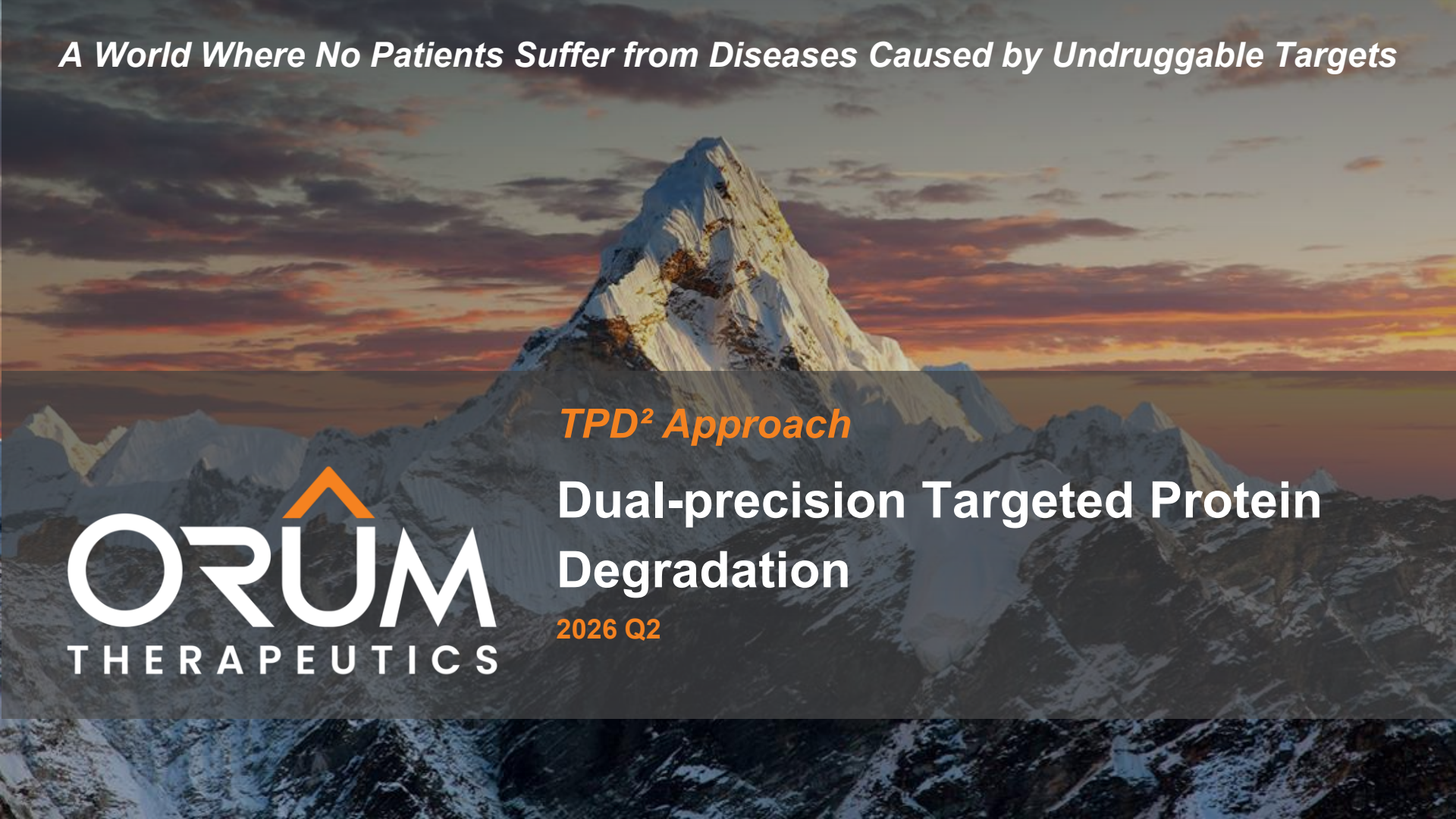
A World Where No Patients Suffer from Diseases Caused by Undruggable Targets



ORUM THERAPEUTICS

TPD² Approach

Dual-precision Targeted Protein Degradation

2026 Q2

Disclaimer

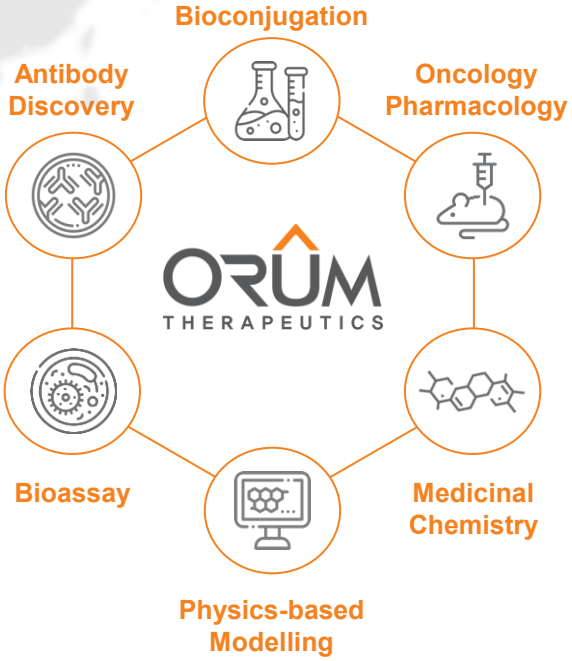
Forward-looking Statements

This presentation contains forward-looking statements that are based on the current expectations and beliefs of Orum Therapeutics, Inc. (“Orum”). Statements in this presentation regarding matters that are not historical facts, including, but not limited to, statements relating to the potential range of usable antibodies, targetable proteins, degrader payloads, and indications, and the potential market for Orum’s products and technologies; the successful development of a multiple-platform portfolio; the modularity of the PROTAb platform; the selection of targets; the translation of preclinical studies to successful clinical trials; the commercial and therapeutic potential of Orum’s technologies and product candidates; Orum’s expectations with respect to its pipeline, including, but not limited to, timelines, and the potential for the filing of Investigational New Drug applications; the potential to receive milestone and royalty payments from technology sales and licensing transactions and to enter into additional transactions in the future; and the adequacy of past, and the potential for future, fundraising are forward-looking statements. These forward-looking statements are based on management’s expectations and assumptions as of the date of this presentation and are subject to numerous risks and uncertainties, which could cause actual results to differ materially from those expressed or implied by such statements. These risks and uncertainties include, without limitation, the uncertainty of success in research and development activities; risks related to clinical trials, including potential delays, safety issues, or negative results; competition from alternative therapies; reliance on third-party decisions in regard to the technologies we license to them; the risk that we may not be able to maintain and enforce our intellectual property; the risk that our product candidates may not be successfully commercialized or adopted; the impact of any pandemic or other global event; the availability of financing; and risks related to the recruitment and retention of key employees, fluctuating markets and economic conditions, health care reform, prices, and reimbursement rates. The forward-looking statements in this presentation speak only as of the date of this presentation, and we undertake no obligation to update or revise any of the statements. We caution investors not to place considerable reliance on the forward-looking statements contained in this presentation. The contents of this presentation are the proprietary information of Orum and may not be reproduced without its prior consent.

Orum is a Leader in Degrader Antibody Conjugates

Daejeon, KR

Boston, US



First company to develop a clinical stage **DAC**

Two FDA IND packages:
ORM-5029, ORM-6151

Innovative and **proprietary** platform technologies

Two validating transactions:
BMS and Vertex

Upcoming key events:
ORM-1153 IND/CTA filing expected in Q4 2026

Our team:
56 employees
>60% R&D | Daejeon & Boston

Experienced Team to Drive Orum Programs and Platforms Forward

From discovery to clinical execution

Founder and CEO
SJ, Sung Joo Lee

sanofi
LG Chem

Chief Scientific Officer
Chad May

serotiny
MAVERICK THERAPEUTICS
Pfizer
ImClone Systems Incorporated

Chief Medical Officer
Olaf Christensen

Bristol Myers Squibb
BAYER
EMD SERONO

Head of Platform Technology
Dorin Toader

Mersana THERAPEUTICS
AstraZeneca
MedImmune

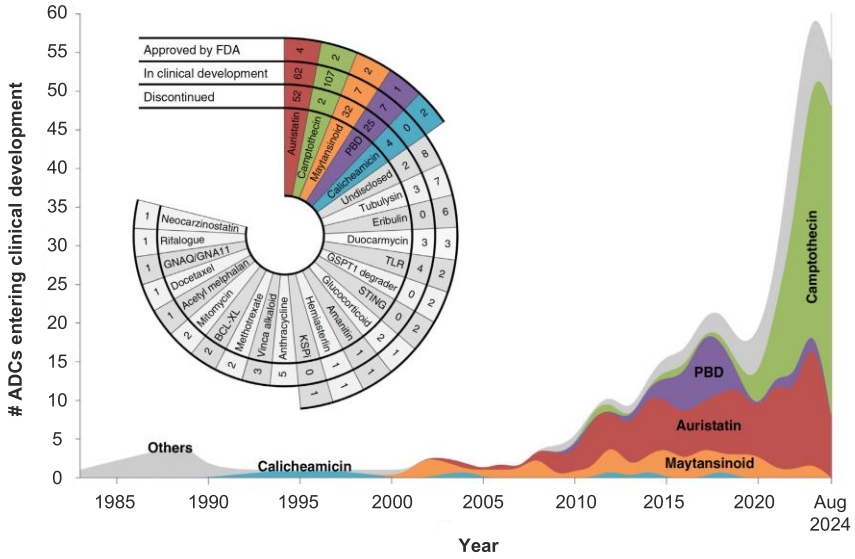
Head of BD
Greg Dwyer

Bristol Myers Squibb
immunogen
magenta THERAPEUTICS
DRG

Robust CAGR Expected in ADC Market Amid Increasing Demand for Novel MoA Payload

Market expected to reach $28B by 2028 (23% CAGR), with ADCs accounting for 30% of global biopharma licensing agreements

