




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

Corporate Presentation

March 2025

NASDAQ STOCKHOLM MAIN LIST (CANTA.ST)

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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 differentiating mechanism for treatment in inflammatory diseases
- Convincing results in several models of inflammation and fibrosis
- Phase 1 clinical trial towards finalization – Prioritized development in Hidradenitis Suppurativa (HS)



NADUNOLIMAB: CLEAR ACTIVITY SIGNALS IN CANCER THERAPY WITH UPCOMING CATALYSTS

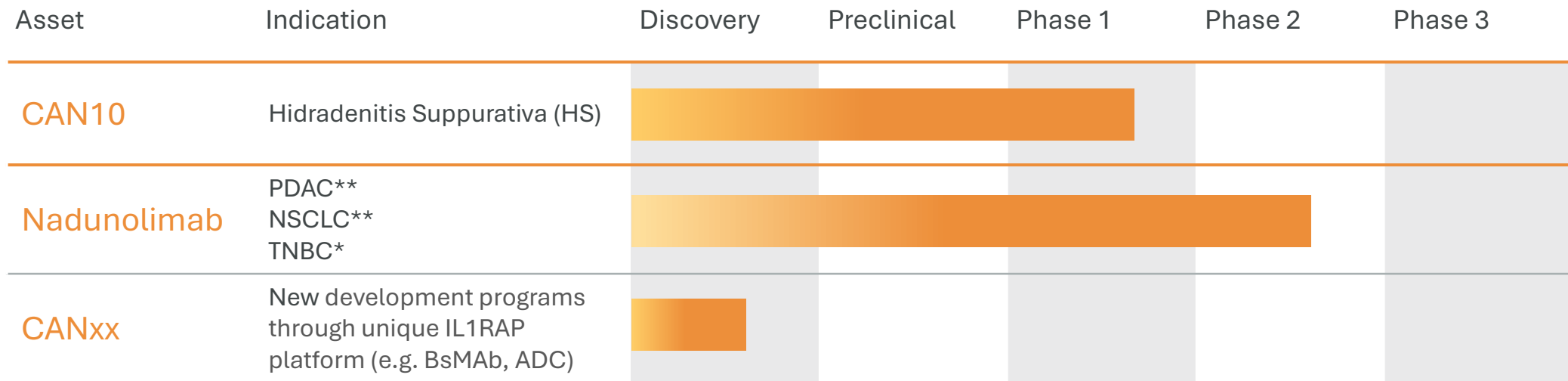
- Strong clinical results in PDAC and NSCLC, and promising initial results in TNBC; >300 patients treated
- Randomized Phase 2 trial in TNBC fully enrolled (Initial data H1 2025); Phase 2b/3 trial in preparation in PDAC
- Broadly applicable due to IL1RAP expression in many solid tumor types



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



PDAC – pancreatic cancer; NSCLC – non-small cell lung cancer; TNBC – triple-negative breast cancer;

*) Recruitment in randomized phase 2 trial ongoing in TNBC

***) Recruitments finalized

Management Team



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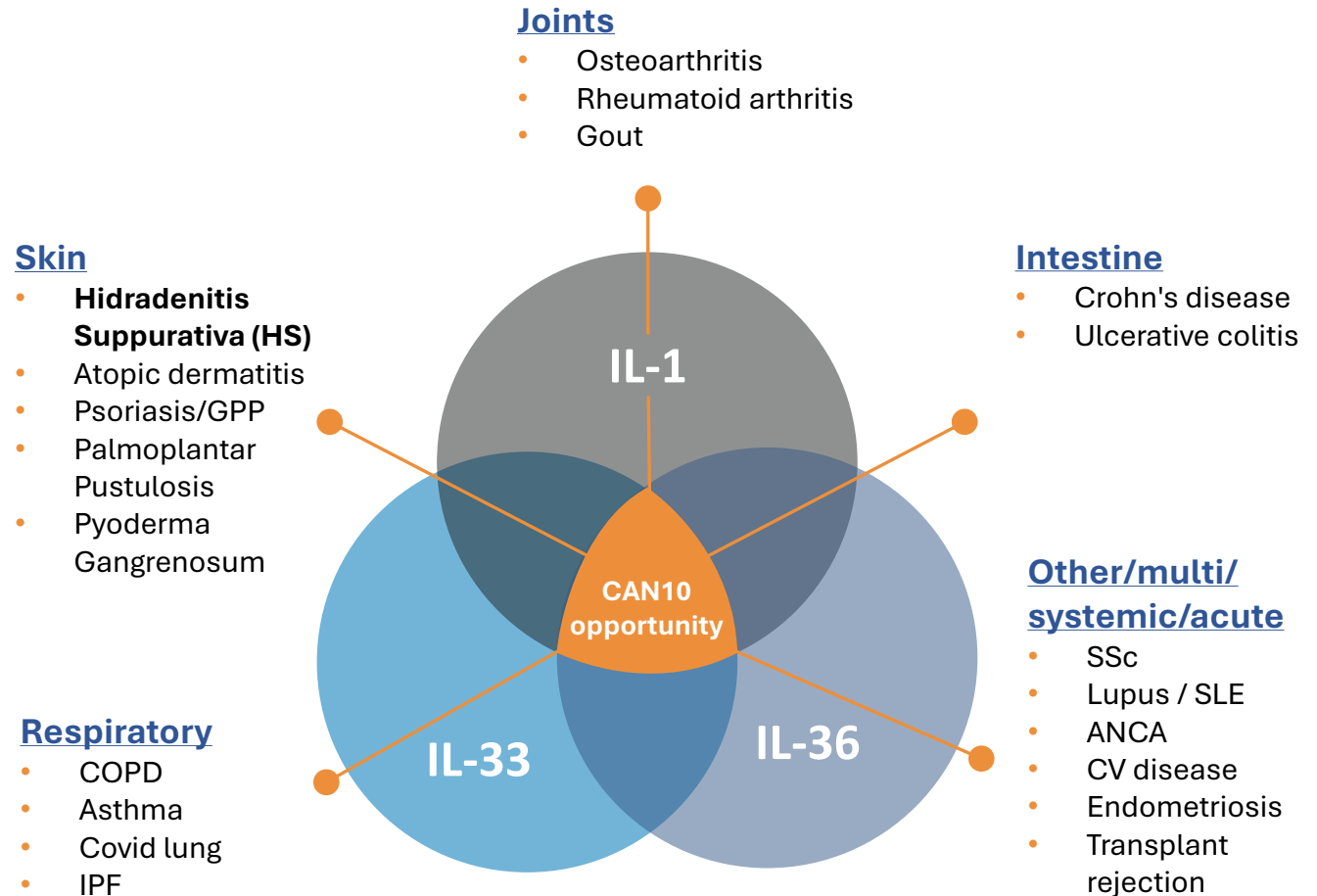


CAN10 OPPORTUNITIES IN AUTOIMMUNE/INFLAMMATORY DISEASES

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 family signaling

- The IL-1 family 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

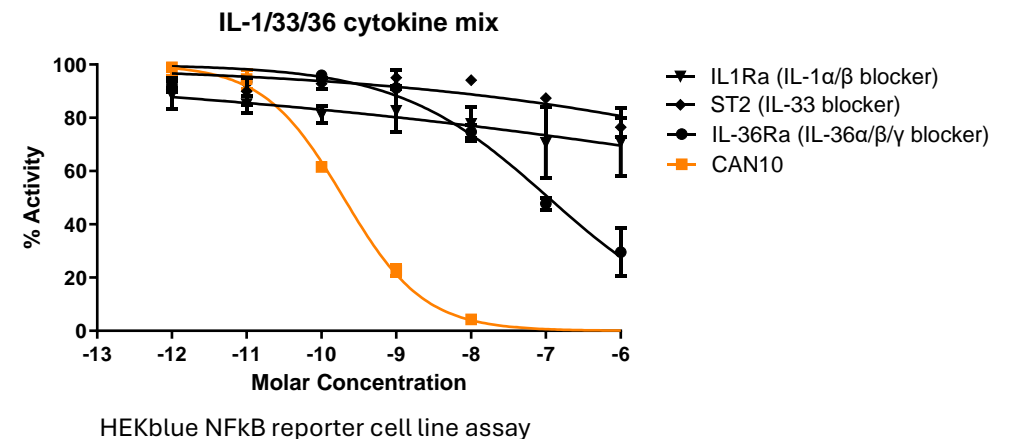
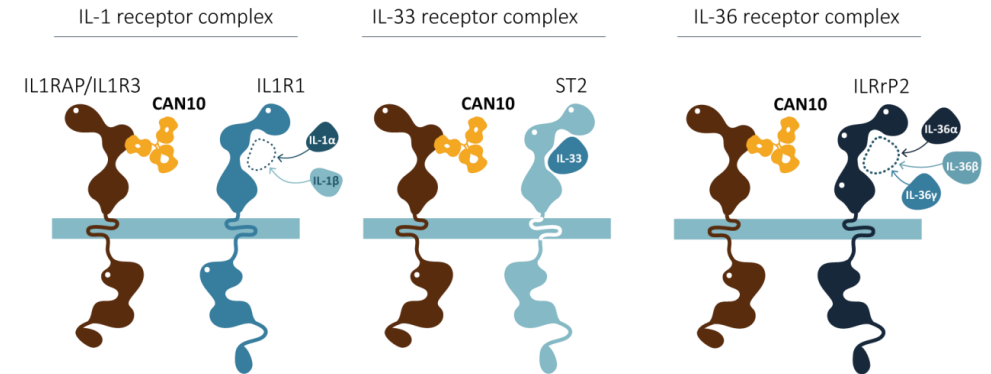


