

DEVELOPING PRECISION MEDICINES FOR THE TREATMENT OF CANCER



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

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

Proprietary Pipeline

- 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 PI3K α 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 $\geq$ 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

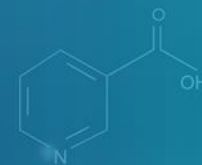
Troy Wilson, Ph.D., J.D.

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			
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:

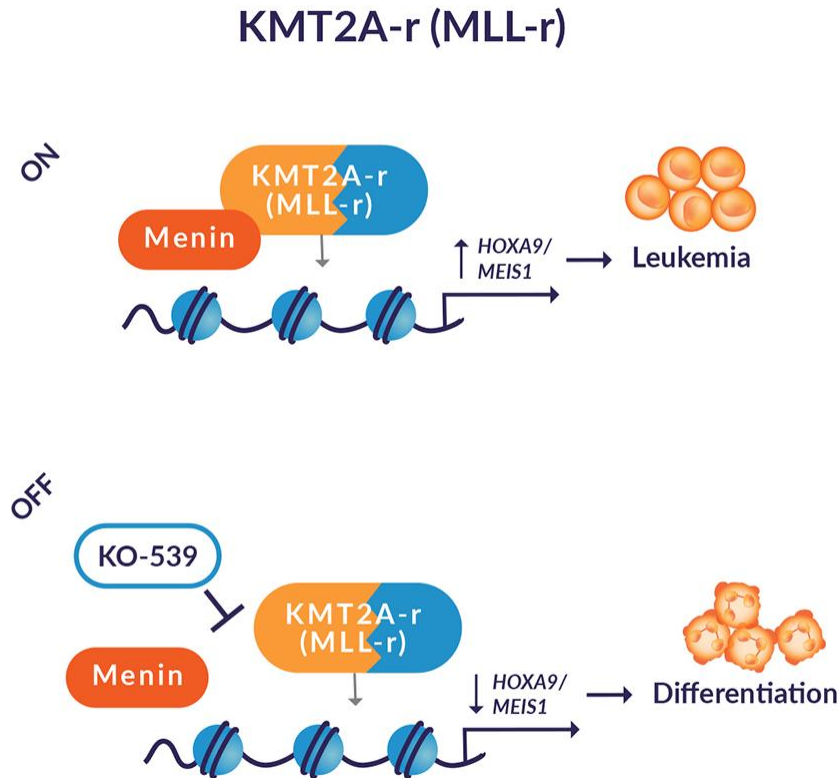
- KMT2A(MLL) rearranged (5-10% of AML)
- NPM1 mutant (~30% of AML)
- Other genetic subtypes (e.g., SETD2/RUNX1-mutant AML)

Potential to combine with other targeted therapies and induction chemotherapy

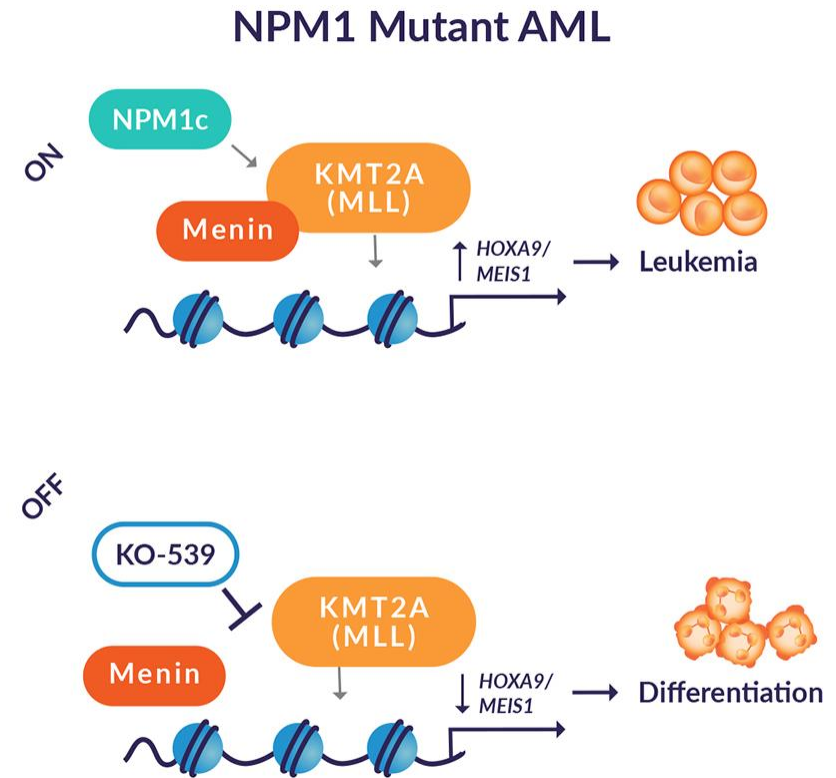


Issued and pending COM patents provide worldwide coverage to 2036

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



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

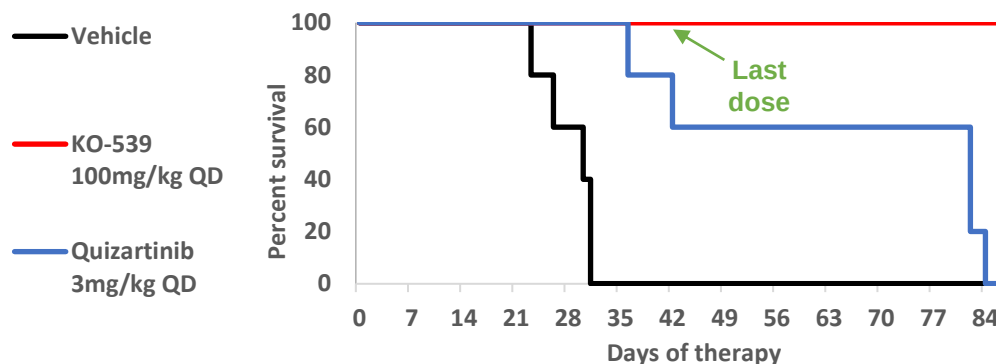


A central role for menin-KMT2A(MLL) interaction in epigenetic dysregulation in NPM1-mutant AML

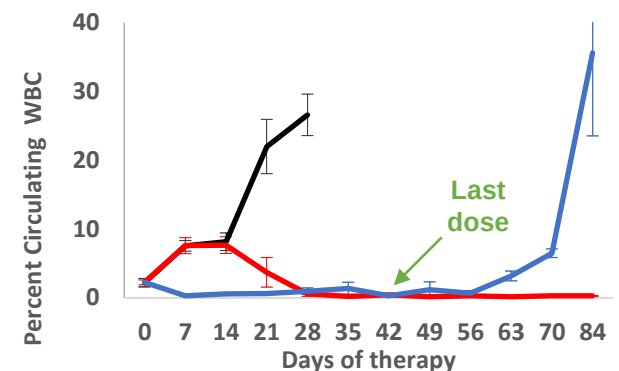
KO-539 Produces Lasting Complete Remissions in a NPM1 / DNMT3A / IDH2 / FLT3-Mutant AML Model

