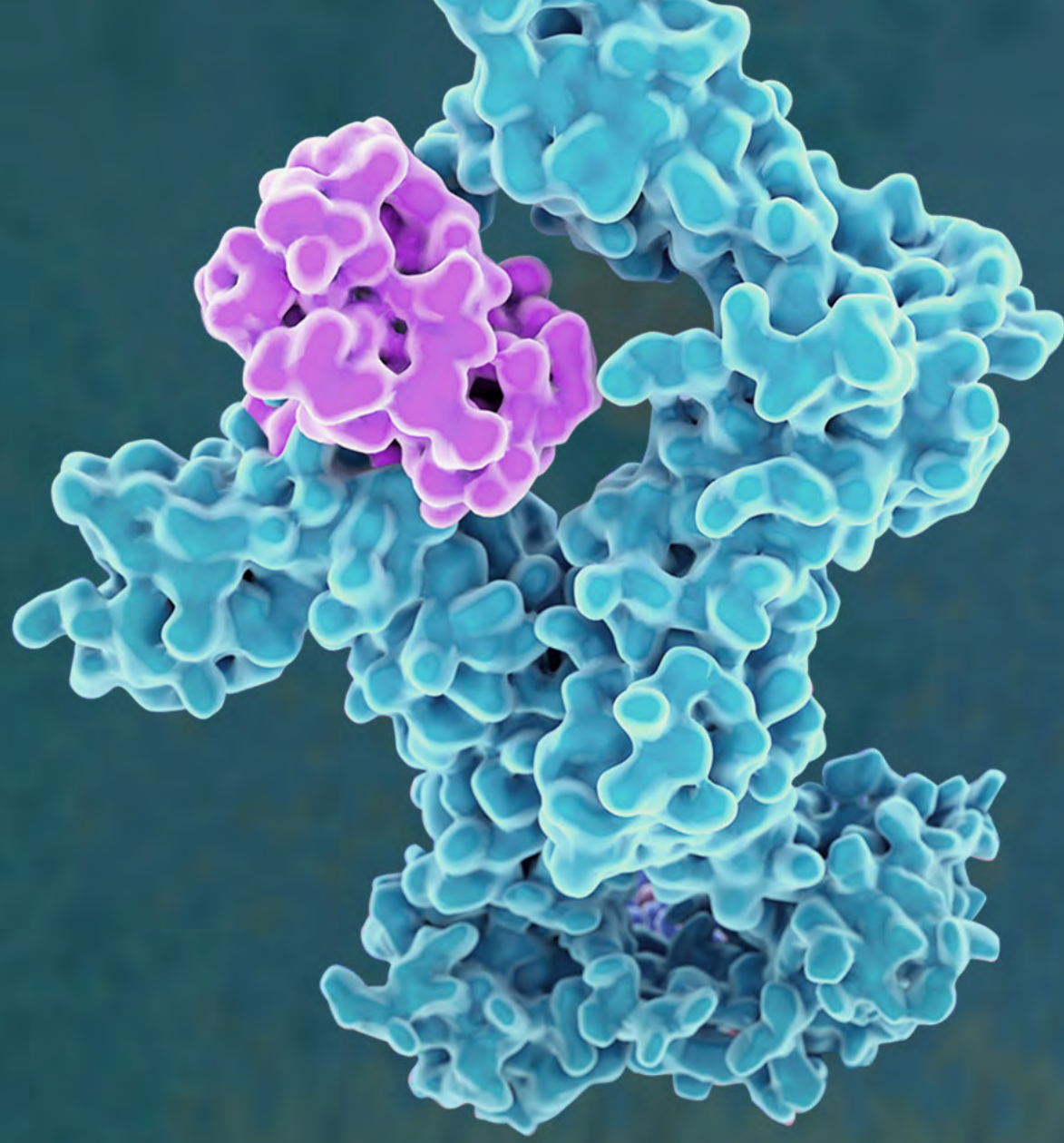
Q3, 2021

Evolutionary Cytokines Revolutionary Medicines



MEDICENNA

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Company Overview

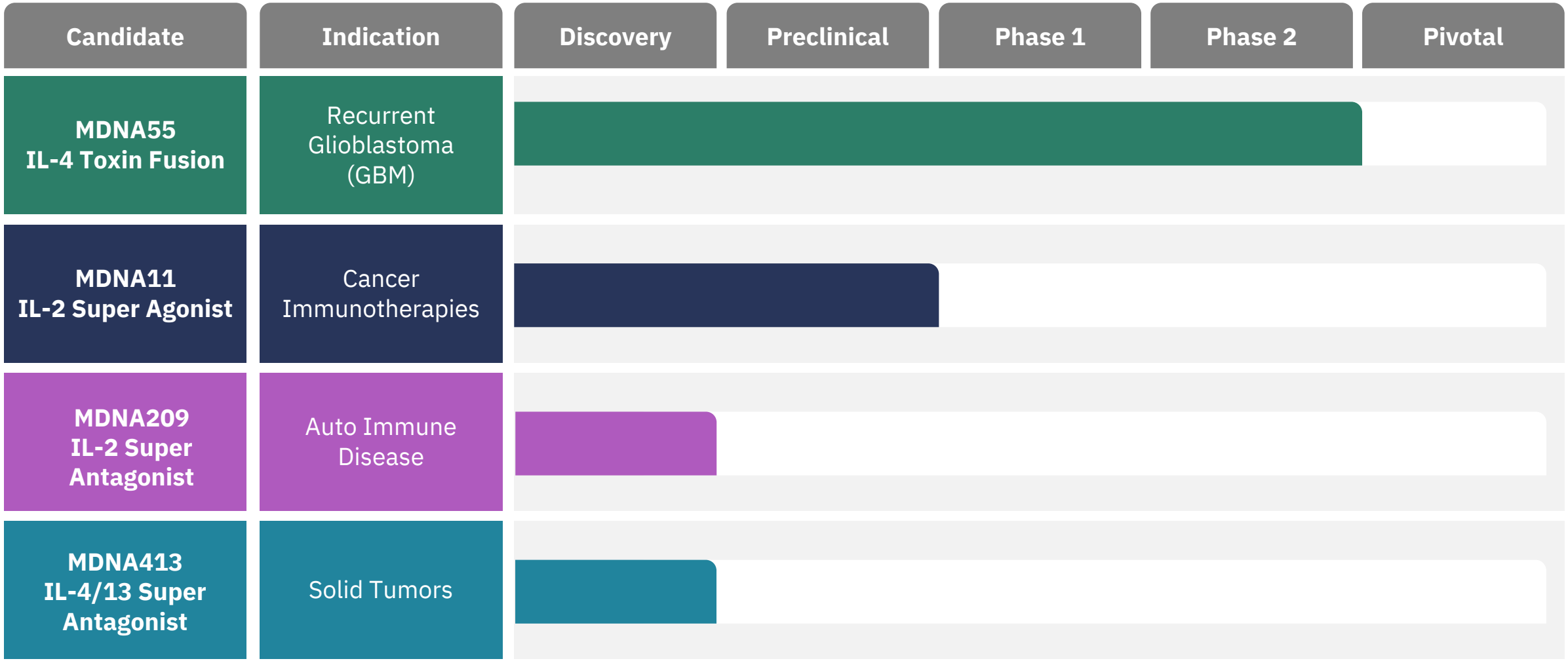
Evolutionary Cytokines, Revolutionary Medicines

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

Nasdaq	**MDNA**
TSX	**MDNA**
Headquarters	**Toronto, CA**
Cash	**CDN $35.9 million ****
Debt	**$0**
Preferred Shares	**0**
Issued and Outstanding	**53,742,255***
Fully Diluted	**62,011,316***

*As of August 12, 2021
**As of June 30, 2021

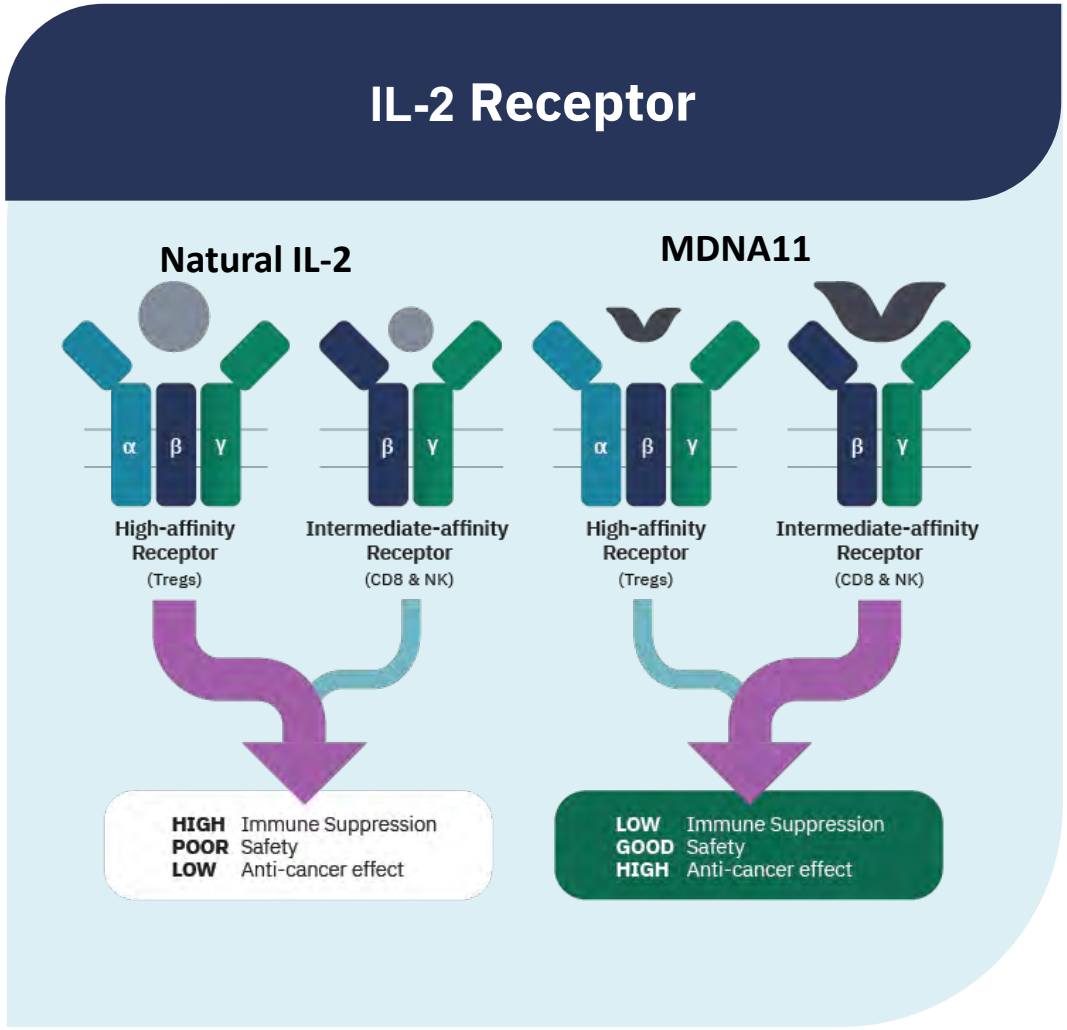
Expanding Pipeline Anchored by MDNA55 and MDNA11



