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): May 11, 2016

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):

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

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

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

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

Item 2.02 Results of Operations and Financial Condition.

On May 11, 2016, Kura Oncology, Inc. (the "Company") issued a press release announcing its financial results for the first quarter ended March 31, 2016. A copy of this press release is furnished herewith as Exhibit 99.1.

Item 7.01 Regulation FD Disclosure.

Beginning on May 11, 2016, members of the Company's management team will be providing presentation materials (the "Presentation") to certain interested parties. A copy of the Presentation is attached hereto as Exhibit 99.2 and incorporated herein by reference.

The information contained in this Current Report on Form 8-K under Items 2.02 and 7.01, including Exhibits 99.1 and 99.2, 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	Press release dated May 11, 2016
99.2	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: May 11, 2016

By: /s/ Annette North

Annette North
Senior Vice President and General Counsel

Exhibit Index

Exhibit Number	Description
99.1	Press release dated May 11, 2016
99.2	Presentation materials of Kura Oncology, Inc.

Exhibit 99.1

Kura Oncology Reports First Quarter 2016 Financial Results

LA JOLLA, Calif., May 11, 2016 – Kura Oncology, Inc., (NASDAQ: KURA) a clinical stage biopharmaceutical company, today reported financial results for the first quarter ended March 31, 2016.

“Kura has made important progress in advancing its pipeline of precision oncology drug candidates over the past quarter,” said Troy Wilson, Ph.D., J.D., President and Chief Executive Officer of Kura Oncology. “We continue to enroll patients in our on-going Phase 2 clinical trials of tipifarnib in patients with HRAS mutant solid tumors and in relapsed or refractory peripheral T-cell lymphoma and earlier in May 2016, we initiated our Phase 2 trial of tipifarnib in lower risk myelodysplastic syndromes (MDS).”

Dr. Wilson added: “In the second half or 2016, we are also planning to initiate a Phase 2 clinical trial with tipifarnib in patients with chronic myelomonocytic leukemia (CMML), a population for which prognosis is very poor, with a three-year survival rate estimated at less than 30 percent. Objective responses, including complete responses, have been previously observed with tipifarnib in CMML. Moreover, as part of the study, we plan to test a biomarker hypothesis, which may allow us to identify those patients most likely to experience durable responses. We believe our ongoing Phase 2 clinical trials and our additional planned trial in CMML provide us with multiple potential opportunities to position tipifarnib for registration-enabling Phase 3 studies in late 2017 or early 2018.”

Upcoming Clinical and Preclinical Milestones for Kura Oncology Programs

- IND submission for ERK inhibitor, KO-947, is anticipated in the second quarter of 2016 followed by initiation of a Phase 1 clinical trial, which is anticipated in the second half of 2016
- Receipt of topline data from the Phase 2 clinical trial for tipifarnib in HRAS mutant solid tumors is anticipated in the second half of 2016
- Initiation of a Phase 2 clinical trial for tipifarnib in patients with CMML is anticipated in the second half of 2016
- Nomination of a development candidate for the menin-MLL program is anticipated in the second half of 2016

Financial Results for the First Quarter 2016

- Cash, cash equivalents and short-term investments totaled $78.5 million as of March 31, 2016, compared with $85.7 million as of December 31, 2015. Management expects that existing cash, cash equivalents and short-term investments will be sufficient to fund current operations into 2018.
- Research and development expenses for the first quarter of 2016 were $4.6 million, compared to $3.6 million for the first quarter of 2015.
- General and administrative expenses for the first quarter of 2016 were $2.4 million, compared to $1.1 million for the first quarter of 2015.
- Net loss for the first quarter of 2016 was $6.6 million, or $0.36 per share, compared to a net loss of $4.5 million, or $1.41 per share, for the first quarter of 2015.
- Subsequent to the first quarter of 2016, Kura Oncology put in place a $20.0 million long-term debt financing agreement, of which $7.5 million has been drawn down. Use of proceeds is for development programs and general corporate purposes.

About Kura Oncology

Kura Oncology is a clinical-stage biopharmaceutical company focused on the discovery and development of precision medicines for the treatment of solid tumors and blood cancers. Kura's pipeline consists of small molecules that target cancer signaling pathways where there is a strong scientific and clinical rationale to improve outcomes by identifying those patients most likely to benefit from treatment. The company's lead drug candidate is tipifarnib, a farnesyl transferase inhibitor, which is currently being studied in four Phase 2 clinical studies: the first is in patients with locally advanced solid tumors that carry HRAS mutations; the second is in patients with relapsed or refractory peripheral T-cell lymphomas; the third in patients with lower risk myelodysplastic syndromes; and the fourth is an investigator sponsored Phase 2 trial, in patients with urothelial carcinoma tumors characterized by HRAS mutations. Kura’s preclinical pipeline includes KO-947, an ERK inhibitor, and a menin-MLL inhibitor program. For additional information about Kura Oncology, please visit www.kuraoncology.com.

Forward Looking Statements

This news release contains certain forward-looking statements that involve risks and uncertainties that could cause actual results to be materially different from historical results or from any future results expressed or implied by such forward-looking statements. Such forward-looking statements include statements regarding, among other things, the efficacy, safety and therapeutic potential of Kura Oncology’s product candidates and compounds, progress and expected timing of Kura Oncology’s drug development programs and clinical trials, plans regarding regulatory filings and future research and clinical trials, expected timing of data from clinical trials of tipifarnib, the strength of Kura Oncology’s balance sheet and the adequacy of cash on hand. You are urged to consider statements that include the words “may,” “will,” “would,” “could,” “believes,” “estimates,” “projects,” “potential,” “expects,” “plans,” “anticipates,” “intends,” “continues,” “designed,” “goal,” or the negative of those words or other comparable words to be uncertain and forward-looking. These forward-looking statements are based upon Kura Oncology’s current expectations and involve assumptions that may never materialize or may prove to be incorrect. Actual results and the timing of events could differ materially from those anticipated in such forward-looking statements as a result of various risks and uncertainties, which include, without limitation, the risk that compounds that appeared promising in early studies or clinical trials do not demonstrate safety and/or efficacy in later studies or clinical trials, the risk that Kura Oncology may not obtain approval to market its product candidates, uncertainties associated with regulatory filings and applications, the risks associated with reliance on outside financing to meet capital requirements, and other risks associated with the process of discovering, developing and commercializing drugs that are safe and effective for use as human therapeutics, and in the endeavor of building a business around such drugs. For a further list and description of the risks and uncertainties Kura Oncology faces, please refer to Kura Oncology’s periodic and other filings with the Securities and Exchange Commission, which are available at www.sec.gov. Such forward-looking statements are current only as of the date they are made, and Kura Oncology assumes no obligation to update any forward-looking statements, whether as a result of new information, future events or otherwise.

