KURA ONCOLOGY

DEVELOPING PRECISION MEDICINES FOR THE TREATMENT OF CANCER

Stifel Healthcare Conference – November 16, 2020

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 tipifarnib and KO-539, 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,” “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 two wholly owned, targeted oncology drug candidates using a precision medicine approach; fast-to-market strategy

Proprietary Pipeline

Tipifarnib: Farnesyl transferase inhibitor

- Registration-directed trial in HRAS mutant head and neck squamous cell carcinoma (HNSCC) ongoing
- Opportunity to expand to HRAS and PI3Kα dependent tumors
- Multiple clinical proof-of-concept studies support significant lifecycle expansion opportunities

KO-539: Menin inhibitor

- Potent and selective inhibitor of the menin-KMT2A(MLL) protein-protein interaction
- Potential to target ~35% of acute myeloid leukemia (AML)
- Preliminary data trial accepted for oral presentation at ASH 2020

Strong Financials

$325.4 million in cash* provides runway into 2023

* Cash, cash equivalents and short-term investments as of September 30, 2020

Kura Leadership Team and Board of Directors

Proven oncology drug development and commercialization expertise

Leadership Team

Troy Wilson, Ph.D., J.D.
President & Chief Executive Officer

James Basta, J.D.
Chief Legal Officer

Stephen Dale, M.D.
Chief Medical Officer

Kirsten Flowers
Chief Commercial Officer

Kathleen Ford
Chief Operating Officer

Marc Grasso, M.D.
Chief Financial Officer &
Chief Business Officer

Board of Directors

Faheem Hasnain
Executive Chairman, Gossamer Bio

Robert Hoffman
Former Chief Financial Officer, Heron Therapeutics

Thomas Malley
President, Mossrock Capital

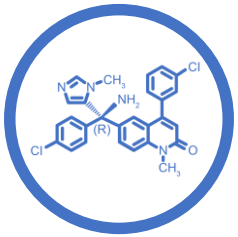
Diane Parks
Former Head of U.S. Commercial, Kite Pharma

Steven Stein, M.D.
Chief Medical Officer, Incyte

Mary Szela
President and CEO, TriSalus Life Sciences

Troy Wilson, Ph.D., J.D.
President and CEO, Kura Oncology

Advancing Targeted Oncology Drug Candidates Using a Precision Medicine Approach



Tipifarnib

Targeting HRAS Mutant Solid Tumors

- Fast Track Designation
- Initial opportunity to address high unmet need in relapsed/refractory HRAS mutant HNSCC
- Opportunities to expand to broader patient populations and to additional indications

KO-539

Targeting KMT2A(MLL)-r and NPM1 Mutant AML

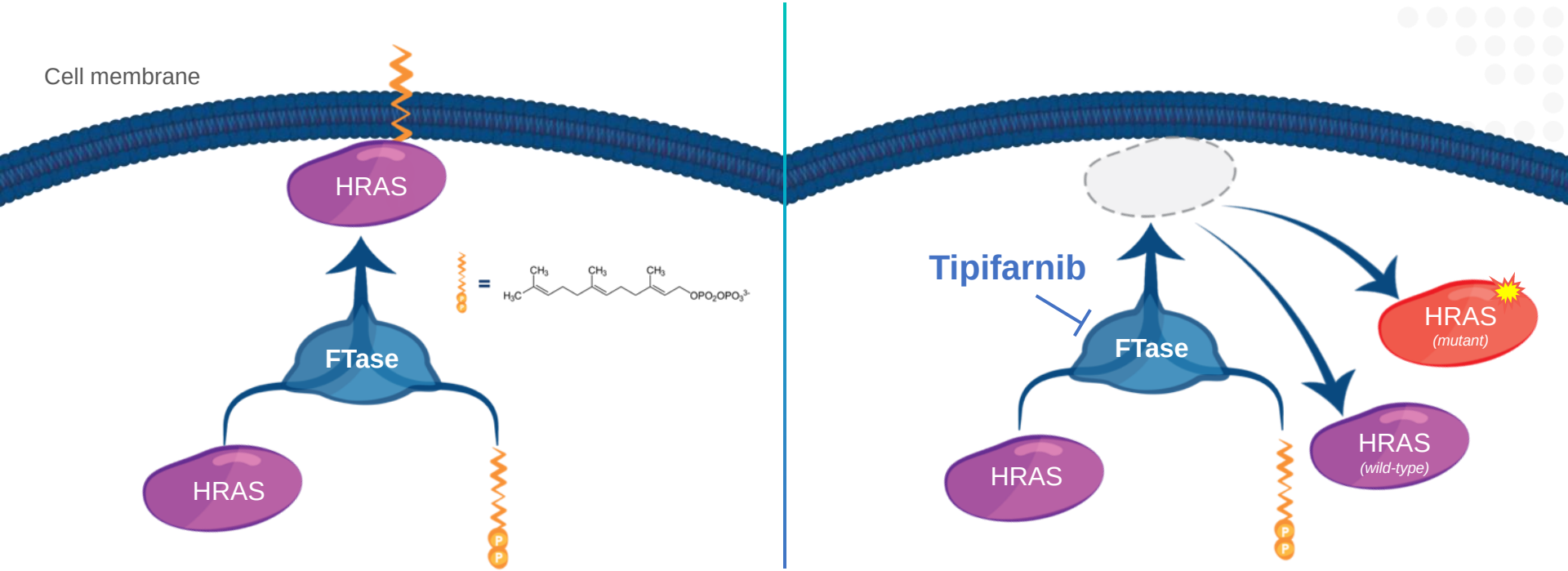
- Orphan Drug Designation
- Opportunity to address large patient population with high unmet need in relapsed/refractory AML
- Publications support potential to drive robust and persistent responses in KMT2A(MLL)-r and NPM1 mutant AML

TIPIFARNIB IN HRAS MUTANT SOLID TUMORS

Tipifarnib in HRAS Mutant Solid Tumors

- Unique MOA targets farnesylation, an essential modification required for activity of the HRAS mutant oncoprotein
- Phase 2 data demonstrates treatment response of ~ 50% ORR, ~ 6 months PFS and ~ 15 months OS in advanced recurrent and metastatic HRAS mutant HNSCC patients
- Favorable safety and tolerability profile supports broad use in advanced patients as well as expansion to earlier therapeutic settings
- Fast Track Designation in HRAS Mutant HNSCC; potential for accelerated approval
- Novel mechanism and well tolerated profile could enable use in combination with standard of care, including immune therapy, targeted therapies and chemo
- Issued and pending patents provide exclusivity to 2036 in major markets

