March 2024

NASDAQ: **IDYA**

IDEAYA Biosciences

Improving Lives Through Transformative Precision Medicines

Safe Harbor Statement

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, including 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; 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 and guidance, manufacturing, release of data or program updates; any statements of expectation or belief regarding future events, potential markets or market size, technology developments, therapeutic benefits, or receipt of cash milestones 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 IDEAYA’s programs’ early stage of development, the process of designing and conducting preclinical and clinical trials, the regulatory approval processes, the timing of regulatory filings, the challenges associated with manufacturing drug products, IDEAYA’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, 2023, and any current and periodic reports filed thereafter. 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.

This presentation concerns anticipated products that are under clinical investigation and which have not yet been approved for marketing by the U.S. Food and Drug Administration (FDA). It is currently limited by Federal law to investigational use, and no representation is made as to its safety or effectiveness for the purposes for which it is being investigated. IDEAYA and the IDEAYA logo are trademarks of IDEAYA Biosciences, Inc. All other trademarks used herein are the property of their respective owners.

IDEAYA Biosciences Highlights

Leading Precision Medicine Oncology Biotechnology Company Advancing Potential First-in-Class Therapies

Broad Pipeline of 4 Clinical Programs with Multiple 2024 Target Milestones and Catalysts

PHASE 2/3	PHASE 1/2	PHASE 1	IND-ENABLING	PRECLINICAL
DAROVASERTIB (PKC) • Daro + Crizo (cMET) 1L MUM Registrational Ph2/3 Program Update(s) – 2024 • Daro + Crizo Phase 2 expansion in GNAQ/11 Cutaneous Melanoma • Neoadjuvant UM Phase 2 Clinical Efficacy Updates – Mid 2024 • Regulatory guidance update in Neoadjuvant UM – 2024	**IDE397 (MAT2A)** • Phase 1/2 expansion in MTAP NSCLC and Bladder **IDE397 + AMG 193 (PRMT5)** • Ongoing Phase 1 enrollment and development of joint publication strategy – 2024 **IDE397 + Trodelvy® (Trop2-ADC)** • Phase 1 FPI in MTAP Bladder – Mid 2024	**IDE161 (PARG)** • Phase 1/2 Expansion in HRD • Program Update(s) - 2024 • Enable Combination(s) - 2024 **IDE161 + KEYTRUDA® (pembrolizumab)** • Phase 1 in Endometrial Cancer **GSK101 (POL THETA)** • Ongoing Phase 1 dose escalation	**WERNER HELICASE** • IND Submission ($7M Milestone upon successful IND clearance) – 2024	**NEXT GEN PROGRAMS** • Development Candidate Nominations, including in MTAP-deletion – 2024

Pharma Collaborations

Pfizer | AMGEN | GILEAD | MERCK | GSK

~$2B in potential milestones

Financials and Investor Relations

~$975M to fund operations into 2028 [1, 2]

NASDAQ: IDYA

IND = Investigational New Drug, UM = Uveal Melanoma, MUM = Metastatic Uveal Melanoma, NSCLC = Non Small Cell Lung Cancer, HRD = Homologous Recombination Deficiency, MTAP = methylthioadenosine phosphorylase

(1) Includes aggregate of $632.6M cash, cash equivalents and marketable securities as of December 31, 2023, plus pro forma $342.3M estimated net proceeds from sales of common stock through at-the-market offerings in January 2024

(2) IDEAYA Form 10-K dated February 20, 2024 as filed with the U.S. Securities and Exchange Commission

IDEAYA BIOSCIENCES

Leading Functional Genomics and Synthetic Lethality Platform

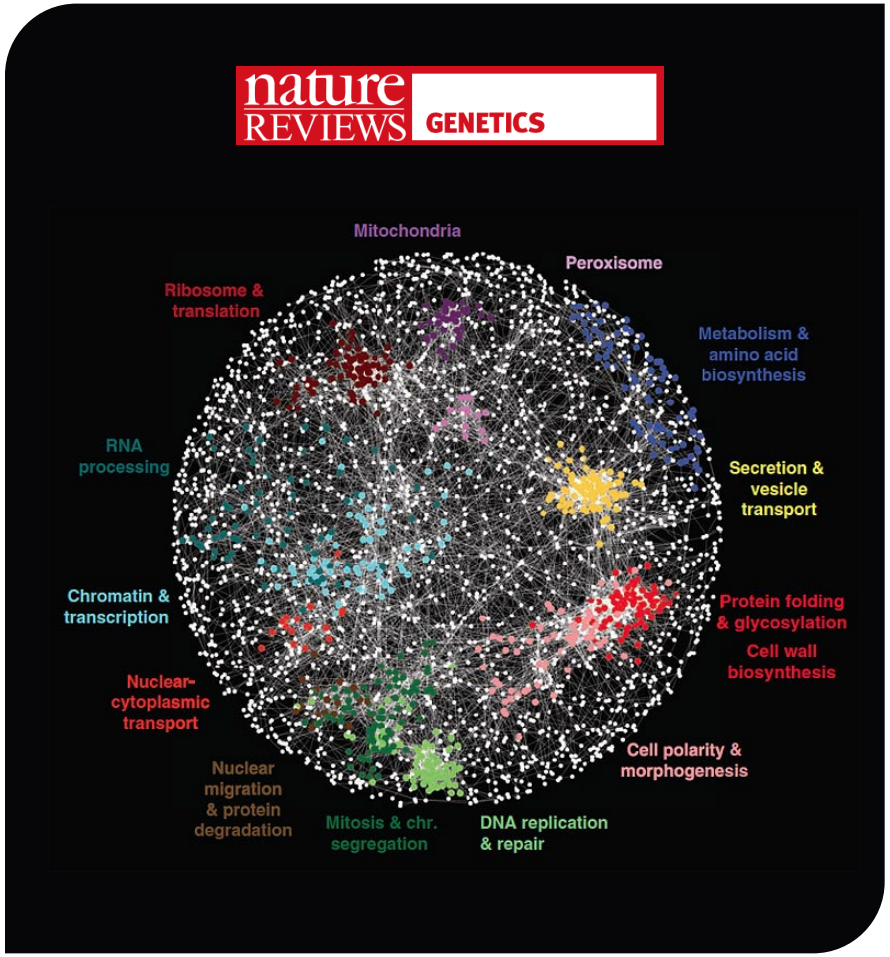
The Next Frontier in Precision Medicine Oncology

Functional Genomics and Synthetic Lethality provides a powerful approach to discover novel precision medicine therapies with patient biomarkers, including MTAP-deletion (~15% of solid tumors), BRCA/HRD (Breast, Prostate, Ovarian, Others), and high-MSI (15% GI Cancers)

Functional genomics combines human genetics with advances in AI and machine learning to develop effective precision medicines

Synthetic lethality occurs when the simultaneous perturbation of two genes results in cell death

Large-scale screening for novel synthetic lethal targets has progressed through advances in molecular biology (e.g., RNA interference, CRISPR-Cas9 editing) and bioinformatics



Nature Reviews Genetics, Vol. 18, 2017, Hieter, et al., as edited by IDEAYA; Image (right) Charles Boone

IDEAYA Precision Medicine Oncology Platform

Fully-Integrated Target, Biomarker, Drug Discovery and Translational Capabilities

Target & Biomarker Discovery and Validation

Bioinformatics, including AI Algorithms

Dual CRISPR, CRISPR, Chemogenomics

Genetically Engineered Models

- Key emerging novel targets identified, such as Werner Helicase, Pol Theta Helicase and PARG
- DECIPHER™ - Dual CRISPR SL Library in DDR Cell Lines in collaboration with UCSD
- PAGEO™ - Paralogous Gene Evaluation in Ovarian in collaboration with Broad Institute
- Machine Learning and Multi-Omics platform

Drug Discovery and Pharmacological Validation

Structure Based Drug Design

Small Molecule Chemistry

Protein Degrader Capabilities

- Crystal structures for SL discovery programs obtained to enable structure-based design
- INQUIRE™ Chemical Library - proprietary, expert-curated small-molecule library
- HARMONY™ Machine-Learning engine empowers drug discovery platform
- Differentiated clinical / candidate compounds discovered, including IDE397, IDE161 and GSK101 / IDE705 (Pol Theta Helicase)

Translational Research and Opportunity Expansion

Genomics – DNA and RNA Analysis

Proteomics – Protein Expression Profiling

Tissue (IHC, IF) and Liquid Biopsies Analysis

- Translational research to define clinical biomarkers and transformative combinations
- Opportunity expansion through broad cell panel screening
- Pharmacodynamic biomarker analysis to confirm target modulation and correlation with clinical activity

IDEAYA Functional Genomics and Synthetic Lethality Platform

Novel Target and Biomarker Discovery and Validation

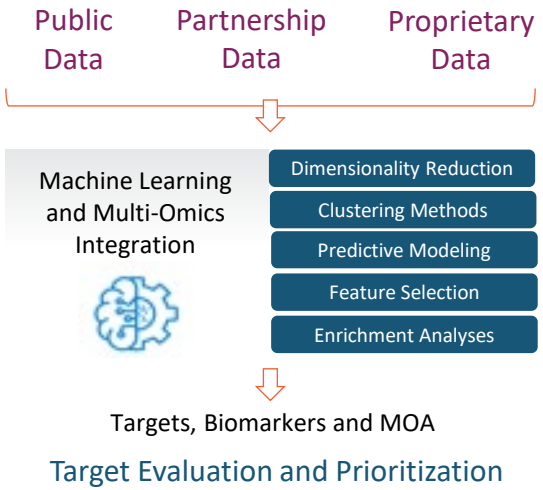
Target and Biomarker Discovery & Validation Platform

IDEAYA Functional Genomics Platform integrates proprietary and public data sets with orthogonal and complementary content

Bioinformatic analysis enables identification and validation of synthetic lethal target / biomarker interactions across vast datasets

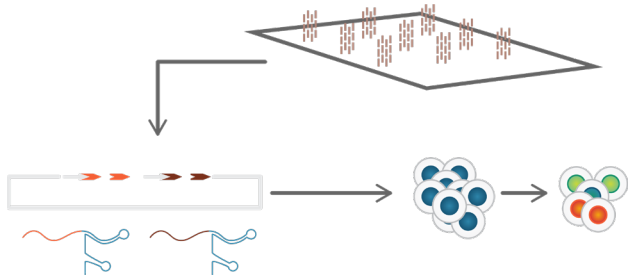
Robust SL interactions validated genetically (Dual CRISPR, paralogues, isogenic pairs, CRISPR/siRNA), pharmacologically and *in vivo*

Data-to-Knowledge Enterprise fueled by multi-omic patient and molecular data across public, partnership and proprietary data sets via IDEAYA Bioinformatics Platform



DECIPHER™

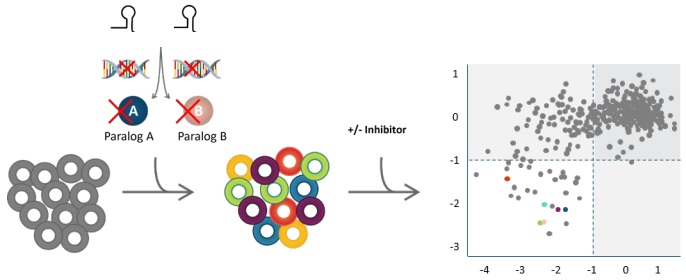
Dual CRISPR SL Library in DNA Damage Repair [2]



Evaluation of DNA Damage Targets synthetic lethal with tumor suppressor or oncogenes

PAGEO™

Paralogous Gene Evaluation in Ovarian Cancer [1]



Evaluation of SL targets in context of functionally redundant paralogous genes in ovarian cancer

Partnership Datasets

depmap portal | FOUNDATIONINSIGHTS™

Public Databases

TCGA | BioPortal | CCLE | PROJECTGENIE | Project Achilles | MSK-IMPACT™

[1, 2] IDEAYA Proprietary Libraries and Datasets – Strategic Collaborations with Broad Institute[1] and UC San Diego[2]

IDEAYA Precision Oncology Drug Discovery Platform & IND Engine

AI/ML & Structure Based Drug Design to Deliver First-in-Class Development Candidates

Structural Biology & Structure Based Drug Design

Full suite of capabilities in structural biology, biophysics, & computational chemistry

Ligand bound co-crystal structures resolved to enable Structure Based Drug Design for each of the Synthetic Lethality drug-discovery programs

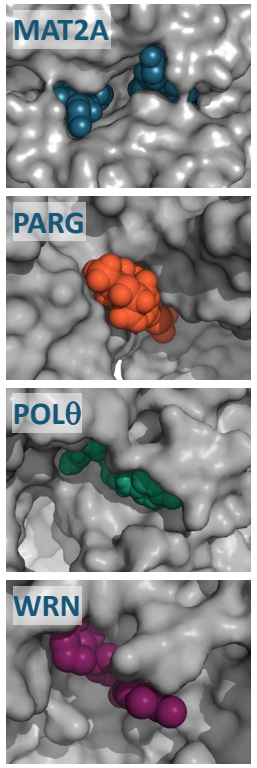
Multiple potential "first-in-world" co-crystal structures resolved, including for PARG, Pol Theta Helicase and Werner Helicase

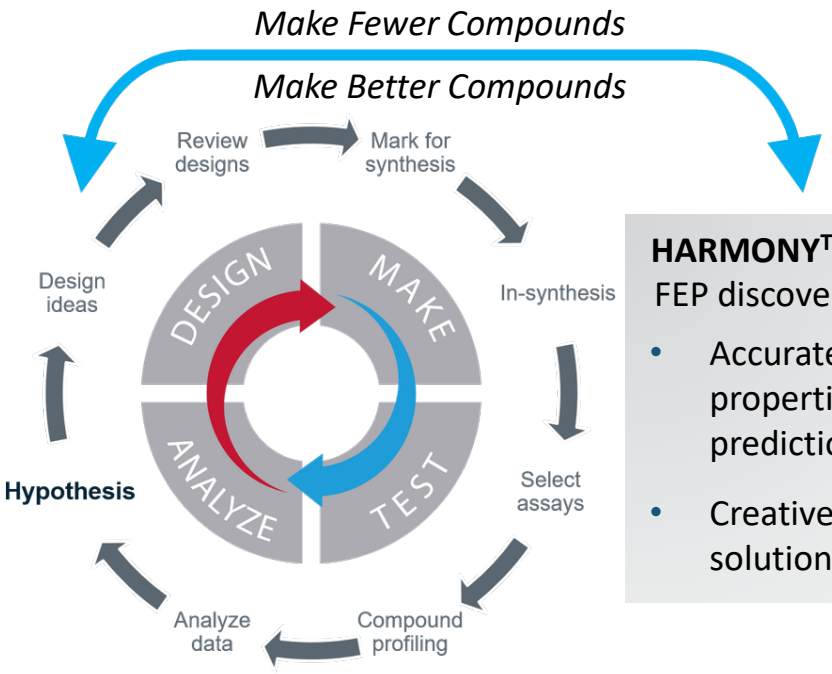
INQUIRE™ Proprietary Chemical Library

Expert-curated HTS library to enhance hit discovery capabilities against novel SL targets classes

Enhances IDEAYA's SL Drug Discovery Platform and competitive differentiation

AI/ML Enabled Computational Drug Discovery*





HARMONY™ ML and FEP discovery engine

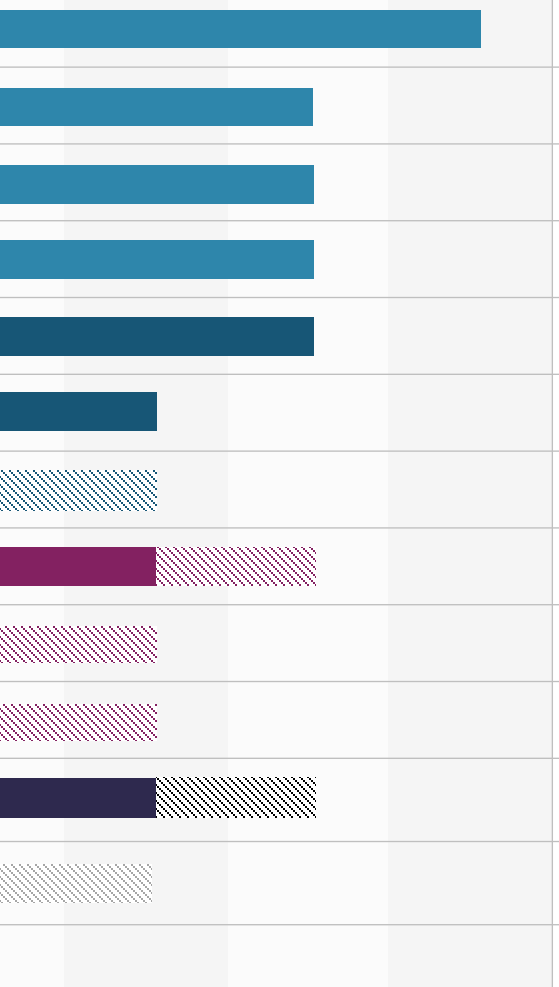
- Accurate properties prediction
- Creative design solutions

AI/ML to Accelerate Time to IND for First-in-Class Targets

*HARMONY™ ML: IDEAYA proprietary AI/Machine Learning platform to enable computational drug discovery and lead optimization

IDEAYA's Potential First-in-Class Precision Medicine Oncology Pipeline

	Modality/Indication	Biomarker	Pre-clinical / IND Enabling / Phase 1 / Phase 2 / Potential Registrational	Program Goals / Achievements	Collaborations	Commercial (IDEAYA)
Darovasertib *PKC*	+cMET[1] Combination 1L HLA-A2(-) MUM	GNAQ/11		Phase 2 (AA) / Phase 3 Registrational Trial ^ Program Update(s) - '24	Pfizer (1)	WW Commercial Rights
	cMET[1] Combination HLA-A2(+) MUM ^	GNAQ/11		HLA-A2(+) Clinical Trial ^^		
	cMET[1] Combination Cutaneous Melanoma	GNAQ/11		Phase 2 Expansion in Metastatic Cutaneous Melanoma		
	(Neo)Adjuvant UM	GNAQ/11		Phase 2 Clinical Efficacy – Mid '24 Regulatory Guidance – '24		
IDE397 *MAT2A*	Monotherapy Solid Tumors	MTAP		Phase 2 Monotherapy Expansion in NSCLC, Bladder		WW Commercial Rights
	Combination Solid Tumors	MTAP		Phase 1 IDE397 + AMG 193 (PRMT5i^{MTA}) ongoing enrollment and joint publication strategy – '24	AMGEN (2)	
	Combination Bladder Cancer	MTAP		Phase 1 IDE397 + Trodelvy® FPI – Mid '24	GILEAD (3)	
IDE161 *PARG*	Monotherapy Solid Tumors	HRD		Phase 2 expansion in priority tumor types (Breast, CRC, Endometrial, Prostate) – '24		WW Commercial Rights
	Combination Endometrial Cancer	High-MSI, MSS		Phase 1 IDE161 + KEYTRUDA® in endometrial cancer	MERCK (4)	
	Combinations Solid Tumors	HRD, Others		Enable IDE161 combination(s) – '24		
GSK101 *Pol Theta Helicase*	+Niraparib Combo[4] Solid Tumors	HR Mutations		Ongoing Phase 1 dose escalation		Global Royalties
WRN *Werner Helicase*	GI Cancers	High-MSI		Targeting IND submission in 2024 ($7M Milestone upon successful IND clearance)	GSK (5)	50% US Profits and 20% costs
Platform	Solid Tumors	Defined Biomarkers		Targeting Multiple DC Nominations, including in MTAP-deletion – '24	GSK (5)	WW Commercial Rights



