Investor Presentation

NASDAQ: ACIU | January 2025

AC Immune

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AC Immune today – an overview

Pioneering next generation Precision Medicine for neurodegenerative diseases

Diverse and balanced pipeline with a large number of wholly-owned assets

Key differentiation: Precision Medicine
Enabled by leadership in Active Immunotherapy

New breakthroughs, e.g. morADC[3]: our platforms have repeatedly created potentially transformative innovations

Partnering: strategic, risk-mitigating, timely, monetization with >CHF 4 billion in potential milestones

Cash reserves on Balance sheet
Funding into 2027

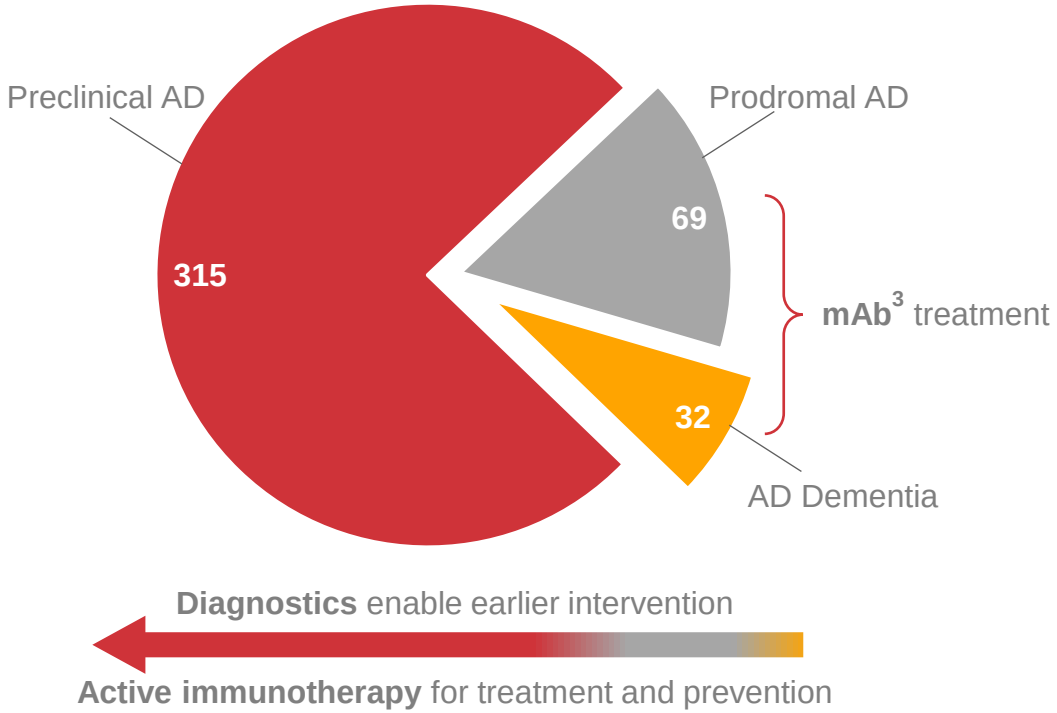
- Based in Lausanne, Switzerland
- ~150 employees
- Listed September 2016 (NASDAQ: ACIU)
- 100 million shares outstanding[1]
- Cash of CHF 182.5 million[2]

(1) As of Sep 30, 2024, 99.986 million shares outstanding; excluding treasury shares; (2) including CHF157.9 million as of Sep 30, 2024 and CHF24.6 m milestone payment received from J&J mid-October; (3) Morphomer-antibody drug conjugate

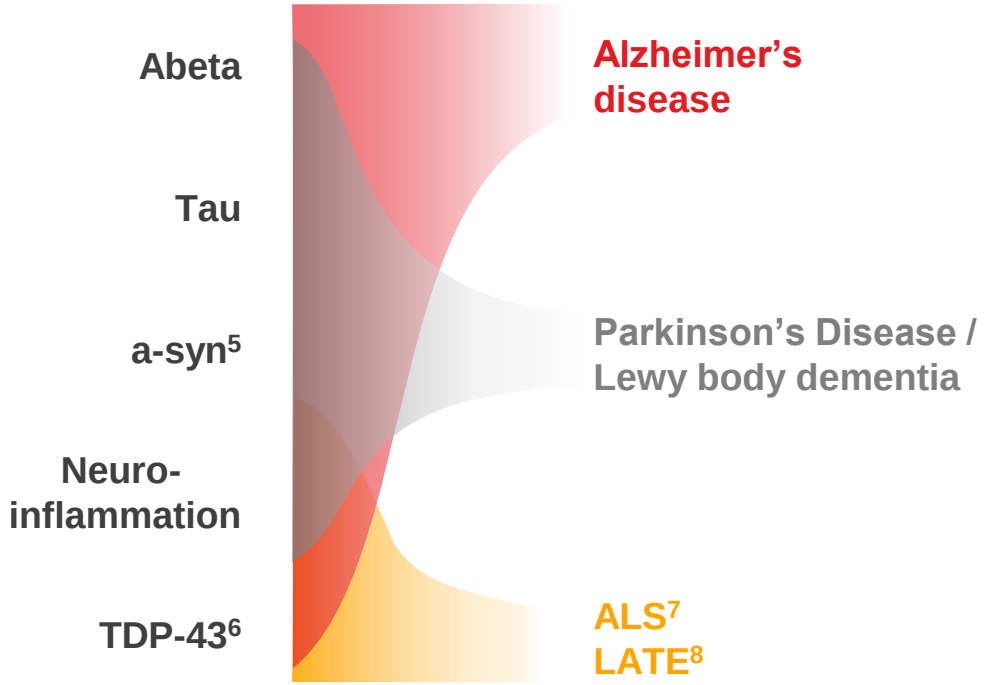
Neurodegenerative diseases

Prevention as the best approach to long-term preservation of neurological health

Global Prevalence of AD[1] Stages[2] (million)



Co-pathologies in NDD[4]



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

(1) Alzheimer's disease; (2) Gustavsson et al. Alzheimer's and Dement. 2023 19:658-670. https://doi.org/10.1002/alz.12694; (3) Monoclonal antibody; (4) Neurodegenerative disease; (5) alpha-synuclein; (6) TAR DNA-binding protein 43; (7) Amyotrophic lateral sclerosis; (8) Limbic-predominant age-related TDP-43 encephalopathy

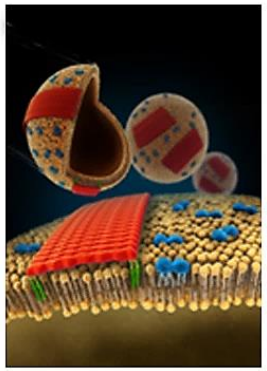
SupraAntigen® and Morphomer® platforms

Deliver conformation-specific candidates for integrated Precision Medicine in CNS

CNS-optimized

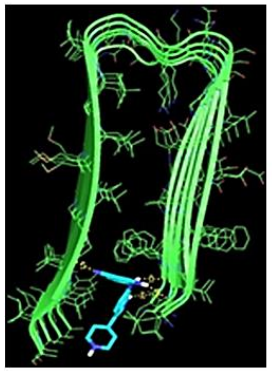
Clinically validated

SupraAntigen®



Vaccines & Antibodies

Morphomer®



Small Molecules

morADC

Conformation-specific

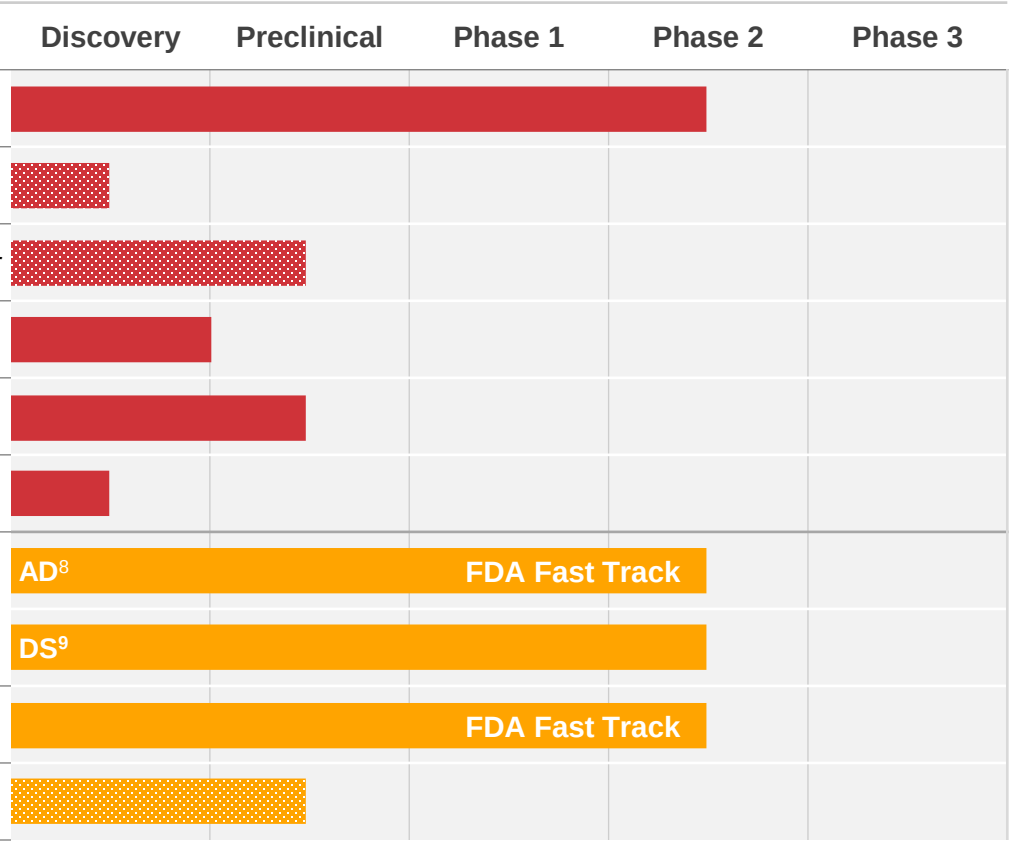
Precision medicine enabling

AC Immune

Broad and robust pipeline in neurodegenerative diseases

Driven by validated proprietary technology platforms for sustained growth

	Indication	Candidate	Partner	Modality
Wholly-owned	Parkinson's disease	ACI-7104.056		*anti-a-syn[1] active immunotherapy*
		Morphomer® a-syn		*anti-a-syn small molecule*
	Neuro-inflammation	ACI-19764		*anti-NLRP3 [3] small molecule inhibitor*
		Anti-NLRP3-ASC[4]		*anti-ASC monoclonal antibody*
	ALS[5]	ACI-5891.9		*anti-TDP-43 [6] monoclonal antibody*
	NDDs[7]	morADC		*Morphomer-antibody drug conjugate*
Partnered	Alzheimer's disease	ACI-24.060	Takeda	*anti-Abeta active immunotherapy*
		ACI-35.030	janssen	*anti-pTau active immunotherapy*
		Morphomer Tau	Lilly	*anti-Tau small molecule inhibitor*



