NOVEL THERAPEUTICS AND DIAGNOSTICS FOR ALZHEIMER'S AND OTHER NEURODEGENERATIVE DISEASES

AC Immune

Disclaimer

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About AC Immune

Based at the EPFL campus in Lausanne, Switzerland

Nasdaq listed in September, 2016 with net proceeds of $70.5 million

Ticker symbol: Nasdaq: ACIU

Approximately $725 million[1] market cap, 56.8 million shares outstanding

90 full-time employees

(1) as of February, 2018

Vision

*To become a global leader in **precision medicine**[1] of neurodegenerative diseases leveraging dual proprietary technology platforms to develop breakthrough therapies*

SupraAntigen™

Vaccines and antibodies specific to disease causing conformations

Morphomer™

Conformation-sensitive small molecules

(1) The goal of precision medicine is to deliver optimally targeted and timed interventions tailored to an individual's molecular drivers of disease.

Investment highlights

AC Immune: a leader in neurodegenerative diseases

1. Large and growing neurodegenerative disease market driven by significant unmet medical need

2. Proprietary technology platforms (SupraAntigen, Morphomer) as engines for sustained growth

3. Diverse product pipeline with complementary diagnostic agents in clinical development

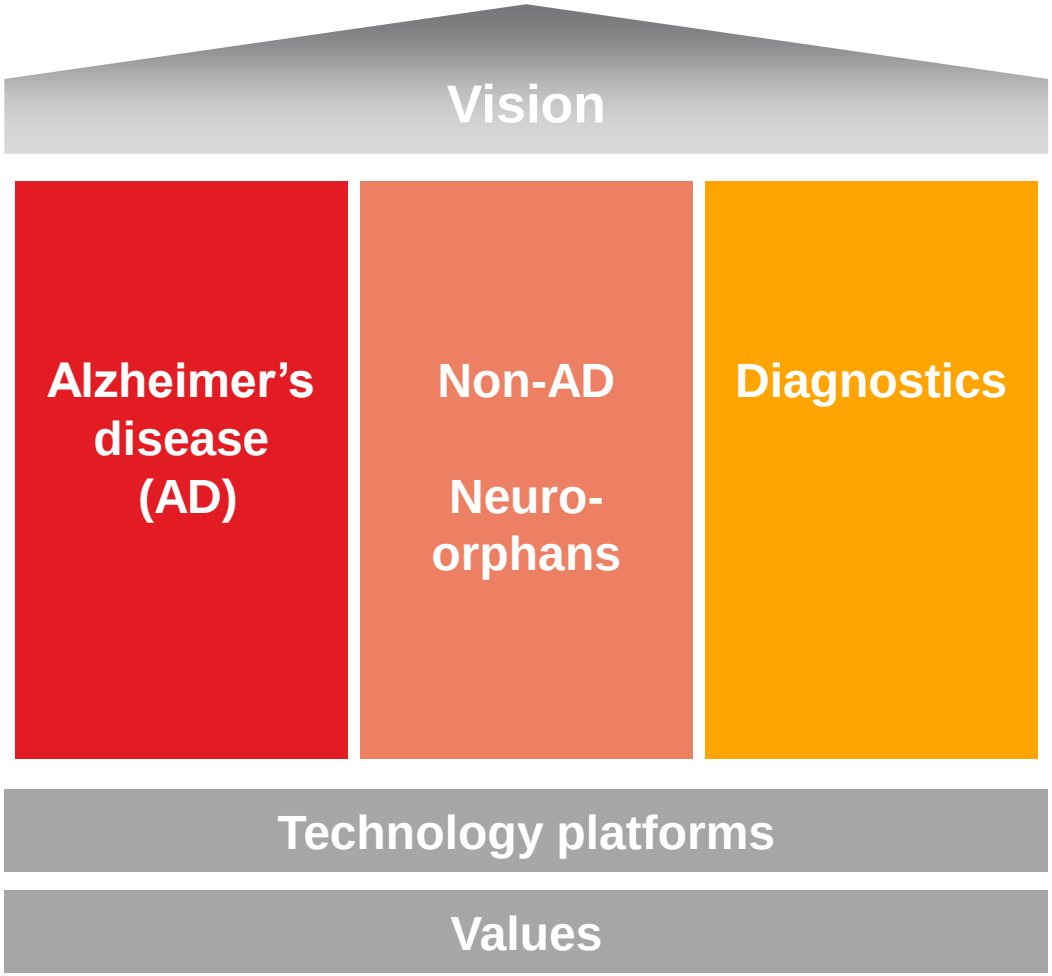
4. Lead product, crenezumab, in Phase 3 development with compelling Phase 2 data and favorable safety profile

5. Well-positioned financially with CHF 117.2 m in cash, enough through min. Q1 2019. Increasing investment into key areas of neuro-orphan and neuro-inflammation

6. Multiple high-profile strategic alliances with leading industry partners

Business strategy: 3-pillar approach

Precision medicine creates ultimate differentiation



Alzheimer's disease

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

Non-AD, neuro-orphans

- Discover therapeutics in Parkinson's disease
- Leverage AD therapeutics in Down syndrome (DS), PSP[1] and other neuro-orphan diseases

Diagnostics

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

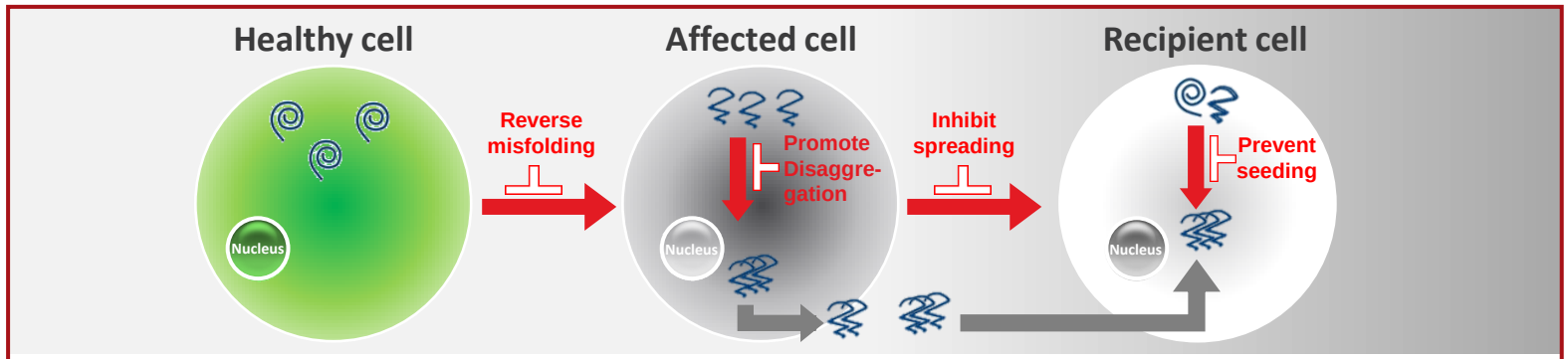
(1) Progressive supranuclear palsy

High-Science approach to proteinopathies

Dual platforms enable discovery and opportunity for synergistic development

De-risked

- Global disease mechanism of proteino-pathies
- Protein targets
- Dual tech platforms
- Precision medicine approach and analysis
- Multiple indications



Abeta	Tau	α-synuclein	Other proteinopathy targets, i.e. TDP-43

SupraAntigen Platform | **Morphomer Platform**

- Proprietary diagnostics
- Biomarkers
- BBB/Target engagement

AD and non-AD proteinopathies including neuro-orphan[1]

- **Proteinopathies are efficiently adressed by a common scientific approach, complemented with proprietary diagnostic**
- **Many proteinopathies have orphan status**

(1) non-AD proteinopathies: Parkinson's disease; Down syndrome, progressive supranuclear palsy (PSP); Frontotemporal dementia (FTD); Dementia with Lewy Bodies; cerebral amyloid angiopathy; myotonic dystrophy; corticobasal degeneration; Pick's disease; amyotrophic lateral sclerosis; chronic traumatic encephalopathy

