KURA ONCOLOGY

Cowen and Company Health Care Conference

March 3, 2020

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, KO-947 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 “aim,” “target,” “next steps,” “would,” “opportunity,” “expected,” “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; 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 pipeline of targeted drug candidates for selected solid tumors and hematologic malignancies Utilizing precision medicine approach; Fast-to-market strategy
Proprietary Pipeline	**Tipifarnib**: Farnesyl transferase inhibitor • Clinical proof of concept achieved in HRAS mutant solid tumors and CXCL12-dependent hematologic malignancies • 1st Phase 2 registration-directed trial ongoing; 2nd planned in 2020 • Significant lifecycle expansion opportunities in solid and liquid tumors **KO-947**: ERK inhibitor • Recommended Phase 2 dose potentially defined; expansion cohort pending **KO-539**: Menin-MLL inhibitor • First-in-class drug candidate; Phase 1 dose-escalation trial ongoing
Near-Term Milestones	Multiple anticipated development milestones across the pipeline in 2020
Financials	Approximately $237 million in cash as of December 31, 2019*

* Includes cash, cash equivalents and short-term investments

Kura Leadership Team and Board of Directors

Leadership Team

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

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

Kathleen Ford
Chief Operating Officer

James Basta, J.D.
Chief Legal Officer

Kirsten Flowers
Chief Commercial Officer

Bridget Martell, M.A., M.D.
Acting Chief Medical Officer

Board of Directors

Faheem Hasnain
Executive Chairman, Gossamer Bio

Robert Hoffman
Chief Financial Officer, Heron Therapeutics

Thomas Malley
President, Mossrock Capital

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

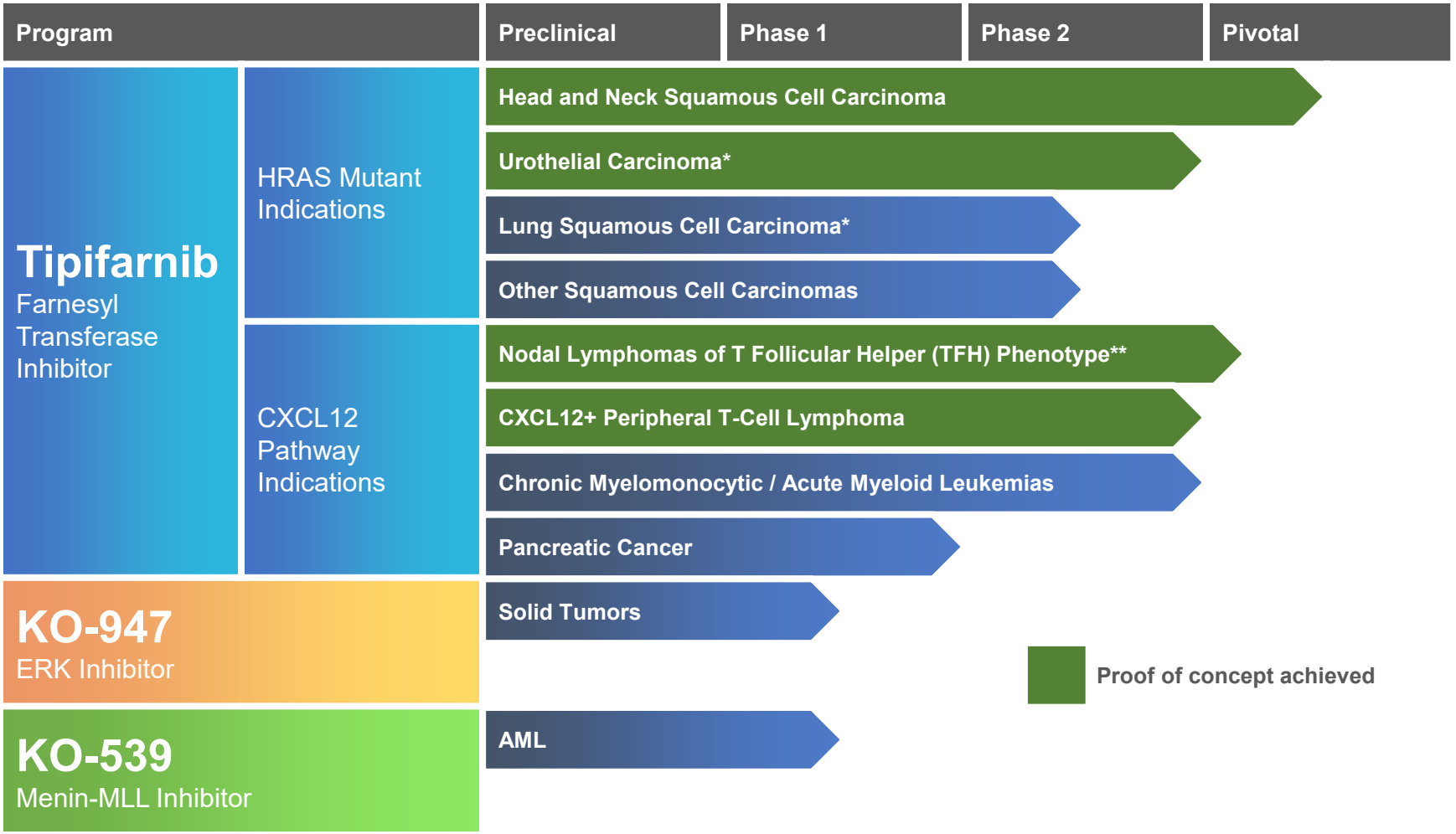
Steven Stein, M.D.
Chief Medical Officer, Incyte

Mary Szela
President and CEO, TriSalus Life Sciences

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

Proven oncology drug development and commercialization expertise

Product Candidate Pipeline



* Investigator-sponsored trials
** Includes angioimmunoblastic T-cell lymphoma (AITL)

01 • *Tipifarnib (Farnesyl Transferase Inhibitor)*

02 *KO-947 (ERK Inhibitor)*

03 *KO-539 (Menin-MLL Inhibitor)*

Farnesyl Transferase Inhibitors (FTIs): A Leadership Opportunity in Targeted Oncology

