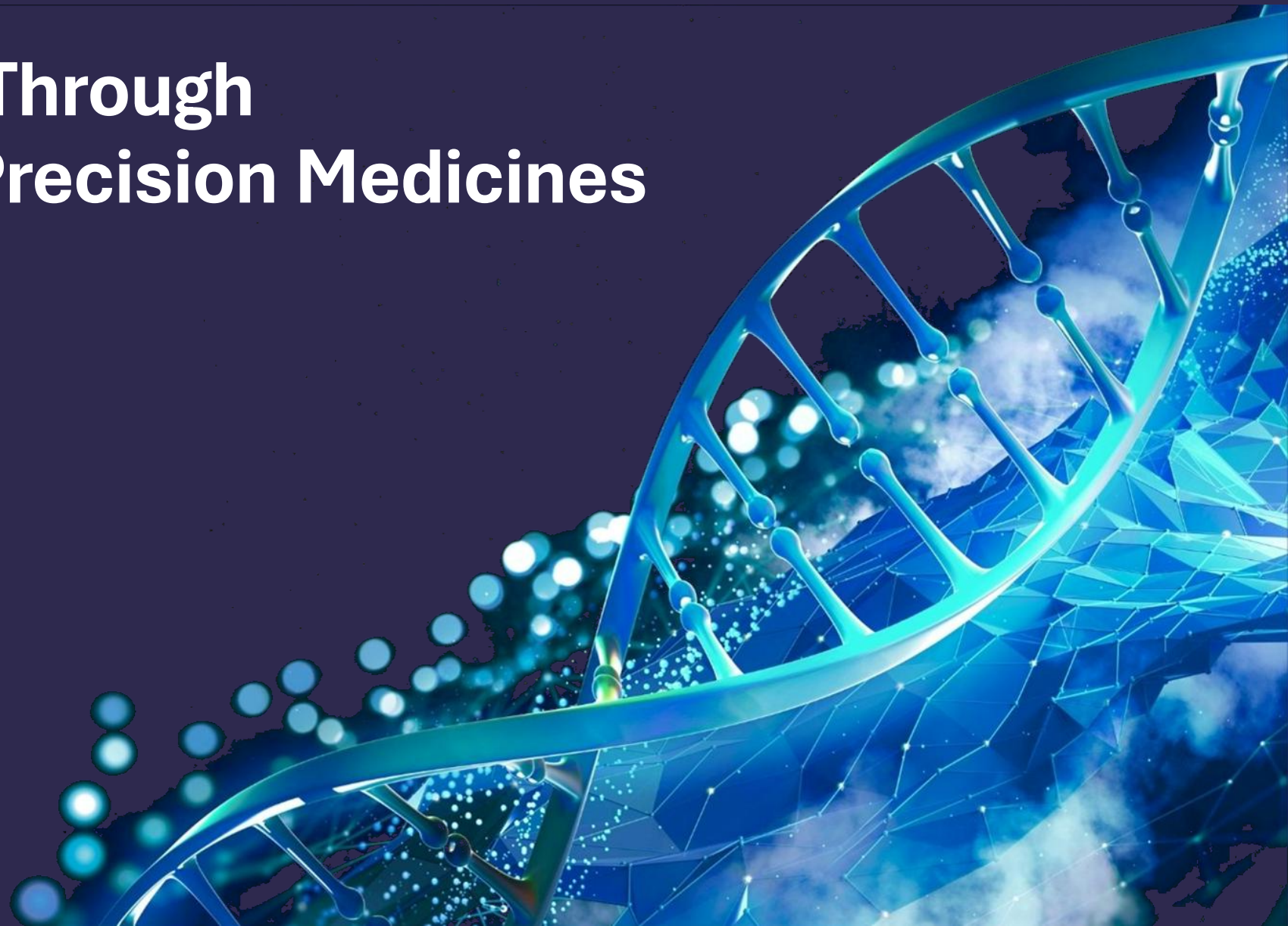
May 2026

Improving Lives Through Transformative Precision Medicines

Corporate Presentation

IDEAYA BIOSCIENCES

NASDAQ: **IDYA**



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Certain statements in this presentation and the accompanying oral commentary are forward-looking statements. These statements relate to future events or the future financial performance of IDEAYA Biosciences, Inc. (the “Company”) and involve known and unknown risks, uncertainties and other factors that may cause the actual results, levels of activity, performance or achievements of the Company or its industry to be materially different from those expressed or implied by any forward-looking statements. In some cases, forward-looking statements can be identified by terminology such as “may,” “will,” “could,” “would,” “should,” “expect,” “plan,” “anticipate,” “intend,” “believe,” “estimate,” “predict,” “potential” or other comparable terminology. All statements other than statements of historical fact could be deemed forward-looking, assumptions, estimates or projections that are subject to change, including expectations regarding the clinical activity profile, potential clinical benefit and potential advantages of the Company’s clinical programs; the translation of preliminary clinical trial results into future clinical trial results; the enrollment of clinical trials; the potentially addressable patient population for the Company’s programs; any expectations regarding the Company’s target discovery platform or new target validation efforts as creating opportunities for research and development initiatives; any projections of financial information, market opportunities, cash runway or profitability, including the estimated funding of operations into 2030; any statements about historical results that may suggest trends for the Company's business; any statements of the plans, strategies, and objectives of management for development programs or future operations; any statements about the timing of preclinical research, clinical development, regulatory filings, regulatory approvals, manufacturing or release of data; any statements of expectation or belief regarding future events, potential market dynamics , technology developments, or receipt of cash milestones, option exercise fees or royalties; and any statements of assumptions underlying any of the items mentioned. The Company has based these forward-looking statements on its current expectations, assumptions, estimates and projections. While the Company believes these expectations, assumptions, estimates and projections are reasonable, such forward-looking statements are only predictions and involve known and unknown risks and uncertainties, many of which are beyond the Company's control. Such risks and uncertainties include, among others, the uncertainties inherent in the drug development process, including the Company’s programs’ early stage of development, the process of designing and conducting preclinical and clinical trials, serious adverse events, undesirable side effects or unexpected characteristics of drug development, the regulatory approval processes, the timing of regulatory filings, the challenges associated with the manufacturing and/or commercialization of the Company’s product candidates; timing of product launches, potential pricing and reimbursement; potential revenue, expected breakthrough, best- or first-in-class or blockbuster status, regulatory landscape, economic conditions, competitive landscape, the Company’s ability to successfully establish, protect and defend its intellectual property, and other matters that could affect the sufficiency of existing cash to fund operations. These and other important factors may cause actual results, performance or achievements to differ materially from those expressed or implied by these forward-looking statements. The forward-looking statements in this presentation are made only as of the date hereof. For a further description of the risks and uncertainties that could cause actual results to differ from those expressed in these forward-looking statements, as well as risks relating to the business of the Company in general, see the Company's periodic filings with the Securities and Exchange Commission (the "SEC"), including its Annual Report on Form 10-K for the year ended December 31, 2025 and any current or periodic reports filed with the SEC. Except as required by law, the Company assumes no obligation and does not intend to update these forward-looking statements or to conform these statements to actual results or to changes in the Company's expectations.

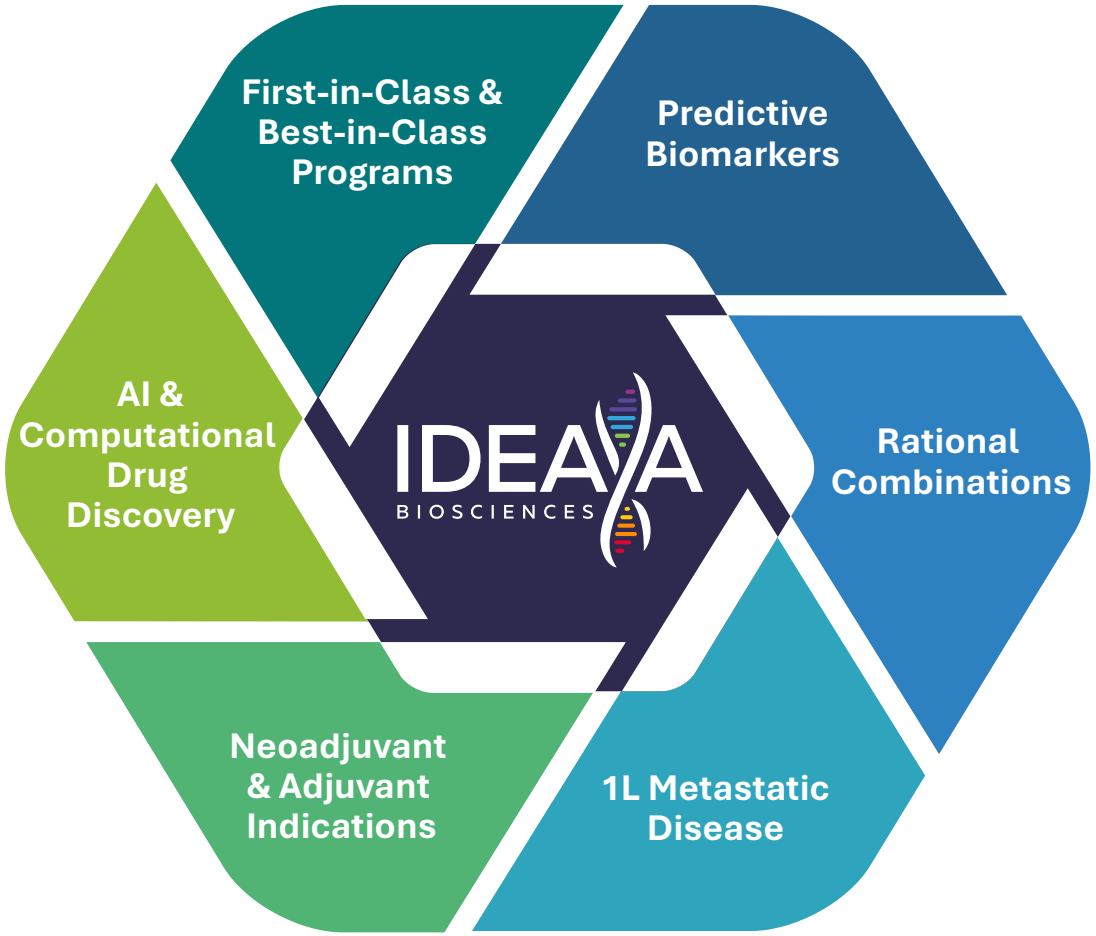
Other

This presentation concerns anticipated products that are under clinical investigation and which have not yet been approved for marketing by the FDA or any other country regulatory authority. These anticipated products are currently limited by Federal law to investigational use, and no representation is made as to their safety or effectiveness for the purposes for which they are being investigated.

Certain information contained in this presentation relates to or is based on studies, publications, surveys and other data obtained from third-party sources and the Company’s own internal estimates and research. The Company has not independently verified, and makes no representation as to the adequacy, fairness, accuracy or completeness of any information obtained from third-party sources. In addition, all of the market data included in this presentation involves a number of assumptions and limitations, and there can be no guarantee as to the accuracy or reliability of such assumptions. Finally, the Company's own internal estimates and research have not been verified by any independent source.

