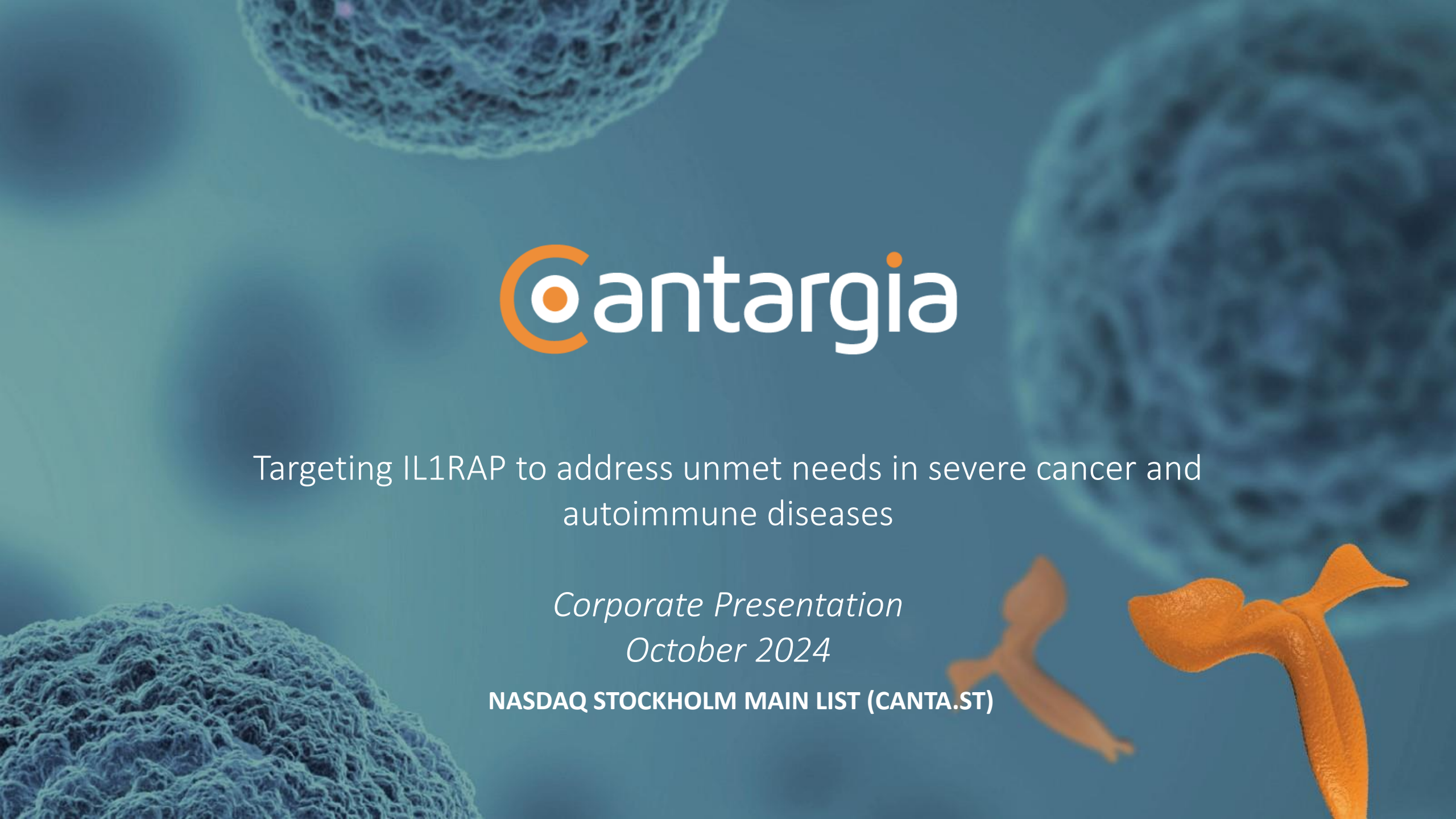
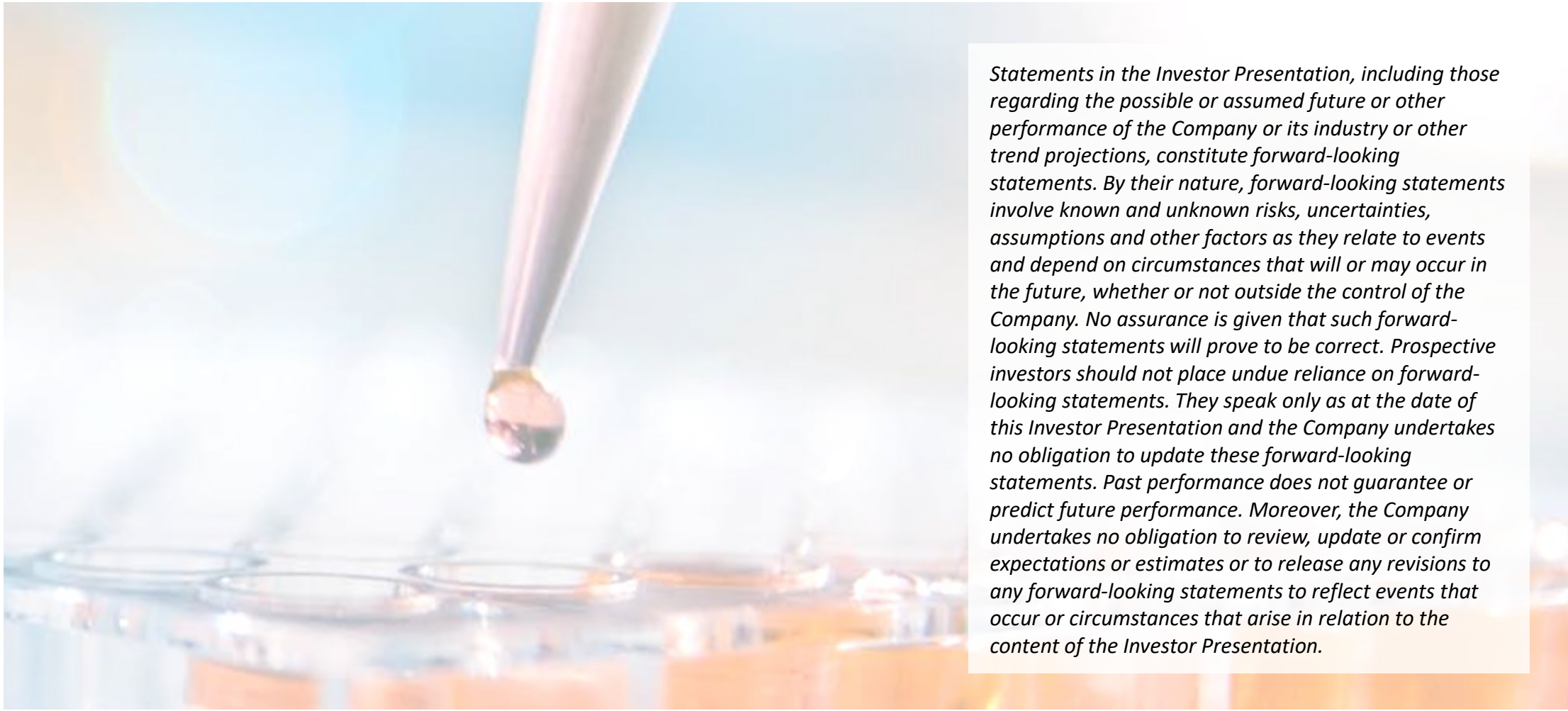


Targeting IL1RAP to address unmet needs in severe cancer and autoimmune diseases

Corporate Presentation
October 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; >300 patients treated
- Randomized Phase 2 trial ongoing in TNBC (initial data H1 2025); Phase 2b trial in preparation in PDAC

CAN10: OPPORTUNITY IN AUTOIMMUNITY/INFLAMMATION

- Pronounced activity in models of systemic sclerosis, myocarditis, psoriasis, atherosclerosis and inflammation
- Phase 1 clinical trial ongoing, initial results show good safety, receptor occupancy and potent PD-effects.

CORPORATE STRENGTH DRIVING INNOVATION

- Solid cash position with runway into 2025 (105MSEK (~10 MUSD) cash & equivalents at Q2 2024)
- Robust patent portfolio: IL1RAP antibody target in oncology (2032), nadunolimab (2035) and CAN10 (2041)

Current pipeline

Asset	Indication	Discovery	Preclinical	Phase 1	Phase 2	Phase 3
Nadunolimab	*PDAC**, TNBC*, NSCLC**					
CAN10	*Hidradenitis Suppurativa Systemic Sclerosis*					
CANxx	*New opportunities within IL1RAP platform*					

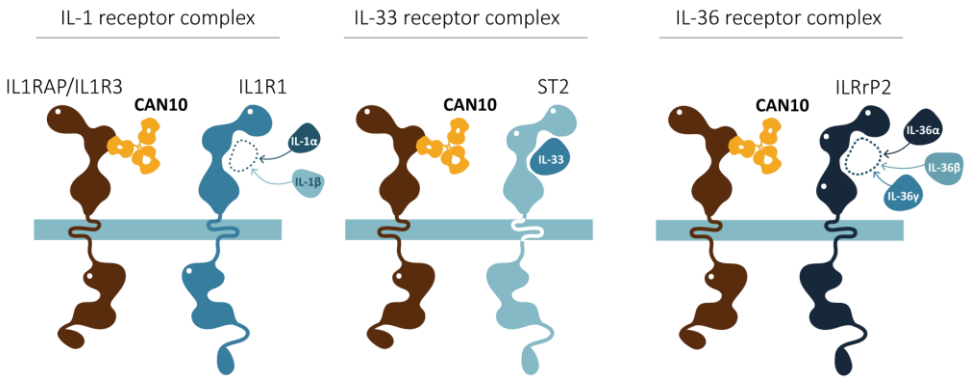
PDAC – pancreatic cancer; TNBC – triple-negative breast cancer; NSCLC – non-small cell lung cancer
**) Recruitment in randomized phase 2 trial ongoing in TNBC*
***) Recruitments finalized*

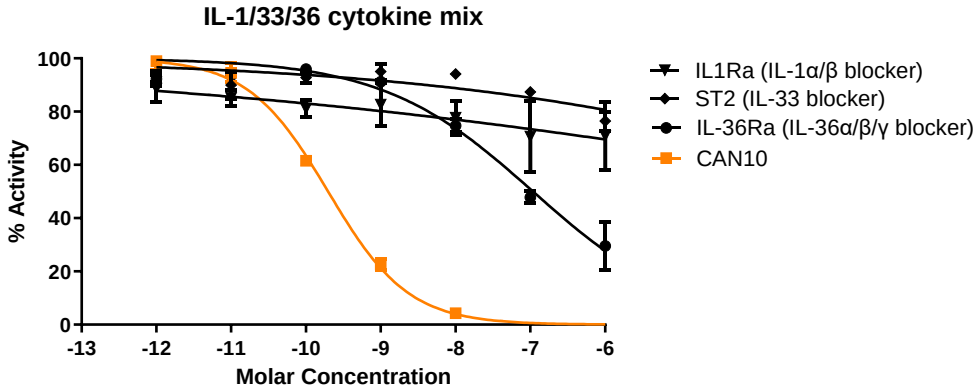
cantargia

CAN10 – OPPORTUNITY IN AUTOIMMUNE/INFLAMMATORY DISEASE

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





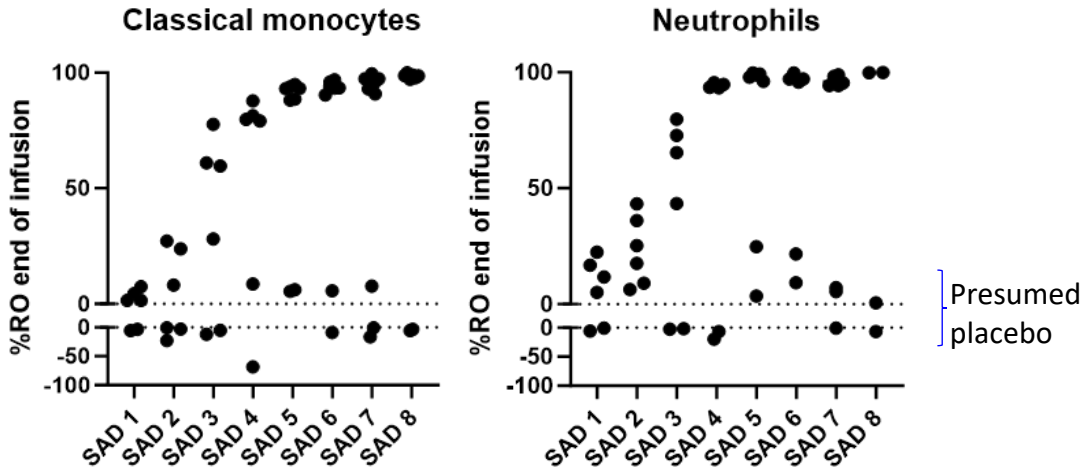
CAN10 first-in-human study - SAD part

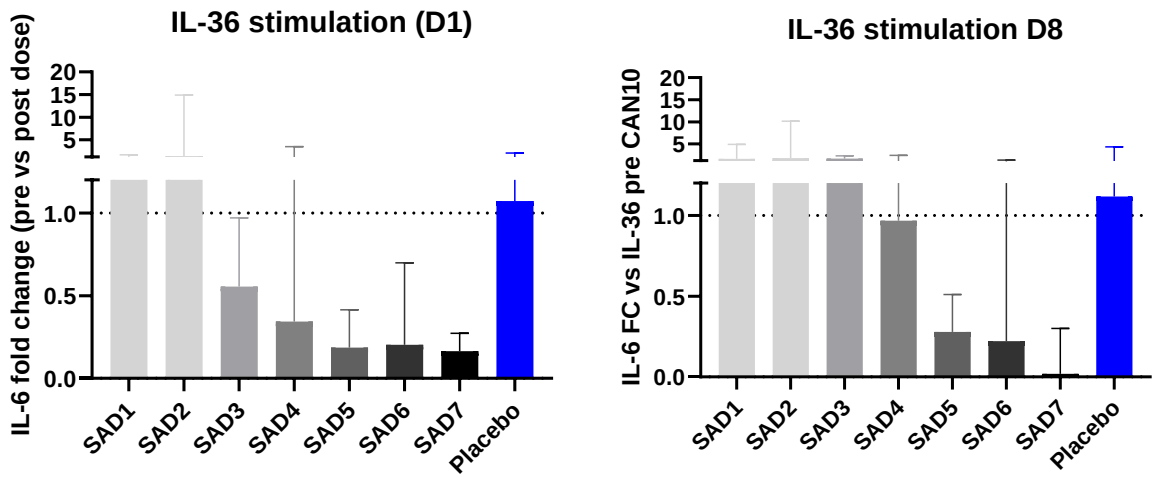
Design

- Blinded, placebo-controlled study
- Nine dose groups from 1 to 400 mg CAN10 incl. 2 patients on placebo in each group

