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

Corporate Presentation

January 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: Redefining Cancer & Inflammation Therapy

Global Leader in IL1RAP Targeting

Nadunolimab Oncology	**Pivotal trial ready in PDAC** FDA Fast Track Designation
CAN10 Inflammatory diseases	**Target validated by Otsuka**
CAN14 & CANxx ~200 mAB ILRAP library	**A Unique platform of IL1RAP mAbs**

Strong Corporate Position

Otsuka deal:

- → Otsuka to develop and commercialize CAN10 following acquisition with upfront of **$33** million and total deal value of **$613** million
- → Validation of target & technology

Key Financials:

- → Cash position: **SEK 339m** (end Q3, 2025)
- → Runway into 2028, with current commitments

CURRENT DEVELOPMENT FOCUSES ON NADUNOLIMAB IN 1L METASTATIC PDAC AND NEXT GENERATION IL1RAP ANTIBODIES

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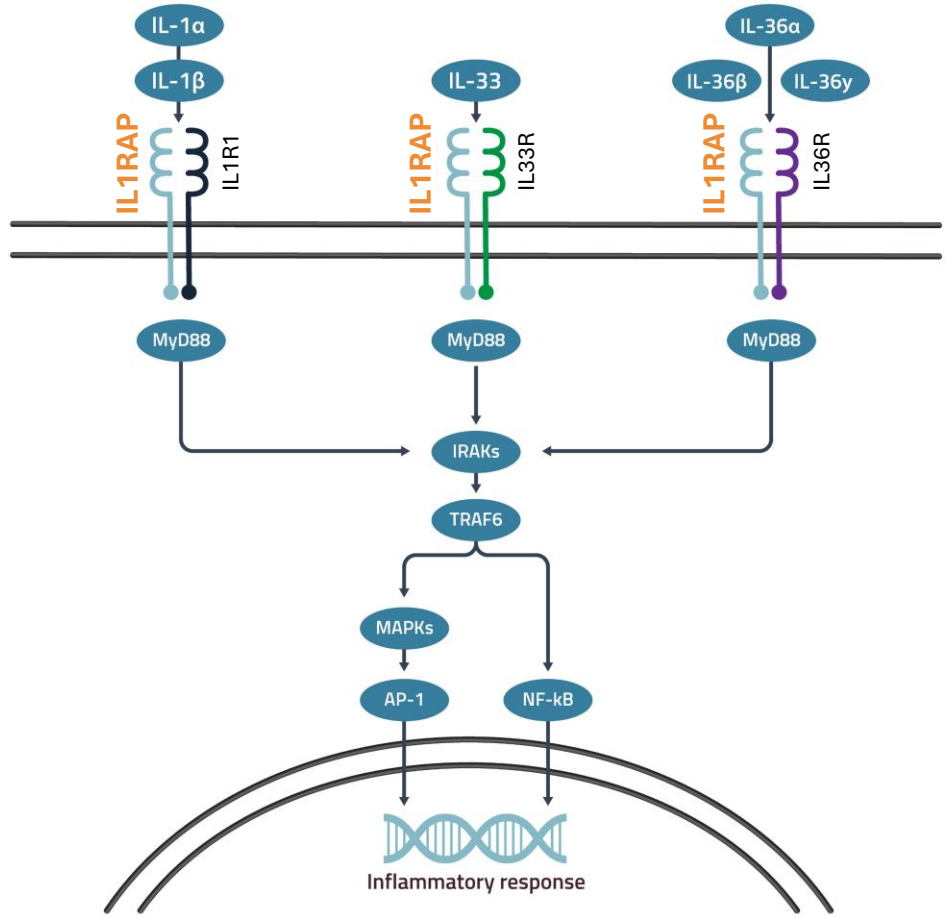
IL1RAP – Target with Numerous Opportunities

- Opportunity to target three signaling systems, IL-1, IL-33 and IL-36, counteracting redundancy and increasing efficacy
- Distinct activity in several hard-to-treat models of inflammation and cancer
- Cantargia antibodies developed for inflammation and cancer, technology validated by Otsuka deal
- Framework for additional signaling blockade at inflammatory sites (Bispecifics) or for delivery of payloads into a tumor microenvironment (ADCs)



NUMEROUS OPPORTUNITIES IN COMMON AND DIFFICULT TO TREAT DISEASES WITH HIGH UNMET MEDICAL NEED

IL1RAP Pipeline supported by Unique Platform

Asset	Target	Indication	Discovery	IND Enabling	Phase 1	Phase 2	Phase 3	Partner
Proprietary Pipeline								
Nadunolimab	IL1RAP	PDAC*						
		NSCLC						
CAN14	IL1RAP BsAb**	Autoimmune diseases						
CANxx	New development programs*** through unique IL1RAP platform							
Strategic Partnership								
CAN10	IL1RAP	Autoimmune diseases						Otsuka

PDAC – pancreatic ductal adenocarcinoma; **NSCLC** – non-small cell lung cancer

*) FDA Fast Track Designation & Orphan Drug Designation; EU Orphan Drug Designation

**) IL1RAP Bispecific Antibody, 2nd target undisclosed

***) E.g. Anti-IL1RAP mAbs, Anti-IL1RAP BsAbs, Anti-IL1RAP ADCs

Executive Management Team with Proven and Relevant Expertise

Hilde Steineger
CEO

Patrik Renblad
CFO

David Liberg
CSO

Ton Berkien
CBO

Wolfram Dempke
CMO

~~AN~~ 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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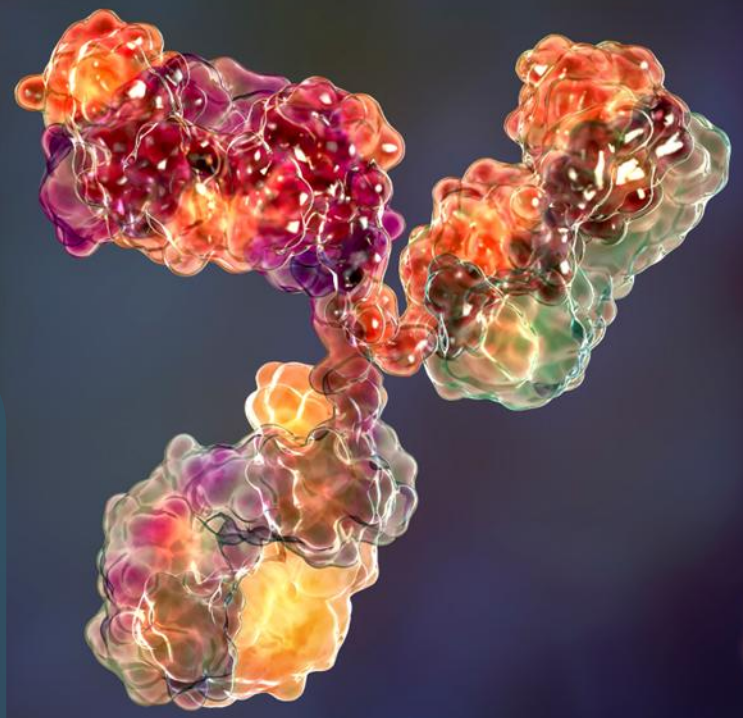
Nadunolimab

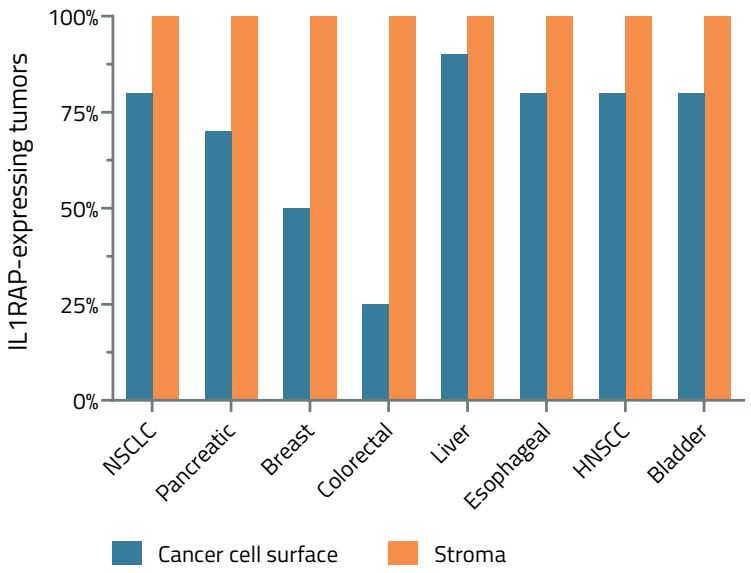
Combat cancer and enhance the immune system's ability to destroy cancer cells



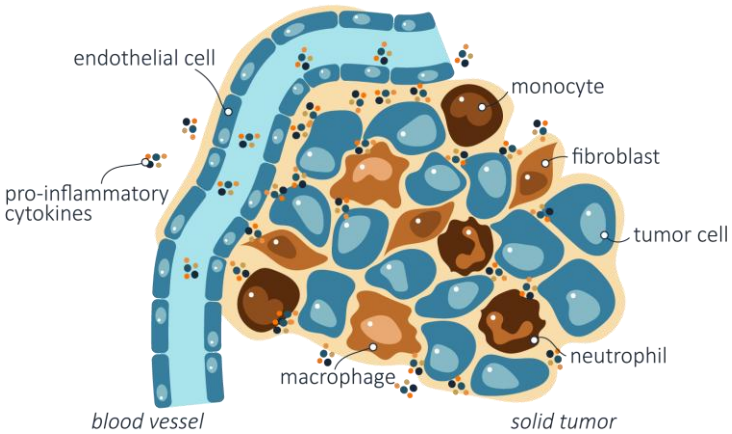
Nadunolimab (CAN04) is an Anti-IL1RAP antibody for treatment of various cancer types. Nadunolimab binds strongly to its target molecule IL1RAP, expressed on tumor cells from many types of cancer. Nadunolimab blocks the signaling of interleukin-1, alpha and beta, thereby limiting tumor development as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

IL1RAP Overexpressed in Most Solid Tumors

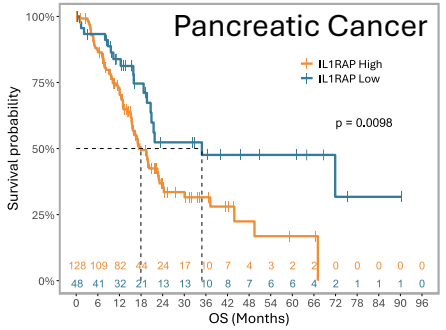
IL1RAP IS EXPRESSED ACROSS SOLID TUMOR TYPES

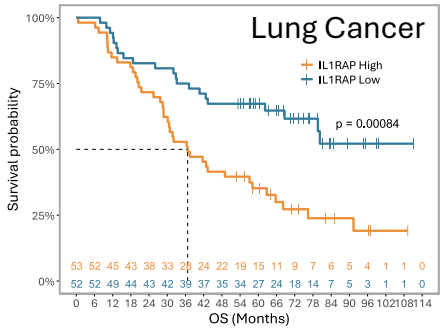


TUMOR-PROMOTING CELLS EXPRESS IL1RAP IN THE TUMOR MICROENVIRONMENT



IL1RAP IS A PROGNOSTIC MARKER IN SEVERAL CANCER INDICATIONS





RNA data from TCGA

IL1RAP – DISTINCTLY OVEREXPRESSED IN TUMORS; LOW EXPRESSION IN NORMAL TISSUE

NSCLC – Non-Small Cell Lung Cancer, HNSCC – Head and Neck Squamous Cell Carcinoma

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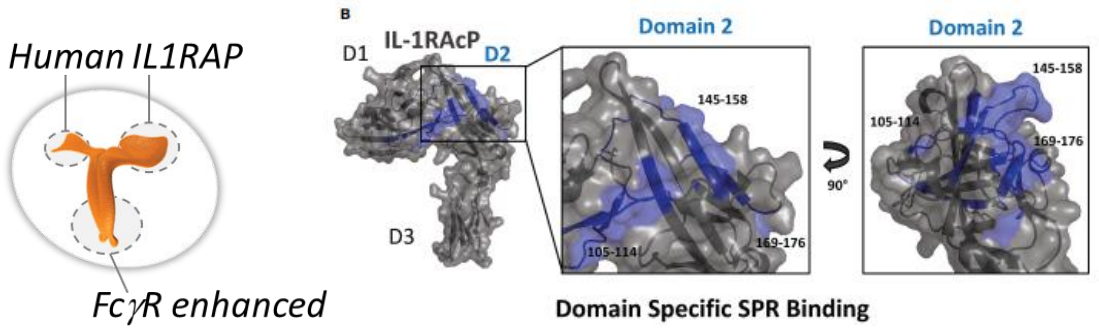
Nadunolimab mAb Properties

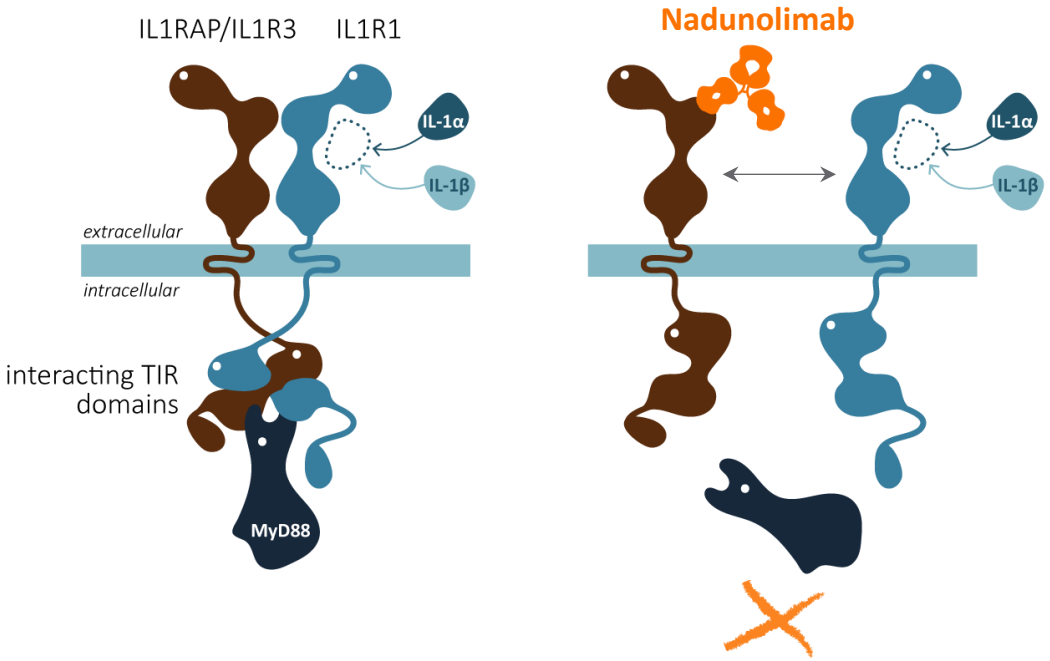
- Nadunolimab is a humanized anti-IL1RAP monoclonal immunoglobulin G1 (IgG1) antibody with a molecular weight of 144 kDa (non-glycosylated)
- It has 2 glycan moieties deficient of fucose in the Fc-region for enhanced antibody-dependent cellular cytotoxicity (ADCC) and antibody dependent cellular phagocytosis (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.

