Pioneering Precision Medicine for Neurodegeneration

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AC Immune

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www.acimmune.com

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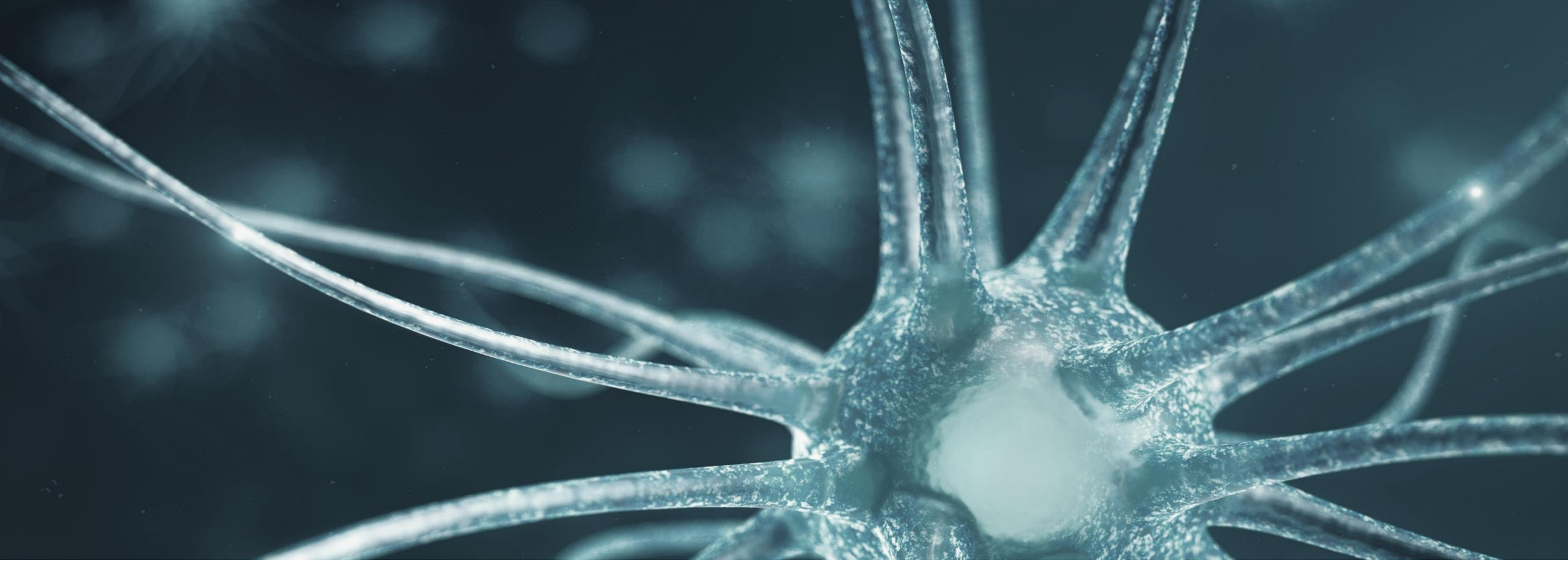
Agenda

1. AC Immune's leadership in neurodegenerative diseases
2. Achievements 2019/20
3. AC Immune's business strategy
4. Pipeline update
5. Novel drug targets for neurodegenerative diseases
6. Near-term inflection points
7. Financial figures
8. Strategic outlook



1. AC Immune’s leadership in neurodegenerative diseases

Andrea Pfeifer, CEO

Roadmap to successful therapies for NDDs[1]

AC Immune

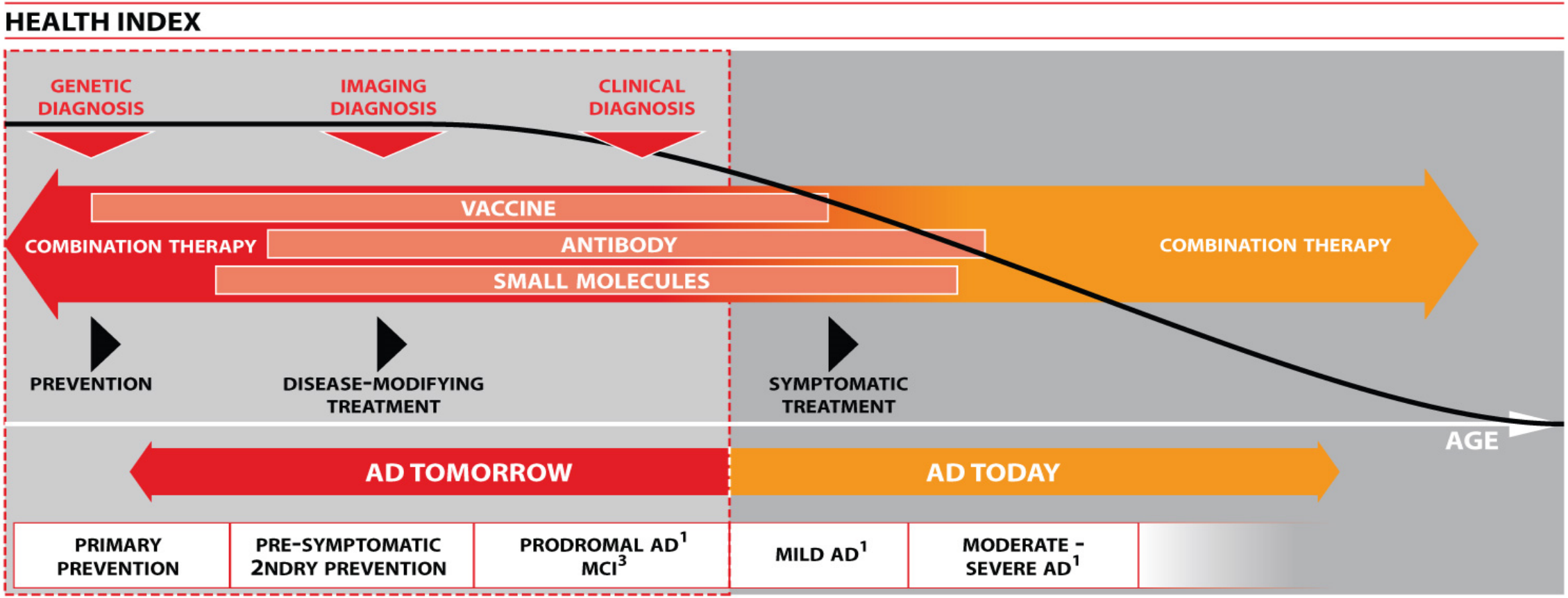
Precision medicine is a key driver for effective treatments

Treat earlier[2]	▪ Prioritize primary and secondary prevention trials ▪ Access diagnostic tools for patient identification
Target Tau[3,4]	▪ Develop therapies and use Tau PET[5] tracers to diagnose/select patients
More homogeneous populations[6,7]	▪ Familial Alzheimer's disease, Down syndrome ▪ NeuroOrphan indications
Precision medicine[8]	▪ Diagnose co-pathologies with selective diagnostics ▪ Select study population based on specific proteinopathies
Target neuroinflammation[9,10]	▪ Develop monoclonal antibodies and small molecules for microglial balance targeting the inflammasome NLRP3[11]-ASC[12] pathway

(1) Neurodegenerative diseases; (2) Reardon S, Nature 2018; (3) Pontecorvo MJ, *et al*., Brain 2019; (4) Gordon BA, *et al*., Brain 2019 ; (5) Positron emission tomography; (6) Strydom A, *et al*., Alzheimers Dement (NY) 2018; (7) Lott IT and Head E.,Nat Rev Neurol. 2019; (8) Robinson JL, *et al*., Brain 2018; (9) Heneka MT *et al*., Nat Rev Neurosci. 2018; (10) Wang S *et al*., Int Immunopharmacol. 2019 ; (11) (NOD)-like receptor protein 3; (12) Apoptosis-associated speck protein containing a CARD

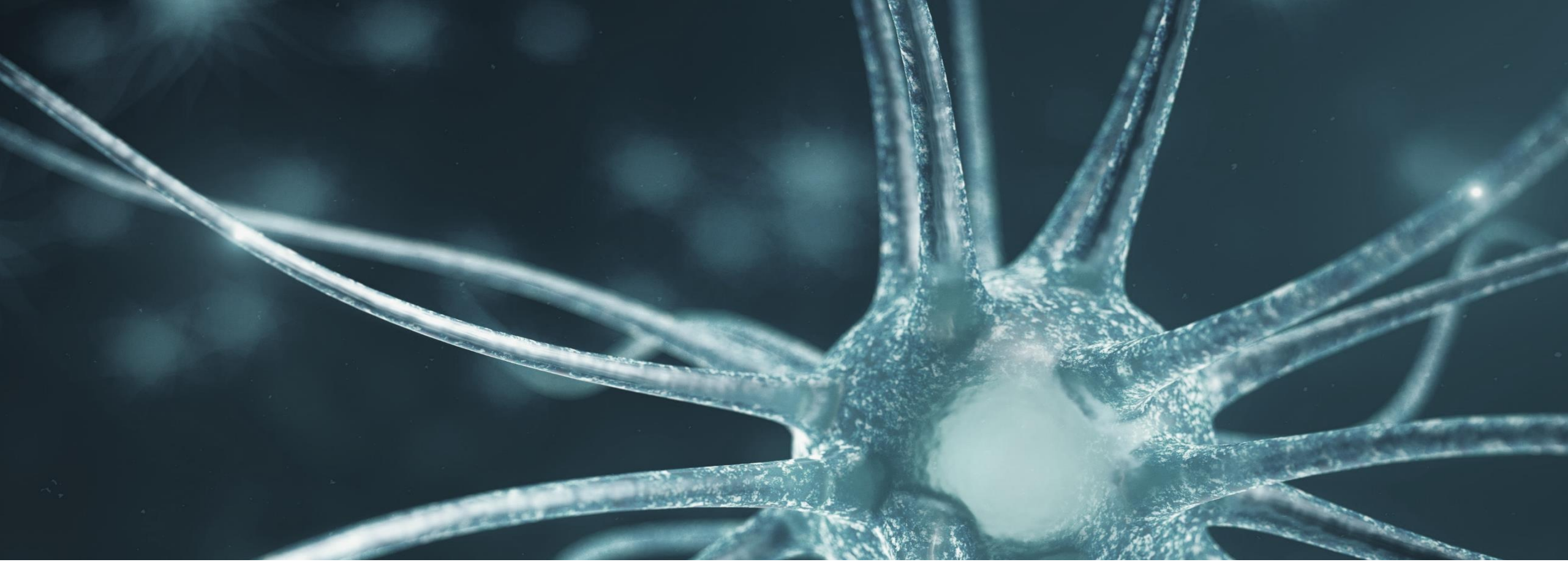
AC Immune is focused on precision medicine in AD[1] and NDD[2]

