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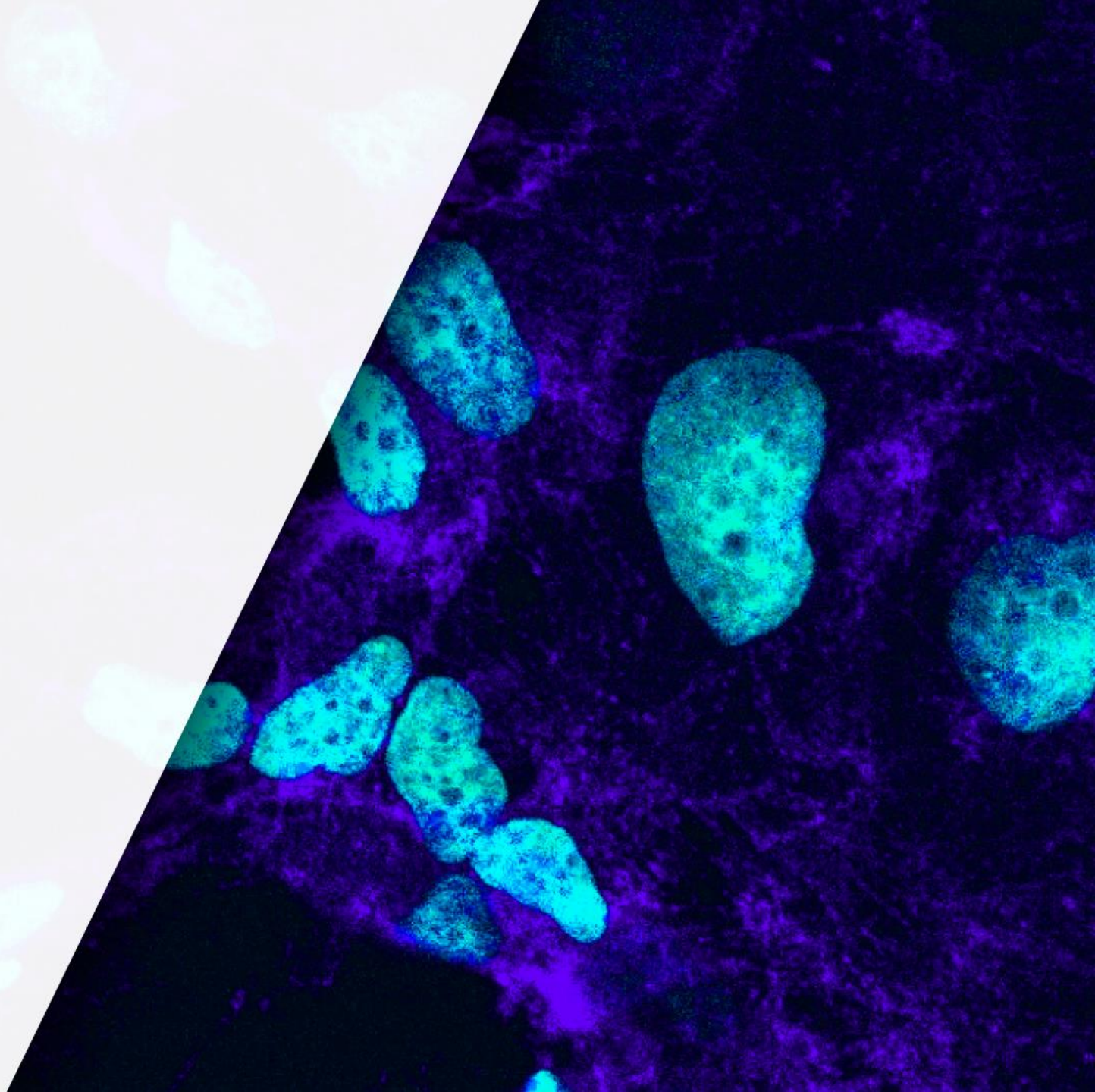
UNMASKING CANCER CELL CAMOUFLAGE

and activating a targeted immune response

COMPANY PRESENTATION | FEB. 2021



SPECIAL NOTE REGARDING FORWARD LOOKING STATEMENTS

This presentation contains forward-looking statements about our expectations, beliefs and intentions regarding, among other things, our product development efforts, business, financial condition, results of operations, strategies, plans and prospects. In addition, from time to time, we or our representatives have made or may make forward-looking statements, orally or in writing. Forward-looking statements can be identified by the use of forward-looking words such as “believe”, “expect”, “intend”, “plan”, “may”, “should”, “could”, “might”, “seek”, “target”, “will”, “project”, “forecast”, “continue” or “anticipate” or their negatives or variations of these words or other comparable words or by the fact that these statements do not relate strictly to historical matters. Forward-looking statements relate to anticipated or expected events, activities, trends or results as of the date they are made. Because forward-looking statements relate to matters that have not yet occurred, these statements are inherently subject to risks and uncertainties that could cause our actual results to differ materially from any future results expressed or implied by the forward-looking statements. Many factors could cause our actual activities or results to differ materially from the activities and results anticipated in forward-looking statements.

We believe these forward-looking statements are reasonable; however, these statements are only current predictions and are subject to known and unknown risks, uncertainties and other factors that may cause our or our industry’s actual results, levels of activity, performance or achievements to be materially different from those anticipated by the forward-looking statements.

All forward-looking statements speak only as of the date hereof, and we undertake no obligations to update or revise forward-looking statements to reflect events or circumstances that arise after the date made or to reflect the occurrence of unanticipated events, except as required by applicable law. In evaluating forward-looking statements, you should consider these risks and uncertainties.

INVESTMENT HIGHLIGHTS

MIRP™
Multifunctional Immuno-Recruitment Proteins - A family of Immuno-therapeutic drugs for multiple cancer types

CURRENT STATUS
- Robust preclinical results
- Phase I/II for solid tumors
- Collaboration with ROCHE to combine with Atezolizumab

PIPELINE
-1st product | Phase I/II CD47/41BB
-2nd & 3rd products | IND H2 2022
-Multiple future candidates in R&D

MARKET
Immuno-therapeutics
$56.5B by 2025

IP
15 families
2 granted (US and other territories),
13 pending (NP worldwide and PCT stage)

STRONG TEAM
Experienced management, supported by reputable KOLs, amongst which is technology inventor, Prof. Mark Tykocinski, Dean of the School of Medicine and Provost, Jefferson University.

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LEADERSHIP TEAM

Management

Yaron Pereg, PhD
CEO
Genentech A Member of the Roche Group · BIOLINERX · CELLECT · GLOSENSE

Adam Foley-Comer, MD
CMO
Roche · BIOLINERX · QUINTILES · IMMUNE Pharmaceuticals · Quark Pharmaceuticals

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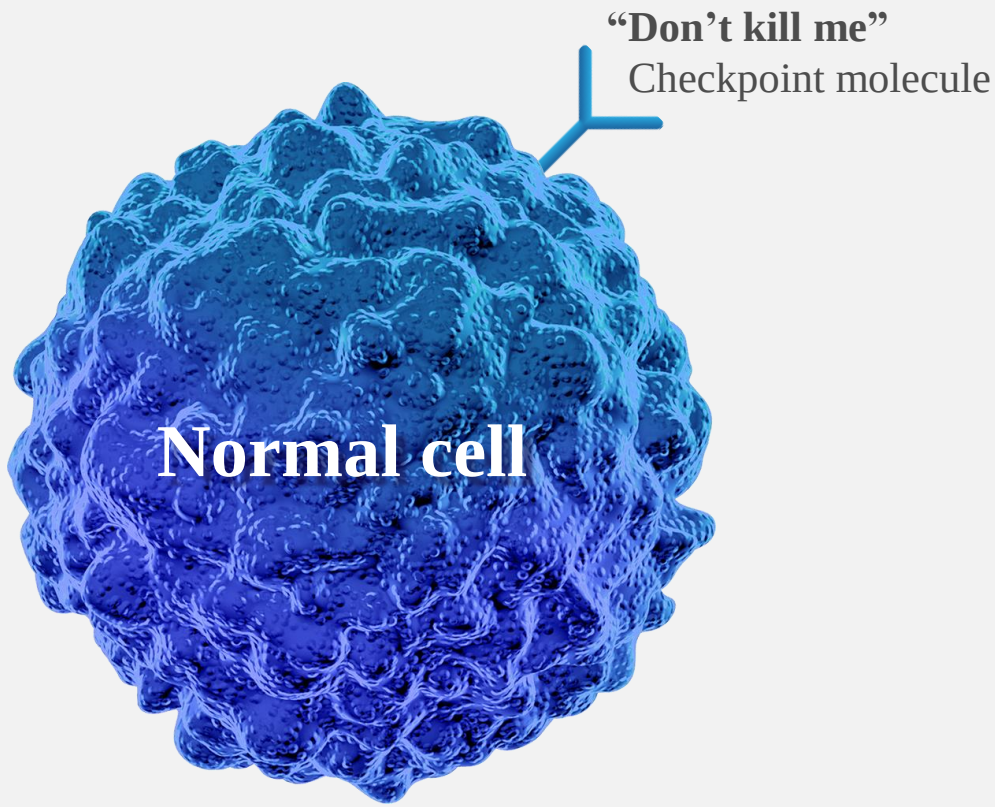
Associate Professor, Harvard Medical School and Director Cell Biology, Beth Israel Deaconess Medical Center

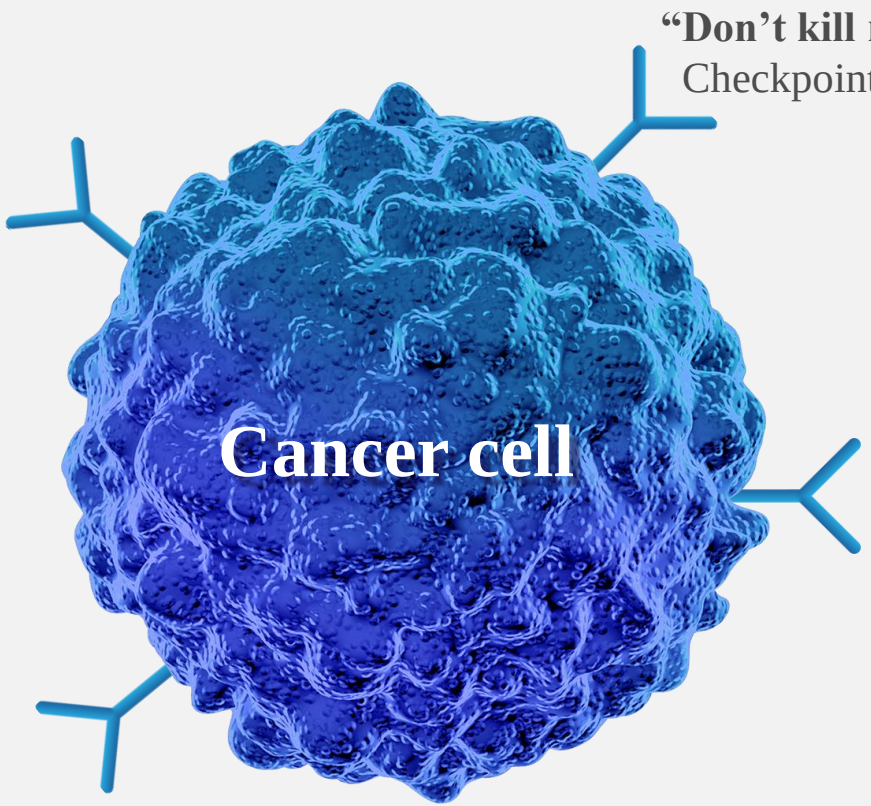
THE CAMOUFLAGE CHALLENGE IN TREATING CANCER



Immune checkpoints are molecules expressed on all cells in the body that regulate the immune system’s self-tolerance, to **prevent indiscriminate attack** of healthy cells.

