



Blocking the **RIGHT** signals to improve treatment options

Corporate Presentation

August 2026

NASDAQ STOCKHOLM MAIN LIST (CANTA.ST)

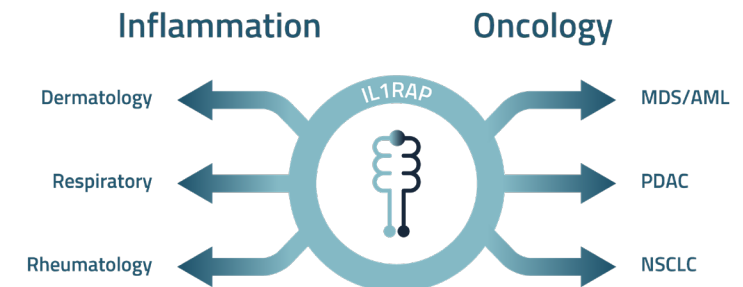
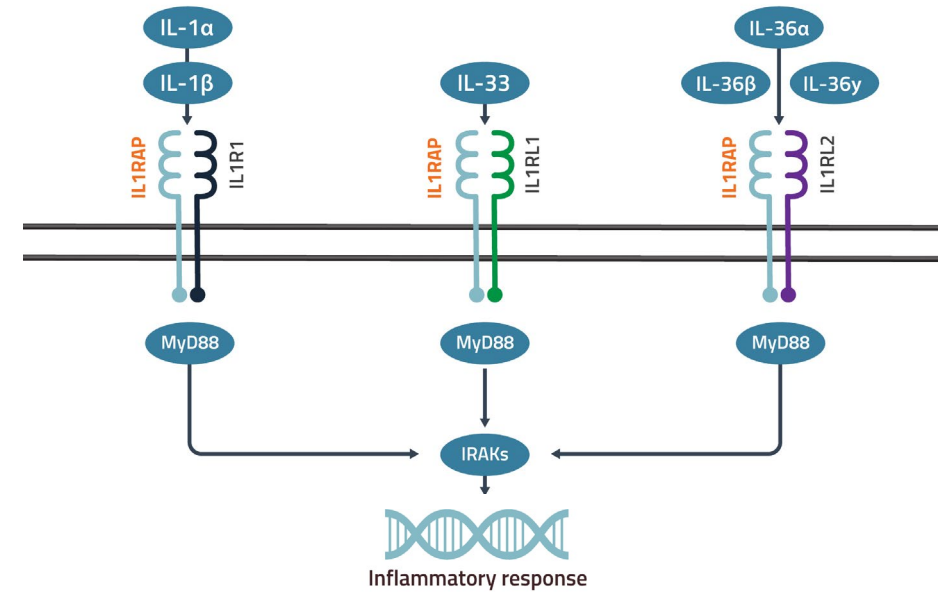
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Cantargia Is the Preeminent IL1RAP-Focused Company

- IL1 Receptor Accessory Protein (IL1RAP) is a co-receptor required for downstream signaling of IL-1, IL-33 and IL-36
- Targeting IL1RAP impacts three signaling systems to counteract redundancy and increase efficacy
- IL1RAP biology links innate and adaptive immune processes, unlocking therapeutic potential across oncology and autoimmune disease
- Preclinical data demonstrates meaningful activity in settings with high unmet medical need, including refractory inflammation and cancer
- Cantargia's CANxx program is a scalable platform for next-generation constructs, ranging from bispecific inhibitors to targeted drug conjugates (ADCs)



TARGETING A CENTRAL NODE ACROSS CANCER, INFLAMMATION AND FIBROTIC INDICATIONS

Our IL1RAP-Focused Pipeline

Asset	Target	Indication	Discovery	Preclinical	Phase 1	Phase 2	Phase 3	Partner
Oncology								
Nadunolimab	IL1RAP	PDAC	+ Daraxonrasib - Phase 1b Planned					
		MDS/AML ¹	Investigator-Initiated Trial					THE UNIVERSITY OF TEXAS MD Anderson Cancer Center [®]
CANxx	Unique IL1RAP platform ²							
Autoimmune								
CAN14	IL1RAP BsAb ³	Autoimmune diseases						
CAN10	IL1RAP	Autoimmune diseases						

IL1RAP – Interleukin-1 Receptor Accessory Protein; **PDAC** – pancreatic ductal adenocarcinoma;
MDS – Myelodysplastic Syndrome; **AML** – Acute Myeloid Leukemia; **BsAB** – Bispecific Antibody

- 1) Investigator-initiated study conducted by Texas MD Anderson Cancer Center with funding from the US Department of Defense
2) E.g. IL1RAP mAbs, IL1RAP BsAbs, IL1RAP ADCs
3) IL1RAP Bispecific Antibody, 2nd target undisclosed

Executive Management Team with Extensive Experience



Hilde Steineger
CEO



Patrik Renblad
CFO



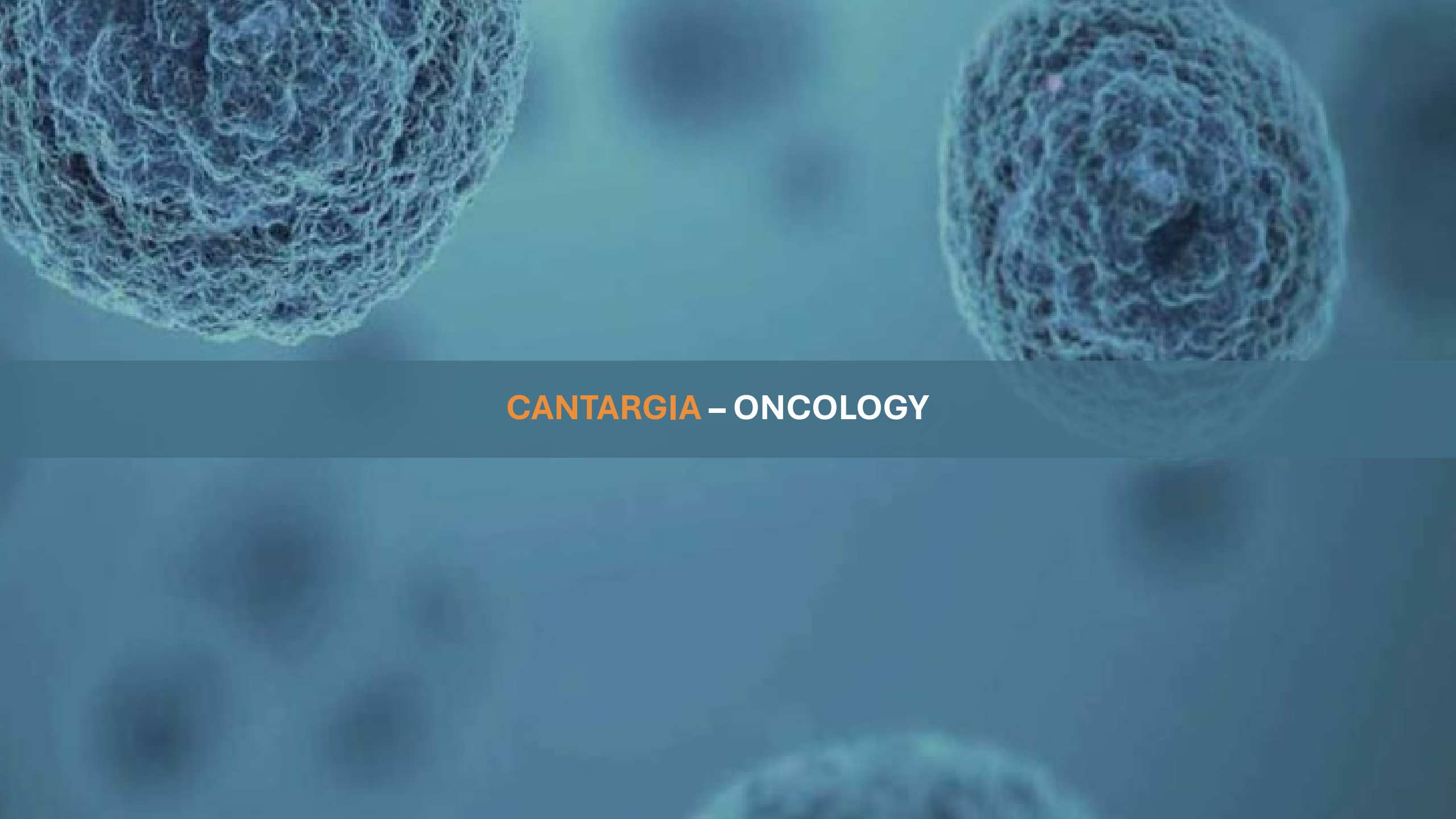
David Liberg
CSO



Ton Berkien
CBO

EXECUTIVE TEAM WITH COMPREHENSIVE INDUSTRY INSIGHT

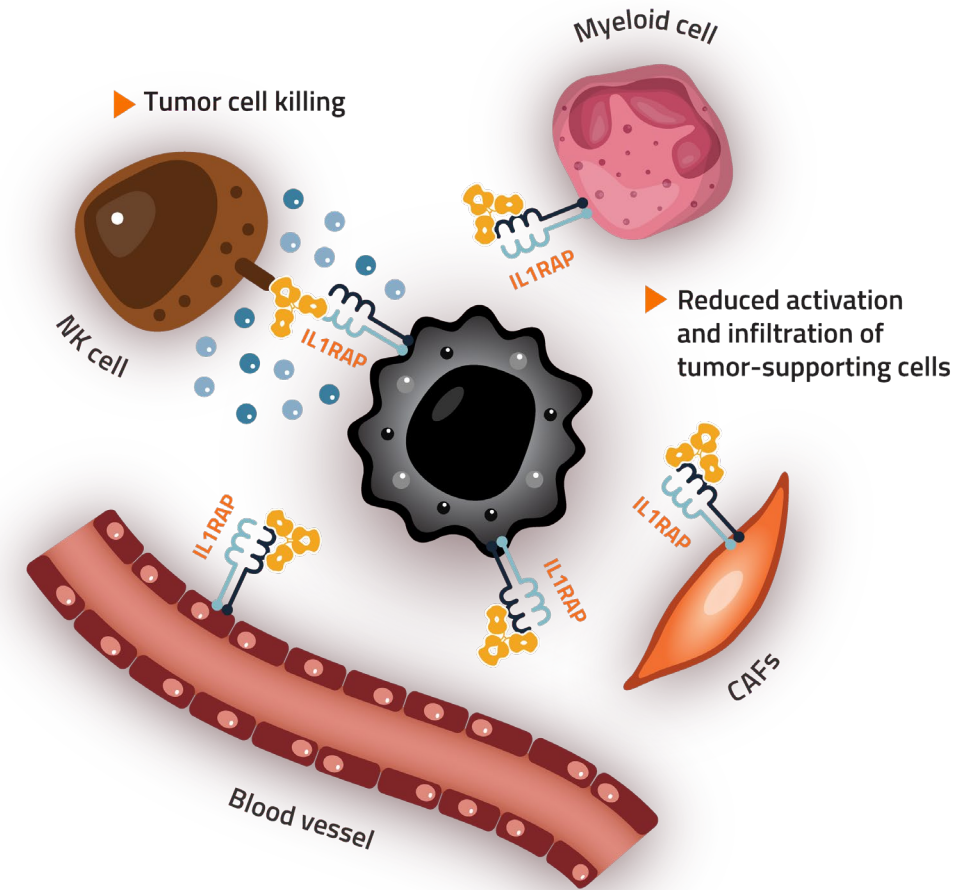


A microscopic image showing several cells with a textured, mesh-like surface. The cells are primarily blue and purple, with some showing internal structures. A semi-transparent dark blue horizontal band is positioned across the middle of the image, containing the text 'CANTARGIA – ONCOLOGY'.

CANTARGIA – ONCOLOGY

Why We're Pursuing IL1RAP with Nadunolimab in Oncology

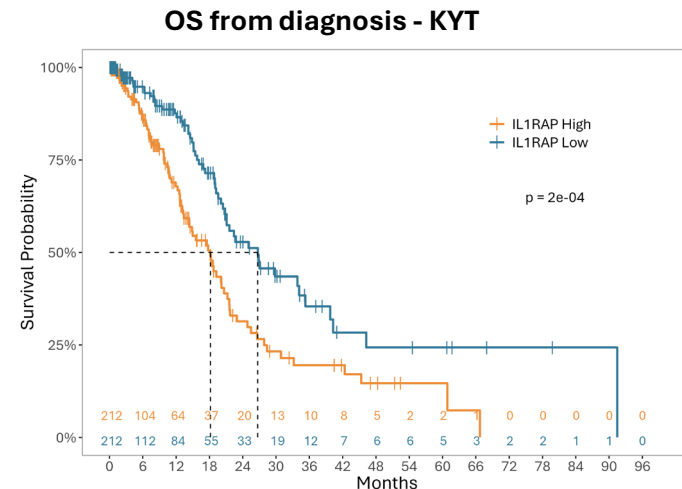
- **High expression of IL1RAP strongly correlates to poorer prognosis** in multiple tumor types
- **Intricately involved in tumorigenesis** across multiple downstream pathways; Transcription factor NFκB and AP-1 are associated with cellular survival, proliferation, metastasis, angiogenesis and chronic inflammation
- **Acts as a gatekeeper** in the tumor microenvironment with expression on stromal cells mediating infiltration of neutrophils/monocytes and establishing tumor-supportive and immunosuppressive TME
- **Highly combinable**; the IL1RAP circuitry is involved in immunosuppression and chemoresistance and may have strong roles in adaptive resistance to kinase-inhibitors or RAS-inhibitors
- **Nadunolimab's mechanism is multi-pronged** interdicting the intracellular signaling in both tumor and supporting cells, as well as inducing ADCC/ADCP in IL1RAP-expressing cells
- **Potential to improve safety** by virtue of reducing neuropathy driven by IL-1-induced inflammation



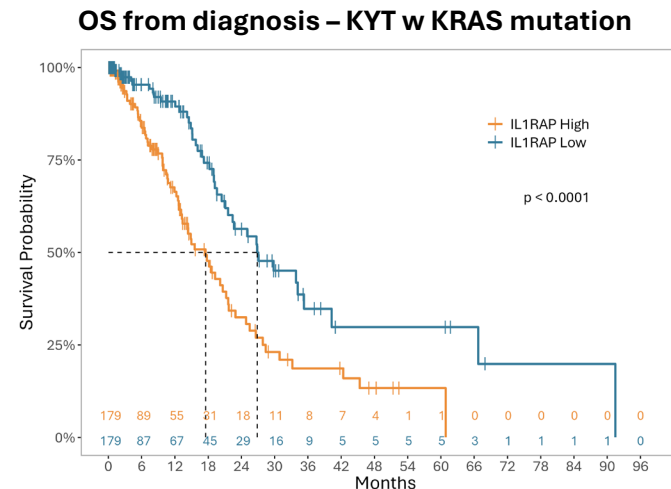
A microscopic image of pancreatic tissue, showing a dense network of cells and ducts. A central blue overlay contains the text "PANCREATIC DUCTAL ADENOCARCINOMA – PDAC".

