UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
WASHINGTON, D.C. 20549

FORM 8-K

CURRENT REPORT

Pursuant to Section 13 or 15(d) of the Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): March 6, 2017

KURA ONCOLOGY, INC.

(Exact name of Registrant as Specified in Its Charter)

Delaware	**001-37620**	**61-1547851**
(State or Other Jurisdiction of Incorporation)	**(Commission File Number)**	**(IRS Employer Identification No.)**

11119 North Torrey Pines Road, Suite 125 **La Jolla, CA** **(Address of Principal Executive Offices)**	**92037** **(Zip Code)**

Registrant's Telephone Number, Including Area Code: (858) 500-8800

Not Applicable
(Former Name or Former Address, if Changed Since Last Report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions (see General Instructions A.2. below):

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Item 7.01 Regulation FD Disclosure.

Beginning on March 6, 2017, members of the management team of Kura Oncology, Inc. (the "Company") will be providing presentation materials (the "Presentation") to certain interested parties. A copy of the Presentation is attached hereto as Exhibit 99.1 and incorporated herein by reference.

The information contained in this Current Report on Form 8-K and in the accompanying Exhibit 99.1 are being furnished and shall not be deemed to be "filed" for the purposes of Section 18 of the Securities and Exchange Act of 1934, as amended, or otherwise subject to the liabilities of that section and will not be incorporated by reference into any registration statement filed by the Company, under the Securities Act of 1933, as amended, unless specifically identified as being incorporated therein by reference. This Current Report on Form 8-K will not be deemed an admission as to the materiality of any information in this Current Report on Form 8-K that is being disclosed pursuant to Regulation FD.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits.

Exhibit Number	Description
99.1	Presentation Materials of Kura Oncology, Inc.

SIGNATURE

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned thereunto duly authorized.

KURA ONCOLOGY, INC.

Date: March 6, 2017

By: /s/ Annette North

Annette North
SVP, General Counsel

Exhibit 99.1

Corporate Overview

March 2017

KURA ONCOLOGY

DEVELOPING PRECISION MEDICINES TO TREAT CANCER

Forward Looking Statements

This presentation contains forward-looking statements. Such statements include, but are not limited to, statements regarding our research, pre-clinical 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 "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 future 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; we may not be able to obtain additional financing. New risk factors 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 Opportunity: Kura Oncology

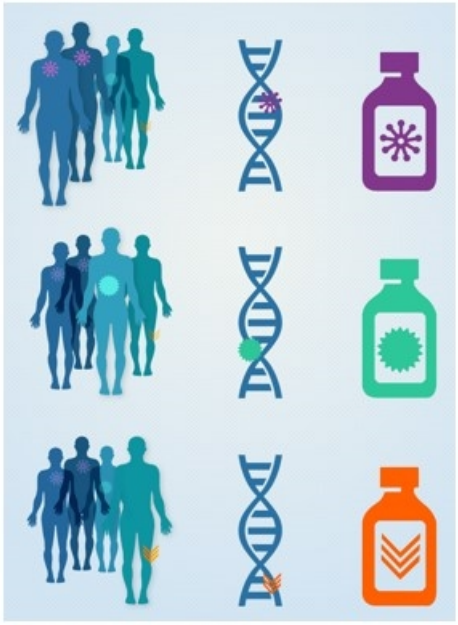
- Product candidates focused on indications with **significant sales potential** to support future commercial build out
- Lead program, tipifarnib, in **multiple Phase 2 trials**
- Multiple **data readouts anticipated in 2017** with potential to initiate first pivotal study in 2018
- Pipeline programs **advancing**
- Solid financials with **$74.6M cash** as of Sept. 30, 2016*; resources expected to fund current operations into 2018

KURA ONCOLOGY

* Includes Cash, Cash Equivalents, and Short-Term Investments

Precision Medicines in Cancer Treatment

Discovery and development of targeted therapies that treat cancer based upon the specific molecular or genetic characteristics of the patient's tumor



Source: www.cancer.gov

ADVANTAGES:

- High translatability from preclinical to clinical studies
- Leverage clinical and pathology trends towards comprehensive tumor profiling
- Potential to drive enhanced efficacy and better tolerability
- Potential for expedited clinical development

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Rapid Progress Since Inception and Multiple Near-term Milestones

2015

- ✓ Initiated P2 HRAS trial for tipifarnib
- ✓ Listed on NASDAQ
- ✓ Initiated P2 PTCL trial

2016

- ✓ Initiated P2 lower-risk MDS trial
- ✓ Reported positive preliminary data from P2 HRAS trial
- ✓ IND accepted for KO-947
- ✓ KO-539 selected as development candidate

2017 (ANTICIPATED)

1H 2017

- ✓ Initiated P2 CMML trial
- ❑ Initiate P1 trial for KO-947
- ❑ Translational data for tipifarnib, KO-947 and KO-539
- ❑ Additional data from P2 HRAS trial
- ❑ Data from PTCL P2 trial

