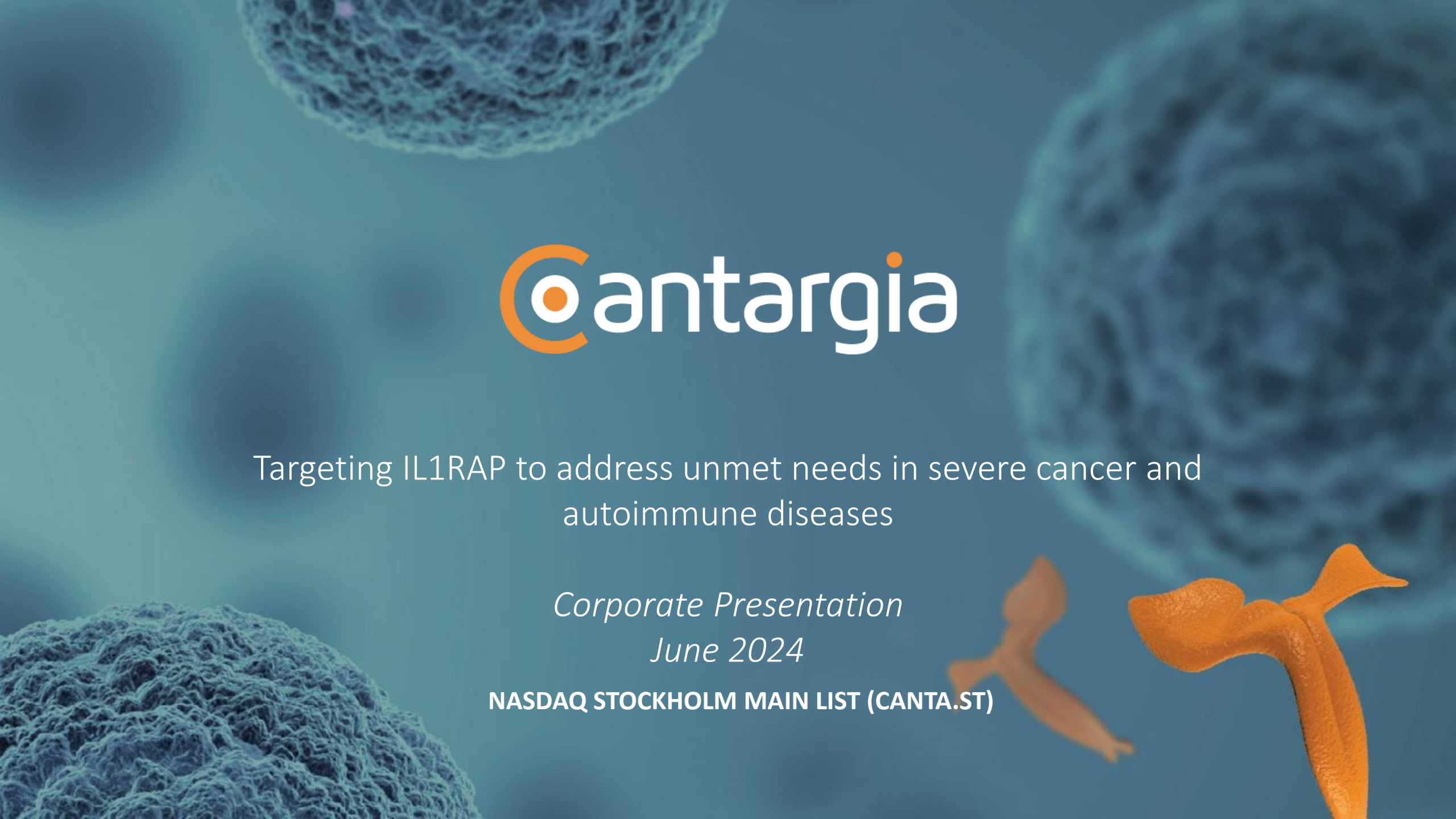
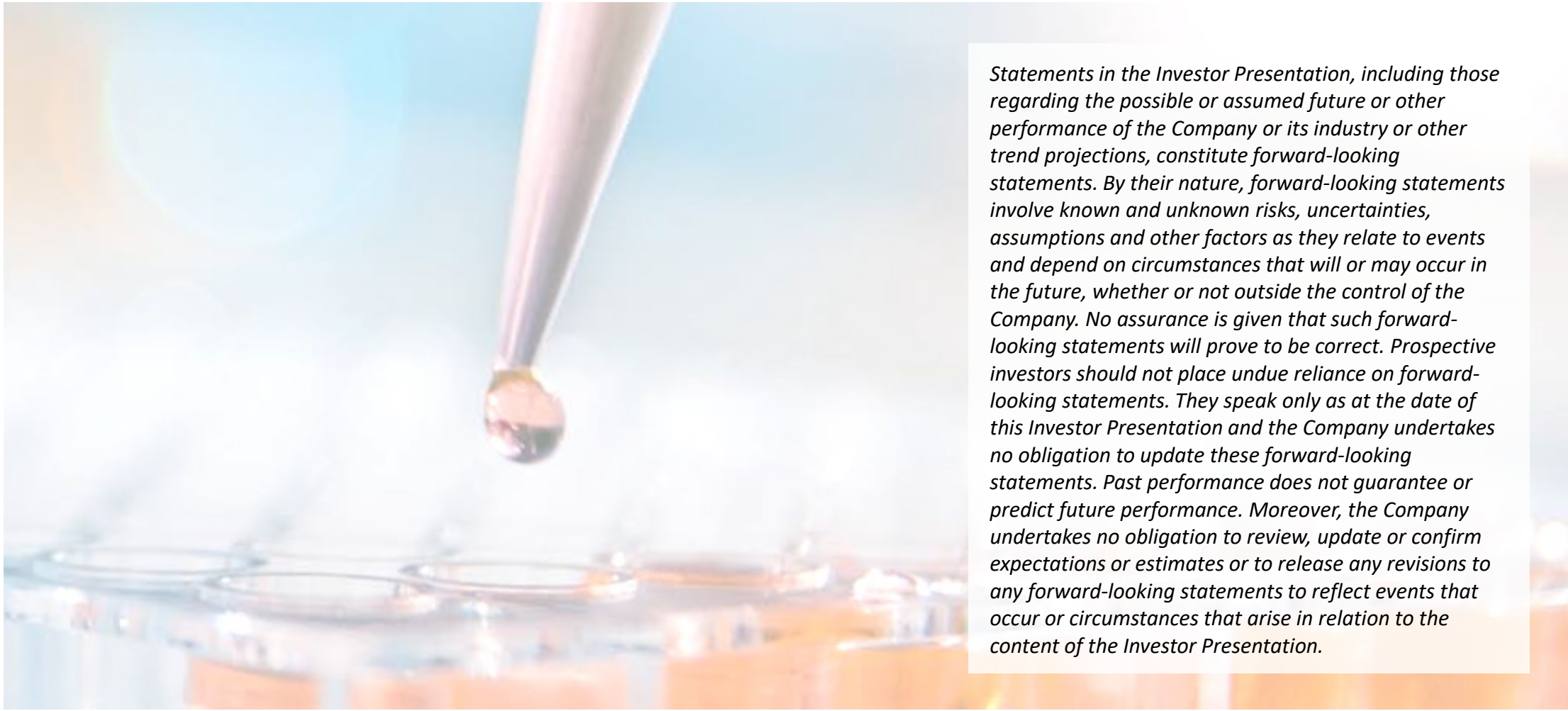


Targeting IL1RAP to address unmet needs in severe cancer and autoimmune diseases

Corporate Presentation
June 2024

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 – Investment highlights

NOVEL IL1RAP ANTIBODIES, POTENTIAL TO TREAT CANCER & INFLAMMATORY DISEASE

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

NADUNOLIMAB: CLEAR ACTIVITY SIGNALS IN CANCER THERAPY WITH UPCOMING CATALYSTS

- Strong clinical interim results in PDAC and NSCLC, and promising initial results in TNBC; >250 patients treated
- Randomized Phase II trial ongoing in TNBC (initial data late 2024); Phase IIb trial in preparation in PDAC (top-line data 2025)

CAN10: OPPORTUNITY IN AUTOIMMUNITY/INFLAMMATION

- Pronounced activity in models of systemic sclerosis, myocarditis, psoriasis, atherosclerosis and inflammation
- Phase I clinical trial ongoing, initial results show good safety and receptor occupancy. New data Q2 2024

CORPORATE STRENGTH DRIVING INNOVATION

- Solid cash position with runway into 2025 (143MSEK (14 MUSD) cash & equivalents at Q1 2024)
- Robust patent portfolio: IL1RAP antibody target in oncology (2032), nadunolimab (2035) and CAN10 (2041)

Current pipeline

Project	Disease	Type of treatment	Discovery phase	Preclinical phase	Clinical phase I	Clinical phase II	Clinical phase III
Nadunolimab	PDAC	1st line				Gemcitabine/nab-paclitaxel	
	TNBC	1st/2nd line				Carboplatin/gemcitabine	
	NSCLC/ non-squamous NSCLC	1st/2nd line				Platinum doublets	
CAN10	Myocarditis, Systemic sclerosis						
CANxx	New opportunities within IL1RAP platform						

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

CAN10 – OPPORTUNITY IN AUTOIMMUNE/INFLAMMATORY DISEASE

CAN10 - Targeting IL-1 family in inflammation

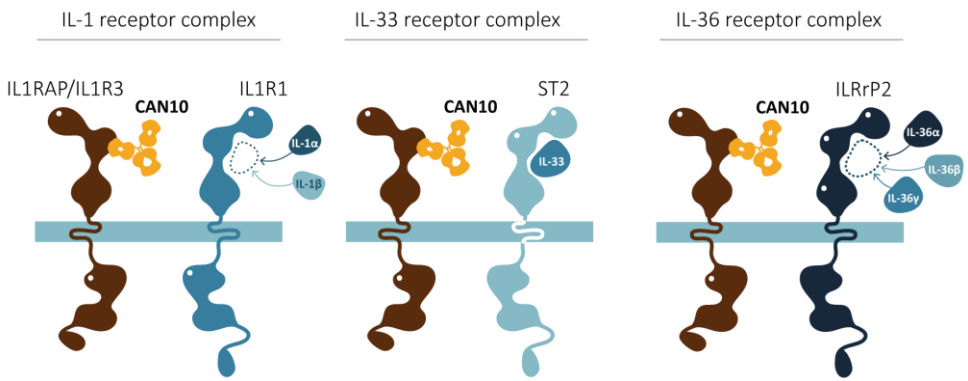
- **Evidence of IL-1 family cytokines (IL-1, IL-33, IL-36) driving inflammatory diseases**
  - These cytokines are commonly upregulated and operate together in several diseases

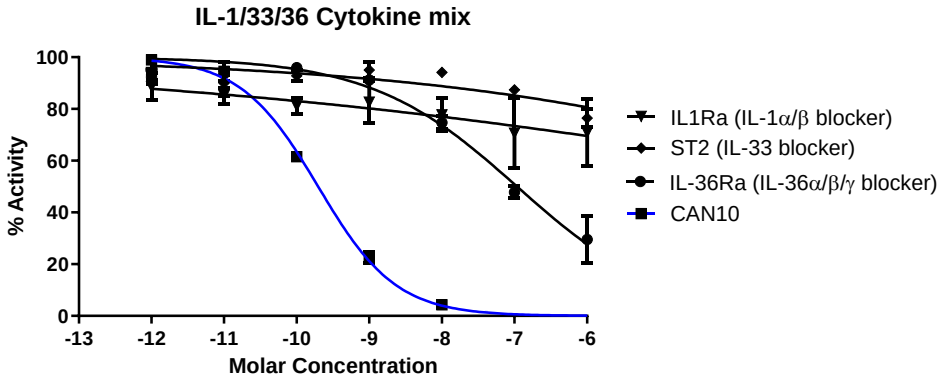
- **Blockade of individual IL-1 family members insufficient**
  - IL-1β and IL-36 targeting drugs only approved in rare diseases with strong elements of dysregulation of the respective cytokines
  - In larger and more diverse diseases, where IL-1 family pathways overlap, signs of clinical benefit reported for therapies targeting individual IL-1 members have been observed, but **not translated into strong clinical efficacy**

- **CAN10 provides a unique opportunity to block IL-1 family signaling**
  - Binding to crucial epitope on common accessory protein (IL1RAP)
  - Solid biological evidence underscores CAN10's potential in several dermatological, fibrotic and cardiovascular diseases

CAN10 developed to block IL-1 family with precision

- **CAN10 prevents signaling from IL1α/β, IL-33 and IL36α/β/γ**
  - CAN10 binds IL1RAP with pM affinity and prevents IL1RAP interaction with the IL-1, IL-33 and IL-36 receptors
- **CAN10 has shown robust efficacy in preclinical models of several diseases**
  - Potent effects in several hard-to-treat models, blocks inflammation and fibrosis **where IL-1α/β or IL-1β blockade only does not**
- **CAN10 is undergoing phase 1 development**
  - No safety issues, including at doses where high level receptor occupancy have been reached
  - SAD portion includes IV administration in healthy volunteers
  - MAD performed with SC administration in psoriasis patients to enable proof-of-mechanism

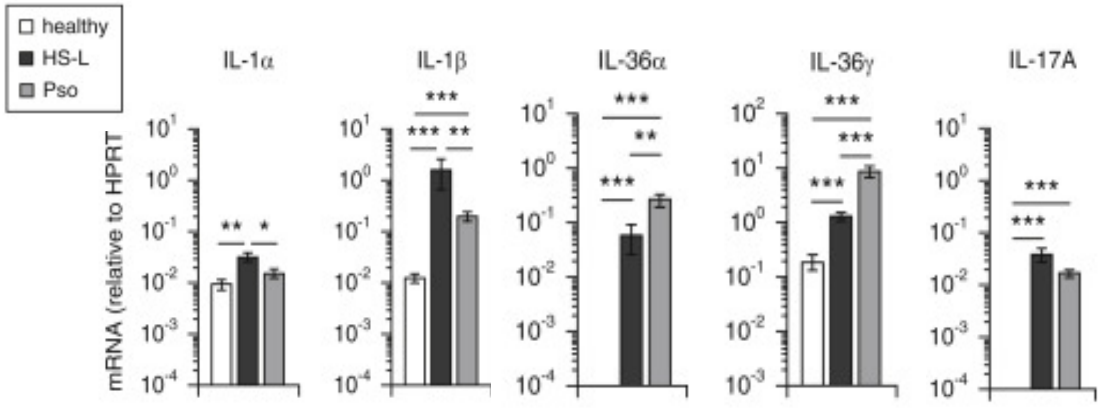




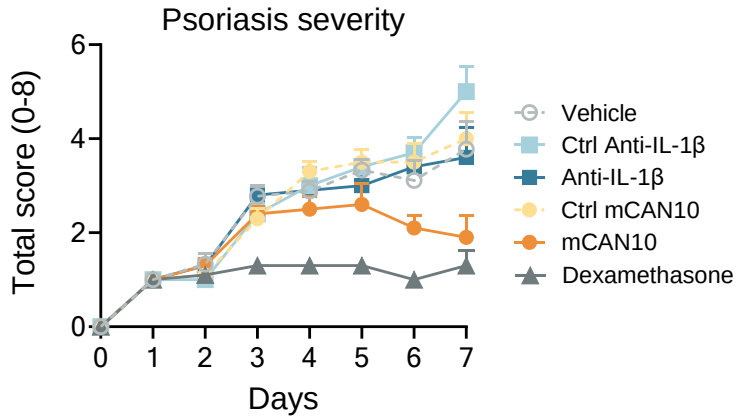
CAN10 first-in-human study ongoing

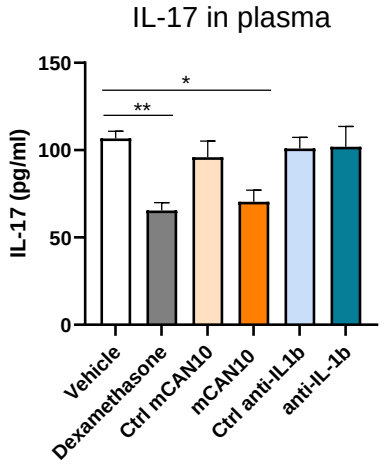
- **IV administration in healthy volunteers (SAD)**
  - Ongoing, receptor occupancy documented
  - No safety signals
- **SC administration in subjects with mild to moderate plaque psoriasis (MAD)**
  - Strong rational for IL1RAP blockade in psoriasis (blocks skin inflammation and IL-17 where anti-IL1β does not)
  - MAD planned to start Q3 2024
  - Psoriasis chosen as phase 1 indication to enable mechanistic studies, no plans to develop in phase 2
- **Building value by including additional PD analyses**
  - Receptor occupancy
  - Ex vivo inhibition assay
  - Psoriasis severity scoring
  - Skin biopsy and skin tape strips
- **Preparations for phase 2 clinical trials ongoing**

