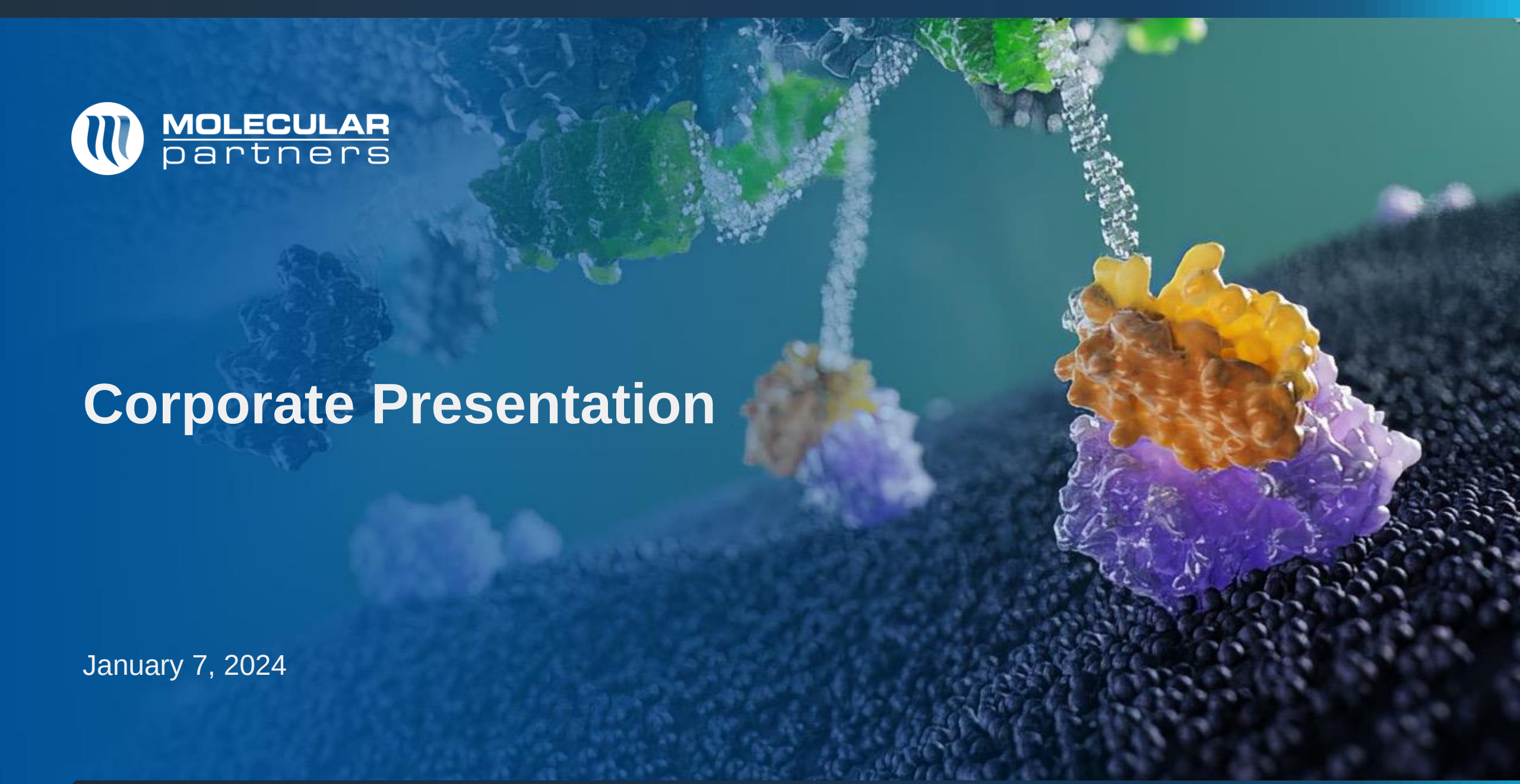


Corporate Presentation

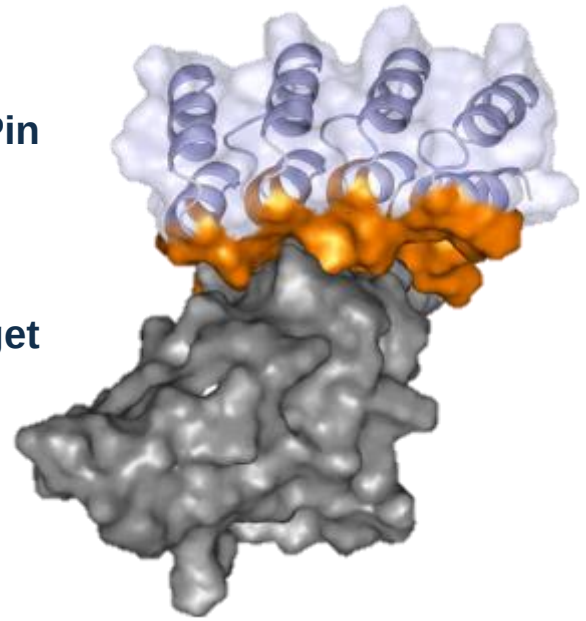
January 7, 2024

Disclaimer

This presentation contains forward looking statements. Any statements contained in this press release that do not describe historical facts may constitute forward-looking statements as that term is defined in the Private Securities Litigation Reform Act of 1995, as amended, including, without limitation, implied and express statements regarding the clinical development of Molecular Partners' current or future product candidates, expectations regarding timing for reporting data from ongoing clinical trials or the initiation of future clinical trials, the potential therapeutic and clinical benefits of Molecular Partners' product candidates, the selection and development of future antiviral or other programs, and Molecular Partners' expected business and financial outlook, including expenses and cash utilization for 2023 and its expectation of its current cash runway. These statements may be identified by words such as "believe", "expect", "may", "plan", "potential", "will", "would" and similar expressions, and are based on Molecular Partners' current beliefs and expectations. These statements involve risks and uncertainties that could cause actual results to differ materially from those reflected in such statements. Some of the key factors that could cause actual results to differ from Molecular Partners' expectations include its plans to develop and potentially commercialize its product candidates; Molecular Partners' reliance on third party partners and collaborators over which it may not always have full control; Molecular Partners' ongoing and planned clinical trials and preclinical studies for its product candidates, including the timing of such trials and studies; the risk that the results of preclinical studies and clinical trials may not be predictive of future results in connection with future clinical trials; the timing of and Molecular Partners' ability to obtain and maintain regulatory approvals for its product candidates; the extent of clinical trials potentially required for Molecular Partners' product candidates; the clinical utility and ability to achieve market acceptance of Molecular Partners' product candidates; the impact of any health pandemic, macroeconomic factors and other global events on Molecular Partners' preclinical studies, clinical trials or operations, or the operations of third parties on which it relies; Molecular Partners' plans and development of any new indications for its product candidates; Molecular Partners' commercialization, marketing and manufacturing capabilities and strategy; Molecular Partners' intellectual property position; Molecular Partners' ability to identify and in-license additional product candidates; and other risks and uncertainties that are described in the Risk Factors section of Molecular Partners' Annual Report on Form 20-F for the fiscal year ended December 31, 2022, filed with Securities and Exchange Commission (SEC) on March 9, 2023 and other filings Molecular Partners makes with the SEC. These documents are available on the Investors page of Molecular Partners' website at www.molecularpartners.com.

Any forward-looking statements speak only as of the date of this press release and are based on information available to Molecular Partners as of the date of this release, and Molecular Partners assumes no obligation to, and does not intend to, update any forward-looking statements, whether as a result of new information, future events or otherwise.

The DARPin Modality and Molecular Partners' Strategy



What we invented

- New class of therapeutics: Designed Ankyrin Repeat Proteins **(DARPins)**
- DARPins to **close the gap between small molecules and antibodies**
- 7 clinical-stage compounds, **>2500 patients treated**

How we apply it

- **Unique DARPin solutions** for a defined medical problems not addressable by antibody designs
- Demonstrate **true patient value** with **early clinical readouts**
- Combine our **capabilities with world-class partners** to deliver innovative therapeutics

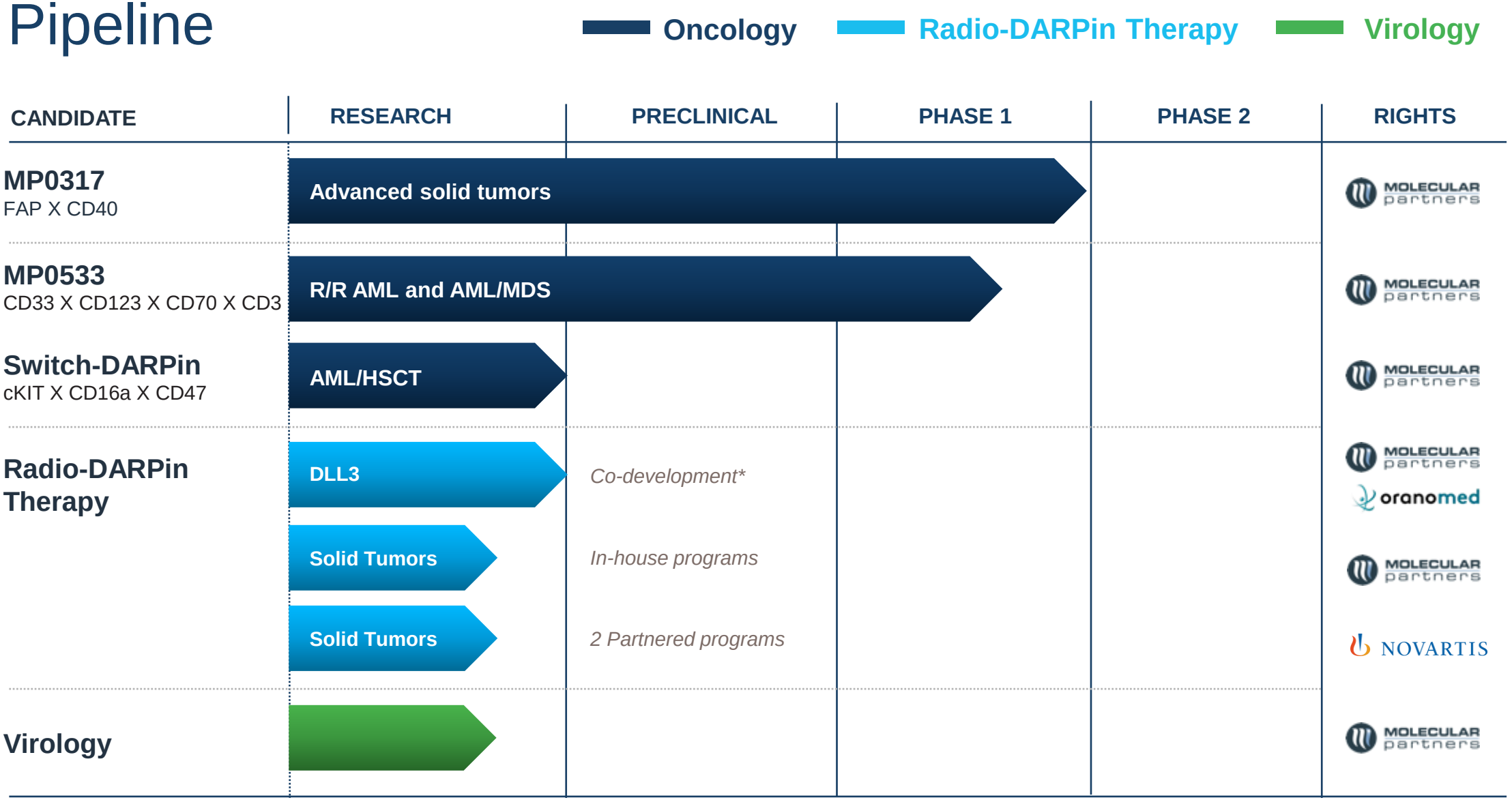
H2 2023 Highlights

MP0533	• Novel **tetra-specific T cell engager** for R/R AML and high-risk MDS/AML patients • Phase 1/2a study with dose-escalation well on track; **DR 6 enrolling patients** • **ASH 2023: encouraging initial clinical data** with acceptable safety and initial activity
Switch-DARPin	• Demonstrated **proof-of-concept** for **Switch-DARPin platform: cKIT – CD16a – CD47 in AML & HSCT**
Radio-DARPin Therapy	• Successful RDT platform optimization to **reduce kidney accumulation and increase tumor uptake** • **Collaboration agreement with Orano Med** to co-develop RDT with up to three targets, including DLL3 • Novartis collaboration progressing further
MP0317	• Bi-specific CD40 agonist targeting FAP for tumor-localized immune activation with **favorable safety profile** confirmed tumor-localized CD40 activation leading to **remodeling of TME** in patients
Operations	• Strong financial position with CHF ~187 M in cash (unaudited financials) as of Dec. 31, 2023 • **Capitalized well into 2026**

MOLECULAR partners

AML, acute myeloid leukemia; ASH, American Society of Hematology; DLL3, Delta-like ligand 3; DR, dose-regimen; EANM, European Association of Nuclear Medicine; FAP, fibroblast activation protein; MDS, myelodysplastic syndrome; RDT, Radio-DARPin Therapy; R/R, relapsed/refractory; SITC, Society for Immunotherapy of Cancer; TME, tumor microenvironment.

Pipeline



*The co-development agreement with Orano Med includes up to 3 potential oncology targets including DLL3.
AML, acute myeloid leukemia; DLL3, Delta-like ligand 3; HSCT, hematopoietic stem cell transplant; MDS, myelodysplastic syndrome; R/R, relapsed/refractory.

MP0533

Tetra-specific T cell Engager for AML

Patients with AML Have a High Unmet Medical Need

69 YEARS OLD

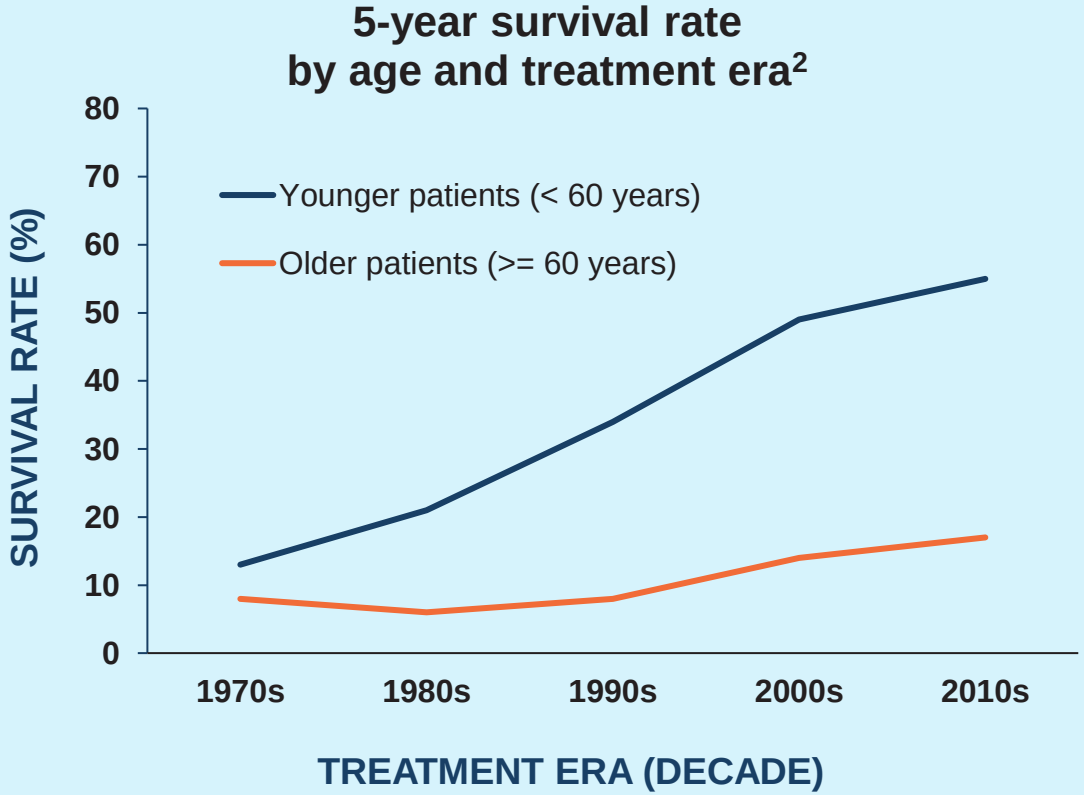
Median age of AML patients at diagnosis[1]

31.7%

Overall 5-year survival rate[1]

Despite 50 years of progress, elderly and frail patients are often not eligible for high-intensity conditioning and HSCT, and thus have limited treatment options and poor survival outcomes[2]

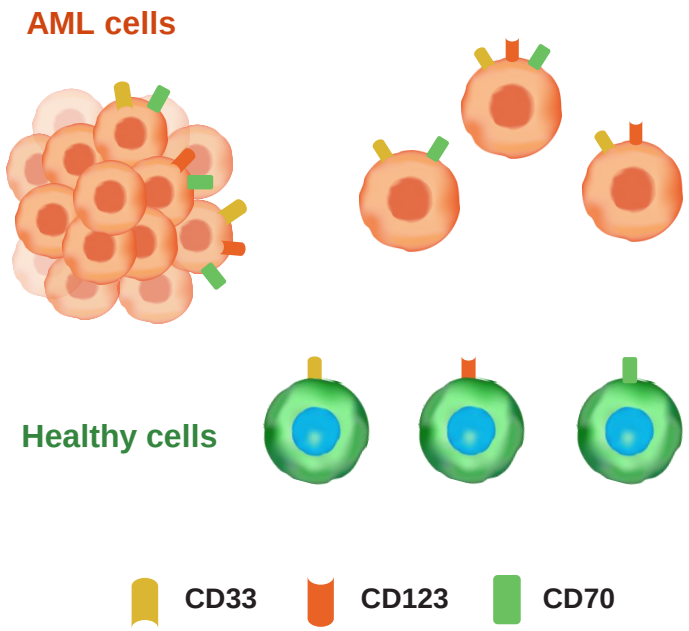
- Lack of broad and clean AML surface targets
- Risk of clonal escape even after high-intensity conditioning/HSCT



1. https://seer.cancer.gov/statfacts/html/amyl.html (based on data: 2016-2020; accessed 3 Jan 2024)
2. Kantarjian HM, *et al*. *Clin Lymphoma Myeloma Leuk* 2021; **21:**580–597.
AML, acute myeloid leukemia; HSCT, hematopoietic stem cell transplantation.

