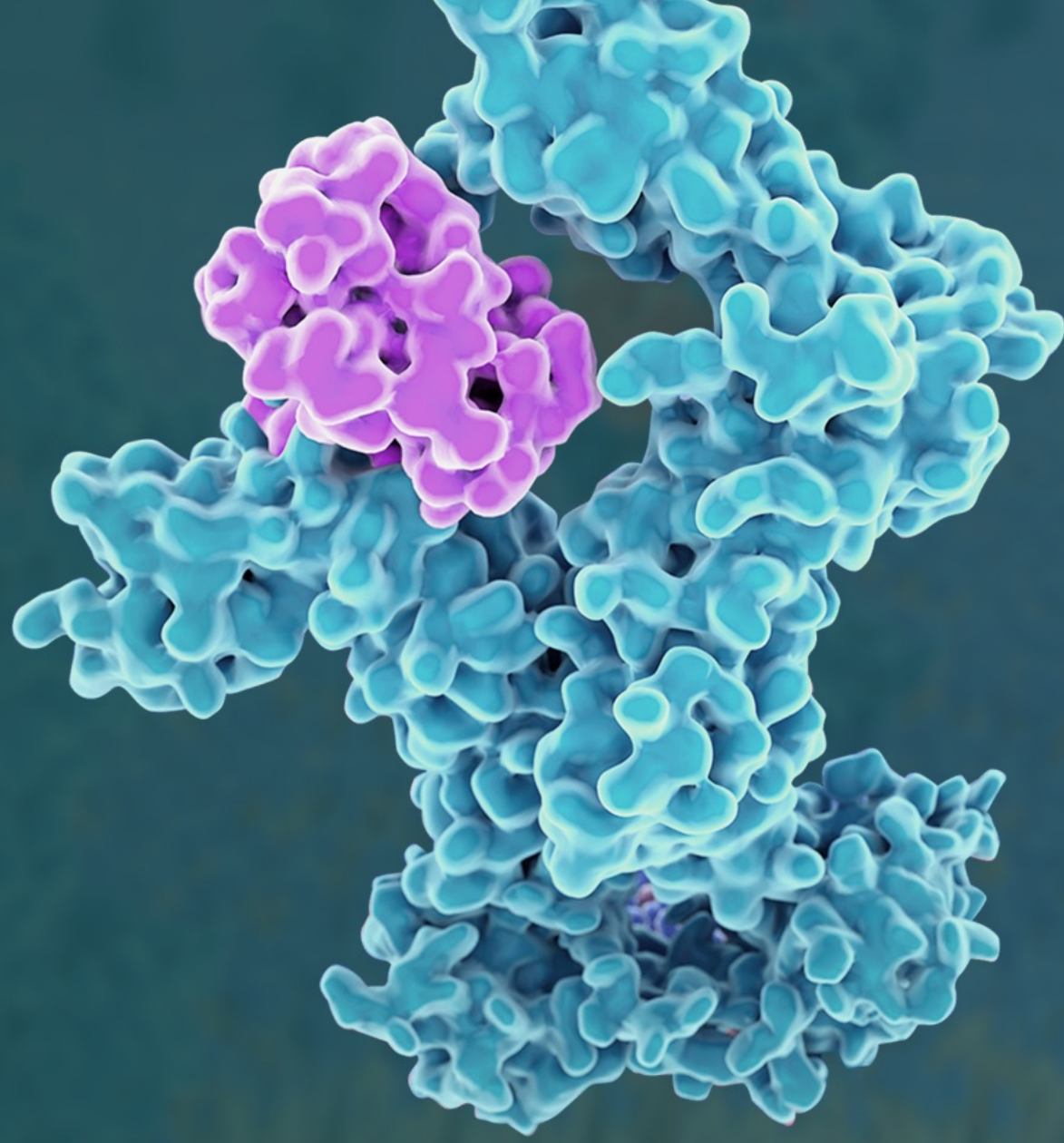
Q4, 2022

Evolutionary Cytokines Revolutionary Medicines



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Investment Highlights

Regular Clinical Data Updates from MDNA11 Program Expected

Superkine Platform: Drug Discovery Engine

- Directed evolution **enhances the desired properties** of IL-2, IL-4, & IL-13 to generate Superkines
- Protein fusion can **improve PK, add an MOA, or confer new capabilities** to Superkines
- IL-2, IL-4 , & IL-13 are known to modulate immune activity against **2,000 different diseases**

MDNA11: "Beta-only" & Long-acting IL-2 Super-agonist in Phase 1/2

- Super-agonist against IL-2R, a **clinically validated anti-cancer target**
- Enhanced IL-2Rβ binding and lack of IL-2Rα affinity position MDNA11 to be **best-in-class**
- Clinical data updates expected **at regular intervals**

MDNA55: Phase 3 Ready Empowered IL-4 Superkine

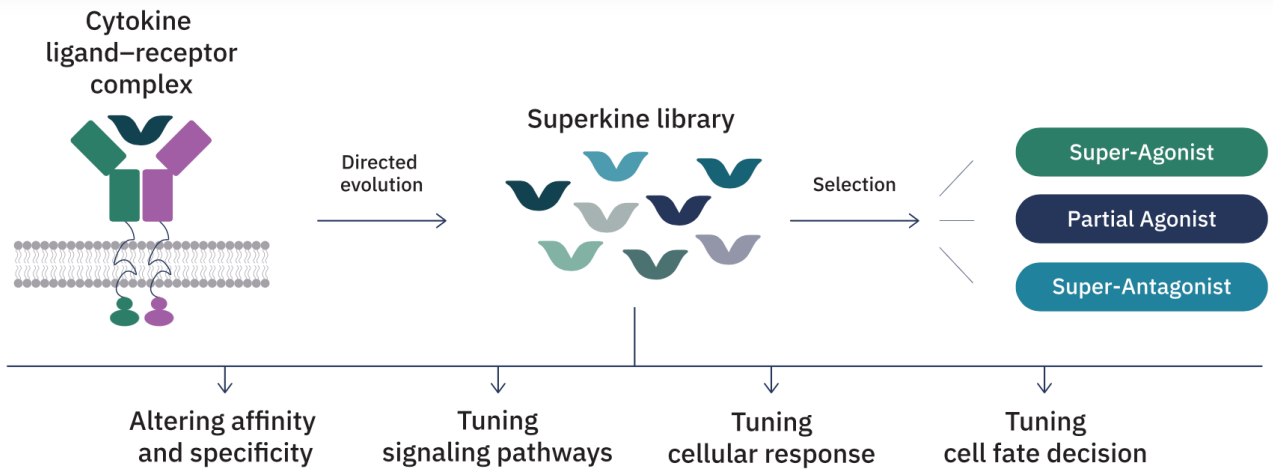
- Targeting recurrent glioblastoma, the most aggressive form of brain cancer
- Phase 2b data show **~100% improvement in median OS** vs. a matched external control arm
- Pursing a **partnership** to advance development

BiSKIT Platform: **Bi**functional **S**uper**K**ines for **I**mmuno**T**herapy

- Fusion of two Superkines or a Superkine and an antibody (e.g. a checkpoint inhibitor)
- Incorporate **two synergistic MOAs** into a single molecule

Superkine Platform Powers Drug Discovery Engine

Transforming IL-2, IL-4 and IL-13 into Druggable Superkines Using Directed Evolution



Our IL-2, IL-4 and IL-13 Superkines are known to modulate immune activity in many diseases, each providing "A Pipeline in a Product" opportunity

