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): January 7, 2019

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 or Rule 12b-2 of the Securities Exchange Act of 1934.

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 January 7, 2019, 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.

SIGNATURES

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: January 7, 2019

By: /s/ Annette North

Annette North
Senior Vice President and General Counsel

KURA ONCOLOGY

Exhibit 99.1

Developing Precision Medicines
for the Treatment of Cancer

Corporate Presentation

January 2019

Forward-Looking Statements

This presentation contains forward-looking statements. Such statements include, but are not limited to, statements regarding our research, preclinical and clinical development activities, plans and projected timelines for tipifarnib, KO-947 and KO-539, plans regarding regulatory filings, our expectations regarding the relative benefits of our product candidates versus competitive therapies, and our expectations regarding the therapeutic and commercial potential of our product candidates. The words "believe," "may," "will," "estimate," "promise," "plan", "continue," "anticipate," "intend," "expect," "potential" and similar expressions (including the negative thereof), are intended to identify forward-looking statements. Because such statements are subject to risks and uncertainties, actual results may differ materially from those expressed or implied by such forward-looking statements. Risks that contribute to the uncertain nature of the forward-looking statements include: our preclinical studies and clinical trials may not be successful; the U.S. Food and Drug Administration (FDA) may not agree with our interpretation of the data from clinical trials of our product candidates; we may decide, or the FDA may require us, to conduct additional clinical trials or to modify our ongoing clinical trials; we may experience delays in the commencement, enrollment, completion or analysis of clinical testing for our product candidates, or significant issues regarding the adequacy of our clinical trial designs or the execution of our clinical trials may arise, which could result in increased costs and delays, or limit our ability to obtain regulatory approval; our product candidates may not receive regulatory approval or be successfully commercialized; unexpected adverse side effects or inadequate therapeutic efficacy of our product candidates could delay or prevent regulatory approval or commercialization; and we may not be able to obtain additional financing. Additional risks and uncertainties may emerge from time to time, and it is not possible for Kura's management to predict all risk factors and uncertainties.

All forward-looking statements contained in this presentation speak only as of the date on which they were made. Other risks and uncertainties affecting us are described more fully in our filings with the Securities and Exchange Commission. We undertake no obligation to update such statements to reflect events that occur or circumstances that exist after the date on which they were made.

Investment Highlights

Targeted Oncology	Advance pipeline of targeted drug candidates for selected solid tumors and hematologic malignancies Utilize precision medicine approaches; Fast-to-market potential
Proprietary Pipeline	**Tipifarnib:** Potent farnesyl transferase inhibitor; Registration-directed and multiple Phase 2 trials ongoing; Biomarker-guided development; Issued patents and potential for regulatory exclusivity **KO-947:** ERK inhibitor; Phase 1 dose-escalation trial ongoing **KO-539:** Inhibitor of menin-MLL interaction; IND filing Q1 2019
Near-Term Milestones	Additional Phase 2 data in HRAS mutant SCCs and CXCL12+ hematologic malignancies
Team	Proven oncology drug development experience
Financials	$187.4M cash as of September 30, 3018*

* Includes cash, cash equivalents and short-term investments

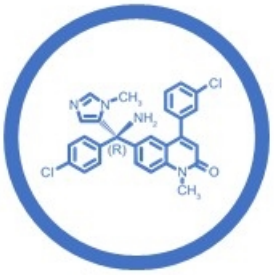
Advancing Pipeline of Targeted Drug Candidates

	Tipifarnib	KO-947	KO-539
Therapeutic Target	• Farnesyl transferase	• ERK kinase	• Menin-MLL interaction
Biomarker Strategies	• HRAS mutant solid tumors • CXCL12-expressing hematologic malignancies and solid tumors	• MAPK-pathway dysregulated tumors • 11q13 amplified solid tumors	• MLL-rearranged (MLL-r) leukemias • NPM1 and DNMT3A mutant liquid tumors
Development Status	• Registration-directed study and multiple Phase 2 trials ongoing* • Two biomarkers identified with issued patents	• Ongoing Phase 1 dose-escalation trial	• IND anticipated Q1 2019

* Tipifarnib previously studied in > 5,000 patients in > 70 studies with a manageable safety profile as a single agent
Note: Chemical structures of KO-947 and KO-539 not published

Biomarker Strategies May Unlock Clinical Activity and Commercial Value

Targeted Therapy



Analytical Technologies

(next-generation sequencing, expression profiling, etc.)

Selected Patient Population

Potential Value

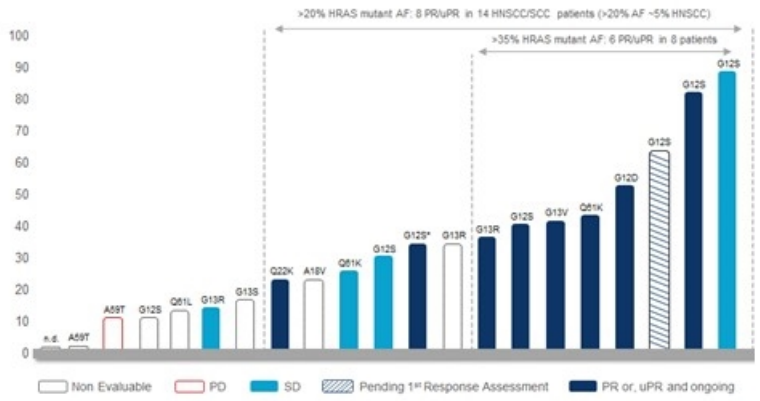
- Enrichment of clinical activity
- Higher probability of success
- Expedited development and regulatory path
- Strong commercial case

Multiple Clinical Proof-of-Concepts Reinforce Precision Medicine Approach

HRAS Mutant
HNSCC

HRAS Mutant Allele Frequency

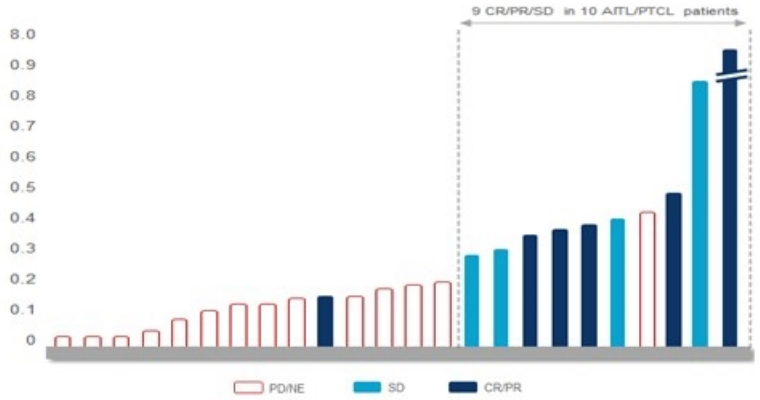
Clinical benefit observed in **high frequency HRAS mutant** population