AC Immune

Technology platforms

Product-focused and highly versatile platforms drive growth

SupraAntigen™

Vaccines and antibodies specific to disease causing conformations

Morphomer™

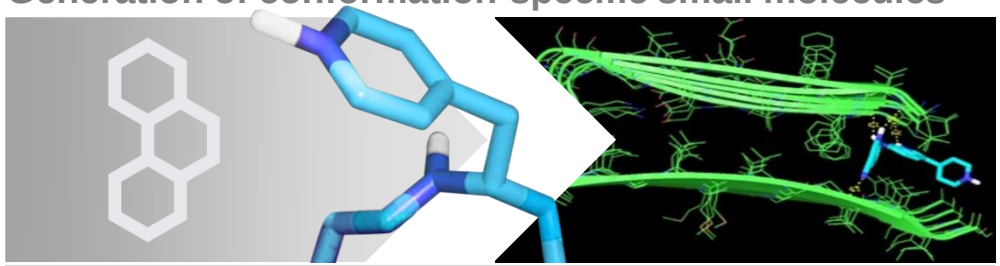
Conformation sensitive small molecules

Immunotherapy against conformation-specific targets

- Highly selective conformation-specific immunotherapy
- Antibodies and vaccines
- Rapid antibody response
- Favorable safety (T-cell independent)

- **Crenezumab**[1] in AD (Ph 3)
- **ACI-24**[1] in AD (Ph 1/2a) and DS (Ph1b)
- **ACI-35**[2] in AD (Ph 1b)
- **Anti-Tau antibody**[2] in AD (Ph 2)
- **α-synuclein**[3]**/TDP-43**[4] **antibodies** in PD and neuro-orphan indications (pre-clinical)

Generation of conformation-specific small molecules



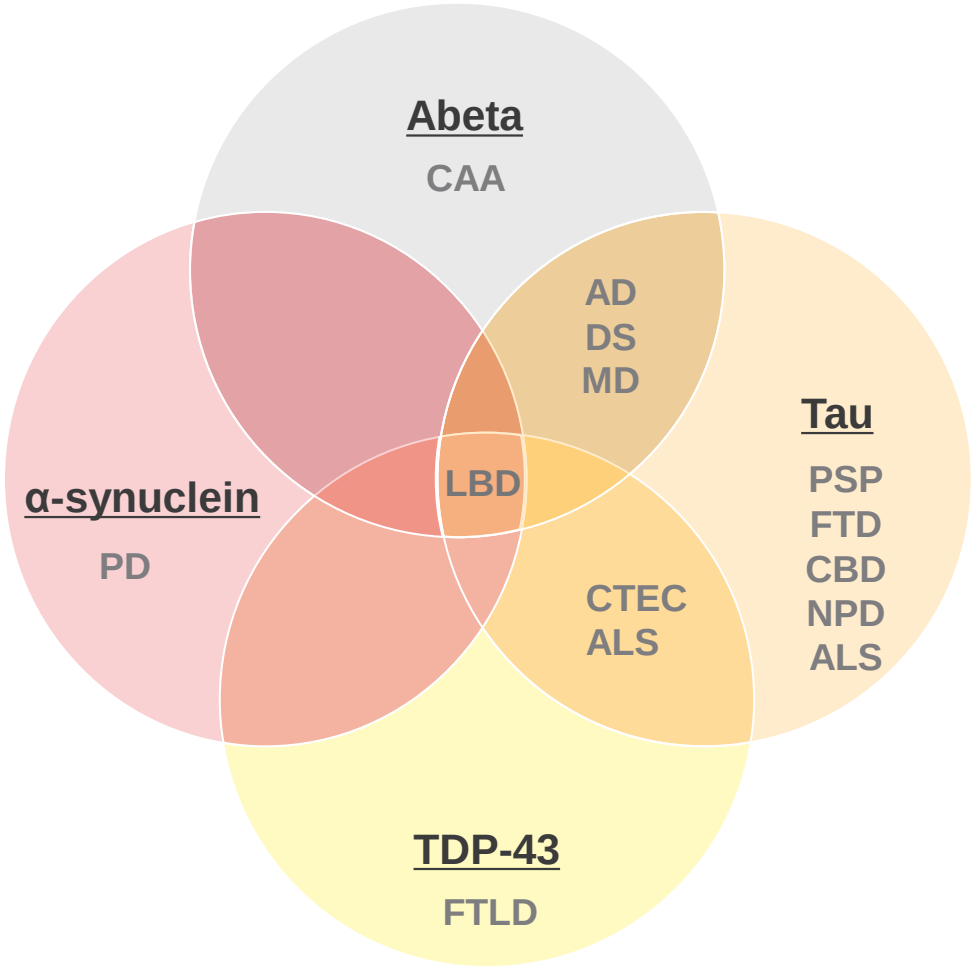
- Conformation specific small molecules through rational design
- Robust library of small molecules
- Protein propagation inhibitors

- **Tau-PET imaging agent**[2] in AD and PSP (Ph 1)
- **Morphomers for different targets**[1,2,3] in AD and PD (discovery / pre-clinical)
- **α-syn-PET imaging agent**[3] in PD (pre-clinical)

(1) Abeta (2) Tau (3) α-synuclein (4) TDP-43

AD and other neurodegenerative diseases share MoA and targets

Significant market potential



Market opportunity

Disease	US data	
	Incidence (per 100,000)	Patient population ('000)[1]
Alzheimer's (AD)	1,500	5,000
Parkinson's (PD)	160	500
Frontotemporal dementia (FTD)	15[2]	–
Amytrophic lateral sclerosis (ALS)	1[3]	30
Dementia with Lewy bodies (LBD)	400	1,300
Frontotemporal lobar degeneration (FTLD)	17	55
Cerebral amyloid angiopathy (CAA)[5]	–	–
Down's syndrome (DS)	79	255
Corticobasal degeneration (CBD)	6	19
Pick's (NPD)	7-43[4]	–
Myotonic dystrophy (MD)	13[3]	–
Progressive supranuclear palsy (PSP)	1	3
Chronic traumatic encephalopathy (CTEC)[5]	–	–

Source: Industry publications and World Bank
(1) Calculated as incidence multiplied by US population of 323m as of 2016 year end; (2) Patients aged between 45-64 years; (3) Worldwide incidence; (4) European incidence;
(5) Estimated prevalence data unavailable