Success of maytansinoids, auristatins, and campothecin payload classes has ushered in a golden era of ADCs, but has also intensified competition as dearth of payload MOA remains



Raffaele Colombo et al. Cancer Discovery (2024)

ADC payload types

Tubulin inhibitors

e.g. auristatin, maytansinoid

MoA

Inhibit microtubule polymerization, causing mitotic arrest

Known Limitation

Peripheral neuropathy, Efflux resistance

DNA damaging agents

e.g. calicheamicin, PBD

MoA

Induce DNA strand crosslinking or cleavage, halting replication

Known Limitation

Severe toxicity, Poor stability

Topoisomerase1

e.g. DXd, SN-38

MoA

Inhibit Topo1, leading to accumulation of DNA breaks during replication

Known Limitation

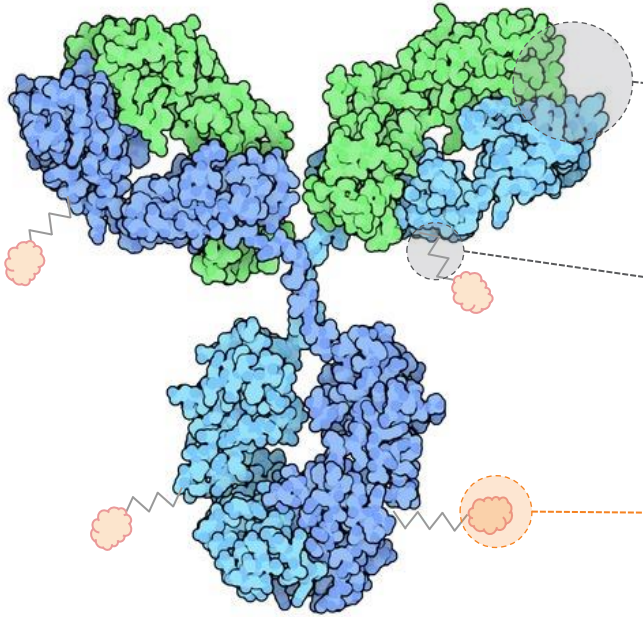
Off-target toxicity from free payload, GI toxicity, Interstitial lung disease

Next-generation payloads are emerging

Novel payloads with improved stability, lower toxicity, new MoAs and the ability to overcome resistance are critical to advance next wave of ADC innovation

What is a Degrader Antibody Conjugate (DAC)?

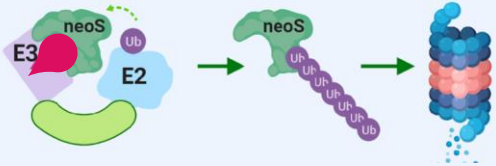
Next generation antibody drug conjugate (ADC) with potential for broad intracellular target diversity



	Conventional ADC	VS	Orum TPD² DAC
Antibody	Confers selectivity and binding against cell surface antigen, allowing **targeted delivery to target cell**		
Linker	Connects payloads to antibody with **stability during circulation**, and **effective release of payload** in presence of cleavage triggers		
Payload	• Cytotoxic payload • Limited MoA		• Targeted protein degrader • Differentiated MoA

Differentiated Approach of Delivering Protein Degraders with Precision

Targeted Protein Degraders



Molecular glues

Monovalent protein degraders engaging E3 ligase and neosubstrate

Heterobifunctional degraders

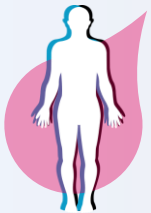
Modular small molecule comprised of target protein and E3 ligase binder

Current Approaches in Protein Degradation

All cells

Penetrate cells indiscriminately, including healthy cells.

Increased risk of off-target toxicity



Tissue-specific

Tissue specific E3 ligases

Still cannot distinguish healthy and disease-causing cells

Orum's Next Gen Approach

Cell-specific

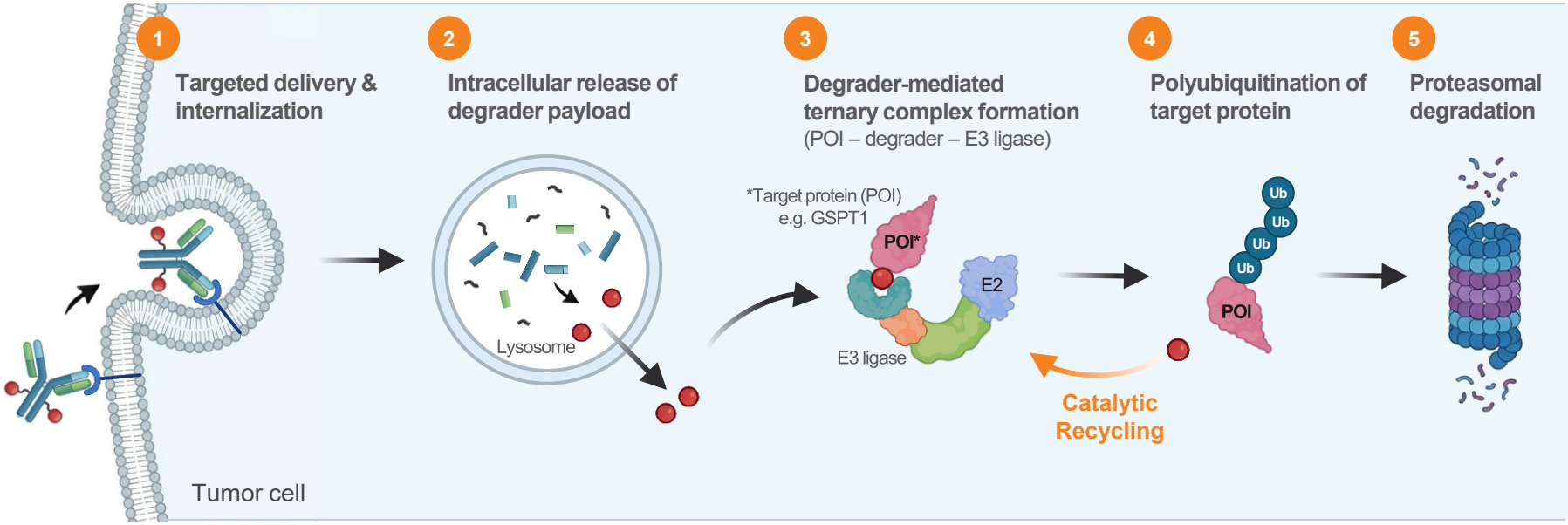
Cell-targeting degraders **delivered specifically to disease-causing cells** (eg. tumor cells)

More precision

Images: flaticon.com

TPD^2 Dual Precision Mechanism of Action: Targeting for Enhanced Safety & Efficacy

Selective tumor targeting combined with targeted protein degradation drives tumor cell death

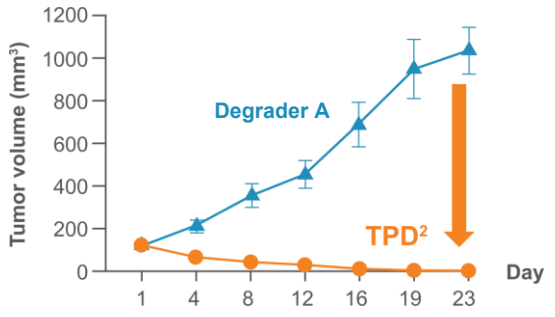


- *Potentially Improved Efficacy*
- *Potential Improvement in PK*
- *Potentially Improved Safety & Therapeutic Index*

Orum's TPD² Presents Potential Improved Efficacy, PK and Safety in Pre-Clinical Studies

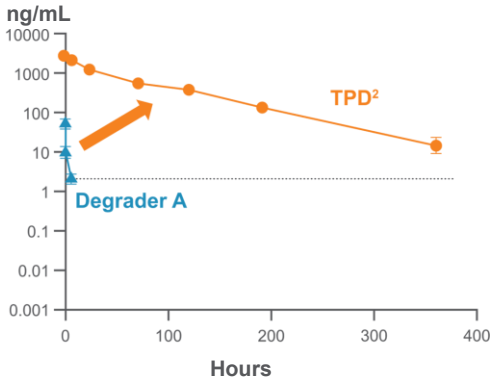
In pre-clinical studies, Orum's TPD² approach presented the potential for each of the following:

A Improved Efficacy
Strong activity in vitro and in vivo



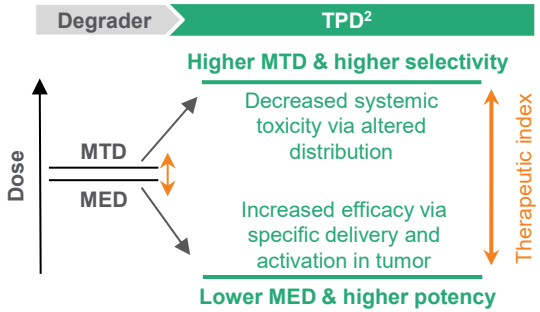
TPD² shows greater in vivo tumor suppression

B Significant Improvement in PK
Better and optimal pharmacokinetics



Exposure of degraders increased by >100x

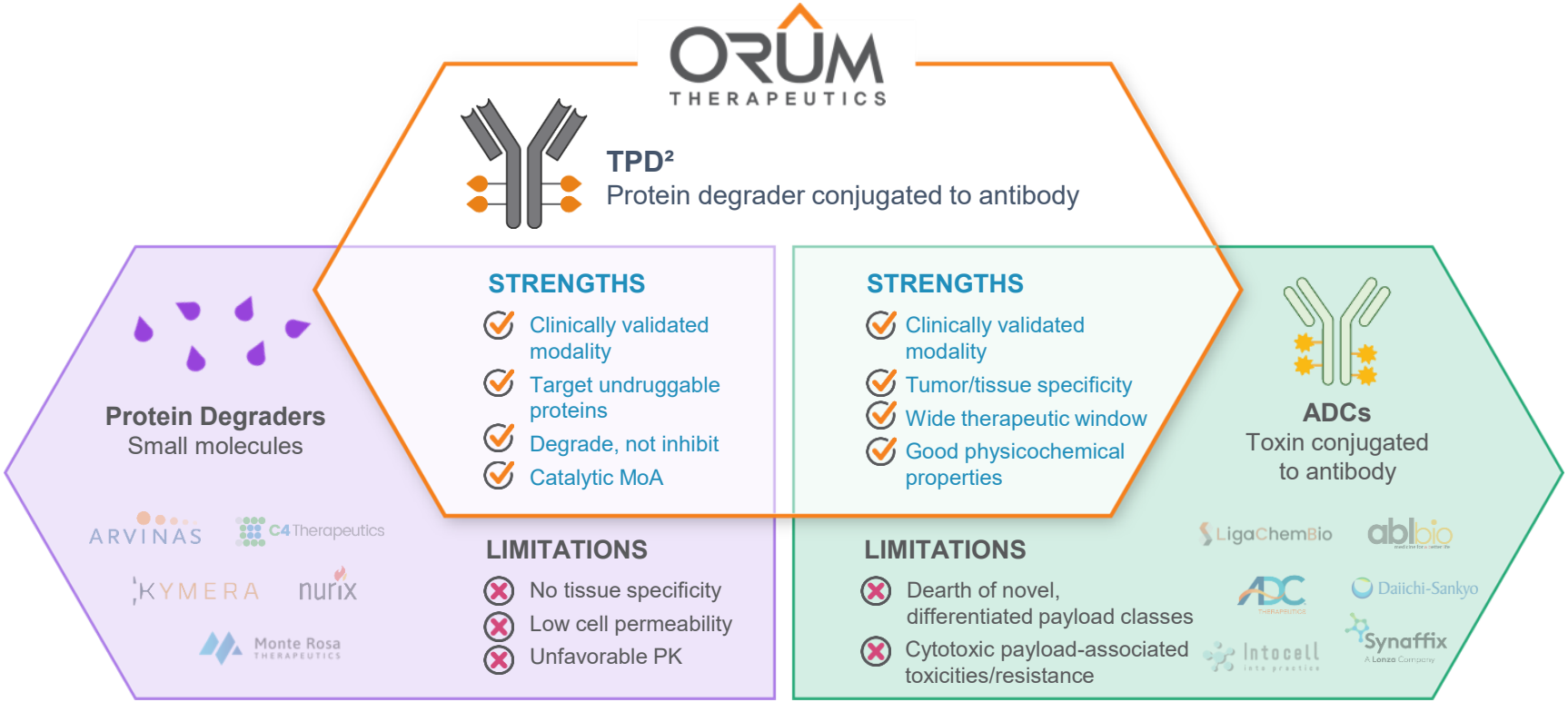
C Improved Therapeutic Index (TI)
Better selectivity and safety profile



Enables selective tumor activation, improving the TI by lowering MED and raising MTD

Dual-Precision Approach Uniquely Positions Orum Amongst Competitors

TPD² uniquely combines the specificity of antibody-drug conjugation with the robust efficacy potential of targeted protein degradation



Two Deals Completed to Highlight the Value of TPD^2 Technology

Strategic partnerships enabling clinical development and broader application of TPD^2

Asset Sale — Bristol Myers Squibb™

Partner overview

- Global biopharma leader in oncology, hematology, and molecular glue degraders

Deal summary

- Acquired ORM-6151 (BMS-986497)
- **$100M** upfront; up to **$80M** in milestones
- BMS assumed full responsibility for all aspects of the program

Program highlights

- Phase 1 commenced in May 2024 (NCT06419634)
- Target Indications: AML and MDS

Platform Deal — VERTEX

Partner overview

- Global biotech leader with approved medicines for genetic diseases and a robust pipeline across modalities targeting serious diseases grounded in human biology

Deal overview

- Platform deal focused on TPD^2 DACs as targeted conditioning agents for use with gene editing
- License & option agreement: up to **3 target**-exclusive licenses

Deal economics

- **$15M** upfront payment
- Up to **$310M** per target in development and commercialization
- Tiered royalties on future net sales

Multiplatform Pipeline to Address High Unmet Medical Need

Dual-precision targeted protein degradation (TPD^2) approach technologies

Platform	Program	Target	Indication	Discovery / IND-enabling / Phase 1
TPD^2 GSPT1	ORM-1153	CD123	AML, Heme-onc	
	ORM-1023	Undisclosed	SCLC, NET	
	BMS-986497 (ORM-6151)	CD33	AML, MDS	BMS Acquired
TPD^2 New Payloads	Multiple	Undisclosed	Solid Tumors	
	Multiple	Undisclosed	Immunology & Inflammation	
TPD^2 Undisclosed	Vertex Partnership	Undisclosed	Pre-conditioning	VERTEX Up to 3 exclusive licenses

SCLC: Small Cell Lung Cancer. NET: Neuroendocrine Tumor. AML: Acute Myeloid Leukemia. MDS: Myelodysplastic Syndromes

Adaptor linker platform

Platform	Program	Description
TPD^2 PROTAb	E3 Ligase-DAC Adaptor	Adaptor linker platform enabling E3 ligase-based DACs

TPD² GSPT1 Platform

Antibody-enabled Molecular Glues

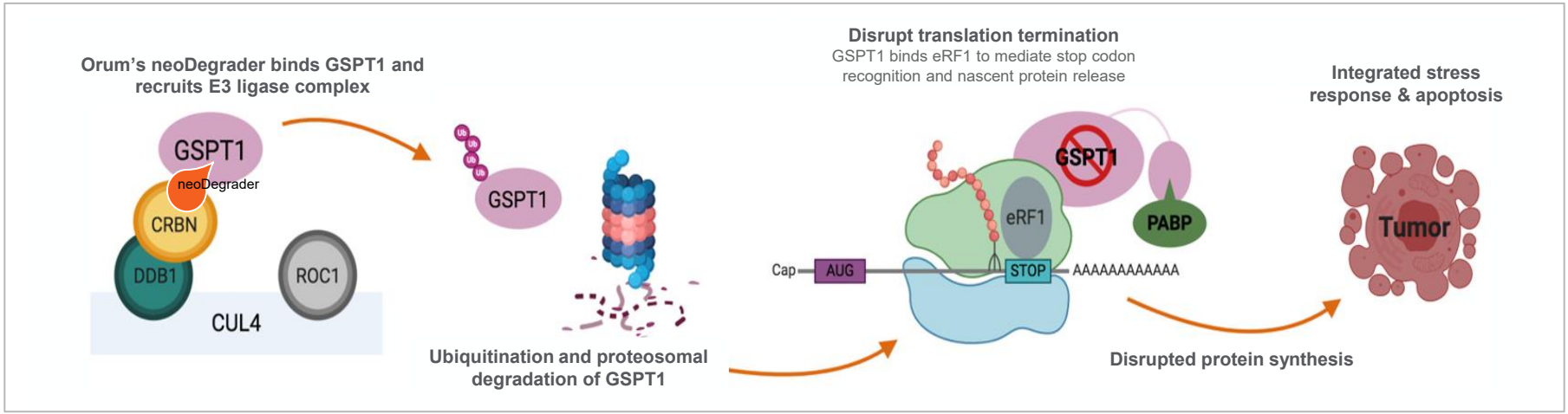
- ORM-1153
- ORM-1023

GSPT1 Degradation Leads to Apoptosis

GSPT1 is a housekeeping protein involved in translation termination

Degradation of GSPT1 → Integrated stress response → Prolonged response → Caspase activation and apoptosis

