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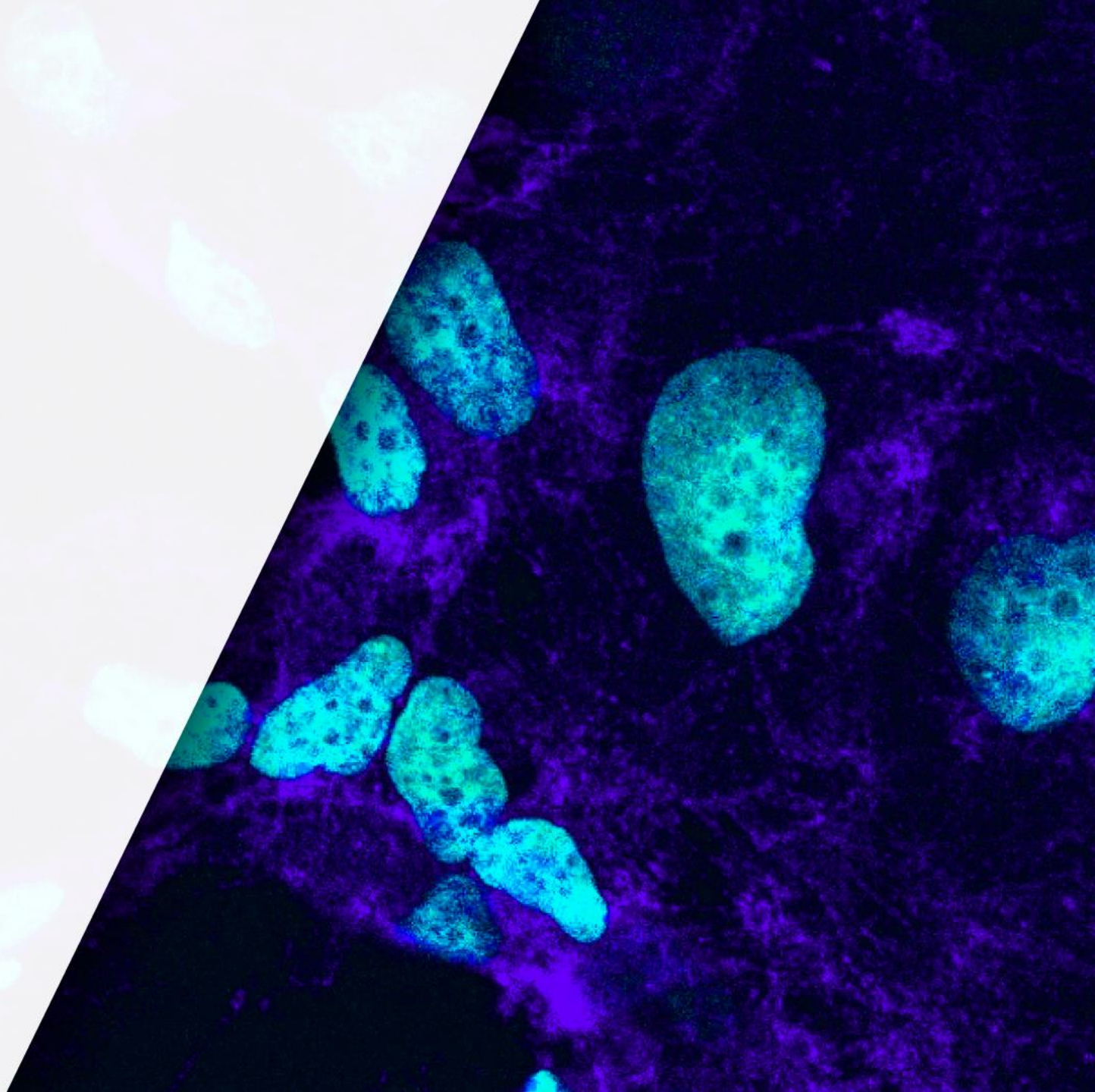
UNMASKING CANCER CELL CAMOUFLAGE

COMPANY PRESENTATION | May 2022



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Company Highlights

Differentiated Pipeline

- Novel, next generation potential across **three programs in solid and hematological malignancies**
- Lead candidate DSP107 designed to target **CD47/SIRPα and 41BB specifically and conditionally** in the tumor microenvironment
- Versatile MIRP platform **targeting innate and adaptive immunity**

Multiple Near-Term Catalysts

- **Anticipated data readouts** for DSP107 in 2022 in NSCLC (phase II) and in hematological malignancies (phase I)
  - Dose escalation portion of Phase I/II data **demonstrates favorable preliminary safety profile and monotherapy activity** in solid tumors
- Expected IND filing for DSP502 and DSP216 in 2023 and 2024

Cash Runway

- Raised **~$100 million** to date
- Investors include **aMoon, BVF, DAFNA, Cancer Focus Fund**

Experienced Leadership

- **Experienced leadership** and executive team with track record of success
- Supported by leading scientific advisory board

Experienced Leadership Team

Aron Knickerbocker, MBA

Board Chairman

aulos

Genentech

FivePrime

Bristol Myers Squibb

AMGEN

Yaron Pereg, PhD

Chief Executive Officer

Genentech

BIOLINERX

CELLECT

Tomer Cohen, MBA

Chief Financial Officer

LOCUST WALK

BARCLAYS

Goldman Sachs

Adam Foley-Comer, MD

Chief Medical Officer

Roche

BIOLINERX

QUINTILES

IMMUNE Pharmaceuticals

Ayelet Chajut, PhD

Chief Technology Officer

Pluristem

ROSETTA GENOMICS

Quark Pharmaceuticals

compugen

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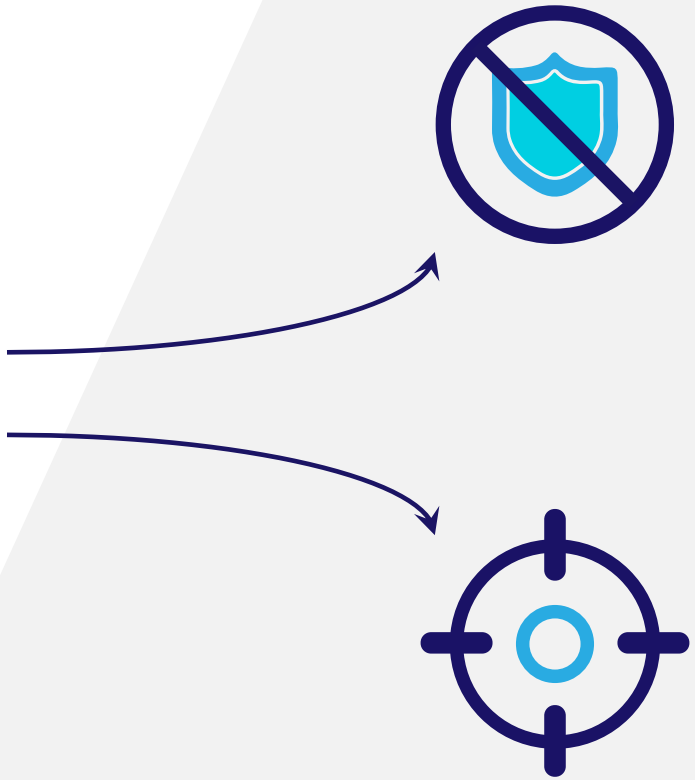
Effectively treating cancer requires a multifaceted approach



Selectively disabling cancer defense mechanism

WHILE

Recruiting a local targeted immune attack

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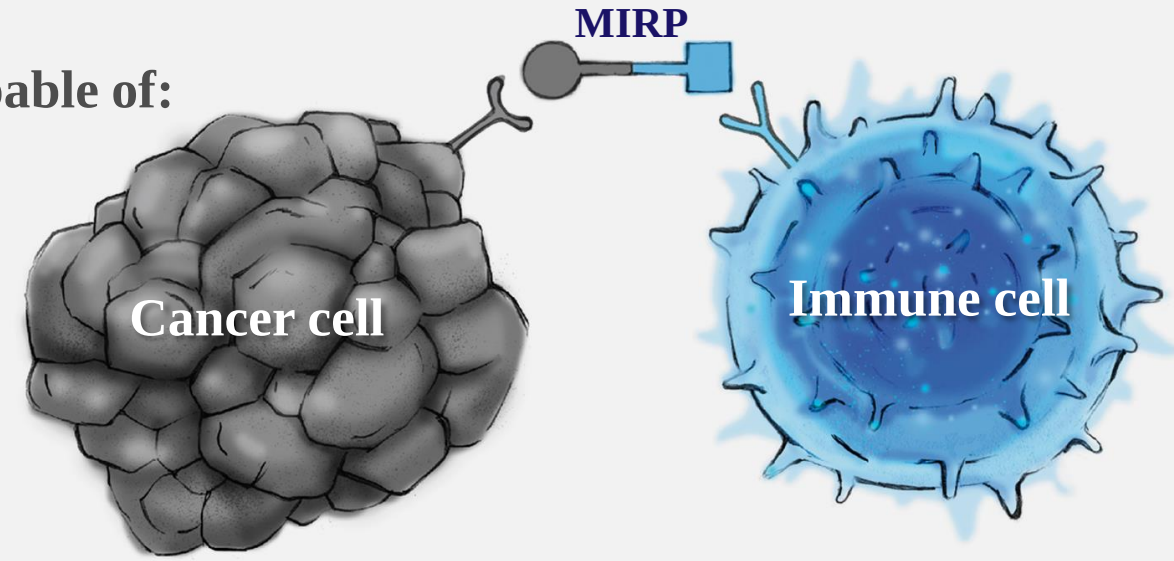
Versatile Multifunctional Immunotherapeutic Platform for Solid and Hematological Malignancies

MULTIFUNCTIONAL IMMUNE RECRUITMENT PROTEIN (MIRP)

Enabled us to design dual-targeting fusion proteins capable of:

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



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MIRP Platform - Customized, Modular and Flexible Design

ECD Type I protein
Ig superfamily
- ✓ Receptors
- ✓ Ligands
- ✓ Tumor antigens

(e.g. PD-L1, PVR, CD47, HLA-G)

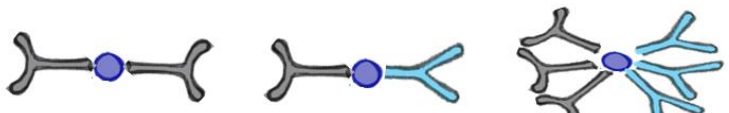
ECD Type I or II protein
Ig superfamily or TNF superfamily
- ✓ Receptors
- ✓ Ligands
- ✓ Immune checkpoint

(e.g. 4-1BBL, TIGIT, PD1, LILRB)

Linker of choice
- ✓ Short peptide
- ✓ Single amino acid
- ✓ Unstructured ECD sequence
- ✓ Fc region (IgG1 or IgG4)

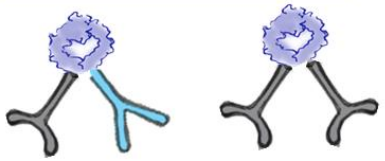
Customized composition

DSP



Type I – Type I | Type I – Type II | Trimeric Type I – Type II

DSP-Fc



Type I – Type II | Type I – Type I

Enhanced, Tumor-Localized Immune Cell Recruitment & Activation With Potential to Improve Safety and Efficacy

