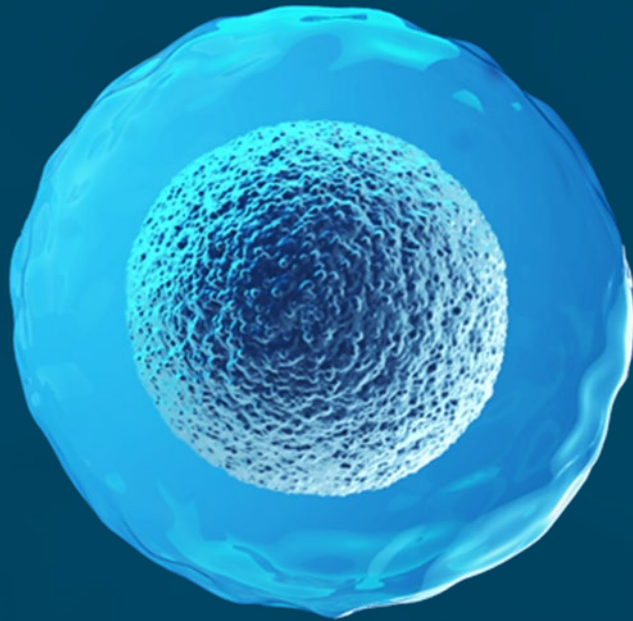




Change lives through living medicines

Corporate Overview

December 2023

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 THERAPEUTICS HIGHLIGHTS



Unique Cell Therapy Platform

Off-the-shelf, potent invariant natural killer T (iNKT) cells administered without lymphodepletion or host rejection



Robust Pipeline of Allogeneic Products

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



Clinically Advanced Programs

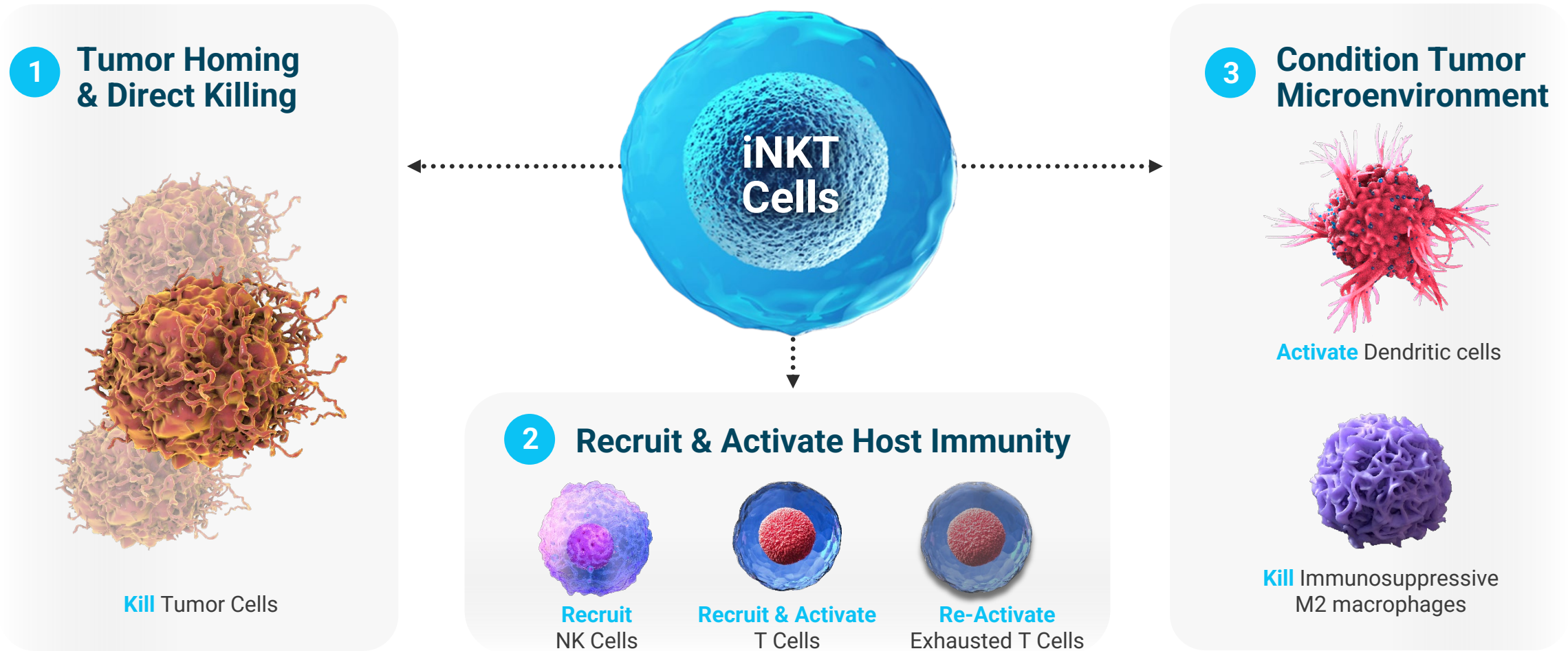
Ongoing clinical trials in oncology & immune-mediated diseases demonstrate clinical benefit with favorable safety



Proprietary Manufacturing

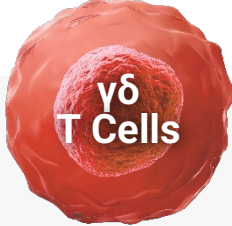
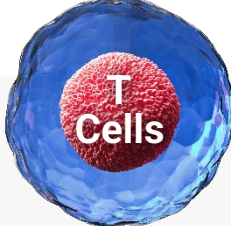
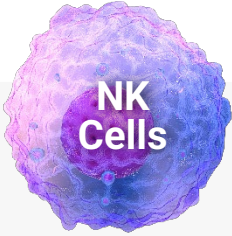
In-house, scalable, closed automated process to produce functional iNKTs at scale

INKT CELLS EMPLOY COMPLEMENTARY MECHANISMS TO TARGET CANCER



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 INKTS DEMONSTRATED IN THE CLINIC

EASILY ADMINISTERED, SAFE AND DURABLE RESPONSES



**No
lymphodepletion**
prior to
administration



**Unprecedented
persistence**
of up to 6 months
**independent of
HLA matching**



**Long-term
disease
stabilization**
(6M+) in
advanced solid
tumors refractory
to prior standard
of care



**First immune cell
therapy**
to improve
survival in patients
with
respiratory
distress syndrome
(>70% vs 10-39%
control)



**Excellent safety
profile**
No GvHD, CRS or
ICANS

INKT CELLS CAN BE ARMORED TO ENHANCE TUMOR KILLING

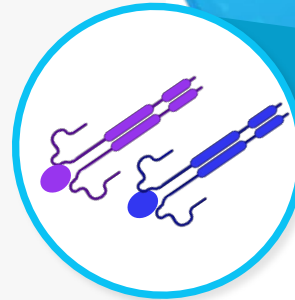
CAR/TCR Engineering

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



Cytokine Engineering

- In vivo expansion
- Improved persistence



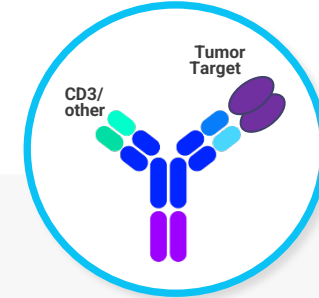
**iNKT
Cells**



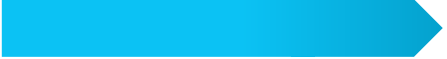
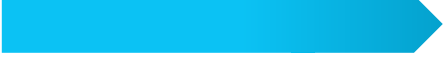
Bispecific Engagers

Enhanced activity via synergies with

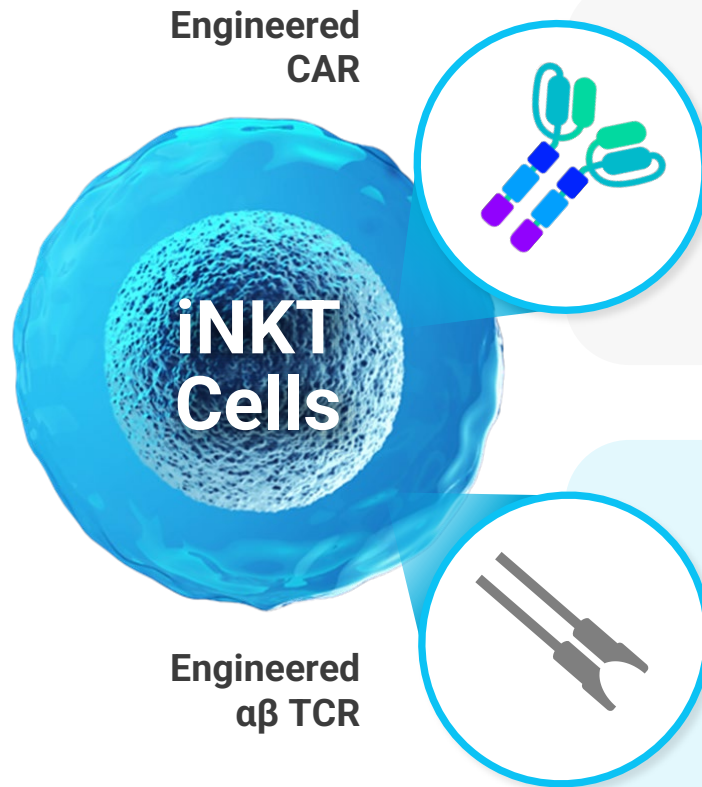
- CD3 based engagers
- Proprietary iNKT engagers



