

Changing lives through living medicines

## **Corporate Overview**

August 2024

# DISCLAIMER AND FORWARD-LOOKING STATEMENTS

*This presentation contains forward-looking statements that are based on management's beliefs and assumptions and on information currently available to management. All statements other than statements of historical facts contained in this presentation are forward-looking statements. Forward-looking statements include, but are not limited to, statements concerning: the therapeutic and curative potential of agentT-797 and iNKT cells, the mechanism of action, potency and safety of agentT-797 and iNKT cells, interim or top-line data, future development plans and timelines (including pre-clinical, clinical, regulatory, manufacturing and commercial), estimated treatment costs, our ability to continue to successfully manufacture iNKT cells (including capacity and scalability), and any other statements containing the words "may," "believes," "expects," "anticipates," "hopes," "intends," "plans," "forecasts," "estimates," "will" and similar expressions are intended to identify forward-looking statements. These forward-looking statements are subject to risks and uncertainties, including the factors described under the Risk Factors section of the most recent Form 10-K, Form 10-Q and the S-1 Registration Statement filed with the SEC. Actual results could differ materially and adversely from those anticipated or implied in the forward-looking statements. There are several important factors that could cause MiNK's actual results to differ materially from those indicated by such forward-looking statements, including a deterioration in MiNK's business or prospects; adverse developments in clinical development, including unexpected safety issues observed during a clinical trial; adverse developments in the U.S. or global capital markets, credit markets or economies generally; and changes in regulatory, social, and political conditions. For instance, actual results may differ materially from those indicated by such forward-looking statements as a result of various important factors, including the uncertainties inherent in the initiation, enrollment and maintenance of patients, and completion of clinical trials, availability and timing of data from ongoing clinical trials, expectations for the timing and steps required in the regulatory review process, including our ability to obtain regulatory clearance to commence clinical trials, expectations for regulatory approvals, the impact of competitive products, our ability to enter into agreements with strategic partners. When evaluating MiNK's business and prospects, careful consideration should be given to these risks and uncertainties. These statements speak only as of the date of this presentation, and MiNK undertakes no obligation to update or revise these statements.*

# MINK PIONEERS ALLOGENEIC IMMUNE CELL THERAPIES IN ARDS AND CANCER

## MiNK Therapeutics

### Universal Cell Therapy Platform

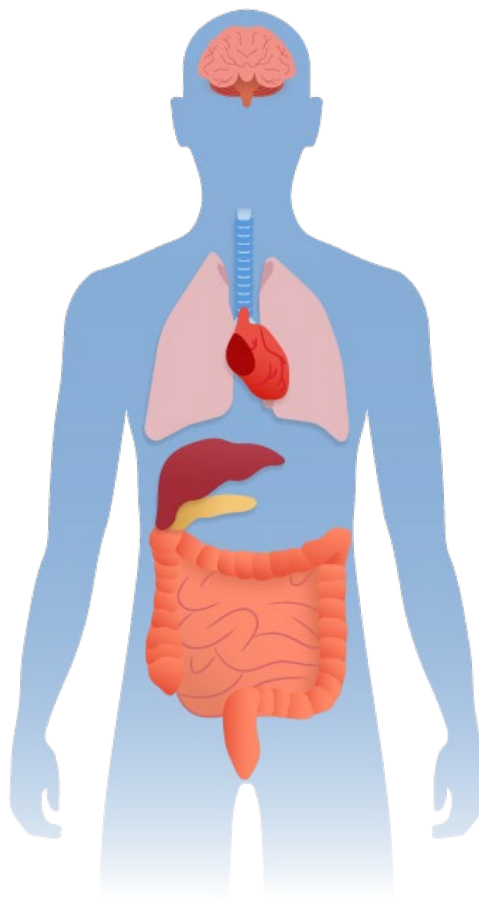
Readily available, allogeneic, innate T cells (iNKT) administered without lymphodepletion or tissue matching

### Robust Pipeline of Allogeneic Products

Fully internal capabilities to manufacture, engineer CARs, TCRs, and bispecific engagers

### Proprietary Manufacturing

In-house, scalable, closed automated process to produce functional iNKTs at scale



## agenT-797: Unmodified iNKT cells

3

Completed Phase 1 trials in respiratory distress and oncology

>80

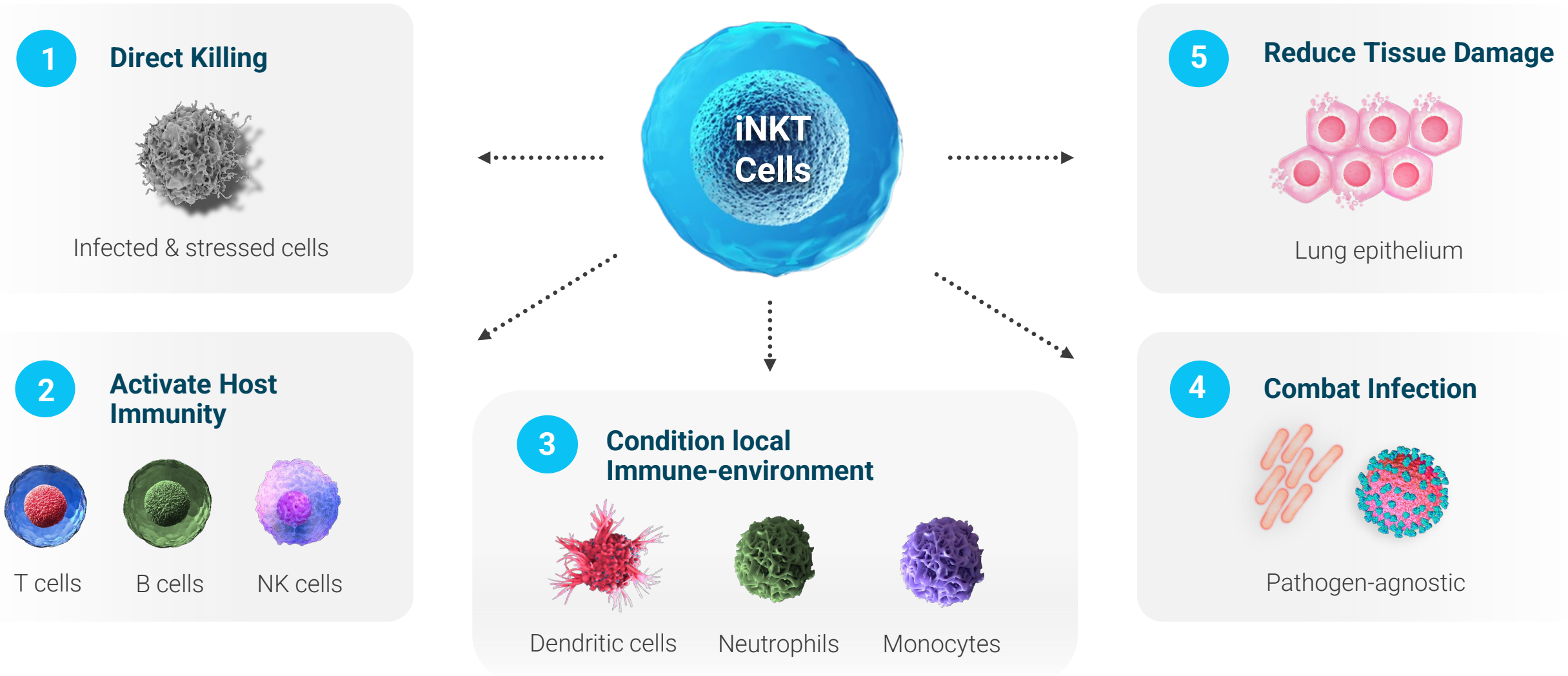
Patients treated with no CRS, no GvHD, no ICANS

No lymphodepletion or HLA matching needed

70%

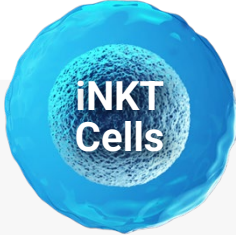
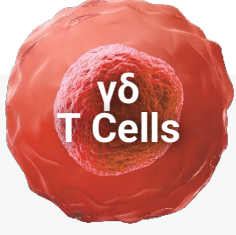
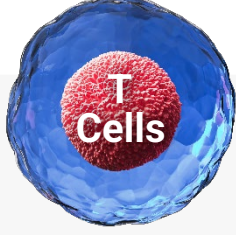
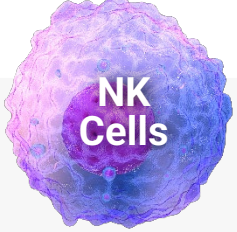
30-day survival rate in patients with moderate to severe ARDS<sup>1</sup>

# iNKT CELLS HAVE MULTIPLE INDIRECT AND DIRECT MODES OF ACTION



# INKT CELLS ARE POWERING THE NEXT GENERATION OF CELL THERAPIES

OVERCOMES PRACTICAL AND MECHANISTIC CHALLENGES OF OTHER CELL TYPES

				
Innate AND adaptive immune modulation	✓	✓	✗	✗
Tumor homing and persistence	✓	✓	✗	✗
No Lymphodepletion	✓	?	✗	?
Naturally suppresses GvHD	✓	✗	✗	✗
No exhaustion	✓	✗	✗	✗
Potential to multi-dose without lymphodepletion	✓	✗	✗	✗

# UNIQUE BENEFITS OF iNKT CELLS OBSERVED IN THE CLINIC TO DATE

SAFE AND CLINICALLY BENEFICIAL RESPONSES WHERE STANDARD TREATMENTS HAVE FAILED



**First immune cell therapy to improve survival in ARDS**

- **30-day survival of 70%** (14/20) vs 10% in-hospital control
- **90-day survival of 75%** (3/4) in VV-ECMO patients



**Long-term disease stabilization in solid tumors**

- **6M+ Stable Disease** in relapsed/refractory solid tumors
- **Partial response** observed in PD-1 refractory Gastric cancer with **42% reduction** in tumor lesion



**Excellent safety profile in ARDS and solid tumor patients**

- **No GvHD, CRS or ICANS**
- **No treatment discontinuation**, dose interruption, nor death due to TRAEs



**HLA unmatched allogeneic therapy with scalable capacity**

- Easily administered **without lymphodepletion**
- Manufactured in Lexington, MA with **easy distribution globally**

# INKT CELLS CAN BE ARMORED TO ENHANCE TUMOR KILLING

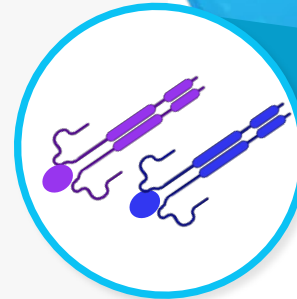
## CAR/TCR Engineering

- Proprietary discovery platforms without additional gene edits
- Targeted tumor cell killing



