Cantargia

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

Corporate Presentation

May 2026

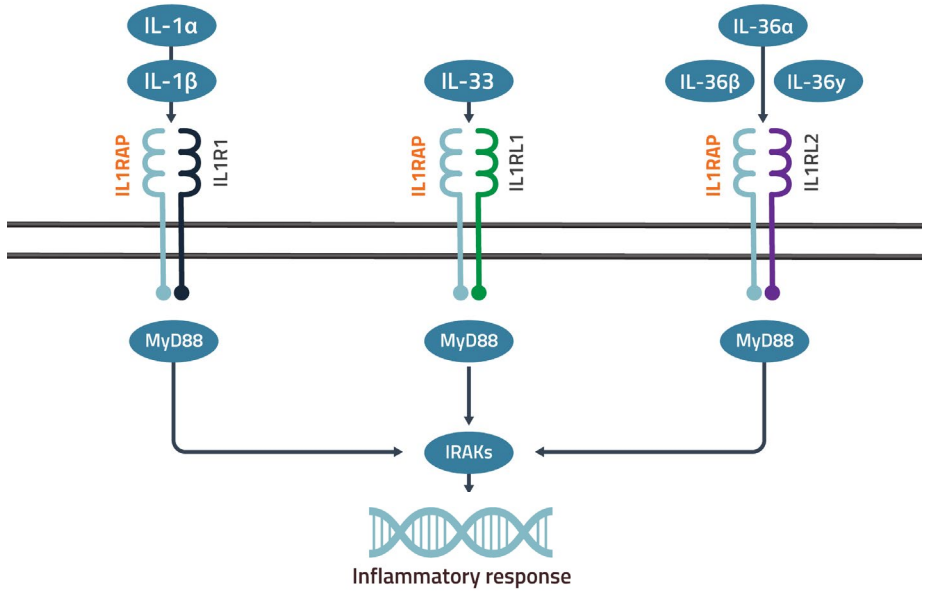
NASDAQ STOCKHOLM MAIN LIST (CANTA.ST)

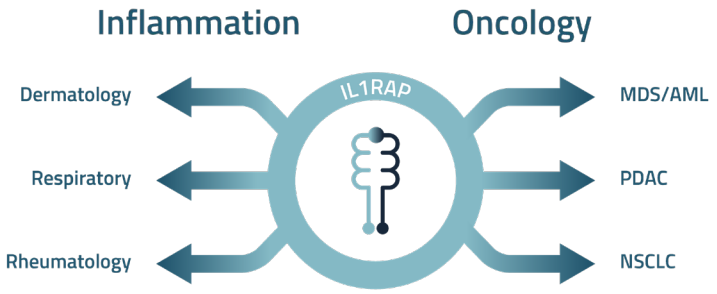
Safe Harbor Statement

Statements in the Investor Presentation, including those regarding the possible or assumed future or other performance of the Company or its industry or other trend projections, constitute forward-looking statements. By their nature, forward-looking statements involve known and unknown risks, uncertainties, assumptions and other factors as they relate to events and depend on circumstances that will or may occur in the future, whether or not outside the control of the Company. No assurance is given that such forward-looking statements will prove to be correct. Prospective investors should not place undue reliance on forward-looking statements. They speak only as at the date of this Investor Presentation and the Company undertakes no obligation to update these forward-looking statements. Past performance does not guarantee or predict future performance. Moreover, the Company undertakes no obligation to review, update or confirm expectations or estimates or to release any revisions to any forward-looking statements to reflect events that occur or circumstances that arise in relation to the content of the Investor Presentation.

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	FDA Fast Track, Orphan Drug; Orphan Drug					
		MDS/AML[1]	Investigator-Initiated Trial					MD Anderson Cancer Center
		MSS CRC[2]	Investigator-Initiated Trial					Mount Sinai
CANxx	Unique IL1RAP platform[3]							
Autoimmune								
CAN14	IL1RAP BsAb[4]	Autoimmune diseases						
CAN10	IL1RAP	Autoimmune diseases						Otsuka

PDAC – pancreatic ductal adenocarcinoma; **MDS** – Myelodysplastic Syndrome; **AML** – Acute Myeloid Leukemia; **MSS CRC** – Metastatic microsatellite stable colorectal cancer; **BsAB** – Bispecific Antibody

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

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Executive Management Team with Extensive Experience

Hilde Steineger
CEO

Patrik Renblad
CFO

David Liberg
CSO

Ton Berkien
CBO

EXECUTIVE TEAM WITH COMPREHENSIVE INDUSTRY INSIGHT

LEO · AstraZeneca · novo nordisk · Nordea · Genmab · mAbs Therapeutics, Inc. · northsea THERAPEUTICS · AMGEN · Takeda · FERRING PHARMACEUTICALS · SYNACT PHARMA

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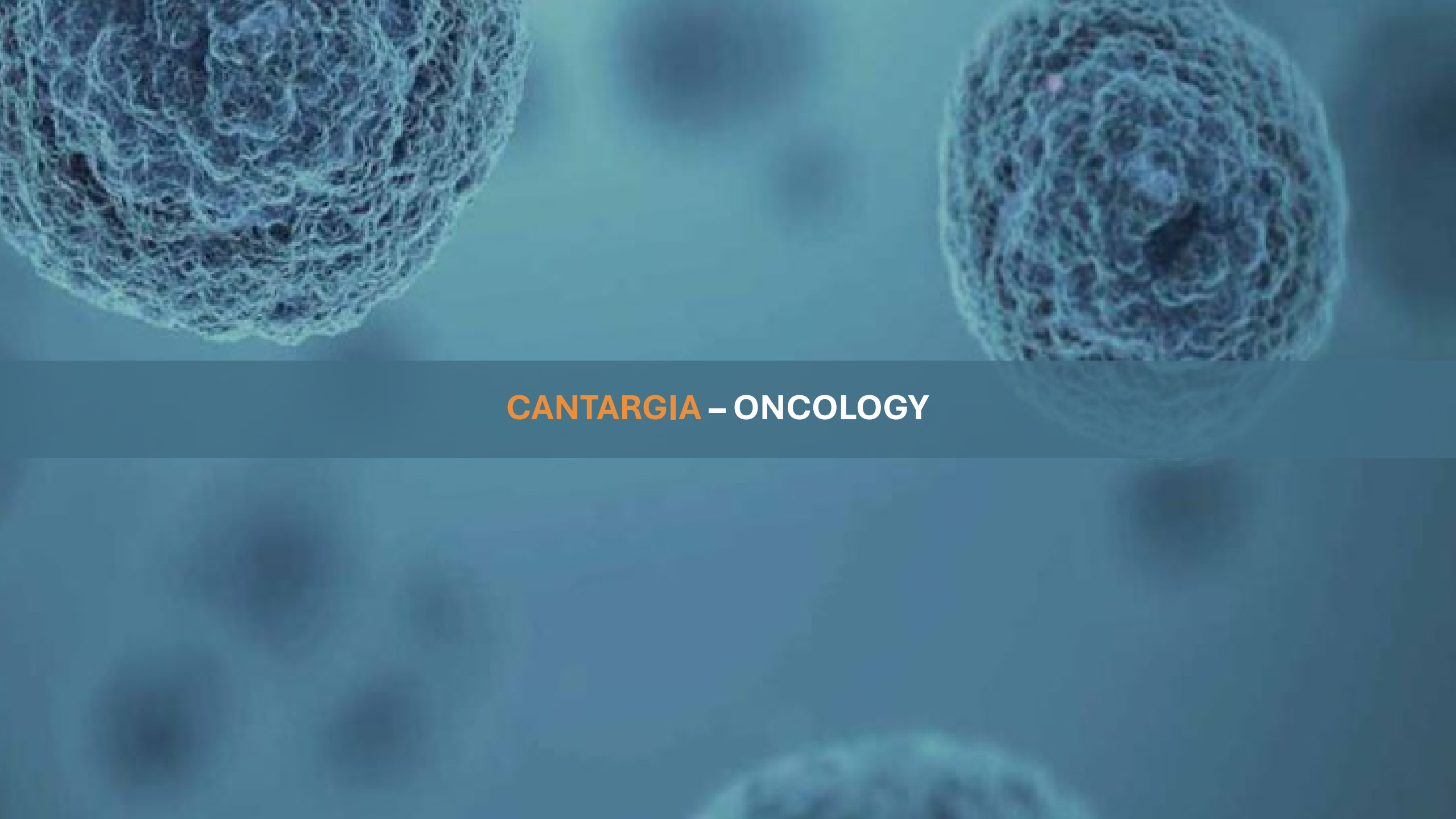
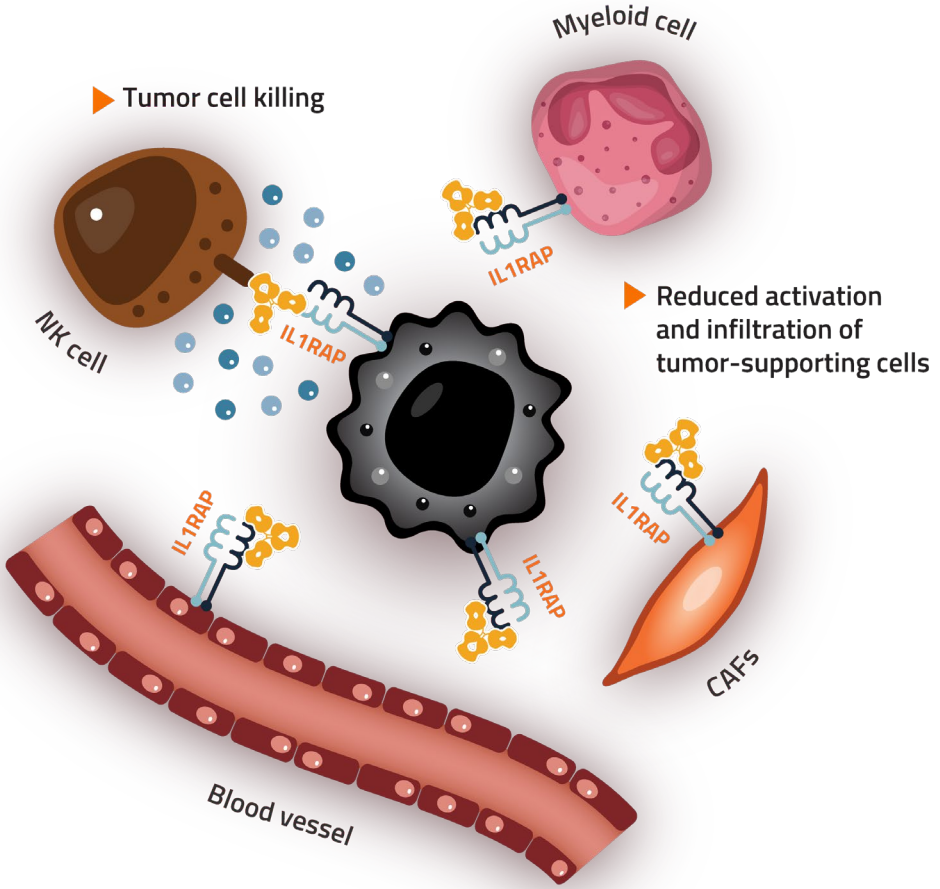