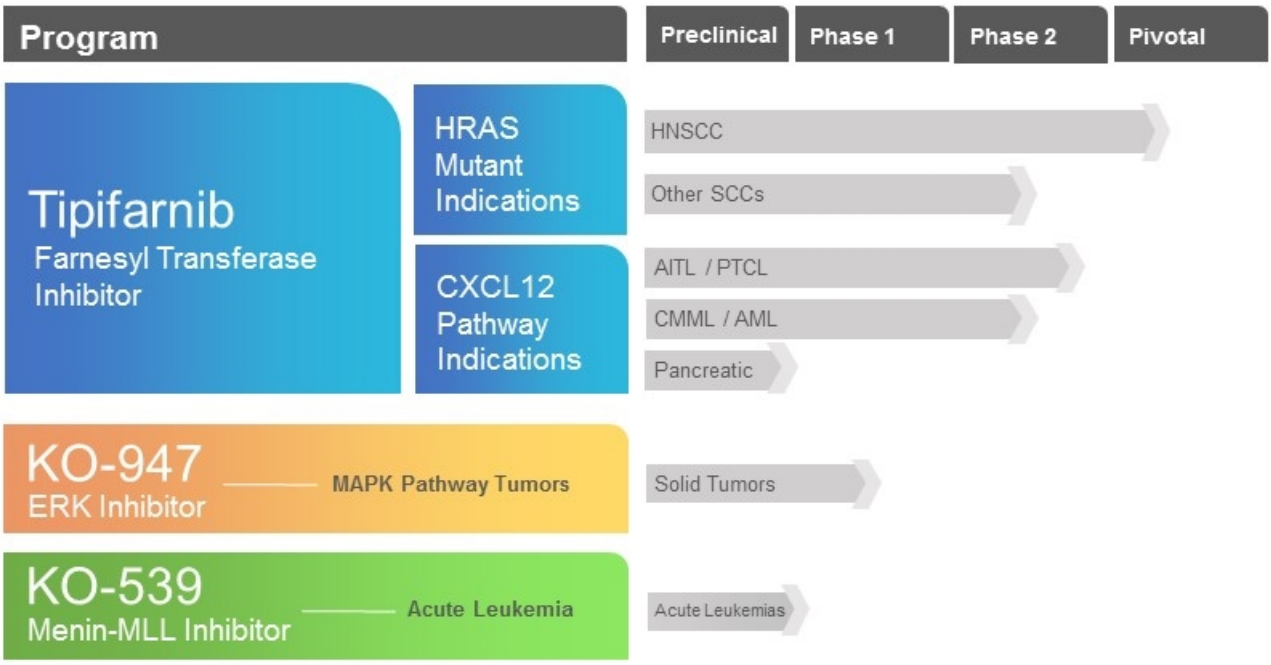
CXCL12+
AITL/PTCL

High CXCL12 Levels

Clinical benefit observed in **high CXCL12 AITL / PTCL** population



Product Candidate Pipeline



Investigator-Sponsored Trials | HRAS Mutant Urothelial Carcinomas, Samsung Medical Center | HRAS Mutant Lung Squamous Cell Carcinomas (LSCC), Spanish Lung Cancer Group

Note: Given current focus in HRAS mutant and CXCL12 pathway indications, Phase 2 study in MDS has been deprioritized and is not currently enrolling new patients

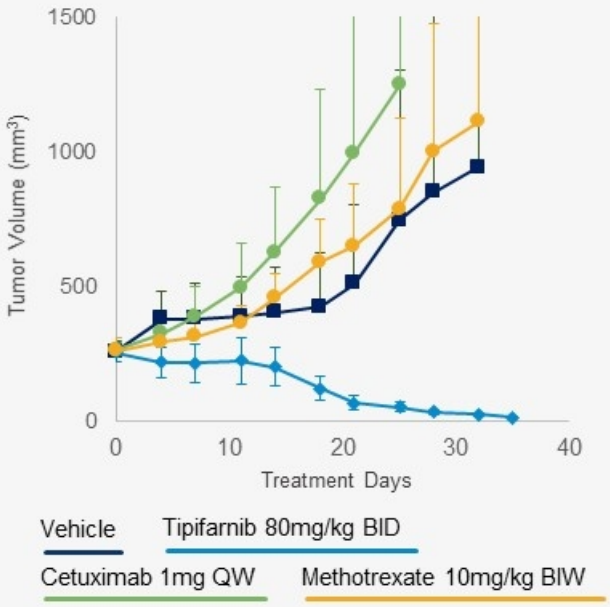
01 • *Tipifarnib in HRAS Mutant Solid Tumors*

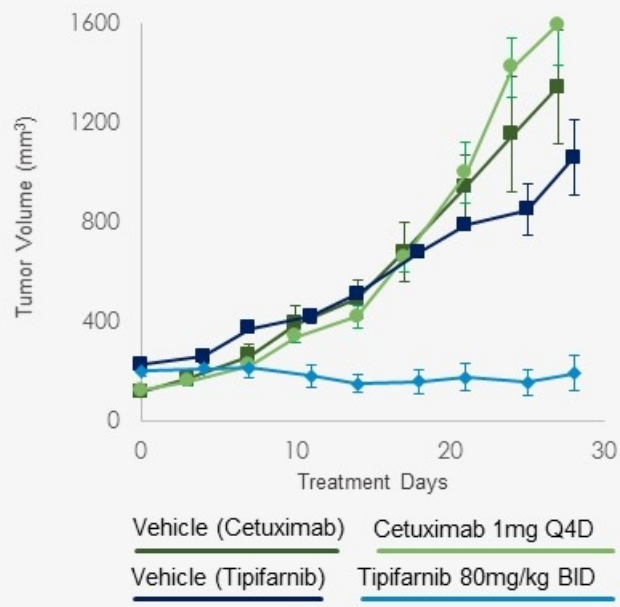
02 • *Tipifarnib Using CXCL12 Pathway Biomarkers*

03 • *KO-947 (ERK Inhibitor)*

04 • *KO-539 (Menin-MLL Inhibitor)*

Tipifarnib is a Potent Inhibitor of HRAS Mutant Tumors

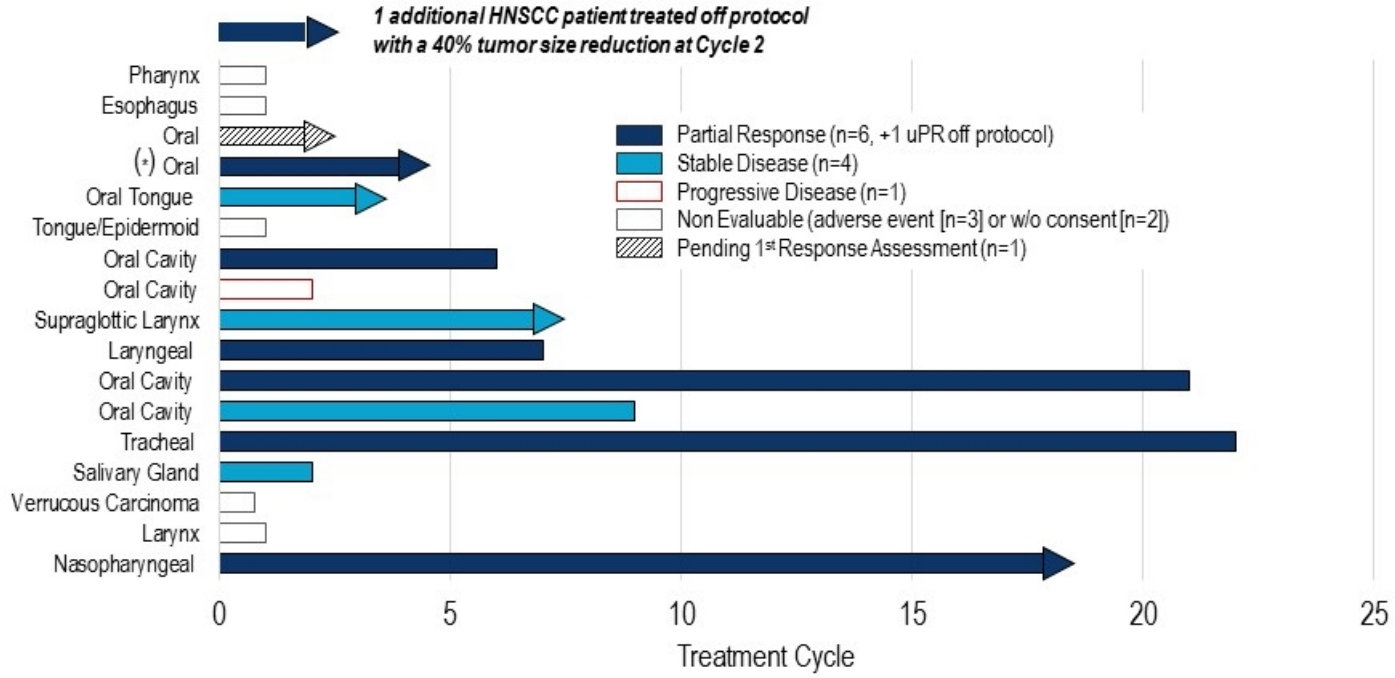




- Regressions observed in preclinical PDX models of SCC carrying the HRAS mutant oncogene
- Stasis or regression observed in other tumor types carrying HRAS mutations