## Cytokine Engineering

- In vivo expansion
- Improved persistence



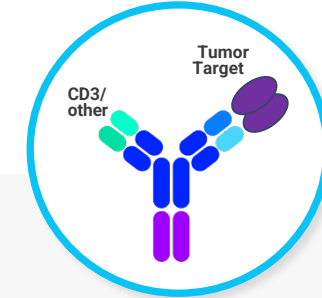
iNKT  
Cells



## Bispecific Engagers

Enhanced activity via synergies with

- CD3 based engagers
- Proprietary iNKT engagers

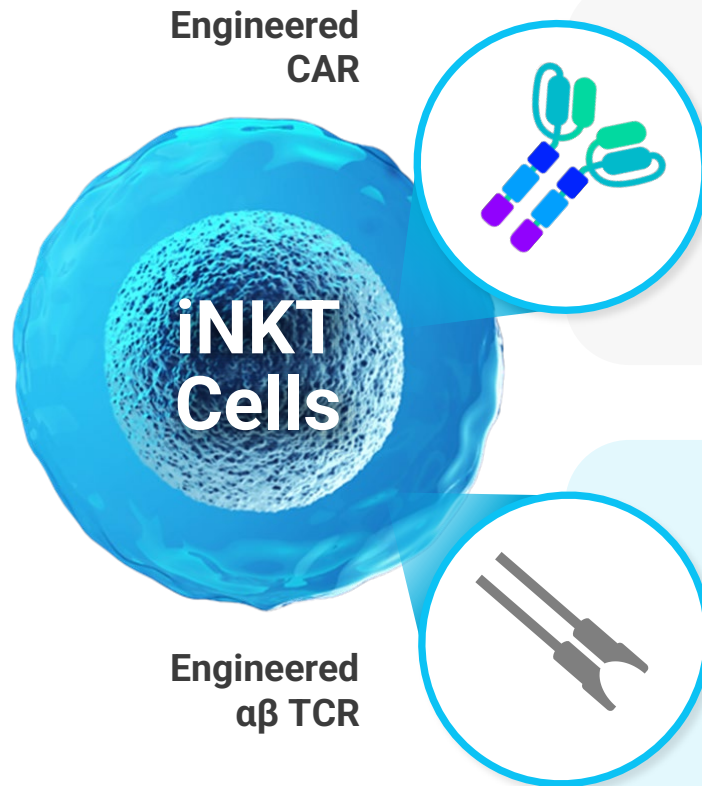


# INNOVATIVE PIPELINE WITH NATIVE AND ENGINEERED INKTS

Product	Target	Indication and Approach	Preclinical	IND-enabling	Phase 1	Phase 2	Latest & Upcoming Milestones
agenT-797	Native iNKT	Solid tumors ± anti-PD1					<ul style="list-style-type: none"> <li>Updated data at SITC 2023</li> </ul>
		Gastric cancer + SOC ± BOT/BAL <sup>1</sup>					<ul style="list-style-type: none"> <li>First patient dosed 2024</li> </ul>
		Acute Respiratory Distress Syndrome (ARDS)					<ul style="list-style-type: none"> <li>Updated data at ATS 2024</li> </ul>
MiNK-215	FAP CAR	Solid tumors					<ul style="list-style-type: none"> <li>Potential IND filing 2025</li> <li>Updated data at AACR 2024</li> </ul>
MiNK-413	BCMA CAR	Multiple myeloma					<ul style="list-style-type: none"> <li>IND ready 2024</li> </ul>
MiNK-PRAME-TCR	PRAME TCR	Solid tumors					<ul style="list-style-type: none"> <li>Candidate nomination 2024</li> </ul>
MiNK-Engagers	Undisclosed	Solid tumors					<ul style="list-style-type: none"> <li>Candidate nomination 2024</li> </ul>



# MINK THERAPEUTICS HAS ROBUST DISCOVERY PLATFORMS FOR CAR AND TCR



## CARDIS enables high-throughput DISCOVERY of functional cars

CAR Expressed into mammalian display library

Identify strong binders and activators

Eliminate false binders and non-functional CARs

## Trx platform enables efficient TCR discovery & engineering

TCR expressed in mammalian display cell line

High throughput functional and specificity testing

In vitro characterization for candidate selection

# RAPID CLINICAL DEVELOPMENT THROUGH PARTNERSHIPS

HIGH IMPACT COLLABORATIONS AND NON-DILUTIVE FINANCING



**PRINCIPAL INVESTIGATOR:**  
**Dr. Terese C. Hammond**  
Pulmonology and Critical Care

**Phase 2: agenT-797 in viral ARDS**



**Discovery: novel TCR targets**



**PRINCIPAL INVESTIGATOR:**  
**Dr. Yelena Janjigian**  
Chief Gastrointestinal Oncology

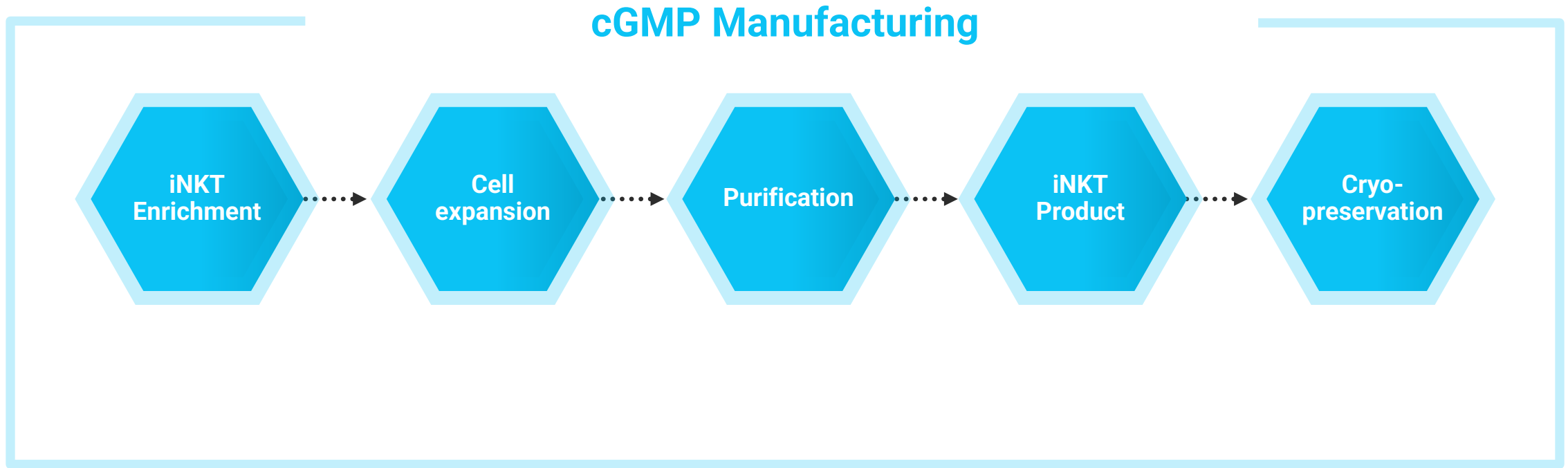
**Phase 2: agenT-797 + chemotherapy ± PD-1/CTLA-4 in Gastric Cancer**



**Clinical and Research: agenT-797 combination with immune checkpoint inhibitors**

# MINK MANUFACTURING PROCESS TO ACHIEVE $\leq$ \$10K PER DOSE

OFF-THE-SHELF, COST-EFFECTIVE AND SCALABLE TO >5000 DOSES



In-house manufacturing



Automatic and fully closed process

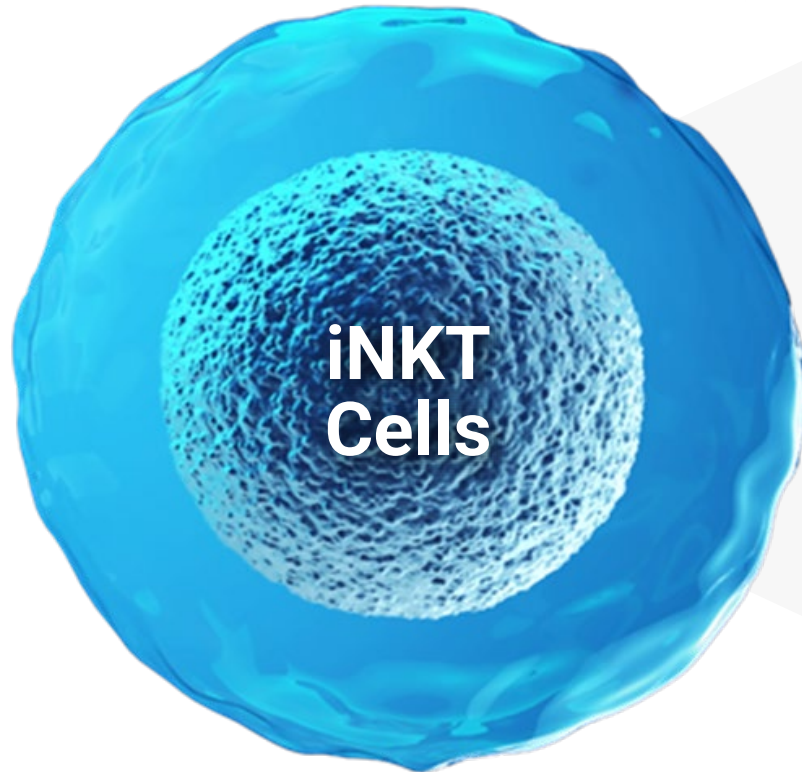


Commercial ready

# agenT-797

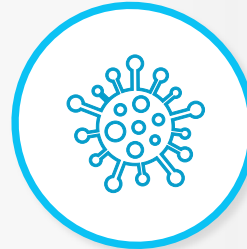
*Clinical data in immune dysfunction*

# INKT CELLS PLAY A PROTECTIVE ROLE IN INFECTION AND INFLAMMATION



## Infections

- Bacterial and viral infections
- Promotes CD8+ cytotoxic response
- Protects against tissue damage



## Autoimmune Diseases

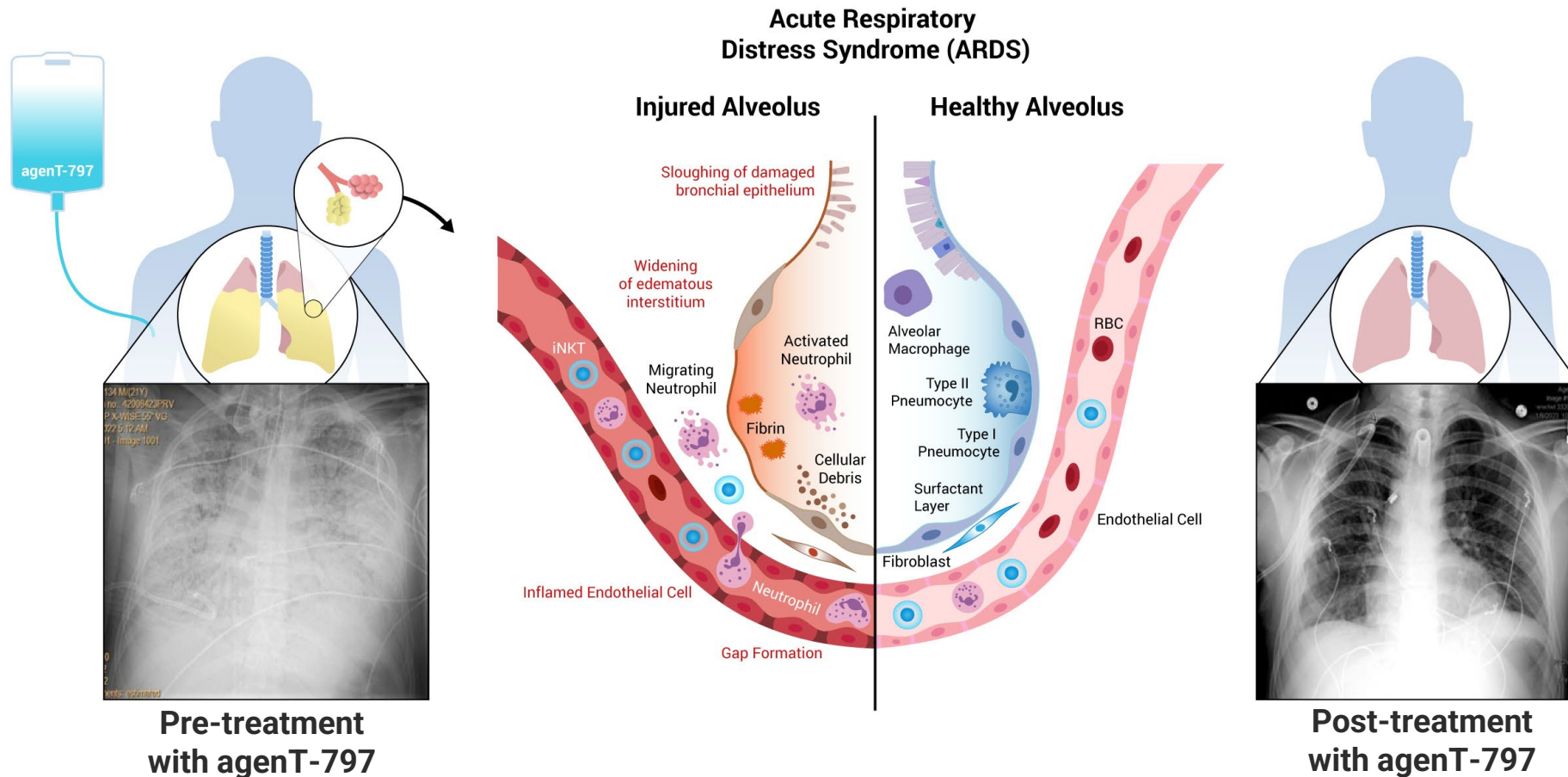
- Lupus, Multiple Sclerosis, Arthritis, Diabetes
- Induction of suppressive cells
- Modulating cytokine and Th profile



