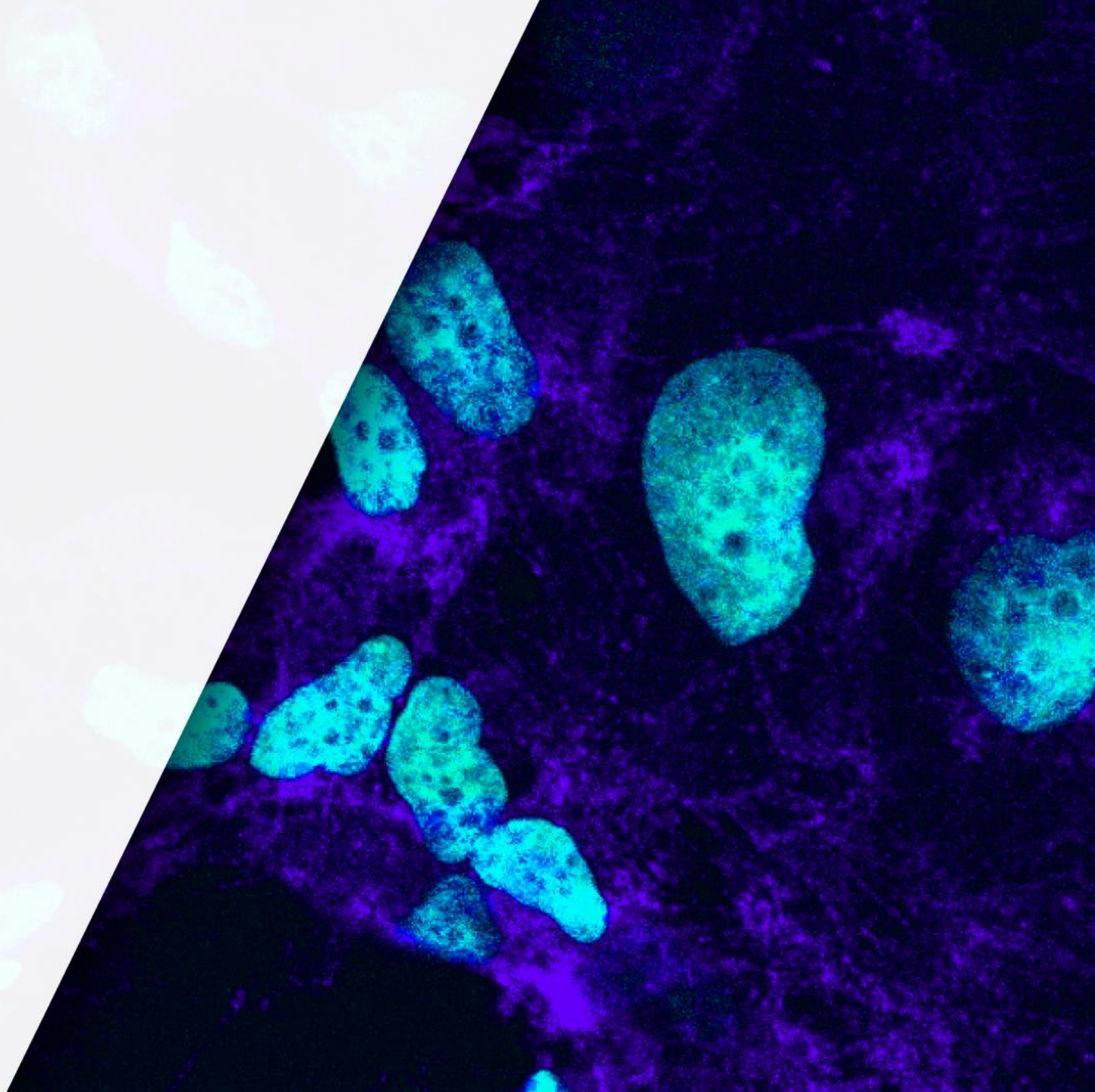
IMMUNITY.
RECRUITED.

UNMASKING CANCER CELL CAMOUFLAGE

COMPANY PRESENTATION | Oct. 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.

COMPANY HIGHLIGHTS

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

CURRENT STATUS
- Phase I/II studies for solid tumors and heme malignancies
- Collaboration with ROCHE to combine with Atezolizumab

PIPELINE
-1st product | Phase I/II CD47/41BB
-2nd & 3rd products | IND H1 2023
-Multiple future candidates in R&D

MARKET
Immuno-therapeutics
$56.5B by 2025

IP
15 families
3 granted (US and other territories),
12 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.

LEADERSHIP TEAM

Management

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

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

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

Tomer Cohen, MBA
CFO
LOCUST WALK · BARCLAYS · Goldman Sachs

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

Iris Pecker, PhD
VP CMC
InSight Biopharmaceutical

Rinat Tabakman, PhD
VP Development
BIOLINERX · XTLbio

Board of Directors

Aron Knickerbocker
Chairman of the Board

Thomas Eldered
Director

Gur Roshwalb
Director

Merav Kaye
Director

Carl-Johan Spak
Director

Tamar Raz
Director

Eyal Lifschitz
Director

Michel Habib
Director

Scientific and Clinical Advisory Board

Mark L. Tykocinski, M.D.
KAHR technology inventor; BOD Observer; Provost Jefferson Thomas University

Martin S. Tallman, MD
Chief Leukemia Service, Memorial Sloan Kettering Cancer Center

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

Hagop Kantarjian, M.D.
Chair Department of Leukemia at The University of Texas MD Anderson Cancer Center

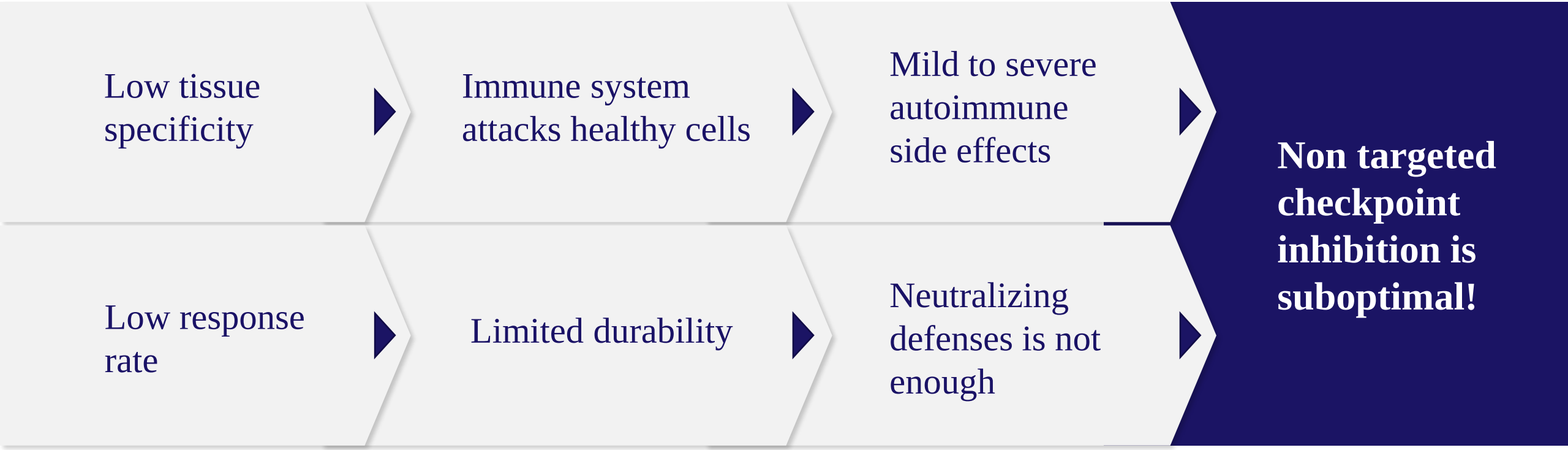
Edwin Bremer, PhD
Professor at the Translational Surgical Oncology at the University Medical Center Groningen

Samir Khelif, MD
Director, Loop Immuno-Oncology Research Lab, Georgetown Lombardi Comprehensive Cancer Center

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

IMMUNITY. RECRUITED.

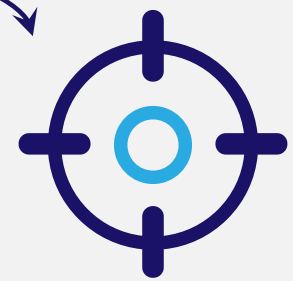
CURRENT CHECKPOINT IMMUNOTHERAPY HAS ITS DOWNSIDES



EFFECTIVELY TREATING CANCER REQUIRES A MULTIFACETED APPROACH

Selectively disabling cancer defense mechanism

WHILE



Recruiting a local targeted immune attack

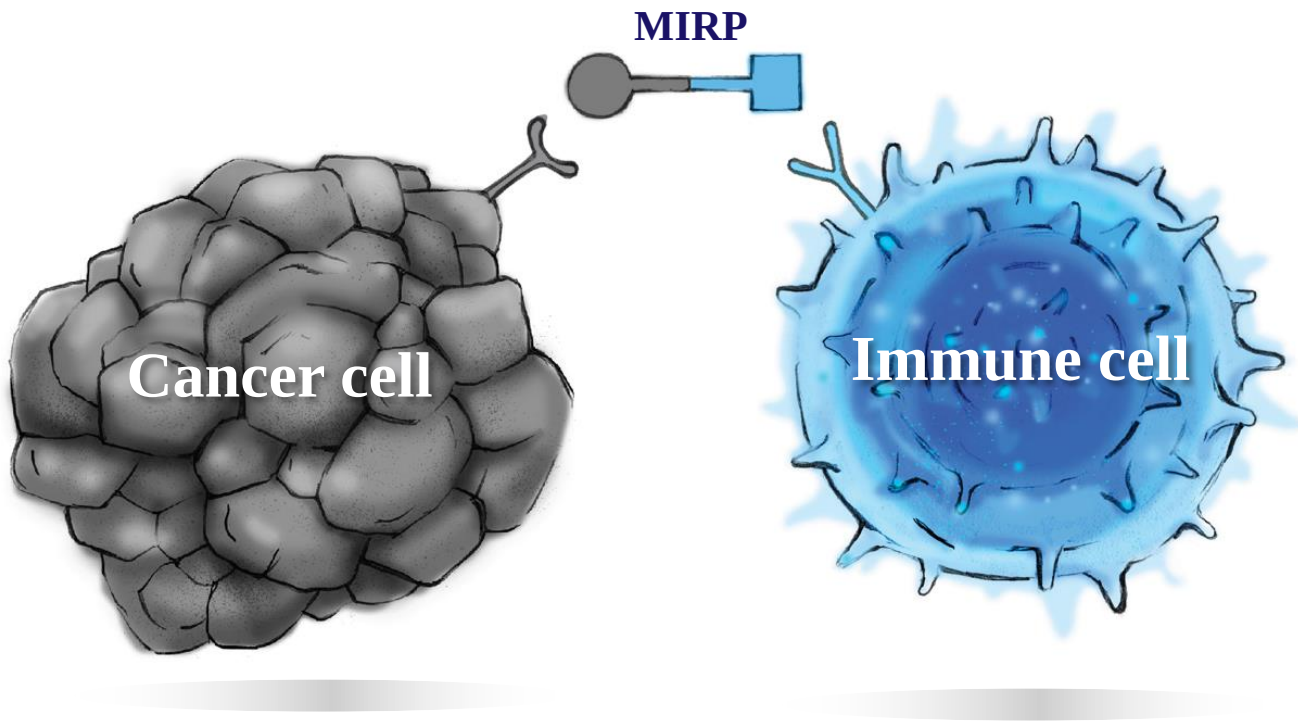
IMMUNITY. RECRUITED.

