UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
Washington, D.C. 20549

FORM 6-K

REPORT OF FOREIGN PRIVATE ISSUER PURSUANT TO RULE 13a-16
OR 15d-16 UNDER THE SECURITIES EXCHANGE ACT OF 1934

For the month of November, 2019

Commission File Number: 001-37891

AC IMMUNE SA

(Exact name of registrant as specified in its charter)

EPFL Innovation Park
Building B
1015 Lausanne, Switzerland
(Address of principal executive office)

Indicate by check mark whether the registrant files or will file annual reports under cover of Form 20-F or Form 40-F:

Form 20-F X Form 40-F

Indicate by check mark if the registrant is submitting the Form 6-K in paper as permitted by Regulation S-T Rule 101(b)(1):

Yes No X

Indicate by check mark if the registrant is submitting the Form 6-K in paper as permitted by Regulation S-T Rule 101(b)(7):

Yes No X

AC IMMUNE SA

On November 6, 2019, representatives from AC Immune will present at its Key Opinion Leader ("KOL") meeting held in New York City, using the presentation slides attached as Exhibits 99.1 and 99.2 hereto.

This report on Form 6-K (including Exhibits 99.1 and 99.2 hereto) shall be deemed to be incorporated by reference into the registration on Form F-3 (Registration Number: 333-224694), the registration statement on Form F-3 (Registration Number: 333-227016) and the registration statement on Form S-8 (Registration Number: 333-216539) of AC Immune and to be a part thereof from the date on which this report is filed, to the extent not superseded by documents or reports subsequently filed or furnished.

SIGNATURE

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned, thereunto duly authorized.

AC IMMUNE SA

By: /s/ Andrea Pfeifer
Name: Andrea Pfeifer
Title: Chief Executive Officer

By: /s/ Joerg Hornstein
Name: Joerg Hornstein
Title: Chief Financial Officer

Date: November 6, 2019

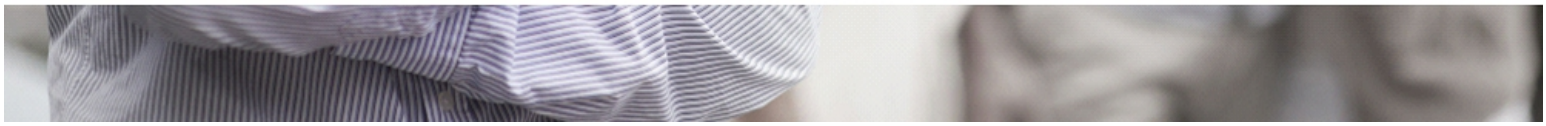
EXHIBIT INDEX

Exhibit Number	Description
99.1	Presentation dated November 6, 2019
99.2	Presentation dated November 6, 2019

Novel Therapeutics and Diagnostics for Neurodegenerative Diseases

AC Immune

Andrea Pfeifer, Ph.D., CEO AC Immune



Disclaimer

This presentation may contain statements that constitute "forward-looking statements" within the meaning of Section 27A of the Securities Act of 1933 and Section 21E of the Securities Exchange Act of 1934. Forward-looking statements are statements other than historical fact and may include statements that address future operating, financial or business performance or AC Immune's strategies or expectations. In some cases, you can identify these statements by forward-looking words such as "may," "might," "will," "should," "expects," "plans," "anticipates," "believes," "estimates," "predicts," "projects," "potential," "outlook" or "continue," and other comparable terminology. Forward-looking statements are based on management's current expectations and beliefs and involve significant risks and uncertainties that could cause actual results, developments and business decisions to differ materially from those contemplated by these statements. These risks and uncertainties include those described under the captions "Item 3. Key Information – Risk Factors" and "Item 5. Operating and Financial Review and Prospects" in AC Immune's Annual Report on Form 20-F and other filings with the Securities and Exchange Commission. Forward-looking statements speak only as of the date they are made, and AC Immune does not undertake any obligation to update them in light of new information, future developments or otherwise, except as may be required under applicable law. All forward-looking statements are qualified in their entirety by this cautionary statement.

About AC Immune

- Pioneering new ways to treat neurodegenerative diseases associated with misfolded proteins
- Listed on Nasdaq since September 2016 (ticker: ACIU)
- 71.1 million shares outstanding[1] (free float approximately 50%)
- Cash position of CHF 286 million as of Q2 2019
- Based at the EPFL campus in Lausanne, Switzerland
- 123 full-time employees

(1) As of April 25, 2019

Investment highlights

AC Immune: a leader in neurodegenerative diseases

1. Addressing 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

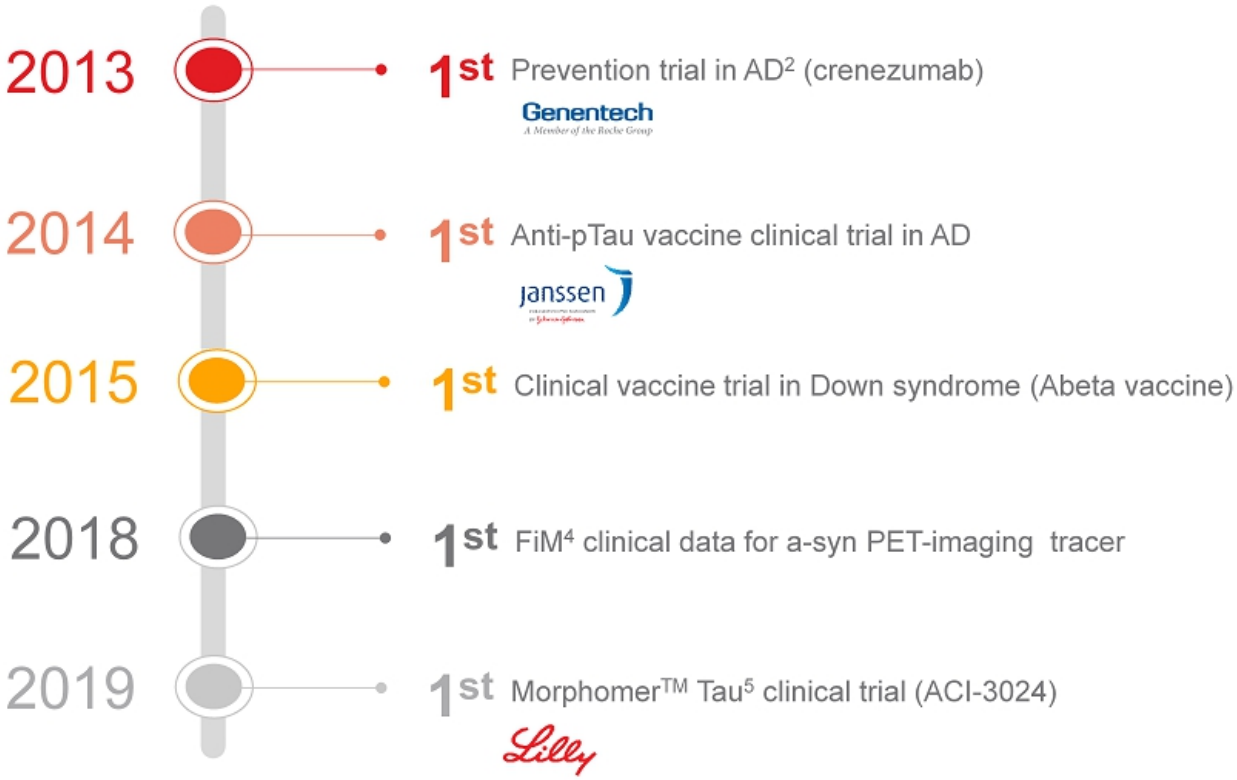
3. Broad pipeline with four therapeutic candidates in Phase 2
   - Multiple near-term value inflection points
   - Partnerships with Roche, Janssen and Eli Lilly

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

5. CHF 286 million in cash, supports operations through Q3 2023[1]
   - Increasing investment into key areas of NeuroOrphan and neuroinflammation

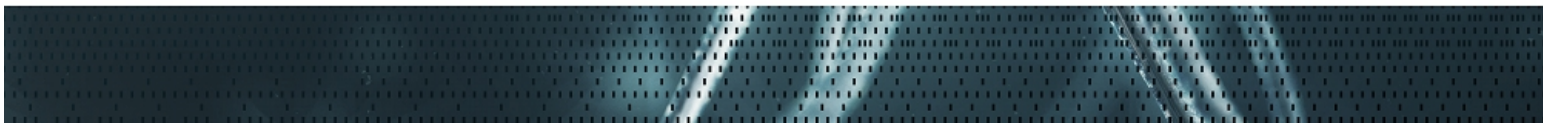
(1) As of Q2 2019. Expected cash runway, excluding potential incoming milestones.

“Firsts” reflect ACIU’s leadership in NDD[1]

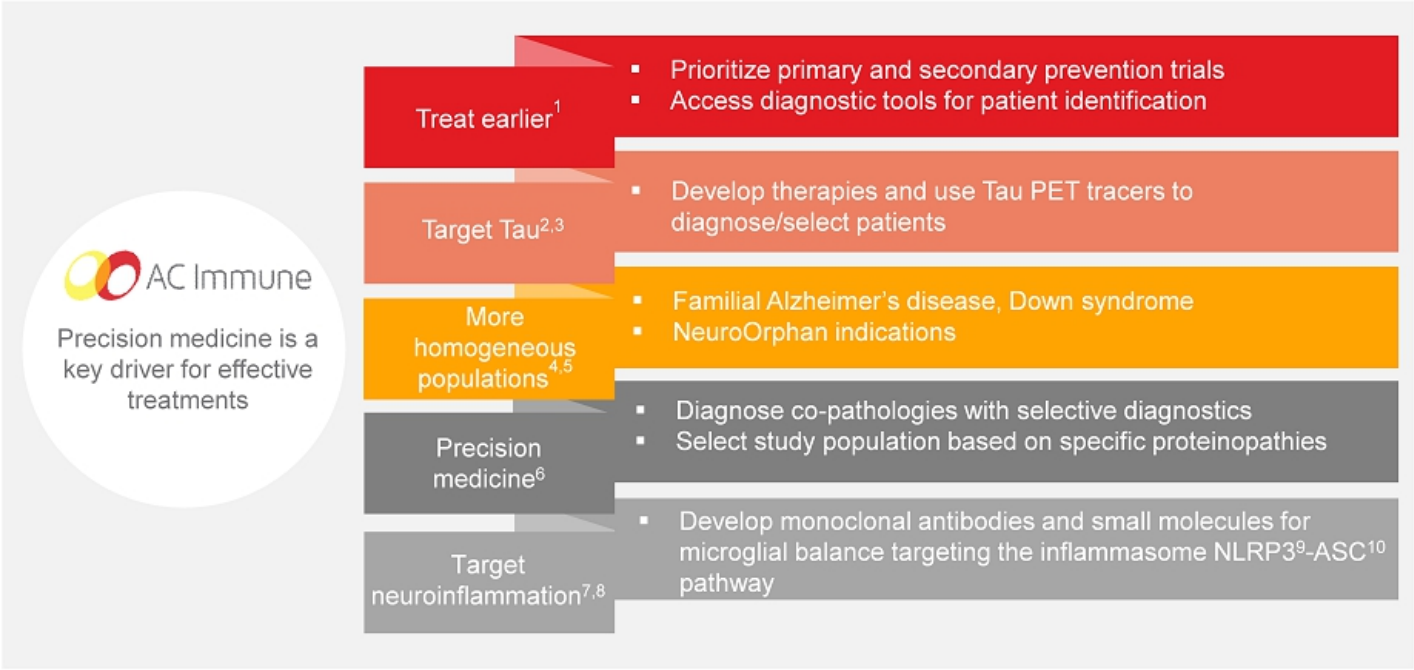


(1) Neurodegenerative diseases; (2) Alzheimer's disease; (3) Progressive supranuclear palsy; (4) First-in-Man; (5) Small molecule Tau-specific aggregation inhibitor