When immune cells bind to checkpoint molecules, their activity is inhibited

THE CAMOUFLAGE CHALLENGE IN TREATING CANCER



Cancer cells overexpress immune checkpoint molecules to camouflage themselves from the immune system by **pretending to be normal cells**, thus eluding immune recognition and attack.

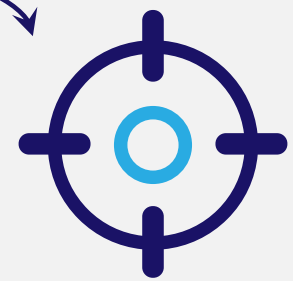
CURRENT CHECKPOINT IMMUNOTHERAPY HAS ITS DOWNSIDES

Low tissue specificity	Immune system attacks healthy cells	Mild to severe autoimmune side effects	**Non targeted checkpoint inhibition is suboptimal!**
Low response rate	Non durable responses in subset of patients	Neutralizing defenses is not enough	

EFFECTIVELY TREATING CANCER REQUIRES A MULTIFACETED APPROACH

Selectively disabling its defenses

WHILE



Recruiting a local targeted immune attack

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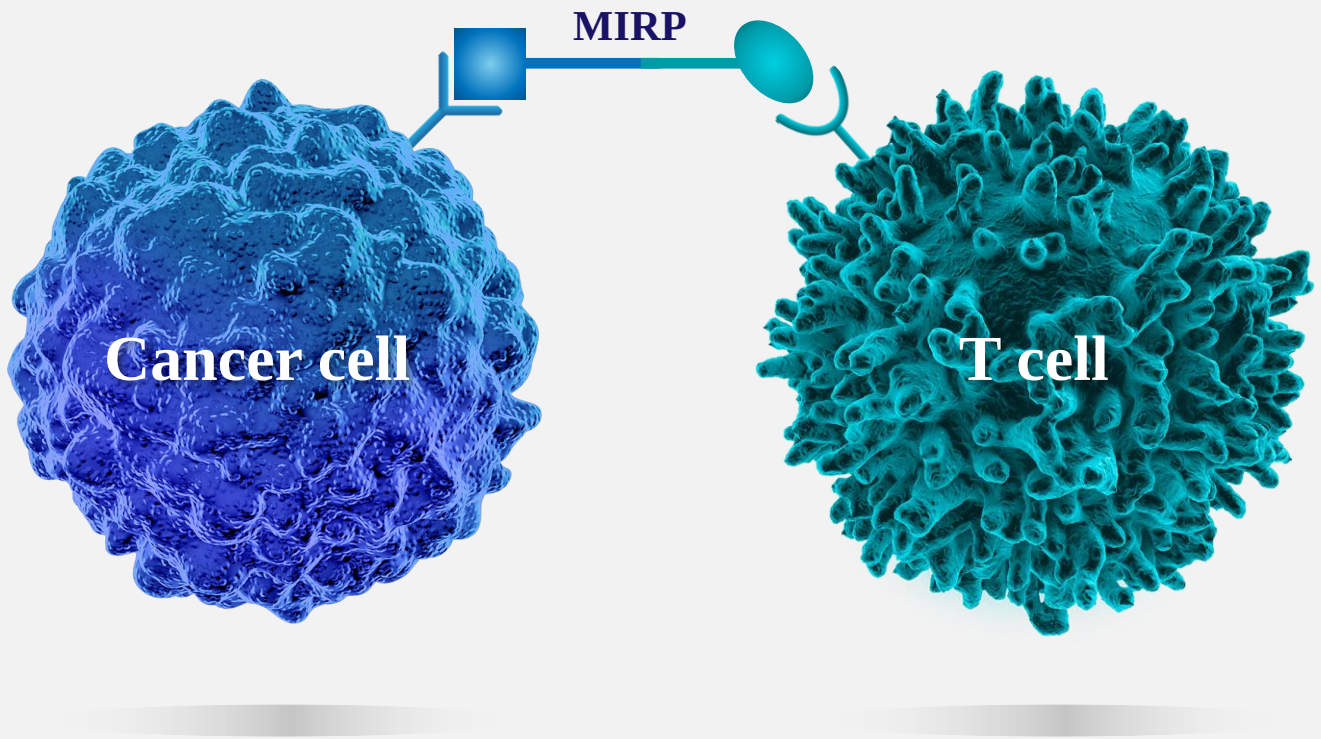
KAHR develops smart immuno-recruitment cancer drugs that activate a targeted immune response by converting cancer camouflage into beacons for the immune system to attack

OUR PLATFORM TECHNOLOGY

MIRP (MULTI-FUNCTIONAL IMMUNO-RECRUITMENT PROTEINS)

MIRPs deliver a multilayered attack by binding cancer cells and T-cells to produce a targeted synergistic effect, combining immune checkpoint inhibition with selective T-cell activation.



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HOW IT WORKS

Targeting checkpoint overexpression

MIRPs utilize cancer cell overexpression of checkpoint antigens to selectively target and bind to the cancer

Inhibiting cancer checkpoints

Checkpoint binding and inhibition unmasks the cancer cell's camouflage and enables immune response

Recruiting adaptive immunity

MIRPs bind to T-cells and activate them in the cancer environment

Activating immune response

Activated T-cells initiate a selective and local immune response to kill the cancer cells

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PIPELINE

Targets	MIRP Type / MOA	Discovery	Preclinical	Clinical	Licensor
DSP107 CD47 x 41BB	**DSP** Activating both, innate and adaptive immunity	Phase I/II Solid Tumors ongoing; Phase I/II AML/MDS Q3/2021; Phase II 2L NSCLC Q4/2021			
DSP502 PVR x PD-L1	**DSP-Fc (IgG1)** Dual checkpoint inhibition	IND ready by Q4/2022			Jefferson Thomas Jefferson University
DSP216 HLA-G x CD47	**DSP-Fc** Enhancing tumor targeting through dual checkpoint binding & immune stimulation	IND ready by Q1/2023			Jefferson Thomas Jefferson University
DSP105 PD-L1 - 41BB	**DSP** Expanding and stimulating Tumor reactive circulating effector T cells	Assessed for ex-vivo use as novel adoptive T cell therapy			Beth Israel Deaconess Medical Center

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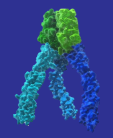
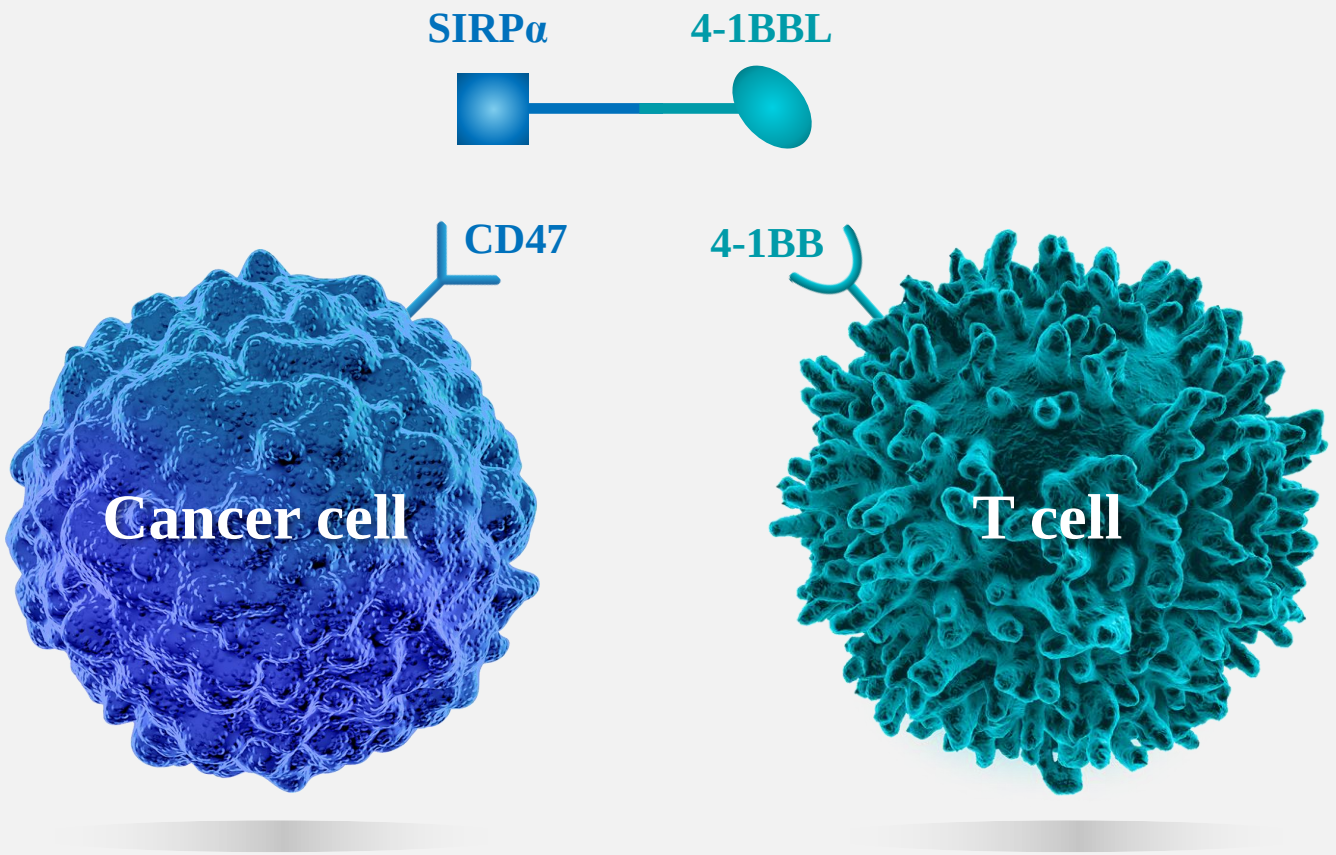