2H 2017

- ❑ Additional data from P2 HRAS trial
- ❑ Data from lower-risk MDS trial

2018 (ANTICIPATED)

- ❑ Potential to initiate first pivotal trial for tipifarnib
- ❑ Data from P2 CMML trial
- ❑ Phase 1 data from KO-947
- ❑ Initiate P1 trial for KO-539

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Pipeline of Selective Drug Candidates For Genetically Defined Cancers

STAGES OF DEVELOPMENT				
PROGRAM	PRECLINICAL	PHASE 1	PHASE 2	ANTICIPATED MILESTONES
Tipifarnib (Farnesyl Transferase Inhibitor)	HRAS Mutant Solid Tumors			Data updates in 1H and 2H 2017
	Peripheral T-cell Lymphomas			Data in 1H 2017
	Lower-risk Myelodysplastic Syndromes			Data in 2H 2017
	Chronic Myelomonocytic Leukemia			Data in 1H 2018
KO-947 (ERK Inhibitor)				Phase 1 Initiation in 1H 2017
KO-539 (Menin-MLL Inhibitor)				Phase 1 Initiation in 2018

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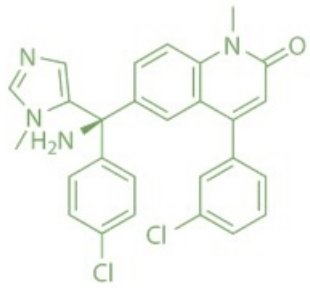
Tipifarnib

(Farnesyl Transferase Inhibitor)

Tipifarnib: A Drug Candidate Developed Ahead of Its Time

- Targeted therapy developed before the advent of personalized medicine approaches
- Extremely potent and highly selective inhibitor of protein farnesylation
- In-licensed from Janssen



CAPITALIZING ON PREVIOUS CLINICAL EXPERIENCE

- > 5,000 patients treated
- Manageable safety profile as single agent therapy
- Objective responses observed with evidence of durable clinical benefit

GOALS TO ADVANCE AS A PRECISION MEDICINE

- Confirm clinical activity
- Validate biomarker hypotheses
- Optimize dose and schedule
- Build data package supporting advancement to pivotal study

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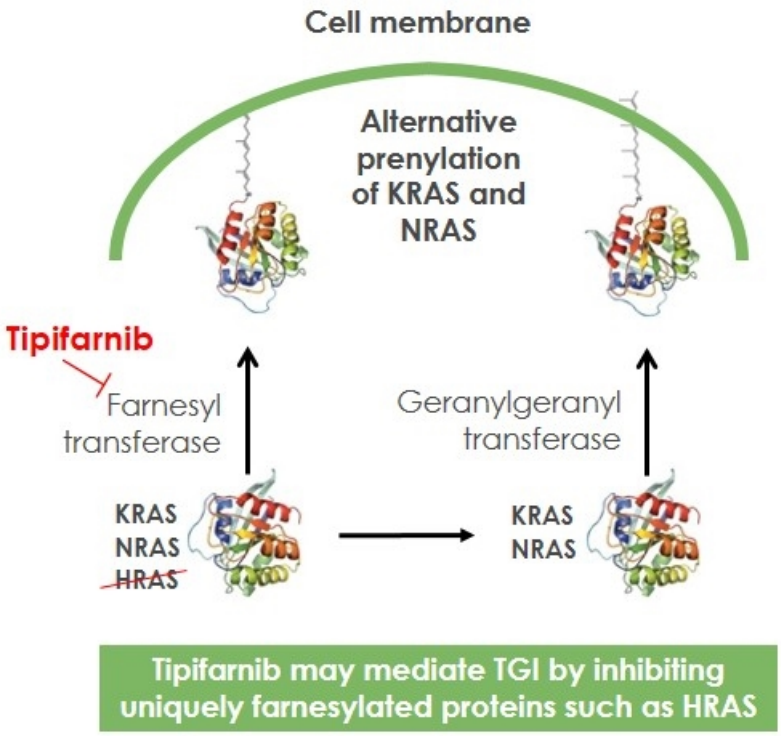
Mechanism of Action of Tipifarnib

- Farnesyl transferase (FT) enzyme attaches farnesyl group to proteins, facilitating localization to the inner membrane of the cell
- FT targets include members of the Ras superfamily (KRAS/NRAS/HRAS) and other proteins critical for cell signaling
- Blocking farnesylation prevents HRAS membrane localization, whereas KRAS and NRAS have an alternate pathway in geranylgeranylation



Tipifarnib may mediate TGI by inhibiting uniquely farnesylated proteins such as HRAS

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Multiple Shots on Goal Position Tipifarnib Favorably For a First Pivotal Trial

4 ONGOING KURA PHASE 2 TRIALS	SUCCESS CRITERA	OUTCOME: 1 OR MORE PIVOTAL TRIALS
HRAS Mutant Tumors PTCL Lower-risk MDS CMML	❑ Biomarker validation ❑ Evidence of durable, clinical benefit ❑ Sufficient ORR ❑ Potential for rapid clinical development ❑ Opportunity to move into earlier lines of therapy ❑ Attractive U.S. oncology commercial market ❑ Potential for regulatory exclusivity and/or patent protection	Potential Pivotal Trial