(1) alpha-synuclein; (2) Parkinson's disease; (3) (NOD)-like receptor protein 3; (4) Apoptosis-associated speck-like protein containing a CARD, also PYCARD; (5) Amyotrophic lateral sclerosis; (6) TAR DNA-binding protein 43; (7) Neurodegenerative diseases; (8) Alzheimer's disease; (9) Down syndrome

Key milestones

Multiple catalysts across pipeline including selected 2025 milestones

● Readouts
○ Other development events

Active immunotherapies		H1'24	H2'24	2025	
ACI-24.060 (Takeda)	Abeta		✓		**ABATE Phase 2 trial to complete enrolment of cohort 3 in AD[1]**
			✓		**ABATE: First DS[2] data on safety and immunogenicity**
				●	**ABATE Phase 2 trial: interim results (AD; DS)**
ACI-35.030 (Janssen)	pTau		✓		First patient in Phase 2b clinical trial (ReTain)
ACI-7104.056	a-syn[3]		✓		**Phase 2 VacSYn trial in PD[4]: Part 1 interim safety and immunogenicity**
				●	**Phase 2 VacSYn trial in PD: Part 1 interim results, pharmacodynamics, biomarkers**
				○	**Phase 2 VacSYn trial in PD: Part 2 Initiation[5]**
Monoclonal antibodies and small molecule drugs					
Monoclonal antibody	TDP-43[6]	✓		○	Reg. tox studies completed; validated pharmacodynamic assay for clinical readout
Morphomer-NLRP3 (ACI-19764)	NLRP3[7]		✓	●	Lead candidate declaration; ***In vivo* PoC**
				○	IND[9]/CTA[10] filing
Morphomer-Tau	Tau			●	***In vivo* PoC study** and initiation of IND-enabling studies
morADC	Platform (a-syn)			●	***In vivo* PoC study** of proprietary brain delivery platforms
Diagnostics					
TDP-43-PET[11] tracer	TDP-43		✓	●	Phase 1 initiation; initial readout in genetic FTD
a-syn-PET tracer (ACI-15916)	a-syn		✓		PD candidate, IND-enabling studies completed
				●	Phase 1 readout

(1) Alzheimer's disease; (2) Down syndrome; (3) alpha-synuclein; (4) Parkinson's disease; (5) IND/CTA approval; (6) TAR DNA-binding protein 43; (7) (NOD)-like receptor protein; (8) Central nervous system; (9) Investigational new drug; (10) Clinical Trial Application; (11) Positron emission tomography

AC Immune strong Balance Sheet

Operations well-funded into 2027

Cash of **CHF 182.5 million**[1]

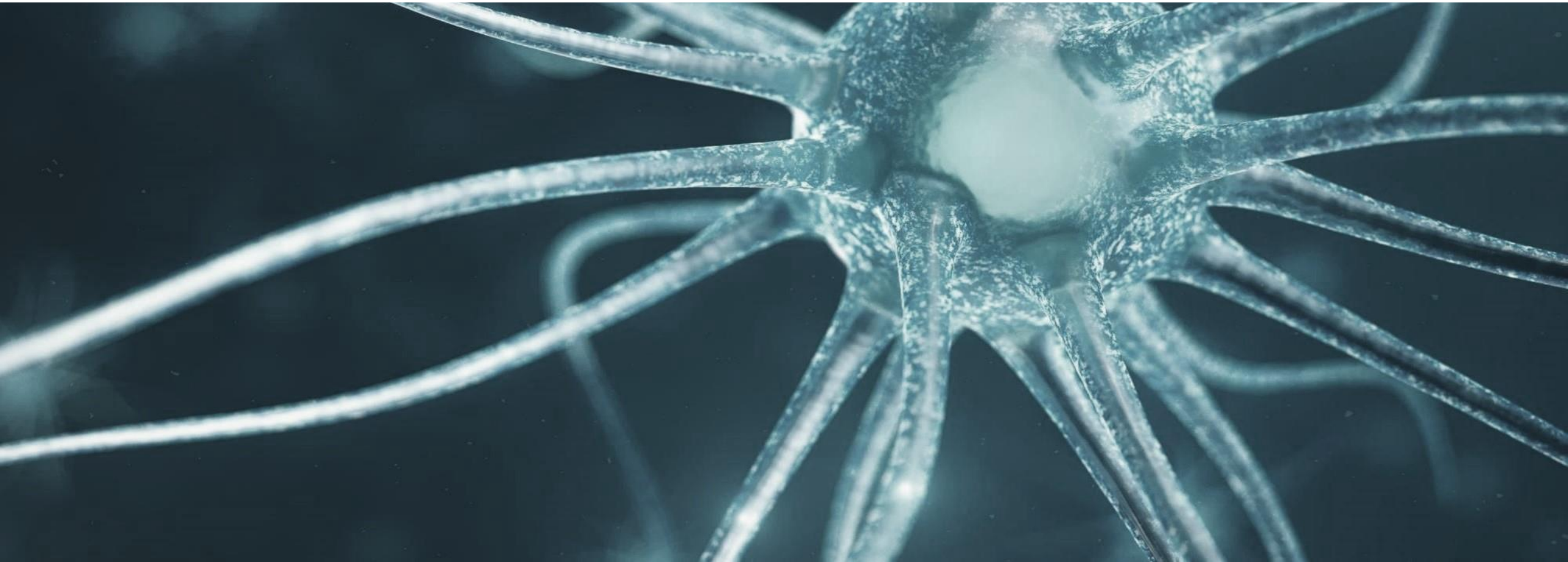
2024 annual cash burn guidance
CHF 65m – 75m

Strong Balance Sheet[2]
Cash runway into 2027

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

(1) Includes CHF157.9 million as of Sep 30, 2024 and CHF24.6 m milestone payment received from J&J mid-October; (2) Assumes no other milestones or deals included

Alpha-synuclein-targeted programs for Parkinson’s disease



Pioneering a-syn[1] modalities to address Parkinson's disease

Unique pipeline assets: 3 therapeutics and 2 diagnostics

- **Active immunotherapy** targeting pathological oligomeric a-syn to prevent spreading and neurodegeneration
- In **Phase 2 trial** in early PD[2] with safety and immunogenicity data reported in H2 2024

ACI-7104.056

Mor-a-syn

- **Small molecule Morphomer®** targets intracellular pathological a-syn aggregates to treat and prevent Parkinson's disease
- **Lead candidate decision** anticipated in 2025

- **Small molecule Morphomer® ADC**[3] with enhanced **brain penetration** and **potency** compared to either parent molecule
- **New Platform technology**: Lead discovery

morADC

ACI-12589
ACI-15916

- **Morphomer® diagnostic PET**[4] **tracers** for pathological a-syn aggregates to detect and differentiate a-synucleinopathies
- Excellent selectivity with **IND**[5]**-enabling studies**[6] completed in H2 2024

- Parkinson's disease affects over 6 million people worldwide
- Challenges remain in diagnosis and there are substantial unmet needs for effective therapeutic interventions

(1) alpha-synuclein; (2) Parkinson's disease; (3) Antibody drug conjugate; (4) Positron emission tomography; (5) Investigational New Drug; (6) IND-enabling studies for ACI-15916

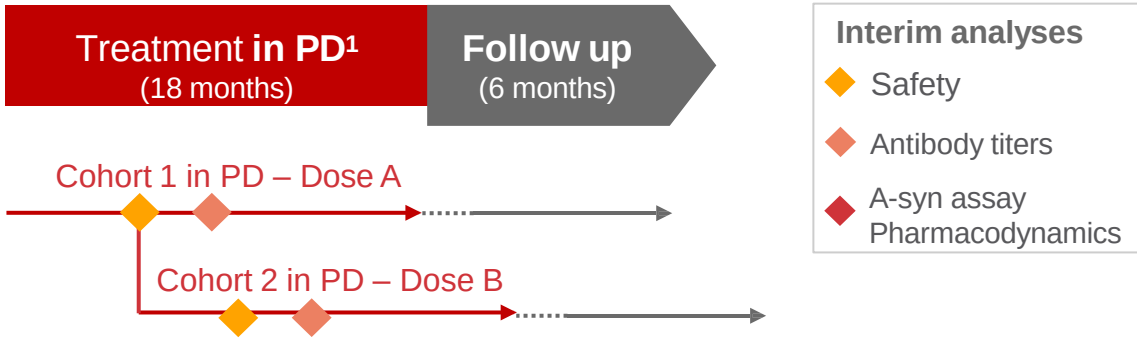
VacSYn: Adaptive biomarker-based Phase 2 study of ACI-7104 in early PD

Placebo-controlled Phase 2 Study Overview – Interim data in H2 '24

- 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

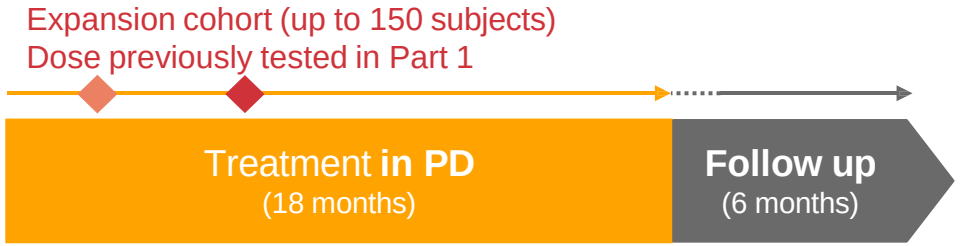
Part 1: Safety & PK/PD

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



Part 2: Proof of Concept in Early PD

- Motor and Non-Motor Functioning (UPDRS[2] based)
- Degeneration of dopaminergic terminals (DaT SPECT[3] imaging)
- Advanced MRI (including ASL[4] and DTI[5])
- Digital biomarkers of motor and non-motor function
- Functional and patient reported outcomes