LEAD MIRP PRODUCT – **DSP107**

DSP107 (MIRP type - Dual Signaling Protein)

SIRPα binds to **CD47** overexpressed on cancer cells, disabling their "don't eat me" signal



4-1BBL side binds to **4-1BB** on tumor-antigen specific T cells, stimulating their expansion, cytokine production, and the development of cytolytic effector functions

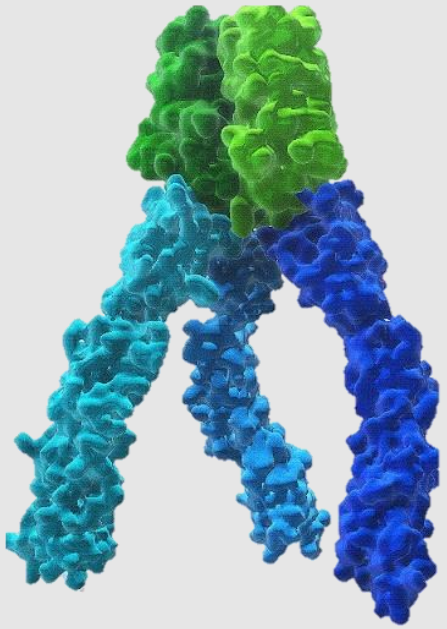
UNIQUE TRIMERIC STRUCTURE ENABLE SPECIFICITY AND SELECTIVITY

DSP107 Structure

Trimeric ligand ends enable both:

- High tissue specificity by binding overexpressed checkpoint molecules driven by affinity and high avidity
- Selective activation of immunity by recruiting and co-stimulating local immune cells

Trimeric 4-1BBL



Immune cell stimulation

Proliferation

Checkpoint inhibition

Tumor microenvironment modulation

3 SIRPα for
CD47 Checkpoint Targeting

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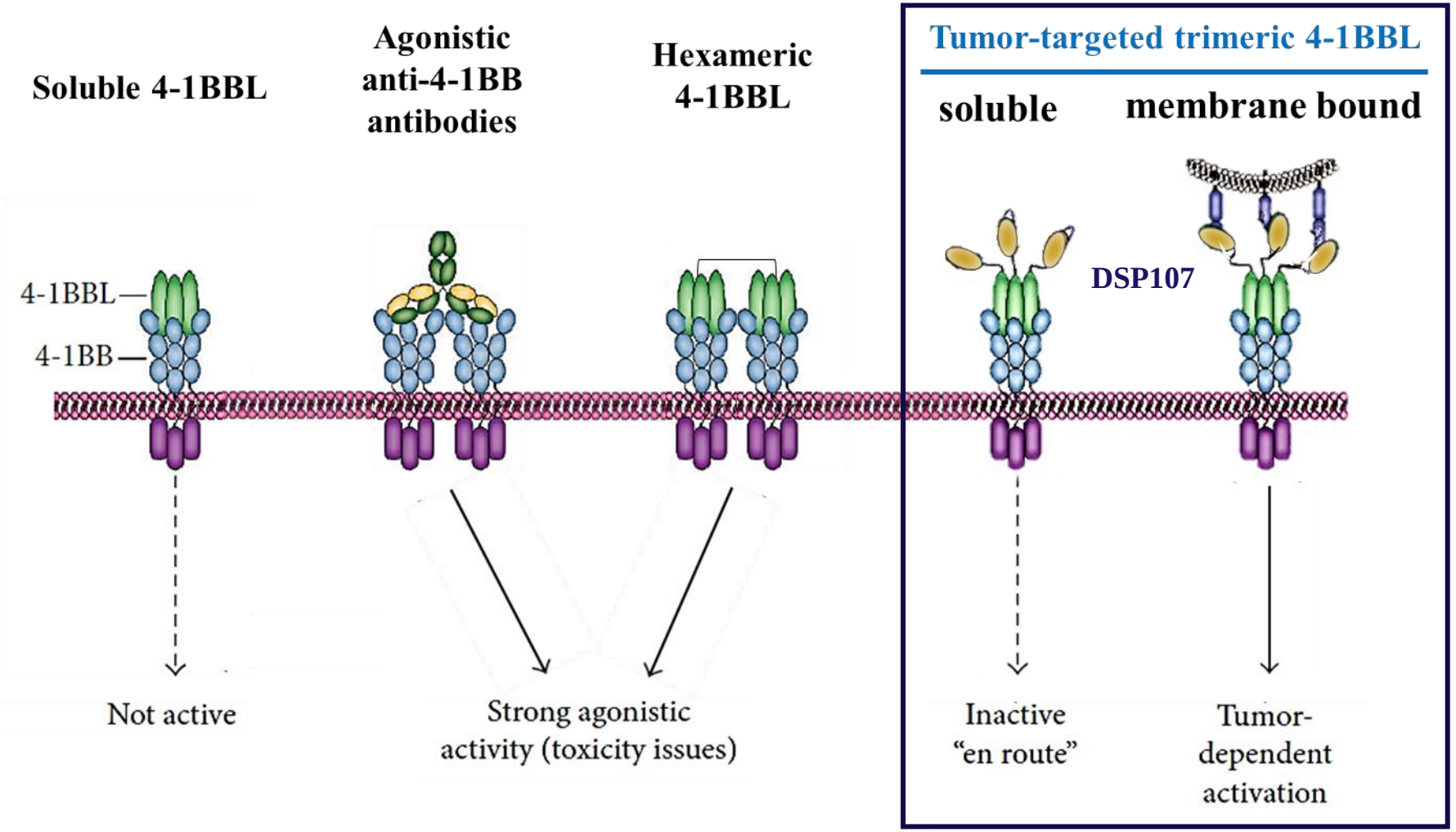
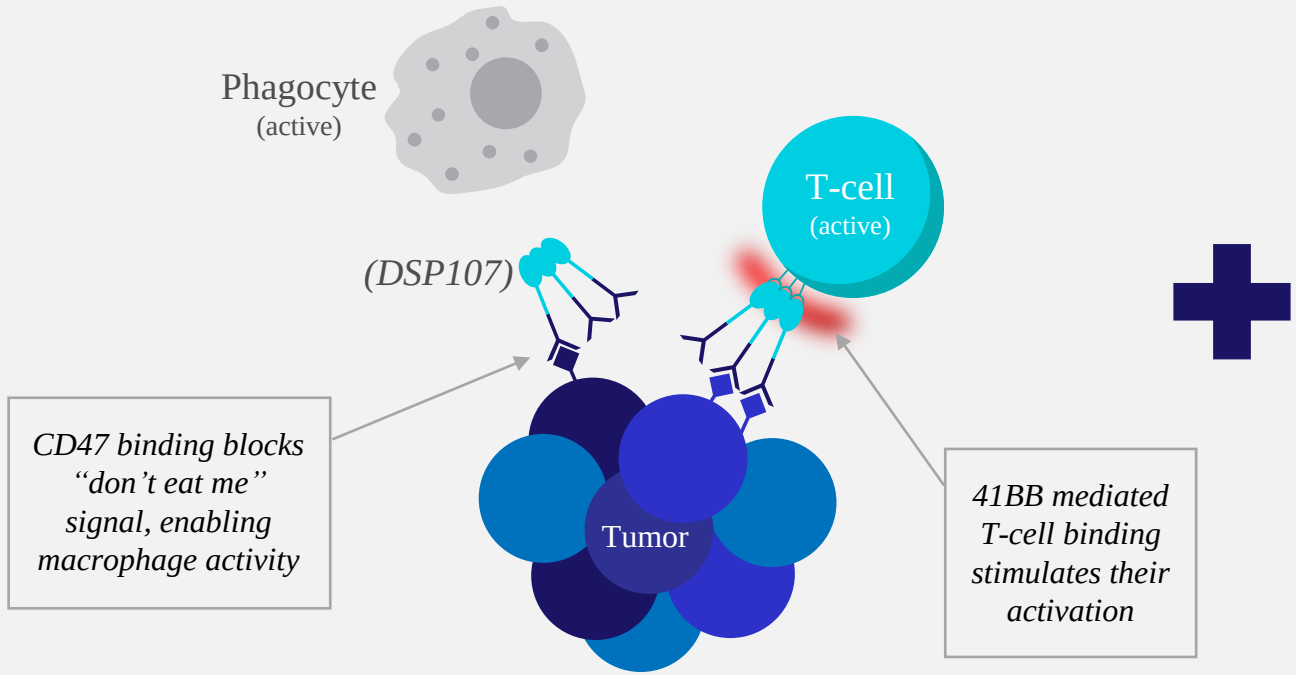
UNIQUE TRIMERIC STRUCTURE ENABLE TUMOR TARGETED 4-1BB CONDITIONAL ACTIVATION



