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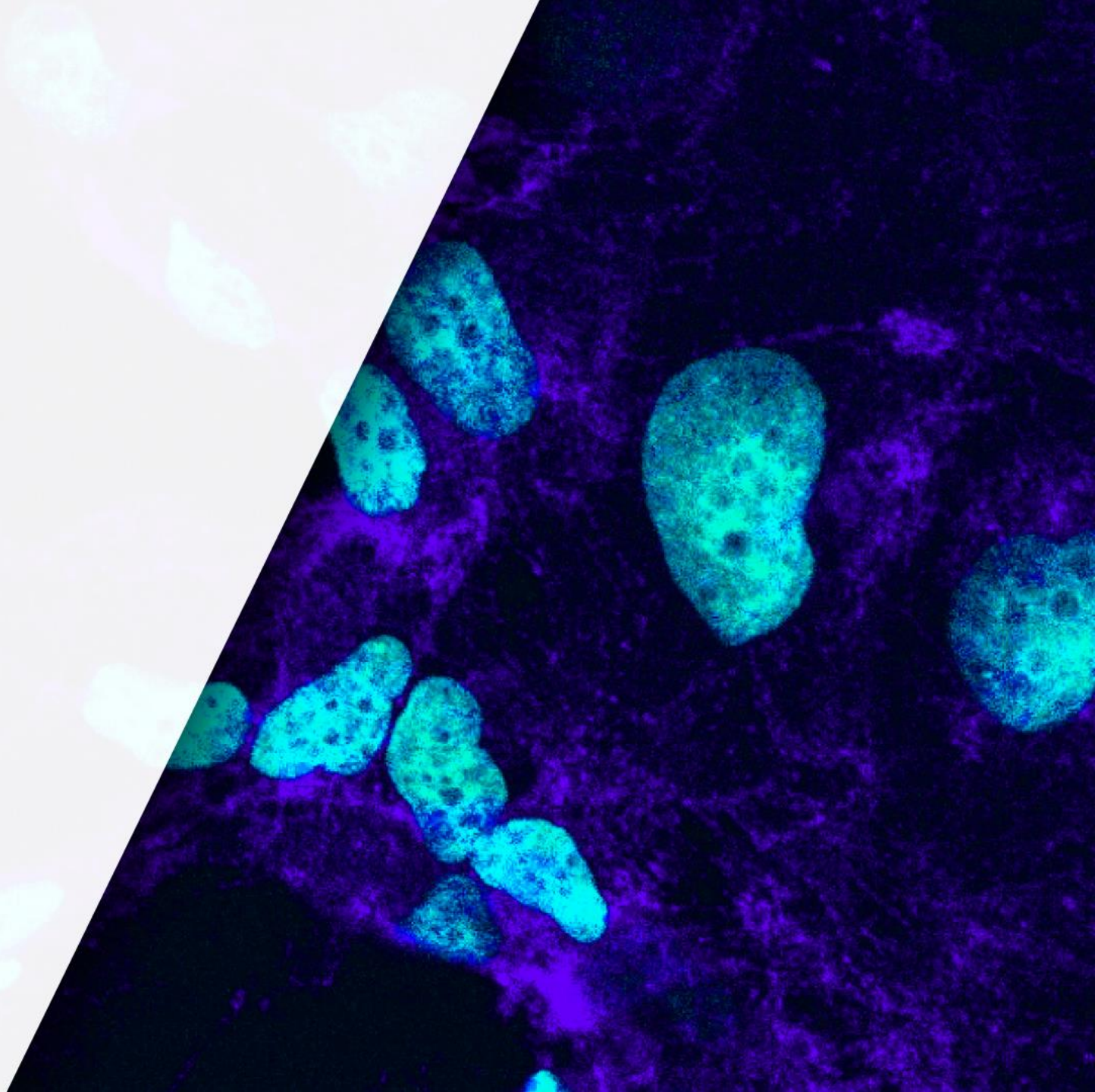
UNMASKING CANCER CELL CAMOUFLAGE

and activating a targeted immune response

COMPANY PRESENTATION | SEPT. 2020



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 product | IND Q4 2021
-Multiple future candidates in R&D

HUGE MARKET
Immuno-therapeutics
$56.5B by 2025

IP
12 families
2 granted (US and other territories),
10 pending (worldwide)

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

Ayelet Chajut, PhD
CTO
Quantomics · Pluristem · ROSETTA GENOMICS · Quark Pharmaceuticals

Oren Gez, MBA
VP Strategy & Corporate Dev.
BARCLAYS · ING · MEITAV DASH

Iris Pecker, PhD
VP CMC
InSight Biopharmaceutical

Rinat Tabakman, PhD
VP Development
BIOLINERX · XTLbio

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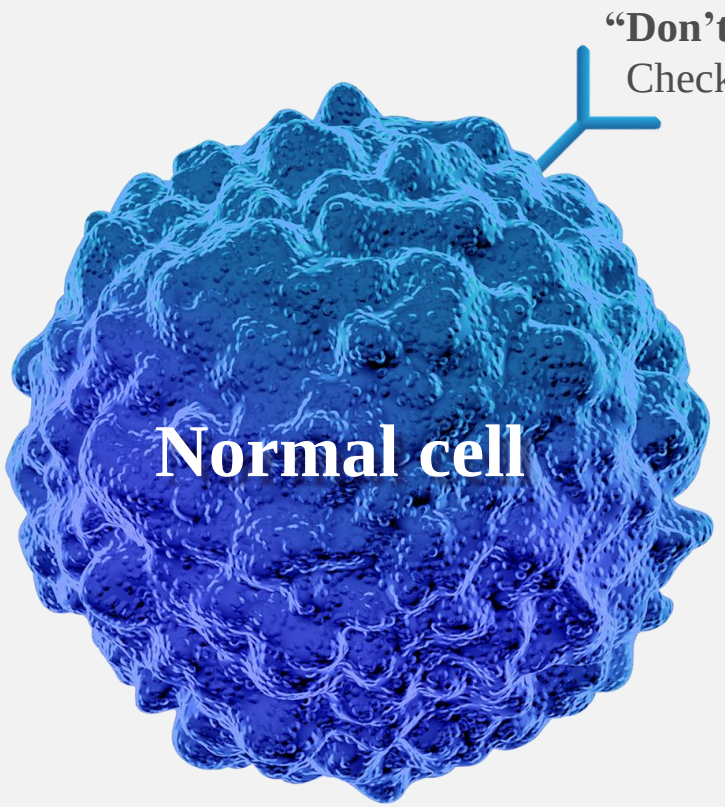
KAHR technology inventor; BOD Observer; Provost Jefferson Thomas University

Bernhard Kirschbaum, PhD
Board consultant; ex-EVP, Global Research & Early Development at Merck Serono

Ezra Cohen, M.D.
SAB member; Director San Diego Center for Precision Immunotherapy

Manuel Hidalgo, M.D., Ph.D
SAB member; Chief Division of Hematology and Medical Oncology, Weill Cornell;