IMMUNITY.
RECRUITED.

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

MIRP (MULTI-FUNCTIONAL IMMUNE-RECRUITMENT PROTEINS)

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



IMMUNITY. RECRUITED.

HOW IT WORKS

Targeting checkpoint overexpression

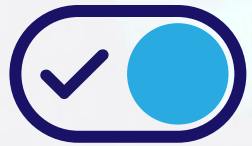
MIRPs utilize cancer cell overexpression of checkpoint surface antigens to selectively target and bind to the cancer

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 tumor environment



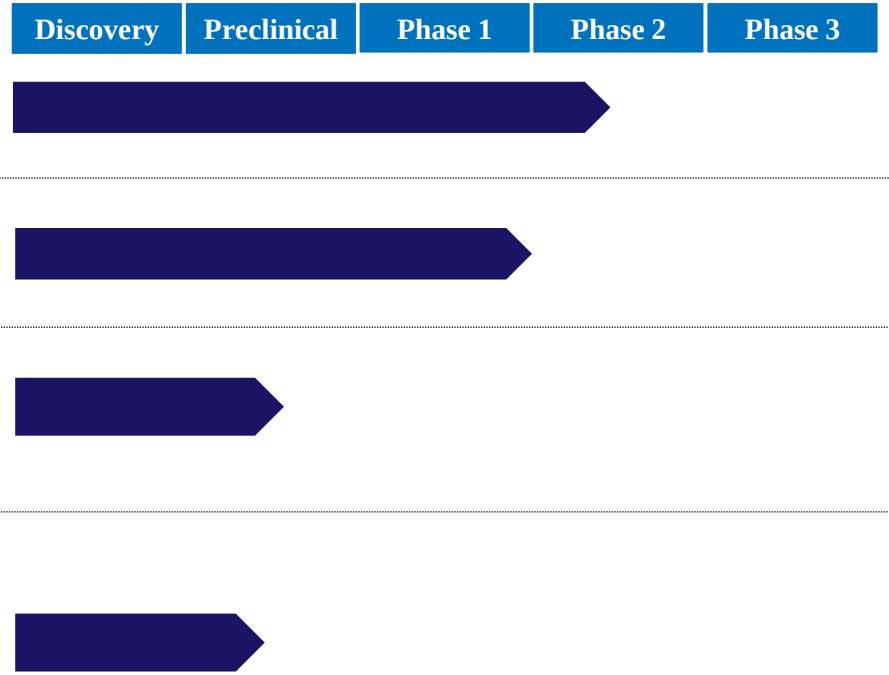
Activating immune response

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

IMMUNITY. RECRUITED.

PIPELINE

Targets	MIRP Type / MOA	Indications	Combinations
DSP107 CD47 x 41BB	DSP Activating both innate and adaptive immunity	Solid Tumors, NSCLC	Monotherapy, Atezolizumab
		AML / MDS	Monotherapy, Azacitidine, Venetoclax
DSP502 PVR x PD-L1	DSP-Fc Dual checkpoint inhibition	To Be Announced	To Be Announced
DSP216 HLA-G x CD47	DSP-Fc Dual checkpoint binding & immune stimulation	To Be Announced	To Be Announced



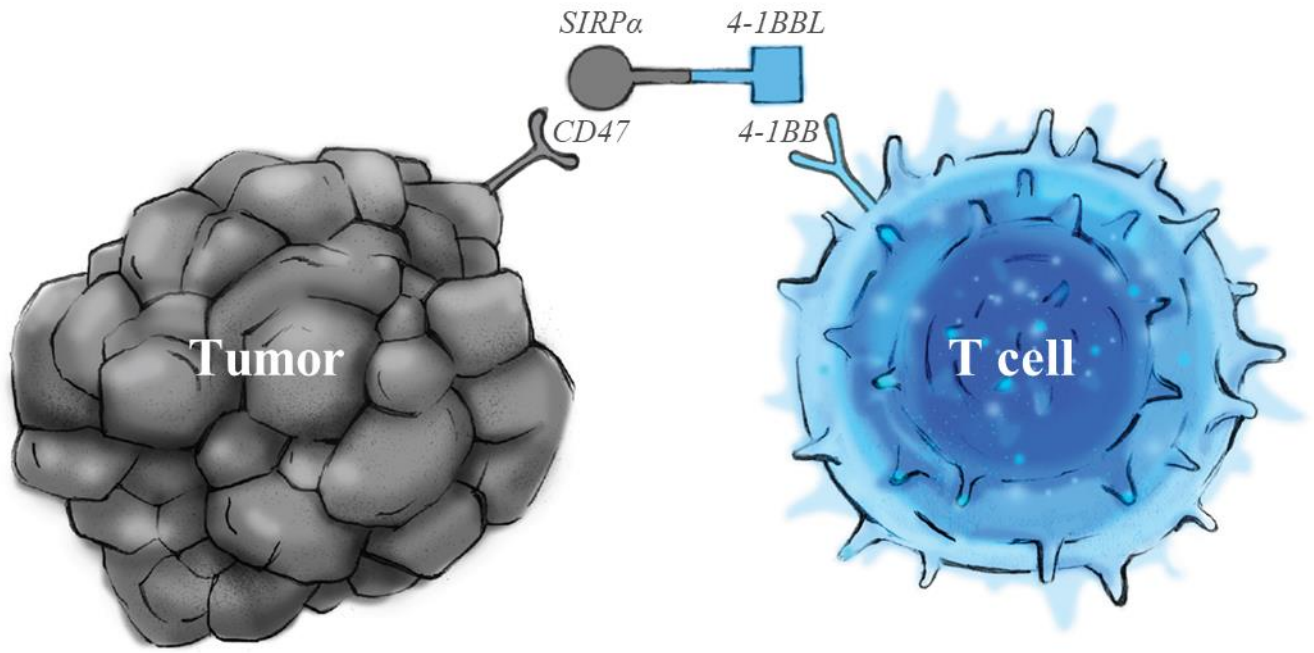
IMMUNITY. RECRUITED.

DSP107

MIRP Type: **DSP**
Targets: **CD47, 41BB**
Primary Cell Target: **mϕ macrophages, T effector cells**
Mechanistic Effect: **Unleash mϕ via ‘Don’t Eat Me’ blockade, Activate Teff**

