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): June 16, 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):

☐ 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 June 16, 2016, 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: June 16, 2016

By: /s/ Annette North

Annette North
SVP, General Counsel

Exhibit 99.1

KURA ONCOLOGY

Troy Wilson, Ph.D., J.D.
President and CEO
June 16, 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

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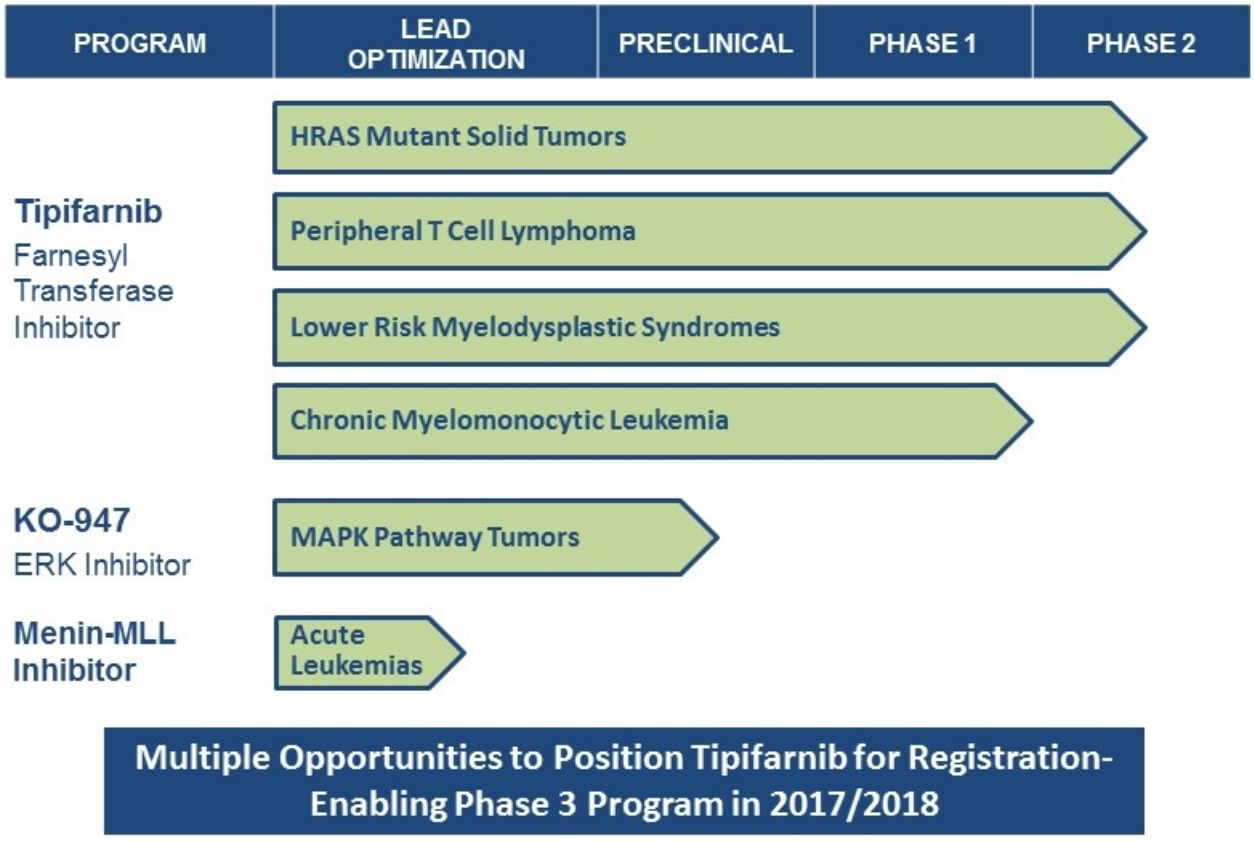
* Includes Cash, Cash Equivalents, and Short-Term Investments

