

DEVELOPING PRECISION MEDICINES FOR THE TREATMENT OF CANCER

Corporate Presentation – February 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, tipifarnib and KO-2806, 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 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; the commencement, enrollment and completion of clinical trials and the reporting of data therefrom; 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 therapies, forging new scientific and clinical paths to give patients a better chance for long-term, durable remissions
Proprietary Pipeline	Menin Inhibitor Program (Ziftomenib) <ul style="list-style-type: none">• Potential to address up to 50% of acute leukemias through monotherapy and combinations• 35% CR rate among 20 patients with NPM1-mutant AML treated at recommended Phase 2 dose• Positive preliminary combination data in NPM1-m and KMT2A-r AML, including 100% CR rate with 7+3 in 1L, 56% CR/CRh rate with ven/aza in R/R menin inhibitor naïve patients and mitigation of differentiation syndrome• Completion of enrollment in Phase 2 registration-directed trial in NPM1-mutant AML expected by mid-2024
	Farnesyl Transferase Inhibitor Programs (Tipifarnib & KO-2806) <ul style="list-style-type: none">• Durable responses observed with tipifarnib as a monotherapy in R/M HRAS-mutant HNSCC patients• Compelling safety profile and activity observed with tipifarnib plus alpelisib in PIK3CA-dependent HNSCC• Preclinical data support clinical combinations of next-gen FTI KO-2806 with adagrasib and cabozantinib• Clinical collaboration with BMS to evaluate KO-2806 and adagrasib in KRAS^{G12C}-mutated NSCLC• Now dosing patients in dose-escalation trial of KO-2806 as monotherapy; combos to start by mid-2024
Strong Financials	<ul style="list-style-type: none">• \$25 million strategic equity investment from Bristol Myers Squibb• \$570 million in <i>pro forma</i> cash* provides runway into 2027

* Includes \$424M in cash, cash equivalents and short-term investments as of 12/31/23 and estimated proceeds net of offering expenses of \$146M from private placement closed on January 26, 2024



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 (Relapsed/refractory)	NPM1-mutant acute myeloid leukemia (AML)				Complete enrollment of 85 patients by mid-2024
		KMT2A-rearranged acute lymphoblastic leukemia				Now dosing patients
		Non-NPM1-mutant / Non-KMT2A-rearranged AML				Dose first patients by mid-2024
	KOMET-007 Combination with venetoclax + azacitidine (Relapsed/refractory)	NPM1-mutant AML				Determine RP2D and initiate dose validation/expansion in frontline AML patients by mid-2024
		KMT2A-rearranged AML				
	KOMET-007 Combination with cytarabine + daunorubicin (Frontline)	NPM1-mutant AML				Determine RP2D by mid-2024
		KMT2A-rearranged AML				
	KOMET-008 Combinations with gilteritinib, FLAG-IDA, LDAC (Relapsed/refractory)	NPM1-mutant AML				Now dosing patients
		KMT2A-rearranged AML				
TIPIFARNIB Farnesyl Transferase Inhibitor (FTI)	KURRENT-HN Combination with alpelisib	PIK3CA-dependent head and neck squamous cell carcinoma (HNSCC)				Currently enrolling patients at two different dose cohorts to determine optimal biologically active dose by end of 2024
KO-2806 Next-Generation FTI	FIT-001 Monotherapy, combinations with cabozantinib and adagrasib	Solid tumors				Now in dose escalation as monotherapy
		Clear cell renal cell carcinoma (ccRCC)				Dose first patients in combo with cabozantinib by mid-2024
		KRAS ^{G12C} -mutant non-small cell lung cancer (NSCLC)				Dose first patients in combo with adagrasib by mid-2024

ZIFTOMENIB: MENIN-KMT2A/MLL INHIBITOR IN ACUTE LEUKEMIAS



Ziftomenib Demonstrates Potential to Become a Cornerstone of AML Therapy

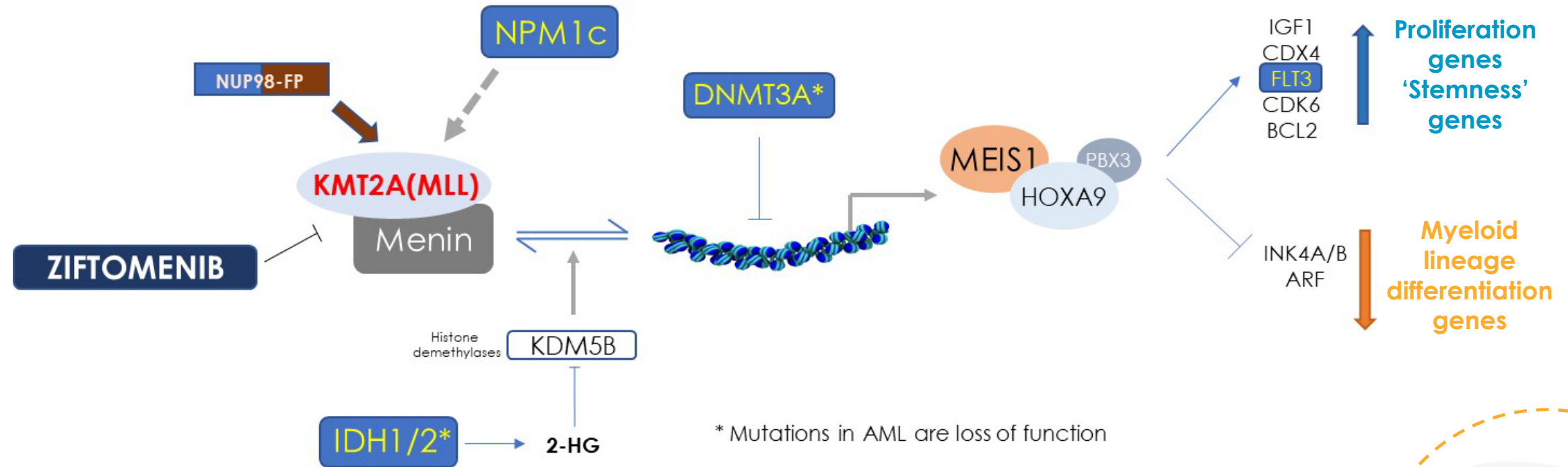
Targets foundational mutations at the core of up to 50% of AML cases

- **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
 - KOMET-001 monotherapy registrational trial expected to complete enrollment by mid-2024