KURA ONCOLOGY, INC.
Statements of Operations Data
(unaudited)
(in thousands, except per share data)

	Three Months Ended March 31,	
	2016	2015
Operating Expenses:		
Research and development	$ 4,649	$ 3,628
General and administrative	2,391	1,060
Total operating expenses	7,040	4,688
Other income, net	414	212
Net loss	$ (6,626)	$ (4,476)
Net loss per share, basic and diluted	$ (0.36)	$ (1.41)
Weighted average number of shares used in computing net loss per share, basic and diluted	18,245	3,184

KURA ONCOLOGY, INC.
Balance Sheet Data
(unaudited)
(in thousands)

	March 31, 2016	December 31, 2015
Cash, cash equivalents and short-term investments	$ 78,466	$ 85,746
Working capital	75,765	81,814
Total assets	80,382	87,259
Long-term liabilities	6	101
Accumulated deficit	(32,922)	(26,296)
Total stockholders' equity	76,066	82,103

CONTACT INFORMATION

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Exhibit 99.2

KURA ONCOLOGY

Troy Wilson, Ph.D., J.D.
President and CEO
May 2016

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, and our other programs, 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," "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.

CORPORATE OVERVIEW

Development Strategy

- Advance pipeline of targeted therapeutics for solid tumors and blood cancers
- Utilize precision medicine approaches to identify patients most likely to benefit from treatments; fast-to market strategy

Pipeline

- Lead product candidate, tipifarnib, in multiple Phase 2 trials; additional Phase 2 trial planned
- Potential to initiate first pivotal study in 2017/18
- Preclinical programs advancing; anticipate KO-947 IND 2Q 2016

Experienced Team

- Key roles in oncology R&D at both biotech and pharma
- Members of team have worked together since 2007 at Intellikine and Wellspring Biosciences

Solid Financials

- $78.5M cash as of March 31, 2016*
- Resources expected to fund current operations into 2018

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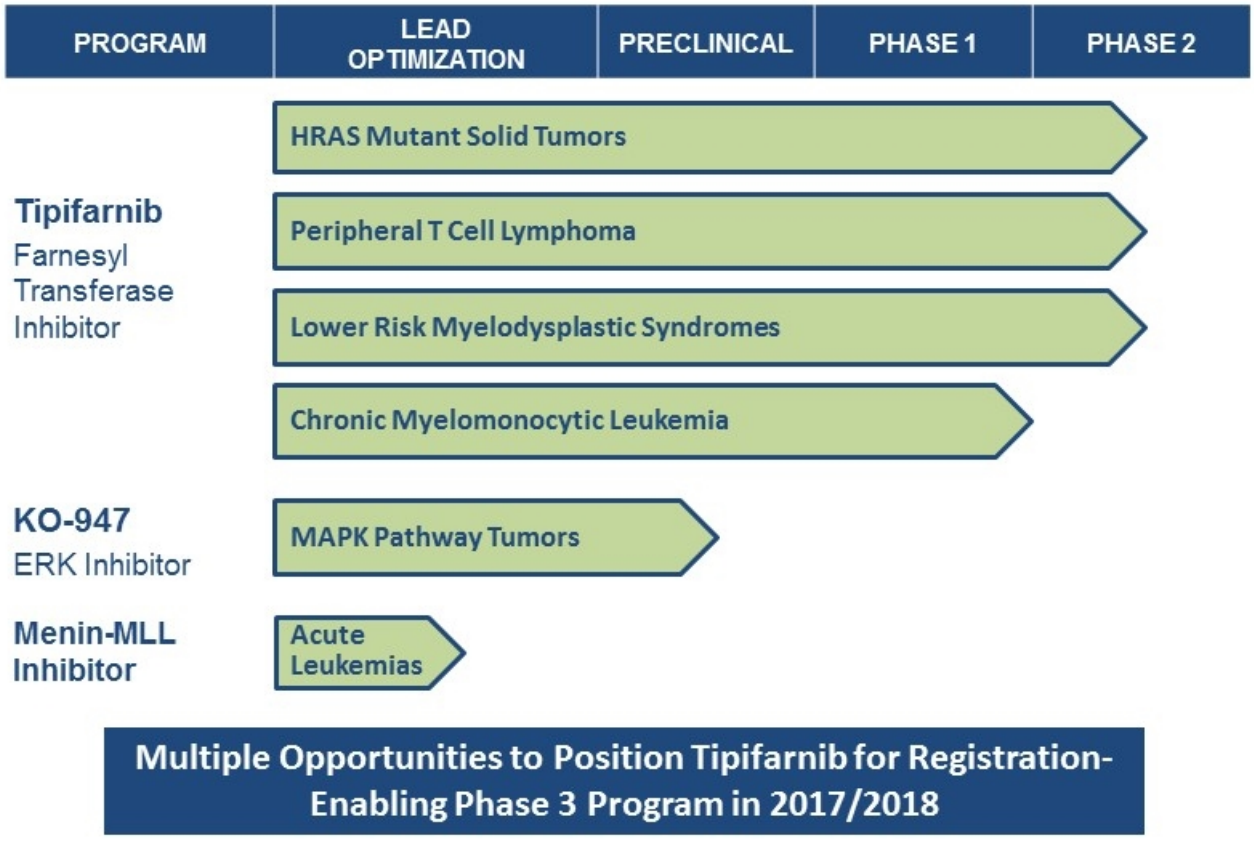
* Includes Cash, Cash Equivalents, and Short-Term Investments

DIVERSE DEVELOPMENT PIPELINE



Multiple Opportunities to Position Tipifarnib for Registration-Enabling Phase 3 Program in 2017/2018

