

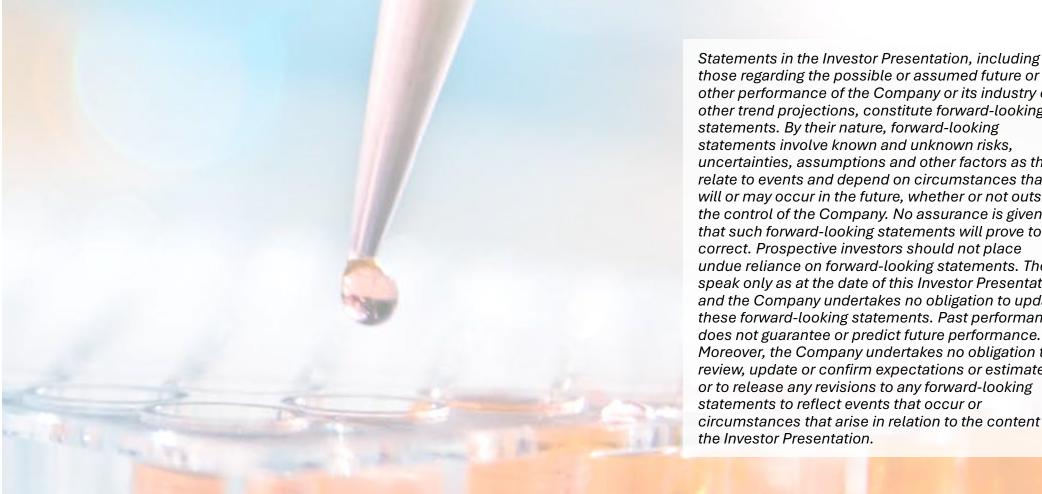
Blocking the **RIGHT** signals to reduce disease severity

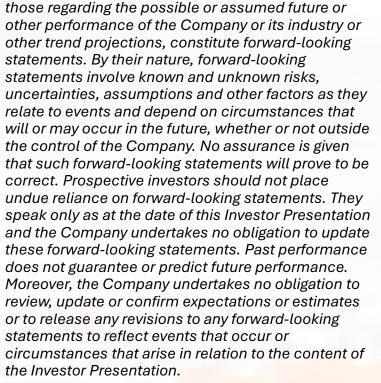
Corporate Presentation

May 2025

NASDAQ STOCKHOLM MAIN LIST (CANTA.ST)

Safe Harbor Statement







Cantargia – Global leader in IL1RAP antibody development



NOVEL IL1RAP ANTIBODIES: BROADLY APPLICABLE TO TREAT INFLAMMATORY DISEASES & CANCER

- IL1RAP signaling drives various autoimmune and inflammatory diseases
- IL1RAP elevated in most solid and liquid tumors

CAN10: OPPORTUNITY IN AUTOIMMUNITY/INFLAMMATION

- CAN10 delivers a broadly applicable and differentiated mechanism for treatment in inflammatory diseases
- Positive Phase 1 PK/PD data supports Q4W dosing in Phase 2
- Prioritized development in Hidradenitis Suppurativa (HS) with Atopic Dermatitis (AD) as second indication

NADUNOLIMAB: CLEAR ACTIVITY SIGNALS IN CANCER THERAPY WITH UPCOMING CATALYSTS

- Randomized Phase 2 trial in TNBC fully enrolled (Initial data mid-2025); IL1RAP diagnostic in development for PDAC
- Strong clinical results in PDAC and NSCLC, and promising initial results in TNBC; >300 patients treated
- Broadly applicable due to IL1RAP expression in many solid tumor types

CORPORATE STRENGTH DRIVING INNOVATION

- CANxx Technology Platform: Worldclass Leader in IL1RAP antibody development
- Robust patent portfolio: IL1RAP antibody target in oncology (2032), nadunolimab (2035) and CAN10 (2041)





Clinical Programs supported by Unique Platform

Asset	Indication	Discovery	Preclinical	Phase 1	Phase 2	Phase 3
CAN10	Hidradenitis Suppurativa (HS) Atopic Dermatitis (AD)*					
Nadunolimab	PDAC*** TNBC**					
CANxx	New development programs through unique IL1RAP platform (e.g. BsMAb, ADC)					

PDAC – pancreatic cancer; TNBC – triple-negative breast cancer; *) Dupilumab non-responders **) Recruitment in randomized phase 2 trial ongoing in TNBC ***) Recruitments finalized