DIVERSE DEVELOPMENT PIPELINE



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

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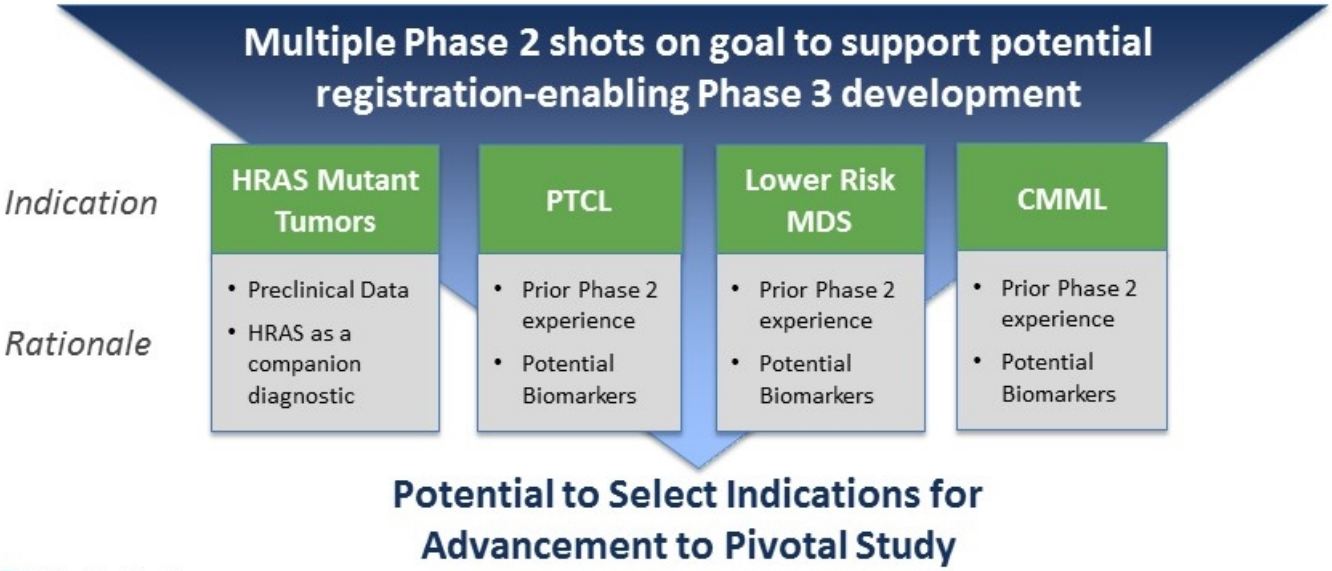
TIPIFARNIB – LEAD PRODUCT CANDIDATE

- 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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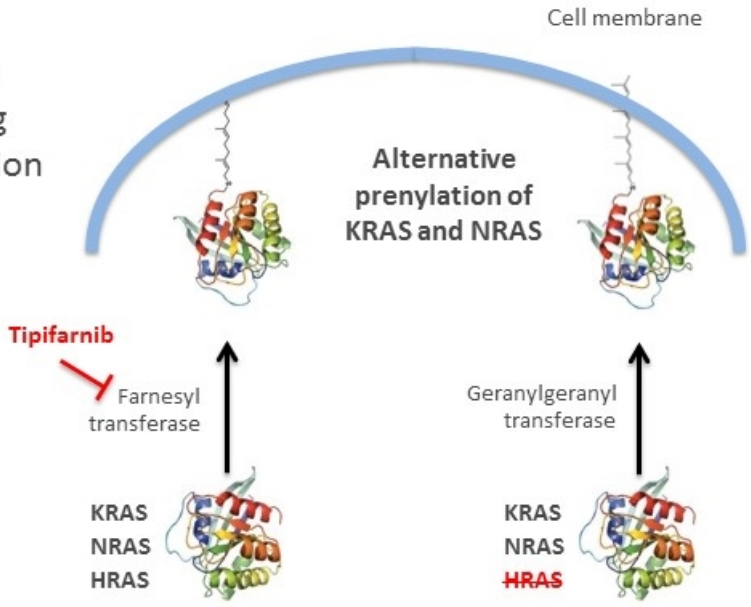
MECHANISM OF ACTION OF TIPIFARNIB