Objective responses with evidence of durable clinical benefit previously observed in each of the disease indications

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Phase 2 Trial in HRAS Mutant Solid Tumors

RATIONALE:

- Preclinical data supports role of HRAS as a tumor oncogene
- Murine models suggested tumor growth inhibition
- Small Phase 2 trial to evaluate whether HRAS mutant tumors would respond to tipifarnib and nature of response (regression versus disease stabilization)

DESIGN OF CURRENT PHASE 2 CLINICAL TRIAL:

- 36 patient study in two 18-patient cohorts with a Simon two-stage design
  - Cohort 1: HRAS mutant thyroid cancers
  - Cohort 2: HRAS mutant solid tumors
- Two responses required in stage 1 (n = 11) to enroll stage 2 (n=7)
- Primary objective: ORR
- Stage 2 of Cohort 2 focused on HRAS mutant SCCHN

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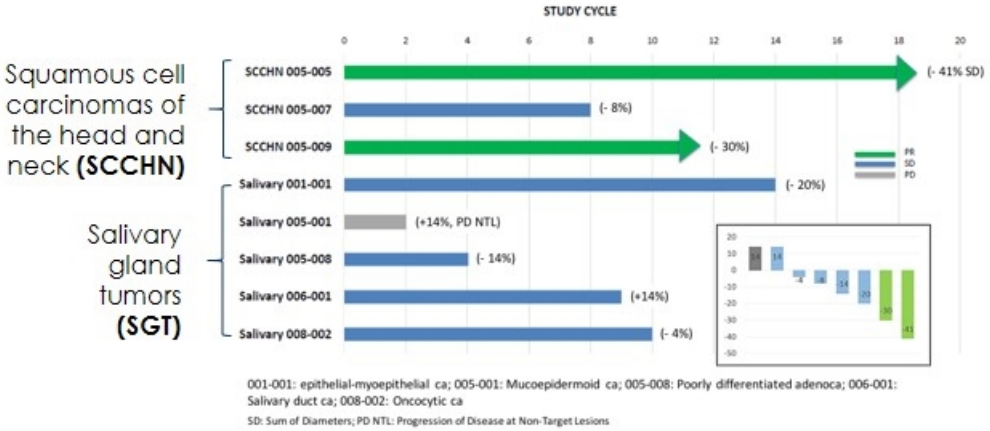
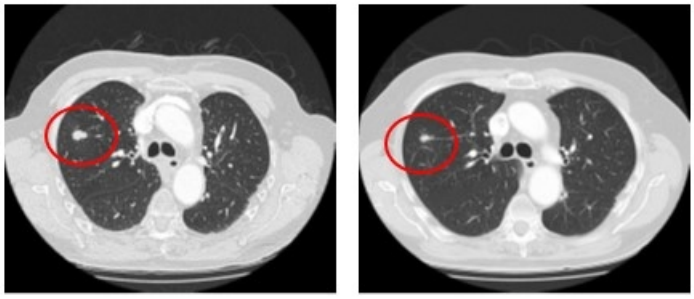
Preliminary Phase 2 Data Supports HRAS Hypothesis For Tipifarnib



001-001: epithelial-myoepithelial ca; 005-001: Mucoepidermoid ca; 005-008: Poorly differentiated adenoca; 006-001: Salivary duct ca; 008-002: Oncocytic ca

SD: Sum of Diameters; PD NTL: Progression of Disease at Non-Target Lesions

Patient 005-005
CT scans courtesy of Dr. Ho, MSKCC



08/17/2015 (Baseline) 12/22/2015 (C4 D22)

- Study has proceeded to 2nd stage for cohort 2 and been amended to enroll additional 7 patients with **HRAS mutant SCCHN**
- Cohort 1 in HRAS mutant thyroid carcinomas still enrolling in 1st stage
- Generally well tolerated, AEs consistent with the known safety profile
- **Encouraging signals of clinical activity, in patients with HRAS mutant SCCHN**

*Preliminary data from Cohort 2, Stage 1, as of 2/28/2017

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HRAS Mutant Squamous Tumors Appear More Sensitive to Tipifarnib than to SOC