Executive Management Team



Damian Marron



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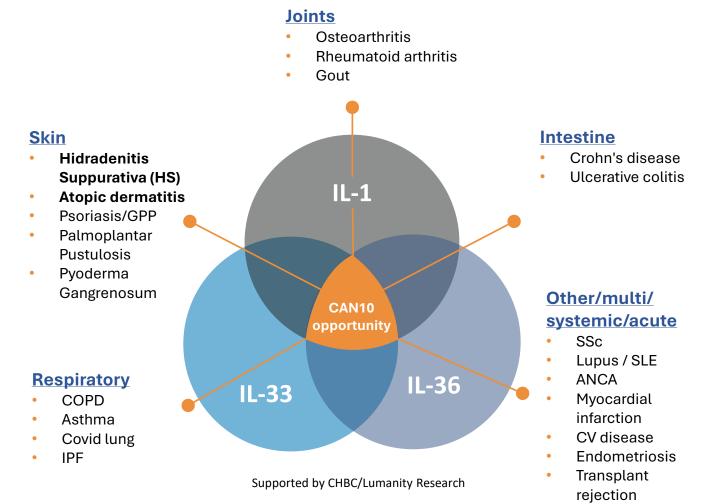


CAN10 OPPORTUNITIES IN AUTOIMMUNE/INFLAMMATORY DISEASES

CAN10 is an Anti-IL1RAP antibody for treatment of autoimmune and inflammatory disease. CAN10 can, by binding IL1RAP, block IL-1, IL-33 and IL-36 signaling pathways simultaneously. This unique function provides CAN10 with great potential for the effective treatment of various diseases whereby CAN10 can achieve a broader and stronger effect compared to treatments aimed at the individual signaling pathways.

CAN10 provides a unique opportunity to block IL-1 superfamily signaling

- The IL-1 superfamily of ligands and receptors is primarily associated with acute and chronic inflammation¹
- Strong evidence of IL-1 family cytokines (IL-1, IL-33, IL-36) is driving multiple inflammatory diseases
- Individual blockade of IL-1 family members² have not resulted in sufficient clinical efficacy in diverse diseases
- CAN10 broader mechanism is highly relevant in dermatological, fibrotic and cardiovascular diseases



1. Interleukin-1 in the pathogenesis and treatment of inflammatory diseases - Charles A. Dinarello, Blood (2011) 117 (14): 3720–3732.

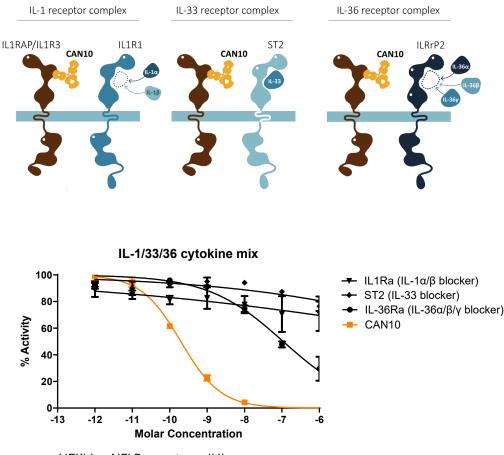
2. Canakinumab, spesolimab

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CAN10 is developed to block IL-1 superfamily with precision

- CAN10 blocks multiple IL-1 family signaling by targeting a single receptor
 - Binds crucial epitope on common accessory protein (IL1RAP)
 - Prevents signaling from IL-1a/ β , IL-33 and IL-36a/ β/γ
- CAN10 has shown robust efficacy in preclinical models of several diseases
 - Differentiation: blocks inflammation and fibrosis where IL-1α/β or IL-1β blockade only does not



HEKblue NFkB reporter cell line assay

CAN10 IS UNDERGOING PHASE 1 (SAD/MAD) DEVELOPMENT - NO SAFETY ISSUES REPORTED



CAN10 First-in-Human study (FIH) – SAD/MAD

SAD (IV)

- \bigcirc
- Healthy volunteers (N=76)
- Placebo controlled
- 10 dose cohorts
- Finalized

MAD – Healthy (SC)

- Healthy volunteers
- 2 dose cohorts, placebo controlled
- SC Day 1, 7 followed by every 14 days
- Phase 2 preparations

Q2 2025: Phase 1

Q4 2025: Start Phase 2

MAD – Psoriasis (SC)

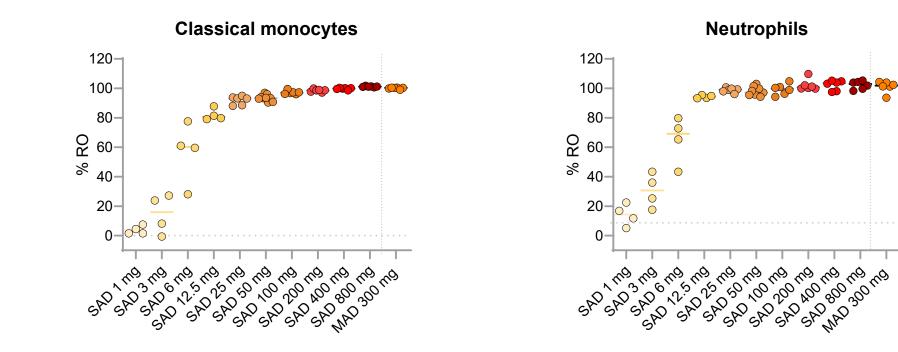
- Mild-Moderate plaque psoriasis
- Enable mechanistic studies