PANCREATIC DUCTAL ADENOCARCINOMA – PDAC

High IL1RAP Expression Is Linked to Poor Outcome in Patients with KRAS mutated Pancreatic Tumors



Know Your Tumor RNA data
PDAC patients stages III/IV

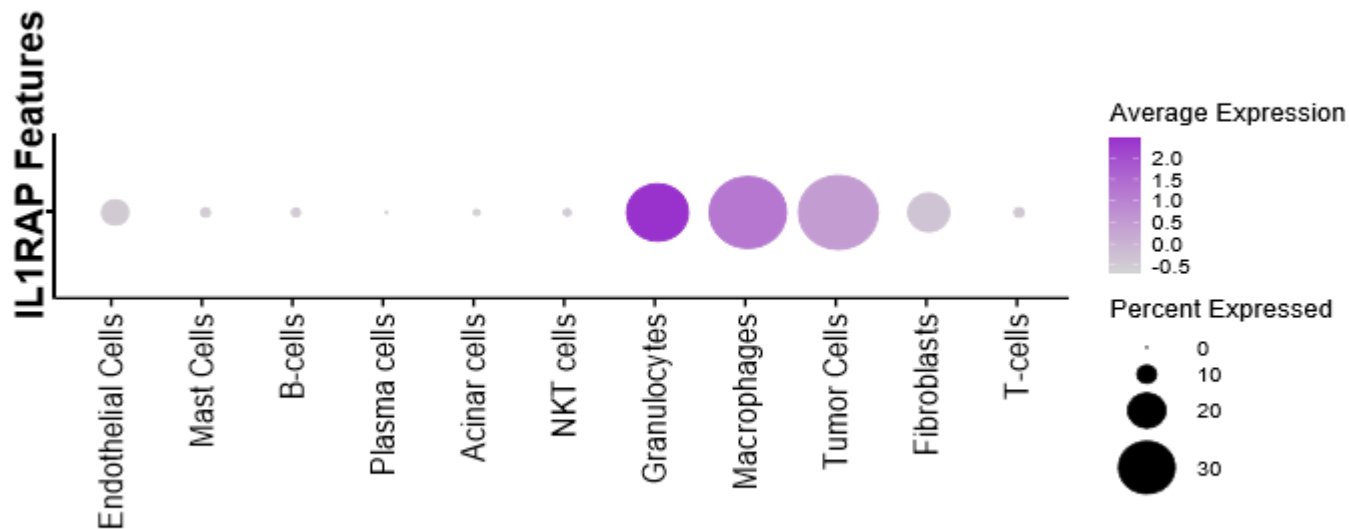


Know Your Tumor RNA data
PDAC patients stages III/IV

- High IL1RAP RNA expression in PDAC tumors correlate with poor survival
- IL1RAP levels strongly influence survival in allcomers as well as patients with KRAS mutations

HIGH IL1RAP EXPRESSION IN PDAC TUMORS IS STRONGLY ASSOCIATED WITH POOR SURVIVAL

IL1RAP Is Expressed by Tumor Cells, Myeloid Cells and Fibroblasts in the PDAC TME



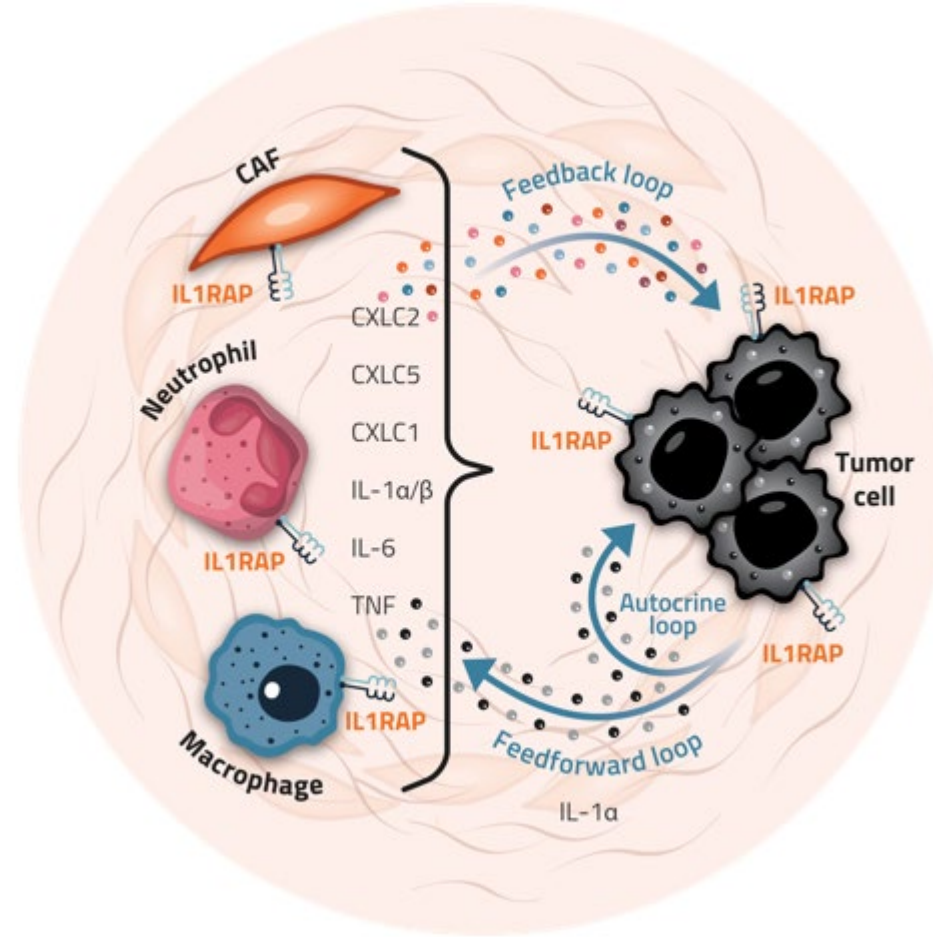
- Single-cell RNA sequencing of tumors from PDAC patients (n=16) shows IL1RAP is mainly expressed by granulocytes, macrophages, tumor cells and fibroblasts in the tumor microenvironment (TME)
- Growing body of evidence implicates myeloid cells as key mediators of resistance to RAS inhibition, as oncogenic RAS secretes chemo-attractants such as CXCR2 and CCL2, which sustain the immunosuppressive TME

Courtesy of Jashodeep Datta, MD, University of Miami, data from Steele et al Nat Cancer (2020)

IL1RAP IS EXPRESSED ON CANCER CELLS, IMMUNE CELLS AND FIBROBLASTS IN THE PDAC TME

IL1RAP Is a Fundamental Driver in the PDAC TME

- IL1RAP is expressed as a signaling receptor **on tumor cells, myeloid cells and cancer-associated fibroblasts (CAFs)**
- IL-1 cytokines are induced in tumor cells and activate myeloid cells and CAFs in the TME, **feedforward signaling**
- Myeloid cells and CAFs secrete mediators that stimulate tumor cells, **feedback signaling**
- IL-1 family cytokines induce collagen formation by CAFs and contribute to establishing a **fibrotic, desmoplastic stroma**
- Activated CAFs attract myeloid cells that fuel the tumor inflammation and create an **immune suppressive and treatment resistant niche**



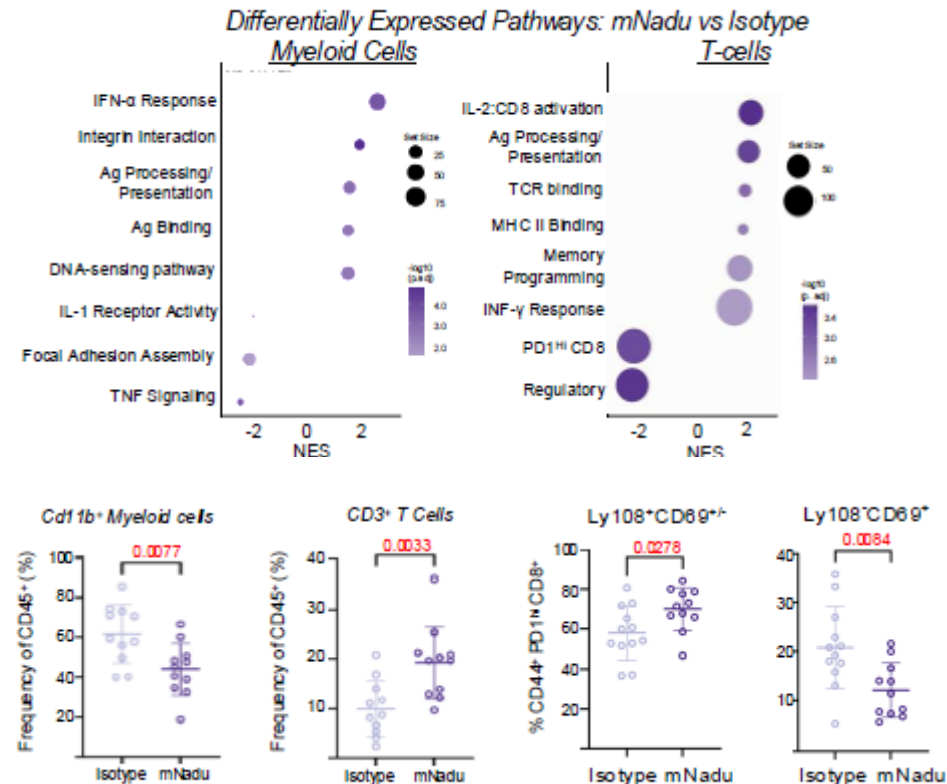
Onco-immune network mediates:

- Tumor survival
- Proliferation
- Migration/invasion
- Chemoresistance
- Immune suppression

IL1RAP IS A KEY FACTOR FOR PDAC TUMOR GROWTH, IMMUNE SUPPRESSION AND THERAPEUTIC RESISTANCE

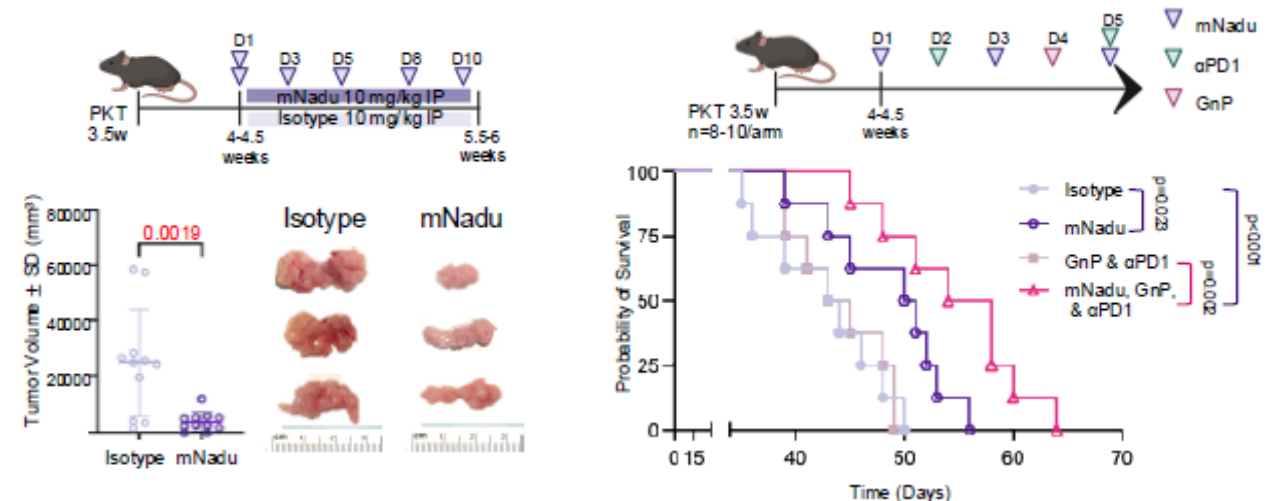
IL1RAP Targeting has Distinct Effects in an Aggressive and Treatment Resistant Preclinical Model of PDAC

m-Nadunolimab reprograms the TME in PKT mice



potently inhibits tumor growth

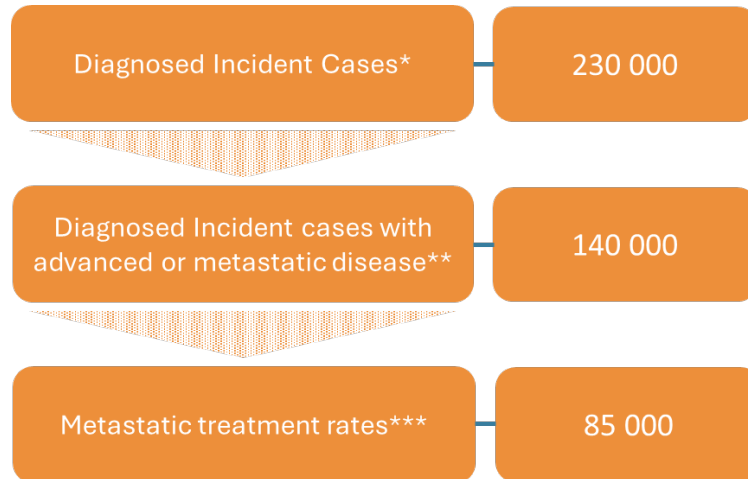
and alleviates immune suppression



In collaboration with Jashodeep Datta, MD PhD, Sylvester Comprehensive Cancer Center, U Miami Miller School of Medicine; PKT mice (Ptf1aCre/+;KrasG12D/+;Tgfb2fl/fl) develop aggressive desmoplastic pancreatic cancer, mNadunolimab = nadunolimab murine surrogate antibody

NADUNOLIMAB SURROGATE INHIBITS TUMOR GROWTH, REPROGRAMS THE TME AND ALLOWS ACTIVITY OF ICI IN A MOUSE MODEL OF PDAC

PDAC: A Deadly Cancer with Limited Treatment Options



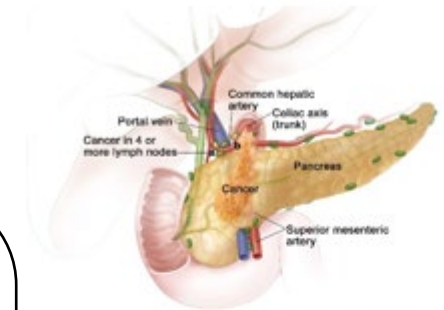
LOCALLY ADVANCED OR METASTATIC DISEASE

Median survival:

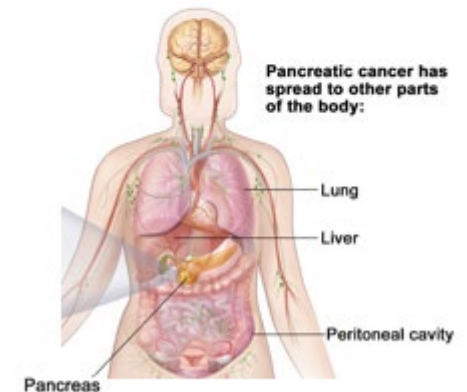
→ 8.5 – 11.7 mo

Treatments:

- Gemcitabine + nab-paclitaxel
- Gemcitabine if condition worsens
- FOLFIRINOX only if good patient condition status
- Jan 2023: 1st line NALIRIFOX
- New products in development have increased sentiment for PDAC treatments. Daraxonrasib from Revolution Medicine is expected to be approved for 2nd Line in Q3 2026.



