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 October, 2018

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 October 27, 2018, representatives from AC Immune will be presenting at the CTAD-Alzheimer conference in Barcelona, Spain, using the presentation slides attached hereto.

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: October 26, 2018

EXHIBIT INDEX

Exhibit Number	Description
99.1	Presentation dated October 27, 2018

Targeting neurodegenerative diseases with novel therapeutics and diagnostics

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.

IDENTIFICATION AND CHARACTERISATION OF SMALL MOLECULE CLINICAL CANDIDATES TARGETING INTRACELLULAR TAU PATHOLOGY

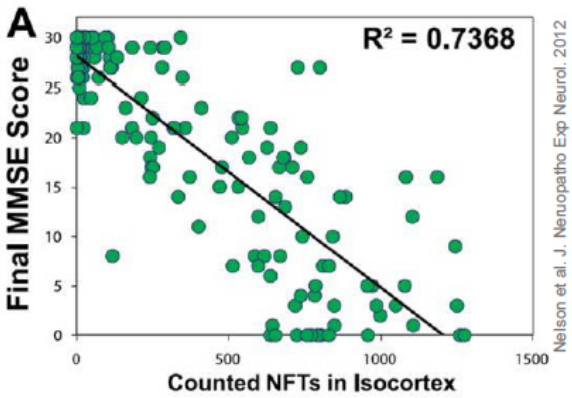
Dr. S. Poli
Head of Translational Science

October 27th , 2018

Rationale for targeting Tau

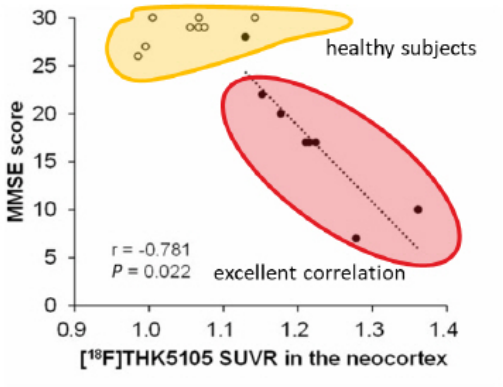
Aggregated Tau is inversely correlated with reduced MMSE score

AD patients brain histology:
Inverse correlation of NFT with MMSE score



Nelson et al. J. Neuropatho Exp Neurol. 2012

AD patients brain Pet Imaging:
Excellent inverse correlation of Pet signal and MME score