GSPT1 degrader cell-killing MOA

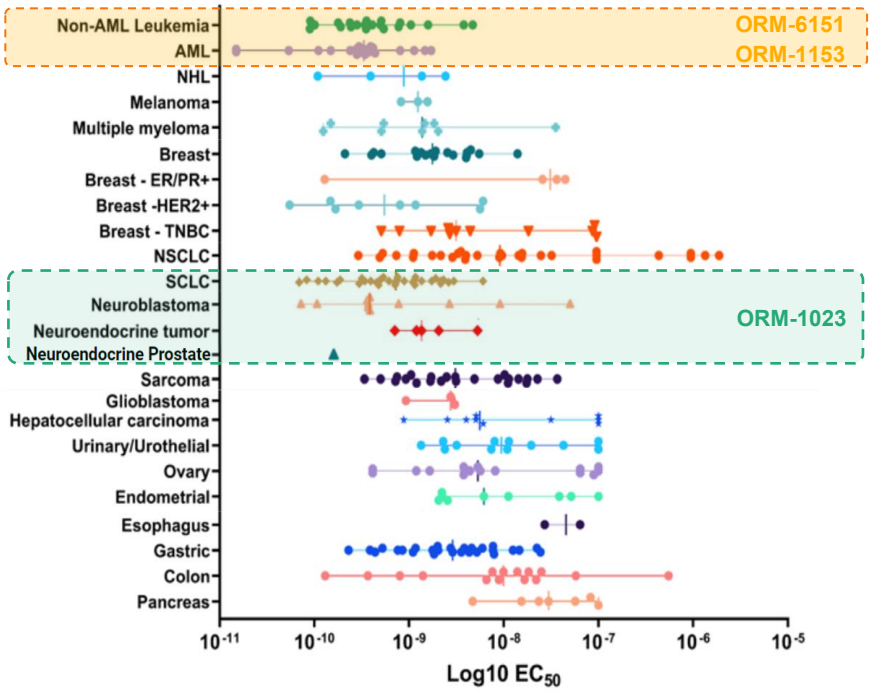


TPD² GSPT1 platform delivers a proprietary GSPT1 degrader (SMol006) specifically to tumor cells, leading to better cell-killing efficacy (>1000-fold) and safety (>10-fold) than CC-90009

* (EHA 2020 #EP598)

GSPT1 Degraders Are Highly Potent Against a Broad Set of Tumor Types

In vitro screen of GSPT1 degrader against 250+ cell lines across 20+ tumor types



Note: Each dot represents a cell line.

Orum's risk-mitigated selection approach

In vitro screening results

Focus on indications with a median *in vitro* potency at or below 1nM

Select targets with clinical validation within indications

Program nomination

GSPT1 degraders show lineage dependency: Activity can be enhanced by antibody conjugation

Current programs yielded from screen

ORUM THERAPEUTICS

ORM-6151/BMS-986497 (CD33)
ORM-1153 (CD123)
ORM-1023 (undisclosed)

ORM-1153: CD123-Targeting GSPT1 DAC

Combining CD123-targeted delivery with GSPT1 degradation

Antigen target:

CD123

Highly expressed in AML
>97% at diagnosis | >98% at relapse

Indication:

Acute myeloid leukemia (AML)

Potential expansion into additional CD123-positive hematologic malignancies

MoA:

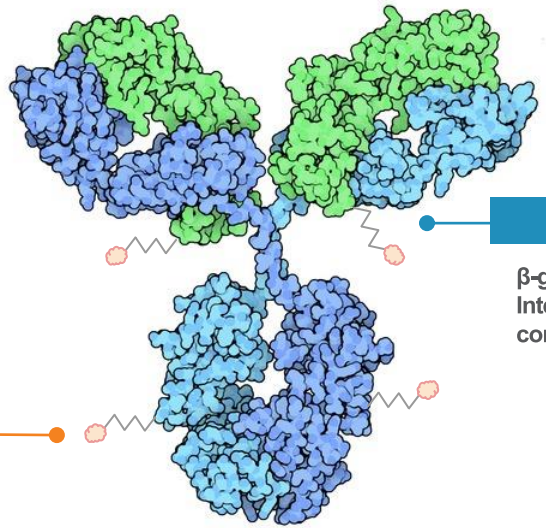
CD123-directed GSPT1 protein degradation

Status:

IND/CTA filing expected in Q4 2026

Antibody

Orum's proprietary Fc-silenced CD123 antibody with enhanced internalization and cross-reactivity to cynomolgus monkey



Linker

β-glucuronide linker. Interchain cysteines for conjugation with DAR ~4[1]

Payload

SMol006: proprietary CRBN-based molecular glue degrader targeting GSPT1. Released in the cytosol with limited intrinsic cell permeability

1. Cartoon location of conjugation only for ease of representation

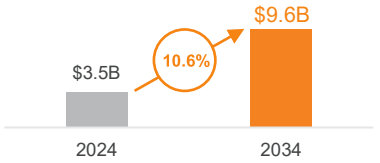
TPD² GSPT1: Opportunity in Acute Myeloid Leukemia (AML)

Unmet Needs

Improving efficacy for R/R patients	Significantly lower efficacy in relapsed/refractory patients than in newly diagnosed AML patients (mOS ~11 mo.)
Demand for Durable & Less Toxic Therapies	Despite initial CR with venetoclax+HMA, long-term remission is difficult to achieve in unfit patients
No SoC for TP53-mutated AML	TP53m patients, accounting for ~5-10% of all cases, show poor response to chemo and face high-risk prognosis, with no established SoC

AML Market Size (global) & Patient Age Trends

Market projected to grow from $3.5B in '24 to $9.6B by '34 (10.6% CAGR), with North America making up 37% in '24

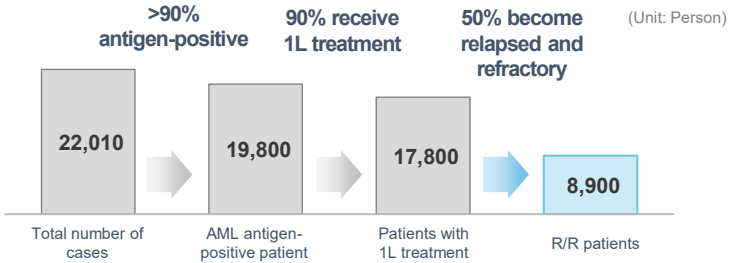


Source : Towards Healthcare; American Cancer Society

AML is uncommon in people under the age of 45

68 Median diagnosis age

AML Patient Segmentation (as of 2025; U.S.)



Standard of Care

Front Line

Unfit: Venetoclax + HMA[1)]

Fit: Intensive Chemotherapy And / or Targeted Therapy[2)] ± Hematopoietic stem cell transplantation

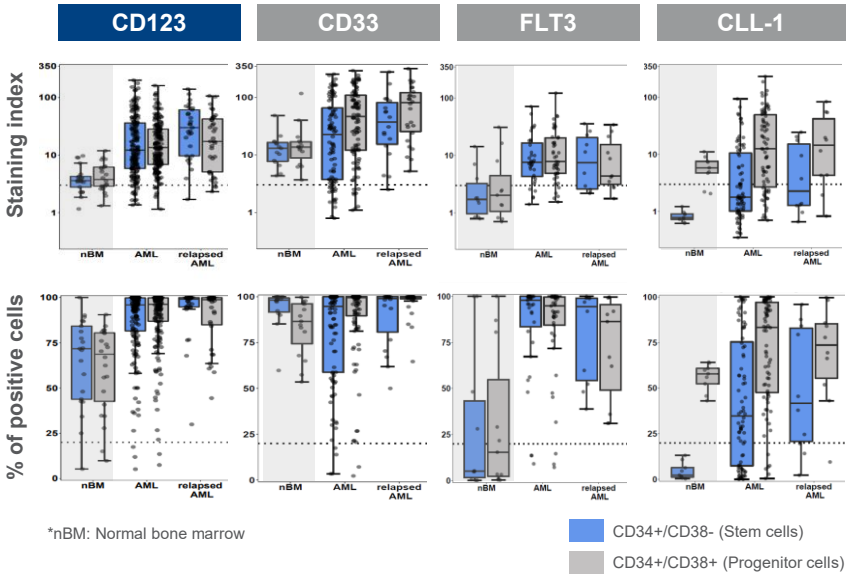
Recurrent Refractory

Intensive Chemotherapy And / or Targeted Therapy[3)] ± Hematopoietic stem cell transplantation

[1)] HMA (Hypomethylating Agent): azacitidine or decitabine
[2)] FLT3i (midostaurin, quizartinib), IDHi (ivosidenib), CD33-ADC (gemtuzumab ozogamicin)
[3)] FLT3i (gilteritinib), IDH1i (ivosidenib, olutasidenib), IDH2i (enasidenib), Menin-i (revumenib, ziftomenib)