Stage III



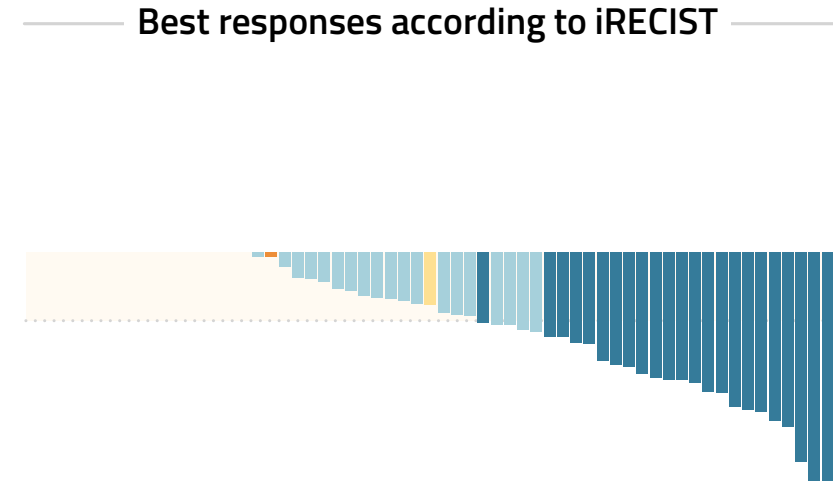
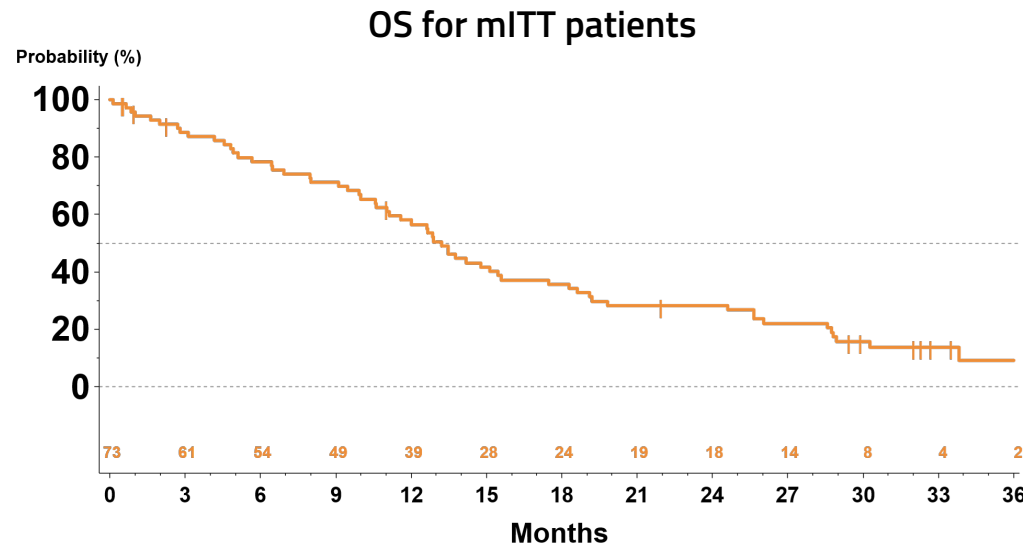
Stage IV

Images from National Cancer Institute

CURRENT DEVELOPMENT FOCUSES ON FIRST-LINE METASTATIC DISEASE

*8MM - 2024 **Stage III unresectable/Stage IV ***1L and 1L maintenance (at 60%)
Source: Global Data, Pancreatic Cancer: Eight-Market Drug Forecast , July 2025

Positive Nadunolimab Efficacy Signals in 1st Line PDAC



Nadunolimab combination with gemcitabine/nab-paclitaxel (GN) in 1st line metastatic PDAC (n=73):

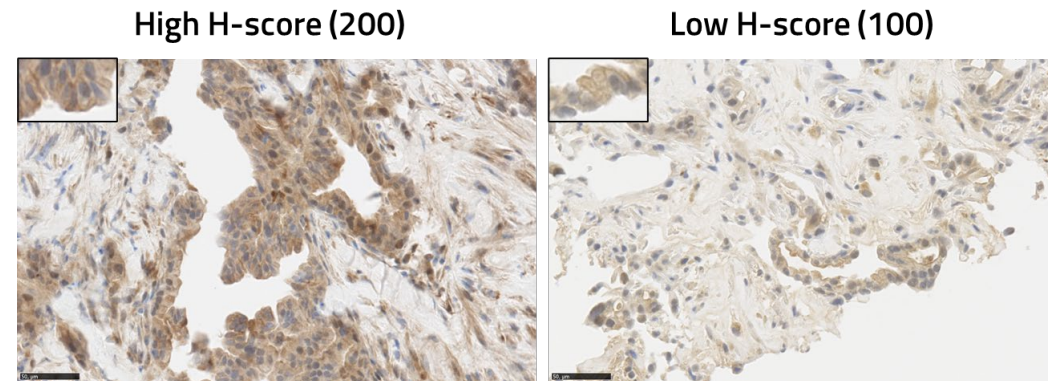
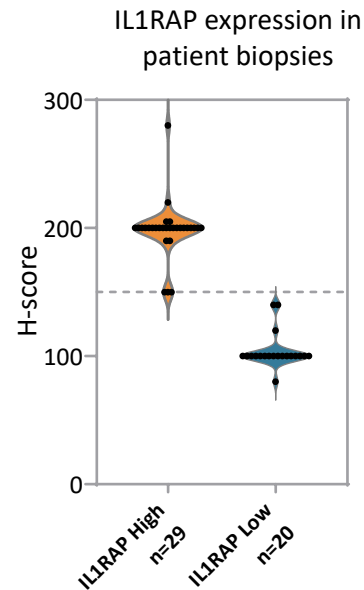
- 32% response rate
- Additional 5 (7%) patients had on-treatment benefit beyond progression
- **Promising OS (13.2 mo)**

LONGER OS THAN EXPECTED GIVEN HISTORICAL CONTROL IN PDAC

Benchmark gemcitabine/nab-paclitaxel: OS 8.5 mo (MPACT, N Engl J Med 2013); OS 9.2 mo (NAPOLI-3, Lancet 2023)

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

IL1RAP Expression in Tumor Biopsies from PDAC Patients

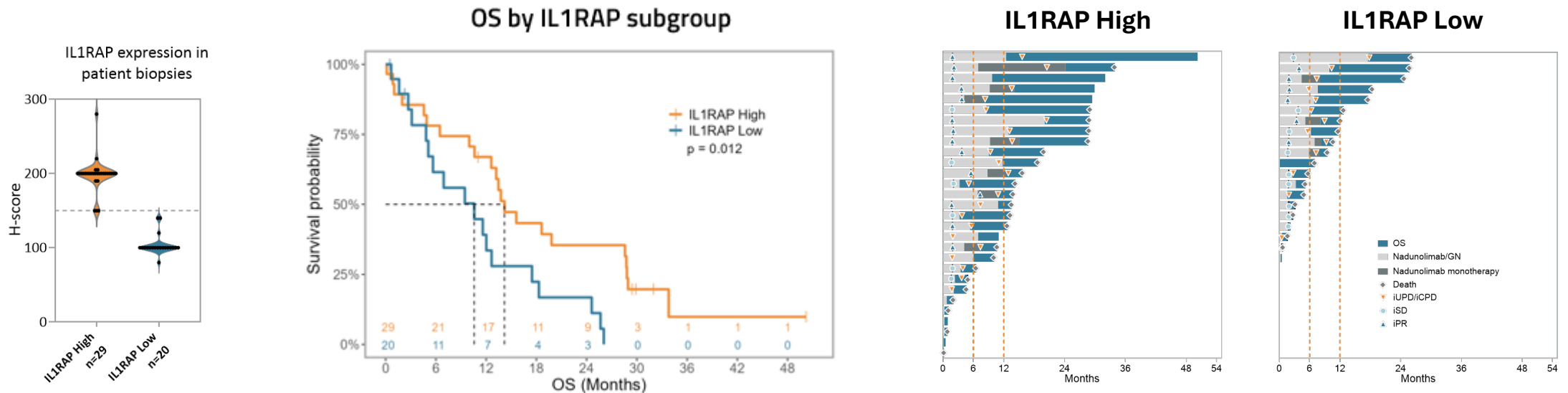


Differential expression of IL1RAP on tumor cells at baseline in PDAC patients:

- IL1RAP expression on tumor cells analyzed by IHC on baseline pre-treatment patient biopsies (n=49)
- Patients were separated in two groups based on IL1RAP expression: High (n=29) vs IL1RAP Low (n=20)
- Correlation between IL1RAP expression levels and survival

PATIENT BIOPSIES HAVE VARYING IL1RAP EXPRESSION LEVELS ON TUMOR CELLS

Stronger OS Benefits in High IL1RAP-Expressing Patients (14.2 vs. 10.6 Months)



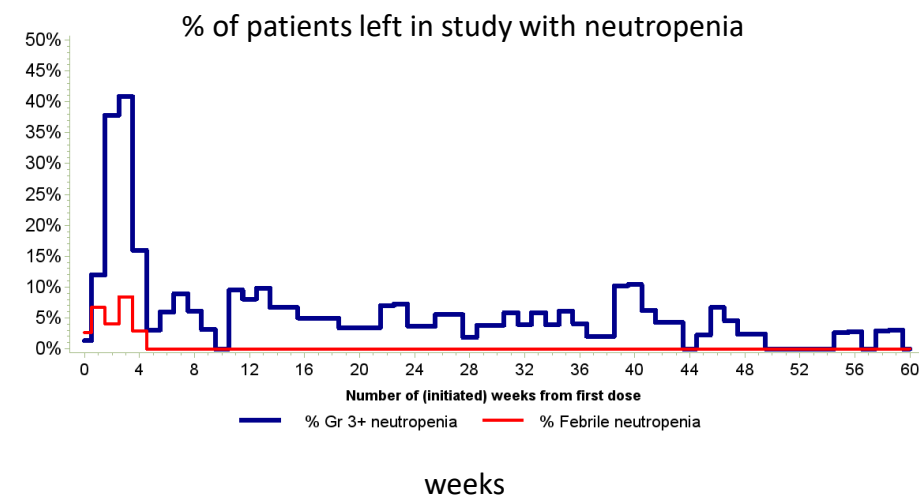
Efficacy analysis for IL1RAP High (n=29, 60%) vs. IL1RAP Low (n=20, 40%) in PDAC patients

- High IL1RAP expression is a marker for poor prognosis after treatment with gem/nab-paclitaxel
- Significantly prolonged OS in ILRAP High vs. IL1RAP Low patients when treated with nadunolimab + gem/nab-paclitaxel (**14.2 vs. 10.6 mo**; p=0.012)
- 12- & 24-month OS of **67% & 35%** in High IL1RAP group

IL1RAP-HIGH PATIENTS SHOW THE STRONGEST BENEFIT

Safety Profile CANFOUR PDAC

Grade 3 or higher AEs	Nadunolimab +Gem/nab-paclitaxel CANFOUR (n=76)	Gem/nab-paclitaxel MPACT, 2013 (n=421)	Gem/nab-paclitaxel NAPOLI 3, 2023 (n=379)
Neutropenia	66%	38%	24%
Leukopenia	24%	31%	5%
Thrombocytopenia	13%	13%	4%
Febrile neutropenia	13%	3%	2%
Anemia	14%	13%	17%
Fatigue	8%	17%	5%
Diarrhea	3%	6%	5%
Peripheral neuropathy	1%	17%	6%

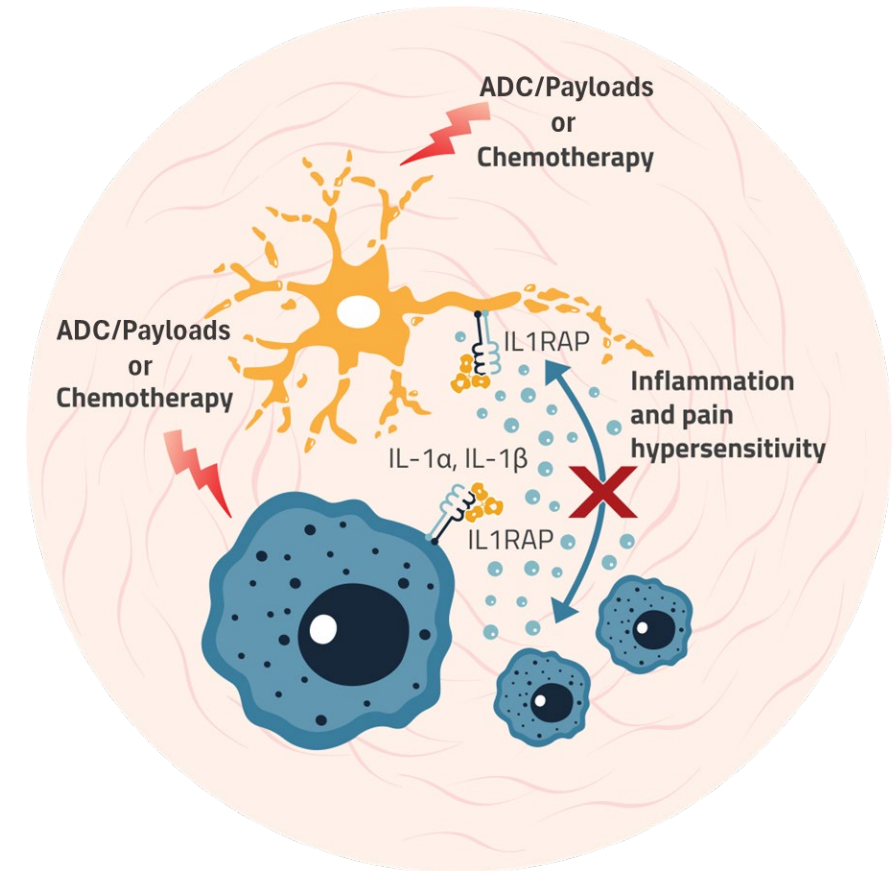


- Elevated levels of neutropenia in patients treated with nadunolimab + chemotherapy
- Only seen in combination with chemotherapy, not as monotherapy
- Effect most prominent in first cycle
- 6 patients were given G-CSF prophylaxis in CANFOUR, none developed grade 3-4 neutropenia
- Only 1 patient with peripheral neuropathy grade 3-4

Most common AEs leading to (any study) treatment discontinuation: general disorders or nervous system disorders.
One patient discontinued any study treatment due to neutropenia.