## Pulmonary Fibrosis & Lung Dysfunction

- Immune or non-immune -mediated
- Suppresses pro-fibrotic factors such as TGFB
- Modulates macrophage polarization

# AGENT-797, MINK'S UNMODIFIED INVARIANT NATURAL KILLER T (iNKT) CELL THERAPY CAN EFFECTIVELY TREAT ARDS



## agentT-797 in ARDS

**30-day survival of 70%** vs. 10% in-hospital control

**15% incidence of secondary bacterial pneumonia** at recommended dose level

**Only 5% (1/20)** experienced a TRAE of grade  $\geq 3$

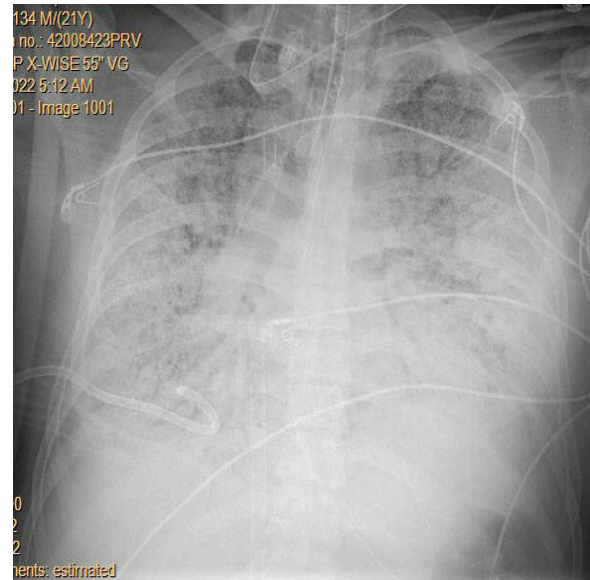
**HLA-unmatched, allogenic, readily available**, cost effective, and scalable cell therapy

# RAPID RESOLUTION OF CARBAPENEM-RESISTANT PNEUMONIA AND SEVERE ARDS WITH AGENT-797 THERAPY

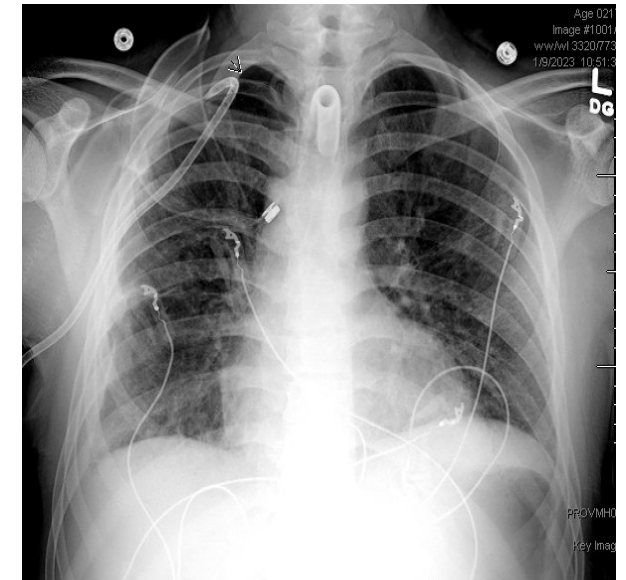
Patient cleared lung infection and stopped VV-ECMO 13 days post agentT-797 infusion

<b>Patient Characteristics</b>	<ul style="list-style-type: none"><li>• 21-year-old male</li><li>• <b>Severe ARDS</b></li><li>• <b>Carbapenem resistant Pseudomonal Pneumonia on VV-ECMO</b></li></ul>
<b>Treatment</b>	<ul style="list-style-type: none"><li>• <b>Emergency use Access (EUA)</b></li><li>• <b>Single dose of agentT-797</b></li><li>• DL2: <math>1 \times 10^9</math> cells</li></ul>
<b>Response</b>	<ul style="list-style-type: none"><li>• Cleared Infection</li><li>• <b>Stopped ECMO 13 days post-infusion</b></li><li>• Reduced pro-inflammatory lung cytokines that drive pathology</li><li>• Increased monocytes in lungs to rapidly clear the infection and resolve inflammation</li></ul>

**Pre-treatment**

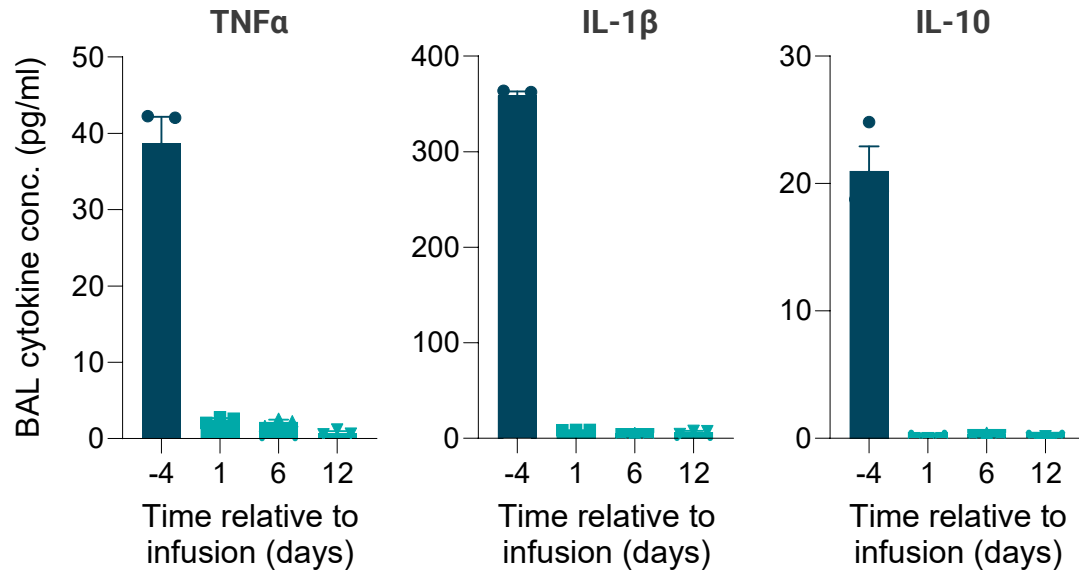


**Post agentT-797**

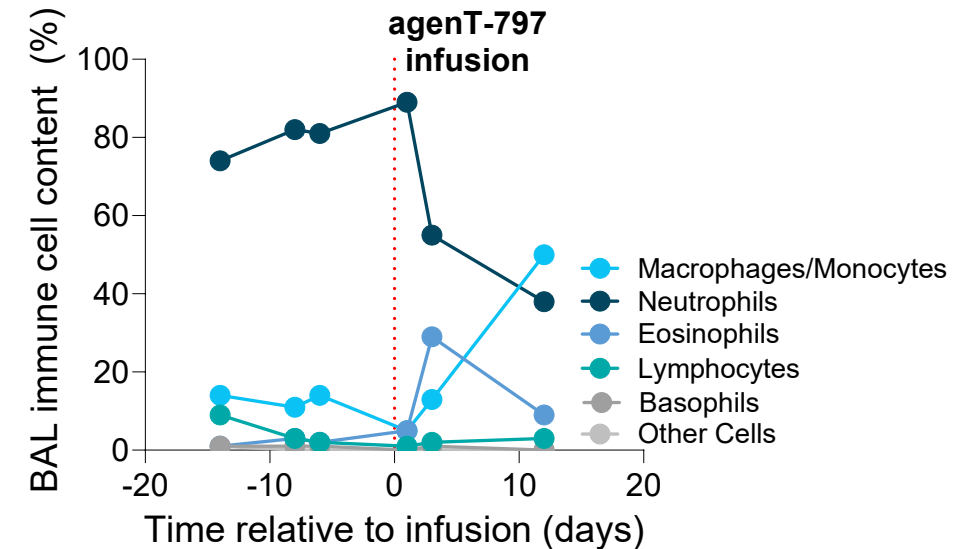


# AGENT-797 REDUCED INFLAMMATORY CYTOKINES AND NEUTROPHILS WITH MONOCYTE-MEDIATED INFECTION CLEARANCE

Reduced pro-inflammatory and myeloid suppressing cytokines in bronchoalveolar lavage



Rapidly reduced neutrophils and increased monocytes/macrophages in bronchoalveolar lavage



Reduction in neutrophil count correlates with resolution of the bacterial infection

Influx of monocyte/macrophages correlates with clearance of infection debris

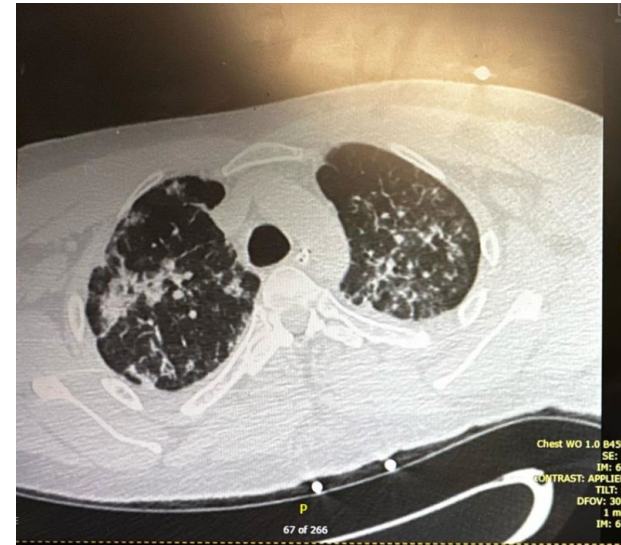


# RAPID CLINICAL RESPONSE TO COVID-19 ARDS REQUIRING VV-ECMO WITH AGENT-797 TREATMENT

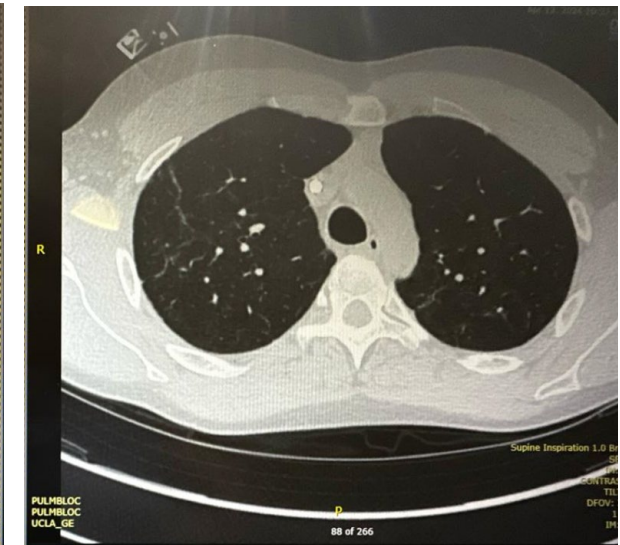
Patient cleared lung infection and stopped VV-ECMO 24 days post agenT-797 infusion

<p><b>Patient Characteristics</b></p>	<ul style="list-style-type: none"> <li>• 26-year-old male</li> <li>• <b>Severe COVID-19 ARDS</b></li> <li>• Unresponsive to remdesivir, baricitinib and convalescent plasma requiring VV-ECMO</li> <li>• Cadaveric renal transplant at age 11, on tacrolimus and prednisone. <b>Required dialysis post-infection.</b></li> </ul>
<p><b>Treatment</b></p>	<ul style="list-style-type: none"> <li>• <b>Emergency use Access (EUA)</b></li> <li>• <b>Single dose of agenT-797</b></li> <li>• DL2: <math>1 \times 10^9</math> cells</li> </ul>
<p><b>Response</b></p>	<ul style="list-style-type: none"> <li>• <b>Extubated, decannulated ECMO 24 days post infusion</b></li> <li>• <b>Stopped dialysis</b></li> <li>• Rapid reduction of proinflammatory cytokines, and VEGF-D, a known marker of alloreactive immune response</li> </ul>