INNOVATIVE PIPELINE WITH NATIVE AND ENGINEERED INKTS

Product	Target	Indication and Approach	Preclinical	IND-enabling	Phase 1/2	Latest & Upcoming Milestones
agenT-797	Native iNKT	Solid tumors ± anti-PD1				Updated data at SITC 2023
		Gastric cancer + SOC ± BOT/BAL ¹				Trial initiation 2023
		Acute Respiratory Distress Syndrome (ARDS)				Updated data at ATS 2023
MiNK-215	FAP CAR	Solid tumors				- Potential IND filing 2024 - Updated data at ASGCT 2023
MiNK-413	BCMA CAR	Multiple myeloma				IND ready 2024
MiNK-PRAME-TCR	PRAME TCR	Solid tumors				Candidate nomination 2024
MiNK-Engagers	Undisclosed	Solid tumors				Candidate nomination 2024

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



Memorial Sloan Kettering
Cancer Center



PRINCIPAL INVESTIGATOR:
Dr. Yelena Janjigian
Chief Gastrointestinal Oncology

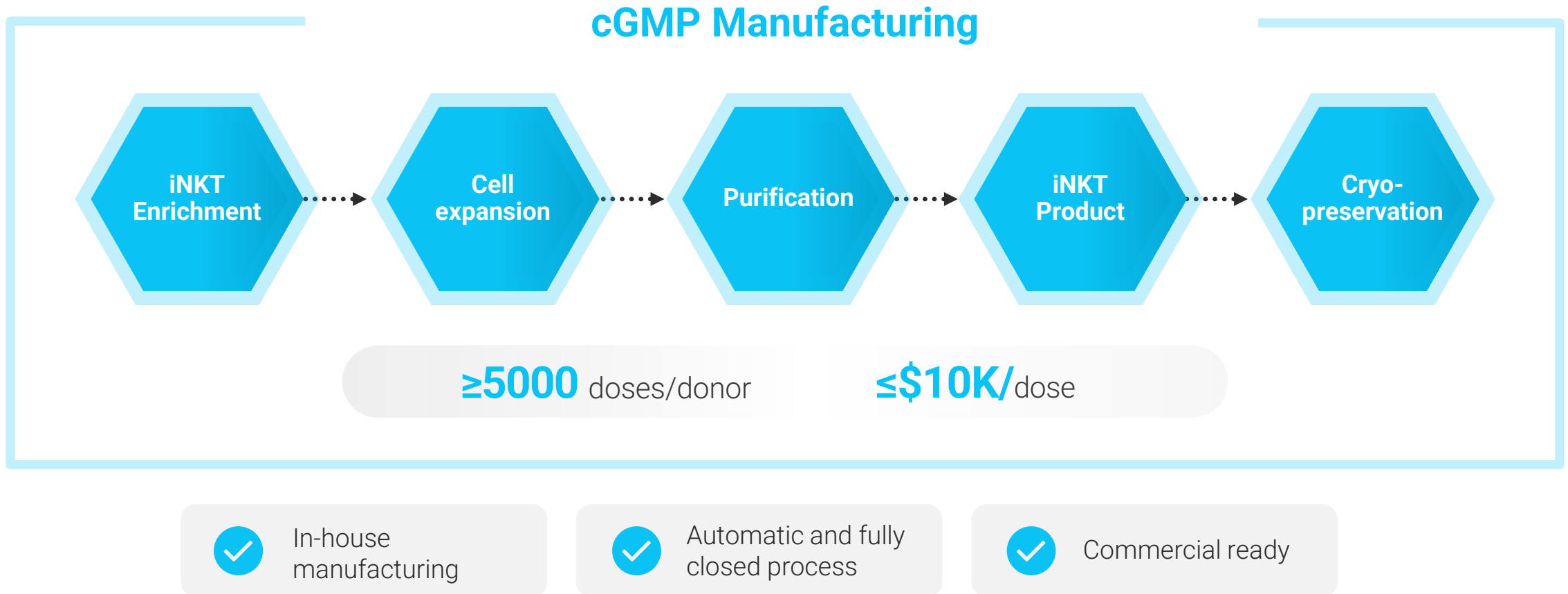
Phase 2: agenT-797 + chemotherapy ± PD-1/CTLA-4 in Gastric Cancer



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



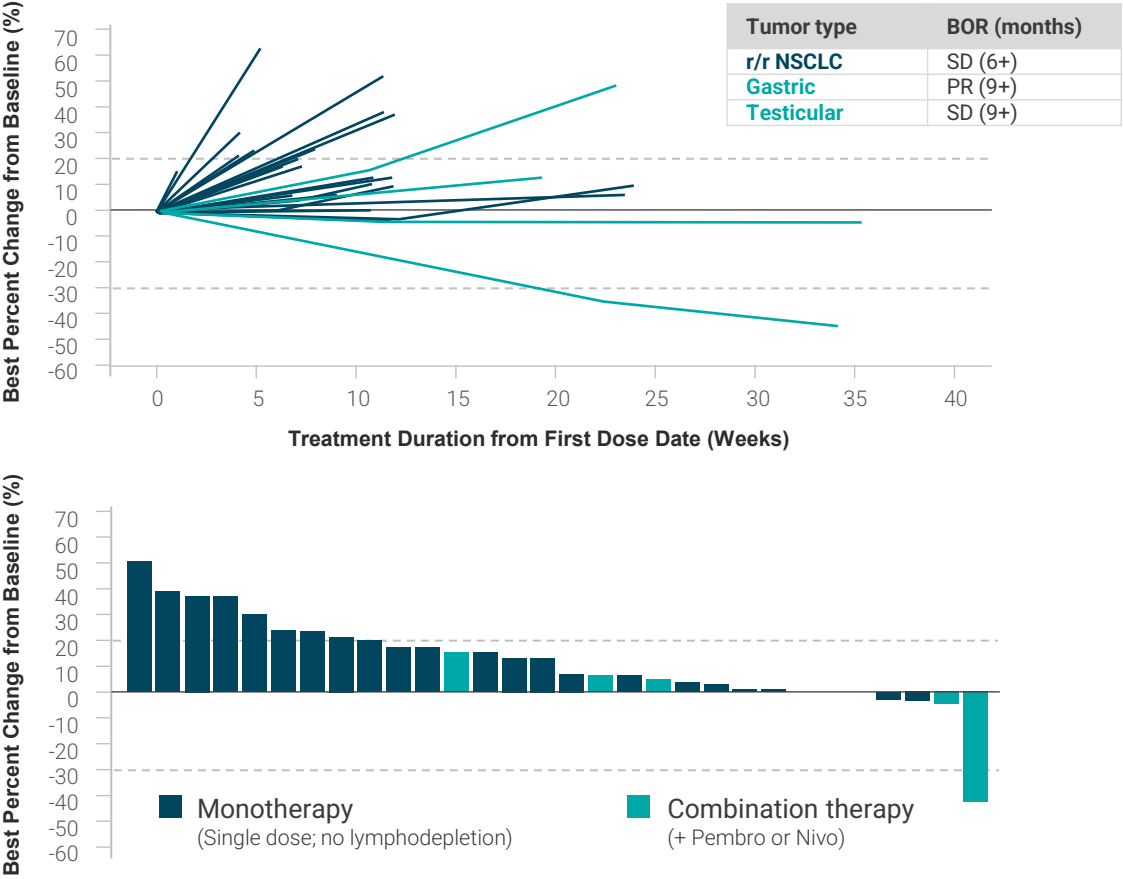
agenT-797

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

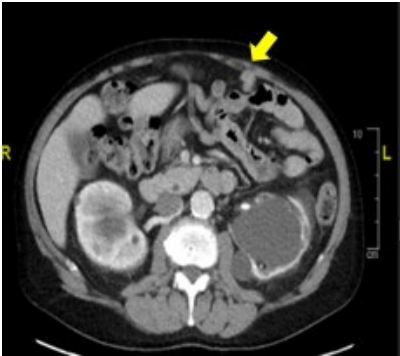
PARTIAL RESPONSE IN PD-1 REFRACTORY GASTRIC CANCER

42% TARGET LESION REDUCTION AT 9 MONTHS; RESPONSE ONGOING

Gastric Cancer Patient

Patient Characteristics	75-year-old male - Failed prior PD-1 therapies	
Prior Therapies	- Pembrolizumab - FOLFOX + nivolumab + oxaliplatin	PD SD
Treatment	- Single dose of agentT-797 + nivolumab (200mg) - DL1: 4.3 x 10⁶ cells/kg	
Response	33% target reduction at 6 months 42% target reduction at 9 months - PFS: 10M+	