(1) 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; (2) Unified Parkinson's disease rating scale; (3) Dopamine Transporter Single Photon Emission Computed Tomography; (4) Arterial spin labeling; (5) Diffusion tensor imaging

VacSYn: Patient baseline characteristics and interim safety/tolerability findings

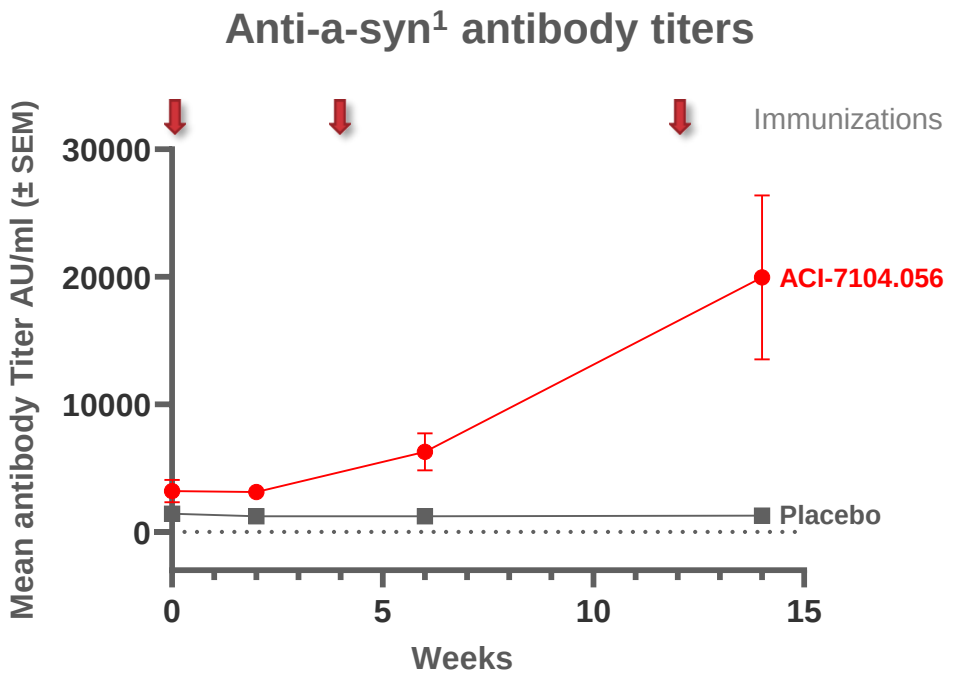
Placebo-controlled Phase 2 Study: No safety concerns raised by DSMB

Baseline profile	Unit	Total[1]
Total number of patients	n	34
Age	Years mean (std)	62.1 (6.7)
Sex		
Male	n (%)	22 (65%)
Female	n (%)	12 (35%)
Hoehn and Yahr stage		
Stage I	n (%)	16 (47%)
Stage II	n (%)	18 (53%)
MDS-UPDRS scores		
Part 1: Non-motor experiences of daily living	mean (std)	4.09 (3.1)
Part 2: Non-motor experiences of daily living	mean (std)	4.09 (3.2)
Part 3: Motor examination	mean (std)	21.09 (9.8)
PD Treatment		
treatment-naïve	n (%)	11 (32%)
on L-Dopa 300mg/day	n (%)	23 (68%)

1. **Overall good safety/tolerability to date**
2. **To date, no death, no serious adverse event and no severe adverse event**
3. **One AE leading to discontinuation from the study[2] unrelated to study drug**
4. **Most common AEs are transient: Injection Site Reactions (49%) and headaches (18%)**
5. **No MRI, lab, ECG abnormalities**

(1) cut-off date September 22, 2024; (2) Worsening of generalized anxiety disorder unrelated to study drug;

ACI-7104.056 VacSYn Phase 2 trial interim results



Strong increase in anti-a-syn antibodies
(after third immunization)

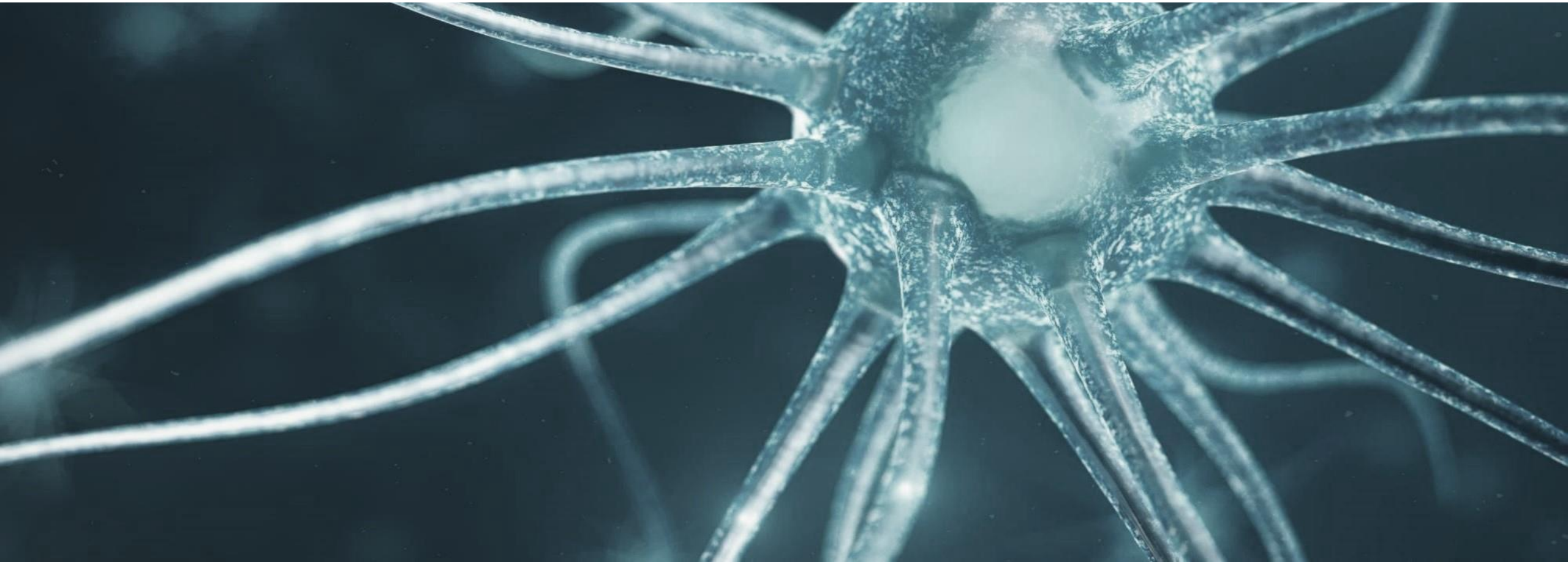
Key results

- Antibody titers against a-syn peptide evident after **2 immunizations**
- ACI-7104.056-treated patients showed an average **16-fold increase**[2] above placebo in anti-a-syn antibodies after 3 immunizations
- No anti-a-syn antibody responses were observed in placebo-treated subjects
- To date, no clinically relevant safety issue reported

(1) alpha-synuclein (peptide aa 115-121); (2) assay background level defined by signal in the placebo group

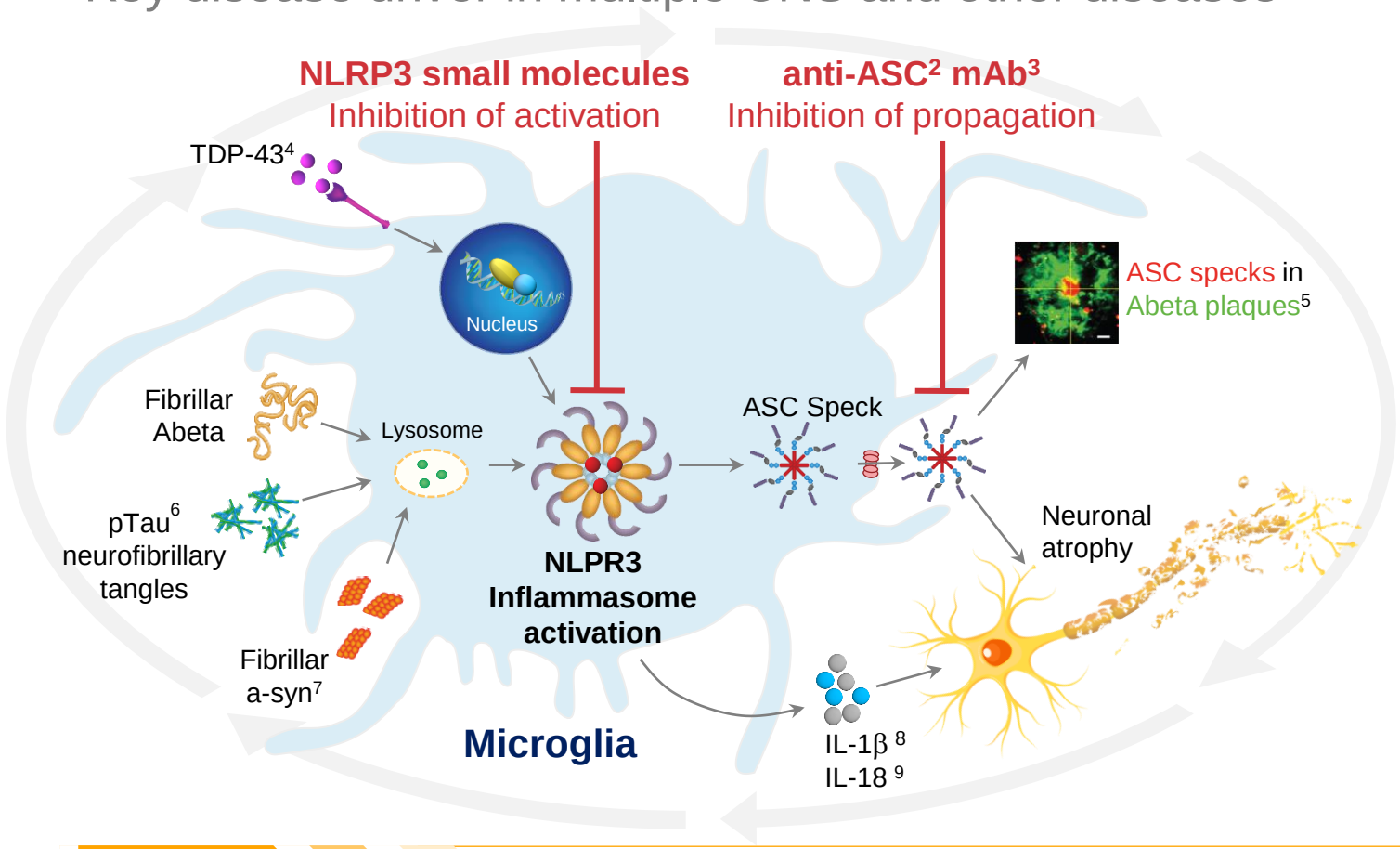
Multiple assets in pipeline targeting NLRP3

