

Aprea AB Names Christian S. Schade President and Chief Executive Officer

June 16, 2016

June 16, 2016 — Boston, MA, USA and Stockholm, Sweden, June 16, 2016 — Aprea AB, a privately held, clinical-stage biopharmaceutical company developing novel anticancer therapies targeting the tumor suppressor protein p53, today announced that Christian S. Schade has been named President and Chief Executive Officer. Mr. Schade, who will be based in Boston, has more than 30 years of private and public pharmaceutical and biotechnology industry experience, as well as broad corporate finance expertise from his tenure in the investment banking industry. Prior to joining Aprea, he was Chief Executive Officer of Novira Therapeutics, which was acquired in December 2015 by Johnson & Johnson in an all-cash transaction.

"I am delighted to welcome Chris to Aprea," said Bernd R. Seizinger, M.D., Ph.D., Chairman of the Board of Directors. "He is an accomplished biopharmaceutical executive with proven experience leading companies through times of change and growth. He has successfully executed major transactions, including IPOs and other financings, and has been on both sides of major mergers and acquisitions. He will be an important asset to Aprea as we continue to develop novel anticancer therapies targeting the tumor suppressor protein, p53. Furthermore, Chris has excellent credentials to build a strategic leadership team in the U.S. to complement the R&D activities of the team in Sweden."

"I am pleased to be joining Aprea at such an important time in the Company's evolution and look forward to helping to build upon its leadership position in developing anticancer therapies targeting p53 suppression," said Christian S. Schade, Aprea's new President and Chief Executive Officer. "With a lead product entering Phase II clinical testing in ovarian cancer, and a strong portfolio of earlier-stage clinical opportunities, Aprea is well-positioned for sustainable innovation in oncology and value creation for shareholders. I look forward to working with the Company's Board of Directors and dedicated employees to develop therapies which hold the potential to deliver better outcomes for cancer patients across an expanding range of indications."

Prior to joining Novira, Mr. Schade was Executive Vice President and Chief Financial Officer at Omthera, a NASDAQ-listed specialty pharmaceuticals company focused on the development and commercialization of new therapies for dyslipidemia. At Omthera, Mr. Schade was responsible for all corporate finance, accounting and business development activities, and led the sale of Omthera in July 2013 to AstraZeneca. He also was Executive Vice President and Chief Financial Officer at NYSE-listed NRG Energy, and from 2000 to 2009, he was Senior Vice President of Administration and Chief Financial Officer at Medarex, a biopharmaceutical company focused on antibody-based therapeutic products for oncology, inflammation, autoimmune disorders and infectious diseases. Mr. Schade played a pivotal role in the acquisition of Medarex by Bristol-Myers Squibb, leading the negotiations for the sale and the eventual merger-integration process of the research, development and administrative functions.

Before joining Medarex, Mr. Schade served as Managing Director at Merrill Lynch in London and held various corporate finance and capital markets positions in New York and London for both Merrill Lynch and JP Morgan Chase & Co. Mr. Schade received an MBA from the Wharton School at the University of Pennsylvania and an AB from Princeton University. Mr. Schade currently serves on the Board of Directors of Indivior PLC.

About p53 and APR-246

The p53 tumor suppressor gene is the most frequently mutated gene in human cancer, occurring in approximately 50% of all human tumors. These mutations are often associated with resistance to anticancer drugs and poor overall survival, representing a major unmet medical need in the treatment of cancer.

APR-246 has been shown to reactivate mutant p53 protein – by reconverting mutant p53 into wild-type p53 conformation and function – and thereby induce programmed cell death in human cancer cells. APR-246 has demonstrated compelling pre-clinical antitumor activity in a wide variety of solid and hematological (blood) tumors, including ovarian cancer, small cell lung cancer, esophageal cancer and AML (acute myeloid leukemia), among others. Additionally, strong synergy has been seen with both traditional anticancer agents, such as chemotherapy, as well as newer mechanism-based anticancer drugs. In addition to pre-clinical testing, a Phase I clinical study has been completed, demonstrating a favorable safety profile and both biological and clinical responses in hematological tumors with mutations in the p53 gene. APR-246 is currently in a Phase Ib/II clinical trial in patients with high-grade serous ovarian cancer. The Phase Ib part has completed. In the Phase II clinical study, Aprea will enroll

up to 400 ovarian cancer patients in Europe and the U.S. Patients will be randomized between carboplatin and pegylated liposomal doxorubicin with or without APR-246; the primary endpoint for the study is progression-free survival (PFS). The Company is also expecting to begin additional clinical studies of APR-246 in other cancer indications.

About Aprea

Aprea AB is a Stockholm, Sweden and Boston, Massachusetts-based biopharmaceutical company focused on the discovery and development of novel anticancer compounds reactivating the tumor suppressor protein, p53. The Company's lead program, APR-246, a first-in-class small molecule drug candidate, is in Phase Ib/II clinical development in ovarian cancer patients, and additional clinical studies with APR-246 in other cancer indications are planned. In March 2016, Aprea completed a EUR 46 million Series B financing with an international syndicate co-led by Versant Ventures and 5AM Ventures, with additional participation by Sectoral Asset Management, HealthCap, acting as local lead investor, and existing investor, Karolinska Development. For more information, please visit www.aprea.com.

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