Pioneering Precision Medicine for Neurodegeneration

NASDAQ: ACIU | Investor Presentation, June 2023

AC Immune

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AC Immune pioneering new ways to treat neurodegenerative diseases

Combining Precision Medicine and early, targeted treatment

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 3 billion in potential milestones

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

Strong Balance sheet
Funded into Q3 2024

- Based in Lausanne, Switzerland
- ~150 employees
- Listed September 2016 (NASDAQ: ACIU)
- 83.6 million shares outstanding[1]
- Cash of CHF 105.4 million[2] (~USD 115 million)

(1) As of March 31, 2023; excluding treasury shares; (2) As of March 31, 2023

Neurodegenerative diseases represent a large and growing market

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

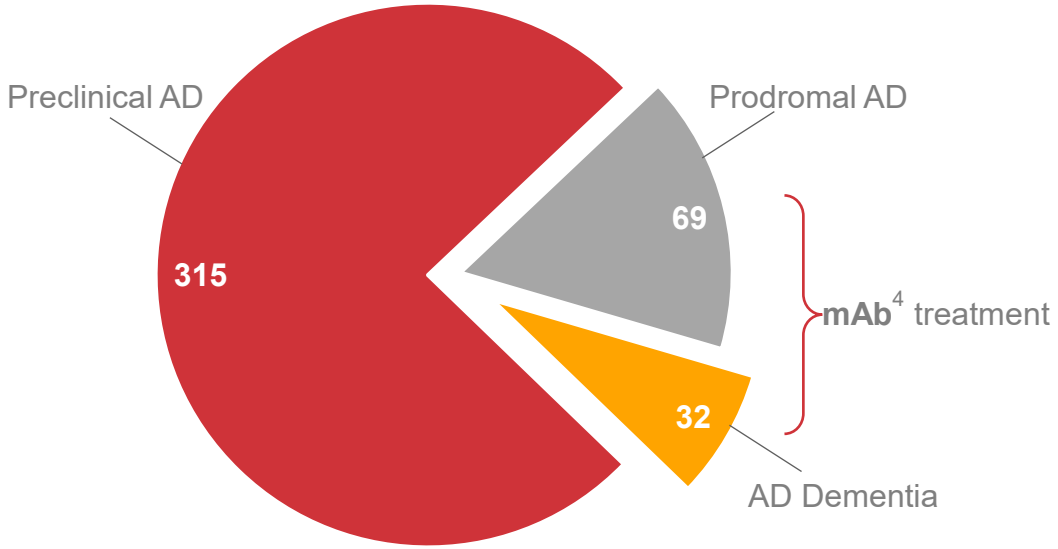
>$1 Trillion global annual cost of dementia[1]

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

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

>400 million people addressable by vaccination

Global Prevalence of AD Stages[2] (million)



Diagnostics enable earlier intervention

Vaccines for treatment and prevention

- AD prevention through combination of earlier diagnosis with early vaccination
- 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

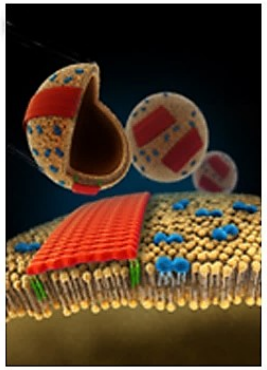
SupraAntigen® and Morphomer® platforms

An integrated approach to Central Nervous System (CNS)-specific therapies

CNS-optimized

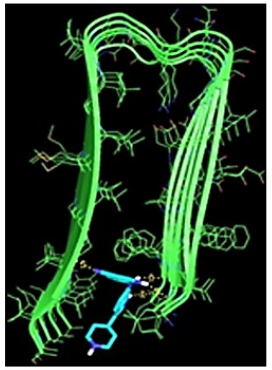
Clinically validated

SupraAntigen®



Vaccines & Antibodies

Morphomer®



Small Molecules

Conformation-specific

Precision medicine enabling

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 340	USD 25	USD 40	Mid-single digits to mid-teens	Genentech A Member of the Roche Group
Biologicals	Semorinemab (anti-Tau antibody)	Phase 2	CHF 430	CHF 17	CHF 42	Mid-single digits to low-double digits	Genentech A Member of the Roche Group
Biologicals	ACI-35.030 (anti-pTau vaccine)	Phase 1b/2a	CHF 500	CHF 26	CHF 5	Low-double digits to mid-teens	Janssen
Small molecules	Tau PET[3] imaging agent	Phase 3[4]	EUR 160	EUR 0.5	EUR 7	Mid-single digits to low-teens	Life Molecular Imaging
Small molecules	Tau Morphomer® small molecules	Phase 1[5]	CHF 1,860	CHF 80 +USD 50[6]	CHF 40	Low-double digits to mid-teens	Lilly
	Total (millions)[7]		**CHF ~3,311**	**CHF 155.2[8]**	**CHF 132.4**		

- Outstanding potential milestone payments exceed CHF 3 billion

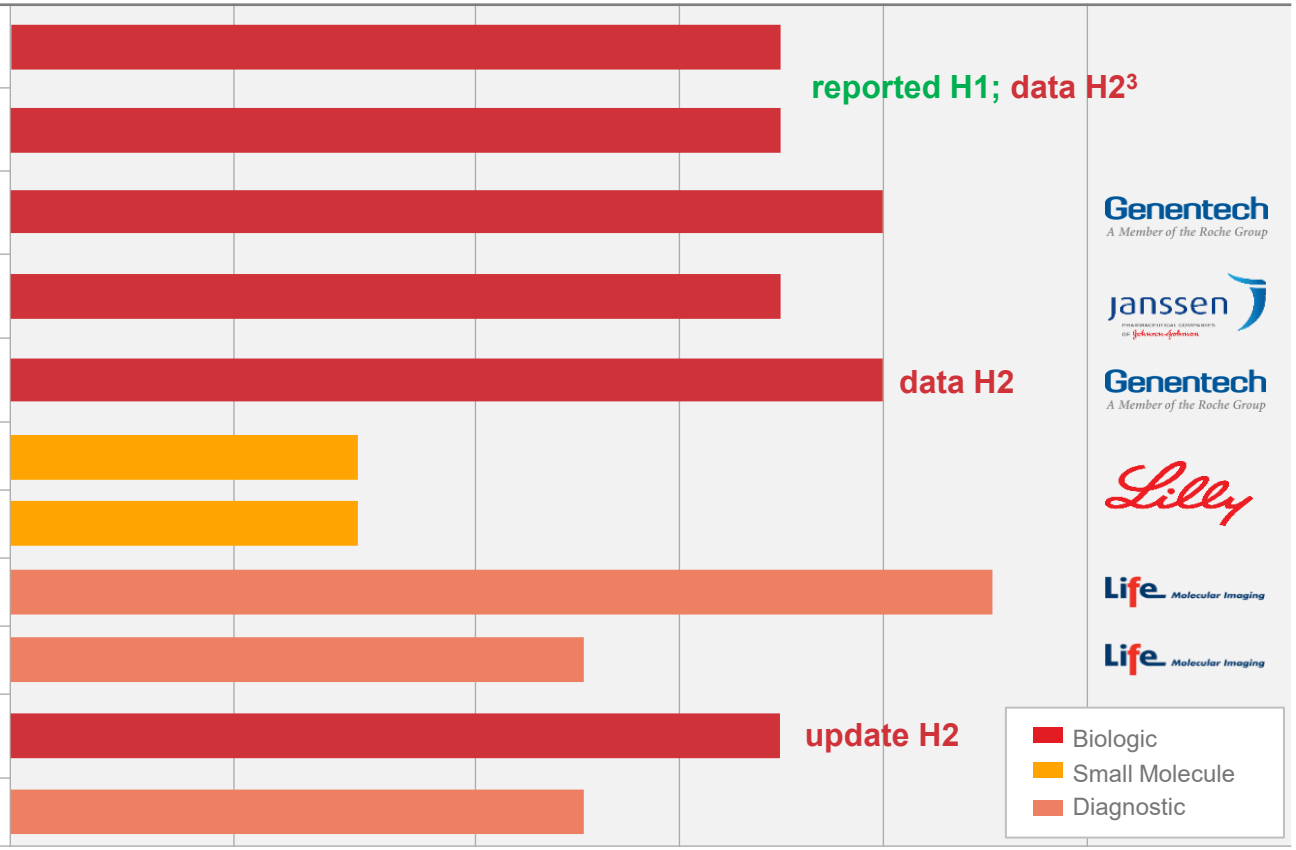
(1) Disclosure limited due to confidentiality agreements with collaboration partners; (2) In millions; (3) Positron emission tomography; (4) In Alzheimer's disease; (5) Phase 1 completed; (6) Equity investment; (7) Converted to CHF on date of receipt; (8) Excludes convertible note agreement of USD 50 million