Rational for psoriasis in MAD



Witte-Händel et al., JID 2018

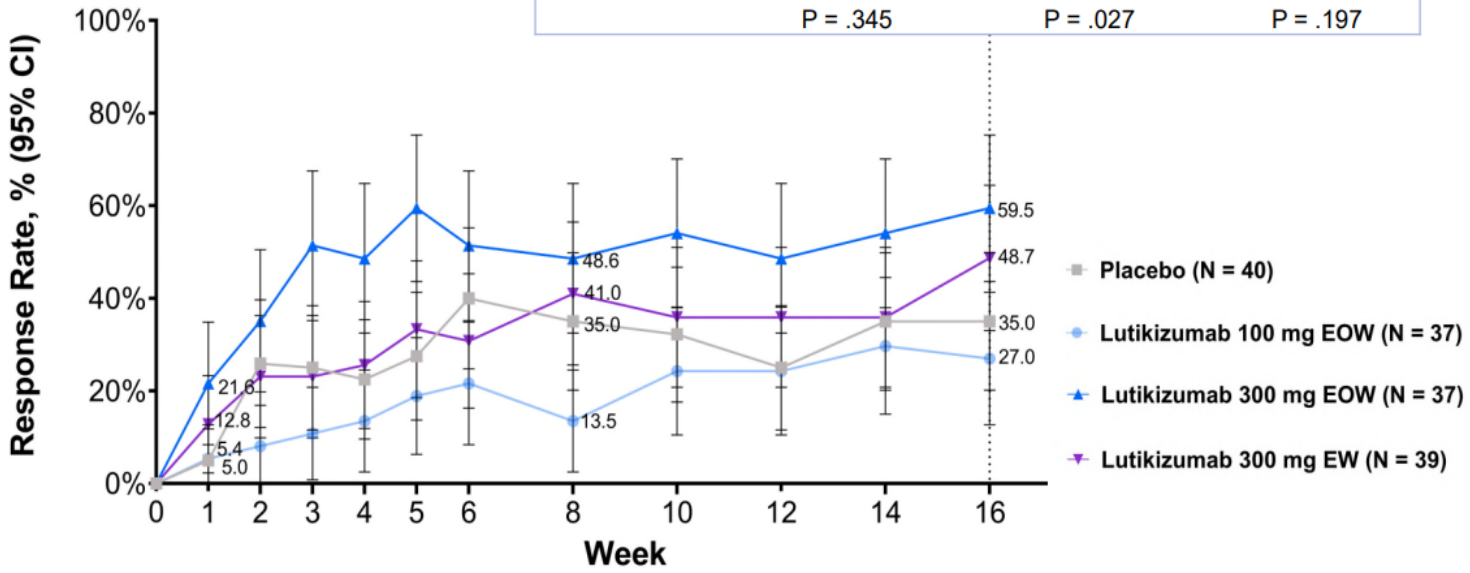




External validation of IL-1 pathway - lutikizumab in HS

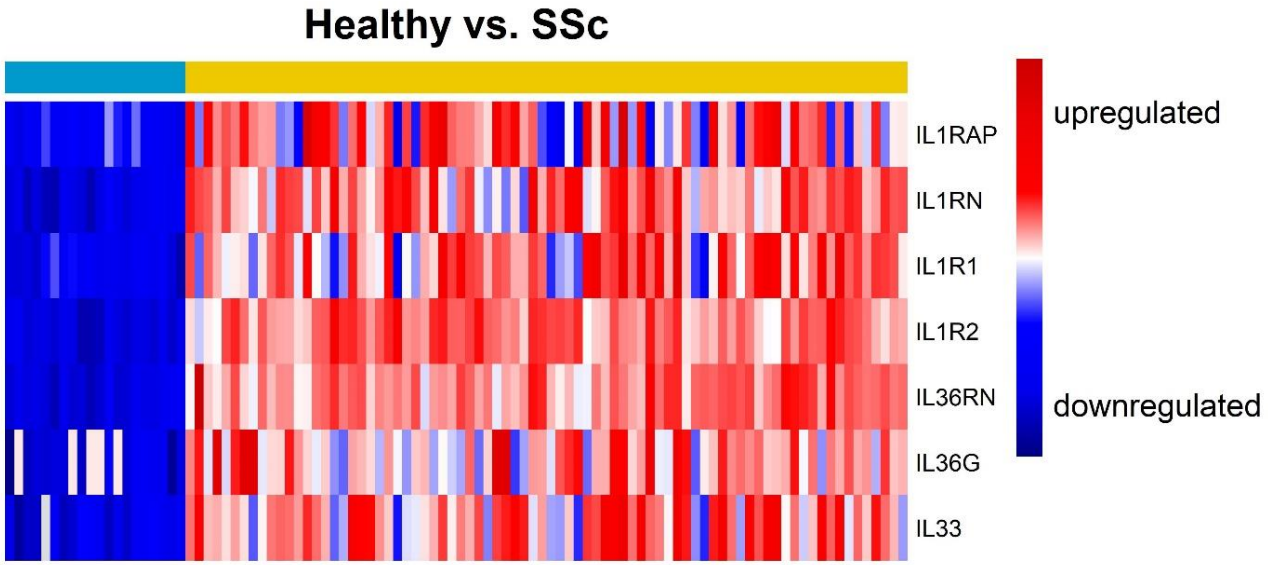
- Lutikizumab is a dual variable domain antibody against IL-1α and IL-β
- Patients treated with lutikizumab experienced higher response rates in the primary endpoint of HiSCR 50 and the secondary endpoint of skin pain NRS30 at week 16 than those treated with placebo
- Patients treated with lutikizumab experienced higher response rates in HiSCR 75 and greater improvement in draining fistula count at week 16 than those treated with placebo
- Lutikizumab entering phase III

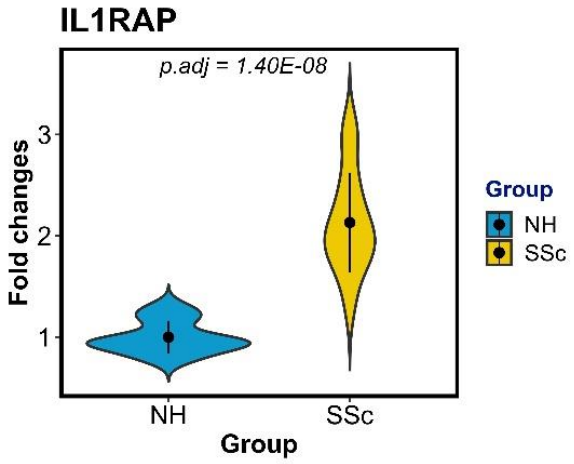
Response (%)	Response (%); Treatment Difference vs. Placebo (%)		
Placebo	Luti 100 mg EOW	Luti 300 mg EOW	Luti 300 mg EW
35.0	27.0 Δ -9.7 P = .345	59.5 Δ 24.1 P = .027	48.7 Δ 13.8 P = .197



IL1RAP and the IL-1/33/36 pathways are upregulated in SSc patient skin

2 publicly available human SSc cohorts show differential expression of IL1RAP and associated genes in SSc skin





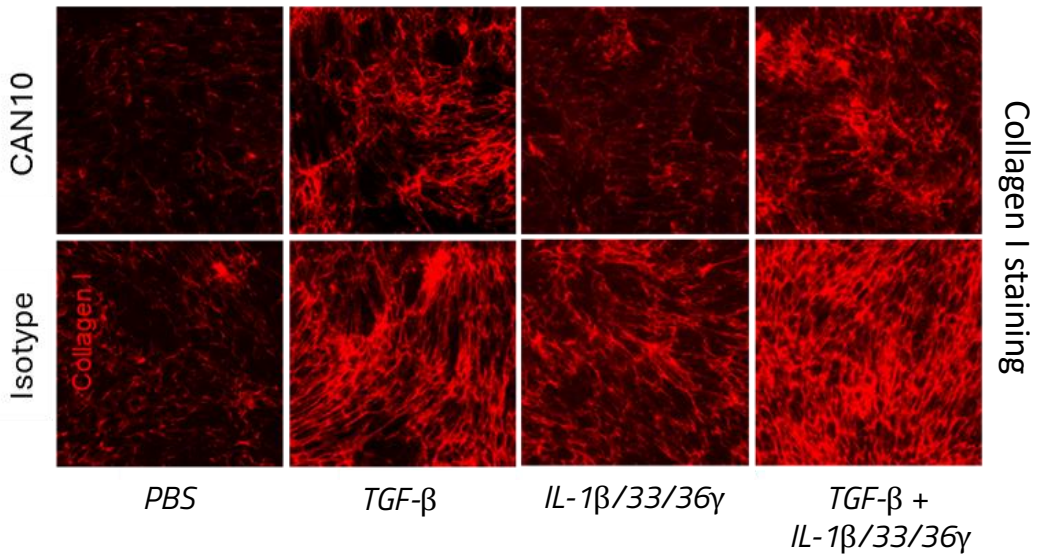
14 SSc vs. 11 healthy

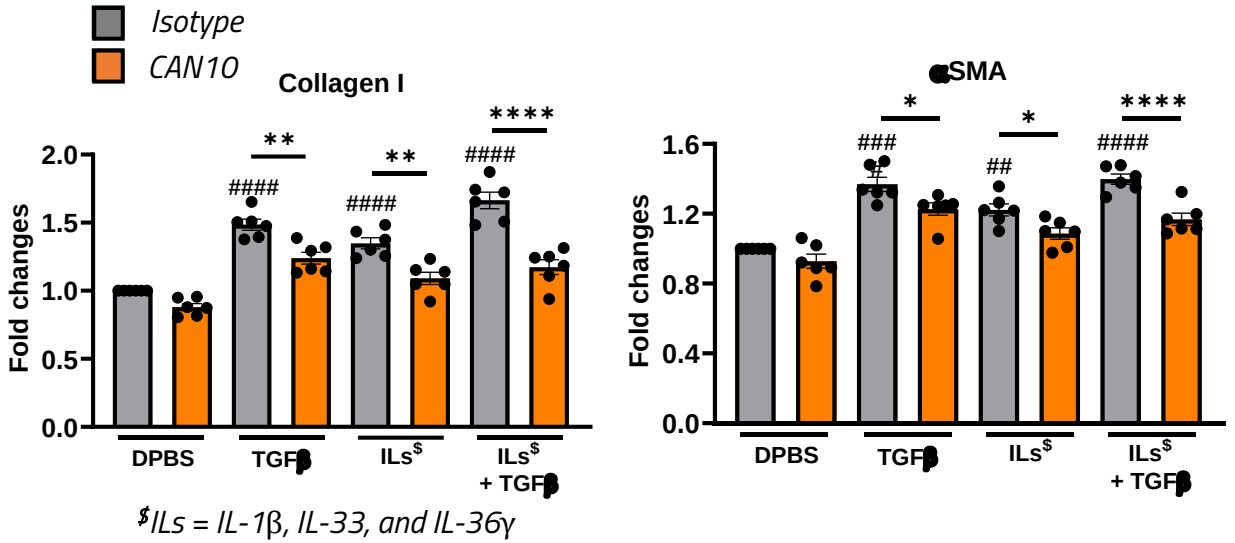
Agilent 2-channel Microarray

Mahoney et al. 2015 GSE59787
Skaug et al. Ann Rheum Dis 2020. GSE130955

 In collaboration with Jörg Distler, Heinrich-Heine-Universität, Düsseldorf, Germany

IL-1, IL-33 and IL-36 directly promotes fibrosis in SSc fibroblasts which can be counteracted by CAN10



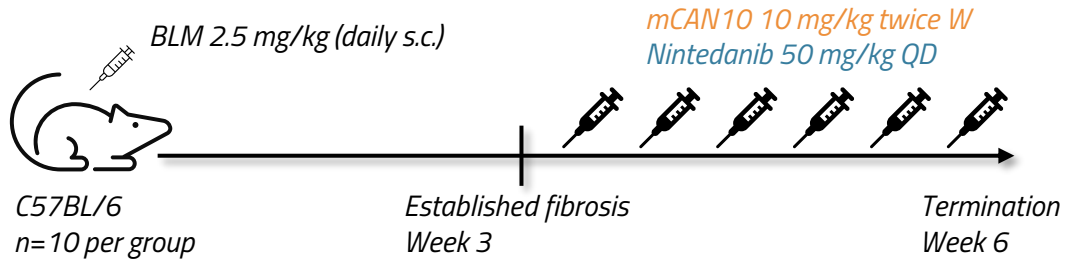


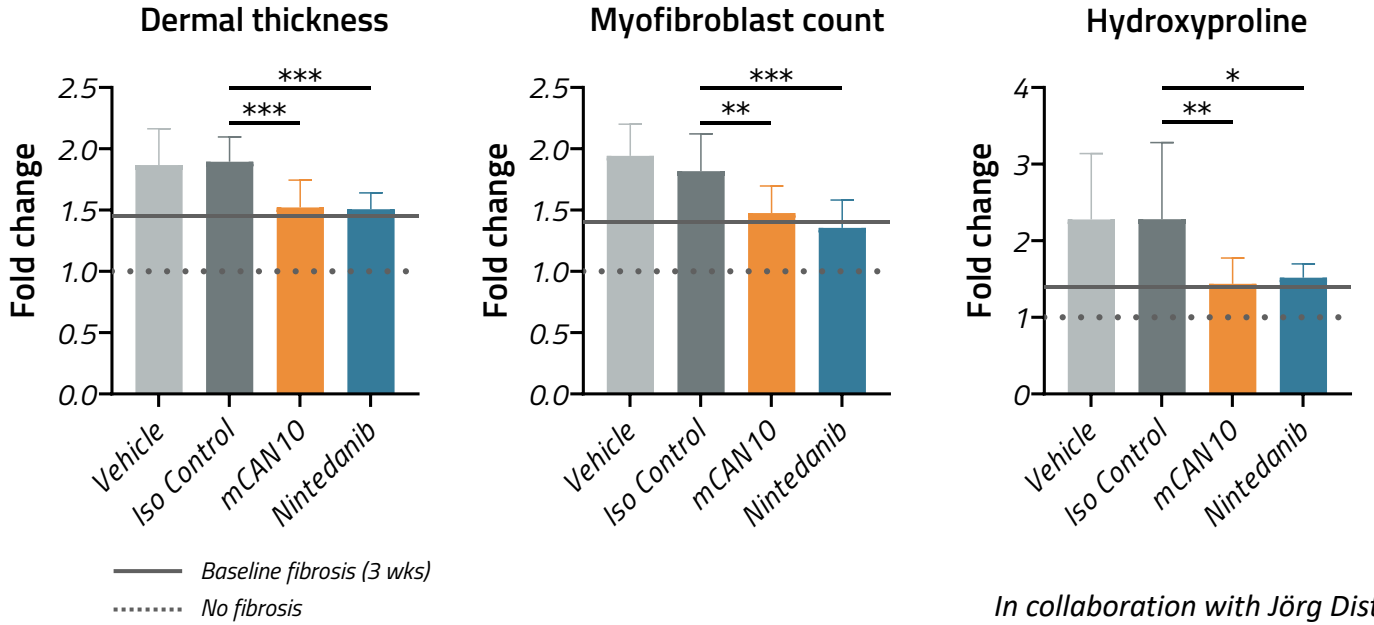
Fibroblasts isolated from SSc patients stimulated with TGFβ or a combination of IL-1β, IL-33, and IL-36γ (abbreviated as ILs) with or without TGFβ in vitro. ILs induced deposition of type I collagen and upregulated the protein levels of αSMA, which could be blocked by CAN10.

