KURA ONCOLOGY

DEVELOPING PRECISION MEDICINES FOR THE TREATMENT OF CANCER

Corporate Presentation – August 2022

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 targeted oncology drug candidates using a precision medicine approach; fast-to-market strategy; global commercial rights

Proprietary Pipeline

Menin Inhibitor Program (Ziftomenib)

- Novel menin inhibitor with potential to target 35% or more of AML
- Encouraging safety, tolerability and clinical activity in multiple genetically defined subgroups of AML
- Recommended Phase 2 dose and data from Phase 1b study in NPM1-mutant and KMT2A-rearranged AML in 2H 2022

Farnesyl Transferase Inhibitor Programs (Tipifarnib & KO-2806)

- Registration-directed trial of tipifarnib in HRAS mutant HNSCC ongoing
- Encouraging safety, tolerability and clinical activity in Phase 1 study of tipifarnib plus alpelisib in PIK3CA-dependent HNSCC
- Phase 1 study of tipifarnib plus osimertinib in NSCLC to start in Q3 2022
- IND for KO-2806, next-generation FTI, on track for Q4 2022

Strong Financials

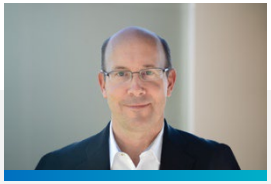
$450.3 million in cash* provides runway through 2024

*Cash, cash equivalents and short-term investments as of June 30, 2022

Kura Leadership Team and Board of Directors

Proven oncology drug discovery, development and commercialization expertise

Leadership Team



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Drug Candidate Pipeline

Program	Preclinical	Phase 1	Phase 2	Registration Directed
Ziftomenib (KO-539) Menin Inhibitor	**Acute Myeloid Leukemia (AML)** *KOMET-001 Trial*			
	• *Topline data from Phase 1b study later this year; full data presentation in Q4 2022*			
Tipifarnib Farnesyl Transferase Inhibitor (FTI)	**HRAS mutant Head & Neck Squamous Cell Carcinoma (HNSCC)** *AIM-HN Trial*			
	• *Enrollment in registration directed trial ongoing*			
	PIK3CA / HRAS Dependent HNSCC *KURRENT-HN Trial*			
	• *Enrollment in PIK3CA dependent and HRAS overexpression cohorts ongoing*			
	EGFR Mutant NSCLC *KURRENT-LUNG Trial*			
	• *Preparing to initiate Phase 1 trial in Q3 2022*			
KO-2806 Next-Generation FTI	**Solid Tumors**			
	• *IND enabling studies ongoing*			

ZIFTOMENIB (KO-539): MENIN INHIBITOR IN ACUTE LEUKEMIAS

Ziftomenib (KO-539): Menin Inhibitor

Potent, selective, reversible, oral inhibitor of menin-KMT2A(MLL) protein-protein interaction for treatment of AML

Novel MOA targeting epigenetic dysregulation leading to differentiation block and anti-tumor activity in 35% or more of AML

Preliminary data from KOMET-001 Phase 1/2 trial show encouraging safety, tolerability and clinical activity in multiple genetically defined subgroups of AML

Focused monotherapy development strategy in multiple genetic subtypes:

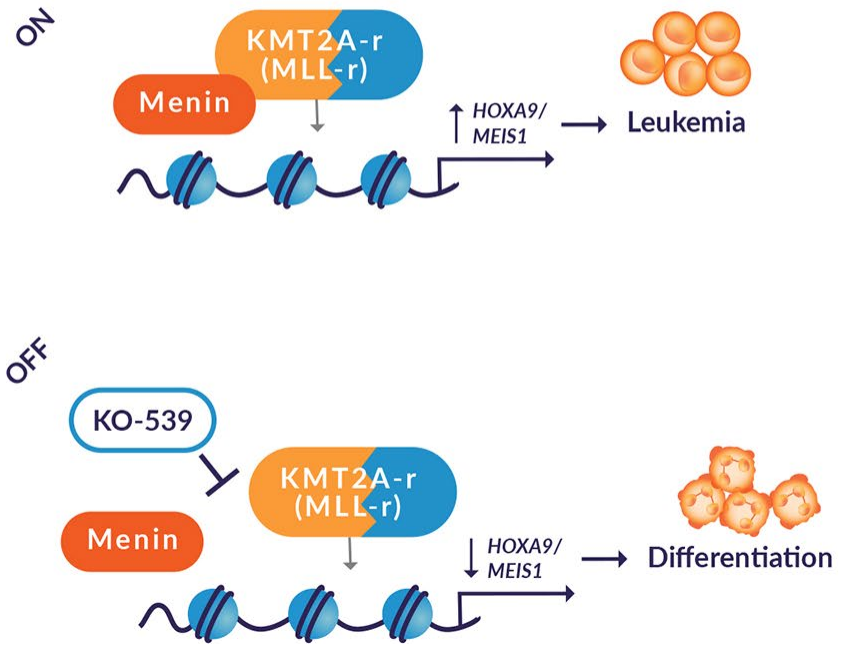
- KMT2A(MLL) rearranged (5-10% of AML)
- NPM1 mutant (~30% of AML)
- Other genetic subtypes (*e.g.,* SETD2/RUNX1-mutant AML)

Potential to combine with other targeted therapies and induction chemotherapy in earlier lines of therapy

Issued and pending COM patents provide worldwide coverage to 2036

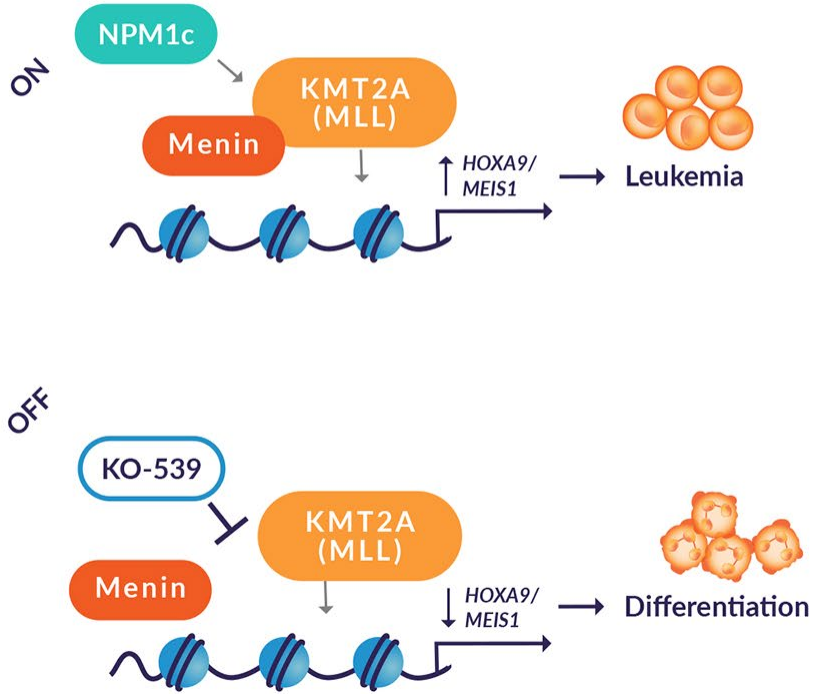
Targeting Menin-KMT2A(MLL) Interaction Provides Potential Therapeutic Intervention into Two Genetic Subsets of AML