IDEAYA's Vision is to Build a Leading Precision Medicine Oncology Company

IDEAYA Research and Development Strategy



Clinical Pipeline Focus Areas

Darovasertib

Prolong survival, preserve the eye and vision in uveal melanoma

ADC+DDR Combos

Improve efficacy and durability of TOP1 ADCs

MTAP Pathway

Exploit synthetic lethality for ~15% of solid tumors

KAT6/7

Target tumor heterogeneity to enhance durability

1L = First-line, AI = artificial intelligence, ADC = antibody drug conjugate, DDR = DNA damage response, MTAP = methylthioadenosine phosphorylase

IDEAYA's Proven Drug Discovery Engine in Precision Medicine Oncology

Integrated Platform to Deliver Renewable Pipeline of Potential First-in-Class Programs

6 ***internally discovered*** clinical stage molecules across six potential ***first-in-class targets***

Candidates delivered against ***difficult-to-drug target classes***, including helicases, polymerases, and glycohyrdolases

Extensive physics and AI-based drug design & lead discovery platform for accelerated IND delivery

Novel selectivity profiles achieved *vs. paralogs and protein families, including against kinases and lysine acetyltransferases*

Core IDEAYA Drug Discovery Capabilities

CRISPR-based target and biomarker discovery

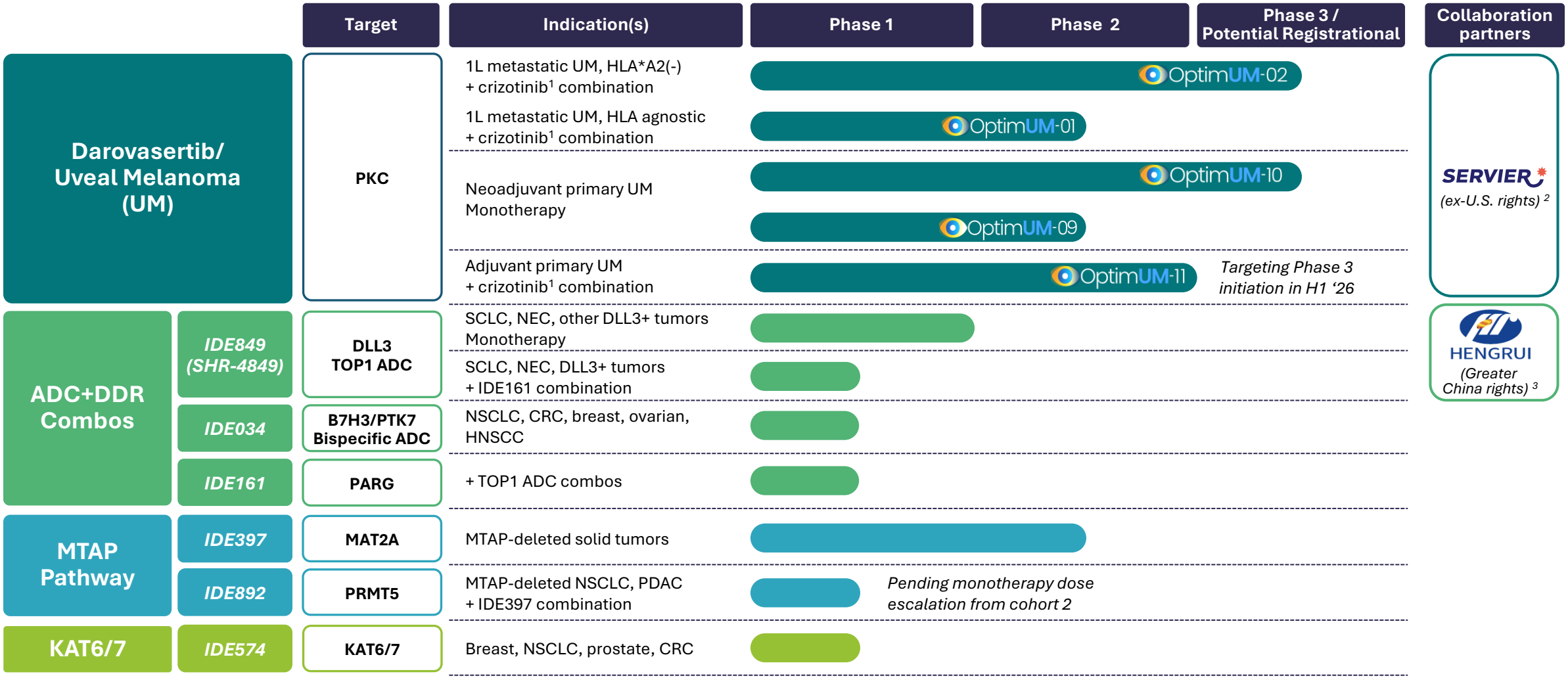
Chemigenomic-enabled translational research

Protein sciences and structural biology

Computational drug discovery enabled by AI & proprietary high-resolution structural data sets across novel target classes

IND = investigational new drug application

Pipeline Enables Combinations and Targeting of Multiple Solid Tumor Indications



(1) Pfizer's oral c-MET inhibitor; (2) Pursuant to an exclusive ex-U.S. licensing deal with Servier, IDEAYA retains all rights to darovasertib in the U.S. and is eligible to receive a total of $320M in regulatory and commercial milestones and double-digit royalties on all ex-U.S. net sales; (3) Pursuant to an exclusive licensing agreement with Jiangsu Hengrui, IDEAYA controls worldwide rights outside of Greater China

HLA = human leukocyte antigen, SCLC = small cell lung cancer, NEC = neuroendocrine carcinoma, NSCLC = non-small cell lung cancer, CRC = colorectal cancer, HNSCC = head and neck squamous cell carcinoma

Uveal Melanoma (UM) is a Rare, Aggressive Form of Cancer with Poor Prognosis

Patients face severe consequences with limited treatment options at all stages of disease

Primary UM
(Localized disease in the eye)

> 3,000 diagnosed in the U.S. per year
> 10,000 globally per year[1]

Enucleation

20%
of patients

lose their eye to surgery,
often within weeks of diagnosis

Radiation
(plaque brachytherapy)

can cause permanent vision loss
and life-long disability

Metastatic UM
(Systemic disease)

~50% progress to metastatic disease

Poor prognosis

- Median OS: **10-12 months**
- Five-year survival rate: **15-20%**
- Frequency of liver metastasis: **~90%**

Limited Treatment Options

< 50% patients eligible for the only approved systemic therapy

Liver-directed therapy invasive and complex

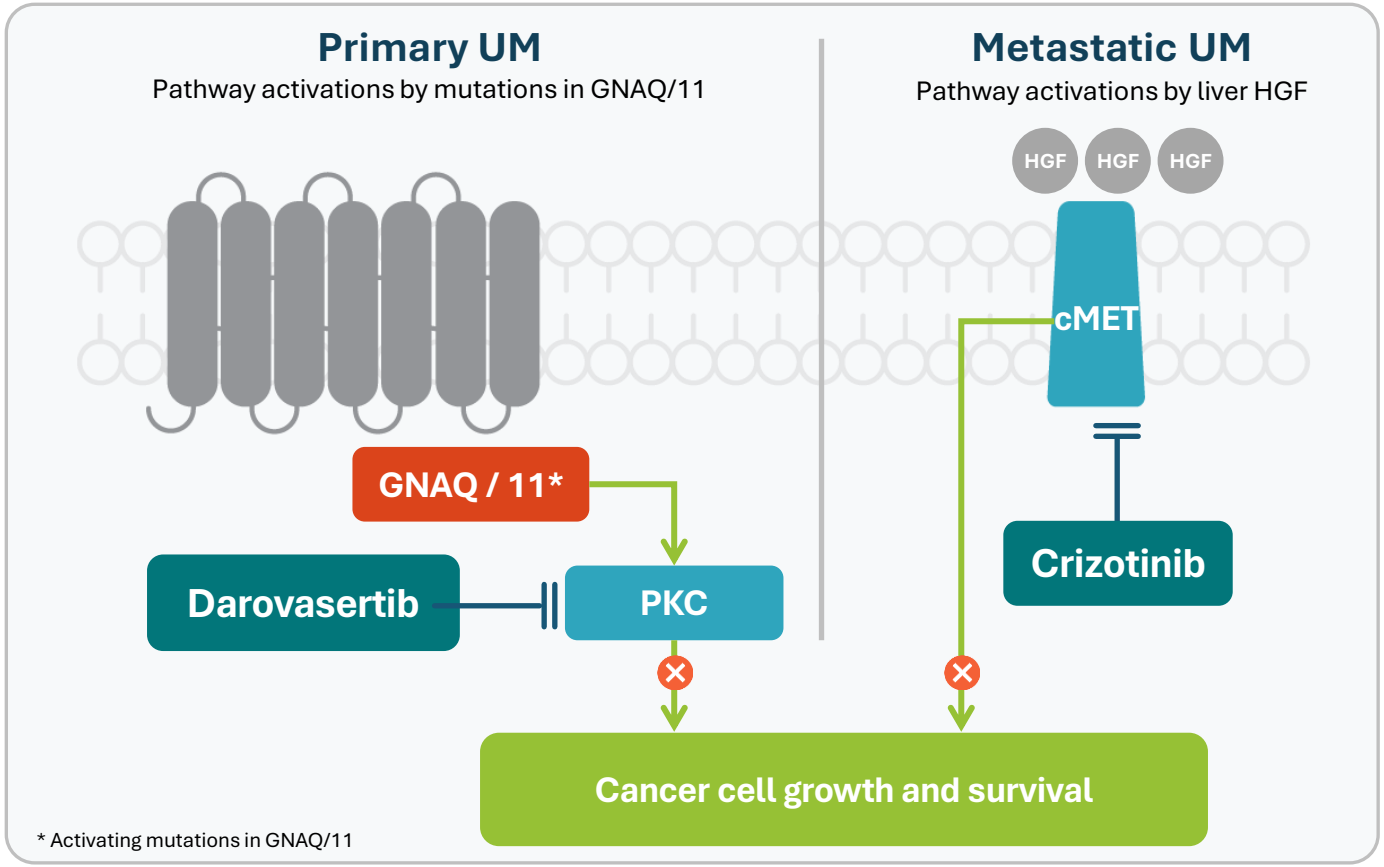
Off-label use of checkpoint inhibitors has limited efficacy

(1) Estimated based on Helgadottir et. al., Appl of Clin Genet (2016) 9: 147–155; and Hou et. al., Adv in Ophth Practice and Research (2024) 4: 226-232
OS = overall survival

Darovasertib Has a Unique Mechanism of Action Targeting the Primary Driver of Disease

Activating mutations in GNAQ/11 drive PKC overactivation in nearly all UM patients

- UM is biologically distinct from cutaneous melanoma, **driven by GNAQ/11 mutations that result in PKC overactivation** and tumor growth
- Darovasertib is an oral, selective inhibitor of **PKC, the key oncogenic driver** in >95% of patients
- Blocking PKC with **darovasertib exploits a common weakness** in all UM tumors
- In metastatic UM (mUM), **darovasertib is combined with crizotinib**, an oral inhibitor of the cMET pathway, which is believed to play a central role in metastatic spread
- This combination has the **potential to improve survival in mUM**, regardless of HLA*A2 status



A daily, all-oral targeted regimen has the potential to improve compliance, treatment outcomes and quality of life for UM patients

PKC = protein kinase C, HGF = hepatocyte growth factor

OptimUM-02 Summary of Topline Data from Phase 2/3 OptimUM-02 trial

Trial met its primary endpoint of statistically significant improvement in median PFS by BICR

Median PFS (1º endpoint)	Statistically significant improvement in median PFS by BICR 6.9 months for the darovasertib combination versus 3.1 months for ICT control (HR: 0.42; 95% CI: 0.30, 0.59; p-value: <0.0001)
ORR/DOR (2º endpoints)	37.1% ORR (including 5 CRs) for the darovasertib combination versus 5.8% for ICT control arm (p-value: <0.0001); median DOR 6.8 months
Overall Survival	Darovasertib combination showed an early trend in improvement for OS, though data still immature
Safety / tolerability	Well-tolerated, with manageable safety profile consistent with previously reported AEs

Complete data to be presented at ASCO 2026

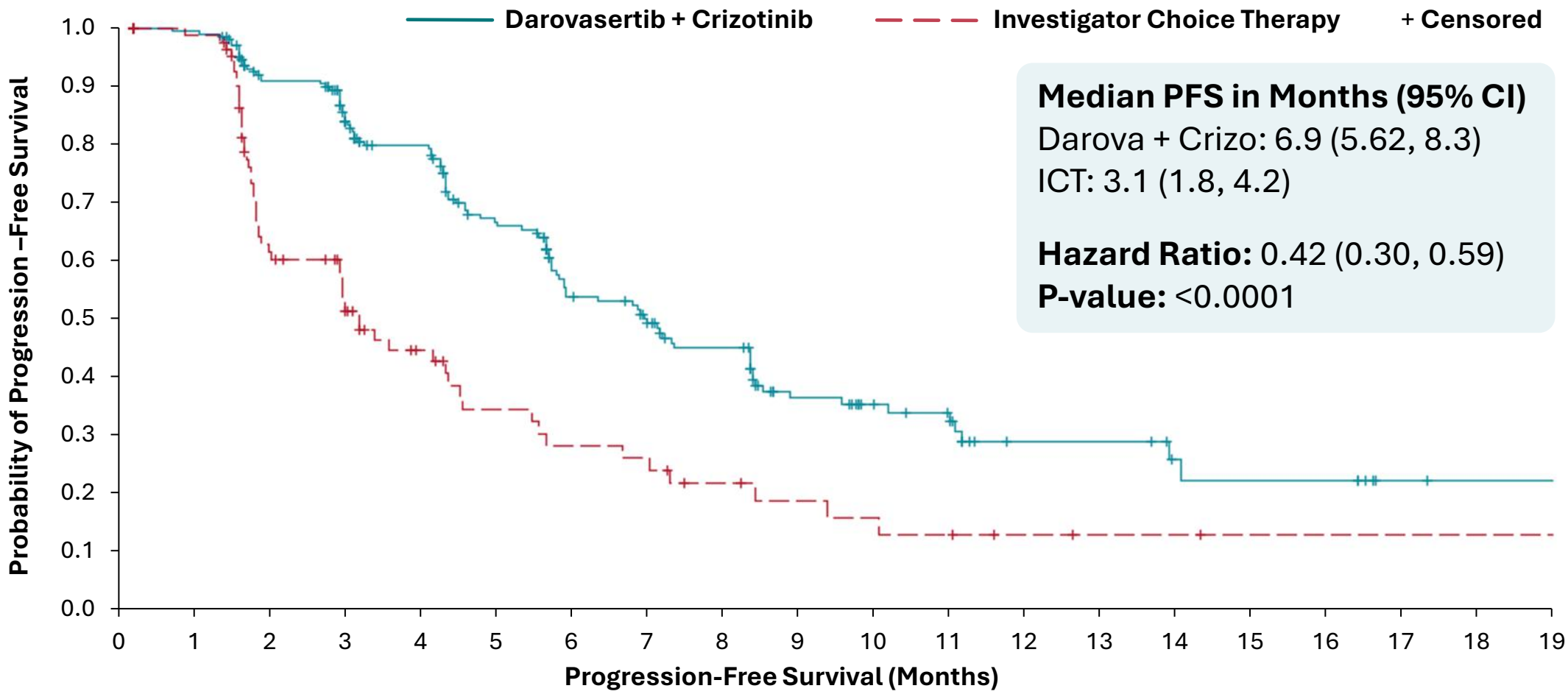
NDA submission to be completed in H2 '26