Superkine Design and Development

Generate Tunable Superkine Library

Transform interleukins using directed evolution to enhance desired properties

Enhance via Protein Fusion

To improve PK, add a second MOA, or confer new capabilities

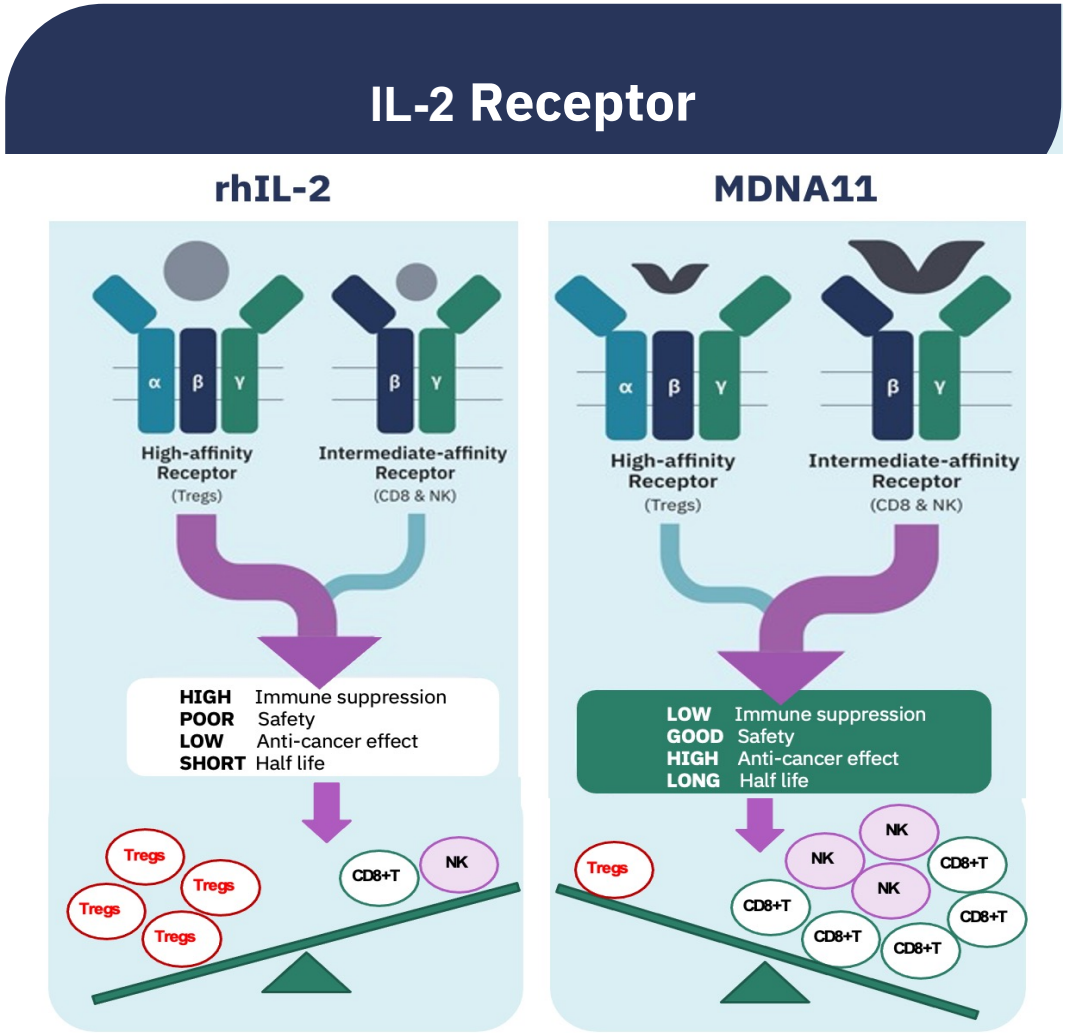
Lead Selection & Development

Advance the most promising candidates towards clinical studies

MDNA11

“Beta-only” & Long-acting IL-2 Super-Agonist for Solid Tumors

Targeting IL-2 Receptor Subunits in Cancer Therapy



The IL-2 receptor (IL-2R) consists of three subunits

- IL-2Rα (CD25)
- IL-2Rβ (CD122)
- IL-2Rγ (CD132)

Stimulation of IL-2Rβ

- Key for the activation of cancer killing immune cells such as CD8+ T cells, naïve T cells, and NK cells.

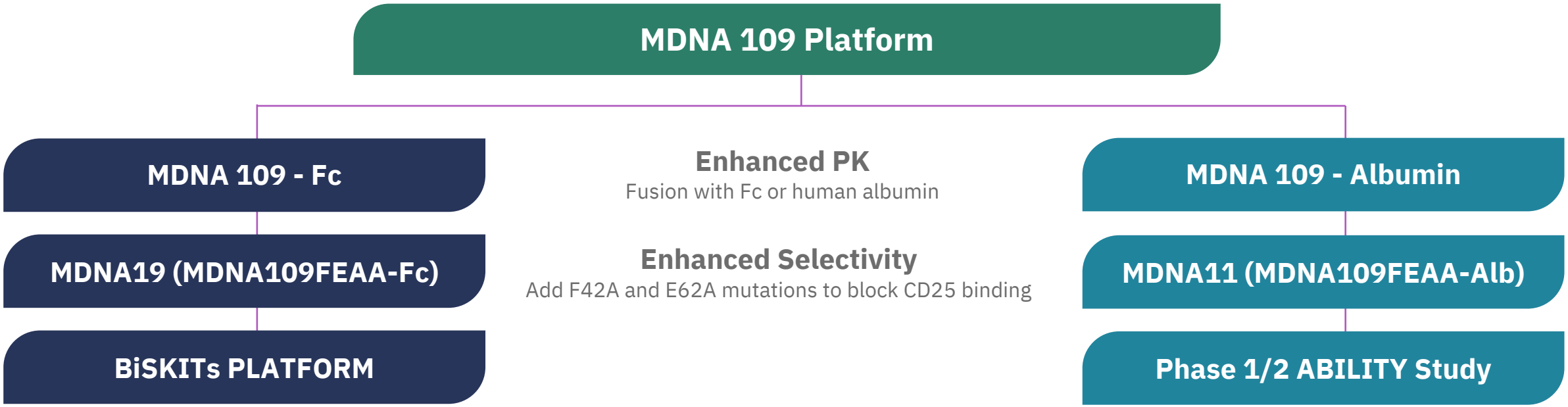
Stimulation of IL-2Rα

- Leads to activation of immunosuppressive Tregs, which abrogate the anti-tumor response
- Causes extreme toxicity

Proleukin (recombinant human [rh] IL-2), which selectively stimulates IL-2Rα, is approved for the treatment of metastatic melanoma and renal cell carcinoma

Engineering MDNA109 to Extend PK & Enhance Receptor Selectivity

Enhanced IL-2Rβ Binding and Abolish IL-2Rα Binding; Fusion to Albumin to Extend Half-Life and Bioavailability

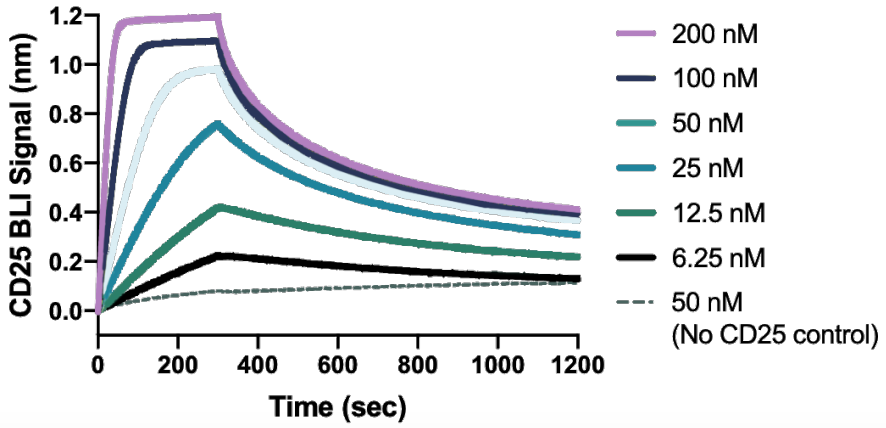


	Enhanced affinity for IL-2Rβ	IL-2Rα affinity abolished
	K_D(nM)	K_D(nM)
rhIL-2	210	24
MDNA11	6.6	No binding

