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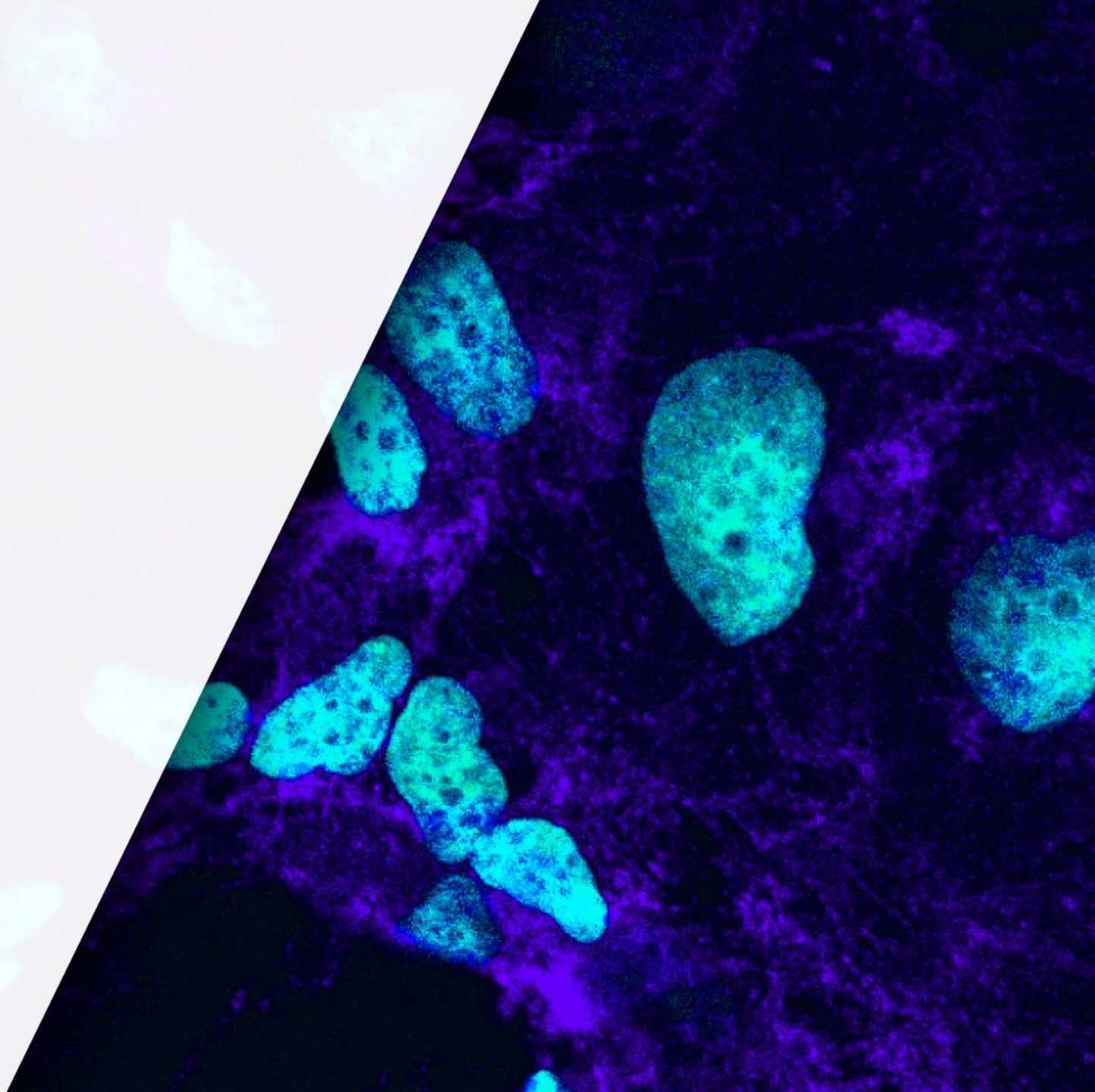
KAHR BIO

Multifunctional Immunotherapeutics

Company Presentation | Feb. 2024



Company Highlights

Our Unique Concept

- Multifunctional fusion proteins targeting innate and adaptive immunity simultaneously to bolster the potential of immunotherapy in unresponsive tumors

Our Lead Drug

- First in class CD47x4-1BB immune cell engager converting cold tumors to hot
- Best in class and differentiated CD47 blocking agent not causing anemia

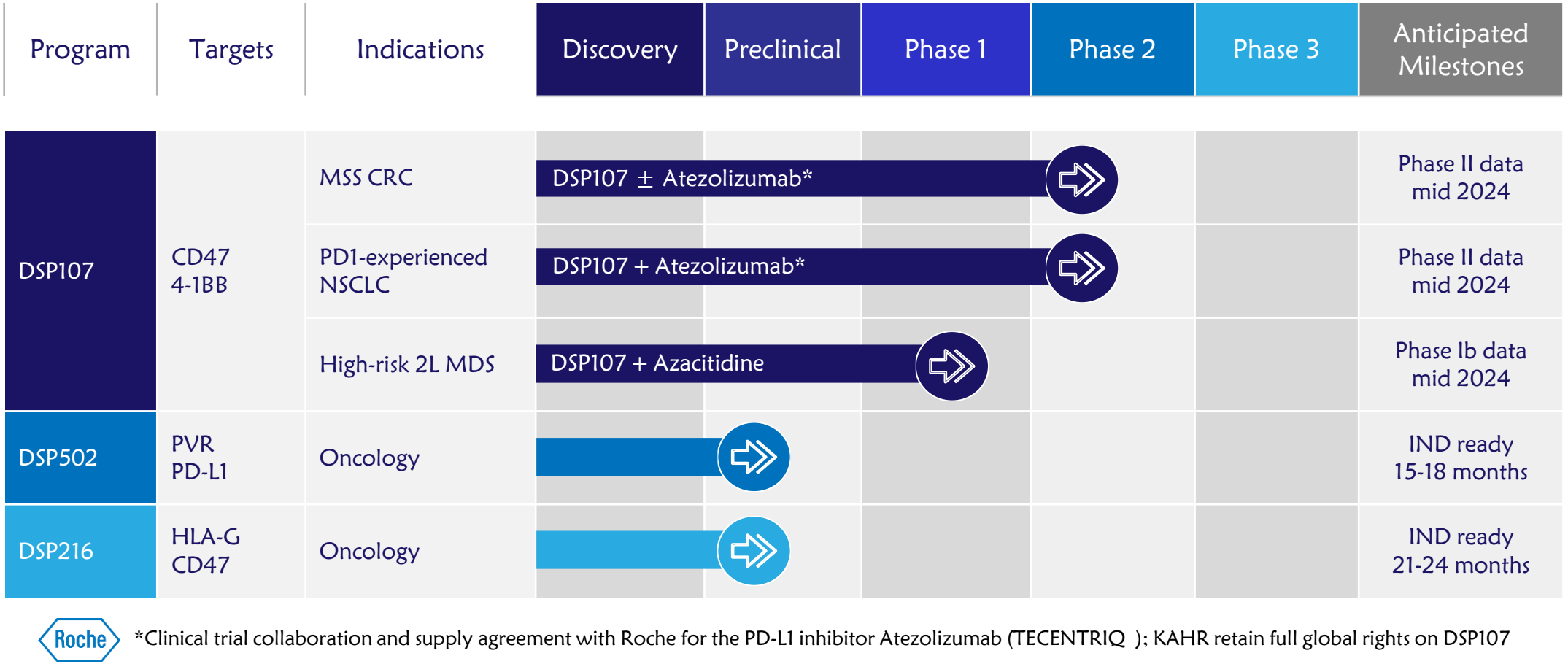
Clinical Validation

- Phase I/II study demonstrates excellent safety profile and responses in cold solid tumors
- Phase Ib study demonstrates responses in HMA-failure high risk r/r MDS patients