Broad and robust pipeline in neurodegenerative diseases

Driven by validated proprietary technology platforms for sustained growth

Clinical Stage Programs

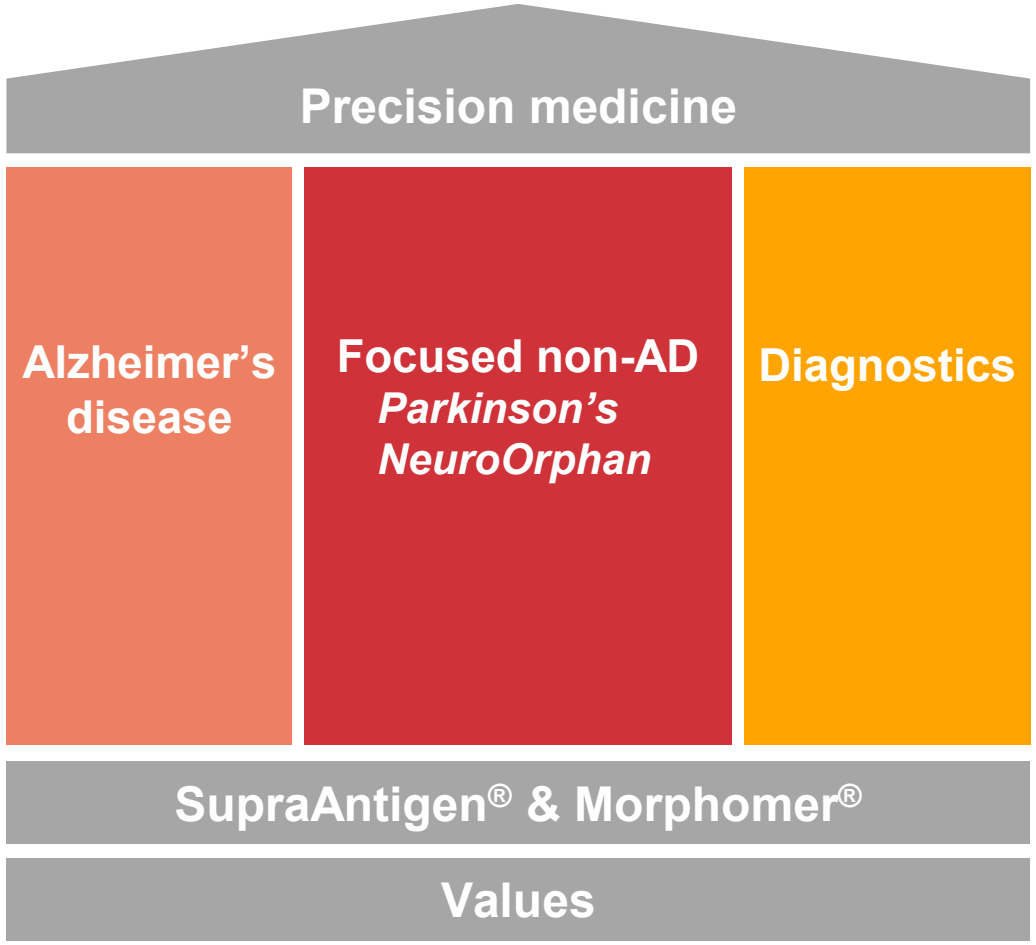
TARGET	PRODUCT CANDIDATE	INDICATION	PARTNER
Abeta	**ACI-24.060** *(anti-Abeta vaccine)*	AD[1] treatment	
		AD treatment (*Down syndrome[2]*)	
	Crenezumab *(anti-Abeta antibody)*	AD prevention[4]	Genentech
Tau	**ACI-35.030** *(anti-pTau vaccine)*	AD treatment	Janssen
	Semorinemab *(anti-Tau antibody)*	AD treatment (*mild-to-moderate)[5]*	Genentech
	Morphomer® Tau aggregation inhibitor	Rare Tauopathies	Lilly
		AD treatment	
	Tau-PET[6] tracer	AD diagnostic	Life Molecular Imaging
		PSP[7] diagnostic	Life Molecular Imaging
a-syn[8]	**ACI-7104.056** *(anti-a-syn vaccine)*	PD[9], a-synucleinopathies	
	a-syn-PET tracer	a-synucleinopathies (e.g. MSA[10])	



(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-26.060 in patients with AD and patients with Down syndrome; (4) Prevention trial API-ADAD in Colombia; (5) Open label extension study is ongoing; (6) Positron emission tomography; (7) Progressive supranuclear palsy; (8) alpha-synuclein; (9) Parkinson's disease; (10) Multiple system atrophy

Business Strategy 2023: advancing vaccine and non-AD portfolio

Focus on delivering Precision Medicine to enhance value creation



Alzheimer's disease

- Accelerate development of novel late-stage therapies with partners
- Accelerate wholly-owned optimized anti-Abeta vaccine (ACI-24.060) with parallel development in AD[1] and DS[2]

Non-AD and NeuroOrphans

- Increase strategic focus in non-AD to Parkinson's disease
- Advance anti-a-syn[3] vaccine into late-stage development

Diagnostics for precision medicine

- Advance our differentiated diagnostic pipeline for Parkinson's disease and TDP-43[4]-based pathologies

(1) Alzheimer's disease; (2) Down syndrome; (3) Alpha-synuclein; (4) TAR DNA-binding protein 43

Key milestones for value creation in 2023

Multiple clinical readouts for wholly-owned vaccines

✓ Achieved
● Clinical readouts
○ Other development events

Vaccines		H1	H2	
ACI-24.060	Abeta	✓		Initiation of Down syndrome cohort of Phase 1b/2 ABATE study
		✓		IND submission to enable expansion of ABATE study to U.S.
		✓	●	**Two interim analyses in AD[1] – safety, immunogenicity**
			●	**Interim analysis in Down syndrome – safety, immunogenicity**
ACI-35.030	Tau		○	Further development with initiation of next trial in AD followed by milestone payment
ACI-7104.056	a-syn[2]		●	**Phase 2 VACSYN study in PD update**
Monoclonal antibodies				
Semorinemab	Tau		●	**Phase 2 Lauriet Trial Open Label Extension results**
Monoclonal antibody	TDP-43[3]		○	Candidate into preclinical development (tox)
Diagnostics				
a-syn-PET[4] tracer	a-syn		○	Next clinical candidate declaration for PD[5]
TDP-43-PET tracer	TDP-43	○		Clinical candidate declaration

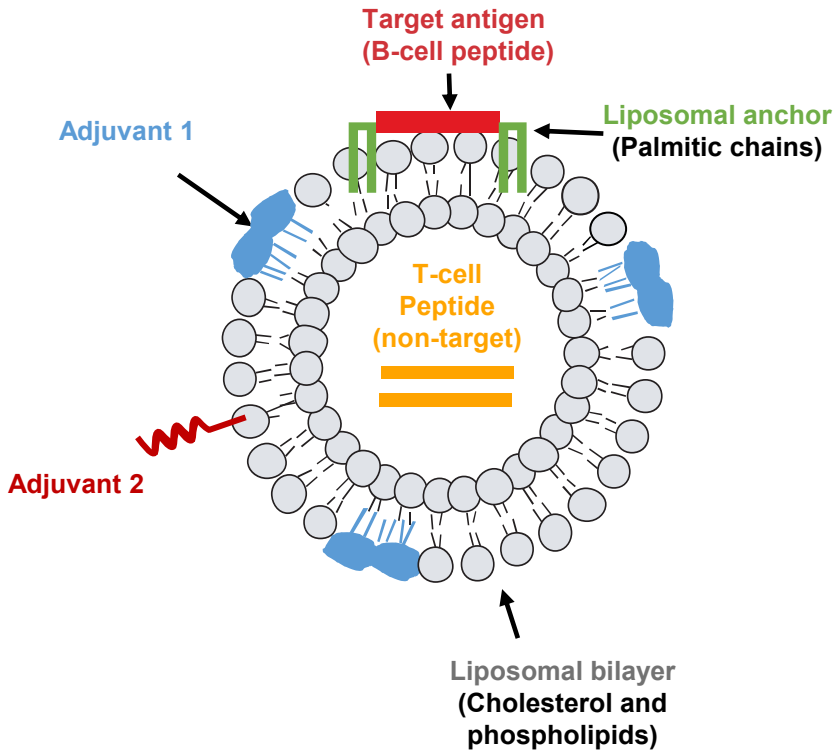
Amyloid-PET Data in H1 2024

(1) Alzheimer's disease; (2) Alpha-synuclein; (3) TAR DNA-binding protein 43; (4) Positron emission tomography; (5) Parkinson's disease