FULL RECEPTOR OCCUPANCY, IL-1 FAMILY CYTOKINE BLOCKADE & NO SAFETY CONCERN DEMONSTRATED



CAN10 FIH – CAN10 saturates IL1RAP on monocytes and neutrophils

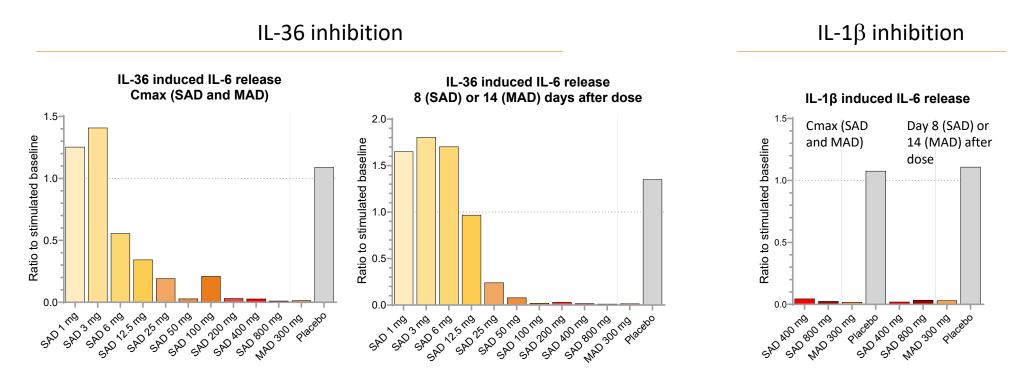
- Receptor occupancy documented (at C_{max})
- Full IL1RAP coverage from 25-50 mg single dose IV





CAN10 FIH – Full blockade of both IL-36 and IL-1 β signaling

• Inhibition of IL-36 and IL-1 β signaling documented at C_{max} (SAD and MAD) and day 8 (SAD) or 14 days after 3rd dose (MAD) \rightarrow long lasting effect

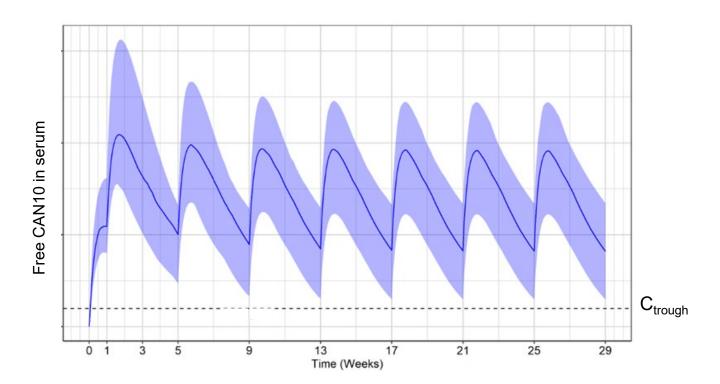


HIGHLIGHTS UNIQUE POTENTIAL OF CAN10 TO HIT DIFFERENT IL-1 SUPERFAMILY PATHWAYS SIMULTANEOUSLY



CAN10 FIH – PK results support Q4W dosing

- Linear PK profile
- High bioavailability
- PK model development has been performed by Certara
- Results from simulations support feasibility of Q4W dosing
- Lowest estimated level for efficacy (C_{trough}) has been set based on ex vivo inhibition assay results and RO



COMBINED RESULTS SO FAR SUGGEST EFFICACY, SAFETY AND CONVENIENCY FOR FUTURE APPLICATIONS



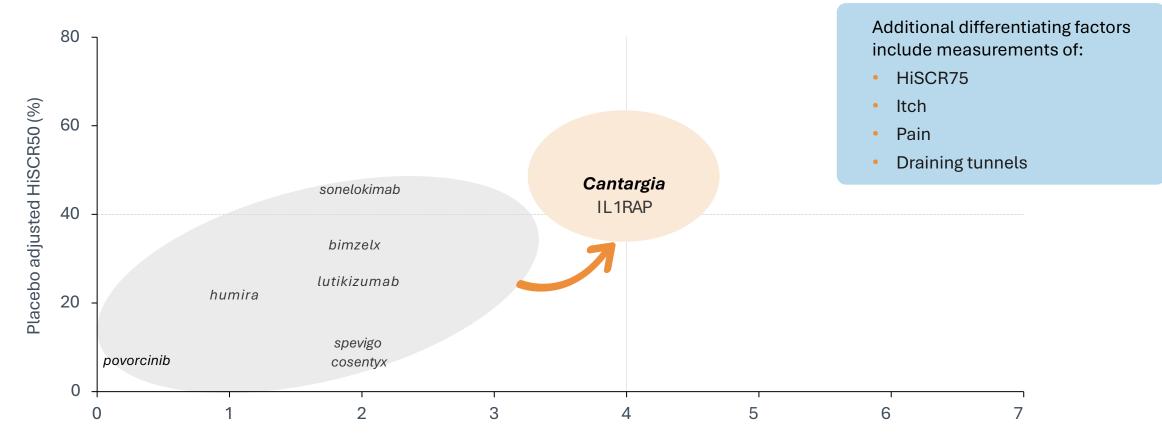
Hidradenitis Suppurativa (HS) – Disease Overview

