MEDICENNA

Master the Signal.
Navigate Complexity.
Create Cures.

Bloom Burton Healthcare Investment Conference
April 21, 2026

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Clinical Stage Immunotherapy Company

3 Lead Programs with First and/or Best-in-Class Potential

MDNA11 – Phase 1/2

Best-in-Class IL-2 Super Agonist

for Advanced Solid Tumors

36% ORR MDNA11 monotherapy

43% ORR MDNA11 + pembrolizumab

as 2/3L Tx or next line following resistance to ICI therapy

MDNA113 – IND-enabling

First-in-Class PD-1 x IL-2 Superkine

for Tumors expressing IL-13Rα2

Tumor Anchoring & Masking

One of the most exciting areas in immuno-oncology
recently validated by multi-billion-dollar pharma transactions

Bizaxofusp (MDNA55): Phase 3 Ready

An IL-4R targeted Immuno-toxin

for Recurrent Glioblastoma (rGBM)

~2x Overall Survival in Deadliest Brain Cancer, rGBM

Median OS ~13.5 months versus ~7.2 months for matched control arm

Multiple recent pharma transactions in brain cancer space

TSX: MDNA | OTCQX: MDNAF

2026 Milestones & Catalysts

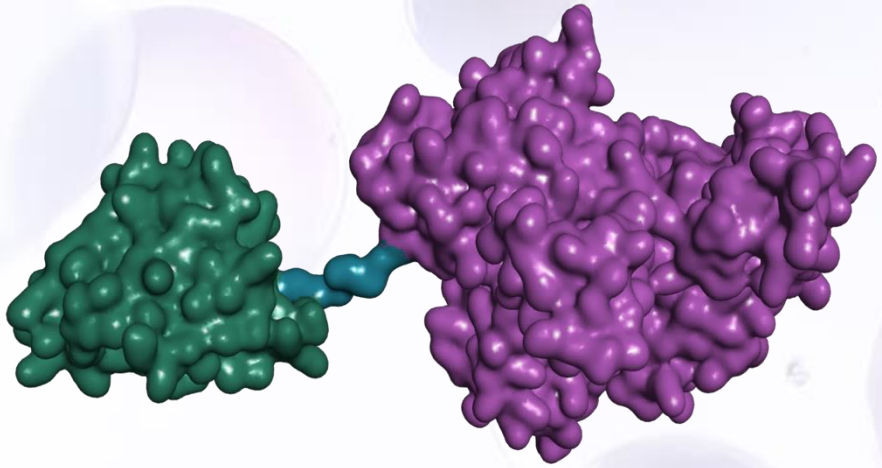
MDNA11	• PoC data post 2L/3L therapy • End of P1 meeting with FDA • Plan P2 registration study with FDA • Neoadjuvant melanoma Ph1b data
MDNA113	• NHP safety/PK/PD results • File IND for first-in-human study
Bizaxofusp	• Partnership • Commence Phase 3 rGBM trial

Funded into Calendar Q3 2026

Creating value by advancing Superkines

A Clinical-Stage Leader in Cytokine Engineering

PLATFORM	CANDIDATE	INDICATION	PRECLINICAL / PHASE 1 / PHASE 2 / PHASE 3	STATUS	PARTNER
Superkine™ BiSKIT™	**Bizaxofusp** IL-4R-Toxin Fusion	Recurrent Glioblastoma		**Pursuing partnership to commence Phase 3**	
Superkine™	**MDNA11** IL-2 Super Agonist	Various solid tumors	**MDNA11 monotherapy**; **MDNA11 + pembrolizumab** — *ABILITY-1 Phase 1/2 Basket Study*	**Plan first registrational trial in 2L/3L checkpoint refractory cancers**	*Clinical Collaborations* MERCK
			MDNA11 + nivolumab ± ipilimumab — *NEO-CYT Phase 1b in Neoadjuvant Melanoma*	**Commence NEO-CYT study H1 2026 with data readouts in H2 2026**	Fondazione Melanoma
T-MASK™ BiSKIT™	**MDNA113** anti-PD-1 x IL-2 Masked Bispecific	Various solid tumors expressing IL-13Rα2		**IND and commence FIH in H2 2026**	
Superkine™	**MDNA209** IL-2/15 Pathway Super Antagonist	Autoimmune diseases		**Select lead**	
Superkine™	**MDNA413** IL-4/13 Pathway Super Antagonist	Inflammatory diseases		**Select lead**	



MDNA11

- A ‘β-enhanced, not-α’ IL-2 superagonist in clinical development for advanced solid tumors
- Clinical-Stage Therapy in Phase 1/2 with a Monotherapy Treatment Arm and a Combination Arm with KEYTRUDA® (pembrolizumab)

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This study is in collaboration with Merck Sharp & Dohme LLC, a subsidiary of Merck & Co., Inc., Rahway, NJ, USA.

MDNA11 is a Differentiated Next-Gen IL-2 with Best-in-Class Potential

WILD-TYPE IL-2

1998

PROLEUKIN (aldesleukin) Recombinant IL-2

- First approved IO drug for RCC and melanoma
- Limited clinical utility because of short half-life, toxicity

2ND GENERATION IL-2

2015 — Roche

2017 — Bristol Myers Squibb

2018 — sanofi

- Discontinued programs, due to poor PK/PD profiles, toxicity or lack of efficacy with PEGylation reducing potency

NEXT-GENERATION IL-2

Present — MEDICENNA

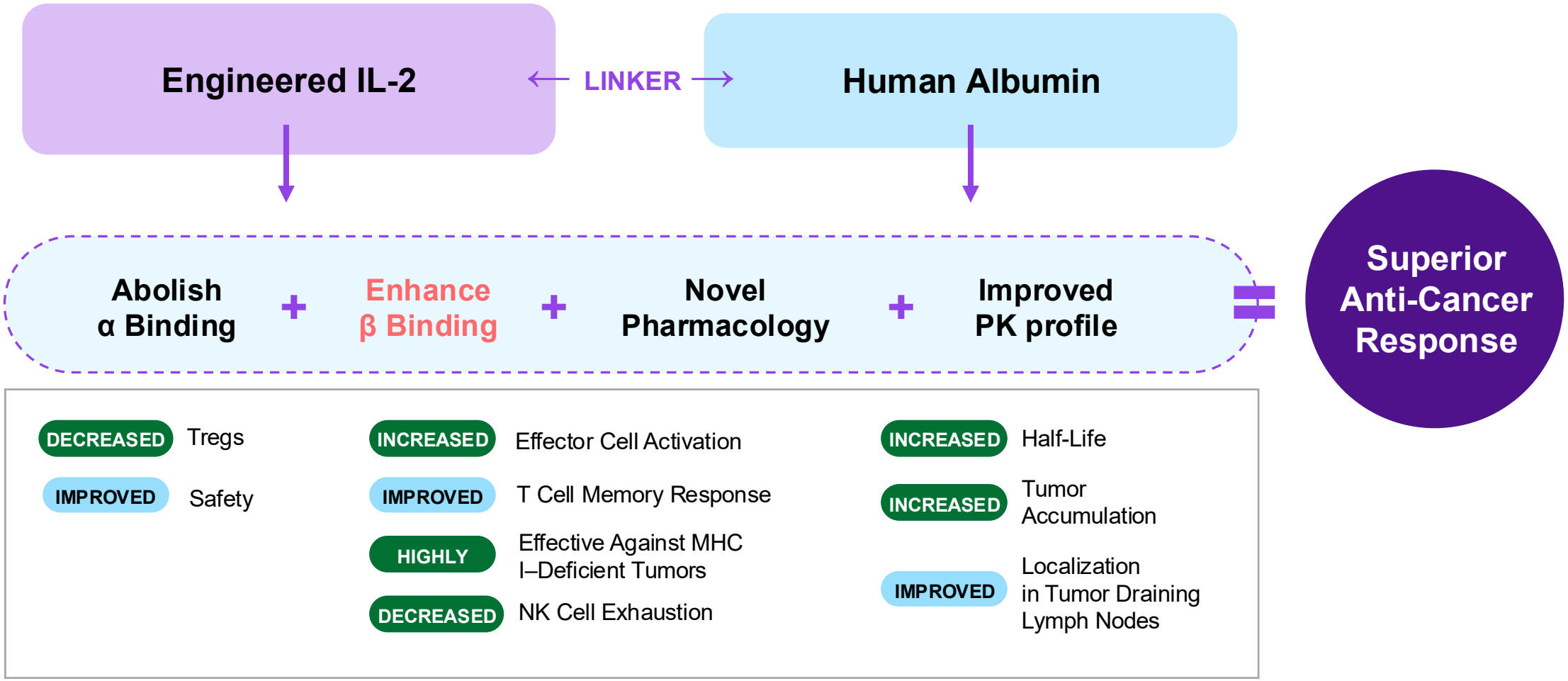
- **Deep and durable single agent activity in various checkpoint resistant cancers**
- **Robust anti-tumor activity demonstrated in earlier 2L/3L treatment setting and as the next treatment line following checkpoint resistance**

NEO-CYT Study Announced H1 2026

ABILITY-1 Study Data Updates H2 2026
Competing Enrollment in Earlier Line Patients

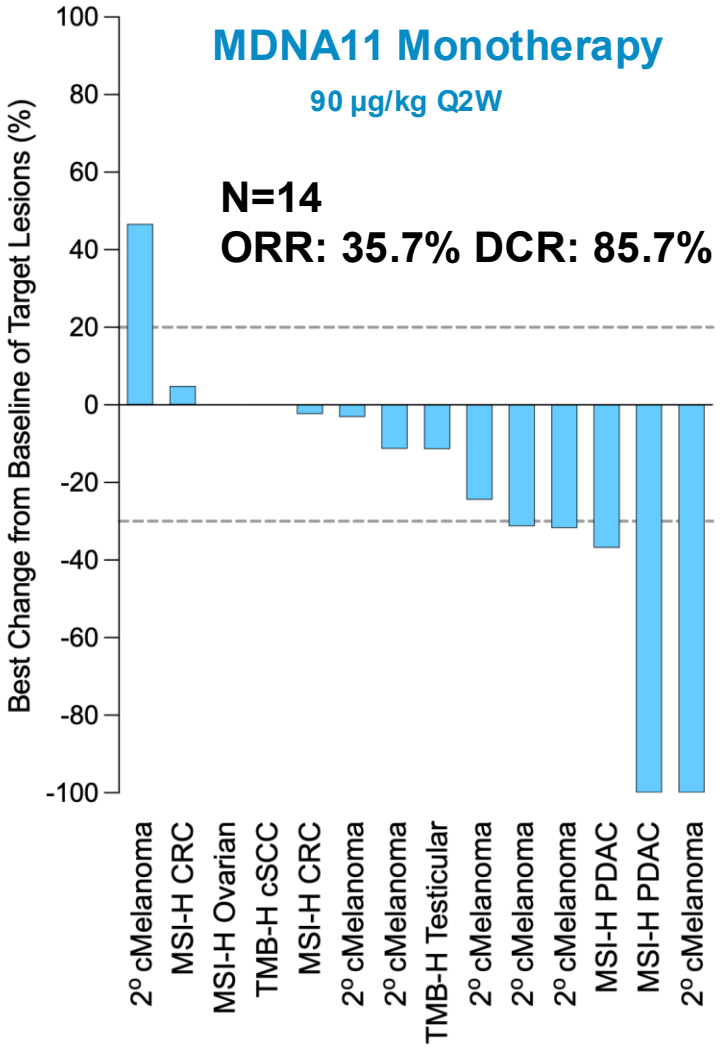
Monotherapy: Melanoma, MSI-H, TMB-H
Combination: Melanoma, MSI-H, TMB-H, Endometrial, NSCLC