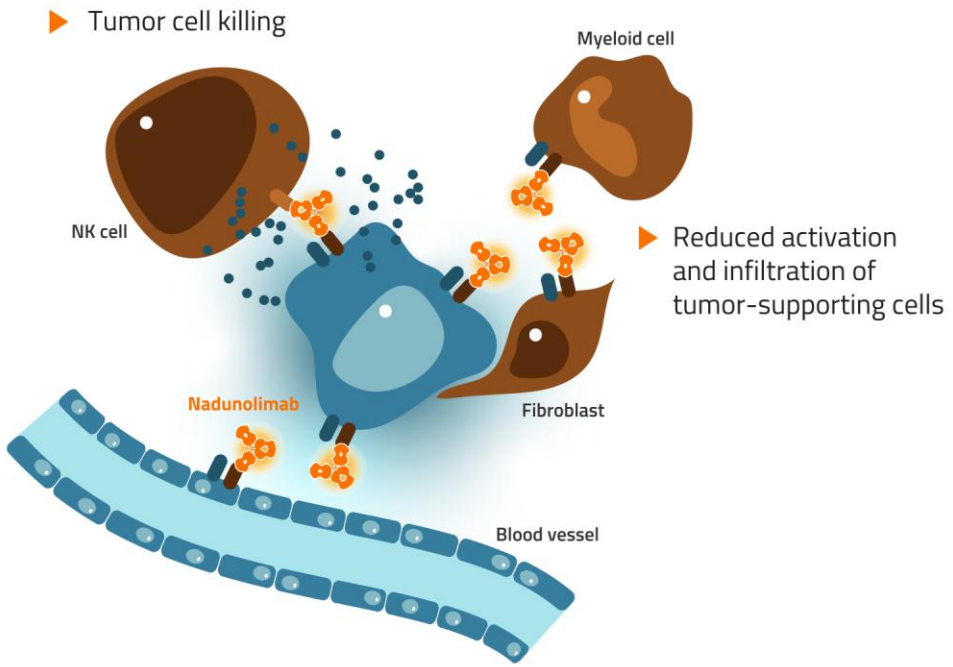


Fields et al Front. Immunol. 2021

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

Nadunolimab Provides Unique Opportunities to Treat Cancer by IL-1α/β Blockade and ADCC





NADUNOLIMAB ATTACKS TUMOR CELLS AND DISRUPTS TUMOR PROMOTING CIRCUITRY

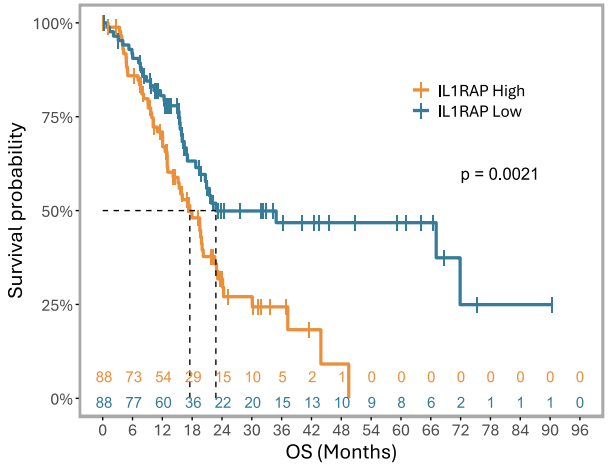
ADCC – Antibody-Dependent Cellular Cytotoxicity; NK – Natural Killer; TIR – Toll-Interleukin-1 Receptor

Overview of Clinical Studies with nadunolimab

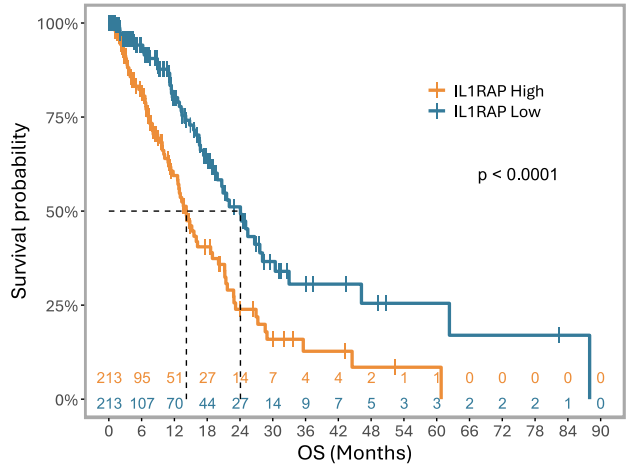
Study (mITT)	NCT number	Design	Status
CANFOUR (n=113 in combination)	NCT03267316	Nadunolimab in combination with gemcitabine/nab-paclitaxel in 1L PDAC and gemcitabine/cisplatin or carboplatin/pemetrexed in NSCLC	**Completed** PDAC manuscript published CCR NSCLC manuscript in Lung Cancer
CIRIFOUR (n=15)	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**

PANCREATIC DUCTAL ADENOCARCINOMA – PDAC

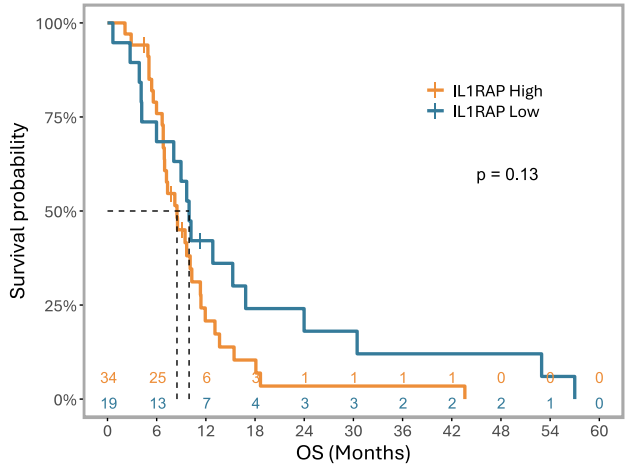
High IL1RAP Expression Linked to Poor Outcome in Patients with Pancreatic Tumors



TCGA RNA data*
PDAC patients all stages



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



BIOPAC biopsy protein stainings***
PDAC patients stage IV

Analysis of RNA-data by AI-company show **two subnetworks enriched for IL1RAP-high genes**, the genes in the two networks correlate to worse OS and basal-like subtype.

HIGH IL1RAP EXPRESSION IN PDAC TUMORS IS STRONGLY CONNECTED TO POOR SURVIVAL

* Zhang et al, Journal of Hematology & Oncology, 2022
** Hansen et al. JITC 2024
*** Millrud-Rydberg et al., AACR Special Conference on advances in Pancreatic Cancer Research 2025, poster #B091

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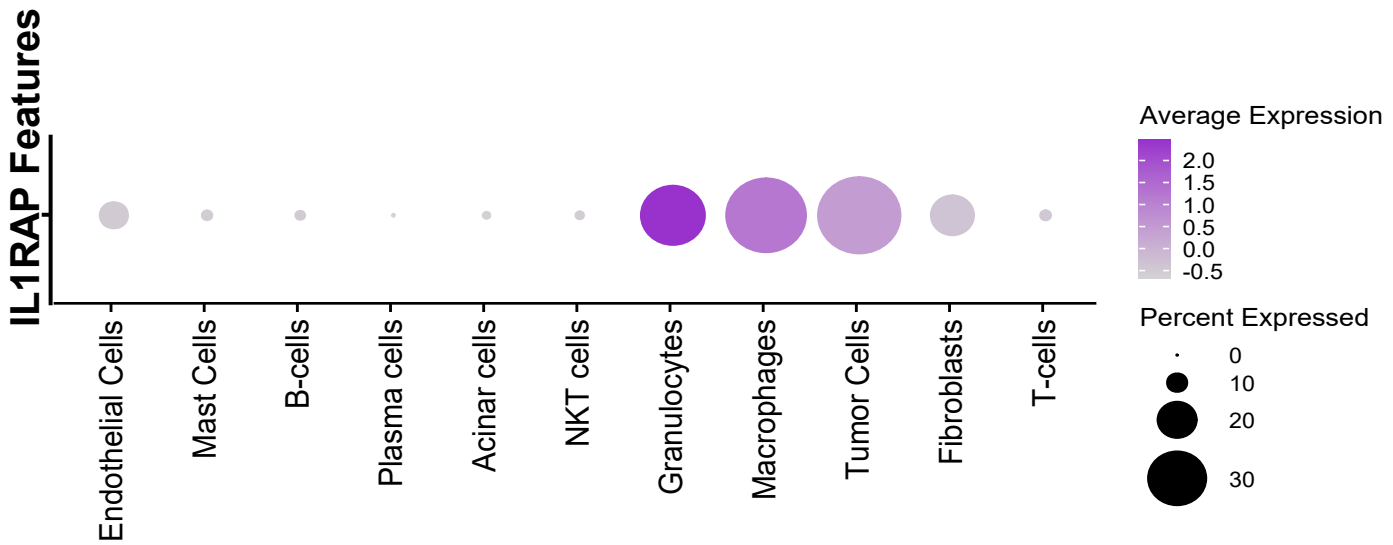
IL1RAP is Expressed by Tumor Cells, Myeloid Cells and Fibroblasts in PDAC



Single-cell RNA sequencing of tumors from PDAC patients (n=16) shows IL1RAP mainly expressed by granulocytes, macrophages, tumor cells and fibroblasts in the TME.

Courtesy of Jashodeep Datta MD, PhD, University of Miami. Ref Steele et al Nat Cancer (2020)

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

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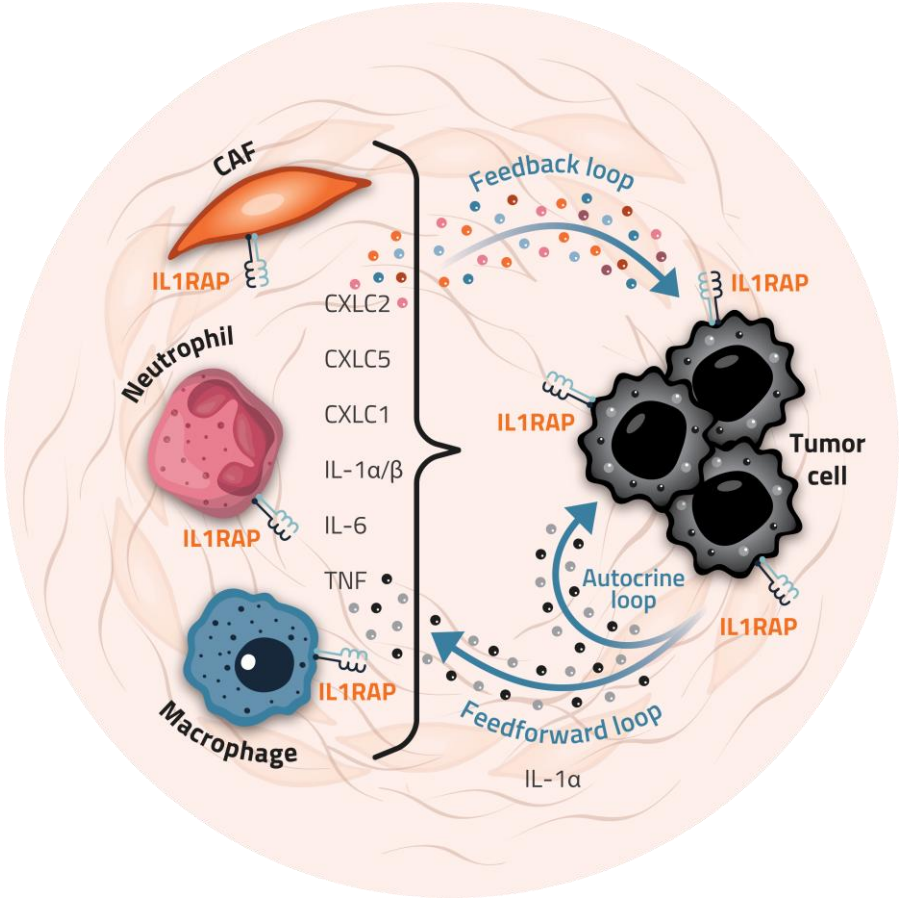
IL1RAP – a Fundamental Driver in the PDAC TME