AC Immune's robust pipeline

Driven by proprietary technology platforms

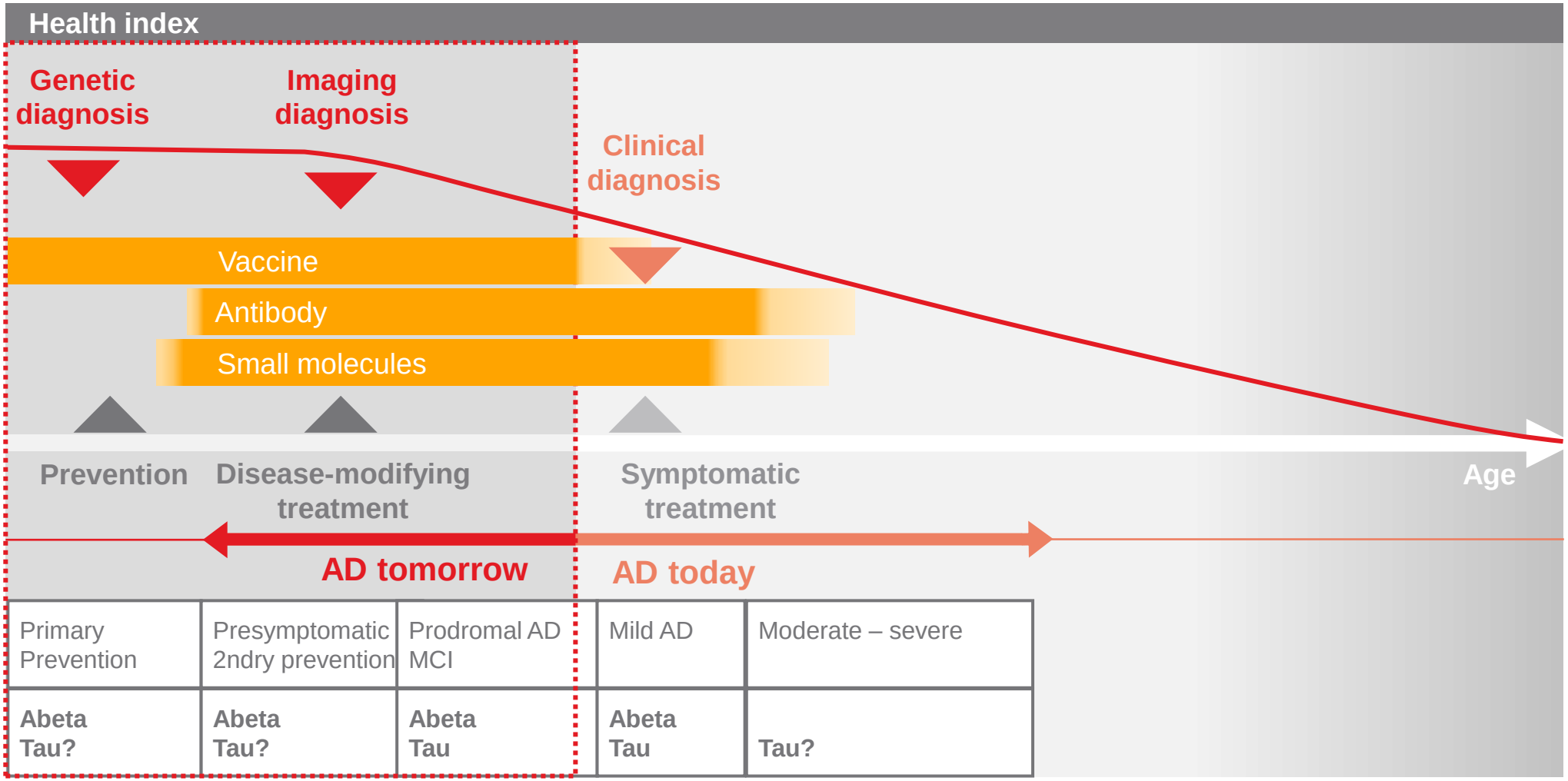
	Product candidate	Target	Partner	Discovery	Pre-clinical	Phase 1	Phase 2	Phase 3
Alzheimer's disease	**Crenezumab** (anti-Abeta antibody)	Abeta	Genentech	AD treatment				
		Abeta		AD prevention				
	ACI-24[1] (anti-Abeta vaccine)	Abeta		AD treatment				
	ACI-35 (anti-pTau vaccine)	Tau	Janssen	AD treatment				
	Anti-Tau antibody	Tau	Genentech	AD treatment				
	Morphomer Tau (Tau inhibitor)	Tau		AD treatment				
Non-AD Neuro-orphan	**ACI-24** (anti-Abeta vaccine)	Abeta		Down sydnrome[2]				
	Morphomer Abeta (Abeta inhibitor)	Abeta		Glaucoma				
	Morphomer α-syn (α-synuclein inhibitor)	α-synuclein		Parkinson's				
	Anti-α-syn antibody	α-synuclein						
	Anti-TDP-43 antibody	TDP-43						
Diagnostics	**Tau-PET imaging agent**[3]	Tau	Piramal Healthcare	AD and PSP				
	IVD[4] (Tau, Abeta)	Abeta/Tau		AD				
	α-syn-PET imaging agent	α-synuclein	Biogen	Parkinson's				

Biologics Small molecules Diagnostics

(1) In process of completing a Phase 1/2a study; (2) AD and cognitive impairment associated with Down syndrome; (3) Positron emission tomography; (4) *in-vitro* diagnostic

Alzheimer's disease treatment

Early diagnosis translates into earlier treatment and better outcome



- The future treatment paradigm for neurodegenerative diseases may involve **different disease-modifying treatments used at various points in the progression of the disease**
- Possible **combination** therapies:
  - Passive immunization targeting Abeta (e.g., crenezumab) together with anti-Tau antibodies
  - Immunotherapies and small molecules targeting Abeta or Tau

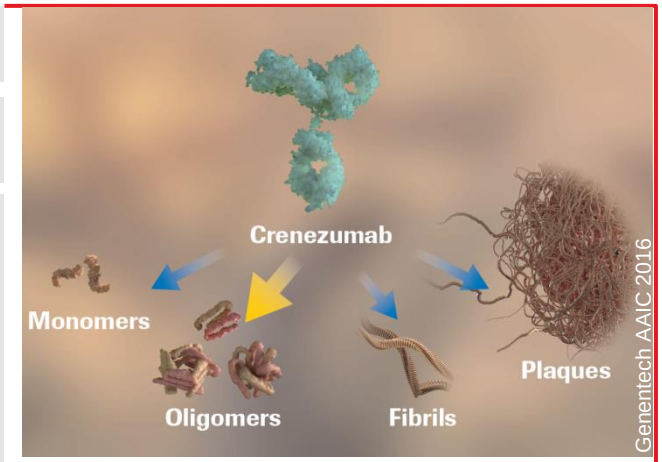
Pipeline

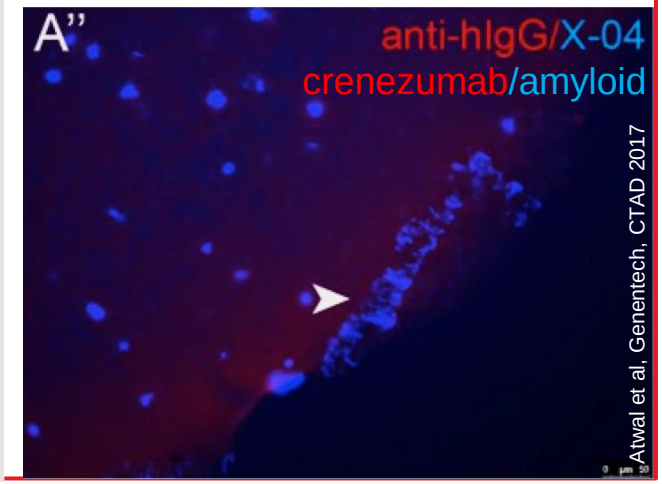
Crenezumab – Phase 3 in AD

Target	Misfolded Abeta
Licensed to	Genentech *A Member of the Roche Group*

Key results in pre-clinical studies	▪ Unique epitope, breaks up Abeta aggregation and prevents assembly
	▪ Binds to monomers, oligomers (10x higher affinity to soluble oligomers) and fibrils of Abeta
	▪ Crystal structure supports ability to block aggregation and promote disaggregation
	▪ Reduced risk of ARIA-E and neuro-inflammation allows for higher dosing attributable to
	  ▪ Low effector function of IgG4 backbone limiting inflammatory cytokines
	  ▪ Lack of binding to vascular amyloid and dense core of Abeta plaques

Development status	▪ Phase 3 commenced in 2016 (CREAD 1) and 2017 (CREAD 2), fast-track designation ▪ Encouraging Phase 2 data in mild patients ▪ First-in-class drug in AD prevention trial (Phase 2)





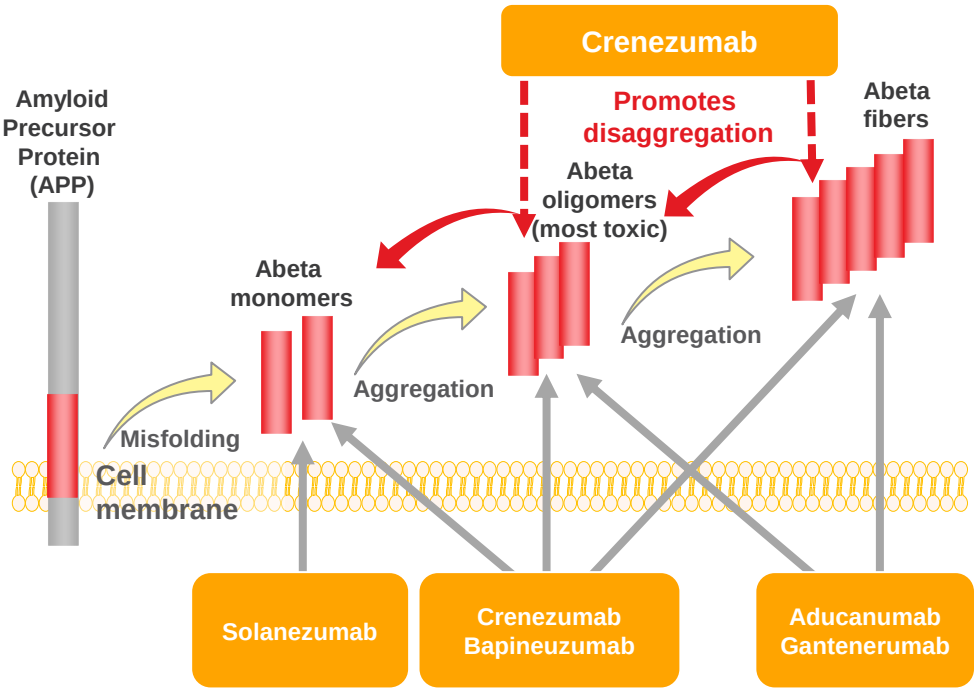
(1) ARIA-E = Amyloid Related Imaging Abnormality-Edema

