KURA ONCOLOGY

Corporate Presentation

November 2019

Developing Precision Medicines for the Treatment of Cancer

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	Advancing pipeline of targeted drug candidates for selected solid tumors and hematologic malignancies Utilizing precision medicine approach; Fast-to-market potential
Proprietary Pipeline	**Tipifarnib**: Potent farnesyl transferase inhibitor • Clinical proof of concept achieved in four indications • Registration-directed AIM-HN study and multiple Phase 2 trials ongoing • Expanding patent portfolio and potential for regulatory exclusivity **KO-947**: ERK inhibitor; Phase 1 dose-escalation trial ongoing **KO-539**: Inhibitor of menin-MLL interaction; First patient dosed in September 2019
Near-Term Milestones	Multiple upcoming development milestones across the pipeline
Financials	$250.1 million in cash as of September 30, 2019*

* 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 heme 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 AIM-HN trial • Multiple Phase 2 trials ongoing	• Ongoing Phase 1 dose-escalation trial	• Phase 1 trial initiated in September 2019

Kura Leadership Team and Board of Directors

Leadership Team

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

Antonio Gualberto, M.D., Ph.D.
Head of Development and Chief Medical Officer

Marc Grasso, M.D.
Chief Financial Officer and Chief Business Officer

Kathleen Ford
Chief Operating Officer

James Basta, J.D.
Chief Legal Officer

Board of Directors

Faheem Hasnain

Robert Hoffman

Thomas Malley

Diane Parks

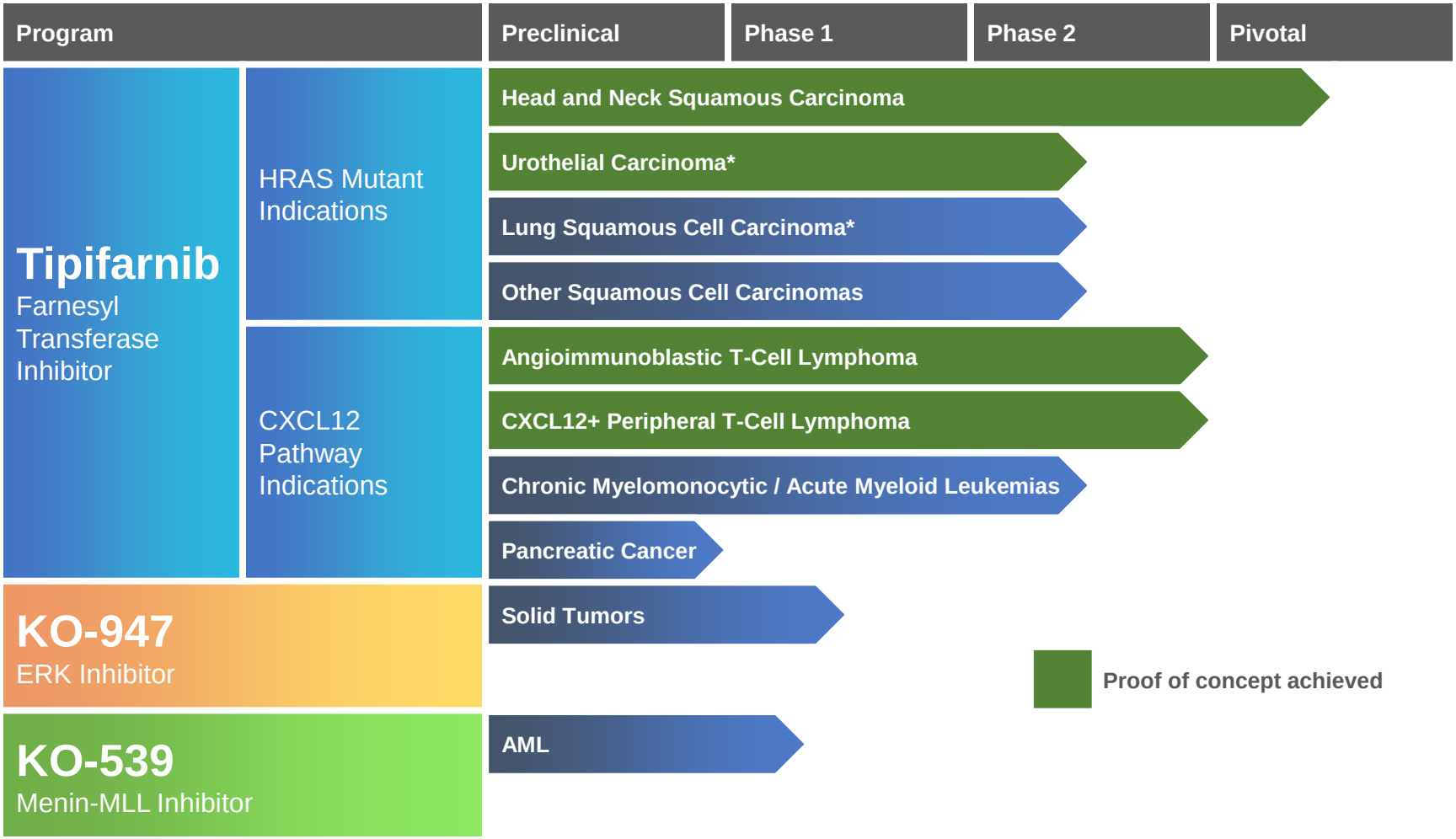
Steven Stein, M.D.

Mary Szela

Troy Wilson, Ph.D., J.D.

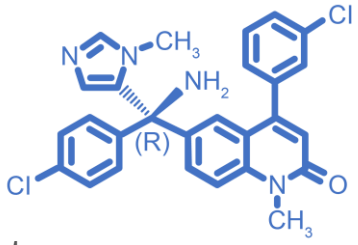
Proven oncology drug discovery, development and commercialization expertise

Product Candidate Pipeline



* Investigator-sponsored trials

Tipifarnib: A First-in-Class Farnesyl Transferase Inhibitor for the Treatment of Cancer



- Extremely potent and selective inhibitor of farnesyl transferase[1] in-licensed from Janssen
- Well characterized with > 5,000 patients treated
- Durable responses were previously reported in selected study patients, but appropriate genetic biomarkers were not available to elucidate the mechanism of action
- Manageable safety profile observed as single-agent therapy (< 25% treatment discontinuation)
- Tipifarnib adverse events (reported from 472 solid tumor patients):
  - Myelosuppression (neutropenia 25%, anemia 31%, thrombocytopenia 19%)
  - Non-heme > 25%: fatigue (41%) and GI unspecific (nausea 47%, anorexia 33%, diarrhea 32%, vomiting 32%)

Kura is executing on a biomarker strategy, including HRAS and CXCL12 pathway biomarkers, to unlock the potential of tipifarnib as a targeted therapeutic

[1] End *et al*. 2001. *Cancer Res*. 61:131-37

01 • *Tipifarnib in HRAS Mutant Solid Tumors*

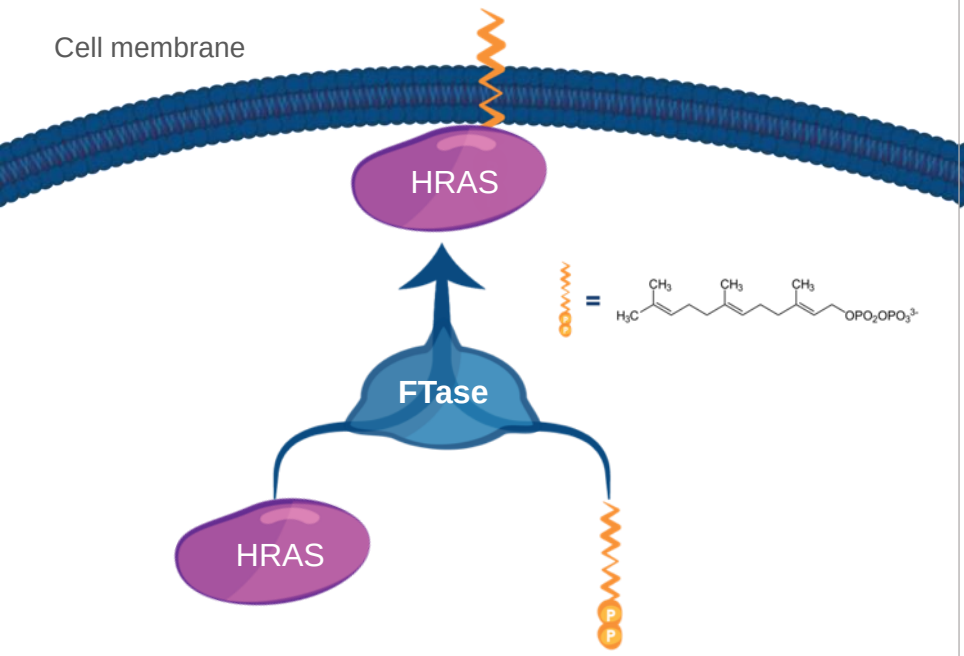
02 *Tipifarnib Using CXCL12 Pathway Biomarkers*