CANTARGIA – ONCOLOGY

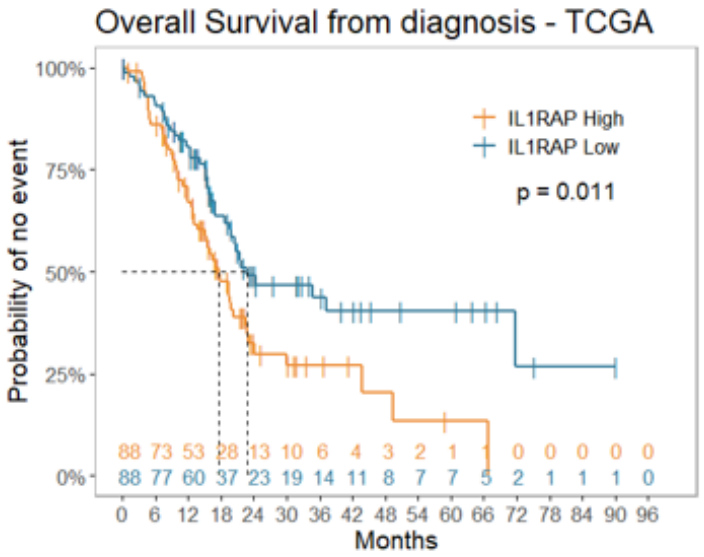
Why We're Pursuing IL1RAP with Nadunolimab in Oncology

- **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

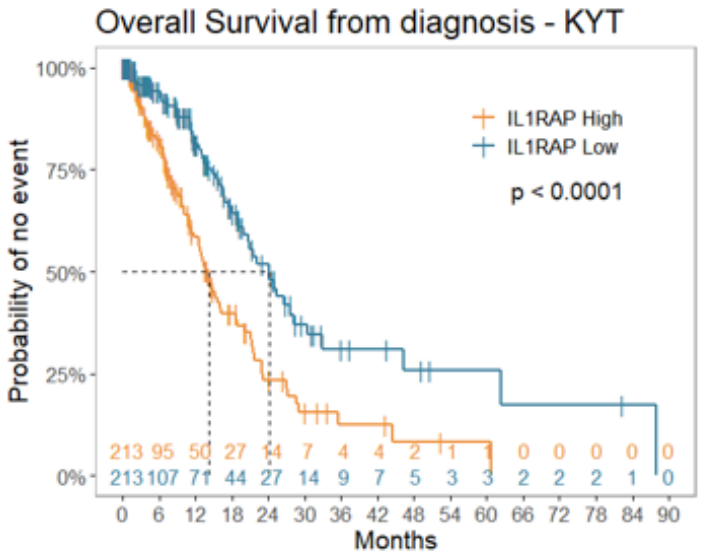


PANCREATIC DUCTAL ADENOCARCINOMA – PDAC

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



TCGA RNA data
PDAC patients all stages (mainly stage I/II)



Know Your Tumor RNA data
PDAC patients stage III/IV

- High IL1RAP RNA expression in PDAC tumors of all stages correlates with poor survival
- Analysis of RNA-data by AI shows two subnetworks enriched for IL1RAP-high genes correlate with worse survival and basal-like subtype

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

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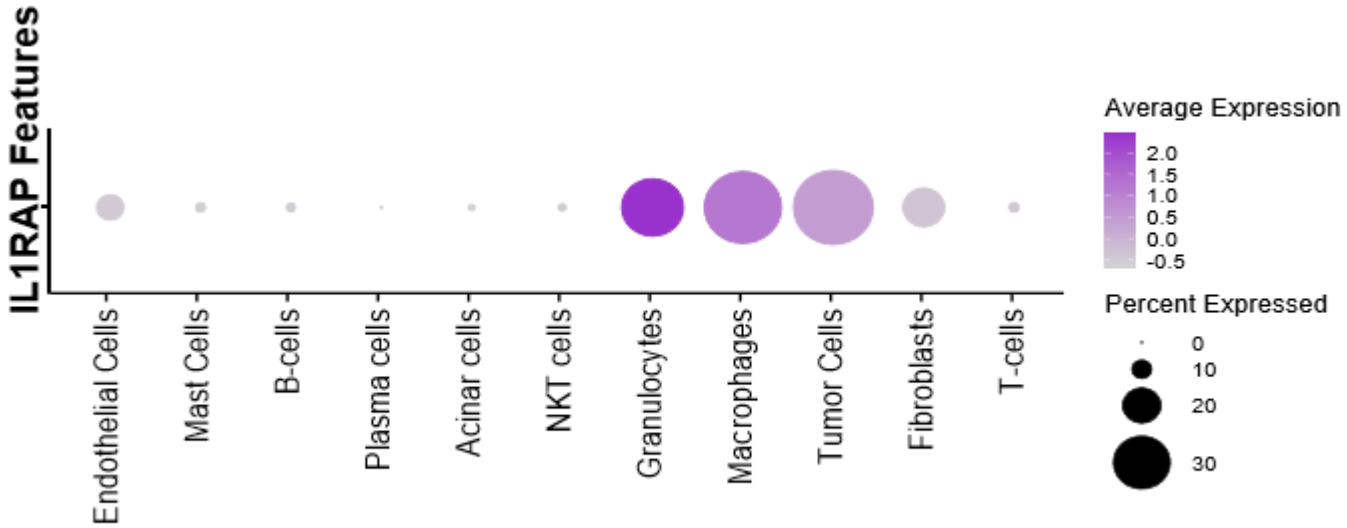
IL1RAP Is Expressed by Tumor Cells, Myeloid Cells and Fibroblasts in the PDAC TME



- 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

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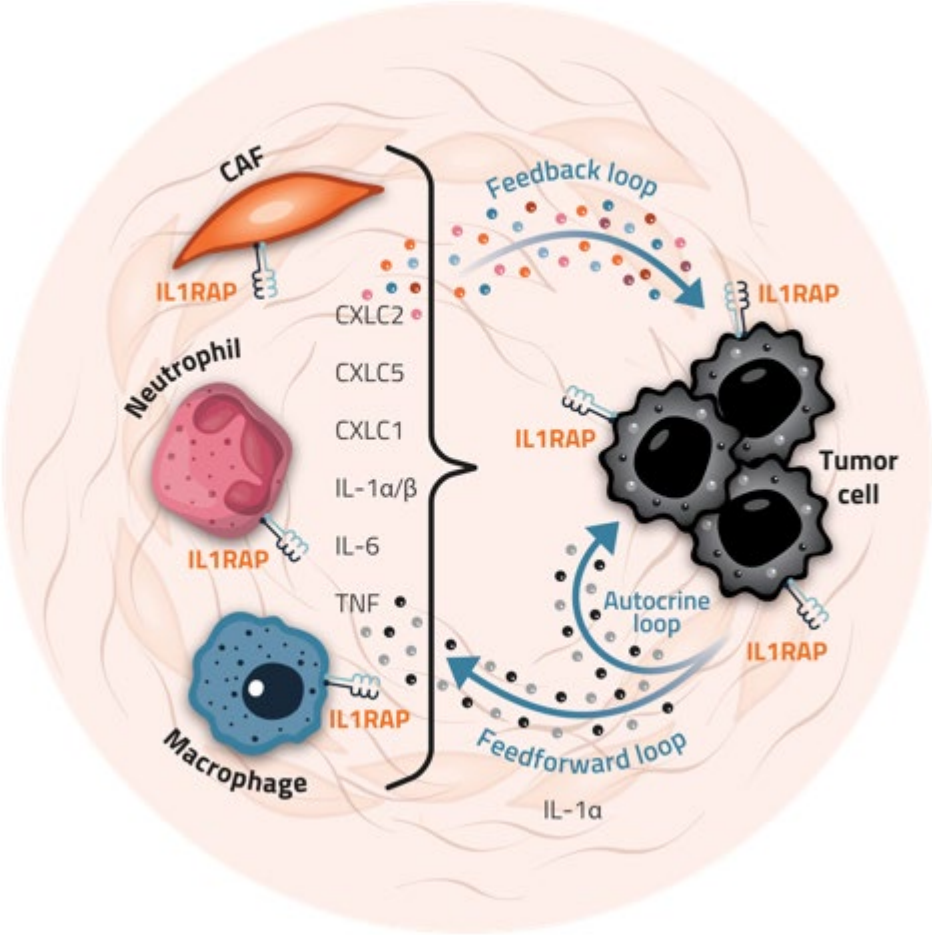
IL1RAP Is a Fundamental Driver in the PDAC TME

- 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

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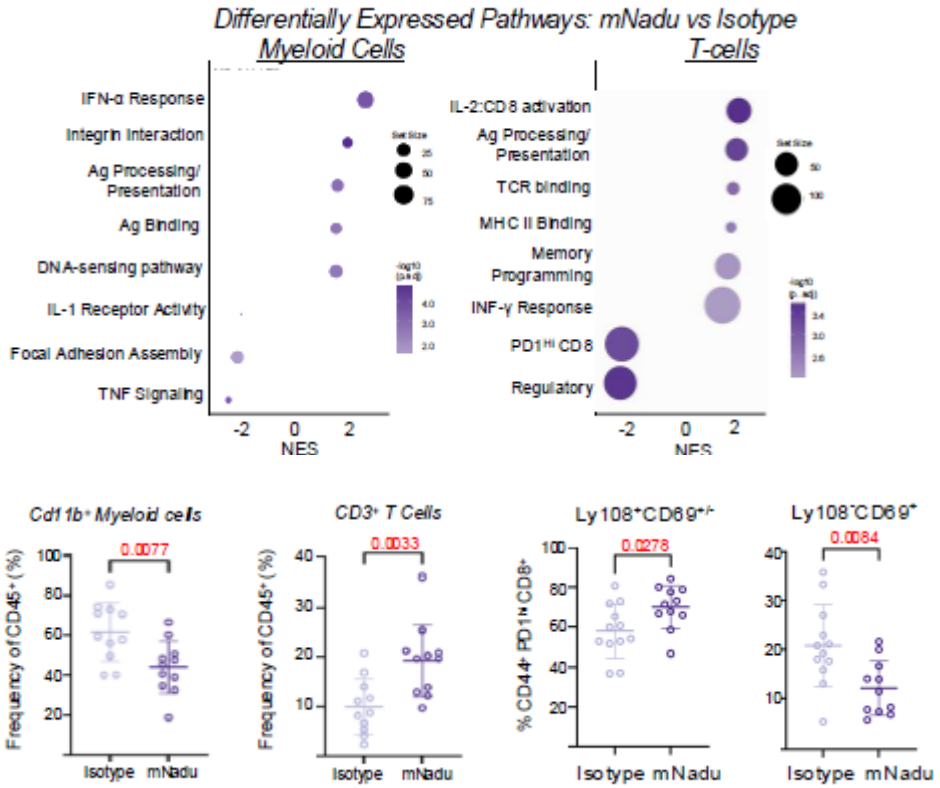
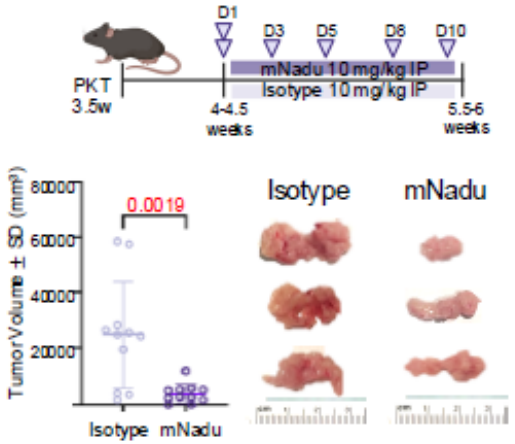
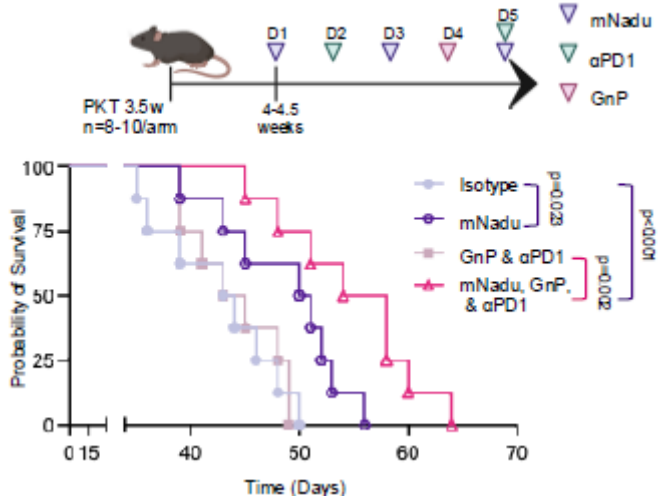
IL1RAP Targeting has Distinct Effects in an Aggressive and Treatment Resistant Preclinical Model of PDAC