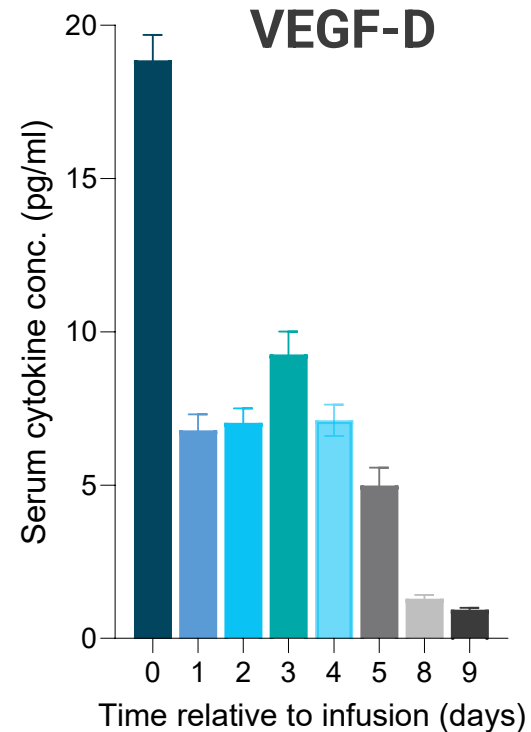
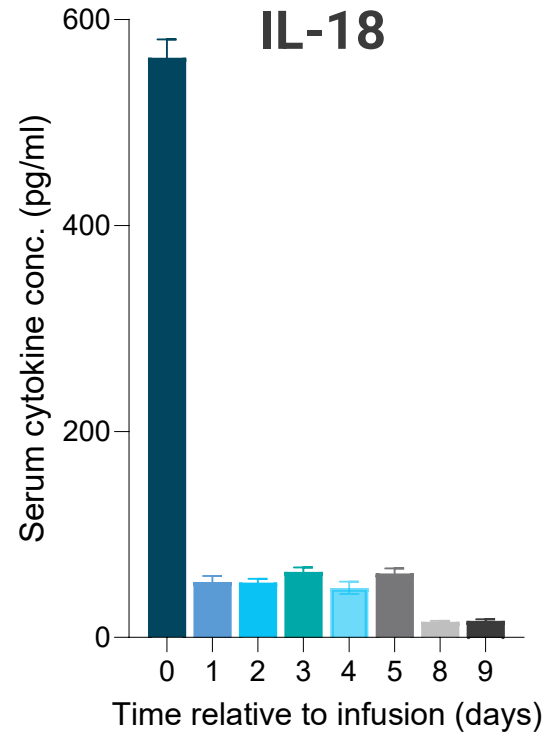
**Pre-treatment**



**Post agenT-797**



# AGENT-797 REDUCES PRO-INFLAMMATORY CYTOKINES AND MARKERS OF ALLOREACTIVITY



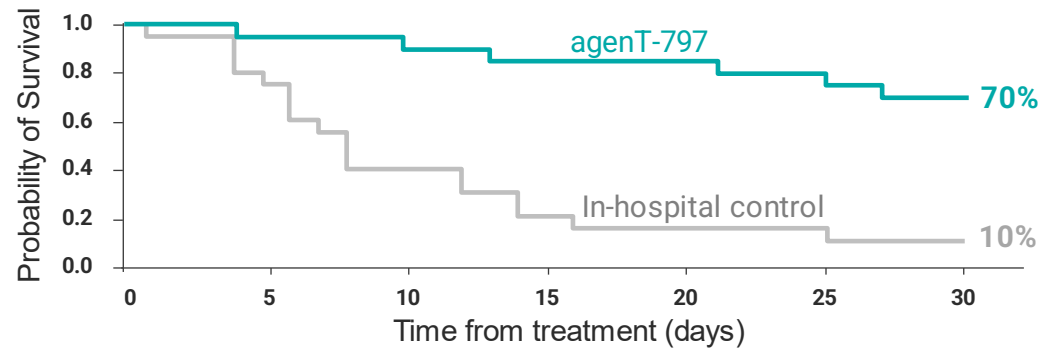
Reduction in **IL-18**, associated with decreased survival in ARDS, and known biomarker of severe Acute Kidney Injury (AKI)

Reduction in **VEGF-D**, an angiogenic growth factor implicated in detrimental alloreactive immune response in kidney transplant

# AGENT-797 IMPROVES SURVIVAL AND LUNG FUNCTION IN SEVERE VIRAL ARDS

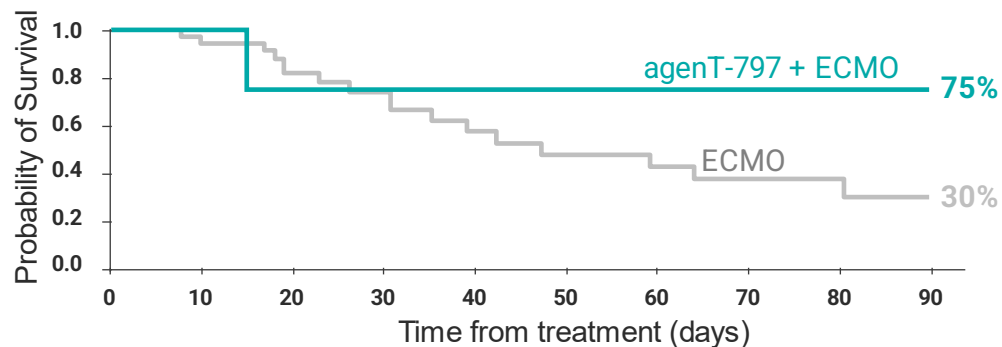
DATA FROM PHASE 1/2 CLINICAL TRIAL

## Increased survival vs case control



At recommended dose of  $10^9$  cells, only **15%** incidence of secondary bacterial pneumonia was observed (N=13)

## Increased survival on VV-ECMO



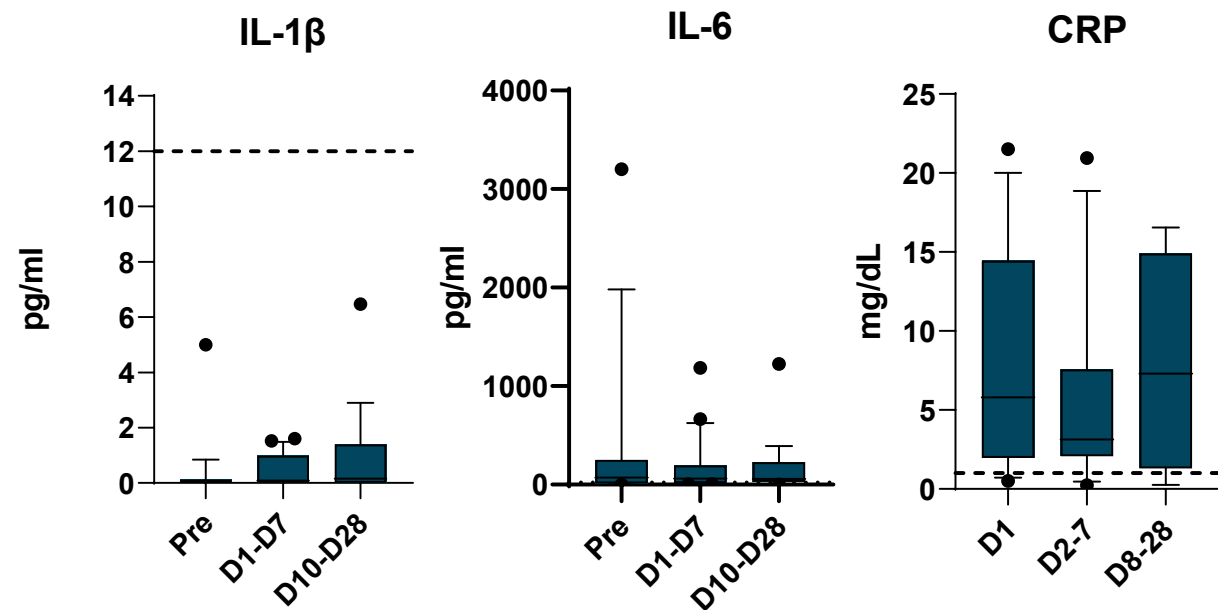
Compare to prevalence of up to **84%** in ventilator-associated pneumonia

# AGENT-797 IS WELL TOLERATED IN SEVERE VIRAL ARDS PATIENTS

## No Significant Adverse Events

	agentT-797 ± ECMO (n=20)	agentT-797 + ECMO (n=4)
	n (%)	n (%)
<b>AE</b>	<b>20 (100)</b>	<b>4 (100)</b>
Any AE of grade ≥ 3	19 (95)	4 (100)
<b>TRAE</b>	<b>5 (25)</b>	<b>0 (0)</b>
Any TRAE of grade ≥ 3	1 (5)	0
Any TRAE leading to discontinuation	0	0
Any TRAE leading to dose interruption	0	0
Any TRAE leading to death	0	0

## No Cytokine Release Syndrome



# ACUTE RESPIRATORY DISTRESS SYNDROME (ARDS)

ARDS Represents a High Unmet Need With 40% Mortality Rate

## Lack of Effective Treatment Options

- Limited response to standard of care corticosteroids and mechanical ventilation
- Significant healthcare costs of up to \$100K/day in the ICU

~200K

annual  
incidence in US

40%

Mortality  
rate

\$100K  
/day

Cost burden to  
US healthcare

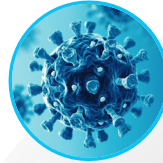
# AGENT-797 IS A VERSATILE THERAPY AGNOSTIC TO CAUSATIVE AGENTS

APPLICABLE TO MULTIPLE OTHER INDICATIONS

## ARDS-related

### Viral

Corona virus, Influenza virus,  
RSV, Herpesvirus, Hantavirus...



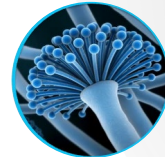
### Fungal

Aspergillus Fumigatus...



### Bacterial

Pneumococcus, Legionella,  
Mycobacterium Tuberculosis...



### Lung Injury (Non-Infectious)

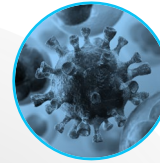
Radiation, Chemical, IPF



## Lifecycle management

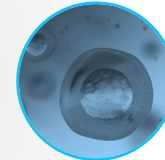
### Cancer

Gastric cancer, NSCLC,  
CRC with liver metastasis



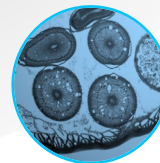
### GvHD

Therapeutic and prophylactic in  
post HSCT



### Metabolic disorders

Senescence, Obesity, CVD



agent-797

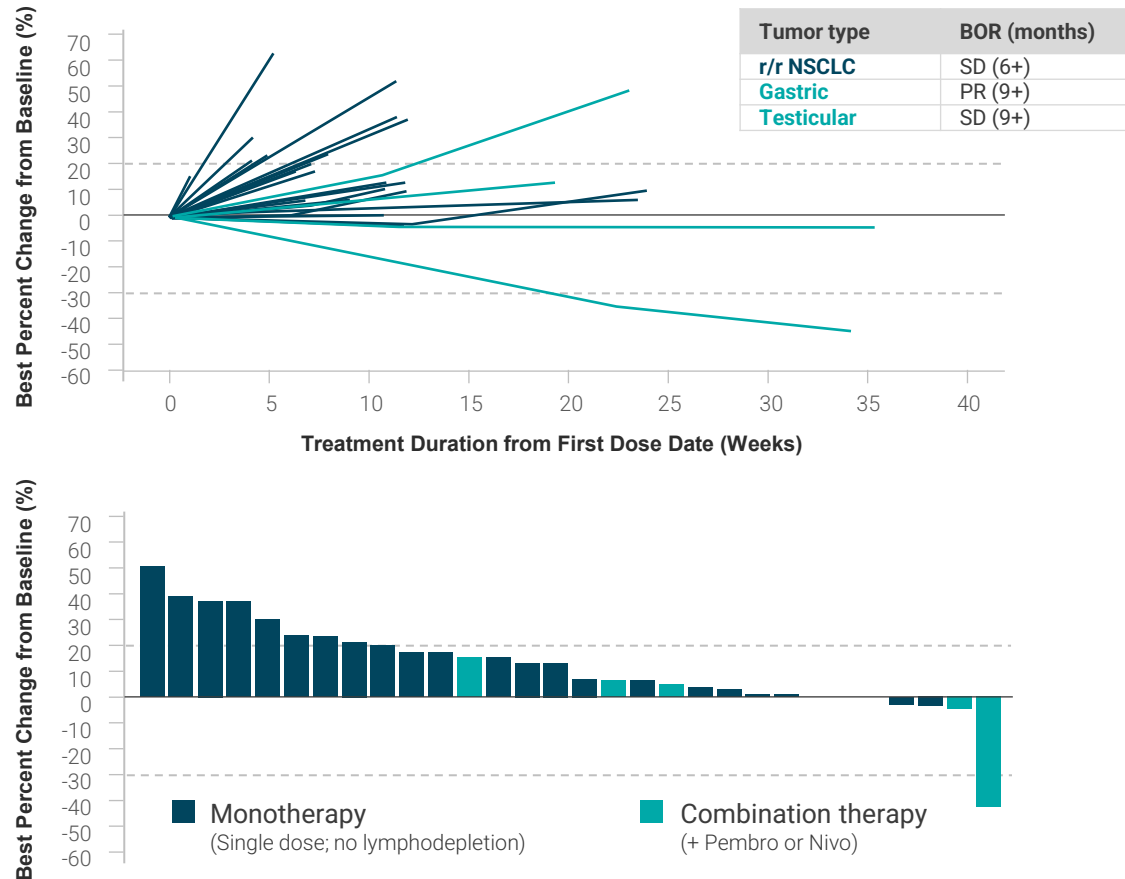
# agenT-797

*Clinical data in solid tumors*

# AGENT-797 SHOWS RESPONSES AND DURABLE STABILIZATION

## SINGLE DOSE WITHOUT LYMPHODEPLETION IN HEAVILY PRE-TREATED PATIENTS

### Target Lesion Change



### 3L+ Solid Tumors

	Monotherapy (n=28)	Combination (n=6)
<b>BOR , n (%)</b>		
Partial Response	0 (0%)	1 (17%)
Stable Disease	7 (25%)	3 (50%)
<b>DCR [CR + PR + SD], n (%)</b>	7 (25%)	4 (67%)
<b>Median PFS (months, 95% CI)</b>	2.3 (1.6, 3.0)	5.5 (1.8, 10.3)
<b>Median follow-up (months)</b>	6.0	10.3



