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): September 7, 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.)**

3033 Science Park Road, Suite 220, San Diego, CA	**92121**
(Address of Principal Executive Offices)	**(Zip Code)**

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

N/A
(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))

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§ 230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§ 240.12b-2 of this chapter).

Emerging growth company ☒

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☒

Item 7.01 Regulation FD Disclosure.

Beginning on September 7, 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 Item 7.01 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 8.01 Other Events.

On September 7, 2017, the Company issued a press release announcing positive topline results from a Phase 2 trial for its lead product candidate, tipifarnib, in patients with HRAS mutant relapsed or refractory squamous cell carcinomas of the head and neck ("HNSCC"). The Phase 2 trial achieved its primary endpoint prior to the completion of enrollment. The trial protocol requires four confirmed, partial responses, per RECIST 1.1 criteria, out of 18 patients to meet its primary endpoint. Four confirmed, partial responses and two patients with disease stabilization have been observed among the first six evaluable HNSCC patients enrolled in the trial. In addition, objective responses greater than one year in duration have already been observed in two patients. All patients joined the study upon progression on prior therapy, including chemotherapy, cetuximab or immune therapy. The Company will continue to enroll HRAS mutant HNSCC patients and plans to present data from the study at an upcoming scientific or medical conference.

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: September 7, 2017

By: /s/ Annette North

Annette North
Senior Vice President and General Counsel

Exhibit Index

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

Exhibit 99.1

Corporate Presentation

September 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.

Kura Oncology Investment Highlights

- Developing **precision medicines for cancer** in diseases with **significant commercial potential**
- Lead program tipifarnib in **multiple Phase 2 trials**
  - Positive Phase 2 trial in head and neck squamous cell carcinomas (HNSCC), reported in September 2017
  - Additional data readouts anticipated in 2017 and 2018
  - Potential to initiate first pivotal study in 2018
  - Patent provides exclusivity in HRAS HNSCC indication to 2036
- **Pipeline programs:** ERK inhibitor in Phase 1 trial with data expected in 2018 and menin-MLL inhibitor in preclinical development
- **Strong cash position:** $53.2 million as of June 30, 2017*; August 2017 financing with net proceeds of approx. $53.2 million
- **Highly experienced oncology drug development team**

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* Cash, cash equivalents and short-term investments

Leadership Team

Proven oncology drug discovery and development expertise

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

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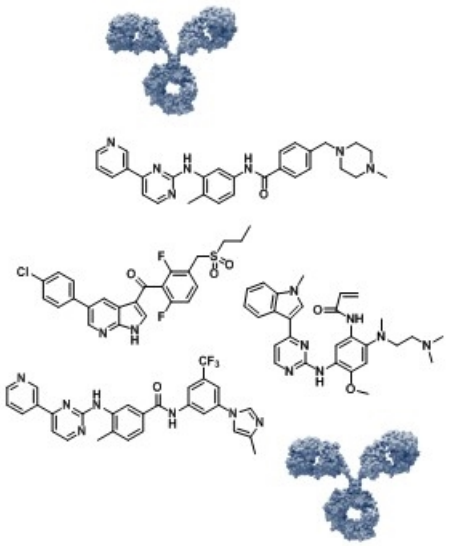
Biomarkers Have Potential to Unlock the Value of Precision Medicines



Precision Medicines

+

Protein expression

Genetics

Histology

Biomarkers

Enhanced Clinical Benefit for Patients

- Zelboraf® vemurafenib (BRAF V600E)
- Herceptin® trastuzumab (HER2)
- glivec® imatinib (BCR-ABL)
- TAGRISSO™ osimertinib (EGFR T790M)
- Tasigna® nilotinib (BCR-ABL)
- KEYTRUDA® (PD-1)

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Product Candidate Pipeline

PROGRAM	PRECLINICAL	PHASE 1	PHASE 2
Tipifarnib (Farnesyl Transferase Inhibitor)	HRAS Mutant Solid Tumors		
	Peripheral T-cell Lymphomas		
	Myelodysplastic Syndromes		
	Chronic Myelomonocytic Leukemia		
KO-947 (ERK Inhibitor)	Solid Tumors		
KO-539 (Menin-MLL Inhibitor)	Liquid Tumors		

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Pipeline Targeting Large Market Opportunity

Product Candidate	Tumor Type	Target Population
Tipifarnib (Phase 2)	$HRAS^{mut}$ HNSCC	2,800-3,400
	$HRAS^{mut}$ Sq-NSCLC	1,000-1,700
	PTCL	1,500-2,500*
	MDS	15,000-18,000**
	CMML	750*
KO-947 (Phase 1)	HNSCC	10,000-20,000*
	$KRAS^{mut}$ NSCLC	23,000
	$BRAF^{mut}$ NSCLC	5,000
KO-539 (Preclinical)	MLL-rearranged leukemias	3,500

* Biomarker still under evaluation for these indications; these reflect estimates of the biomarker-positive subset for each indication.

** Estimate of the prevalence of MDS patients (approx. 50,000-60,000 patients in the U.S.) who would be positive for a tipifarnib biomarker.