KMT2A-r (MLL-r)



Targeting the menin-KMT2A(MLL) interaction to reverse epigenetic dysregulation in MLL-rearranged AML

NPM1 Mutant AML

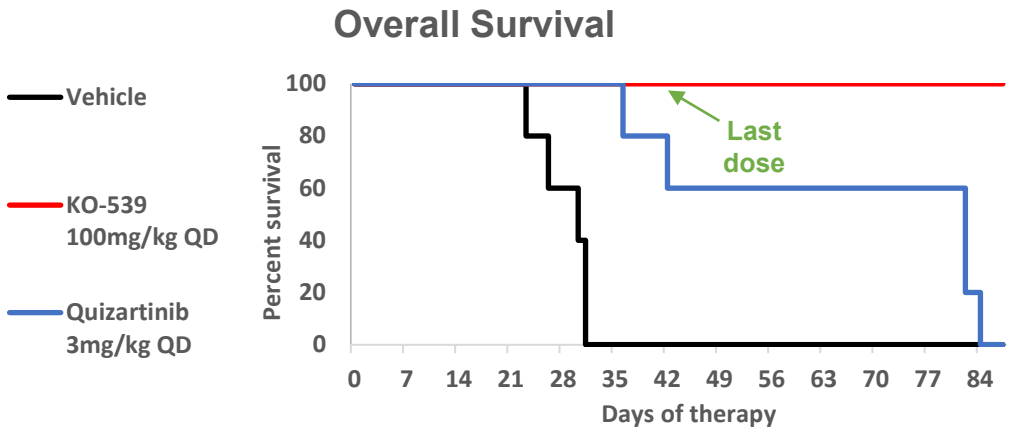


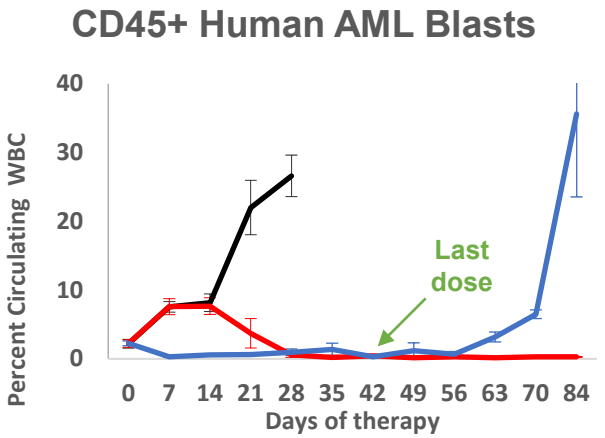
A central role for menin-KMT2A(MLL) interaction in epigenetic dysregulation in NPM1-mutant AML

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

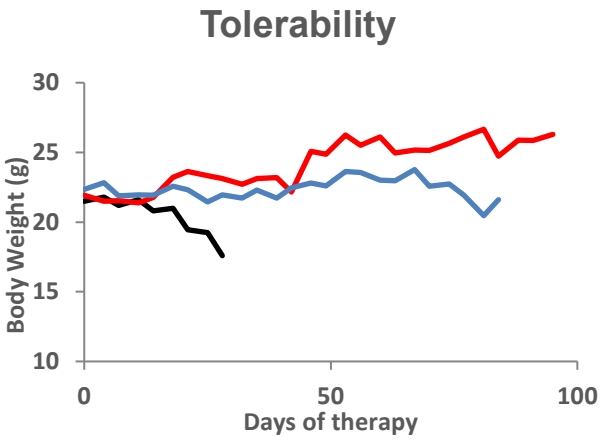
Ziftomenib (KO-539) Produces Lasting Complete Remissions in a NPM1 / DNMT3A / IDH2 / FLT3-Mutant AML Model

AM7577





- 100% (10/10) of animals treated with single-agent ziftomenib cleared their leukemia and became long-term survivors
- Tumor growth inhibition was durable – no leukemia was detectable in blood or bone marrow two months after cessation of dosing
- Ziftomenib was well tolerated at tested dose levels
- Comparator compound (FLT3 inhibitor) was initially active, but all animals eventually relapsed



Ziftomenib (KO-539) Demonstrates Encouraging Early Clinical Activity

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KURA KO-MEN-001

Clinical or biological activity reported in six of eight efficacy-evaluable patients

KOMET-001 (n=12)			
Dose	Mutational Profile	# of Prior Regimens	Clinical Activity
400 mg	*RUNX1, SRSF2, ASXL1, TET2, STAG2, BCOR, PTPN11*	3	Decreased peripheral blasts
	EZH2, DNMT3A, FAT3, RET	3	Progressive disease
	NPM1	2	Not efficacy evaluable at time of data cut
	DNMT3A, CUX1, ASXL1, IDH2, CBL, U2AF1, RUNX1	5	Not efficacy evaluable at time of data cut
200 mg	*NPM1, DNMT3A, KMT2D*	7	Complete remission, MRD-
	NPM1, FLT3-ITD, TET2, CUX1	4	Morphological leukemia-free state
	U2AF1, TET2, p53, DNMT3A, PTPN11	4	Stable disease
	IDH2, SRSF2, DNMT3A, CBL	3	Progressive disease
	TP53, PICALM (MLLT10)	3	Not efficacy evaluable
	KMT2A-r	4	Not efficacy evaluable
100 mg	*SETD2, RUNX1*	2	Complete remission, MRD+
50 mg	*KMT2A*-r	2	Decreasing hydrea requirement

Wang et al. ASH 2020 #115 (preliminary data as of November 2, 2020)

Case Study – *SETD2, RUNX1* Mutant AML

Patient Characteristics	
Demographics	69-year-old male
Mutational profile	*SETD2*, *RUNX1*
Prior lines of therapies	2 (decitabine; CD33/CD3 bispecific antibody)
Ziftomenib dose	100 mg, escalated to 200 mg during cycle 7
# of ziftomenib cycles	8
CYP3A4 inhibitor	Yes (fluconazole)
Baseline bone marrow blasts	56%
Clinical activity	Complete remission, MRD+ (0.8% blasts)
Grade ≥3 TRAEs	Gr. 3 deep vein thrombosis