BICR = blinded independent central review, PFS = progression-free survival, ICT = Investigator Choice Therapy (76% Ipi + Nivo; 24% Pembro), ORR = overall response rate, DOR = duration of response, CR = complete response, AEs = adverse events. Data as of the cut-off date of January 23, 2026

OptimUM-02 Statistically significant improvement in median PFS by BICR

Darovasertib combination reduced the risk of disease progression by 58% vs. ICT control

Kaplan-Meier (KM) Estimate of Progression-Free Survival (PFS)*



Median PFS in Months (95% CI)
Darova + Crizo: 6.9 (5.62, 8.3)
ICT: 3.1 (1.8, 4.2)

Hazard Ratio: 0.42 (0.30, 0.59)
P-value: <0.0001

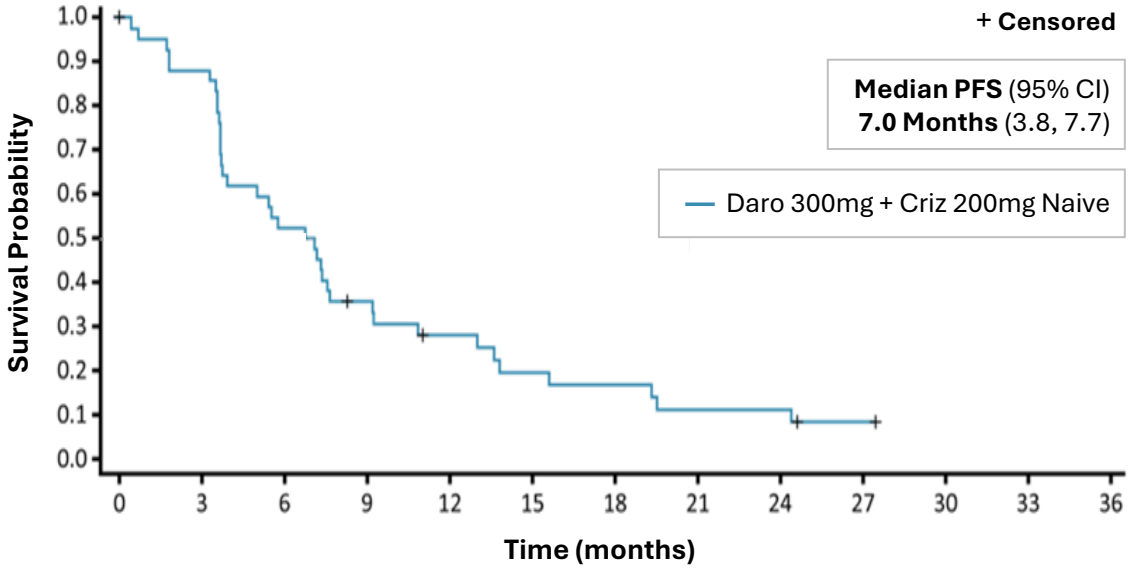
* Data as of the cut-off date of January 23, 2026

OptimUM-01 First Reported Overall Survival with Darovasertib + Crizotinib

2025 SMR CONGRESS

Median PFS and OS compared favorably to historical meta-analyses in front-line mUM

Median PFS

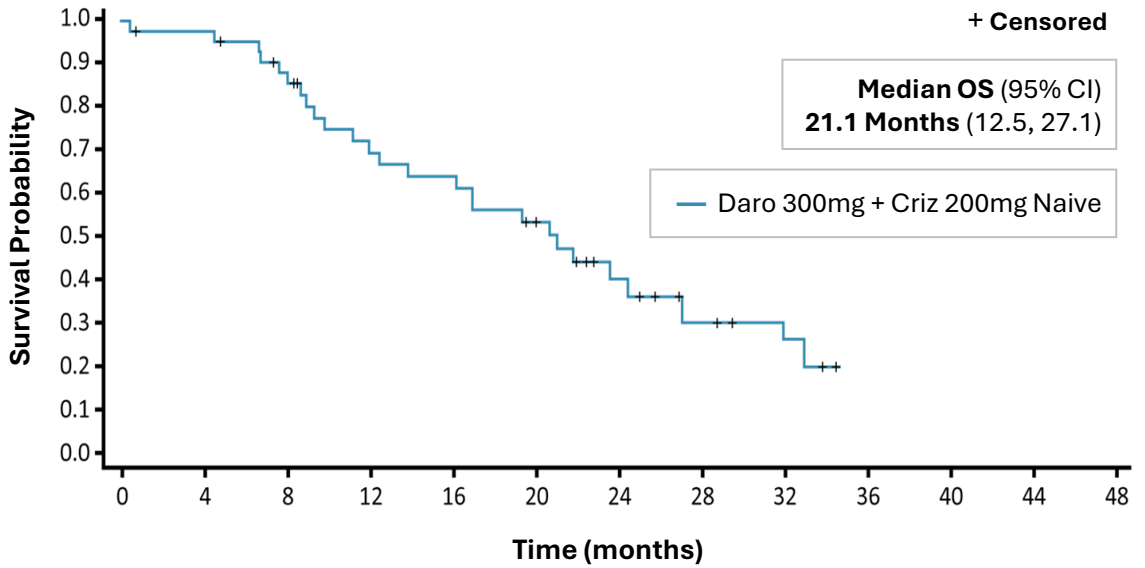


mPFS of 7.0 months
(95% CI: 3.8, 7.7, median follow-up 25 months)

Historical mPFS of 2.8 months[1]

Consistent with 7.1 months and 6.9 months mPFS previously reported at ESMO 2023 and in OptimUM-02

Median OS



mOS of 21.1 months
(95% CI: 12.5, 27.1, median follow-up 25 months)

Historical mOS of 10-12 months[1-2]

(1) Khoja L, et al. J Clin Oncol. 2025;43 (suppl) Abstract 9539; (2) Rantala ES, et al. Melanoma Res. 2019;29:561-568
Cross trial comparisons are not being made and for informational purposes only

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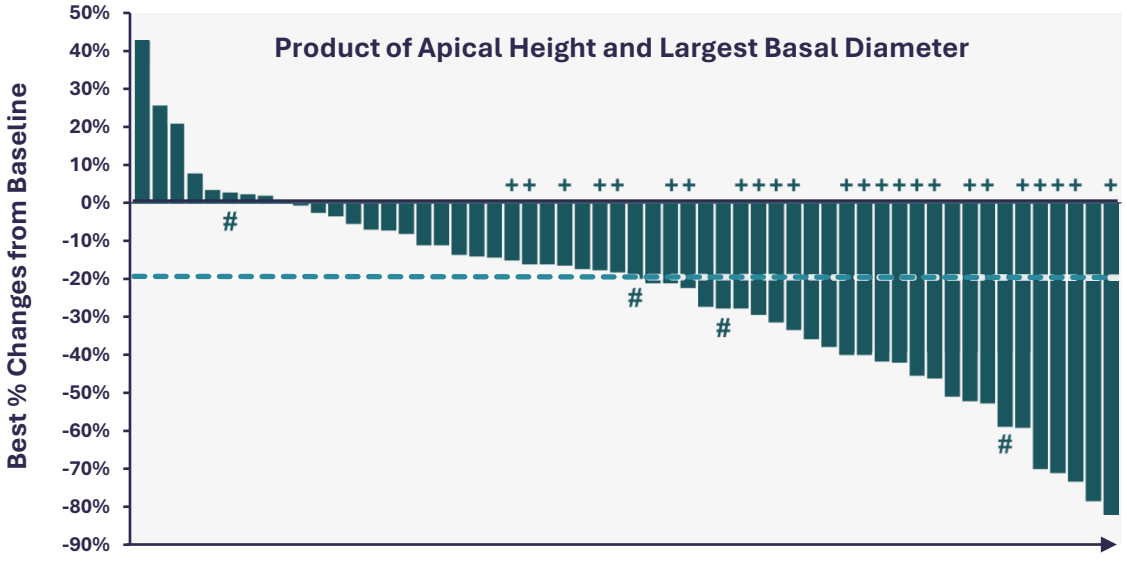
OptimUM-09 Primary Phase 2 Efficacy Results in Neoadjuvant UM

BERLIN 2025 ESMO congress

Robust tumor shrinkage leading to eye preservation and visual improvements

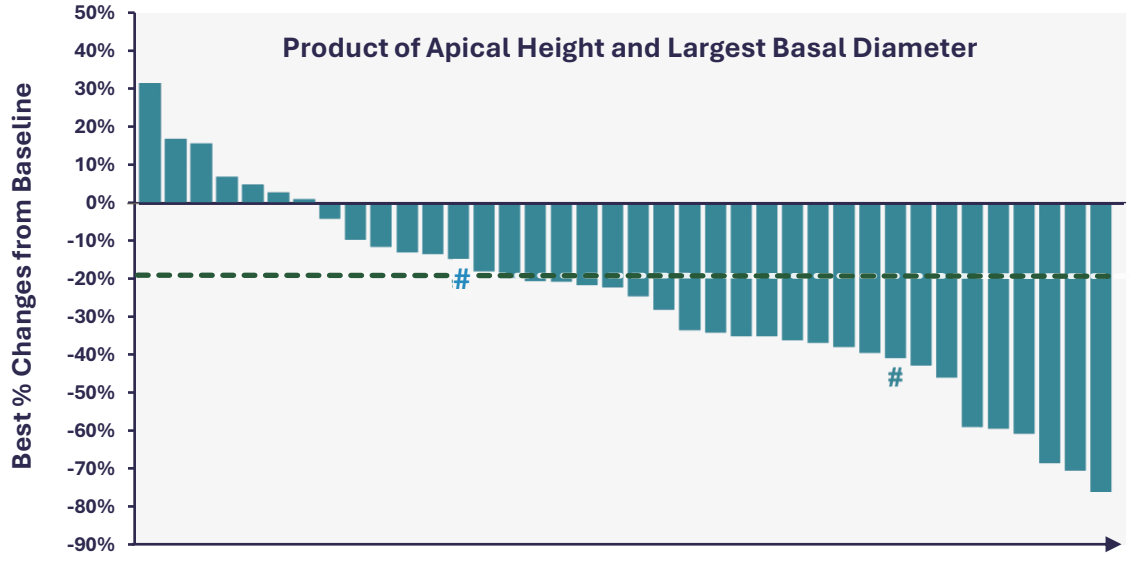
Cohort 1 (Enucleation)

Tumor Shrinkage and Eye Preservation



Cohort 2 (Plaque Brachytherapy)

Tumor Shrinkage and Visual Improvement



50%
of patients had **tumor reduced** by **≥20%**

57%
of patients were able to **preserve their eye**

57%
of patients **had visual gains during therapy,** with an average **14 letters***

60%
of patients had **tumor reduced by ≥20%**

65%
of patients had **reduced risk of predicted vision loss** after 3 years

+ Patient converted from enucleation to eye preserving therapy. # Patients ongoing on neoadjuvant treatment. Per protocol, efficacy evaluable population (N=56) for Cohort 1 and (N=38) for Cohort 2 was defined as all patients who received at least one dose of study drug and have at least one post-baseline tumor assessment. One patient was not evaluable and therefore not included in the efficacy evaluable population in Cohort 2. * Blended average based on patients in enucleation and plaque brachytherapy cohorts