MDNA11: A Unique ‘β-enhanced Not-α’ IL-2 Albumin-fused Superkine



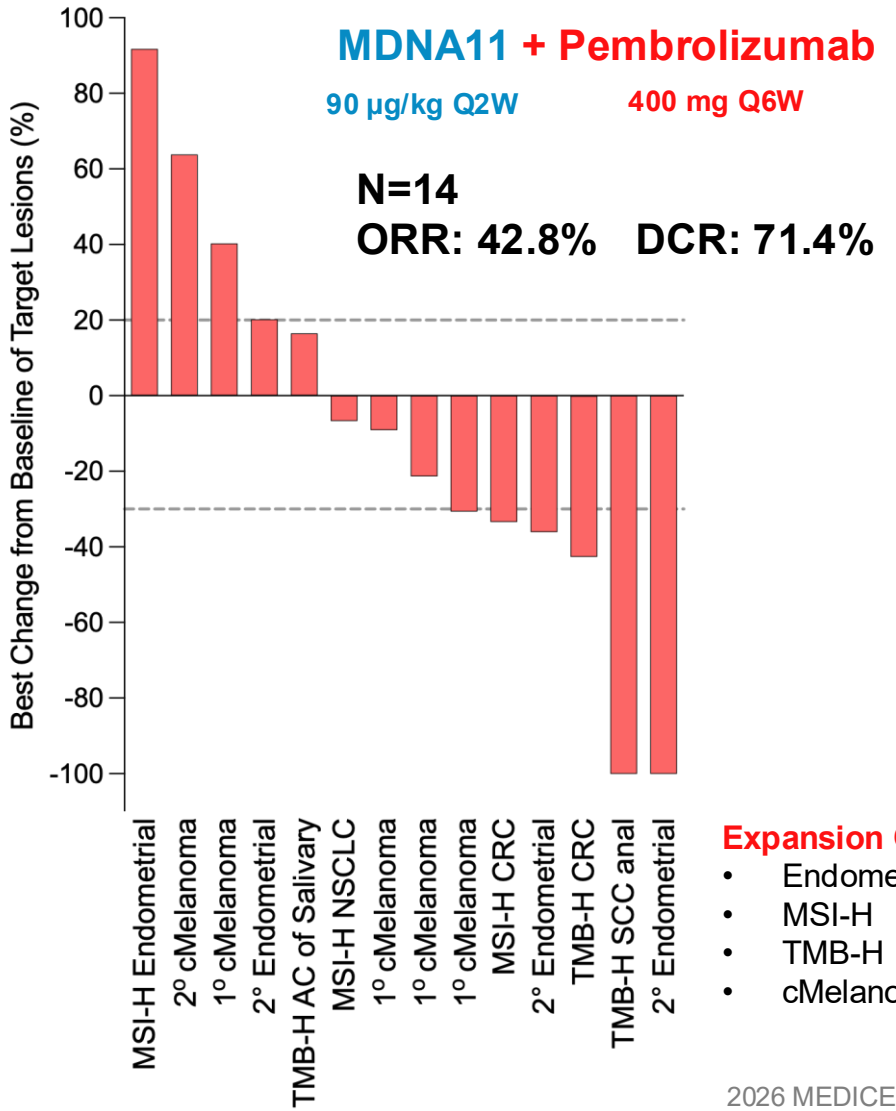
Compelling Anti-Tumor Activity in Earlier Line Therapy Settings

Dose Expansion Cohorts: Last Line ICI OR 1-2 Prior Systemic Treatments



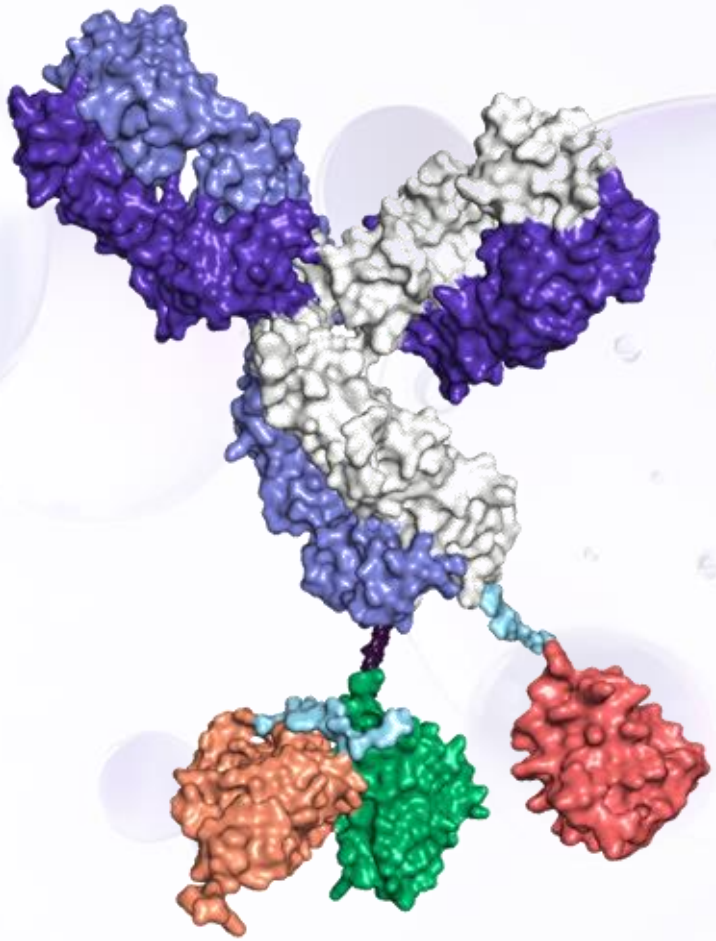
Expansion Cohorts:

- cMelanoma (2° ICI resistance)
- MSI-H
- TMB-H



Expansion Cohorts:

- Endometrial (2° resistance)
- MSI-H
- TMB-H
- cMelanoma



MDNA113

- First-in-class
- Differentiated **PD-1** x **IL-2** Bi-functional Molecule
- Supercharging a **Commercially Validated Anti-PD-1** Inhibitor with a **Clinically Validated IL-2 Superkine**
- Tumor Targeted and Conditionally Activated

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Anti-PD-1 Bispecifics Have Emerged to Compete with PD-(L)1 Therapies

Summit therapeutics | Akesobio

Ivonescimab

MERCK | 礼新医药 LaNova Medicines

LM-299

BIONTECH | Bristol Myers Squibb™

BNT327

Pfizer | 三生制药 3SBIO INC.

SSGJ-707

abbvie | RemeGen Biosciences

RC148

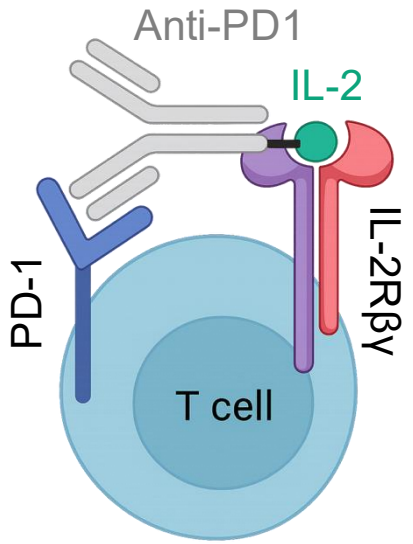
Takeda | Innovent

TAK-928

~$38B USD in PD-(L)1 Bispecific Deal Value Over the Past 24 months

- PD-(L)1 x VEGF combinations dominate early wave approaches
- PD-1 x IL-2 emerging as next frontier
- Medicenna is positioned at this inflection point with its pipeline of superkines

PD-1 x IL-2: The Next Wave of Immuno-Oncology



PD-1 × IL-2 *cis*-binding synergies

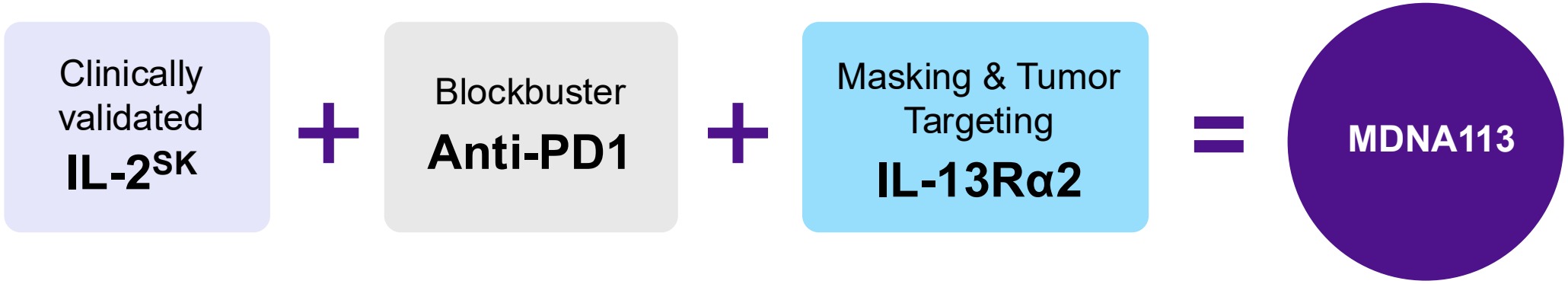
- Targeting the same T cell simultaneously
- **PD-1:** prevents T cell exhaustion
- **IL-2:** proliferates and activates T cells

