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 December 2016

_____

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

Item 7.01 Regulation FD Disclosure.

AC Immune SA ("**AC Immune**") has updated its investor presentation with recent data presented by its partner Genentech, a member of the Roche group, at the 9th Clinical Trials on Alzheimer's Disease Conference (CTAD) in San Diego. The updated investor presentation and informational poster that summarizes the data are attached hereto as Exhibit 99.1 and 99.2, respectively. From time to time, AC Immune will use this updated presentation and the informational poster in conversations with investors, analysts and others.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits

Exhibit Number	Description
99.1	AC Immune SA Investor Presentation
99.2	Informational Poster

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/ Martin Velasco
Name: Martin Velasco
Title: Chairman

Date: December 15, 2016

EXHIBIT INDEX

Exhibit Number	Description
99.1	AC Immune SA Investor Presentation
99.2	Informational Poster

Crenezumab

Phase I Study

AC Immune

Crenezumab Background

Crenezumab (RO5490245) is a humanized anti-amyloid β monoclonal IgG4 antibody

- Binds multiple forms of amyloïdes β (monomers, oligomers, fibrils, plaques) with high affinity for oligomers
- IgG4 hypothesized to reduce risk of amyloid-related imaging abnormalities (ARIA) enabling higher doses
- Clinical studies conducted in sporadic and autosomal dominant Alzheimer's disease.

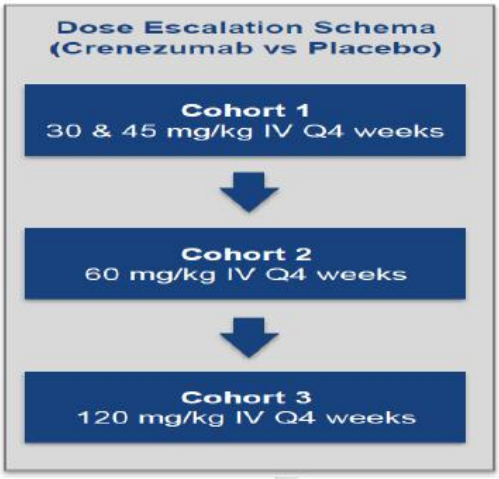
Crenezumab

Phase Ib Study GN29632

Phase Ib study GN29632 serves to inform the safety and tolerability of crenezumab delivered at higher doses

Phase Ib Dosing

- Crenezumab 30, 45, 60 and 120 mg/kg IV Q4 weeks vs placebo



Study Population

- MMSE 18-28
- 50-90 years
- Probable mild to moderate AD by NINCDS-ADRDA criteria
- CDR global score 0.5 to 1
- [18F]-florbetapir PET scan positive for cerebral amyloid
- 50% of the patients enrolled in each dose level are ApoE4 positive

Phase Ib Design

a Cross-over to crenezumab for patients on placebo
b Randomization ratio 5:1 crenezumab:placebo

IV=intravenous; MRI=magnetic resonance imaging

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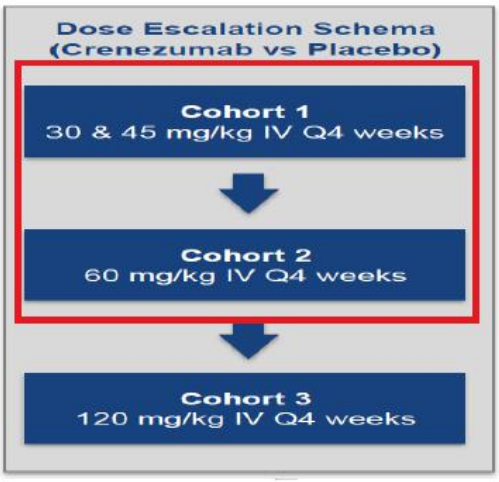

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Crenezumab

Phase Ib Study Assessments

- Safety
  - Brain MRI (central read)
  - Nature, frequency, severity of adverse events (Aes) and serious AEs
  - Examinations: Physical and neurologic, vital signs
  - Laboratory tests (blood and urine)
  - ECG assessments
- PK: Serum crenezumab concentration (peak and through) obtained on dosing days
- Exploratory:
  - Imaging and plasma biomarkers
  - Clinical scales (ADAS-Cog13, ADCS-ADL, CDR-SB)

ADAS-Cog=Alzheimer's disease assessment scale-cognitive; ADCS-ADL=Alzheimer's disease cooperative study-activities of daily living; AE=adverse event; CDR-SB=clinical dementia ration-sum of boxes; ECG=electrocardiogram; MMSE=Mini-Mental State Examination; MRI=magnetic resonance imaging; PK=pharmacokinetics.

