



Press release

Cantargia AB  
556791-6019  
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## Cantargia's CAN10 demonstrates positive activity in systemic sclerosis models in new data presented at ACR Convergence 2022

Strongly supports therapeutic potential in one of CAN10's lead indications

**Cantargia (Cantargia AB; Nasdaq Stockholm: CANTA) today reported new preclinical data demonstrating positive effects of the IL1RAP-binding antibody CAN10 in three different *in vivo* models of systemic sclerosis, a lead indication for the drug candidate. Across the models, which mimic difficult-to-treat forms of the disease, CAN10 reduced both pathological inflammation and fibrosis in skin and lung tissue, factors associated with clinical severity in humans. CAN10 also normalized a number of biomarkers closely associated with the human disease. The data have been selected for an oral presentation at ACR Convergence 2022, held in Philadelphia, November 10-14, 2022.**

*"We are pleased to build on previous exciting data on the effects of CAN10 in a number of models of life-threatening diseases. The new results, selected for oral presentation at the prestigious ACR Convergence, demonstrate the potential of CAN10 in systemic sclerosis. Supported by the strength of the data we have presented to date, Cantargia is fully committed to advancing the development of CAN10 as a potentially game-changing therapy for systemic sclerosis, and we look forward to initiating clinical studies in 2023,"* said Göran Forsberg, CEO of Cantargia.

Systemic sclerosis is a life-threatening autoimmune disease involving inflammation and subsequent fibrosis, i.e. uncontrolled scar tissue formation, in skin and various internal organs. Current treatments for systemic sclerosis focus on symptomatic treatment rather than addressing underlying mechanisms.

The new results show potent activity of a CAN10 surrogate antibody, which reduced inflammation and fibrosis in three different preclinical *in vivo* models of systemic sclerosis. The anti-fibrotic effect was demonstrated by reduced thickening of the skin, decreased levels of collagen deposits that lead to fibrosis, and numbers of fibroblast cells that synthesize the collagen. Reduced fibrosis was also observed in the lungs. Elevated levels of IL1RAP and associated signaling molecules were detected in skin samples, compared to healthy skin. In patient skin biopsies, dysregulation of a range of other markers related to inflammation or fibrosis was also detected, and corresponding markers were normalized by the CAN10 surrogate antibody. CAN10 also reduced the release of inflammatory molecules by human skin fibroblasts in response to IL1RAP stimulation.

The data were generated in collaboration with the world-leading research group headed by Prof. Dr. Jörg Distler at the Heinrich-Heine University/Hiller Research Center Düsseldorf, Germany.

*"The new data provide strong evidence that signaling via IL1RAP regulates disease development in systemic sclerosis. The models used in our research are well-established tools for studying the disease, and CAN10 potently reduced inflammation and fibrosis in these models. These data indicate that CAN10 is a novel and promising therapeutic approach for treatment of systemic sclerosis,"* said Prof. Dr. Jörg Distler.

The results will be presented in an oral presentation by Dr. Caitríona Grönberg; details can be found below. The presentation slides will be made available on Cantargia's webpage (<https://cantargia.com/en/research-development/publications>) after the presentation.

Abstract Number: 1624

Session: Systemic Sclerosis and Related Disorders – Basic Science

Date: Sunday, November 13, 2022

Title: Blocking IL-1, IL-33 and IL-36 Signaling with the Anti-IL1RAP Antibody mCAN10 Ameliorates Inflammation and Fibrosis in Preclinical Models of Systemic Sclerosis

CAN10 strongly binds IL1RAP and simultaneously blocks the function of IL-1, IL-33 and IL-36 signaling, which can be of significant value in the treatment of autoimmune or inflammatory diseases. Cantargia is initially focusing the development of CAN10 on systemic sclerosis and myocarditis, and plans to start clinical phase I studies in early 2023.

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*This is information that Cantargia AB is obliged to make public pursuant to the EU Market Abuse Regulation. The information was submitted for publication, through the agency of the contact person set out above, at 16.00 CET on 8 November 2022.*

#### **About Cantargia**

Cantargia AB (publ), reg. no. 556791-6019, is a biotechnology company that develops antibody-based treatments for life-threatening diseases and has established a platform based on the protein IL1RAP, involved in a number of cancer forms and inflammatory diseases. Cantargia's lead drug candidate, the antibody nadunolimab, is being studied clinically in combination with chemotherapy or immune therapy, with a primary focus on non-small cell lung cancer and pancreatic cancer. Positive interim data from the combination of nadunolimab with chemotherapy indicate stronger efficacy than would be expected from chemotherapy alone. Cantargia's second program, the antibody CAN10, addresses treatment of serious autoimmune/inflammatory diseases, with initial focus on systemic sclerosis and myocarditis.

Cantargia is listed on Nasdaq Stockholm (ticker: CANTA). More information about Cantargia is available at [www.cantargia.com](http://www.cantargia.com).

#### **About CAN10**

The CAN10 antibody binds strongly to its target IL1RAP and has a unique capability to simultaneously inhibit signaling via IL-1, IL-33 and IL-36. Inhibition of these signals can be of significant value in the treatment of several inflammatory or autoimmune diseases. The initial focus of CAN10 will be on two severe diseases: myocarditis and systemic sclerosis. In preclinical in vivo models of myocarditis, a CAN10 surrogate antibody significantly reduced the development of inflammation and fibrosis, and significantly counteracted the deterioration of the cardiac function. CAN10 also inhibited disease development in models of systemic sclerosis, peritonitis, psoriasis and psoriatic arthritis. CAN10 is currently in late-stage preclinical development and the first clinical trial is expected to begin in early 2023.