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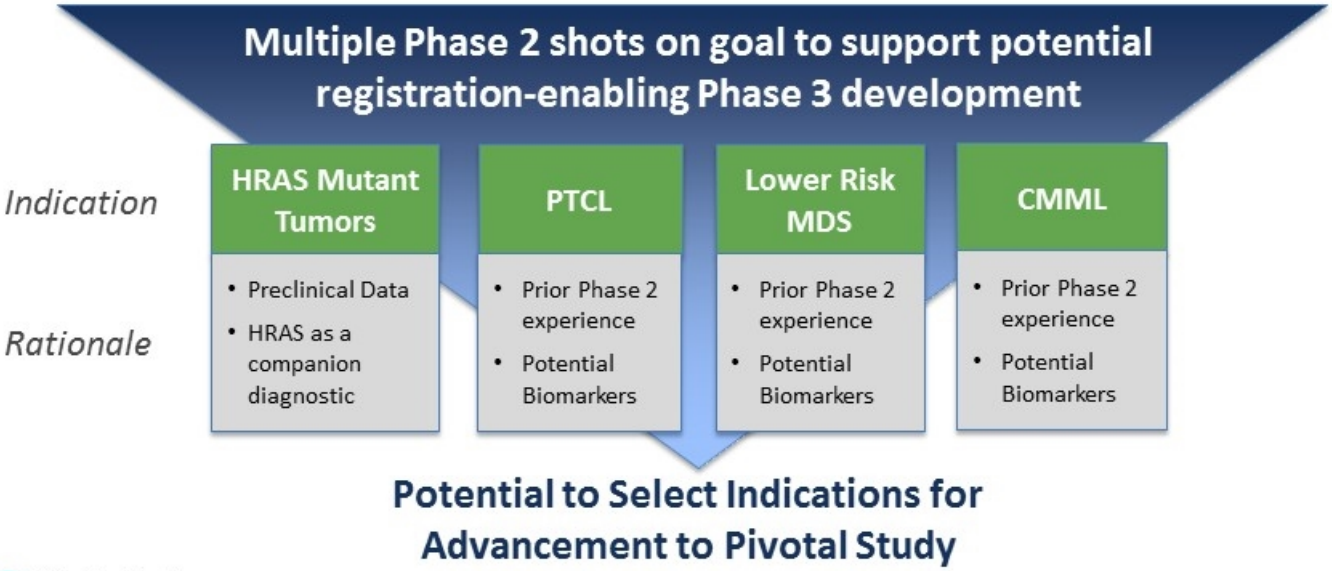
TIPIFARNIB – LEAD PRODUCT CANDIDATE

- Licensed worldwide rights in oncology from Janssen
- Broad development program, which preceded precision medicine approach
  - Studied in > 5,000 patients
  - Generally well-tolerated
  - Objective responses observed in multiple unselected patient populations
  - Evidence of durable clinical benefit



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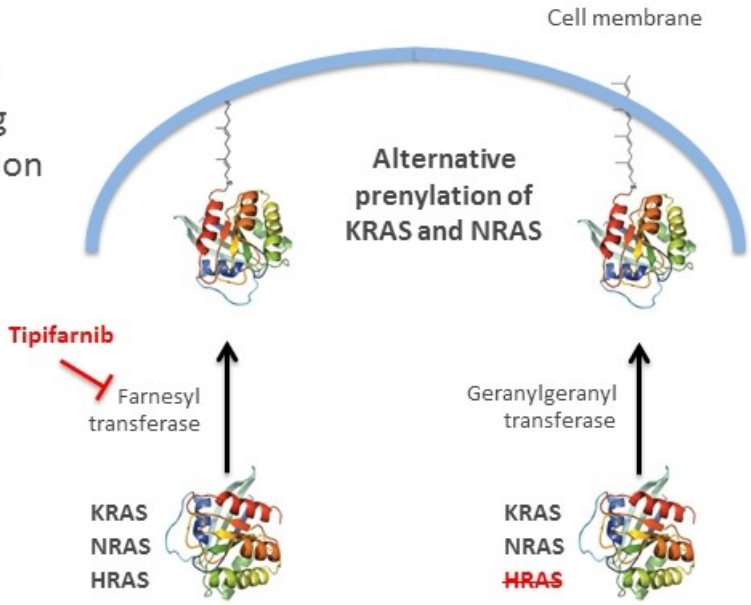
MECHANISM OF ACTION OF TIPIFARNIB

- Farnesyltransferase (FT) attaches farnesyl group to proteins, facilitating their localization to the inner membrane
- Targets of FT include members of the RAS superfamily of small GTP-binding proteins critical to cell cycle progression
- Blocking farnesylation prevents membrane localization
- KRAS and NRAS have an alternate pathway in geranylgeranylation
- **HRAS is solely dependent on farnesylation**
- **Tumors with KRAS and NRAS mutations are less sensitive to FTIs**



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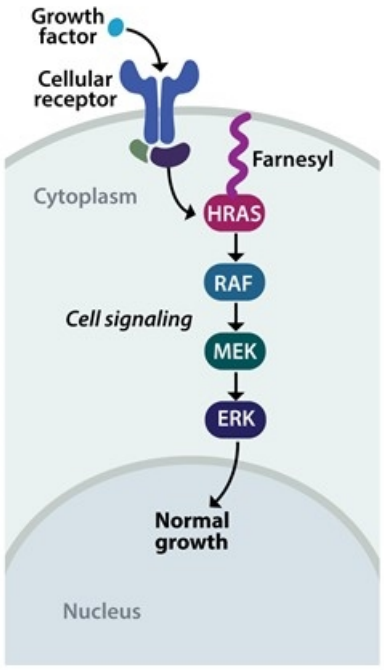
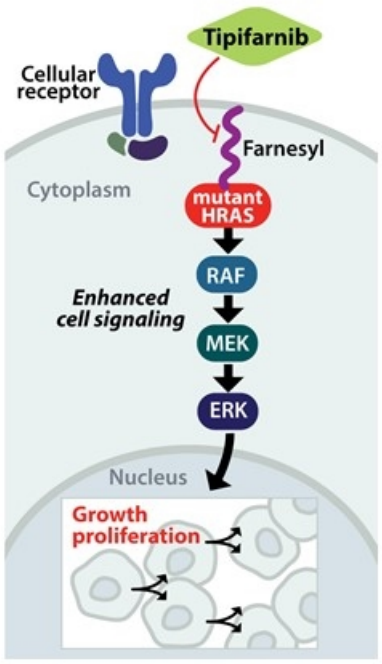
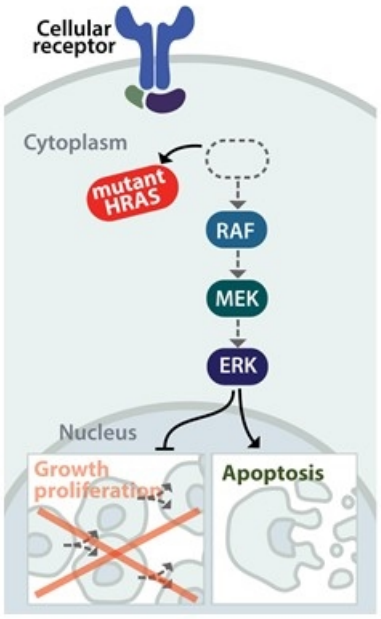
TIPIFARNIB MOA SUPPORTS DEVELOPMENT IN HRAS MUTANT SOLID TUMORS



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, driving tumor growth and proliferation



Blocking farnesylation prevents membrane localization of HRAS, disrupting cellular signaling and inhibiting tumor growth

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OVERVIEW – HRAS MUTANT SOLID TUMORS

What are HRAS Mutant Tumors?

- HRAS mutant solid tumors include salivary gland, urinary tract, cervical, upper aerodigestive tract and other cancers
- Estimated annual incidence of approximately 8,000 patients in U.S.