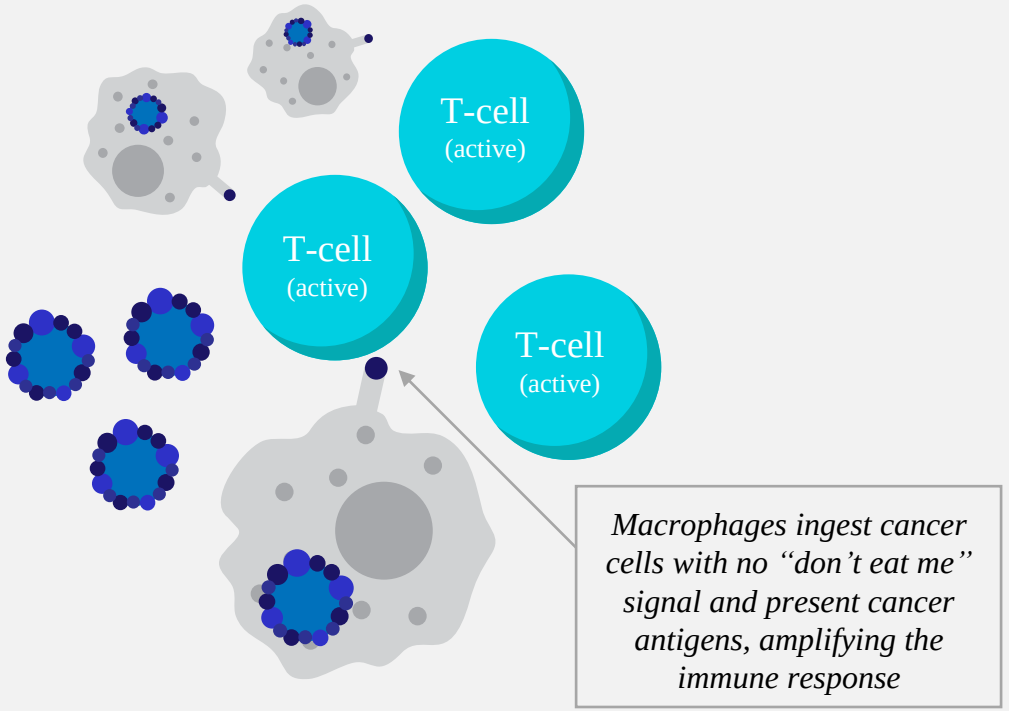
Modified from: Bremer E.; ISRN Oncology; Volume 2013, Article ID 371854

DSP107 – SYNERGISTIC IMMUNE ACTIVATION

Innate immunity enabled + mediated activation of adaptive immunity



Natural adaptive immune response



DSP107 – BEST IN CLASS CD47 TARGETING COMPOUND

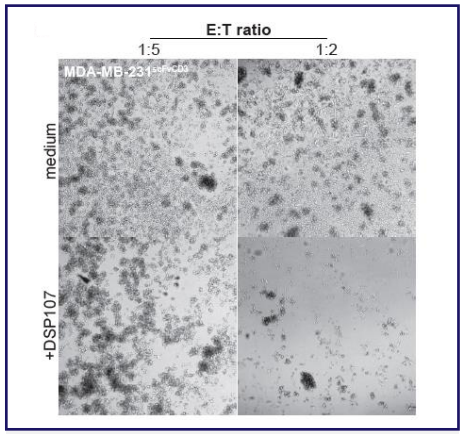
Next generation capabilities

Dual MOA
activates innate and adaptive immunity

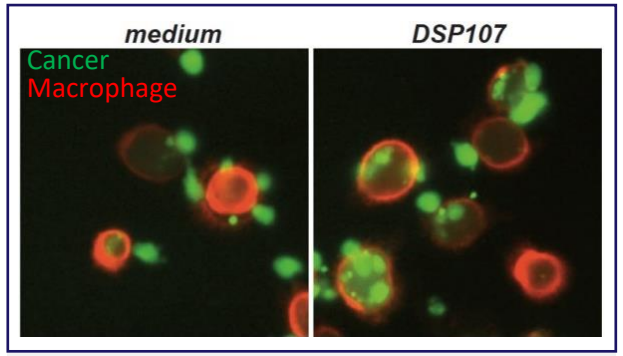
Excellent safety
without hematological toxicities

Strongly positioned
for treatment of solid and hematological malignancies

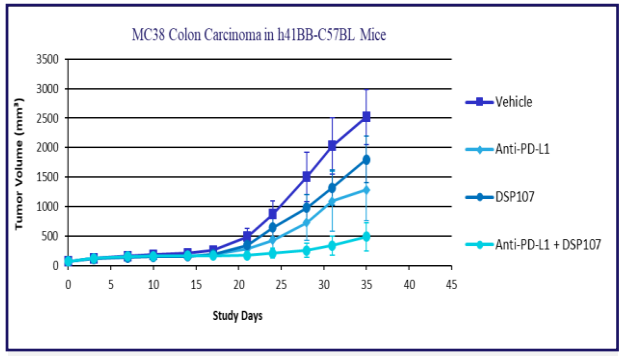
Unique synergistic effects



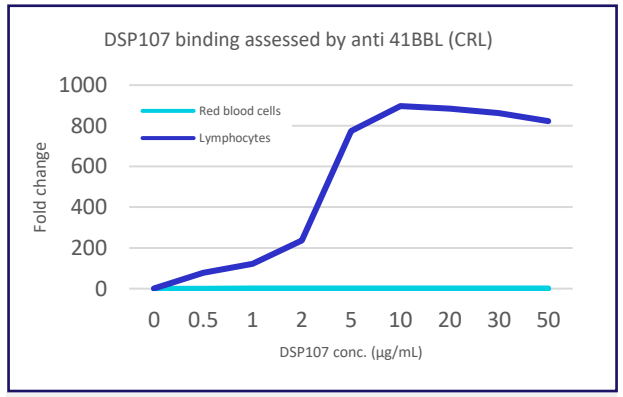
Activates T cells to secrete IFN-γ and augment their cancer cell killing potential



Augments macrophages-mediated phagocytosis of tumor cells as a single agent and synergizes with mAb's



Strong anti tumor activity as a single agent and synergizes with PD1/PD-L1 checkpoint inhibitors in-vivo



Does not bind red blood cells, avoiding antigen sink issues, resulting in a best-in-class safety profile

CD47 AGENT PIPELINE

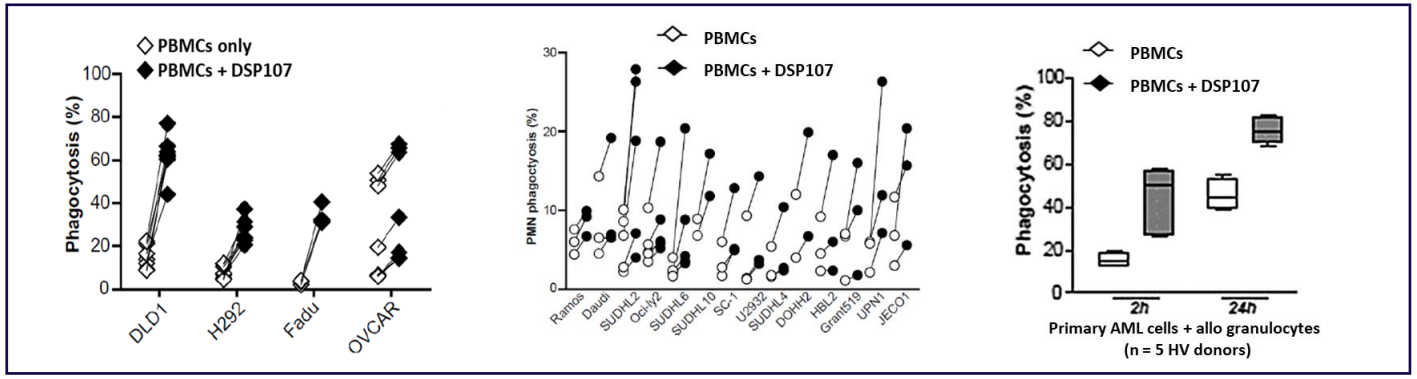
Candidate	DSP107	Magrolimab	ALX148	TTI-622	AO-176	TG-1801
Type	**SIRPα-41BBL** fusion protein	CD47 mAb	SIRPα-Fc fusion protein	SIRPα-Fc fusion protein	CD47 mAb	CD47/CD19 BisAb
Mechanism	**Bi-functional**	Monovalent	Monovalent	Monovalent	Monovalent	Bi-specific (not bi-functional)
Immune activation	**Innate and adaptive**	Innate	Innate	Innate	Innate	Innate
RBC binding - **Antigen Sink issue** - **Hematological toxicities**	**No**	Yes	Low	No	Low	No
Monotherapy (preclinical)	**Yes**	No	No	Yes	Yes	Yes

*Other companies with phase I stage CD47-targeting agents: Innovent Bio, Surface Oncology, Seattle Genetics, Novimmune, I-Mab/Abbvie, OSE, Hengrui, Shattuck Labs