Vaccine programs targeting neurodegenerative diseases

Disruptive potential of SupraAntigen®-V

Optimized vaccines delivering superior results in neurodegenerative diseases



Generates target-specific antibody response

Safely engages target-unrelated T-cells to enhance & maintain response

Unprecedented Clinical Performance

Immunogenicity	++++[1]
Target specificity	++++[2]
Conformation specificity	+++
Avidity increase over time	+++
Sustainability of response	+++
Boosting	+++
Class switching IgM to IgG	+++
Evidence of memory B cells	+++

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

(1) 100% response after 1st injection; (2) Increases over time

SupraAntigen® vaccines offer significant advantages over mAbs[1]

Vaccine–based approach provides opportunity to prevent neurodegenerative diseases globally

SupraAntigen® vaccines	Monoclonal antibodies
Consistent, long-lasting immunity	Transient effect
Limited dosing (annual or bi-annual)	More frequent dosing (bi-weekly or monthly)
No observed ARIA-E[2] to date	ARIA-E rates of concern[3]
Cost-effective	High costs (per patient per year)
Improved access (administration, logistics)	Infrastructure, infusion inconvenience, monitoring

- AD prevention by combining early diagnosis with early vaccination potentially superior to mAb treatment
- Vaccines are believed to be the only realistic possibility for global prevention of neurodegenerative diseases

(1) Monoclonal antibodies; (2) Amyloid-related imaging abnormalities; (3) Lecanemab ARIA-E rate was 13.1% compared to placebo1.5% (CLARITY Phase 3); Donanemab ARIA-E rate was 24.0% (TRAILBLAZER-ALZ2 Phase 3) compared to placebo <1% (TRAILBLAZER-ALZ Phase 2)

ACI-24.060: Vaccine designed to clear Abeta plaques to treat AD[1]

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

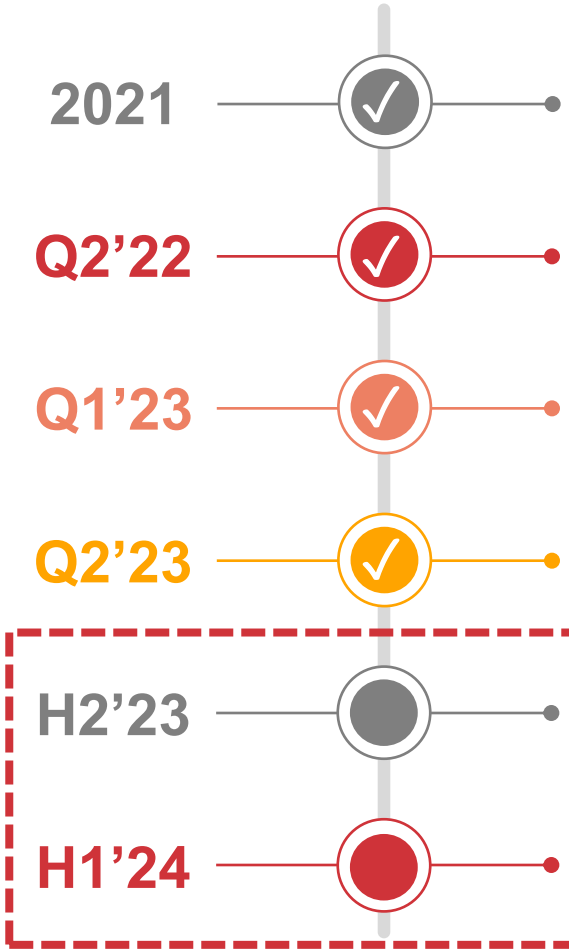
Clinical Stage Programs

TARGET	PRODUCT CANDIDATE	INDICATION	DISCOVERY	PRECLINICAL	PHASE 1	PHASE 2	PHASE 3	PARTNER
Abeta	**ACI-24.060** *(anti-Abeta vaccine)*	AD[1] treatment					reported H1; data H2[3]	
		AD treatment (*Down syndrome[2]*)						
	Crenezumab *(anti-Abeta antibody)*	AD prevention[4]						Genentech
Tau	ACI-35.030 *(anti-pTau vaccine)*	AD treatment						janssen
	Semorinemab *(anti-Tau antibody)*	AD treatment (*mild-to-moderate*)[5]						Genentech
	Morphomer® Tau aggregation inhibitor	Rare Tauopathies						Lilly
		AD treatment						
	Tau-PET[6] tracer	AD diagnostic						Life Molecular Imaging
		PSP[7] diagnostic						Life Molecular Imaging
a-syn[8]	ACI-7104.056 *(anti-a-syn vaccine)*	PD[9], a-synucleinopathies						
	a-syn-PET tracer	a-synucleinopathies (e.g. MSA[10])						

(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-26.060 in patients with AD and patients with Down syndrome; (4) Prevention trial API-ADAD in Colombia; (5) Open label extension study is ongoing; (6) Positron emission tomography; (7) Progressive supranuclear palsy; (8) alpha-synuclein; (9) Parkinson's disease; (10) Multiple system atrophy

ACI-24 program: Achievements and anticipated milestones

Initial Ph 1b/2 data on Abeta plaque reduction measured via PET[1] imaging expected in H1 2024



2021	Biomarker data[2] in previous trials showing evidence of Abeta targeting in AD[3] and AD-DS[4]
Q2'22	Initiation of Phase 1b/2 ABATE study of ACI-24.060 (optimized formulation of ACI-24)
Q1'23	ACI-24.060 shows positive initial interim safety and immunogenicity in ABATE low-dose AD cohort
Q2'23	Expansion of ABATE into participants with Down syndrome (DS) and higher dose AD cohort
H2'23	**Additional interim safety and immunogenicity data in AD and DS from ABATE**
H1'24	**Initial ABATE data on Abeta plaque reduction measured via PET imaging**

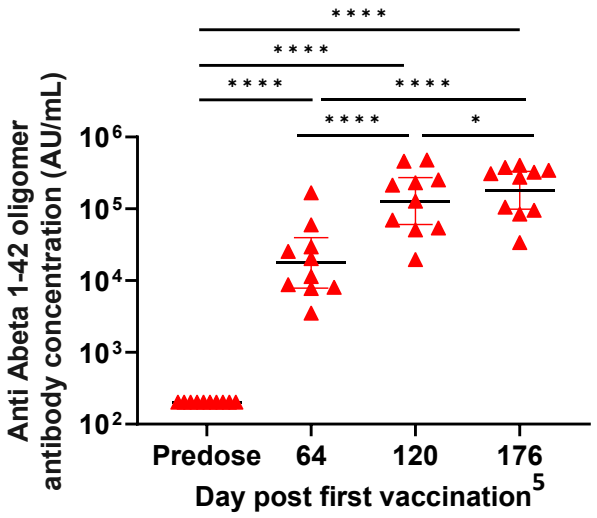
(1) Positron emission tomography; (2) Sol, O. et al., 2021 CTAD poster and Rafii, M. et al., 2022 JAMA Neurology 79:565-574; (3) Alzheimer's disease; (4) Down syndrome

ACI-24.060: Potent immune response against toxic Abeta species

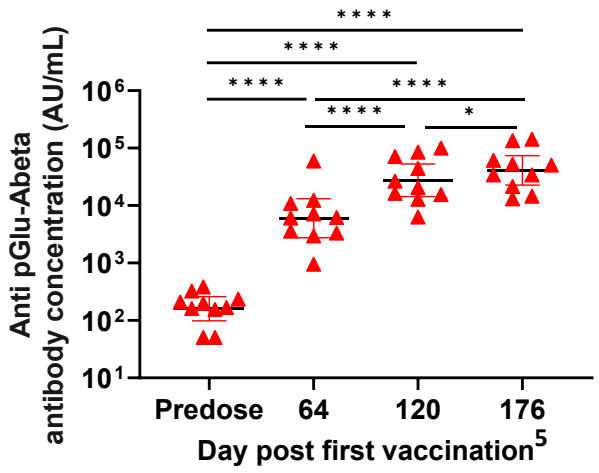
Strong antibody response against targets of lecanemab and donanemab (NHP[1])

