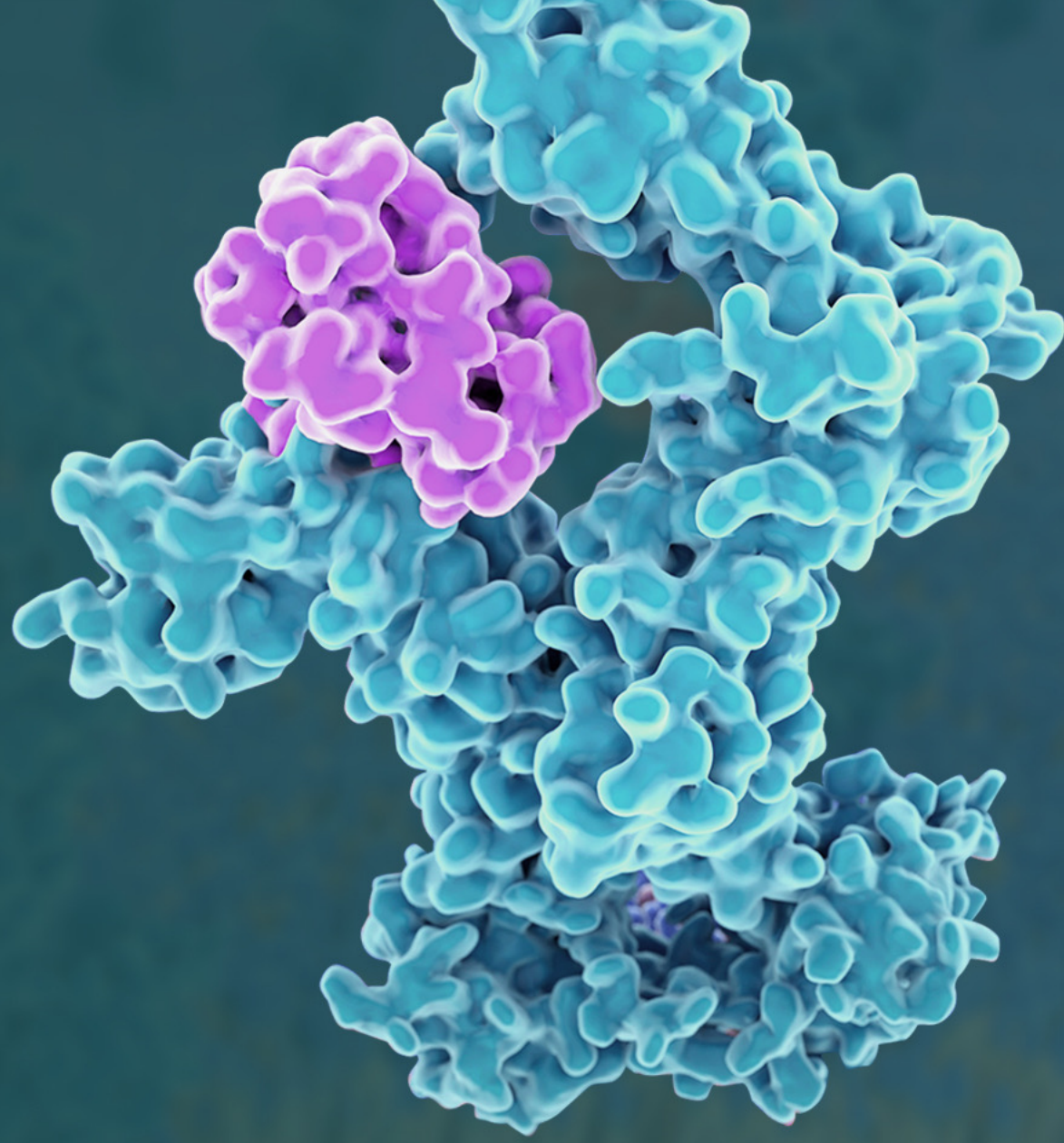
Q4, 2021

Evolutionary Cytokines Revolutionary Medicines



MEDICENNA

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Investment Highlights

Clinical Data Updates from MDNA11 Program Expected in Q4 2021 and H1 2022

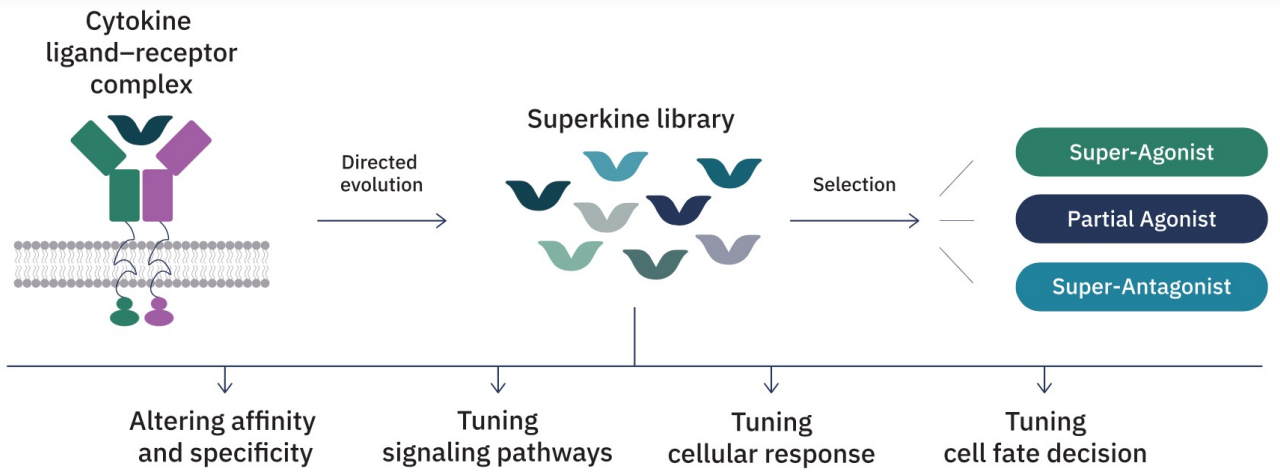
Superkine Platform: Drug Discovery Engine	Directed evolution **enhances the desired properties** of IL-2, IL-4, & IL-13 to generate Superkines Protein fusion can **improve PK, add an MOA, or confer new capabilities** to Superkines IL-2, IL-4 , & IL-13 are known to modulate immune activity against **2,000 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 **in Q4 2021** and **H1 2022**
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

Experienced Management Team

Fahar Merchant, PhD	President and Chief Executive Officer	Protox Therapeutics; KS Biomedix; Avicenna Medics Inc.
Elizabeth Williams, CPA, CA	Chief Financial Officer	Aptose Biosciences; EY
Rosemina Merchant, MESc	Chief Development Officer	Protox Therapeutics; KS Biomedix; GE Healthcare Life Sciences; Sanofi Pasteur
Kevin Moulder, PhD	Chief Scientific Officer	gsk; F-star Therapeutics; Biogen; PolyProx Therapeutics; Tusk Therapeutics
Mann Muhsin, MD	Chief Medical Officer	Nektar; Halozyme; Huyabio International; oncosec Immunotherapies

Superkine Platform Powers Drug Discovery Engine

Transforming Interleukins into Visionary Superkines with Directed Evolution



Our Superkines are derived from IL-2, IL-4, & IL-13, which are known to modulate immune activity against 2,000 different diseases

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

Clinical Stage Pipeline

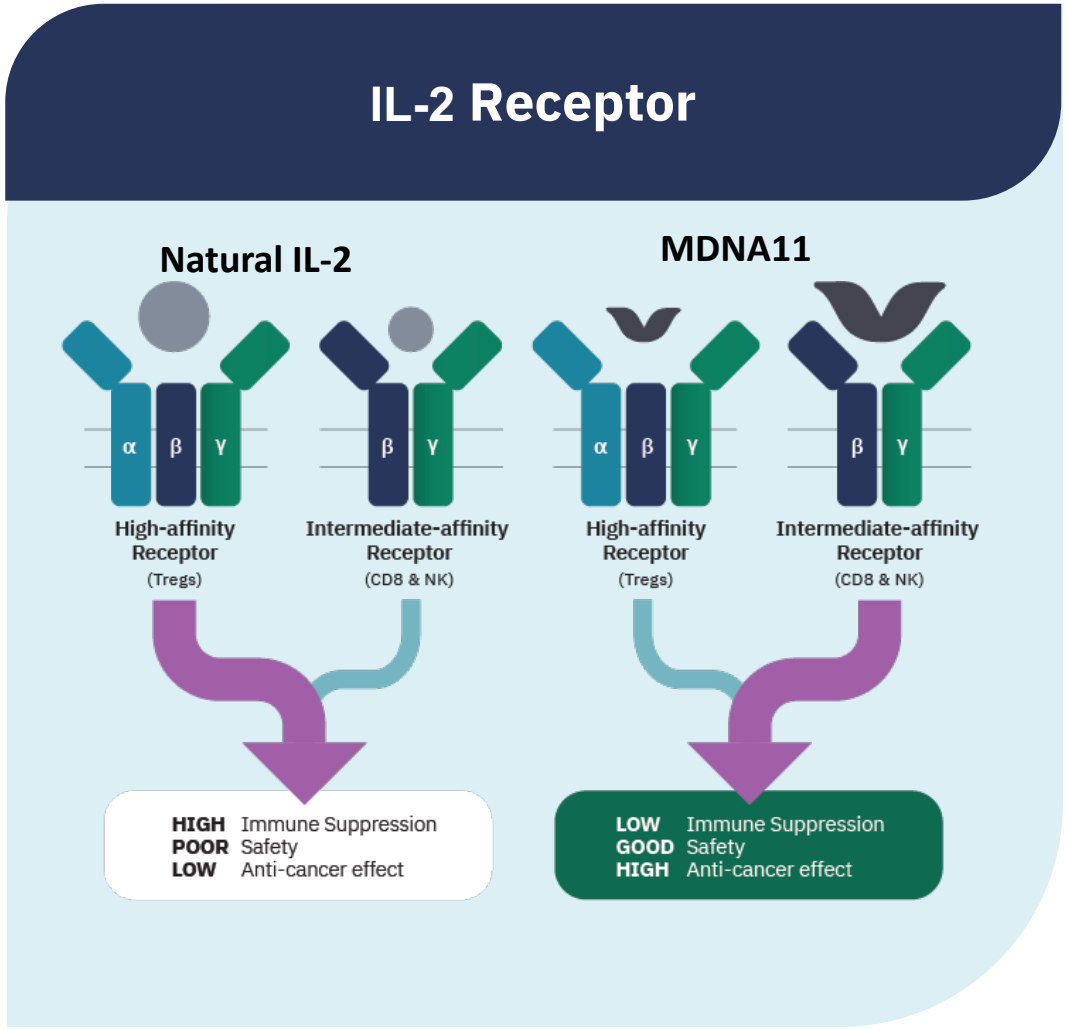
Leveraging the Superkine Platform to Develop Novel Interleukin-based Therapies