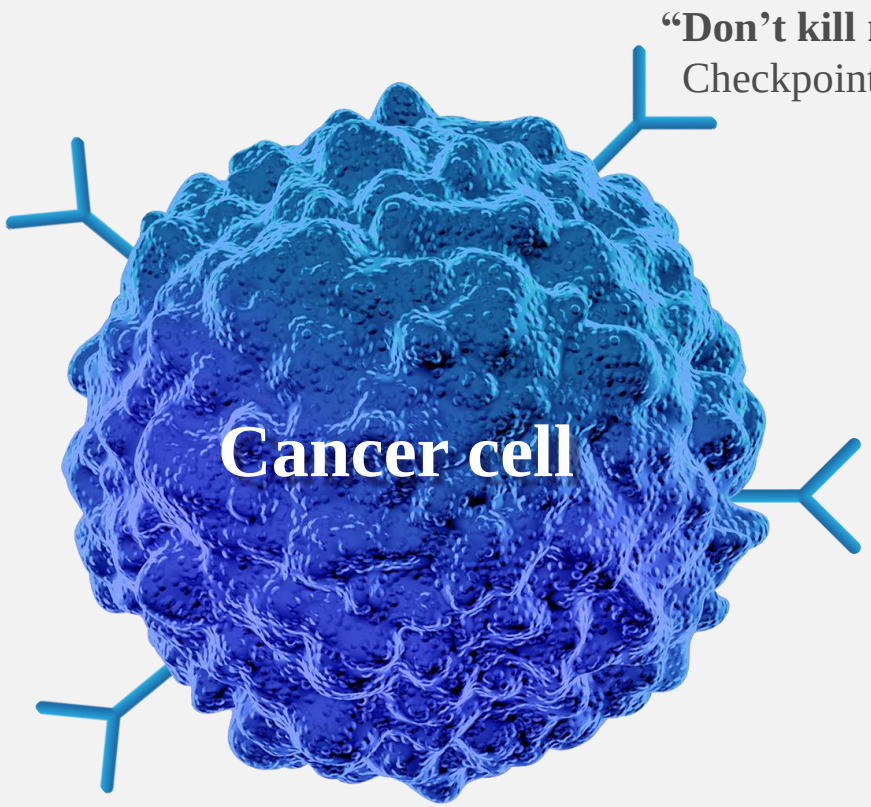
THE CAMOUFLAGE CHALLENGE IN TREATING CANCER



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	

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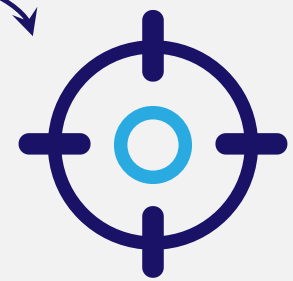
EFFECTIVELY TREATING CANCER REQUIRES A MULTIFACETED APPROACH

Selectively disabling its defenses

WHILE



Recruiting a local targeted immune attack

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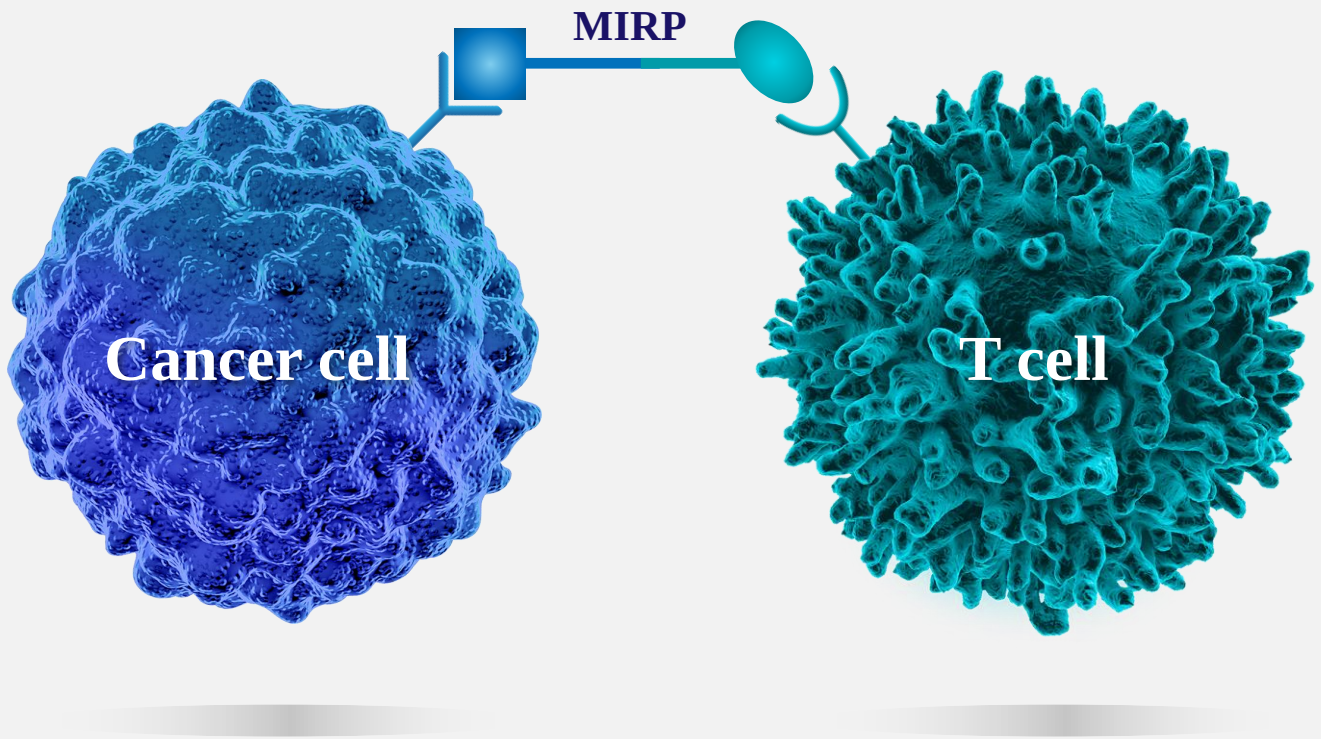
KAHR develops smart immuno-recruitment cancer drugs that activate a targeted immune response by converting cancer camouflage into beacons for the immune system to attack