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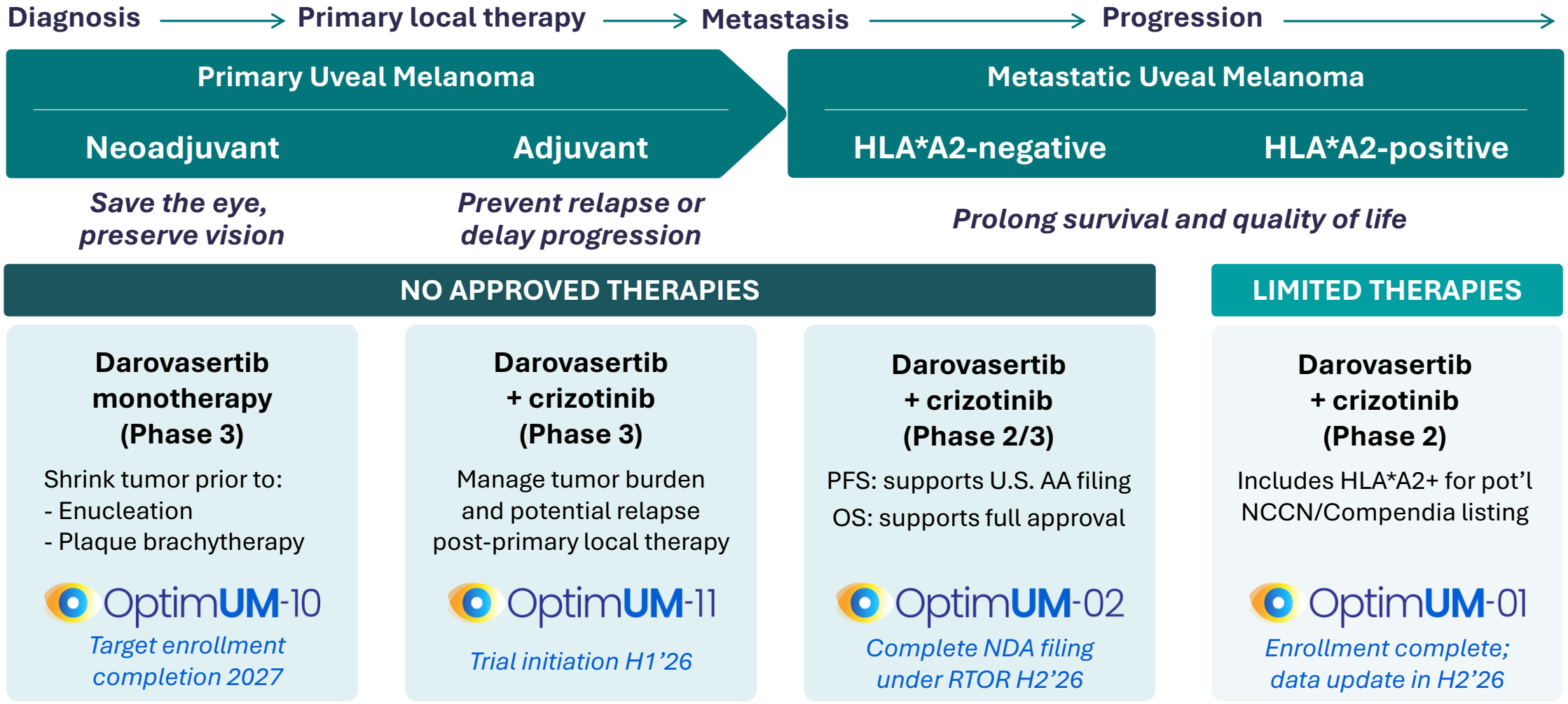
Darovasertib Has Potential To Be the First Targeted Therapy for All Stages of UM

Robust clinical development plan across the uveal melanoma patient journey

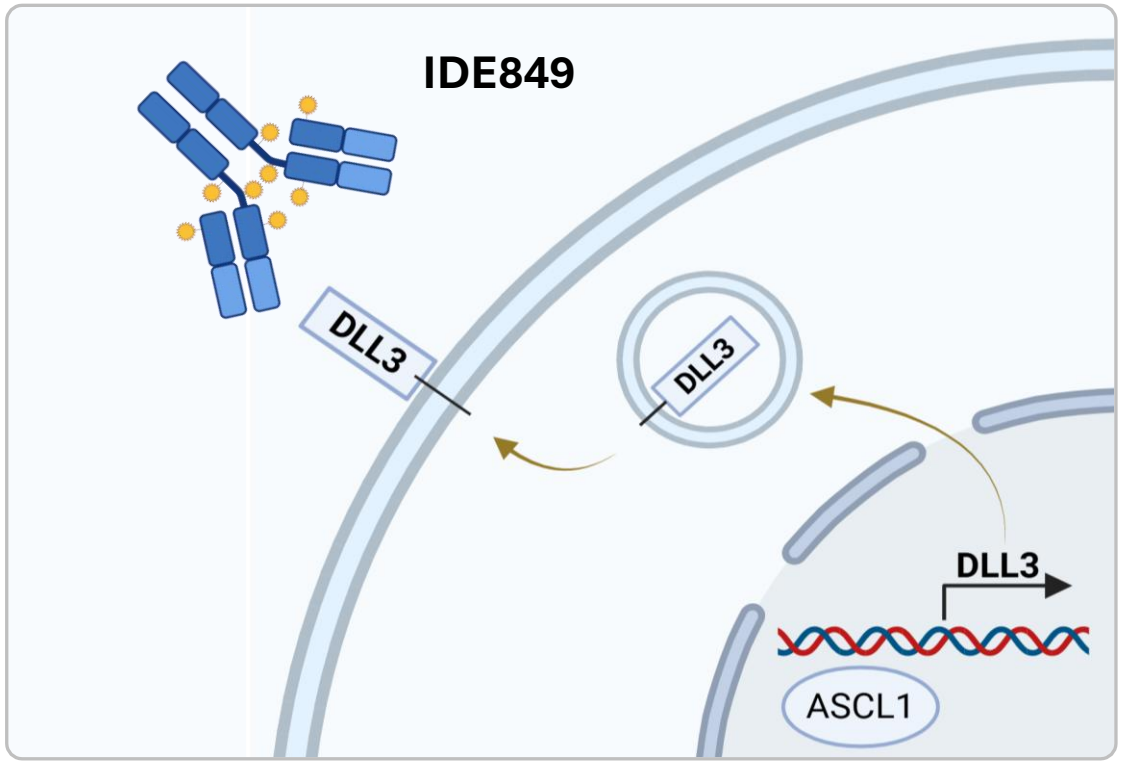


FDA ▶ **Orphan Drug Designation in UM[1]; Fast Track Designation in mUM; Breakthrough Therapy Designation[2]**

(1) Orphan Drugs benefit from certain tax credits and may be excluded from certain mandatory price negotiation provisions of the 2022 Inflation Reduction Act
(2) Breakthrough therapy designation for the neoadjuvant treatment of adult patients with primary uveal melanoma for whom enucleation has been recommended
PFS = progression free survival, OS = overall survival , AA = accelerated approval, NCCN = national comprehensive cancer network, RTOR = real-time oncology review

Building a Franchise of TOP1 ADCs to Synergize with IDE161 (PARG inhibitor)

Lead ADC product candidate, IDE849, has TOP1 payload and high DLL3 affinity and selectivity



- Strong affinity, high selectivity
- Proprietary TOP1 payload with ~4,000 patients treated to date
- Optimized, DAR 8 format
- Internalization-dependent cleavable linker
- High plasma stability

Tumor-selective delivery of **TOP1 ADC in combination with systemic PARG inhibition** via IDE161 may synergize to **increase therapeutic window** and **enhance efficacy**

Tumor-restricted expression

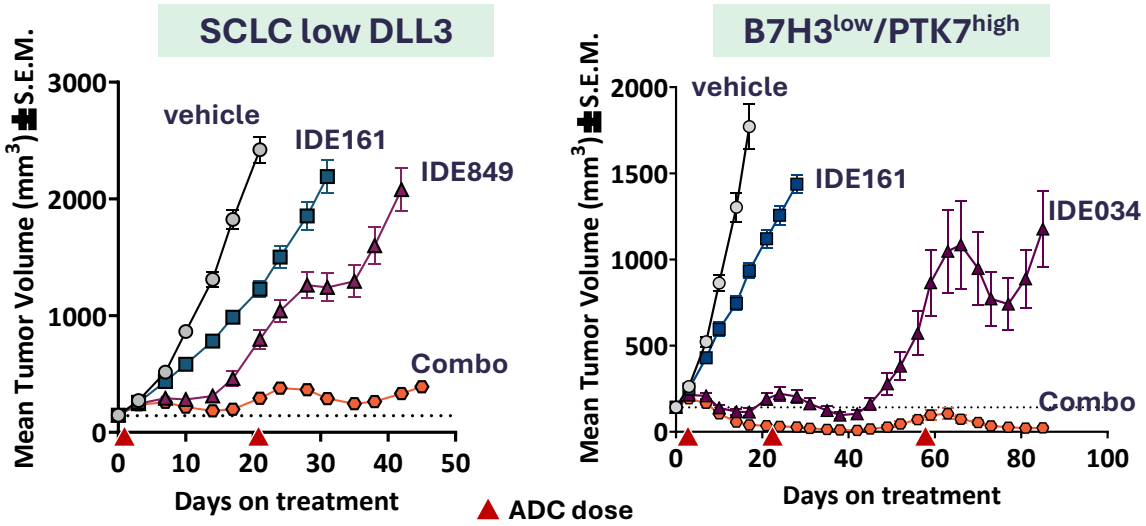
IDE849 *(DLL3)*

IDE034 *(B7H3/PTK7)*

Bispecific, AND gate format

IDE161 *(PARG)*

Chemically-installed synthetic lethality



DLL3 = Delta-like ligand 3, ADC = antibody drug conjugate, TOP1 = topoisomerase I, DAR = drug-to-antibody ratio, ASCL1 = Achaete-Scute family BHLH transcription factor 1, PARG = poly(ADP-ribose) glycohydrolase

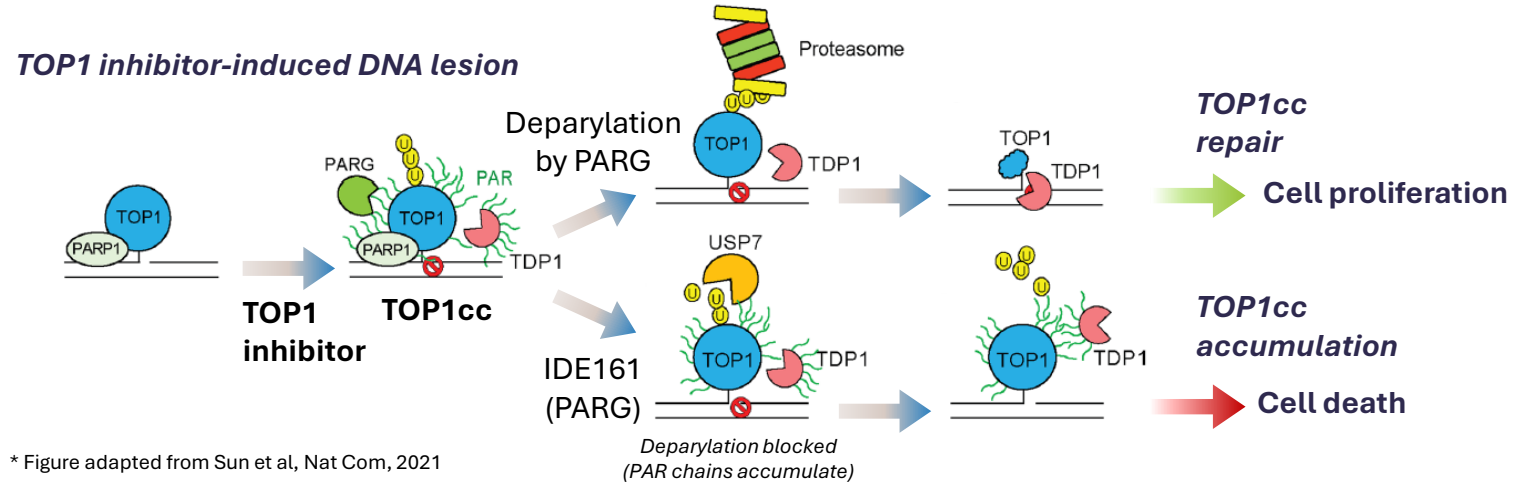
IDE161 Has The Potential to Improve Efficacy and Durability of TOP1 ADCs

Combination mechanism has shown broad potential across multiple solid tumor models

AACR American Association for Cancer Research FINDING CURES TOGETHER™

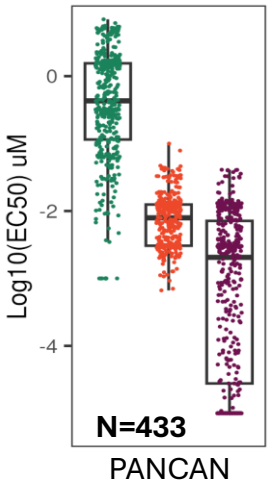
PARG inhibition by IDE161 maximizes TOP1 ADC payload efficacy