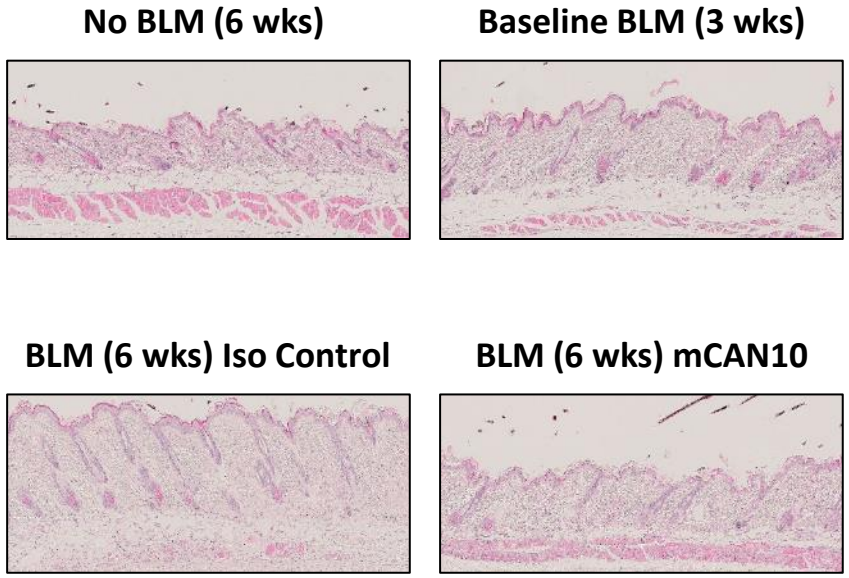
 In collaboration with Jörg Distler, Heinrich-Heine-Universität, Düsseldorf, Germany

Systemic sclerosis: mCAN10 inhibits bleomycin-induced skin fibrosis

Bleomycin (BLM) model

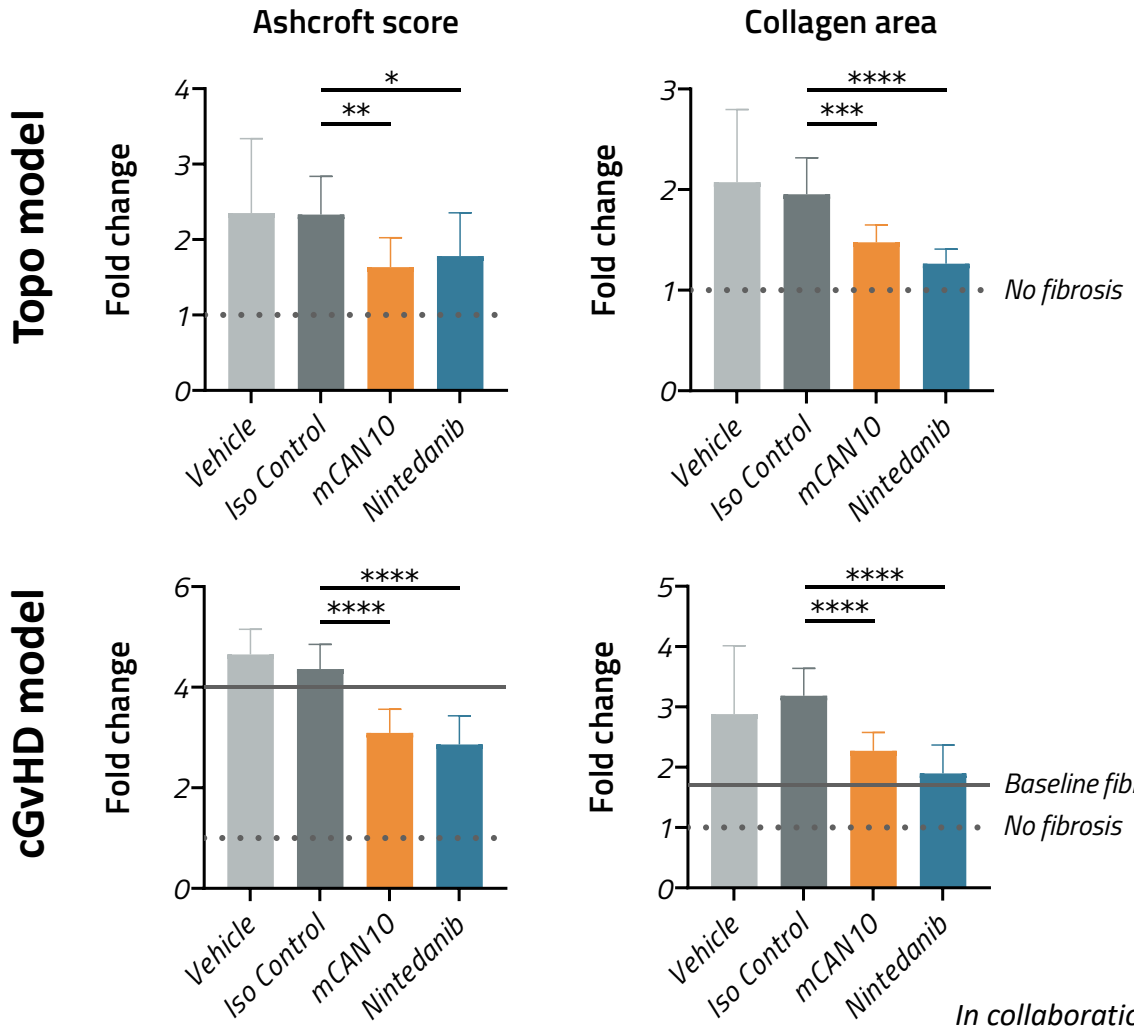


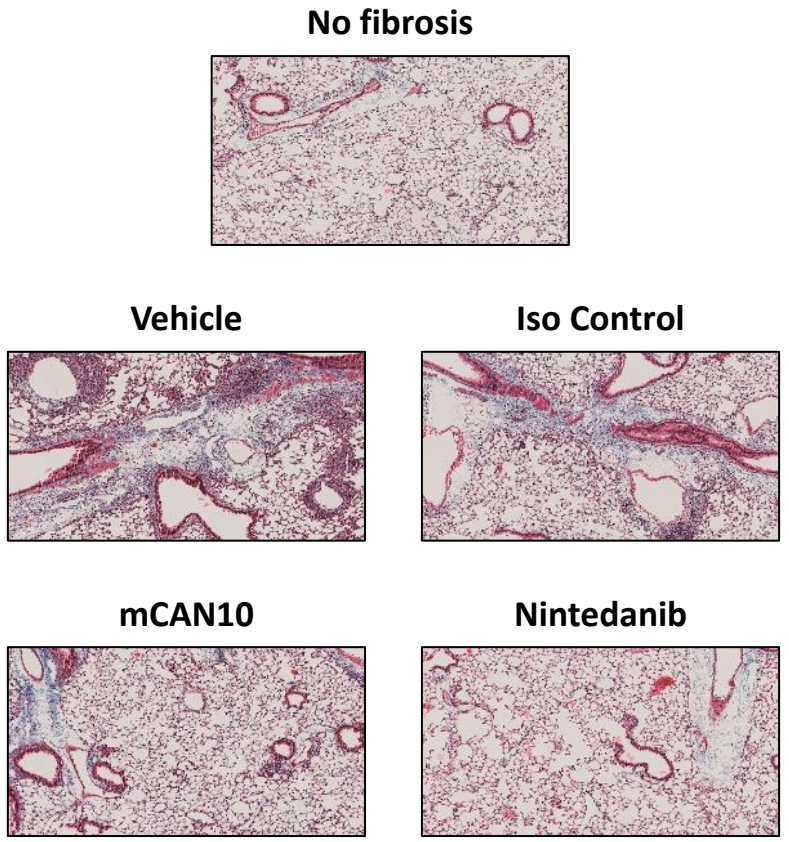




In collaboration with Jörg Distler, Heinrich-Heine-Universität, Düsseldorf, Germany

Systemic sclerosis: Therapeutic mCAN10 treatment reduces lung fibrosis in the Topo and cGvHD models

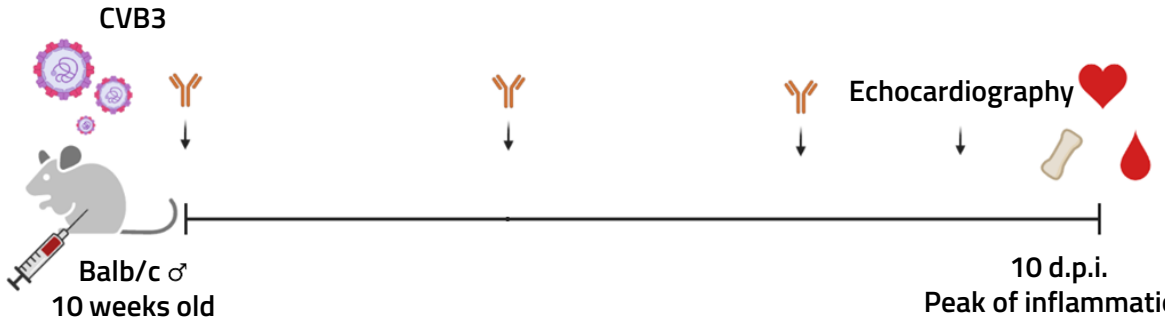


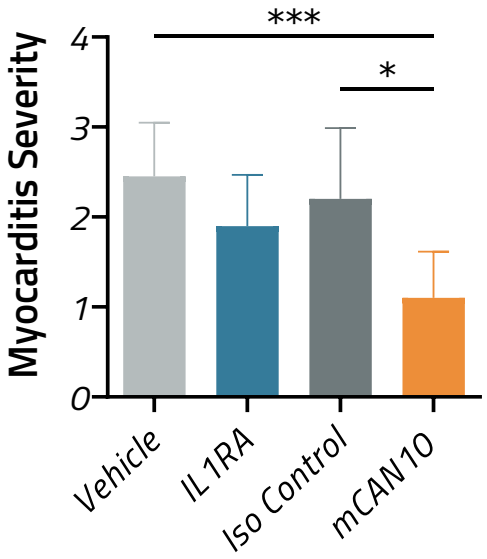


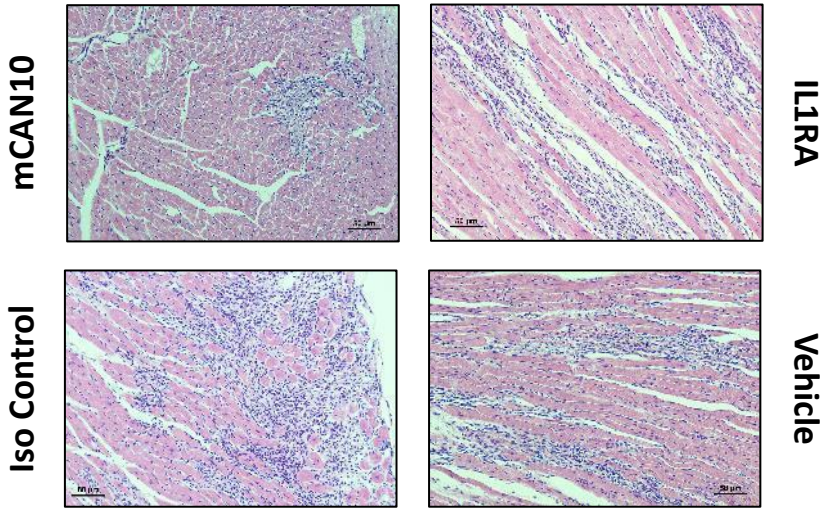
In collaboration with Jörg Distler, Heinrich-Heine-Universität, Düsseldorf, Germany

Viral myocarditis: mCAN10 reduces disease severity

CVB3 myocarditis experimental design







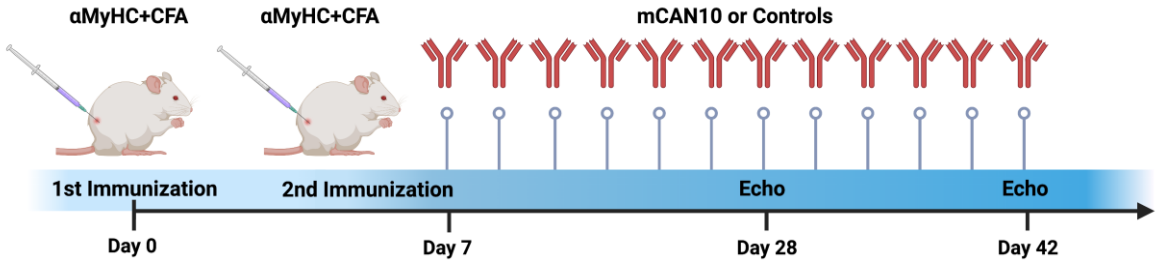
→ mCAN10 reduced disease severity, based on histological scoring of heart sections, and preserved heart function

→ mCAN10 also reduced inflammatory leukocyte populations in the heart tissue

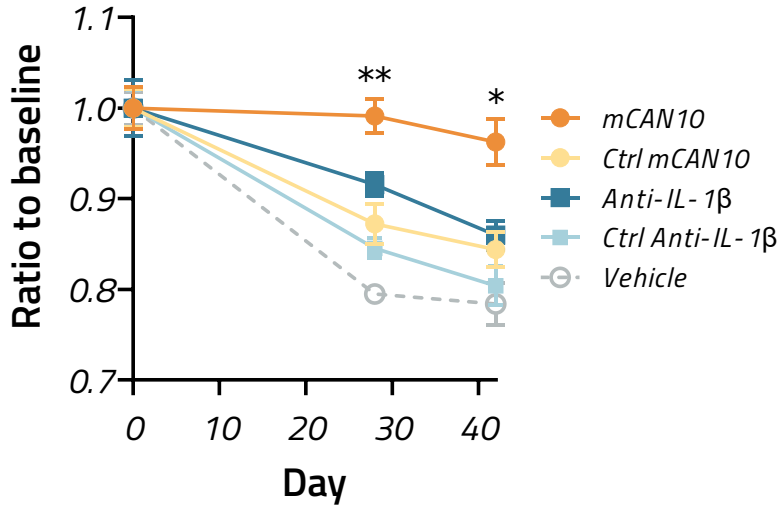
CVB3 – Coxsackievirus B3; IL1RA – IL-1 Receptor Antagonist (blocks IL-1α/IL-β signaling)
n=10 per group

In collaboration with Daniela Cihakova, John's Hopkins University, Baltimore, US

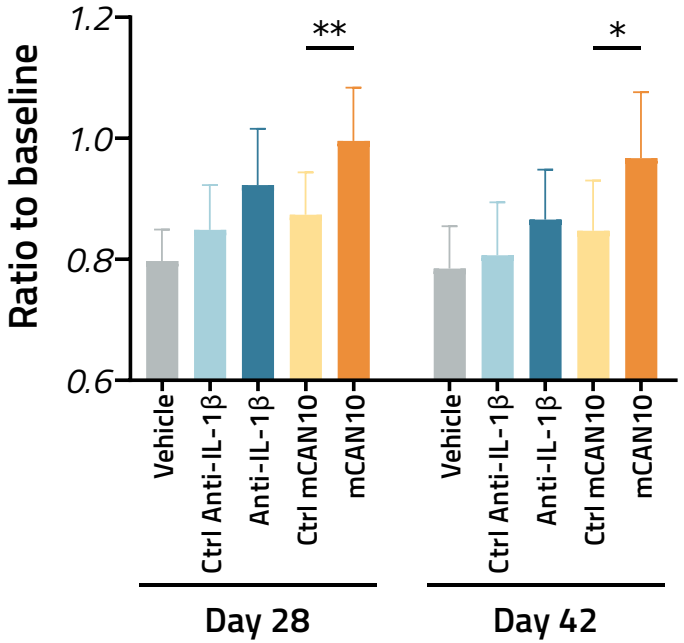
Experimental autoimmune myocarditis: mCAN10 improves heart function



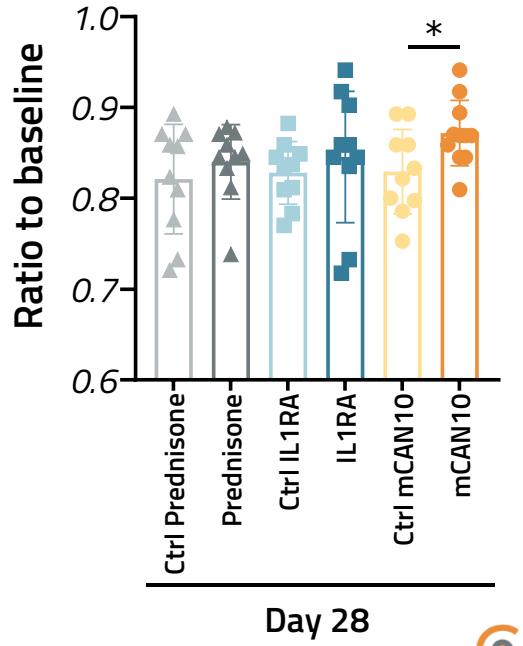
Left Ventricual Ejection Fraction (LVEF)



LVEF (treatment from day 7)

