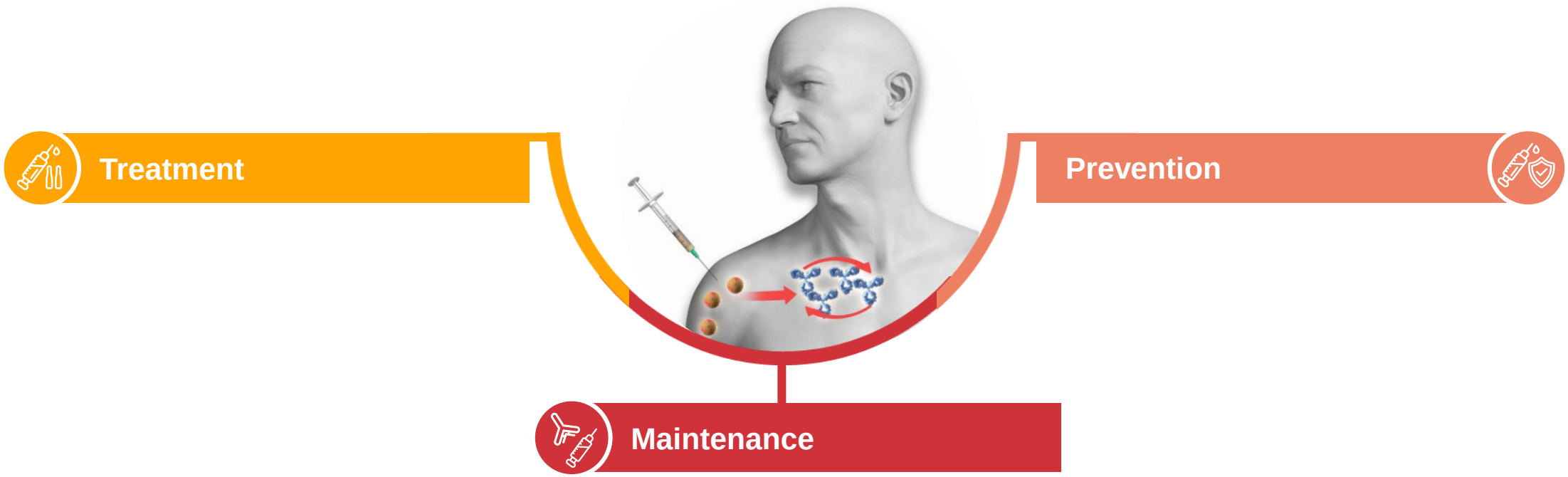


- Active immunotherapy is potentially the only option for global prevention of NDDs[2]