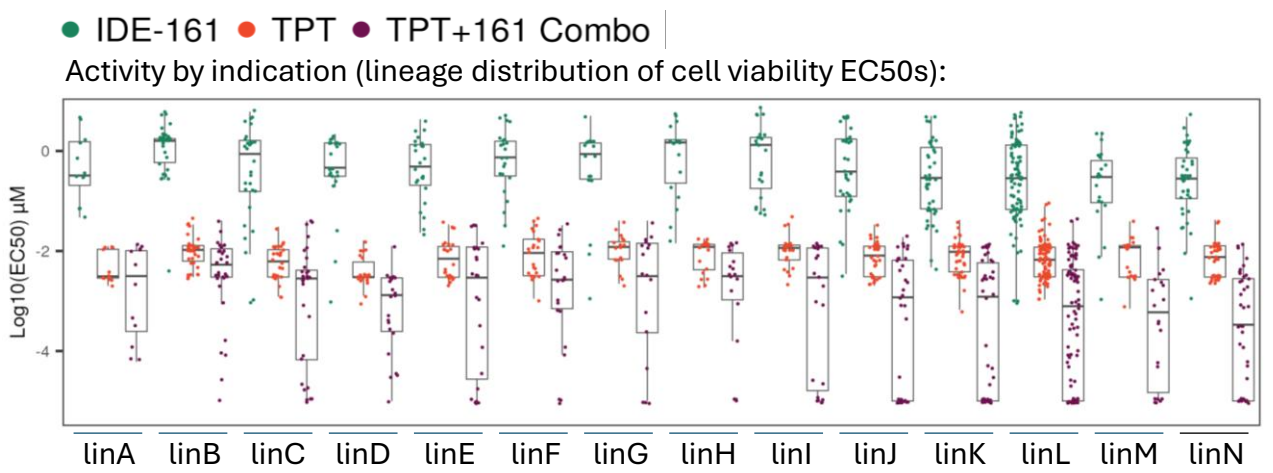
- TOP1 ADCs cause DNA damage by trapping TOP1 in parylated DNA lesions that, if not repaired, result in genetic instability and cell death[1]
- TOP1 lesion repair requires PARG-dependent deparylation
- PARG inhibition in the presence of TOP1 inhibition results in the rapid accumulation of TOP1 lesions
- IDE161 (PARG) induced accumulation of TOP1 lesions amplifies the efficacy of TOP1 ADCs in preclinical models



* Figure adapted from Sun et al, Nat Com, 2021

IDE161/topotecan demonstrate potent activity across majority of cancer cell models[2]





(1) Sun et al, Nat Com, 2021; (2) Munoz et al, AACR 2025
TOP1cc = Topoisomerase I cleavage complex, TPT = topotecan

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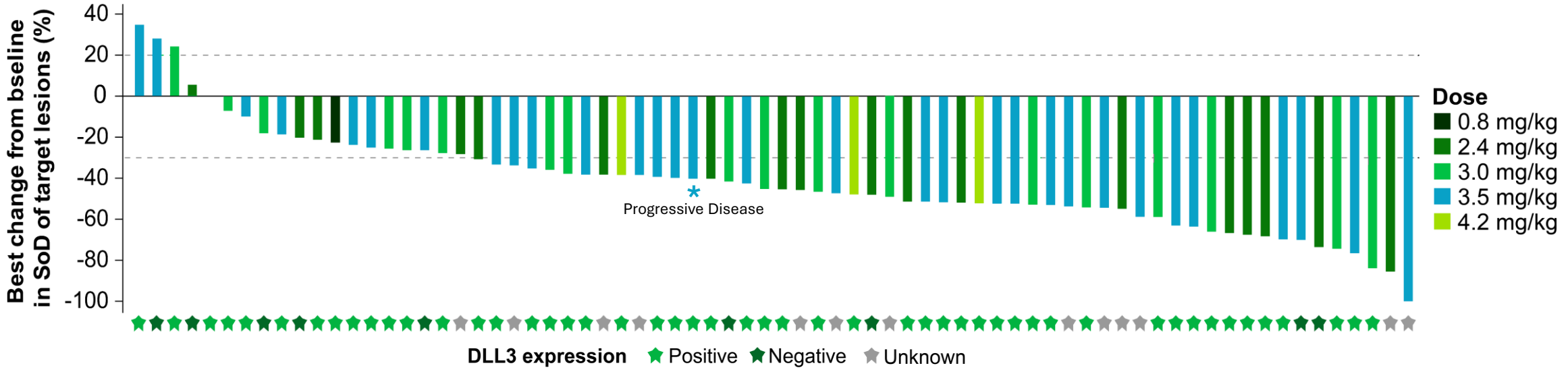
IDE849 (SHR-4849) Demonstrated Compelling Initial Efficacy in SCLC

Robust responses observed across multiple expansion doses tested in Phase 1 study

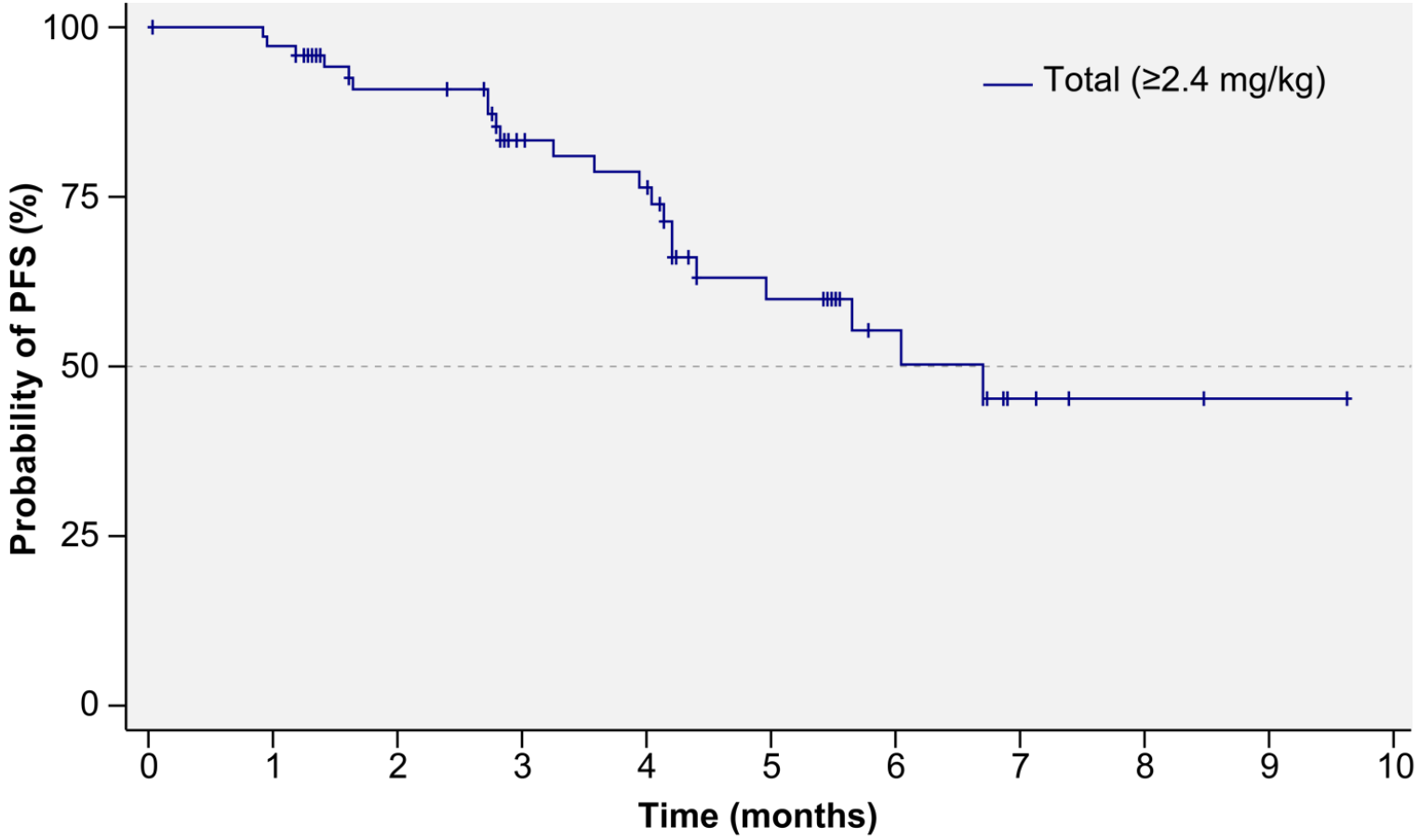


	2.4 mg/kg		3.0 mg/kg		3.5 mg/kg		4.2 mg/kg		Total (≥2.4 mg/kg)	
	2L Setting (n=10)	All (n=19)	2L Setting (n=8)	All (n=18)	2L Setting (n=16)	All (n=31)	2L Setting (n=1)	All (n=3)	2L Setting (n=35)	All (n=71)
ORR, n (%)	8 (80.0%)	14 (73.7%)	6 (75.0%)	12 (66.7%)	12 (75.0%)	23 (74.2%)	1 (100.0%)	3 (100.0%)	27 (77.1%)	52 (73.2%)
Confirmed ORR, n (%)	7 (70.0%)	11 (57.9%)	2 (25.0%)	4 (22.2%)	11 (68.8%)	16 (51.6%)	1 (100.0%)	3 (100.0%)	21 (60.0%)	34 (47.9%)
Response pending confirmation, n (%)	0	1 (5.3%)	4 (50.0%)	8 (44.4%)	0	1 (3.2%)	0	0	4 (11.4%)	10 (14.1%)
DCR, n (%)	10 (100.0%)	18 (94.7%)	8 (100.0%)	17 (94.4%)	15 (93.8%)	28 (90.3%)	1 (100.0%)	3 (100.0%)	34 (97.1%)	66 (93.0%)

DLL3 positivity was defined as H-score >0
Tumor responses were assessed in all enrolled patients who received study treatment and had baseline and at least one post-baseline efficacy assessment
SoD = sum of diameters, 2L = second-line, ORR = objective response rate, DCR = disease control rate

Phase 1 PFS Data in SCLC Patients Treated with IDE849 (SHR-4849)

Encouraging preliminary evidence of durability across all lines of treatment



	Total (≥2.4 mg/kg)	
	2L Setting (n=42)	All (n=86)
Events, n (%)	8 (19.0%)	22 (25.6%)
Median months	NR	6.7
3-month rate (%)	93.3%	83.3%
6-month rate (%)	59.0%	55.3%

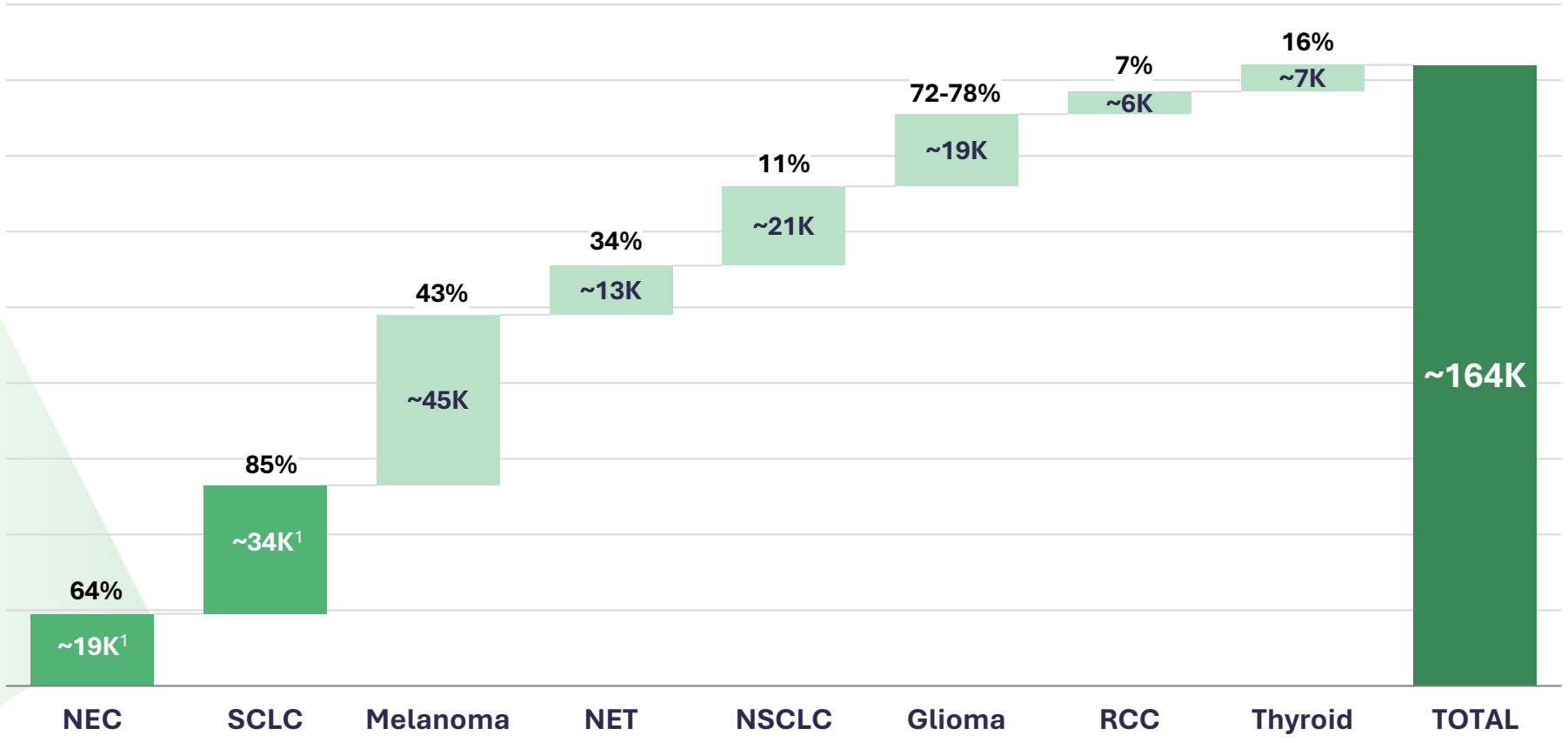
NR = not reached

DLL3 Expression is Upregulated in a Broad Range of Solid Tumor Types

~164,000 potential addressable patients in the U.S. alone

Estimated DLL3+ Frequency by NEC Subtype	
Pulmonary Large-Cell NEC	63%
Gastroentero-pancreatic NEC	44%
Merkel Cell Carcinoma	87%
Neuroendocrine Prostate	77%
Transformed 2L NSCLC	86%