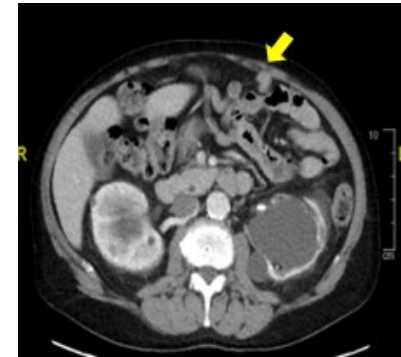
# PARTIAL RESPONSE IN PD-1 REFRACTORY GASTRIC CANCER

42% TARGET LESION REDUCTION AT 9 MONTHS; RESPONSE ONGOING

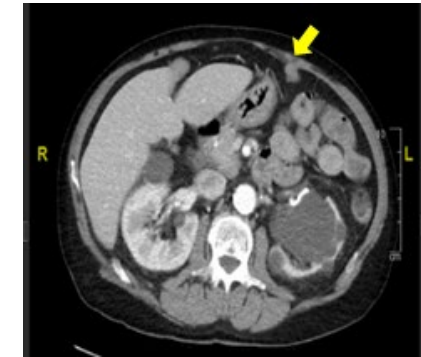
## Gastric Cancer Patient

<b>Patient Characteristics</b>	<ul style="list-style-type: none"><li>75-year-old male</li><li>Failed prior PD-1 therapies</li></ul>
<b>Prior Therapies</b>	<ul style="list-style-type: none"><li>Pembrolizumab PD</li><li>FOLFOX + nivolumab + oxaliplatin SD</li></ul>
<b>Treatment</b>	<ul style="list-style-type: none"><li>Single dose of agenT-797 + nivolumab (200mg)</li><li>DL1: <math>4.3 \times 10^6</math> cells/kg</li></ul>
<b>Response</b>	<ul style="list-style-type: none"><li>33% target reduction at 6 months</li><li>42% target reduction at 9 months</li><li>PFS: 10M+</li></ul>

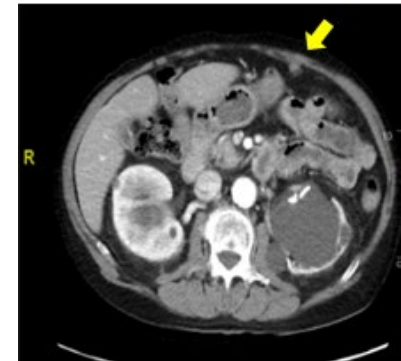
Baseline



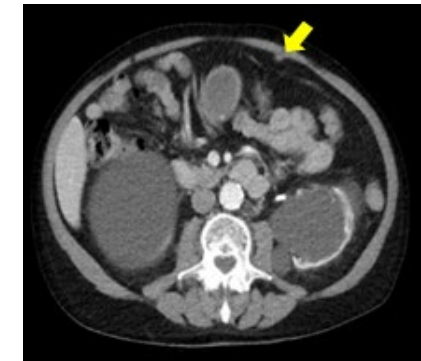
Month 3



Month 6



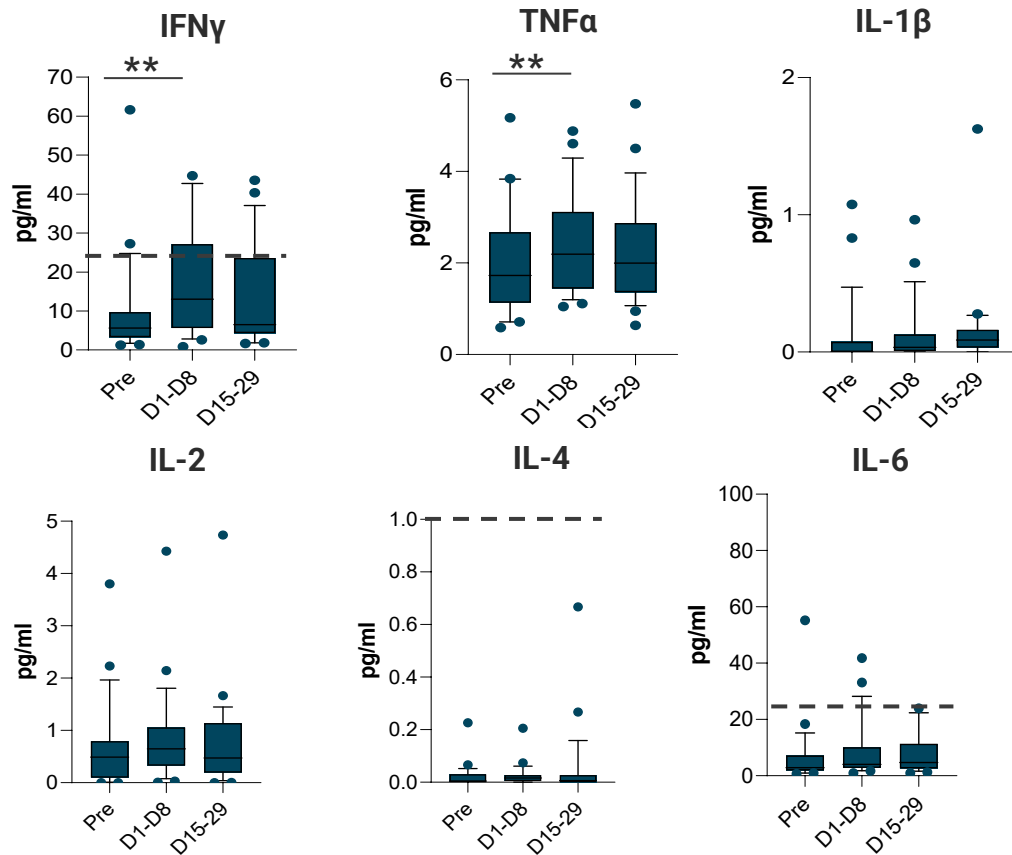
Month 9



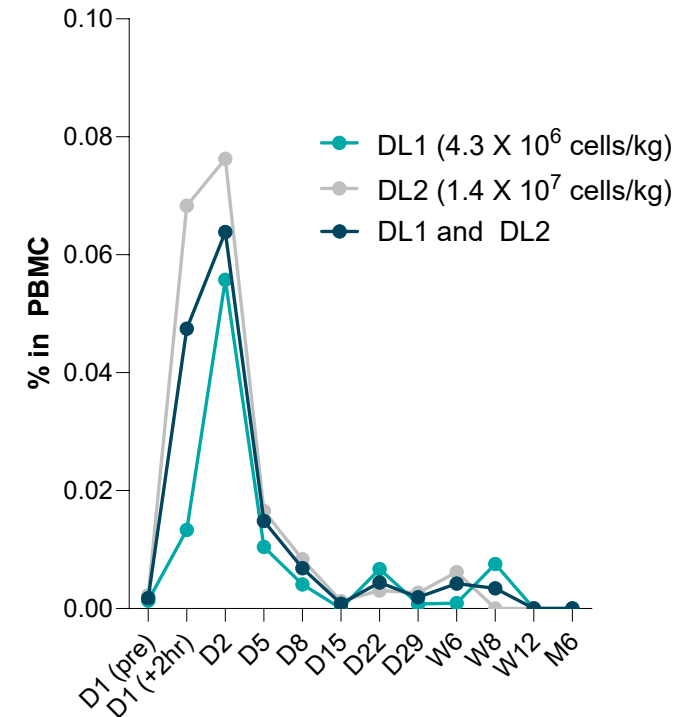
# AGENT-797: PROLONGED PERIPHERAL PERSISTENCE AND TH1 CYTOKINE PROFILE

## ENHANCED IFN $\gamma$ AND TNF $\alpha$ AND UP TO 6 MONTHS PERSISTENCE

### Enhanced Th1 Cytokine Profile



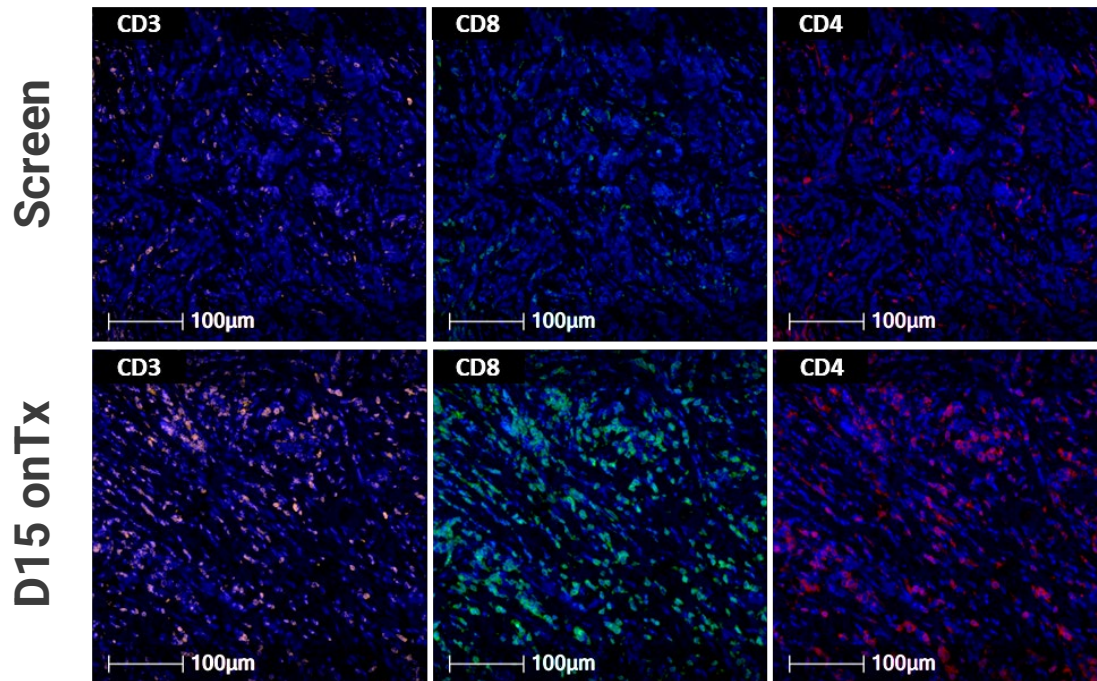
### Long Peripheral Persistence



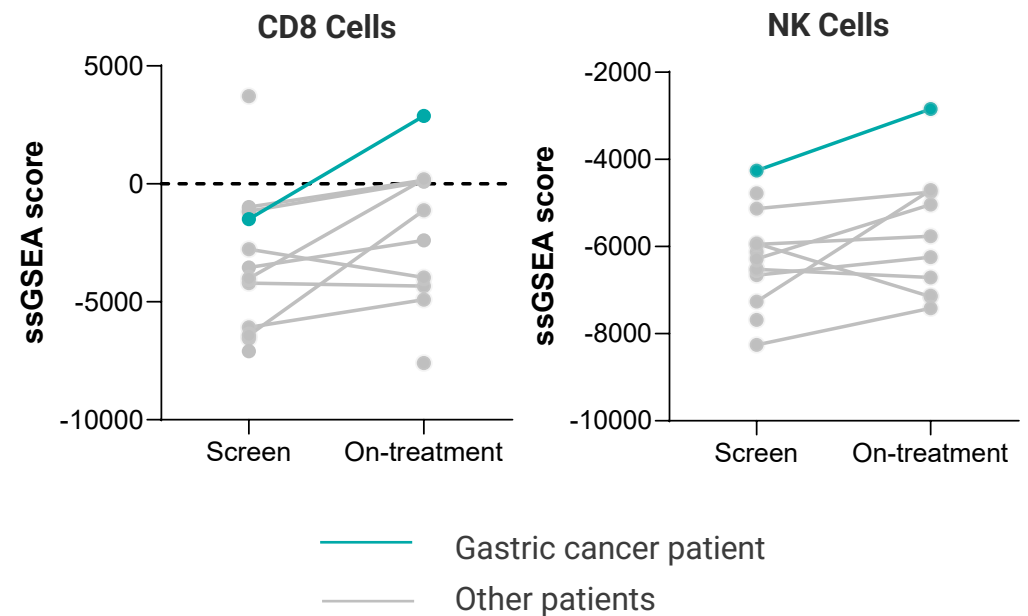
# AGENT-797: PROMOTES IMMUNE CELL INFILTRATION IN TUMOR

INCREASED CD3, CD4, CD8 AND NK CELLS

Increased T Cell Infiltration  
(mIF)