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

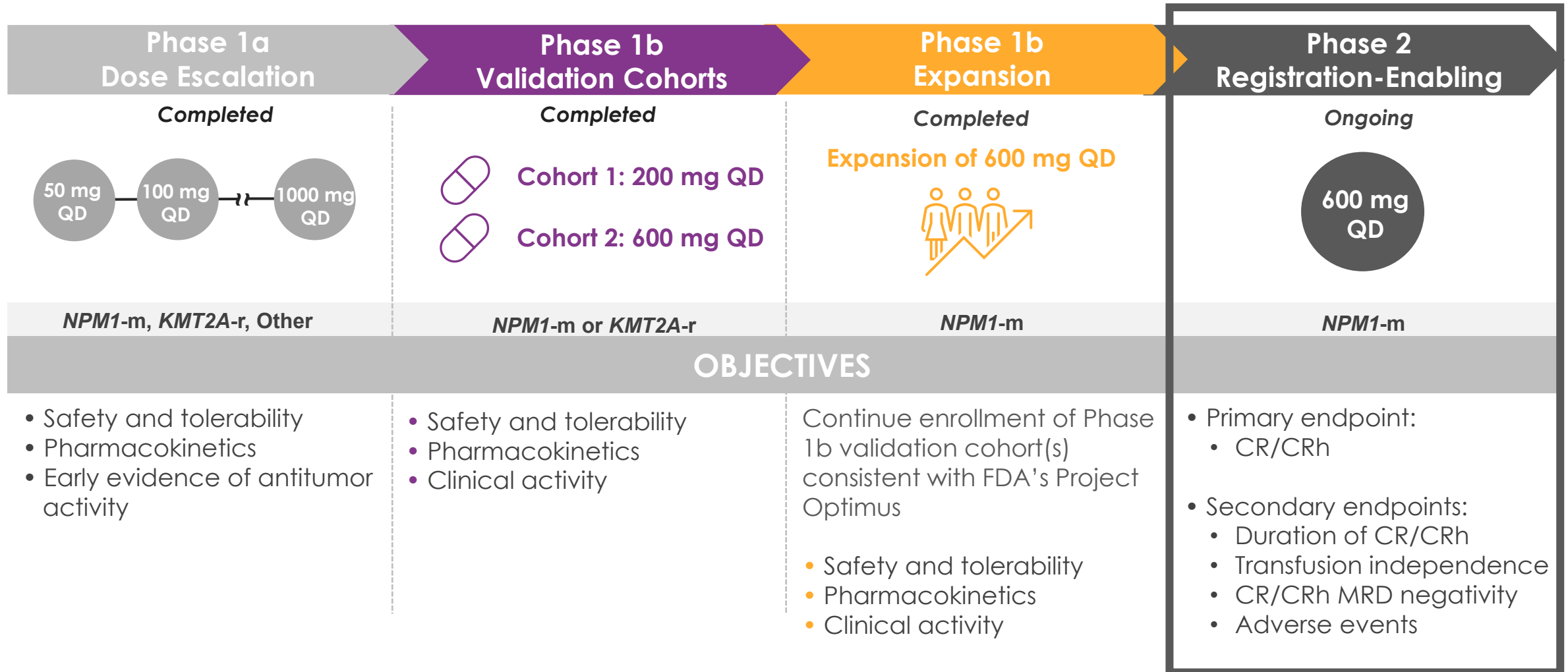


- *NPM1*-m and *KMT2A*-r drive overexpression of *HOXA9/MEIS1* genes, critical for transformation to AML
- *KMT2A*(*MLL*) sits upstream from major AML targets (*i.e.*, *FLT3*, *IDH1/2*, *DNMT3A*)
- *KMT2A*(*MLL*)-dependent genes contribute to therapeutic resistance and relapse to current therapies
-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





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

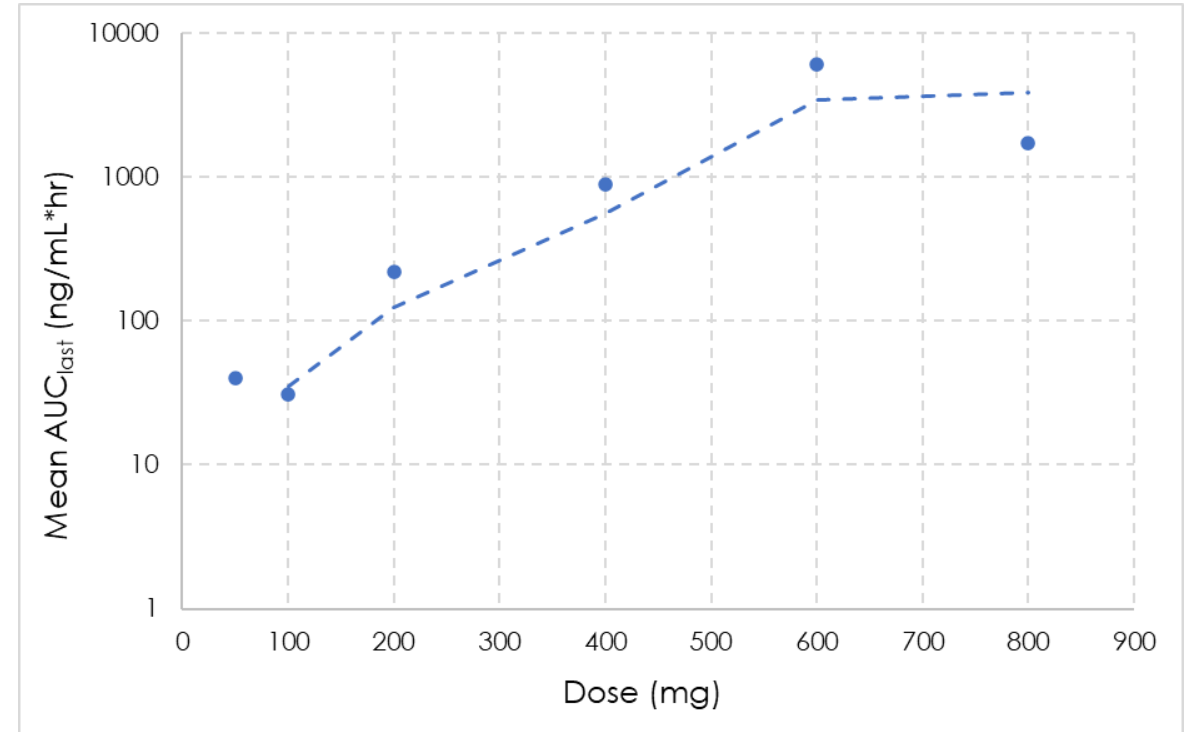




Ziftomenib Demonstrates Optimal Pharmaceutical Properties

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
 - No dose adjustment needed for CYP3A4 substrates (e.g., venetoclax)
- No drug-induced QTc prolongation observed at any dose





Ziftomenib Demonstrates Encouraging Safety Profile in Phase 1b

- 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 38.5% at 200 mg and 37.5% 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



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)

(preliminary data as of April 12, 2023)

Differentiated CR Rates vs. SOC in Heavily Pretreated Patients

	MUTATION	CR %	mDOR	MEDIAN PRIORS
Ziftomenib 600mg QD	NPM1m	35%	8.2 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