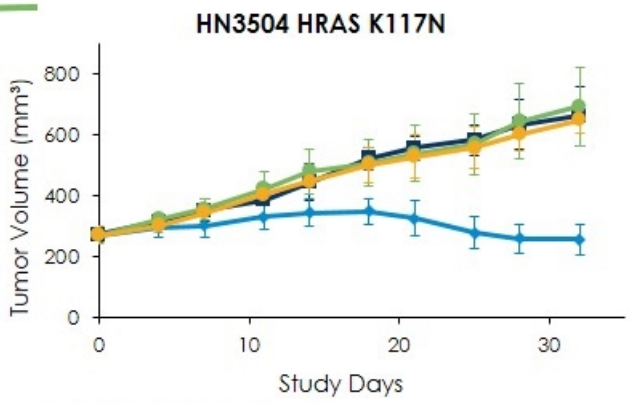
CLINICAL DATA*

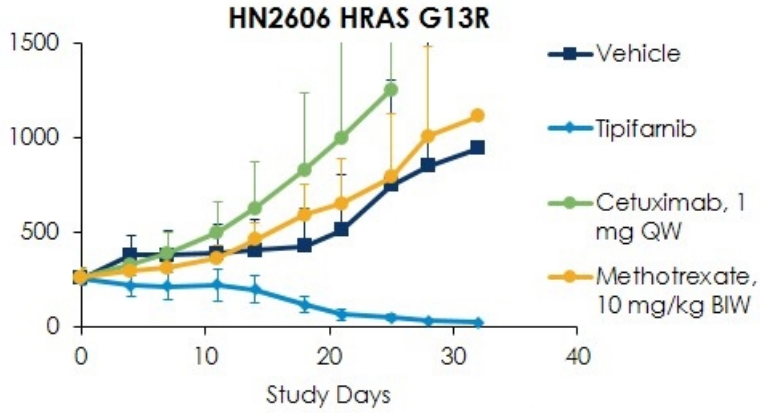
Patient 005-005 (metastatic tracheal tumor)
~ 6 mos. SD (systemic) → Cetuximab/Carboplatin/paclitaxel/RT → ~ 8 mos. SD → PD → PR C2 → Tipifarnib → **Ongoing in C19**

Patient 005-007 (oral cavity SCCNH)
Cetuximab → PD C2 → Tipifarnib → SD C2 → SD C6 → **Censored**

Patient 005-009 (oral cavity SCCNH)
Cetuximab/Paclitaxel → PD C2 → Tipifarnib → PR C2 → **Ongoing in C12**

PRECLINICAL DATA





- *Data source: Kura Oncology*

*Preliminary data from Cohort 2, Stage 1, as of 2/28/2017

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HRAS Mutant SCCHN Represents Significant Unmet Medical Need

- SCCHN comprises different malignant tumors that develop in or around the throat, larynx, nose, sinuses, and mouth.
- Estimated incidence of SCCHN in the U.S. is 56,000 in 2017
  - Estimated frequency of HRAS mutations in SCCHN patients ~ 5-6%
  - HRAS-mediated resistance to anti-EGFR therapies may drive higher numbers

	Keytruda (Pembrolizumab) Merck	Opdivo (Nivolumab) BMS/ONO Pharma		Erbitux (Cetuximab) Eli Lilly
Efficacy Study	Single Arm[1] N = 174	MTX/Doc/Cetu[2] N = 361		Single Arm[3] N = 103
		Active	**Control**	
ORR	16%	13.3%	5.8%	13%
Median OS	--	7.5 mo	5.1 mo	--

[1] Keytruda Package Insert
[2] Opdivo Package Insert
[3] J Clin Oncol. 2007 Jun 1;25(16):2171-7

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Phase 2 Trial in HRAS Mutant Solid Tumors

EVOLVING SCCHN TREATMENT LANDSCAPE

- Recruitment of SCCHN patients has become more challenging post-approval of I/O agents
  - I/O agents have become standard-of-care in the U.S. in the overall SCCHN population
  - Post-I/O patients often have very advanced disease

KURA IS AGRESSIVELY OPENING EX-U.S. SITES AND IS FACILITATING HRAS SCREENING AT CLINICAL SITES

- Kura working to open additional clinical sites in Europe and Asia, where I/O agents are not approved/reimbursed
- Working with academic sites to facilitate screening of tumor samples both in U.S. and ex-U.S
- Contracting with additional labs to facilitate HRAS testing for sites that do not have sequencing capability

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Defined Patient Populations Are Actionable with Targeted Therapies

ONCOGENE	INDICATION	U.S. INCIDENCE	APPROVED DRUGS	2016 REVENUES[1]
Bcr-Abl	CML	~ 9,000	Imatinib, Nilotnib, Dasatinib, Bosutinib, Ponatinib	> $4,000 M
ALK	NSCLC	~ 9,000	Crizotinib, Ceritinib, Alectinib	~ $800M
BRAF	Malignant melanoma	~ 5,000	Vemurafenib, Dabrafinib	~$500M
PARP	Ovarian	~ 3,000	Olaparib, Rucaparib, Niraparib	~$250M

ONCOGENE	INDICATION	U.S. INCIDENCE	DRUG CANDIDATE
IDH1/2	AML	~ 3,000-5,000	AG-120, AG-221, AG-881
TRK+	Various	~1,500-5,000	Larotrectinib, Entrectinib
HRAS	SCCHN Sq-NSCLC	~ 2,800-3,400 ~1,000-1,700	Tipifarnib

[1]2016 global revenue estimates developed by third party market research

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Kura is On Track to Achieve Success Criteria for the First Phase 2 Trial