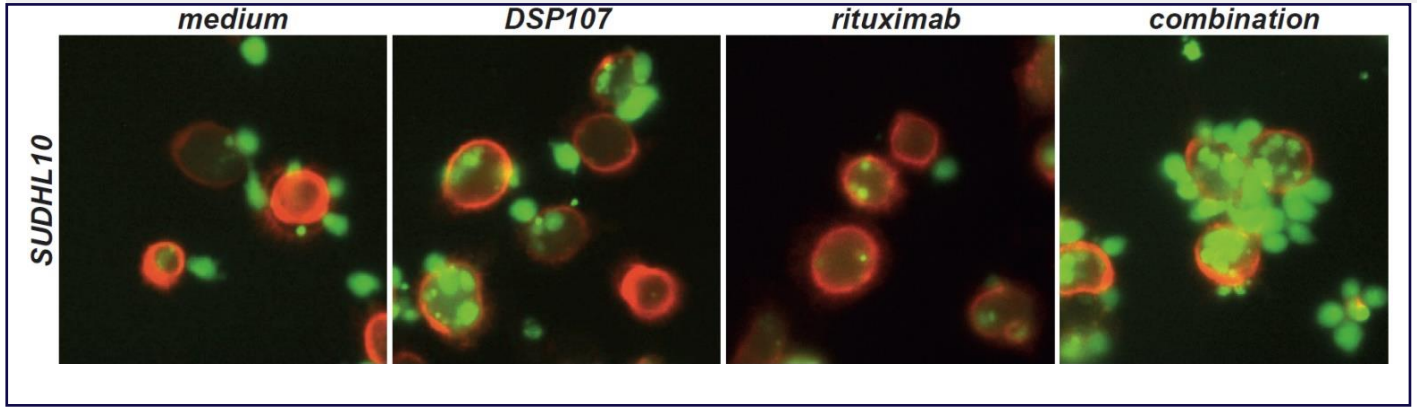
DSP107 - PRE-CLINICAL OVERVIEW

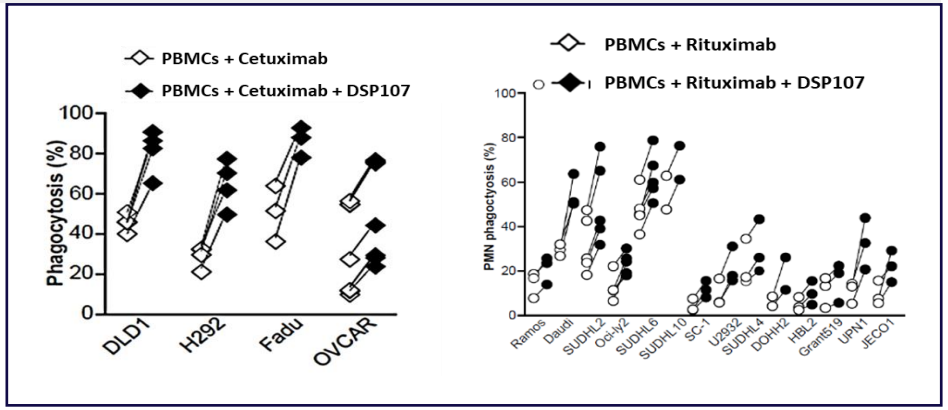
SIRPα – BINDS TUMOR AND INDUCES PHAGOCYTOSIS

Triggers cancer cell death by phagocytosis as a single agent



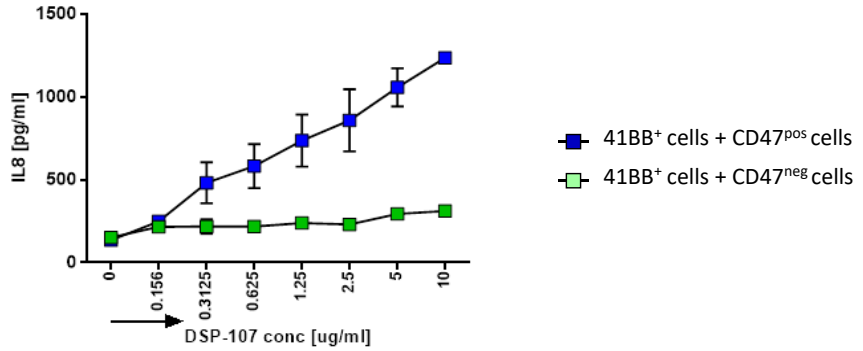
Augments mAb's ADCP-mediated phagocytosis of cancer cells



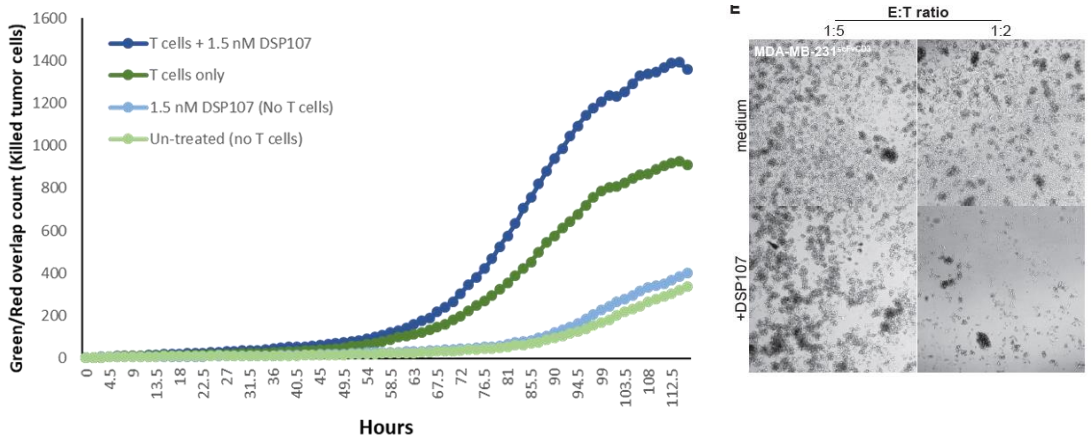


41BBL – ACTIVATES T-CELLS

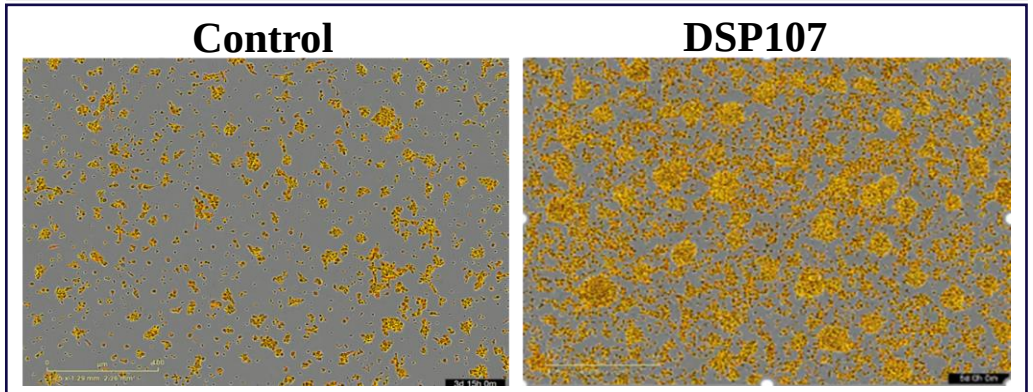
Tumor selective cross presentation activates 41BB signaling



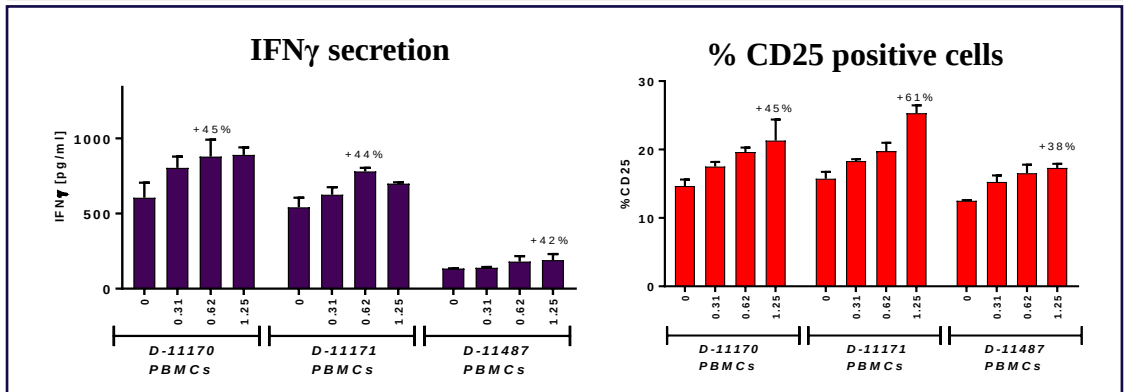
Induces T-cell killing potential against cancer cells