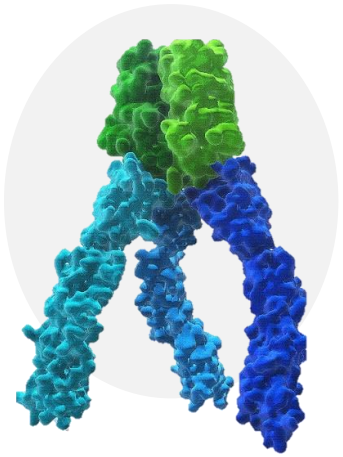
DSP (Dual Signaling Protein)

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)

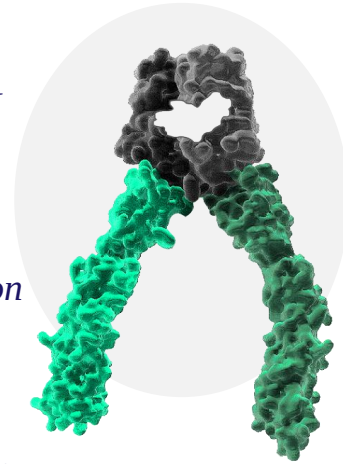
Dual checkpoint inhibition for tumoricidal response

DSP502

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

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

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

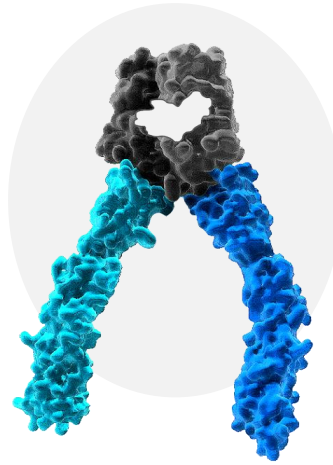


DSP216

CD47 inhibitor – *Avidity driven for cancer specific blocking*

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

Inactive Fc – *Half-life extension potential*



Wholly Owned, Focused and Differentiated Pipeline

Program	Targets	Indications	Discovery	Preclinical	Phase 1	Phase 2	Phase 3	Anticipated Milestones
DSP107	CD47 4-1BB	Advanced Solid Tumors, NSCLC			DSP107 ± atezolizumab*			Phase I/II interim data H2/22
		AML / MDS			DSP107 ± azacitidine + venetoclax			Phase Ib interim data H2/22
DSP502	PVR PD-L1	Oncology						IND submission 2023
DSP216	HLA-G CD47	Oncology						IND submission 2024

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 cells**
Mechanism of Action	**Unleash mϕ via ‘Don’t Eat Me’ blockade, Activate 4-1BB+ T cells**

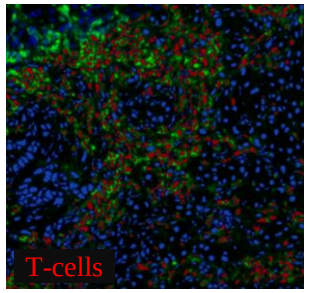
DSP107 – Differentiated CD47 Targeting Compound

Dual MOA
designed to activate innate and adaptive immunity

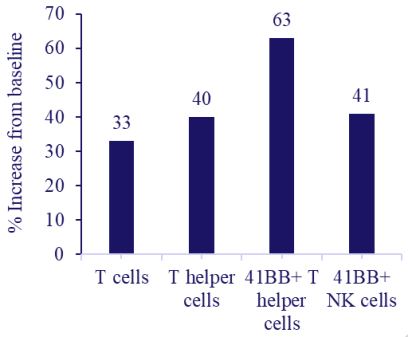
Favorable safety
without hematological toxicities observed

Strongly positioned
for treatment of solid and hematological malignancies

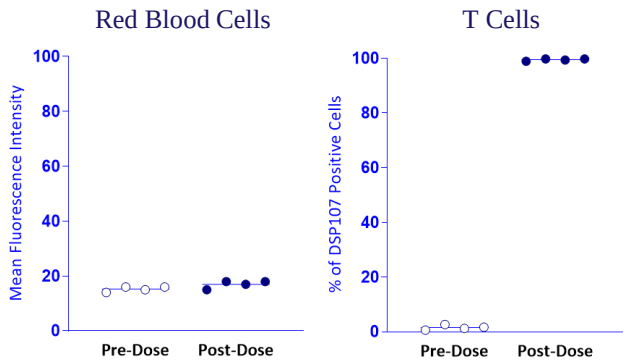
Phase I data demonstrates unique and differentiated features



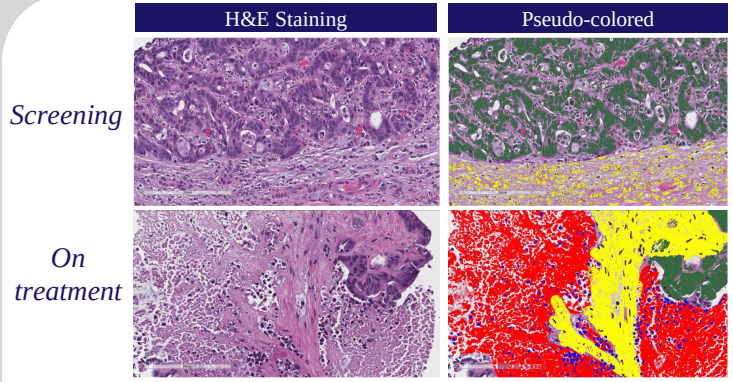
Post DSP107 treatment biopsy data



Triggering T and NK-cell infiltration into tumor microenvironment



No binding to red blood cells observed



Increasing tumor necrosis (red) and immune cell infiltration (blue)

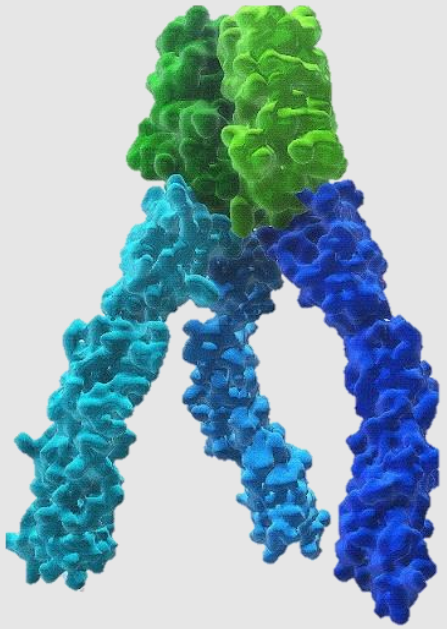
Trimeric Structure Design For Tumor Selectivity and Improved Safety

Trimeric ligand ends designed to enable:

1. Cancer selective binding to overexpressed CD47 on tumor cells, minimizing RBC binding and associated toxicities

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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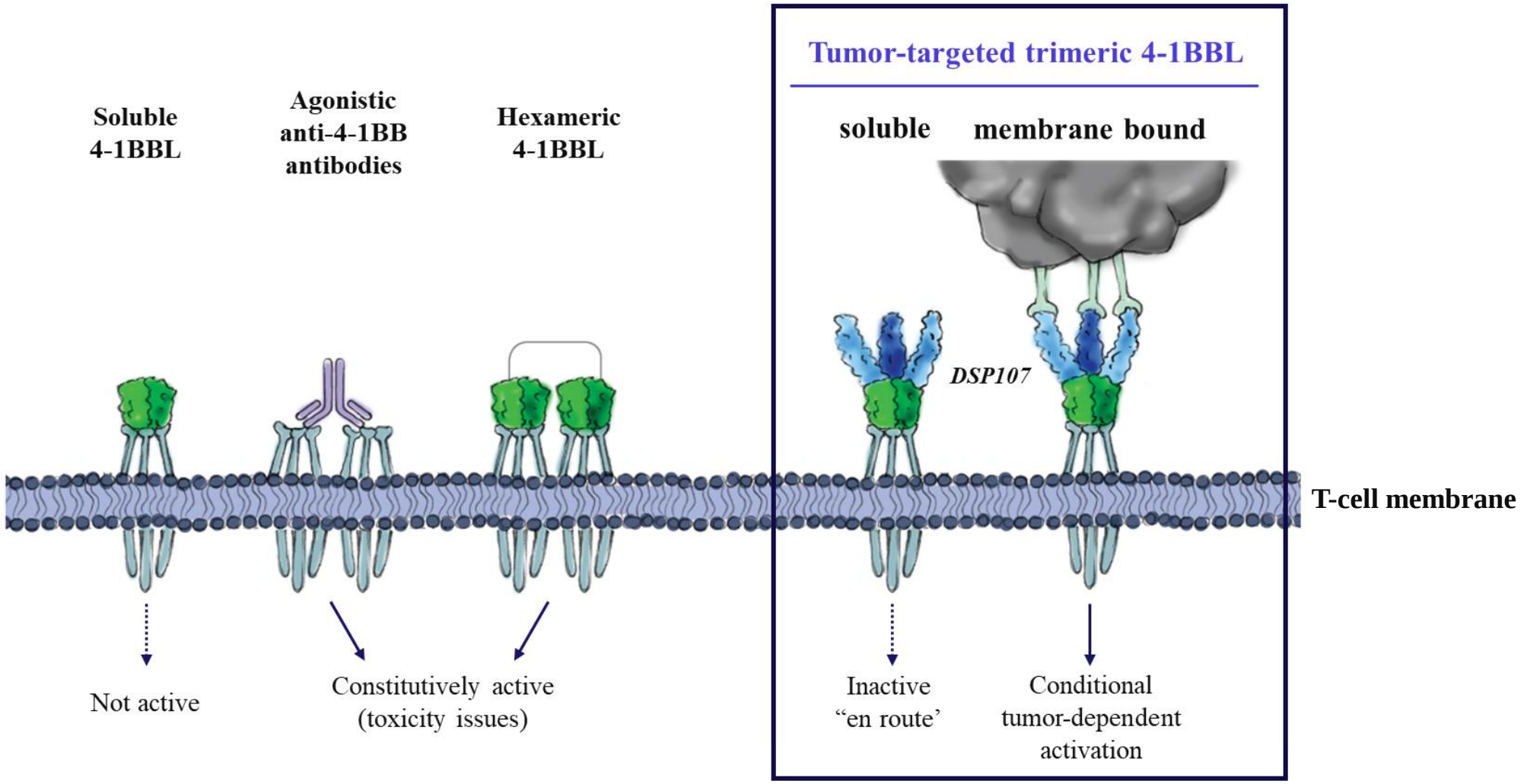
DSP107 Structure Designed to Safely Unlock 4-1BB Potential