Results

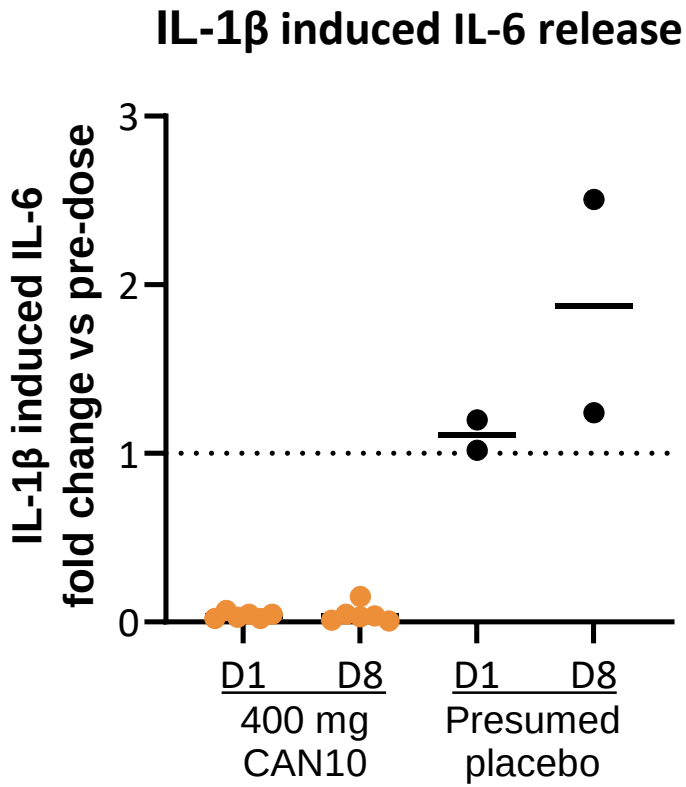
- No safety signals
- Receptor occupancy documented (at Cmax)
- Potent PD effects on IL-1 & IL-36 at Cmax and day 8





AFTER SUCCESSFUL SAD, MULTIPLE DOSING INVESTIGATED IN PSORIASIS PARTICIPANTS

Full blockade of IL-1β signaling at Cmax and at day 8 in subjects treated with CAN10



Whole blood from the participants in the 400 mg cohort stimulated with IL-1β pre-dose, 1 hour post dose (day 1) and day 8. IL-6 levels measured in supernatant

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

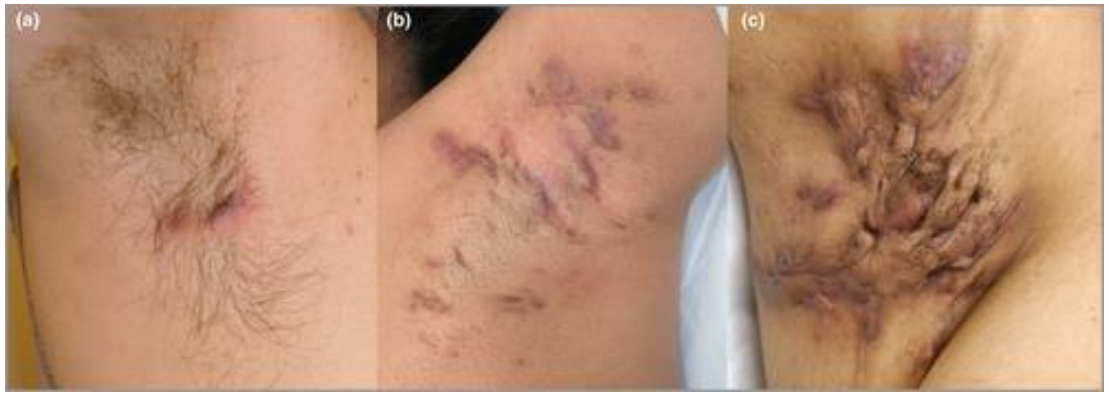
Overview of Hidradenitis Suppurativa (HS)

HS – a severe chronic inflammatory skin disease

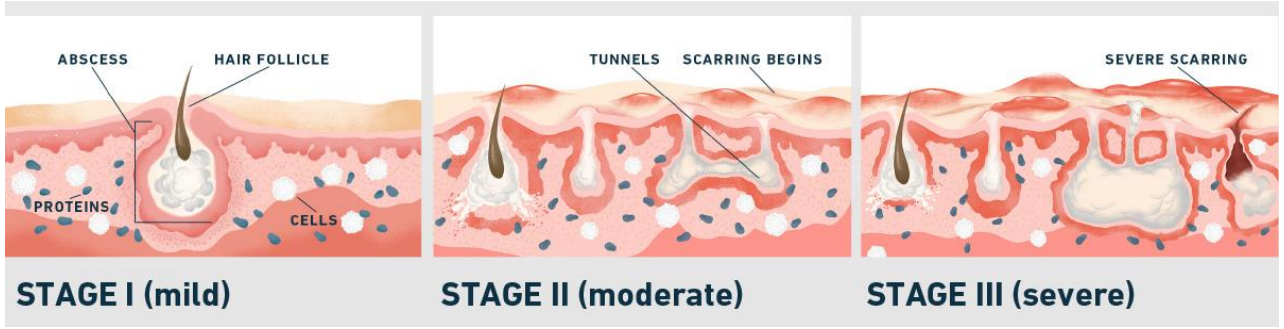
- HS is a diverse disease with several inflammatory components involved in the pathology
- Estimated HS prevalence of 0.7-1.2%

Inadequate current treatments

- Antibiotics
- Steroids
- Anti-TNFα (Humira), anti-IL-17 (Cosentyx)
  - ~50% respond to each in trials
- Huge medical need
  - Non-responders
  - Refractory patients



Hurley stage I (a), II (b) and III (c)[1]



Schematic overview of Hurley stage I-III in HS[2]

1. Lapid et al., BJD 2018; San Diego, CA; 2. AbbVie https://www.nobsabouths.com/what-is-hidradenitis-suppurativa/symptoms accessed 2024-09-05. 3. van der Zee, et al., Dermatology 2018

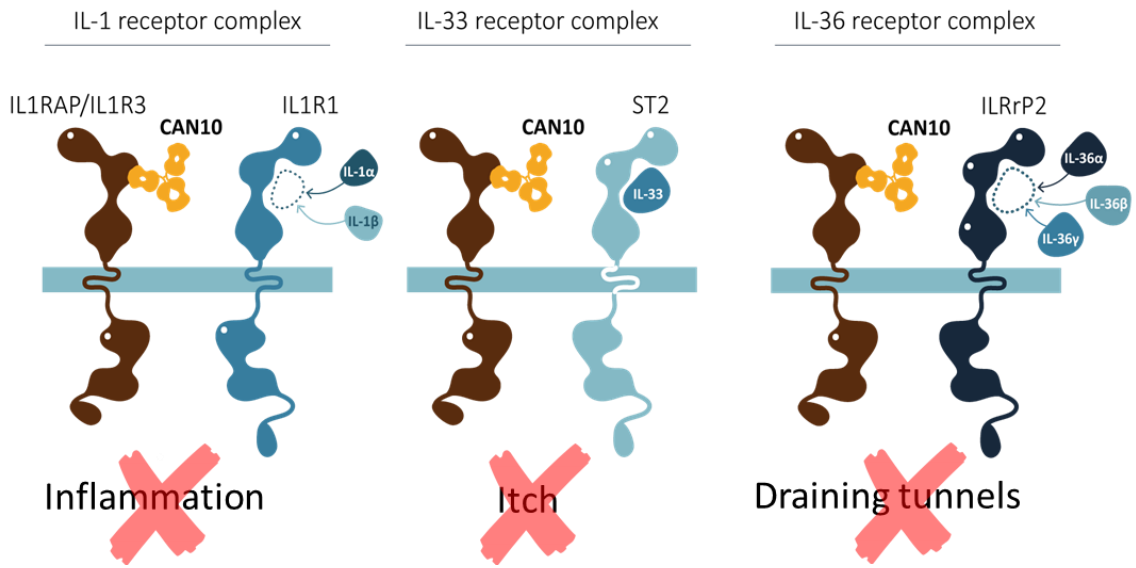
CAN10 for treatment of Hidradenitis Suppurativa (HS)

IL-36R-blockade (spesolimab) elicited positive results on overall disease severity[1]

- Efficacy shown in Phase 2 randomized controlled study (NCT04762277) by changes in iHS4, HASI-R, and HiSCR50, with a particular effect on draining tunnels (dTs)
- Phase 2b/3 study ongoing

Combined IL-1α and IL-1β blockade (lutikizumab) generated high response rates in anti-TNFα refractory patients[2]

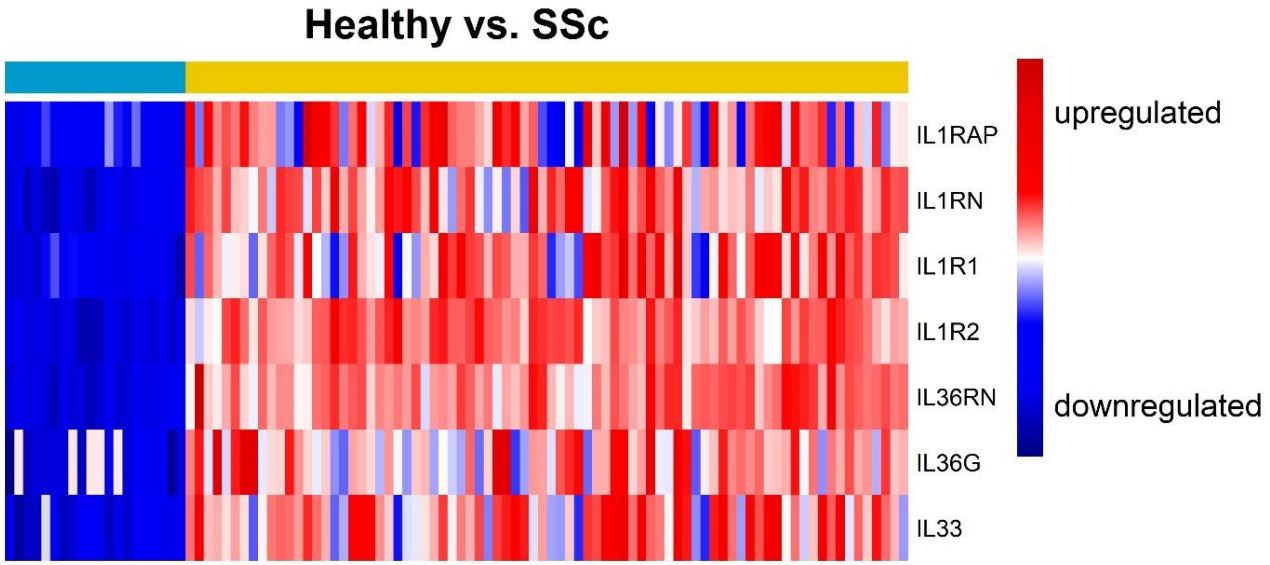
- Efficacy in phase 2 study on primary (HiSCR50) and secondary endpoints (NRS30, skin pain) as well as HiSCR75 at 16 weeks
- Phase 3 study ongoing

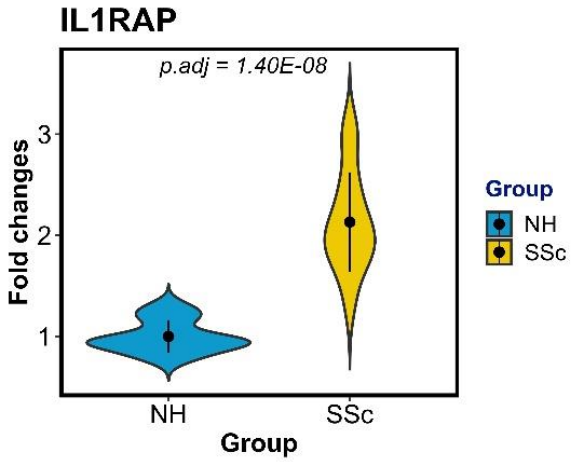


1. Kimball AB, et al. Presented at: American Academy of Dermatology; March 8-12, 2024; San Diego, CA; 2. Alavi A, et al. Br J Dermatol. 2024.

IL1RAP and the IL-1/33/36 pathways are upregulated in skin from Systemic Sclerosis (SSc) patients

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





14 SSc vs. 11 healthy

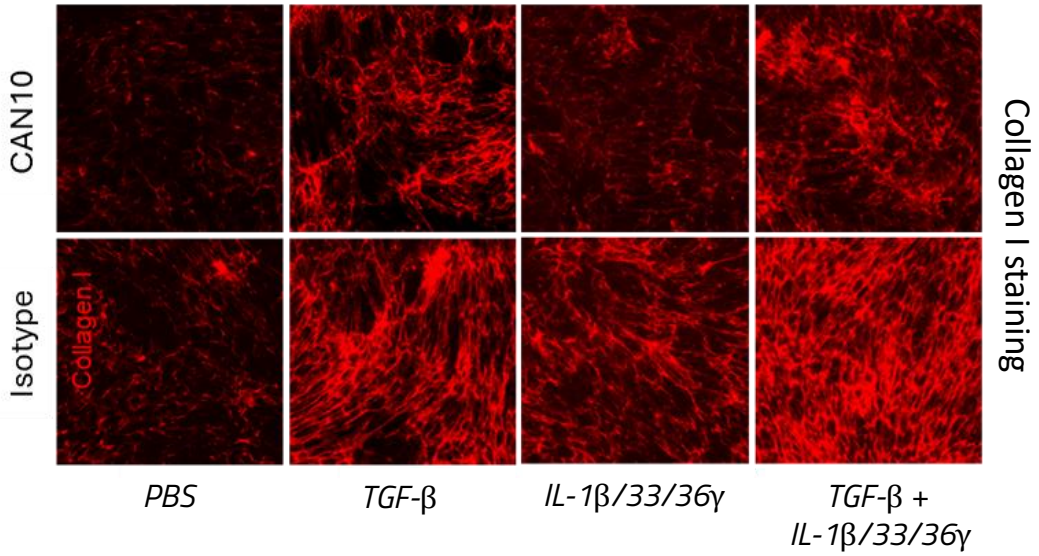
Agilent 2-channel Microarray

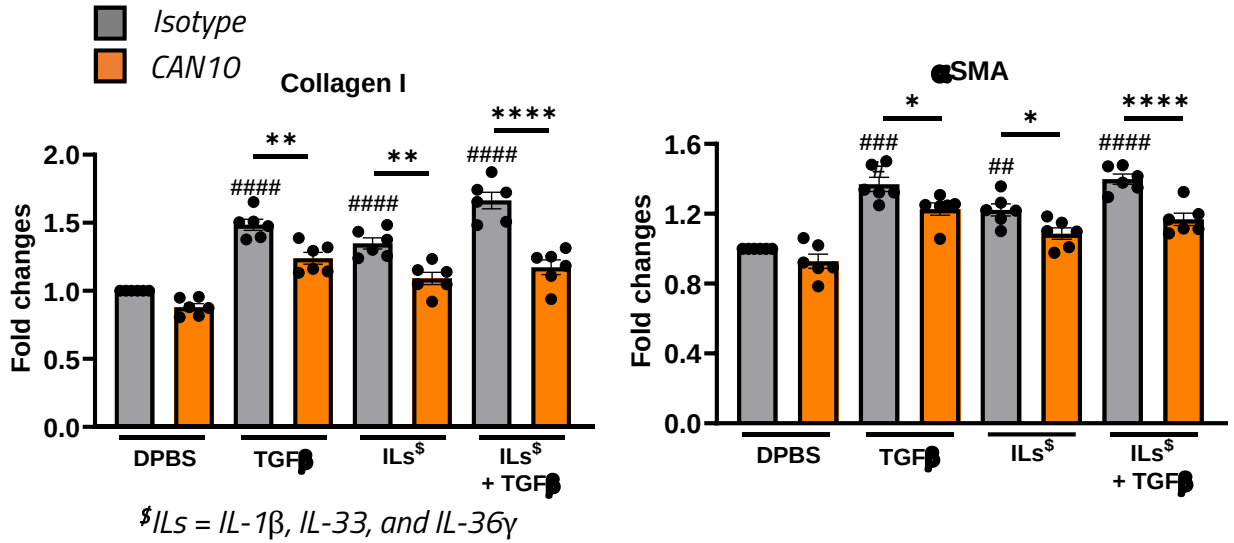
Mahoney et al. 2015 GSE59787
Skaug et al. Ann Rheum Dis 2020. GSE130955