Corporate Highlights

- Runway into 2025 and through multiple clinical milestones:
  - ❑ Phase II cohorts in 3L MSS CRC and PD1-experienced NSCLC topline data
  - ❑ Phase Ib 2L MDS topline data

Focused and Differentiated Pipeline



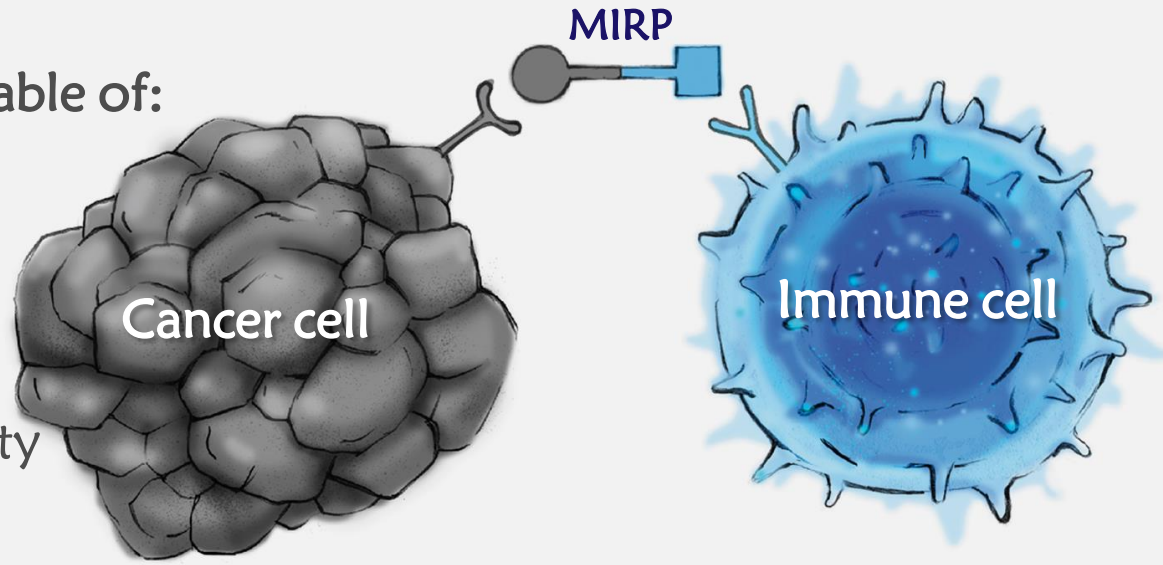
Roche *Clinical trial collaboration and supply agreement with Roche for the PD-L1 inhibitor Atezolizumab (TECENTRIQ); KAHR retain full global rights on DSP107

Multifunctional Immunotherapeutic Platform (MIRP) for Solid and Hematological Malignancies

MULTIFUNCTIONAL IMMUNE RECRUITMENT PROTEIN (MIRP)

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

1. Inhibiting key immune checkpoints on tumors
2. Activating innate and adaptive anti-tumor immunity



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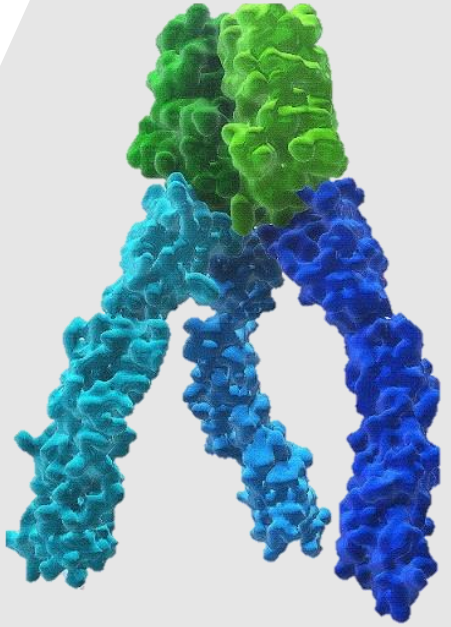
DSP107 Structure Designed For Tumor Selectivity and Enhanced Efficacy

Trimeric ligand ends designed to enable:

1. Cancer selective binding to overexpressed CD47 on tumor cells, sparing RBC binding and associated toxicities
2. Conditional 4-1BB-mediated T cell activation dependent on trimeric binding to CD47 on cancer cells

4-1BB is a costimulatory receptor that when activated enhances T-cell activation

Trimeric 4-1BBL



- Cytolytic T cell activation
- T cell Proliferation
- Checkpoint inhibition
- Tumor microenvironment modulation

3 SIRPα for CD47 Blockade

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

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Unlocking CD47 and 4-1BB Potential

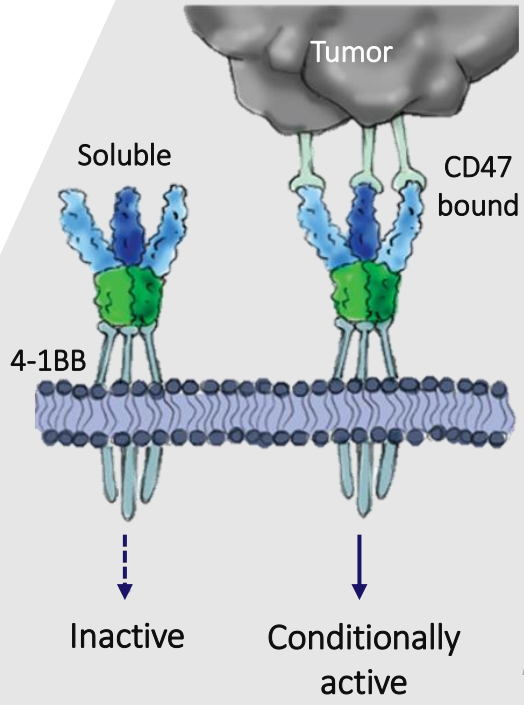
Challenges and opportunities

- Validated targets but their inherent toxicities limited therapeutic window of 1st generation agents
- Complimentary mechanism - without active T cells CD47 inhibitors are inactive
- Tumor microenvironment (TME) mainly contains inactive macrophages and lower counts of cytotoxic T cells

DSP107 solutions

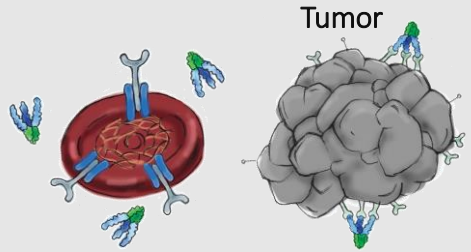
- CD47 blockade without binding RBCs and anemia
- 4-1BB conditional activation restricted to TME
- Blockade of CD47 activates macrophages
- T-cell engagement modulates TME composition