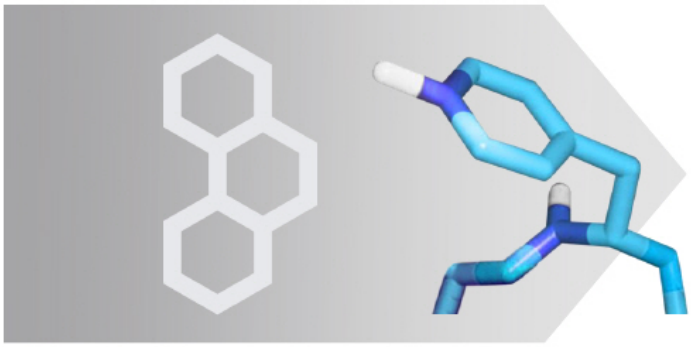


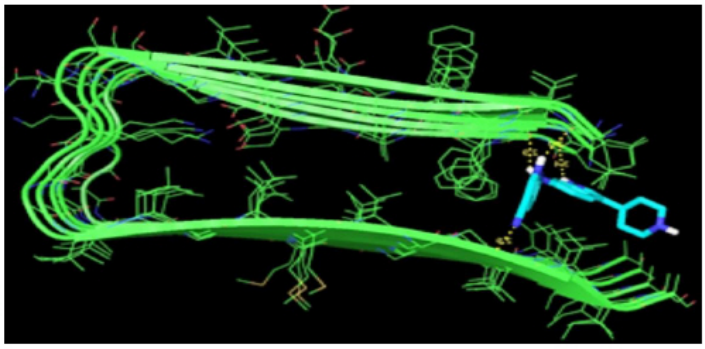
Ref: Okamura et al, Brain 2014

- Tau pathology correlates well with disease severity

Morphomer platform: Discovery of ACI-3024

Generation of conformation–specific small molecules

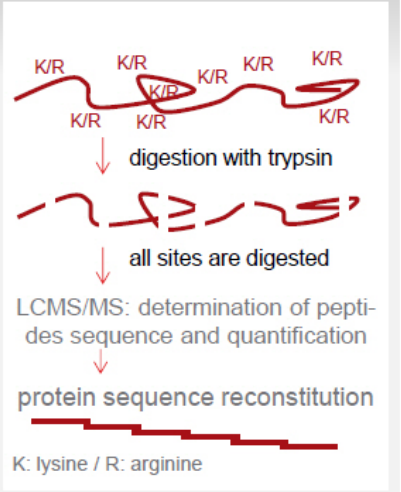




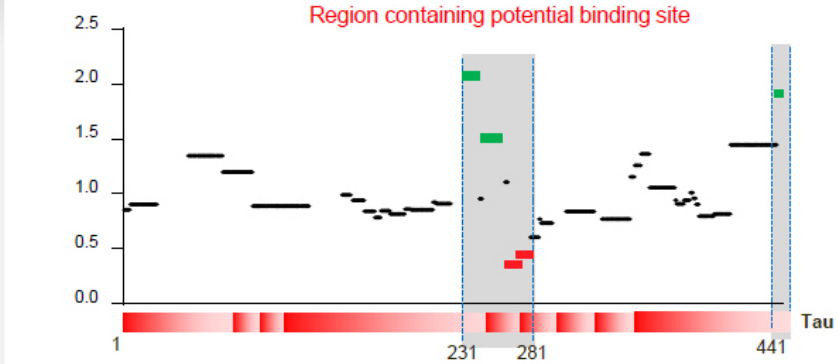
- Conformation-specific, non-peptidic, small molecules with drug like properties
- Protein propagation inhibitors (*Kroth et al., 2012)*
- Validated for selective binding to Abeta, Tau and alpha-Synuclein through *in vitro* efficacy
- Robust library of around 3000 compounds with desirable properties including brain penetration
- Around 1000 compounds screened so far for the Tau SME program
- Combination of library and medicinal chemistry program led to the discovery of ACI-3024

Mechanism of action of Morphomers
Change of conformation of aggregated Tau

Limited proteolysis in absence (A) or presence (B) of Morphomer



Relative abundance of peptides generated by limited digestion of B/A



More **protected** regions have B/A < 0.5
More **accessible** regions have B/A > 1.5
Grey box indicates non significant ratio of peptides abundance

Ref.: AC Immune unpublished data

- **Morphomer binding induced conformational changes in Tau aggregates**
- **Most of the conformational changes in Tau are located between amino acids 231-281**