For neuroinflammatory conditions like Parkinson’s disease and obesity



NLRP3[1] Inflammasome is a promising therapeutic target

Key disease driver in multiple CNS and other diseases



AC Immune's unique positioning

- Potential best-in-class compounds
  - Small molecule therapeutics
  - Monoclonal antibody diagnostics
- Multiple substantial indications

CNS[10]
- Parkinson's disease
- Alzheimer's disease
- Multiple sclerosis
- Other NDDs[11]

Other
- Obesity
- Type 2 diabetes
- Rheumatoid arthritis
- IBD[12]

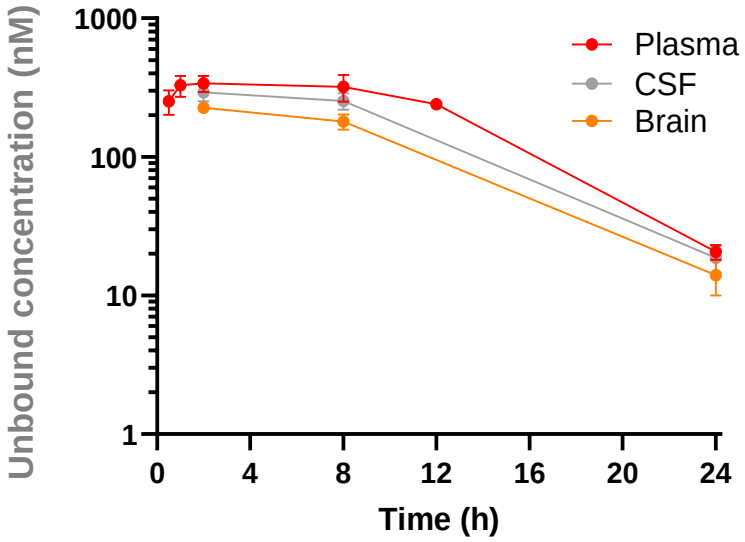
- Mechanism of action can be applied across a broad range of neuroinflammatory and other diseases
- Pharmacological inhibition of NLRP3 lowers aberrant cytokine release and reduces disease pathology [13,14,15]

(1) Nod-Like Receptor protein containing Pyrin 3; (2) Apoptosis-associated speck-like protein containing a CARD, also called PYCARD; (3) monoclonal antibody; (4) TAR DNA binding protein-43; (5) Venegas *et al.*, 2017; (6) phosphorylated Tau; (7) alpha-synuclein; (8) Interleukin-1 beta; (9) Interleukin-18; (10) Central nervous system; (11) neurodegenerative diseases; (12) Inflammatory bowel disease; (13) Stancu *et al.*, 2019; (14) Dempsey *et al.*, 2018; (15) Gordon *et al.*, 2018

ACI-19764 small molecule candidate targeting NLRP3 [1]

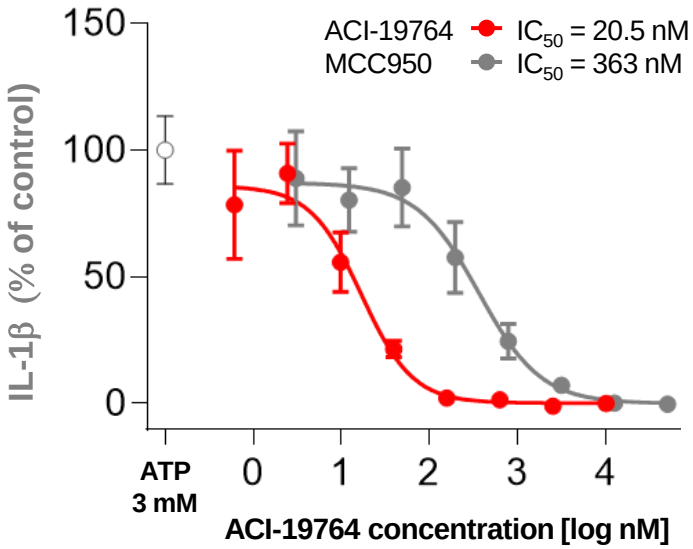
Preclinical efficacy demonstrated in mouse models of neuroinflammation

Brain penetration



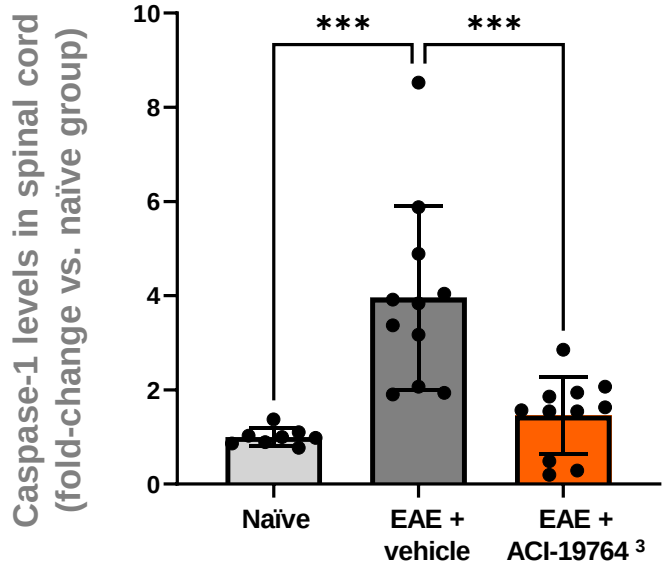
Highly brain penetrant molecule with a brain/plasma[4] ratio of 0.7 after a single oral dose in rats (5 mg/kg)

Human whole blood IL-1β



ACI-19764 exhibits low nM efficacy in translationally relevant *in vitro* assays

Multiple sclerosis model (EAE[2])



Potent NLRP3 inhibition leads to reduction in pro-pyroptotic caspase-1

- Mechanism of action can be applied across a broad range of neuroinflammatory diseases including: AD[5], PD[6], ALS[7], FTD[8], MS[9] and traumatic brain and spinal cord injury

(1) (NOD)-like receptor protein 3; (2) Experimental autoimmune encephalomyelitis; (3) Dosed at 5mg/kg in medicated chow; (4) Kp,uu – unbound partition coefficient; (5) Alzheimer's disease; (6) Parkinson's disease; (7) Amyotrophic lateral sclerosis; (8) Frontotemporal dementia; (9) Multiple sclerosis

ACI-19764 small molecule candidate targeting NLRP3

Excellent brain penetrance, safety and efficacy (preclinical tox / PK data)

Optimal brain PK and developability

- Mouse brain Kp,uu[1] : 0.3
- Rat brain Kp,uu : 0.7
- Dog CSF[2] Kp,uu : 1
- BCS[3] class 1

- **Predicted human oral dose below 100mg/day**

High potency and selectivity

- Strong IL-1β[4] inhibition
  - Human macrophages IC_{50} **:** 2nM
  - Human WB[5] IC_{50} **:** 20.5nM
  - Mouse WB IC_{50} **:** 84.4nM
  - Mouse microglia IC_{50} **:** 5.6nM
- *In vivo* (EAE[6] model) inhibition of inflammation (IL-1β; caspase-1; GFAP[7]; CD4[8])
- No inhibition of other types of inflammasome[9]

Excellent safety and tolerability

- No adverse findings up to 400 mg/kg in short toxicology rat study
- No adverse effects upon chronic treatment in several *in vivo* studies
  - Tg83 mice (3 months)
  - EAE mice (30 days)
  - DIO mice (28 days)

- ACI-19764 shows excellent brain penetration, efficacy, safety, and developability profile
- IND-enabling studies to be completed in 2025

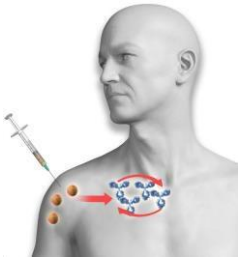
(1) Optimal brain to plasma ration (Kp,uu) = 1.0; (2) cerebrospinal fluid; (3) Biopharmaceutics Classification System; class 1 defines high soluble and high permeable drugs (4) interleukin-1 beta; (5) whole blood; (6) Experimental autoimmune encephalomyelitis; (7) Glial fibrillary acidic protein; (8) cluster of differentiation 4, marker of T helper cells; (9) AIM2, NLRP1, NLRC4