4 ONGOING KURA PHASE 2 TRIALS	SUCCESS CRITERA	OUTCOME: 1 OR MORE PIVOTAL TRIALS
HRAS Mutant Tumors	✓ Biomarker validation ✓ Evidence of durable, clinical benefit ❑ Sufficient ORR ❑ Potential for rapid clinical development ✓ Opportunity to move into earlier lines of therapy ✓ Attractive U.S. oncology commercial market ❑ Potential for regulatory exclusivity and/or patent protection	Potential Pivotal Trial **
PTCL		
Lower-risk MDS		
CMML		

* Kura is currently prosecuting patent applications to cover HRAS indication(s)

** Potential for registration-enabling study of tipifarnib in relapsed and/or refractory HRAS mutant SCCHN subject to data from 2nd stage of ongoing Phase 2 trial

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Three Additional Tipifarnib Phase 2 Trials Ongoing with 2 Data Readouts Anticipated in 2017

	PTCL	LOWER RISK MDS	CMML
Subjects	n=18 (Potential for expansion to n=30)	n = up to 58	n ~ 20
Primary Endpoints	ORR (IWC)	RBC transfusion independence	ORR using MDS/MPN IWG criteria
Rationale	• Prior Phase 2 experience • Patient biomarker analysis	• Prior Phase 2 experience • Patient biomarker analysis	• Prior Phase 2 experience • Patient biomarker analysis
Biomarkers	Exploratory	NK cell markers, including KIR2DS2	NRAS/KRAS wild-type versus mutant
Est. U.S. Incidence	5,000	9,750	1,100
Milestone	**Data 1H 2017**	**Data 2H 2017**	**Data 1H 2018**

SUCCESS CRITERA

- ❑ Biomarker validation
- ❑ Evidence of durable, clinical benefit
- ❑ Sufficient ORR
- ❑ Potential for rapid clinical development
- ❑ Opportunity to move into earlier lines of therapy
- ❑ Attractive U.S. oncology commercial market
- ❑ Potential for regulatory exclusivity and/or patent protection

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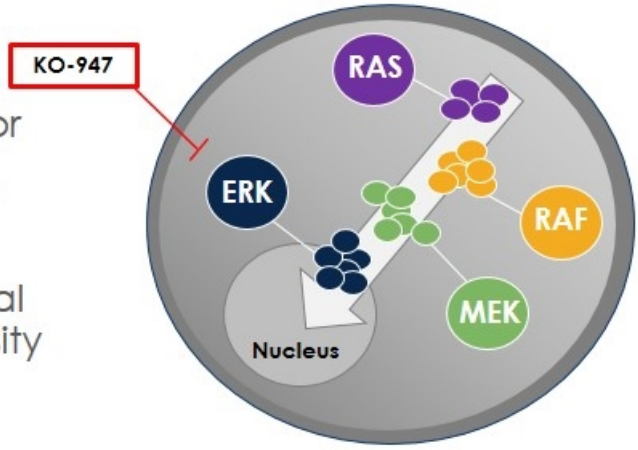
KO-947
(ERK Inhibitor)

KO-947: Potent Inhibitor of Extracellular Signal-regulated Kinase (ERK)

- Aberrant signaling caused by mutations or dysregulation of the MAPK pathway associated with numerous tumor types
- Inhibitors of RAF and MEK have validated the MAPK pathway in cancer
- Competitors have demonstrated clinical activity in selected patients, but it has been challenging to drive durable PD and clinical activity

KO-947

- Potent and selective ERK inhibitor
- Prolonged pathway modulation enables intermittent dosing
- IV route selected for initial clinical study to drive higher dose intensity