Designing the Optimal PD-1 x IL-2 Bifunctional

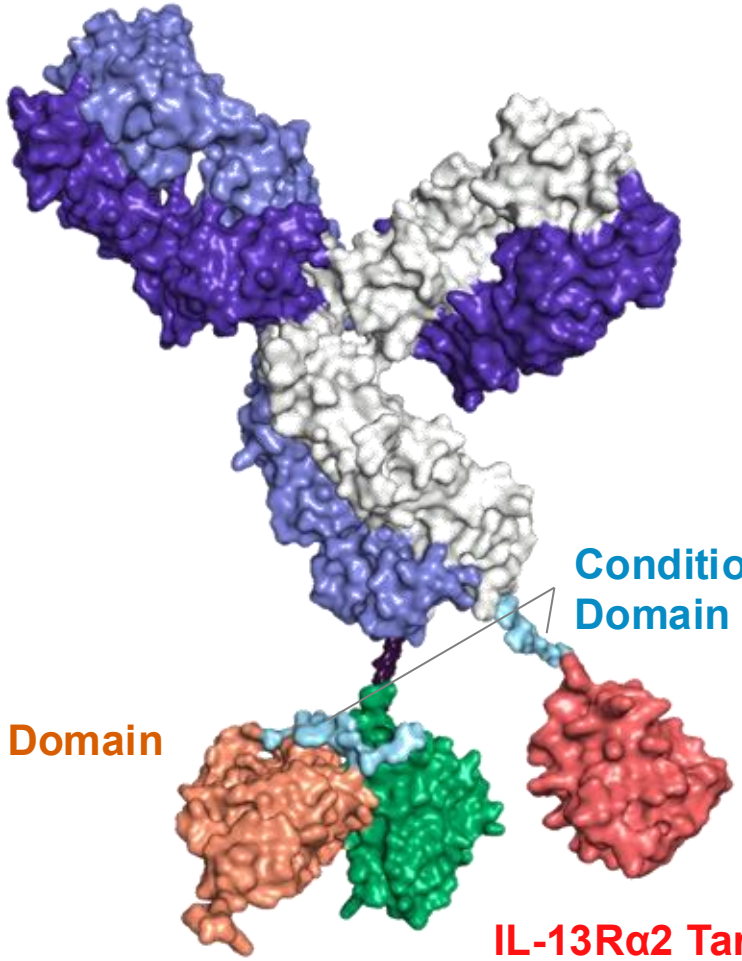
- **Anti-PD-1 inhibitors:** typically dosed 3 mg/kg Q2W for near complete receptor occupancy
- **1:1 fusion of PD-1 x IL-2:** to ensure PD-1 saturation, dosing of 3 mg/kg IL-2 is required
- **IL-2 is more potent** and typically dosed at much lower levels (e.g. 0.09 mg/kg Q2W MDNA11)

Objective: Deliver a Validated Anti-PD-1 Inhibitor & Unleash Full IL-2 Potency at the Tumor

MDNA113: A Differentiated First-in-Class, Tumor Anchored Anti-PD1 x IL-2 Bi-functional Molecule



MDNA113: Designed to be the Optimal PD-1 x IL-2 Bifunctional



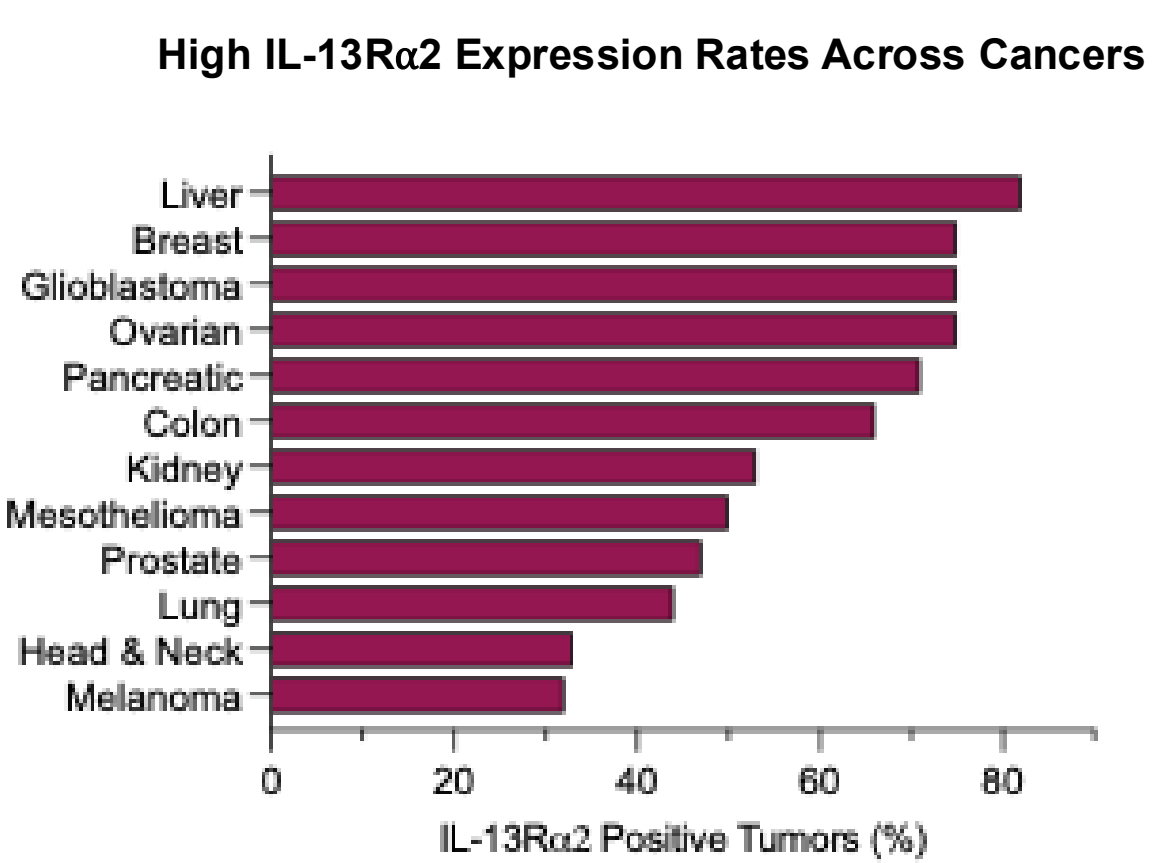
IL-2^{SK} Derived from MDNA11: demonstrating single agent activity in patients with checkpoint resistance

MDNA113: First-in-class

1. **Delivering a commercially validated blockbuster anti-PD-1 inhibitor**
2. **Supercharging PD-1 with a clinically validated IL-2 superkine**
3. **Masking IL-2 systemically such that 3 mg/kg Q2W can be administered to saturate PD-1**
4. **IL-13Rα2 Tumor Targeting: anchoring MDNA113 at the tumor**
5. **Ensuring activation at the TME by two unmasking mechanisms**

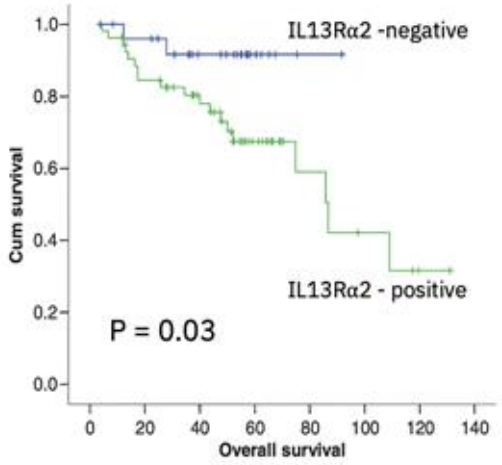
IL-13Rα2 is Expressed in Many Tumors Affecting 2M+ Patients Annually

Blockbuster potential: High IL-13Rα2 expression is associated with poor clinical outcomes, making it an ideal TAA target