AM7577

Overall Survival

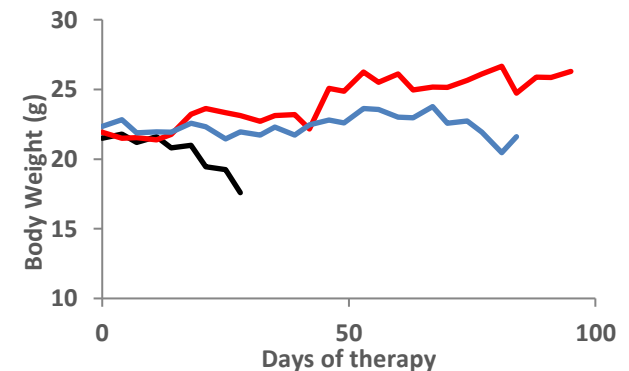


CD45+ Human AML Blasts



- 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

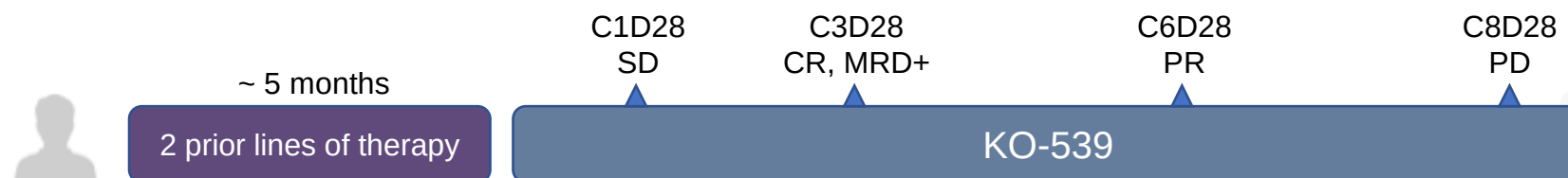


KO-539 Demonstrates Encouraging Early Clinical Activity

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	<i>RUNX1, SRSF2, ASXL1, TET2, STAG2, BCOR, PTPN11</i>	Yes	3	Decreased peripheral blasts
	<i>EZH2, DNMT3A, FAT3, RET</i>	Yes	3	Progressive disease
	<i>NPM1</i>	No	2	Not efficacy evaluable at time of data cut
	<i>DNMT3A, CUX1, ASXL1, IDH2, CBL, U2AF1, RUNX1</i>	Yes	5	Not efficacy evaluable at time of data cut
200 mg	<i>NPM1, DNMT3A, KMT2D</i>	Yes	7	Complete remission, MRD-
	<i>NPM1, FLT3-ITD, TET2, CUX1</i>	Yes	4	Morphological leukemia-free state
	<i>U2AF1, TET2, p53, DNMT3A, PTPN11</i>	No	4	Stable disease
	<i>IDH2, SRSF2, DNMT3A, CBL</i>	Yes	3	Progressive disease
	<i>TP53, PICALM (MLLT10)</i>	Yes	3	Not efficacy evaluable
	<i>KMT2A-r</i>	Yes	4	Not efficacy evaluable
100 mg	<i>SETD2, RUNX1</i>	Yes	2	Complete remission, MRD+
50 mg	<i>KMT2A-r</i>	Yes	2	Decreasing hydra requirement

Case Study – *SETD2*, *RUNX1* Mutant AML



Patient Characteristics

Demographics	69-year-old male
Mutational profile	<i>SETD2</i> , <i>RUNX1</i>
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

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



Patient Characteristics

Demographics	44-year-old female
Mutational profile	<i>NPM1</i> , <i>DNMT3A</i> , <i>KMT2D</i> , <i>FLT3-TKD</i>
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

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

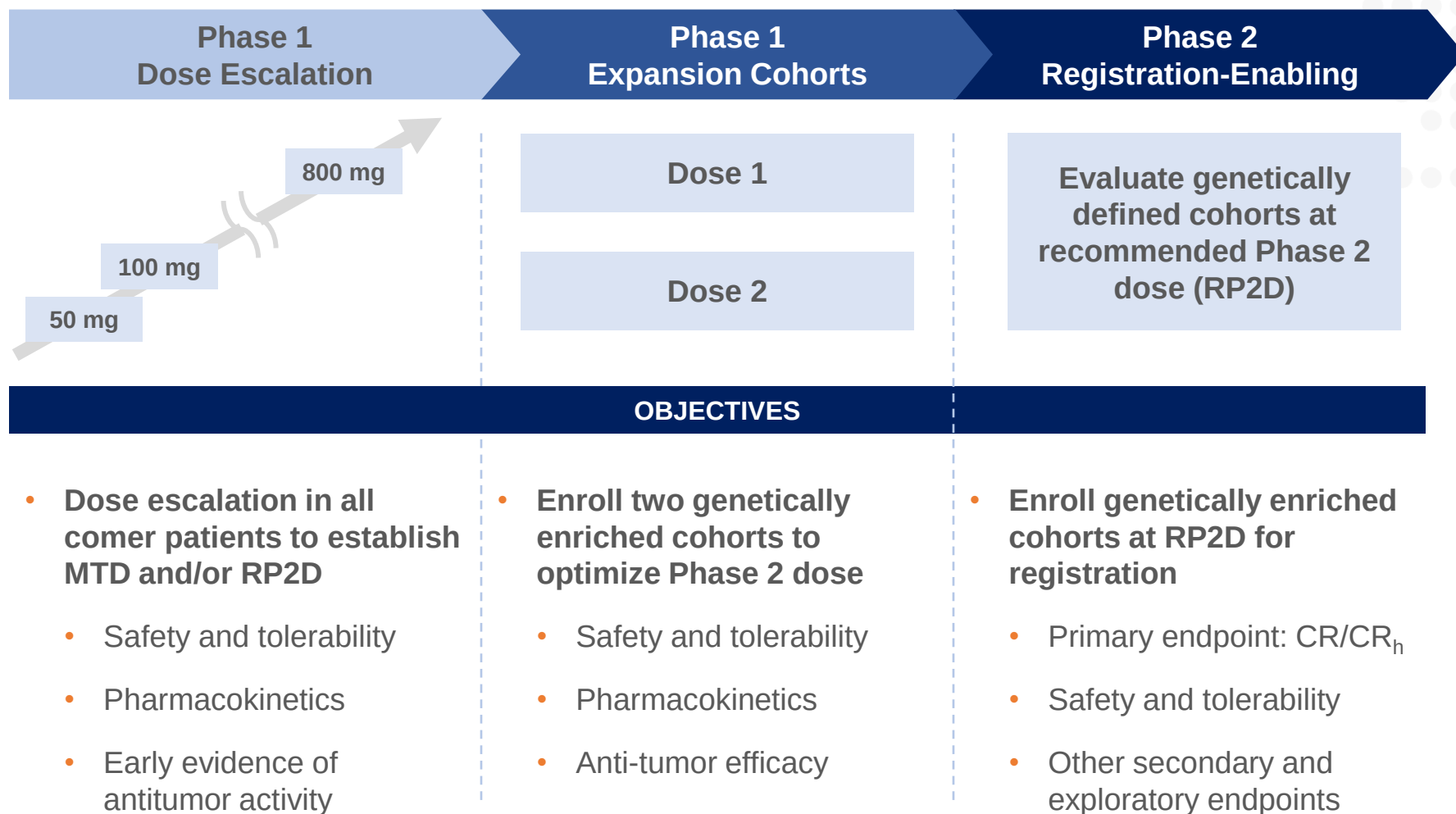
Treatment-related AEs (N=12)	Grade ≥ 3 (all)	Grade 1,2 ($\geq 10\%$)
Pancreatitis	1* (8.3%)	0%
Lipase increased	1* (8.3%)	0%
Neutrophil count decreased	1* (8.3%)	0%
Tumor lysis syndrome	1 (8.3%)	0%
Deep vein thrombosis	1 (8.3%)	0%
Nausea	0%	3 (25%)
Rash	0%	2 (16.7%)
Diarrhea	0%	2 (16.7%)