AC Immune's Roadmap to successful therapies for neurodegenerative diseases

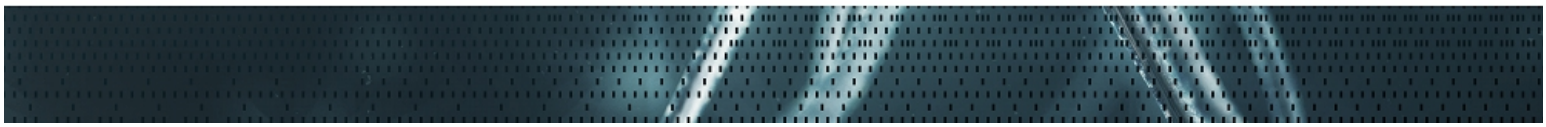


Roadmap to successful therapies for neurodegenerative diseases



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

AC Immune’s business strategy



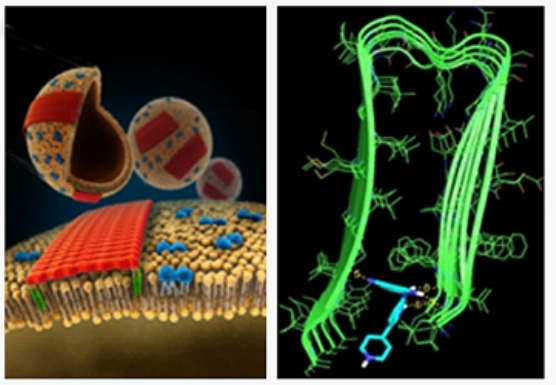
Vision

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

Dual Proprietary Technology Platforms

SupraAntigen™

Vaccines and antibodies specific to disease causing conformations



Morphomer™

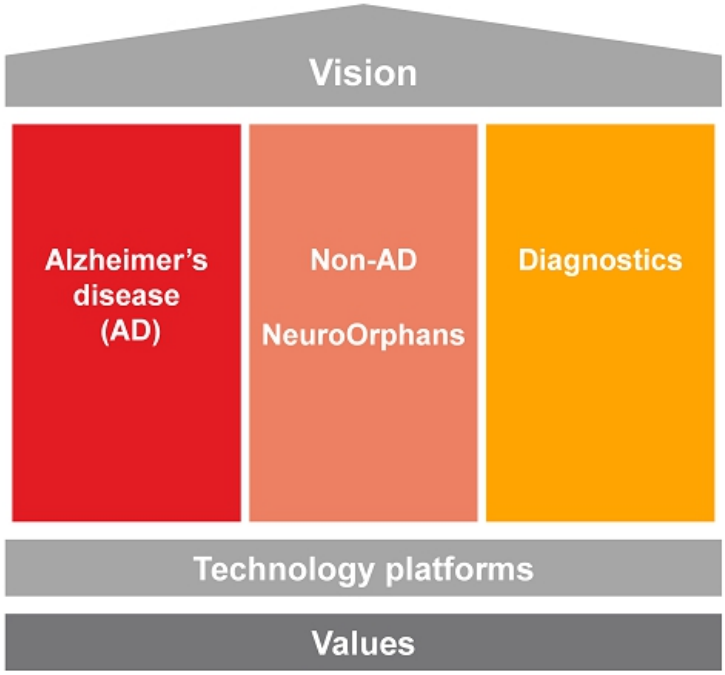
Conformation-sensitive small molecules

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

Business strategy: 3-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 NDDs[2] as mono- and 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

Investors and funds from partnerships

Highly committed institutional investors[1]

B.A:M:

BlackRock

BVF PARTNERS L.P.

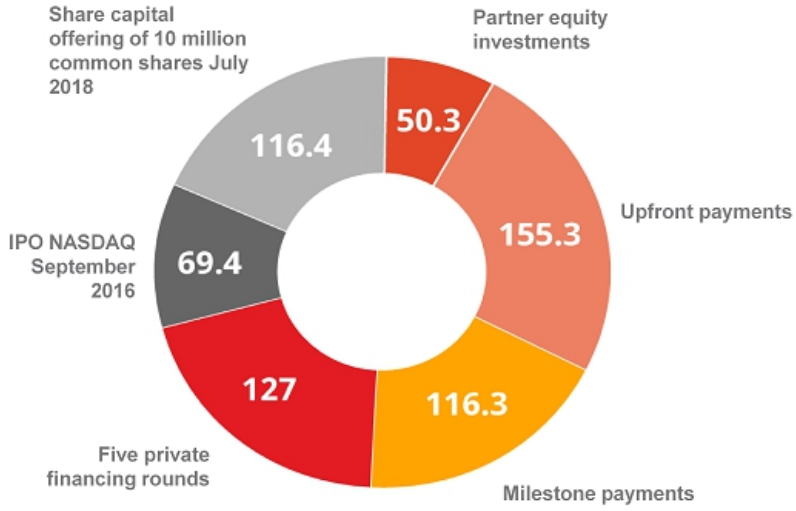
INVUS

Fidelity

TEKLA Capital Management LLC

TEMASEK

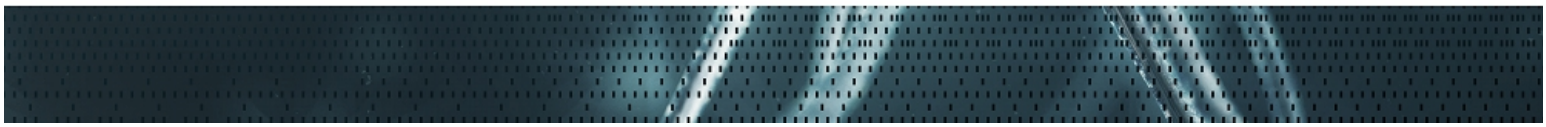
Corporate funding to date[2] (in CHF millions)



- CHF 313 million from investor funds
- CHF 322 million in partnering related funds[3,4]
- CHF 3 billion in total potential payments plus potential royalties outstanding

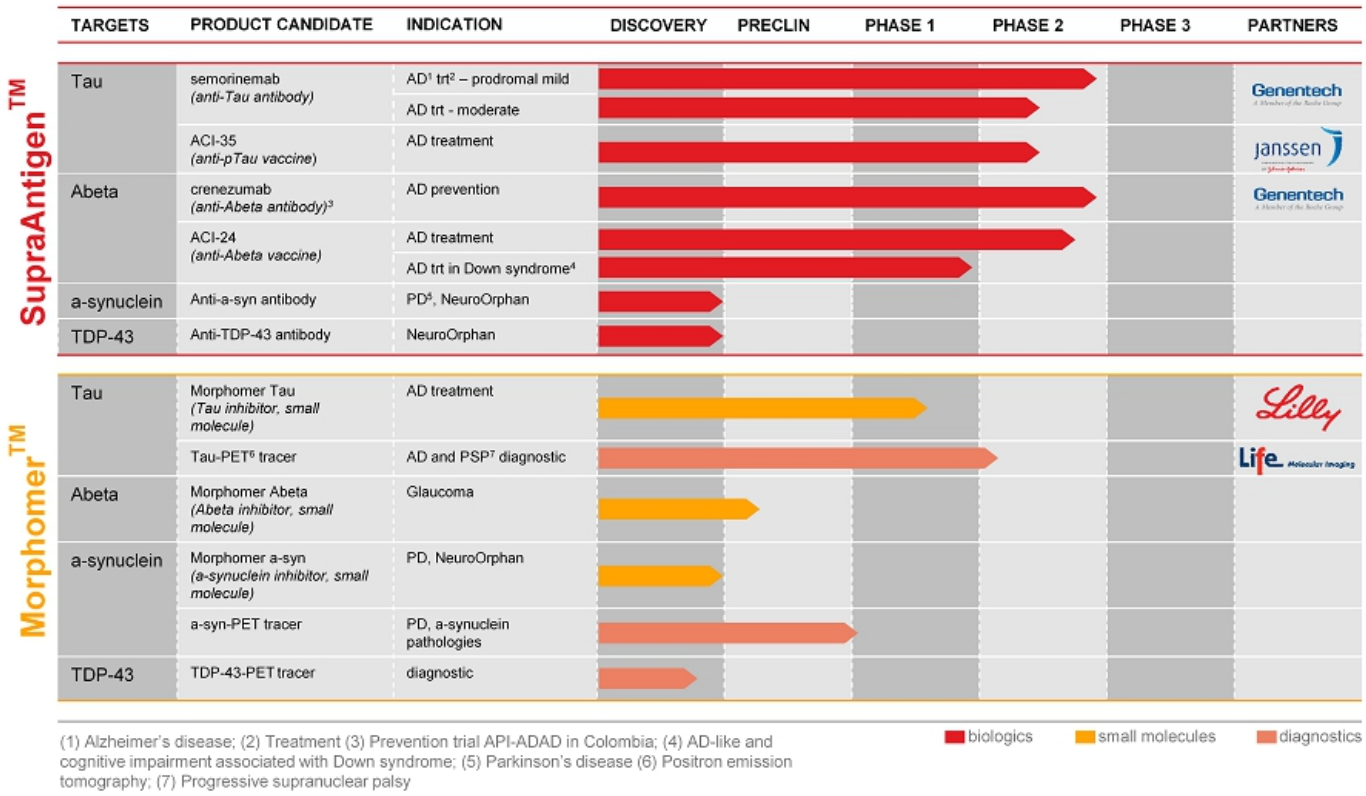
1) Based on latest schedule 13G and 13F filings; (2) Converted to CHF based on exchange rates at times of receipt; (3) Milestone payments as of 30 September, 2019; (4) With Lilly convertible loan