Multiple targets and approaches enable mono- and combination therapies



(1) Alzheimer's disease; (2) Neurodegenerative diseases; (3) Mild cognitive impairment

- Future treatment paradigms for NDD may involve different combinations of disease modifiers at various stages of a disease
- Combination therapies may include combinations of immunotherapies or combinations of small and large molecules targeting proteinopathies and neuroinflammation



2. Achievements 2019/20

Covid-19: minimal anticipated impact on milestone achievement

Not modifying guidance: remain on track to deliver five clinical readouts in 2020

Executing robust business continuity plan

- Making every provision to protect the health of patients, staff, and investigators
- In continuous coordination with partners and important stakeholders including the Swiss government, trial investigators and contractors
- Maintaining productivity and integrity of our clinical development

On track to deliver 2020 milestones

- Many key trials are already fully enrolled
- Patient follow up continuing virtually

Additional considerations

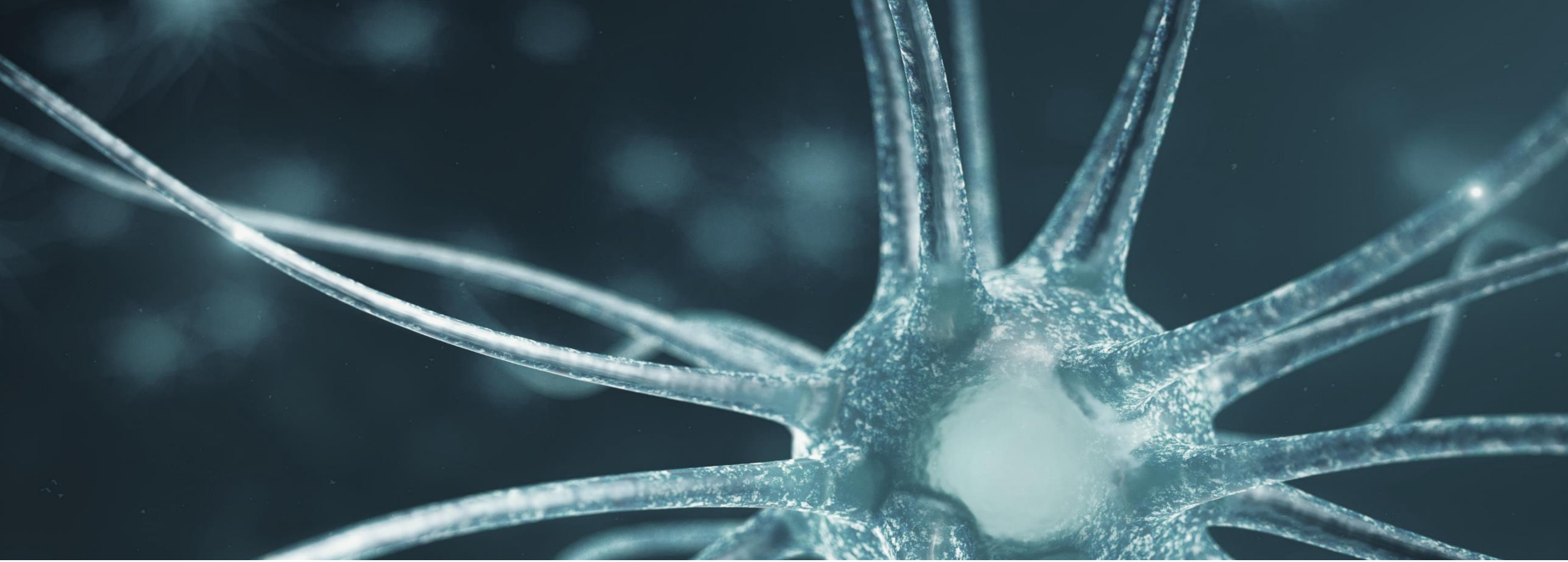
- ACI-24 in AD[1]: Phase 2, 12-month interim data analysis will proceed as planned on a reduced patient data-set
- ACI-24 in DS[2]: Plans to initiate Phase 2 in H2 2020 are progressing; to be initiated in line with public health guidance at that time
- Crenezumab: Phase 2 (API) study: Dosing temporarily interrupted due to country-wide order; patients dosed for >5 years as part of prevention study and data still anticipated in 2022

(1) Alzheimer's disease; (2) Down syndrome

2019: Strong pipeline progress and further improved balance sheet

Building on our track record of successful execution

Achieving partnership milestones	✓ CHF 110 million from Lilly for small molecule Tau inhibitor and $50 million in exchange for a note, convertible to equity at a premium ✓ Milestone payment for Tau PET tracer entering Phase 2 ✓ New grant from MJFF for a-synuclein PET tracer
Advancing/expanding clinical pipeline	✓ Second Phase 2 trial of semorinemab in moderate AD ✓ Phase 1b/2a trial of anti-phospho-Tau vaccine ✓ Phase 1 trial of small molecule Tau inhibitor ✓ Tau imaging substudy in ongoing API study of crenezumab
Delivering program readouts	✓ a-synuclein and TDP-43 Proof of Concept (PoC) studies in Q3 2019 ✓ Interim Phase 1b data for anti-Abeta vaccine in Down syndrome ✓ Interim Phase 1b of anti-pTau vaccine ACI-35 ✓ Phase 1 SAD results for small molecule Tau inhibitor
Establishing thought leadership	✓ Key opinion leader (KOL) event on “untangling Tau pathology” ✓ KOL event on treated AD in people with Down syndrome
Unlocking platform potential	✓ Novel biparatopic antibodies against a-synuclein



3. AC Immune’s business strategy

AC Immune strengths

A leader in neurodegenerative diseases

1. Addressing the largest market opportunity in healthcare
   - Pioneering precision medicine in neurodegenerative diseases

2. Highly productive validated discovery platforms for sustained growth to address misfolded proteins applicable across multiple diseases
   - SupraAntigen™: vaccines and antibodies specific to disease-causing conformations
   - Morphomer™: conformation-sensitive small molecules

3. Broad pipeline with four therapeutic candidates in Phase 2
   - Multiple near-term value inflection points
   - Partnerships with Roche, Janssen Pharmaceuticals and Eli Lilly and Company

4. Complementary diagnostics in clinical development
   - Highly-valued preclinical assets in Tau, a-syn and TDP-43

5. CHF 277.9 million in cash[1]; sufficiently funded to reach multiple value inflection points through at least Q1 2024
   - Increasing investment into key areas of NeuroOrphan and neuroinflammation

(1) As of March 31, 2020

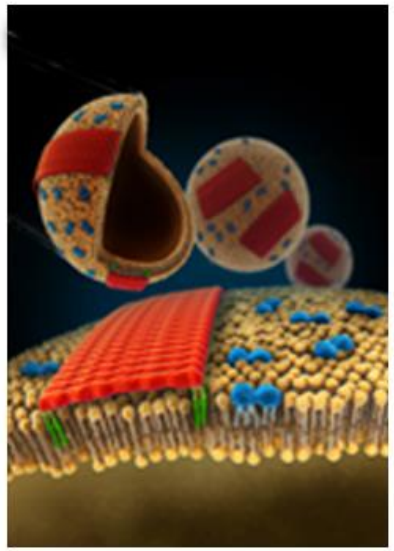
Vision

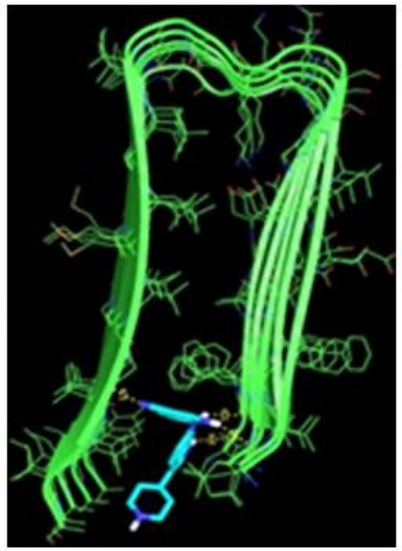
*To become a global leader in **precision medicine**[1] for neurodegenerative diseases leveraging dual proprietary technology platforms to develop breakthrough mono- and combination therapies*