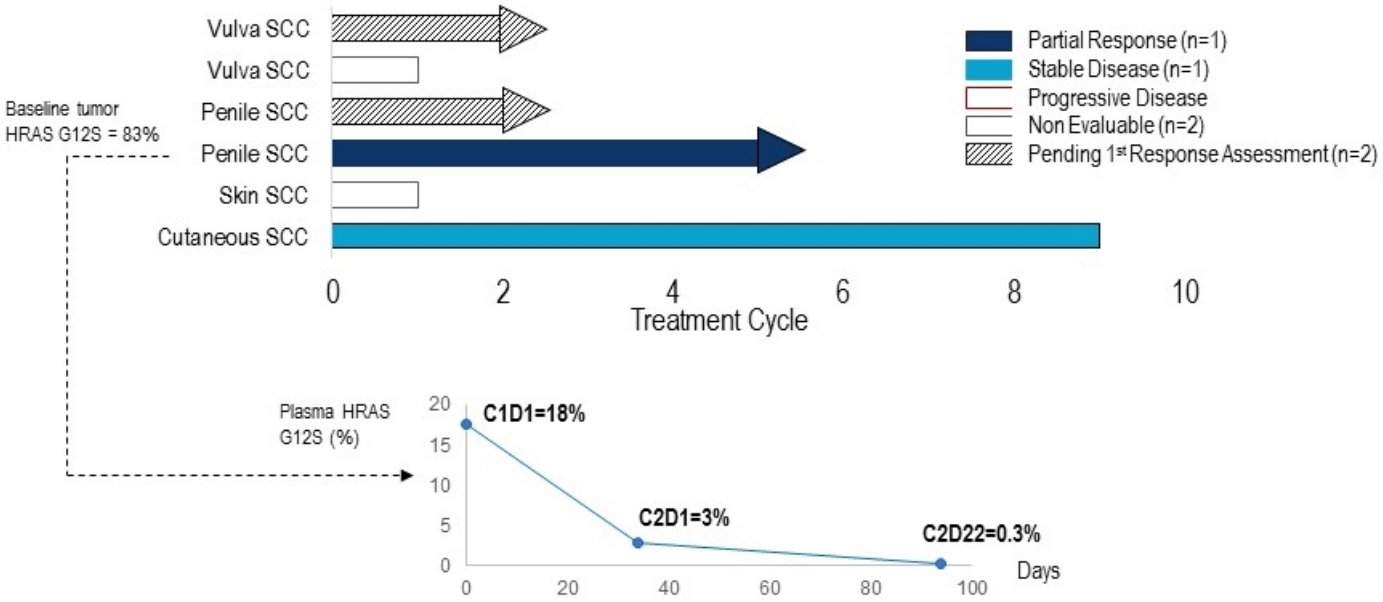
Phase 2 Study of Tipifarnib: HNSCC Patients

(n=17 on study + 1 patient treated off protocol)



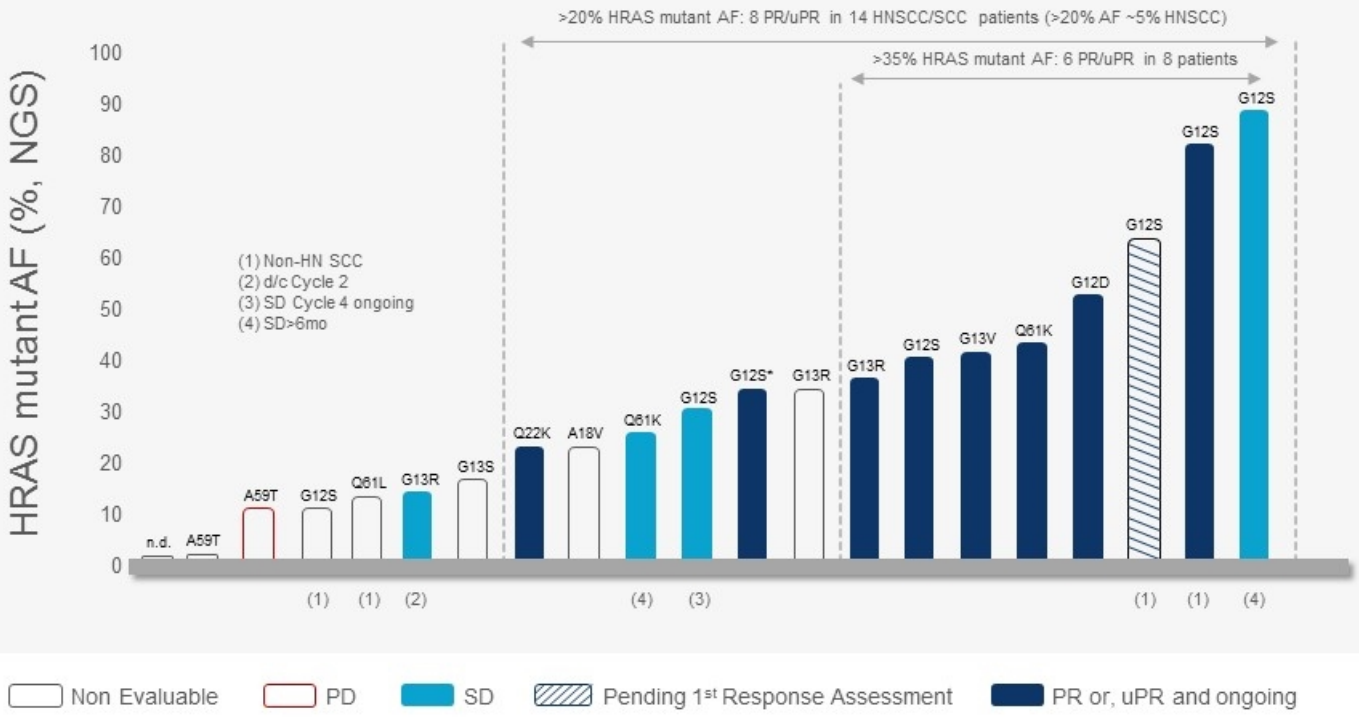
Ho *et al*. European Society for Medical Oncology 2018 Congress #1046O | Preliminary results as of 9/7/18
* Response confirmed on 10/15/18

Phase 2 Study of Tipifarnib: Other SCC Patients (n=6)



Association of HRAS Mutant Allele Frequency with Clinical Benefit from Tipifarnib

(HNSCC, SCC, n=21)



Ho *et al*. European Society for Medical Oncology 2018 Congress #1046 | Study KO-TIP-001 patients with HN and non-HN SCC tumors with available HRAS mutant allele data (10/17/18); one additional HNSCC patient was treated off protocol |
* Allele frequency obtained post-ESMO | Pending analysis: 1 HNSCC pending 1st scan, 1 SCC pending 1st scan, 1 SCC SD