Supported by CHBC/Lumanity Research

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

CAN10 is developed to block IL-1 family 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-1 α/β , IL-33 and IL-36 $\alpha/\beta/\gamma$
- **CAN10 has shown robust efficacy in preclinical models of several diseases**
 - Differentiation: blocks inflammation and fibrosis **where IL-1 α/β or IL-1 β blockade only does not**



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

First-in-Human study – SAD/MAD

SAD (IV)


- Healthy volunteers (N=76)
- Cohorts: CAN10 (6), Placebo (2)
- 10 dose cohorts
- Finalized

MAD – Healthy (SC)

- Healthy volunteers
- 2 dose cohorts
- Phase 2 preparations

MAD – Psoriasis (SC)

- Mild-Moderate plaque psoriasis
- 2 dose cohorts
- Enable mechanistic studies

- Q2 2025: Phase 1 
- Q4 2025: Start Phase 2

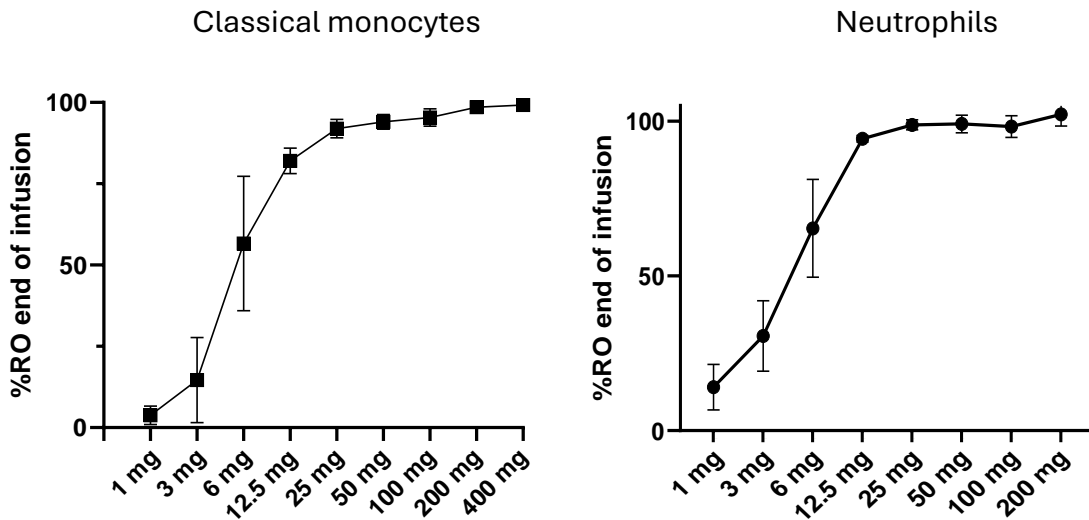
FULL RECEPTOR OCCUPANCY AND IL1 FAMILY CYTOKINE BLOCKING DEMONSTRATED IN SAD

CAN10 first-in-human study - SAD part*

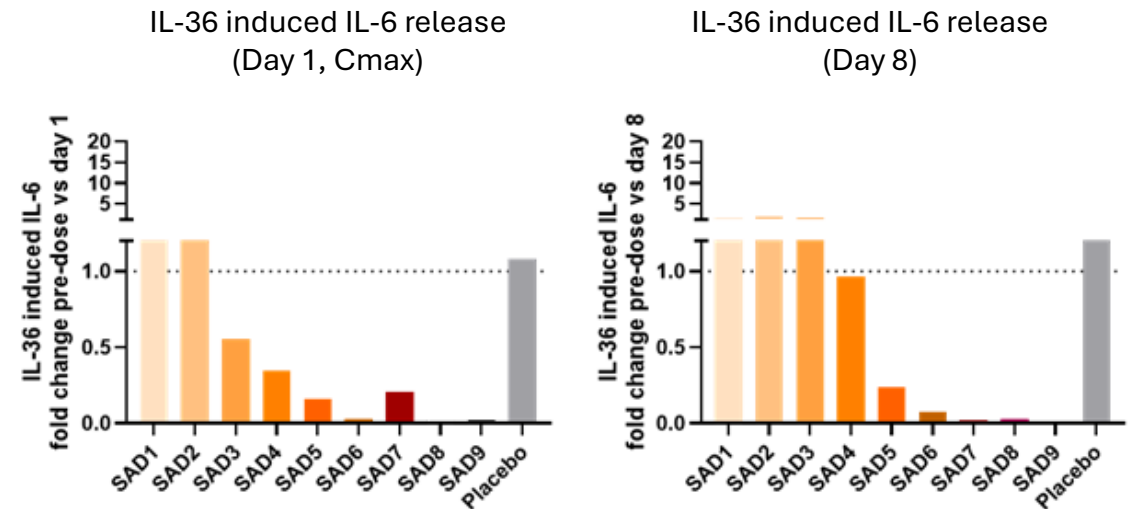
Results – PD effects

- Receptor occupancy documented (at C_{max})
- Inhibition of IL-36 signaling documented at C_{max} and day 8 → long lasting effect