Crenezumab

Phase Ib Safety Results: Patient baseline characteristics and treatment exposure

Characteristics	Cohort 1 (n=26)	Cohort 2 (n=26)
▪ Age, mean (range)	73.5 (54-82)	72.7 (51-87)
▪ Male, n (%)	14 (54%)	15 (58%)
▪ ApoE status, n (%)		
E2/E3	1 (2%)	0
E3/E3	5 (19%)	5 (19%)
E3/E4	17 (65%)	18 (69%)
E4/E4	3 (12%)	3 (12%)
▪ Baseline MMSE, mean (range)	22.4 (18-28)	22.7 (18-29)
▪ Medium duration of exposure	52.1 weeks (4-64)	32.1 weeks (12-40)

ApoE=apolipoprotein E; MMSE=Mini-Mental State Examination.

Crenezumab

Phase Ib Safety Results: Cohort I (30 & 45 mg/kg)

Adverse event (AE) summary, n (%)	Cohort 1 (n=26)
▪ Total number of patients with at least one AE	21 (81%)
▪ AE related to study treatment (as assessed by investigator)	7 (27%)
▪ AE Grade ≥ 3 (severe, life-threatening or resulting in death)[1]	1 (4%)
▪ Serious AE[2]	1 (4%)
▪ Adverse events of special interest[3]	4 (15%)
▪ Treatment withdrawal due to AE[4]	1 (4%)
• No dose-limiting toxicities	
• No deaths	
Common and Selected AEs, n (%)	**Cohort 1 (n=26)**
▪ Headache	4 (15%)
▪ Cerebral microhemorrhage	4 (15%)
▪ Anxiety	5 (19%)
▪ Fatigue	0
▪ Muscle spams	3 (12%)
▪ Infusion-related reactions[6]	3 (12)

1. Common Terminology Criteria for Adverse Events (CTCAE) v4.0 2. Serious AEs Cohort 1: Malignant melanoma (1 patient) 3. AESI: Cohort I: Cerebral microhemorrhage (3 patients); Cerebrallar microhemorrhage (1 patient) 4. Discontinuations due to adverse event. Cohort 1: Malignant melanoma diagnosis 5. Includes one subject with reported term of cerebrelar microhemorrhage 6. Per protocol, infusion-related reactions defined as adverse events occurring during or within 24 hours after study drug administration and judged to be related to study drug.

Crenezumab

Phase Ib Safety Results: Cohort II (60 mg/kg)

Adverse event (AE) summary, n (%)	Cohort 2 (n=26)
Total number of patients with at least one AE	20 (77%)
AE related to study treatment (as assessed by investigator)	4 (15%)[1]
AE Grade ≥ 3 (severe, life-threatening or resulting in death)	1 (4%)
Serious AE[2]	2 (8%)
	2 (8%)
Adverse events of special interest[3]	2 (8%)
Treatment withdrawal due to AE[4]	

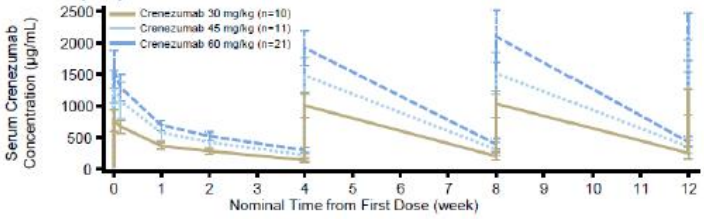
Common and Selected AEs, n (%)	Cohort 2 (n=26)
Headache	2 (8%)
Cerebral microhemorrhage	2 (8%)
Anxiety	1 (4%)
Fatigue	4 (15%)
Muscle spams	0
Infusion-related reactions[5]	2 (12%)

1. Common Terminology Criteria for Adverse Events (CTCAE) v4.0 2. Serious AEs Cohort 2: Accidental overdose, pneumonia and subdural hematoma (1 patient), atypical chest pain (1 patient) 3. AESI: Cohort 2: Cerebral microhemorrhage (2 patients), pneumonia (1 patient) 4. Discontinuation due to adverse event: Cohort 2: Confusional state, atrial fibrilation (both non serious events) 5. Per protocol, infusion-related reactions defined as adverse events occurring during or within 24 hours after study drug administration and judged to be related to study drug.