03 *KO-947 (ERK Inhibitor)*

04 *KO-539 (Menin-MLL Inhibitor)*

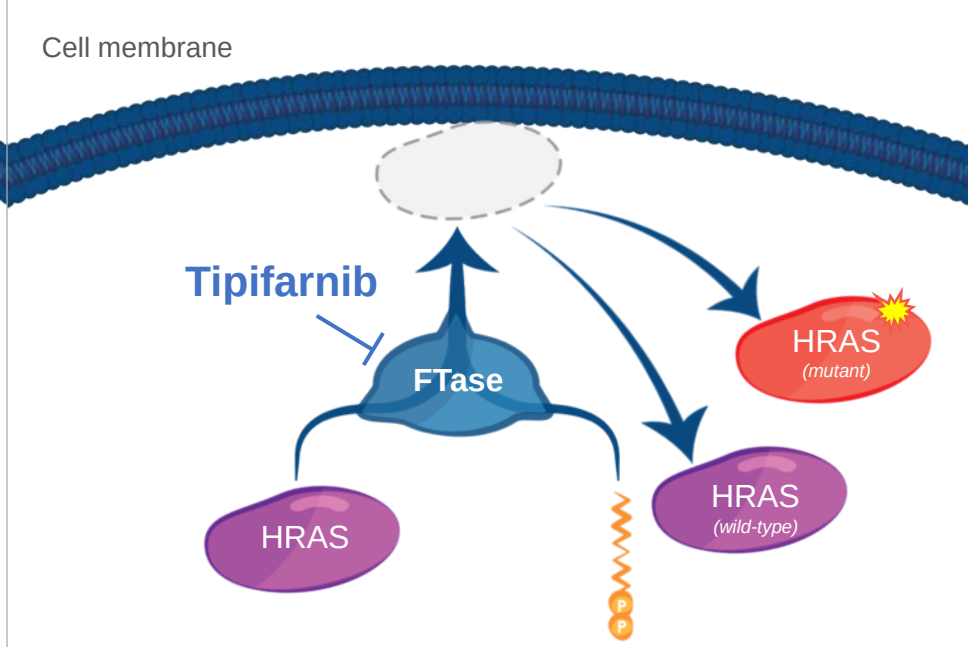
Tipifarnib Inhibits Farnesylation – An Essential Modification Required for HRAS Activity

FTase Required for Pathway Signaling



- **Farnesyl transferase (FTase) attaches farnesyl group,** or lipid side chain, that facilitates localization to the inner cell membrane
- Membrane localization is **required for HRAS signal transduction activity**

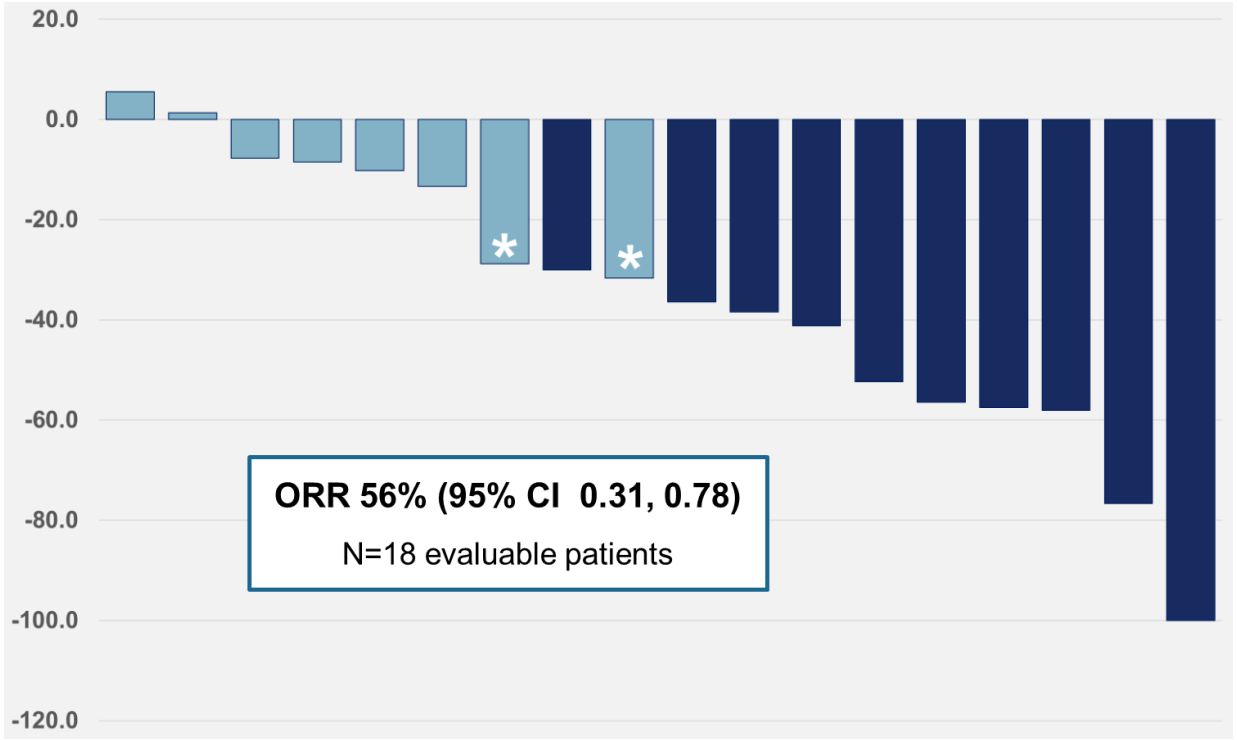
Tipifarnib Inhibits FTase



- Blocking farnesylation **prevents wild-type and mutant HRAS membrane localization**
- While NRAS and KRAS can be anchored to the membrane via an alternative pathway, **HRAS is solely dependent on FTase**

Proof of Concept in HRAS Mutant Head and Neck Squamous Cell Carcinomas (HNSCC)

Maximum Change in Tumor Burden



RUN-HN
KO-TIP-001

PR SD Not yet efficacy evaluable Not efficacy evaluable

* unconfirmed PR (uPR) ◇ 600 mg starting dose

Ho *et al*. AACR-NCI-EORTC International Conference on Molecular Targets and Cancer Therapeutics #384 | Preliminary data as of 10/17/19

Efficacy-evaluable patients with HRAS mutant variant allele frequency (VAF) ≥ 20% and serum albumin ≥ 3.5 g/dL, or HRAS VAF ≥ 35%

One patient treated off-protocol through compassionate use

Tumor measurements not available from three patients (not evaluable/not yet efficacy evaluable)

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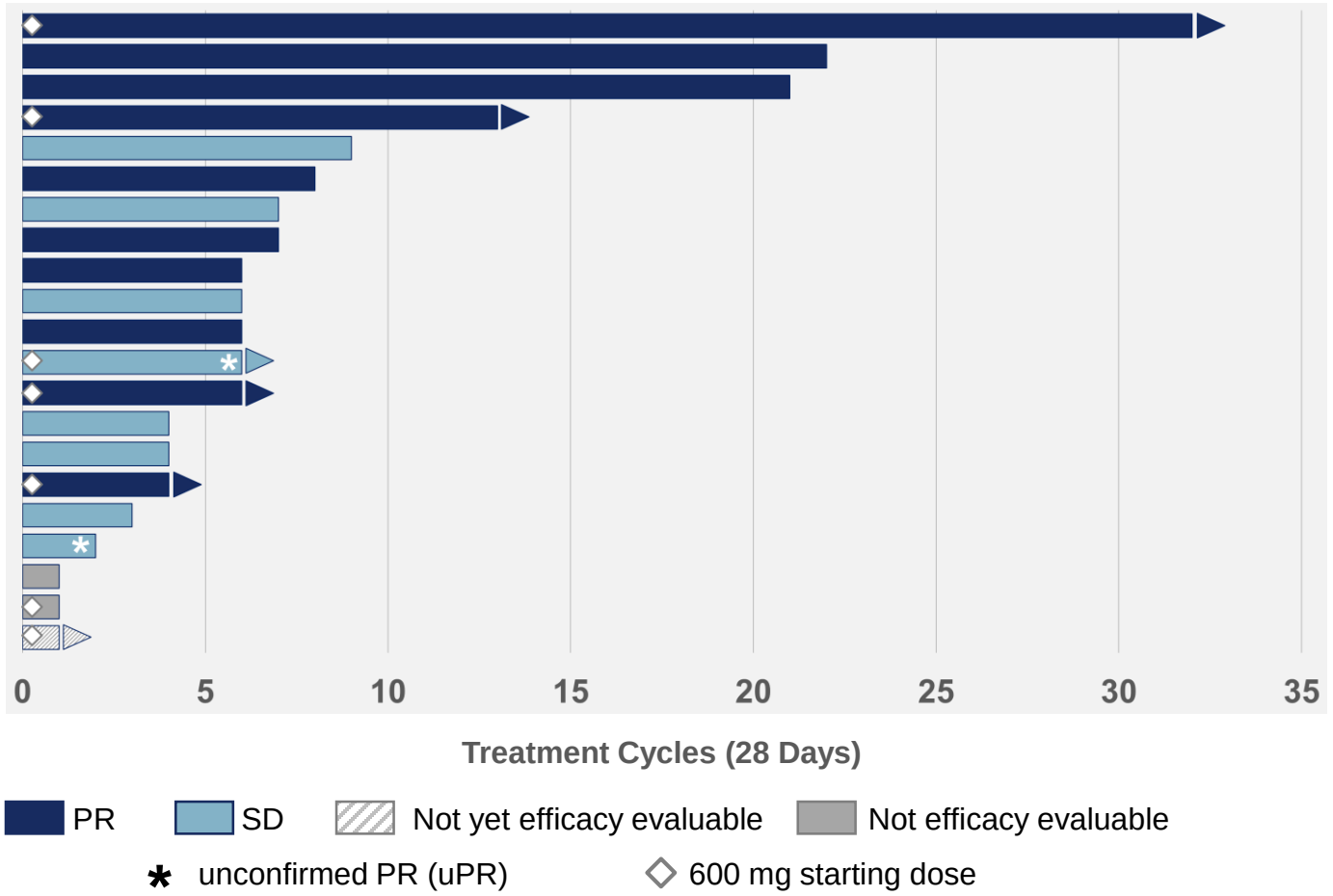
Durable Anti-Tumor Activity as a Single Agent in HRAS Mutant HNSCC