Pipeline and catalysts



Broad and robust pipeline in neurodegenerative diseases

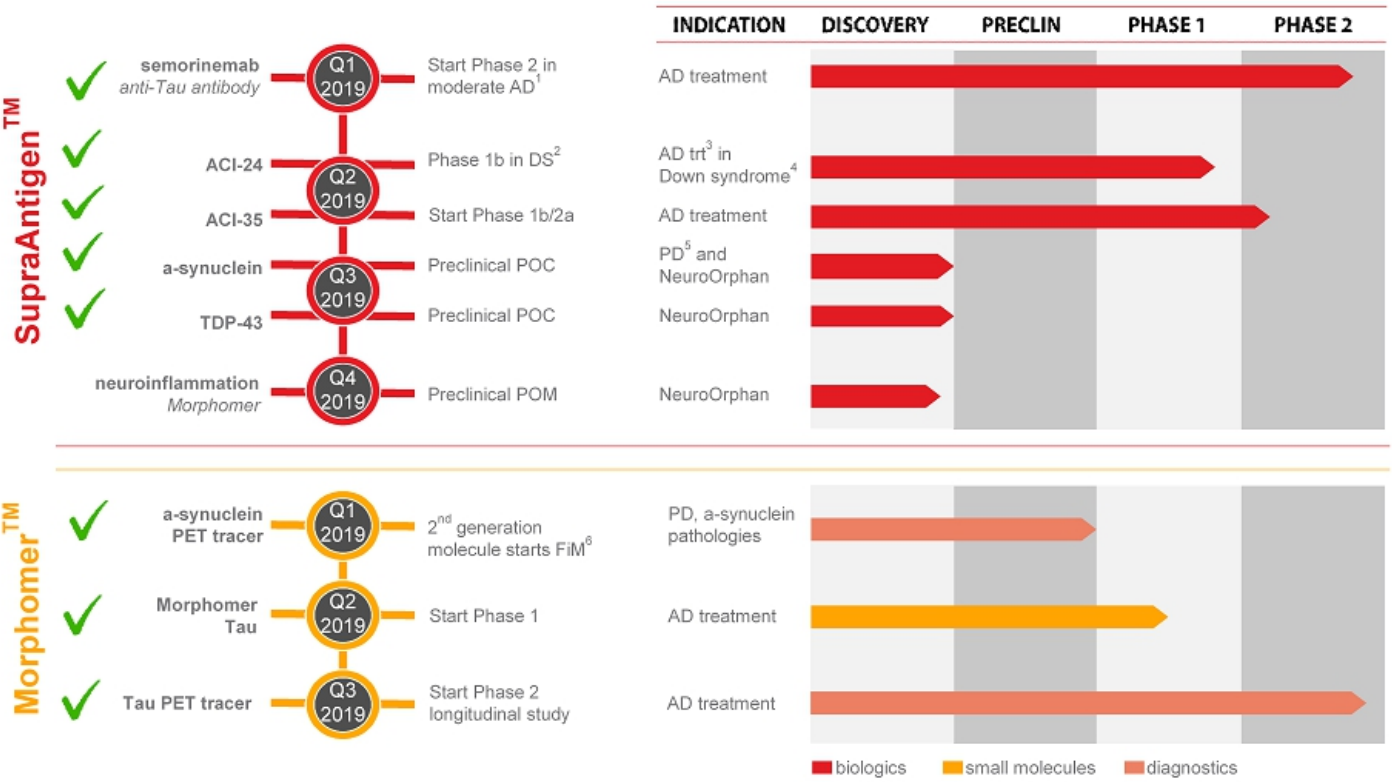
Driven by proprietary technology platforms for sustained growth



(1) Alzheimer's disease; (2) Treatment (3) Prevention trial API-ADAD in Colombia; (4) AD-like and cognitive impairment associated with Down syndrome; (5) Parkinson's disease (6) Positron emission tomography; (7) Progressive supranuclear palsy

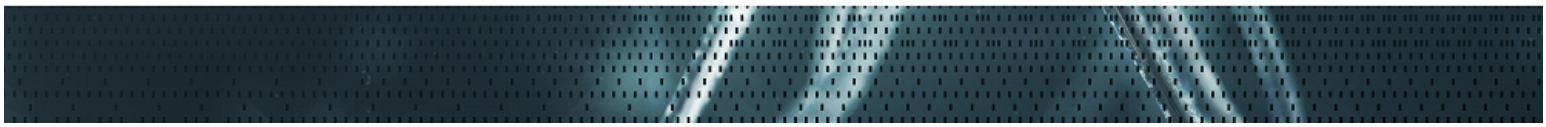
Key milestones for 2019

Successful delivery of strategy with multiple near-term catalysts

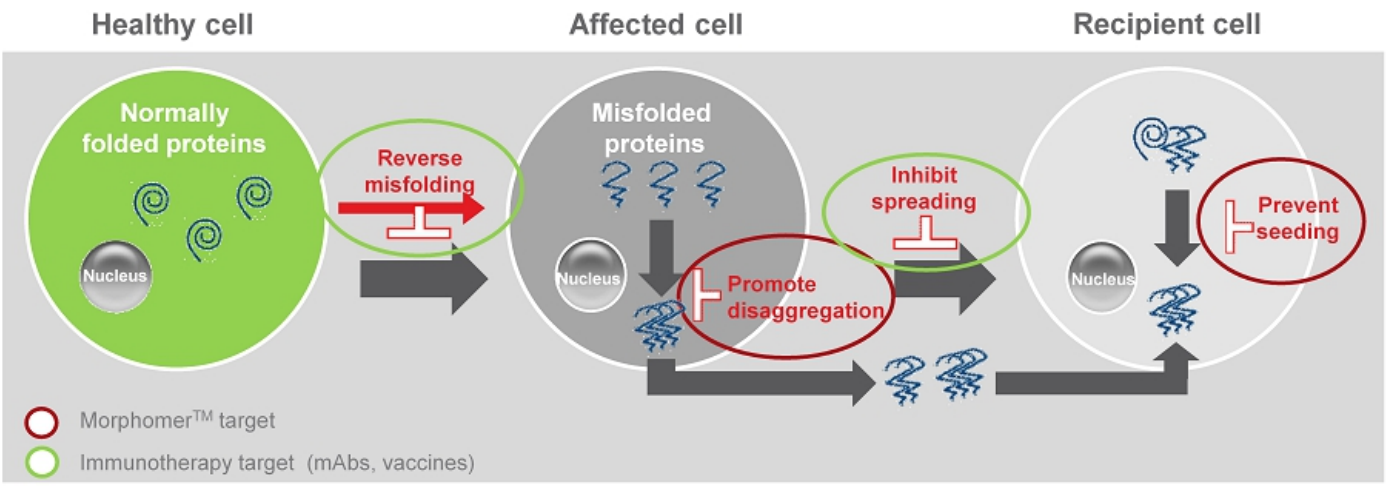


(1) Alzheimer's disease; (2) Preliminary interim data look (high dose cohort) (3) Treatment; (4) AD-like and cognitive impairment associated with Down syndrome; (5) Parkinson's disease; (6) First-in-Man

Focus on Tau



AC Immune's molecules target pathological Tau at key points in the disease pathway



- Targeting both intracellular seeds and extracellular spreading by combination therapy of Morphomers and immunotherapy enables full control of Tau pathology progression
- Highly selective Tau imaging diagnostic enables more precise patient characterization and potentially more precise prediction of AD[1] progression

(1) Alzheimer's disease

2019 Tau pipeline news

Lilly

30m
CHF milestone payment

Genentech

ALZHEIMER'S PREVENTION INITIATIVE
Tau PET substudy initiated

Life Molecular Imaging

Ph2
2nd anti-Tau monoclonal antibody trial in moderate AD

€
Milestone payment in connection with initiation of a Phase 2 trial

Ph1b/2a
clinical trial started to evaluate ACI-35.030 vaccine

janssen
PHARMACEUTICAL COMPANIES OF Johnson & Johnson

AC Immune: broadest anti-Tau pipeline with key clinical newsflow

Product candidates	Diagnostics and therapies: antibody	vaccine	small molecule	diagnostic
Status	Phase 2[1,2]	Phase 1b/2a[3]	Phase 1	Phase 2
Partner	Genentech, A Member of the Roche Group	janssen	Lilly	Life Molecular Imaging
Next milestone	**Q2 2020**[1] followed by **Q3 2021**[2]	**Q2 2020** interim analysis[4]	**Q1 2020** with interim in Q4 2019	**Q4 2020** PET tracer[5]

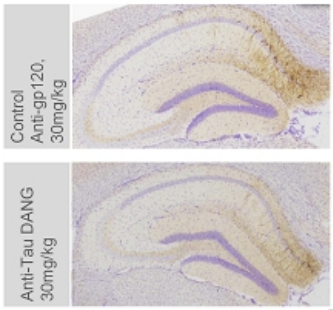
(1) Prodromal/mild trial primary completion estimated; (2) Moderate trial primary completion estimated; (3) In Alzheimer's disease; (4) Trial of anti-pTau vaccine (safety and immunogenicity); (5) Trial in progressive supranuclear palsy patients (test/ retest) results

Semorinemab – Phase 2 in AD[1]

Anti-Tau antibody

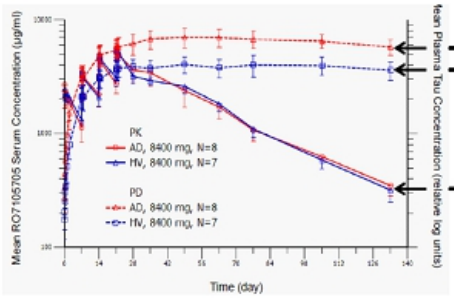
Target	Designed to intercept the cell-to-cell spread of pathological Tau in extracellular space of brain
Licensee	Genentech A Member of the Roche Group
Key differentiator	▪ Tau pathological spread is dose dependently reduced independent of effector function ▪ Proven target engagement through dose-dependent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