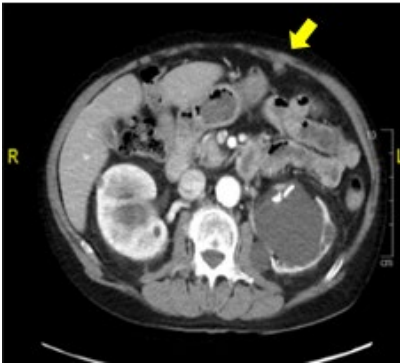
Baseline



Month 3



Month 6



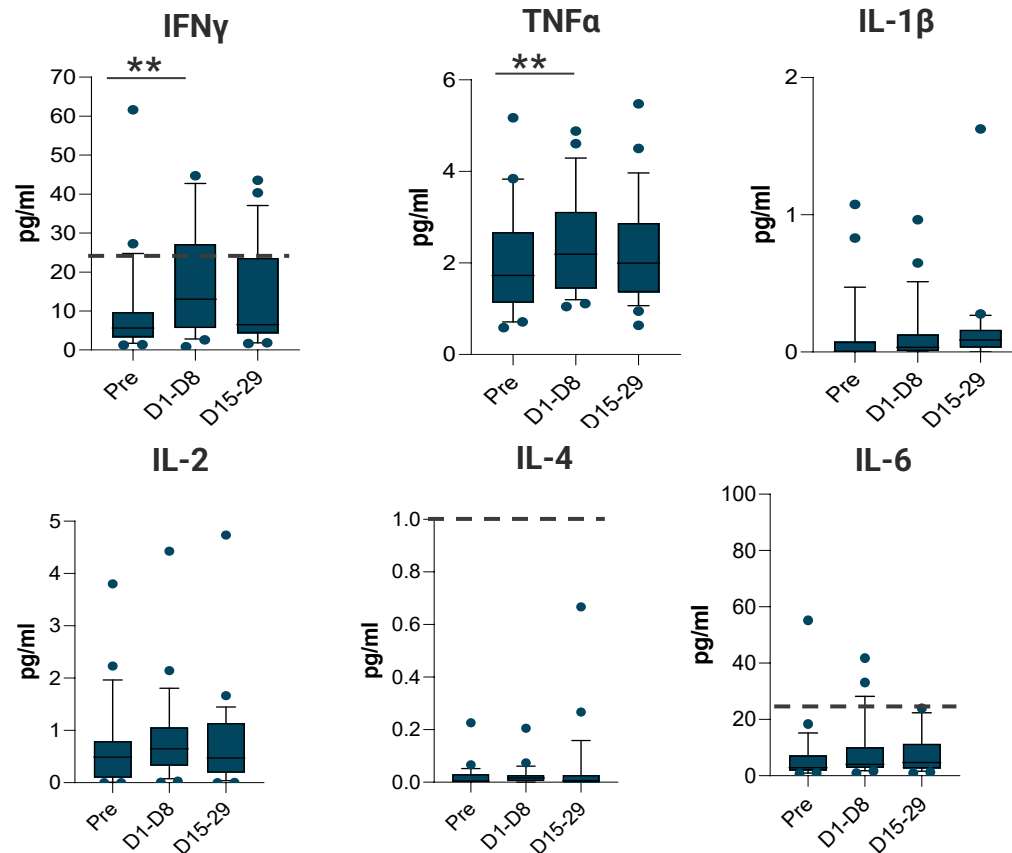
Month 9



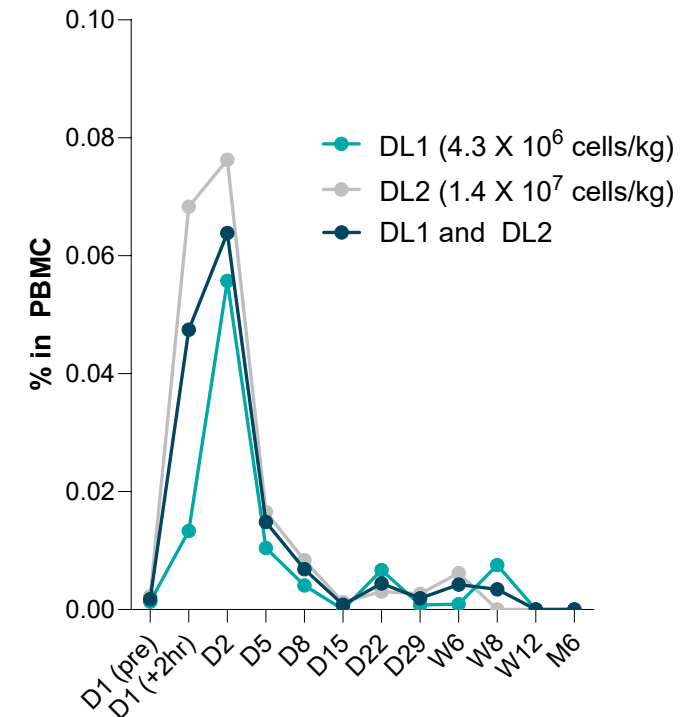
AGENT-797: PROLONGED PERIPHERAL PERSISTENCE AND TH1 CYTOKINE PROFILE

ENHANCED IFN γ AND TNF α AND UP TO 6 MONTHS PERSISTENCE

Enhanced Th1 Cytokine Profile



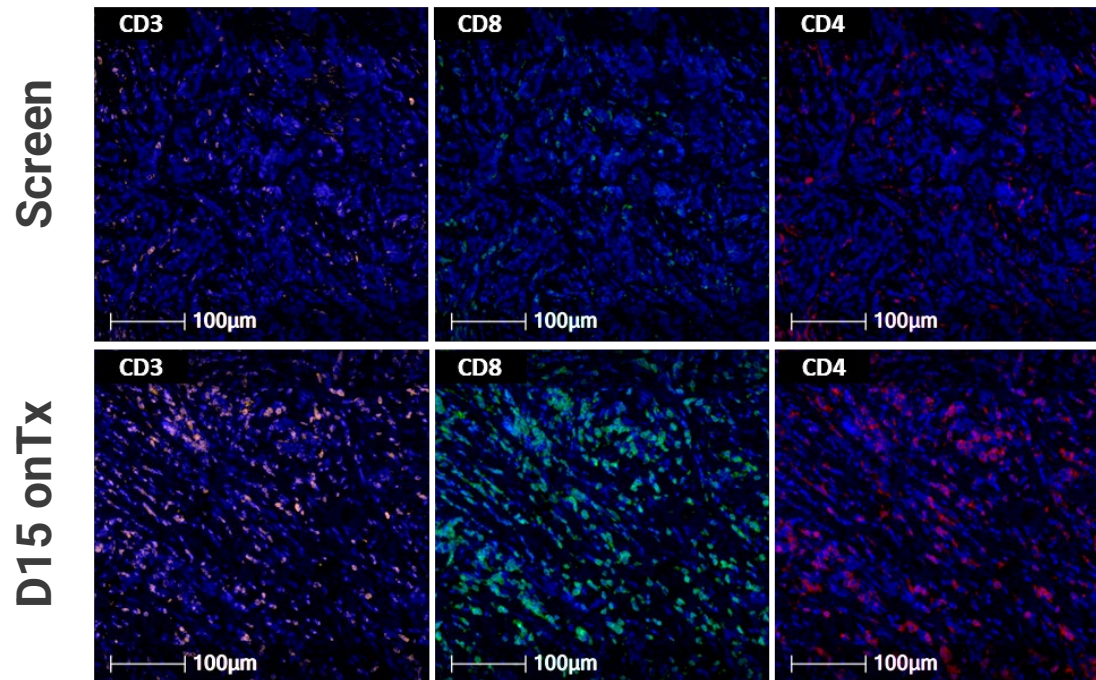
Long Peripheral Persistence



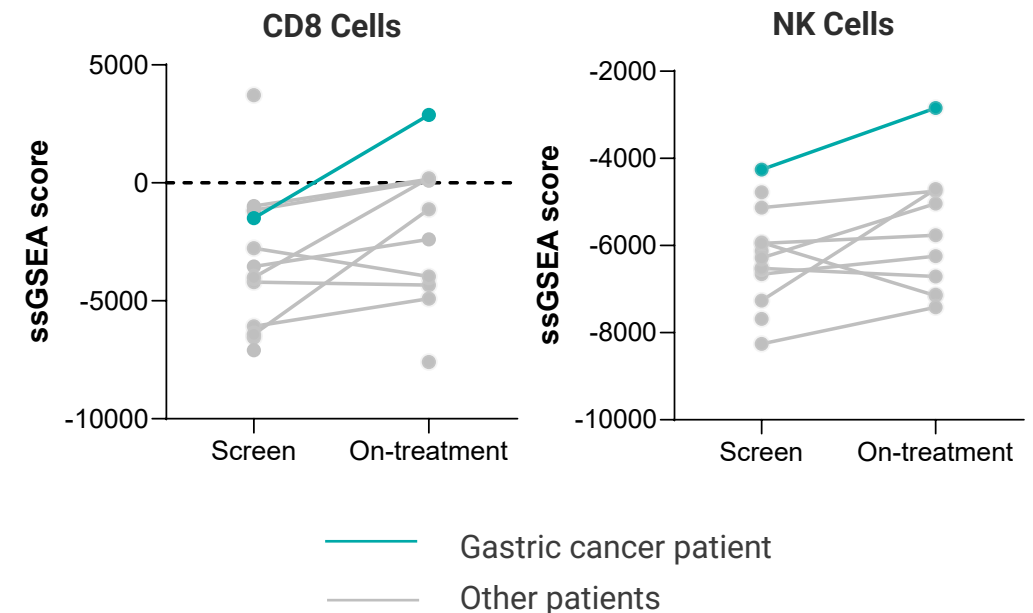
AGENT-797: PROMOTES IMMUNE CELL INFILTRATION IN TUMOR

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

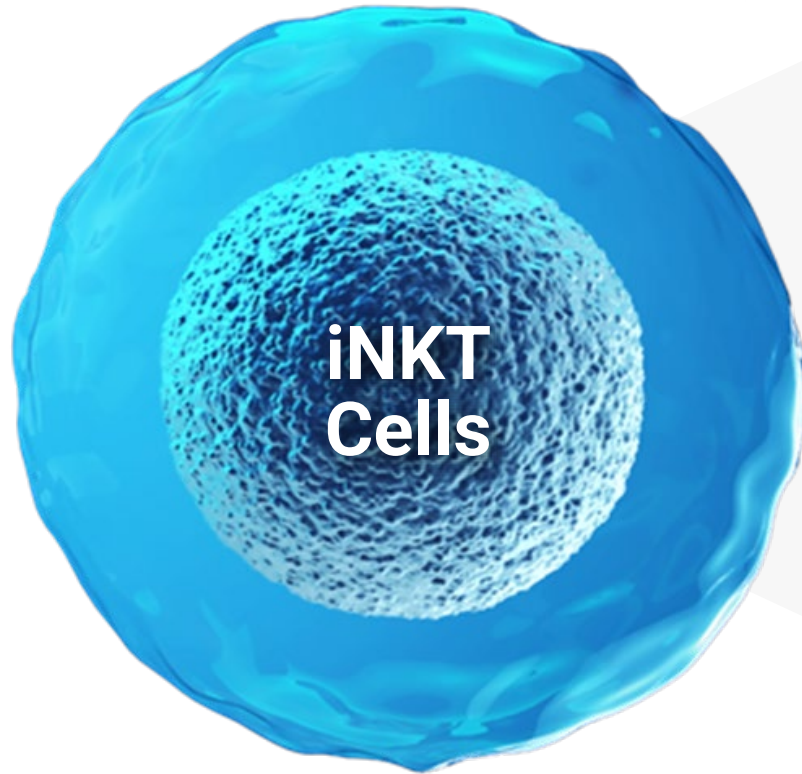
NO DLTS AND FEW RELATED ADVERSE EVENTS

	Total	agenT-797 MonoTx		agenT-797 + anti-PD-1	
Dose level		DL1: 4.3 x 10 ⁶ cells/kg	DL2: 1.4 x 10 ⁷ cells/kg	DL1: 4.3 x 10 ⁶ cells/kg	DL2: 1.4 x 10 ⁷ cells/kg
	N = 34	N = 8	N = 20	N = 3	N = 3