IL1RAP and Alleviation of Neuropathy

- Chemotherapy and ADCs induce neuropathy by several pathways including IL-1 (neuroinflammation)
- High levels of inflammatory cytokines such as IL1 β have been shown to correlate with higher risk of chemotherapy induced neuropathy¹
- Preclinically, anti-IL1RAP mAb completely blocks chemotherapy induced peripheral neuropathy in animal models^{2, 3}
- Anti-IL1RAP mAb treatment also blocks ADC payload induced peripheral neuropathy in animal models⁴



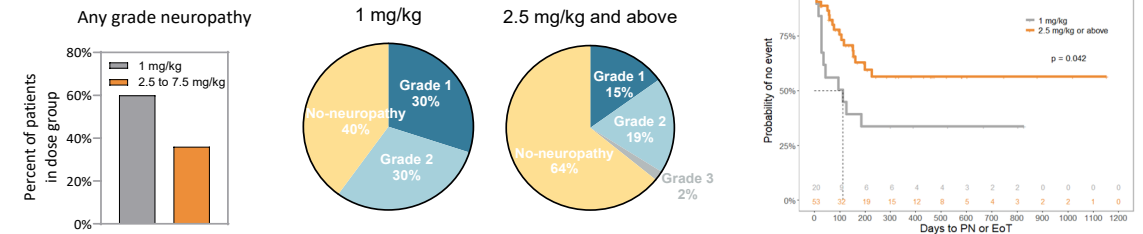
IN ADDITION TO PROMISING EFFICACY NADUNOLIMAB COULD CONTRIBUTE TO SAFER COMBINATION THERAPIES

1. Kleckner et al Breast Cancer Research and Treatment, Volume 189, pages 521–532, (2021) 2. SITC Annual meeting 2024, 3. ASCO Annual meeting 2024, 4. AACR Annual meeting 2025

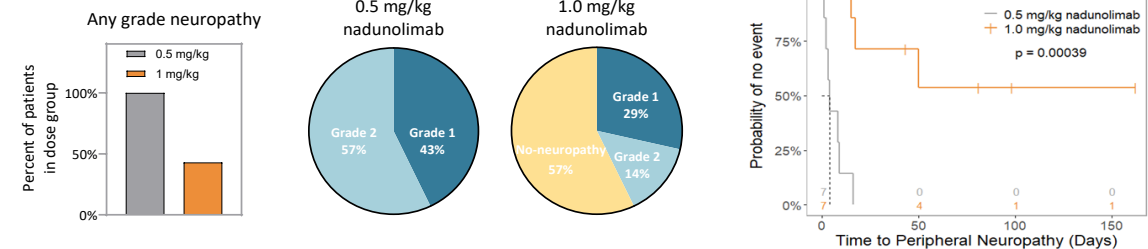
Neuropathy Clinical Data with nadunolimab

- Ph2 study data from CANFOUR in 1L PDAC patients treated with nadunolimab and gemcitabine/nab-paclitaxel
- Lower Grade 3-4 peripheral neuropathy than expected from historical controls (1% vs 17%)
- Reduced incidence as well as delayed onset of all grade neuropathies
- Correlation between nadunolimab dose level and protective effect
- Dose dependent reduction of neuropathies in patients treated with nadunolimab observed in two additional chemotherapy combinations:
 - mFOLFOX: Late-stage patients with solid tumor indications treated with nadunolimab + mFOLFOX
 - FOLFIRINOX: 1L PDAC patients treated with nadunolimab + FOLFIRINOX

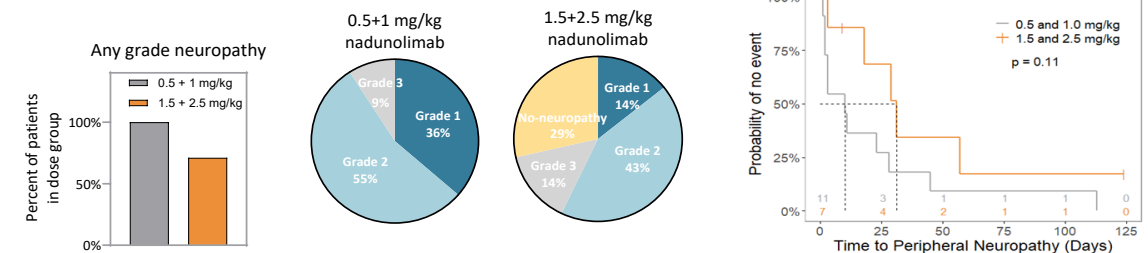
Gemcitabine/nab-paclitaxel



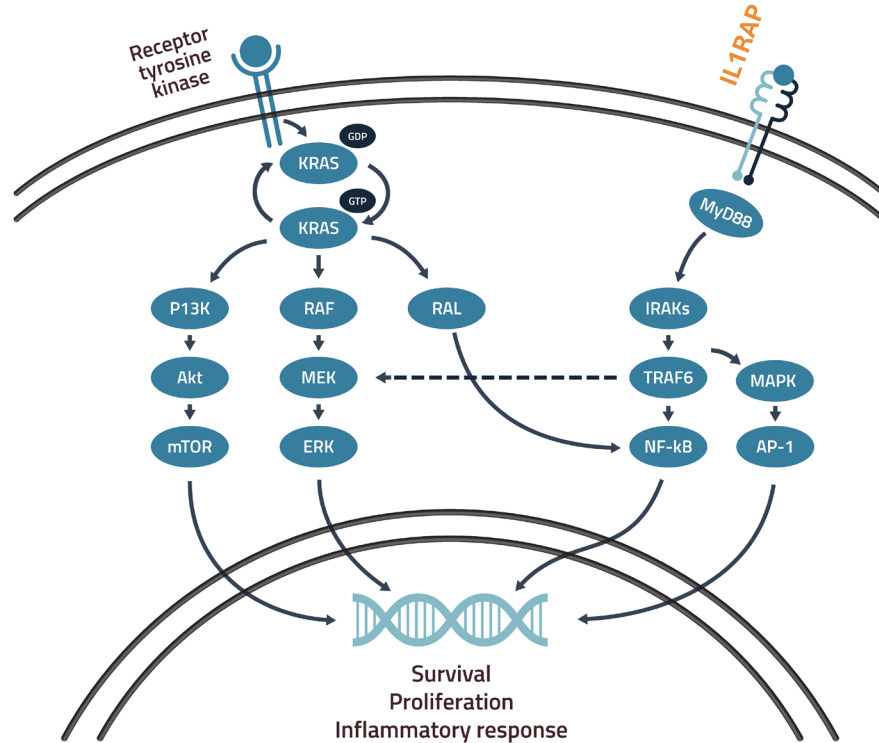
mFOLFOX



mFOLFIRINOX



Future of PDAC Management – Two Targetable Pathways with Strong Combination Potential

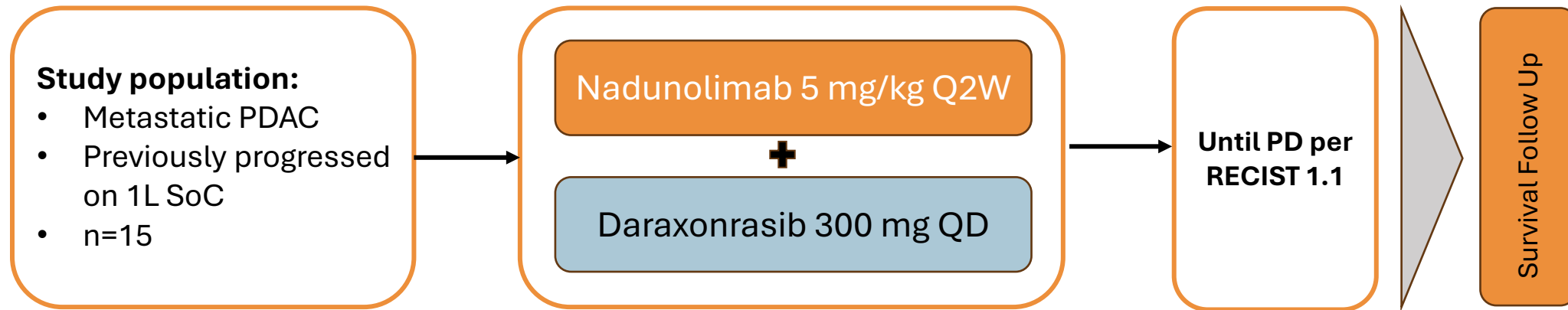


- KRAS fuels IL-1 dependent tumor inflammation
- Autocrine IL-1 activates IL1RAP and triggers tumor cell intrinsic IRAK-dependent signaling
- IL-1 signaling shapes the TME by activating IL1RAP on myeloid cells and CAFs, leading to a **self-sustaining inflammatory circuit**
- **High levels of IL1RAP strongly correlated to poor survival** in patients with or without KRAS mutations.
- **KRAS mutations (in particular G12D) correlate to higher levels of IL1RAP expression**

IL1RAP AND KRAS SIGNALING ACT IN CONCERT TO SHAPE THE PHENOTYPE OF PDAC

Preparation for Multiple Treatment Options: Ph1b/2a in Combination with Nadunolimab + RASi

An open-label Phase 1b trial designed to assess safety and tolerability of nadunolimab combined with daraxonrasib in PDAC patients who had previously progressed on 1L SoC



Primary objective:

- To determine the safety and tolerability of nadunolimab combined with daraxonrasib

Secondary objectives:

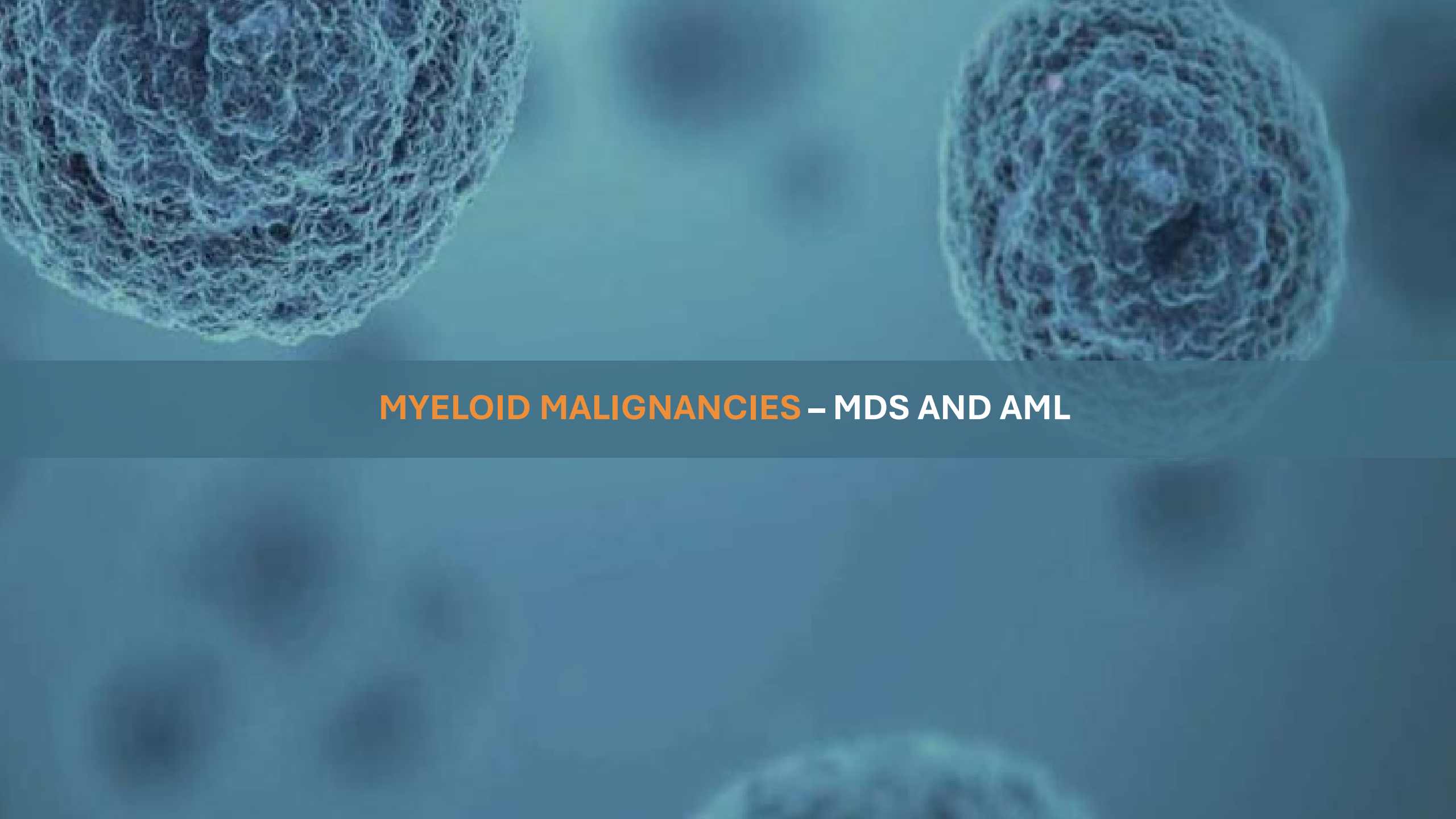
- To determine preliminary signs of clinical efficacy

Exploratory objectives:

- To evaluate disease-related inflammatory, immune or microenvironment-related parameters related to the study drugs, in the circulation and in tumor tissue

Status:

- Study to be initiated Q1 2027, subject to regulatory approval and daraxonrasib availability

A microscopic view of cells, likely blood smears, with a blue overlay. Two large, irregularly shaped cells with a textured, almost fibrous surface are prominent in the upper half. The background is a soft, out-of-focus blue. A dark blue horizontal band spans the middle of the image, containing white and orange text.