^ Integrated Phase 2/3 enables potential Accelerated Approval (AA, Phase 2) and potential Full Approval (Phase 3) based on FDA Type C Meeting Q1 2023 , ^^ Targeting enrollment of additional HLA-A2(+) patients in a separate clinical trial (e.g., ongoing IDE196-001 Phase 2 clinical trial)

(1) Pursuant to Pfizer Clinical Trial Collaboration and Supply Agreements for Darovasertib/Crizotinib Combination; IDEAYA retains all Darovasertib Commercial Rights

(2) Pursuant to Amgen Clinical Trial Collaboration and Supply Agreement for IDE397 + AMG 193, an investigational MTA-cooperative PRMT5 inhibitor; Amgen is the sponsor the study and the parties jointly share external costs of the study

(3) Pursuant to Gilead Clinical Study Collaboration and Supply Agreement for IDE397 + Trodelvy®, a Trop-2 directed antibody-drug conjugate (ADC); the Company will sponsor the study and Gilead will provide Trodelvy at no cost

(4) Pursuant to Merck Clinical Trial Collaboration and Supply Agreement for IDE161 + Keytruda®, an anti-PD-1 therapy; the Company will sponsor the study and Merck will provide Keytruda at no cost

(5) Pursuant to GSK Collaboration, Option and License Agreement: Polθ: Global Royalties; WRN: 50/50 US Profits + ex-US Royalties

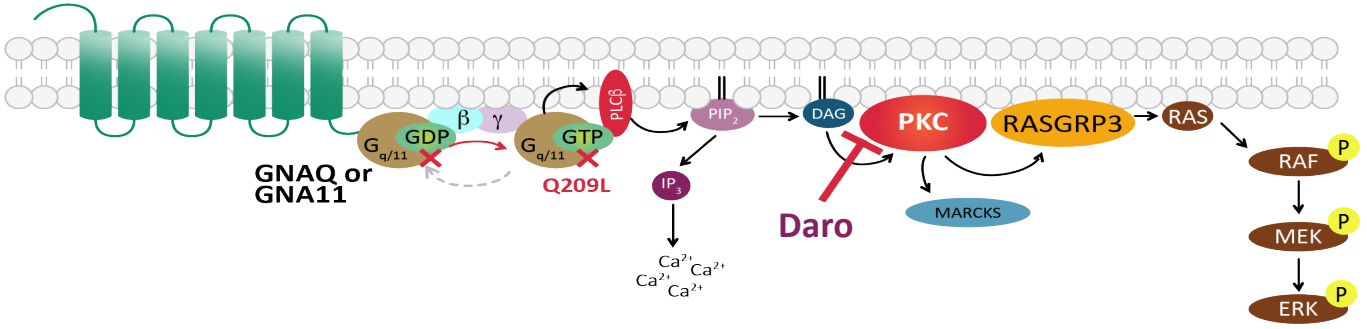
MAT2A=methionine adenosyltransferase 2a, MTAP=methylthioadenosine phosphorylase, MTA=methylthioadenosine, PRMT5=protein arginine methyltransferase 5 (PRMT5), PARG= poly (ADP-ribose) glycohydrolase, WRN = Werner Helicase, Polθ = DNA Polymerase Theta, HRD = homologous recombination deficiency, MSI = microsatellite instability, PKC = protein kinase C, MUM = metastatic uveal melanoma, UM = uveal melanoma, Crizo = crizotinib, NSCLC = non-small cell lung cancer, WW = worldwide, HLA-A2(-) = HLA-A2*02:01 Negative; HLA-A2(+) = HLA-A2*02:01 Positive, DC = development candidate

= Target 2024 Program Milestones

Darovasertib – Potential to Broadly Impact Uveal Melanoma

Potential First-in-Class and Best-in-Class in (Neo)adjuvant UM and Metastatic UM

Mutations in GNAQ / GNA11 activate PKC Signaling, a genetic driver of Uveal Melanoma



Darovasertib is an oral, potent and selective PKC inhibitor

GNAQ or GNA11 (~95%) and other upstream mutations activate PKC signaling in UM and MUM patients

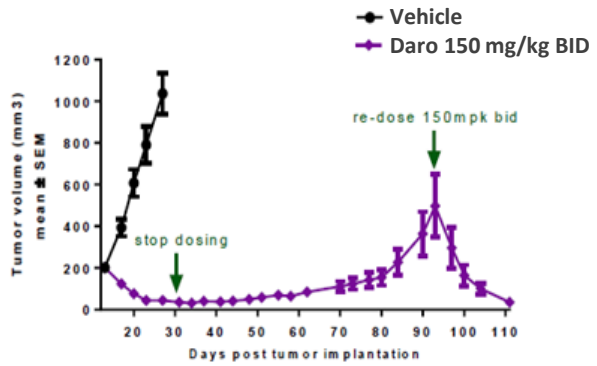
UM is typically treated with radiation and/or enucleation, with no approved systemic therapies for Neoadjuvant UM

MUM occurs in approximately 50% of UM patients and predominantly as liver metastasis in ~90% of MUM patients, with no approved therapies for HLA-A*02:01 negative MUM

Daro Mono Rationale in Primary UM

Single Agent Daro Induces Tumor Regression

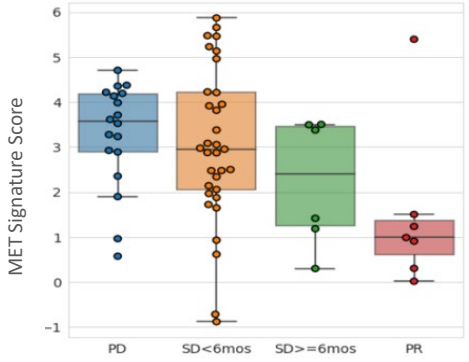
Uveal Melanoma Xenograft (92.1 mutant GNAQ)



Van Raamsdonk, CD, et. al, Nature 2009; Van Raamsdonk CD, et. al, NEJM 2010; Piperno-Neumann S, et. al, J Clin Oncol 2014

Daro + Crizo Combo Rationale for Use in Metastatic Uveal Melanoma (MUM)

Daro Phase 1 Monotherapy Efficacy Association with cMET Expression

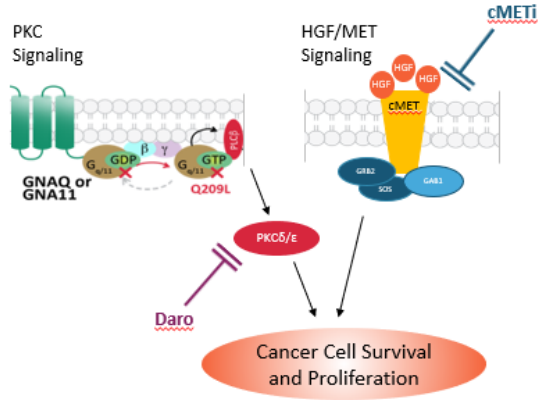


Ph 1 Clinical Outcomes

PD=Progressive Disease, SD=Stable Disease, PR=Partial Response

IDEAYA Data, AACR 2021

Activation of PKC and cMET Pathways with Observed cMET Overexpression in MUM Liver Metastases



Pfizer +

+ Pfizer Clinical Trial Collaboration and Supply Agreements for Darovasertib + Crizotinib Combination in MUM

IDEAYA owns or controls all commercial rights in darovasertib, including in Primary UM and MUM

IDEAYA BIOSCIENCES

Phase 2 Clinical Trial - Comparatively High-Risk, Poor Prognosis Population

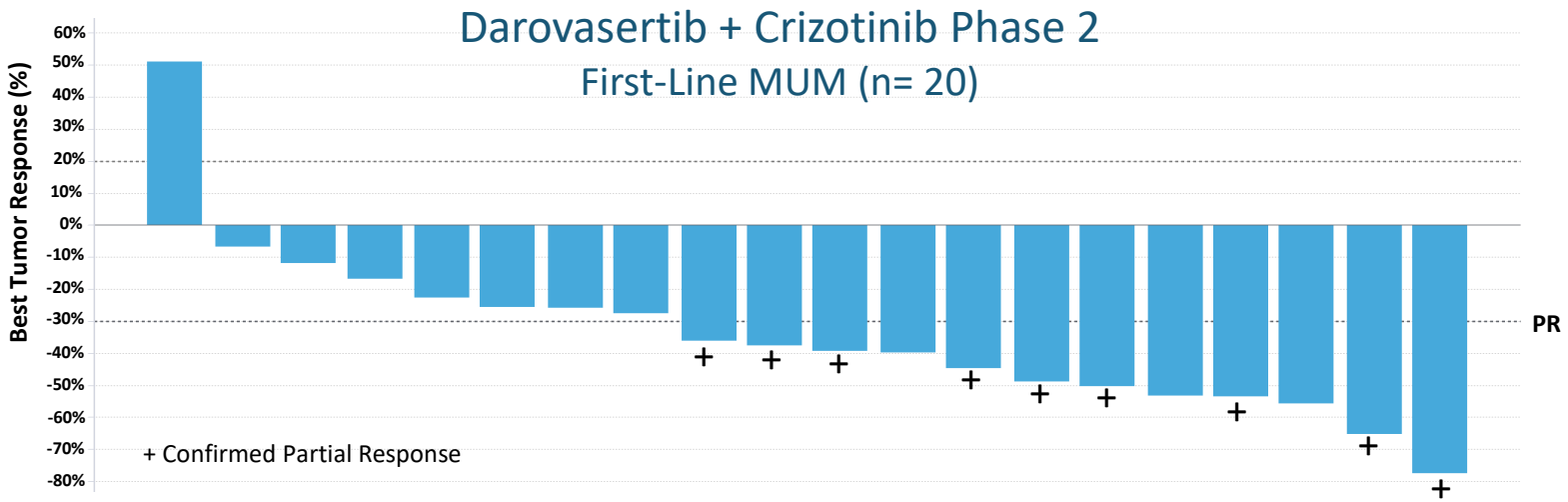
Disease Burden Significantly Higher in Both Any-Line and First-Line MUM Population+

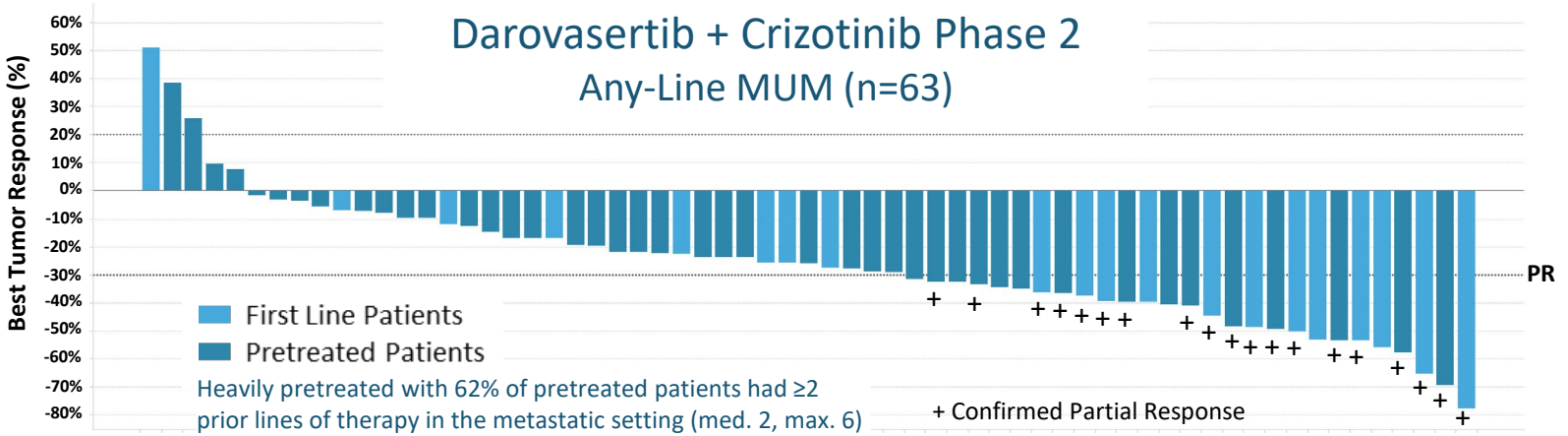
Baseline Characteristics		IDE196-001 Phase 2* Darovasertib + Crizotinib		Tebentasfusp First-Line Phase 3#	
		Any-Line n=63 (%)	First-Line n=20 (%)	Tebe Arm n=252 (%)	Control Arm^ n=126
Age (Years)	< 65	35 (56)	10 (50)	64 Median	66 Median
	≥65	28 (44)	10 (50)		
Sex	F	32 (51)	9 (45)	124 (49)	64 (51)
	M	31 (49)	11 (55)	128 (51)	62 (49)
ECOG PS	0	43 (68)	14 (70)	192 (76)	85 (67)
	1	20 (32)	6 (30)	49 (19)	31 (25)
Baseline LDH	Normal	25 (40)	10 (50)		
	>ULN	**38 (60)**	**10 (50)**	**90 (36)**	**46 (37)**
Largest metastatic lesion	≤3.0 cm	22 (35)	8 (40)	139 (55)	70 (56)
	3.1 to 8.0 cm	**35 (56)**	**9 (45)**	**92 (37)**	**46 (37)**
	≥ 8.1 cm	**6 (10)**	**3 (15)**	**21 (8)**	**10 (8)**
Location of metastases	Hepatic Only	19 (30)	10 (50)	131 (52)	59 (47)
	Extrahepatic Only	3 (5)	0	9 (4)	10 (8)
	Hepatic and Extrahepatic	**41 (65)**	**10 (50)**	**111 (44)**	**55 (44)**

+ Cross-trial comparisons are not based on head-to-head studies and are presented for informational purposes; no direct comparisons are being made
* IDEAYA Data as of August 22, 2023 (based on preliminary analysis of unlocked database by investigator review)
# N Engl J Med 2021; 385:1196-1206; ECOG data missing in Tebentafusp and Control arm of 4% and 7% respectively
^ Investigator Choice distribution in the control group: 103 (82%) received pembrolizumab, 16 (13%) received ipilimumab, and 7 (6%) received dacarbazine

Daro + Crizo Phase 2 Efficacy: First-Line MUM and Any-Line MUM

Compelling Overall Response Rate (ORR) by RECIST 1.1 Observed





Confirmed 45% ORR and 90% DCR

Response by RECIST 1.1 First-Line MUM	Evaluable (N=20)
Confirmed ORR (9/20)	45%
Tumor Shrinkage (19/20)	95%
>30% Tumor Shrinkage (12/20)	60%
Best Overall Response	
cPR (9/20)	45%
SD (9/20)	45%
DCR (18/20)	90%

Confirmed 30% ORR and 89% DCR

Response by RECIST 1.1 Any-Line MUM	Evaluable (N=63)
Confirmed ORR (19/63)	30%
Tumor Shrinkage (58/63)	92%
>30% Tumor Shrinkage (27/63)	43%
Best Overall Response	
cPR (19/63)	30%
SD (37/63)	59%
DCR (56/63)	89%

ESMO 2023 Proferred Presentation M McKean et al: preliminary analysis of unlocked database as of 8/22/2023 by investigator review, C1D1 cutoff as of 9/22/2022, based on 20 evaluable 1L MUM patients and 63 evaluable Any-Line (includes 1L and 2L+/pre-treated) MUM patients

ORR = Overall Response Rate; DCR = Disease Control Rate; cPR = Confirmed Partial Response; uPR = Unconfirmed Partial Response; SD = Stable Disease

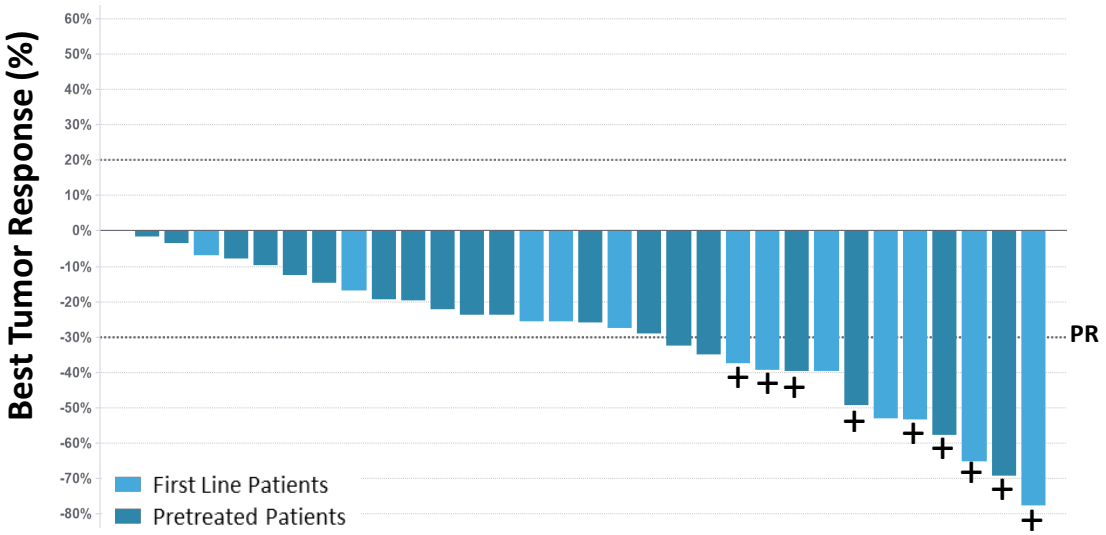
IDEAYA BIOSCIENCES

Daro + Crizo Phase 2 Efficacy: HLA-A2-Negative and HLA-A2-Positive MUM

Clinical Combination Observes Clinical Efficacy Irrespective of HLA-A2 Status

HLA-A2 Negative (n=31)

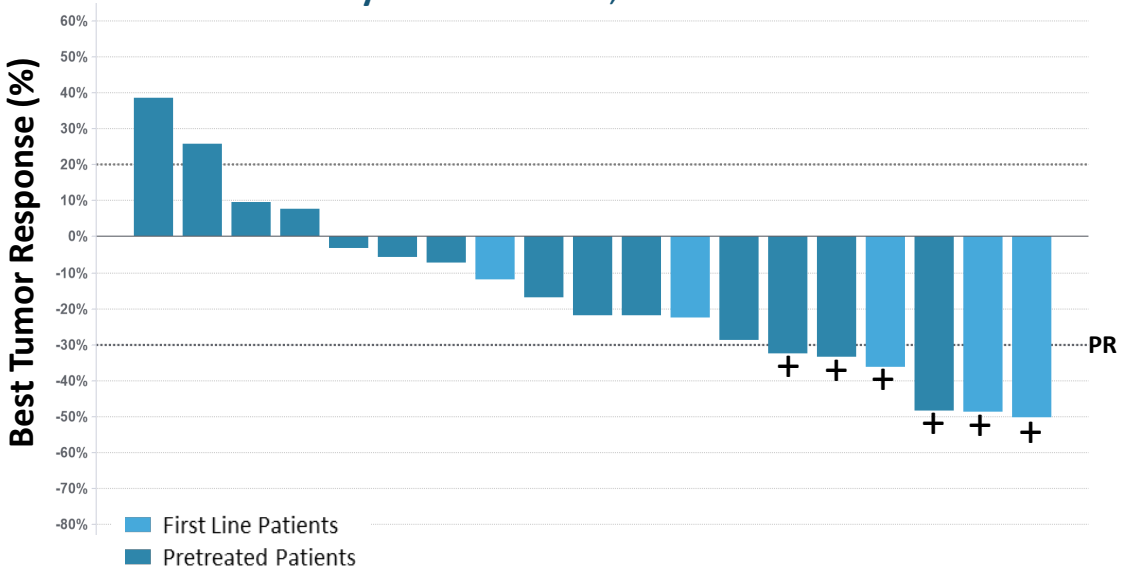
First-Line: 42% ORR; 100% DCR
Any-Line: 29% ORR; 94% DCR