AE, n (%)	32 (94)	8 (100)	18 (90)	3 (100)	3 (100)
Any AE of grade ≥ 3	19 (56)	7 (88)	11 (55)	0	1 (33)
	3 (9)	0	2 (10)	0	1 (33)
	1 (3)	0	0	0	1 (33)
TRAE, n (%)	9 (27)	3 (38)	2 (10)	2 (67)	2 (67)
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
TRAE by System Organ Class, n (%)					
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

agenT-797

Clinical data in immune dysfunction

INKT CELLS PLAY A PROTECTIVE ROLE IN INFECTION AND INFLAMMATION



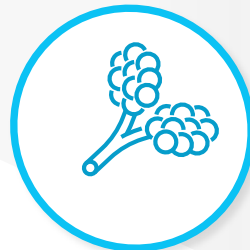
Infections

- 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



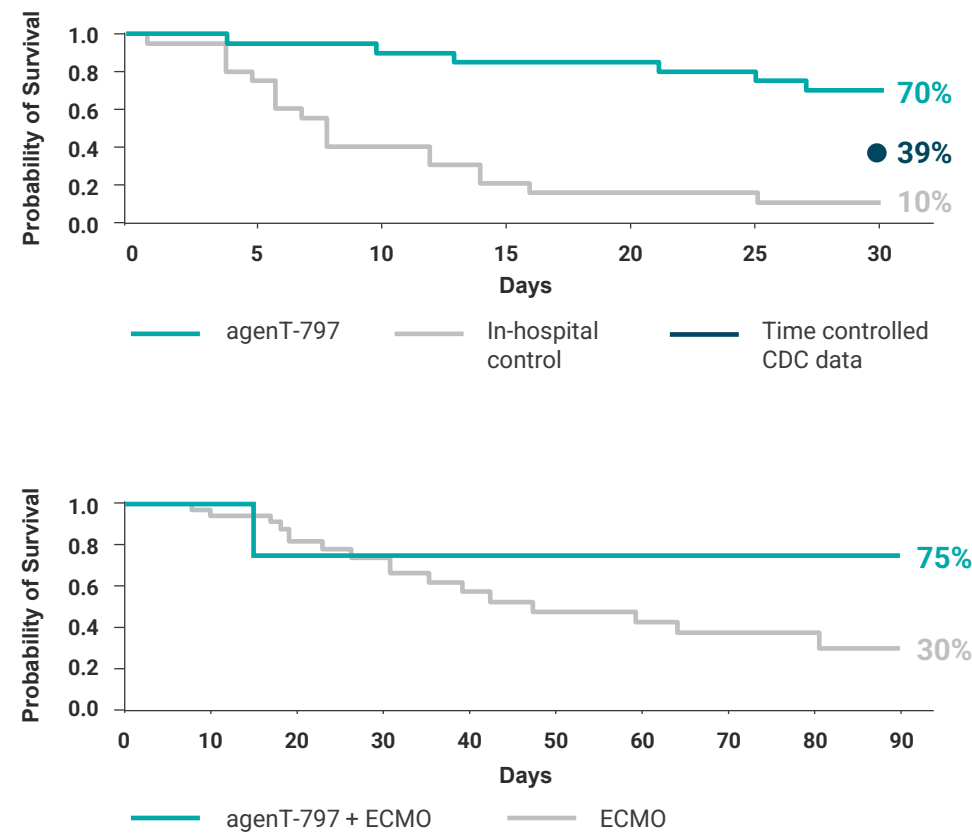
Pulmonary Fibrosis & Lung Dysfunction

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

AGENT-797 IMPROVES SURVIVAL AND LUNG FUNCTION IN SEVERE CARDS

AGENT-797 TREATMENT: 30-DAY SURVIVAL RATE OF 70% (VS 10% SITE-BASED CONTROL)