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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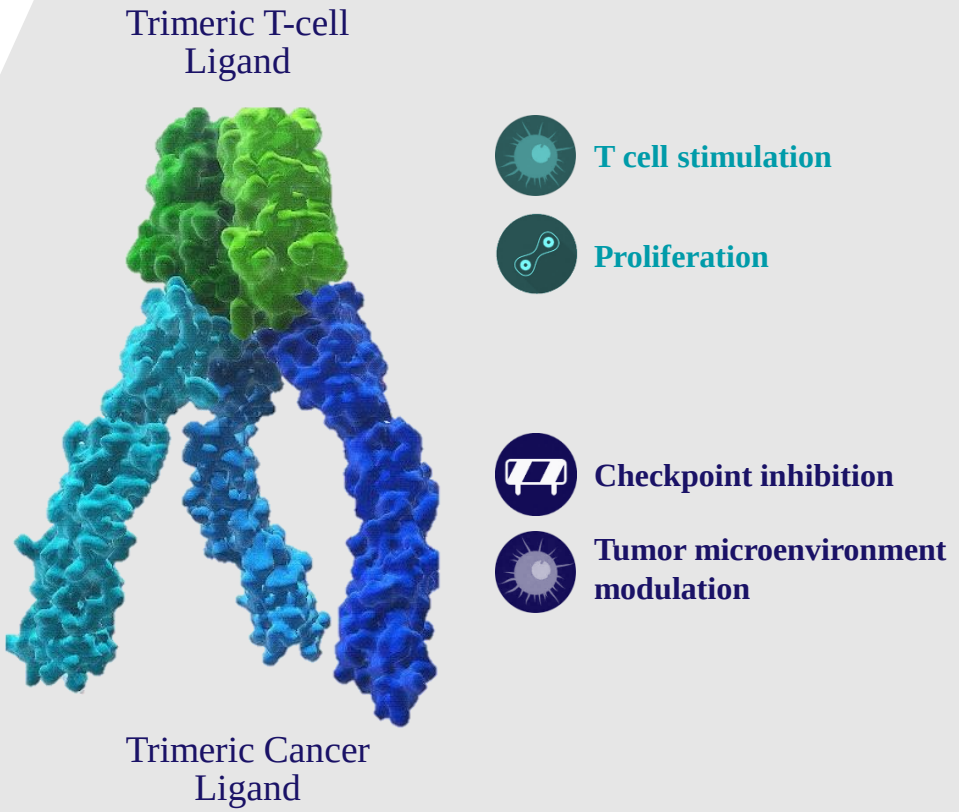
UNIQUE TRIMERIC STRUCTURES ENABLE SPECIFICITY AND SELECTIVITY

Trimeric ligand ends enable both:

- High tissue specificity, by binding only to overexpressed checkpoint molecules
- Selective activation of adaptive immunity by binding local T-cells

MIRP Structure



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HOW IT WORKS

Targeting checkpoint overexpression

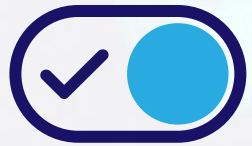
MIRPs utilize cancer cell overexpression of checkpoint antigens to selectively target and bind to the cancer

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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A UNIQUE COMBINATION OF FEATURES

Multi-specific targeting for synergistic immune effect

Trimeric binding structures for optimal activation and increased affinity

Overexpressed checkpoint binding for cancer site specificity

Local T-cell recruitment to avoid systemic toxicity

Differentiated PK/PD relationships for wider therapeutic window

Suited for solid and hematological malignancies

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PIPELINE

	Discovery	Preclinical	Clinical	Collaborators
DSP107 Bi-functional Protein SIRPα-41BBL Activating both, innate and adaptive immunity	IND cleared; Phase I/II Solid Tumors commence Q3/2020			Roche Genentech A Member of the Roche Group
DSP105 Bi-functional Protein PD1-41BBL Releasing the brakes while pushing the accelerator of effector T cells	IND ready by Q4/2021			Beth Israel Deaconess Medical Center
TSP Tri-functional Protein TIGIT/LILRB /SIGLEC Enhancing tumor targeting through dual type checkpoint binding & immune stimulation	12 Programs			Jefferson Philadelphia University + Thomas Jefferson University HOME OF SIDNEY KIMMEL MEDICAL COLLEGE

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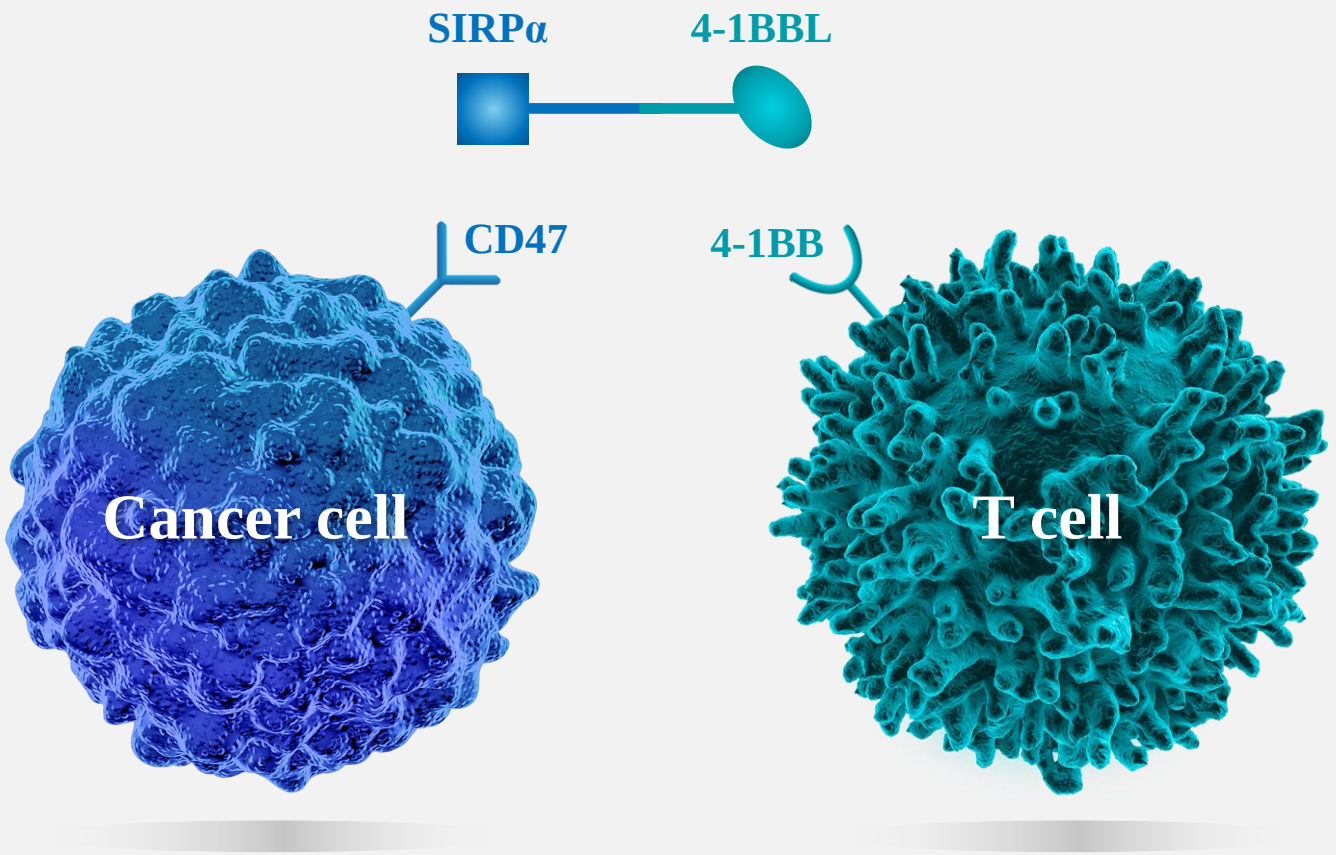
LEAD MIRP PRODUCT – **DSP107**