ACI-24.060 in NHPs

Anti Abeta1-42 oligomer IgG

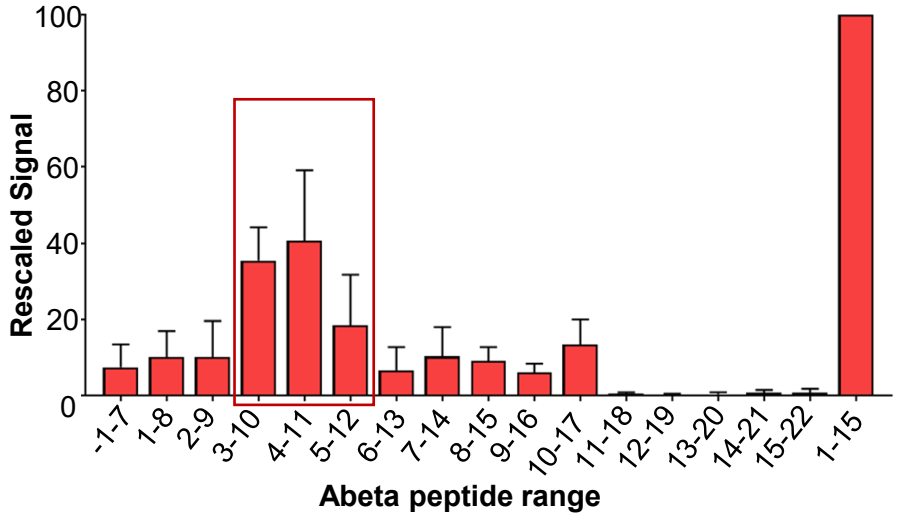


Anti-pyroGlu[2]-Abeta IgG



* 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

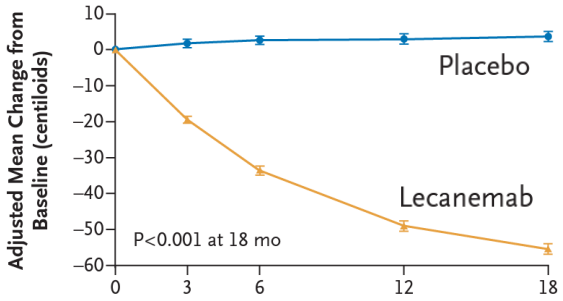
- Sustained, boostable IgG response against Abeta oligomers[3] and pyroglutamate[4] Abeta
- The optimized vaccine represents a potential breakthrough compared to previous anti-Abeta vaccines

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

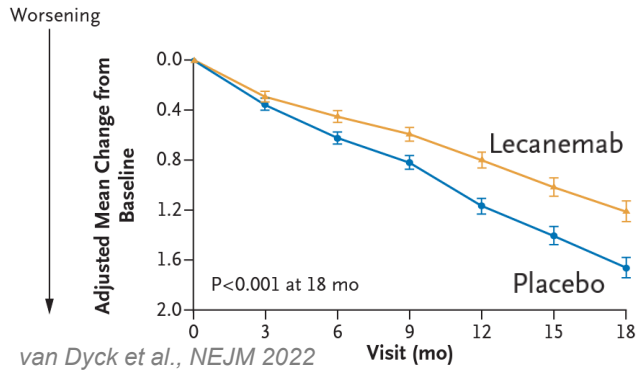
Lowering of Amyloid PET[1] burden is valid as a biomarker for clinical effect

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

Amyloid Burden on PET

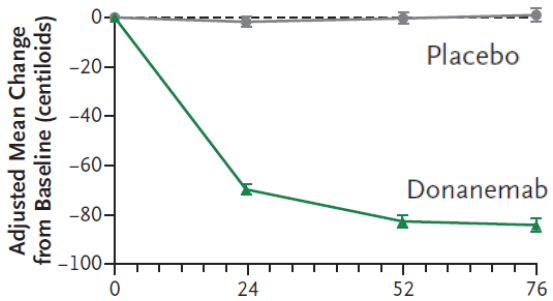


Primary endpoint: CDR-SB[2]

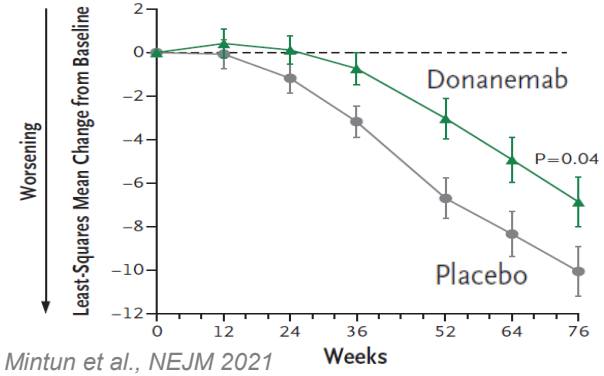


van Dyck et al., NEJM 2022

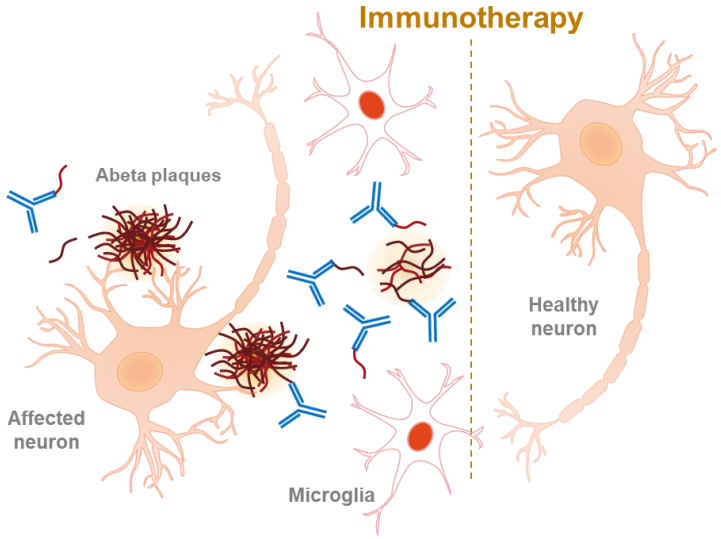
Amyloid Burden on PET



Primary endpoint: iADRS[3]



Mintun et al., NEJM 2021



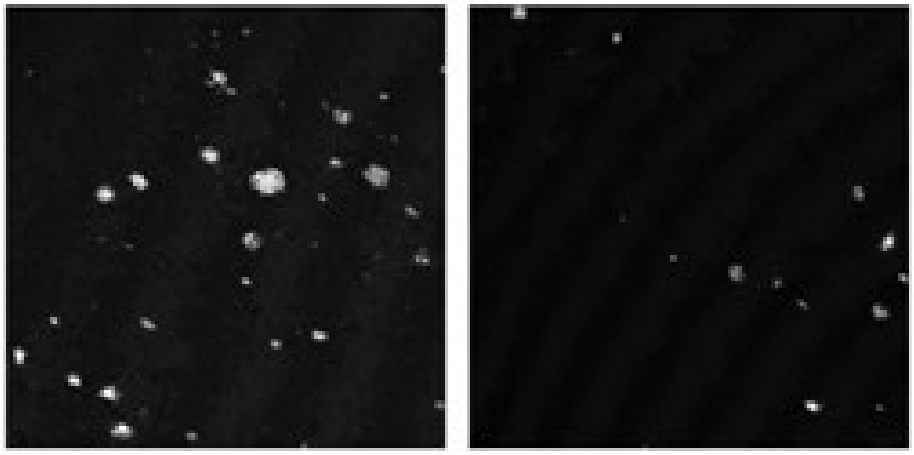
- Targeting small aggregates such as Abeta oligomers or fragments such as Abeta 1-42 and pyroglutamate Abeta3-42 (pGlu-Abeta3-42) has demonstrated clinical utility
- Reductions in Abeta plaques can be detected as early as 3 months after the start of treatment

