DEVELOPING PRECISION MEDICINES FOR THE TREATMENT OF CANCER

Corporate Presentation – January 2023

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; the COVID-19 pandemic may disrupt our business and that of the third parties on which we depend, including delaying or otherwise disrupting our clinical trials and preclinical studies, manufacturing and supply chain, or impairing employee productivity; 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.

All forward-looking statements contained in this presentation speak only as of the date on which they were made. Other risks and uncertainties affecting us are described more fully in our filings with the Securities and Exchange Commission. We undertake no obligation to update such statements to reflect events that occur or circumstances that exist after the date on which they were made.

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)** • Potential to address 35% or more of acute leukemias • Encouraging safety, tolerability and clinical activity observed in relapsed/refractory AML patients • 30% CR rate among 20 patients with NPM1 mutations at 600 mg RP2D • Phase 2 registration-directed trial in NPM1-mutant AML expected to begin in Q1 2023 • Combination studies with standards of care expected to begin in 1H 2023
	Farnesyl Transferase Inhibitor Programs (tipifarnib & KO-2806) • Durable responses as a monotherapy in recurrent/metastatic HRAS-mutant HNSCC patients • Proof of mechanism demonstrated in combination with alpelisib in PIK3CA-dependent HNSCC • Potential to prevent emergence of resistance to osimertinib in EGFR-mutant NSCLC • IND for KO-2806, next-generation FTI, on track for Q1 2023
Strong Financials	• $438 million in Cash as of December 31, 2022* • $25 million equity investment from Bristol Myers Squibb and $125 million term loan facility, if fully drawn, extend cash runway into 2026

* Unaudited, preliminary cash, cash equivalents and short-term investments as of 12/31/22

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DRUG CANDIDATE PIPELINE

PROGRAM	CLINICAL TRIAL	STUDY STARTUP / PHASE 1 / REGISTRATION DIRECTED
ZIFTOMENIB Menin Inhibitor	**KOMET-001** Monotherapy	NPM1-mutant acute myeloid leukemia (AML) Non-NPM1-m/KMT2A-r AML KMT2A-rearranged ALL
	KOMET-007/008 Combination with standards of care	NPM1-mutant AML KMT2A-rearranged AML
TIPIFARNIB Farnesyl Transferase Inhibitor (FTI)	**AIM-HN** Monotherapy	HRAS-mutant head and neck squamous cell carcinoma (HNSCC)*
	KURRENT-HN Combination with alpelisib	PIK3CA-dependent HNSCC HRAS-dependent HNSCC
	KURRENT-LUNG Combination with osimertinib	EGFR-mutant NSCLC
KO-2806 Next-Generation FTI	Combination with targeted therapies	Solid Tumors

* Trial closed to further enrollment

KURA ONCOLOGY

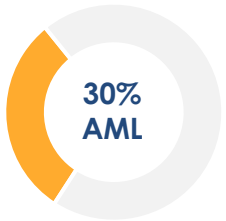
ZIFTOMENIB: MENIN-KMT2A/MLL INHIBITOR IN ACUTE LEUKEMIAS

NPM1-MUTANT AND KMT2A-REARRANGED AML REPRESENT AREAS OF SIGNIFICANT UNMET NEED

No FDA-Approved Targeted Therapies Exist Today

NPM1-mutant AML

~ **6,000** new cases annually in the U.S.[1]



Adult patients with NPM1-mutant AML and select co-mutations and/or relapsed/refractory disease are associated with poor prognosis[2]

5-year Overall Survival ~ 50%[3]

Median Overall Survival in patients with R/R NPM1 AML is ~ 6.1 mo.[4]

KMT2A-rearranged AML

~ **1,000-2,000** new cases annually in U.S.[1]

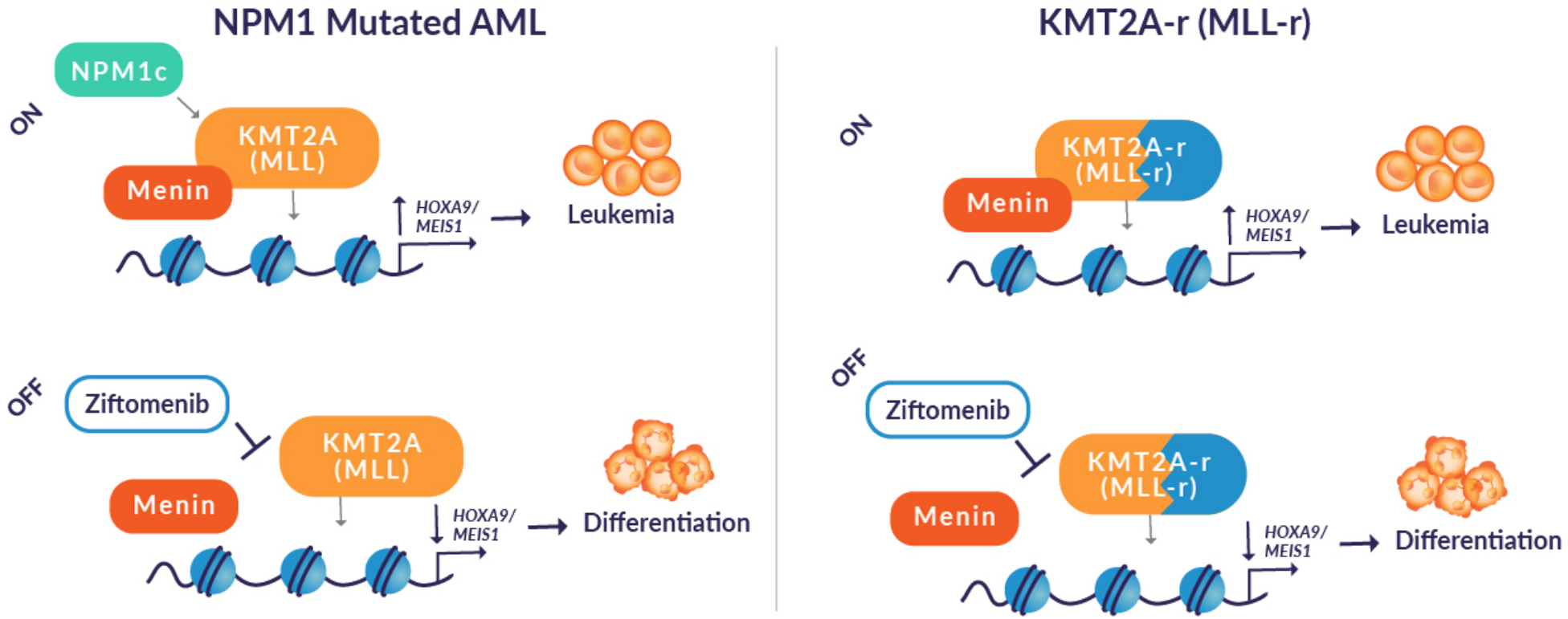
Adult patients with KMT2A-rearranged AML have poor prognosis with high rates of resistance and relapse following current standard of care [5, 6]

5-year Overall Survival < 20%[5]

Median Overall Survival in patients with R/R KMT2A-r AML is 6 mo. following 2L treatment and 2.4 mo. following 3L treatment[5]

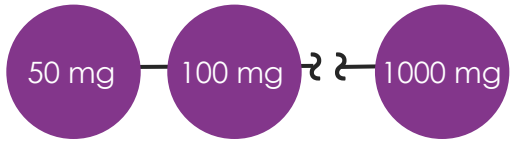
[1] SEER statistics for AML in the US, accessed April 2020.
[2] Döhner *et al*. *Blood*. 2017 Jan 26;129(4):424-447.
[3] Angenendt L, *et al*. *J Clin Oncol*. 2019;37(29):2632-2642.
[4] Venugopal S, et al. ASH Abstract 2287, 2021.
[5] Issa GC, *et al*. *Blood Cancer J*. 2021;11(9):162.
[6] Vetro C, *et al*. *Cancer Genet*. 2020;240:15-22.

