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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 August, 2022

**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 Offices)

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

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**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

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Name: Andrea Pfeifer

Title: Chief Executive Officer

Date: August 2, 2022

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## EXHIBIT INDEX

Exhibit Number	Description
99.1	Press Release dated August 2, 2022

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## Detailed data from the phase II Crenezumab Alzheimer's Prevention Initiative Study in Autosomal Dominant Alzheimer's Disease Presented at AAIC

- Crenezumab was safe and well tolerated with no cases of ARIA-E observed during the up to eight-year study
- As previously reported, numerical differences favoring crenezumab over placebo were confirmed across the co-primary, multiple secondary and exploratory endpoints, none statistically significant
- Demographic and baseline biomarker data indicate a confluence of factors which may have caused the study to have lower than expected statistical power
- All mutation carriers may continue to receive crenezumab while the data are further analyzed

**Lausanne, Switzerland, August 2, 2022** -- AC Immune SA (Nasdaq: ACIU), a clinical-stage biopharmaceutical company pioneering precision medicine for neurodegenerative diseases, today announced that its partners presented the first quantitative data from the Alzheimer's Prevention Initiative (API) Autosomal Dominant Alzheimer's Disease (ADAD) Colombia Trial during a *Focused Topic Session* at the Alzheimer's Association International Conference (AAIC). The study evaluated the potential of the anti-amyloid beta antibody crenezumab to slow or prevent Alzheimer's disease in cognitively unimpaired people who carry a specific genetic mutation which causes early-onset Alzheimer's disease.

As previously reported on [June 16, 2022](#), the trial did not meet its co-primary endpoints (API ADAD composite cognitive total score and the Free and Cued Selective Reminding Test Cueing Index). Numerical differences favoring crenezumab over placebo were observed across both of these co-primary endpoints; statistical significance was not reached. Additional clinical and biomarker measures also showed numerical differences favoring crenezumab over placebo that did not reach statistical significance.

**Dr. Andrea Pfeifer, CEO of AC Immune commented:** "When viewed in totality, we believe the results of the API ADAD study are encouraging and important for the Alzheimer's field. Given the consistency of results favoring crenezumab over placebo, and the slower than expected decline in the placebo group as measured by the API ADAD composite score, the numerical differences seen in clinical and biomarker analyses provide intriguing evidence of potential activity impacting the pathogenic pathway. These results, together with the trial's remarkable safety data showing no cases of ARIA-E over the eight-year trial, support the decision to allow for the continued treatment of mutation carriers with crenezumab while further analysis of the data is ongoing. We would like to extend our sincere gratitude to all those who participated in this landmark trial, which will undoubtedly shape the future of Alzheimer's disease drug development."

Clinical endpoints showed the following relative changes in annualized scores compared to placebo, in all cases favoring crenezumab though in all cases not statistically significant:

- Cognitive test scores: API ADAD composite 22.9% (p=0.43); FCSRT 19.9% (p=0.16); RBANS total score 43.8% (p=0.55)
- Clinical/function: Time to MCI/dementia due to AD 20.8% (p=0.48); time to non-Zero in CDR-GS 8.1% (p=0.76); CDR Sum of Boxes 8.8% (p=0.64)

Biomarker results also favored crenezumab with the following relative changes compared to placebo, all favoring crenezumab though not statistically significant:

- PET measures: A $\beta$  PET SUVR 3.6% (p=0.69); Tau-PET SUVR 51.1% (p=0.20); FDG PET SUVR 18.1% (p=0.25)
- CSF measures: t-tau 28.7% (p=0.53); p-tau-181 37.4% (p=0.28); NfL 18.2% (p=0.46)

The API ADAD trial enrolled 252 people who are members of the world's largest extended family with ADAD in Colombia. Two-thirds of participants carried the Presenilin 1 E280A mutation which typically causes cognitive impairment due to Alzheimer's disease around age 44. Approximately half of enrolled mutation carriers were negative for amyloid beta at enrolment. Participants were randomised to receive crenezumab or placebo for a planned duration of five to eight years. During the trial, the dose of crenezumab was increased more than seven-fold as knowledge about potential treatment approaches for Alzheimer's disease evolved. The average age of mutation carriers enrolled in the trial was 37.

Limitations of the API ADAD study noted in the AAIC presentation included limited statistical power to determine whether treatment with crenezumab at the optimal dose, which was received by most subjects for only about two out of five years, would have a clinical benefit. Demographic and baseline data indicate the study's lower-than-expected statistical power may have been due to a confluence of factors, including a study population that was on average younger and at an earlier preclinical Alzheimer's disease stage than expected.

**Dr Johannes Streffer, Chief Medical Officer of AC Immune added:** "The API ADAD study has provided a wealth of data that will be instrumental in gaining a better understanding of Alzheimer's disease progression. Though not statistically significant, numerical differences favoring crenezumab on clinical endpoints were importantly supported by consistent numerical differences observed on biomarker measures. We found the relative 51.1% reduction in accumulation of tau in the entorhinal cortex to be of particular interest, despite the lack of statistical significance, as prior clinical data have shown correlations between brain Tau levels and disease progression."