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

ACI-24 vaccination reduces Abeta plaque burden

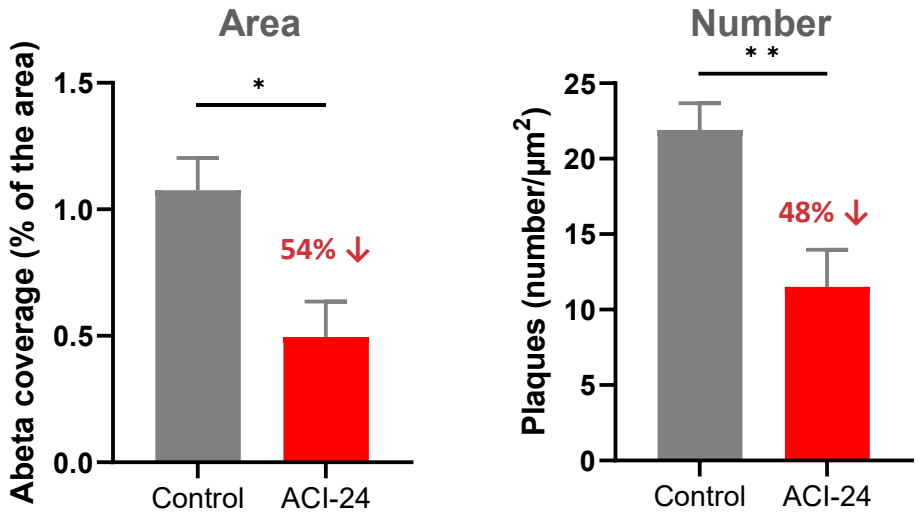
Significant Abeta plaque reduction *in vivo* in preclinical APPxPS1 model[1]

Abeta Plaque Staining in Control and ACI-24 Vaccinated Mice



Control ACI-24

Quantification of Abeta Plaques



* p<0.05 ** p<0.01

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

- ACI-24 vaccination significantly reduces Abeta plaque burden in a *preventive* 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

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

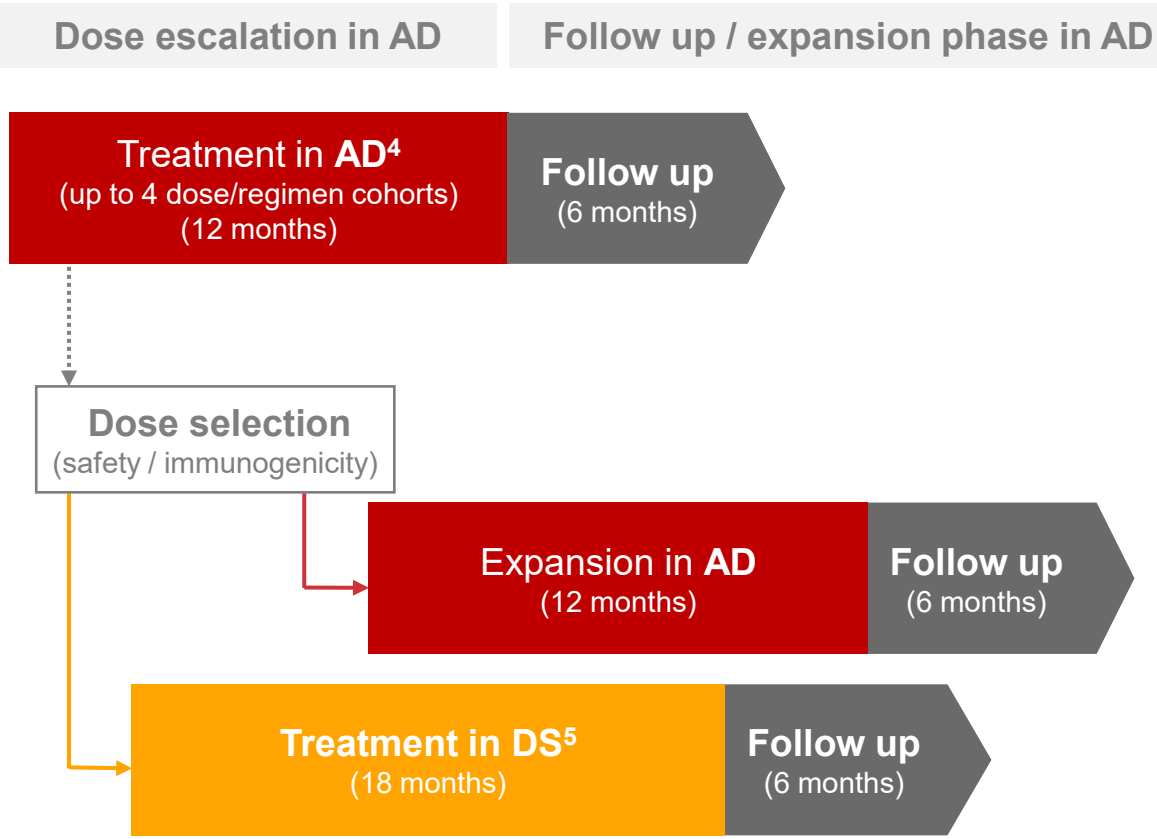
AD
- Up to 4 different doses and/or dose regimens
- Expansion of one cohort to assess effect on Abeta PET

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 – 75 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

ACI-7104: Anti-a-syn[1] vaccine being developed for Parkinson's disease

Update on Phase 2 trial expected in H2

Clinical Stage Programs

TARGET	PRODUCT CANDIDATE	INDICATION	DISCOVERY	PRECLINICAL	PHASE 1	PHASE 2	PHASE 3	PARTNER
Abeta	ACI-24.060 *(anti-Abeta vaccine)*	AD[2] treatment						
		AD treatment (*Down syndrome[3]*)						
	Crenezumab *(anti-Abeta antibody)*	AD prevention[4]						Genentech
Tau	ACI-35.030 *(anti-pTau vaccine)*	AD treatment						Janssen
	Semorinemab *(anti-Tau antibody)*	AD treatment (*mild-to-moderate)[5]*						Genentech
	Morphomer® Tau aggregation inhibitor	Rare Tauopathies						Lilly
		AD treatment						
	Tau-PET[6] tracer	AD diagnostic						Life Molecular Imaging
		PSP[7] diagnostic						Life Molecular Imaging
a-syn	**ACI-7104.056** *(anti-a-syn vaccine)*	PD[8], a-synucleinopathies				update H2		
	a-syn-PET tracer	a-synucleinopathies (e.g. MSA[9])						

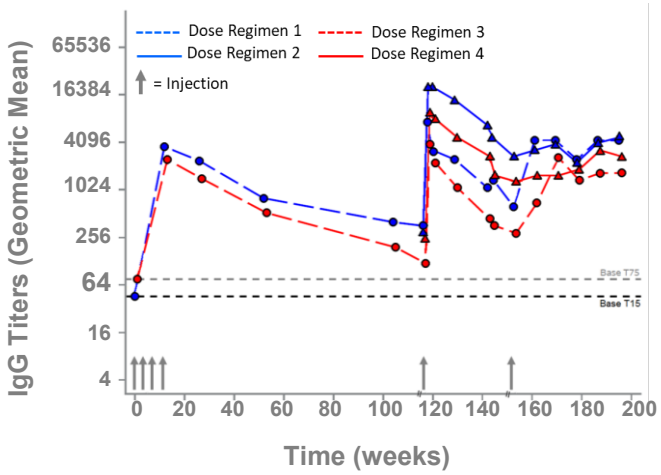
(1) alpha-synuclein; (2) Alzheimer's disease; (3) Down syndrome-related Alzheimer's disease; (4) Prevention trial API-ADAD in Colombia; (5) Open label extension study is ongoing; (6) Positron emission tomography; (7) Progressive supranuclear palsy; (8) Parkinson's disease; (9) Multiple system atrophy

Anti-a-syn[1] vaccine is clinically validated[2] in Parkinson's disease

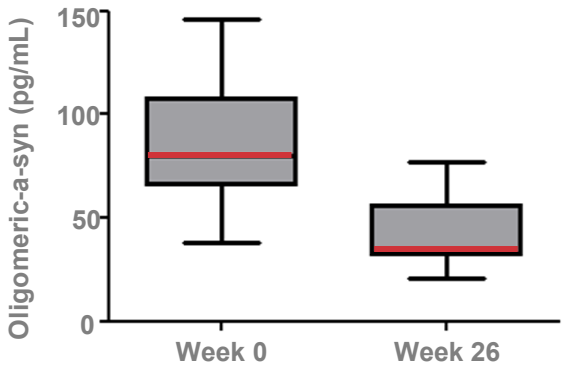
Phase 1 results in *The Lancet Neurology* support best-in-class profile