DSP107 – First-in-Class CD47x41BB Targeting Product

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

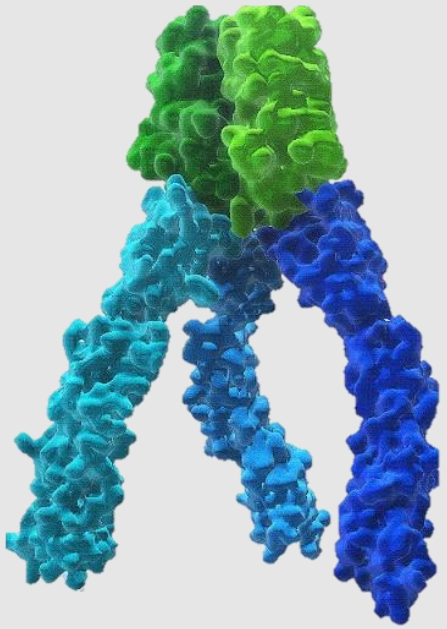
UNIQUE TRIMERIC STRUCTURE ENABLE SPECIFICITY AND SELECTIVITY

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

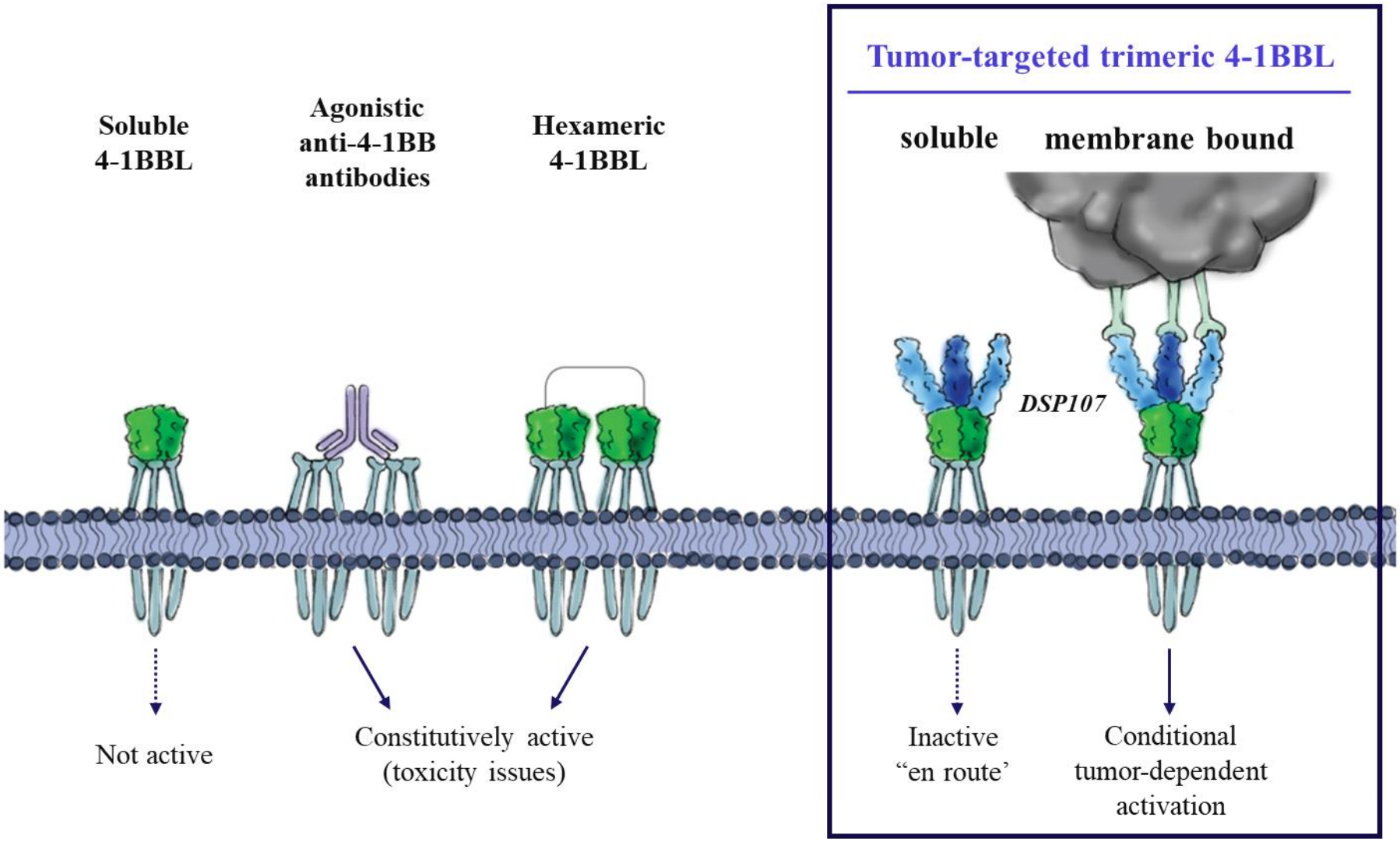
Checkpoint inhibition

Tumor microenvironment modulation

3 SIRPα for
CD47 Checkpoint Targeting

IMMUNITY. RECRUITED.

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



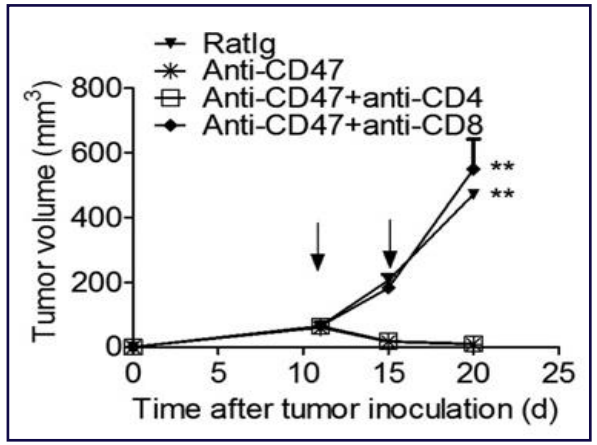
Modified from: Bremer E.; ISRN Oncology; Volume 2013, Article ID 371854

CD47 AND 4-1BB – RATIONALE

THE PROMISE OF COMBINING CHECKPOINT INHIBITION WITH IMMUNE CELL CO-STIMULATION

DSP107 is a first-in-class therapeutic agent that effectively combines CD47 checkpoint inhibition with 4-1BB-mediated activation of tumor specific T-cells

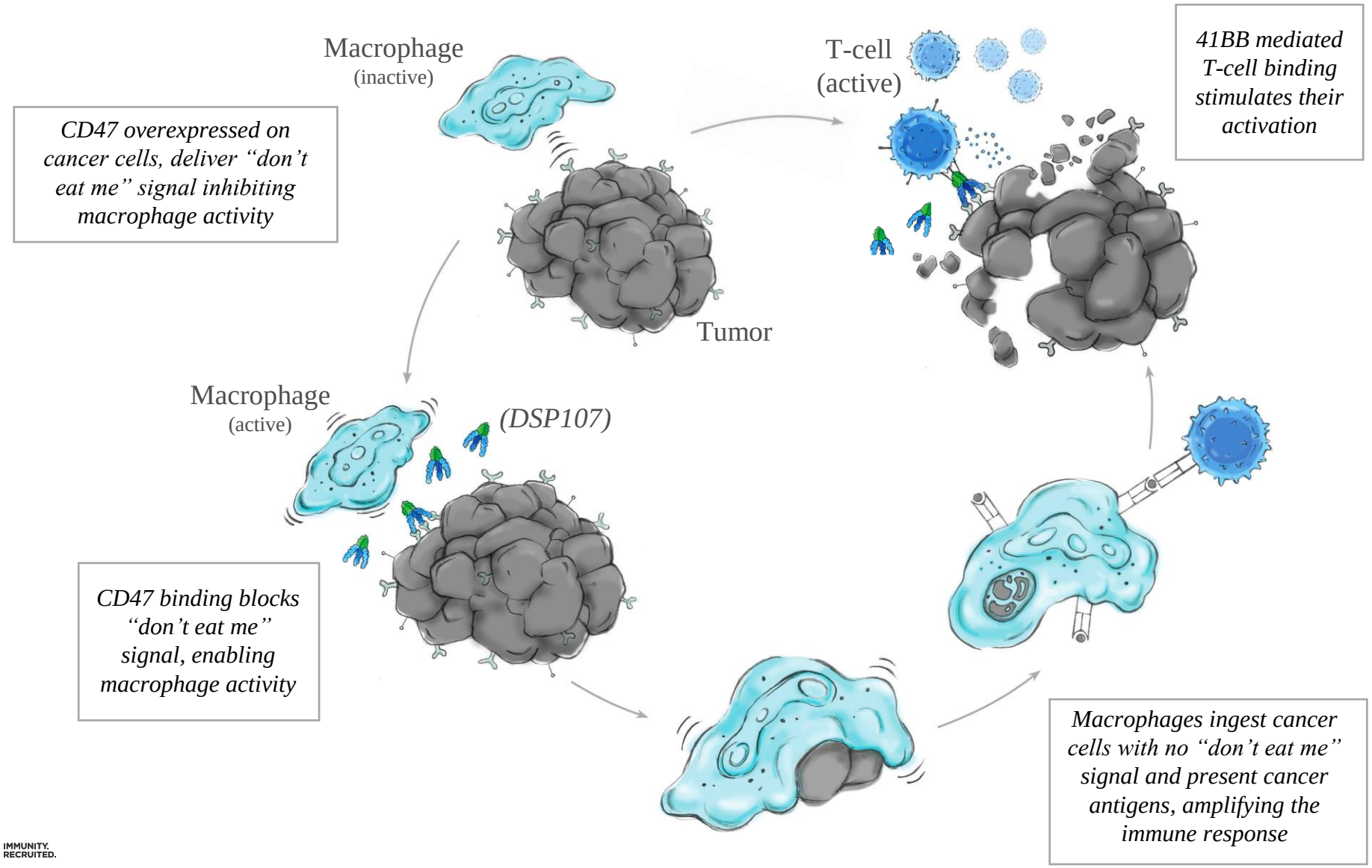
- T-cell activation is a pre-requisite for CD47 therapy, with T-cell depletion abrogating its anti tumor activity[1]
- Blockade of CD47 reactivates macrophages against cancer cells, enhances antigen presentation and induces specific anti-tumor T-cell activity[2]
- 4-1BB has been used in various studies to identify tumor-reactive T-cells in the tumor microenviroment[3]



Liu X et al. Nat Med. 2015 21:1209-15

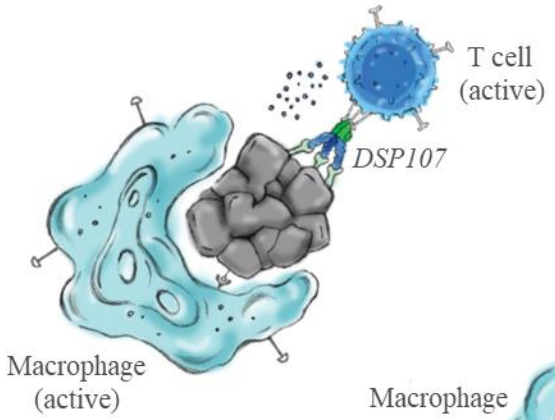
[1] Liu X et al. Nat Med. 2015 21:1209-15; [2] Tseng T et al. PNAS 2013 110: 11103-11108; [3] Chacon JA et al. PLoS ONE. 2013;8(4). [4] Bartkowiak T & Curran MA. Frontiers in Oncology. 2015 5:1-16;

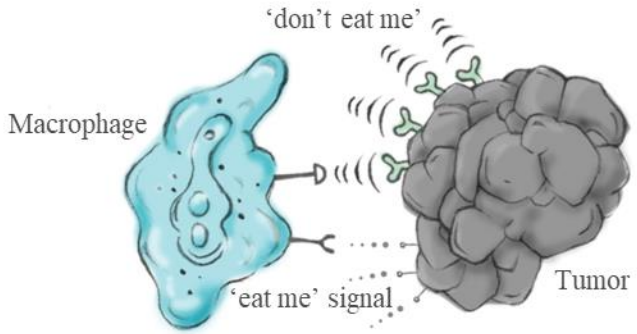
SYNERGISTIC INNATE & ADAPTIVE IMMUNE ACTIVATION

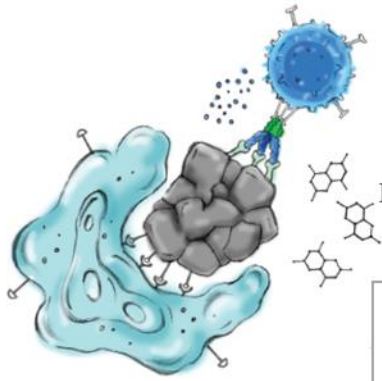


DSP107 MONOTHERAPY AND COMBINATION APPROACHES

Monotherapy potential *triggering macrophage mediated phagocytosis and T cell cytotoxicity*

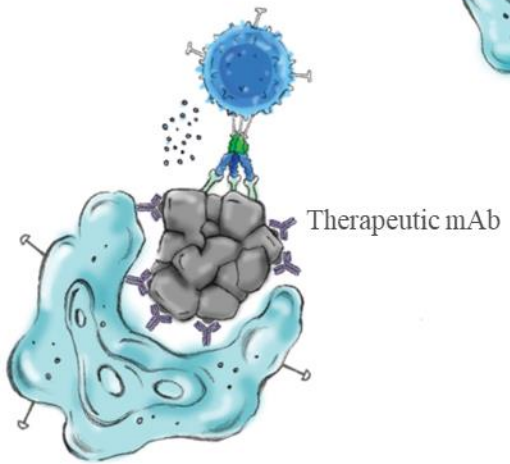


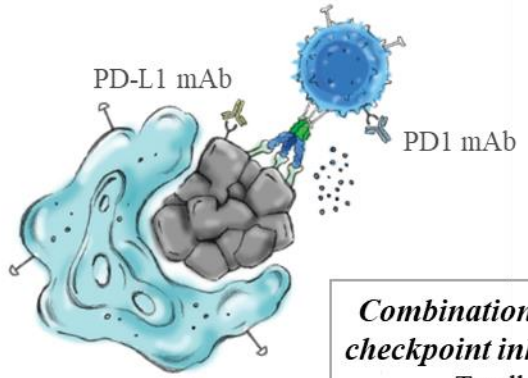




Combination with chemotherapy, hypomethylating agents and BCL2 inhibitors *to increase "eat me" signals*

Combination with therapeutic antibodies *enhancing tumor killing by* antibody-dependent cellular phagocytosis (ADCP)





Combination with PD1/PD-L1 checkpoint inhibitors to enhance T-cell activation

IMMUNITY. RECRUITED.