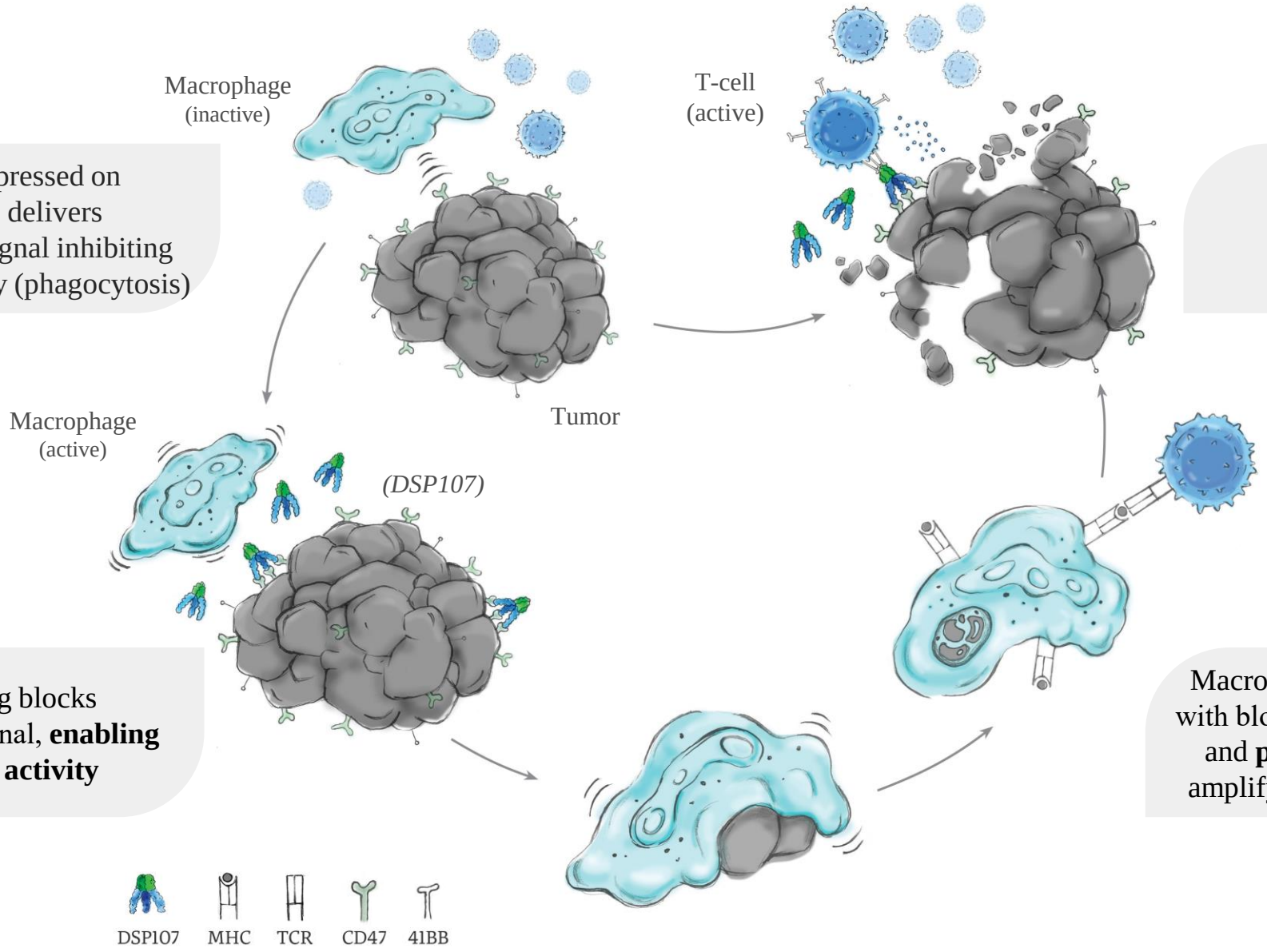
Phase I/II Data Demonstrated Absence of 4-1BB Related Hepatotoxicity



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

Designed for 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**

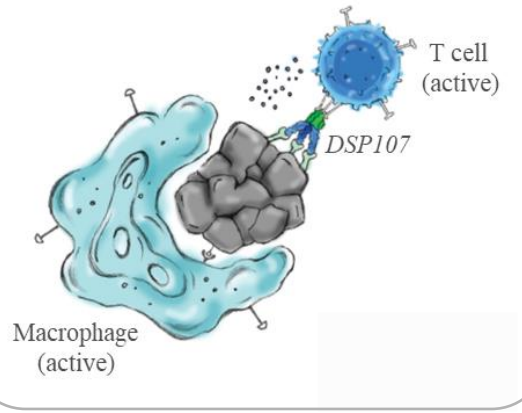
CD47 binding blocks “don’t eat me” signal, **enabling macrophage activity**

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

DSP107 Potential as a Monotherapy and in Combination Therapies

DSP107 monotherapy

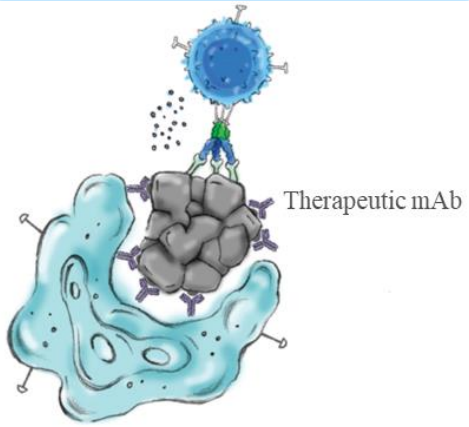
Triggers macrophage mediated phagocytosis and T cell cytotoxicity



DSP107 combination with therapeutic Antibodies IgG1 mAbs

(cetuximab, trastuzumab…)

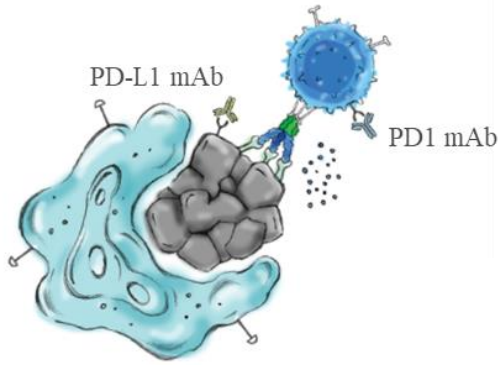
Enhances antibody-dependent cellular phagocytosis (ADCP)



DSP107 combination with PD1/PD-L1 Checkpoint Inhibitors

(atezolizumab, pembrolizumab…)

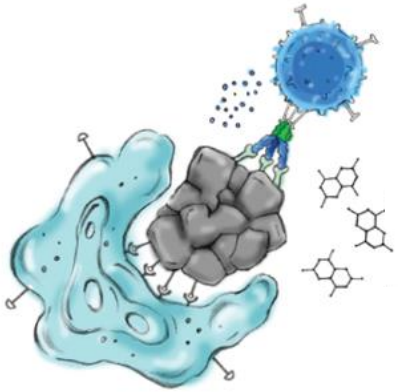
Enhances T cell activation



DSP107 combination with pro-apoptotic agents

(chemotherapy, hypomethylating agents and BCL2 inhibitors)

Increases “eat me” signals



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Preclinical Studies Support Differentiated Potential Dual MoA

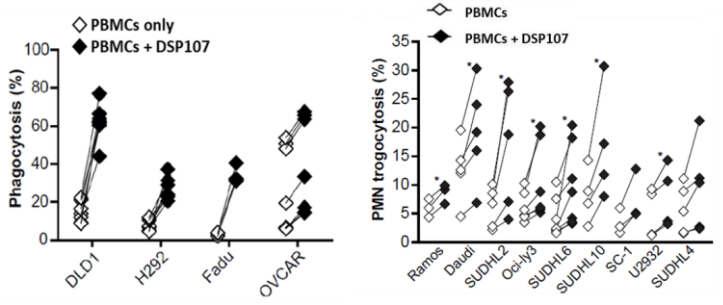
Dual MOA
designed to activate innate and adaptive immunity

Favorable safety
without hematological or hepato-toxicities in NHP observed

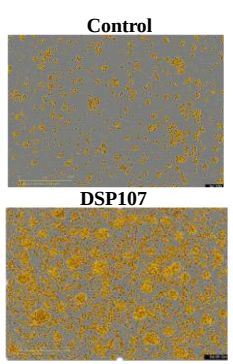
Monotherapy potential
for treatment of solid and hematological malignancies

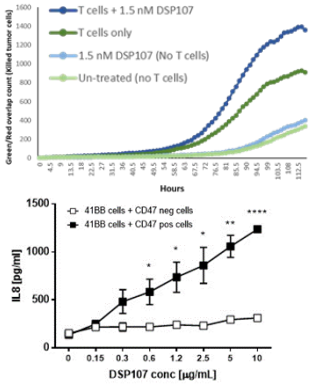
Comprehensive preclinical package demonstrated differentiated features

Carcinoma and Lymphoma cell lines



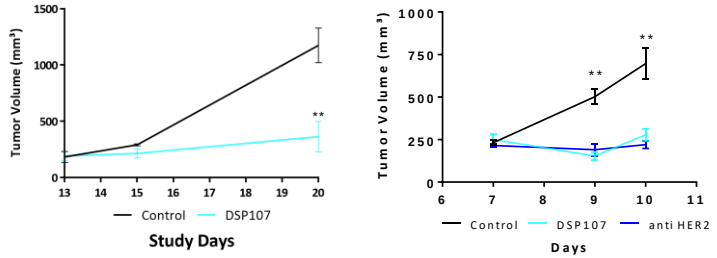
Triggering cancer cell death by phagocytosis as a single agent





Inducing 4-1BB activation, T-cell proliferation and killing potential against cancer cells

Lymphoma and Ovarian Carcinoma models



Demonstrating single agent anti tumor activity in mice models

DSP107 in Clinical Trials for Advanced Solid and Hematological Malignancies

Advanced Solid Tumors

Phase I/II – Dose escalation (n ~ 30)

Accelerated dose escalation; Then 3+3

- DSP107
- DSP107 + atezolizumab

RP2D

Phase I/II – Expansion Cohort (n = 70)

- DSP107
- DSP107 + atezolizumab

Potential indication

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 Ib – Dose escalation (n ~ 36)

Dose escalation N=6-8/cohort

- DSP107 ± azacytidine
- DSP107 + azacytidine + venetoclax

High risk R/R MDS/CMML or AML patients who failed up to 2 prior lines

RP2D

Planned phase 2 – Expansion
Details to be announced following EOP1 meeting with the FDA

Lead site: MD Anderson Cancer Center

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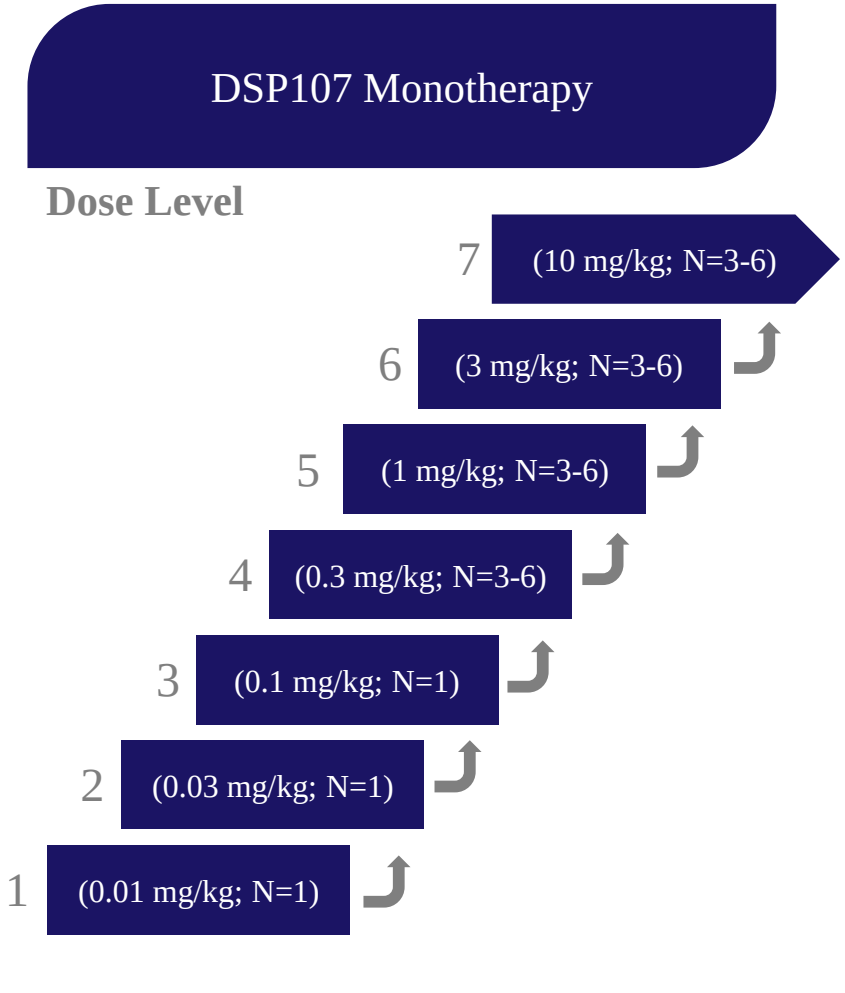
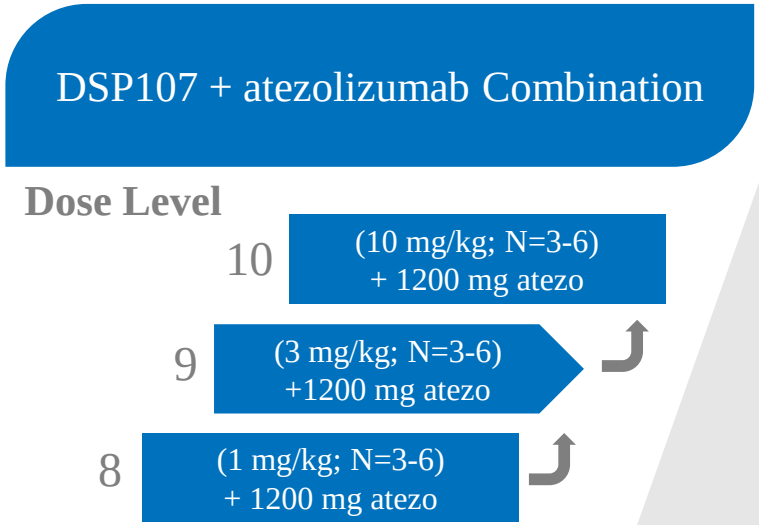
RP2D: Recommended Phase 2 dose; NSCLC: Non-small cell lung cancer; MDS: Myelodysplastic syndromes; AML: Acute myeloid leukemia