Preliminary data as of November 2, 2020

Case Study – *NPM1, DNMT3A, KMT2D, FLT3-TKD* Mutant AML

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Patient Characteristics	
Demographics	44-year-old female
Mutational profile	*NPM1*, *DNMT3A*, *KMT2D*, *FLT3-TKD*
Prior lines of therapies	7 (incl. decitabine+venetoclax, gilteritinib, itacitinib, fludarabine, bortezomib)
Ziftomenib dose	200 mg
# of ziftomenib cycles	3+ (on treatment)
CYP3A4 inhibitor	Yes (posaconazole)
Baseline bone marrow blasts	14%
Clinical activity	Complete remission, MRD- (0% blasts)
Grade ≥3 TRAEs	Gr. 4 lipase increased, Gr. 3 pancreatitis, Gr. 3 neutrophil count decreased

Preliminary data as of November 2, 2020

Continuous Daily Dosing of Ziftomenib in Patients with Relapsed or Refractory AML

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- No dose discontinuations due to treatment-related adverse events (AEs)
- No evidence of QT prolongation or other clinically significant ECG changes

Treatment-related AEs (N=12)	Grade ≥ 3 (all)	Grade 1,2 (≥ 10%)
Pancreatitis	1* (8.3%)	0%
Lipase increased	1* (8.3%)	0%
Neutrophil count decreased	1* (8.3%)	0%
Tumor lysis syndrome	1 (8.3%)	0%
Deep vein thrombosis	1 (8.3%)	0%
Nausea	0%	3 (25%)
Rash	0%	2 (16.7%)
Diarrhea	0%	2 (16.7%)

* Pancreatitis, increased lipase and decreased neutrophil count were observed in an NPM1 mutant AML patient who went on to achieve a complete remission (CR) with no measurable residual disease (MRD) after seven prior regimens

Wang et al. ASH 2020 #115 (preliminary data as of November 2, 2020)

Summary of Preliminary Data from KOMET-001

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- Ziftomenib (KO-539) is a potent and selective inhibitor of the menin-KMT2A/MLL complex
- Encouraging safety profile and tolerability to date
  - No evidence of QTc prolongation
  - Differentiation syndrome, a known on-target effect, appears manageable with enhanced mitigation strategy
- Encouraging signs of clinical activity in multiple genetically defined subgroups of AML
- Phase 1b expansion cohorts comprised of patients with NPM1-mutant or KMT2A-rearranged relapsed/refractory AML
- Recommended Phase 2 dose and data from Phase 1b study expected in 2H 2022

KOMET-001: Phase 1/2 Clinical Trial of Ziftomenib (KO-539) in Patients with Relapsed or Refractory AML

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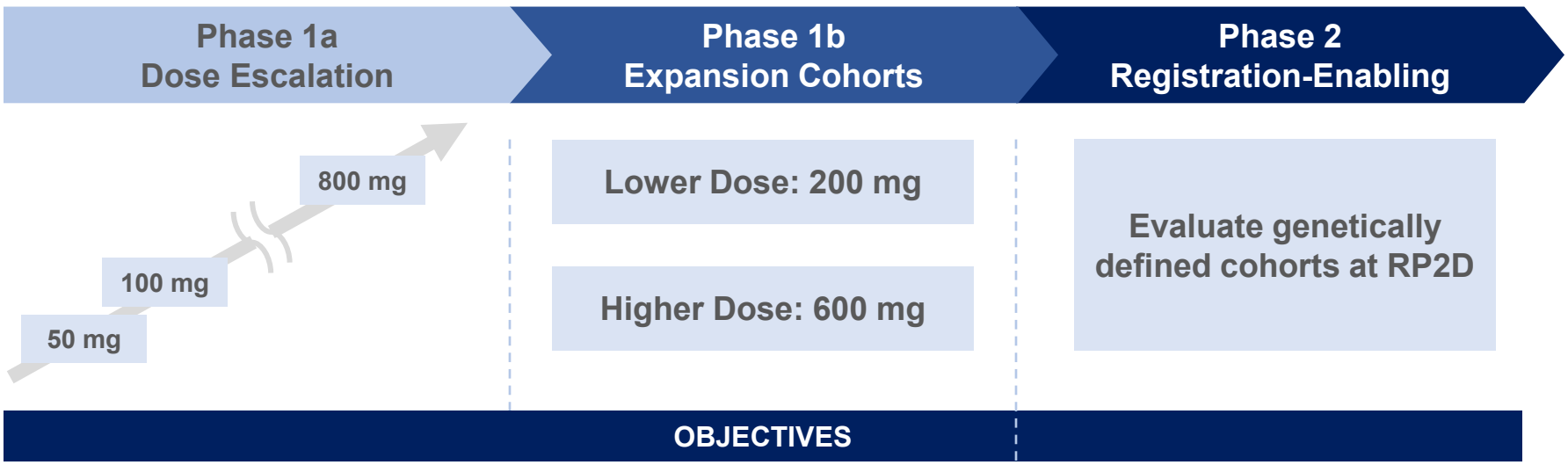

Dose escalation in all comer patients to establish MTD and/or recommended Phase 2 dose (RP2D)

- Safety and tolerability
- Pharmacokinetics
- Early evidence of anti-tumor activity

Enroll two genetically enriched cohorts to enable refinement of RP2D

- Safety and tolerability
- Pharmacokinetics
- Anti-tumor efficacy

Enroll genetically enriched cohorts at RP2D for registration

- Primary endpoint: CR/CRh
- Safety and tolerability
- Other secondary and exploratory endpoints

