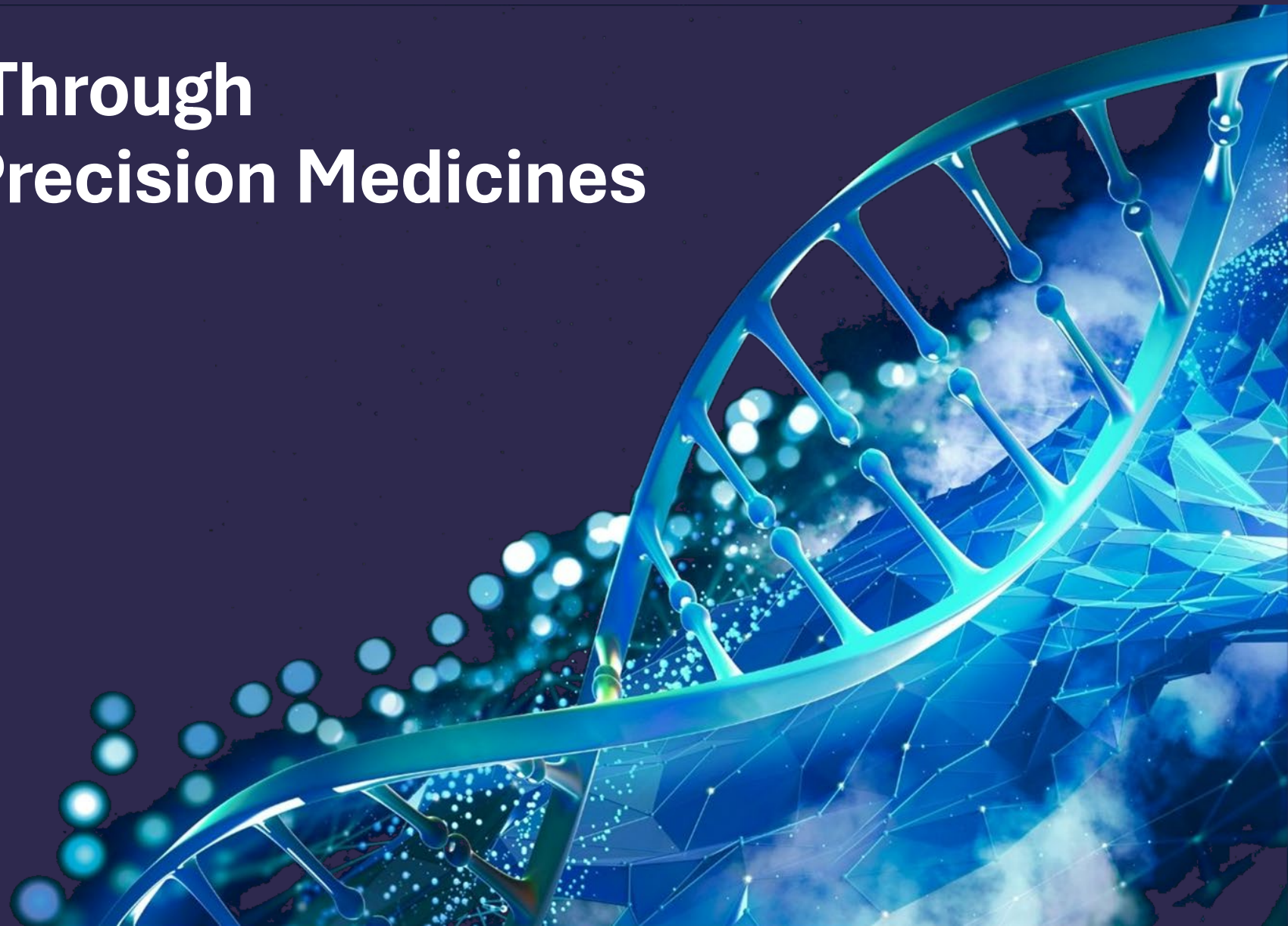
March 2026

Improving Lives Through Transformative Precision Medicines

Corporate Presentation

IDEAYA BIOSCIENCES

NASDAQ: **IDYA**



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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.

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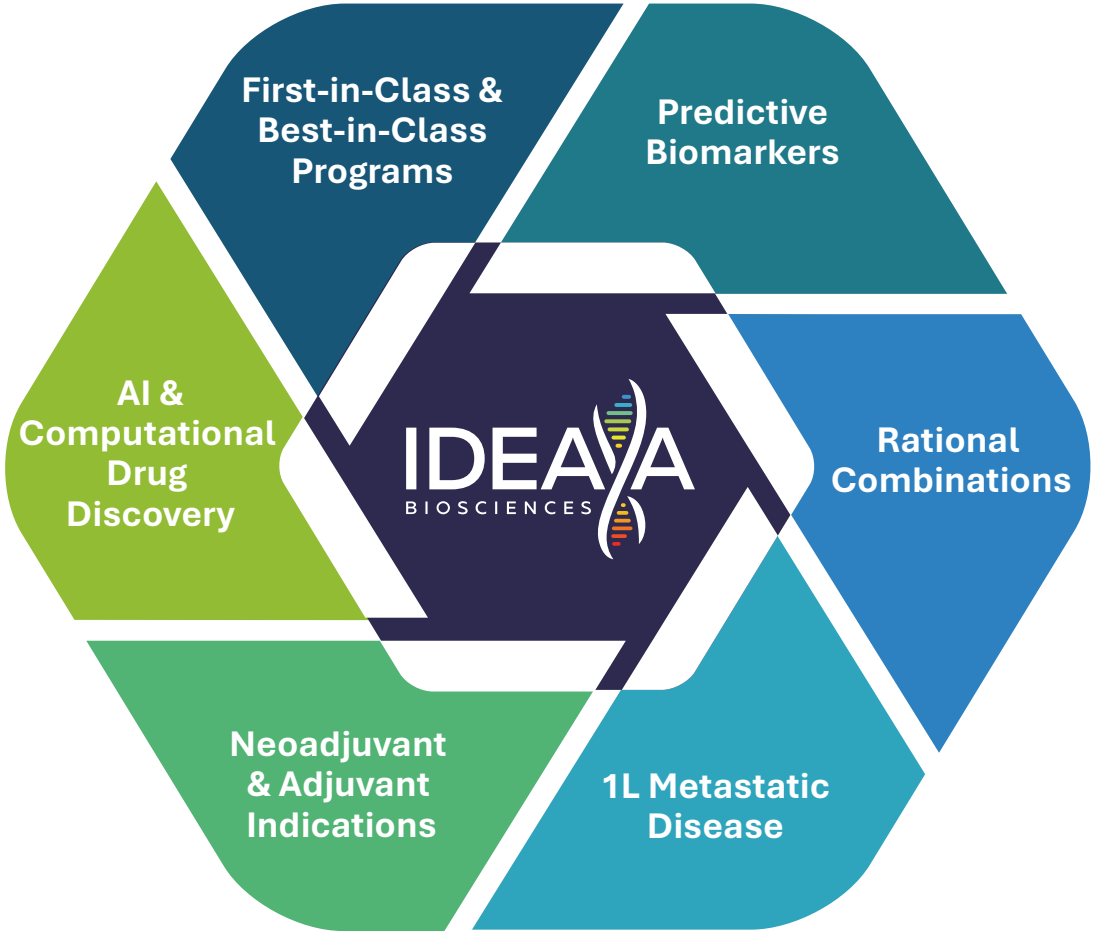
IDEAYA's Vision is to Build a Leading Precision Medicine Oncology Company