Candidate	Indication	Preclinical	Phase 1	Phase 2	Pivotal
MDNA55 **IL-4 Empowered Superkine**	Recurrent Glioblastoma (rGBM)	**Future development to be funded by partner*			
MDNA11 **"Beta-only" & Long Acting IL-2 Super-Agonist**	Solid Tumors	*Initiated September 2021*			

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

Improved IL-2 Variants are Needed

Proleukin and “Pegylated Not-alpha” IL-2 Variants Have Substantial Shortcomings

Proleukin (Recombination Human IL-2)

Poor safety profile due to selective stimulation of IL-2Rα

- Patients are often unable to receive a full course of therapy
- Patients must be treated in the intensive care unit

Poor pharmacokinetic profile

- Half-life on the order of minutes
- Requires dosing every 8 hours for 5 days

“Pegylated Not-alpha” IL-2 Variants

Have low IL-2Rβ affinity

- Limits efficacy due to poor stimulation of immune effector cells

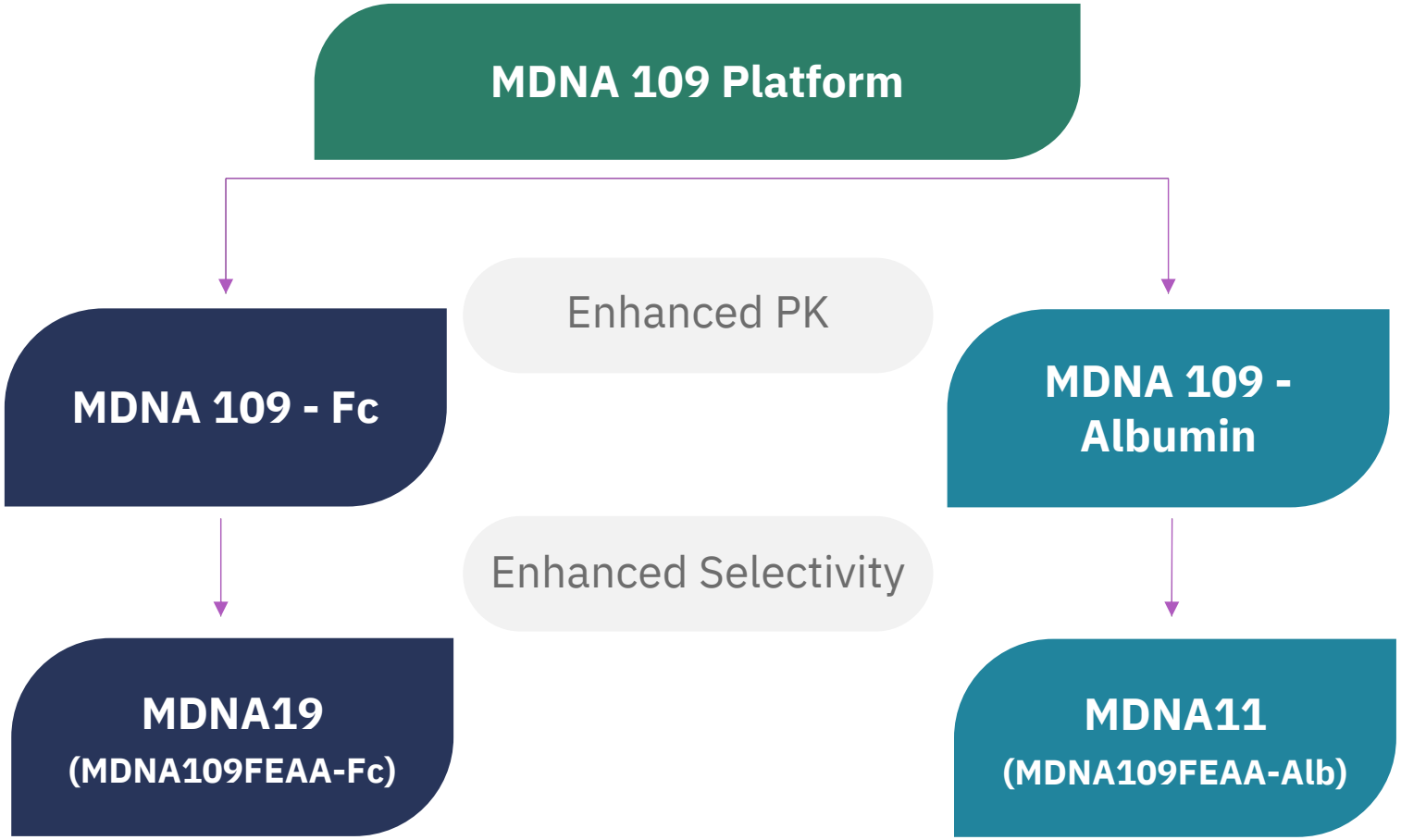
Require complex manufacturing processes

- Increases cost of goods
- Batch-to-batch heterogeneity

To overcome these shortcomings MDNA11 utilizes a differentiated “beta-only” approach with albumin to extend the half life (not PEG)

MDNA11: A “Beta-only” IL-2 Super-agonist

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

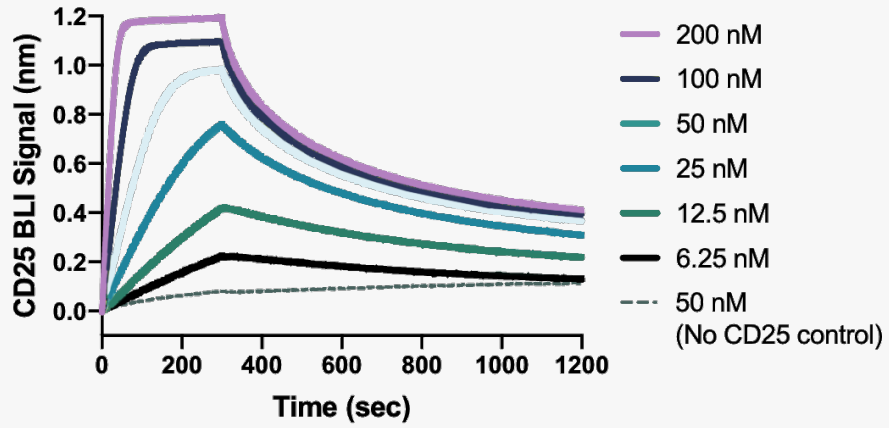


	Enhanced affinity for IL-2Rβ	IL-2Rα affinity abolished
	K_D(nM)	K_D(nM)
rhIL-2	210	24
MDNA11	6.6	No binding