Time on Treatment



RUN-HN
KO-TIP-001

Ho *et al*. AACR-NCI-EORTC International Conference on Molecular Targets and Cancer Therapeutics #384 | Preliminary data as of 10/17/19
Efficacy-evaluable patients with HRAS mutant variant allele frequency (VAF) ≥ 20% and serum albumin ≥ 3.5 g/dL, or HRAS VAF ≥ 35%
One patient treated off-protocol through compassionate use

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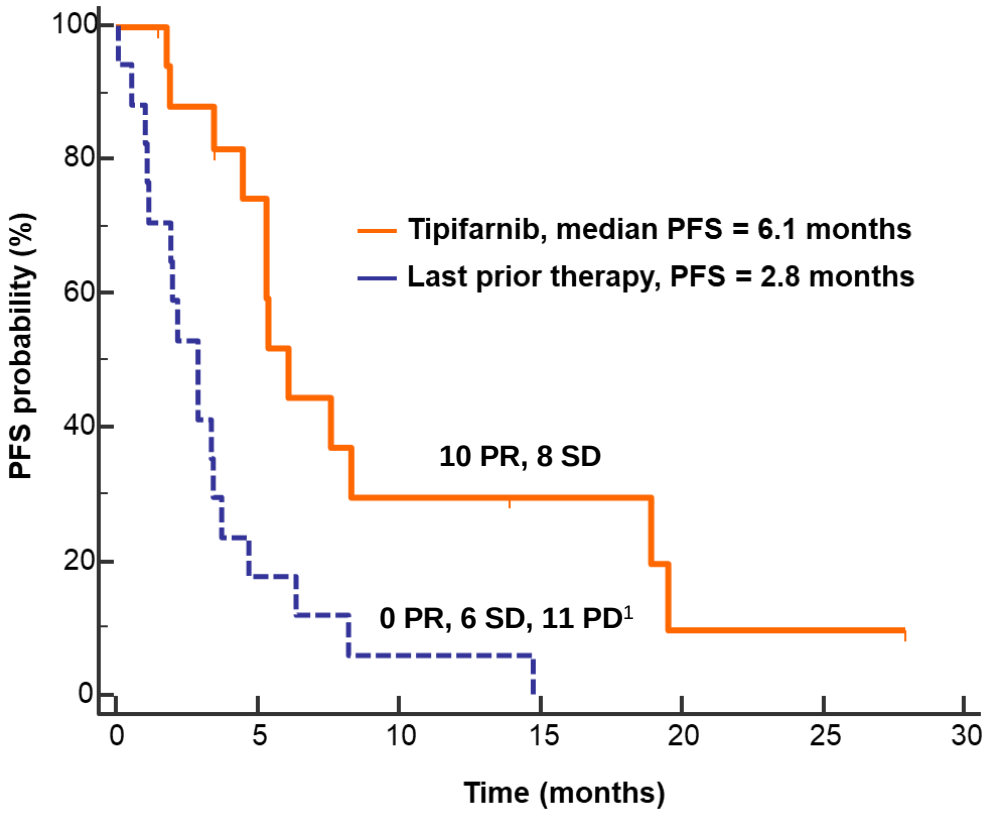
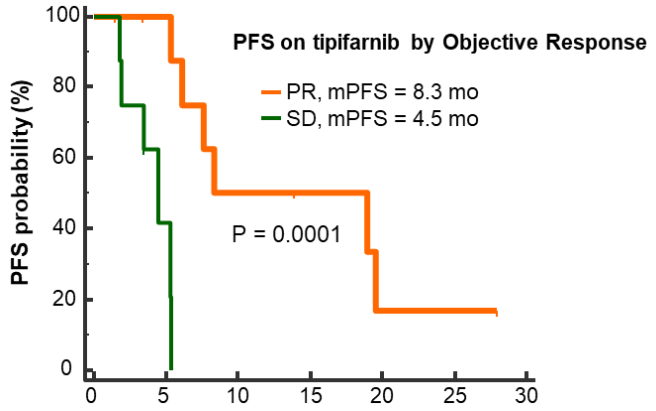
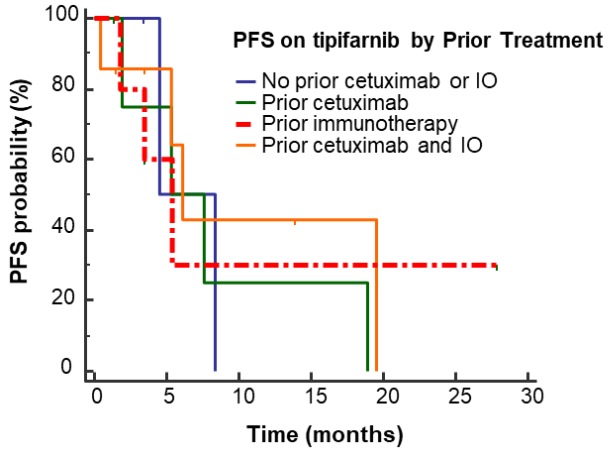
Tipifarnib Appears Better than Standard of Care for Treatment of HRAS Mutant HNSCC







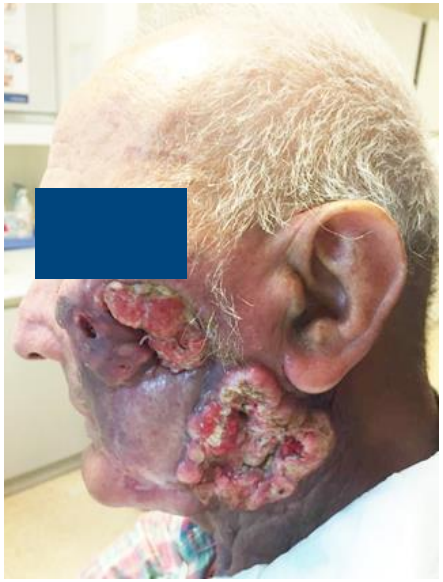
Ho *et al*. AACR-NCI-EORTC International Conference on Molecular Targets and Cancer Therapeutics #384 | Preliminary data as of 10/17/19

Efficacy-evaluable patients with HRAS mutant variant allele frequency (VAF) ≥ 20% and serum albumin ≥ 3.5 g/dL, or HRAS VAF ≥ 35%

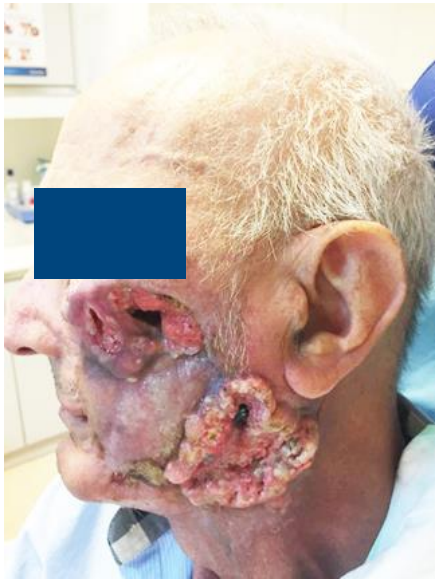
One patient treated off-protocol through compassionate use | [1] One patient did not receive prior systemic therapy

Resolution of Disfiguring Skin Lesions with Tipifarnib Post-Immunotherapy Failure

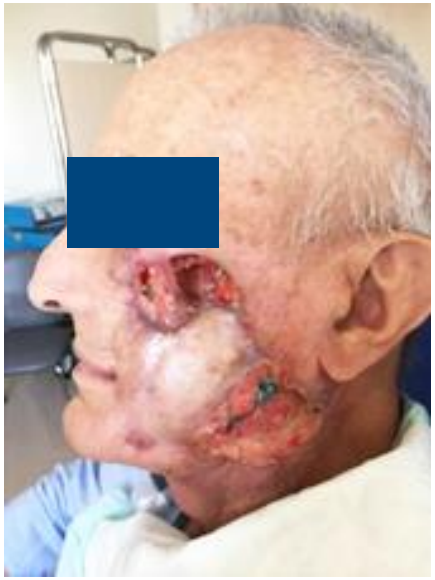
- Patient 012-001: 69-year-old male with recurrent oral cavity SCC
- Prior therapies: TPEx (docetaxel CDDP cetuximab), nivolumab + lirilumab
- Molecular status: HRAS G12S, TP53 R248Q
- 27.5% HRAS mutant allele frequency
- Initial PR (40% tumor reduction) on Cycle 1 Day 15 (7 days tipifarnib + 7 days rest; 56% reduction at Cycle 3



Cycle 1 Day 1



Cycle 1 Day 7



Cycle 1 Day 20

Cycle 2 Day 1

Ho *et al*. 2018 Multidisciplinary Head and Neck Cancers Symposium #217 | Results based on preliminary data as of 2/8/18
Images provided by Dr. Caroline Even and Dr. Charles Ferte, IGR, Paris

Registration Strategy in HRAS Mutant HNSCC

AIM-HN: Registration-directed trial of tipifarnib in HRAS mutant HNSCC

- Recurrent or metastatic patients after one prior line of platinum therapy
- At least 59 evaluable patients, 15% ORR (null hypothesis)
- Trial initiated in November 2018; Approximately two years to fully enroll
- Now open in 75+ clinical sites in the U.S., Europe and Asia
- Intended to support an NDA seeking accelerated approval*