~ 1-2% ¹	Of people worldwide have Hidradenitis Suppurativa	80-160 million people	
~ 50%²	Of patients moderate/severe HS (Hurley stage II/III)	(C) SEVERE SCAREING SEVERE SCAREING SE	
~ 55% ³	Of HS Patients are not well controlled on Standard Of Care	SoC: TNFa & IL-17	
10 years ⁴	Time that patients suffer from symptoms prior to accurate diagnosis	Significant burden on Healthcare systems	

1. James G. Krueger et..al, Br J Dermatol 2024; 190:149–162. <u>Note</u> that Globally, prevalence studies report higher results of up to 4.1% (3. Jemec GB, Kimball AB. Hidradenitis suppurativa: epidemiology and scope of the problem. J Am Acad Dermatol. (2015)) 2. van der Zee, et al., Dermatology 2018. 3. TD Cowen March 2024 Health Care Conference 4. Joslyn Kirby et..al, Front. Med. Technol. , 25 March 2024



Despite advances in HS, new approaches are needed to break the efficacy ceiling, improve convenience and safety



Maintenance dosing frequency (weeks)

The HS market is expected to grow to USD 10 billion by 2030 in the seven major markets*



Unique MoA in the treatment of HS

IL-1 superfamily has roles integrated with and in parallel to the IL-17 pathway

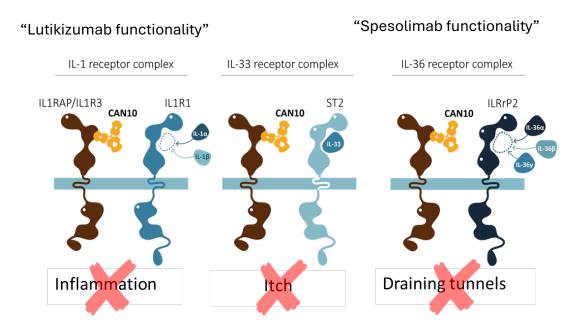
- IL-1 superfamily cytokines are jointly upregulated in HS skin lesions
- Unique CAN10 MoA differentiates from current treatments and create opportunity in poor responders and refractory patients (~50%)

IL-1 superfamily blockade may provide a competitive advantage, particularly in the severe patient population

- IL-36 blockade showed effects on draining tunnels
- Efficacy indicated in severe patient group (majority Hurley stage III)

CAN10 integrates several functionalities in one molecule, combines the effects of lutikizumab and spesolimab

 Broad mode of action expected by IL1RAP blockade, targeting several of the pathophysiological changes in HS





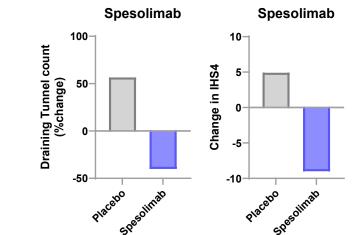
CAN10 is validated by external clinical results in HS

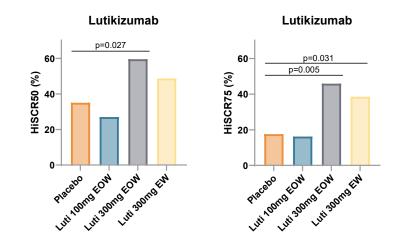
IL-36R-blockade (spesolimab) positive results on disease severity¹

- Efficacy in Phase 2 randomized controlled study: iHS4 and HASI-R, with a particular effect on draining tunnels (dTs)
- Phase 2b/3 study ongoing

Combined IL-1 α/β blockade (lutikizumab) generated good response rates in anti-TNF α refractory patients²

- Efficacy in phase 2 study on primary (HiSCR50) and secondary endpoints (NRS30, skin pain) as well as HiSCR75 at 16 weeks
- Phase 3 study ongoing