MP0533: Avidity-Driven Selectivity for Cancer Cells in AML

PROBLEM: AML-associated antigens are also expressed on healthy cells



AML remains a deadly disease and persistence of **LSCs** drives relapse

AML cell population is heterogeneous:

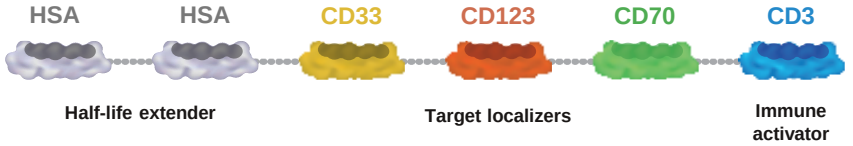
- Individual AML blasts and LSCs lack a clean target
- AML cells can be differentiated from healthy cells (e.g. HSCs) by their **co-expression of specific targets** (e.g. CD33, CD123, CD70)

AML, acute myeloid leukemia; HSC, hematopoietic stem cell; HSA, human serum albumin; LSC, leukemic stem cells.

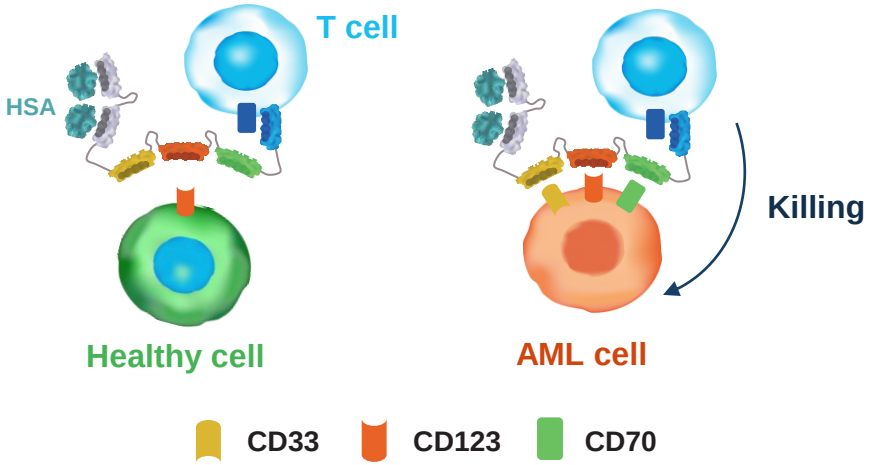
MP0533: Avidity-Driven Selectivity for Cancer Cells in AML

SOLUTION: MP0533 induces T cell-mediated killing of cells co-expressing TAAs

MP0533 design:



MP0533 MoA:



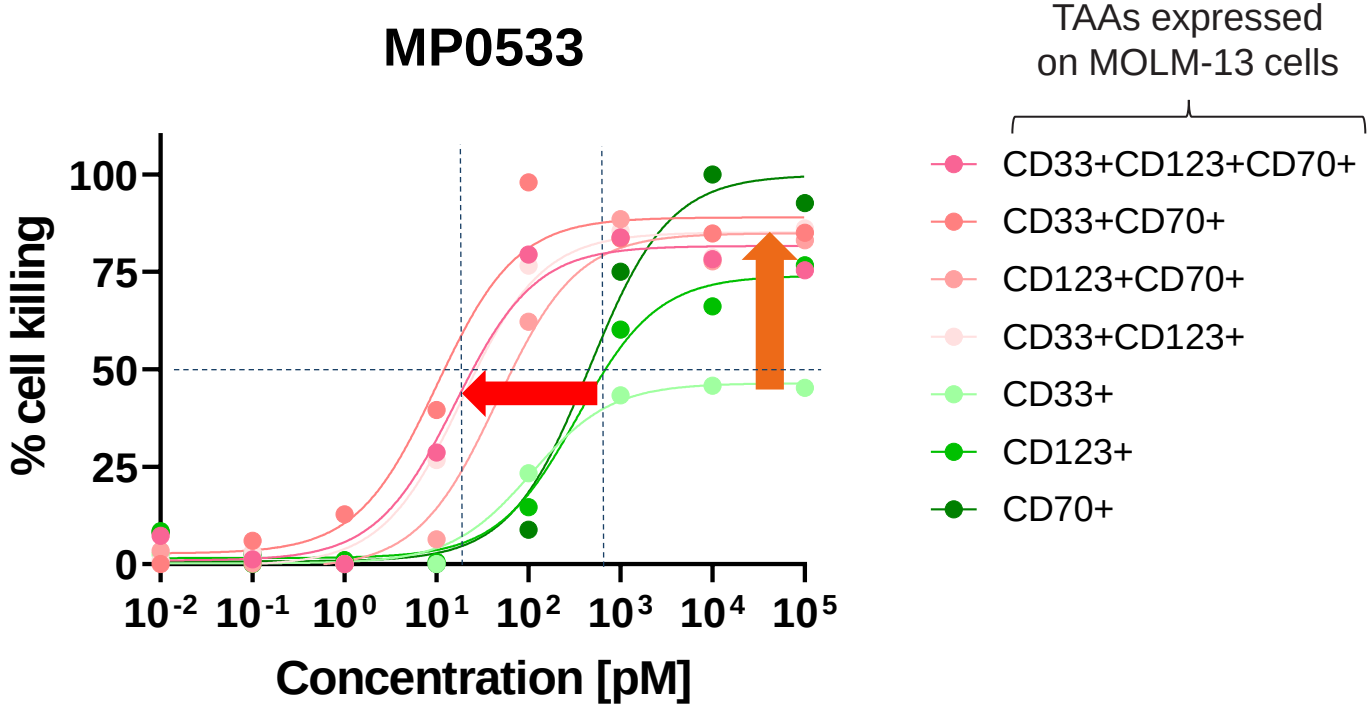
MP0533 is designed to induce **T cell-mediated killing preferentially when 2 or 3 target antigens** (CD33, CD123, CD70) **are co-expressed**

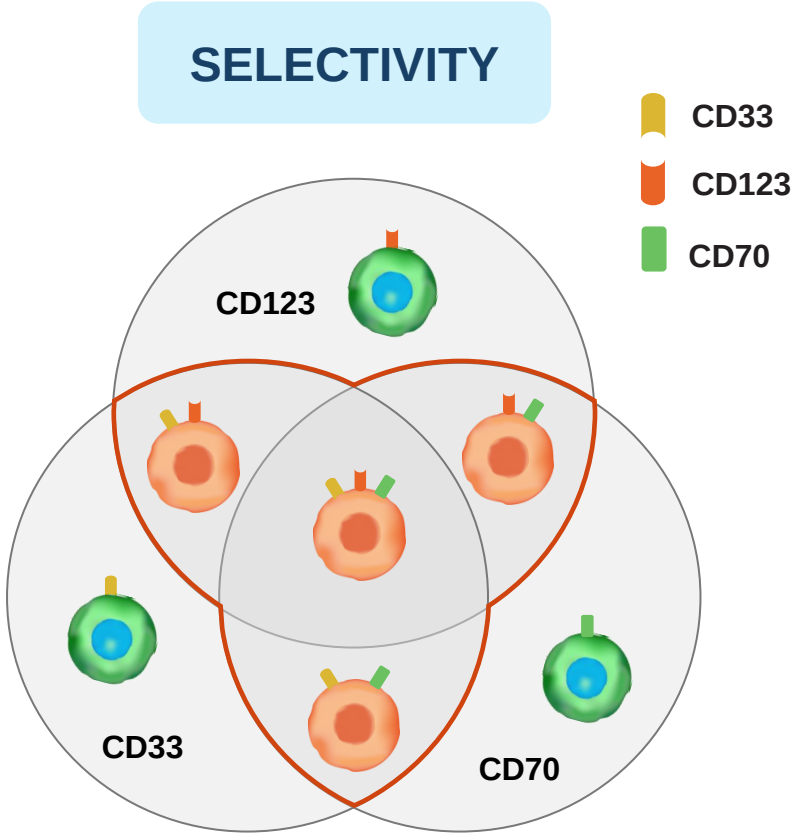
MP0533 is hypothesized to preserve healthy cells, hence **opening a therapeutic window**

MP0533 has the potential to kill all AML cells (blasts and LSCs) despite heterogeneity, ensuring **long-term disease control**

AML, acute myeloid leukemia; HSC, hematopoietic stem cell; HSA, human serum albumin; LSC, leukemic stem cells; TAA, tumor-associated antigen.

MP0533 Induces Specific Killing of AML Cells Expressing Two or Three TAAs





TAA, tumor-associated antigen.

MP0533 Phase 1 Dose-escalation Trial in R/R AML patients

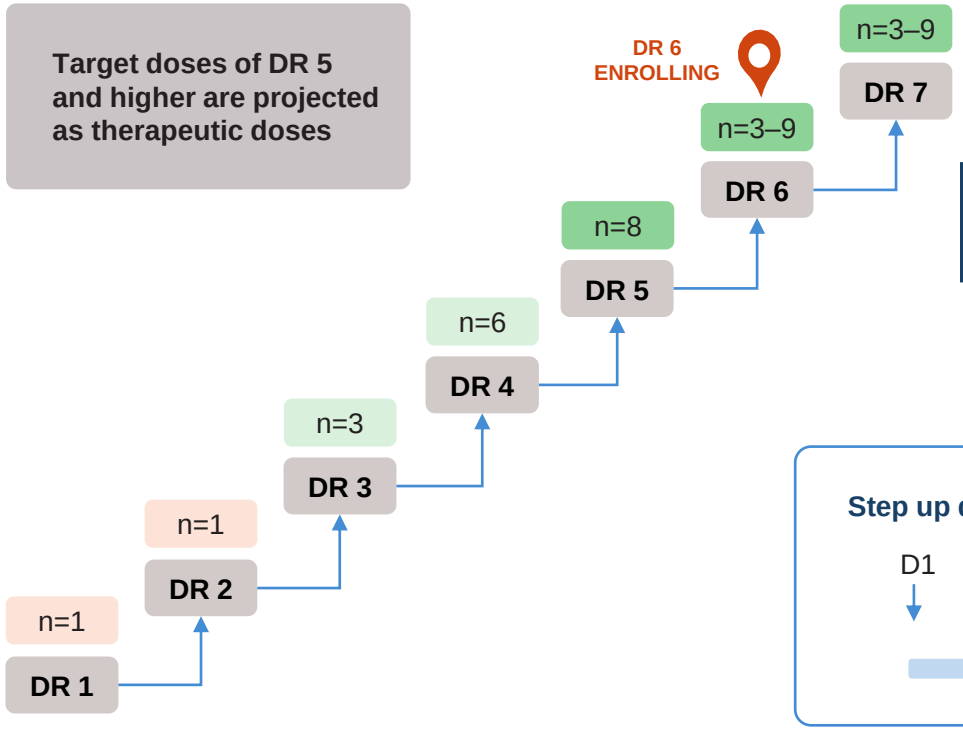
STUDY DESIGN

- FIH, multicenter, single-arm, open-label, Phase 1/2a study of MP0533 monotherapy (NCT05673057)
- Objectives: Safety/tolerability, PK/PD, and preliminary activity
- Eligible patients: Adults with R/R AML or MDS/AML
- Centers: 9 sites initiated across Europe

PHASE 1
DR ESCALATION OF MP0533 MONOTHERAPY

PHASE 2A
POC OF MP0533 MONOTHERAPY

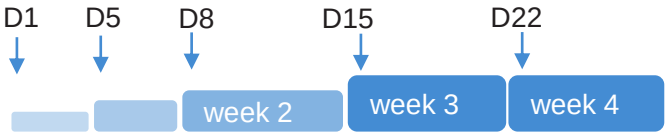
Target doses of DR 5 and higher are projected as therapeutic doses



Expansion with RP2D-R

n=30

Step up dosing in cycle 1 (= DLT period) of each DR



Study currently enrolling patients in DR 6, plans to present data at projected therapeutic doses in H1 2024

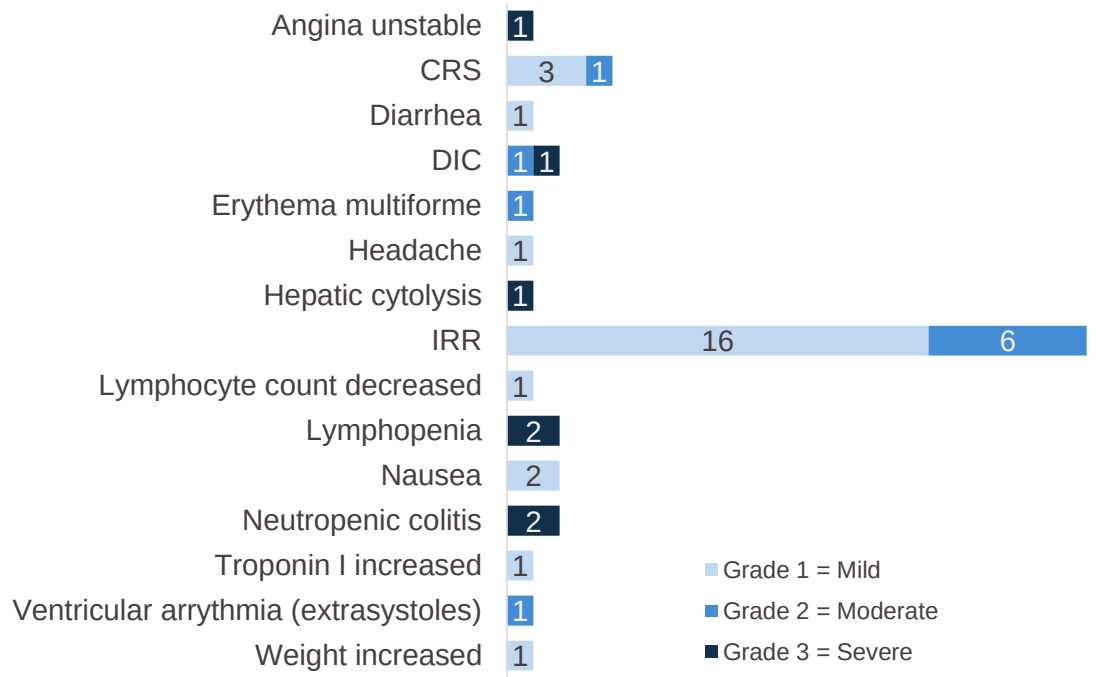
AML, acute myeloid leukemia; D, treatment cycle day; DLT, dose-limiting toxicity; DR, dose regimen; MDS, myelodysplastic syndrome; n, number of patients; PD, pharmacodynamic; PK, pharmacokinetics; POC, proof of concept; RP2D-R, recommended phase 2 DR; R/R, relapsed/refractory.

MP0533 - Patient Characteristics and Safety Profile

PATIENT CHARACTERISTICS	DR COHORTS 1–4 (n=11)
Sex, n (%)	
Female / male	5 (45) / 6 (55)
Age	
Mean / Median (range)	66 / 75 (26–81)
ECOG PS, n (%)	
0 / 1 / 2	4 (36) / 5 (46) / 2 (18)
Hematologic malignancy, n (%)	
AML / MDS/AML	9 (82) / 2 (18)
ELN risk category, n (%)	
Intermediate / adverse	1 (9) / 10 (91)*
No. of prior systemic treatment lines, n (%)	
1 / 2 / 3	4 (36) / 5 (46) / 2 (18)

*TP53 mutated: 3 (27%)

MP0533-RELATED TEAEs (n=43 reported)



Acceptable safety profile for MP0533 reported for DR 1–4 (11 patients):

- Overall, AE profile consistent with AML and elderly/heavily pretreated patients with many comorbidities
- IRR and CRS are the most frequent MP0533-related TEAEs (Grade 1–2)
- No DLTs in any of the MP0533 DRs to date

Pabst et al, ASH 2023 poster presentation (data cut-off 24 Oct 2023)

AE, adverse event; CRS, cytokine release syndrome; DIC, disseminated intravascular coagulation; ELN, European LeukemiaNet; IRR, infusion-related reaction; TEAE, treatment-emerging AE.