- 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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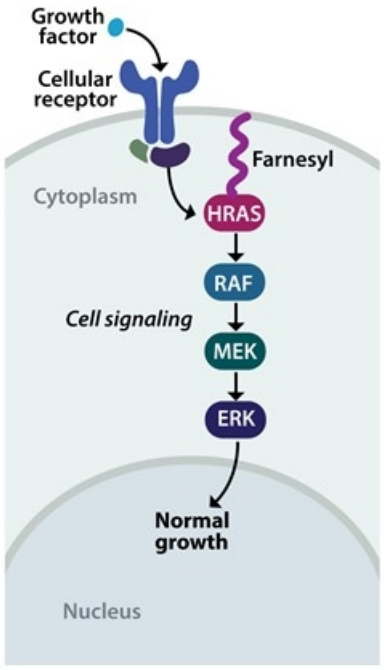
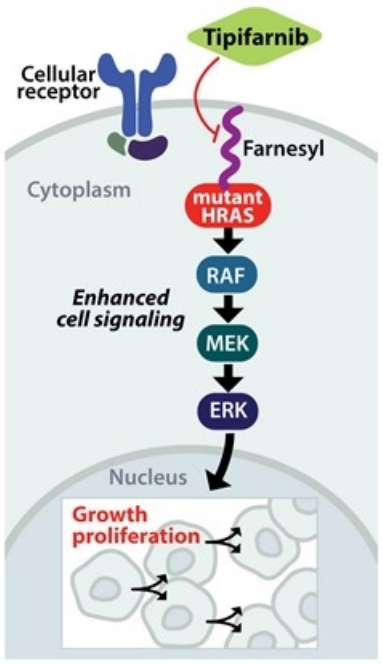
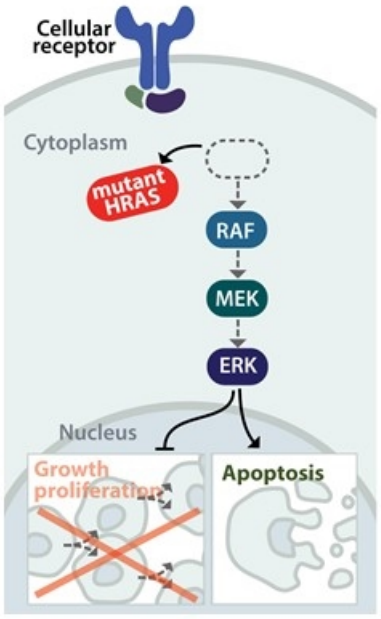
TIPIFARNIB MOA SUPPORTS DEVELOPMENT IN HRAS MUTANT SOLID TUMORS



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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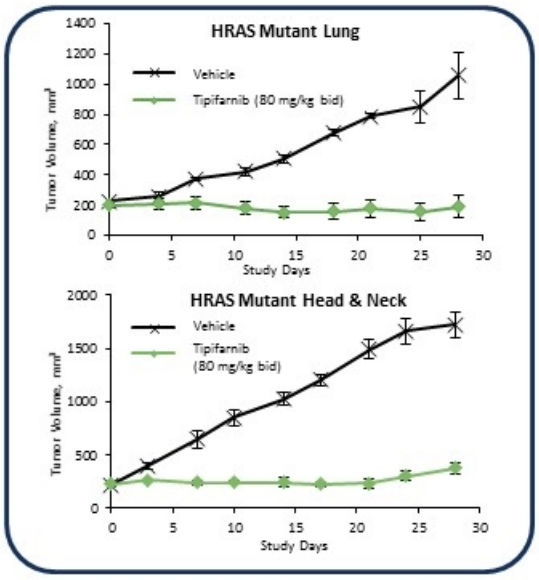
RATIONALE AND DESIGN OF PHASE 2 TRIALS IN HRAS MUTANT TUMORS



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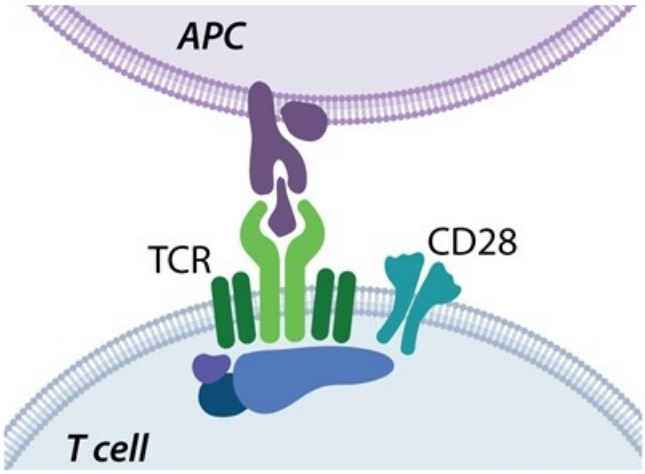
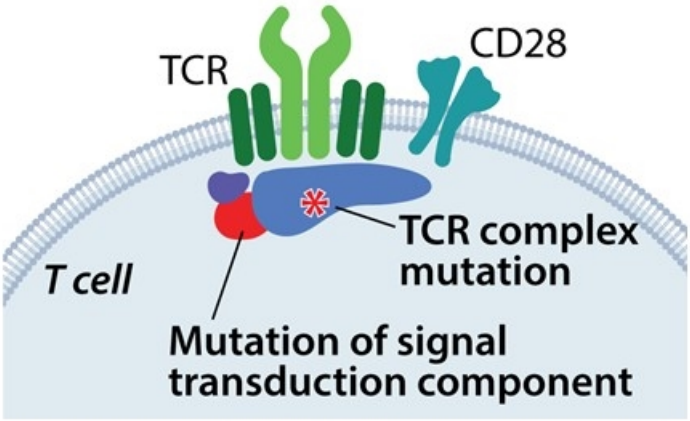
*NCI-Sponsored Phase 2 Trial in Relapsed/Refractory Lymphomas; *Blood. 2011;118(18):4882-9*