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/+;Tgfbr2fl/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

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PDAC: A Deadly Cancer with Limited Treatment Options

Diagnosed Incident Cases*	230 000
Diagnosed Incident cases with advanced or metastatic disease**	140 000
Metastatic treatment rates***	85 000

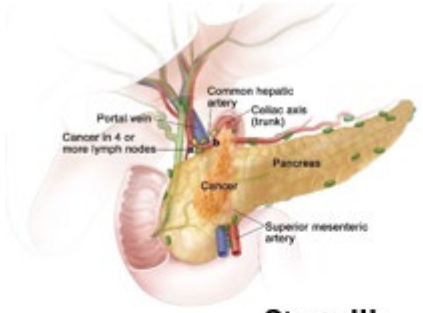
LOCALLY ADVANCED OR METASTATIC DISEASE

Median survival:

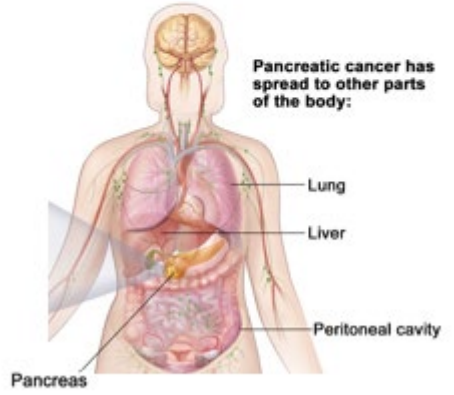
- **8.5 – 11.7 mo**

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

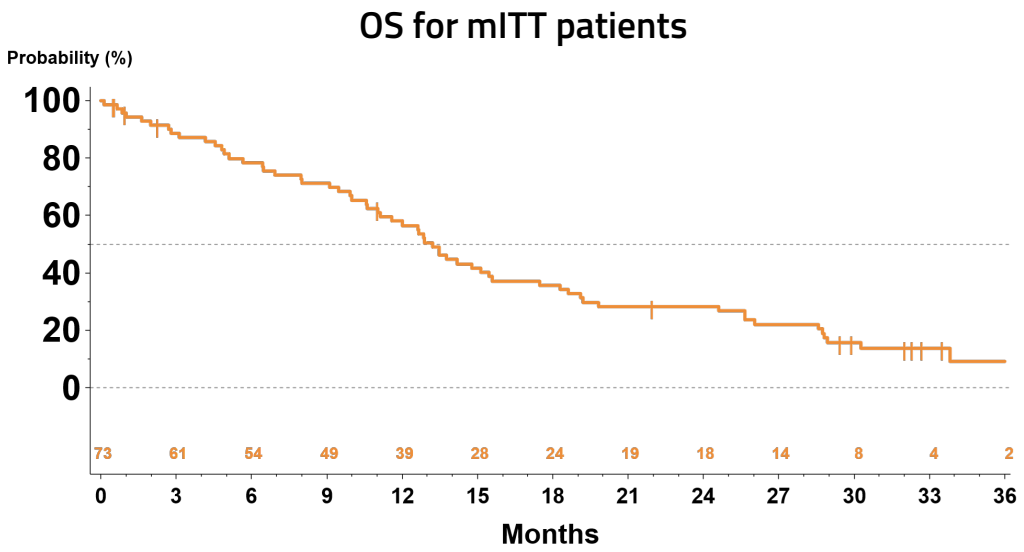
Images from National Cancer Institute

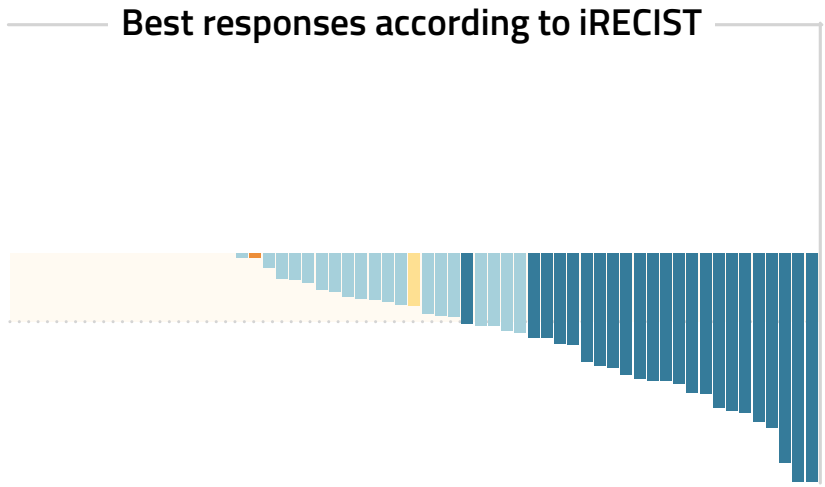
CURRENT DEVELOPMENT FOCUSES ON FIRST-LINE METASTATIC DISEASE

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

PDAC: Pancreatic Ductal Adenocarcinoma

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)

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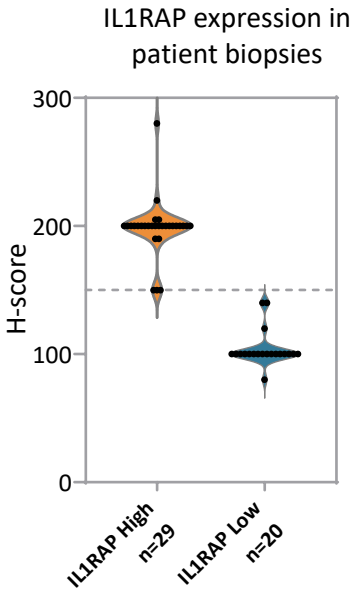
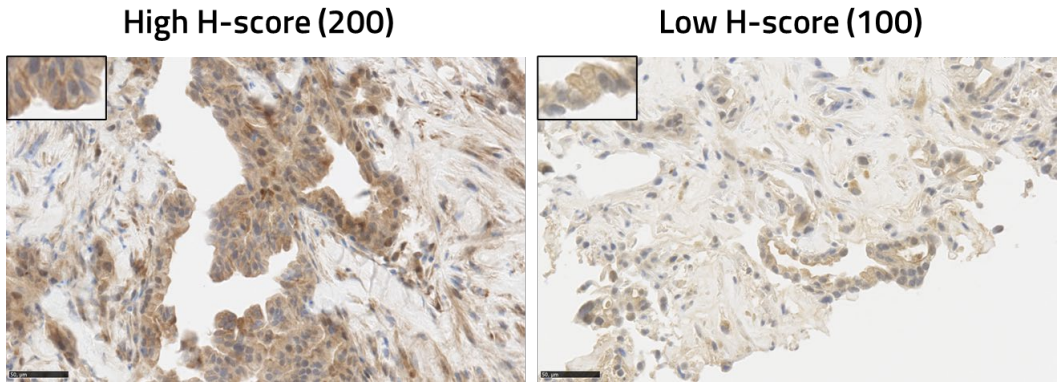
IL1RAP Expression in Tumor Biopsies from PDAC Patients





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

Van Cutsem et al, Clinical Cancer Research 2024

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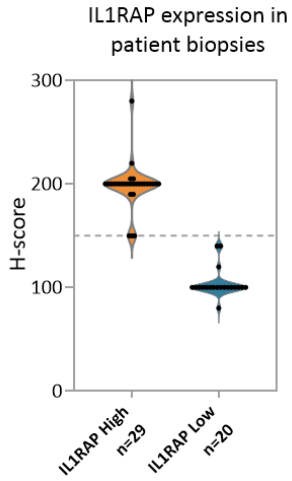
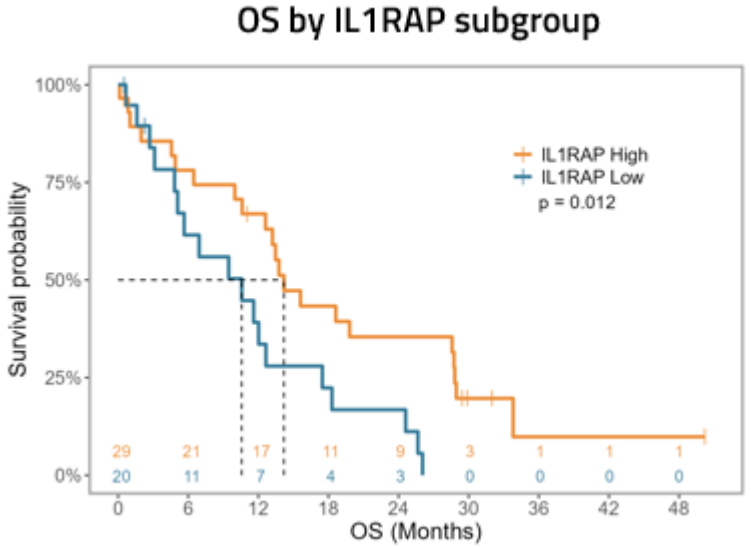
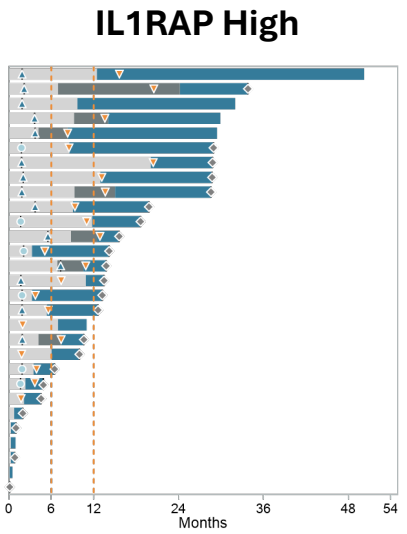
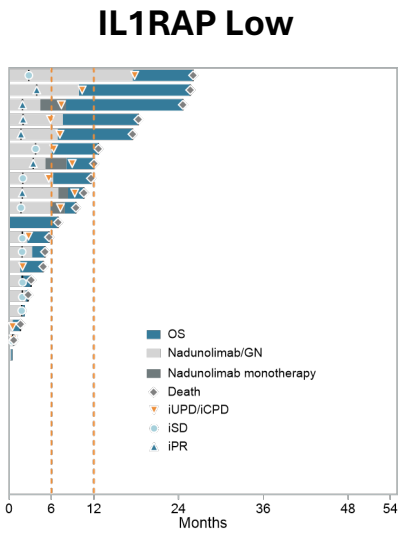
Stronger OS Benefits in High IL1RAP-Expressing Patients (14.2 vs. 10.6 Months)









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

GnP mOS=8.5 mo MPACT (N Engl J Med 2013) and 9.2 mo NAPOLI-3 (Lancet, 2023)

Van Cutsem et al, Clinical Cancer Research 2024

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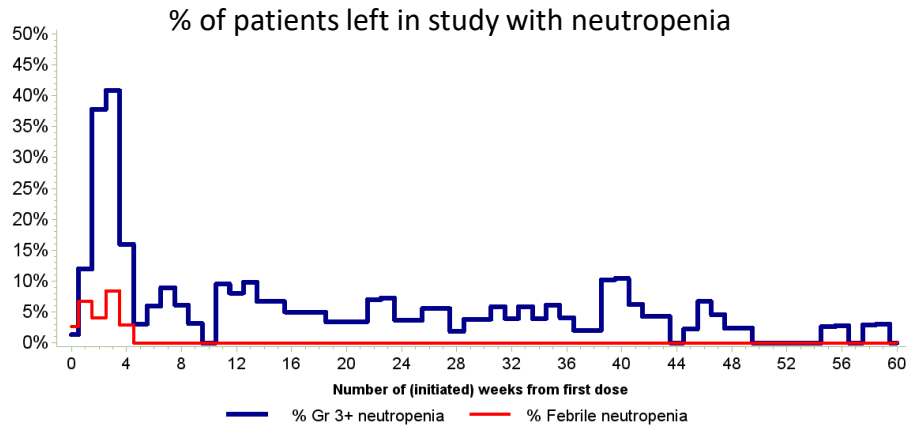
Safety Profile CANFOUR PDAC