DSP107 DIFFERENTIATED CD47 TARGETING COMPOUND

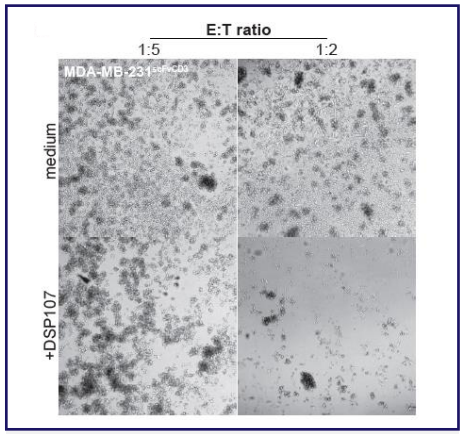
Next generation capabilities

Dual MOA
activates innate and adaptive immunity

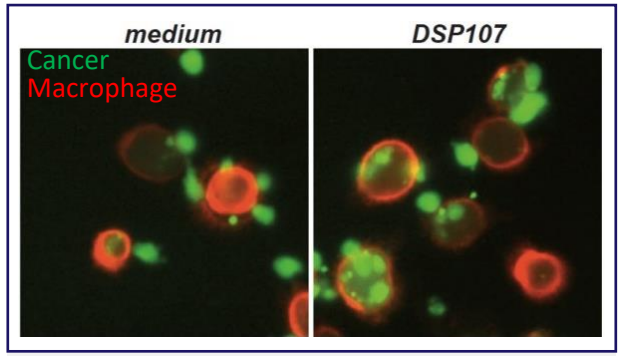
Excellent safety
without hematological toxicities

Strongly positioned
for treatment of solid and hematological malignancies

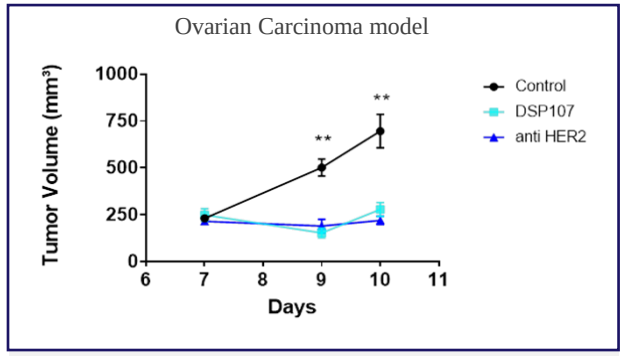
Unique and differentiated features



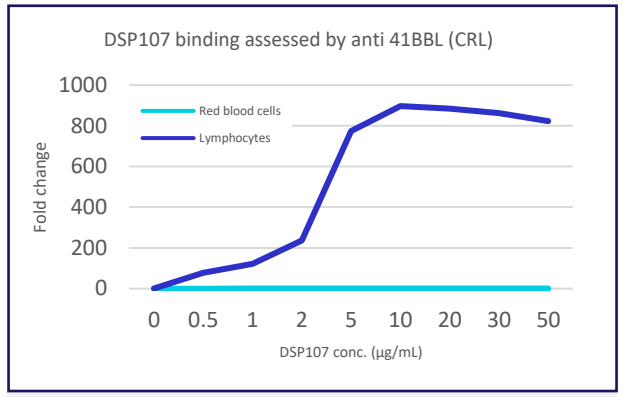
Activates T cells to secrete IFN-γ and augment their cancer cell killing potential



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



Strong anti tumor activity as a single agent in solid tumors and liquid tumors in-vivo models



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

CD47 AGENT PIPELINE

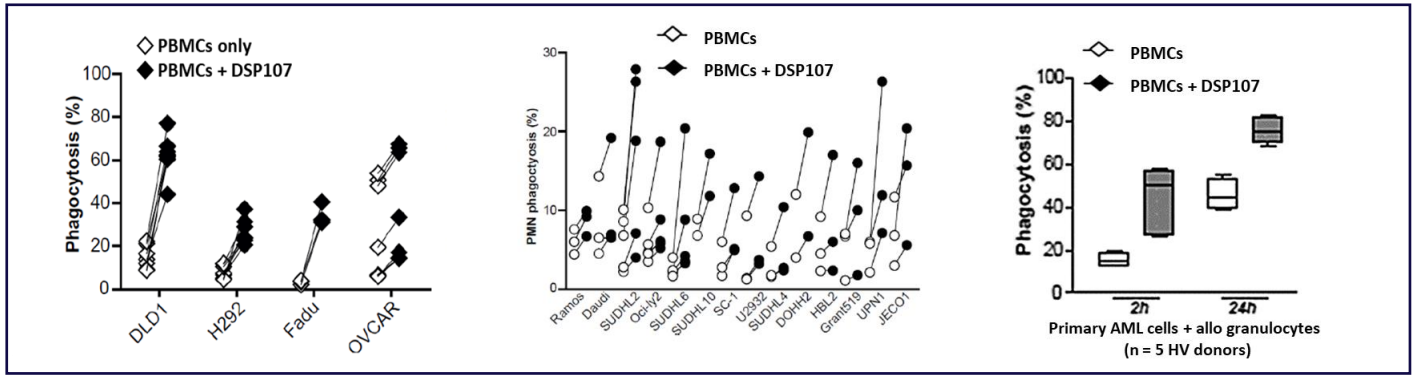
Candidate	DSP107	Magrolimab	ALX148	TTI-622	AO-176	TG-1801	SL-172154
Type	**SIRPα-41BBL** fusion protein	CD47 mAb	SIRPα-Fc fusion protein	SIRPα-Fc fusion protein	CD47 mAb	CD47/CD19 BisAb	SIRPα-Fc-CD40L Fusion protein
Mechanism	**Bi-functional**	Monovalent	Monovalent	Monovalent	Monovalent	Bi-specific	Bi-functional
Immune activation	**Innate and adaptive**	Innate	Innate	Innate	Innate	Innate	Innate
RBC binding - **Antigen Sink issue** - **Heme toxicities**	**No**	Yes	Low	No	Low	No	??
Monotherapy (preclinical)	**Yes**	No	No	Yes	Yes	Yes	Yes
Clinical indication	**NSCLC, AML, MDS**	MDS, AML, NHL and Solid tumors	NHL, HNSCC, G/GEJ	NHL	Solid tumors	NHL	Ovarian
Efficacy (ORR/CR)	**N/A**	MDS (91%/42%) AML (64%/55%) With Azacytidine NHL (50%/36%) With Rituximab	NHL (55%/18%) With Rituximab HNSCC (20%) With Pembro G/GEJ (21%) With Hercprtin	(33%/6%) Monotherapy	N/A	N/A	N/A

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

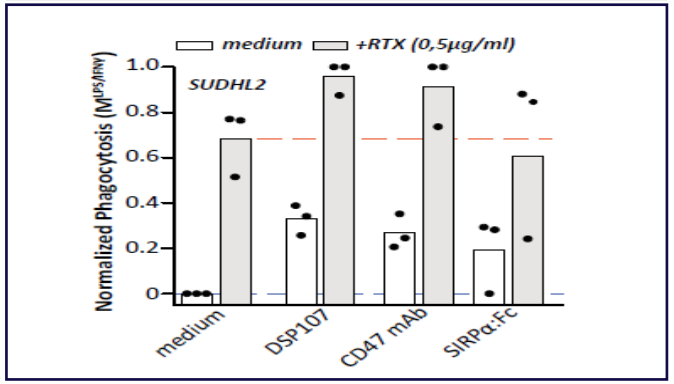
DSP107 - PRE-CLINICAL OVERVIEW

SIRPα – BINDS TUMOR AND INDUCES PHAGOCYTOSIS

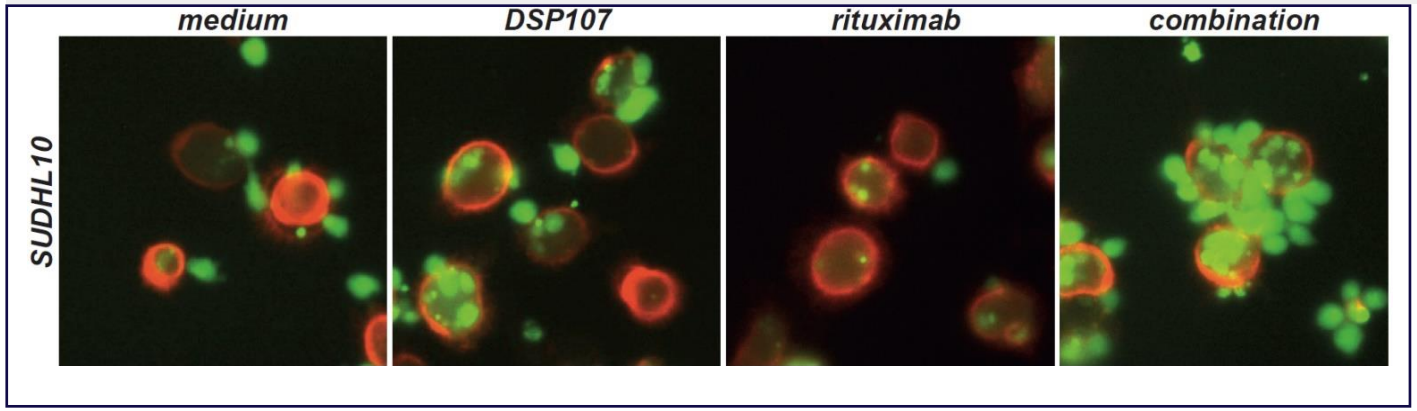
Triggers cancer cell death by phagocytosis as a single agent