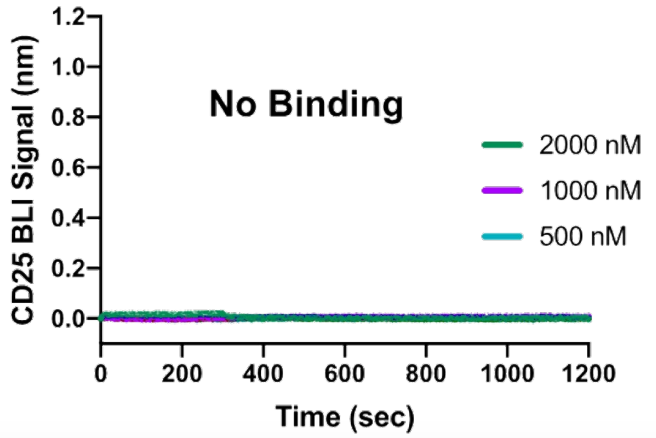
MDNA11's IL-2 Binding is Highly Differentiated vs. rhIL-2

No IL-2Rα (CD25) Binding and Enhanced Affinity and Selectivity for IL-2Rβ (CD122) Compared to rhIL-2

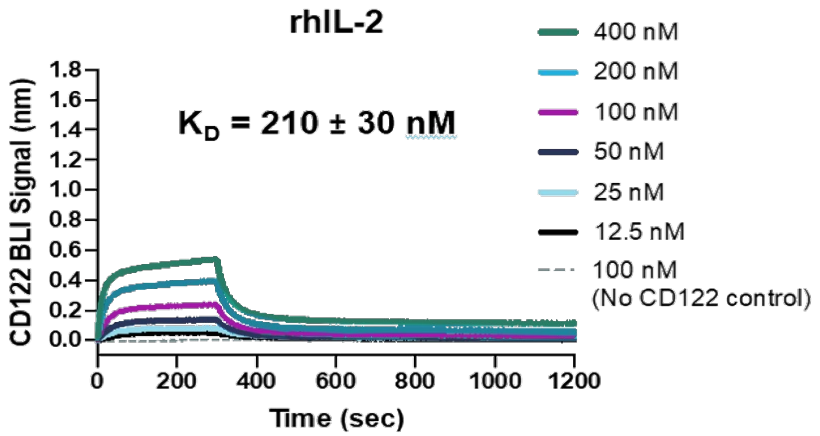
rhIL-2 – IL-2Rα Binding



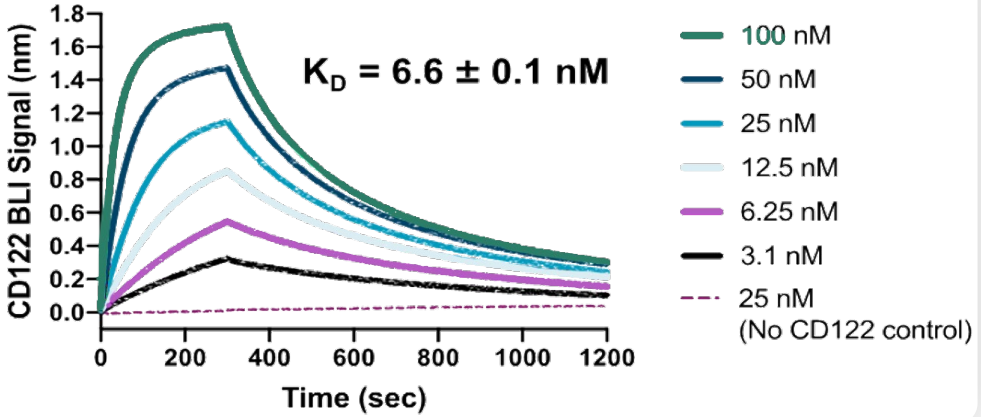
MDNA11 – IL-2Rα Binding



rhIL-2 – IL-2Rβ Binding



MDNA11 – IL-2Rβ Binding



MDNA11 –Best in Class Potential

MDNA11's strong anti-tumor activity, preliminary safety profile and convenient outpatient dosing regimen paves the way for a potential best-in-class therapy with significant commercial potential

	MDNA11	Proleukin[1]	NKTR-214	SAR'245[2]	ALKS 4230[3]	NL-201[4]	WTX-124[5]	XTX202[6]	STK-012[7]
No binding to IL-2Rα	√	✗	✗	√	√	√	✗	√	✗
Enhanced IL-2Rβγ Binding	√	✗	✗	✗	✗	√	✗	✗	✗
QW, Q2W or Q3W Dosing	√	✗	√	√	✗	√	Unknown	√	√
Tumor Accumulation	√	✗	✗	✗	✗	✗	✗	✗	✗
No Pegylation Liabilities	√	√	✗	✗	√	✗	√	√	✗
Pipeline Potential	√	√	✗	✗	✗	√	✗	✗	√

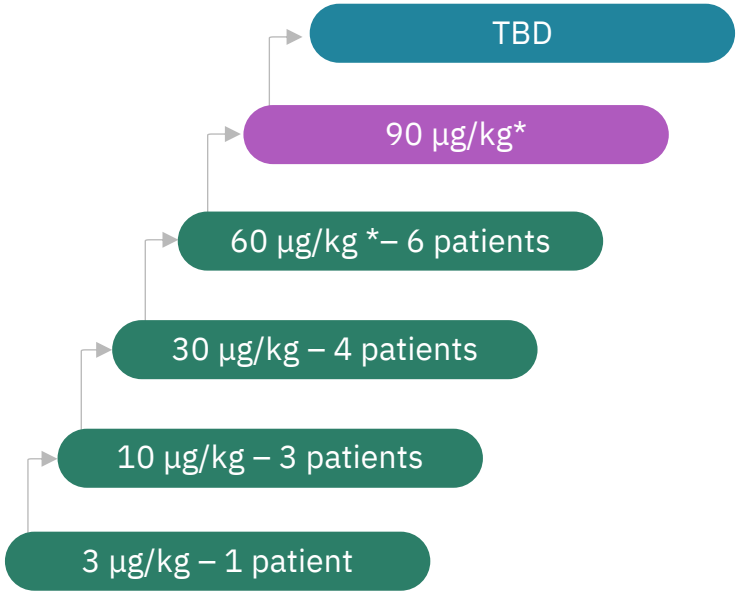
[1] Nature Rev. Drug Discovery (2021). [2] Ptacin et al., Nat Comm (2021). [3] Lopes et al., JITC (2020). [4] Da Silva et al., Nature (2019). [5] Nirschl et al, Cancer Immunol Res (2022). [6] O'Neil et al., ASCO (2021). [7] Oft et al, AACR (2021). Additional information from https://clinicaltrials.gov/

Phase 1/2 ABILITY Study Schema: Enrolling Dose Level 5

MDNA11

Monotherapy Dose Escalation

N ~ 20: Select tumors



Modified, accelerated 3+3 design

Intra-patient dose escalation permitted on sponsor approval

DLT assessment

Identify RP2D

MDNA11 Monotherapy Dose Expansion

N~ 40: Melanoma, RCC and other select tumors (1:1:1)

MDNA11 administered alone at RP2D via IV infusion Q2W or Q3W

Signals of anti-tumor activity

MDNA11 + CPI Dose Expansion

N~ 40: Melanoma, RCC and other select tumors (1:1:1)

Safety run-in

MDNA11 administered at RP2D in combination with CPI via IV infusion Q3W (planned)

Signals of anti-tumor activity

Endpoints:

Safety and tolerability

ORR (RECIST 1.1)

Clinical Benefit Rate (CBR) (CR+PR+SD)

Survival EPs (TTE Analysis) PFS/OS

Disease Control Rate (DCR)

Duration of Response (DoR)

Time to Relapse (TTR)

Pharmacodynamic Assessment:

Immune Cell Profiling (Blood)