KOMET: Kura Oncology Menin-MLL Trial

Multiple Expansion Opportunities in Acute Leukemias

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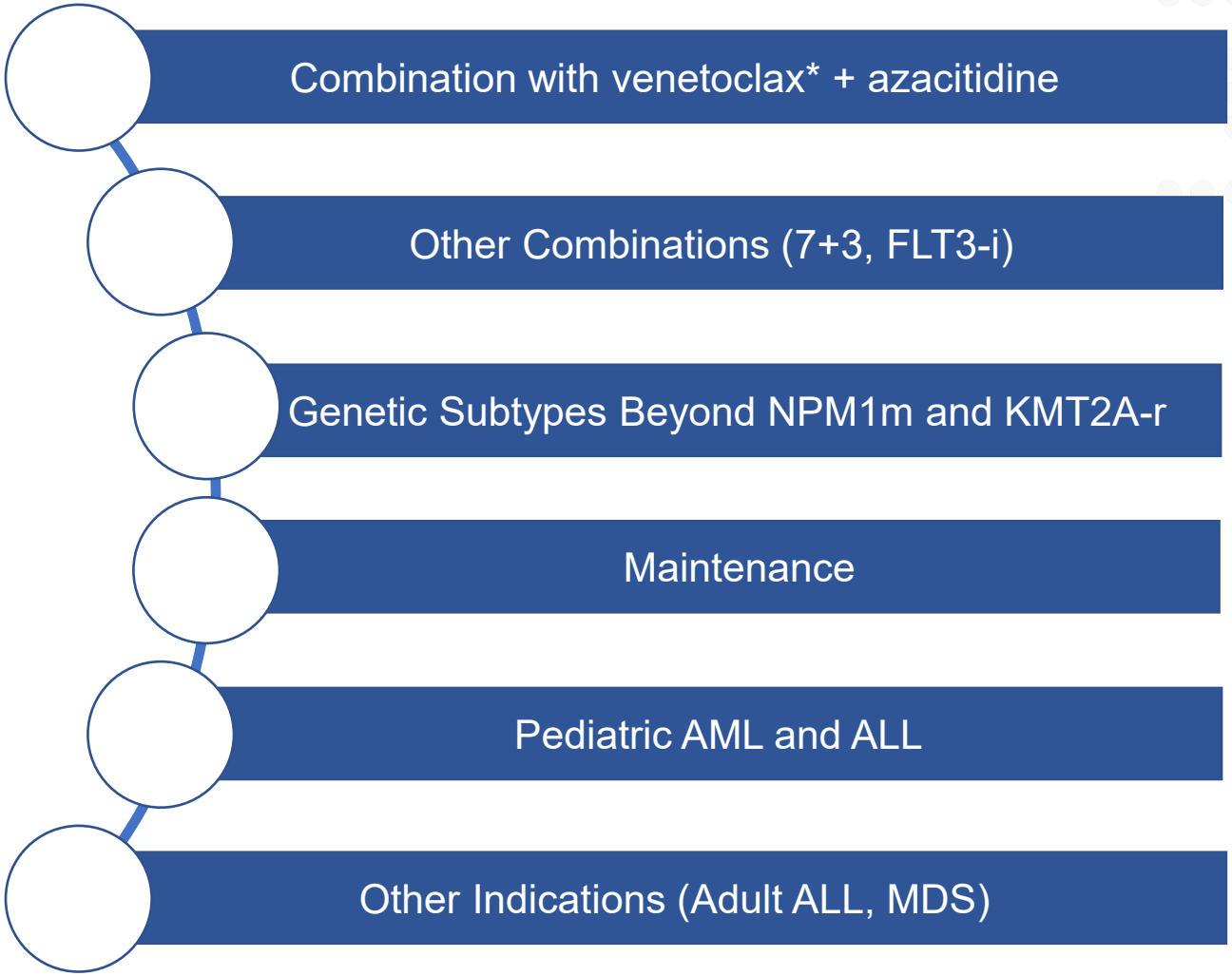
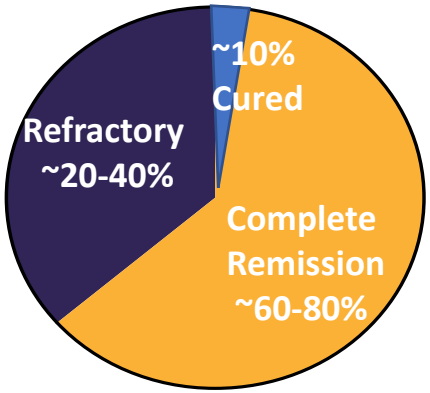


* Pre-clinical abstract showing synergistic activity of ziftomenib in combination with venetoclax presented at ASH 2021

Prognosis Remains Poor for Most Patients with NPM1m or KMT2A-r AML; No FDA-Approved Targeted Therapies Exist

AML Disease Status Following First Induction[1]



- ~ 1/2 of patients who achieve a CR* **will relapse**[1]
- R/R treatments are **sub-optimal**[2-4]
- Less than 10% of patients in R/R are **alive at 3 years**[5]

*CR; complete response

NPM1-Mutant AML

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

Adult patients with NPM1m and select co-mutations and/or R/R disease are associated with poor prognosis[7]

5-year Overall Survival ~50%[8]

KMT2A-Rearranged AML

~1,000-2,000 new cases annually in U.S.[6]

Adult patients with KMT2A-r have poor prognosis with high rates of resistance and relapse following current SoC[9 10]

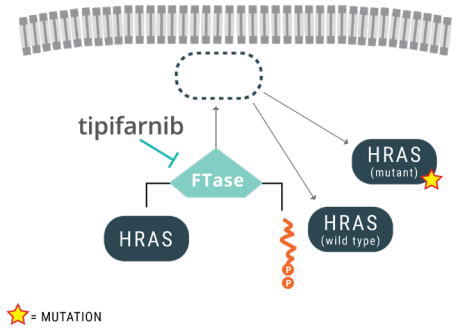
5-year Overall Survival <20%[9]

[1] Megías-Vericat JE, et al. Ann Hematol. 2018;97(7):1115-1153.
[2] DeWolf S, Tallman MS. Blood. 2020 Aug 27;136(9):1023-1032.
[3] Roboz *et al*. J Clin Oncol. 2014 Jun 20;32(18):1919-26.
[4] Perl *et al*. Engl J Med. 2019 Oct 31;381(18):1728-1740.
[5] Bose P, et al. Curr Treat Options Oncol. 2017;18(3):17.
[6] SEER statistics for AML in the US, accessed April 2020.
[7] Döhner *et al*. Blood. 2017 Jan 26;129(4):424-447.
[8] Angenendt L, et al. J Clin Oncol. 2019;37(29):2632-2642.
[9] Issa GC, et al. Blood Cancer J. 2021;11(9):162.
[10] Vetro C, et al. Cancer Genet. 2020;240:15-22.