ACI-3024 - Lead characterization

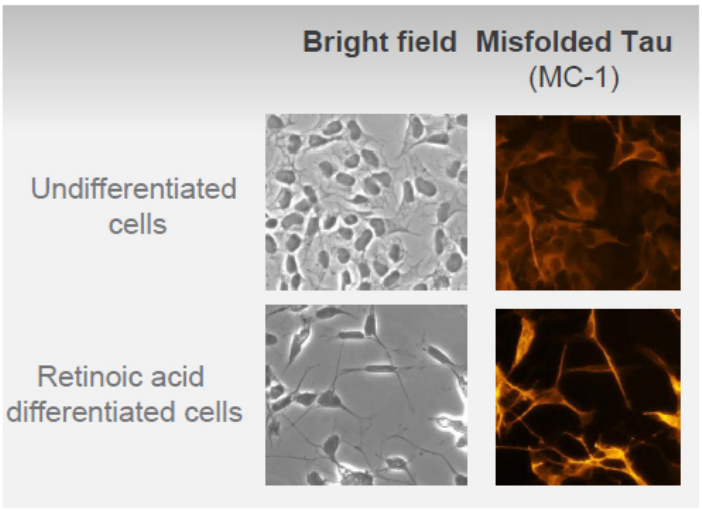
Summary of *in vitro* results

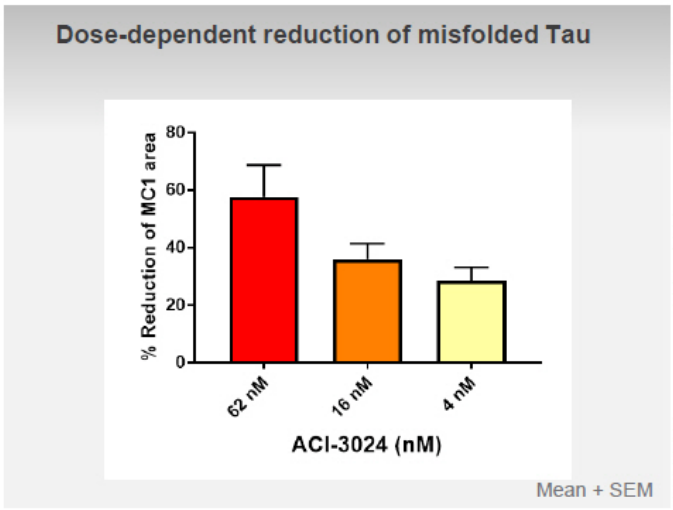
Tau aggregation inhibition	▪ Potent reduction of Tau aggregation ▪ Effect independent of Tau and FTDP-17 isoform mutants
Target engagement	▪ Selective binding to aggregated Tau (25.1 nM) ▪ No binding to monomeric forms of Tau ▪ Selective binding to AD brain-derived pathological Tau (Ki 11.7 nM)
Cross-reactivity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

ACI-3024 – *In vitro* Pharmacology

Dose-dependent reduction of intracellular pathological Tau

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



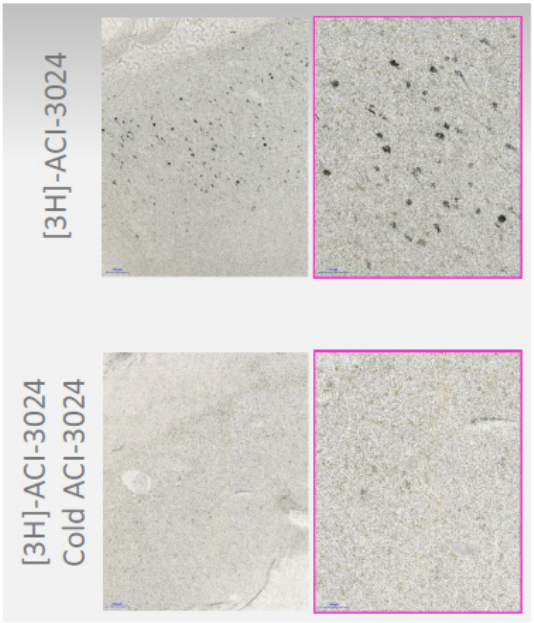


Ref.: AC Immune unpublished data

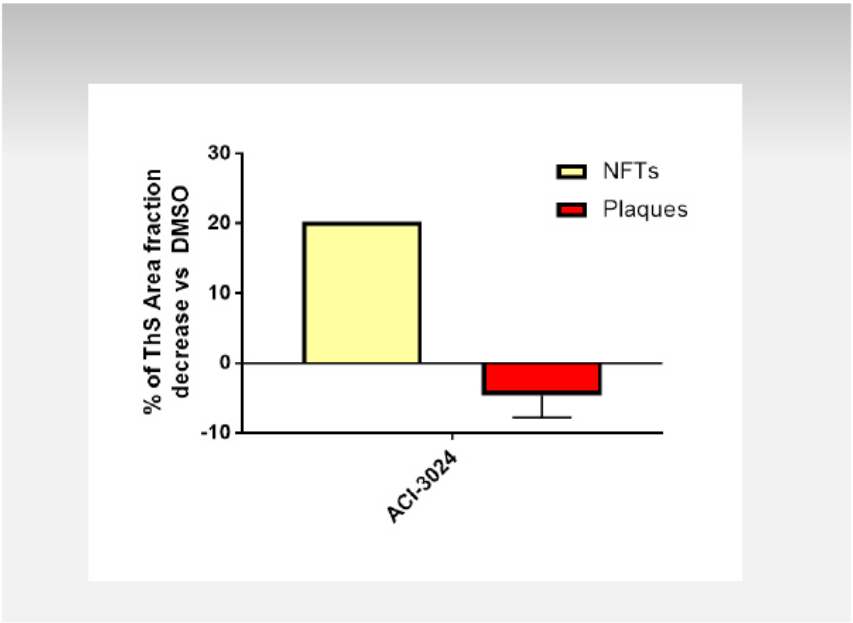
- ***In vitro* treatment with ACI-3024 led to a dose-dependent decrease of misfolded Tau at low nM concentrations**