Serum Cytokines

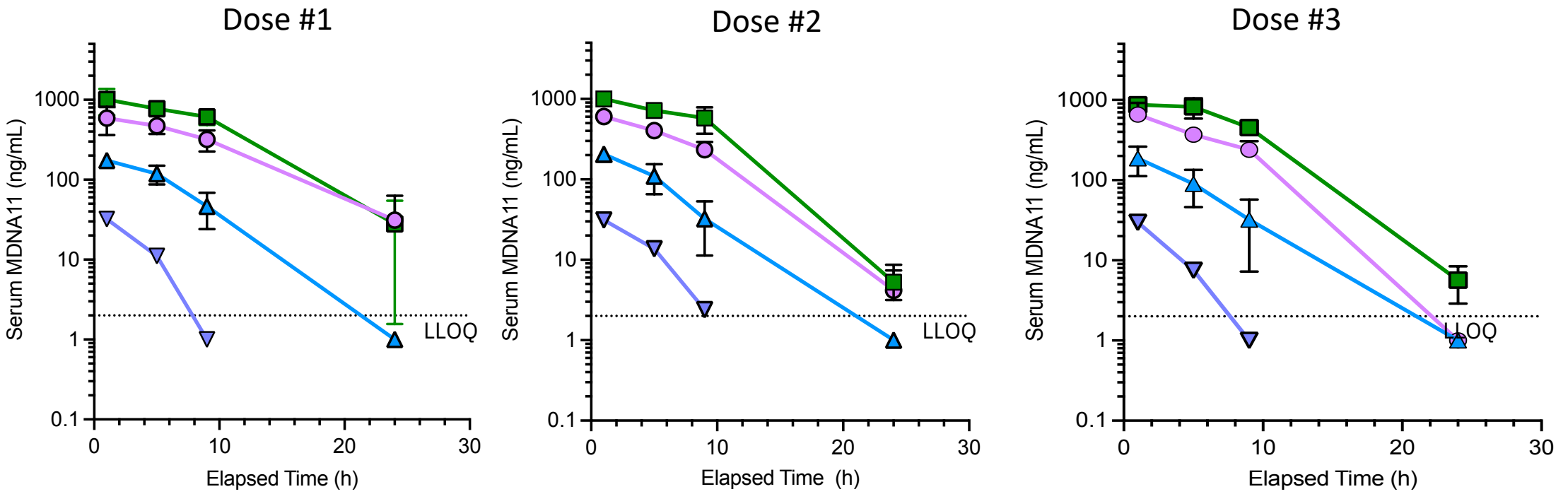
Multiplex Immunofluorescence (Paired tumor biopsies)

NanoString Gene Expression (Paired tumor biopsies)

*Step-up dosing utilized: two priming doses of 30µg/kg given before target dose. Protocol Version 5

MDNA11 PK Profile in Cancer Patients

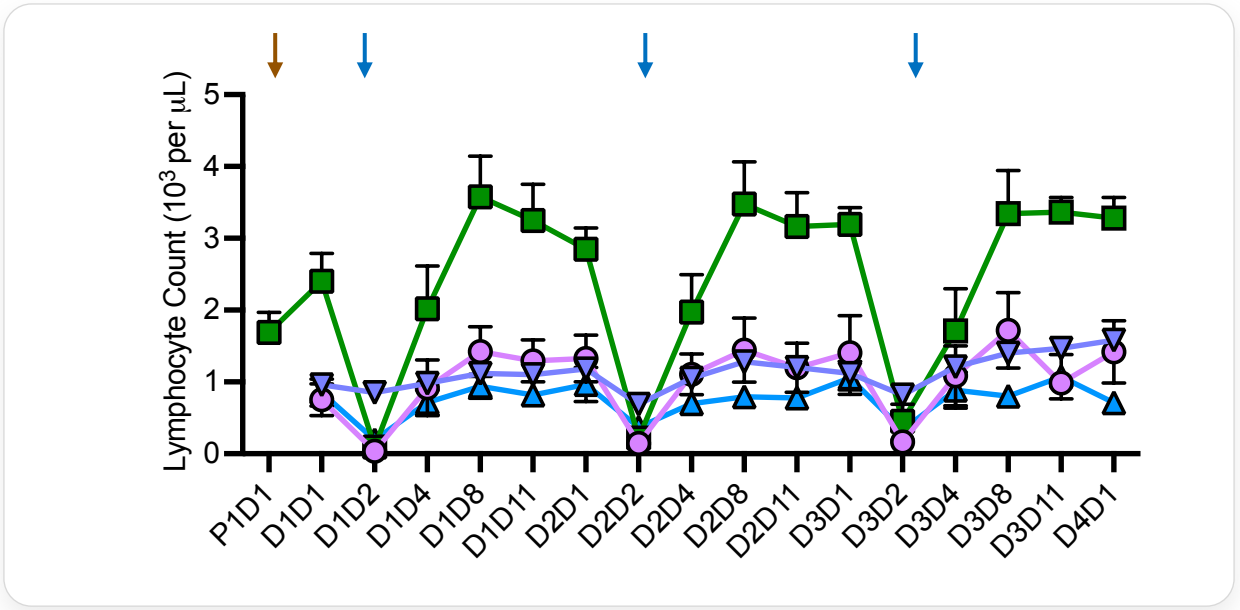
- MDNA11 PK exhibits saturable rapid clearance and a slower parallel linear clearance process
- Dose-dependent increase in exposure (C_{max} and AUC_{last})
- Variability is low between Dose 1-3, suggesting that there is no clinically significant ADA response



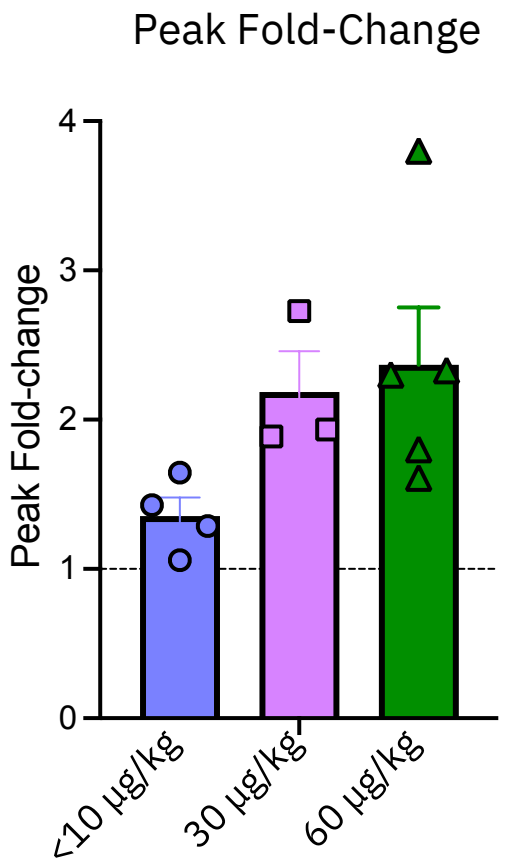
LLOQ = lower limit of quantification
Values < LLOQ plotted as 0.5 x LLOQ

MDNA11 Induced Lymphocyte Expansion

Expansion of cancer killing immune cells



	Average AUC (day.10³ cells/µL) (Average of Dose 1 & 2)
< 10 µg/kg	3
30 µg/kg	4.8
60 µg/kg	12.2



Peak fold-change relative to baseline.