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%



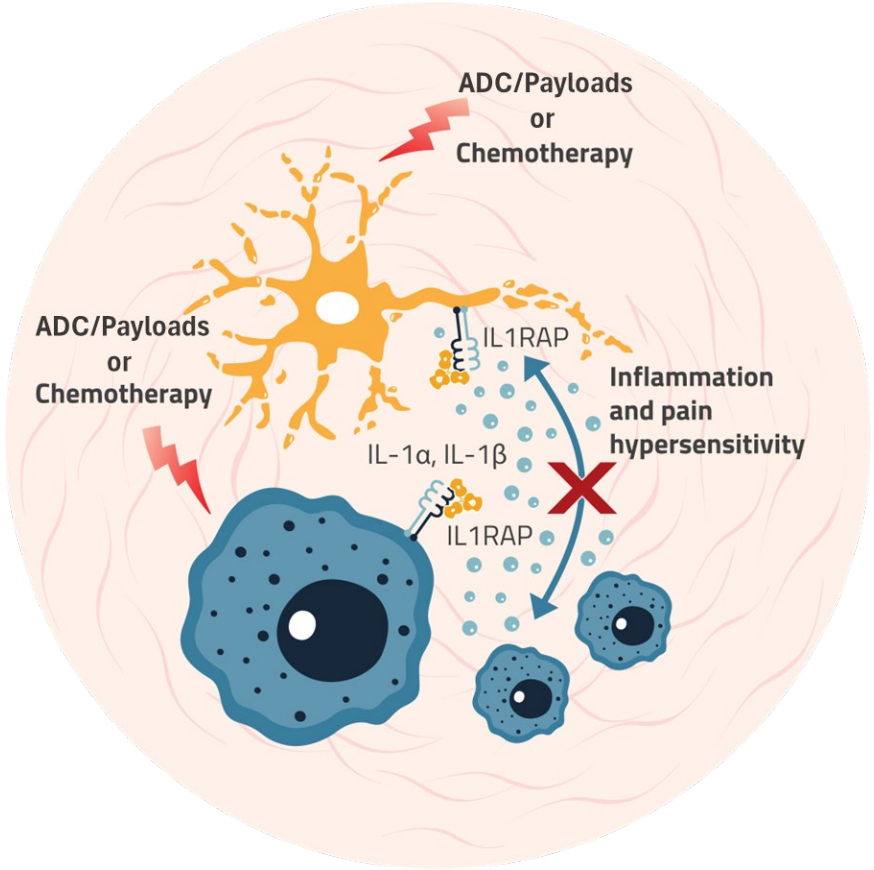
weeks

- Elevated levels of neutropenia in patients treated with nadunolimab + chemotherapy
- Only seen in combination with chemotherapy, not as monotreatment
- 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 IL1β have been shown to correlate with higher risk of chemotherapy induced neuropathy[1]
- 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 models[4]



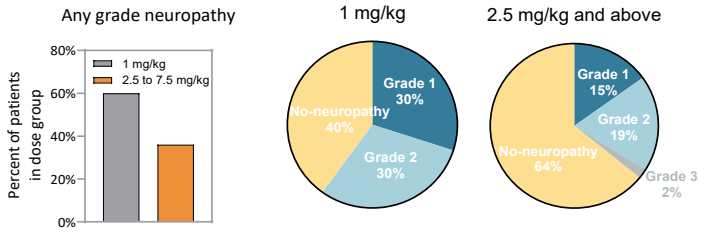
IN ADDITION TO PROMISING EFFICACY NADUNOLIMAB COULD CONTRIBUTE TO SAFER COMBINATION THERAPIES

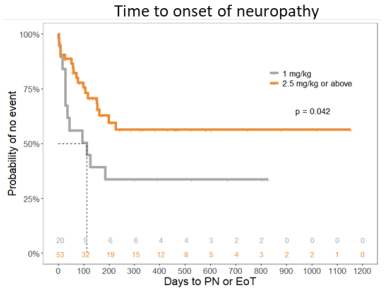
1. Kleckner et al Breast Cancer Research and Treatment, Volume 189, pages 521–532, (2021) 2. SITC Annual meeting 2024, 3. ASCO Annual meeting 2024, 4. AACR Annual meeting 2025

Neuropathy Clinical Data with nadunolimab

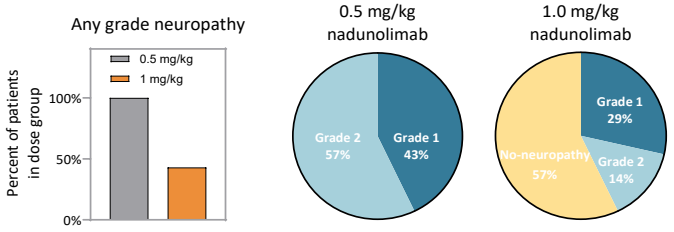
- Ph2 study data from CANFOUR in 1L PDAC patients treated with nadunolimab and gemcitabine/nab-paclitaxel
  - Lower Grade 3-4 peripheral neuropathy than expected from historical controls (1% vs 17%)
  - Reduced incidence as well as delayed onset of all grade neuropathies
- Correlation between nadunolimab 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

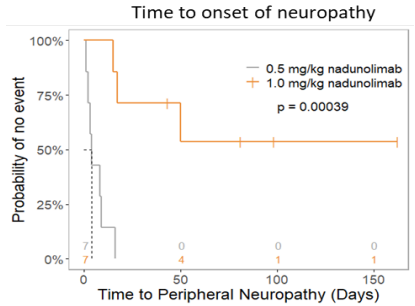
Gemcitabine/nab-paclitaxel



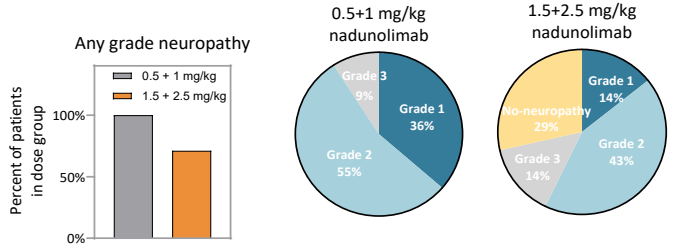


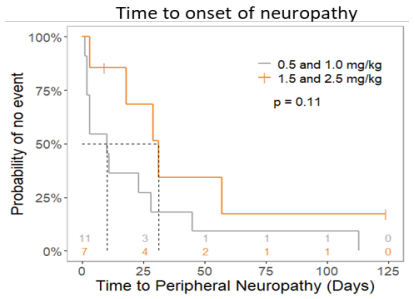
mFOLFOX



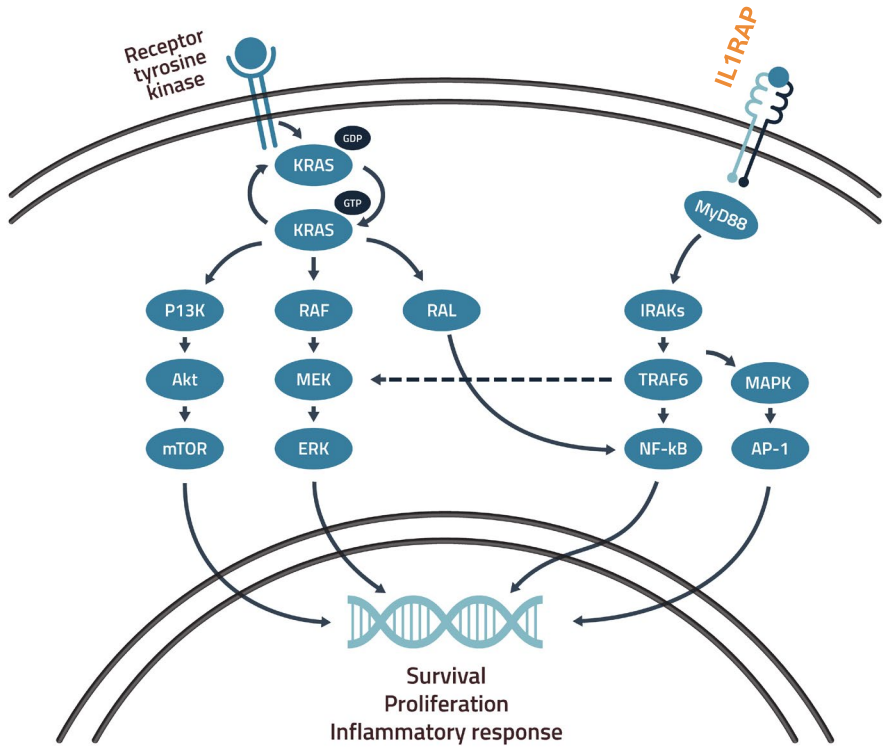


mFOLFIRINOX





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

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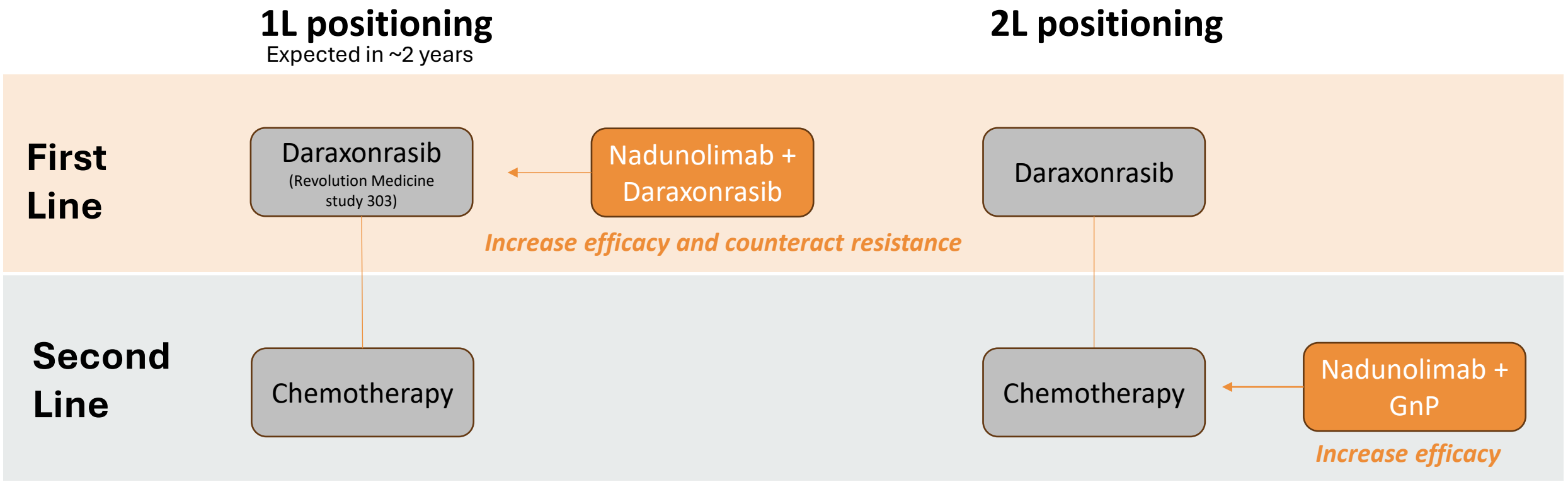
Nadunolimab's Potential Roles in the Evolving Landscape of Metastatic PDAC Treatment