Estimated DLL3-Positive U.S. Annual Incidence by Tumor Type



IDEAYA is prioritizing trials in NEC and SCLC

(1) Based on 100% as no need to stratify NEC or SCLC population

Sources: SEER 2025, Rojo, F., at al. Lung Cancer. 2020;147:237–243. Lozada, JR, at al. Expression Patterns of DLL3 across NENs Cancer Res Commun. 2025 Feb;1;5(2):318-326. Schmitt, M.et al. DLL3 Expression in NEC and NETs. Endocr Pathol 36, 9 (2025), Tanaka, K., at al. Lung Cancer. 2018 Jan;115:116-120. Yao, J., at al. The Oncologist. 2022;27:940–951. Ali, G., at al. Front Oncol. 2021;11:729765. Song, H., at al. Exp Ther Med. 2018;16:53-60.

NET = neuroendocrine tumor, RCC = renal cell carcinoma

IDE849 (SHR-4849) Clinical Development Overview

Potential monotherapy and combination opportunities in multiple DLL3-overexpressing tumors

Preliminary Strategy

- Pursue potential mono-therapy AA path in 2L+ SCLC and/or 2L+ NEC
- Evaluate clinical combinations, including with SOC, in 1L SCLC
- Evaluate DLL3+ basket trial as monotherapy
- Target to enhance durability with IDE849 + IDE161/PARG combo

Target Clinical Development Plan

IDE849

Monotherapy Dose Escalation and Expansion

- Dose Escalation
- Expansion Cohort: SCLC
- Expansion Cohort: NEC

IDE849

Combination with IDE161 (PARG)

(to be initiated once monotherapy expansion cohorts are underway)

- IDE849 + IDE161: SCLC and NEC

SOC = Standard of Care

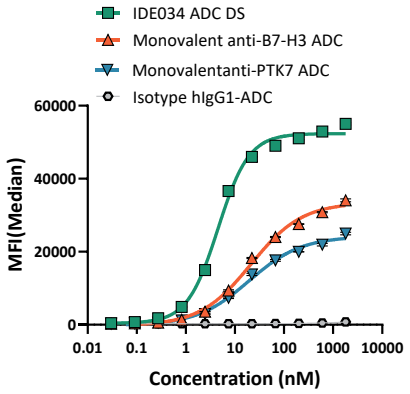
IDE034 is a Potential First-in-Class Phase 1 B7H3/PTK7 TOP1 Bispecific ADC

Dual tumor-antigen binding to maximize tumor-specific PARG combination benefit in multiple solid tumors

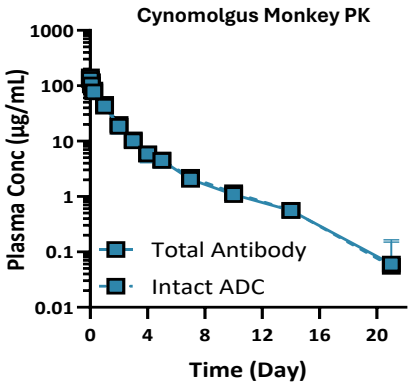
αB7H3 αPTK7

Human IgG1
TOP1: BLD1102
DAR=8
Internalization-dependent payload release

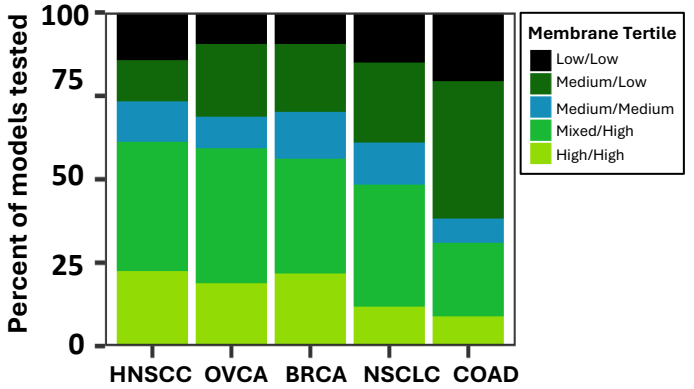
Enhanced Tumor Cell Binding vs Matched Monospecific ADCs



Robust Linker-Payload Stability in Circulation



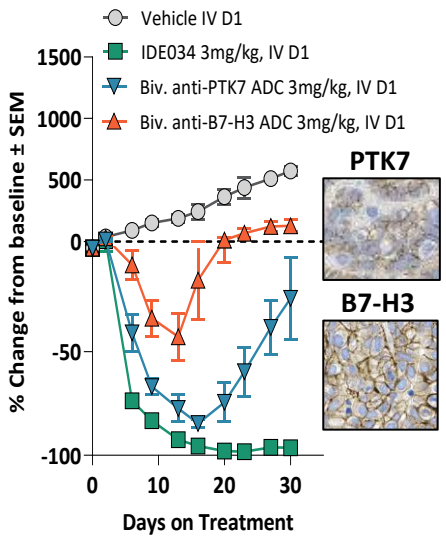
Extensive Dual B7H3/PTK7 Surface Protein Expression Across Tumor Types



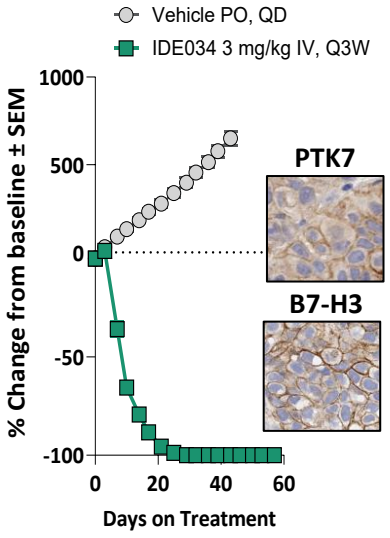
- Dual-antigen dependence delivers potential efficacy and safety advantages over B7H3 or PTK7 mono-specific ADCs
- Large dual-positive indication opportunities
- Minimal dual-antigen expression in normal tissues
- Targets tumor-initiating cells to potentially inhibit resistance

IDE034 has demonstrated exceptional preclinical anti-tumor activity across priority indications

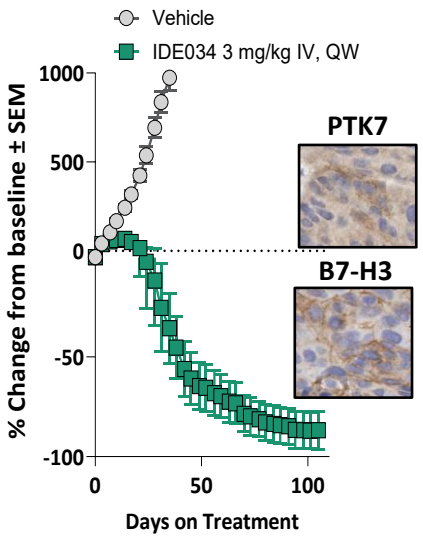
Breast- TNBC PDX



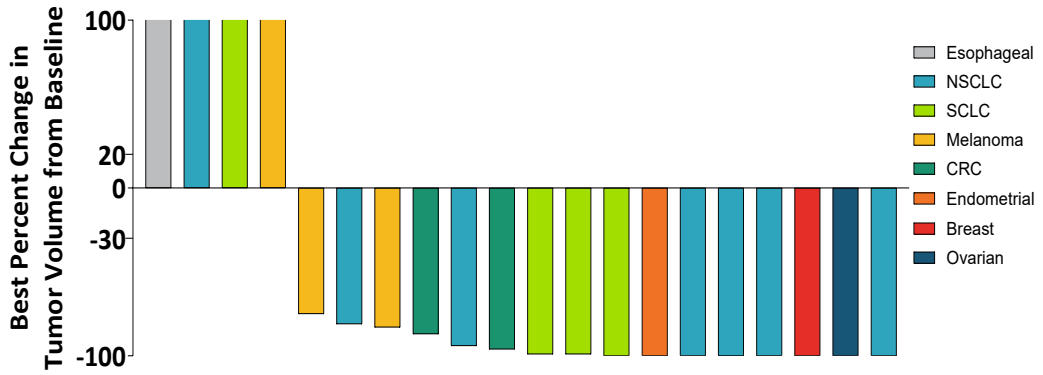
Ovarian PDX



Colorectal PDX



B7H3/PTK7 Dual Positive CDX (Inferred from baseline mRNA Expression)



IDE397 is the Backbone of 3 Combination Strategies for MTAP-Deleted Solid Tumors

MAT2A is central to support protein methylation and DNA repair in MTAP$^{-/-}$ tumors

Combination strategies designed to amplify metabolic liabilities and genomic instability conferred by loss of MTAP:

1) MAT2A + TOP1 ADC

IDE397 *(MAT2A)*	***IDE034**** *(B7H3/PTK7)*

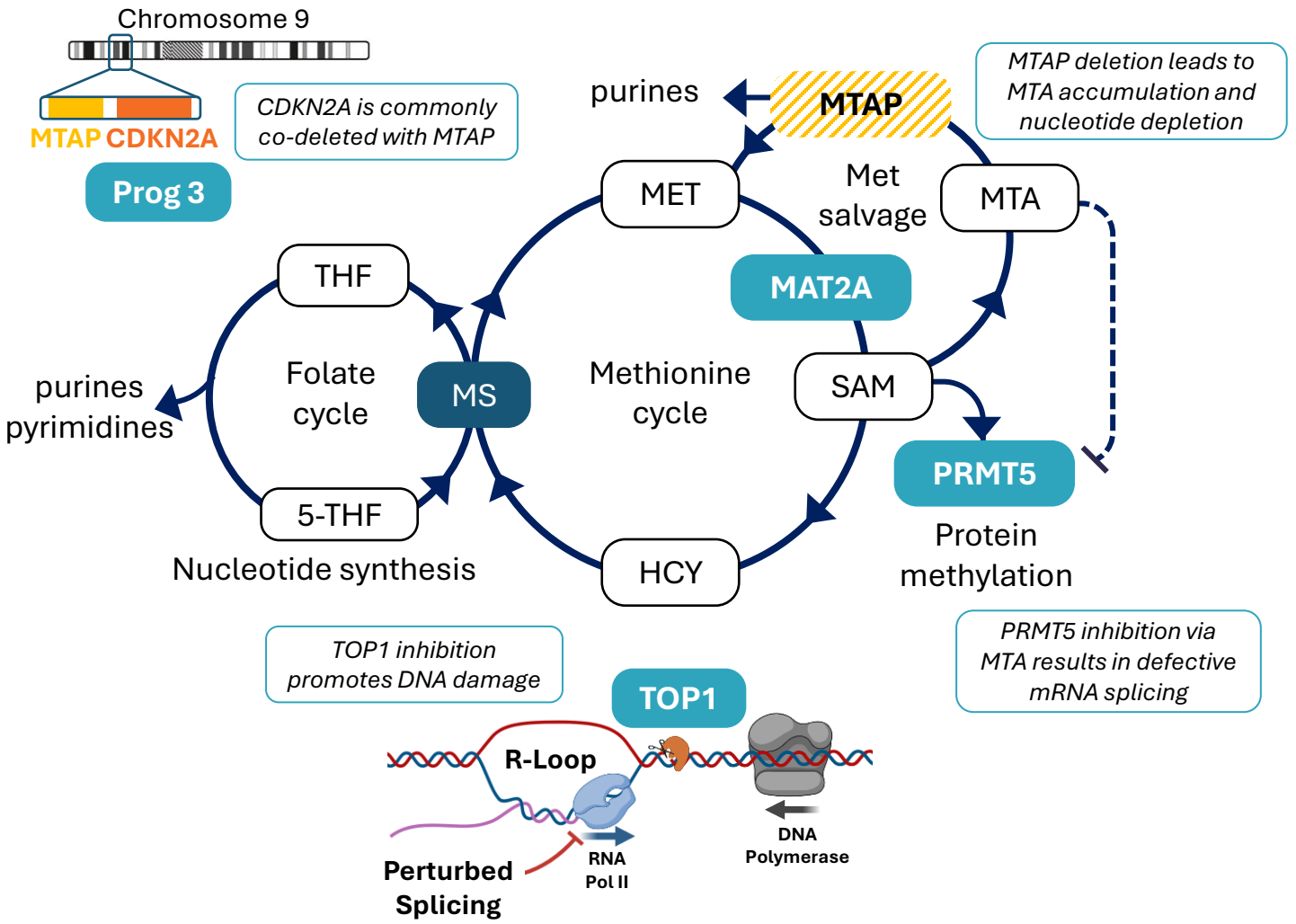
** Potential example*

2) MAT2A + PRMT5

IDE397 *(MAT2A)*	***IDE892*** *(PRMT5)*

3) MAT2A + co-alterations of MTAP

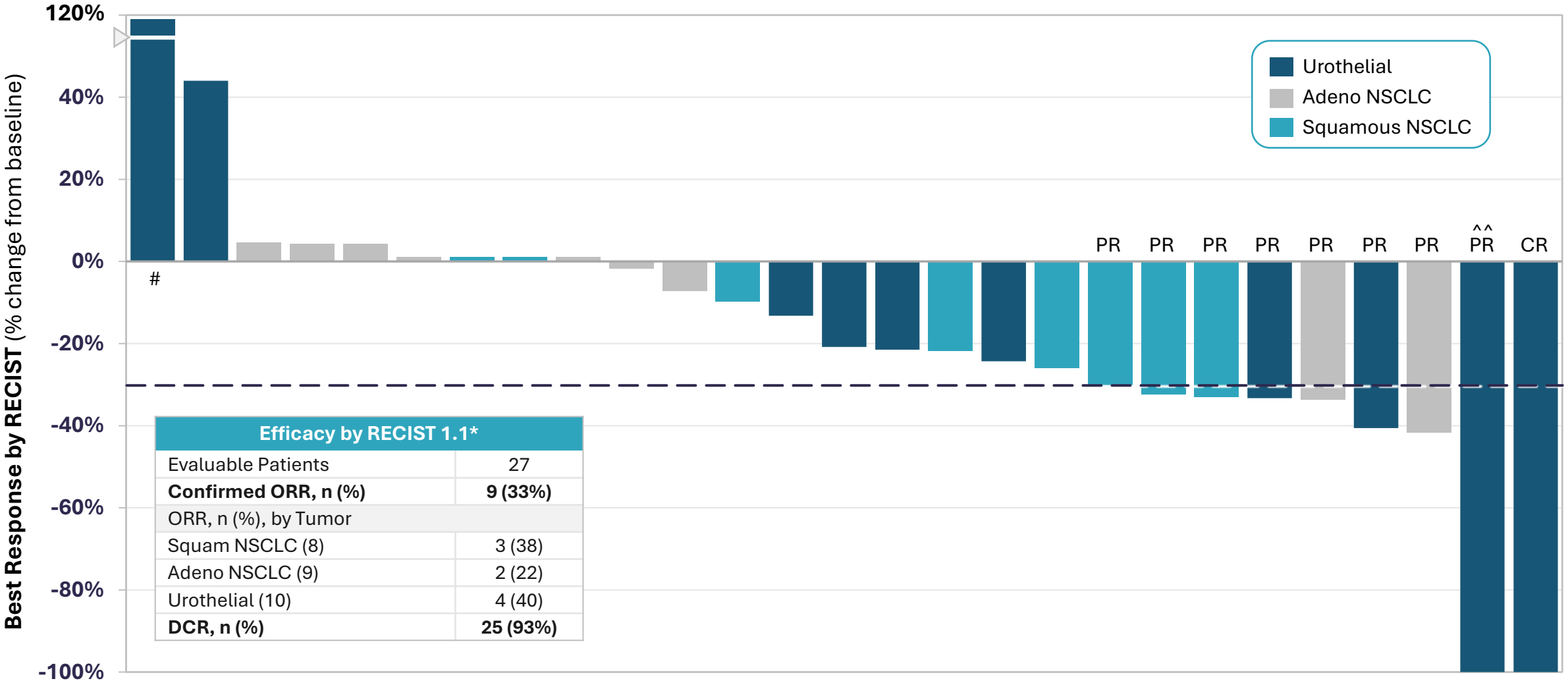
IDE397 *(MAT2A)*	***CDKN2A*** *(preclinical)*



MTAP = methylthioadenosine phosphorylase, MAT2A = methionine adenosyltransferase 2a, DC = development candidate, MTA = methylthioadenosine, PRMT5 = protein arginine methyltransferase 5, SAM = S-adenosylmethionine, MET=methionine, HCY=homocysteine, THF= tetrahydrofolate, 5-THF= 5-methytetrahdrofolate; MS= methionine synthase

Phase 1 IDE397 Monotherapy in MTAP-Deleted Lung and Urothelial Cancers

Best response by RECIST 1.1 at 30mg Phase 2 expansion dose

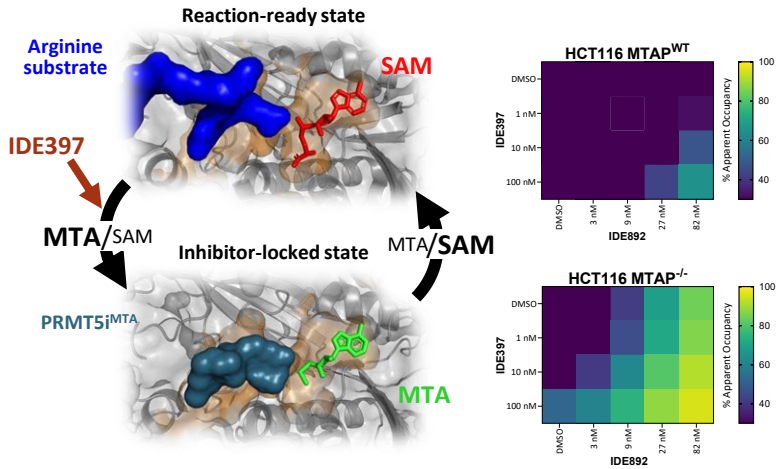


Efficacy by RECIST 1.1*	
Evaluable Patients	27
Confirmed ORR, n (%)	**9 (33%)**
ORR, n (%), by Tumor	
Squam NSCLC (8)	3 (38)
Adeno NSCLC (9)	2 (22)
Urothelial (10)	4 (40)
DCR, n (%)	**25 (93%)**

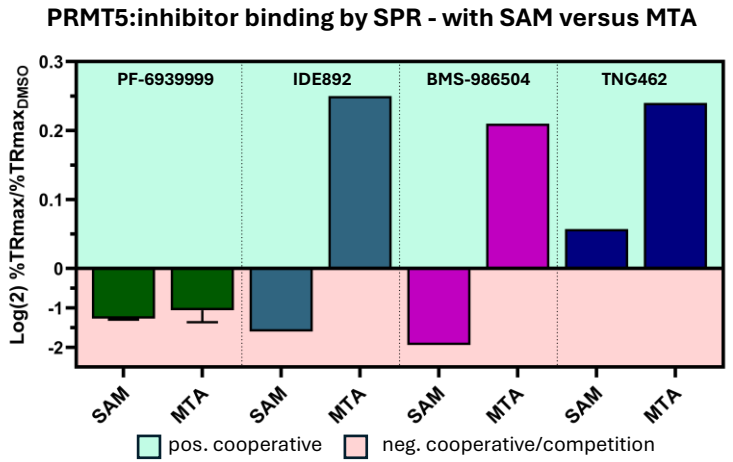
* Evaluable Patients: Treated with >1 cycle (21 days) of IDE397 at the 30 mg expansion dose and with >1 post-baseline scan(s); # Patient received less than 75% of planned dosing prior to the first scan due to unrelated AEs in cycle 2;
^^ PR with -100% best response had complete resolution of the target lesion. Data as of 22AUG2024 data cut off; two patients confirmed response after the data cut

IDE892 (PRMT5) Exhibits Robust Selectivity and Combination Potential with IDE397 in MTAP$^{-/-}$ Preclinical Models

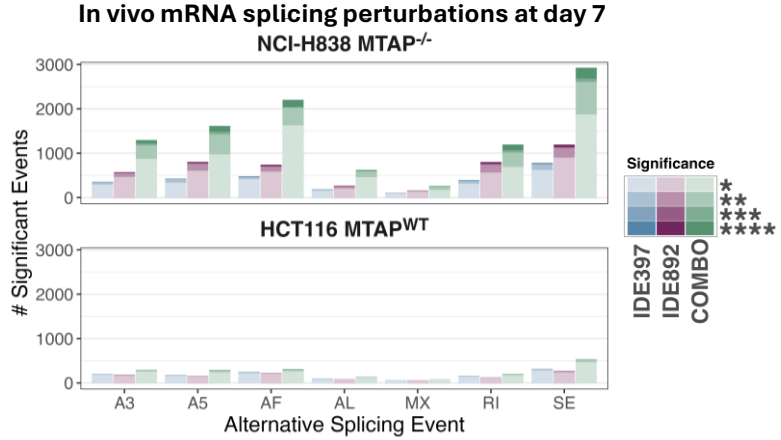
IDE397 enhances a rationally designed PRMT5i^{MTA}



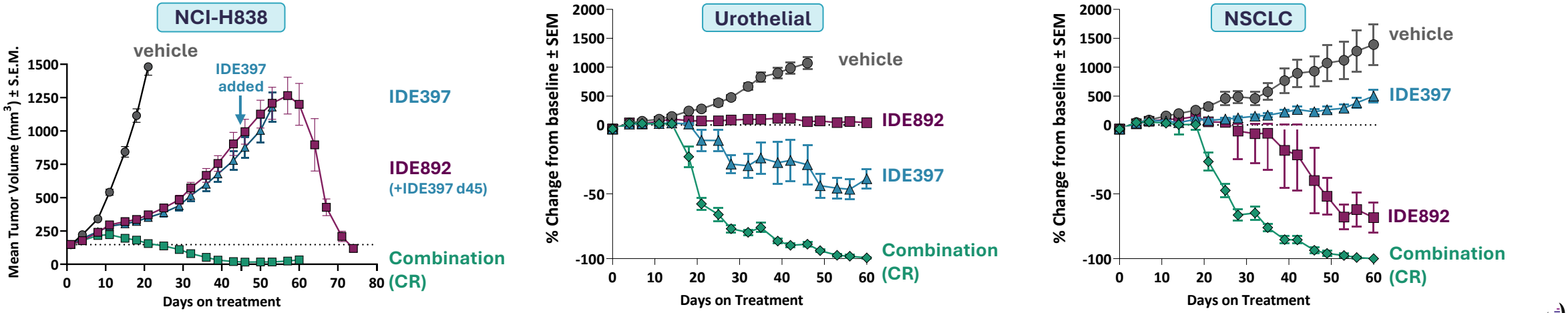
IDE892 displays positive cooperativity with MTA and negative cooperativity with SAM



IDE892/IDE397 combination delivers strong on-target MTAP-selective PD



Strong monotherapy and combination benefit observed in MTAP$^{-/-}$ CDX and PDX models