Tipifarnib Development Program in HRAS Mutant HNSCC

RUN-HN
KO-TIP-001

Phase 2
Proof-of-Concept Study

SEQ-HN
KO-TIP-007

Non-Interventional
Screening & Outcomes Cohort

AIM-HN
KO-TIP-007

Registration-Directed Trial

HRAS mutant patients who are not eligible for participation in AIM-HN may be referred to RUN-HN

AIM-HN: Global, multi-center registration directed trial of tipifarnib in HRAS mutant HNSCC

SEQ-HN: Matched control study to identify HRAS mutant HNSCC patients and characterize activity of standard of care

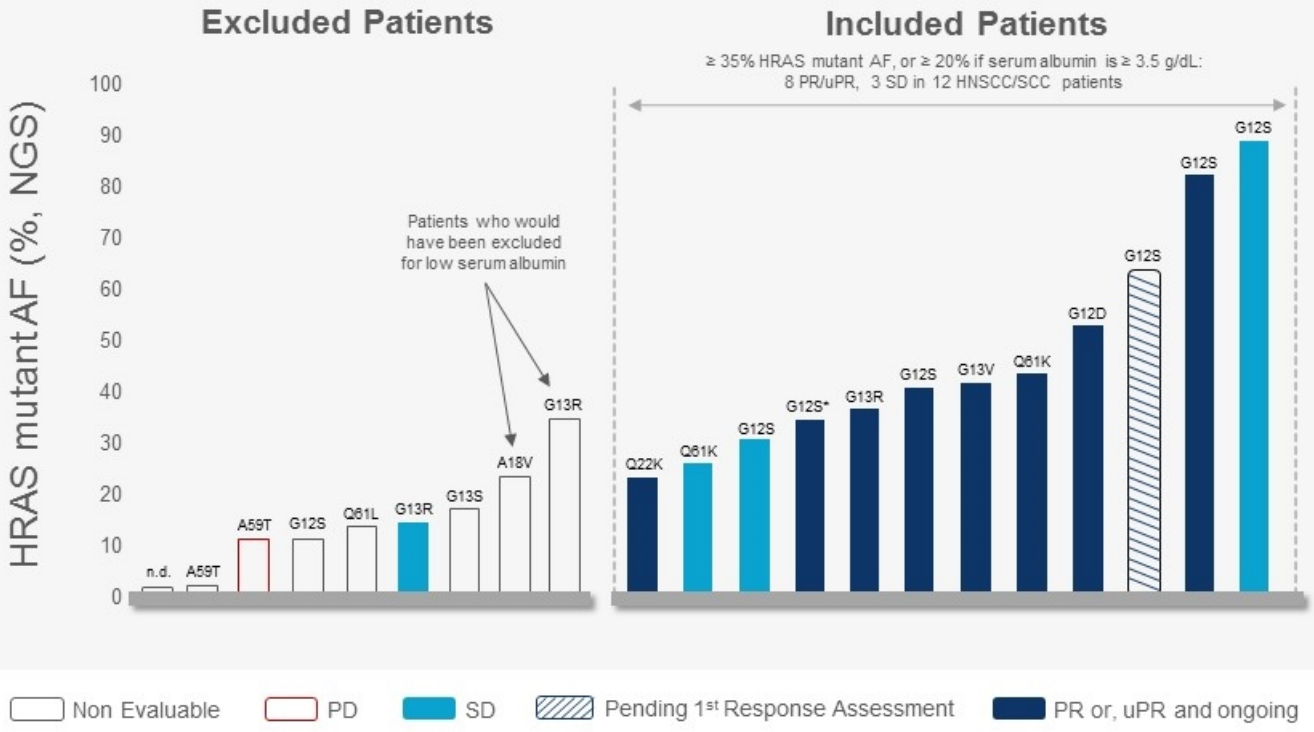
AIM-HN: Trial Design

AIM-HN
KO-TIP-007

- **Global, registration-directed trial**
  - Targeting ~ 100 clinical sites worldwide
  - Anticipate ~ two years to enroll
- **Primary endpoint: ORR by IRR**
- **Statistical assumptions**
  - At least 59 subjects, 80% power, 15% ORR (null hypothesis) and 30% ORR (response rate of interest)
- **Minimum tumor HRAS mutant allele frequency of 20%**
  - Tumor HRAS mutation with an allele frequency ≥ 35%, or ≥ 20% if serum albumin is ≥ 3.5 g/dL
- **600 mg BID starting dose given daily in alternate weeks**
- **As currently designed, AIM-HN may be adequate to support an NDA seeking accelerated approval (FDA end of Phase 2 meeting)**
  - SEQ-HN data to provide a benchmark of the activity of standard of care in HRAS mutant HNSCC (relevant for potential label discussion and post approval commitments)
- **Trial initiated and open for enrollment in November 2018**

Activity When AIM-HN Criteria Applied Retrospectively to Phase 2 Study

(HNSCC, SCC, n=21)



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* Allele frequency obtained post-ESMO | Pending analysis: 1 HNSCC pending 1st scan, 1 SCC pending 1st scan, 1 SCC SD

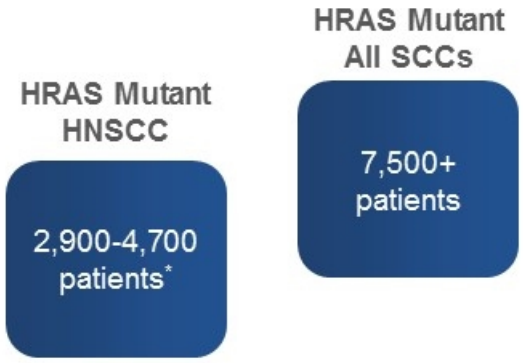
HRAS Mutant Cancers: Market Opportunity

HNSCC Represents Significant Unmet Need[1]

1L	2L
ORR 36%	ORR 13-16%
PFS 5.6 months	PFS ~2 months
OS ~10 months	OS 6-8 months

- Outcome of SOC in unselected populations
- Lower response rate expected in HRAS mutant patients[2]

Populations Based on Annual U.S. Incidence



[1] N Engl J Med. 2008 Sep 11;359(11):1116-27 | Keytruda & Opdivo package inserts | J Clin Oncol. 2007 Jun 1;25(16):2171-7
[2] Journal of Clinical Oncology 2012 30:15_suppl, 5574-5574
* Estimate is between 5-8% of total HNSCC population, depending on allele frequency of HRAS mutations (Source: TCGA, internal data)

02. Tipifarnib Using CXCL12 Pathway Biomarkers

01 • Tipifarnib in HRAS Mutant Solid Tumors

03 • KO-947 (ERK Inhibitor)

04 • KO-539 (Menin-MLL Inhibitor)

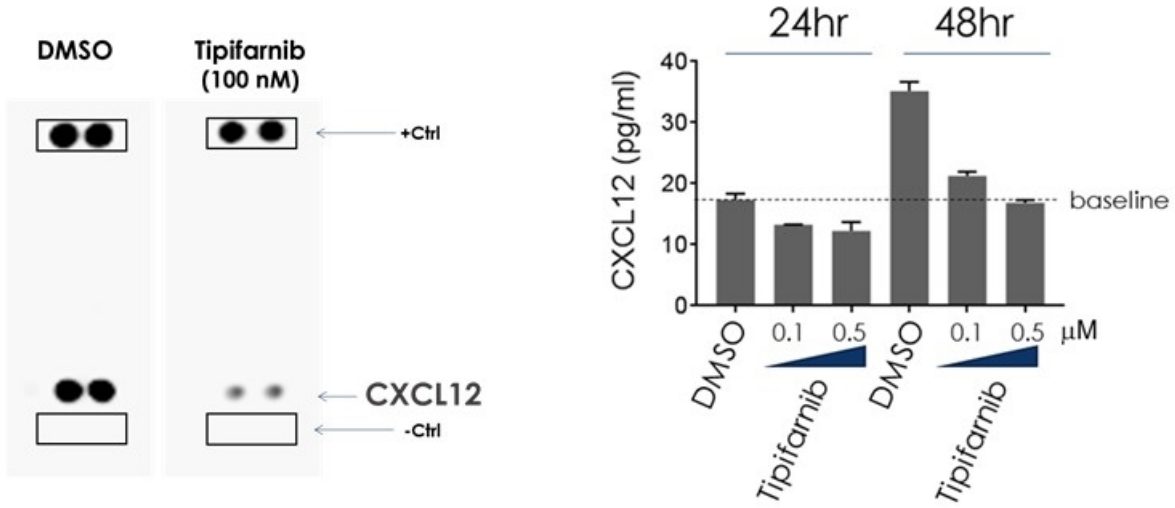
Relevance of CXCL12 Inhibition as a Targeted Therapy