AIM-HN
KO-TIP-007

SEQ-HN: Non-interventional screening and outcomes cohort

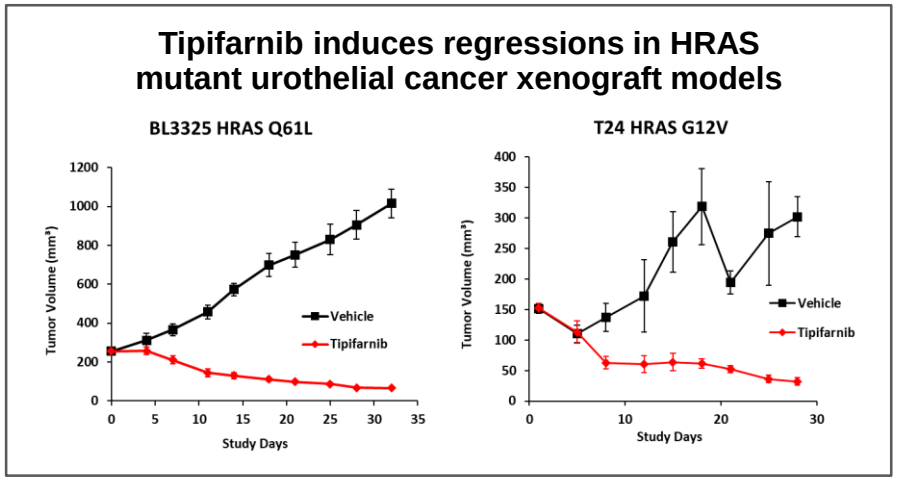
- Matched case-control study designed to:
  - Characterize natural history of HRAS mutant HNSCC patients
  - Enable identification of patients for potential enrollment into AIM-HN
- May support potential FDA labelling discussions, post-approval commitments and commercial considerations

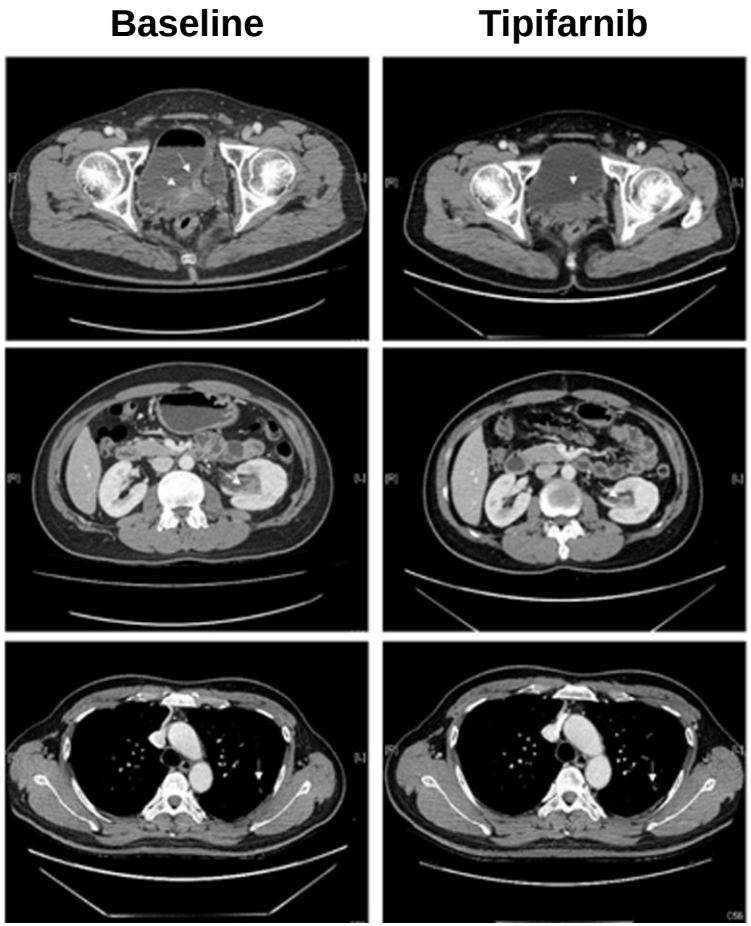
SEQ-HN
KO-TIP-007

* Feedback from end-of-Phase 2 meeting with FDA

Proof of Concept Achieved in HRAS Mutant Urothelial Carcinomas

- Confirmed objective responses achieved in 5 of 13 evaluable patients (38% ORR)
- Primary endpoint met prior to completion of enrollment with 4 patients experiencing PFS > 6 mo.
- Phase 2 proof-of-concept trial sponsored by Samsung Medical Center in Seoul, Korea
- Full data to be presented at future medical meeting





Tumor scan Images were provided by Dr. Se Hoon Park, Samsung Medical Center | Tumor patient-derived xenograft data are proprietary

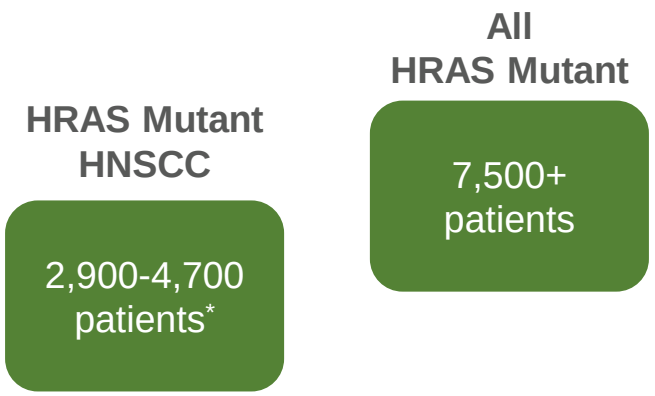
HRAS Mutant Cancers: Market Opportunity

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)

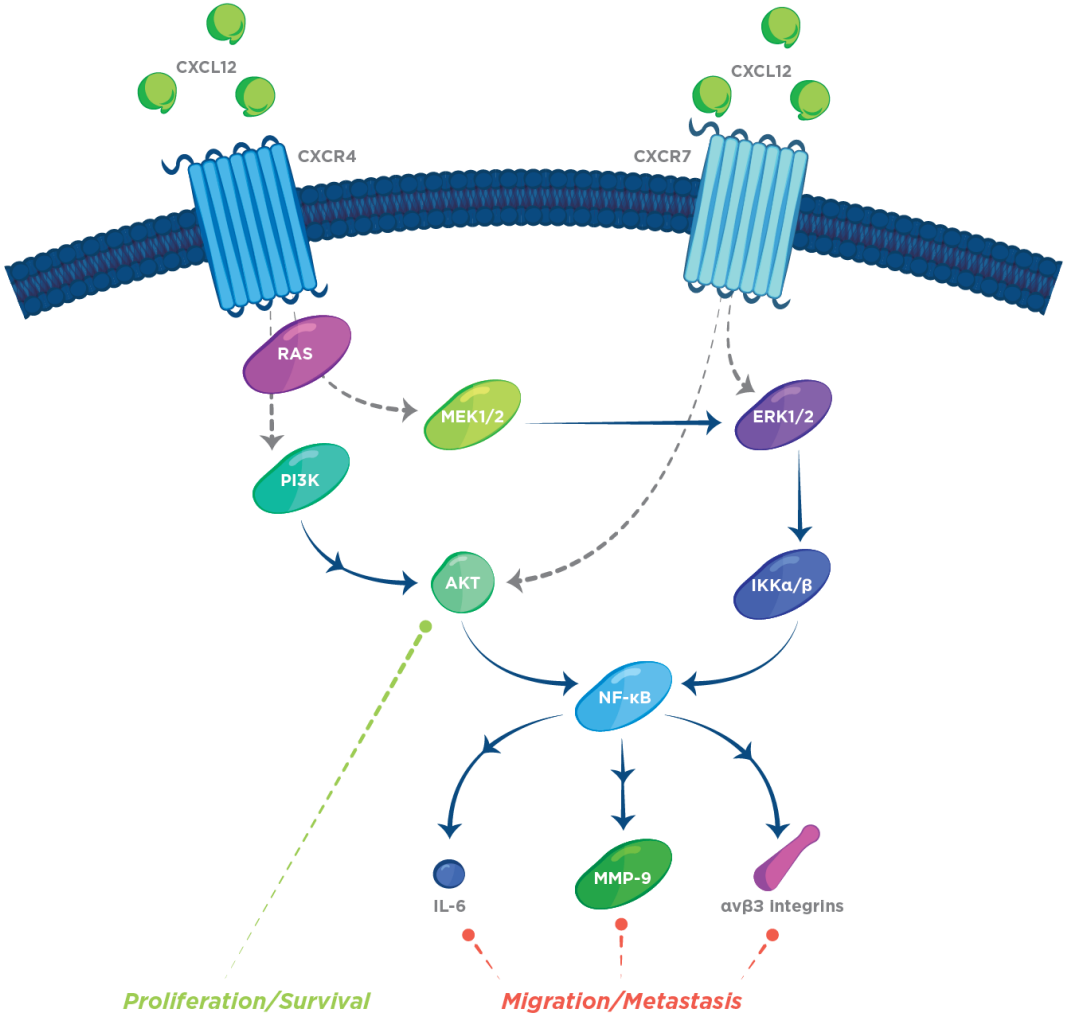
01 *Tipifarnib in HRAS Mutant Solid Tumors*

02. *Tipifarnib Using CXCL12 Pathway Biomarkers*

03 *KO-947 (ERK Inhibitor)*

04 *KO-539 (Menin-MLL Inhibitor)*

CXCL12 and CXCR4/7 Signaling Drive Multiple Downstream Pathways Critical to Cancer