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

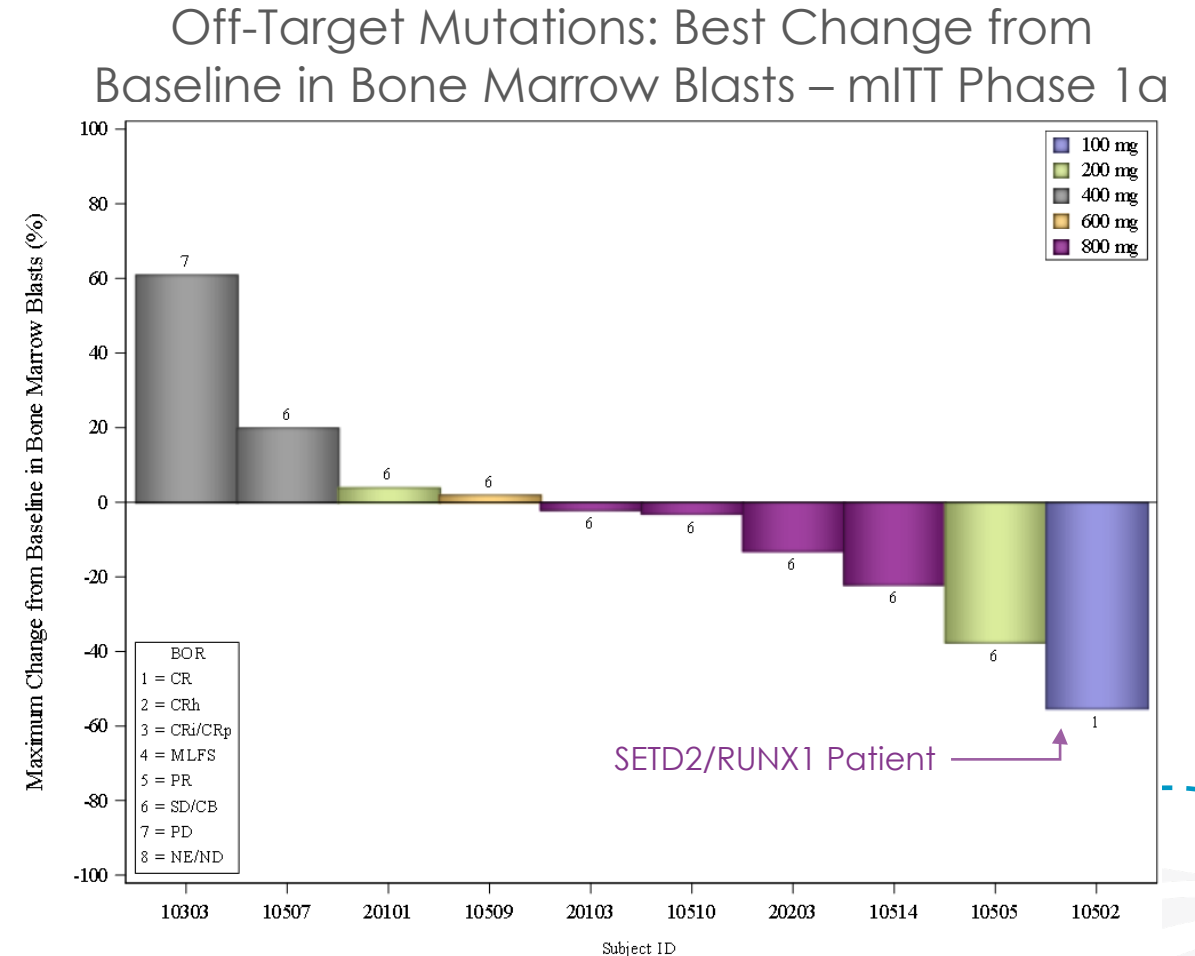
- CRmrd- after Cycle 1
- HSCT
- CRmrd- maintained on Cycle 2 post-HSCT

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/*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





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

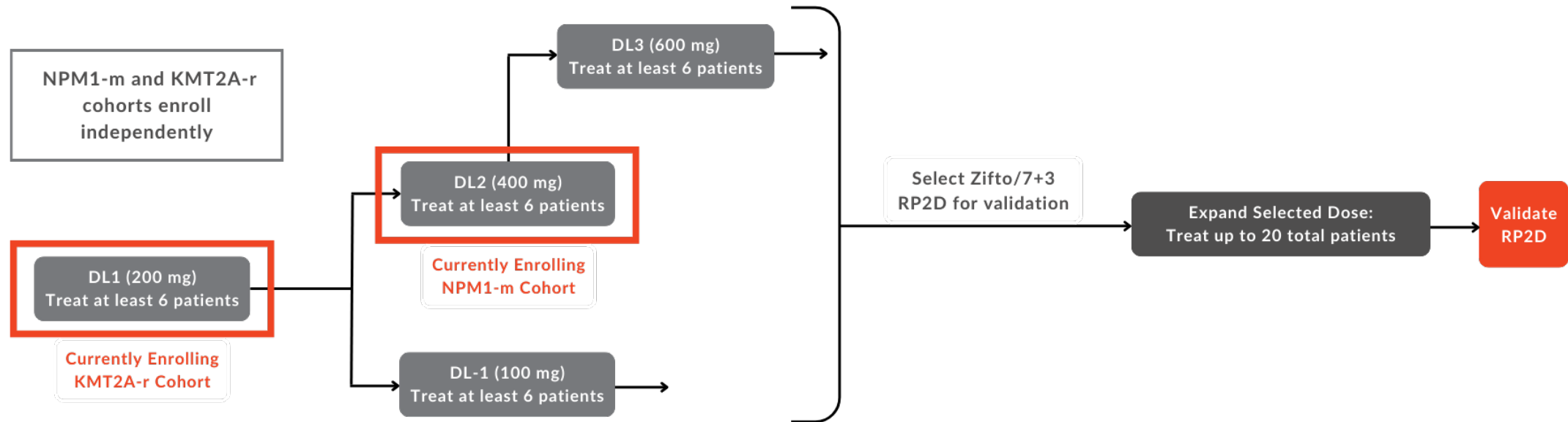
Monotherapy data supportive of combination strategies

- 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



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 be administered continuously 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*

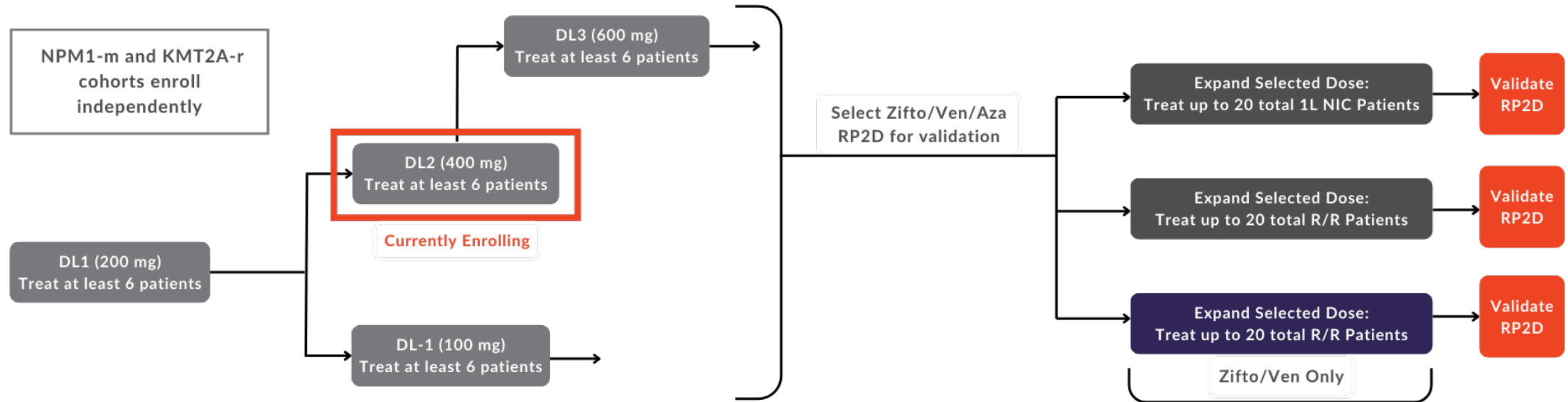
*Age ≥ 60 years and/or treatment-related AML and/or adverse risk cytogenetics per ELN