MDNA11

IL-2 Super Agonist for Cancer Immunotherapy

Targeting IL-2 Receptor Subunits in Cancer Therapy



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

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

Stimulation of CD122

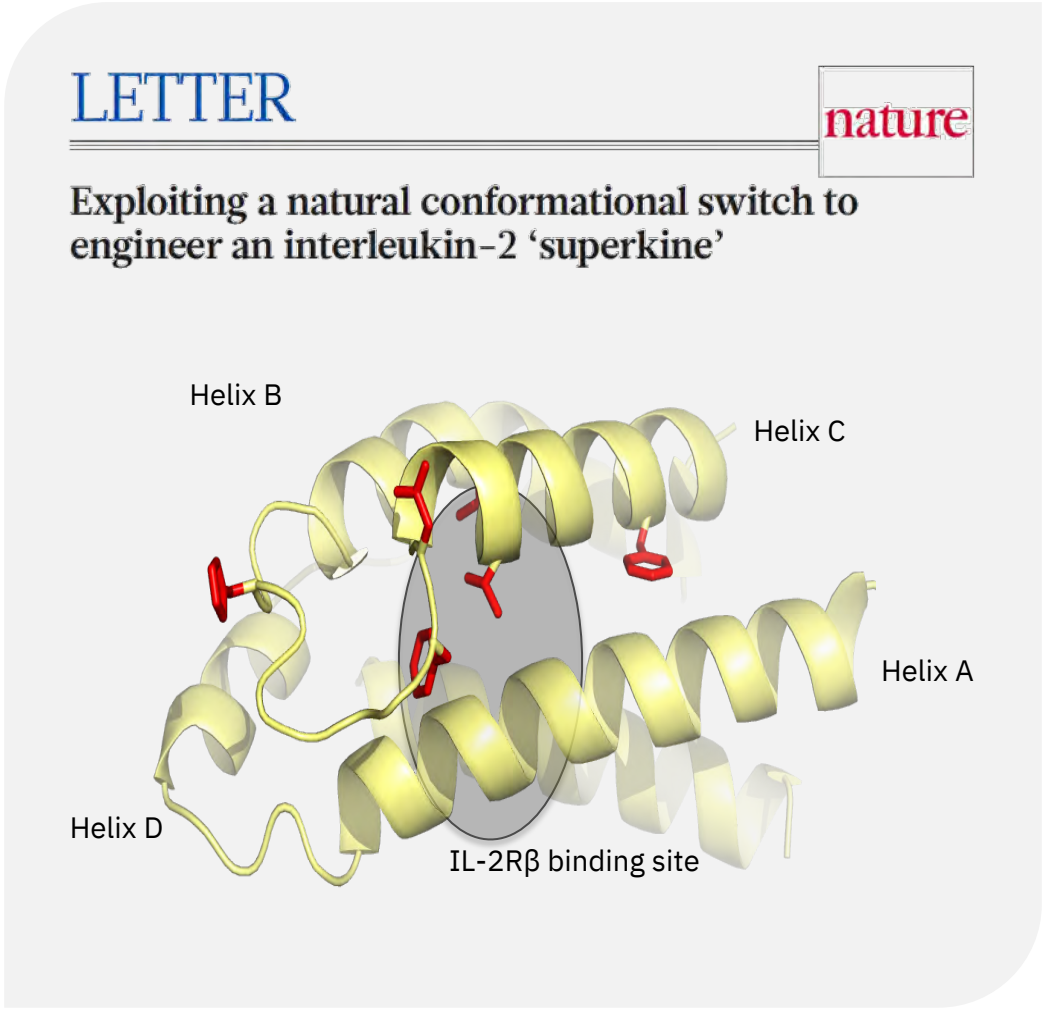
- Key for the activation of cancer killing immune cells such as CD8+ T cells, naïve T cells, and NK cells.

Stimulation of CD25

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

Proleukin (recombinant human [rh] IL-2), which selectively stimulates CD25, is approved for the treatment of metastatic melanoma and renal cell carcinoma

Superkines: First-Generation IL-2 Variants

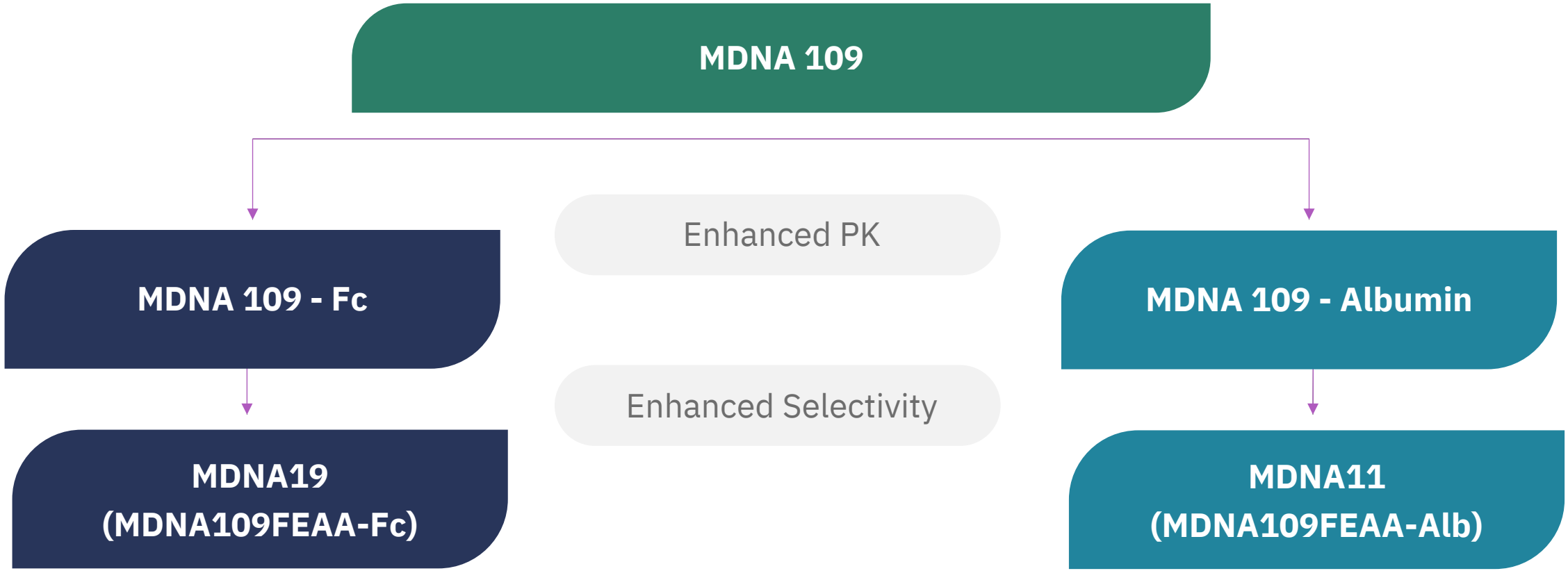


Levin, Bates, and Ring et. al, Nature, 2012

Medicenna's MDNA109 platform produced first generation IL-2 variants with 200-fold higher affinity for CD122 (IL-2Rβ), which is key for the activation of immune cells responsible for cancer killing (CD8+ T cells, naïve T cells, NK cells), yet similar affinity to CD25

	Similar affinity to CD25	200X increased affinity to CD122
SPR data (nM)	CD25	CD122
IL-2	6.6	280
MDNA109	**6.6**	**1.4**

MDNA11: Next-Generation IL-2 Superkine

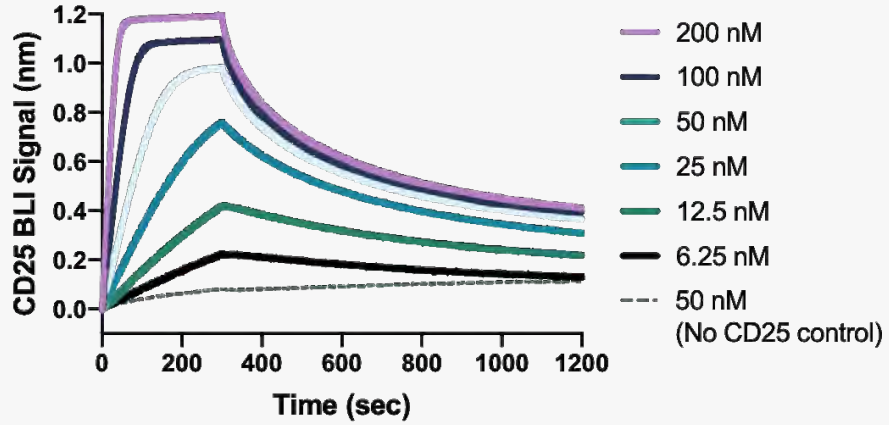


MDNA11 is a next-generation IL-2 superkine with superior CD122 binding without CD25 affinity, thereby preferentially stimulating cancer killing effector T cells and NK cells when compared to competing IL-2 programs.