Prognosis

- Prognosis varies with certain histologies having very poor prognoses with limited treatment options
  - HRAS mutant salivary gland cancers have no effective treatment
- Multiple HRAS mutant tumor types constitute an unmet medical need

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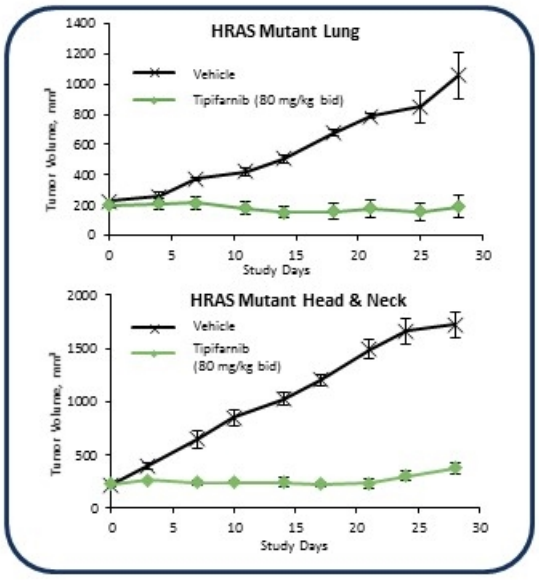
RATIONALE AND DESIGN OF PHASE 2 TRIALS IN HRAS MUTANT TUMORS



- **Rationale:**
  - HRAS has potential to be driver oncogene (Costello Syndrome, urothelial cancer)
  - Tipifarnib is active in PDX models of HRAS mutant tumors

- **Design of Current Phase 2 Clinical Trials:**
  - Primary Objective: ORR
  - 18 patient Phase 2 study with Simon two-stage design (11+7); 2 responses required after the first 11 evaluable patients to proceed to stage 2
  - Kura is supporting an investigator-sponsored Phase 2 study (urothelial cancer)
  - Dosing at 900 mg bid for 7 days in alternate week dosing

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DEVELOPMENT OF TIPIFARNIB IN HEMATOLOGY / ONCOLOGY DISORDERS

- Previous clinical activity observed across multiple hematologic malignancies, including:
  - Lymphomas
  - Leukemias
  - Myeloproliferative disorders
  - Myelodysplastic diseases
- Multiple farnesylated proteins / pathways implicated in heme/onc disorders
- Evaluate prior clinical data and samples where available
- Develop biomarker hypotheses
- Confirm clinical activity
- Validate biomarker hypotheses

Biomarker Strategies

- Potential to improve response rate and duration of response
- Potential to reduce clinical development risk
- Potential to extend IP protection
- Commercial competitive advantage

OVERVIEW – PERIPHERAL T-CELL LYMPHOMA

What is PTCL?

- Peripheral T-cell lymphomas (PTCLs) are a diverse group of usually aggressive non-Hodgkin lymphomas
- Characterized by the presence of malignant T-cells or natural killer (NK) cells
- Estimated annual U.S. incidence of approximately 5,000 patients

Prognosis

- Overall prognosis is poor with 5 year OS approximately 35%
- Clear unmet need as few treatment options provide durable benefit

Agent#	N	Prior Therapy median	CR (%)	ORR (%)	Median PFS/TTP (mos)	Median OS (mos)
Beleodaq	120	2	11	26	1.6	7.9
ISTODAX (romidepsin)	130	2	15	25	4.0	11.3
FOLOTYN	109	3	8	27	3.5	14.5

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RATIONALE AND DESIGN OF TRIALS IN PTCL

- **Rationale:**
  - Previous Phase 2 study in patients with relapsed/ refractory PTCL showed encouraging activity*
  - Durable responses - median DOR: ~11 months
  - Potential biomarkers that may predict activity of tipifarnib

Disease Indication	n	CR n (%)	PR n (%)	ORR (%)
Overall	36	6 (17)	5 (14)	31%
Hodgkin Lymphoma	19	2 (11)	2 (11)	21%
Mycosis Fungoides	4	0 (0)	2 (50)	50%
Peripheral T-Cell Lymphoma	**8**	**3 (38)**	**1 (13)**	**50%**
Anaplastic Large Cell Lymphoma	5	1 (20)	0 (0)	20%

- **Design of Current Phase 2 Clinical Trial:**
  - Primary Objective: ORR
  - 18 patient Phase 2 study with Simon two-stage design (11+7): 2 responses required after the first 11 evaluable patients to proceed to stage 2
  - Enrollment to be extended to 30 patients if 5 responses seen in stage 1
  - Dosing at 900 mg bid for 7 days in alternate week dosing

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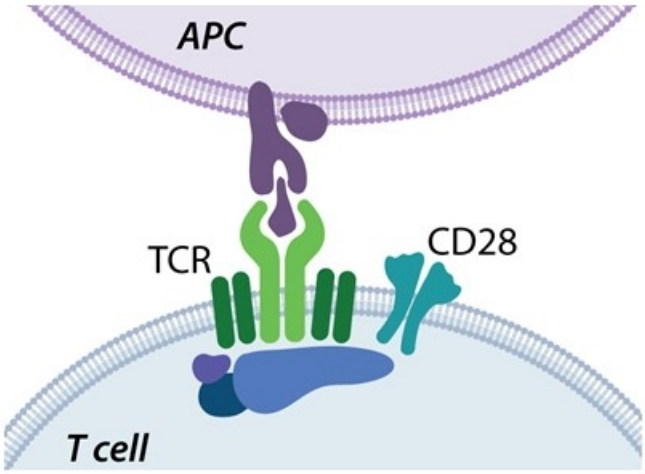
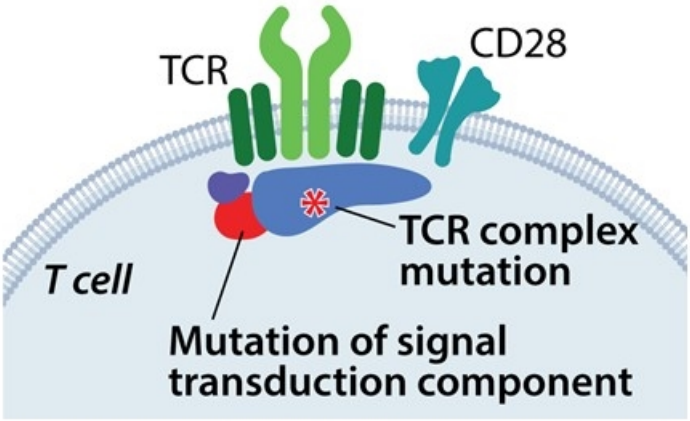
*NCI-Sponsored Phase 2 Trial in Relapsed/Refractory Lymphomas; *Blood. 2011;118(18):4882-9*