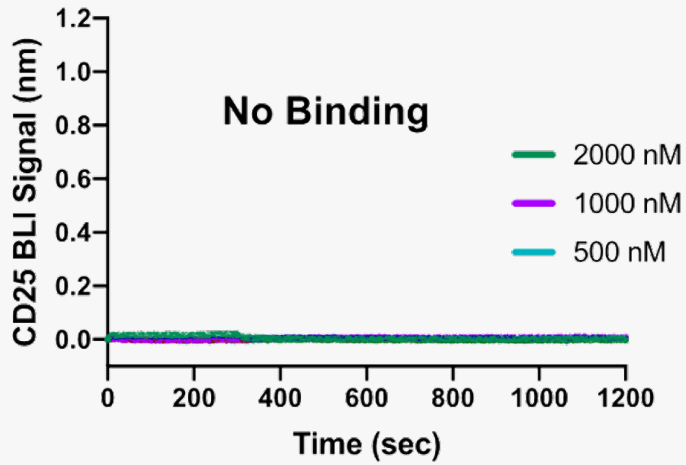
MDNA11

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

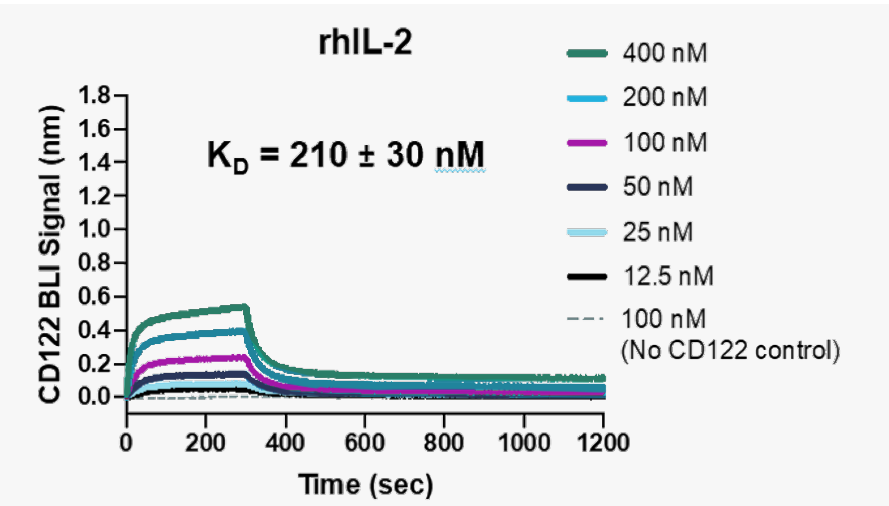
rhIL-2 – IL-2Rα Binding



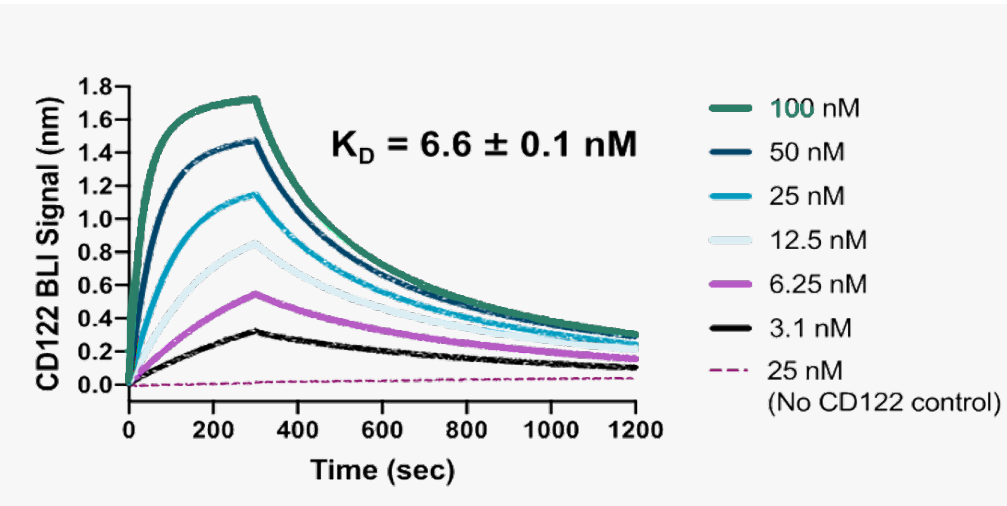
MDNA11 – IL-2Rα Binding



rhIL-2 – IL-2Rβ Binding

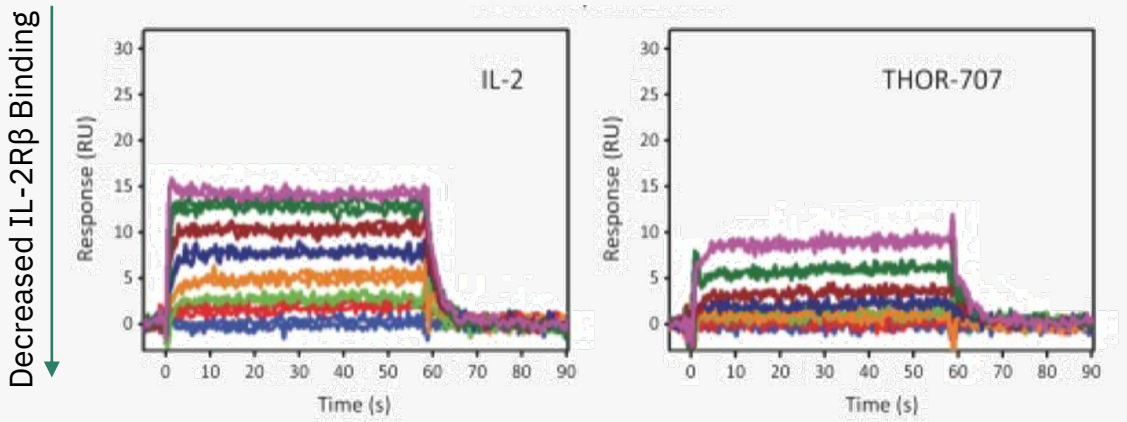


MDNA11 – IL-2Rβ Binding

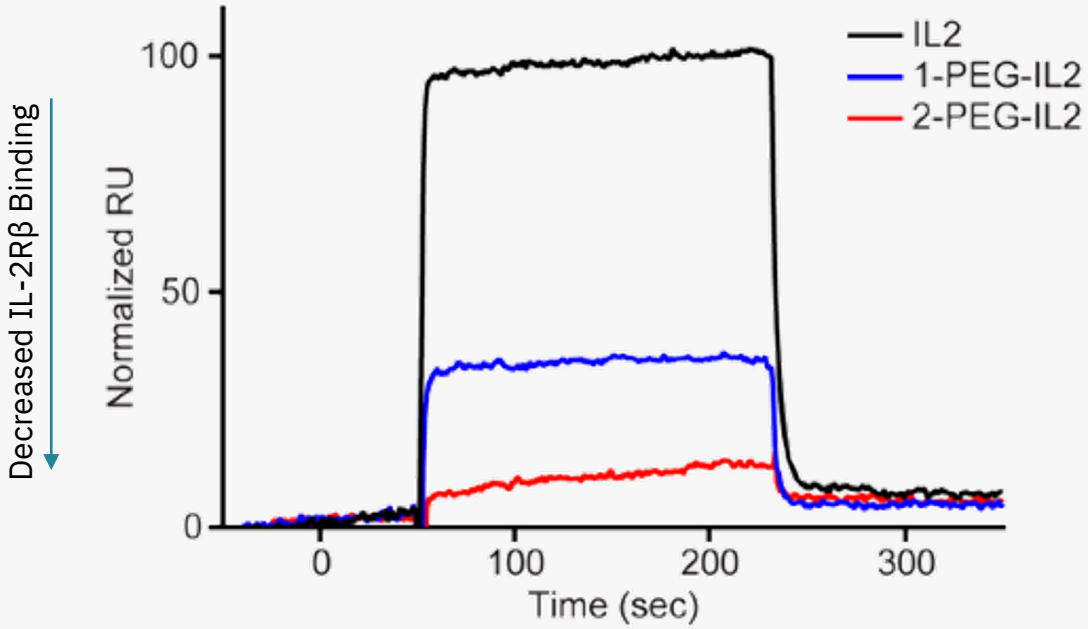


Competing IL-2 Variants are Weak IL-2Rβ (CD122) Binders

Sanofi's THOR707 (aka SAR444245): Reduced Binding to IL-2Rβ (CD122)



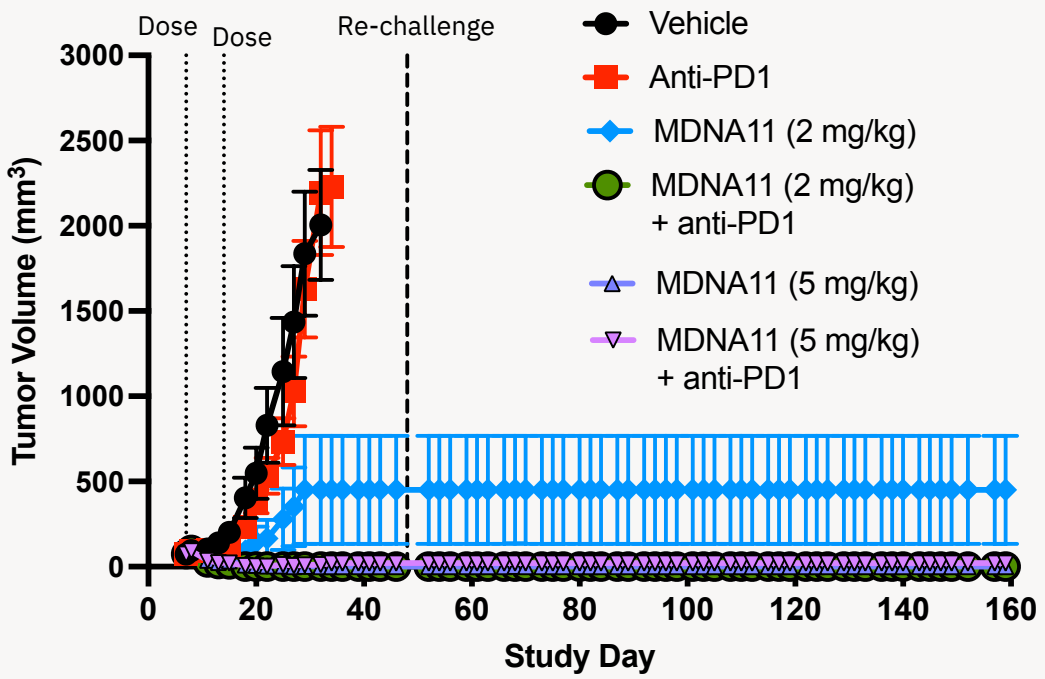
Nektar's 1-PEG-IL2 (Most Active Form of Bempeg) is a Weak IL-2Rβ (CD122) Binder



Strong Monotherapy and Anti-PD1 Combo Effect

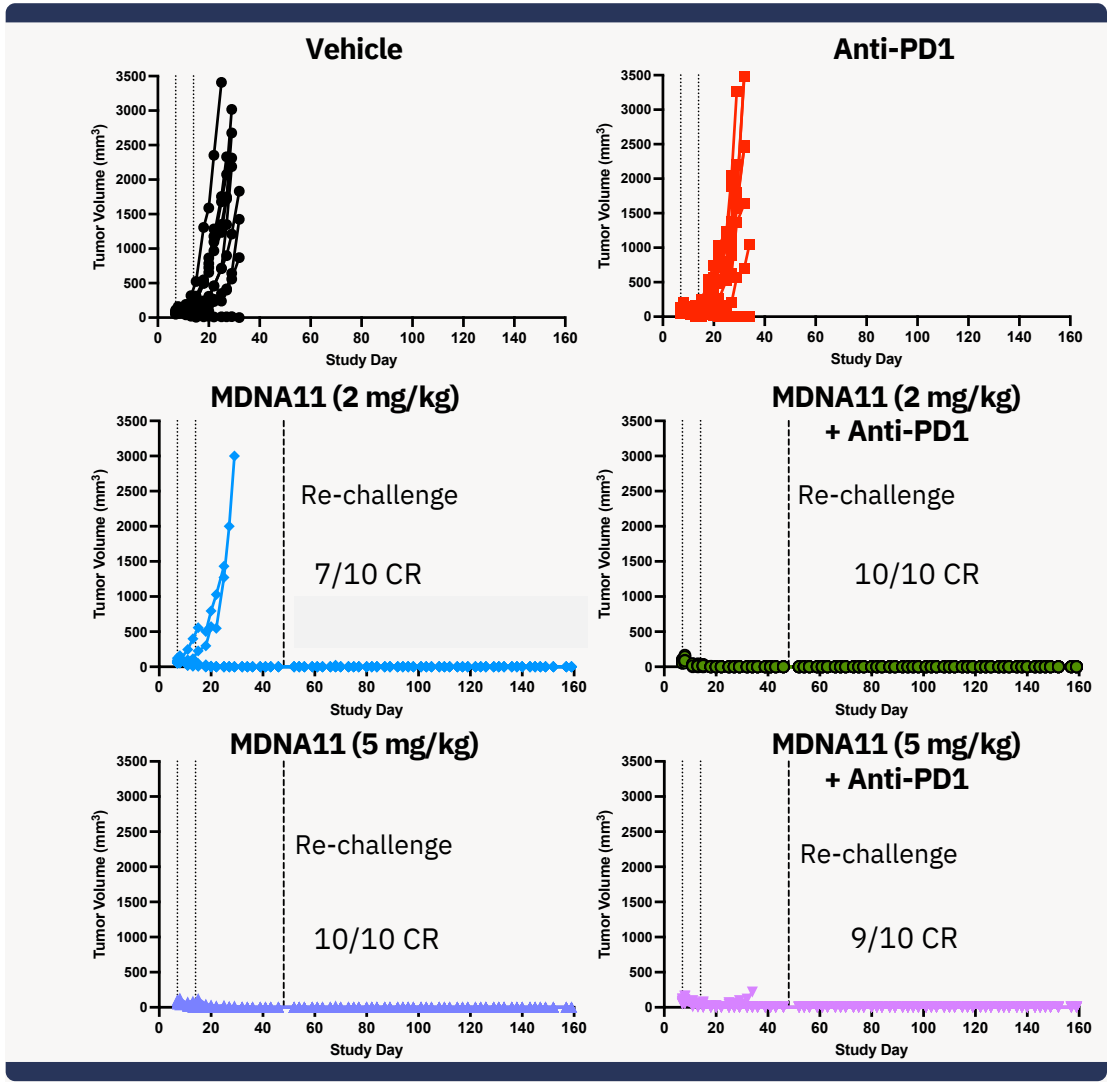
Potent Anti-Tumor Efficacy With or Without anti-PD1 in MC38 Tumor Model

MC38 Tumor Model



MDNA11: IP QWx2
Anti-PD1 (RMP1-14; 10 mg/kg): IP BIWx3
Average size at initiation of dosing ~ 75 mm^3
Study in C57Bl/6 mice.