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

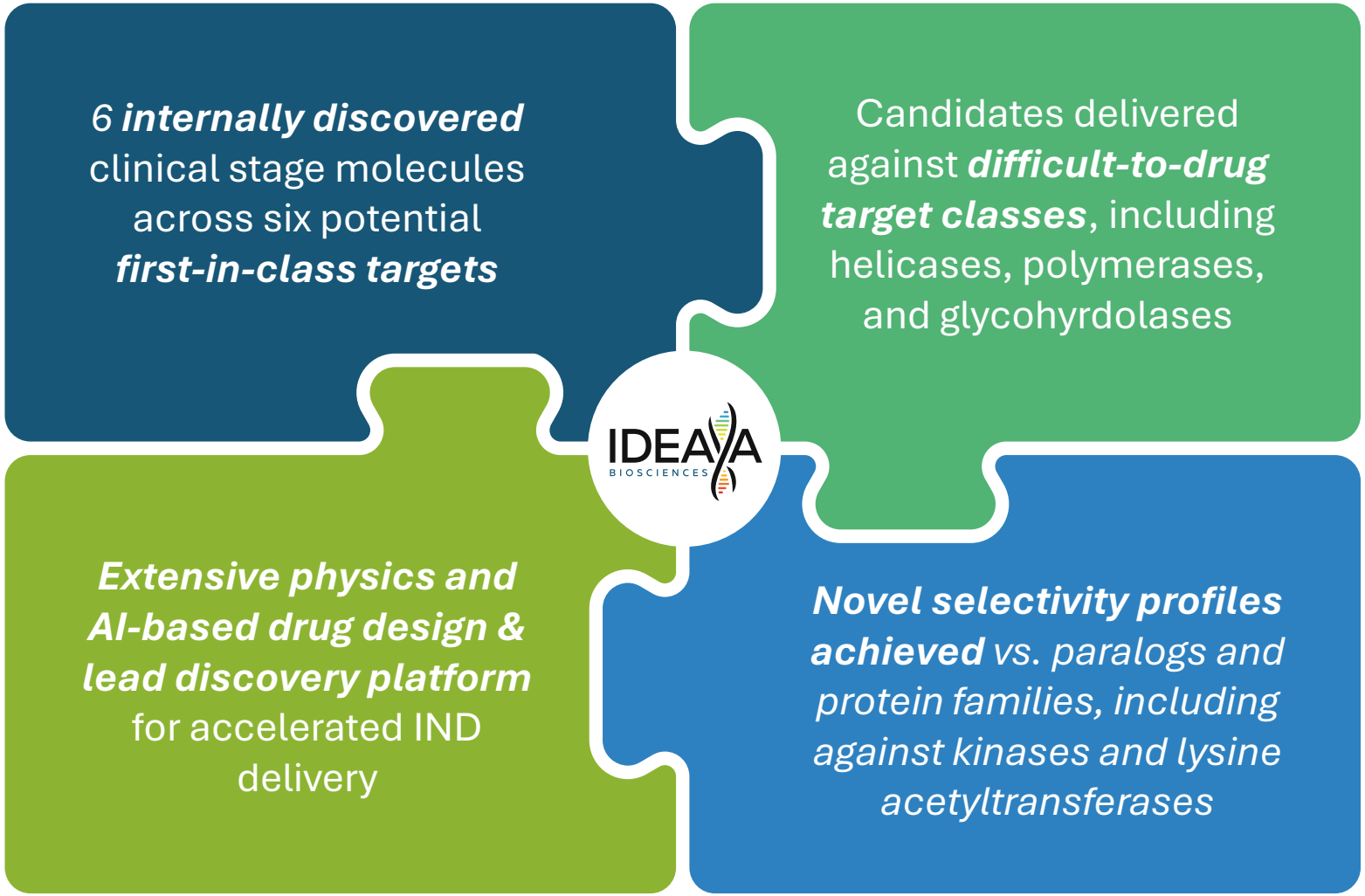
Next Gen Therapies

Target tumor heterogeneity to enhance durability

1L = 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



Core IDEAYA Drug Discovery Capabilities

CRISPR-based target and biomarker discovery

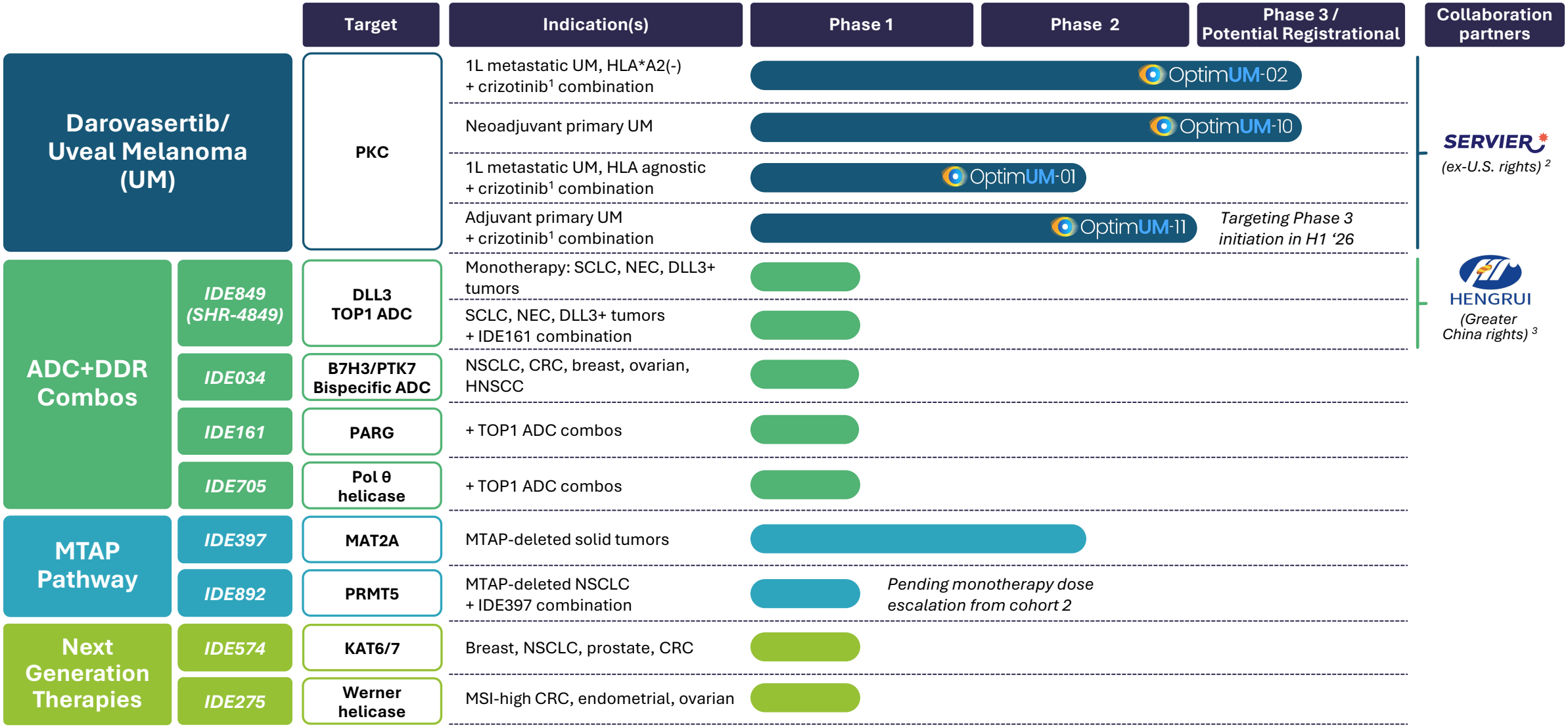
Chemigenomic-enabled translational research

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

Deep 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

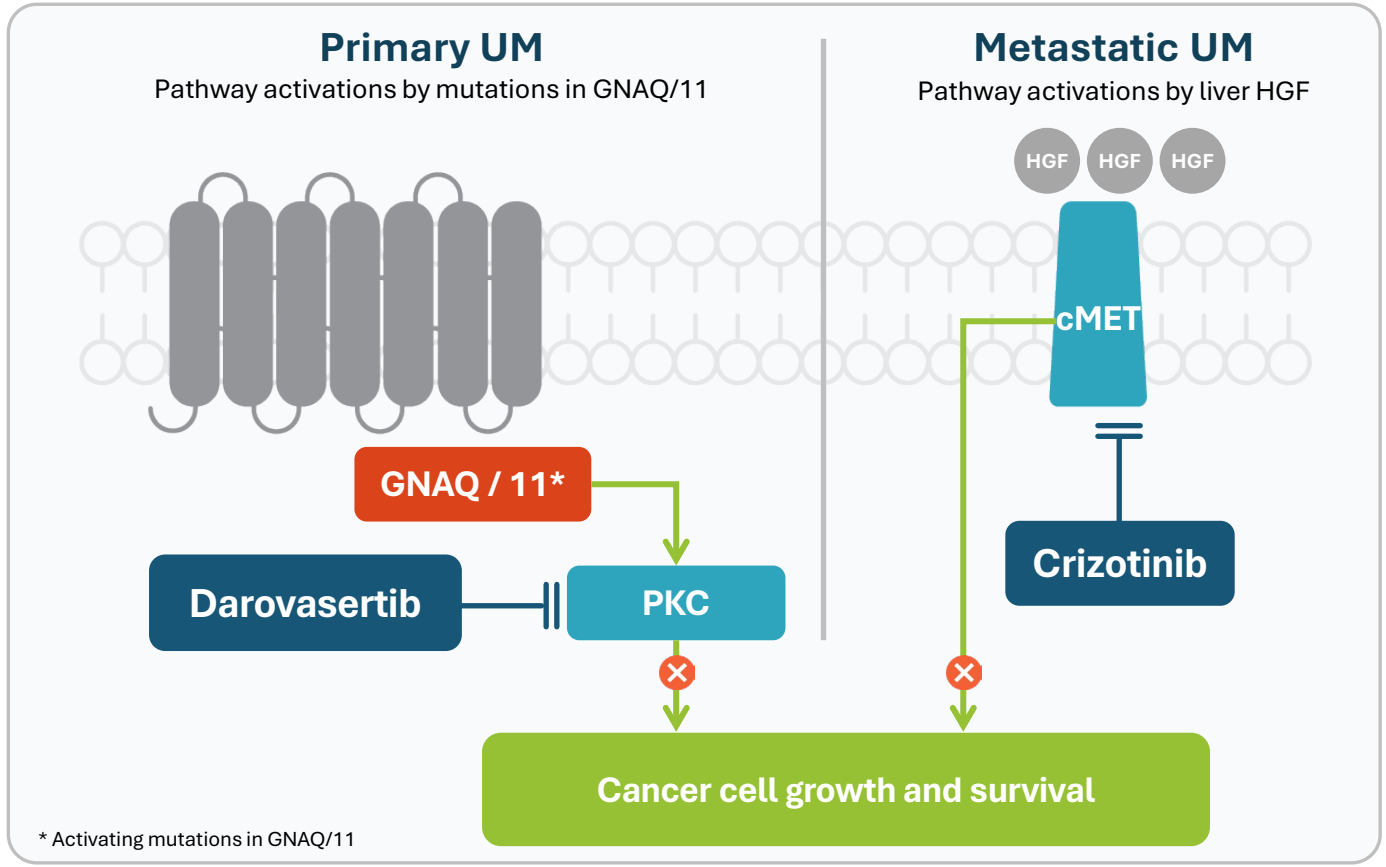
Off-label use of checkpoint inhibitors has limited efficacy

(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

- Oral, selective inhibitor of **PKC, the key oncogenic pathway** in >95% of UM patients
- Activating **mutations in GNAQ/11** proteins result in PKC overactivation and tumor cell growth
- 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

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

Robust clinical development plan across the uveal melanoma patient journey

Diagnosis → **Primary local therapy** → **Metastasis** → **Progression** →

Primary Uveal Melanoma

Neoadjuvant | **Adjuvant**

Save the eye, preserve vision

Prevent relapse or delay progression

Metastatic Uveal Melanoma

HLA*A2-negative | **HLA*A2-positive**

Prolong survival and quality of life

NO APPROVED THERAPIES

Darovasertib monotherapy (Phase 3)

Shrink tumor prior to:
- Enucleation
- Plaque brachytherapy

OptimUM-10

Target enrollment completion H1'27

Darovasertib + crizotinib (Phase 3)

Manage tumor burden and potential relapse post-primary local therapy

OptimUM-11

Trial initiation H1'26

Darovasertib + crizotinib (Phase 2/3)

PFS: supports U.S. AA filing

OS: supports full approval

OptimUM-02

Enrollment complete

LIMITED THERAPIES

Darovasertib + crizotinib (Phase 2)