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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*

- ✓ Initiated P2 CMML trial
- ✓ Additional data from P2 HRAS trial
- ✓ Translational data for tipifarnib, KO-947 and KO-539
- ✓ Initiated P1 trial for KO-947
- ✓ Efficacy and biomarker data from PTCL P2 trial
- ✓ U.S. patent for use of tipifarnib in HRAS mutant HNSCC
- ✓ Additional data from P2 HRAS trial
- ❑ Additional data from P2 PTCL trial

2018*

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

* Anticipated milestones

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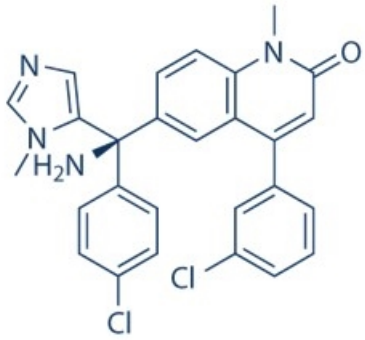
Tipifarnib in HRAS Mutant Solid Tumors

Tipifarnib: Unlocking Value in a Lower Risk Asset

SUBSTANTIAL CLINICAL EXPERIENCE

- Extremely potent and selective inhibitor of protein farnesylation
- Well characterized > 5,000 patients with activity in subpopulations
- Developed before advent of personalized medicine approaches
- Licensed from Janssen



KURA'S KEYS TO UNLOCK VALUE

- Identifying and validating biomarkers for enhanced efficacy
- Confirming activity in biomarker subsets
- Optimizing dose and schedule
- Building data package to support advancement to pivotal study

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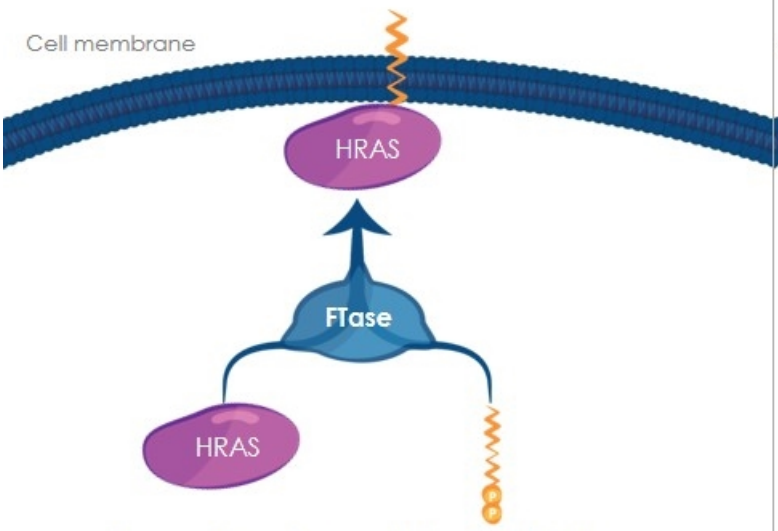
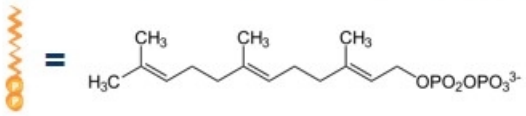
Tipifarnib Inhibits Cancer Cell Growth Signaling Pathways

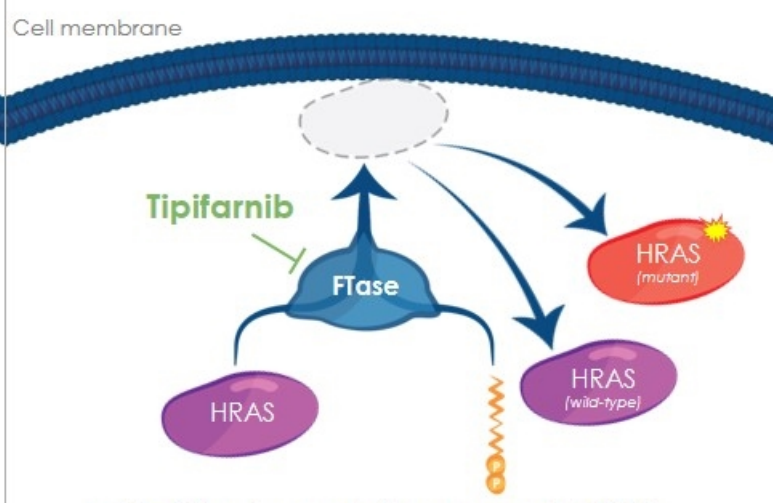
Normal FTase



- **Farnesyl transferase (FTase) attaches farnesyl group to proteins**, facilitating localization to the inner cell membrane



Tipifarnib Inhibits FTase



- Blocking farnesylation **prevents HRAS membrane localization**
- Tipifarnib can block farnesylation and membrane localization of **both wild-type and mutant HRAS proteins**

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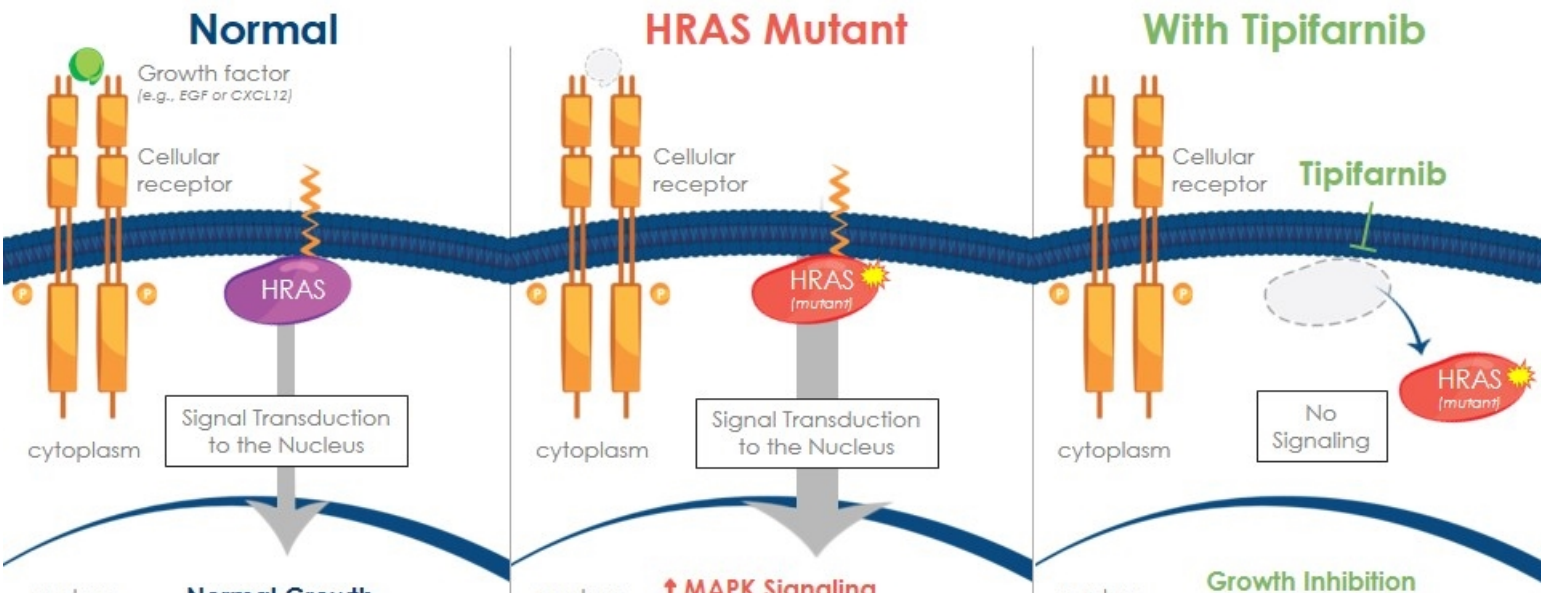
Tipifarnib Inhibits HRAS Mutant Tumors