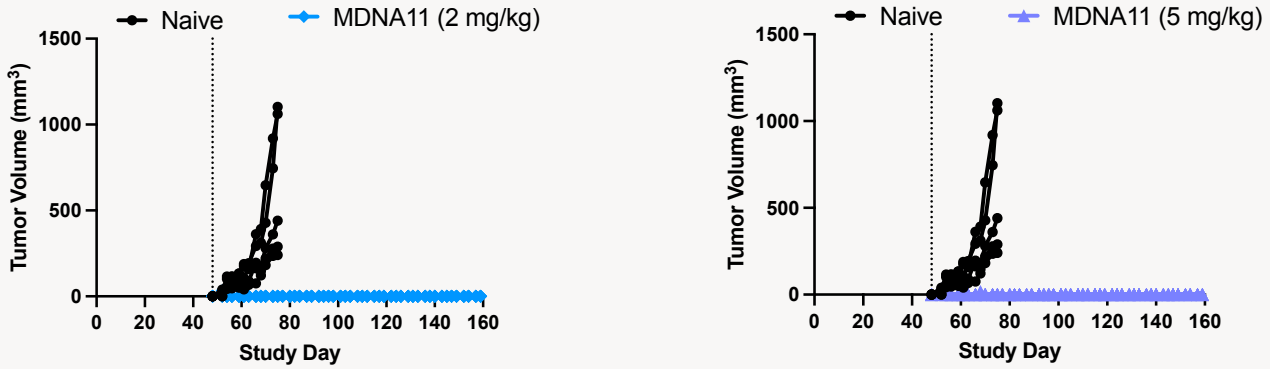
CR: Tumor volume = 0



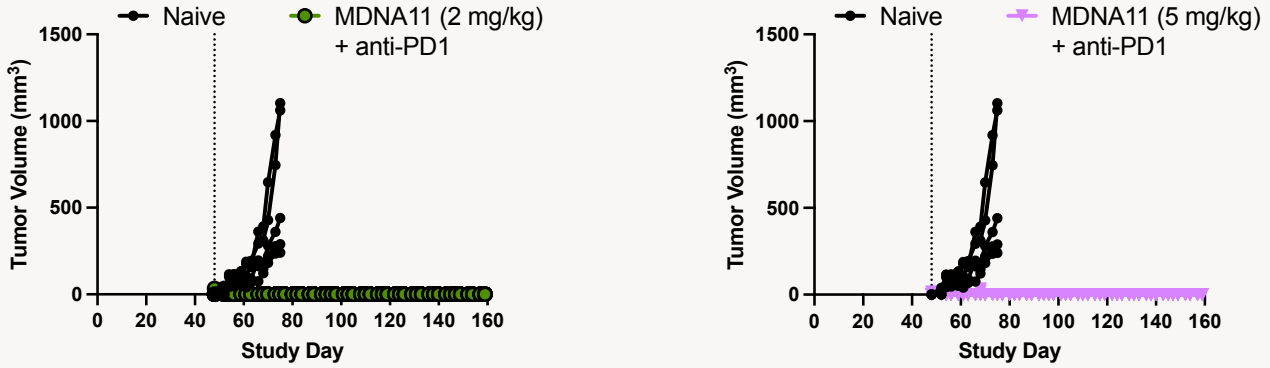
MDNA11 ± Anti-PD-1 - Long Term Memory Response

Inhibits Tumor Growth and Induces Memory Response

MC38 Re-challenges: MDNA11 Monotherapy

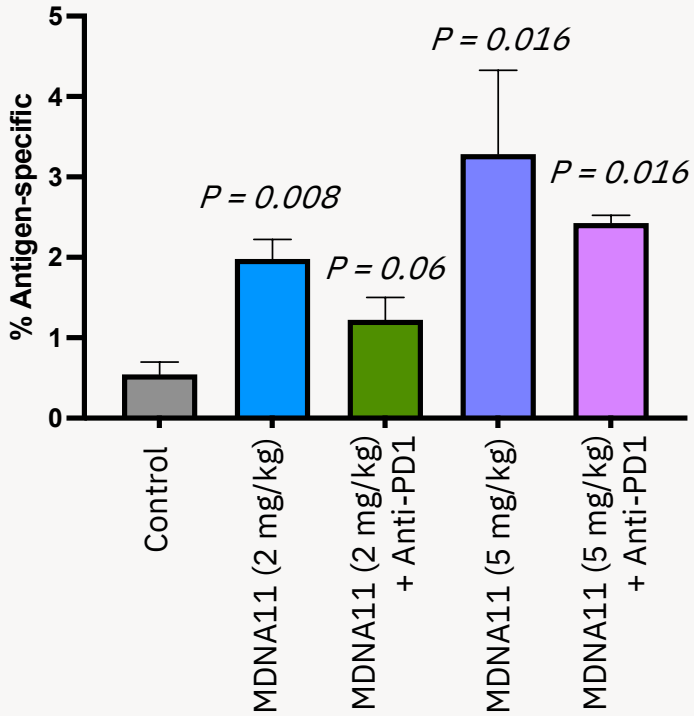


MC38 Re-challenges: MDNA11 + Anti-PD-1



- Avg. tumor size in the treatment group at time of dosing: ~75 mm^3
- MDNA11: IP QWx2 ; Anti-PD1 (RMP1-14; 10 mg/kg): IP BIWx3
- Study in C57Bl/6 mice;

Antigen-specific CD8+ T-cells



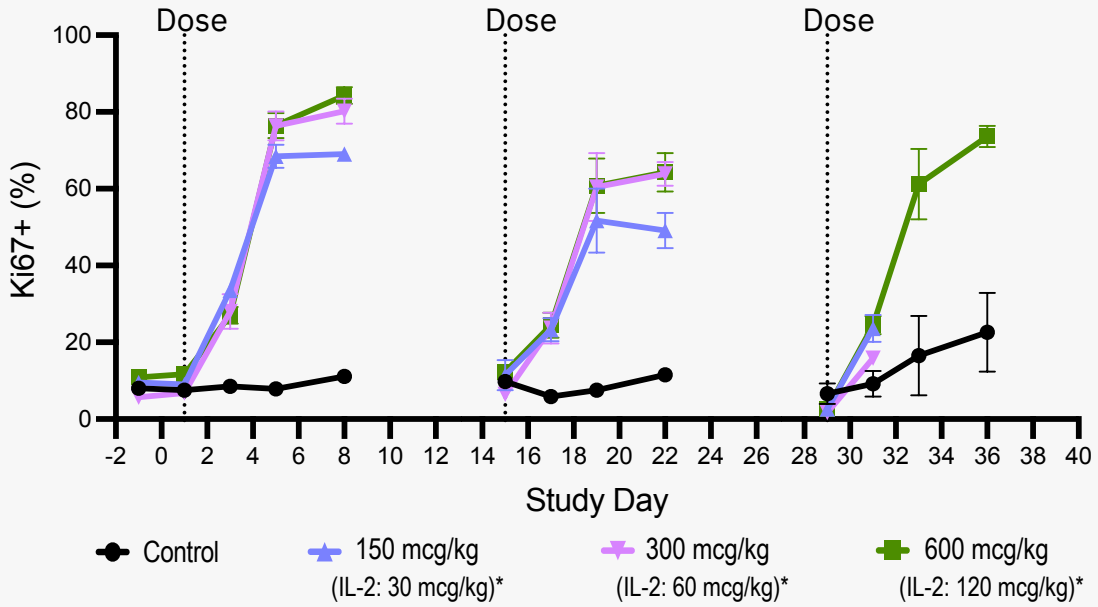
P- value vs Control

- Antigen-specific CD8+ T-cells by flow cytometry using H-2K MuLV p15E tetramer
- All mice boosted with MC38 5 days prior to flow cytometry analysis

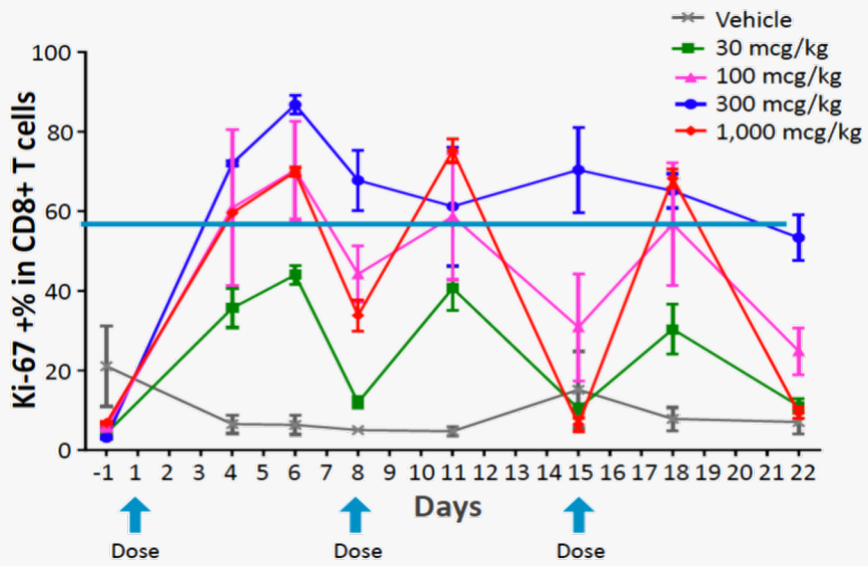
Durable, Dose-Dependent Ki67 Expression in NHP