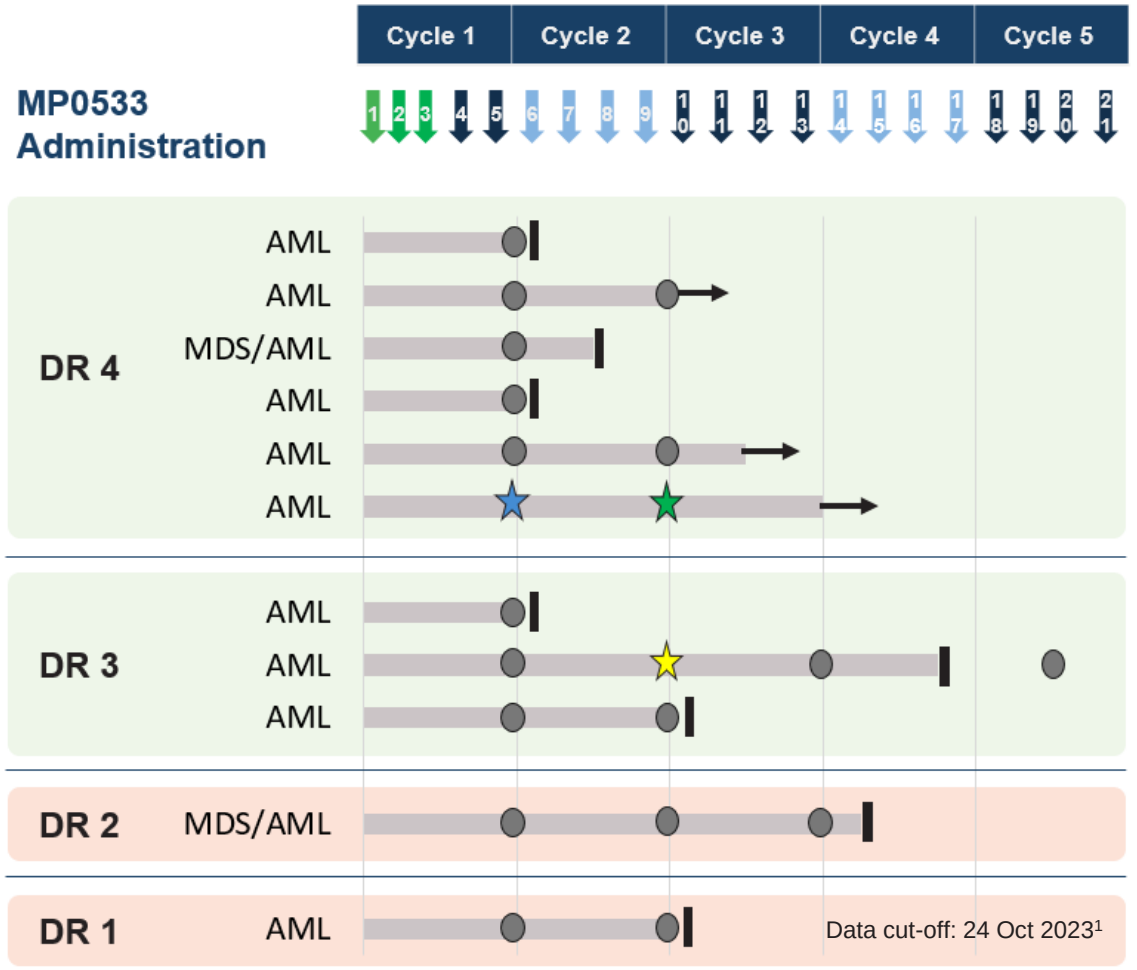
MP0533 Treatment and Clinical Response

Encouraging initial clinical data with two responders[1]

- CR in 1 patient at DR 4
- MLFS in 1 patient at DR 3

Currently enrolling patients in DR 6

- DR 5 and above are projected as therapeutically active doses
- Next update in H1 2024



LEGEND

- CR
- CRi
- MLFS
- No response

Response (2022 ELN[2]) was assessed every 4 weeks until disease progression and results are presented as indicated

- MP0533 treatment
- Treatment continuation at data cut-off
- Treatment discontinuation

Arrows at the top indicate MP0533 administration at D1, D5, D8, D15 and weekly thereafter

- Step-up dosing is presented in green arrows
- Color changes in blue arrows indicate start of a new 28-day cycle

[1]Pabst et al. ASH 2023 poster presentation; [2]Döhner et al. Blood 2022;140(12)1345-77.
CR, complete response; CRi, CR with incomplete hematologic recovery; ELN, European LeukemiaNet; MLFS, morphologic leukemia-free state.

Switch-DARPin Platform & first program for HSCT in AML

Targeted and conditional activation of immune cells

Next-Generation Conditioning for HSCT in AML and Beyond

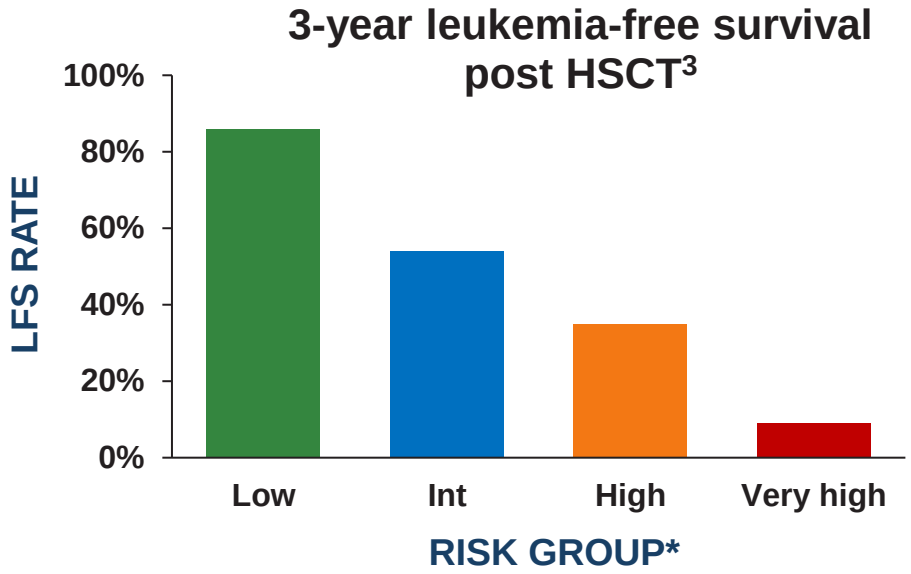
HSCT is potentially curative for AML, however:

Conditioning regimens followed by HSCT do not always kill all AML cells[1,2]

→ Many patients **relapse post HSCT**, especially AML patients with poor cytogenetic risk profile

High-intensity conditioning regimen bears high toxicity[1,2]

→ Many patients receive **reduced intensity conditioning with higher risk of relapse** or do not qualify for HSCT



Opportunity for next-generation conditioning regimen

- Induce deep molecular remission to kill all AML clones, including in patients with poor genetic risk profile
- Limit toxicity to allow access to HSCT for more AML patients, including elderly or frail patients
- Beyond AML: broaden applicability of HSCT for other diseases (e.g., genetic diseases) by improving safety of conditioning regimen

1.Jethava et al. Bone Marrow Transplant 2017;52:1504-11; 2. Döhner et al. Blood 2022;140:1345-77; 3. Moses Murdock et al. Blood 2022;139:3546-57.
*Risk groups defined based on different variables, including age, cytogenetic risk profile, type of and response to conditioning.
AML, acute myeloid leukemia; HSCT, hematopoietic stem cell transplantation; Int, intermediate; LFS, leukemia-free survival.

Next-Generation Conditioning for HSCT in AML

Target cKIT to eliminate HSCs/LSCs

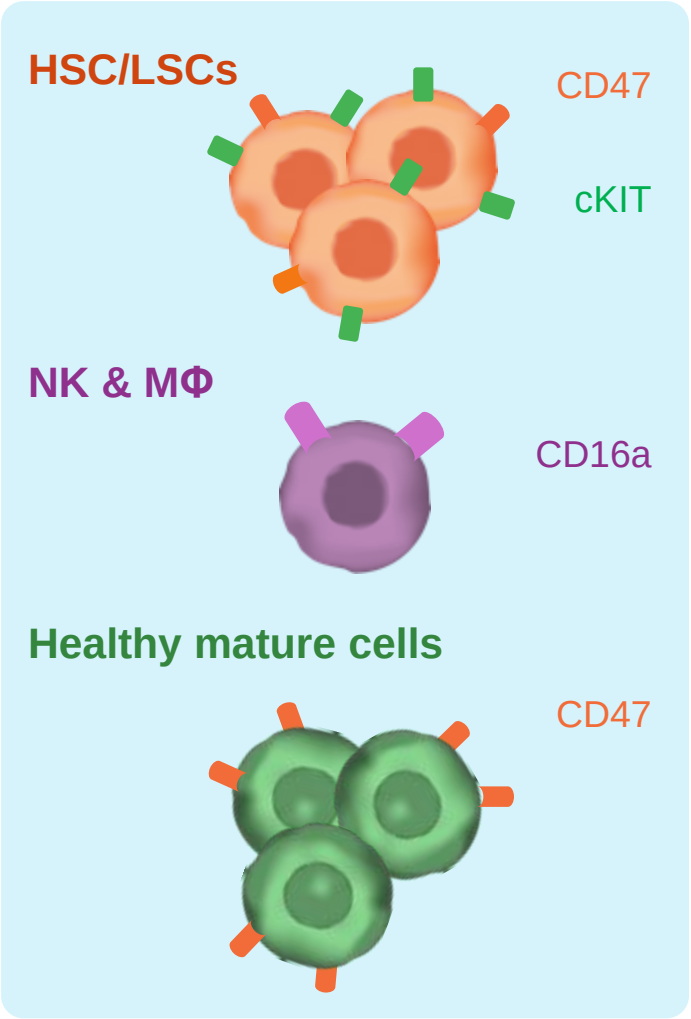
- cKIT is critical for stem cell maintenance and renewal[1, 2]
- Simple antagonists (mAbs) to cKIT are not potent enough

Engage NK cells and macrophages (MΦ) via CD16a to kill HSCs/LSCs

- Effective and safe approach
- NK and MΦ activity is limited by CD47 expression on HSC/LSCs[3]

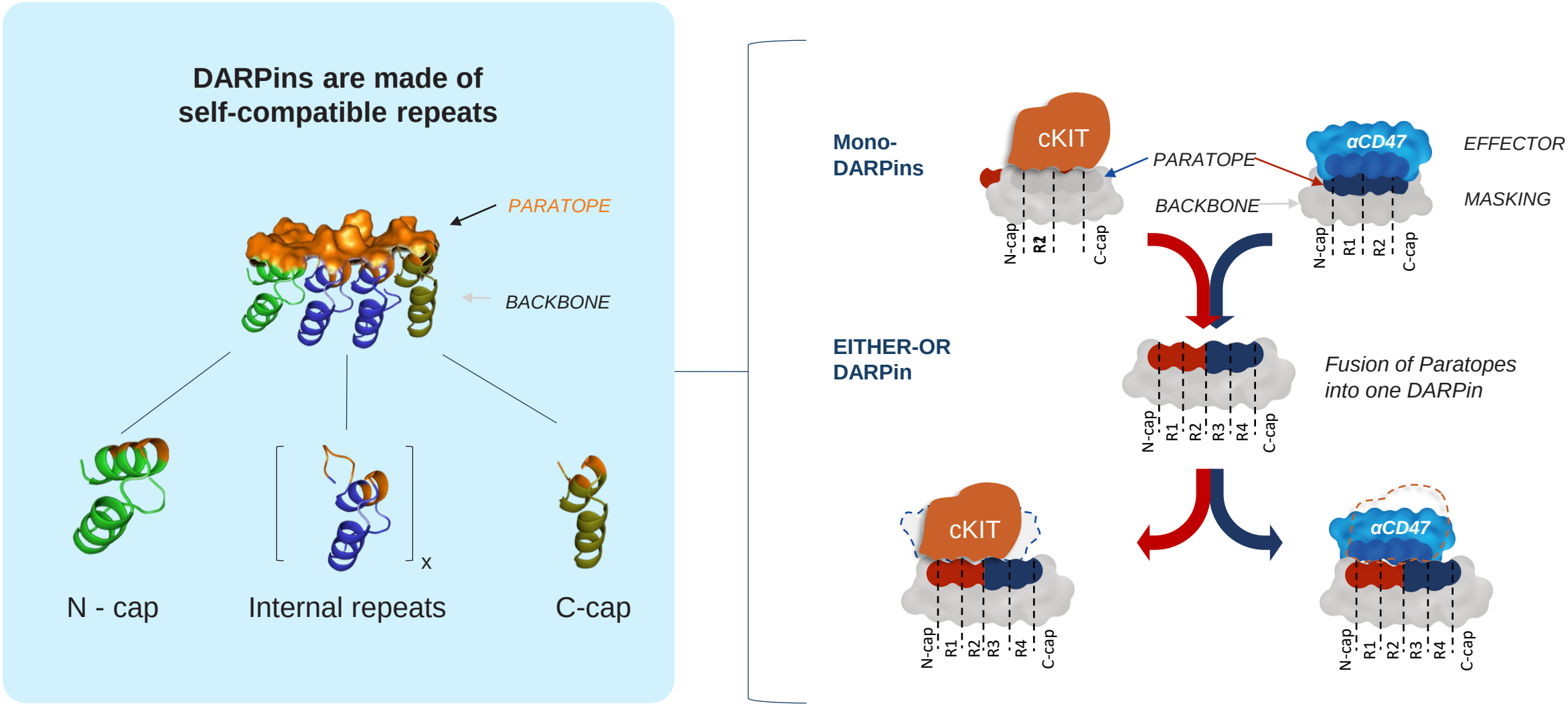
Conditionally block CD47 on LSCs/HSCs to boost NK cell and MΦ killing activity

- CD47 is widely expressed as "do-not-eat-me signal" and prevents killing of cells, including HSCs/LSCs[1,3]
- Swich MoA allows conditional local blocking of CD47 on HSCs/LSCs



1. Valent et al. Int J Mol Sci 2019; 2. Rev in Kent et al. molecular pathways 2008; 3.Chhabra, Weissman and Shizuru, STM 2016.
HSC, hematopoietic stem cell; LSC, leukemic stem cell; mAb, monoclonal antibody; MoA, mode of action; MΦ, macrophage.

Either-Or DARPin: exclusive binding to cKIT protein or to αCD47 DARPin



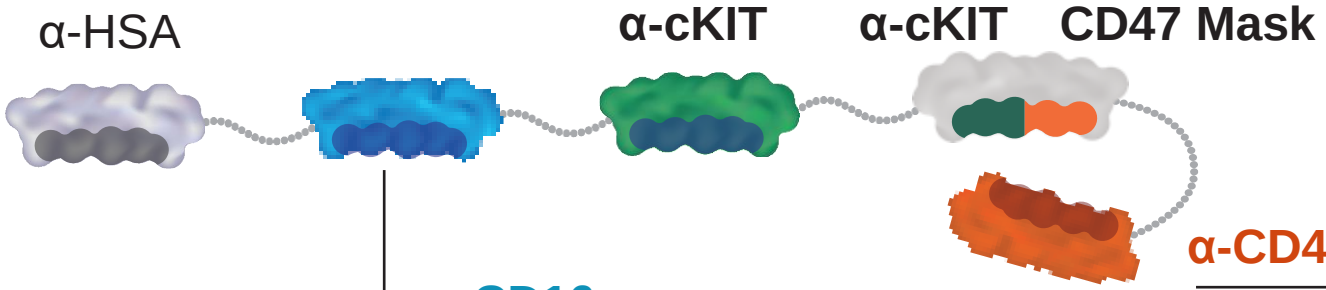
cKIT x CD16a x CD47 Switch-DARPin

Our solution for a safe conditioning regimen and long-term disease control

cKIT (CD117)

HSC marker essential for HSC maintenance and renewal

- **Challenge:** optimal HSC depletion requires both cKIT blocking AND potent immune cell mediated killing
- cKIT-CD16a-CD47 Switch-DARPin proposed to demonstrate full activity



Switch-DARPin

Prevents peripheral CD47 blockade

- Higher safety
- Expected better biodistribution
- Allows use of Fc-engaging modalities

α-CD16a

Innate immune cell engager

CD16a effector function

- ADCC and ADCP induction
- No impact of inhibitory Fc
- Reduced CRS (compared to TCE)