AC Immune

Crenezumab

Compelling binding characteristics with unique disaggregation and safety profile

Multiple neuroprotective mechanisms of action



Uniquely differentiated binding profile with favorable preliminary safety profile

Antibody	Binding profile	Stage	Phase 3 dosage Clinicaltrials.gov	Iso-type	ARIA-E (safety)
Crenezumab (GNE/Roche/ AC Immune)	Monomers + Oligomers +++ Fibrils ++	Ph 3	60mg/kg	IgG4	< 0.2% in Ph2[1]
Aducanumab (Biogen/Eisai)	Oligomers +++ Fibrils +++	Ph 3	ApoE4+: 3 or 10 mg/kg ApoE4-: 6 or 10mg/kg	IgG1	41% , 37% and 35% in Ph1b (DB)[2]
Gantenerumab (Roche/ Morphosys)	Oligomers ++ Fibrils +++	Ph 3	Double blind (DB): 1.5 or 3.2mg/kg Open Label (OLE): up to 17.1mg/kg	IgG1	10% in DB 22.9% in OLE[3]
Solanezumab (Eli Lilly)	Monomers +++	Ph 3 failed	5.7 mg/kg	IgG1	1% in Ph3[4]
BAN2401 (Eisai/Biogen)	Soluble Protofibrils +++ Fibrils +	Ph 2	2.5mg/kg 5 mg/kg 10mg/kg	IgG1	0% in Ph1[5]
Bapineuzumab (Elan/Pfizer/J&J)	Monomers ++ Oligomers +++ Fibrils ++	Ph 3 failed	0.5mg/kg 1 mg/kg	IgG1	~10% in Ph3[6]

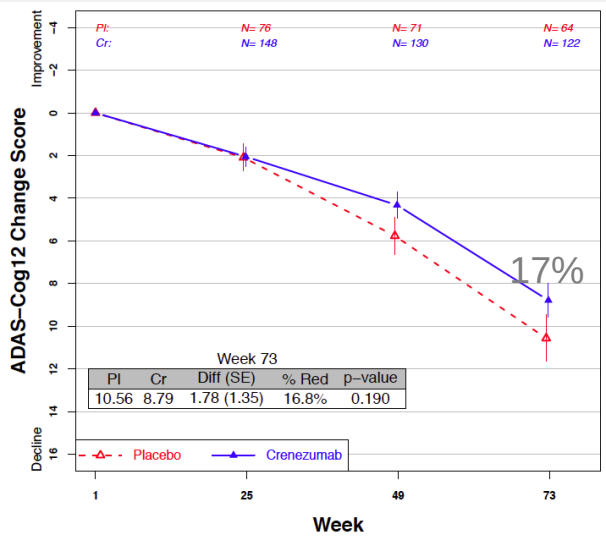
Crenezumab's multiple neuroprotective mechanisms of action, in particular direct binding and inhibition of toxic Abeta oligomers, may differentiate crenezumab's clinical benefit

(1) Lin et al, CTAD 2017; (2) Budd-Heaberlein, JPAD 2017; (3) Andelkovic, CTAD 2017; (4) Siemers et al, Alzheimer's & Dementia 2016; (5) Logovinsky et al, Alzheimer's Research & Therapy 2016; (6) Salloway et al, New Engl J Med 2014

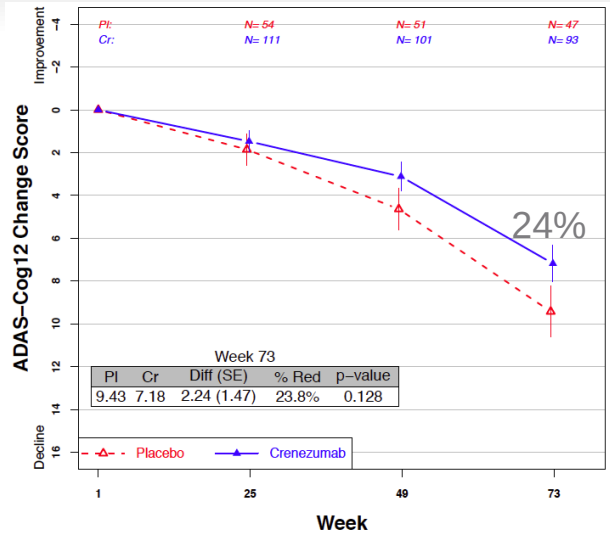
Crenezumab – Phase 2 results

ABBY cognition study high dose IV cohort
Stronger performance in milder patients (ADAS-cog 12)

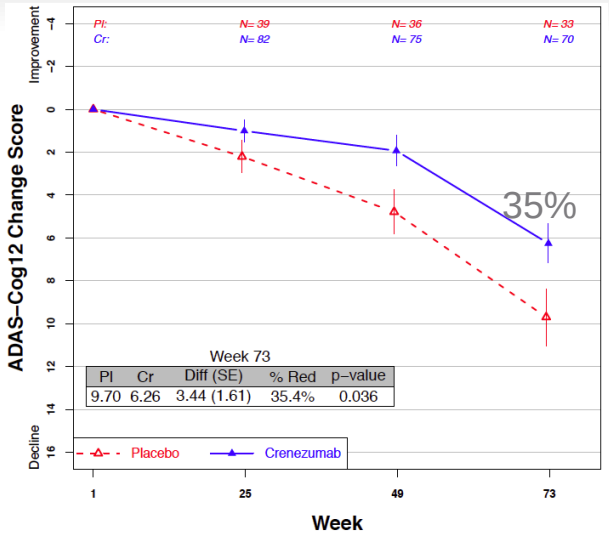
Mild-to-moderate (MMSE 18-26)



Mild (MMSE 20-26)



Milder (MMSE 22-26)



Cummings, presentation AAIC, July 2014

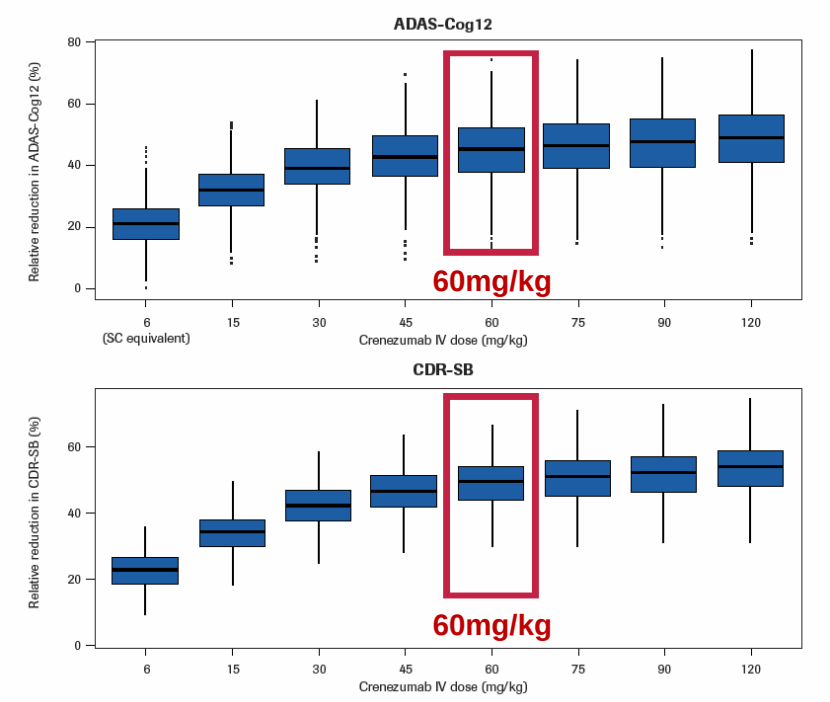
Mild (MMSE 20-26): pre-specified analysis of data
Milder (MMSE 22-26): non-prespecified exploratory analysis of data