ZIFTOMENIB IS A POTENT AND SELECTIVE ORAL INHIBITOR OF THE MENIN-KMT2A/MLL COMPLEX



Kühn MW, *et al*. *Cancer Discov*. 2016;6(10):1166-1181
Thorsteinsdottir U, *et al*. *Mol Cell Biol*. 2001;21(1):224-234
Patel SS, *et al*. *Curr Hematol Malig Rep*. 2020;15(4):350-359
Brunetti L, *et al*. *Cancer Cell*. 2018;34(3):499-512

KOMET-001 PHASE 1 CLINICAL TRIAL OF ZIFTOMENIB IN PATIENTS WITH RELAPSED OR REFRACTORY (R/R) AML

PHASE 1A DOSE ESCALATION	PHASE 1B VALIDATION COHORTS	PHASE 1B EXPANSION
	 Cohort 1: 200 mg  Cohort 2: 600 mg	 Expansion of 600 mg
OBJECTIVES		
Dose escalation in all-comer patients to establish maximum tolerated dose and/or RP2D • Safety and tolerability • Pharmacokinetics • Early evidence of antitumor activity	Enroll 2 genetically enriched cohorts to determine the optimal RP2D in line with FDA's Project Optimus • Safety and tolerability • Pharmacokinetics • Antitumor efficacy	Continue enrollment of Phase 1b validation cohort(s) meeting go/no-go criteria • Safety and tolerability • Pharmacokinetics • Antitumor efficacy

ZIFTOMENIB DEMONSTRATES ENCOURAGING SAFETY PROFILE AND TOLERABILITY IN PHASE 1B

≥Gr 3 TEAEs Occurring in >10% Participants
(Regardless of Causal Assessment)

	200 mg	600 mg
NPM1-m	(N = 4)	(N = 20)
	0	0
KMT2A-r	(N = 13)	(N = 16)
Differentiation Syndrome	4 (30.8)	4 (25.0)
Febrile Neutropenia	0	2 (12.5)

Erba *et al*. ASH 2022 #64 (preliminary data as of October 24, 2022)

CHARACTERIZATION OF DIFFERENTIATION SYNDROME WITH ZIFTOMENIB

Any Grade and ≥ G3 DS in Phase 1a/b population

	200 mg N = 17, n (%)	600 mg N = 36, n (%)
NPM1-m (all grades)	0/4 (0)	4/20 (20.0)
≥ Gr3	0/4 (0)	1/20 (5.0)
KMT2A-r (all grades)	5/13 (38.5)	6/16 (37.5)
≥ Gr3	4/13 (30.8)	4/16 (25.0)
Patients with DS event at 600 mg ORR: 3/4 (75%) for *NPM1*-m; 1/6 (16.7%) for *KMT2A*-r		

Extramedullary involvement has a significantly higher frequency in patients with KMT2A(MLL) rearrangements vs. all others, including NPM1[1]

Erba *et al*. ASH 2022 #64 (preliminary data as of October 24, 2022)

[1] *Fianchi et al. Mediterr J Hematol Infect Dis. 2021; 13(1): e2021030; DOI: https://doi.org/10.4084/MJHID.2021.030*

ZIFTOMENIB DEMONSTRATES ENCOURAGING ANTILEUKEMIC ACTIVITY AT 600 MG

Best Overall Response	200 mg	600 mg
NPM1-m Phase 1a + 1b	**(n=6)**	**(n=20)**
CR	**1 (16.7)**	**6 (30.0)**
CR/CRh	1 (16.7)	6 (30.0)
CRc	1 (16.7)	7 (35.0)
MRD negativity	1 (100.0)	3 (42.9)[1]
ORR	2 (33.3)	8 (40.0)
KMT2A-r Phase 1a + 1b	**(n=14)**	**(n=18)**
CR/CRh	0	1 (5.6)
CRc	0	2 (11.1)
MRD negativity	0	2 (100.0)
ORR	0	3 (16.7)

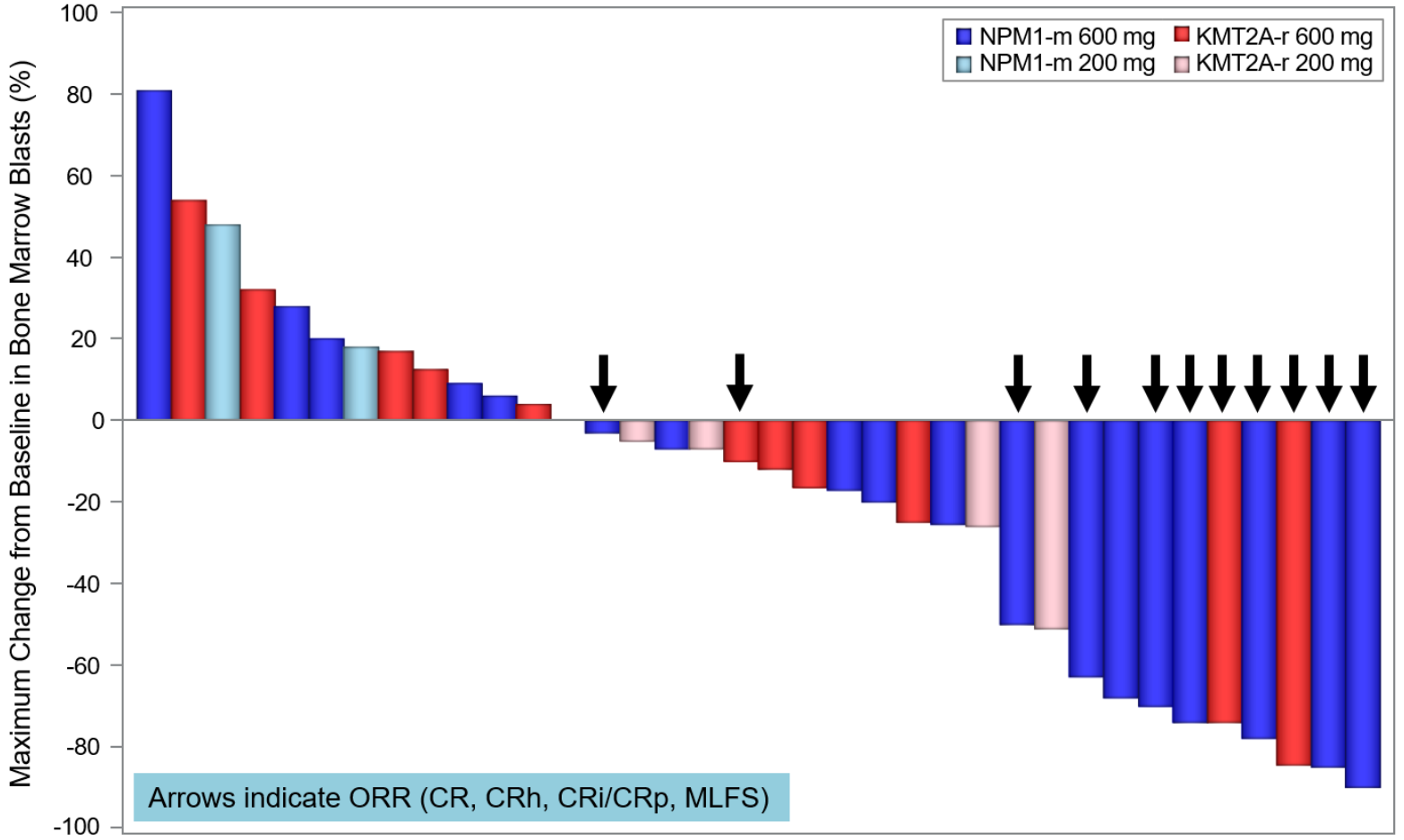
- 2 pts had concurrent *IDH1/2*
- 2 pts had both *IDH1/2* and *FLT3-ITD/TKD*

Of IDH1/2 co-mutants (7), 57% experienced a CR

[1] MRD was assessed for 5/7 CRc patients; 3 of those 5 patients (60%) tested were MRD negative
CRc includes CR, CRh, CRi, CRp
ORR includes CR, CRh, CRi, CRp, MLFS

