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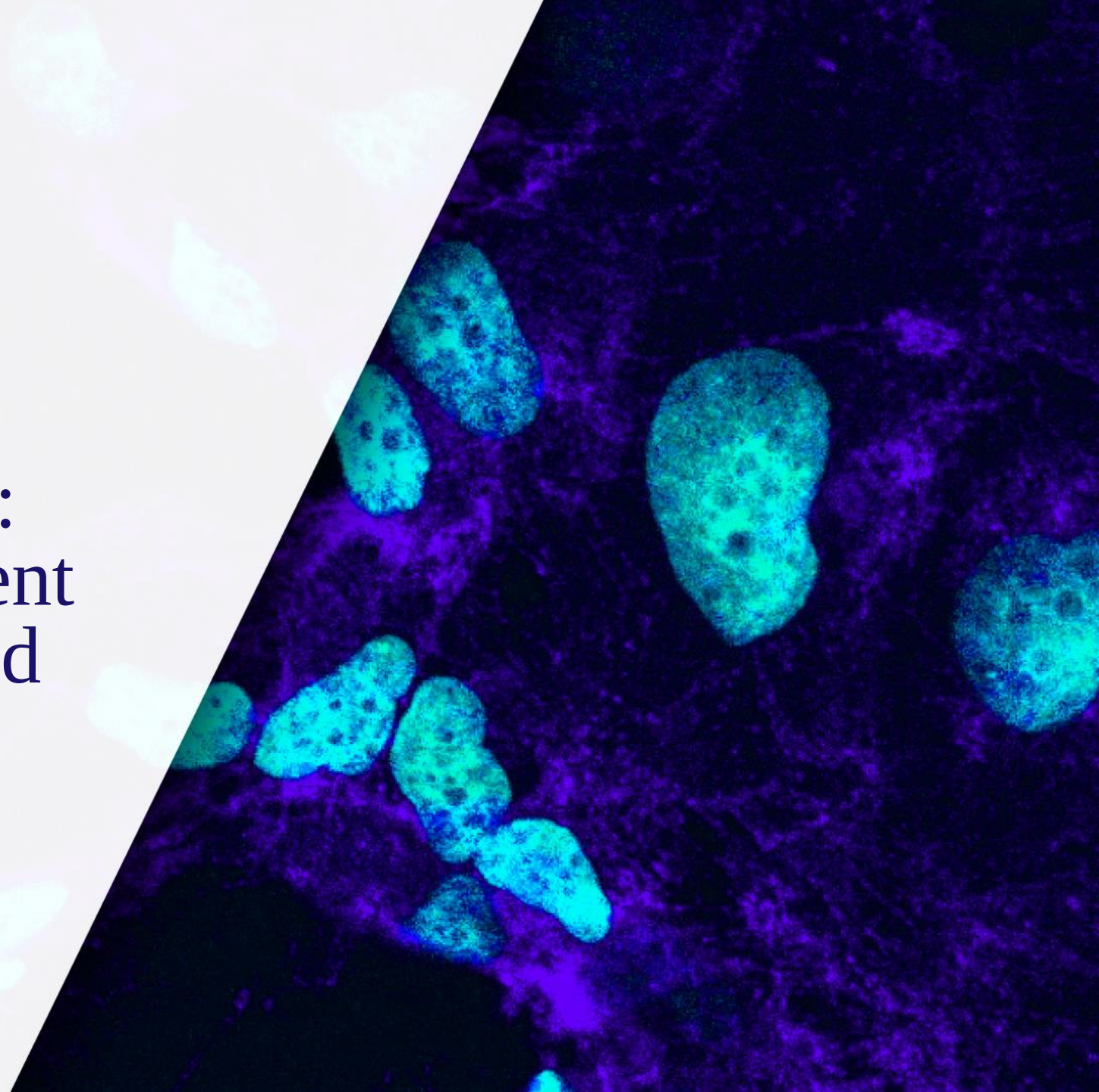
KOL Webinar on CD47 Therapeutics: A Potential Treatment For Solid Tumor and Hematological Malignancies



December 2021

AGENDA

Welcome and Introduction	**Yaron Pereg, PhD**, CEO, KAHR, Jerusalem, Israel
Therapeutic potential of CD47 therapies for solid tumors	**Ezra Cohen, MD, FRCPSC, FASCO,** Chief of Hematology-Oncology at UC San Diego Moores Cancer Center, and o-Director of the San Diego Center for Precision Immunotherapy, San Diego, California
Unmet need in MDS and AML and CD47 landscape	**Naval G. Daver, MD,** Associate Professor of Leukemia at MD Anderson Cancer Center, Houston, Texas
KAHR Pipeline Overview	**Yaron Pereg, PhD**, CEO, KAHR, Jerusalem, Israel
DSP107 Initial Phase 1 Data	**Adam Foley-Comer, MD**, CMO, KAHR, Jerusalem, Israel
Q&A Session	

EZRA COHEN, MD, FRCPSC, FASCO

CHIEF OF HEMATOLOGY-ONCOLOGY AT UC SAN DIEGO MOORES CANCER CENTER, AND CO-DIRECTOR OF THE SAN DIEGO CENTER FOR PRECISION IMMUNOTHERAPY

Ezra Cohen, MD, FRCPSC, FASCO is a board-certified oncologist and an internationally renowned cancer researcher. Dr. Cohen serves as co-director of UC San Diego Health's Precision Immunotherapy Clinic, which offers the most promising investigational immunotherapy treatments for many types of cancer, including head and neck cancers. At UC San Diego Health's Moores Cancer Center, he is associate director for translational science and the leader of the Solid Tumor Therapeutics research program. As a physician-scientist, Dr. Cohen also leads a laboratory that studies novel cancer treatments, including immunotherapy, with a particular focus on squamous cell carcinomas and cancers of the thyroid, salivary gland, and HPV-related oropharyngeal cancers. A frequent speaker at national and international meetings, he has authored more than 170 peer-reviewed papers and has been the principal investigator of multiple clinical trials of new drugs for head and neck cancer and other solid tumors in all phases of development. Dr. Cohen completed a hematology/oncology fellowship at the University of Chicago, where he was named chief fellow. He completed residencies in family medicine at the University of Toronto and in internal medicine at Albert Einstein College of Medicine. Dr. Cohen earned his medical degree at University of Toronto. He is board certified in medical oncology, and a fellow of the Royal College of Physicians and Surgeons of Canada (FRCPSC) and the American Society of Clinical Oncology (FASCO).

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NAVAL G. DAVER, MD

ASSOCIATE PROFESSOR OF LEUKEMIA AT MD ANDERSON CANCER CENTER, HOUSTON, TEXAS

Naval G. Daver, MD is an Associate Professor in the Department of Leukemia at MD Anderson Cancer Center. He completed his medical school from Grant Medical College and Sir J group of Hospitals Mumbai, followed by a residency and fellowship in hematology-oncology from Baylor College of Medicine. He is a clinical investigator with a focus on molecular and immune therapies in AML and Myelofibrosis and is principal investigator on >25 ongoing institutional, national and international clinical trials in these diseases. These trials focus on developing a personalized therapy approach by targeting specific mutations or immune pathways expressed by patients with AML, evaluating novel combinations of targeted, immune and cytotoxic agents, and identifying and overcoming mechanism of resistance. He is especially interested in developing monoclonal and bispecific antibodies, immune checkpoint and vaccine based approaches in AML, MDS, and myelofibrosis and is leading a number of these trials at MDACC. Dr. Daver has published >150 peer-reviewed manuscripts and is on the editorial board of numerous hematology specific journals. He has also authored numerous abstracts at national and international conferences.