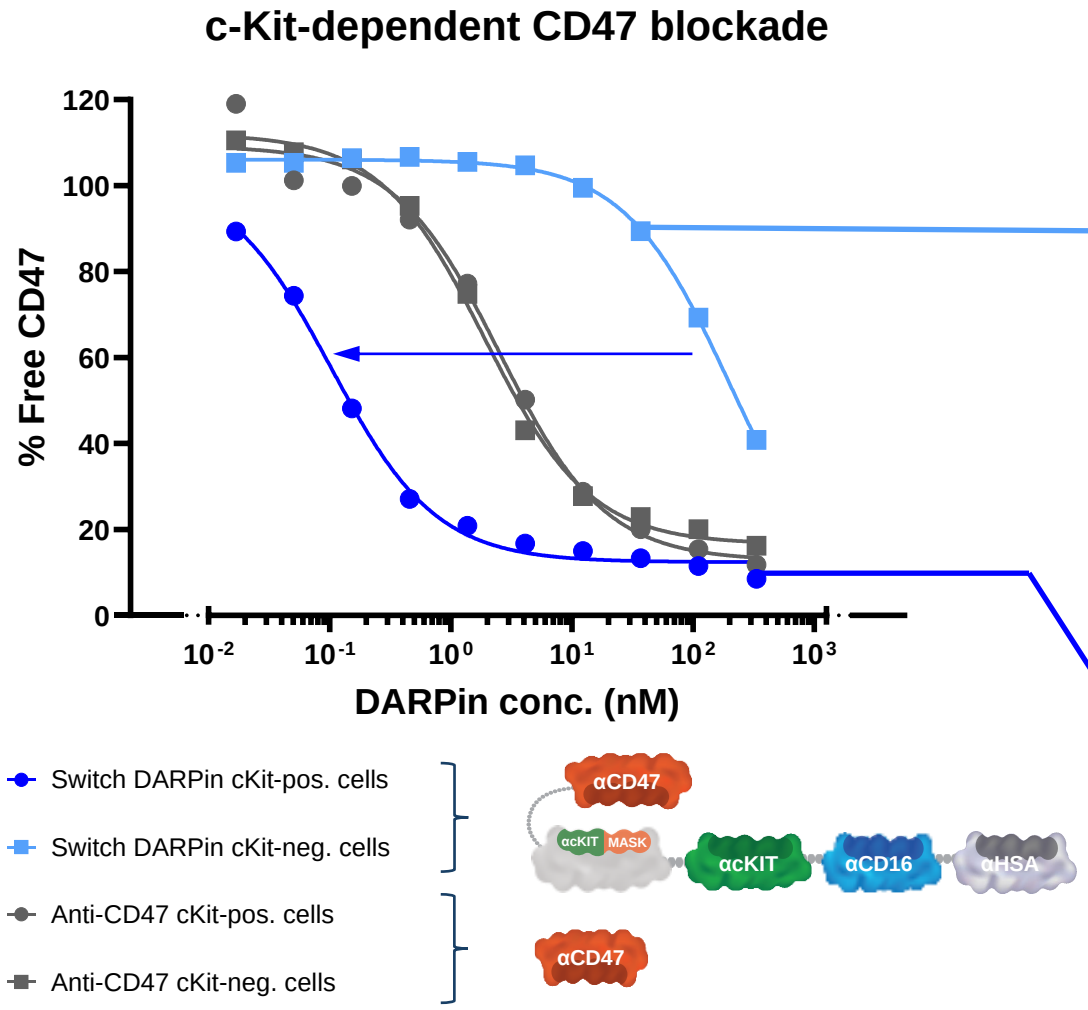
α-CD47

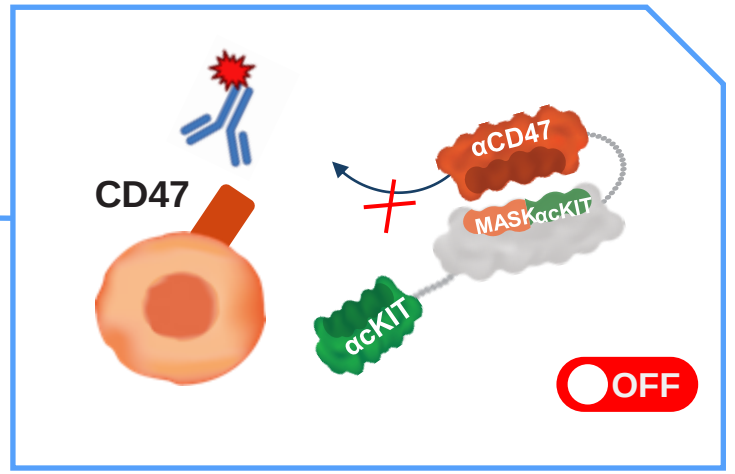
CD47 innate checkpoint blockade

- Block "do-not-eat-me" signal = enhances phagocytosis
- High expression on HSC = target for ADCC and ADCP

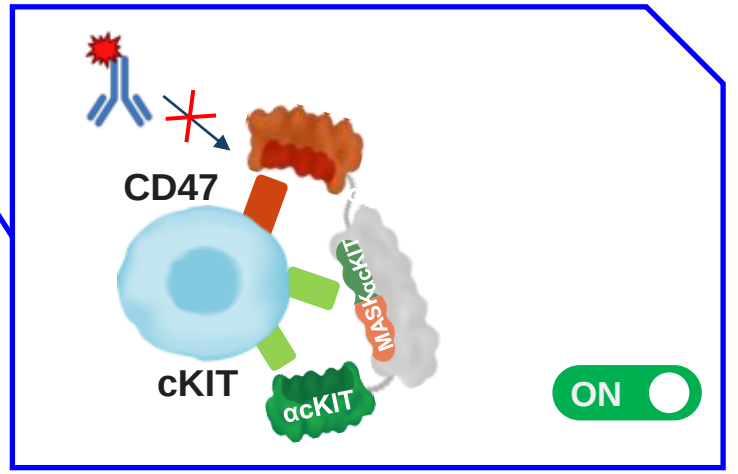
ADCC, antibody-dependent cell-mediated cytotoxicity; ADCP, antibody-dependent cellular phagocytosis; CRS, cytokine release syndrome; TCE, T-cell engager

Switch-DARPin POC - CD47 is Blocked Only on cKIT Positive Cells





cKIT Negative cells
Switch is OFF
CD47 is NOT blocked



cKIT Positive cells
Switch is ON
CD47 is Blocked

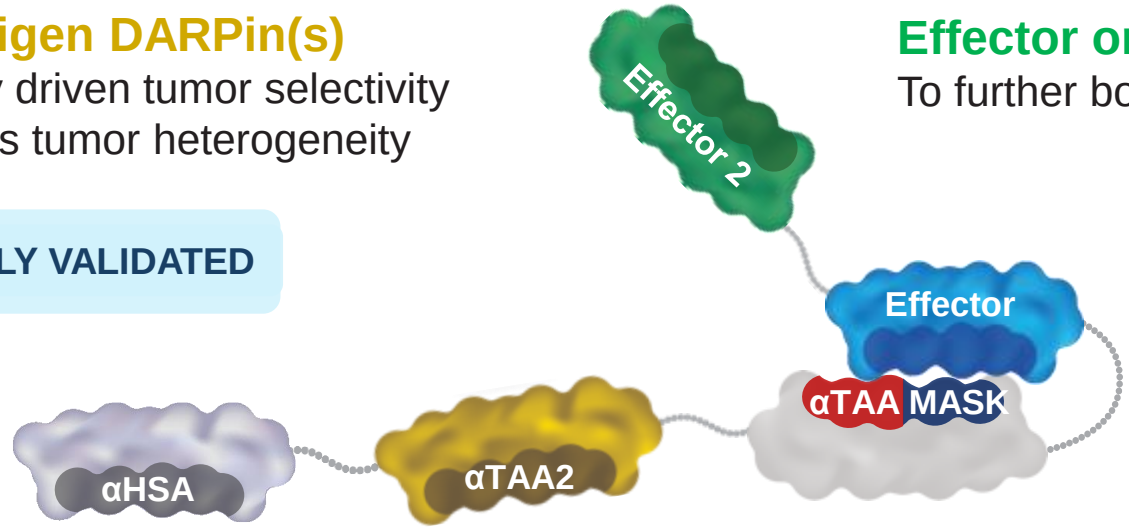
anti-CD47
detection agent

POC, proof-of-concept;

Logic-gated Switch-DARPins
Swiss knives for enhanced immune engagers

Tumor Antigen DARPin(s)
+1 for avidity driven tumor selectivity
+2 to address tumor heterogeneity

✓CLINICALLY VALIDATED



Effector or Co-stimulating DARPin
To further boost immune response

Effector DARPin
CD3 engager, CD47 Blocker, ...

✓CD3 ENG CLINICALLY VALIDATED

HSA DARPin(s)
For Half life extension

✓CLINICALLY VALIDATED

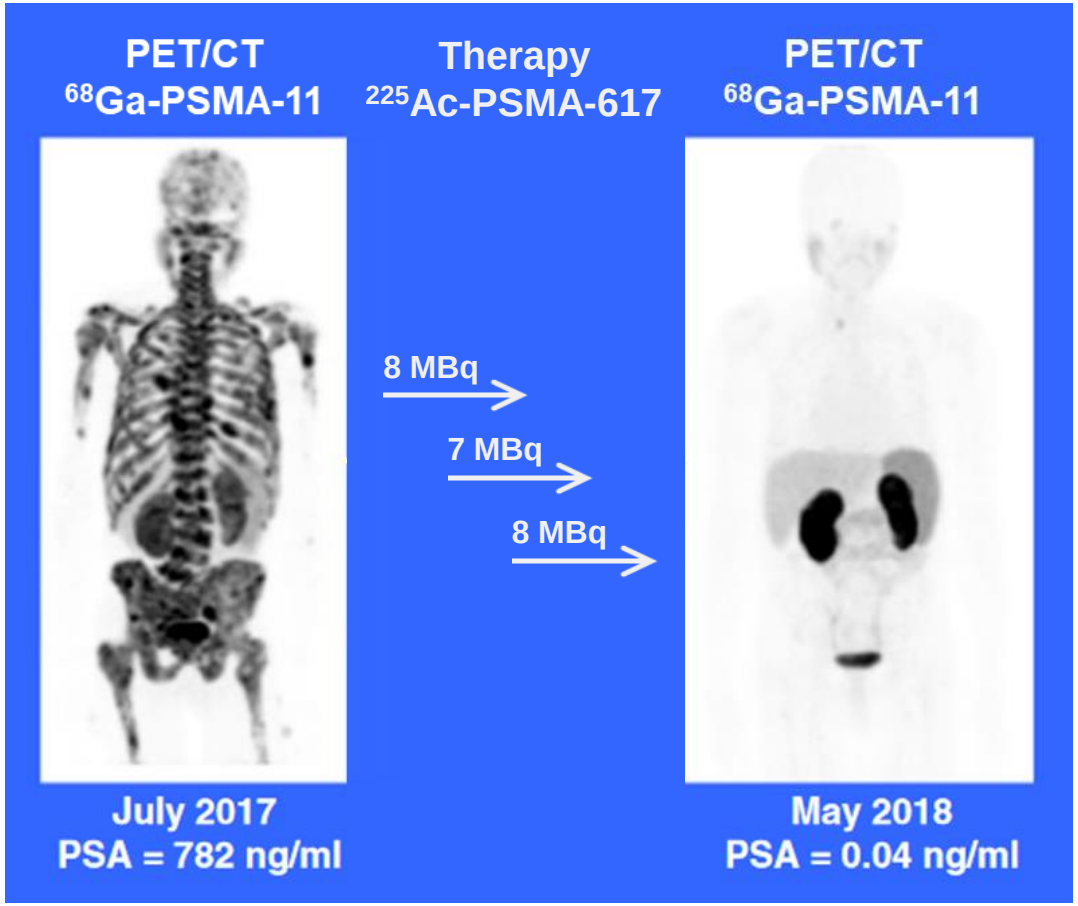
SWITCH DARPin
to prevent systemic immune-cell activation
- Allows safe use of potent immune-cell effectors
- Better biodistribution (no immune target-mediated sink)

hypothetical sketch

Radio-DARPin Therapy

Platform & Pipeline

Targeted Radiotherapy: “Old” Modality Turned Hot Through Precision

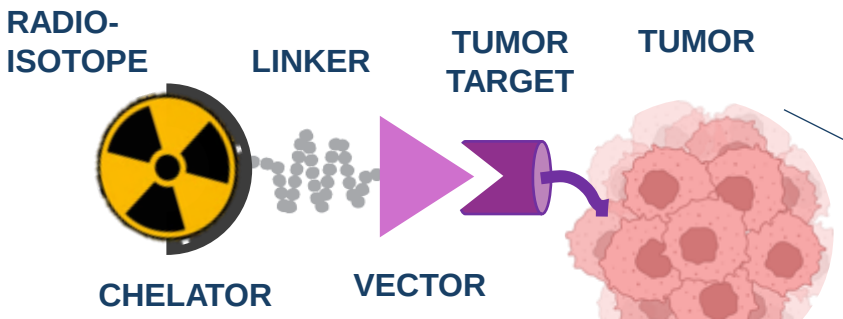


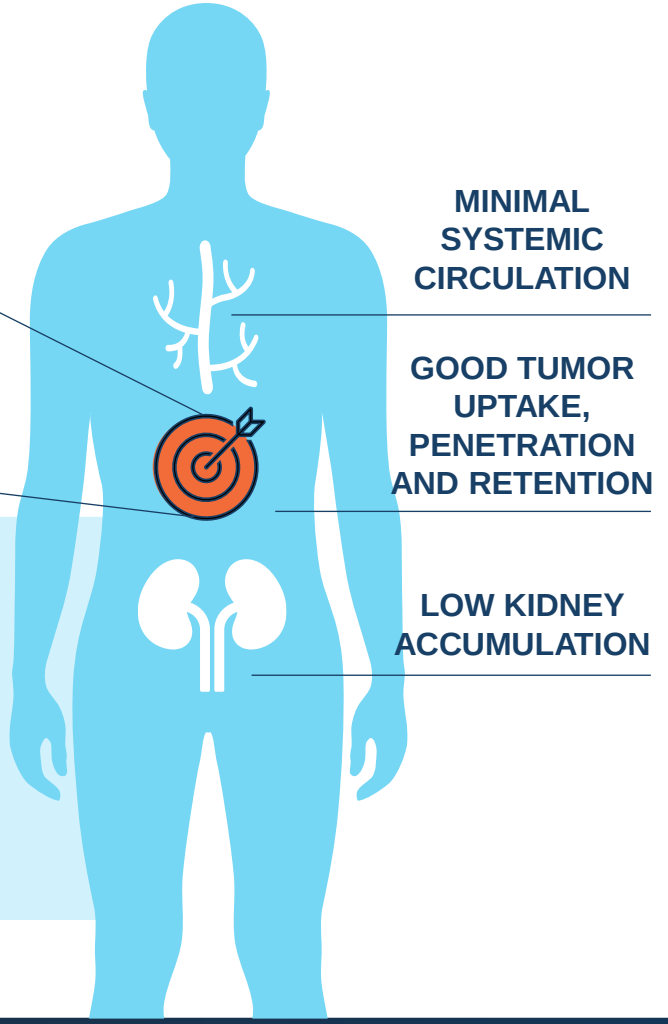
Example: Treatment of naïve prostate cancer patient with extensive bone metastasis with ^{225}Ac-PSMA-617

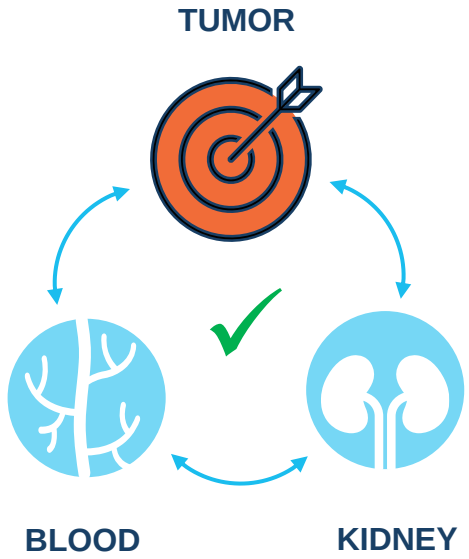
- ✓ Proven strong clinical **efficacy**, including oligo- and multi-metastatic disease, and good **tolerability**
- ✓ Theranostics approach **“see what you treat”**
- ✓ **Supply chain** challenges being solved (next 3-5 years)
- ✓ **Increased coordination** among Oncologists, Radiologists and Nuclear Medicine Docs
- X **Vectors** matching targeted radiotherapy requirements & spanning a broad tumor target space are limiting the expansion to other relevant cancer types

PET/CT scan pictures adapted from Sathekge M, et al. 225Ac-PSMA-617 in chemotherapy-naive patients with advanced prostate cancer: a pilot study. Eur J Nucl Med Mol Imaging 46, 129–138 (2019). https://doi.org/10.1007/s00259-018-4167-0.