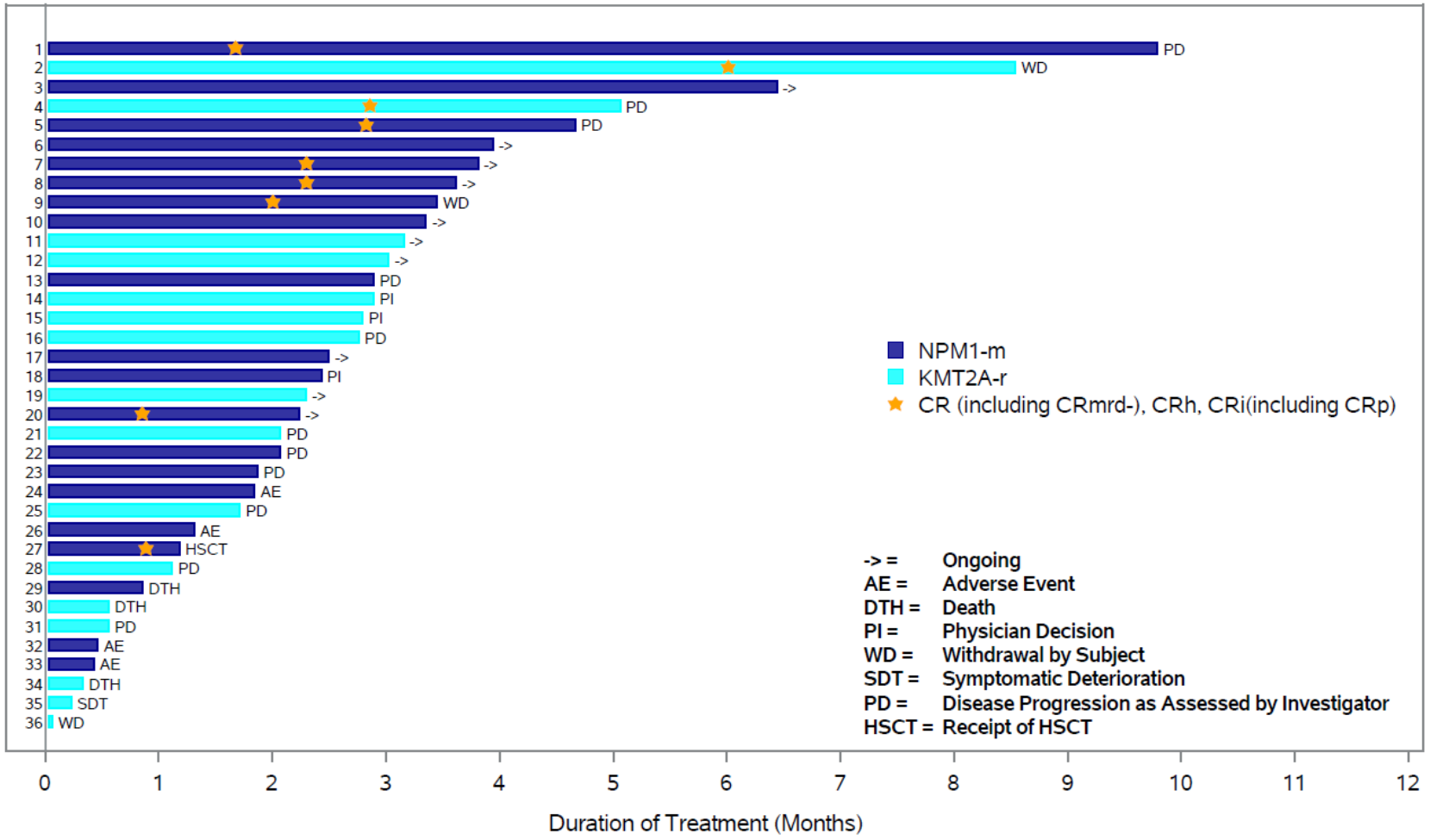
Erba *et al*. ASH 2022 #64 (preliminary data as of October 24, 2022)

DECREASING BONE MARROW BLAST COUNTS CONSISTENTLY REPORTED



Erba *et al*. ASH 2022 #64 (preliminary data as of October 24, 2022)

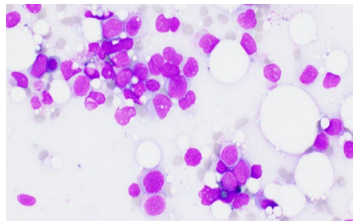
CLINICAL ACTIVITY OF ZIFTOMENIB OPTIMAL AT 600 MG ORAL, DAILY DOSING



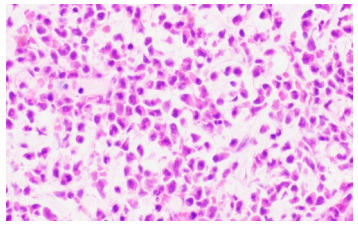
Preliminary data as of October 24, 2022

ZIFTOMENIB INDUCES RAPID AND EXTENSIVE DIFFERENTIATION OF NPM1-MUTANT LEUKEMIA

61 yo male with NPM1-m, FLT3-ITD, and IDH2 AML *Baseline bone marrow blasts: 75%*	
Prior therapies	7+3, Midostaurin, HiDAC, gilteritinib
Initiated ziftomenib at 600 mg	
DS during C1	Bone pain, ↓BP WBC ↑58K
Response	• MLFS after Cycle 1 • CR after Cycle 3



Baseline Bone Marrow
Cellular BM (40%) with 75% blasts consistent with relapsed AML



Cycle 1 Day 28 ziftomenib
Hypercellular BM (>95%) with striking granulocytic hyperplasia and <1% blasts

Erba *et al*. ASH 2022 #64 (preliminary data as of October 24, 2022)

EVIDENCE OF CLINICAL BENEFIT IN PATIENTS WITH NPM1-MUTANT AML

44 yo female with NPM1-m, DNMT3A and IKZF1 AML Baseline bone marrow blasts: 14%	
Prior therapies	Cytarabine + anthracycline NOS; mitoxantrone, etoposide + cytarabine; HiDAC+ fludarabine + melphalan; **1st SCT** + cyclophosphamide; lenalidomide + bortezomib; decitabine + venetoclax + gilteritinib; ASP1235; busulfan + fludarabine; **2nd SCT** + methotrexate
Initiated ziftomenib at 200 mg	
No DS	Experienced TRAEs of Gr4 lipase increased and Gr3 pancreatitis at C2D28; Gr3 pulmonary embolus during C17
Response	• CRmrd- after Cycle 1 • CRmrd- maintained and currently at Cycle 31

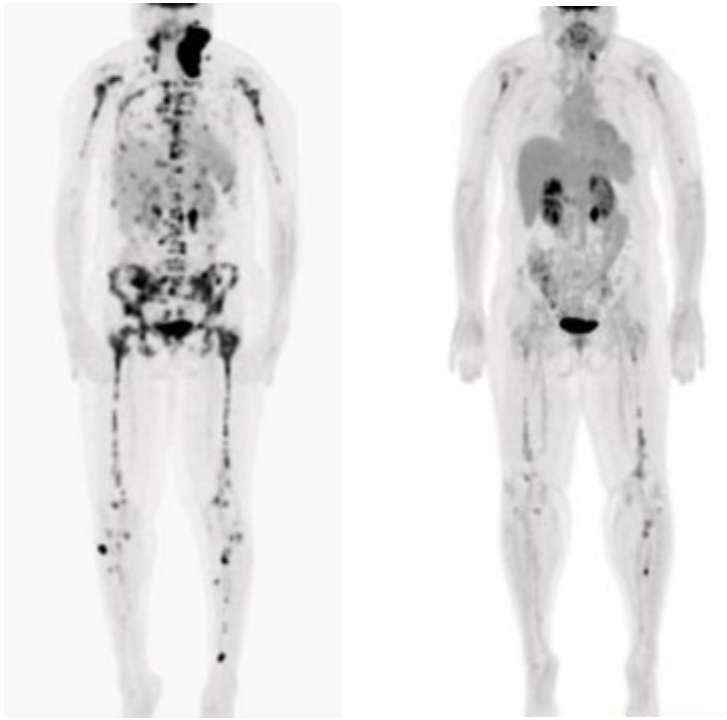
22 yo male with *NPM1*-m AML Baseline bone marrow blasts: 90%	
Prior therapies	Cytarabine + idarubicin (7+3)
Initiated ziftomenib at 600 mg	
DS during Cycle 1 (Gr2; non-serious)	Non-cardiac chest and bone pain; ↓ fibrinogen (89 from 456 at baseline)
Response	• CRmrd- after Cycle 1 • Transplant scheduled