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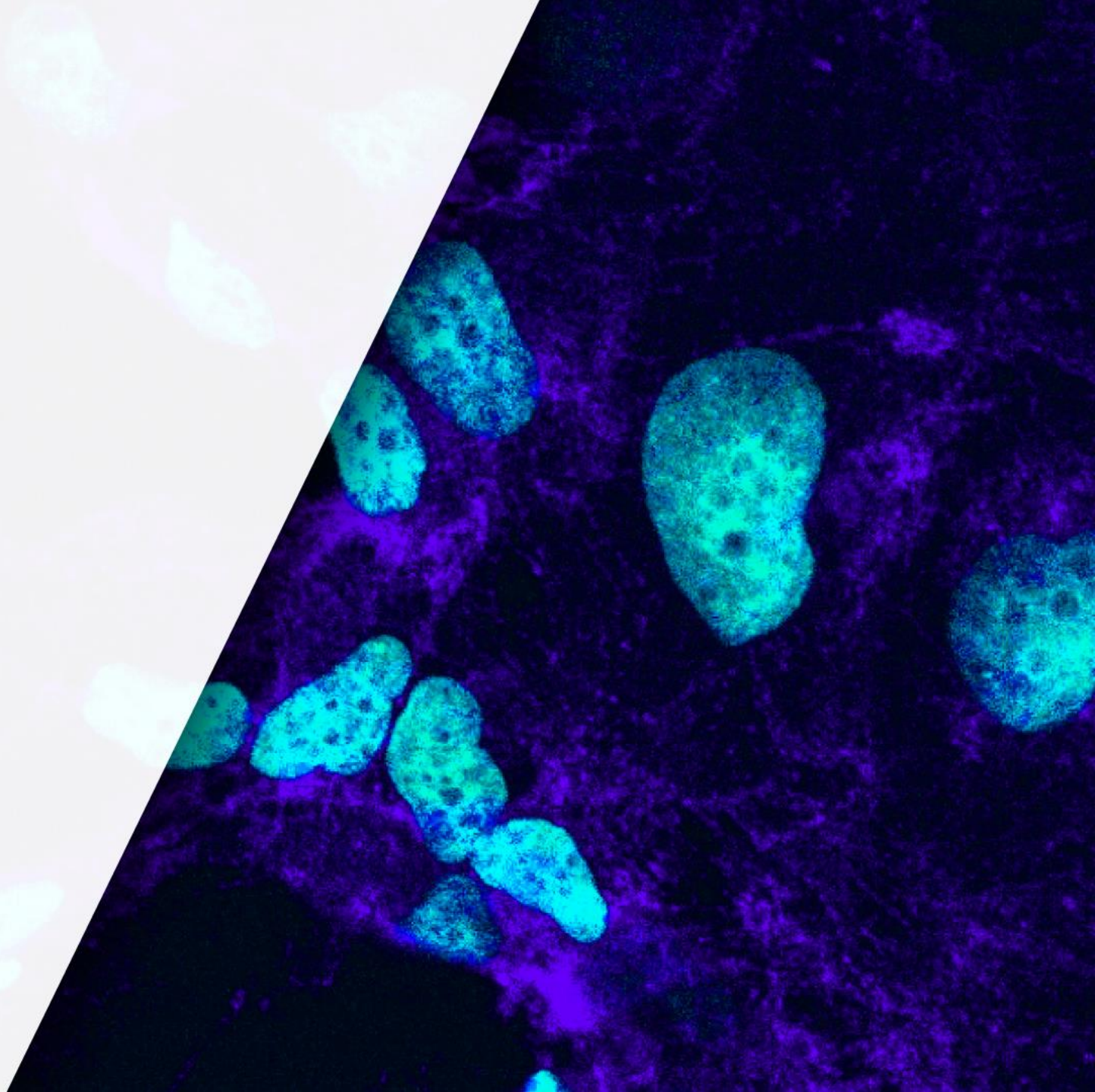
UNMASKING CANCER CELL CAMOUFLAGE

COMPANY PRESENTATION | Dec. 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.

MULTIFUNCTIONAL CANCER IMMUNOTHERAPIES TARGETING INNATE AND ADAPTIVE IMMUNE SYSTEMS

NOVEL MIRPs

Multifunctional Immuno-Recruitment Proteins – versatile platform targeting both innate & adaptive immunity across cancers

UNIQUE PIPELINE

- First-in-class potential across 3 programs
- Lead candidate DSP107 –
  CD47 inhibition (Cancer specific)
  4-1BB activation (CD47-conditional)

UPCOMING MILESTONES

- **DSP107**| Initial Ph I/II combo data H1 2022
- **DSP502 & DSP216** | IND 2023
- **Multiple future candidates** in research pipeline

MARKET

Immuno-therapeutics
$56.5B by 2025
(Source: Allied Market Research)

IP

13 families
4 granted (US and other territories),
73 pending (NP worldwide and PCT stage)

Experienced Leadership

Management team, BOD and SAB comprised of leading experts including technology inventor, Prof. Mark Tykocinski, Dean of the School of Medicine and Provost, Jefferson University.

EXPERIENCED LEADERSHIP TEAM

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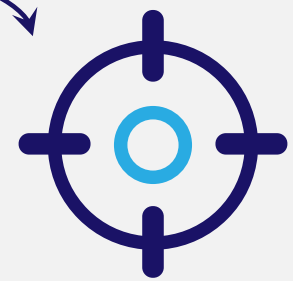
Co-Founder & Managing General Partner at ALIVE Israel HealthTech Fund; 20+ yrs investing in biotech

EFFECTIVELY TREATING CANCER REQUIRES A MULTIFACETED APPROACH

Selectively disabling cancer defense mechanism

WHILE



Recruiting a local targeted immune attack

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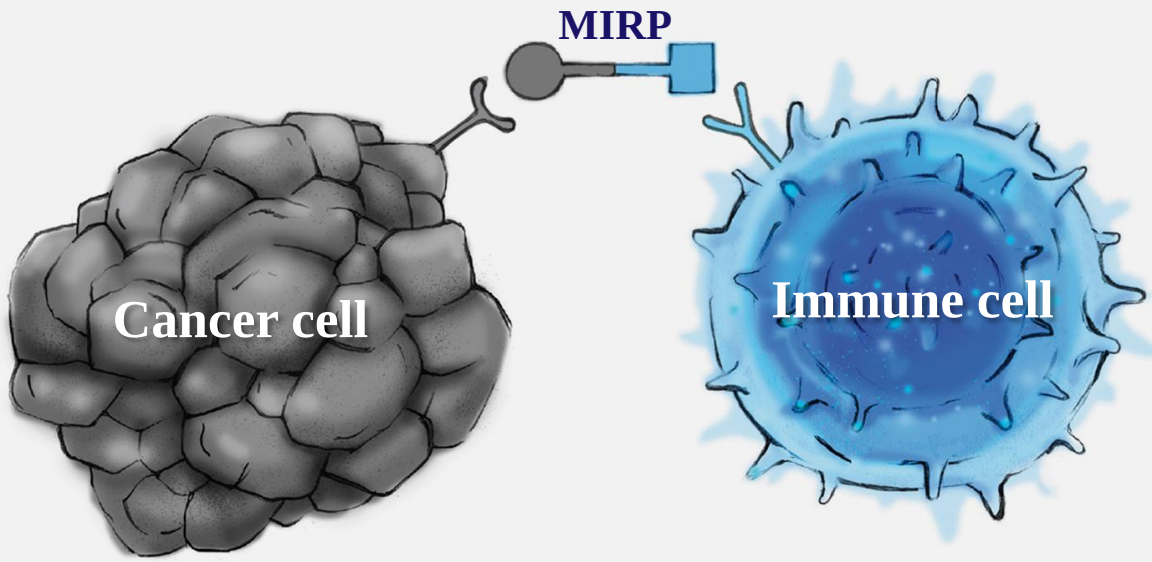
MULTIFUNCTIONAL IMMUNO-RECRUITMENT PROTEIN (MIRP) VERSATILE IMMUNO-THERAPEUTIC PLATFORM DESIGNED TO SAFELY OVERCOME CANCER EVASION

MIRPs trigger a multilayered immune response by:

1. Inhibiting key evasion markers on cancer cells
2. Activating innate and adaptive anti-tumor immunity



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MIRP STRATEGIES FOR IMMUNE RECRUITMENT & ACTIVATION

Two configurations utilize different target-dependent strategies designed to improve safety and efficacy

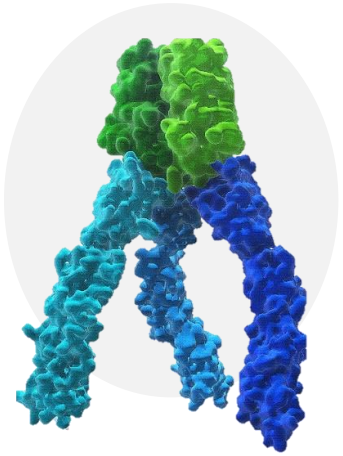
DSP (Dual Signaling Protein)