* 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

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

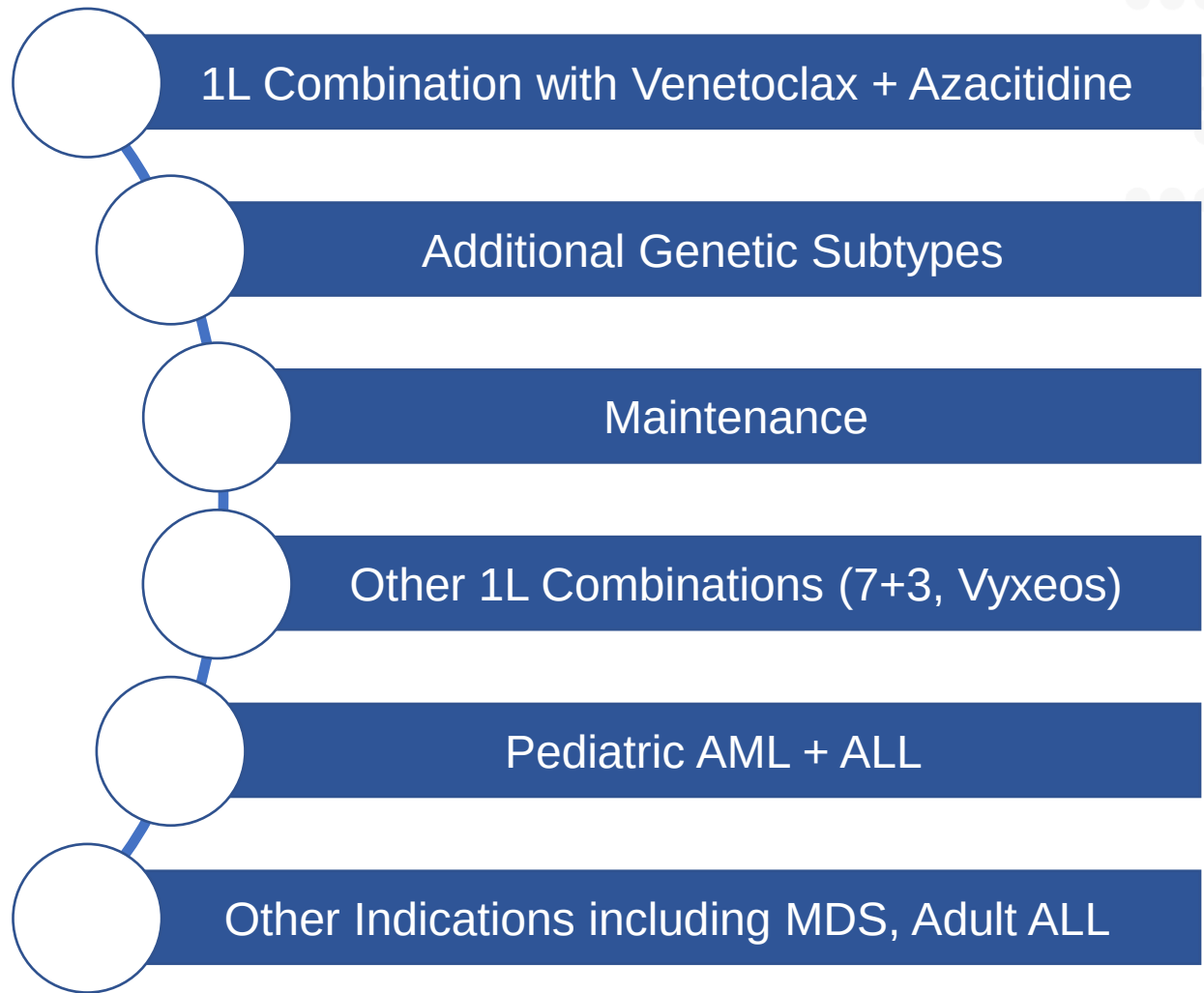
KOMET-001: Phase 1/2 Clinical Trial of KO-539 in Patients with Relapsed or Refractory AML



Multiple Expansion Opportunities in Acute Leukemias

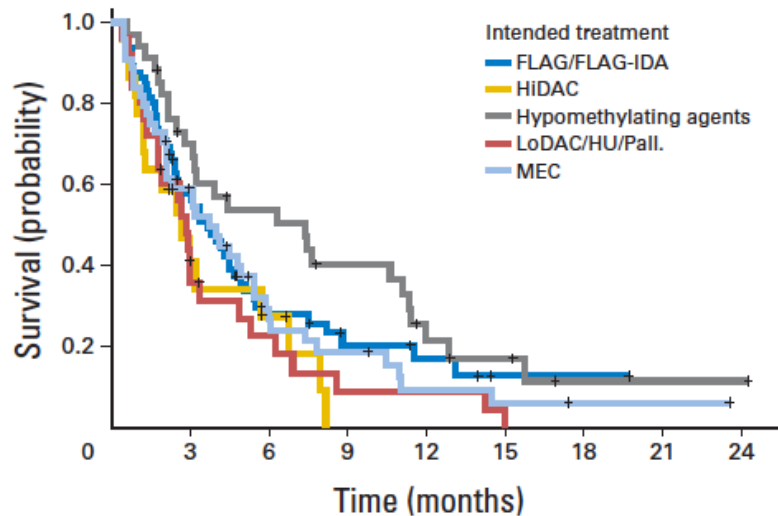


R/R NPM1 and
KMT2A (MLL)-r AML



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

Chemotherapy¹



Targeted Therapies

Drug Name	AML Subset	Median OS
Enasidenib	<i>IDH2</i> mutant	9.3 mos ²
Ivosidenib	<i>IDH1</i> mutant	8.8 mos ³
Gemtuzumab ozogamicin	CD33+ AML	11.6 mos ⁴
Gilteritinib	<i>FLT3</i> mutant	9.3 mos ⁵
Quizartinib	<i>FLT3-ITD</i> 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 year⁷