Preliminary data as of October 24, 2022

EVIDENCE OF CLINICAL BENEFIT: EXAMPLE OF A KMT2A-REARRANGED NON-RESPONDING PATIENT

47 yo female with KMT2A-r, TERT and BRAF AML Baseline bone marrow blasts: 52%	
Prior therapies	ddAC + paclitaxel, CPX-35, SCT, Aza, FLAG Ida-ven, DLI, RT - gums
Initiated ziftomenib at 200 mg	
DS during C1	Muscle and EMD pain, ↑temp, ↓BP, WBC ↑ 5.2
Response	• Bone marrow blasts 2% end of Cycle 2 • Best overall response for the patient of SD due to residual extramedullary disease

Baseline **After 2 cycles**



Erba *et al*. ASH 2022 #64 (preliminary data as of October 24, 2022)

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 30% CR rate (n=20)
- High levels of ziftomenib tissue penetration likely drive clearance of extramedullary disease

Monotherapy data supportive of combination strategies

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

Erba *et al*. ASH 2022 #64 (preliminary data as of October 24, 2022)

ZIFTOMENIB CLINICAL DEVELOPMENT PATH

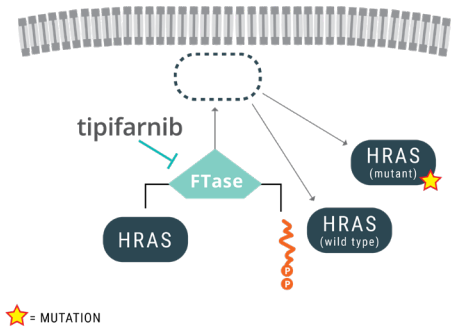
DEVELOPMENT APPROACH	STUDY STARTUP / PHASE 1 / REGISTRATION DIRECTED	TRIAL
MONOTHERAPY (Relapsed/refractory)	NPM1-mutant acute myeloid leukemia (AML) Non-NPM1-m/KMT2A-r AML KMT2A-rearranged ALL	komet ACUTE LEUKEMIAS KURA KO-MEN-001
COMBINATION WITH VENETOCLAX + AZACYTIDINE (Relapsed/refractory, frontline)	NPM1-mutant AML KMT2A-rearranged AML	komet ACUTE LEUKEMIAS KURA KO-MEN-007
COMBINATION WITH CYTARABINE + DAUNORUBICIN (7+3) (Frontline)	NPM1-mutant AML KMT2A-rearranged AML	
COMBINATION WITH GILTERITINIB (Relapsed/refractory)	NPM1-mutant AML	komet ACUTE LEUKEMIAS KURA KO-MEN-008
COMBINATION WITH FLAG-IDA (Relapsed/refractory)	NPM1-mutant AML KMT2A-rearranged AML	
COMBINATION WITH IDAC/LDAC (Relapsed/refractory)	NPM1-mutant AML KMT2A-rearranged AML	
POST-TRANSPLANT MAINTENANCE	NPM1-mutant AML KMT2A-rearranged AML	**Investigator-sponsored studies**
COMBINATION WITH FLA (Relapsed/refractory)	Pediatric AML & ALL	
COMBINATION WITH BV-DAM (Frontline)	Pediatric ALL	

KURA ONCOLOGY

FARNESYL TRANSFERASE INHIBITOR PROGRAMS

EVOLUTION IN THE THERAPEUTIC APPLICATIONS OF FARNESYL TRANSFERASE INHIBITORS

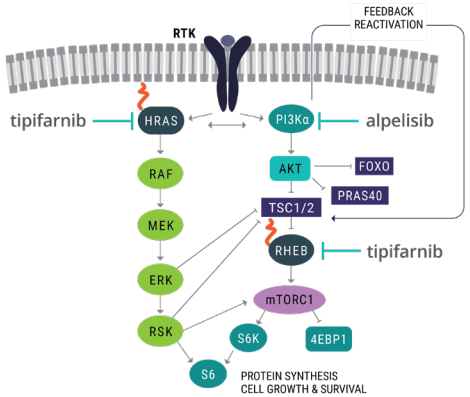
Direct Inhibition of Oncogenic Proteins



- Monotherapy activity in mutant tumors

AIM-HN
KURA KO-TIP-007

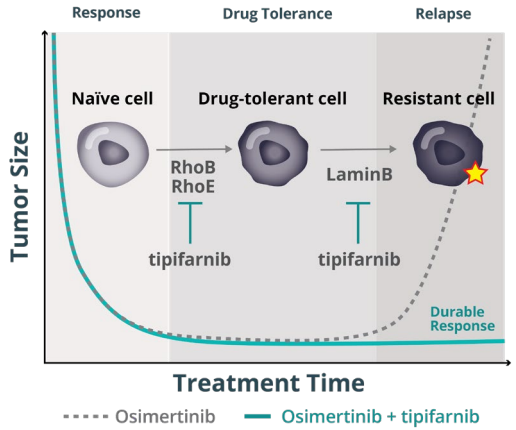
Overcoming Drug Resistance



- Overcome drug resistance to PI3Kα inhibitor in HNSCC

KURRENT-HN
KURA KO-TIP-013

Preventing Emergence of Resistance

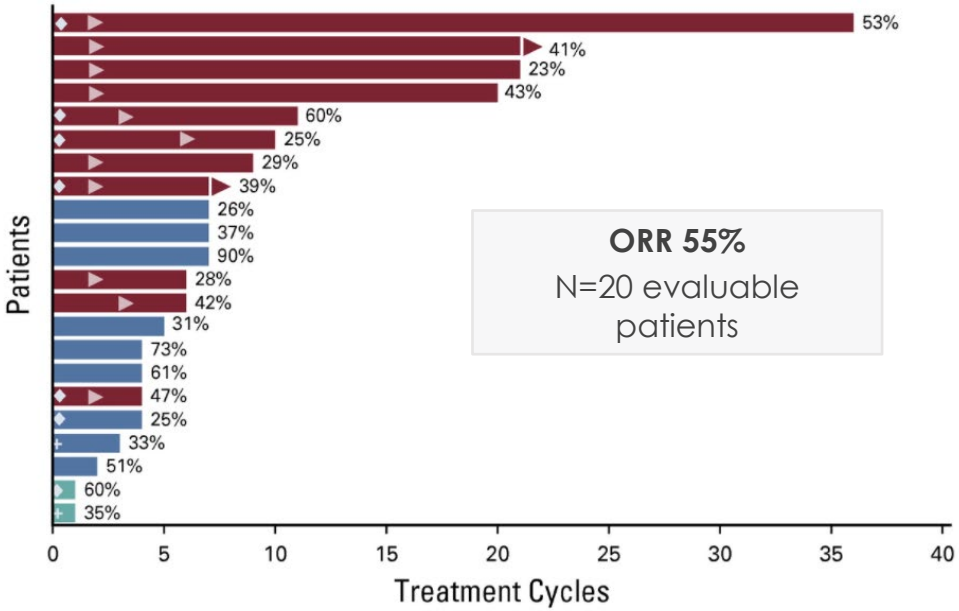


- Prevent emergence of resistance to EGFR inhibitor in NSCLC

KURRENT-LUNG
KURA KO-TIP-015

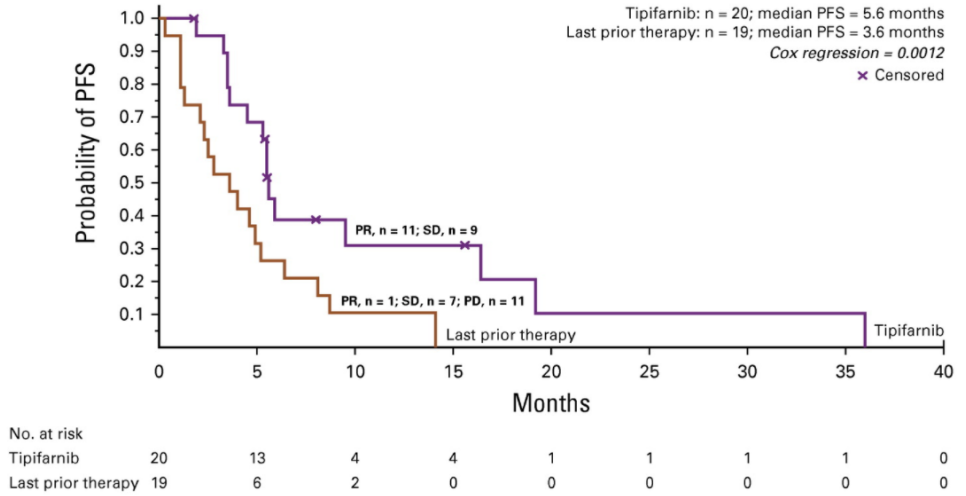
TIPIFARNIB DEMONSTRATES DURABLE ANTI-TUMOR ACTIVITY IN PATIENTS WITH RECURRENT OR METASTATIC HRAS-MUTANT HNSCC