+ Confirmed Partial Response

HLA-A2 Positive (n=19)

First-Line: 60% ORR; 100% DCR
Any-Line: 32% ORR; 84% DCR



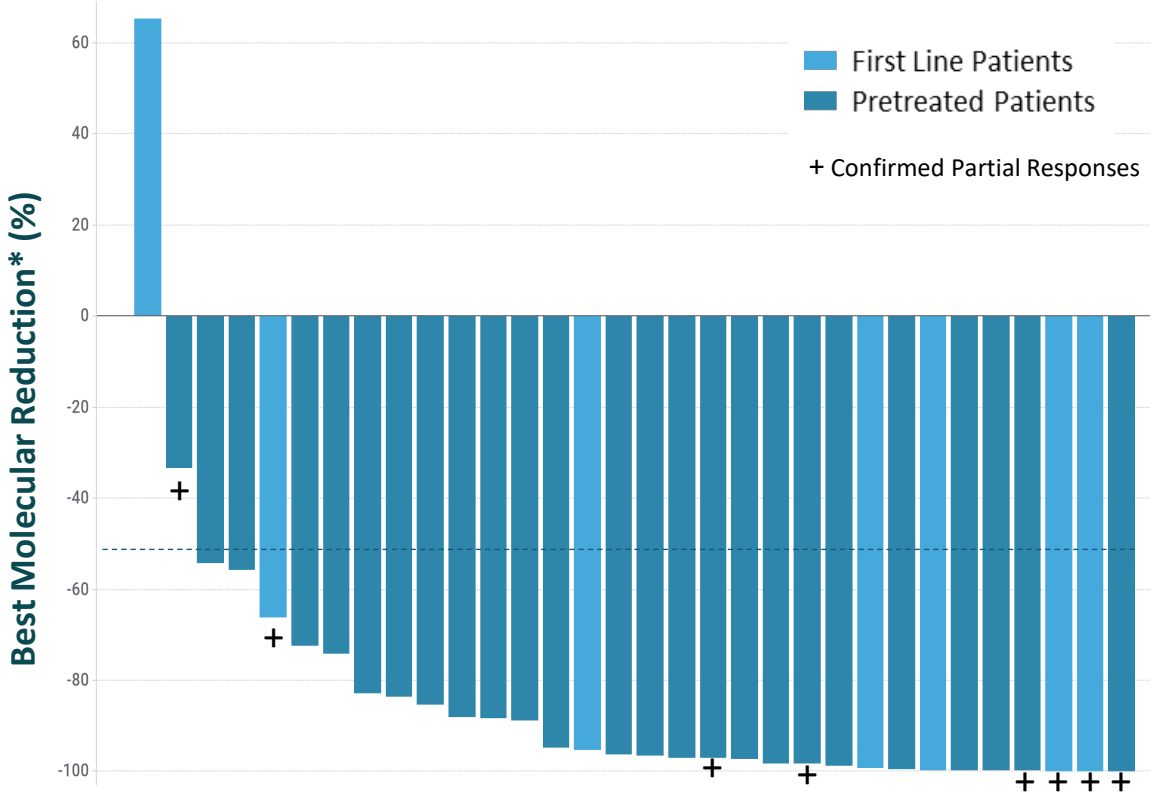
+ Confirmed Partial Response

ESMO 2023 Proferred Presentation M McKean et al : preliminary analysis of unlocked database as of 08/22/2023 by investigator review; data cutoff based on treatment Day 1 of Cycle 1 (C1D1) as of 9/22/2022

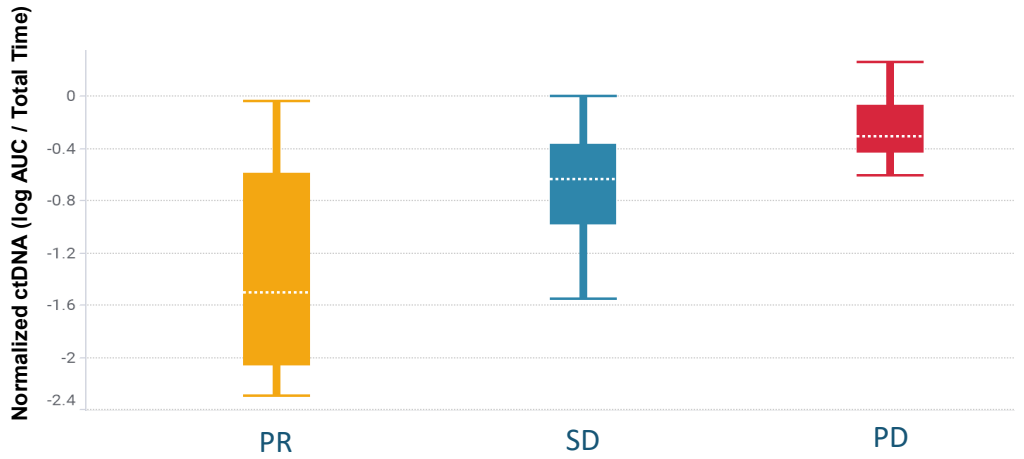
Observed 94% ctDNA Molecular Response Rate with Deep & Sustained MRs*

Any-Line MUM Patients Treated with the Darovasertib + Crizotinib Combination

Molecular Response Regardless of BAP1 Mutation



Molecular Response Correlates with RECIST Response ^



High ctDNA Molecular Response Rate of 94% in Any-Line MUM

Deep and Sustained MRs with approximately 80% of patients showing >80% reduction in MAF

ctDNA MRs correlate with Clinical Efficacy (PR, SD, PD) by RECIST

ESMO 2023 Proferred Presentation M McKean et al: Preliminary analysis of unlocked database as of 08/22/2023 by investigator review; data cutoff based on treatment Day 1 of Cycle 1 (C1D1) as of 9/22/2022

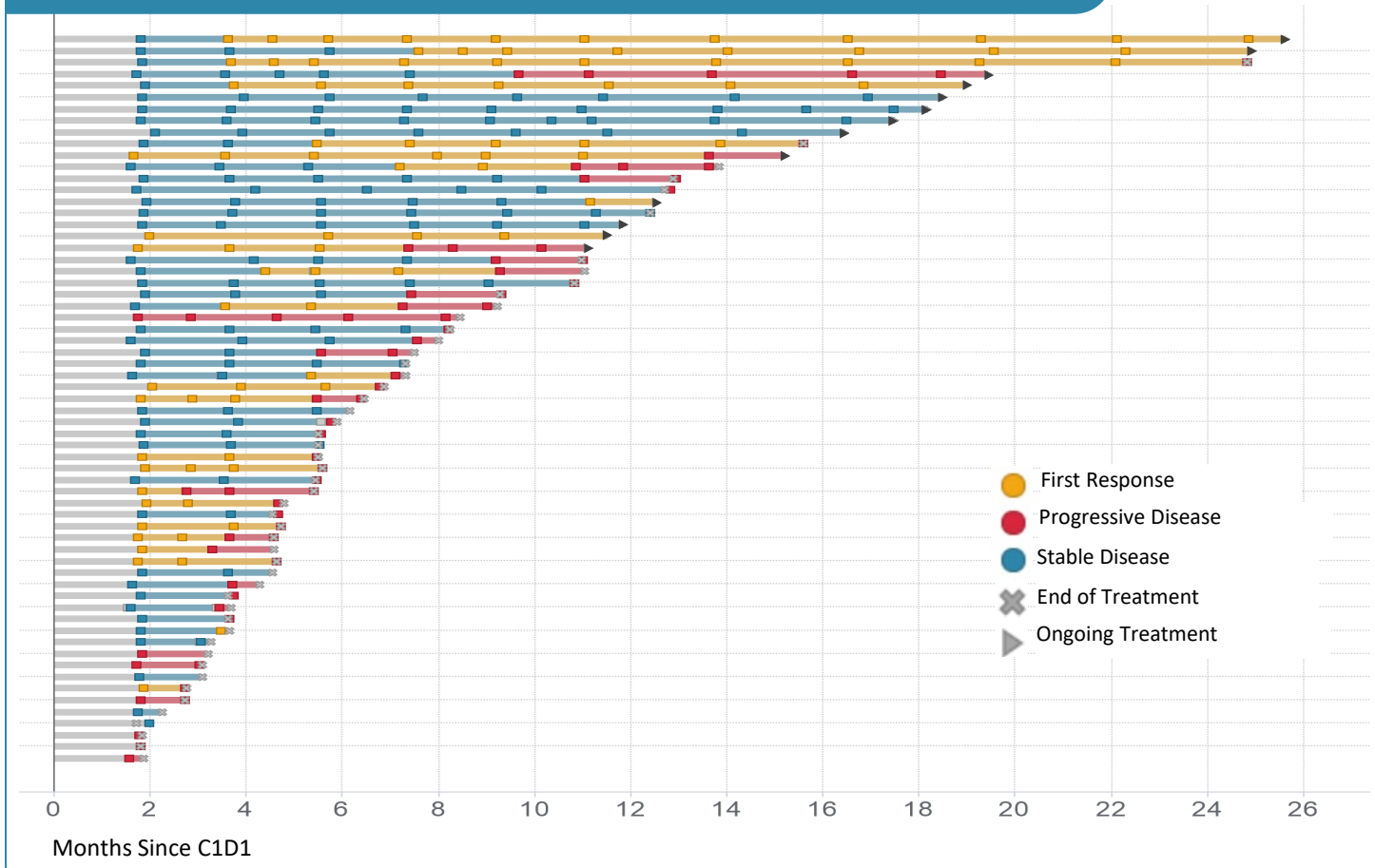
*Molecular response (MR) defined as at least 50% reduction in percentage of Mean Allele Frequency (MAF) at any timepoint

^ Best Overall Response

Median PFS in First-Line, Any-Line and Hepatic-Only MUM

Observed Compelling Median Progression Free Survival with Encouraging Trend

Any-Line MUM Swimlane Plot



Darovasertib + Crizotinib Phase 2

Median Progression Free Survival

- First-Line (n=20): 7.1 months
- Any-Line (n=63): 6.8 months
- Hepatic-Only (n=19): 11.0 months

Treatment Duration – Observations

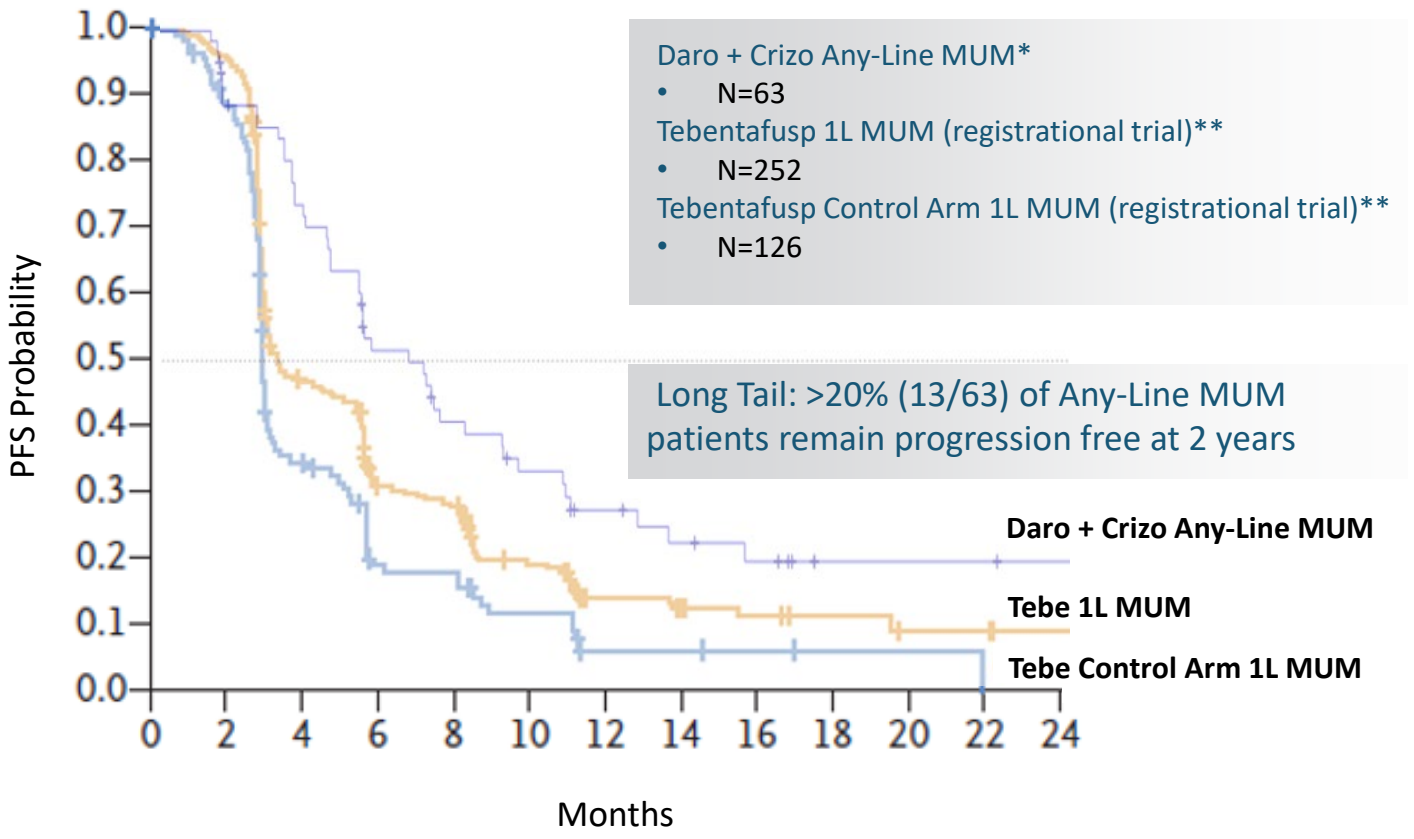
- ~50% of patients treated > 6 months
- ~30% of patients treated > 1 year

ESMO 2023 Proferred Presentation M McKean et al: Preliminary analysis of unlocked database as of 08/22/2023 by investigator review, C1D1 cutoff as of 9/22/2022, based on 63 evaluable Any-Line MUM patients

2-Year PFS Kaplan Meier (KM) Curve: Daro + Crizo in Any-Line MUM*

Daro + Crizo Combo Observes a Promising 2-Year PFS KM Curve and a “Long Tail” Effect

2-Year PFS Kaplan Meier Curves in MUM



Daro + Crizo Any-Line MUM*
- N=63

Tebentafusp 1L MUM (registrational trial)**
- N=252

Tebentafusp Control Arm 1L MUM (registrational trial)**
- N=126

Long Tail: >20% (13/63) of Any-Line MUM patients remain progression free at 2 years

* IDEAYA Data: preliminary analysis of unlocked database as of 08/22/2023 by investigator review, C1D1 cutoff as of 9/22/2022, based on 63 evaluable Any-Line MUM patients. Direct comparisons are not being made and the historical data for tebentafusp is being shared for informational purposes only

** N Engl J Med 2021;385:1196-206; Tebentafusp Phase 3 registrational trial, PFS curves

Darovasertib + Crizotinib Combination Clinical Summary in MUM

Highly Differentiated Clinical Efficacy & AE Profile Observed[+, ++]

	Darovasertib + Crizotinib	Cabozantinib Mono / Crizotinib Mono	Selumetinib + DTIC	Ipi + Nivo	Tebentafusp
Target / Mechanism	PKC + cMET	cMET	MEK + Chemotherapy	CTLA4 + PD1	HLA-A2-0201 Bi-Specific
Study Name(s)	NCT03947385	A091201^ / NCT05063058 ^^^^	NCT01974752^^^	NCT02626962[##]	IMCgp100-102[#]
Population	1L/2L/3L+ MUM (n=63)	1L+ MUM (n=31) / 1L (n=6) 2L (n=1) MUM	1L+ MUM (n=97)	1L MUM (n=52)	2L+ MUM (n=127)
Patient Selection	NA	NA / MET Overexpression	NA	NA	HLA-A2-positive
Drug Form	Oral Tablets	Oral Capsules	Oral Capsules + chemo	IV infusion	IV Infusion (Weekly)
Tolerability (Grade ≥3 Drug-Related AE)	31%	51.6% / NA	63% (All Cause)	58%	46.5%
% of Patients with Tumor Shrinkage	First-Line = 95% / Any-Line = 92% / Hepatic Only = 100%*	23% ^^ / NA	35% ^^	27% ^^	44% ^^
Confirmed ORR% (by RECIST 1.1)	**First-Line = 45% / Any-Line = 30% / Hepatic Only = 37%***	**0% / 0%**	**3%**	**11.5% (not confirmed ORR)**	**4.7%**
Median PFS	**First-Line: 7.1 months / Any-Line: 6.8 months / Hepatic-Only: 11.0 months***	**2 months / NA**	**2.8 months**	**3 months**	**2.8 months**

+ Cross-trial comparisons are not based on head-to-head studies and are presented for informational purposes; no direct comparisons are being made

++ ESMO 2022: F. Dimitriou, et. al: IPI + Nivo Combo in HLA-A2-0201 MUM reports ~6% ORR (2 PRs out of 33 patients)

* ESMO 2023 Proferred Presentation M McKean et al: Preliminary analysis of unlocked database as of 08/22/2023 by investigator review; data cutoff based on treatment Day 1 of Cycle 1 (C1D1) as of 9/22/2022

Based on Immunocore reported 2L+ study data (to reflect comparative patient population) and by independent review and ORR% was with confirmed PRs; ## ASCO 2021, J. Piulats, et. Al, Ipi = ipilimumab, nivo = nivolumab, ORR% did not require PR/CR confirmation

^ Randomized Phase II Trial and Tumor Mutational Spectrum Analysis from Cabozantinib versus Chemotherapy in Metastatic Uveal Melanoma (Alliance A091201); Clin Cancer Res 2020;26:804–11

^^ Estimated from Waterfall plot

^^^ Journal of Clinical Oncology, Carjaval, et. al, 2018; 1232-1239; ^^^^ European Journal of Cancer, Leyraz, et. al, 2022; 146-155

IDEAYA BIOSCIENCES

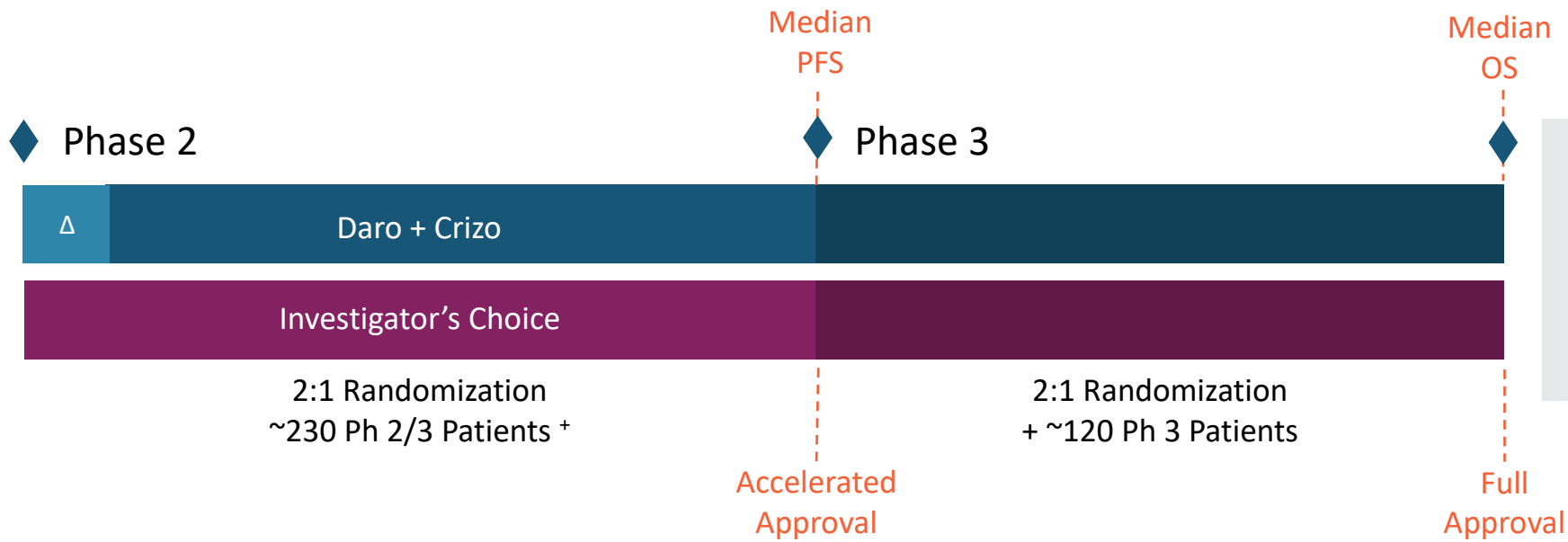
Accelerated Approval Trial Design in First-Line HLA-A2 Negative MUM