Increased Survival vs Case control



Reduced Incidence of Secondary Infections, including Pneumonia

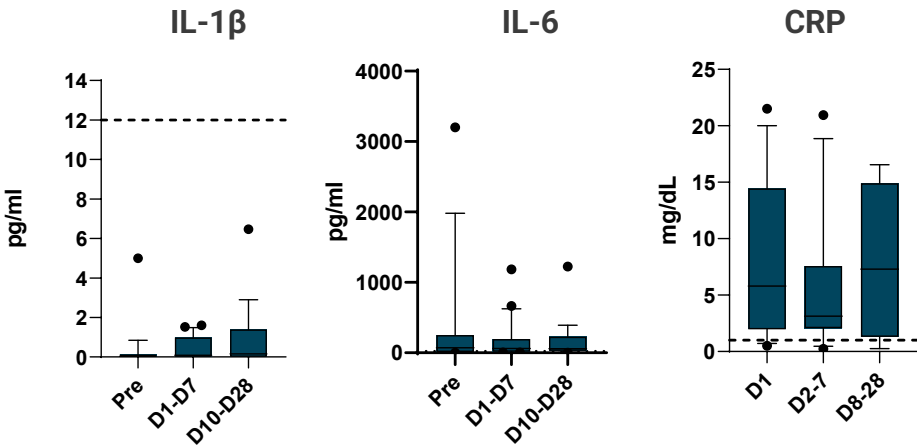
	Dose Level 1 (n=3)	Dose Level 2 (n=4)	Dose Level 3 (n=13)
	n (%)	n (%)	n (%)
Pneumonia	2 (67)	3 (75)	2 (15)
Bacteraemia	2 (67)	0	1 (8)
Urinary tract infection	0	3 (75)	1 (8)
Fungaemia	0	1 (25)	1 (8)
Cytomegalovirus viraemia	0	0	1 (8)
Lung abscess	1 (33)	0	0
Pneumonia klebsiella	0	1 (25)	0
Sepsis	1 (33)	0	0
Septic shock	0	0	1 (8)
Upper respiratory tract infection	1 (33)	0	0

AGENT-797 IS WELL TOLERATED IN SEVERE CARDS PATIENTS

No Significant Adverse Events

	agentT-797 ± ECMO (n=20)	agentT-797 + ECMO (n=4)
	n (%)	n (%)
AE	20 (100)	4 (100)
Any AE of grade ≥ 3	19 (95)	4 (100)
TRAE	5 (25)	0 (0)
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



SIGNIFICANT IMPROVEMENT NOTED IN CARBAPENEM-RESISTANT PNEUMONIA

PATIENT CLEARED LUNG INFECTION AND STOPPED VV-ECMO 13 DAYS POST AGENT-797 INFUSION

Carbapenem-resistant Severe ARDS Patient

Patient Characteristics	• 21-year-old male • Severe ARDS • Carbapenem resistant Pseudomonal Pneumonia on VV-ECMO
Treatment	• Single dose of agentT-797 • DL2: 1 x 10⁹ cells
Response	• Cleared Infection • Stopped ECMO 13 days post-infusion