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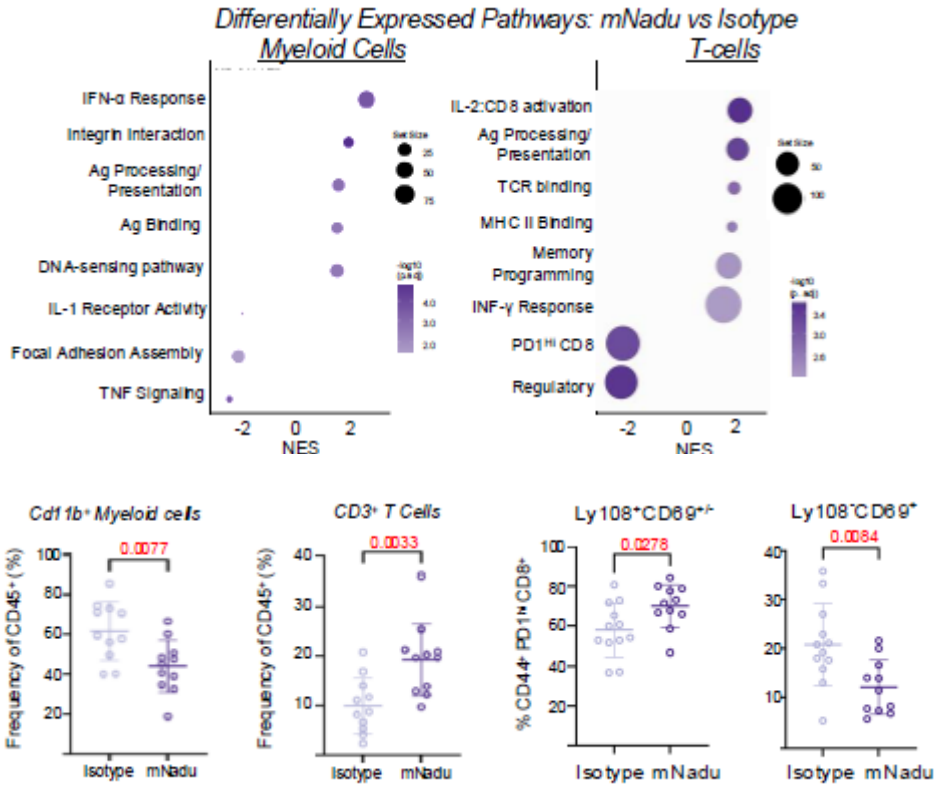
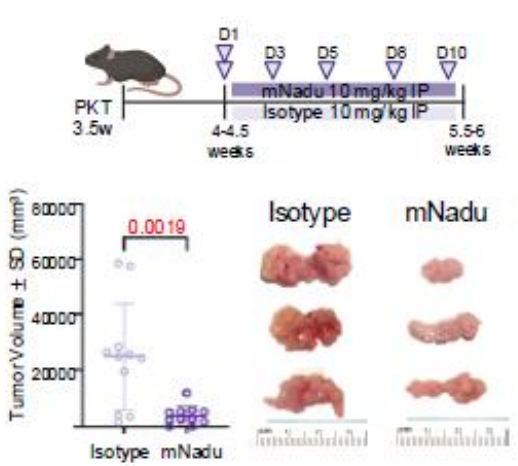
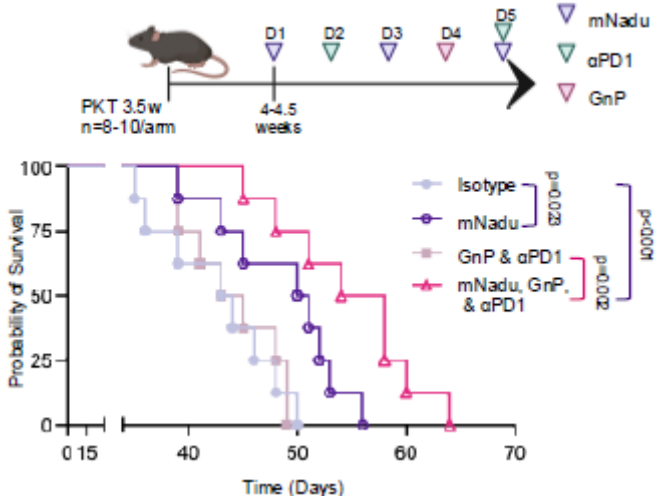
IL1RAP Targeting has Distinct Effects in an Aggressive and Treatment Resistant Preclinical Model of PDAC

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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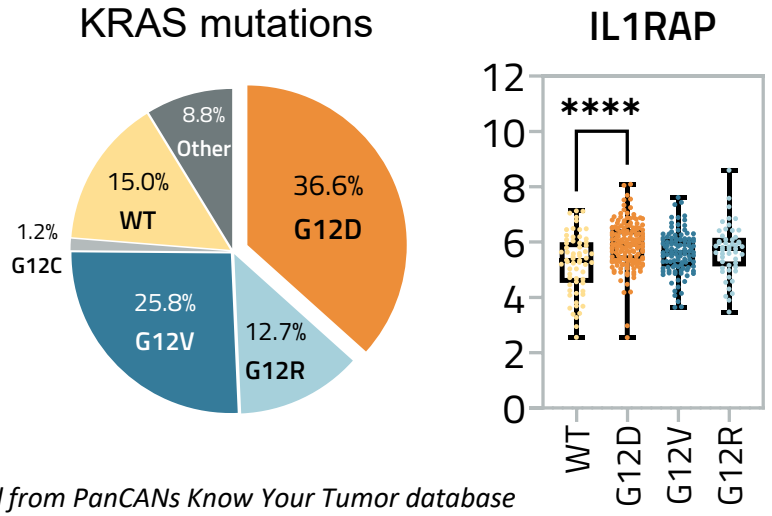
KRAS in PDAC – Connections to IL1RAP

KRAS and PDAC

- The KRAS gene encodes a protein involved in cell signaling and regulation of cell growth
- Mutations in KRAS lead to the constant activation of the RAS signaling pathway, which drives uncontrolled cell proliferation and survival
- KRAS mutations are key drivers of pancreatic cancer, present in **~90% of PDAC tumors**
  - G12D and G12V are most frequent, accounting for 65% (Hoffman 2022)
- G12D shows worse outcome compared to other KRAS status

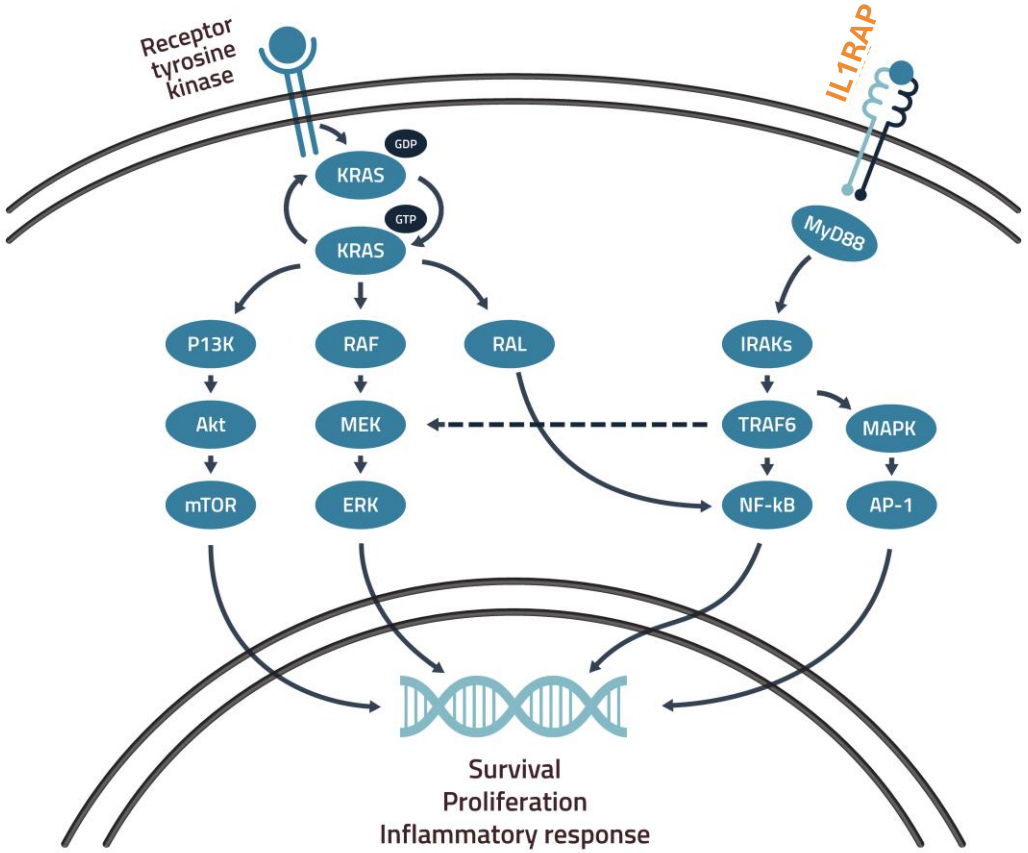
IL1RAP and KRAS

- IL1RAP RNA expression is higher in pancreatic tumors compared to normal pancreas
- High IL1RAP RNA expression strongly correlates to poor survival
- IL1RAP RNA expression is significantly increased in KRAS mutated PDAC, especially in the G12D population, compared to WT
- IL1RAP and KRASG12D increase in late stage PDAC



Data retrieved from PanCANs Know Your Tumor database

IL1RAP Signaling Acts in Parallel to KRAS to Shape the Phenotype of PDAC



- 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 correlate to poor survival in patients with or without KRAS mutations, **IL1RAP act independently of KRAS**
- Preclinical data show both strong tumor growth inhibition and TME remodeling by IL1RAP targeting, also in an aggressive KRAS-driven model*

IL1RAP IS A MARKER FOR POOR SURVIVAL AND THERAPEUTIC RESISTANCE INDEPENDENTLY OF KRAS

**in collaboration with Dr. Jashodeep Datta, Sylvester Comprehensive Cancer Center, Miller School of Medicine, U Miami*

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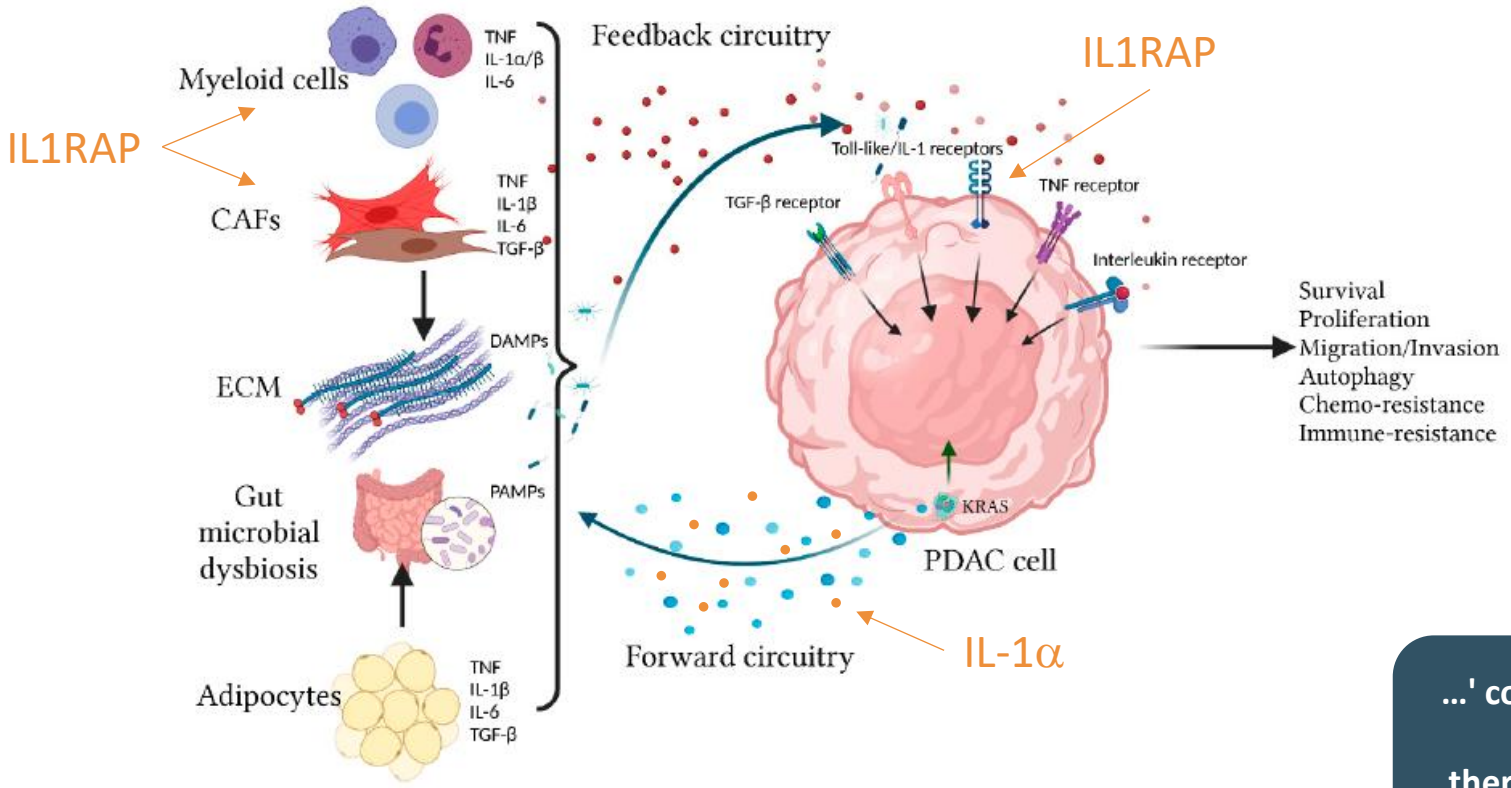
KRAS and IL1RAP in Pancreatic Cancer – Setting Up and Maintaining the Tumor Microenvironment