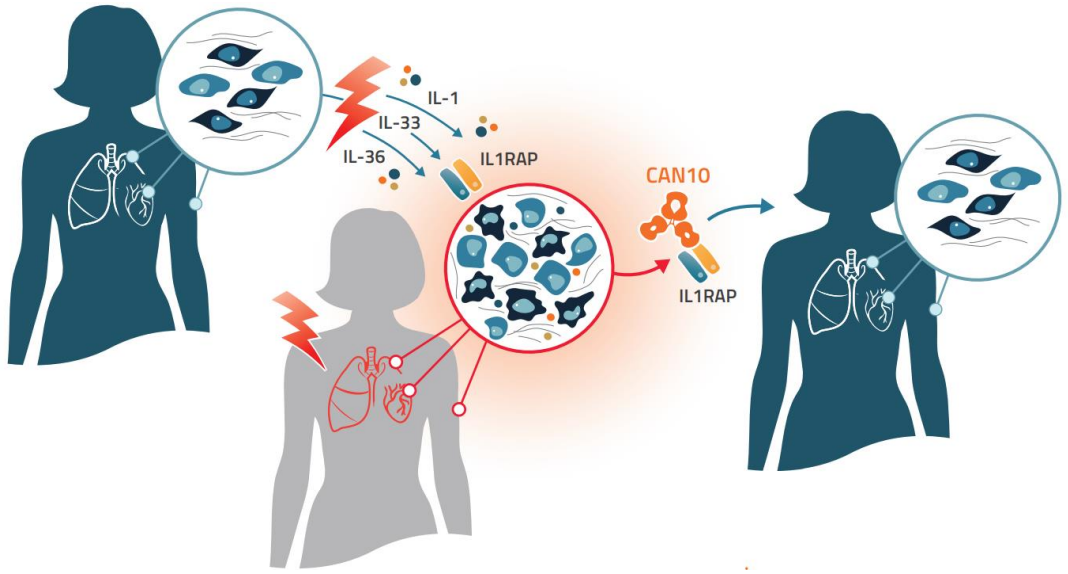


LVEF (treatment from day 7)



αMHC – α-Myosin Heavy Chain; CFA – Complete Freund's Adjuvant
n=10 per group

CAN10 – a potent blocker of IL1RAP function with effects in skin, lung, heart and vasculature



Status

- → CAN10 safe in GLP tox study
- → Strong results in several preclinical models, including lead indications systemic sclerosis and myocarditis
- → Phase I ongoing, early planning of patient studies (phase IIa)

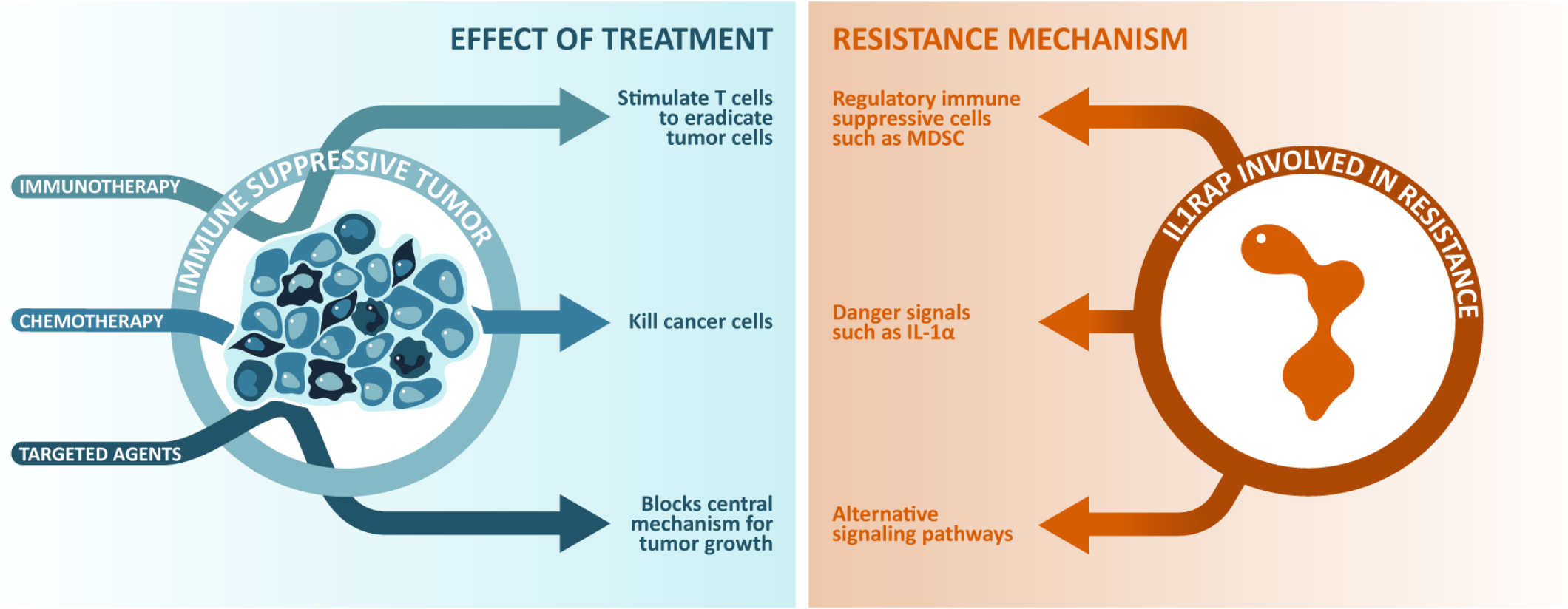
Clinical phase I study – Following plan

- → Phase I in healthy volunteers (SAD) followed by psoriasis patients (MAD); ongoing in Germany
- → No safety findings in first 4 SAD groups. Receptor occupancy confirmed to be in line with preclinical model
- → Up to 80 individuals (safety, pharmacokinetics, biomarkers)

SAD – Single Ascending Dose; MAD – Multiple Ascending Dose

NADUNOLIMAB (CAN04) OVERVIEW

Cantargia – Strategy to improve current cancer therapies

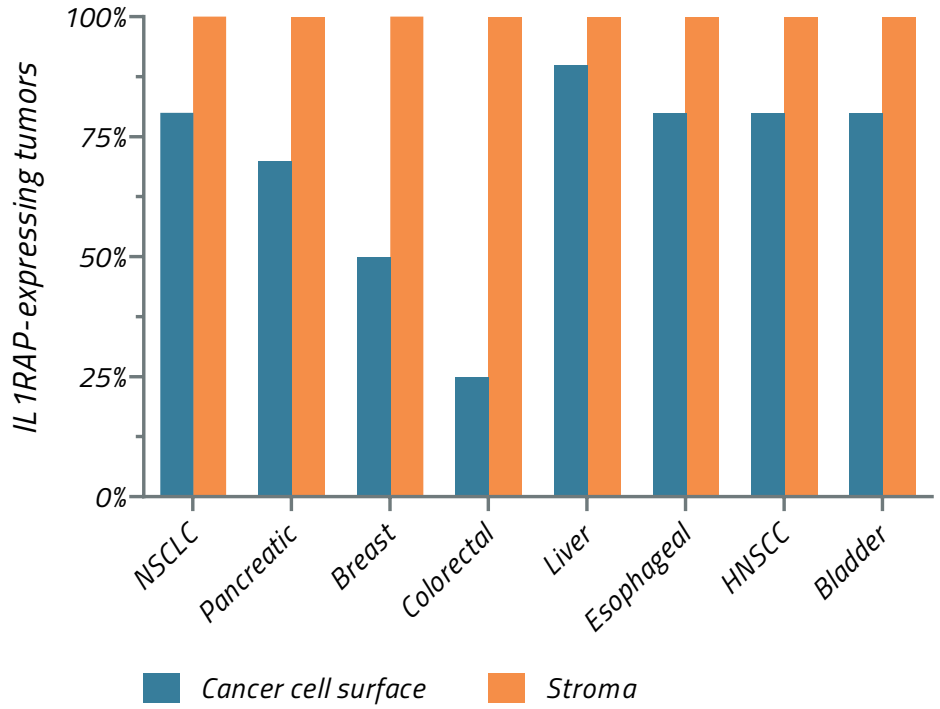


IL1RAP – A NOVEL TARGET WITH SEVERAL OPPORTUNITIES; CURRENT FOCUS ON SYNERGISTIC COMBINATIONS

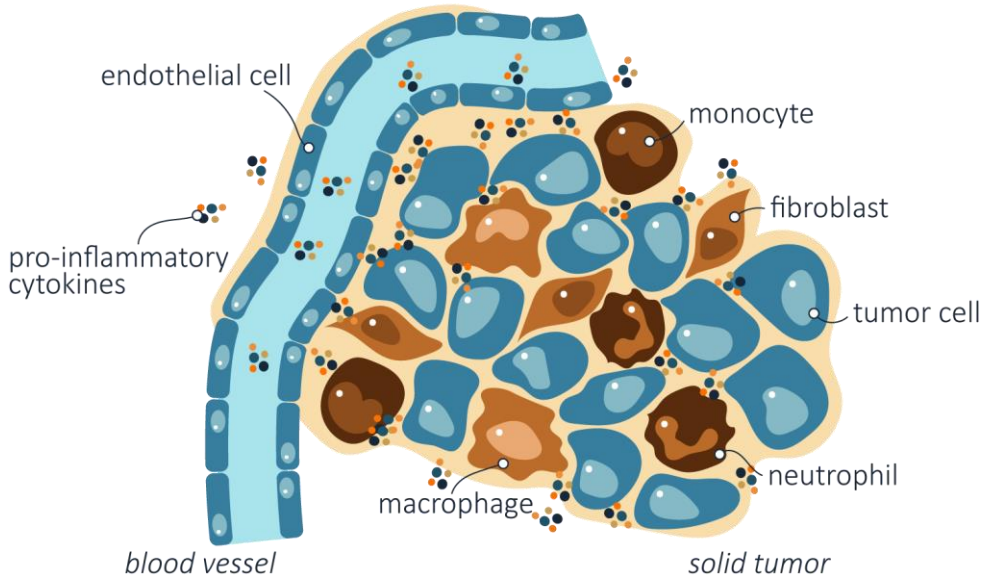
MDSC – Myeloid-Derived Suppressor Cells

IL1RAP overexpressed in most solid tumors

IL1RAP EXPRESSION IN SOLID TUMOR TYPES



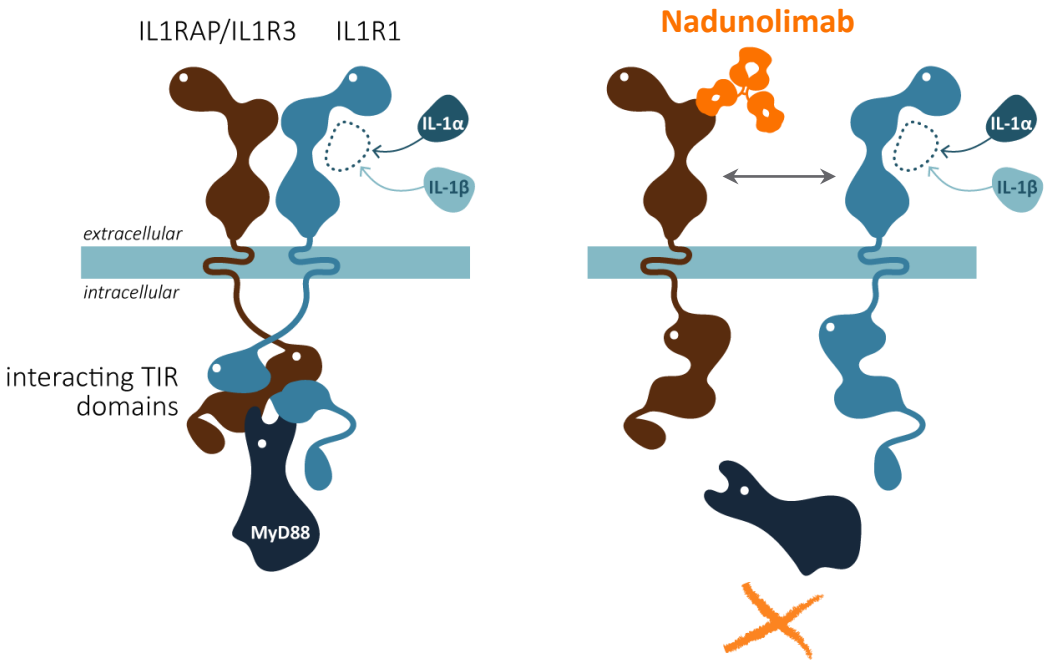
SEVERAL TUMOR-PROMOTING CELLS EXPRESSING IL1RAP IN THE TUMOR MICROENVIRONMENT

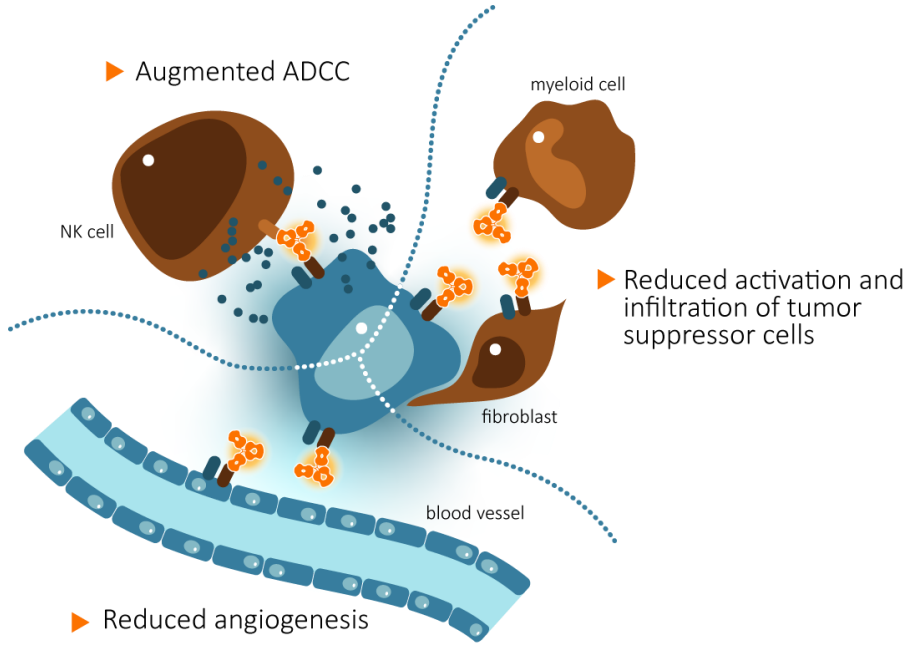


IL1RAP – DISTINCTLY OVEREXPRESSED IN TUMORS; LOW EXPRESSION IN NORMAL TISSUE

HNSCC – Head and Neck Squamous Cell Carcinoma

Targeting IL1RAP provides unique opportunities to treat cancer by IL-1α/β blockade and ADCC

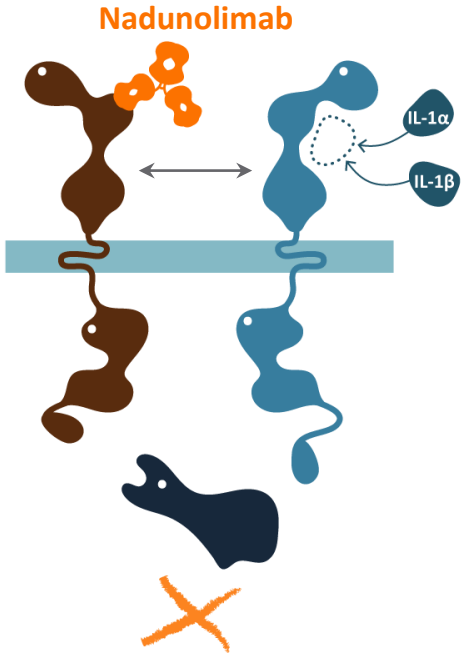


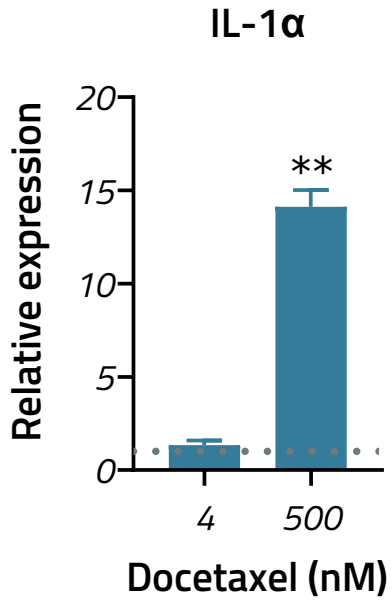


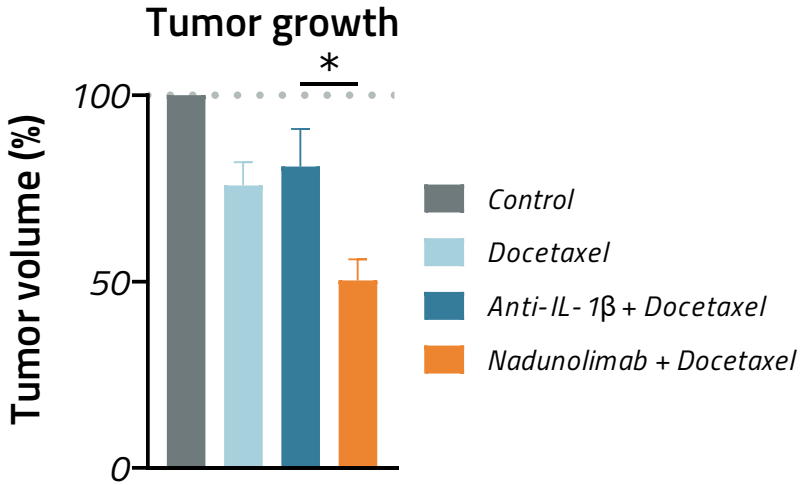
NADUNOLIMAB COUNTERACTS IMMUNE SUPPRESSION AND POTENTIATES THERAPY

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

Nadunolimab potentiates antitumor activity of chemo-therapy







→ **Nadunolimab blocks both IL-1α and IL-1β signaling**

→ **Docetaxel induces IL-1α release by tumor cells in vitro**

→ **Nadunolimab + docetaxel reduces in vivo tumor growth more potently than anti-IL-1β + docetaxel**