Clinically Validated Technology Platforms

SupraAntigen™

Vaccines and antibodies specific to disease causing conformations





Morphomer™

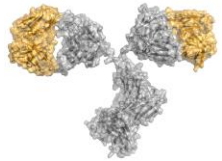
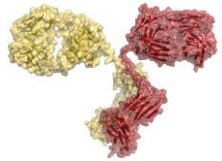
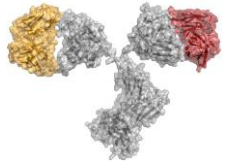
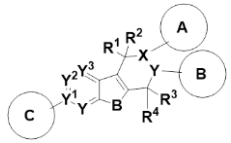
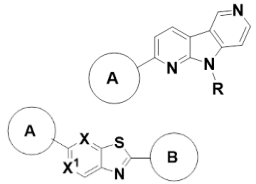
Conformation-sensitive small molecules

Images: Hickman *et al.*, JBC 2011; Kroth *et al.*, JBC 2012

(1) The goal of precision medicine is to deliver optimally targeted and timed interventions tailored to the individual disease drivers

Unlocking platform potential for next generation of innovations

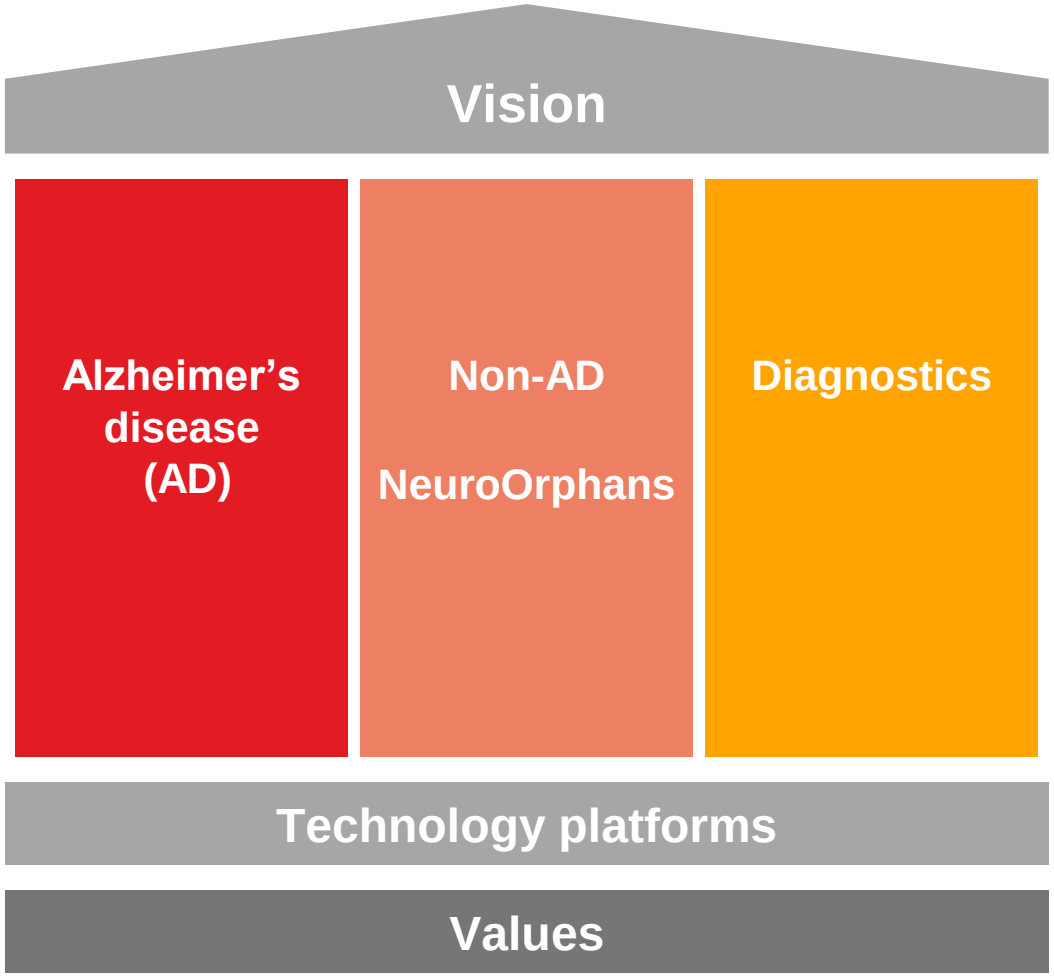
Multiple opportunities for value creation and future partnership

Monoclonal antibodies	Bispecific antibodies	Biparatopic antibodies	Conformational liposomal vaccines	Small molecule therapeutics	PET[1] tracers diagnostics
					
SupraAntigen™				**Morphomer™**	
▪ semorinemab *(anti-Tau)* Genentech A Member of the Roche Group ▪ crenezumab *(anti-Abeta)* Genentech A Member of the Roche Group ▪ anti-a-syn ▪ anti-TDP-43 ▪ anti-ASC[3] (NI[4])	▪ undisclosed	▪ a-syn ▪ undisclosed	▪ ACI-35 *(anti-pTau vaccine)* janssen ▪ ACI-24 *(anti-Abeta vaccine)*	▪ ACI-3024 *(Tau inhibitor)* Lilly ▪ a-syn inhibitor ▪ NLRP3[2] inhibitor	▪ PI-2620 *(Tau tracer)* Life Molecular Imaging ▪ a-syn tracer ▪ TDP-43 tracer

(1) Positron emission tomography; (2) (NOD)-like receptor protein 3; (3) Apoptosis-associated speck protein containing a CARD; (4) Neuroinflammation

Business strategy: three-pillar approach

Precision medicine ultimately creates differentiation



Alzheimer's disease (AD)

- Develop best-in-class late stage assets in partnership
- Develop preventive/therapeutic vaccines as fully owned assets (ACI-24)
- Establish a pipeline of disease-modifying small molecules

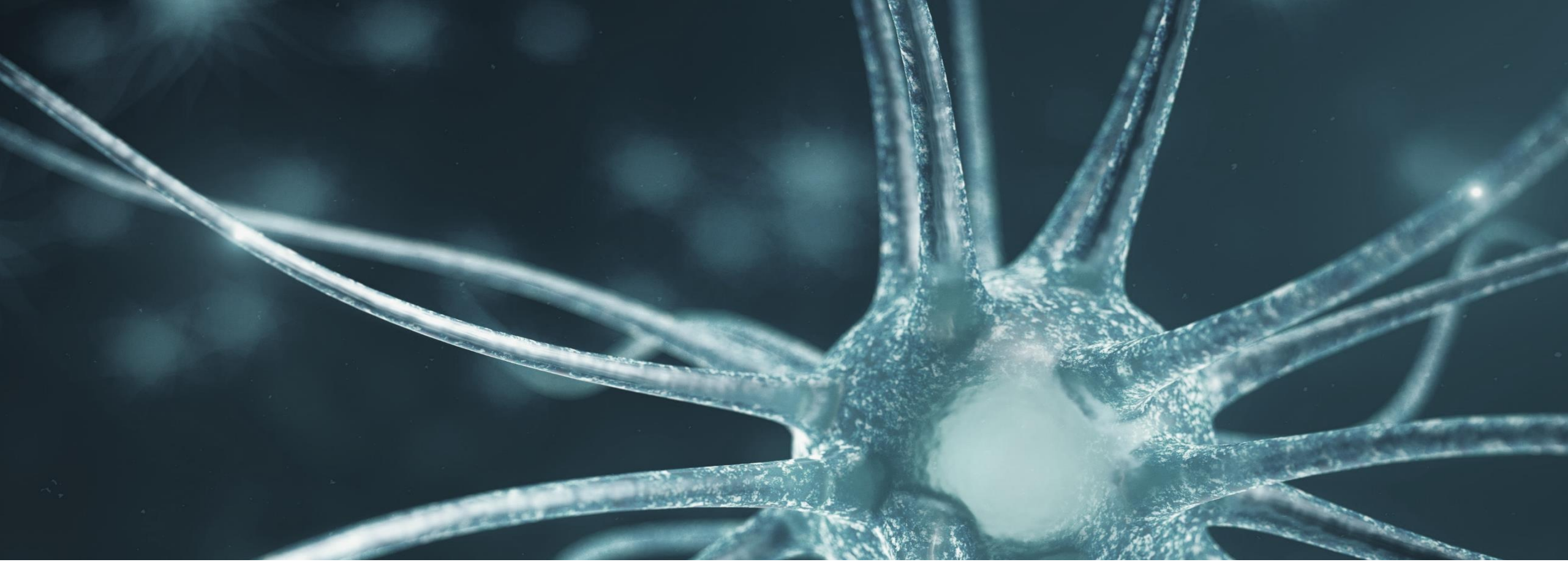
Non-AD, NeuroOrphans

- Discover therapeutics in Parkinson's disease
- Leverage AD therapeutics in Down syndrome, PSP[1] and other NeuroOrphan diseases
- Target neuroinflammation for NDD[2] as mono- and/or combination therapy

Diagnostics

- Accelerate diagnostic pipeline to late stage development
- Use diagnostics for improved clinical trials and external partnerships