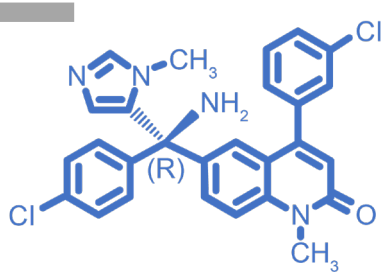
Accelerated approval paths with single-arm, ORR-driven studies in relapsed/refractory solid and liquid tumors

Lifecycle expansion opportunities with validated biomarkers in multiple solid and liquid tumors

Issued and pending patents provide exclusivity for FTIs in major markets

Kura is the leader in the research, development and commercialization of tipifarnib and FTIs

Tipifarnib: A First-in-Class Farnesyl Transferase Inhibitor for the Treatment of Cancer



- Potent, selective inhibitor of farnesyl transferase[1]
- Well characterized with > 5,000 patients treated
- Durable responses previously reported in selected study patients, but appropriate genetic biomarkers were not identified at that time
- Kura scientists discovered proprietary biomarkers (HRAS and CXCL12 pathway); validated in five Phase 2 proof-of-concept studies
- Manageable safety profile observed as monotherapy (< 25% treatment discontinuation)
- Tipifarnib adverse events[2]:
  - Myelosuppression (neutropenia 25%, anemia 31%, thrombocytopenia 19%)
  - Non-heme > 25%: fatigue (41%) and GI unspecific (nausea 47%, anorexia 33%, diarrhea 32%, vomiting 32%)

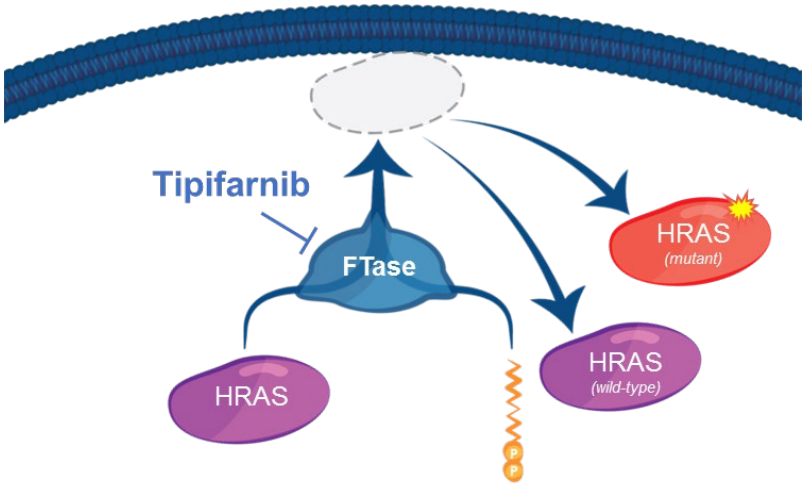
Kura unlocked the potential of tipifarnib and FTIs as targeted therapeutics in oncology

[1] End *et al*. 2001. *Cancer Res*. 61:131-37

[2] Adverse events reported from 472 solid tumor patients

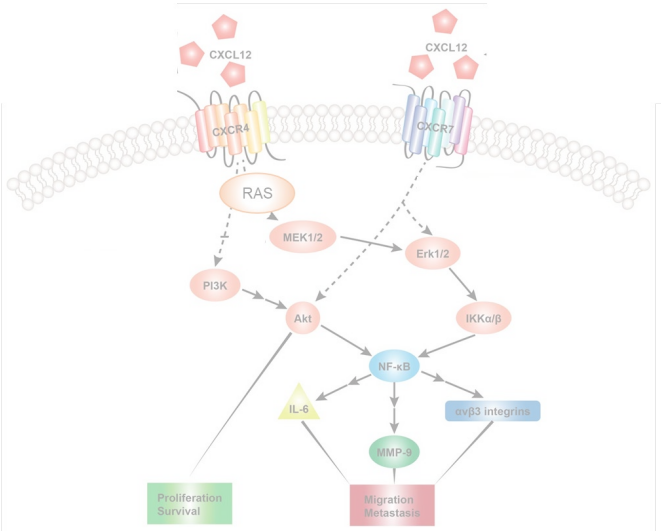
FTIs Inhibit Two Distinct Critical Pathways and Drive Activity in Biomarker-Defined Tumors

HRAS Mutant Solid Tumors



- HRAS mediates signal transduction and growth and proliferation of tumor cells
- HRAS mutations drive resistance to SOC therapies; poor prognosis
- Tipifarnib inhibits farnesylation and signaling activity of the HRAS oncoprotein

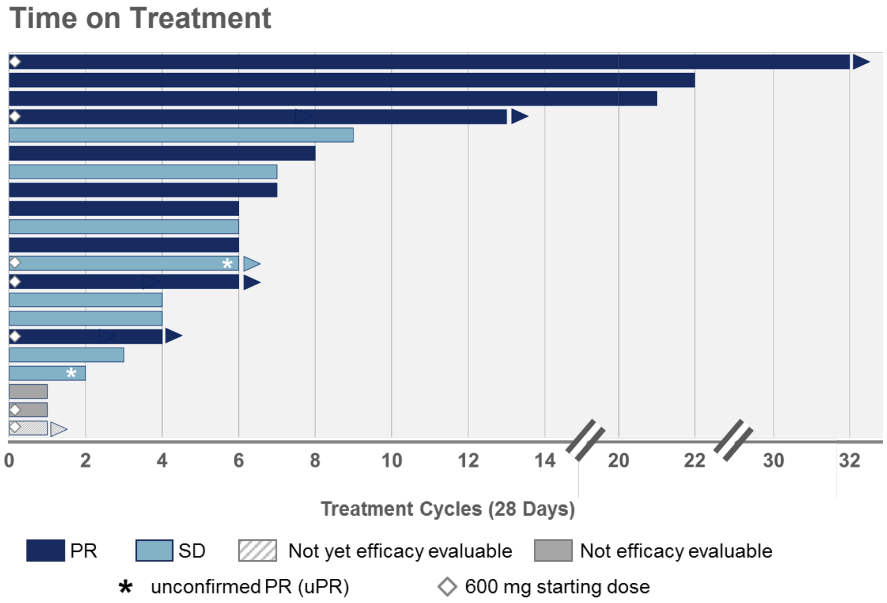
CXCL12-Dependent Tumors



- CXCL12 and its receptors (CXCR4, CXCR7) link cancer cells to the tumor microenvironment
- CXCL12 pathway activation drives cancer phenotype; poor prognosis
- Tipifarnib inhibits farnesylation of key regulatory proteins involved in CXCL12 production