Augments T-cell proliferation

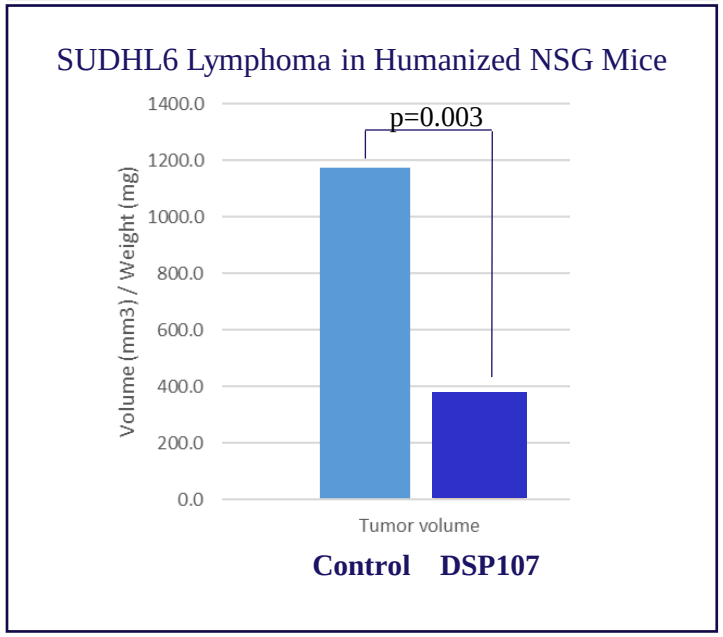


Activates T cells and increases IFNγ secretion

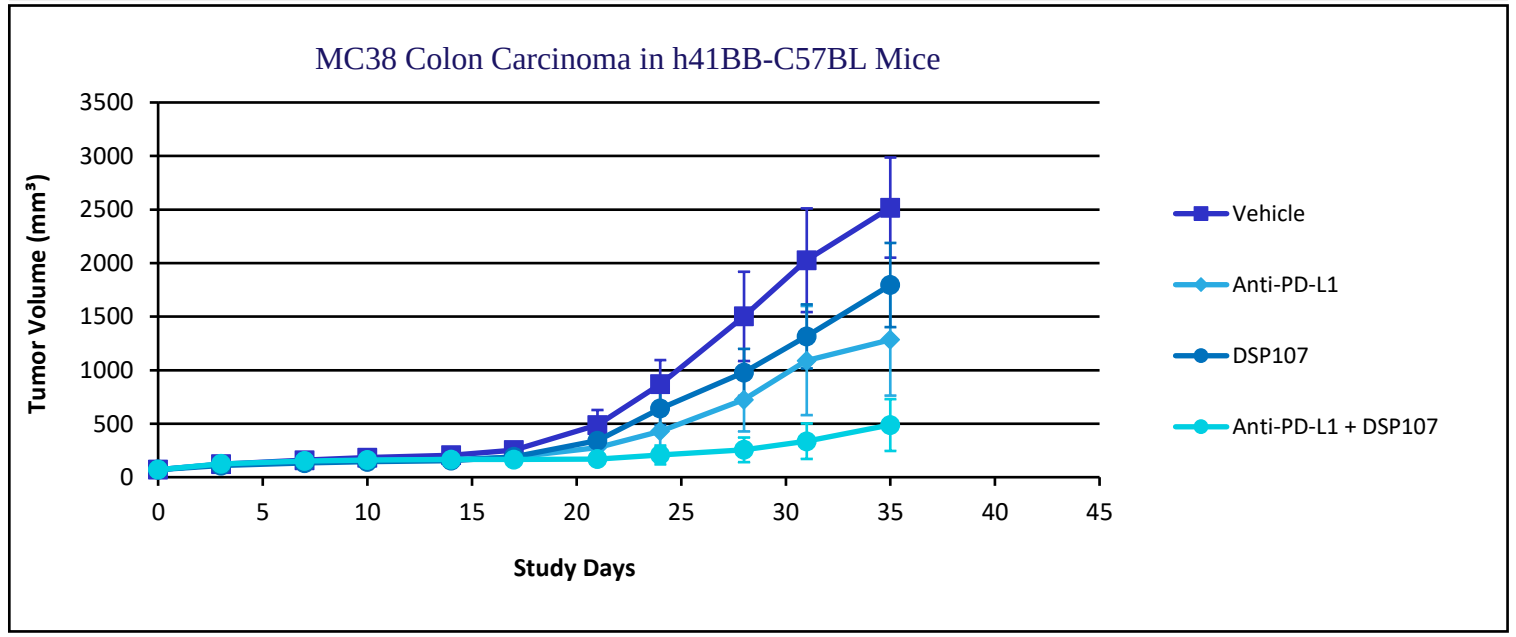


POTENT IN VIVO EFFICACY

Shows strong anti tumor activity as a single agent



Significant tumor inhibition and extended survival when combined with anti PD-L1

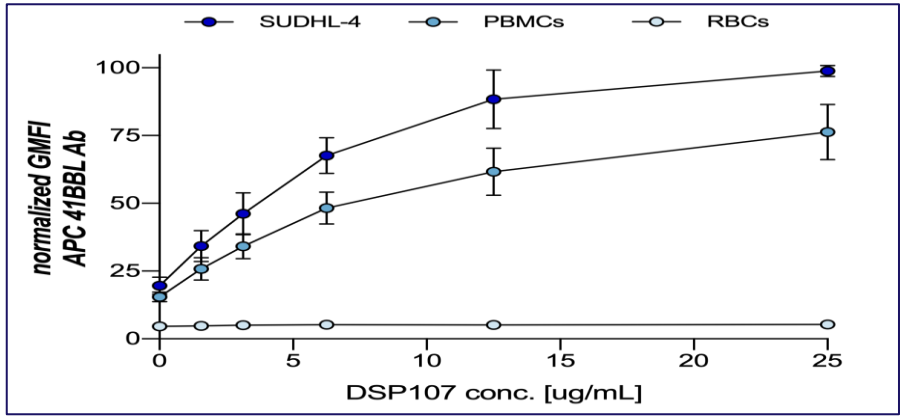


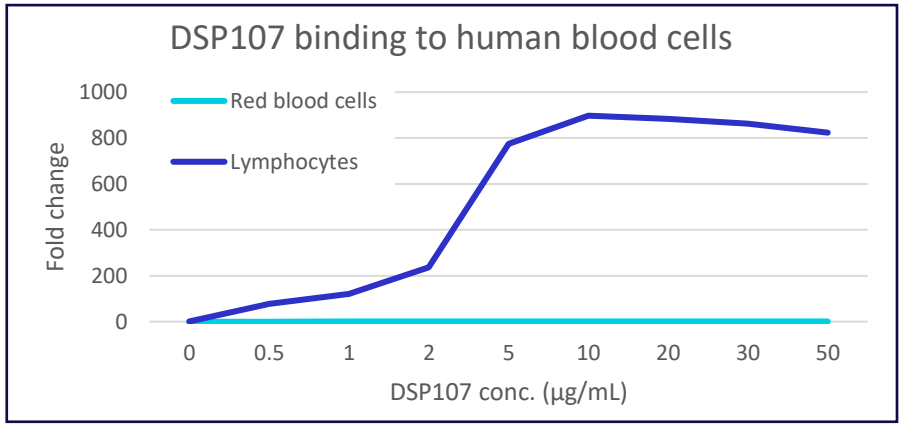
EXCELLENT SAFETY - NO HEMATOLOGICAL TOXICITIES

GLP Toxicology - Monkey study results

- Repeated administrations (up to 4) with doses of up to 50 mg/kg were safe & well tolerated
- No reduction in RBC count and Hb and no effect on platelets or white blood cells
- No changes in clinical chemistry parameters following repeated administration of DSP107
- No DSP107 related microscopic/macroscopic changes or findings (liver, spleen, kidneys, lung, lymph node)
- No treatment related changes in the cytokine levels

Increased Affinity to Cancer Cells and Negligible binding to RBCs

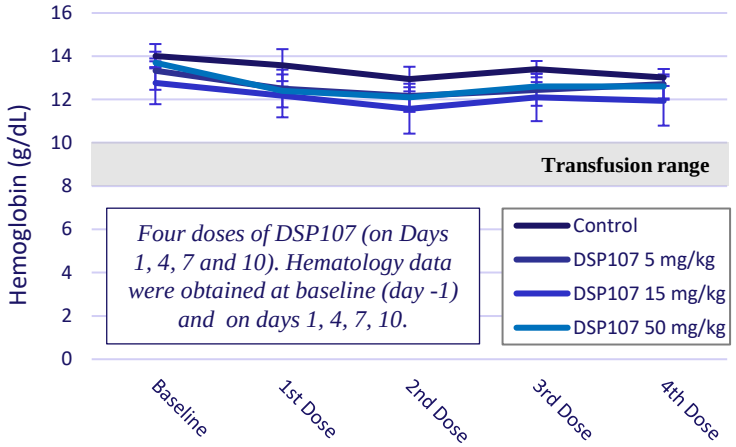


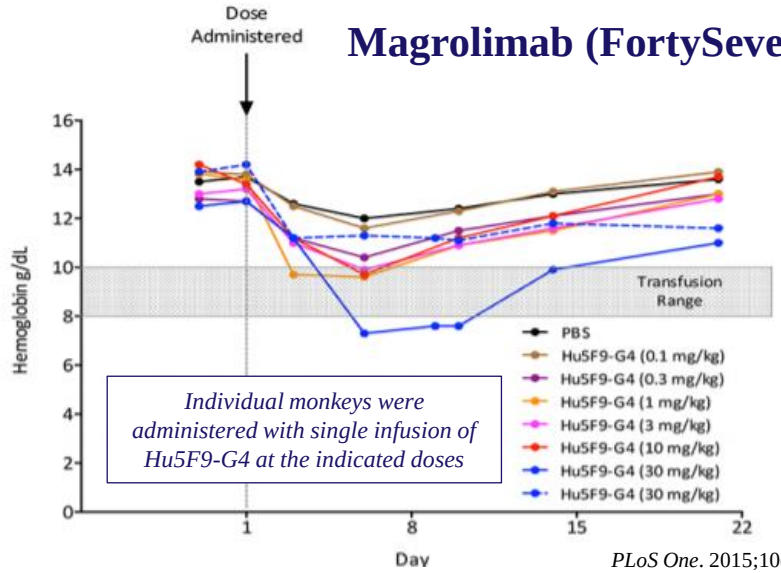


BETTER SAFETY COMPARED TO OTHER CD47 AGENTS

Reported cynomolgus monkey's safety data

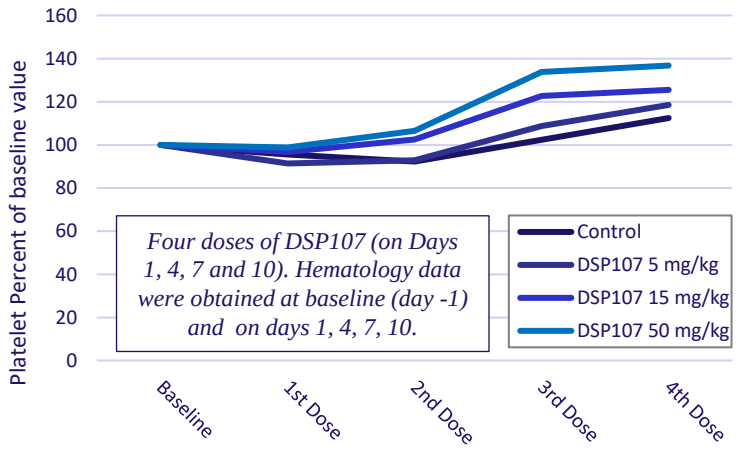
Hemoglobin

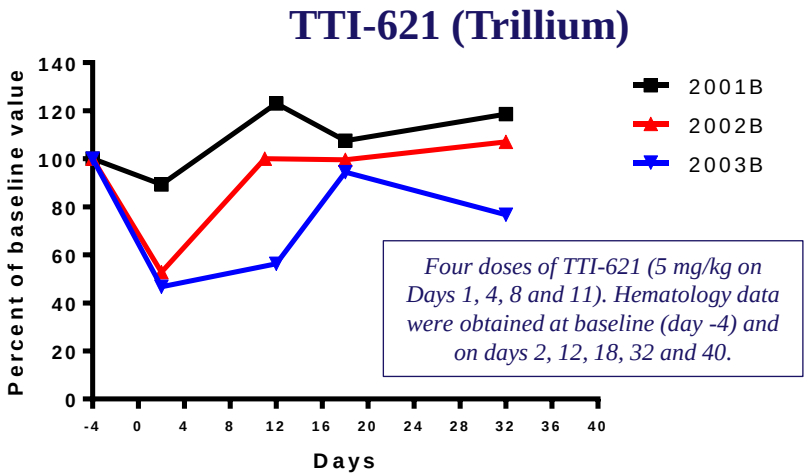




PLoS One. 2015;10(9):e0137345.

Platelets





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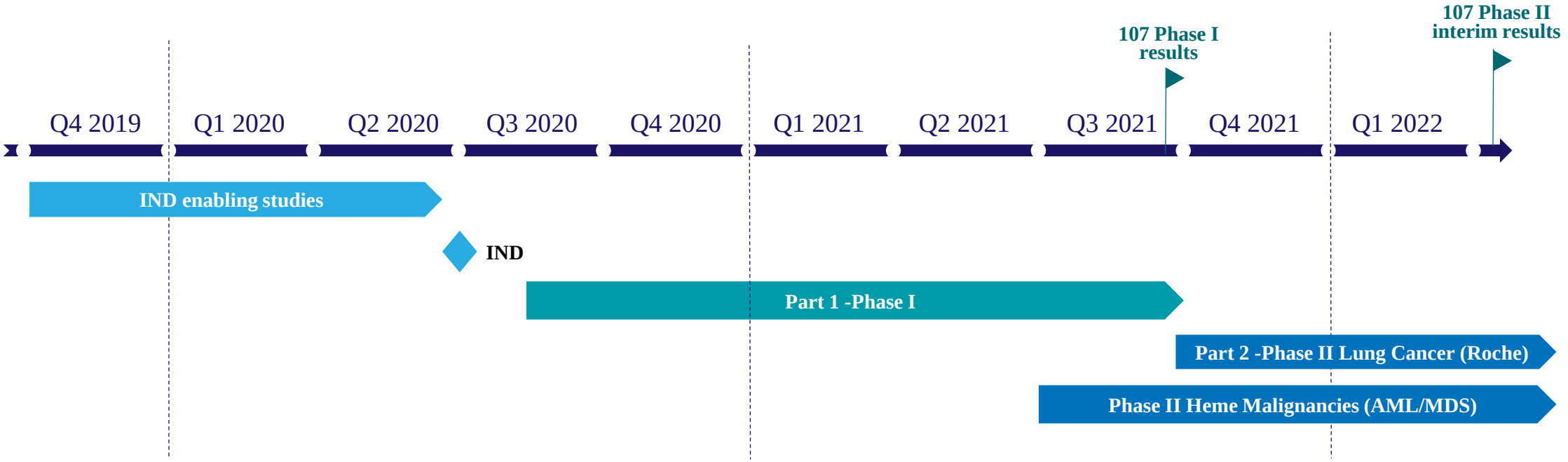
CLINICAL DEVELOPMENT PLAN