CD123 is a Clinically Validated Target with High Selectivity for AML Cells

Expression of AML targets in AML & normal bone marrow



Target coverage, selectivity and indication scope

	CD123	CD33	FLT3	CLL1
Patients expressing at initial diagnosis	97.0%	96.4%	88%	80.01%
Patients expressing at relapse	98.1%	98.1%	80%	71.4%
Stem vs. progenitor expression	Similar	Higher in progenitors	Similar	Higher in progenitors
Normal tissue expression	Eosinophil, neutrophil, plasmacytoid DC	Hematopoietic cell, myeloid cell	HSC, progenitors	Granulocyte, monocyte
Alternative indications	MDS, CML, B-ALL, CLL, HL, BPDCN	MDS	MDS, ALL	MDS
Response-limiting genetic polymorphism	n/a	rs12459419 CT & TT genotypes (49%) less responsive to GO	n/a	n/a

GO: Gemtuzumab ozogamicin

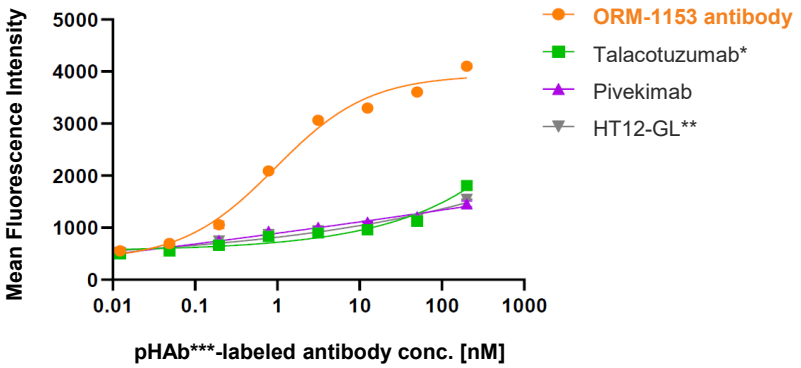
- **High AML expression at diagnosis and relapse**
- **Low expression in normal hematopoietic stem/progenitor cells**
- **Targeting both AML stem/progenitor cells to reduce relapse**
- **Broad therapeutic potential in hematopoietic cancers**

Herrmann H, et al. Blood Adv. 2020 Oct 27;4(20):5118-5132. Haubner S, et al. Leukemia. 2019;33(1):64-74. Carow CE et al. Blood. 1996;87(3):1089-1096; Lamba JK, et al. J Clin Oncol. 2017 Aug 10;35(23):2674-2682

ORM-1153's Antibody Demonstrates Enhanced Internalization Efficiency

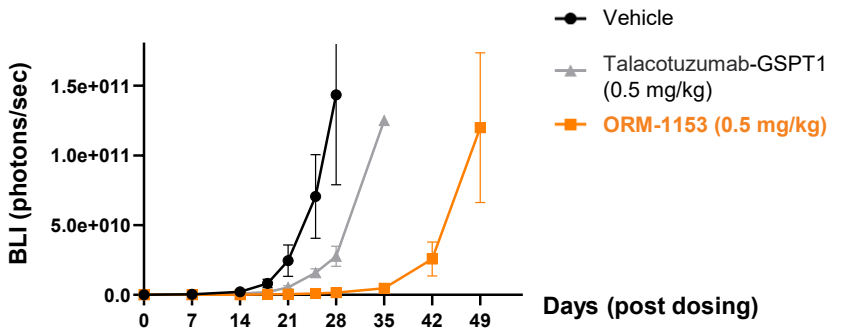
Sub-nanomolar CD123 binding enables rapid and efficient internalization, and ORM-1153 shows markedly greater in vivo efficacy vs. talacozotuzumab-GSPT1

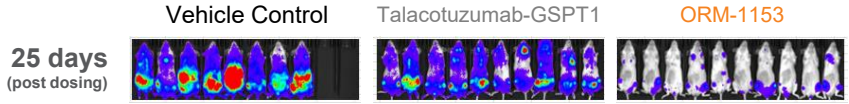
Internalization efficiency in MV-4-11 (24 hrs)



Binding kinetics	ka (1/Ms)	kd (1/s)	KD (M)
ORM-1153 antibody	**8.33E+06**	**5.38E-03**	**6.46E-10**
Talacotuzumab	8.24E+05	5.20E-06	6.30E-12
Pivekimab	2.90E+06	7.35E-05	2.53E-11
HT12-GL	6.01E+05	4.89E-05	8.13E-11

Disseminated MV-4-11-Luc xenograft mouse model

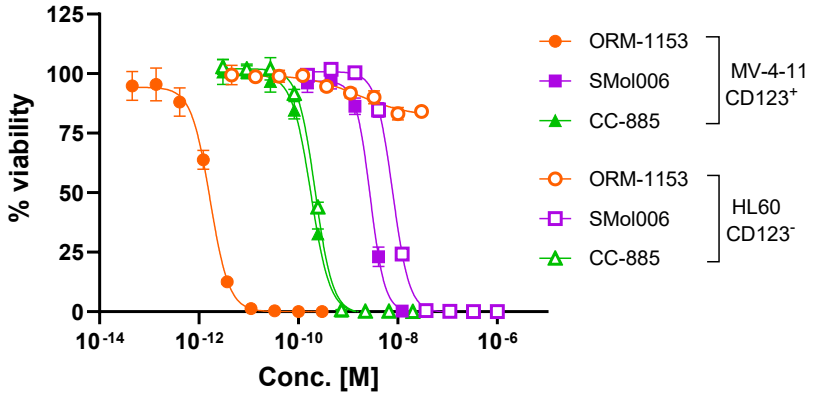




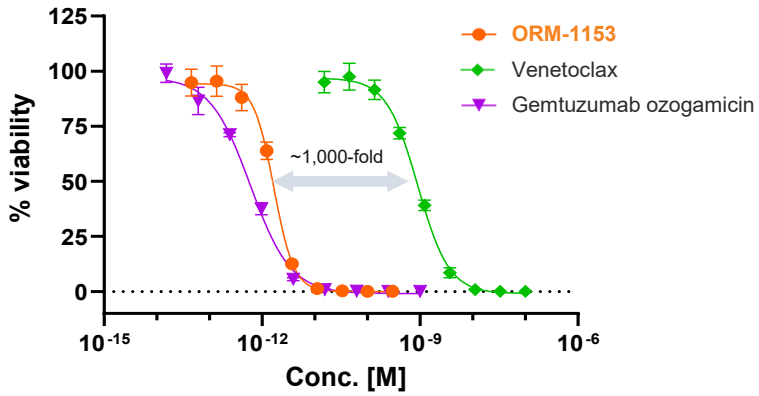
*Talacotuzumab: Anti-CD123 humanized monoclonal antibody (formerly CSL362)
**HT12-GL: antibody portion of AZD9829 (CD123-Topo1 ADC)
***pHAb Reactive dye

ORM-1153 Exhibits CD123-Dependent Activity and Strong Potency in AML Cell Lines

CD123-dependent cytotoxicity in CD123+/- AML cells



Comparative potency against SoC agents in MV-4-11



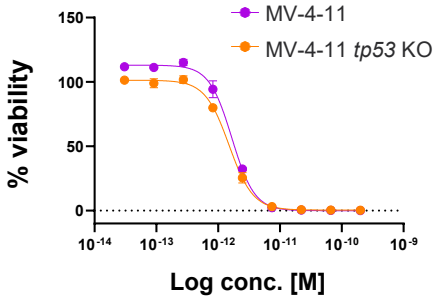
- Robust CD123-dependent cytotoxicity is observed, with strong activity in CD123$^+$ AML cells and negligible activity in CD123$^-$ cells. ORM-1153 demonstrates ~1,000-fold higher activity than the non-conjugated GSPT1 degrader.
- In MV-4-11(CD123$^+$/CD33$^+$) cells, ORM-1153 shows ~1,000-fold greater potency than venetoclax and is comparable to gemtuzumab ozogamicin (Mylotarg)

ORM-1153 Potently Inhibits Growth in AML TP53 Isogenic Cell Lines

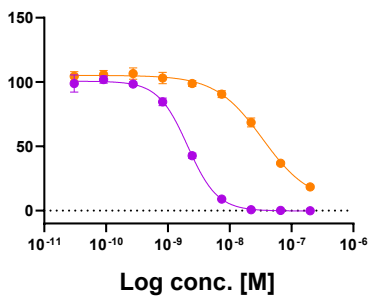
Cytotoxicity retained in TP53 KO cells with ORM-1153 indicating potential activity in TP53 mutant AML

MV-4-11 TP53 WT/KO cells

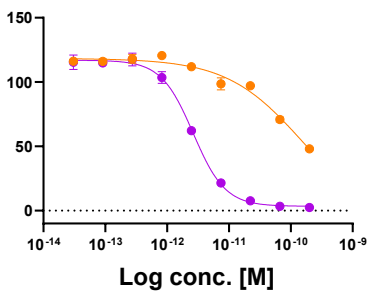
ORM-1153



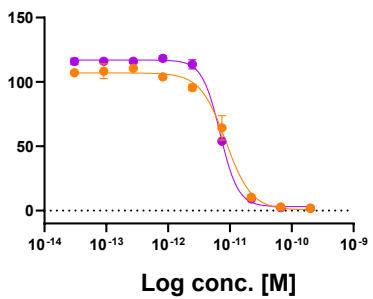
Venetoclax



Pivekimab sunirine (IMGN632)