DSP107 (MIRP type - Dual Signaling Protein)

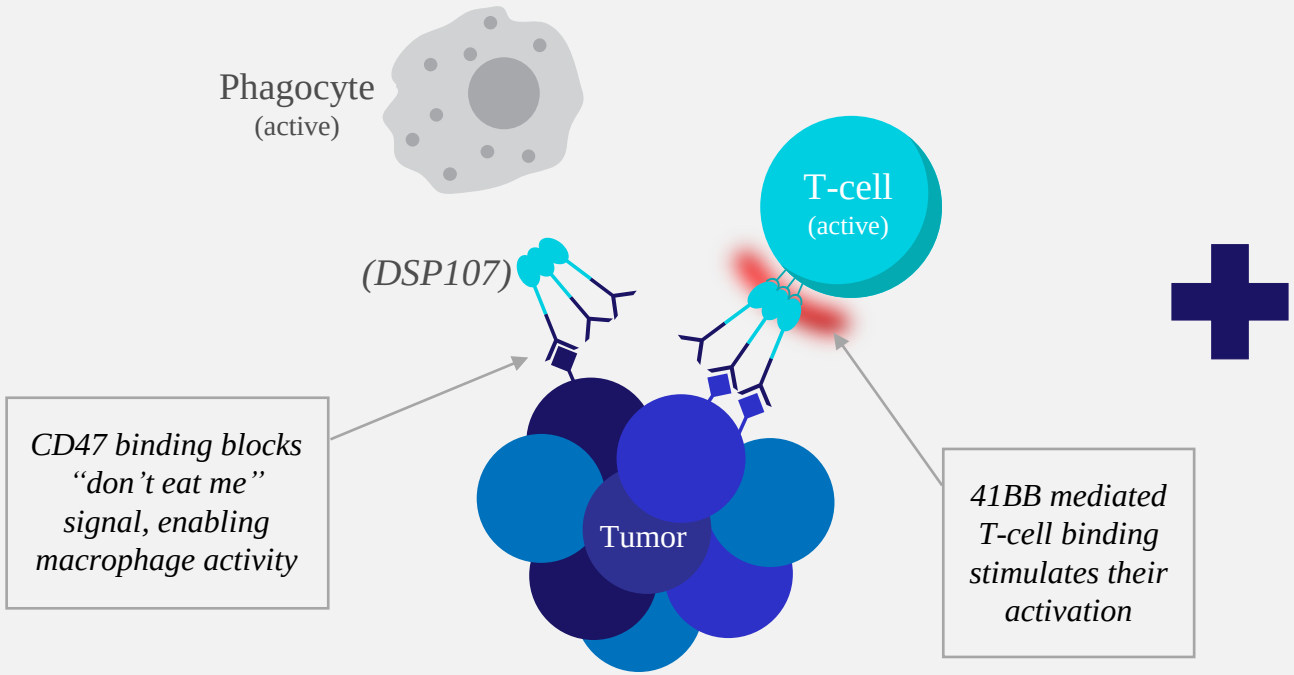
SIRPα binds to **CD47** overexpressed on cancer cells, disabling their "don't eat me" signal



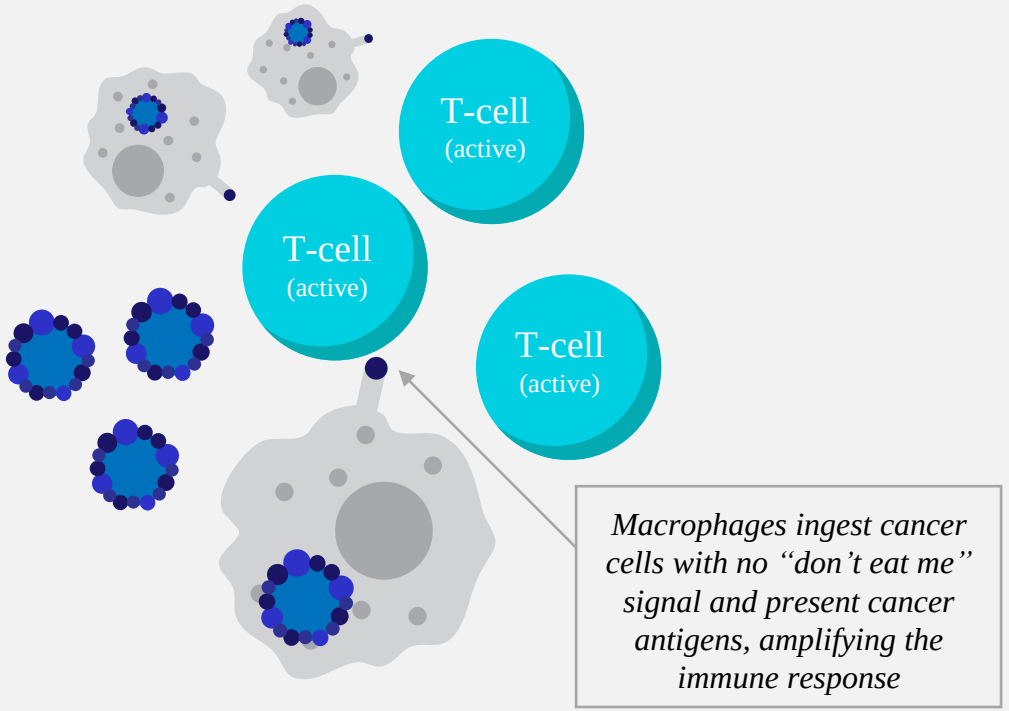
4-1BBL side binds to **4-1BB** on tumor-antigen specific T cells, stimulating their expansion, cytokine production, and the development of cytolytic effector functions

DSP107 – SYNERGISTIC IMMUNE ACTIVATION

Innate immunity enabled + mediated activation of adaptive immunity



Natural adaptive immune response



DSP107 – BEST IN CLASS CD47 TARGETING COMPOUND

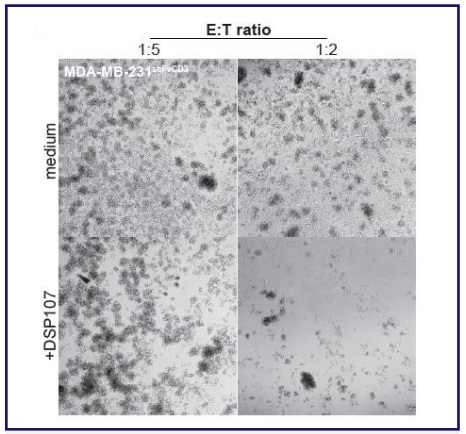
Next generation capabilities

Dual MOA
activates innate and adaptive immunity

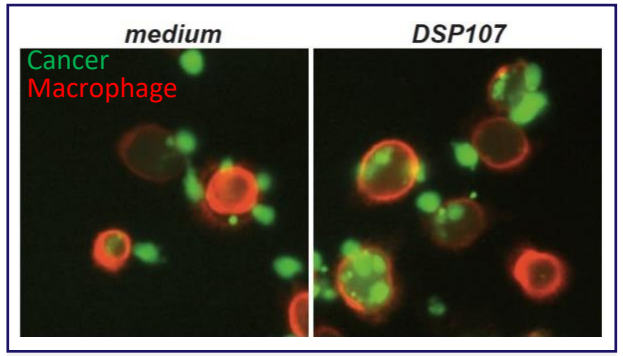
Excellent safety
without hematological toxicities