THE LANCET Neurology

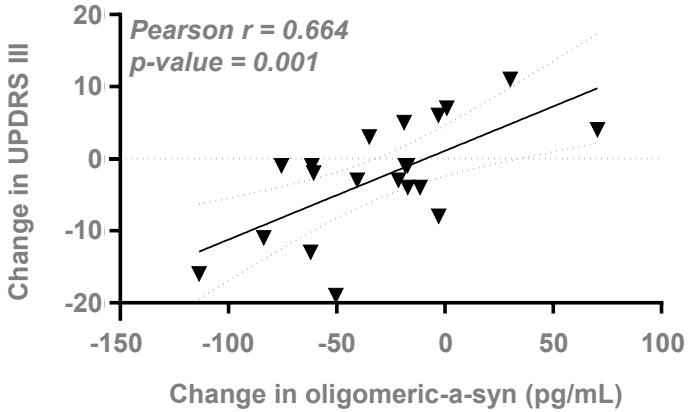
Strong and boostable antibody response



50% reduction[3] of pathological a-syn in CSF[4]



Changes[5] 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[6] III scores correlated with reductions in oligomeric a-syn

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

AC Immune

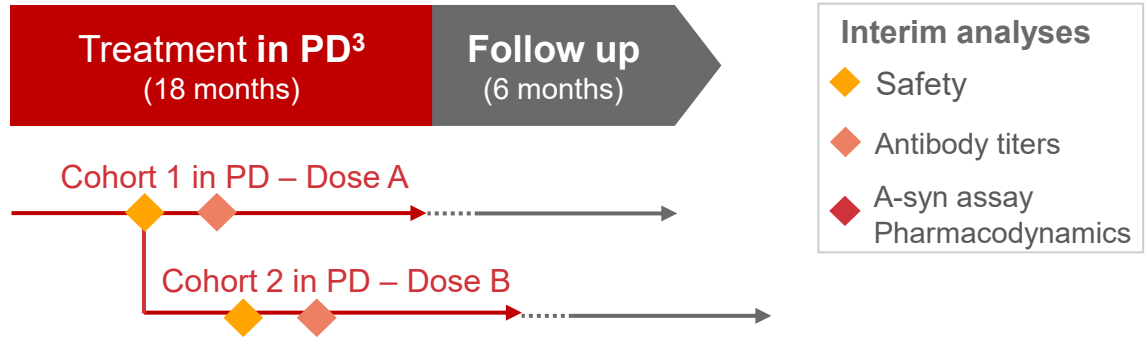
VacSYn: an adaptive biomarker-based Phase 2 study of ACI-7104 in early PD[1]

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

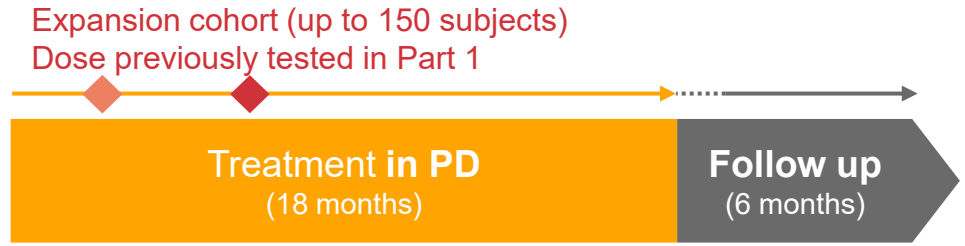
Part 1: Safety & PK/PD[2]

- 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

ACI-35.030: Anti-pTau vaccine being developed for AD[1]

Further clinical development in AD and milestone payment expected in H2

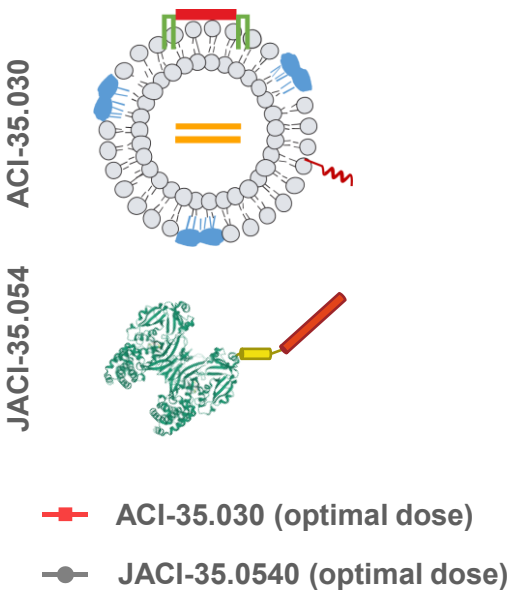
Clinical Stage Programs

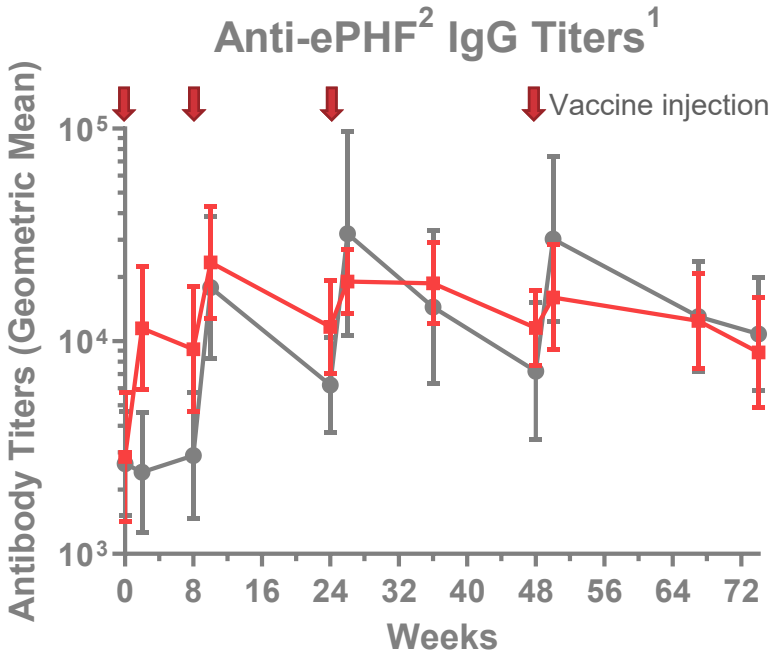
TARGET	PRODUCT CANDIDATE	INDICATION	DISCOVERY	PRECLINICAL	PHASE 1	PHASE 2	PHASE 3	PARTNER
Abeta	ACI-24.060 *(anti-Abeta vaccine)*	AD treatment						
		AD treatment (*Down syndrome*[2])						
	Crenezumab *(anti-Abeta antibody)*	AD prevention[3]						Genentech
Tau	**ACI-35.030** *(anti-pTau vaccine)*	AD treatment						janssen
	Semorinemab *(anti-Tau antibody)*	AD treatment (*mild-to-moderate*)[4]						Genentech
	Morphomer® Tau aggregation inhibitor	Rare Tauopathies						Lilly
		AD treatment						
	Tau-PET[5] tracer	AD diagnostic						Life Molecular Imaging
		PSP[6] diagnostic						Life Molecular Imaging
a-syn[7]	ACI-7104.056 *(anti-a-syn vaccine)*	PD[8], a-synucleinopathies						
	a-syn-PET tracer	a-synucleinopathies (e.g. MSA[9])						