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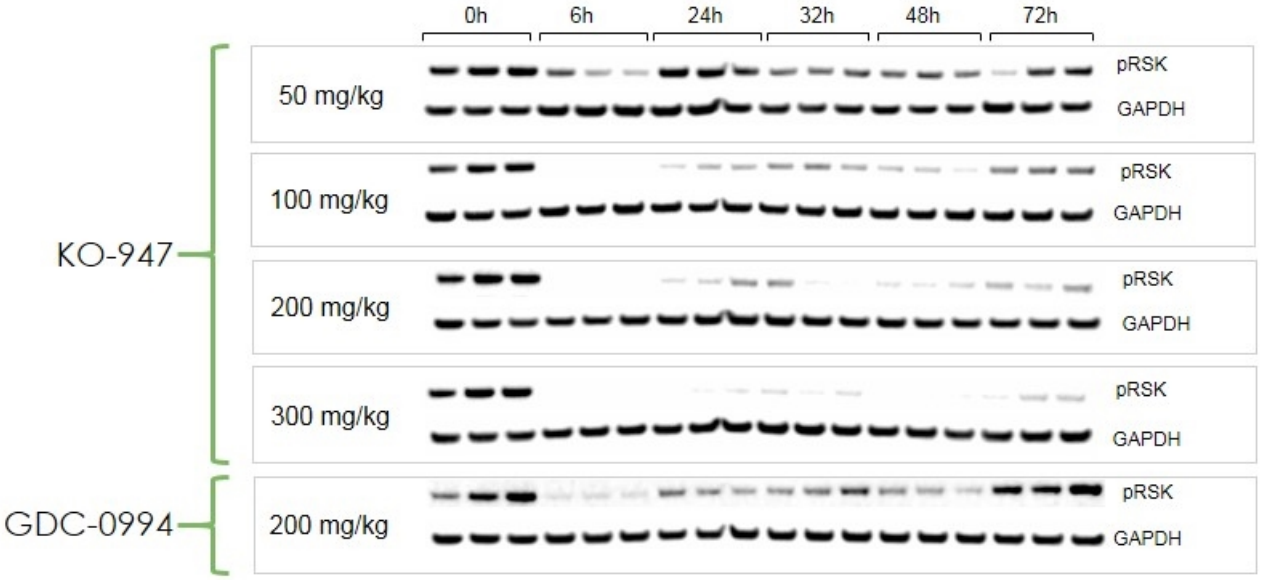
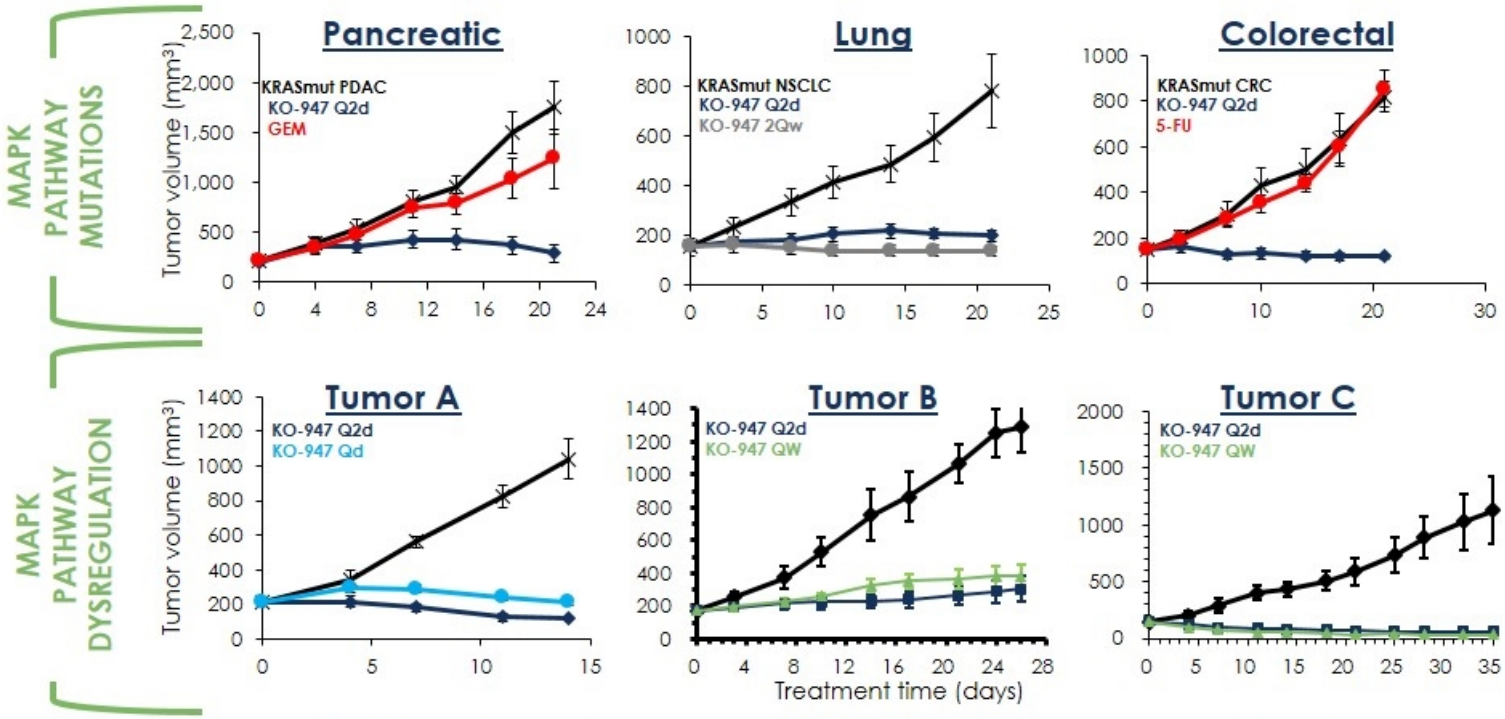
KO-947 Demonstrates Prolonged MAPK Pathway Modulation In Vivo

Pharmacodynamic Modulation After a Single Oral Dose KRAS H2122 Model



Extended pharmacology of KO-947 supports potential for intermittent dosing schedules