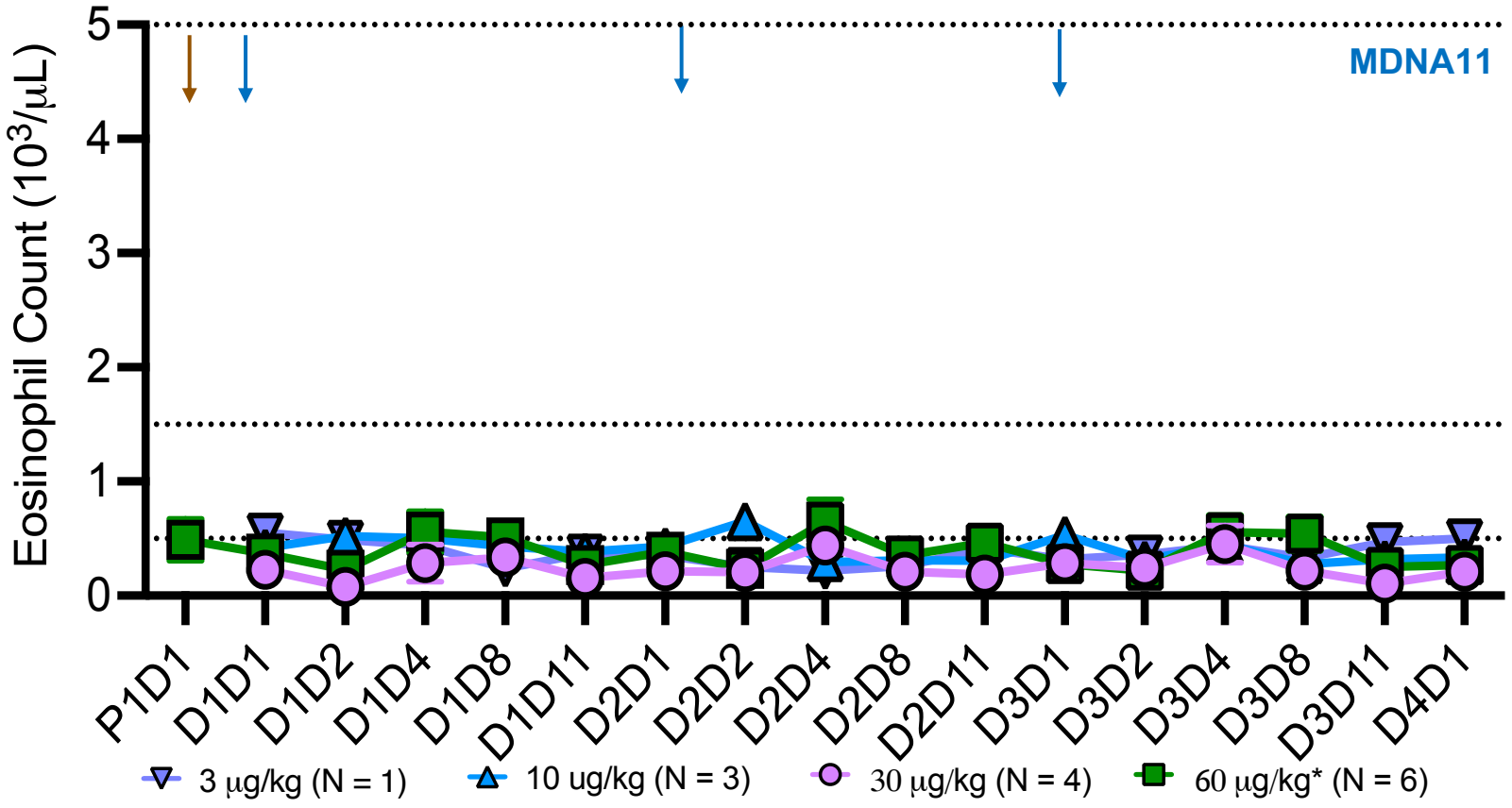
Graph shows mean ± SEM

For < 10 µg/kg and 30 µg/kg, peak data for Dose 3

For 60 µg/kg, peak data for Target Dose 1

DL4 patients received 2 priming doses (30 µg/kg Q2W) prior to target dose (60 µg/kg Q2W)
Graph shows mean ± SEM. AUC measured as area between minimum lymphocyte count values

Improved Safety Profile - No Evidence of Eosinophilia



Severe Eosinophilia

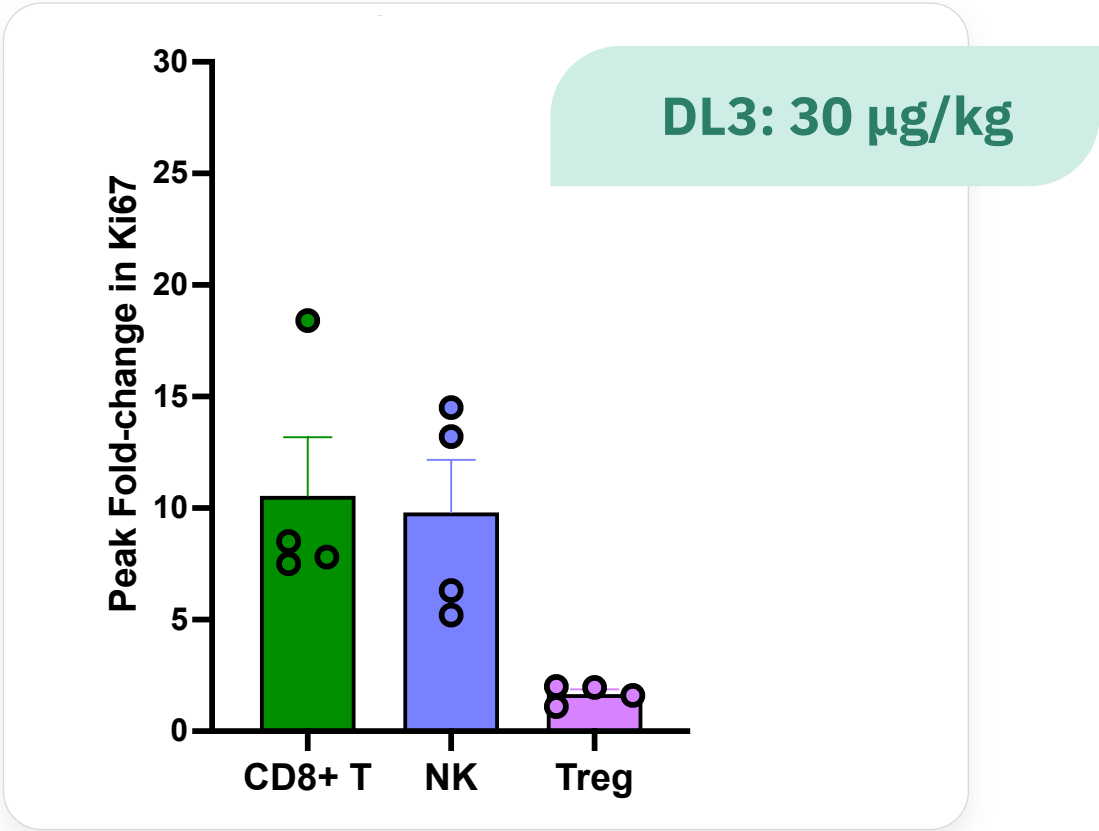
Moderate Eosinophilia

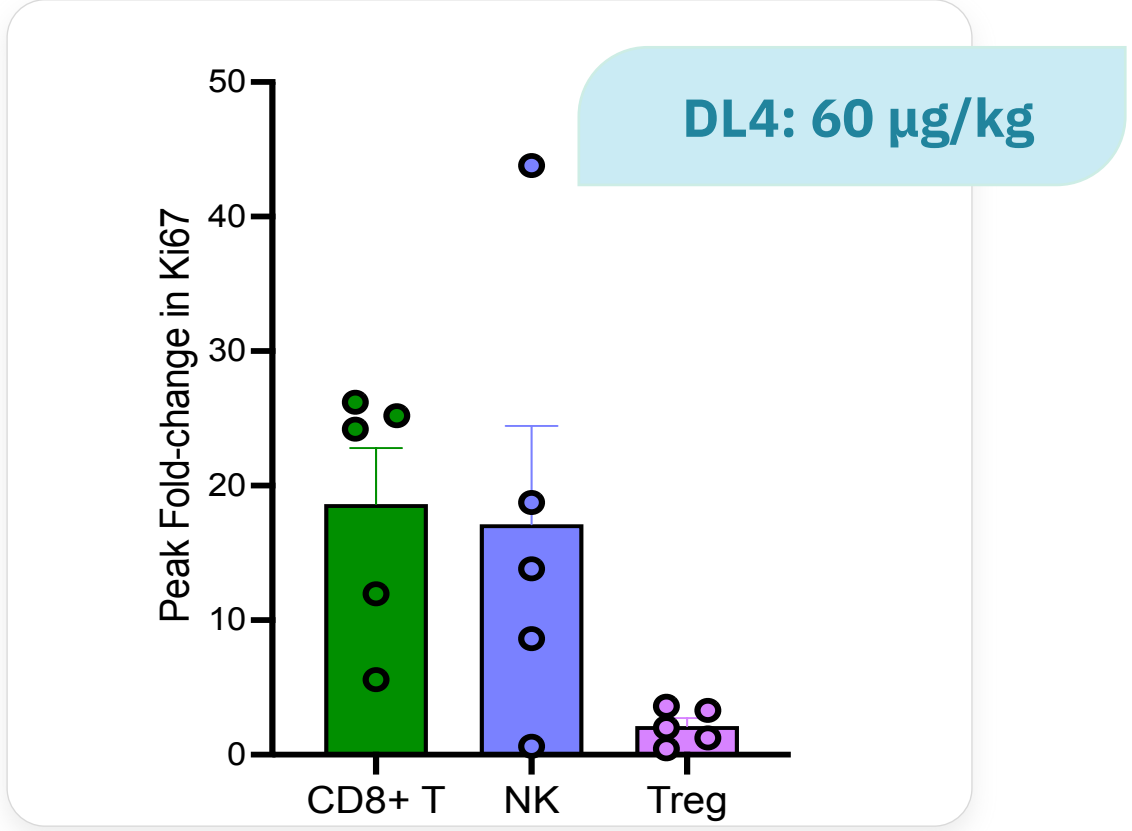
Mild Eosinophilia

Graphs show mean ± SEM.