4-1BB conditional activity



CD47 tumor selectivity

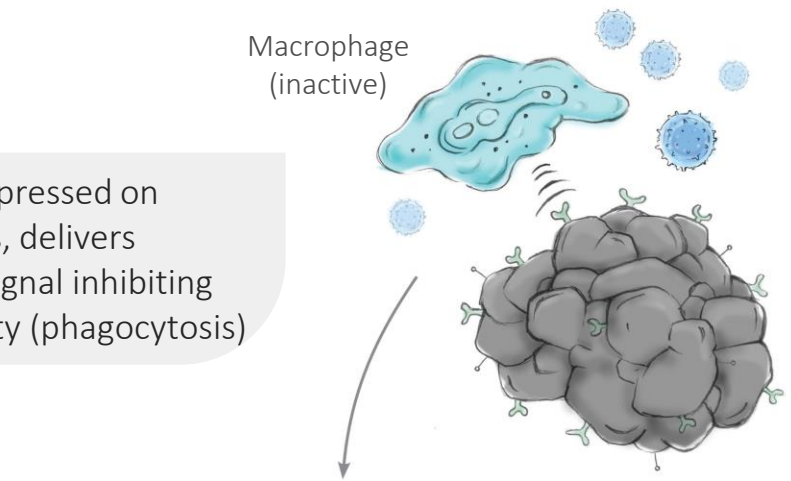
High affinity/avidity of DSP107 to CD47 clusters on cancer cells

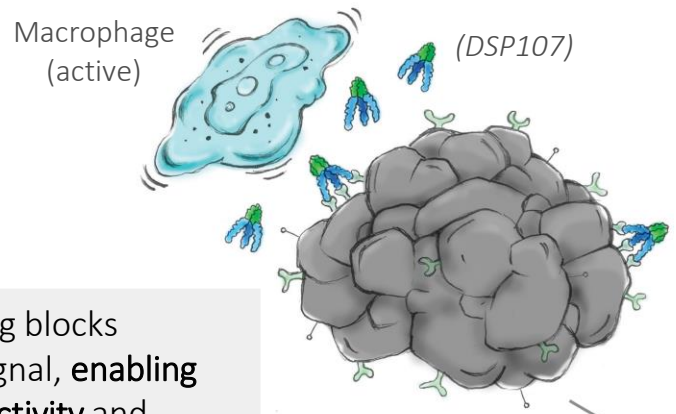


On RBCs CD47 protein complex is anchored to cytoskeleton resulting in its immobilization and low affinity of DSP107 to the monomeric CD47

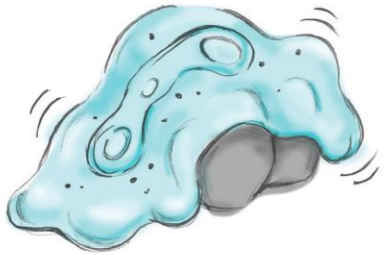
Designed for Synergistic Innate & Adaptive Immune Activation

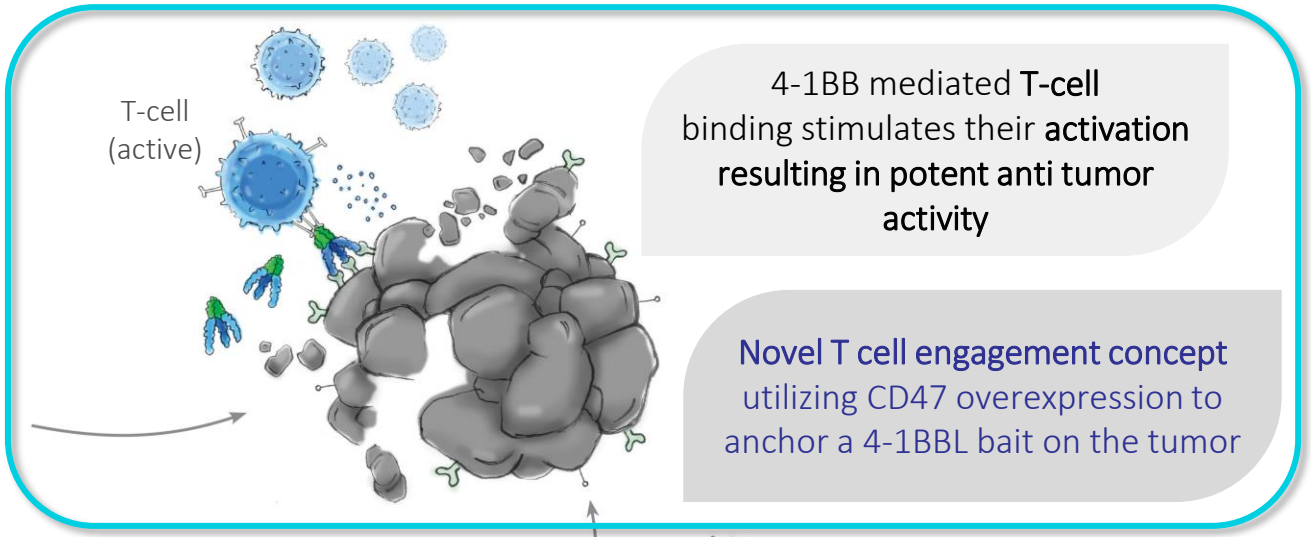
CD47 overexpressed on cancer cells, delivers **“don’t eat me”** signal inhibiting macrophage activity (phagocytosis)





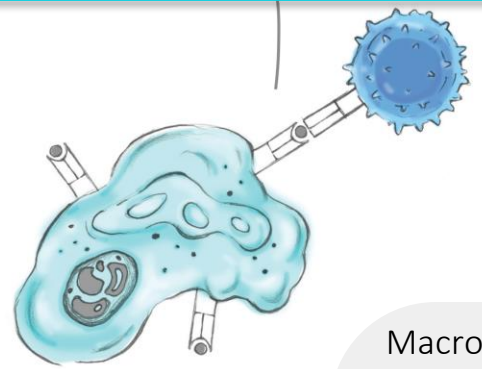
CD47 binding blocks “don’t eat me” signal, **enabling macrophage activity** and anchoring 4-1BBL to the tumor to **engage effector T cells**



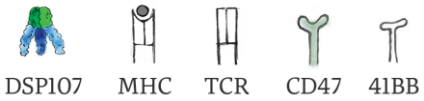


4-1BB mediated **T-cell** binding stimulates their **activation resulting in potent anti tumor activity**

Novel T cell engagement concept utilizing CD47 overexpression to anchor a 4-1BBL bait on the tumor



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

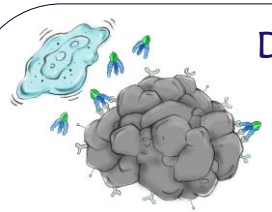


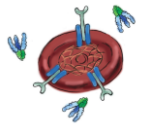
Solid Tumors Program

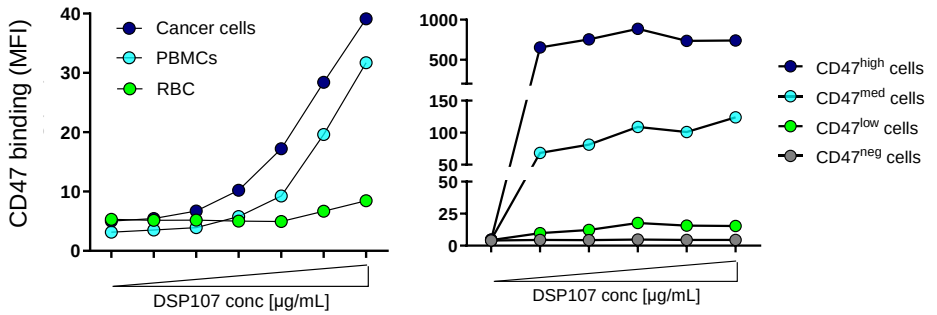
Preclinical Data Supports DSP107 Potential for Solid Tumors