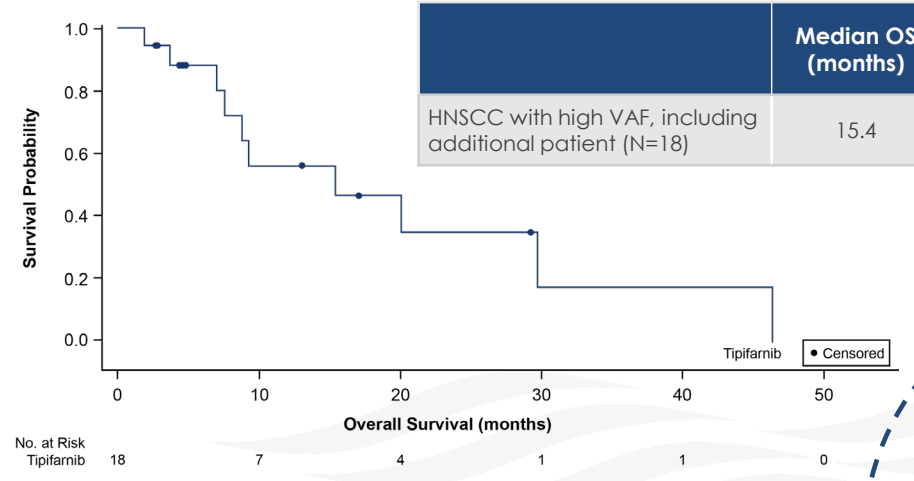
RUN-HN
KO-TIP-001



ORR 55%
N=20 evaluable patients

Red, PR; *blue*, SD; *green*, not evaluable for efficacy; *diamond*, patient initiated treatment at 600 mg twice a day; *cross*, patient withdrew consent; *arrow in bar*, start of response; *arrow*, active treatment. Numbers at the end of the bars represent VAF for each patient.





	Median OS (months)	95% CI	
		Lower	Upper
HNSCC with high VAF, including additional patient (N=18)	15.4	7.0	46.4

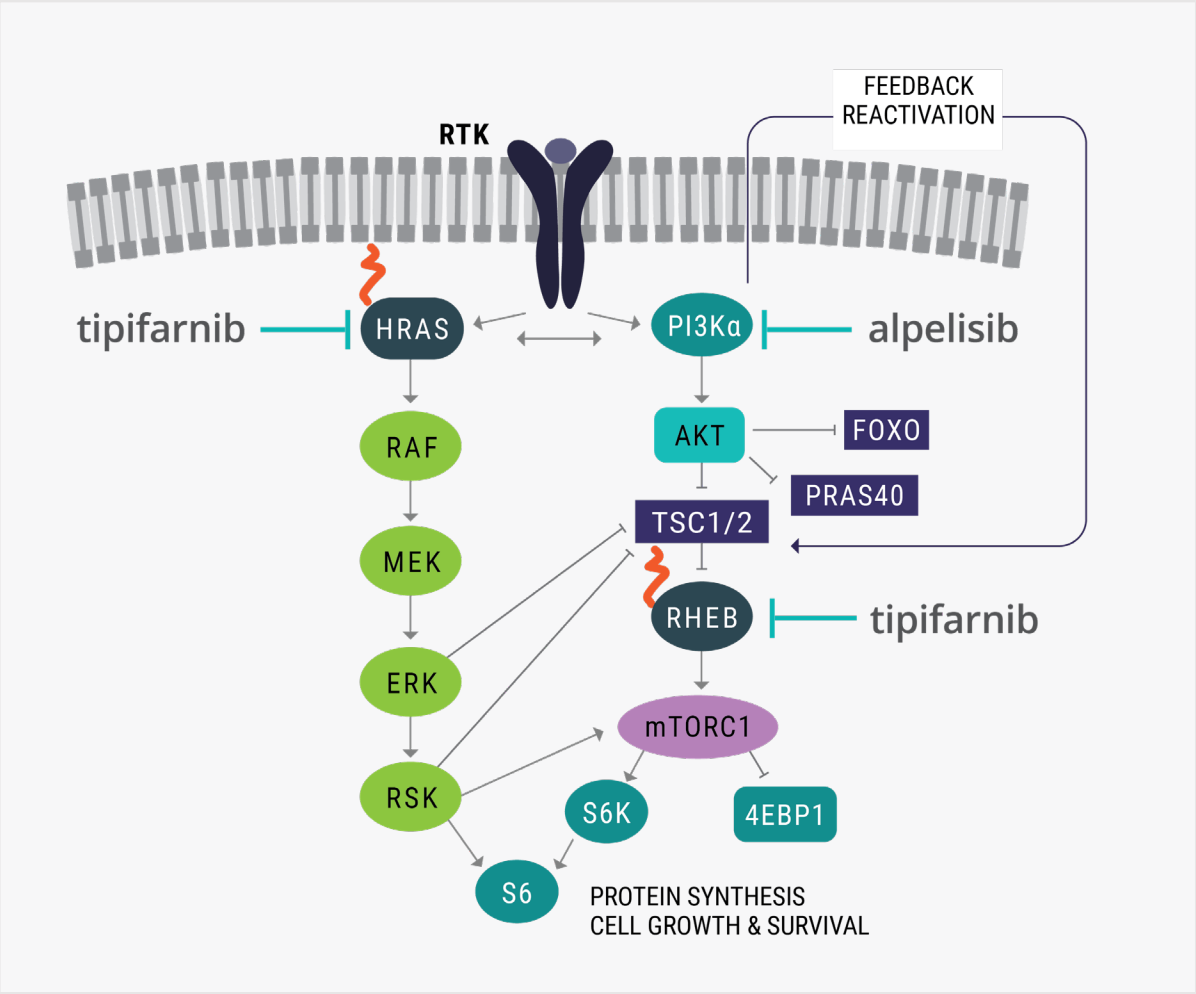
Ho, et al. *J Clin Oncol*. 2021 June 10;39(17):1856-1864. doi: 10.1200/JCO.20.02903.
Ho *et al*. ASCO 2020 #6504 (preliminary exploratory data as of 9/30/19)
Efficacy-evaluable patients with HRAS mutant variant allele frequency (VAF) ≥ 20% and serum albumin ≥ 3.5 g/dL, or HRAS VAF ≥ 35%
One patient treated off-protocol through compassionate use

