KURA ONCOLOGY

DEVELOPING PRECISION MEDICINES FOR THE TREATMENT OF CANCER

Corporate Presentation – February 2021

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,” “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

KO-539

- 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
- Continuing in Phase 1 dose escalation, Phase 1 expansion cohorts upcoming

Tipifarnib

- Breakthrough Therapy* and Fast Track Designations from FDA
- Registration-directed trial in HRAS mutant HNSCC ongoing
- Opportunity to expand to HRAS and PI3Kα dependent tumors

Next-Generation Farnesyl Transferase Inhibitor

- Nomination of development candidate expected in mid-2021

Strong Financials

$633.3 million in cash** provides runway into 2024

* For the treatment of patients with recurrent or metastatic HRAS mutant HNSCC with variant allele frequency ≥ 20% after disease progression on platinum-based chemotherapy

** Cash, cash equivalents and short-term investments as of December 31, 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
Chairman and Chief Executive Officer, 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

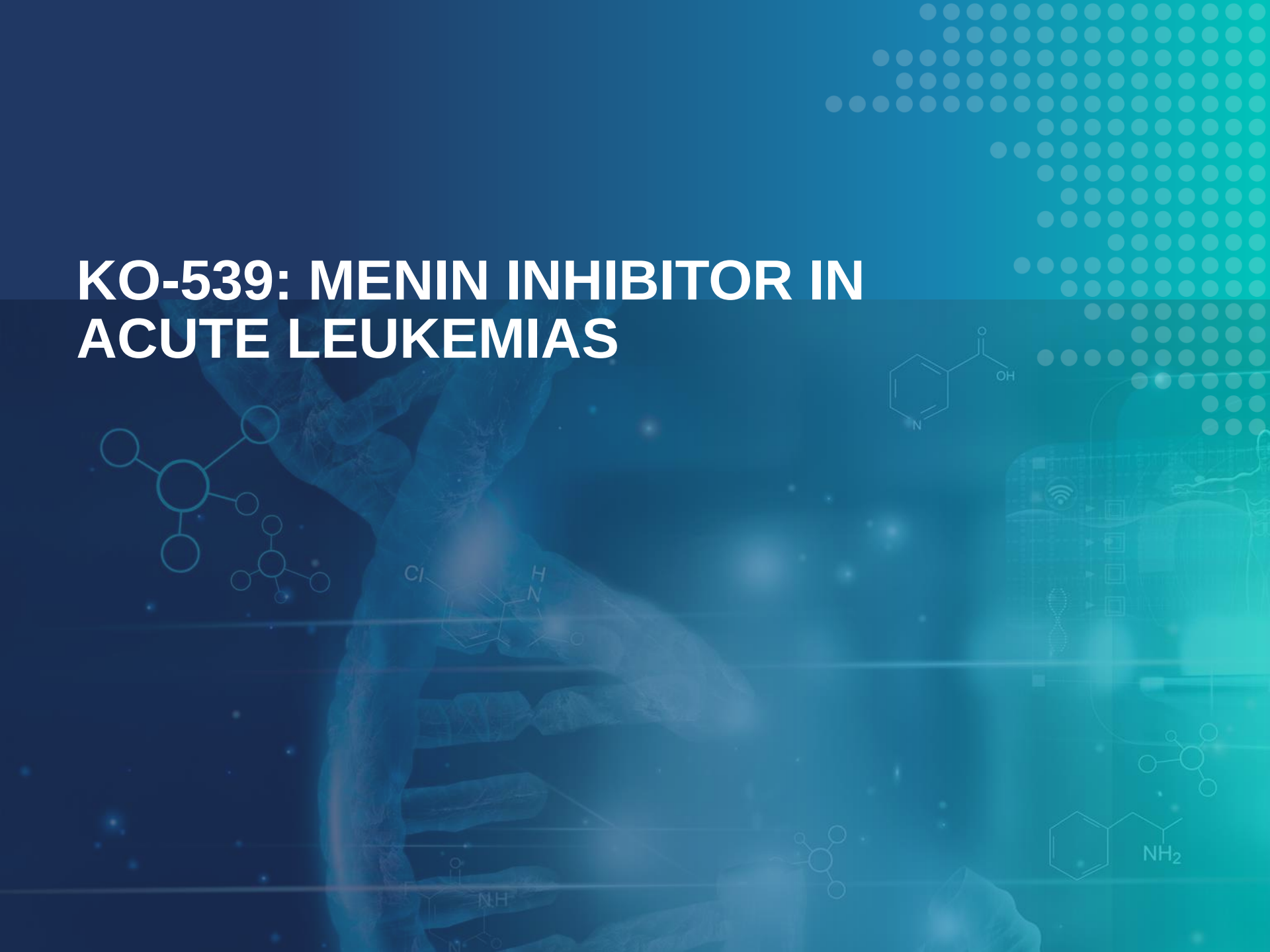
Mary Szela
President and CEO, TriSalus Life Sciences

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

Drug Candidate Pipeline

Program	Preclinical	Phase 1	Phase 2	Registration Directed
KO-539 Menin Inhibitor	**Acute Myeloid Leukemia (AML)** • *Enrollment in Phase 1 expansion cohorts expected to begin in mid-2021*			
Tipifarnib Farnesyl Transferase Inhibitor	**HRASm Head & Neck Squamous Cell Carcinoma (HNSCC)** • *Enrollment in AIM-HN registration-directed trial ongoing* **PI3Kα / HRAS dependent HNSCC** • *Initiation of PI3Kα inhibitor combination study expected in 2H 2021*			
Next-Generation Farnesyl Transferase Inhibitor	**Solid tumors** • *Nomination of development candidate expected in mid-2021*			

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% or more of AML

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

Focused monotherapy development strategy in multiple genetic subtypes:

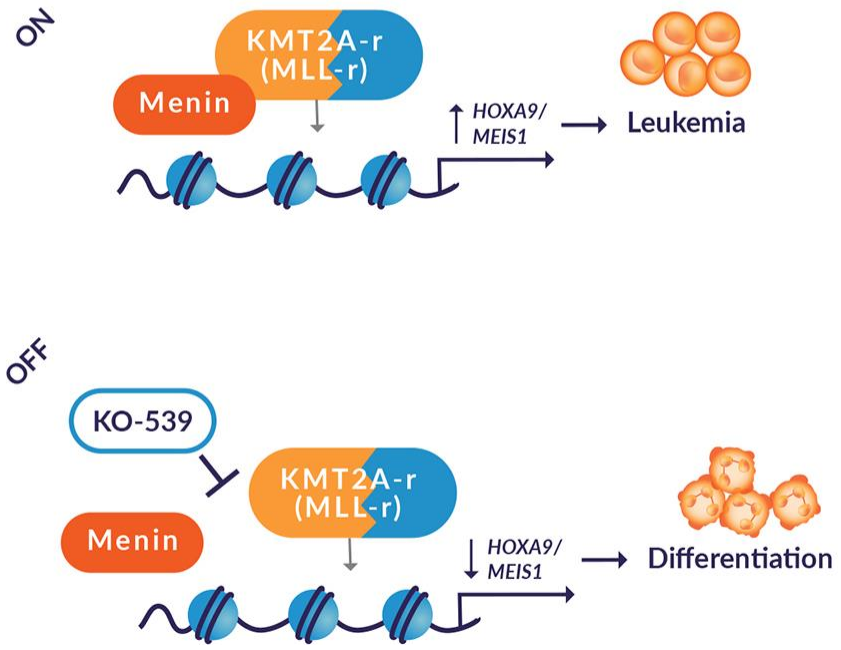
- KMT2A(MLL) rearranged (5-10% of AML)
- NPM1 mutant (~30% of AML)
- Other genetic subtypes (*e.g.,* SETD2/RUNX1-mutant AML)