TRIAL DESIGN AND KEY INCLUSION CRITERIA

Part 1 – Monotherapy and Combination Dose Escalation in Advanced Solid Tumor Patients





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 = 23 (cohorts 1 – 7)
Sex	10 (43%) ♀; 13 (57%) ♂
Age	Median 63 (Range 29-78)
Tumor types	
Colorectal	7 (30%)
Pancreas	5 (22%)
Head and Neck	3 (13%)
NSCLC	1 (4%)
Ovarian	2 (9%)
Rare tumor types	5 (22%)
Previous lines of therapy	Median 3 (Range 2-8)
PD1/PD-L1 experienced	11 (48%)

Well Tolerated Without DLTs, Hematological or Hepato-Toxicities

Summary

- DSP107 doses up to and including 10 mg/kg well 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 treating patients with DSP107 and Atezolizumab combination

Treatment-Related AEs in ≥ 2 Patients	
Total No of Patients	**N = 23 (cohorts 1 - 7)**
Treatment-related AEs (any grade)	**n (%)**
Any	16 (70)
IRR*	6 (26)
Diarrhea	4 (17)
Fatigue	4 (17)
Nausea	3 (13)
Constipation	2 (9)

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

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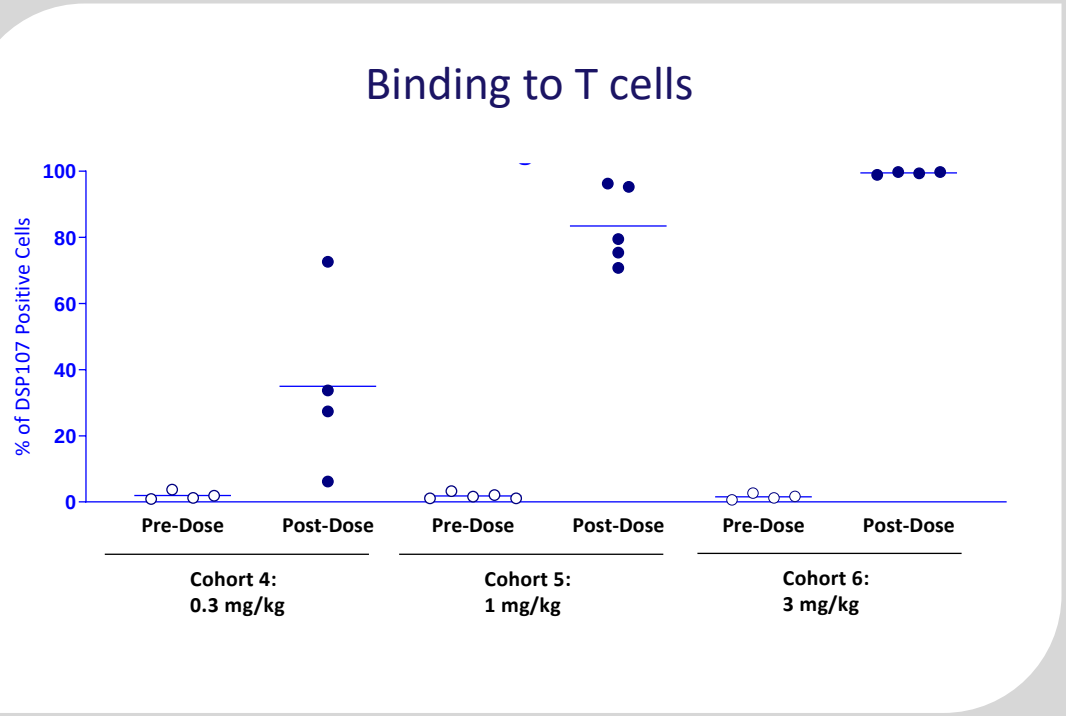
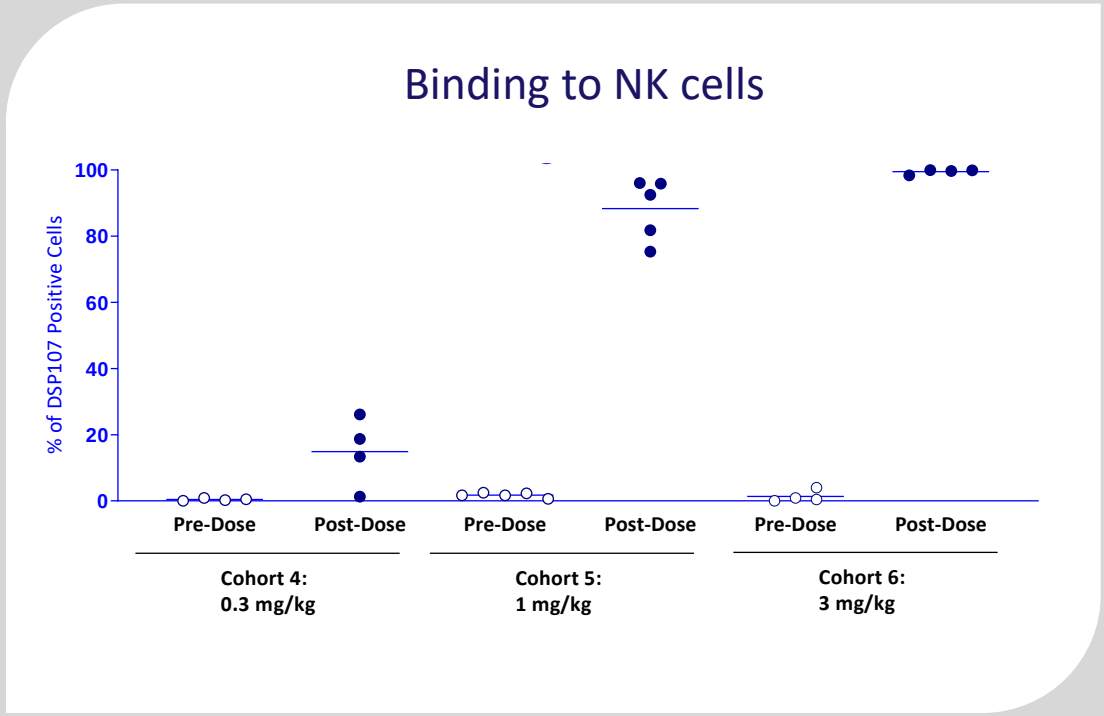
Receptor Occupancy Data Showed Immune Cell Target Engagement With No RBC Binding

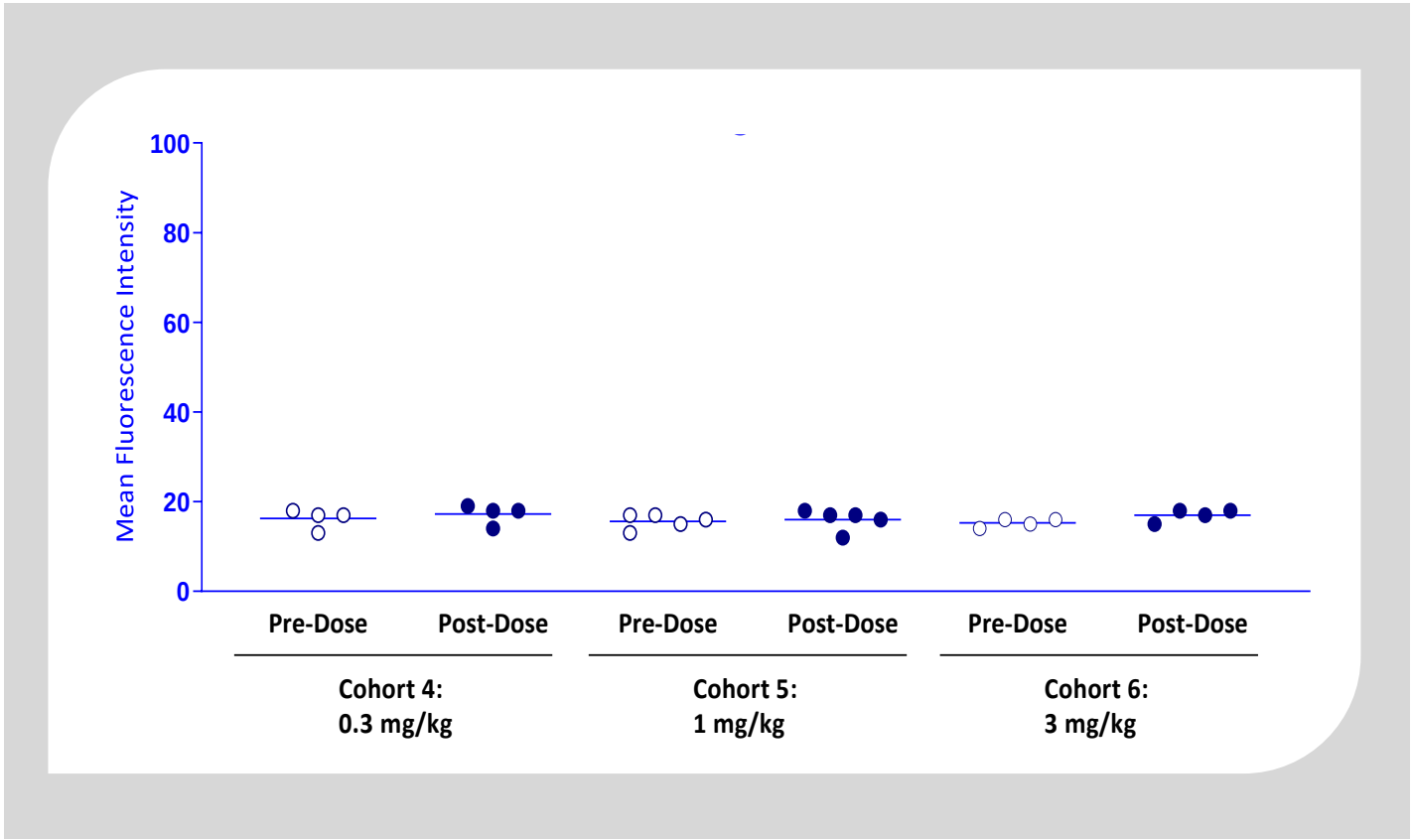
Dose dependent target engagement achieved with 100% receptor occupancy on circulating immune cells observed at 3 mg/kg