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.



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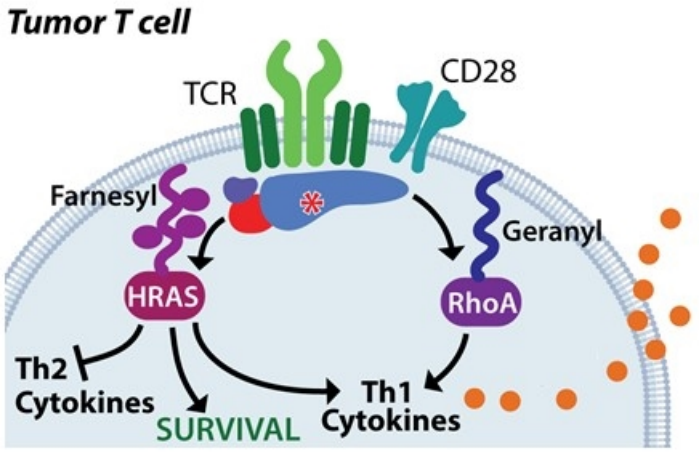
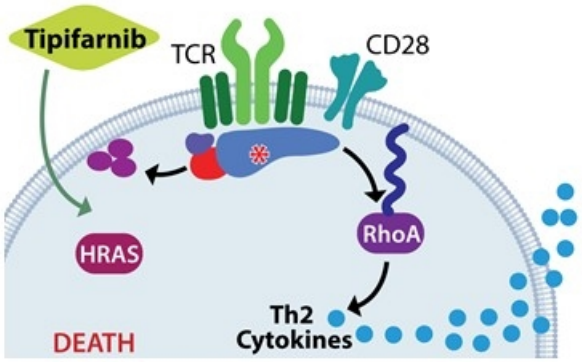
FARNESYLATION OF KEY SIGNALING PROTEINS IN T CELLS SUPPORTS DEVELOPMENT APPROACH



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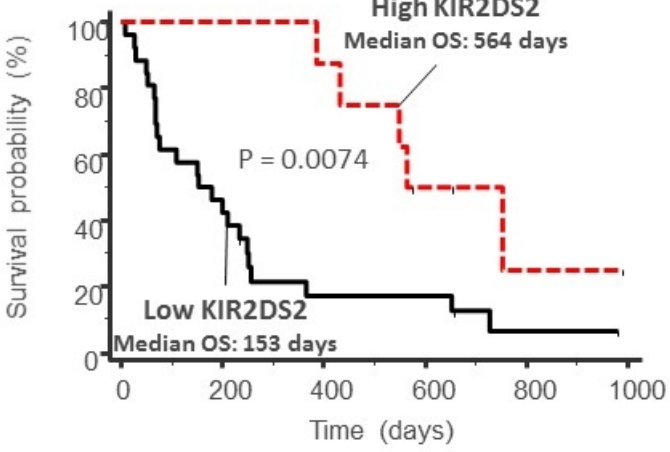
*Study INT-28; Blood. 2007;109(10):4158-63