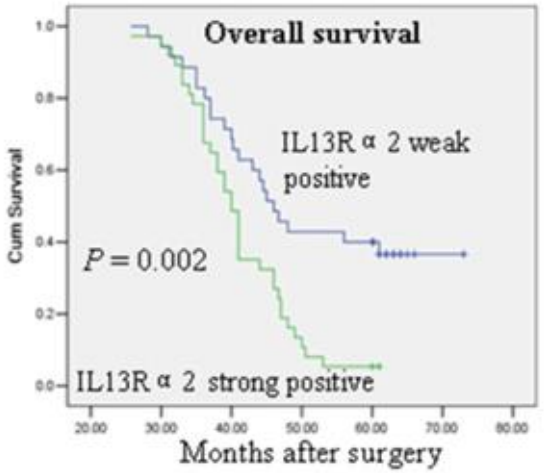
COLON CANCER

Barderas et al., Cancer Res, 2012



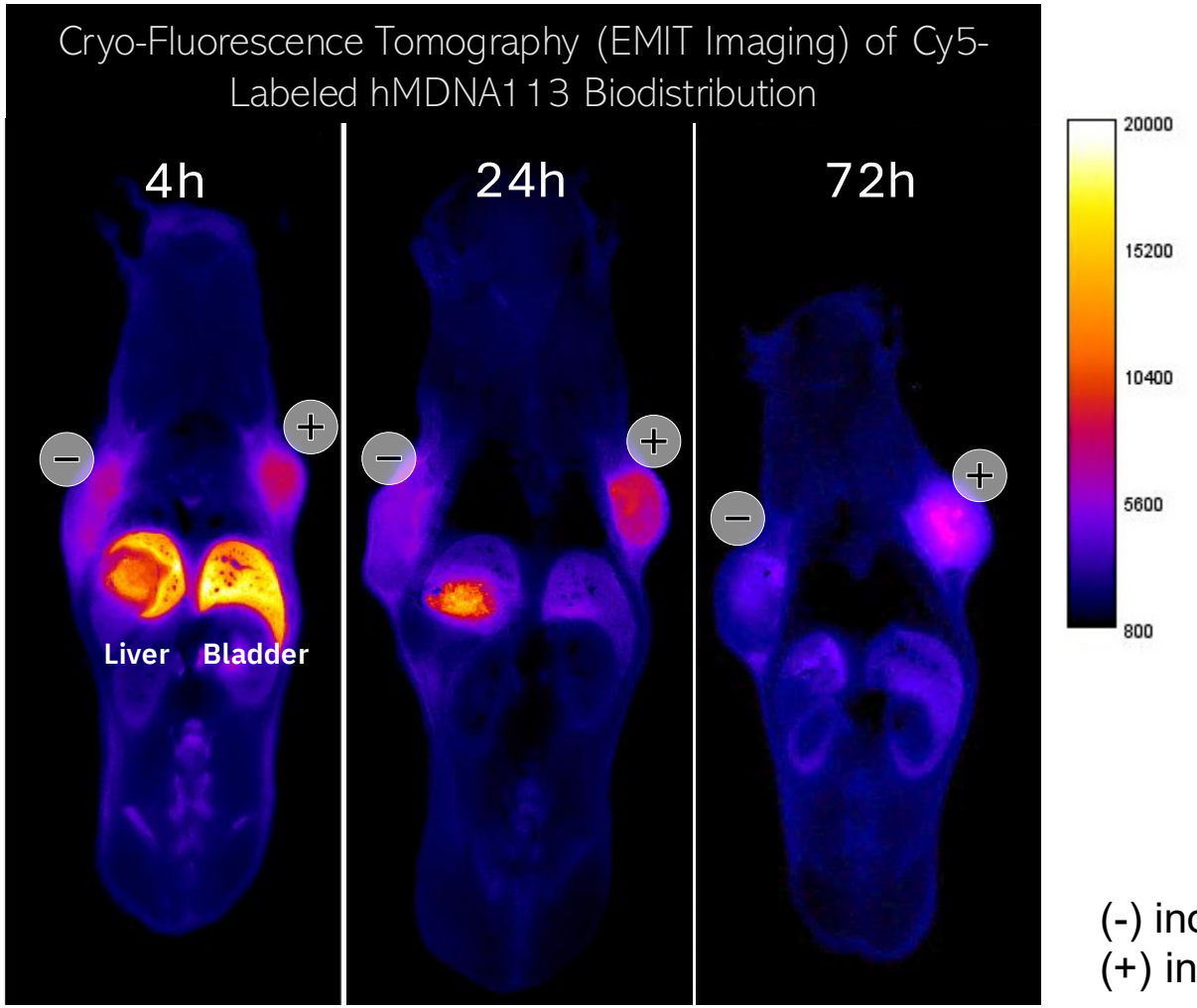
LUNG CANCER

Xie et. al. Oncotarget, 2015



1. Hou et al., J Cancer Res & Clinical oncol (2009); 2. Papageorgis et al., Br Cancer Res (2015); 3. Joshi et al., Cancer Res (2000); 4. Kioi et al., Cancer (2006); 5. Shimamura et al., Clin Cancer Res (2010); 6. Barderas et al., Cancer Res (2012); 7. Kang et al., J Per Med (2021); 8. Oncomine Cancer MicroArray (OMCA Database); 9. Nagai et al., Cancer Reprots (2023); 10. Xiw et al., Oncotarget (2015); 11. Kawakami et al., Clin Cancer Res (2003); 12. Beardi et al., Clin Cancer Res (2013)

Tumor-Anchoring: Preferential Tumor Localization and Retention of MDNA113 in IL-13Rα2 Expressing Tumors

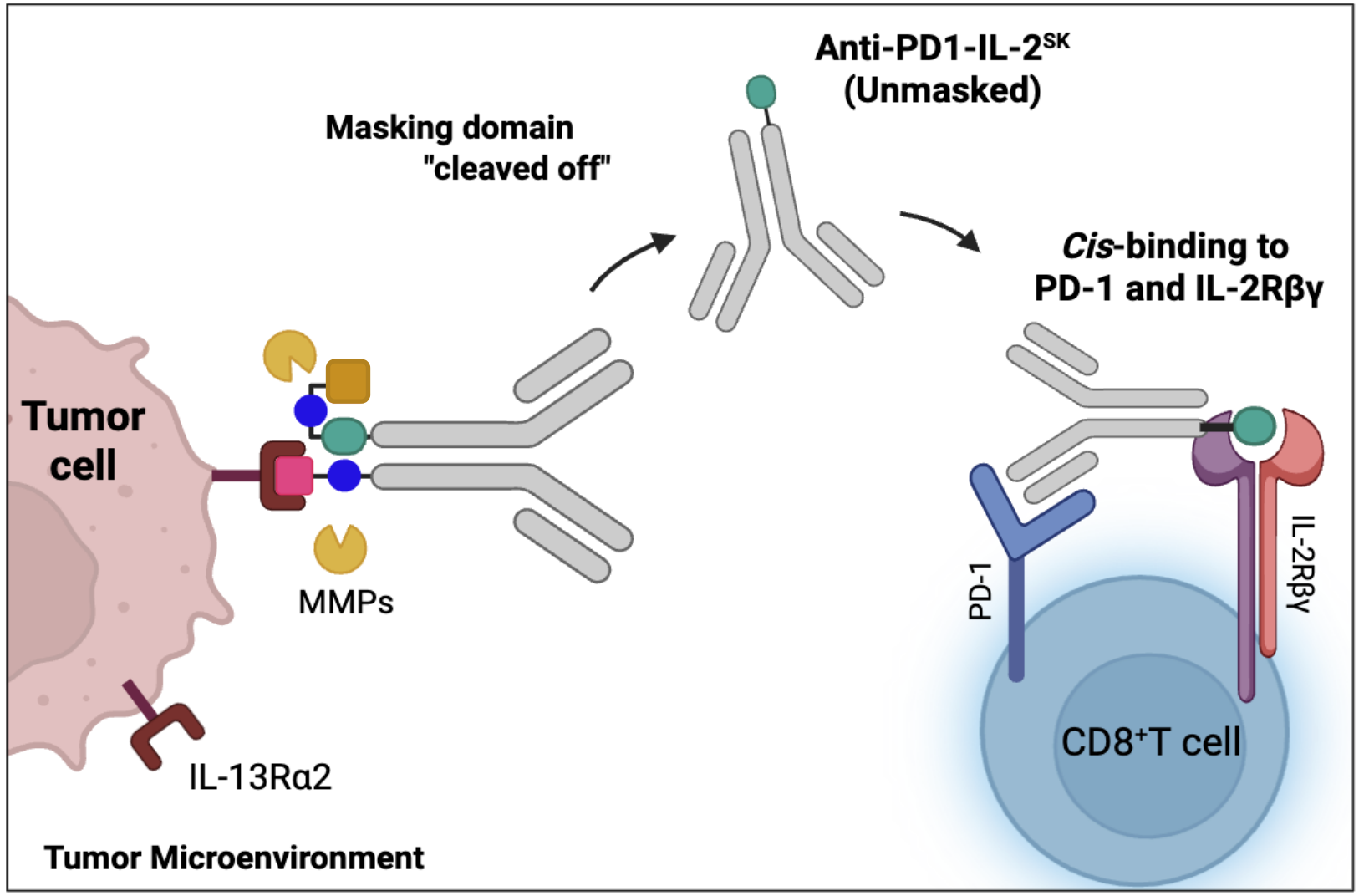


Delivering PD-1 and Unleashing IL-2^{SK}

- MDNA113 is localized to **IL-13Rα2$^{(+)}$ tumors** and anchored for conditional activation

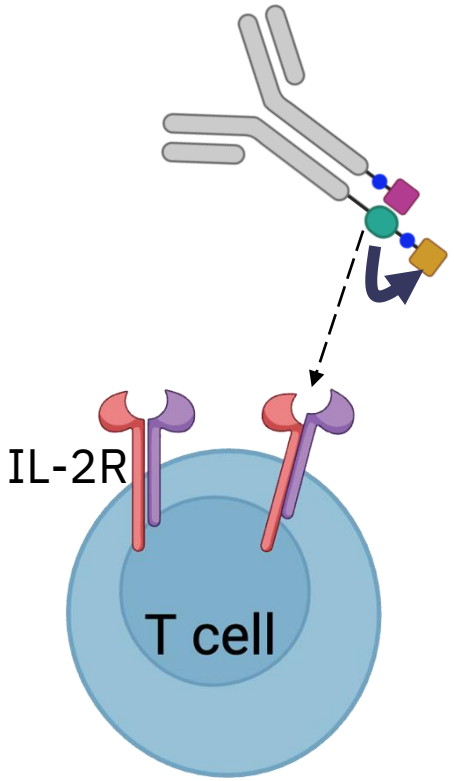
(-) indicates IL-13Rα2-negative
(+) indicates IL-13Rα2-positive tumors

Mechanism 1: MDNA113 is Anchored to IL-13Rα2 and is then Activated in the TME via MMP Cleavage



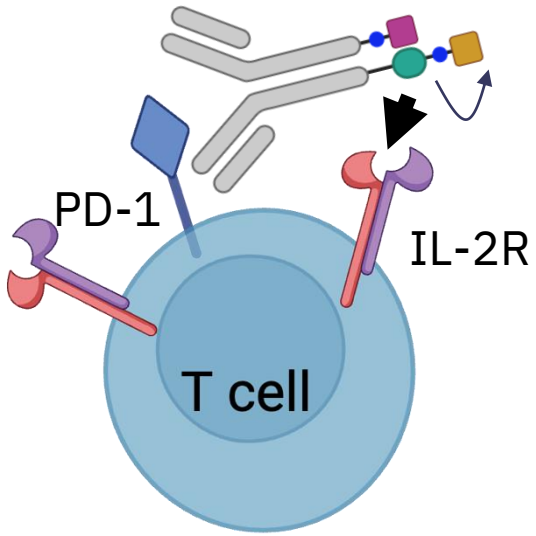
Mechanism 2: cis-Expression of PD-1 and IL-2R on Target Cells Unmasks MDNA113 Without Requirement for MMP Cleavage

Periphery: No PD-1 binding, IL-2 masking intact



PD1^{Neg} CD8^{+} T Cells
(predominantly in circulation)

TME: PD-1 binding enables IL-2 x IL-2R engagement without need for cleavage



PD1^{Pos} CD8^{+} T Cells
(predominantly within TME)

MDNA113 Builds Upon Established Proof of Concept in Late-Stage Development

Takeda Innovent

PD-1/IL-2$^{\alpha\text{-bias}}$

- Early signs of efficacy in CPI-naïve patients
- Advancing into Phase 2 and Phase 3 Registrational Trials
- **Grade 5 events consistent with IL-2Rα binding and native IL-2**
- **Due to toxicity, TAK928 is limited on its dosage**

2025 Licensing Transaction including one other asset:
$1.2B upfront, up to $11.4B in milestones

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PD-1/IL-2$^{\text{non-}\alpha,\ \beta\ \text{enhanced}}$

- IL-13Rα2 tumor-anchoring
- Activation of drug at tumor site
- Designed to be safer via with conditional activation (i.e. not capped on dosage)
- Targets cold tumors
- **IL-2 component alone of MDNA113 has similar efficacy to IBI363 in cutaneous melanoma (~30% ORR)**

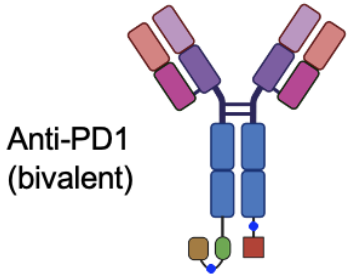
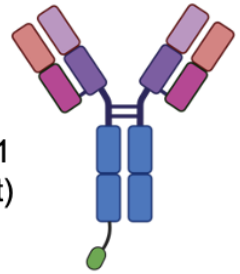
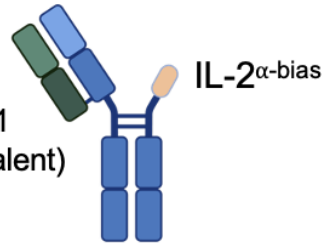
IBI363 Ph1/2 Data Adapted from ASCO and ESMO Plenary 2024

Differentiation of MDNA113 to other Bifunctional Anti-PD-1-IL-2 Programs

KEY FEATURES	Medicenna MDNA113	Other IL2/anti-PD1 candidates
β-enhanced and not-α IL-2^{SK} (clinically validated)	✓	✗
Tumor Specific Targeting (IL-13Rα2)	✓	✗
Dual conditional activation mechanisms	✓	✗
PD-1/PD-L1 Blockade (clinically validated vs. novel)	✓	✓/✗
Cis-binding (IL-2R/PD-1)	✓	✓/✗
IL-2^{SK} attenuated in periphery	✓	✓/✗
IL-2^{SK} activated in TME	✓	✓/✗