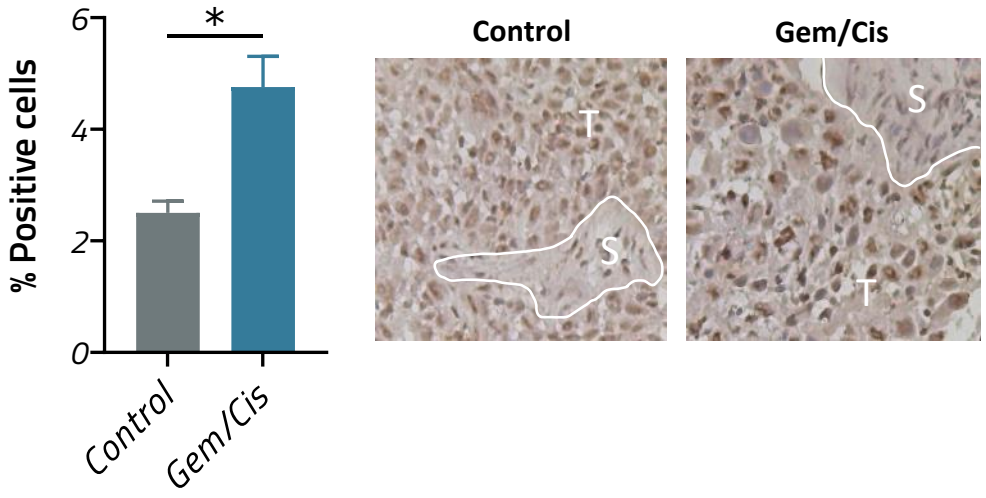
NADUNOLIMAB INCREASES DOCETAXEL EFFICACY IN CONTRAST TO IL-1β BLOCKADE

Rydberg-Millrud et al, Cancer Immunol Immunother 2022, https://rdcu.be/cUz5Y
n=3 per group in mid graph; n=20 per group in right graph

Cantargia

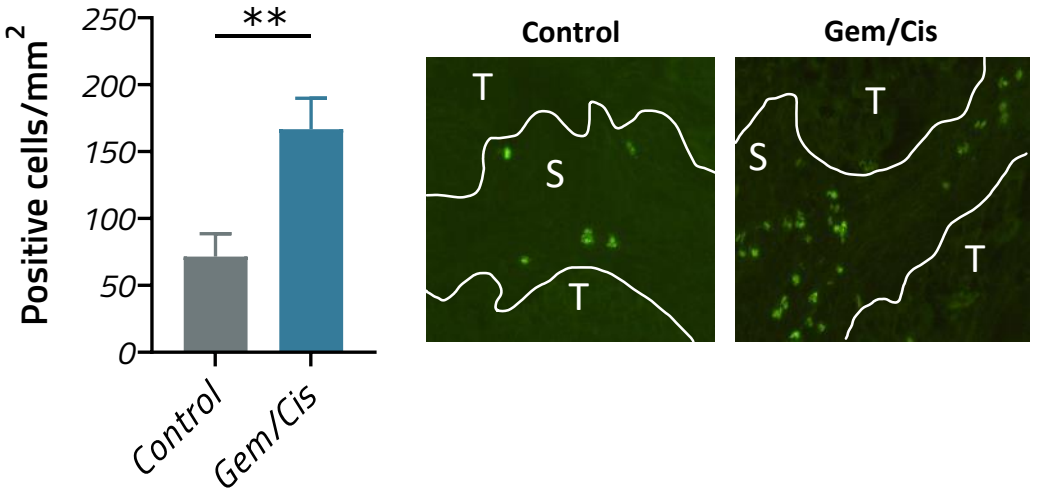
Chemotherapy induces IL-1α and IL-1β in the tumor

IL-1α
Tumor cell region (T)



→ **Gem/Cis induces release of IL-1α by tumor cells in tumors grown in vivo**

IL-1β-converting enzyme
Stromal cell region (S)



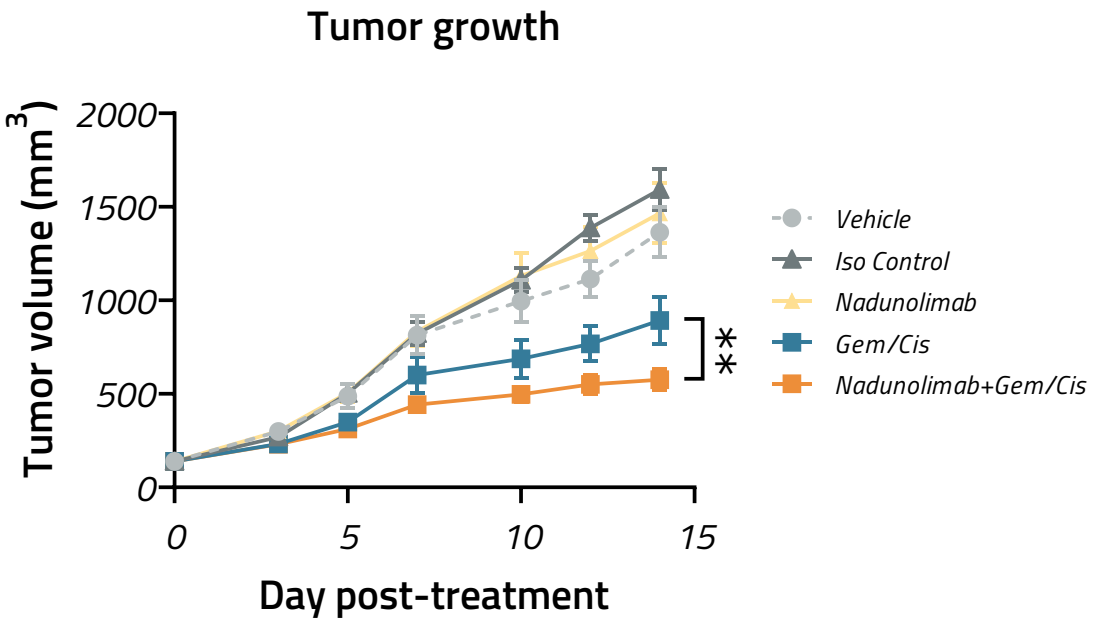
→ **Gem/Cis also induces release of IL-1β-converting enzyme (ICE) by stromal cells**

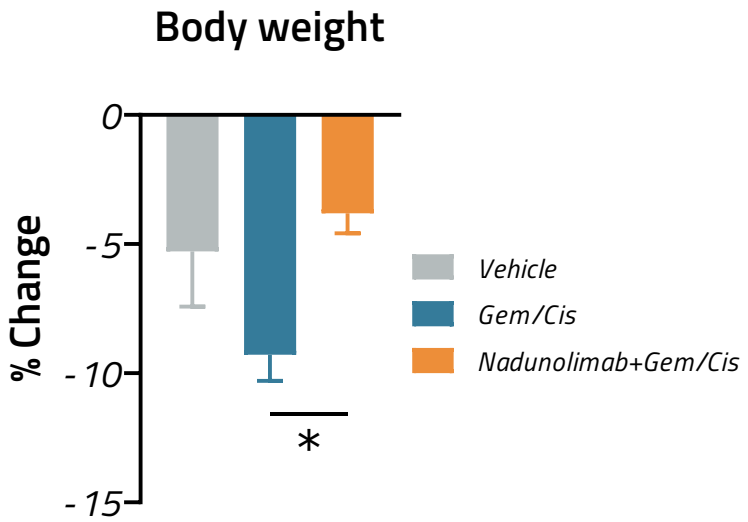
INCREASED LEVELS OF IL-1α AND IL-1β RESULTS IN CHEMORESISTANCE

Rydberg-Millrud et al, Cancer Immunol Immunother 2022, https://rdcu.be/cUz5Y
n=5 per group

Cantargia

Targeting IL1RAP uniquely synergizes with chemotherapy





→ **Nadunolimab increases efficacy of platinum-based chemotherapy in vivo**

→ **Nadunolimab also counteracts weight loss after chemo-therapy**

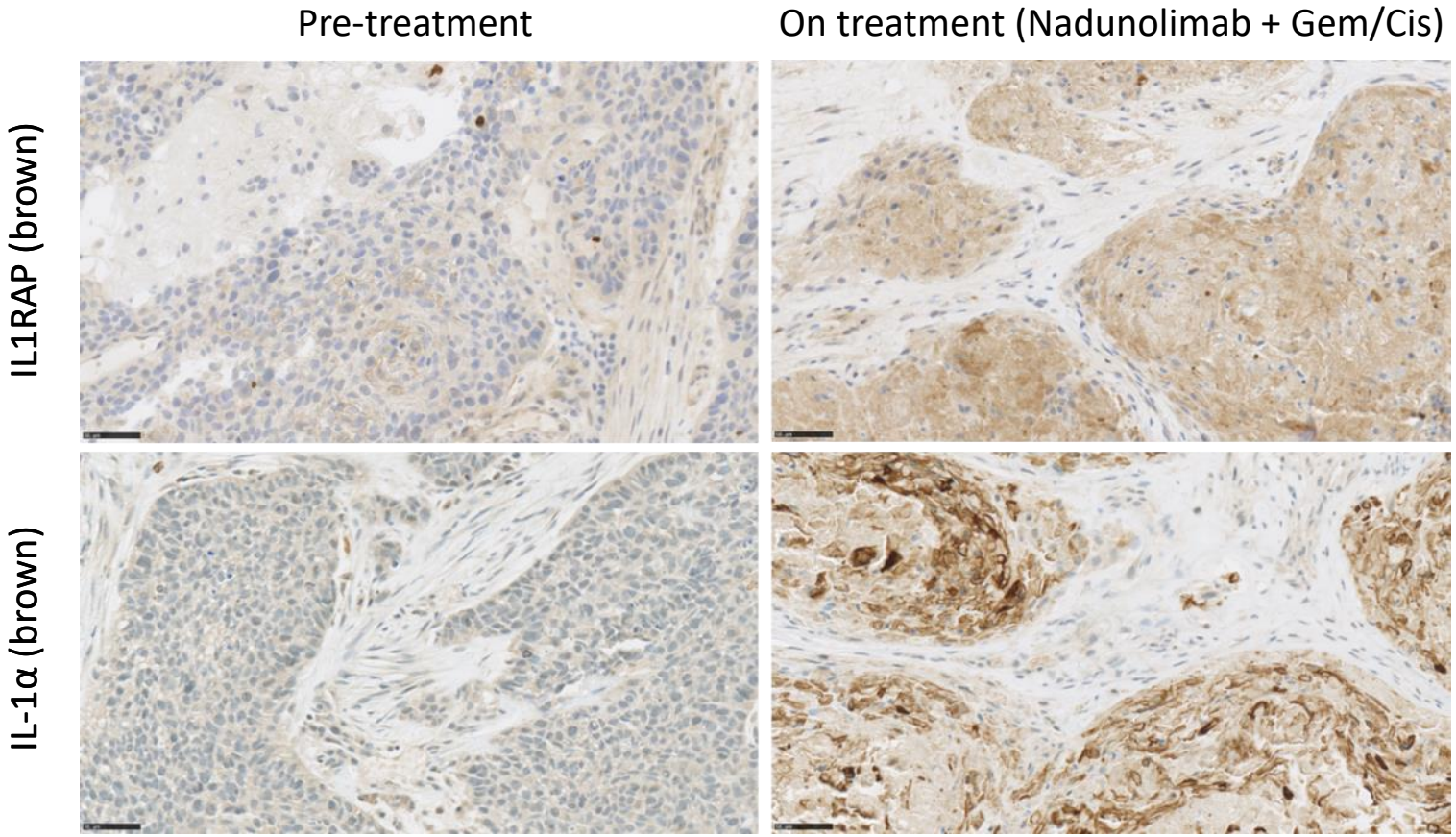
NADUNOLIMAB HAS POTENTIAL TO IMPROVE CHEMOTHERAPY EFFICACY AND TOLERABILITY

Rydberg-Millrud et al, Cancer Immunol Immunother 2022, https://rdcu.be/cUz5Y
n=10 per group

cantargia

NADUNOLIMAB (CAN04) CLINICAL RESULTS

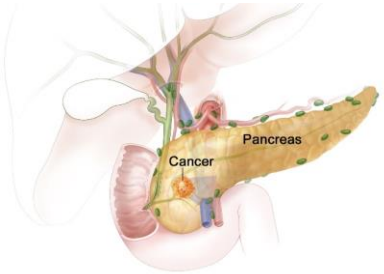
NSCLC – Induction of IL1RAP and IL-1α with therapy



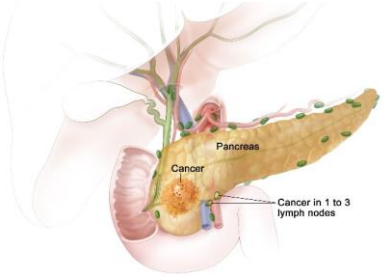
IL-1α INDUCED BY CHEMOTHERAPY IN LINE WITH PRECLINICAL FINDINGS;
WELL ESTABLISHED DANGER SIGNAL – ACTIVITY BLOCKED BY NADUNOLIMAB

PDAC – Staging and treatment

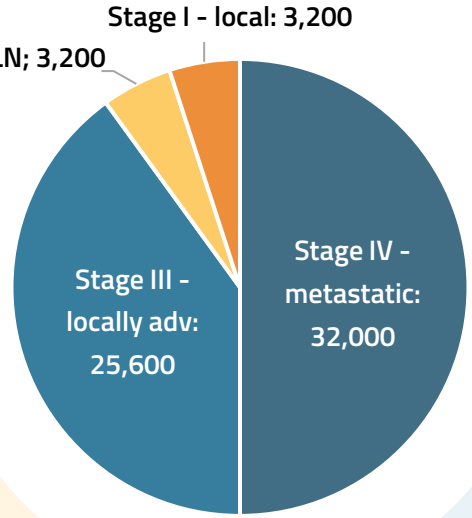
Expected number of cases US 2023: 64,000

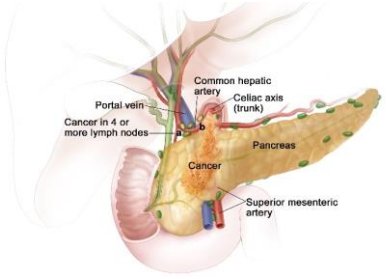


Stage I

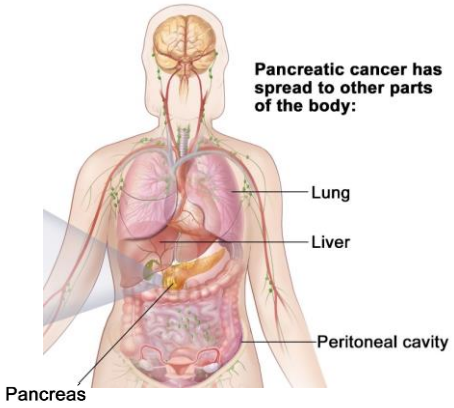


Stage II





Stage III



Stage IV

RESECTABLE OR BORDERLINE RESECTABLE DISEASE

Survival:

- → 15.7 – 28.0 mo

Treatment:

- → Neoadjuvant: gemcitabine+radiotherapy or mFOLFIRINOX
- → Radical pancreatic resection
- → Post-op chemo (gemcitabine+capecitabine or mFOLFIRINOX) or chemo+radiotherapy

LOCALLY ADVANCED OR METASTATIC DISEASE

Survival:

- → 8.5 – 11.1 mo

Treatment:

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

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

Images from National Cancer Institute

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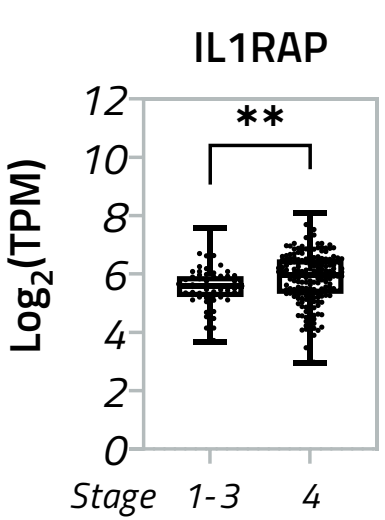
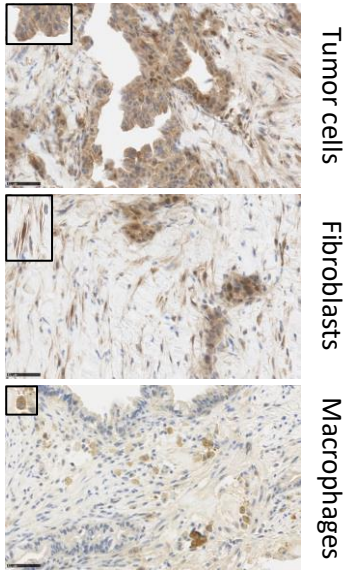
PDAC – IL1RAP linked to poor prognosis

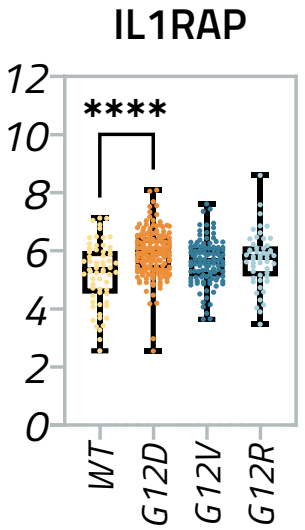
IL1RAP IN PDAC

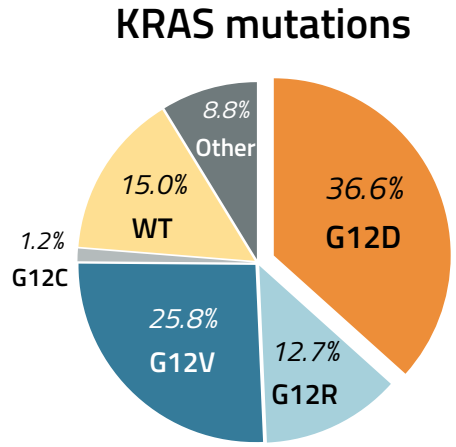




- IL1RAP levels increase with tumor stage
- IL1RAP expressed on both tumor cells, cancer-associated fibroblasts and macrophages in tumor microenvironment
- High IL1RAP correlates with lower efficacy after 1st line Gem/Abraxane

KRAS MUTATIONS IN PDAC





- Over 80 % of PDAC patients have a KRAS mutation; G12D is the most common
- KRAS G12D has a worse prognosis with HR 1.47 (Bournet et al, 2016)
- IL1RAP is overexpressed in patients with KRAS G12D

CLEAR LINK BETWEEN IL1RAP, KRAS G12D AND PDAC PROGNOSIS

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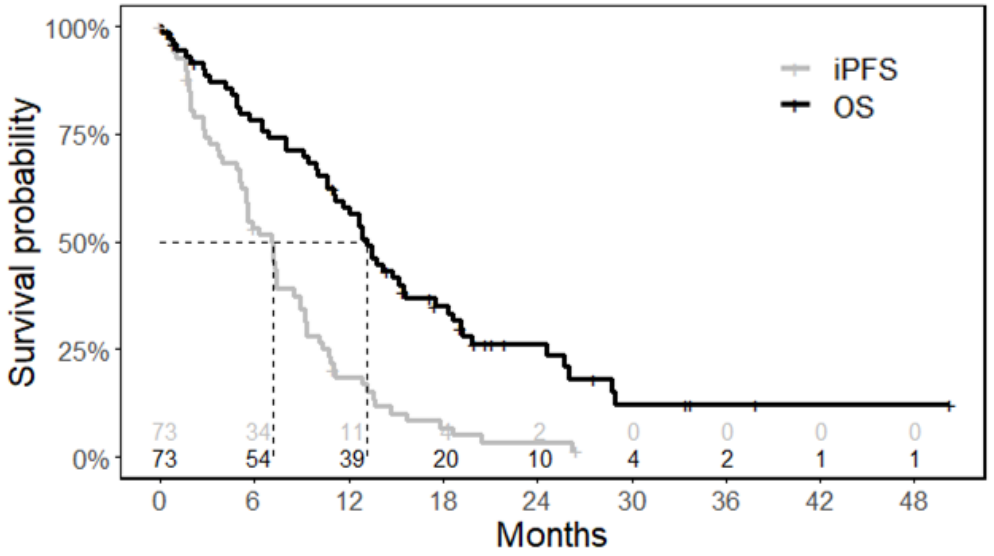
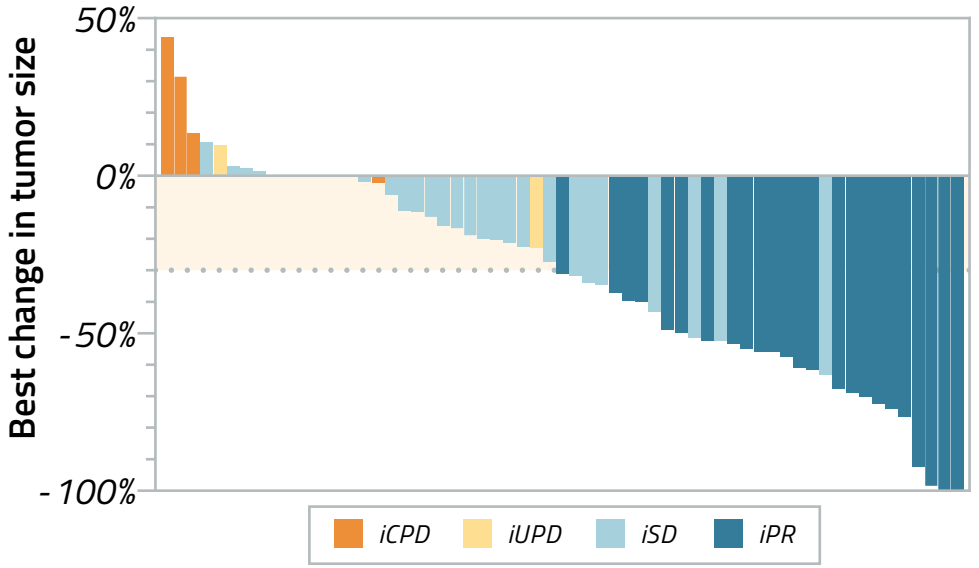
PDAC – Positive interim data in 1st line patients

OS and iPFS for mITT patients



Best responses according to iRECIST



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

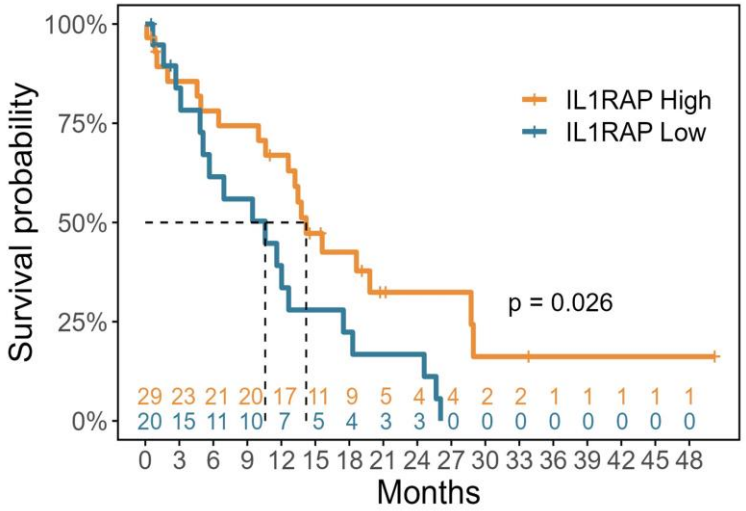
- → 33% response rate with long OS and iPFS
  - → Additional 5 (7%) patients had on-treatment benefit beyond progression
- → Promising OS (13.2 mo), iPFS (7.2 mo) and DCR (71%); 2 patients still on treatment

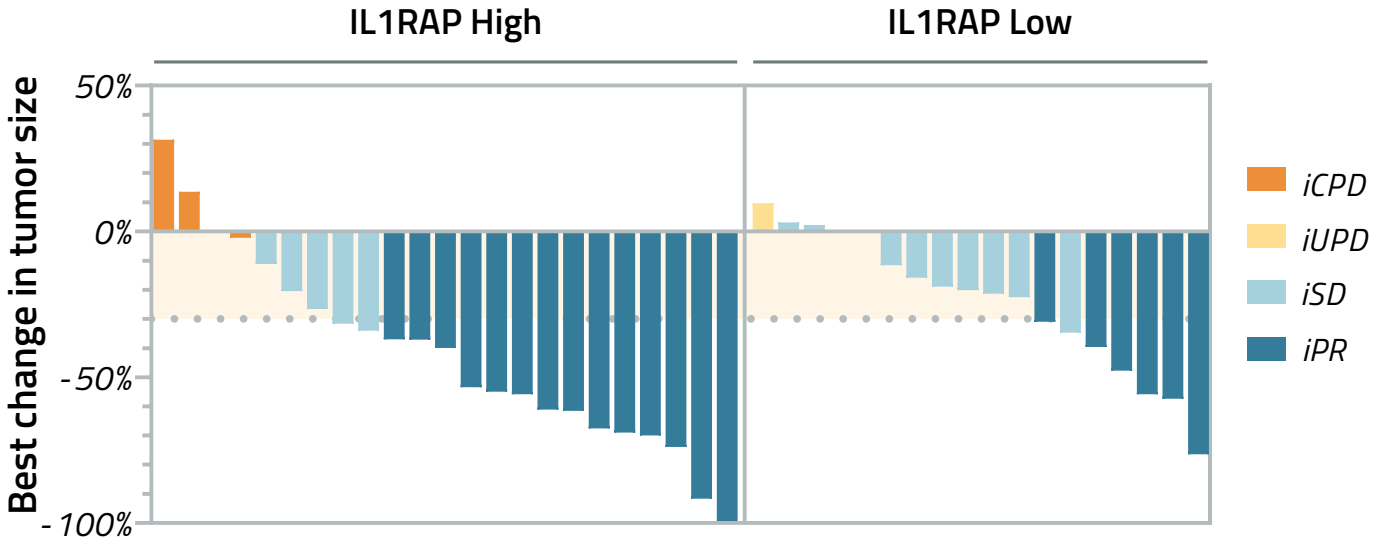
PFS AND OS LONGER THAN EXPECTED GIVEN HISTORICAL CONTROL IN PDAC – PHASE IIB TRIAL IN PREPARATION

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

PDAC – Strong efficacy in patients with high tumor IL1RAP level

OS by IL1RAP subgroup





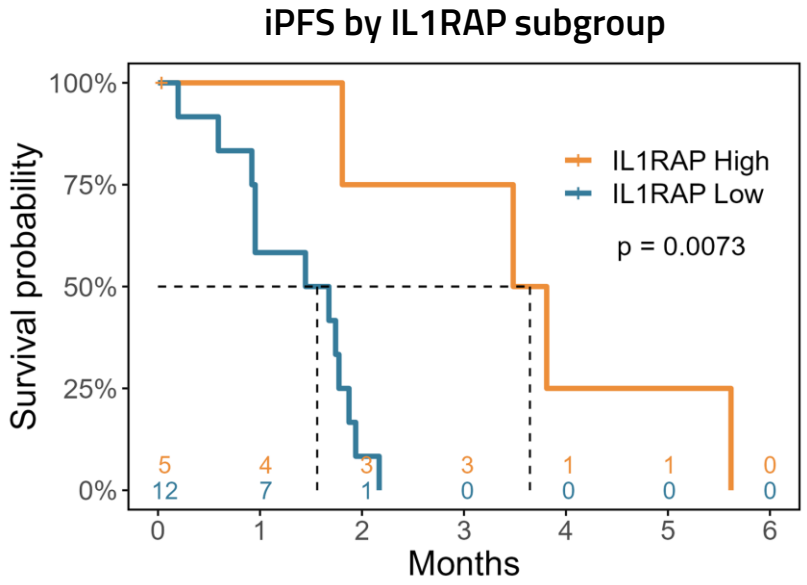
Efficacy analysis for IL1RAP High (n=29) vs IL1RAP Low (n=20) PDAC patients (1st line, combination with Gem/Abraxane):

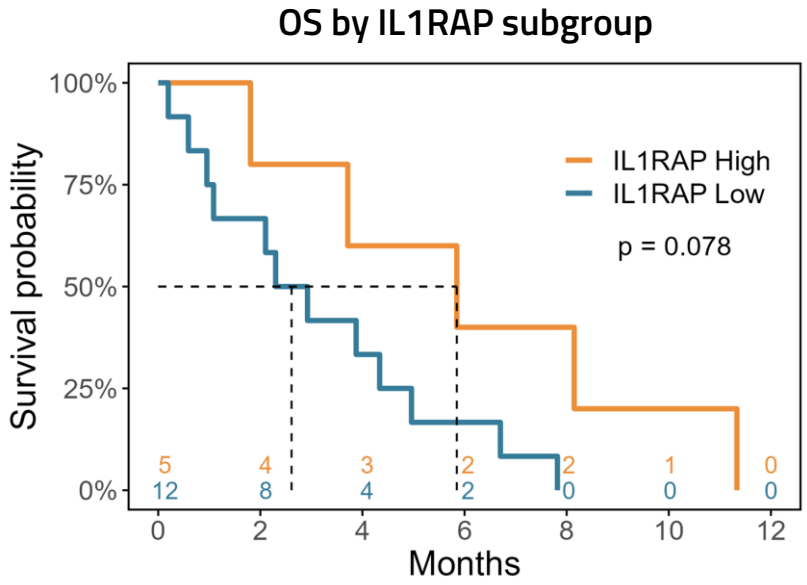
- → Significantly prolonged OS in ILRAP High vs IL1RAP Low patients (14.2 vs 10.6 mo; p=0.026)
- → Deeper and more durable responses in IL1RAP High subgroup: 11 patients had 50% or more tumor size decrease

NEW DATA IN IL1RAP HIGH PATIENTS SUPPORT ONGOING DEVELOPMENT AND EXPLORATION OF NEW OPPORTUNITIES

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

PDAC – Strong efficacy in patients with high tumor IL1RAP level





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

→ Significantly prolonged iPFS in IL1RAP High vs IL1RAP Low patients (3.6 vs 1.6 mo; p=0.0073)

→ Trend for OS advantage in IL1RAP High patients (5.8 vs 2.6 mo; p=0.078)

NADUNOLIMAB MONOTHERAPY RESULTS SUPPORT EFFECTS IN IL1RAP HIGH PATIENTS

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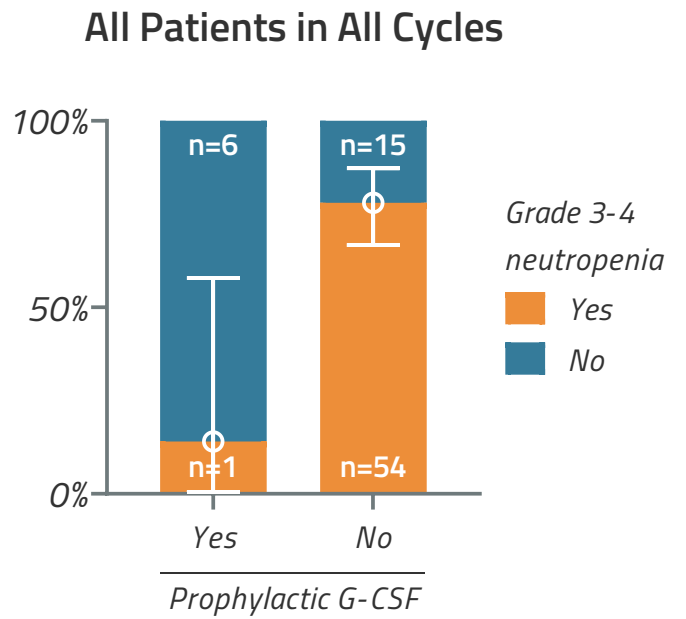
PDAC – Safety profile is manageable and supports MOA

- Neutropenia manageable through G-CSF prophylaxis
  - In 7 patients given G-CSF prophylaxis, only 1 developed grade 3-4 neutropenia
- Only 1 % peripheral neuropathy grade 3-4 observed
  (17% in historical controls)