Strongly positioned
for treatment of solid and hematological malignancies

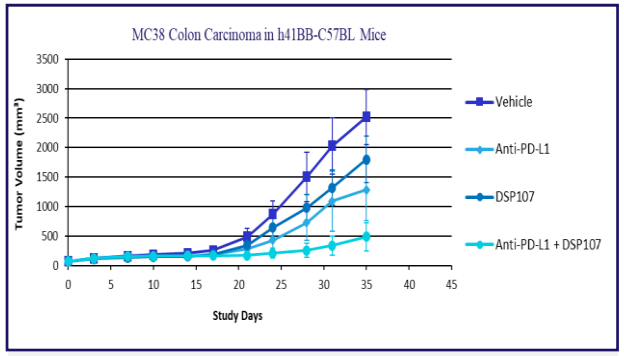
Unique synergistic effects



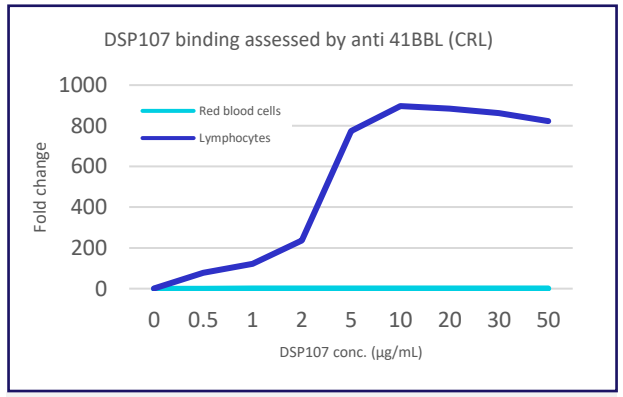
Activates T cells to secrete IFN-γ and augment their cancer cell killing potential



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

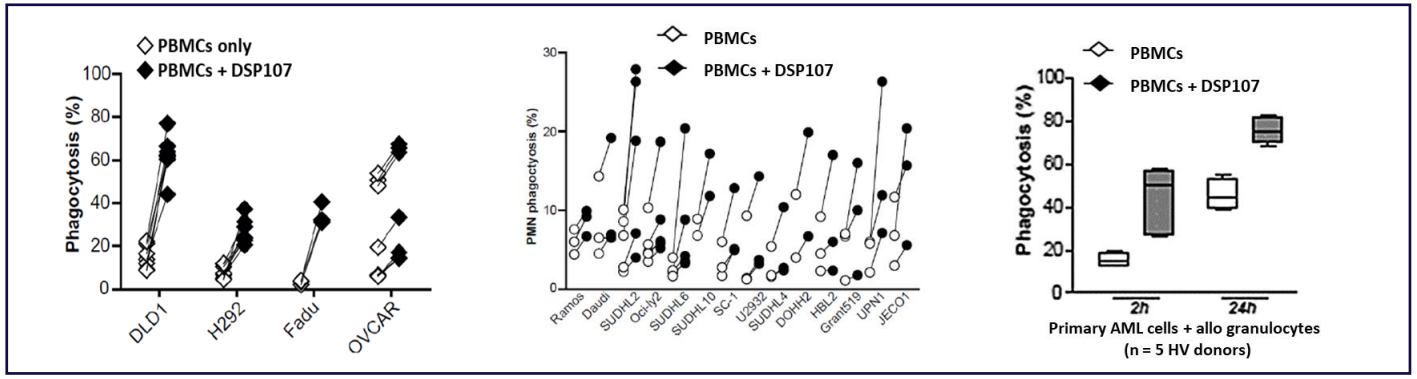


Does not bind red blood cells, avoiding antigen sink issues, resulting in a best-in-class safety profile