(~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 year⁷

(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

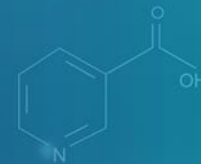
⁶ Cortes *et al.* Lancet Oncol. 2019 Jul;20(7):984-997

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



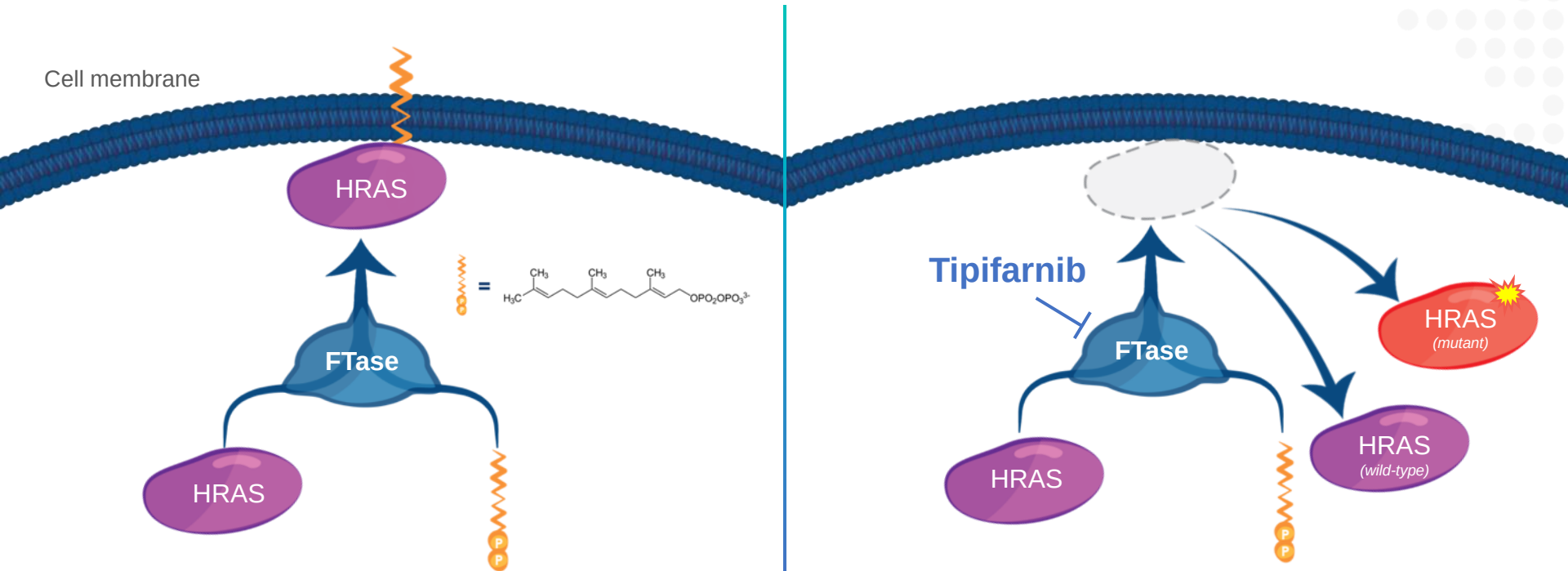
Novel mechanism and well tolerated profile could enable use in combination with standard of care, including immune therapy, targeted therapies and chemo



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 $\geq$ 20% after disease progression on platinum-based chemotherapy

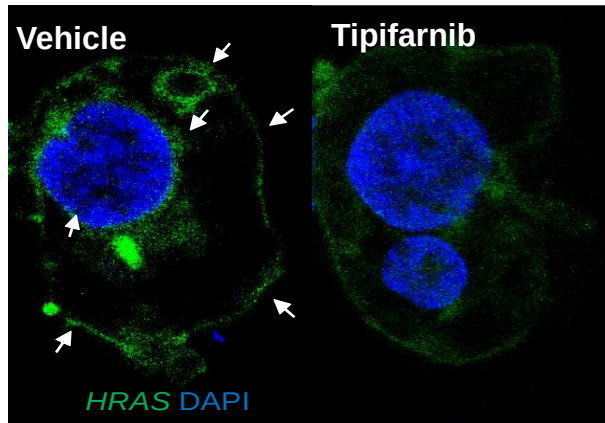
Tipifarnib Inhibits Farnesylation – An Essential Modification Required for HRAS Activity