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

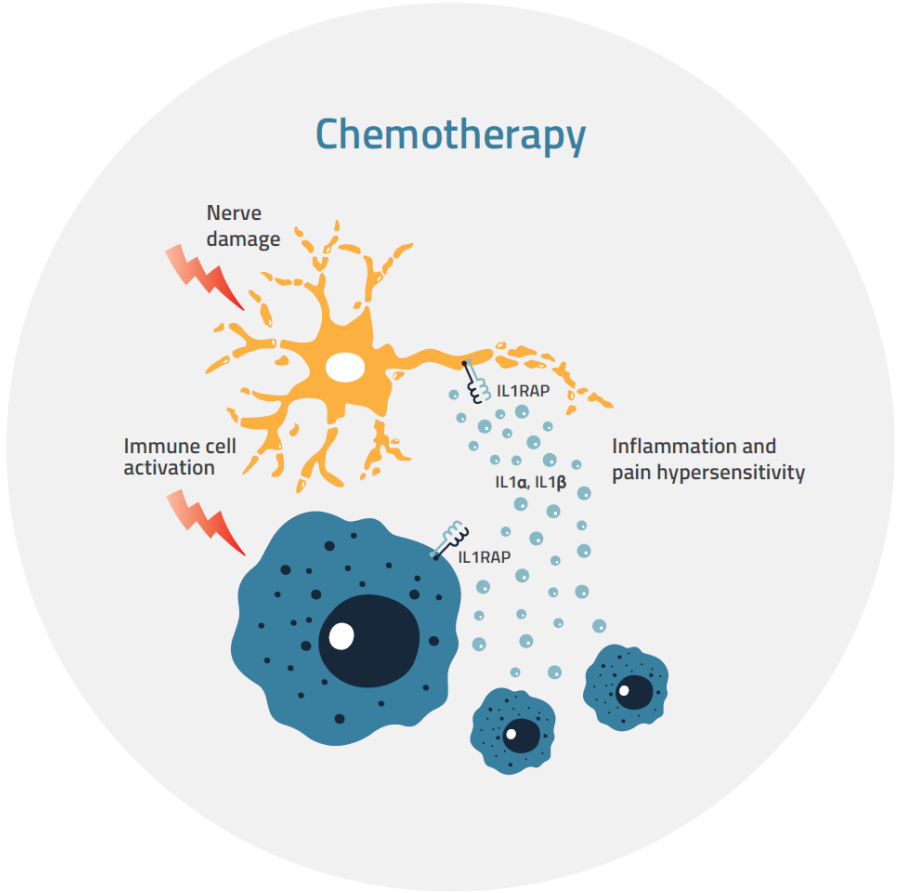


G-CSF PROPHYLAXIS IMPLEMENTED IN FUTURE TRIALS;
POTENTIAL REDUCTIONS OF SOME SIDE EFFECTS TO BE DOCUMENTED IN RANDOMIZED TRIALS

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

Nadunolimab and alleviation of neuropathy

- Chemotherapy induce neuropathy by several pathways including IL-1 (neuroinflammation)
- Nadunolimab, phase 2 data in PDAC with Gem/nabP
  - lower Grade 3-4 peripheral neuropathy than expected from historical controls (1% vs 17%).
- Correlation between nadunolimab dose level and protective effect
- Counteraction of chemotherapy-induced neuropathy in animal models



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

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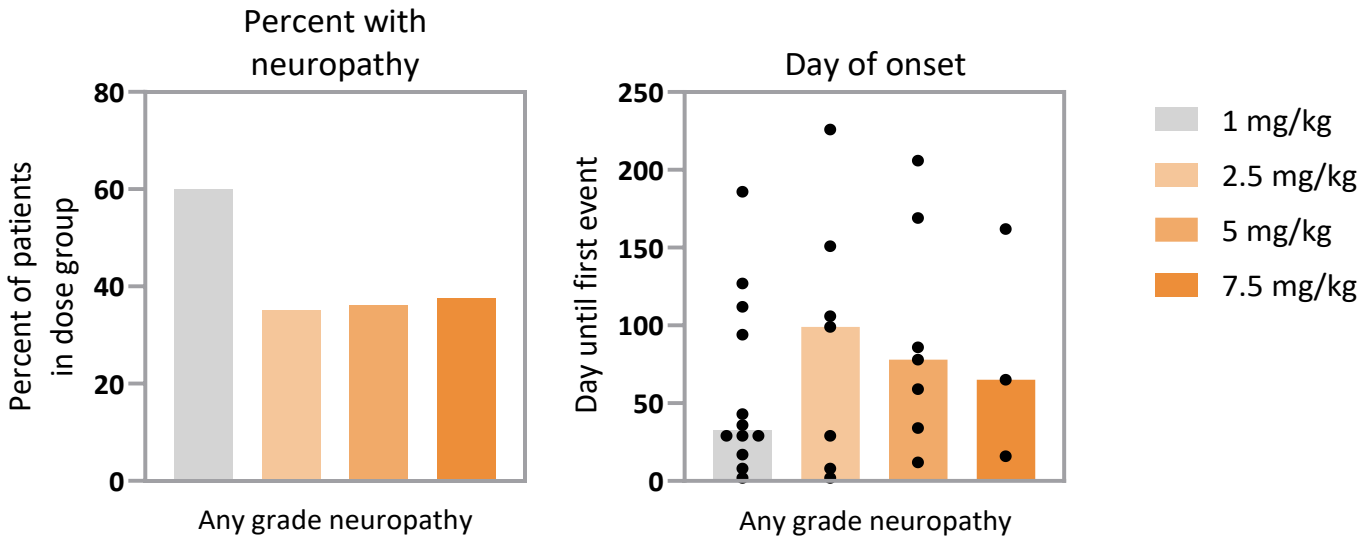
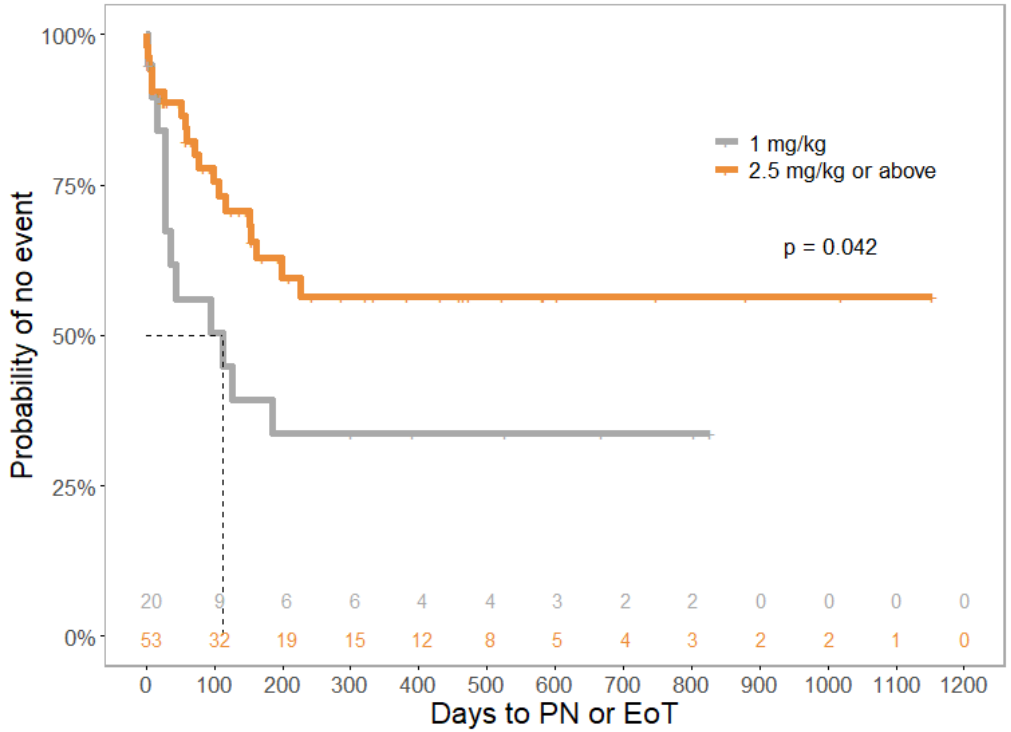
Nadunolimab and alleviation of neuropathy





CORRELATION WITH NADUNOLIMAB AND DECREASE IN NEUROPATHY

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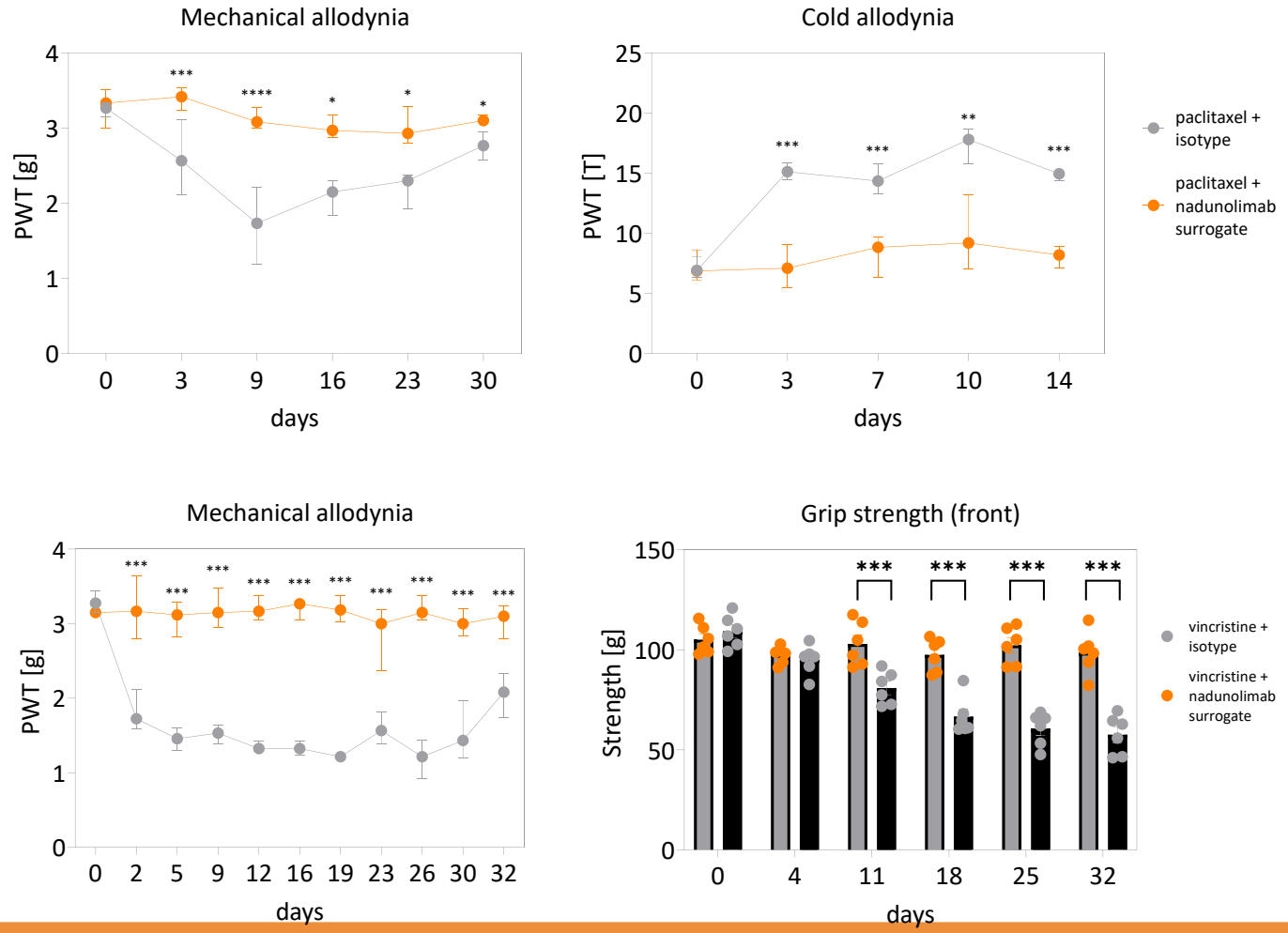
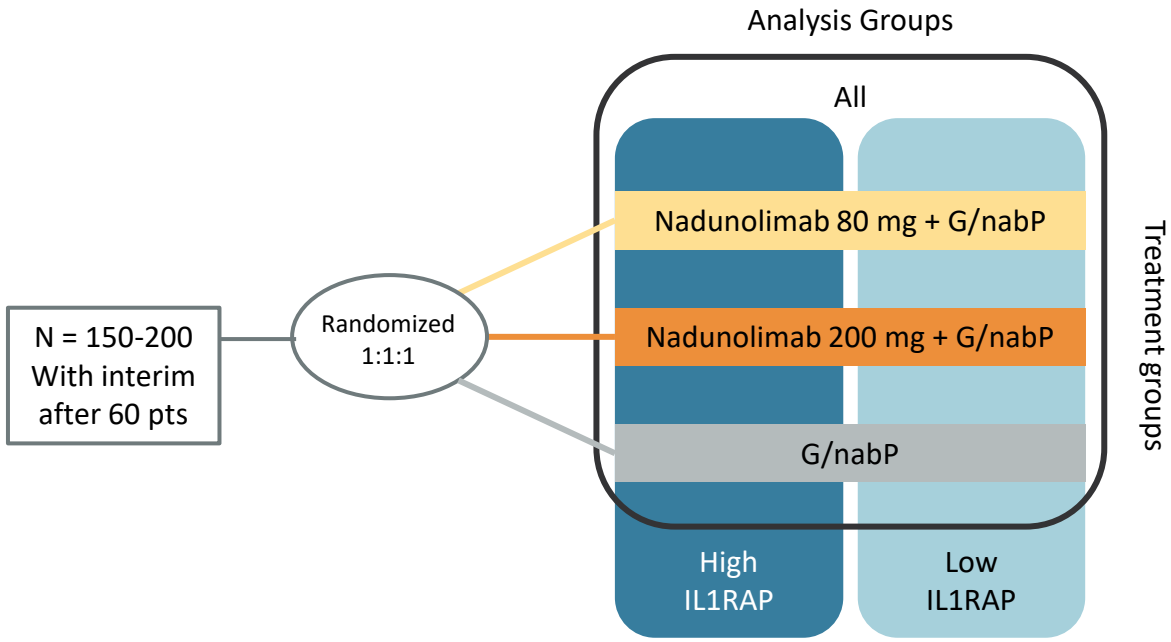
Nadunolimab and alleviation of neuropathy



NADUNOLIMAB COUNTERACT NEUROPATHY INDUCED BY PACLITAXEL OR VINCRISTINE IN ANIMAL MODELS

PDAC – Phase IIb study design



Primary endpoint:

→ PFS

Pre-planned interim review :

→ After 60 pts to allow strategic next steps incl. regulatory

Timelines:

→ FPI planned for mid 2024 (US regulatory approval obtained)

Geography:

→ USA and Europe

PHASE IIB TRIAL TO VALIDATE STRONG SIGNAL OF ACTIVITY IN IL1RAP HIGH PATIENTS

Nadunolimab PDAC milestone targets

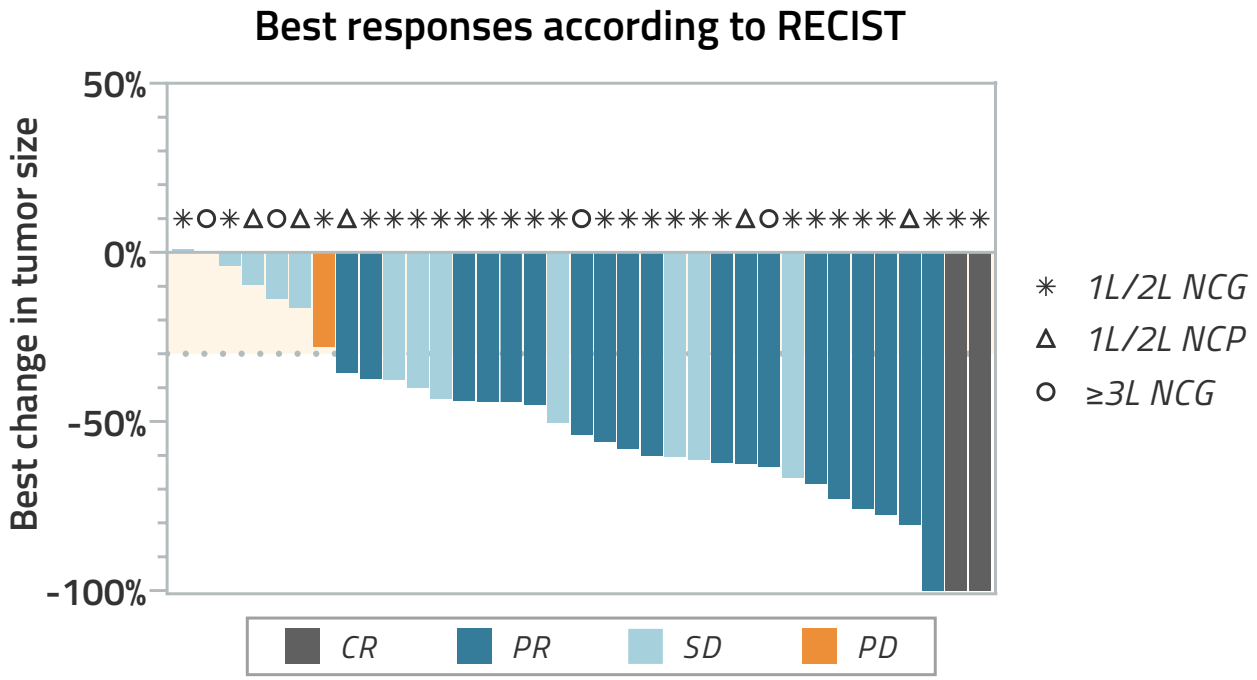
mid-2024	H1 2025	H2 2025	H1 2026	H1 2027	H1 2028	H2 2028
Start PANFOUR study	*PANFOUR enrolment completed*	*PANFOUR study results*				
	FDA meeting	***FDA EOP2 meeting***				
	Phase III study	*preparation*	*Start Phase III study*	*Phase III enrolment completed*	*Phase III study results*	
					Potential BLA / MAA submission	***Potential US market launch***