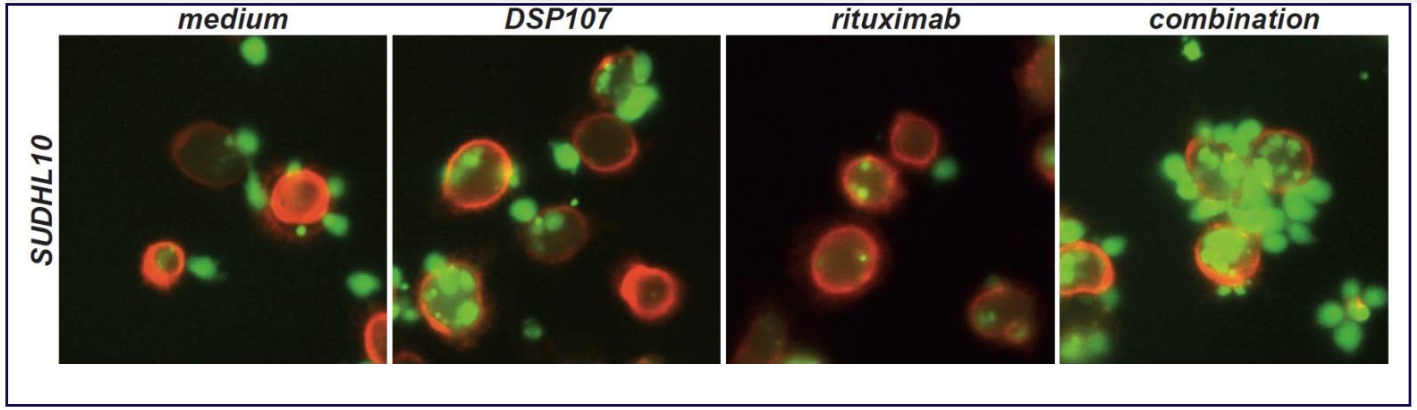
DSP107 - PRE-CLINICAL OVERVIEW

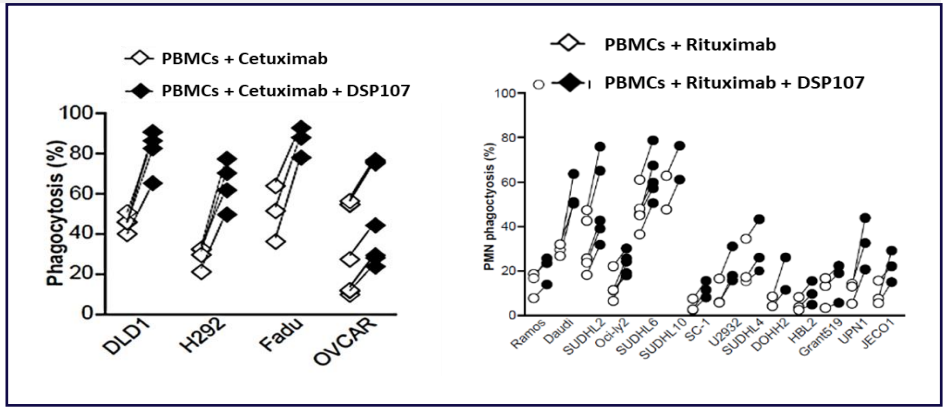
SIRPα – BINDS TUMOR AND INDUCES PHAGOCYTOSIS

Triggers cancer cell death by phagocytosis as a single agent



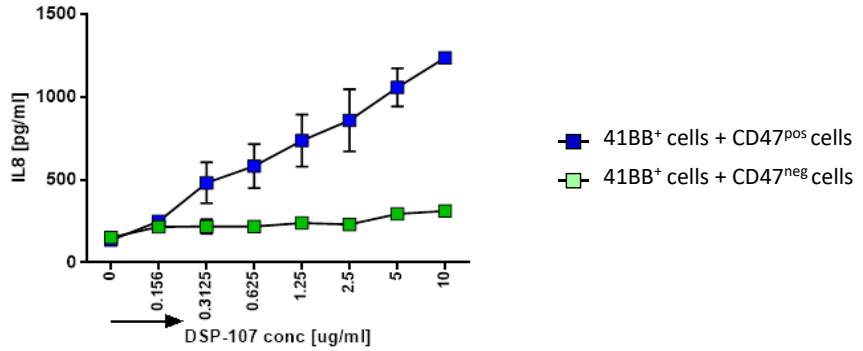
Augments mAb's ADCP-mediated phagocytosis of cancer cells



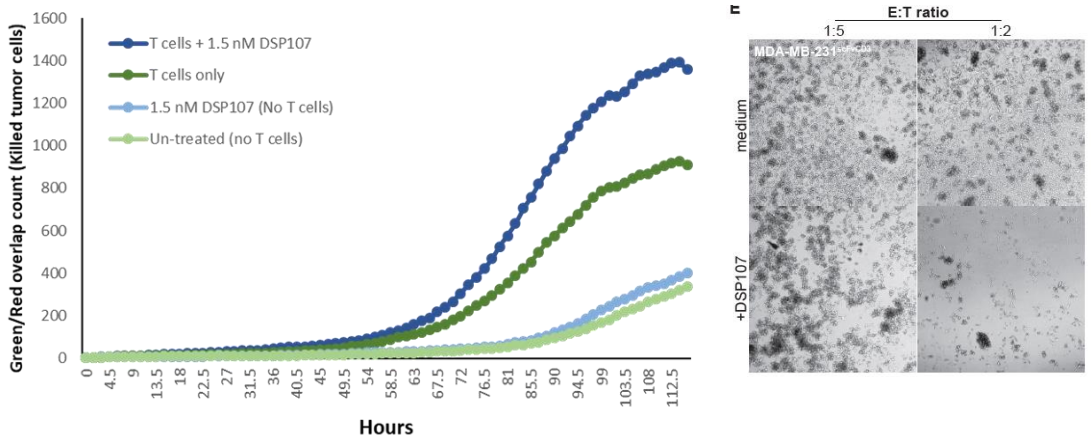


41BBL – ACTIVATES T-CELLS

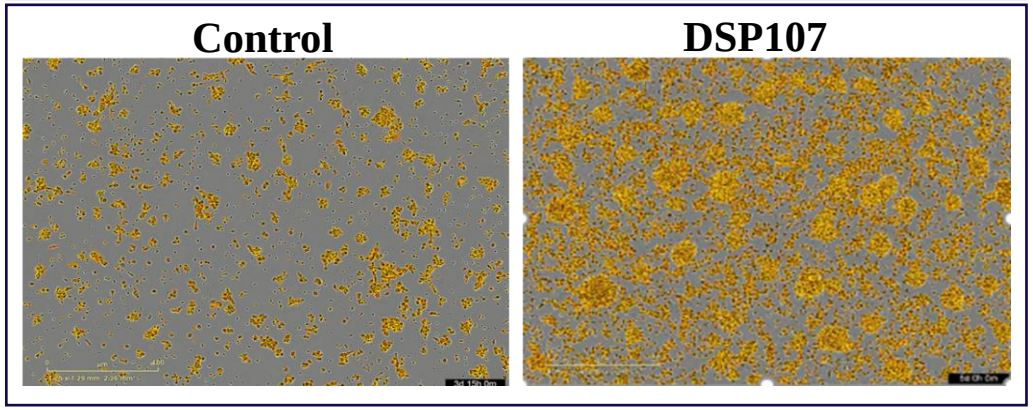
Tumor selective cross presentation activates 41BB signaling



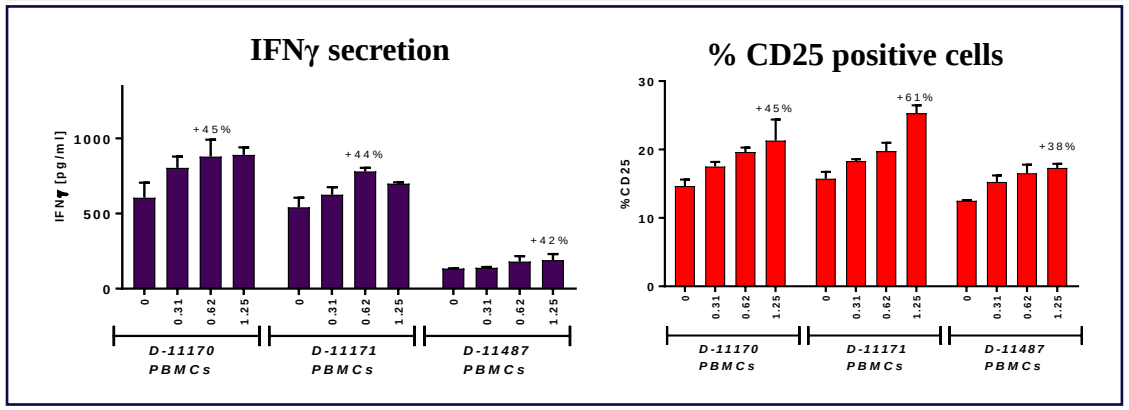
Induces T-cell killing potential against cancer cells