- Significant 35% reduction in cognitive decline in milder patients (p=0.036)
- In the mild and moderate patient population, a positive trend in cognition was observed although statistical significance was not achieved
- Consistent effects increasing over time

Crenezumab – Phase 3

Anti-Abeta antibody with potential to become best-in-class disease modifying treatment for AD

Dose-response simulation on cognitive endpoints in patients with mild AD (MMSE 22-26)



Polhamus et al., poster CTAD, 2016

- Choice of the dose for Phase 3 based on modelling of results from the Phase 2 in a drug-disease model
- Antibody exposure needed for maximal cognitive and clinical effect reached at 60/mg/kg
- Phase 1 safety results support use of 60mg/kg in Phase 3

Key ongoing clinical studies

Pivotal CREAD 1 and CREAD 2 trial design builds on ABBY/BLAZE findings and latest Abeta understanding

Study design

- 750 patients with prodromal to mild AD per study
- 60mg/kg every four weeks (4x higher than Phase 2 ABBY)

Key Eglibility

- MMSE 22+ and CDR-GS 0.5/1.0
- Brain amyloid positivity
- 50-80 years of age

Endpoints

- Primary endpoint: CDR-SB at 105 weeks
- Key secondary endpoint: ADAS-cog 13 at 105 weeks
- Other endpoints: safety, biomarkers and economic

Study timelines

- CREAD 1 started in Q1 2016 – expected data 2020
- CREAD 2 started in Q1 2017 – expected data 2021

API-ADAD prevention trial in Colombian population

- 300 cognitively healthy individuals of whom 200 are genetically predisposed to develop early AD
- Study started in Q4 2013

www.clinicaltrials.gov

ACI-24 – Phase 1/2a in AD and Phase 1b in DS

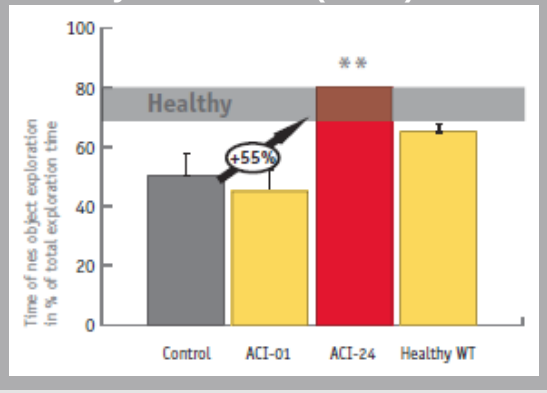
Anti-Abeta therapeutic vaccine

Target	Misfolded Abeta
Key results in pre-clinical studies	▪ Strong and robust antibody response[1] specific for oligos and fibrils ▪ Favorable safety profile with lack of local inflammation and T-cell independent mode-of-action[1] ▪ Significant reduction of Abeta levels in brain and compelling memory enhancement (AD and DS models)

AD development status	▪ **Clinical Phase 1/2a (in-house) with interim data**
	  ▪ Positive safety and tolerability
	  ▪ Cohort 3 showed trend of reduction of accumulation of brain amyloid (PET imaging)
	  ▪ Cohort 3 showed trend of reduction of clinical decline (CDR-SB)

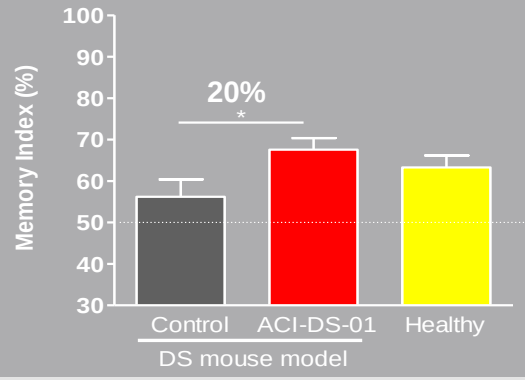
DS development status	**Clinical Phase 1b with interim data expected in 2018** ▪ World first clinical trial for vaccine targeting AD in people with Down syndrome ▪ Dose escalation study in up to 24 adults with Down syndrome (25-45 years) ▪ Endpoints: safety and tolerability, effect on induction of anti-Abeta antibodies, biomarkers for Abeta brain and CSF load ▪ Recruitment of low-dose cohort completed in Q3 2017

Memory restoration (ORT[3]) in AD model



Muhs et al, PNAS 2007

Memory restoration (ORT[3]) in DS model



Belinchenko et al, PLOS ONE 2016

(1) Pihlgren et al., Blood 2013; (2) ELISA = Enzyme Linked Immunosorbent Assay; (3) ORT = Object Recognition Test

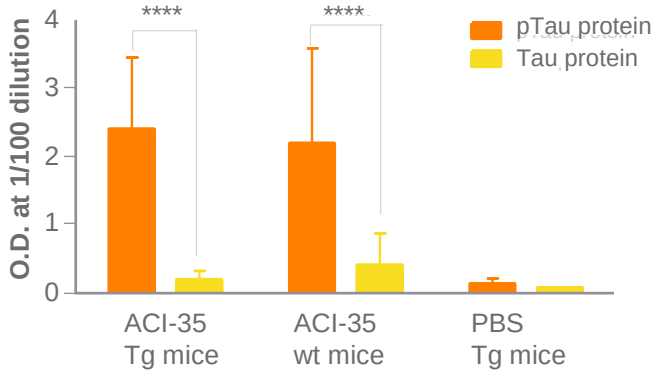
ACI-35 - Phase 1b in AD

Anti-pTau therapeutic vaccine

Target	Aggregated pTau
Licensed to	janssen | PHARMACEUTICAL COMPANIES of Johnson & Johnson
Key results in pre-clinical studies	▪ High specific antibody response to pathogenic Tau ▪ Improvement of cognition, physical performance, behavior and prolongation of survival ▪ Favorable safety profile with T-cell independent mode-of-action
Development status	▪ Clinical Phase 1b with interim data ▪ Acceptable safety and tolerability ▪ Dose-dependent and target-specific antibody response to pTau ▪ AC Immune and Janssen jointly decided to advance anti-Tau vaccine program ▪ Scientific advisory meeting with regulatory agencies to support next phase of development

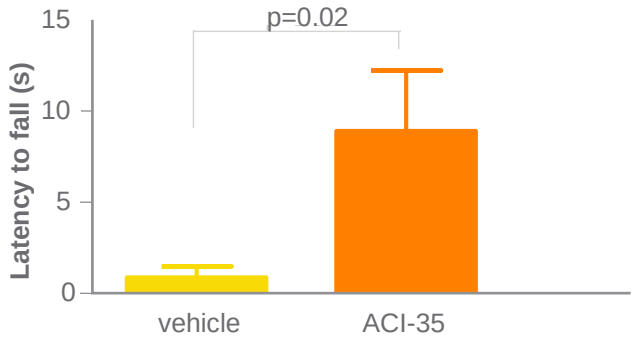
Immune response highly specific to phosphorylated Tau

ACI-35 vaccinated mice - pTau vs.Tau protein after 5 immunizations



Highly significant improvement of behavior (P301S)