Ideal Properties of Radiotherapy Product Candidate







Deliver radioisotope selectively to the tumor while sparing healthy tissues

"special focus on kidneys and bone marrow (blood), which are the most frequent dose-limiting organs"

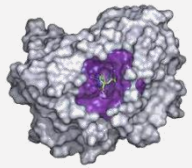
LMW Molecules as Ideal Vectors but Limited Target Space

LMW MOLECULES

Targets with cavity where a Low Molecular Weight (LMW) vector with high affinity and specificity can be identified

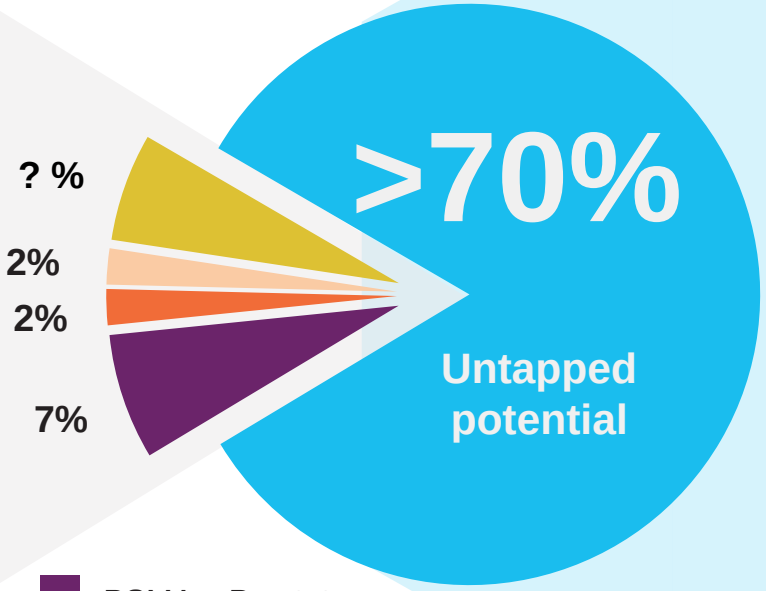
Target Examples:

PSMA
SSTR2



PSMA (1Z8L)

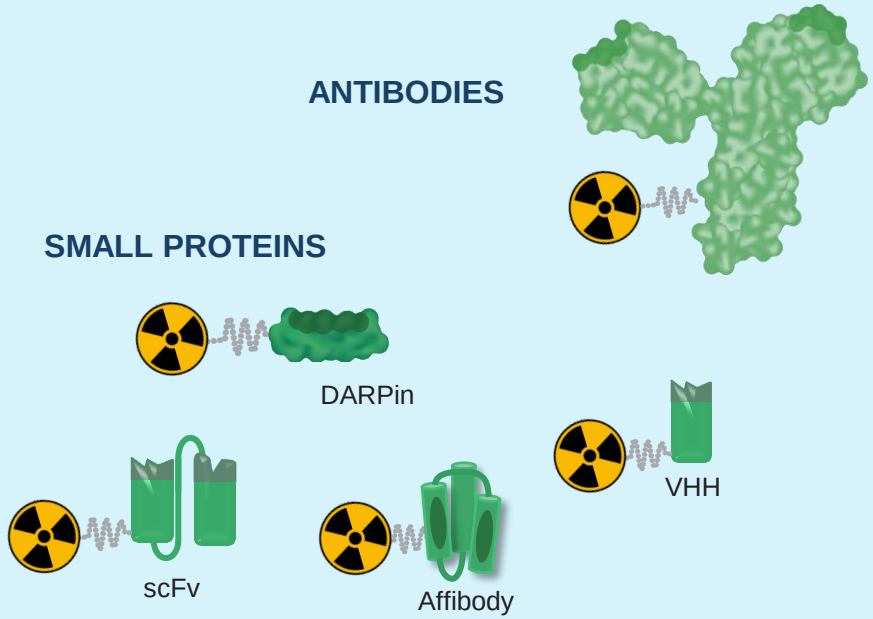
TARGET SPACE*



- PSMA + Prostate
- SSTR2 + NETs
- SSTR2 expansion
- Potential other LMW suitable targets**
- Other tumor targets & cancers

PROTEIN - PROTEIN BINDERS

Proven class for high affinity & specificity binding of protein surfaces of broad range of tumor targets



However, all protein-based vectors have key limitations for effective and safe targeted radiotherapy!

* Source: Guggenheim Securities Report 2023
** e.g. FAP, CAIX, FOLR1, NTSR1, Eph2A, GPC3, MC2R, GRPR, ITGB6

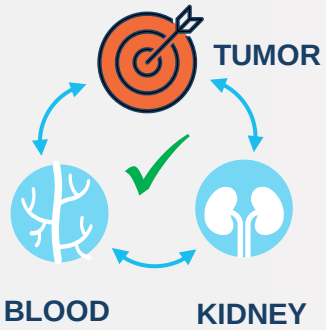
Strengths and Weaknesses of Vectors for Targeted Radiotherapy

LMW molecules

50-1500 Da

- ✓ Fast entry and exit from the body to limit exposure of healthy tissue
- ✓ Good tumor uptake and penetration
- X Limited target space due to nature of binding (affinity and selectivity)

20% of targets?

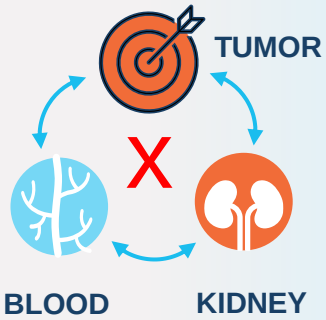


Peptides

500-5´000 Da

- ✓ Fast entry and exit from the body to limit exposure of healthy tissue
- ✓ Good tumor uptake and penetration
- X Low predictability of vector discovery (+ target space?)
- X Often high kidney accumulation

+10% of targets?

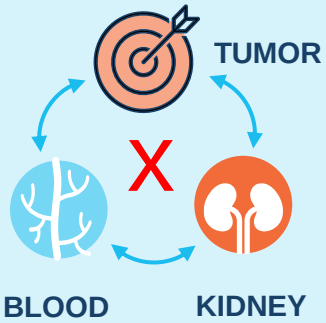


Small proteins

5´000 – 30´000 Da

- ✓ Fast entry and exit from the body to limit exposure of healthy tissue
- ✓ Proven class of binding proteins for broad range of tumor targets
- ~ Lower tumor uptake
- X High kidney accumulation

+70% of targets?



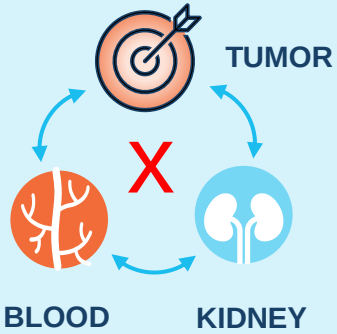
Radio-DARPin Therapeutics Opportunity

Antibodies

150´000 Da

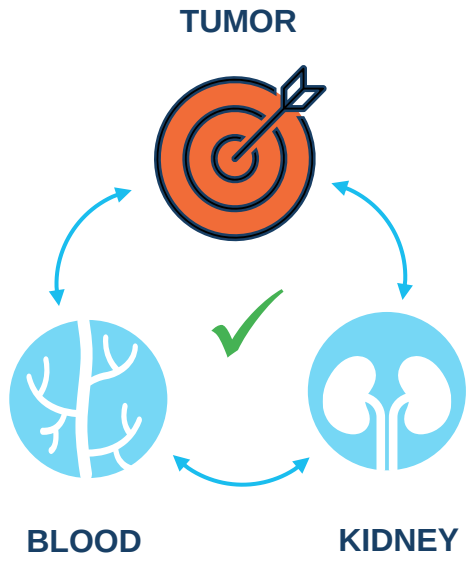
- ✓ Proven class of binding proteins for broad range of tumor targets
- ✓ Good tumor uptake
- X Limited tumor penetration
- X Stay in the body too long, exposing bone marrow to radiation

+70% of targets?



LMW, low molecular weight

The Ideal Targeted Radiotherapy Vector Platform



Efficacy – Tumor

High tumor uptake:
Concentration at site of action

Deep tumor penetration:
Access to site of action

Long tumor retention:
Maintenance at site of action

Safety – Blood & Kidney

Short systemic half-life:
Low risk for bone marrow tox & early imaging

Low kidney accumulation:
Low risk of kidney toxicity

Low accumulation in other healthy tissue:
Low risk of healthy organ tox

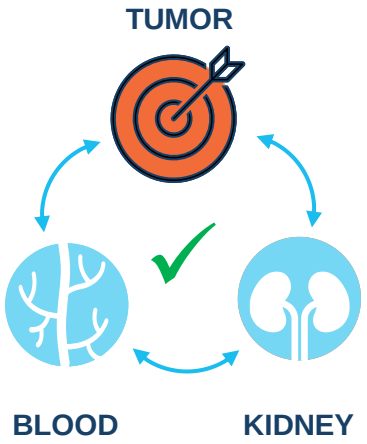
Target Space & Product Engine

- **Broad target range:**
  Cover many tumor targets and cancer types
- **Predictable vector discovery:**
  High PoTs and short timelines to lead
- **Developability**: Simple coupling chemistry & tolerance of harsh conditions

PoTs, probability of technical success

DARPins' Innate Properties Favorable for Radiotherapy Vector Use

Build on DARPin advantages to resolve limiting dimensions



Efficacy – Tumor

High tumor uptake:
Concentration at site of action
~ **BOOST BY BUILDING ON HLE TUNING EXPERTISE**

Deep tumor penetration:
Access to site of action
✓ **SMALL SIZE**

Long tumor retention:
Maintenance at site of action
✓ **HIGH AFFINITY**

Safety – Blood & Kidney

Short systemic half-life:
Low risk for bone marrow tox & early imaging
✓ **SMALL SIZE**

Low kidney accumulation:
Low risk of kidney toxicity
X **STEALTH KIDNEY DARPin (ROBUST ARCHITECTURE)**

Low accumulation other tissue:
Low risk of healthy organ tox
✓ **HIGH SELECTIVITY**

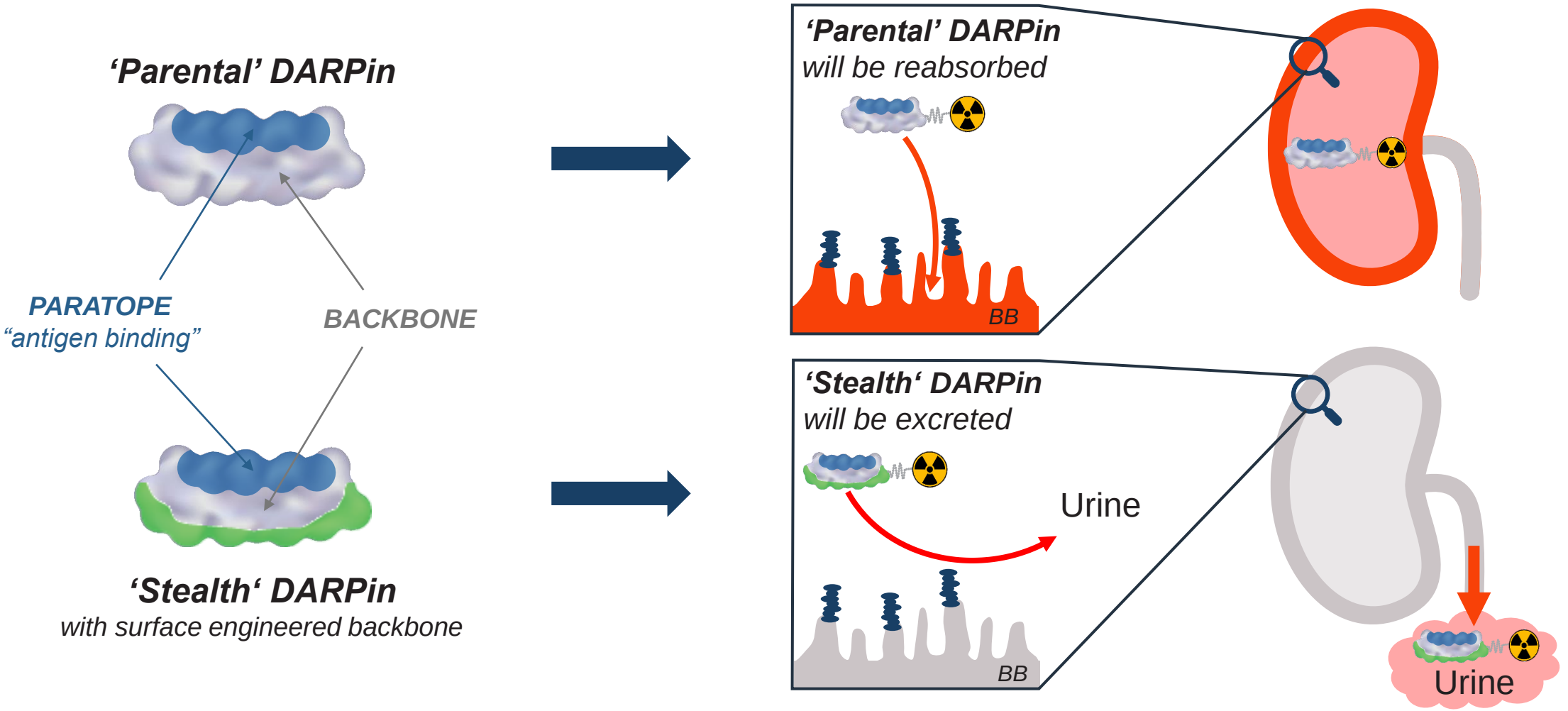
Target Space & Product Engine

- **Broad target range:**
  Cover many tumor targets and cancer types
  ✓ **DARPins TO >100 TARGETS***
- **Predictable vector discovery:**
  High PoTs and short timelines to lead
  GOAL TRANSFERABLE PLATFORM LEARNINGS
- **Developability**:
  Simple coupling chemistry & tolerance of harsh conditions
  ✓ **ROBUST ARCHITECTURE**

PoTs, probability of technical success; HLE, half-life extension
*14 mono-DARPins are components of multi-DARPins clinical stage compounds

Surface Engineering to Reduce Kidney Accumulation

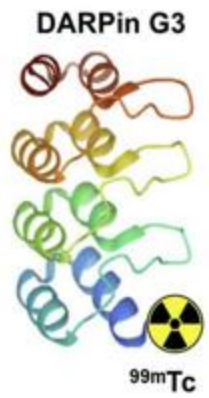
Enabled by the robust architecture of DARPin scaffold

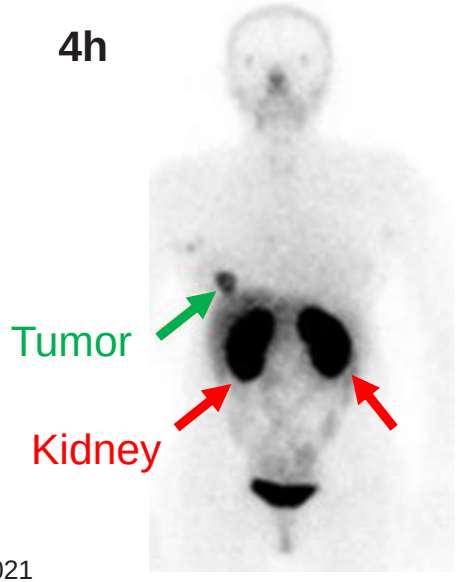


Surface Engineering to Reduce Kidney Accumulation

Enabled by the robust architecture of DARPin scaffold

Polypeptides & proteins < 60 kDa are reabsorbed by kidneys

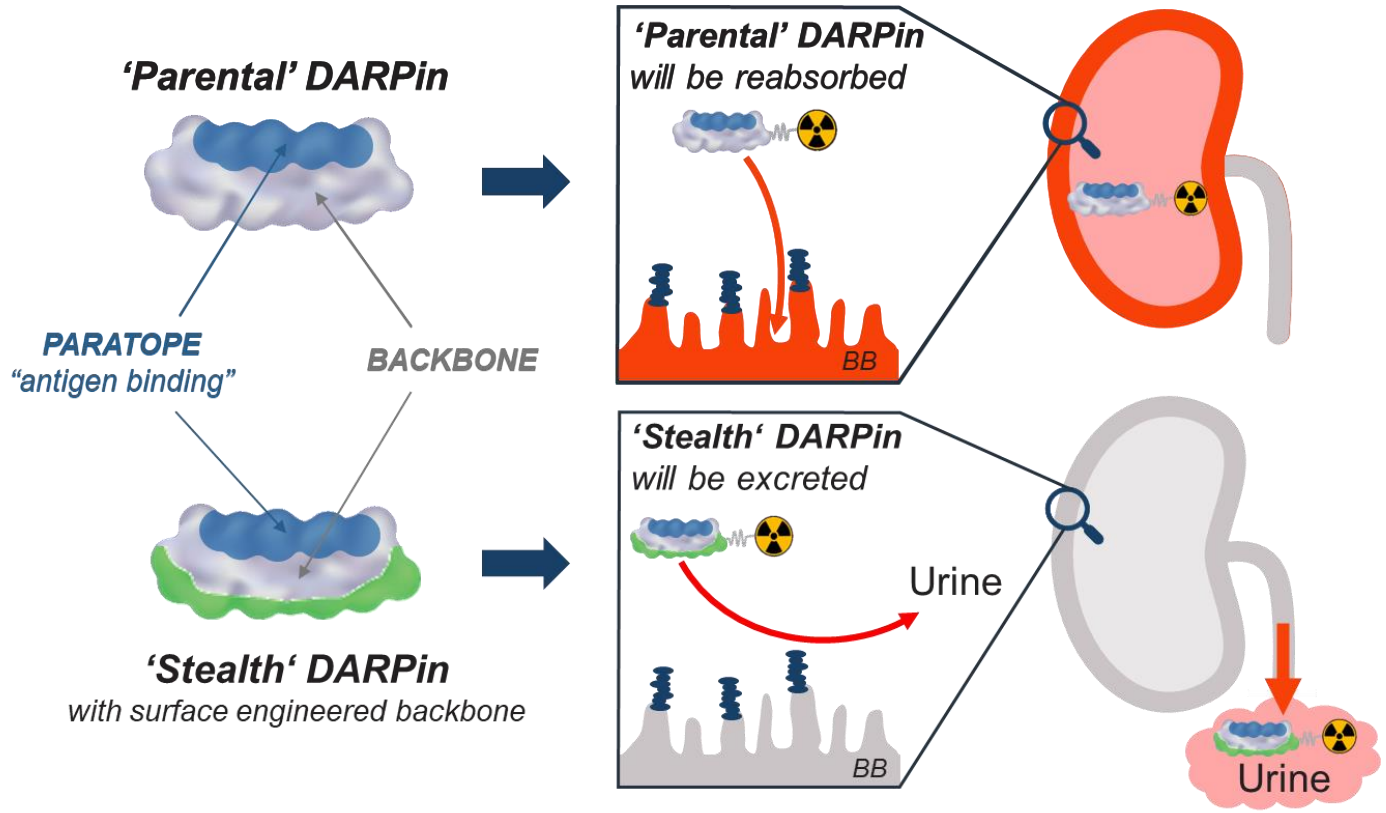




Bragina *et al., J Nuc Med*, 2021

- Strong kidney accumulation of residualizing radionuclides
- Kidney toxicity with therapeutic radionuclides