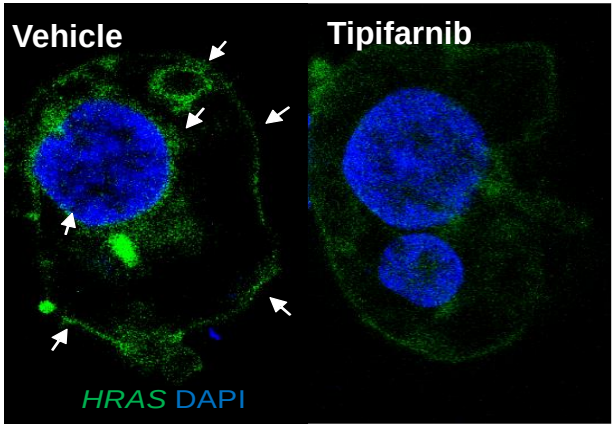
Tipifarnib Inhibits Farnesylation – An Essential Modification Required for HRAS Activity



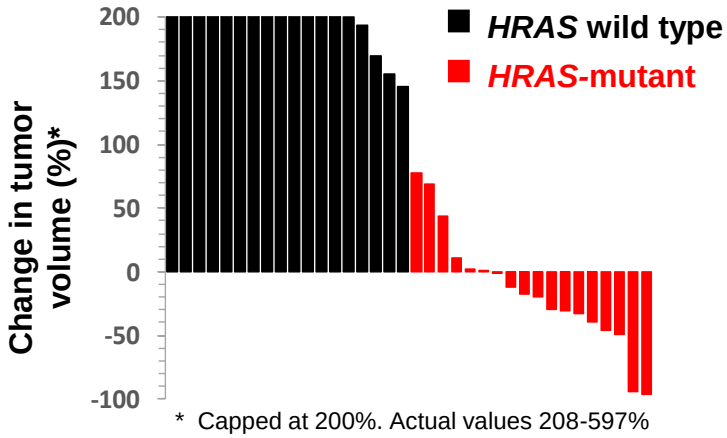
- Tipifarnib inhibits farnesylation and signaling activity of the HRAS oncoprotein
- Farnesylation is essential for HRAS signal transduction activity
- HRAS mutations drive proliferation and resistance mechanisms in solid tumors
- Incidence of HRAS mutations in HNSCC is approximately 4-8% and varies by region

Tipifarnib Displays Robust, Selective Activity in HRAS Mutant HNSCC Models

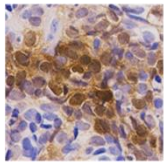
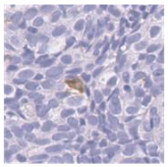
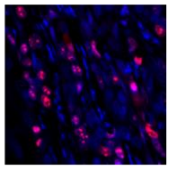
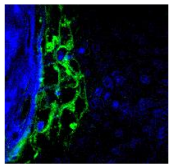
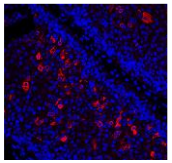
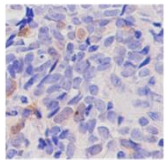
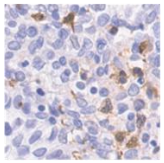
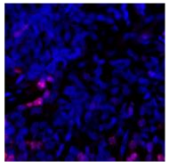
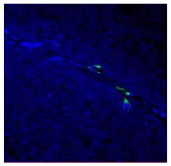
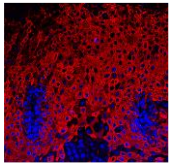
HRAS membrane displacement



Antitumor activity in PDX models



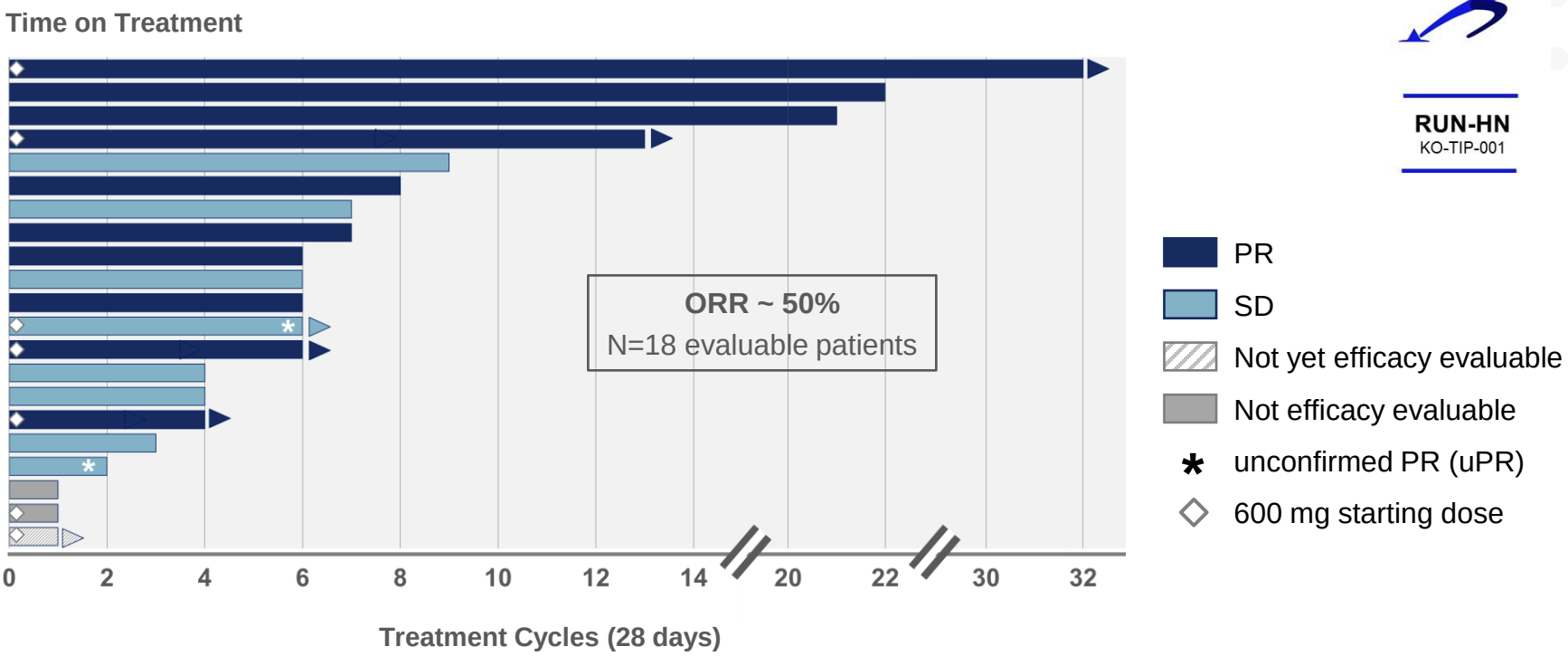
* Capped at 200%. Actual values 208-597%

	MAPK signaling	Apoptosis	Cell cycle arrest	Angiogenesis	Squamous differentiation
Vehicle					
Tipifarnib					
	pERK	c.CSP3	Ki67	CD31	KRT4