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

antargia

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



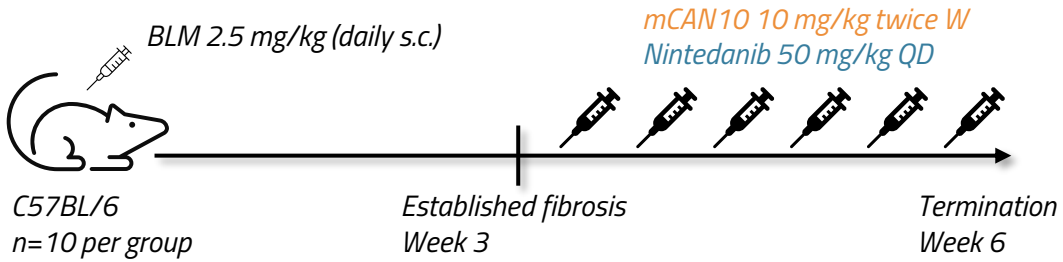


Fibroblasts isolated from SSc patients stimulated with TGFβ or a combination of IL-1β, IL-33, and IL-36γ (abbreviated as ILs) with or without TGFβ in vitro. ILs induced deposition of type I collagen and upregulated the protein levels of αSMA, which could be blocked by CAN10.

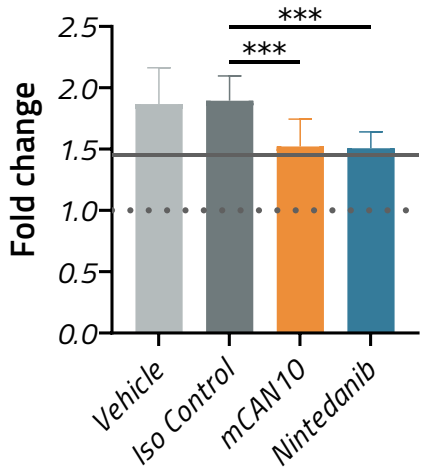
 In collaboration with Jörg Distler, Heinrich-Heine-Universität, Düsseldorf, Germany

Systemic sclerosis – mCAN10 inhibits bleomycin-induced skin fibrosis

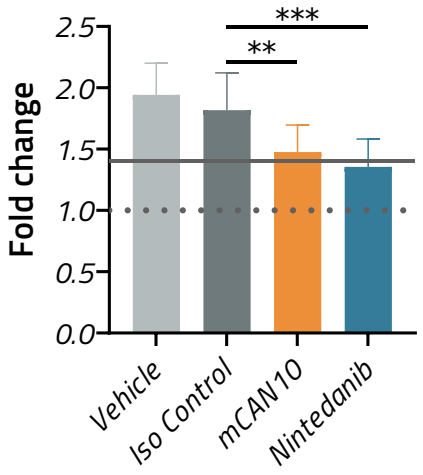
Bleomycin (BLM) model



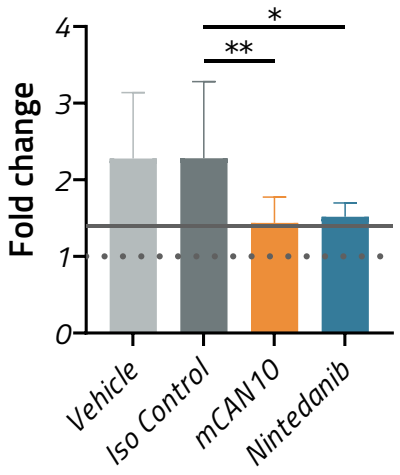
Dermal thickness



Myofibroblast count



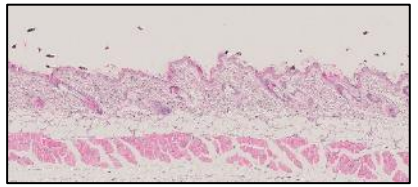
Hydroxyproline



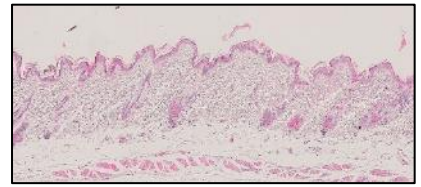
Baseline fibrosis (3 wks)

No fibrosis

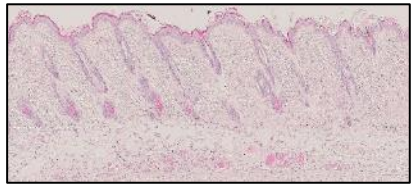
No BLM (6 wks)



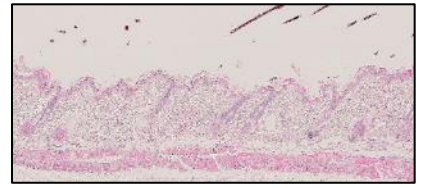
Baseline BLM (3 wks)



BLM (6 wks) Iso Control

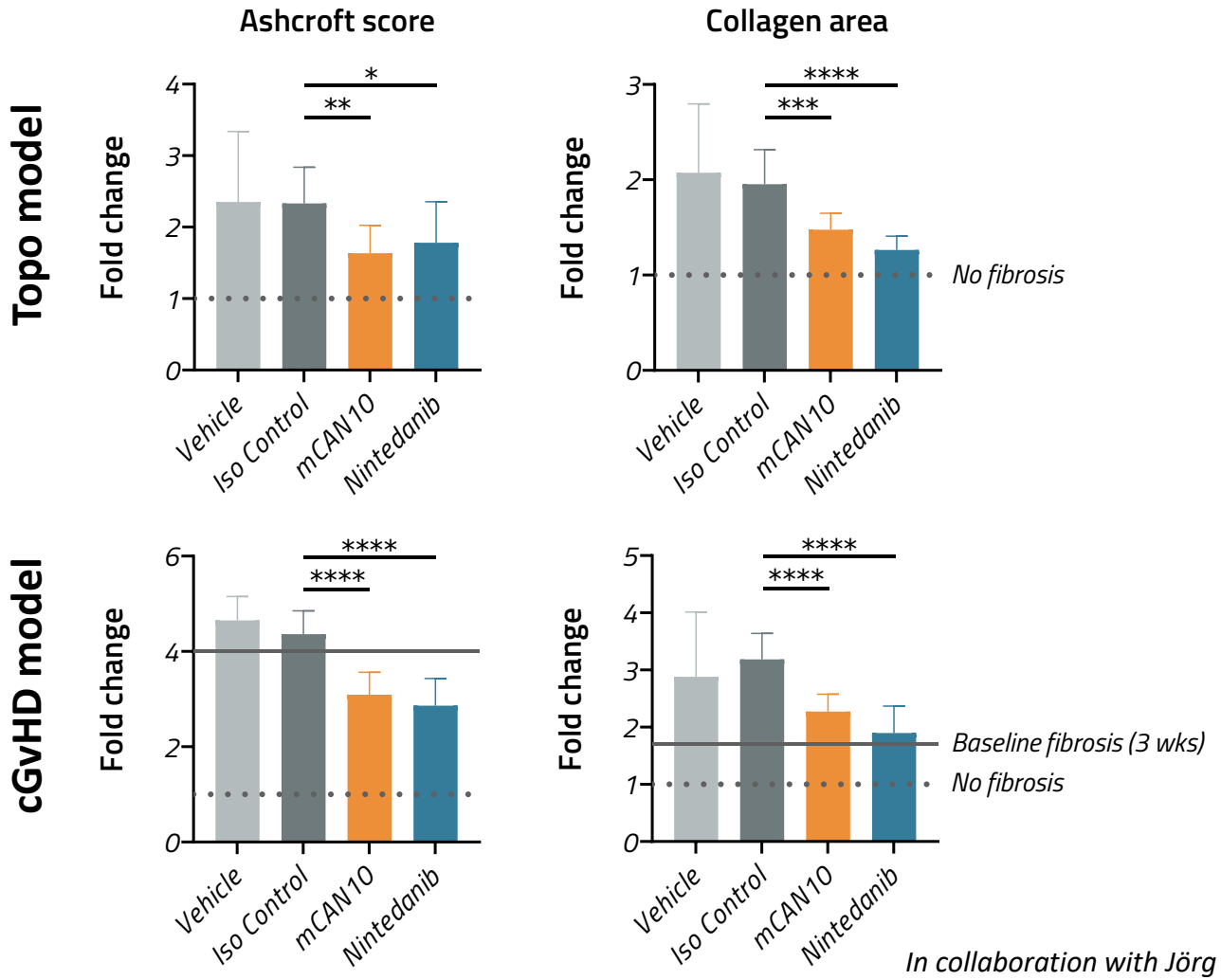


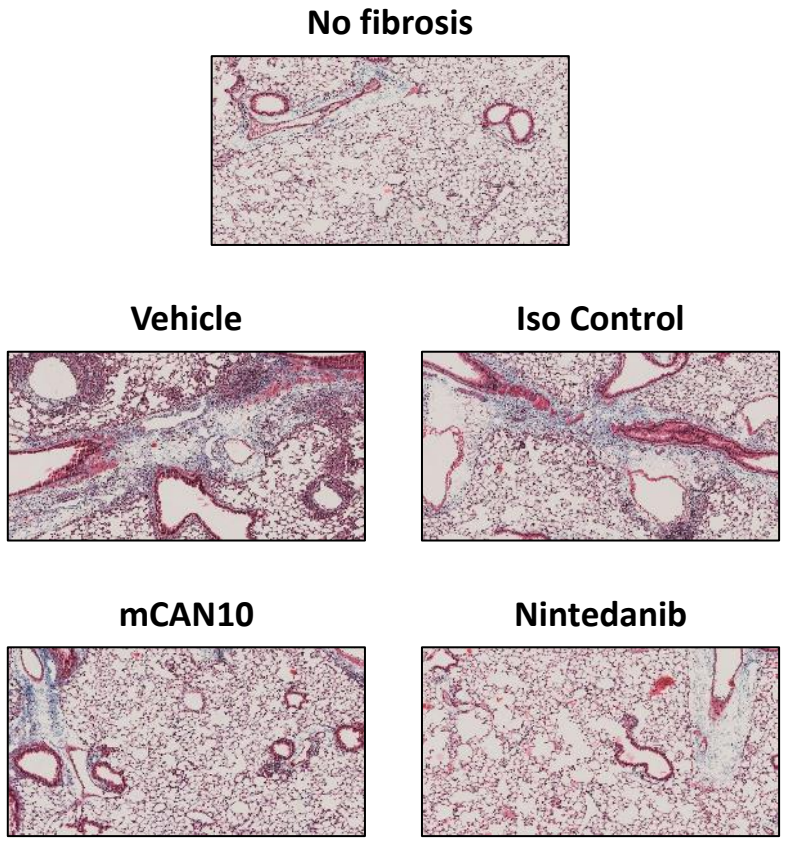
BLM (6 wks) mCAN10



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

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

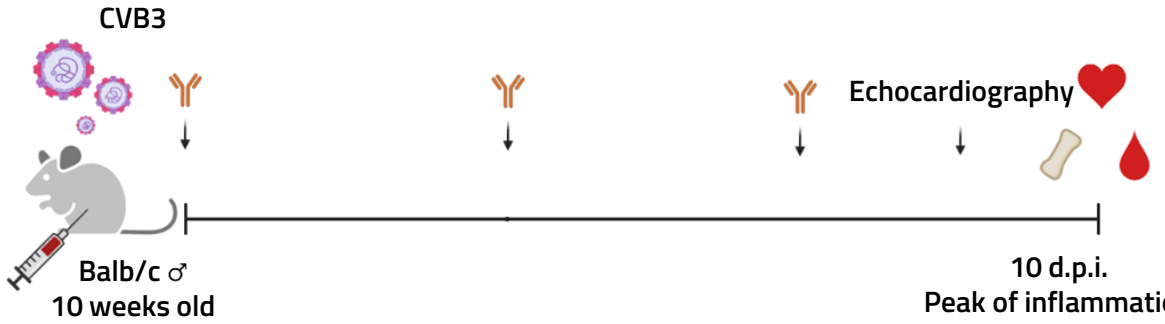


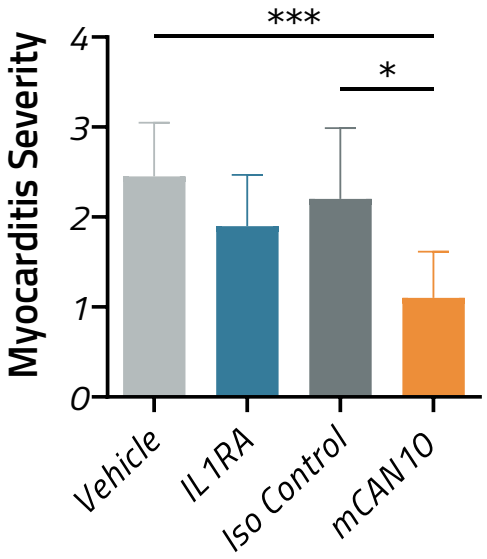


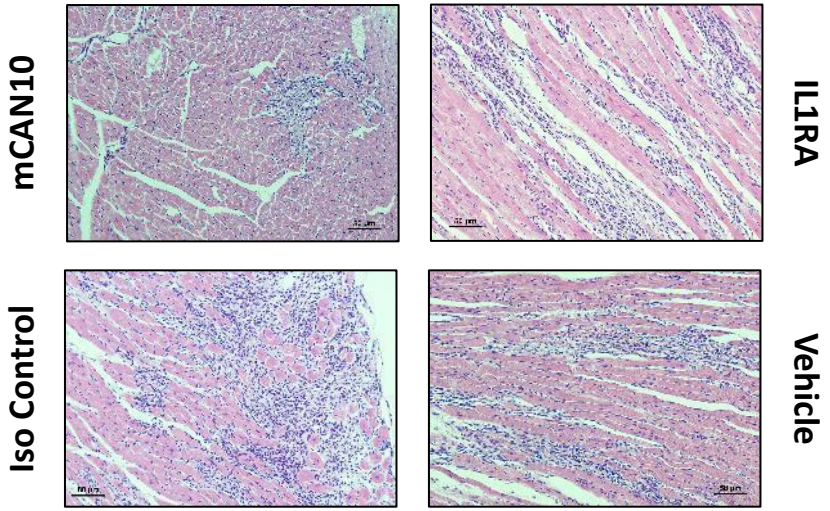
In collaboration with Jörg Distler, Heinrich-Heine-Universität, Düsseldorf, Germany