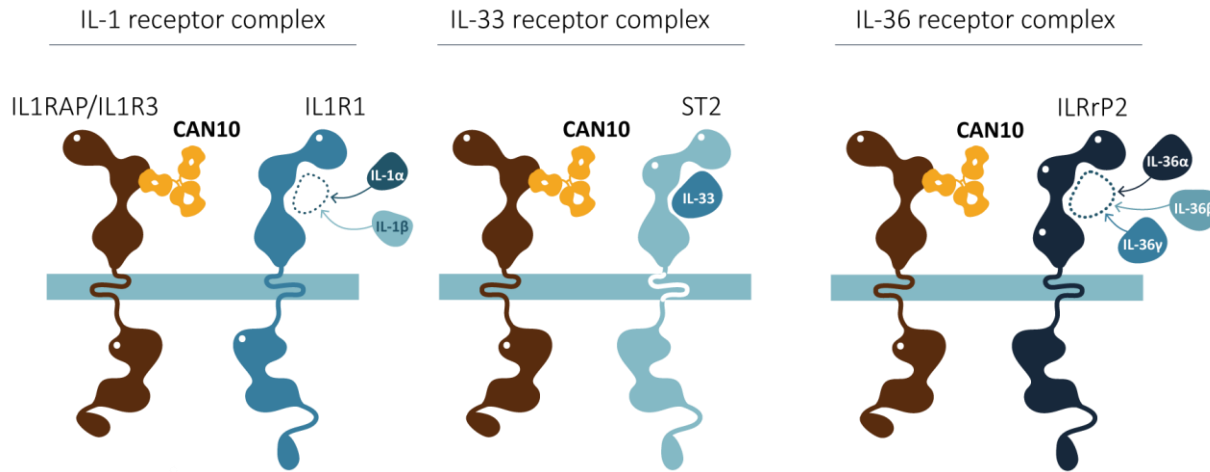
Receptor Occupancy



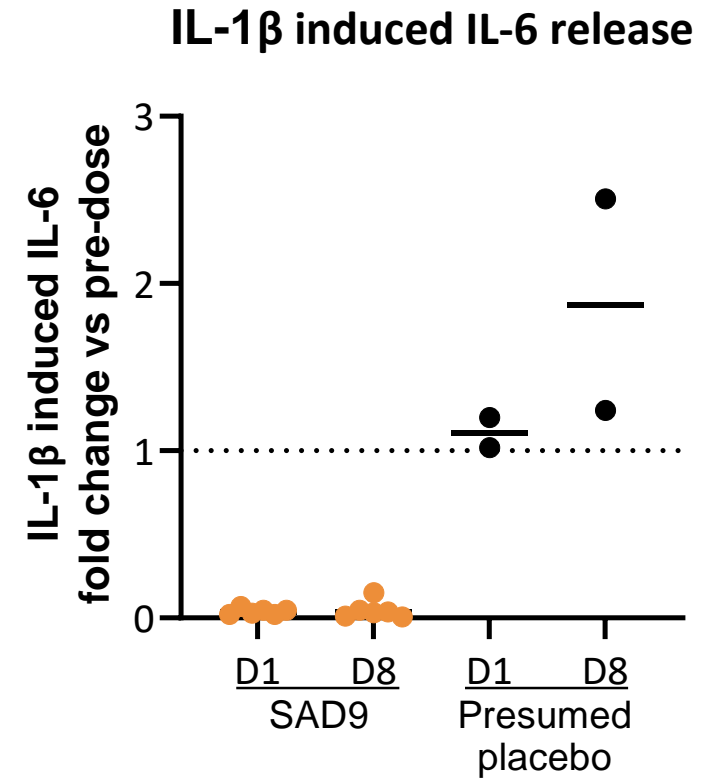
Whole blood ex vivo inhibition assay



Full blockade of IL-1 β signaling at Cmax and Day 8



- **Unique function of CAN10 and differentiating from approved treatments**
 - Making immune cell non-responsive to IL-1 and IL-36



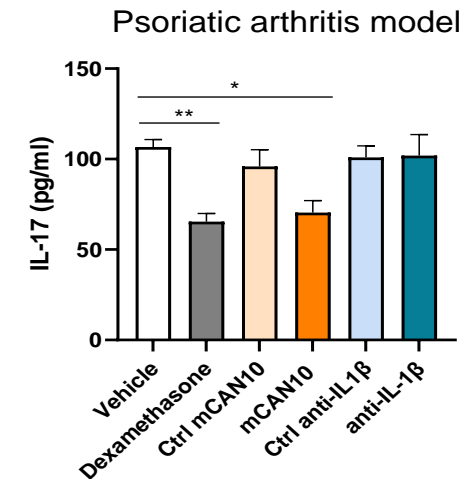
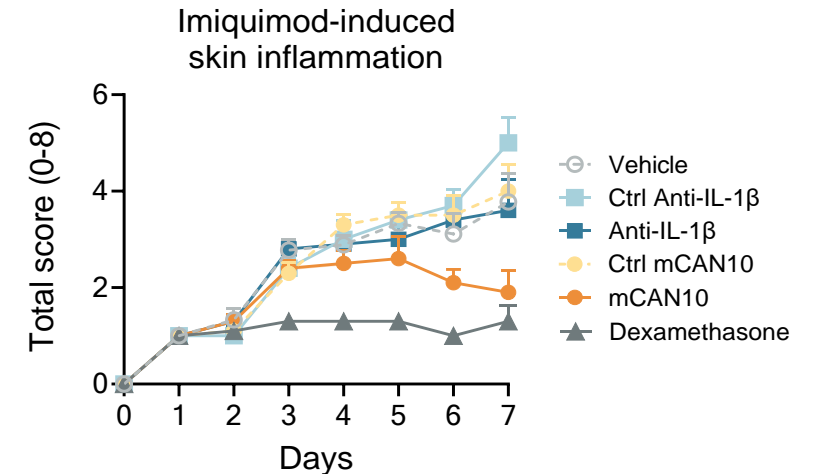
SUCCESSFULLY COMPLETED SAD, MULTIPLE DOSING INVESTIGATED IN VOLUNTEER & PSORIASIS COHORTS

CAN10 First-in-Human study - MAD

- Strong rationale for IL1RAP blockade in inflammatory skin diseases
 - Blocks skin inflammation and IL-17 where anti-IL-1 β does not
- Psoriasis chosen for phase 1 to enable mechanistic studies; no plans to develop further
- Initial PK data supports potential 4-weekly (Q4W) dosing
- Positive FDA feedback on phase 2 trial, with strong endorsement from KOL's on lead indication

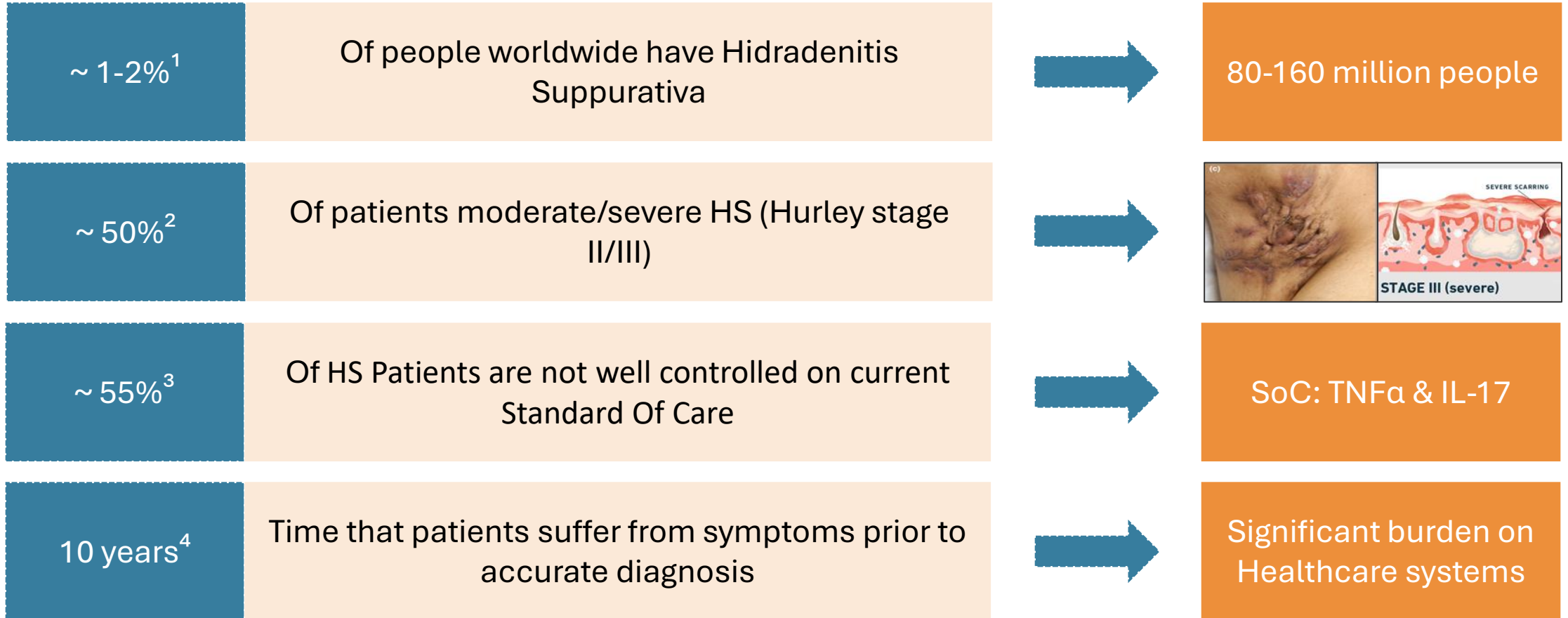
Planned PD analyses

- Receptor occupancy
- Ex vivo inhibition assay
- Psoriasis severity scoring
- Skin biopsies



HIDRADENITIS SUPPURATIVA AS LEAD INDICATION

Hidradenitis Suppurativa (HS) – Disease Overview



1. James G. Krueger et al., Br J Dermatol 2024; 190:149–162. **Note** 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

Unique MoA in the treatment of HS

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

- IL-1 family 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%)