MYELOID MALIGNANCIES – MDS AND AML

Myelodysplastic Syndrome (MDS) and Acute Myeloid Leukemia (AML) – IL1RAP at the Center of Disease

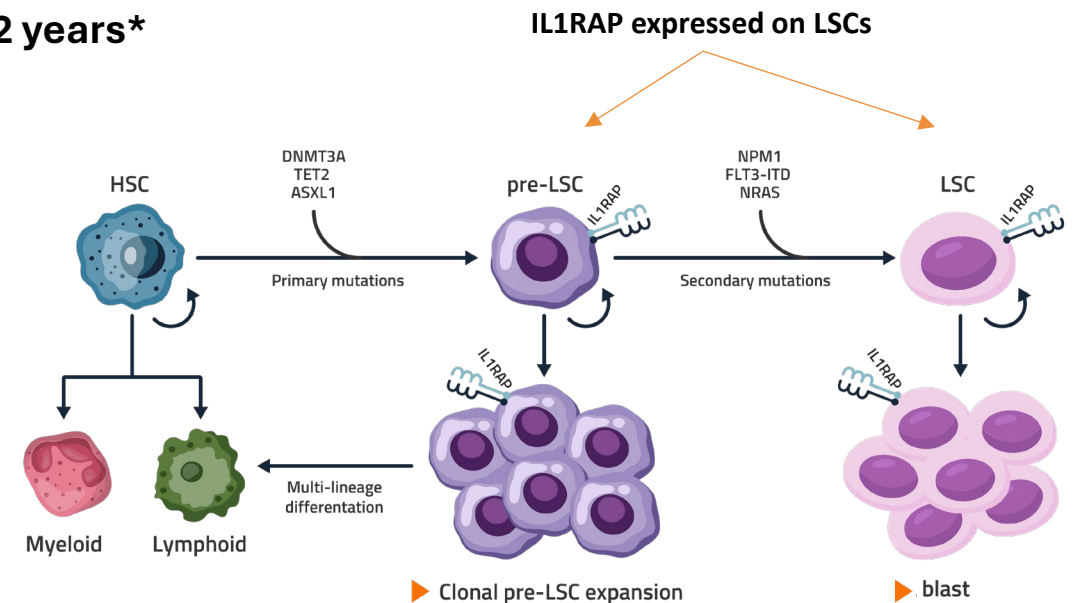
MDS:

- Originates from IL1RAP⁺ leukemia stem cells (LSC) in the bone marrow (BM)
- LSC produces immature, non-functional, blast cells, in MDS <20% of BM
- Divided into low, intermediate and high risk
- **High-risk MDS (30 - 40%) likely to transform into AML within 2 years***
- Molecularly heterogenous



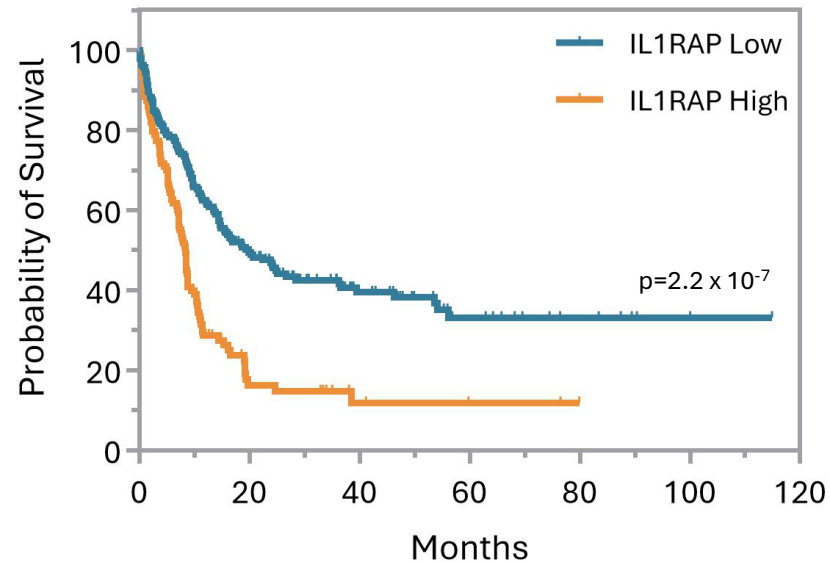
AML:

- Blast cells >20% of BM, produced by IL1RAP⁺ LSC
- Highly lethal blood cancer (10-30% survival)
- Molecularly highly heterogenous
- Main treatment: chemotherapy, stem cell transplantation
- Few targeted therapies (FLT3, IDH1/2, BCL2)



Encouraging Leukemia Data Reinforce IL1RAP as a High-Risk Target

IL1RAP in AML



IL1RAP overexpression is associated with poor clinical outcome in AML with normal karyotype. Barreyro et al., Blood 2012 .

- IL1RAP is overexpressed in leukemic disease and linked to adverse inflammatory and signaling profiles
- In AML, higher IL1RAP expression has been associated with poorer outcomes in independent clinical studies
- Supports IL1RAP as a target in patients with particularly high unmet medical need

HIGH IL1RAP EXPRESSION IN LEUKEMIC MALIGNACIES IS ASSOCIATED WITH POOR SURVIVAL

IL1RAP Expression increases with Disease Aggressiveness

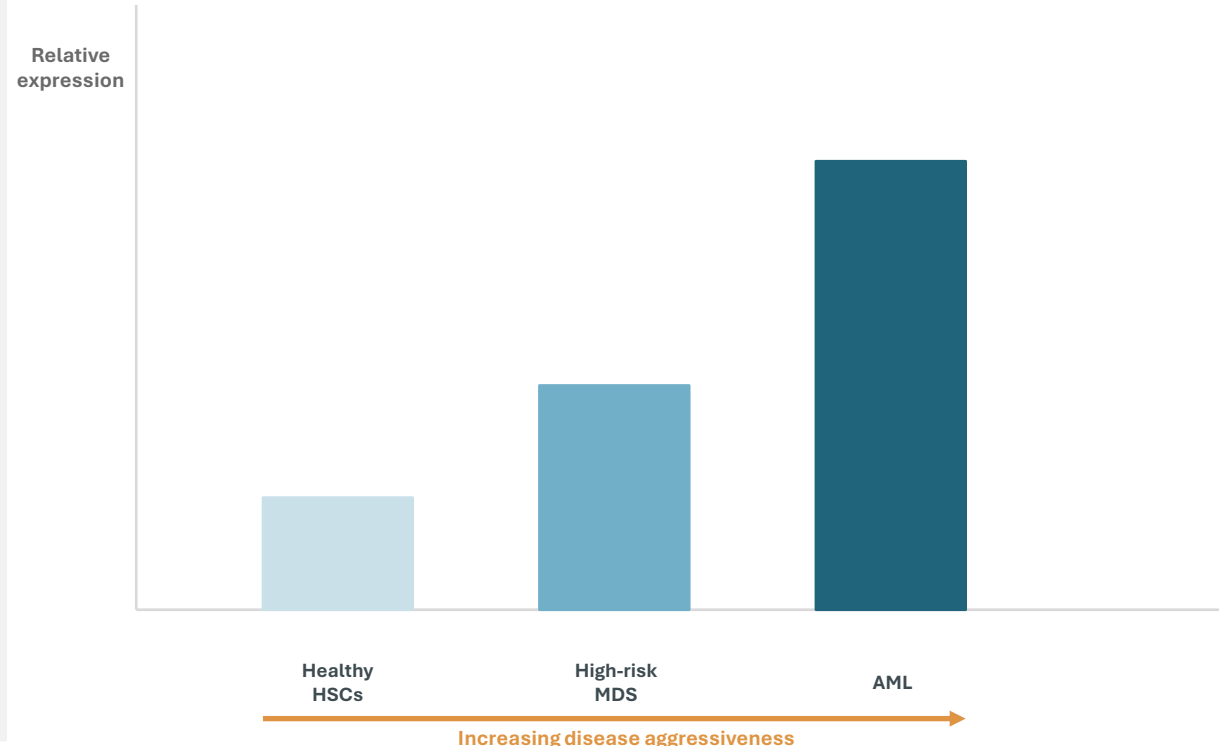
Relative IL1RAP expression across myeloid disease states

Conceptual scale based on published flow-cytometry observations

Key message

- ▶ Healthy hematopoietic stem cells (HSCs) show very low or undetectable IL1RAP expression.
- ▶ Expression increases in MDS and is most pronounced in high-risk MDS and AML.
- ▶ High IL1RAP expression is associated with leukemic stem/progenitor cells and more aggressive disease biology.
- ▶ This provides biologic rationale for IL1RAP-directed therapy in high-risk MDS and AML.

IL1RAP follows a disease continuum rather than an absolute blood reference range.



Note: Relative, conceptual scale; not absolute MFI values across studies.

IL1RAP-high biology associated with:

- ▶ Leukemic stem/progenitor cells
- ▶ Aggressive disease biology
- ▶ Therapeutic target rationale

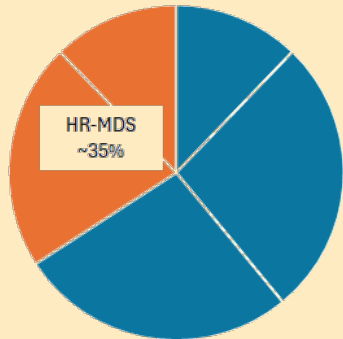
IL1RAP EXPRESSION IS LOW IN HEALTHY HSCs AND INCREASES WITH DISEASE AGGRESSIVENESS

High-risk (HR) Myelodysplastic Syndrome (MDS) comprises ~35% of all new MDS cases

Global Prevalence – MDS*

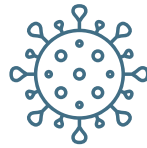


- ~400.000 MDS patients
- HR patients: ~ 35%



- mSurvival: 1 - 2 years**

Treatment (HMA)



Backbone (HMA) treatment:

- Vidaza (5-azacitidine)
- Dacogen (decitabine)
- Inaqovi/Inqovi (Decitabine cedaruzidine combination)

Treatment shift:

- From symptom mgt. toward disease modification and overall survival

Unmet need



Stem Cell transplantation

- (HSCT) - the only intervention curative potential in HR-MDS
- However, 70% of patients not eligible for transplant
- 50% do not respond to HMA; lack of effective post-HMA therapies → mOS at 5 – 6 mths***

Value



Delay leukemic (AML) progression in HR-MDS populations:

- Global HR-MDS patient population: ~140.000
- Market value: ~\$4.5 bn (2028) with projections to ~\$11.2 bn by 2034****
- Orphan Drug potential
- Large pharma: BMS, Gilead, Novartis, Abbvie

* Subject to patient status (refractory/resistant) and treatment history - TD Securities, October 2025

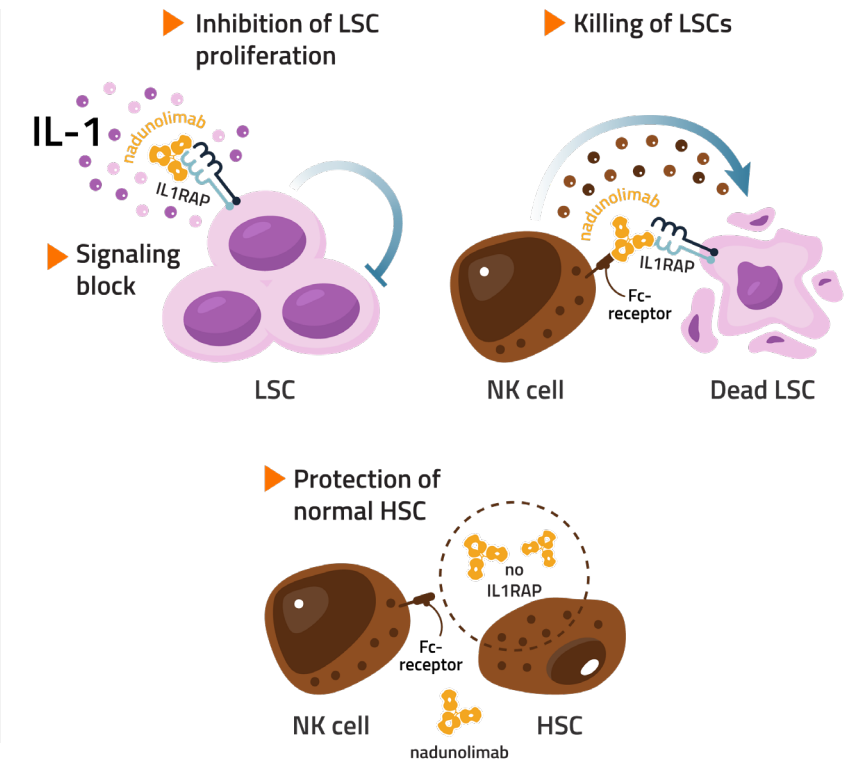
**50-75% of HR MDS patients have progressed to AML @ 5 years. Median survival rate: 0,8 – 1,6 years (Source: mdspatientsupport.org.uk) – Note: includes HR and VHR MDS patients

*** HC Wainwright, December 2025

****InnoCare Q3 2025 report

Nadunolimab in MDS and AML – Targeting the Root Cause of the Disease

- **LSCs escape current chemotherapeutic treatment strategies**, thereby providing a reservoir for disease relapse
- **Strong medical** need for targeting LSCs with specificity
- **IL1RAP is expressed in AML and high-risk MDS**
- **IL1RAP is expressed on leukemic stem cells**, but not on normal hematopoietic stem cells (HSC)
- **IL1 α/β signals through the IL1R1/IL1RAP complex** and promotes the survival of LSCs
- Nadunolimab targets IL1RAP on LSC to block proliferation and label cells for killing by ADCC



References: Askmyr et al. *Blood* (2013), Houtsma et al. *Blood Adv* (2022), Barreyro et al. *Blood* (2012); Ågerstam et al. *PNAS* (2015), de Boer et al. *Haematologica* 2021

IIT at MD Anderson: Ph1b/2a Study in MDS/AML

Principal Investigator: Gautam Borthakur, MD, MD Anderson Cancer Center

Grant support: U.S. Department of Defense (DOD)

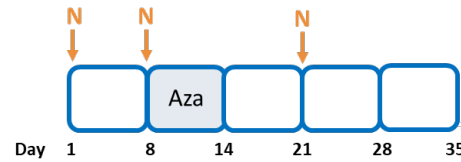
Study start 2025

Arm 1 HR-MDS (n=20):

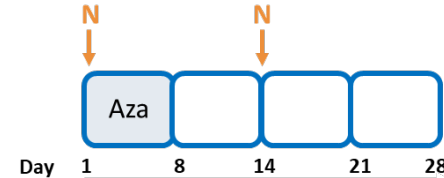
- Intermediate/high/very high risk MDS*
- Untreated or up to 2 prior treatments

*IPSS-R scoring

Cycle 1



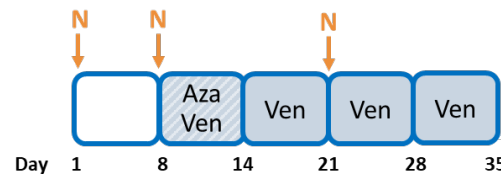
Cycle 2-24



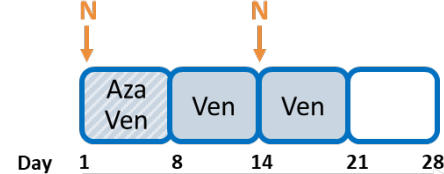
Arm 2 AML (n=20):