Registration Strategy in HRAS Mutant HNSCC: Potential for Accelerated Approval

RUN-HN: HRAS Mutant Head & Neck Squamous Cell Carcinomas[1]



- HNSCC represents a significant unmet need as standard of care provides limited clinical benefit (ORR ~13-16%, PFS ~2 months) in 2nd line
- HRAS mutations are a negative prognostic factor and primary mechanism of resistance to standard of care
- In RUN-HN study, tipifarnib showed durable anti-tumor activity (ORR ~50%, PFS ~6 months) as a single agent in heavily pretreated patients with HRAS mutant HNSCC

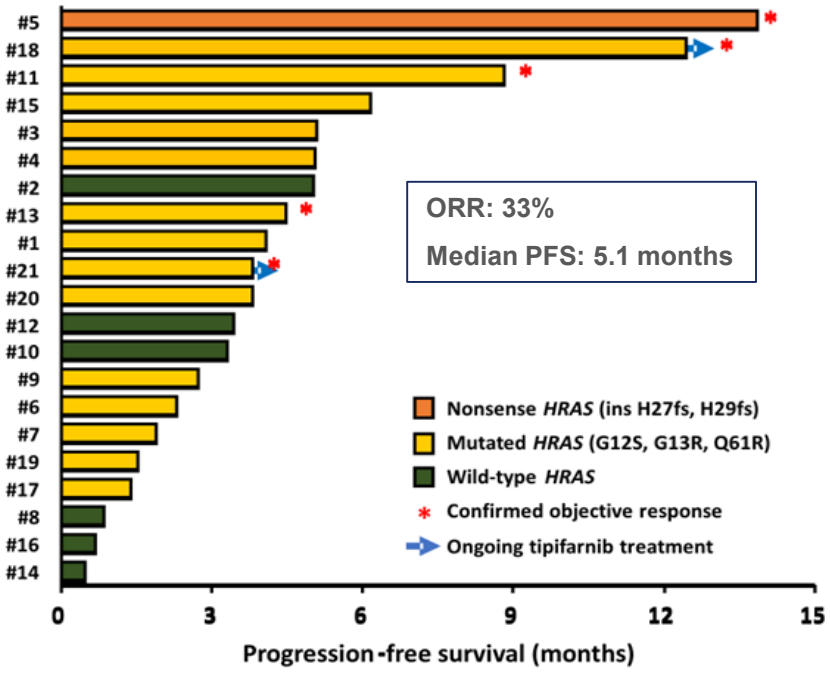
AIM-HN: Registration-Directed Trial of Tipifarnib in HRAS Mutant HNSCC

- At least 59 evaluable recurrent or metastatic patients after platinum therapy
- Trial initiated in November 2018; Full enrollment projected in Q1 2021
- Intended to support an NDA seeking accelerated approval[2]

[1] Ho et al. AACR-NCI-EORTC #384 (preliminary data as of 10/17/19)
[2] Feedback from end-of-Phase 2 meeting with FDA

Proof-of-Concept in Urothelial Carcinoma Demonstrates Potential for Label Expansion

HRAS Mutant Urothelial Carcinoma*



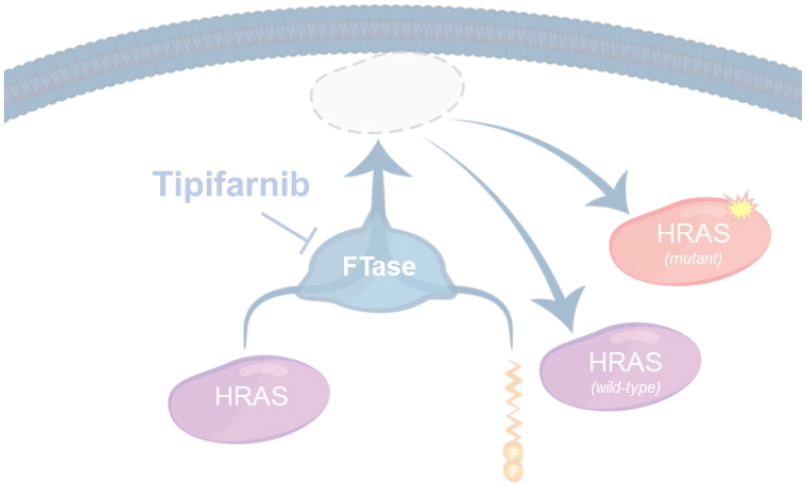
Potential for Label Expansion in HRAS Mutant Tumors

- HNSCC
  - Earlier lines of therapy: 1st line combination with SOC I/O and adjuvant setting
  - Broaden patient pool in low HRAS mutant variant allele frequency in combination
- Urothelial carcinoma
- Lung squamous cell carcinoma
- Other SCCs
  - Penile
  - Vulvar
  - Cutaneous

* Dr. Se Hoon Park, Samsung Medical Center (unpublished data)

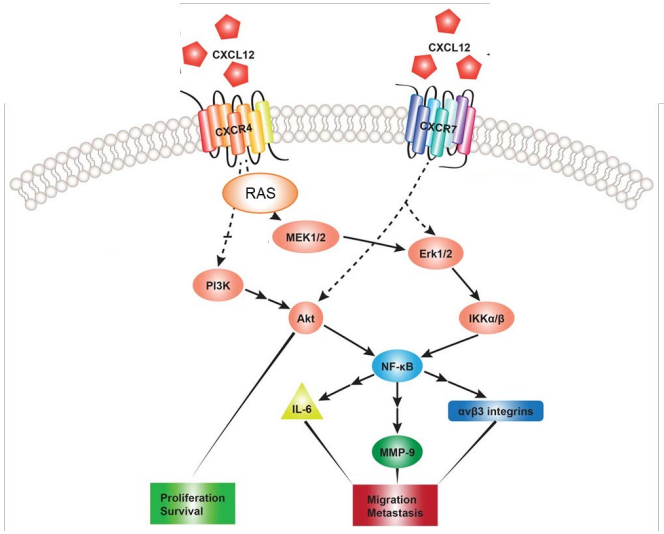
FTIs Inhibit Two Distinct Critical Pathways and Drive Activity in Biomarker-Defined Tumors