KO-947: Translational Research Identified Potential Lead Clinical Indications



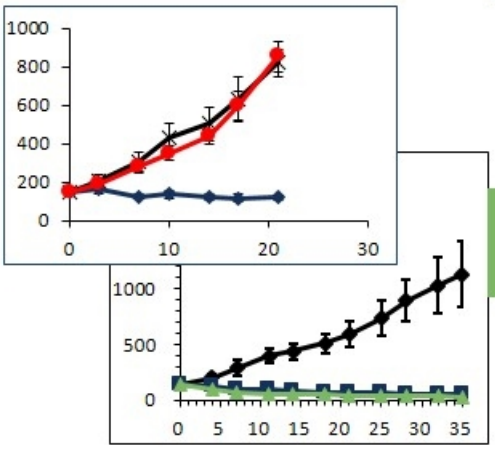
- Robust anti-tumor activity demonstrated in two broad tumor classes with > 50% response rates in preclinical models
- Potential biomarkers have been identified to support development
- Evaluated KO-947 in 138 PDX models across 20 potential indications

Kura Oncology data

KURA ONCOLOGY

KURA is Pursuing a Precision Medicine-Based Approach Toward Development of KO-947

PRECLINICAL DATA



PHASE 1
Anticipated 1H 2017

PHASE 1b/2

SUCCESS CRITERA

- ❑ Biomarker validation
- ❑ Evidence of durable, clinical benefit
- ❑ Sufficient ORR
- ❑ Potential for rapid clinical development
- ❑ Opportunity to move into earlier lines of therapy
- ❑ Attractive U.S. oncology commercial market
- ❑ Potential for regulatory exclusivity and/or patent protection

GOAL

Indications with potential for single agent activity, enabling accelerated development

ADVANTAGES OF A PRECISION MEDICINE-BASED APPROACH

- High potential for translatability from preclinical to clinical studies
- Leverage clinical and pathology trends towards comprehensive tumor profiling
- Meaningful single agent activity may permit more rapid clinical development

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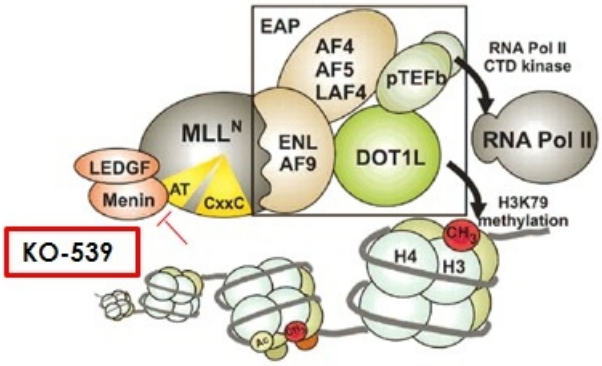
KO-539
(Menin-MLL Inhibitor)

KO-539: Potential First-in-class Inhibitor of the Menin-MLL Interaction

- Chromosomal translocations of the MLL gene play a causative role in the onset, development and progression of a subset of acute leukemias
- MLL-r fusion proteins and a similar mutation, MLL partial tandem duplication, drive overexpression of leukemogenic proteins
- Leukemogenic activity of MLL is critically dependent on binding the protein menin
- Estimated U.S. incidence of 3,500 patients with MLL-rearranged and MLL-PTD acute leukemias (AML and ALL)
- Opportunities to target menin dysregulation in additional tumor types
- Licensed worldwide rights from University of Michigan



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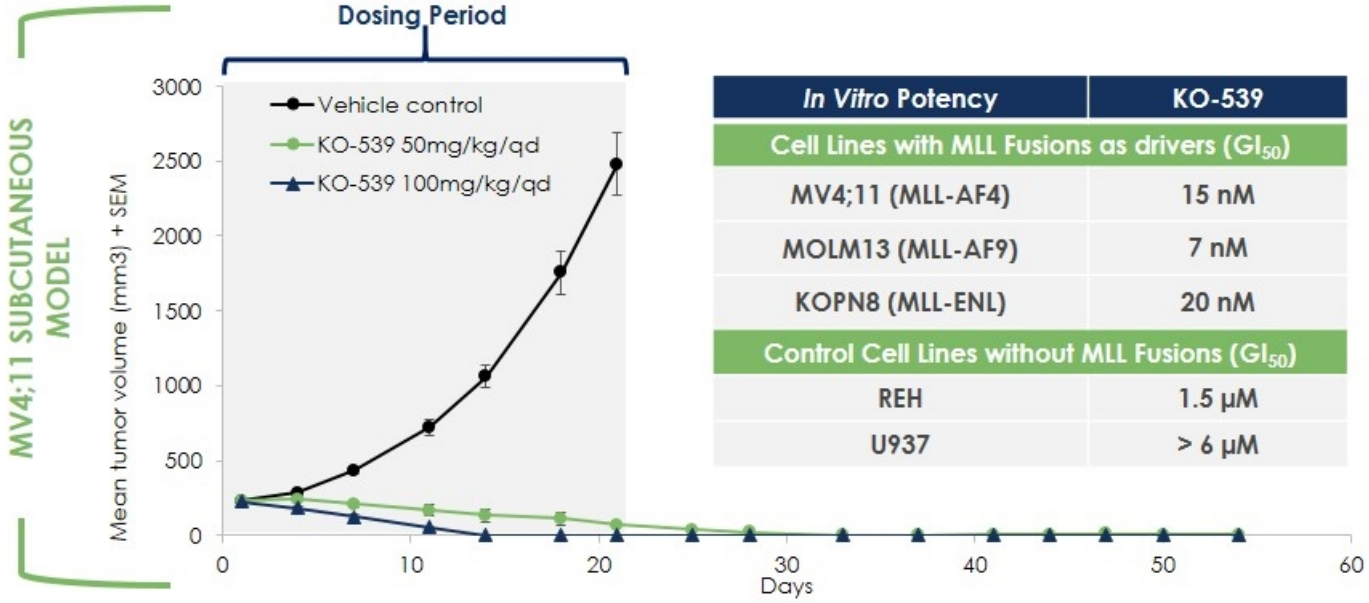
KO-539 Displays Prolonged Efficacy in Xenograft Model