ACTIVE Immune Therapy

targeting neurodegenerative diseases

Major advantages

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

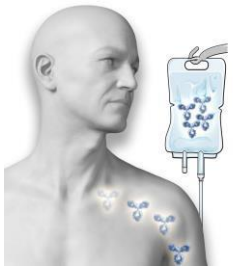


Active immunotherapy

Stimulates the patient's immune system to produce their own antibodies

Passive immunotherapy

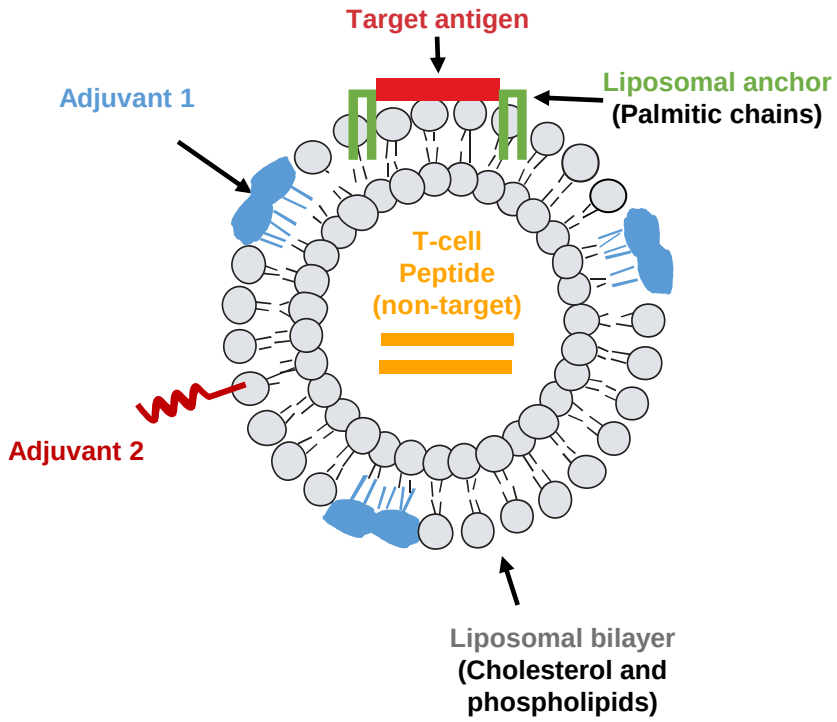
Externally generated mAb requires administration every two to four weeks



(1) Amyloid-related imaging abnormalities-edema

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

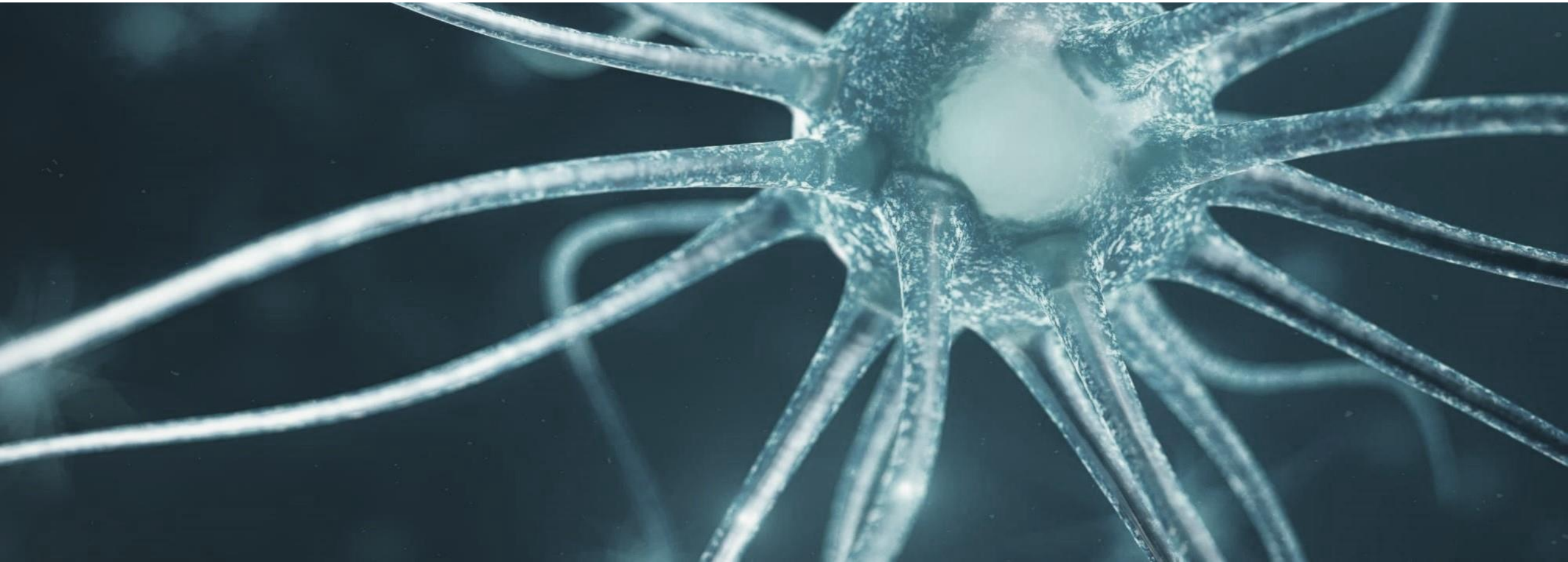
Immunogenicity	✓
Target & conformation specificity	✓
Avidity increase over time	✓
Sustainable response	✓
Boostable response	✓

- 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

ACTIVE Immune Therapy

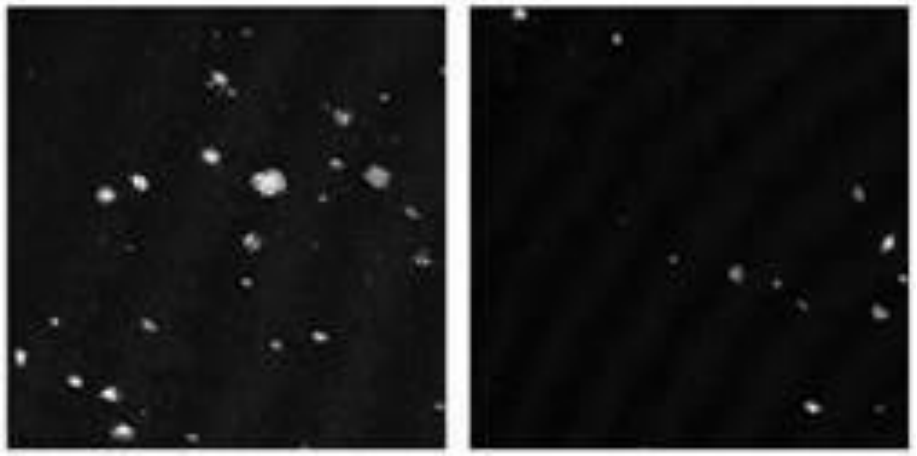
ACI-24.060: Anti-Abeta for Alzheimer’s disease partnered with Takeda



ACI-24 active immunotherapy reduces Abeta plaque burden

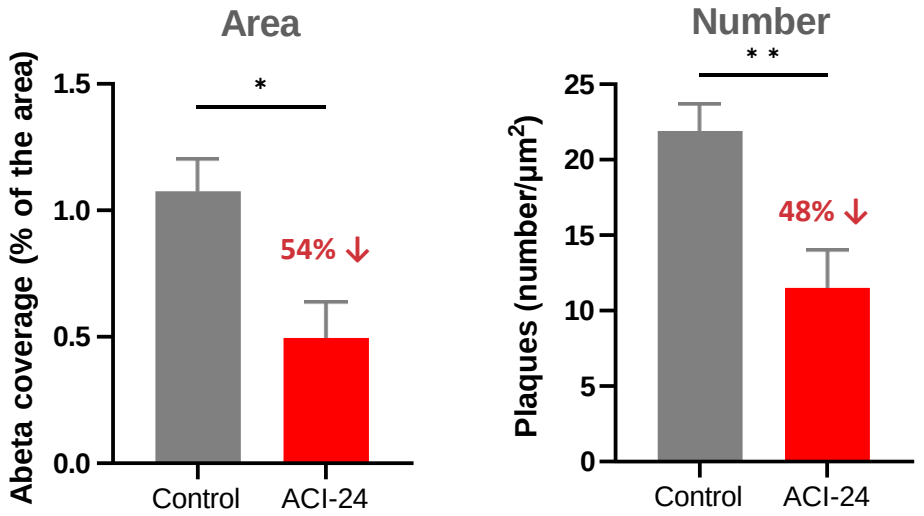
Significant Abeta plaque reduction *in vivo* in preclinical APPxPS1 model[1]

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

ABATE: 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

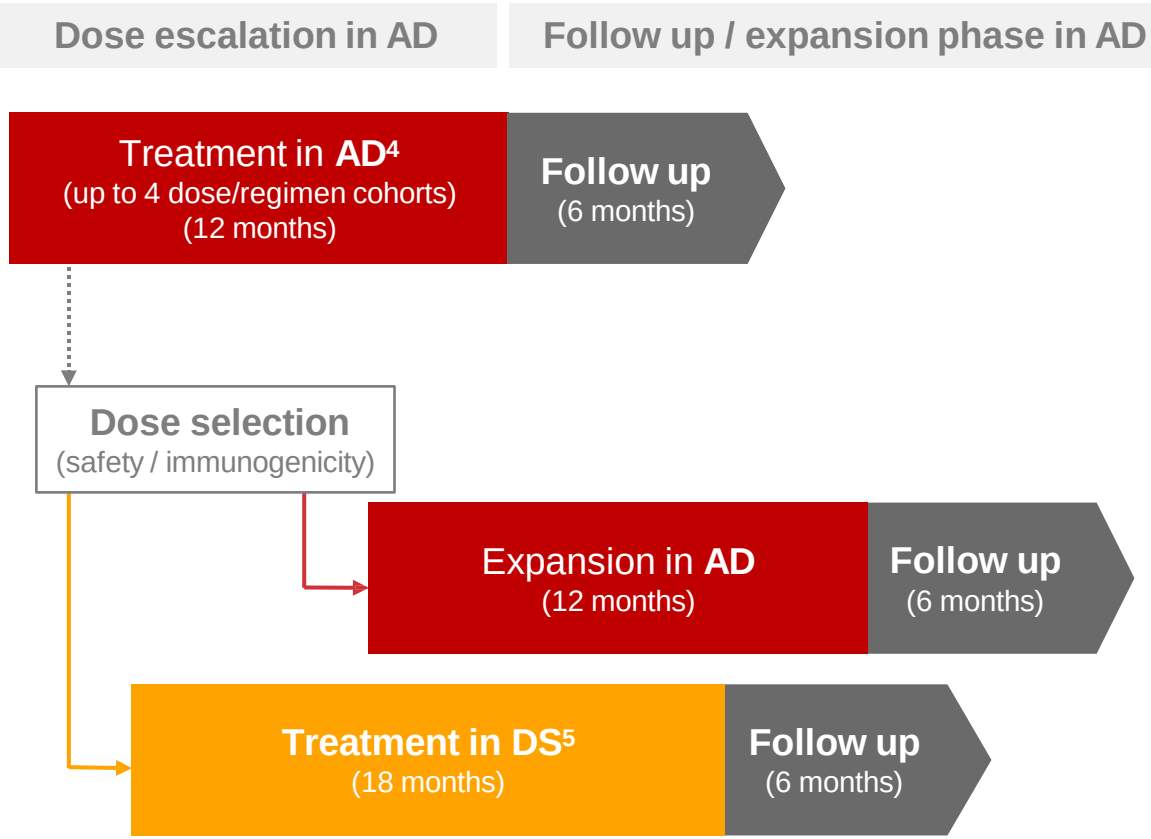
AD
- Up to 4 different doses and/or dose regimens
- Expansion of one cohort to assess effect on Abeta PET