Potential to combine with other targeted therapies and induction chemotherapy

Issued and pending COM patents provide worldwide coverage to 2036

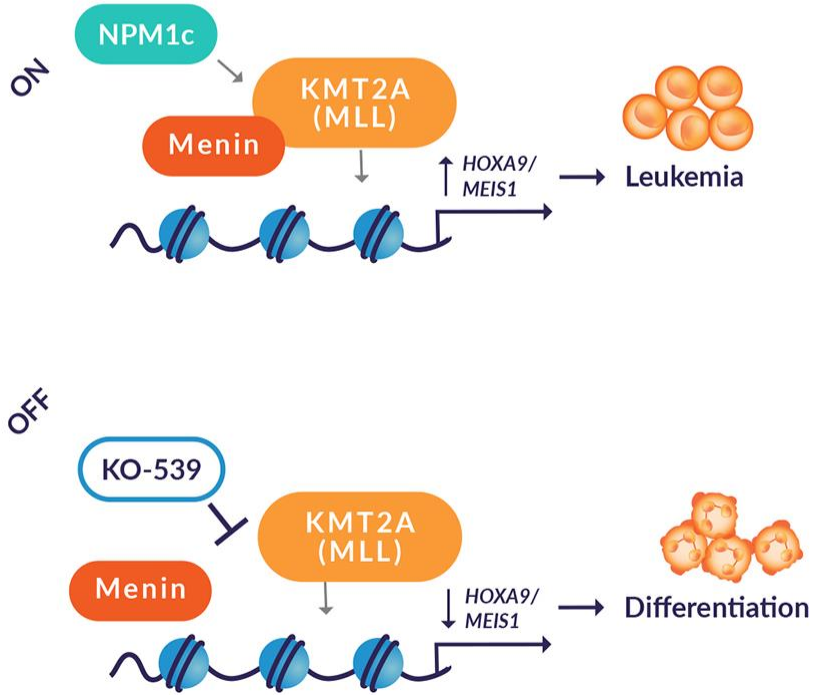
Targeting Menin-KMT2A(MLL) Interaction Provides Potential Therapeutic Intervention into Two Genetic Subsets of AML

KMT2A-r (MLL-r)



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

NPM1 Mutant AML



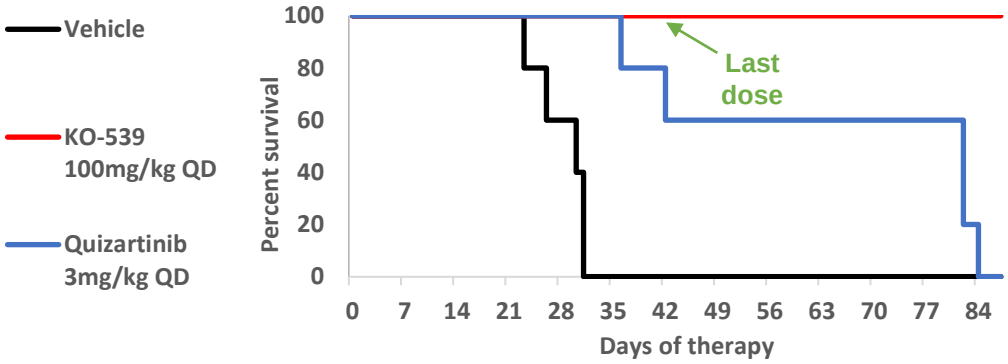
A central role for menin-KMT2A(MLL) interaction in epigenetic dysregulation in NPM1-mutant AML

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

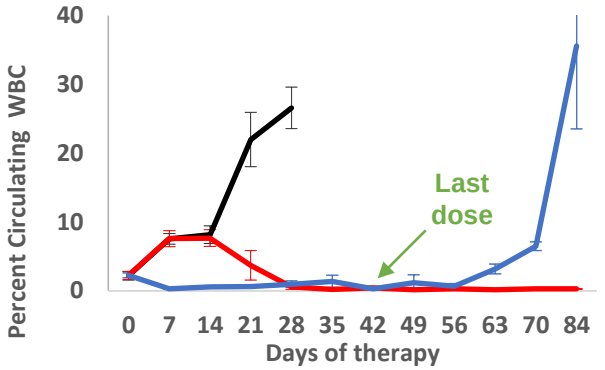
KO-539 Produces Lasting Complete Remissions in a NPM1 / DNMT3A / IDH2 / FLT3-Mutant AML Model

AM7577

Overall Survival

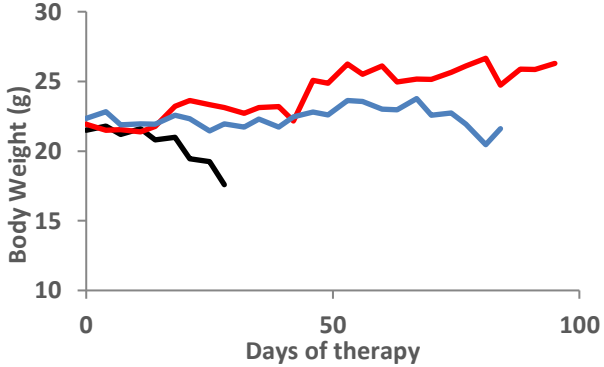


CD45+ Human AML Blasts



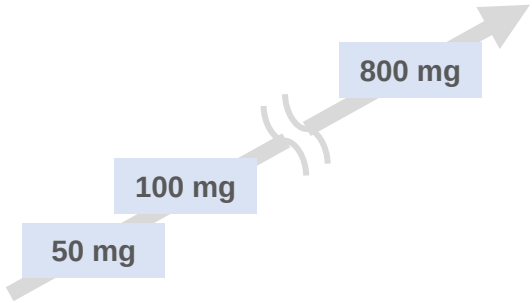
- 100% (10/10) of animals treated with single-agent KO-539 cleared their leukemia and became long-term survivors
- Tumor growth inhibition was durable – no leukemia was detectable in blood or bone marrow two months after cessation of dosing
- KO-539 was well tolerated at tested dose levels
- Comparator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