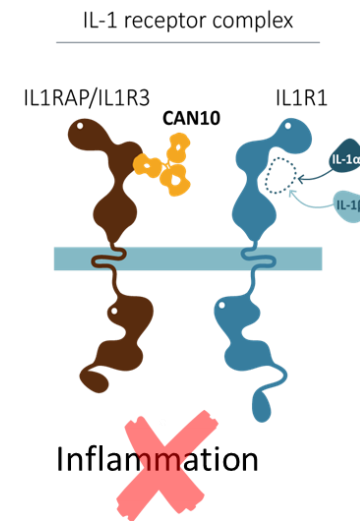
IL-1 family blockade may provide a competitive advantage, particularly in the severe patient population

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

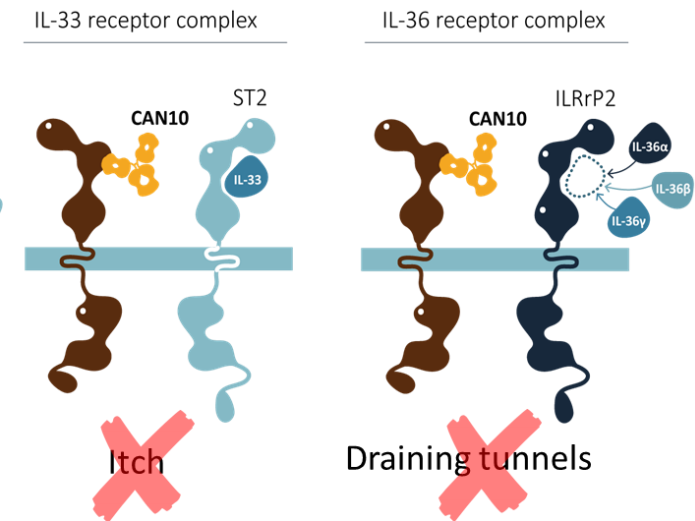
CAN10 integrates several functionalities in one molecule, combines the effects of lutikizumab and spesolimab

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

“Lutikizumab functionality”



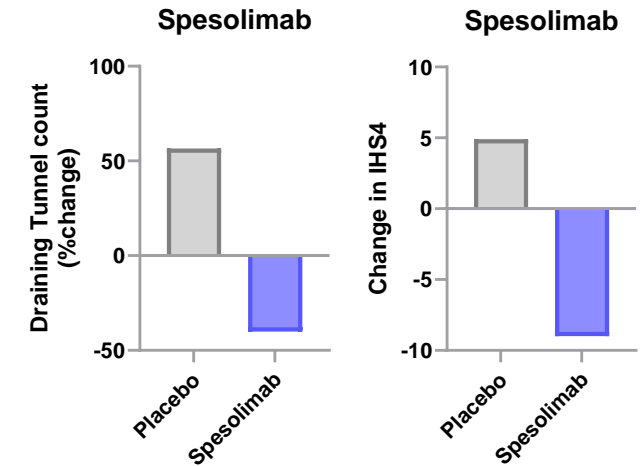
“Spesolimab functionality”



CAN10 is validated by External clinical results in HS

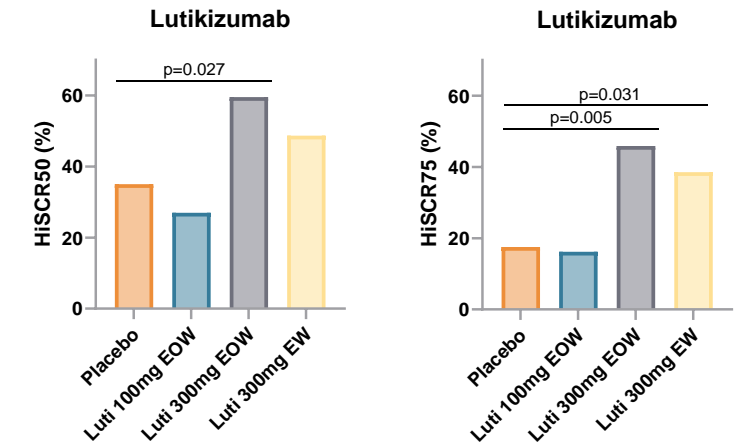
IL-36R-blockade (spesolimab) positive results on disease severity¹

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



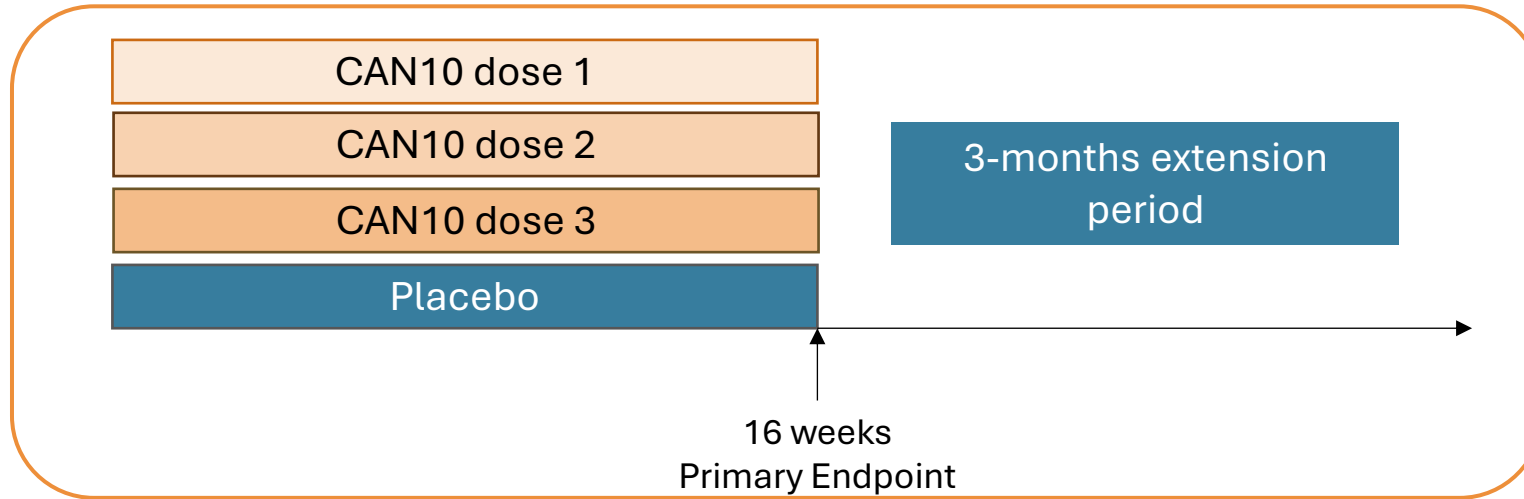
Combined IL-1 α / β blockade (lutikizumab) generated good response rates in anti-TNF α refractory patients²

- 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



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



- N = ~40 pts per active arm, N = ~40-60 in placebo
- All-comers, with a set number of patients previously failing TNF α or IL-17 blockade
- ~50 EU/US sites

Patient population

- Biological naïve and refractory patients

Primary Endpoint

- HiSCR75

Secondary Endpoints

- HiSCR50, 90 and 100
- IHS4
- dTs, abscess, nodules
- NRS30
- QoL
- Safety and tolerability