KIR2DS2 AS A POTENTIAL BIOMARKER IN MDS

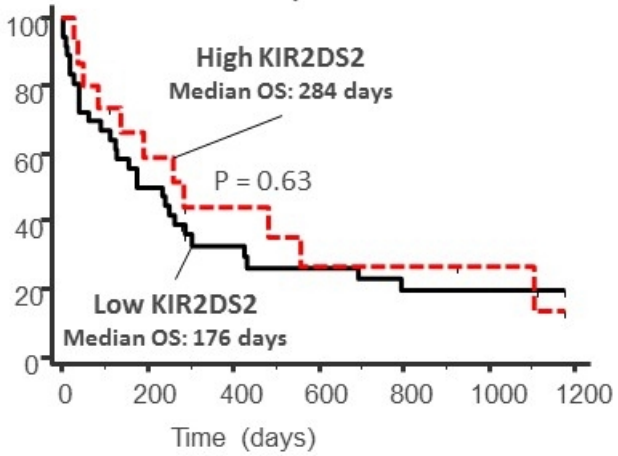
Tipifarnib Treated Patients (n=34)

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

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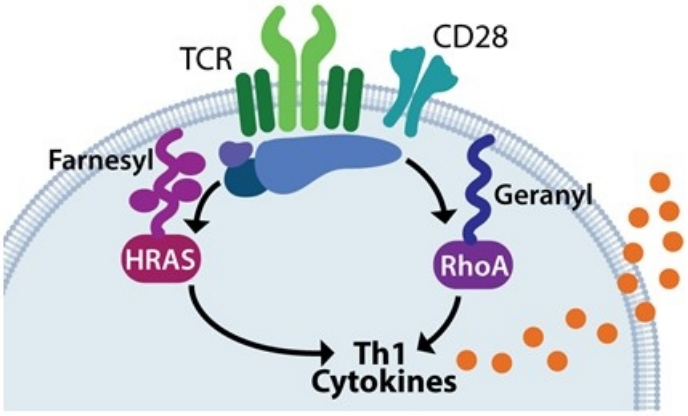
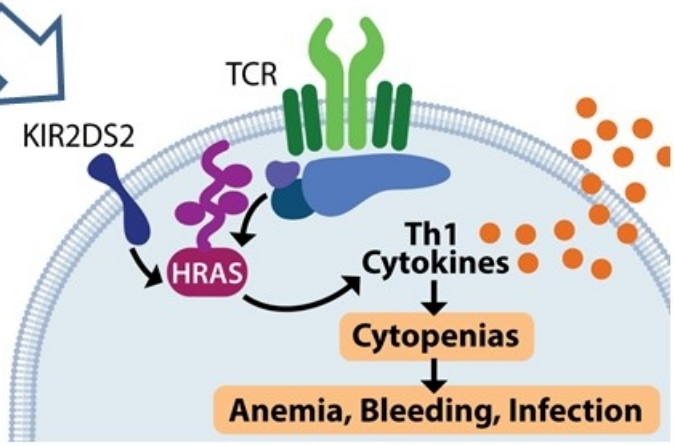
SIGNALING PATHWAYS IN MDS HAVE POTENTIAL TO YIELD PREDICTIVE BIOMARKERS



Aging



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

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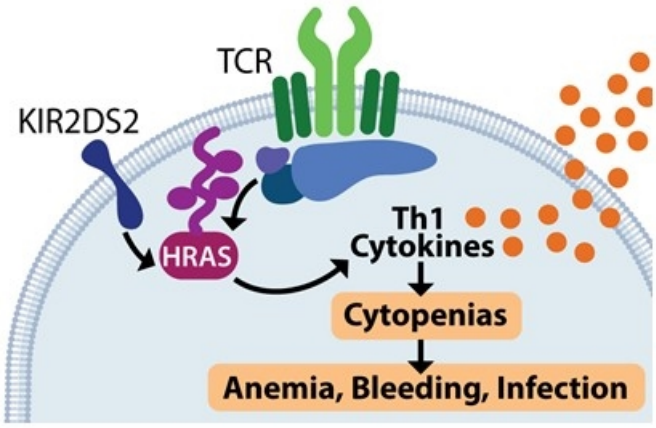
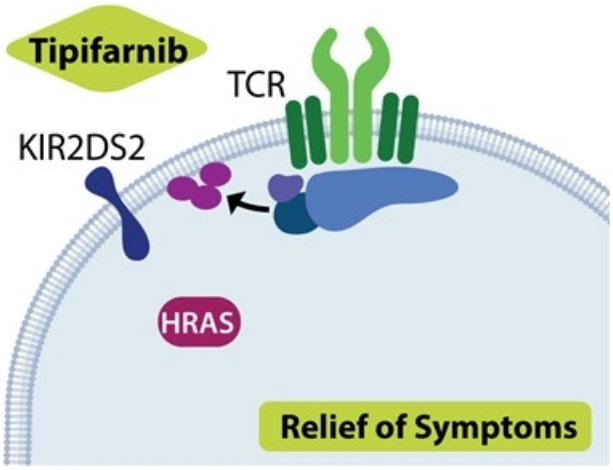
SIGNALING PATHWAYS IN MDS HAVE POTENTIAL TO YIELD PREDICTIVE BIOMARKERS