(1) Amyloid-related imaging abnormalities; (2) Neurodegenerative diseases

Active immunotherapy: a new class of treatment for neurodegenerative disease

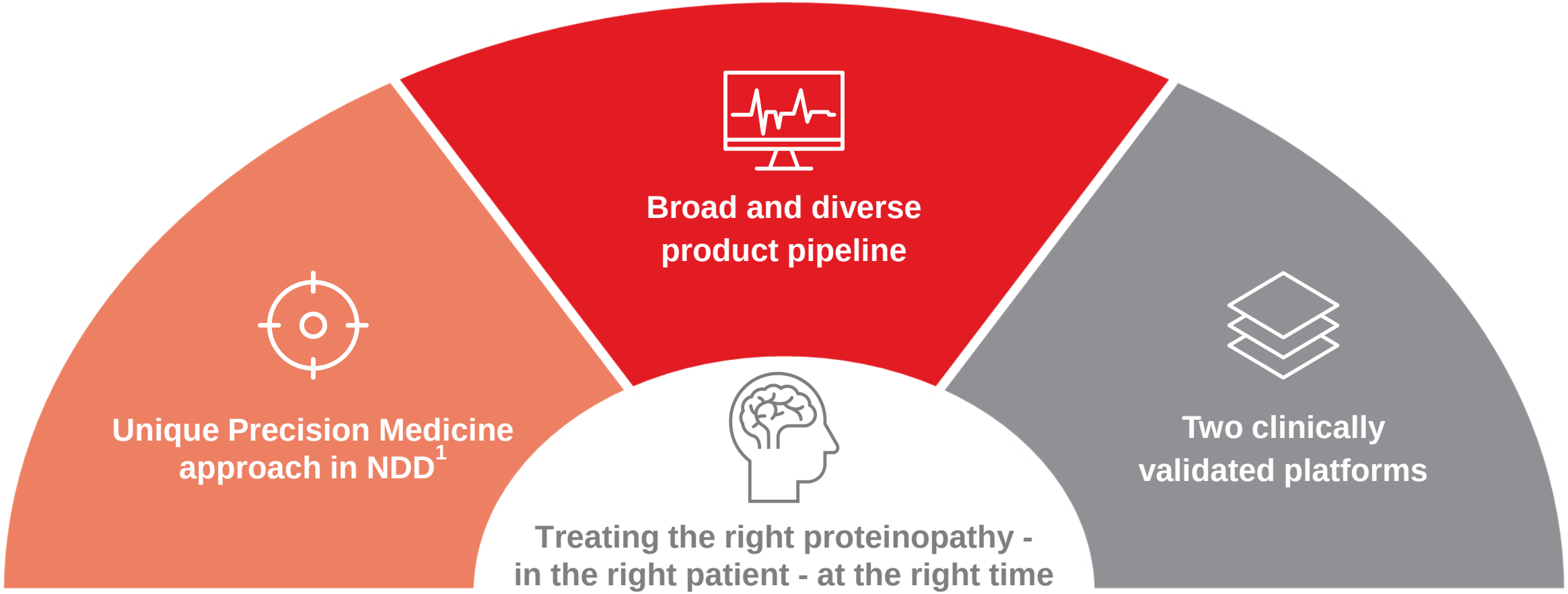
Potential for profound social and economic impact



ACTIVE Immune Therapy for global treatment and prevention of neurodegenerative diseases

Today’s strengths predict future success

Precision Medicine for mono- and combination therapy



(1) Neurodegenerative diseases

AC Immune: Pioneering science and precision medicine

Shifting the treatment paradigm for neurodegenerative disease towards precision medicine and disease prevention

Supplementary information

External validation and cash generated by 5 partnering[1] deals

Managing risk and retaining significant upside

	Product	Dev. phase	Total value[2]	Upfront[2]	Milestones received to date[2]	Royalties	Partners
Biologicals	Crenezumab (anti-Abeta antibody)	Phase 2	USD 65 [3]	USD 25	USD 40		*
Biologicals	Semorinemab (anti-Tau antibody)	Phase 2	CHF 59 [3]	CHF 17	CHF 42		*
Biologicals	ACI-35.030 (anti-pTau active immunotherapy)	Phase 1b/2a	CHF 500	CHF 26	CHF 20	Low-double digits to mid-teens	Janssen
Small molecules	Tau PET[4] imaging agent	Phase 3 [5]	EUR 160	EUR 0.5	EUR 7	Mid-single digits to low-teens	Life Molecular Imaging
Small molecules	Tau Morphomer® small molecules	Phase 1 [6]	CHF 1,860	CHF 80 +USD 50 [7]	CHF 40	Low-double digits to mid-teens	Lilly
	Total (millions)[8]		**CHF ~2,600**	**CHF 155.2 [9]**	**CHF 147.4**		

- Outstanding potential milestone payments exceed CHF ~2.5 billion

(1) Disclosure limited due to confidentiality agreements with collaboration partners; (2) In millions; (3) Total payments received from partner until termination of agreement; (4) Positron emission tomography; (5) In Alzheimer's disease; (6) Phase 1 completed; (7) Equity investment; (8) Converted to CHF on date of receipt; (9) Excludes convertible note agreement of USD 50 million ; * licensed to Genentech (a member of the Roche Group) until April 19, 2024