FARNESYL TRANSFERASE INHIBITOR PROGRAMS

Therapeutic Applications of Farnesyl Transferase Inhibitors

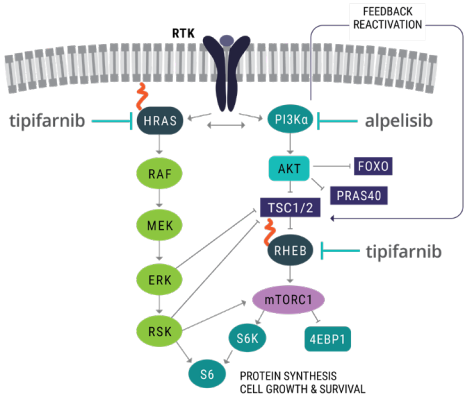
Direct Inhibition of Oncogenic Proteins



- Monotherapy activity in mutant tumors

AIM-HN
KURA KO-TIP-007

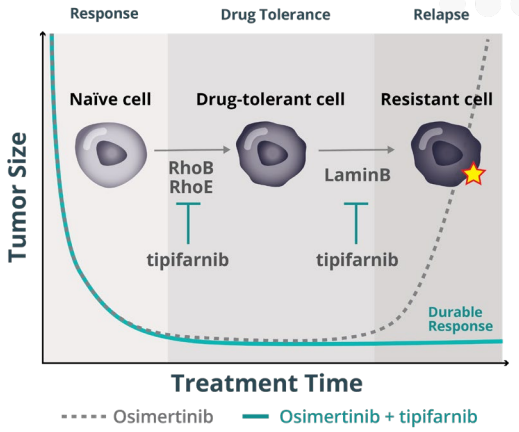
Overcoming Drug Resistance



- Overcome drug resistance to PI3Kα inhibitor in HNSCC

KURRENT-HN
KURA KO-TIP-013

Preventing Emergence of Resistance

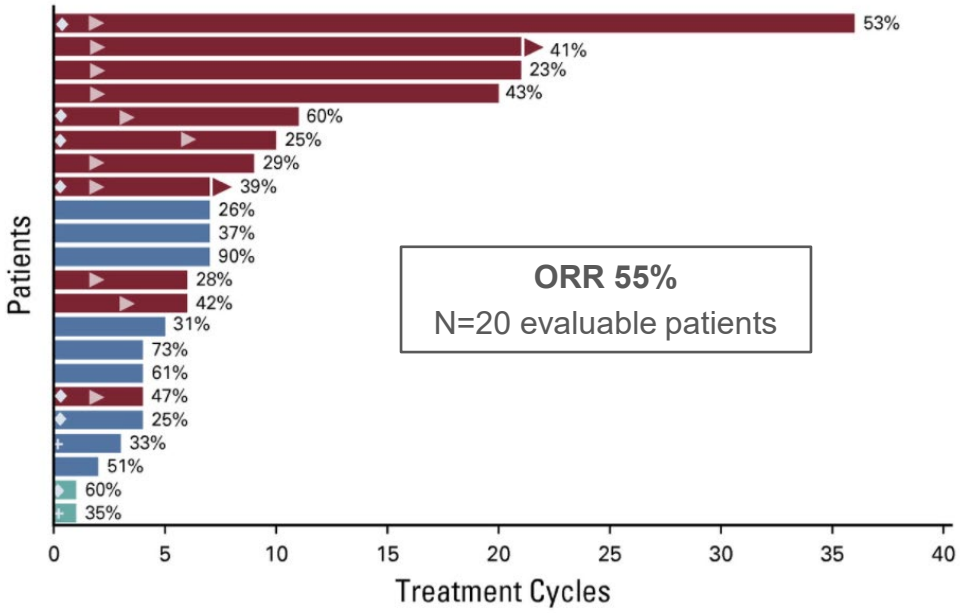


- Prevent emergence of resistance to EGFR inhibitor in NSCLC

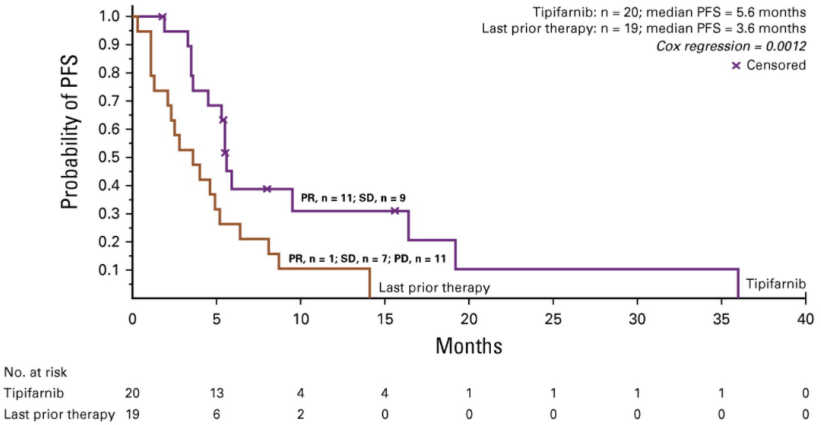
KURRENT-LUNG
KURA KO-TIP-015

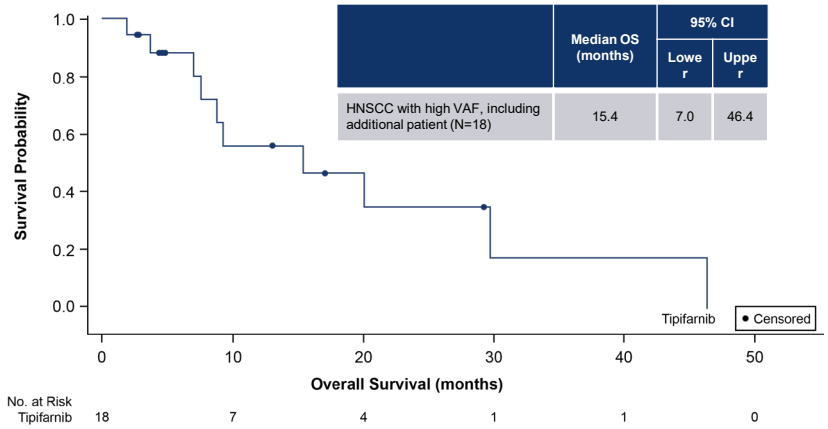
Tipifarnib: Durable Anti-Tumor Activity in Patients with Recurrent or Metastatic HRAS Mutant HNSCC

RUN-HN
KO-TIP-021



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.





Ho, et al. J Clin Oncol. 2021 Mar 22;JCO2002903. doi: 10.1200/JCO.20.02903. Online ahead of print.
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

Ongoing Registrational Program for Tipifarnib Monotherapy in HRAS mutated HNSCC

AIM-HN: Registration-directed trial of tipifarnib in HRAS mutant HNSCC

- Recurrent or metastatic patients after one prior line of platinum therapy
- Now open in > 100 clinical sites in the U.S., Europe and Asia
- Amended trial to enroll all HRAS mutant HNSCC patients regardless of variant allele frequency
- Intended to support an NDA seeking accelerated approval*