FARNESYLATION OF KEY SIGNALING PROTEINS IN T CELLS SUPPORTS DEVELOPMENT APPROACH



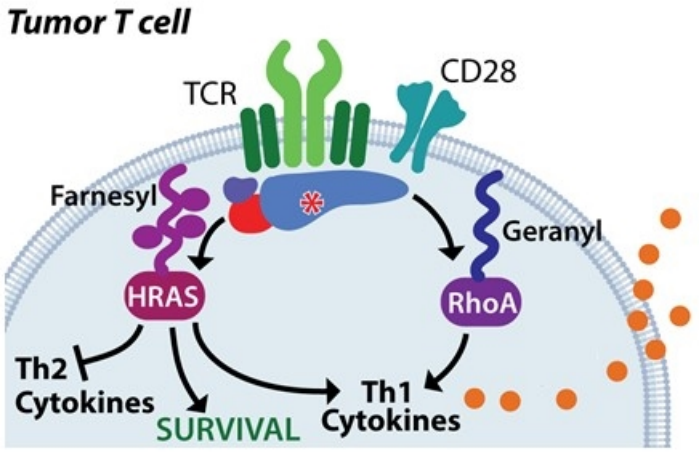
Normal T cells are activated by interaction of their T cell receptors (TCR) and CD28 with molecules in antigen presenting cells (APC).

T cell lymphoma cells are constantly activated due to TCR complex mutations, dysregulation or mutations of signal transduction components.



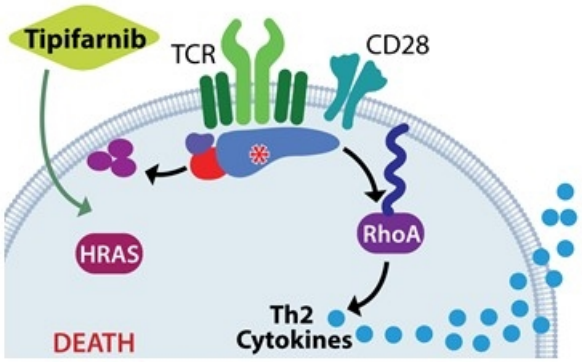
KURA ONCOLOGY

FARNESYLATION OF KEY SIGNALING PROTEINS IN T CELLS SUPPORTS DEVELOPMENT APPROACH



A subset of T cell lymphomas is characterized by Th1 cytokine production. Both Th1 cytokine production and tumor cell survival are regulated by farnesylated HRAS.

Upon treatment of PTCL with tipifarnib, we expect to inhibit or kill the population of Th1 cells and shift the population to Th2 cells. By monitoring production of Th1 and Th2 cytokines and other markers, we may be able to identify those T-cell tumors sensitive to treatment with tipifarnib.



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OVERVIEW – LOWER RISK MYELODYSPLASTIC SYNDROMES

What is MDS?

- Myelodysplastic syndromes (MDS) are a group of blood and bone marrow disorders with both proliferative and dysplastic phenotypes
- Characterized by ineffective hematopoiesis leading to cytopenias
- Autoimmunity known to play a role in the onset of lower risk MDS
- Estimated annual U.S. incidence of 13,000; 75% of patients (~ 9,750) comprise lower risk MDS

Prognosis

- Median age of patients with MDS is 70 to 75 years
- Lower-risk MDS patients are at high risk of infection, require regular transfusions and have a generally poor quality of life
- ~25% of MDS patients transform to AML
- Limited therapeutic options

PREVIOUS PHASE 2 DATA SUPPORTS DEVELOPMENT IN LOWER RISK MDS

- **Rationale:**
  - Previous Phase 2 study sponsored by J&J demonstrates tipifarnib is active in MDS*
  - Identification of potential biomarkers

Intermediate / High Risk MDS Including CMML	Overall (N = 82)
ORR (CR+HI)	26 (31.7%)
Complete Response (CR)	12 (14.6%)
Hematologic Improvement (HI)	14 (17.1%)

- **Design of Current Phase 2 Clinical Trial:**
  - Primary Objective: RBC transfusion independence
  - Initially, 44 eligible subjects stratified into one of 4 biomarker-defined strata
  - Patients will be analyzed retrospectively for the presence/absence of various NK- and T-cell markers
  - Dosing at 900 mg bid for 7 days in alternate week dosing

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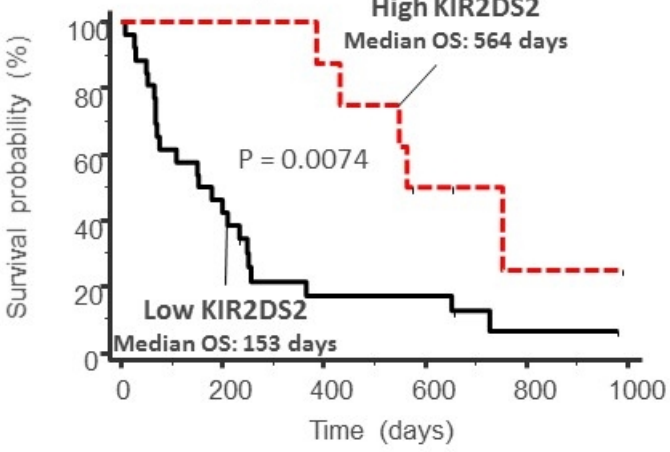
*Study INT-28; Blood. 2007;109(10):4158-63

KIR2DS2 AS A POTENTIAL BIOMARKER IN MDS

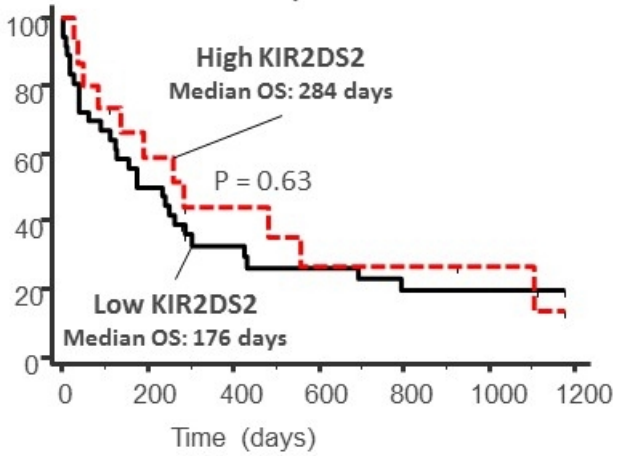
Tipifarnib Treated Patients (n=34)

NCI CTEP-20 Study



Chemotherapy Treated Patients (n=51)