15 rpm ACI-R-40 Rotarod 5M vehicle vs. ACI-35



AC Immune unpublished data

(1) Tg = Transgenic; (2) wt = wild type

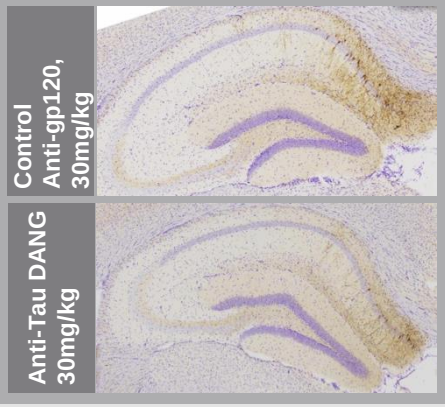
Anti-Tau antibody - Phase 2 in AD

Anti-Tau antibody (RO7105705)

Target	Designed to intercept the cell-to-cell spread of pathological tau in extracellular space of brain
Licensed to	Genentech, A Member of the Roche Group
Key pre-clinical results	▪ Tau pathological spread is dose dependently reduced independent of effector function ▪ Proven target engagement through dose-dependent rise of plasma Tau (mice, cynos)

Pre-clinical results

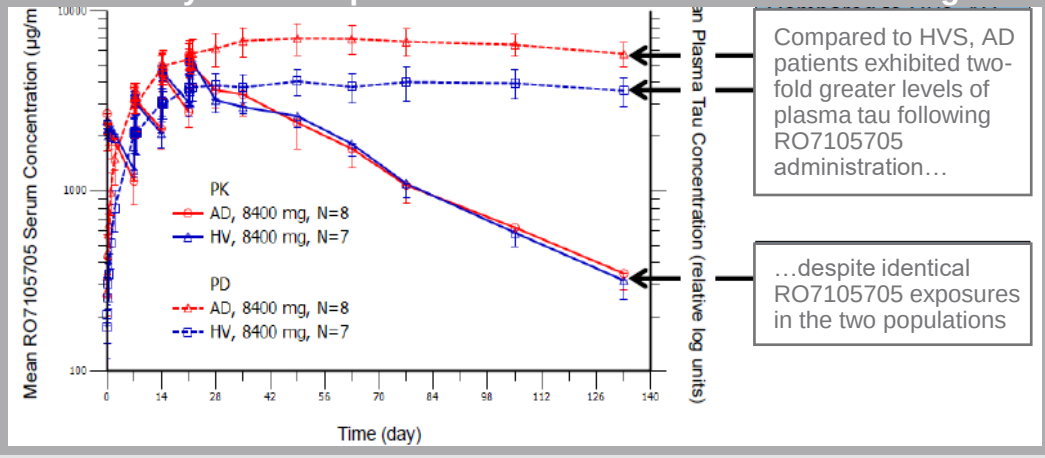
Dose dependent reduction of Tau pathology



AD/PD conference, Vienna, April 2017

Clinical results

Pharmacodynamic response: Plasma Tau concentration 2x higher in AD than in HV[1]



Kerchner et al, CTAD 2017

Development status	**Phase 1 data** ▪ No dose-limiting toxicities up to high doses ▪ Dose-proportional PK with median half-life of 32.3 days ▪ Detectable in CSF, indicating CNS exposure ▪ Pharmacodynamic response: 2x greater plasma Tau concentrations observed in patients with AD than in HVs **Phase 2 design** ▪ 360 prodromal-to-mild AD patients (MMSE 20-30, CDR-GS 0.5 or 1) ▪ 3 active doses or placebo for 72 weeks, followed by 96 week open label study ▪ Primary endpoints: safety measures and CDR-SB

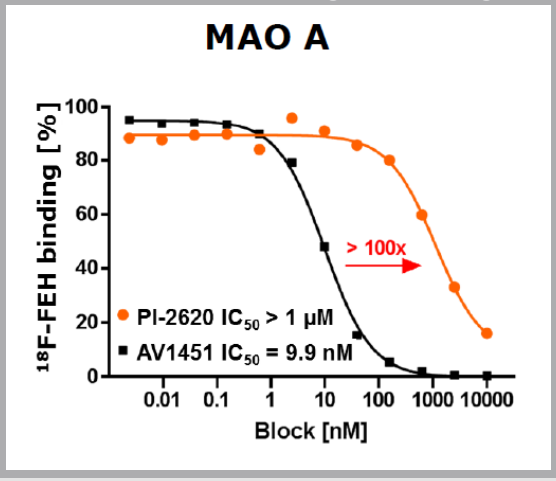
(1) Healthy volunteers

Tau-PET imaging – Phase 1 in AD and PSP

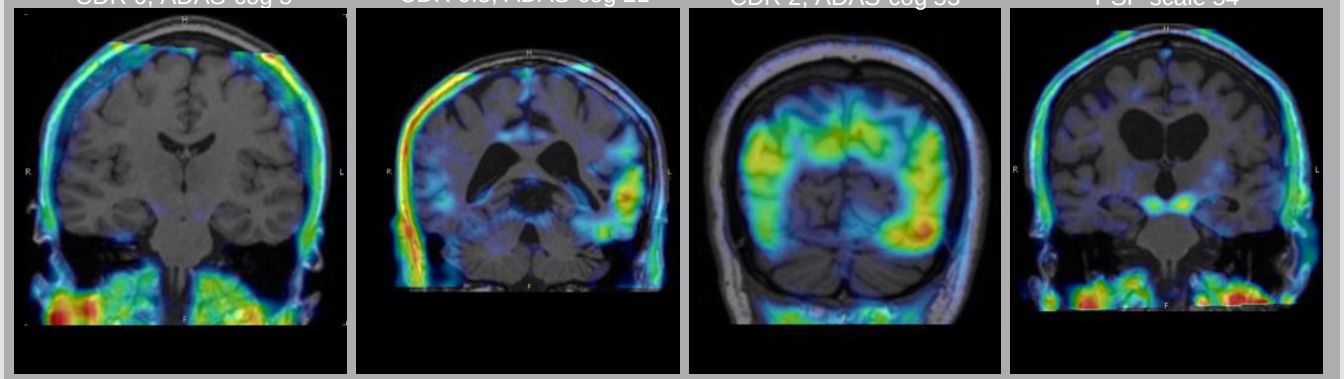
Morphomer Tau PI-2620

Target	Misfolded Tau (4R and 3R)
Licensed to	Piramal Imaging
Key results	▪ High specificity for pathological forms of human Tau in AD and other tauopathies ▪ Outstanding PET tracer-profile – excellent brain penetration and high selectivity even in early disease stage

Pre-clinic: High selectivity and absence of off-target binding



Phase 1 clinical study: distinct, specific Tau distribution pattern in AD and PSP

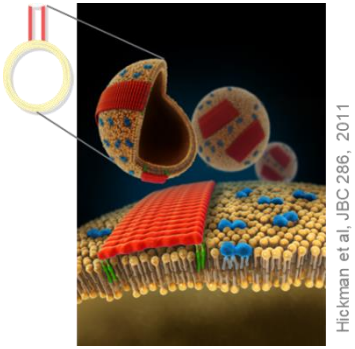
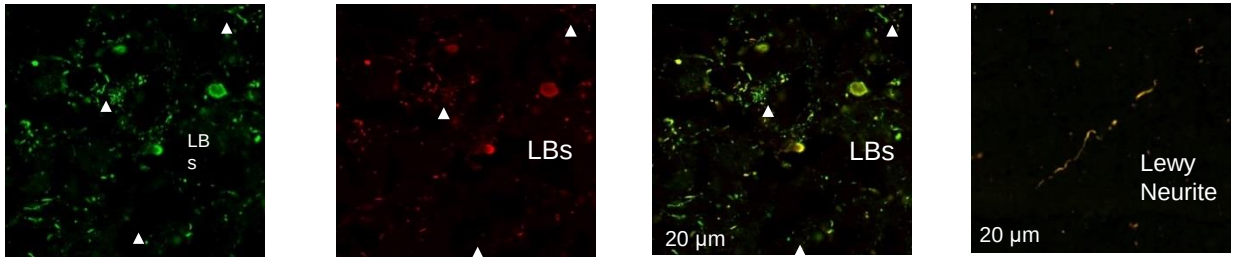


Stephens, AD/PD conference, Vienna, April 2017

Development status	▪ Clinical Phase with interim data
	  ▪ Fast kinetics with robust brain uptake, fast wash-out in non-target regions and low off-target uptake
	  ▪ Distinct and specific Tau distribution pattern in AD and PSP subjects
	  ▪ Good reproducibility of PET-scans confirmed by test-retest study