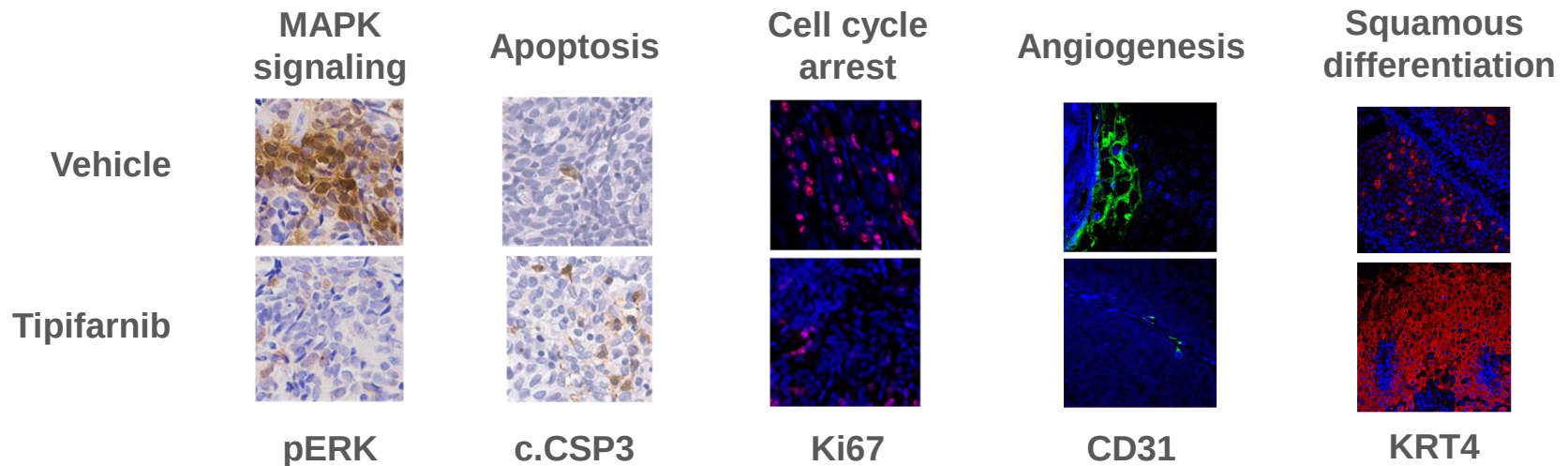
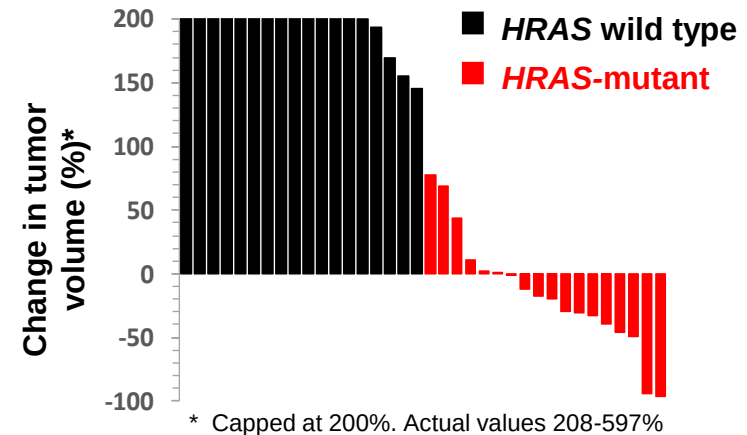
- Tipifarnib inhibits farnesylation and signaling activity of the HRAS oncoprotein
- Farnesylation is essential for HRAS signal transduction activity
- HRAS mutations drive proliferation and resistance mechanisms in solid tumors
- Incidence of HRAS mutations in HNSCC is approximately 4-8% and varies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