Viral myocarditis – mCAN10 reduces disease severity

CVB3 myocarditis experimental design







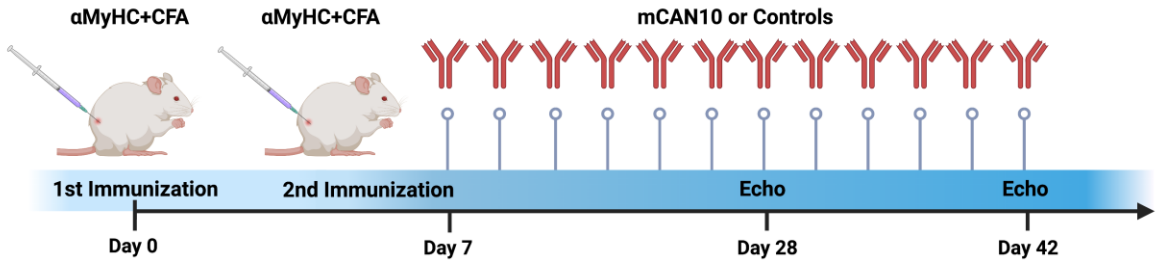
→ mCAN10 reduced disease severity, based on histological scoring of heart sections, and preserved heart function

→ mCAN10 also reduced inflammatory leukocyte populations in the heart tissue

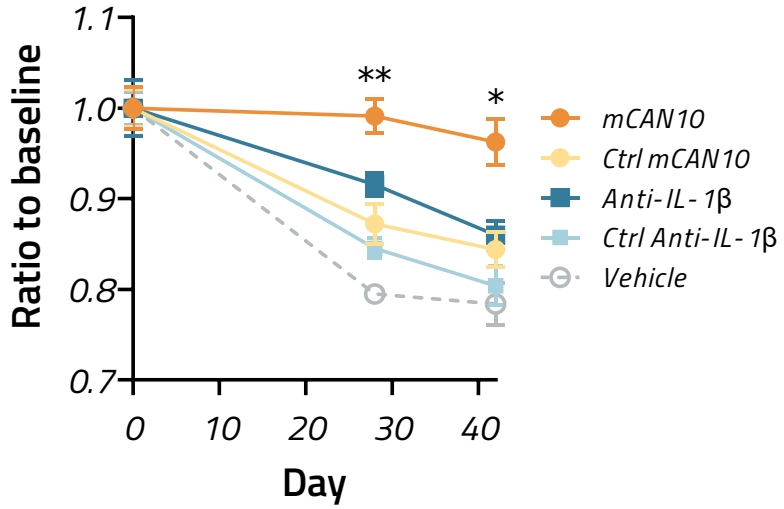
CVB3 – Coxsackievirus B3; IL1RA – IL-1 Receptor Antagonist (blocks IL-1α/IL-β signaling)
n=10 per group

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

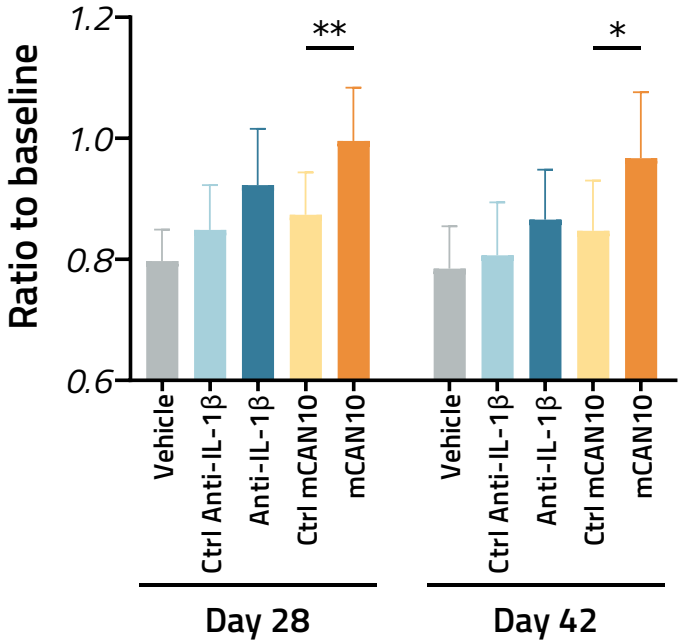
Experimental autoimmune myocarditis – mCAN10 improves heart function



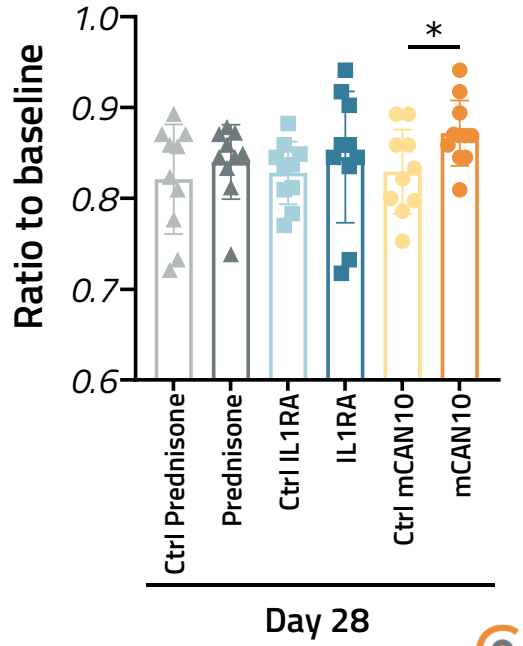
Left Ventricual Ejection Fraction (LVEF)



LVEF (treatment from day 7)



LVEF (treatment from day 7)



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

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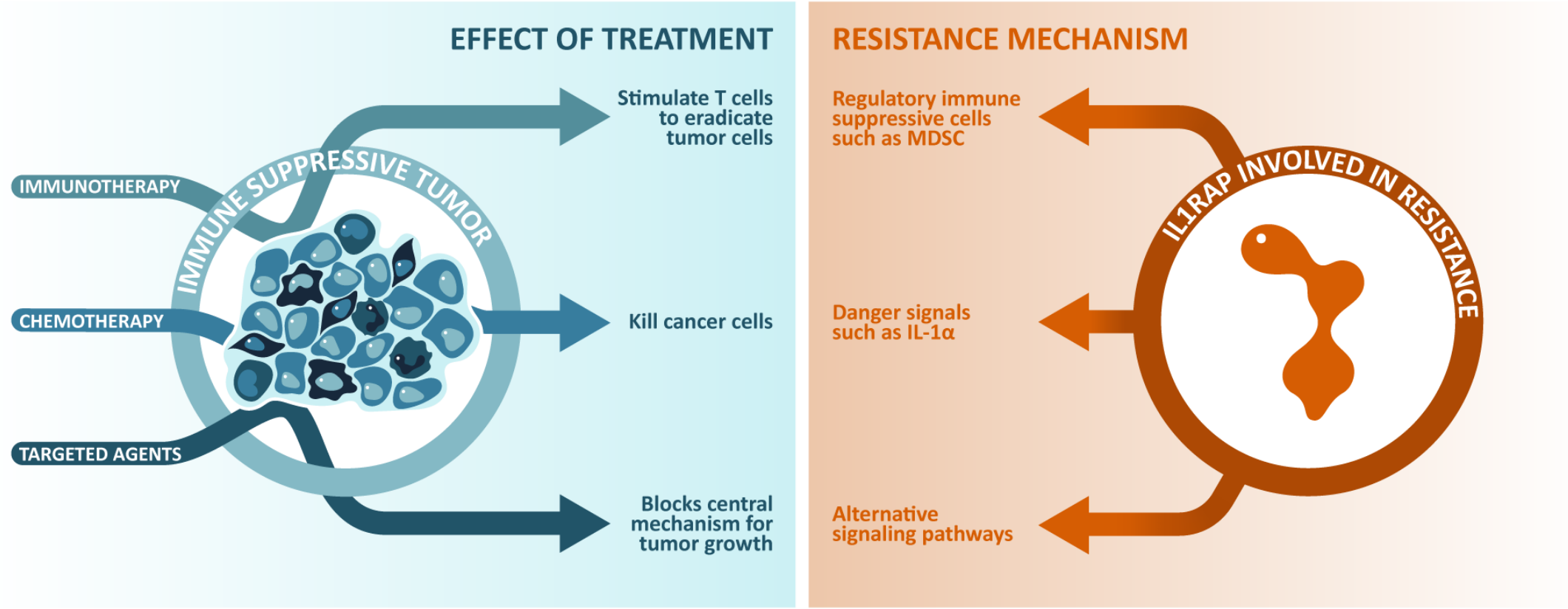
NADUNOLIMAB (CAN04) OVERVIEW