FDA Guided Design: Open Label Ph2/3 Evaluation of Daro + Crizo vs. Investigator's Choice ^

FDA Project FrontRunner: Target First-Line approval strategy to enhance patient benefit in MUM

FDA Accelerated Approval: Address unmet need in MUM, which has limited effective treatment options

Integrated Phase 2/3 Study within Study Enables Potential Accelerated Approval & Full Approval



2:1 Randomization
Daro + Crizo vs Investigator's Choice
(IC = Ipi + Nivo, PD1, or DTIC)

Primary Endpoints (BICR)
Accelerated Approval: mPFS
Full Approval: mOS

FDA Fast Track Designation for Daro + Crizo in MUM

Δ Nested study to confirm move forward dose: (i) Daro 300 mg BID + Crizo 200 mg BID or (ii) Daro 200 mg BID + Crizo 200 mg BID

+ Phase 2 study contemplates data set of n=200 patients randomized 2:1 with treatment arm at move forward dose in support of potential accelerated approval based on mPFS

Daro = Darovasertib, Crizo = Crizotinib, MUM=Metastatic Uveal Melanoma, HLA-A2 = HLA-A2*02:01 Serotype, IC = Investigator's Choice, Ipi = ipilimumab, nivo = nivolumab; DTIC = dacarbazine

^ Clinicaltrials.gov: NCT05987332

Darovasertib Monotherapy in (Neo)adjuvant Primary Uveal Melanoma

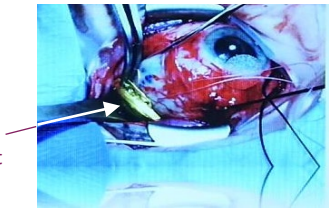
3 of 6 evaluable (50%) UM Patients Observed Eye Preservation in Enucleation Cohort^

IDEAYA Phase 2 and IST ongoing to evaluate Darovasertib

Current Treatment Approach following diagnosis of UM depends on tumor size and location:

- Enucleation in Large Tumors (~20%)
- Radiation in Small / Medium Tumors (~80%)

Plaque Brachytherapy



Iodine-125 Plaque Surgery, UCLA

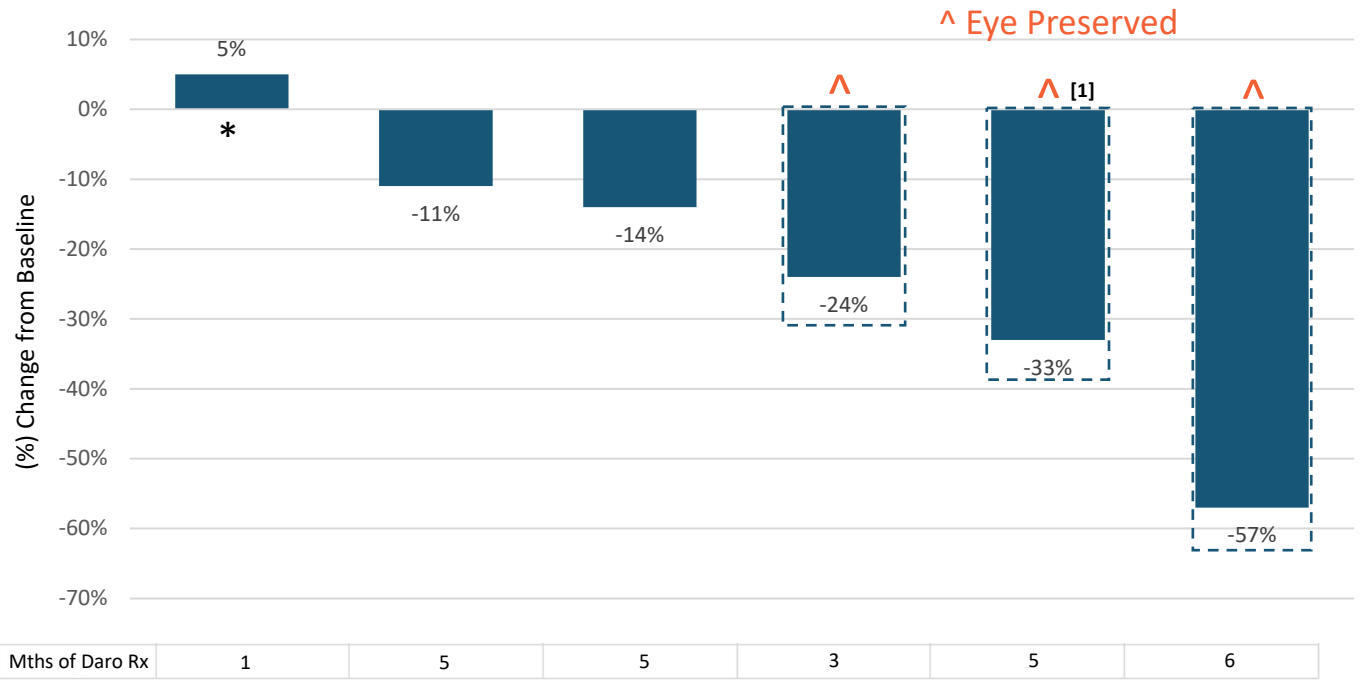
Poor Vision (≤20/200) occurs in about 70%-80% of patients with UM (including enucleation)

Neoadjuvant / Adjuvant Systemic Therapy goals:

- Avoid Enucleation → Save the Eye
- Reduce Tumors and Radiation Dose → Protect Vision
- Reduce Occurrence of Metastasis → Save Lives

Paradigm Shifting Opportunity to Broadly Impact UM, with annual incidence of ~8,000 – 10,000 patients in US, EU

Evaluable UM Patients Treated to Maximal Benefit Φ+



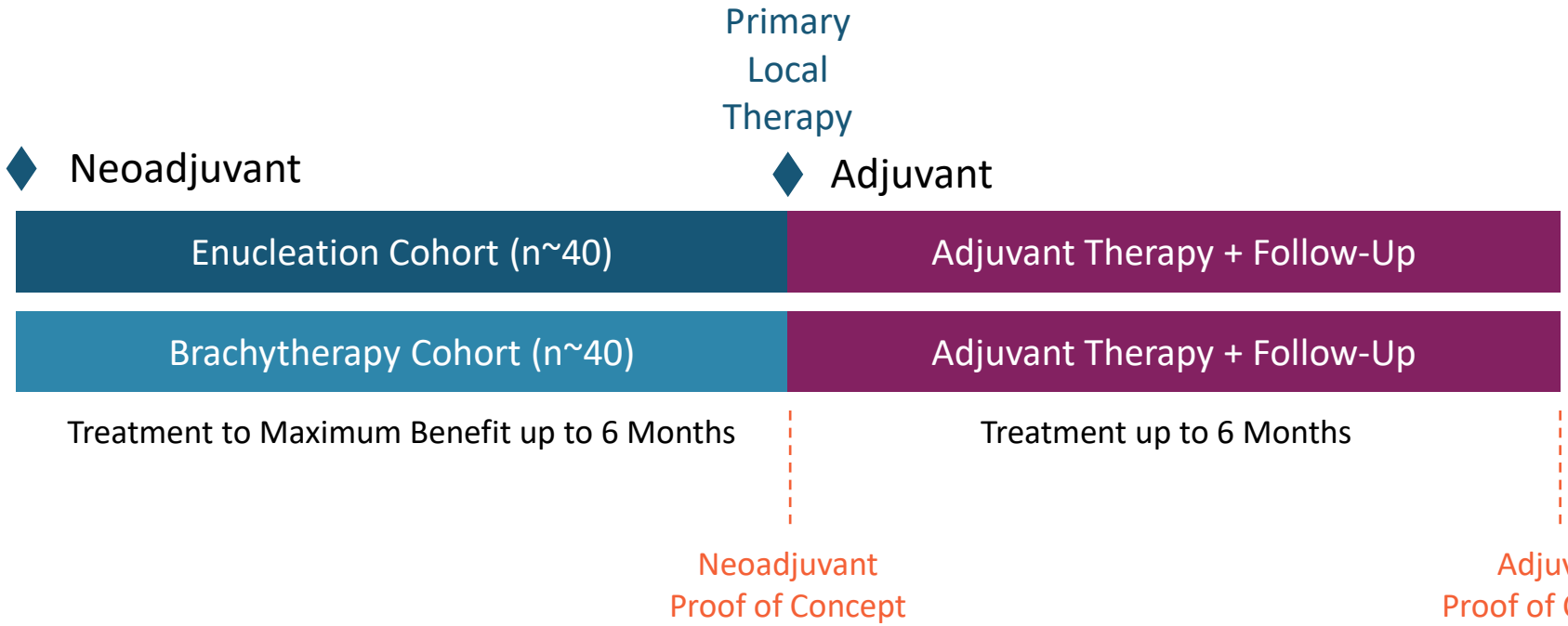
2 out of 4 additional patients after the enrollment cutoff date are likely plaque eligible (20% tumor reduction in 2 months, 22% tumor reduction in 1 month) and continuing on darovasertib until maximal benefit

Φ Data by investigator assessment with enrollment cut-off of July 17, 2023, from NADOM IST courtesy of Professor Anthony Joshua, MBBS, PhD, FRACP, St. Vincent's Hospital
+ Maximal % reduction in measured apical height or longest basal diameter
* Patient had sub-retinal blood present at baseline and with lack of shrinkage and visual deterioration discontinued after 6 weeks. One additional patient had Grade 3 drug related dermatitis and discontinued treatment before 1st scan.
[1] Patient was plaque-eligible and ongoing with darovasertib neo-adjuvant treatment to maximal benefit

(Neo)Adjuvant Darovasertib in Primary Uveal Melanoma (UM)

Paradigm Shifting Opportunity to Save the Eye, Protect Vision and Save Lives

Phase 2 Study of Darovasertib Monotherapy as Neoadjuvant Therapy followed by Adjuvant Therapy ^



Primary Endpoints
- Safety
- Cohort 1: Eye Preservation
- Cohort 2: Decrease in Radiation Dose

Secondary Endpoints
- Useful Vision (1 yr)
- Relapse Free Survival (RFS)
  - Local (1 yr)
  - Distant (3 yr)

Three Independent Approaches for demonstrating Clinical Benefit and defining potential Approval Pathways

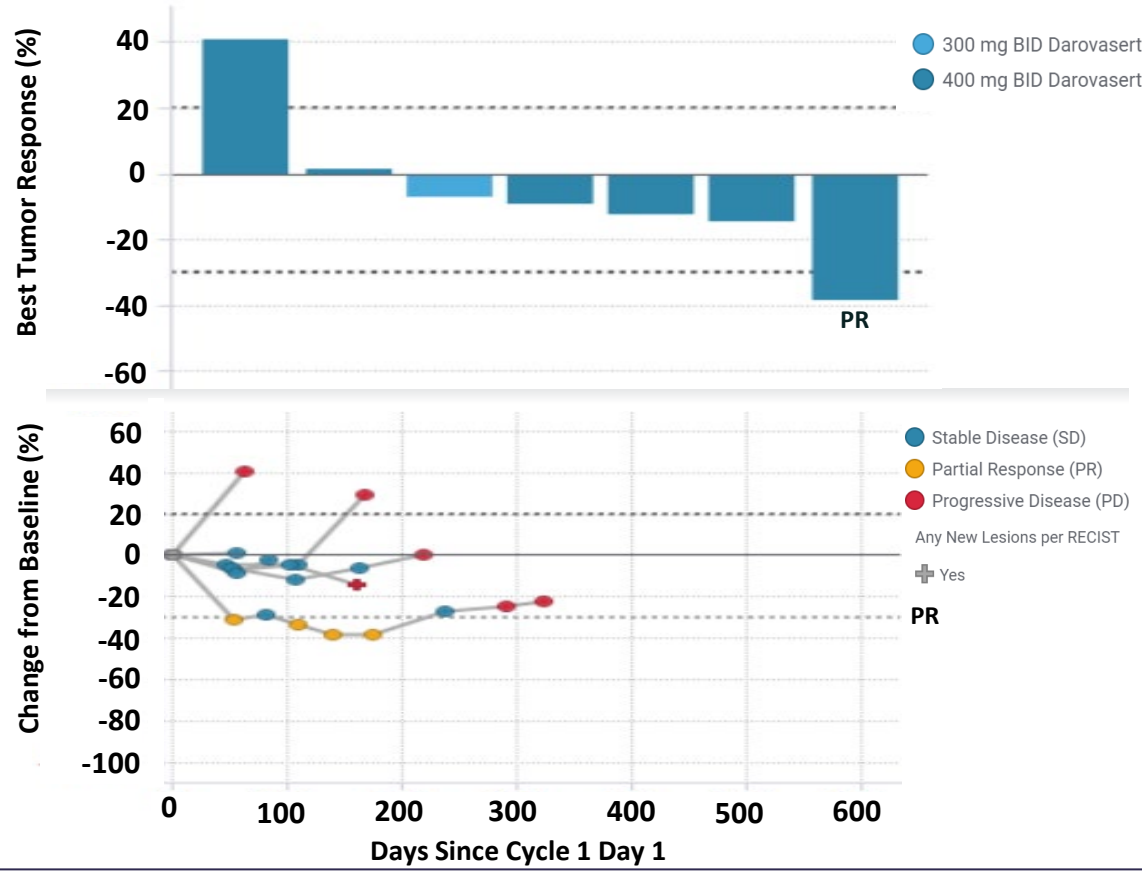
Enucleation Cohort → Save the Eye Brachytherapy Cohort → Protect Vision Adjuvant Therapy → Save Lives

^ Clinicaltrials.gov: NCT05907954

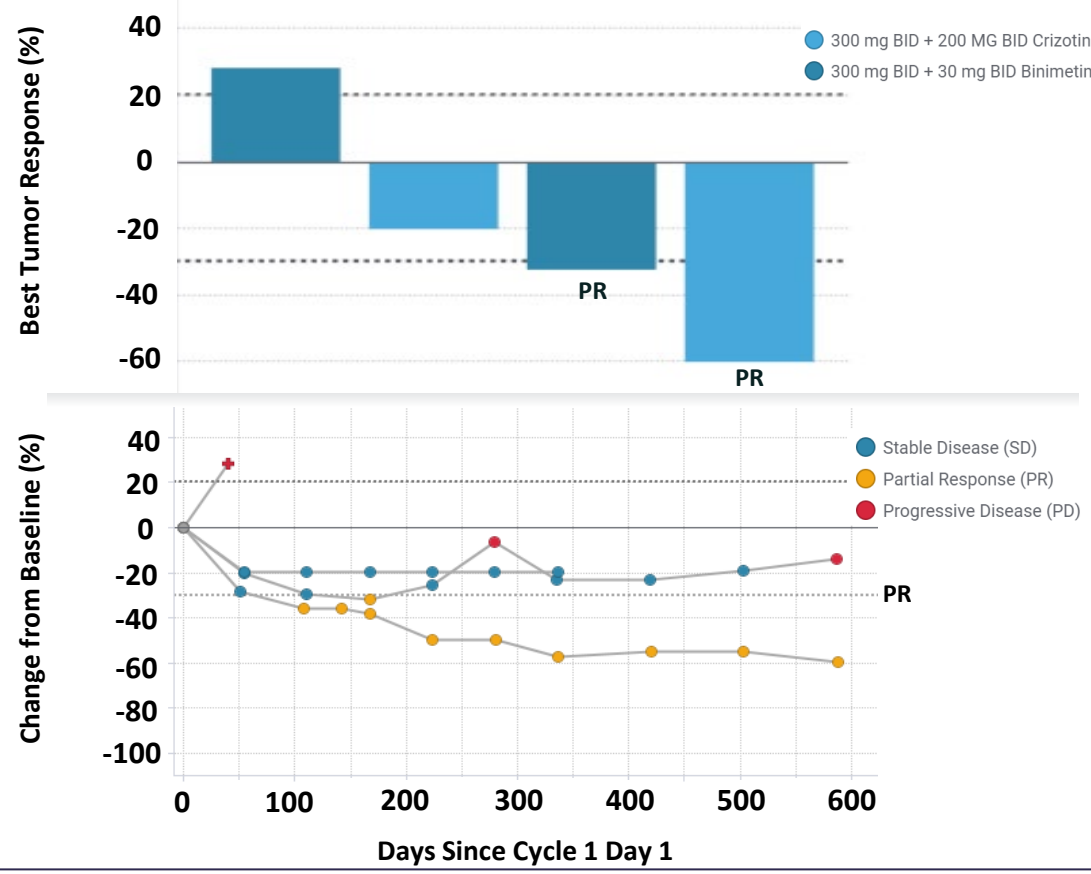
GNAQ/11 Cutaneous Melanoma Patients Treated With Darovasertib

2 of 4 (50%) Observed Durable Partial Responses by RECIST 1.1 with Daro Combination

Cutaneous Melanoma Patients Treated with Daro Mono (n=7)



Cutaneous Melanoma Patients Treated with Daro Combo (n=4)



IDEAYA Data: preliminary analysis of unlocked database as of 24Dec2023 by investigator review

Darovasertib Clinical & Commercial Strategy in Uveal Melanoma and CM

High Unmet Need and Multiple First-Line Opportunities

Uveal Melanoma Patient Journey

	Neoadjuvant UM	Adjuvant UM	MUM	Metastatic CM
HLA-A2-Negative (~70% of UM / MUM)**	No FDA Approved Therapies* **Daro** **Phase 2** **Enucleation** **Define Accelerated Approval Path** **Daro** **Phase 2** **Radiation** **Define Accelerated Approval Path**	No FDA Approved Therapies* **Daro** **Phase 2** **Define Accelerated Approval Path**	No FDA Approved Therapies* **Daro + Crizo** **Registrational Trial** **Accelerated Approval**	**Daro + Crizo** **Phase 2** Define Accelerated Approval Path
HLA-A2-Positive (~30% of UM / MUM)**			**Daro + Crizo** **Target NCCN / Compendia Listing**	
Target Treatment Duration	≥6 months	≥6 months	mPFS + ~3 months	mPFS + ~3 months
Target Clinical Endpoints	Eye & Vision Preservation	Relapse Free Survival	ORR, mPFS, mOS	ORR, mPFS, mOS
Annual Incidence US/EU**	~8-10k	~8-10k	~4-5k	>5K [1]
Total Prevalence US/EU**	~100k	~100k	~14k	~180K[2]

+95% of UM and ~5% of Cutaneous Melanoma (CM) patients harbor GNAQ/GNA11 mutation

FDA Orphan Drug Designation in Uveal Melanoma⁺

*No FDA approved systemic therapies in multiple UM and MUM indications across the patient journey