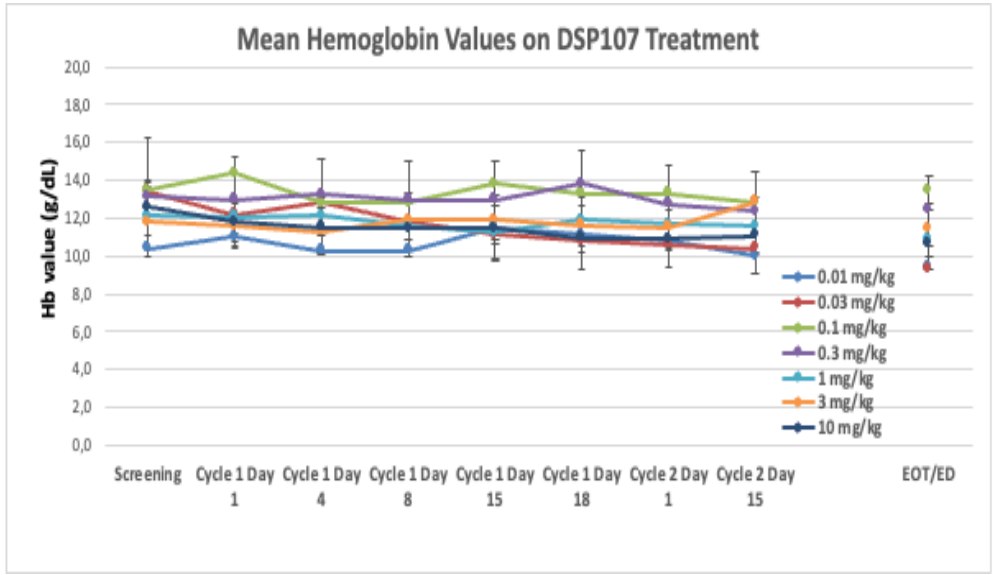




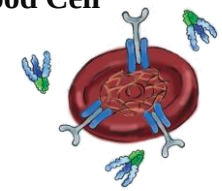
DSP107 Does Not Bind Red Blood Cells

Resulting in Favorable Safety Profile With No Anemia or Antigen Sink Issues





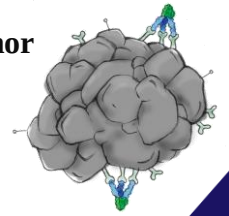
Red Blood Cell



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

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

Tumor



DSP107 Treatment Resulted in Tumor Necrosis

Key paired biopsies data

- All biopsies collected from hepatic metastases pre-treatment and following cycle 2 (6 doses)
- H&E stained slides assessed by independent, blinded pathologist
- In 3 out of 4 paired biopsies significant increase in necrotic (dead) tumor tissue was observed
- Necrosis 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

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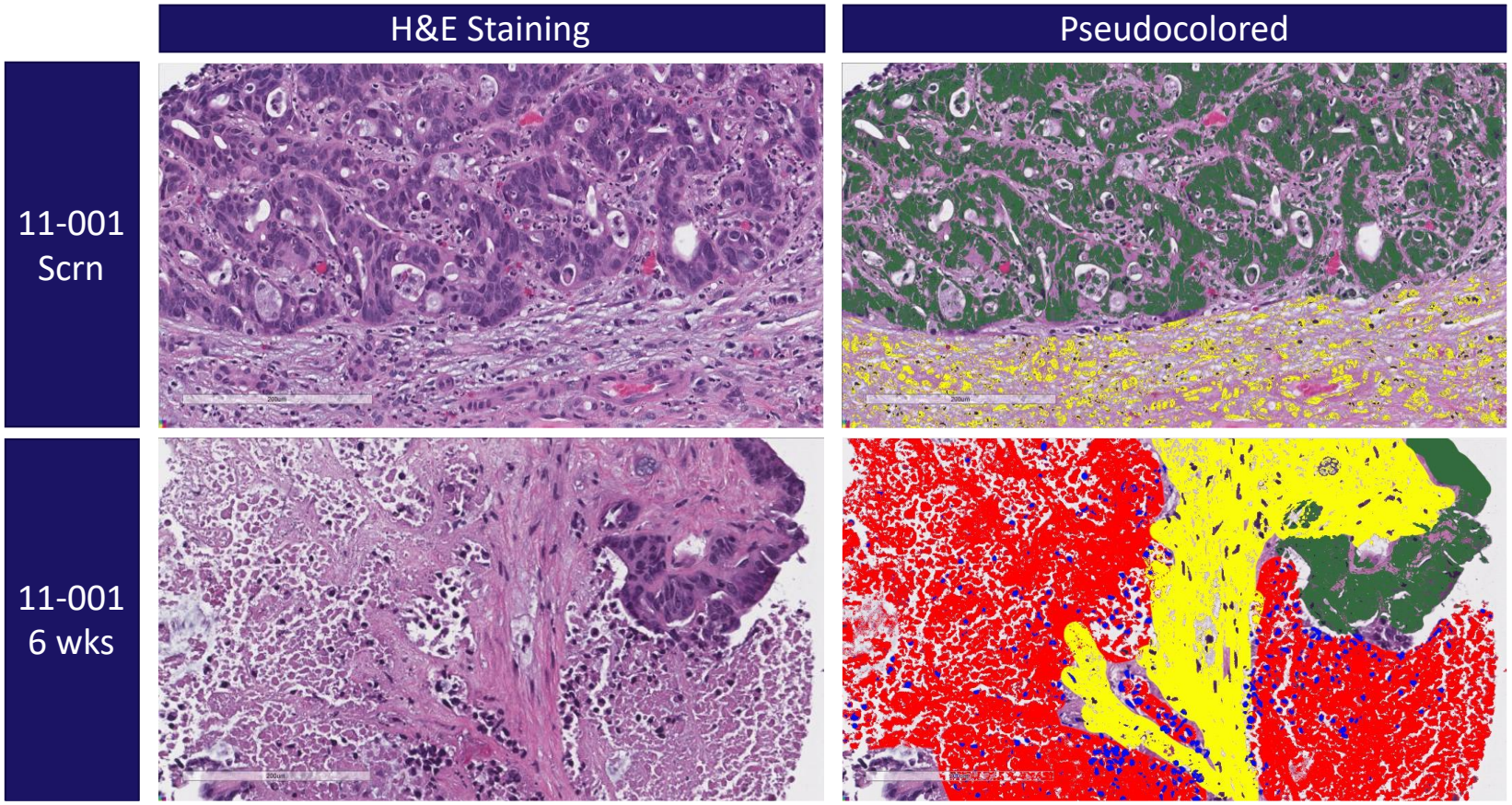
Case study: Increased Tumor Necrosis Associated With Immune Cell Infiltration



~ 65%

of the tumor tissue necrotic 6 weeks post DSP107 treatment (no necrosis at baseline)

Paired biopsy from colon carcinoma patient (11-001) in dose level 4 (0.3 mg/kg) pre- treatment and following cycle 2 (6 doses)

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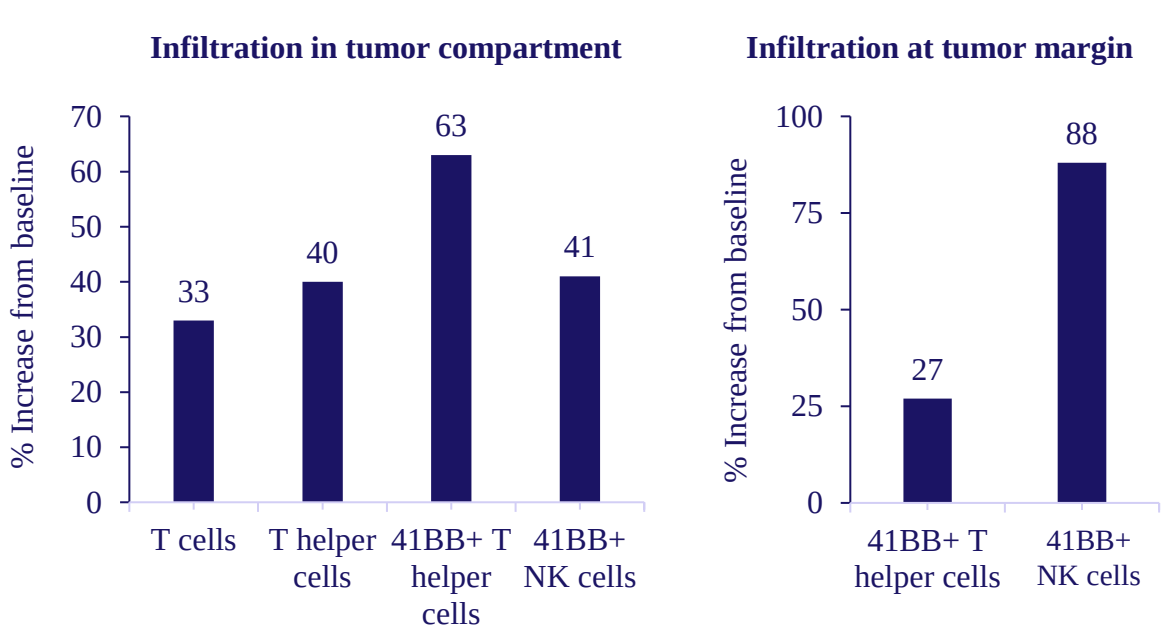
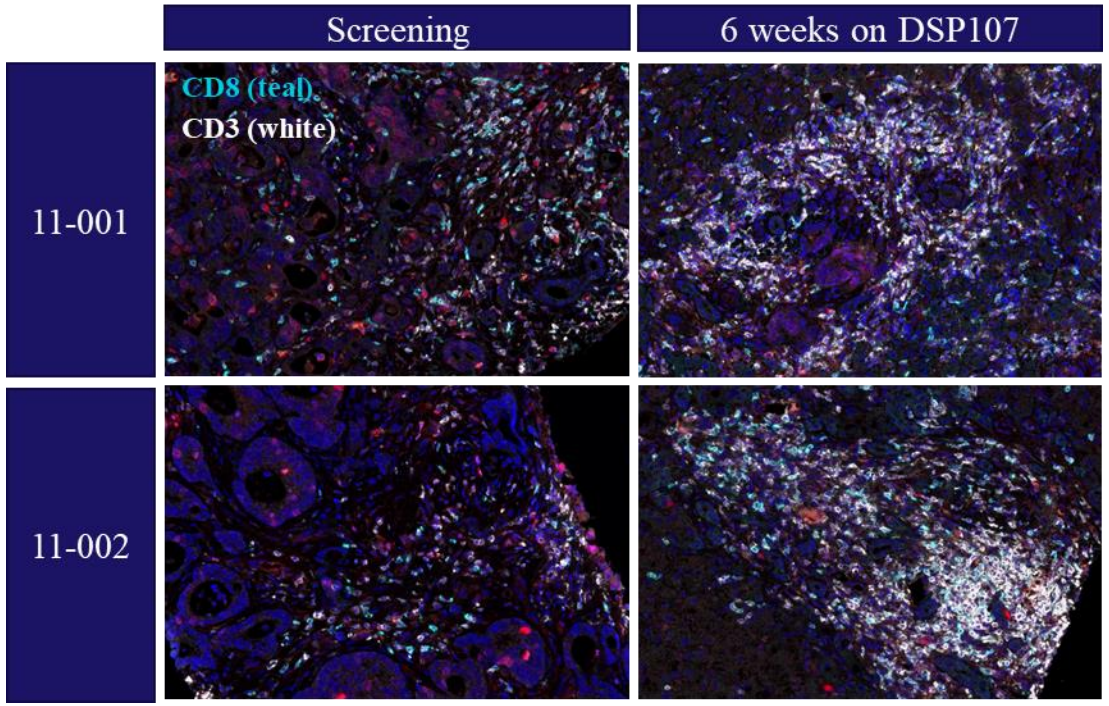
Case Study: Paired Biopsies Demonstrated Adaptive Immune Engagement in Ongoing Clinical Trial