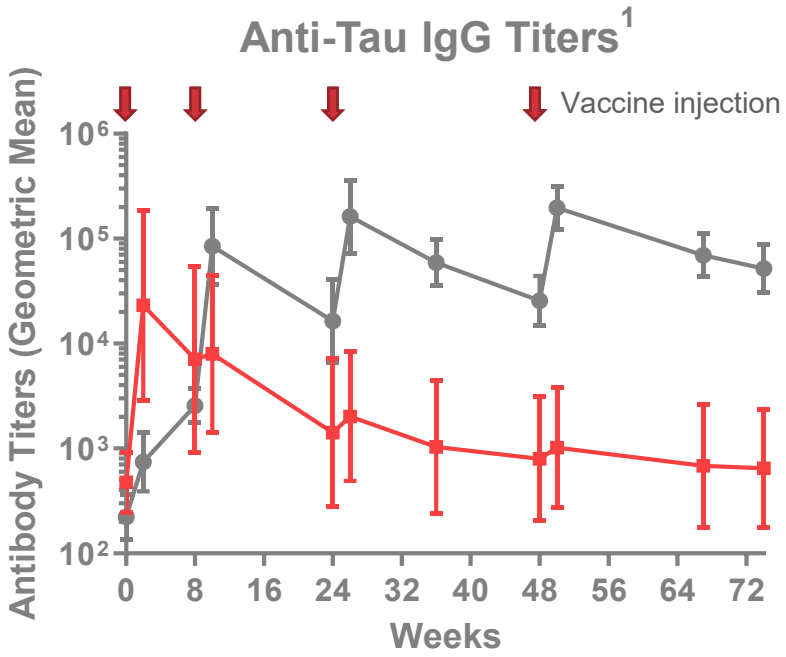
(1) Alzheimer's disease; (2) Down syndrome-related Alzheimer's disease; (3) Prevention trial API-ADAD in Colombia; (4) Open label extension study is ongoing; (5) Positron emission tomography; (6) Progressive supranuclear palsy; (7) alpha-synuclein; (8) Parkinson's disease; (9) Multiple system atrophy

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 vaccine 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

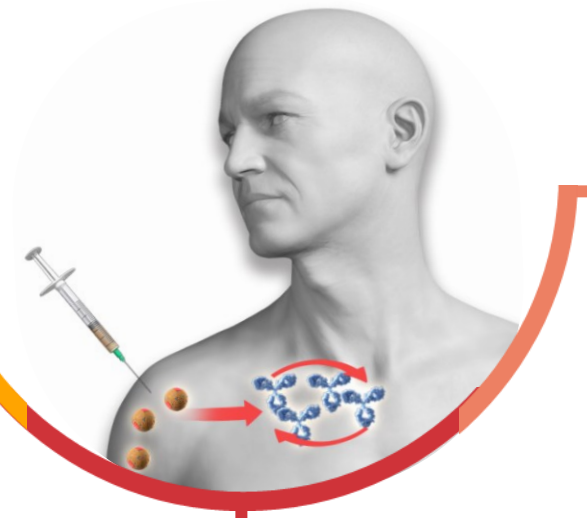
(1) ACI-35.030 original sub-cohort 1.2 data; (2) Enriched paired helical filaments; (3) Phosphorylated Tau; (4) Alzheimer's disease; (5) Antibody

Vaccines as a new class of treatment for neurodegenerative disease

AC Immune vaccines: Potential for profound social and economic impact

Treatment

- High efficacy and safety/tolerability
- Convenient, annual dosing



Prevention

- Vaccination is the best strategy to preserve function and quality of life
- Cost-effective and global application

Maintenance

- Use as maintenance therapy after monoclonal anti-Abeta antibodies
- Convenient, annual dosing

- Goal: Global vaccines for neurodegenerative diseases

Clinical-stage monoclonal antibodies targeting neurodegenerative diseases

Semorinemab: Anti-Tau monoclonal antibody being developed for AD[1]

New Phase 2 open-label extension data expected in H2

Clinical Stage Programs

TARGET	PRODUCT CANDIDATE	INDICATION	DISCOVERY	PRECLINICAL	PHASE 1	PHASE 2	PHASE 3	PARTNER
Abeta	ACI-24.060 *(anti-Abeta vaccine)*	AD[1] treatment						
		AD treatment (*Down syndrome[2]*)						
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Tau	ACI-35.030 *(anti-pTau vaccine)*	AD treatment						janssen
	Semorinemab *(anti-Tau antibody)*	AD treatment (*mild-to-moderate)[4]*					data H2	Genentech
	Morphomer® Tau aggregation inhibitor	Rare Tauopathies						Lilly
		AD treatment						
	Tau-PET[5] tracer	AD diagnostic						Life Molecular Imaging
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Lauriet study evaluating the mAb[1] semorinemab in mild-to-moderate AD[2]

One co-primary endpoint met: first positive cognitive results for an anti-Tau mAb therapy in AD

1. Observed a statistically significant 2.89 point (42.2%) reduction in cognitive decline vs. placebo as measured by ADAS-Cog11[3] at week 49 (p=0.0008)
2. ADAS-Cog11 findings were consistent across prespecified subgroups and at week 61[4]
3. Results showing semorinemab's significant treatment effect on cognition achieved in a population where limited or no effect of anti-Abeta mAbs is observed
4. Semorinemab was well tolerated with an acceptable safety profile and no unanticipated safety signals
5. ADCS-ADL[5] co-primary endpoint and secondary efficacy endpoints (MMSE[6]; CDR-SB[7]) were not met

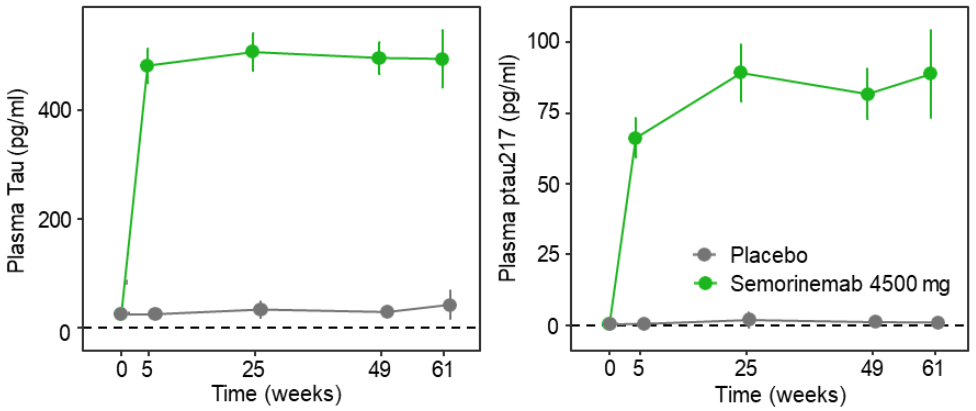
First evidence of therapeutic impact on cognition for a disease-modifying anti-Tau mAb in mild-to-moderate AD patients[8]

(1) Monoclonal antibody; (2) Alzheimer's disease; (3) Alzheimer's Disease Assessment Scale, Cognitive Subscale, 11-item Version; (4) In the subset of patients for whom the double-blind treatment period was extended to 60 weeks; (5) Alzheimer's Disease Cooperative Study - Activities of Daily Living; (6) Mini-mental state exam; (7) Clinical Dementia Rating-Sum of the Boxes; (8) MMSE of 16-21;

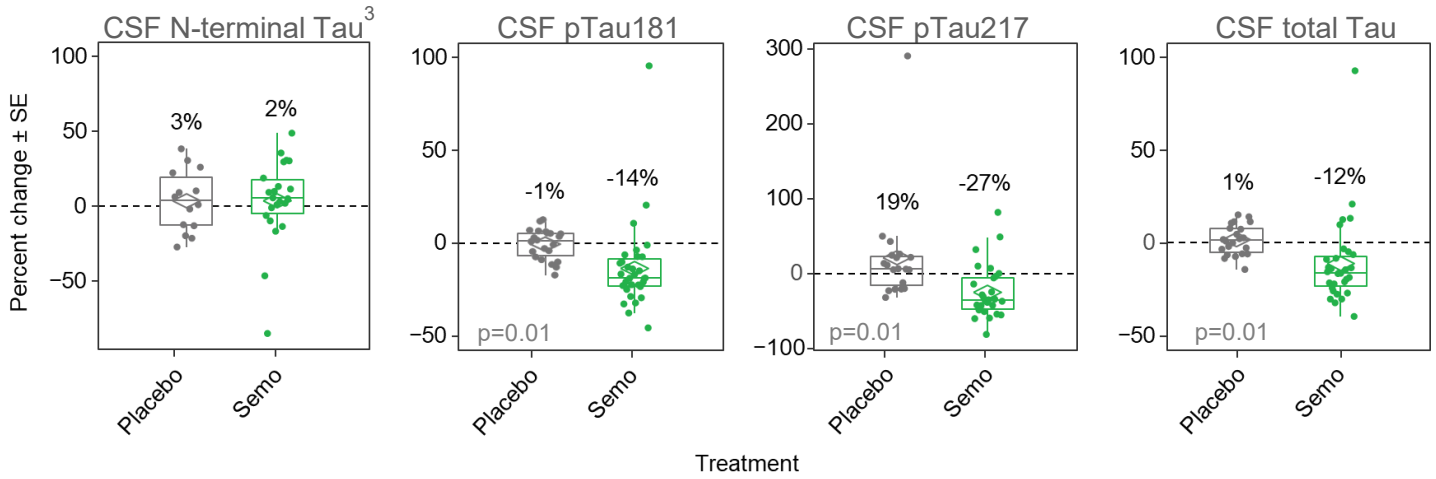
Key biomarker findings from Lauriet Phase 2 trial of semorinemab in AD[1]