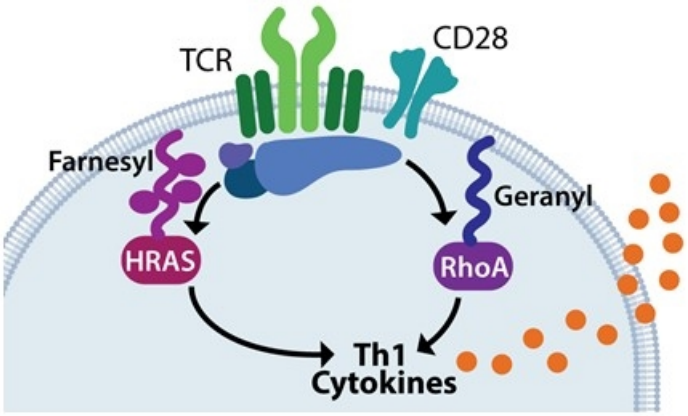
German AMLCG 1999 Study



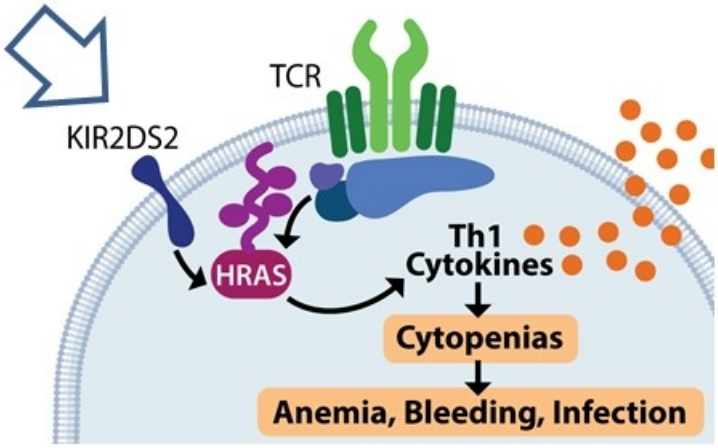
- High expression of KIR2DS2, an NK/T cell marker, correlated with clinical benefit in tipifarnib-treated AML patients; no correlation in chemo treated patients
- Expression of KIR2DS2 has been shown to predispose patients to the development of MDS and other autoimmune disorders
- Activating KIRs, such as KIR2DS2, are known to signal in part through the RAS pathway

KURA ONCOLOGY

SIGNALING PATHWAYS IN MDS HAVE POTENTIAL TO YIELD PREDICTIVE BIOMARKERS



Aging

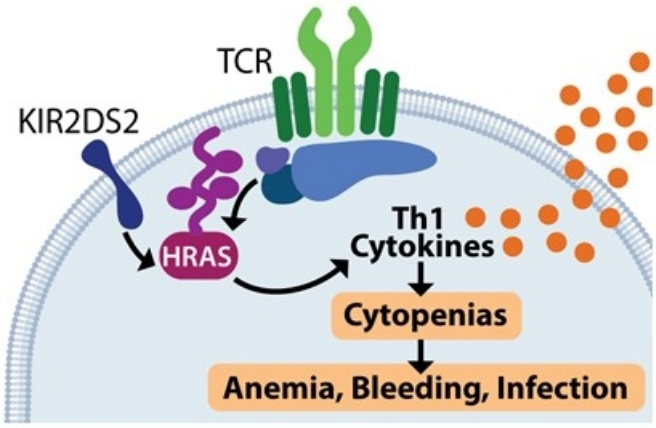


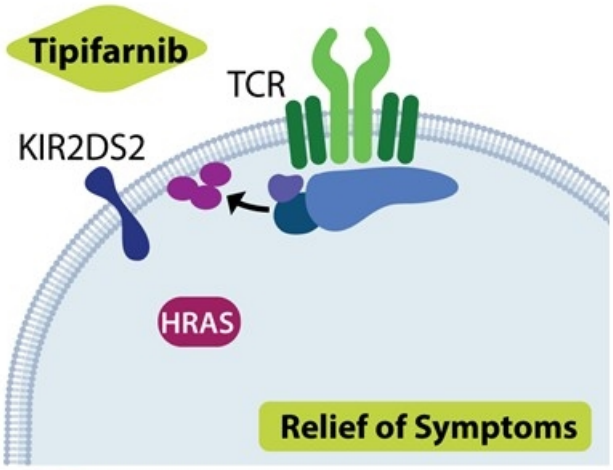
During aging, loss of CD28 and gain of KIR2DS2 result in overproduction of Th1 cytokines and may induce autoimmunity against the bone marrow.

KURA ONCOLOGY

SIGNALING PATHWAYS IN MDS HAVE POTENTIAL TO YIELD PREDICTIVE BIOMARKERS

By monitoring KIR2DS2 and other markers, we may be able to identify those patients with bone marrow dysplasia who could benefit from tipifarnib.





KURA ONCOLOGY

OVERVIEW – CHRONIC MYELOMONOCYTIC LEUKEMIA

What is CMML?

- Chronic myelomonocytic leukemia (CMML) is a clonal disorder of bone marrow stem cells that shares characteristics of both myoproliferative and myelodysplastic diseases
- Increased monocytes and blasts in the peripheral blood and bone marrow, as well as dysplasia in at least one type of blood cell
- Estimated U.S. incidence of 1,400 patients

Prognosis

- Prognosis of CMML is very poor
- 3 year survival is approximately 29%
- Limited therapeutic options

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PREVIOUS CLINICAL DATA SUPPORTS DEVELOPMENT IN CMML

- **Rationale:**
  - Observed clinical activity in previous studies including CMML patients*
  - Opportunity to use response as primary endpoint
  - Potential to treat 1st line patients
  - Data from previous Phase 1 AML study supports higher dose in alternate week dosing schedule†

	CMML (N = 19)
ORR (CR+CRp+PR)	4 (21.1%)
CR	1 (5.3%)
CRp	3 (15.8%)
HI	3 (15.8%)
Duration of Response, median	7.5 mo
Time to AML, median	Not Estimable
Overall Survival, median	14.7 mo

- **Design of Current Phase 2 Clinical Trial:**
  - Primary Objective: ORR
  - Two exploratory cohorts (RAS wild type and RAS mutant)
  - Dosing up to 1,200 mg bid for 7 days in alternate week dosing
  - Retrospective analysis of RAS mutational status of patients