Includes HLA*A2+ for pot'l NCCN/Compendia listing

OptimUM-01

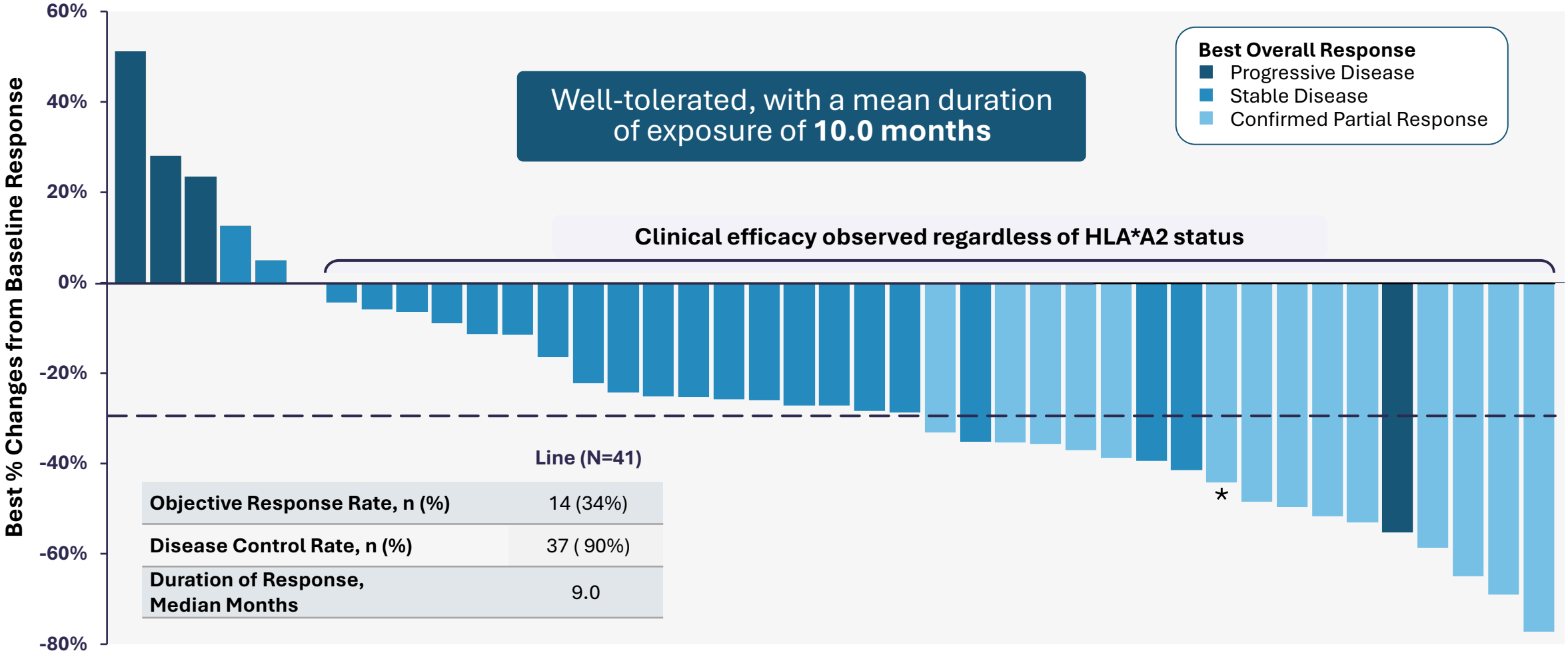
Currently enrolling

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

OptimUM-01 Darovasertib + Crizotinib Continue to Drive Robust Responses

Single-arm trial demonstrated favorable ORR versus historical trials and meta-analysis in mUM



	Line (N=41)
Objective Response Rate, n (%)	14 (34%)
Disease Control Rate, n (%)	37 (90%)
Duration of Response, Median Months	9.0

(1) Nathan P, et al, NEJM 2021; 385:1196-1206; Wang Y, et al, Front. Oncol., 14 October 2025; Volume 15. Single-digit % ORR reported in historical MUM trials and meta-analyses. Cross trial comparisons are not being made and for informational purposes only

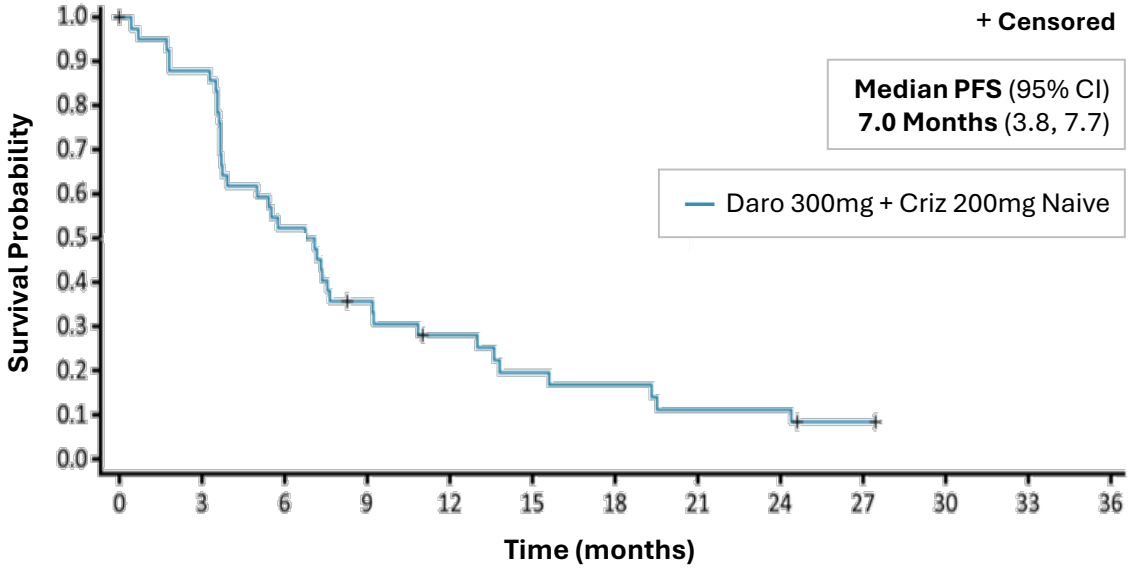
* By RECIST v1.1, patient had target lesion response but progression detected with new lesions and non-target lesions. ORR = objective response rate

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

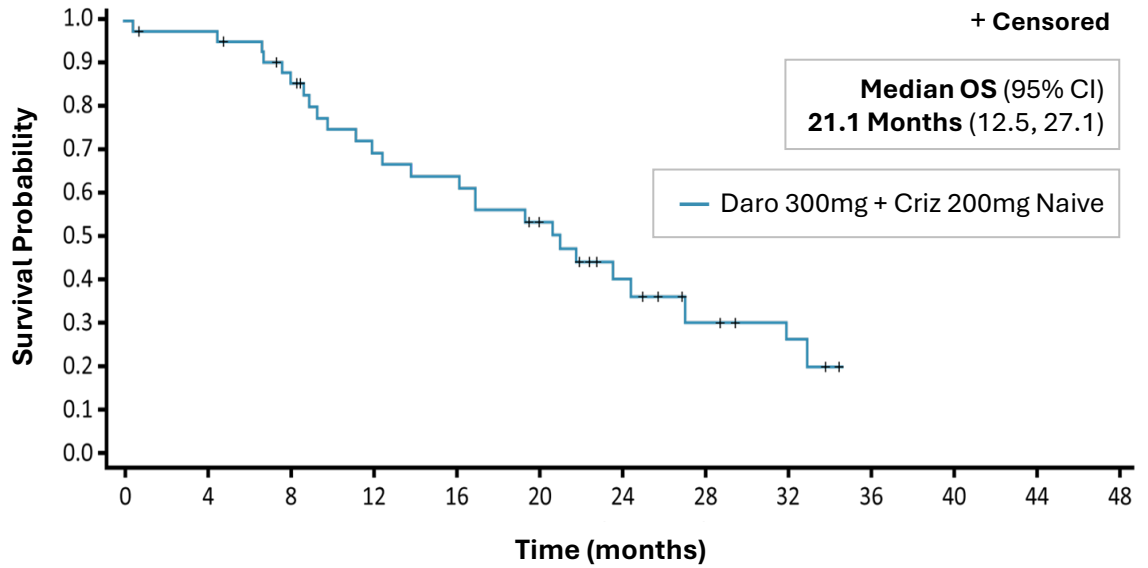


mPFS of 7.0 months
(95% CI: 3.8, 7.7, median follow-up 25 months)

Historical mPFS of 2.8 months[1]

Consistent with 7.1 months previously reported at ESMO 2023

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

IDEAYA BIOSCIENCES

OptimUM-02 Pivotal Phase 2/3 Trial in HLA*A2-Negative mUM

Pursuing approval of the first systemic therapy for an underserved metastatic population

Trial Design

Multi-arm, Multi-stage, Open-label Phase 2/3 trial

(N=437) Full enrollment complete in Dec 2025*

2:1 Randomization

Patients receive darovasertib + crizotinib

300mg BID darovasertib

200mg BID crizotinib

Or

Patients receive investigator's choice of:

- *Pembrolizumab*
- *Ipilimumab + nivolumab*
- *Dacarbazine*

Clinical Endpoints

Primary:

- PFS per RECIST 1.1 (BICR) for potential U.S. accelerated approval filing
- OS for potential U.S. full approval filing

Secondary:

- PFS per RECIST 1.1 (investigator)
- ORR per RECIST 1.1
- DOR per RECIST 1.1
- QoL via EORTC QLQ-C30 questionnaire
- General health status via EQ-5D questionnaire
- Safety

Randomized PFS results to enable a potential U.S. accelerated approval filing

Source: clinicaltrials.gov (NCT05987332)

* Eligibility criteria included patients 18+ years with HLA*A2-negative mUM with histological/cytological confirmation. Randomized PFS analysis is based on the first 130 PFS events from the intent-to-treat population (ITT) enrolled in the Phase 2b/3 portion of the trial, which comprises approximately 313 patients randomized 2:1 to the treatment versus control arm.

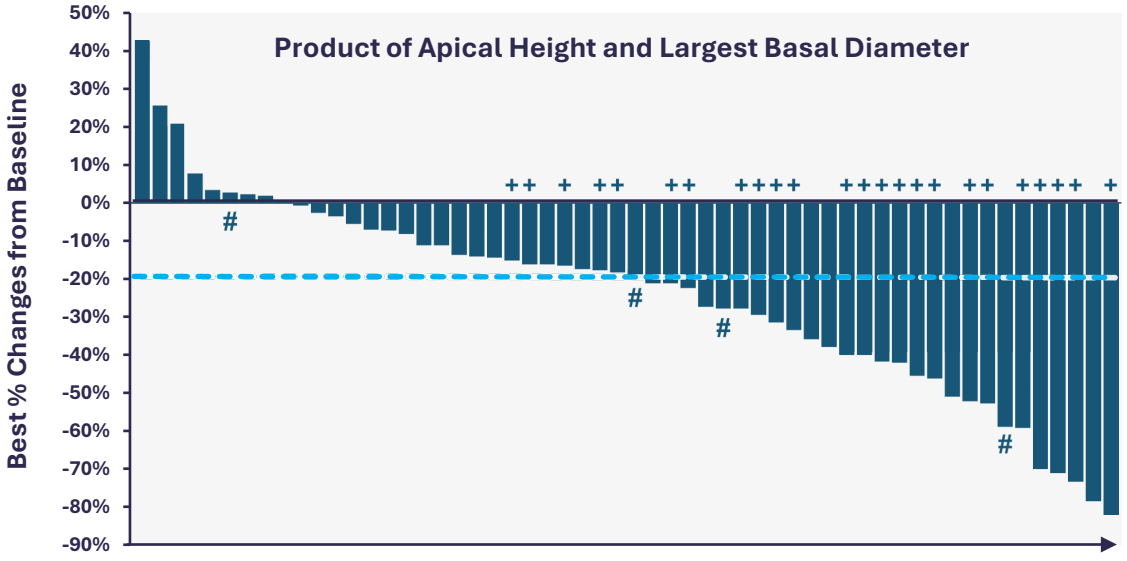
BICR = blinded independent central review, DCR = disease control rate, QoL = quality of life, QLQ = quality of life questionnaire, EORTC = European Organization for Research and Treatment of Cancer, EQ-5D = EuroQol 5 dimension