Combined checkpoint inhibition and immune co-stimulation

DSP107

4-1BB activator – *CD47-conditional T-cell activation*

CD47 inhibitor – *Trimeric binding for cancer specific blocking*



DSP-Fc (Dual Signaling Protein With Fc Domain)

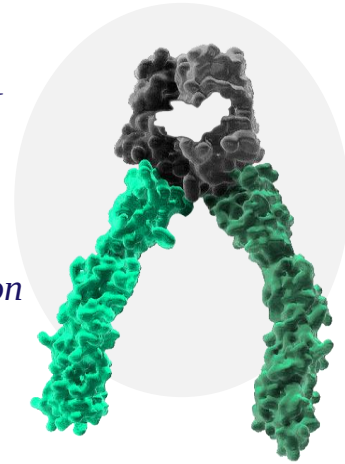
Dual checkpoint inhibition for diverse immune modulation

DSP502

PVR inhibitor – *Dual PD1/TIGIT inhibition with DNAMI potentiation*

PD-L1 inhibitor – *T and NK cell activation*

Active IgG1 Fc – *Half-life extension, ADCC activity*

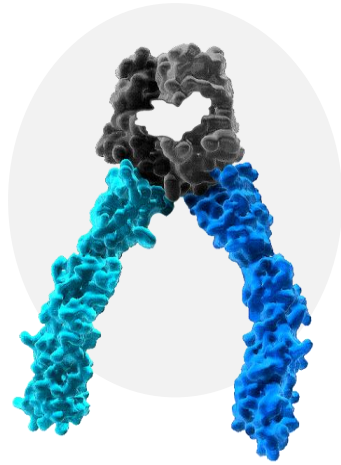


DSP216

CD47 inhibitor – *Avidity driven for cancer specific blocking*

HLA-G inhibitor – *Inhibition of LILRB1,LILRB2*

Inactive Fc – *Half-life extension*



FOCUSED AND DIFFERENTIATED PIPELINE

MIRP Type	Program	Targets	Indications	Discovery	Preclinical	Phase 1	Phase 2	Phase 3	Upcoming Milestones	Commercial Rights
DSP	DSP107	CD47 4-1BB	Solid Tumors, NSCLC			DSP107 ± atezolizumab*			Initial Ph I/II combo results 1H 2022	
			AML / MDS			DSP107 ± azacitidine + venetoclax			Initial Ph Ib results Q4 2022	
DSP-Fc	DSP502	PVR PD-L1	Oncology						IND Filing 1H 2023	
	DSP216	HLA-G CD47	Oncology						IND Filing 2H 2023	

Roche *Clinical trial collaboration and supply agreement with Roche for the PD-L1 inhibitor atezolizumab (TECENTRIQ®)

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DSP107

MIRP Type	**DSP**
Targets	**CD47, 4-1BB**
Primary Cell Target	**mϕ macrophages, T-effector cells**
Mechanistic Effect	**Unleash mϕ via ‘Don’t Eat Me’ blockade, Activate T-eff**

DSP107
UNIQUE TRIMERIC STRUCTURE ENABLES TUMOR SELECTIVITY

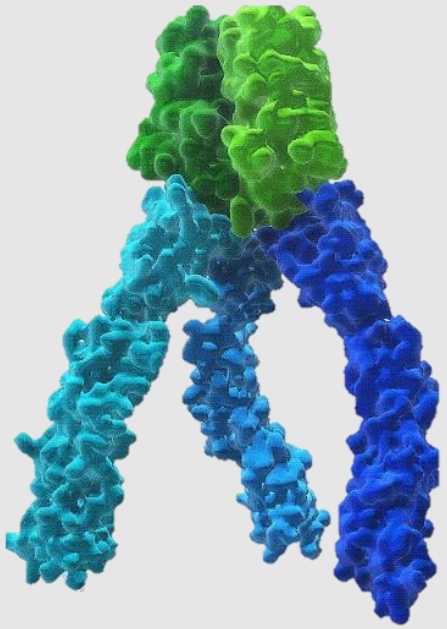
DSP107 Structure

Trimeric ligand ends enable:

1. Cancer selective binding driven by high affinity and avidity to overexpressed CD47

2. Conditional 4-1BB mediated T cell activation dependent on trimeric binding to CD47 on cancer cells

Trimeric 4-1BBL



3 SIRPα for
CD47 Checkpoint Targeting

Cytolytic T cell activation

T cell Proliferation

Checkpoint inhibition

Tumor microenvironment modulation

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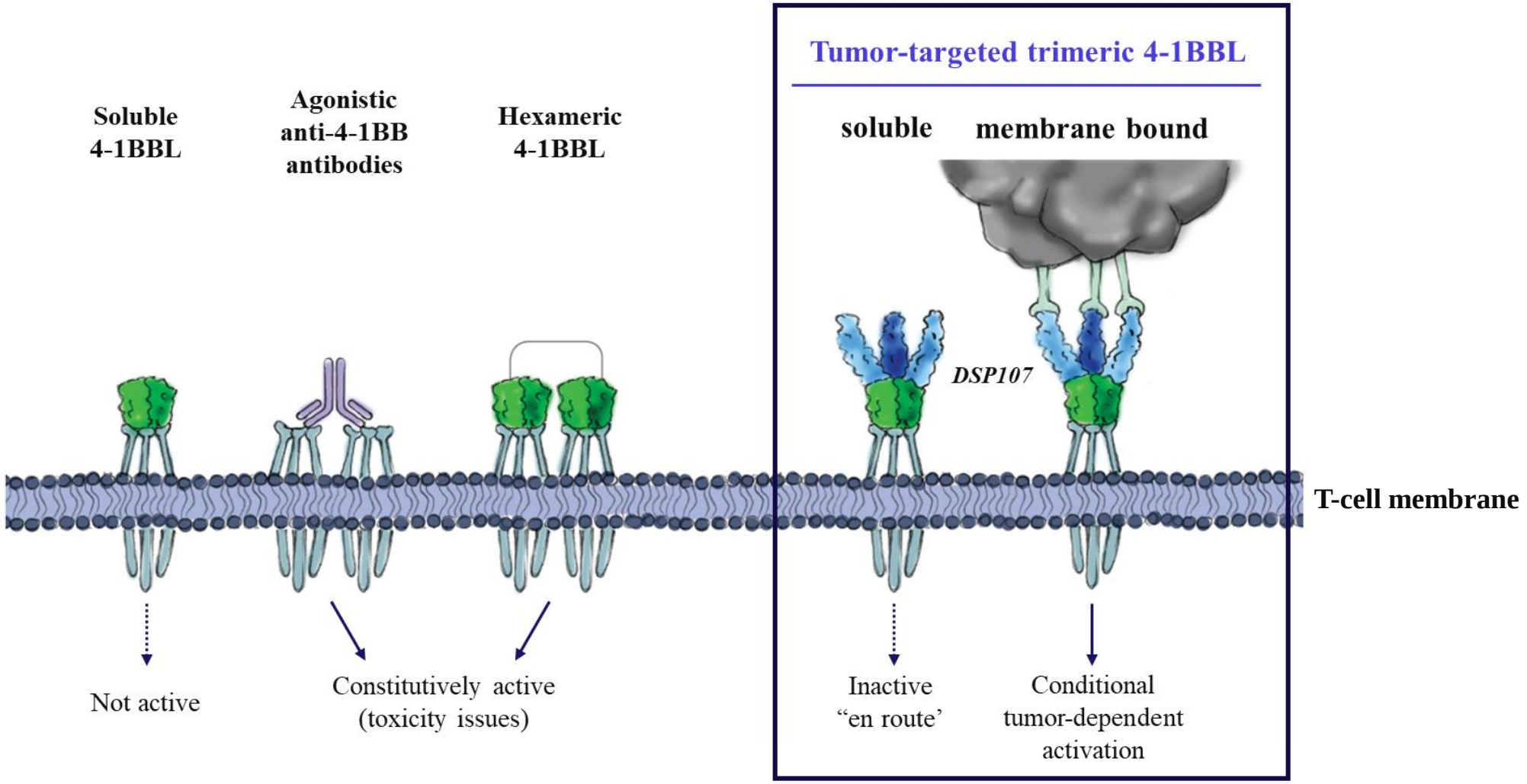
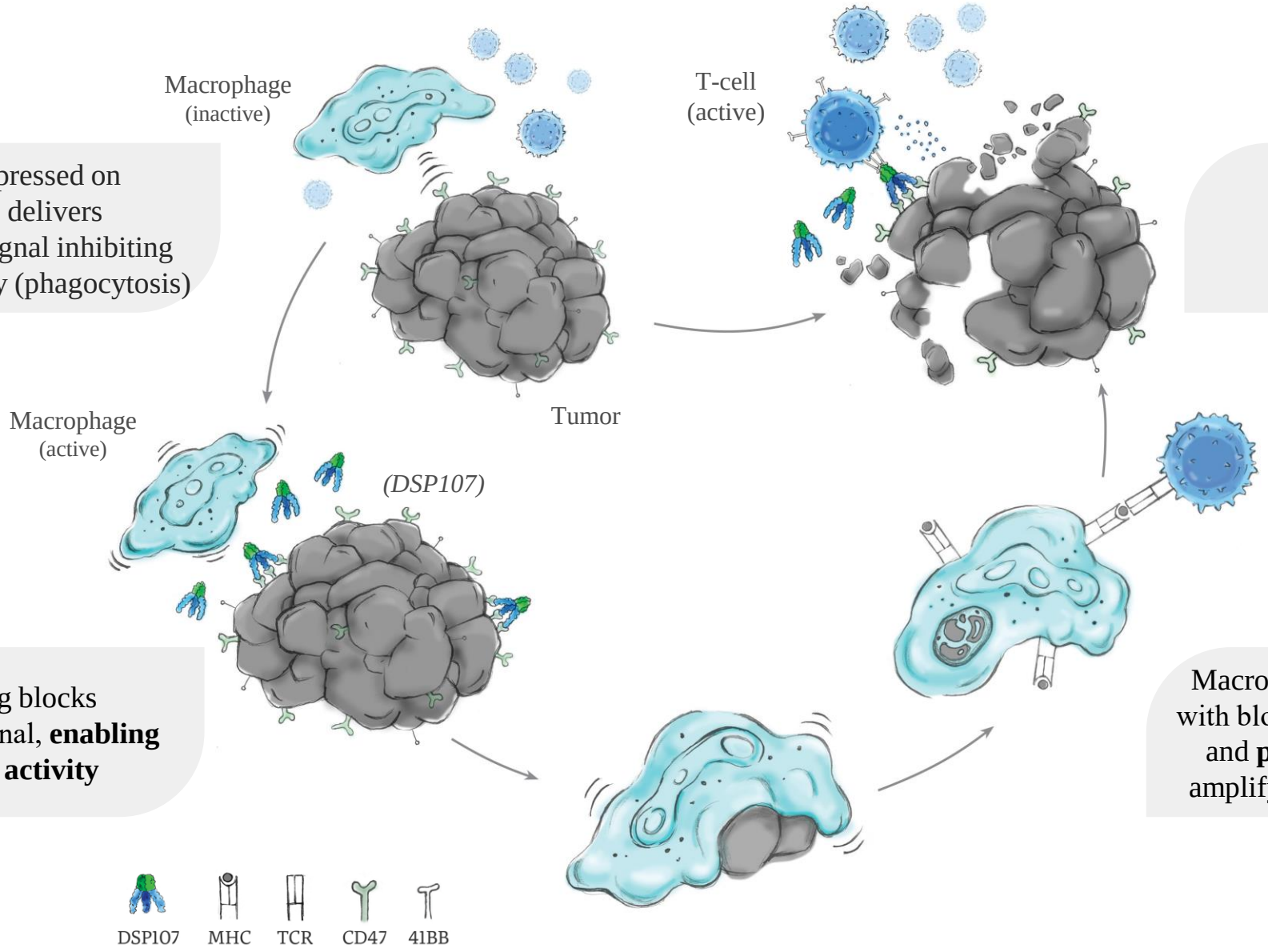
UNIQUE TRIMERIC STRUCTURE ENABLES TUMOR TARGETED 4-1BB CONDITIONAL ACTIVATION