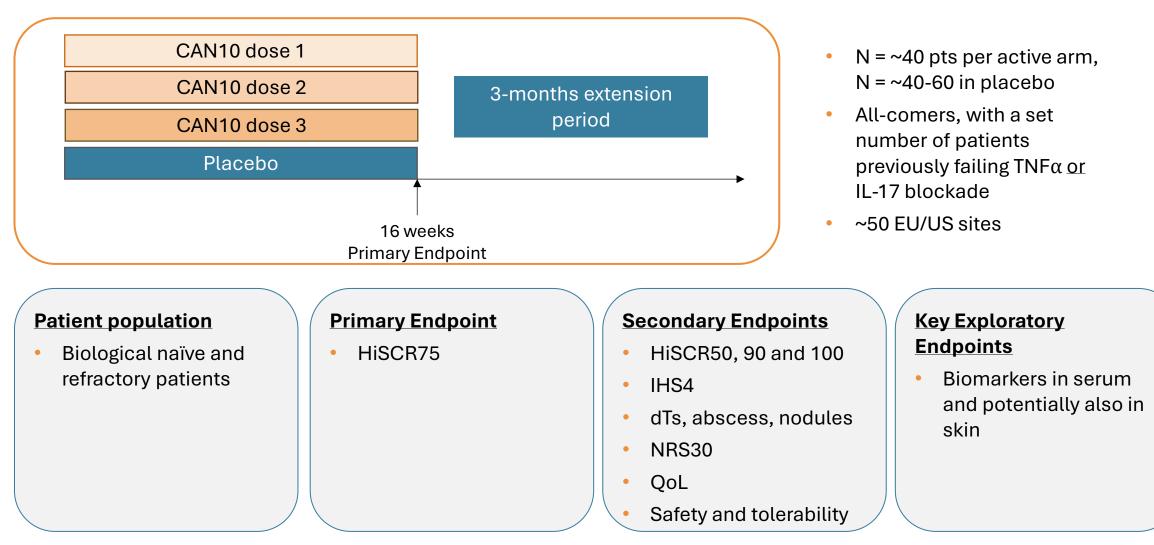




International Activity (International Activity) (International Activ

1. Alavi A, et al. Br J Dermatol. 2024 2. Kimball AB, et al. Presented at: American Academy of Dermatology; March 8-12, 2024; San Diego, CA; **Note:** Spesolimab is developed by Boehringer Ingelheim; Lutikizumab is developed by Abbvie

Next: Proposed Phase 2 study in HS





Atopic Dermatitis (AD) – Disease Overview & Treatment Approach

~ 46 million ¹	Prevalent diagnosed patient population of people suffering from AD	
~ \$22bn \$8.5bn	Expected global sales in AD treated population in 2033	
~ 30% ²	Even among patients treated with the most prescribed biologic for AD, dupilumab, ~ 30% have inadequate response	
ini ini ini ini	Broad activation of multiple inflammatory pathways and disease heterogeneity ³ drive the high need for broader MoAs and personalized treatments	
	Blockade of multiple cytokine signaling with CAN-10 supports a broader approach to address heterogeneity in acute and chronically treated patients	and the second

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¹ Global Data: Atopic Dermatitis: Global Drug Forecast and Market Analysis to 2033, March 2025

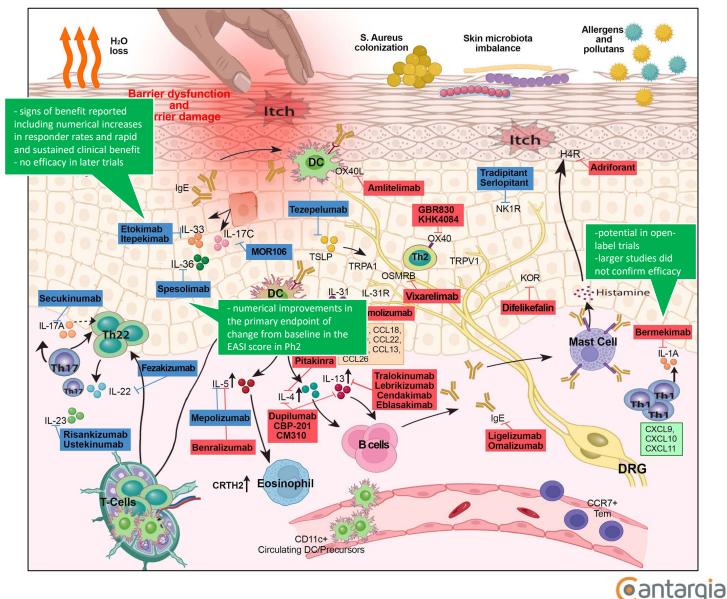
² Diamant Thaçi, et..al, Journal of Dermatological Science 94 (2019) 266–275, A Literature Review of Real-World Effectiveness and Safety of Dupilumab for Atopic Dermatitis, Kamata, Masahiro et al., JID Innovations, Volume 1, Issue 3, 100042

³ Facheris, P., Jeffery, J., Del Duca, E. et al. The translational revolution in atopic dermatitis: the paradigm shift from pathogenesis to treatment. Cell Mol Immunol 20, 448–474 (2023).