ACI-3024 - Target engagement and functional selectivity

High resolution autoradiography on human AD brain sections



***Ex vivo* disaggregation on Tau NFT on human AD brain sections**



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

ACI-3024 - *In vivo* Evaluation in rTg4510 mice

Treatment study in aged transgenic mice

Mice

- rTg4510 tauopathy model expresses repressible (Tet promotor Tau on/off) human 4R0N Tau carrying the P301L mutation (SantaCruz, 2005)

Treatment

- Oral administration for 1 month starting at 5 months of age
  - ACI-3024 30mg/kg bi-daily
  - Dose and dosing regimen selected based on the assumption that efficacy is driven by 24 h CSF concentrations above target EC_{50}

Read-out

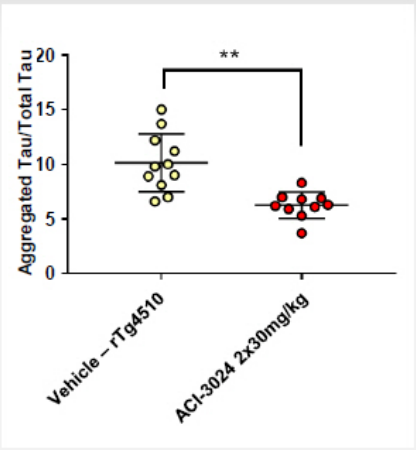
- Biochemistry: total, aggregated, and hyperphosphorylated brain Tau and CSF Tau
- Immuno-histochemistry: misfolded Tau
- Neuroinflammation: microglial analysis

Treatment study results

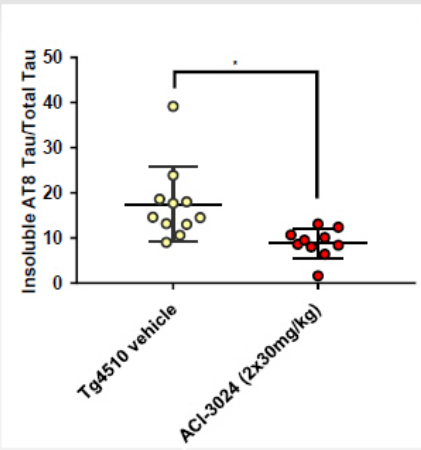
Assessment of compound efficacy in an aggressive Tauopathy model

Biochemistry: Analysis of pathological Tau in Tau ON/OFF rTg4510 mice

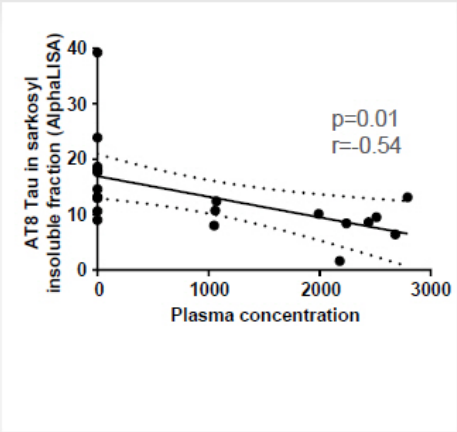
Aggregated Tau[2] normalized to total Tau[1]



Insoluble hyper-phosphorylated Tau[3] normalized to total Tau[1]



