PRESS RELEASE
14 March 2024 17:31:00 CET

Cantargia presents new human data further supporting CAN10 in the treatment of systemic sclerosis

Cantargia (Cantargia AB; Nasdaq Stockholm: CANTA) today reported new data highlighting the potential of the CAN10 antibody, currently in phase I clinical development, as therapy in systemic sclerosis. All signaling systems targeted by CAN10 (IL-1, IL-33 and IL-36) were found to be highly upregulated in patient skin biopsies. These cytokines induce fibrosis in skin fibroblasts isolated from patients, and this manifestation of disease was blocked by CAN10. In mouse models, CAN10 reversed the aberrant expression of several genes involved in the systemic sclerosis pathogenesis in humans. The data is presented at the Systemic Sclerosis World Congress in Prague 14-16 March 2024.

"We are very excited about the progress in the CAN10 program which is quickly advancing from the ongoing healthy volunteer investigations towards studies in patients. The new data provides important information further increasing our confidence in CAN10 as a future therapy of inflammatory diseases" said Göran Forsberg, CEO of Cantargia.

CAN10 is currently in phase I clinical development with transition from healthy volunteers to patients planned for Q3, 2024. One of the lead indications, systemic sclerosis, is a life-threatening autoimmune disease resulting in fibrosis in the skin, lung, and other internal organs. Current treatments focus on symptomatic treatment rather than addressing underlying disease mechanisms. It is estimated that in the US, approximately 100,000 patients suffer from the disease. CAN10 has orphan drug status in the US for treatment of systemic sclerosis.

The new results demonstrate that both the target for CAN10, IL1RAP, and the IL1RAP-dependent signaling molecules IL-1, IL-33 and IL-36, are upregulated in skin from systemic sclerosis patients. This disease develops as fibroblasts promote the formation of an excessive buildup of connective tissue in skin and internal organs, so-called fibrosis. The results show that IL-1, IL-33 and IL-36 collagen production on skin fibroblasts from systemic sclerosis patients, leading to fibrosis. These disease-associated mechanisms can be reduced by CAN10.

These new data strengthen previously reported beneficial effects of IL1RAP-blockade in three mouse models of systemic sclerosis, where treatment with a mouse surrogate of CAN10 reduced both skin and lung fibrosis. Additional gene expression analysis of mouse systemic sclerosis skin showed decreased expression of several key disease-related profibrotic factors in the skin by CAN10. In summary, the data show that CAN10 targets central processes important for systemic sclerosis and that CAN10 provides an opportunity to treat underserved systemic sclerosis patients.

These data were generated in collaboration with a world-leading research group headed by Prof. Dr. Jörg Distler at the Heinrich-Heine University, Düsseldorf, Germany and will be presented as a poster at the 8th Systemic Sclerosis World Congress March 14-16, 2024 in Prague, Czech Republic. The poster is now also available at Cantargia's web page **https://cantargia.com/en/research-development/publications**.

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Title: Combined blockade of IL-1, IL-33 and IL-36 signaling by targeting IL1RAP ameliorates skin and lung fibrosis in preclinical models of systemic sclerosis
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Poster Number: 317

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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. The main program, the antibody nadunolimab (CAN04), is being studied clinically primarily in combination with chemotherapy with a focus on pancreatic cancer, non-small cell lung cancer and triple-negative breast cancer. Positive interim data for the combinations indicate stronger efficacy than would be expected from chemotherapy alone. Cantargia's second development program, the antibody CAN10, blocks signaling via IL1RAP in a different manner than nadunolimab and 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**.

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. The CAN10 surrogate also inhibited disease development in models of systemic sclerosis, psoriasis, psoriatic arthritis, atherosclerosis and peritonitis. A clinical phase I study, investigating CAN10 in healthy volunteers and psoriasis patients, is ongoing. Up to 80 subjects may be included in the trial, the first clinical data set shows good safety. Additional data from the trial are expected continuously during 2024.

Attachments
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