Surface engineering of DARPins as a strategy to increase renal excretion

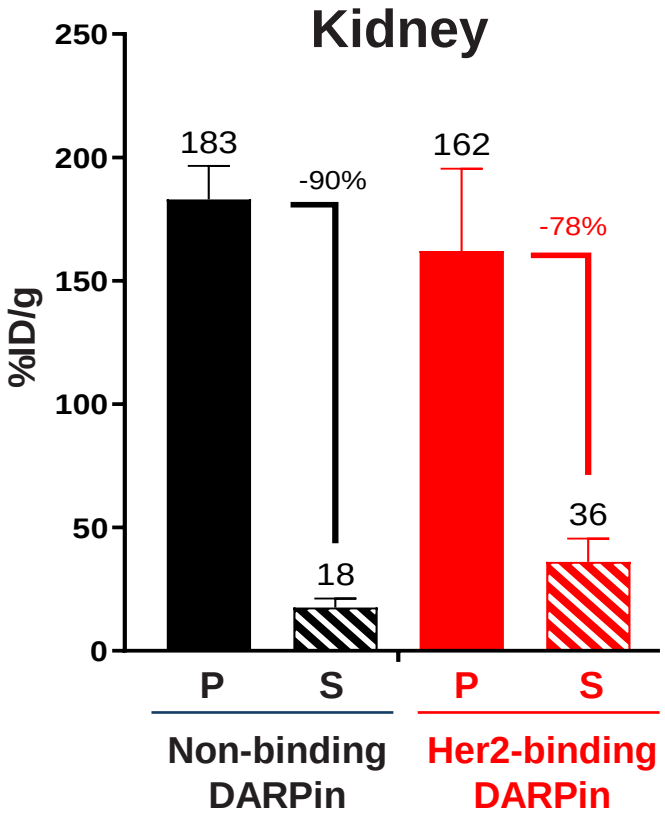


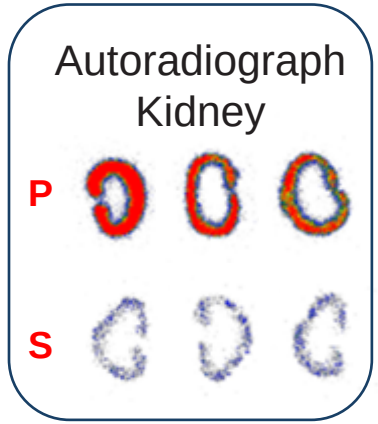
BB: *Brush border of proximal tubular cells in the kidney with megalin/cubilin receptor complex*

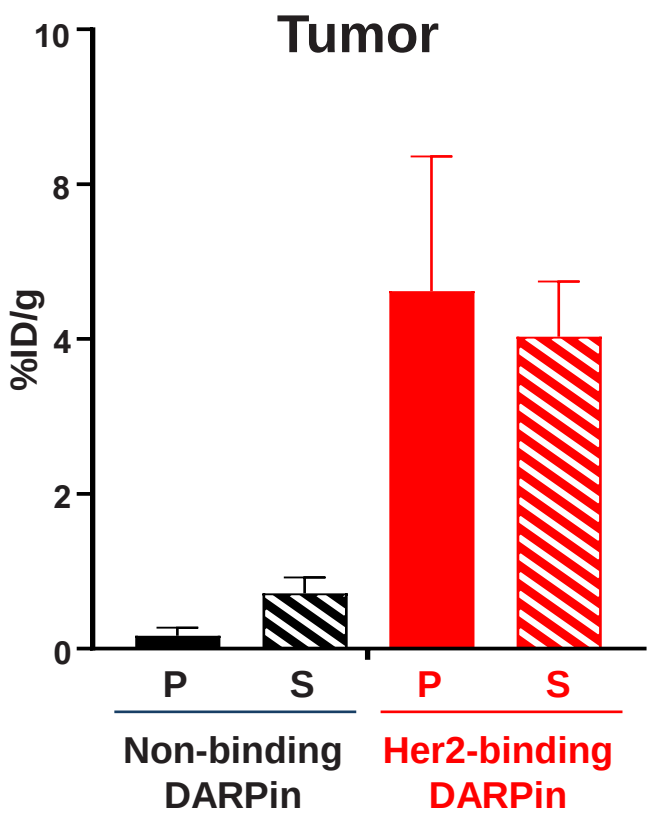
Stealth DARPins Show Strongly Reduced Kidney Accumulation

P: Parental

S: Stealth



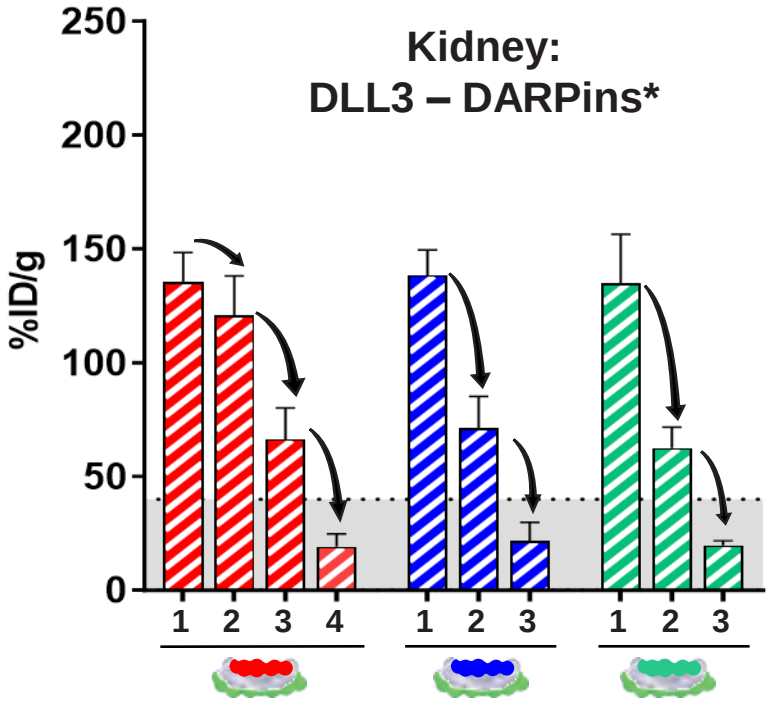




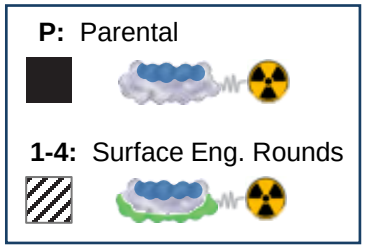
→ **Up to 90% reduction in kidney accumulation with maintained tumor uptake**

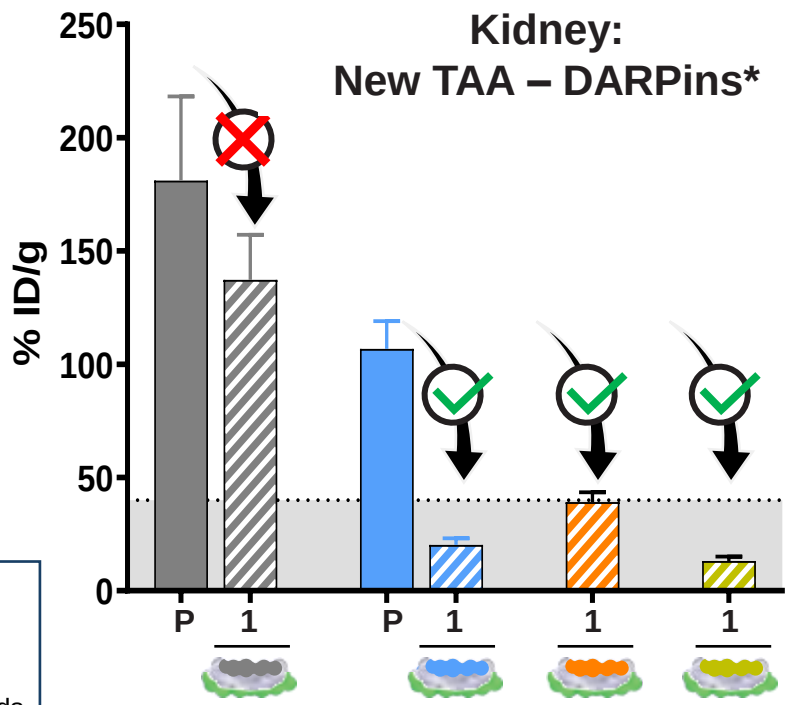
SKOV3 tumor mouse model,
111-In/DTPA labelled DARPin
4 h post injection

Evolution of Surface Engineering for our RDT Engine



Integration of learnings across different TAAs and >140 engineered DARPins





LEARNING PHASE: Iterative rounds of DARPin surface engineering and *in-vivo* testing needed to reach low kidney accumulation

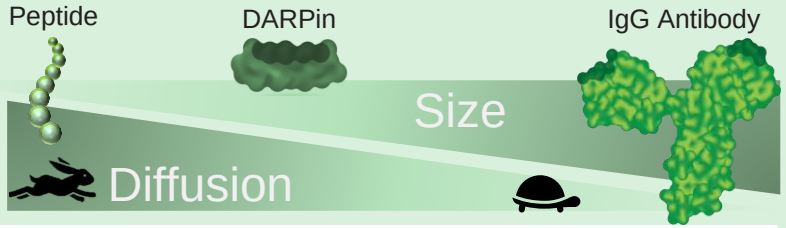
TODAY: Single round of DARPin surface engineering to reach low kidney values for most DARPin binders

MOLECULAR partners

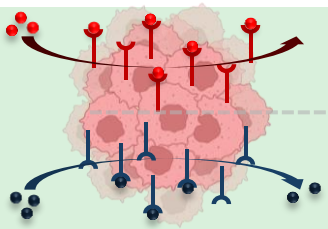
** Kidney value of best surface variant per engineering round displayed in graph: 4h timepoint in wt or tumor bearing mice; DARPins conjugated to different chelators and labelled with different radioisotopes; TAA: tumor-associated antigen*

Multi Parameter Optimization to Improve Tumor Uptake

1. Small vectors extravasate and diffuse faster



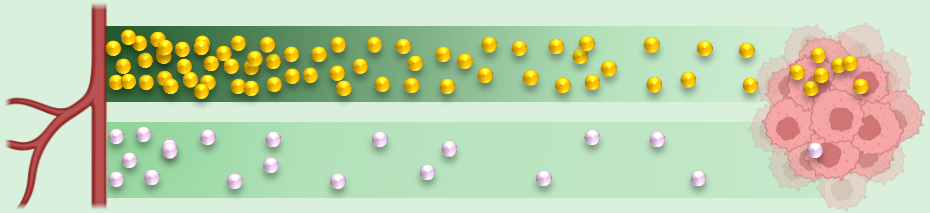
2. High affinity increases tumor uptake of small vectors [1,2] & residence



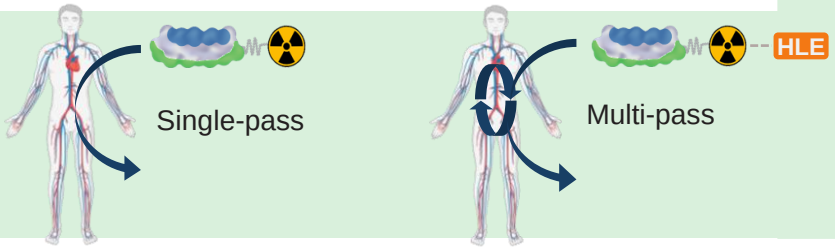
High affinity → slow wash-out & long residence time on tumor

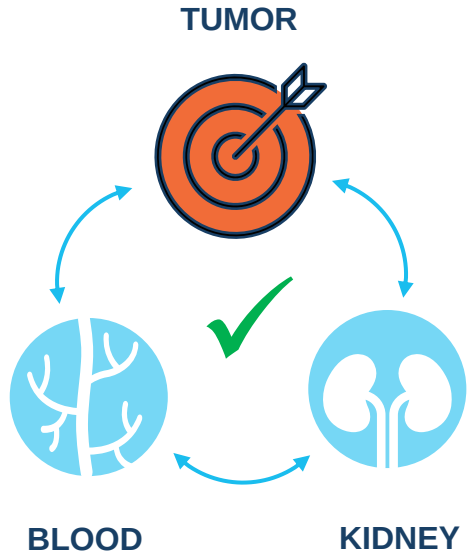
Low affinity → faster wash-out & shorter residence time on tumor

3. Concentration gradient from blood to tumor



4. Extended half-life to maintain concentration gradient





Optimizing Tumor : Kidney : Blood ratio

1. Schmidt & Wittrup, Mol Cancer Ther, 2009
2. Zahnd et al., Cancer Res, 2010

Systemic Half-life Extension (HLE) Increases Tumor Uptake

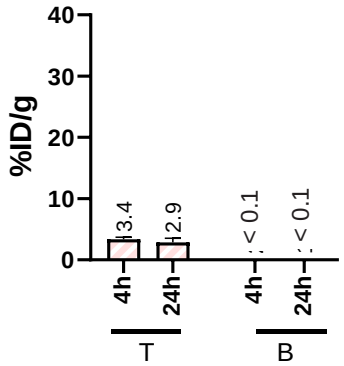
Establishing a HLE toolbox with different “strengths & properties” to tailor to specific needs

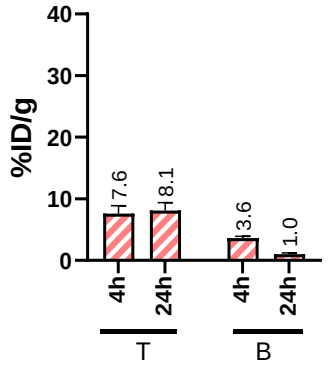
Stealth DARPin (no HLE)
Tumor up to 6% ID/g

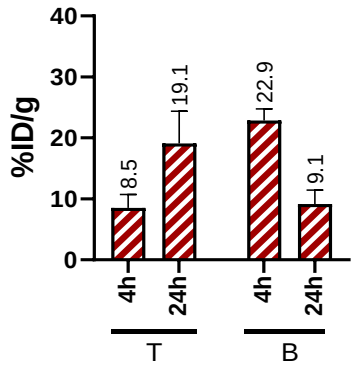
Stealth DARPin + HLEs
Very low blood level increase
Tumor up to 10% ID/g

Stealth DARPin + HLEs
Low to medium blood level increase
Tumor up to 30% ID/g

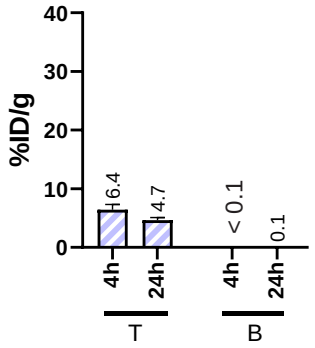
Her2 DARPin

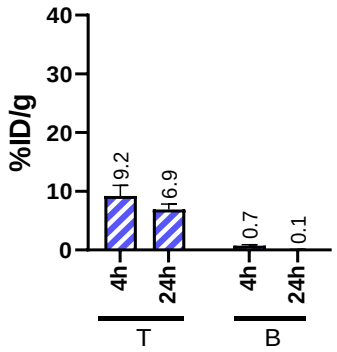


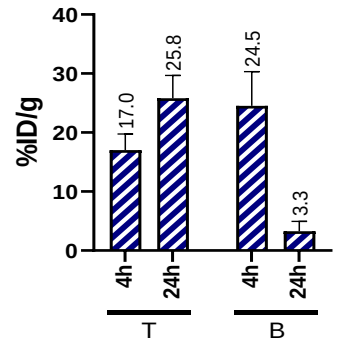


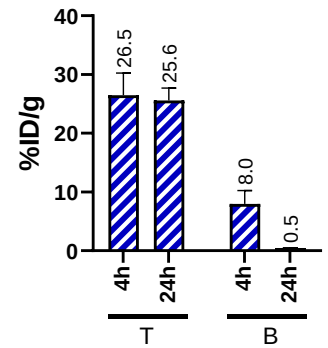


DLL3 DARPin









T: Tumor; **B**: Blood

Her2 and DLL3 DARPins conjugated to different chelators, labelled with different radioisotopes; and tested in different mouse tumor models; αSA: HLE moieties binging to serum albumin