Ki67 is a key marker of anti-tumor CD8+ T-cell proliferation

MDNA11 – Dosed 14 days apart



THOR-707 – Dosed 7 days apart



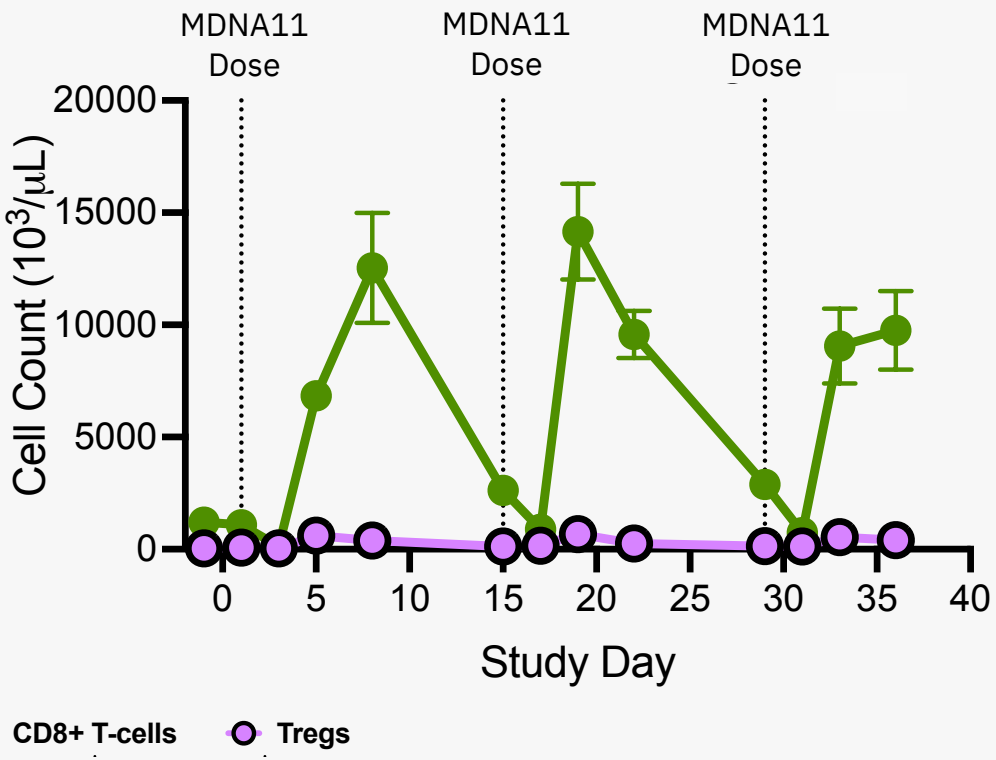
Target Ki67 expression of >50% clearly demonstrated with MDNA11 treatment

* refers to dose based on IL-2 content

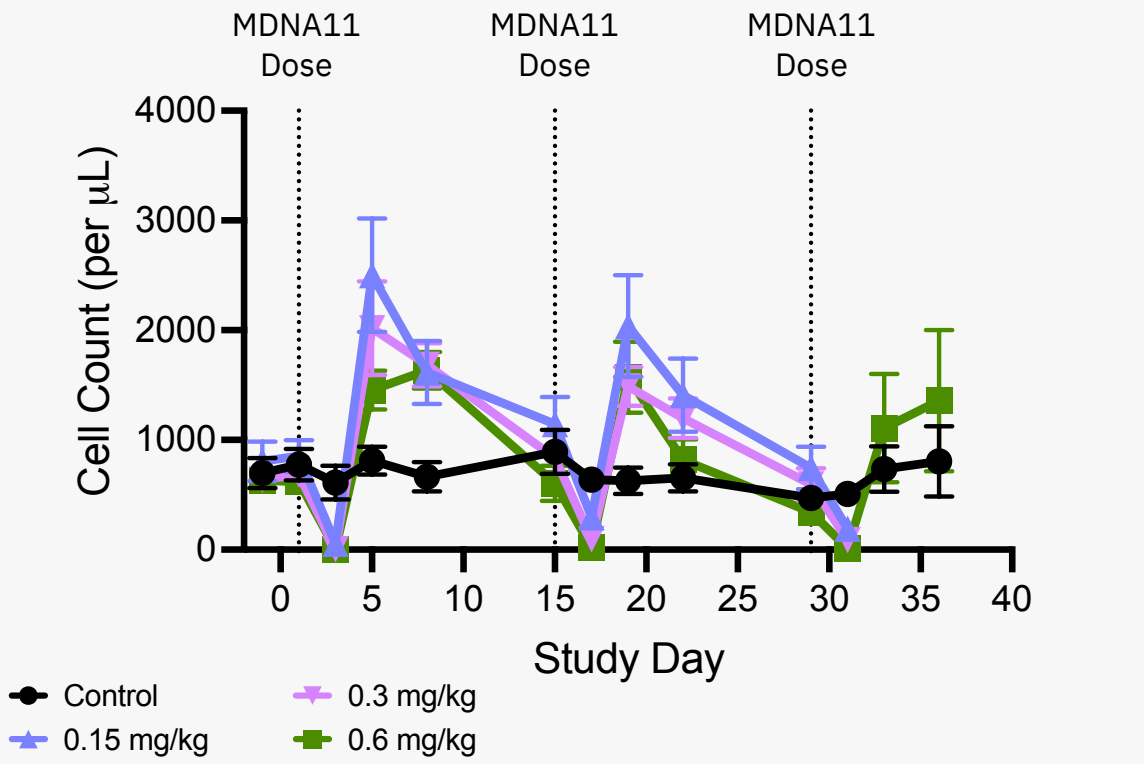
Preferential Expansion of Anti-Cancer Immune Cells Over T_{regs}

Activation of Immunosuppressive T_{regs} Abrogates the Anti-cancer Immune Response

Preferential Expansion of CD8+ T Cells over Tregs at Highest Dose Tested (0.6 mg/kg)



Induction of NK Cell Expansion



Phase 1/2 ABILITY Study Schema

First Patient Dosed in September 2021

US, UK and Canada expansion in 2022

Basket, Accelerated Sequential Dose Escalation and Expansion Study of MDNA11 +/- CPI

MDNA11: Monotherapy Dose Escalation

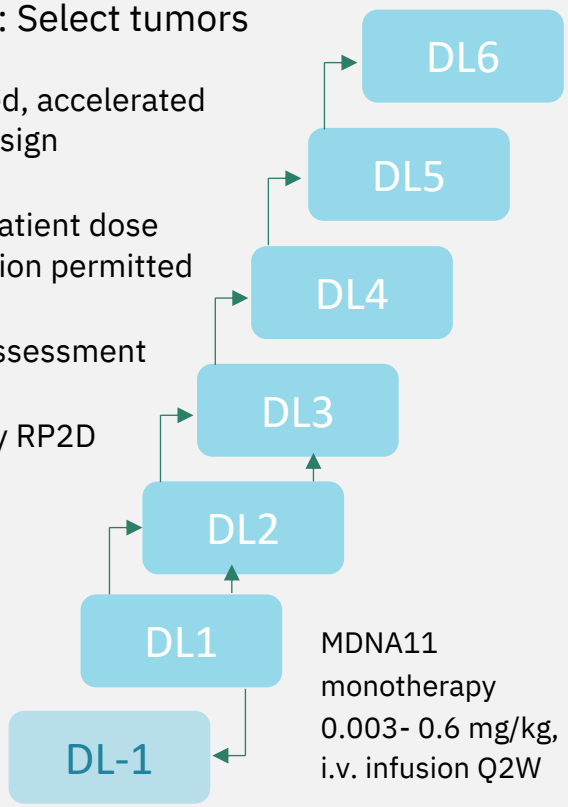
N~ 20: Select tumors

Modified, accelerated 3+3 Design

Intra-patient dose escalation permitted

DLTs assessment

Identify RP2D



MDNA11 monotherapy 0.003- 0.6 mg/kg, i.v. infusion Q2W

MDNA11 Monotherapy Dose Expansion

N~ 30: Melanoma, renal cell carcinoma and other select tumors (1:1:1)

MDNA11 administered alone at RP2D via i.v. infusion Q2W or Q3W

Signals of anti-tumor activity

MDNA11 + Checkpoint Inhibitor (CPI) Dose Expansion

N~ 30: Melanoma, renal cell carcinoma and other select tumors (1:1:1)

Safety run-in

MDNA11 administered at RP2D in combination with CPI via i.v. infusion Q3W (planned)