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

SYNERGISTIC INNATE & ADAPTIVE IMMUNE ACTIVATION

CD47 overexpressed on cancer cells, delivers **“don’t eat me”** signal inhibiting macrophage activity (phagocytosis)



4-1BB mediated **T-cell** binding stimulates their **activation**

CD47 binding blocks “don’t eat me” signal, **enabling macrophage activity**

Macrophages ingest cancer cells with blocked “don’t eat me” signal and **present cancer antigens**, amplifying the immune response

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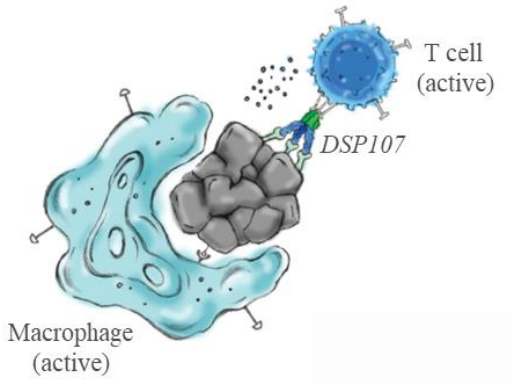
DSP107 WELL POSITIONED TO SHOW ANTI-CANCER ACTIVITY AS MONOTHERAPY AND IN COMBINATION THERAPIES

DSP107 monotherapy

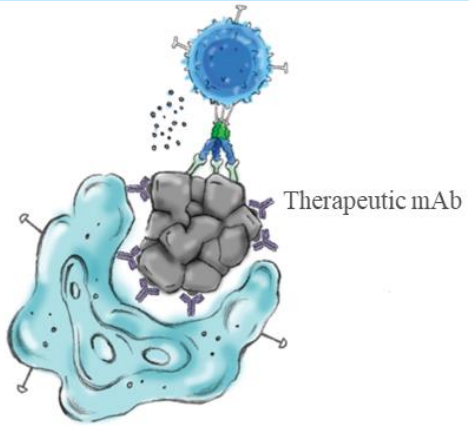
Triggers macrophage mediated phagocytosis and T cell cytotoxicity



DSP107 combination with therapeutic Antibodies IgG1 mAb's

(cetuximab, trastuzumab..)

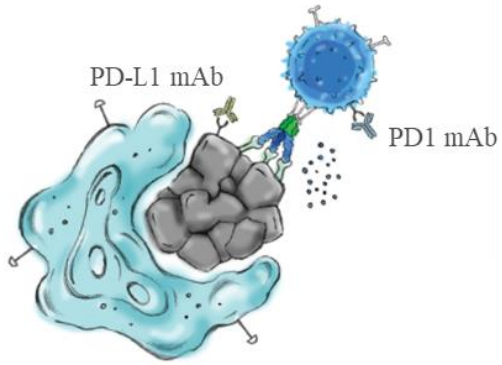
Enhances antibody-dependent cellular phagocytosis (ADCP)



DSP107 combination with PD1/PD-L1 Checkpoint Inhibitors

(atezolizumab, pembrolizumab…)

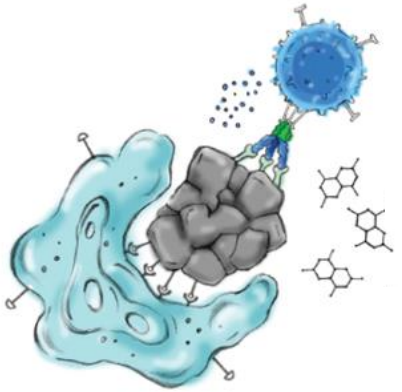
Enhances T cell activation



DSP107 combination with pro-apoptotic agents

(chemotherapy, hypomethylating agents and BCL2 inhibitors)

Increases "eat me" signals



DSP107 - DIFFERENTIATED CD47 TARGETING COMPOUND

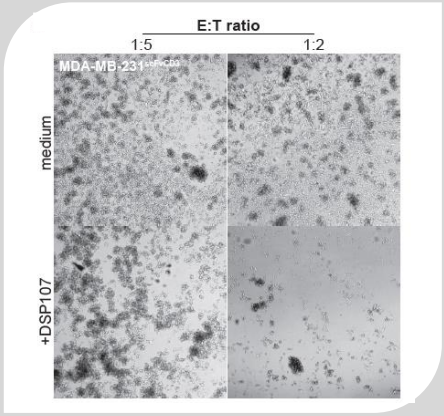
Next generation capabilities

Dual MOA
activates innate and adaptive immunity

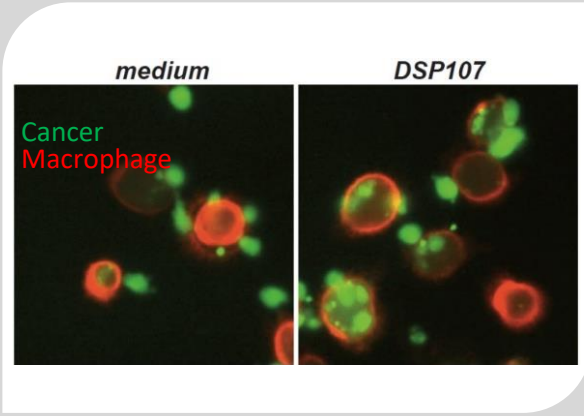
Excellent safety
without hematological toxicities