Two Phase II studies to commence H2/2021:

- 2L NSCLC patients who progressed on PD1/PD-L1 therapies to evaluate safety and efficacy of DSP107 monotherapy and when combined with Atezolizumab
- High risk MDS/AML patients to evaluate safety and efficacy of DSP107 either as monotherapy or when combined with azacitidine

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PHASE I/II STUDY DESIGN

PART I — Dose escalation study

DSP107 administered as monotherapy and in combination with Atezolizumab

Dosing regimen - iv administration once weekly

Population (N=~45) - patients with advanced solid tumors not suitable for curative therapy and without approved treatment options

Accelerated dose escalation in single patient cohorts until pre determined PK, PD or safety signals observed, followed by standard 3+3 design

PART II — Expansion cohort

Dose selection based on safety results from part 1

Single expansion cohort comparing DSP107 monotherapy to combination with Atezolizumab in patients with NSCLC who progressed after PD-1/PD-L1 targeting agents (N=~70 patients)

CLINICAL COLLABORATION WITH ROCHE

KAHR and Roche entered clinical collaboration agreement to evaluate DSP107 in combination with Atezolizumab in Advanced Lung Cancer patients

Roche

Study will evaluate the potential of DSP107 and Atezolizumab (PD-L1 inhibitor) in NSCLC patients who have progressed following first line treatment with PD1/PD-L1 inhibitors

Patient enrollment expected to commence in H2/2021

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PLANNED **DSP107 PHASE I/II AML/MDS** STUDY DESIGN
(COLLABORATION WITH MDACC)

PART I — Dose escalation study

DSP107 administered as monotherapy (treatment cycle 1) and in combination with Azacitidine (treatment cycle 2)

DSP107 dosing regimen - iv administration once weekly

Population (N=18-36) – patients R/R with AML or MDS/CMML who have failed up to 2 prior therapeutic regimes

Dose selection based on solid tumor RP2D, dose level -1 RP2D, and dose level +1 for DSP107

PART II — Expansion cohorts

Dose and regime (monotherapy or azacitidine combination) selection based on safety and efficacy results from part 1

Three expansion cohorts with Simon 2-stage design and clear "go/no-go" decisions in patients with:

Cohort I - **FRONTLINE AML** (N=10+10)

Cohort II – **FRONTLINE MDS/CMML** (N=10+10)

Cohort III – **R/R MDS/CMML** (N=10+10)

Endpoints

Safety – maximum tolerated dose, DLTs, RP2D of DSP107 monotherapy and Combination with azacitidine

Efficacy – (1) Overall response rate (CR+CRi+PR) within 3 months
(2) PFS (Time to next treatment), EFS and OS
(3) Exploratory biomarkers – 81-gene mutational profiling at MDACC, MRD by flow and NGS, CYTOF (Mass cytometry) customized panel for macrophages and T-cells in AML, TCR repertoire (Adaptive).

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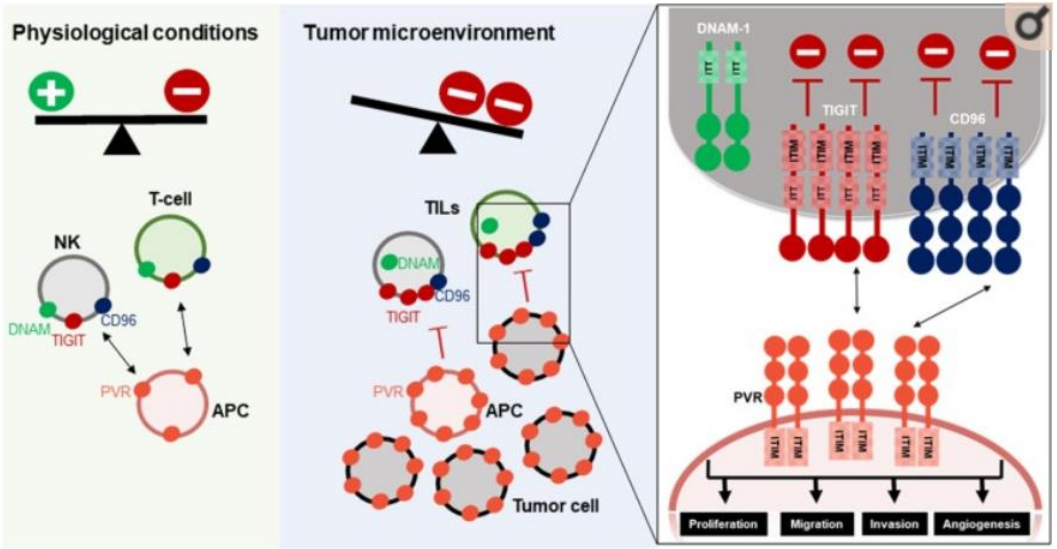
PRECLINICAL PIPELINE

DSP502 (DSP-Fc)

TIGIT (PVR) – BACKGROUND

- PVR is the ligand of TIGIT, CD96 and DNAM1
- Under physiological conditions, PVR is expressed at low levels and limited to certain cell types
- Balance between activating and inhibitory PVR mediated signals maintains normal function of immune cells
- In the tumor microenvironment PVR is dramatically overexpressed and acts in trans to reduce DNAM1 protein levels
- Balance between immune activating and inhibitory signals is often disturbed in the TME: inhibitory receptors (TIGIT and CD96) are upregulated while activating receptor (DNAM1) is downregulated
- PVR blockade by mAb or genetic ablation increases DNAM1 in both NK and T cells in vivo



[1] Brlić et al. Cellular & molecular immunology (2019) 16, 40–52.

[2] Li, X. et al. The Journal of clinical investigation (2018) 128, 2613–2625.

[3] Stengel at al. PNAS (2012) 109 , 5399-5404.

DSP502- TIGIT–PD1 – UNIQUE CONCEPT

- KAHR's TIGIT based product **targets PVR and PD-L1 on tumors**
- PVR blockade provides dual stimulatory mechanism to enhance anti-tumor immunity and promote DNAM1 costimulatory signaling for effective anti-tumor immunity
- High PVR expression was recently found to predict non-responders to PD1 therapy in NSCLC patients (with 100% accuracy)[1] and in melanoma[2]
- **Inhibition of TIGIT/PVR pathway in clinical studies shows efficacy only when combined with PD-1 blockade**

MoA	**PVR targeting** (KAHR's approach)	**TIGIT Ab** (Competitors)
Inhibit TIGIT signaling	✓	✓
Inhibit CD96 signaling	✓	–
Increase DNAM1 surface expression and signaling	✓	–

[1] *Lee et al. JCI insight* (2020). 10.1172
[2] *Lepletier et al. Clinical Cancer Research* (2020)

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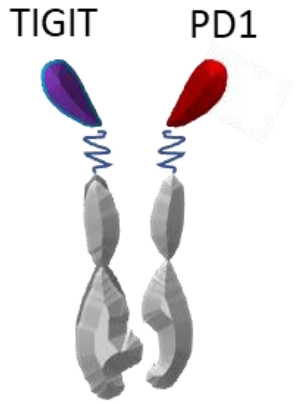
DSP502 – TIGIT-PD1 – SCIENTIFIC RATIONALE



Unique concept

- Enhancing tumor targeting through dual checkpoint inhibition
- Tumor selectivity by dual binding relaying on avidity (‘AND GATE’)
- Multiple activities to overcome resistance pathways affecting both TIGIT/PVR and PD/PD-L1 to activate effector T and NK cells and reduce Treg cells
- DSP502 is based on an IgG1 and expected to have Fc-mediated ADCC activity
- Inhibition of TIGIT/PVR pathway in clinical studies shows efficacy only when combined with PD-1 blockade

Mechanism of Action – Targeting PVR and PD-L1

- **TIGIT** will block endogenous **PVR** on cancer cells unleashing the activity of the adaptive immune system to attack the tumor cells
- **PD1** will block **PD-L1** on tumor cells to activate effector T cells and increase tumor specificity

DSP216 (DSP-Fc)

LILRB2(ILT4)/HLA-G – BACKGROUND

- Leukocyte Immunoglobulin Like Receptor B (LILRB; ILT) are immune checkpoint proteins expressed on macrophages and other myeloid cells
- HLA-G, the main ligand for LILRB1 (ILT2) and LILRB2 (ILT4), is a critical protein responsible for immunotolerance in placenta preventing the mother's immune system from destroying the fetus
- By over-expressing HLA-G, tumor cells use the same escape mechanism to evade immune surveillance
- HLA-G expression is associated with cancer immune evasion, disease progression and poor prognosis

- HLA-G serves as a broad-range Immune Checkpoint protein which:
  - inhibits all immune cell subsets including macrophages, NK, B and APCs, as well as T cells
  - recruits suppressive immune cells inducing an immunosuppressive microenvironment for tumors

DSP216 - LILRB2-SIRPA – UNIQUE CONCEPT

- KAHR's LILRB based products target HLA-G expressed exclusively on tumors
- HLA-G blockade will interfere with both LILRB1 and LILRB2 binding to avoid redundancy compensation
- HLA-G blockade activates both innate (macrophages) and adaptive (T cells) immune systems

MoA	**HLA-G targeting** (KAHR's approach)	**LILRB1/2 Ab** (Competitors)
Inhibit both LILRB1 and LILRB2	✔	—
Tumor selectivity (HLA-G expression)	✔	—
Activates both innate (macrophages) and adaptive (T cells)	✔	✔

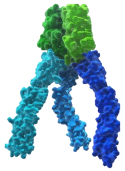
DSP216 – LILRB2-SIRPA – SCIENTIFIC RATIONALE

Unique concept

- Enhancing tumor targeting through dual checkpoint inhibition
- Tumor selectivity by dual binding relaying on avidity (‘AND GATE’)
- Multiple activities to overcome resistance pathways:
  - Affecting both LILRB1 and LILRB2 to avoid redundancy compensation
  - Activating both innate (macrophages) and adaptive (T cells) immune systems
  - Blocking CD47 to trigger phagocytosis of tumor cells