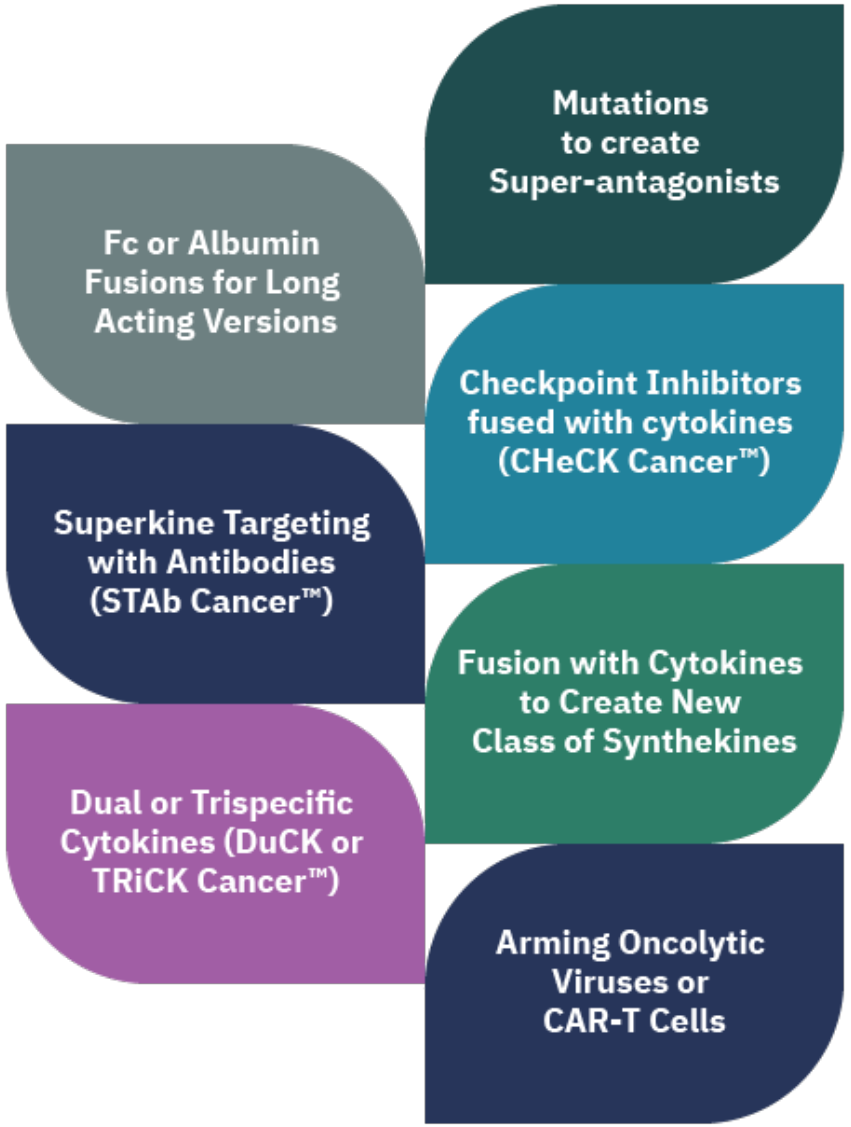
Signals of anti-tumor activity

IL-2 Superkine Program

Next Steps

MDNA11 Recent & Upcoming Milestones

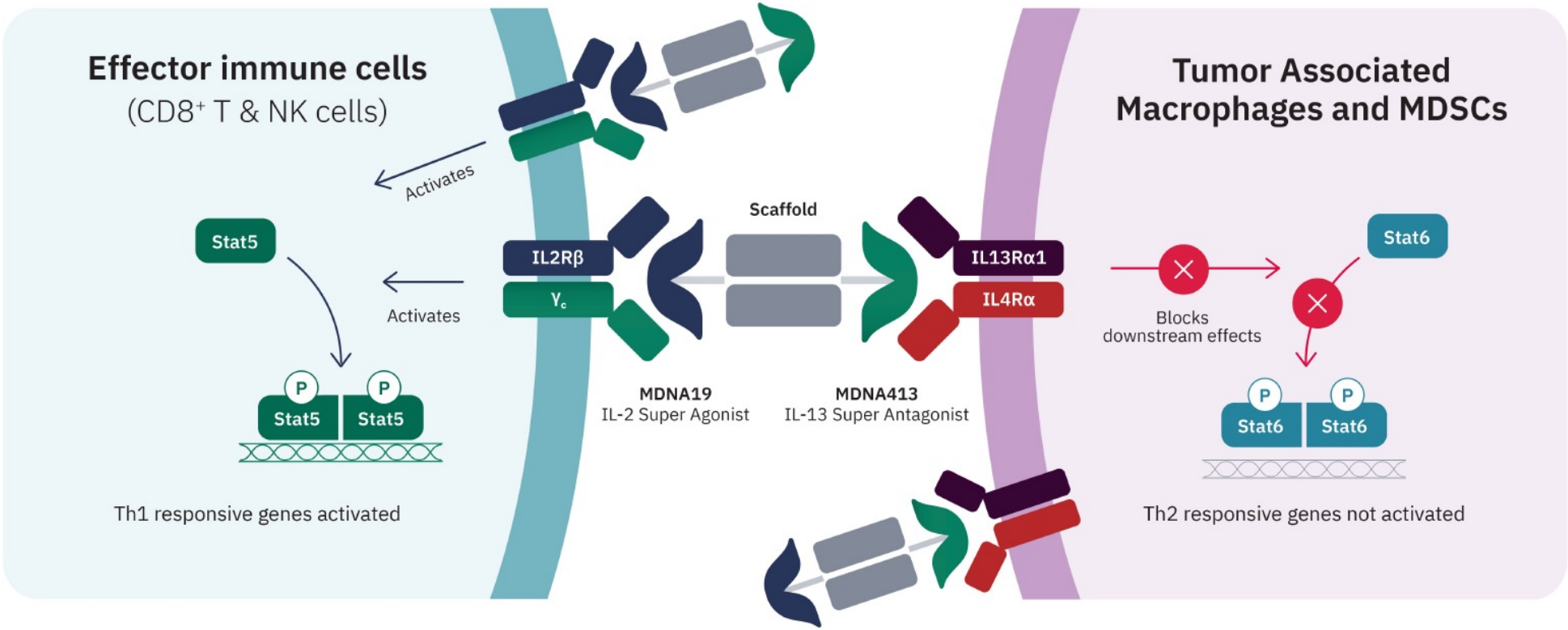
- Initiated Phase 1/2 clinical trial **(September 2021)**
- Safety, PK/PD and biomarker data update from Phase 1/2 monotherapy study **(End 2021)**
- Phase 1/2 efficacy data update **(Mid 2022)**



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

Dual Cytokines: Mechanism of Action

Targeting immunologically “cold” tumors by modulation of the Tumor Microenvironment (TME)



Increased activation of CD8⁺ T & NK Cells to attack the tumor

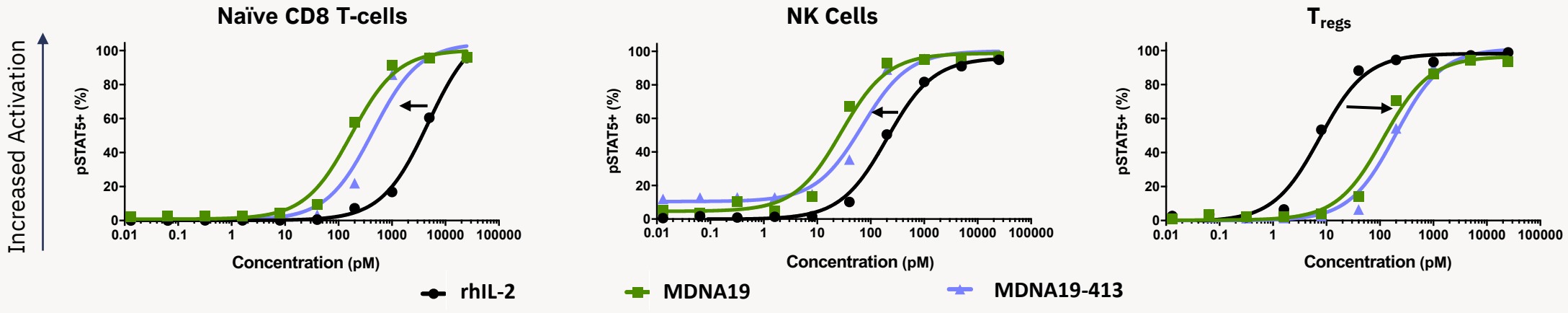
Lowered Immune Suppressing effects in the TME

- Blocking pSTAT6 signaling
- Suppressing M2 polarization
- Preventing MDSCs suppression of T cells

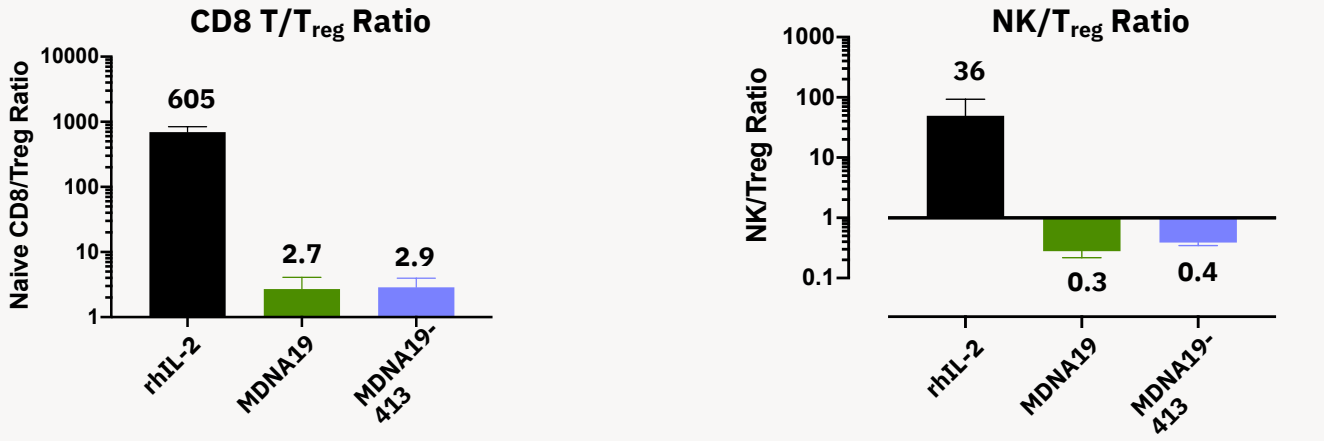
MDNA19-413: IL-2 Agonism Maintained

Enhanced Signaling in Anti-tumor CD8+ T and NK cells; Diminished Signaling in Pro-tumor T_{regs}

Immune Cell Selectivity Retained



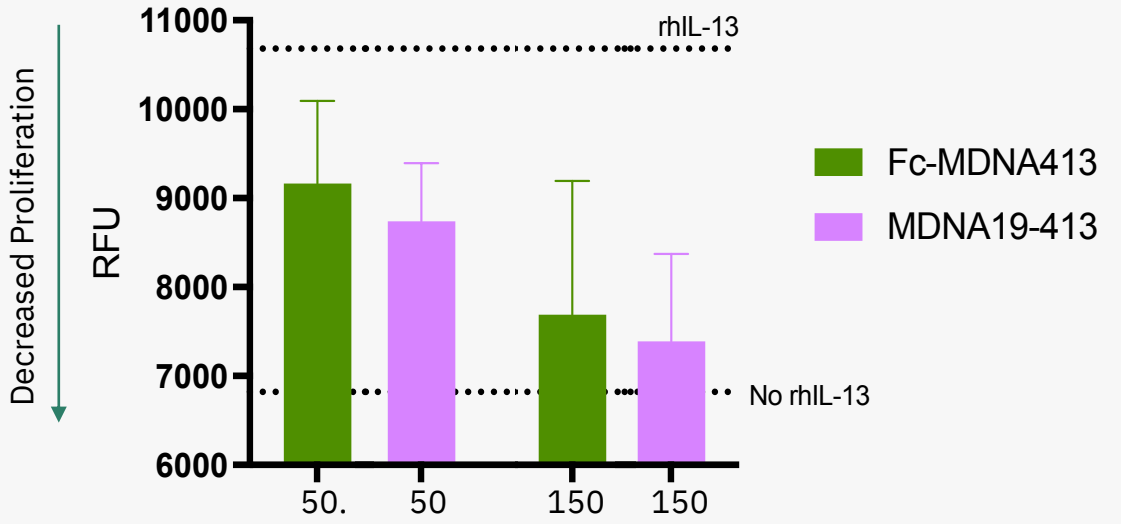
Preferential Stimulation of CD8+ T and NK Cells