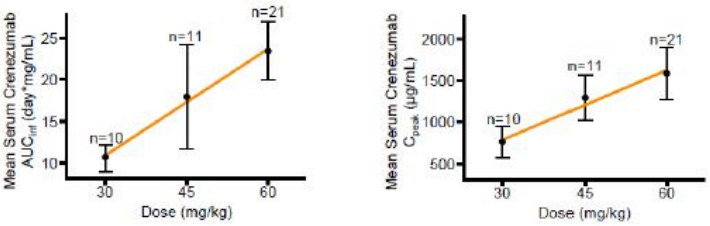
Crenezumab

Phase Ib Serum Pharmacokinetics

Mean (SD) Serum Crenezumab Concentrations



Serum exposure (AUC_{inf}, C_{peak}) vs Dose



- Serum PK of crenezumab exhibits biphasic disposition with a long half-life (approx. 25 days)
  - Consistent with other LgG antibodies that exhibit linear kinetics[2]
- Steady-state concentrations are achieved within 13 weeks, with modest accumulation
- Exposure increase proportional to dose
- The observed serum concentration at 60 mg/kgIVQ4W, agrees with projected exposure based on historical data
  - i.e. four-fold higher than following the 15 mg/kg dose

1. Deng R. et al. Mabs 2011; 31: 61-66
2. Projection was based on a two-compartment linear population PK model developed on PK data from the Phase I (ABE4427g and ABE4662g) and Phase II studies (ABE4869g and ABE4955g).

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Crenezumab

Phase Ib Study Conclusions: Analysis of Cohort I and II

- Safety of crenezumab for doses of 30, 45 and 60 mg/kg
  - Majority of Aes were low-grade and non-serious
  - No dose-limiting toxicities
  - **No investigator-assessed drug-related serious adverse events**
  - ARIA
    - **No events of ARIA-E**
    - Few patients (6 of 52) had ARIA-H; all were asymtomatic, and did not result in treatment disontinuation

- Crenezumab serum PK for doses of 30, 45, and 60 mg/kg
  - Dose-proportional up to 60 mg/kg IV Q4 weeks, and consistent with historical data
    - Serum concentrations are four-fold higher than following the 15 mg/kg dose

- Safety, tolerability, and PK profiles of crenezumab
  - Supportive of continued development at doses higher than 15 mg/kg IV Q4 weeks
  - 60 mg/kg dose implemented in the ongoing Phase III program

Crenezumab exposure-response across AD endpoints supports a higher dose for Phase 3

Dan Polhamus PhD[1], James Rogers PhD[2], Robert Paul MD[2], Smita Kshirsagar PhD[2], Srikumar Sahasranaman PhD[2], Jin Y Jin PhD[2], Angelica L Quartino PhD[2]

(1) Metrum Research Group, Tariffville, CT. USA

(2) Genentech, Inc., San Francisco, CA. USA

Background

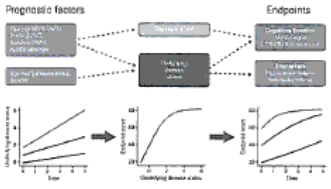
- Crenezumab (MABT5102A, RO5490258) is a humanized anti-Aβ monoclonal IgG4 antibody in development for the treatment of AD.
- Crenezumab binds to multiple forms of Aβ (monomers, oligomers, fibers/plaques)—notably with high affinity for Aβ oligomers—and is hypothesized to reduce oligomer neurotoxicity and accumulation.[1,2]
- Crenezumab was tested in two Phase 2 trials (ABBY, NCT01343966; BLAZE, NCT01397578)[3,4] conducted in a mild-to-moderate AD population, evaluating a high 15 mg/kg IV Q4W dose and a low 300 mg Q2W SC dose. In ABBY, there was no enrichment for presence of Aβ pathology.
- The Phase 2 studies demonstrated a consistent treatment effect on cognition with the 15 mg/kg IV dose for the milder population (MMSE ≥ 22) in a post hoc analysis, while the low 300 mg Q2W SC dose level lacked a consistent treatment effect, suggesting that higher doses in more mildly affected patients may be associated with greater efficacy signals. In both Phase 2 studies, crenezumab was generally well tolerated, with only one case of ARIA-E across both studies, indicating that higher doses could be investigated further.
- Safety, tolerability and PK of higher IV doses in mild-to-moderate AD is currently being investigated in a Phase Ib study (NCT02353598). Blinded safety and PK data from 30, 45 and 60 mg/kg IV Q4W are presented.[5]