Key Exploratory Endpoints

- Biomarkers in serum and potentially also in skin



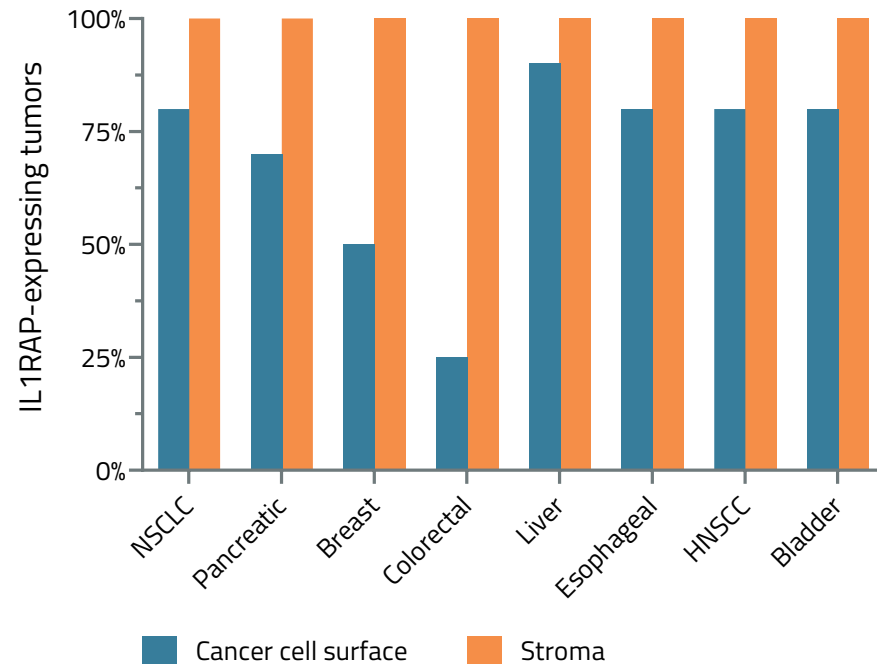
NADUNOLIMAB

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

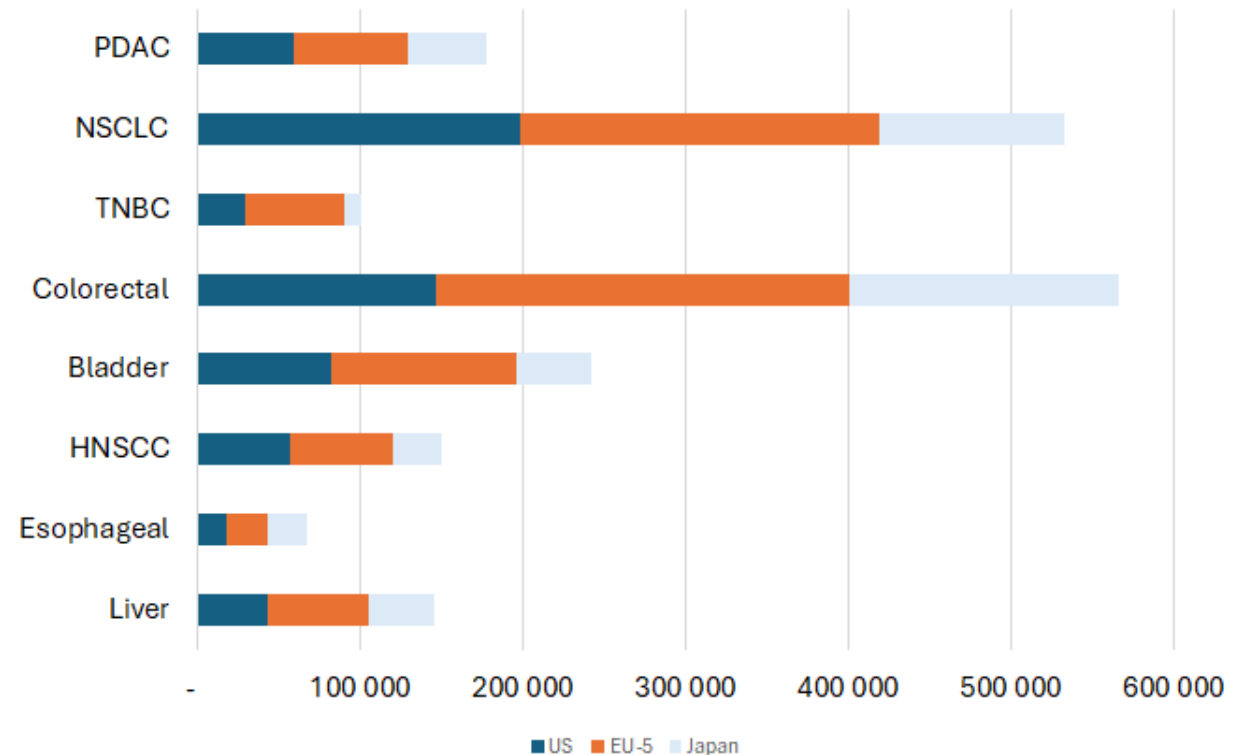
Nadunolimab (CAN04) is an Anti-IL1RAP antibody for treatment of various cancer types. CAN04 binds strongly to its target molecule IL1RAP, expressed on tumor cells from many types of cancer. CAN04 blocks the signaling of interleukin-1, alpha and beta, thereby limiting tumor development as well as working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

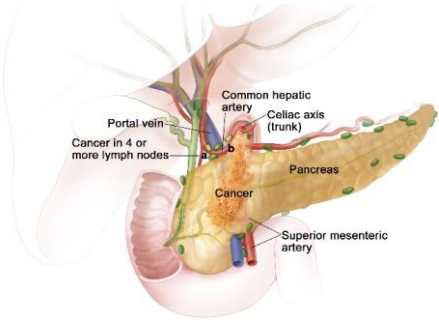
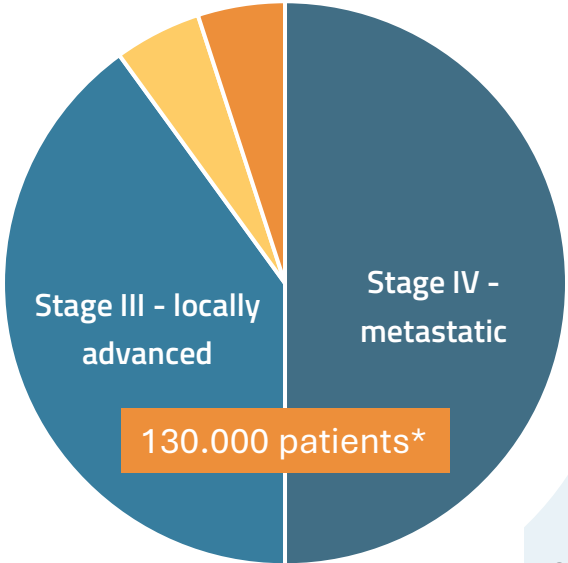
IL1RAP EXPRESSION IN SOLID TUMOR TYPES



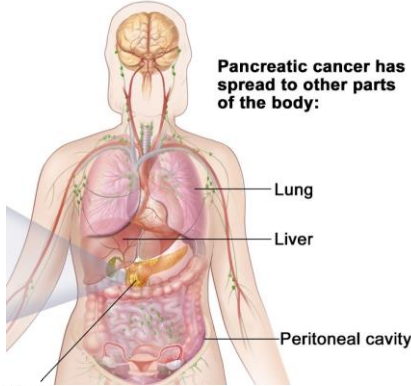
INCIDENCE RATES SHOW BROAD OPPORTUNITY IN VARIOUS CANCER TYPES



Pancreatic Cancer (PDAC) as primary indication



Stage III



Pancreatic cancer has spread to other parts of the body:

Stage IV

LOCALLY ADVANCED OR METASTATIC DISEASE

Survival:

- 8.5 – 11.1 mo

Treatment:

- Gemcitabine/nab-paclitaxel
- Gemcitabine if poorer performance
- FOLFIRINOX only if good patient condition status
- Jan 2023: 1st line NALIRIFOX (OS 11.1 mo)

Images from National Cancer Institute

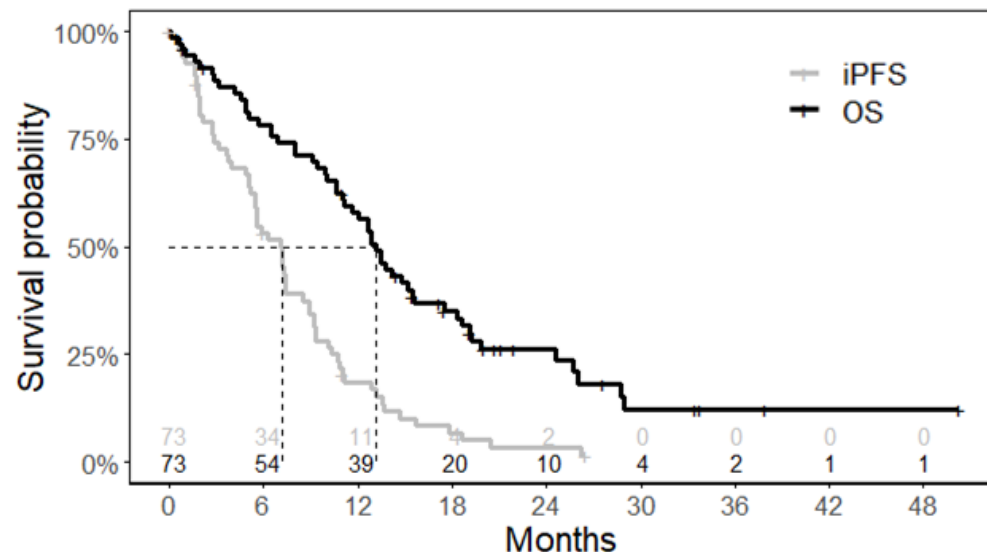
CURRENT DEVELOPMENT FOCUSES ON FIRST-LINE METASTATIC DISEASE WITH POTENTIAL TO MOVE TO EARLIER TREATMENT SETTINGS