Increased CD8 and NK cell Infiltration  
(RNA-Seq)



# AGENT-797 IS WELL-TOLERATED

## NO DLTS AND FEW RELATED ADVERSE EVENTS

	Total	agenT-797 MonoTx		agenT-797 + anti-PD-1	
Dose level	N = 34	DL1: 4.3 x 10 <sup>6</sup> cells/kg N = 8	DL2: 1.4 x 10 <sup>7</sup> cells/kg N = 20	DL1: 4.3 x 10 <sup>6</sup> cells/kg N = 3	DL2: 1.4 x 10 <sup>7</sup> cells/kg N = 3
<b>AE, n (%)</b>	<b>32 (94)</b>	<b>8 (100)</b>	<b>18 (90)</b>	<b>3 (100)</b>	<b>3 (100)</b>
Any AE of grade ≥ 3	19 (56)	7 (88)	11 (55)	0	1 (33)
	<b>3 (9)</b>	<b>0</b>	<b>2 (10)</b>	<b>0</b>	<b>1 (33)</b>
	1 (3)	0	0	0	1 (33)
<b>TRAE, n (%)</b>	<b>9 (27)</b>	<b>3 (38)</b>	<b>2 (10)</b>	<b>2 (67)</b>	<b>2 (67)</b>
Any TRAE of grade ≥ 3	1 (3)	1 (13)	0	0	0
Any TRAE leading to discontinuation	0	0	0	0	0
Any TRAE leading to dose interruption	0	0	0	0	0
Any TRAE leading to death	0	0	0	0	0
<b>TRAE by System Organ Class, n (%)</b>					
General (Fatigue, Chills)	5 (15)	1 (13)	1 (5)	1 (33)	2 (67)
Skin (Pruritus, Odor)	2 (6)	1 (13)	0	1 (33)	0
Immune system (CRS)	1 (3)	0	1 (5)	0	0
Nervous system (Dysgeusia)	1 (3)	0	0	0	1 (33)
Psychiatric (Insomnia)	1 (3)	0	0	1 (33)	0
Respiratory (Dyspnoea)	1 (3)	0	1 (5)	0	0
Blood and lymphatic system (Anemia)	1 (3)	1 (13)	0	0	0

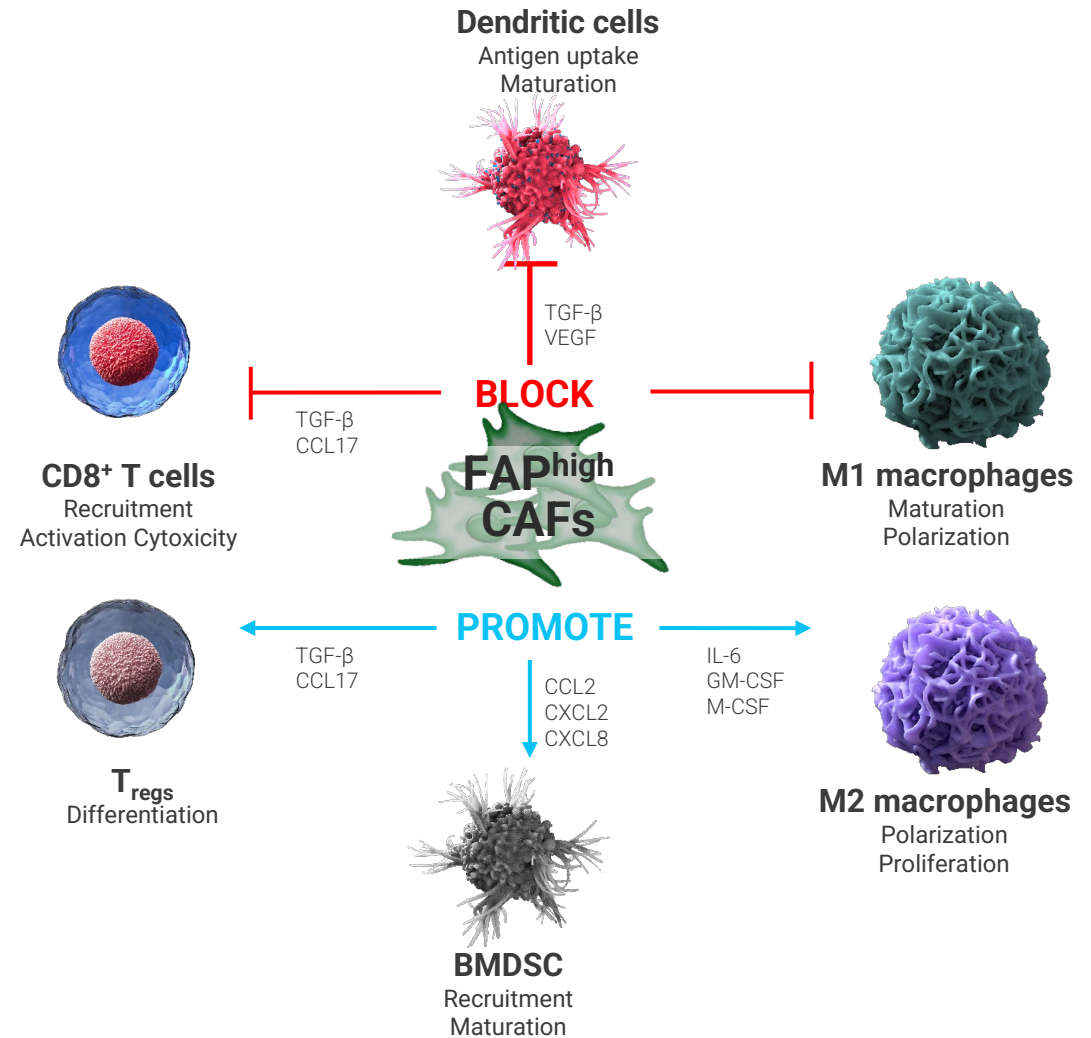
# MiNK-215

*Preclinical data from Engineered FAP-CAR-iNKT cells*

# TARGETING TUMOR-PROMOTING STROMAL CELLS IN SOLID TUMORS

FAP<sup>HIGH</sup> CAFs OCCUR IN >90% OF EPITHELIAL-DERIVED TUMORS

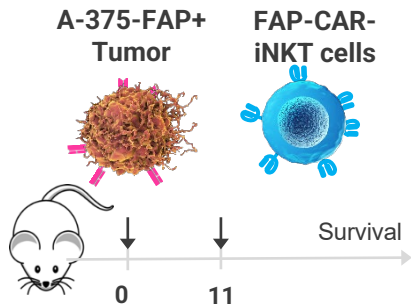
- FAP<sup>high</sup> CAFs are highly immune-suppressive and tumor-promoting in the TME
- FAP<sup>high</sup> CAFs secrete a variety of cytokines to modulate immune activity
- Targeting FAP<sup>high</sup> CAFs may result in tumor cell death in highly stromagenic cancers without IO success



# MINK FAP-CAR-iNKT PROMOTES SURVIVAL IN FAP+ TUMOR-BEARING MICE

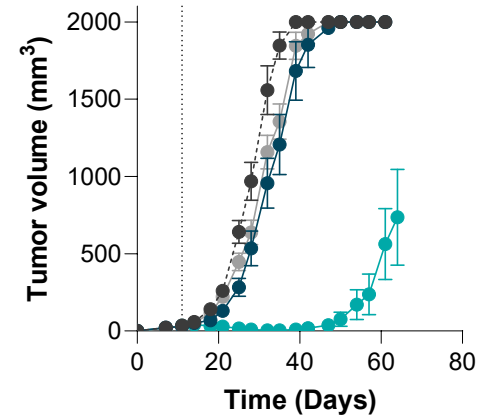
## SUPERIOR ANTI-TUMOR ACTIVITY TO CLINICAL REFERENCE CAR (SIBROTUZUMAB)

### A375 Melanoma Model



- Day 0: FAP+ A-375 tumor cells injected subcutaneously into NOG-tg (IL15) mice
- Day 11: FAP-CAR- iNKT cells administered

### Improved Tumor Control



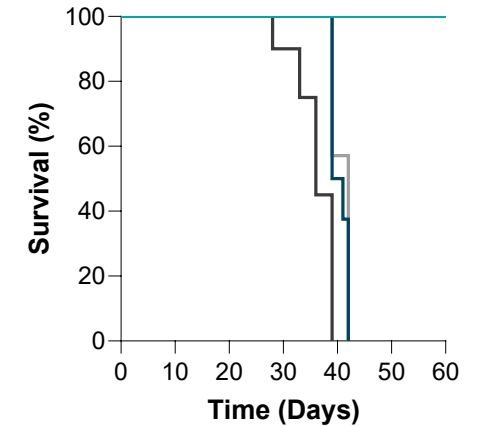
● Untreated

● Negative Control BCMA-CAR-iNKT

● Sibrotuzumab-CAR-iNKT

● MiNK-FAP-CAR-iNKT

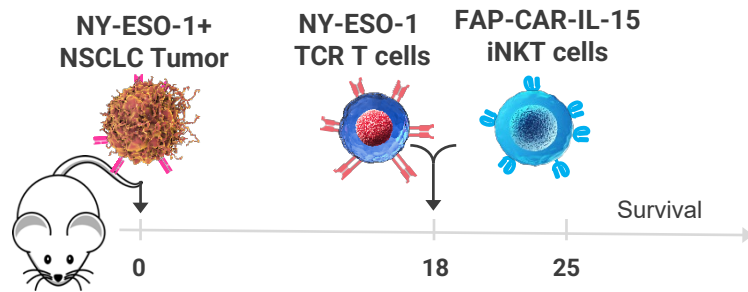
### Superior Survival



# NSCLC MOUSE ORTHOTOPIC MODEL RECAPITULATES TUMOR STROMA

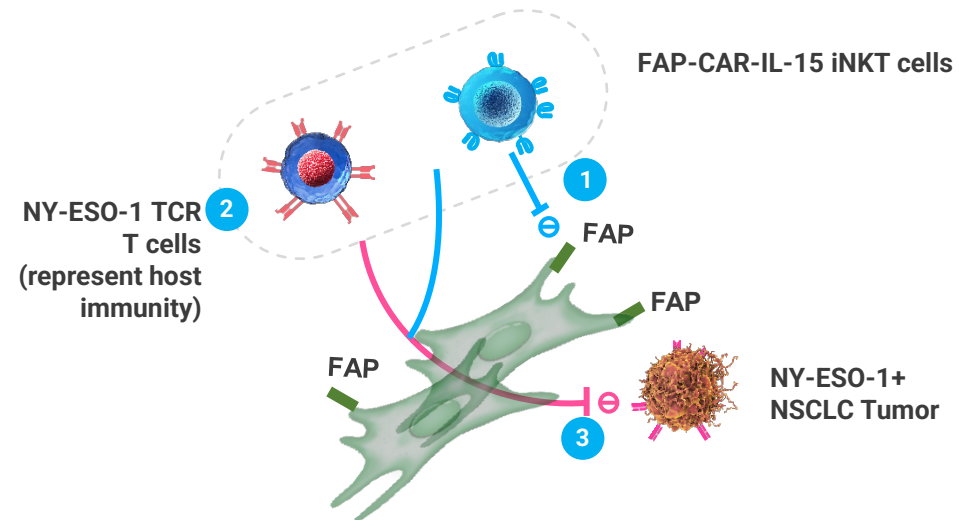
## IN VIVO ASSESSMENT OF TARGETING FAP+ CAFs

### Orthotopic NSCLC Model



- Day 0: A-549 tumor cells expressing NY-ESO-1 antigen injected into immunodeficient mice
- Day 18: FAP-CAR-IL-15 iNKT cells and/or NY-ESO-1 TCR T cells administered
- NY-ESO-1 TCR T cells mimic host T cells