Source: Kura internal data

Durable Anti-Tumor Activity with Tipifarnib as a Monotherapy in Patients with HRAS Mutant HNSCC

RUN-HN: Phase 2 Trial in HRAS Mutant HNSCC



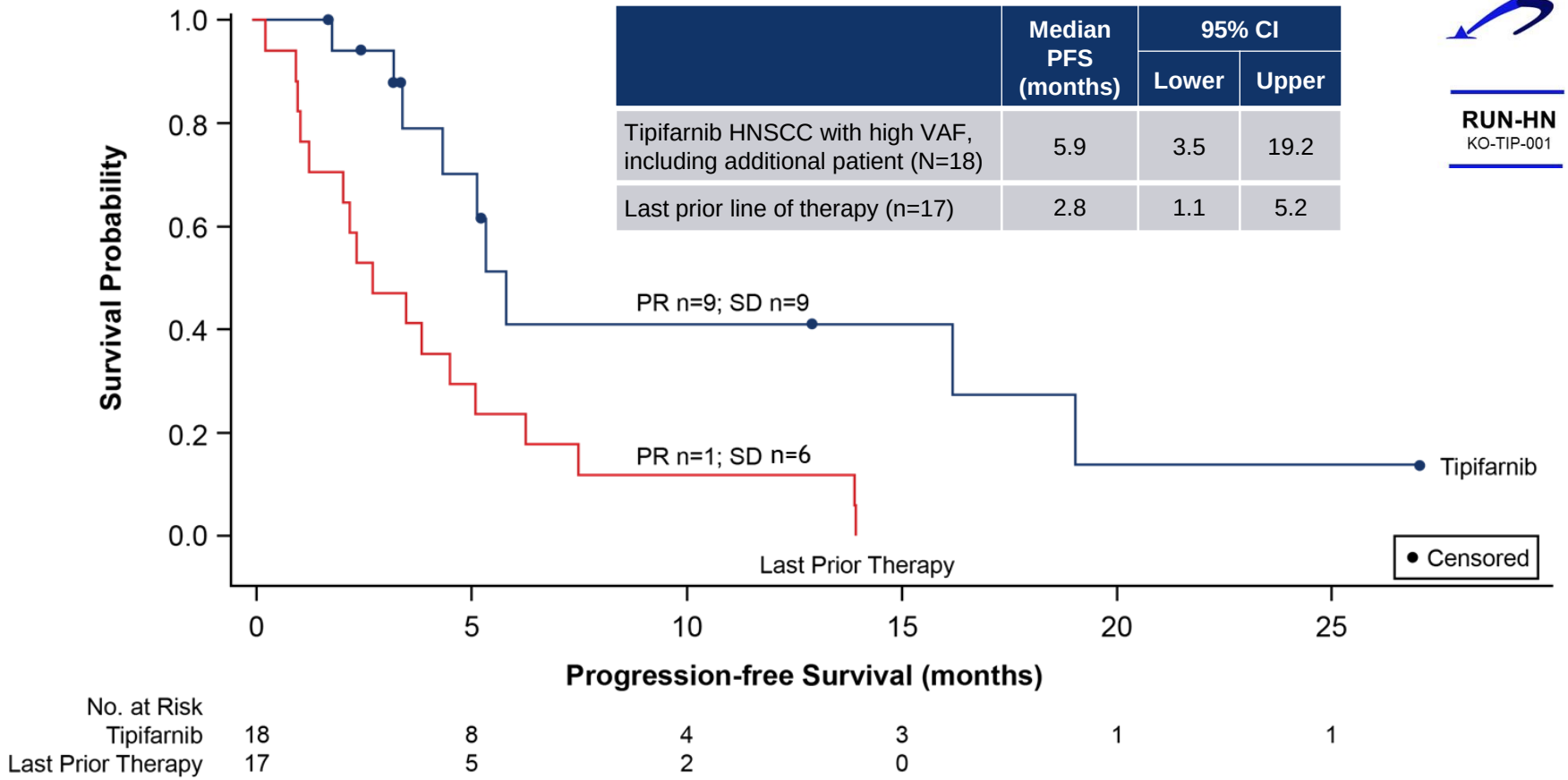
Ho *et al*. AACR-NCI-EORTC 2019 #384 (preliminary data as of 10/17/19)
Efficacy-evaluable patients with HRAS mutant variant allele frequency (VAF) ≥ 20% and serum albumin ≥ 3.5 g/dL, or HRAS VAF ≥ 35%
One patient treated off-protocol through compassionate use

Progression-Free Survival with Tipifarnib and Last Prior Therapy in Patients with HRAS Mutant HNSCC

RUN-HN: Phase 2 Trial in HRAS Mutant HNSCC

	Median PFS (months)	95% CI	
		Lower	Upper
Tipifarnib HNSCC with high VAF, including additional patient (N=18)	5.9	3.5	19.2
Last prior line of therapy (n=17)	2.8	1.1	5.2