(1) Progressive supranuclear palsy; (2) Neurodegenerative diseases



4. Pipeline update

Broad and robust pipeline in neurodegenerative diseases

Driven by validated proprietary technology platforms for sustained growth

Clinical-stage pipeline (★ data readout expected in 2020)

TARGET	PRODUCT CANDIDATE	INDICATION	DISCOVERY	PRECLINICAL	PHASE 1	PHASE 2	PHASE 3	PARTNER
Tau	**semorinemab** *(anti-Tau antibody)*	AD[1] treatment *(prodromal / mild)*					★	Genentech A Member of the Roche Group
		AD treatment *(moderate)*						
	ACI-35.030 *(anti-pTau vaccine)*	AD treatment					★	Janssen
	ACI-3024 *(Tau inhibitor)*	AD treatment				★		Lilly
	Tau-PET[2] tracer	AD and PSP[3]						Life Molecular Imaging
Abeta	**crenezumab** *(anti-Abeta antibody)*	AD prevention[4]						Genentech A Member of the Roche Group
	ACI-24 *(anti-Abeta vaccine)*	AD treatment (*Down syndrome[5]*)				★		
		AD treatment					★	

Biologic
Small Molecule
Diagnostic

(1) Alzheimer's disease; (2) Positron emission tomography; (3) Progressive supranuclear palsy; (4) Prevention trial API-ADAD in Colombia; (5) AD-like cognitive impairment associated with Down syndrome

Broad and robust pipeline in neurodegenerative diseases

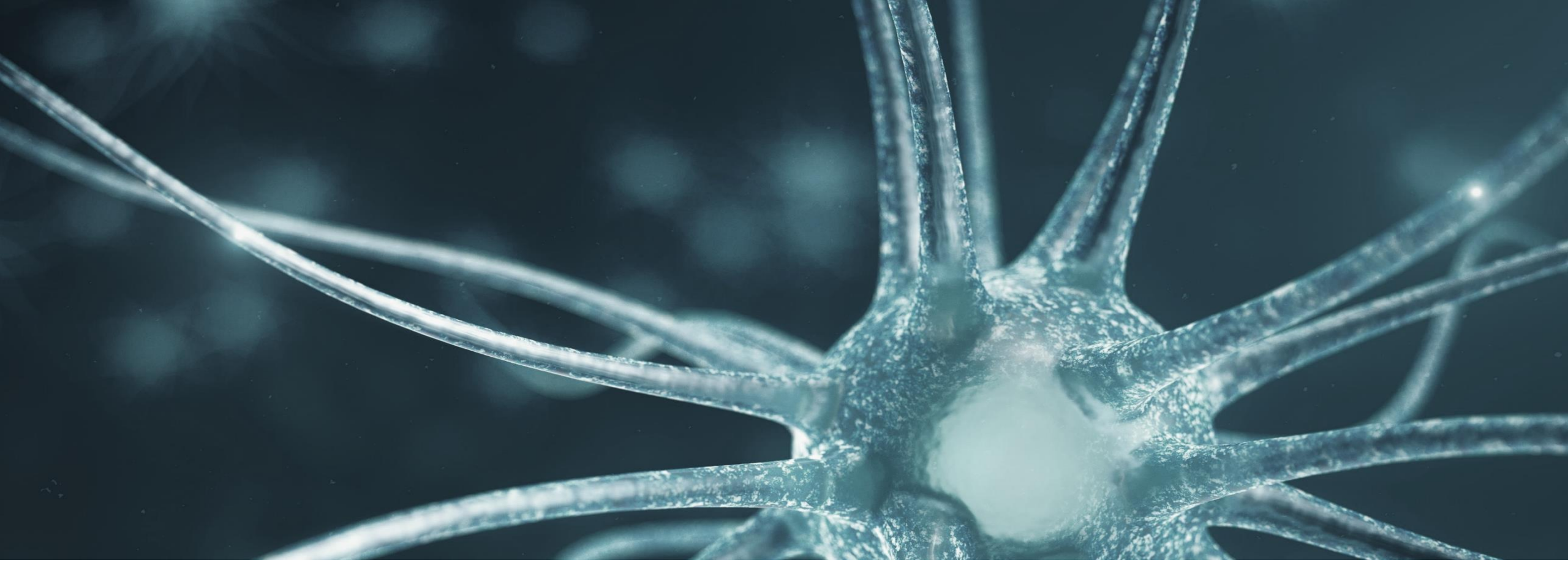
Driven by validated proprietary technology platforms for sustained growth

Early-stage pipeline (★ key milestone in 2020)

TARGET	PRODUCT CANDIDATE	INDICATION	DISCOVERY	PRECLINICAL	PHASE 1
a-synuclein (a-syn)	**a-syn-PET[1] tracer**	PD[2], a-synucleinopathies			★
	anti-a-syn antibody	PD, NeuroOrphan		★	
	Morphomer a-syn *(a-syn inhibitor)*	PD, a-synucleinopathies		★	
TDP-43[3]	**anti-TDP-43 antibody**	NeuroOrphan		★	
	TDP-43-PET tracer	TDP-43-opathies			
Inflammasome	**anti-NLRP3[4]-ASC[5] antibody**	NeuroOrphan		★	
	Morphomer-NLRP3-ASC	Non-CNS[6]		★	
	Morphomer-NLRP3-ASC	NeuroOrphan			

Biologic
Small Molecule
Diagnostic

(1) Positron emission tomography; (2) Parkinson's disease (3) TAR DNA-binding protein 43; (4) (NOD)-like receptor protein 3; (5) Apoptosis-associated speck protein containing a CARD; (6) Central nervous system



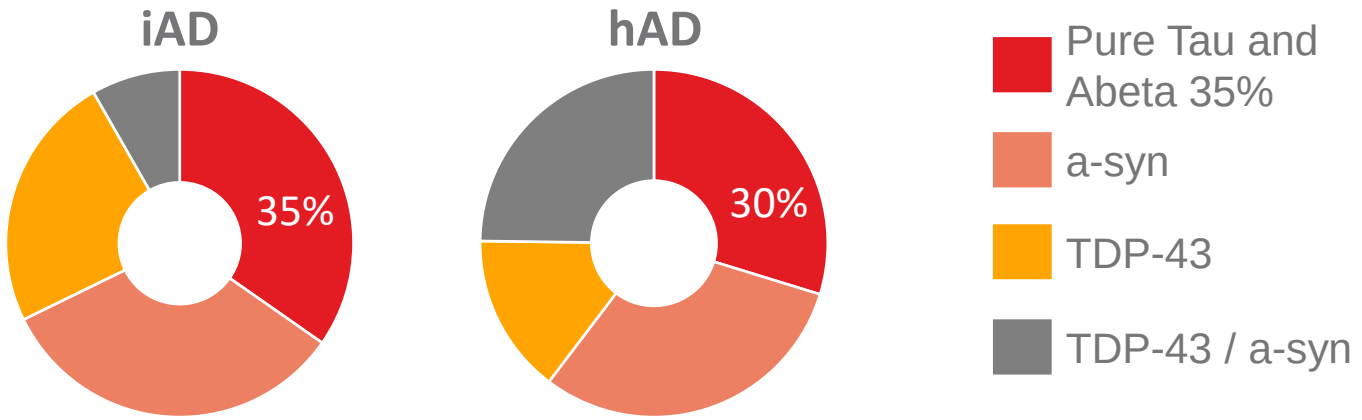
5. Novel drug targets for neurodegenerative diseases

Why do we need precision medicine in AD[1]?

High level of other proteinopathies and co-pathologies in AD

hAD[2] (iAD[3]) shows high levels of co-pathologies:

- 55% (41%) a-syn;
- 40% (33%) TDP-43 with an overall prevalence of 70% (65%) of co-pathologies



Adapted from Robinson *et al*, Brain, 2018

iAD[3]; intermediate; hAD[2], high levels of co-pathologies

- The prevalence of co-pathologies in AD[1] and other NDD[4] may indicate a need for different therapies at different stages
- Clinical trial participants may be better defined by their various proteinopathies
- Patient sub-classification may lead to improved clinical outcome
- Combination therapy may be the ultimate requirement

(1) Alzheimer's disease; (2) High level of Alzheimer's disease neuropathological change; (3) Intermediate level of Alzheimer's disease neuropathological change; (4) Neurodegenerative diseases