HRAS Mutant Solid Tumors



- HRAS mediates signal transduction and growth and proliferation of tumor cells
- HRAS mutations drive resistance to SOC therapies; poor prognosis
- Tipifarnib inhibits farnesylation and signaling activity of the HRAS oncoprotein

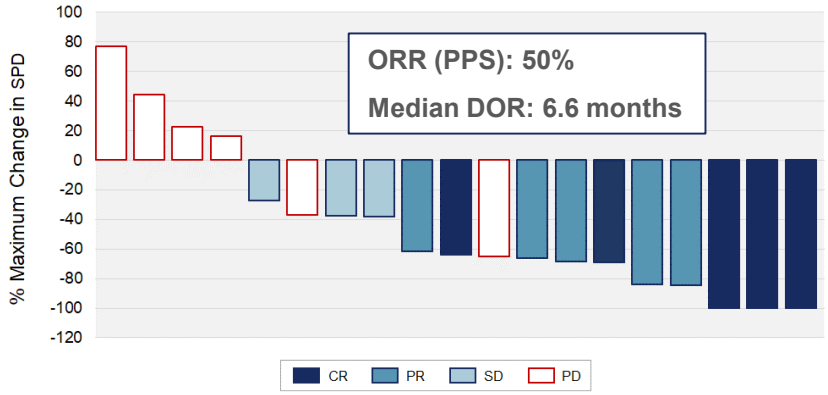
CXCL12-Dependent Tumors



- CXCL12 and its receptors (CXCR4, CXCR7) link cancer cells to the tumor microenvironment
- CXCL12 pathway activation drives cancer phenotype; poor prognosis
- Tipifarnib inhibits farnesylation of key regulatory proteins involved in CXCL12 production

Single-Arm ORR Trial Provides Potential for Accelerated Approval in Advanced Nodal Lymphomas of TFH Phenotype

Phase 2 Trial Results in Relapsed/Refractory AITL



- A significant unmet need as standard of care provides limited clinical benefit (ORR ~25%, PFS 2-3 months)
- CXCL12 is a negative prognostic factor for standard of care PTCL therapy
- AITL is an aggressive form of TFH lymphoma characterized by high levels of CXCL12
- Enhanced activity in patients with KIR mutations (ORR 70%, CR 40%)

Registration-Directed Trial in Nodal Lymphomas of TFH Phenotype, Including AITL

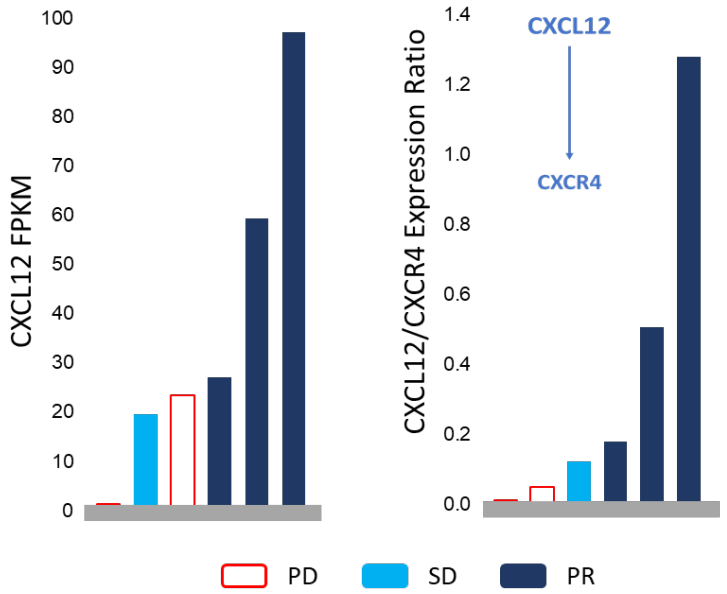
- Single-arm trial of 128 relapsed/refractory patients who have had at least one prior systemic chemotherapy
- Trial has two independent primary objectives:
  1) ORR in all patients enrolled
  2) ORR in patients with KIR mutations (determined retrospectively)
- Each objective has a 9% null hypothesis and can be met independently
- Plan to initiate in 2H 2020, intended to support NDA seeking accelerated approval

Witzig *et al*. ASH 2019 #468 (preliminary data as of 11/11/19)

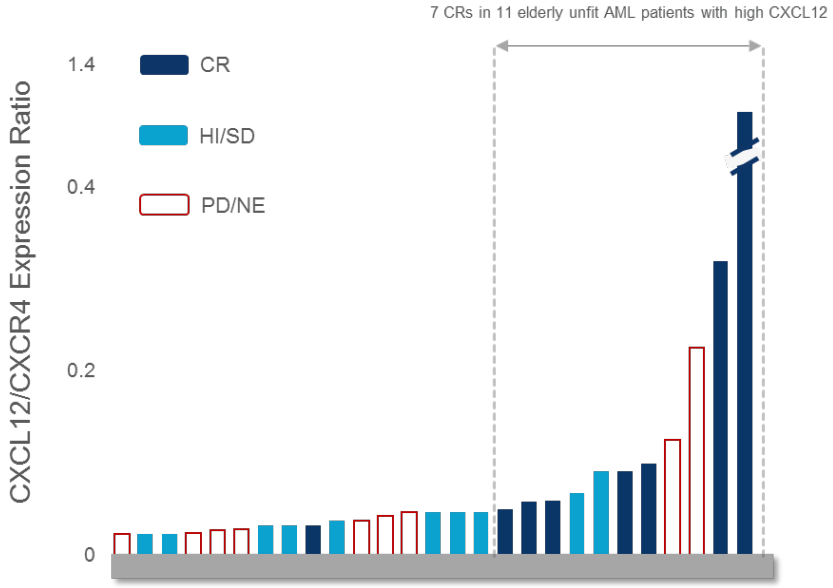
Proof-of-Concept in AITL/CXCL12-High PTCL Demonstrates Potential for Label Expansion

- Phase 2 PTCL study shows potential to utilize CXCL12 to enrich for clinical activity
- Retrospective analyses demonstrate association between CXCL12 pathway activation and clinical activity in DLBCL, CTCL and AML
- Potential indications of interest: Post ASCT AITL/TFH; CXCL12-dependent PTCL; 1L AITL/TFH, combination with SOC; 2L, 3L DLBCL; Acute leukemias