Tipifarnib Displays Robust, Selective Activity in HRAS Mutant HNSCC Models

HRAS membrane displacement



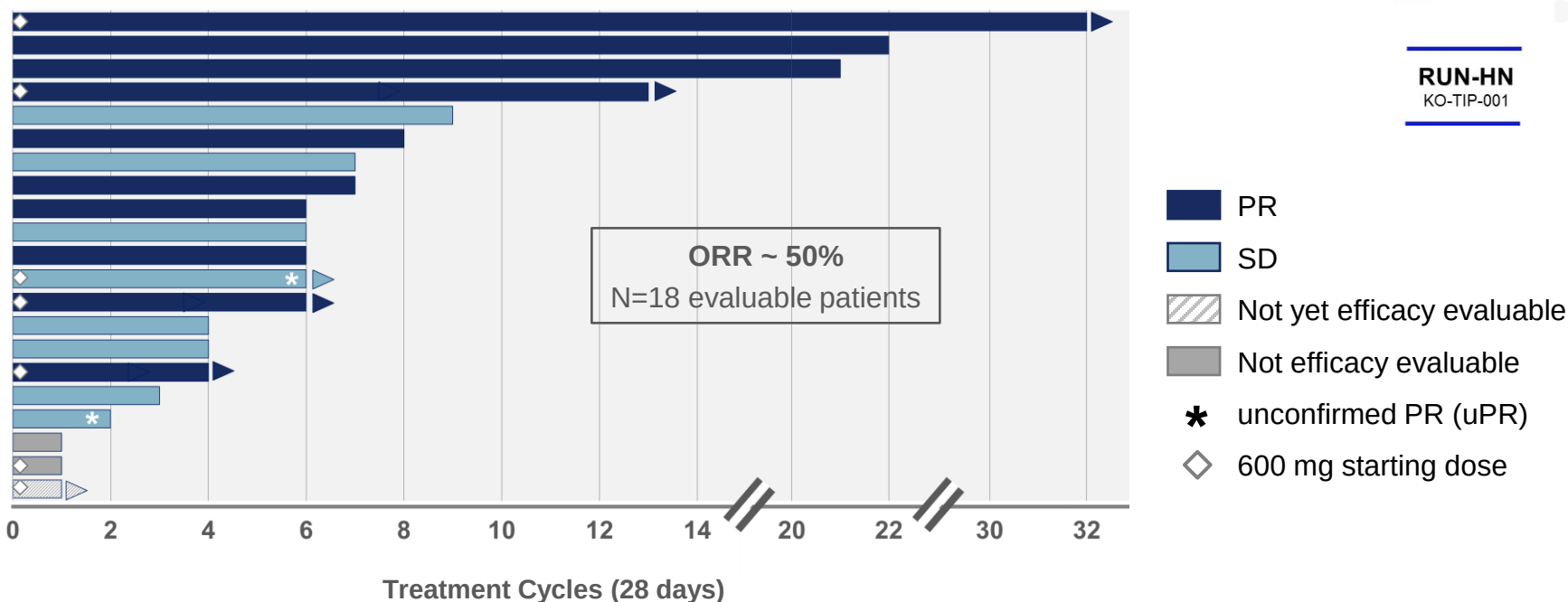
Antitumor activity in PDX models



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

RUN-HN: Phase 2 Trial in HRAS Mutant HNSCC

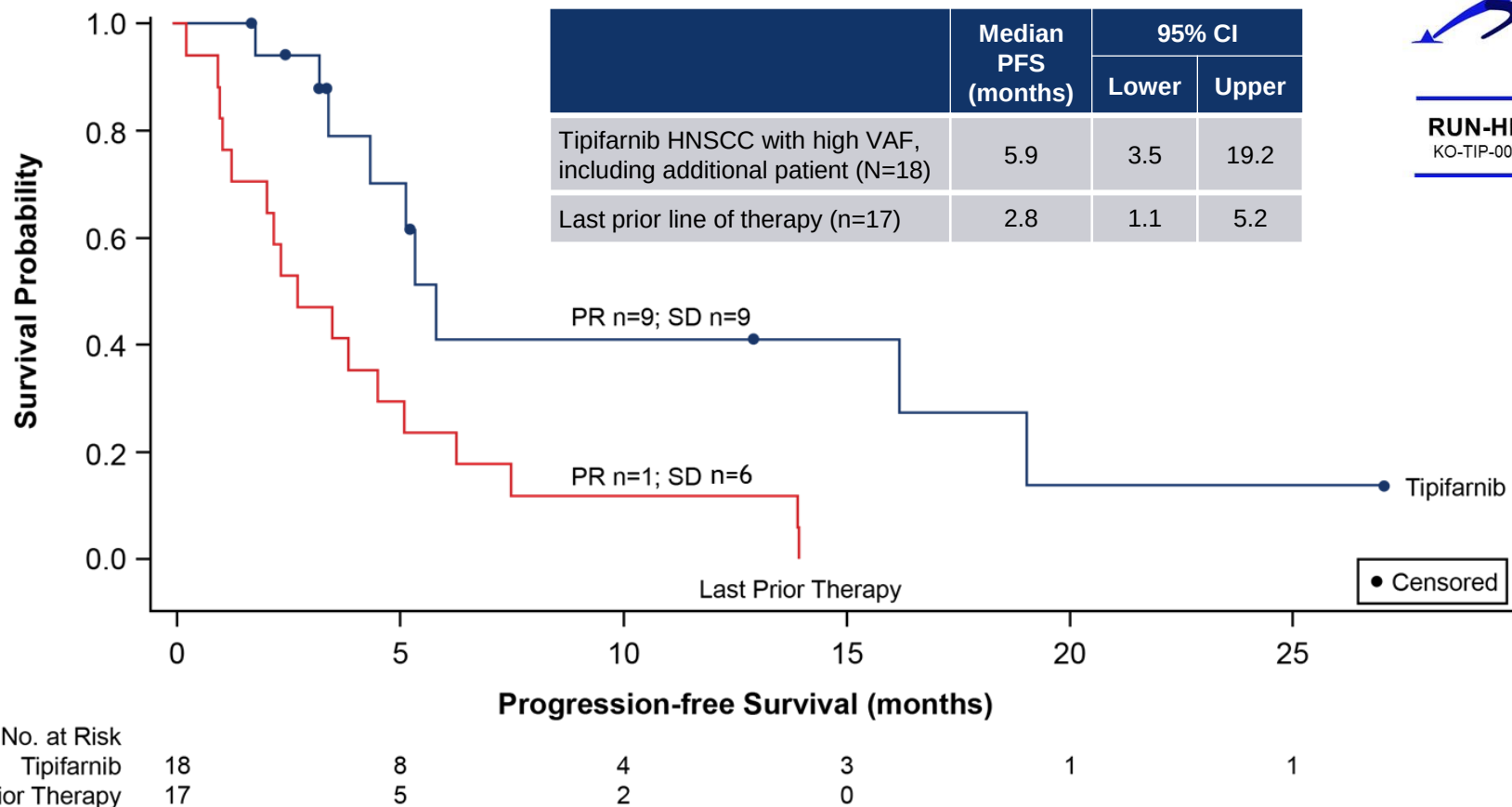
Time on Treatment



RUN-HN
KO-TIP-001

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

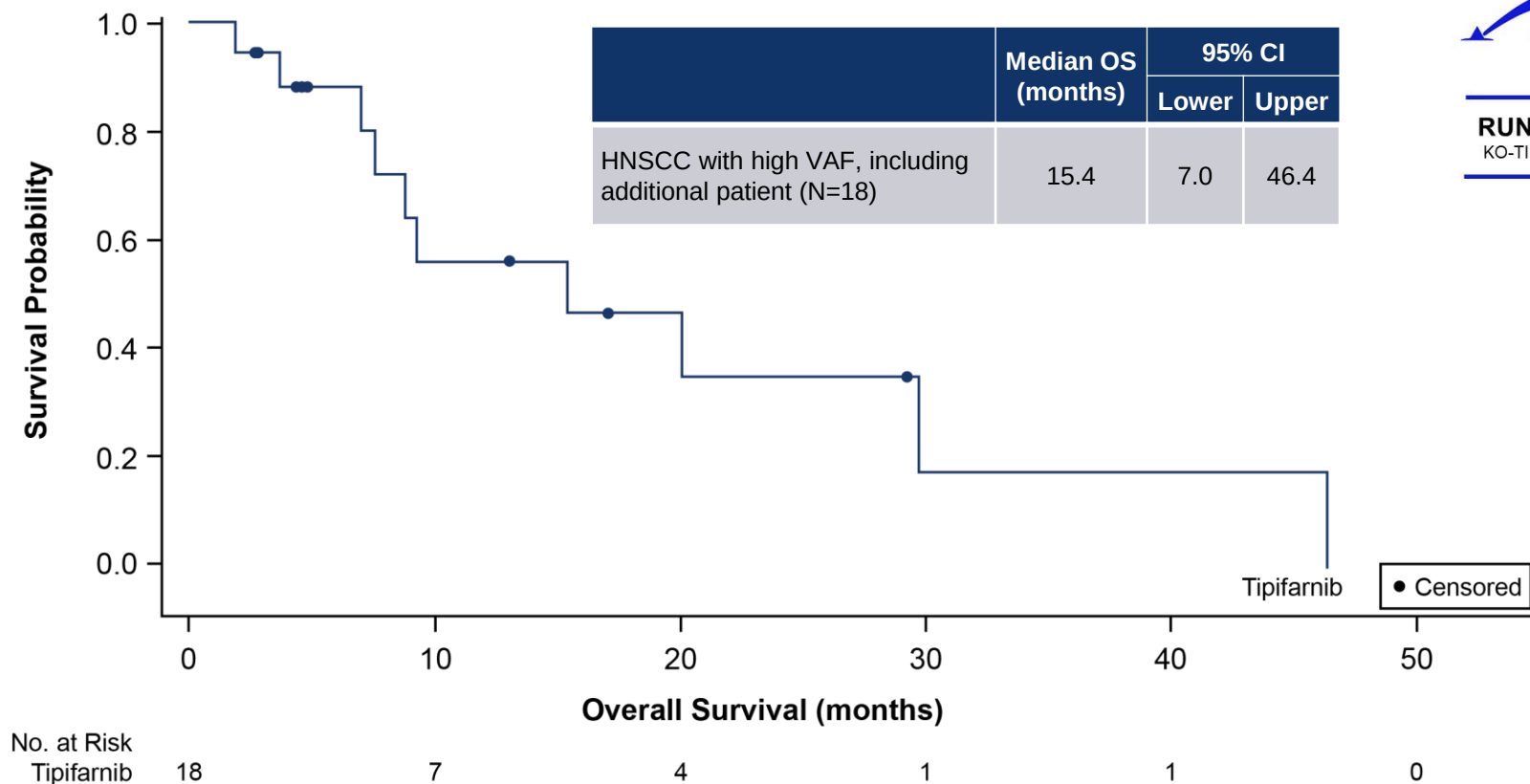
RUN-HN: Phase 2 Trial in HRAS Mutant HNSCC



RUN-HN
KO-TIP-001

Overall Survival in Patients with HRAS Mutant HNSCC

RUN-HN: Phase 2 Trial in HRAS Mutant HNSCC



RUN-HN
KO-TIP-001

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 > 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:
 - 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