- Relapsed/refractory AML
- First or second salvage

Cycle 1



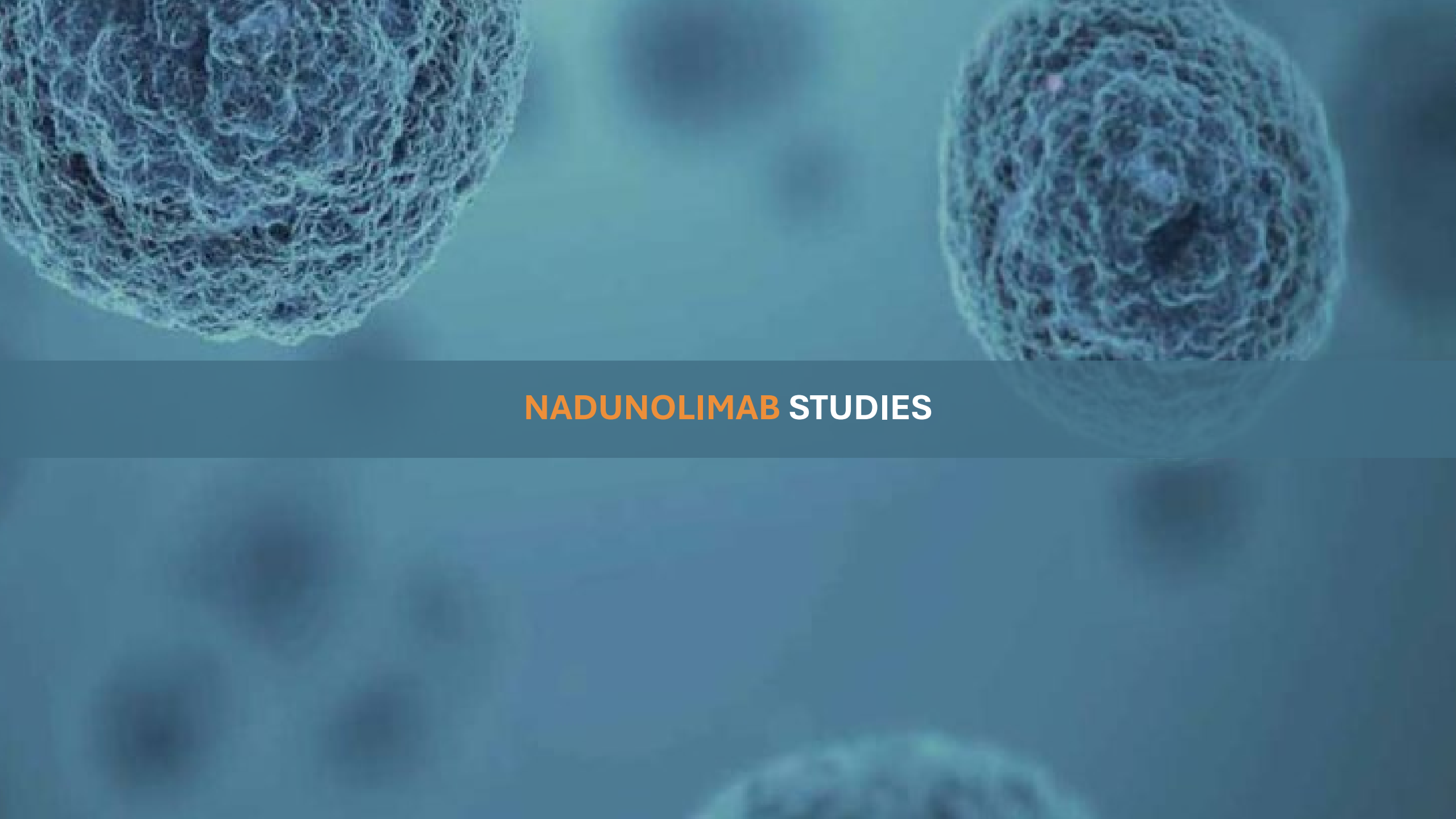
Cycle 2-24



Treatment until no protocol defined response

Study status per August 19, 2026:

- Phase 1b completed, study progressing into Phase 2a
- Nadunolimab in combination with azacitidine or azacitidine and venetoclax was generally well tolerated across both patient groups, with an acceptable safety profile
- **Notably, in the high-risk MDS cohort, 5/5 patients achieved complete remissions with the 6th pending, at a January 2026 data-cut.**

A microscopic image of cells, possibly lymphocytes, with a blue overlay. The cells have a granular texture and are arranged in a cluster. The background is a soft, out-of-focus blue.

NADUNOLIMAB STUDIES

Ongoing Investigator- Initiated Trials with nadunolimab

Study	Disease	Combination therapy	Number of patients	Status	NCT-number
Leukemia*	MDS/AML	Azacitidine and/or venetoclax	40	Recruiting	NCT06548230
Colorectal**	MSS CRC	Toripalimab (anti-PD-1)	24	Recruiting	NCT07281716
Lung Cancer Prevention**	High-risk Lung Nodules	-	Up to 59	Recruiting	NCT07284485

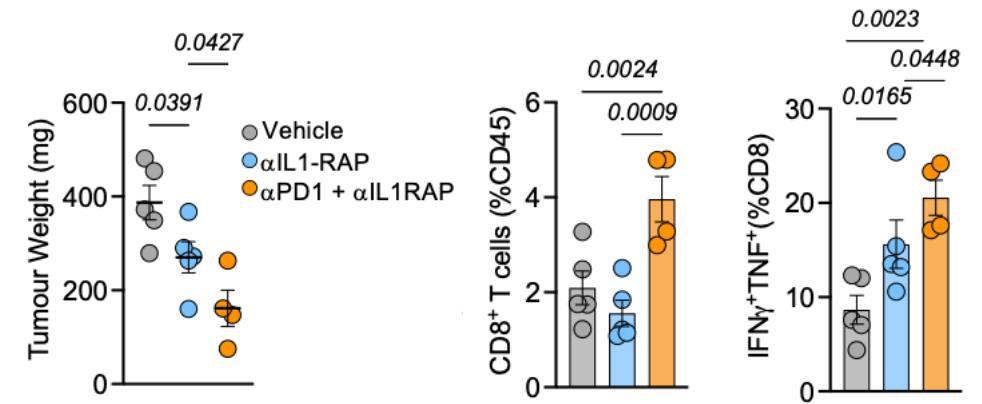
TNBC – Triple Negative Breast Cancer; **MDS** – Myelodysplastic Syndrome; **AML** – Acute Myeloid Leukemia; **MSS CRC** – Metastatic microsatellite stable colorectal cancer

*) Investigator-led study conducted by Texas MD Anderson Cancer Center with funding from the US Department of Defense.

**) Investigator-led study conducted by Mount Sinai Tisch Cancer Center, NY.

Combining Nadunolimab with Immunotherapy in Patients with Metastatic MSS CRC

- The clinical study at Mount Sinai aims to investigate the combination of nadunolimab and immune checkpoint inhibitor (ICI) therapy in patients with chemotherapy-refractory metastatic microsatellite stable (MSS) colorectal cancer (CRC)
- T cell infiltration in MSS CRC strongly correlates with improved outcome, ICI aims to activate these T cells but show limited efficacy in MSS CRC due to potent immune suppression
- Immune suppressive mechanisms in CRC involve IL1RAP-dependent cytokines and IL1RAP-expressing cell types in the tumor microenvironment
- Consistent with the above, preclinical data indicate that IL1RAP-blockade with nadunolimab in combination with ICI treatment reduce tumor burden and induce a productive T cell response in a CRC model otherwise unresponsive to ICI treatment

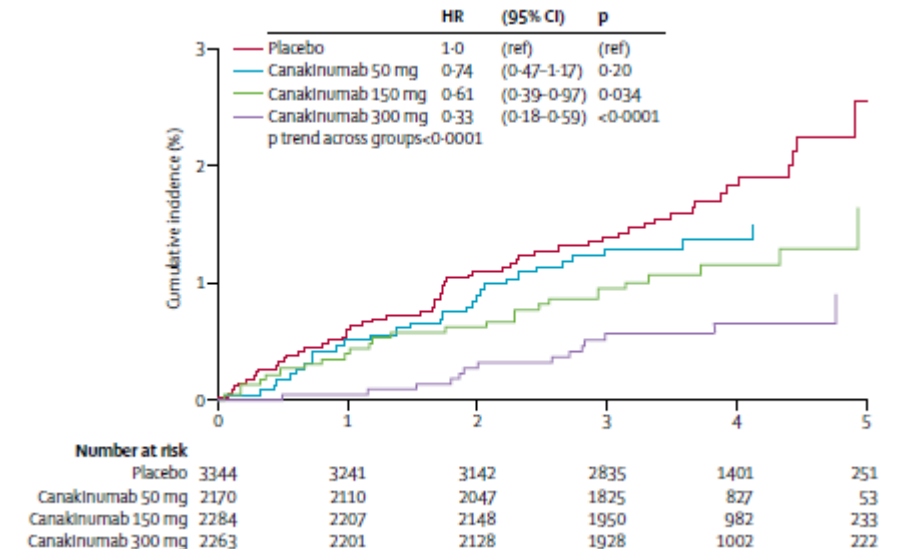


Clinical and preclinical studies performed by Mount Sinai Tisch Cancer Center, NY; αIL1RAP=nadunolimab murine surrogate antibody; CD8⁺ T cells=cytotoxic T cells; IFN_γ⁺ TNF⁺ (%CD8)=activated cytotoxic T cells

NADUNOLIMAB MAKES THE TUMOR SUSCEPTIBLE TO ICI TREATMENT BY COUNTERACTING THE IMMUNE SUPPRESSIVE TME

Treatment of High-Risk Lung Cancer Patients with nadunolimab

- The investigator initiated clinical trial at Mount Sinai aims to determine if nadunolimab can induce regression of lung nodules in patients with risk of developing lung cancer.
- There is evidence that IL-1 is involved in tumor initiation, and pre-clinical data show that early disruption of IL-1 signaling delay lung cancer progression.
- Clinical data from the CANTOS study indicate that IL-1 blockade drastically reduces lung cancer incidence in a population with no previously diagnosed lung cancer.
- The clinical trial hypothesis is that nadunolimab can enhance clearance of early lung lesions and delay development of lung cancer.



Lower incidence of lung cancer in patients treated with canakinumab compared to placebo treatment. Data from CANTOS, Ridker et al 2017.

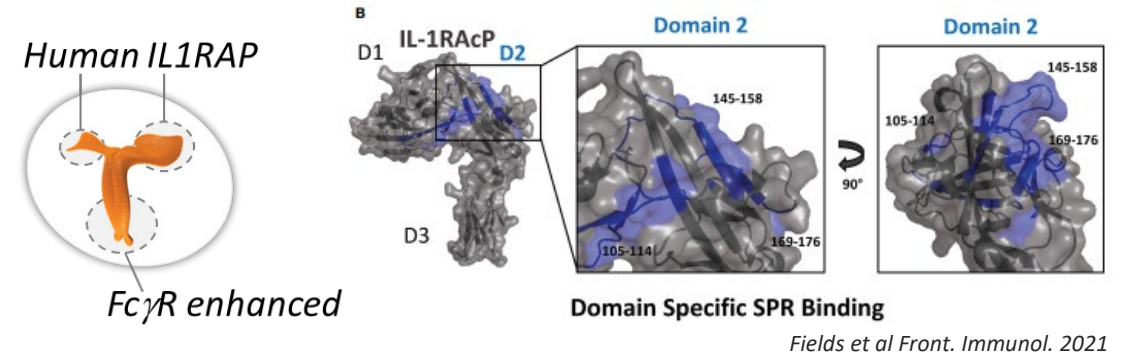
BLOCKADE OF IL-1 SIGNALING PREVENTS LUNG CANCER INCIDENCE

A microscopic image showing several cells with a blue overlay. Two cells in the upper half are in focus, showing a detailed, textured surface. The rest of the image is blurred, showing other cells in the background.

NADUNOLIMAB – PROPERTIES AND CLINICAL STUDIES

Nadunolimab mAb Properties

- Nadunolimab is a humanized anti-IL1RAP monoclonal immunoglobulin G1 (IgG1) antibody with a molecular weight of 144 kDa (non-glycosylated)
- It has 2 glycan moieties deficient of fucose in the Fc-region for enhanced antibody-dependent cellular cytotoxicity (ADCC) and antibody dependent cellular phagocytosis (ADCP)
- Nadunolimab binds domain 2 of IL1RAP with high affinity
- Nadunolimab fully blocks IL-1 α and IL-1 β signaling, and partially blocks IL-33 and IL-36 signaling in a HEK reporter cell system
- Nadunolimab induces ADCC with an IC50 in the single digit nM range, using human SK-MEL-5 melanoma cells as target cells and PBMC or NK-cells as effector cells.



Nadunolimab attributes	Details
Human IL1RAP, K_D SPR	5.62 pM
Function blocking IL-1 α /IL-1 β , IC50	2.7 nM/0.2 nM
Function blocking IL-33, EC50	7.5 nM
Function blocking IL-36 α / β / γ , IC50	0.2 nM/1.0 nM/0.3 nM
ADCC, IC50	<10 nM
Binding to Cyno, K_D SPR	5.49 pM
Binding to rat and mouse	No cross reactivity

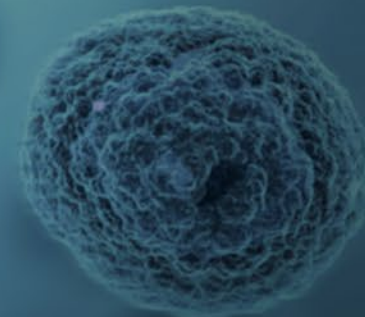
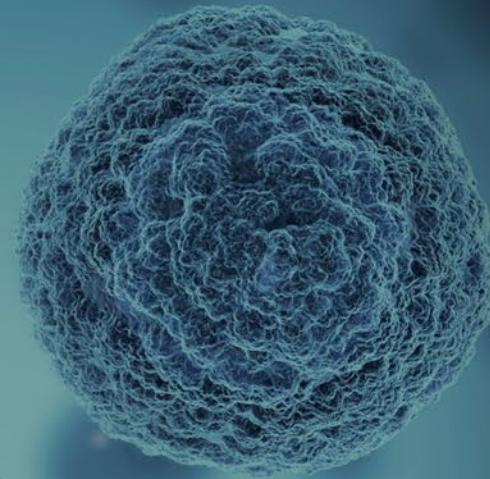
Overview of Clinical Studies with nadunolimab

Study (mITT)	NCT number	Design	Status
CANFOUR (n=113 in combination)	NCT03267316	Nadunolimab in combination with gemcitabine/nab-paclitaxel in 1L PDAC and gemcitabine/cisplatin or carboplatin/pemetrexed in NSCLC	Completed PDAC manuscript published CCR NSCLC manuscript in Lung Cancer
CIRIFOUR (n=15)	NCT04452214	Nadunolimab in combination with pembrolizumab in patients that progressed on ICI (HNSCC, NSCLC, MM)	Completed Manuscript published Invest New Drugs
CAPAFour (n=18)	NCT04990037	Nadunolimab + mFOLFIRINOX in 1L PDAC	Completed
CESTAFour (n=36)	NCT05116891	Nadunolimab in combination with 1. mFOLFOX, 2. docetaxel, or 3. gem/cisplatin. Solid tumor indications.	Completed
TRIFour (n=15 part 1, n=102 part 2)	NCT05181462	Nadunolimab in combination with gem/carbo in patients with TNBC. Control arm in part 2 (1:1 gem/carbo).	Fully recruited