MDNA11 Stimulated CD8+ T and NK Cell Proliferation (Ki67)

No increase in Tregs



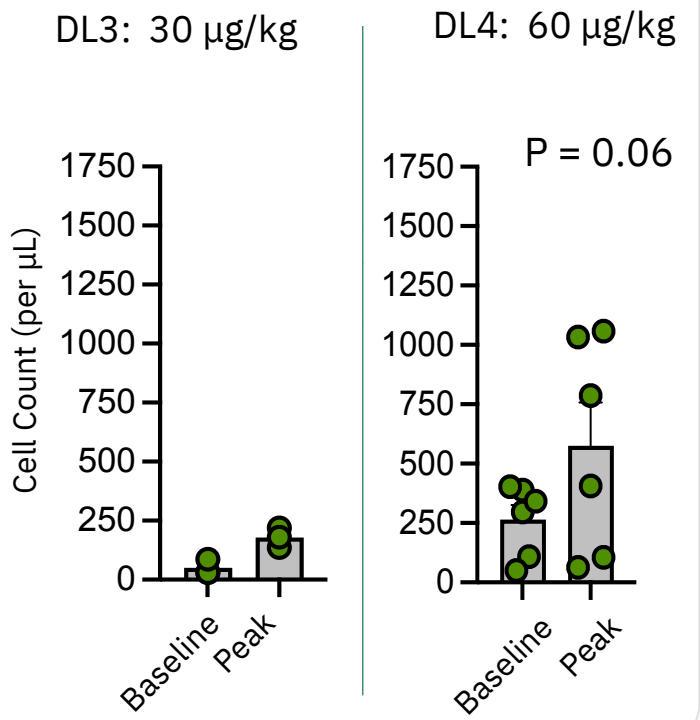


- Peak fold-change relative to respective baseline (D1D1 for DL3; P1D1 for DL4)
- DL4 patients received 2 priming doses (30 µg/kg Q2W) prior to start of target dose of 60 µg/kg (Q2W)
- DL3 data based on 2nd/3rd dose cycle
- Graphs show mean ± SEM

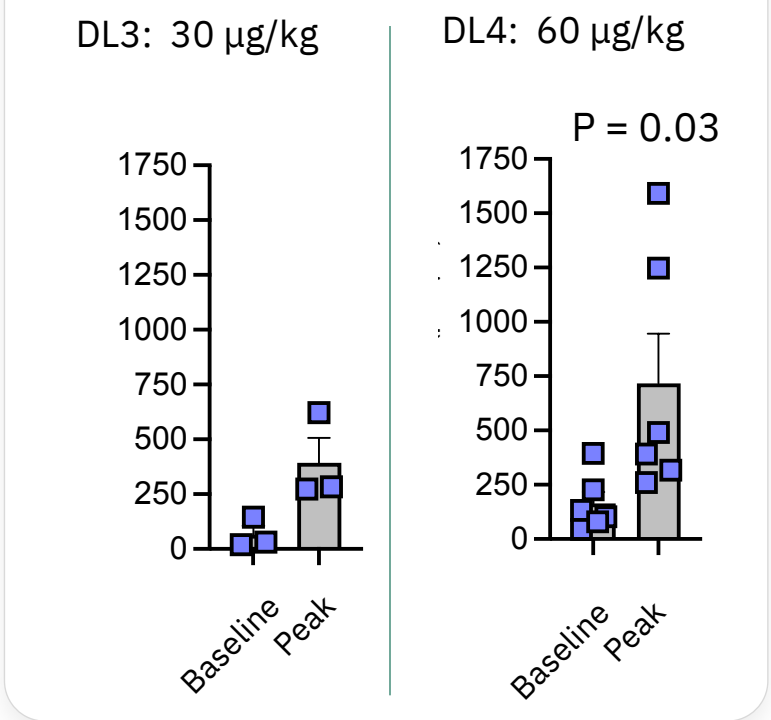
MDNA11 Preferentially Expanded CD8+ T & NK Cells Over Tregs

Peak fold change in cell count

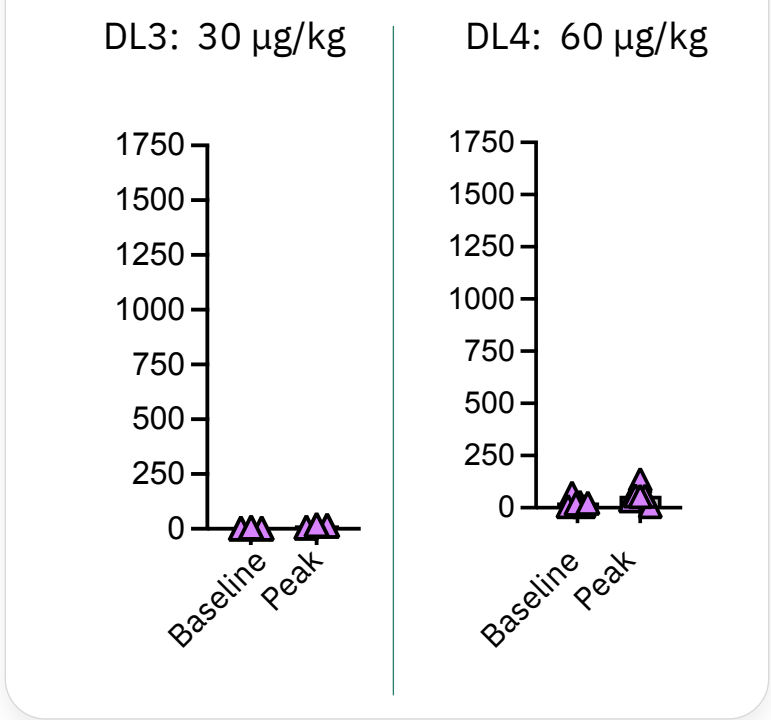
CD8+ T Cells



NK Cells



Tregs



Peak fold-change relative to baseline
Patients received 2 priming 30 µg/kg doses (Q2W) prior to the targeted 60 µg/kg (at 3rd administration).
Data shown for 30 µg/kg cohort are based on 3rd administration for comparison