### **About the Alzheimer's Prevention Initiative and the API ADAD (Colombia) Trial**

The Alzheimer's Prevention Initiative (API) is an international collaborative formed in 2009 to launch a new era of Alzheimer's prevention research. Led by the Banner Alzheimer's Institute, the API conducts prevention trials in cognitively healthy people at increased risk for Alzheimer's disease. API continues to establish brain imaging, fluid biomarker and cognitive endpoints needed to rapidly test promising prevention therapies. It also leads participant recruitment registries to accelerate enrolment into Alzheimer's-focused studies. API is intended to provide the scientific means, accelerated approval pathway and enrolment resources needed to evaluate the range of promising Alzheimer's prevention therapies and find ones that work without losing another generation.

First proposed by investigators from the Banner Alzheimer's Institute (BAI) the API ADAD trial (NCT01998841) was a prospective, randomised, double-blind, placebo-controlled, parallel-group label enabling Phase II study of the efficacy of crenezumab versus placebo in cognitively unimpaired individuals who have no clinical symptoms of Alzheimer's disease and carry the PSEN1 E280A autosomal dominant mutation. Participants who are mutation carriers were randomised in a 1:1 ratio to receive either crenezumab or placebo for at least 260 weeks. Crenezumab was initially administered at 300 mg subcutaneously every two weeks. Dosing was amended in 2015 to 720 mg subcutaneously every two weeks and in 2019 the option to increase the dose to 60 mg/kg, delivered intravenously every four weeks, was offered to participants. A cohort of participants (non-mutation carriers) were also enrolled and dosed solely on placebo.

The trial, which was supported by National Institute on Aging (NIA) generous philanthropic contributions to Banner Alzheimer's Foundation and Roche, was the first NIH-supported prevention trial of an experimental prevention therapy in cognitively unimpaired persons at known risk for the disease.

For more information, go to <https://alzheimerspreventioninitiative.com/>.

## About Autosomal Dominant Alzheimer's Disease

Autosomal Dominant Alzheimer's Disease (ADAD; also known as familial AD or dominantly-inherited AD [DIAD]) is a rare, inherited form of Alzheimer's disease caused by single gene mutations in the APP, PSEN1 or PSEN2 genes. Less than 1% of all Alzheimer's cases worldwide are thought to be caused by genetic mutations. It usually has a much earlier onset than the more common sporadic Alzheimer's disease, with symptoms developing in people in their 30s to 60s. If an individual has one of these mutations they are nearly certain to develop Alzheimer's and there is a 50% chance they will pass it on to each of their children.

## About the PSEN1 E280A mutation and the Antioquia kindreds

The PSEN1 E280A mutation virtually guarantees that carriers will develop Alzheimer's at the average age of 44 and dementia at the average age of 49. The Colombian PSEN1 E280A kindred are the world's largest extended family with ADAD, with ~6,000 family members and ~1,200 with the mutation.

The API ADAD trial was conducted in collaboration with neurologist Francisco Lopera and his team, Grupo de Neurociencias de Antioquia (GNA), at the University of Antioquia in Medellín, Colombia. Dr Lopera followed the kindred for three decades prior to the start of the trial and has established a close relationship with many members.

## About crenezumab

Crenezumab is a humanised monoclonal antibody, an investigational treatment designed to slow Alzheimer's disease progression by neutralising neurotoxic beta-amyloid oligomers. It was designed by AC Immune to be a conformation-specific monoclonal antibody targeting multiple forms of misfolded Abeta. Crenezumab has an antibody backbone (IgG4) designed to minimise the inflammatory response in the brain, which may result in a lower risk of certain MRI (magnetic resonance imaging) abnormalities known as ARIA (Amyloid-Related Imaging Abnormalities). The investigational medicine is being developed by Genentech and is part of a collaboration with AC Immune SA.

## About AC Immune SA

AC Immune SA is a clinical-stage biopharmaceutical company that aims to become a global leader in precision medicine for neurodegenerative diseases, including Alzheimer's disease, Parkinson's disease, and NeuroOrphan indications driven by misfolded proteins. The Company's two clinically validated technology platforms, SupraAntigen<sup>®</sup> and Morphomer<sup>®</sup>, fuel its broad and diversified pipeline of first- and best-in-class assets, which currently features ten therapeutic and three diagnostic candidates, six of which are currently in phase 2 clinical trials. AC Immune has a strong track record of securing strategic partnerships with leading global pharmaceutical companies including Genentech, a member of the Roche Group, Eli Lilly and Company, and Janssen Pharmaceuticals, Inc., resulting in substantial non-dilutive funding to advance its proprietary programs and >\$3 billion in potential milestone payments.

SupraAntigen<sup>®</sup> is a registered trademark of AC Immune SA in the following territories: AU, EU, CH, GB, JP, RU and SG. Morphomer<sup>®</sup> is a registered trademark of AC Immune SA in CN, CH, GB, JP, NO and RU.

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**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. These include: the impact of Covid-19 on our business, suppliers, patients and employees and any other impact of Covid-19. 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.