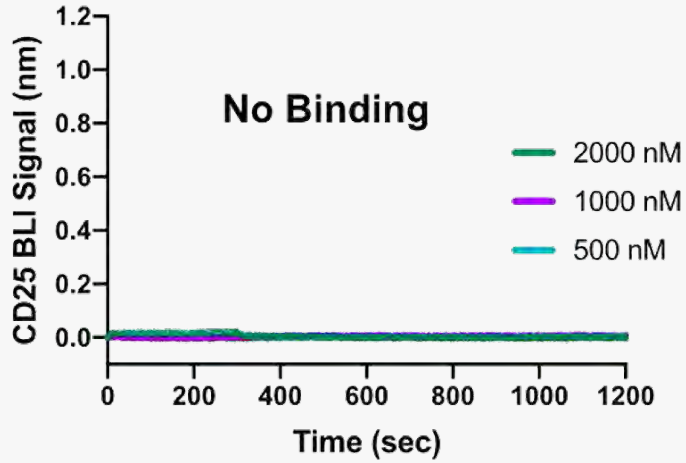
MDNA11

No CD25 Binding and Enhanced Affinity and Selectivity for CD122 Compared to rhIL-2

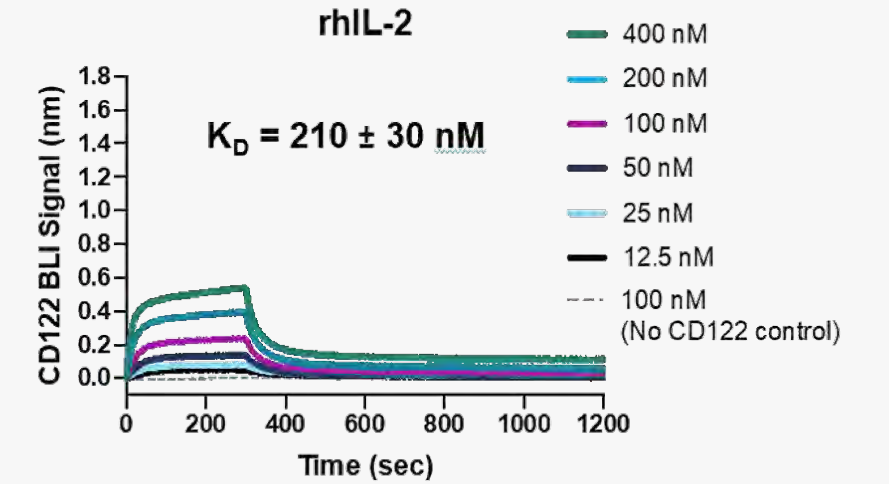
rhIL-2 – CD25 Binding



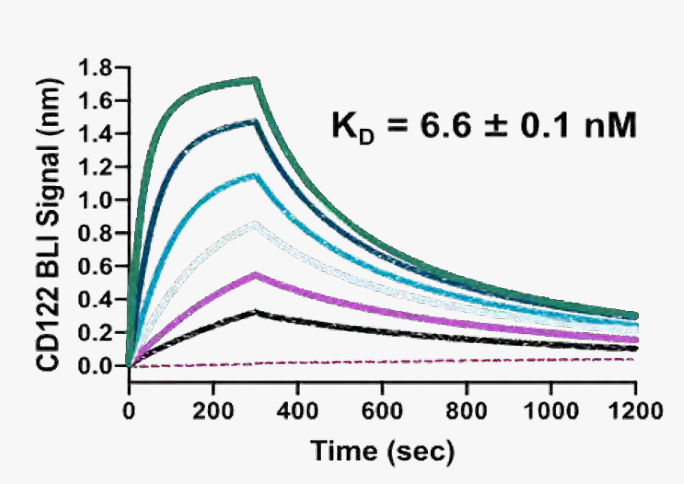
MDNA11 – CD25 Binding



rhIL-2 – CD122 Binding



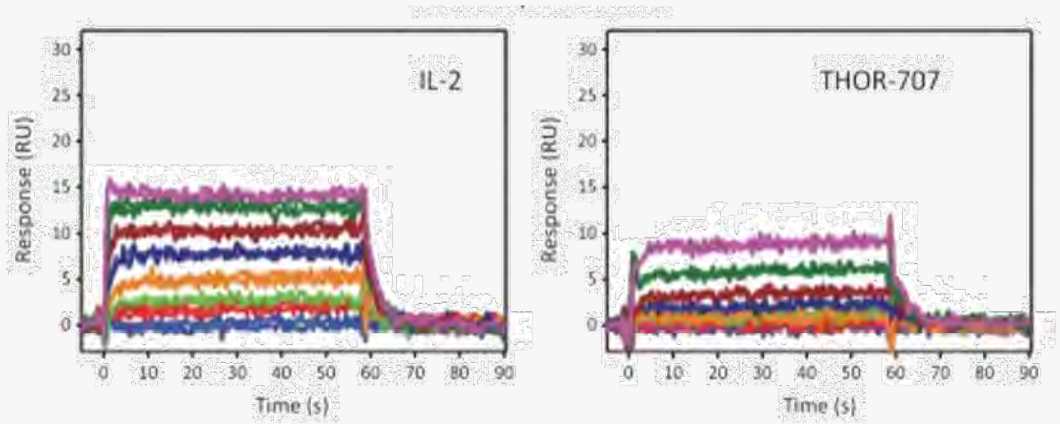
MDNA11 – CD122 Binding



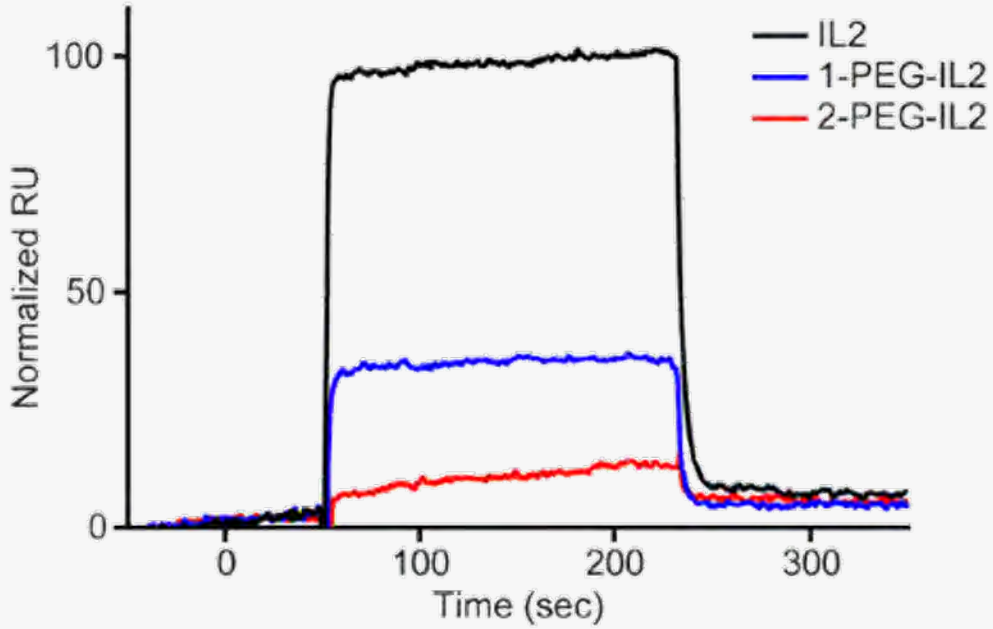
Competing IL-2 Variants are Weak CD122 Binders

THOR-707: Reduced Binding to IL2Rβ (CD122)

IL2Rβ (CD122)



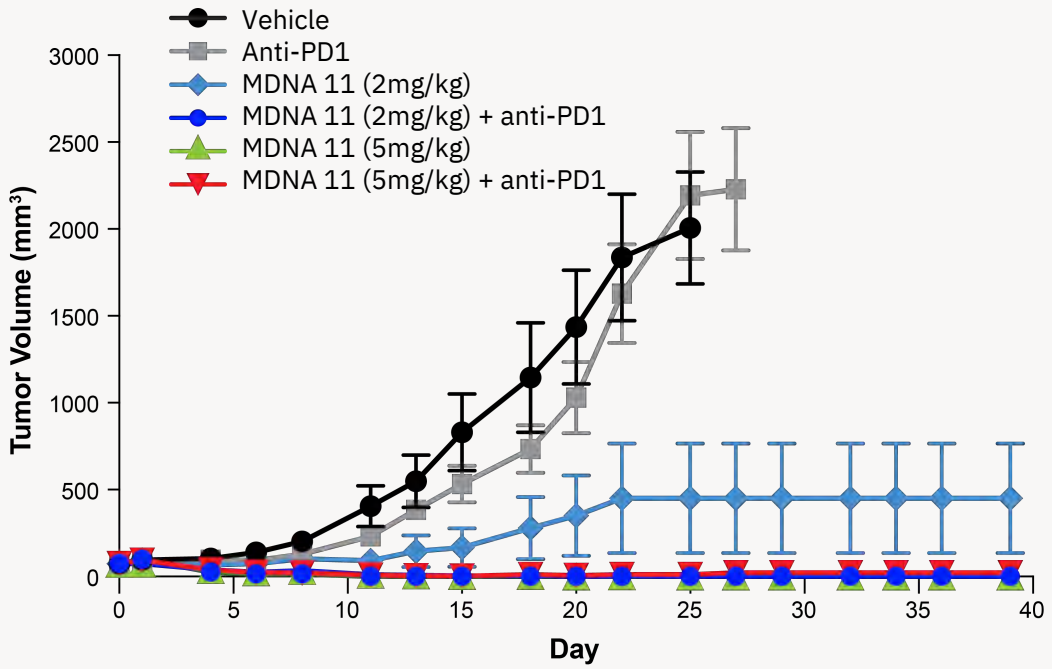
1-PEG-IL2 (Most Active Form of NKTR-214) is a Weak IL2Rβ (CD122) Binder