Augments T-cell proliferation

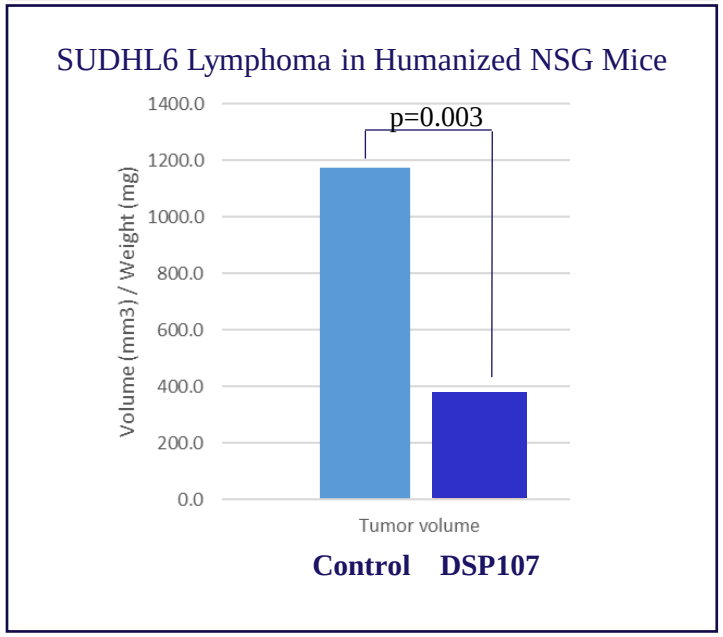


Activates T cells and increases IFNγ secretion

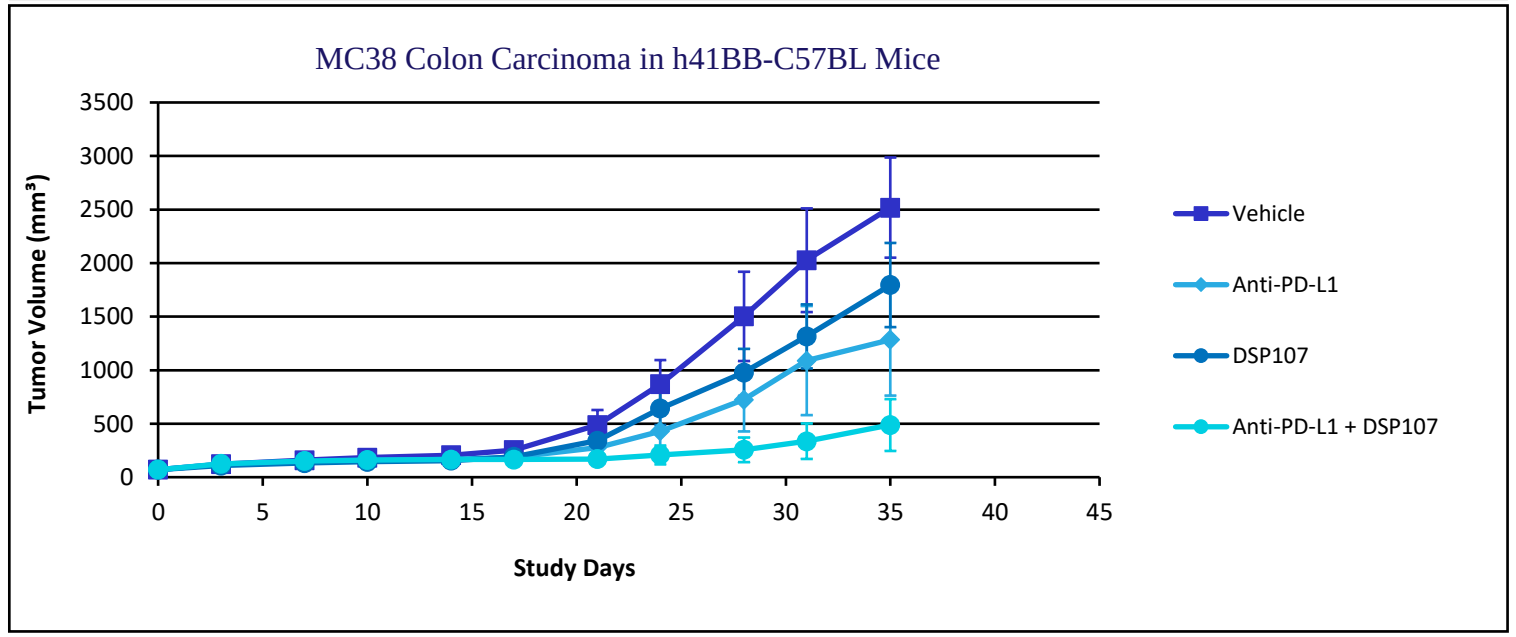


POTENT IN VIVO EFFICACY

Shows strong anti tumor activity as a single agent



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

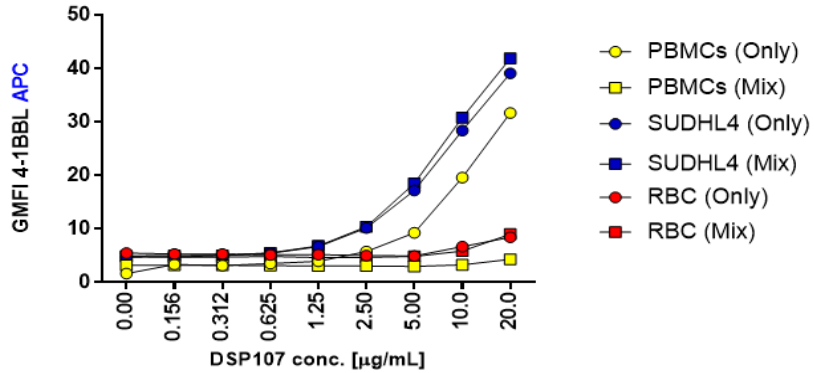


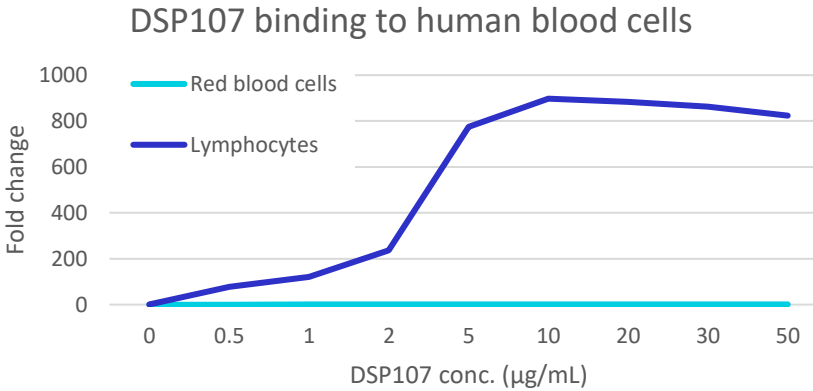
EXCELLENT SAFETY - NO HEMATOLOGICAL TOXICITIES

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 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