Strongly positioned
for treatment of solid and hematological malignancies

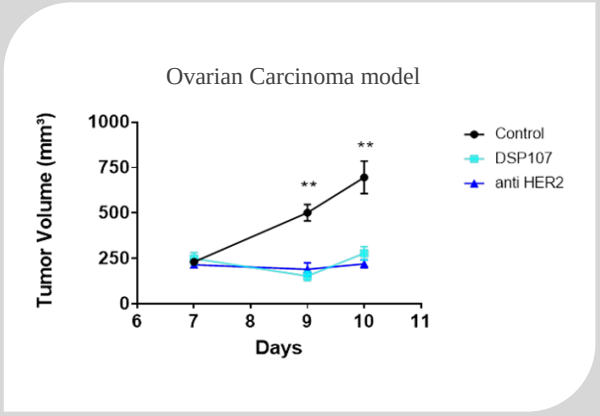
Unique and differentiated features



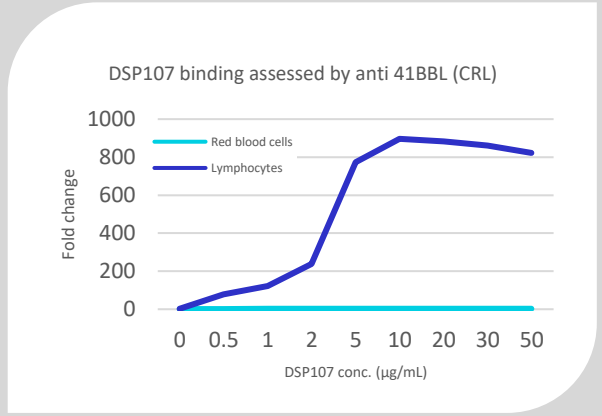
Activates T cells to secrete IFN-γ and augments their cancer cell killing potential



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

DSP107 – CLINICAL DEVELOPMENT

DSP107 – CLINICAL DEVELOPMENT STRATEGY

Advanced Solid Tumors

Phase 1 – Dose escalation (n = 30)

Accelerated dose escalation; Then 3+3

- DSP107
- DSP107 + atezolizumab

RP2D

Phase 2 – Expansion Cohort (n = 70)

- DSP107
- DSP107 + atezolizumab

Potential regulatory path

2L NSCLC patients who progressed on PD1/PD-L1 therapies

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

Hematological Malignancies

Phase I Dose escalation (n = 36)

Dose escalation N=6-8/cohort

- DSP107 ± azacytidine
- DSP107 + azacytidine + venetoclax

High risk R/R MDS/CMML and AML who failed up to 2 prior therapeutic regimes

RP2D

Phase 2 – Expansion
Details to be announced following EOPI meeting with the FDA

Lead site: MD Anderson Cancer Center

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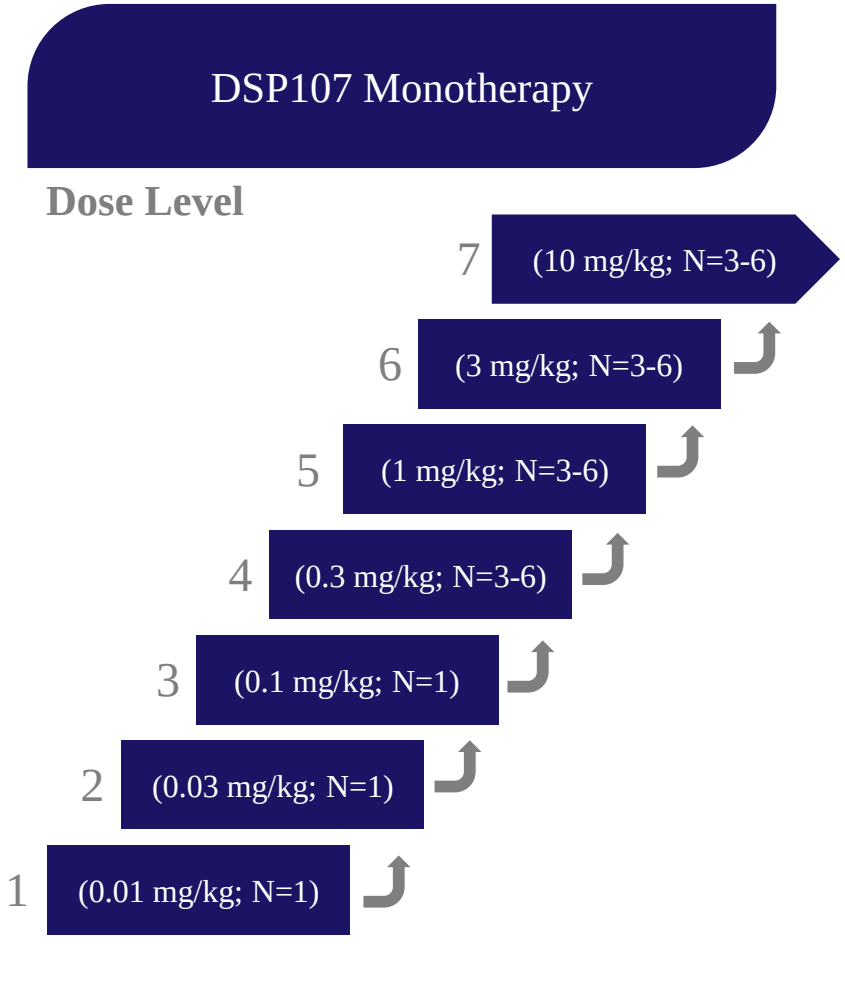
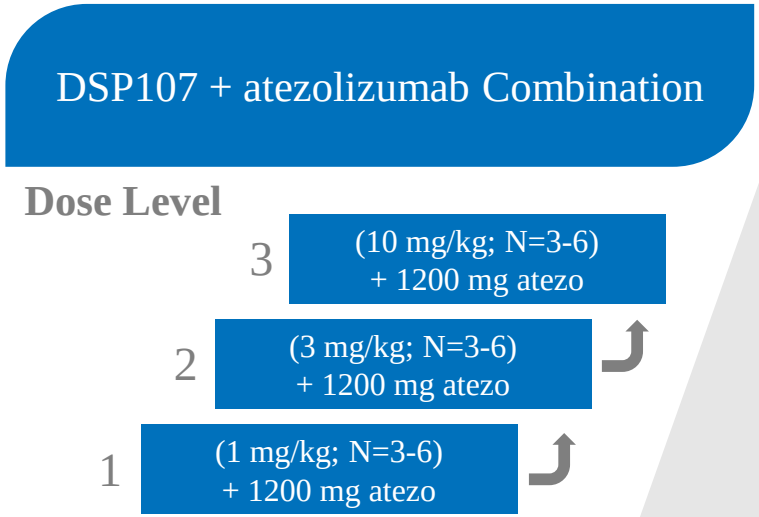
RP2D: Recommended Phase 2 dose; NSCLC: Non-small cell lung cancer; MDS: Myelodysplastic syndromes; AML: Acute myeloid leukemia

TRIAL DESIGN AND KEY INCLUSION CRITERIA

Part 1 – Monotherapy and Combination Dose Escalation





Trial Design:

- Patients with advanced solid tumors (N=30) not suitable for curative therapy and without approved treatment options
- IV administration once weekly
- Accelerated dose escalation in single patient cohorts followed by standard 3+3 design

Key Inclusion Criteria:

- Histologically confirmed advanced solid tumor with no approved therapeutic options
- Age 18 years or older
- ECOG performance status 0 or 1
- Measurable disease per RECIST v 1.1

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PATIENTS WITH ADVANCED SOLID TUMORS – NEARLY HALF FAILED PRIOR IMMUNOTHERAPY AND/OR COLD TUMORS