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

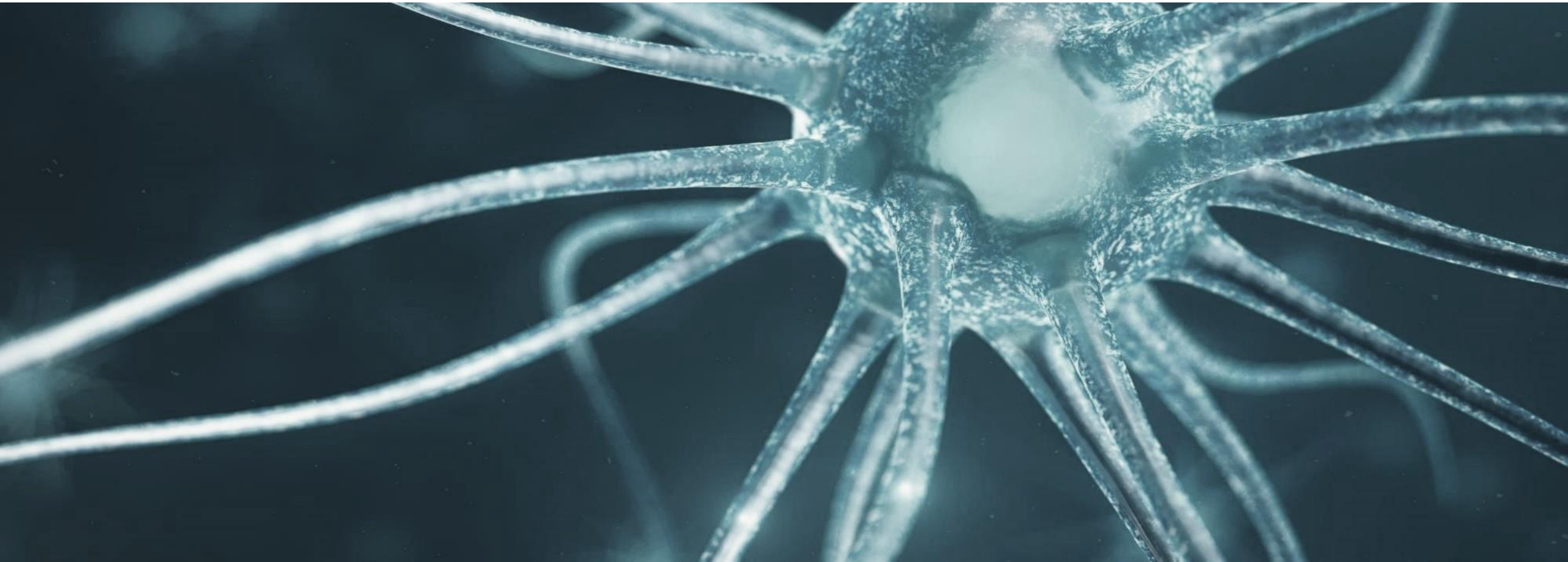
Trial Schematic



(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

ACTIVE Immune Therapy

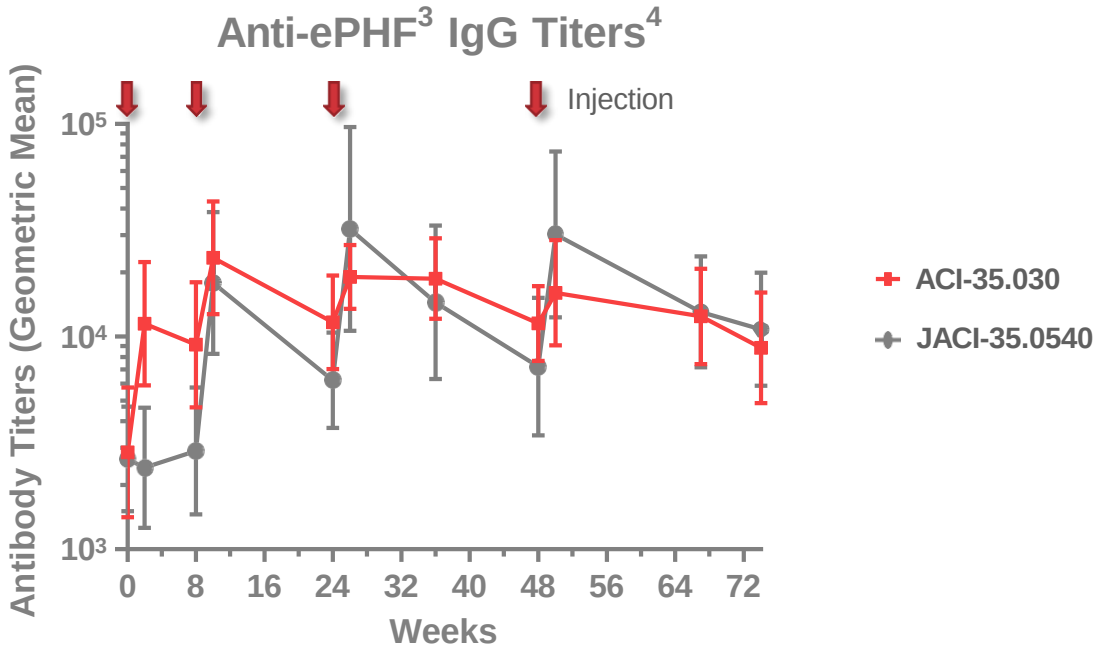
ACI-35.030: Anti-pTau for Alzheimer's disease partnered with janssen



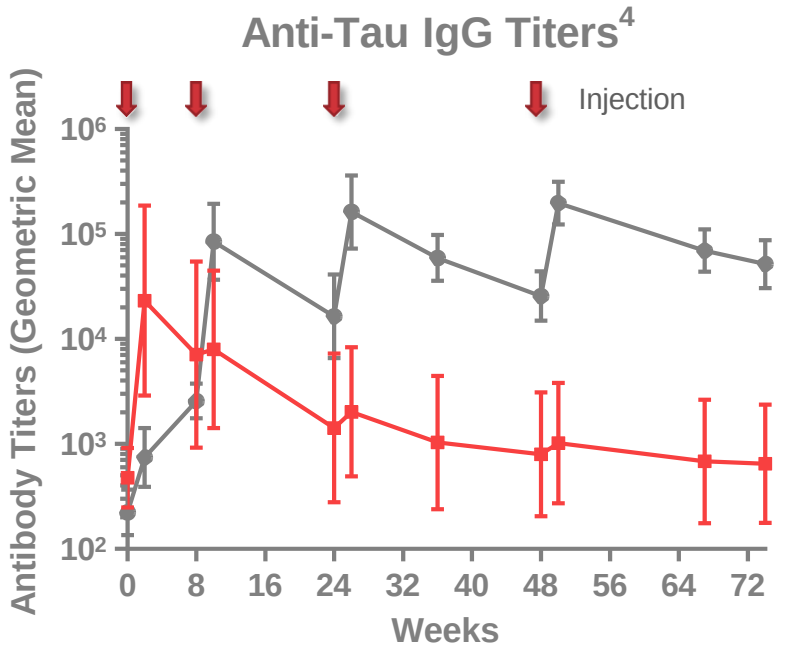
ACI-35.030 selected for further development by partner Janssen

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

ACI-35.030 and JACI-35.054 utilized the same pTau[1] epitope – compared head-to-head in Phase 1b/2a trial in AD[2] patients



ACI-35.030 induced anti-ePHF Ab[5] responses in 100% of patients after 1st injection



ACI-35.030-induced Abs against normal Tau returned to baseline levels as Ab response matured

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

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

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

Study population

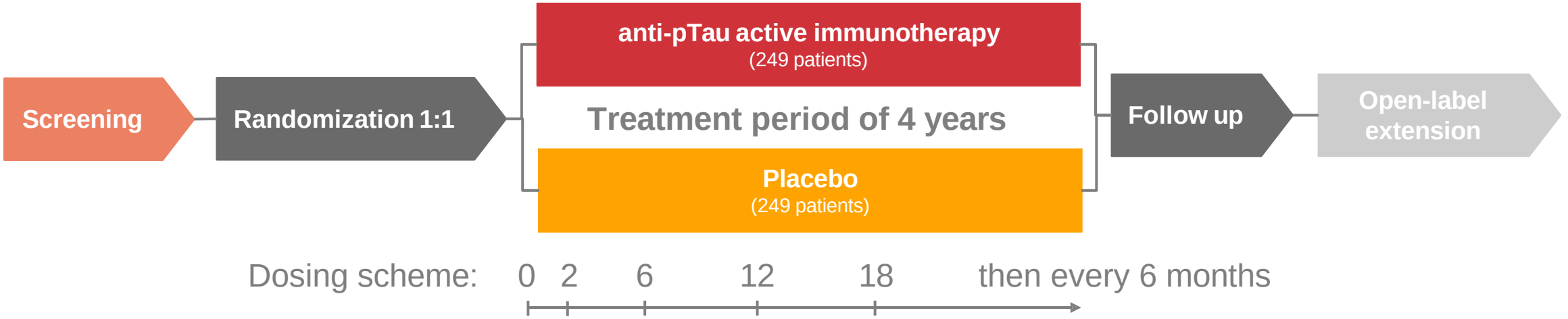
- ~500 participants with **preclinical AD**:
  - Cognitively normal
  - Tau PET positive
  - Amyloid positive[2]
- Prior to appearance of clinical symptoms