AIM-HN: REGISTRATION-DIRECTED TRIAL OF TIPIFARNIB

AIM-HN
KO-TIP-007

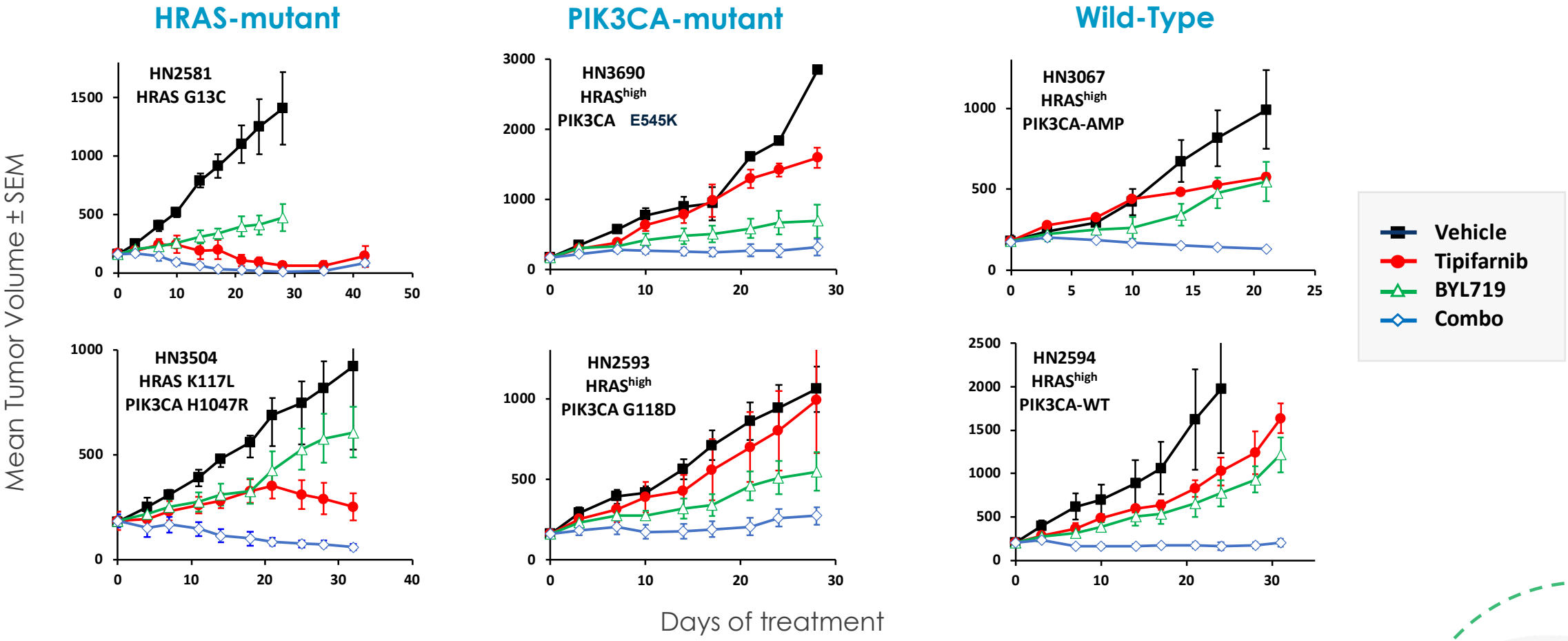
- Tipifarnib granted Breakthrough Therapy Designation for the treatment of patients with HRAS-mutant HNSCC based on data from RUN-HN study
- AIM-HN is a global, multi-center, registration-directed trial in patients with recurrent or metastatic HNSCC after one prior line of platinum therapy
- Evidence of meaningful clinical activity observed in AIM-HN; however, trial closed to further enrollment due to significant feasibility challenges
- Currently evaluating clinical data from RUN-HN and AIM-HN to inform future development of the program
- Given significant overlap between patients with HRAS overexpression and mutation, HRAS-mutant HNSCC patients in the U.S. may be eligible to enroll in ongoing KURRENT-HN study

TIPIFARNIB HAS POTENTIAL TO OVERCOME RESISTANCE TO TREATMENT WITH PI3Kα INHIBITORS IN HNSCC



- The PI3K pathway is the most frequently activated pathway in HNSCC
  - ~30% of tumors harbor *PIK3CA* mutation or amplification
- Feedback reactivation of PI3K –mTOR signaling drives innate resistance to PI3K inhibitors
  - Necessitates development of rational combination strategies
- Tipifarnib blocks hyperactivated growth factor signaling via multiple farnesylation-dependent proteins, including HRAS and RHEB

COMBINATIONS OF TIPIFARNIB AND PI3Kα INHIBITOR DEMONSTRATE ROBUST ACTIVITY IN HNSCC PDX MODELS



Malik *et al*. EORTC-NCI-AACR 2020 #159
Tipifarnib used at reduced dose to simulate potential lower doses in combination (80→60mg/kg BID)
BYL-719 used at reduced dose to simulate potential lower doses in combination (60→40mg/kg QD)

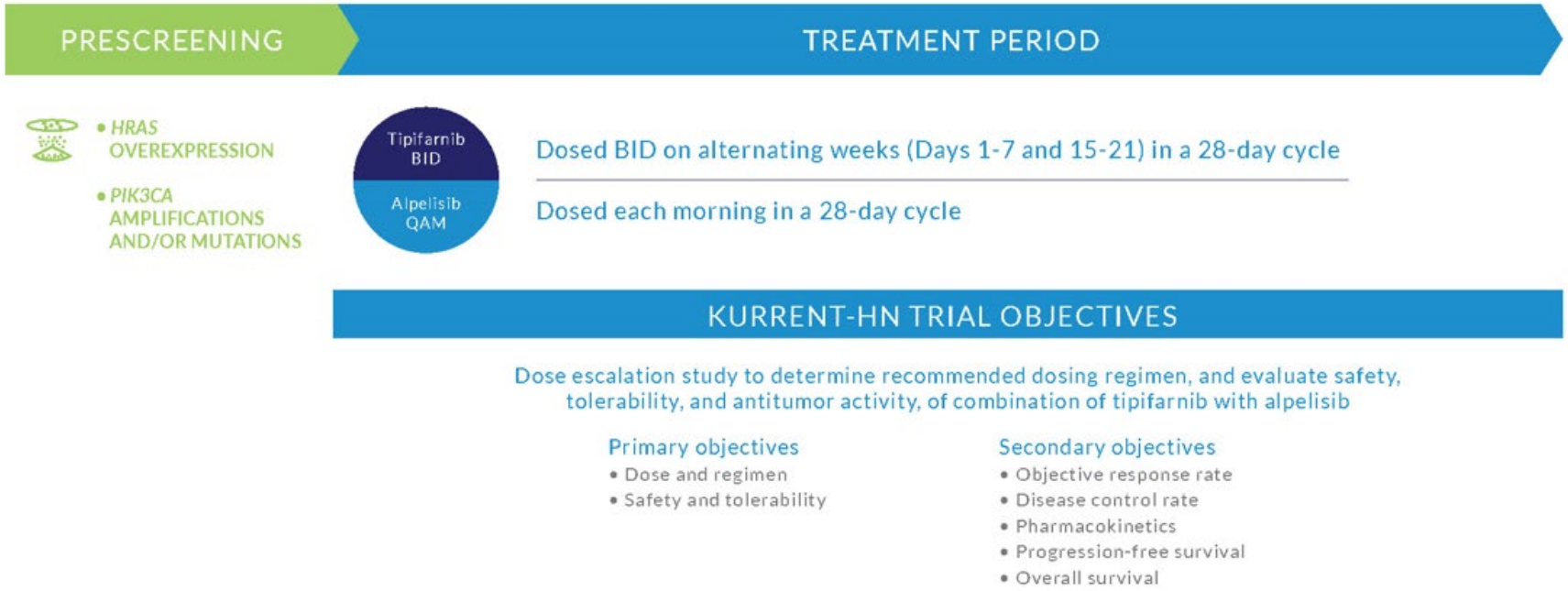
COMBINATION OF TIPIFARNIB AND PI3Kα INHIBITOR HAS SIGNIFICANT THERAPEUTIC POTENTIAL IN HNSCC

- HRAS-MAPK and PI3K-mTOR are complementary pathways in HNSCC
  - Overexpression of WT HRAS reported to induce resistance to PI3Kα inhibition
  - HRAS is reported to preferentially activate PI3K (vs. KRAS; vs. MAPK)
- HRAS mutation/overexpression and PIK3CA mutations/amplifications account for up to 45% of HNSCC[1]
  - PIK3CA mutations/amplification: 30-40% (25% estimated overlap with HRAS-overexpressing tumors)
  - HRAS overexpression: 20-30%
  - HRAS mutations: 4-8% (83% overexpress HRAS)
- Preclinical data is supportive of the combination; enhanced activity observed in both HRAS-mutant/overexpressed and PIK3CA-mutant/amplified populations of HNSCC
- Preliminary clinical data demonstrate that tipifarnib plus alpelisib can induce a durable clinical response in PIK3CA-dependent HNSCC[1]

[1] Soifer H et al, ENA 2022 PB041

References: Yan J et al (1998) *J Bio Chem* 273:24052 ; Gupta S et al (2007) *Cell* 129:957 ; Zhao L et al (2008) *Proc Natl Acad Sci* 105:2652