Tolerability



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

komet Trial 001



OBJECTIVES

- **Dose escalation in all comer patients to establish MTD and/or RP2D**
  - Safety and tolerability
  - Pharmacokinetics
  - Early evidence of antitumor activity
- **Enroll two genetically enriched cohorts to optimize Phase 2 dose**
  - Safety and tolerability
  - Pharmacokinetics
  - Anti-tumor efficacy
- **Enroll genetically enriched cohorts at RP2D for registration**
  - Primary endpoint: CR/CR_h
  - Safety and tolerability
  - Other secondary and exploratory endpoints

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

Continuous Daily Dosing of KO-539 Has Been Well-Tolerated with a Manageable Safety Profile

- 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)

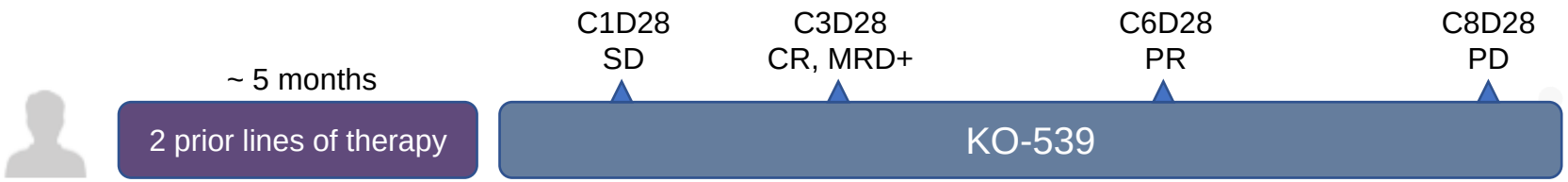
KO-539 Demonstrates Encouraging Early Clinical Activity

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

KOMET-001 (n=12)				
Dose	Mutational Profile	CYP3A Inhibitor	# of Prior Regimens	Clinical Activity
400 mg	*RUNX1, SRSF2, ASXL1, TET2, STAG2, BCOR, PTPN11*	Yes	3	Decreased peripheral blasts
	EZH2, DNMT3A, FAT3, RET	Yes	3	Progressive disease
	NPM1	No	2	Not efficacy evaluable at time of data cut
	DNMT3A, CUX1, ASXL1, IDH2, CBL, U2AF1, RUNX1	Yes	5	Not efficacy evaluable at time of data cut
200 mg	*NPM1, DNMT3A, KMT2D*	Yes	7	Complete remission, MRD-
	NPM1, FLT3-ITD, TET2, CUX1	Yes	4	Morphological leukemia-free state
	U2AF1, TET2, p53, DNMT3A, PTPN11	No	4	Stable disease
	IDH2, SRSF2, DNMT3A, CBL	Yes	3	Progressive disease
	TP53, PICALM (MLLT10)	Yes	3	Not efficacy evaluable
	KMT2A-r	Yes	4	Not efficacy evaluable
100 mg	*SETD2, RUNX1*	Yes	2	Complete remission, MRD+
50 mg	*KMT2A*-r	Yes	2	Decreasing hydrea requirement

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

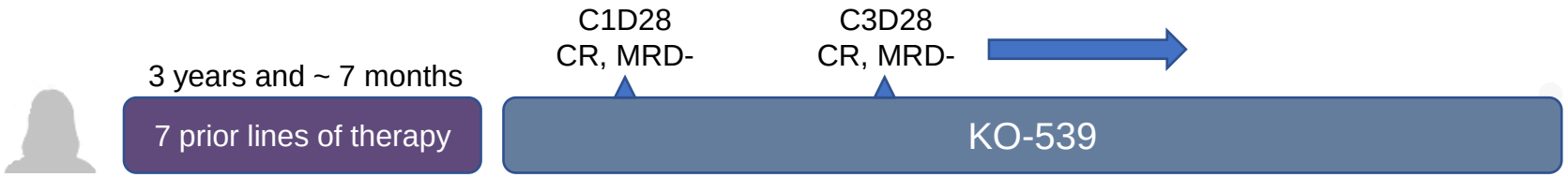
Case Study – *SETD2, RUNX1* Mutant AML



Patient Characteristics	
Demographics	69-year-old male
Mutational profile	*SETD2*, *RUNX1*
Prior lines of therapies	2 (decitabine; CD33/CD3 bispecific antibody)
KO-539 dose	100 mg, escalated to 200 mg during cycle 7
# of KO-539 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



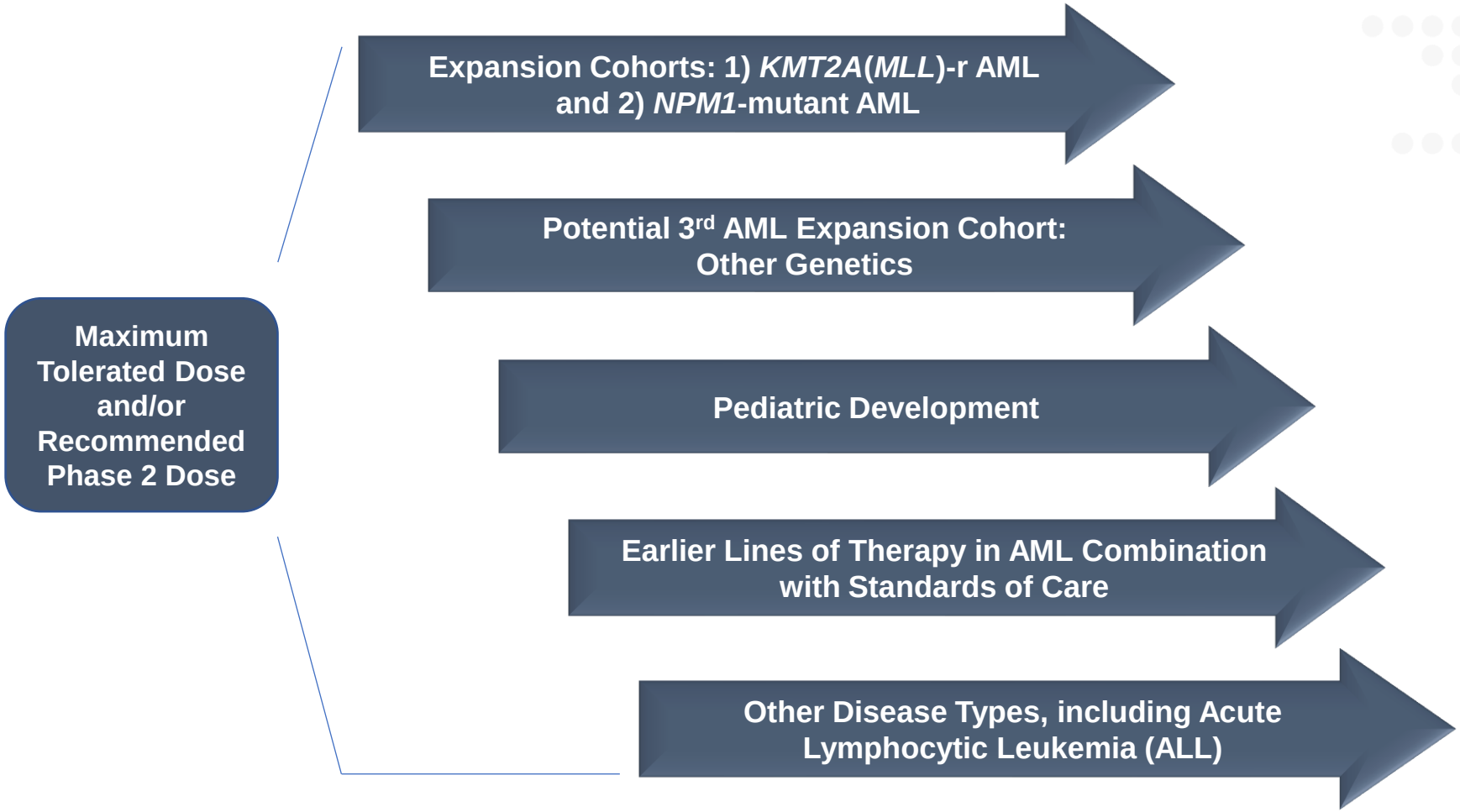
Patient Characteristics	
Demographics	44-year-old female
Mutational profile	*NPM1*, *DNMT3A*, *KMT2D*, *FLT3-TKD*
Prior lines of therapies	7 (incl. decitabine+venetoclax, gilteritinib, itacitinib, fludarabine, bortezomib)
KO-539 dose	200 mg
# of KO-539 cycles	3+ (on treatment)
CYP3A4 inhibitor	Yes (posaconazole)
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

