

---

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 July, 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  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

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

---

**AC IMMUNE SA**

On July 11, 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 Exhibit 99.1 hereto. A related press release describing certain of the information being presented at the KOL meeting is also attached as Exhibit 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333-216539) of AC Immune and to be a part thereof from the date on which this report is filed, to the extent not superseded by documents or reports subsequently filed or furnished.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: July 11, 2019

---

## EXHIBIT INDEX

<b>Exhibit Number</b>	<b>Description</b>
---------------------------	--------------------

---

99.1	Presentation dated July 11, 2019
99.2	Press Release dated July 11, 2019

---



**ACI-24 anti-Abeta vaccine for prevention and therapy in Alzheimer's disease and Down syndrome**



NASDAQ: ACIU | KOL Breakfast NYC | July 2019

© 2019 AC Immune. Not to be used or reproduced without permission.

Version 07 July 2019

[www.acimmune.com](http://www.acimmune.com)



# Disclaimer

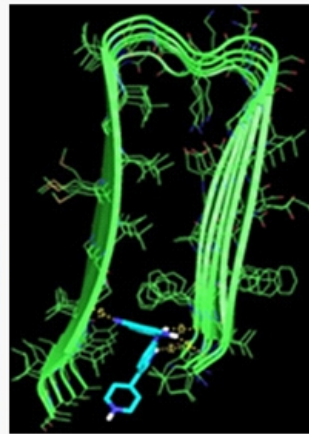
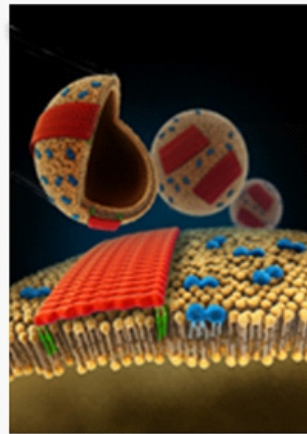
*This presentation may contain statements that constitute “forward-looking statements” within the meaning of Section 27A of the Securities Act of 1933 and Section 21E of the Securities Exchange Act of 1934. Forward-looking statements are statements other than historical fact and may include statements 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.*

*This presentation is strictly confidential, is being distributed to a limited range of invited persons solely for their own information, may not be distributed to the press or any other person, and may not be reproduced or published, in whole or in part, in any form.*

# AC Immune's dual technology platforms

## SupraAntigen™

Vaccines and antibodies specific to disease causing conformations



## Morphomer™

Conformation-sensitive small molecules

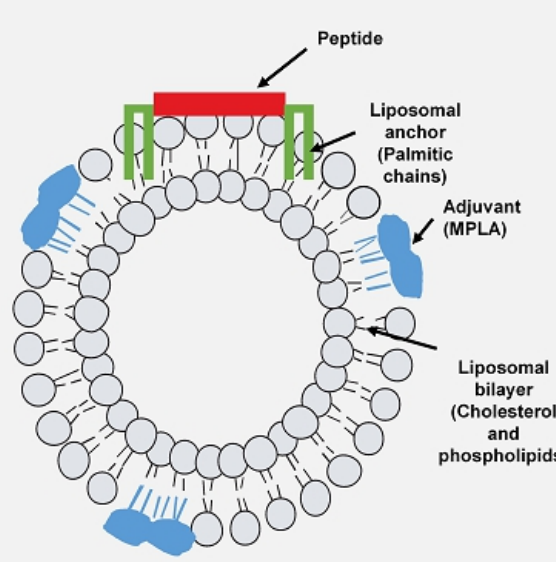
- Highly selective conformation-specific immunotherapy
- Antibodies and vaccines
- Rapid antibody response
- Favorable safety (T-cell independent MoA<sup>1</sup>)

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

(1) Mode of action

## ACI-24 structure

Strong antibody response against pathological human Abeta without activation of Abeta-specific T-cells



The diagram illustrates the structure of an ACI-24 liposome. It is a spherical vesicle composed of a bilayer of lipids, including cholesterol and phospholipids. The liposome is anchored to a peptide (red bar) via palmitic chains (green bars). The liposome also contains an adjuvant (MPLA), shown as blue structures. Labels include: Peptide, Liposomal anchor (Palmitic chains), Adjuvant (MPLA), and Liposomal bilayer (Cholesterol and phospholipids).

**ACI-24 is an active immunotherapeutic based on AC Immune's proprietary SupraAntigen™ technology**

- Peptide: Epitope Binding
  - ACI-24 (Pa11-15) peptide produced by chemical synthesis
- Linker: Conformation
  - Palmitic chains anchor peptides to generate *conformation-specific β-sheets*
- Liposome + Adjuvant: Immunogenicity
  - ACI-24 liposomes contain a vaccine adjuvant – Synthetic Monophosphoryl Lipid A (MPLA)

### Major strengths

- Pathological conformation of Abeta ( $\beta$ -sheets) to crosslink B cell receptor
- Lack of Abeta specific T cell epitopes
- Toll-like receptor triggering