Selective binding, phagocytosis induction, T cell activation and synergism with anti PD-L1 demonstrated

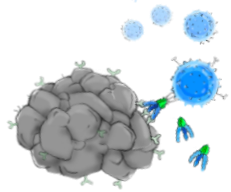
DSP107 selectively binds tumor cells while sparing RBCs

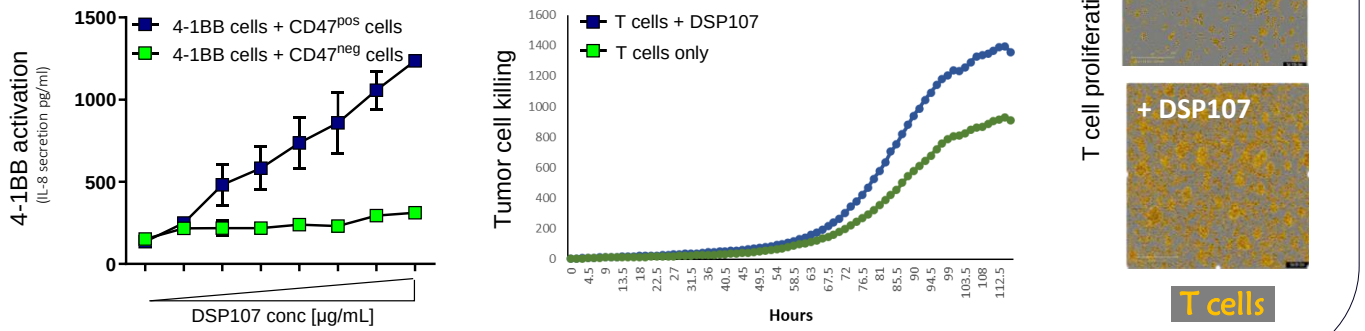






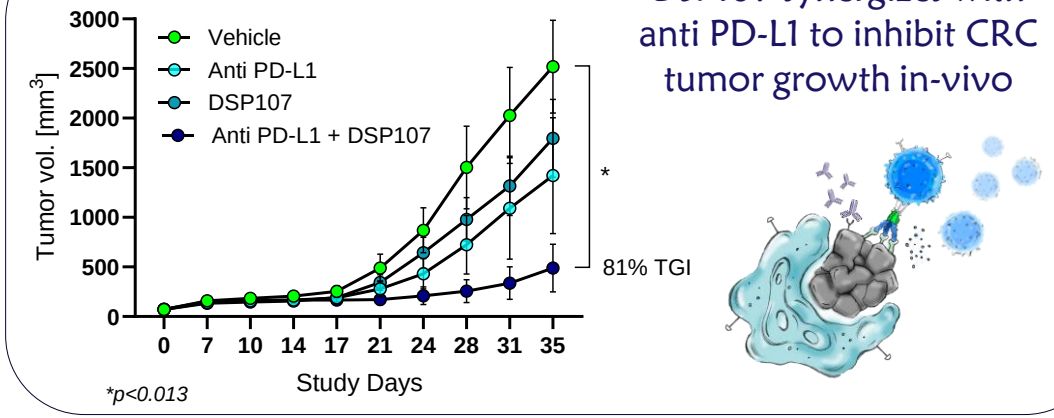
DSP107 increases T cell activation, cytotoxicity and expansion





DSP107 induces tumor cell phagocytosis

DSP107 synergizes with anti PD-L1 to inhibit CRC tumor growth in-vivo



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Phase I Dose Escalation Study - Design

First-in-human dose escalation trial to establish safety of DSP107 mono and Atezolizumab combo therapy

Advanced Solid Tumors

Phase 1 Dose escalation (n = 42)

Enrolling sites: Pittsburgh, Colorado, Kansas, Thomas Jefferson; SCRI

- DSP107 dose levels 0.01 - 10 mg/kg administered IV Q1W
- Atezolizumab 1200 mg administered IV Q3W
- Single patient dose escalation up to 0.3 mg/kg, then standard 3+3 design
- Approximately half of patients with cold tumors and/or progressed on prior PD1/PD-L1 therapy

Study Population

Sample size	N = 42
Sex	17 (40%) ♀ ; 25 (60%) ♂
Age	Median 61 (29-78)
Tumor types	
MSS Colorectal	12 (29%)
Pancreas	9 (21%)
Head and Neck	4 (9.5%)
Sarcomas	4 (9.5%)
Ovarian	2 (5%)
NSCLC	1 (2.5%)
Rare and other	10 (24%)
# Previous lines	Median 3 (Range 1-8)
PD1/PD-L1 experienced	16 (38%)

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DSP107 Monotherapy and Atezo Combination Safety Results

Clean and differentiated safety profile; no overlapping toxicities with common PD-(L)1 CPIs

Summary

- DSP107 monotherapy and in combination with Atezo well tolerated
- No DLTs up to 10 mg/kg, and no treatment-related SAEs
- No hematological toxicities
- No hepato-toxicities
- Very few AEs considered related to DSP107. Most related AEs Grade 1-2 in severity

Treatment-Related AEs in ⩾ 2 Patients		
Regimen	Monotherapy	Combo with Atezolizumab
Total No of Patients	N = 23 (0.01 - 10 mg/kg)	N = 19 (1 - 10 mg/kg)
Treatment-related AEs (any grade)	n (%)	n (%)
Any	16 (70)	13 (68)
IRR*	8 (35)	4 (21)
Diarrhea	4 (17)	4 (21)
Fatigue	4 (17)	4 (21)
Nausea	3 (13)	2 (10)
Constipation	2 (9)	NA
Arthralgia	NA	2 (10)
Myalgia	NA	2 (10)
Decreased Appetite	NA	2 (10)
Anemia**	NA	2 (10)

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

**Anemia Grade 1 in severity

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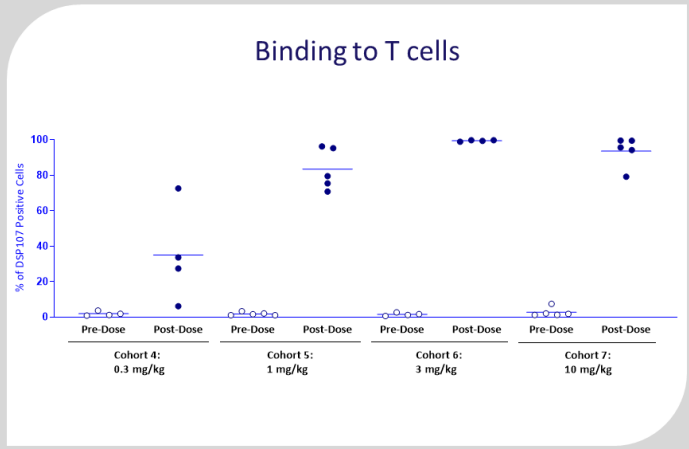
Phase I Dose Escalation Study - Results