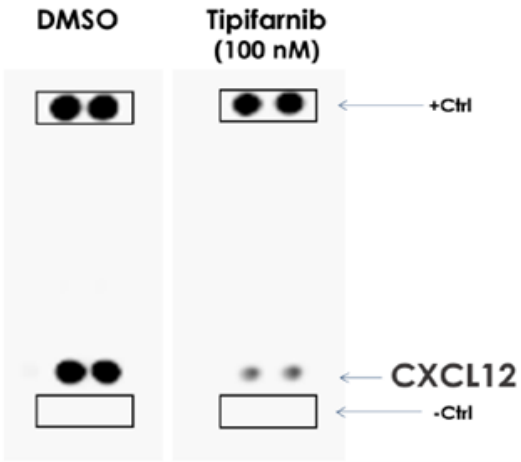


Key Features:

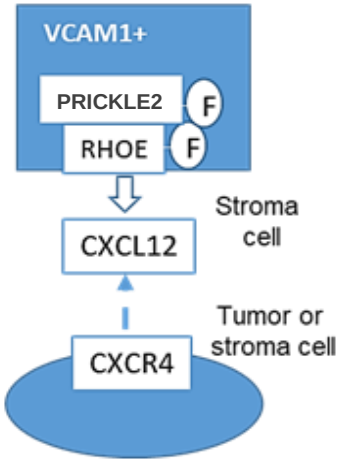
- CXCL12 is expressed primarily by immune cells, endothelial cells and stromal fibroblasts
- CXCL12 and its receptors (CXCR4, CXCR7) are key factors linking cancer cells with the tumor microenvironment
- Pathway activation (high CXCL12; receptor activation) can drive cancer phenotype
- Trend for worse prognosis across multiple tumors with high CXCL12 expression

Int J Mol Med. 2013 Dec;32(6):1239-46

Tipifarnib is a CXCL12 Pathway Inhibitor



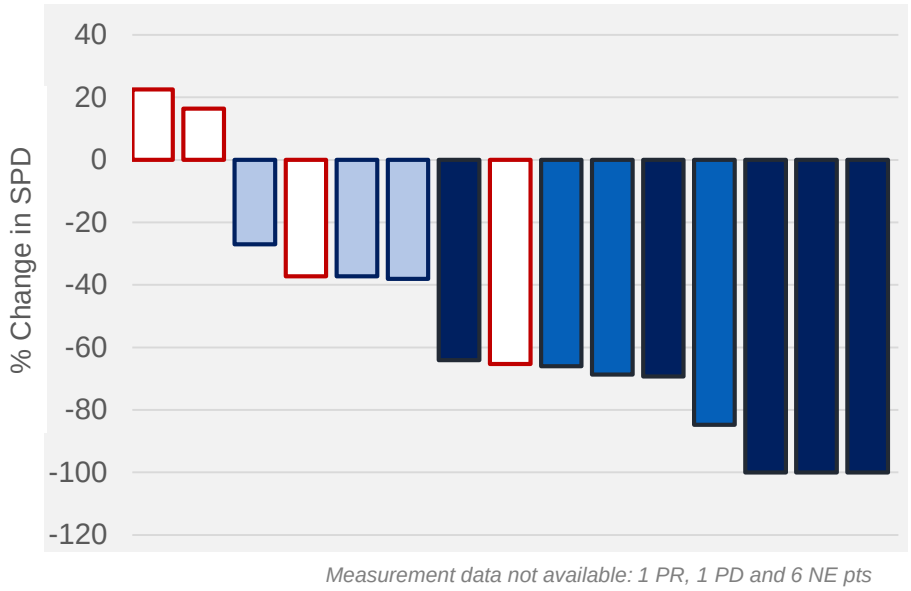
Tipifarnib downregulates CXCL12 secretion ex-vivo in CD1 mouse bone marrow stroma cultures



Gene expression of the uniquely farnesylated RHOE (RND3) and PRICKLE2 proteins is strongly associated with bone marrow stroma CXCL12 expression, suggesting potential CXCL12-related tipifarnib targets[1]

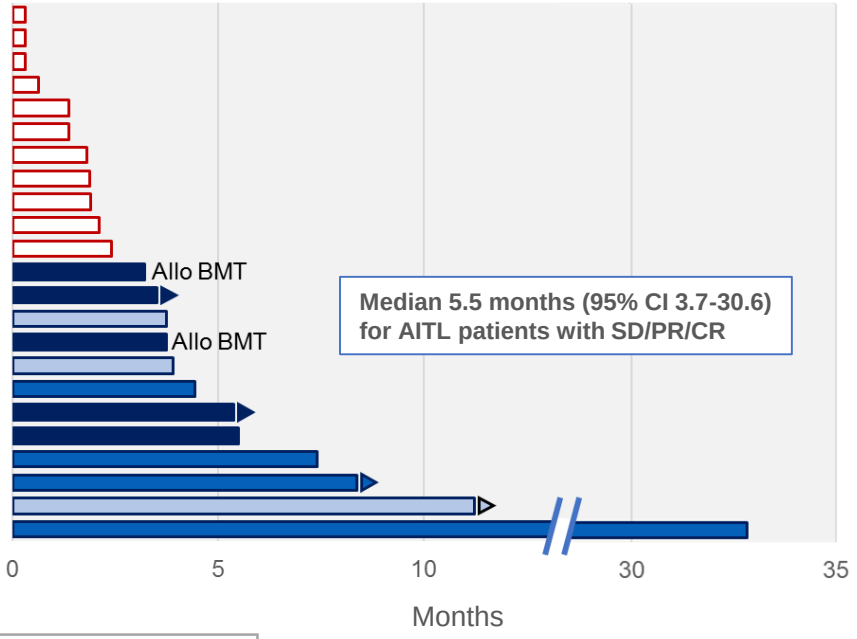
Proof-of-Concept in Angioimmunoblastic T-Cell Lymphoma (AITL)

Maximum Change in Tumor Burden



Measurement data not available: 1 PR, 1 PD and 6 NE pts

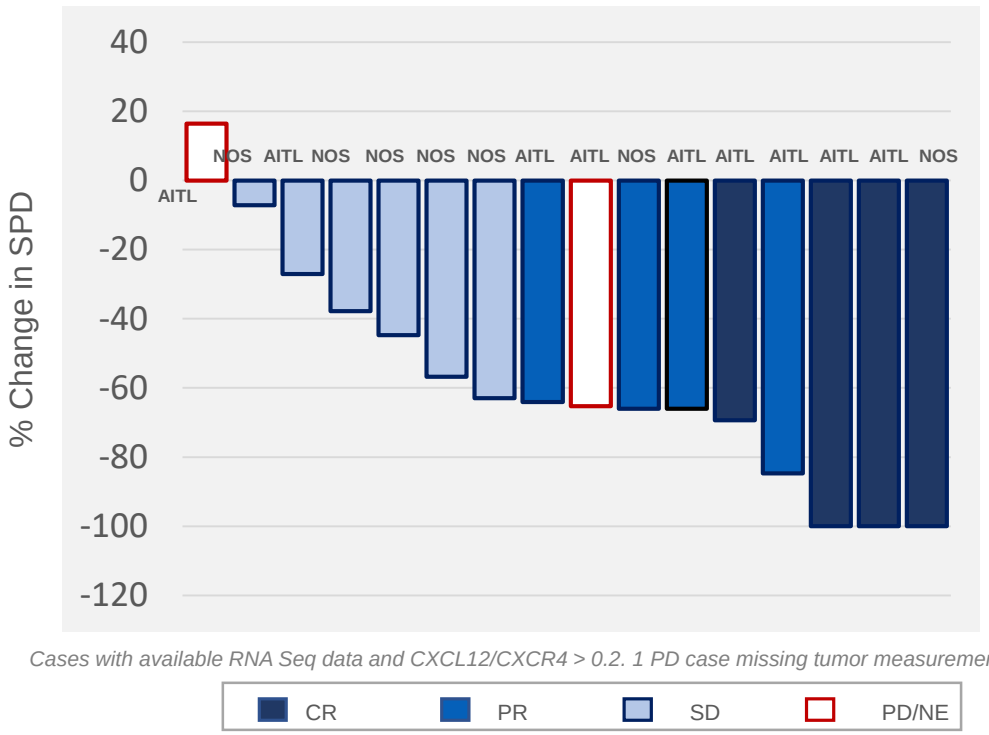
Time on Treatment



Preliminary data as of May 24, 2019

Tipifarnib Treatment Resulted in Durable Clinical Responses and Enabled Subsequent Transplant in Patients Achieving a Complete Response

Tipifarnib is Active in High CXCL12- Expressing AITL and PTCL NOS Tumors



Cases with available RNA Seq data and CXCL12/CXCR4 > 0.2. 1 PD case missing tumor measurements

sample	HISTOL	RESP	CXCL12/CXCR4
6	AITL	PR	7.42
28	AITL	CR	2.4
34	AITL	PD/NE	
36	NOS	SD	1.3
17	NOS	SD	0.85
32	AITL	PD/NE	
37	NOS	SD	0.6
1	AITL	CR	0.48
30	AITL	PR	0.4
7	AITL	PD/NE	
18	NOS	SD	0.40
12	NOS	CR	0.37
2	AITL	CR	0.36
3	AITL	PR	0.35
16	NOS	SD	0.30
31	NOS	PR	0.28
5	AITL	SD	0.28
8	AITL	PD/NE	0.19
23	NOS	PD/NE	0.18
40	NOS	PD/NE	0.17
19	NOS	PD/NE	0.16
4	AITL	PR	
26	ALCL	PD/NE	0.14
41	NOS	SD	0.1
10	AITL	PD/NE	0.12
11	AITL	PD/NE	0.12
20	NOS	PD/NE	0.10
25	NOS	PD/NE	0.08
33	AITL	SD	0.1
21	NOS	PD/NE	0.05
29	AITL	CR	
13	NOS	PD/NE	0.03
9	AITL	PD/NE	0.03
24	NOS	PD/NE	0.03

Tipifarnib targets CXCL12 and is active in tumors with high CXCL12 expression[1].

