PRESS RELEASE

AC Immune Announces Late-Breaker Presentation by Genentech at CTAD on Phase 2 Lauriet Study of Semorinemab in Mild-to-Moderate Alzheimer's Disease

Analysis of the broader mITT population is consistent with the previously reported success in meeting one of the two co-primary endpoints (ADAS-Cog11) with statistically significant reduction in the rate of cognitive decline vs. placebo

Benefit of semorinemab on ADAS-Cog11 in all prespecified subgroups was consistent with the treatment effect in the overall cohort, regardless of disease severity, baseline Tau load, and ApoE carrier status

Benefit on cognition was driven primarily by the memory domain subcomponent of ADAS-Cog11, a core feature of AD

Further analyses evaluating semorinemab's effects on cerebrospinal fluid (CSF) biomarkers are ongoing as is the open label portion of the study

Lausanne, Switzerland, November 10, 2021 – AC Immune SA (NASDAQ: ACIU), a clinical-stage biopharmaceutical company pioneering precision medicine for neurodegenerative diseases, today announced that Genentech, a member of the Roche Group, presented the full topline data from Lauriet, a placebo-controlled Phase 2 study evaluating the safety and efficacy of the investigational anti-Tau monoclonal antibody, semorinemab, in mild-to-moderate Alzheimer's disease (AD) during a late-breaking session at the 14th Clinical Trials on Alzheimer's Disease (CTAD) Conference.

Previously announced topline results showed that the trial met one of its two co-primary endpoints by demonstrating a statistically significant 43.6% reduction in the rate of cognitive decline with semorinemab compared to placebo ($p<0.0025$), as measured by the Alzheimer's Disease Assessment Scale, Cognitive Subscale, 11-item Version (ADAS-Cog11) at week 49 in a prespecified modified intent to treat (mITT) population. The second co-primary endpoint, Alzheimer's Disease Cooperative Study-Activities of Daily Living (ADCS-ADL), was not met nor the secondary efficacy endpoints including the Mini-Mental State Examination (MMSE) or the Clinical Dementia Rating-Sum of Boxes (CDR-SB). The prespecified mITT population consisted of 204 patients with mild-to-moderate AD who had missed $\leq$1 dose of study drug.

The full topline data presented at CTAD show that semorinemab's treatment effect on ADAS-Cog11 was confirmed in a larger (n=241) mITT population that included all trial participants who had received $\geq$1 dose of study drug and had at least one post-baseline ADAS-Cog11 assessment. Data from this population show a 42.2% reduction in the rate of cognitive decline with semorinemab compared to placebo ($p=0.0008$), as measured by ADAS-Cog11. This treatment effect was observed consistently in prespecified subgroups based on disease severity, baseline Tau load, and ApoE carrier status. ADAS-Cog11 domain analyses show that semorinemab's treatment effect was

driven predominantly by the memory domain, which is a core feature of AD. As previously announced, no significant treatment effect was observed on the trial's other co-primary endpoint or the secondary endpoints.

Biomarker analyses reported during the CTAD presentation included Tau positron emission tomography (PET) scans and plasma Tau levels. The biomarker dataset from cerebrospinal fluid (CSF) samples is not available at this time. There was no identifiable treatment effect on global or regional Tau distribution as assessed by PET analysis. Analysis of plasma Tau showed a pronounced increase of plasma Tau levels with semorinemab treatment, which is suggestive of peripheral tau binding and similar to previous studies. Levels of semorinemab in plasma were in the expected range as was the ratio of the level of semorinemab in the CSF to that in plasma (mean 0.29%).

Safety data from the trial confirmed that semorinemab is well tolerated with an acceptable safety profile, consistent with previous data. Adverse events and serious adverse events were well balanced between the two treatment arms, and there were no unanticipated safety signals. The trial's open label extension remains ongoing.

Prof. Andrea Pfeifer, CEO of AC Immune SA, commented: "We are pleased that the data presented at CTAD confirm Lauriet's remarkable findings, which provide the first evidence of therapeutic impact on cognition for an anti-Tau monoclonal antibody in mild-to-moderate AD, by showing a statistically significant slowing of the rate of cognitive decline. We thus remain encouraged by the Lauriet data, while still being cautious about what it may mean for patients, given the lack of effect on functional endpoints. AD is a slow progressing chronic disease, therefore, we look forward to learning about semorinemab's longer-term effects through the ongoing open-label extension, which is being conducted by our partners at Genentech."

Prof. Johannes Streffer, CMO of AC Immune SA, commented: "The finding of significant slowing of cognitive loss assessed by ADAS-Cog 11 in the Lauriet study is an important result and one that should be further analyzed. It is the first example of a monoclonal anti-Tau antibody slowing cognitive decline in mild-to-moderate AD, adding to an emerging dataset that provides a strong scientific rationale for Tau as a valid target in Alzheimer's disease."

About the Lauriet study

Lauriet is a double-blind, placebo-controlled, randomized Phase 2 trial assessing semorinemab, an investigational anti-Tau monoclonal antibody, compared to placebo in 272 adult participants with mild-to-moderate AD across 43 study centers globally. The co-primary endpoints of the study evaluated the change from baseline at week 49 in cognition as measured by the Alzheimer's Disease Assessment Scale, Cognitive Subscale, 11-Item Version (ADAS-Cog11) and the change from baseline in activities of daily living as measured by the Alzheimer's Disease Cooperative Study-Activities of Daily Living (ADCS-ADL) scale. Secondary endpoints evaluated cognitive and functional measures including changes from baseline as measured by the Clinical Dementia Rating-Sum of Boxes (CDR-SB) and the Mini-Mental State Examination (MMSE). The Lauriet open label extension is ongoing. For more information, visit ClinicalTrials.gov (NCT03828747).

About semorinemab

Semorinemab is an investigational monoclonal anti-tau antibody that targets the N-terminal portion of the Tau protein, and is designed to bind to Tau and slow its spread between neurons. In tauopathies such as AD, Tau misfolds and forms tangles, which cause cell damage and ultimately neuronal death. It is hypothesized that abnormal Tau protein then spreads between neurons, gradually involving more areas of the brain, and leading to clinical disease progression. Tau-targeting antibody therapies are designed to slow or stop this process of tau spread. Semorinemab is being developed by Genentech and was identified in collaboration with AC Immune (Nasdaq: ACIU, Lausanne, Switzerland). Semorinemab has been studied in two Phase 2 studies – Lauriet in mild-to-moderate AD and Tauriel in early (prodromal-to-mild) AD, where the primary efficacy endpoint was not met.

About AC Immune SA

AC Immune SA is 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® and Morphomer®, 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 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.

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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.