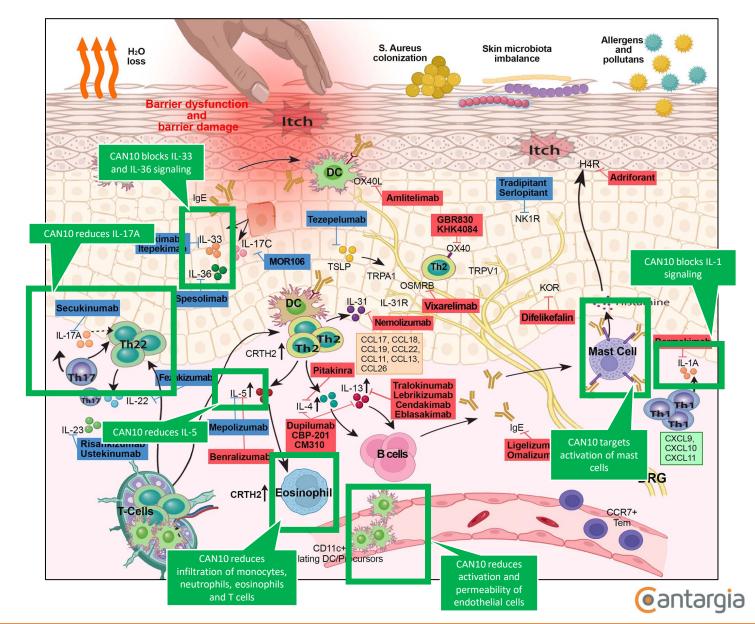
CAN10 rationale in AD patients not responding to dupilumab

- Broad activation of inflammatory pathways in AD
 - IL-1, IL-33 and IL-36 affects several of the inflammatory pathways implicated in AD
- Clinical data and commercial interest indicating benefit of individual cytokine blockade
 - Signs of efficacy, but not sufficient, from bermekimab (anti-IL-1α), etokimab/itepekimab (anti-IL-33), spesolimab (anti-IL-36R)
 - Lutikizumab (IL-1α/β blockade; Abbvie), two Ph2 trials ongoing
- Combined blockade set to increase efficacy



Non-clinical data supporting CAN10 in AD

- Multiple pathways and signaling levels targeted simultaneously
- MOA supported by results from several non-clinical activities
 - Reduces IL-17, IL-5
 - Blocks tissue inflammation superior to IL-1 blockade (including skin)
 - Blocks IL-1/33-induced endothelial and mast cell inflammation
 - Reduces skin (and lung) fibrosis
- MoA supported by human data of whole blood
 - Several key biomarkers affected by CAN10



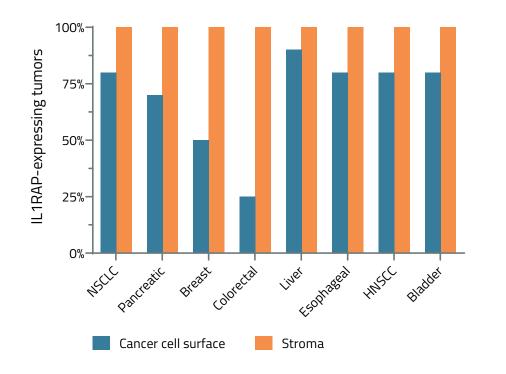


NADUNOLIMAB COMBAT CANCER AND ENHANCE THE IMMUNE SYSTEM'S ABILITY TO DESTROY CANCER CELLS

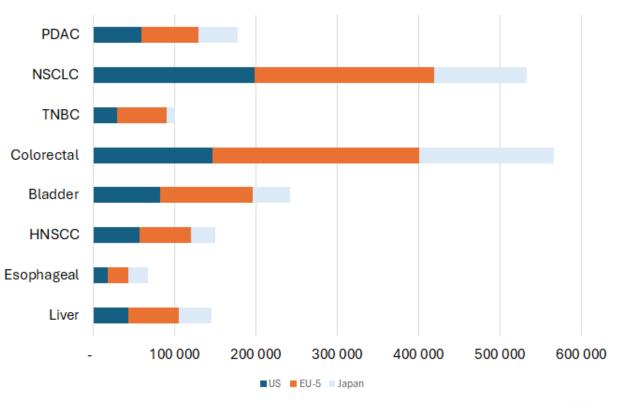
Nadunolimab (CAN04) is an Anti-IL1RAP antibody for treatment of various cancer types. CAN04 binds strongly to its target molecule IL1RAP, expressed on tumor cells from many types of cancer. CAN04 blocks the signaling of interleukin-1, alpha and beta, thereby limiting tumor development as well as working synergistically with chemotherapy and adding functionality through Antibody-Dependent Cellular Cytotoxicity (ADCC)

IL1RAP overexpressed in most solid tumors provides treatment opportunity in various cancer types