Diffuse Large B-Cell Lymphoma



Acute Myeloid Leukemia



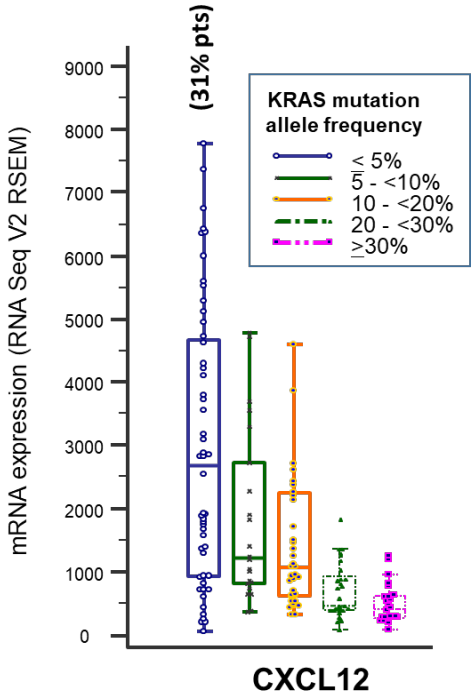
Potential for Label Expansion in CXCL12-Dependent Solid Tumors: Pancreatic Cancer

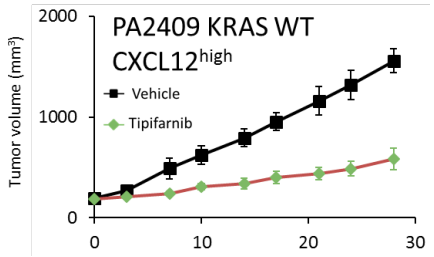
Rationale

- High CXCL12 expression is associated with reduced overall survival in patients with pancreatic cancer[1]
- CXCL12 expression and KRAS mutant allele frequency (MAF) are inversely related: ~30% of pancreatic tumors carry <5% KRAS MAF and express high levels of CXCL12[2]
- Tipifarnib downregulates CXCL12 secretion from pancreatic stellate cells and inhibits the growth of high CXCL12, low KRAS mutant pancreatic xenografts[3]
- Overall survival benefit observed with tipifarnib treatment in patients with CXCL12-expressing pancreatic tumors (identified by clinical characteristics) in retrospective analyses[2]

Next Steps

- Anticipate initiating a Phase 2 POC study in 2H 2020

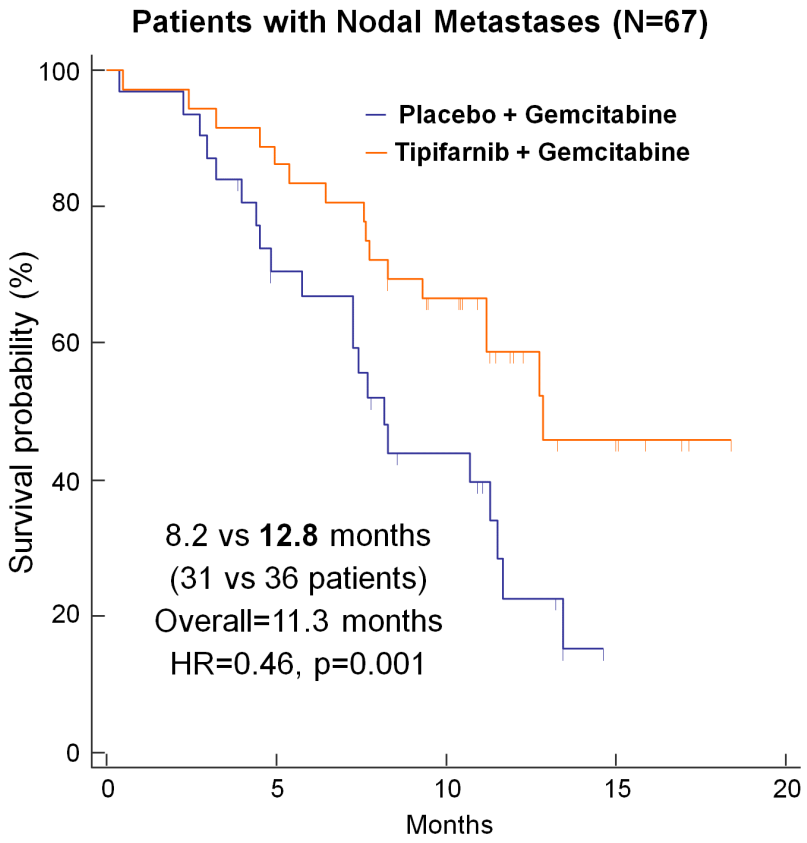


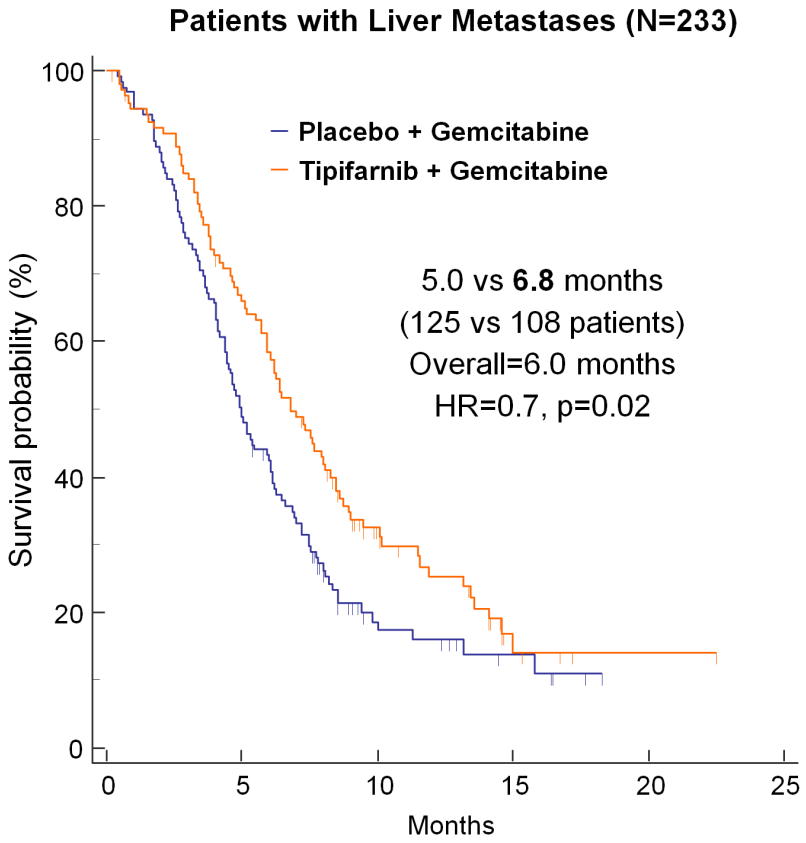


[1] Samarendra, *et al. Br J Cancer.* 2017;117:124–15
[2] Gualberto *et al. JCO* 2019. 37, suppl: 275 (gene expression data from TCGA)
[3] Kura Oncology, data on file