**IDEAYA data: ~70% of MUM patients were HLA-A2-negative based on 149 patients tested for HLA-A2 status as presented at ESMO 2023; US/EU MUM annual incidence and total prevalence based on market research analysis

⁺ Orphan Drugs benefit from certain tax credits and may be excluded from certain mandatory price negotiation provisions of the 2022 Inflation Reduction Act

[1] GNAQ/11 cutaneous melanoma estimated annual incidence is approximately 5,000 patients in the US and 8,000 patients in the EU28. Based on several metastatic cancer patient databases, including Memorial Sloan Kettering Cancer Center (MSKCC) Impact, we project GNAQ/11 metastatic cutaneous melanoma has the potential to double or more the annual addressable metastatic patient population of metastatic uveal melanoma alone

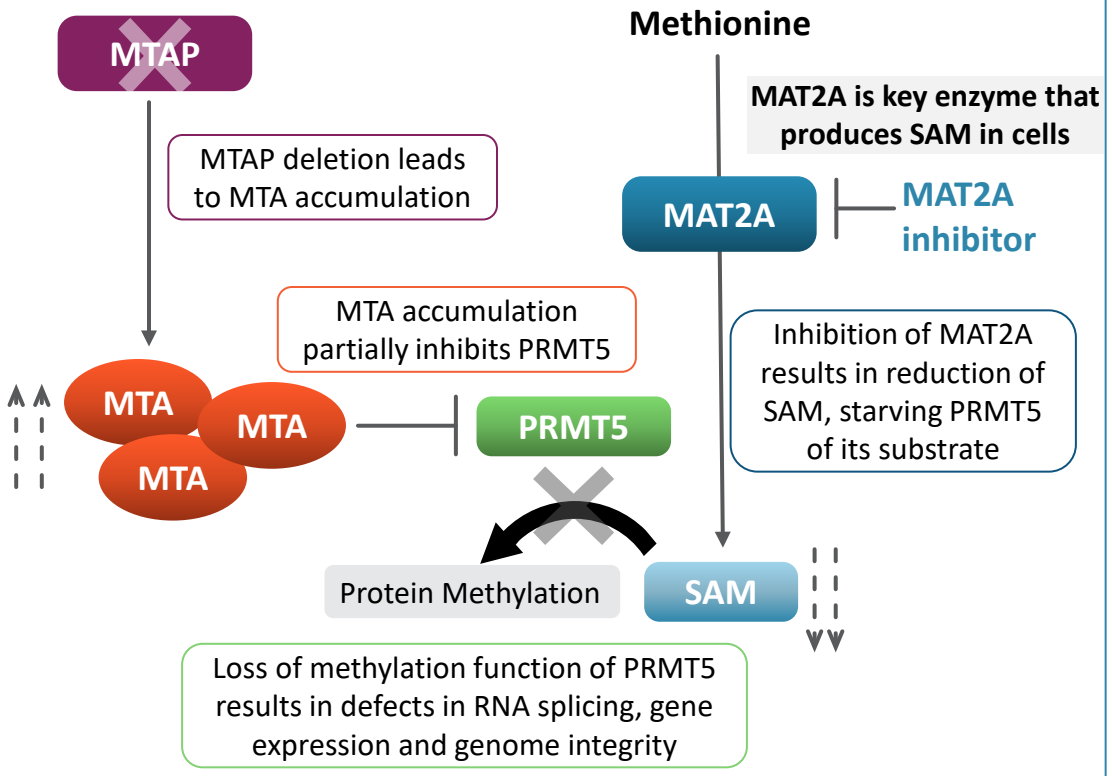
[2] The estimated total prevalence of primary GNAQ/11 cutaneous melanoma is approximately 70,000 patients in the US and 110,000 patients in the EU28

IDEAYA BIOSCIENCES

MAT2A Inhibition is Synthetic Lethal with MTAP-Deletion

Strategies to address MTAP$^{-/-}$ Prevalence in ~15% of all Solid Tumors

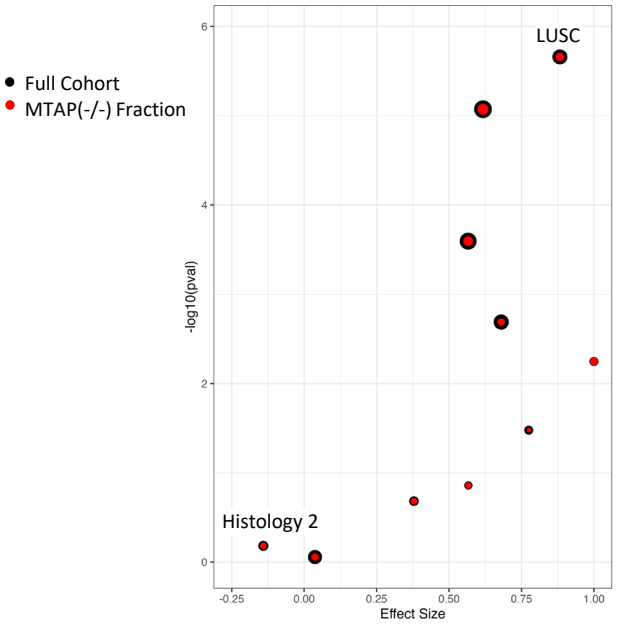
MTAP-MAT2A Synthetic Lethality Biology

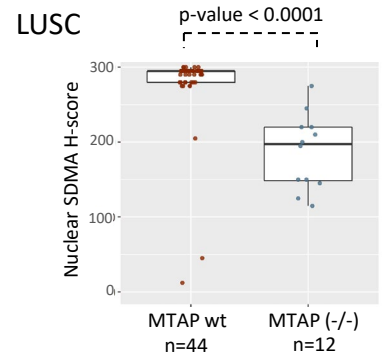


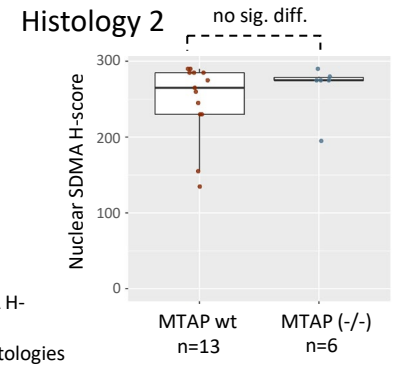
Endogenous Suppression in MTAP$^{-/-}$ PDX Models

Methylation Pathway Suppression in MTAP(-/-) Squamous Lung (LUSC)

SDMA Effect Size (PDX Tissue Microarray)







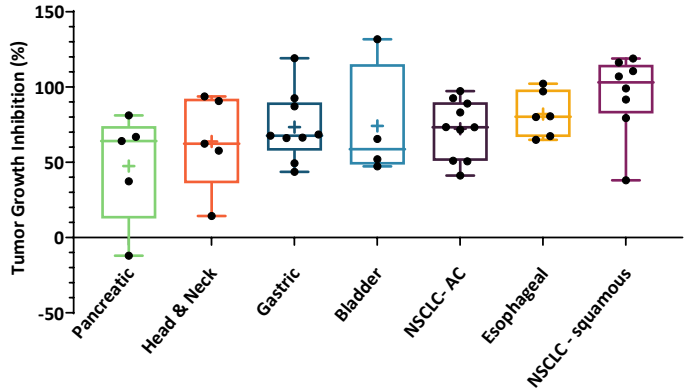
IDEAYA Data: AACR 2023 (M. Fischer et al.) – Volcano plot comparing nuclear SDMA H-Score by IHC in MTAP(-/-) relative to MTAP wt across tissue microarray (TMA) of treatment-naïve PDX models; LUSC shows most significance effect across tumor histologies

IDE397 Demonstrates Broad Efficacy across MTAP-Deletion PDX Models

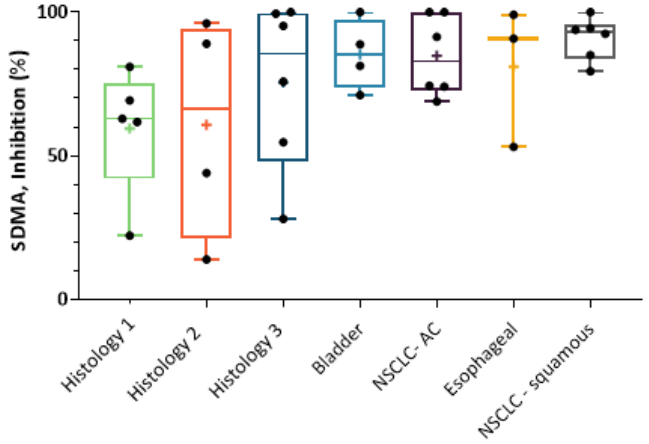
Deep Regressions observed in NSCLC-squamous (LUSC), Esophageal and Bladder Cancers

IDE397 Efficacy: 47 MTAP$^{-/-}$ PDX Models

TGI with IDE397 (30mpk) in MTAP-/- PDX Panel



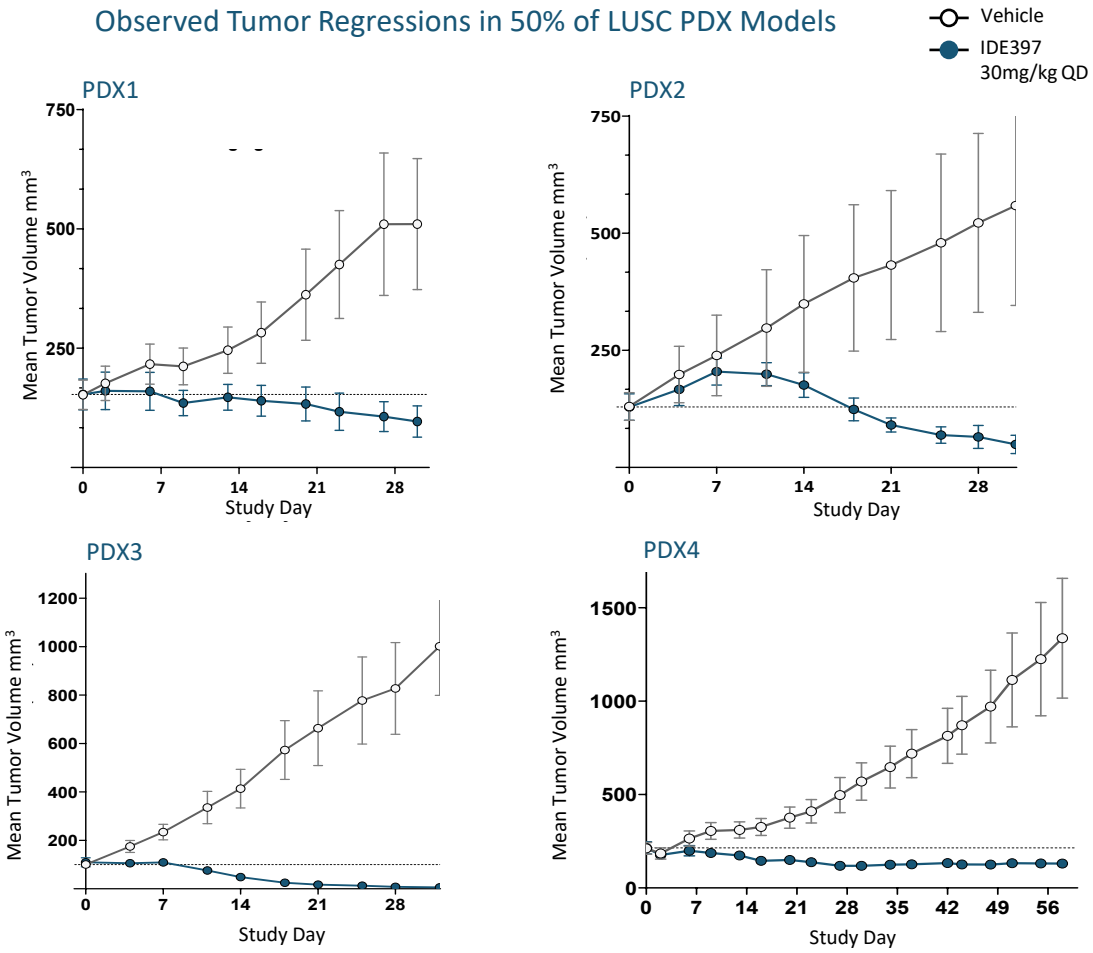
SDMA Suppression in Residual Tumors* at End of Study



IDEAYA Data; *2 of 8 LUSC unevaluable due to insufficient residual tumor burden

IDE397 In Vivo Efficacy in LUSC PDX Models

Observed Tumor Regressions in 50% of LUSC PDX Models



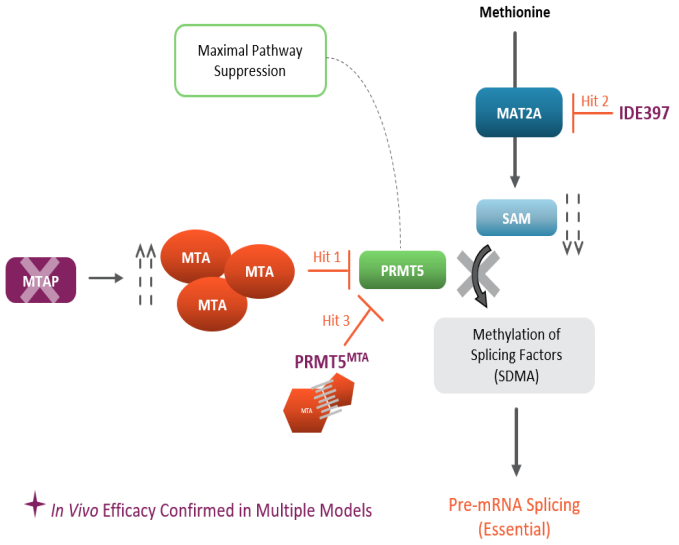
IDEAYA Data

IDE397 Clinical Combination Strategy in MTAP-Deletion NSCLC

Phase 1 Study of IDE397 + AMG 193 (Amgen PRMT5) Clinical Combination Enrolling

IDE397 + MTA-Cooperative PRMT5i

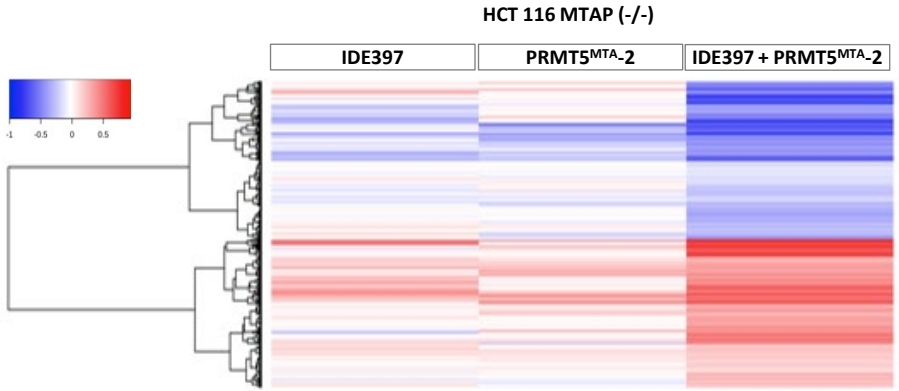
IDE397 + MTA-Cooperative PRMT5 Inhibitor enables Maximal Pathway Suppression



Enhanced Combination Efficacy Observed in multiple Tumor Indications and Across Representative PRMT5^{MTA} Inhibitors

Alternative mRNA Splicing Analysis

Combination Highly Perturbs Splicing Fidelity



Quantitative Assessment of IDE397 / PMRT5i Effect on pre-mRNA Splicing

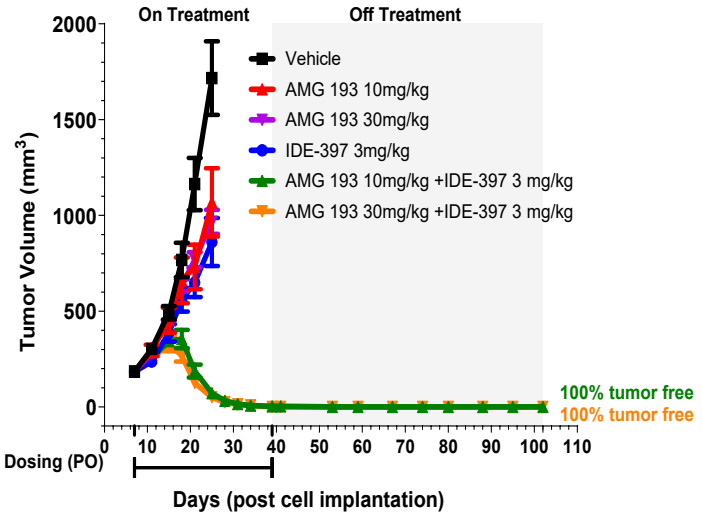
⬇

>2800 significant Splicing Events only in the Combination Treatment Arm+

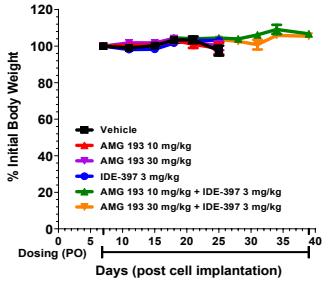
Identified as novel splice junctions or as not meeting significance criteria in monotherapy arms
Color = heatmap of Z-scored TMM-normalized counts per million

Preclinical Efficacy

Observed Durable Complete Responses



No Body Weight Loss



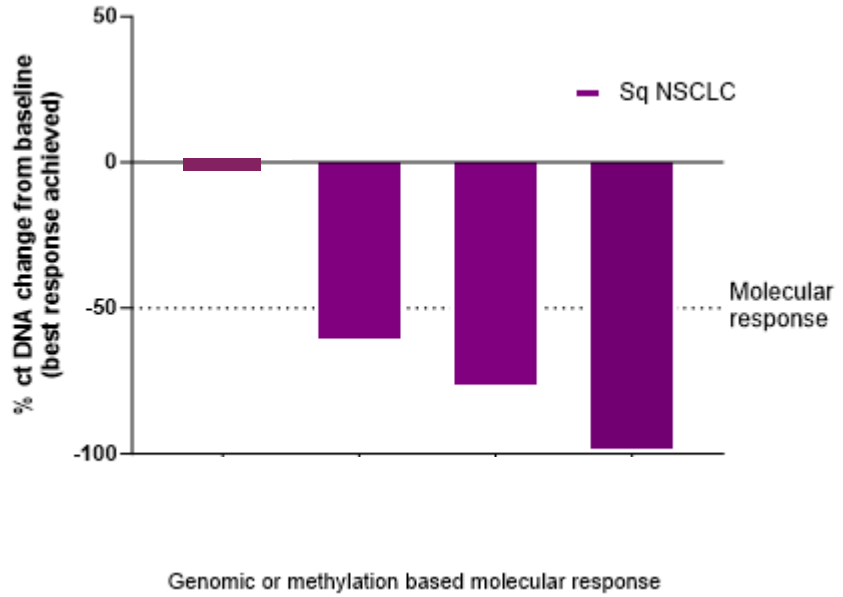
IDEAYA / Amgen Data: AACR 2023, M. Fischer et al.