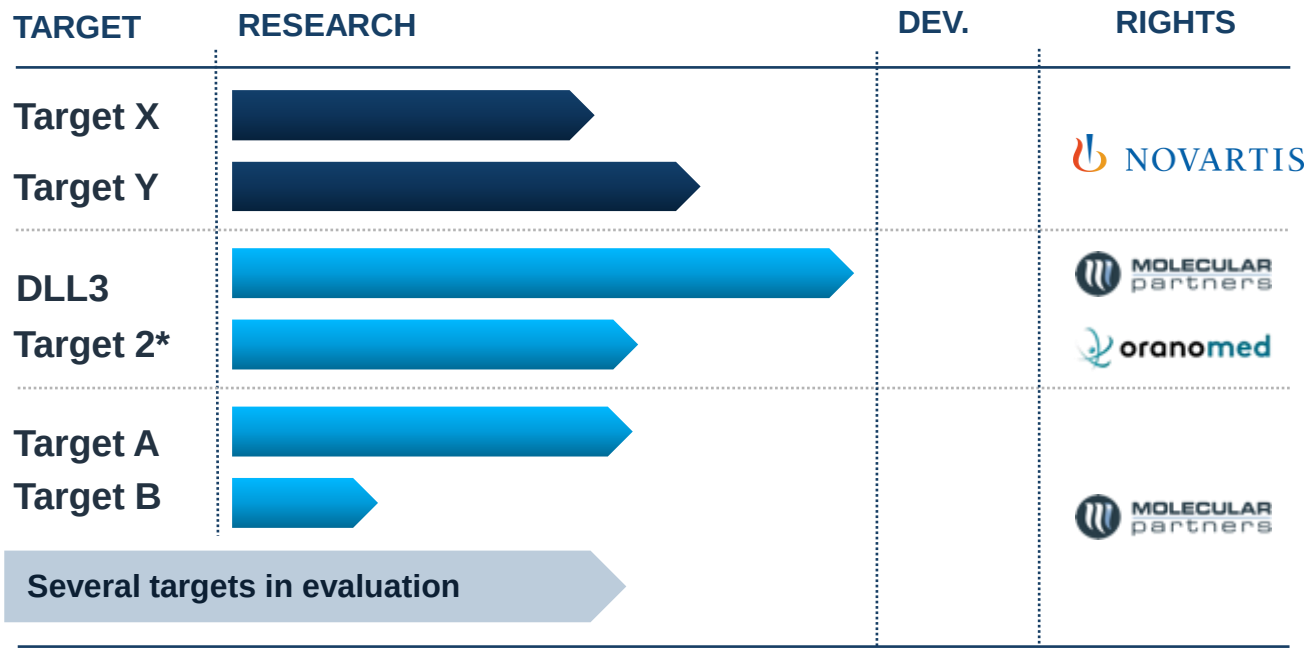
RDT Engine & Pipeline

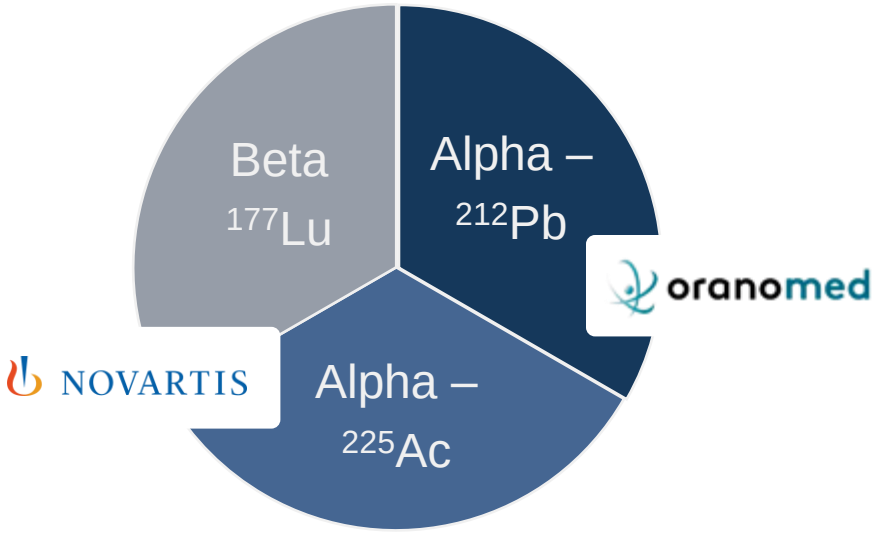
Leverage Radio DARPin Engine & build pipeline

- Tailor candidate properties to specific target needs and radioisotope

Partnering model to join forces with leader in the field

- Cross-pollination of R&D knowledge
- Access radioisotopes & supply chain



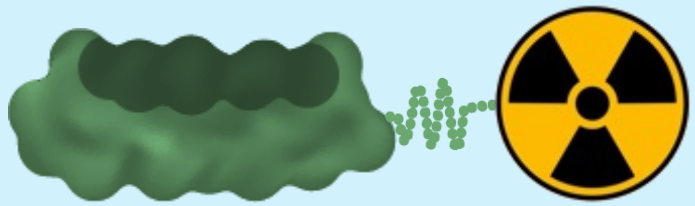


*The co-development agreement with Oranomed includes up to 3 potential oncology targets including DLL3 (Delta-like ligand 3)

Co-development of Radio-DARPin Therapeutics with Orano Med

oranomed
^{212}Pb for Targeted Alpha Therapy

MOLECULAR partners



- Co-development collaboration*, 50:50 cost and profit share
- Access to future manufacturing applying ^{212}Pb
- Up to three tumor antigens incl. DLL3
- Molecular Partners commercialization rights for DLL3

** deal signed January 5, 2024*

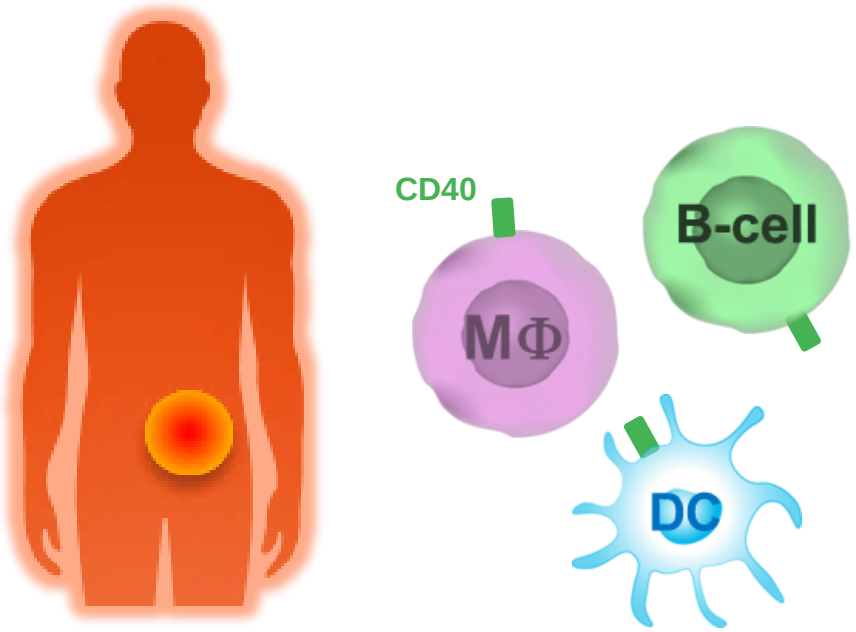
MOLECULAR partners

MP0317

Tumor-localized Immunotherapy

MP0317: Unlocking CD40 Activity Through Local Activation

PROBLEM: Toxicity of CD40 Agonists has so far limited their potential



CD40 agonists can activate **B cells, DCs and MΦ** to enhance the efficacy of anticancer treatment, especially in “cold tumors”

Systemic activation of CD40 via mAbs has been hampered by **significant toxicities**

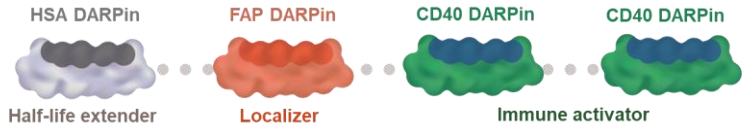
- Limiting potential CD40 agonists to reach therapeutically active doses

DC, dendritic cell; MΦ, macrophage; mAb, monoclonal antibody.

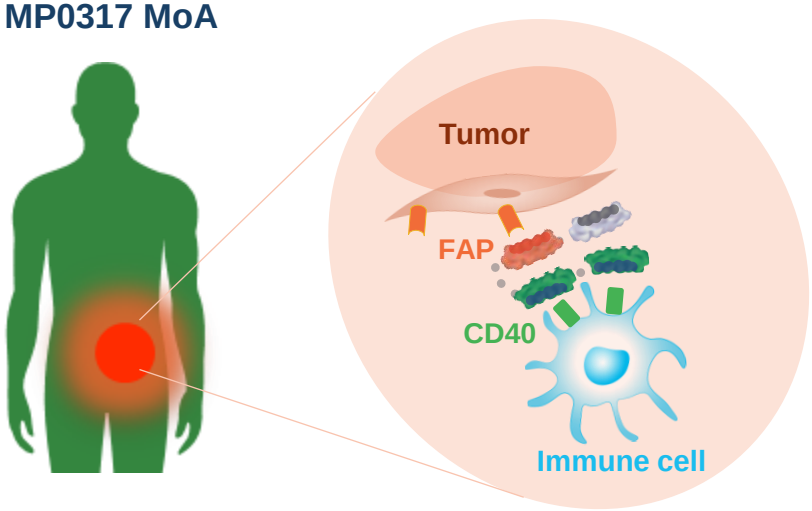
MP0317: Unlocking CD40 Activity Through Local Activation

SOLUTION: MP0317 – FAP-dependent tumor-localized CD40 activation

MP0317 design



MP0317 MoA



FAP is a **validated tumor target**

- Overexpressed in ≥ 28 different cancer types
- Expression not downregulated during disease progression

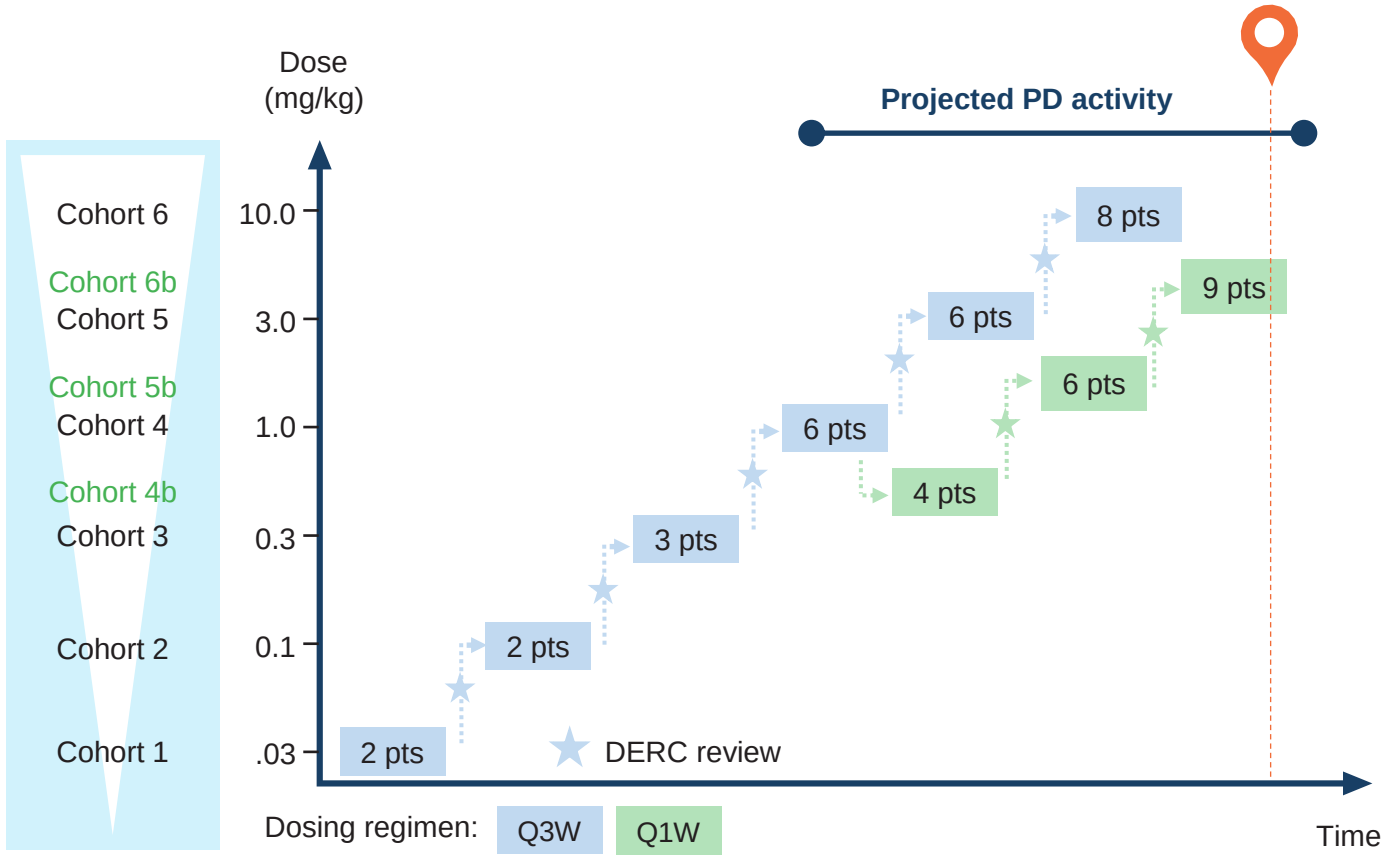
MP0317 designed to

- **Bind tumor-localized FAP** and induce **CD40-mediated activation** of immune cells **in the tumor**
- Overcome systemic toxicity, allowing a wider therapeutic dosing range

FAP, fibroblast activation protein; HSA, human serum albumin.

MP0317 Phase 1 Study Design and Status

First-in-human, multicenter, dose-escalation study in adults with advanced solid tumors



Primary Study Objectives

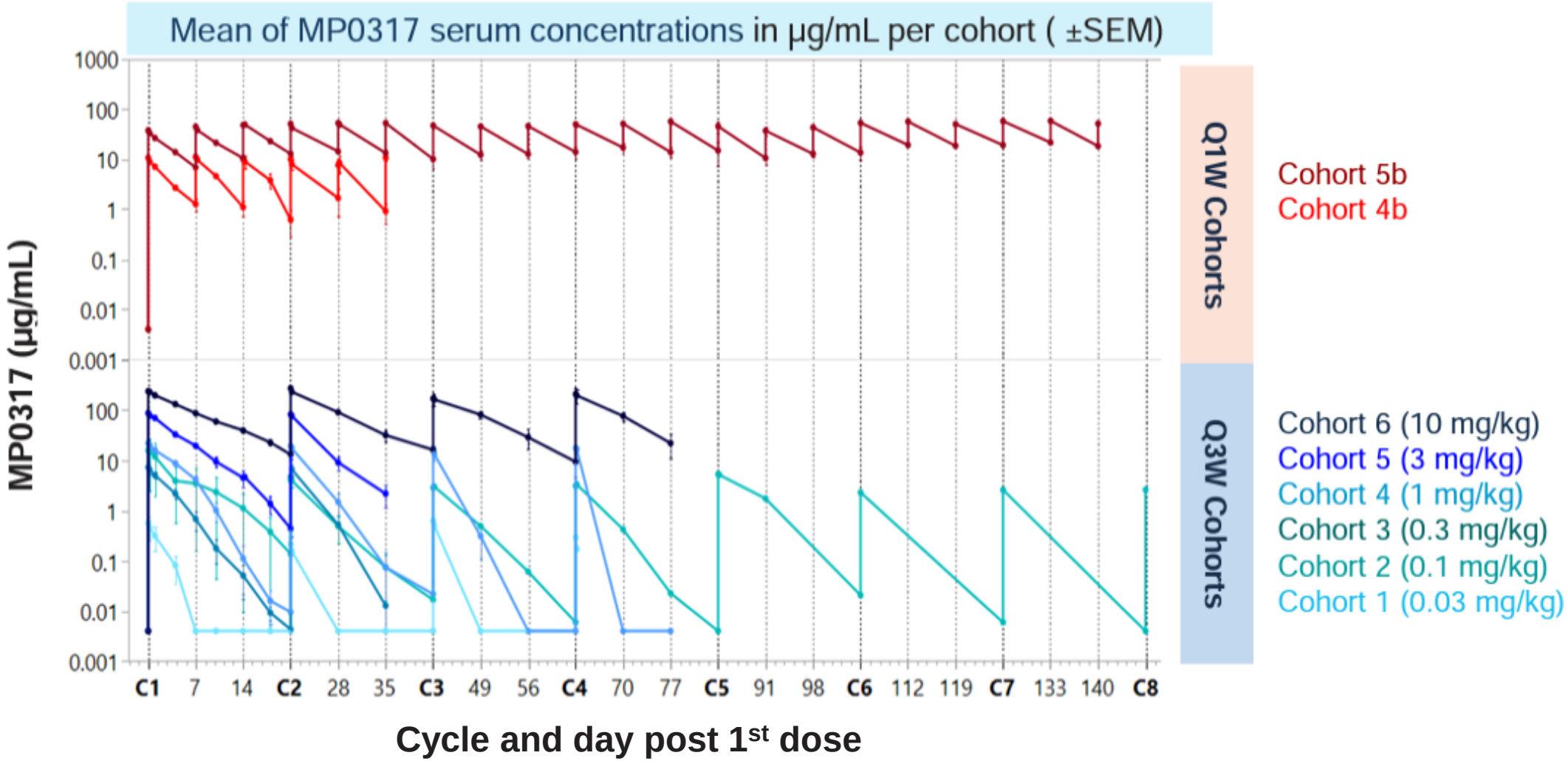
- MP0317 safety and tolerability
- Recommended dose for expansion and combination

Updated Data Presented at SITC 2023[1]

- **Enrollment completed** in dose-escalation part; 46 patients treated
- **Favorable safety profile** up to highest planned dose (10 mg/kg); one DLT
- **Clinical evidence** of tumor-localized CD40 pathway and immune cell activation, leading to **TME remodeling**