Cantargia – Strategy to improve current cancer therapies

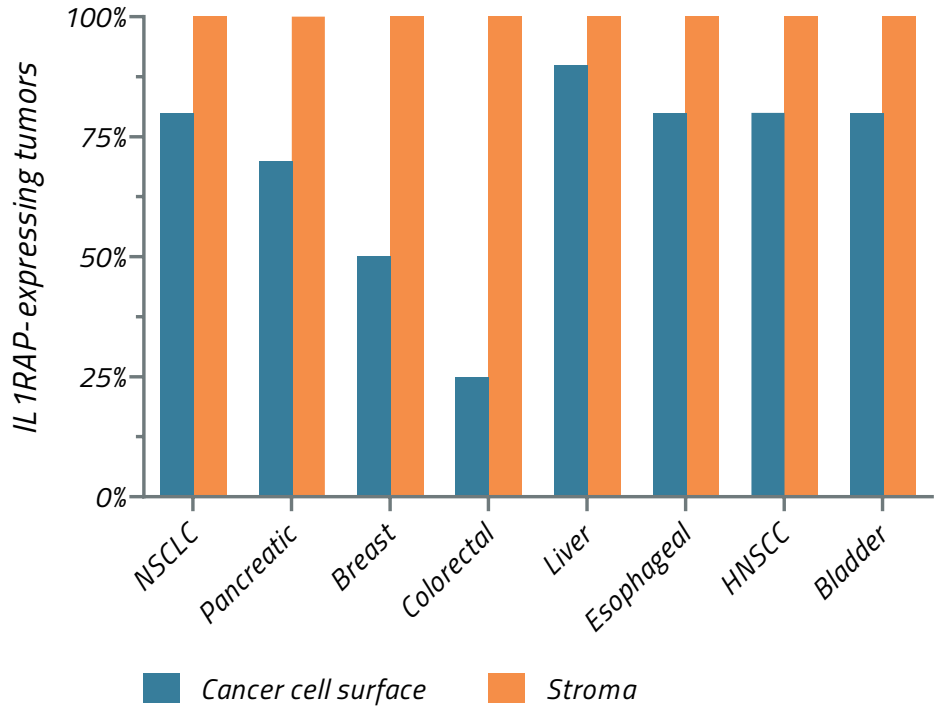


IL1RAP – A NOVEL TARGET WITH SEVERAL OPPORTUNITIES; CURRENT FOCUS ON SYNERGISTIC COMBINATIONS

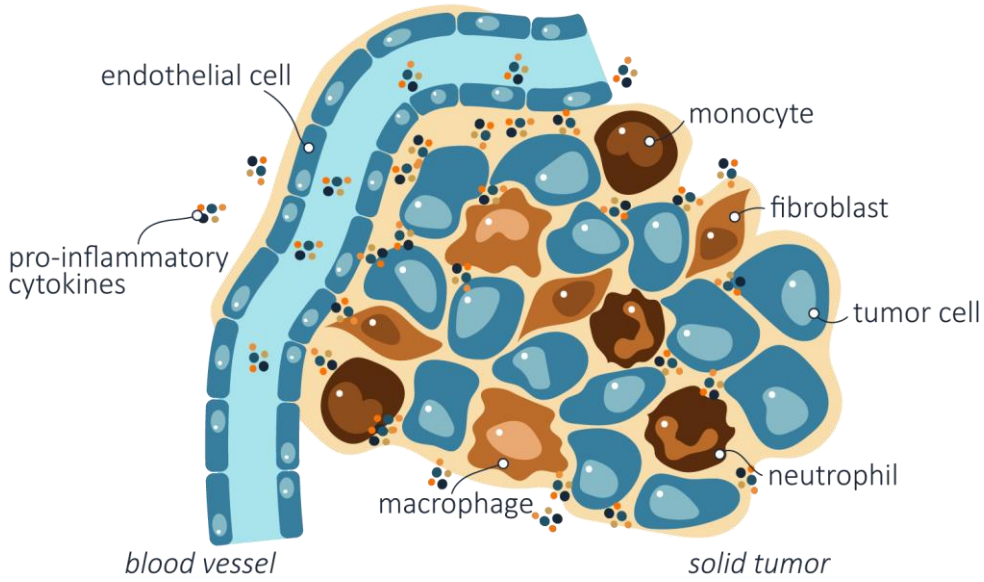
MDSC – Myeloid-Derived Suppressor Cells

IL1RAP overexpressed in most solid tumors

IL1RAP EXPRESSION IN SOLID TUMOR TYPES



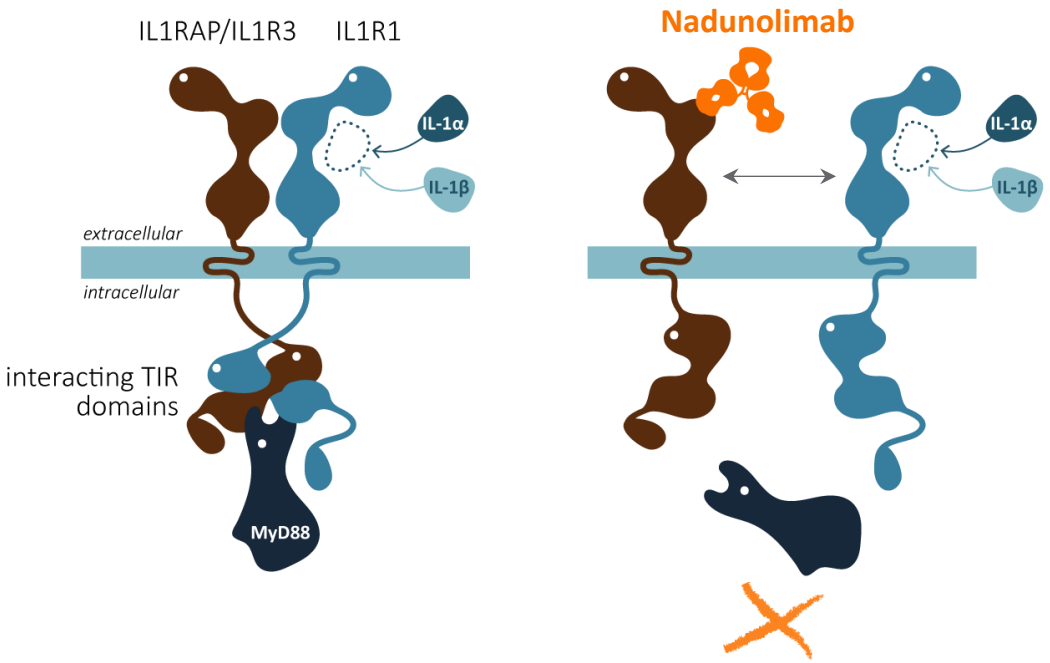
SEVERAL TUMOR-PROMOTING CELLS EXPRESSING IL1RAP IN THE TUMOR MICROENVIRONMENT

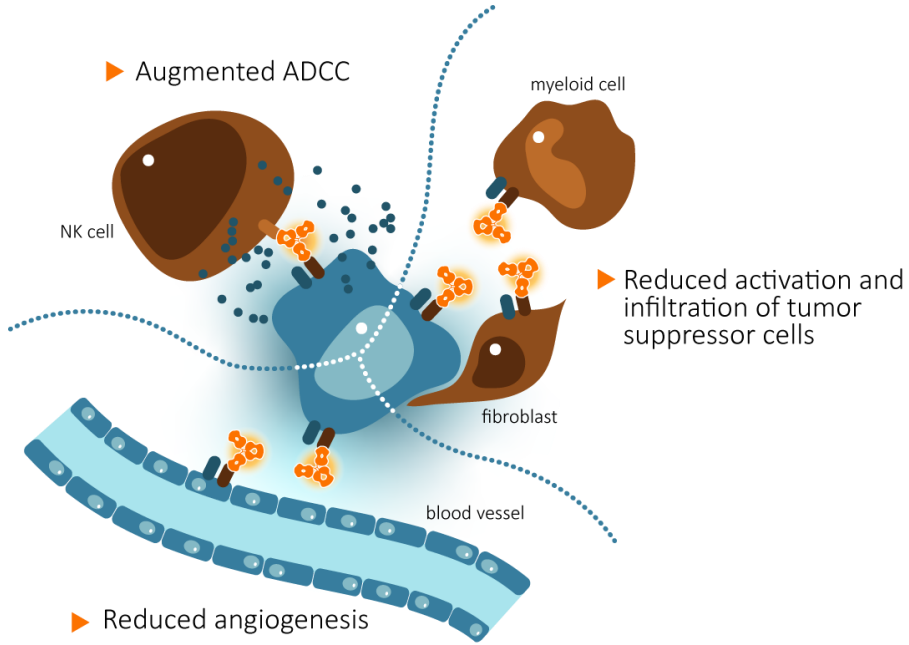


IL1RAP – DISTINCTLY OVEREXPRESSED IN TUMORS; LOW EXPRESSION IN NORMAL TISSUE

HNSCC – Head and Neck Squamous Cell Carcinoma

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

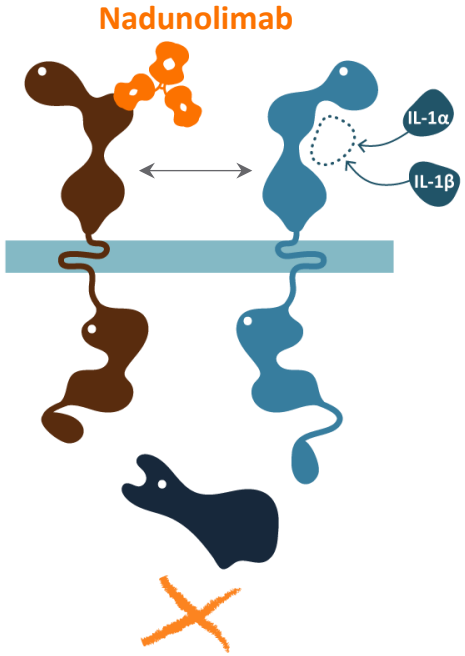


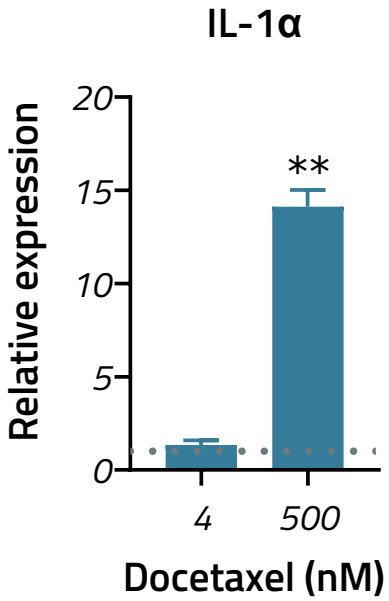


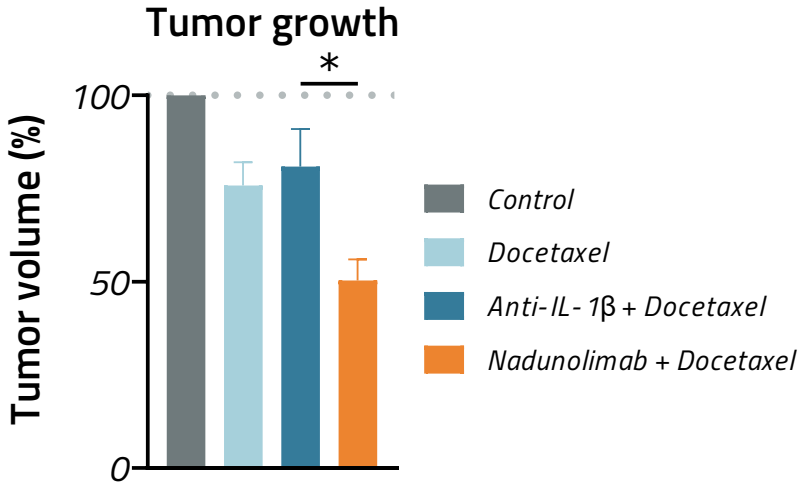
NADUNOLIMAB COUNTERACTS IMMUNE SUPPRESSION AND POTENTIATES THERAPY

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**

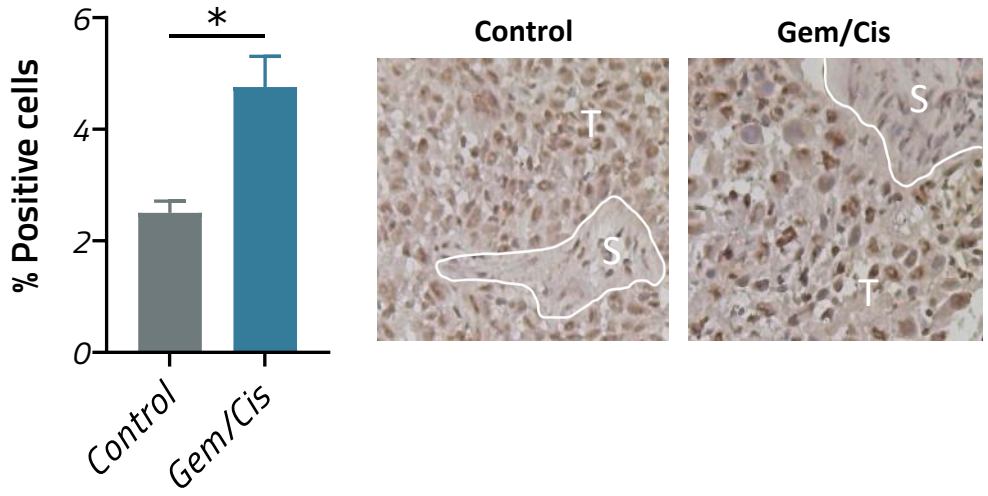
NADUNOLIMAB INCREASES DOCETAXEL EFFICACY IN CONTRAST TO IL-1β BLOCKADE

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

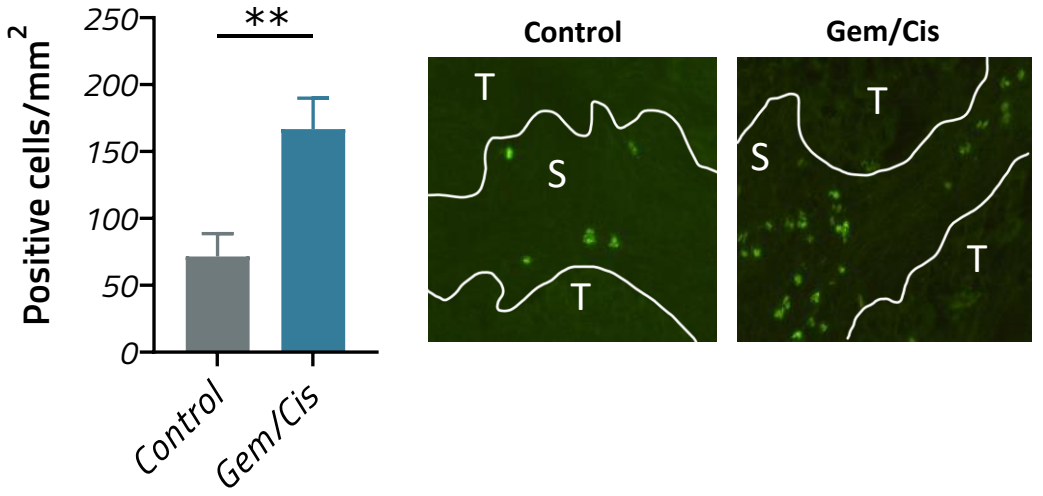
Chemotherapy induces IL-1α and IL-1β in the tumor

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)

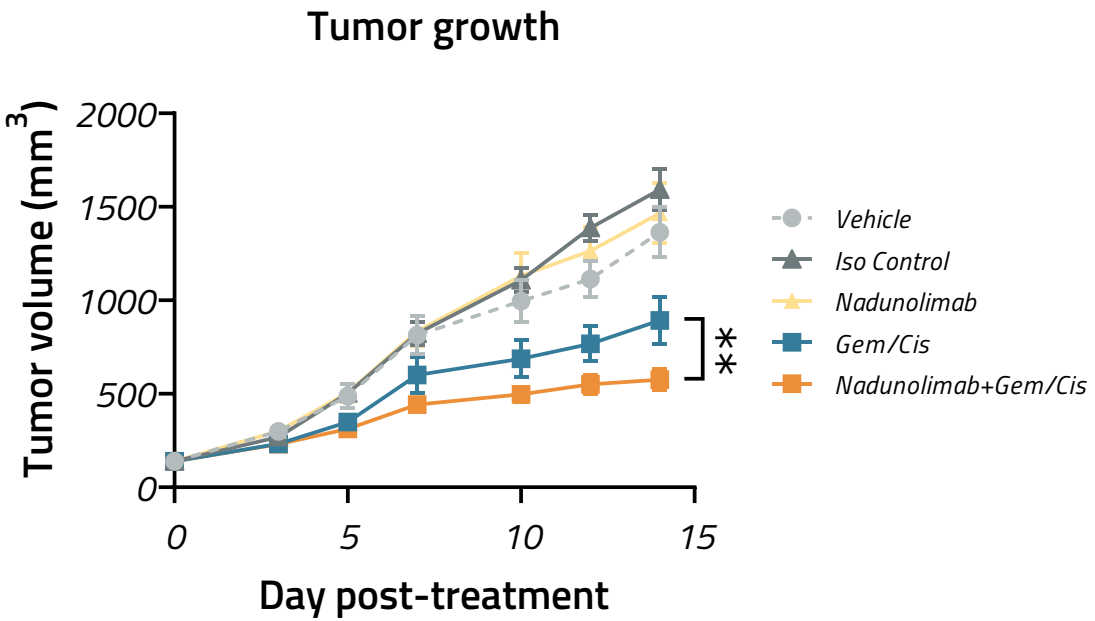


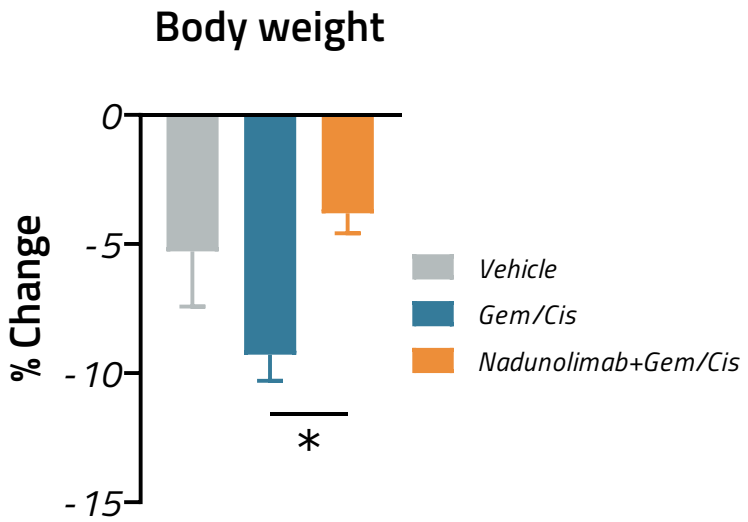
→ **Gem/Cis also induces release of IL-1β-converting enzyme (ICE) by stromal cells**

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

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

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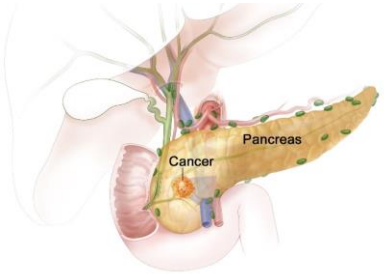
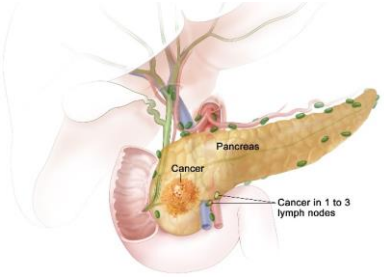
NADUNOLIMAB (CAN04) CLINICAL RESULTS

Pancreatic Cancer (PDAC) – Staging and treatment

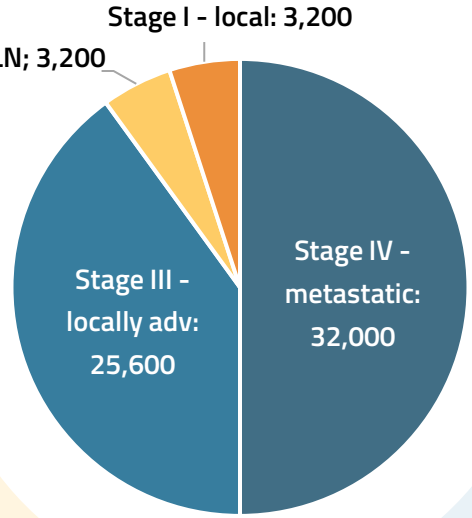
Expected number of cases US 2023: 64,000