Characteristics	
Total number of patients	N = 17
Sex	6 (35%) ♀; 11 (65%) ♂
Age	Median 62 (Range 29-78)
Tumor types	
Colorectal	4 (24%)
Pancreas	4 (24%)
Head and Neck	3 (18%)
NSCLC	1 (6%)
Ovarian	1 (6%)
Rare tumor types	4 (24%)
Previous lines of therapy	Median 2 (Range 2-8)
PD1/PD-L1 experienced	8 (47%)

NO DLTs, HEMATOLOGICAL TOXICITIES OR HEPATO-TOXICITIES

Summary

- DSP107 doses up to and including 3 mg/kg considered safe and tolerated
- No DLTs and no treatment-related SAEs
- No hematological toxicities
- No hepato-toxicities
- Very few AEs considered related to DSP107 and almost all mild or moderate in severity
- Most related AEs Grade 1-2 in severity. Only 2 related Grade 3 AEs – transient hypertension and fatigue (at EOT visit)

Now enrolling patients to Dose Level 7 (10 mg/kg)

Treatment-Related AEs in ≥ 2 Patients	
Total No of Patients	**N = 17**
Treatment-related AEs (any grade)	**n (%)**
Any	12 (71)
Diarrhea	4 (24)
IRR*	3 (18)
Fatigue	3 (18)
Nausea	3 (18)
Constipation	2 (12)

*IRRs Grade 1-2 in severity. Easily abrogated in subsequent infusions by reduced rate of infusion and concomitant IV fluids.

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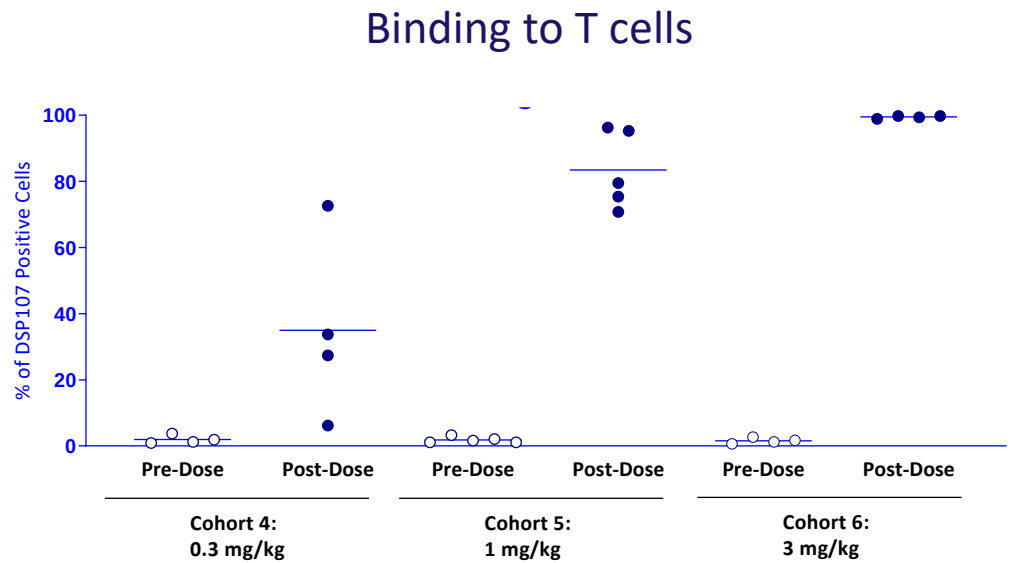
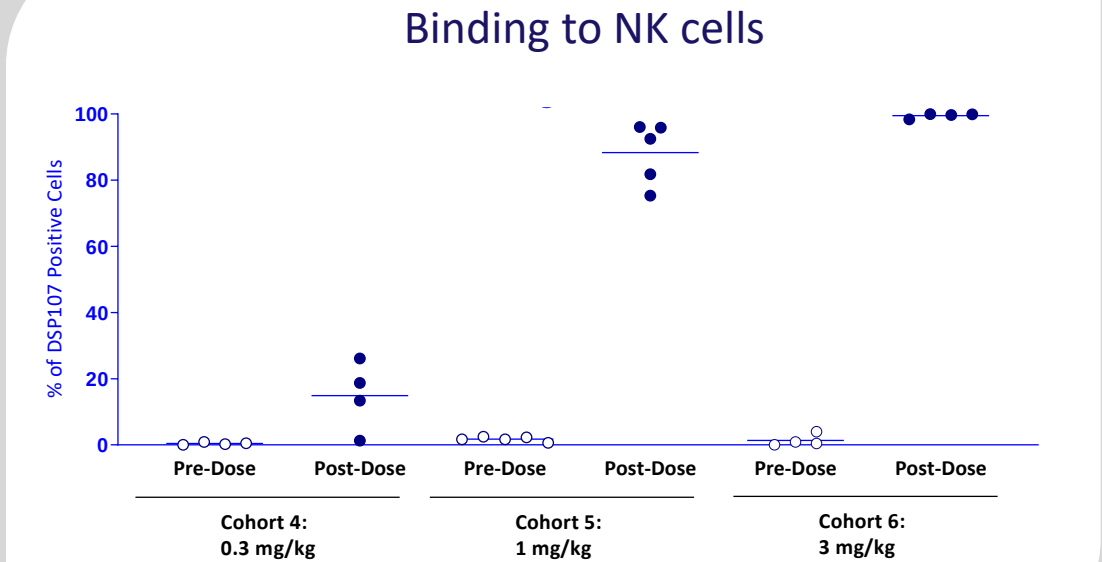
FULL TARGET ENGAGEMENT ON CIRCULATING IMMUNE CELLS

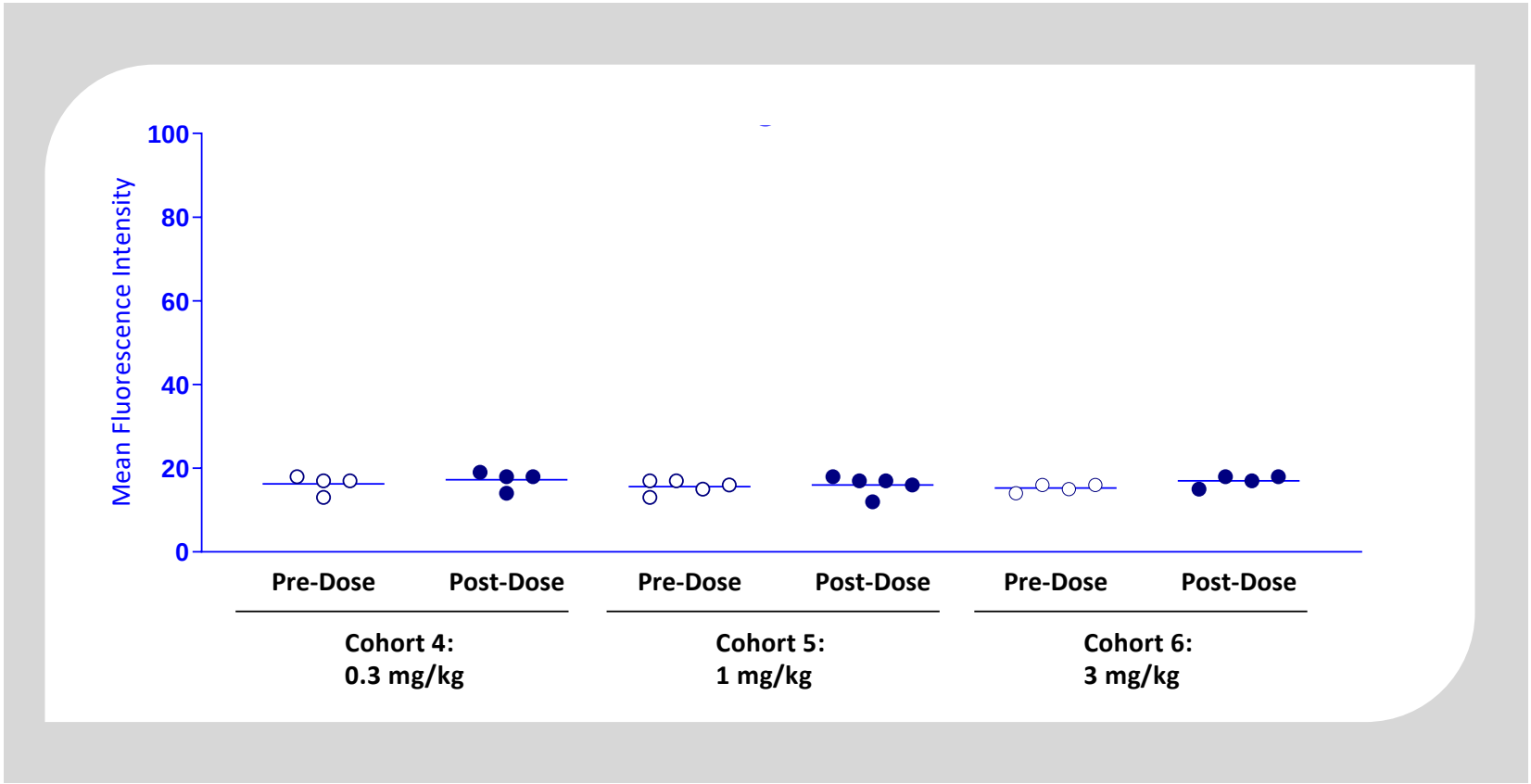
- Dose dependent target engagement achieved across T cells and NK cells
- 100% receptor occupancy on circulating immune cells observed at ≥3 mg/kg