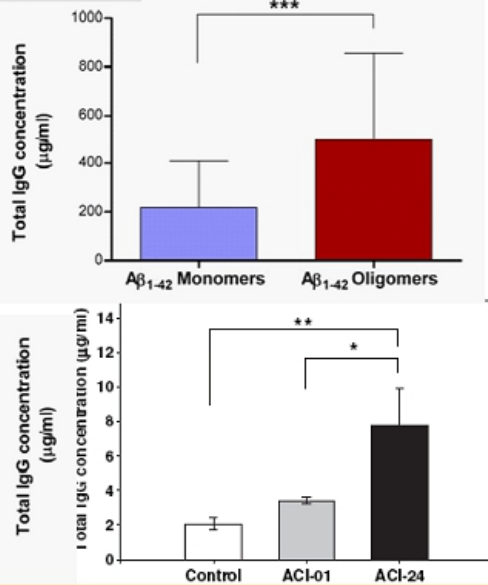
# ACI-24: proof-of-concept

Key preclinical results: preferential binding to Abeta oligomers and plaques

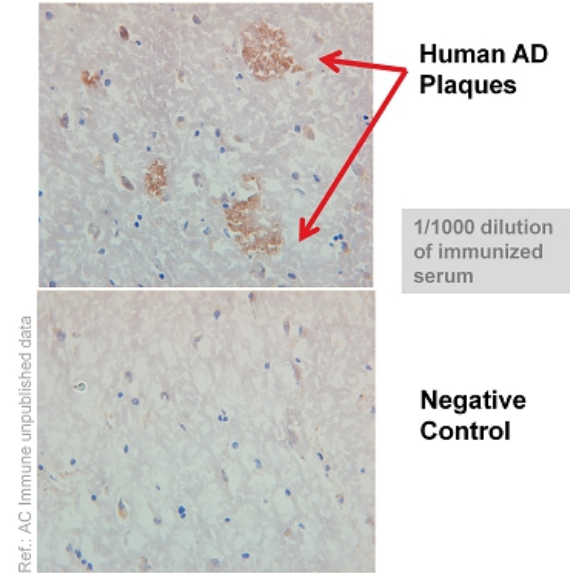
## Binding of ACI-24 immunized mouse sera to Abeta monomers, oligomers and fibers

Anti-Abeta IgG (Elisa)

Ref.: Hickman et al., JBC, 2011  
Ref.: Muhs et al., PNAS, 2007



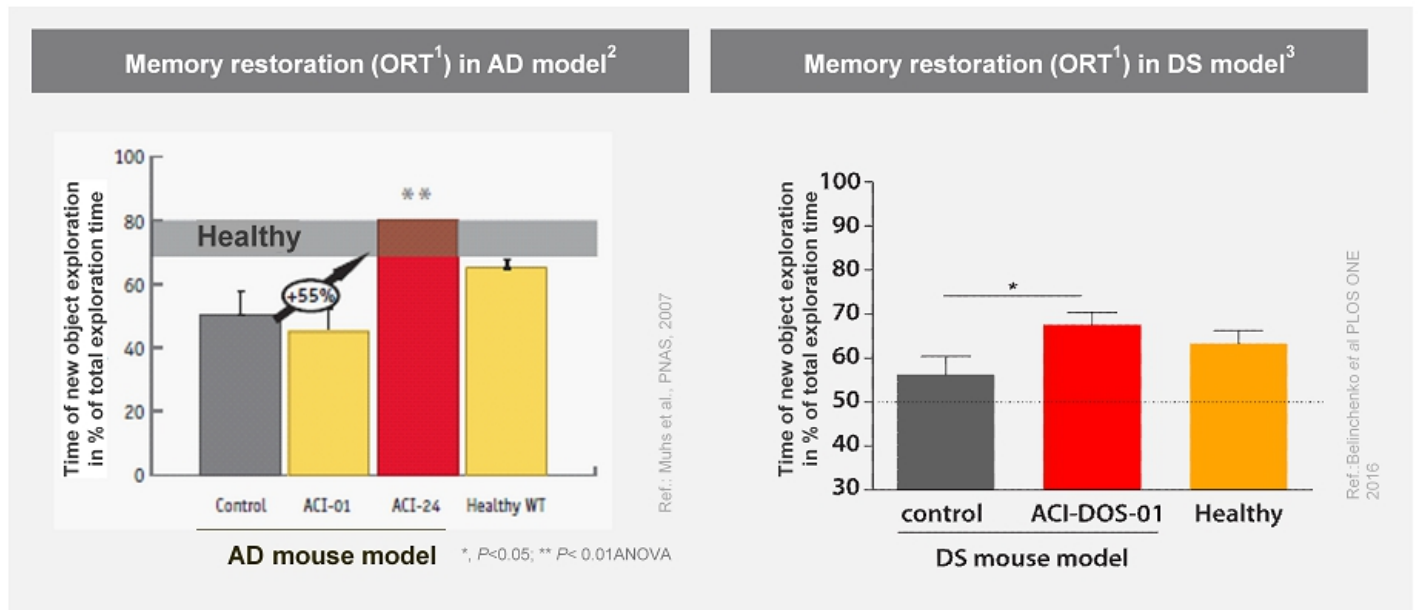
## Binding of ACI-24 immunized monkey sera to human brain



- Strong binding to the pathological forms of Abeta
- Binding of immunized monkey sera to human AD brain

# ACI-24: proof-of-concept

Key preclinical results: memory restoration in AD and DS mouse models



ACI-24 improves cognition in AD and DS transgenic mouse models

(1) Object recognition test; (2) APPxPS-1 model; (3) TS65Dn model

# ACI-24: summary of preclinical studies

Non-clinical and toxicology studies for AD and DS indications

## ACI-24 in non-clinical studies:

- Strong and consistent anti-Abeta IgG response
- High specificity towards the pathological Abeta
- Efficacy in AD and DS transgenic mouse models
- No Abeta-specific T-cell activation

## ACI-24 in toxicology studies:

- No systemic adverse effects up to highest dose in the following studies
  - Single dose study in mice (up to 385 µg s.c., 65 µg i.m.)
  - Single dose study in monkeys (up to 385 µg s.c.)
  - Repeat dose studies in APP<sup>1</sup> tg-mice (up to 400 µg s.c.) and in old APPxPS<sup>2</sup>-1 tg mice (up to 26 µg i.p.)
  - 2 repeat dose studies in monkeys (up to 1320 µg s.c.)



