DEVELOPING PRECISION MEDICINES FOR THE TREATMENT OF CANCER

Corporate Presentation – November 2024

Forward-Looking Statements

This presentation contains forward-looking statements. Such statements include, but are not limited to, statements regarding our research, preclinical and clinical development activities, plans and projected timelines for ziftomenib, KO-2806 and tipifarnib, plans regarding regulatory filings, our expectations regarding the relative benefits of our product candidates versus competitive therapies, and our expectations regarding the therapeutic and commercial potential of our product candidates. The words "believe," "may," "should," "will," "estimate," "promise," "plan", "continue," "anticipate," "intend," "expect," "potential" and similar expressions (including the negative thereof) are intended to identify forward-looking statements. Because such statements are subject to risks and uncertainties, actual results may differ materially from those expressed or implied by such forward-looking statements. Risks that contribute to the uncertain nature of the forward-looking statements include: our preclinical studies and clinical trials may not be successful; the U.S. Food and Drug Administration (FDA) may not agree with our interpretation of the data from clinical trials of our product candidates; we may decide, or the FDA may require us, to conduct additional clinical trials or to modify our ongoing clinical trials; we may experience delays in the commencement, enrollment, completion or analysis of clinical testing for our product candidates, or in the reporting of data from such clinical testing, or significant issues regarding the adequacy of our clinical trial designs or the execution of our clinical trials may arise, which could result in increased costs and delays, or limit our ability to obtain regulatory approval; our product candidates may not receive regulatory approval or be successfully commercialized; unexpected adverse side effects or inadequate therapeutic efficacy of our product candidates could delay or prevent regulatory approval or commercialization; and we may not be able to obtain additional financing. Additional risks and uncertainties may emerge from time to time, and it is not possible for Kura's management to predict all risk factors and uncertainties.

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Investment Highlights

Targeted Oncology	Advancing a pipeline of novel investigational therapies, forging new scientific and clinical paths to give patients a better chance for long-term, durable remissions
Proprietary Pipeline	**Menin Inhibitor Programs – Ziftomenib & Next Generation** • Potential to address up to 50% of acute leukemias through monotherapy and combinations • Breakthrough Therapy Designation granted by FDA for treatment of R/R NPM1-mutant AML • Upcoming data from 100 patients in combo with ven/aza and 7+3 in NPM1-m and KMT2A-r AML • Topline data from registration-directed trial in NPM1-mutant AML expected in early 2025 • IND cleared for ziftomenib in GIST; preclinical data support opportunity for menin inhibitor in diabetes
	Farnesyl Transferase Inhibitor Programs (KO-2806 & Tipifarnib) • Dose-escalation study of next-gen FTI KO-2806 continues as monotherapy and in combo with cabozantinib in ccRCC and adagrasib in NSCLC • Clinical collaboration with BMS to evaluate KO-2806 and adagrasib in $KRAS^{G12C}$-mutated NSCLC • Durable responses observed with tipifarnib as monotherapy in HRAS-mutant HNSCC • Compelling safety profile and activity with tipifarnib plus alpelisib in PIK3CA-dependent HNSCC
Strong Financials	• $25 million strategic equity investment from Bristol Myers Squibb • $455 million in cash as of September 30, 2024* provides runway into 2027

* Cash, cash equivalents and short-term investments

Experienced Leadership Team and Board of Directors

Leadership Team

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Drug Candidate Pipeline

PROGRAM	CLINICAL TRIAL	STUDY STARTUP / DOSE-ESCALATION / DOSE-VALIDATION / REGISTRATION DIRECTED	ANTICIPATED MILESTONE
ZIFTOMENIB Menin Inhibitor	**KOMET-001** Monotherapy	NPM1-mutant acute myeloid leukemia (AML)	Topline data in early 2025
		KMT2A-rearranged acute lymphoblastic leukemia (ALL)	Now dosing patients
		Non-NPM1-mutant / Non-KMT2A-rearranged AML	Now dosing patients
	KOMET-007 Combinations with venetoclax/azacitidine, cytarabine + daunorubicin (7+3)	NPM1-mutant AML	Phase 1b expansion study now enrolling
		KMT2A-rearranged AML	
	KOMET-008 Combinations with gilteritinib, FLAG-IDA, LDAC	NPM1-mutant AML	Now dosing patients
		KMT2A-rearranged AML	
	KOMET-015 Combination with imatinib	Advanced GIST	Initiate proof-of-concept study in 1H 2025
KO-2806 Next-Generation Farnesyl Transferase Inhibitor (FTI)	**FIT-001** Monotherapy, combinations with cabozantinib and adagrasib	Solid tumors	Now in dose escalation as monotherapy
		Clear cell renal cell carcinoma (ccRCC)	Now dosing patients in combo with cabozantinib
		$KRAS^{G12C}$-mutant non-small cell lung cancer (NSCLC)	Now dosing patients in combo with adagrasib
TIPIFARNIB FTI	**KURRENT-HN** Combination with alpelisib	PIK3CA-dependent head and neck squamous cell carcinoma (HNSCC)	Present preliminary data in 1H 2025

KURA ONCOLOGY

ZIFTOMENIB: MENIN-KMT2A/MLL INHIBITOR

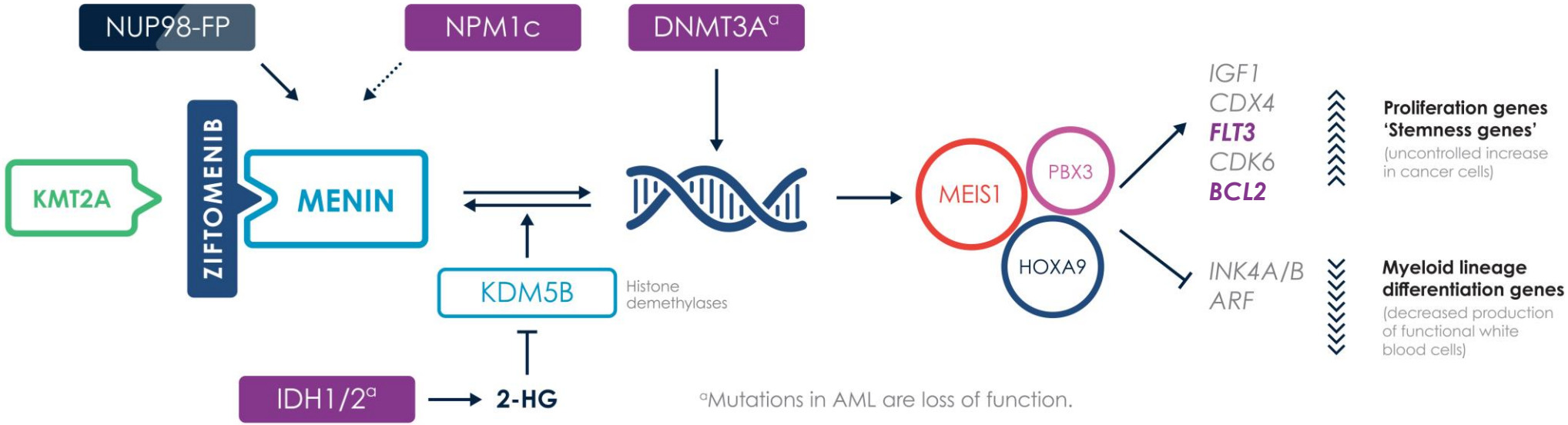
Ziftomenib Demonstrates Potential to Become a Cornerstone of AML Therapy

Targets foundational mutations in up to 50% of AML cases

- **Compelling clinical data support frontline opportunity**
  - Good tolerability profile, enabling continuous administration in combination with SOC
  - Combinations appear to mitigate the risk of differentiation syndrome
  - No observed or predicted drug-drug interactions
  - Encouraging preliminary evidence of clinical activity
- **Strong investigator enthusiasm as evidenced by rapid enrollment across studies**
  - First 20 patients enrolled in KOMET-007 combination trial in less than four months
  - Now dosing patients in KOMET-008 combination trial with SOCs, including FLT3 inhibitor
  - Enrollment in KOMET-001 monotherapy registrational trial completed in < 16 months

Ziftomenib Targets the Menin-KMT2A Pathway, A Foundational Target in AML

- *NPM1*-m and *KMT2A*-r drive overexpression of *HOXA9/MEIS1* genes, critical for transformation to AML
- KMT2A(MLL) sits upstream from major AML targets (*i.e.*, *FLT3*, *BCL2*, *IDH1/2*, *DNMT3A*)
- KMT2A(MLL)-dependent genes contribute to therapeutic resistance and relapse to current therapies
- Menin inhibition downregulates HOXA9/MEIS1, leading to differentiation of leukemic blasts



1. Lu et al. Cancer Cell 2016;30(1):92–107; 2. Ferreira et al. Oncogene 2016;35(23):3079-82; 3. Jeong et al. Nat. Genet 2014;46(1):17-23; 4. Wang et al. Blood 2005;106(1):254–64; 5. Chowdhury et al. EMBO Rep 2011;12(5):463-9; 6. Schmidt et al. Leukemia 2019;33(7):1608-19; 7. Xu et al. Cancer Cell 2016;30(6):863-78; 8. Collins & Hess. Curr Opin Hematol 2016;23(4):354-61; 9. Brunetti et al. Cancer Cell 2018; 34(3):499–512.

KOMET-001 Phase 1/2 Study of Ziftomenib in Relapsed / Refractory AML

komet-001
ACUTE LEUKEMIAS
KURA KO-MEN-001

Phase 1a Dose Escalation	Phase 1b Validation Cohorts	Phase 1b Expansion	Phase 2 Registration-Enabling
Completed	*Completed*	*Completed*	*Enrollment Completed*
50 mg QD — 100 mg QD — 1000 mg QD	Cohort 1: 200 mg QD Cohort 2: 600 mg QD	Expansion of 600 mg QD	600 mg QD
NPM1-m, *KMT2A*-r, Other	*NPM1*-m or *KMT2A*-r	*NPM1*-m	*NPM1*-m
OBJECTIVES			

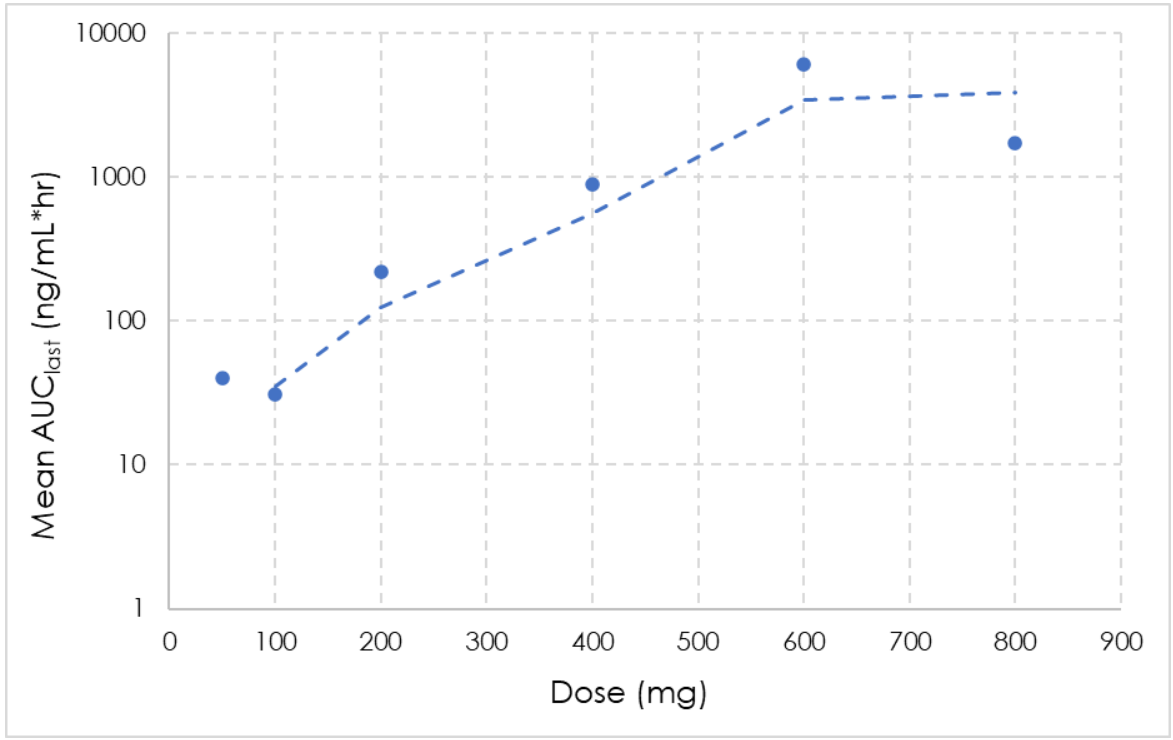
• Safety and tolerability
• Pharmacokinetics
• Early evidence of antitumor activity
	• Safety and tolerability
	• Pharmacokinetics
	• Clinical activity
		Continue enrollment of Phase 1b validation cohort(s) consistent with FDA's Project Optimus
		• Safety and tolerability
		• Pharmacokinetics
		• Clinical activity
			• Primary endpoint:
			  • CR/CRh
			• Secondary endpoints:
			  • Duration of CR/CRh
			  • Transfusion independence
			  • CR/CRh MRD negativity
			  • Adverse events

CR, complete remission; CRh, complete remission with partial hematological recovery; FDA, United States Food and Drug Administration; MRD, measurable residual disease; R/R, relapsed/refractory; RP2D, recommended phase 2 dose.