(1) NDC = non-demented control; (2) SN = substantia nigra

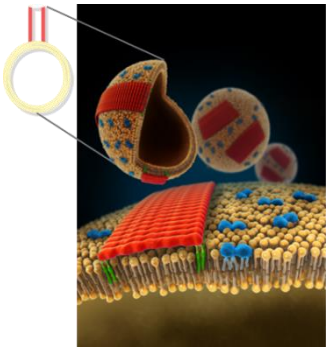
Anti-a-synuclein (a-syn) antibody – Discovery in PD

Target	Misfolded, aggregated a-syn
Target characteristics	▪ Pathological a-syn aggregates and forms oligomers and fibrils ▪ Mutated and post-translational mutations enhance a-syn misfolding ▪ Aggregation and spreading of misfolded a-syn are linked to synucleinopathies as shown in patients and animal models
Preclinical results	▪ Several antibodies with specificity and high target affinity for pathological human a-syn generated (KD down to 100pM) ▪ Target binding shown for PD[1], DLB[2] and MSA[3] in human brains from multiple patients
SupraAntigen Platform	 SupraAntigenTM platform is ideally positioned to generate antibodies selective for the alpha-syn pathology **Target staining (IHC) on PD brain sections** Amygdala | Cingulate Cortex a-syn Clone 1 | p-syn[4] | a-syn Clone1/ p-syn[4] | a-syn Clone 1/ p-syn[4] 

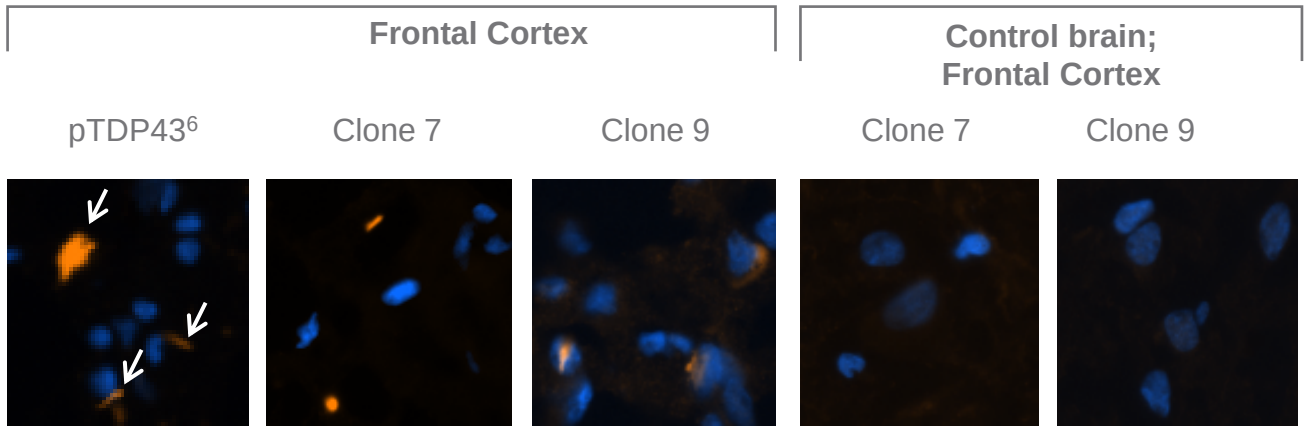
Next steps	▪ Select lead antibodies ▪ Efficacy studies in a-syn animal models ▪ IND enabling studies

(1) PD, Parkinson's Disease; (2) Dementia with Lewy bodies; (3) MSA, Multiple System Atrophy, (4) p-syn antibody (pSer129; Abcam UK)

Anti-TDP-43[1] antibodies – Discovery phase

Target	Aggregated TDP-43
Target characteristics	▪ TDP-43 is a RNA/DNA binding protein involved in RNA metabolism ▪ Aggregated TDP-43 loses its physiological function and the extracellular pathological protein is involved in spreading of the pathology ▪ TDP-43 pathology is found in multiple neurodegenerative diseases such as FTD[2], AD, HD[3], ALS[4] and CTE[5]
Preclinical results	▪ Several antibodies generated with unique binding profiles to the pathological, aggregated human TDP-43 ▪ High target binding shown for human FTD brain; binding affinity range (KD 0.2 – 1.6 nM)
SupraAntigen Platform	 Hickman et al, JBC 286, 2011 SupraAntigenTM platform is ideally positioned to generate antibodies with selectivity to TDP-43 pathology

Target staining (IHC) on FTD brain sections



Next steps	▪ Expansion of anti-TDP-43 antibody library ▪ Analysis of extra-cellular TDP-43 in human brain tissue and CSF samples ▪ Selection of lead antibodies for efficacy animal studies

(1) TDP-43, TAR DNA—binding protein 43; (2) FTD, Fronto-temporal dementia; (3) HD, Huntington's Disease; (4) ALS, Amyotropic Lateral Sclerosis; (5) CTE, Chronic Traumatic Encephalopathy; (6) pTDP-43 antibody; CosmoBio

Financial overview and catalyst timeline

Financial highlights

- Cash position as of Sept. 30, 2017: CHF 117.2 million
- Quarterly burn-rate: CHF 13.5 to 15 million
- Cash runway: Fully funded through 2019
- Pre-IPO financing rounds raised approx. $130 million[1]
- Net proceeds from September 2016 IPO: $70.5 million
- Funding through partnering activities including potential payments of more than $1.4 billion; $1.24 billion outstanding
  - CHF 14 million milestone for first patient dosing in anti-Tau antibody phase 2 accrued in Q4 2017
- Analyst coverage: Credit Suisse, Leerink, Jefferies

(1) exchange rate fixed as of closing date of last financing round

Successful execution of strategy with supportive near-term milestones

	Achievements 2017	Key milestones for 2018/19
Data read-outs	✓ ACI-24 in AD: Encouraging interim data of Phase 1/2a of ACI-24 ✓ ACI-35: Encouraging interim data of Phase 1b; Joint decision with Janssen Pharma to move program forward and scientific advice from regulatory authorities for next phase of development ✓ Crenezumab Phase 1 study findings support use of a 4x higher dose (60 mg/kg) in Phase 3 than in Phase 2 ✓ Tau-PET imaging agent in AD: Encouraging pre-clinical and Phase 1 data with favorable kinetics and densitometry; specific binding to different Tauopathies	▪ ACI-24 in AD Phase 1/2a (safety data) reporting Q1 2018 ▪ ACI-35 in AD Phase 1b reporting Q1/Q2 2018 ▪ ACI-24 Phase 1b in DS interim data in 2018 ▪ Morphomer Tau IND enabling studies in 2018 ▪ α-Synuclein PET imaging IND enabling studies in H1 2018 ▪ α-Synuclein antibodies lead selection in 2018 ▪ TDP-43 antibodies lead selection in 2019
Study initiations	✓ Crenezumab: Second pivotal Phase 3 trial of CREAD 2 started by Genentech ✓ anti-Tau antibody: Phase 2 based on Phase 1 data started by Genentech	▪ ACI-24 in AD Phase 2 in 2018 ▪ ACI-35 next phase of clinical development based on Phase 1b data and scientific advice in 2018 ▪ Tau-PET imaging agent longitudinal study in 2018 ▪ α-Synuclein PET imaging agent start of Phase 1 in H2 2018

Strategy for value creation

CONTINUE to leverage our dual platform technologies to efficiently advance commercially viable product candidates

INVEST resources to further establish leadership in neurodegenerative diseases and complement existing technology leads

- Accelerate the advancement of our diagnostic portfolio
- Pursue research in neuroinflammation
- Continue to explore new targets

EVOLVE strategy to develop late stage assets in-house

EXPAND into other neurodegenerative and neuro-orphan diseases

- Pursuing neuro-orphan indications may enable us to obtain a streamlined regulatory approval pathway and favorable reimbursement treatment of any approved product

Additional information

Leadership team

Proven management and world-leading science

Executive officers

Andrea Pfeifer, Ph.D.