NHP: MDNA113 Was Well-tolerated at Doses up to 50 mg/kg

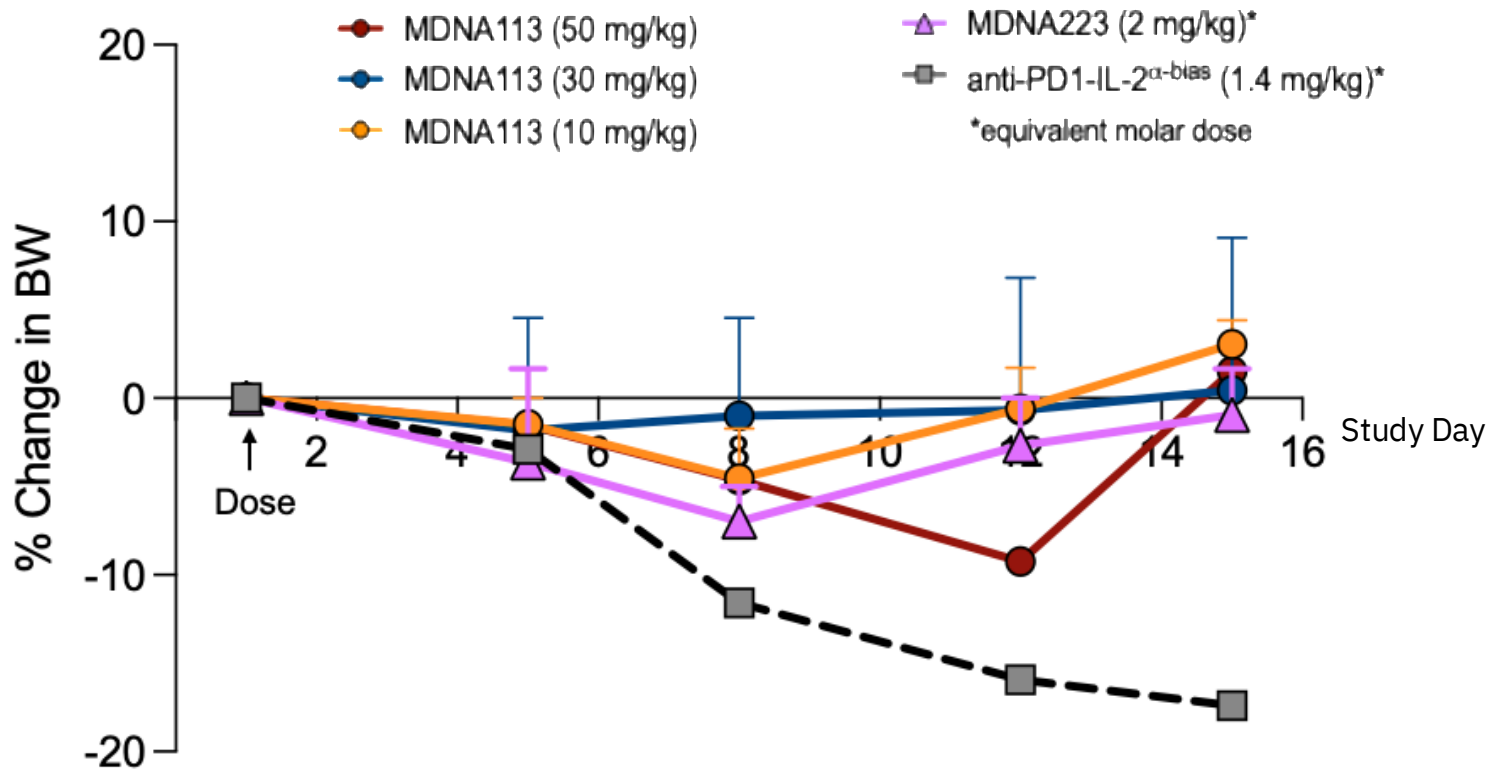
Head-to-Head: Unmasked MDN113 was Better Tolerated vs. Anti-PD-1-IL-2$^{\alpha\text{-bias}}$

Test Construct	Dose	Clinical Observations
MDNA113 (Masked) 	10 mg/kg (n = 2)	1 of 2 monkeys experienced diarrhea (7 days)
	30 mg/kg (n = 2)	No abnormal findings
	50 mg/kg (n = 1)	Monkey experienced diarrhea (7 days)
MDNA223 (Unmasked) 	2 mg/kg (n = 2)	1 of 2 monkeys experienced diarrhea (5 days)
Anti-PD1-IL-2$^{\alpha\text{-bias}}$ 	1.4 mg/kg (n = 1) Equivalent molar dose as MDNA223	Skin erythema in eye and inguinal regions (> 9 days) Decreased appetite (> 9 days) Decreased activity (7 days)

Naive cynomolgus monkeys; IV infusion

NHP: Body Weight in NHP Maintained with High Dose MDNA113

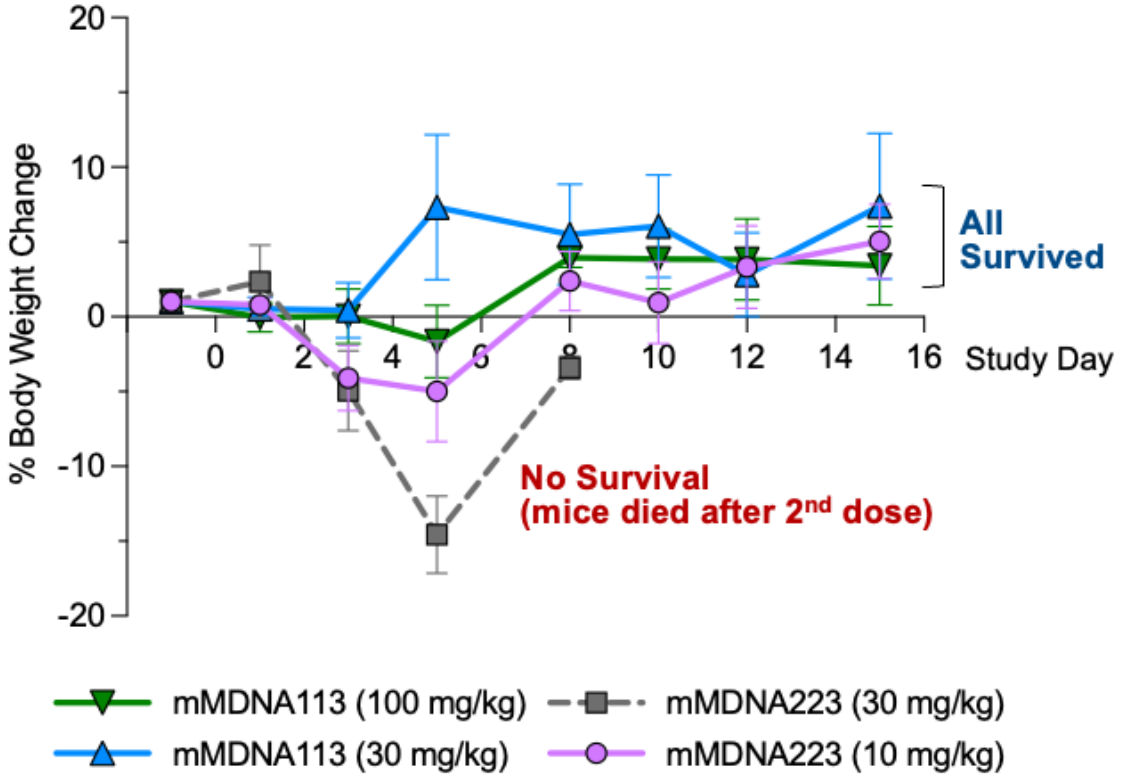
Head-to-Head: Unmasked MDNA113 was Better Tolerated vs. Anti-PD-1-IL-2$^{\alpha\text{-bias}}$



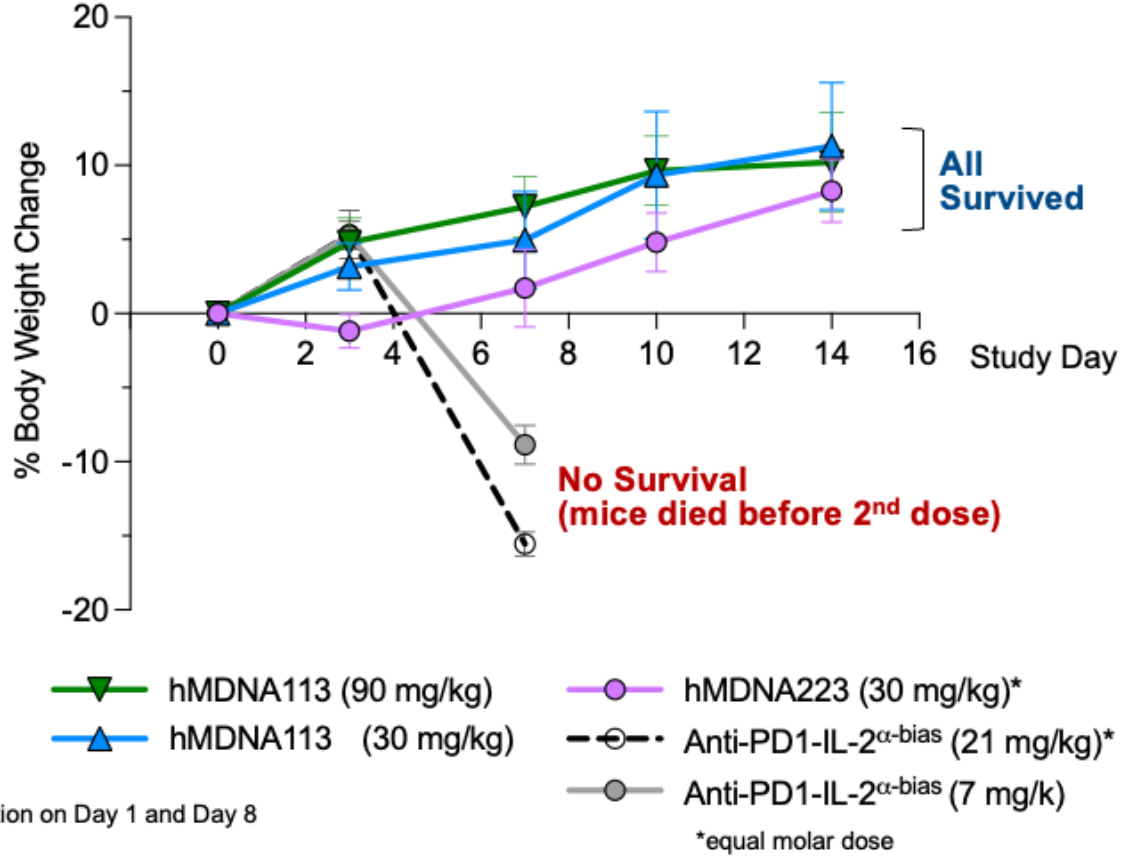
In vivo: MDNA113 was Well Tolerated in Mice up to At least 100 mg/kg