### FAP-CAR-IL-15 iNKT Mechanism of Action



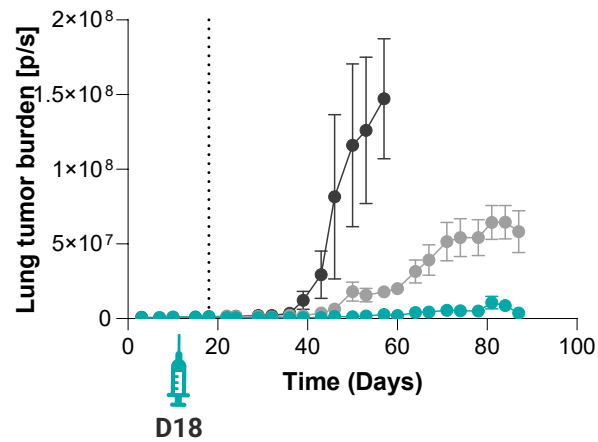
- 1 FAP-CAR-IL-15 iNKT cells directly target and kill FAP-expressing CAFs
- 2 FAP-CAR-IL-15 iNKT cells recruit T cells to the tumor microenvironment
- 3 T cells infiltrate the tumor tissue and directly kill tumor cells



# MINK-215 HALTS TUMOR GROWTH AND IMPROVES SURVIVAL IN MICE

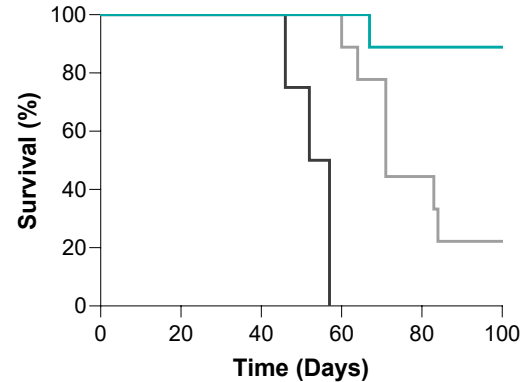
## SYNERGIZES WITH HOST T CELLS FOR ENHANCED ACTIVITY

### Reduced Tumor Burden

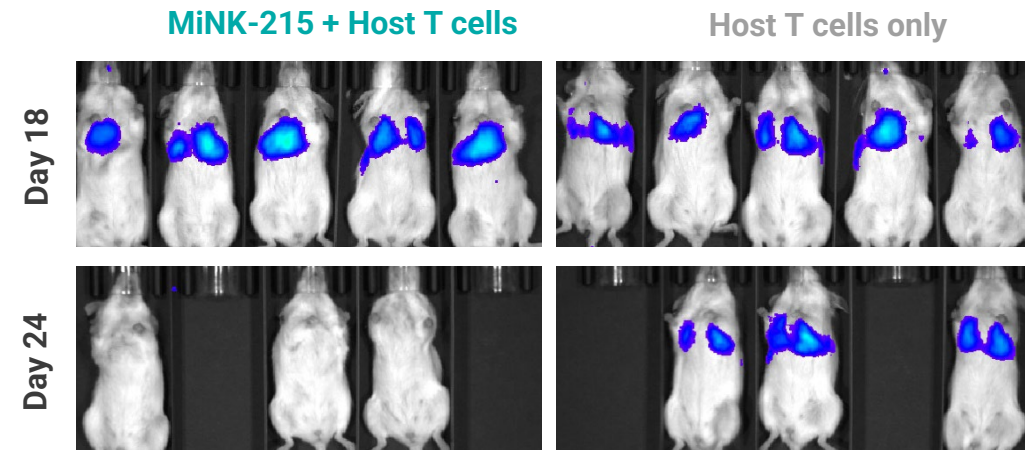


● Untreated    ● Host T cells only    ● MiNK-215 + Host T cells

### Improved Survival



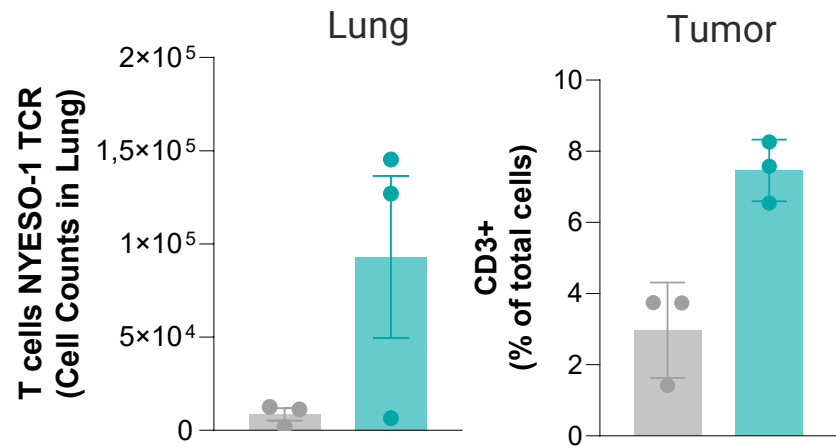
### Superior Tumor Clearance



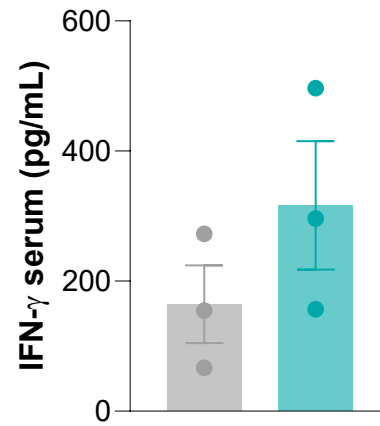
# MINK-215 PROMOTES T CELL INFILTRATION & CYTOKINE SECRETION

## DECREASES FAP EXPRESSION IN TUMOR STROMA

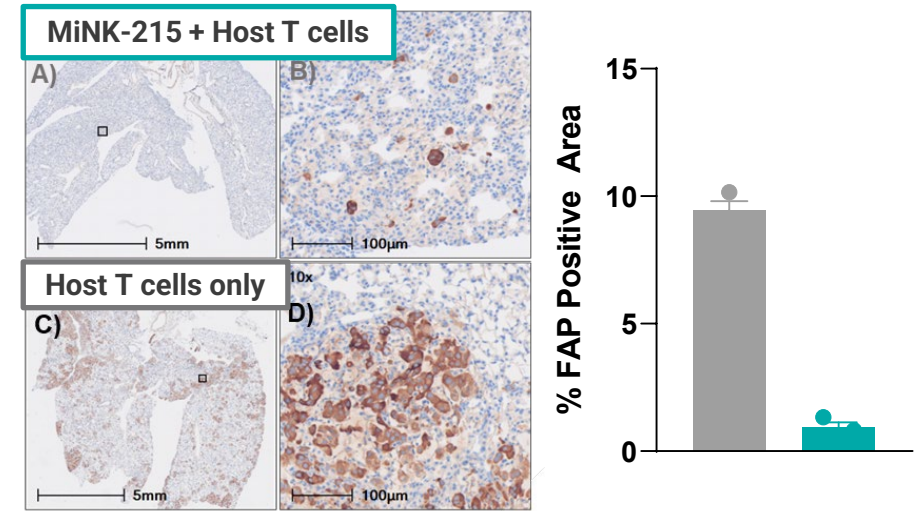
### Increased T Cell Infiltration



### Enhanced Cytokine Secretion



### Reduction in FAP+ CAFs

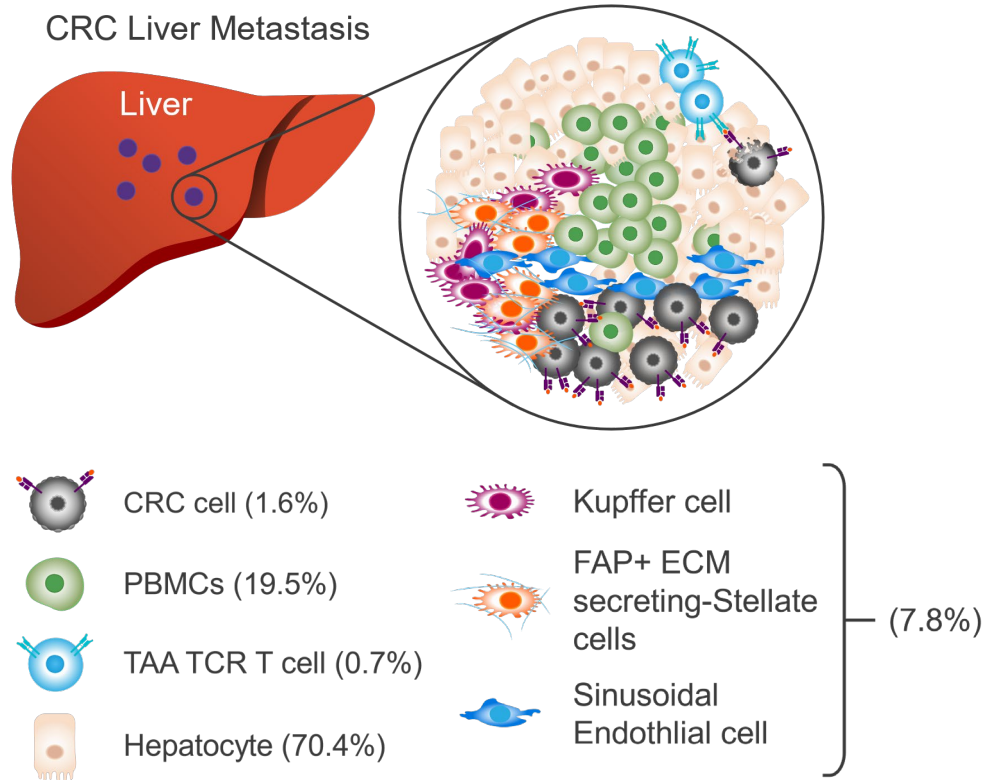


● Host T cells only    ● MiNK-215 + Host T cells

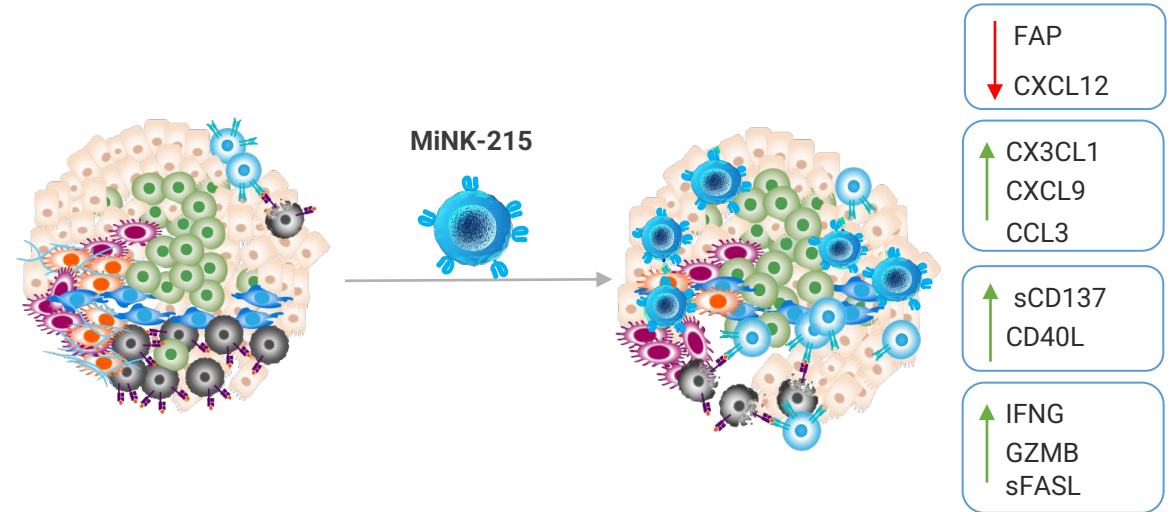
# CRC LIVER MET ORGANOIDS RECAPITULATE FAP EXPRESSION

MIMICS HUMAN METASTATIC LESIONS REFRACTORY TO IMMUNE CHECKPOINT BLOCKADE

## CRC Liver Metastasis Organoid Model



## MiNK-215 Mechanism of Action

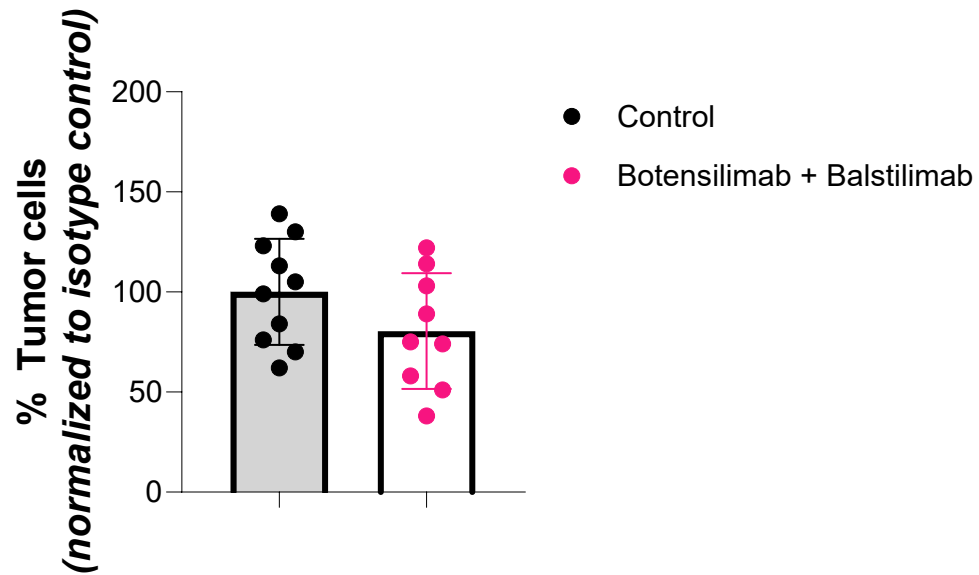


- FAP-CAR-IL-15 iNKT cells directly target and kill FAP-expressing CAFs
- FAP-CAR-IL-15 iNKT cells increase chemokines (CX3CL1, CXCL9, CCL3) associated with infiltration of T cells in the tumor microenvironment
- FAP-CAR-IL-15 iNKT cells enhance T cell activation (sCD137, CD40L) and cytotoxic function (IFN $\gamma$ , GZMB, sFASL)

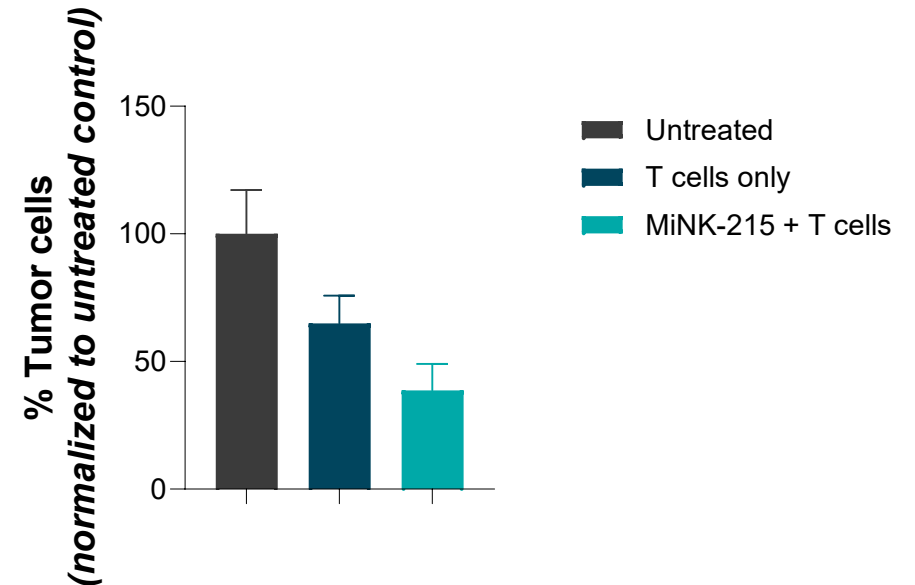
# MiNK-215 OVERCOMES LIMITATIONS OF PD-1/CTLA-4 BLOCKADE

PROMISING APPROACH FOR TREATMENT OF CRC-LIVER METASTASES

CRC Liver Metastasis Organoids are Refractory to Immune Checkpoint Blockade



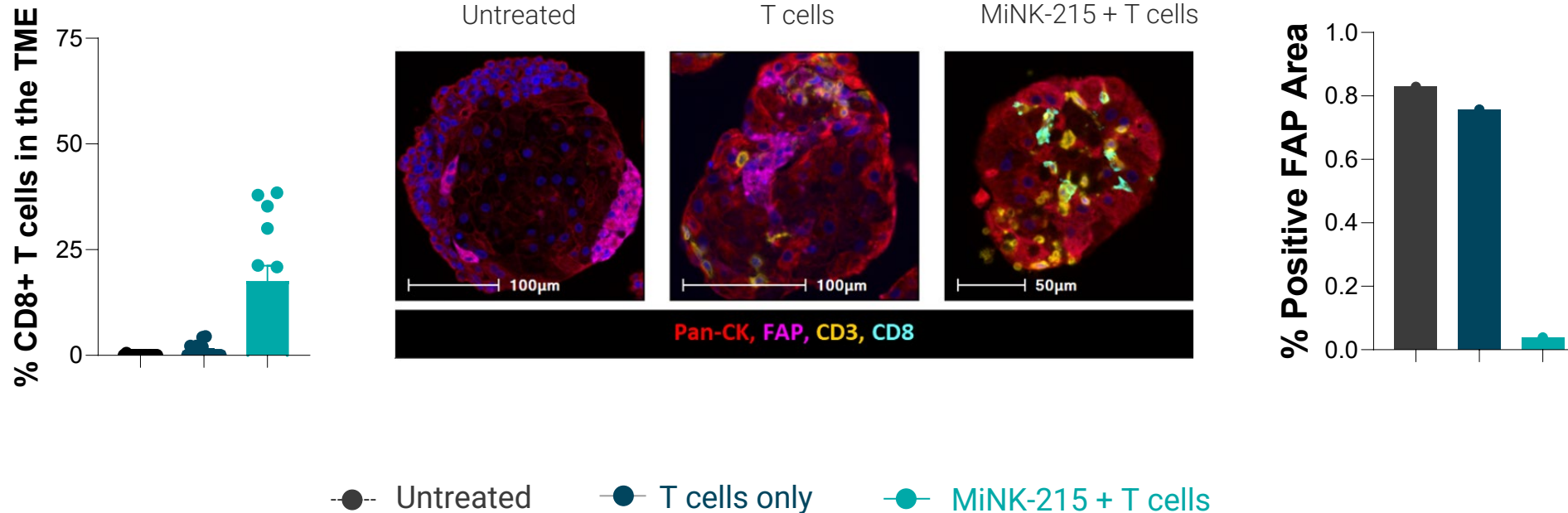