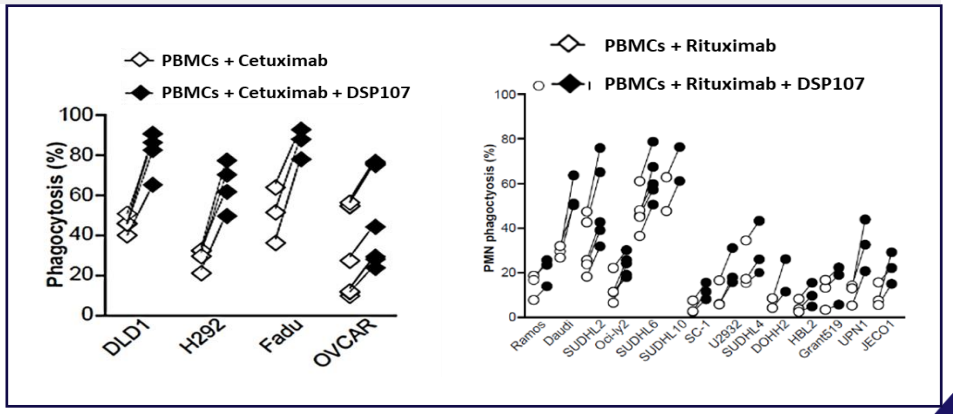


Phagocytic effect better than other CD47 targeting agents



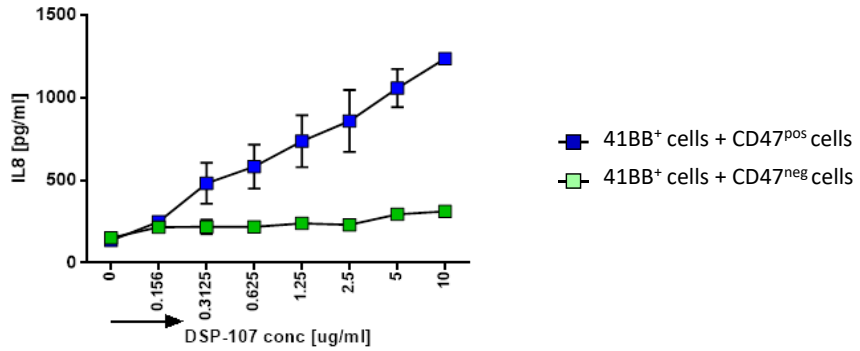
Augments mAb's ADCP-mediated phagocytosis of cancer cells



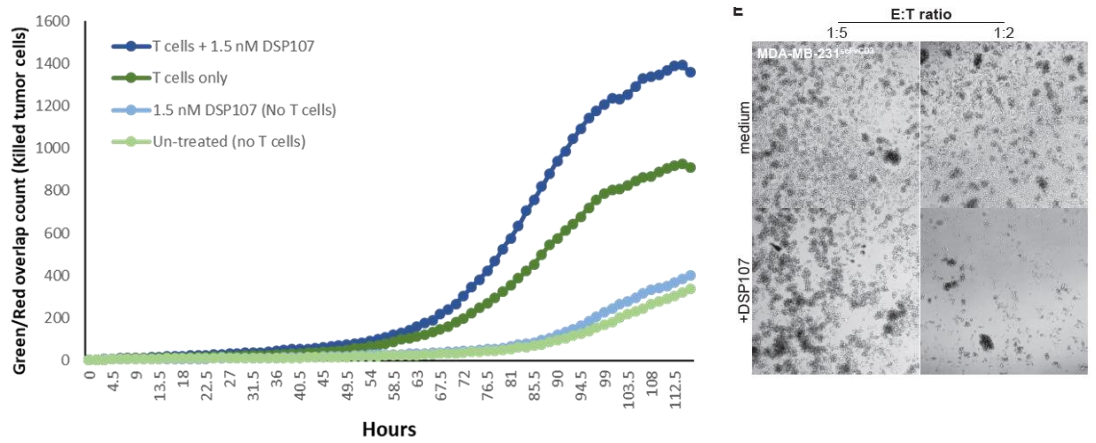


41BBL – ACTIVATES T-CELLS

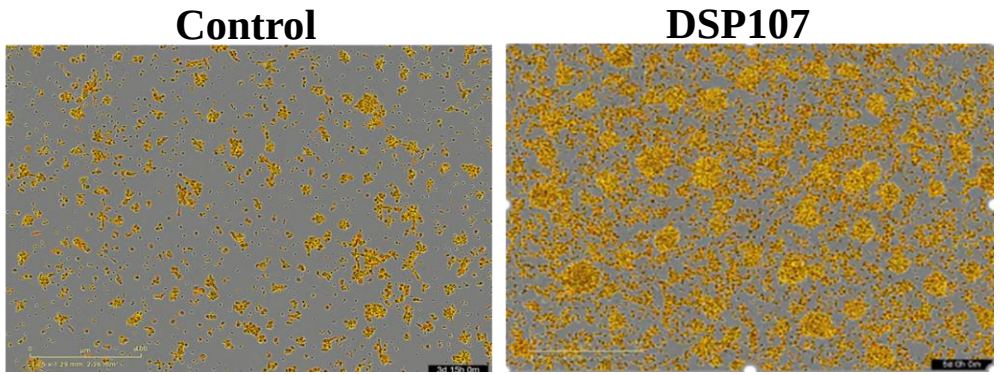
Tumor selective cross presentation activates 41BB signaling



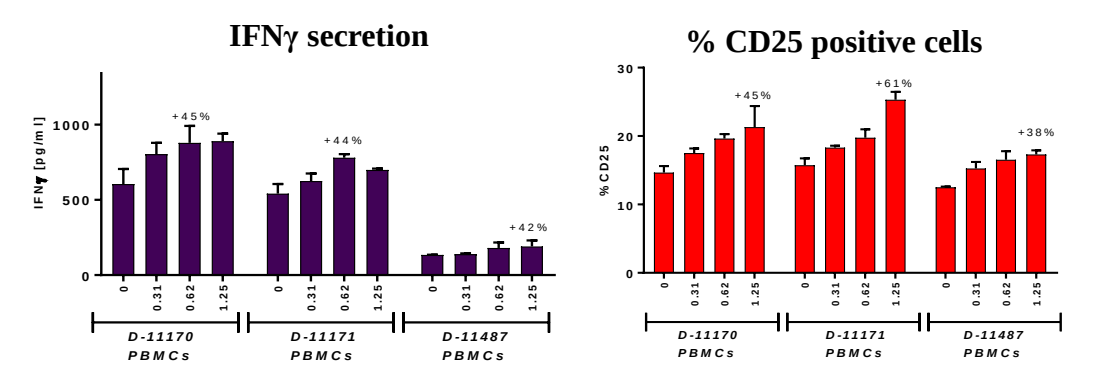
Induces T-cell killing potential against cancer cells



Augments T-cell proliferation



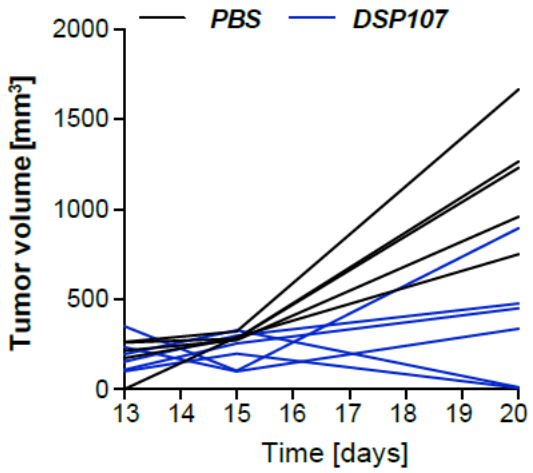
Activates T cells and increases IFNγ secretion

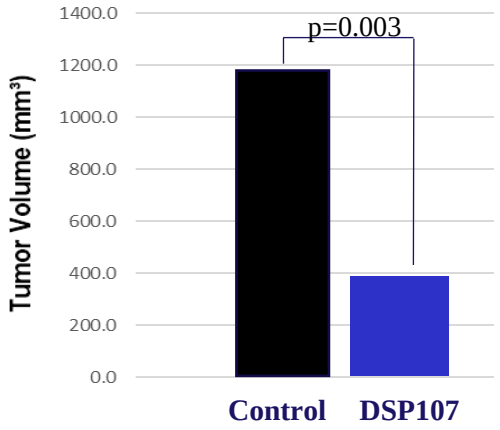


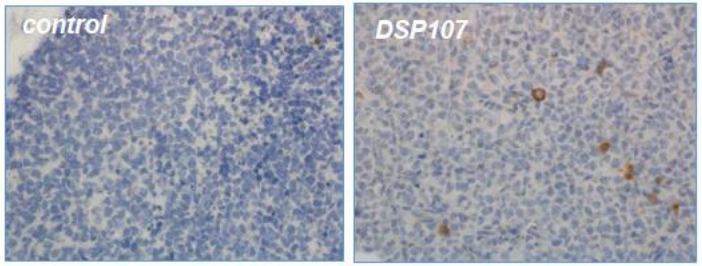
DSP107 DEMONSTRATES POTENT IN VIVO EFFICACY

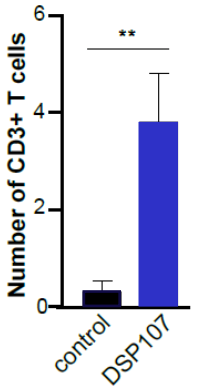
Strong single agent anti tumor activity in lymphoma model

SUDHL6 Lymphoma in Humanized NSG Mice



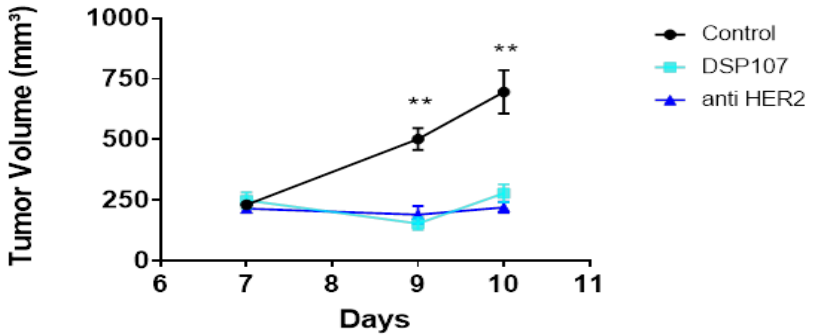






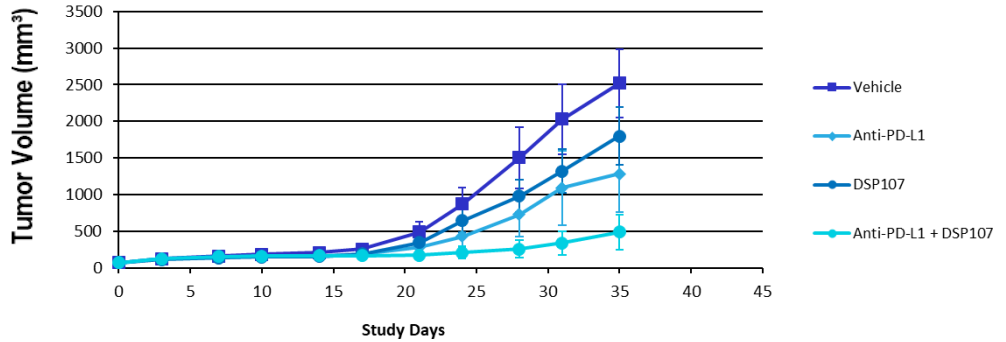
Strong single agent anti tumor activity in solid tumors