- KRAS induces a microenvironment that may ultimately lead to a self-sustaining inflammatory loop, that will reduce the dependency of the KRAS mutation itself
- KRAS inhibitors and IL1RAP blockade is an opportunity to strike both tumor driving intrinsic and extrinsic pathways
- IL1RAP targeting and *e.g.* chemotherapy synergizes through cell killing and inhibition of induced resistance pathways, a similar mechanism may be highly relevant for IL1RAP and KRAS inhibition

…' co-targeting the key signaling nodes within the onco-inflammatory network represents a promising therapeutic strategy that can enhance clinical efficacy'.
(Bansod 2021)

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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

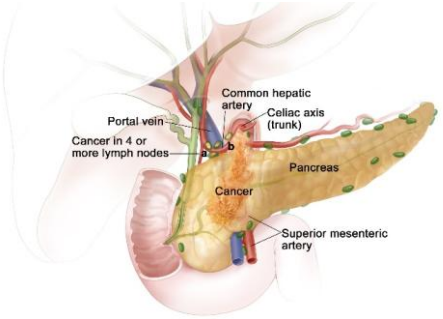
LOCALLY ADVANCED OR METASTATIC DISEASE

Median survival:

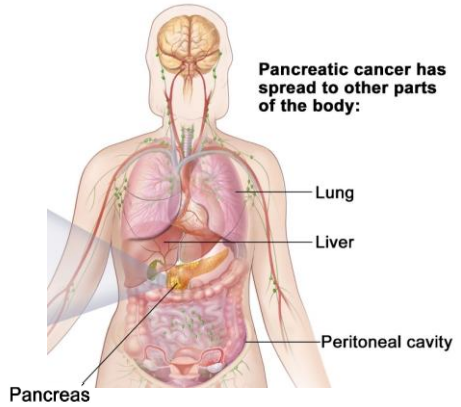
- 8.5 – 11.7 mo

Treatments:

- Gemcitabine + nab-paclitaxel
- Gemcitabine if poorer performance
- FOLFIRINOX only if good patient condition status
- Jan 2023: 1st line NALIRIFOX



Stage III



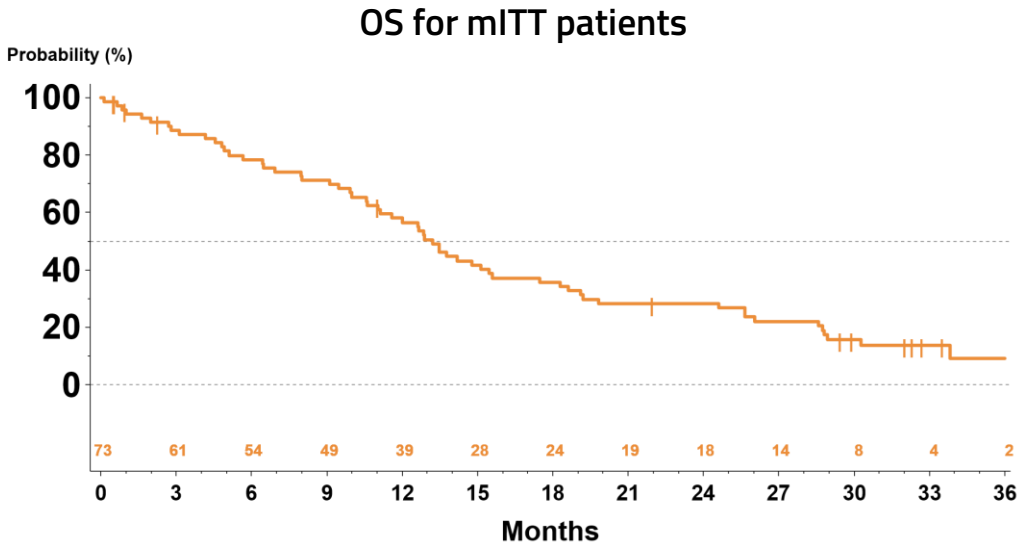
Stage IV

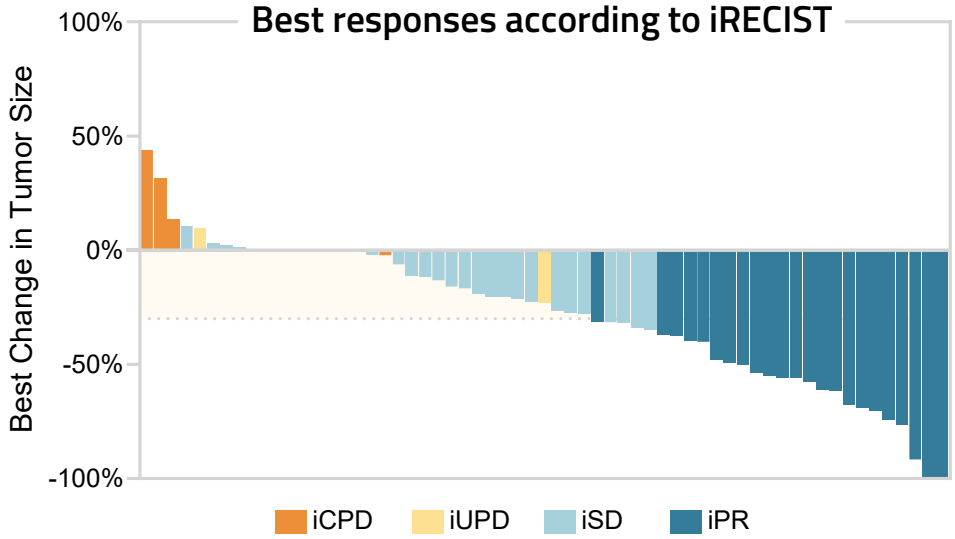
Images from National Cancer Institute

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

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

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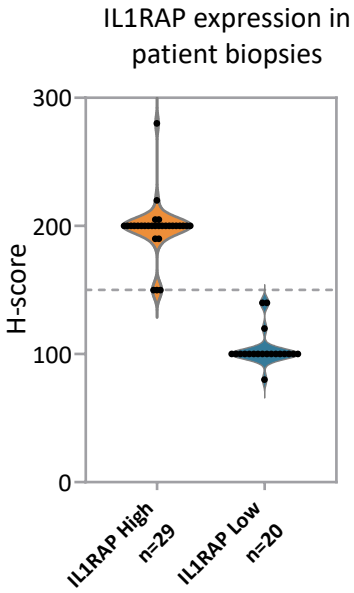
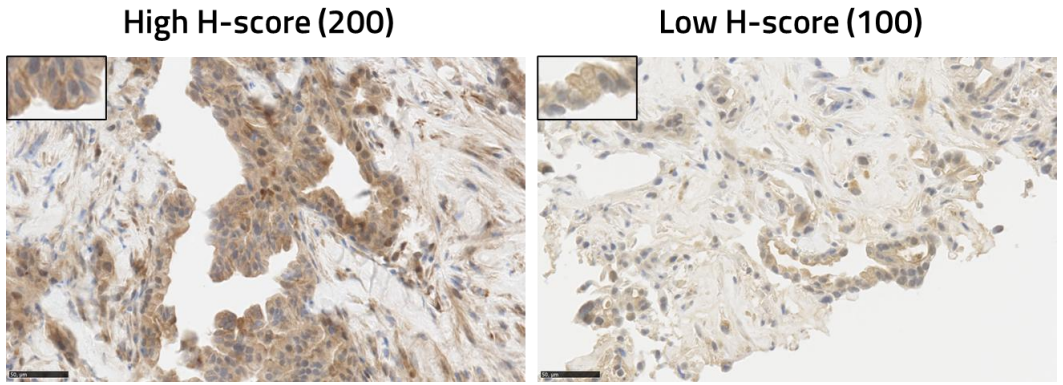
IL1RAP Expression in Tumor Biopsies from PDAC Patients





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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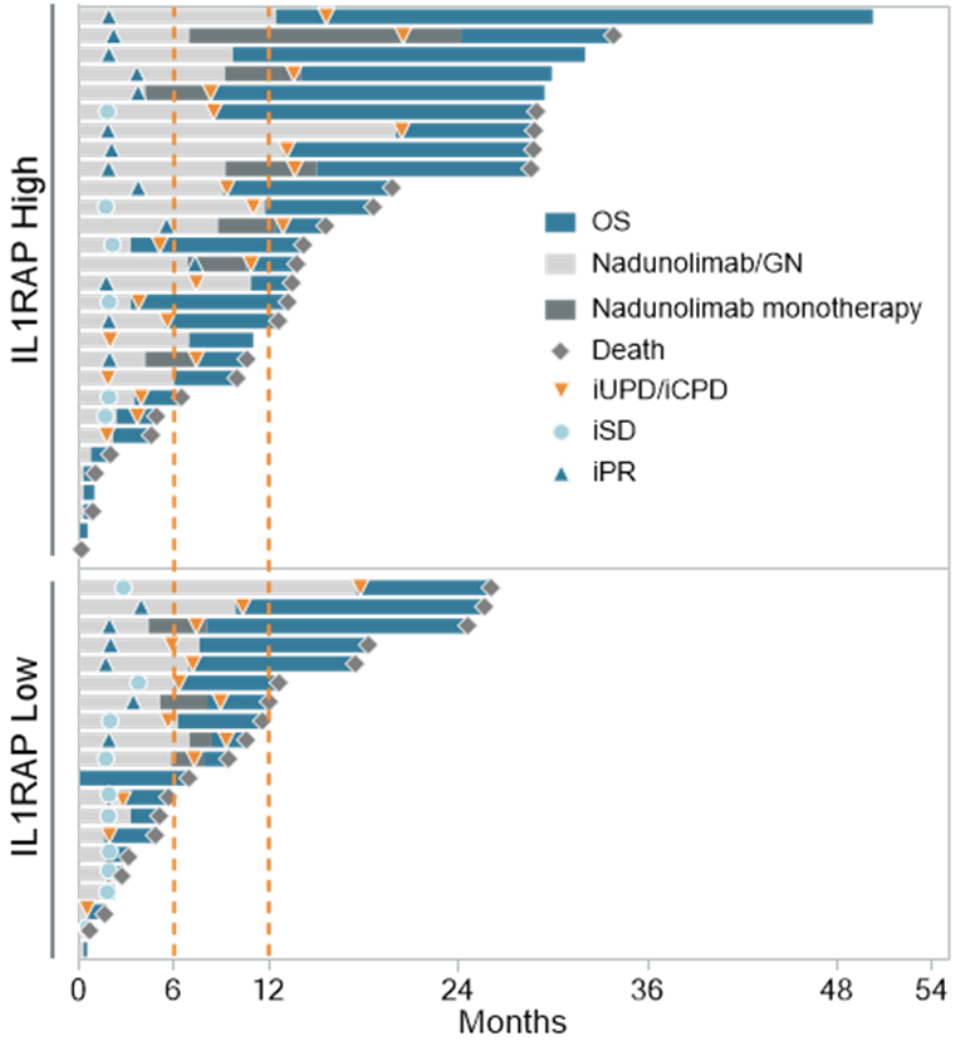
Efficacy and IL1RAP Levels on Tumor Cells in PDAC



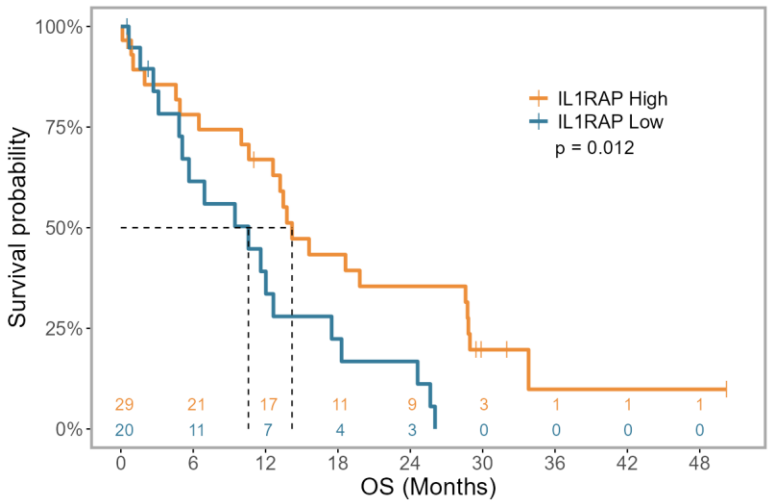
Efficacy analysis for IL1RAP High (n=29) vs IL1RAP Low (n=20) PDAC patients

- Individual assessment of patients in the IL1RAP high and IL1RAP low groups
- Prolonged survival in the IL1RAP high group
- 1-year survival was 67% and 2-year survival 35% in the IL1RAP high group

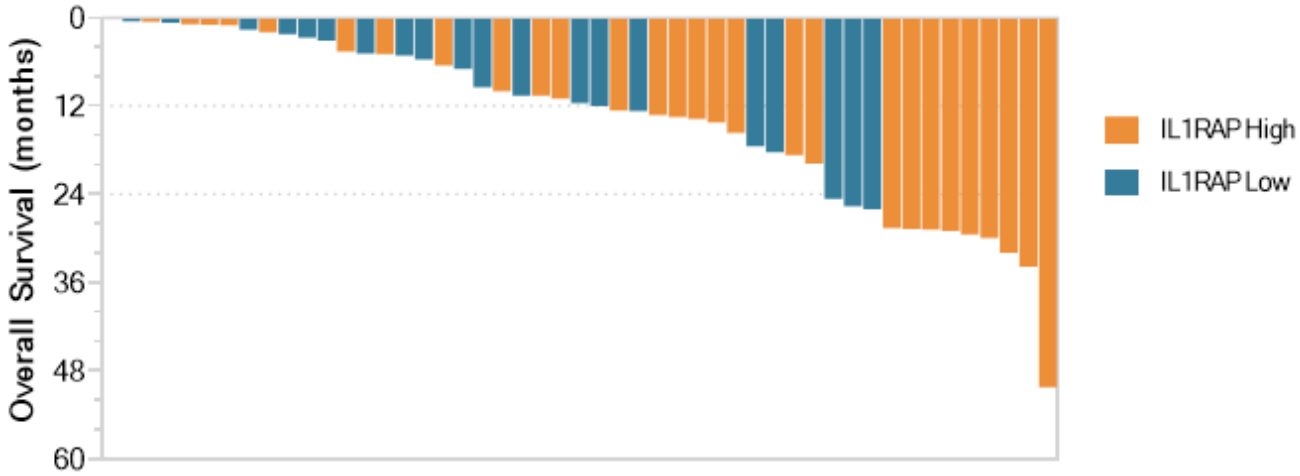
iCPD – Confirmed Progressive Disease; iUPD – Unconfirmed Progressive Disease; iSD – Stable Disease; iPR – Partial Response (all according to iRECIST)

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

OS by IL1RAP subgroup



OS by IL1RAP subgroup



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

- → High IL1RAP expression is a marker for poor prognosis after treatment with gem/nab-paclitaxel
- → A 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). 24-month OS of **35%** in High IL1RAP group.