- Growth and survival of normal cells is driven by growth factor interaction with cell receptors and intracellular signaling

- Mutation of HRAS protein can switch signaling into a permanently "on" state, making tumor growth and proliferation independent of growth factor and dependent on HRAS signaling

- Blocking farnesylation disrupts cellular signaling in the tumor cell and can induce either growth inhibition or apoptosis

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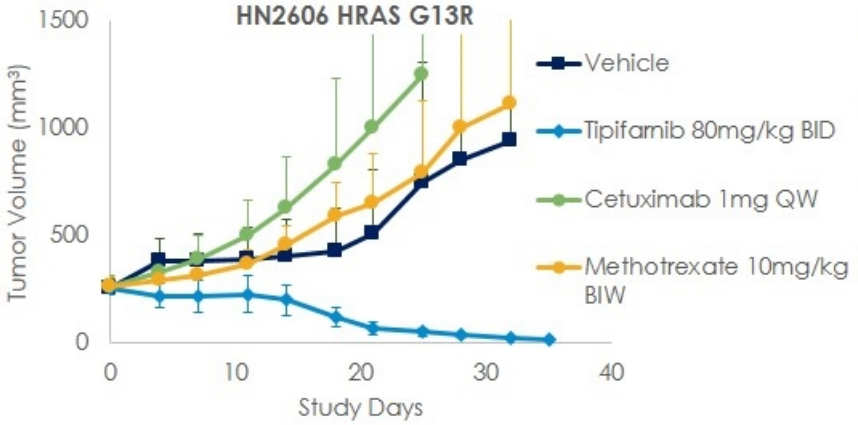
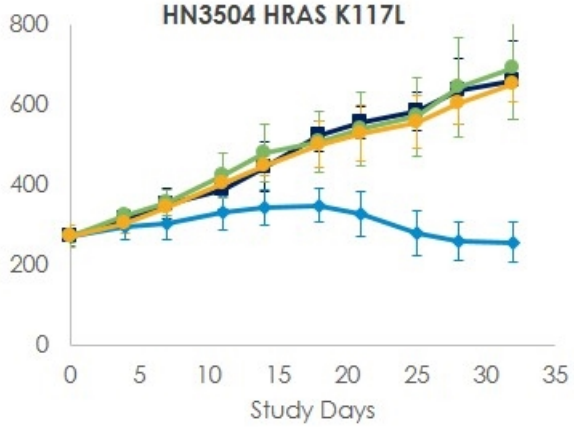
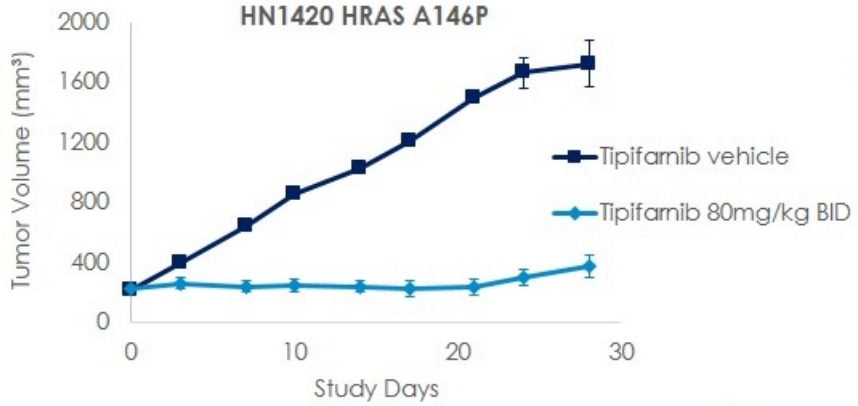
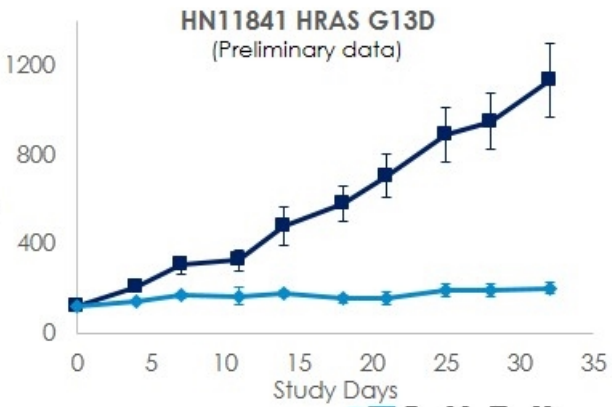
Tipifarnib is Active in Preclinical PDX Models of HRAS Mutant HNSCC









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HNSCC Represents Significant Unmet Medical Need

- HNSCC comprises different malignant tumors that develop in or around the throat, larynx, nose, sinuses and mouth
- Low response rates and limited duration of clinical benefit with existing therapeutic options for HNSCC
- Estimated incidence of HNSCC in the U.S. is 56,000 in 2017
  - Estimated frequency of HRAS mutations in HNSCC patients ~ 5-6%
  - HRAS-mediated resistance to anti-EGFR therapies may drive greater market opportunity