Ziftomenib Demonstrates Optimal Pharmaceutical Properties

Clinical data from KOMET-001 demonstrate:

- Ziftomenib demonstrates a dose-dependent increase in exposure up to RP2D at 600 mg
- Ziftomenib is not a clinically meaningful CYP3A4 substrate
  - No dose adjustment of ziftomenib needed when administered with a CYP3A4 inhibitor (*e.g.*, azoles)
- Ziftomenib is not a clinically meaningful CYP3A4 inhibitor[1]
  - No dose adjustment needed for CYP3A4 substrates (*e.g.*, venetoclax)
- No drug-induced QTc prolongation observed at any dose



[1] FDA Guidance (2020): Clinical Drug Interaction Studies – Cytochrome P450 Enzyme- and Transporter-Mediated Drug Interactions Guidance for Industry

Ziftomenib Demonstrates Encouraging Safety Profile in Phase 1

- Differentiation syndrome (DS) appears manageable in NPM1-m monotherapy patients with mitigation strategy
    - 20% rate of mild to moderate DS
- Rates of DS in KMT2A-r monotherapy patients were 42.9% at 200 mg and 38.9% at 600 mg; potential to mitigate in combination
- DS is an on-target adverse event and represents evidence of clinical activity
- No reports of drug-induced QTc prolongation
- Maintained count recovery suggests no drug-induced myelosuppression

Wang *et al*. Lancet Oncol. 2024 Oct;25(10):1310-1324.

Ziftomenib Has Highly Differentiated Monotherapy Activity

40% of NPM1 patients achieved a CR during course of study

Best Overall Response	600 mg
NPM1-m Phase 1a + 1b	**(n=20)**
CR	**7 (35.0)**
CR/CRh	7 (35.0)
CRc	8 (40.0)
MRD negativity	4 (50.0)[1]
ORR	9 (45.0)
KMT2A-r Phase 1a + 1b	**(n=18)**
CR/CRh	2 (11.1)
CRc	3 (16.7)
MRD negativity	3 (100.0)
ORR	3 (16.7)

Differentiated CR Rates vs. SOC in Heavily Pretreated Patients

	MUTATION	CR %	mDOR	MEDIAN PRIORS
Ziftomenib 600mg QD	NPM1m	**35%**	**7.7** mo*	**3**
	FLT3m	**33%**	-	
	IDH 1/2	**50%**	-	
Gilteritinib	FLT3m	14.2%	14.8 mo	1
Enasidenib	IDH2	19%	8.2 mo	2
Ivosidenib	IDH1	25%	10.1 mo	2

*Median DoR for CRc without censoring at HSCT
Source: USPI's

- **High activity, durable responses and favorable profile suggest potential for ziftomenib to become a backbone therapy across the continuum of AML care**

[1]MRD was assessed for 6/8 CRc patients; 4 of those 6 patients (67%) tested were MRD negative
CRc includes CR, CRh, CRi, CRp; ORR includes CR, CRh, CRi, CRp, MLFS
Wang *et al*. Lancet Oncol. 2024 Oct;25(10):1310-1324.

Case Studies Highlight Meaningful Durability and Favorable Tolerability

Durable CR for 36 cycles on ziftomenib in 8th line including 2 HSCTs

44 yo female with *NPM1*-m, *DNMT3A* and *IKZF1* AML
7 Prior Tx
Baseline bone marrow blasts: 14%

ziftomenib at 200 mg

Response
- CRmrd- after Cycle 1
- CRmrd- through Cycle 36

Enthusiasm among investigators and patients to utilize ziftomenib earlier and initiate maintenance

22 yo male with *NPM1*-m AML
1 Prior Tx (refractory to 7+3)
Baseline bone marrow blasts: 90%

ziftomenib at 600 mg

Response
- CRmrd- after Cycle 1
- HSCT
- CRmrd- maintained on Cycle 2 post-HSCT

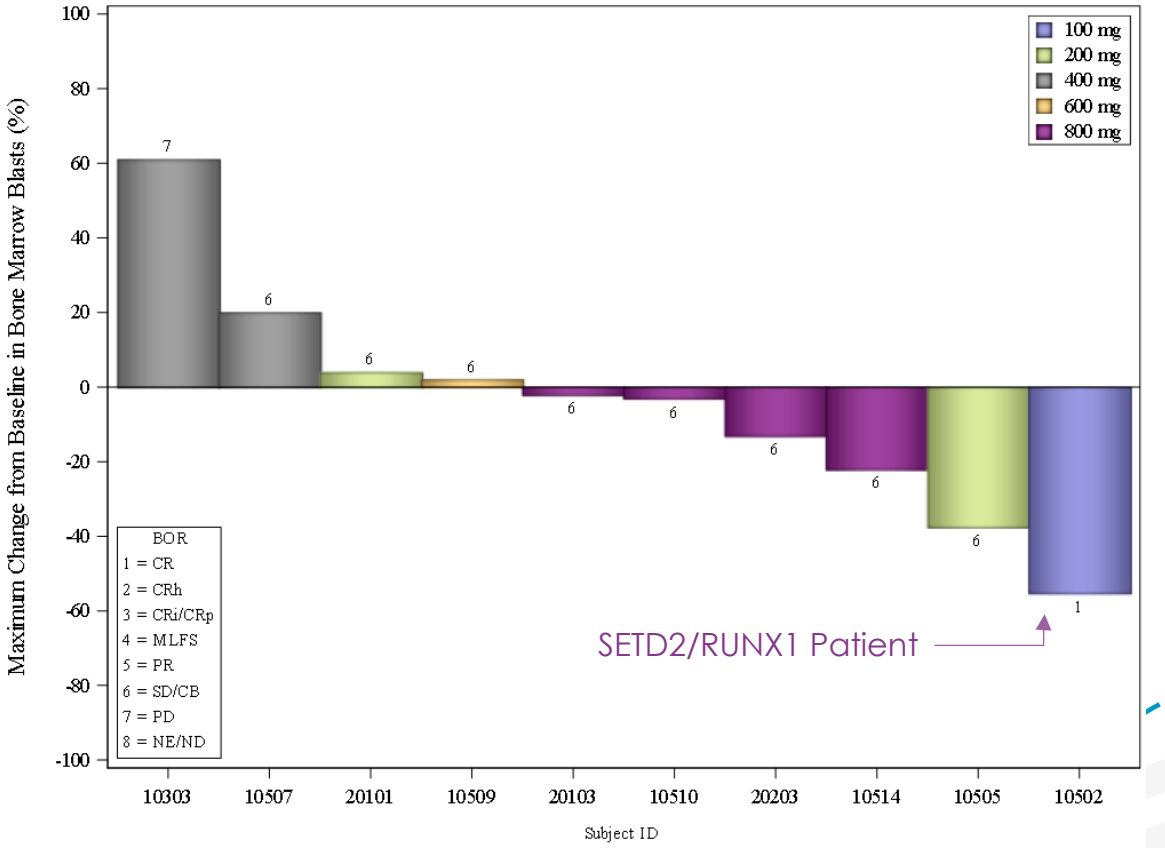
Fathi *et al*. EHA 2023 #LB2713 (preliminary data as of April 12, 2023)

Targeting the Menin-KMT2A Pathway has Potential to Benefit a Broader Subset of AML Patients

Ziftomenib has Demonstrated Evidence of Activity in Non-*NPM1*-m/Non-*KMT2A*-r Patients

- *SETD2/RUNX1* patient achieved a CR at 100mg dose in Phase 1a
- Notable evidence of blast reduction in range of off-target patients
- KOMET-001 study will continue to evaluate additional AML populations
- Potential to be incorporated into KOMET-007/008 combination studies

Off-Target Mutations: Best Change from Baseline in Bone Marrow Blasts – mITT Phase 1a



Graphic excludes subjects where post-baseline BM samples not collected

Summary: KOMET-001 Phase 1 Clinical Trial of Ziftomenib

Ziftomenib demonstrates an encouraging safety profile and tolerability

- Reported events most often consistent with features and manifestations of underlying disease
  - No evidence of drug-induced QTc prolongation
  - Differentiation syndrome, an on-target effect, manageable with mitigation strategy

Clinical activity of ziftomenib monotherapy is optimal at the 600 mg daily dose

- Positive *NPM1*-m benefit/risk balance with pronounced activity and 35% CR rate (n=20)
- High levels of ziftomenib tissue penetration likely drive clearance of extramedullary disease
- Emergence of resistance mutations has been observed at a much lower rate relative to certain competition

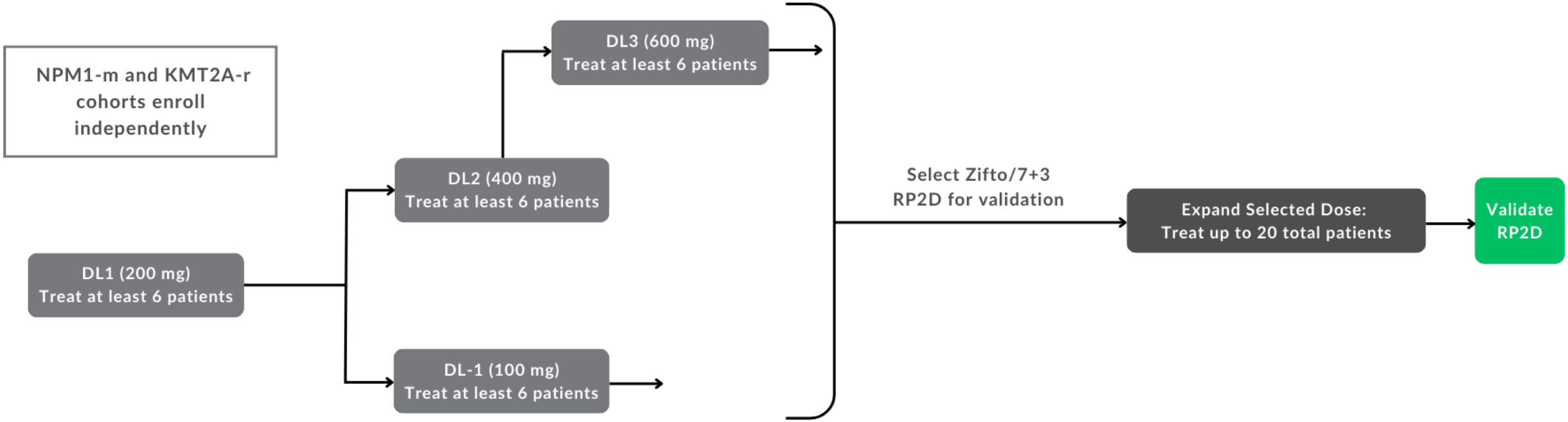
Monotherapy data supportive of combination strategies

- No predicted adverse drug-drug interactions
- Optimization of *KMT2A*-r benefit/risk planned via combination strategies to maximize time on treatment
- Oral, QD dosing allows for convenient administration and combination with standards of care

Wang et al. Lancet Oncol. 2024 Oct;25(10):1310-1324

KOMET-007: Phase 1 Combination Trial of Ziftomenib in Patients with Newly Diagnosed or R/R AML

Ziftomenib/cytarabine/daunorubicin (7+3) combination

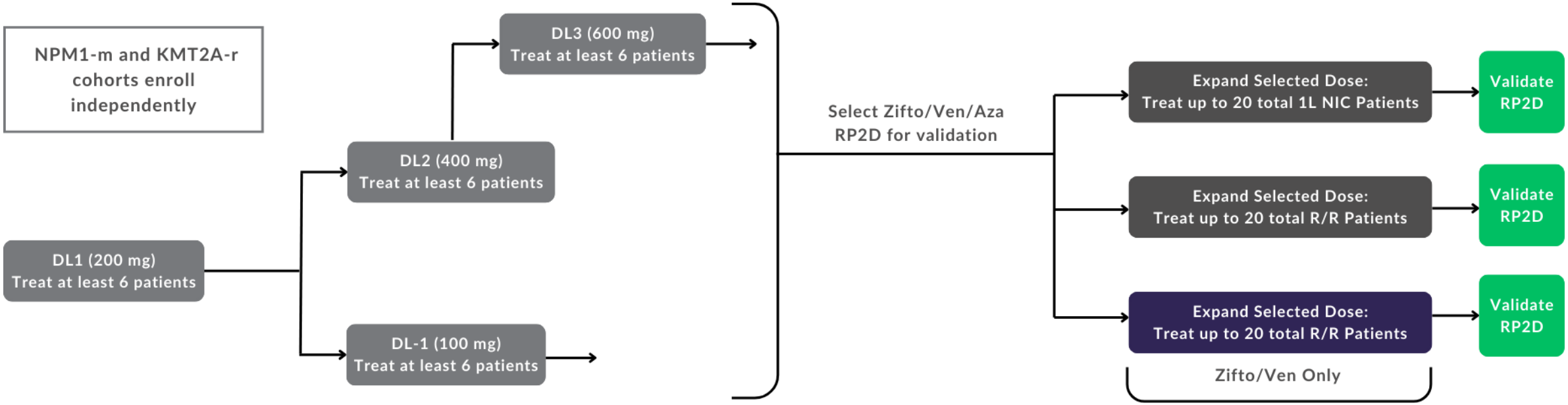


- Ziftomenib dosing begins on Cycle 1 Day 8 and is continuous thereafter
- Cytarabine administered on Cycle 1 Day 1-7; administration of an additional cycle based on C1 bone marrow biopsy results
- Daunorubicin administered on Cycle 1 Day 1-3; administration of an additional cycle based on C1 bone marrow biopsy results
- Dose escalation conducted in patients with adverse risk*