Dose dependent reduction of Tau pathology (preclinical)



AD/PD conference, Vienna, April 2017

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



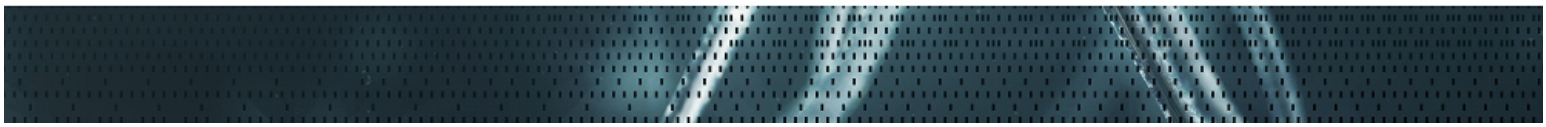
- Compared to HV, AD patients exhibited two-fold greater levels of plasma Tau following RO7105705 administration, despite identical RO7105705 exposures in the two populations
- Median half-life of 32.3 days
- No dose-limiting toxicities at high doses

Kerchner *et al*, CTAD 2017

Development status	**Phase 2 design (prodromal to mild AD; Tauriel): primary completion estimated in Q2 2020** ▪ 360 prodromal-to-mild AD patients (MMSE[3] 20-30, CDR-GS[4] = 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[5]; secondary endpoints: RBANS[6] and ADAS-cog13[7] **Phase 2 design (moderate; Lauriet): primary completion estimated in Q3 2021** ▪ 260 patients (MMSE 16-21; CDR-GS = 1 or 2) ▪ 1 dose or placebo for 49 weeks, followed by open-label study ▪ Primary endpoints: ADAS-cog11 and ADCS-ADL; secondary endpoints: CDR-SB, MMSE, safety

(1) Alzheimer's disease; (2) Healthy volunteers; (3) Mini-mental state exam; (4) Clinical Dementia Rating-Global Score; (5) Clinical Dementia Rating-Sum of the Boxes; (6) Repeatable Battery for Assessment of Neuropsychological Status; (7) Alzheimer's Disease Assessment Scale-cognitive subscale

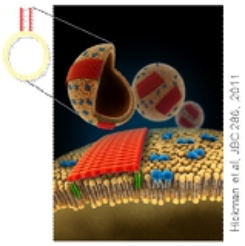
Preclinical programs with newsflow in Q3



Anti-a-synuclein (a-syn) antibody – Discovery in PD[1]

Target	Misfolded, aggregated a-synuclein (a-syn)
Target characteristics	▪ Pathological a-syn aggregates and forms oligomers and fibrils ▪ Aggregation and spreading of misfolded a-syn are linked to synucleinopathies as shown in patients and animal models
Key results	▪ Antibodies with specificity and high affinity for pathological human a-syn (KD[2] down to 3pM) ▪ Target binding shown for PD, DLB[3] and MSA[4] in human brains from multiple patients ▪ A-syn antibodies, ACI-5755 and ACI-5756, show aggregation inhibition *in vitro* ▪ *In vivo*, ACI-5755 and ACI-5756 significantly decrease pathological a-syn spreading

SupraAntigen™ platform

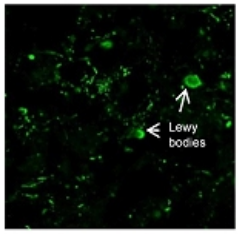


SupraAntigen platform is ideally positioned to generate antibodies with selectivity to:

- Misfolded, aggregated a-syn

Binding preference

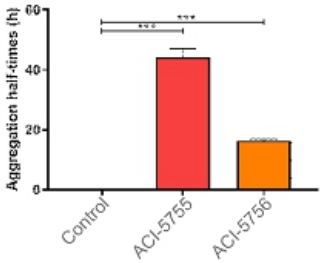
Target engagement on Lewy bodies of PD amygdala



PD patient

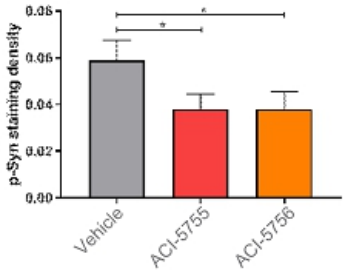
Delay of a-syn aggregation *in vitro*

Recombinant a-syn aggregation assay



Reduction of pathological a-syn *in vivo*[5]

Phosphorylated S129 a-syn (p-Syn[6]) IHC[7]



AC Immune unpublished data

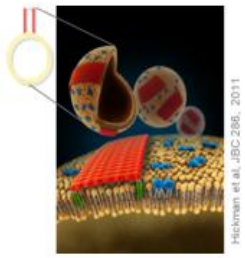
Key differentiation	▪ High affinity for pathological a-syn; significant decrease of a-syn spreading
Next steps	▪ IND[8] enabling studies start in Q1 2020

(1) Parkinson's disease; (2) Equilibrium dissociation constant; (3) Dementia with Lewy bodies; (4) Multiple system atrophy; (5) Cortex; (6) p-syn antibody (pSer129; Abcam, UK); (7) Immunohistochemistry; (8) Investigational new drug

Anti-TDP-43 antibodies – Discovery Phase

Target	Aggregated TDP-43[1]
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[3], HD[4], ALS[5] and CTE[6]
Key results	▪ ACI-5891 antibody binds to all forms of pathological human TDP-43 with high affinity ▪ *In vivo*, ACI-5891 significantly decreases aggregated TDP-43

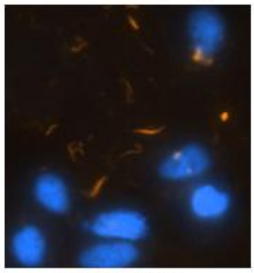
SupraAntigen™ platform



SupraAntigen platform is ideally positioned to generate antibodies with selectivity to:
- Misfolded, aggregated TDP-43

Binding preference

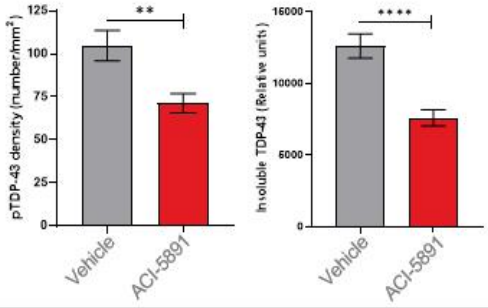
Target engagement on FTD frontal cortex (IHC[8])



FTD patient

Reduction of pathological TDP-43 *in vivo*[7]

pTDP-43 (IHC[8]) | Insoluble TDP-43 (biochemistry)

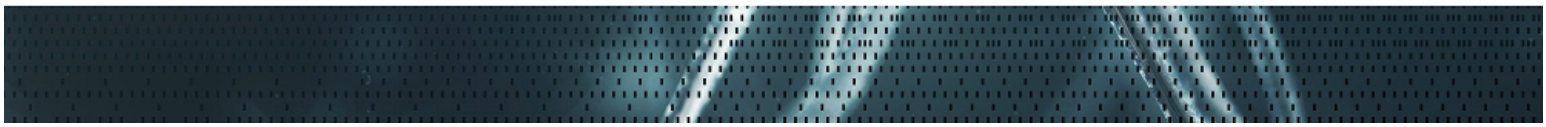


Key differentiation	▪ Only antibody reported with demonstrated *in vivo* activity
Next steps	▪ IND enabling studies start in Q2 2020

Ref: AC Immune unpublished data

(1) TAR DNA-binding protein 43; (2) Frontotemporal dementia; (3) Alzheimer's disease; (4) Huntington's disease; (5) Amyotrophic lateral sclerosis; (6) Chronic traumatic encephalopathy; (7) Walker *et al* Acta Neuropathol 2015; (8) Immunohistochemistry

Strategic outlook



Strategy for value creation

1. ENFORCE focus on early treatment and prevention trials in homogeneous patient populations

2. EVOLVE strategy to add precision medicine and combination therapy approaches based on patients' specific proteinopathies

- Leverage diagnostics portfolio to identify and track patients

3. INVEST to further build leadership in neurodegenerative diseases

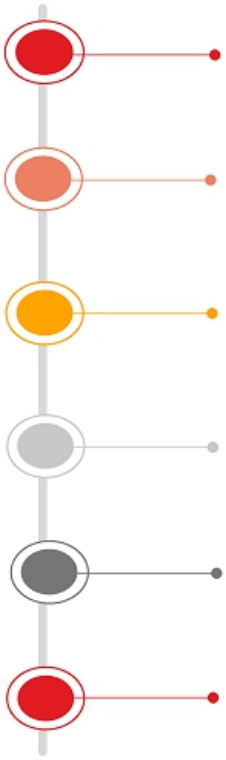
- Execute in line with our **"Roadmap"**
- Focus on Tau and new targets in neuroinflammation

4. DIVERSIFY into other neurodegenerative and NeuroOrphan diseases

- Potential for streamlined regulatory pathway
- Favorable pricing and reimbursement

5. CAPTURE maximum upside by partnering assets at optimal point in development

Milestone highlights H1 2020



Semorinemab anti-Tau antibody Phase 2 primary completion estimated in prodromal/mild AD[1]

ACI-24-DS[2] Phase 1b full study results and Phase 2 initiation

ACI-35.030 Phase 1b/2a in AD interim analysis of anti-pTau vaccine (safety and immunogenicity)

Mor-Tau Phase 1 MAD[3] readout

Mor-a-syn PET tracer FiM[4] start (3rd gen)

Identification of first **NLPR3-ASC inflammasome inhibitor** with *in vivo* activity

(1) Alzheimer's disease; (2); Down syndrome; (3) Multiple Ascending Dose; (4) First-in-Man

AC Immune

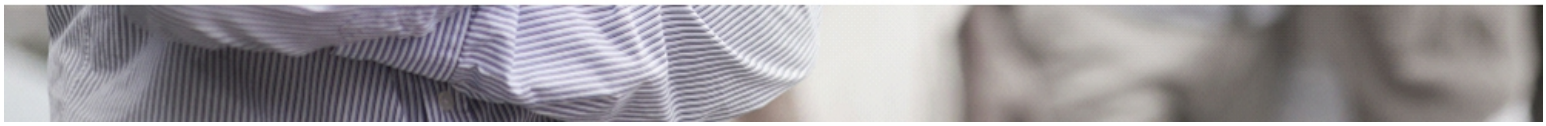
We continue to shape the future of neurodegeneration by discovering and developing breakthrough therapies through pioneering science and precision medicine