In Vitro Potency	KO-539
Cell Lines with MLL Fusions as drivers (GI_{50})	
MV4;11 (MLL-AF4)	15 nM
MOLM13 (MLL-AF9)	7 nM
KOPN8 (MLL-ENL)	20 nM
Control Cell Lines without MLL Fusions (GI_{50})	
REH	1.5 µM
U937	> 6 µM

- KO-539 is a potent and selective inhibitor of the menin-MLL interaction
- KO-539 demonstrated robust efficacy in *in vivo* models of MLL-r AML
- Tumor regressions sustained at 30 days following end of dosing period

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Anticipated Milestones

PROGRAM	UPDATE	ESTIMATED TIMEFRAME
Tipifarnib (Farnesyl Transferase Inhibitor)	Additional data from Phase 2 study in HRAS mutant SCCHN	**1H and 2H 2017**
	Data from Phase 2 study in PTCL	**1H 2017**
	Data from Phase 2 clinical trial in lower risk MDS	**2H 2017**
	Data from Phase 2 clinical trial in CMML	**1H 2018**
KO-947 (ERK Inhibitor)	Initiate Phase 1 clinical trial	**1H 2017**
	Additional translational data on KO-947	**1H 2017**
KO-539 (Menin-MLL Inhibitor)	Additional translational data on KO-539	**1H 2017**
	Initiate Phase 1 study	**2018**

KURA ONCOLOGY

Experienced Management Team

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

Antonio Gualberto, M.D., Ph.D.
Chief Medical Officer

Yi Liu, Ph.D.
Chief Scientific Officer

Heidi Henson, CPA
Chief Financial Officer

Pingda Ren, Ph.D.
SVP, Chemistry and Pharmaceutical Sciences

Annette North, LLB
SVP and General Counsel

wellspring biosciences

Pfizer

EMD SERONO

Takeda

intellikine

ambit

Genomics Institute of the Novartis Research Foundation

KURA ONCOLOGY

Board and Advisors

BOARD OF DIRECTORS	
Faheem Hasnain	Former President and CEO, Receptos, Inc.
Robert Hoffman	EVP and CFO, Innovus Pharmaceuticals, Inc.
Thomas Malley	Mossrock Capital
Steven Stein, M.D.	Chief Medical Officer, Incyte Corporation
Troy Wilson, Ph.D., J.D.	CEO and President, Kura Oncology, Inc.
SCIENTIFIC ADVISORS	
Kevin Shokat, Ph.D.	Professor and Chairman, Dept. Cellular & Molecular Pharmacology, UCSF
Frank McCormick, Ph.D., FRS	Director Emeritus of the UCSF Helen Diller Cancer Center and Professor, UCSF
Neal Rosen, M.D., Ph.D.	Director of the Center for Molecular Therapeutics at Memorial Sloan-Kettering Cancer Center
Sir Simon Campbell, CBE, FRS	Former Senior VP Worldwide Discovery & Medicinal R&D Europe at Pfizer

Why Invest in Kura Oncology?

Precision Medicine Strategy in Oncology

Advancing Therapeutic Pipeline

- Lead product candidate in multiple Phase 2 trials
  - Encouraging clinical data in ongoing Phase 2 study of HRAS mutant squamous cell carcinomas of the head and neck (SCCHN)
  - Potential to select indication for pivotal study in 2017
  - Multiple Phase 2 trials may provide additional development opportunities
- Preclinical programs advancing
  - IND for KO-947 accepted; Phase 1 anticipated 1H 2017
  - KO-539 advancing as development candidate for menin-MLL program

Solid Financials

- NASDAQ: KURA
- Shares Outstanding*: 19.0M basic, 1.2M options ($6.43 weighted avg strike price)
- $74.6M cash as of September 30, 2016**; resources expected to fund current operations into 2018

* Excludes 2.3M shares of common stock subject to repurchase as of Sept 30, 2016; weighted average strike price as of Sept 30, 2016

** Includes Cash, Cash Equivalents, and Short-Term Investments

KURA ONCOLOGY

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DEVELOPING PRECISION MEDICINES TO TREAT CANCER