*Age ≥ 60 years and/or treatment-related AML and/or adverse risk cytogenetics per ELN
DL = ziftomenib dose level; zifto = ziftomenib; 7+3 = cytarabine/daunorubicin; RP2D = recommended Phase 2 dose; 1L = first-line; IC = intensive chemotherapy

KOMET-007: Phase 1 Combination Trial of Ziftomenib in Patients with Newly Diagnosed or R/R AML

Ziftomenib/venetoclax/azacitidine combination



- Ziftomenib dosing begins on Cycle 1 Day 8 and is continuous thereafter
- Venetoclax administered per label in 28-day cycles with adjustments to cycle length based on Cycle 1 bone marrow biopsy results
- Azacitidine administered per label on Cycle 1 Days 1-7 with additional cycles based on bone marrow biopsy results

DL = ziftomenib dose level; zifto = ziftomenib; ven = venetoclax; ven = venetoclax; RP2D = recommended Phase 2 dose; NIC = non-intensive chemotherapy; 1L = first-line; R/R = relapsed/refractory

KOMET-007: Promising Safety and Tolerability Profile in Combination

Combinations mitigate risk of differentiation syndrome (DS)

Grade ≥ 3 TEAEs (≥ 10%)	n (%)
Patients with Grade ≥ 3 TEAEs	**18 (90)**
Platelet count decreased	6 (30)
Febrile neutropenia	5 (25)
White blood cell count decreased	4 (20)
Pneumonia	3 (15)
Hypoxia	2 (10)
Neutrophil count decreased	2 (10)
Sepsis	2 (10)
Thrombocytopenia	2 (10)

Grade ≥ 3 Ziftomenib-Related AEs (All)	n (%)
Patients with Grade ≥ 3 Ziftomenib-Related AEs	**6 (30)**
Platelet count decreased	3 (15)
Anemia	1 (5)
Febrile neutropenia	1 (5)
Leukopenia	1 (5)
Neutrophil count	1 (5)
Thrombocytopenia	1 (5)

- No DS events reported
- No dose-limiting toxicities (DLTs) observed, including delayed hematologic count recovery
- No QTc prolongation observed
- TEAEs consistent with underlying disease and backbone therapies

Preliminary data as of January 11, 2024
TEAEs = Treatment-emergent adverse events

100% CR rate with Ziftomenib and 7+3 in 1L Patients with Adverse-Risk AML*

- Anticipated CR/CRi rate with 7+3 in all-comer 1L adverse risk patients: 32-33%[1,2]

1L Adverse-Risk Group n=5	CR Rate (n)
Overall (NPM1-m + KMT2A-r)	100% (5)
NPM1-m only (n=4)	100% (4)
KMT2A-r only (n=1)	100% (1)

- All patients treated in initial dose cohort (200 mg) in combination with 7+3

Preliminary data as of January 11, 2024

[1] Lancet et al. *Blood*. 2014 May 22;123(21):3239-46.

[2] Lin et al. *Blood Adv*. 2021 Mar 23;5(6):1719-1728.

*Age ≥ 60 years and/or treatment-related AML and/or adverse risk cytogenetics per ELN

Ziftomenib + Ven/Aza with Pronounced Activity in Menin Inhibitor Naïve Patients

- ~35-45% CR/CRi rate is expected in ven-naïve relapsed/refractory patients[1]
- Anticipated CR/CRi rate in KMT2A-r AML following two prior therapies <10%[2]
- 53% ORR in mITT population (n=15, including six menin experienced patients)
- 40% (6/15) of patients treated with ven/aza received prior treatment with a menin inhibitor

Menin Inhibitor Naïve Group n=9	ORR (n)	CR/CRi Rate (n)	CR/CRh Rate (n)
Overall (NPM1-m + KMT2A-r)	78% (7)	67% (6)	56% (5)
NPM1-m (n=5)	100% (5)	80% (4)	60% (3)
KMT2A-r (n=4)	50% (2)	50% (2)	50% (2)

- All patients treated in initial dose cohort (200 mg) in combination with Ven/Aza
- Enrollment in 600 mg dose cohort ongoing

Preliminary data as of January 11, 2024
[1] Stahl, M. *et al.*, *Blood Advances* 5(5), 1552-1564 (2021)
[2] Issa, G. et al. Blood Cancer J. 11, 162 (2021)
ORR includes CR, CRh, CRi, MLFS

Ziftomenib + Ven/Aza Able to Drive Responses in Venetoclax Failures

- Expected response rates following ven/aza ~ 0-20%[1-4]
- Anticipated CR/CRi rate in KMT2A-r AML following two prior therapies < 10%[5]

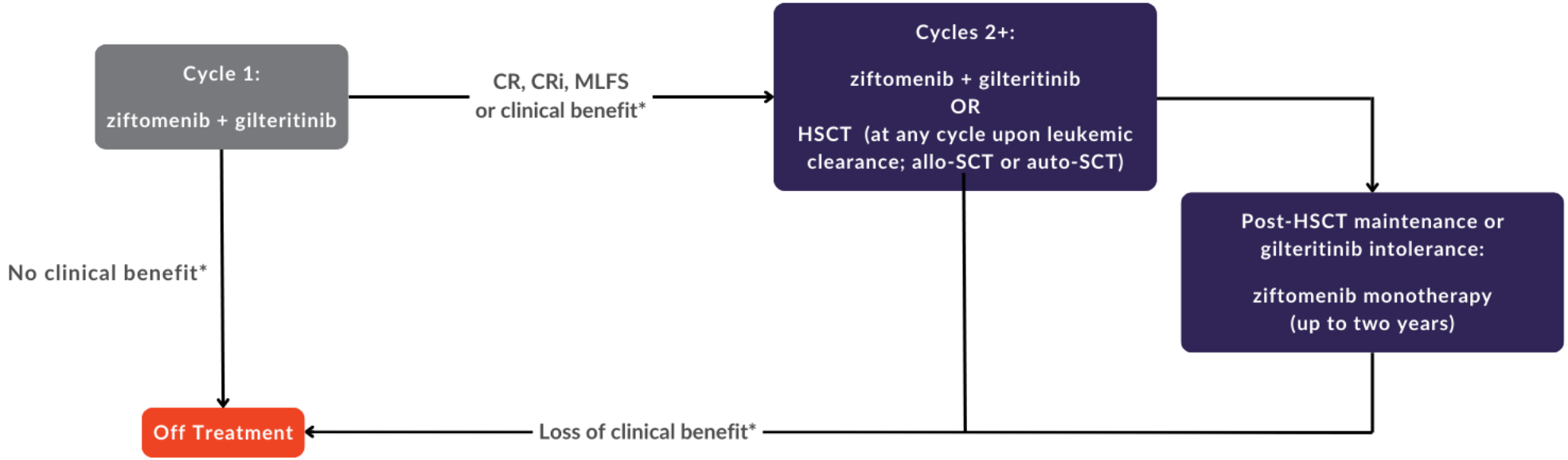
Venetoclax Experienced Group n=10	ORR (n)	CR/CRi Rate (n)	CR/CRh Rate (n)
Overall (NPM1-m + KMT2A-r)	40% (4)	30% (3)	30% (3)
NPM1-m (n=5)	60% (3)	40% (2)	40% (2)
KMT2A-r (n=5)	20% (1)	20% (1)	20% (1)

- All patients treated in initial dose cohort (200 mg) in combination with Ven/Aza
- Enrollment in 600 mg dose cohort ongoing

Preliminary data as of January 11, 2024
[1] Zainaldin, C. et al., Lymphoma 63(13):3245-3248 (2022);
[2] Chan, O. and Walker, A., *Hematology* 702-708 (2023);
[3] Maiti A, et al., Haematologica. 2021; 106(3):894-898;
[4] Issa, Syndax ASH Investor Event (Dec. 2023)
[5] Issa, G. et al. Blood Cancer J. 11, 162 (2021)
ORR includes CR, CRh, CRi, MLFS

KOMET-008: Phase 1 Combination Trial of Ziftomenib in Patients with R/R AML

Ziftomenib + gilteritinib combination



- Phase 1a Dose Escalation
  - Arm A: Ziftomenib in combination with FLAG-IDA or LDAC or gilteritinib (illustrated above) in relapsed or refractory (R/R) NPM1-mutant AML
  - Arm B: Ziftomenib in combination with FLAG-IDA or LDAC in R/R KMT2A-rearranged AML
- Patients must also have documented FLT3 mutation if receiving gilteritinib

*Per investigator discretion

CR, complete remission; CRi, CR with incomplete count recovery; MLFS, morphologically leukemia-free state; HSCT, hematopoietic stem-cell transplantation; allo-SCT, allogenic stem-cell transplantation; auto-SCT, autologous stem-cell transplantation

Targeting Foundational Mutations has Transformed Deadly Hematologic Cancers into Chronic Diseases

Multiple Myeloma

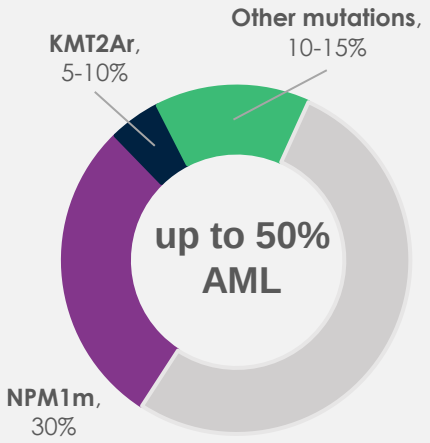
- Until the 2000's, there were few treatment options for multiple myeloma, and the median survival was 2–3 years
- With the advent of immunomodulatory drugs (IMiDs) and proteasome inhibitors (PIs) in the 2000's, the outcomes of patients are now significantly improving
- Many patients can now live with their disease > 10 years
- **IMiDs have become a cornerstone of treatment for patients with multiple myeloma and are used in combinations at all stages of disease**

IMiD combinations increased 5-year overall survival from 35% to > 65%; class generated ~$15B in revenues at peak

Holstein and McCarthy, *Drugs* (2017) 77(5), 505-520
Bird, S. and Pawlyn, C. *Blood* (2023) 142(2): 131-140
SEER Statistics (accessed on Jan 4, 2024)
Gulla A. and Anderson K. *Haematologica* (2020) 105(10): 2358-2367
Bristol Myers Squibb 2022 Financial Report

Investigating Ziftomenib Across the AML Continuum in up to 50% of Patients for Whom Menin-KMT2A Pathway is a Disease Driver

Prevalence of Ziftomenib Eligible Patients



1L Treatable ~17K → **Relapsed / Refractory** ~14K

1L Treatable			Relapsed / Refractory			
FIT IC* or UNFIT NIC*	Transplant/ No Transplant	Post-Transplant Maintenance	IC or NIC Tx or tolerable therapy	Transplant/ No Transplant	Targeted Tx if FLT3m, IDH1/2m and/or NPM1m	Non-Intensive therapy / Palliative Care

komet ACUTE LEUKEMIAS
Ziftomenib Clinical Trials for Acute Leukemias

KOMET-007
- 1L Zifto + Ven / Aza
- 1L Zifto + 7+3

Investigator/ Company Sponsored Studies
- Post-HSCT Maintenance

KOMET-001
- R/R NPM1m AML

KOMET-007
- R/R Zifto + Ven/Aza

KOMET-008
- R/R Zifto + FLAG-IDA
- R/R Zifto + LDAC
- R/R Zifto + gilteritinib

*FIT IC = patients eligible for induction chemotherapy; UNFIT NIC = patients eligible for non-intensive chemotherapy

Ziftomenib: A Multi-Billion-Dollar Opportunity in AML and Beyond

Potential to Transform Outcomes Across the Continuum of Care

Relapsed / Refractory

- Initial approval would represent **30% of potential AML patients**
- KOMET-001 registration-directed study for FDA full approval
- **BTD granted in R/R NPM1-mutant AML** indicating potential for substantial improvement over available therapies

Frontline / Maintenance

- Significant opportunity in 1L AML and Maintenance
- Safety, tolerability and clinical activity anticipated to be ideal for combinations with SOC and with maintenance indication

Other Indications

- Compelling **additional opportunities beyond AML** offer multi-billion-dollar potential
- Translational data support potential in **GIST and diabetes**
- **Next-generation menin inhibitors** currently under development