Note: two-thirds of pancreatic cancer patients are diagnosed with unresectable Stage III or Stage IV metastatic disease, and a significant proportion of resectable Stage III disease (85%) progresses to metastatic pancreatic cancer (Global Data, (accessed 2025))

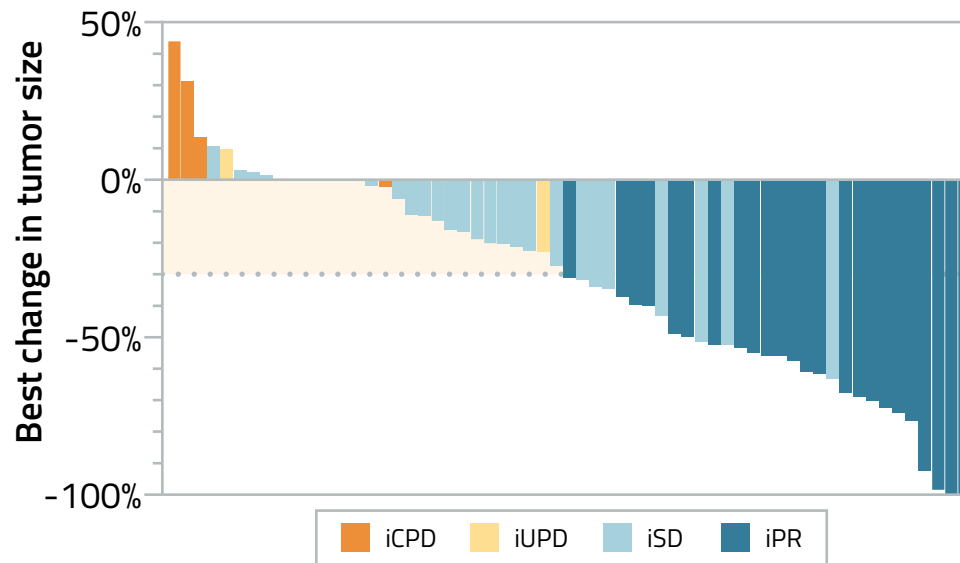
*US, EU4+UK, JPN

Pancreatic Cancer – Positive data in 1st line patients

OS and iPFS for mITT patients



Best responses according to iRECIST



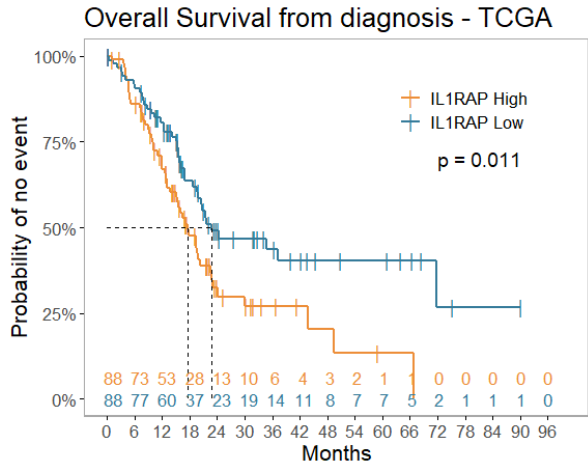
Nadunolimab combination with Gem/Abraxane in 1st line PDAC (n=73):

- 33% response rate with long OS and iPFS
 - Additional 5 (7%) patients had on-treatment benefit beyond progression
- Promising OS (13.2 mo), iPFS (7.2 mo) and DCR (71%); 2 patients still on treatment

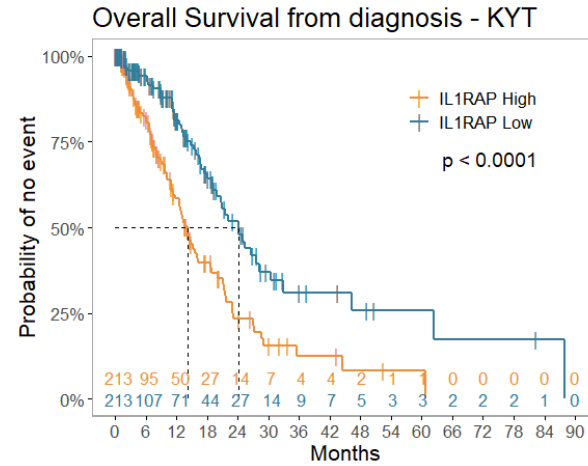
OS AND PFS SUPERIOR TO HISTORICAL CONTROLS IN PDAC

Benchmark Gem/Abraxane: OS 8.5 mo, PFS 5.3 mo, ORR 23%, DCR 48% (Von Hoff et al, N Engl J Med 2013); OS 9.2 mo, PFS 5.6 mo, ORR 36%, DCR 62%, (NAPOLI-3, ASCO GI 2023)
 iCPD – Confirmed Progressive Disease; iUPD – Unconfirmed Progressive Disease; iSD – Stable Disease; iPR – Partial Response (all according to iRECIST)

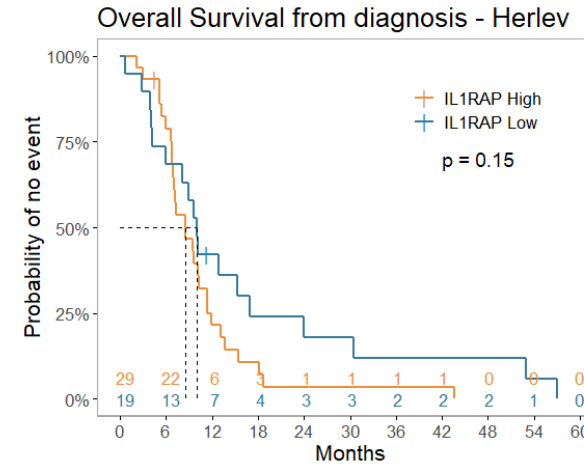
High IL1RAP expression is linked to poor outcome in patients with pancreatic tumors



Protein Atlas RNA data
 PDAC patients all stages
 Global



Know Your Tumor RNA data
 PDAC patients stage III/IV
 US

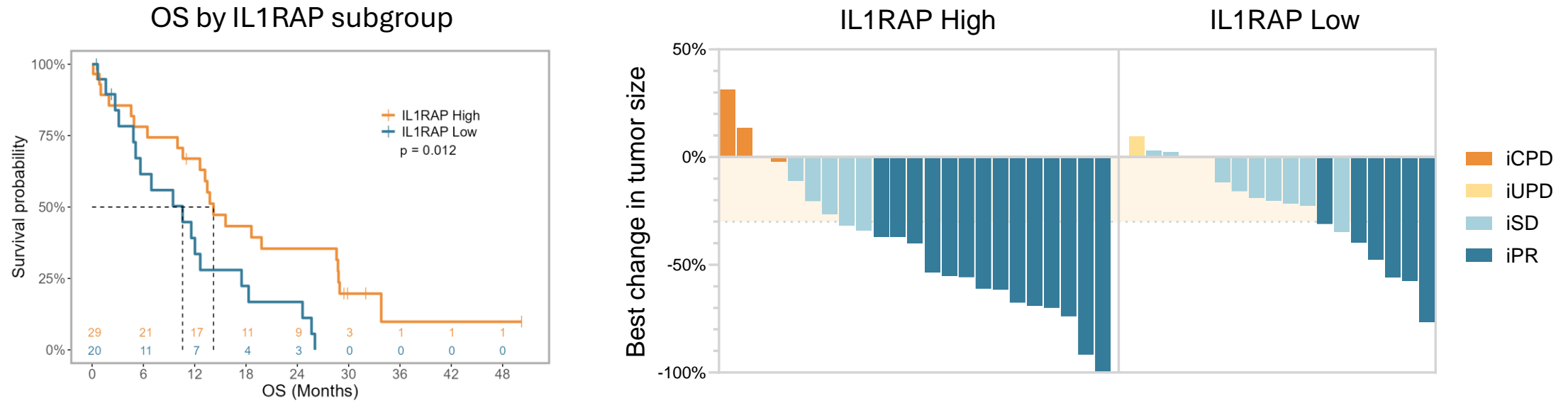


Herlev biopsy protein stainings
 PDAC patients stage IV treated with
 gem/nab-pac
 DK