- Strong and specific antibody response
- Excellent safety profile

(1) Amyloid precursor protein; (2) presenilin



ACI-24-0701 study: A Phase 1/2 Double-Blind, Randomized, Placebo-Controlled, Adaptive Design Study of the Safety, Tolerability, Immunogenicity and Efficacy of ACI-24 in Patients with Mild to Moderate Alzheimer's Disease





# ACI-24-0701 study in AD

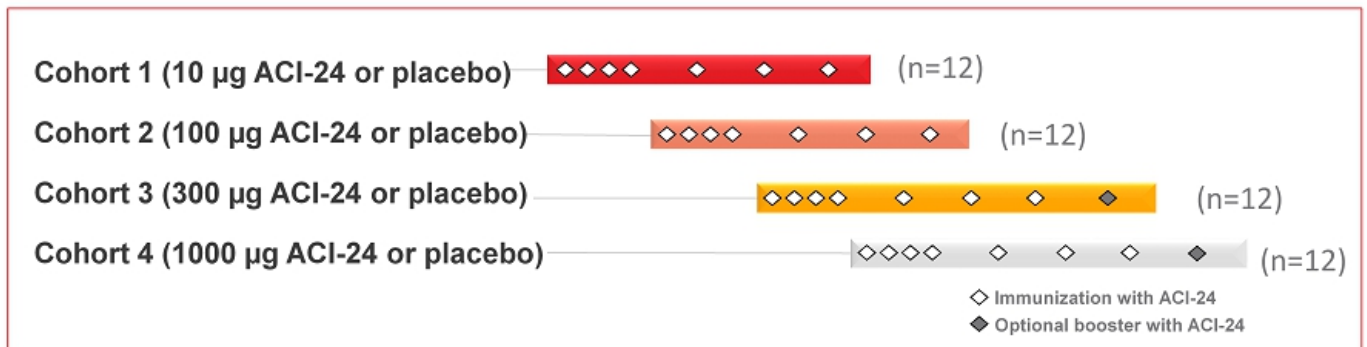
## Study overview

Study population	<ul style="list-style-type: none"><li>▪ Participants with mild to moderate probable Alzheimer's disease (NINCDS-ADRDA<sup>1</sup> criteria)</li><li>▪ MMSE<sup>2</sup>: 18–28; age &gt;40 and &lt;90 yo</li></ul>
Primary endpoints	<ul style="list-style-type: none"><li>▪ Safety and tolerability</li><li>▪ Induction of anti-Abeta 1-42 IgG titers in serum</li></ul>
Secondary endpoints	<ul style="list-style-type: none"><li>▪ Reduction of brain Abeta levels using amyloid PET-scan</li><li>▪ Induction of T-cell activation</li><li>▪ Putative biomarkers of the progression of AD i.e., Tau, phosphorylated Tau and Abeta levels (A<math>\beta</math>1–42 and A<math>\beta</math>1–40) in blood and CSF</li><li>▪ Efficacy on clinical/cognitive parameters</li><li>▪ Immune response in serum and/or CSF including, but not limited to anti-Abeta1-42 IgM titers in blood</li><li>▪ Inflammatory cytokines in blood</li></ul>

(1) National Institute of Neurological and Communicative Disorders and Stroke and the Alzheimer's Disease and Related Disorders Association; (2) Mini-Mental State Examination

# ACI-24-0701 study in AD

## Clinical study design



- Ratio of 3:1 active (ACI-24) versus placebo per cohort
- Administration subcutaneously:
  - Cohorts 1 to 3 : Weeks 0, 4, 8, 12, 24, 36 and 48 with optional booster injection in cohort 3
  - Cohort 4 : Weeks 0, 4, 8, 12, 24, 36, 48 and 74 with booster injection at week 74

# ACI-24-0701 study in AD

## Demographics and baseline characteristics

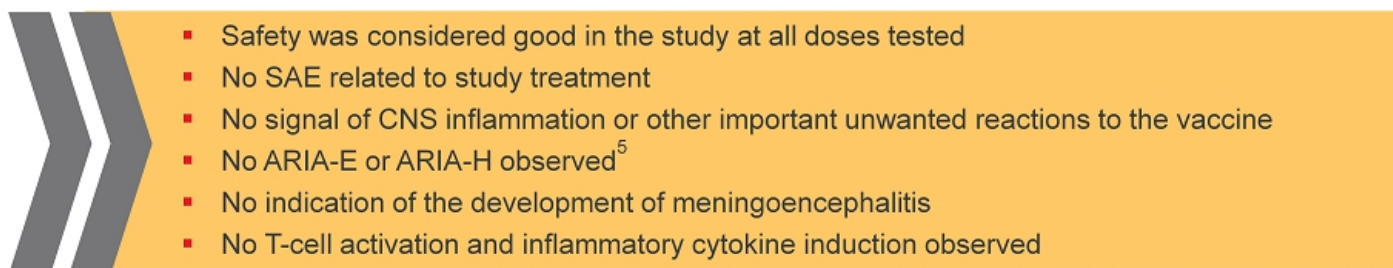
Characteristic	ACI-24				Placebo
Dose	10 µg	100 µg	300 µg	1000 µg	-
Number of patients	9	9	9	9	12
Age, mean (SD <sup>1</sup> )	69.4 (8.7)	65.2 (8.3)	71.1 (4.9)	62.2 (8.5)	69.1 (4.8)
Female, number (%)	5 (55.6)	4 (44.4)	6 (66.7)	3 (33.3)	5 (41.7)
MMSE <sup>2</sup> , mean (SD)	21.9 (2.7)	22.7 (2.4)	22.7 (3.0)	24.8 (2.1)	22.8 (2.7)

(1) Standard deviation; (2) Mini-Mental State Examination

# ACI-24-0701 study in AD

Overall summary of AEs<sup>1</sup> – safety analysis set

Characteristic	ACI-24				Placebo
	10µg (9)	100µg (9)	300µg (9)	1000µg (9)	
Dose (# of patients)					- (12)
Treatment exposure in days (SD <sup>2</sup> ) Min; Max <sup>3</sup>	336 (1.4) 334; 338	337 (1.9) 334; 341	337 (0.7) 336; 338	462 (124.1) 252; 644	325 (98.7) 85; 532
Nb patients with ≥ 1 AE (%)	8 (88.9)	9 (100.0)	9 (100.0)	9 (100.0)	9 (100.0)
Nb patients with severe AEs (%)	1 (11.1)	1 (11.1)	0	3 (33.3)	3 (25.0)
AEs leading to withdrawal (%)	0	0	0	1 (11.1)	0
Serious AEs (%)	2 (22.2)	1 (11.1)	0	3 (33.3)	4 (33.3)
Death (%)	0	1 (11.1)		1 (11.1)	1 (8.3)
Treatment related AEs <sup>4</sup> (%)	5 (55.6)	4 (44.4)	5 (55.6)	8 (88.9)	6 (50)