Patients in ABILITY's Dose Escalation Cohorts Heavily Pre-treated

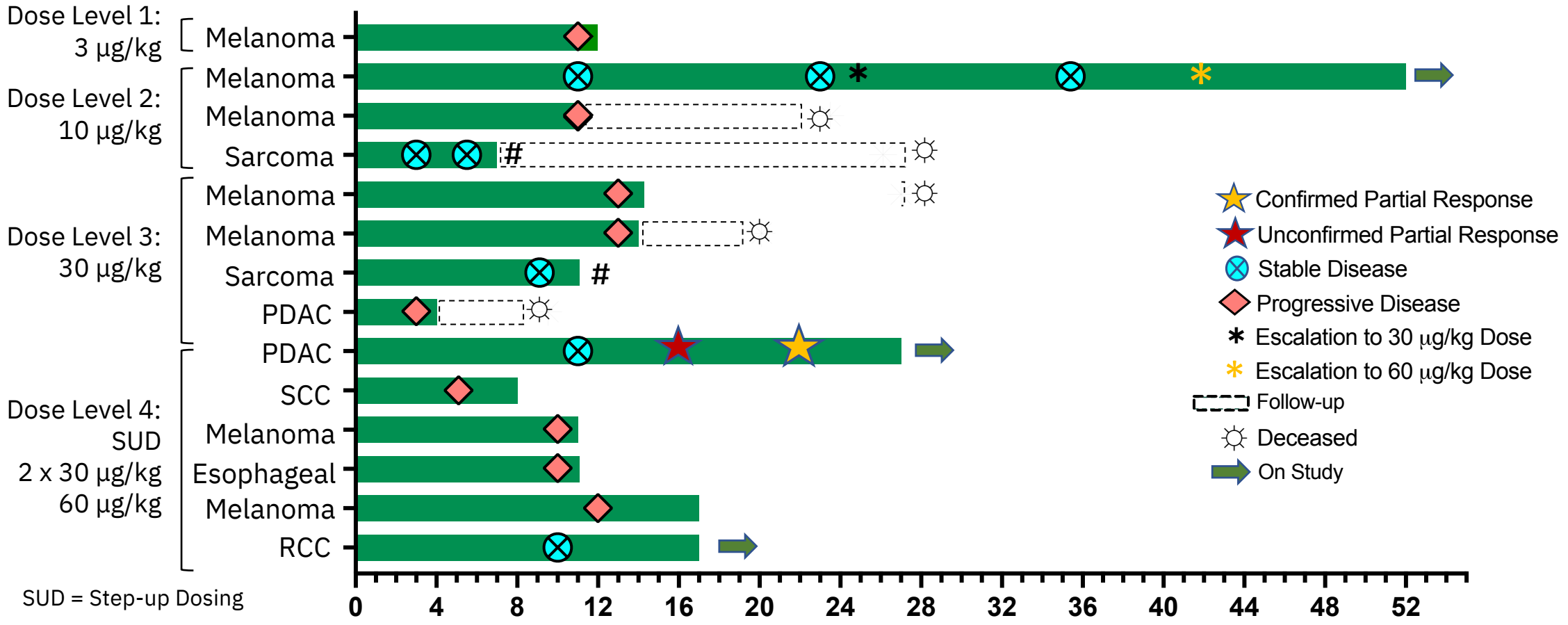
- All patients have advanced solid tumors and failed prior therapy

Demographics/Performance	
Median age (range), years	63 (27-78)
Male (%)	11/14 (79%)
Baseline ECOG = 0	10/14 (71%)
Baseline ECOG = 1	4/14 (29%)
Primary Cancer Diagnosis	
Melanoma	7/14 (50%)
Renal Cell Carcinoma (non-clear cell)	1/14 (7%)
Pancreatic Ductal Adenocarcinoma (PDAC)	2/14 (14%)
Sarcoma	2/14 (14%)
Squamous Cell Carcinoma	1/14 (7%)
Gastro-esophageal Adenocarcinoma	1/14 (7%)

Prior Systemic Therapies	
Prior Lines of Therapy: 1-2	9/14 (64%)
Prior Lines of Therapy: 3-4	5/14 (36%)
Prior use of immunotherapy	11/14 (79%)
Prior use of targeted therapy	4/14 (28%)
Prior use of chemotherapy	7/14 (50%)

Treatment Duration and Tumor Response

- Tumor control in 5 of 14 evaluable patients (including 1 confirmed PR in PDAC) despite low dose levels and heavily pre-treated patients



SUD = Step-up Dosing

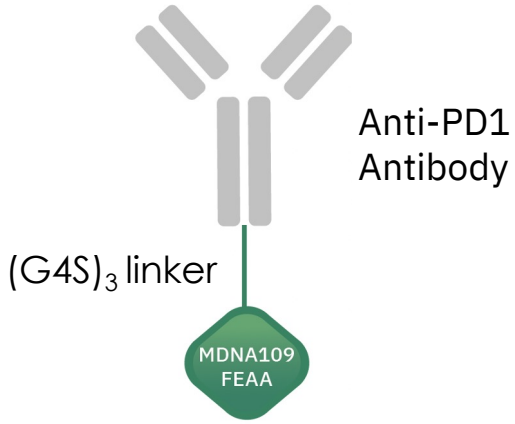
Target lesions exhibit SD;
PD due to clinical progression or patient withdrawal

Data cut off: October 21, 2022

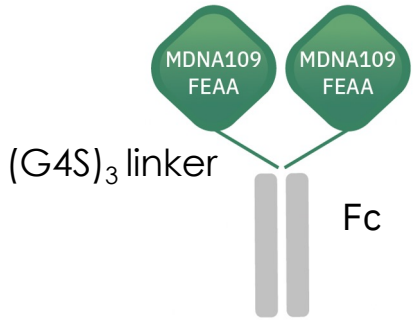
Bifunctional **S**uper**K**ines for **I**mmuno**T**herapy (BiSKIT)

Overview of Anti-PD1-IL-2 Superkine BiSKIT (MDNA223)

MDNA223

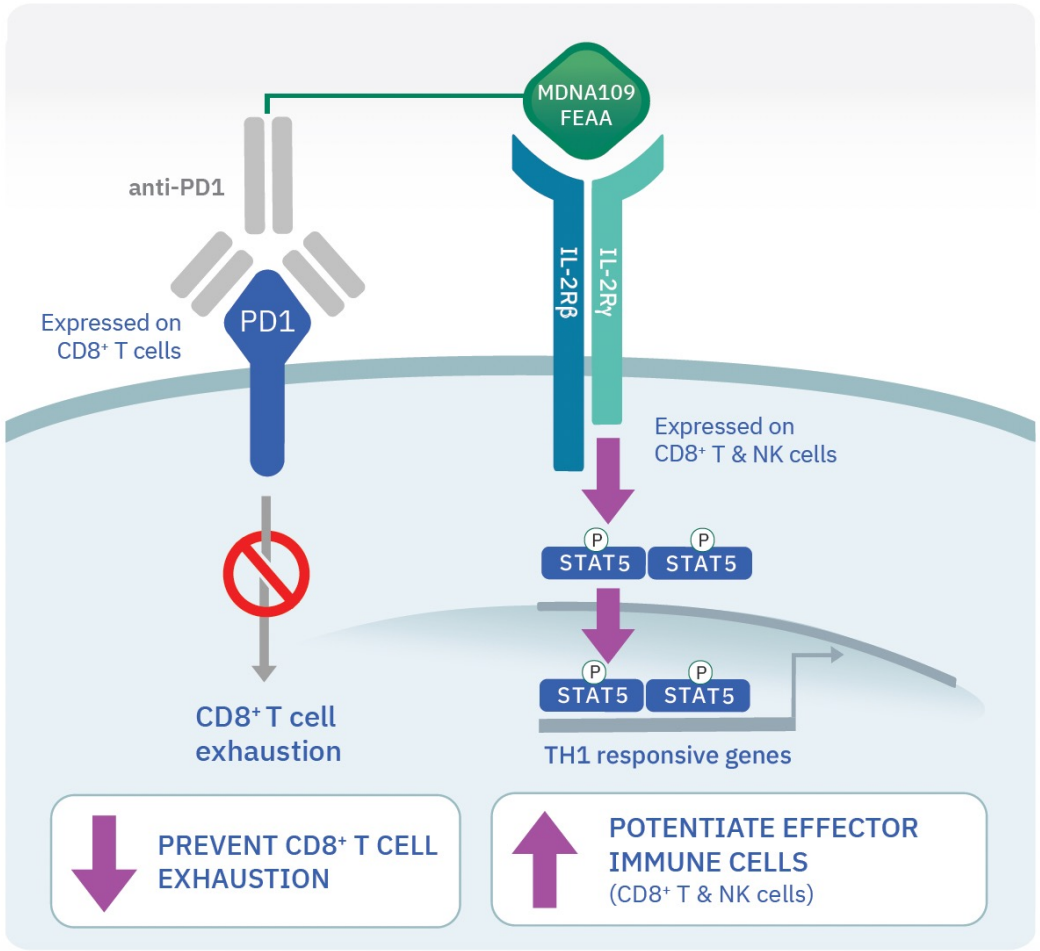


MDNA19
(MDNA109FEAA-Fc)