Isolated effect of IL-2R demonstrated poor tolerability of Anti-PD-1-IL-2$^{\alpha\text{-bias}}$ vs. Unmasked MDNA113

mMDNA113 (with anti-mPD1):
(effect of PD1/PDL-1 and IL-2R)

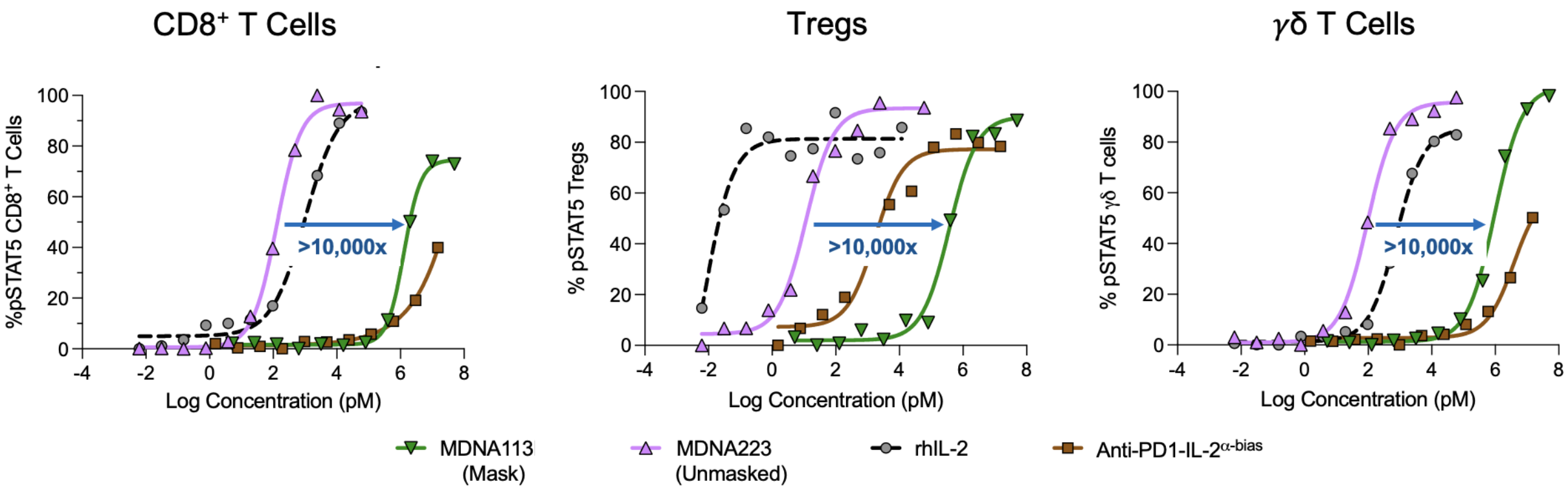


MDNA113 (with anti-hPD1):
(effect of IL-2R only)



Female C57Bl/6 mice treated by IV injection on Day 1 and Day 8

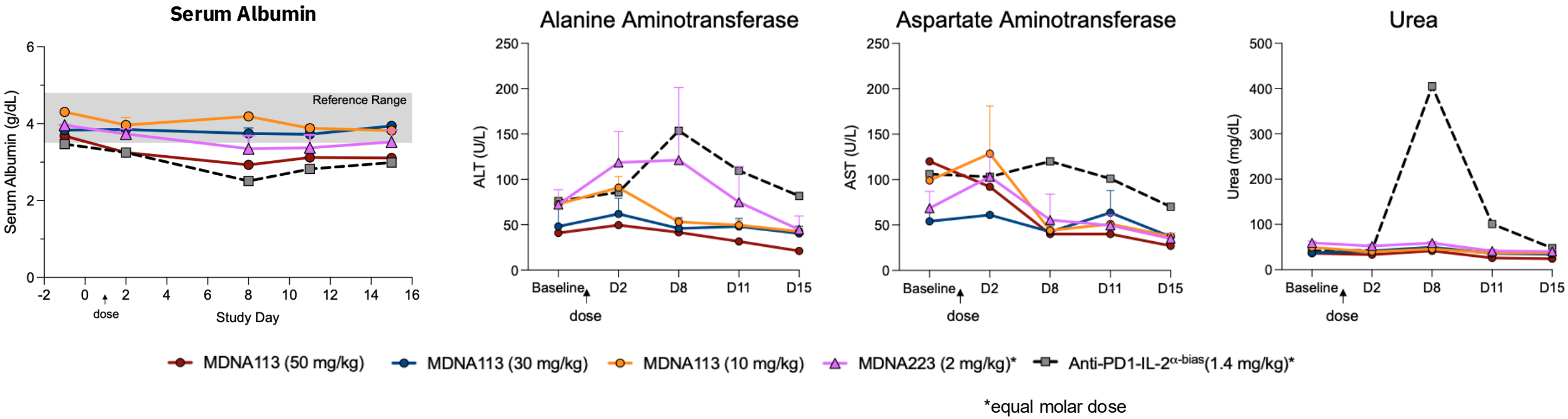
PBMC: 10,000-Fold Blockade of pSTAT5 Signaling in Human PBMCs Demonstrates Superior Masking Capacity



Data from representative normal human PBMC donor. Analysis by flow cytometry; normalized pSTAT5 signal shown

NHP: MDNA113 has Minimal Effect on Serum Albumin, Liver Enzymes and Urea

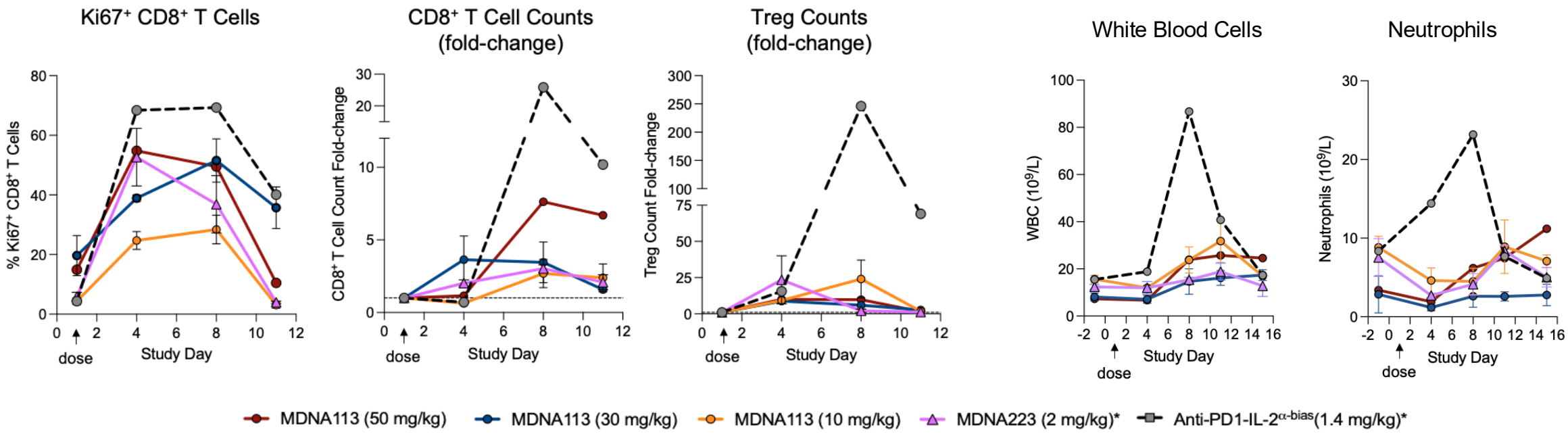
Head-to-Head: Anti-PD-1-IL-2$^{\alpha\text{-bias}}$ Demonstrates Impact on Renal Function



*equal molar dose

NHP Masking: Dampened Systemic Immune Activation in NHP with MDNA113

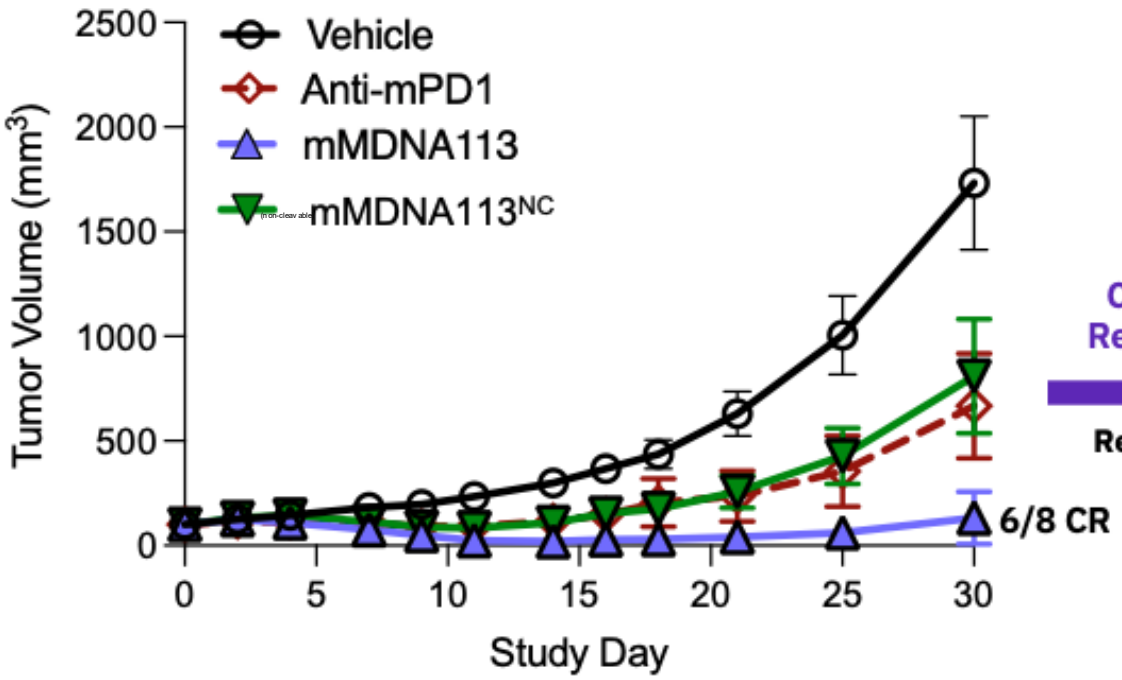
Head-to-Head: Anti-PD-1-IL-2α-bias Demonstrated Peripheral Activation of Tregs



*equal molar dose

In vivo: MDNA113 Inhibits IL-13Rα2-Expressing MC38 Tumors and Promotes Memory Response Against Tumor Rechallenge