However, high CXCL12 could not explain all the activity/resistance to tipifarnib in AITL.

Molecular screenings were conducted to identify other drivers of the activity of tipifarnib in AITL.

Preliminary data as of May 24, 2019

High Activity of Tipifarnib in KIR3DL2 Mutant AITL Supports CXCL12 / CXCL5 Hypothesis

- CXCL12 and CXCL5, respectively, drive sensitivity and resistance to tipifarnib
- AITL expresses high levels of CXCL12 and is sensitive to tipifarnib
- AITL also expresses CXCL5; however, ~50% of AITL carry mutations of KIR3DL2, express low levels of CXCL5 and are highly sensitive to tipifarnib (50% CR rate)
- High Allele Frequency of KIR3DL2 mutation predicted complete response to tipifarnib treatment (ROC AUC=0.94, p<0.0001)
- AITL patients carrying KIR3DL2 mutations experienced a better outcome with tipifarnib treatment than with prior SOC treatment

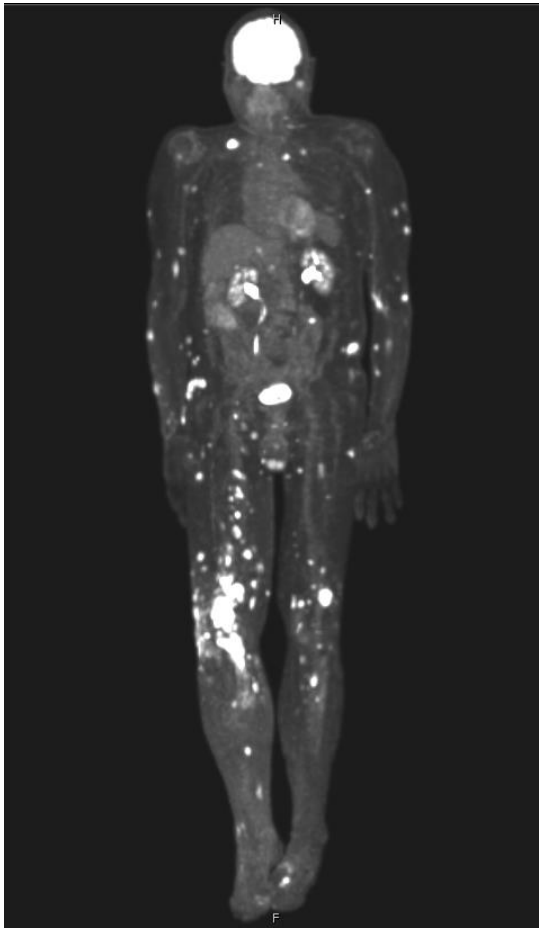
Best Response to Tipifarnib (N=16 AITL with sequenced tumors)

	KIR3DL2 Mutant	KIR3DL2 Wild Type
N	8	8
Overall Best Response		
Complete Response (CR)	4	-
Partial Response (PR)	2	2
Stable Disease (SD)	2	-
Progressive Disease (PD)	-	6
Not evaluable (NE)	-	-
Overall Response Rate (CR + PR)	**75%**	**25%**
95% CI	35.9 - 95.4	4.6 - 64.1
Clinical Benefit Rate (CR + PR + SD)	**100%**	**25%**
95% CI	64.1 - 100.0	4.6 - 64.1

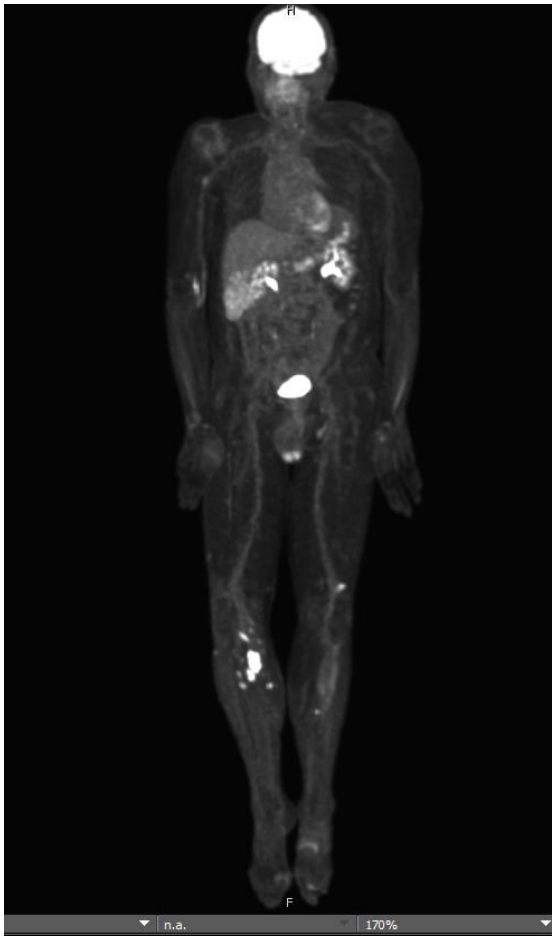
Preliminary data as of May 24, 2019

Witzig *et al*. EHA 2019 #S869 | KIR data analyses to be presented at 15-ICML: Gualberto et. al. Abstract 156-P

Tumor Reduction in PTCL-NOS Patient, wild type CXCL12 3'UTR



Baseline



End of Cycle 2

- 77 yo male with PTCL-NOS Stage IV
- CHOP x 5 with initial response then progression in skin
- At baseline visit had multiple skin nodules biopsy proven relapsed PTCL
- After two cycles of tipifarnib patient had near CR

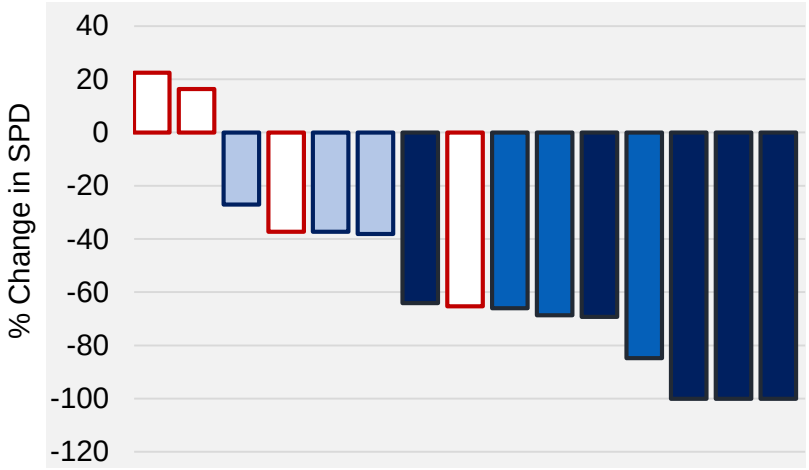
AITL/CXCL12+ PTCL Indications

CXCL12+ PTCL

AITL/PTCL NOS

Clinical benefit observed in
high CXCL12-expressing PTCL

Maximum Change in Tumor Burden (AITL Cohort)



- AITL and CXCL12+ cohorts met pre-specified statistical hypotheses with 45% and 42% ORR, respectively, supporting proof of concept for tipifarnib in PTCL
- Tipifarnib is active in AITL patients and in PTCL-NOS patients with wild type CXCL12 3'UTR
  - AITL: 53% ORR (all subjects, PPS)
  - PTCL-NOS with wild type CXCL12 3'UTR: 33% ORR (all subjects, PPS), 100% clinical benefit rate
- Robust tools for the selection/stratification of patients

PTCL Remains a High Unmet Need

	BELEODAQ® (belinostat)	ISTODAX® (romidepsin)	FOLOTYN® (pralatrexate)	ADCETRIS® (brentuximab)
Efficacy Study	Single Arm[1] N=120	Single Arm[2] N=130	Single Arm[3] N=109	Single Arm[4] N=58
Prior Therapies (range)	2 (1-8)	2 (1-8)	3 (1-12)	2 (1-6)
Overall Response Rate	**25.8%**	**26.2%**	**27%**	**86% (in ALCL)**
Median PFS/TTP	1.6 months	4.0 months	3.5 months	13.2 months
Median Overall Survival	7.9 months	11.3 months	14.5 months	Not reached
Dosing	IV infusion[5]	IV infusion[6]	IV push[7]	IV infusion[8]

- All the approved second line agents, with the exception of Adcetris, which targets ALCL patients only, have a limited efficacy with an average ORR of 26%
- Folotyn, Beleodaq and Istodax are not approved in EU

[1] Beleodaq® package insert
[2] Istodax® package insert
[3] Folotyn® package insert
[4] Adcetris® package insert
[5] 1,000 mg/m^2 administered over 30 mins by IV infusion once daily on days 1-5 of a 21-day cycle
[6] 14 mg/m^2 administered over a 4-hour period by IV on days 1, 8 and 15 of a 28-day cycle
[7] 30 mg/m^2 administered over 3-5 mins as an IV push once weekly for 6 weeks in 7-week cycles
[8] 1.8 mg/kg administered intravenously over 30 minutes every 3 weeks for up to 16 cycles