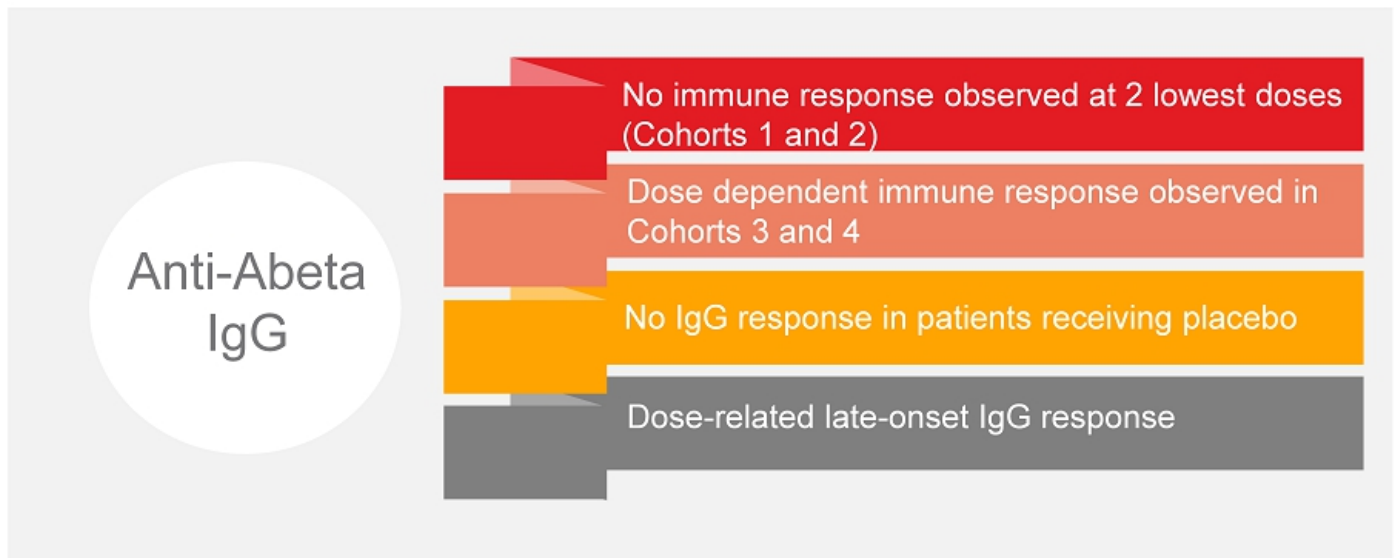
- Safety was considered good in the study at all doses tested
- No SAE related to study treatment
- No signal of CNS inflammation or other important unwanted reactions to the vaccine
- No ARIA-E or ARIA-H observed<sup>5</sup>
- No indication of the development of meningoencephalitis
- No T-cell activation and inflammatory cytokine induction observed

(1) Adverse Event; (2) Standard deviation; (3) Cohort 3 booster not counted in the table; (4) AE assessed by investigator as possibly/probably related to investigational product; (5) One tiny lesion with low signal on hemosequence suspicious for a microbleed observed at 100 µg dose of ACI-24 (possible artefact)



# ACI-24-0701 study in AD

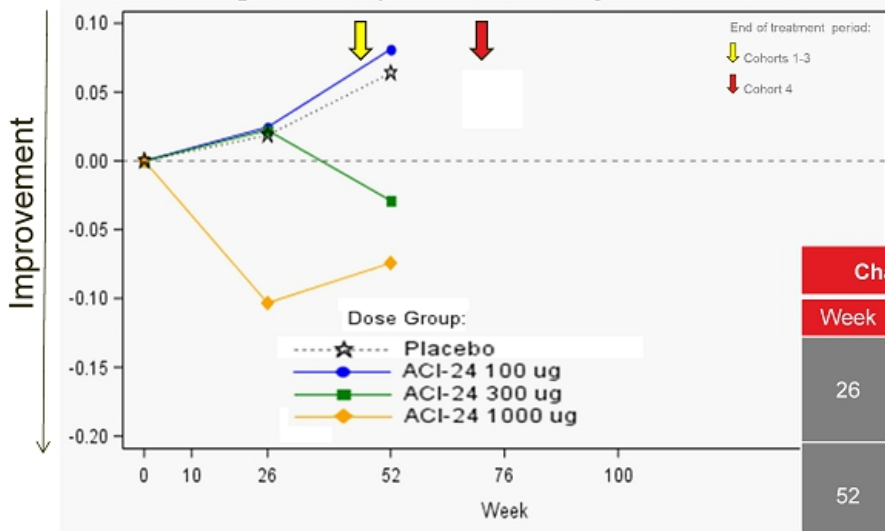
## Immunogenicity study results



# ACI-24-0701 study in AD

Abeta PET<sup>1</sup> exploratory analysis

Change in composite summary SUVR-MCG<sup>2</sup>



■ A dose dependent<sup>3</sup> trend in reduction of brain amyloid accumulation observed in cohorts 3 and 4 at week 52

Characteristic		ACI-24			Placebo
Week	Dose	100 µg	300 µg	1000 µg	-
26	# patients	9	9	3	7
	Mean Comp. score (SD)	0.02 (0.21)	0.02 (0.19)	-0.10 (0.10)	0.02 (0.28)
52	# patients	9	9	7	8
	Mean Comp. score (SD)	0.08 (0.25)	-0.03 (0.22)	-0.07 (0.07)	0.06 (0.22)