Evidence of Tipifarnib Activity in CXCL12-Associated Pancreatic Cancer

Retrospective analysis of INT-11, a Phase 3 trial of gemcitabine with or without tipifarnib in advanced pancreatic cancer

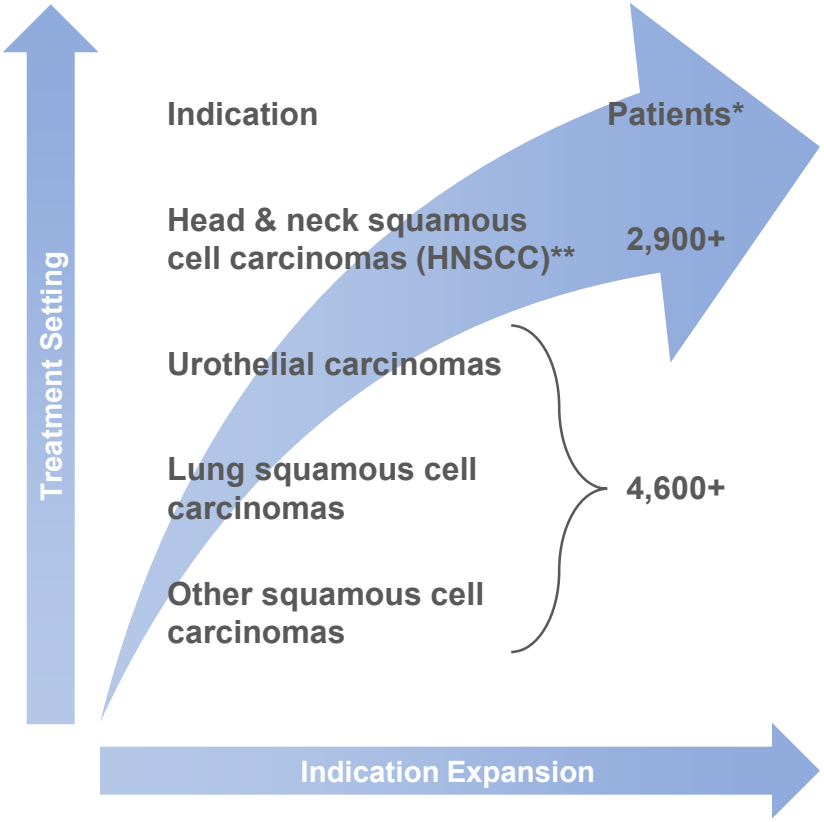




Tipifarnib: Broad Potential Market Expansion Opportunity

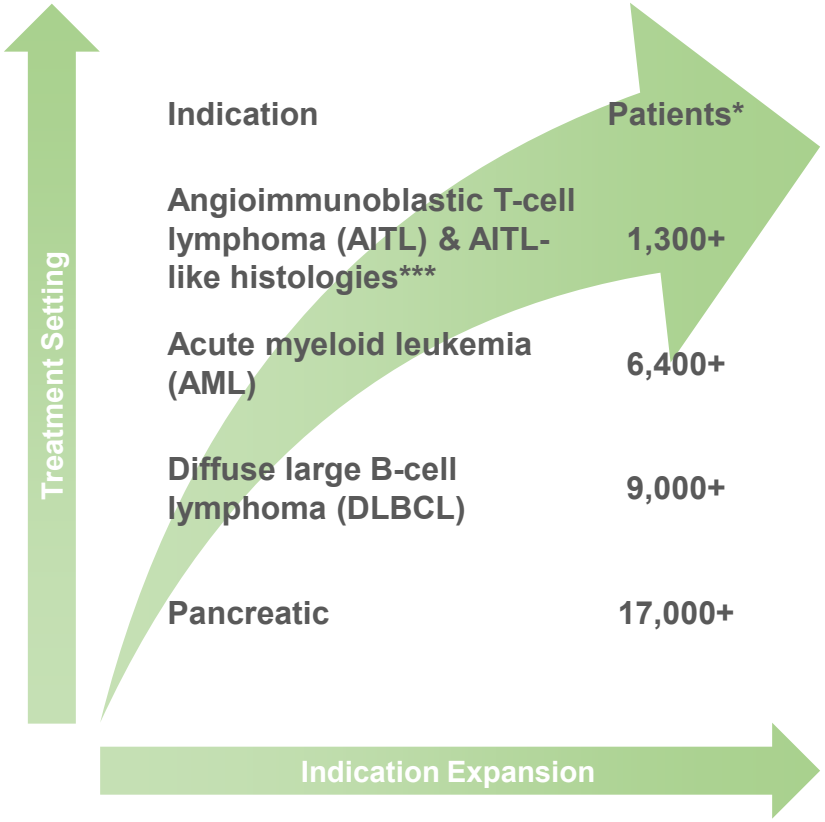
HRAS Mutant Solid Tumors

Populations Based on Annual U.S. Incidence



CXCL12 Pathway Indications

Populations Based on Annual U.S. Incidence



* Estimates of the biomarker-positive subsets across all lines of therapy

** HNSCC population with HRAS variant allele frequency ≥ 20% (TCGA)

*** Does not include additional opportunities in peripheral T-cell lymphoma (PTCL) or cutaneous T-cell lymphoma (CTCL)

Tipifarnib / FTI Patent Exclusivity

Layered patent strategy provides patent exclusivity to 2036 and beyond in major markets

Proprietary Biomarkers and Methods

- Multiple issued U.S. patents covering biomarker-guided indications (e.g., HRAS mutant HNSCC, CXCL12-expressing PTCL) and provide patent exclusivity to 2036 and beyond
- Include claims to biomarker, dose, schedule and tumor
- 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