Pivekimab-SMol249*



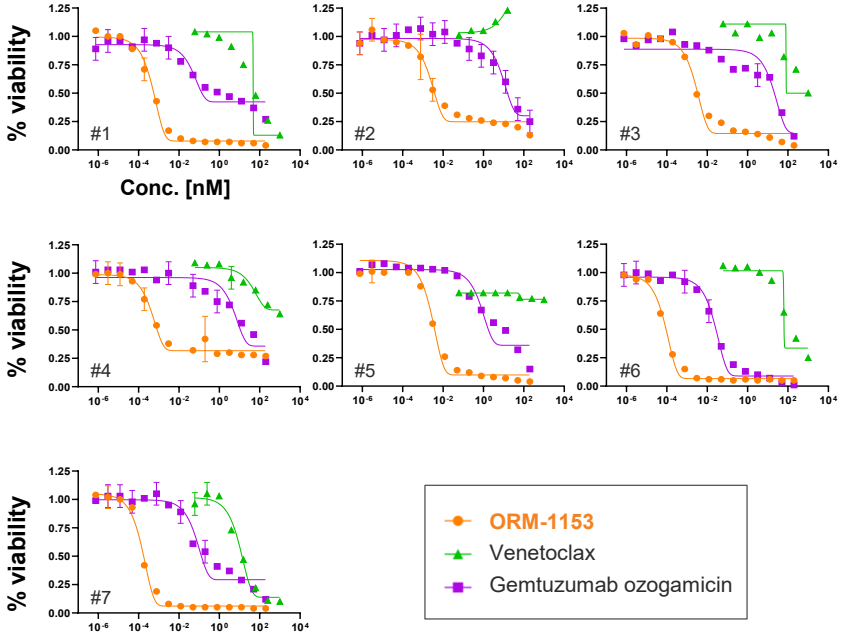
Test article	DAR	Fold change (KO / WT)	TP53 WT IC_{50} (M)	TP53 KO IC_{50} (M)
ORM-1153	**4**	**0.90**	**1.66E-12**	**1.50E-12**
Venetoclax	-	16.26	2.09E-09	3.40E-08
Pivekimab sunirine	2	57.95	2.64E-12	1.53E-10
Pivekimab-SMol249*	2	1.23	6.93E-12	8.58E-12

*Pivekimab-SMol249 is a control generated by conjugating SMol249, the linker-payload used in 1153, to the 442C mutation site of pivekimab, the antibody used in IMGN632

Robust ORM-1153 Activity in AML PDX Models Regardless of Gene Alterations

Consistent efficacy maintained across high-risk profiles including TP53 and FLT3-ITD

Ex vivo cytotoxicity and patient sample characteristics (#1 - #7)



Sample	Gender	Age	Diagnosis	Treatment	Gene Alternation	CD123 (ABC)
#1	F	59	Relapsed	Pretreated	IDH1, NPM1, FLT3-ITD	11131
#2	F	54	De novo	Naïve	TP53, NPM1, FLT3-ITD, KMT2A	4775
#3	M	NA	De novo	Naïve	WT**	4503
#4	F	68	De novo	Naïve	TP53, FLT3-ITD	3566
#5	M	80	De novo	Pretreated	FLT3-ITD	3485
#6	M	60	Relapsed	Pretreated	TP53, IDH2, NPM1	3197
#7	F	21	De novo	NA	WT**	2453

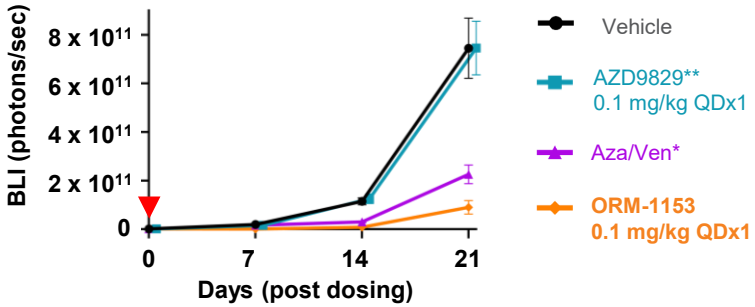
*Mylotarg is a CD33-targeting ADC approved for the treatment of AML
** Not associated with TP53, FLT3, IDH1/2, NPM1, or KMT2A gene alterations
NA – not available, ABC – antibodies bound per cell

Robust Tumor Control and Survival Benefit of ORM-1153 in Disseminated AML Model

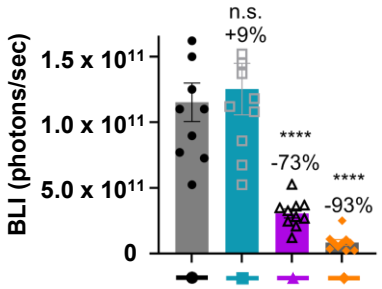
ORM-1153 drives greater reduction in tumor burden and robust survival compared to SoC and benchmark ADC

Disseminated MV4-11-Luc xenograft mouse model

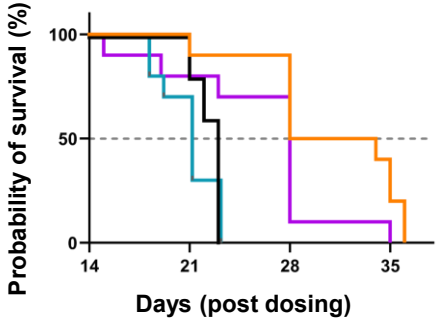
Greater reduction in AML burden over time

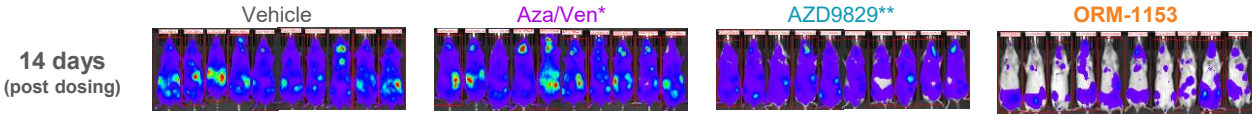


93% tumor reduction at Day 14



Robust overall survival at MED

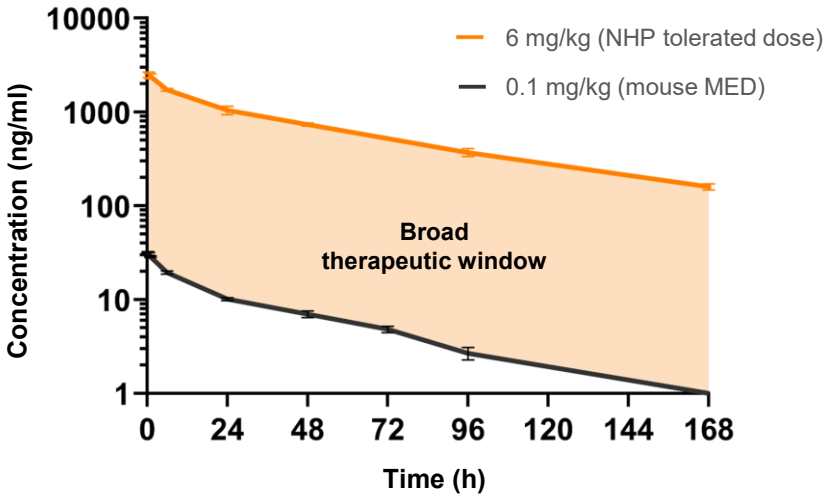




▲ = Dosing day
*Aza/Ven = Standard of care, azacitidine (2.5 mg/kg, IP QDx5) + venetoclax (100mg/kg, PO 5days/week x 3)
**AZD9829 is a discontinued clinical anti-CD123 ADC with a TOP1 inhibitor payload at DAR8

Broad Therapeutic Window and Favorable Safety Profile of ORM-1153

Exposure-based preliminary therapeutic window



- ✓ **Wide therapeutic margin established** between the minimal efficacious dose (MED; 0.1 mg/kg in mice) and the anticipated highest non-severely toxic dose (6.0 mg/kg in NHPs).
- ✓ **Favorable safety profile** in repeat-dose NHP studies, with off-target effects strictly limited to mild, transient, and reversible reductions in platelets and increases in liver enzymes

Broad Potential of ORM-1153 Across CD123+ Hematologic Malignancies

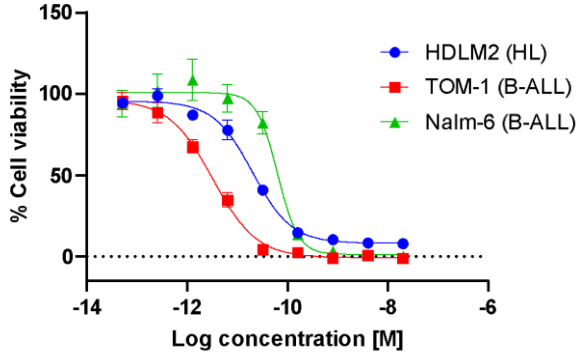
Demonstrated in vitro activity across other CD123+ models validates the expansive market potential for CD123+ indications beyond AML