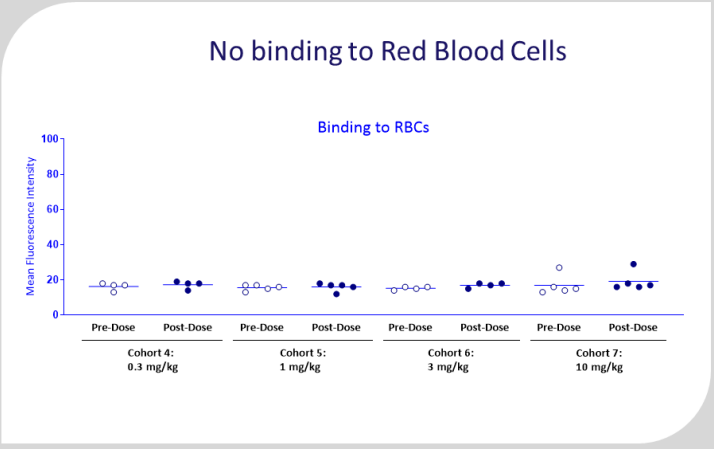
Demonstrated infiltration of 4-1BB T-cells in cold tumors

High disease control rate and objective responses including in metastatic MSS-CRC patients

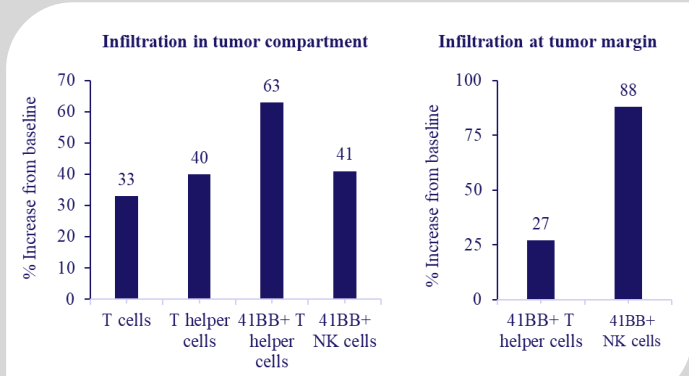
Summary

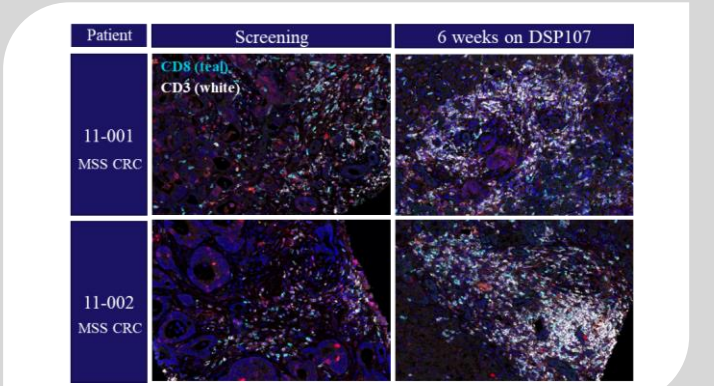
- Dose-dependent target engagement with 100% receptor occupancy from 3 mg/kg
- Paired biopsies demonstrate immune cell infiltration in cold tumors
- 55% disease control rate including durable objective responses in cold tumors when combined with Atezo
- 10 mg/kg selected for expansion cohorts in MSS-CRC and NSCLC





Paired biopsies data





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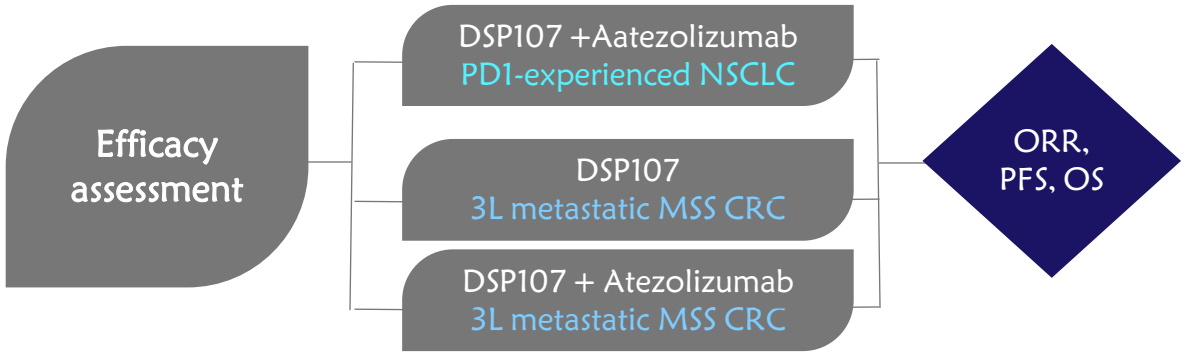
Phase II Dose Expansion Cohorts Design

Expansion cohorts in largest oncology indications where immunotherapy found unresponsive to date

NSCLC and MSS CRC

Phase 2 Expansion Cohorts (n = 60)



Enrolling sites: Pittsburgh, Colorado, Kansas, Thomas Jefferson; SCRI

	SOC	Historical benchmark	Response to CPI
NSCLC 2L/3L PD1-experienced	Chemo. or targeted therapies	ORR= 6% - 14% PFS ~ 3 months OS ~ 6 months	ORR = 3% - 7% PFS = 1.5 m - 4 m OS ~ 7 months
CRC 3L MSS	Lonsurf + Bevacizumab	ORR=5.4% PFS = 5.6 months OS = 10.8 months	ORR = 0 - 2% PFS ~2 months OS = 5-7 months

Study Population

- **2L or 3L NSCLC**
  - Histologically confirmed, inoperable non-small cell lung cancer (Stage 3b or 4)
  - Received up to 2 lines of prior systemic treatment, including anti PD-1/PD-L1 therapeutic agent ± chemotherapy
- **3L MSS CRC**
  - Histologically confirmed, inoperable microsatellite stable colorectal carcinoma (Stage 3b or 4)
  - Received two previous lines of therapy including standard chemotherapy and/or targeted antibodies

Refs: N Engl J Med 2023, 388:1657-1667 | Thorac Cancer, 2020, 11(7):1927-1933 | Jpn J Clin Oncol. 2019, 49(8):762-765
J. Clin. Med. 2020, 9(1), 102 | Lung Cancer,. 2023: 178:116-122 | https://doi.org/10.1097/JTO.0b013e3181bbf223