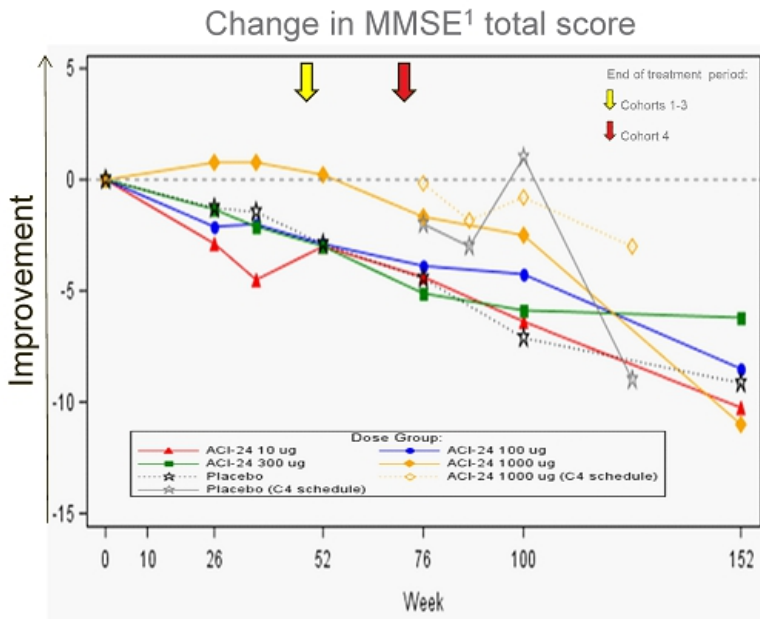


■ Tendency for reduction in brain amyloid load observed in cohorts 3 and 4

(1) Florbetaben Positron Emission Tomography; (2) Standardised Uptake Value Ratio-Mean Cerebellar Gray; (3) PET scans not conducted for Cohort 1

# ACI-24-0701 study in AD

## Exploratory analysis on cognition



- A positive trend on cognition measured by MMSE
- Observed during the treatment period for the highest dose versus placebo and lower doses



- Tendency for effect on cognition in a limited population; study not powered on this parameter

(1) Mini-mental state examination

# ACI-24-0701 study in AD

## Key learnings

1	<ul style="list-style-type: none"><li>▪ Safe in AD participants with no study treatment-related SAEs<sup>1</sup></li></ul>
2	<ul style="list-style-type: none"><li>▪ Dose-related anti-Abeta IgG responses observed</li></ul>
3	<ul style="list-style-type: none"><li>▪ A potential dose-dependent positive trend on Abeta in brain (PET<sup>2</sup>), MMSE<sup>3</sup> total score and functional outcome (CDR-SB<sup>4</sup>) observed, however, results derived from a small study population</li></ul>
4	<ul style="list-style-type: none"><li>▪ For ongoing Phase 2 trial:<ul style="list-style-type: none"><li>▪ Use 1000 µg dose</li><li>▪ Administer intramuscularly</li><li>▪ Measure free, total and immune complexed IgG titers</li></ul></li></ul>

(1) Serious adverse events; (2) Positron Emission Tomography; (3) Mini-mental state examination; (4) Clinical dementia rating scale-sum of boxes



ACI-24-1301 study: Phase 1b Multi-Center, Double-Blind, Randomized, Placebo-Controlled Dose Escalation Study of the Safety, Tolerability and Immunogenicity of ACI-24 in Adults with Down Syndrome – currently ongoing





# ACI-24-1301 study in DS

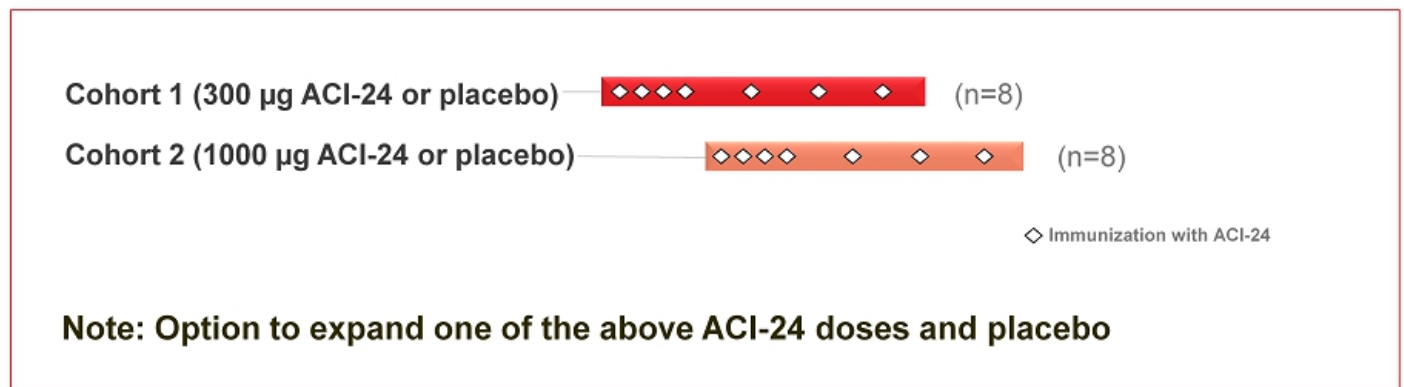
## Study overview – Study currently ongoing

Study population	<ul style="list-style-type: none"><li>▪ Male or female participants with DS age <math>\geq 25</math> to <math>\leq 45</math> years</li><li>▪ Cytogenetic diagnosis of either Trisomy 21 or complete unbalanced translocation of Chromosome 21; IQ <math>\geq 40</math> (KBIT-2)<sup>1</sup></li></ul>
Primary endpoints	<ul style="list-style-type: none"><li>▪ Safety and tolerability</li><li>▪ Induction of anti-Abeta Ig titers in serum</li></ul>
Secondary endpoints	<ul style="list-style-type: none"><li>▪ Efficacy on CGIC<sup>2</sup></li><li>▪ Cognition (CANTAB<sup>3</sup> motor control, reaction time, paired associative learning; BPT<sup>4</sup>) and behavior (VABS<sup>5</sup>, NPI<sup>6</sup>)</li><li>▪ Whole brain, ventricle and hippocampal volume by MRI</li><li>▪ Induction of T-cell activation</li><li>▪ Putative biomarkers of Alzheimer pathology including Abeta levels, total tau, phosphorylated Tau protein, sAPP<math>\alpha</math><sup>7</sup>, sAPP<math>\beta</math><sup>8</sup>, Orexin-A, inflammatory cytokines, angiogenic proteins and vascular injury markers in plasma and/or in CSF, TLR-4 expression</li><li>▪ Induction of anti-Abeta Ig titers in CSF</li></ul>