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 be administered continuously 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 Day 1-7 of each cycle with additional cycles based on bone marrow biopsy results



KOMET-007: Promising Safety and Tolerability Profile in Combination

Combinations mitigate risk of differentiation syndrome (DS)

Grade \geq 3 TEAEs (\geq 10%)	n (%)
Patients with Grade \geq 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 \geq 3 Ziftomenib-Related AEs (All)	n (%)
Patients with Grade \geq 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 to date, including delayed hematologic count recovery
- No QTc prolongation observed
- TEAEs consistent with underlying disease and backbone therapies



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 patients¹
- 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 400 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 response rate in KMT2A-r R/R AML < 10% ORR⁴

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 400 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)

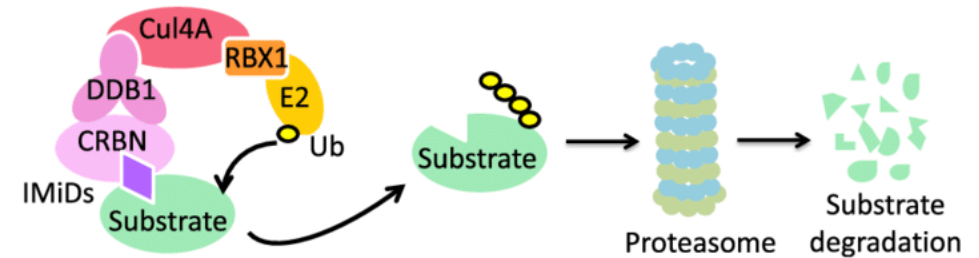
ORR includes CR, CRh, CRi, MLFS



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.**

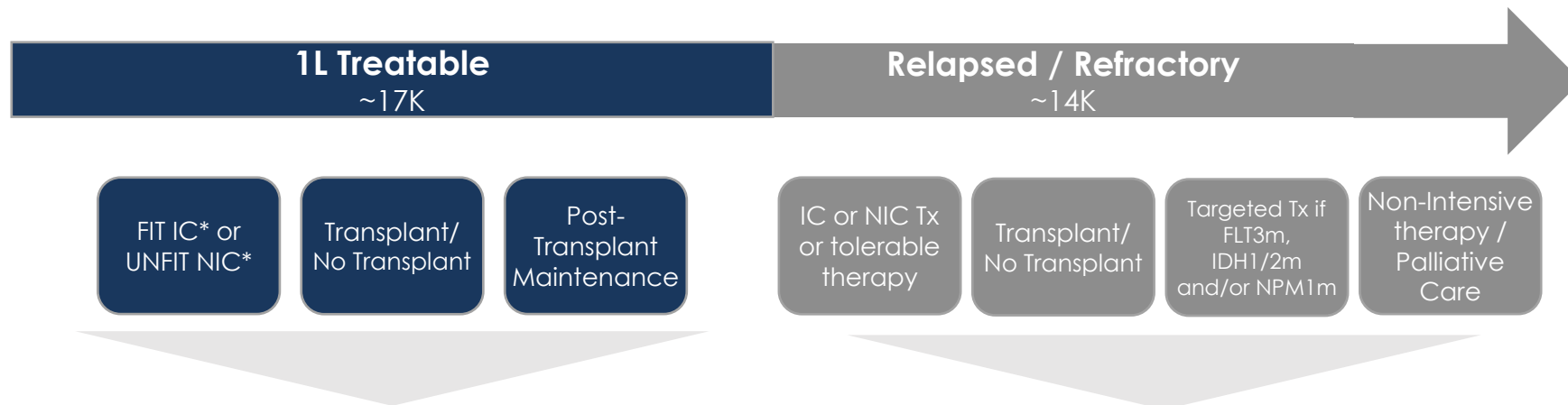
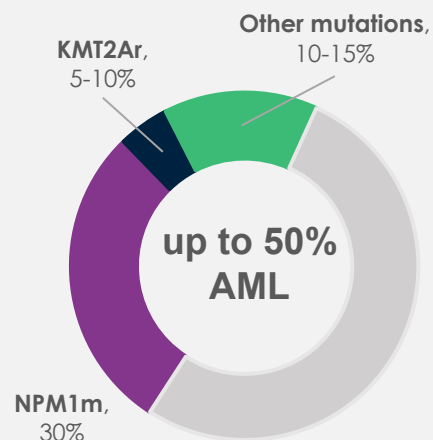


IMiD combinations increased 5yr OS from 35% to > 65%; class generated ~\$15B in revenues at peak



We are 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



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 Offers a Multi-Billion-Dollar Opportunity in AML and Beyond

Potential to Transform Outcomes Across the Continuum of Care

Relapsed / Refractory

- Initial approval represents **30% of potential patients**
- KOMET-001 registration-directed study for FDA full approval

Frontline / Maintenance

- Significant opportunity in 1L AML and Maintenance
- **Potential to drive > 50% revenue**
- Safety, tolerability and clinical activity anticipated to be ideal for combinations with SOC and with maintenance indication