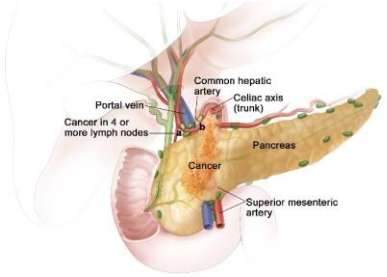


Stage I

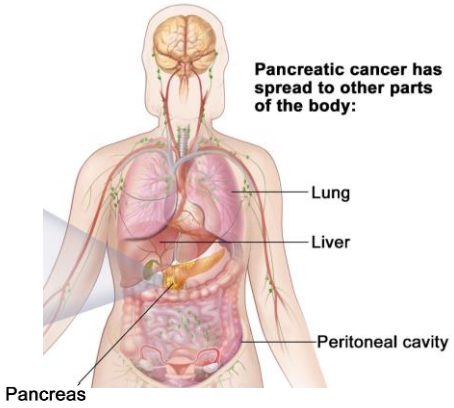


Stage II





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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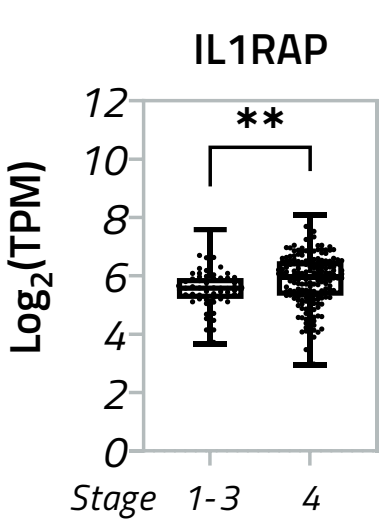
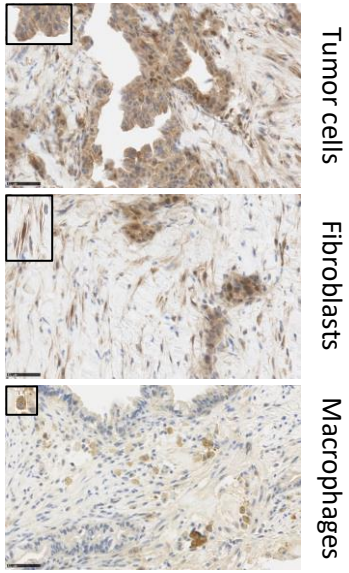
Pancreatic Cancer – IL1RAP linked to poor prognosis

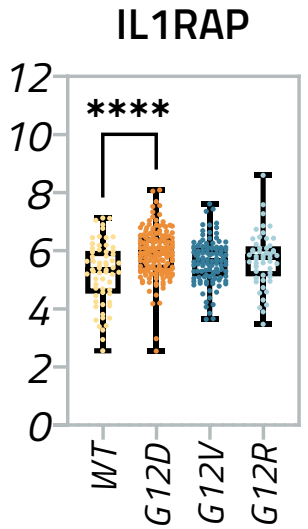
IL1RAP IN PDAC

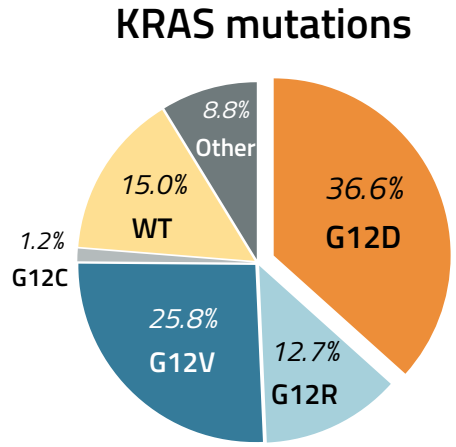




- IL1RAP levels increase with tumor stage
- IL1RAP expressed on both tumor cells, cancer-associated fibroblasts and macrophages in tumor microenvironment
- High IL1RAP correlates with lower efficacy after 1st line Gem/Abraxane

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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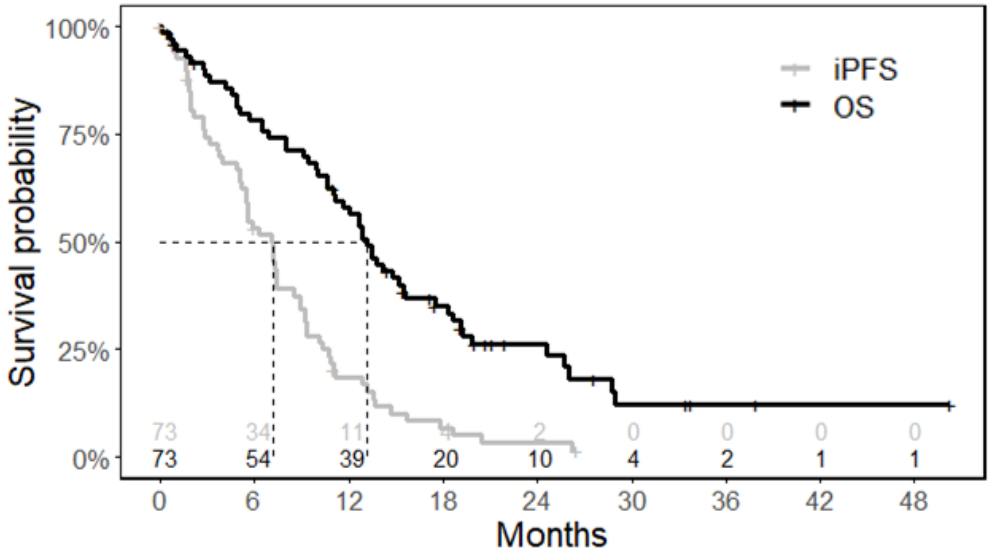
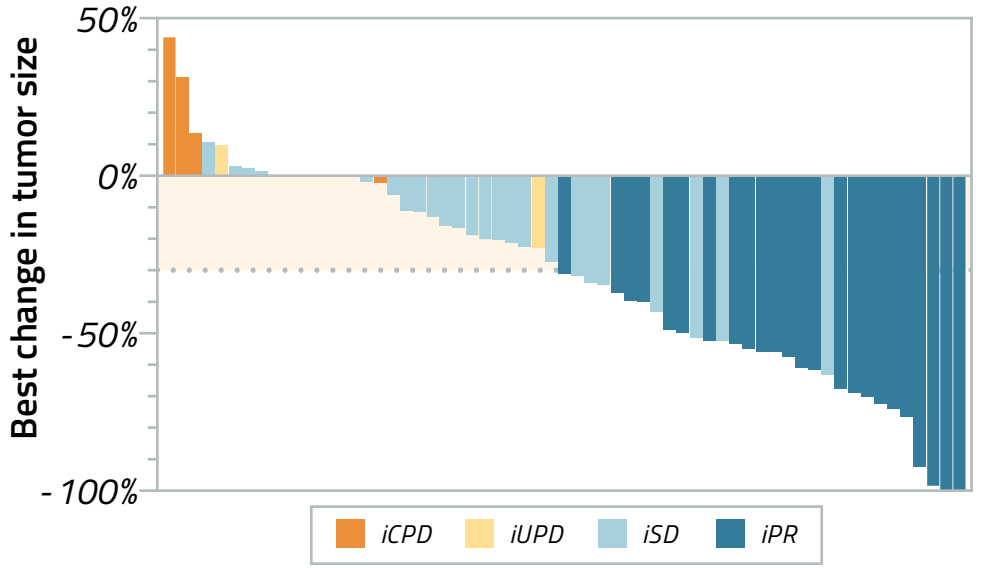
Pancreatic Cancer – Positive interim data in 1st line patients

OS and iPFS for mITT patients



Best responses according to iRECIST



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

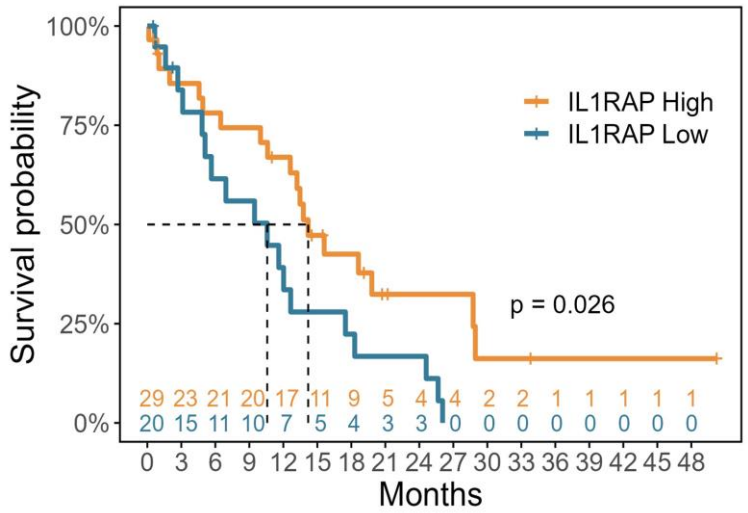
- 33% response rate with long OS and iPFS
  - Additional 5 (7%) patients had on-treatment benefit beyond progression
- Promising OS (13.2 mo), iPFS (7.2 mo) and DCR (71%); 2 patients still on treatment

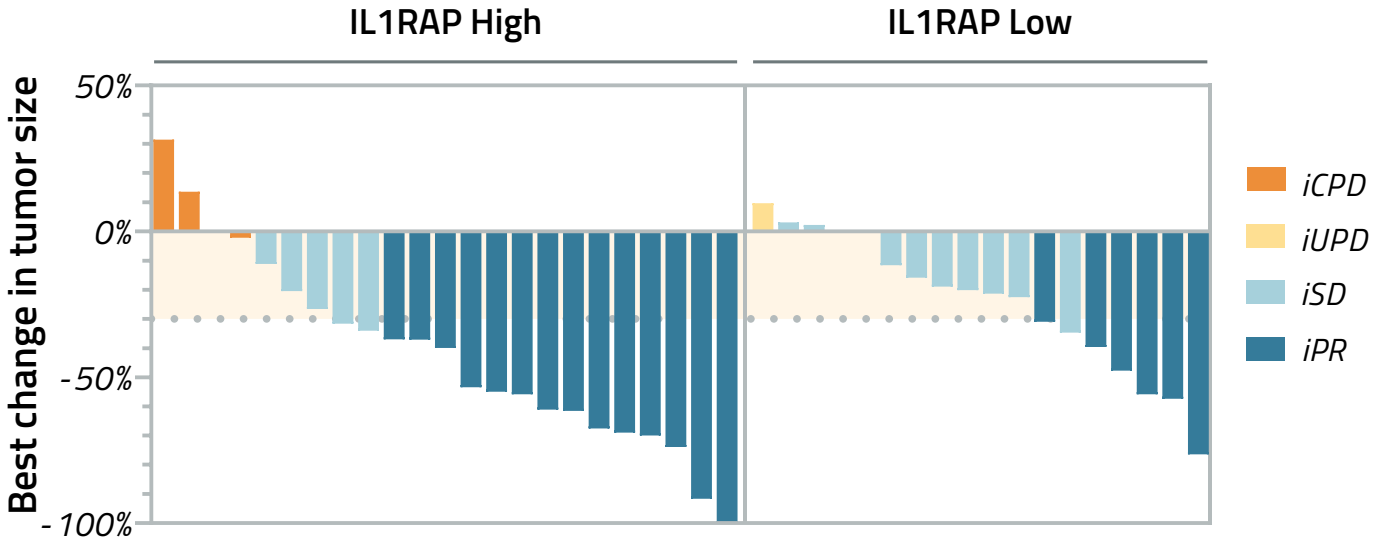
PFS AND OS LONGER THAN EXPECTED GIVEN HISTORICAL CONTROL IN PDAC

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)

Pancreatic cancer – Efficacy and IL1RAP level

OS by IL1RAP subgroup





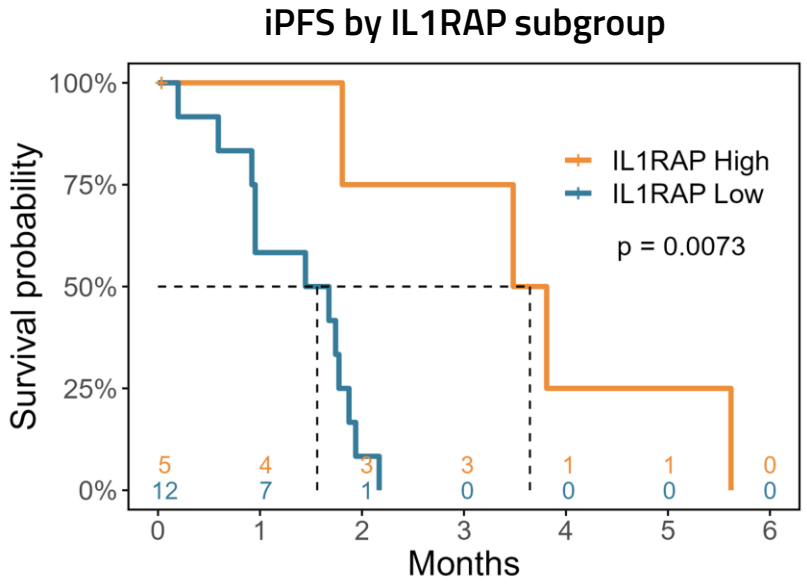
Efficacy analysis for IL1RAP High (n=29) vs IL1RAP Low (n=20) PDAC patients (1st line, combination with Gem/Abraxane):

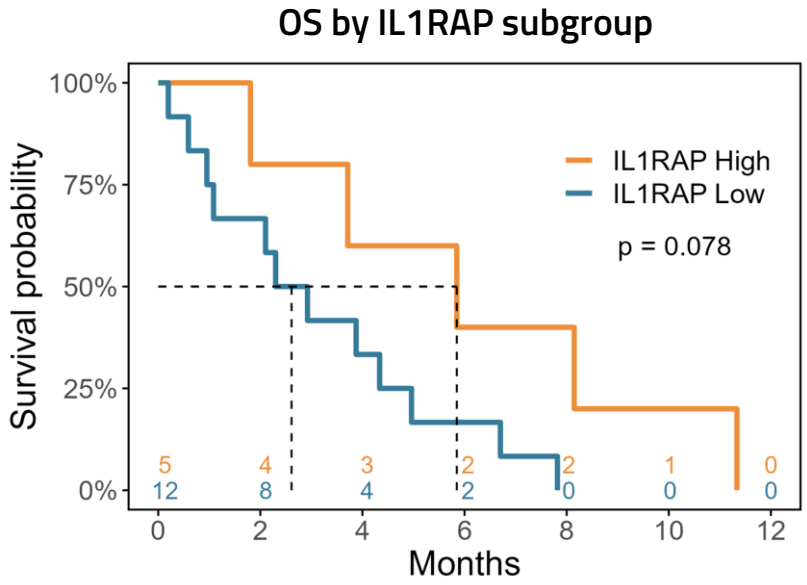
- → Significantly prolonged OS in ILRAP High vs IL1RAP Low patients (14.2 vs 10.6 mo; p=0.026)
- → Deeper and more durable responses in IL1RAP High subgroup: 11 patients had 50% or more tumor size decrease