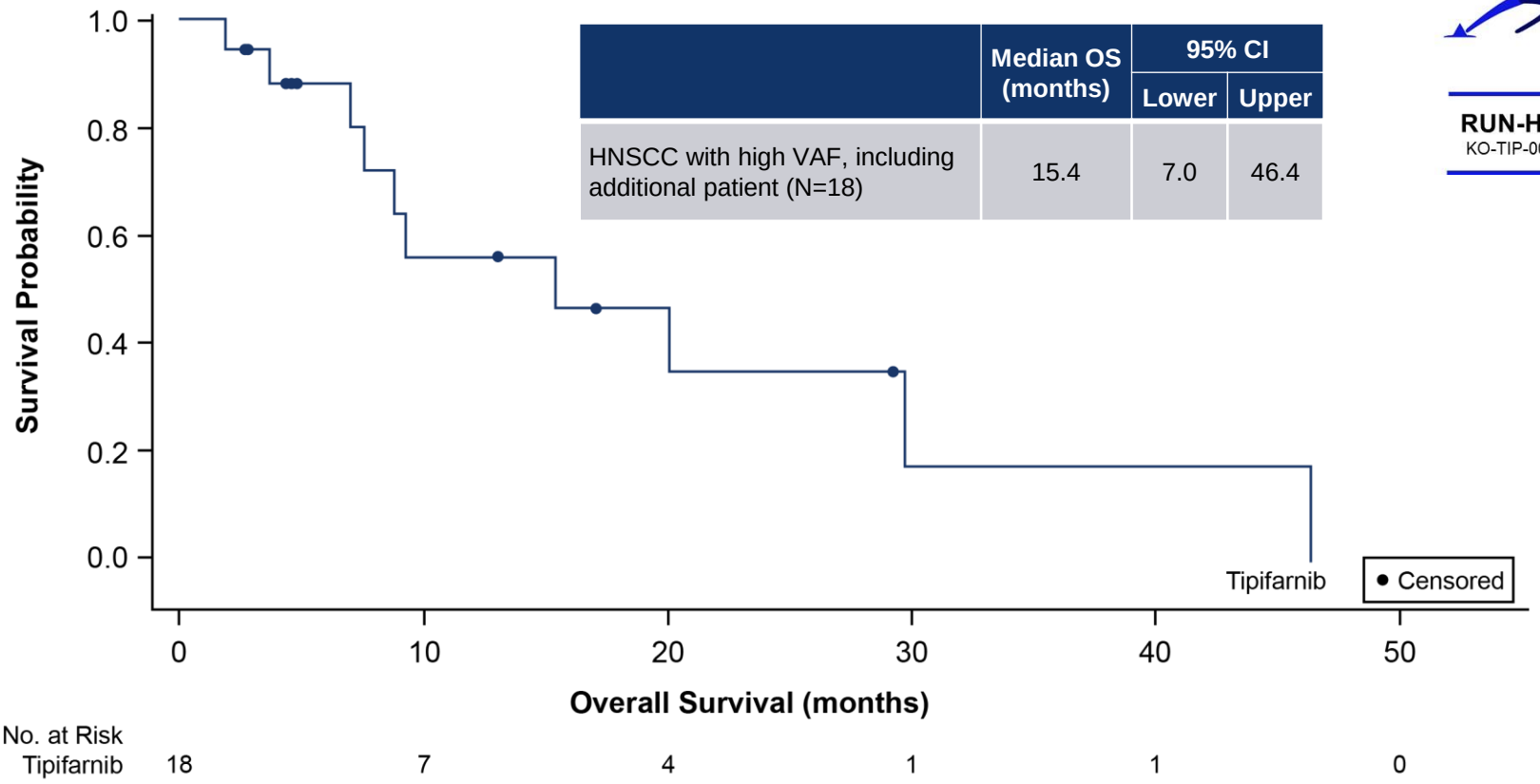
RUN-HN
KO-TIP-001



Ho *et al*. ASCO 2020 #6504 (preliminary 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

Overall Survival in Patients with HRAS Mutant HNSCC

RUN-HN: Phase 2 Trial in HRAS Mutant HNSCC



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

RUN-HN
KO-TIP-001

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

Registration Strategy in HRAS Mutant 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 ~90 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:
  - Characterize natural history of HRAS mutant HNSCC patients and their outcomes after first line therapy
  - 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

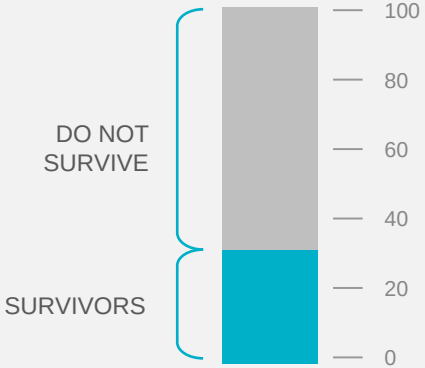
Tipifarnib Has the Potential to be the First Small Molecule Targeted Therapy for HNSCC Patients

Globally, **~885,000** people develop head and neck cancer annually and **~450,000** die of HNSCC each year[1]

60,000+ cases of HNSCC per year in the **U.S.**[2]

Head and neck squamous cell carcinoma ranks as the **7th leading cancer worldwide**[3]

Only **~1/3 of patients** with advanced diagnosis **survive 5 years**[4]



Outcomes with currently available therapies (including I-O therapy) are poor[5]

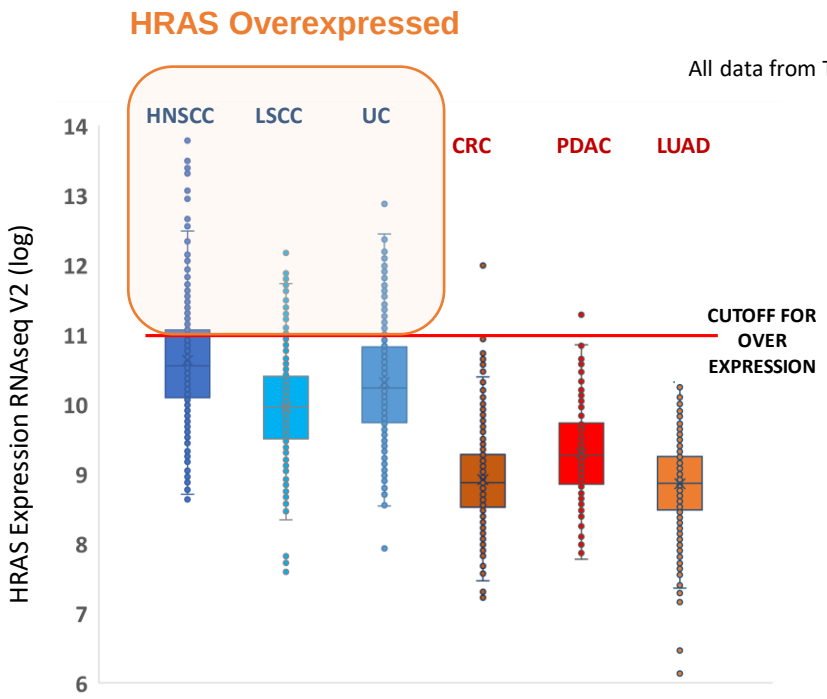
OS	PFS	ORR
First line: 10-15 mo Second line: 5-8 mo	First line: 3-5 mo Second line: 2-3 mo	First line: 20-36% Second line: 13-16%

[1] Bray et al. CA Cancer J Clin. 2018;68(6):394-424
[2] Cramer et al. Nat Rev Clin Oncol. 2019 Nov;16(11):669-683 | ACS Cancer Facts and Figures 2020
[3] Siegel et al. CA Cancer J Clin. 2020;70(1):7-30
[4] National Cancer Institute. Introduction to head & neck cancer. https://training.seer.cancer.gov/head-neck/intro/. Accessed March 4, 2019
[5] N Engl J Med. 2008 Sep 11;359(11):1116-27 | Keytruda & Opdivo package inserts | J Clin Oncol. 2007 Jun 1;25(16):2171-7 | J Clin Oncol. 2012 30:15_suppl, 5574-5574

EXPANSION OPPORTUNITIES FOR TIPIFARNIB IN HRAS AND PI3Kα DEPENDENT HNSCC

HRAS Dependent Tumors Represent a Significant Subset of HNSCC with Distinct Biology

- Several independent studies cluster HRAS mutant HNSCCs as part of a larger subset[1]
- TCGA cohort shows overexpression of HRAS gene in 25-30% of HNSCC[2]
- Average HRAS expression in HNSCC is 5-10x higher than in other tumor types
- Together with HRAS mutant tumors, HRAS-overexpressing HNSCC may represent a significant subset of **HRAS dependent tumors** with distinct biology that is targeted by tipifarnib