Strong Monotherapy and Anti-PD1 Combo Effect

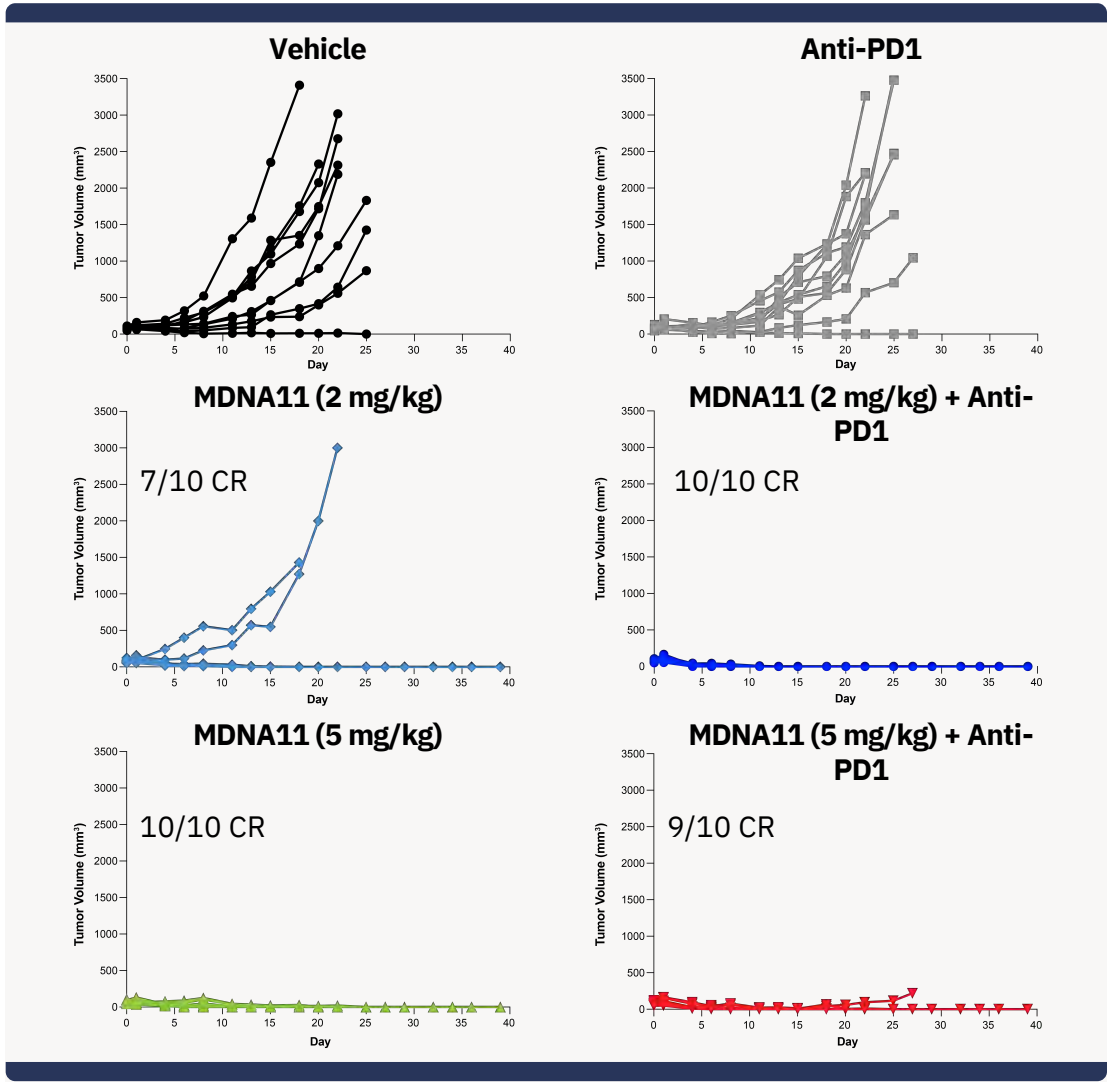
Anti-Tumor Efficacy & Combination Effect with Anti-PD1 in MC38 Tumor Model

MC38 Tumor Model



MDNA11: IP Q7Dx2
Anti-PD1 (RMP1-14; 10 mg/kg): IP BIWx3
Average size at initiation of dosing ~ 75 mm^3
Study in C57Bl/6 mice.

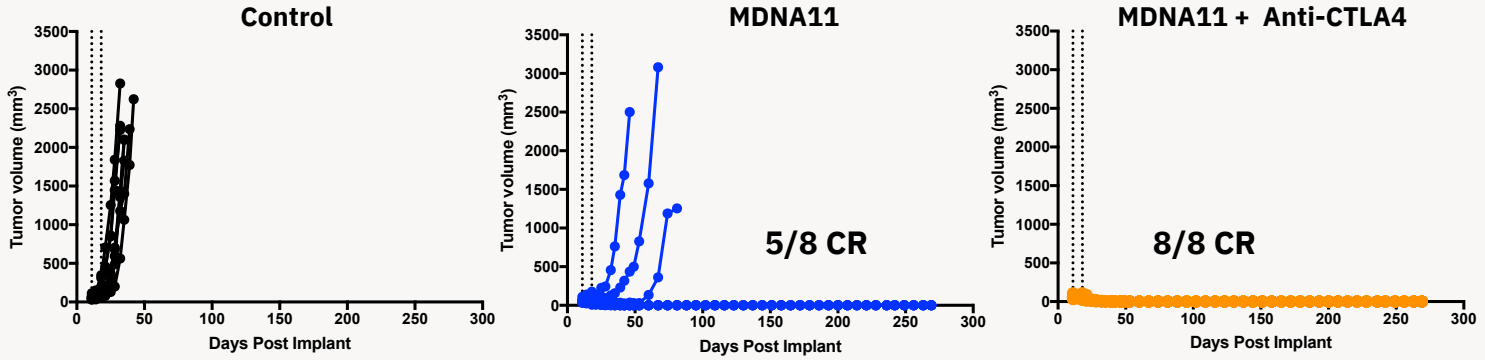
CR: Tumor volume = 0
Re-challenge study on-going



MDNA11 + αCTLA4

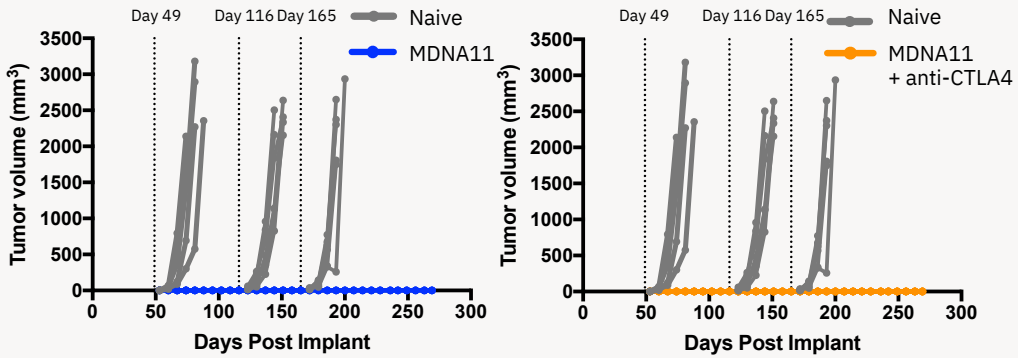
Inhibits Tumor Growth and Induces Memory Response

Primary Tumors (CT26 in Balb/c Mice)



Re-challenges

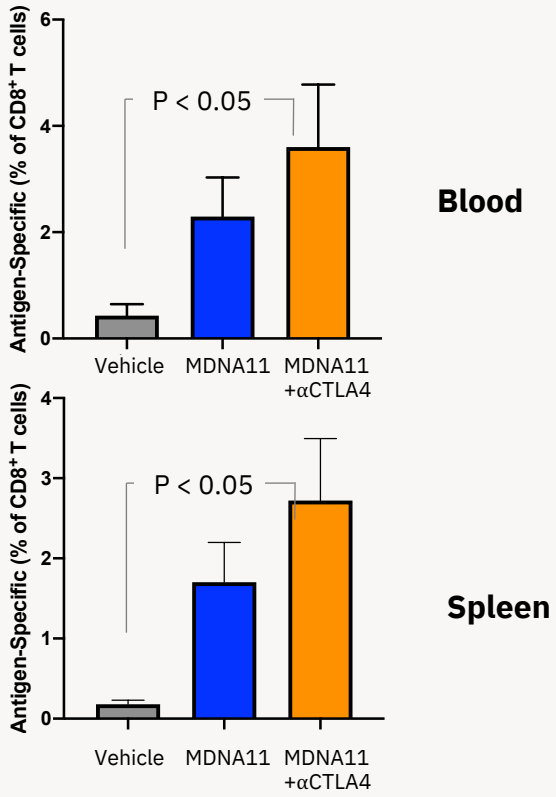
Mice re-challenged with CT26 tumor cells at different sites on their flanks



- Avg. tumor size in the treatment group at time of dosing: ~60 mm^3
- MDNA11 (5 mg/kg, IP, Q.W x 2wks); Anti-CTLA4 (9D9; 200 µg, IP, Q2W x 2wks)

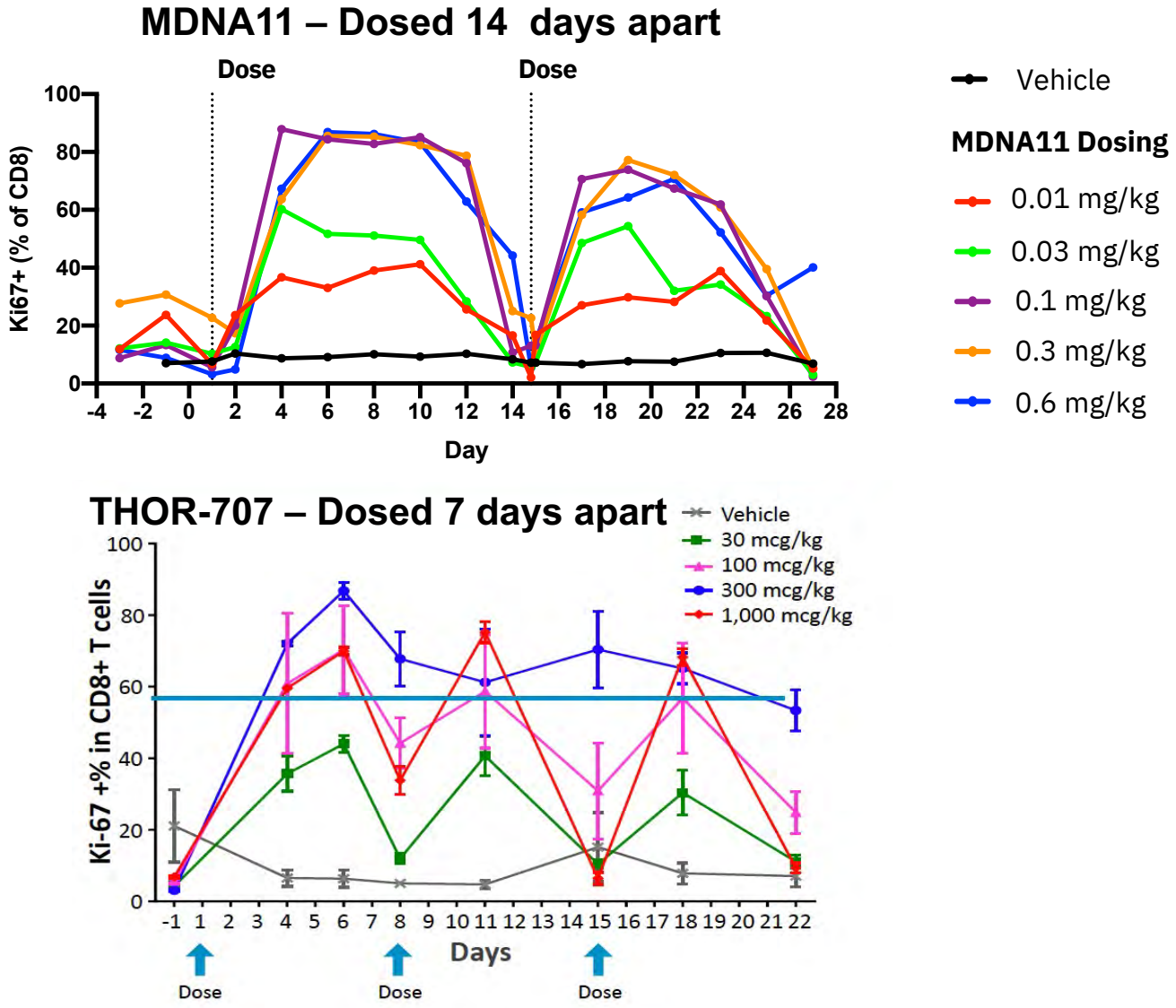
Antigen-specific CD8 T-cells on Day 270

(MDNA11 treatment on Day 11 & 18)