Total US Addressable Market of ~27.7k Patients in Top 5 CD123+ Indications

	US incidence (2024)	CD123 (%)*	Pts expressing CD123**	Potential target unmet needs
MDS (High-risk)	~20K	~53%	~10.7k	HMA failure
Chronic myeloid leukemia (CML)	~10K	80 - 85%	~7.5k	TKI resistance
B-ALL	~5K	~90%	~4.4k	Post-SoC R/R
Hodgkin Lymphoma (HL)	~17K	60 - 90%	~1.5k	Post-SoC R/R
Chronic lymphocytic leukemia (CLL)	~2K	~20%	~3.6k	Post-SoC R/R

*Percentage of the patients expressing CD123 within each indication
**Calculated by applying CD123 expression rates to the total annual US incidence
Source : ClearView Analysis (2025)

ORM-1153 in vitro cytotoxicity in CD123+ cell lines



Cell line	Disease	In vitro cytotoxicity IC_{50} (M)
HDLM2	Hodgkin's lymphoma	2.03E-11
TOM-1	B-ALL	3.27E-12
Nalm-6	B-ALL	6.41E-11

ORM-1023: Clear Opportunity in SCLC and Neuroendocrine Tumors

GSPT1 activity in SCLC/NET

- Orum's in vitro and in vivo data show robust activities
- Clinical stage GSPT1 small molecule degrader has demonstrated activity in patients, albeit with a narrow therapeutic index

Broad antigen expression

- Incident cases of ES-SCLC in the US are >24K/year with ~90% of patients expressing target antigen
- Limited development of therapies targeting antigen, making ORM-1023 a potentially "first-in-class" opportunity

Expansion beyond SCLC

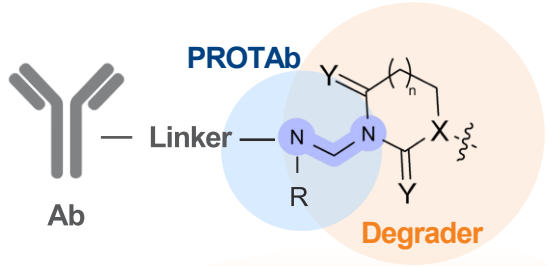
- Target also expressed in a high percentage of patients in several other high-unmet need tumors: neuroendocrine prostate, neuroblastoma, and breast cancer

TPD^2 PROTAb Platform

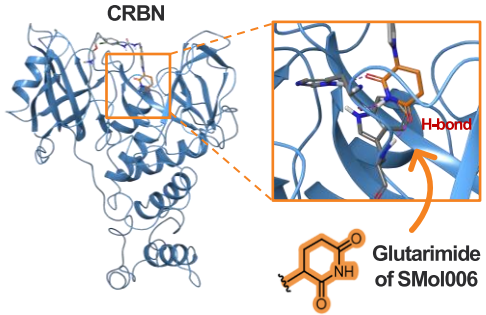
Traceless adaptor linker for degrader payloads

TPD² PROTAb: Linker Platform to Facilitate the Development of DACs

Adaptor for E3 ligase-based degrader conjugation



1 CRBN-based degrader | 2 Other E3 ligase degrader

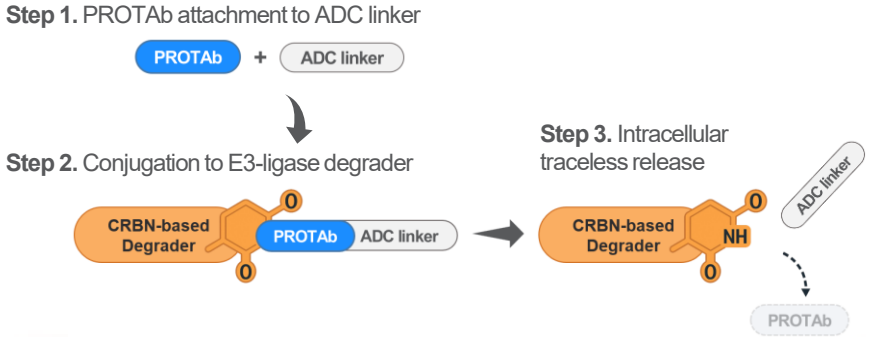


Extended to other E3 ligases

Note: Structure of SMol006 molecular glue binding to CRBN E3 ligase

2024 ADC Linker and Conjugation Summit, PROTAb Poster

PROTAb-enabled traceless conjugation process



Efficient, time-saving development path

- ✓ Reduces DAC development time by 6 to 9 months by eliminating trial-and-error chemistry through moiety-driven molecular adaptation for efficient linking and conjugation

Compatibility with diverse degrader payloads

- ✓ Offers a universal conjugation handle for both MGDs and heterobifunctional degraders

Broad linker compatibility

- ✓ Compatible with diverse linker designs, including protease-cleavable, thiol-sensitive, and β-glucuronide-cleavable linkers, etc.

PROTAb Enables Targeted Degradation with Diverse Hetero-Bifunctional Degraders

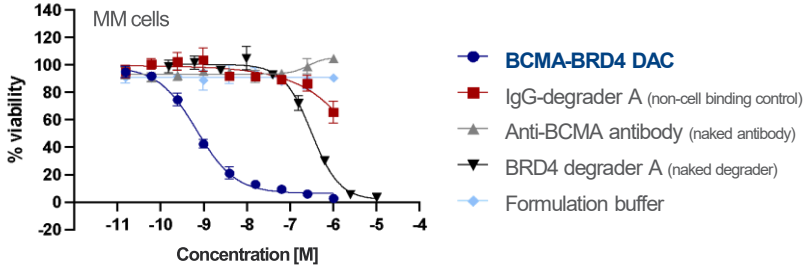
Demonstrates effective use of PROTAb with non-GSPT1 targets and hetero-bifunctional degraders

BCMA-BRD4

Anti-BCMA mAb

BRD4 Degrader

Targeted BRD4 degradation drives cytotoxicity



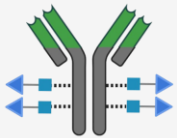
POC molecules PROTAb-DACs are highly effective *in vitro*

- ✓ BCMA-BRD4 improved potency by >3 logs compared to a small molecule alone
- ✓ CD79B-IRAK4 induced IRAK4 degradation at low nanomolar range, while maintaining antibody-level target binding affinity