Data provide further support for Tau as a target in AD

Plasma pharmacodynamics from Lauriet study[2]



Lauriet study (mild-to-moderate AD): CSF Tau pharmacodynamics



1 Observed rapid rise in plasma Tau levels following treatment with semorinemab, providing evidence of target engagement

2 CSF[4] biomarker analyses confirm target engagement and show significant reduction of pTau[5]

3 Treatment effect on Tau PET[6] signal was not observed

4 Data support the importance of soluble forms of pathological Tau in driving cognitive decline and warrant further analysis

(1) Alzheimer's disease; (2) Week 61 timepoint corresponds to a subset of Cohort 2 patient samples; (3) N-terminal Tau findings potentially linked to antibody-target binding; (4) Cerebrospinal fluid; (5) Phosphorylated Tau; (6) Positron emission tomography

Diagnostics to enable precision medicine

Successfully treating neurodegeneration requires precision medicine

From a mono- to a multi-target combination approach informed by cutting edge diagnostics

Imaging: AC Immune's Unique Capabilities

Positron Emission Tomography

- Tau
- a-syn[1]
- TDP-43[2]

Biofluids

- **Blood / Serum**
- **Cerebrospinal Fluid**

- a-syn
- TDP-43

Future Technologies

In collaboration:

- **Digital Health Technologies & Wearable Devices**

Treating the

right proteinopathies,

in the right patient,

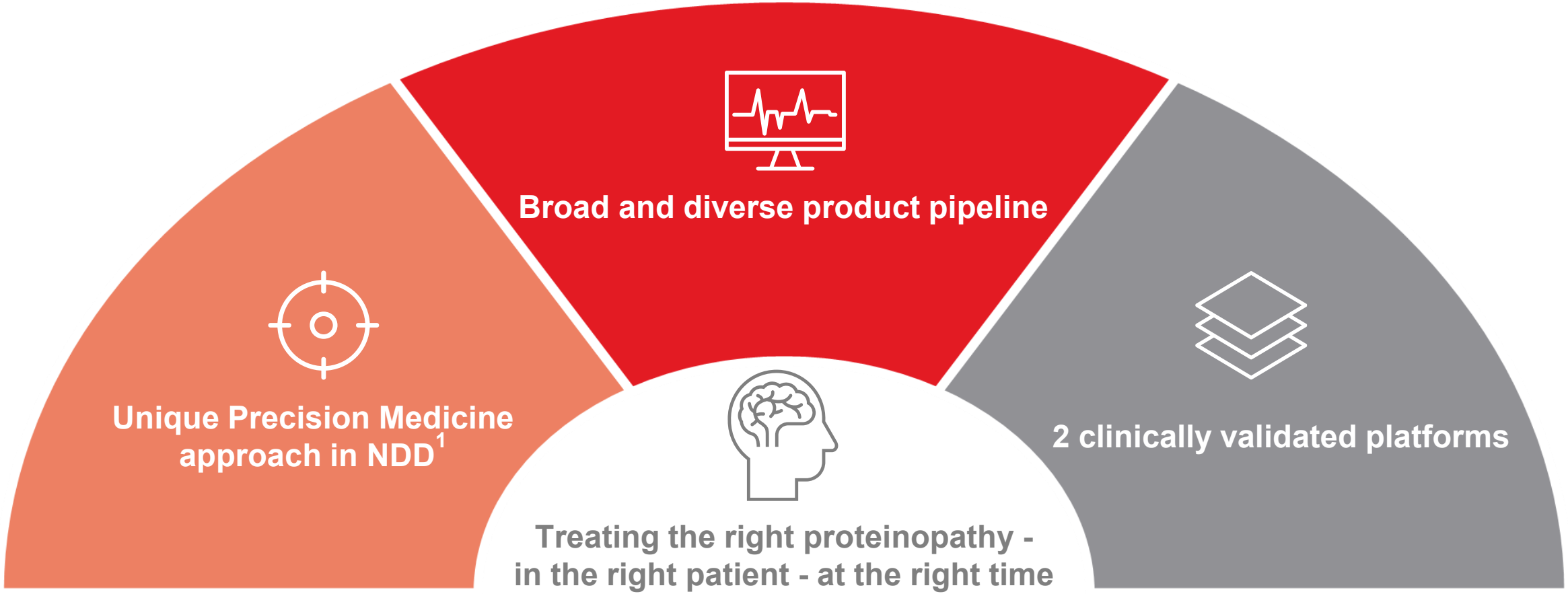
at the right time

- Non-invasive diagnostics are critical for accurate patient selection and treatment to improve clinical outcomes
- Early and comprehensive diagnosis may eventually lead to disease prevention and combination therapy

(1) alpha-synuclein; (2) TAR DNA-binding protein 43;

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

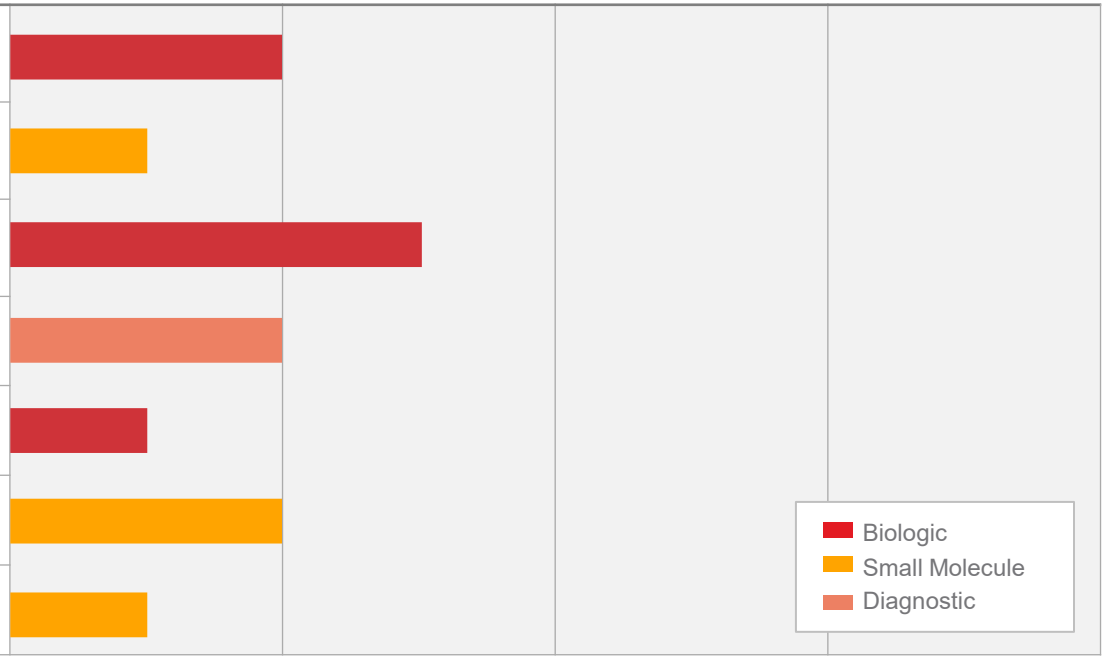
Supplementary information

Broad and robust pipeline in neurodegenerative diseases

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

Novel Targets Pipeline

TARGET	PRODUCT CANDIDATE	INDICATION
a-synuclein (a-syn)	**Anti-a-syn antibody**	PD[3], NeuroOrphan
	Morphomer® a-syn *(a-syn inhibitor)*	PD, a-synucleinopathies
TDP-43	**Anti-TDP-43[4] antibody**	LATE[5], NeuroOrphan
	TDP-43-PET[6] tracer	TDP-43-opathies
Inflammasome	**Anti-NLRP3[7]-ASC[8] antibody**	NeuroOrphan
	Morphomer® NLRP3-ASC	Non-CNS
	Morphomer® NLRP3-ASC	NeuroOrphan; non-CNS



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