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

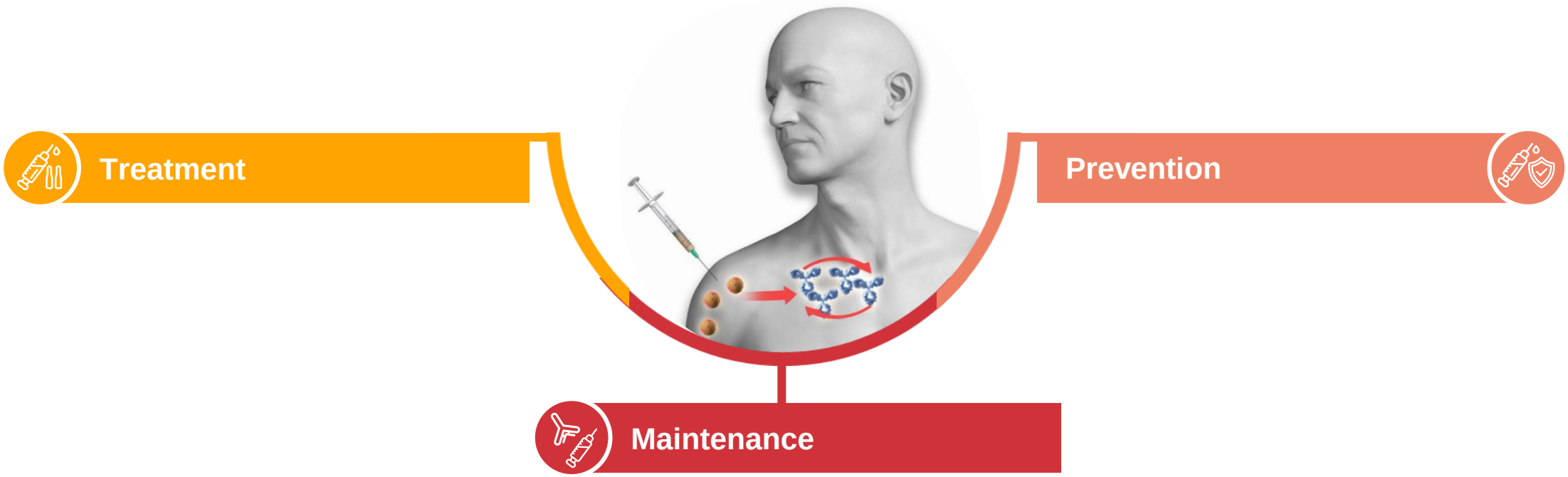
- Preclinical AD Cognitive Composite 5[4]:
  - Episodic memory
  - Timed executive function
  - Global cognition
- Potential **traditional approval**



(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

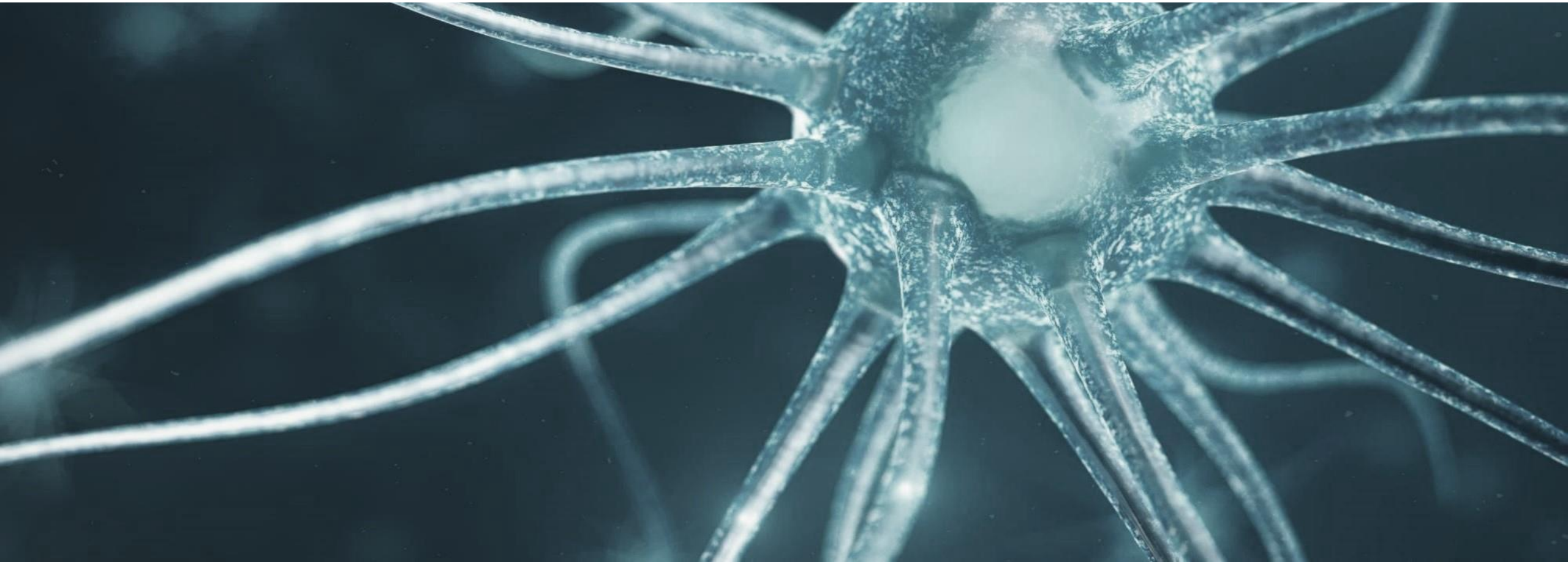
Active immunotherapy: a new class of treatment for neurodegenerative disease

Potential for profound social and economic impact



ACTIVE Immune Therapy for global treatment and prevention of neurodegenerative diseases

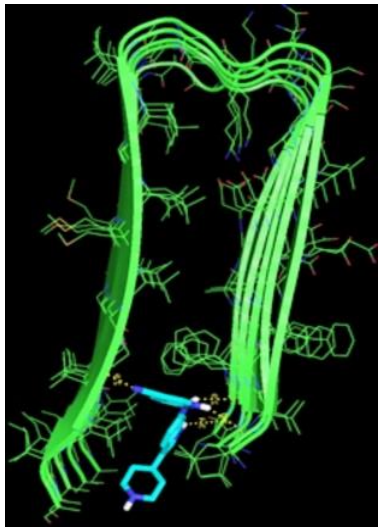
Tracers from the Morphomer® platform

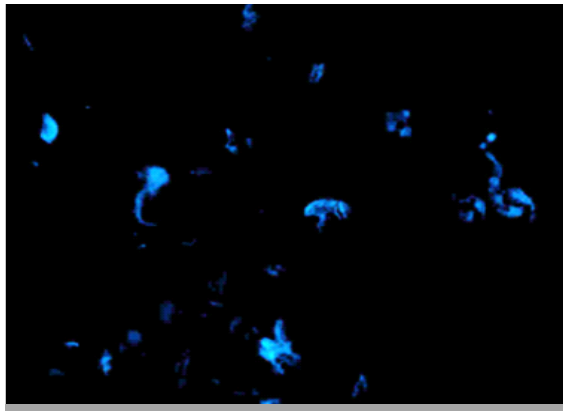


Precision medicine approach enabled by the Morphomer® platform

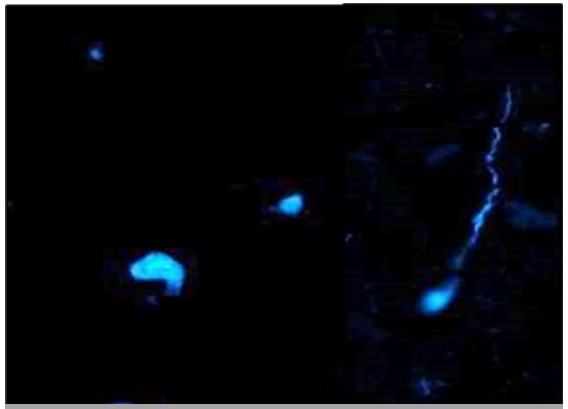
Developing a suite of tracers against emerging targets in neurodegenerative diseases

	Indication	Candidate	Partner	Modality	Discovery	Preclinical	Phase 1	Phase 2	Phase 3
Wholly-owned	Parkinson's disease	ACI-15916		*a-syn-PET[3] tracer (diagnostic)*					
Wholly-owned	MSA[4]	ACI-12589		*a-syn-PET tracer (diagnostic)*					
Wholly-owned	ALS[5]	ACI-19626		*TDP-43-PET tracer (diagnostic)*					
Partnered	Alzheimer's disease	PI-2620	Life Molecular Imaging	*Tau-PET tracer (diagnostic)*					FDA Fast Track