All data from TCGA PanCancer Atlas studies

	Mean	SEM	z-score*
HNSCC	10.63	0.04	24.96
LSCC	9.96	0.03	19.75
UC	10.31	0.04	21.56
CRC	8.92	0.03	NA
PDAC	9.30	0.05	6.37
LUAD	8.86	0.03	1.13

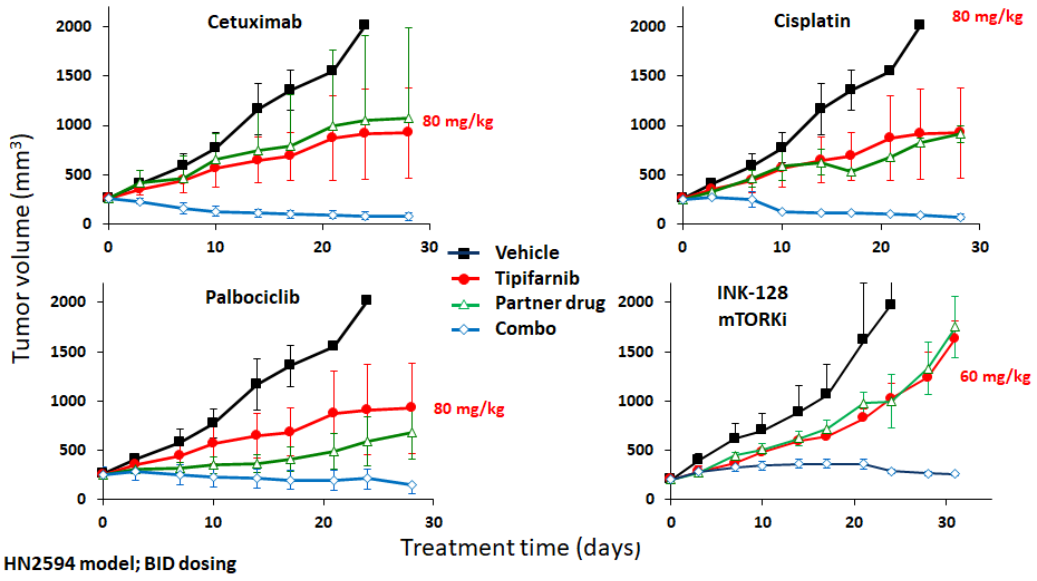
* vs. CRC

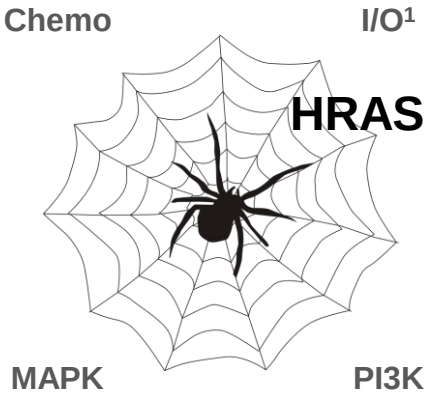
	# > CUTOFF	TOTAL	% HIGH
HNSCC	146	482	30.3
LSCC	39	461	8.5
UC	78	306	25.5
CRC	1	520	0.2
PDAC	2	163	1.2
LUAD	1	498	0.2

[1] Campbell et al. (2018), Cell Rep. 23:194; Su et al. (2017), Theranostics, 7:1088;
[2] International Cancer Genome Consortium (2013), Nat. Commun.| 4:2873

HRAS is a Central Resistance Mechanism to Other Therapies in PDX Models of HRAS Dependent HNSCC

- Tipifarnib displays additive or synergistic anti-tumor activity with a range of other drugs in HRAS-overexpressing patient-derived xenograft (PDX) models

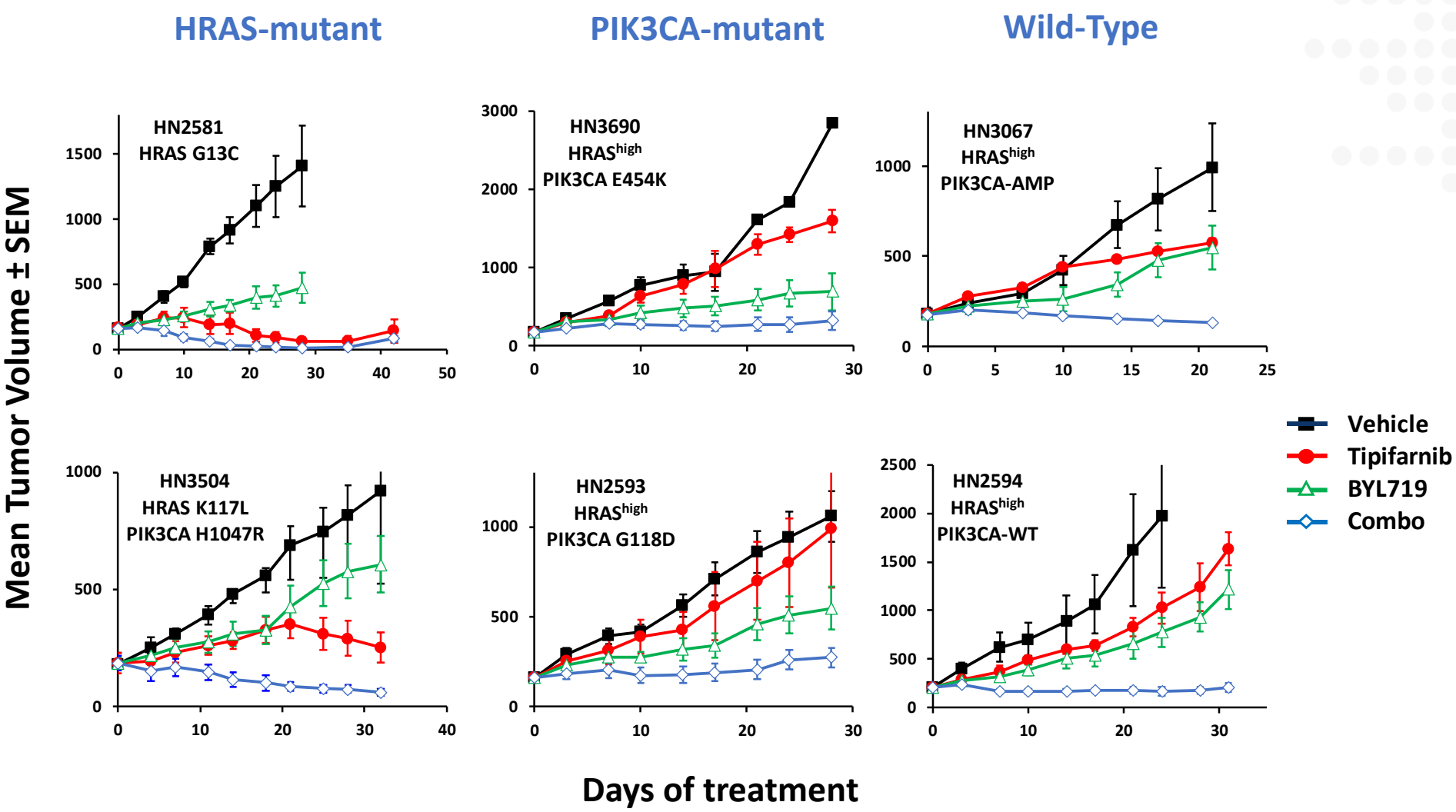




- HRAS represents a key node at the center of HNSCC tumor biology, driving resistance to other therapies and reinforcing the potential for combination strategies with tipifarnib