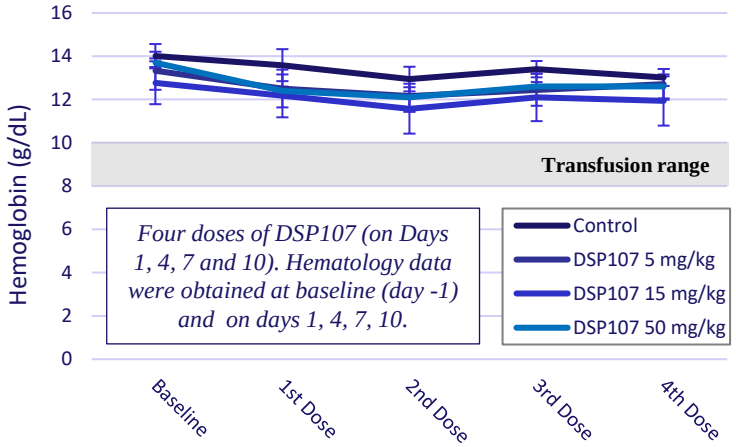


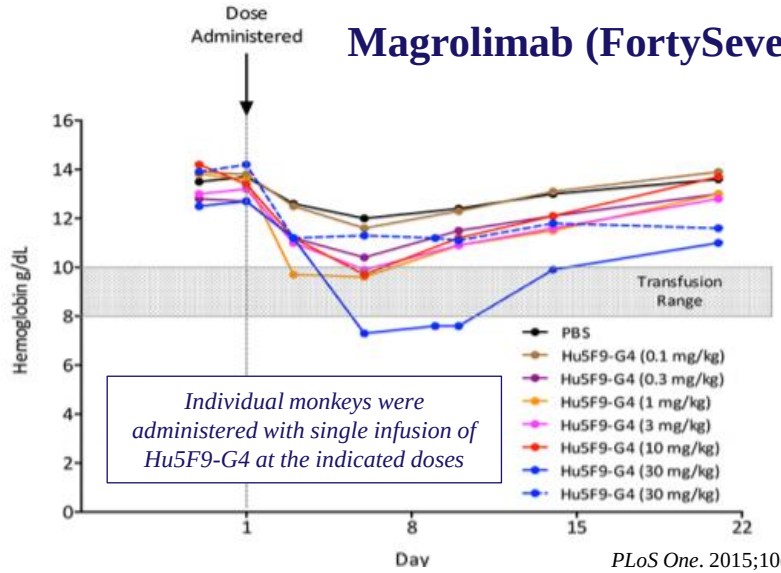


BETTER SAFETY COMPARED TO OTHER CD47 AGENTS

Reported cynomolgus monkey's safety data

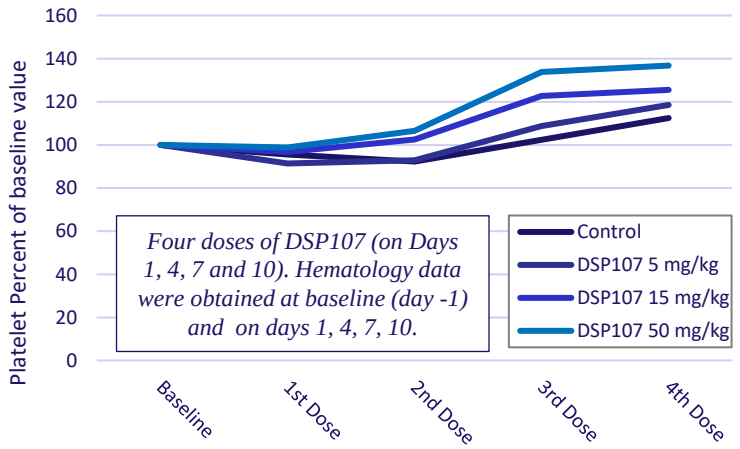
Hemoglobin

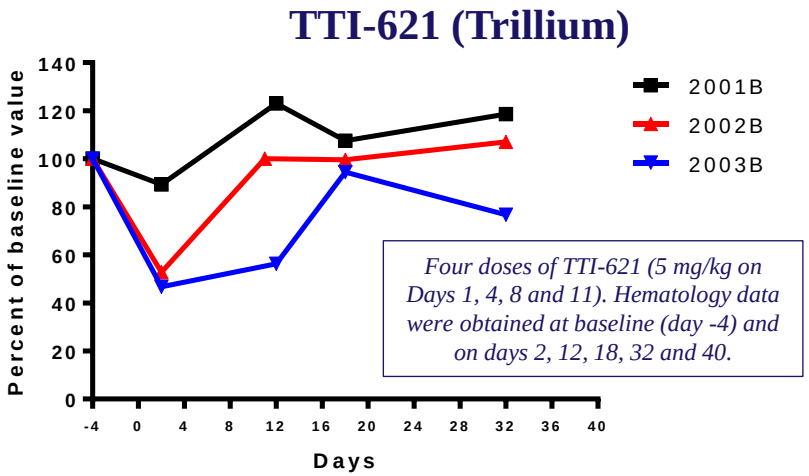




PLoS One. 2015;10(9):e0137345.

Platelets





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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)

Patient enrollment expected to commence in Q3/2020

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CLINICAL COLLABORATION WITH ROCHE

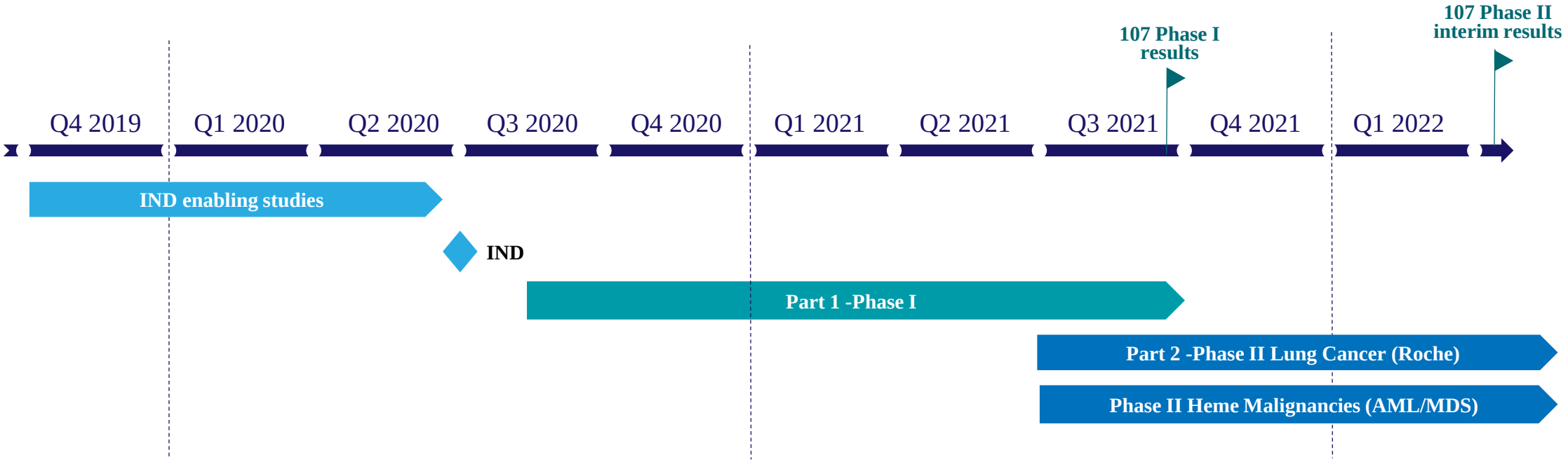
KAHR and Roche entered clinical collaboration agreement to evaluate DSP107 in combination with Atezolizumab in Advanced Lung Cancer patients

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

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 when combined with azacitidine and/or venetoclax

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THANK YOU!