KURA ONCOLOGY

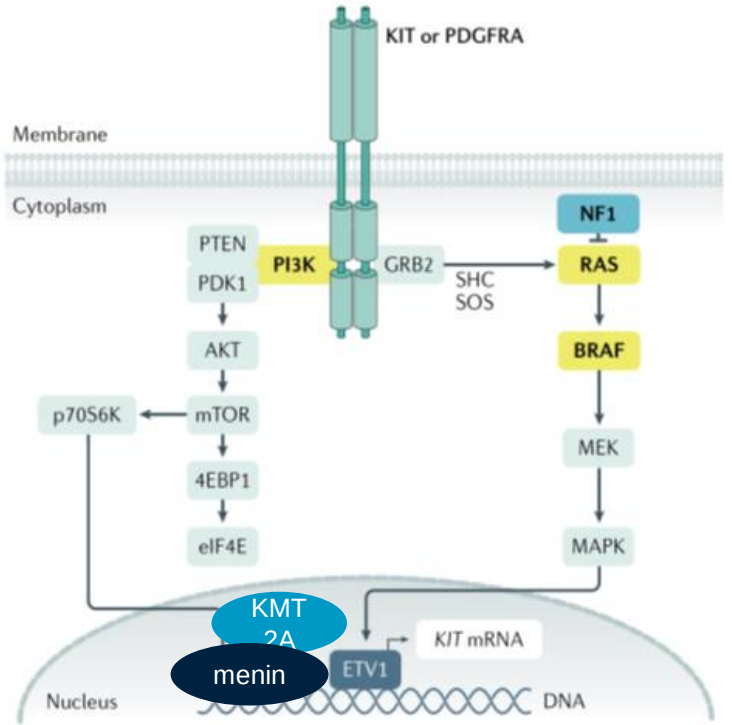
ZIFTOMENIB IN GASTROINTESTINAL STROMAL TUMORS (GIST)

GIST: A Unique Solid Tumor Opportunity for Ziftomenib

Advancing the treatment of TKI-resistant GIST

- Ziftomenib-imatinib combination treatment showed robust antitumor activity in both imatinib-sensitive and imatinib-resistant GIST PDX models representing the full GIST treatment continuum
- The combination of ziftomenib + imatinib exerts antitumor activity by a synthetic lethal mechanism through which ziftomenib epigenetically targets a vulnerability of GIST tumors actively induced by TKI treatments
- IND cleared for ziftomenib in advanced GIST in combination with imatinib
- Proof-of-concept study of ziftomenib + imatinib in patients with advanced GIST after imatinib failure expected to start in 1H 2025

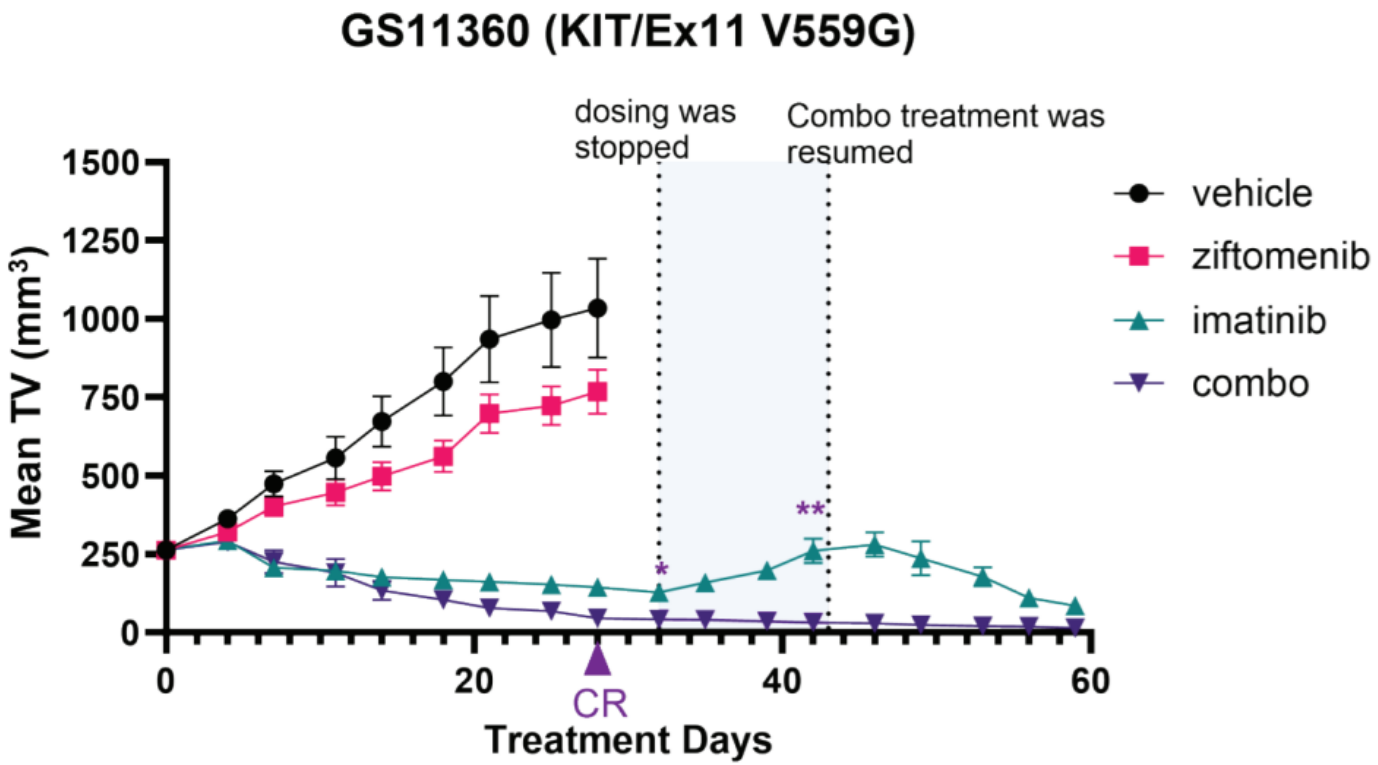
Signaling pathways of KIT-mutated GIST[1]



[1] Blay JY *et al*. Primer 2021, modified

Ziftomenib-Imatinib Combination Displays Durable Antitumor Activity in First Line GIST PDX Model

The combination induces regressions in tumors that have relapsed following imatinib treatment

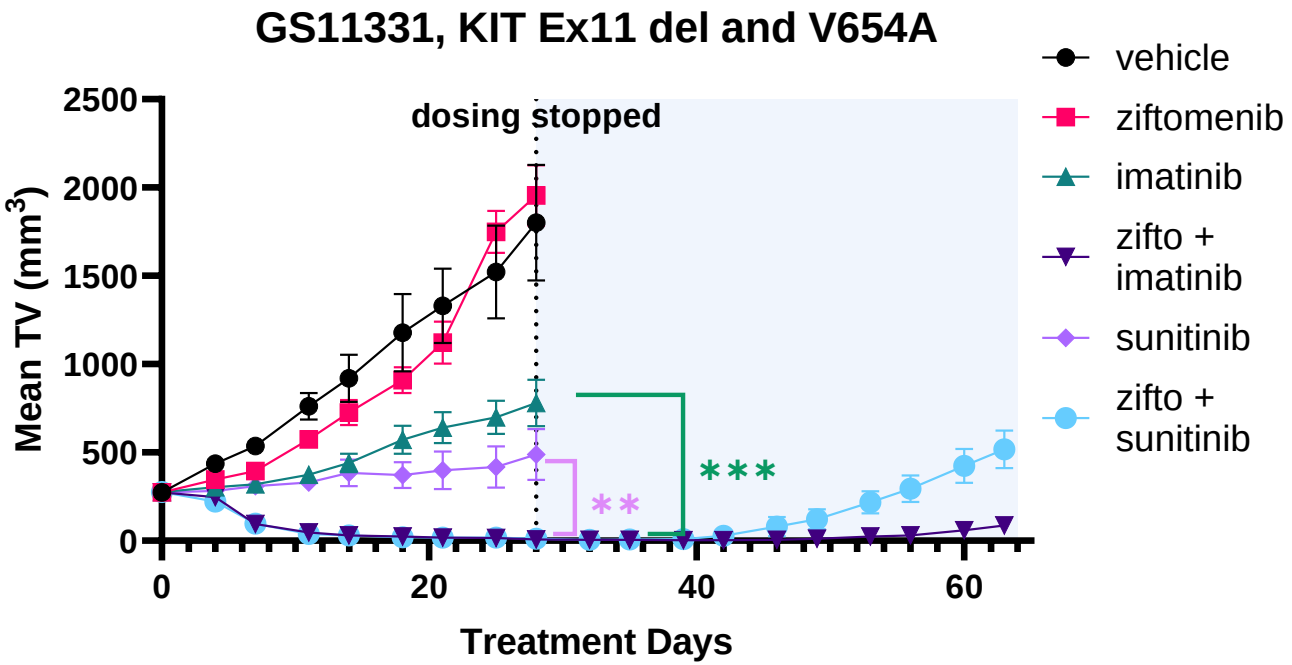


Data were presented as Mean ± SEM; t-test; *P<0.05,**P<0.01,***P<0.001,****P<0.0001, not significant (ns)

- Imatinib monotherapy was tumoristatic, but the ziftomenib-imatinib combination induced deep regressions in all animals, including some CRs
- Cessation of dosing resulted in rapid regrowth in imatinib-treated tumors, but tumors treated with the combination continued to regress
- The relapsed imatinib-treated tumors regressed when exposed to the combination

Ziftomenib and TKIs synergize in imatinib-resistant PDX models

Robust and durable responses seen in Ex13 GIST PDX model with combo



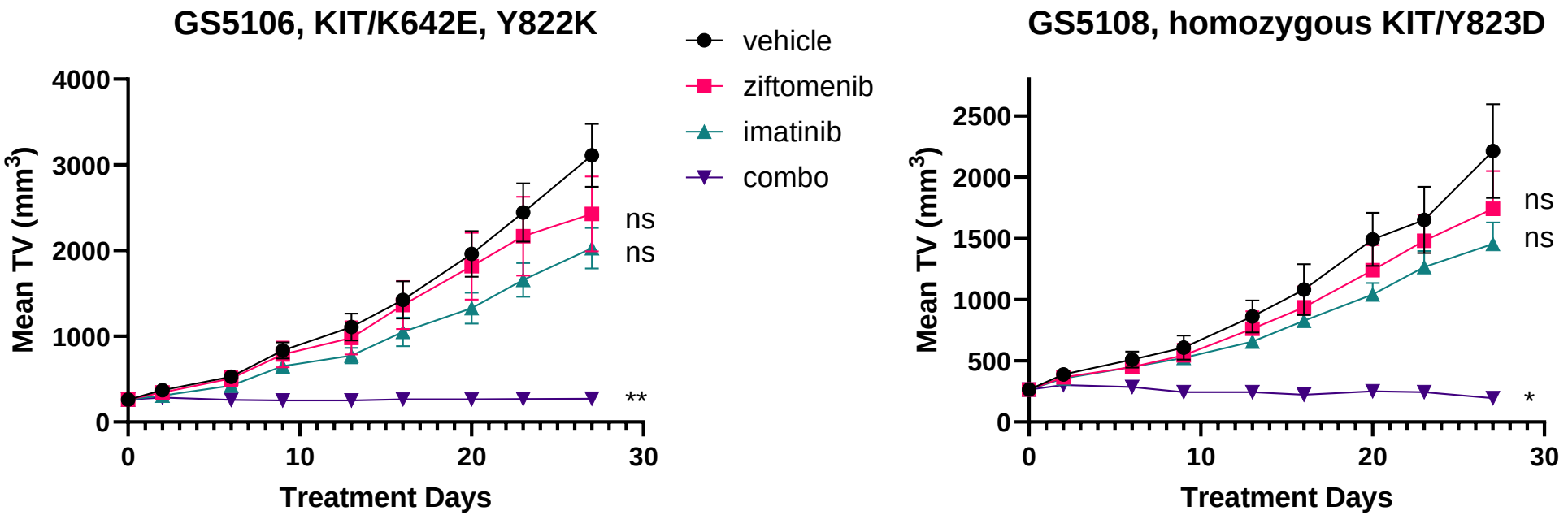
- Both TKIs synergized with ziftomenib, inducing deep regressions in all animals, including some complete responses
- Full suppression of tumor growth was maintained for up to four weeks after dosing stop

Data were presented as Mean ± SEM; t-test; *P<0.05,**P<0.01,***P<0.001,****P<0.0001, not significant (ns)

Ziftomenib and TKIs synergize in imatinib-resistant PDX models

Specific KIT mutation type does not seem to matter, with consistent imatinib-ziftomenib combo activity seen across different KIT mutant models

Synthetic lethal activity in Imatinib resistant Ex17 GIST PDX models



Data were presented as Mean ± SEM; t-test; *P<0.05,**P<0.01,***P<0.001,****P<0.0001, not significant (ns)