IL1RAP HIGH PATIENTS SHOW THE STRONGEST BENEFIT

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PDAC – Next steps

IL1RAP Assay Feasibility
Assay development; selection of one clone, one platform and one protocol

Validation
Assay validation; enable patient selection in clinical study

Interaction with regulatory bodies
Approval of clinical development plan including patient selection of high IL1RAP patient population

CDx assay (kit)
CDx kit development at commercial CDx company

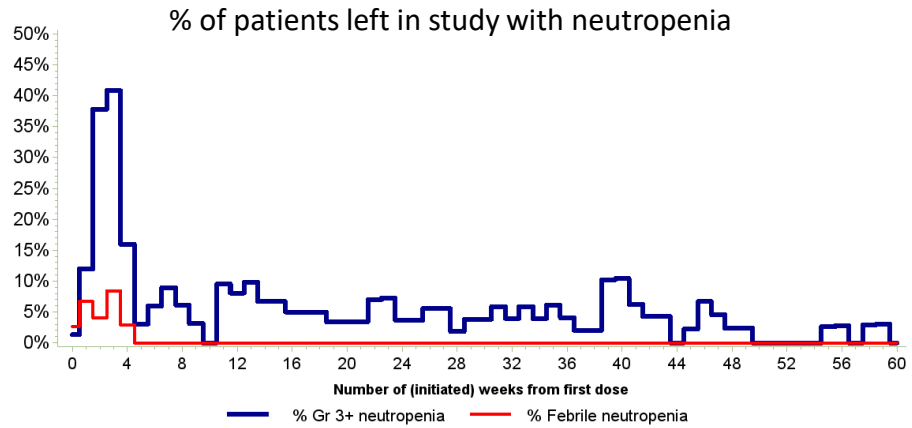
Proposed PDAC clinical study design

- Metastatic PDAC patients selected for high IL1RAP expression
- Treatment naive patients
- Combination with gemcitabine/nab-paclitaxel
- Randomized controlled study
- Primary read-out: OS

IL1RAP ASSAY ALLOWS FOR PATIENT SELECTION IN FUTURE CLINICAL STUDIES

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%



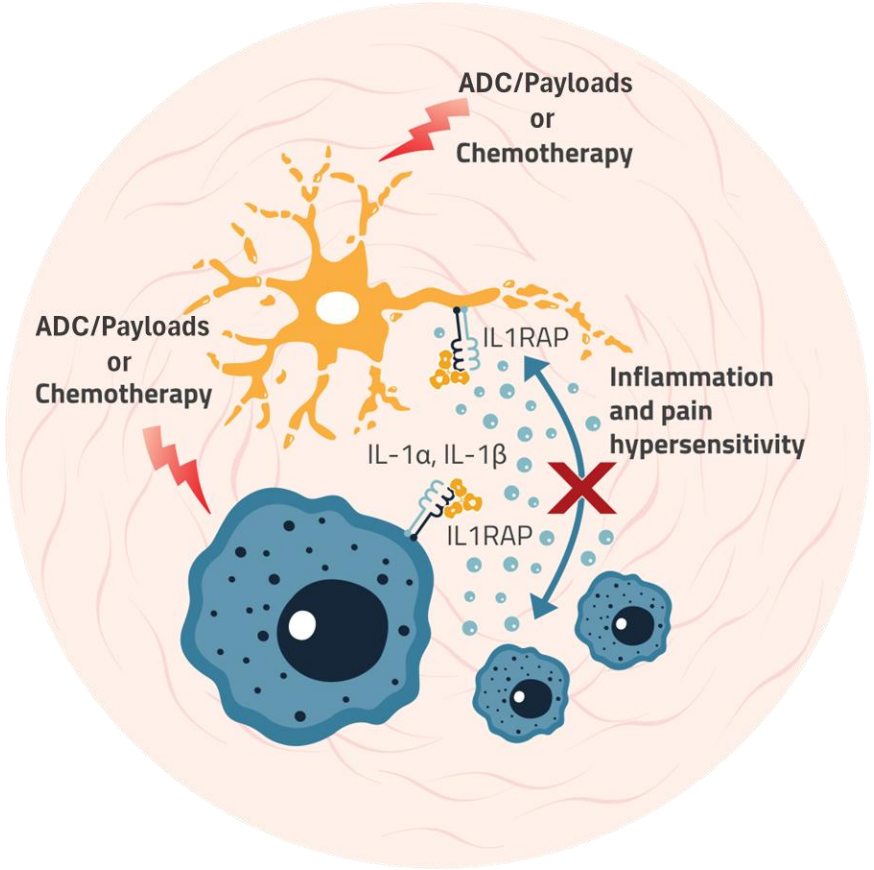
weeks

- 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
- Mandatory G-CSF in cycle 1 to be implemented in upcoming studies
- 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

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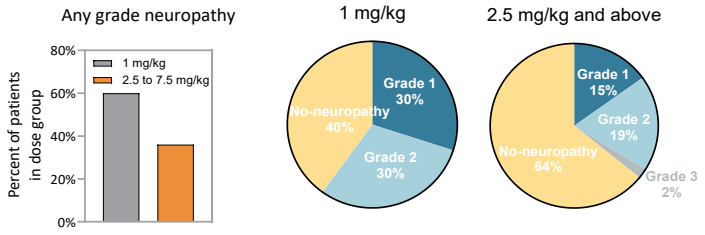
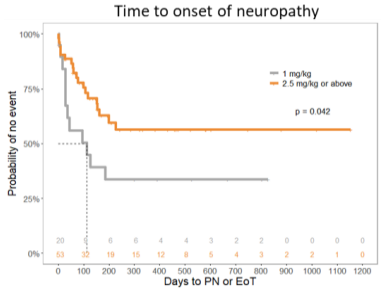
Neuropathy Clinical Data with nadunolimab

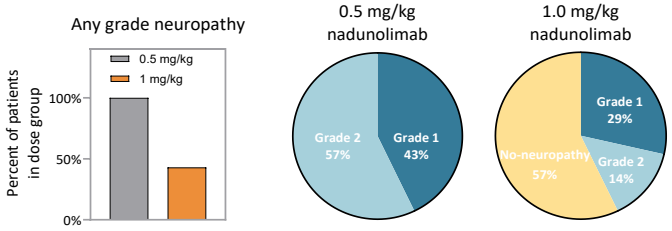
- Ph2 study data from CANFOUR in 1L PDAC patients treated with nadunolimab and gemcitabine/nab-paclitaxel
  - Lower Grade 3-4 peripheral neuropathy than expected from historical controls (1% vs 17%)
  - Reduced incidence as well as delayed onset of all grade neuropathies
- Correlation between nadunolimab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

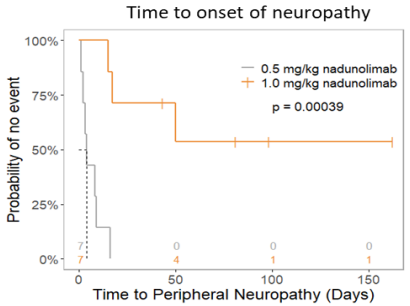
Gemcitabine/nab-paclitaxel



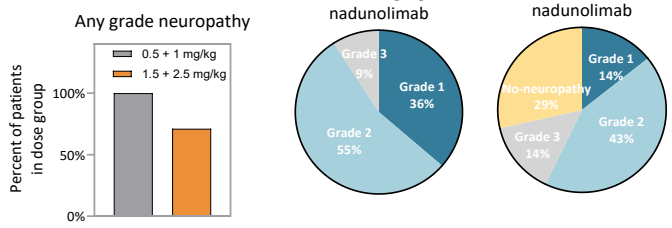


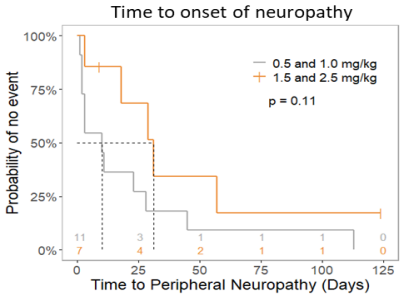
mFOLFOX





mFOLFIRINOX

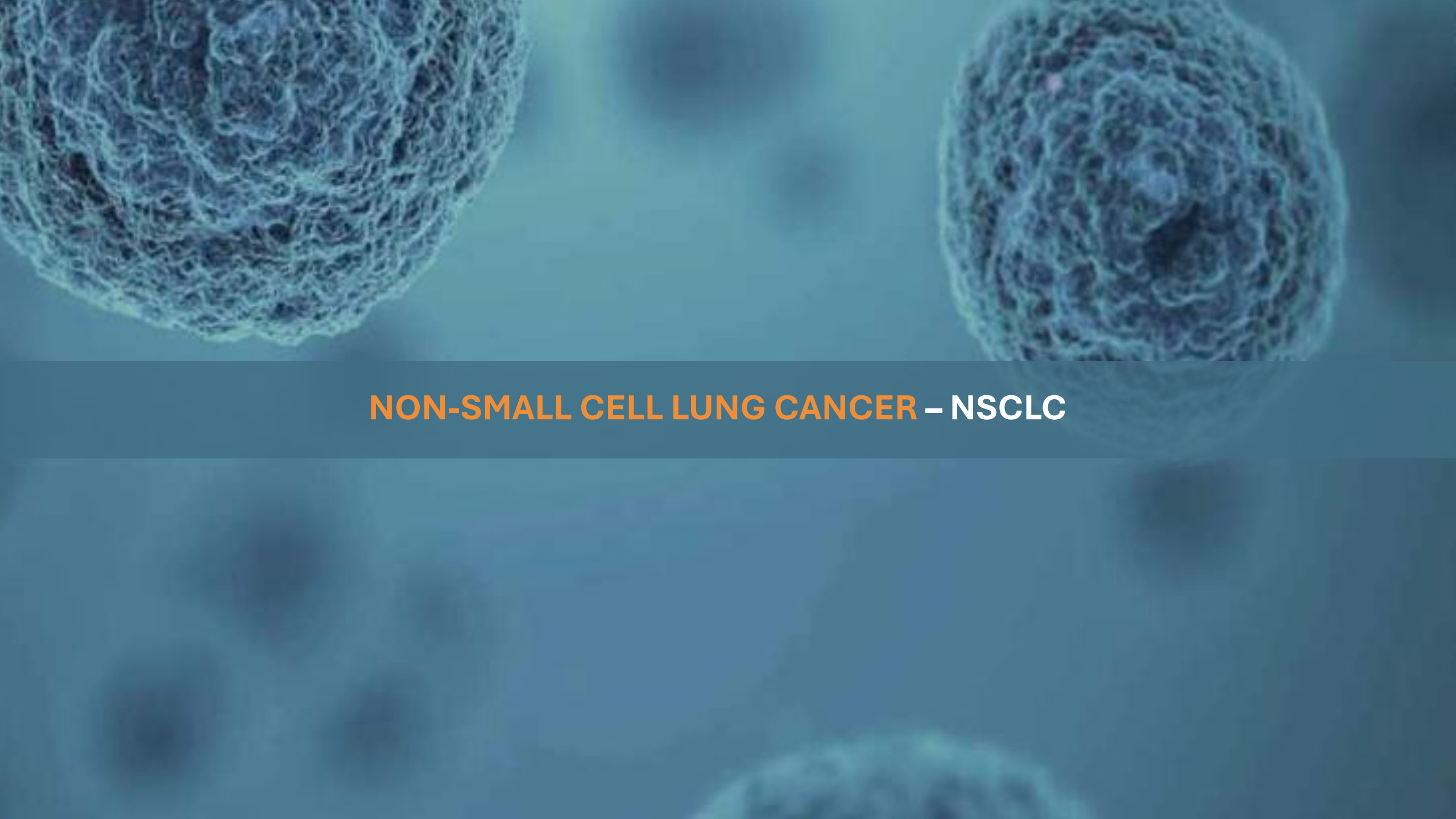




Nadunolimab – A Leading Candidate in the 1L PDAC Race

	Phase	N	mOS (months)	PFS (months)	ORR (%)	6m OS (%)	9m OS (%)	12m OS (%)	24m OS (%)
SOC									
GN (NAPOLI 3[1])	3	387	9.2	5.6	36.2	68.4	-	39.5	.
FOLFIRINOX[2] (AVENGER500)	3	262	11.7	8.0	21	-	-	-	-
NALIRIFOX (NAPOLI 3[1])	3	383	11.1	7.4	41.8	72.4	-	45.6	.
In development									
Nadunolimab + GN[3]	**2a**	**73**	**13.2**	**5.6**	**32**	**78**	**71**	**58**	**28**
Nadunolimab + GN IL1RAP high[3]	**2a**	**29**	**14.2**	**7.4**	**48**	**78**	**74**	**67**	**35**
Daraxonrasib[4]	2a	38	-	-	47	-	-	-	-
Daraxonrasib + GN[4]	2a	31	-	-	55	-	-	-	-
IMM-1-104 + mGN[5]	2a	34	-	-	39	94	86	-	-