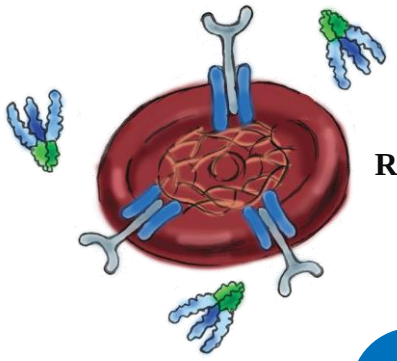


RECEPTOR OCCUPANCY DATA CONFIRMS LACK OF RBC BINDING

No binding to red blood cells at doses 0.3, 1 and 3 mg/kg

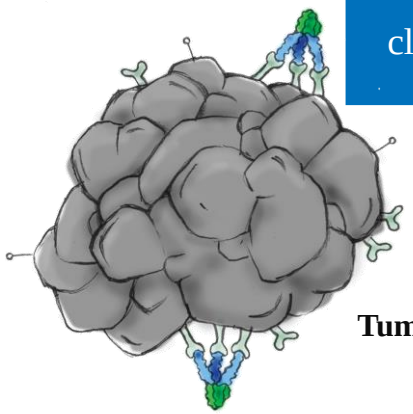


On RBCs CD47 protein complex is anchored to cytoskeleton resulting in its immobilization and low affinity of DSP107 to the monomeric CD47



Red Blood Cell

High affinity/avidity of DSP107 to CD47 clusters on cancer cells



Tumor

INCREASED NECROSIS IN PAIRED BIOPSIES AFTER DSP107

Key Findings:

- Notable increase in necrotic tumor tissue was observed in 3 out of 4 paired biopsies compared to screening
- Necrosis was associated with immune cell infiltration
- No evidence of vascular necrosis

Patient Number	Dose (mg/kg)	Tumor Type	Timepoint	% Necrosis
11-001	0.3	Colorectal	Screening	0
			6 weeks	**65**
11-002	0.3	Colorectal	Screening	2
			6 weeks	**35**
10-003	1	Pancreatic	Screening	10
			6 weeks	**50**
13-005	1	Pancreatic	Screening	4
			6 weeks	3

All biopsies collected from hepatic metastases pre-treatment and following cycle 2 (6 doses). H&E stained slides assessed by independent, blinded pathologist.

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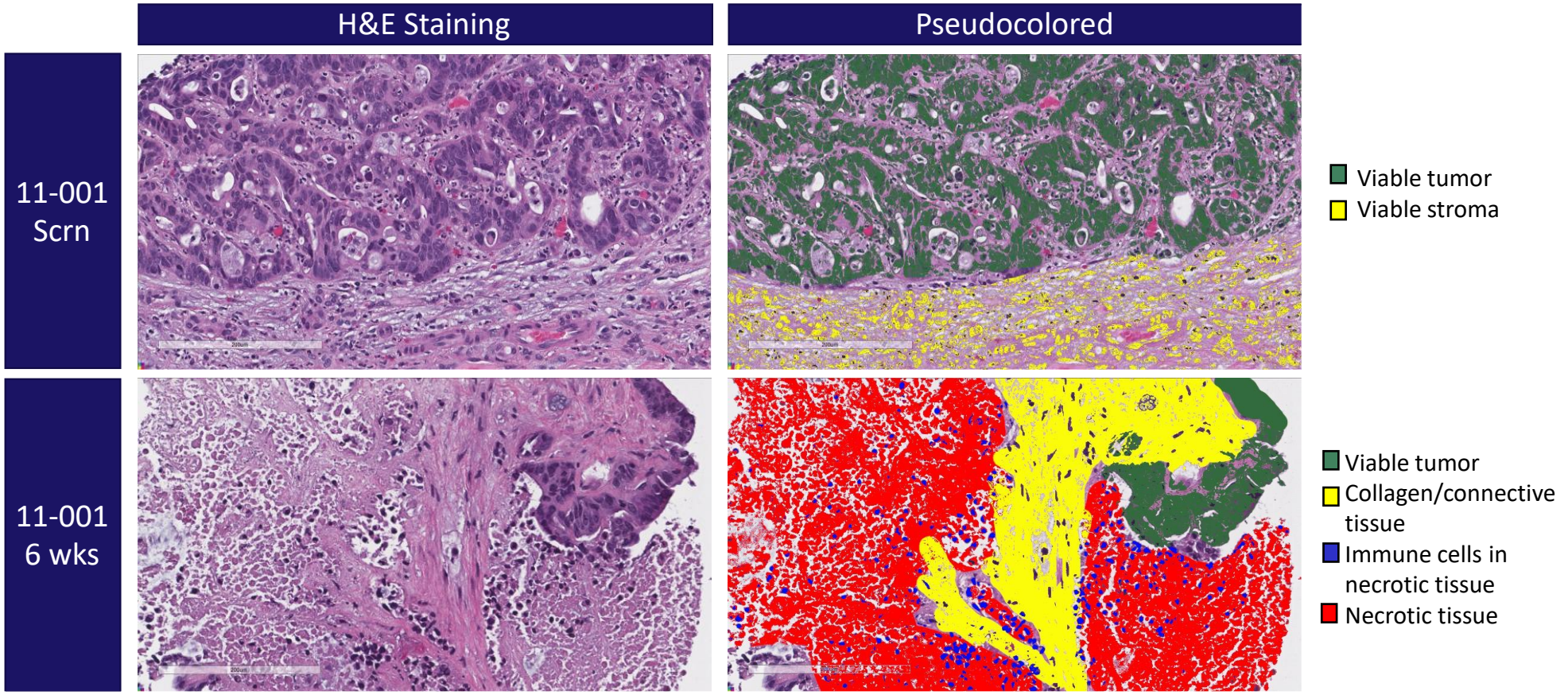
CASE STUDY: NECROSIS ASSOCIATED WITH INCREASED IMMUNE CELL INFILTRATION AFTER DSP107



~ 65%

Necrotic tumor tissue

6-wks post DSP107 treatment

Paired biopsy from colon carcinoma patient (11-001) in dose level 4 (0.3 mg/kg) pre- treatment and following cycle 2 (6 doses). No necrosis at baseline.