- Antigen-specific CD8T cells detected by anti-CD8 (KT15) and H-2Ld MuLV gp70 Tetramer
- All mice boosted with CT26 cells 5 days prior to analysis

Durable, Dose-Dependent Ki67 Expression



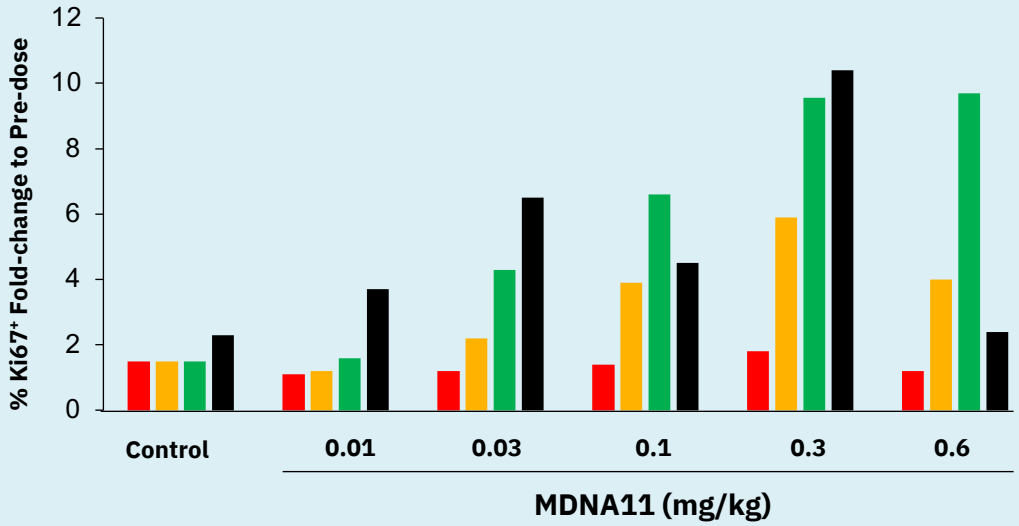
Ki67 is a key marker of anti-tumor CD8+ T-cell proliferation

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

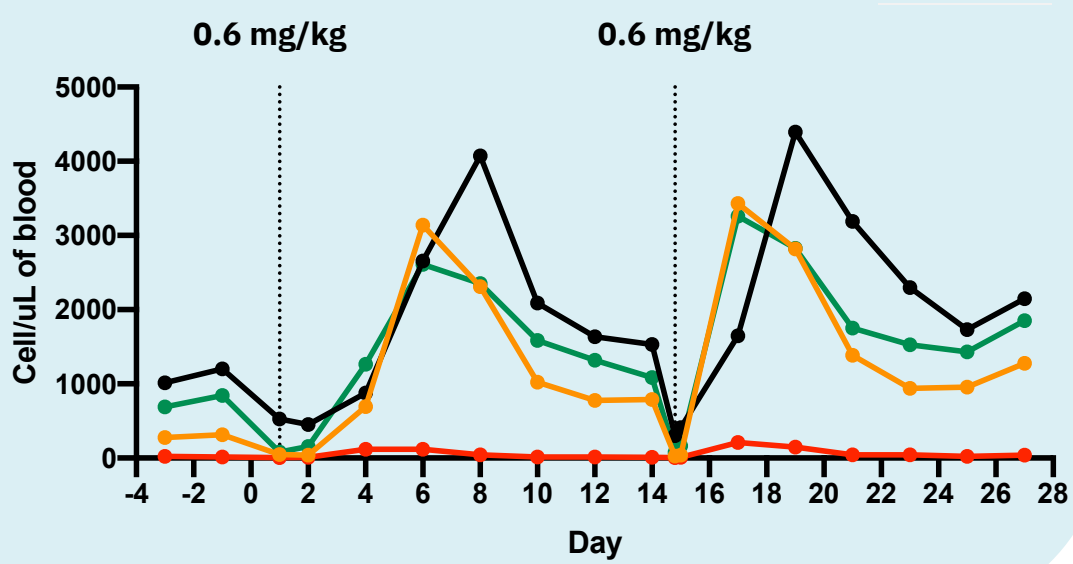
Non-Human Primates – Increased Immune Cells but Not Tregs

MDNA11 induced up to 10-fold expansion in cancer-fighting immune cells (CD4+ T, CD8+ T, and NK Cells) in non-human primate study without: (a) Treg expansion, (b) generating anti-drug antibodies, (c) causing hypotension associated with vascular leak syndrome, (d) cytokine storms, or (e) other undesirable immune mediated side effects.

Percent Ki67⁺ Cell Fold Change



Immune Cell Counts



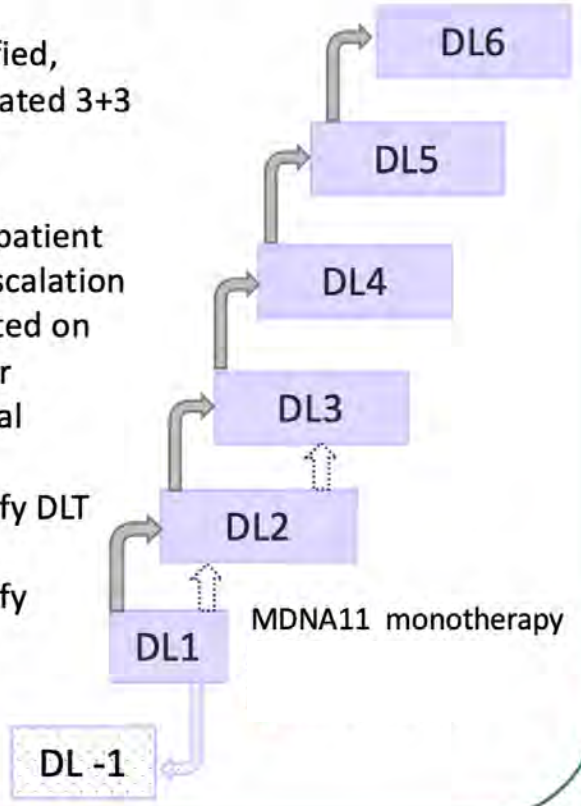
Tregs CD4⁺ T Cell CD8⁺ T Cell NK Cell

MDNA11 Phase 1/2 Trial Design

MDNA11
Monotherapy Dose Escalation

N~ 20: Select tumors

- Modified, accelerated 3+3 Design
- Intra-patient dose escalation permitted on sponsor approval
- Identify DLT
- Identify RP2D



MDNA11
Monotherapy Dose Expansion

N~ 30: Melanoma, RCC and other select tumors (1:1:1)

MDNA11 administered alone at RP2D via i.v infusion Q2W

Signals of anti-tumor activity

MDNA11 + CPI
Dose Expansion

N~ 30: Melanoma, RCC and other select tumors (1:1:1)

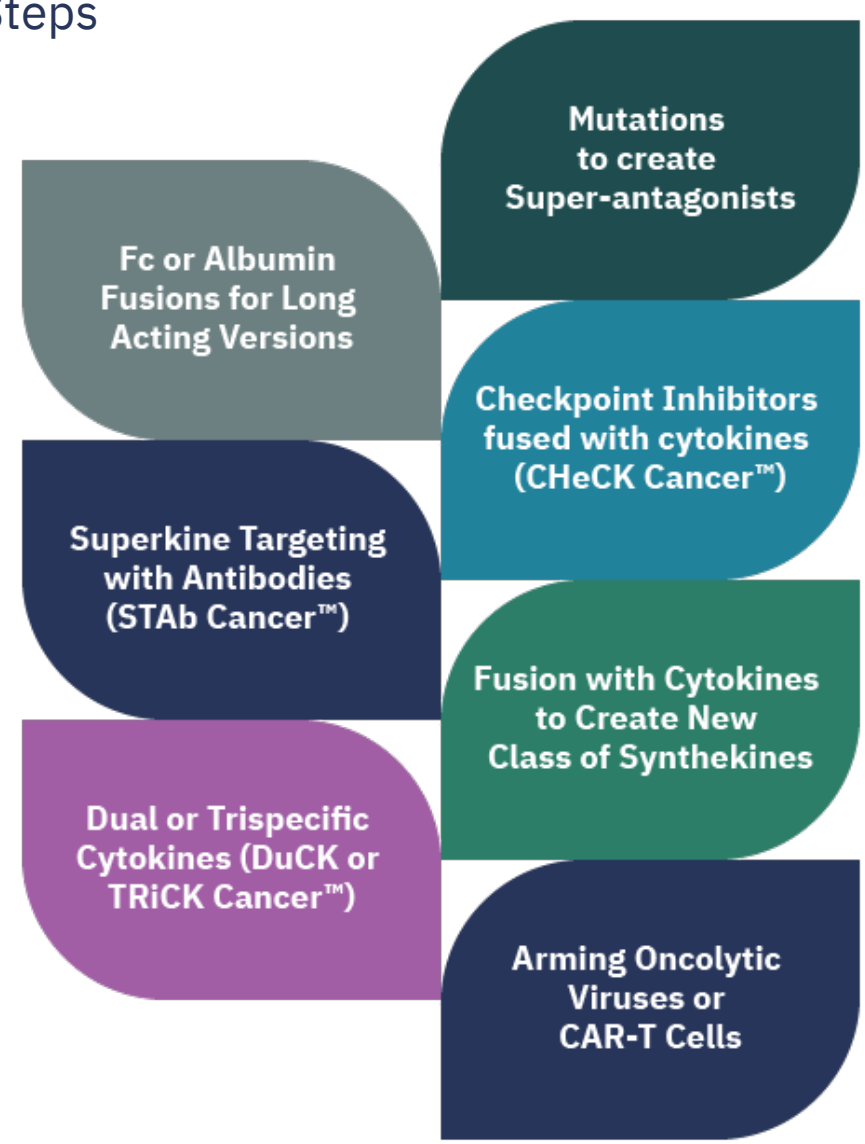
Safety run-in (21 days)