[1] HRAS likely drives immunosuppression in HNSCC, and tipifarnib may also sensitize to immunotherapy via inhibition of CXCL12 production by activated carcinoma-associated fibroblasts

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)

Combinations of Tipifarnib and PI3Kα inhibitors Have Broad 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 25-50% 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

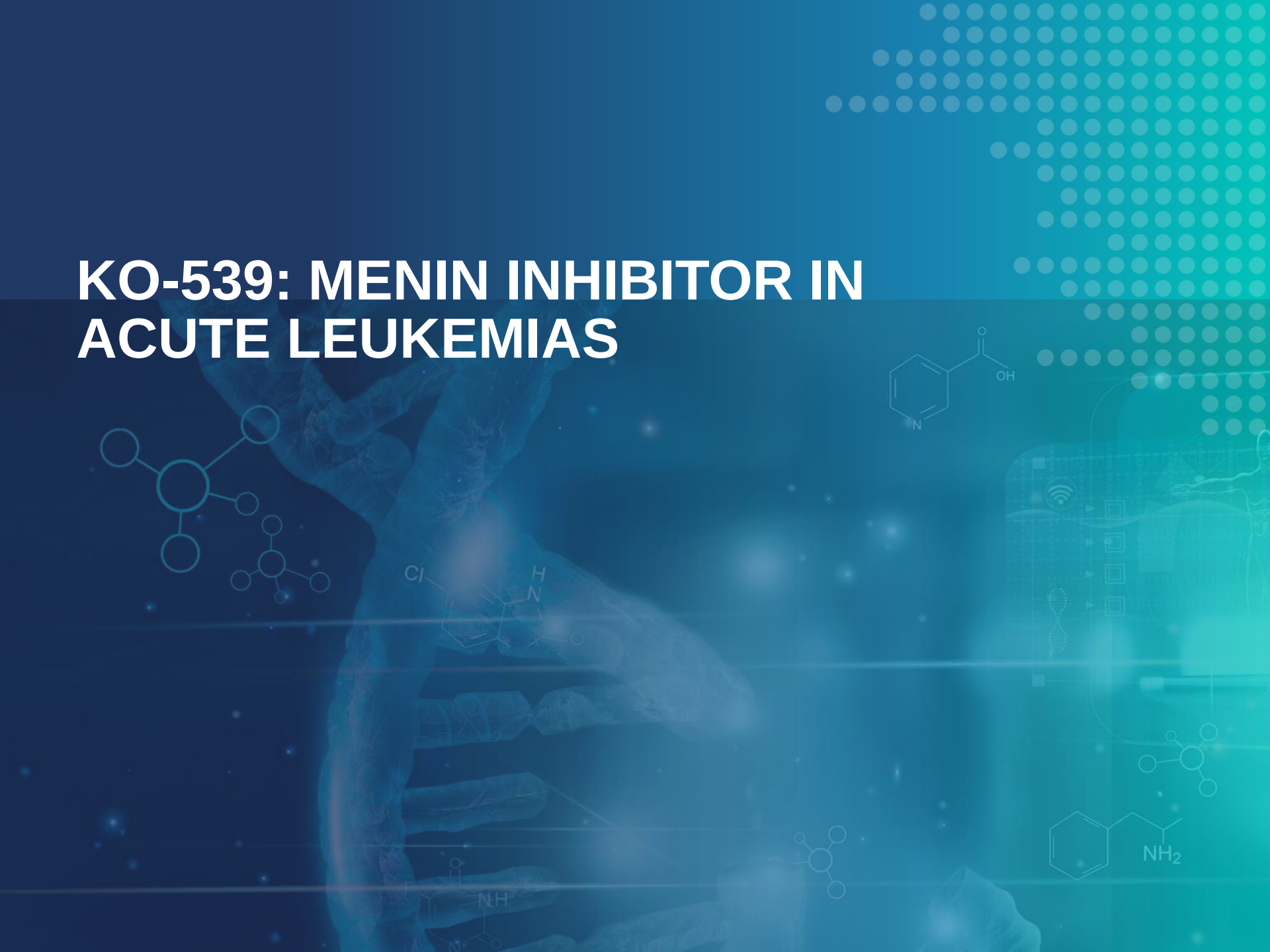
Tipifarnib / FTI Patent Exclusivity

Layered patent strategy provides patent exclusivity to 2036 in major markets

Proprietary Biomarkers and Methods	• Multiple issued U.S. patents covering biomarker-guided indications provide patent exclusivity to 2036 • Additional patent applications pending in the U.S. and foreign countries for tipifarnib in other biomarkers and disease indications • U.S. patents cover use of “any farnesyl transferase inhibitor”
Combinations	• Patents cover combinations of tipifarnib with other agents (*e.g.*, I/O) • Additional patents possible with specific agents, doses, schedules, etc.
Novel FTI Program	• Researching FTIs with superior properties to tipifarnib • Expect composition of matter IP on new discoveries

Broadest claims cover *any FTI*, providing Kura an opportunity to have an exclusive leadership position for FTIs in oncology

KO-539: MENIN INHIBITOR IN ACUTE LEUKEMIAS



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% of AML

Preliminary data from KOMET-001 Phase 1/2A dose-escalation study accepted for oral presentation at ASH on December 5, 2020