IDE397 Phase 2 Monotherapy Expansion in MTAP-Deletion Squamous NSCLC

Robust Tumor Shrinkage and ctDNA Molecular Responses Observed

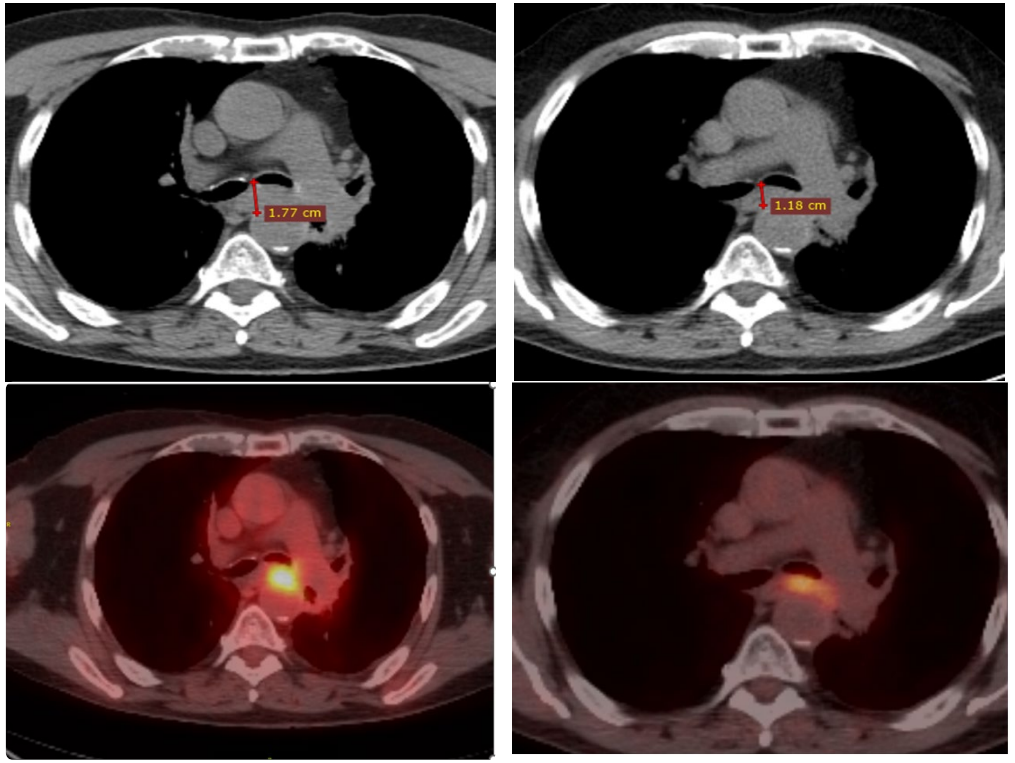
Best Response from Baseline among subjects with Squamous NSCLC with evaluable ctDNA samples*

75% Molecular Response Rate



33% shrinkage by PET/CT and decreased hypermetabolism noted in the mediastinal nodal mass in Squamous NSCLC patient**

Baseline PET/CT | **12-week on study PET/CT**



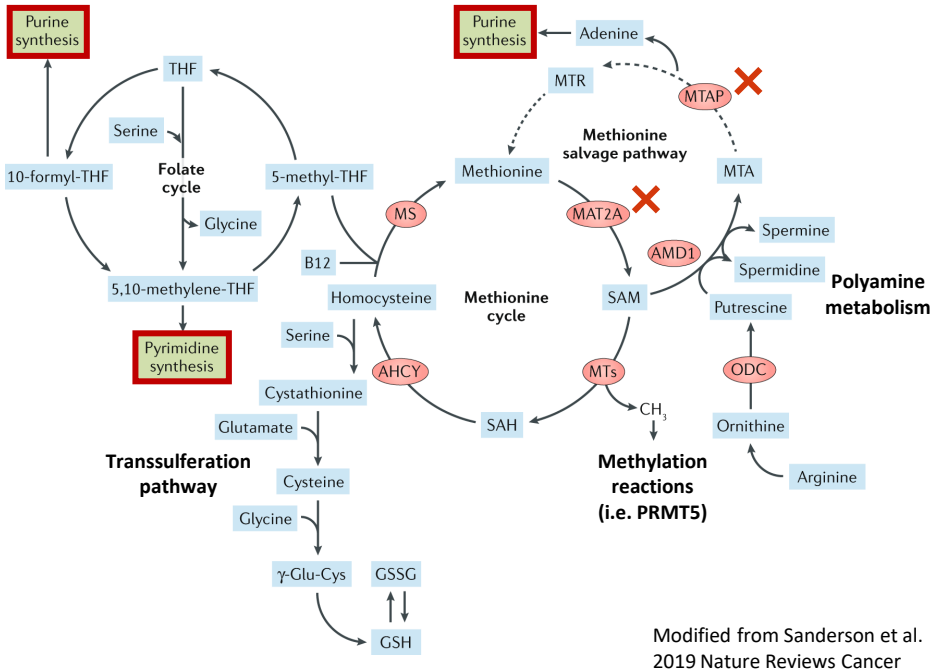
*1 patient sample failed QC for ctDNA analysis

IDEAYA Data: preliminary analysis of unlocked database as of November 21, 2023
** Radiologic and Clinical response noted (decreased dyspnea and hoarseness) in recurrent tumor in the mediastinum after prior platinum chemotherapy and consolidation anti-PD1 antibody treatment

TRODELVY® + IDE397 Combination in MTAP-Deletion Urothelial Cancer

Disordered methionine metabolism underpins IDE397 combination opportunity with TOP1i-based ADC

IDE397 perturbs both nucleotide pools and mRNA splicing in MTAP$^{-/-}$ cells

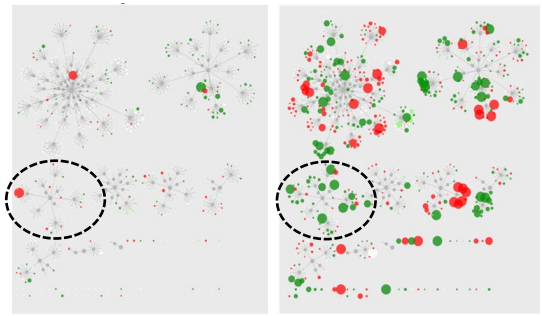


Modified from Sanderson et al. 2019 Nature Reviews Cancer

Metabolic perturbation by IDE397 selectively interacts with MTAP

Metabolite Cytoscape

Global (untargeted) metabolic profiling of MTAP^{wt} vs MTAP$^{-/-}$ +/- IDE397

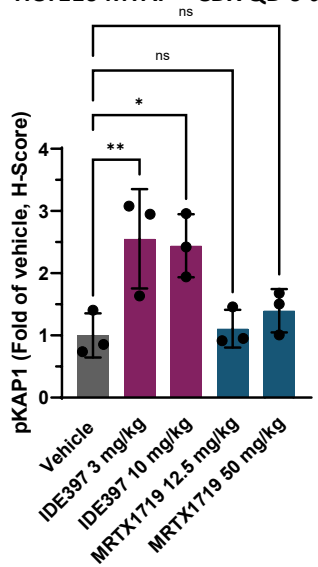


MTAP WT +/- IDE397 **MTAP$^{-/-}$ +/- IDE397**

Ovals indicate nucleotide subcluster (purine/pyrimidine); green-decrease, red-increase FDR< 0.05

IDE397 provokes DDR response in vivo

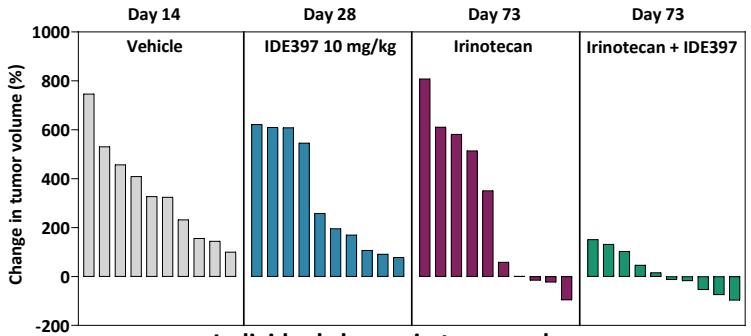
HCT116 MTAP$^{-/-}$ CDX QD 6 days



Key clinical correlates underscore combination opportunity

- MTAP$^{-/-}$ UC: shorter survival, lower RR to CPI, shorter PFS and OS with Enfortumab-Vedotin (EV)
- MTAP$^{-/-}$ status is associated with pemetrexed antitumor activity in UC
- A small study evaluating Trodelvy activity in UC post-EV suggested preferential activity in MTAP$^{-/-}$ tumors (RR 50% vs. 19% post EV)
- IDE397 has monotherapy efficacy in MTAP$^{-/-}$ UC (CR observed in a patient with short DFI post platinum and CPI, other tumor reductions and molecular responses seen)

TOP1i combination delivers durable benefit in challenging RT112/84 UC CDX model



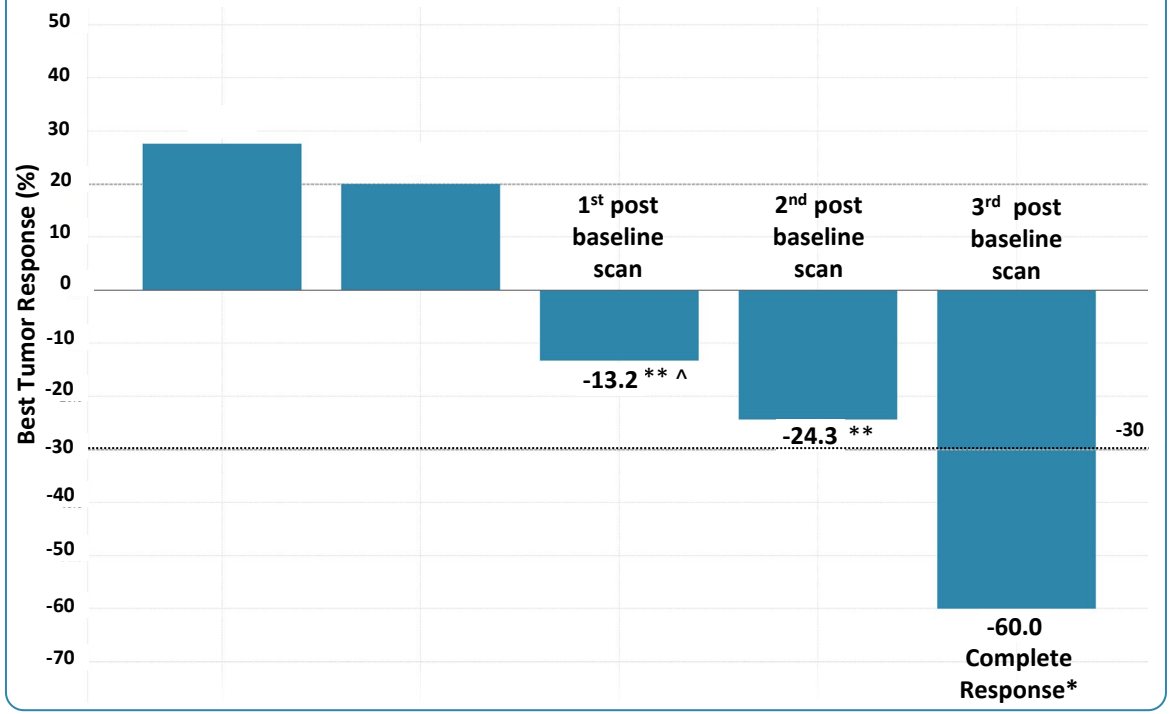
Individual change in tumor volume

Individual tumors measured on day of study group termination as indicated; termination timing was based on endpoint criteria for tumor volume

IDE397 Phase 2 Monotherapy Expansion in MTAP-Deletion Urothelial Cancer

Robust Tumor Shrinkage and ctDNA Molecular Responses Observed

Best Overall Response by RECIST 1.1 in Urothelial / Bladder Cancer

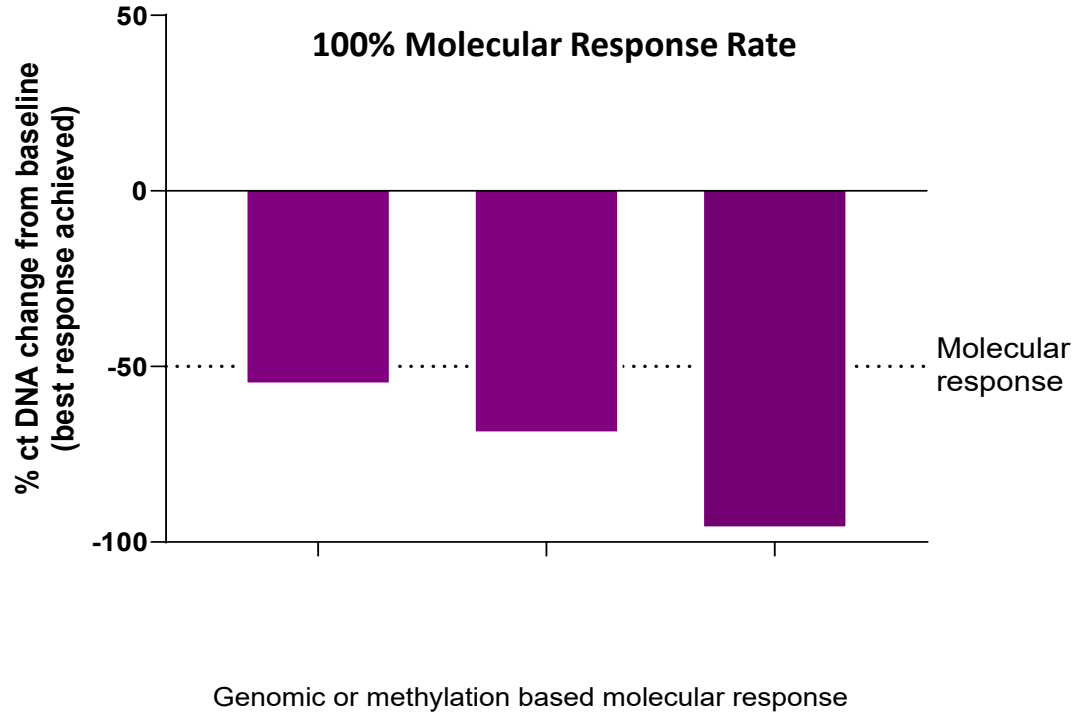


IDEAYA Data: preliminary analysis of unlocked database as of November 21, 2023

* Decrease of all nodes selected as target lesions to < 10mm in short axis is assessed as a complete response per RECIST v1.1 in cases where no other target lesions are present at baseline. Target lesion sum for lymph nodes may not be zero even if CR criteria are met

** Patients had visceral metastases with target lesions in the liver and lung. ^ 6-week on study scan

ctDNA: Best Response From Baseline in Urothelial / Bladder Cancer*



IDEAYA Data: preliminary analysis of unlocked database

*2 of the patient samples failed QC for ctDNA assessment

IDE397 Phase 1/2 Clinical Development Plan in MTAP-Deletion Solid Tumors

Strategic Focus in Select Monotherapy Indications and High Conviction Clinical Combinations

IDE397 – Clinical Profile

Exposure-Dependent Pharmacokinetic (PK) Profile with low C_{max}:C_{min}

Robust Pharmacodynamic (PD) Response observed

Monotherapy Expansion demonstrates clinical efficacy with Responses in Multiple High-Priority Tumor Types in Dose Expansion, including a Complete Response

IDE397 is strategically well positioned to evaluate both monotherapy and clinical combinations in MTAP-deletion solid tumors

IDE397 Monotherapy Expansion in Select MTAP-Deletion Solid Tumor Types

- IDE397 RDE Monotherapy Expansion: Squamous NSCLC and Bladder Cancer
- Focus or Additional Indication Expansion

PRMT5 Combination – Targeting Clinical PoC in MTAP-Deletion NSCLC

AMGEN

- IDE397 + AMG 193 MTA-Cooperative PRMT5i#^[1]: Expansion Focus in NSCLC
- Other Potential Indications and/or Combinations

IDE397 + AMG 193
Enrollment
Ongoing

Trop2-ADC Combination – Targeting Clinical PoC in MTAP-Deletion Bladder Cancer

GILEAD

- IDE397 + Trodelvy®*: Bladder Cancer

IDE397 + Trodelvy®
Targeting
Mid 2024 FPI

Addressable MTAP-Deletion Annual Incidence of >50,000 patients in the US, EU5 and Japan across priority solid tumor types of NSCLC, bladder, gastric, and esophageal cancers

AMG 193 = Amgen's investigational MTA-cooperative PRMT5 inhibitor; * Trodelvy® = Gilead's Trop-2 directed ADC

^ Amgen reported initial clinical data at the 2023 AACR-NCI-EORTC that showed 5/18 (ORR=28%) for AMG 193 monotherapy expansion at dose > 800 mg QD in esophageal, pancreatic, renal cell, gallbladder, and Sertoli-Leydig cell cancers

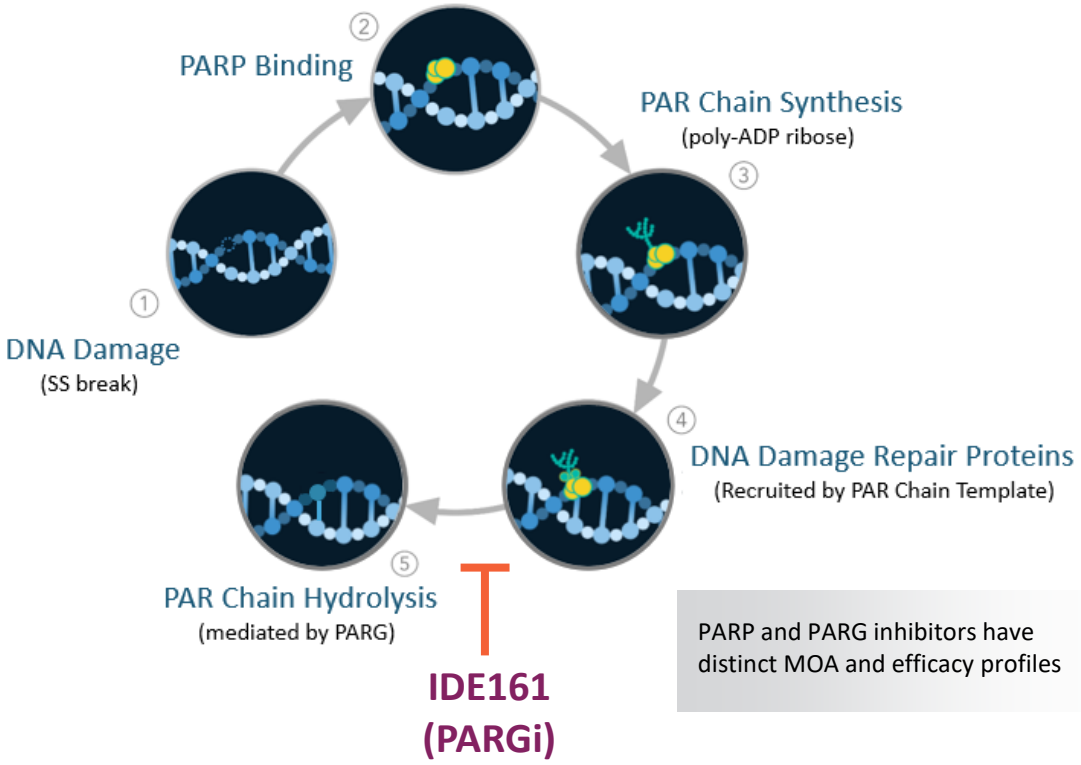
[1] Clinicaltrials.gov: NCT05975073

IDEAYA BIOSCIENCES

PARG Inhibition is Synthetic Lethal with HRD

Novel, Mechanistically-Differentiated Target in a Clinically Validated Pathway

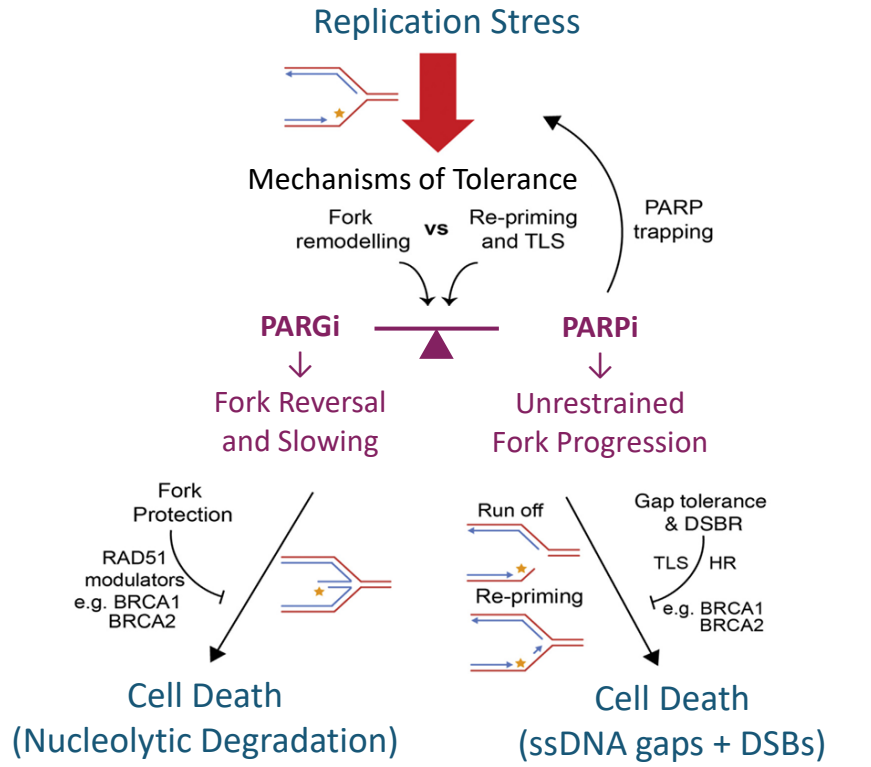
PARG Activity is required to resolve DNA Repair