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 Next Steps

- Initiate Phase 1/2a clinical trial **(Mid 2021)**
- Report top-line Safety, PK/PD and Biomarker Results from Phase 1/2a monotherapy study **(End 2021)**
- Phase 1/2a Efficacy Data **(1H 2022)**

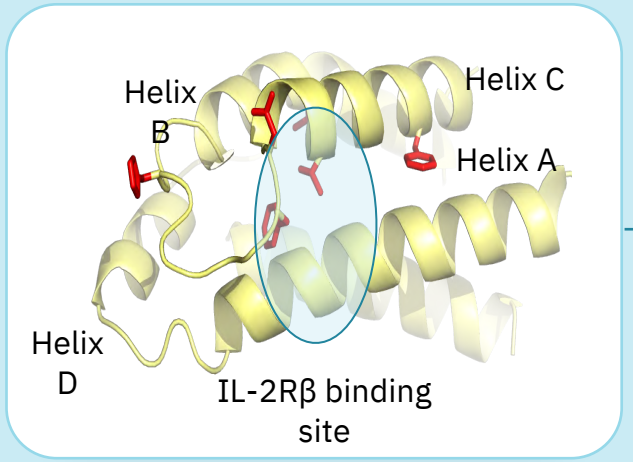
Bifunctional Superkines for Immunotherapy (BiSKITs)

Superkine Targeted with Antibody (STAb™)

Enhances accumulation in tumors

nature COMMUNICATIONS

Sun et. Al., Nat comm, 2019



+FA **SumIL-2** (MDNA109FA)

αEGFR-SumIL-2 (αEGFR-MDNA109FA)

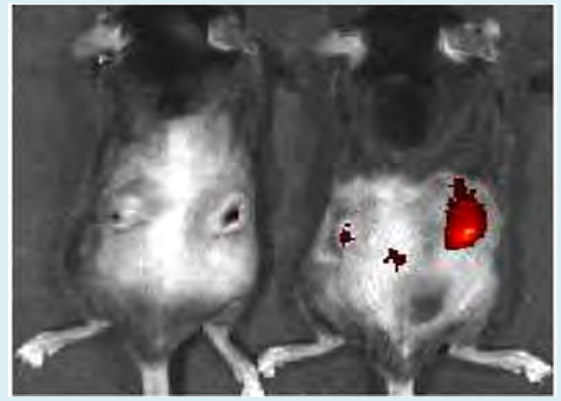
+FEAA **MDNA19**

MDNA19 (MDNA109FEAA-Fc)

Medicenna Therapeutics

Tumor Accumulation

Control αEGFR-MDNA109FA



Left tumor: MC38
Right tumor: MC38-EGFR5

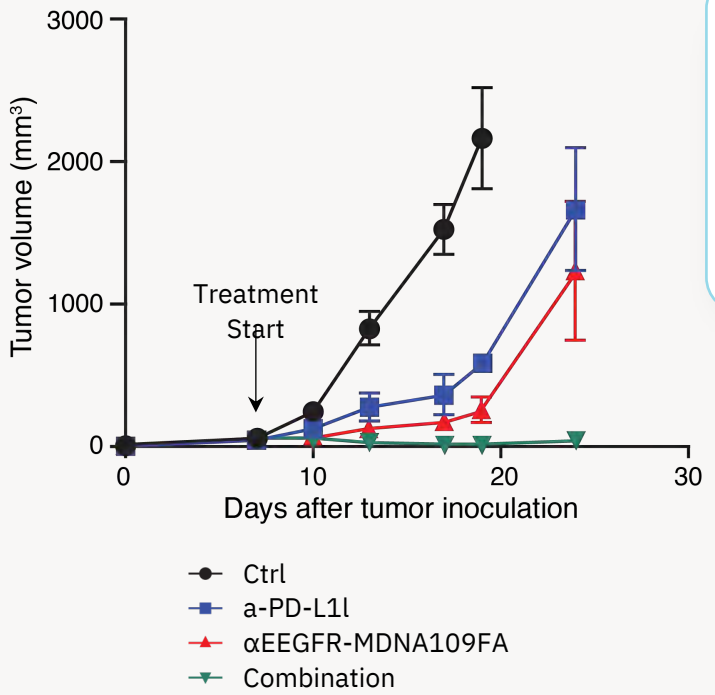
Fluorescence images of MC38 (left) and MC38-EGFR5 (right) tumor-bearing mice treated with a single dose of PBS or αEGFR-MDNA109FA (25 μg, IV)

Sun et al., Nature Communications, 2019

STAb™ Overcomes Checkpoint Resistance and 'Cold' Tumors

Overcoming Checkpoint Resistance

B16F10-EGFR5 Tumors



αEGFR-SumIL-2
(αEGFR-MDNA109FA)

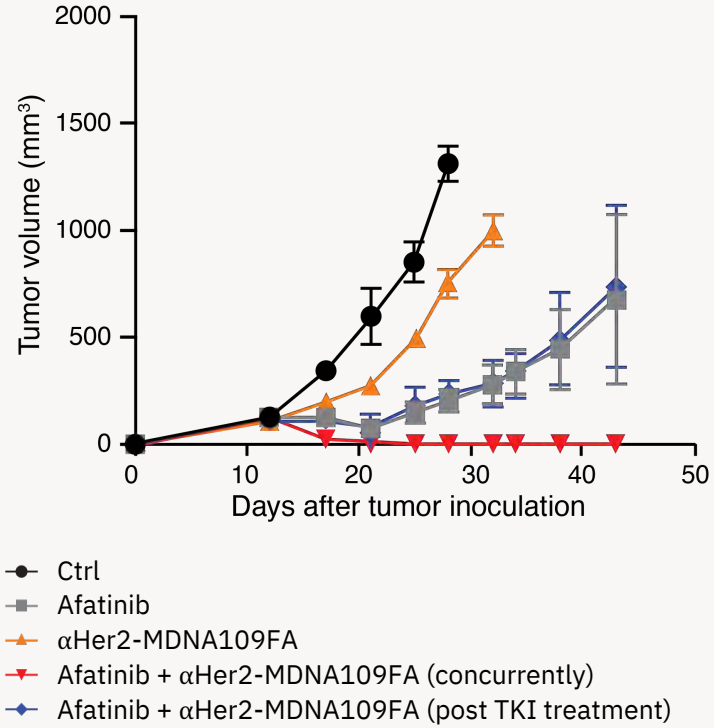
IP treated with 25 µg of αEGFR-MDNA109FA-Fc. Intratumorally treated with 50 µg of anti-PD-L1 on days 8, 11, and 14.

nature COMMUNICATIONS

Synergy with TKI to Tackle Immunological 'Cold' Tumors

αHer2-SumIL-2
(αHer2-MDNA109FA)

TUBO Tumors (overexpress Her2)

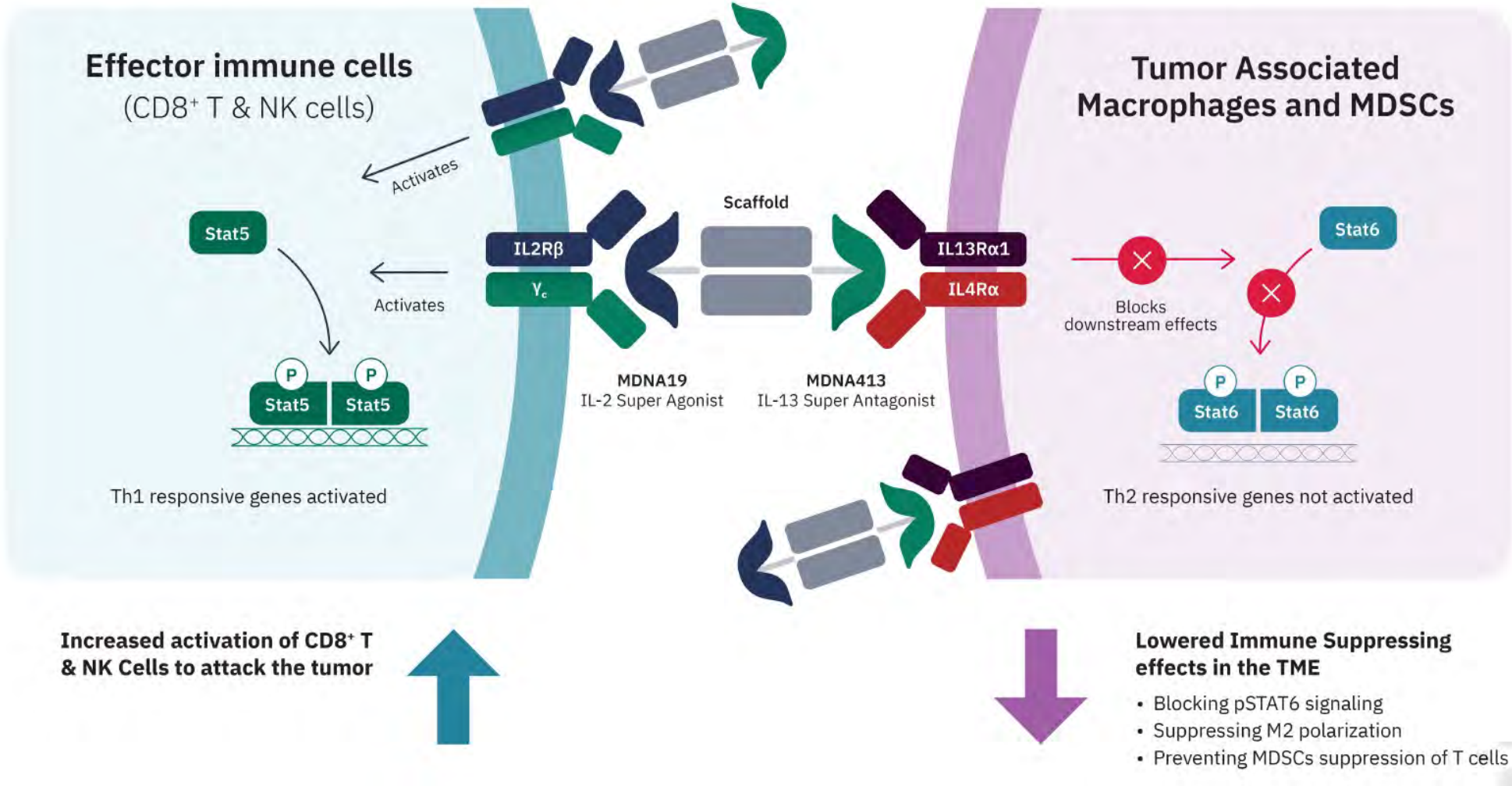


IP treated with 20 µg of anti-αHer2-MDNA109FA on either days 12, 15, and 18 or days 25, 28, and 31. Orally with 1 mg of Afatinib on days 12 and 17.