Here we present the exposure–response analysis supporting a higher dose of 60 mg/kg IV Q4W in the ongoing Phase 3 study CREAD (NCT02670083).[6]

Methods

- Using data from the ADNI study,[7] a disease progression model for mild-to-moderate AD was developed. The model adequately described the longitudinal changes of the clinical endpoints (ADAS-Cog12 and CDR-SB) and the biomarkers (hippocampal and ventricular volumetric MRI) simultaneously for subjects in the ADNI study.[8-11] The model included analysis of key baseline characteristics that are thought to influence disease progression (Figure 1).
- The disease progression model was extended to describe the drug effect of crenezumab on each clinical endpoint separately, as a hyperbolic function (E_{max} model). No drug effect was seen on the volumetric MRI (Roche data on file). Therefore, no MRI data are shown here.
- The model was used to analyze the Phase 2 studies (ABBY and BLAZE) simultaneously. However, to account for the staggered enrollment and within-cohort randomization to crenezumab or placebo, factors for study and route were included for two parameters in the model: disease progression and maximum drug effect (E_{max}). This allows for a separate estimation of the placebo response and drug effect for each cohort.

Figure 1. Schematic of the AD disease progression model.[8,9]

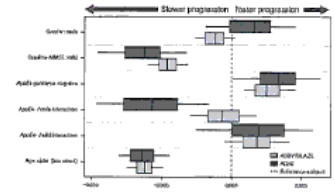


- Clinical trial simulations representing 1000 replications of the Phase 3 study design were conducted across a range of doses, assessing the likelihood of achieving a relative reduction in disease progression in treated patients compared with placebo as measured by ADAS-Cog12 and CDR-SB.

Results

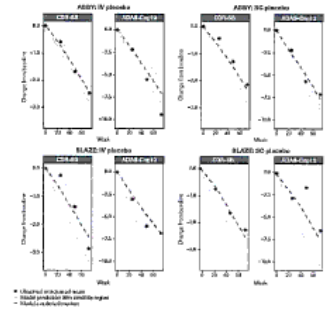
- Key baseline characteristics that influence disease progression were consistent between the ADNI population and the ABBY/BLAZE population (Figure 2). The analysis showed faster disease progression in:
  - moderate AD (lower baseline MMSE)
  - *ApoE4*-positive genotype
  - female gender
  - early onset (age).

Figure 2. Analysis of influence of key baseline patient characteristic on rate of disease progression.



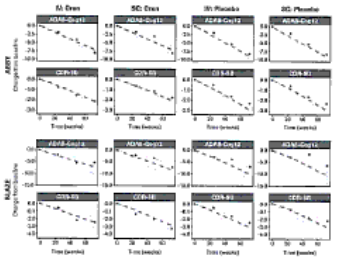
- Simulations using the disease model developed on ADNI data alone demonstrated that the observed placebo decline in both cohorts of ABBY and BLAZE is largely consistent with expectations (Figure 3).

Figure 3. Observed cognitive decline as measured by CDR-SB and ADAS-Cog12 compared with disease progression model simulations for ABBY and BLAZE placebo cohorts.



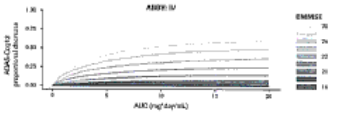
- Validation of the extended disease progression model fitted to Phase 2 data demonstrated that the model replicated the Phase 2 longitudinal data accurately. The model is therefore fit for purpose for simulation of the disease progression and crenezumab treatment effect in the population of interest (milder AD population, baseline MMSE 22–26; Figure 4).