CAN14 and CANXX

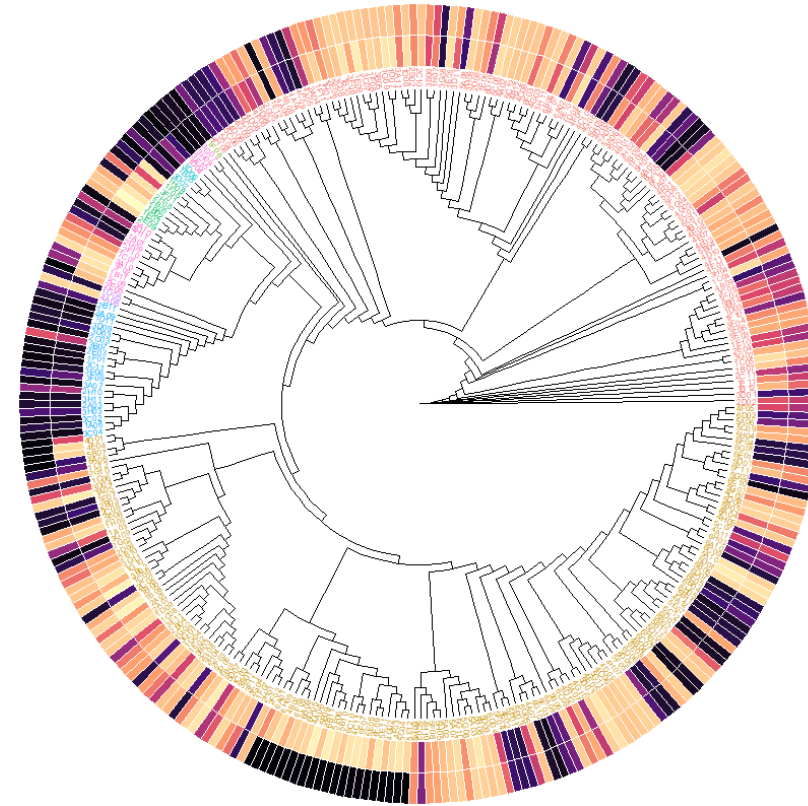
Next generation IL1RAP therapeutics

CANXX is a program for new therapeutics and reagents comprising unique antibodies, reagents and knowledge around IL1RAP as a drug target. CAN10 was the first program originating from the CANXX platform, CAN14 is the second project, adding new features to IL1RAP-blockade



CANxx – a Generator for New Drugs and Reagents

- **Knowledge** of IL1RAP and its role in disease
- **A library of ~500 anti-IL1RAP clones** within the CANxx platform
- CANxx antibodies bind to different domains of IL1RAP and have **different functional properties**



CANXX – A COMPREHENSIVE SOURCE FOR NEW DRUG PROJECTS

Next Generation IL1RAP Therapeutics

IL1RAP blockade is a potent way to block inflammation in preclinical and translational ex vivo models

Bispecific mAbs

Add new functionalities to IL1RAP blockade for stronger efficacy – tailor for specific diseases

Anti-IL1RAP

Second target

IL1RAP is expressed in a large number of solid and hematological tumors with limited normal tissue expression

ADCs

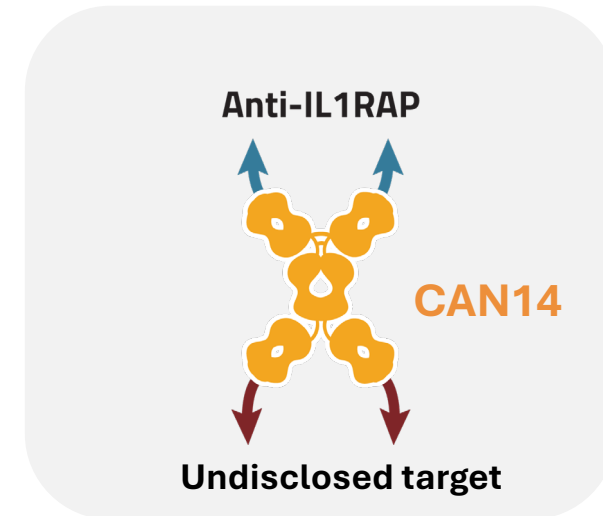
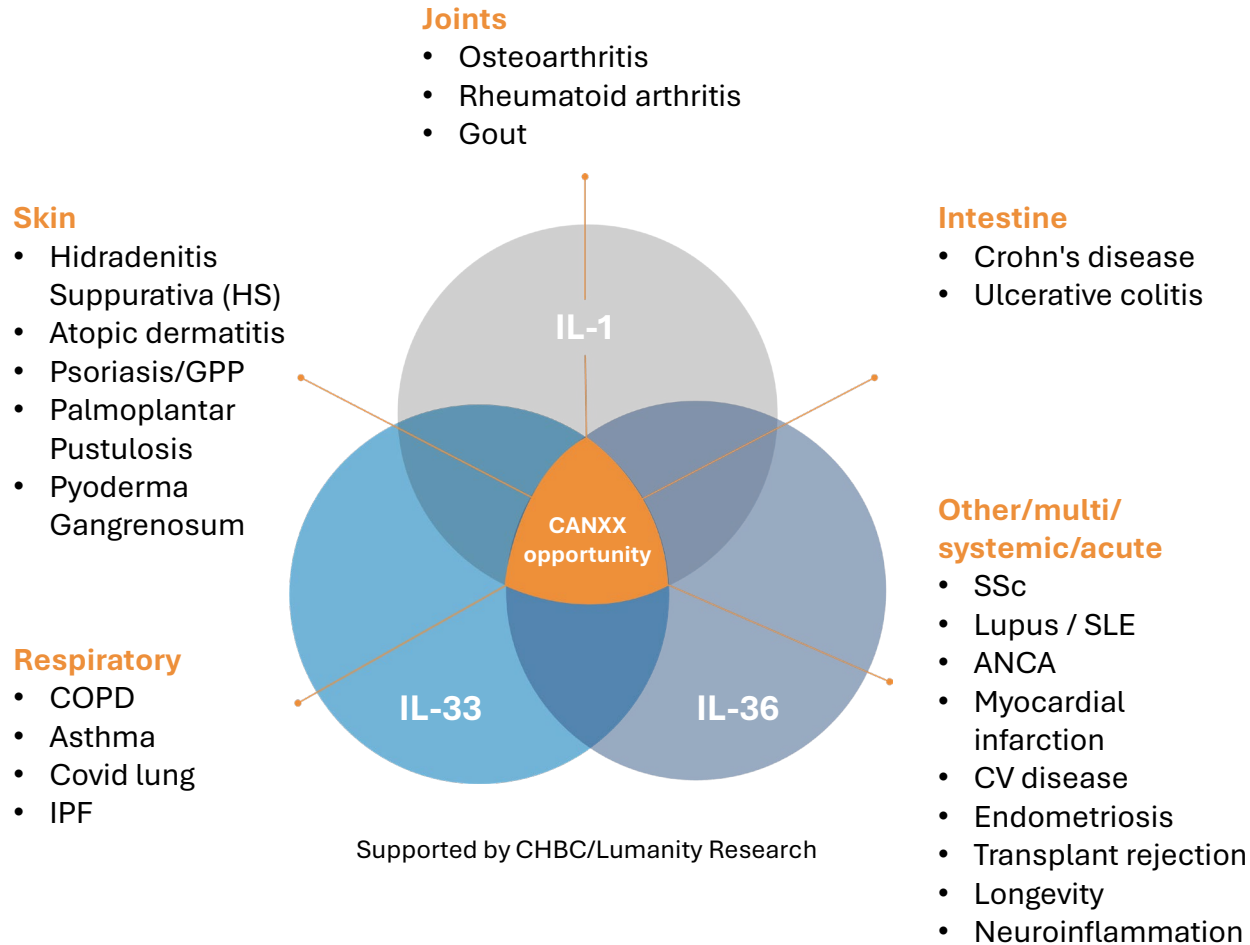
Increase efficacy and concentrate effect by combining cytotoxicity and IL1RAP-targeting in one molecule

Anti-IL1RAP

Toxic payload

CAN14 and IL1RAP-based Bispecific Antibodies

Anti-IL1RAP as a framework for efficacious treatments tailored for specific diseases

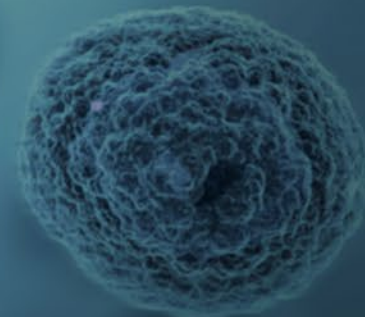
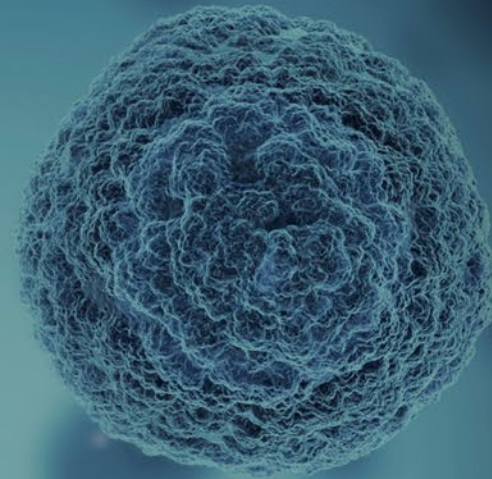


- Dual Targeting for Better Efficacy
- Overcoming Resistance or Redundance
- Targeting to specific tissues
- Candidate Selection by Year-End 2026

CAN10

Opportunities in autoimmune/inflammatory diseases

CAN10 is an Anti-IL1RAP antibody for treatment of autoimmune and inflammatory disease. By its binding to IL1RAP, CAN10 can 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.



Transformational CAN10 Deal with Otsuka



- Marks a transformative milestone for Cantargia – providing external validation of its antibody platform and CAN10 target mechanism
- Demonstrates the scientific and commercial value of Cantargia's technology in autoimmune and inflammatory diseases
- Enables long-term value creation through non-dilutive funding from upfront, milestones, and royalties
- Expands global recognition of Cantargia's R&D capabilities, paving the way for future pipeline partnerships

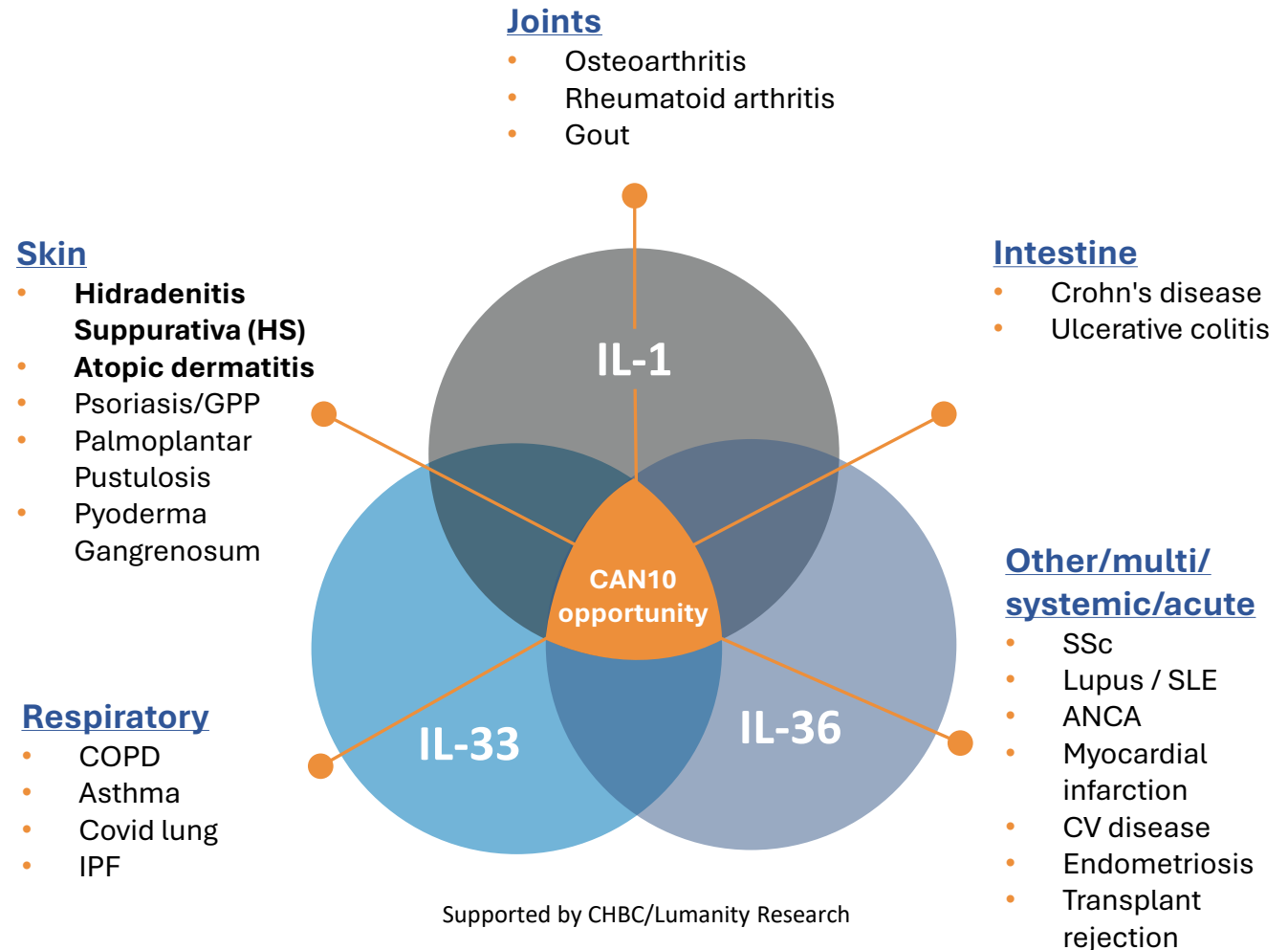
Deal Summary:

- Global development, manufacturing & commercialization rights asset purchase agreement by Otsuka, a global leader in field of neuroscience, oncology, nephrology and immunology
- Financial terms (total deal value USD **613** million):
 - Upfront payment: USD **33** million (received upon closing)
 - Development, regulatory & commercial milestone payments: up to USD 580 million
 - Royalties on net sales: double digits tiered

VALIDATION OF IL1RAP AS A TARGET IN INFLAMMATION AND OF CANTARGIA'S ANTIBODY PLATFORM

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

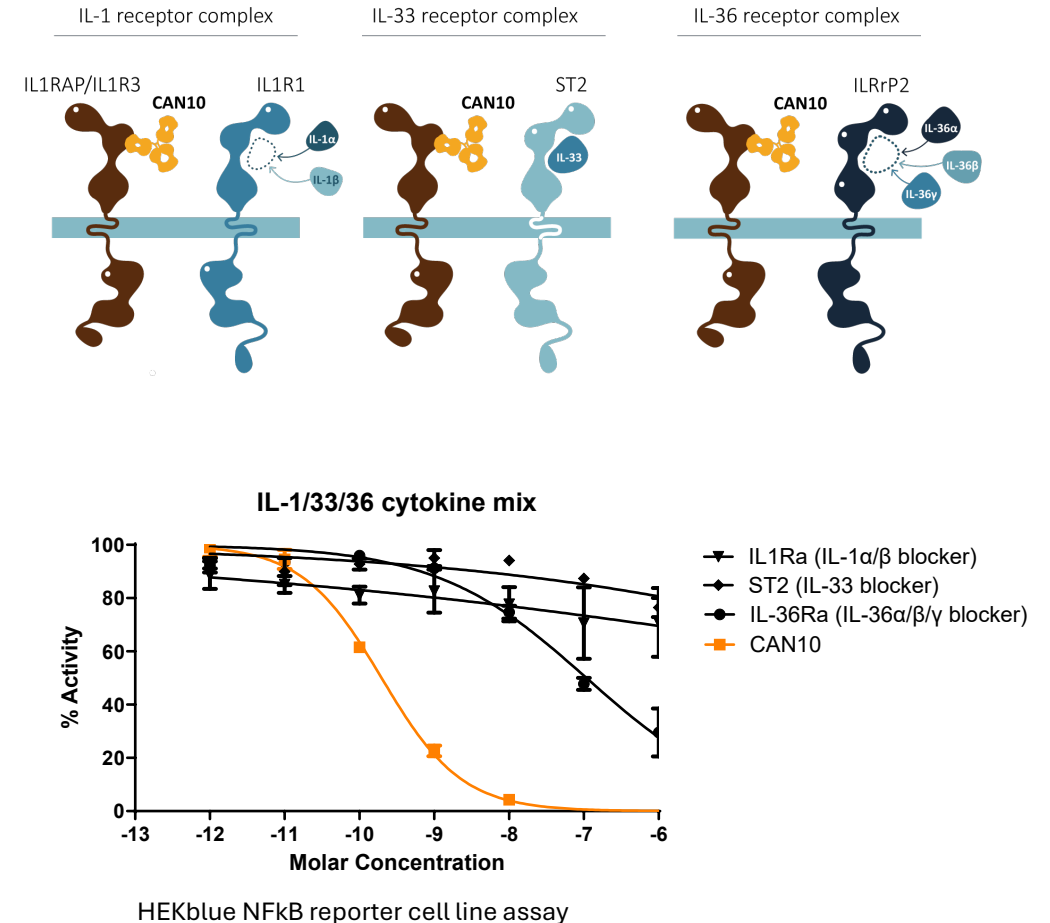


1. Interleukin-1 in the pathogenesis and treatment of inflammatory diseases - Charles A. Dinarello, Blood (2011) 117 (14): 3720–3732.