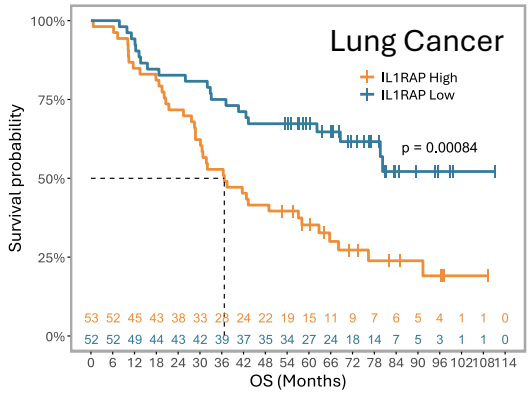
1: Weinberg et al, Lancet, Vol 402 ,Issue 10904, 2023
2: Philip et al, Journal of Clinical Oncology, Vol 42, number 31, 2024
3: van Cutsem et al, Clin Cancer Res, vol 30, 2024
4: Revolution Medicine company presentation 2025: da93b210-ee1b-452e-97a8-7d973c694310
5: Immuneering company presentation 2025: 455a4d85-9f90-41bb-8ede-54a62d6f5e09



NON-SMALL CELL LUNG CANCER – NSCLC

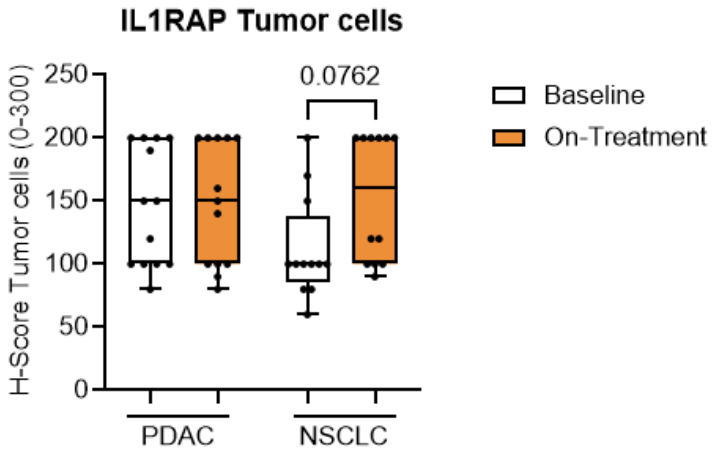
Tumor IL1RAP Expression in NSCLC

Protein Atlas RNA



RNA data from TCGA

CANFOUR protein expression



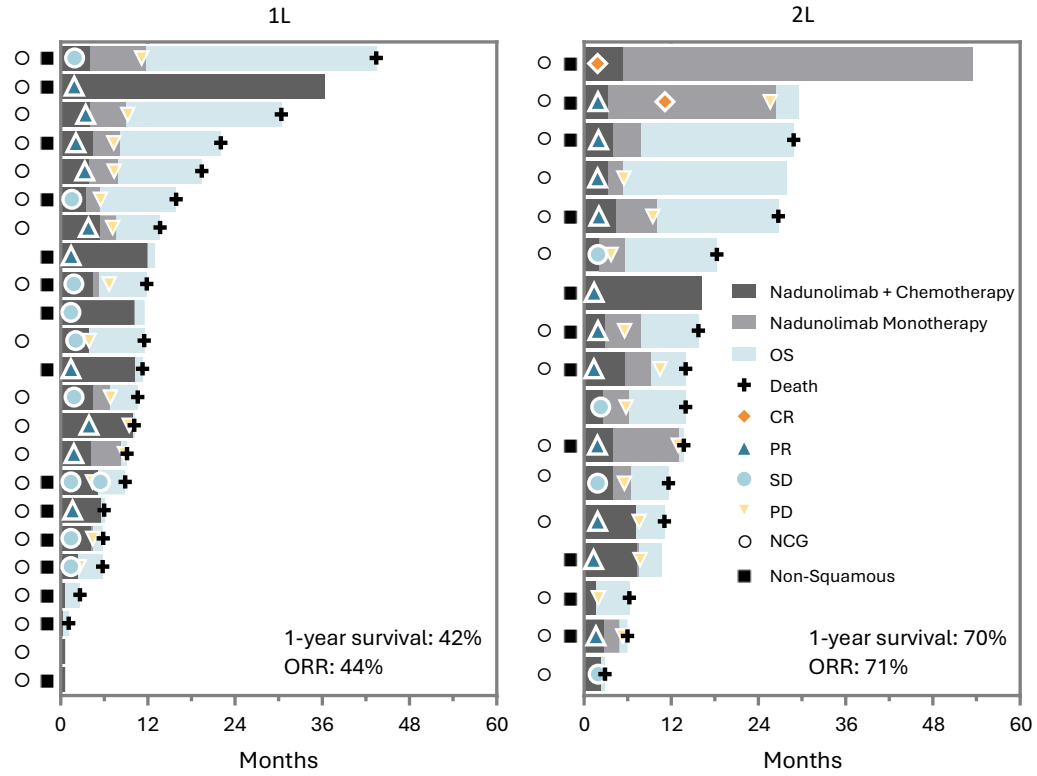
- High IL1RAP RNA expression is a prognostic marker for poor survival in lung cancer, as in PDAC
- CANFOUR NSCLC patients do not show a correlation between high IL1RAP on tumor cells and improved response, which may be due to an upregulation of IL1RAP induced by the platinum-based chemotherapy or IL1RAP expression on supporting cells
- Nadunolimab monotherapy treated patients do not show upregulation of IL1RAP, in line with preclinical data showing that IL1RAP is upregulated in response to platinum containing chemotherapy

IL1RAP EXPRESSION IS UPREGULATED BY TREATMENT WITH PLATINUM CHEMO

Promising Efficacy with nadunolimab plus Platinum Doublets: Longer Survival in Post-Pembrolizumab 2L

Efficacy parameter (95% CI)	Total (n=40)	1L (n=23)	2L (n=17)
OS; median, months	13.7 (11.1-18.3)	11.5 (8.9-19.4)	15.7 (11.1-28.8)
PFS; median, months	7.2 (5.6-9.2)	7.2 (4.4-9.2)	7.6 (5.3-10.4)
1-year survival*	54% (37-69)	42% (21-62)	70% (42-86)
ORR	55% (38-71)	44% (23-66)	71% (44-90)
DoR; median, months	6.4 (4.4-9.9)	5.7 (3.4-9.9)	7.5 (3.7-20.3)

*The proportion of patients with 1-year survival is based on Kaplan-Meier estimation.

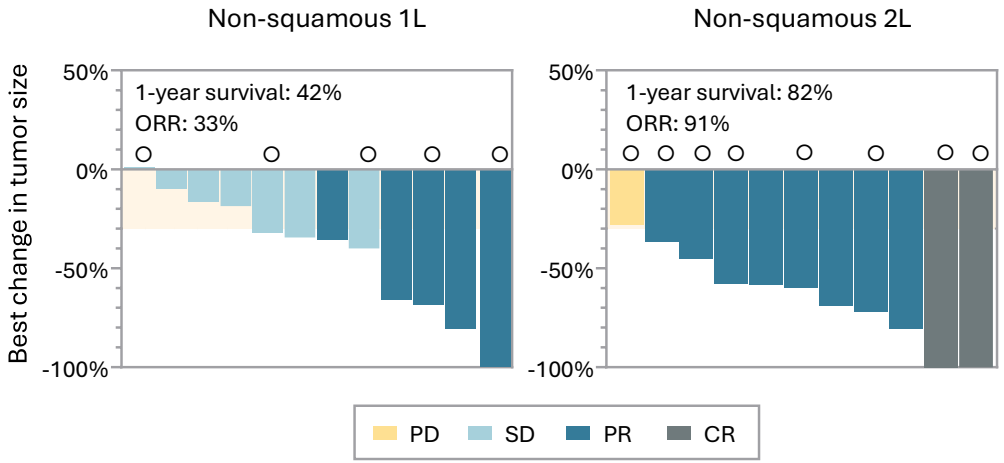


- Platinum was given for 4-6 cycles and then patients continued either on nadunolimab monotherapy or in combination with gemcitabine or pemetrexed.
- At data cut-off 3 patients were still receiving nadunolimab based therapy.
- All 2L patients received pembrolizumab as 1L treatment

Best Efficacy in Non-Squamous NSCLC Patients Receiving nadunolimab plus Platinum doublets Post-Pembrolizumab

	Non-Squamous		Reference**
Efficacy parameter (95% CI)	**1L (n=15)**	**2L (n=11)**	**2L (n=118)**
OS; median, months	11.6 (5.8-22.0)	26.7 (6.2-NE)	OS 13.3 months after last dose of 1L pembro
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
**Rittberg et al Curr Oncol, 2023. Patients are 82% NonSq, and 88% were treated with 2L platinum doublet



BEST RESPONSE WAS SEEN 2L POST-PEMBROLIZUMAB IN NON-SQUAMOUS PATIENTS WITH AN OS OF 26.7 MONTHS AND ORR OF 91%

CR1: Achieved during NGC treatment, after 2 months
CR2: Achieved 8 months post-NCG, during treatment with nadunolimab monotherapy
Reference value Non-Sq 1L: OS 11.3 months Gandhi 2018
***Rittberg et al Curr Oncol, 2023. Patients were 82% NonSq, and 88% were treated with 2L platinum doublet (n=118)*

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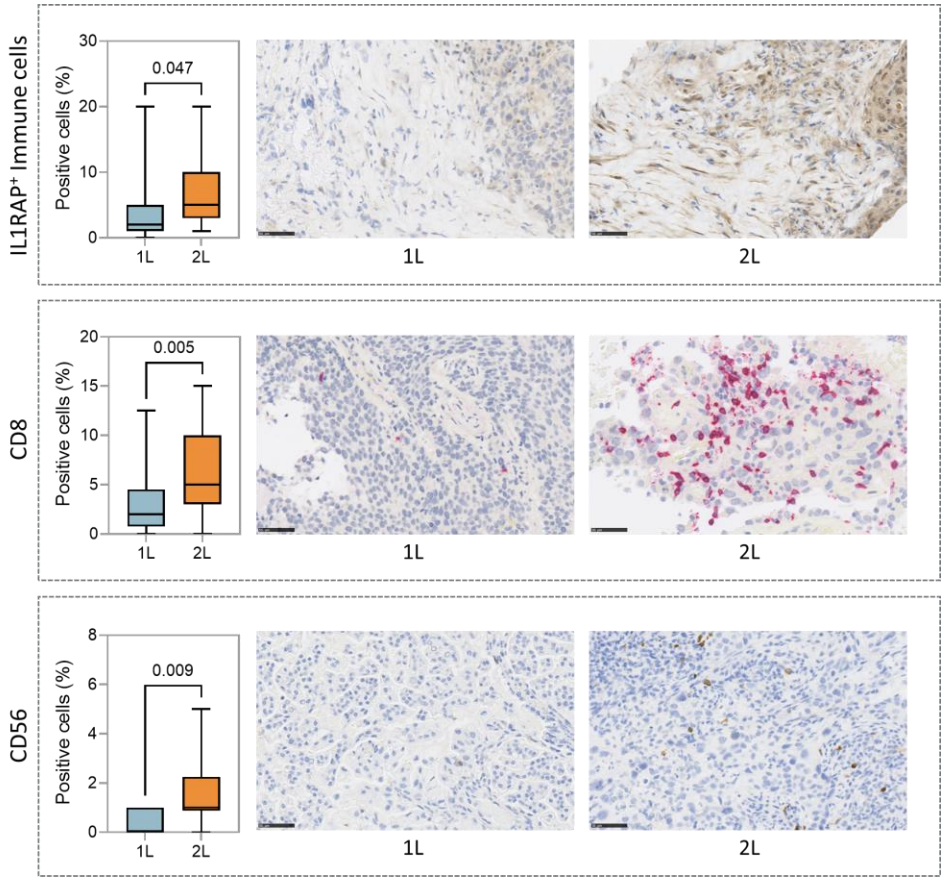
NSCLC Biomarkers: Patients receiving pembrolizumab have Altered TME as compared to Naïve



- Screening biopsies were stained for the presence of IL1RAP$^+$ immune cells, CD8$^+$ T cells, NK cells and CD163+ macrophages
- Patients previously treated with pembrolizumab appear to have altered TME with high infiltration of immune cells
- Serum markers are also altered in 2L patients as compared to naïve patients

A significantly altered tumor microenvironment with high levels of immune cells in the patients that responds best to nadunolimab + platinum doublet treatment

HIGH IMMUNE CELL INFILTRATION IN TME CORRELATES WITH RESPONSE TO NADUNOLIMAB AND PLATINUM DOUBLETS

NSCLC – 2nd Line Non-Squamous

Diagnosed NSCLC Incident Cases*	640 000*
Diagnosed Incident Cases of NSCLC – stage IIIB/IV Non-squamous	300 000 (~47%)
NSCLC – stage IIIB/IV Non-squamous Patients without any AGAs or not tested for biomarkers	190 000 (~60%)
NSCLC - 2L Non-squamous patients irrespective PD-L1 expression status	100 000 (~53%)