KURA ONCOLOGY

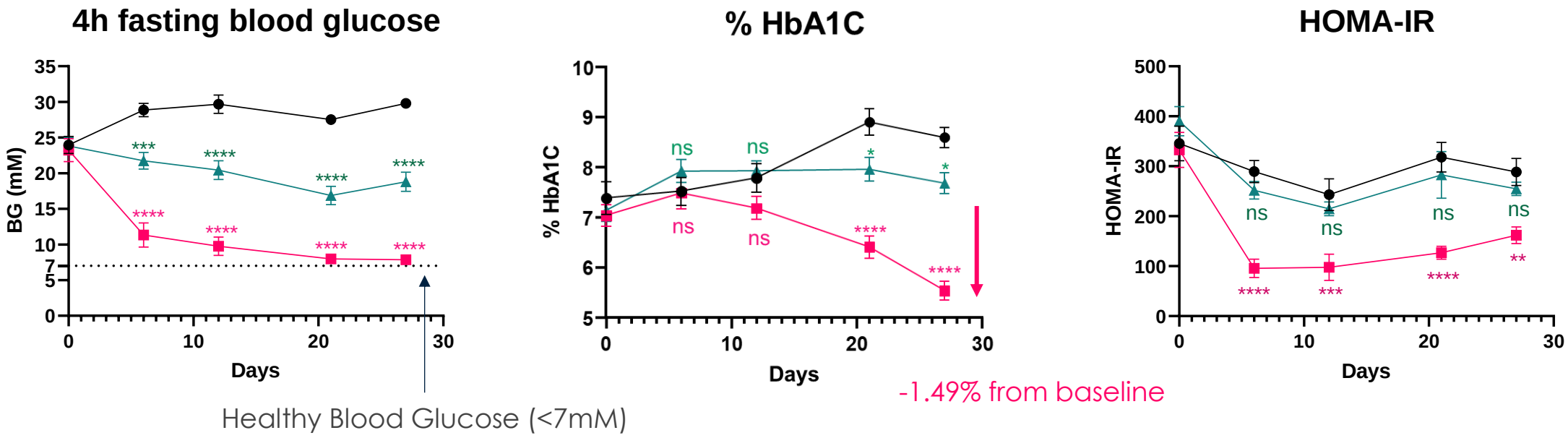
OPPORTUNITY FOR MENIN INHIBITOR IN DIABETES

Kura Menin Inhibitor Opportunity in Diabetes

Unique combination of effects on both insulin deficiency and insulin resistance

- Potential first-in-class regenerative medicine to restore β-cell mass
  - Prolonged activity observed for a month after dosing cessation
  - Selectivity for β-cells in islets offers safety advantages over e.g., DYRK1 inhibitors
- Robust increase in β-cell mass could extend the therapeutic application of menin inhibitors to T1D patients
- Additional unpredicted effects of menin inhibition on insulin resistance suggest combination opportunities with T2D SOC, e.g., metformin, semaglutide
- Nomination of next generation development candidate expected in 1H 2025

Ziftomenib Reduces Blood Glucose and HbA1C Levels and Improves Insulin Sensitivity in ZDF Rat Model of Type 2 Diabetes

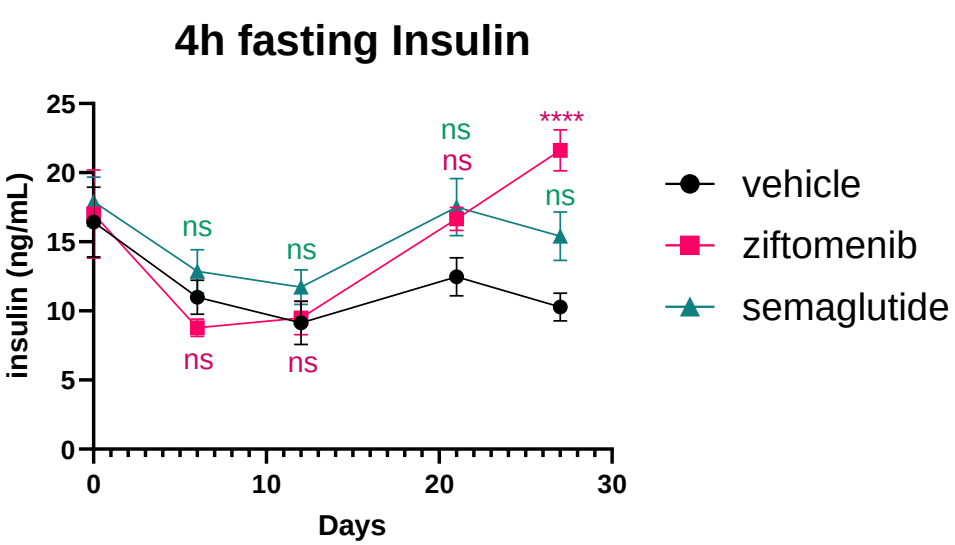


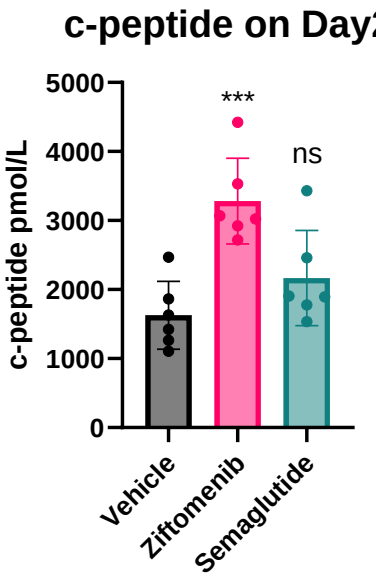
ns, not significant; *, p<0.05; **, p<0.01; ***, p<0.005; ****, p<0.001

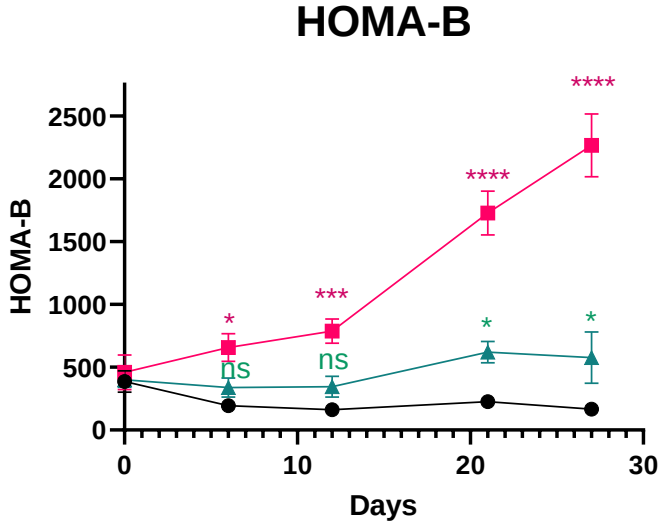
McCloskey *et al*. ADA 2024 #845-P
ZDF: Zucker Diabetic Fatty

Ziftomenib Stimulates Insulin Production

Ziftomenib significantly increased serum insulin levels and serum c-peptide levels, indicating significant improvement to steady-state β-cell function



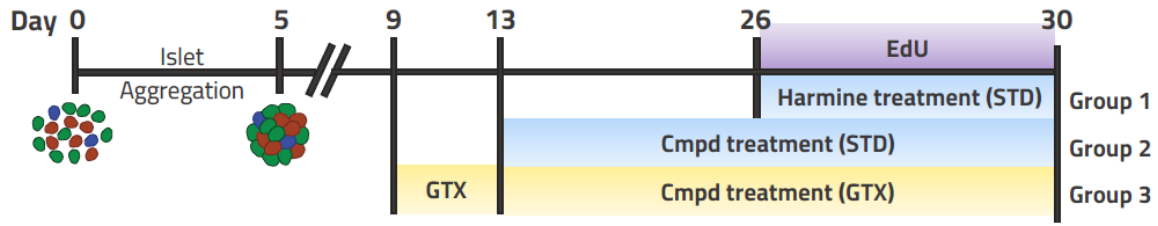




ns, not significant; *, p<0.05; **, p<0.01; ***, p<0.005; ****, p<0.001

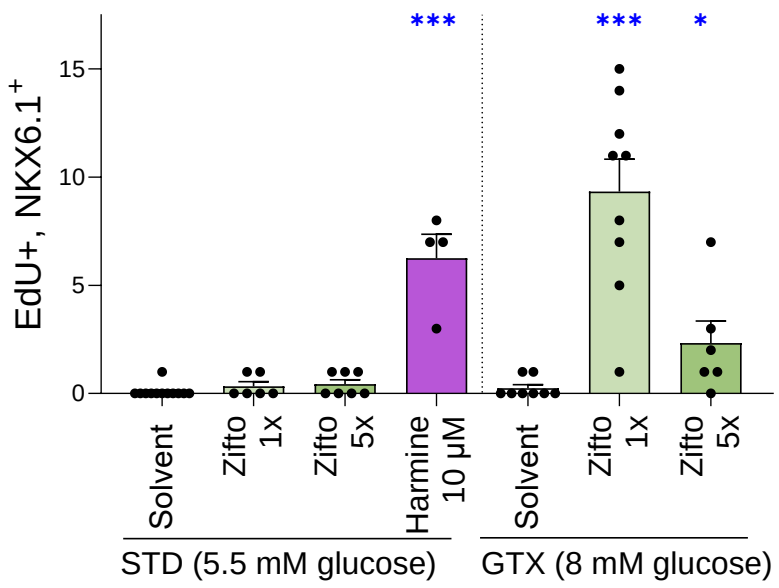
Ziftomenib Stimulates β-cell Proliferation with Minimal Effects on Non-β-cells in Human Pancreatic Islet Microtissues

Ziftomenib stimulated the proliferation of β-cells specifically in donor samples

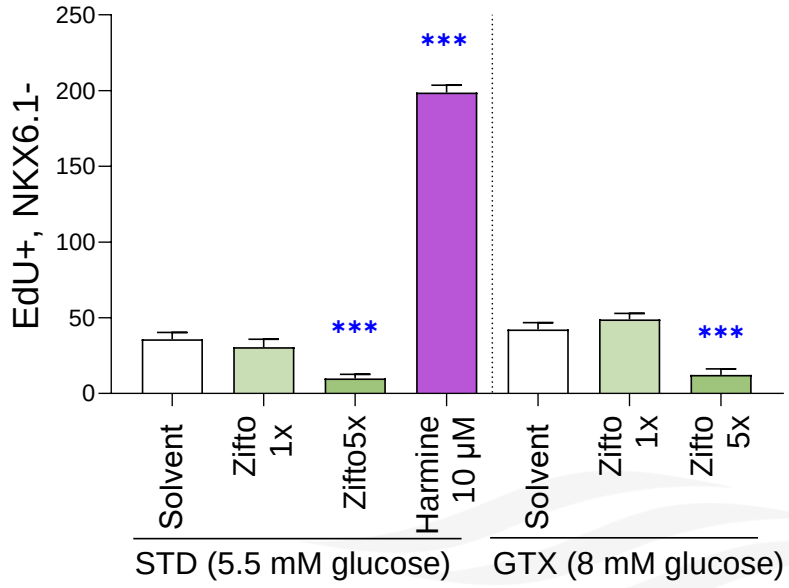


- Ziftomenib: 1x (nM) or 5x (nM)
- Harmine: 10 µM (only used in STD, toxic under GTX)
- Proliferating β-cells: NKX6.2+, EdU+
- Proliferating non-β-cells: NKX6.2-, EdU+