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





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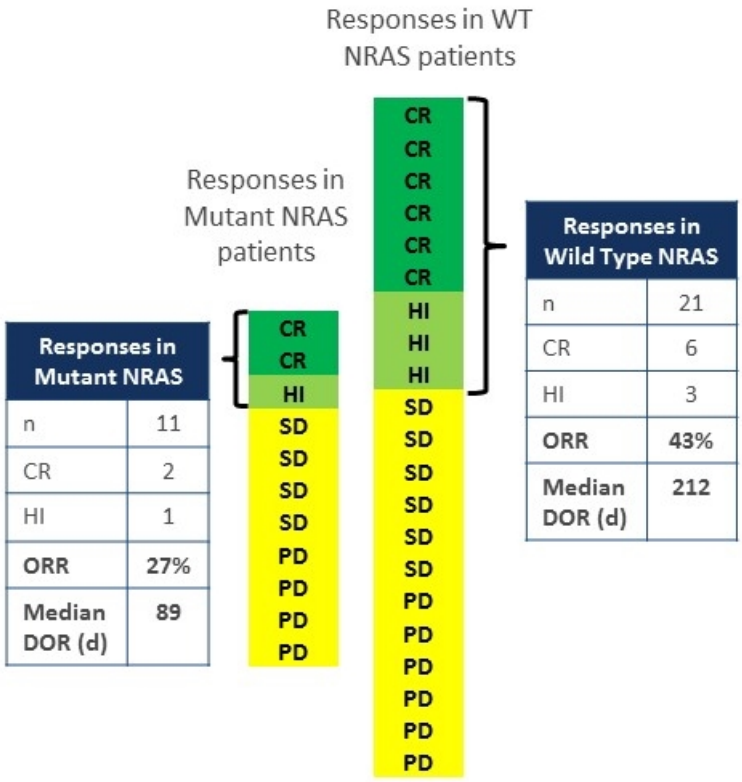
*INT-28: J&J clinical data; ITT population; CMML defined by FAB criteria. Response criteria as in Cheson et al. Blood 2000;96:3671-4

† 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: J&J clinical data; 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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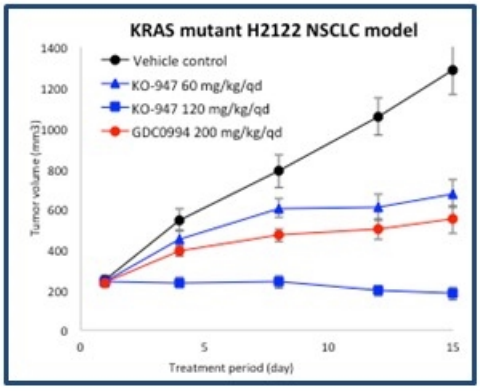
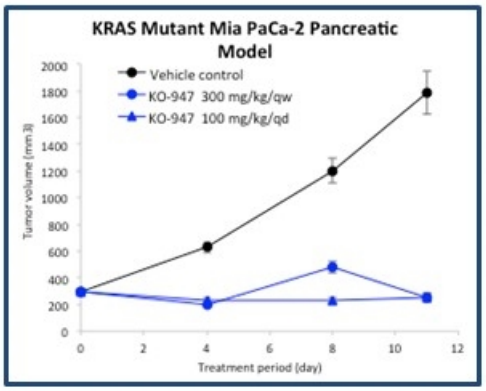
KO-947 – STRONG ACTIVITY IN MAPK PATHWAY MODELS WITH FLEXIBLE DOSING

- 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 multiple tumor models at tolerable doses and compares favorably to clinical-stage reference compounds
- Advancing an intravenous administration, which has the potential to improve exposure and tolerability
- IND submission pending completion of drug product manufacturing activities