*7MM – 2024e (i.e. 8MM) ** Actionable genomic alterations
Source: Global Data, Non-small Cell Lung Cancer (NSCLC): Seven-Market Drug Forecast Update, May 2025

2L treatment for stage IV NSCLC (ASCO)

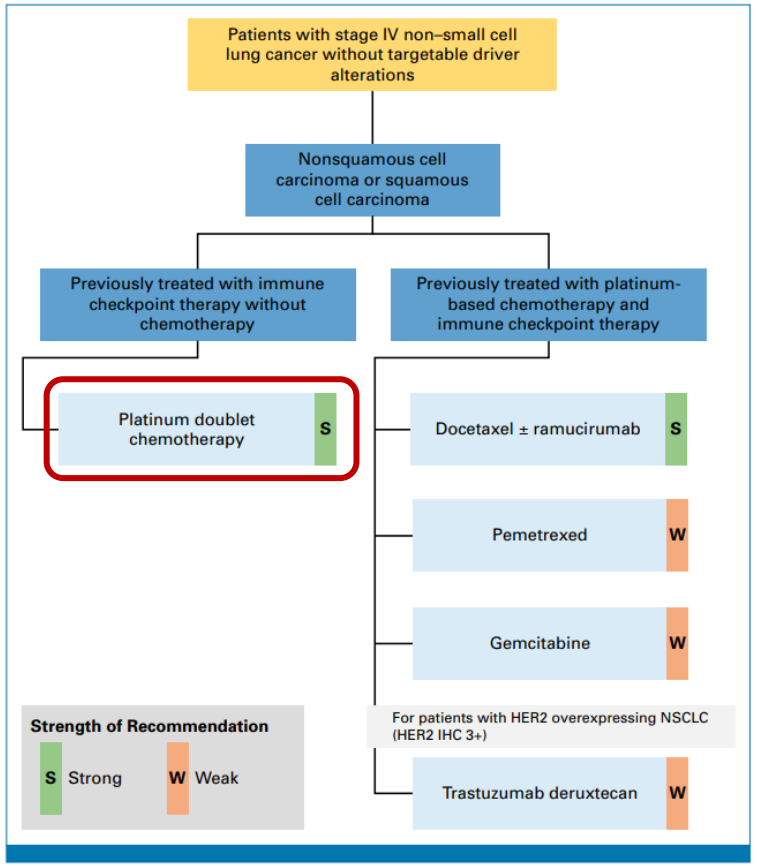


FIG A2. Second-line and subsequent treatment options for patients with stage IV NSCLC without driver alterations. For recommendations with multiple treatment options of the same evidence quality and strength of recommendation, the decision of which agent to offer should be tailored based on discussion of efficacy and toxicity with each patient. HER2, human epidermal growth factor receptor 2; IHC, immunohistochemistry; NSCLC, non–small cell lung cancer; SCC, squamous cell carcinoma.

ASCO Living Guideline, Version 2025.1

TRIPLE-NEGATIVE BREAST CANCER – TNBC

TRIFOUR Preliminary Results in TNBC

- No difference observed between the group treated with gemcitabine/carboplatin (GC) and the chemotherapy-only control group, with regards to the primary endpoint, overall response rate (ORR). Both arms report somewhat higher than historical benchmarks for first- to third-line treated patients (~30%).
- Preliminary median overall survival (mOS) after 39 events for the 99 patients were 26 months in both study groups, substantially longer than expected for this patient population. There were no differences observed in sub-group analyses. Based on these results, Cantargia will discontinue further development in TNBC.
- Safety was in line with previous nadunolimab data, with neutropenia and asthenia being the most common adverse events and no significant safety differences between treatment groups, confirming that nadunolimab can be added to standard chemotherapy without increasing toxicity.
- Since some patients in both groups continue to benefit from the study therapy, treatment and follow-up will continue for ethical reasons, although the overall assessment of the study is unlikely to change.

Tumor Pathology in PDAC vs TNBC Overlaps & Differences

	IL1RAP levels correlate to survival	KRAS driven	IL1RAP on tumor cells	IL1RAP on stromal/ immune cells	CAF/myeloid dependent desmoplastic stroma	IL-1 involved in chemotherapy resistance	Defined responder subgroup	Nadunolimab safety & tolerability
PDAC	√	√	√	√	√	√	√	√
TNBC	X	X	√	√	X	√	X	√

PDAC

- IL1RAP mRNA levels strongly correlate to survival
- Characteristic desmoplastic (fibrotic) stroma where IL1RAP-expressing cells play leading roles (CAFs, myeloid cells) and IL-1 signaling is important
- CANFOUR homogenous population of metastatic PDAC patients receiving first line treatment

TNBC

- IL1RAP mRNA levels are not prognostic
- Different TME to PDAC, heterogeneous with higher infiltration of immune cells
- Heterogeneous patient population with diverse medical history, no single driver mutation

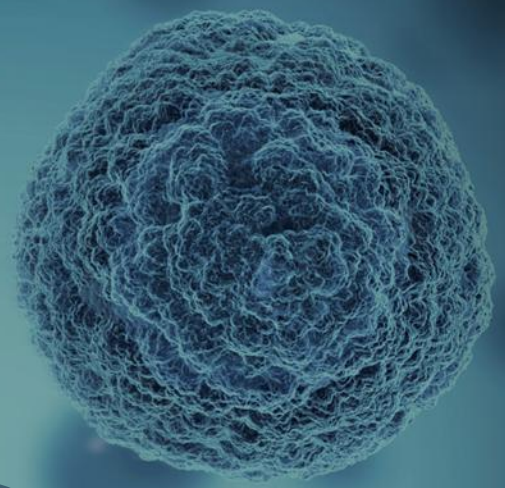
Nadunolimab – Key Takeaways

- Nadunolimab, investigated in more than 300 pts, shows promising safety & efficacy.
- The mechanism of nadunolimab includes ADCC and blocking of both IL-1α and IL-1β signaling through IL1RAP.
- Clinical results strongly support potential unique first-in-class opportunities in PDAC and NSCLC.
- PDAC patients with high IL1RAP level respond best to nadunolimab combination therapy despite having a worse prognosis.
- Development of IL1RAP assay for selection of patients on track for study initiation in 2026.
- Nadunolimab is Pivotal Study Ready and has Fast Track Designation for IL1RAP high expressing patients in PDAC.

TARGETING HIGH IL1RAP PDAC PATIENTS INCREASE PROBABILITY OF SUCCESS

CAN14 and CANXX

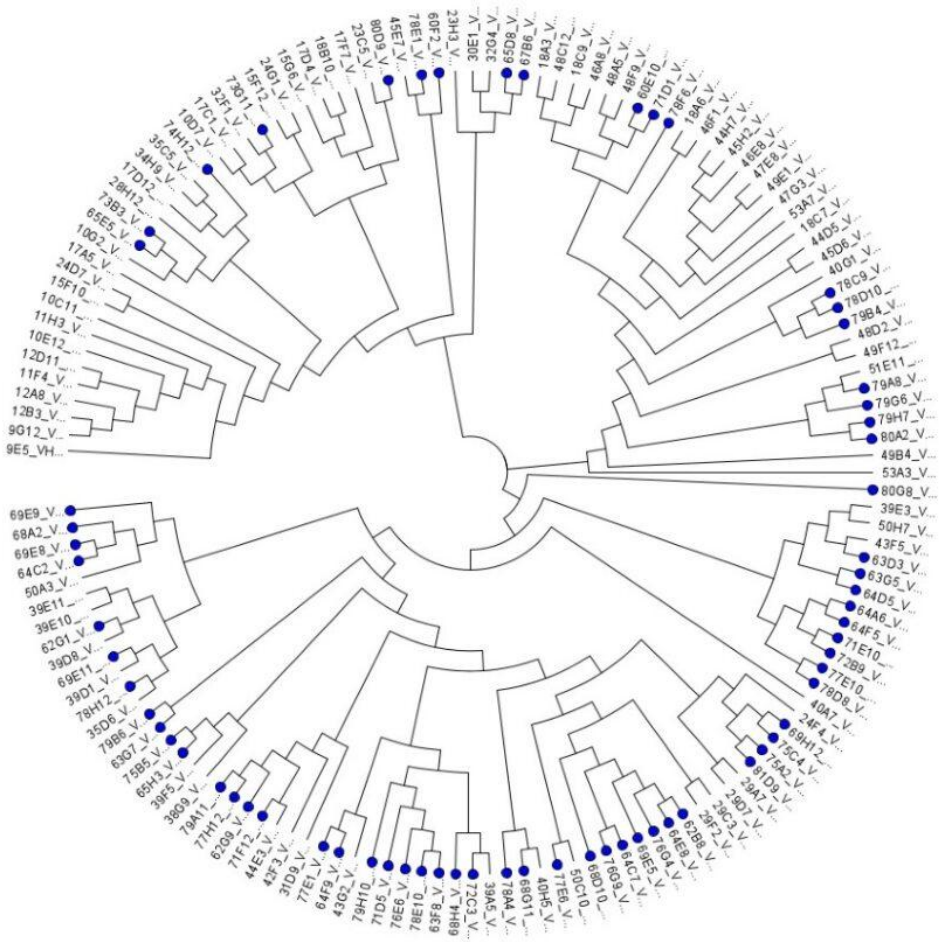
Next generation IL1RAP therapeutics



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 that adds 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**

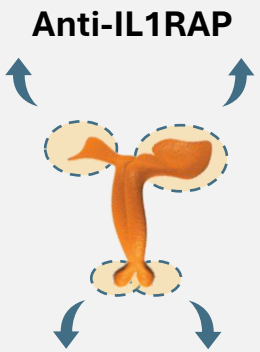


CANXX – A COMPREHENSIVE SOURCE FOR NEW DRUG PROJECTS

Next Generation IL1RAP Therapeutics

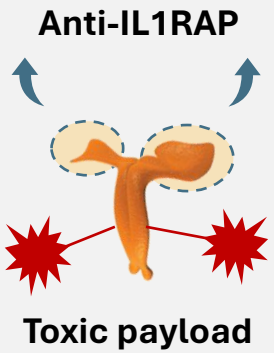
IL1RAP blockade is a potent way to block inflammation in preclinical and translational ex vivo models

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



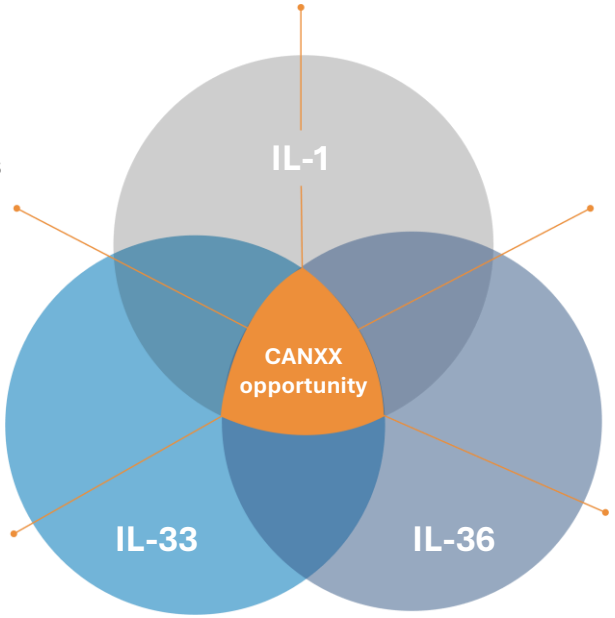
CAN14 and IL1RAP-based Bispecific Antibodies

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

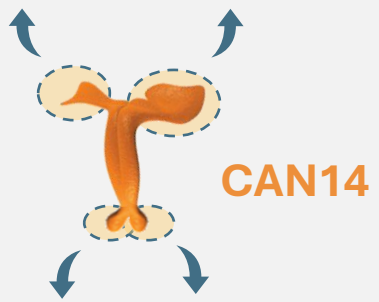


Supported by CHBC/Lumanity Research

Intestine
- Crohn's disease
- Ulcerative colitis

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

Anti-IL1RAP

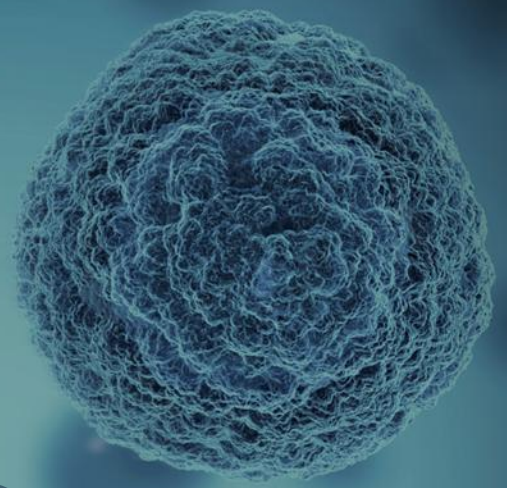


Undisclosed target

- 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

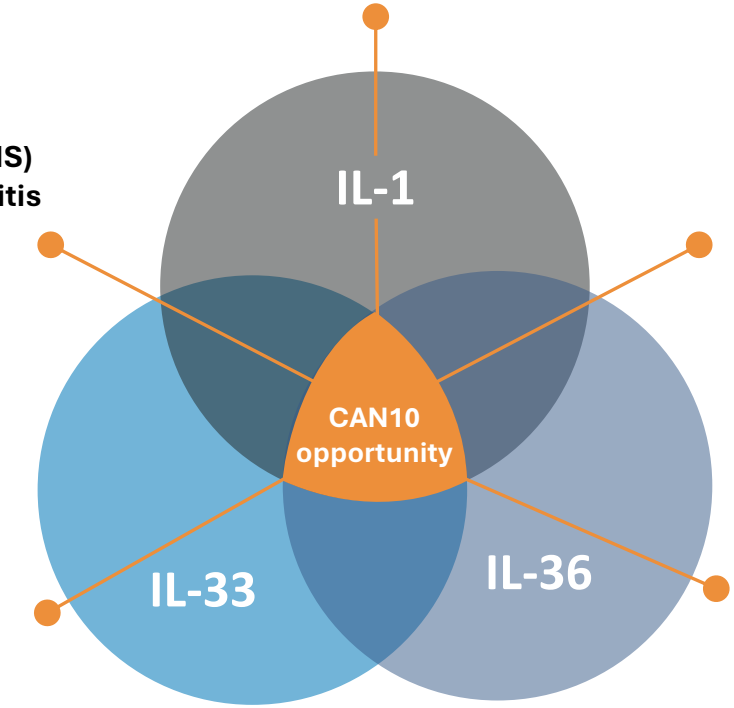
VALIDATION OF IL1RAP AS A TARGET IN INFLAMMATION AND OF CANTARGIA'S ANTIBODY PLATFORM