Summary of Preliminary Data from KOMET-001

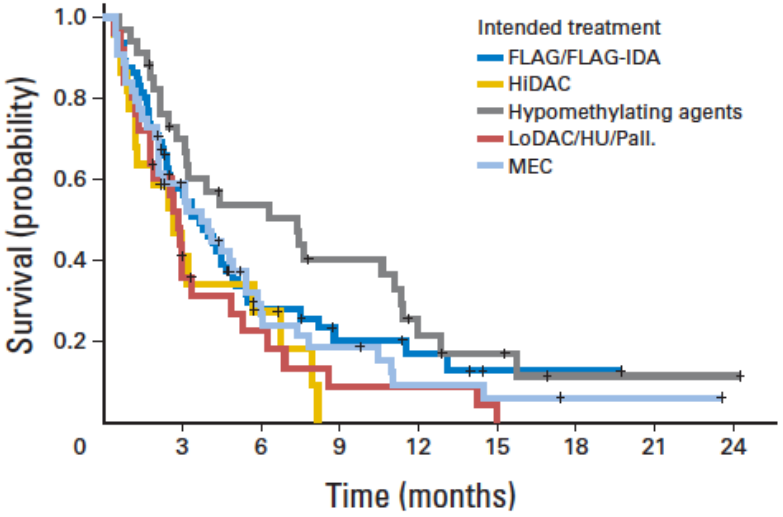
- KO-539 is a potent and selective inhibitor of the menin-KMT2A/MLL complex
- KO-539 has been well tolerated with a manageable safety profile to date
  - Observed toxicities appear to be reversible and manageable
  - No evidence of QTc prolongation
- KO-539 demonstrates encouraging signs of clinical activity in multiple genetically defined subgroups of AML
- KO-539 pharmacokinetics and clinical activity do not appear to be affected by co-administration of a CYP3A4 inhibitor
- Continue to enroll patients in dose escalation; plan to initiate genetically enriched Phase 1 expansion cohorts in mid-2021

Multiple Expansion Opportunities in Acute Leukemias



Relapsed/Refractory AML is a Challenging Disease Associated with Poor Outcomes

Chemotherapy[1]



Targeted Therapies

Drug Name	AML Subset	Median OS
Enasidenib	*IDH2* mutant	9.3 mos[2]
Ivosidenib	*IDH1* mutant	8.8 mos[3]
Gemtuzumab ozogamicin	CD33+ AML	11.6 mos[4]
Gilteritinib	*FLT3* mutant	9.3 mos[5]
Quizartinib	*FLT3-ITD* mutant	6.2 mos[6]

Credit: Dr. Wang, Roswell Park Comprehensive Cancer Center

NPM1-Mutant AML

Estimated **6,000** new cases in the U.S. per year[7]

(~30% of AML)

Known co-mutations confer **worse prognosis**[8] and represent rational combination approaches

KMT2A(MLL)-Rearranged AML

Estimated **1,000-2,000** new cases in the U.S. per year[7]

(5-10% of AML)

NCCN guidelines denote that MLL-r confers **poor prognosis**[9]

[1] Roboz *et al*. J Clin Oncol. 2014 Jun 20;32(18):1919-26
[2] Stein *et al*. Blood. 2017 Aug 10;130(6):722-731
[3] DiNardo *et al*. N Engl J Med. 2018 Jun 21;378(25):2386-2398
[4] Taksin *et al*. Leukemia. 2007 Jan;21(1):66-71
[5] Perl *et al*. Engl J Med. 2019 Oct 31;381(18):1728-1740
[6] Cortes *et al*. Lancet Oncol. 2019 Jul;20(7):984-997
[7] SEER statistics for AML in the US, accessed April 2020
[8] Döhner *et al*. Blood. 2017 Jan 26;129(4):424-447
[9] NCCN. AML Guidelines (version 3.2020). Accessed May 2020