PARP and PARG inhibitors have distinct MOA and efficacy profiles

Cancer Research (Aug 2019), Cancer Research (Jul 2019), Cancer Cell (Mar 2019)

PARG Inhibition is Mechanistically Distinct from PARPi

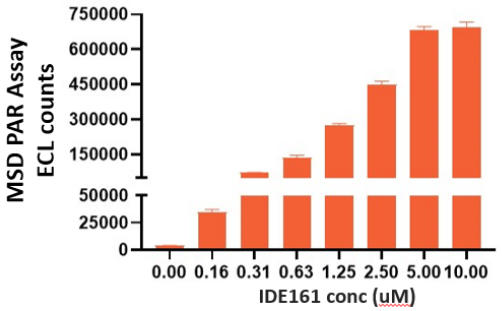


Pillay et al., Progress in Biophysics and Molecular Biology 2021; McDermott et al., Cancer Cell 2019; Zeman and Cimprich, Nature Cell Biology 2014

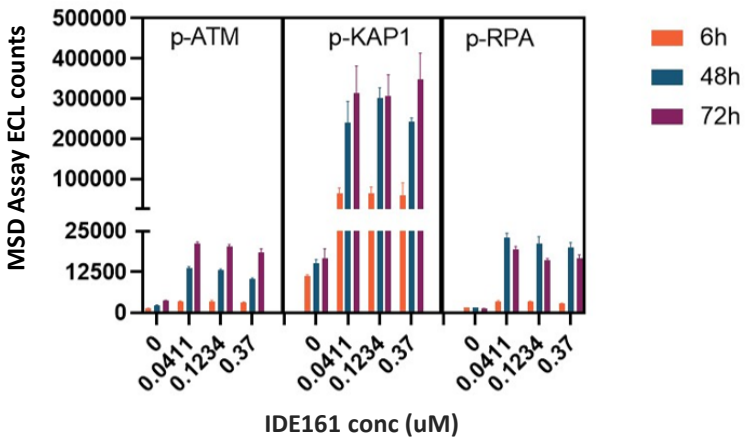
IDE161: Potential First-in-Class Phase 1 PARG Inhibitor

IDE161 Profile: Potent, Selective with Favorable Properties

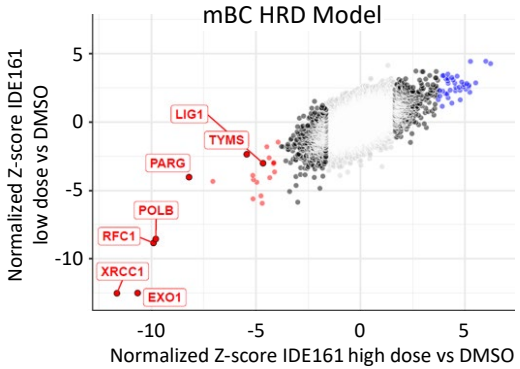
IDE161-induced Cellular PAR Accumulation

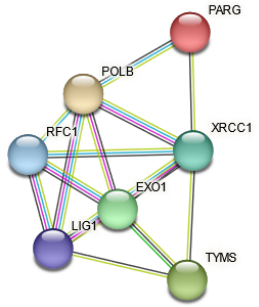


IDE161-induced DNA Damage Response



IDE161 is Synthetic Lethal with BER Gene Disruption



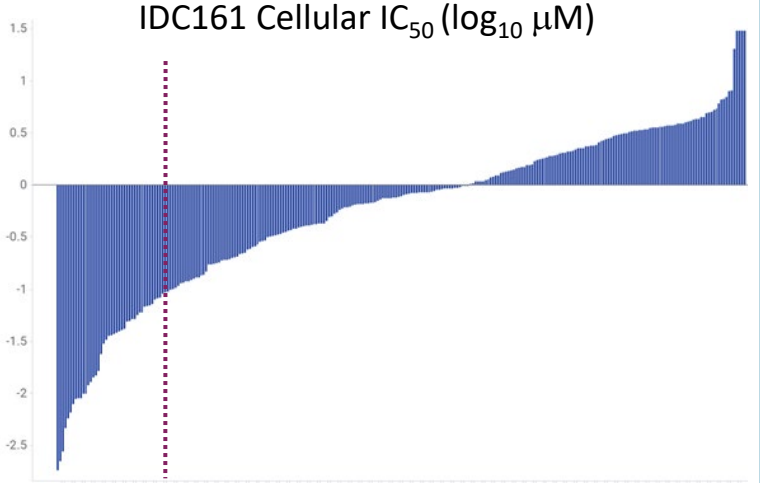


Gene Network generated from whole genome CRISPR screens anchored with IDE161 using significant synthetic lethality hits across 4 models

IDE161 Sensitivity Profile in Cell Panel

Cellular response profiles reveal mechanistic associations with PARGi sensitivity

IDC161 Cellular IC_{50} (log_{10} µM)



314 cell lines across 31 lineages

Top predictive response biomarkers* include HRD, replication stress, nucleotide excision repair and parylation cycle

* Based on univariate and multivariate pan-cancer and lineage-specific molecular feature analysis

IDEAYA Data: AACR 2023 D. Munoz et al.

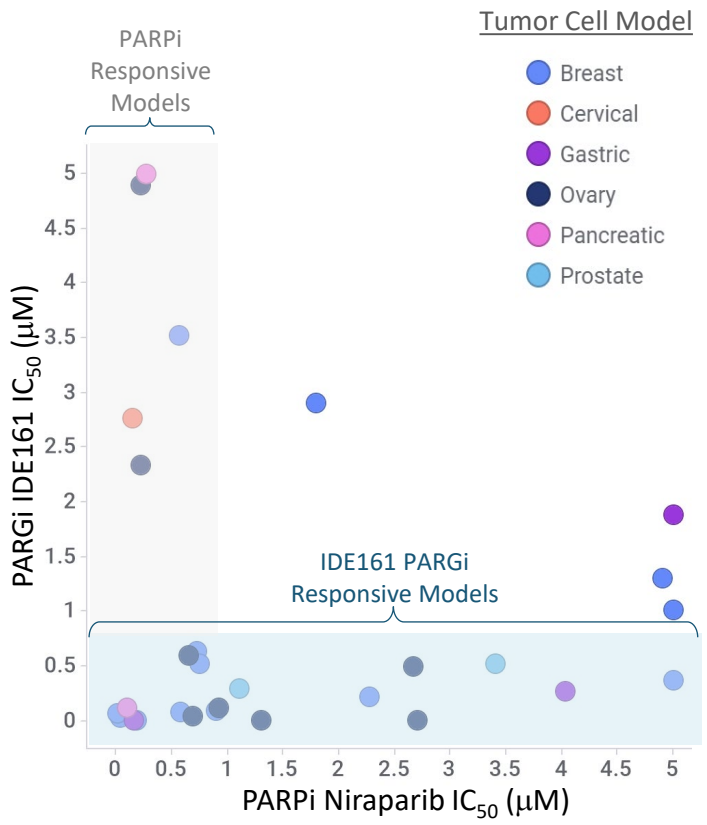
PARG = poly (ADP-ribose) glycohyrdolase; PAR = poly (ADP-ribose); DDR = DNA Damage Response; HRD = Homologous Recombination Deficiency; BER = Base Excision Repair, mBC = Metastatic Breast Cancer

IDE161 is Active and Well-Tolerated in HRD ER+ Her2- Breast Cancer Models

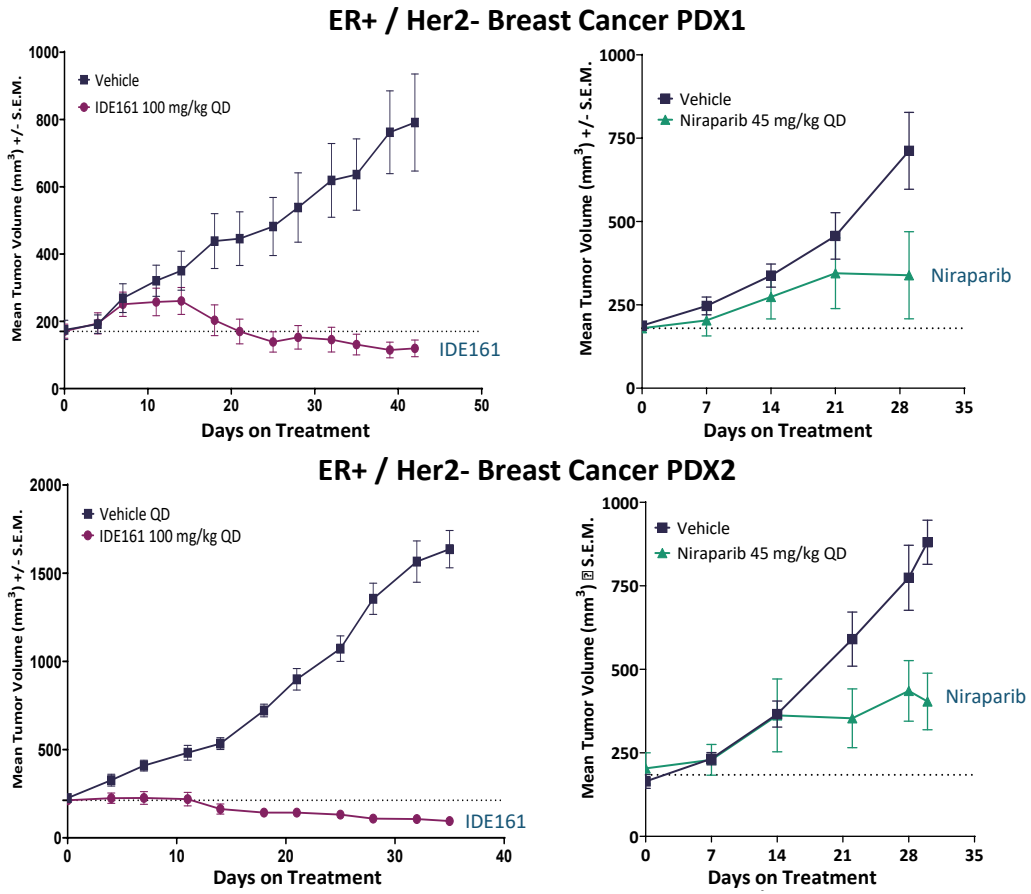
Observed PARG inhibitor Activity is Distinct from PARP Inhibition

IDE161 Selective Sensitivity vs PARPi

HRD cell lines are selectively sensitive to IDE161 versus PARPi



Regression in BRCA-altered Breast Cancer PDX Models



IDEAYA Data: 2023 AACR, D. Munoz et al.

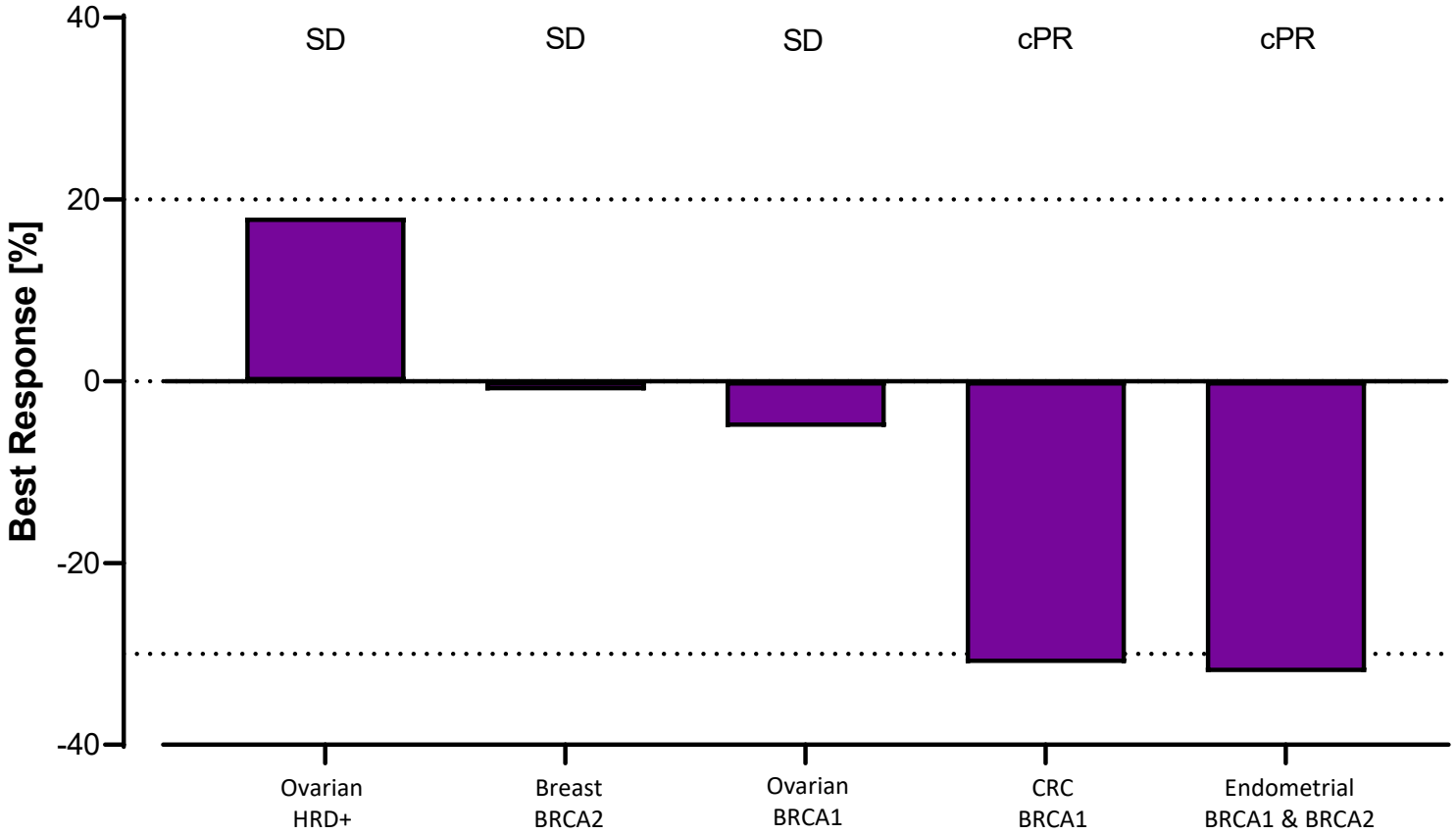
Preliminary IDE161 Clinical Efficacy at Phase 1 Expansion Dose

2 PRs by RECIST 1.1 and 100% DCR in Priority Solid Tumor Types with HRD

Initial tumor scans support favorable efficacy of IDE161 in HRD solid tumors:

- 100% DCR (5 of 5): 4 patients with tumor shrinkage & 3 Stable Disease
- Partial Response in a CRC patient at 2nd scan which was subsequently confirmed.
- Patient with Endometrial Cancer showed 87% reduction in CA125 (2760 U/mL at baseline and 360 U/mL at nadir). First scan showed Partial Response with 31% reduction in tumor size which was confirmed on subsequent scan.
- Fast track designation granted for BRCA1/2 HR+HER2- BC and ovarian cancer post PARPi therapy

Subjects with Priority Tumor Types at Phase 1 Expansion Dose



IDEAYA Data: Preliminary analysis of unlocked database as of 09Nov23; data cutoff based on treatment Day 1 of Cycle 1 (C1D1) of 21Aug23 in patients at clinically relevant doses in priority tumor types; SD = Stable Disease, cPR = confirmed Partial Response

IDE161 Phase 1/2 Clinical Development Plan in HRD Solid Tumors

Strategic Focus in Endometrial, Colorectal, Prostate, Breast & Other Solid Tumor Types

IDE161 Phase 1/2 – Monotherapy and Combination Clinical Development Plan

IDE161 Monotherapy Dose Escalation and Expansion in HRD Solid Tumors[1]

- Dose Escalation
- Expansion Cohort: ER+, Her2-, HRD Breast Cancer
- Expansion Cohort: HRD Tumors (EC, CRC, Prostate Cancer)
- Expansion Opportunities beyond HRD Tumors

IDE161 Combination Opportunities Validated Preclinically

- Dose Escalation
- IDE161+ Rational Combination Assets

Activity in PARPi- and Platinum-Resistant Settings	Differentiated Sensitivity relative to PARPi's	Targeting Improved Safety Profile relative to PARPi's

Preliminary IDE161 monotherapy clinical efficacy observed, including RECIST 1.1 Responses and >50% reduction in PSA

ER+, Her2- Breast Cancer Patients with HRD Tumors → ~10% to ~14% of Breast Cancer

Facile peripheral PD Biomarker for PARGi based on measurement of PAR in blood samples (PBMC's)

FDA Fast Track Designation for IDE161 in BRCA1/2 Ovarian and Breast Cancers*

*Fast Track Designations include (i) Pretreated, Platinum-Resistant Advanced or Metastatic BRCA1/2 mutant Ovarian Cancer, and (ii) Pretreated, Advanced or Metastatic HR+, Her2-, BRCA1/2 mutant Breast Cancer
PARG = poly (ADP-ribose) glycohyrdolase; PAR = poly (ADP-ribose; PBMC = peripheral blood mononuclear cells, PSA = prostate specific antigen, EC = endometrial cancer, CRC = colorectal cancer
[1] Clinicaltrials.gov: NCT05787587

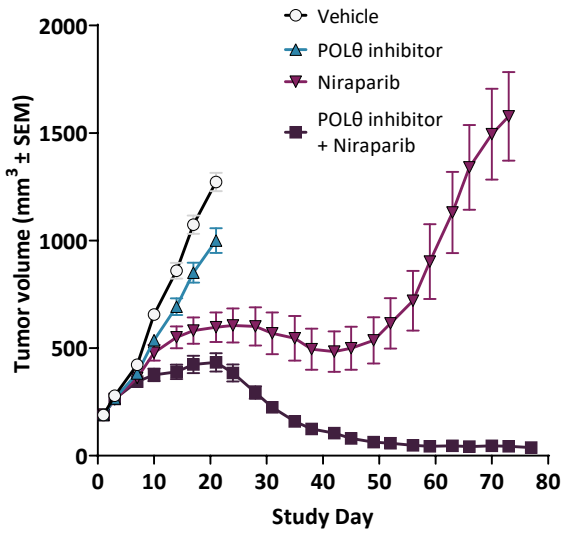
IDEAYA BIOSCIENCES

GSK101 (IDE705): Potential First-in-Class Pol Theta Helicase Inhibitor

Phase 1 in Combination with Niraparib (PARPi)