TDP-43[1] and alpha-synuclein: drivers of value creation in 2020 and beyond

Broad applications in NDD and AD

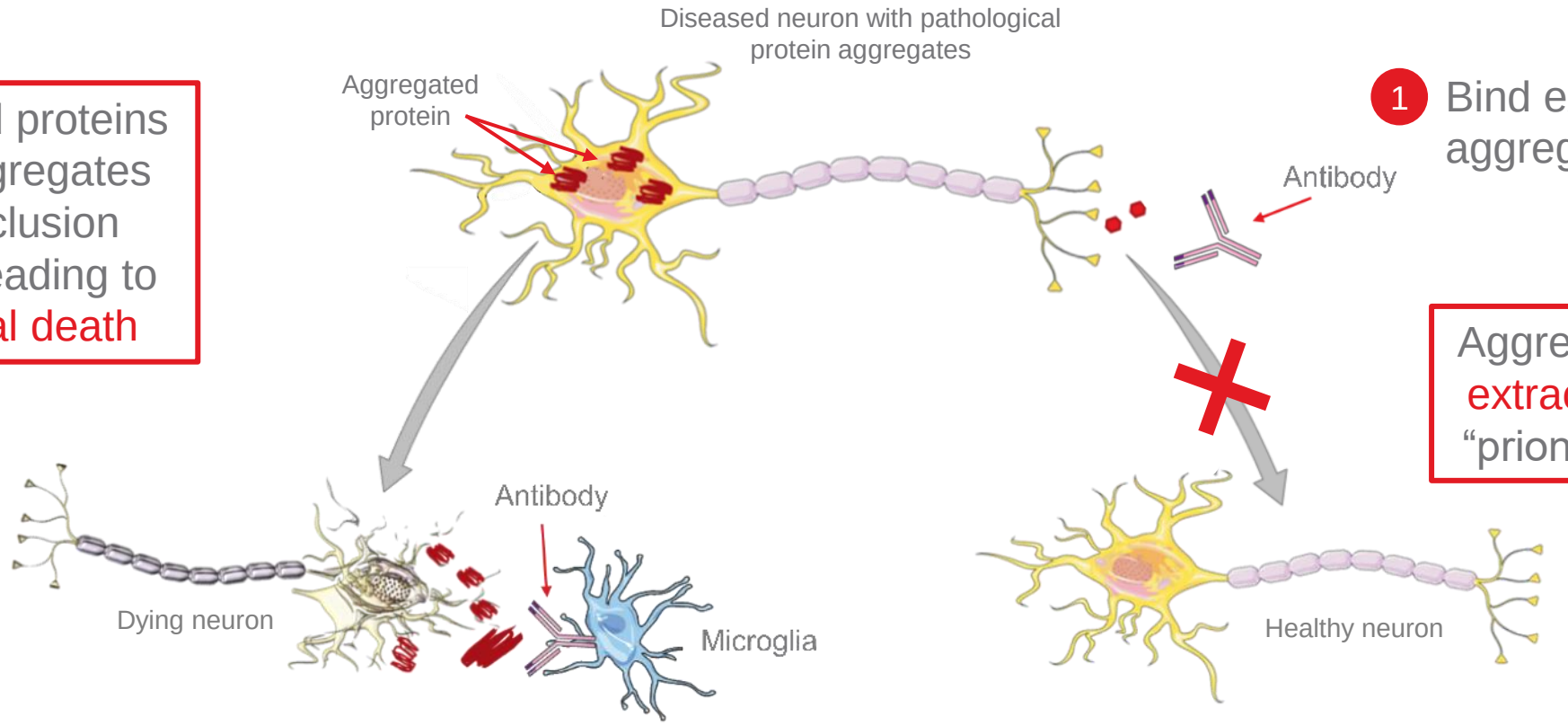
Established hallmarks in NDD	Including NeuroOrphan indications and Parkinson's disease
Novel therapeutic targets in Alzheimer's disease	High levels of a-syn and/or TDP-43 co-pathology
Highlights the need for precision medicine	For faster and more accurate diagnosis, treatment and monitoring of disease progression
Significant market opportunity	AC Immune's therapeutic and diagnostic programs are amongst the most comprehensive in the field

(1) TAR DNA-binding protein 43

Emerging targets in neurodegenerative disease

Antibodies targeting TDP-43[1] and a-syn[2]

Misfolded proteins form aggregates and inclusion bodies leading to neuronal death



1 Bind extracellular aggregates

Aggregates spread extracellularly in a "prion-like" fashion

2 Enhance phagocytic clearance

3 Inhibit seeding and spreading

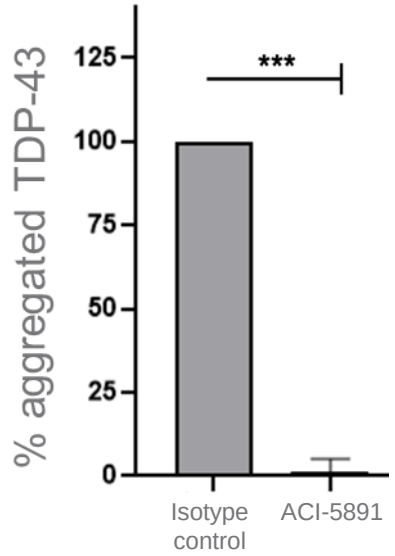
(1) TAR DNA-binding protein 43; (2) Alpha-synuclein; (3) Neurodegenerative diseases; (4) Alzheimer's disease

Only anti-TDP-43 antibody reported with demonstrated *in vivo* activity

Established preclinical proof-of-concept

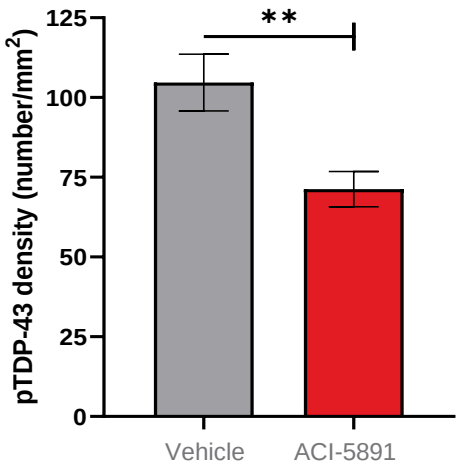
Inhibition of TDP-43 aggregation *in vitro*

Recombinant TDP-43 aggregation assay

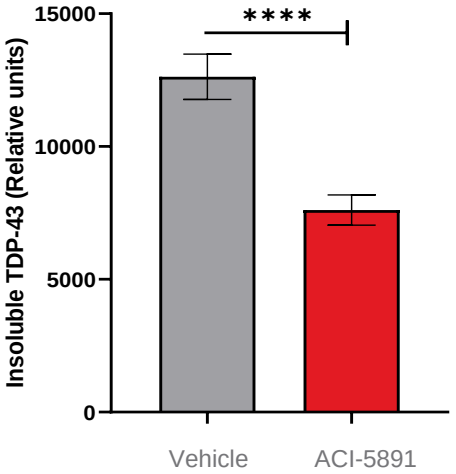


Reduction of pathological TDP-43 *in vivo*[1]

pTDP-43[2] (Immunohistochemistry)



Insoluble TDP-43 (biochemistry)



Ref: AC Immune unpublished data

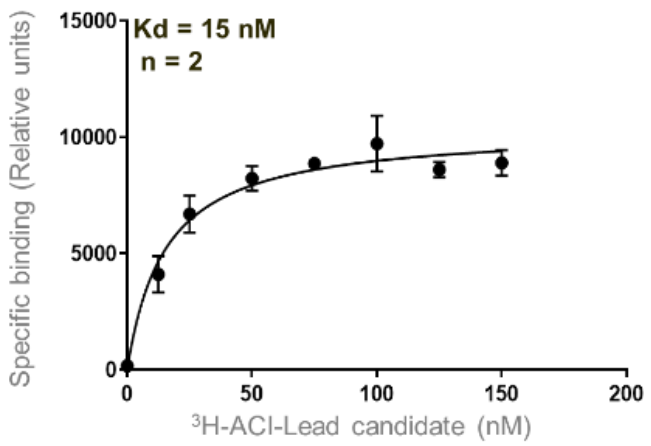
Key results	▪ *In vitro*, 98% inhibition of TDP-43 aggregation ▪ *In vivo*, significant reduction in TDP-43 neuro-pathology
Next steps	▪ Complete humanization of lead candidate; start IND[3]-enabling studies in **Q2 2020**

(1) rNLS8 TDP-43 transgenic mouse model; Walker *et al.,* Acta Neuropathol., 2015; (2) Phosphorylated TDP-43; (3) Investigational new drug

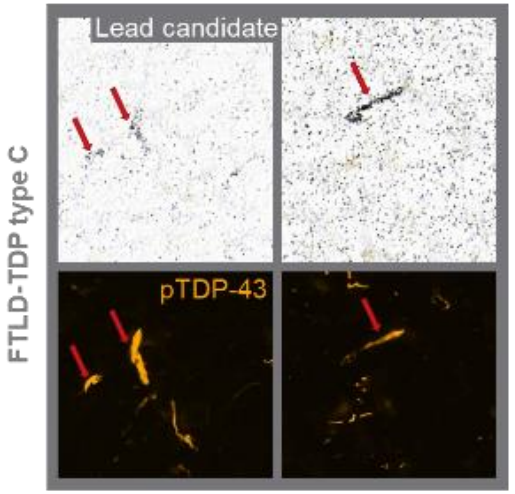
First-in-class TDP-43 PET[1] imaging tracer – Discovery Phase

Designed to facilitate clinical development and enable precision medicine

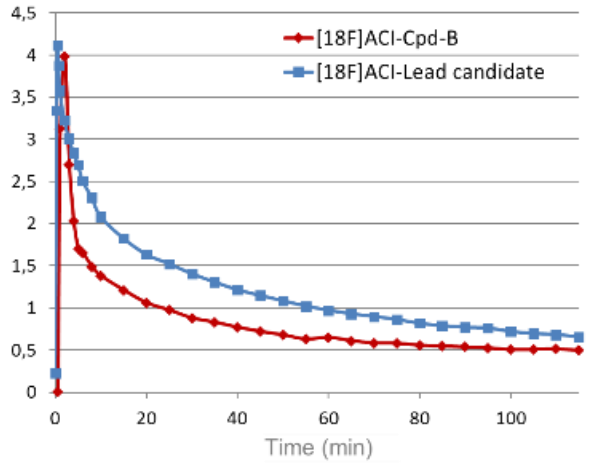
Binding affinity on FTD type-C brain-derived TDP-43 aggregates