Correlation between hyper-phosphorylated Tau in brain and ACI-3024 plasma exposure



[1] HT7xTau13; [2] HT7xHT7; [3] AT8xHT7

Mean + SEM
1-way ANOVA
*** p< 0.001; * p< 0.05

Ref.: AC Immune unpublished data

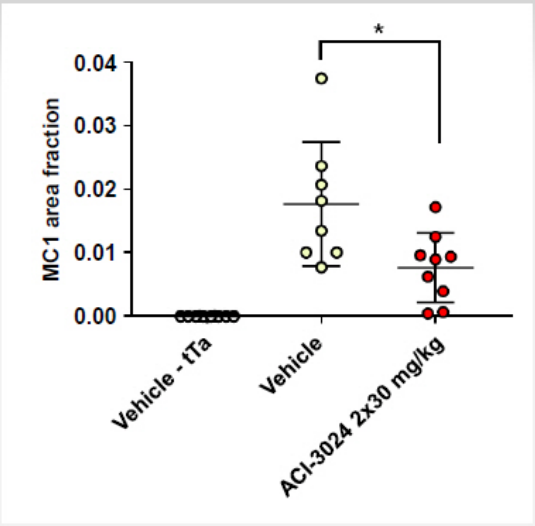
- **Treatment with ACI-3024 significantly reduced aggregated and insoluble pS202/pT205 hyper-phoshorylated Tau in cortical homogenates. The decrease was proportional to the plasma exposure to ACI-3024**

Treatment study results

Assessment of ACI-3024 treatment effects on misfolded Tau

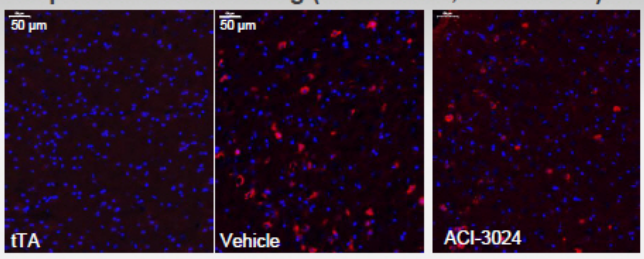
Immunohistochemistry: Analysis of misfolded Tau (MC1) in rTg4510 brain section

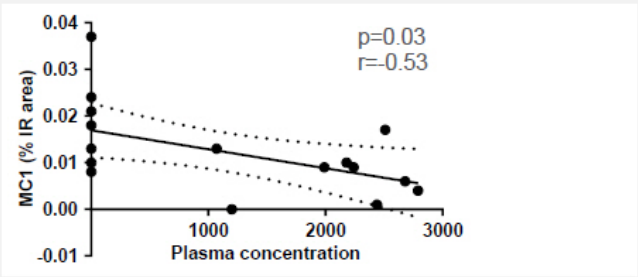
MC1 in brain sections



Mean + SEM
1-way ANOVA
*** p< 0.001
* p< 0.05

Representative staining (MC1 in red, DAPI: blue)





Ref.: AC Immune unpublished data

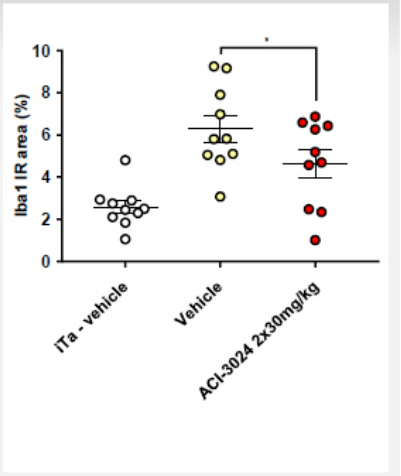
- **Treatment with ACI-3024 significantly reduced misfolded Tau.**
- **The decrease was proportional to the plasma exposure to ACI-3024**