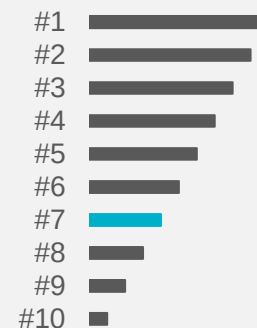
SEQ-HN
KO-TIP-007

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 year¹

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 poor⁵

OS

First line: 10-15 mo
Second line: 5-8 mo

PFS

First line: 3-5 mo
Second line: 2-3 mo

ORR

First line: 20-36%
Second line: 13-16%

¹ Bray et al. CA Cancer J Clin. 2018;68(6):394-424

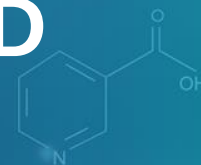
² Cramer et al. Nat Rev Clin Oncol. 2019 Nov;16(11):669-683 | ACS Cancer Facts and Figures 2020

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

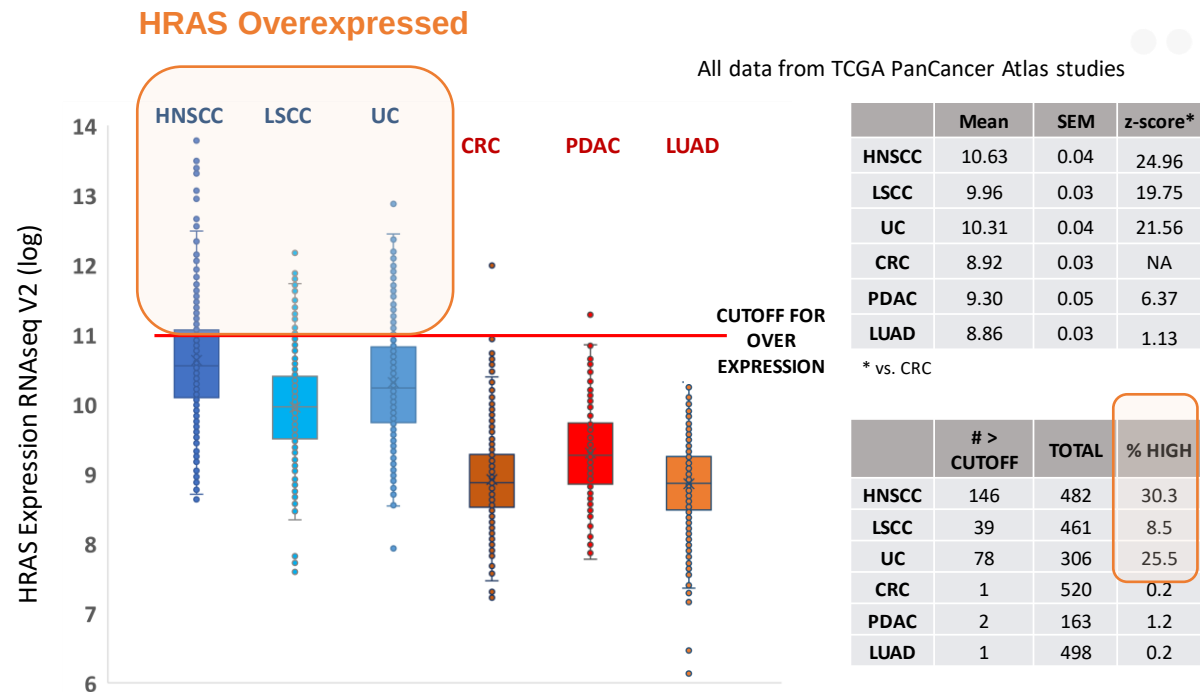
⁵ N Engl J Med. 2008 Sep 11;359(11):1116-27 | Keytruda & Opdivo package inserts | J Clin Oncol. 2007 Jun 1;25(16):2171-7 | J Clin Oncol. 2012 30:15_suppl, 5574-5574

EXPANSION OPPORTUNITIES FOR TIPIFARNIB IN HRAS AND PI3K α 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 subset¹
- TCGA cohort shows overexpression of HRAS gene in 25-30% of HNSCC²
- 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

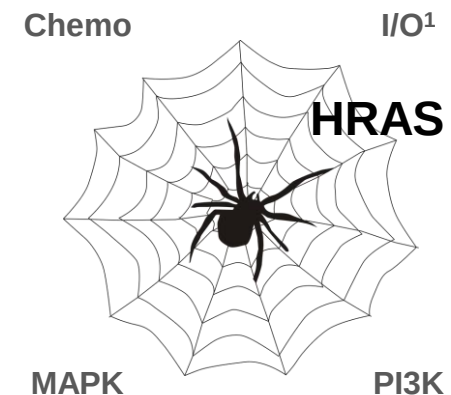
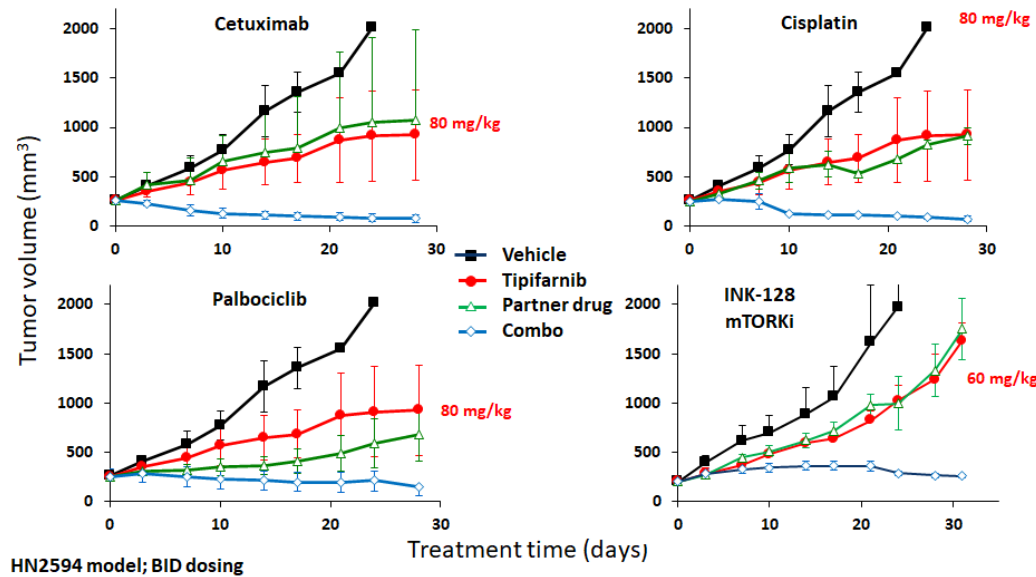


¹ Campbell et al. (2018), Cell Rep. 23:194; Su et al. (2017), Theranostics, 7:1088;