IL1RAP HIGH PATIENTS SHOW THE STRONGEST BENEFIT

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

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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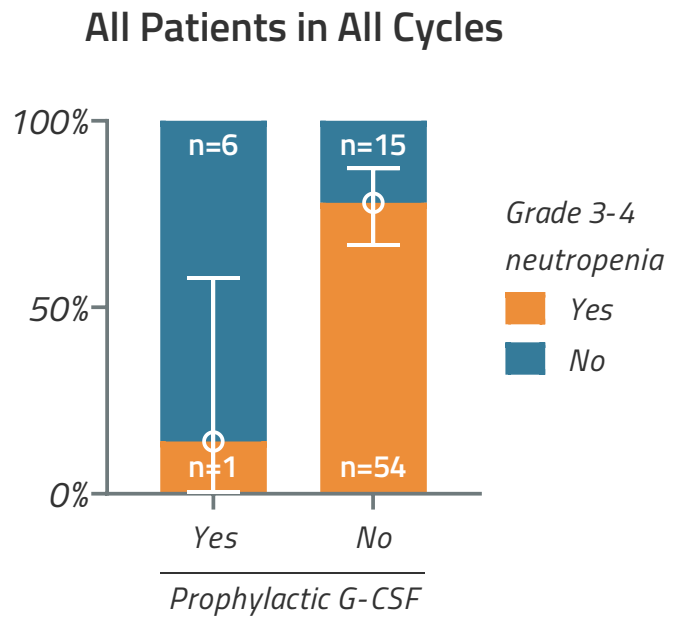
PDAC – Safety profile is manageable and supports MOA

- 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%

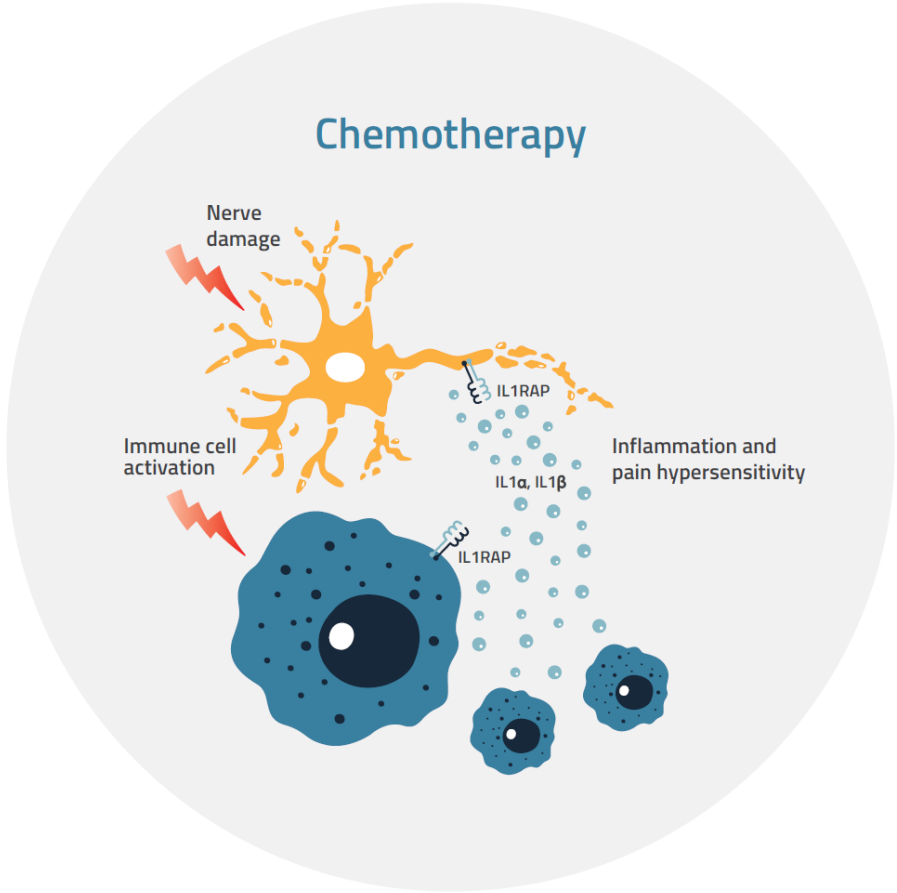


G-CSF PROPHYLAXIS IMPLEMENTED IN FUTURE TRIALS;
POTENTIAL REDUCTIONS OF SOME SIDE EFFECTS TO BE DOCUMENTED IN RANDOMIZED TRIALS

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.

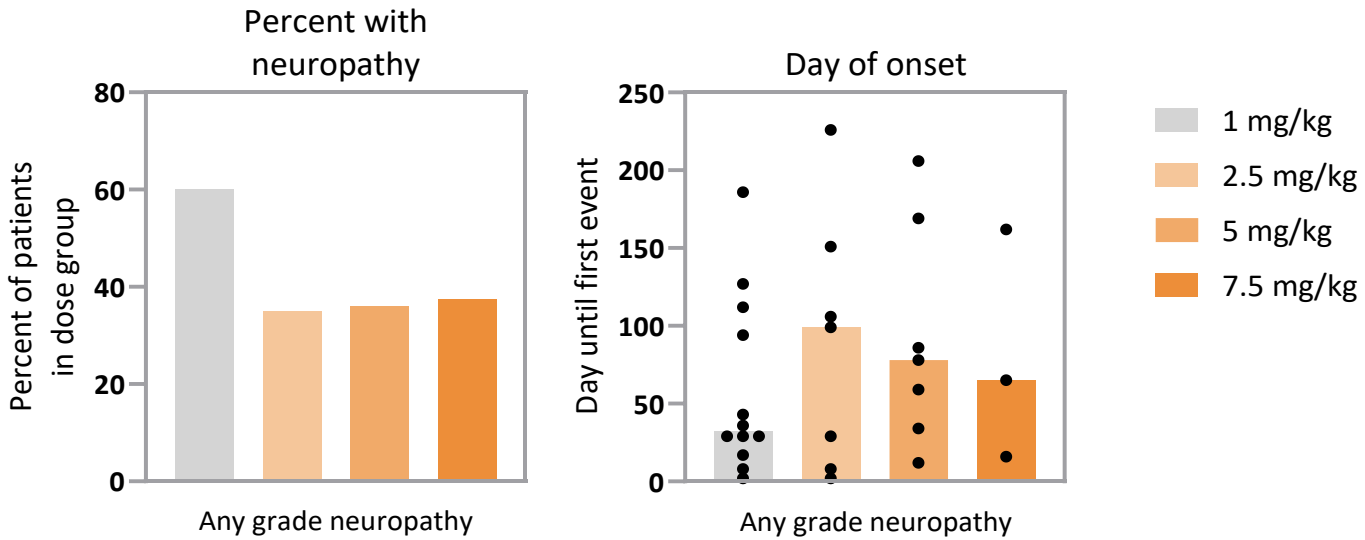
Nadunolimab and alleviation of neuropathy

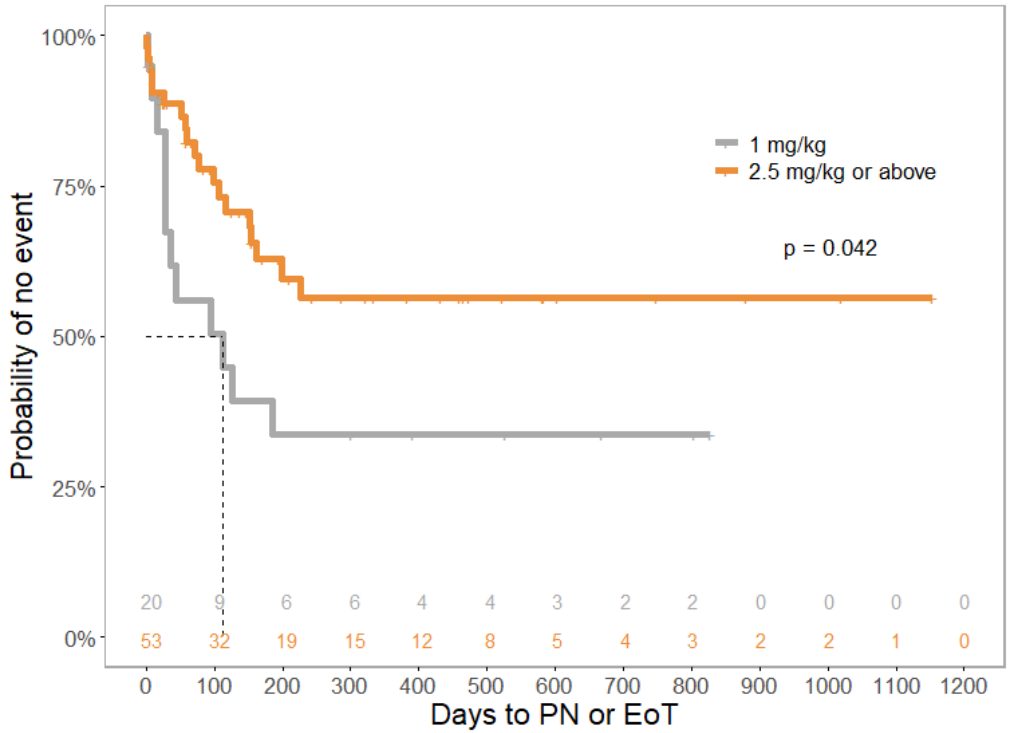
- Chemotherapy induce neuropathy by several pathways including IL-1 (neuroinflammation)
- Nadunolimab, phase 2 data in PDAC with Gem/nabP
  - lower Grade 3-4 peripheral neuropathy than expected from historical controls (1% vs 17%).
- Correlation between nadunolimab dose level and protective effect
-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



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

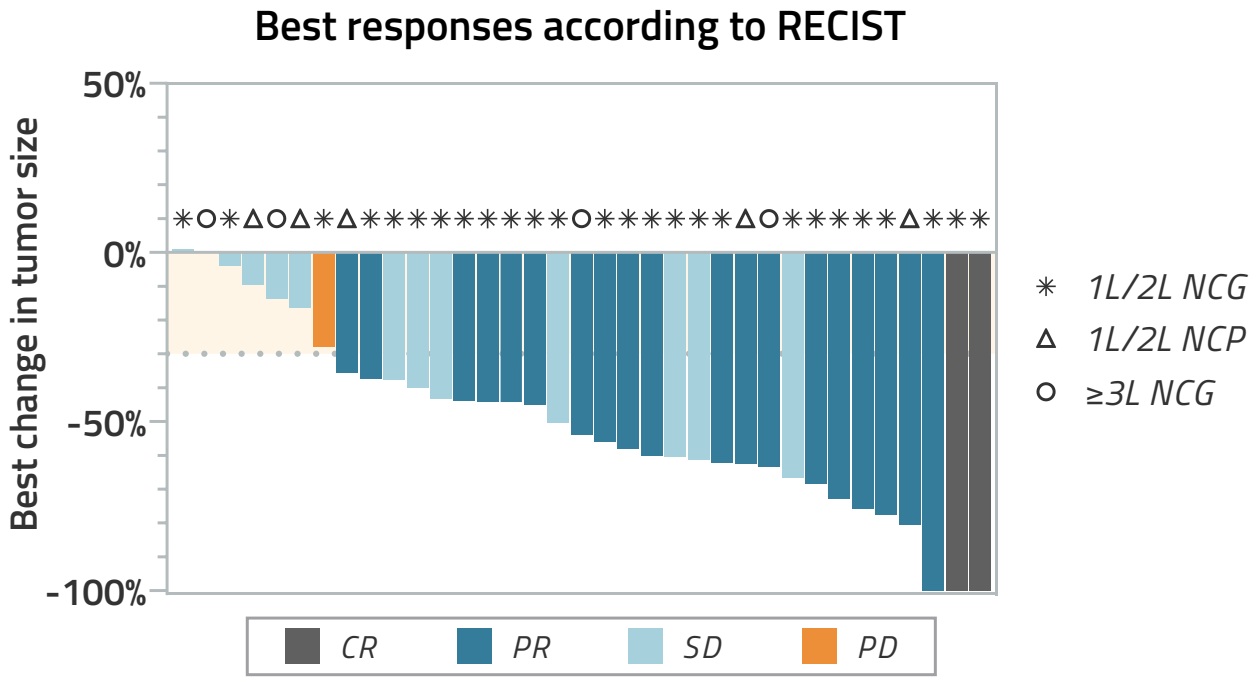
Nadunolimab and alleviation of neuropathy





CORRELATION BETWEEN NADUNOLIMAB DOSE LEVEL AND DECREASE IN NEUROPATHY

Non-small cell lung cancer (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

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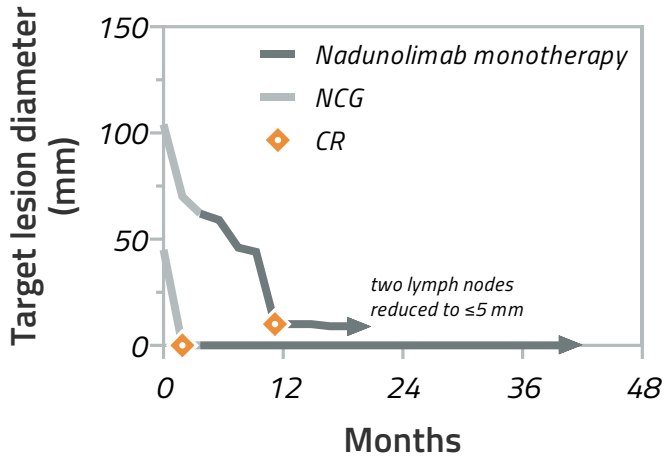
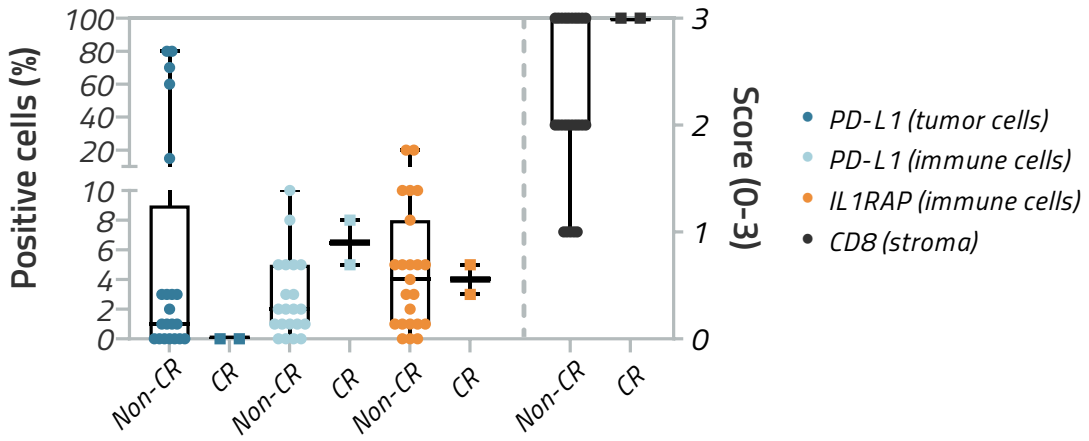
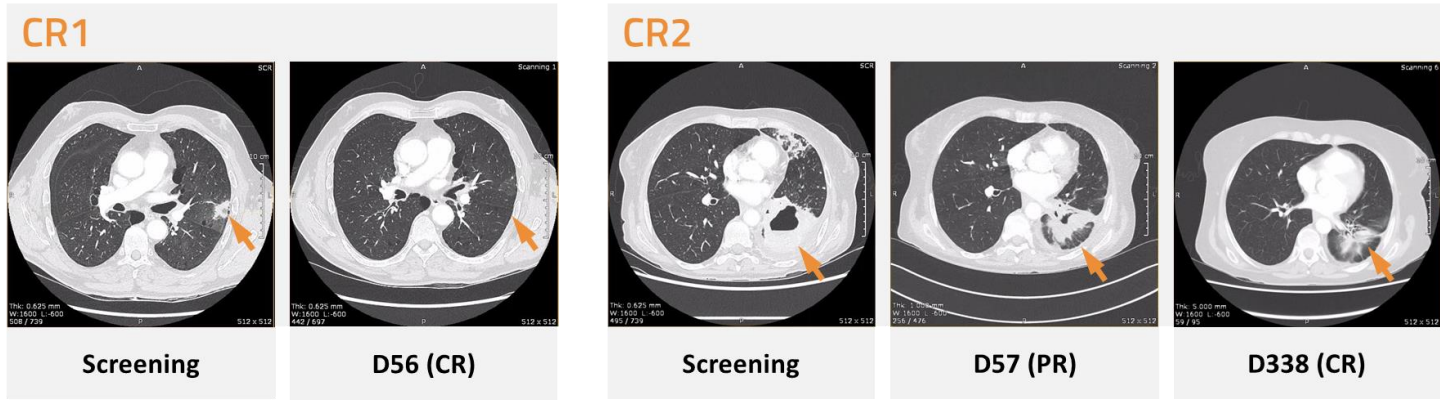
NSCLC – Complete responders with distinct biomarker profile