Pre-infusion



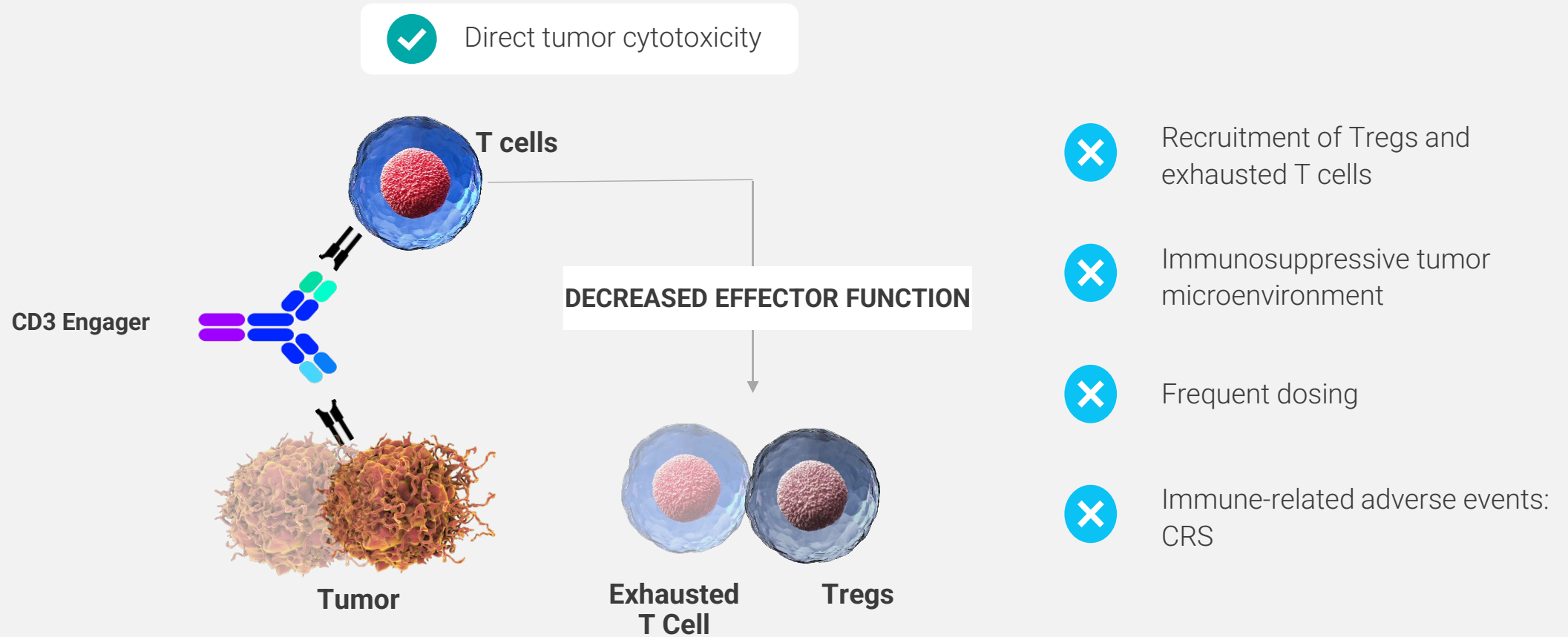
Post agentT-797 infusion



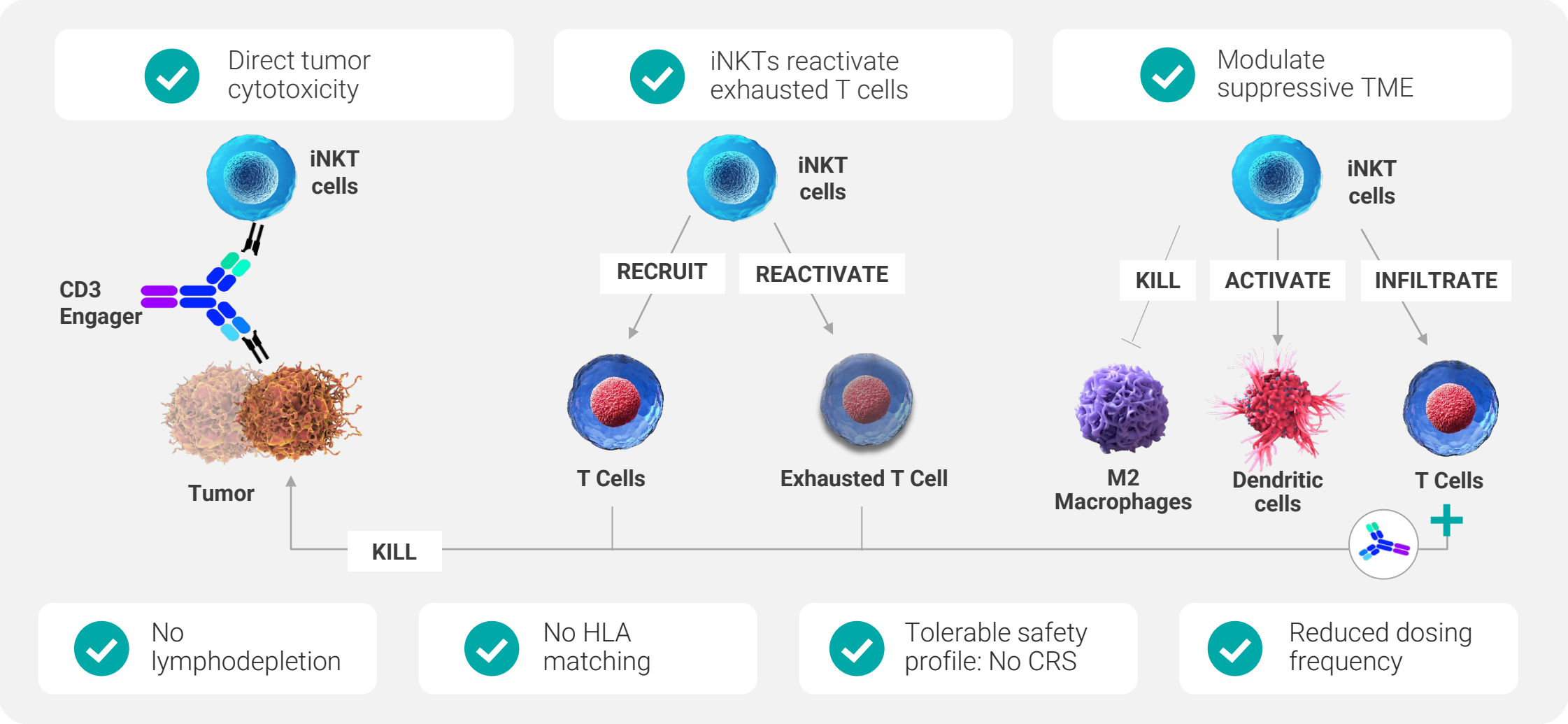
agenT-797

Preclinical data on synergy with CD3 engagers

LIMITATIONS OF CURRENT T CELL ENGAGERS

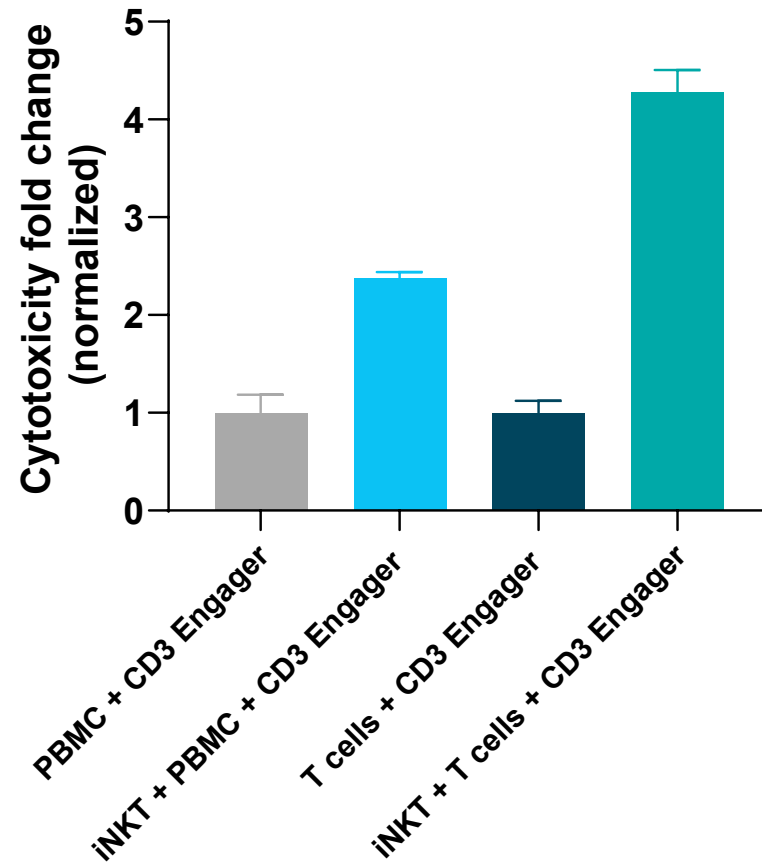


INKTS POTENTIATE A SUSTAINED IMMUNE RESPONSE



INKTS AND CD3 ENGAGER SHOW 2X TUMOR KILLING

INKT CELLS WILL SYNERGIZE WITH PATIENTS' ENDOGENOUS IMMUNE CELLS



- PBMCs and T cells mimic host immune system
- Cytotoxicity normalized to CD3 engager activity with PBMCs or T cells aligning with current therapy
- CD3 engager + allogeneic iNKT cells (as low as 0.2×10^5) boost cytotoxicity >2 fold

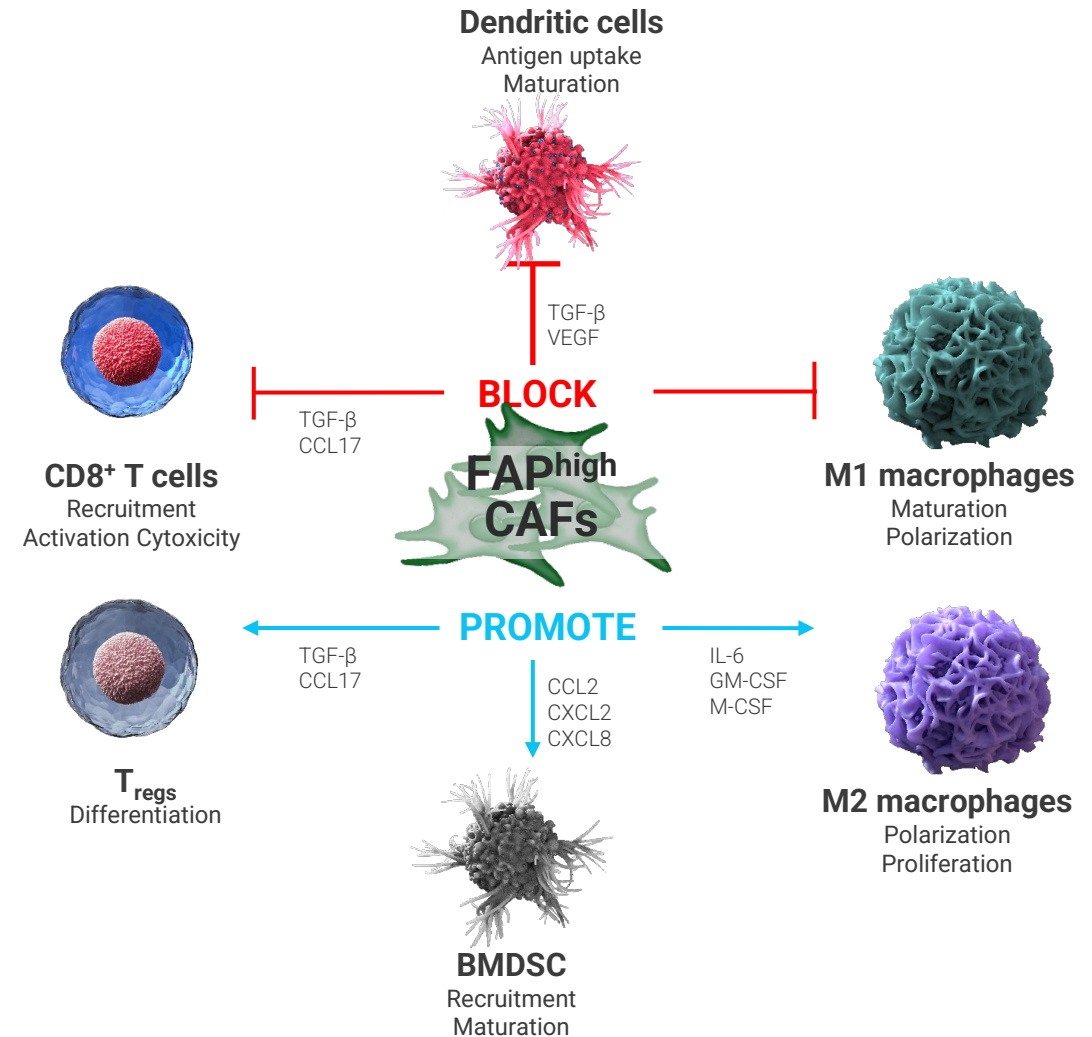
MiNK-215

Preclinical data from Engineered FAP-CAR-iNKT cells

TARGETING TUMOR-PROMOTING STROMAL CELLS IN SOLID TUMORS

FAP^{HIGH} CAFs OCCUR IN >90% OF EPITHELIAL-DERIVED TUMORS

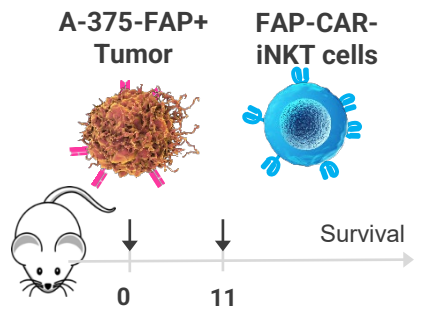
- FAP^{high} CAFs are highly immune-suppressive and tumor-promoting in the TME
- FAP^{high} CAFs secrete a variety of cytokines to modulate immune activity
- Targeting FAP^{high} CAFs may result in tumor cell death in highly stromagenic cancers without IO success