	Keytruda (Pembrolizumab) Merck	**Opdivo (Nivolumab) BMS/ONO Pharma**		**Erbitux (Cetuximab) Eli Lilly**
Efficacy Study	Single Arm[1] N = 174	MTX/Doc/Cetuximab[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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Tipifarnib: Phase 2 in HRAS Mutant Solid Tumors

- **Design of Phase 2 Trial**
  - Originally designed to enroll patients into two single-arm study cohorts, each with a 2-stage design (11+7 evaluable patients)
    - Cohort 1: HRAS mutant thyroid cancers
    - Cohort 2: Other (non-thyroid) HRAS mutant solid tumors
  - Primary objective: Objective response rate (ORR)[1]
- **Status:**
  - Cohort 1 (thyroid): ongoing in first stage
  - Cohort 2 (other HRAS mutant)
    - Pre-specified activity for first stage of accrual was met.
    - Based on data observed in the first stage, enrollment limited to HRAS mutant HNSCC since August 2016
    - **Trial positive in September 2017 with confirmed PR's observed in four of first six evaluable patients with HRAS mutant HNSCC**

[1] Objective response is ≥ 30% tumor shrinkage as defined in RECIST 1.1 guideline (See A. Eisenhauer *et al., Eur. J. Cancer* 45 (2009): 228-247)

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Phase 2 HRAS Mutant HNSCC – Key Takeaways

- **Positive Phase 2** results obtained in September 2017
  - Partial responses observed in **four of the first six** evaluable patients and disease stabilization observed in two remaining patients with HRAS mutant HNSCC
  - Primary endpoint has been met; patient enrollment will continue
- Patients on the study who had **failed cetuximab, with or without chemotherapy, or immune therapy**, have experienced objective partial responses upon treatment with tipifarnib
- **Patients received limited clinical benefit from prior therapies**
- Two of the responses have demonstrated **durability beyond one year**
- **Very uncommon level of activity** in the relapsed/refractory setting
- **Updated data to be presented** at an upcoming scientific or medical meeting

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Tipifarnib: Foundation Medicine Collaboration

- Leader in tissue-based next-generation sequencing (NGS) using a comprehensive genomic profiling assay
- Focused on outreach to patients with HRAS mutant HNSCC, particularly those in the community setting
- SmartTrials Precision Enrollment program will contact physicians treating individuals across the U.S. diagnosed with mutant HNSCC
- Complements ongoing efforts to open additional clinical sites and facilitate HRAS mutation screening

FOUNDATION MEDICINE

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Tipifarnib: Patent Protects Use in HRAS Mutant HNSCC Until August 2036

- U.S. patent 9,707,221 provides exclusivity in U.S. for tipifarnib in HRAS mutant HNSCC indication
- Kura is pursuing U.S. and foreign patent protection in this and other indications
- This patent illustrates potential of broader strategy to generate intellectual property related to tipifarnib and its use in treating human diseases

Claim 1: "A method of treating an H-Ras mutant head and neck squamous cell carcinoma (HNSCC) in a subject, comprising administering a therapeutically effective amount of tipifarnib to said subject, wherein said HNSCC is at an advanced stage, metastatic, relapsed or refractory, and wherein said HNSCC is human papillomavirus (HPV)-negative."

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Potential to Initiate 1st Pivotal Trial for Tipifarnib in 2018

Additional Phase 2 results anticipated

FOUR ONGOING KURA PHASE 2 TRIALS	CRITERIA FOR ADVANCEMENT TO PIVOTAL DEVELOPMENT
HRAS Mutant Tumors	✓ Biomarker validation ✓ Evidence of durable, clinical benefit
PTCL	✓ Sufficient ORR ✓ Potential for rapid clinical development
Lower-risk MDS	✓ Opportunity to move into earlier lines of therapy ✓ Attractive U.S. oncology commercial market
CMML	✓ Potential for regulatory exclusivity and/or patent protection

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Tipifarnib in Peripheral T-Cell Lymphoma (PTCL)

Tipifarnib: Phase 2 Trial in PTCL

RATIONALE:

- Diverse group of aggressive non-Hodgkin lymphomas characterized by presence of malignant T-cells or natural killer (NK) cells
- Effective options for relapsed patients are limited with objective responses
- Approved therapies show 25-30% response rate and a median PFS/TTP of 1.6-4.0 months
- Patients have a poor overall survival of about 40%
- Previous investigator-sponsored Phase 2 study at Mayo Clinic showed objective responses in patients with relapsed/refractory PTCL[1]

INITIAL DESIGN OF PHASE 2 CLINICAL TRIAL:

- 18 patient Phase 2 study with Simon two-stage design (11+7)
- Primary objective: ORR
- Exploratory biomarker assessments

1. Witzig *et al.*, 2011, *Blood* 118:4882-9.

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Preliminary Phase 2 Data Identify CXCL12 as a Potential Biomarker in PTCL

- 7/18 patients (39%) experienced PR/SD with a median of 4 prior therapies[1]
- Patients with SD had meaningful tumors size reductions[2]
- Gene expression data available for 13 patients
- Patients with tumors with high expression of CXCL12 experienced better responses and longer time to progression (TTP)
  - Differences in CXCL12 expression appeared related to genetic variation in the CXCL12 gene
  - 2/2 patients with Angioimmunoblastic T-cell lymphoma (AITL) experienced objective responses

1. Preliminary data as of July 15, 2017.
2. Only two cases met all criteria for objective response. Two patients had tumor size reductions >50% in target lesions but persistent non target lesions and splenomegaly, respectively