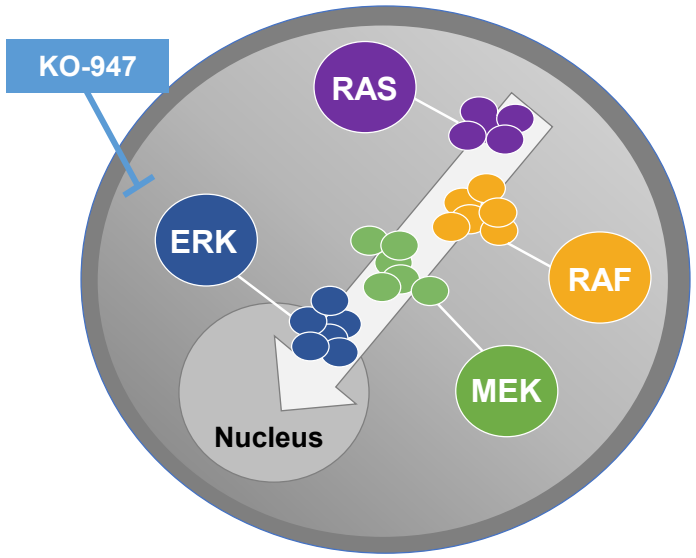
01 *Tipifarnib (Farnesyl Transferase Inhibitor)*

02 • *KO-947 (ERK Inhibitor)*

03 *KO-539 (Menin-MLL Inhibitor)*

KO-947: Potent Inhibitor of ERK1/2

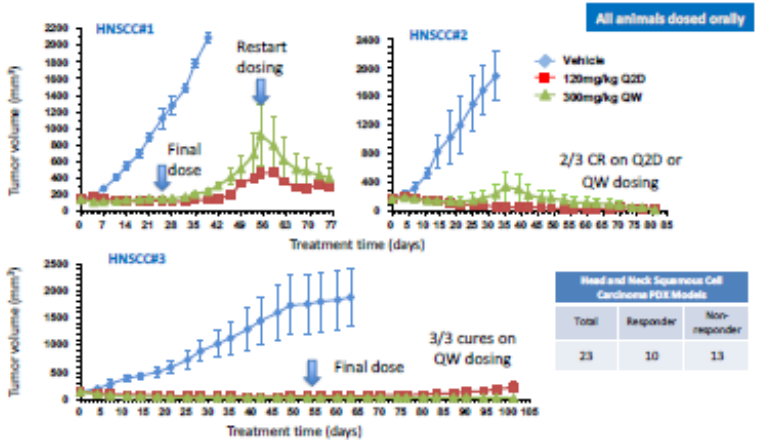
- Potent, selective small molecule inhibitor of ERK1/2
- Demonstrates prolonged pathway modulation in preclinical tumor models
- Multiple tumors, including molecularly-defined squamous cell carcinomas and adenocarcinomas, identified as sensitive to KO-947 as monotherapy in preclinical models
- Mechanism-based and SOC combinations under evaluation
- Favorable pharmacology enables intermittent dosing schedules
- Potential biomarkers, including 11q13 amplifications in SCCs, have been identified for patient enrichment



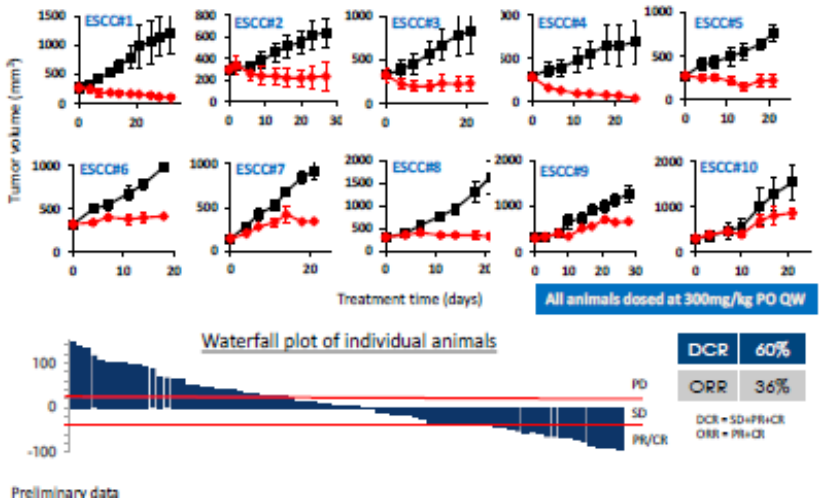
KO-947 Demonstrates Robust Single-Agent Activity in Preclinical Studies

- Broad profiling in ~ 200 patient-derived xenograft (PDX) models
- Consistent and compelling activity in diverse indications on intermittent schedules
- Robust activity in preclinical models of HNSCCs and ESCCs with 11q13 amplifications
- Leverages existing HNSCC clinical and diagnostic infrastructure for tipifarnib

KO-947 induces complete responses and regressions of large tumors in head and neck squamous cell carcinoma



KO-947 is highly active in PDX models of esophageal squamous cell carcinoma



KO-947: Phase 1 Clinical Trial

- First-in-human study in patients with advanced solid tumors
- Two dosing regimens evaluated, including once-weekly intravenous (IV) dosing and a more frequent intermittent IV schedule
- Potential recommended Phase 2 dose as a monotherapy for ESCC and HNSCC
- Currently on partial clinical hold to amend the protocol for additional safety monitoring based on the predefined DLT (dose limiting toxicity) stopping criteria
- Patients receiving clinical benefit continue to be dosed
- Pending amendment agreement with FDA, plan to initiate expansion cohort in HNSCC and ESCC patients with 11q13 amplifications

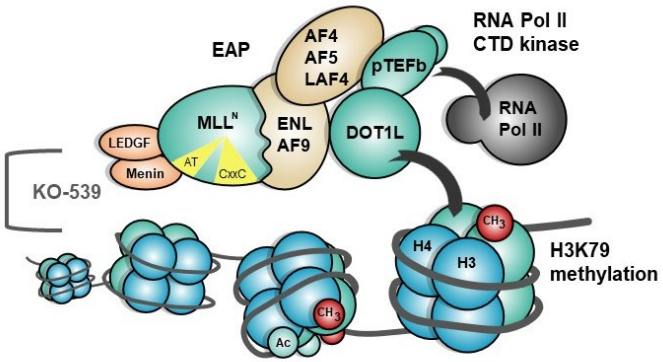
01 *Tipifarnib (Farnesyl Transferase Inhibitor)*