Donor-1 Proliferating β-Cell Count



Donor-1 Proliferating Non-β-Cell Count



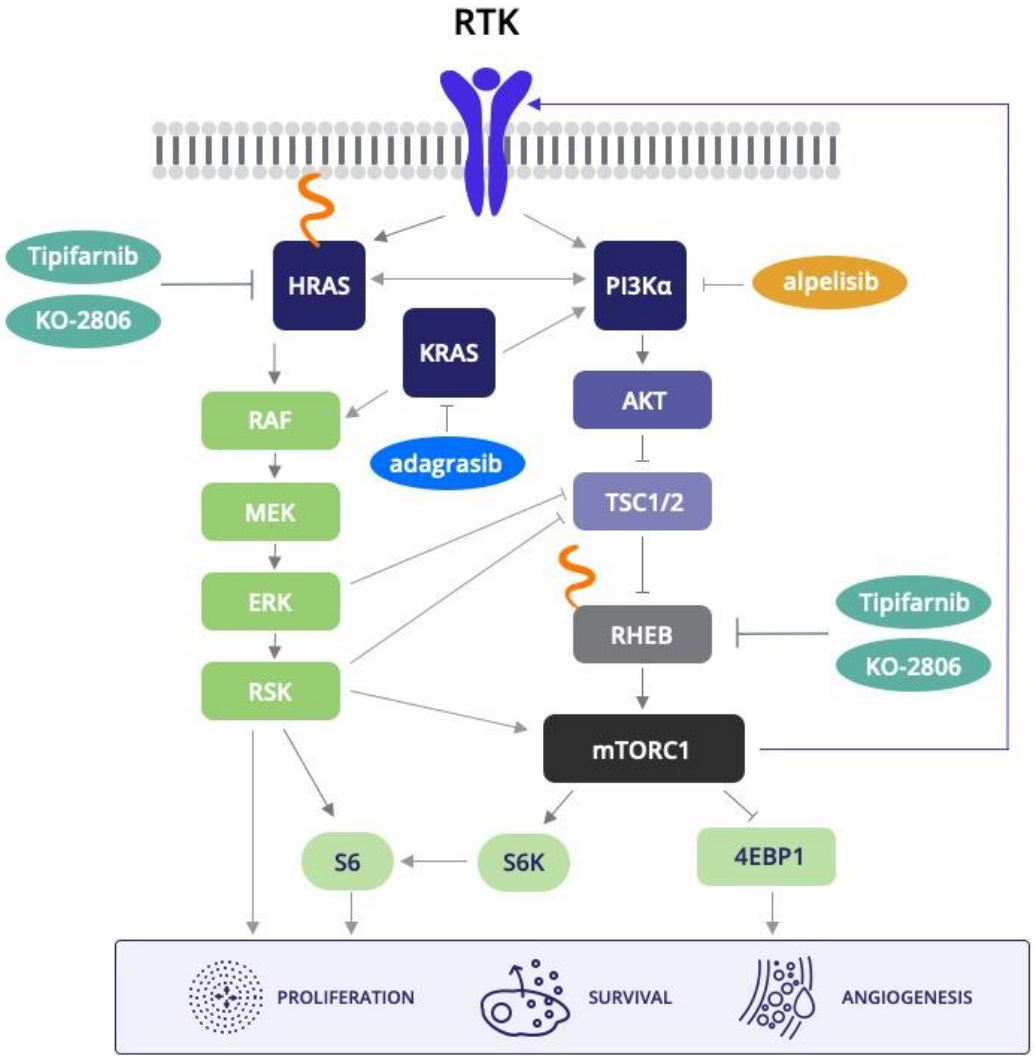
McCloskey *et al*. ADA 2024 #845-P

*, p<0.05; ***, p<0.005

GTX, glucotoxic conditions; STD, standard conditions

KO-2806: NEXT-GEN FARNESYL TRANSFERASE INHIBITOR

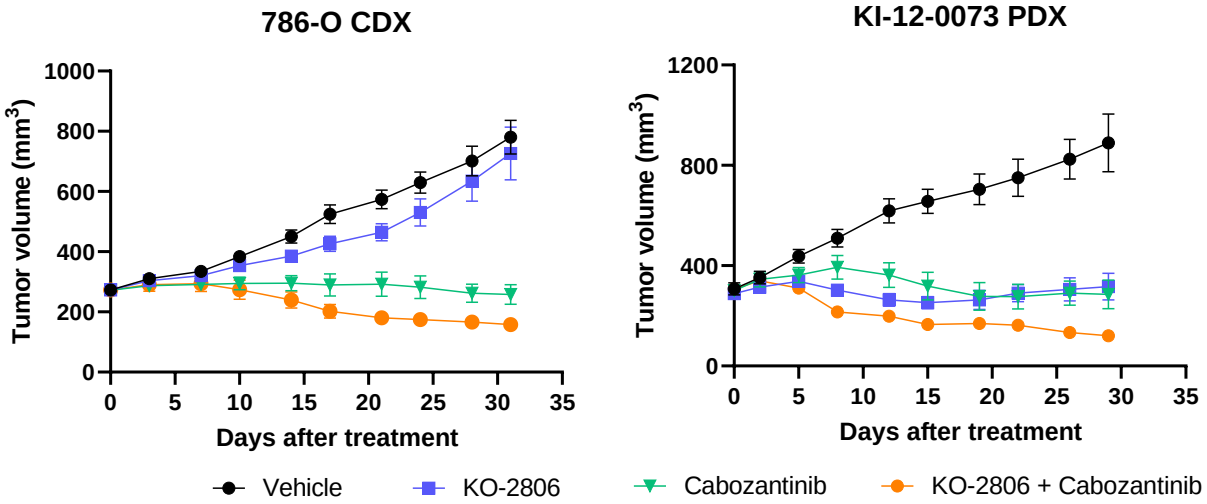
Therapeutic Applications of Farnesyl Transferase Inhibitors

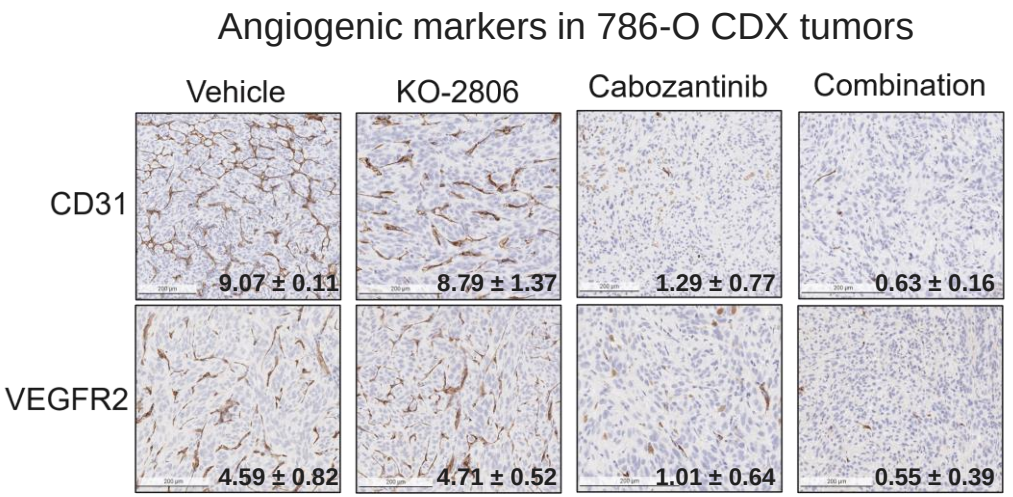


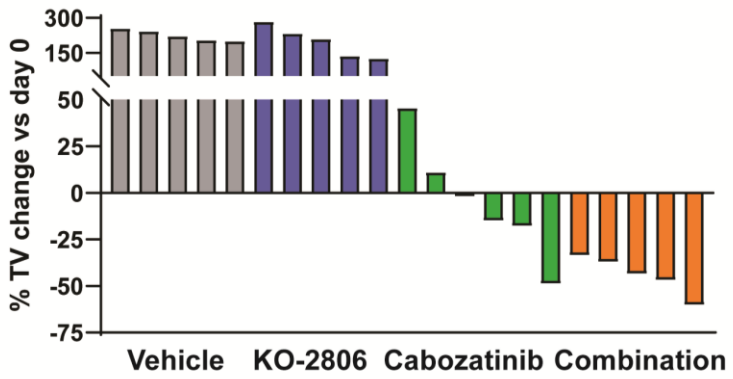
- Dysregulated RAS-MAPK and PI3Kα/AKT/mTOR signaling are key drivers of various cancers. Targeted cancer therapies such as alpelisib and adagrasib slow tumor progression by inhibiting individual elements in this complex signaling pathway
- However, resistance to these treatments develops through compensatory activation of complementary proteins, including receptor tyrosine kinases and mTOR
- Farnesyl Transferase Inhibitors (FTIs) can blunt the compensatory reactivation process by inhibiting farnesylation-mediated activation of additional proteins in the pathway – HRAS and RHEB
- By combining targeted therapies with FTIs, we believe we can reshape treatment options for many cancer patients

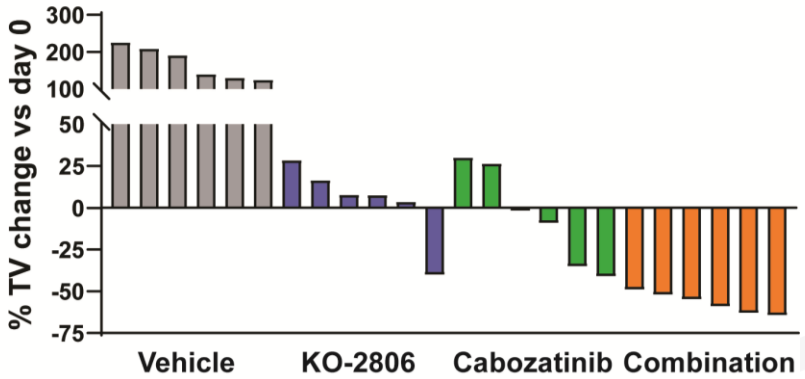
KO-2806 Potentiates the Antitumor Activity of Cabozantinib in ccRCC Models

The anti-angiogenic activity of cabozantinib is enhanced by addition of KO-2806





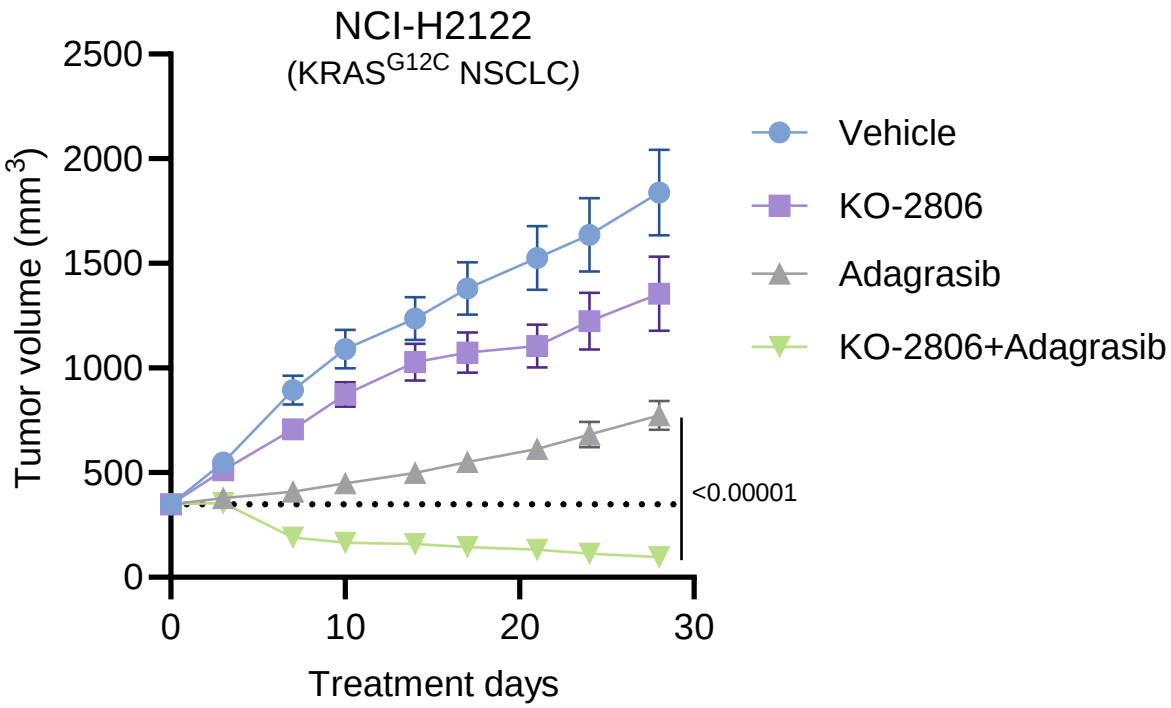


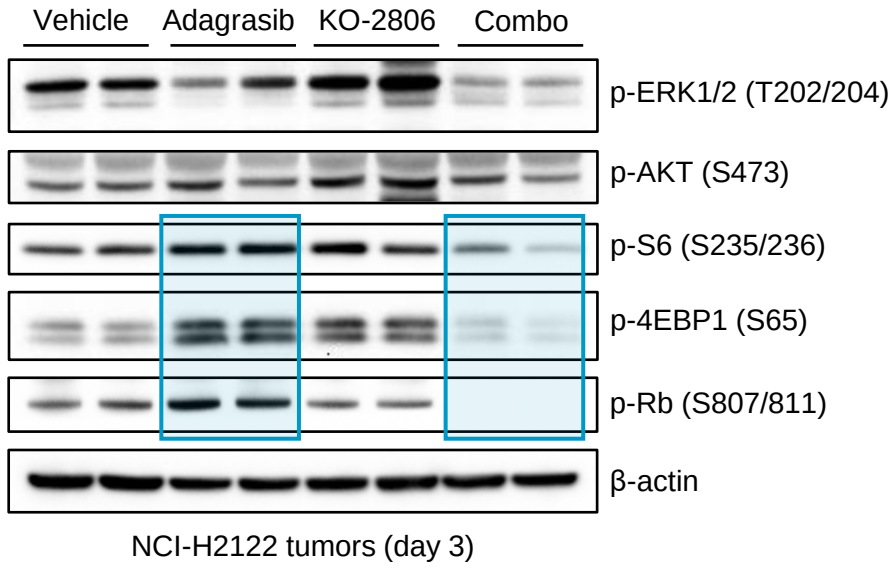


Gatchalian J. et al, AACR-NCI-EORTC 2023 Abstract #34634

Combination of KO-2806 Enhances the Antitumor Efficacy of a KRAS^{G12C} Inhibitor in NSCLC

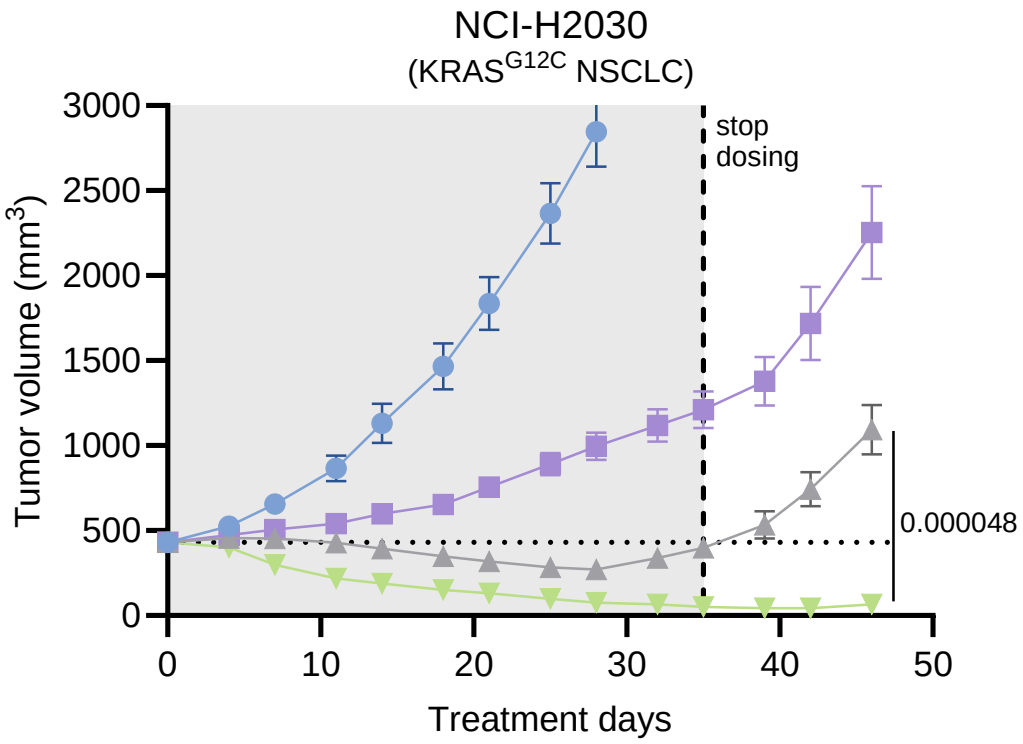
Combination of KO-2806 with adagrasib causes tumor regressions through suppression of mTOR signaling in KRAS^{G12C} NSCLC xenografts