Target engagement by micro-autoradiography



Brain PK[2] profile



Ref: AC Immune unpublished data

Key results	▪ Lead candidate shows selective TDP-43 binding ▪ Target engagement confirmed by micro-autoradiography ▪ PK study confirmed good, rapid brain uptake (4.11%)
Next steps	▪ Further optimize target affinity and PK profile; declare clinical lead candidate

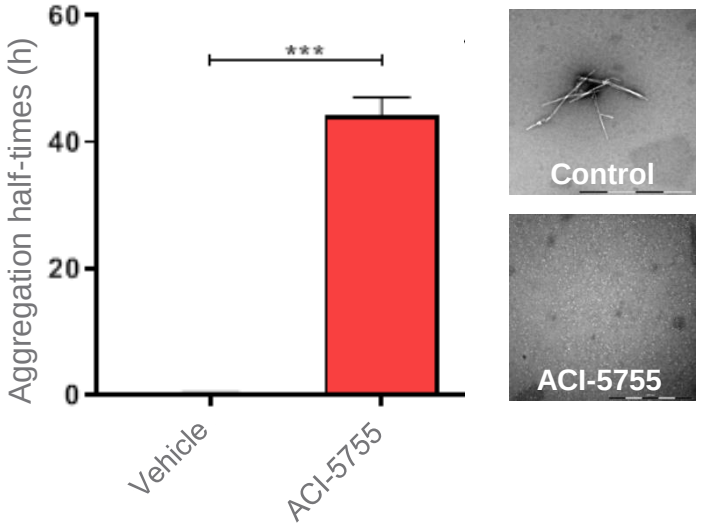
(1) Positron emission tomography; (2) Pharmacokinetic

Lead candidate ACI-5755 currently in IND[1]-enabling studies in PD[2]

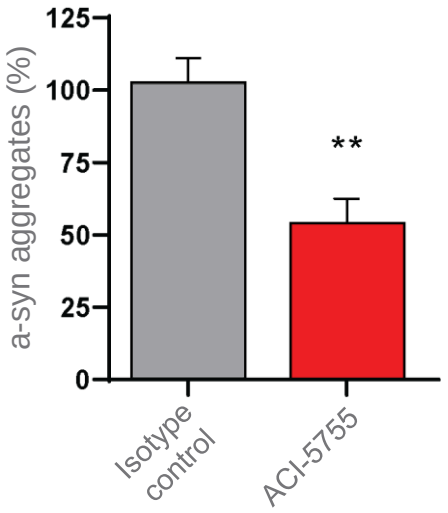
Targeting spreading of pathological a-syn with selective antibody

Inhibition of a-syn aggregation *in vitro*

Recombinant a-syn aggregation assay

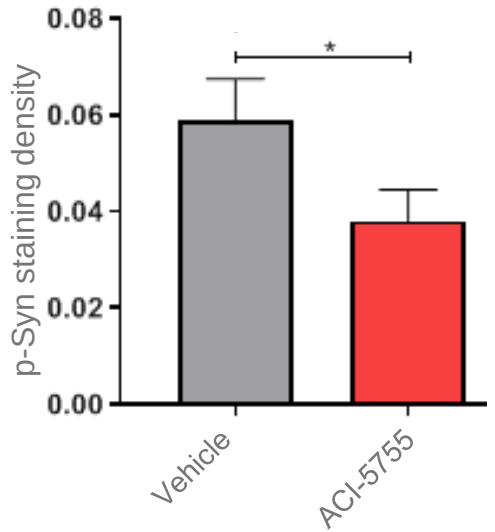


Cellular spreading assay[4]



Reduction of pathological a-syn *in vivo*[3]

Phosphorylated S129 a-syn (p-Syn[5]) cortex immunohistochemistry



Ref: AC Immune data presented at AD/PD 2020

Key results	▪ *In vitro*, ACI-5755 significantly inhibits a-syn aggregation and inhibits uptake and seeding capacity of a-syn aggregates in cells ▪ *In vivo*, ACI-5755 significantly decreases a-syn spreading
Next steps	▪ Advance towards IND filing

(1) Investigational new drug; (2) Parkinson's disease; (3) M83 transgenic mice inoculated with human a-syn preformed fibrils; (4) Human cell line susceptible to a-syn seeding; (5) p-syn antibody (pSer129; Abcam, UK)

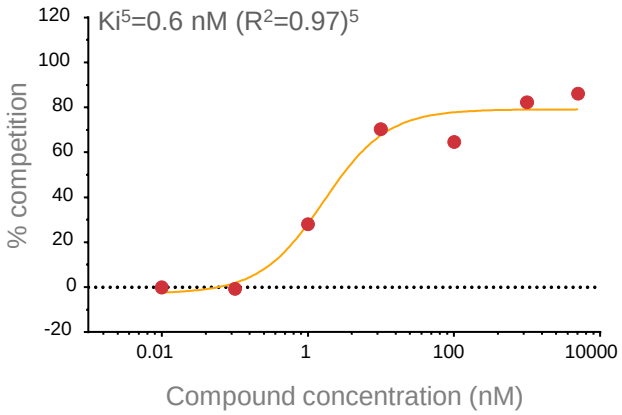
A-syn PET[1] imaging tracer – First-in-Human

Potentially the first selective diagnostic agent for PD[2]

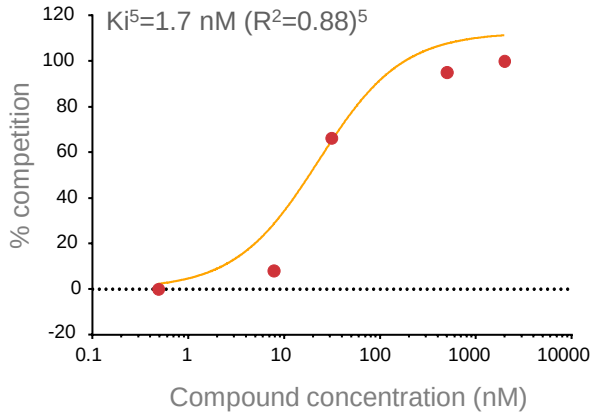
THE MICHAEL J. FOX FOUNDATION FOR PARKINSON'S RESEARCH

Biochemical and histological radiography assays

Binding to Lewy bodies in PD brains

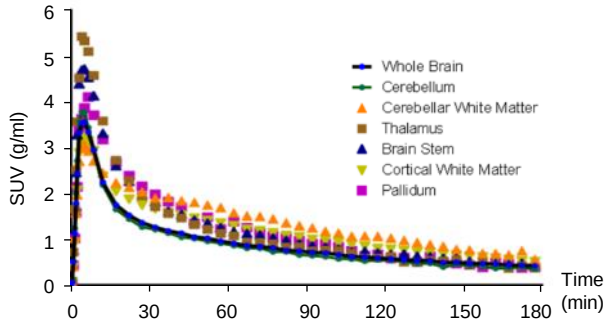


Binding to PD-derived a-syn aggregates



Pharmacokinetic (PK) profile in NHP[3]

18F-PK profile in different brain regions[4]



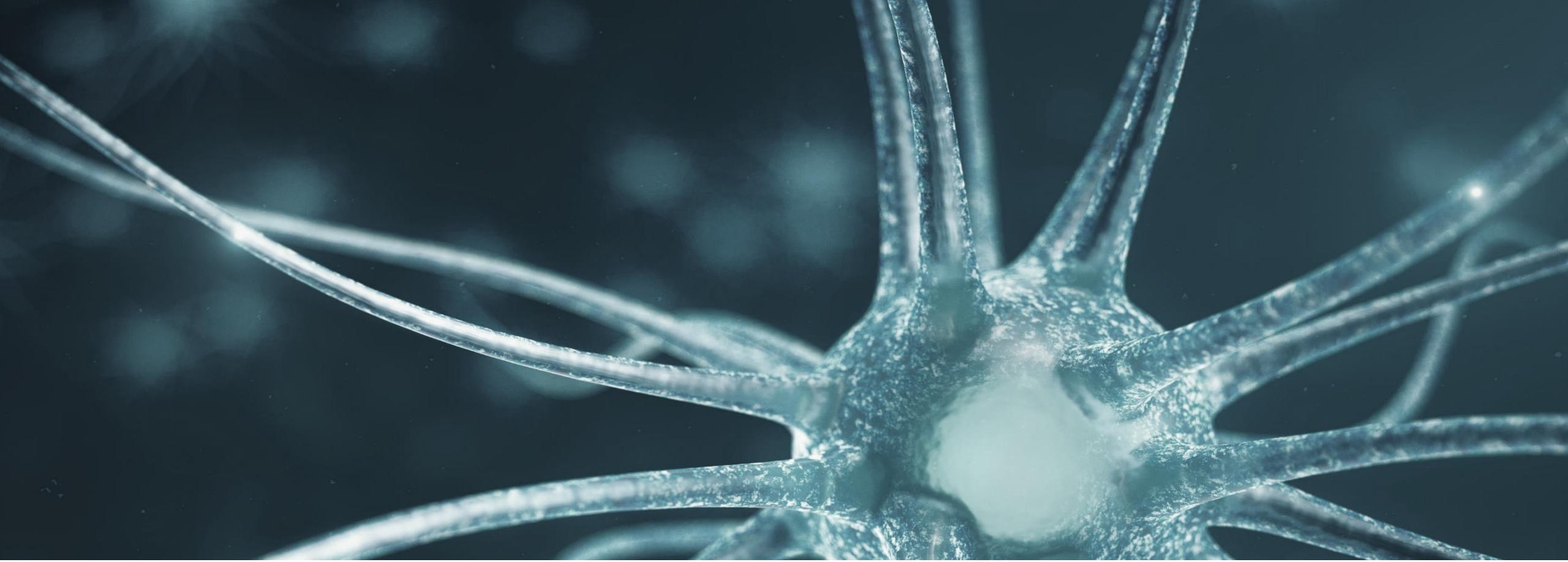
Capotosti, AD/PD Conference, Lisbon 2019