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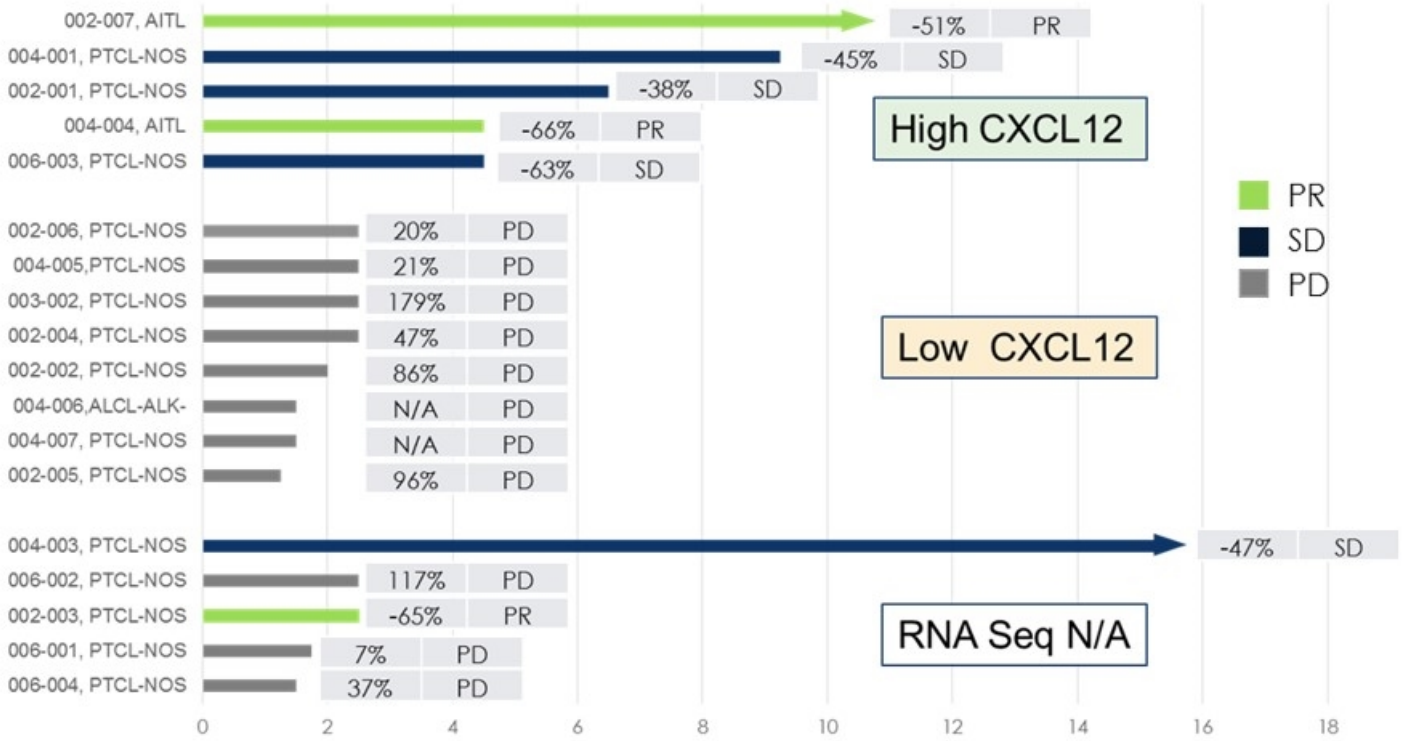
Preliminary Phase 2 Data Identify CXCL12 as a Potential Biomarker in PTCL*



* Preliminary data as of July 15, 2017

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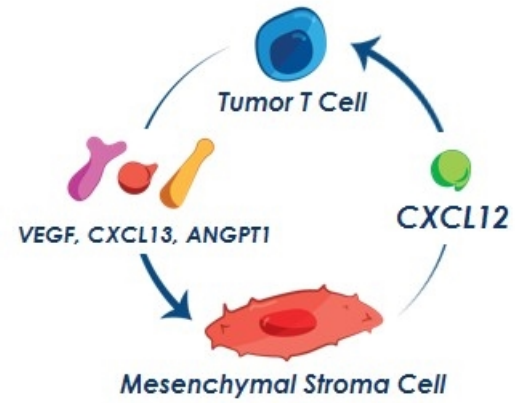
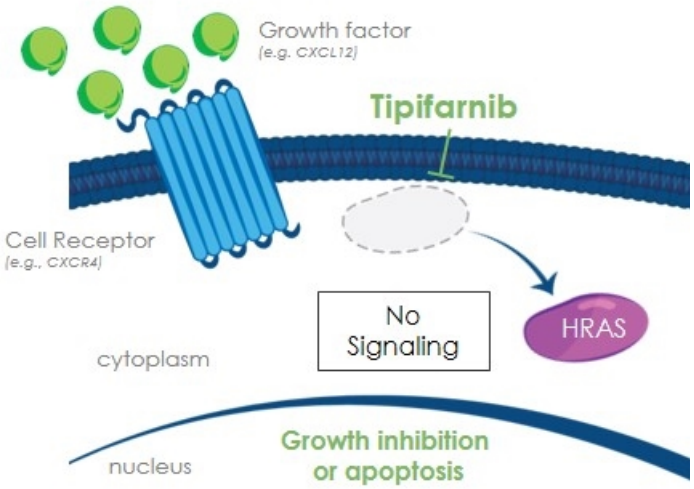
Rationale for CXCL12 Biomarker

CXCL12 is Necessary for Maintenance of T-cell Tumors



- T-cell tumors secrete growth factors for stromal cells, which in turn produce growth factors for T-cells, including CXCL12 (SDF-1)[1-5]

Blocking HRAS Farnesylation Disrupts CXCL12 Signaling



- CXCL12 postulated to signal, in part, through **HRAS**. HRAS requires farnesylation for activity.