ACI-3024 - Effect on neuro-inflammation

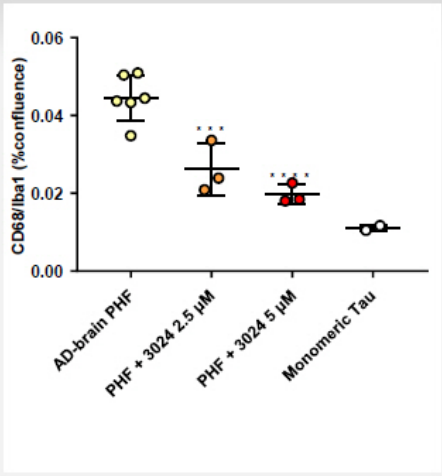
Assessment of compound efficacy on pathological Tau-induced neuro-inflammation

In vivo treatment study	Human AD-brain derived Tau activation of rat primary microglial cells

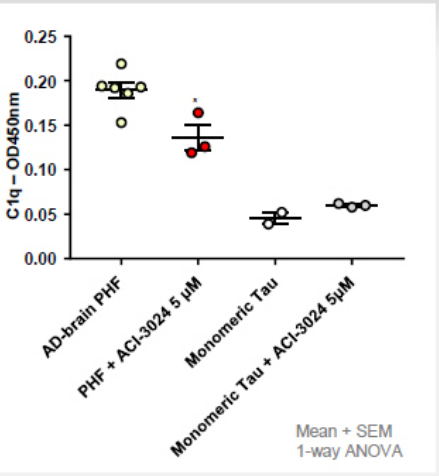
Total Microglia in frontal cortex



CD68+ microglia cells



C1q release from microglia cells



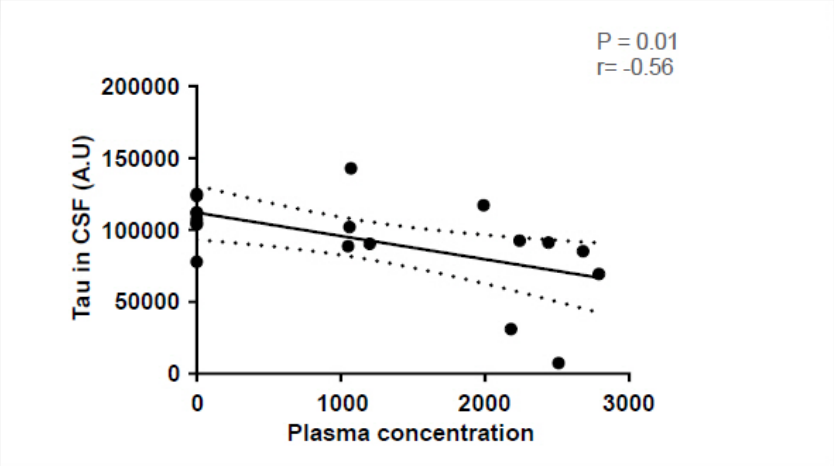
Ref.: AC Immune unpublished data

- **In rTg4510 mice, treatment with ACI-3024 reduced microgliosis. This was likely due to a detoxification of Tau aggregates that consequently decreases pathological Tau induced-microglial activation**