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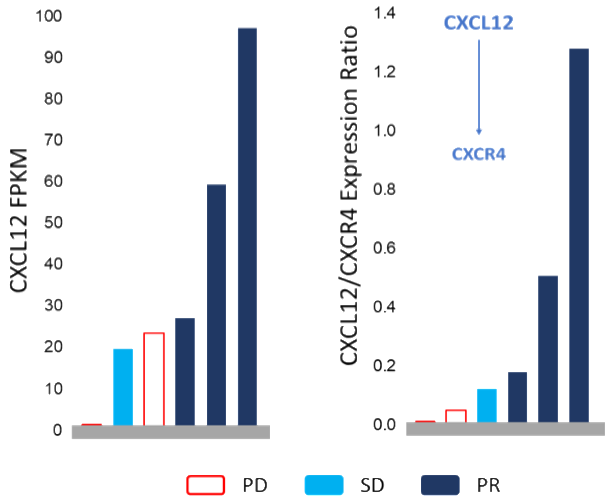
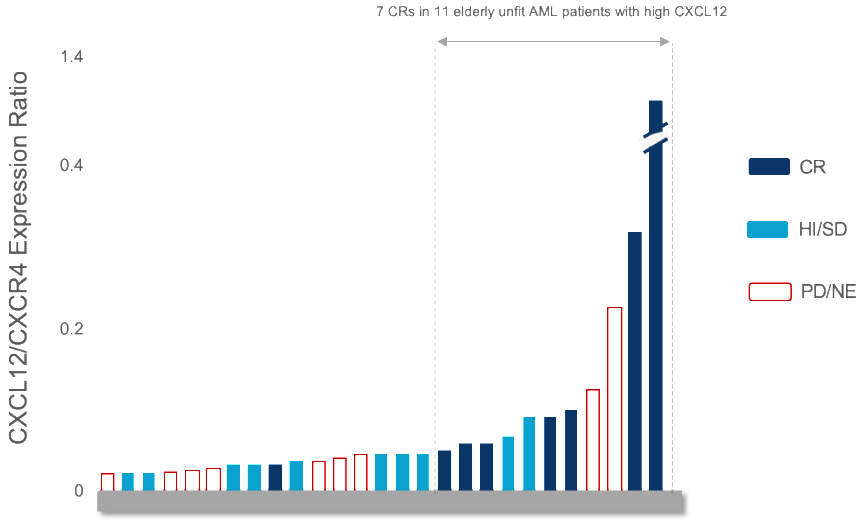
Prior Data Supports Expansion of CXCL12 Pathway Inhibition Beyond AITL/PTCL-NOS[1]

Objective Responses in Tipifarnib-treated DLBCL Patients with High Tumor CXCL12 Expression



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

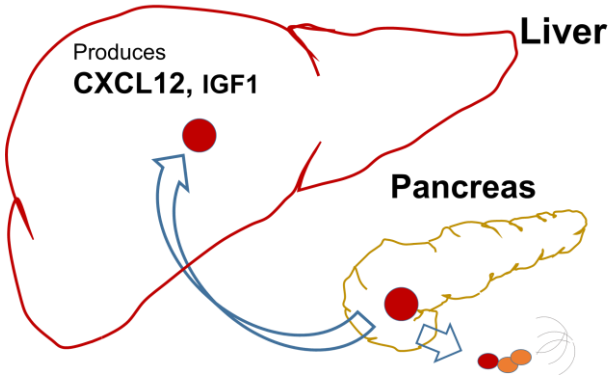


- Data from prospective AITL//PTCL-NOS trial and retrospective analysis from previous Janssen studies in DLBCL and AML suggests inhibition of CXCL12 pathway may drive clinical benefit across different hematologic malignancies
- Additional non-clinical studies planned and in progress to further validate association between CXCL12 pathway biomarkers and potential for clinical activity

[1] Gualberto *et al*. AACR 2019 #CT191

Inhibition of CXCL12 May Drive Clinical Benefit in Pancreatic Cancer

Disease Model for CXCL12 Expressing Sites



Rationale:

- CXCL12/IGF1 is known to induce tumor homing to liver and lymph nodes[1]
- Retrospective analysis of prior Janssen pancreatic cancer study (INT-11) suggests advantage in patient subsets with high CXCL12
- Potential to enrich for clinical benefit:
  - High CXCL12 expression in pancreatic tumors
  - ≤ 5% KRAS mutant allele frequency

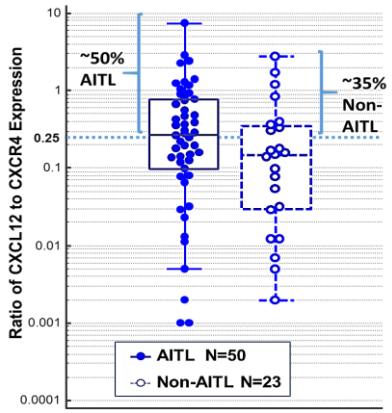
Next Steps:

- Additional non-clinical work and input from key opinion leaders and investigators
- Initiation of a proof-of-concept study in pancreatic cancer in the first half of 2020

Gualberto *et al*. ASCO GI 2019 #275 | [1] Akekawatchai *et al*. 2005 *J Biol Chem*. Dec 2;280(48):39701-8 |

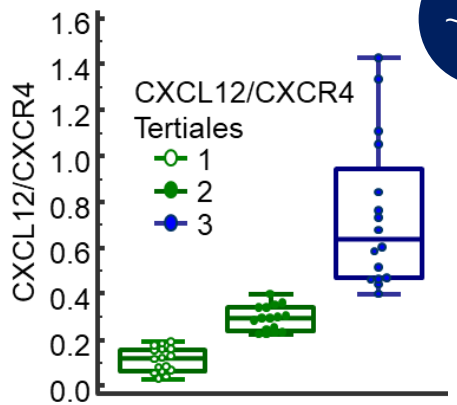
Tipifarnib Has Potential to Expand to Additional CXCL12-High Populations

PTCL / AITL[1] ~40%



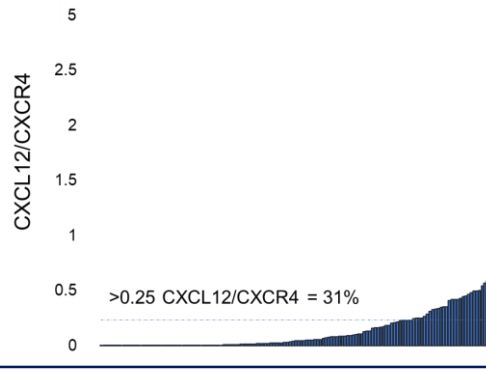
Est. Annual U.S. Incidence: 3,950[5]

DLBCL[2] ~33%



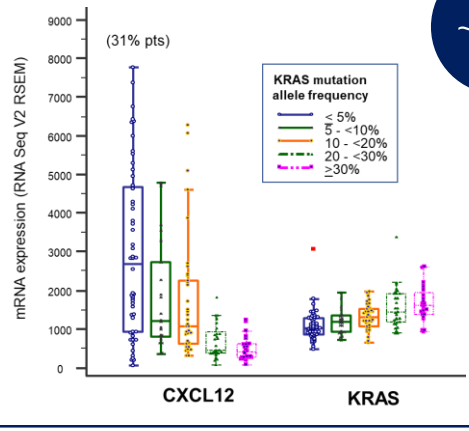
Est. Annual U.S. Incidence: 27,650[5]

AML[3] ~30%



Est. Annual U.S. Incidence: 21,450[6]

PANCREATIC[4] ~30%

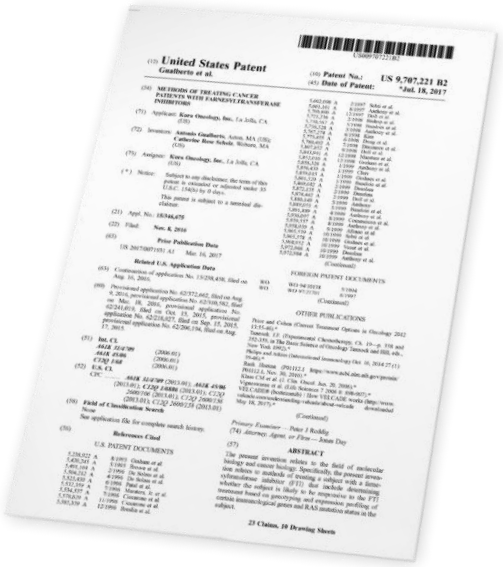


Est. Annual U.S. Incidence: 56,770[6]

[1] Witzig ASH 2018 #2937 | [2] Kura Oncology ASH 2018 Data Review | [3] Gualberto ASH 2017 #3957 | [4] Gualberto AACR 2019 #CT191 |
[5] Teras *et al*. 2016 *CA Cancer J Clin*. Nov 12;66(6):443-459 | [6] American Cancer Society | Incidence not adjusted for CXCL12-high subset

Multiple Issued Patents Provide Patent Exclusivity in U.S. and Foreign Countries

- Multiple issued U.S. patents covering biomarker-guided indications, providing patent exclusivity to 2036 and beyond
  - HRAS mutant HNSCC
  - HRAS mutant NSCLC
  - CXCL12-expressing cancers
  - Angioimmunoblastic T-cell lymphomas
- U.S. patents issued covering ”any farnesyl transferase inhibitor” for treatment of HRAS mutant HNSCC and CXCL12-expressing PTCL and AML
- First European patent granted for HRAS mutant HNSCC
- 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


United States Patent
Gaulberts et al.
Patent No.: US 9,707,221 B2
Date of Patent: *Jul. 18, 2017
METHODS OF TREATING CANCER PATIENTS WITH FARNESYLTRANSFERASE INHIBITORS
23 Claims, 19 Drawing Sheets