(1) Kaufman Brief Intelligence Test – Second Edition ; (2) Clinical Global Impression of Change; (3) Cambridge Neuropsychological Test Automated Battery; (4) Brief Praxis Test; (5) Vineland Adaptive Behavior Scale; (6) Neuropsychiatric Inventory; (7) Soluble amyloid precursor protein alpha; (8) Soluble amyloid precursor protein beta

# ACI-24-1301 study in DS

## Clinical study design



- Ratio of 3:1 active (ACI-24) versus placebo per cohort (n=8)
- Administration subcutaneously at weeks 0, 4, 8, 12, 24, 36 and 48
- Sequential and progressive dosing to enable a better control of safety and tolerability

# ACI-24-1301 study in DS

## Demographics and baseline characteristics

Characteristic	ACI-24		Placebo
Dose	300 µg	1000 µg	-
Number of subjects	6	6	4
Age, mean (SD <sup>1</sup> ) (min; max)	33.5 (4.6) (28;41)	31.5 (4.9) (25;36)	33.0 (4.2) (28;38)
Female, number (%)	2 (33.3)	4 (66.7)	3 (75.0)
Years of education, mean (SD <sup>1</sup> )	12.0 (0.0)	14.2 (3.7)	15.0 (3.5)

(1) Standard deviation



## ACI-24-1301 study in DS

Overall summary of AEs<sup>1</sup> – Safety Analysis Set – interim data as of June 26, 2019

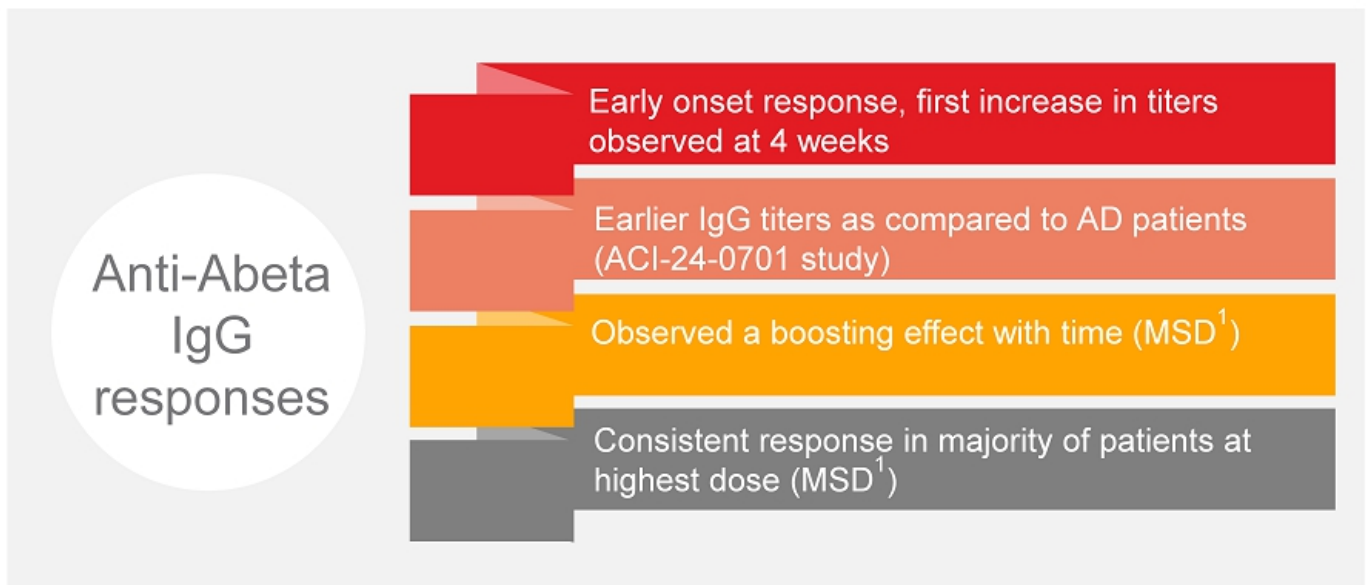
Characteristic	ACI-24		Placebo
Dose of study treatment (# of patients)	300µg (6)	1000µg (6)	- (4)
Early withdrawal (%)	0 (0.0)	0 (0.0)	0 (0.0)
Serious AEs (%)	0 (0.0)	0 (0.0)	0 (0.0)

- Safety is considered good in the study at all doses tested so far
- No SAE<sup>2</sup> reported
- No signal of CNS inflammation or other important unwanted reactions to the vaccine
- No ARIA-E or ARIA-H<sup>3</sup> observed
- No indication of the development of meningoencephalitis
- No T-cell activation and inflammatory cytokine induction observed so far

(1) Adverse Event; (2) Serious Adverse Event; (3) Alzheimer's Related Imaging Abnormality-Edema or -Haemorrhages