MiNK-215 Enhances Tumor Killing in Organoids



# MINK-215 OVERCOMES IMMUNE SUPPRESSION IN ORGANOIDS

HIGH T CELL INFILTRATION AND FAP+ CELL KILLING IN REFRACTORY 3D TUMOR MODEL

Increased CD8+ Infiltration and Reduced FAP expression

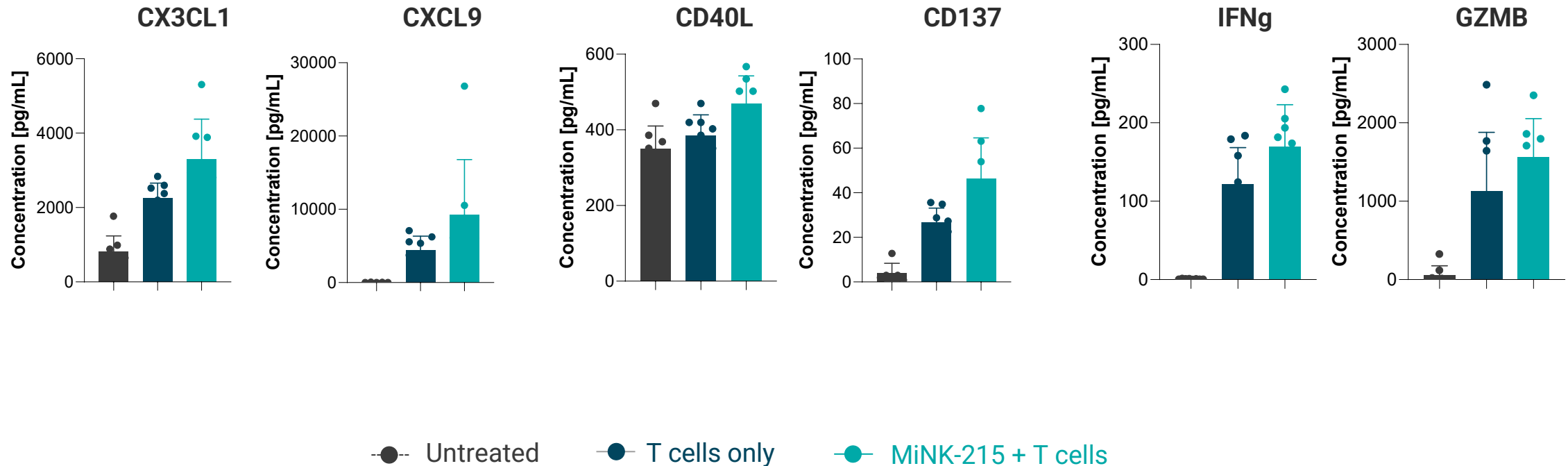


# MINK-215 PROMOTES T CELL TRAFFICKING, ACTIVATION AND EFFECTOR FUNCTION

## Increased T Cell Infiltration

## Enhanced T Cell Activation

## Increased Th1 Cytokines



# Summary & Milestones

# MINK IS PIONEERING ALLOGENEIC INKT CELL THERAPIES FOR ONCOLOGY



## **iNKTs Bridge Adaptive and Innate Immunity**

Directly attack tumor cells, recruit host immunity, reshape tumor microenvironment



## **Broad Therapeutic capability**

Opportunities in oncology and immune-mediated diseases



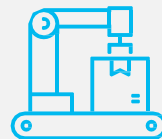
## **Clinical Proof-of-Concept**

3 Phase 1 trials show tolerability and immune-modulating activity



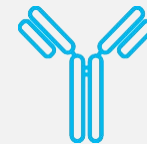
## **Proprietary Cell Engineering**

Platform for discovery of CARs, TCRs, and bispecific engagers



## **Proprietary Manufacturing at Scale**

Efficient isolation process from healthy donors can generate >5,000 doses per donor















## **Access to Validated Immuno-oncology Therapies**

Access to Agenus' immuno-oncology antibodies for combinations



# NEAR TERM-MILESTONES

	2023		2024		
<b>agenT-797</b> Native iNKT	 Initial clinical and persistence readout in solid tumors	 Initiate Phase 2 in 2L gastric cancer	 Launch Phase 2 in viral ARDS (externally funded)	 Preliminary data readout for 2L Gastric cancer	 Launch Phase 1/2 combination studies with BOT/BAL in solid tumors
<b>MiNK - 215</b> FAP-CAR	 Proof-of concept in vivo studies in solid tumor model	 Analytical and Process Development activities	 IND-enabling studies	 Universal Donor selection for Drug Product	 Planned IND Filing
<b>MiNK - 413</b> BCMA-CAR	 Proof-of concept studies in multiple myeloma model		 Explore strategic partnering in oncology and non-oncology indications		