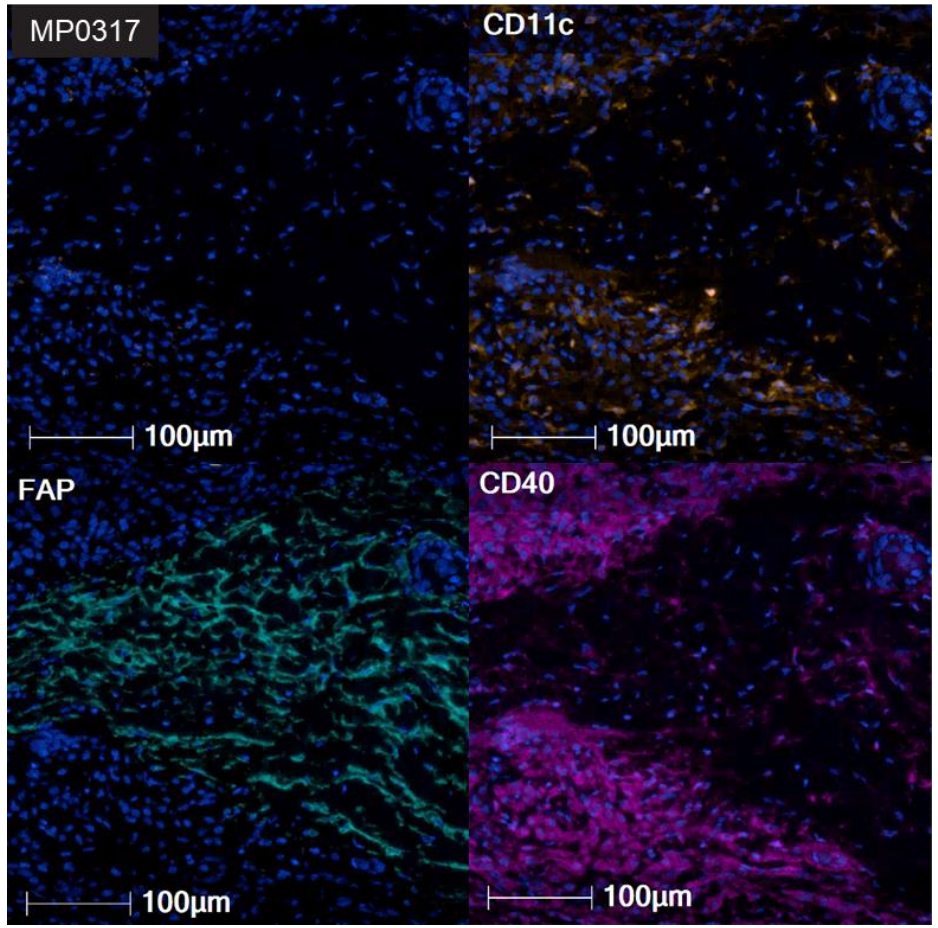
[1]Gomez-Rocca et al. SITC 2023 poster presentation. The study is registered at ClinicalTrials.gov (NCT05098405).
PD, pharmacodynamic; Q1W, weekly dosing; Q3W, every-3-weeks dosing;
DERC, dose escalation review committee; DLT, dose-limiting toxicity; TME, tumor microenvironment.

Exposure and Dosing



MP0317 Co-localizes with FAP and CD40 in Tumors – Concomitant Increase in Intra-tumoral DCs Observed

PRIOR TO TREATMENT

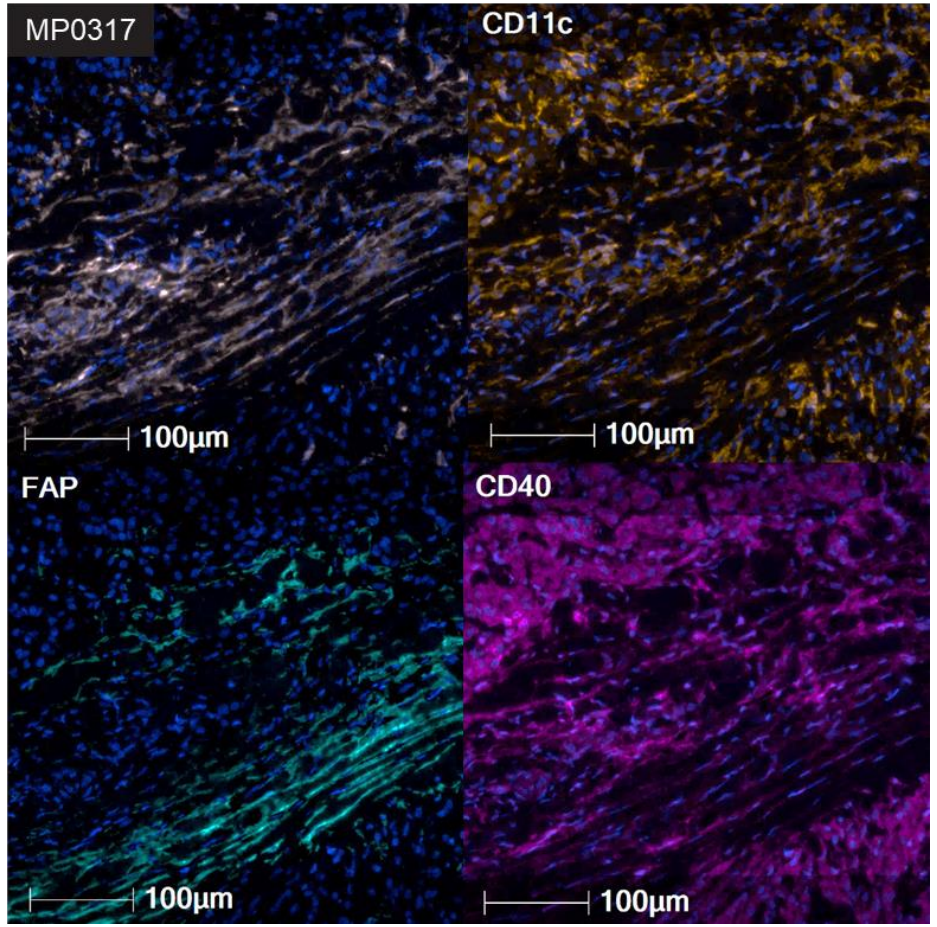


Minimal DC presence in FAP-positive tumor area

DC infiltration

MP0317

CYCLE 2 DAY 8

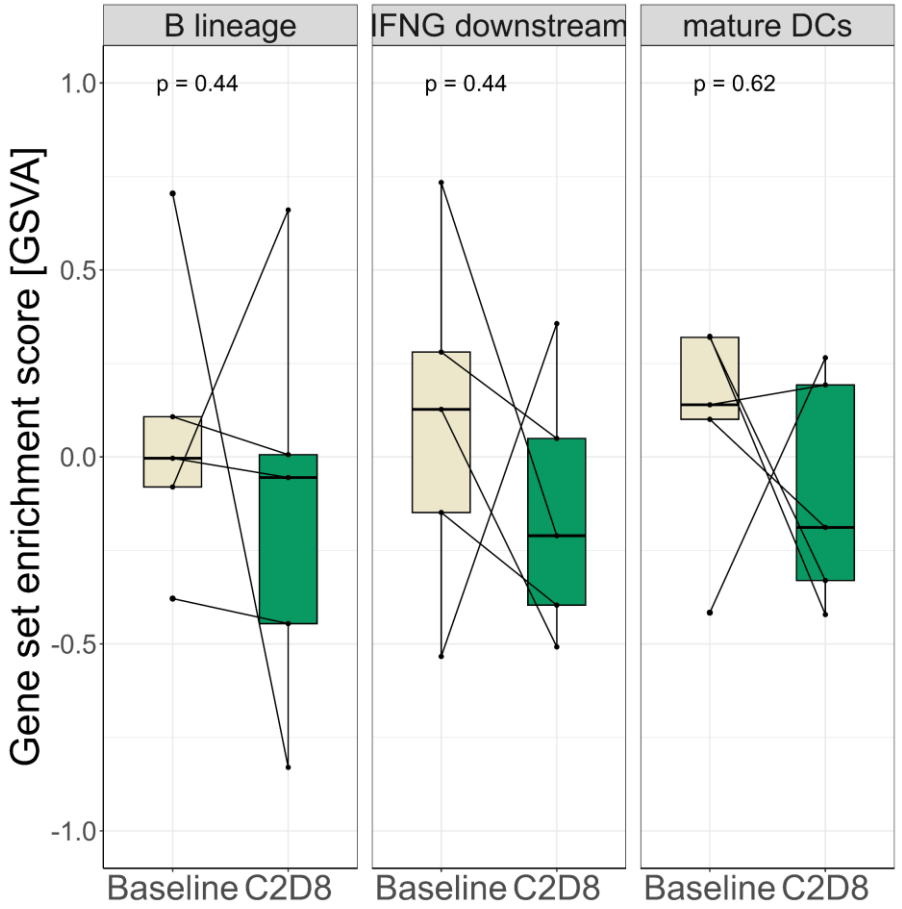


High DC infiltration in FAP-positive tumor area in MP0317 presence

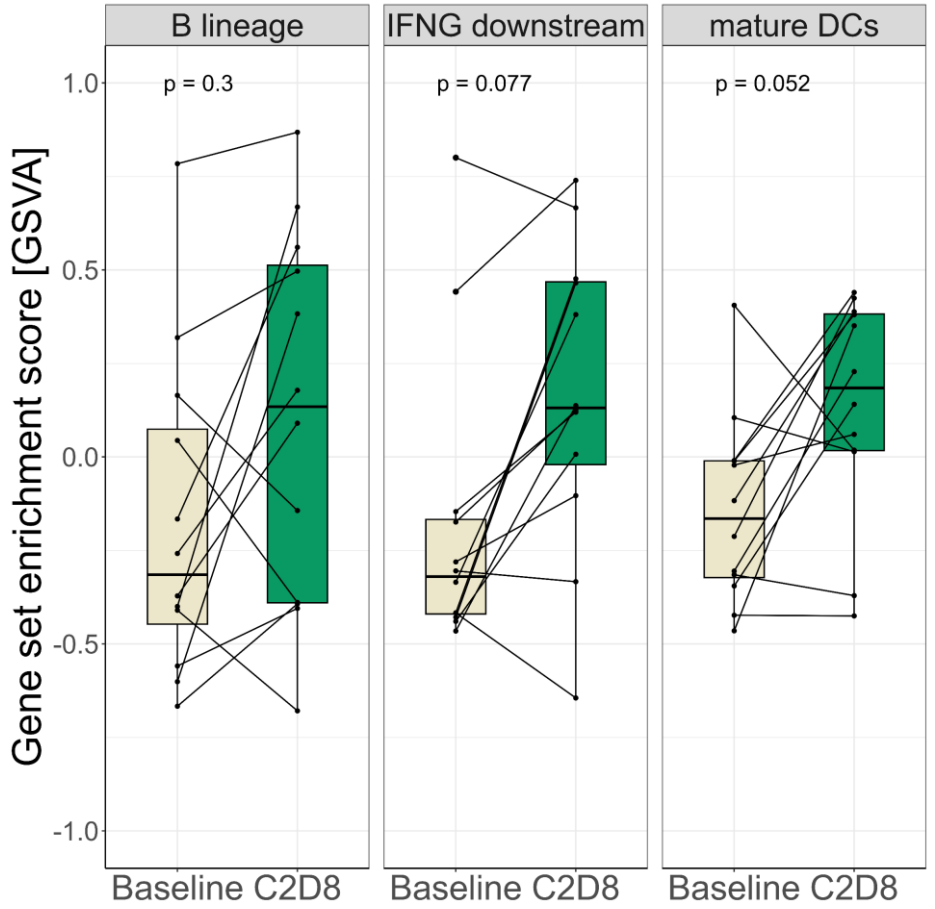
Tumor biopsy of a patient with GIST in Cohort 5b (Q1W); biopsy location: peritoneum.
DC, dendritic cell; FAP, fibroblast activation protein; GIST, gastrointestinal stromal tumor.

Increased immune cell infiltration, DC maturation and IFNγ production observed in tumors post MP0317 treatment

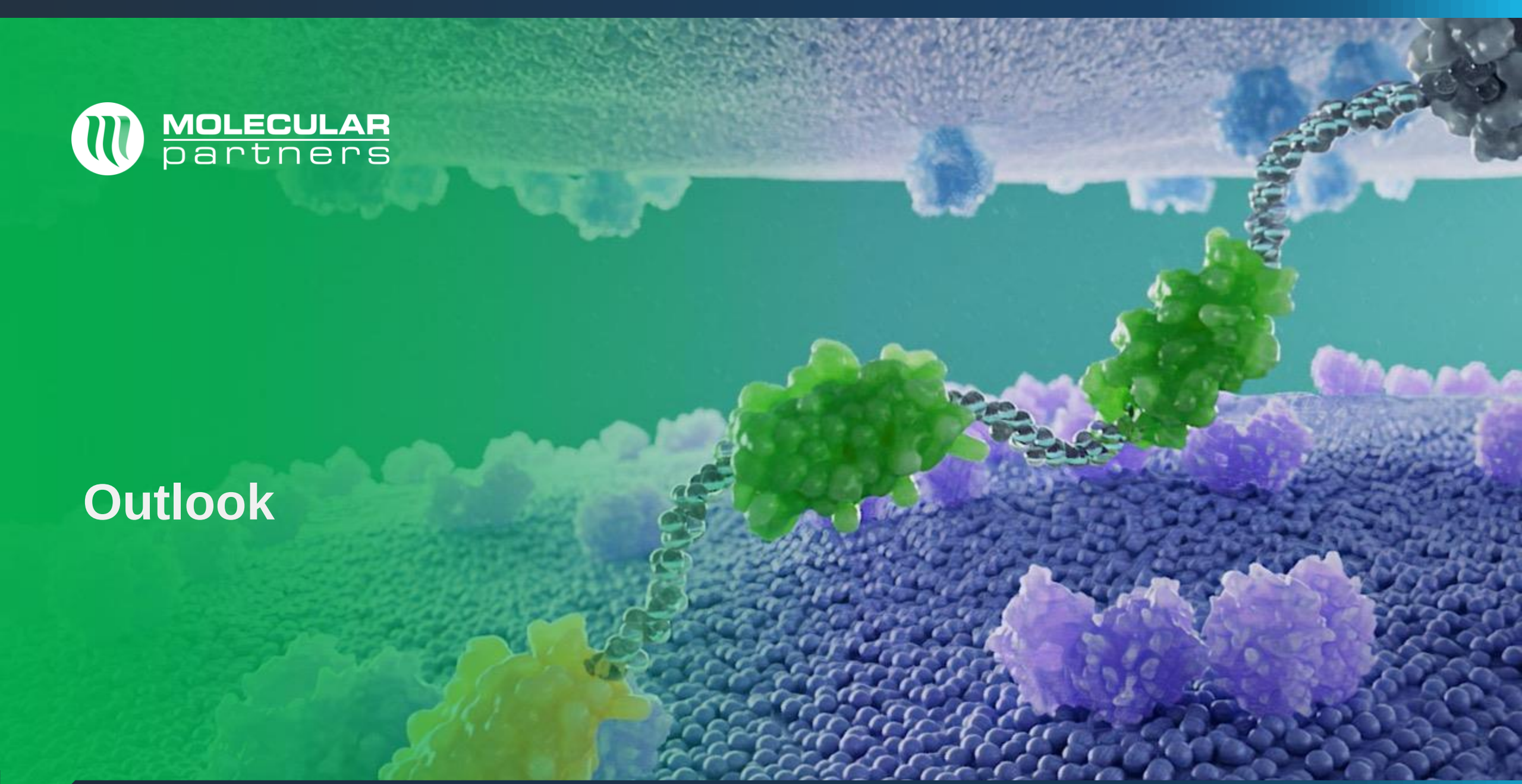
MP0317 low* doses or not detected in tumor (n=5)



MP0317 higher doses and detected in tumor (n=12)**



Treated patients up to Cohort 6 with evaluable paired biopsies for transcriptomics (n=17).
*Low doses=≤0.1mg/kg; **Higher doses=≥0.3mg/kg. Statistical analysis was done using a signed rank Wilcoxon test.
Gomez-Roca et al, SITC 2023 poster presentation



Outlook

Outlook and Upcoming Milestones

MP0533	• Data from projected therapeutically active doses in H1 2024 • Plans for future clinical development strategy • Clinical expansion in Europe and preparation of potential US IND application
Switch-DARPin	• Data presentation on cKIT x CD16a x CD47 program in H1 2024 • Initiate IND-enabling studies in H2 2024 • Leverage Switch-DARPin platform for next-generation immune cell engagers
Radio-DARPin Therapy	• DLL3 data and lead RDT candidate selection in H1 2024 to advance into IND-enabling studies with FIH in 2025 • Nominate additional RDT targets and pipeline candidates in H1 • Broaden clinical and supply collaborations with radionuclide companies
MP0317	• Full Phase 1 proof-of-mechanism and safety data in H1 2024 • Partnering for clinical development in combination settings

CHF ~187 million cash* (incl. short-term time deposits) ensures **funding well into 2026**

*As of December 31, 2023, unaudited financials.
AML, acute myeloid leukemia; IND, investigational new drug;
R/R, relapsed/refractory; ASH, American Society of Hematology.

Thank You