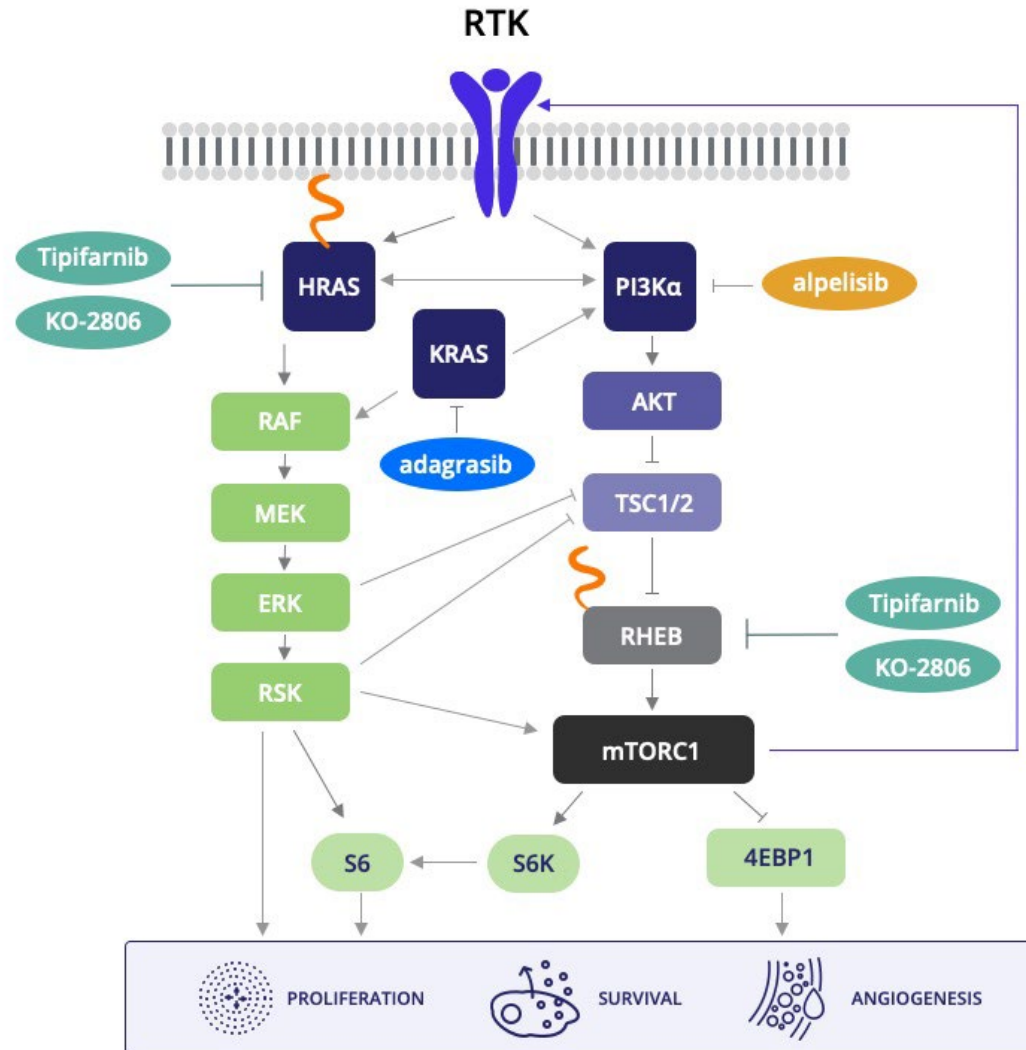
Other Indications

- Compelling **additional opportunities beyond AML** offer multi-billion-dollar potential
- Early translational data supports potential in **solid tumor and non-oncology indications**

TIPIFARNIB: FARNESYL TRANSFERASE INHIBITOR (FTI)



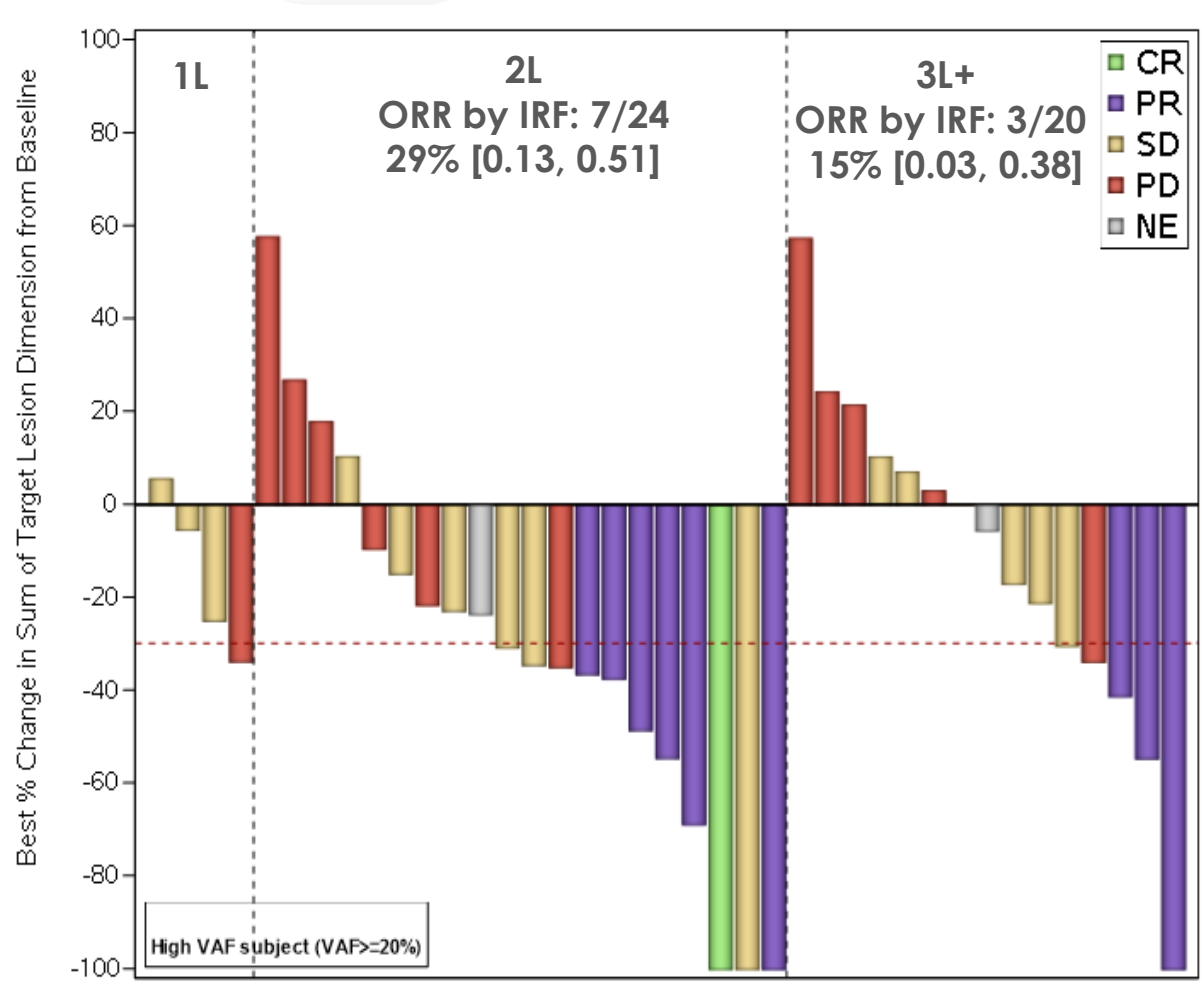
Therapeutic Applications of Farnesyl Transferase Inhibitors