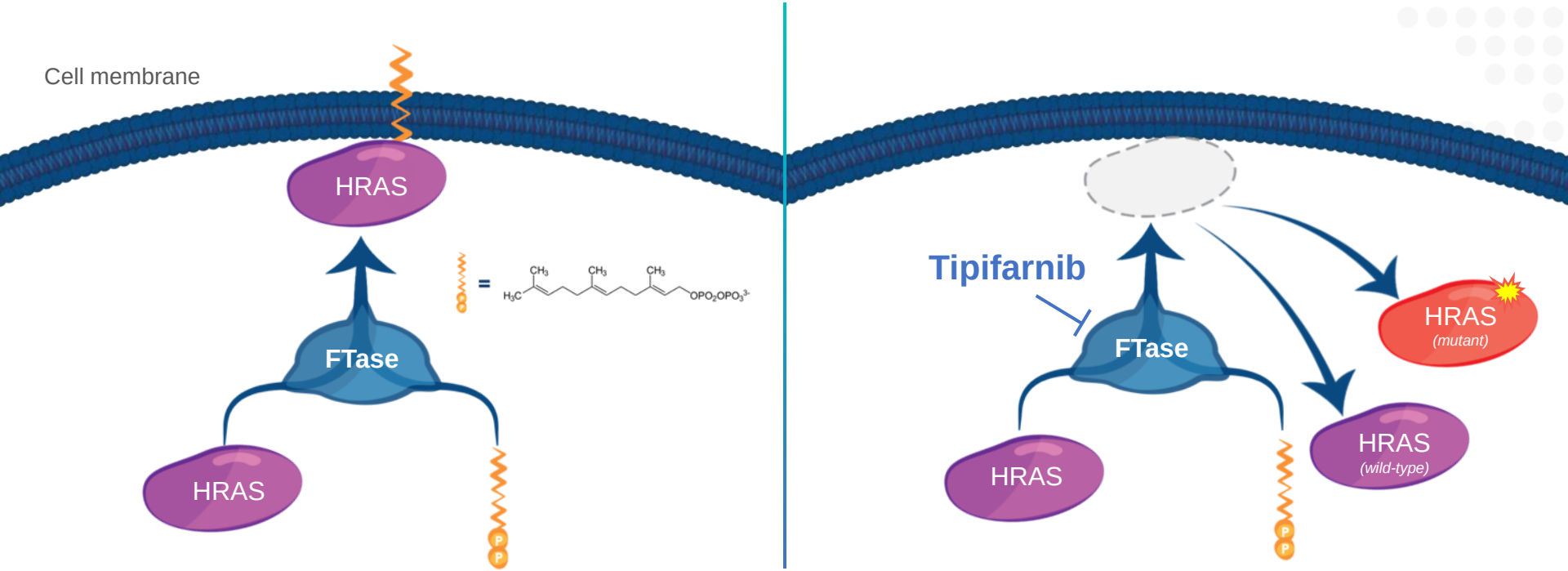
TIPIFARNIB IN HRAS MUTANT SOLID TUMORS

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
- Breakthrough Therapy* and Fast Track Designations from FDA; 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

* For the treatment of patients with recurrent or metastatic HRAS mutant HNSCC with variant allele frequency ≥ 20% after disease progression on platinum-based chemotherapy

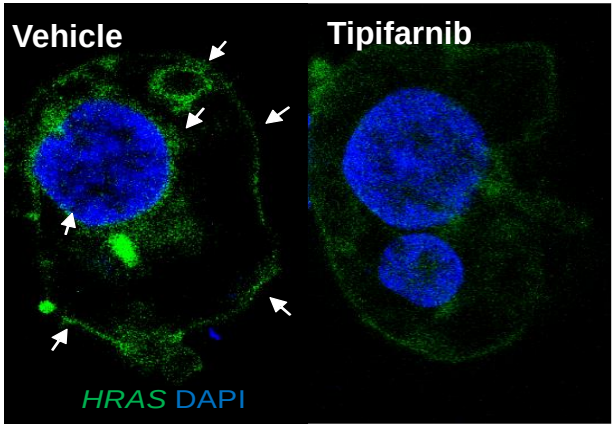
Tipifarnib Inhibits Farnesylation – An Essential Modification Required for HRAS Activity



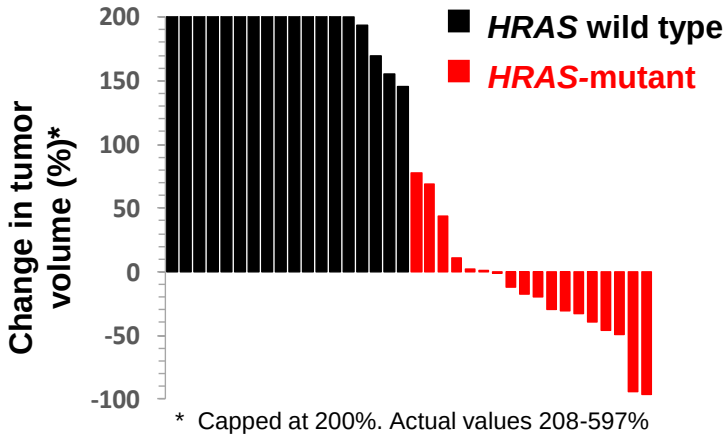
- Tipifarnib inhibits farnesylation and signaling activity of the HRAS oncoprotein
- Farnesylation is essential for HRAS signal transduction activity
- HRAS mutations drive proliferation and resistance mechanisms in solid tumors
- Incidence of HRAS mutations in HNSCC is approximately 4-8% and varies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

Tipifarnib Displays Robust, Selective Activity in HRAS Mutant HNSCC Models

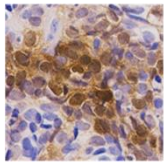
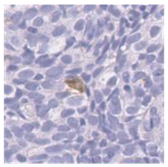
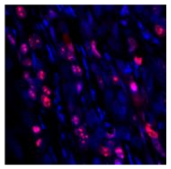
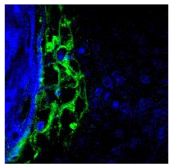
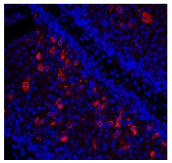
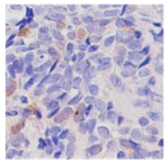
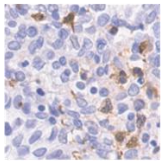
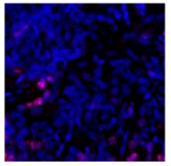
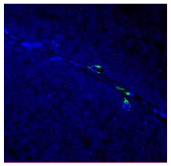
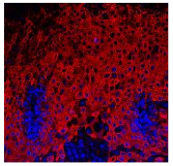
HRAS membrane displacement



Antitumor activity in PDX models



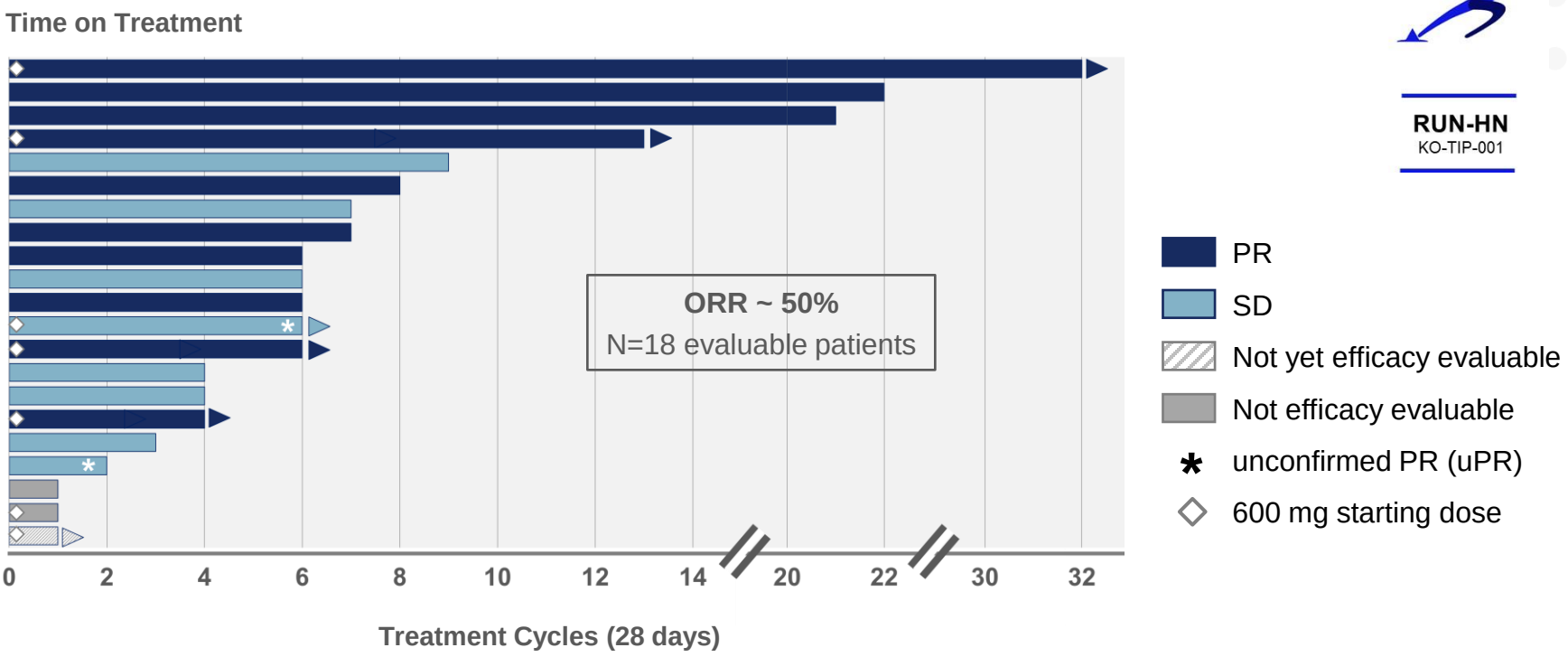
* Capped at 200%. Actual values 208-597%