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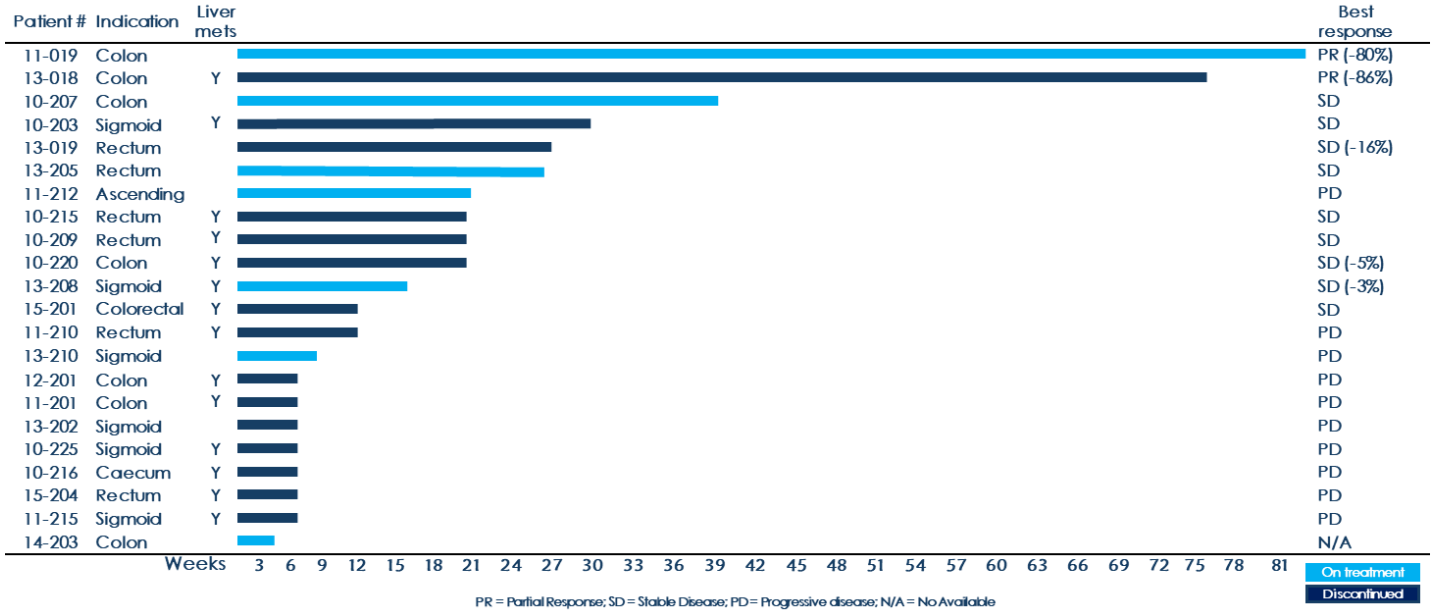
Phase II MSS CRC Dose Expansion Cohort Results

High disease control rate and objective responses in 3L MSS-CRC patients

Summary

- DSP107 combination with Atezolizumab safe and well tolerated
- Two patients (10%) with deep, durable objective responses (~ 18 months)
- 55% disease control rate in patients with KRAS / BRAF mutations and liver metastases
- Anti-tumor effect on target lesions including liver metastases in 50% patients
- ORR and DCR results twice better than SOC Lonsurf + Bevacizumab
- Prolonged treatment durability without safety concern may predict PFS and OS advantage

Best response swimmer plot



Phase II MSS CRC Dose Expansion Cohort Results

DSP107 + Atezo active in MSS CRC patients with liver metastasis

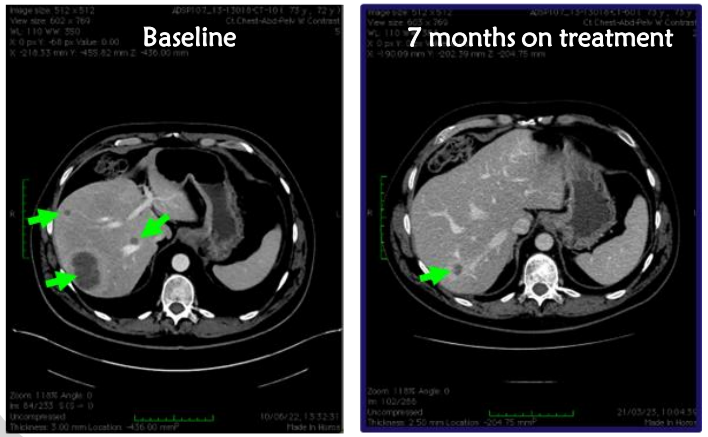
MSS CRC with Liver Metastases

- Liver is the most common site of metastases in MSS CRC and appear in 80% of 3L patients
- Prognosis of patients with liver metastases is poor, and most patients cannot undergo surgery
- Conventional antitumor approaches including chemo, radiotherapy and targeted therapy result in unsatisfactory outcomes
- Immunotherapy provided no clinical benefit owing to the inhibitory impact of the immunosuppressive TME in the liver
- DSP107 with Atezolizumab may become the first effective therapy for CRC patients with liver metastases

13-018 MSS CRC patient

KRAS mutated; with liver and lung metastases

- 46 year old male patient
- Adenocarcinoma of sigmoid colon
- Moderately to well-differentiated
- Previous lines of systemic therapy include: XELOX, XELOX + Bev, FOLFIRI + Bev, JAB-3312 + binemitinib, Lonsurf
- Received 2 cycles of DSP107 + Atezo and achieved mixed response; on DSP107 monotherapy since week 9 deepen response to PR (-86%) until week 74



Patient #	Liver mets	Effect on target lesions
11-019		-
13-018	Y	-/-/-/-/-
10-207		0/+
10-203	Y	+/+/+/+/+
13-019		-/-/-
13-205		+/+/-
11-212		N/A
10-215	Y	+/+/+
10-209	Y	-/-/-/-/+
10-220	Y	-/-/+
13-208	Y	0/0/-/-
15-201	Y	+/+
11-210	Y	N/A
13-210		N/A
12-201	Y	+/+/-
11-201	Y	N/A
13-202		0/+
10-225	Y	+/+/-/+/+
10-216	Y	+/+/+/+/+
15-204	Y	
11-215	Y	
14-203		N/A

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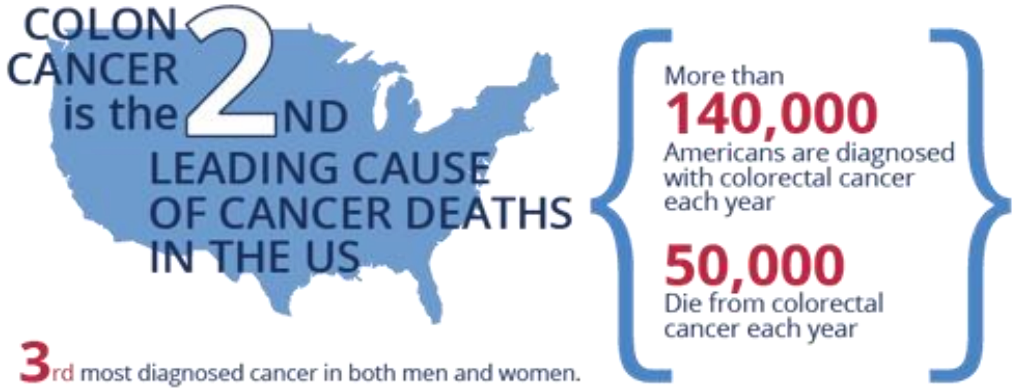
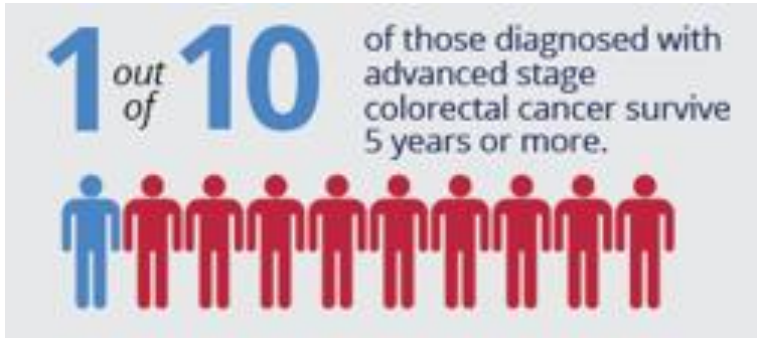
DSP107 MSS-CRC Opportunity