Current Standard-of-Care therapies includes the following chemotherapies: Gemcitabine/Nab-paclitaxel (GnP), Folfirinox, Nalirifox

NADUNOLIMAB HAS THE POTENTIAL TO DELIVER EFFICACY AND OVERCOME TREATMENT RESISTANCE IN BOTH 1ST & 2ND LINE

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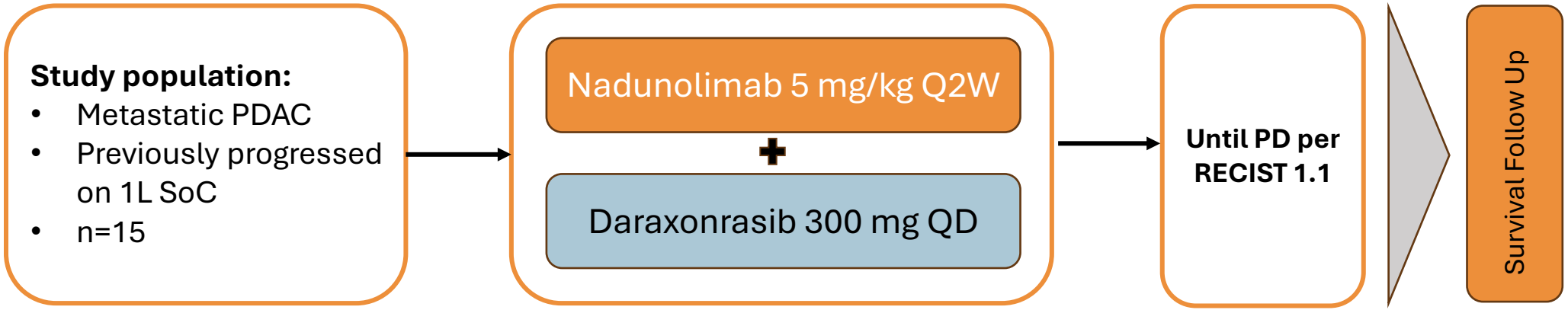
Preparation for Multiple Treatment Options: Ph1b/2a in Combination with Nadunolimab + RASi

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

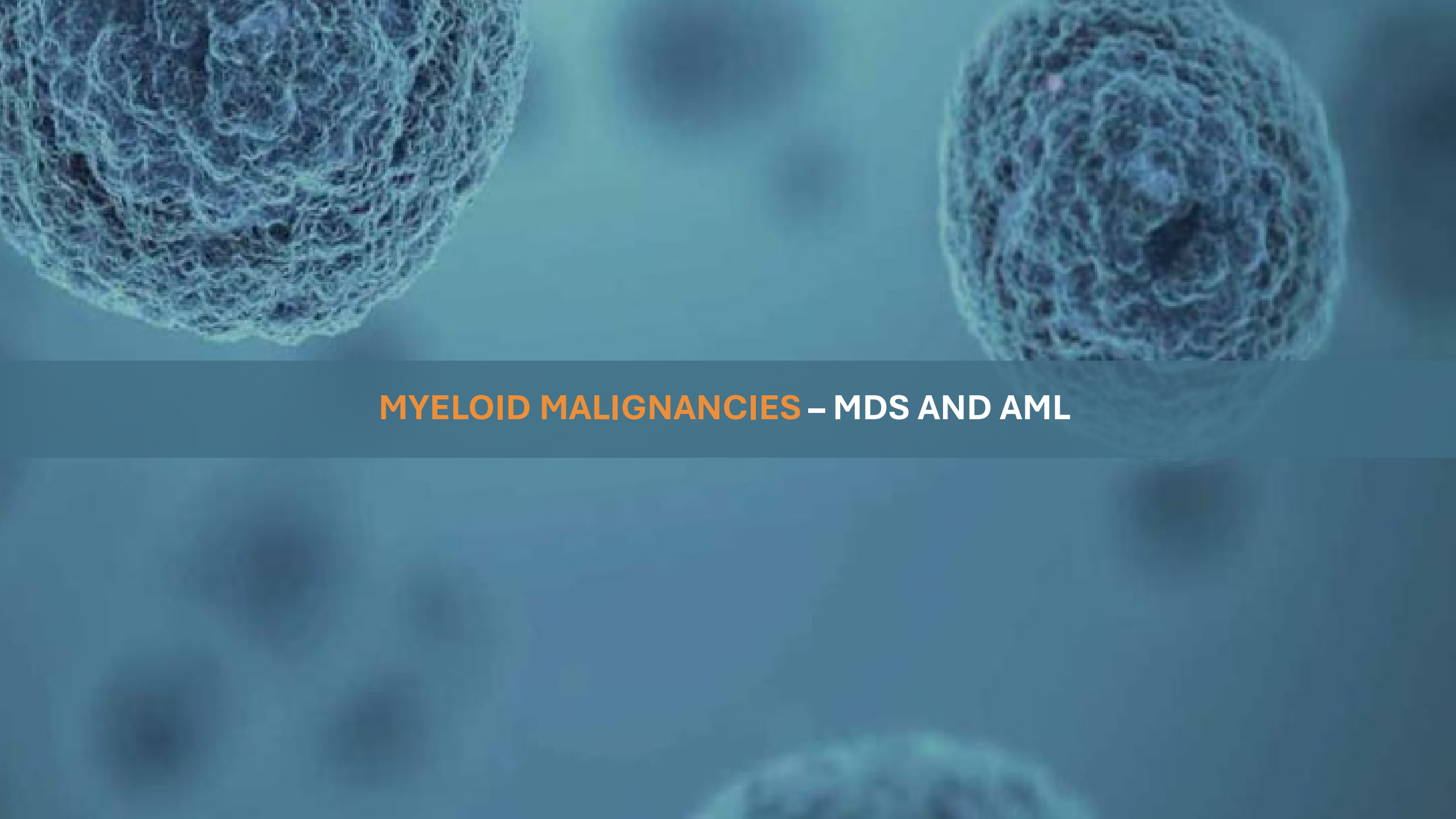


Primary objective:
- To determine the safety and tolerability of nadunolimab combined with daraxonrasib

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 Q4 2026/Q1 2027, subject to funding, regulatory approval and daraxonrasib availability



MYELOID MALIGNANCIES – MDS AND AML

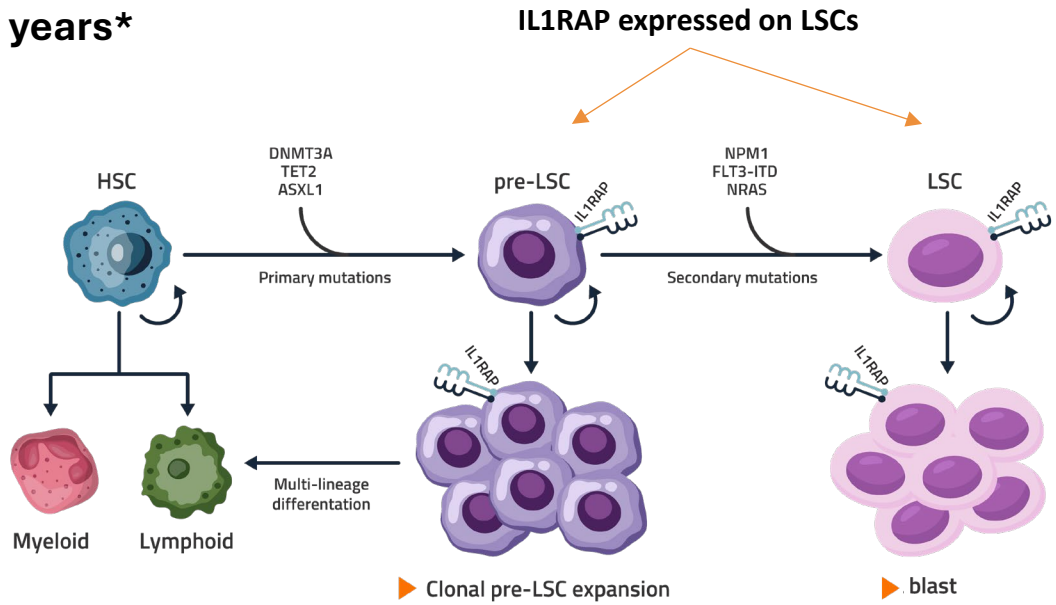
Myelodysplastic Syndrome (MDS) and Acute Myeloid Leukemia (AML) – IL1RAP at the Center of Disease

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

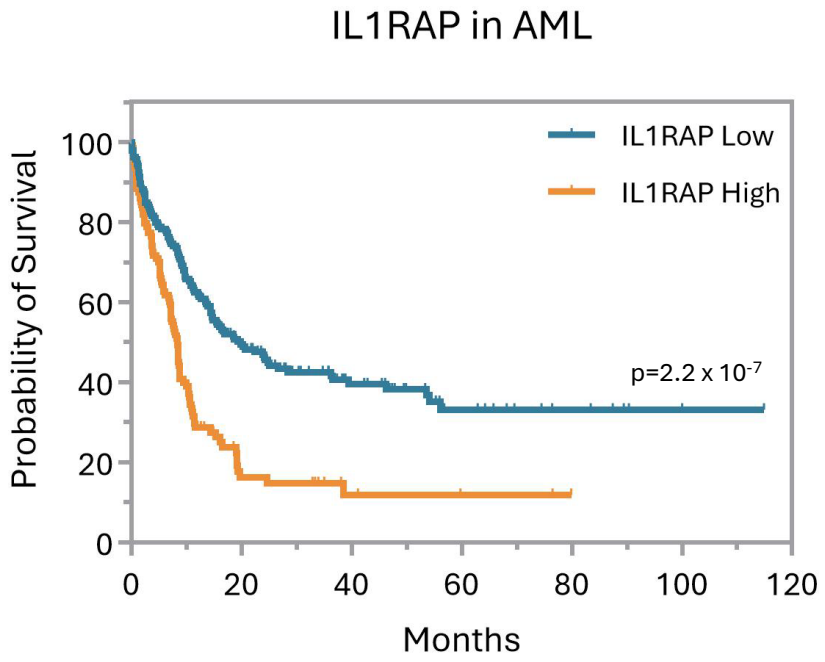
AML:

- 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)



*Deutsche Bank Research - 23 Apr 2026

Encouraging Leukemia Data Reinforce IL1RAP as a High-Risk Target



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

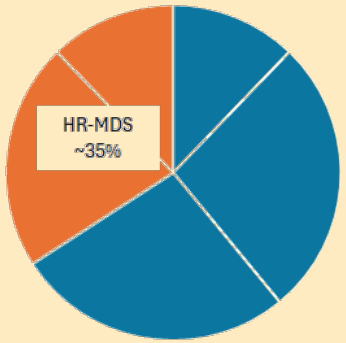
HIGH IL1RAP EXPRESSION IN LEUKEMIC MALIGNACIES IS ASSOCIATED WITH POOR SURVIVAL

Cantargia

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***

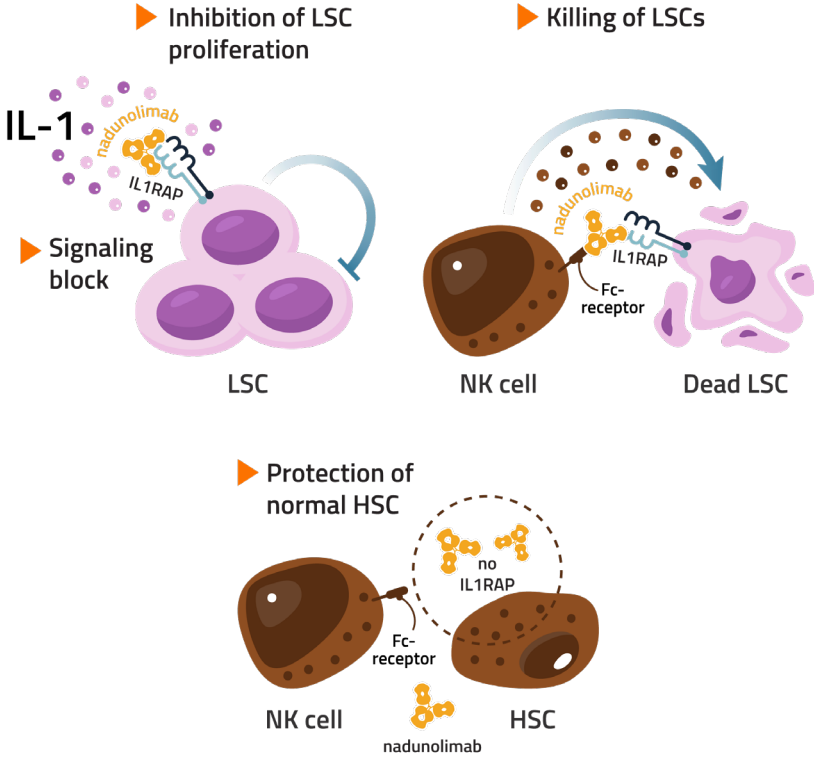
Value

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)
- **IL1α/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