- 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



Tipifarnib Shows Clinical Benefit in HRAS mutant HNSCC



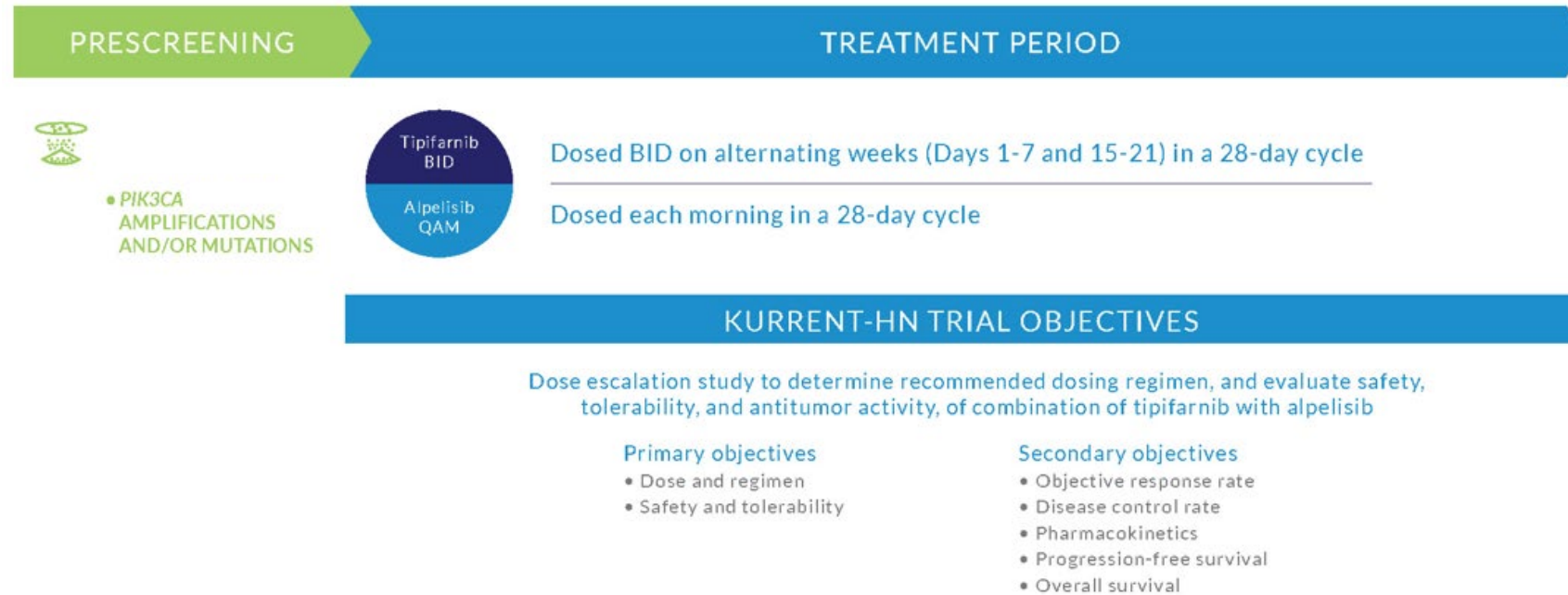
6/10 responders had BOR of PD in the last prior line with IO-based therapies
PFS in these ranged from 1-5 months vs. 6 –27 months on tipifarnib

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.

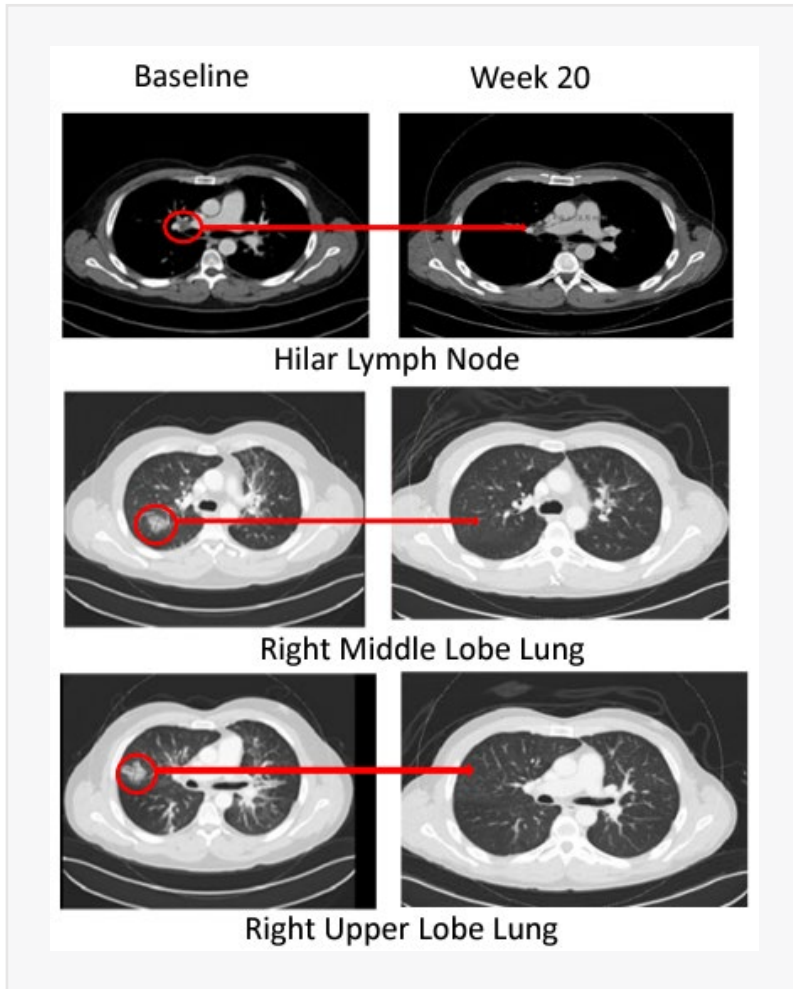
KURRENT-HN: PHASE 1/2 Combination Trial of Tipifarnib and Alpelisib in Patients with HNSCC