ACI-3024 - Correlations between Tau in CSF and plasma exposure in rTg4510 mice

Evaluation of a potential biomarker for efficacy

Correlation between total Tau in CSF and ACI-3024 plasma exposure

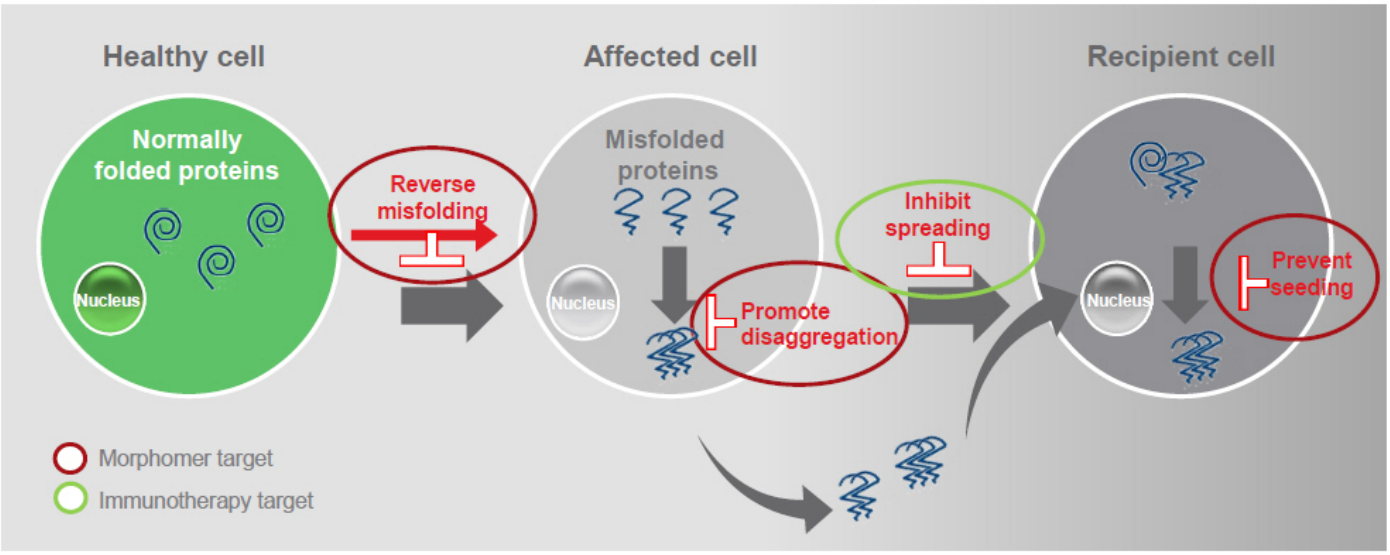


Ref.: AC Immune unpublished data

- **The significant inverse correlation between CSF Tau and ACI-3024 exposure in plasma might indicate an increase of Tau clearance from the brain**
- **CSF Tau concentrations will be explored as a biomarker for efficacy**

AC Immune's targets in spreading hypothesis of misfolded tau in neuro-degenerative diseases

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



- **Targeting both intracellular seeds and extracellular spreading by combination therapy of Morphomers and immunotherapy enables to fully control Tau pathology progression**
- **High selective Tau imaging diagnostic enables more precise patient characterization and potentially more precise prediction of AD progression**

ACI-3024 – Summary of Preclinical evaluation

GLP-toxicology package for CTA submission for FiH studies

***In vitro* on- and off-target activity**	• ACI-3024 is active and selective in multiple *in vitro* pharmacology assays • Binding assessed on 138 targets (Cerep Bioprint profile) shows good selectivity
***In vivo* studies**	• In an *in vivo* therapeutic study ACI-3024 showed compound related treatment effects by biochemistry and IHC (brain, CSF and microglia)
ADME	• ACI-3024 has good *in vitro* and *in vivo* ADME properties, including low clearance, long half-life and good CNS disposition as assessed by brain and CSF concentrations
***In vitro* tox and DDI**	• ACI-3024 has low potential for DDI in vitro (EC_{50} on CYP > 25uM) • It has and no PgP interaction • It is negative in *in vitro* genotoxicity assays (AMES and MNT), and in the *in vivo* MLY
GLP tox in rodents and non rodents	• 4-week toxicology study with 2-week recovery successfully completed • NOAEL established at 300 mg/kg in rodent and 450 mg/kg in non rodent
GLP safety pharmacology	• ICH S7 safety pharmacology battery successfully completed: cardiovascular telemetry study in non rodent; respiratory and Irwin study in rodents
CTA submission	• Preclinical safety evaluation completed and preparation for First in Human studies ongoing

DDI drug-drug interaction; AMES bacterial mutagenesis and carcinogenesis test; MNT micronucleus test in human cell lines; MLY *in vivo* mouse mymphoma

ACI-3024 - Selective Tau aggregation in inhibitors Conclusions

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 efficacy in pathological and functional read-outs in an aggressive transgenic Tauopathy model, with a strong PKPD correlation

4. ACI-3024 has shown excellent preclinical safety and tolerability profile and is entering clinical development as disease-modifying agent for neurodegenerative diseases characterized by misfolded tau

AC Immune

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