# ACI-24-1301 study in DS

Preliminary immunogenicity results – interim data as of June 26, 2019



(1) Initial MesoScale Discovery immunoassay platform data

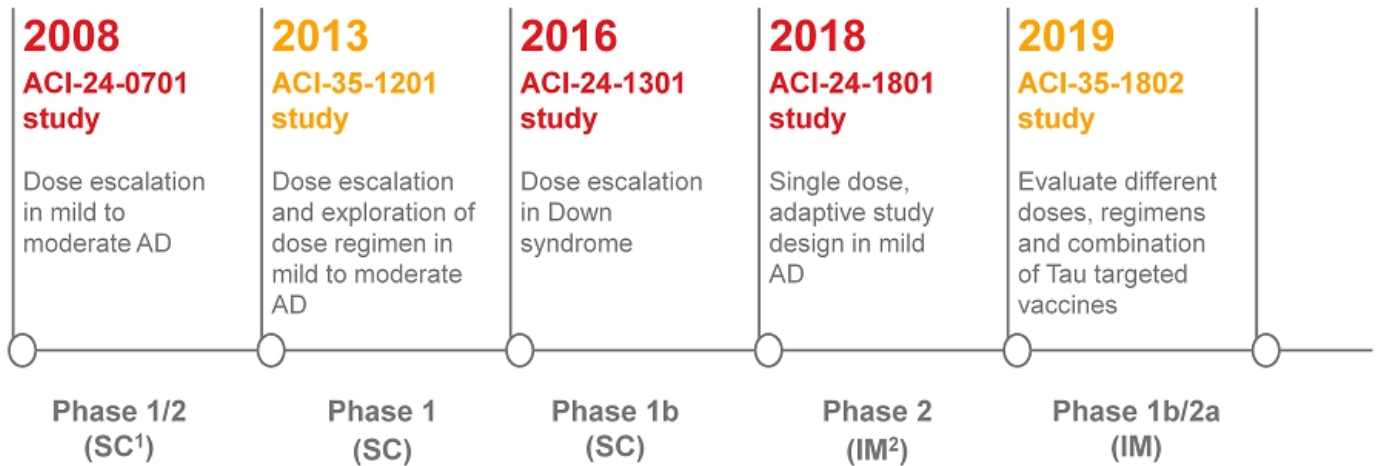
# ACI-24-1301 study in DS

Key learnings – interim data as of June 26, 2019

1	<ul style="list-style-type: none"><li>▪ Safe in DS subjects with no SAEs<sup>1</sup> and no early withdrawal at any dose to date. No indication of CNS inflammation or meningoencephalitis. No ARIA-E or -H<sup>2</sup> observed</li></ul>
2	<ul style="list-style-type: none"><li>▪ Early and consistent onset of anti-Abeta IgG response</li></ul>
3	<ul style="list-style-type: none"><li>▪ Plans for accelerated development:<ul style="list-style-type: none"><li>▪ Focus on prevention therapy using biomarker endpoints</li><li>▪ Use highest dose via the intramuscular route to further boost immunogenicity</li><li>▪ Measure the impact of ACI-24 on biomarkers and clinical efficacy</li><li>▪ Include PET<sup>3</sup>-scan imaging</li><li>▪ Measure free, total and immune complexed IgG titers</li></ul></li></ul>

(1) Serious adverse events; (2) Alzheimer's Related Imaging Abnormality-Edema or -Haemorrhages; (3) Positron Emission Tomography

# AC Immune: pioneers in anti-Abeta and anti-Tau vaccines



(1) Subcutaneous; (2) Intramuscular



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

## AC Immune Hosts KOL Event and Reports Initial Interim Clinical Data for ACI-24 Vaccine to Treat Alzheimer's Disease-like Symptoms in Subjects with Down Syndrome

*Anti-Abeta vaccine demonstrates strong safety and preliminary immunogenicity results in subjects with Down syndrome*

*Presentations underscore significant need and opportunity for studying Alzheimer's disease-like symptoms in this high-risk and genetically homogeneous population*

**Lausanne, Switzerland, 11, July 2019** – AC Immune SA (NASDAQ: ACIU), a Swiss-based, clinical-stage biopharmaceutical company with a broad pipeline focused on neurodegenerative diseases, today announced initial interim data from an ongoing Phase 1b trial of the ACI-24 anti-Abeta vaccine to treat Alzheimer's disease (AD) like symptoms in subjects with Down syndrome (DS) as well as key takeaways from its Key Opinion Leader (KOL) meeting, held in New York City.

**Professor Andrea Pfeifer, CEO of AC Immune, commented:** "These initial interim Phase 1b data support the continued study of ACI-24 in this trial to treat AD-like symptoms in DS as well as in our ongoing Phase 2 trial in subjects with mild AD. We thank Dr. Skotko and Professor Mobley, leading Key Opinion Leaders, whose participation today highlights the unmet need and the significant opportunity that exists for studying AD-like symptoms in this more homogeneous population, which may yield critical information for the potential benefit of DS subjects as well as the broader AD community."

The event, which will be recorded and can be viewed [here](#), is being co-chaired by Professor Pfeifer and Professor William Mobley, Executive Director of the University of California San Diego's Down Syndrome Center for Research and Treatment. It features presentations from Professor Mobley and Dr. Brian Skotko, Associate Professor at Harvard Medical School and the Emma Campbell Endowed Chair on Down Syndrome and Director of the Down Syndrome Program at Massachusetts General Hospital, as well as Professor Pfeifer and Dr. Marie Kosco-Vilbois, Chief Scientific Officer of AC Immune.

### Highlights from Dr. Kosco-Vilbois' presentation include:

- The trial is a fully enrolled, placebo-controlled, 16 subjects Phase 1b study of AC Immune's ACI-24 anti-Abeta vaccine
  - To date, ACI-24 was well tolerated by DS subjects, demonstrating a favourable safety profile at all doses tested, mirroring previous clinical trial results
  - There were no subjects withdrawals from the study or serious adverse events (SAE) reported, no signals of CNS inflammation or other pro-inflammatory reactions, no amyloid-related imaging abnormalities (ARIA-E/ARIA-H) or indication of meningoencephalitis
-

- Preliminary immunogenicity data showed an anti-Abeta IgG response that was detectable at week 4
- Pending the final outcome, the Phase 2 study of ACI-24 in subjects with DS would likely focus on disease prevention and will include biomarkers and Positron Emission Tomography (PET) imaging to monitor disease progression, in addition to quantifying anti-Abeta IgG titers generated by the vaccine.

**Dr. Kosco-Vilbois, CSO of AC Immune, commented:** "We are pleased that to date in this study our proprietary, highly selective, conformation-specific anti-Abeta vaccine has been safe and was well tolerated and demonstrated preliminary signals of activity in DS subjects. These early immunogenicity data, showing an IgG response, are encouraging. Given the Abeta-driven nature of AD-like symptoms in DS subjects, we believe that ACI-24, which targets pathologic forms of Abeta, may reduce the appearance of amyloid burden and related brain lesions and potentially slow or halt progression of the disease."