OptimUM-09 Primary Phase 2 Efficacy Results in Neoadjuvant UM

Robust tumor shrinkage leading to eye preservation and visual improvements

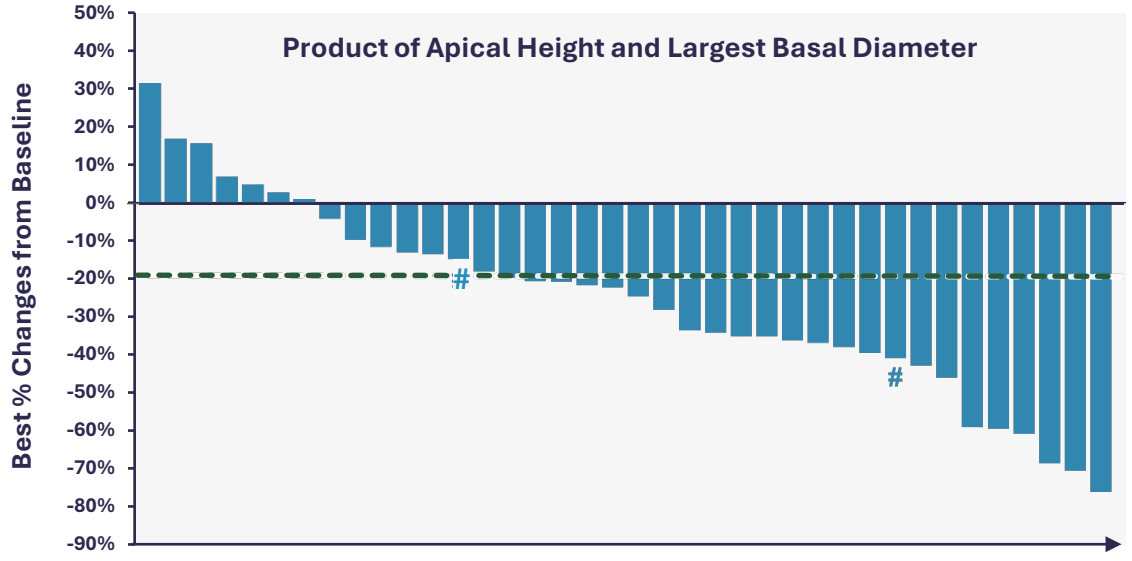
Cohort 1 (Enucleation)

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

OptimUM-10 Pivotal Phase 3 Trial in Neoadjuvant UM

Evaluating the ability of darovasertib to preserve eyes and protect long-term vision

Trial Design

Multi-center, Open-label, Phase 3 Study

2:1 Randomization

Enucleation *(N=120)**

- **Neoadjuvant darovasertib** *300mg BID up to 6 months* → **Primary Local Therapy** *(if needed)*
- **Immediate Enucleation (standard of care)**

Plaque Brachytherapy *(N=330)**

- **Neoadjuvant darovasertib** *300mg BID up to 6 months* → **Radiation Therapy**
- **Radiation Alone (standard of care)**

Clinical Endpoints

Primary:

- Enucleation Cohort: Eye preservation rate >10% with 95% CI
- Plaque Brachytherapy Cohort: Proportion of patients with ≥ 15-letter BCVA loss

Secondary required for approval:

- Event-Free Survival (no detriment) pooled from both cohorts

Additional Secondaries:

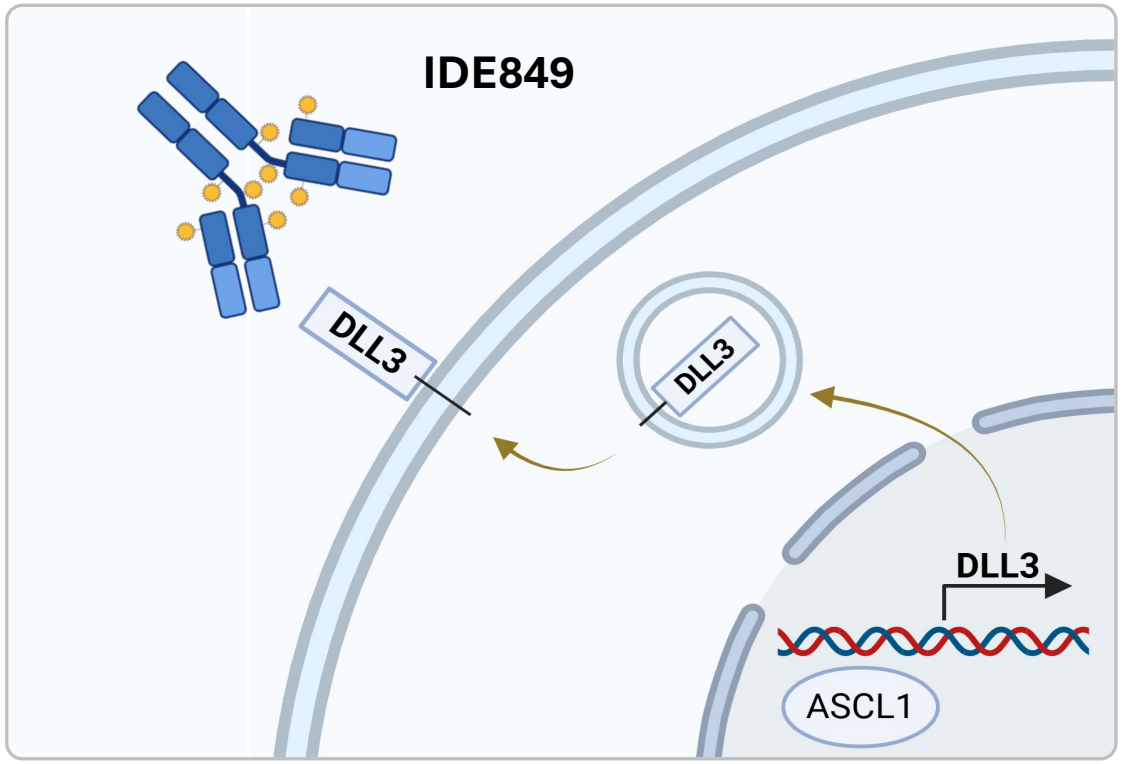
- Overall Response Rate; Radiation dose reduction to key visual structures; Visual acuity outcomes; Quality of life measures; Safety and tolerability

Source: clinicaltrials.gov (NCT07015190)

*Planned sample size and treatment duration, CI = confidence interval, BCVA = best corrected visual acuity

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

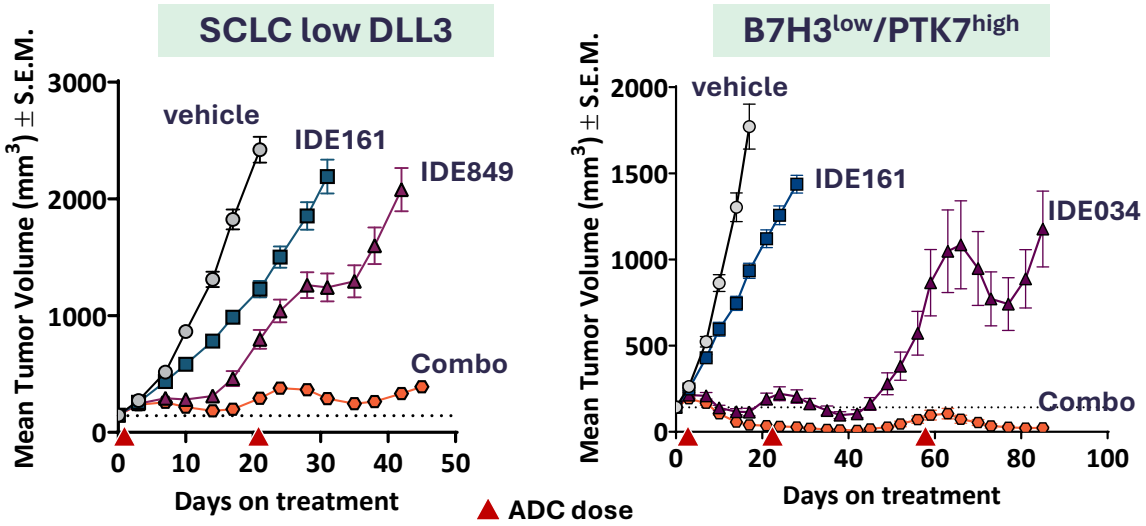
IDE849 *(DLL3)*

IDE034 *(B7H3/PTK7)*

Bispecific, AND gate format

IDE161 *(PARG)*

Chemically-installed synthetic lethality



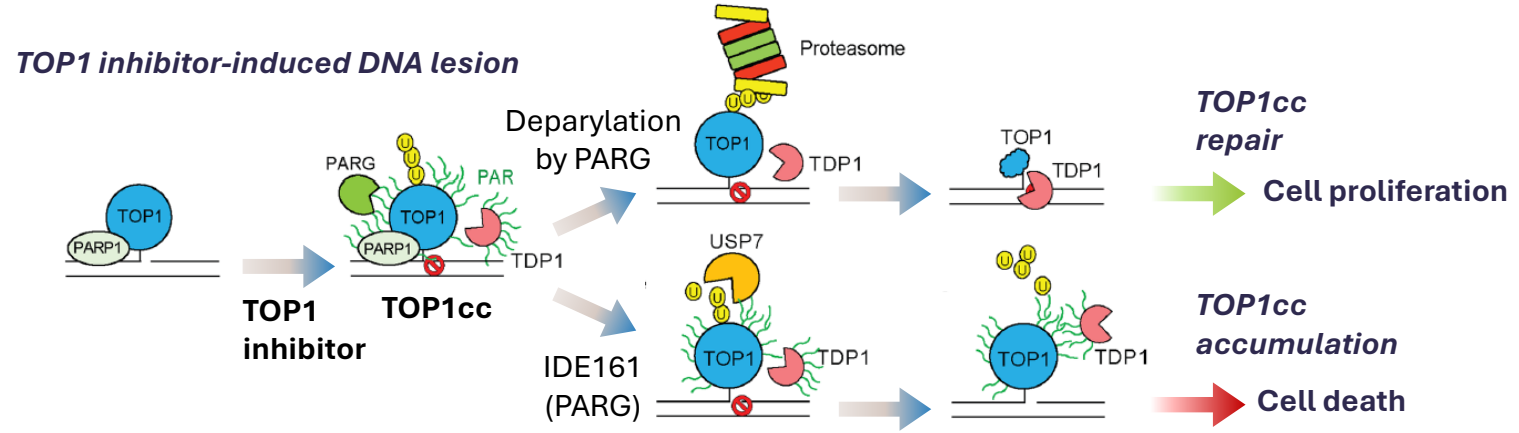
DLL3 = Delta-like ligand 3, ADC = antibody drug conjugate, TOP1 = topoisomerase I, DAR = drug-to-antibody ratio, ASCL1 = Achaete-Scute family BHLH transcription factor 1, PARG = poly(ADP-ribose) glycohydrolase