cis-Binding to IL-2R and PD1 on Same CD8⁺ T Cell

Anti-PD1-MDNA109FEAA **(MDNA223)**



Synchronized

IL-2R Activation
+
PD1 Blockade

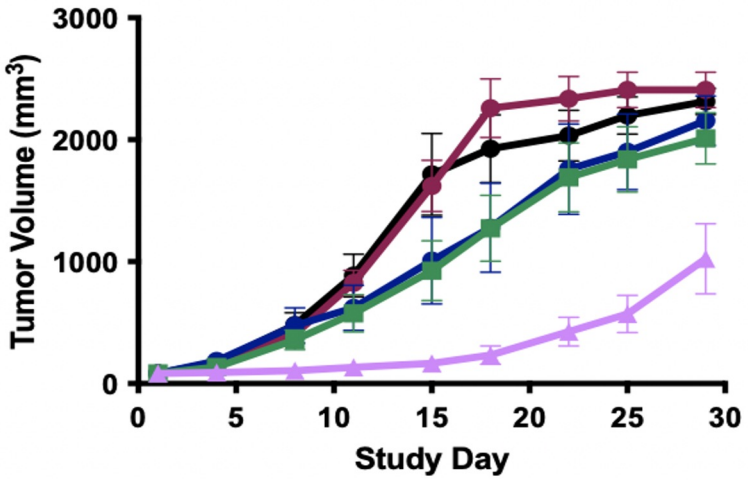
↓

Superior
Anti-Tumor
Activity

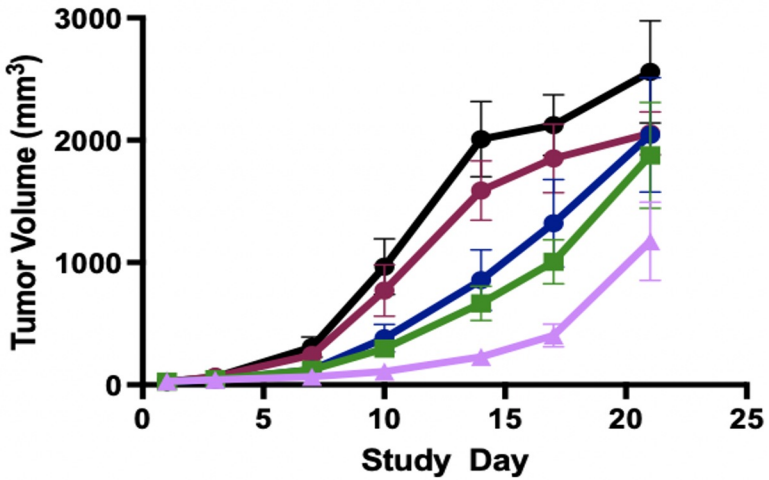
MDNA223m Demonstrated Superior Anti-Tumor Activity

MDNA223m showed higher levels of anti-tumor activity than co-administration in pre-clinical studies

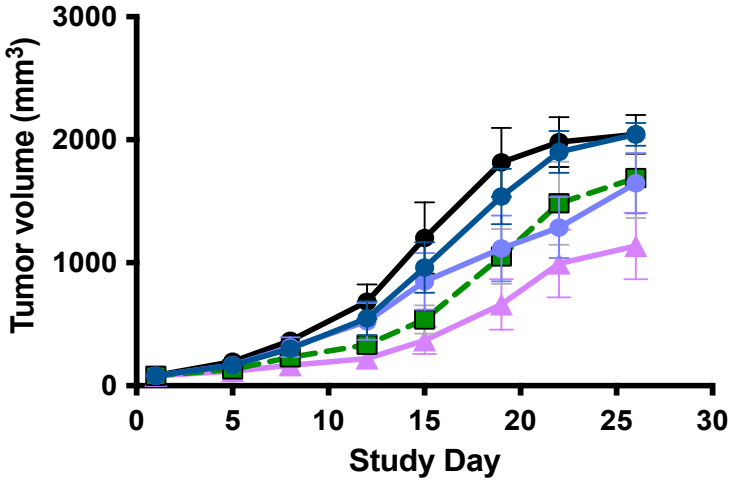
CT26 Colon Tumor Model



B16F10 Melanoma Model



E0771 Breast Tumor Model



- Vehicle
- Anti-PD1 (1.8 mg/kg)
- MDNA19 (1 mg/kg)
- Anti-mPD1 (1.8 mg/kg) + MDNA19
- MDNA223m (2 mg/kg)

- Vehicle
- MDNA19 (1 mg/kg)
- Anti-mPD1 (10 mg/kg)
- Anti-mPD1 (10 mg/kg) + MDNA19
- MDNA223m (2 mg/kg)

Treatment with molar equivalent doses of anti-PD1 (150 Kda), MDNA19 (83 Kda) or MDNA223m (165 Kda).
IP treatment QWx2 (CT26 and E0771) and QWx3 (B16F10)
Avg tumor size at initiation of dosing: 127 mm^3 (CT26), 80 mm3 (E0771) or 30 mm^3 (B16F10)

MDNA55

Empowered IL-4
Superkine Targeting
Glioblastoma

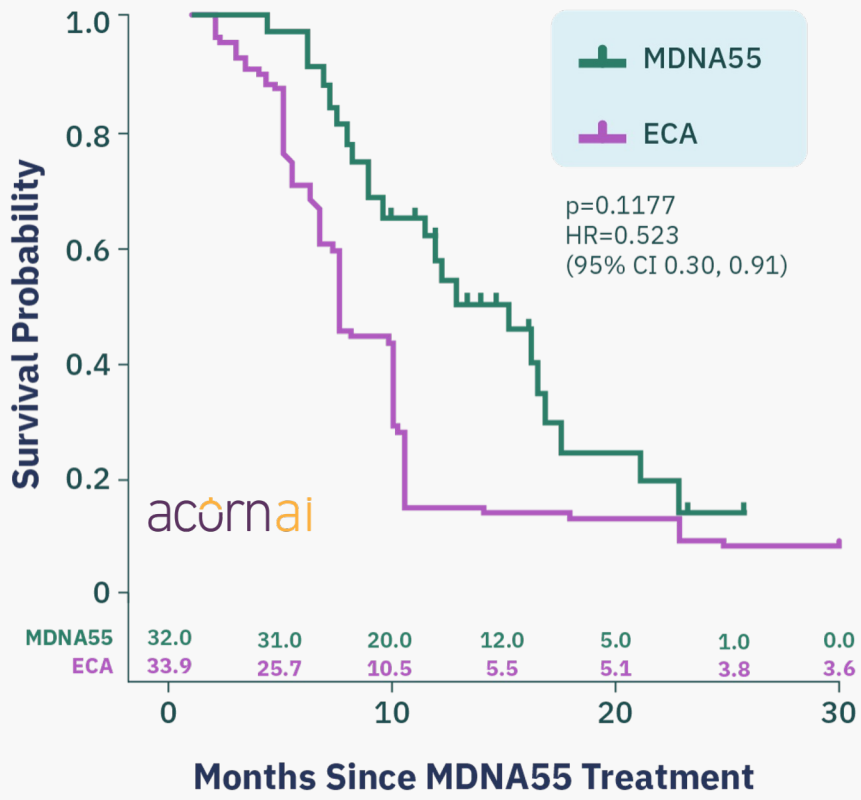
Improvement of ~ 100% in mOS vs External Control Arm (ECA)

Results*

Weighted IL4R High + IL4R Low$^{\text{High Dose}}$ (n=32)
mOS is 15.7 months vs 7.2 months in ECA

→ Survival time more than doubled in the IL4R High + IL4R Low$^{\text{High Dose}}$ group compared to ECA

IL4R High + IL4R Low$^{\text{High Dose}}$



*Survival was calculated from time of relapse

Upcoming Anticipated Milestones & Financial Summary

- ABILITY Study Fully Funded – Cash Runway into Q2 2024

Anticipated Milestones

Dose Escalation Cohorts Anti-Tumor Update	Activity Data **(Q1 2023)**
Early Single Agent Expansion Anti-Tumor	Activity Data **(Mid 2023)**
Early Combination Study Anti-Tumor	Activity Data **(Q4 2023)**

Financial Highlights

Nasdaq/TSX	MDNA
Headquarters	Toronto, CA
Cash	CDN $40M **
Debt	$0
Preferred Shares	None
Issued and Outstanding	~70 Million*
Fully Diluted	~91 Million*

* As of November 3, 2022. **As of September 30, 2022

Thank you

Fahar Merchant, PhD
President and CEO

Elizabeth Williams
Chief Financial Officer

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