- CRC is one of the most common cancers with annual 1.8M new cases worldwide and >$16B market
- >90% of the cases with microsatellite stable (MSS) profile
- Recent FDA approvals for third line therapy in metastatic CRC:
  - **Trifluridine/tipiracil** (Lonsurf, Taiho Oncology) **in combination with bevacizumab** (Avastin, Roche) approved based on **mOS of 10.8 months, mPFS of 5.6 months, ORR of 5.4% and a DCR of 29%** (Aug. 2023)
  - **Fruquintinib** (Fruzaqla, Takeda) approved based on **mOS of 7.4 months, mPFS of 3.7 months, ORR of 1.5% and a DCR of 56%** (Nov 2023)





PD-(L)1 CPI Monotherapy is Inactive in MSS-CRC

- MSS CRC harbors a disrupted immune response in the TME, characterized by low infiltrated T cells and reduced anti-tumor immune cytotoxicity
- PD1/PD-L1 CPIs are inactive in MSS CRC with **ORR 0-2% and PFS ~2 months**
- DSP107 mediated TME modulation by engaging and activating immune effector cells may prime MSS tumors to CPIs

PD1/PD-L1 Agent	Phase	N	Population	ORR	Median PFS	Median OS
Pembrolizumab	2	18	MSS	0	2.2 months	5 months
Nivolumab ± Ipilimumab	2	23	MSS	N/A	1.4 months	N/A
Durvalumab + Tremelimumab	2	119	98% MSS	1%	1.8 months	6.6 months
Atezolizumab	3	90	92% MSS	2%	1.8 months	7.1 months

Sahin et al., ASCO Educational Book, Volume 42, Number 42, 2022; https://doi.org/10.1200/EDBK_349811

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Hematological Malignancies Program

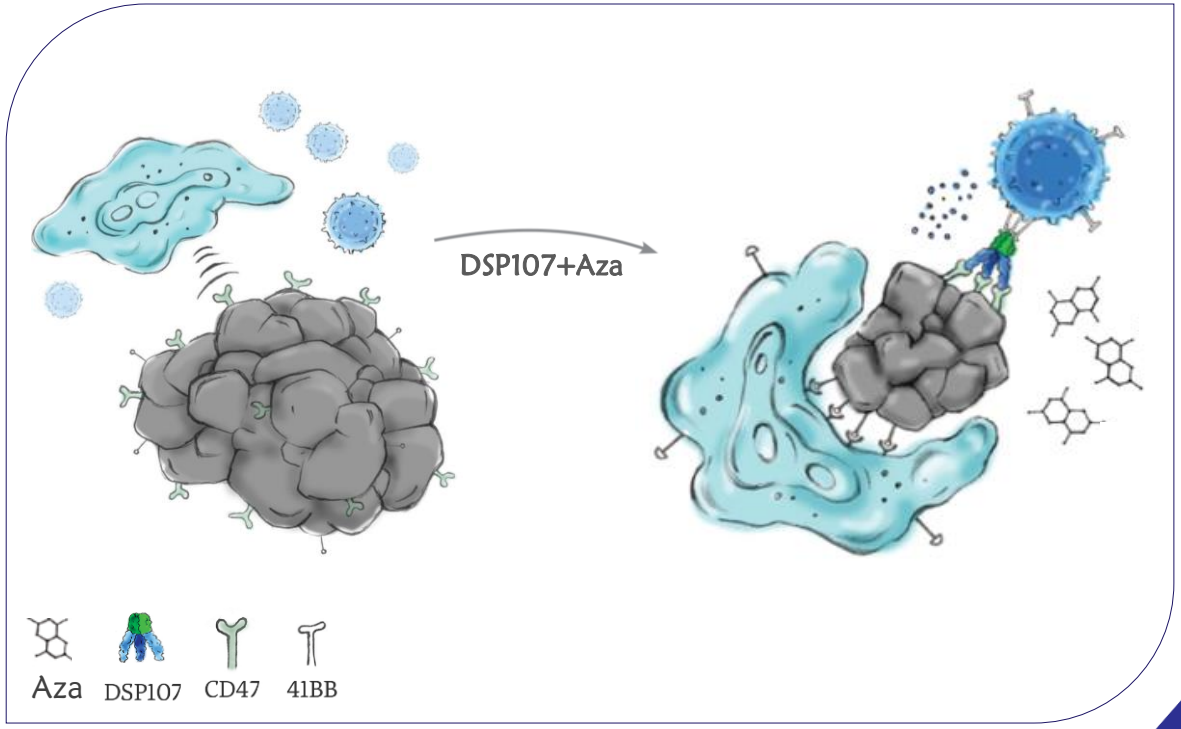
DSP107 combination with Azacytidine

Strong scientific rationale for DSP107 in MDS

- CD47 expression in MDS correlates with IPSS-R risk score suggesting a role in disease progression
- Phagocytosis induction is dependent on blocking CD47 efficiently and on the expression of pro-phagocytic "eat me" signal
- Azacitidine increases the expression of pro-phagocytic markers leading to enhanced macrophage homing and attack
- In MDS, Azacitidine induces calreticulin (eat me signal) and CD47 expression providing optimal conditions for DSP107 to work

CD47 expressed on cancer cells, delivers "dont eat me" signal inhibiting macrophages

Azacitidine increases "eat me" signal enhancing DSP107-mediated phagocytosis activity



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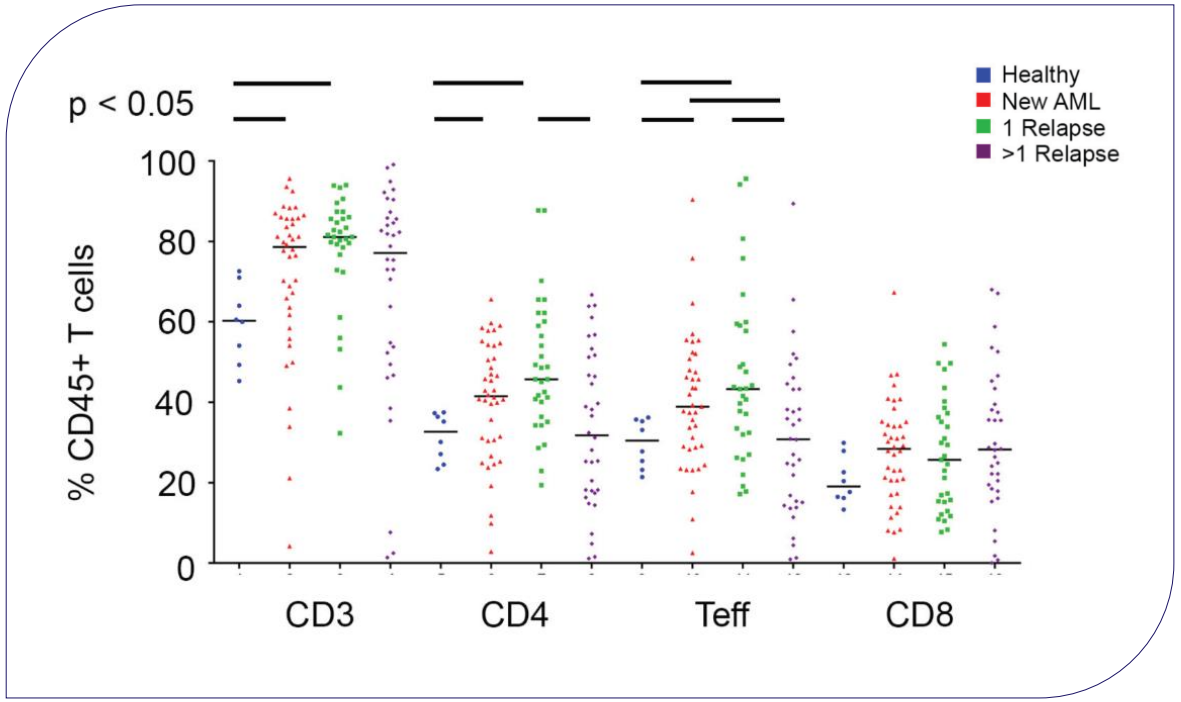
T-cell-harnessing Therapies in AML/MDS