1. *Lab Invest.* 2004; 84:1512-9
2. *Cancer Cell.* 2015 ;27:755-68
3. *Stem Cells Int.* 2015;2015:63230
4. *Haematologica.* 2014; 99:997-1005
5. *Cancer Cell* 2015; 27:755–68

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Tipifarnib in MDS and CMML

Tipifarnib: Phase 2 Trial in MDS

RATIONALE AND UNMET NEED:

- MDS are a group of blood and bone marrow disorders with both proliferative and dysplastic phenotypes characterized by ineffective hematopoiesis leading to cytopenias
- Estimated annual U.S. incidence of 13,000; estimated U.S. prevalence of 50,000-60,000 patients
- Effective options for relapsed patients are limited
- Previous Phase 2 study from Johnson & Johnson showed objective responses in patients with intermediate and high-risk MDS[1]

1. Study INT-28; *Blood*. 2007;109(10):4158-63

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Tipifarnib: Role of CXCL12 and Isolated Neutropenia in MDS

HYPOTHESIS:

- Neutropenia at study entry may enrich for response to tipifarnib in patients with MDS

RATIONALE FOR TESTING:

- CXCL12 implicated in the homing of myeloid cells in the bone marrow. Activating mutations in the CXCL12 pathway known to cause neutropenia.
- We hypothesized that the observation of isolated neutropenia could be a surrogate of high CXCL12 activity in bone marrow and sensitivity to tipifarnib.
- Retrospective analysis of data from the previous J&J Phase 2 study in MDS showed the majority of responders to tipifarnib were patients with neutropenia at study entry.

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Tipifarnib: Phase 2 Trial in MDS

DESIGN OF PHASE 2 CLINICAL TRIAL:

- Two cohorts of 18 patients, each with a Simon 2-stage design
- The trial will enroll patients who have failed up to two prior therapies; inclusion to be opened to all MDS patients independent of AML risk
- Trial inclusion to be limited to patients with neutropenia (<1,000) (estimated ~ 30% of MDS population)
- Primary endpoint: ORR
- Two responses needed to move to the 2nd stage of each cohort and the trial is positive with four or more responses in either cohort
- Retrospective analyses of the CXCL12 pathway markers

DATA: Anticipated 1H 2018

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Tipifarnib: Phase 2 Trial in CMML

RATIONALE:

- CMML is a disorder of bone marrow stem cells with characteristics of both myoproliferative and myelodysplastic diseases
- Prognosis of CMML is very poor; 5 year survival is ~18-20%
- Limited therapeutic options
- Previous Phase 2 study from Janssen supports continued development in CMML[1]

DESIGN OF PHASE 2 CLINICAL TRIAL:

- Primary Objective: ORR
- Retrospective analysis of RAS mutational status of patients and CXCL12 pathway markers

DATA: Anticipated 1H 2018

1 INT-28: J&J clinical data; ITT population; CMML defined by FAB criteria. Response criteria as in Cheson *et al. Blood* 2000;96:3671-4

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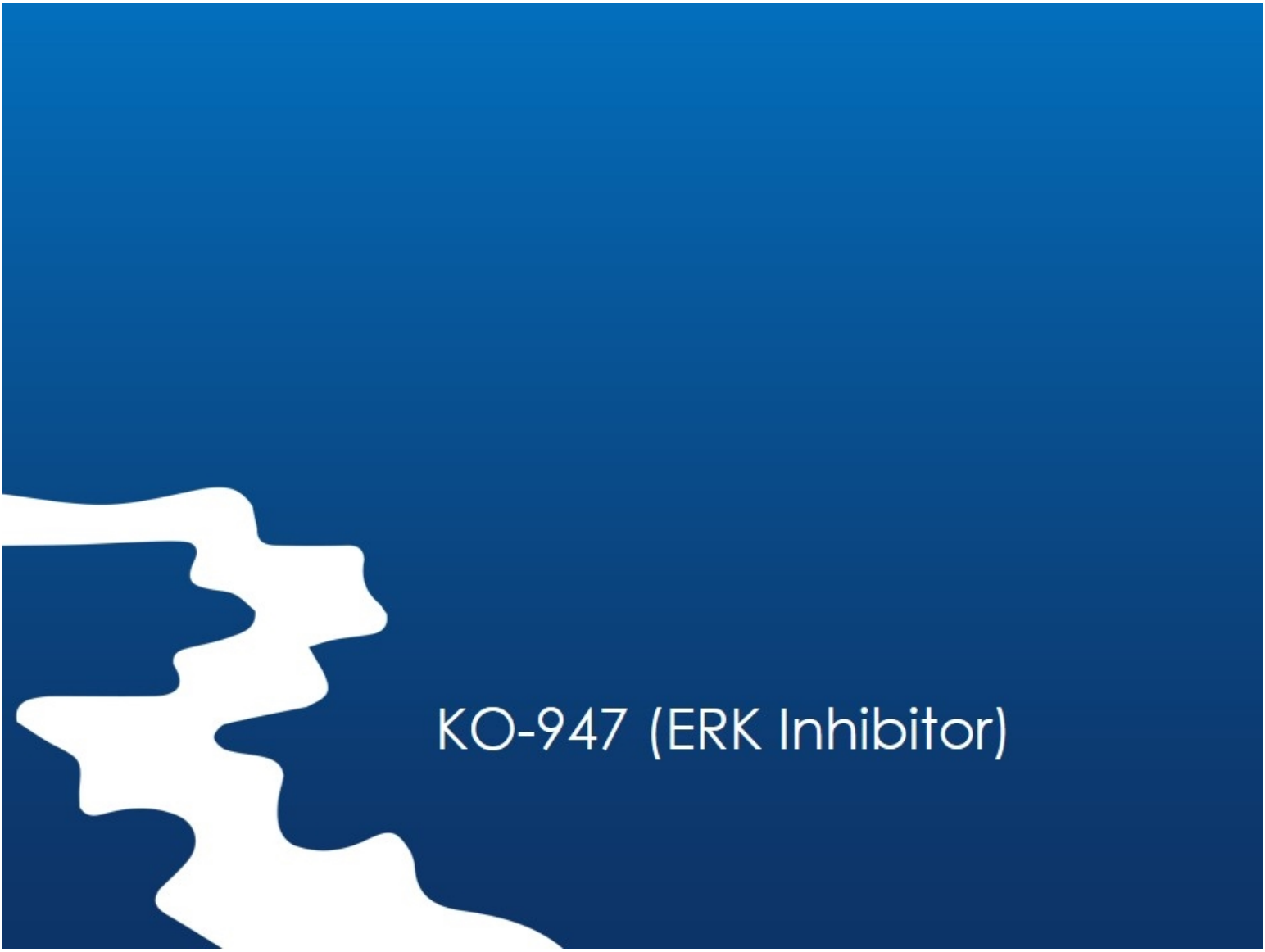
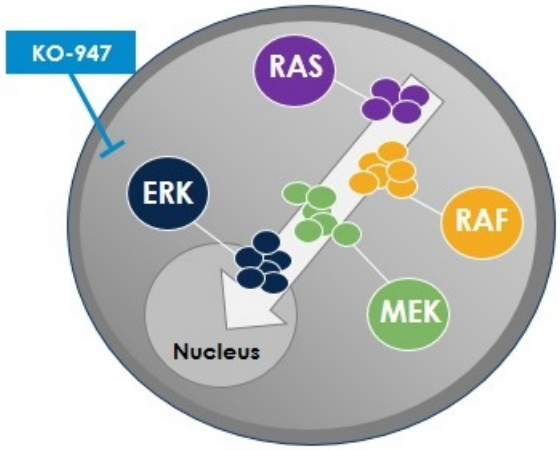