01 *Tipifarnib in HRAS Mutant Solid Tumors*

02 *Tipifarnib Using CXCL12 Pathway Biomarkers*

03 • *KO-947 (ERK Inhibitor)*

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

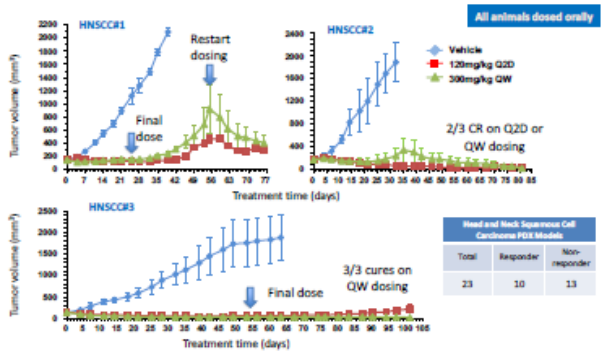
ERK inhibitor

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
- Goal: Reach recommended Phase 2 dose / maximum tolerated dose by end of 2019 or early 2020

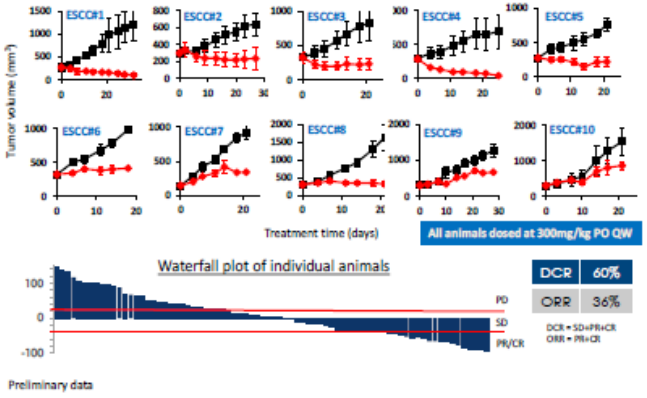
KO-947 Demonstrates Robust Single-Agent Activity in Preclinical Studies

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

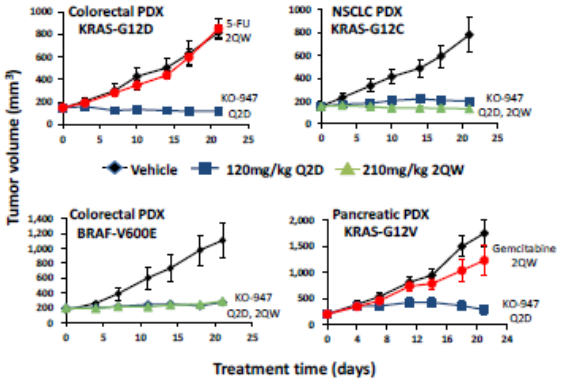


Head and Neck Squamous Cell Carcinoma PDX Models		
Total	Responder	Non-responder
23	10	13

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



01 *Tipifarnib in HRAS Mutant Solid Tumors*

02 *Tipifarnib Using CXCL12 Pathway Biomarkers*

03 *KO-947 (ERK Inhibitor)*

04 • *KO-539 (Menin-MLL Inhibitor)*

KO-539: Potent Inhibitor of Menin-MLL Interaction

Summary

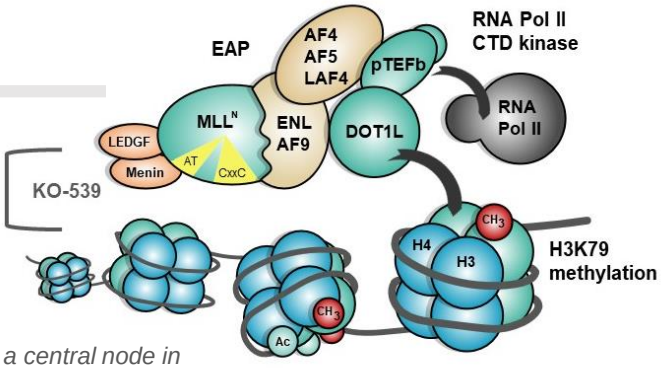
- Potent, selective small molecule inhibitor of the menin-MLL interaction
- Robust antitumor activity observed in mixed lineage leukemias rearranged (MLL-r) as well as disseminated NPM1mut and DNMT3Amut AML PDX models
- Preliminary data suggests anti-leukemic activity by induction of myeloid differentiation in AML blasts
- NPM1, MLL-r and MLL-PTD mutations occur in ~40% of AML patients[1]

KO-539

Menin-MLL inhibitor

Clinical Development and Status

- First patient dosed in Phase 1 trial in Sep 2019
- Goal: Achieve recommended Phase 2 dose in 2020



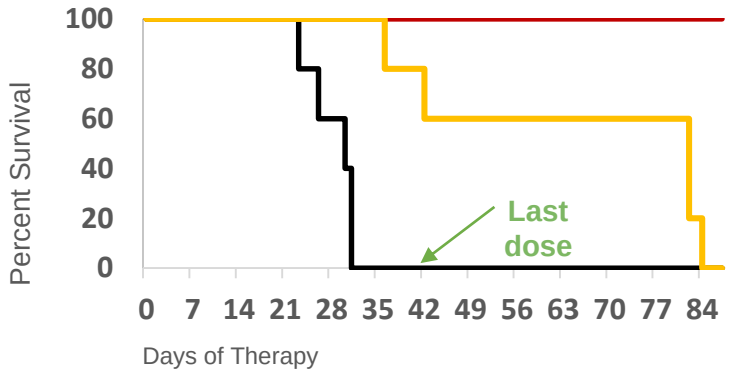
The menin-MLL complex appears to be a central node in epigenetic dysregulation driven by several distinct oncogenic driver mutations important in diverse leukemias and myeloproliferative disorders

[1] N Engl J Med. 2012 Mar 22;366(12):1079-89 | Haematologica. 2007 Jun;92(6):744-52 | Anticancer Res. 2005 May-Jun;25(3B):1931-44

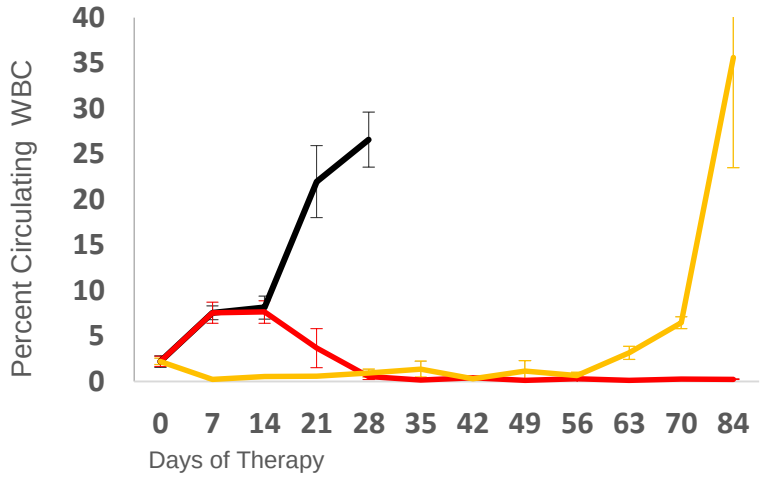
KO-539 Produces Lasting Complete Remissions in a NPM1/DNMT3A/IDH2/FLT3-Mutant AML Model

AM7577 Model

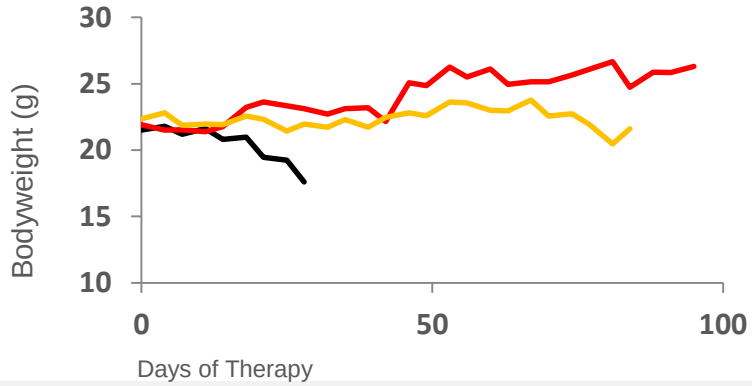
Overall survival



CD45+ human AML blasts



Tolerability



Vehicle

KO-539 100 mg/kg QD

Quizartinib 3 mg/kg QD

Anticipated Milestones & Financial Highlights

Program		Milestone	Status
Tipifarnib Farnesyl Transferase Inhibitor	HRAS Mutant Indications	Initiation of registration-directed trial in HNSCC (AIM-HN)	✓
		Data from Phase 2 trial in urothelial carcinoma	2020
		Potential for full enrollment in AIM-HN	End of 2020
	CXCL12 Pathway Indications	Proof of concept in AITL and CXCL12+ PTCL	✓
		Data from AITL expansion cohort in Phase 2 trial	ASH 2019
		Regulatory feedback from Phase 2 trial in AITL	1H 2020
		Data from Phase 2 trial in CMML	1H 2020
		Initiation of proof-of-concept study in pancreatic cancer	2020
KO-947 ERK Inhibitor		Potential biomarker of activity	✓
		Completion of dose escalation in Phase 1 trial	End of 2019/Early 2020
KO-539 Menin-MLL Inhibitor		First patient dosed in Phase 1 trial	✓
		Recommended Phase 2 dose in Phase 1 trial	2020

Financial Highlights	Nasdaq: KURA
	Shares outstanding: 45.3M basic, 4.1M options*
	Cash, cash equivalents and short-term investments: $250.1M*

* As of September 30, 2019

KURA ONCOLOGY

Developing Precision Medicines for the Treatment of Cancer