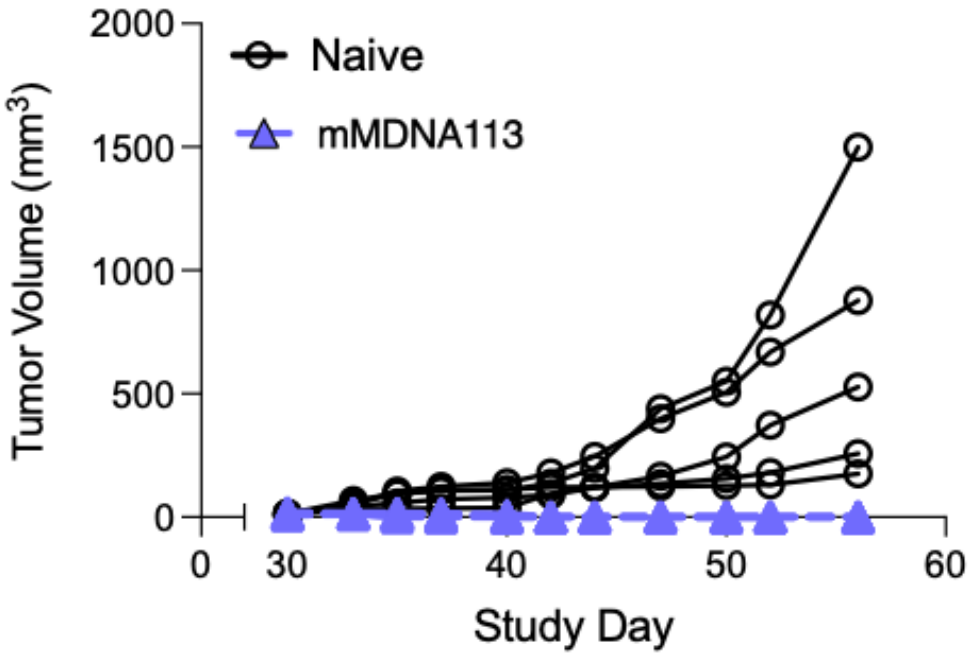
MC38-IL-13Rα2 Colon Tumor Model



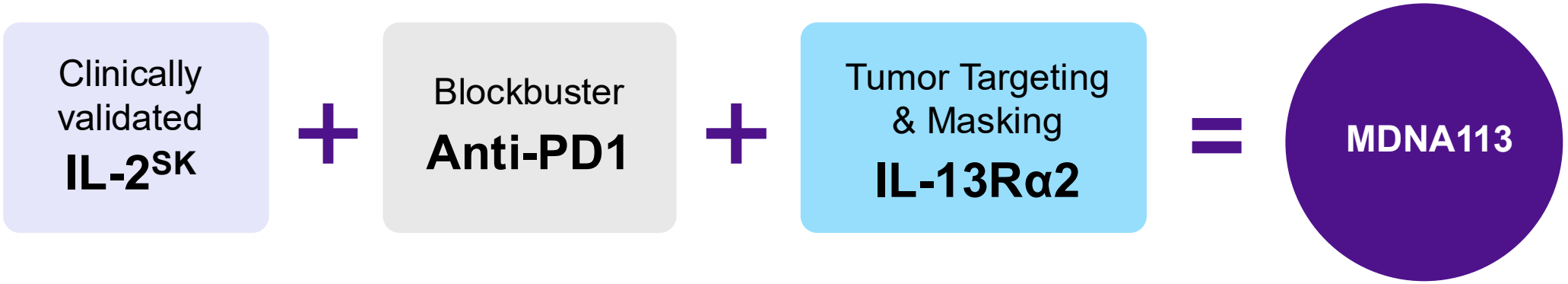
Complete Responders

Re-challenge

Memory Response



MDNA113: A Differentiated First-in-Class, Tumor Anchored Anti-PD1 x IL-2 Bi-functional Molecule



- **Differentiated** masking approach allows for saturation of PD-1 whilst unleashing IL-2^{SK} within the TME
- **Tumor targeting** to IL-13Rα2, **anchors** the PD-1 x IL-2 to the TME for a sustained period of time
- **MOA 1 for Activation:** activation by tumor specific proteases MMPs
- **MOA 2 for Activation:** *cis*-expression of PD-1 and IL-2R on target effector cells unmasks MDNA113

MDNA113 demonstrates:

- Exceptional tumor selectivity, localization, and potency with improved systemic tolerability
- Superior tolerability and significantly wider therapeutic window vs. PD-1 x IL-$2^{\alpha\text{-bias}}$ comparator
- Well tolerated up to 50 mg/kg in non-human primates

Thank you

TSX: MDNA
OTCQX: MDNAF

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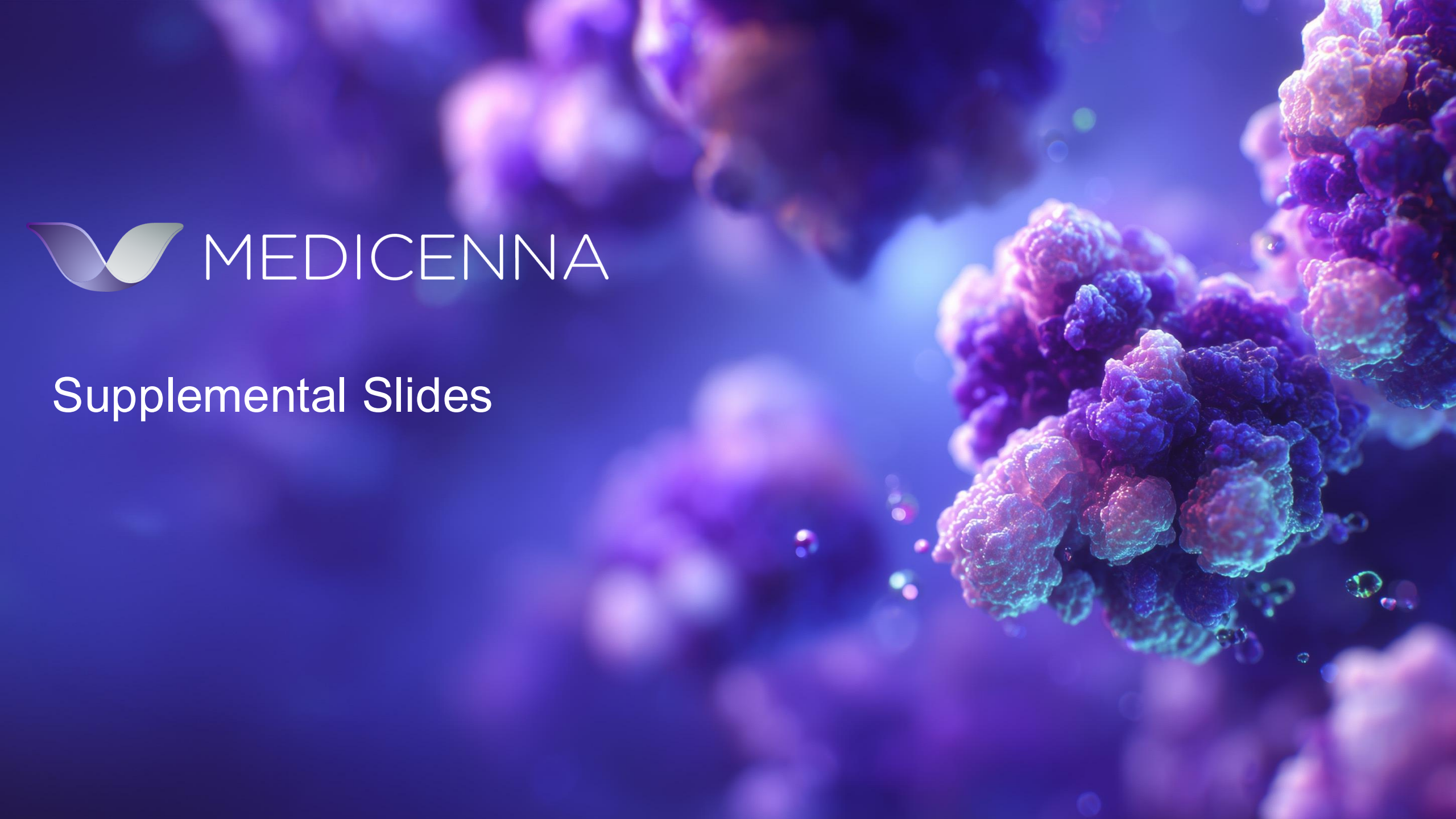
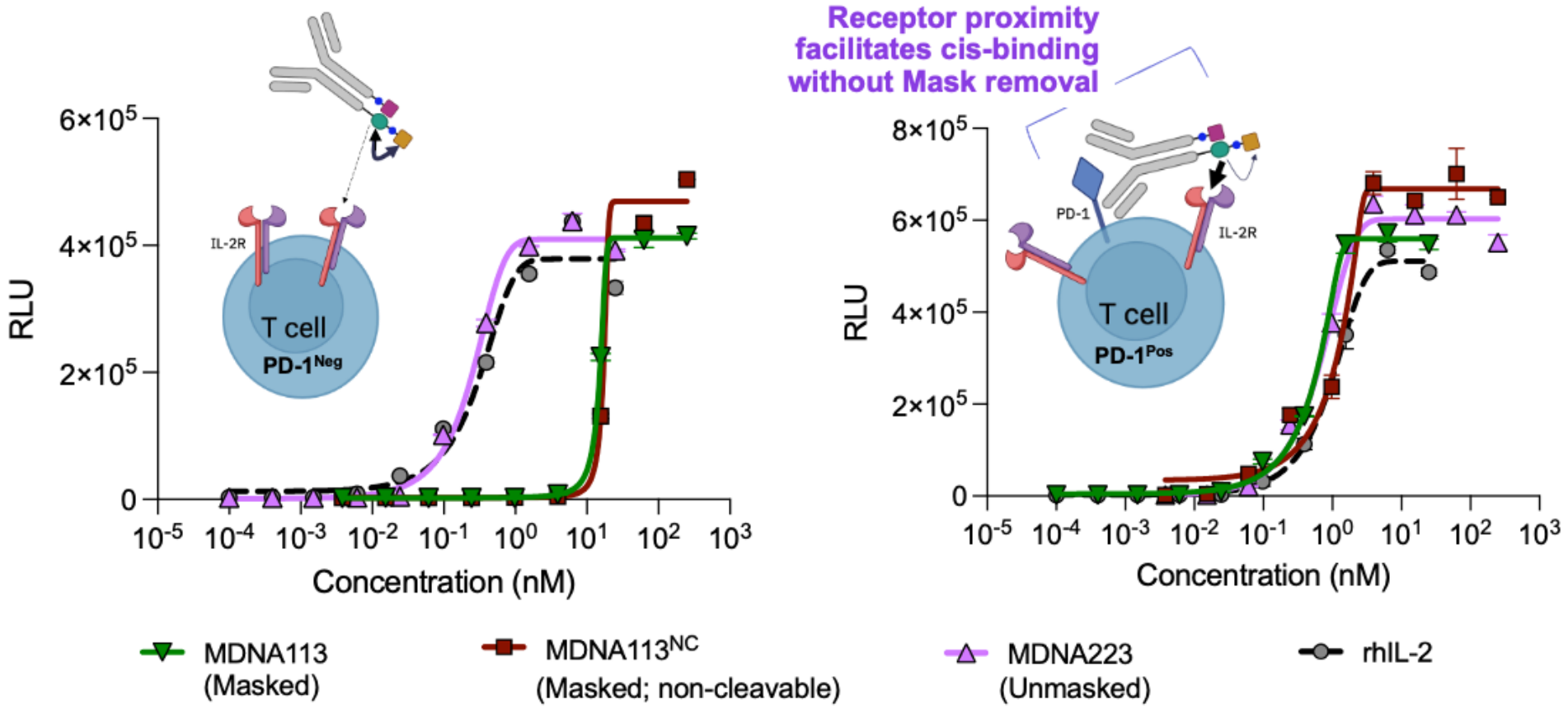