- **Key characteristics of CXCL12**
  - Expressed primarily by immune cells, endothelial cells and stromal fibroblasts that constitute the tumor microenvironment
  - Binds and activates two receptors, CXCR4 and CXCR7
  - CXCL12 and its receptors are key factors linking cancer cells with tumor microenvironment

- **Potential role of CXCL12 inhibition in cancer therapy**
  - Inhibition of growth and homing of lymphoid and myeloid tumors[1]
  - Interference with tumor cell metastasis into secondary organs, *e.g.* inhibition of bone recurrence of solid tumors in adjuvant settings – after primary tumor is removed by surgery/chemoradiation[2]
  - Reversion of the tolerogenic effect of a tumor microenvironment rich in immunosuppressive cells such as regulatory T-cells and neutrophils, *e.g.* synergy of CXCL12 and PD-L1 inhibition in pancreatic tumor models[3]

[1] Burger *et al.* 2007. *Br J Haematol.* 137:288-96 | [2] Epstein 2004. *Nat Rev Cancer* 4:901-9 |
[3] Feig *et al.* 2013. *Proc Natl Acad Sci U S A.* 110:20212-7

Tipifarnib is a Potent Inhibitor of CXCL12 Secretion by Stromal Cells



- Primary CD1 mouse model of bone marrow cultures secrete abundant CXCL12 (SDF1, stroma derived factor 1) that was decreased by tipifarnib
- Research on specific farnesylated targets ongoing

02a • *Tipifarnib Using CXCL12 Pathway Biomarkers: PTCL / AITL*

01 • *Tipifarnib in HRAS Mutant Solid Tumors*

03 • *KO-947 (ERK Inhibitor)*

04 • *KO-539 (Menin-MLL Inhibitor)*

PTCL: CXCL12-Expressing Lymphoma with a Significant Unmet Need

	BELEODAQ® (BELINOSTAT)	ISTODAX® (ROMIDEPSIN)	FOLOTYN® (PRALATREXATE)
Efficacy Study	Single Arm[1] N=120	Single Arm[2] N=130	Single Arm[3] N=109
Prior Therapies (range)	2 (1-8)	2 (1-8)	3 (1-12)
Overall Response Rate	**25.8%**	**26.2%**	**27%**
Median PFS/TTP	1.6 months	4.0 months	3.5 months
Median Overall Survival	7.9 months	11.3 months	14.5 months
Dosing	IV infusion[4]	IV infusion[5]	IV push[6]

Approved therapies in relapsed / refractory PTCL approved based on single-arm clinical trials of 130 patients or fewer with response rates in the range of 25-27% and limited duration of clinical benefit in unselected populations

[1] Beleodaq® package insert
[2] Istodax® package insert
[3] Folotyn® package insert
[4] 1,000 mg/m² administered over 30 mins by IV infusion once daily on days 1-5 of a 21-day cycle
[5] 14 mg/m² administered over a 4-hour period by IV on days 1, 8 and 15 of a 28-day cycle
[6] 30 mg/m² administered over 3-5 mins as an IV push once weekly for 6 weeks in 7-week cycles

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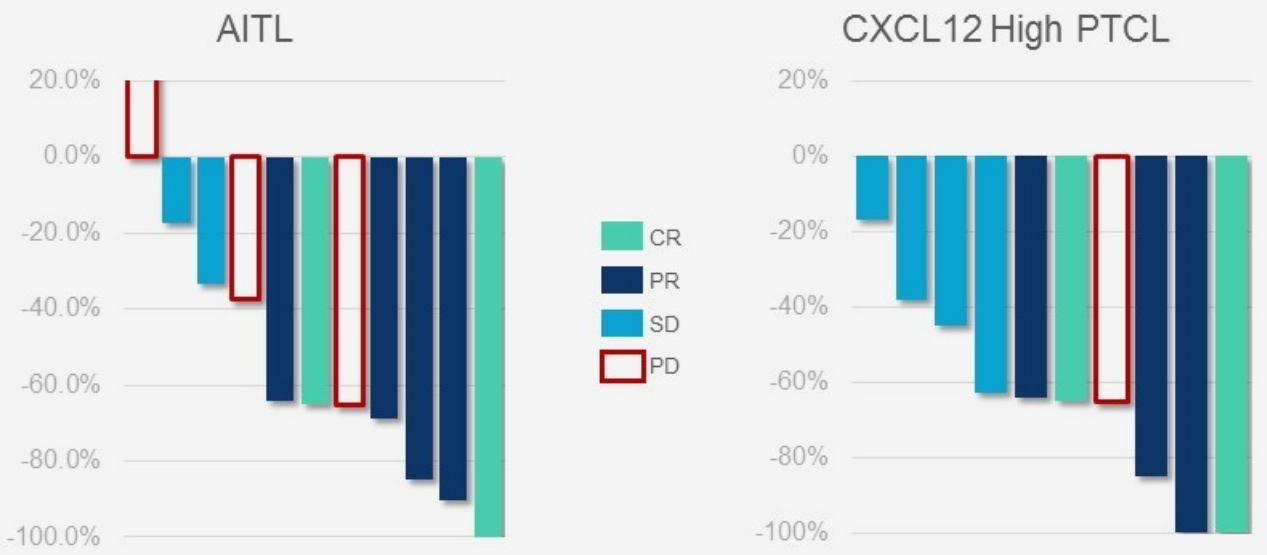
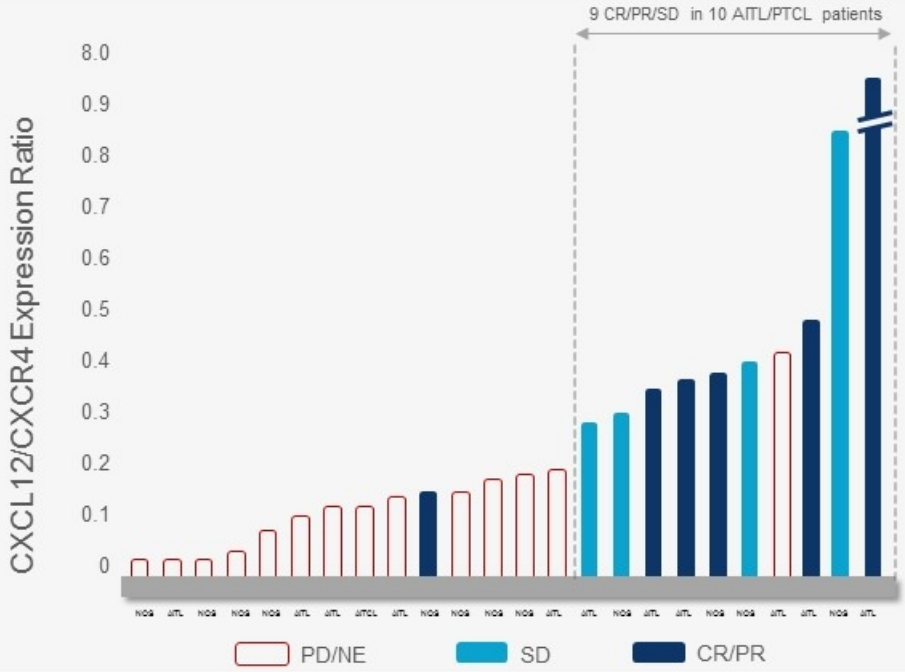
Clinical Activity in Phase 2 Study of Tipifarnib