MINK FAP-CAR-INKT PROMOTES SURVIVAL IN FAP+ TUMOR-BEARING MICE

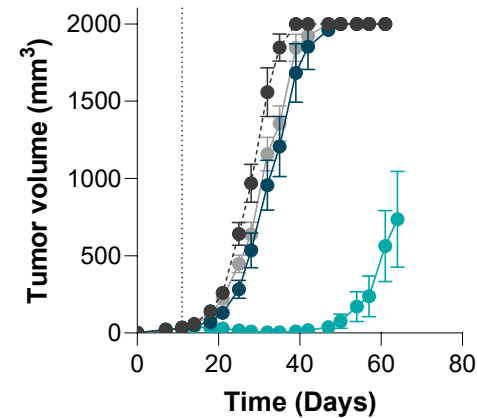
SUPERIOR ANTI-TUMOR ACTIVITY TO CLINICAL REFERENCE CAR (SIBROTUZUMAB)

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



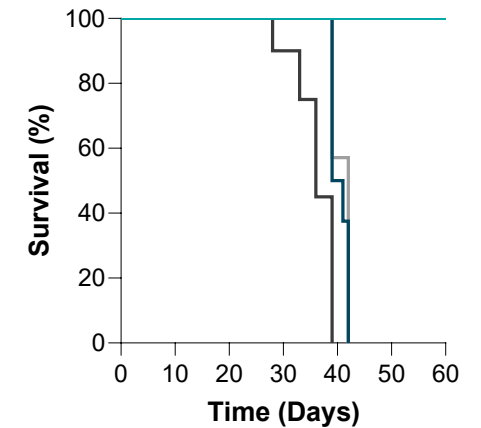
●-- Untreated

●-- Negative Control BCMA-CAR-iNKT

●-- Sibrotuzumab-CAR-iNKT

●-- MiNK-FAP-CAR-iNKT

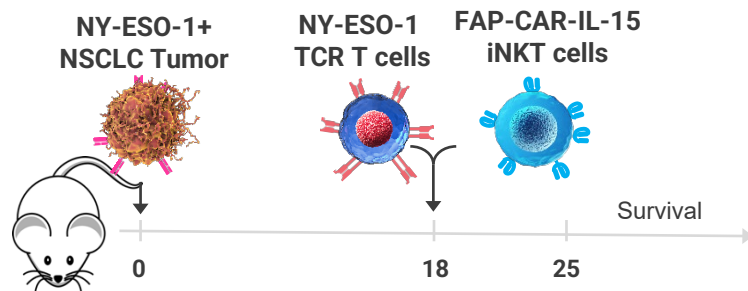
Superior Survival



NSCLC MOUSE ORTHOTOPIC MODEL RECAPITULATES TUMOR STROMA

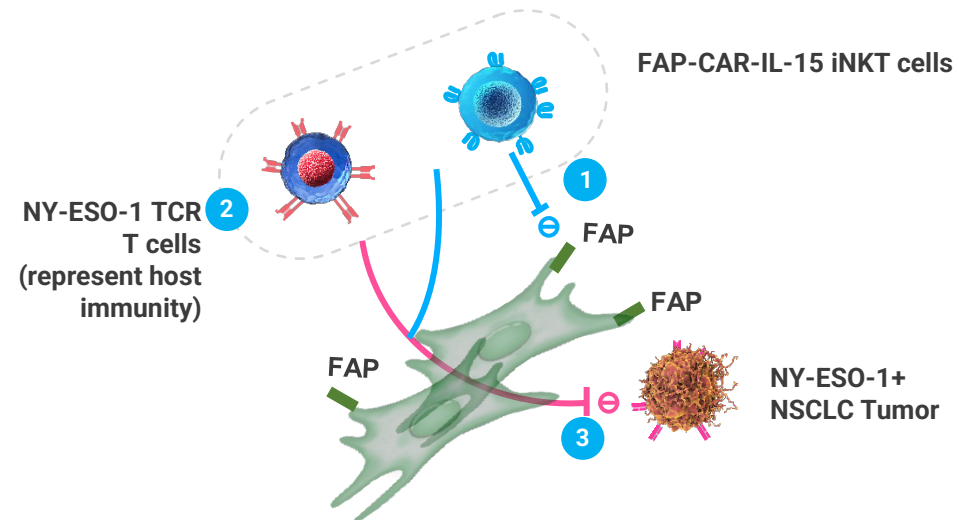
IN VIVO ASSESSMENT OF TARGETING FAP+ CAFs

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

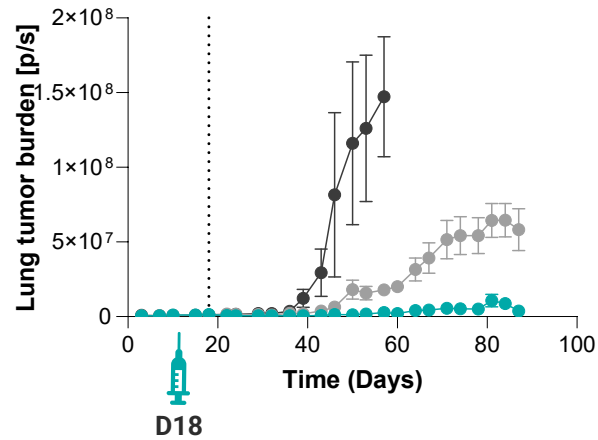


- 1 FAP-CAR-IL-15 iNKT cells directly target and kill FAP-expressing CAFs
- 2 FAP-CAR-IL-15 iNKT cells recruit T cells to the tumor microenvironment
- 3 T cells infiltrate the tumor tissue and directly kill tumor cells