Sun et al., Nature Communications, 2019

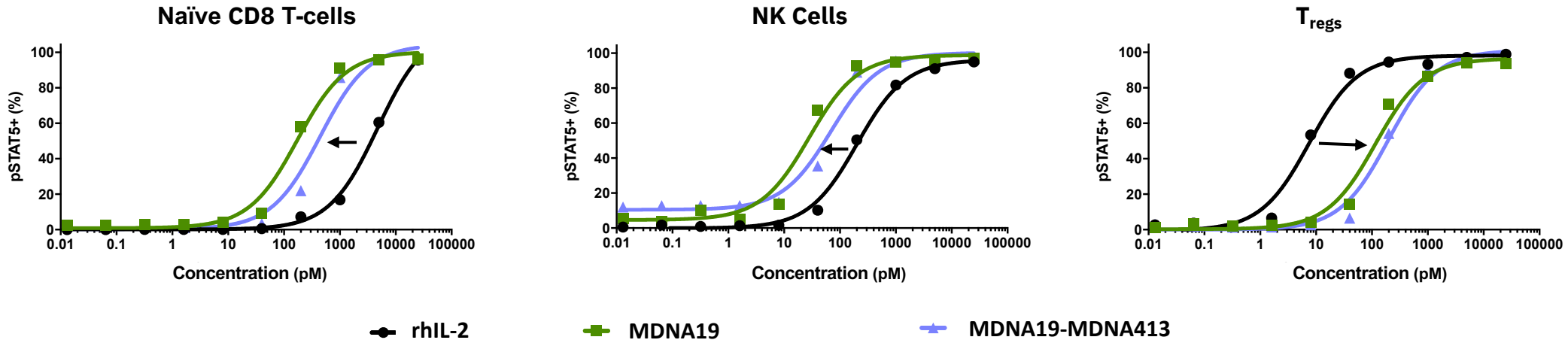
Dual Specific Cytokine (DUCK Cancer™) Mechanism of Action

Targeting immunologic ‘cold tumors’ by modulation of the Tumor Microenvironment (TME)



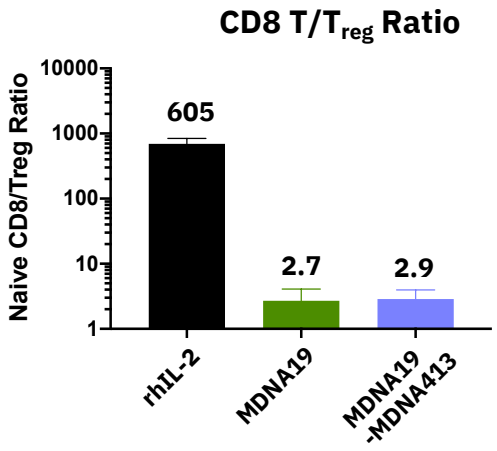
Bi-Specific Superkine

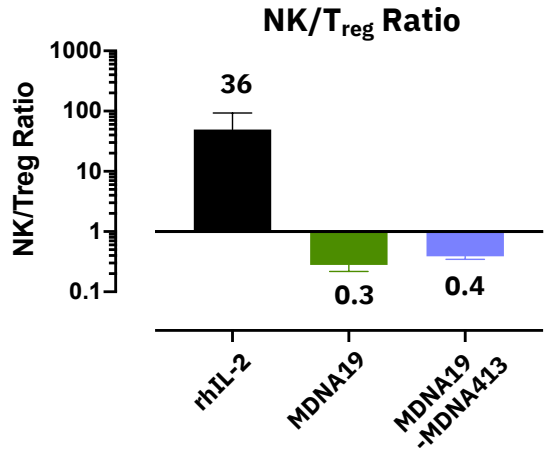
Enhanced signaling in CD8⁺ T and NK cells; diminished signaling in T_{regs} IL-2 Agonism maintained



P-STAT5 (EC_{50}, pM)	rhIL-2	MDNA19	MDNA19-MDNA413
Naïve CD8⁺ T cells	3389.5	370.6	575.8
NK cells	201.5	71.0	80.1
T_{regs}	5.6	135.5	210.3

Studies in human PBMC

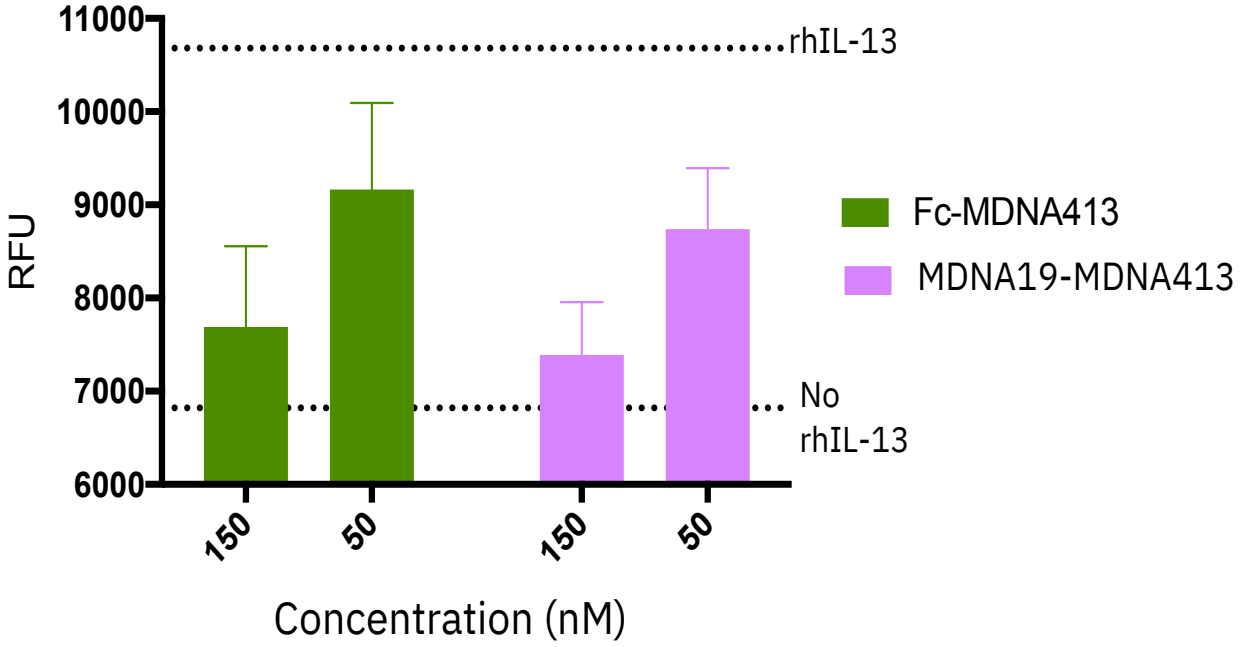


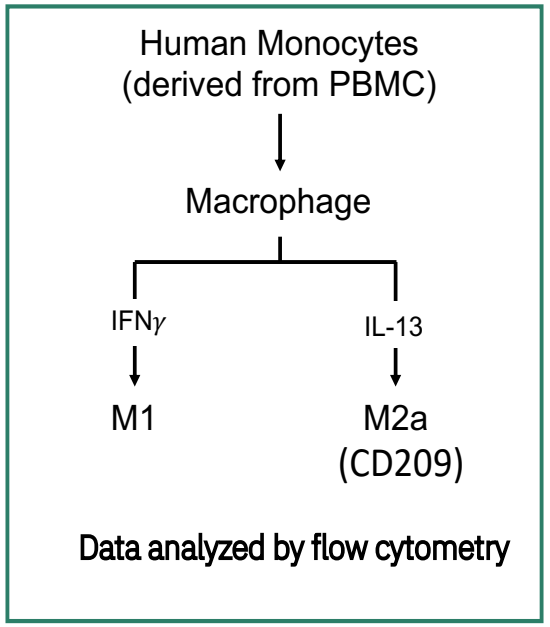


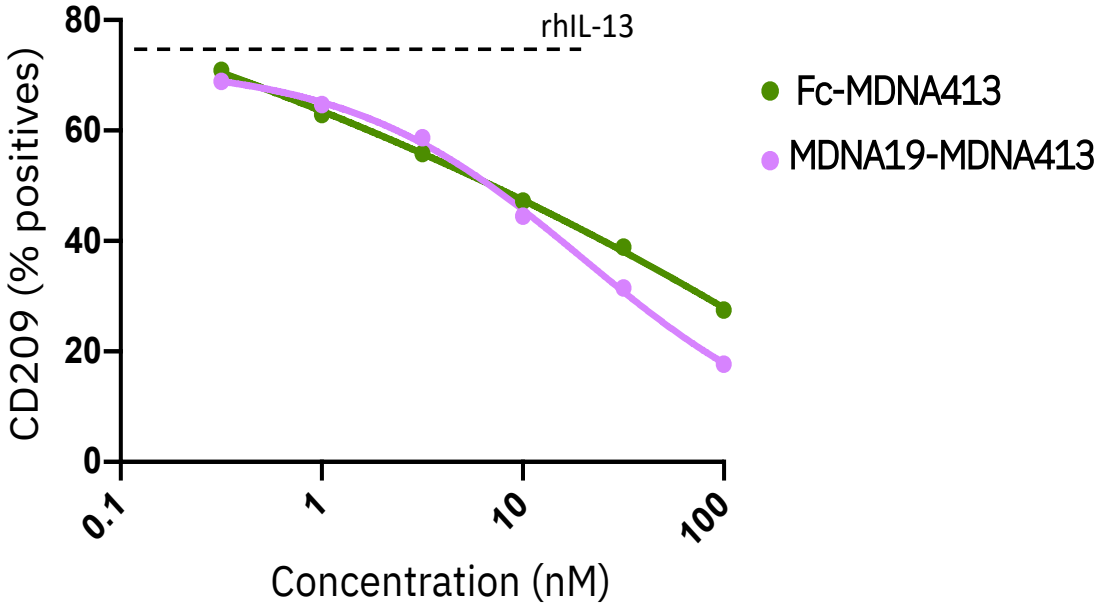
Bi-Specific Superkine

Inhibits IL-4 & IL-13 Induced Signaling, TF-1 Proliferation & M2a Polarization

TF-1 is a human erythro-leukemia cell line that is highly dependent on IL-4/IL-13 for proliferation