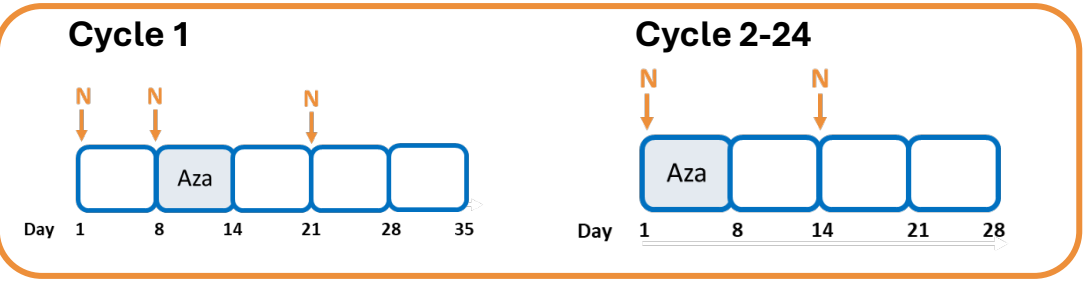
References: Askmyr et al. Blood (2013), Houtsma et al. Blood Adv (2022), Barreyro et al. Blood (2012); Ågerstam et al. PNAS (2015), de Boer et al. Haematologica 2021

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

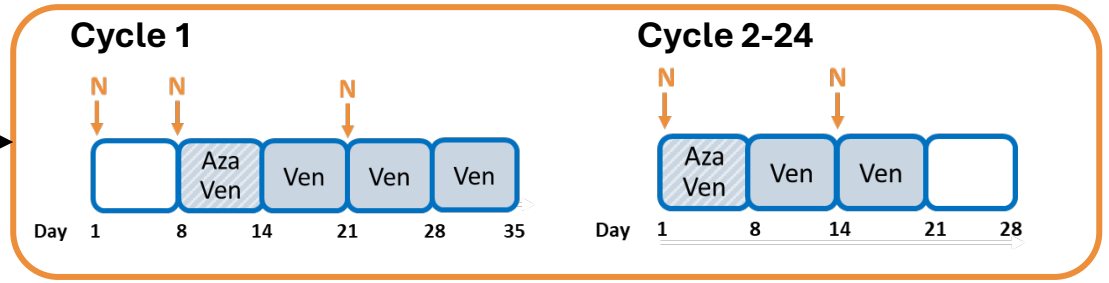
Principal Investigator: Gautam Borthakur, MD, MD Anderson Cancer Center
Grant support: U.S. Department of Defense (DOD)
Study start 2025

Arm 1 HR-MDS (n=20):
- Intermediate/high/very high risk MDS*
- Untreated or up to 2 prior treatments

*IPSS-R scoring



Arm 2 AML (n=20):
- Relapsed/refractory AML
- First or second salvage



Treatment until no protocol defined response

Study status per May 1, 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**

NADUNOLIMAB IIT STUDIES

Ongoing Investigator-Initiated Trials with nadunolimab

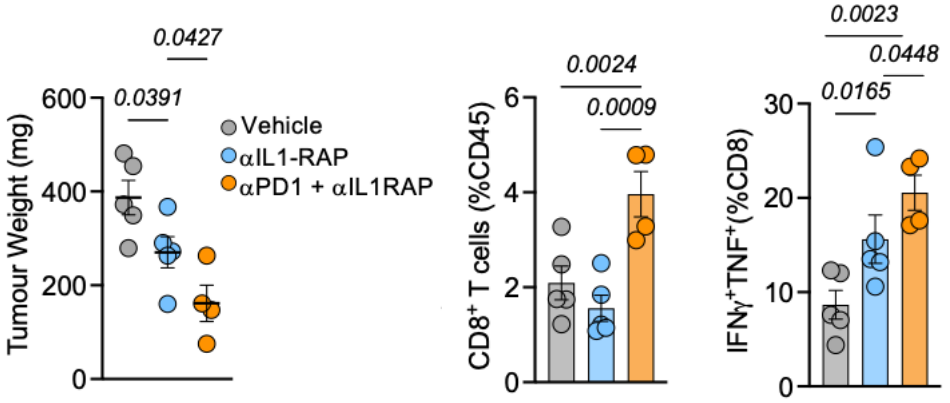
Study	Disease	Combination therapy	Number of patients	Status	NCT-number
Leukemia*	AML/MDS	Azacitidine and/or venetoclax	40	Recruiting	NCT06548230
Colorectal**	MSS CRC	Toripalimab (anti-PD-1)	24	Recruiting	NCT07281716

AML - Acute Myeloid Leukemia; **MDS** - Myelodysplastic Syndrome; **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; IFNγ+ TNF+ (%CD8)=activated cytotoxic T cells

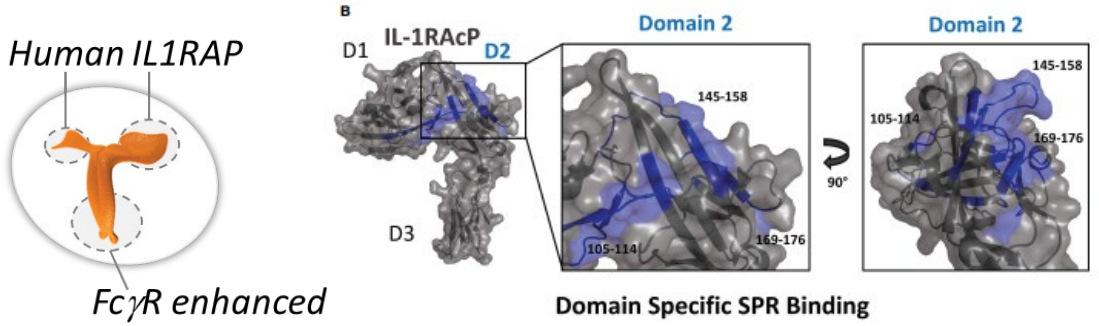
NADUNOLIMAB MAKES THE TUMOR SUSCEPTIBLE TO ICI TREATMENT BY COUNTERACTING THE IMMUNE SUPPRESSIVE TME

cantargia

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-1α and IL-1β 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.



Fields et al Front. Immunol. 2021

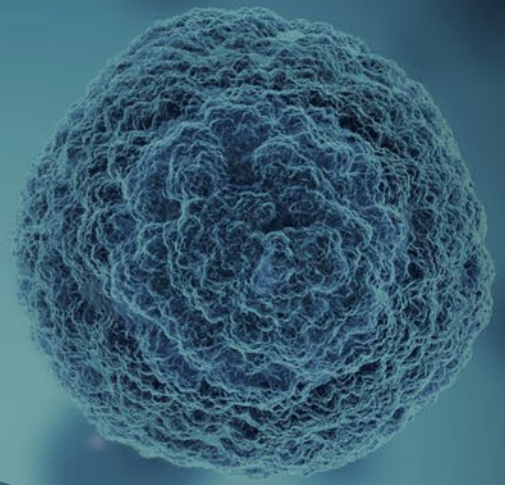
Nadunolimab attributes	Details
Human IL1RAP, K_D SPR	5.62 pM
Function blocking IL-1α/IL-1β, IC50	2.7 nM/0.2 nM
Function blocking IL-33, EC50	7.5 nM
Function blocking IL-36α/β/γ, IC50	0.2 nM/1.0 nM/0.3 nM
ADCC, IC50	<10 nM
Binding to Cyno, K_D SPR	5.49 pM
Binding to rat and mouse	No cross reactivity

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

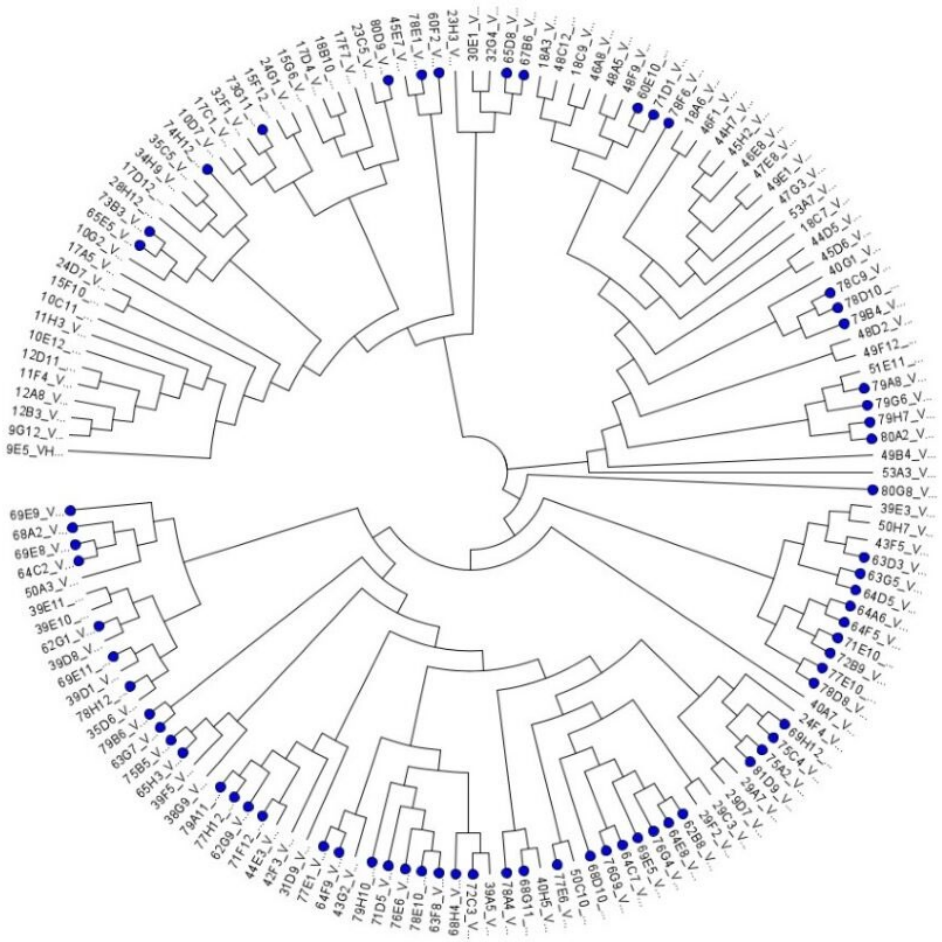
Next generation IL1RAP therapeutics



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 ~200 anti-IL1RAP antibodies** within the CANxx platform
- CANxx antibodies bind to different domains of IL1RAP and have **different functional properties**