Confirm CANFOUR high IL1RAP results and accelerated path to market

- *Interim efficacy & subgroup analysis*
- *Discuss and agree dose and data driven patient selection strategy for Phase III / BLA*
  - *IL1RAP or KRAS or serum BM patient selection*

PANFOUR study design address FDA Project Optimus and Frontrunner guidelines and de-risks development with interim snapshot to evaluate efficacy, safety and biomarker subgroup analysis

NSCLC – Promising efficacy of nadunolimab combination therapy



High ORR to nadunolimab and platinum doublets in different lines of therapy:

- Gem/Cis 1st/2nd line: ORR 53% (n=30)
- Carbo/Pemtrex 1st/2nd line: ORR 60% (n=5)
- Gem/Cis ≥3rd line: ORR 50% (n=4)

CONSISTENTLY HIGH RESPONSE RATES WITH NADUNOLIMAB AND PLATINUM DOUBLETS

CR – Complete Response; PR – Partial Response; SD – Stable Disease; PD – Progressive Disease
NCG – Nadunolimab/Cisplatin/Gemcitabine; NCP – Nadunolimab/Carboplatin/Pemetrexed

NSCLC – Long-term benefit with strong signal in non-squamous subtype

	All (n=30)	Historical data[1,2]	Non-squamous (n=16)	Non-squamous, historical data[3]
Median OS	**13.7 mo**	10.3 mo	**15.9 mo**	11.3 mo
Median PFS	**7.0 mo**	5.1 mo	**7.3 mo**	4.9 mo
ORR	**53%**	22-28 %	**56%**	19%
Complete response	**6.7% (n=2)**	<1%	**12.5% (n=2)**	<1%

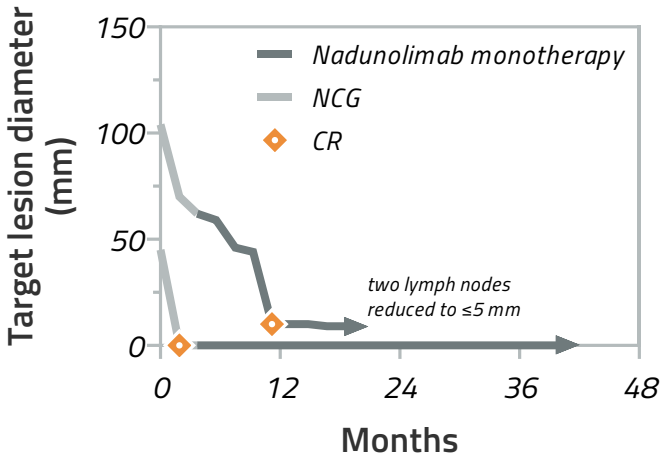
- Strongest efficacy in 16 non-squamous patients
- Long-term benefit of nadunolimab combination therapy, including two complete responses

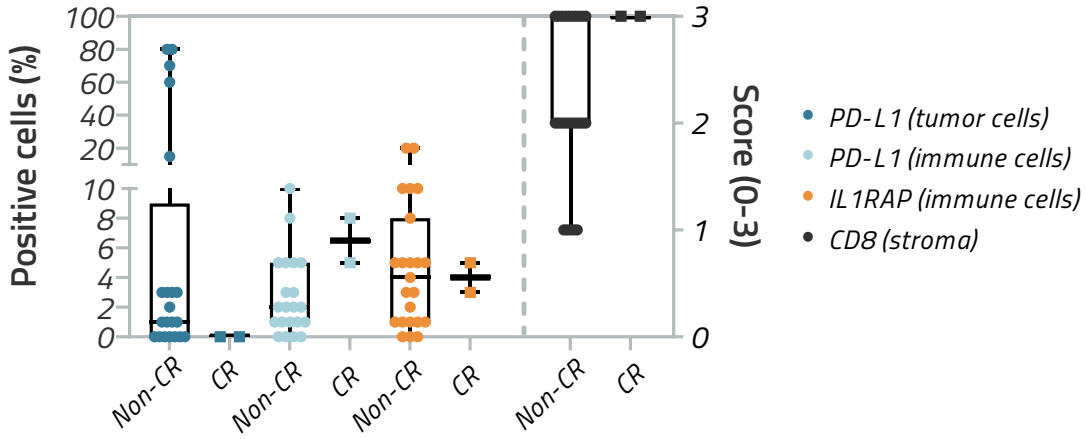
Treatment course for each individual patient

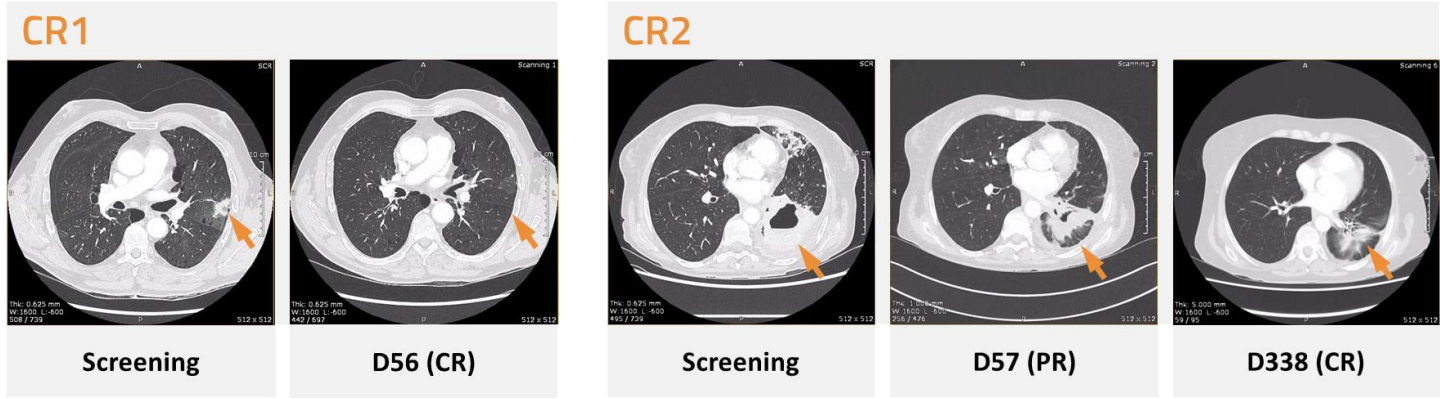
NADUNOLIMAB COMBINATION THERAPY COMPARES VERY FAVORABLY TO HISTORICAL DATA FOR CHEMOTHERAPY ALONE

[1] Schiller et al, N Engl J Med 2002; [2] Scagliotti et al, J Clin Oncol 2008; [3] Gandhi et al, N Engl J Med 2018
PD – Progressive Disease; SD – Stable Disease; PR – Partial Response; CR – Complete Response; NCG – Nadunolimab/Cisplatin/Gemcitabine

NSCLC – Complete responders with distinct biomarker profile







- One CR maintained over 3 yrs (CR1), second CR achieved on nadunolimab monotherapy approx. 9 mo post-chemo (CR2)
- Both non-squamous, progressed on pembro, with PD-L1^{-} tumor cells, PD-L1^{+} immune cells in tumor

SIGNAL OF NADUNOLIMAB MONOTHERAPY ACTIVITY RESULTING IN COMPLETE RESPONSE
FURTHER BIOMARKER ANALYSES ONGOING FOR FUTURE DEVELOPMENT STRATEGY

CR – Complete Response; NCG – Nadunolimab/Cisplatin/Gemcitabine

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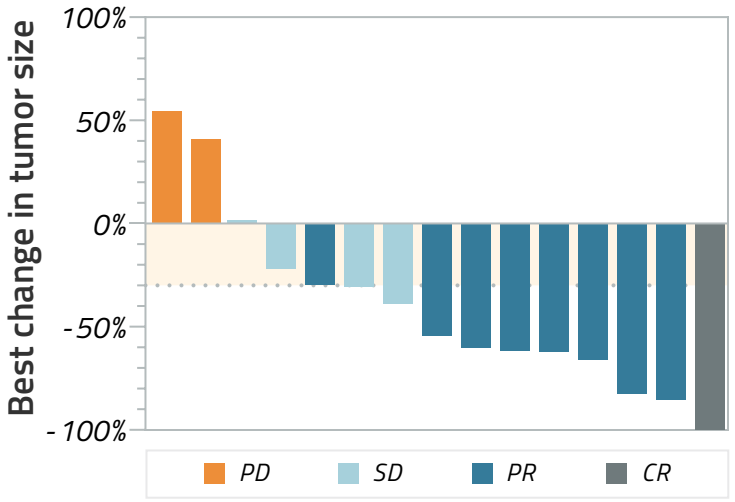
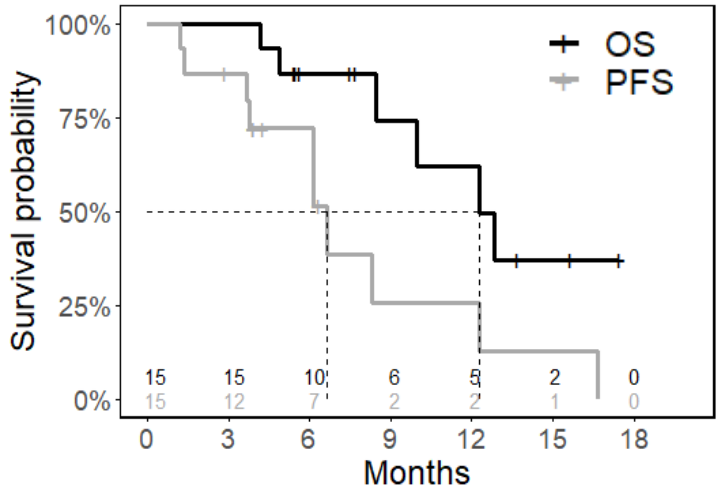
TNBC – Promising early safety and efficacy

Best responses according to RECIST



OS and PFS



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

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

15 patients enrolled in the dose-escalation phase:

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

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

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

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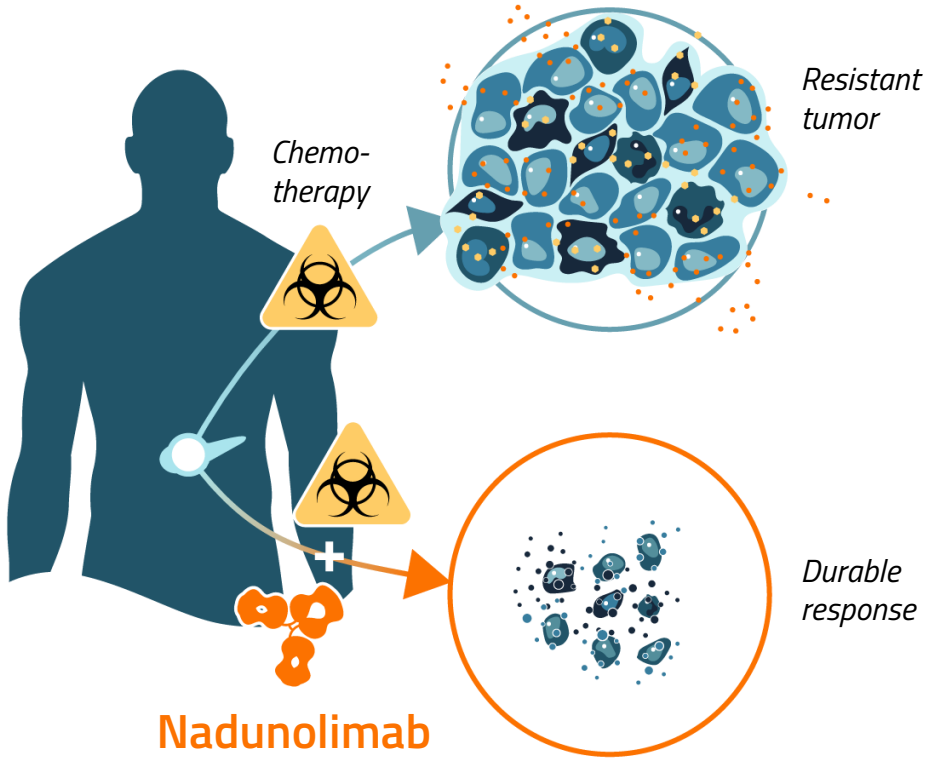
Key messages

- Nadunolimab, investigated in almost 300 pts, shows efficacy both as monotherapy as well as in combination.
- Clinical results strongly support potential unique first-in-class opportunities in PDAC, NSCLC and TNBC. Controlled phase 2 trial ongoing in TNBC and in preparation for PDAC
- PDAC patients with high IL1RAP level respond best to nadunolimab combination therapy despite having a worse prognosis.
- The mechanism include counteracting chemotherapy resistance through upregulatation of both IL-1α and IL-1β, signaling through IL1RAP. The mechanism is highly relevant for ADC combination strategies



PROMISING EFFICACY OF NADUNOLIMAB– CURRENT FOCUS ON RANDOMIZED CLINICAL TRIALS AGAINST CHEMOTHERAPY

MILESTONES & INVESTMENT HIGHLIGHTS

Upcoming milestones

Nadunolimab

PDAC

- Start of Phase IIb trial in 150-200 patients
- Phase IIb top-line data in 2025

TNBC

- Full recruitment H2 2024
- Randomized Phase II top-line data in late 2024

AML/MDS

- Start phase I/II mid 2024 (DOD sponsored with MDA)

CAN10

- Phase I data updates during 2024 (including safety and biomarkers)

Additional milestones

- NSCLC – Efficacy & biomarker data from CANFOUR during 2024
- New clinical data presented from CIRIFOUR, CAPAFOUR and CESTAFOUR trials
- New preclinical and translational results

EXTENSIVE NEWS FLOW EXPECTED DURING 2024

Cantargia – Investment highlights

NOVEL IL1RAP ANTIBODIES, POTENTIAL TO TREAT CANCER & INFLAMMATORY DISEASE

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

NADUNOLIMAB: CLEAR ACTIVITY SIGNALS IN CANCER THERAPY WITH UPCOMING CATALYSTS

- Strong clinical interim results in PDAC and NSCLC, and promising initial results in TNBC; >250 patients treated
- Randomized Phase II trial ongoing in TNBC (initial data late 2024); Phase IIb trial in preparation in PDAC (top-line data 2025)

CAN10: OPPORTUNITY IN AUTOIMMUNITY/INFLAMMATION

- Pronounced activity in models of systemic sclerosis, myocarditis, psoriasis, atherosclerosis and inflammation
- Phase I clinical trial ongoing, initial results show good safety and receptor occupancy. New data Q2 2024

CORPORATE STRENGTH DRIVING INNOVATION

- Solid cash position with runway into 2025 (143MSEK (14 MUSD) cash & equivalents at Q1 2024)
- Robust patent portfolio: IL1RAP antibody target in oncology (2032), nadunolimab (2035) and CAN10 (2041)

Cantargia IP

- Lead candidate anti-IL1RAP antibody **CAN04**
  - Expiry year **2035**
  - Granted (e.g. Europe, USA, China, Japan)
  - Mother patent and divisionals
- Lead candidate anti-IL1RAP antibody **CAN10**
  - Expiry year **2041**
  - Granted (USA)
  - Examination at early stage in remaining territories
- Anti-IL1RAP for treatment of **solid tumors**
  - Expiry year **2032**
  - Granted (e.g. Europe, USA, China, Japan)
  - Mother patent and divisionals
- Anti-IL1RAP for treatment of **hematological disorders**
  - Expiry year **2030**
  - Granted (e.g. Europe, USA, China, Japan)
  - Mother patent and divisionals
- Anti-IL1RAP for treatment of **myeloproliferative disorders**
  - Acquired from Cellerant; expiry year **2029**
  - Granted (USA)
- Additional patent families covering alternative anti-IL1RAP antibodies
  - Starting point for CANxx project(s)

Expiry dates conservative, not incorporating potential prolongations of market exclusivity