→ **Supports platform scalability across diverse intracellular targets**

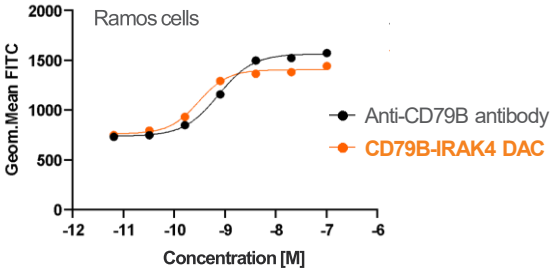
CD79B-IRAK4

Anti-CD79B mAb



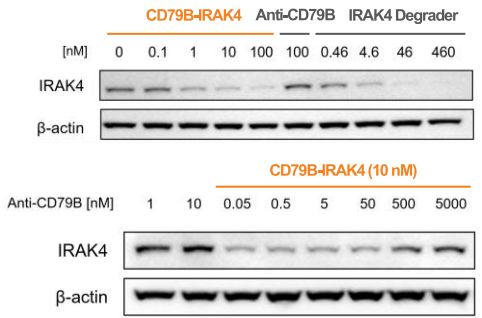
IRAK4 Degrader

Maintains antibody-level target binding affinity

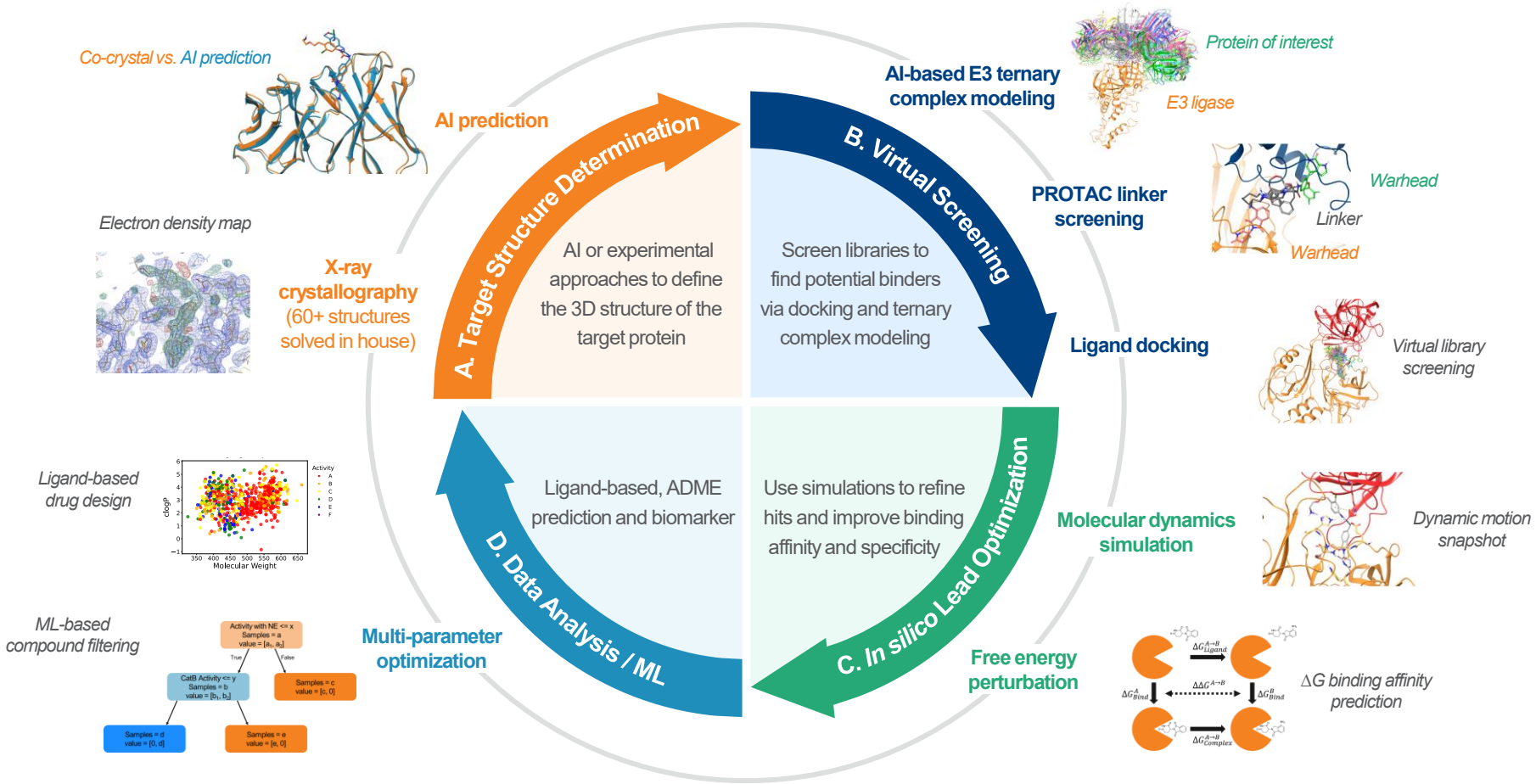


Dose-dependent degradation of IRAK4 at low nanomolar range

Target-mediated degradation via CD79B binding



Structure-based Discovery Workflow at Orum: From Target to Lead Candidate



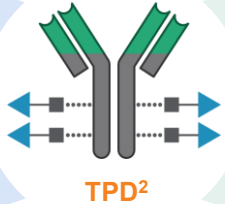
TPD² : Significant Potential to Unlock a Vast Number of Indications

Modular platform technology allows for creation of a multitude of targeted therapies

- **Orum's strength** lies in its ability to **develop** the antibody and degrader payload separately, tailoring these components to **enhance therapeutic benefit** in each indication

Degrader Targets (Intracellular)

Oncology		Non-Oncology
AR Bcl-xL BRD4 BRD9 BTK CDK9	ER GSPT1 IKAROS IRAK4 STAT3	BTK IKAROS IRAK4 JAK NLRP3 RIPK2 STAT3



Antibody Targets* (Surface)

Oncology		Non-oncology	
B7-H3 BCMA CD19 CD20 CD38 CD56 CD123 CLND18.2	DLL3 EGFR HER2 HER3 Nectin-4 PSMA ROR1 TROP2	BAFF-R BCMA CD19 IL-1R IL-3R IL-4R IL-5R	IL-6R IL-11R IL-13R TACI TNFR Family Integrin α4/β7

* CD33 excluded from target candidate in accordance with terms of BMS asset purchase agreement

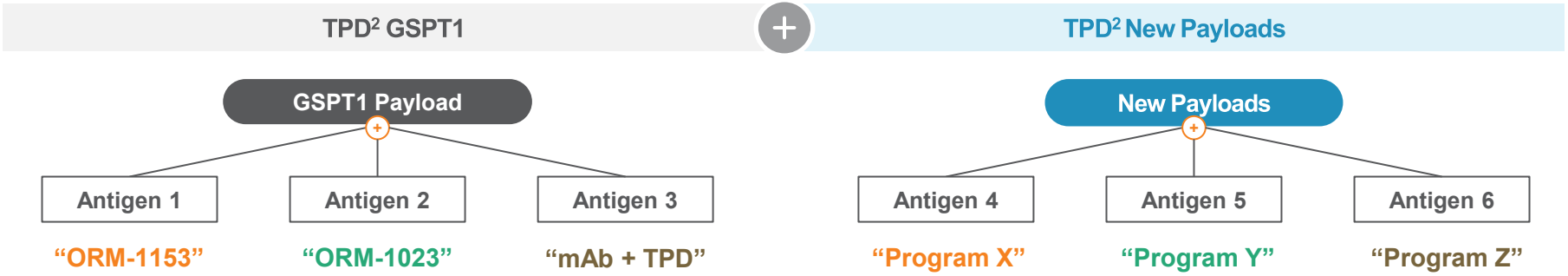
Indications

Oncology		Non-oncology
ALL AML Breast CCL Colon Gastric	MM NHL NSCLC Sarcoma SCLC	Asthma Atopic dermatitis Castleman disease Lupus Multiple sclerosis Rheumatoid arthritis Sjögren's syndrome Ulcerative colitis

Orum DAC Discovery Strategy for 2026 and Beyond

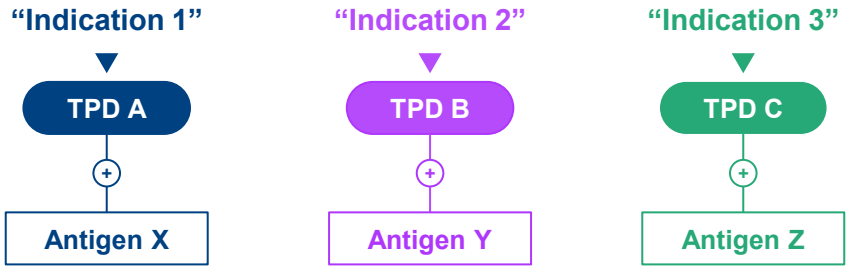
GSPT1 and next-generation payload : Unlocking platform potential across diverse targets

- Address limited activity or resistance associated with conventional payloads

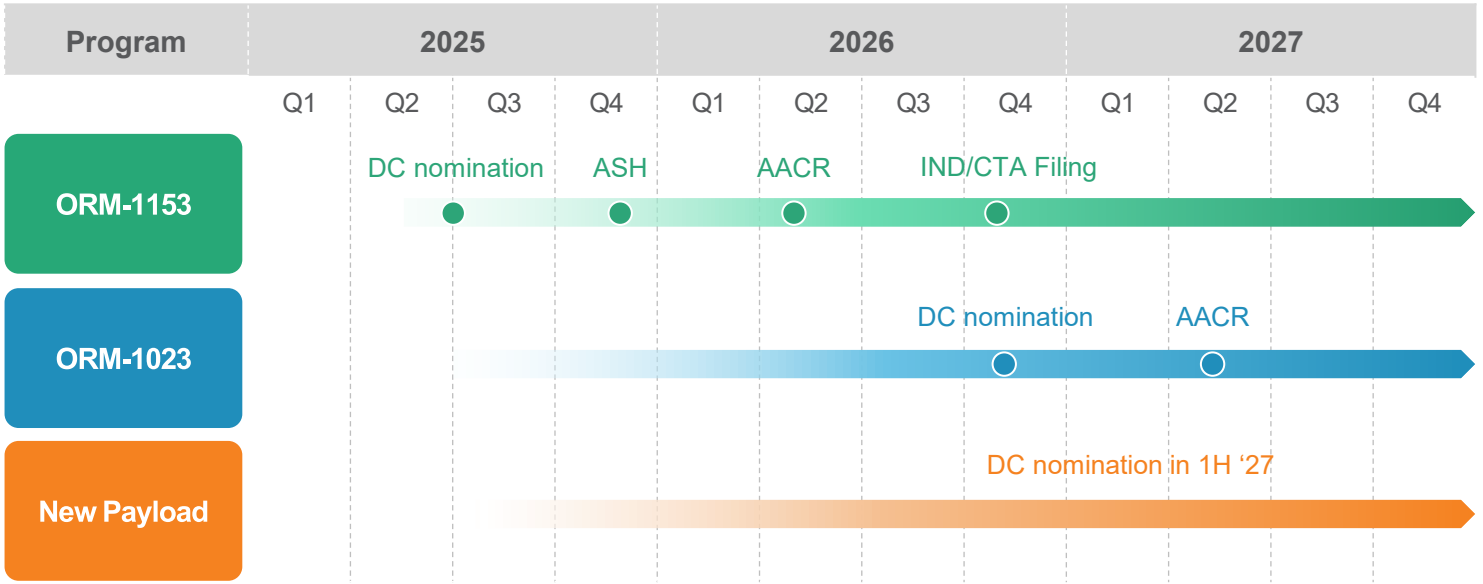


Indication-tailored payloads: Matching payload-antigen pairs to disease contexts

- "Ultra-specific" dual-targeting approach, with both antibody and payload aligned to disease-specific biology to maximize therapeutic index



Expected Timeline for 2026 and Beyond



Current Partnering Focus

- **Out-license** Orum's programs and platform technology
- Access to complementary external technologies through **collaborations** or **in-licensing**


ORÛM
THERAPEUTICS
Thank you
A world where no patients suffer from diseases caused by undruggable targets
ir@orumrx.com