OVCAR8 Ovarian Carcinoma in NSG Mice



Significant tumor inhibition when combined with anti PD-L1

MC38-hCD47 Colon Carcinoma in C57BL-KI-h41BB Mice

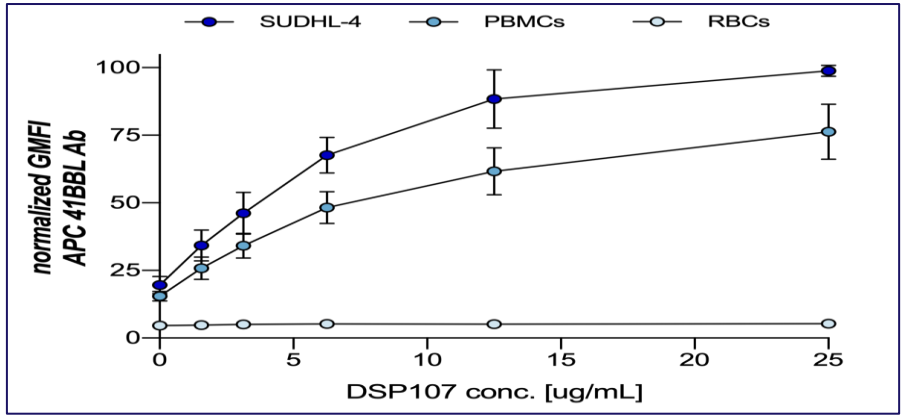


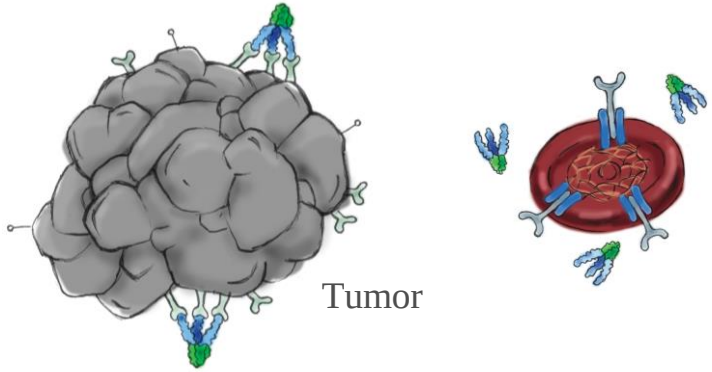
EXCELLENT SAFETY - NO HEMATOLOGICAL TOXICITIES

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



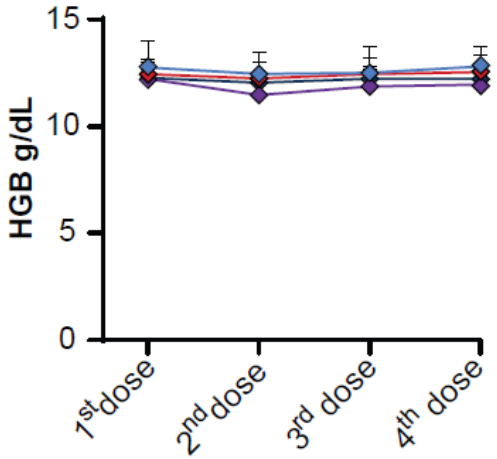


High affinity/avidity of DSP107 to CD47 clusters

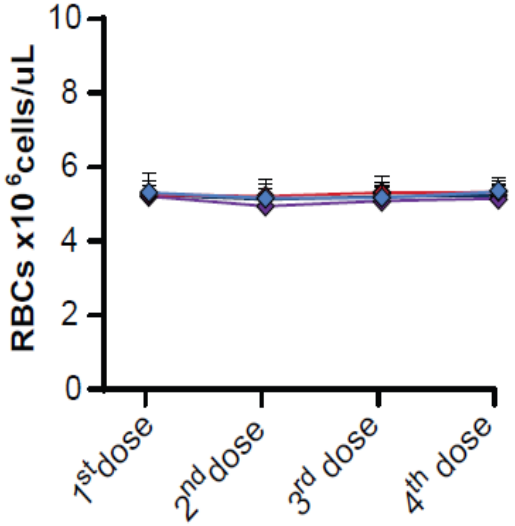
CD47 protein complex anchored to cytoskeleton resulting in its immobilization and low affinity of DSP107 to the monomeric CD47

EXCELLENT SAFETY PROFILE IN NHP

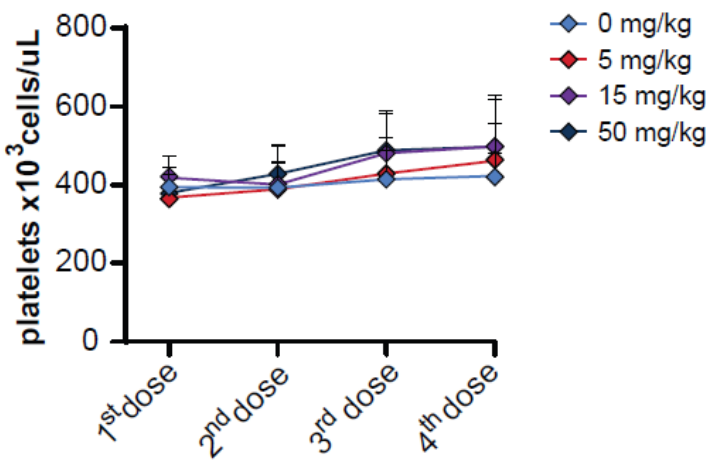
Hemoglobin

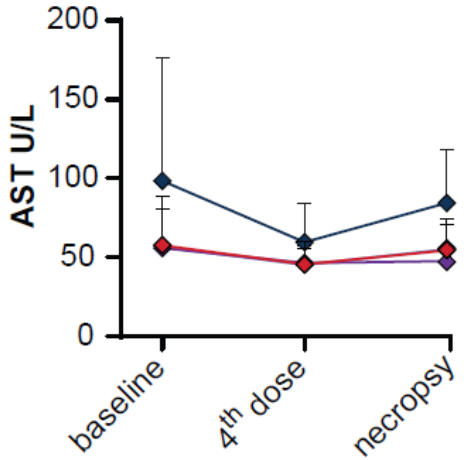


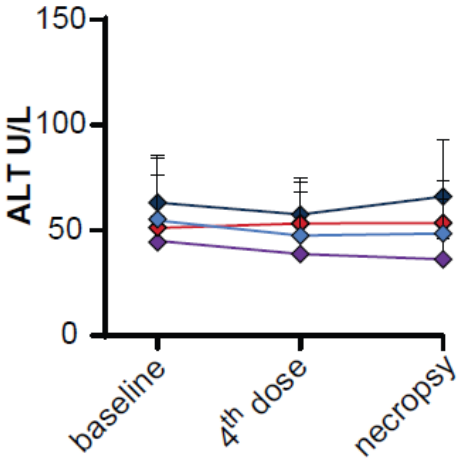
RBCs



Platelets



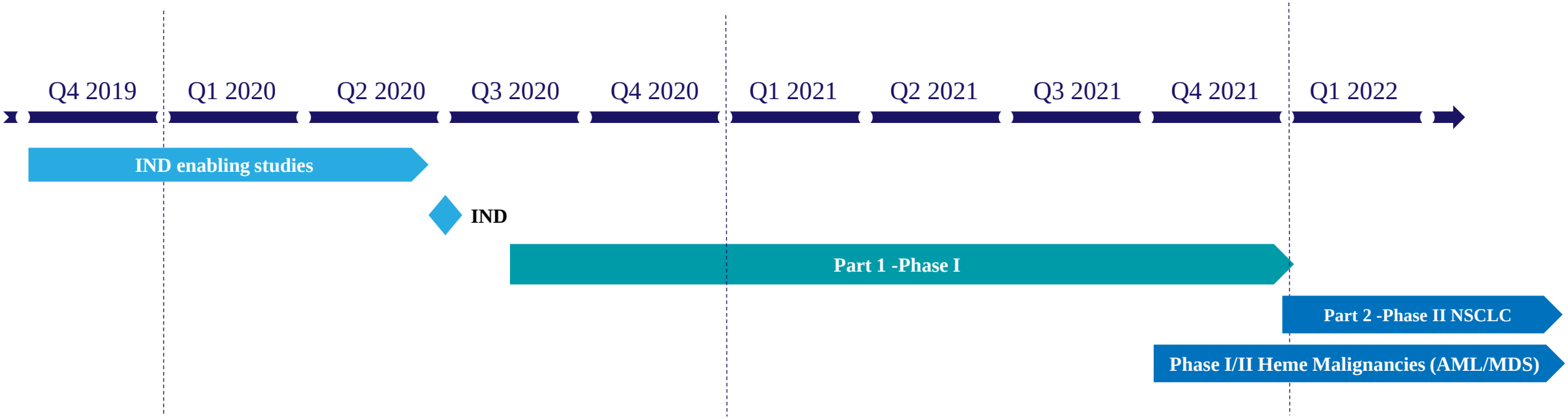




Four doses of DSP107 (on Days 1, 4, 7 and 10). Hematology data were obtained at baseline (day -1) and on days 1, 4, 7, 10

DSP107 – CLINICAL DEVELOPMENT

CLINICAL DEVELOPMENT PLAN



Two Phase I/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 azacytidine or Aza + Venetoclax

DSP107_001 PHASE I/II SOLID TUMOR STUDY

Enrolling sites: Pittsburgh, Colorado, Kansas, Thomas Jefferson, San-Diego
Additional sites under evaluation: Augusta, Chapel Hill, University of Texas

PART I — Dose escalation study

- DSP107 administered as monotherapy and in combination with Atezolizumab
- Dosing regimen - iv administration once weekly
- Population (N=~30) - 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 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)

IMMUNITY. RECRUITED.