IDE161 Has Potential to Improve Efficacy and Durability of TOP1 ADCs

Combination mechanism has shown broad potential across multiple solid tumor models

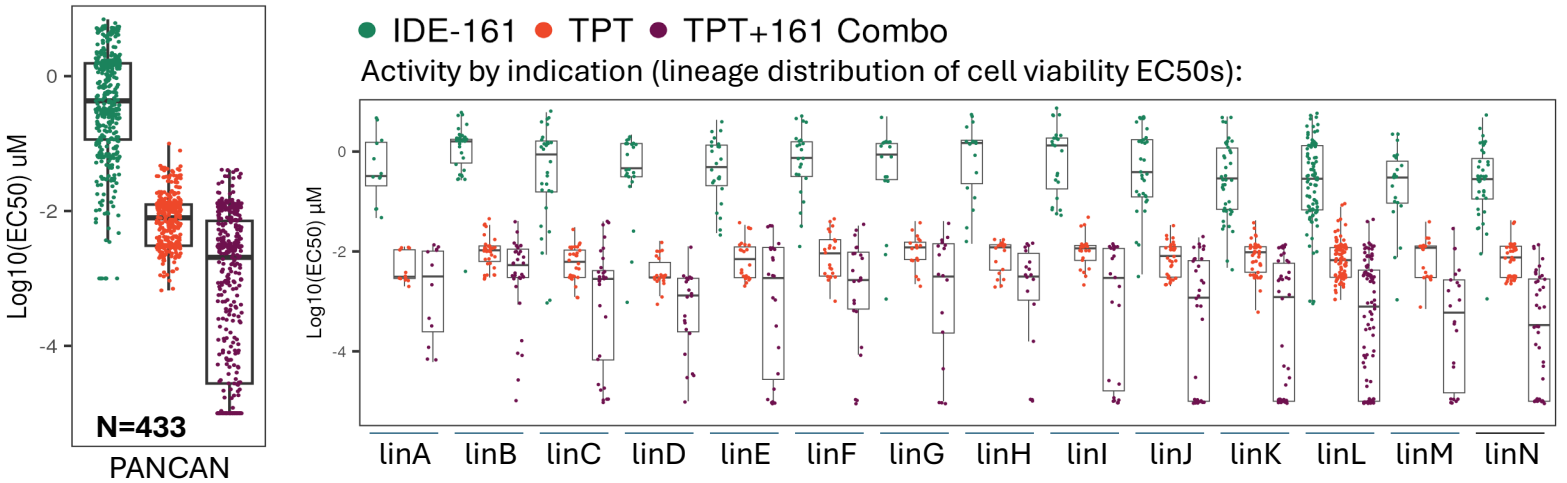
PARG inhibition by IDE161 maximizes TOP1 ADC payload efficacy

- 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]

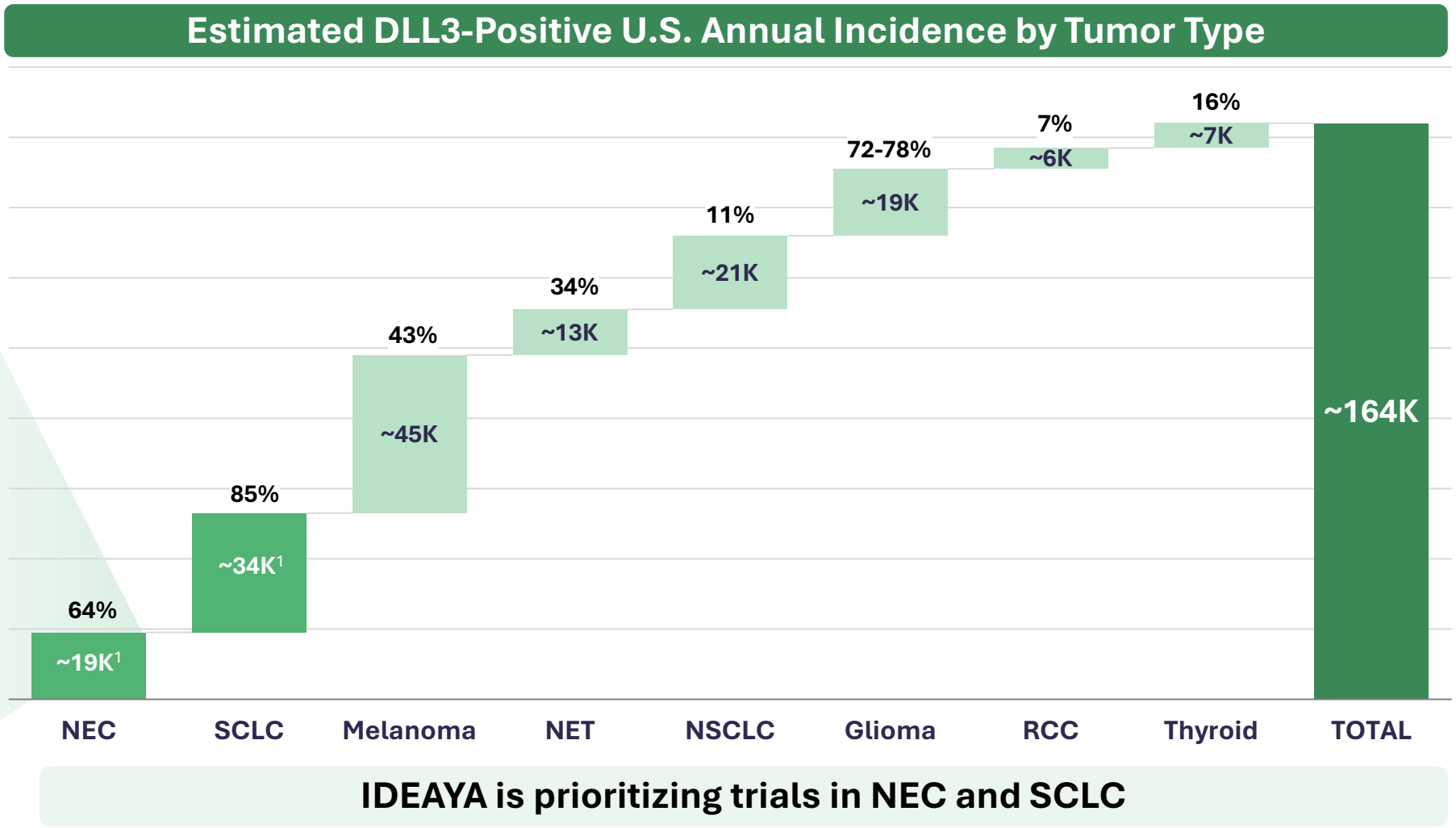


(1) Sun et al, Nat Com, 2021; (2) Munoz et al, AACR 2025
TOP1cc = Topoisomerase I cleavage complex, TPT = topotecan

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%



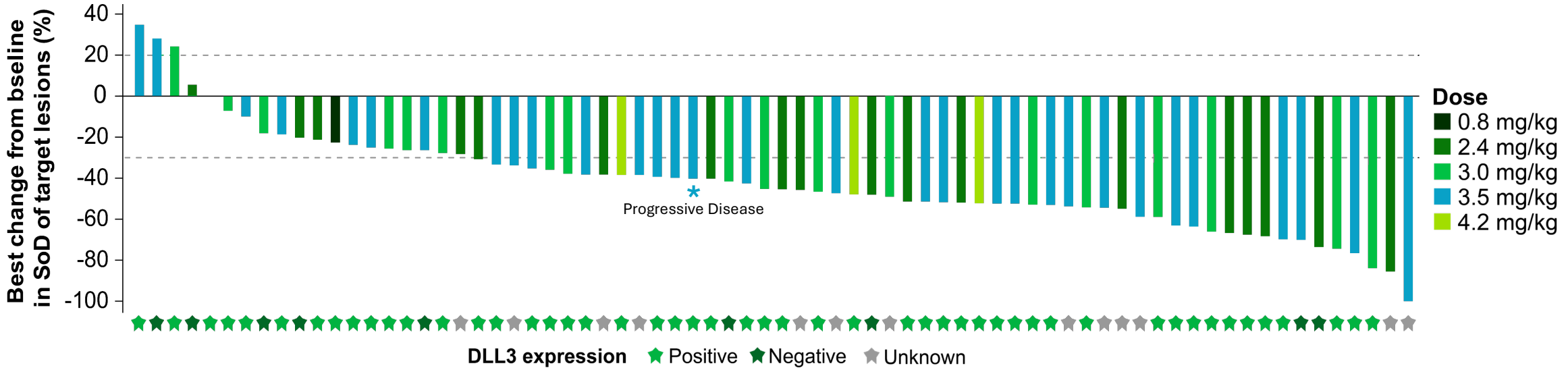
(1) Based on 100% as no need to stratify NEC or SCLC population

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) Demonstrated Compelling Initial Efficacy in SCLC