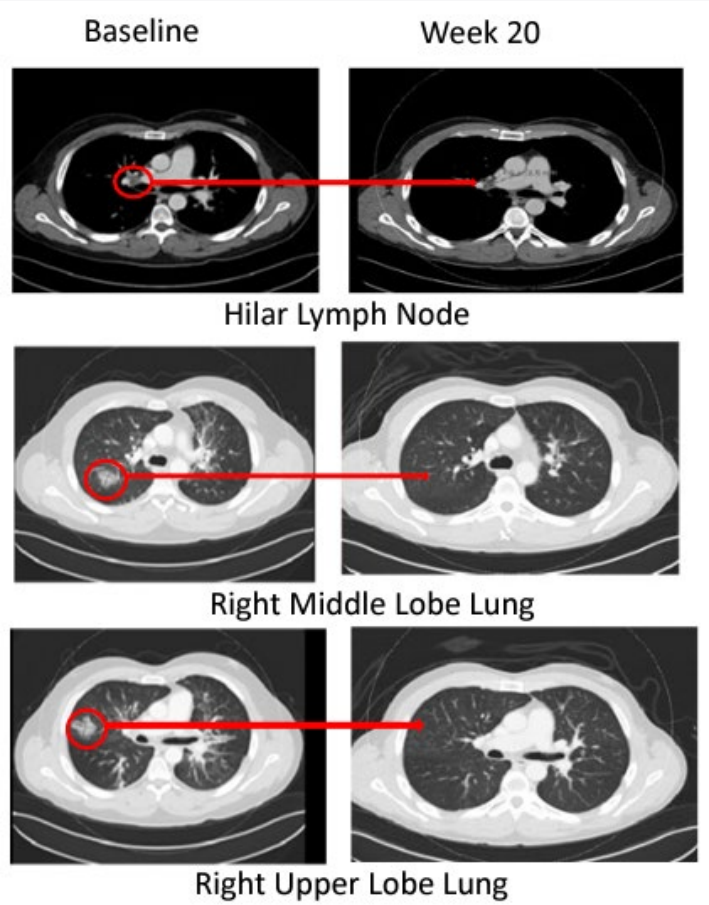
KURRENT-HN: PHASE 1/2 COMBINATION TRIAL OF TIPIFARNIB AND ALPELISIB IN PATIENTS WITH HNSCC



Phase 1 clinical trial of tipifarnib and alpelisib in patients with recurrent/metastatic *HRAS*-overexpressed and/or *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 HRAS overexpression and/or PIK3CA mutation and/or amplification
- Under the collaboration, Kura sponsors the trial and supplies tipifarnib and Novartis supplies alpelisib
- Enrolling patients in PIK3CA-dependent and HRAS-overexpression cohorts

DURABLE CLINICAL RESPONSE OBSERVED IN PATIENT WITH PIK3Cα -DEPENDENT HNSCC

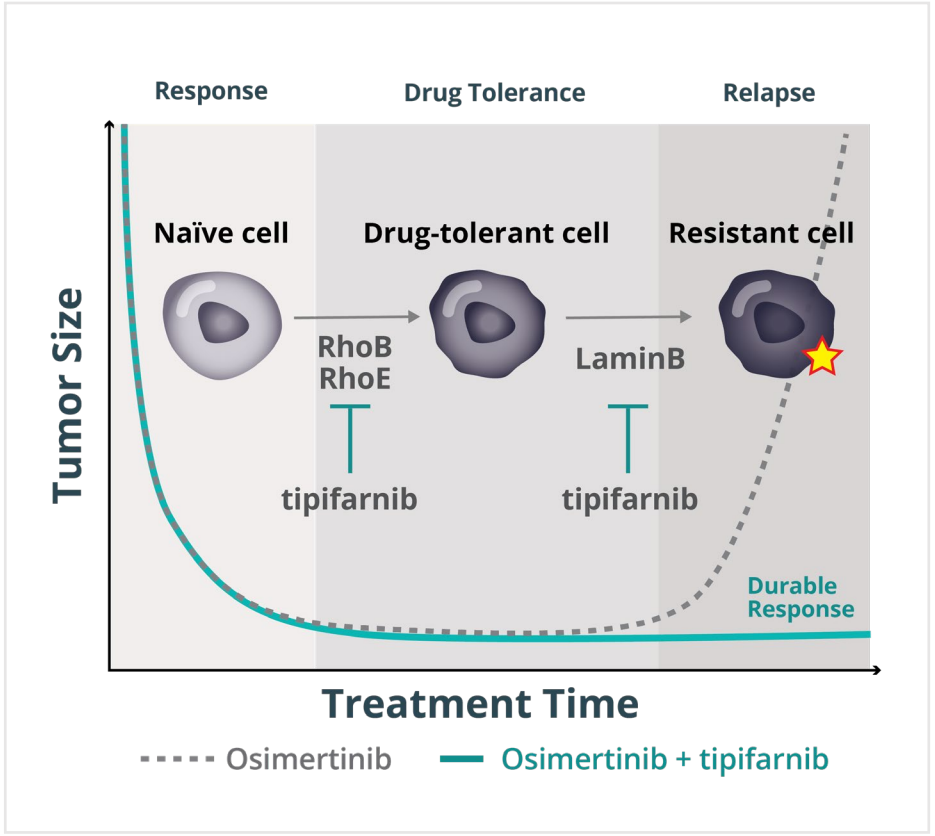


- 35yo, male, nonsmoker, HPV16 positive
- SCC of tonsil Stage III cT4N2M0; PD-L1 CPS = 60
- Prior Treatments
  - CDDP/rad for 1 mo (Nov-Dec2019), BOR UNK
  - Cemiplimab/ISA101b (Jun-Nov2021), BOR PD
- PIK3Ca R88Q mutation (44%) and HRAS OE (3+ staining in 100% of tumor cells) by IHC from May 2021 biopsy
- 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

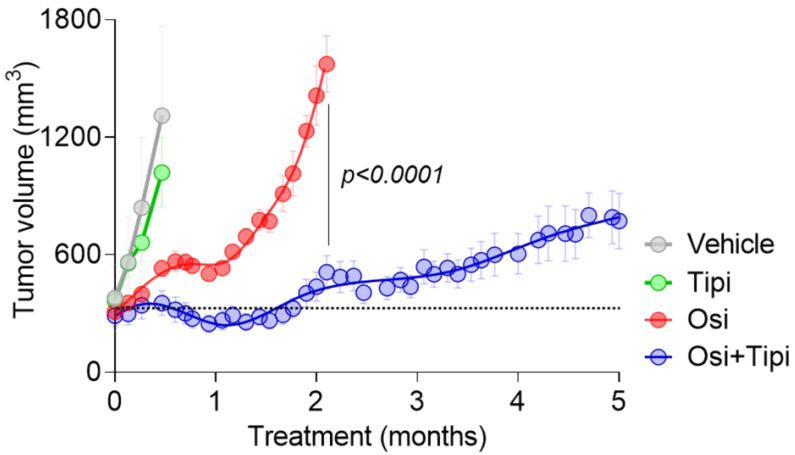
Soifer H et al, ENA 2022 PB041; Data cut as of 14Sep2022; Preliminary raw data

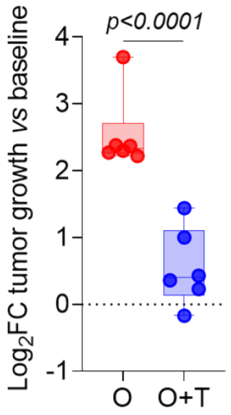
TIPIFARNIB PREVENTS EMERGENCE OF RESISTANCE TO OSIMERTINIB IN VIVO

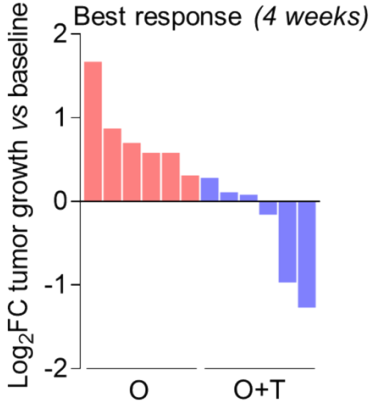
- Drug-tolerant cells (DTCs) arise within days of osimertinib exposure
- DTCs are characterized by Rho pathway activation
- RhoB, RhoE and LaminB are farnesylation-dependent proteins that are selectively upregulated in DTCs
- Genetic or pharmacologic inhibition of these targets kills DTCs and prevents the emergence of osimertinib-resistant mutant cells
- Combination of tipifarnib and osimertinib delays relapse *in vivo*