Focused monotherapy development strategy

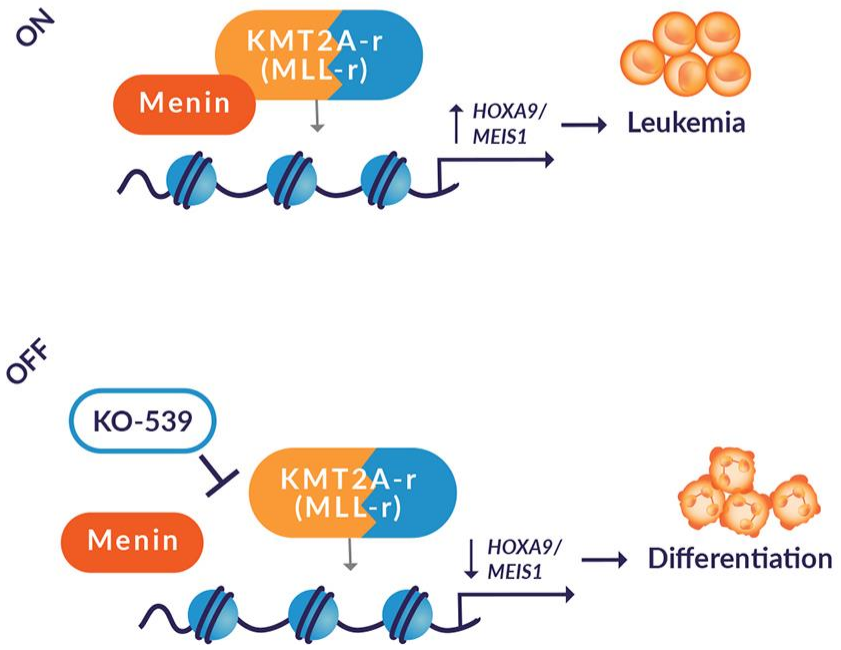
- KMT2A(MLL)-rearranged (5-10% of AML)
- NPM1 mutant (30% of AML)

Potential to combine with other targeted therapies and induction chemotherapy

Issued and pending COM patents provide worldwide coverage to 2036

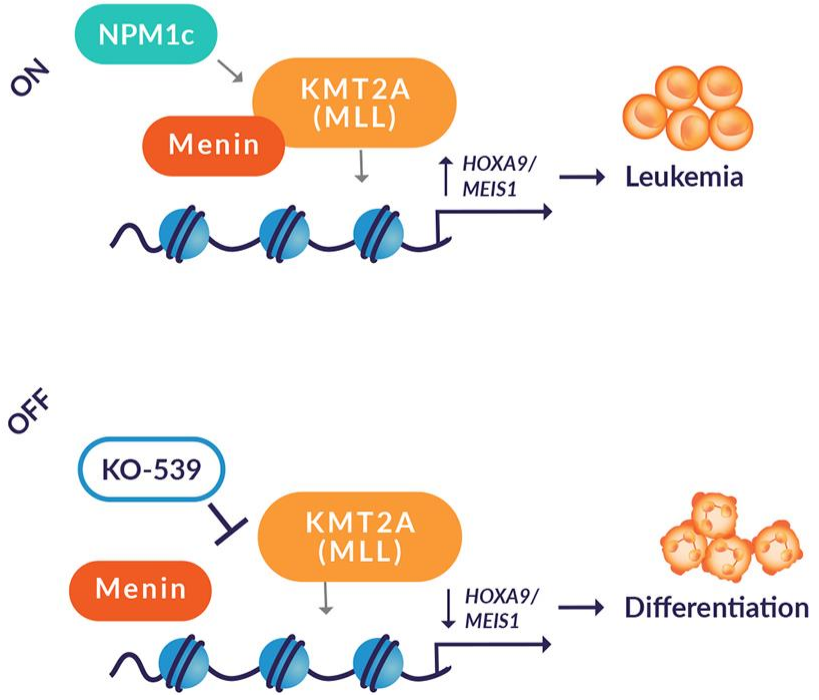
Targeting Menin-KMT2A(MLL) Interaction Provides Potential Therapeutic Intervention into Two Genetic Subsets of AML

KMT2A-r (MLL-r)



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

NPM1 Mutant AML

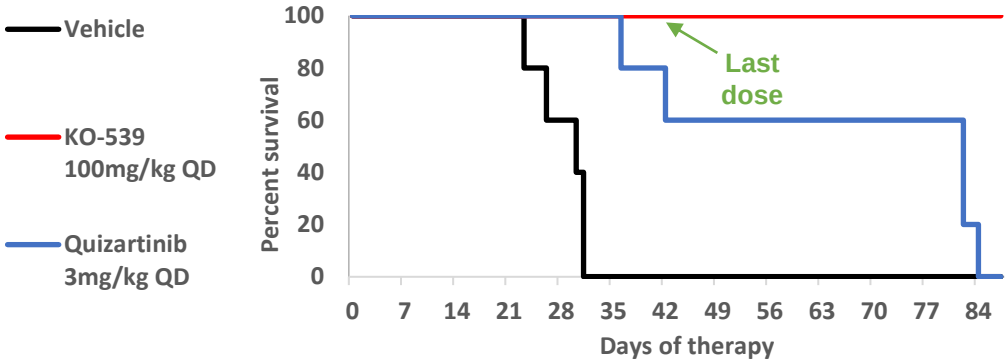


A central role for menin-KMT2A(MLL) interaction in epigenetic dysregulation in NPM1-mutant AML

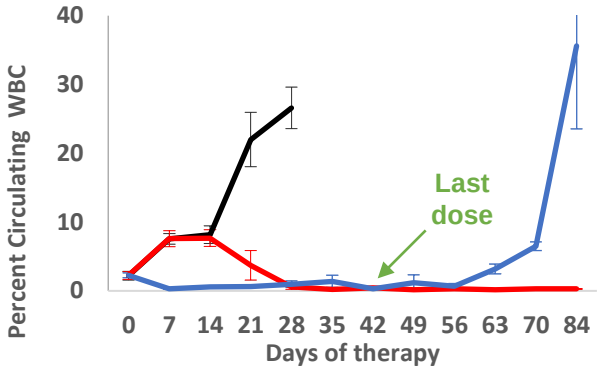
KO-539 Produces Lasting Complete Remissions in a NPM1 / DNMT3A / IDH2 / FLT3-Mutant AML Model