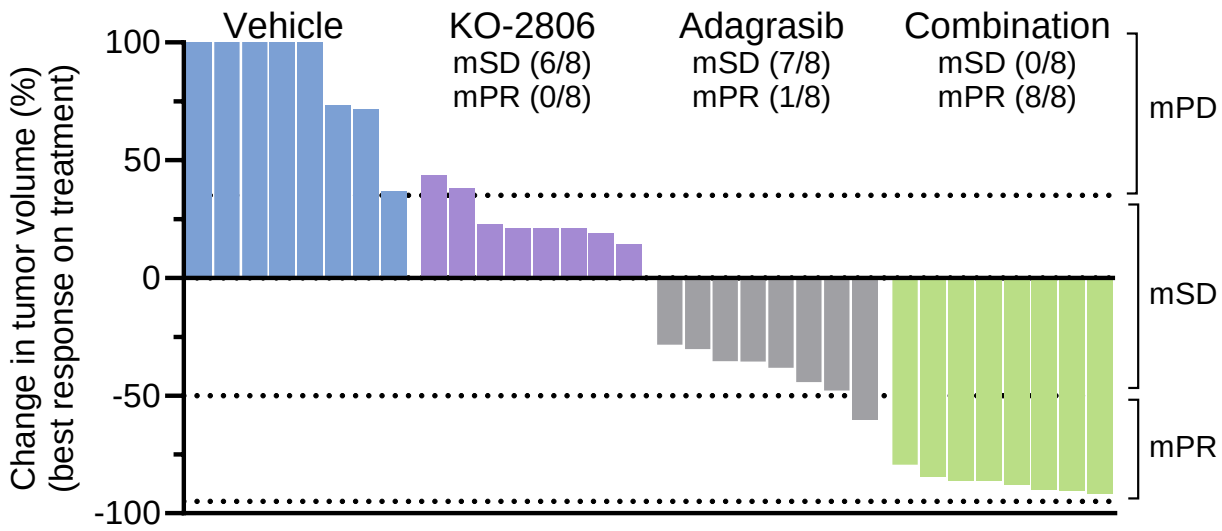




NCI-H2122 tumors (day 3)

Combination of KO-2806 Enhances the Depth and Duration of Response Compared to Adagrasib Alone

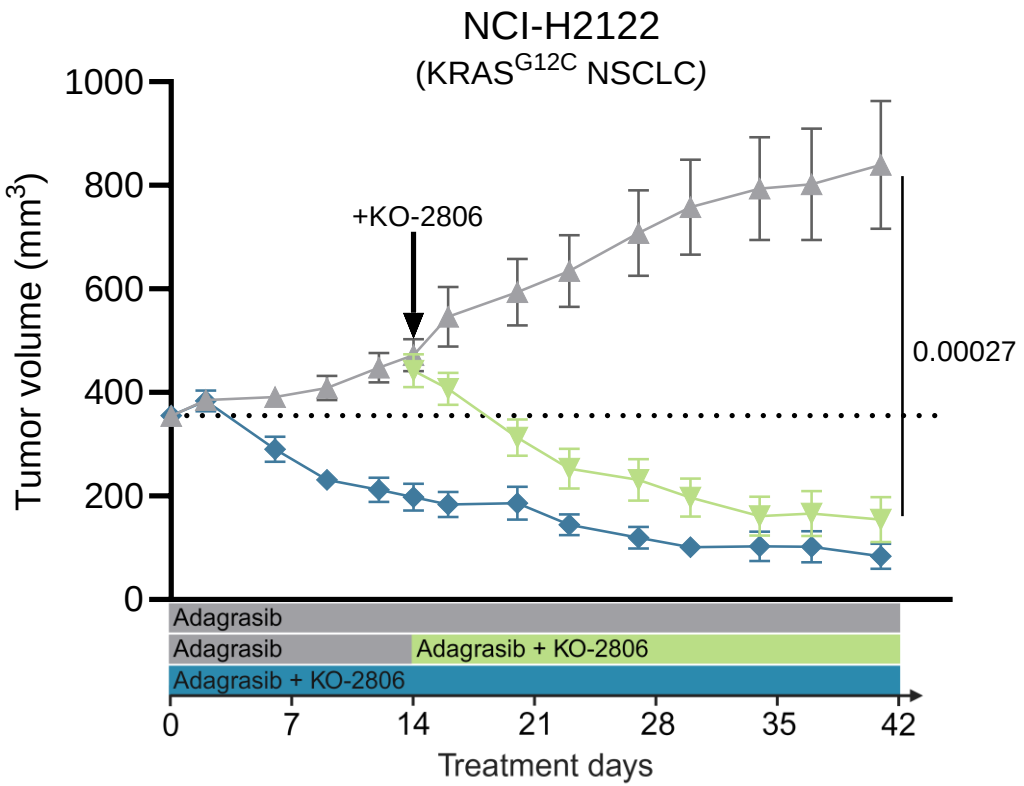


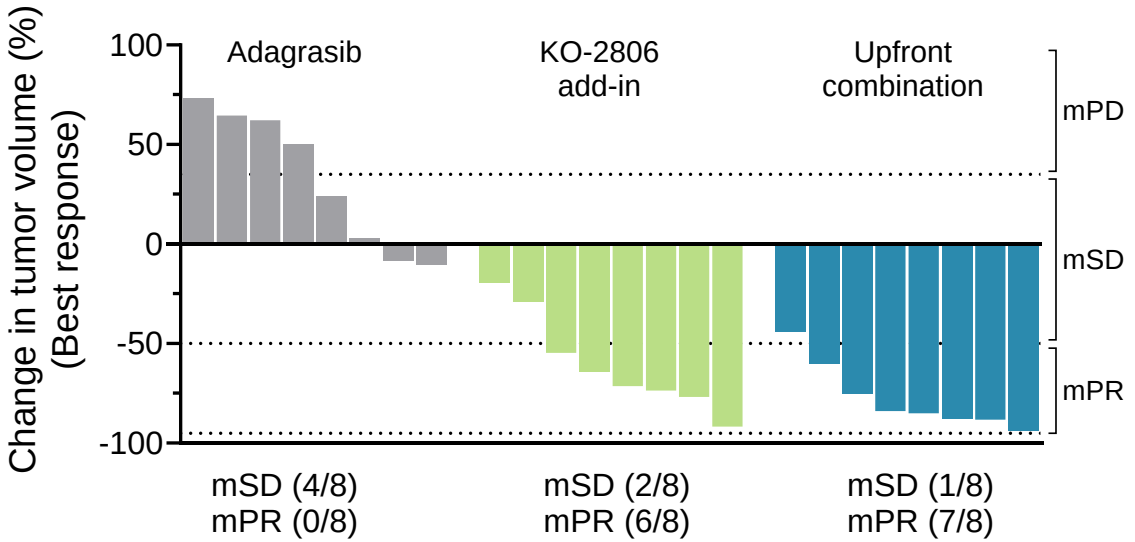


Patel H. *et al.* EORTC-NCI-AACR 2023 Abstract #34968

Tumors Progressing on the KRAS^{G12C} Inhibitor, Adagrasib, are Re-Sensitized by the Addition of KO-2806

Tumor regressions in the KO-2806 add-in group were comparable to the upfront combination of KO-2806 with adagrasib





FIT-001 Phase 1 First-in-Human Clinical Trial of KO-2806 in Patients with Advanced Solid Tumors

OBJECTIVES

Primary
- Evaluate the safety and tolerability of KO-2806 (dose escalation)
- Determine the MTD/HPDD and/or the OBAD of KO-2806 (dose escalation)
- Define the RP2D of KO-2806 (dose expansion)
- Evaluate the antitumor activity of KO-2806 in combination with cabozantinib in ccRCC and adagrasib in $KRAS^{G12C}$-mutant NSCLC (dose expansion)

Secondary
- Evaluate the safety and tolerability of KO-2806 (dose expansion)
- Evaluate the preliminary antitumor activity of KO-2806 (dose escalation and dose expansion)
- Characterize the PK of KO-2806 when administered as monotherapy, and the PK of KO-2806 and the combination agents when administered in combination therapy (dose escalation and expansion)

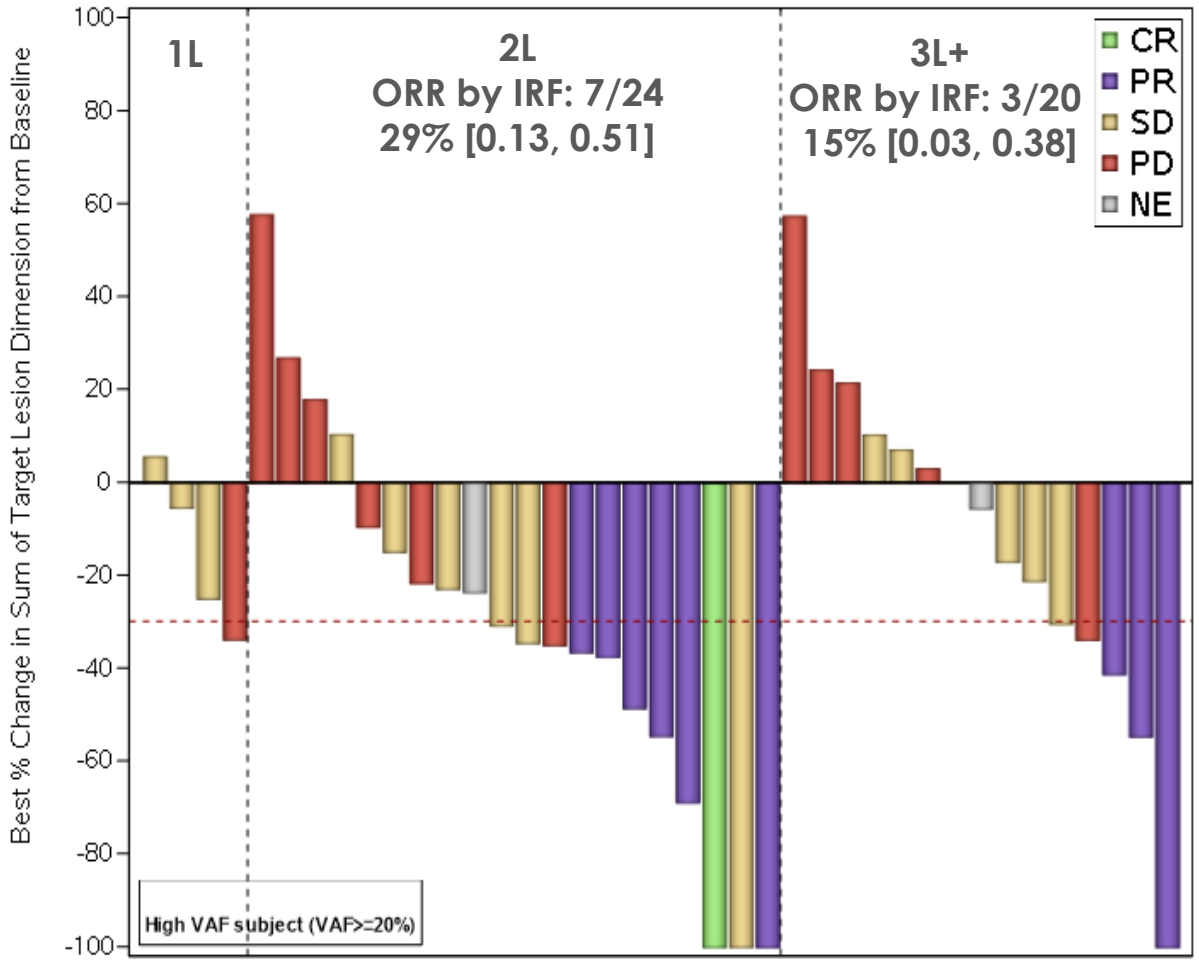
Ongoing enrollment in FIT-001 Phase 1 dose-escalation trial of KO-2806 as a monotherapy and in combination

MTD/HPDD = maximum tolerated dose/highest protocol-defined dose; OBAD = optimal biologically active dose