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

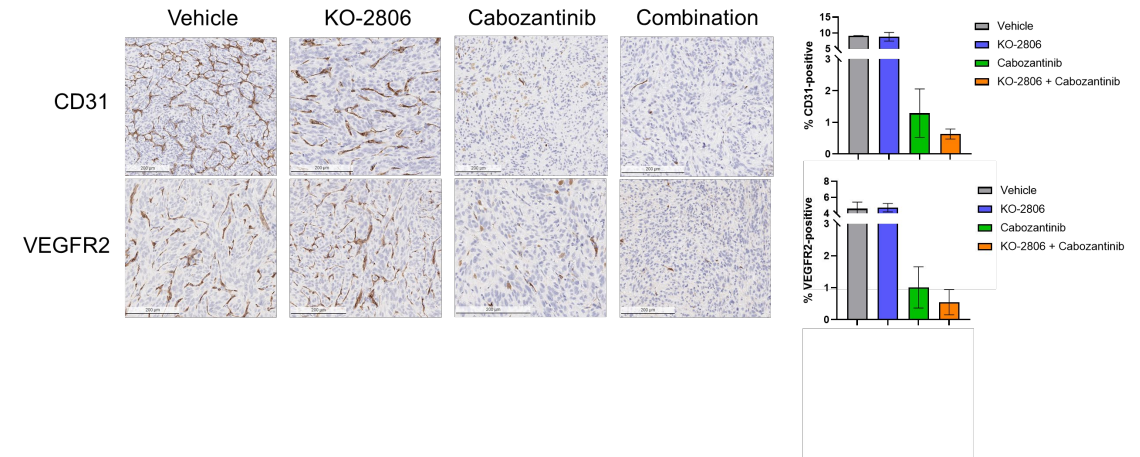
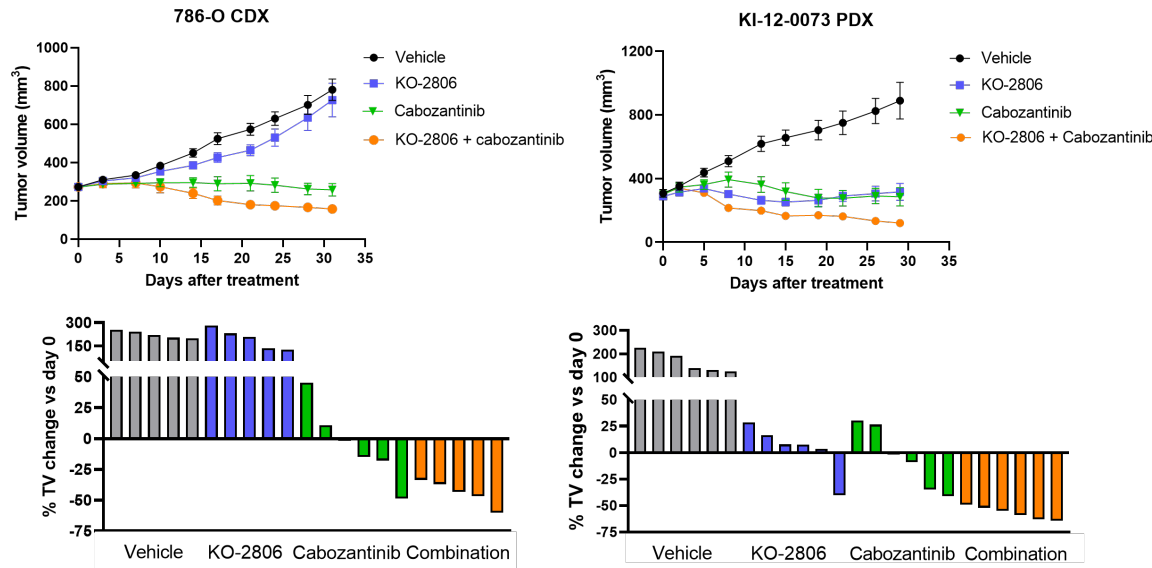
KO-2806: NEXT- GENERATION FTI

Combination of KO-2806 and Cabozantinib Demonstrates Synergistic Activity in ccRCC CDX & PDX Models



KO-2806 potentiates antitumor activity of cabozantinib in ccRCC models

KO-2806 enhances the anti-angiogenic activity of cabozantinib *in vivo*



IHC of 786-O tumors and quantitation

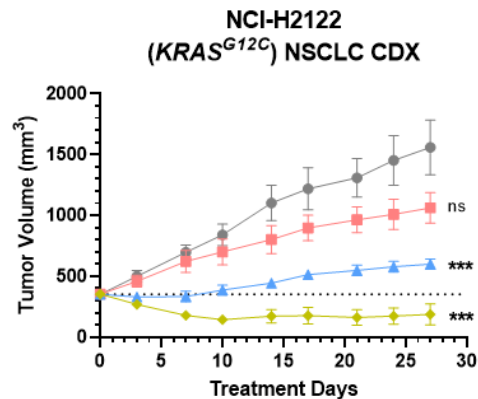
- KO-2806 enhances the anti-angiogenic activity of cabozantinib *in vivo*, as observed by decreased expression of vascular markers in 786-O tumors
- Studies are ongoing to further define the basis of the synergy of the combination

Combination of KO-2806 to Enhance Antitumor Efficacy of KRAS^{G12C} Inhibitor in NSCLC



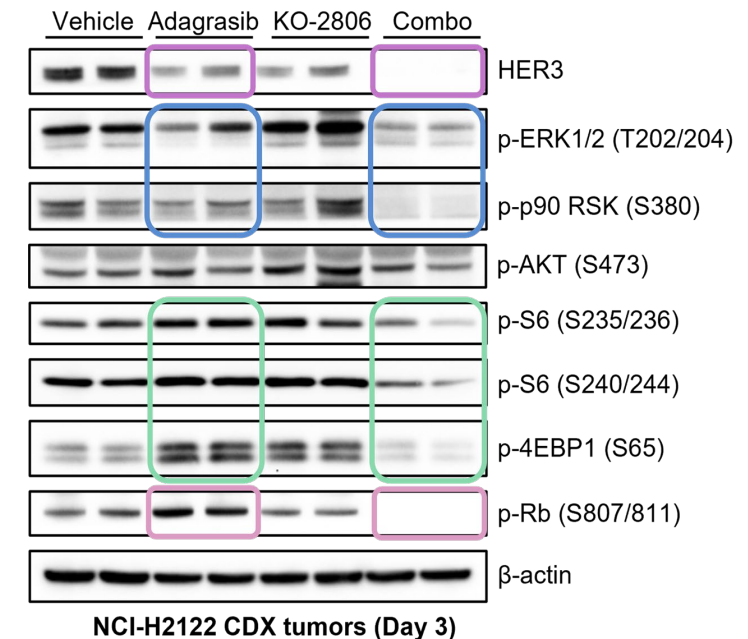
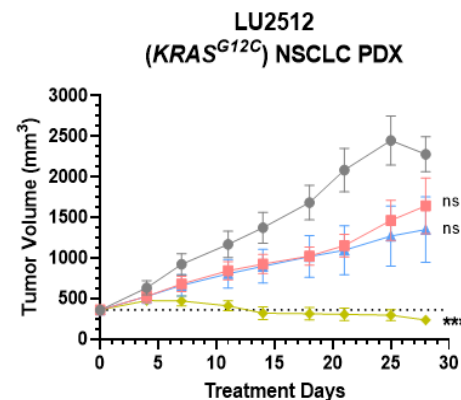
Combination of KO-2806 with a KRAS^{G12C} inhibitor causes tumor regressions in patient-derived and cell-derived NSCLC xenografts

Combination of KO-2806 with a KRAS^{G12C} inhibitor suppresses mTOR and MAPK signaling and decreases proliferation