AM7577

Overall Survival

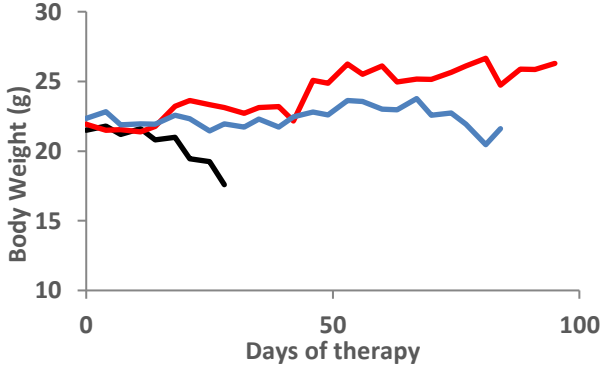


CD45+ Human AML Blasts



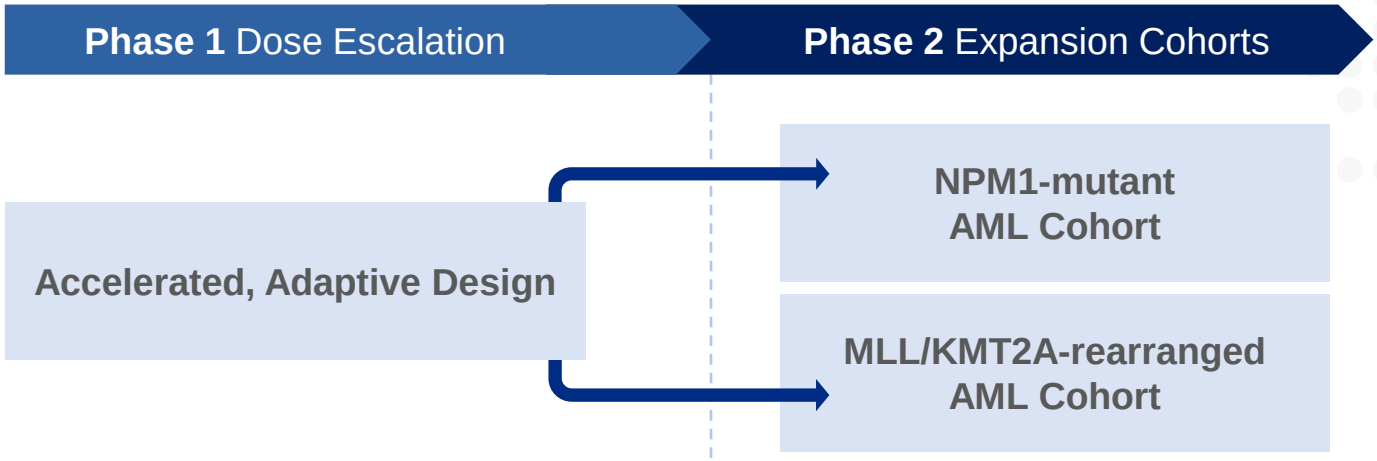
- 100% (10/10) of animals treated with single-agent KO-539 cleared their leukemia and became long-term survivors
- Tumor growth inhibition was durable – no leukemia was detectable in blood or bone marrow two months after cessation of dosing
- KO-539 was well tolerated at tested dose levels
- Comparator compound (FLT3 inhibitor) was initially active, but all animals eventually relapsed

Tolerability



KOMET-001: Phase 1/2A First-in-Human Study of KO-539 in Patients with Relapsed or Refractory AML

komet Trial 001



OBJECTIVES

- Determine recommended Phase 2 dose and/or MTD
- Safety and tolerability
- Pharmacokinetics
- Early evidence of antitumor activity

- Safety and tolerability
- Antitumor activity

KOMET: **K**ura **O**ncology **M**enin-MLL **T**rial

KOMET-001 Update: Abstract Published for ASH 2020

- Six patients with relapsed/refractory AML enrolled, three evaluable as of data cutoff on August 10, 2020
- Dose escalation began with single-patient cohorts at 50 mg po qd in 28-day cycles, advanced to 3 + 3 design at 200 mg dose
- Evidence of biologic activity observed at first three dose levels, including a CR in patient with SETD2/RUNX1 co-mutations at 100 mg dose
- KO-539 has been well tolerated and with a manageable safety profile to date
  - No dose-limiting toxicities, dose interruptions or discontinuations due to drug-related AEs
  - No treatment-related deaths, two patients discontinued treatment due to disease progression
- Peak drug concentrations attained between 2-3 hours after daily oral dosing with elimination half-life > 24 hours
- Updated safety, PK and efficacy data to be presented at ASH on December 5, 2020

Preliminary Data for KO-539 at ASH

Preliminary Data on a Phase 1/2A First in Human Study of the Menin-KMT2A (MLL) Inhibitor KO-539 in Patients with Relapsed or Refractory Acute Myeloid Leukemia

Publication Number: 115

Session Name: 616. Acute Myeloid Leukemia: Novel Therapy, excluding Transplantation: Novel promising therapies for relapsed/refractory AML

Session Date: Saturday, December 5, 2020

Session Time: 9:30 am - 11:00 am PT

Presentation Time: 10:30 am PT

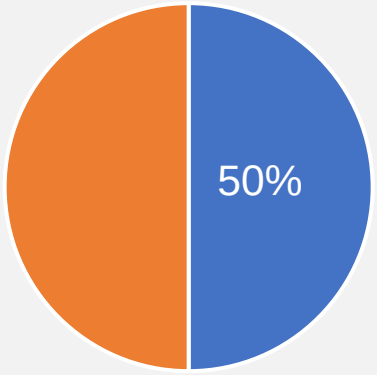
Kura Virtual Investor Event

Date: Saturday, December 5, 2020

Time: 11:00 am - 12:00 pm PT (immediately following oral session)

Featuring two investigators from KOMET-001, Kura's Phase 1/2A study of KO-539 in patients with relapsed or refractory AML

NPM1-Mutant and KMT2A(MLL)-Rearranged AML are Challenging Diseases Associated with Poor Outcomes



>50% of AML patients who initially achieve a complete remission after induction therapy **relapse within 1-3 years**[1]

Only **~1 in 10** relapsed / refractory AML patients survive **five years**[2]

NPM1-Mutant AML

Estimated **6,000** new cases in the U.S. per year[3]

(~30% of AML)

Known co-mutations confer **worse prognosis**[4] and represent rational combination approaches

KMT2A(MLL)-Rearranged AML

Estimated **1,000-2,000** new cases in the U.S. per year[3]

(5-10% of AML)

NCCN guidelines denote that MLL-r confers **poor prognosis**[5]

[1] Wiese *et al*. Am J Manag Care. 2018 Aug;24(16 Suppl):S347-S355
[2] Breems *et al*. J Clin Oncol. 2005 Mar 20;23(9):1969-78
[3] SEER statistics for AML in the US, accessed April 2020
[4] Döhner *et al*. Blood. 2017 Jan 26;129(4):424-447
[5] NCCN. AML Guidelines (version 3.2020). Accessed May 2020

Investment Highlights

Targeted Oncology

Advancing two wholly owned, targeted oncology drug candidates using a precision medicine approach; fast-to-market strategy

Proprietary Pipeline

Tipifarnib: Farnesyl transferase inhibitor

- Registration-directed trial in HRAS mutant head and neck squamous cell carcinoma (HNSCC) ongoing
- Opportunity to expand to HRAS and PI3Kα dependent tumors
- Multiple clinical proof-of-concept studies support significant lifecycle expansion opportunities

KO-539: Menin inhibitor

- Potent and selective inhibitor of the menin-KMT2A(MLL) protein-protein interaction
- Potential to target ~35% of acute myeloid leukemia (AML)
- Preliminary data trial accepted for oral presentation at ASH 2020

Strong Financials

$325.4 million in cash* provides runway into 2023

* Cash, cash equivalents and short-term investments as of September 30, 2020

KURA ONCOLOGY

DEVELOPING PRECISION MEDICINES FOR THE TREATMENT OF CANCER

Stifel Healthcare Conference – November 16, 2020