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

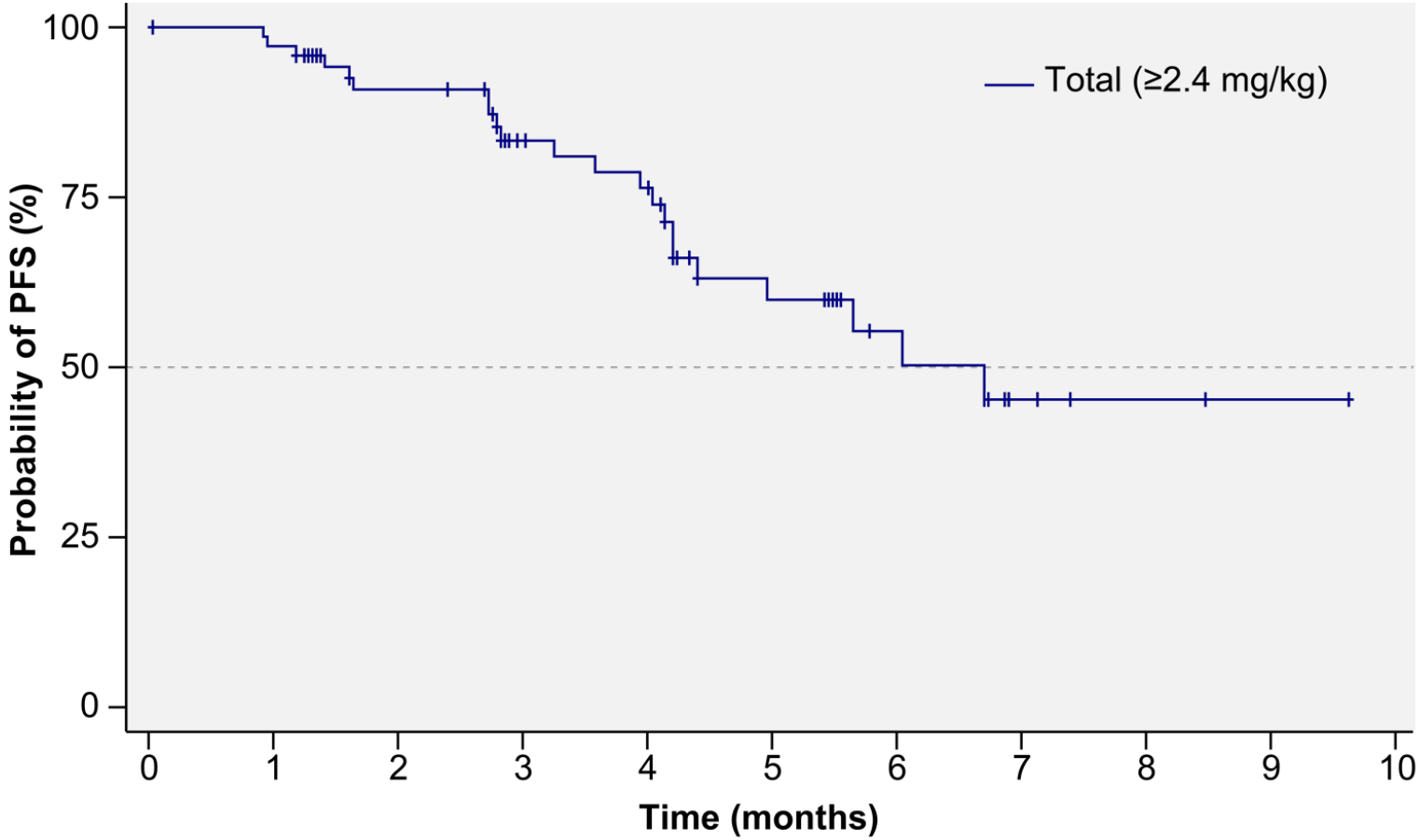


	2.4 mg/kg		3.0 mg/kg		3.5 mg/kg		4.2 mg/kg		Total (≥2.4 mg/kg)	
	2L Setting (n=10)	All (n=19)	2L Setting (n=8)	All (n=18)	2L Setting (n=16)	All (n=31)	2L Setting (n=1)	All (n=3)	2L Setting (n=35)	All (n=71)
ORR, n (%)	8 (80.0%)	14 (73.7%)	6 (75.0%)	12 (66.7%)	12 (75.0%)	23 (74.2%)	1 (100.0%)	3 (100.0%)	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

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)

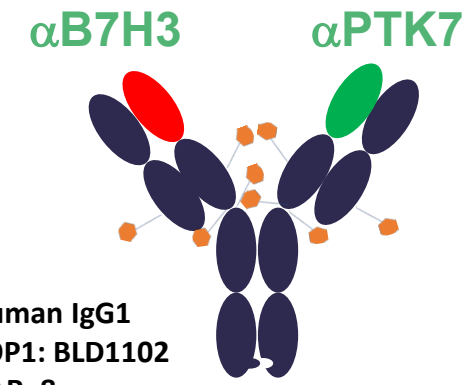
(to be initiated once monotherapy expansion cohorts are underway)

- 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



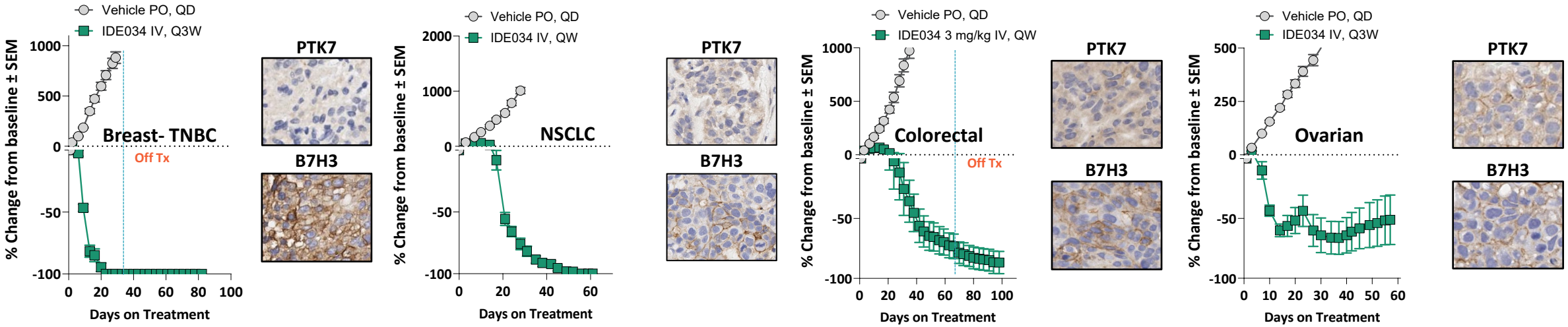
Human IgG1
TOP1: BLD1102
DAR=8

- Enhanced binding and internalization in double-positive cells to potentially enhance efficacy and safety versus B7H3 and PTK7 mono TOP1 ADCs
- Internalization-dependent payload release

Indication	B7H3/PTK7 % Double Positive[1]
Lung	≥ 30%
Colorectal[2]	≥ 40%
Breast	≥ 40%
Ovarian	≥ 35%
HNSCC	≥ 30%

- Substantial B7H3/PTK7 patient population across tumor types
- Targets tumor-initiating cells to potentially inhibit resistance
- Minimal dual antigen expression in normal tissues
- Important IDE161 combination opportunity

IDE034 has demonstrated robust anti-tumor activity across priority indications



(1) IDEAYA analysis of Human Protein Atlas; (2) Human Protein Atlas annotates colorectal cancer as bowel cancer
TNBC = triple negative breast cancer