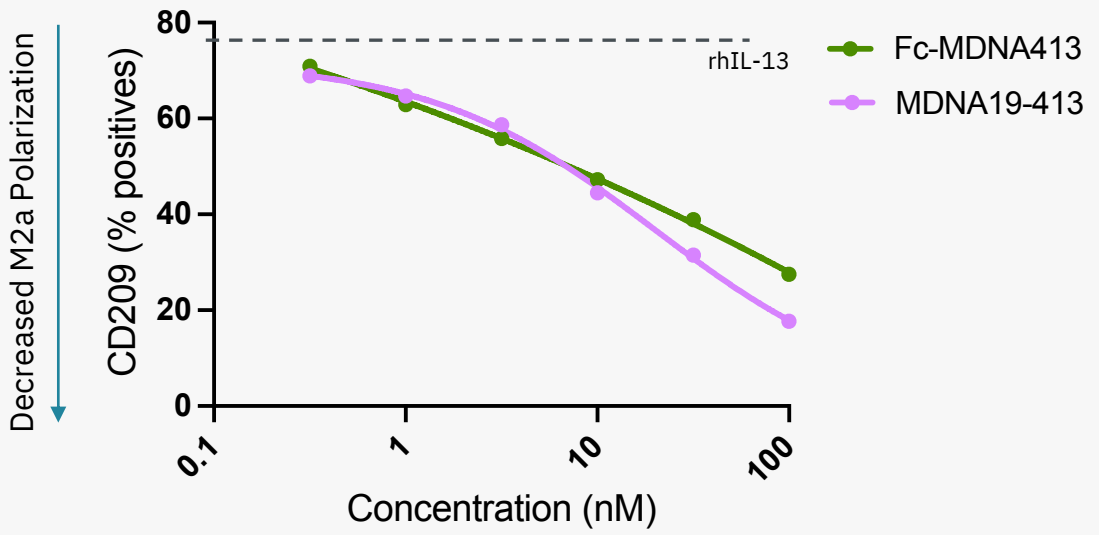
MDNA19-413: IL-4/IL-13 Antagonism Maintained

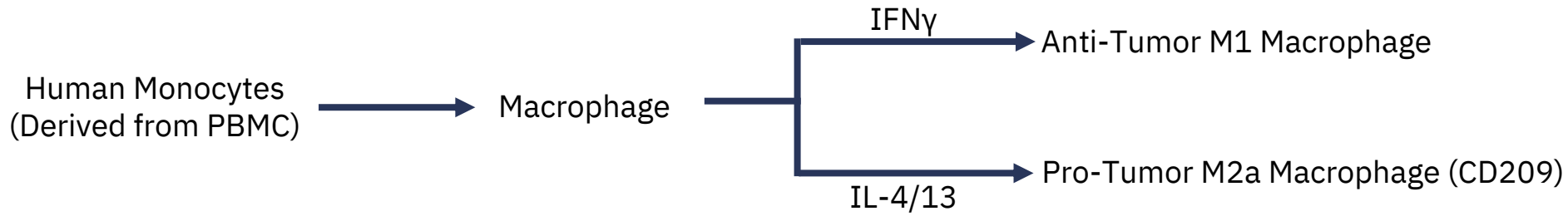
Inhibits Pro-tumor IL-4/IL-13 Induced Proliferation and M2a Polarization

Suppression of TF-1 Cell Proliferation
(TF-1 proliferation is highly dependent on IL-4/IL-13 signaling)



Prevention of M2a Polarization
(M2a macrophages promote tumor growth)

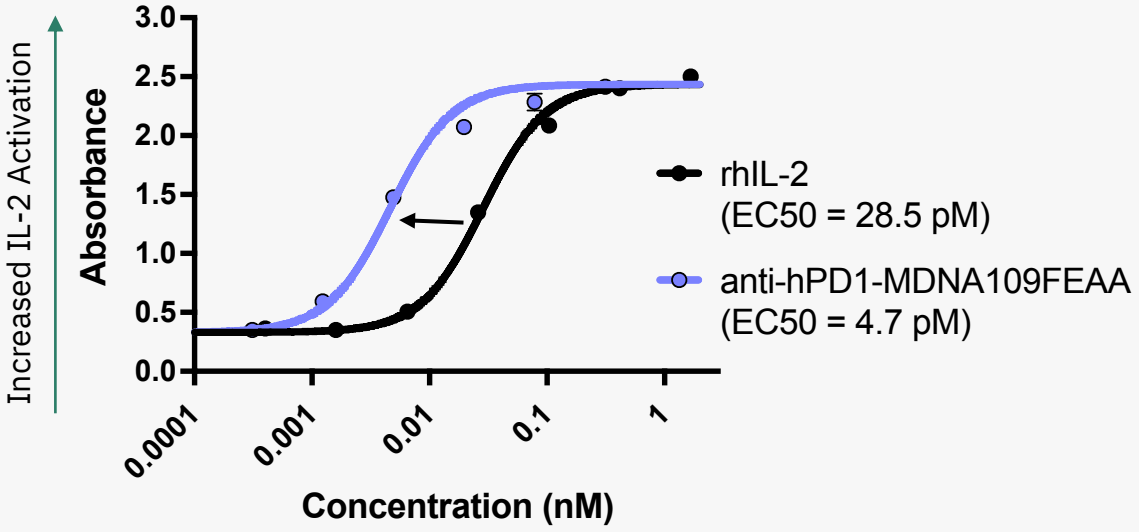




Checkpoint Inhibitors Fused to Cytokines

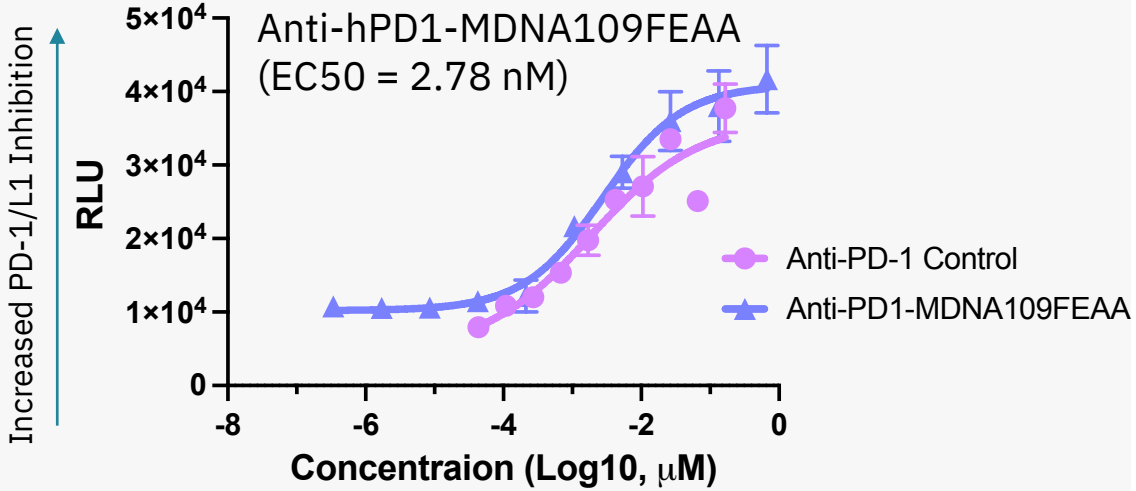
T-cell activation + Suppression of Exhaustion = Superior Therapeutic Efficacy

IL-2 Reporter Assay



HEK Blue IL-2 assay
Measures P-STAT5 signaling

Human PD-1/PD-L1 Blockade Bioassay (Promega)



PD1 effector cells co-cultured with PD-L1 aAPC/CHO-K1 cells
Blockade of PD-1/PD-L1 interaction induces NFAT-luciferase reporter

Anti-PD1-MDNA109FEAA = Anti-PD1 fused with IL-2 Agonist

MDNA55

Empowered IL-4
Superkine Targeting
Glioblastoma

Current Treatment Strategies for GBM are Ineffective

85-90% of patients

55% of GBM Temodar-Resistant*

RELAPSE

Even after going through the standard treatment regimen, **all tumors recur** invariably

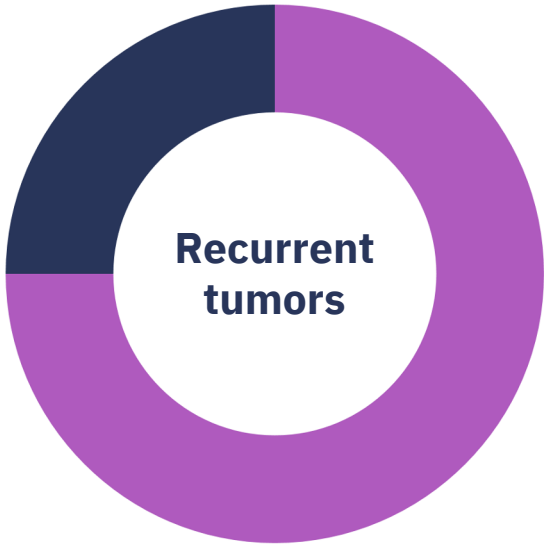
- Not all patients are eligible for surgery and chemotherapy regimen in the first place, while the vast majority of recurrent tumors are inoperable
- Survival for patients undergoing the standard therapy is extremely low: The two-year survival rate is 5-10%

25%

OPERABLE rGBM

75%

INOPERABLE rGBM but **can be treated by MDNA55** via a direct infusion into tumor



* Expression of the DNA repair protein O6-methylguanine-DNA methyltransferase (MGMT) is responsible for resistance to Temodar