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). 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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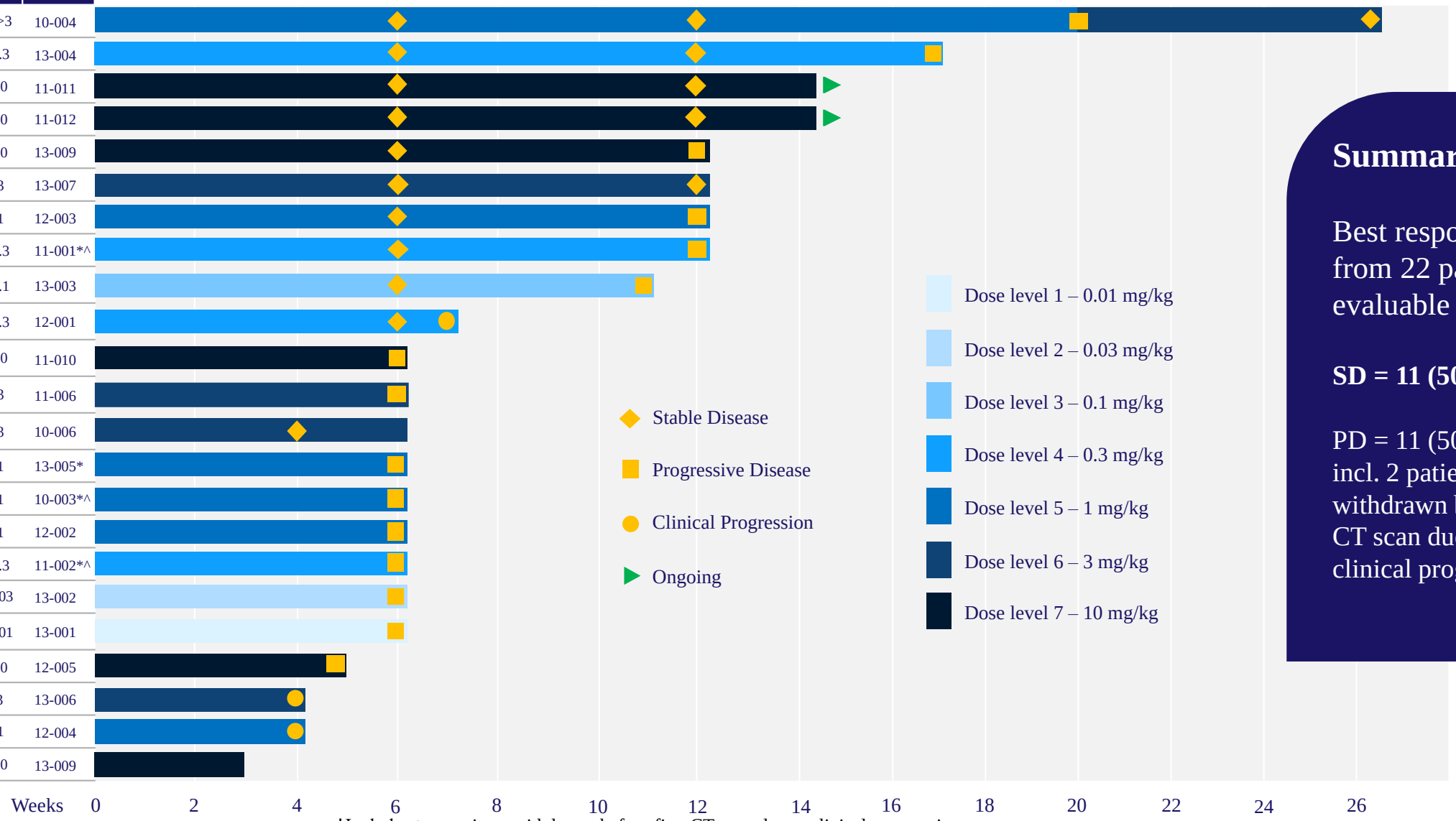
Best Overall Response After DSP107 Monotherapy

Indication	Dose	Patient #
NSCLC	1>3	10-004
Colorectal	0.3	13-004
Colorectal	10	11-011
Anal	10	11-012
Colorectal	10	13-009
Neuroendocrine GI	3	13-007
Ovarian	1	12-003
Colorectal	0.3	11-001*^
Head & Neck	0.1	13-003
Pancreas	0.3	12-001
Appendiceal	10	11-010
Colorectal	3	11-006
Eccrine Porocarcinoma	3	10-006
Pancreas	1	13-005*
Pancreas	1	10-003*^
Pancreas	1	12-002
Colorectal	0.3	11-002*^
Head & Neck	0.03	13-002
Neuroendocrine Lung	0.01	13-001
Ovarian	10	12-005
Adrenal	3	13-006
Head & Neck	1	12-004
Pancreas	10	13-009



Summary

Best response from 22 patients evaluable to date:

SD = 11 (50%)

PD = 11 (50%) incl. 2 patients withdrawn before 1st CT scan due to clinical progression

†Includes two patients withdrawn before first CT scan due to clinical progression;
*Paired biopsy data available; ^Necrosis induction evident in on treatment biopsy

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DSP107 Phase I/II Data: Favorable Preliminary Safety and Activity in Advanced Solid Tumors

Clinical Overview

- DSP107 as monotherapy and in combination with atezolizumab is being evaluated in a dose escalation trial
- 23 patients with diverse solid tumors have been treated with DSP107 monotherapy with 22 patients evaluable for efficacy analysis
- Now treating patients with DSP107 and atezolizumab
- Phase Ib in r/r AML and MDS is ongoing assessing the safety of DSP107 monotherapy, DSP107 with Azacytidine and DSP107 with Azacytidine plus Venetoclax

Key Findings

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

Next steps: Further evaluate potential safety and preliminary efficacy of DSP107 with atezolizumab

DSP107 Highlights

MOA

- CD47 inhibition (Cancer specific)
- 4-1BB activation (CD47-conditional)

Potential Efficacy - Preclinical

- Activates T cells, increases IFNγ secretion and anti-tumor killing
- Increased macrophage phagocytosis of tumor cells
- Augments mAbs' ADCP phagocytosis of cancer cells
- Potential efficacy as monotherapy and synergistic activity in combination

Differentiation

- Activates both adaptive and innate immunity
- No RBC binding observed
- Potentially favorable safety profile suitable for combination therapy

Potential Safety - Preclinical

- No binding to human RBCs observed
- No CD47 related hematological toxicities observed
- No 41BB related hepato-toxicities observed

Clinical Opportunities

- Solid tumors
  - DSP107 ± atezolizumab (intended to enhance T cell activation)
- Hematological Malignancies
  - DSP107 ± azacytidine + venetoclax (intended to enhance eat me signal)

Preliminary Clinical Data

Well tolerated

- No DLTs
- No hematological toxicities
- No hepato-toxicities

Preliminary efficacy signals as a monotherapy in solid tumors

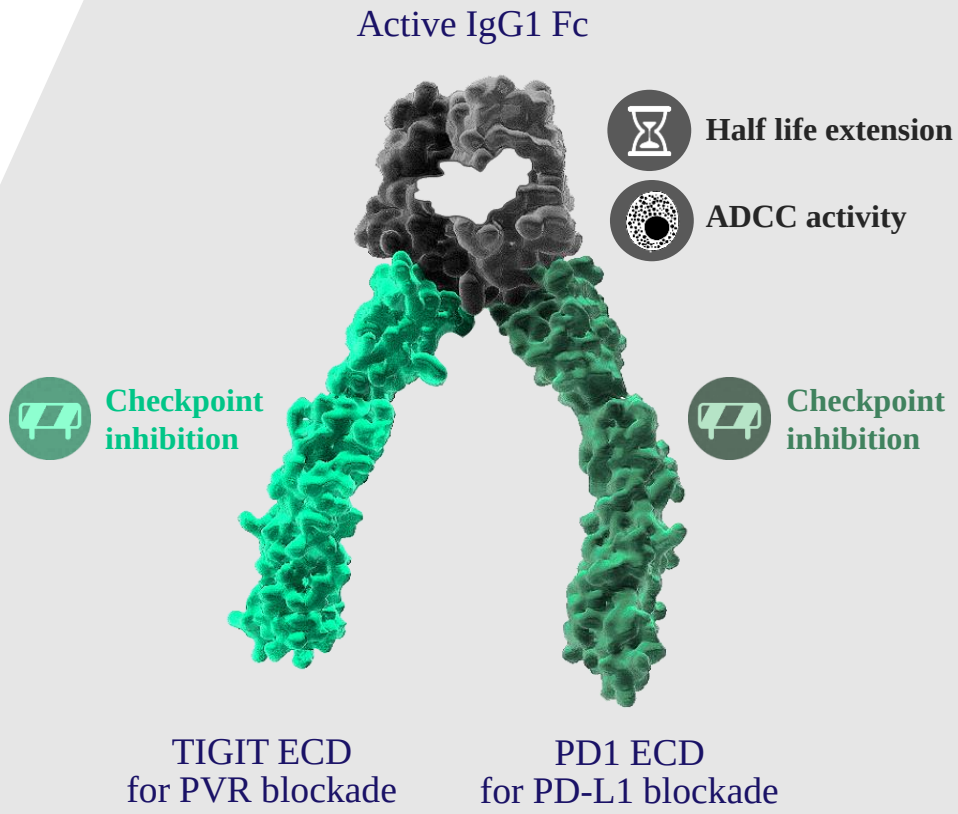
- Paired biopsies demonstrate tumor necrosis
- Stable disease achieved in eleven of twenty-two patients (50%)

DSP502

MIRP Type	**DSP-Fc**
Targets	**PVR, PD-L1, FcR**
Primary Cell Target	**NK cells, T effector cells**
Mechanism of Action	**Dual checkpoint inhibition designed to unleash NK and T-cells, ADCC**

Dual Checkpoint Binding Aimed at Enhancing Selectivity and Synergistic Immunity

1. Dual binding to overexpressed checkpoints may enable high tumor specificity
2. Potential anti-tumor immunity via simultaneous checkpoint inhibition of PVR and PD-L1
3. Active Fc may extend half-life and enhance ADCC activity