KO-947 (ERK Inhibitor)

KO-947: Potent ERK1/2 Inhibitor

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

A Differentiated ERK Inhibitor

- Potent and selective
- Prolonged pathway modulation intermittent dosing
- IV route selected for initial clinical to drive higher dose intensity
- **Currently in Phase 1 clinical testing**



ERK1/2: Extracellular Signal-Regulated Kinases 1/2

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KO-947: Translational Research Identified Potential Lead Clinical Indications

EXTENSIVE PRECLINICAL EVALUATION OF KO-947 ANTI-TUMOR ACTIVITY IN MAPK DYSREGULATED TUMORS

- KO-947 evaluated in ~200 PDX models across 20 potential indications
- Identified broad tumor classes sensitive to ERK inhibition (> 50% response rates in preclinical models)
  - KRAS- and BRAF-mutant adenocarcinomas
  - Squamous cell carcinomas
- Potential biomarkers have been identified to support development

POTENTIAL INDICATIONS REPRESENT HIGH UNMET NEED

POTENTIAL INDICATION	U.S. INCIDENCE
Head & Neck Squamous Cell Carcinomas (HNSCC)	56,000*
$KRAS^{mut}$ Non-Small Cell Lung Cancer	23,000
$BRAF^{mut}$ Non-Small Cell Lung Cancer	5,000

* Represents total est. U.S. incidence for the tumor type; biomarkers are being evaluated, which will result in lower incidence estimates

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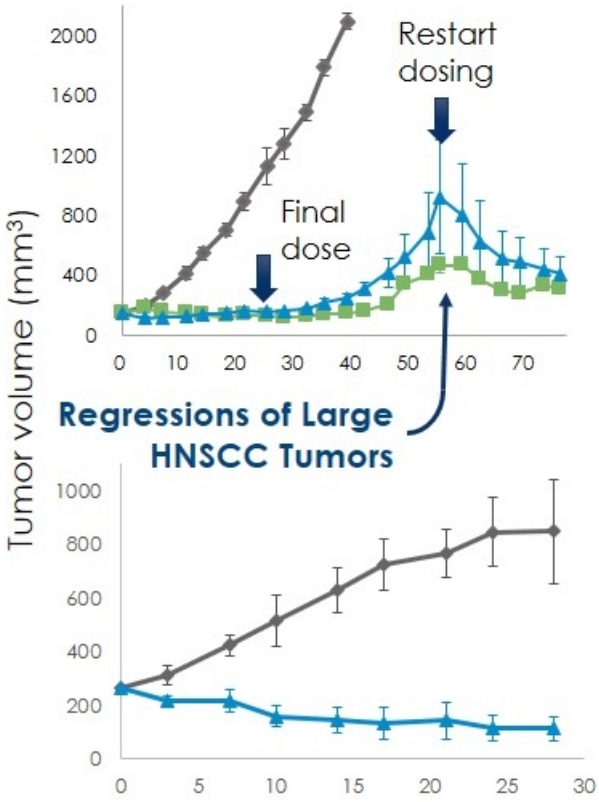
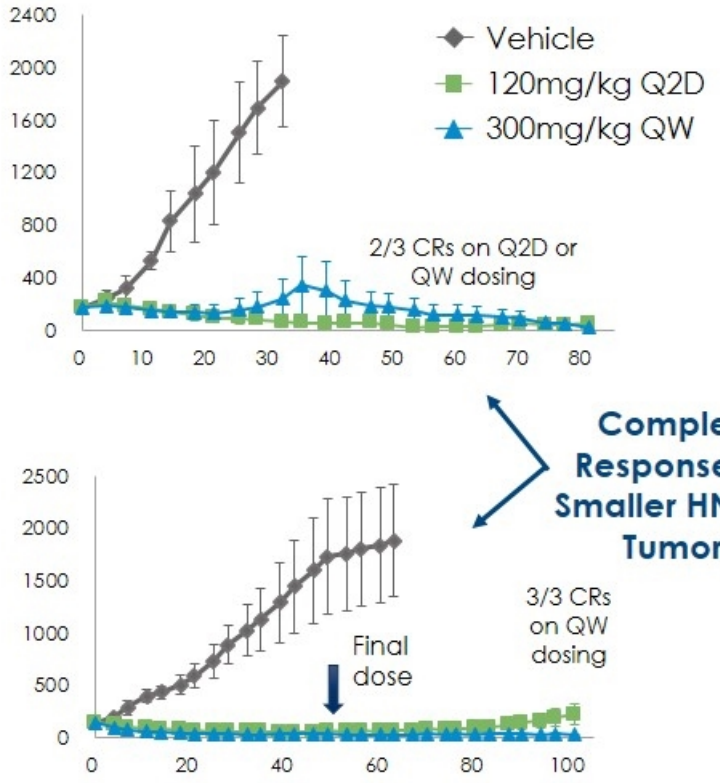
KO-947 is Highly Active in Preclinical Models in Biomarker Subsets of HNSCC