IL1RAP EXPRESSION IN SOLID TUMOR TYPES

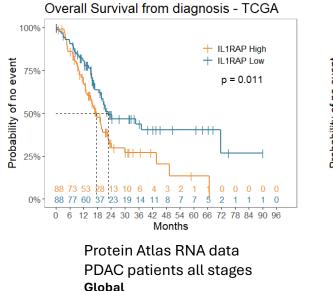


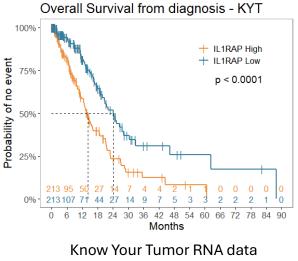
INCIDENCE RATES SHOW BROAD OPPORTUNITY IN VARIOUS CANCER TYPES



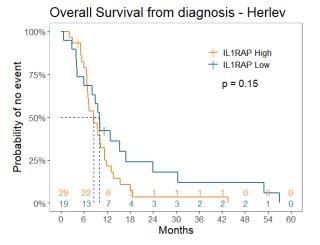


High IL1RAP expression is linked to poor outcome in patients with pancreatic tumors





PDAC patients stage III/IV US



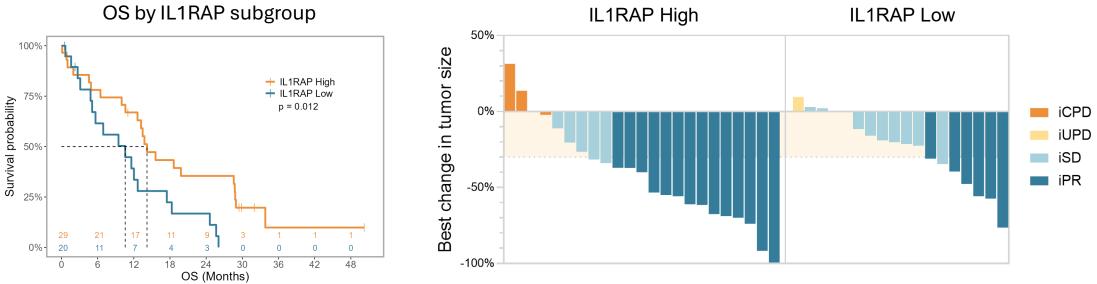
Herlev biopsy protein stainings PDAC patients stage IV treated with gem/nab-pac **DK**

- → IL1 family members are upregulated in PDAC tumors as compared to normal pancreatic tissue
- IL1RAP is expressed on tumor cells, cancerassociated fibroblasts and immune cells in tumor microenvironment
- → High IL1RAP RNA and protein expression is associated with poor outcome in PDAC

HIGH IL1RAP EXPRESSION IS A PROGNOSTIC FACTOR FOR POOR SURVIVAL



PDAC - Strong efficacy in patients with high tumor IL1RAP expression level



Efficacy analysis for IL1RAP High (n=29) vs IL1RAP Low (n=20) PDAC patients

- → High IL1RAP expression is a poor prognostic marker for treatment with gem/nab-paclitaxel
- > Significantly prolonged OS in ILRAP High vs IL1RAP Low patients (14.2 vs 10.6 mo; p=0.012)
- > Deeper and more durable responses in IL1RAP High subgroup: 11 patients had 50% or more tumor size decrease

IL1RAP HIGH PATIENTS SHOW THE STRONGEST BENEFIT

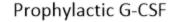
iCPD – Confirmed Progressive Disease; iUPD – Unconfirmed Progressive Disease; iSD – Stable Disease; iPR – Partial Response (all according to iRECIST)

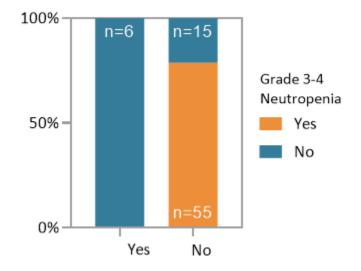
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PDAC – Manageable safety with a potential benefit

- \rightarrow Neutropenia manageable through G-CSF prophylaxis
 - → In 6 patients given G-CSF prophylaxis, none developed grade 3-4 neutropenia
- Only 1 % peripheral neuropathy grade 3-4 observed (17% in historical controls)

Grade 3 or higher AEs	Gem/Abraxane Von Hoff, 2013 (n=421)	Nadunolimab+Gem/Abraxane CANFOUR (n=76)	
Neutropenia	38%	65%	
Leukopenia	31%	24%	
Thrombocytopenia	13%	15%	
Febrile neutropenia	3%	13%	
Anemia	13%	13%	
Fatigue	17%	8%	
Diarrhea	6%	3%	
Peripheral neuropathy	17%	1%	



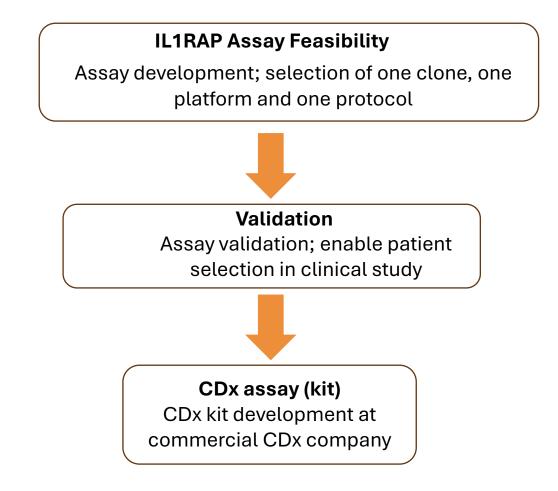


G-CSF PROPHYLAXIS IMPLEMENTED IN FUTURE TRIALS; POTENTIAL REDUCTION OF NEUROPATHY TO BE DOCUMENTED IN RANDOMIZED TRIALS

Median duration of treatment 5.5 mo (ref 3.9 mo); most common reasons for termination: gastrointestinal events or general health deterioration. No patients discontinued due to neutropenia.