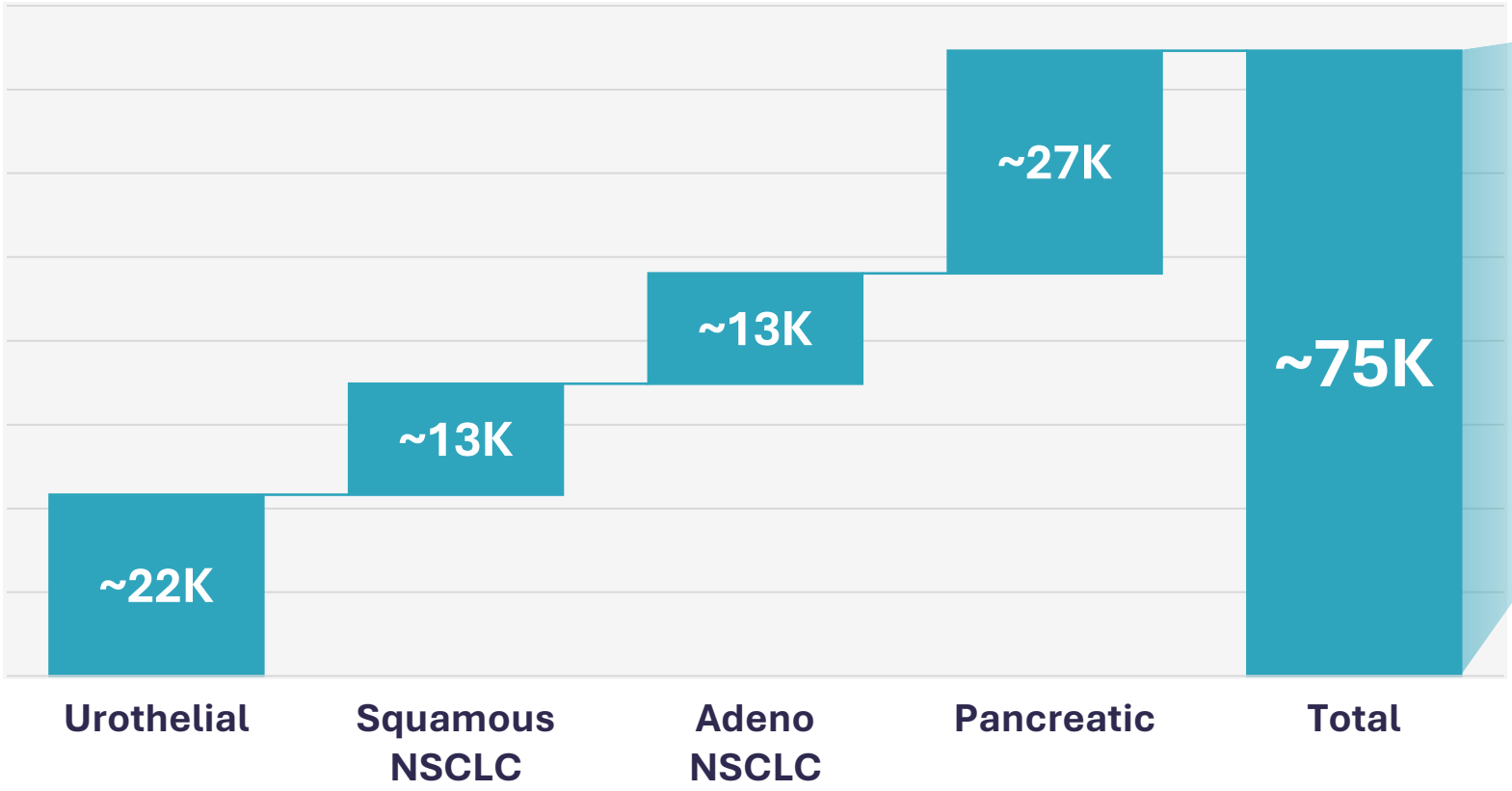


MTAP$^{-/-}$ = MTAP deleted, WT= wild type, CR = complete response. SPR = surface plasmon resonance

There Are No FDA-Approved Therapies for MTAP-Deleted Solid Tumors

IDEAYA's clinical strategy is focused on lung, urothelial and pancreatic cancers

Estimated U.S. Annual Incidence in Priority Tumor Types



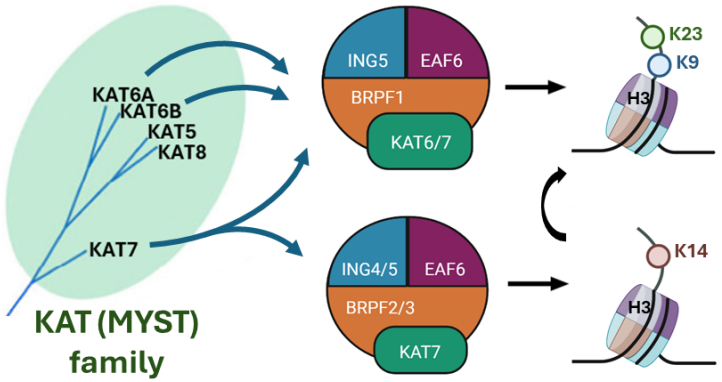
MTAP-Deletion Frequency by Tumor Type[1]	
Urothelial Cancer	**26%**
Squamous NSCLC	**19%**
Adeno NSCLC	**11%**
Pancreatic[2]	**40%**
Head and Neck	14%
Gastric	10%
Squamous Esophageal Cancers	28%

(1) Estimated addressable patient population based on SEER 2024 incidence and MTAP-deletion frequency from TCGA PanCancer Atlas
(2) Estimated frequency up to ~40% based on Gorbokon et. al., Cancers (Basel). 2025 Apr 1;17(7):1205

IDE574 is an equipotent dual KAT6/7 Inhibitor with Broad Potential in Solid Tumors

IDE574: potent dual KAT6/7 inhibitor to overcome paralog bypass

Histone acetyltransferases KAT6A/B and KAT7 collaboratively promote tumorigenic gene expression programs

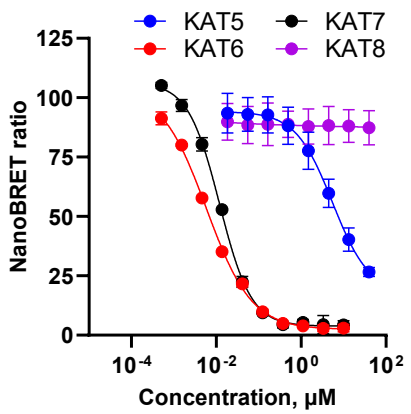


Adapted from White et al. Trends in PS 2024; LaLonde et al. Genes & Dev. 2013

Therapeutic Opportunity

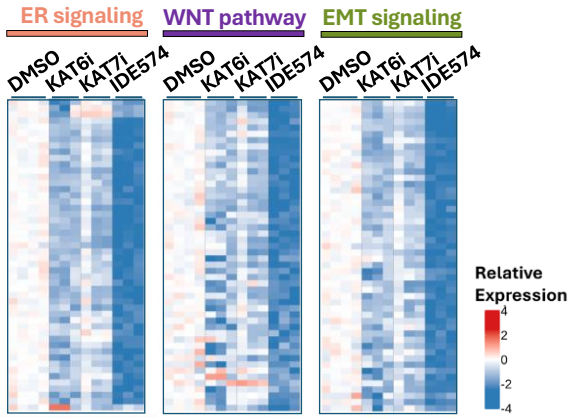
- Drug lineage-survival oncogenic TF networks
- Disrupt tumor initiating cell renewal
- Block evolution of drug-tolerant persister cells

IDE574: BRET engagement assay



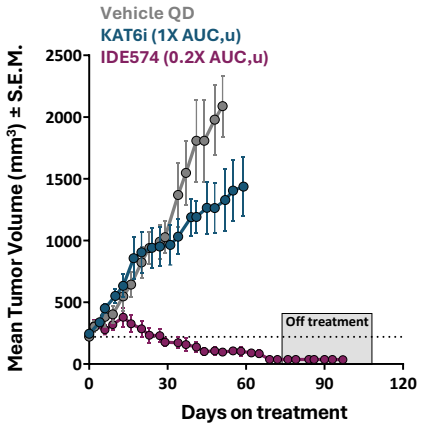
mBC PDX ST941C cell line, RNA-seq

IDE574 delivers deep and sustained suppression of key oncogenic and drug resistance pathways

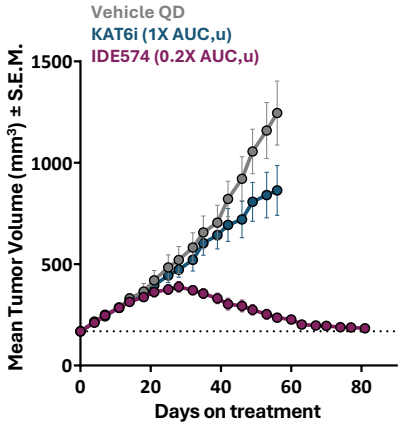


Robust mono and combo opportunity across mBC, Lung, and CRC

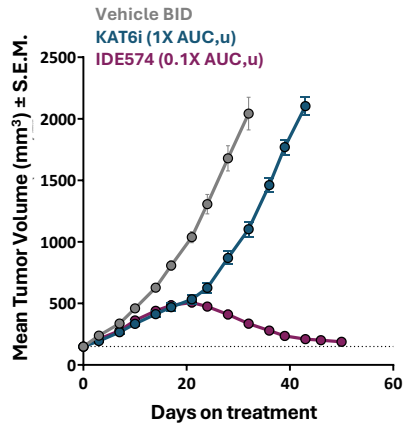
HR+ mBC (PDX ST941, ESR1 Y537S)



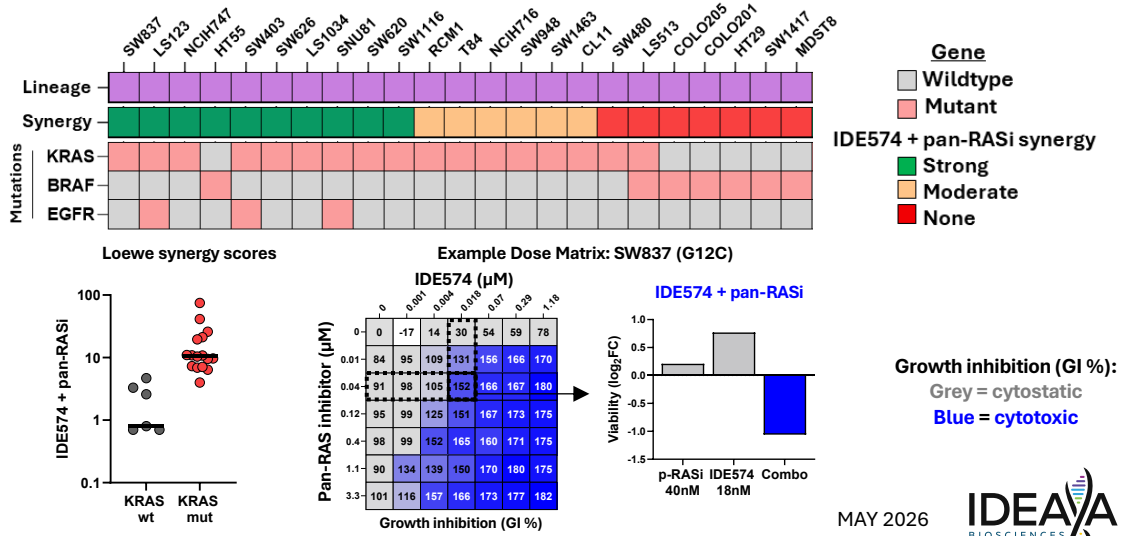
NSCLC (PDX, KEAP1/STK11 mut)



CRC (CDX, MSS/TP53 mut)



IDE574 demonstrates synergy with pan-RAS inhibition to kill CRC cells



HR+ mBC = hormone receptor positive metastatic breast cancer, KAT6i = clinical KAT6 selective inhibitor with preclinical exposure @RP2D

IDEAYA Biosciences: Building a Leading Precision Medicine Oncology Company

Clinical stage pipeline advancing across multiple solid tumor indications

Anticipated 2026 Pipeline Milestones

		Q1 2026	Q2 2026	H2 2026
Darovasertib/ Uveal Melanoma (UM)		✓ **Metastatic UM (HLA*A2-neg)** Topline OptimUM-02 data	✓ **Metastatic UM (HLA*A2-pos)** Complete OptimUM-01 enrollment **Metastatic UM (HLA*A2-neg)** OptimUM-02 ASCO presentation **Adjuvant UM** Initiate OptimUM-11 trial	**Metastatic UM (HLA*A2-pos)** OptimUM-01 data update **Metastatic UM (HLA*A2-neg)** NDA submission **Neoadjuvant UM** OptimUM-09 data update
ADC+DDR Combos	*IDE849 (DLL3)*			**Phase 1 data update (Hengrui)** SCLC, NEC, monotherapy **Phase 1/2 data (IDEAYA)** SCLC, NEC, monotherapy **Initiate Registrational Trial** DLL3+ tumors, monotherapy (YE '26)
	IDE034 (B7H3/PTK7)	✓ **Phase 1 FPI (Monotherapy)** Solid tumors		**Phase 1 Update (Monotherapy)** Solid tumors (YE '26)
	IDE161 (PARG)	✓ **Phase 1 FPI (+IDE849 combo)** SCLC, NEC, DLL3+		
MTAP Pathway	*IDE892 (PRMT5)*	✓ **Phase 1 FPI (Dose escalation)** NSCLC, PDAC	**Phase 1 FPI (+IDE397 combo)** Solid tumors (mid-2026)	
KAT6/7	*IDE574 (KAT 6/7)*	✓ **Phase 1 FPI (Monotherapy)** Solid tumors		

Highlights

Darovasertib commercial readiness activities ongoing

~$973M in cash and equivalents with runway into 2030[1]

Strong partnerships

SERVIER

HENGRUI

NASDAQ: IDYA

(1) Includes aggregate of approximately $972.9 million of cash, cash equivalents and marketable securities as of Mar 31, 2026, as detailed on IDEAYA's Form 10-Q filed with the U.S. SEC; runway projections based on current operating plan

FPI = first-patient-in, RWE = real world evidence

May 2026

Improving Lives Through Transformative Precision Medicines

Corporate Presentation

IDEAYA BIOSCIENCES

NASDAQ: **IDYA**