AIM-HN
KO-TIP-007

SEQ-HN: Prospective observational cohort of HNSCC

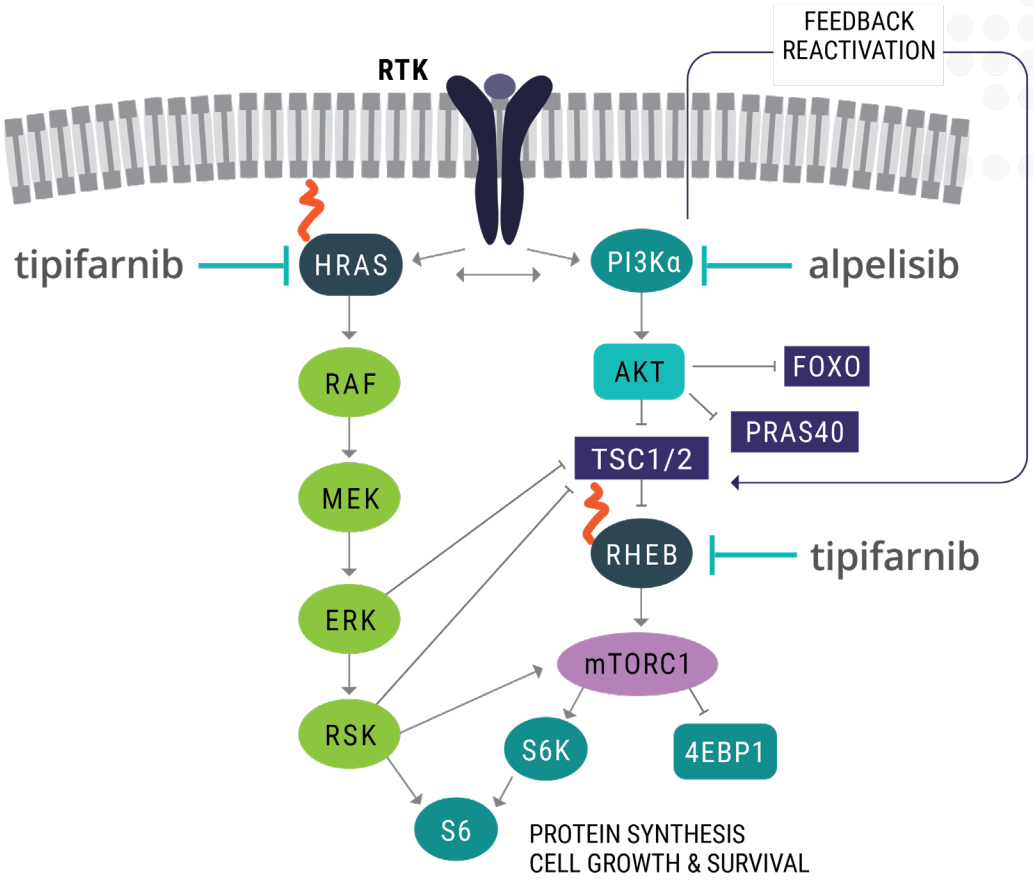
- Matched case-control study designed to:
  - Understand natural history of HRAS mutant HNSCC patients and their outcomes after first line therapy compared to wild-type controls
  - Enable identification of patients for potential enrollment into AIM-HN
- May support potential FDA labelling discussions, post-approval commitments and commercial considerations

SEQ-HN
KO-TIP-007

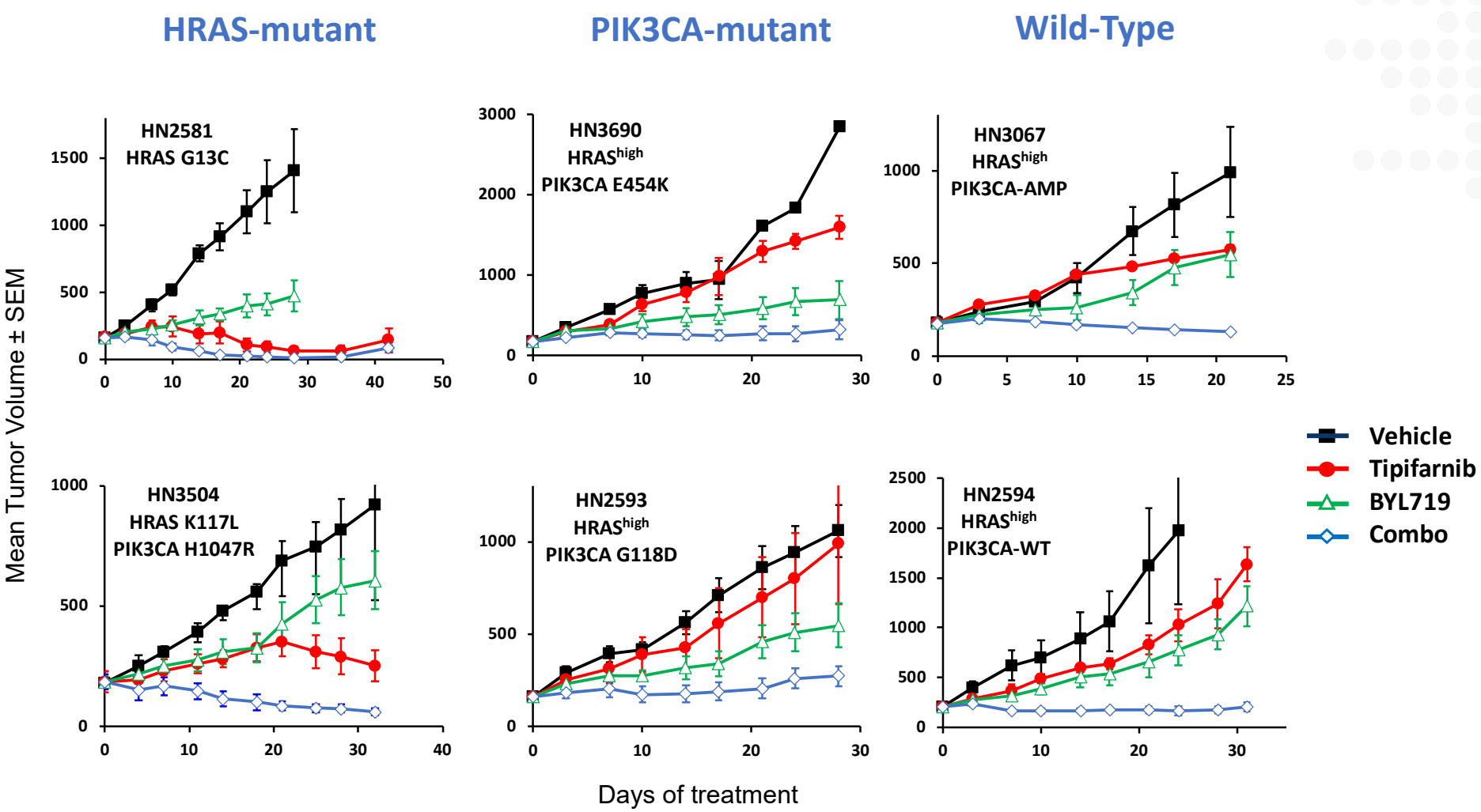
* Feedback from end-of-Phase 2 meeting with FDA 2018