02 *KO-947 (ERK Inhibitor)*

03 • *KO-539 (Menin-MLL Inhibitor)*

KO-539: Potent Inhibitor of Menin-MLL Interaction

- Potent, selective small molecule inhibitor of the menin-MLL protein-protein interaction
- Robust antitumor activity observed in mixed lineage leukemias rearranged (MLL-r) as well as disseminated NPM1 mutant and DNMT3A mutant AML PDX models
- Preliminary preclinical data suggests anti-leukemic activity by induction of myeloid differentiation in AML blasts
- NPM1, MLL-r and MLL-PTD mutations occur in ~40% of AML patients[1]
- Granted Orphan Drug Designation for the treatment of AML in July 2019
- Phase 1/2 dose-escalation trial ongoing

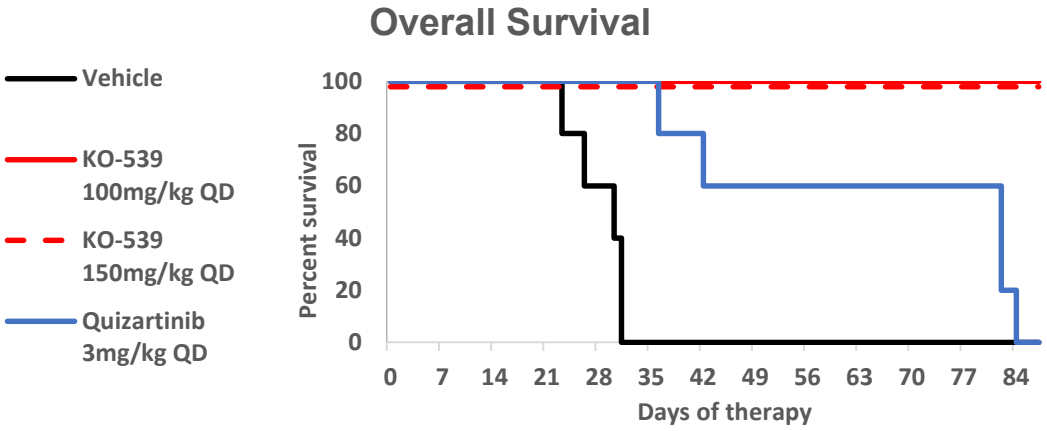


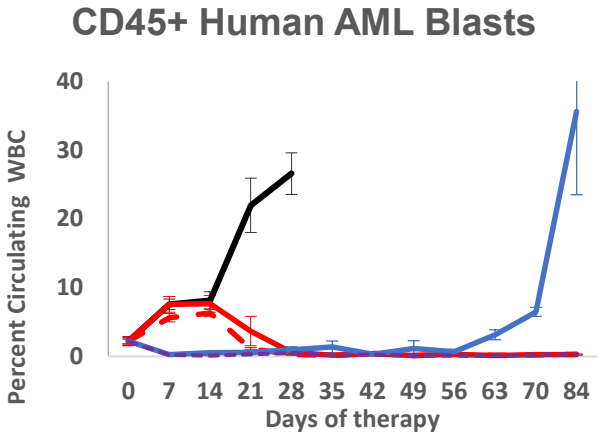
The menin-MLL complex appears to be a central node in epigenetic dysregulation driven by several distinct oncogenic driver mutations important in diverse leukemias and myeloproliferative disorders

[1] N Engl J Med. 2012 Mar 22;366(12):1079-89 | Haematologica. 2007 Jun;92(6):744-52 | Anticancer Res. 2005 May-Jun;25(3B):1931-44

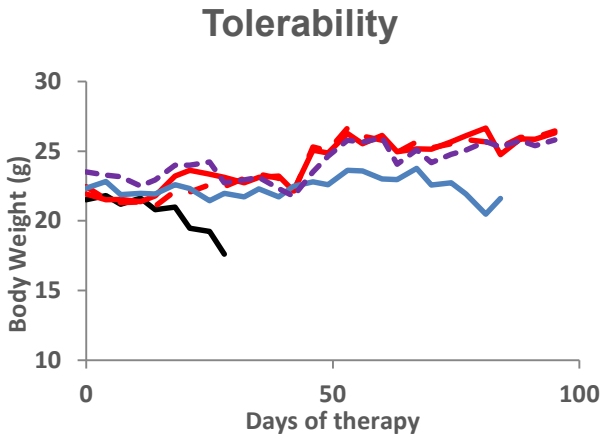
KO-539 Produces Lasting Complete Remissions in a NPM1 / DNMT3A / IDH2 / FLT3-Mutant AML Model

AM7577





- 100% (10/10) of animals treated with single-agent 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 was initially active, but all animals eventually relapsed



KO-539: Phase 1/2 Clinical Trial

- First-in-human study in patients with relapsed/refractory AML
- First patient dosed in September 2019
- Administered as a once daily oral dose in 28-continuous-day cycles
- Dose escalation objectives:
  - Determine recommended Phase 2 dose and/or MTD
  - Investigate safety and tolerability
  - Characterize pharmacokinetics
  - Assess early evidence of antitumor activity
- Anticipate reaching RP2D with potential to enrich in NPM1-mutant AML and MLL-rearranged genetically defined subgroups this year

Forecasted Milestones & Financial Highlights

Program		Milestone	Status
Tipifarnib Farnesyl Transferase Inhibitor	HRAS Mutant Indications	Data from Phase 2 trial in urothelial carcinoma	2020
		Potential for full enrollment in AIM-HN	Q1 2021
	CXCL12 Pathway Indications	Data from Phase 2 trial in CMML	2020
		Initiation of registration-directed trial in T-cell lymphoma	2H 2020
		Initiation of proof-of-concept study in pancreatic cancer	2H 2020
KO-947 ERK Inhibitor		Open 11q13-amplified HNSCC/ESCC expansion cohort	2020
KO-539 Menin-MLL Inhibitor		Achievement of recommended Phase 2 dose	2020

Financial Highlights	Nasdaq: KURA
	Shares outstanding: 45.4M basic, 4.1M options*
	Cash, cash equivalents and short-term investments: $236.9M*

* As of December 31, 2019

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Developing Precision Medicines for the Treatment of Cancer