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

- The IL-1superfamily 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

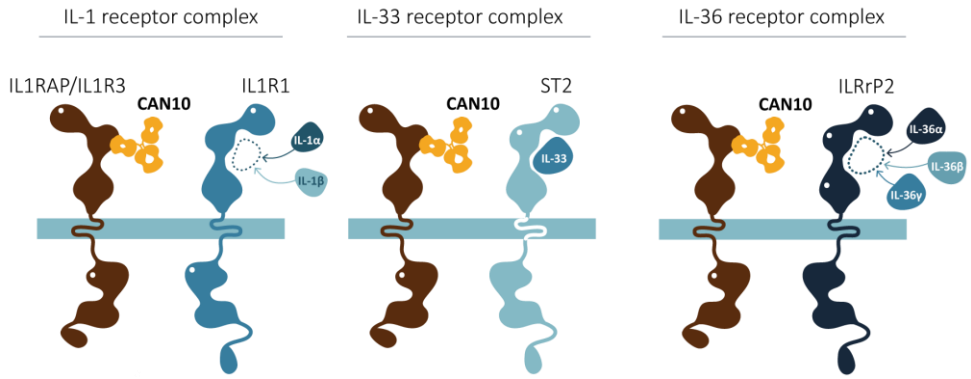
1. Interleukin-1 in the pathogenesis and treatment of inflammatory diseases - Charles A. Dinarello, Blood (2011) 117 (14): 3720–3732.
2. Canakinumab, spesolimab

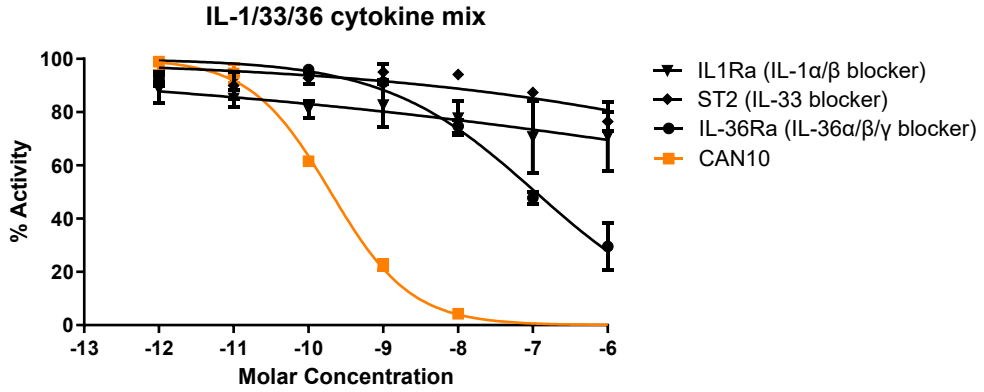
Cantargia

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 First-in-Human study (FIH) – SAD/MAD

SAD (IV)

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

MAD – Healthy (SC)

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

MAD – Psoriasis (SC)

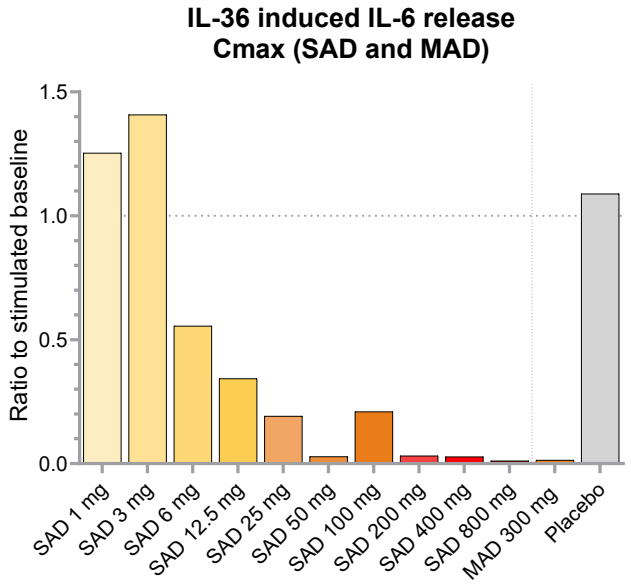
- Mild-Moderate plaque psoriasis
- Enable mechanistic studies

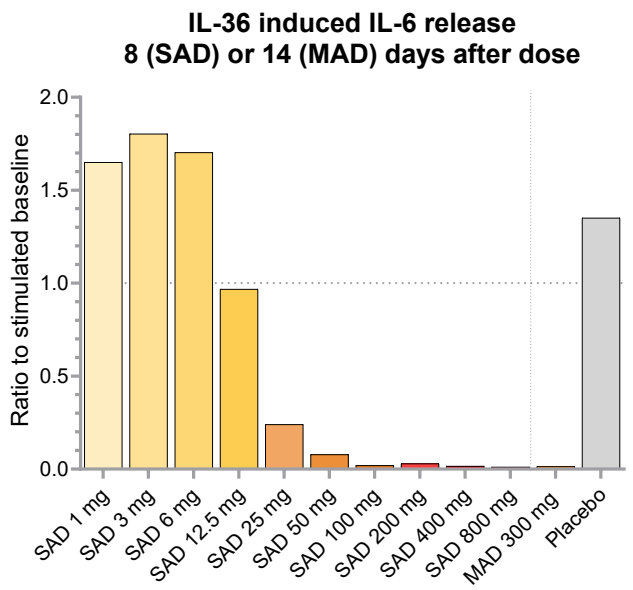
FULL RECEPTOR OCCUPANCY, IL-1 FAMILY CYTOKINE BLOCKADE & NO SAFETY CONCERN DEMONSTRATED

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

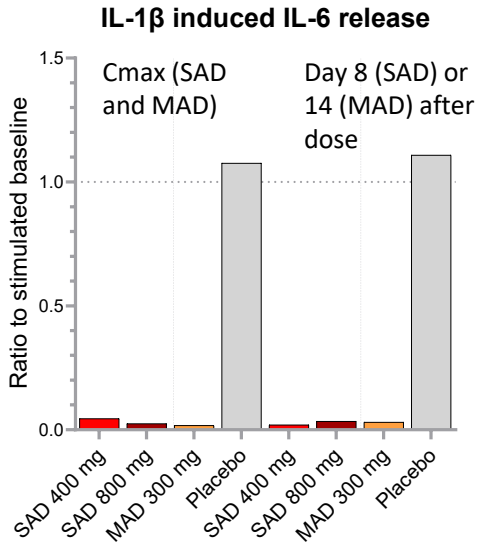
- Inhibition of IL-36 and IL-1β signaling documented at C_{max} (SAD and MAD) and day 8 (SAD) or 14 days after 3rd dose (MAD) → long lasting effect

IL-36 inhibition



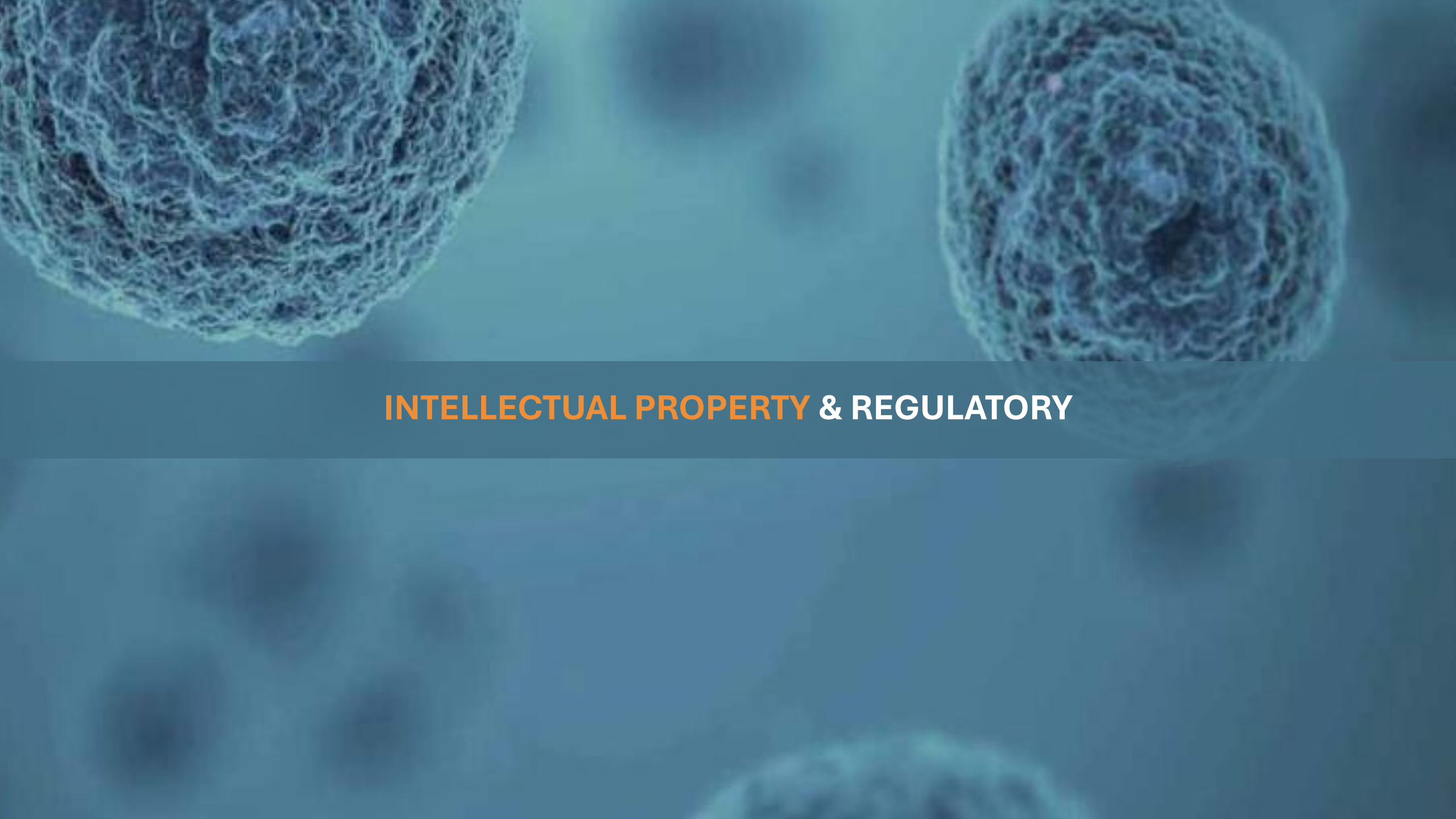


IL-1β inhibition



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 **339m** (September 30, 2025)
- Runway: Into 2028 (with current commitments)

The Cantargia Share

- Listing: **Nasdaq Stockholm** main market
- Ticker: **CANTA.ST**
- Number of shares: **248,611,655** (September 30, 2025)
- Share price: SEK **2.56** (September 30, 2025)
- Market Cap: SEK **635m** (September 30, 2025)
- Average Daily Liquidity: SEK **6.6m** (September 30, Year-to-Date), SEK **15.8m** (Q3 2025)

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, September 30

#	Owner	Number of Shares	%
1	Fjärde AP-fonden	24 800 000	10%
2	Första AP-fonden	16 493 130	7%
3	Avanza Pension	15 625 939	6%
4	Henrick Schill	4 325 663	2%
5	The Invus Group	3 752 923	2%
6	Brushamn Invest AB	3 391 740	1%
7	Tibia Konsult AB	2 806 052	1%
8	Nordnet Pensionsförsäkring	2 431 647	1%
9	Pia Althin	1 864 824	1%
10	Rafi Barsum	1 803 147	1%
	Other	171 316 590	69%
	Total	**248 611 655**	**100%**

A Global Leader in IL1RAP Antibody Development

Potential to become best-in-class in 1L PDAC: Lead Candidate nadunolimab ready to start pivotal trials on the back of outstanding efficacy and safety POC results, especially in high IL1RAP-expressing tumors; FDA Fast Track and Orphan Drug designations supporting accelerated path

Clear Biomarker Strategy: IL1RAP-based Companion Diagnostic Test in development to identify high-expression PDAC patients, enabling targeted treatment and patient stratification

Key External Validation: 2025 transformational deal with Otsuka for CAN10 in immunology indications, validating IL1RAP biology and Cantargia's antibody platform : $613m total deal value of which $33m upfront payment

Unique Next-Gen IL1RAP Platform: proprietary modular antibody platform enabling precision IL1RAP inhibition, a well- defined mechanism of action with potential across oncology and immunology, and expansion into next-gen therapeutics (bispecifics and ADCs)

Several Near-Term Milestones: i) Nadunolimab FDA pivotal trial initiation in Mid-2026 ii) IL1RAP assay clearance, FDA mid 2026 and EMA 2H26 iii) CAN14 Candidate Selection year-end 2026 iv) Nadunolimab interim data with Accelerated Approval window in 2028

Thank you

Contact details:

info@cantargia.com