Tau Morphomer™ Therapeutics and ACI-35 Anti-pTau Therapeutic Vaccine

Marie Kosco-Vilbois, Ph.D, CSO AC Immune

AC Immune

Version Nov 4



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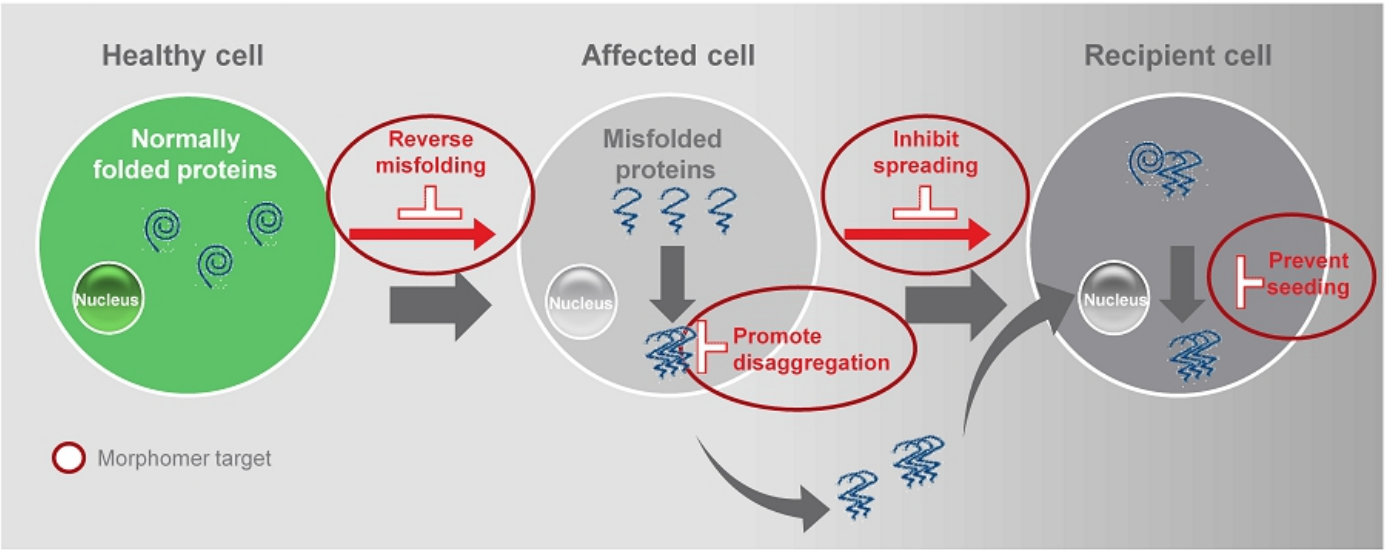
ACI-3024: Potential First in Class Small Molecule Tau Aggregation Inhibitor

AC Immune

Lilly

Morphomers™ address spreading hypothesis of misfolded Tau in neurodegenerative diseases

Multiple points of potential intervention in the disease pathway

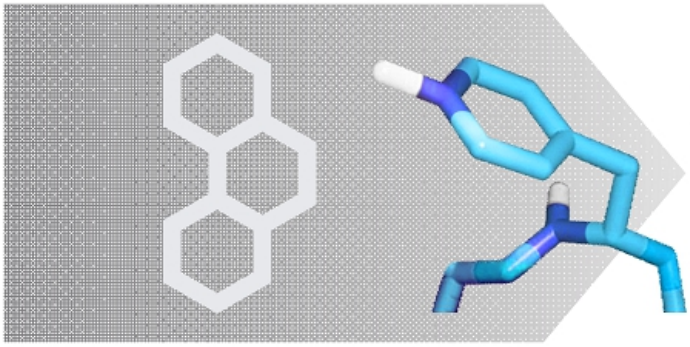


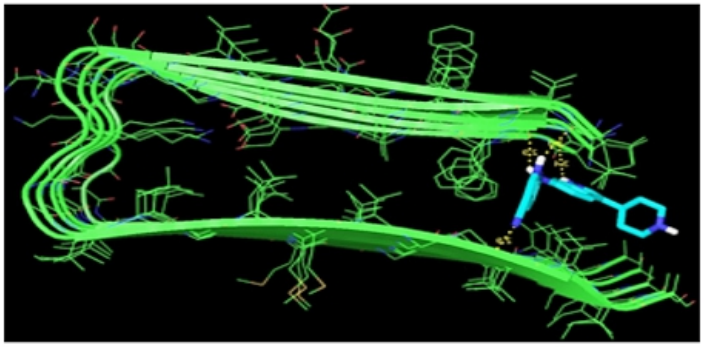
- Targeting both intracellular seeds and extracellular spreading would lead to control of Tau pathology progression
- Combined with our highly selective Tau imaging diagnostic, enables more precise patient characterization and potentially more precise prediction of AD[1] progression and successful clinical outcome

(1) Alzheimer's disease

Morphomer™ platform: discovery of ACI-3024

Generation of conformation-specific small molecules





- Conformation-specific, non-peptidic, small molecules with drug like properties
- Protein propagation inhibitors (*Kroth et al., 2012)*
- Robust library contains >7000 compounds with desirable properties including brain penetration
- Validated for selective binding to Abeta, Tau, alpha-synuclein and TDP-43
- Combination of library and medicinal chemistry program led to the discovery of ACI-3024

ACI-3024: lead characterization

Summary of *in vitro* results

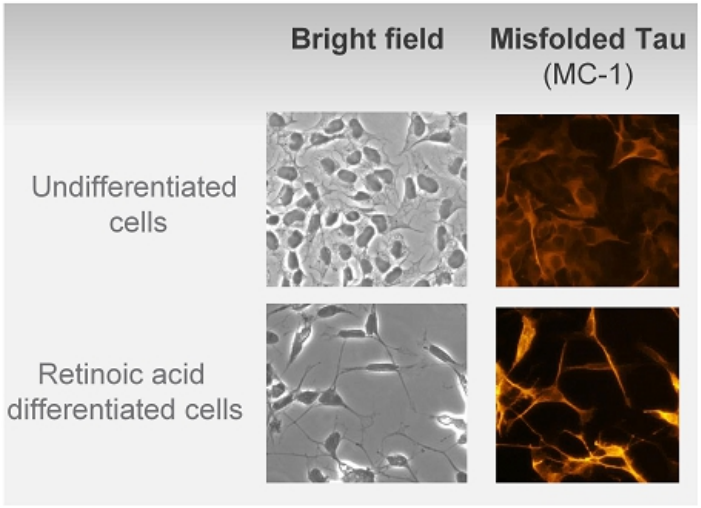
Tau aggregation inhibition	▪ Potent reduction of Tau aggregation ▪ Effect independent of Tau and FTDP-17[1] isoform mutants
Target engagement	▪ Selective binding to aggregated Tau (25.1 nM) ▪ No binding to monomeric forms of Tau ▪ Selective binding to AD[2] brain-derived pathological Tau (Ki 11.7nM)
Cross-reactivity to Abeta and alpha-synuclein	▪ No binding to human brain-derived Abeta ▪ No binding to human brain-derived alpha-synuclein ▪ No binding to healthy brain tissue

(1) Frontotemporal dementia with parkinsonism-17; (2) Alzheimer's disease

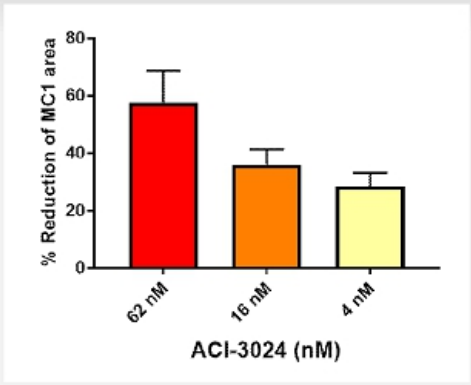
ACI-3024: *in vitro* pharmacology

Dose-dependent reduction of intracellular pathological Tau

Intracellular Tau misfolding in *in vitro* differentiated neuroblastoma cells expressing Tau P301L



Dose-dependent reduction of misfolded Tau



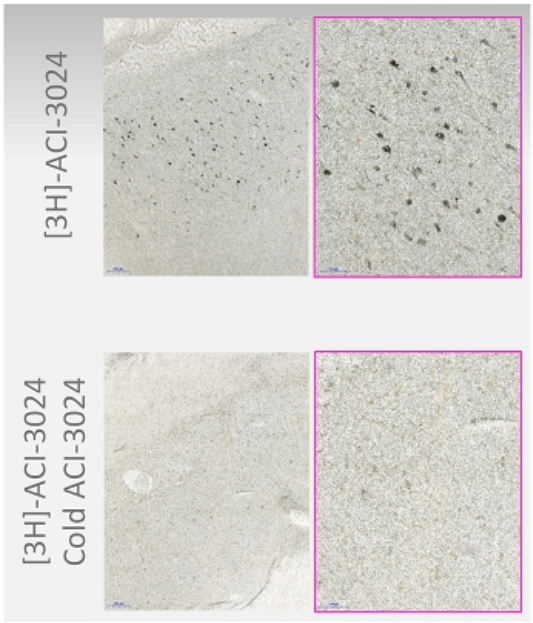
Mean + SEM

Ref.: Poli S, et al., CTAD 2018

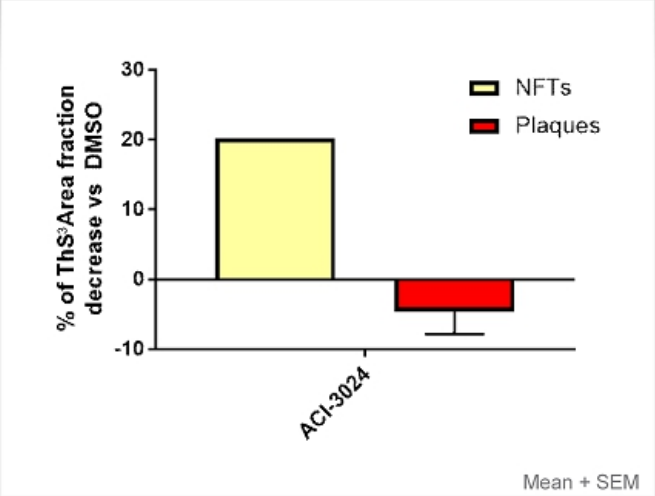
- *In vitro* treatment with ACI-3024 led to a dose-dependent decrease of misfolded Tau at low nM concentrations