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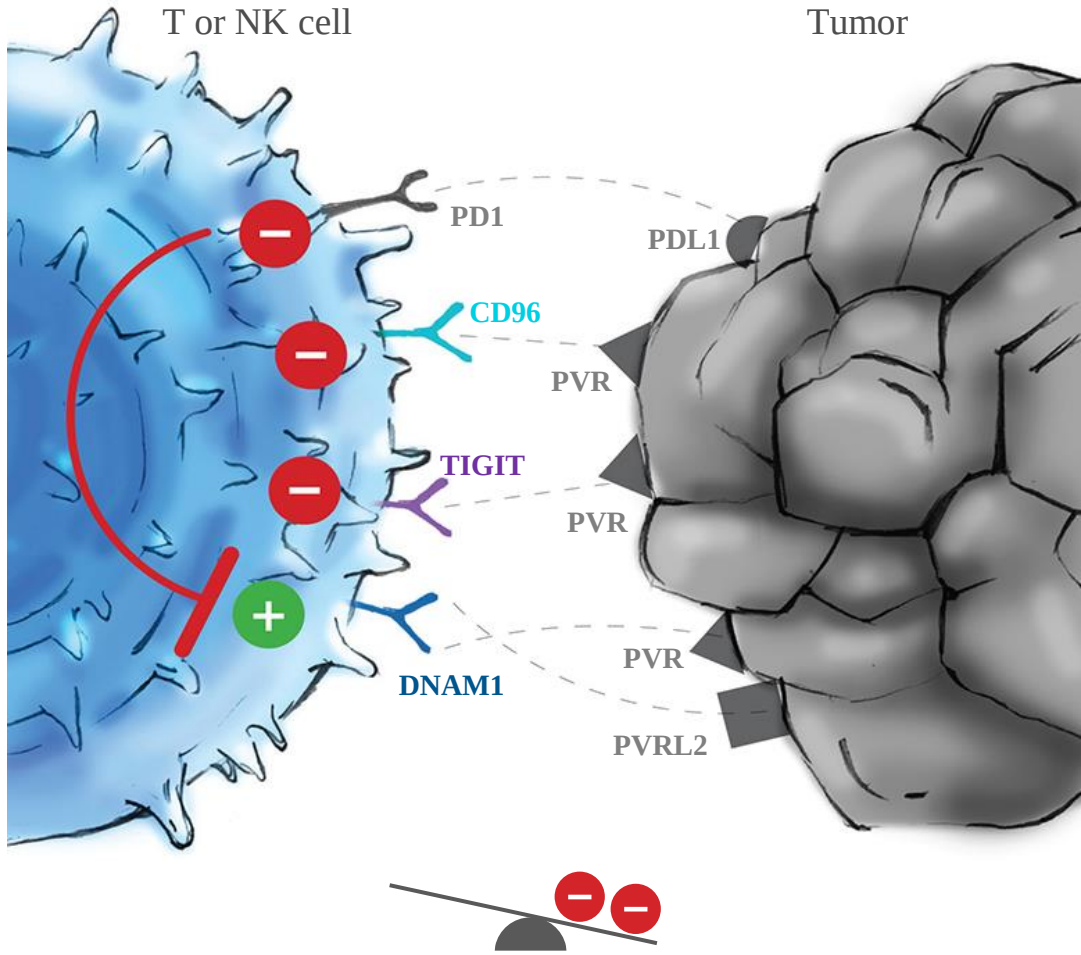
Novel, Potential Next-Generation Approach to Checkpoint Inhibition

- PVR is a ligand for inhibitory (TIGIT, CD96) and stimulatory (DNAM1) immune checkpoint receptors
- Tumor cells overexpress PVR to evade immune surveillance
- Overexpression of PVR is associated with resistance to checkpoint therapy in NSCLC and melanoma patients
- Inhibition of TIGIT/PVR is efficacious in combination with PD1 blockade
- PD1 inactivates DNAM1 stimulatory activity



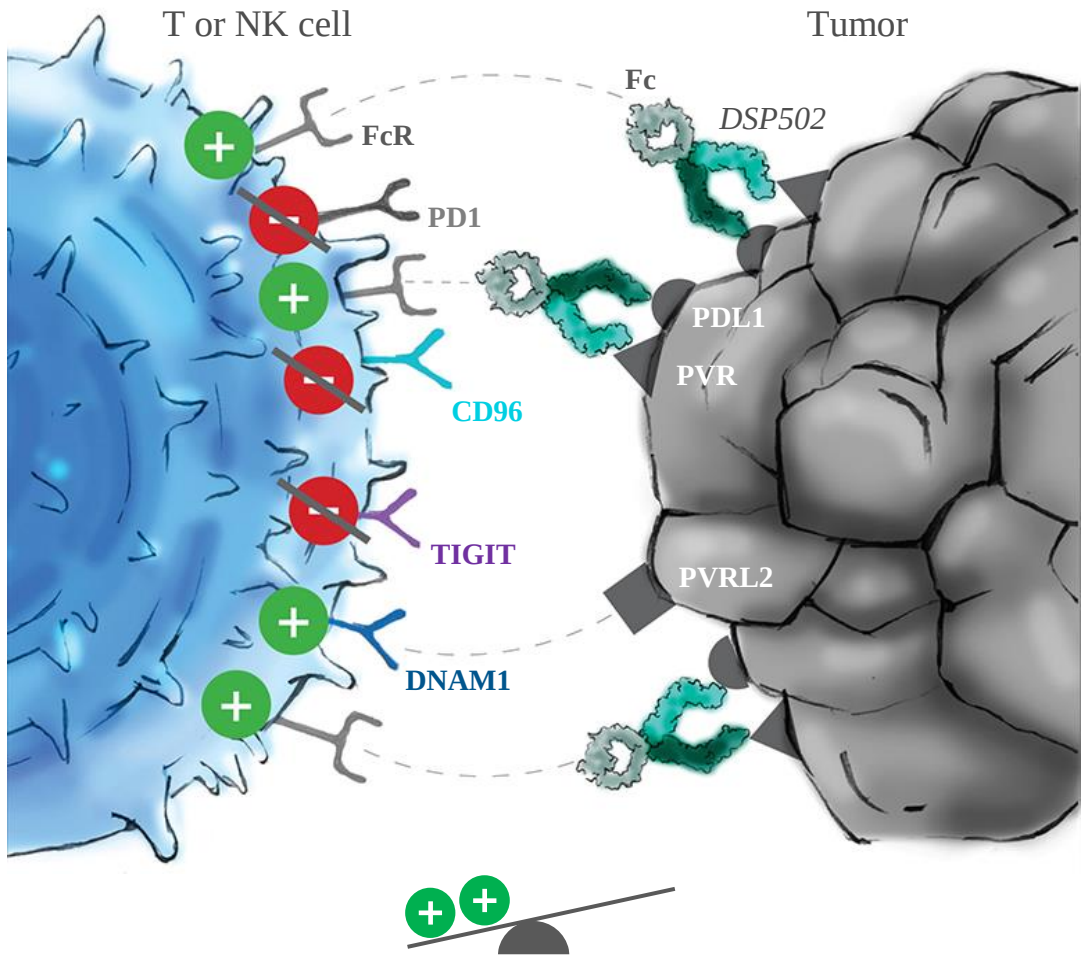
Immunosuppression is the net effect of PVR and PD1 overexpression in the TME

Synergistic Dual Checkpoint Inhibition for Robust Anti-tumor Immunity

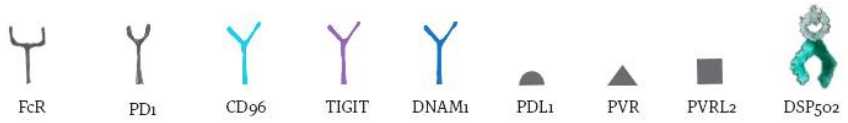
1. Simultaneous TIGIT, CD96 and PD-L1 inhibition with DNAM1 costimulation for enhanced anti-tumor immunity
2. Designed to activate both, T cells and NK cells

Effect	Dual PVR and PD-L1 Targeting (KAHR's approach)	TIGIT Ab* (Competitors)
− TIGIT	✓	✓
− CD96	✓	—
+ DNAM1	✓	—
− PD-1	✓	—

*Company has not undertaken comparative trials of DSP502 against the identified competitors



DSP502 blocks PVR and PD1 signaling for enhanced immune activation and anti-tumor immunity



DSP502 – Differentiated TIGIT/PD1 Targeting Compound

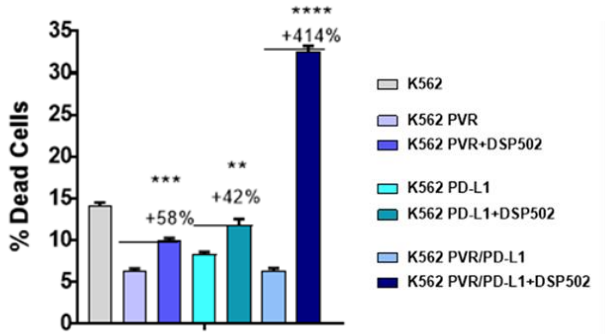
Potential next-generation capabilities

Dual MOA
designed to activate innate and adaptive immunity

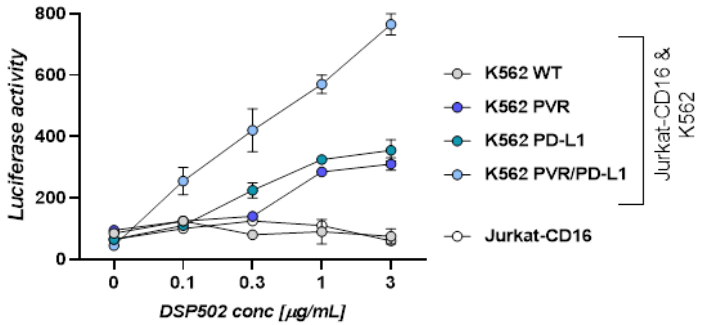
High Tumor Specificity
Concomitant binding to PVR and PD-L1 required for its activity

Designed to Have Unique Features
Multiple functionalities that act simultaneously for synergistic effect