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

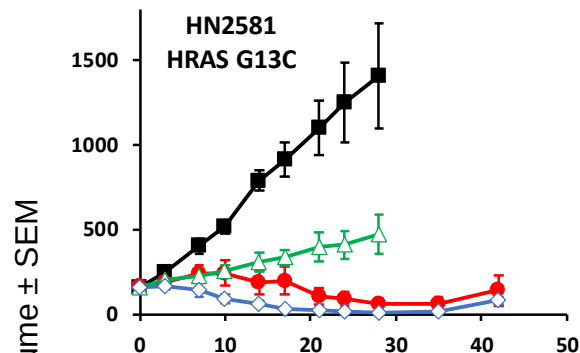


- HRAS represents a key node at the center of HNSCC tumor biology, driving resistance to other therapies and reinforcing the potential for combination strategies with tipifarnib

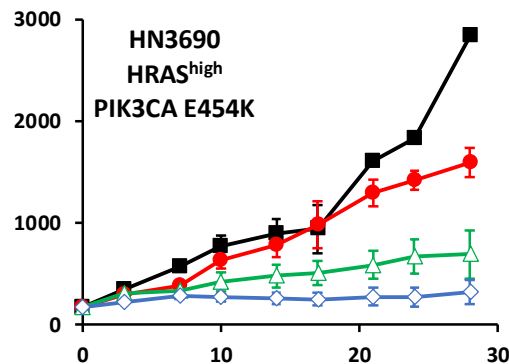
¹ HRAS likely drives immunosuppression in HNSCC, and tipifarnib may also sensitize to immunotherapy via inhibition of CXCL12 production by activated carcinoma-associated fibroblasts

Combinations of Tipifarnib and PI3K α Inhibitor Demonstrate Robust Activity in HNSCC PDX Models

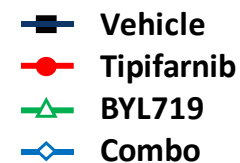
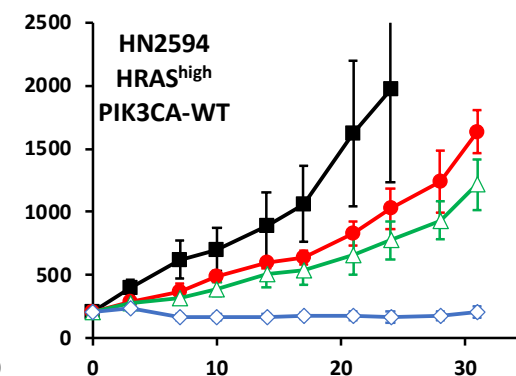
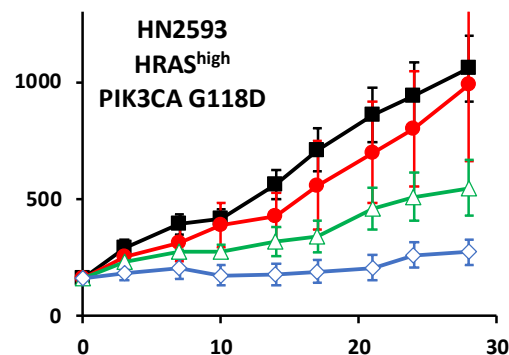
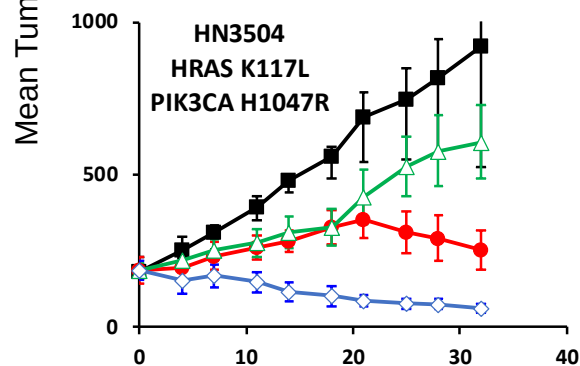
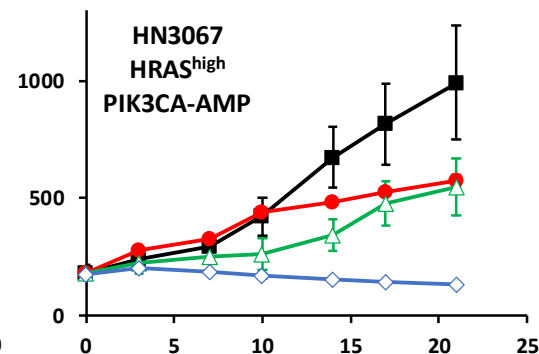
HRAS-mutant



PIK3CA-mutant



Wild-Type



Days of treatment

Combinations of Tipifarnib and PI3K α 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 PI3K α 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 HNSCC¹
 - 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 PI3K α 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

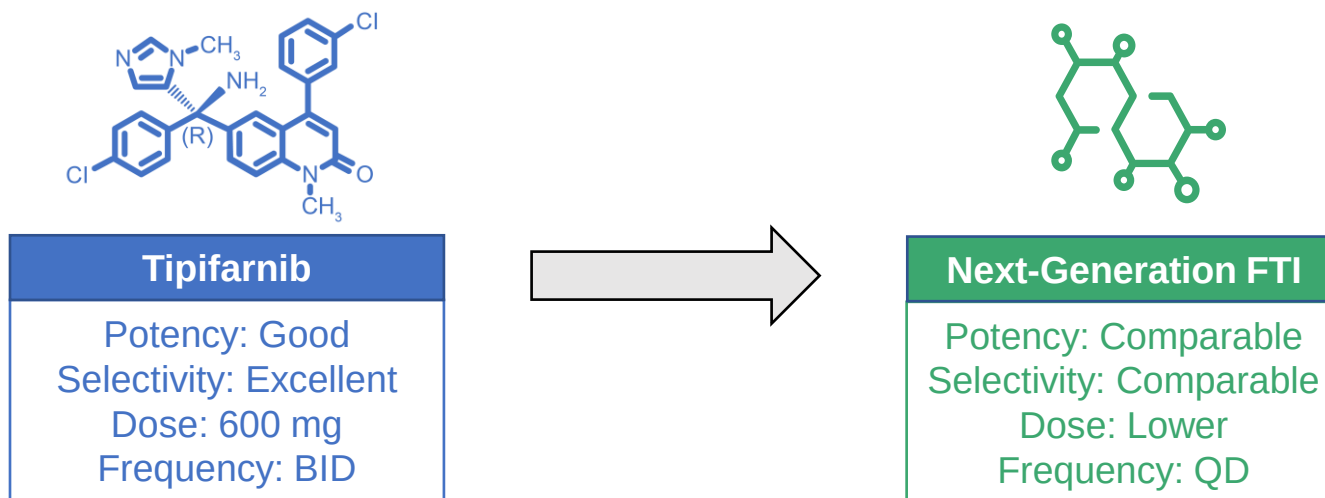
- Patents cover combinations of tipifarnib with other agents (e.g., I/O)
- Additional patents possible with specific agents, doses, schedules, etc.

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



- 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*
---	---

DEVELOPING PRECISION MEDICINES FOR THE TREATMENT OF CANCER