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

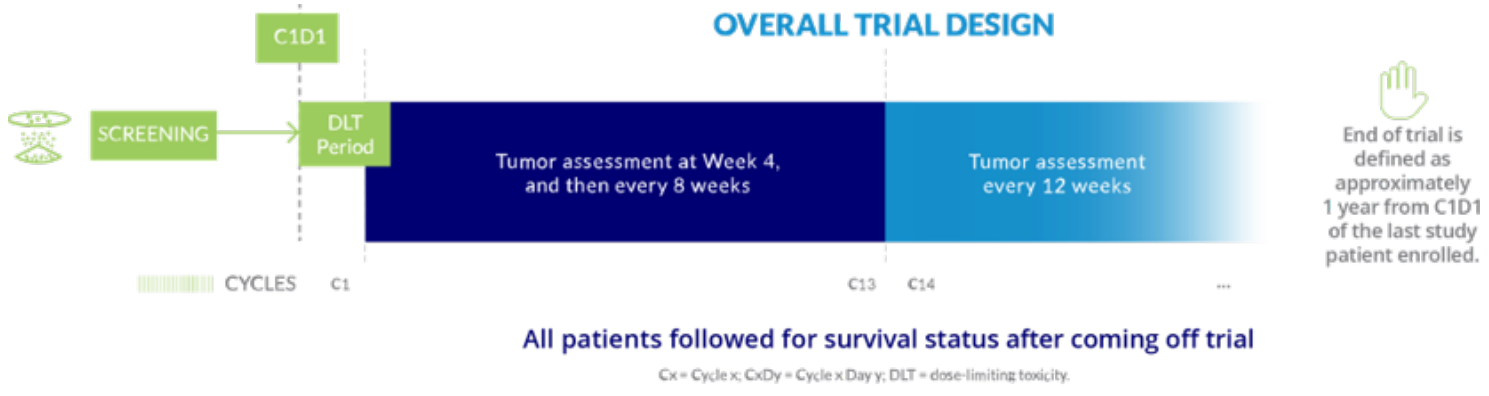
- HRAS-MAPK and PI3K-mTOR are complementary pathways in HNSCC
  - Overexpression of WT HRAS reported to induce resistance to PI3Kα inhibition
  - HRAS is reported to preferentially activate PI3K (vs. KRAS; vs. MAPK)
- HRAS mutation/over expression and PIK3CA mutations/amplifications account for up to 50% 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

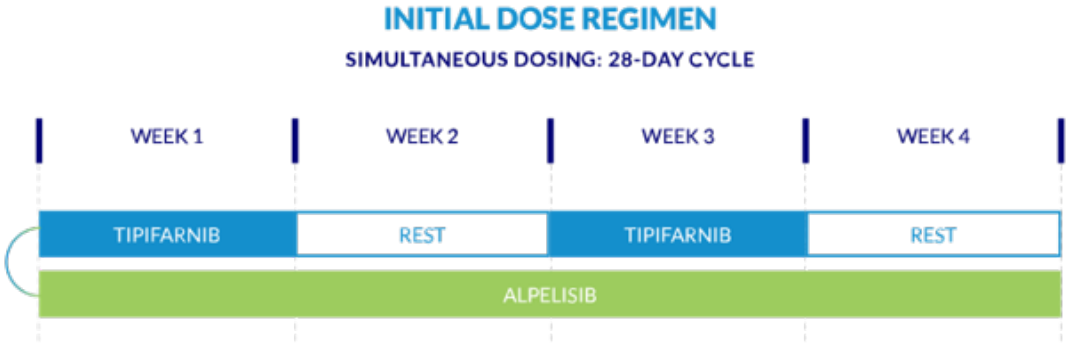
[1] TCGA Data

References: Yan J et al (1998) JBC 273:24052 ; Gupta S et al (2007) Cell 129:957 ; Zhao L et al (2008) PNAS 105:2652

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

KURRENT-HN
KURA KO-TIP-013

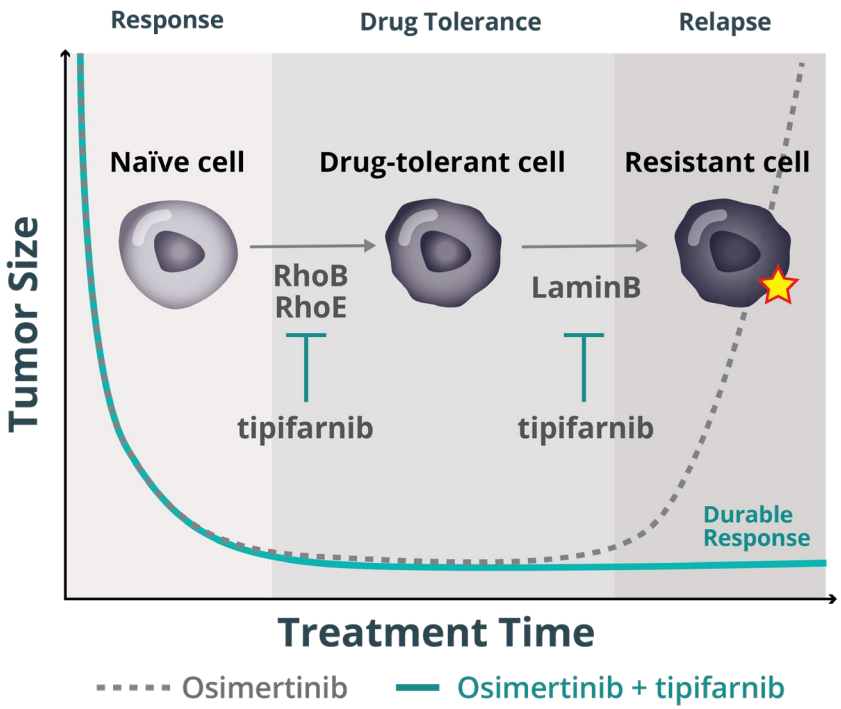




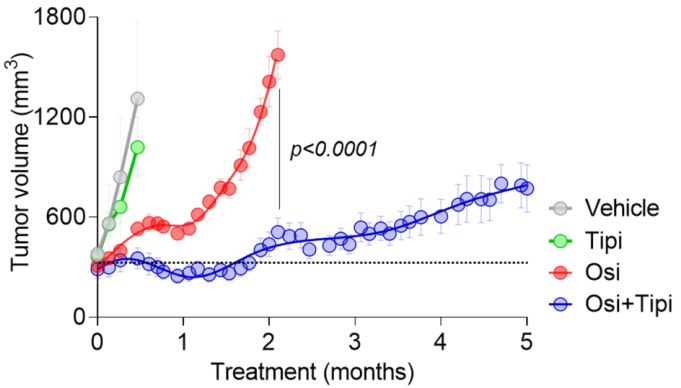
- Clinical collaboration to evaluate tipifarnib in combination with alpelisib for the treatment of patients with HNSCC whose tumors have HRAS overexpression and/or PIK3CA mutation and/or amplification
- Under the collaboration, Kura sponsors the trial and supplies tipifarnib, and Novartis supplies alpelisib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