MDNA55: A Targeted Immunotherapy for GBM

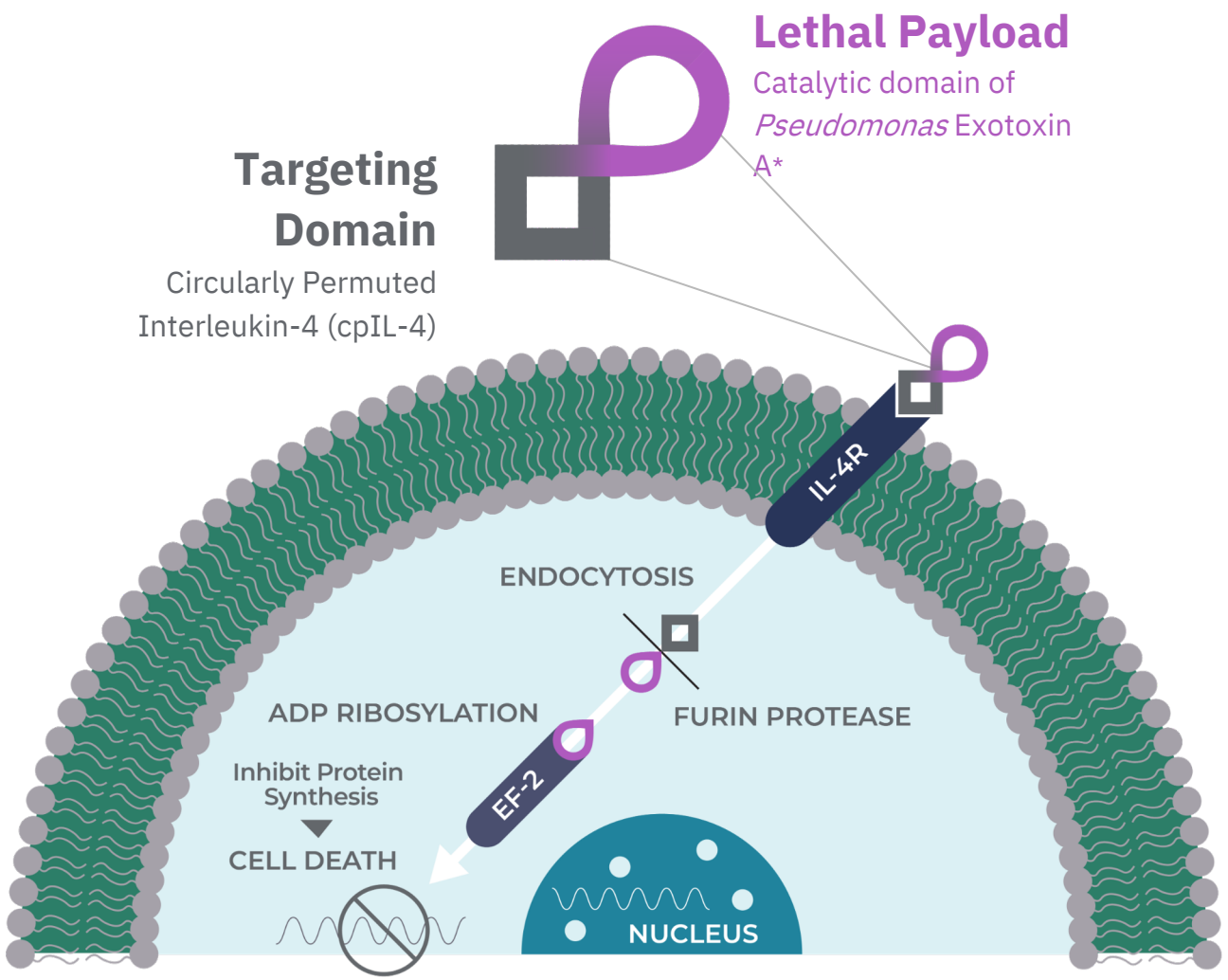
By Passes BBB
Single intra-tumoral infusion avoids systemic toxicity and achieves tumor control

Targets IL4R
Receptor is expressed in brain tumors and in the tumor microenvironment (TME), but not in healthy brain cells

Highly Selective
Avoids off-target toxicity

Disrupts the TME
Targets IL4R positive MDSCs in GBM unblinds the immunosuppressive TME

Causes Immunogenic Cell Death
Anti-tumor immunity is initiated and remains active after MDNA55 is cleared



*FDA approved Moxetumomab pasudotox

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

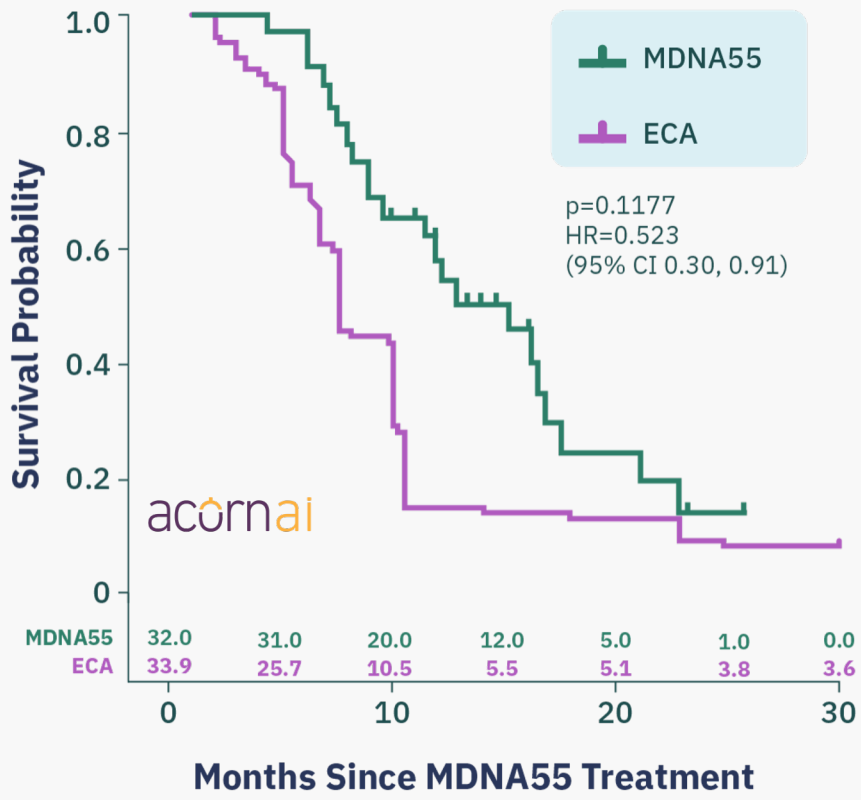
Results*

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

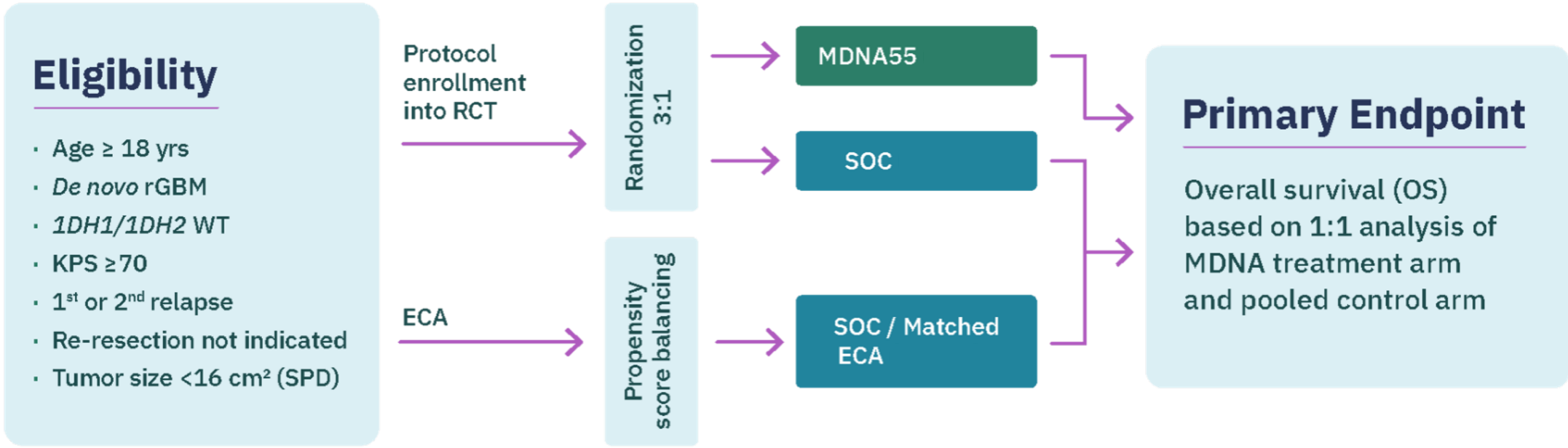
IL4R High + IL4R Low$^{\text{High Dose}}$



*Survival was calculated from time of relapse

Planned MDNA55 Phase 3 Trial – Hybrid Design With ECA

Hybrid Design Trial with an External Control Arm



Study Assumptions

- 90% power
- HR of MDNA55 vs. pooled control = 0.65
- 2-sided alpha = 0.05
- Effect size = 4.6 months in mOS time
- Drop-out rate = ~5%

Financial Overview

Evolutionary Cytokines, Revolutionary Medicines

Medicenna is a clinical stage immunotherapy company that uses directed evolution to generate engineered interleukins called Superkines that can amplify, blunt or fine tune the immune system in order to combat the most challenging diseases and provide hope to patients with unmet needs

Nasdaq	**MDNA**
TSX	**MDNA**
Headquarters	**Toronto, CA**
Cash	**CDN $26.7 million ****
Debt	**$0**
Preferred Shares	**0**
Issued and Outstanding	**53,979,576***
Fully Diluted	**62,387,419***

*As of November 12, 2021
**As of September 30, 2021

Near-Term Value Inflection Milestones

	H2 2021	H1 2022
MDNA11	**Dosed 1st Patient in Phase 1/2 Clinical Study** ✓ **Safety, PK/PD and Biomarker Data Update**	**Initial Efficacy Data Update**
BiSKIT Platform	**Identify new lead candidate**	**Initiate IND Enabling Studies**

Thank you

Fahar Merchant, PhD
President and CEO

Elizabeth Williams
Chief Financial Officer

MEDICENNA