Brain uptake		Wash-out
Time to Cmax	ID/g[6] brain (%)	Peak/half Peak
5min	4.0	~ 20min

Key results	▪ Highly specific, low nanomolar binding in human PD, DLB[7] and MSA[8] brains ▪ Between 500 to 1000-fold selectivity over potential Abeta co-pathologies ▪ Favorable PK profile in NHPs and mice
Next steps	▪ 2nd-gen study in genetic populations, i.e. MSA and SNCA[9] ▪ Advance 3rd-gen candidate to clinical stage in **Q4 2020**

(1) Positron emission tomography; (2) Parkinson's disease; (3) Non-human primates; (4) Data shown for 18F-labeled ACI-3710 by positron emission tomography (PET); (5) Square of the coefficient of multiple correlation; (3) Non-human primates; (4) Data shown for 18F-labeled ACI-3710 by PET[1]; (6) Injected dose per gram of brain; (7) Dementia with Lewy bodies; (8) Multiple system atrophy; (9) Alpha-synuclein gene mutation

AC Immune



6. Near-term inflection points

Multiple upcoming clinical catalysts to drive value in 2020

1st
Tau antibody
Phase 2 data expected

3
Tau programs reporting clinical data

5
clinical readouts expected this year

Phase 2 readouts

- **semorinemab** — mild / prodromal AD
- **ACI-24** — AD (interim data)

Phase 1b readouts

- **ACI-35.030**[1] — AD (interim data)
- **ACI-24** — Down syndrome

Phase 1 readouts

- **ACI-3024** — healthy volunteers

Initiation

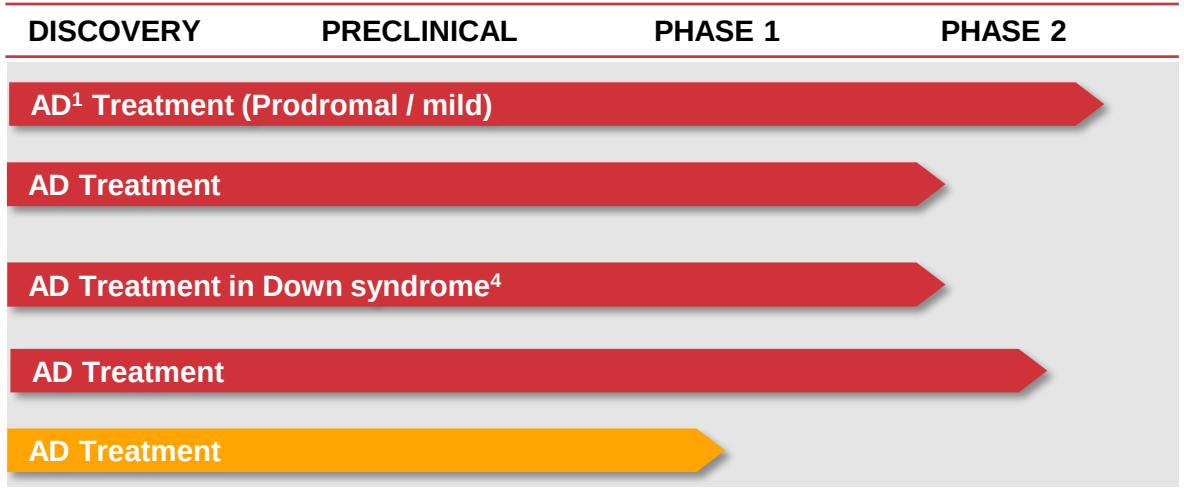
- **ACI-24** — Phase 2 – Down syndrome

(1) Phase 1b/2a study

Multiple upcoming catalysts to drive value in 2020

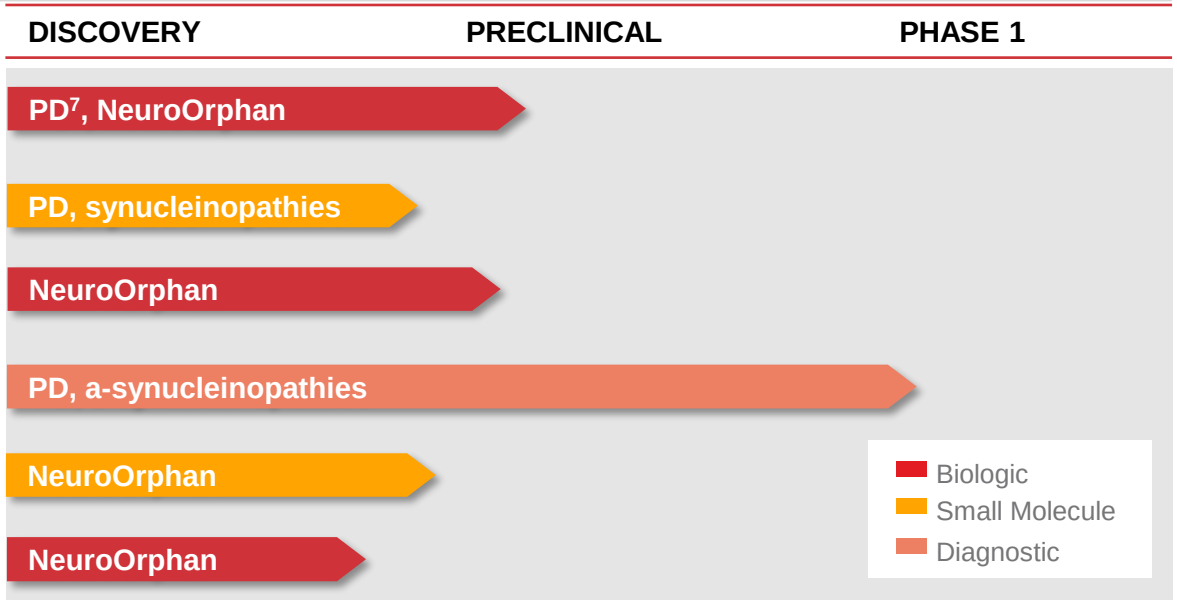
Clinical

Q2	**semorinemab** *(anti-Tau antibody)*	Phase 2 primary completion (estimated; last patient, last visit)
Q2	**ACI-35.030** *(anti-pTau vaccine)*	Phase 1b/2a in AD interim analysis[2]
H2	**ACI-24** *(anti-Abeta vaccine)*	Phase 1b full study reporting in Down syndrome[3]
H2	**ACI-24** *(anti-Abeta vaccine)*	Phase 2 12-month interim analysis in AD
H2	**ACI-3024** *(Tau inhibitor)*	Phase 1 results (healthy volunteers) disclosed by partner (expected)[5]

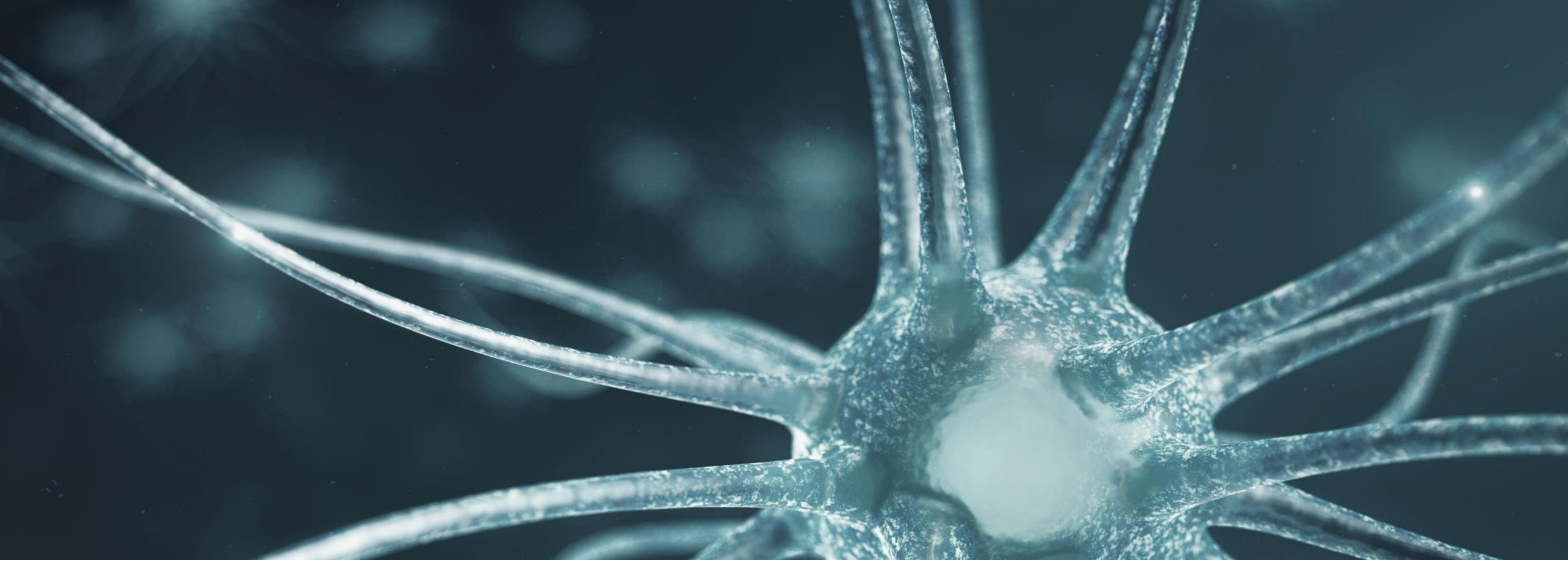


Preclinical

Q1	**anti-a-syn anibody**	Start IND[6]-enabling studies for lead candidate **(achieved ☑)**
Q2	**Morphomer a-syn** *(a-syn inhibitor)*	Identify first biologically active small molecule
Q2	**anti-TDP-43[8] antibody**	Declare clinical lead; start IND-enabling studies
Q4	**a-syn PET tracer**	Advance 3rd-gen candidate to clinical stage
Q4	**Morphomer-NLRP3[9]-ASC[10]**	Declare lead (non-CNS[11])
Q4	**anti-NLRP3-ASC antibody**	Declare pre-lead