	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



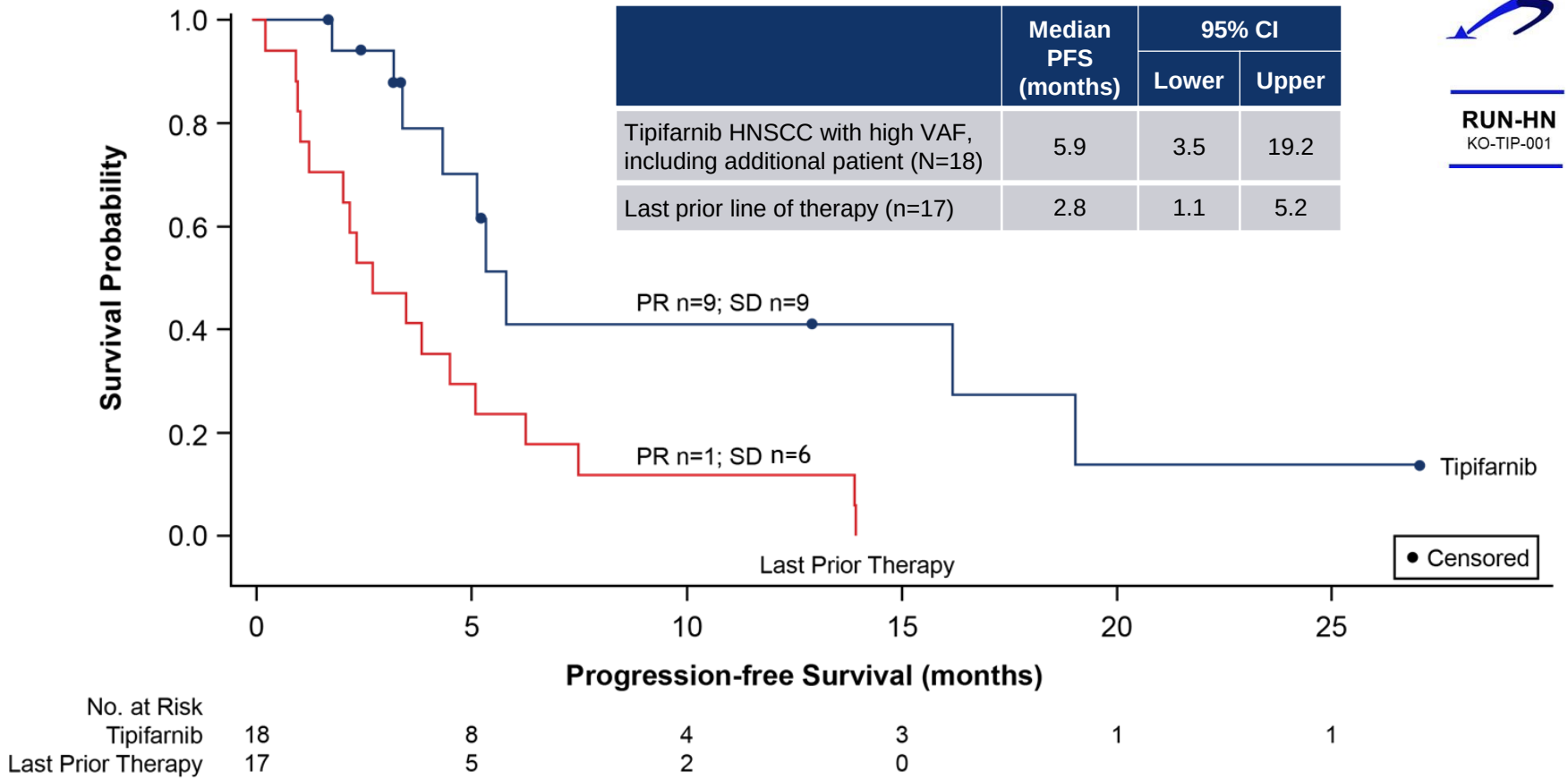
Ho *et al*. AACR-NCI-EORTC 2019 #384 (preliminary data as of 10/17/19)
Efficacy-evaluable patients with HRAS mutant variant allele frequency (VAF) ≥ 20% and serum albumin ≥ 3.5 g/dL, or HRAS VAF ≥ 35%
One patient treated off-protocol through compassionate use