Change in SPD (%)



Witzig *et al.* ASH 2018 #2937 | Preliminary data as of 11/21/18 | Missing measurement data from 2 subjects with best response of PD
SPD: Sum of the products of diameters

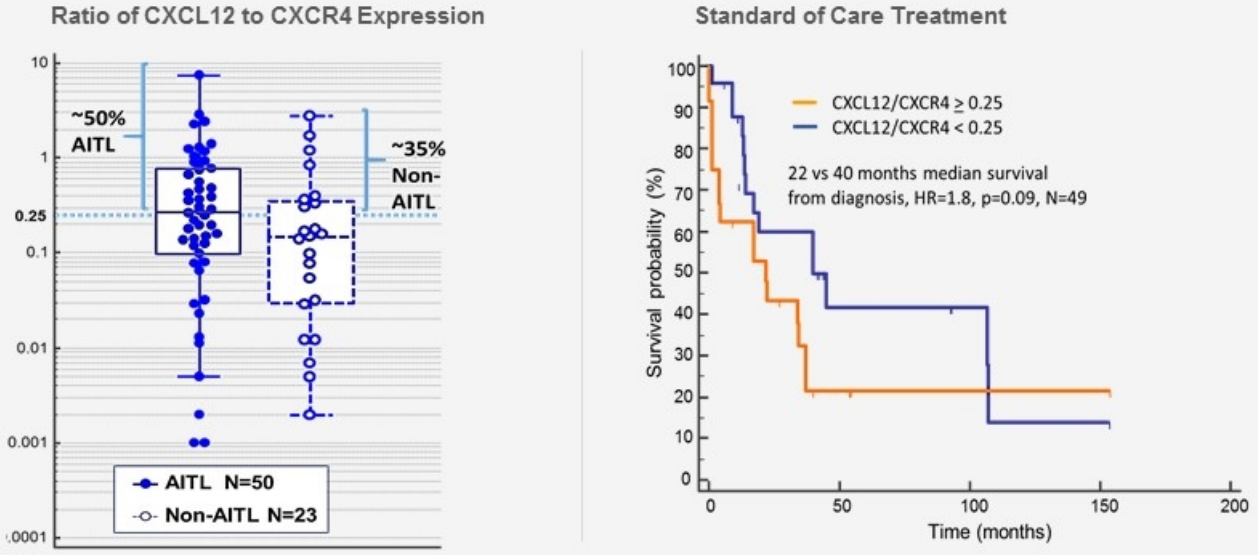
Association of High CXCL12 with Clinical Benefit from Tipifarnib in AITL/PTCL



- The High CXCL12/CXCR4 subset of PTCL patients experienced **50% ORR and 90% clinical benefit** with tipifarnib after a median of 3 prior therapies
- High CXCL12/CXCR4 expression ratio had 90% sensitivity and 93% specificity to identify PTCL patients likely to benefit from tipifarnib

Witzig *et al.* ASH 2018 #2937 | Preliminary data as of 11/21/18 | Expression data generated using RNA-Seq

High CXCL12 Defines Poor Prognosis with Standard of Care Therapy in PTCL



- A trend for worse prognosis was observed in PTCL patients with high CXCL12/CXCR4 expression ratio when treated with standard of care therapy
- Increasing levels of CXCL12 resulted in significantly more negative prognosis for SOC (not shown)
- CXCL12 high subset of patients represents ~40% of PTCL

Witzig *et al*. ASH 2018 #2937

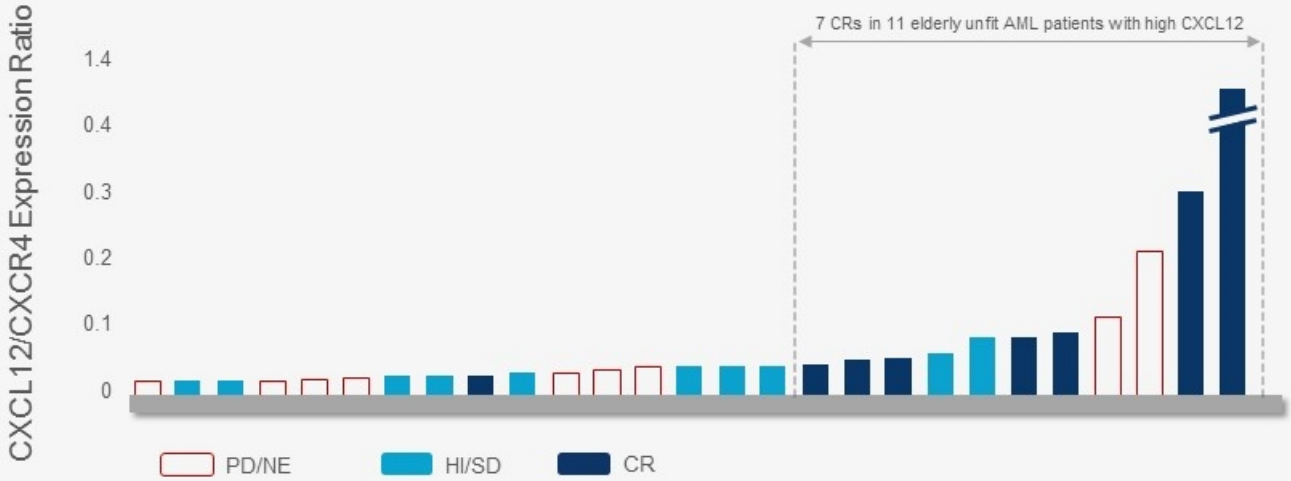
02b • *Tipifarnib Using CXCL12 Pathway Biomarkers: Other Hematologic Malignancies*

01 • *Tipifarnib in HRAS Mutant Solid Tumors*

03 • *KO-947 (ERK Inhibitor)*

04 • *KO-539 (Menin-MLL Inhibitor)*

Association of High CXCL12 with Activity of Tipifarnib in Elderly Unfit AML



- CTEP20 study: Patient subset – available AML marrow samples with NRAS WT or unknown, N=27 (NCBI GEO, GSE8970)[1]
- Analysis limited to K/NRAS-wild type patients as K/NRAS mutant tumors are unlikely to be CXCL12-dependent

[1] Blood. 2008 Mar 1;111(5):2589-96 | Expression data generated using microarray | HI: Hematologic Improvement

Opportunities in Other CXCL12-Expressing Hematologic Indications

Disease Type	n	CR, n (%)	PR, n (%)	ORR, (%)
Diffuse large B-Cell Lymphoma (DLBCL)	37	0	7 (19)	19
Hodgkin Lymphoma	19	2 (11)	2 (11)	21
Mycosis Fungoides	4	0	2 (50)	50

- Responses previously observed in patients with other relapsed/refractory hematologic tumors (unselected population) in NCI-sponsored Phase 2 trial of tipifarnib