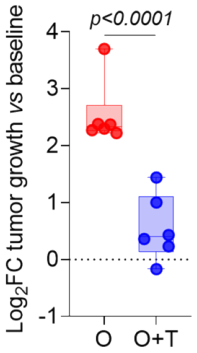
Compelling Preclinical Data Supports Development of FTI / Osimertinib Combinations in Frontline NSCLC

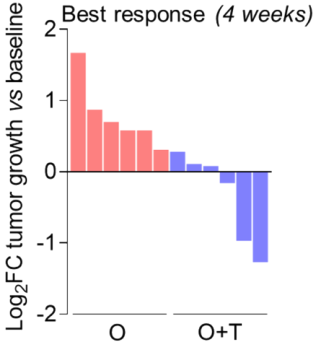
- Drug-tolerant cells (DTCs) arise within days of osimertinib exposure
- DTCs are characterized by Rho pathway activation
- RhoB, RhoE and LaminB are farnesylation-dependent proteins that are selectively upregulated in DTCs
- Genetic or pharmacologic inhibition of these targets kills DTCs and prevents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

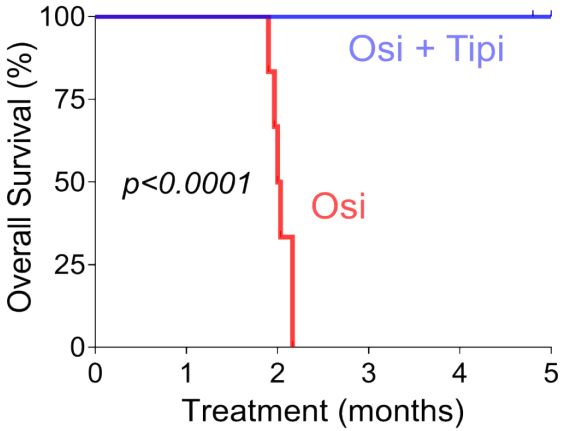


Tipifarnib Prevents Resistance to Osimertinib In Vivo







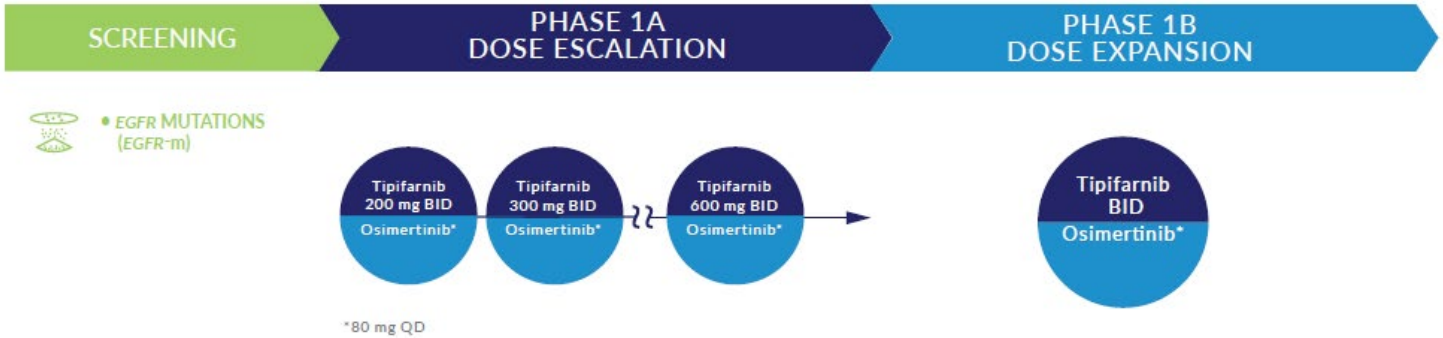


- New findings generated through a collaboration with INSERM (the French National Institute of Health and Medical Research)

KURRENT-LUNG: A Phase 1 Combination Trial of Tipifarnib and Osimertinib in Patients with NSCLC

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



KURRENT-LUNG TRIAL OBJECTIVES

Dose escalation to evaluate safety/tolerability in the *EGFR*-mutated NSCLC population

PRIMARY OUTCOMES
- Characterize the safety of the combination (DLTs) per NCI CTCAE v5.0

SECONDARY OUTCOMES
- Antitumor efficacy (ORR, DOR, PFS)
- Pharmacokinetics
- Evaluate circulating tumor DNA as an indicator of response

Dose expansion to characterize the safety profile of tipifarnib in combination with osimertinib

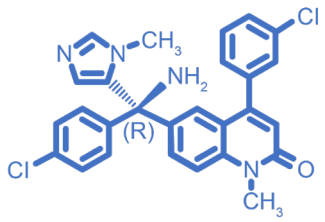
PRIMARY OUTCOME
- Safety and tolerability per NCI CTCAE v5.0

SECONDARY OUTCOMES
- Antitumor efficacy (ORR, DOR, PFS)
- Pharmacokinetics
- Evaluate circulating tumor DNA as an indicator of response

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

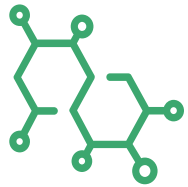
Next-Generation Farnesyl Transferase Inhibitor (FTI)

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



Tipifarnib

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



KO-2806

Potency: Improved
Selectivity: Comparable
Dose: Lower
Frequency: QD

- 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 to submit IND application for KO-2806 in Q4 2022

Forecasted Milestones & Financial Highlights

Program	Milestone	Status
Ziftomenib (KO-539) **Menin Inhibitor**	Determine recommended Phase 2 dose and report top-line data	2H 2022
	Present updated data from KOMET-001 at medical meeting	Q4 2022
Tipifarnib **Farnesyl Transferase Inhibitor (FTI)**	Enrollment in AIM-HN registration-directed study	Ongoing
	Dose first patient in HRAS overexpression cohort in KURRENT-HN	✓
	Initiate KURRENT-LUNG study in combination with osimertinib	Q3 2022
KO-2806 **Next-Generation FTI**	Submit IND application for KO-2806	Q4 2022

Financial Highlights* **Nasdaq: KURA**	Cash, cash equivalents and short-term investments: $450.3M* Shares outstanding: 66.8M basic; 9.1M options, RSU's & warrants

*As of June 30, 2022

KURA ONCOLOGY

DEVELOPING PRECISION MEDICINES FOR THE TREATMENT OF CANCER

Corporate Presentation – August 2022