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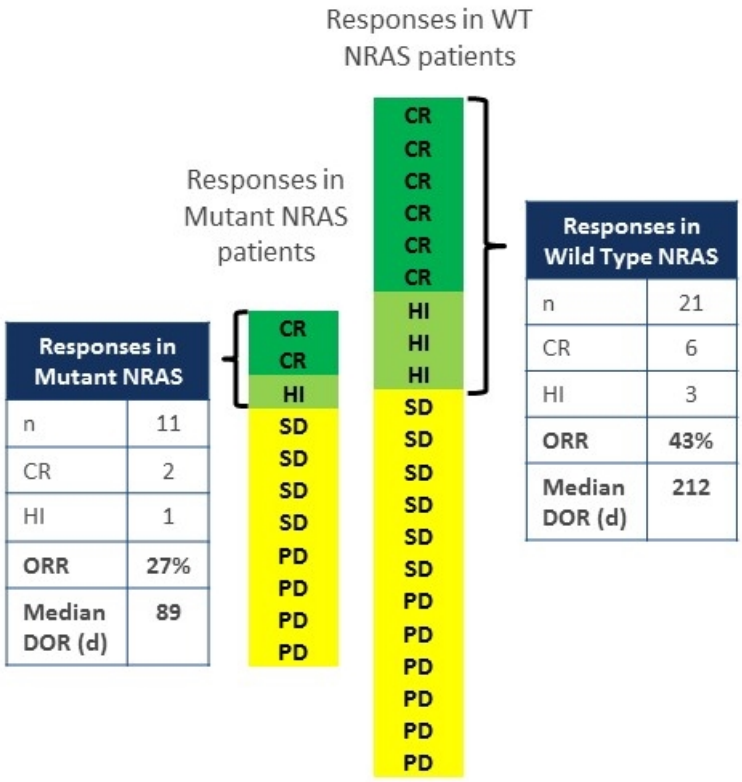
*INT-28: J&J clinical data; ITT population; CMML defined by FAB criteria. Response criteria as in Cheson et al. Blood 2000;96:3671-4

† Kirschbaum *et al.*, *Leukemia*. 2011; 25(10):1543-7.

EXCLUDING RAS MUTANTS MAY ENHANCE EFFICACY IN CMML



Responses in Mutant NRAS	
n	11
CR	2
HI	1
ORR	**27%**
Median DOR (d)	**89**

Responses in Wild Type NRAS	
n	21
CR	6
HI	3
ORR	**43%**
Median DOR (d)	**212**

- NCI CTEP-20 Phase 2 trial in 34 previously untreated elderly or unfit AML patients*
- NRAS gene status determined for 32 patients
- Higher response rate in AML patients with WT NRAS treated with tipifarnib may translate to CMML population
- Analysis of patient RAS mutational status will be conducted retrospectively in the planned Phase 2 CMML clinical trial

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*CTEP-20; *Blood. 2008; 111: 2589-96*

MULTIPLE PATHS TO REGISTRATION-ENABLING PHASE 3 STUDIES

Identify Therapeutic Opportunities
Facilitate Rapid & Efficient Clinical Validation

	HRAS Solid Tumors			PTCL	Lower Risk MDS	CMML*
Subjects	Thyroid n=18	Solid n=18	Urothelial n=18	n=18 (Potential for expansion to n=30)	n = 54	n = 20
1° Endpoint	ORR (RECIST v1.1)			ORR (IWC)	RBC transfusion independence	ORR using MDS/MPN IWG criteria
Rationale	• Preclinical data • Pathology series (urothelial)			• Prior Phase 2 experience • Patient biomarker analysis	• Prior Phase 2 experience • Patient biomarker analysis	• Prior Phase 2 experience • Patient biomarker analysis
Biomarkers	Documented HRAS mutations			Exploratory	NK cell markers including KIR2DS2	NRAS/KRAS wild-type versus mutant
Est. U.S. Incidence	8,000			5,000	9,750	1,400

* Proposed clinical plan

Potential to Select Indications for Advancement to Pivotal Study

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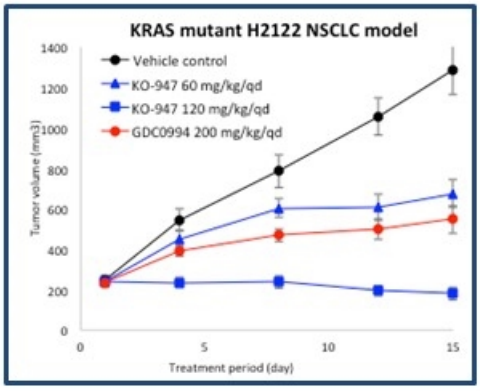
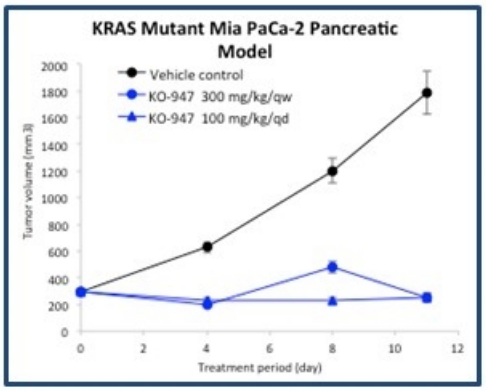
KO-947 – STRONG ACTIVITY IN MAPK PATHWAY MODELS WITH FLEXIBLE DOSING

- ATP competitive inhibitor of the extracellular receptor kinase (ERK)
- Aberrant signaling caused by mutations or dysregulation of the MAPK pathway associated with numerous tumor types
- KO-947 induced tumor regression in KRAS mutated tumor models at tolerable doses
- KO-947 compares favorably to clinical-stage reference compounds
- Advancing an intravenous administration, which has the potential to improve exposure and tolerability
- IND anticipated 2Q 2016





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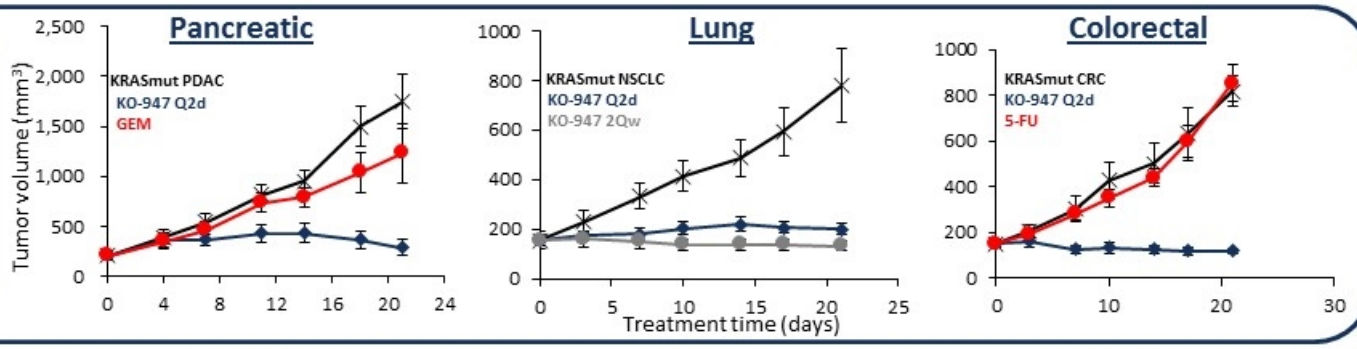
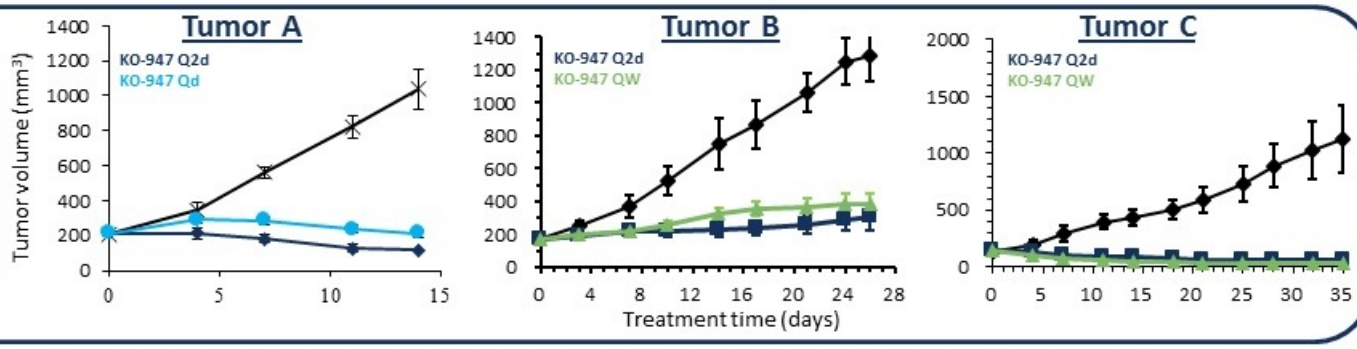
KO-947: TRANSLATIONAL RESEARCH IDENTIFIED POTENTIAL LEAD CLINICAL INDICATIONS