Mechanism of Action – Targeting HLA-G and CD47

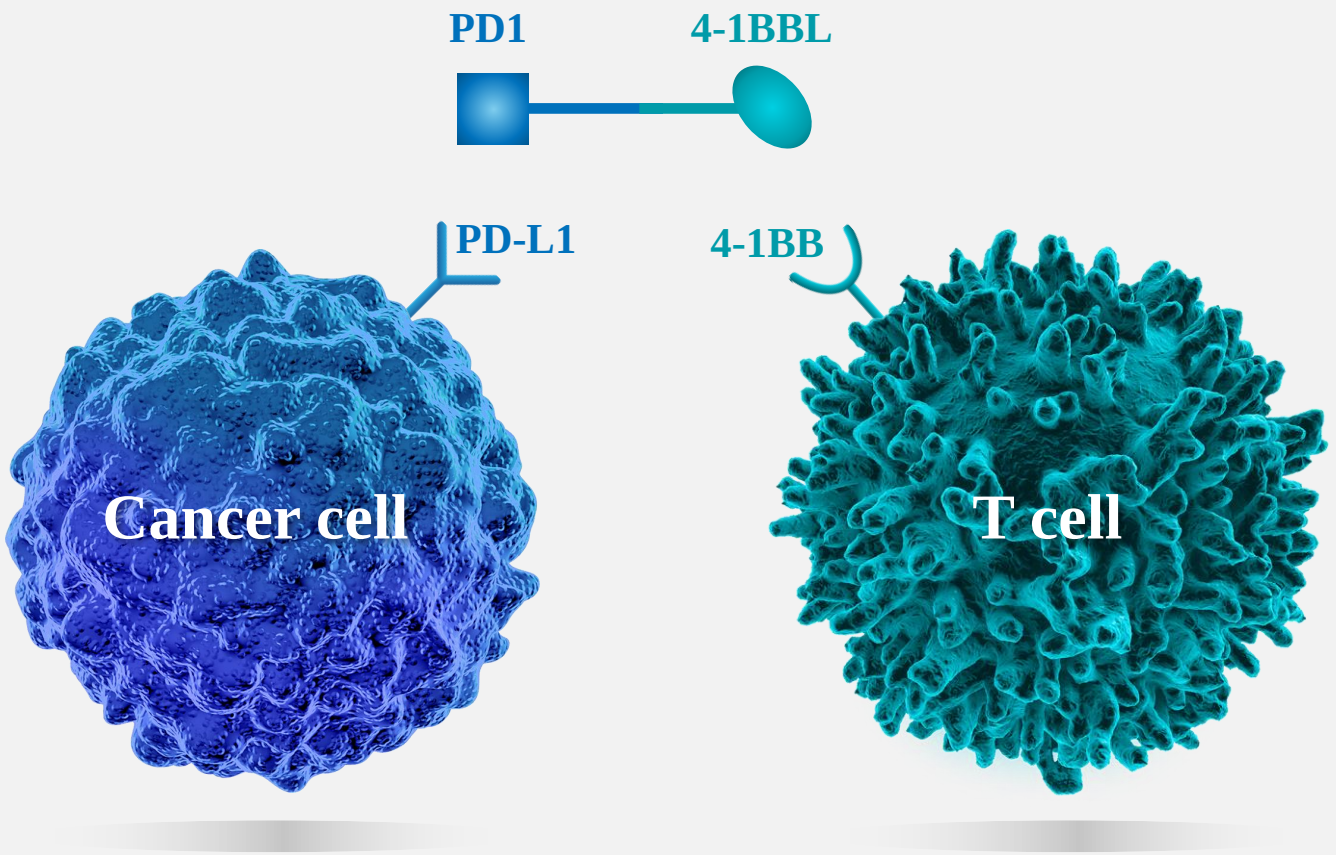
- **LILRB2 (ILT4)** will block over-expresses **HLA-G** on cancer cells unleashing the activity of innate and adaptive immune systems
- **SIRPα** will block **CD47** on tumor cells inhibiting ‘don’t eat me’ signals to activate macrophages



DSP105 (DSP)

DSP105 – PD1-41BBL

PD1 binds to **PD-L1** overexpressed on cancer cells disabling their inhibitory signal



4-1BBL side binds to **4-1BB** on tumor-antigen specific T cells, stimulating their expansion, cytokine production, and the development of cytolytic effector functions

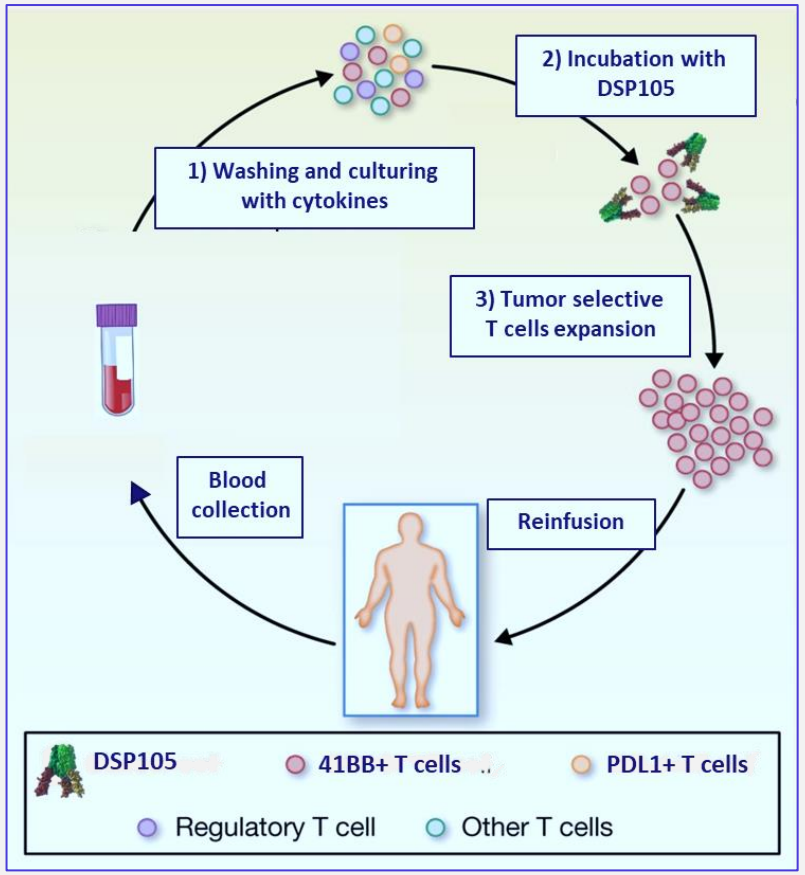
DSP105 – NOVEL ADOPTIVE T-CELL THERAPY

DSP Primed Tumor Reactive T-cells (DpT)

- Process utilizes PBMCs from standard blood sample collected from the patient
- Cells are washed and incubated with proprietary mix of clinical grade compliant cytokines
- 41BB positive T cells are enriched by incubation with DSP105
- Anti tumor specific T-cells are re-infused to patient

Value Proposition

- Derived from standard blood sample
- Does not require tumor biopsy or target identification
- Multi-antigen approach reducing relapse risk
- Simple, low cost and straightforward process (currently <14 days)



DSP105 – NEXT GENERATION CELL THERAPY APPROACH

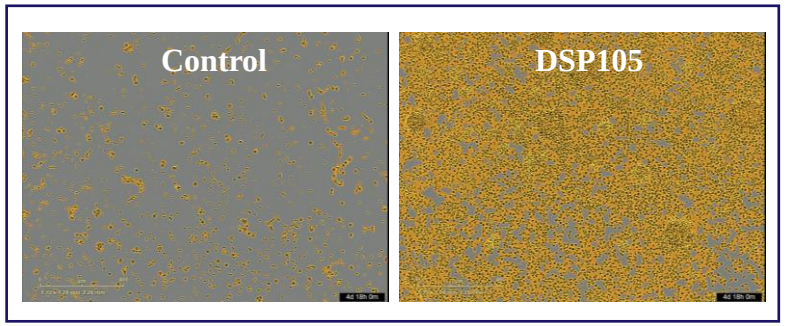
Unique value proposition

Simplicity
Does not require gene modification or tumor biopsy

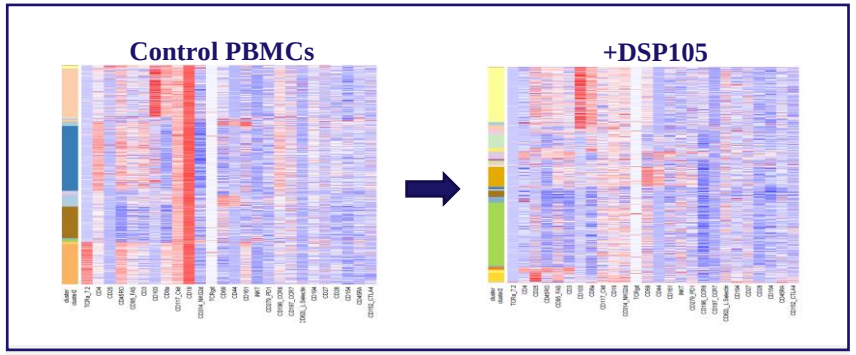
Excellent safety
Targeting patient-specific antigens expressed by the tumor

Solid tumors opportunity
Multi antigen approach reducing risk for relapse

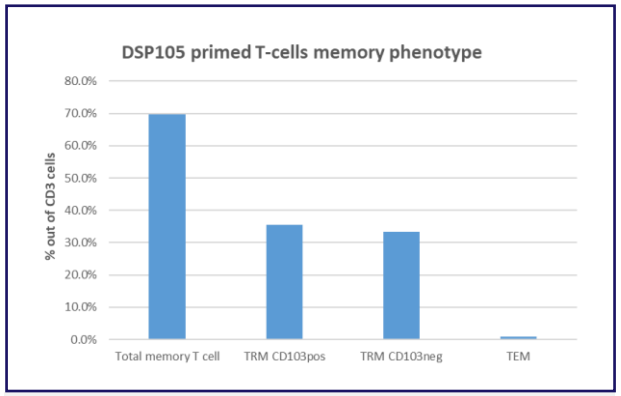
Combination of unique features



Selectively activate and expand the low percentage of tumor reactive 41BB positive T-cells



DSP105 in the DpT process results in clonal expansion of tumor reactive T cell clones



The DpT selected T cells have a memory phenotype and are Phenotypically Similar to tumor primed T-cells

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Pre-clincial development ongoing; IND by H1/2022

THANK YOU!