ACI-3024: target engagement and functional selectivity

High resolution autoradiography on human AD[1] brain sections



***Ex vivo* disaggregation on Tau NFT[2] on human AD brain sections**



Ref.: Poli S, et al., ADPD 2019

- ACI-3024 specifically binds Tau NFTs and is able to disaggregate Tau NFTs from human AD brain sections even in presence of amyloid plaques

(1) Alzheimer's disease; (2) Neurofibrillary tangles; (3) Thioflavin S

ACI-3024: *in vivo* evaluation in rTg4510 mice

Treatment study in aged transgenic mice

Tauopathy model	▪ rTg4510 Tauopathy mouse model expressing repressible (Tet promotor Tau on/off) human 4R0N Tau carrying the P301L mutation (SantaCruz, 2005)

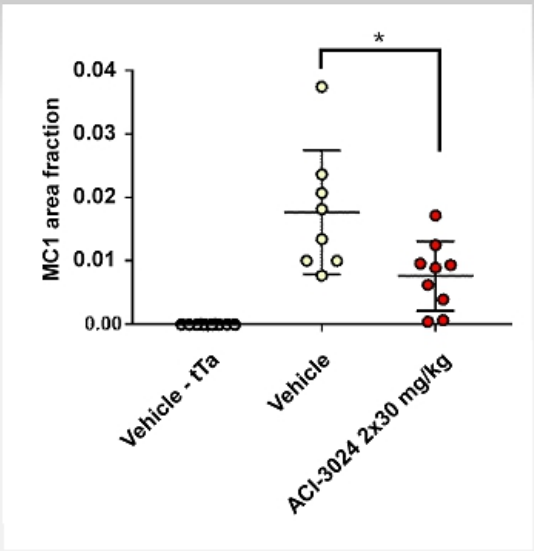
Protocol design	▪ Independent experiments with different sources of rTg4510 mouse colony
	  ▪ 30mg/kg bi-daily (BID) dose randomized to vehicle starting at 5 months of age
	  ▪ 10, 30 or 100 mg/kg BID dose randomized to vehicle starting at 5 months of age

Treatment results	▪ Significant reduction in misfolded, hyper-phosphorylated and aggregated Tau at 30mg/kg; Proof of mechanism confirmed at 100 mg/kg BID in a follow up dose-response experiment

ACI-3024: decrease in misfolded Tau

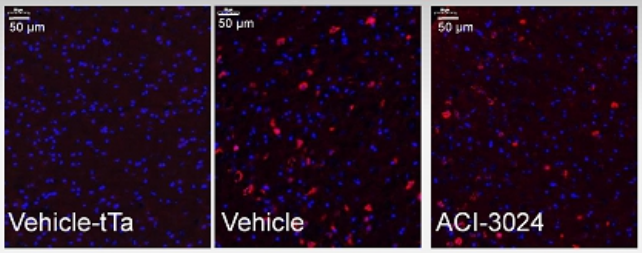
Immunohistochemistry: analysis of misfolded Tau (MC1) in rTg4510 brain section

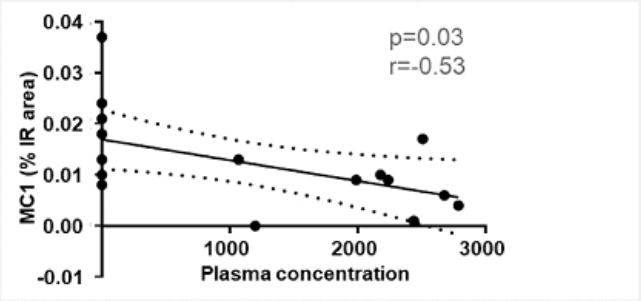
Misfolded Tau levels in brain sections



Mean + SEM
ANOVA
* p< 0.05

Representative staining (MC1 in red, DAPI in blue)





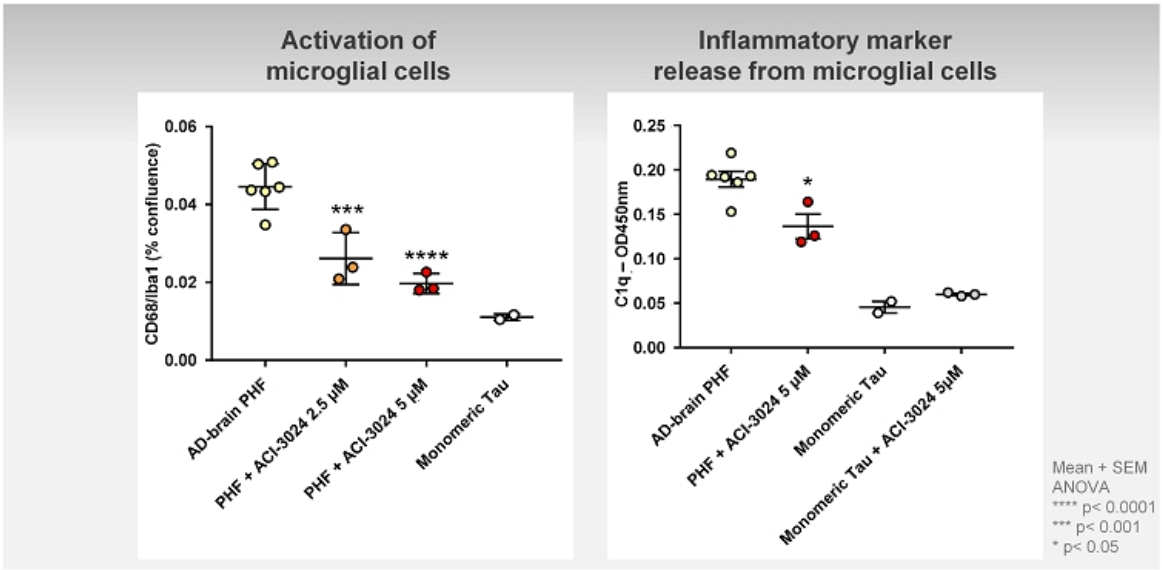
Ref.: Poli S, et al., CTAD 2018

- Treatment with ACI-3024 significantly reduced misfolded Tau
- The decrease was proportional to the plasma exposure to ACI-3024

ACI-3024: effect on neuroinflammation

Assessment of compound effect on Tau-induced microglial activation

Human AD-brain derived Tau activation of rat primary microglial cells



Ref.: Poli S, *et al.*, CTAD 2018

- Treatment with ACI-3024 significantly decreases microglial activation induced by human-derived Tau aggregates

ACI-3024: summary of preclinical evaluation

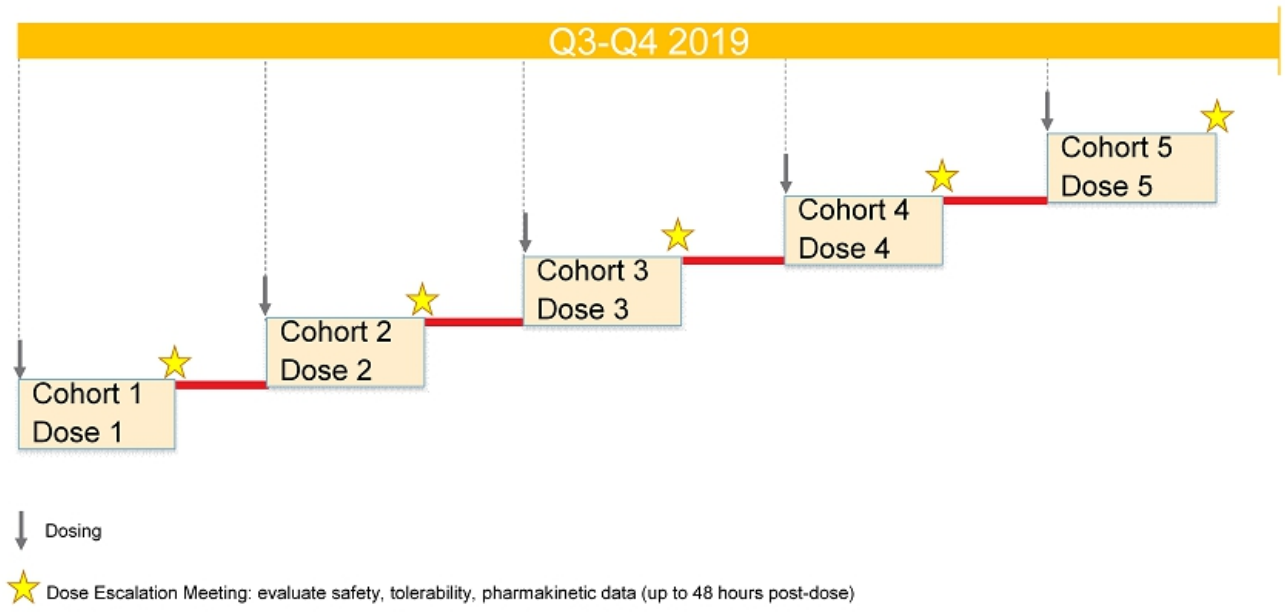
Nonclinical safety package for regulatory submission for first in human study

***In vitro* on- and off-target activity**	• Active and selective in multiple *in vitro* pharmacology assays • Good selectivity as assessed on 138 targets (Cerep Bioprint profile)
***In vivo* studies**	• *In vivo* study showed compound related treatment effects by biochemistry and immunohistochemistry (brain, CSF[1] and microglia)
ADME[2]	• Good *in vitro* and *in vivo* ADME properties, including low clearance, long half-life and good CNS[3] disposition as assessed by brain and CSF concentrations
***In vitro* tox and drug-drug interaction**	• Low potential for drug-drug interaction *in vitro* (EC_{50} on CYP > 25uM) • No P-glycoprotein[4] interaction • Negative in *in vitro* genotoxicity assays (AMES[5] and MNT[6]) and in the *in vivo* MLY[7]
Toxicology in rodents and non rodents	• In 4-week oral repeated-dose toxicity studies, NOAEL[8] established at 300 mg/kg in rodents and 450 mg/kg in non-rodents
Safety pharmacology	• ICH S7 safety pharmacology battery successfully completed: cardiovascular telemetry study in non rodent; respiratory and Irwin study in rodents
Clinical trial	• First-in-Human study initiated Q3 2019

(1) Cerebral spinal fluid; (2) Absorption, distribution, metabolism, and elimination; (3) Central nervous system; (4) Permeability-glycoprotein; (5) Bacterial mutagenesis and carcinogenesis test; (6) Micronucleus test in human cell lines; (7) *In vivo* mouse lymphoma; (8) No observed adverse event level