MAPK Pathway Mutations



MAPK Pathway Dysregulation



- Robust anti-tumor activity demonstrated in two broad tumor classes with > 50% response rates in preclinical models
- Potential biomarkers have been identified

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Evaluated KO-947 in 138 PDX models across 20 potential indications

KO-947: RATIONALE FOR FURTHER STUDY

Development Plans

- IND-enabling studies and preparation ongoing
- Translational research identified potential lead indications
- Potential biomarkers identified
- IV route of administration selected for initial clinical study
- Opportunity to advance oral program to broaden indication set

Anticipated Milestones

• IND submission	Q2 2016
• Initiate Phase 1 study	2H 2016

U.S. Commercial Opportunity

- KRAS mutant tumors incidence
  - Pancreatic cancer: 45,000
  - Colorectal cancer: 53,000
  - Non-small cell lung cancer: 23,000
- MAPK pathway dysregulated tumors

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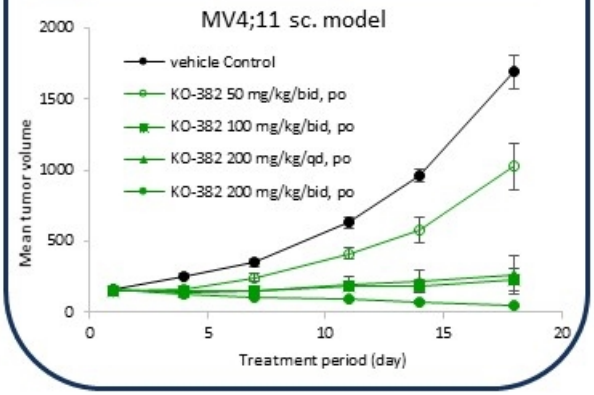
MENIN-MLL INHIBITOR PROGRAM

- Chromosomal translocations of the MLL gene play a causative role in the onset, development and progression of a subset of acute leukemias
- Potential first-in-class program targeting menin-MLL interaction for treatment of MLL-rearranged and MLL-PTD AML and ALL
- Estimated U.S. incidence of 3,200 patients with MLL-rearranged and MLL-PTD
- Opportunities to target menin overexpression in additional tumor types
- Lead optimization underway; development candidate anticipated 2H 2016
- Licensed worldwide rights from University of Michigan

Potent and Selective Advanced Leads

KO-382 IC_{50}	MLL Leukemia Cells		Normal Cells	
	MV4;11	35 nM	K562	1500 nM
	MOLM13	75 nM	REH	2000 nM



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NEAR-TERM MILESTONES

PROGRAM	EVENT	ANTICIPATED TIMING
Tipifarnib	Phase 2 clinical trial in HRAS mutant solid tumors	Ongoing
Tipifarnib	Phase 2 clinical trial in PTCL	Ongoing
Tipifarnib	Phase 2 IST in HRAS mutant urothelial cancer	Ongoing
Tipifarnib	Phase 2 clinical trial in lower risk MDS	Ongoing
KO-947	IND submission	1H 2016
KO-947	Initiate Phase 1 clinical trial	2H 2016
Tipifarnib	Initiate Phase 2 clinical trial in CMML	2H 2016
Tipifarnib	Topline data from Phase 2 study in HRAS mutant tumors	2H 2016
Menin-MLL	Nomination of development candidate	2H 2016
Tipifarnib	Topline data from Phase 2 study in low-risk MDS	1H 2017
Tipifarnib	Topline data from Phase 2 clinical trial in PTCL	2H 2017
Tipifarnib	Topline data from Phase 2 clinical trial in CMML	1H 2018

EXPERIENCED MANAGEMENT TEAM

Troy Wilson, Ph.D., J.D.	Chief Executive Officer
Yi Liu, Ph.D.	Chief Scientific Officer
Pingda Ren, Ph.D.	SVP, Chemistry and Pharm. Sciences
Antonio Gualberto, M.D., Ph.D.	Chief Medical Officer
Heidi Henson, CPA	Chief Financial Officer
Annette North, LLB	SVP and General Counsel

wellspring biosciences
intellikine
EMD SERONO
ambit
Pfizer
Takeda
Genomics Institute of the Novartis Research Foundation

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BOARD AND ADVISORS

BOARD OF DIRECTORS

Faheem Hasnain	Former President and CEO, Receptos, Inc.
Robert Hoffman	SVP Finance and CFO, AnaptysBio, Inc.
Thomas Malley	Mossrock Capital
Troy Wilson, Ph.D., J.D.	CEO and President, Kura Oncology, Inc.

SCIENTIFIC ADVISORS

Kevan 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

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CORPORATE OVERVIEW

Development Strategy

- Advance pipeline of targeted therapeutics for solid tumors and blood cancers
- Utilize precision medicine approaches to identify patients most likely to benefit from treatments; fast-to market strategy

Pipeline

- Lead product candidate, tipifarnib, in multiple Phase 2 trials; additional Phase 2 trial planned
- Potential to initiate first pivotal study in 2017/18
- Preclinical programs advancing; anticipate KO-947 IND 2Q 2016

Experienced Team

- Key roles in oncology R&D at both biotech and pharma
- Members of team have worked together since 2007 at Intellikine and Wellspring Biosciences

Solid Financials

- $78.5M cash as of March 31, 2016*
- Resources expected to fund current operations into 2018

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


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