CEO

- Head of Nestlé Research
- Co-founder of Nestlé's VC-fund

Andreas Muhs, Ph.D.

CSO

- Director of Preclinical Research, ViaCell
- Director of Pharmacology, Cardion

Joerg Hornstein

CFO

- VP/Divisional CFO, Merck Millipore
- CFO, Merck Serono China, Merck Indonesia

Jean-Fabien Monin

CAO

- CFO, bioMérieux Central Europe

Other members of the leadership team

Olivier Sol, M.D.

Head of Clinical Team

- Medical & Regulatory Aff. Director, Diaxonhit
- Clinical and medical expert Janssen, UCB-Pharma, GlaxoSmithKline and Sanofi

Julian Gray, M.D., Ph.D.

Clinical Advisor

- Clinical development expert neurological diseases (Roche, Eisai, Sandoz)

Joseph Wettstein, Ph.D.

CSO Deputy

- Head of Functional Neuroscience, Hoffman-La Roche

David Lowe, Ph.D.

Innovation Fellow

- CSO and VP R&D, Psychogenics
- CSO, Head R&D, Memory Pharmaceuticals

Key partners

External validation of technologies and platforms

Well-regarded foundations / institutions

- The Michael J. Fox Foundation for Parkinson's Research
- Banner Alzheimer's Institute
- NIH National Institutes of Health – Turning Discovery Into Health
- Research Down Syndrome Lumind Foundation

Highly committed investors

- dievini – advisors in health sciences
- VARUMA
- Fidelity Investments
- TEMASEK
- ESSEX

High-value partnerships

- Genentech – A Member of the Roche Group
  - Up to $340m in potential payments; signed 2006
  - Up to $418m in potential payments; signed 2012
- Janssen – Pharmaceutical Companies of Johnson & Johnson
  - Up to $509m in potential payments; signed 2014
- Piramal | Life Sciences – knowledge action care
  - 2014, significant value
- Nestlé Institute of Health Sciences
  - 2015
- Biogen
  - 2016
- ESSEX
  - 2017

- **Four out-licensing agreements over $1.4 billion in value and three research collaborations**
- **Five private financing rounds totalling ~$130 million[1]**
- **IPO NASDAQ September 2016 raised $70.5 (CHF 69.4) million in net proceeds**
- **More than 300 pending patent applications**
- **More than 260 granted patents**

(1) exchange rate fixed as of closing date of last financing round

Collaboration agreements

Summary overview[1]

	(in millions)	Total Value[2]	Upfront	Clinical milestones[3]	Regulatory/ Marketing	Sales	Royalties
Genentech A Member of the Roche Group	**Crenezumab**	**$340**	**$25**	**$40**	---	---	Net high single digits to the mid-teens
Genentech A Member of the Roche Group	**Anti-Tau antibodies**	**CHF400+**	**CHF17**	**CHF42[4]**	---	---	Mid-single digits to low double digits
janssen	**ACI-35**	**CHF500**	**CHF26**	**CHF5**	---	✓	Low double digits to mid-teens
Piramal | Life Sciences knowledge action care	**Tau-PET imaging agent**	**€157**	**€0.5**	**€1.0**	✓	✓	Mid-single digits to low teens
Nestlé Institute of Health Sciences	**Tau diagnostic assay**	Not disclosed	NA	NA	NA	NA	NA
Biogen	**α-syn-PET imaging agent**	Not disclosed	Not disclosed	NA	NA	NA	NA
ESSEX	**Therapeutic for neuroprotection**	Not disclosed	Not disclosed	NA	NA	NA	NA

(1) Disclosure limited due to confidentiality agreements with collaboration partners
(2) Figures are rounded numbers
(3) Received to date (4) including CHF 14 million milestone for first patient dosing in Phase 2 accrued in Q4 2017

Financial overview (IFRS)

Key financial data

(all figures in CHF millions, except for share and per share data)	Nine Months ended Sept 30, 2017	Nine Months ended Sept 30, 2016
Income statement		
Revenues	3.8	21.7
R&D expenses	22.5	18.7
G&A expenses	7.0	4.5
Income (loss) for the period	(30.6)	(2.3)
Adjustments[1]	5.6	1.1
Adjusted income (loss)[1]	(25.0)	1.2
EPS – basic and diluted	(0.54)	(0.05)
Adjusted EPS – basic and diluted [1]	(0.44)	(0.03)
	As of	
Balance sheet	**Sept. 30, 2017**	**Dec. 31, 2016**
Cash and cash equivalents	117.2	152.2
Total shareholder's equity	112.9	142.4

(1) Adjustments are comprised of non-cash share based compensation totalling CHF 0.8 million and 0.4 million, respectively, and foreign currency remeasurement losses totalling CHF 4.8 million and CHF 0.7 million, respectively

AC Immune diagnostics

Creation of precision medicine in neurodegenerative diseases

IVD (Tau, Abeta)
- CSF
- Blood

Tau-PET imaging agent
- Brain imaging

Piramal | Life Sciences
knowledge action care

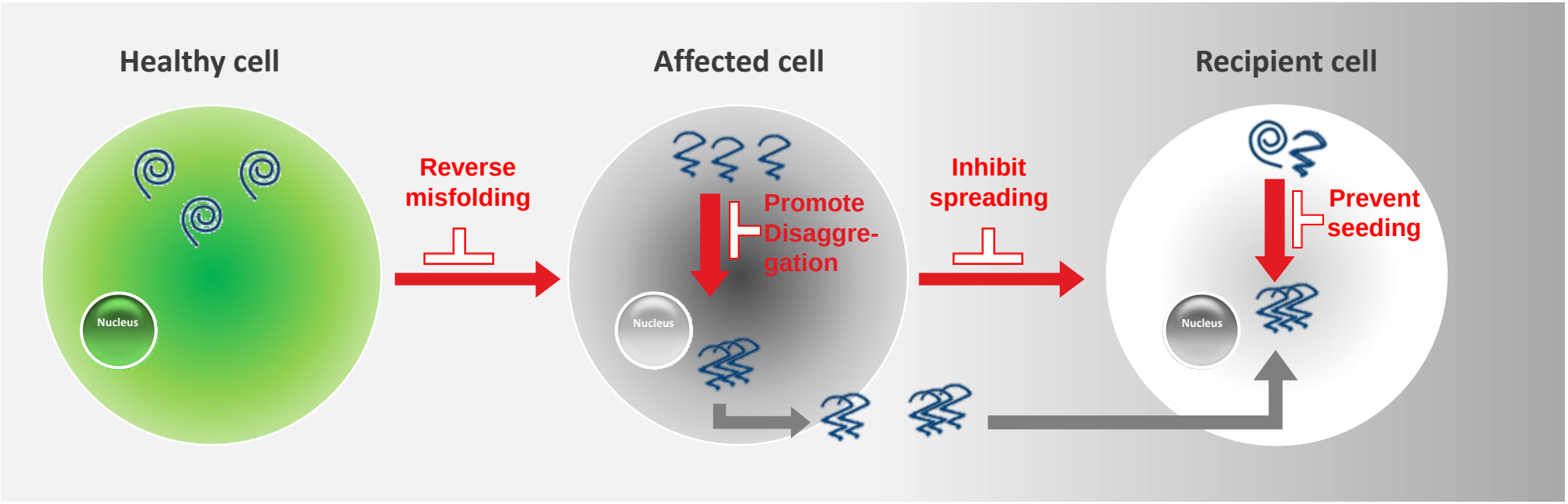
α-syn-PET + TDP-43 imaging agent
- Brain imaging

Biogen

Strategic value for AC Immune	▪ Enable early and better diagnosis of patients ▪ Improved selection of patients ▪ Early detection / diagnosis significantly increases probability of clinical success ▪ Attractive assets for partnering and revenue generation
Benefit for patients and healthcare systems	▪ Early treatment start for patients with demonstrated disease ▪ Improvement in patient safety and outcome ▪ Lowering costs of treatment

Misfolded Tau – a compelling therapeutic target in AD

AC Immune's Tau therapies intervene at key points in the disease pathway



Morphomer Tau

- ACI-35
- Anti-Tau antibody

Morphomer Tau

Tau-PET imaging agent for companion diagnosis