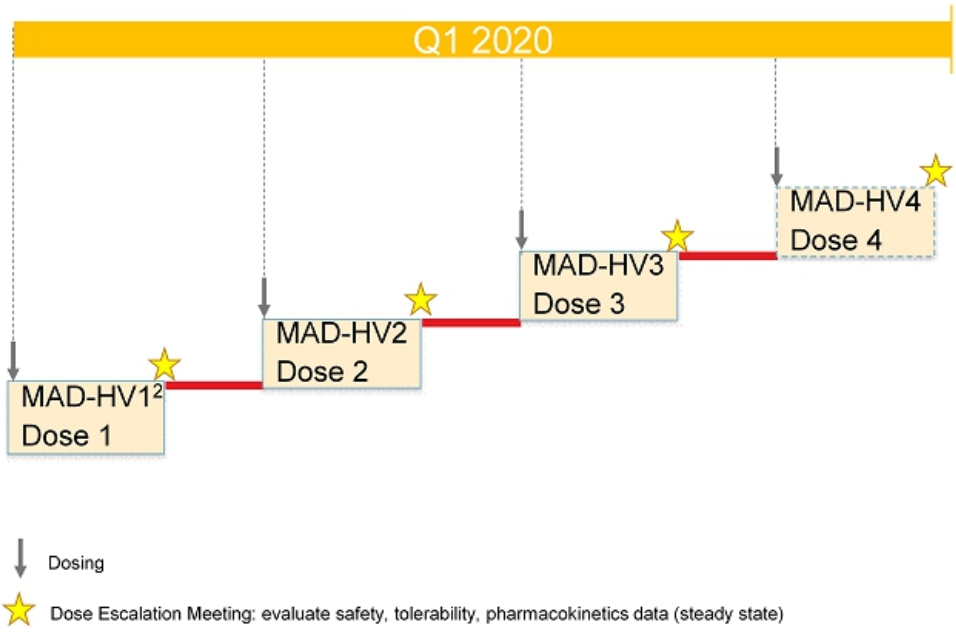
ACI-3024: First in Human study

Escalation scheme SAD[1] (ongoing)



(1) Single ascending dose

Escalation scheme MAD[1] (next)



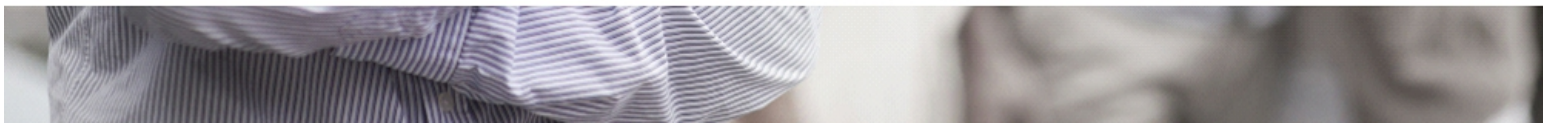
(1) Multiple ascending dose; (2) Healthy volunteer

ACI-3024: selective Tau aggregation inhibitor

Summary

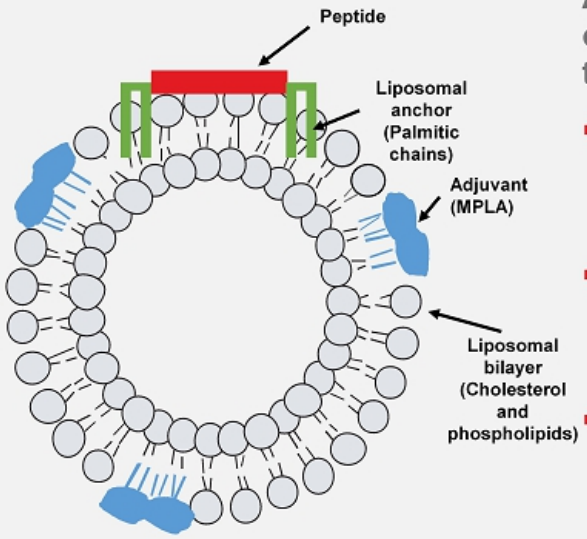
1	▪ The Morphomer™ platform has enabled identification of a new class of low molecular weight compounds, which specifically target misfolded and aggregated Tau
2	▪ Through a thorough medicinal chemistry program, ACI-3024 was identified as lead candidate with optimal drug like properties suitable for clinical development
3	▪ ACI-3024 has shown effects on Tau pathology and downstream neuroinflammation and neurodegeneration in an aggressive animal model of Tau pathology
4	▪ ACI-3024 has shown excellent preclinical safety and tolerability profile and has entered clinical development as a first in class, Tau-specific disease-modifying small molecular weight compound for the treatment of neurodegenerative diseases characterized by misfolded Tau

ACI-35 1st and 2nd Generation Anti-pTau Vaccines



1st generation vaccine: ACI-35 structure

Anti-pTau therapeutic vaccine



ACI-35 is an active immunotherapeutic based on AC Immune's proprietary SupraAntigen™ technology

- Peptide: Epitope Binding
  - Human phospho-Tau synthetic peptide T3 as antigen (Tau 393-408 (pS396/pS404))
- Linker: Conformation
  - Palmitic chains anchor peptides to generate desired conformation
- Adjuvant: Immunogenicity
  - Synthetic Monophosphoryl Lipid A (MPLA)

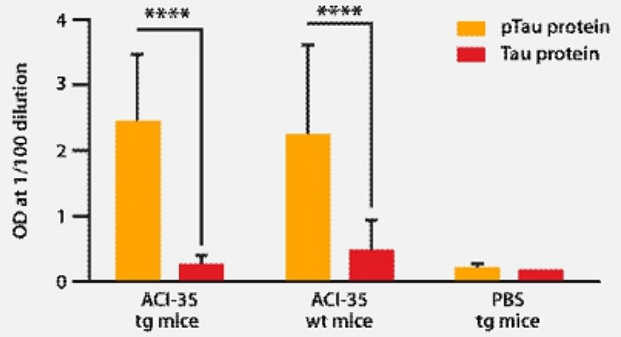
- Aim: to generate a T-cell independent anti-Tau antibody response

ACI-35: proof-of-concept

Key preclinical results

Immune response specific to phosphorylated Tau (pTau)

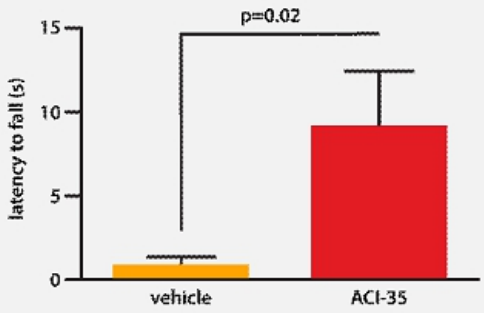
ACI-35 vaccinated mice – anti-pTau vs.Tau response after 5 immunizations



**** p<0.0001; ANOVA

Improvement of behavior (hTauP301S mice)

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

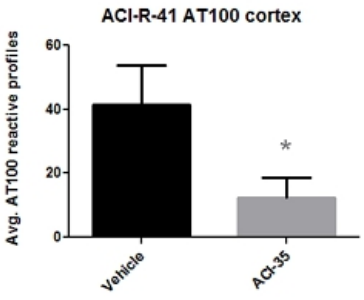


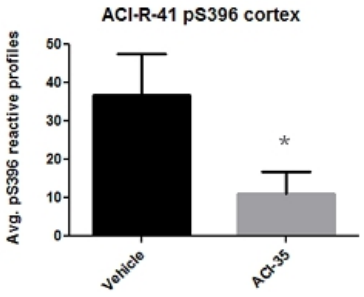
- Specific antibody response to phosphorylated Tau; improvement of behavior observed

(1) tg, transgenic; (2) wt, wild type; (3) PBS, phosphate-buffered saline

ACI-35: proof-of-concept

Key preclinical results

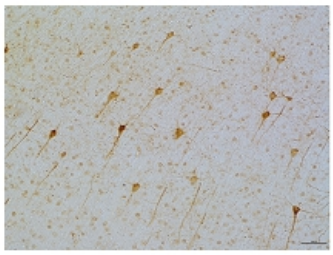


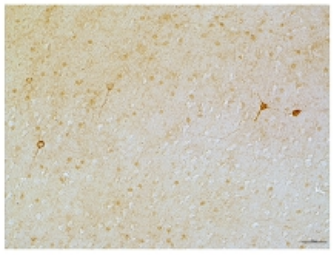


* p<0.05

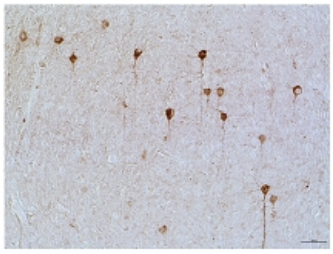
PBS

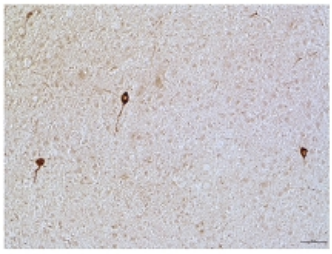
ACI-35





AT-100





Anti-pS396

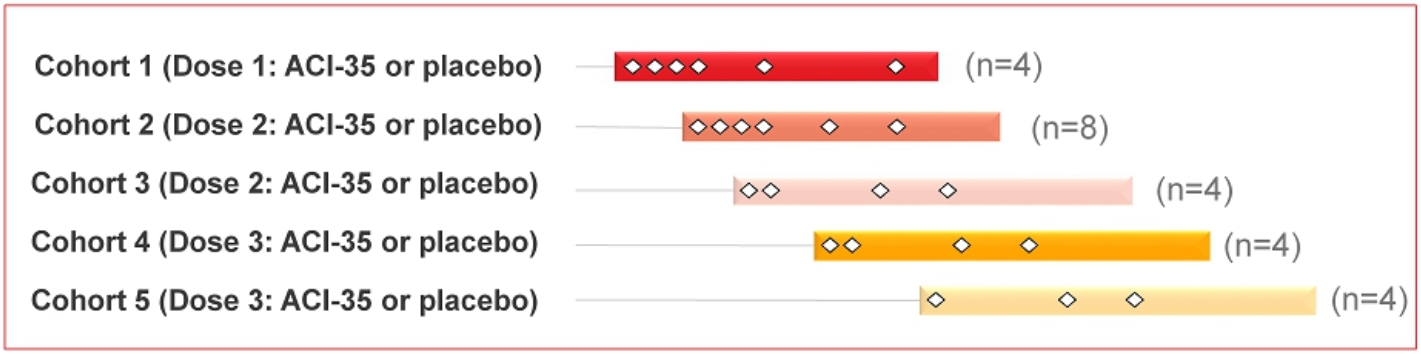
AC Immune, unpublished data

- Specific antibody response to pathogenic Tau; Decrease of pT212/pS214 and pS396 Tau in the cortex