Supplemental Slides

Mechanism 2: cis-Expression of PD-1 and IL-2R on Target Cells Unmasks MDNA113 Without Requirement for MMP Cleavage

Periphery: No PD-1 binding, IL-2 masking intact

TME: PD-1 binding enables IL-2 x IL-2R engagement without need for cleavage

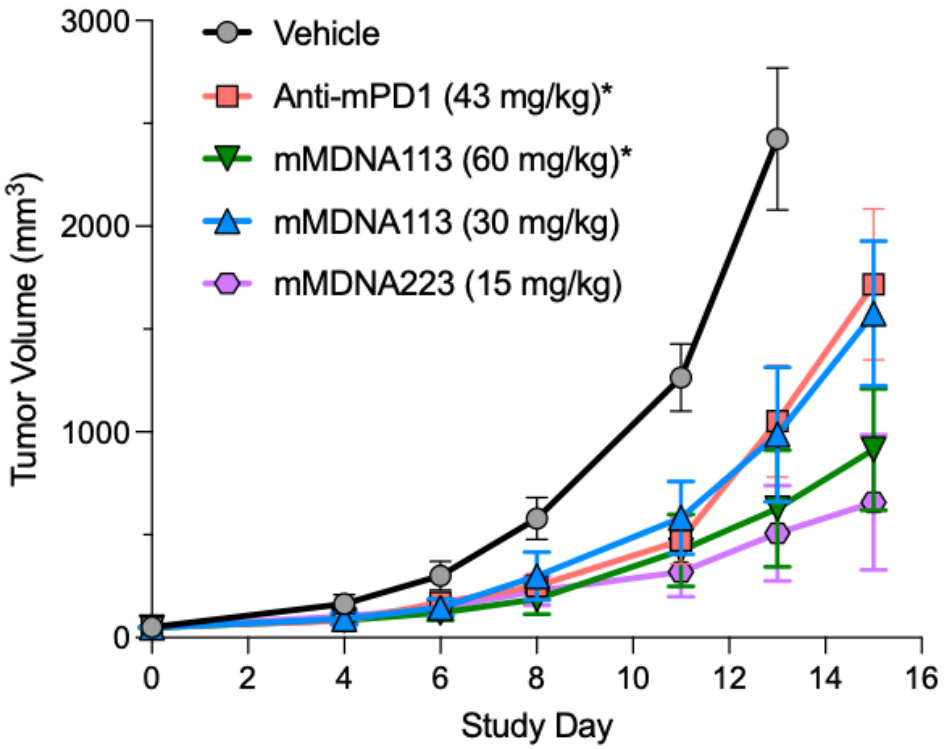


293T-STAT5-Luc2-IL2Rαβγ (left) and 293T-STAT5-Luc2-IL2Rαβγ-PD1 (right) reporter assays. Incubation at 37 °C for ~22 hours and measured relative luminescence unit (RLU)

In vivo: MDNA113 Enhances Infiltration of Functionally Active CD8⁺ T Cells in the B16F10/IL-13Rα2 Melanoma Model

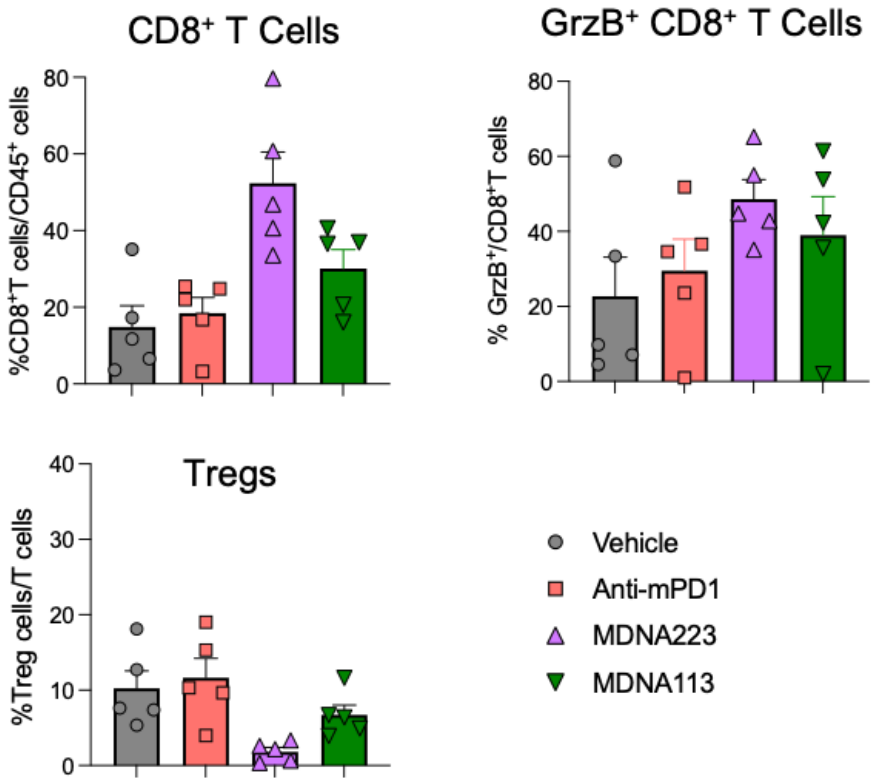
No Increase in Tregs

B16F10 Melanoma Model



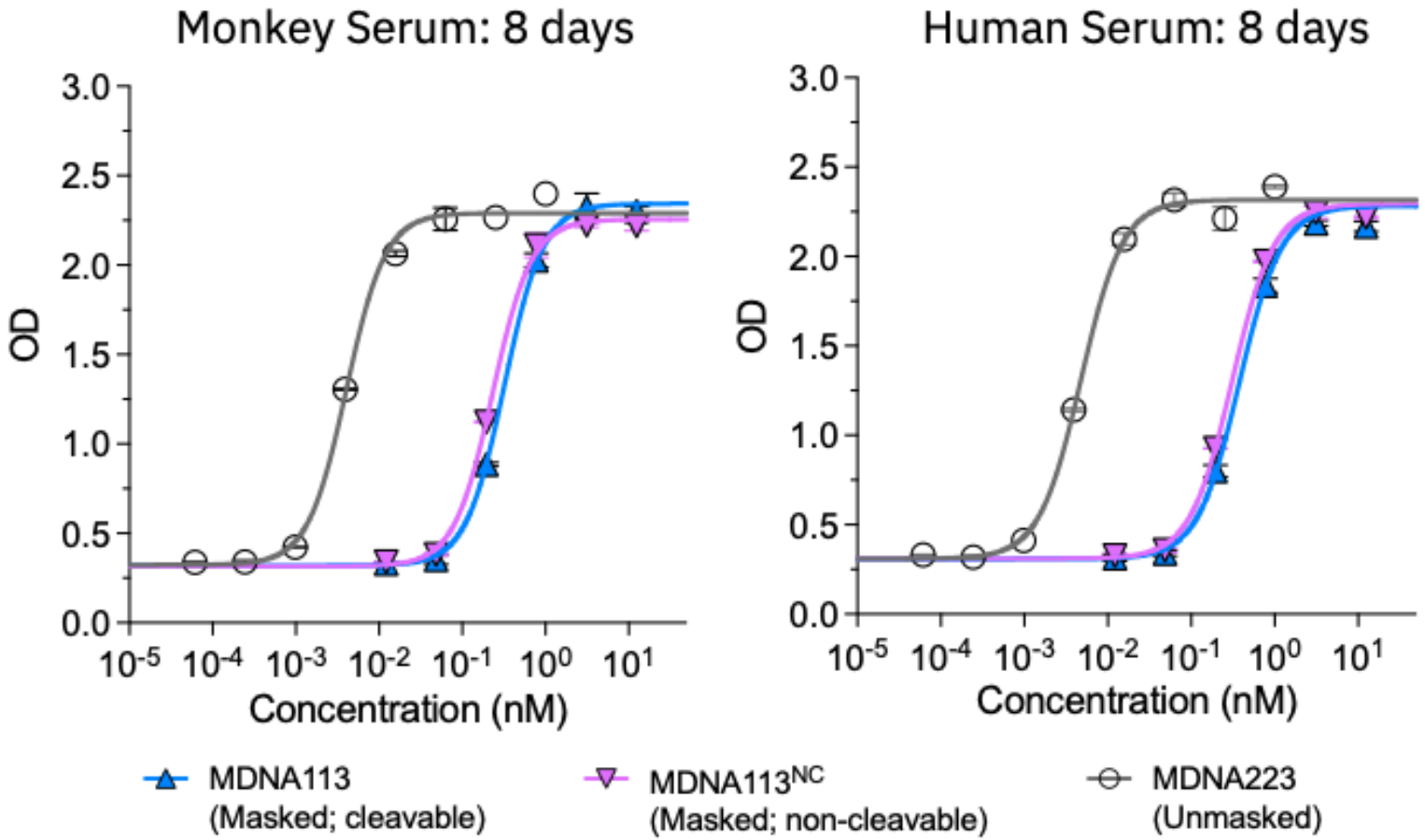
B16F10 tumor size of 70 mm³ at start of treatment; IV (Day 1 & 8)
*Equal molar dose of test articles used

Tumor Infiltrating Immune Cells



Mice with B16F10/IL-13Rα2 tumors (150 mm³) received 1 dose by IP injection.
Molar-equivalent dose of 10 mg/kg. Tumors harvested on Day 8 for flow cytometry

Masking: No Linker Cleavage Following Prolonged Incubation in Normal Sera



Constructs incubated in pooled normal serum at **37 ºC for 8 days** and evaluated for JAK-STAT activation in HEK-Blue IL-2Rαβγ reporter cells.

MDNA113 Pharmacokinetics Consistent with Approved Anti-PD-1 Therapies