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

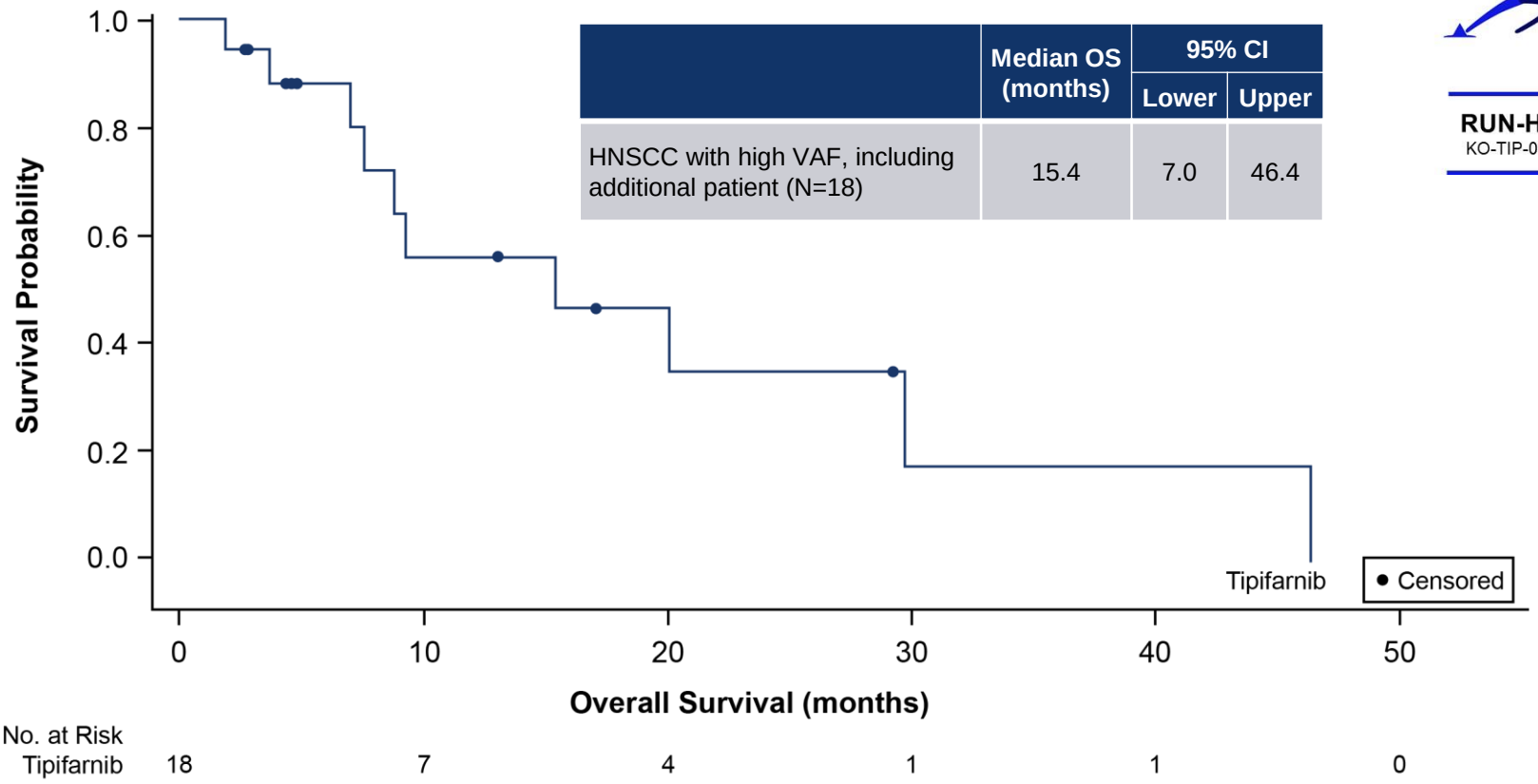
RUN-HN
KO-TIP-001



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

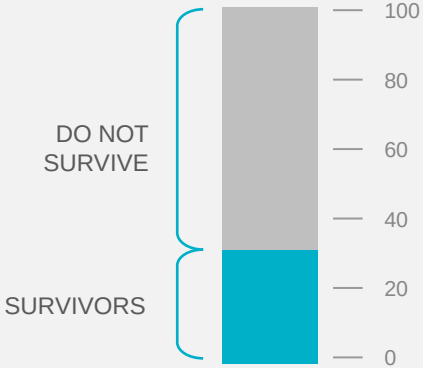
Tipifarnib Has the Potential to be the First Small Molecule Targeted Therapy for HNSCC Patients

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]

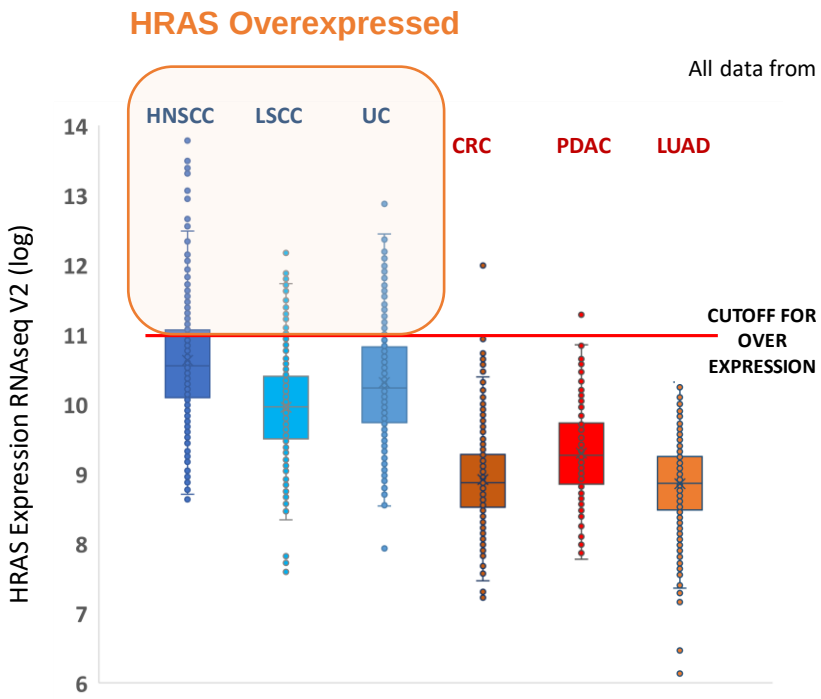
OS	PFS	ORR
First line: 10-15 mo Second line: 5-8 mo	First line: 3-5 mo Second line: 2-3 mo	First line: 20-36% Second line: 13-16%