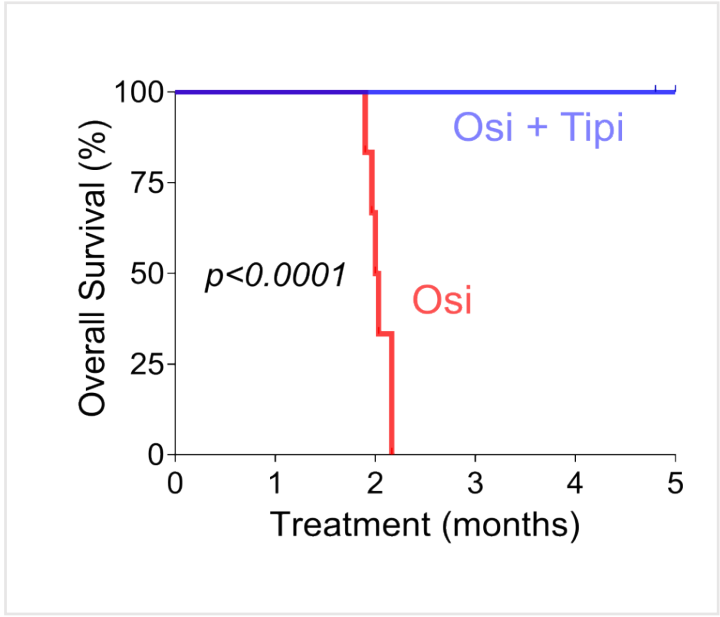


TIPIFARNIB PREVENTS EMERGENCE OF RESISTANCE TO OSIMERTINIB IN VIVO







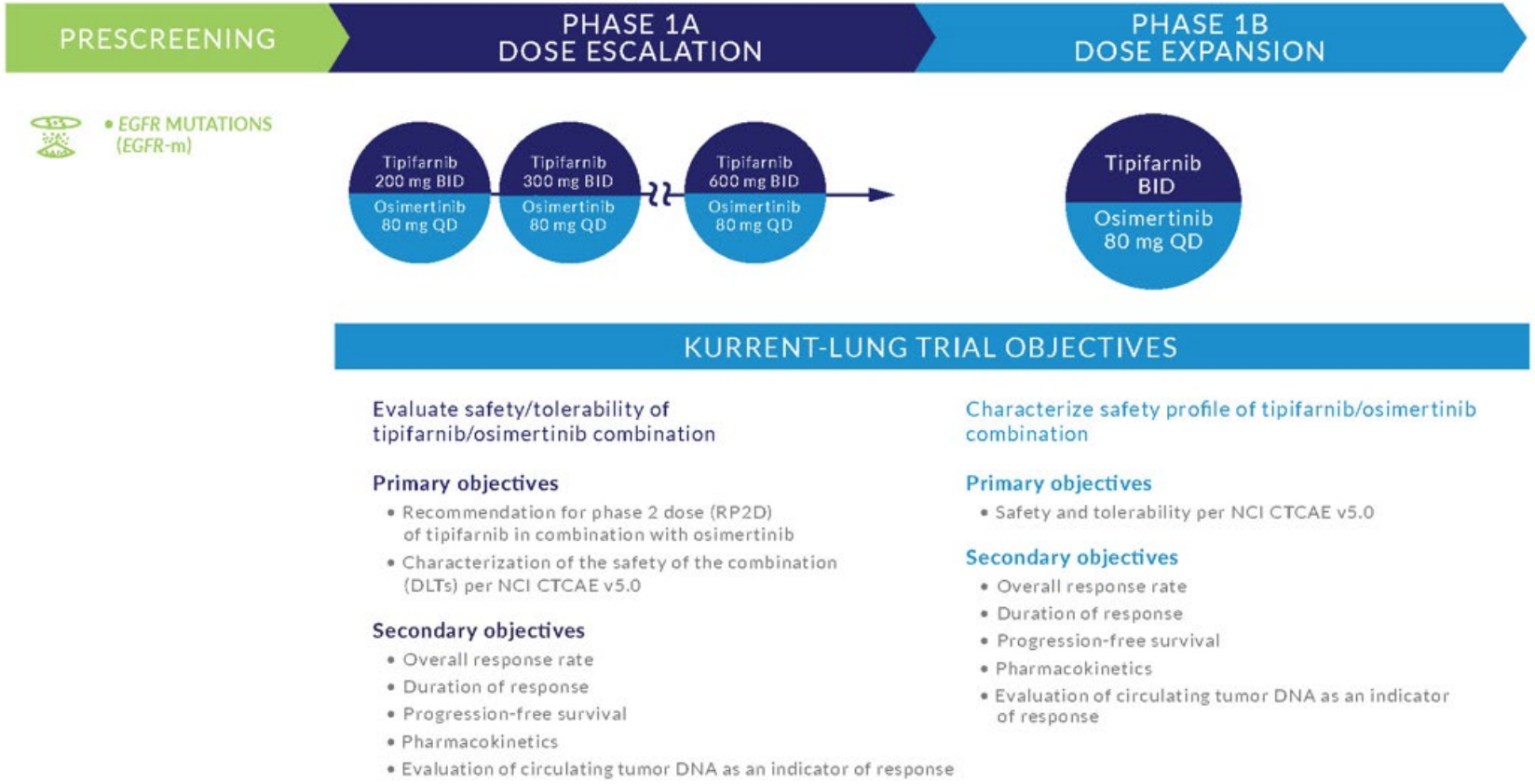


- Preclinical data generated through a collaboration with INSERM (the French National Institute of Health and Medical Research), suggest the potential to prevent emergence of resistance to EGFR inhibitor, osimertinib

Figarol *et al.* AACR 2022 #7934

TIPIFARNIB PREVENTS EMERGENCE OF RESISTANCE TO OSIMERTINIB IN VIVO

KURRENT-LUNG
KURA KO-TIP-015

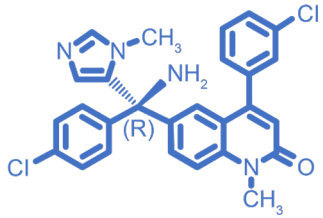


Phase 1 clinical trial of tipifarnib and osimertinib in patients with treatment-naïve, locally advanced, or metastatic *EGFR*-Mutant NSCLC

ORR = overall response rate; DOR – duration of response;
PFS = progression-free survival

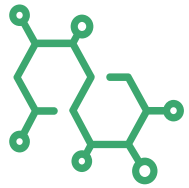
NEXT-GENERATION FARNESYL TRANSFERASE INHIBITOR (FTI)

KO-2806 nominated as development candidate for IND-enabling studies



Tipifarnib

Potency: Good
Selectivity: Excellent
Dose: 600 mg
Frequency: BID



KO-2806

Improved potency, pharmacokinetic and physicochemical properties

- FTIs represent an attractive therapeutic target and commercial franchise in oncology with compelling opportunities in combination with other targeted therapies
- Goal is to develop a next-generation FTI with improved potency, pharmacokinetic and physicochemical properties
- IND-enabling studies ongoing; on track for IND application acceptance for KO-2806 in Q1 2023

FORECASTED MILESTONES & FINANCIAL HIGHLIGHTS

PROGRAM	MILESTONE	ESTIMATED TIME OF ACHIEVEMENT
ZIFTOMENIB (KO-539) Menin Inhibitor	Dose first patient in Phase 2 registration-directed portion of KOMET-001	Q1 2023
	Dose first patient in KOMET-007 (venetoclax+azacitidine, 7+3)	1H 2023
	Dose first patient in KOMET-008 (gilteritinib, FLAG-IDA, IDAC/LDAC)	2H 2023
TIPIFARNIB Farnesyl Transferase Inhibitor (FTI)	Dose first patient in KURRENT-LUNG study (osimertinib)	1H 2023
	Determine OBAD* for PIK3CA cohort in KURRENT-HN study (alpelisib)	Mid-2023
KO-2806 Next-Generation FTI	Acceptance of Investigational New Drug application	Q1 2023

Financial Highlights* Nasdaq: KURA	$438M in Cash as of December 31, 2022**
	Shares outstanding: 68.3M basic; 9.3M options, RSU's & warrants

* Optimal biologically active dose
** Unaudited, preliminary cash, cash equivalents and short-term investments as of 12/31/2022

DEVELOPING PRECISION MEDICINES FOR THE TREATMENT OF CANCER

Corporate Presentation – January 2023