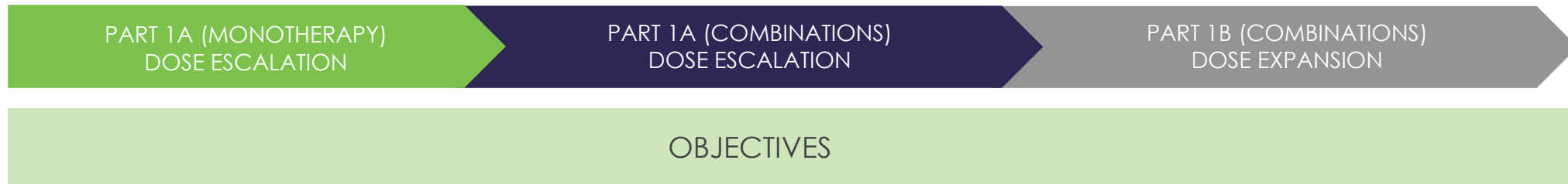
● Vehicle
■ KO-2806
▲ Adagrasib
◆ KO-2806 + Adagrasib

*** = p < 0.001



- Combination of KO-2806 with adagrasib enhances the depth and duration of response compared with single-agent KRAS^{G12C} inhibitor treatment

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



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

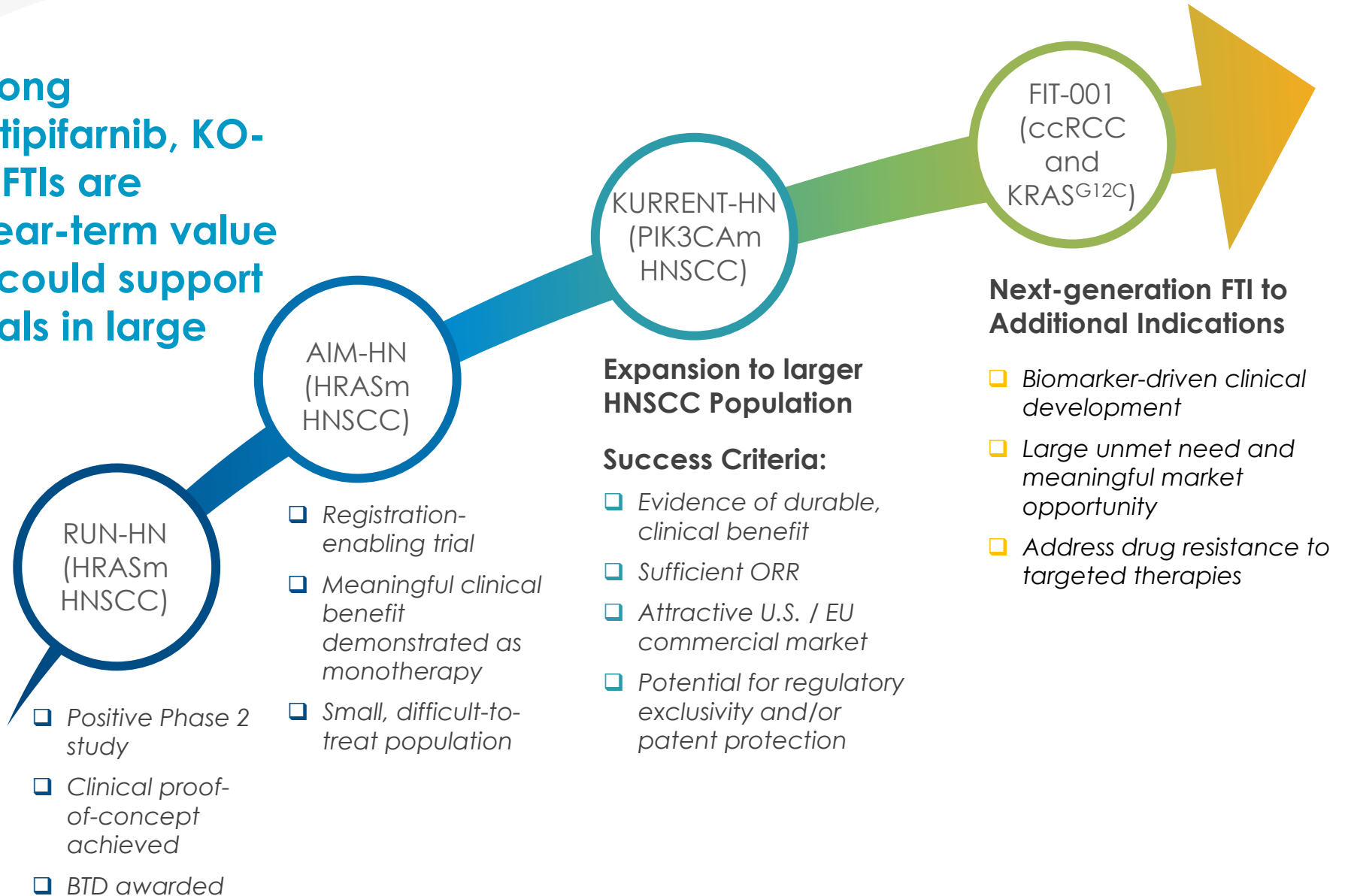
- Evaluate the safety and tolerability of KO-2806 (dose expansion)
- Evaluate the preliminary antitumor activity of KO-2806 (dose escalation and dose expansion)
- Characterize the PK of KO-2806 when administered as monotherapy, and the PK of KO-2806 and the combination agents when administered in combination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

Ongoing enrollment in FIT-001 Phase 1 dose-escalation trial of KO-2806



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	Dose first patients in KMT2A-r acute lymphoblastic leukemia (ALL)	✓
	Dose first patient in KOMET-008 combination trial	✓
	Initiate the post-transplant maintenance program	Q1 2024
	Complete enrollment of 85 patients in KOMET-001 registration-directed trial	Mid-2024
	Initiate expansion cohort in non-NPM1-m/non-KMT2A-r AML	Mid-2024
	Determine RP2D in combination with ven/aza and initiate dose validation/expansion in 1L AML	Mid-2024
	Determine RP2D in combination with 7+3	Mid-2024
TIIFARNIB Farnesyl Transferase Inhibitor (FTI)	Positive results from AIM-HN monotherapy trial in HRAS-mutant HNSCC	✓
	Determine OBAD in combination with alpelisib in PIK3CA-dependent HNSCC	End of 2024
KO-2806 Next-Generation FTI	Dose first patient in FIT-001 dose-escalation trial as monotherapy	✓
	Dose first patients in FIT-001 trial in combination with cabozantinib in ccRCC	Mid-2024
	Dose first patients in FIT-001 trial in combination with adagrasib in KRAS ^{G12C} -mutated NSCLC	Mid-2024
Financial Highlights Nasdaq: KURA	\$570M in pro forma cash* provides runway into 2027	
	Shares outstanding as of Feb 27, 2024: 76.1M basic; 25.3M options, RSUs, PSUs, warrants & pre-funded warrants	

OBAD = optimal biologically active dose; RP2D = recommended Phase 2 dose

* Includes \$424M in cash, cash equivalents and short-term investments as of 12/31/23 and estimated proceeds net of offering expenses of approximately \$146M from private placement closed on January 26, 2024

DEVELOPING PRECISION MEDICINES FOR THE TREATMENT OF CANCER

Corporate Presentation – February 2024