- 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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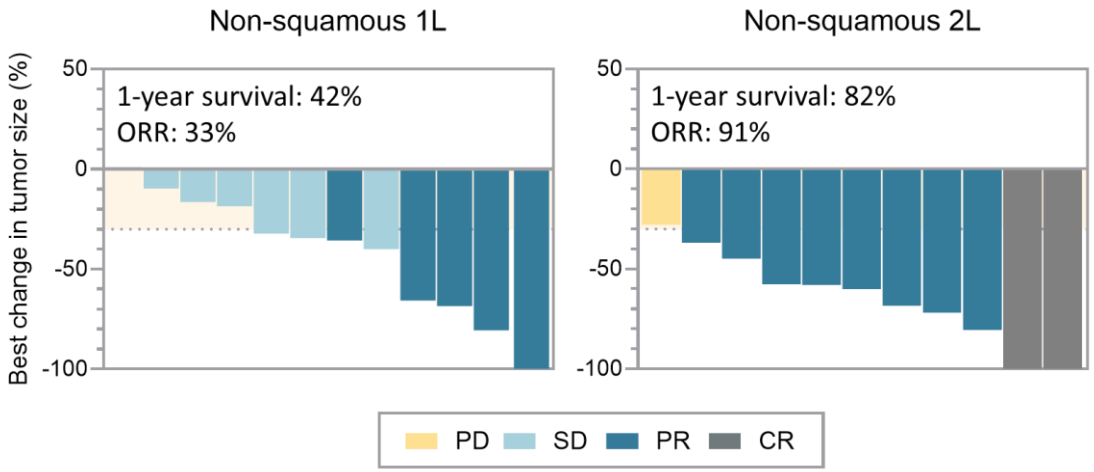
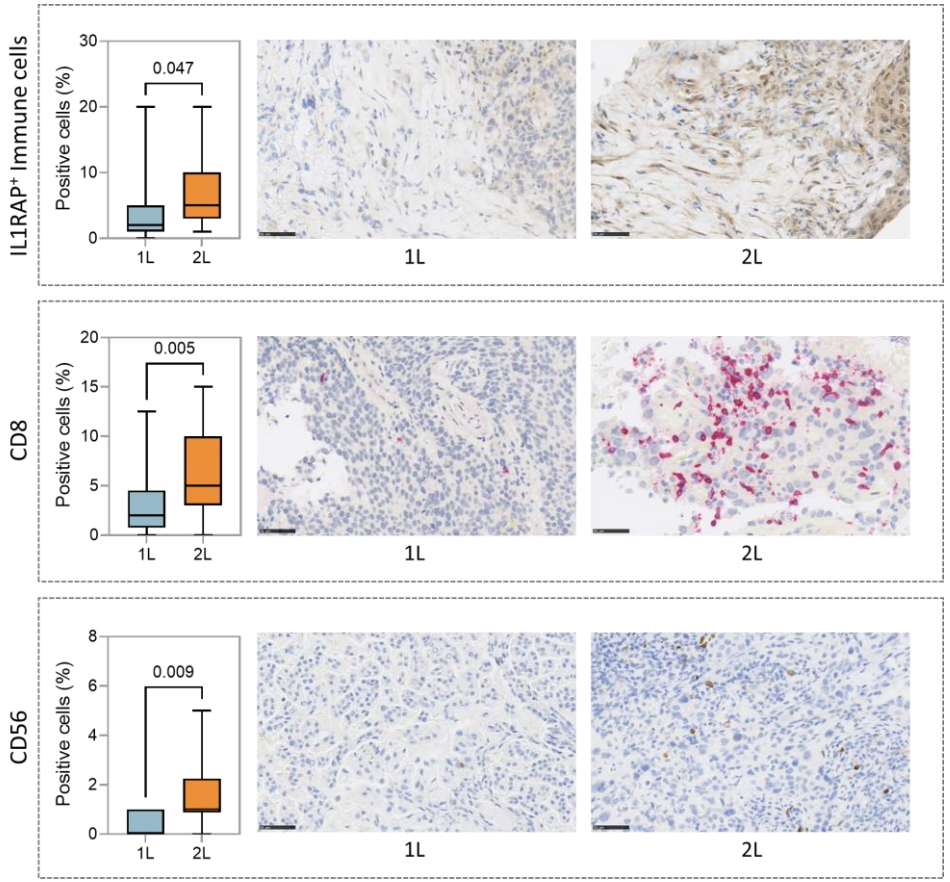
NSCLC – Strongest effects in patients no longer responding to PD1-inhibitors



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

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



MODIFICATIONS IN TUMOR MICROENVIRONMENT
FAVORABLE FOR NADUNOLIMAB THERAPY AND MAY BE LINKED TO STRONG EFFICACY OBSERVED

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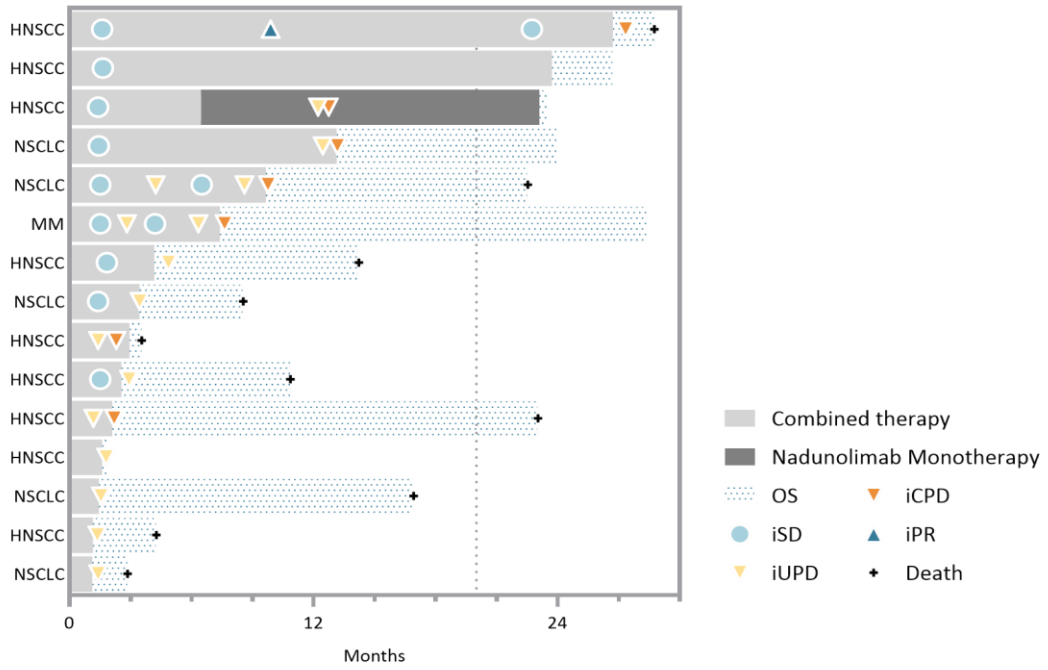
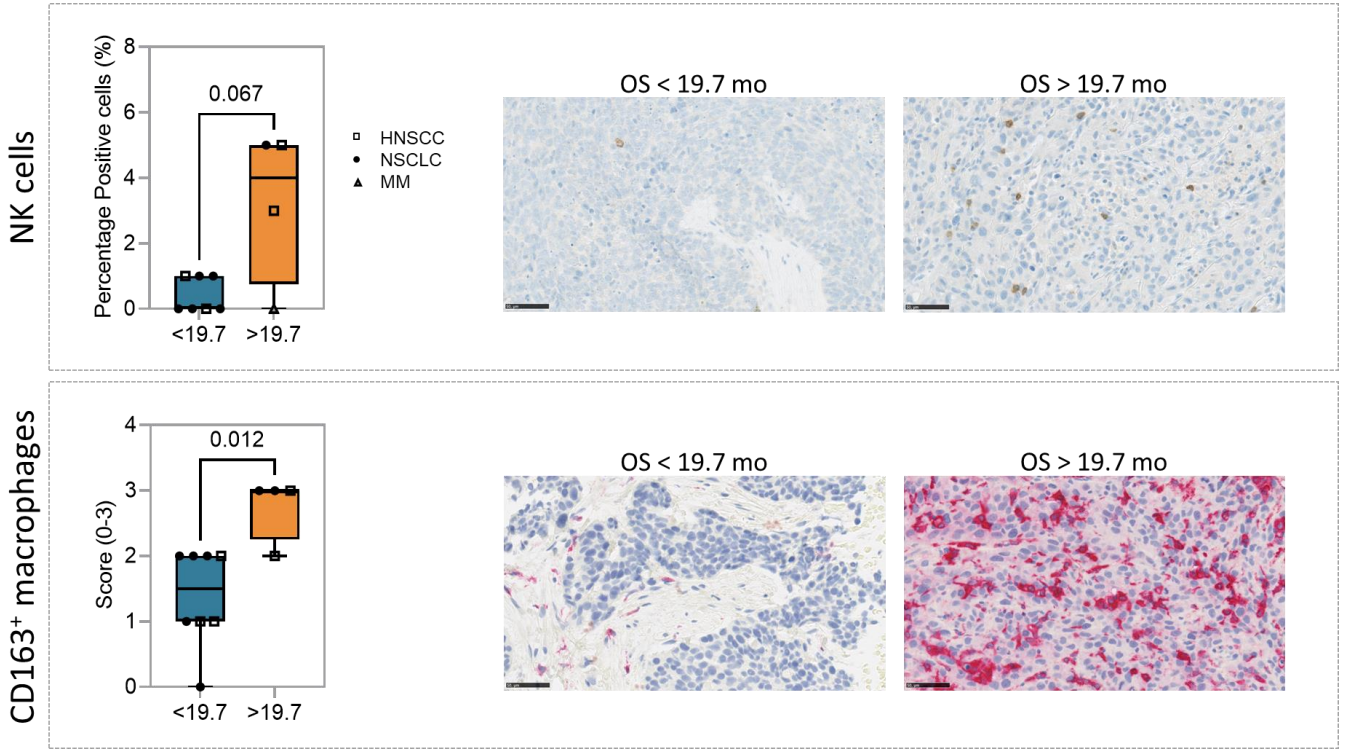
Keytruda combination – Promising signs of clinical activity with remarkable benefit in a subset of patients





COMBINATION WITH KEYTRUDA SHOWS LONG SURVIVAL CORRELATING WITH TUMOR MICROENVIRONMENT CHARACTERISTICS

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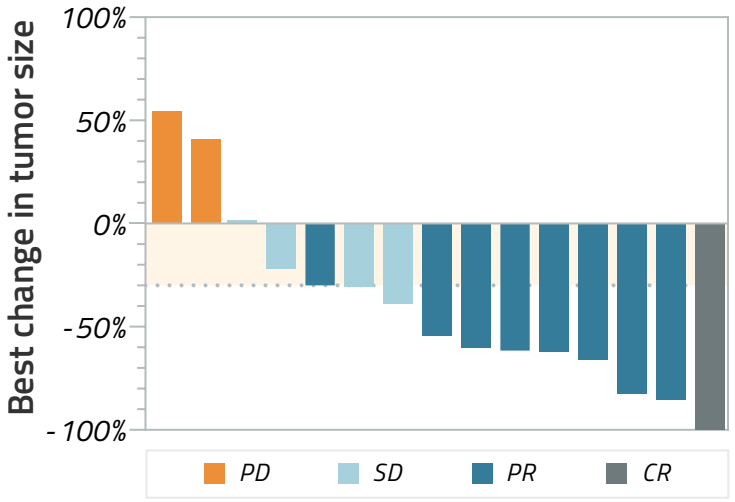
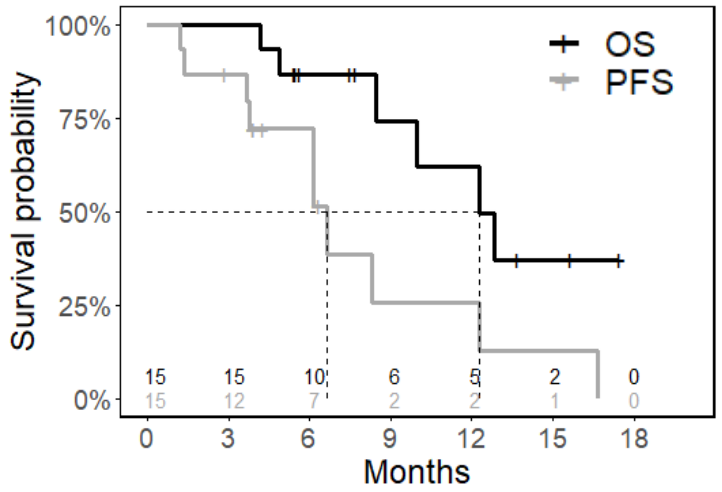
Triple-negative breast cancer (TNBC) – Promising early safety and efficacy

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 2 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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MILESTONES & INVESTMENT HIGHLIGHTS

Upcoming milestones

Nadunolimab

PDAC

- Phase 2b trial in 150-200 patients

TNBC

- Randomized Phase 2 top-line data in H1 2025

AML/MDS

- Start phase 1/2 Q4 2024 (DOD sponsored with MDA*)

THE UNIVERSITY OF TEXAS MD Anderson Cancer Center

CAN10

- Phase 1 data updates during 2024 (including safety and biomarkers)
- Phase 1 final data H1 2025
- Start phase 2 H2 2025

Additional milestones

- New clinical data presented from CAPAFOUR and CESTAFOUR trials
- New preclinical and translational results

EXTENSIVE NEWS FLOW EXPECTED DURING 2024

* MD Anderson

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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