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)*

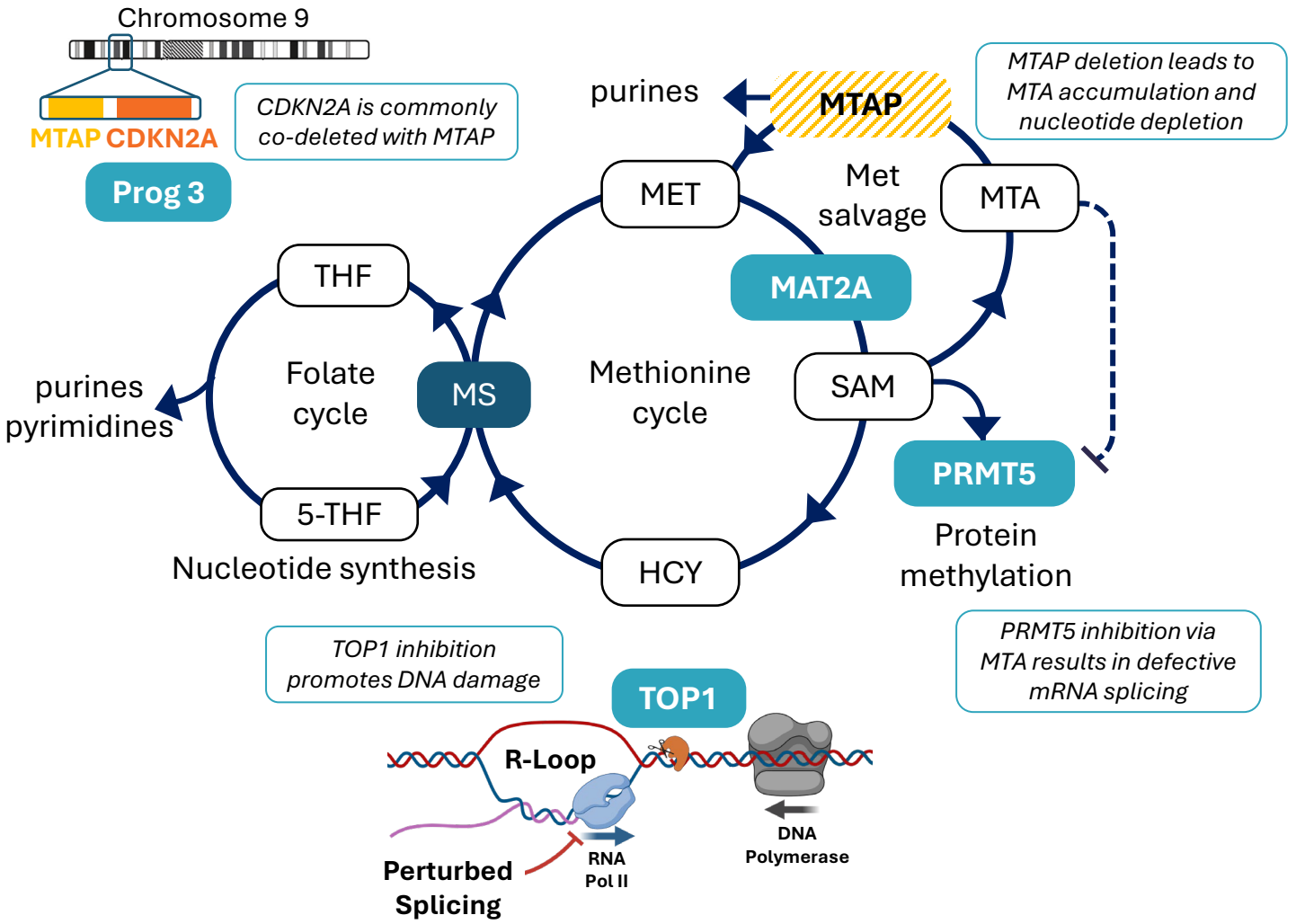
** Potential example*

2) MAT2A + PRMT5

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

3) MAT2A + co-alterations of MTAP

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

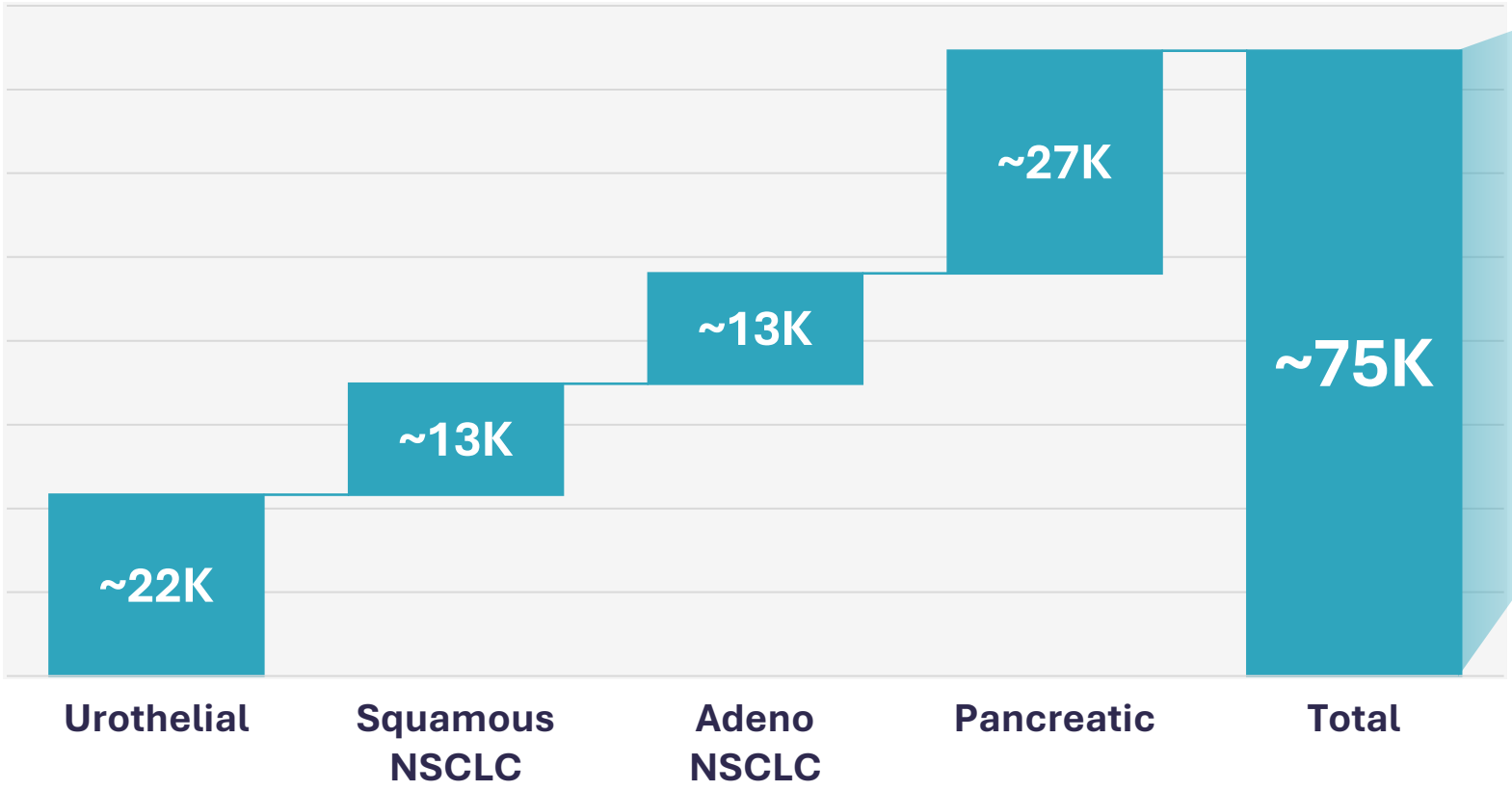


MTAP = methylthioadenosine phosphorylase, MAT2A = methionine adenosyltransferase 2a, DC = development candidate, MTA = methylthioadenosine, PRMT5 = protein arginine methyltransferase 5, SAM = S-adenosylmethionine, MET=methionine, HCY=homocysteine, THF= tetrahydrofolate, 5-THF= 5-methytetrahdrofolate;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

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

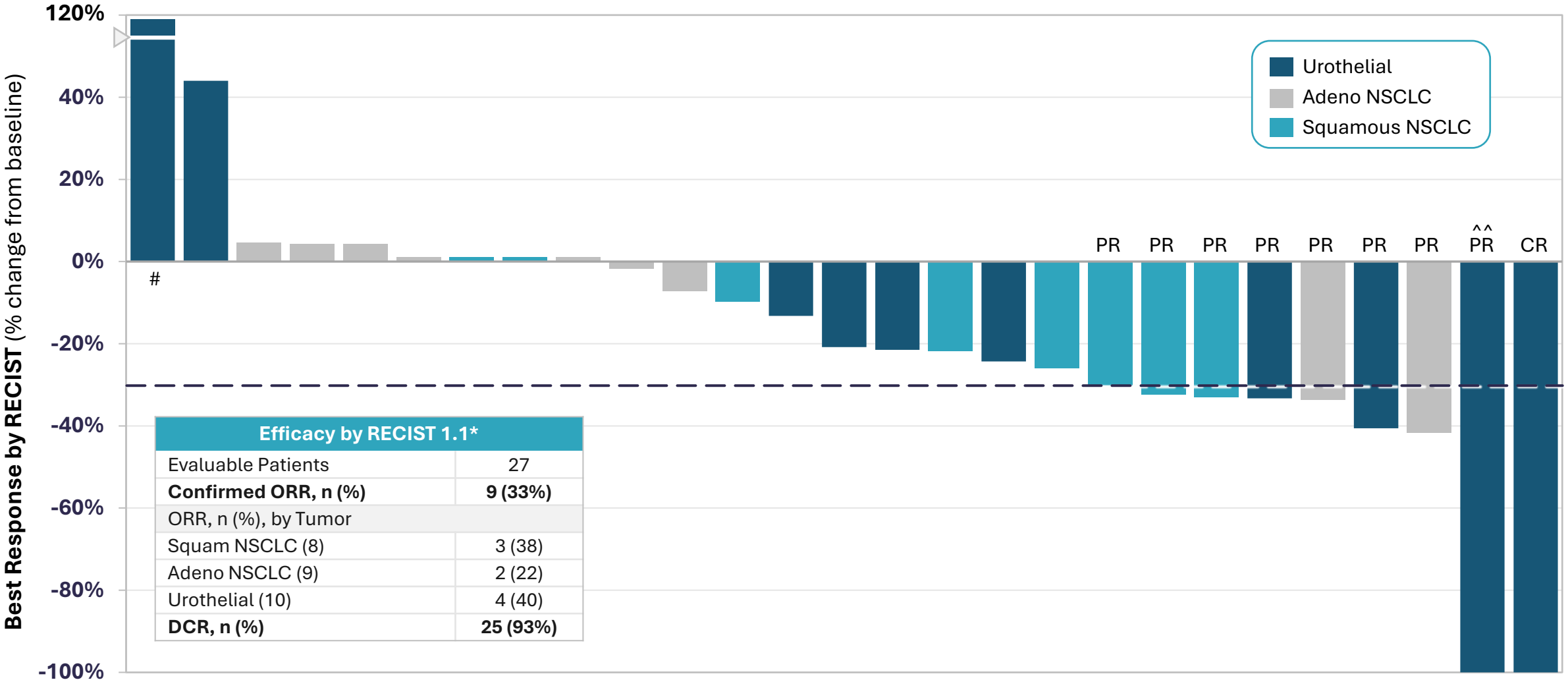


MTAP-Deletion Frequency by Tumor Type[1]	
Urothelial Cancer	**26%**
Squamous NSCLC	**19%**
Adeno NSCLC	**11%**
Pancreatic[2]	**40%**
Head and Neck	14%
Gastric	10%
Squamous Esophageal Cancers	28%

(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

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

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



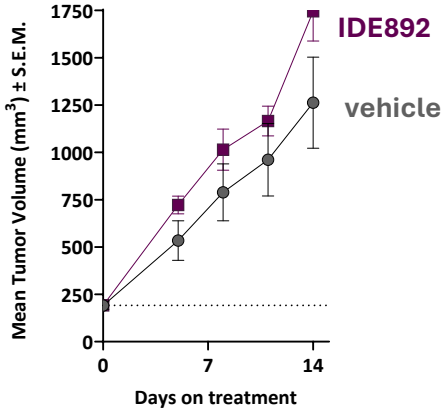
Efficacy by RECIST 1.1*	
Evaluable Patients	27
Confirmed ORR, n (%)	**9 (33%)**
ORR, n (%), by Tumor	
Squam NSCLC (8)	3 (38)
Adeno NSCLC (9)	2 (22)
Urothelial (10)	4 (40)
DCR, n (%)	**25 (93%)**

* 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

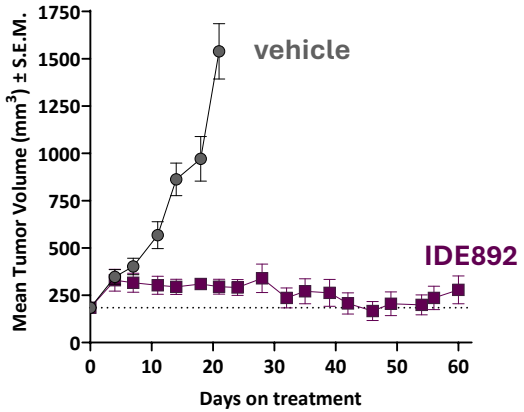
Phase 1 PRMT5 Inhibitor, IDE892, Exhibits Robust Selectivity and Combination Potential with Phase 2 MAT2A Inhibitor IDE397 in MTAP$^{-/-}$ Preclinical Models

Only active in MTAP$^{-/-}$ cells (HCT116 isogenic pair)

HCT116 WT

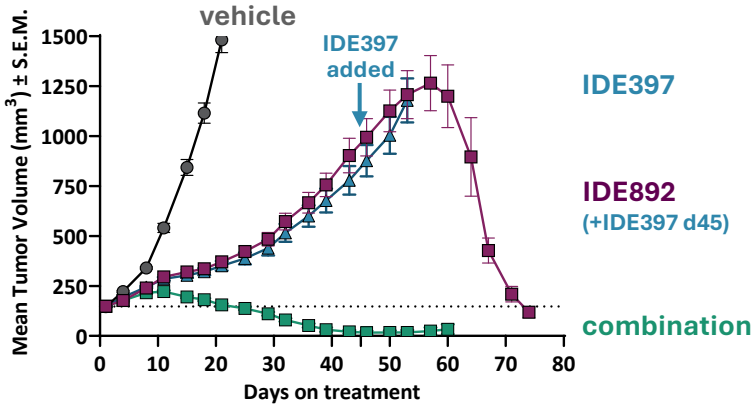


HCT116 MTAP$^{-/-}$



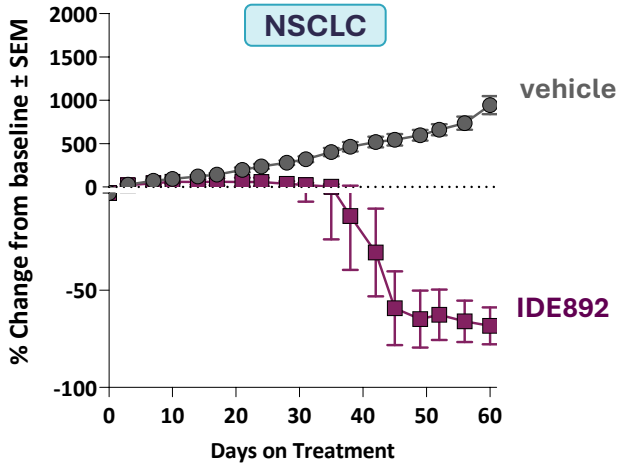
IDE397 combination delivers CRs and reverses relapse