PDAC – Next steps



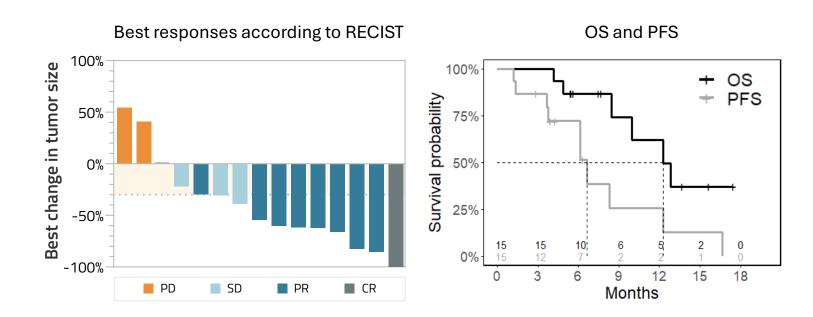
Proposed PDAC study design

- Metastatic PDAC patients selected for high IL1RAP expression
- Treatment naive patients
- Combination with gemcitabine/nabpaclitaxel
- Controlled study with 2-dose lead-in
- Adaptive phase 2/3
- Primary read-out: OS
- Interim analysis allows possibility for early MAA

IL1RAP ASSAY ALLOWS FOR PATIENT SELECTION IN FUTURE CLINICAL STUDIES



Triple-negative breast cancer (TNBC) Promising early safety and efficacy



Benchmark Gem/Carbo: OS 11.1 mo, PFS 4.1 mo, ORR 30% (O'Shaughnessy et al, J Clin Oncol 2014)

Nadunolimab combination with Gem/Carbo in 1st/2nd line metastatic TNBC:

15 patients enrolled in the doseescalation phase:

- Preliminary ORR: 60%
 (1 CR, 8 PR, 4 SD, 2 PD)
- Preliminary median OS 12.3 mo, median PFS 6.6 mo
- Acceptable safety profile (G-CSF given prophylactically to control neutropenia)

Randomized phase 2 fully enrolled

• Preliminary efficacy results expected mid-2025

RESPONSE RATE OF NADUNOLIMAB COMBINATION THERAPY WELL ABOVE HISTORICAL DATA FOR CHEMOTHERAPY ONLY





MILESTONES & INVESTMENT HIGHLIGHTS

Upcoming Milestones

2025		Q1	Q2	Q3	Q4
CAN10		Initial Phase 1 MAD results	Phase 1 MAD study completed		Phase 2 start in HS & AD
Nadunolimab	PDAC			Regulatory Update	
	TNBC	TRIFOUR recruitment completed	TRIFOUR initial results		
	AML	Study start with MD Anderson*			
Other		Additional new preclinical and translational results			

EXTENSIVE NEWS FLOW EXPECTED DURING 2025

PDAC: Pancreatic ductal adenocarcinoma; NSCLC: Non-Small Cell Lung Cancer; TBNC: Triple Negative Breast Cancer; HS: Hidradenitis Suppurativa; AD: Atopic Dermatitis * US Department of Defense, The University of Texas MD Anderson Cancer Center



Investment Highlights

CORPORATE STRENGTH DRIVING INNOVATION

- Dedicated Team with experience from Pharma, Biotech & Academia
- Two clinical programs ongoing: CAN10 and nadunolimab
- CANxx Technology Platform (ADC + Bi-Specific mAb): Worldclass Leader in IL1RAP antibody development

CAN10: STRONG PHASE 1 DATA – NEXT PRIORITIZED DEVELOPMENT IN HIDRADENITIS SUPPURATIVA

- Phase 1 indicates possible Q4W dosing regimen in HS
- Positive FDA feedback on HS phase 2 trial initiation, incl. strong endorsement from KOL's
- Broadly applicable and differentiating mechanism for treatment in inflammatory diseases (e.g. HS & AD)

NADUNOLIMAB: CLEAR ACTIVITY IN CANCER THERAPY WITH PATH TOWARDS MARKET

- IL1RAP diagnostic in development for PDAC;
- Randomized Phase 2 trial in TNBC fully enrolled (initial data mid-2025);
- To date >300 patients treated



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Thank you

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