- IL1 family members are upregulated in PDAC tumors as compared to normal pancreatic tissue
- IL1RAP is expressed on tumor cells, cancer-associated 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

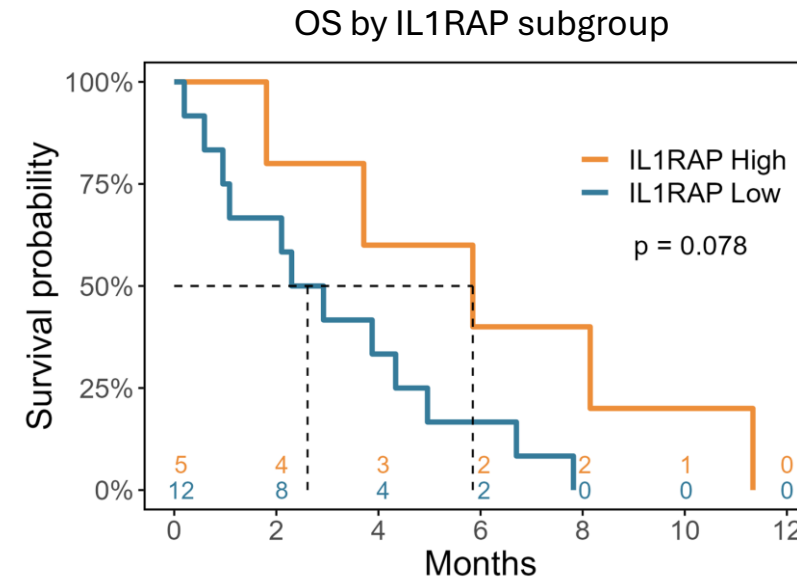
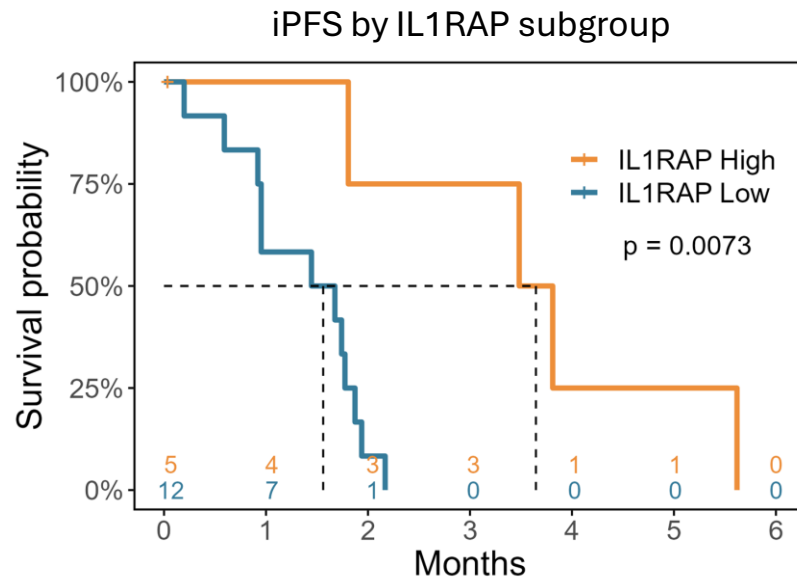
- High IL1RAP expression is a poor prognostic marker for treatment with gem/nab-paclitaxel
- Significantly prolonged OS in ILRAP High vs IL1RAP Low patients (14.2 vs 10.6 mo; p=0.012)
- Deeper and more durable responses in IL1RAP High subgroup: 11 patients had 50% or more tumor size decrease

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)

Van Cutsem et al, Clinical Cancer Research 2024

Strong efficacy reflected in “IL1RAP high” patients



Monotherapy efficacy analysis for IL1RAP High (n=5) vs IL1RAP Low (n=12) PDAC patients (late-stage, typically progressed after two lines of chemotherapy):

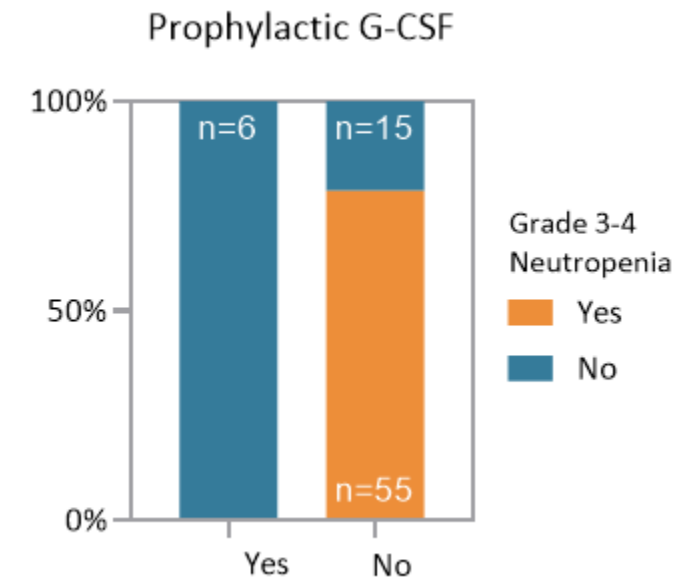
- Significantly prolonged iPFS in IL1RAP High vs IL1RAP Low patients (3.6 vs 1.6 mo; $p=0.0073$)
- Trend for OS advantage in IL1RAP High patients (5.8 vs 2.6 mo; $p=0.078$)

NADUNOLIMAB MONOTHERAPY RESULTS SUPPORT EFFECTS IN IL1RAP HIGH PATIENTS

PDAC – Manageable safety and supports MOA

- Neutropenia manageable through G-CSF prophylaxis
 - In 7 patients given G-CSF prophylaxis, only 1 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%

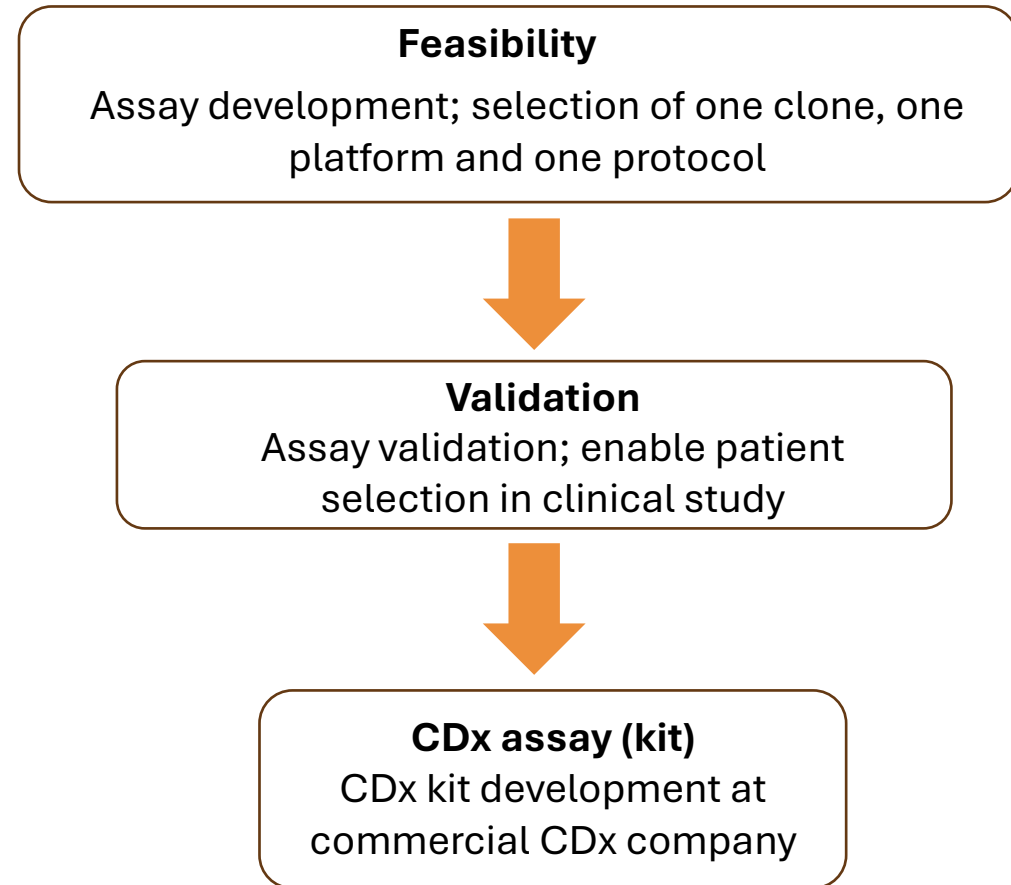


**G-CSF PROPHYLAXIS IMPLEMENTED IN FUTURE TRIALS;
POTENTIAL REDUCTIONS OF SOME SIDE EFFECTS (E.G. NEUROPATHY) TO BE DOCUMENTED IN RANDOMIZED TRIALS**

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.