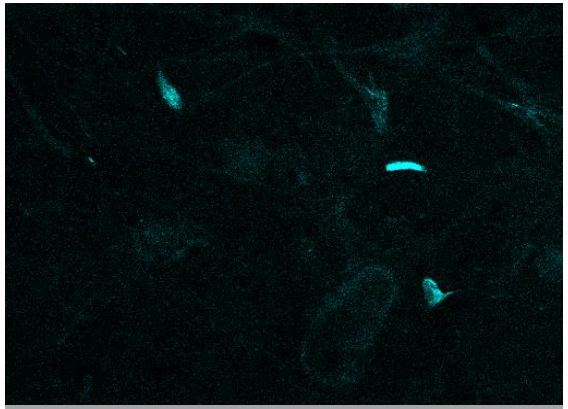




Tau neurofibrillary tangles
PI-2620



a-syn[1] Lewy bodies
ACI-12589

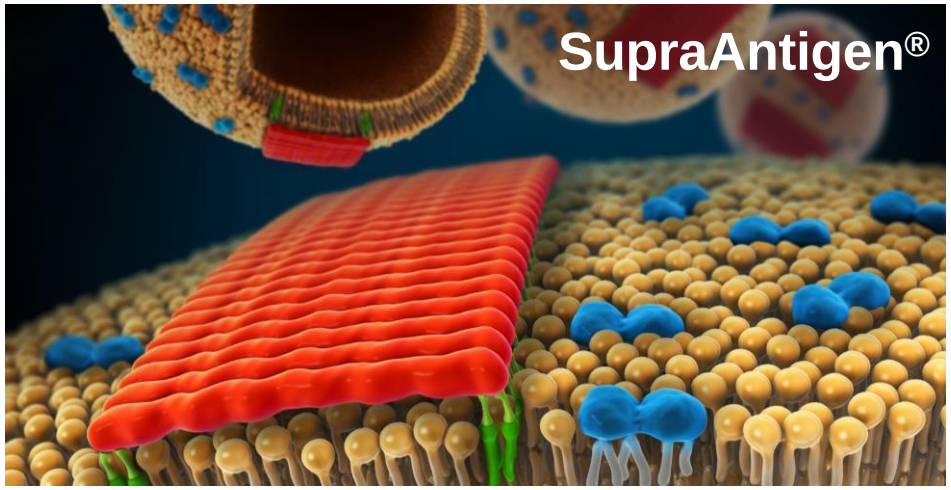
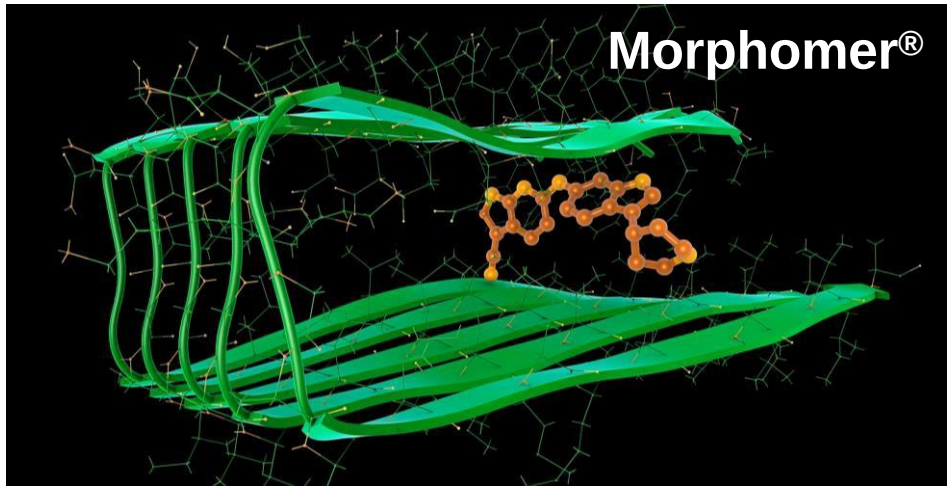


TDP-43[2] inclusions

(1) alpha-synuclein; (2) TAR DNA-binding protein 43; (3) Positron emission tomography; (4) Multiple system atrophy; (5) Amyotrophic lateral sclerosis; (6) Alzheimer's disease

Technology platforms driving value-creating pharma deals

Strategy: optimize value to risk ratio and retain significant upside

Platform		
Wholly-owned Programs	■ a-syn active immunotherapy ■ Anti-TDP-43 mAb[1] ■ Anti-NLRP3-ASC mAb	■ morADC	■ Mor-a-syn ■ Mor-TDP-43 PET / Mor-a-syn PET ■ Mor-NLRP3-ASC

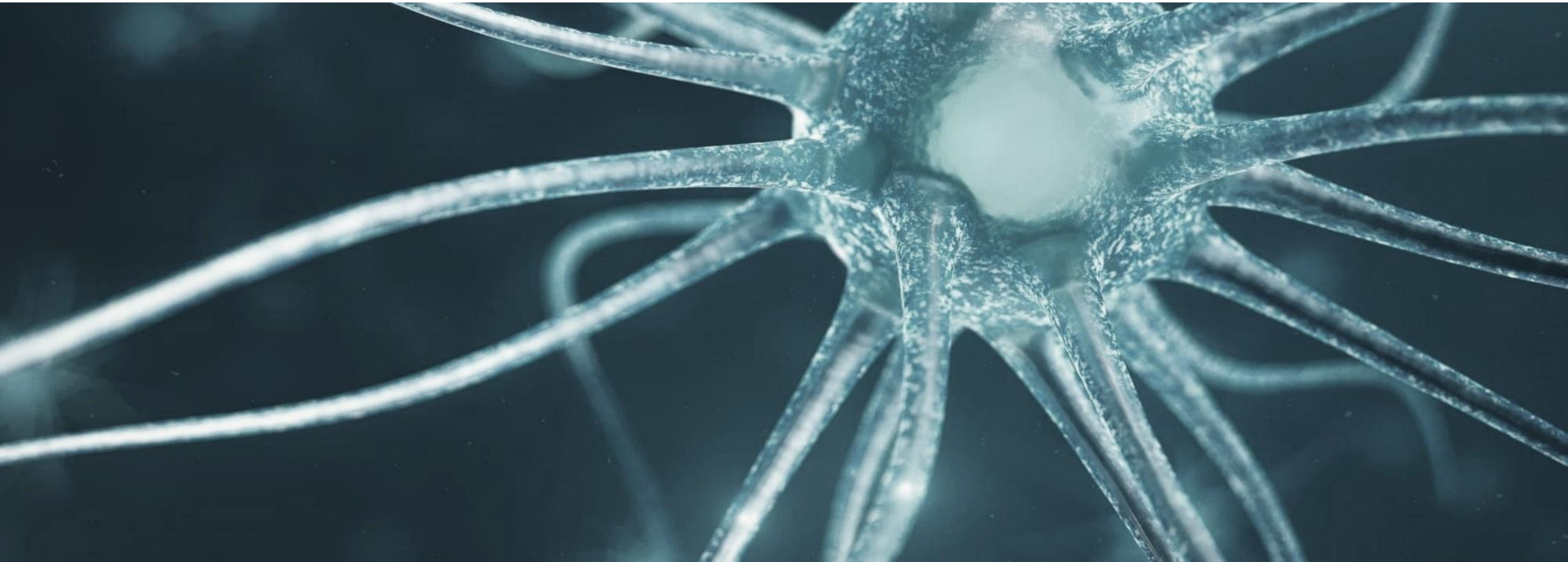
- Over CHF 400 million in upfront payments from deals; further >CHF 4.3 billion possible
- Considerable additional potential value in our unpartnered clinical and preclinical programs

(1) Monoclonal antibody

AC Immune: Pioneering science and precision medicine

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

Supplementary information



AC Immune's strong track record in deals[1] with leading pharma companies

Strategy: optimize value to risk ratio and retain significant upside

Program	Phase	Total value[2]	Upfront[2]	Milestones received[2]	Royalties	Partner
ACI-24.060 (anti-Abeta active immunotherapy)	Phase 1b/2	>USD 2,100	USD 100		Mid-to-high teens	Takeda
ACI-35.030 (anti-pTau active immunotherapy)	Phase 2b	CHF 500	CHF 26	CHF 45	Low-double digits to mid-teens	janssen
Tau Morphomer® drugs	Phase 1 [6]	CHF 1,860	CHF 80 +USD 50 [7]	CHF 40	Low-double digits to mid-teens	Lilly
PI-2620 (Tau PET[4] tracer)	Phase 3 [5]	EUR 160	EUR 0.5	EUR 7	Mid-single digits to low-teens	Life Molecular Imaging
Crenezumab (anti-Abeta antibody)	Phase 2	USD 65 [3]	USD 25	USD 40		*
Semorinemab (anti-Tau antibody)	Phase 2	CHF 59 [3]	CHF 17	CHF 42		*
Total (millions)[8]		**CHF ~4,750**	**CHF 255.2 [9]**	**CHF 172**		

- Outstanding potential milestone payments exceed ~CHF 4.3 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 ; * previously licensed to Genentech (a member of the Roche Group)

morADC platform offers enhanced targeting of brain proteinopathies

Synergistic inhibition of aggregation and seeding and increased brain penetration

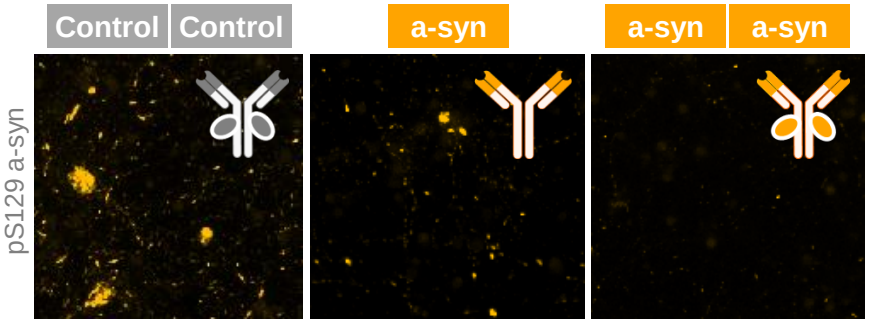
Enables **single** or **dual targeting** strategies to deliver **combination therapy** in a single agent

Single targeted morADC (a-syn/a-syn) shows up to **80x** higher anti-aggregation effects than parental molecules

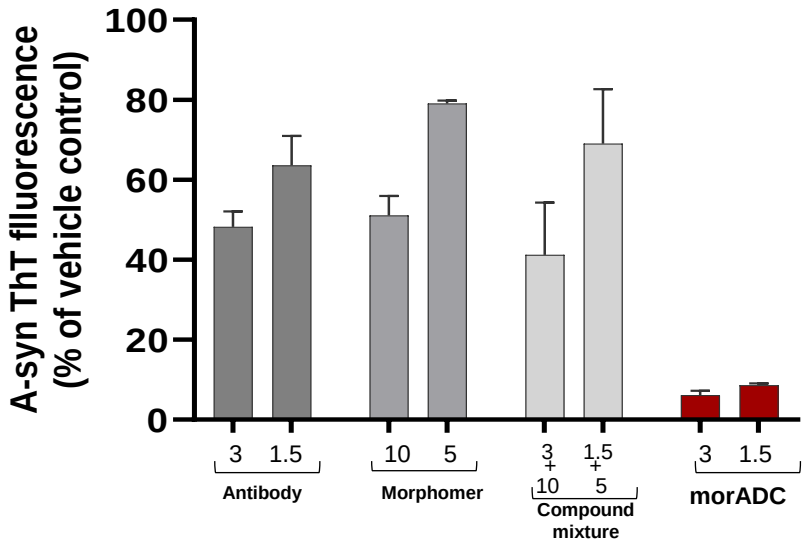
Dual targeted morADC (Abeta/Tau) shows **3x** and **15x** higher anti-aggregation effects than parental molecules

Additionally, **enhanced brain exposure (up to 8x higher)** was observed for the monoclonal antibody within the morADC

Seeding inhibition



Aggregation inhibition



AC Immune unpublished data

- morADCs: important synergistic effects on targeted proteinopathies