All animals dosed orally

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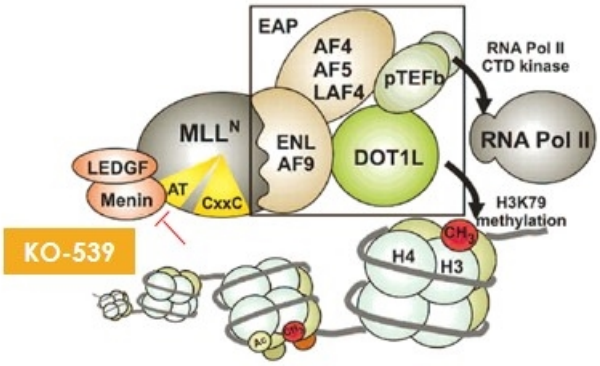
KO-539 (Menin-MLL Inhibitor)

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-MLL dysregulation in additional tumor types
- Licensed worldwide rights from University of Michigan



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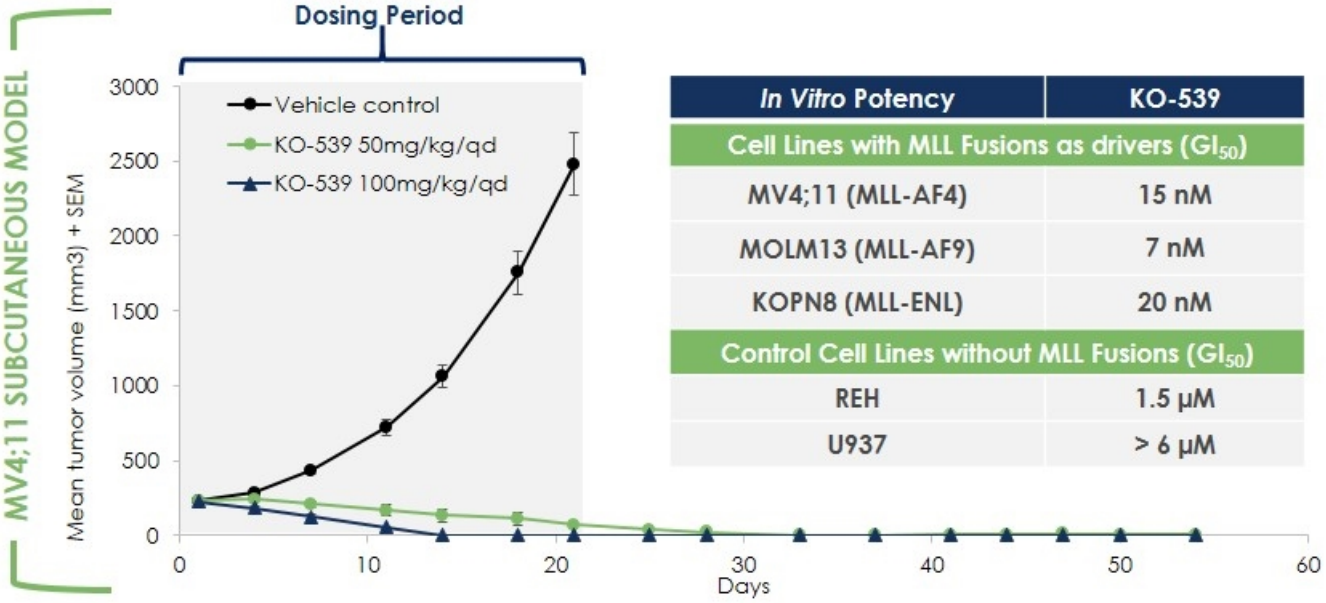
KO-539 Displays Prolonged Efficacy in Xenograft Model



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

KURA ONCOLOGY

Kura Is Building a Robust Patent Portfolio

- **Tipifarnib (Farnesyl Transferase Inhibitor)**
  - U.S. patent 9,707,221 provides exclusivity in U.S. for tipifarnib in HRAS mutant HNSCC indication until August 2036
  - Additional patent applications pending in the U.S. and foreign countries for HRAS mutant HNSCC as well as other biomarkers and disease indications
- **KO-947 (ERK Inhibitor)**
  - U.S. patent 9,624,228 provides exclusivity in the U.S. for KO-947 and structurally-related compounds as well as methods of using the compounds for the treatment of diseases including cancer until May 2036
  - Additional patent applications pending in the U.S. and foreign countries
- **KO-539 (Menin-MLL Inhibitor)**
  - Kura is pursuing U.S. and foreign patent protection relating to compositions of matter, methods of use, etc.

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Anticipated Milestones

PROGRAM	MILESTONES	ESTIMATED
Tipifarnib (Farnesyl Transferase Inhibitor)	Initiate Phase 2 clinical trial in CMML	✓
	Additional data from Phase 2 clinical trial in HRAS mutant HNSCC	✓
	Data from Phase 2 clinical trial in PTCL	✓
	Additional data from Phase 2 clinical trial in HRAS mutant HNSCC	✓
	Additional data from Phase 2 clinical trial in PTCL	2H 2017
	Data from Phase 2 clinical trial in MDS	1H 2018
	Data from Phase 2 clinical trial in CMML	1H 2018
KO-947 (ERK Inhibitor)	Initiate Phase 1 clinical trial	✓
	Translational data presentation at AACR	✓
	Data from Phase 1 clinical trial	2018
KO-539 (Menin-MLL Inhibitor)	Translational data presentation at AACR	✓

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DEVELOPING PRECISION MEDICINES TO TREAT CANCER