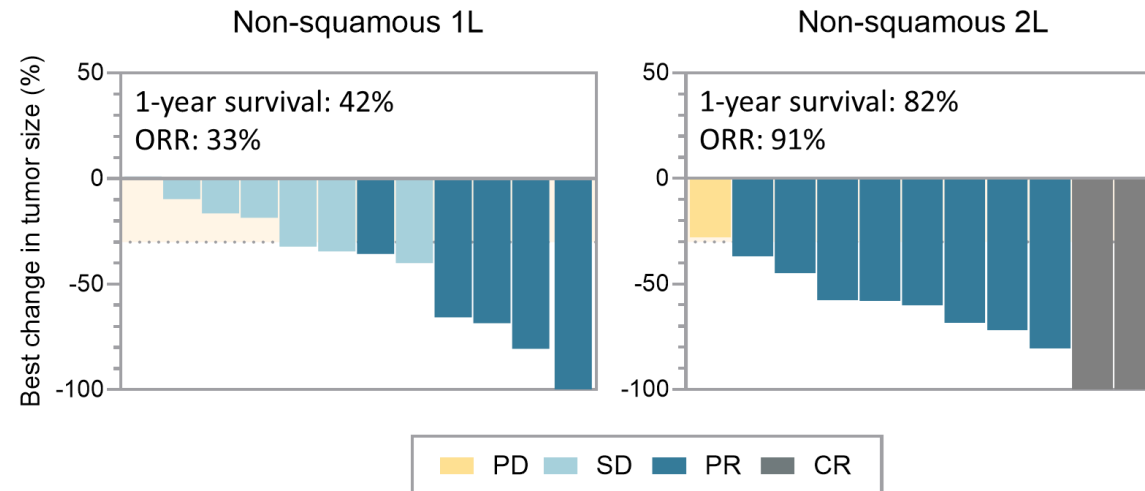
PDAC – Next steps

- Metastatic PDAC patients selected for high IL1RAP expression
- Treatment naive patients
- Combination with gemcitabine/nab-paclitaxel
- Controlled study with 2-dose lead-in
- Recruitment in Europe and US
- Primary read-out: OS
- Interim analysis opens for early MAA and supports the start of the CDx assay kit development



PHASE 3 DESIGN IN SELECTED PATIENTS WITH FAST TO MARKET APPROACH

NSCLC – Strongest effects in patients no longer responding to PD(L)-1



	Non-squamous	
Efficacy parameter (95% CI)	1L (n=15)	2L (n=11)
OS ; median, months	11.6 (5.8-22.0)	26.7 (6.2-NE)
PFS ; median, months	6.3 (2.7-11.3)	10.4 (5.3-22.2)
1-year survival*	42% (16-65)	82% (45-95)
ORR	33% (12-62)	91% (59-100)
DoR ; median, months	9.9 (4.4-NE)	9.1 (3.7-NE)

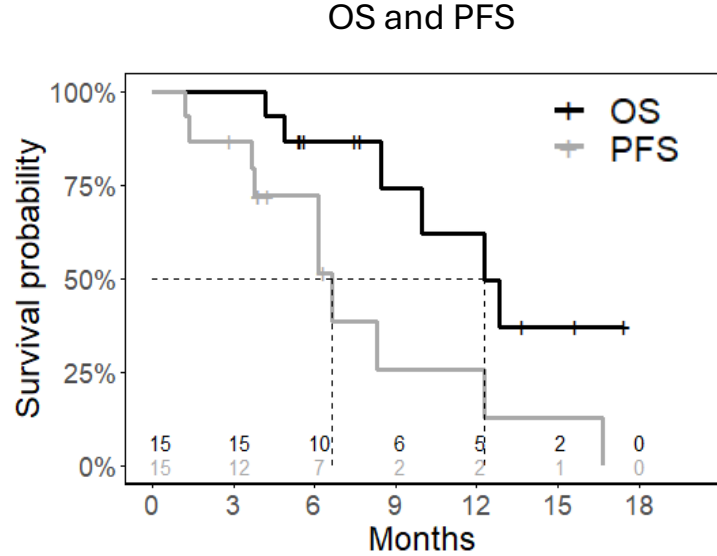
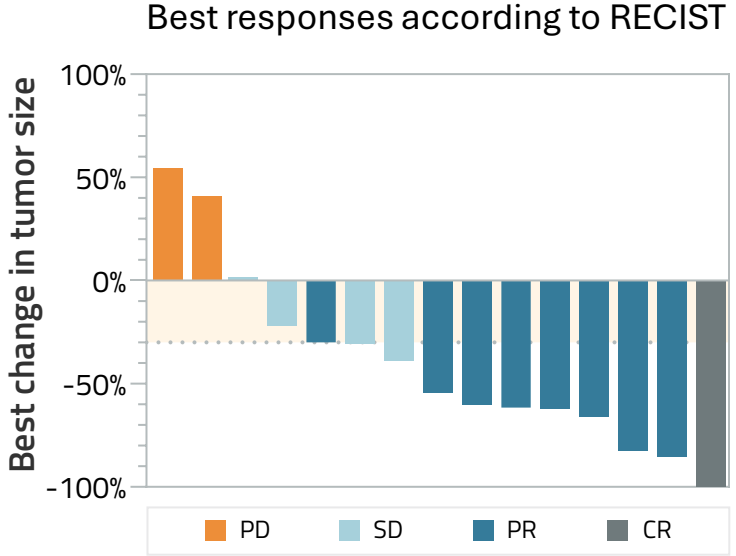
*The proportion of patients with 1-year survival is based on Kaplan-Meier estimation
NE; not estimable

SHOW VERY STRONG DATA IN 2ND LINE NON-SQ NSCLC - HIGH UNMET MEDICAL NEED

Note: subgroup analysis from 40 patients

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 dose-escalation 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

PD – Progressive Disease; SD – Stable Disease; PR – Partial Response; CR – Complete Response

A microscopic view of several cells, likely yeast or similar microorganisms, showing a textured, spherical surface. The image is overlaid with a semi-transparent blue filter. A horizontal band across the middle contains the text 'MILESTONES & INVESTMENT HIGHLIGHTS'.

MILESTONES & INVESTMENT HIGHLIGHTS

Investment Highlights

CORPORATE STRENGTH DRIVING INNOVATION

- Dedicated Team with experience from Pharma, Biotech & Academia
- Two clinical programs ongoing
- 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 phase 2 trial initiation, incl. strong endorsement from KOL's
- Broadly applicable and differentiating mechanism for treatment in inflammatory diseases

NADUNOLIMAB: CLEAR ACTIVITY IN CANCER THERAPY WITH PATH TO MARKET

- Phase 2b/3 trial in preparation in PDAC. To date >300 patients treated
- Randomized Phase 2 trial in TNBC fully enrolled (initial data H1 2025);

Upcoming milestones

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

EXTENSIVE NEWS FLOW EXPECTED DURING 2025

PDAC: Pancreatic ductal adenocarcinoma; NSCLC: Non-Small Cell Lung Cancer; TNBC: Triple Negative Breast Cancer

* US Department of Defense, The University of Texas MD Anderson Cancer Center



Thank you

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