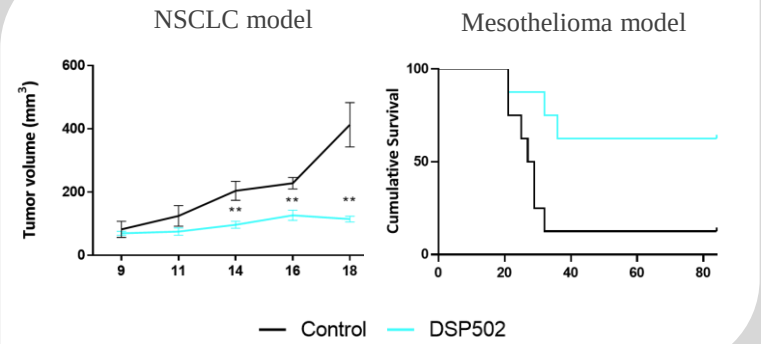
Differentiated mechanism of action



Enhanced NK cells cancer killing potential



Augmented NK cells ADCC activity



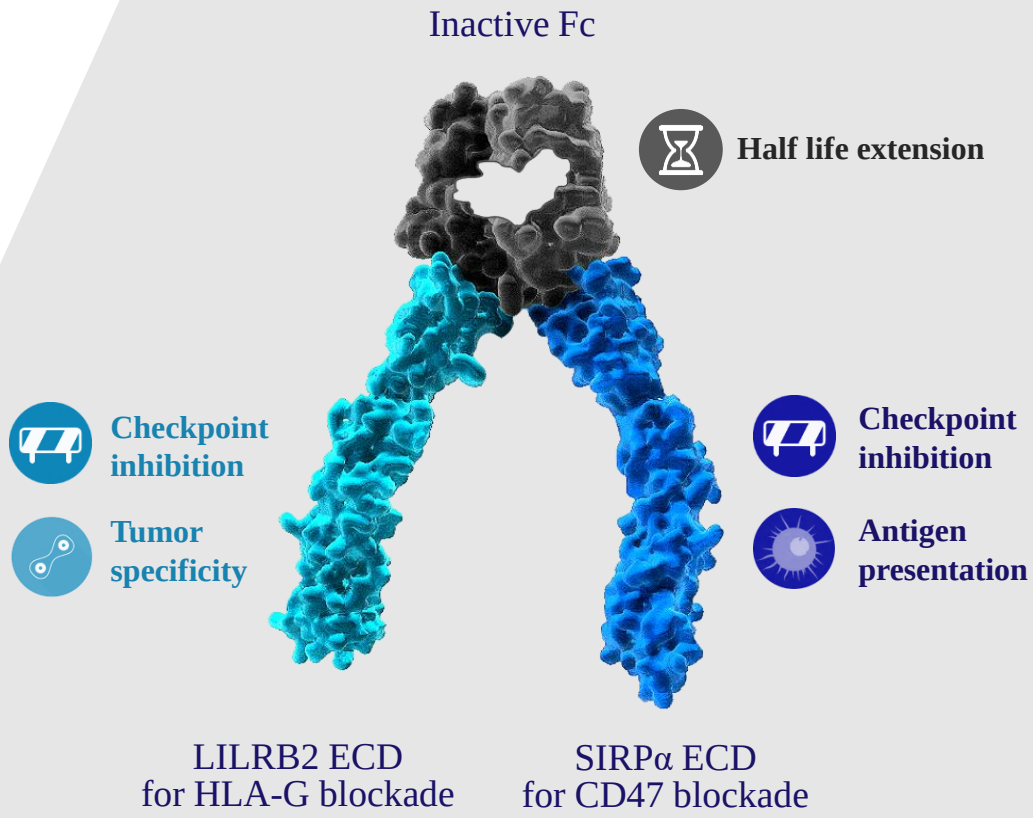
Demonstrated potent single agent anti tumor activity

DSP216

MIRP Type	**DSP-Fc**
Targets	**CD47, HLA-G**
Primary Cell Target	**mϕ macrophages, T effector cells, NK cells, Myeloid cells**
Mechanism of Action	**Dual checkpoint inhibition unleash macrophage, NK and Teff**

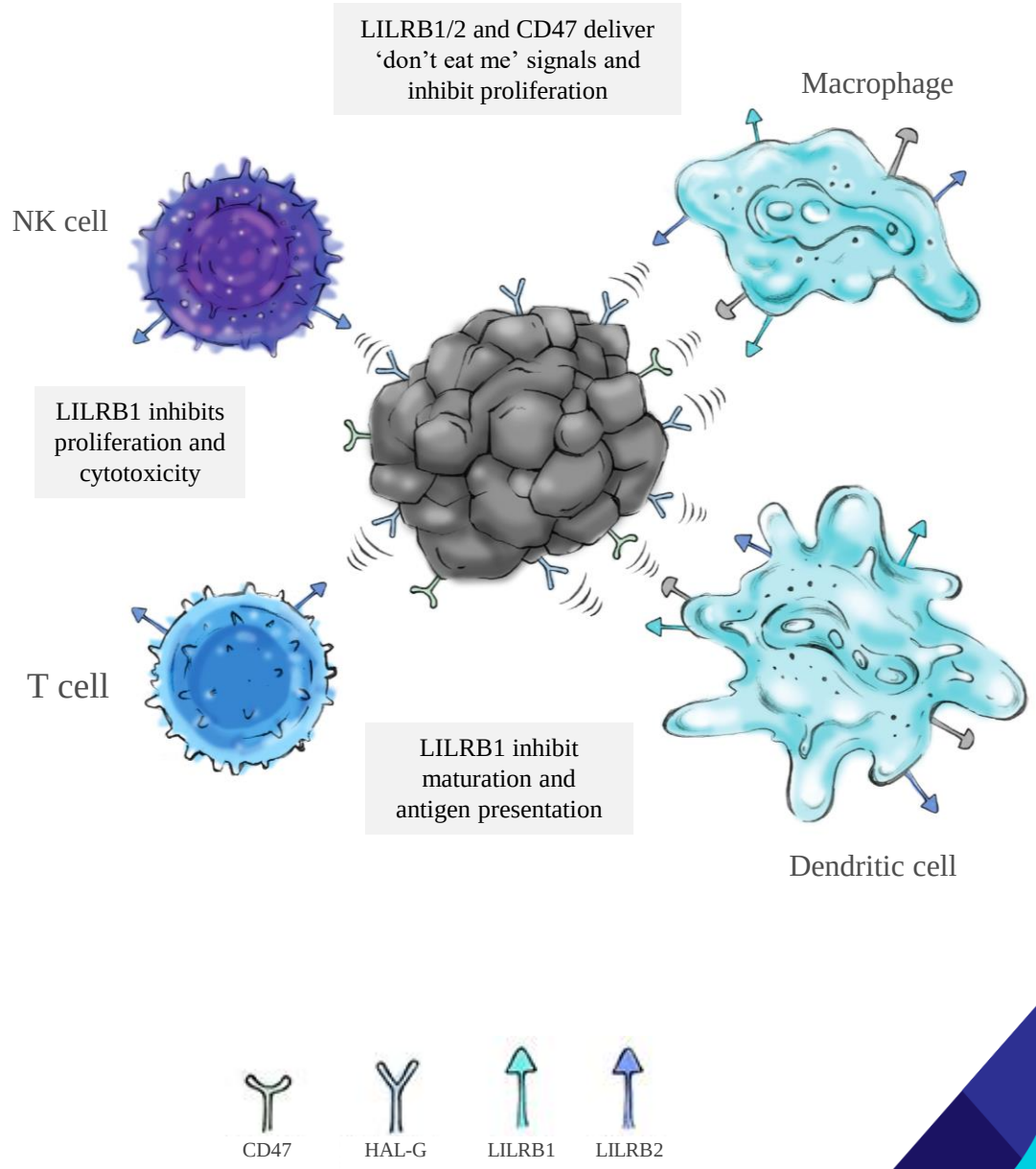
Dual Checkpoint Binding Designed For Enhanced Selectivity and Broad Immunity

1. Dual binding to overexpressed cancer checkpoints may enable high tumor specificity
2. HLA-G and CD47 blockade designed to activate innate and adaptive immunity
3. Inactive Fc may extend half-life



HLA-G Blockade Designed To Prevent Immunotolerance Across Immune Cells

- Placenta expressed HLA-G triggers immunotolerance to prevent the mother's immune system from attacking the fetus
- Tumor cells overexpress HLA-G to evade immune surveillance
- HLA-G is a broad-range immune checkpoint that is the main ligand for the LILRB/ILT receptor family
- HLA-G inhibits multiple immune cell subsets and recruits suppressive immune cells to the tumor microenvironment



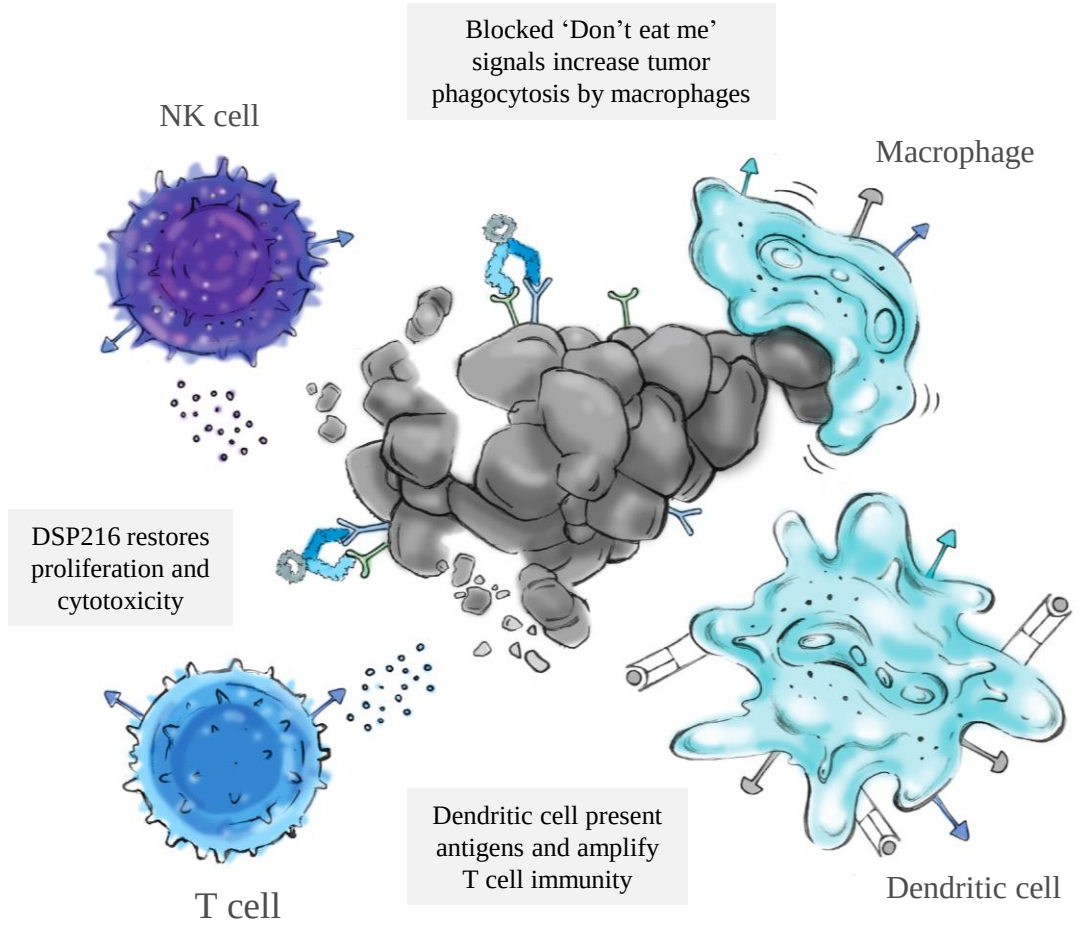
Dual Targeting Designed For Robust Tumor Selective Innate & Adaptive Immune Activation

1. HLA-G blockade aims to prevent cancer immunotolerance by **multiple immune cell subsets**

2. CD47 blockade removes ‘don’t eat me’ signal, triggering macrophage phagocytosis of tumor cells

Effect	HLA-G & CD47 (KAHR’s approach)	LILRB1/2 Ab* (Competitors)
− LILRB1 & LILRB2	✓	—
+ Tumor selectivity	✓	—
+ Innate & adaptive immunity	✓	—

*Company have not undertaken comparative trials of DSP502 against the identified competitors



CORPORATE HIGHLIGHTS

Multifunctional Cancer Immunotherapy Candidates Targeting Innate and Adaptive Immune Cells

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

DIFFERENTIATED PIPELINE
- Novel, next-generation potential across three programs
- Lead candidate DSP107 –
  CD47 inhibition (Cancer specific)
  41BB activation (CD47-conditional)

ANTICIPATED MILESTONES
-DSP107| Interim Ph II solid tumor mono and combo data and interim Ph I hematological malignancy data H2 2022
-DSP502 & DSP216 | IND 2023

MARKET
Immuno-therapeutics
$50.2B by 2026

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, Thomas Jefferson University.

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