**Highlights of Dr. Skotko's presentation include:**

- DS is caused by having a third copy (trisomy) of chromosome 21 and occurs in 1/~792 live births; ~5,000 children per year
- ~212,000 people living with DS in the US and ~359,000 in Europe
- Adults with DS are increasingly moving into semi-independent living situations and securing paid employment, are involved in romantic relationships and marry, participate in day habilitation services or are active participants in sports (Special Olympics)
- Initial symptoms are typically changes in behavior; seizures can also be an early warning sign, followed by memory loss
- Based on surveys, family members and people with DS say they are satisfied, even positive, about their lives despite acknowledging the challenges that accompany DS

**Highlights from Professor Mobley's presentation include:**

- The neuropathological changes in DS subjects are very similar but not identical to typical AD. People with Down syndrome have an extra copy of chromosome 21, which houses the gene that codes for amyloid precursor protein (APP). APP is the parent protein of Abeta, a protein fragment that accumulates into amyloid plaques, a key feature of AD
  - Studying AD-like symptoms in the DS population addresses many of the key dilemmas that hinder the discovery of new treatments: uncertain mechanisms and timing of disease-induced brain changes, difficulty offering treatment before disease onset, genetic and age-related variability, and the risk of including subjects with other forms of age-related dementia
  - For AD in DS, the disease mechanism and approximate timing of onset are known; readily detectable pathological changes occur prior to AD-like symptoms, enabling treatment prior to disease onset; the DS population is far more homogeneous and carries minimal risk of having coexisting conditions causing dementia at such a young age
  - In mouse models of DS, vaccination against Abeta by ACI-24 improved cognition and prevented neuronal atrophy
-

**Professor Mobley commented:** “DS is an underrepresented, overlooked population at increased risk for AD-like disease. It offers opportunities for exploring effective treatments for AD that will benefit both the DS and general populations. Homogeneity in pathogenesis, age-related disease onset and absence of other dementias powerfully enable prevention trials of AD-like symptoms in DS. I am encouraged by the body of evidence supporting this rationale and personally gratified to be conducting the first clinical trial of a vaccine against Abeta in individuals with DS.”

DS is characterized by the onset of AD-like symptoms by age 20, and nearly all DS subjects display AD-like symptoms by age 40. Treating AD-like symptoms in DS subjects is imperative and information from this patient population may also be pivotal for developing successful treatments for the broader AD population. Many KOLs – and AC Immune – believe that this approach may make it possible to more rapidly identify successful treatment strategies, including combination therapies for AD, to benefit DS subjects as well as the AD community. This was the theme of AC Immune’s KOL Breakfast today, which examined the Company’s Roadmap for developing innovative treatment paradigms for neurodegenerative diseases.

#### **Key Opinion Leader Biographies:**

Professor William C. Mobley, M.D., Ph.D., is an Executive Director of UCSD’s Down Syndrome Center for Research and Treatment and the Florence Riford Chair of Alzheimer’s Disease Research. Professor Mobley came to UCSD in 2009 from Stanford University where he served as the John E. Cahill Family Professor in the Department of Neurology and Neurological Sciences and was the founding director of the Neuroscience Institute. Professor Mobley earned his M.D. and Ph.D. in Neuro- & Behavioral Science, as well as an internship in pathology, all at Stanford University in Palo Alto, California. He then went on to complete a residency and fellowship in neurology and pediatric neurology at The Johns Hopkins University. While there, he was selected to serve as Chief Resident in Pediatric Neurology. He is certified by the American Board of Pediatrics and the American Board of Psychiatry and Neurology with Special Competence in Child Neurology.

Dr. Brian Skotko, M.D., M.P.P., is a Board-certified medical geneticist and the Emma Campbell Endowed Chair on Down Syndrome at Massachusetts General Hospital. As the Director of the hospital’s Down Syndrome Program, he has dedicated his professional energies toward children with cognitive and development disabilities. Dr. Skotko co-authored the national award-winning books, *Common Threads: Celebrating Life with Down Syndrome* and *Fasten Your Seatbelt: A Crash Course on Down Syndrome for Brothers and Sisters*. He is a graduate of Duke University, Harvard Medical School, and Harvard Kennedy School, and he is currently an Associate Professor at Harvard Medical School. Dr. Skotko is a leader on clinical and translational research in the field of Down syndrome. He has been featured in *The Wall Street Journal*, *The New York Times*, *The Washington Post*, *The L.A. Times*, NPR’s “On Point,” and ABC’s “Good Morning America.” Dr. Skotko serves on the Honorary Board of Directors for the Massachusetts Down Syndrome Congress. Dr. Skotko has a sister with Down syndrome.

#### **About AC Immune SA**

AC Immune SA is a Nasdaq-listed clinical-stage biopharmaceutical company, which aims to become a global leader in precision medicine for neurodegenerative diseases. The Company is utilizing two proprietary discovery platforms, SupraAntigen™ and Morphomer™, to design, discover and develop small molecule and biological therapeutics as well as diagnostic products intended to diagnose, prevent and modify

---



neurodegenerative diseases caused by misfolding proteins. The Company's pipeline features nine therapeutic and three diagnostic product candidates, with five currently in clinical trials. It has collaborations with major pharmaceutical companies including Roche/Genentech, Eli Lilly and Janssen.

**For further information, please contact:**

**US Investors**

Joshua Drumm, Ph.D.  
AC Immune Investor Relations  
Phone: +1 646 876 5538  
E-mail: [joshua.drumm@acimmune.com](mailto:joshua.drumm@acimmune.com)

**US Media**

Katie Gallagher or Sharon Correia  
LaVoieHealthScience  
Phone: +1 617 792 3937  
E-mail: [kgallagher@lavoiehealthscience.com](mailto:kgallagher@lavoiehealthscience.com)  
[scorreia@lavoiehealthscience.co](mailto:scorreia@lavoiehealthscience.co)

**European Media**

Judith Moore  
AC Immune Global Communication Lead  
Phone: +44 7932 053829  
E-mail: [judith.moore@acimmune.com](mailto:judith.moore@acimmune.com)

**European Investors**

Chris Maggos  
LifeSci Advisors  
Phone: +41 79 367 6254  
E-mail: [chris@lifesciadvisors.com](mailto:chris@lifesciadvisors.com)

**Forward looking statements**

This press release contains 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.

---