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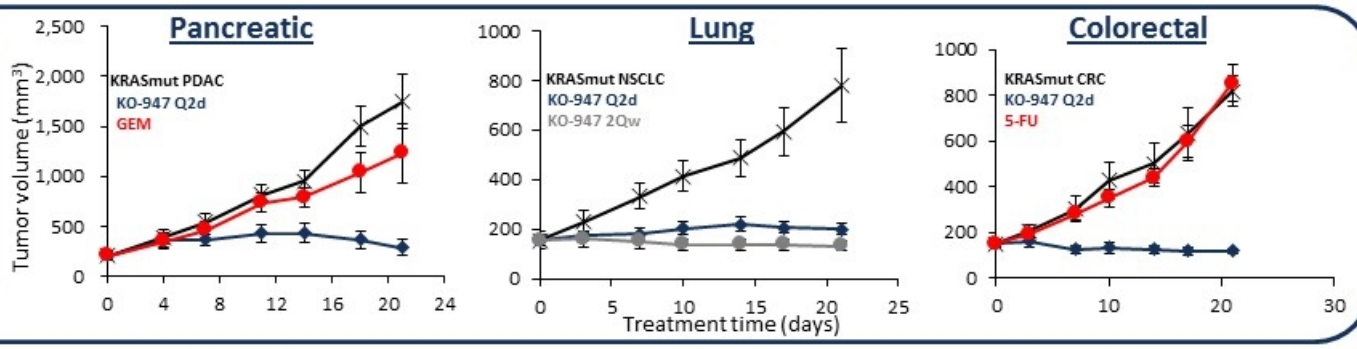
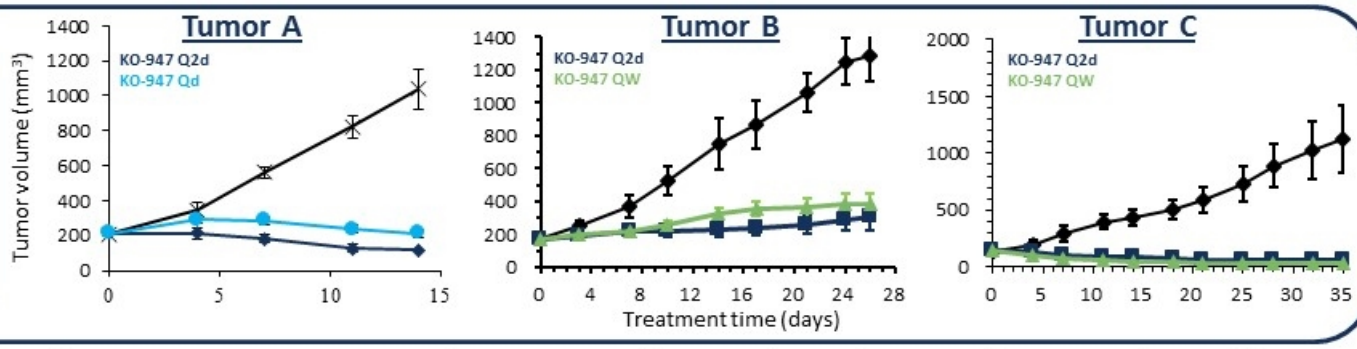
KO-947: TRANSLATIONAL RESEARCH IDENTIFIED POTENTIAL LEAD CLINICAL INDICATIONS

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	2H 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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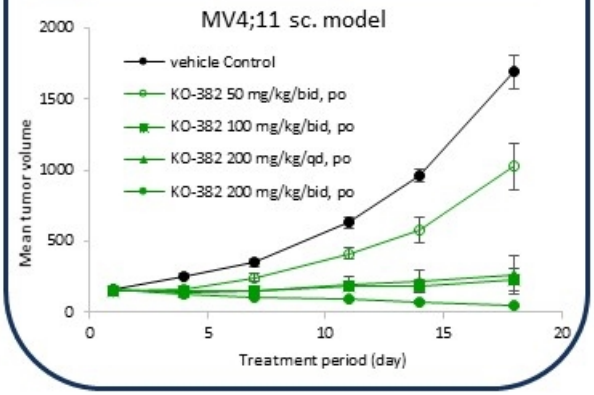
MENIN-MLL INHIBITOR PROGRAM

- 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	2H 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

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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

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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