DSP107_002 PHASE I AML/MDS STUDY

Lead site: MD Anderson Cancer Center

PART I — Dose escalation study

Part A - DSP107 administered as monotherapy (Cycle 1) and in combination with AZA (Cycle 2 and onwards)

Part B - DSP107 in combination with AZA + VEN

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

Part A dose selection based on safe, pharmacologically active dose from solid tumor study. Part B dose selection based on data from Part A.

Subject to protocol amendment post EOPI meeting with the FDA

PART II — Expansion cohorts

Dose selection based on safety and efficacy from part I after EOPI meeting

Four expansion cohorts with ongoing monitoring for treatment futility, toxicity and 4-week mortality so that enrollment can be stopped if predefined stopping boundaries are met:

Cohort I - **FRONTLINE AML** (N=28) DSP107 + AZA + VEN

Cohort II – **FRONTLINE MDS/CMML** (N=28) DSP107 + AZA

Cohort III – **R/R MDS/CMML** (N=28) DSP107 + AZA

Cohort IV - **R/R T-cell lymphoproliferative diseases** (N=28) DSP107

Endpoints

Safety and RP2D of DSP107 monotherapy and combination with AZA and AZA+VEN

Efficacy – (1) Primary efficacy endpoint - response rate (CR+CRi or CR+PR) within 6 months
(2) DOR, EFS and OS, bridging to HSCT
(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.

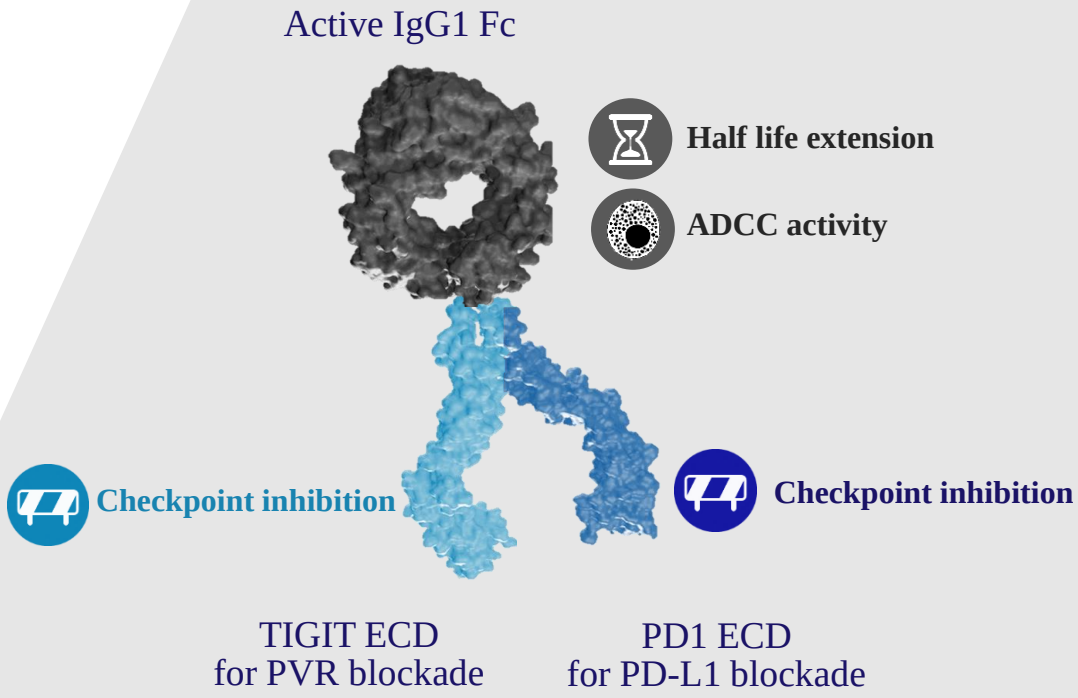
DSP502

MIRP Type: **DSP-Fc**
Targets: **PVR, PD-L1, FcR**
Primary Cell Target: **NK cells, T effector cells**
Mechanistic Effect: **Dual checkpoint inhibition unleash NK cells and Teff, ADCC**

DUAL CHECKPOINT BINDING ENABLES SPECIFICITY AND SELECTIVITY

- High tumor specificity by “And gate” binding of overexpressed checkpoints
- Active Fc backbone for mAb properties and enhanced tumor killing by ADCC

DSP502 Structure

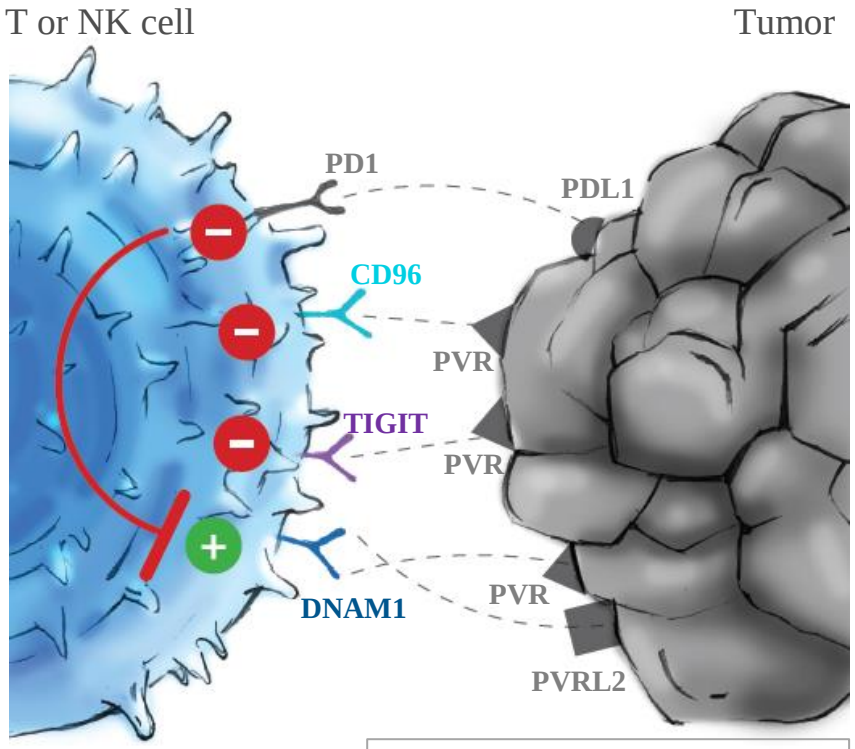


IMMUNITY. RECRUITED.

THE RATIONALE OF COMBINING PVR AND PDL1 BLOCKADE

- PVR is the ligand of TIGIT, CD96 and DNAM1
- Under normal conditions, PVR balances stimulatory (DNAM1) and inhibitory (TIGIT and CD96) signals maintain normal immune cell function
- In tumor cells, PVR is overexpressed, upregulating inhibitory receptors and downregulating stimulatory receptor to create immunosuppression

- PD1 blockade inactivates DNAM1 costimulatory downstream signaling and reduces its expression
- High PVR expression associates with resistance to PD1 checkpoint therapy in NSCLC and Melanoma patients
- Inhibition of TIGIT/PVR pathway in clinical studies shows efficacy when combined with PD-1 blockade



PD1 inhibits downstream stimulatory DNAM1

PVR overexpression in the tumor microenvironment imbalances inhibitory (TIGIT and CD96) and stimulatory (DNAM1) receptors resulting in immunosuppression

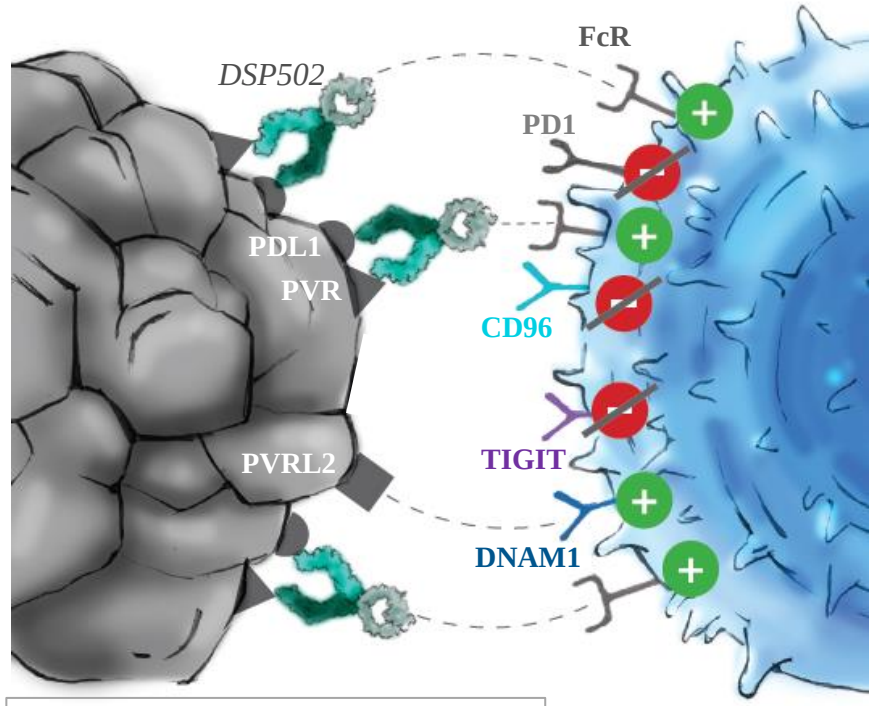
The net effect is immunosuppression

DSP502 – NOVEL SYNERGISTIC DUAL CHECKPOINT INHIBITION APPROACH

Simultaneous PVR and PD-L1 blockade enables multi checkpoint inhibition and promotes DNAM1 costimulatory signaling for effective anti-tumor immunity activating effector T and NK cells

Effect	**PVR targeting** (KAHR's approach)	**TIGIT Ab** (Competitors)
Inhibit TIGIT signaling	✔	✔
Inhibit CD96 signaling	✔	—
Increase DNAM1 surface expression and signaling	✔	—

DSP502 interferes with PD1/PD-L1 checkpoint and its IgG1-Fc delivers an immune-activating signal



DSP502 blocks PVR to potentiate DNAM1 costimulatory activity while inhibiting TIGIT/CD96 signaling