Witzig *et al. Blood.* 2011 Nov 3;118(18):4882-9

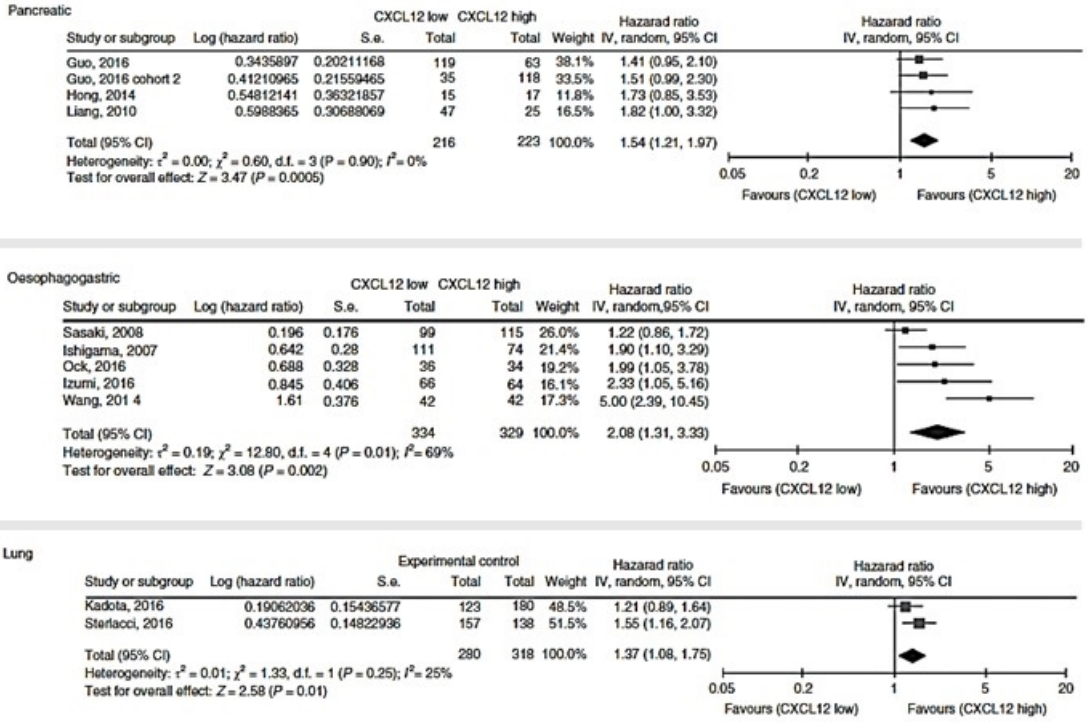
02c • Tipifarnib Using CXCL12 Pathway Biomarkers: Solid Tumors

01 • Tipifarnib in HRAS Mutant Solid Tumors

03 • KO-947 (ERK Inhibitor)

04 • KO-539 (Menin-MLL Inhibitor)

Solid Tumors: Known Unmet Need in High CXCL12 Tumor Subsets



- High CXCL12 expression consistently associated with reduced overall survival with standard of care in patients with pancreatic, esophagogastric and lung tumors[1]

[1] Samarendra et al 2017. *Br J Cancer* 117, 124–135

Solid Tumors: Potential CXCL12-Driven Tumor Indication in Pancreatic Cancer

2019 ASCO Gastrointestinal Cancers Symposium

January 18, 2019 | San Francisco, CA

Poster Session B: Cancers of the Pancreas, Small Bowel and Hepatobiliary Tract

Abstract 275: Patient reported abdominal pain as a surrogate of the clinical benefit of tipifarnib in pancreatic cancer patients

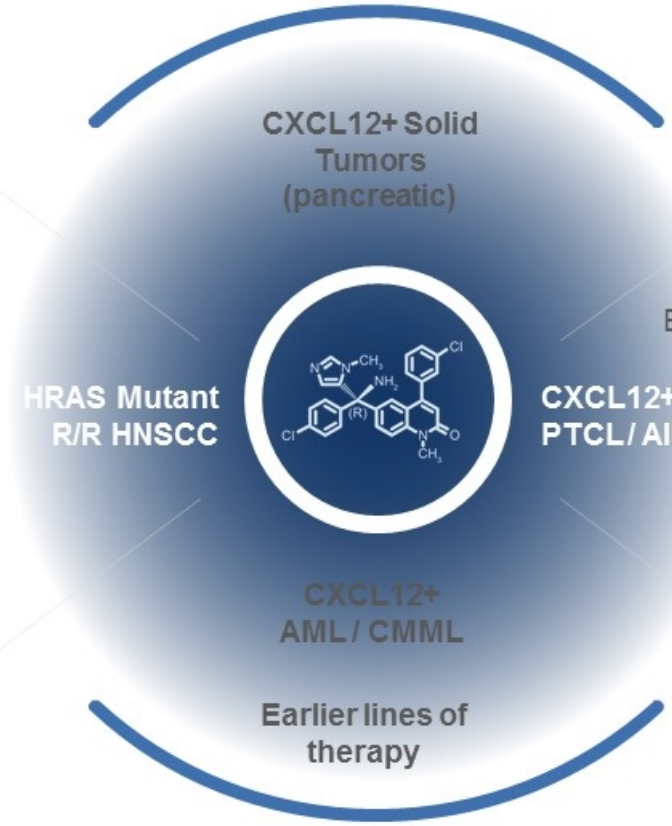
Authors: Antonio Gualberto, Catherine Scholz, Eric Van Cutsem

Demir et al. 2017. Early pancreatic cancer lesions suppress pain through CXCL12-mediated chemoattraction of Schwann cells. PNAS 114:E85-E94

Cornerstone Proof-of-Concepts Support Expansion to Additional Indications

Tipifarnib

Farnesyl transferase (FT) inhibitor



Other HRAS Mutant SCCs

Earlier lines of therapy

Other HRAS Mutant Solid Tumors

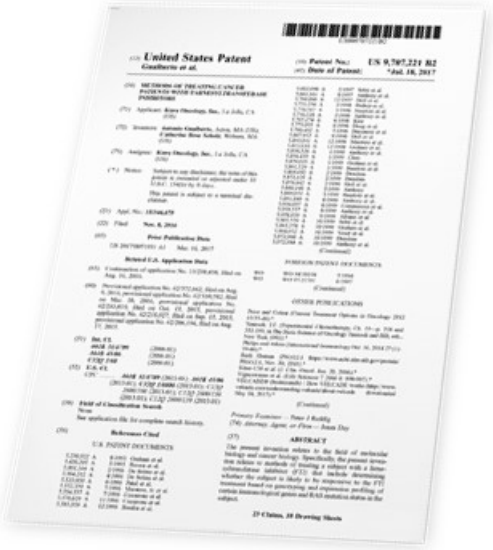
Other CXCL12+ R/R B-cell Lymphomas

Earlier lines of therapy

CXCL12+ R/R CTCL, HL

Biomarker Strategies Enlarge Patent Estate for Therapeutic Uses of Tipifarnib

- U.S. patent 9,707,221 issued in July 2017 provides exclusivity for tipifarnib in **HRAS mutant HNSCC indication to 2036**
- Corresponding patents beginning to issue in foreign countries
- U.S. patent 9,956,215 issued in May 2018 provides exclusivity for tipifarnib in certain **CXCL12-expressing cancers to 2037**
- U.S. patent 10,137,121 issued in November 2018 provided exclusivity for tipifarnib **in AITL to 2037**
- Additional patent applications pending in the U.S. and foreign countries for tipifarnib in other biomarkers and disease indications
- Patents illustrate potential of broader strategy to generate intellectual property related to use of drug candidates in biomarker-defined populations



03 • KO-947 (ERK Inhibitor)

01 • Tipifarnib in HRAS Mutant Solid Tumors

02 • Tipifarnib Using CXCL12 Pathway Biomarkers

04 • KO-539 (Menin-MLL Inhibitor)