- T-cell populations in Bone Marrow (BM) are preserved in AML patients
- The CD3 and CD8 T-cell subsets were more frequent in BMs from patients with relapsed AML vs healthy donors
- The preserved T-cell populations in AML BMs suggest a role for T-cell-harnessing therapies

T-cell subset distribution in BM aspirates from healthy donors and AML patients

Increased frequency of BM CD3, CD4, Teff and CD8 T cells in AML patients compared to healthy donors



Source: Cancer 2019;125:1470-1481

Phase Ib Dose Escalation in r/r AML/MDS Study - Design

Dose escalation trial to establish safety of DSP107 plus Azacitidine combo therapy

Relapsed/refractory AML/MDS

Phase 1 Dose escalation (n= up to 36)

Enrolling sites: MD Anderson Cancer Center

- High risk R/R MDS/CMML and AML who failed up to 2 prior therapeutic regimes
- DSP107 dose levels 0.3 - 10 mg/kg administered IV Q1W
- Objectives:
  - Assess safety and preliminary efficacy
  - Select RP2D for Phase 2 in HMA-failure high-risk MDS

Study Population

Sample size	N = 20 (0.3 - 3 mg/kg)
Sex	7 (35%) ♀ ; 13 (65%) ♂
Age	Median 68 (Range 40-81)
Tumor types	
AML	14 (70 %)
MDS	6 (30 %)
# Previous lines	Median 2 (Range 1-2)

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Phase Ib Dose Escalation in r/r AML/MDS Study - Results

Combination well Tolerated; Preliminary signal in r/r MDS patients refractory to hypomethylating agents

Summary

- Combination therapy well tolerated; No DLTs up to and including 3 mg/kg
- 4/5 r/r MDS patients with objective response in suboptimal dose levels
- All responding patients are HMA-failure MDS patients with higher risk disease including p53 mutations
- Final dose level (10 mg/kg) ongoing
- No standard of care therapy for 2nd line high risk r/r MDS; OS for these patients is less than 4 months

Patient #	Cohort	Dose (mg/kg)	Indication	Disease Detail	Previous Lines of Therapy	Previous HMA/VEN	Best response
20-010	2	1	**MDS**	High-risk, ASXL1 and TP53 mutation	2	Y/N	**PR**
20-012	2	1	AML	2ry AML, TP53 mutation	2	Y/Y	**SD**
20-013	2	1	AML	2ry AML, IDH2 and TP53 mutation	1	Y/Y	
20-015	2	1	AML	t-AML, TP53 mutation and complex monosomal karyotype	1	Y/Y	
20-016	2	1	**MDS**	Int-risk, JAK2 and TET2 mutations with monosomy 7 and trisomy 8	2	Y/Y	**CR**
20-018	2	1	AML	De novo AML with IDH1, SRSF2, RUNX1, FLT3-ITD And TAG2 mutation	2	Y/Y	
20-019	2	1	**MDS**	TP53 and IDH1 mutation	2	Y/Y	
20-020	3	3	**MDS**	Complex karyotype incl deletion 9q and TP53 mutation. IPSS-R very high	2	Y/N	**mCR**
20-021	3	3	AML	t-AML, complex karyotype and 2 TP53 mutations	2	N/Y	
20-022	3	3	AML	Diploid cytogenetics and evidence of FLT-3 ITD	2	Y/Y	
20-023	3	3	AML	Complex karyotype, KMT2A, RUNX1, loss of TP53 and t(15;17)	1	N/N	
20-025	3	3	AML	2ry AML; **MDS** with TP53 mutation and IPSS-R very high risk	1	Y/Y	**CRp**
20-026	3	3	AML	AML with MDS-related changes and complex karyotype, NPM1+, TP53+	2	Y/Y	
20-027	3	3	**MDS**	TP53 mutation and complex karyotype, IPSS-R high/very-high risk	2	Y/N	**mCR**

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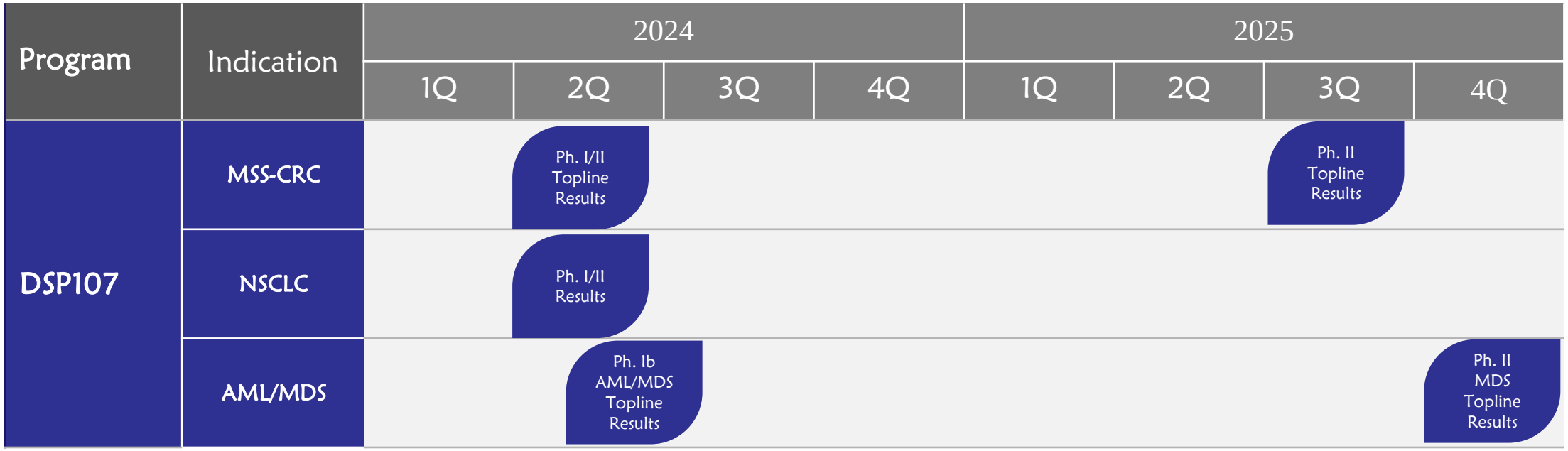
Our Path to Success

Anticipated Near Term Milestones

Multiple clinical readouts expected in the next 18 months

- Progress the clinical development of DSP107 in MSS CRC to establish a first evidence of robust efficacy for immunotherapy in the 3rd largest oncology indication (>$16B market)
- Confirm efficacy signal among MDS HMA-failure patients from phase Ib trial in dose expansion phase II trial
- Establish evidence for DSP107 potential in NSCLC



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Thank You!