Figure 4. Comparison of observed cognitive decline as measured by CDR-SB and ADAS-Cog12 and extended disease progression model simulations for ABBY and BLAZE Phase 2 studies.



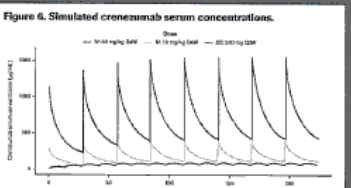
- A correlation was observed between crenezumab exposure (AUC at steady state) and treatment effect (ADAS-Cog12 and CDR-SB), which appeared to reach an asymptote at the projected exposures of the dose of 60 mg/kg Q4W. The treatment effect of crenezumab was associated with high baseline MMSE, suggesting better treatment effect earlier in AD (Figure 5). In addition, *ApoE4* genotype had a minor influence on the treatment effect (Roche data on file).

Figure 5. Association between ADAS-Cog12 and crenezumab exposure given the patient baseline MMSE.



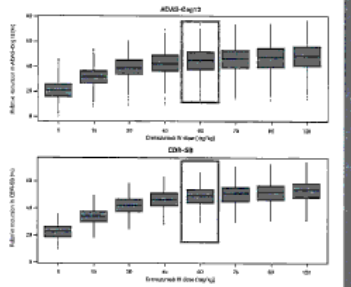
- A population PK model was developed using serum crenezumab concentration data from the Phase 1 (ABE4427g and ABE4662g) and Phase 2 studies (ABE4889g and ABE4955g). Model-simulated serum concentrations for a 15 mg/kg IV Q4W (gray), 300 mg SC Q2W (red), and 60 mg/kg IV Q4W shown in Figure 6. An IV dose of 15 mg/kg Q4W provided ~1.5-, 2.5- and 5-fold higher exposure (C_{min}, AUC and C_{max} at steady state) compared with the 300 mg Q2W SC dose. Using the model, a dose of 60 mg/kg IV Q4W is predicted to achieve 4-fold higher serum crenezumab concentrations compared with 15 mg/kg Q4W (Figure 6).

Figure 6. Simulated crenezumab serum concentrations.



- Clinical trial simulations of the Phase 3 study design using the drug-disease model showed that a 4-fold increase in dose to 60 mg/kg Q4W is predicted to achieve a 41% relative reduction on ADAS-Cog12, and 44% on the CDR-SB in the milder AD population (baseline MMSE 22–26) (Figure 7). However, since Phase 2 data were used as the training set for the model, uncertainty in estimated efficacy is greater where exposure is outside the levels observed in Phase 2.

Figure 7. Dose–response of crenezumab dose IV Q4W on cognitive endpoints (ADAS-Cog12 and CDR-SB) in patients with mild AD (baseline MMSE 22–26) based on clinical trial simulations using the drug-disease model.



Conclusions

- A drug-disease progression model of both crenezumab and placebo cohorts adequately summarized the longitudinal decline in ADAS-Cog12 and CDR-SB in mild-to-moderate patients in the crenezumab Phase 2 studies (ABBY/BLAZE).
- A 60 mg/kg IV Q4W dose was selected for Phase 3, supported by clinical trial simulations using the drug-disease model, which predicts greater treatment effect at higher exposures in patients with mild AD.

References

1. Adolfsson O, *et al. J Neurosci* 2012; 32:9677-89.
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Abbreviations

Aβ, amyloid-beta; AD, Alzheimer's disease; ADAS-Cog12, Alzheimer's Disease Assessment Scale Cognition subscale 12; ADNI, Alzheimer's Disease Neuroimaging Initiative; ApoE4, apolipoprotein E4; ARIA-E, amyloid-related imaging abnormalities-edema; AUC, area under the curve; BMMSE, baseline Mini-Mental State Examination; CDR-SB, Clinical Dementia Rating-Sum of Boxes; C_{max}, maximum concentration; C_{min}, minimum concentration; Cren, crenezumab; E_{max}, maximum drug effect; IgG4, immunoglobulin G4; IV, intravenous; MMSE, Mini-Mental State Examination; MRI, magnetic resonance imaging; PK, pharmacokinetic; Q2W, dose every 2 weeks; Q4W, dose every 4 weeks; SC, subcutaneous.

Acknowledgments

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