KO-947: Potent Inhibitor of ERK1/2

- **Summary**
  - Potent, selective small molecule inhibitor of ERK1/2
  - Demonstrates prolonged pathway modulation in preclinical tumor models
  - Multiple tumors, including SCCs and KRAS mutant adenocarcinomas, identified as sensitive to KO-947 as monotherapy in preclinical models
  - Mechanism-based and SOC combinations under evaluation

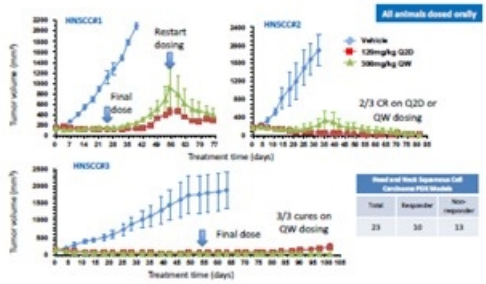
KO-947

ERK inhibitor

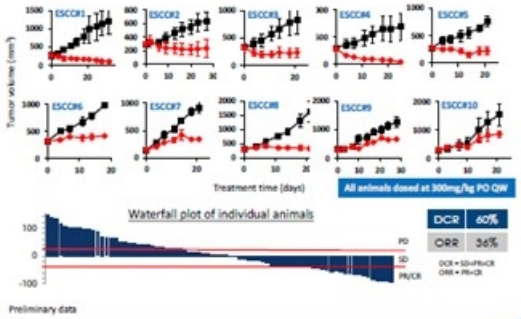
- **Clinical Development and Status**
  - Unique pharmacology enables intermittent dosing schedules
  - Potential biomarkers, including 11q13 amplifications in SCCs, for sensitive subsets have been identified
  - Phase 1 dose escalation trial ongoing
  - Initial Phase 1 clinical data anticipated in 2019

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

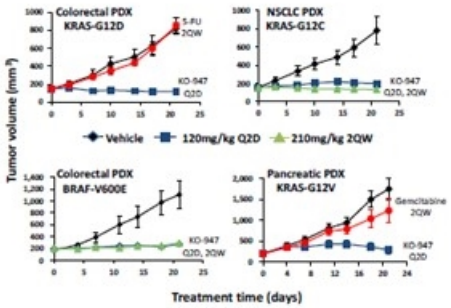
KO-947 induces complete responses and regressions of large tumors in head and neck squamous cell carcinoma



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



KO-947 is active in a subset of KRAS- or BRAF-mutant colon, lung and pancreatic PDX models



Burrows *et al*. AACR 2017 #5168/11

04 • KO-539 (Menin-MLL Inhibitor)

01 • Tipifarnib in HRAS Mutant Solid Tumors

02 • Tipifarnib Using CXCL12 Pathway Biomarkers

03 • KO-947 (ERK Inhibitor)

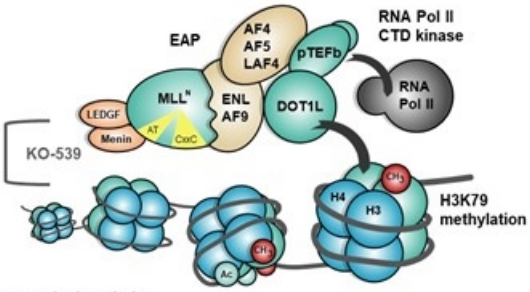
KO-539: Potent Inhibitor of Menin-MLL Interaction

- **Summary**
  - Potent, selective small molecule inhibitor of the menin-MLL interaction
  - Robust antitumor activity observed in mixed lineage leukemias rearranged (MLL-r) as well as disseminated NPM1mut and DNMT3Amut AML PDX models
  - Preliminary data suggests anti-leukemic activity by induction of myeloid differentiation in AML blasts
  - **Menin-MLL inhibitors have the potential to treat approximately 50% of acute leukemias**

KO-539

Menin-MLL inhibitor

- **Status**
  - IND submission anticipated in Q1 2019
  - Initiation of Phase 1 study anticipated in Q2 2019

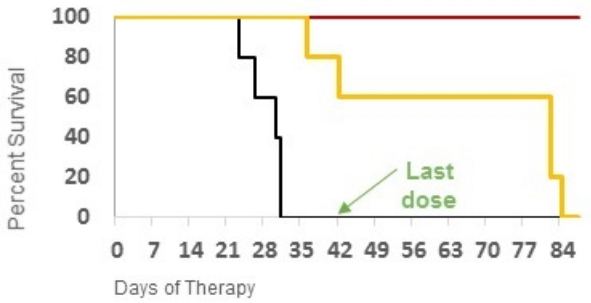


The menin-MLL complex appears to be a central node in epigenetic dysregulation driven by several distinct oncogenic driver mutations important in diverse leukemias and myeloproliferative disorders

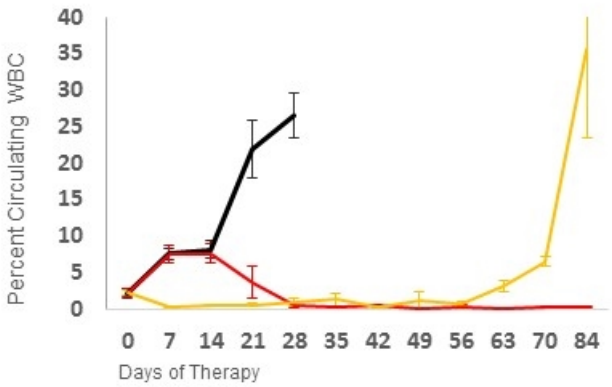
KO-539 Produces Lasting Complete Remissions in a NPM1/DNMT3A/IDH2/FLT3-Mutant AML Model

AM7577 Model

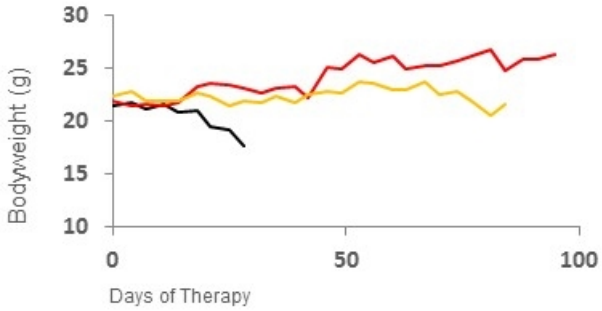
Overall survival



CD45+ human AML blasts



Tolerability



Vehicle

KO-539 100 mg/kg QD

Quizartinib 3 mg/kg QD

Burrows *et al*. AACR-NCI-EORTC 2017 LB-A27

Anticipated Milestones & Financial Highlights

Program		Milestones	Status
Tipifarnib Farnesyl Transferase Inhibitor	HRAS Mutant Indications	Initiation of registration-directed trial in HNSCC	✓
		Additional data from Phase 2 trial (KO-TIP-001)	2019
	CXCL12 Pathway Indications	Patents for tipifarnib in AITL and CXCL12^{+} PTCL & AML	✓
		Proof-of-concept in AITL	✓
		Data from retrospective study in pancreatic cancer	Q1 2019
		Data update in CXCL12+ hematologic malignancies	2019
KO-947 ERK Inhibitor		Potential biomarker of activity in squamous cell carcinomas	✓
		Data from Phase 1 dose-escalation trial	2019
KO-539 Menin-MLL Inhibitor		Anti-tumor activity in preclinical models of AML	✓
		Submission of IND application	Q1 2019
		Initiation of Phase 1 trial	Q2 2019

Financial Highlights	
	Nasdaq: KURA
	Shares outstanding: 38.0M basic, 3.3M options*
	Cash, cash equivalents and short-term investments: $187.4M*

* As of 3Q 2018 10-Q

KURA ONCOLOGY

Developing Precision Medicines for the Treatment of Cancer

Corporate Presentation

January 2019