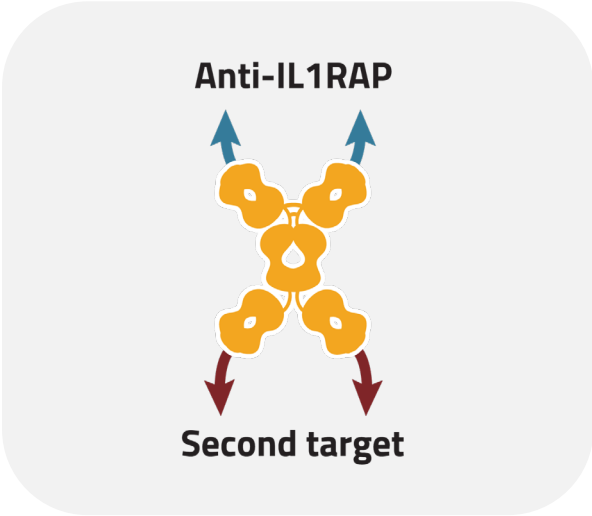


CANXX – A COMPREHENSIVE SOURCE FOR NEW DRUG PROJECTS

Next Generation IL1RAP Therapeutics

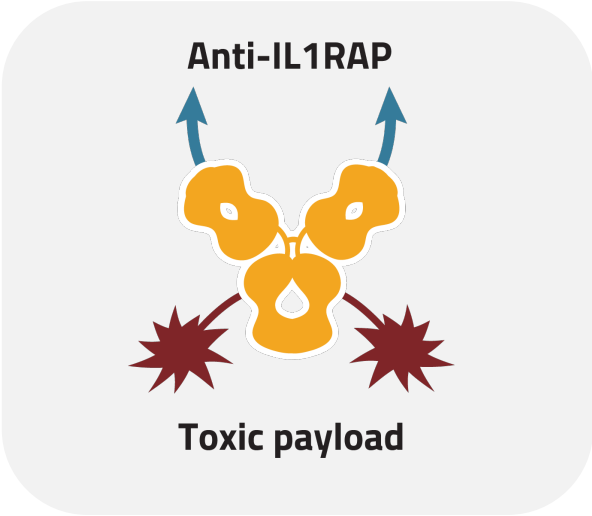
IL1RAP blockade is a potent way to block inflammation in preclinical and translational ex vivo models

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



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



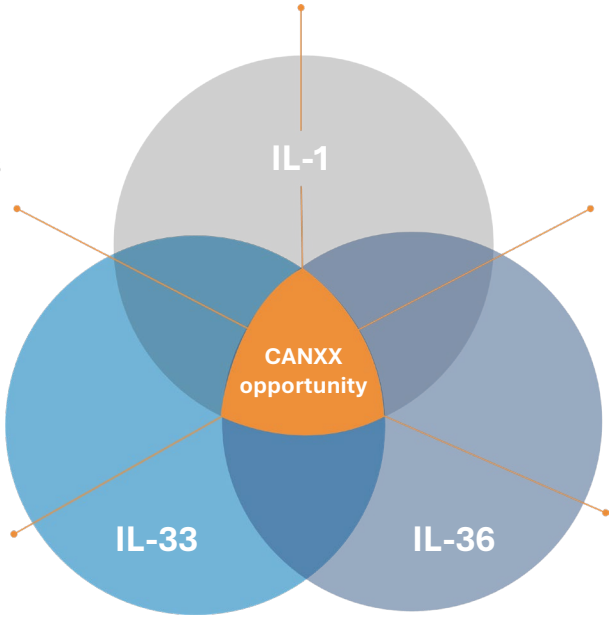
CAN14 and IL1RAP-based Bispecific Antibodies

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

Joints
- Osteoarthritis
- Rheumatoid arthritis
- Gout

Skin
- Hidradenitis Suppurativa (HS)
- Atopic dermatitis
- Psoriasis/GPP
- Palmoplantar Pustulosis
- Pyoderma Gangrenosum

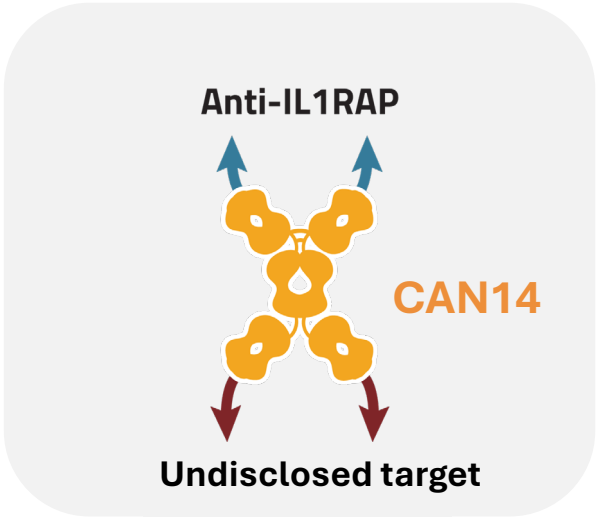
Respiratory
- COPD
- Asthma
- Covid lung
- IPF



Supported by CHBC/Lumanity Research

Intestine
- Crohn's disease
- Ulcerative colitis

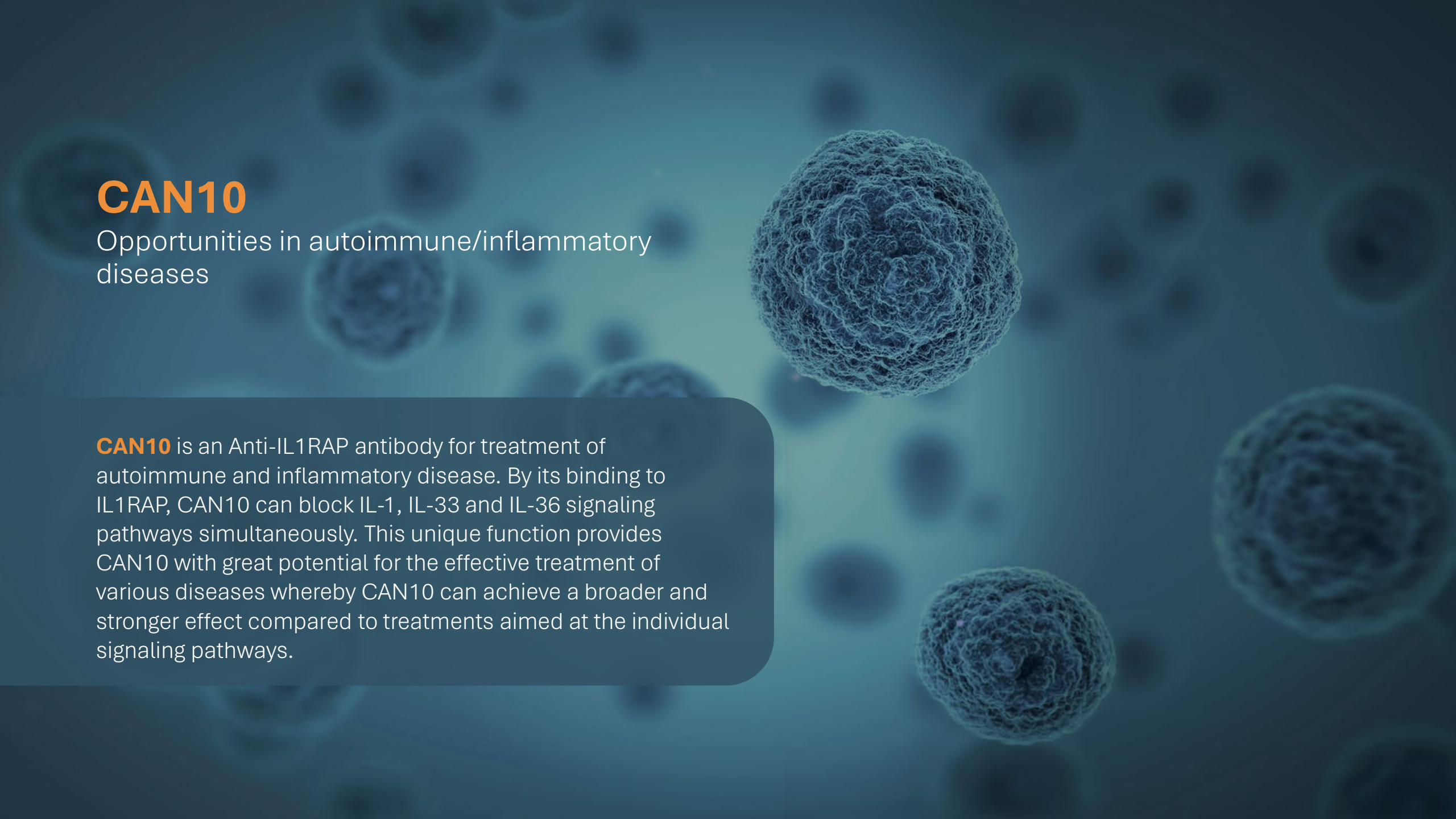
Other/multi/ systemic/acute
- SSc
- Lupus / SLE
- ANCA
- Myocardial infarction
- CV disease
- Endometriosis
- Transplant rejection
- Longevity
- Neuroinflammation



- 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

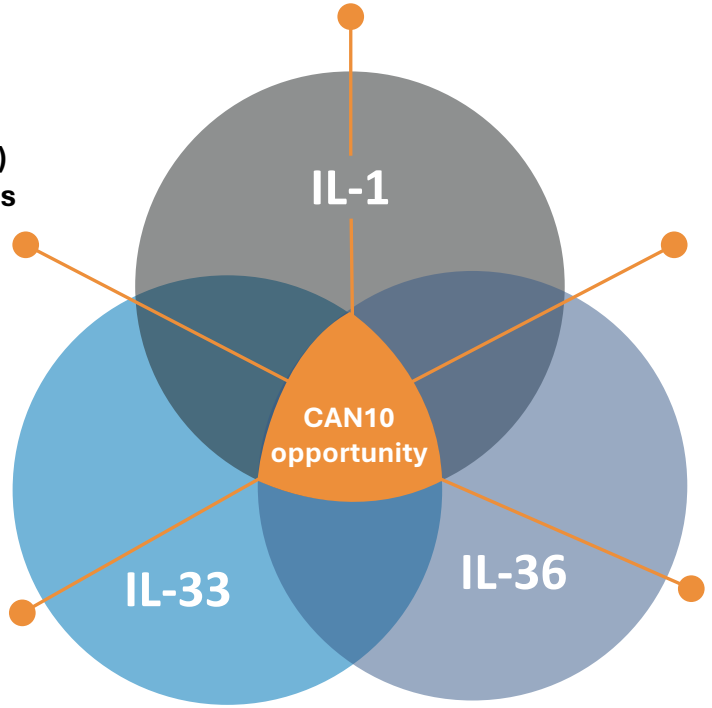
VALIDATION OF IL1RAP AS A TARGET IN INFLAMMATION AND OF CANTARGIA'S ANTIBODY PLATFORM

CAN10 Provides a Unique Opportunity to Block IL-1 Super-family Signaling

- The IL-1 superfamily of ligands and receptors is primarily associated with acute and chronic inflammation[1]
- Strong evidence of IL-1 family cytokines (IL-1, IL-33, IL-36) is driving multiple inflammatory diseases
- Individual blockade of IL-1 family members[2] have not resulted in sufficient clinical efficacy in diverse diseases
- CAN10 broader mechanism is highly relevant in dermatological, fibrotic and cardiovascular diseases

Joints
- Osteoarthritis
- Rheumatoid arthritis
- Gout

Skin
- **Hidradenitis Suppurativa (HS)**
- **Atopic dermatitis**
- Psoriasis/GPP
- Palmoplantar Pustulosis
- Pyoderma Gangrenosum



Intestine
- Crohn's disease
- Ulcerative colitis

Respiratory
- COPD
- Asthma
- Covid lung
- IPF

Other/multi/ systemic/acute
- SSc
- Lupus / SLE
- ANCA
- Myocardial infarction
- CV disease
- Endometriosis
- Transplant rejection