Pol Theta Helicase *In Vivo* Activity

GSK101 + PARPi

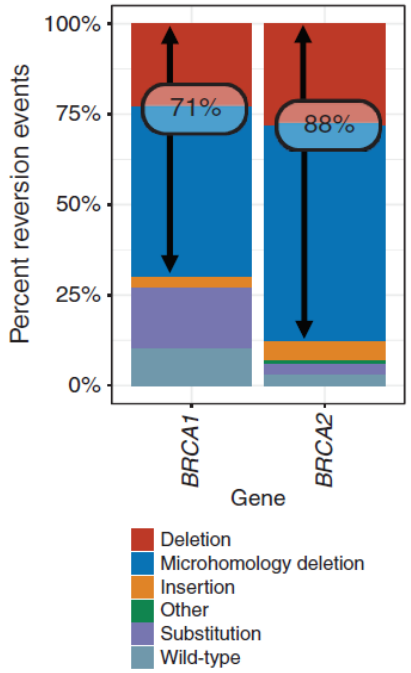


Observed Deep and Durable Responses in Multiple Xenograft Models

IDEAYA / GSK Data

BRCA 1/2 Clinical Reversions

BRCA Reversions Mediated by MMEJ



Cancer Res. 2020, DOI: 10.1158/2159-8290

Clinical Development Strategy

Pol Theta Helicase Inhibitor + PARP Inhibitor

Pol Theta Helicase Inhibitors Disrupt MMEJ Alternative DNA Damage Repair:

- Inhibit DSB Repair by MMEJ
- Dysregulate Replication Fork Stabilization

Potentiate PARPi Efficacy	Prevent PARPi Resistance	Overcome PARPi Resistance

Potential Clinical Opportunities

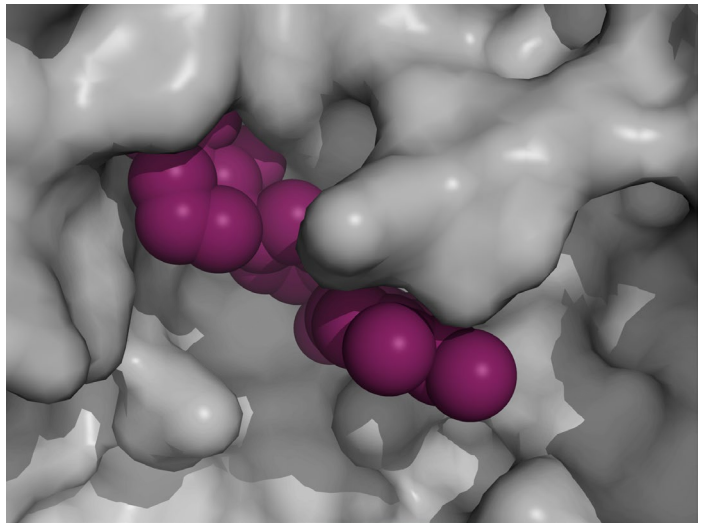
GSK Strategic Partnership: Global Royalties with GSK covering all Costs, ~$1B Milestones, incl up to $20M Preclinical / Ph1 Clinical Potential Combination with GSK's Zejula™, a PARP Inhibitor

IDEAYA's AI/ML Enabled Drug Discovery Platform and IND-Engine

IND-Filing and Multiple Potential First-in-Class Development Candidates (DCs) Targeted in 2024

WRN Helicase

Nominated Werner Helicase Development Candidate

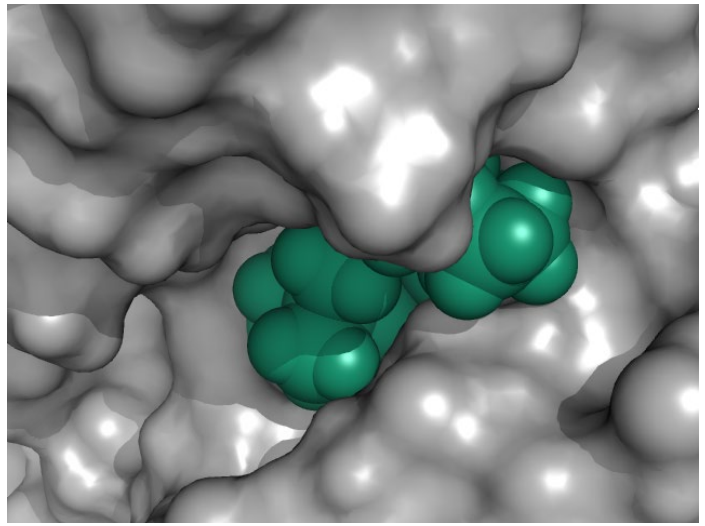


Targeting IND Submission in 2024*
MSI-High Tumor Agnostic

*Pursuant to GSK Collaboration

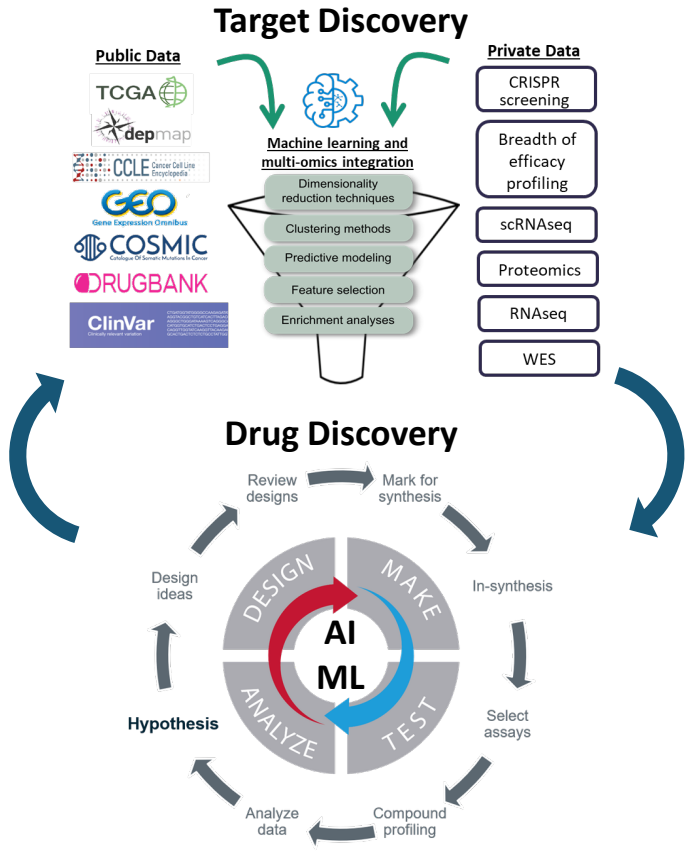
Multi-Pronged Strategy in MTAP$^{-/-}$

Next Generation Programs



Key mechanistic interaction with MTAP-loss, including distinct from PRMT5 pathway

AI/ML-Enabled IND-Engine



Targeting multiple DCs in 2024

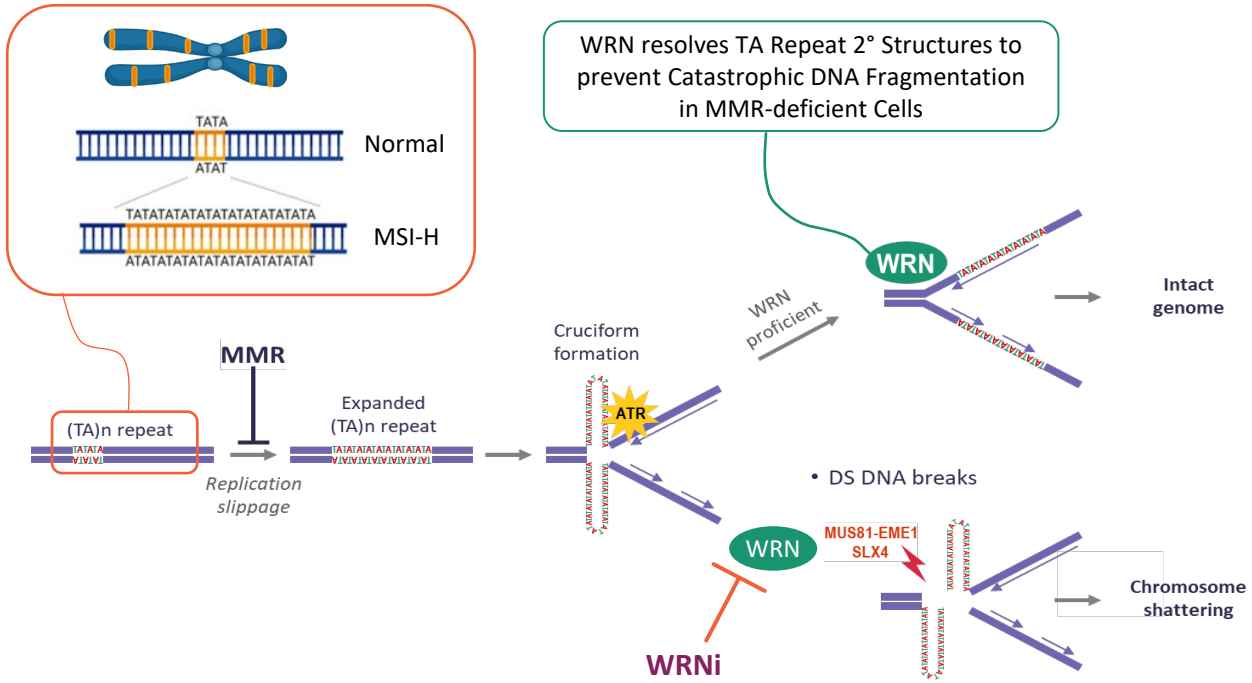
Werner Helicase is Synthetic Lethal with Microsatellite Instability

Development Candidate in IND Enabling Studies

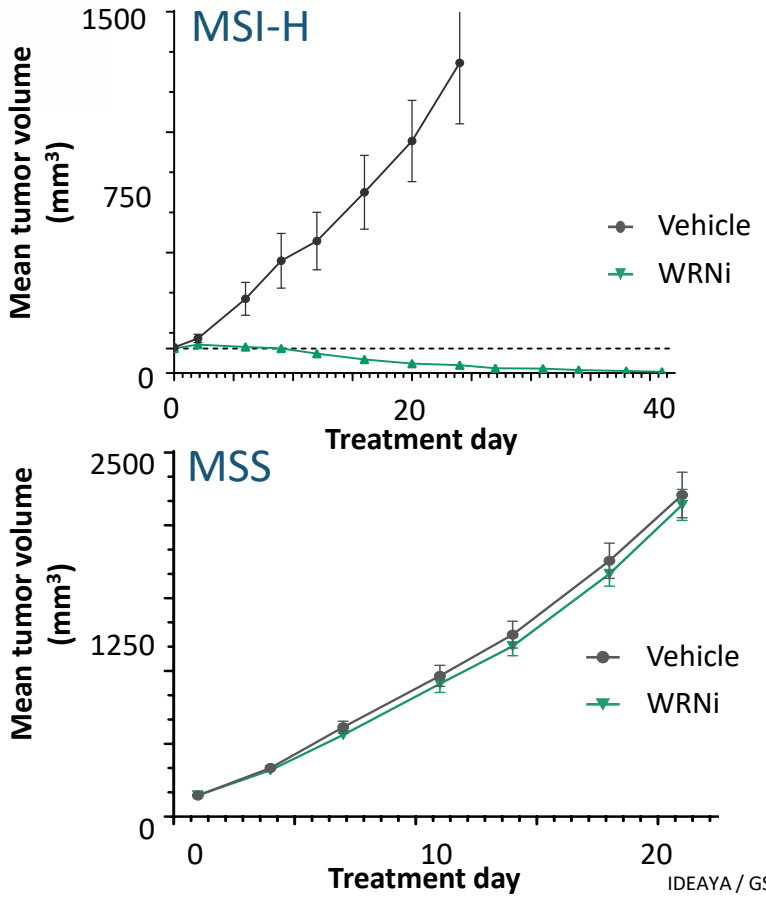
Werner Helicase Inhibitors Phenocopy Genetic SL in MSI-High Cancers

Nanomolar Potency, Biochemical and Cellular Activity, and Selectivity over other RecQ Helicases

Werner Helicase Synthetic Lethality in MSI-High Cancer Cells



Werner Helicase Synthetic Lethal with High-MSI



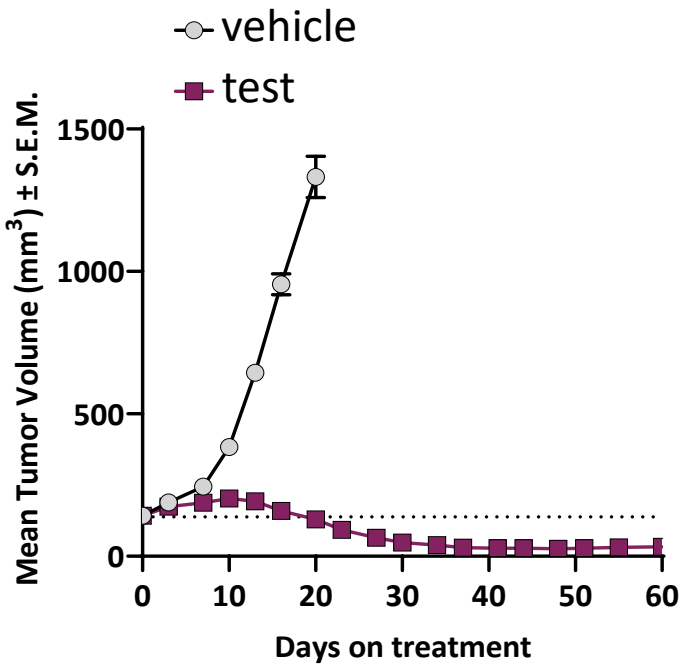
IDEAYA / GSK Data: AACR 2023

GSK Strategic Partnership: 50/50% US Profit Share and ex-US Royalties, ~$1B Milestones, incl up to $20M Preclinical / Ph1 Clinical; Cost Share 80% (GSK) / 20% (IDEAYA); Potential Combination with GSK's Jemperli™, a PD-1 IO Agent

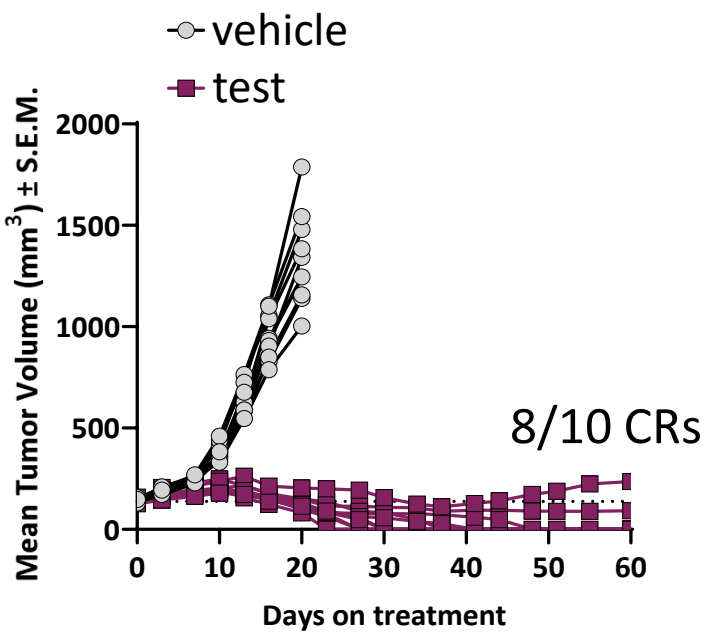
IDEAYA Pipeline: MTAP-Deletion New Target Opportunity

Mechanism-based activity distinct from PRMT5 pathway

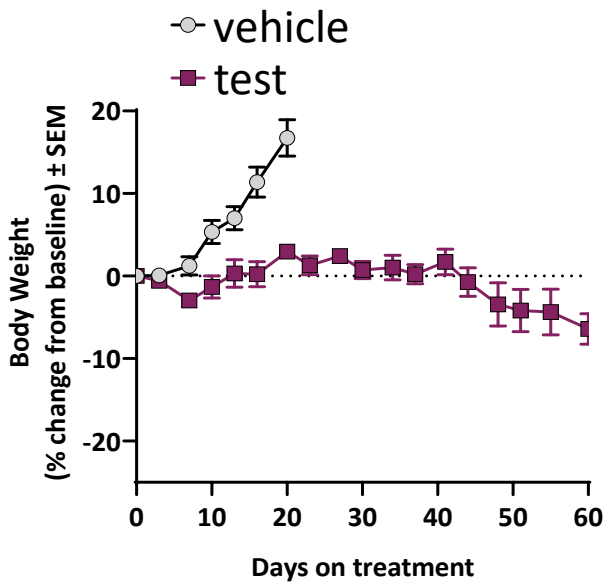
Mean Tumor Volume in NCI-H838



8 of 10 Complete Responses



Minimal Body Weight Change



- First-in-class opportunity not yet evaluated in the clinic
- Cellular screens indicate potential for broad therapeutic benefit in MTAP$^{-/-}$ cancers
- Mechanism anticipated to combine well with MAT2A and PRMT5^{MTA} inhibitors

Building a Fully-Integrated Biotech in Precision Medicine Oncology

Foundational Potential First-in-Class Clinical Pipeline and Drug Discovery Platform

CLINICAL PROGRAMS	DEVELOPMENT CANDIDATES	PRECLINICAL
Ph 2/3 – Darovasertib [1] Ph 2 – IDE397 (MAT2A) [1] Ph 1 – IDE161 (PARG) [1] Ph 1 – GSK101 (Pol Theta Helicase) [2]	Werner DC – Targeting 2024 IND [2]	Targeting Multiple DC Nominations in 2024, including in MTAP-deletion
4 Clinical Programs	5 Clinical Programs	≥7 Clinical Programs

Darovasertib Registration-Enabling Trial with Potential Accelerated Approval in HLA-A2(-) MUM is tractable for commercial execution and provides path to potential product revenue to reinvest in broad *first-in-class* pipeline

Potential First-in-Class Precision Medicine Oncology Pipeline, including Darovasertib (Ph2/3), IDE397 (Ph 2), IDE161 (Ph 1), GSK101 (Ph 1), Werner Helicase (IND-enabling), and multiple Development Candidates targeted in 2024, including in MTAP-deletion

Strong Balance Sheet with ~$975M[3] and opportunity for milestones with cash runway to 2028

Pharma Collaborations include combinations with Pfizer, Amgen, Gilead, Merck, and GSK partnership with ~$2 billion[2] in potential milestones

(1) Clinical Trial Collaboration and Supply Agreements, independently with Pfizer (Darovasertib + Crizotinib), Amgen (IDE397 + AMG193), Gilead (IDE397 + Trodelvy®), and Merck (IDE161 + KEYTRUDA®); IDEAYA retains all commercial rights to its products
(2) GSK101 Pol Theta Program Cost Share = 100% GSK with ~$1B Milestones and WW Royalties; Werner Helicase Program Cost Share = 80% GSK / 20% IDEAYA with ~$1B Milestones, 50/50 US Profit Share and Ex-US Royalties
(3) Includes aggregate of $632.6M cash, cash equivalents and marketable securities as of December 31, 2023, plus pro forma $342.3M estimated net proceeds from sales of common stock through at-the-market offerings in January 2024