KURA ONCOLOGY

TIPIFARNIB: FARNESYL TRANSFERASE INHIBITOR

Tipifarnib Shows Clinical Benefit in HRAS mutant HNSCC



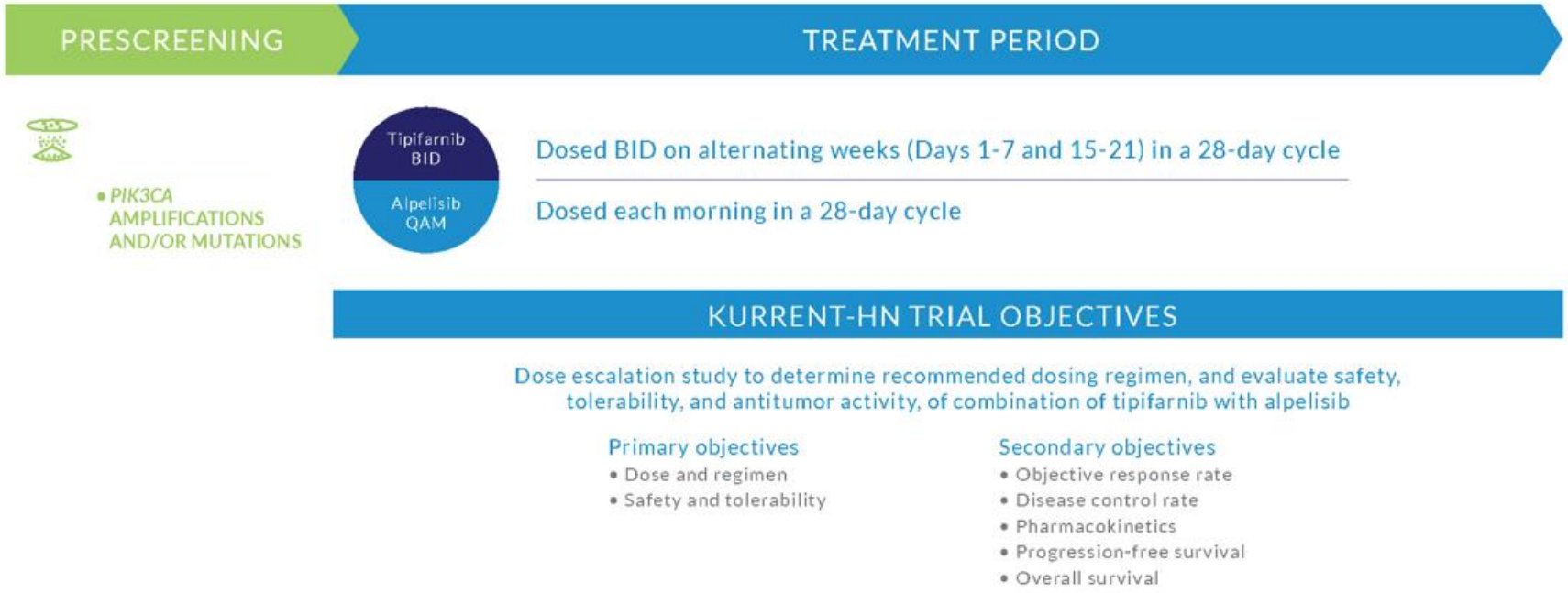
6/10 responders had BOR of PD in the last prior line with IO-based therapies
PFS in these ranged from 1-5 months vs. 6 –27 months on tipifarnib

Patients with **High VAF** in mITT (N=50)		
	Investigator Assessment	**Independent Review Facility**
Best Overall Response, n (%)		
Confirmed CR	1 (2)	1 (2)
Confirmed PR	14 (28)	9 (18)
SD	17 (34)	14 (28)
PD	6 (12)	14 (28)
NE	12 (24)	12 (24)
DCR, n (%) [95% CI]	32 (64) [0.49, 0.77]	24 (48) [0.34, 0.63]
ORR, n (%) [95% CI]	15 (30) [0.18, 0.45]	10 (20) [0.10, 0.34]
mDoR, months [95% CI]	5.6 [3.88, 9.23]	6.5 [3.88, -]
mPFS, months [95% CI]	3.7 [2.60, 5.55]	2.6 [1.87, 4.40]

mITT: Patients treated with at least one dose of Tipifarnib. CR, complete response; PR, partial response; BOR, best overall response; IO, immuno-oncology; SD, stable disease; PD, progressive disease; NE, not evaluable; -, not calculable; ORR: objective response rate; DCR, disease control rate; mDoR, median duration of response; mPFS, median progression free survival.

Ho et al, ESMO 2023 #LBA47

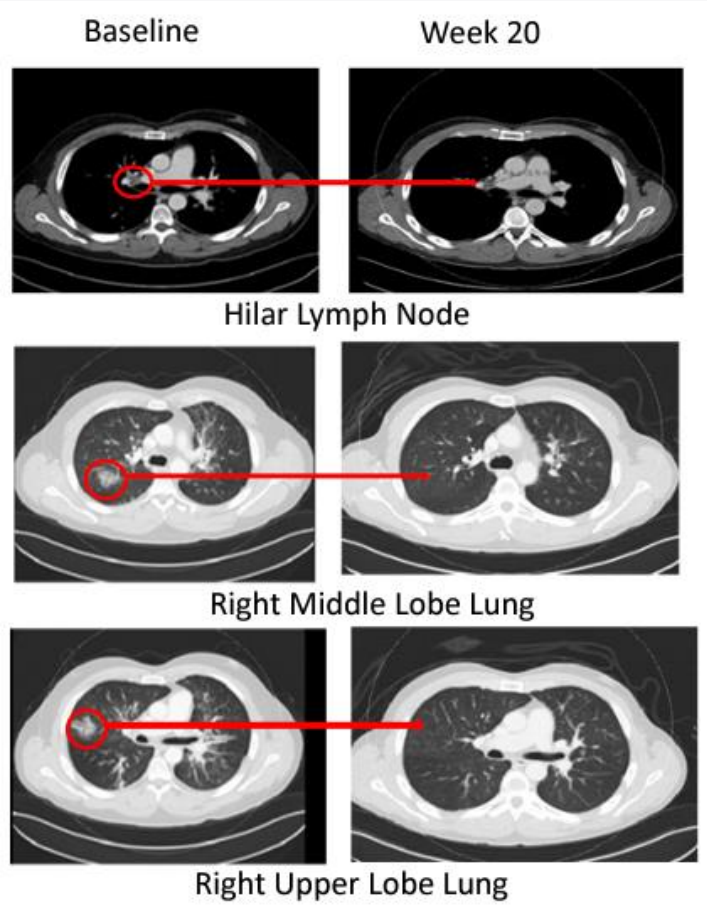
KURRENT-HN: PHASE 1/2 Combination Trial of Tipifarnib and Alpelisib in Patients with HNSCC



Phase 1 clinical trial of tipifarnib and alpelisib in patients with recurrent/metastatic *PIK3CA*-amplified and/or *PIK3CA*-mutated HNSCC

- Clinical collaboration to evaluate tipifarnib in combination with alpelisib for the treatment of patients with HNSCC whose tumors have PIK3CA mutation and/or amplification
- Under the collaboration, Kura sponsors the trial and supplies tipifarnib and Novartis supplies alpelisib

Durable Clinical Response Observed in Patient with PIK3CA-dependent HNSCC



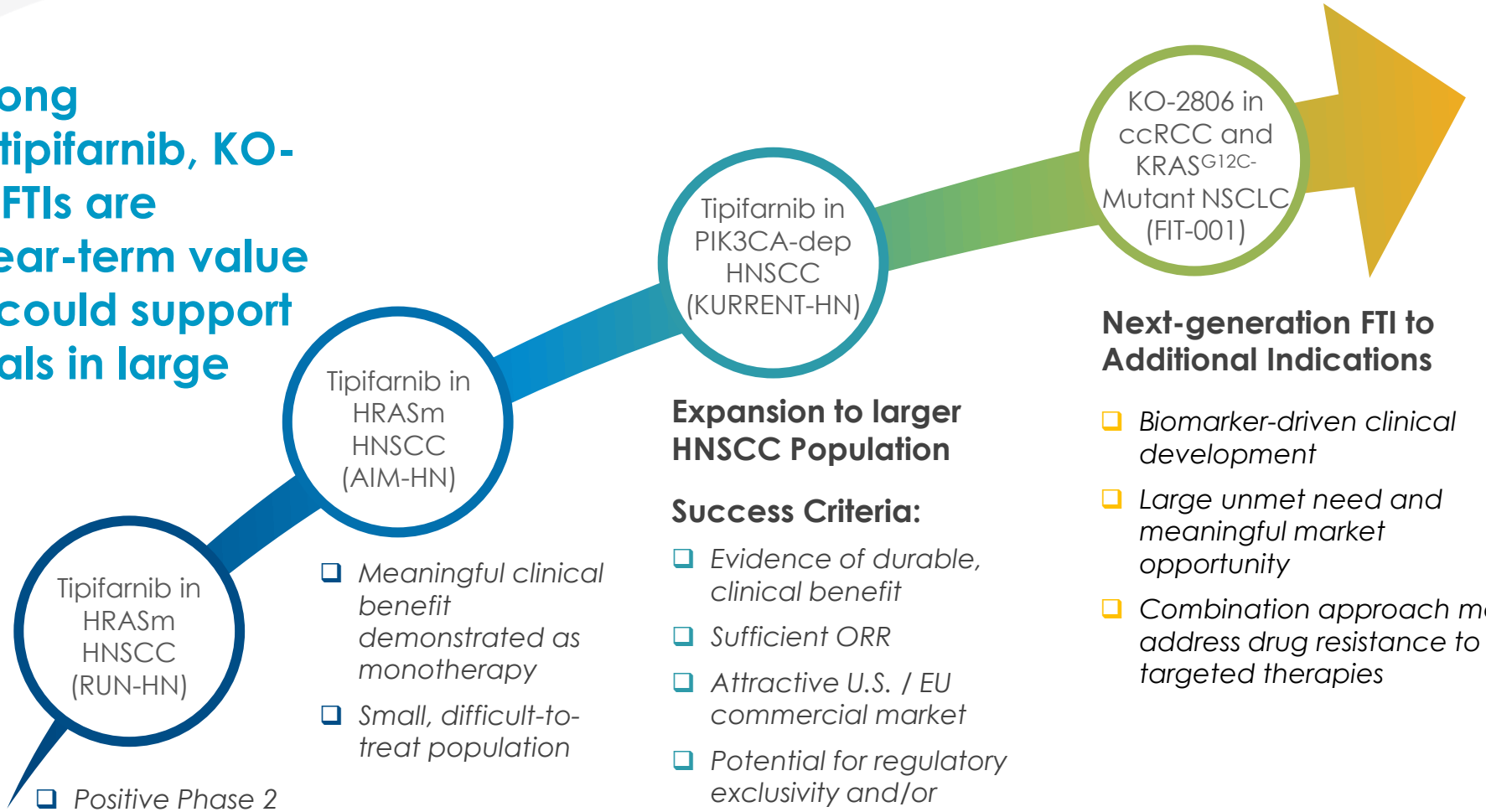
- 35yo, male, nonsmoker, HPV16 positive
- SCC of tonsil Stage III cT4N2M0; PD-L1 CPS = 60
- Prior Treatments
  - CDDP/rad for 1 mo (Nov-Dec2019), BOR UNK
  - Cemiplimab/ISA101b (Jun-Nov2021), BOR PD
- PIK3CA R88Q mutation (44%) and HRAS OE (3+ staining in 100% of tumor cells) by IHC from May 2021 biopsy
- DL1 tipifarnib, DL2 alpelisib; completed 6 cycles
- G1/2 TRAE, G3 lipase elevation; presented clinical benefit and improvement in respiratory symptoms
- 81% reduction in target lesions after 1 cycle of treatment
- 84% reduction in target lesions after 3 cycles (BOR)
- Continued on-study for >27 weeks maintaining QoL

Additional data from KURRENT-HN clinical trial expected to be presented in 1H 2025

Soifer H et al, ENA 2022 PB041; Data cut as of 14Sep2022; Preliminary raw data

FTI Franchise Development Strategy

Building on a strong foundation with tipifarnib, KO-2806 and future FTIs are positioned for near-term value inflections, and could support multiple approvals in large indications



Forecasted Milestones & Financial Highlights

PROGRAM	MILESTONE	ESTIMATED TIME OF ACHIEVEMENT
ZIFTOMENIB **Menin Inhibitor**	Present updated data from KOMET-007 trial in combination with ven/aza and 7+3	ASH 2024
	Report topline results from KOMET-001 registration-directed trial in NPM1-mutant R/R AML	Early 2025
	Present preliminary data from Phase 1b expansion portion of KOMET-007	2025
	Initiate proof-of-concept study in combination with imatinib in patients with advanced GIST	1H 2025
	Nominate a next generation menin inhibitor development candidate	1H 2025
KO-2806 **Next-Generation Farnesyl Transferase Inhibitor**	Identify maximum tolerated dose as monotherapy	2H 2024
	Initiate one or more expansion cohorts in combination with cabozantinib in ccRCC	1H 2025
TIPIFARNIB **Farnesyl Transferase Inhibitor**	Identify OBAD in combination with alpelisib in PIK3CA-dependent HNSCC	End of 2024
	Present data from KURRENT-HN trial in combination with alpelisib in PIK3CA-dependent HNSCC	1H 2025

Financial Highlights Nasdaq: KURA	$455.3M in pro forma cash as of September 30, 2024* provides runway into 2027
	Shares outstanding as of September 30, 2024: 77.7M basic; 24.5M options, RSUs, PSUs, warrants & pre-funded warrants

OBAD = optimal biologically active dose
* Cash, cash equivalents and short-term investments

DEVELOPING PRECISION MEDICINES FOR THE TREATMENT OF CANCER

Corporate Presentation – November 2024