Supported by CHBC/Lumanity Research

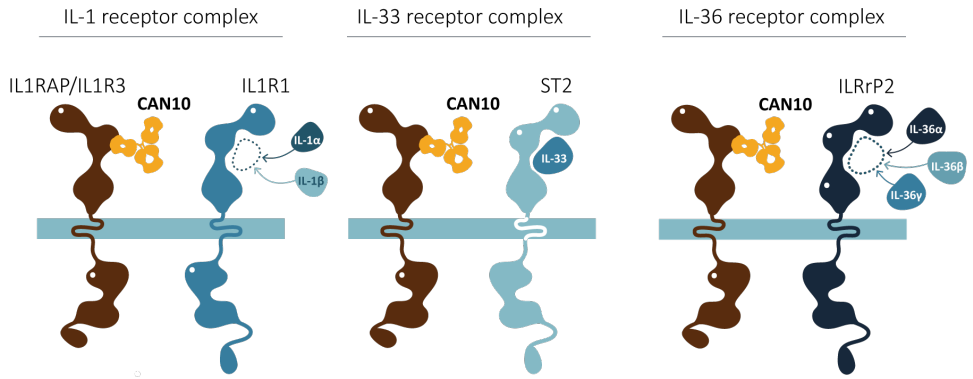
1. Interleukin-1 in the pathogenesis and treatment of inflammatory diseases - Charles A. Dinarello, Blood (2011) 117 (14): 3720–3732.
2. Canakinumab, spesolimab

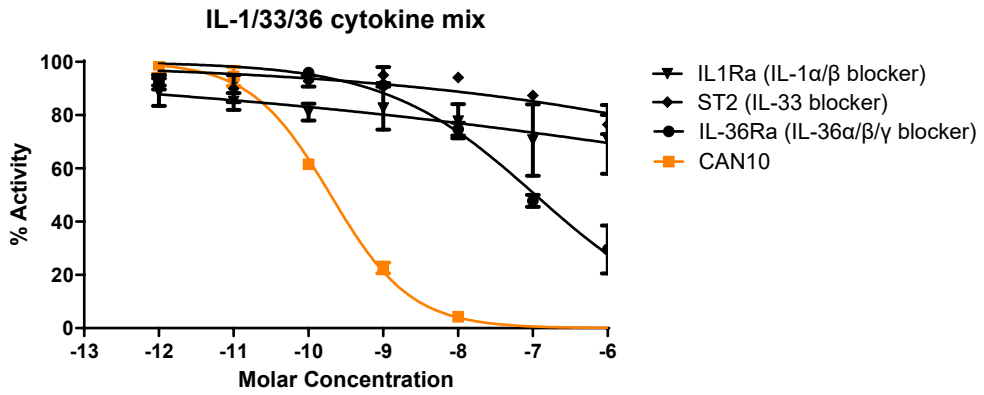
Cantargia

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-1α/β, IL-33 and IL-36α/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**





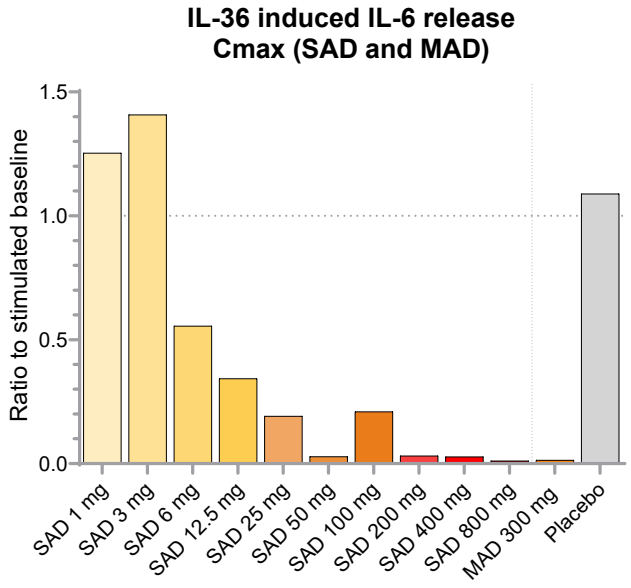
HEKblue NFkB reporter cell line assay

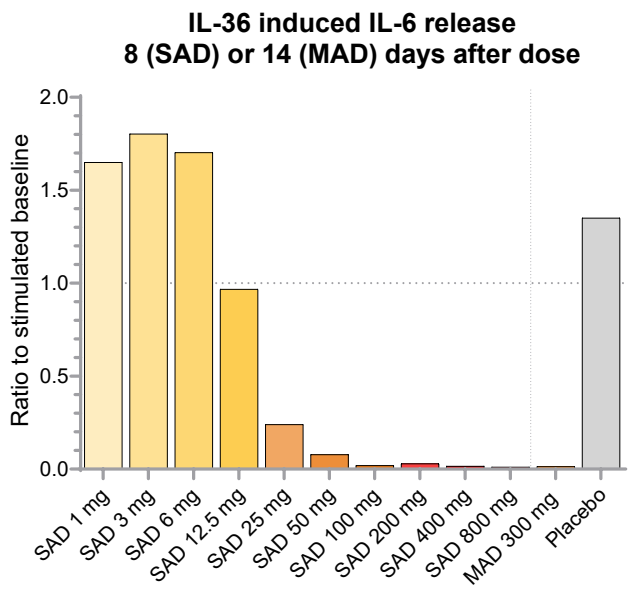
CAN10 IS UNDERGOING PHASE 1 (SAD/MAD) DEVELOPMENT - NO SAFETY ISSUES REPORTED

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

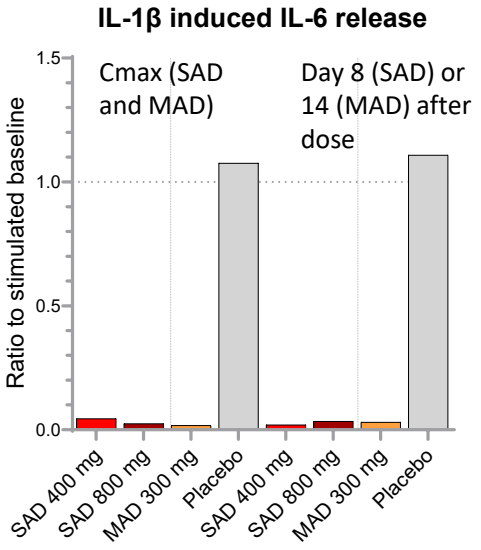
- Inhibition of IL-36 and IL-1β signaling documented at C_{max} (SAD and MAD) and day 8 (SAD) or 14 days after 3rd dose (MAD) → long lasting effect

IL-36 inhibition



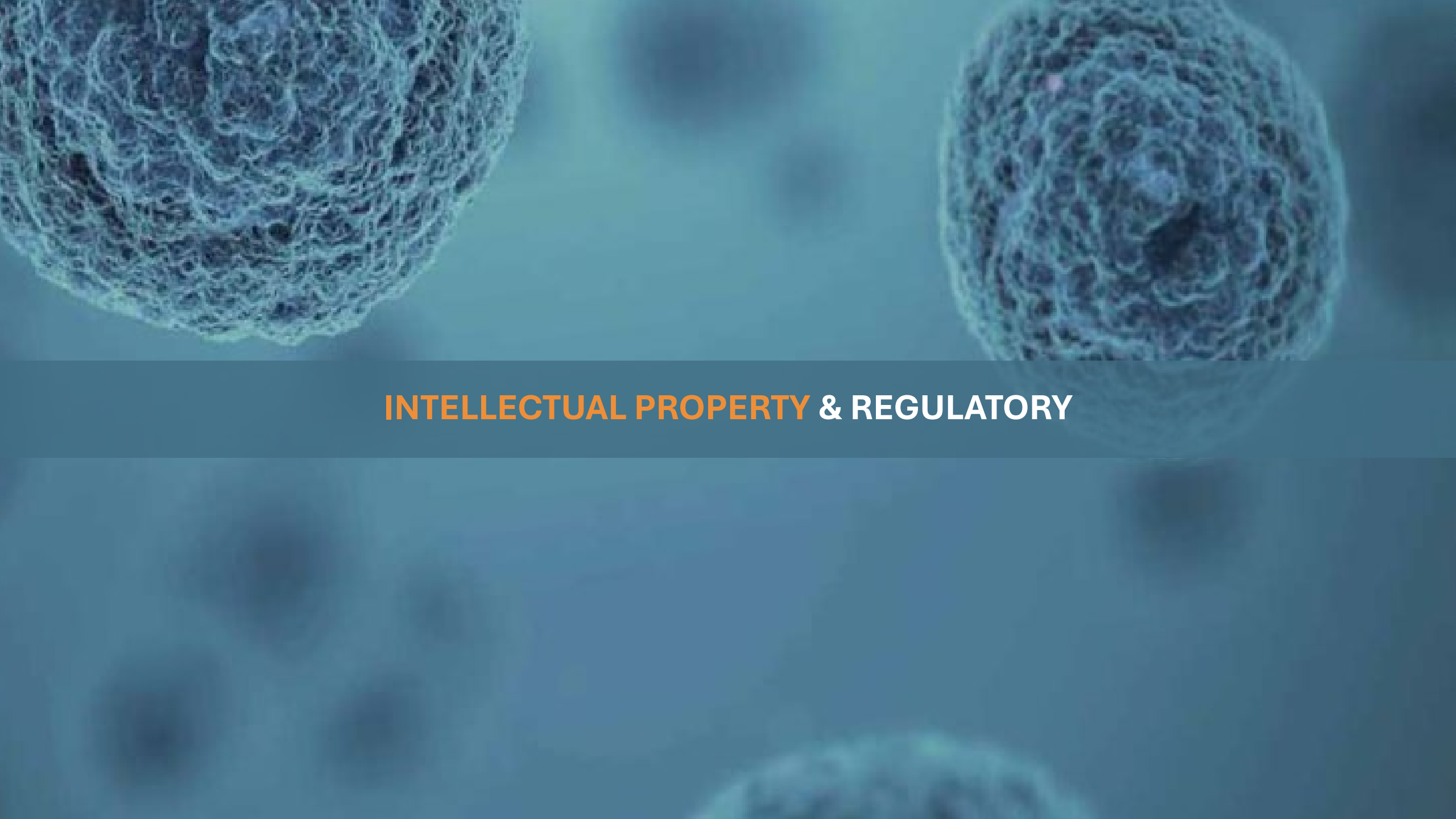


IL-1β inhibition



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

Cantargia



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.

KEY FINANCIALS & INVESTMENT HIGHLIGHTS

Key Financial and Share Information

Financials

- Cash position: SEK **258m** (March 31, 2026)
- Runway: Into 2028 (with current commitments)

The Cantargia Share

- Listing: **Nasdaq Stockholm** main market
- Ticker: **CANTA.ST**
- Number of shares: **248,611,655** (March 31, 2026)
- Share price: SEK **4.085** (March 31, 2026)
- Market Cap: SEK **1,016m** (March 31, 2026)
- Average Daily Liquidity: SEK **17.4m** (March 31, Year-to-Date)

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, March 31, 2026

Owner	Number of shares	Capital/Votes (%)
Fourth Swedish National Pension Fund	23,551,565	9.47%
Avanza Pension	15,217,063	6.12%
Handelsbanken Fonder	7,103,219	2.86%
American Century Investment Management	6,827,250	2.75%
Henrick Schill	4,235,663	1.70%
Brushamn Invest AB	3,391,740	1.36%
The Invus Group	3,161,602	1.27%
Nordnet Pensionsförsäkring	3,076,991	1.24%
Stefan Johansson Restaurang AB	2,236,334	0.90%
Tibia Konsult AB	2,000,000	0.80%
Other	177,810,228	71.52%
Total	**248,611,655**	**100.0%**

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