ACI-35 Phase 1b study - overview of clinical data

ACI-35 Phase 1 study in AD[1]

Clinical study design



- Ratio of 3:1 active (ACI-35) versus placebo per cohort
- Primary readouts: Safety, tolerability and induction of IgG titers against pTau in serum

(1) Alzheimer's disease

Immunogenicity results

Responder analysis (MSD[1] assay in serum)

- Essentially all actively treated patients were anti-pTau responders
- No anti-pTau response observed with placebo
- Early target-specific antibody response against pTau observed after the first injection in the vast majority of patients

(1) Meso Scale Discovery technology

Safety results

- Injection site reactions and fatigue were the most common study adverse events
- Except for injection site reactions, no pattern of adverse events (AE) compared to placebo suggested a relationship to study medication
- Injection site reactions were generally mild and self-limiting, and more frequent at higher doses

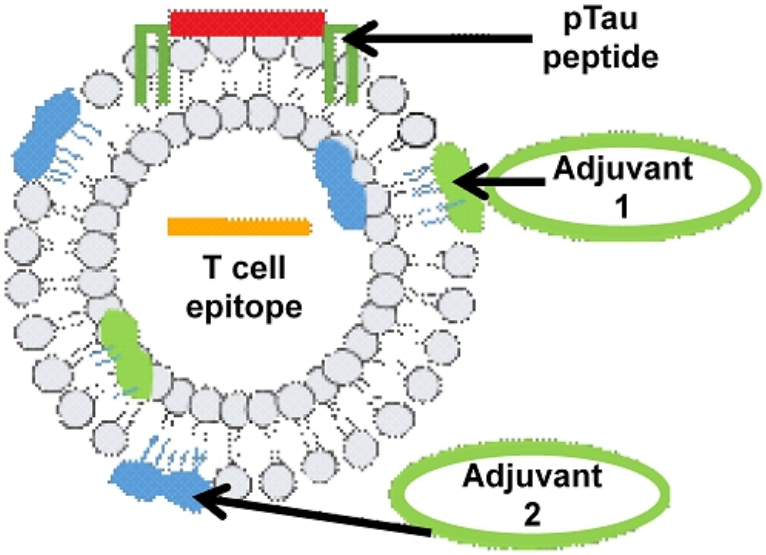
ACI-35: Phase 1b study in AD patients

Key clinical observations

- Safe at all doses tested
- Early target-specific antibody response against pTau after the first injection in the vast majority of patients

2nd generation vaccine: ACI-35.030 structure

Anti-pTau optimized therapeutic vaccine

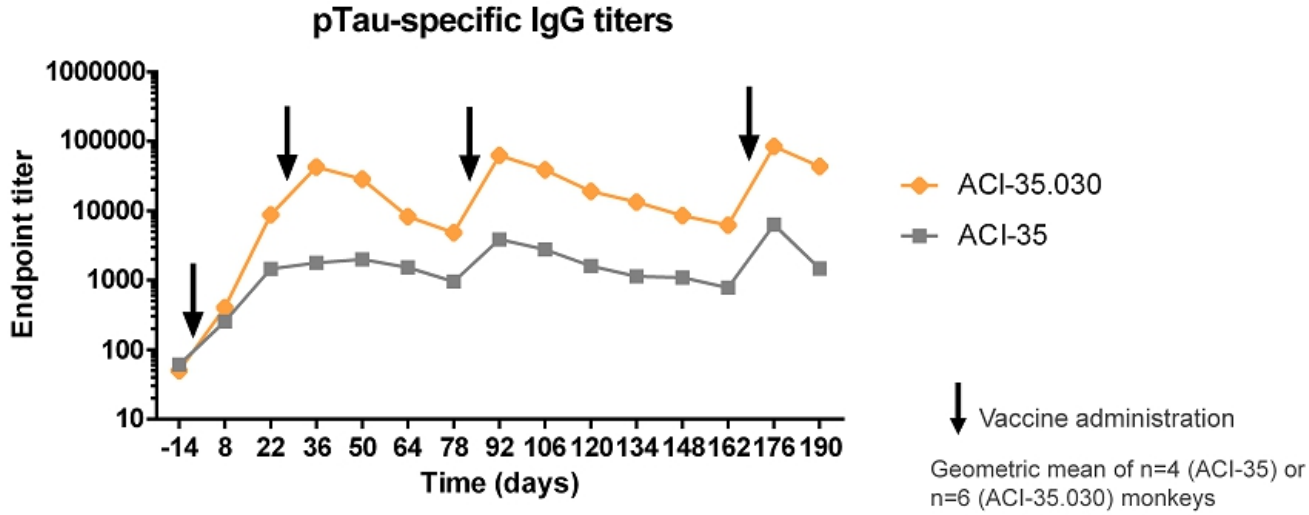


- Aim: An optimized liposomal vaccine comprising a tau peptide and a T cell epitope capable of binding to HLA-DR[1] molecules

(1) Human leukocyte antigen-major histocompatibility complex, class II

ACI-35.030: rationale

pTau-specific IgG titers in immunized rhesus monkeys

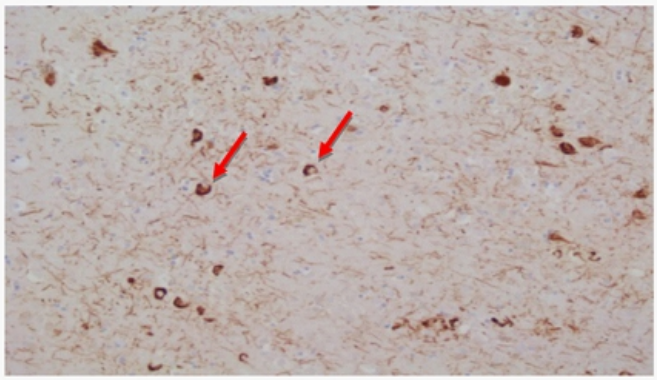


- Increased level of pTau-specific IgG titers induced by ACI-35.030 as compared to ACI-35

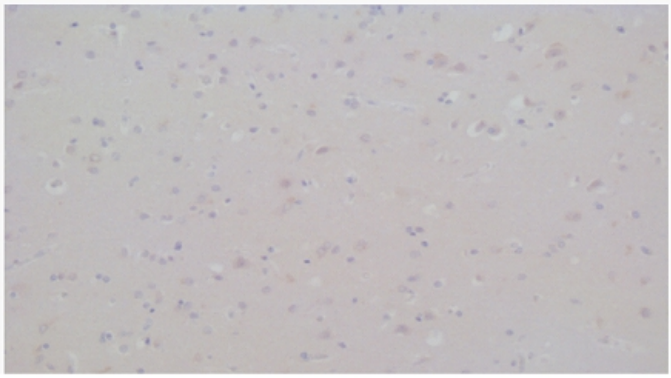
ACI-35.030: results from rhesus immunization

Binding to Tau from human AD[1] brain

AD Braak Stage V/VI



Healthy brain tissue



- Sera from rhesus monkeys immunized with ACI-35.030 binds to pathological Tau in brain sections with AD as compared to healthy human brain tissue

(1) Alzheimer's disease

ACI-35.030 non-clinical studies

Key learnings

- Demonstrates an excellent nonclinical safety profile
- Produces an enhanced homogeneous antibody response with boosting effect
- No Tau-specific T-cell response observed

ACI-35.030: Phase 1b/2a study

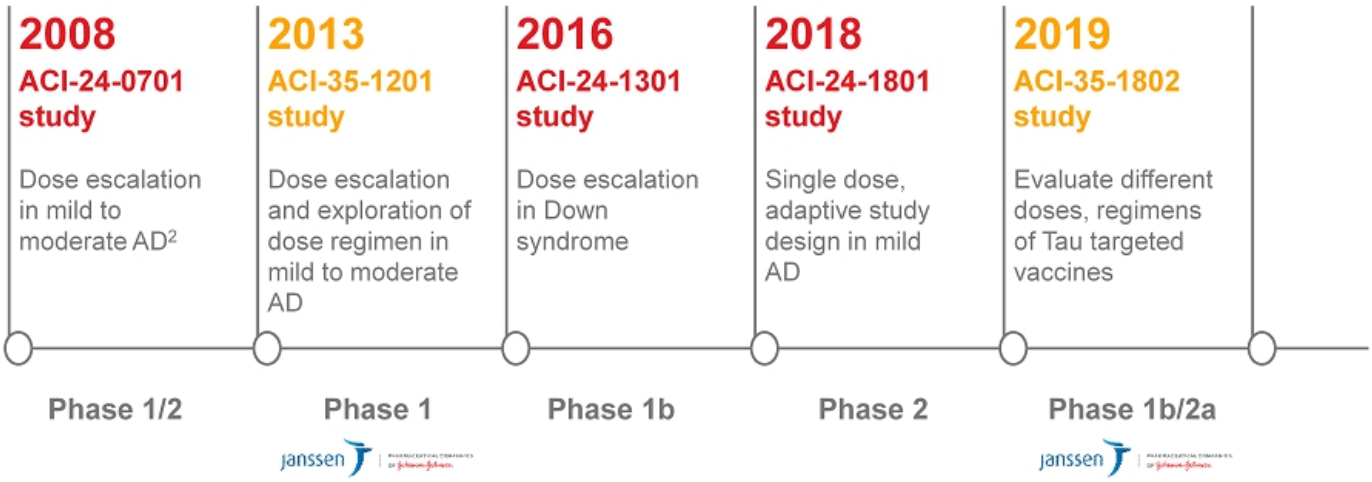
ACI-35.030 Phase 1b/2a study in AD[1]

- A randomized, multicenter, double-blind, placebo-controlled clinical study
- Ratio of 3:1 active (ACI-35.030) versus placebo per cohort
- Primary readouts: safety, tolerability and immunogenicity of different doses of ACI-35.030 in treated AD patients
- First patient in: Q3 2019

(1) Alzheimer's disease

AC Immune: pioneers in anti-Abeta[1] and anti-Tau vaccines

ACI-35.030 clinical trial: first patient dosed Q3 2019



(1) ACI-24; (2) Alzheimer's disease