The net effect is enhanced anti-tumor immunity

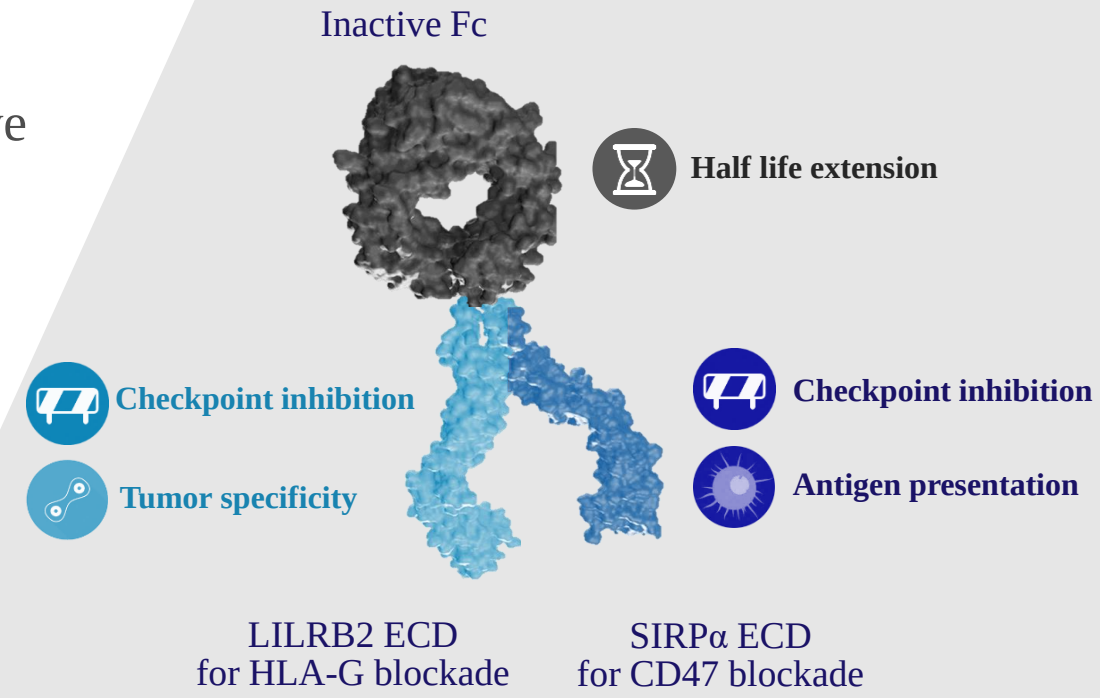
DSP216

MIRP Type: **DSP-Fc**
Targets: **CD47, HLA-G**
Primary Cell Target: **mϕ macrophages, T effector cells**
Mechanistic Effect: **Dual checkpoint inhibition unleash macrophage and Teff**

DUAL CHECKPOINT BINDING ENABLES SPECIFICITY AND SELECTIVITY

- High tumor specificity by dual binding of cancer-exclusive overexpressed checkpoint and “And gate” binding
- Fc backbone for mAb properties

DSP216 Structure

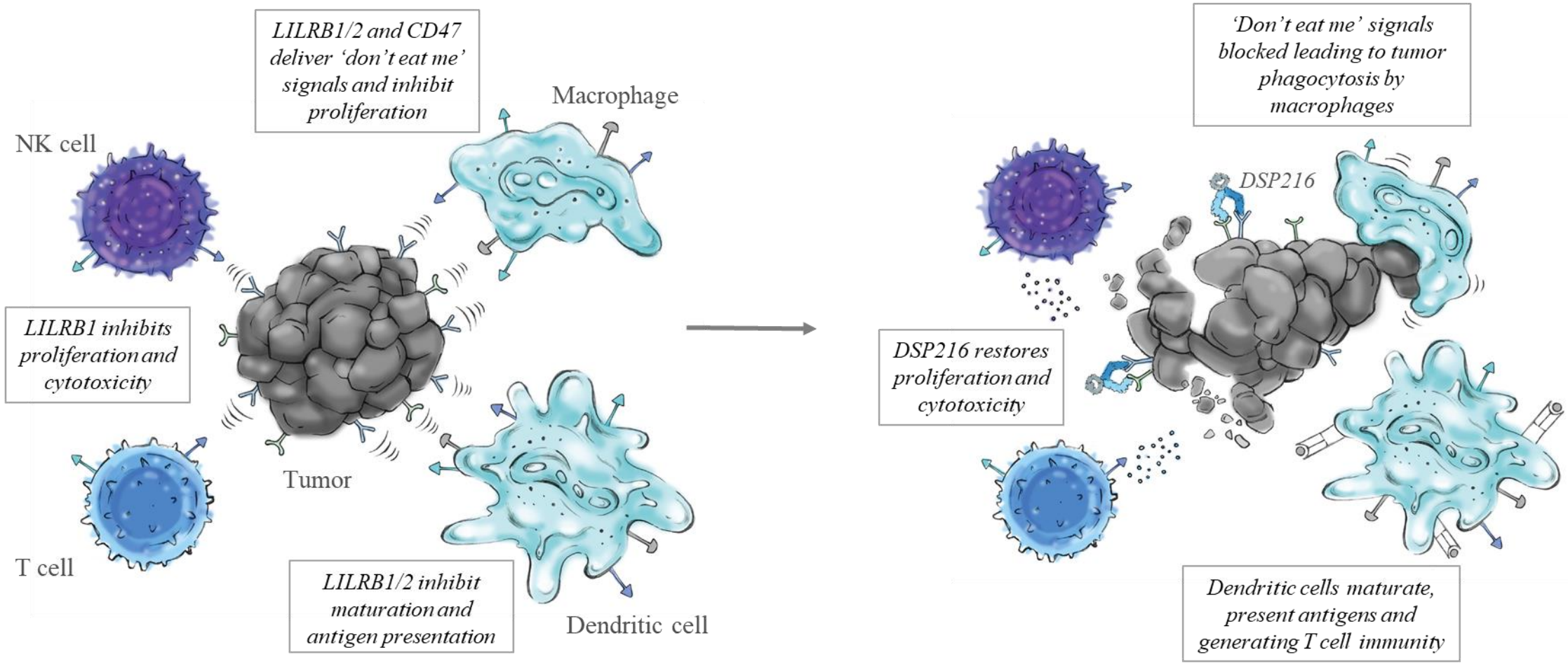


THE RATIONALE OF HLA-G TARGETING

- 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 expressed only on placentas and triggers immunotolerance that prevents the mother's immune system from attacking the fetus
- 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

Tumor cells utilize the same mechanism and evade immune surveillance by over-expressing HLA-G

SIMULTANEOUS INNATE & ADAPTIVE IMMUNE STIMULATION



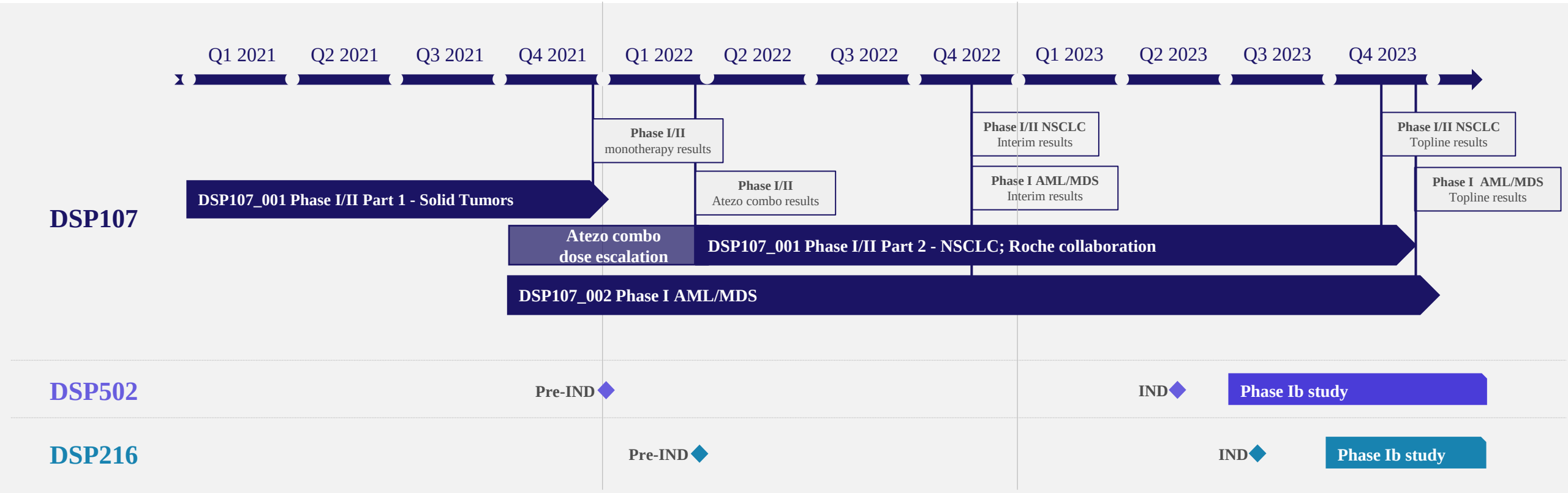
DSP216 – FIRST IN CLASS DUAL CHECKPOINT INHIBITOR

- Targeting HLA-G expressed exclusively on tumors enhances tumor targeting through dual checkpoint inhibition
- HLA-G blockade interferes with both LILRB1 and LILRB2 binding to avoid redundancy compensation
- HLA-G blockade activates both innate (macrophages) and adaptive (T cells) immune systems
- CD47 blockade removes ‘don’t eat me’ signal and triggers phagocytosis of tumor cells

Effects	**HLA-G targeting** (KAHR’s approach)	**LILRB1/2 Ab** (Competitors)
Inhibit both LILRB1 and LILRB2	✓	–
Tumor selectivity	✓	–
Activates both innate and adaptive immunity	✓	✓

BUSINESS OVERVIEW

ROAD MAP


Q1 2021
Q2 2021
Q3 2021
Q4 2021
Q1 2022
Q2 2022
Q3 2022
Q4 2022
Q1 2023
Q2 2023
Q3 2023
Q4 2023
DSP107
Phase I/II
monotherapy results
Phase I/II
Atezo combo results
Phase I/II NSCLC
Interim results
Phase I AML/MDS
Interim results
Phase I/II NSCLC
Topline results
Phase I AML/MDS
Topline results
DSP107_001 Phase I/II Part 1 - Solid Tumors
Atezo combo
dose escalation
DSP107_001 Phase I/II Part 2 - NSCLC; Roche collaboration
DSP107_002 Phase I AML/MDS
DSP502
Pre-IND
IND
Phase Ib study
DSP216
Pre-IND
IND
Phase Ib study

THANK YOU!