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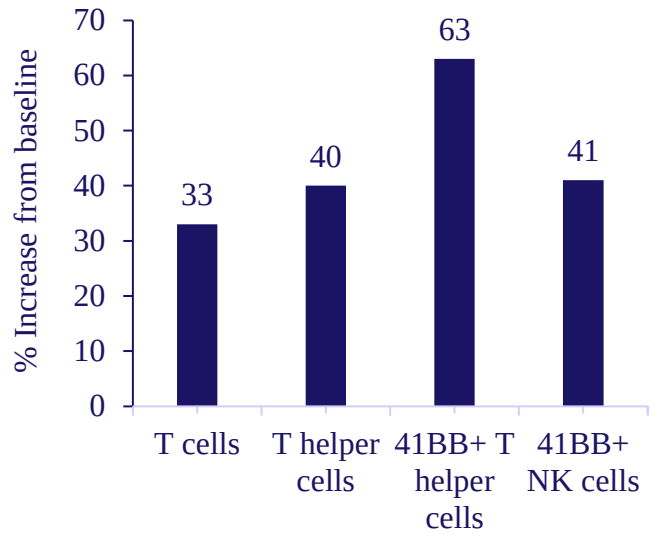
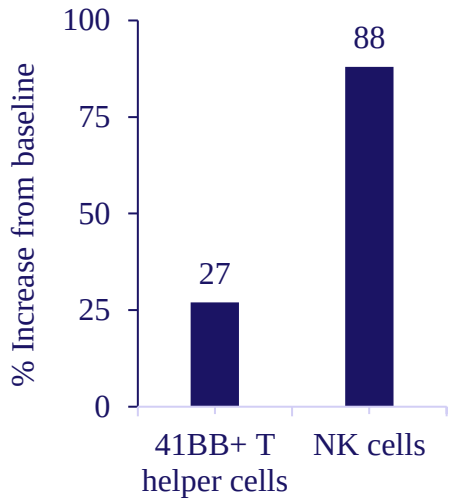
CASE STUDY: INCREASED IMMUNE INFILTRATION AFTER DSP107

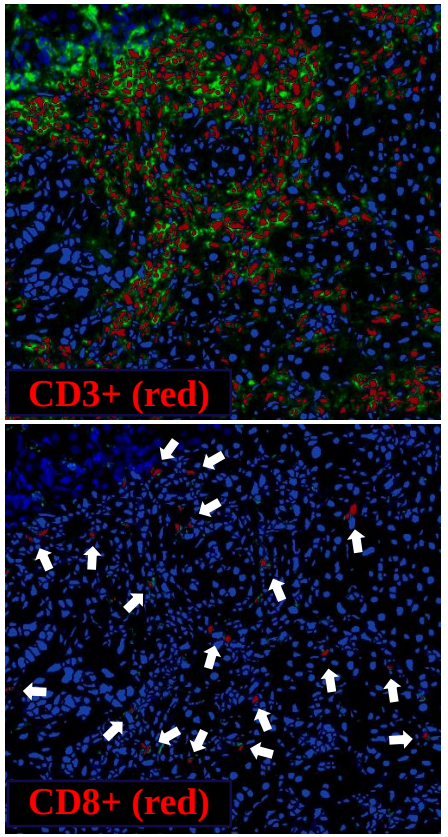
6-wks post DSP107 treatment

Infiltration in tumor compartment



Infiltration at tumor margin





Paired biopsy from colon carcinoma patient (11-001) in dose level 4 (0.3 mg/kg) pre- treatment and following cycle 2 (6 doses). Quantification of multiplex image analysis from biopsy stains.

Significant infiltration of T cells and NK cells in both the tumor compartment and at the tumor margin following DSP107 treatment

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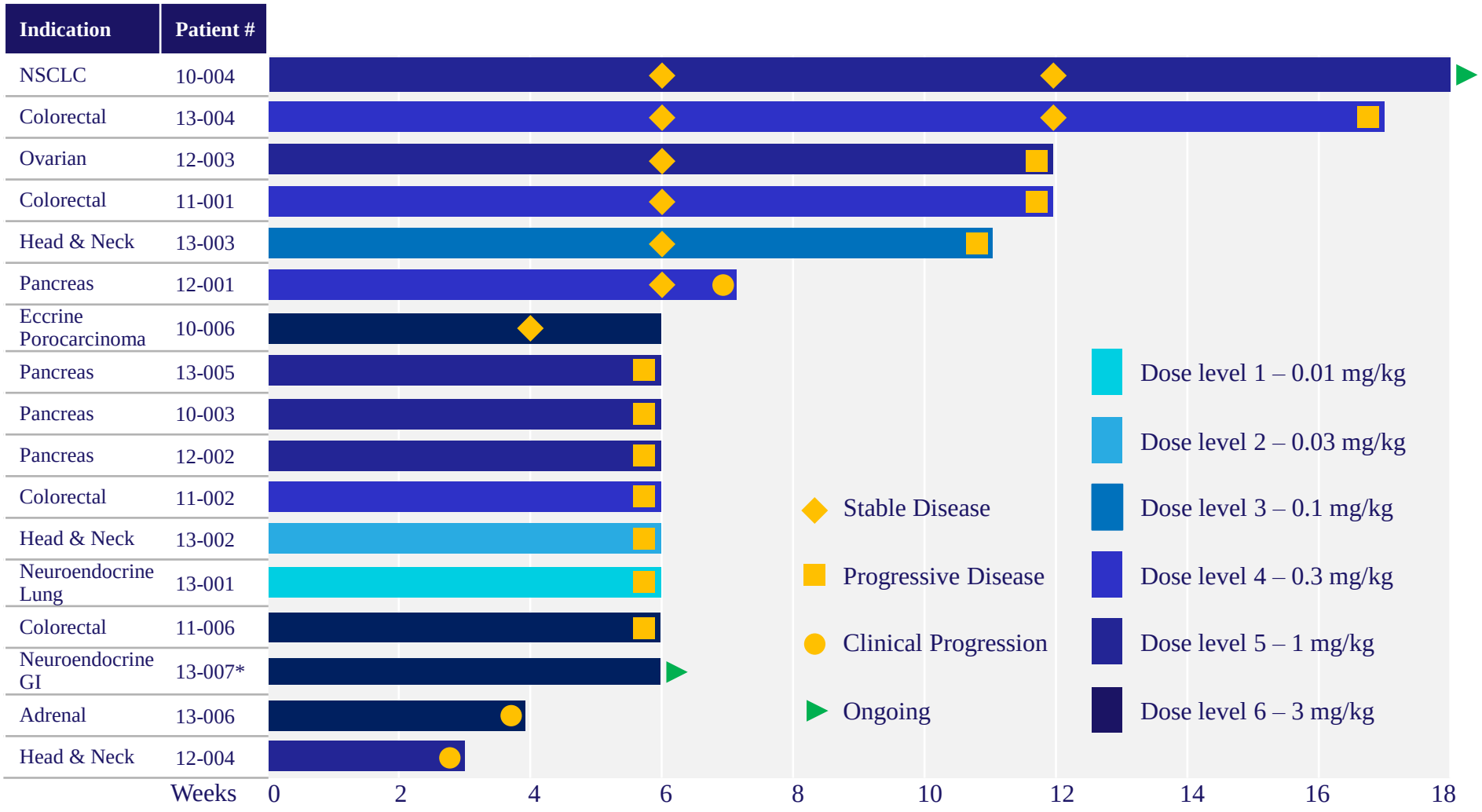
BEST OVERALL RESPONSE TO DATE AFTER DSP107 MONOTHERAPY



Summary

Best response from 16 patients evaluable to date:

SD = 7 (44%)

PD = 9 (56%) incl. 2 patients withdrawn before 1st CT scan due to clinical progression

*Has not reached first CT scan evaluation. †Includes two patients withdrawn before first CT scan due to clinical progression.

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DSP107 PHASE 1 DATA: FAVORABLE SAFETY AND PRELIMINARY ACTIVITY IN SOLID TUMORS

Clinical Overview

- DSP107 alone and in combination with atezolizumab is being evaluated in a dose escalation trial
- 17 patients with diverse solid tumors have been treated as of data cut-off on Nov. 4th 2021, with 16 patients evaluable for efficacy analysis
- Now enrolling patients to cohort 7 (10 mg/kg)

Key Findings

- Mostly low-grade AEs with no DLTs, no hematological toxicities and no hepato-toxicities
- Receptor occupancy data confirming lack of RBC binding and immune cell engagement
- Increase immune cell infiltration into the tumor with increased tumor necrosis

Further evaluate safety and preliminary efficacy of DSP107 alone up to dose level 7, as well in combination with atezolizumab

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KEY UPCOMING MILESTONES

Program MOA	Indication	2021	2022		2023	
		2H	1H	2H	1H	2H
DSP107 CD47 inhibitor 4-1BB activator	**Solid Tumors, NSCLC**	Initial Ph I/II mono data	Initial Ph I/II combo data	Ph I/II NSCLC Interim results		Ph I/II NSCLC Topline results
	AML/MDS			Ph I AML/MDS Interim results		Ph I AML/MDS Topline results
DSP502 PVR inhibitor PD-L1 inhibitor Active IgG1 Fc	**Oncology**		IND-enabling activities		File IND	
DSP216 CD47 inhibitor HLA-G inhibitor Inactive Fc	**Oncology**		IND-enabling activities			File IND

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Q&A