[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

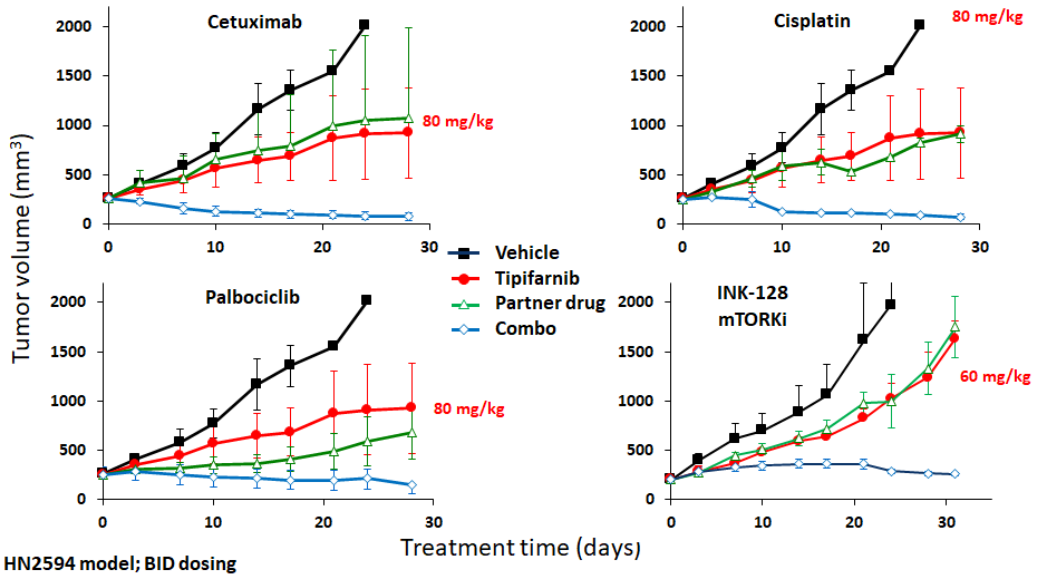
* vs. CRC

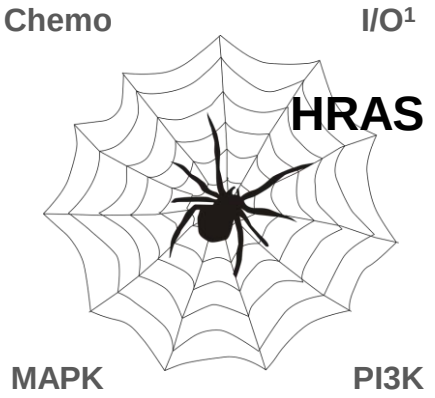
	# > CUTOFF	TOTAL	% HIGH
HNSCC	146	482	30.3
LSCC	39	461	8.5
UC	78	306	25.5
CRC	1	520	0.2
PDAC	2	163	1.2
LUAD	1	498	0.2

[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

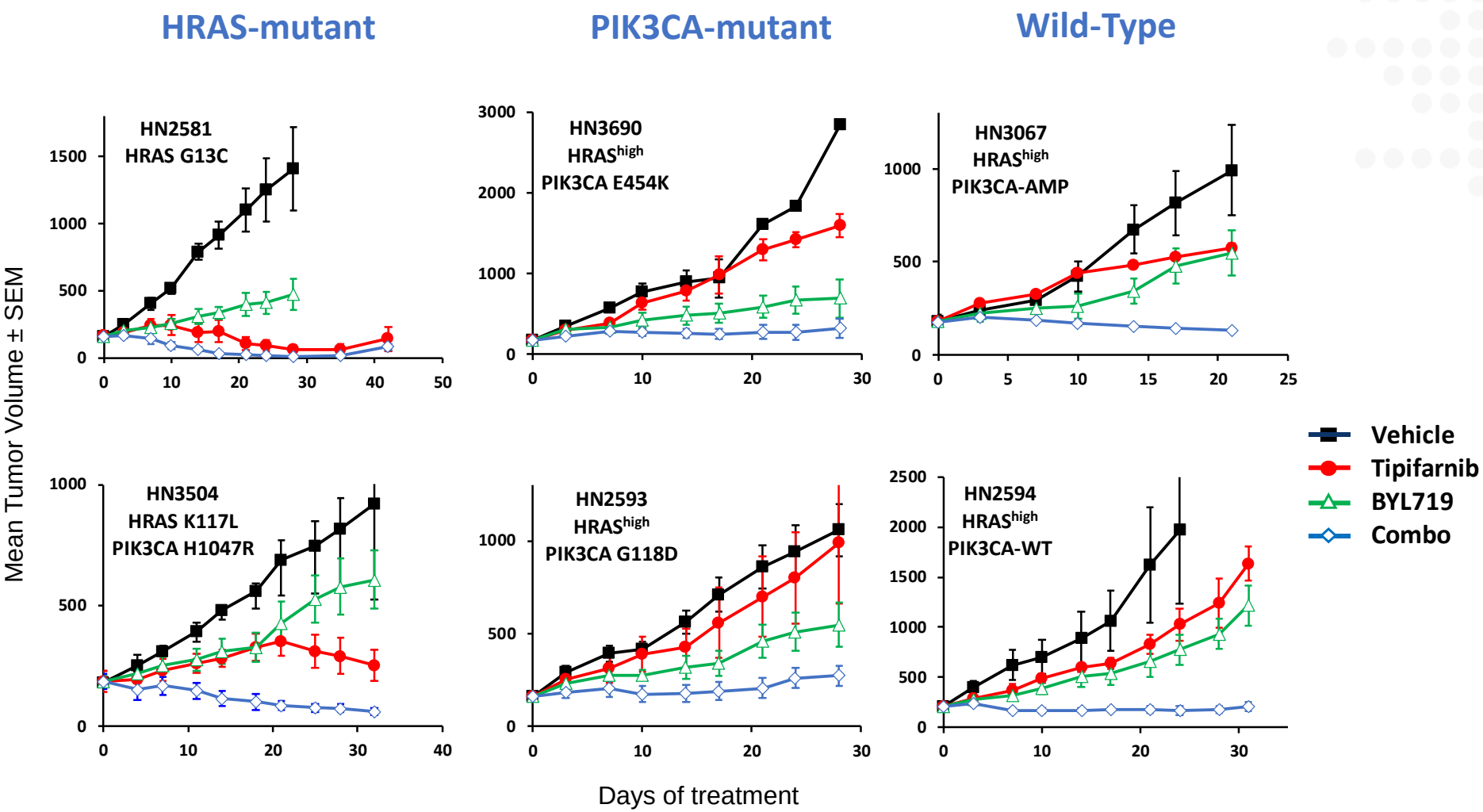




- HRAS represents a key node at the center of HNSCC tumor biology, driving resistance to other therapies and reinforcing the potential for combination strategies with tipifarnib

[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
- Plan to initiate Phase 1/2 proof-of-concept study in combination with PI3Kα inhibitor in second half of 2021

[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

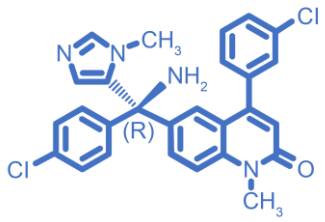
Tipifarnib / FTI Patent Exclusivity

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

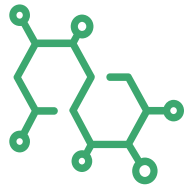
Next-Generation Farnesyl Transferase Inhibitor (FTI)

Nomination of development candidate for IND-enabling studies expected in mid-2021



Tipifarnib

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



Next-Generation FTI

Potency: Comparable
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 comparable potency and selectivity as well as improved pharmacokinetic and physicochemical properties
- Intend to direct next-generation FTI at new biology and larger disease indications
- Expect to nominate development candidate for IND-enabling studies in mid-2021

Forecasted Milestones & Financial Highlights

Program	Milestone	Status
KO-539 **Menin Inhibitor**	Initiation of Phase 1 expansion cohorts	mid-2021
	Additional Phase 1 data from KOMET-001	2H 2021
Tipifarnib **Farnesyl Transferase Inhibitor**	Enrollment in AIM-HN registration-directed study	ongoing
	Initiation of PI3Kα inhibitor combination study	2H 2021
Next-Generation **Farnesyl Transferase Inhibitor**	Nomination of Development Candidate for IND studies	mid-2021

Financial Highlights **Nasdaq: KURA**	Cash, cash equivalents and short-term investments: $633.3M*
	Shares outstanding: 66.2M basic, 5.0M options*

* As of December 31, 2020

KURA ONCOLOGY

DEVELOPING PRECISION MEDICINES FOR THE TREATMENT OF CANCER

Corporate Presentation – February 2021