NCI-H838 MTAP$^{-/-}$

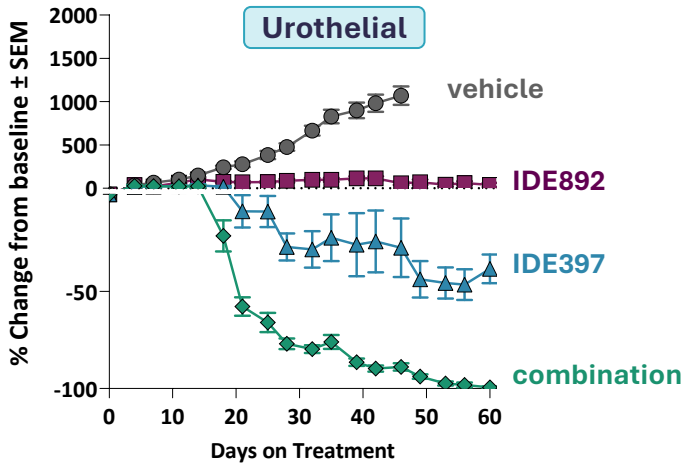


Strong monotherapy and combination benefit observed in MTAP$^{-/-}$ PDX models

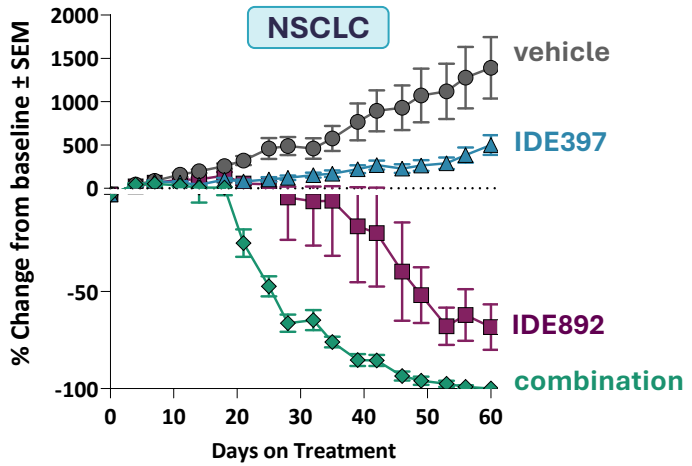
NSCLC



Urothelial



NSCLC

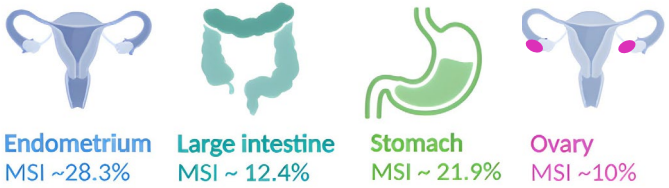


MTAP$^{-/-}$ = MTAP deleted, WT= wild type, CR = complete response

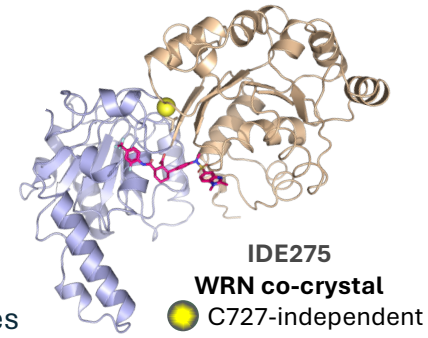
Next Generation, Potential First-In-Class Therapies in Phase 1 for Solid Tumors

IDE275: Werner Helicase (WRN) Inhibitor Targeting MSI-H Cancers

Unique IDE275-bound helicase conformation can overcome intrinsic and acquired resistance to other clinical WRN inhibitors

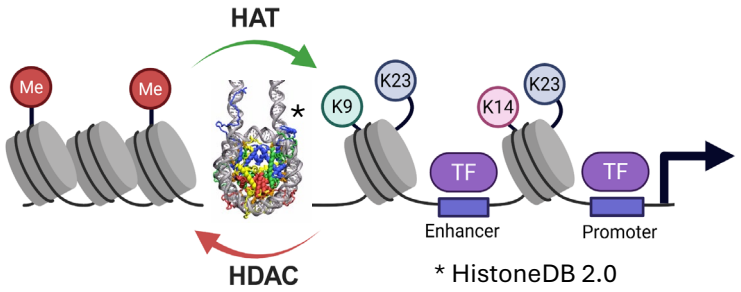


- MSI-H tumors caused by MMR deficiencies
- 3-4% of all cancers
- 30-50% derive no benefit from checkpoint therapies



IDE574: dual KAT6/7 Inhibitor for mBC, Lung, Prostate and CRC

Histone acetyltransferases (HATs) KAT6 and KAT7 collaboratively promote tumorigenic gene expression programs

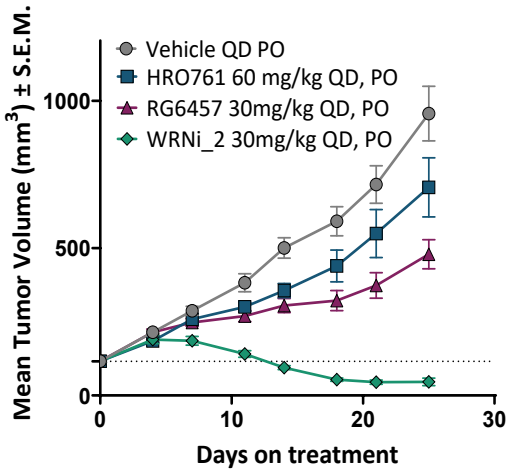


Lineage-survival oncogene TF networks

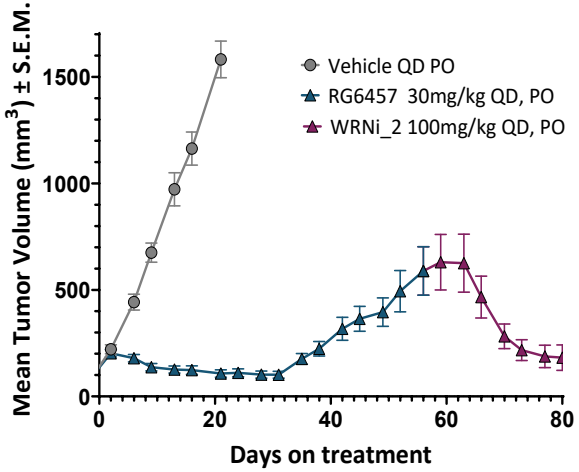
Tumor initiating cell maintenance

Evolution of drug-tolerant persister cells

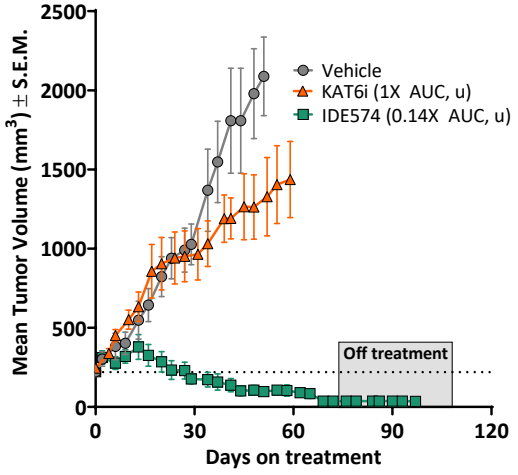
MSI-H Gastric Cancer
(Chemo-refractory PDX)



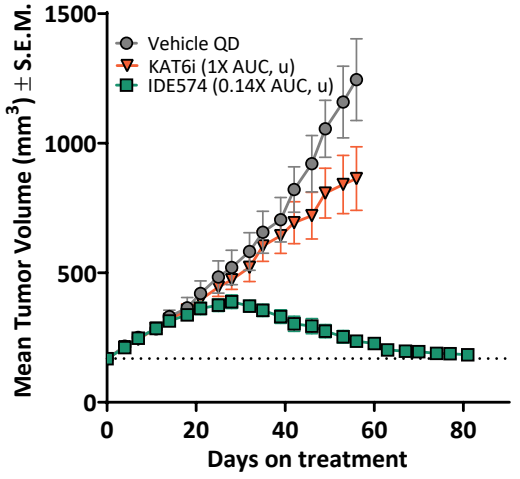
MSI-H CRC
(SW48)



HR+ mBC
(PDX ST941, ESR1 Y537S)



NSCLC
(PDX LU5209, AT2, KEAP1/STK11 mut)



WRNi_2 = in vivo tool analog of IDE275, RG6457 = Bayer/Roche compound, HRO761 = Novartis compound, MSI-H = microsatellite instability-high, MMR = mismatch repair, HDAC = histone deacetylase, mBC = metastatic breast cancer, TF = transcription factor, 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

Advancing a pipeline of 9 clinical stage programs across multiple solid tumor indications

Target 2026 Pipeline Milestones

		Q1 2026	Q2 2026	H2 2026
Darovasertib/ Uveal Melanoma (UM)		**1L Metastatic UM, Phase 2/3** Topline randomized PFS results to enable potential US AA filing[2]	**Metastatic UM** Complete HLA*A2 positive enrollment for RWE/NCCN **Adjuvant UM** Initiate Phase 3 trial	
ADC+DDR Combos	*IDE849 (DLL3)*			**Phase 1 data (monotherapy)** Global study (YE'26) **Initiate registrational trial** 2L+ setting, SCLC/NEC (YE '26)
	IDE034 (B7H3/PTK7)	**Phase 1 FPI (monotherapy)** ✓ Solid tumors		
	IDE161 (PARG)		**Phase 1 FPI (+IDE849 combo)** SCLC, NEC, DLL3+	
MTAP Pathway	*IDE397 (MAT2A)*			**Phase 2 data (+Trodelvy)** UC patients (2026)
	IDE892 (PRMT5)	**Phase 1 FPI (dose escalation)** ✓ Solid tumors		**Phase 1 FPI (+IDE397 combo)** NSCLC (mid-2026)
Next Gen Therapies	*IDE574 (KAT 6/7)*	**Phase 1 FPI (monotherapy)** Solid tumors		

Highlights

Darovasertib commercial readiness activities ongoing

~$1.05 B in cash and equivalents with runway into 2030[1]

Strong partnerships

SERVIER

HENGRUI

NASDAQ: IDYA

(1) Includes aggregate of approximately $1,050 million of cash, cash equivalents and marketable securities as of Dec 31, 2025, as detailed on IDEAYA's Form 10-K filed with the U.S. SEC; runway based on current operating plan; (2) 1L Metastatic UM Phase 2/3 trial in HLA*A2-negative setting (OptimUM-02)
FPI = first-patient-in, RWE = real world evidence

March 2026

Improving Lives Through Transformative Precision Medicines

Corporate Presentation

IDEAYA BIOSCIENCES

NASDAQ: **IDYA**