(1) Alzheimer's disease; (2) Cohort 1; safety/tolerability; immunogenicity; (3) Phase 1b completion expected in Q2; (4) AD-like cognitive impairment associated with Down syndrome; (5) Phase 1 completion expected in Q2; (6) Investigational new drug; (7) Parkinson's disease; (8) TAR DNA-binding protein 43; (9) (NOD)-like receptor protein 3; (10) Apoptosis-associated speck protein containing a CARD; (11) Central nervous system



7. Financial figures

Substantial funds from partnerships complement equity investments

Distinguished institutional investors[1]

B.A:M

BlackRock

BVF PARTNERS L.P.

Fidelity INVESTMENTS

INVUS

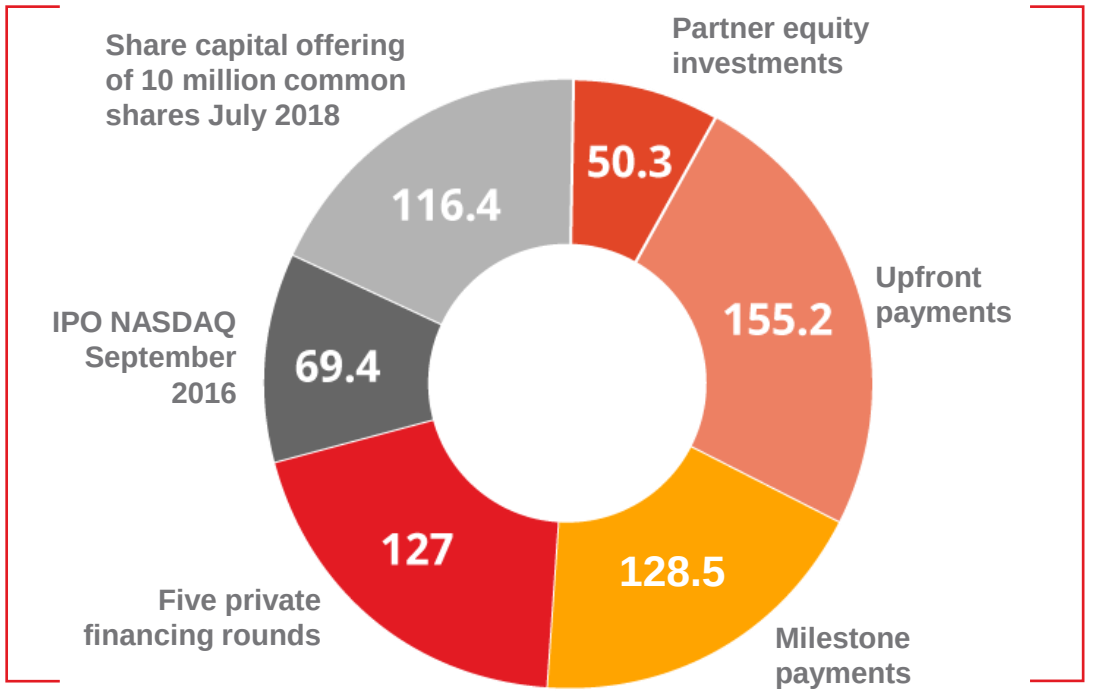
Prosight Capital

TEKLA Capital Management LLC

TEMASEK

Corporate funding to date[2] (in CHF millions)

CHF 313 million



CHF 334 million

- CHF 313 million from investor funds
- CHF 334 million in partnering related funds[3,4]
- CHF 3 billion in total potential payments plus potential royalties outstanding

(1) Based on latest schedule 13G and 13F filings; (2) Converted to CHF based on exchange rates at times of receipt; (3) Milestone payments as of March 31, 2020; (4) With Lilly convertible loan

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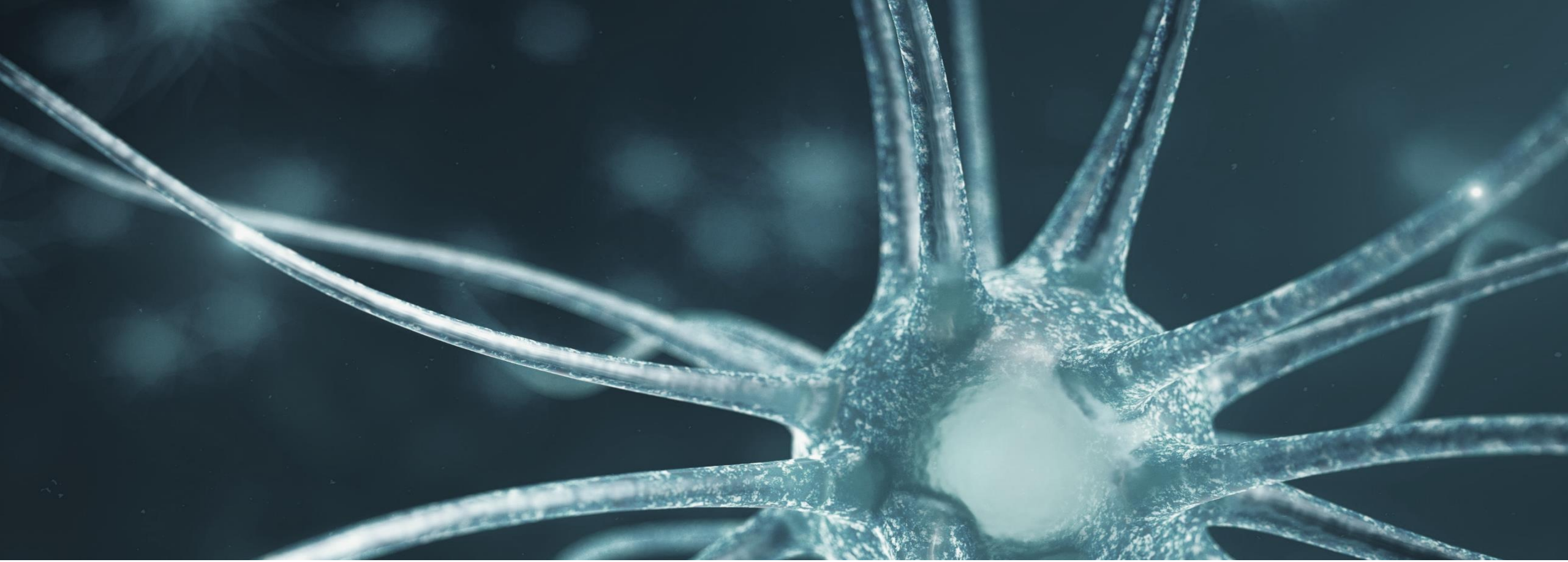
2019 Financial update

Strong cash position	▪ CHF 288.6 million compared to CHF 186.5 million at December 31, 2018
Substantial partnership revenues	▪ Received CHF 111.0 million in 2019, an increase of CHF 103.8 million compared to 2018
R&D expenses	▪ Increased by CHF 6.2 million year-over-year to CHF 50.4 million in 2019
G&A expenses	▪ Increased by CHF 3.6 million year-over-year to CHF 16.1 million in 2019
IFRS income/(loss)	▪ Net income after taxes of CHF 45.4 million in 2019, compared with net loss of CHF 50.9 million in 2018

Sufficiently funded to reach multiple value inflection points through at least Q1 2024[1]

(1) Excluding potential future milestone payments



8. Strategic outlook

Drivers of value creation in 2020 and beyond

Ongoing strong financial position

CHF 277.9 million in cash[1], ensuring the Company is fully financed through Q1 2024

Pipeline progression

Industry-leading molecules against multiple key targets; i.e. anti-a-syn and anti-TDP-43 antibodies advancing to preclinical development

5 clinical data readouts in 2020

Multiple near-term value inflection points, including the 1st Phase 2 readout of an anti-Tau antibody in Alzheimer's disease

Pioneering precision medicine

Addressing large market opportunity with differentiated, patient-focused approach

(1) As of March 31, 2020

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We continue to shape the future of neurodegeneration by discovering and developing breakthrough therapies through pioneering science and precision medicine