MDNA55

A Powerful Molecular Trojan Horse 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

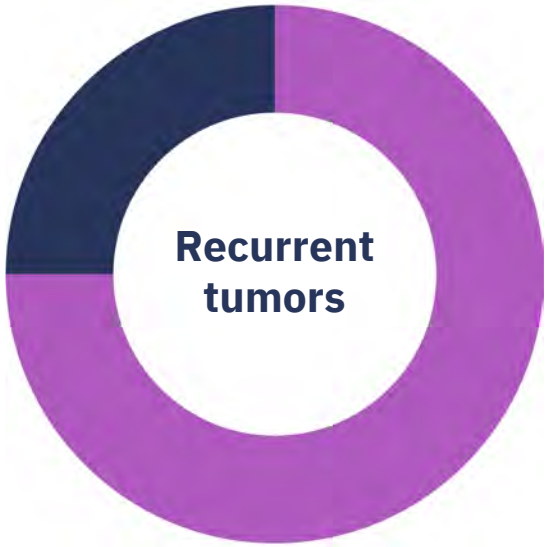
- Not all patients are eligible for surgery and chemotherapy regimen in the first place, while the vast majority of recurrent tumors are inoperable
- Survival for patients undergoing the standard therapy is extremely low: The two-year survival rate is 5-10%

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

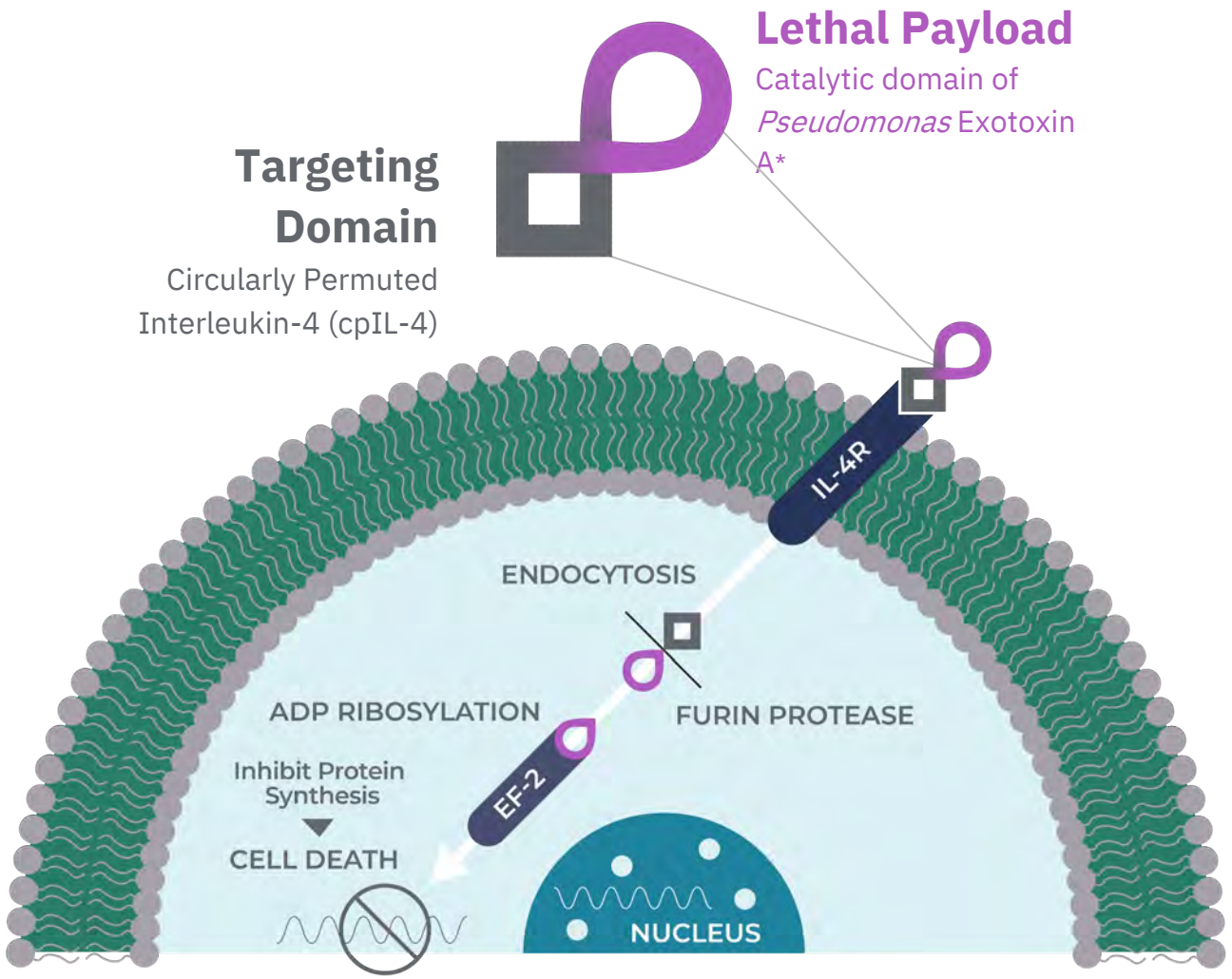
By Passes BBB
Single intra-tumoral infusion avoids systemic toxicity and achieves tumor control

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 All-comers (n=43)

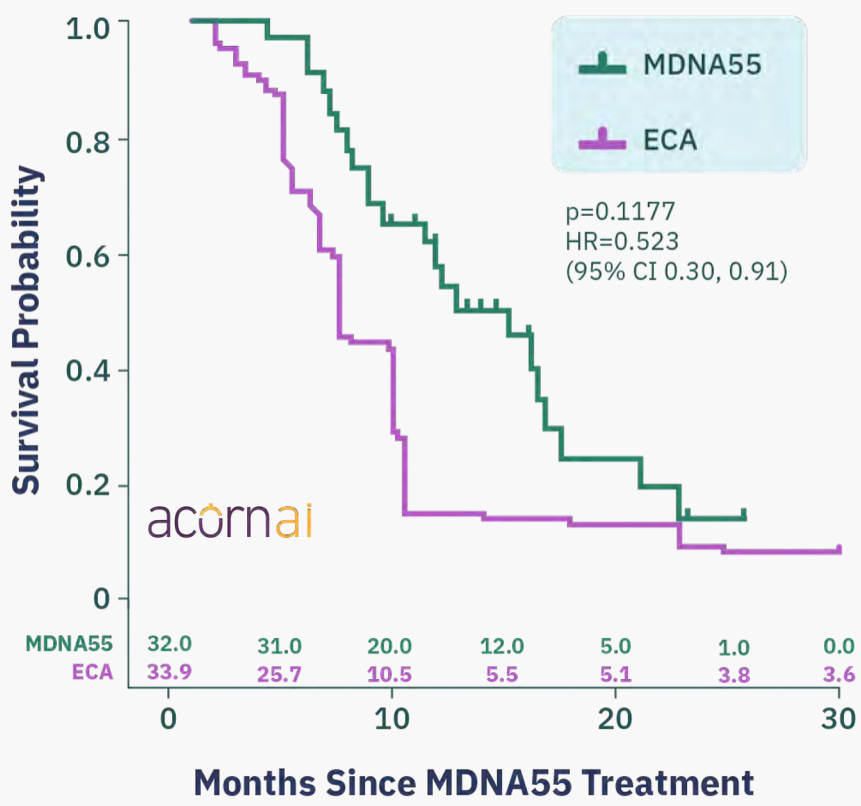
mOS is 12.4** months vs. 7.2 months in ECA

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

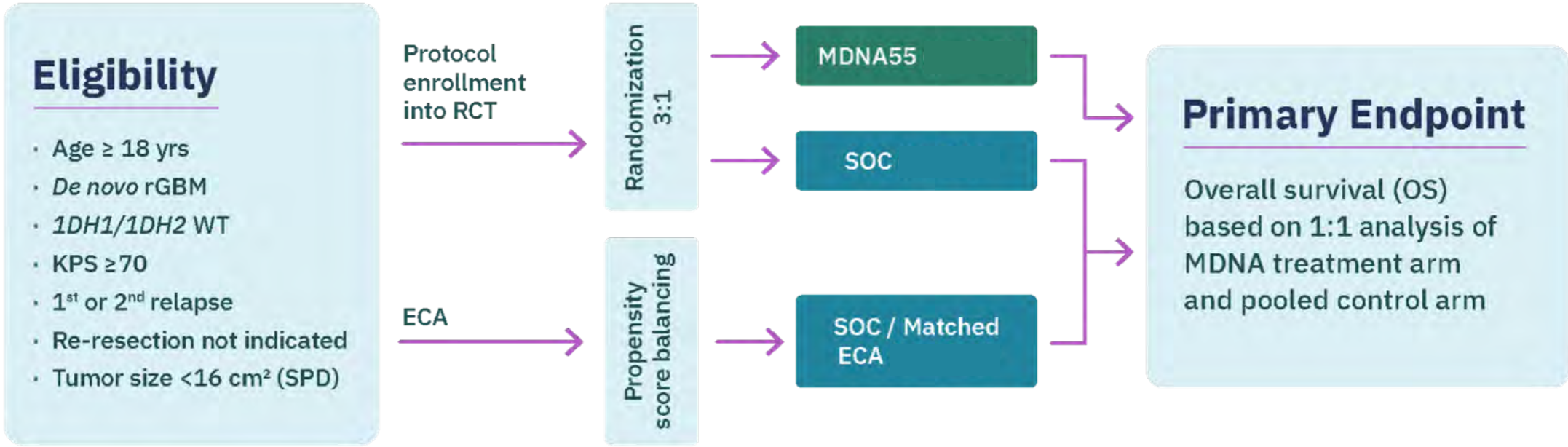
IL4R High + IL4R Low$^{\text{High Dose}}$



*Survival was calculated from time of relapse | **mOS is 12.4 months as of 15 Sep 2020

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%

SOC Therapies Allowed

- Bevacizumab (Avastin®)
- Lomustine (CCNU, CeeNU®, GleostineTM)
- Temozolomide (Temodar®)
- Tumor Treating Fields (Optune®)
- Radiation Therapy

Thank you

Fahar Merchant, PhD
President and CEO

Elizabeth Williams
Chief Financial Officer

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