MINK-215 HALTS TUMOR GROWTH AND IMPROVES SURVIVAL IN MICE

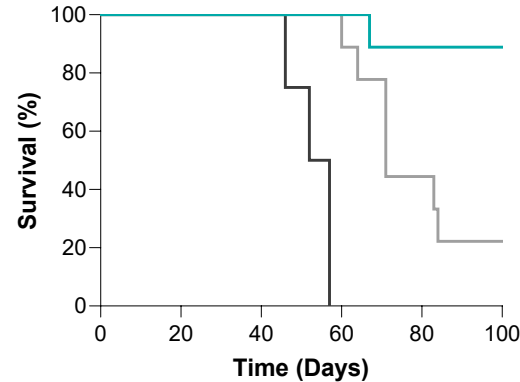
SYNERGIZES WITH HOST T CELLS FOR ENHANCED ACTIVITY

Reduced Tumor Burden

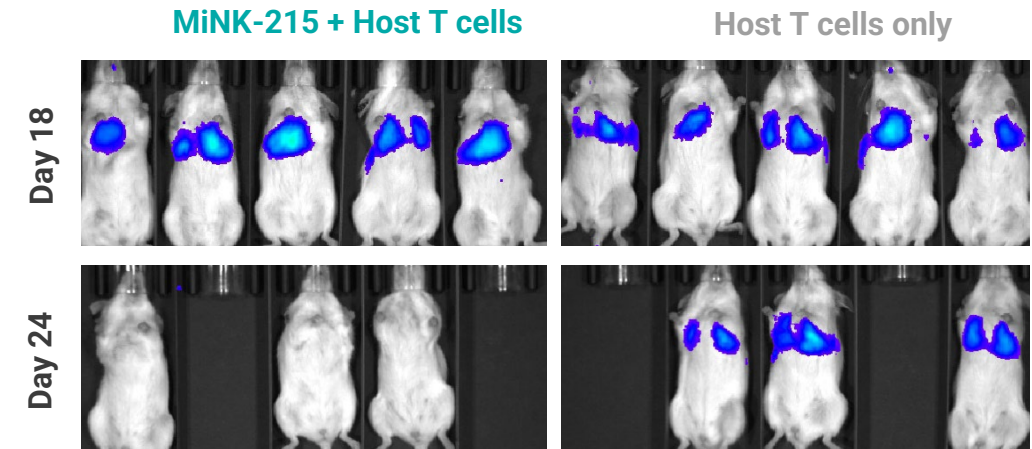


● Untreated ● Host T cells only ● MiNK-215 + Host T cells

Improved Survival



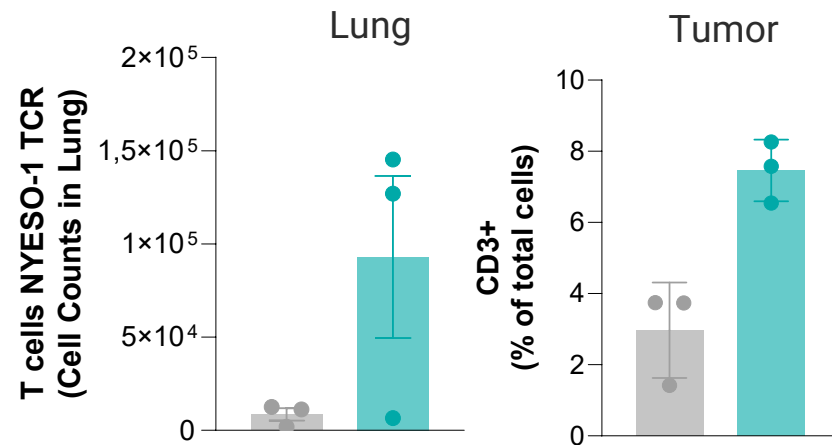
Superior Tumor Clearance



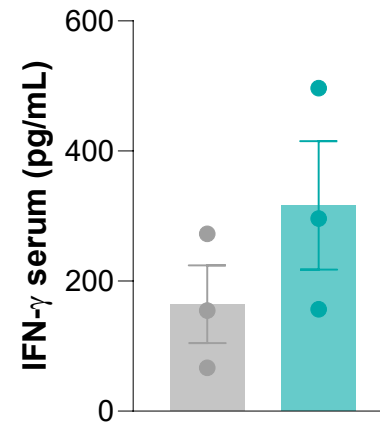
MINK-215 PROMOTES T CELL INFILTRATION & CYTOKINE SECRETION

DECREASES FAP EXPRESSION IN TUMOR STROMA

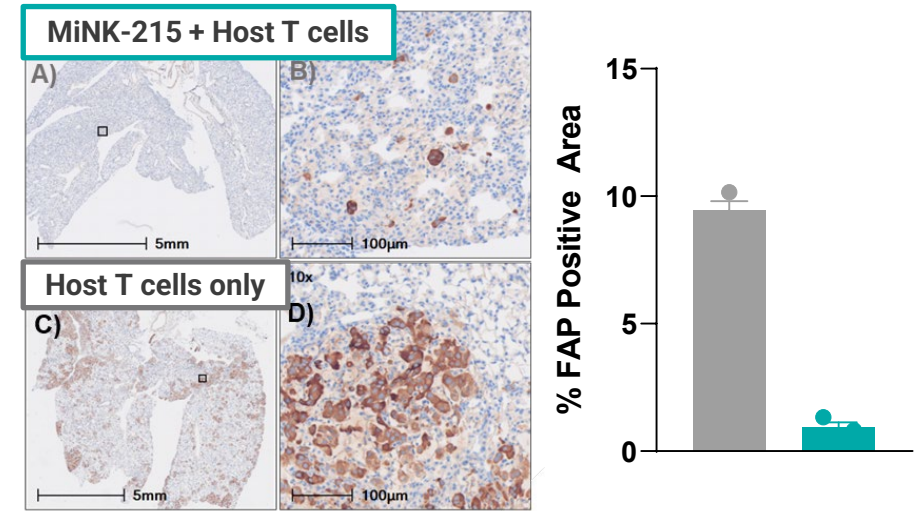
Increased T Cell Infiltration



Enhanced Cytokine Secretion



Reduction in FAP+ CAFs



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

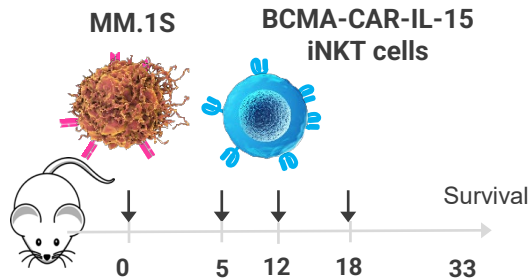
MiNK-413

Preclinical data from Engineered BCMA-CAR-iNKT cells

MINK-413 DELAYED TUMOR ENGRAFTMENT IN XENOGRAFT MICE

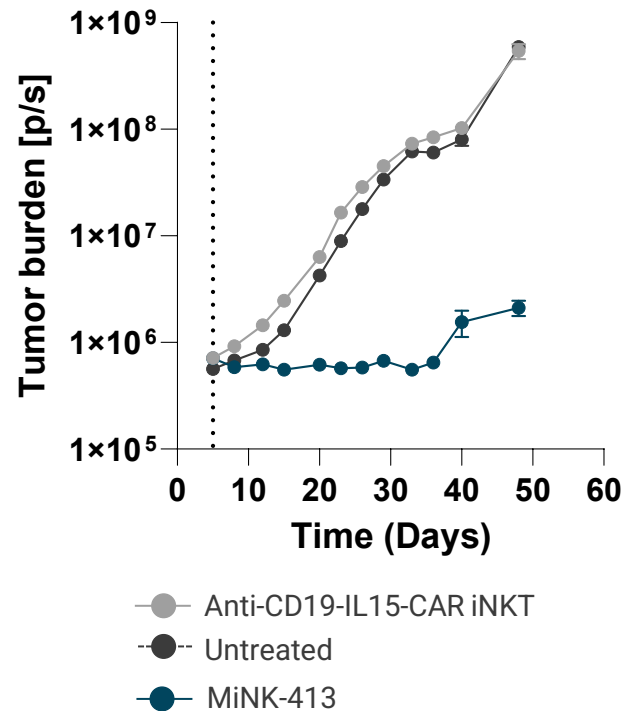
REDUCED TUMOR BURDEN AND IMPROVED SURVIVAL IN MINK-413 TREATED MICE

Multiple Myeloma Mouse Model

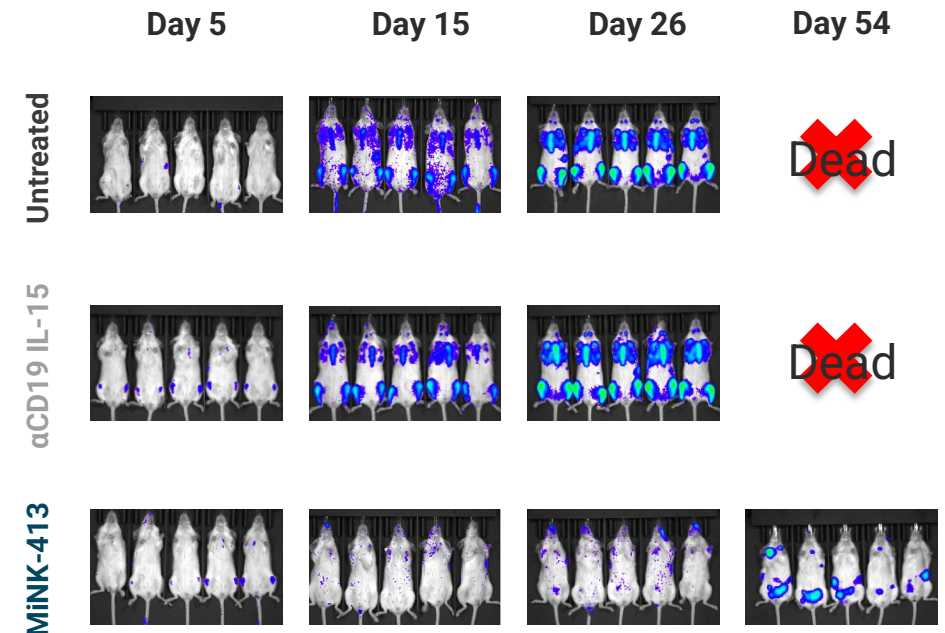


- Day 0: MM.1S tumor cells injected into NOG mice
- Day 5: First dose of BCMA-CAR-IL15 iNKT cells administered
- Day 12, 18: Additional doses of BCMA-CAR-IL-15 iNKT cells

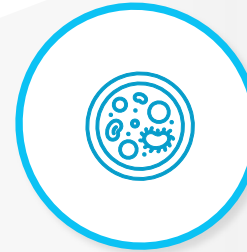
MiNK-413 Delays Tumor Growth



MiNK-413 Promotes Survival

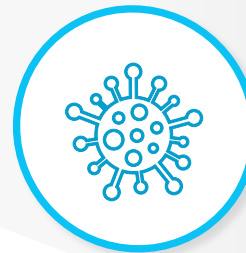


BROAD TARGET APPLICATION IN ONCOLOGY AND INFLAMMATION



Oncology

- Multiple Myeloma
- Potential synergies with non BCMA targeting CD3 engagers



Autoimmune

- SLE, Myasthenia Gravis, Amyloidosis, NMO
- Depletion of BCMA+ cells

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



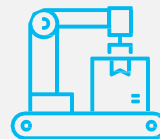
Clinical Proof-of-Concept

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 Agenesis' immuno-oncology antibodies for combinations

NEAR TERM-MILESTONES