2. Canakinumab, spesolimab

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**

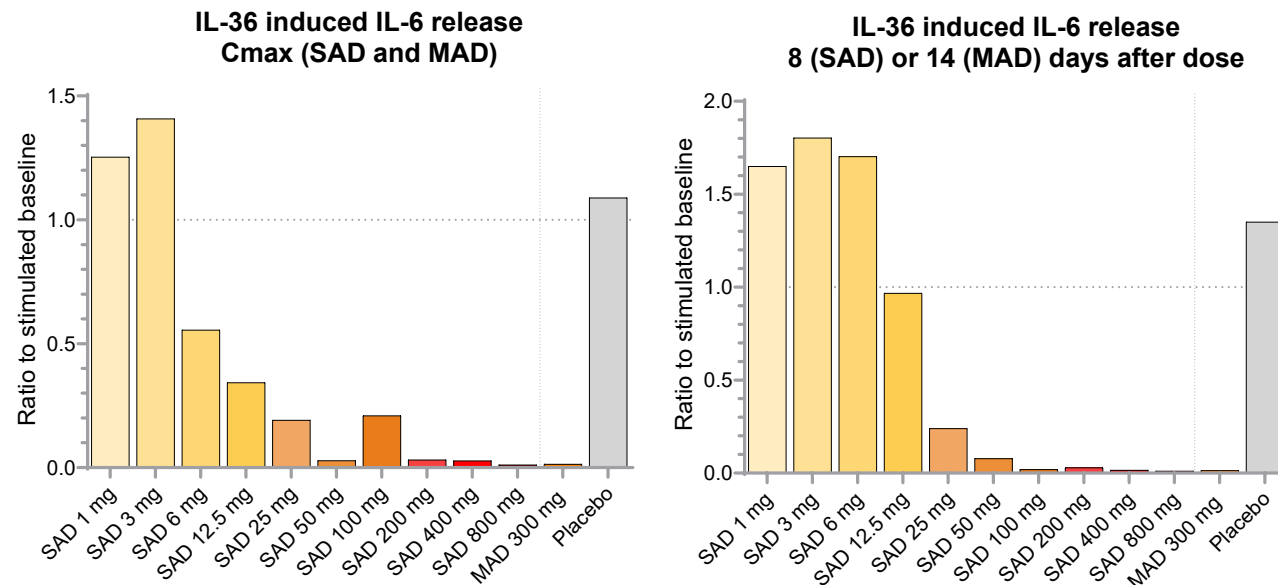


CAN10 IS UNDERGOING PHASE 1 (SAD/MAD) DEVELOPMENT - NO SAFETY ISSUES REPORTED

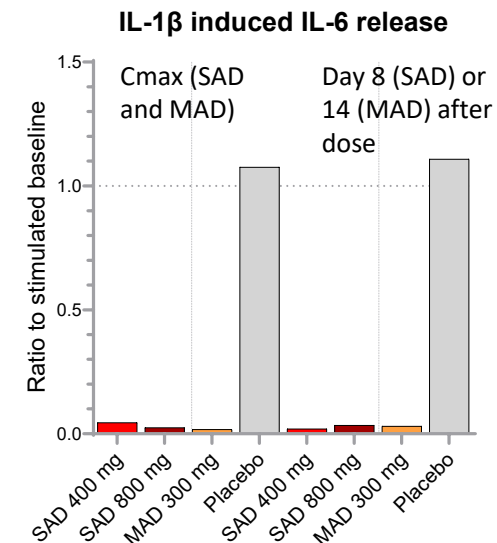
CAN10 FIH – Full blockade of both IL-36 and IL-1 β Signaling

- 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) → long lasting effect

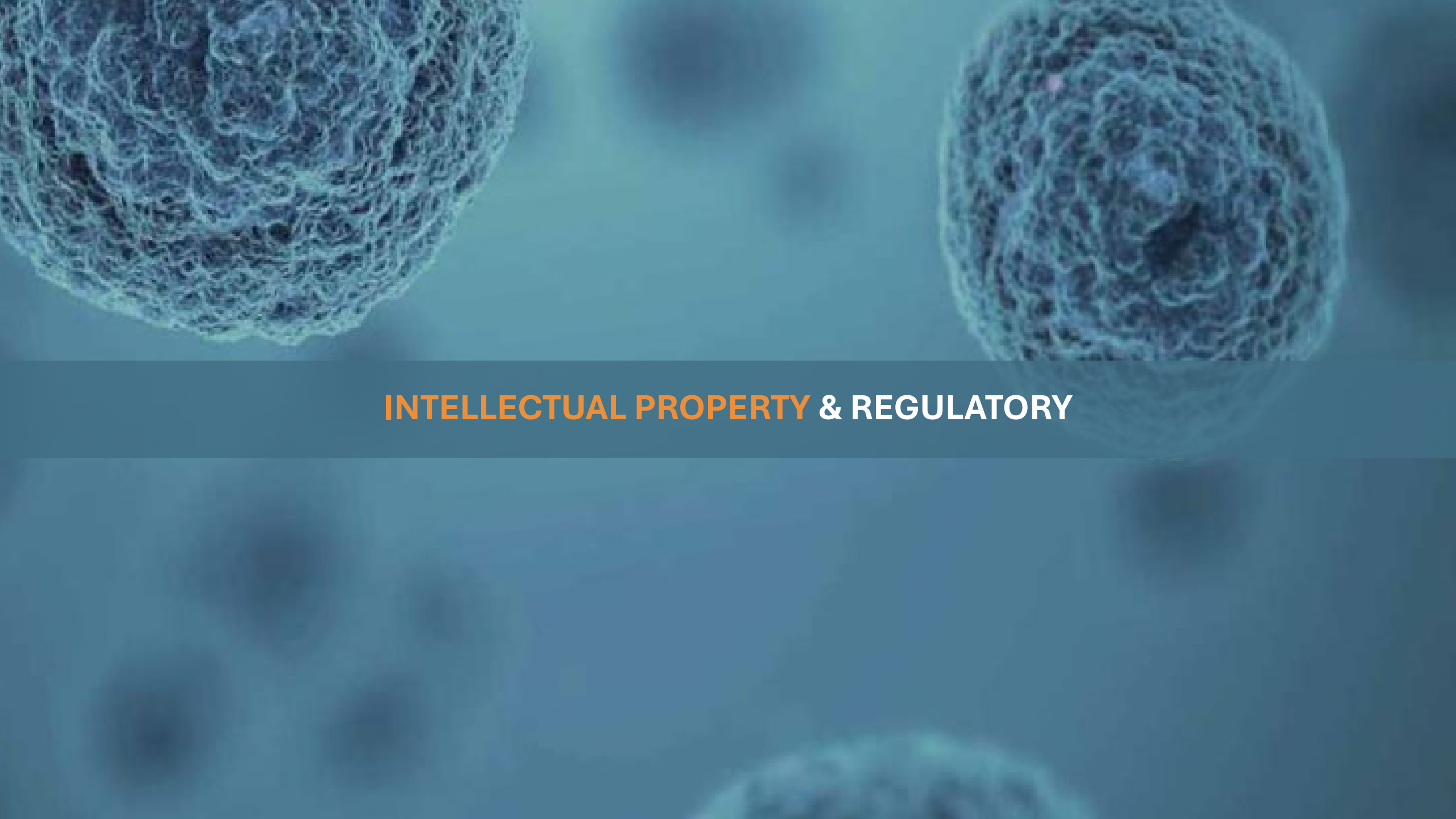
IL-36 inhibition



IL-1 β inhibition



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

A microscopic image showing several cells with a textured, mesh-like surface. The cells are primarily blue and purple, with some internal structures visible. A semi-transparent dark blue horizontal band is positioned across the middle of the image, containing the text 'INTELLECTUAL PROPERTY & REGULATORY'.

INTELLECTUAL PROPERTY & REGULATORY

Cantargia IP

Proprietary Pipeline

- Lead candidate anti-IL1RAP antibody **CAN04**
Expiry year **2035**
Granted patents (e.g. Europe, USA, China, Japan)
- Anti-IL1RAP for treatment of **solid tumors**
Expiry year **2032**
Granted patents (e.g. Europe, USA, China, Japan)
- Anti-IL1RAP for treatment of **hematological disorders**
Expiry year **2030**
Granted patents (e.g. Europe, USA, China, Japan)
- Anti-IL1RAP for treatment of **myeloproliferative disorders**
Expiry year **2029**
Granted patents (USA), acquired
- Additional anti-IL1RAP antibodies of **CANxx**
Expiry year **2037**
Library of anti-IL1RAP antibodies for CANxx project(s)
Granted patents (USA, China, Japan)



Strategic Partnership

- Anti-IL1RAP antibody **CAN10**
Expiry year **2041**
Granted patents (USA) and pending in worldwide territory

Estimated expiry are conservative, not incorporating potential extension periods of market exclusivity rights

US Designation & Protections in Addition to Patents

- **Fast Track Designation Benefits – Granted in 2025 for nadunolimab in high-level IL1RAP PDAC**
 - Provides sponsors with frequent communication and meetings with the FDA, which can help clarify requirements and resolve issues quickly during drug development.
 - Makes drugs eligible for Accelerated Approval and Priority Review, allowing faster access to the market if certain criteria are met.
 - Permits Rolling Review, so sponsors can submit completed sections of a New Drug Application (NDA) or Biologics License Application (BLA) for FDA review rather than waiting until the application is complete.
- **Orphan Drug Designation Benefits – Granted in 2021 for nadunolimab in pancreatic cancer**
 - Grants up to 25-50% tax credits for qualified clinical trial expenses related to orphan drug development.
 - Provides seven years of market exclusivity for the approved indication, independent of patent status—no other company can market the same drug for the same indication during this time.
- **US Exclusivity for Biologics**
 - Biologics approved by the FDA get 12 years of exclusivity from the date of first licensure under the Biologics Price Competition and Innovation Act (BPCIA).
 - For the first 4 years, the FDA does not accept any biosimilar applications relying on reference data; for the full 12 years, biosimilars cannot be approved for that product.
 - Pediatric exclusivity can extend this period by 6 months.

EU Designations & Protections in Addition to Patents

- **Orphan Drug designation in EU – Granted in 2021 for nadunolimab in pancreatic cancer**
 - **Grants market exclusivity period of 10 years** from the date of marketing authorization during which no similar medicinal product can be placed on the market for the same therapeutic indication.
 - **Potential to extend by 2 additional years (making a total of 12 years) if the sponsor completes an agreed pediatric investigation plan (PIP)** related to the orphan condition.
- **EU Exclusivity for Biologics**
 - The EU uses an “8+2+1” system for market and data exclusivity:
 - 8 years of data exclusivity, during which competitors cannot rely on the innovator’s data for regulatory approval.
 - 2 additional years of market exclusivity, so biosimilars can be approved but not marketed until after 10 years total.
 - 1 optional year is added for a new indication that demonstrates significant clinical benefit.

A microscopic view of cells, possibly pollen grains, with a blue overlay. The cells are spherical and have a textured, mesh-like surface. The background is a soft, out-of-focus blue.

KEY FINANCIALS & INVESTMENT HIGHLIGHTS

Key Financial and Share Information

Financials

- Cash position: SEK **210m** (June 30, 2026)*
- Runway: Mid-2028 (with current commitments)

The Cantargia Share

- Listing: **Nasdaq Stockholm** main market
- Ticker: **CANTA.ST**
- Number of shares: **248,611,655** (June 30, 2026)*
- Share price: SEK **2.146** (June 30, 2026)
- Market Cap: SEK **533m** (June 30, 2026)

Non-commission Analyst Coverage:

- DnB Carnegie – Arvid Necander
- Van Lanschot Kempen – Sebastiaan van der Schoot
- H.C. Wainwright – Sara Nik, Ph.D. & Joe Pantginis, Ph.D.

Cantargia's Cap Table, June 30, 2026

Owner	Number of shares	Capital/Votes (%)
Fourth Swedish National Pension Fund	23,551,565	9.47%
Avanza Pension	13,649,243	5.49%
American Century Investment Management	7,481,767	3.01%
Handelsbanken Fonder	7,203,219	2.90%
Henrick Schill	4,246,933	1.71%
The Invus Group	3,916,705	1.58%
Brushamn Invest AB	3,391,740	1.36%
Plato Investment Management	3,160,712	1.27%
Nordnet Pensionsförsäkring	3,026,485	1.22%
Acadian Asset Management	2,446,082	0.98%
Other	176,537,204	71.01%
Total	248,611,655	100.0%

*) Excluding the proceeds from the 33,333,333 new shares issued in the 2026 rights issue, registered July 1.

A Global Leader in IL1RAP Antibody Development

- 
- **IL1RAP is a unique target** with dual mechanism addressing inflammation and cancer biology
 - **High IL1RAP expression marks poor prognosis** in multiple malignancies
 - **Nadunolimab is a versatile combination agent** for chemotherapy, immunotherapy, and emerging treatment modalities
 - **Robust oncology pipeline in both MDS and PDAC** demonstrates therapeutic efficacy across multiple tumor types and disease settings



Thank you

Contact details:

info@cantargia.com