



Corporate Presentation

September 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: HRAS mutant HNSCC, HRAS mutant urothelial carcinoma, AITL and CXCL12+ PTCL
- 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; IND cleared March 2019

Near-Term Milestones

Multiple upcoming development milestones across the pipeline in 2H 2019 and early 2020

Financials

\$261 million in cash as of June 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

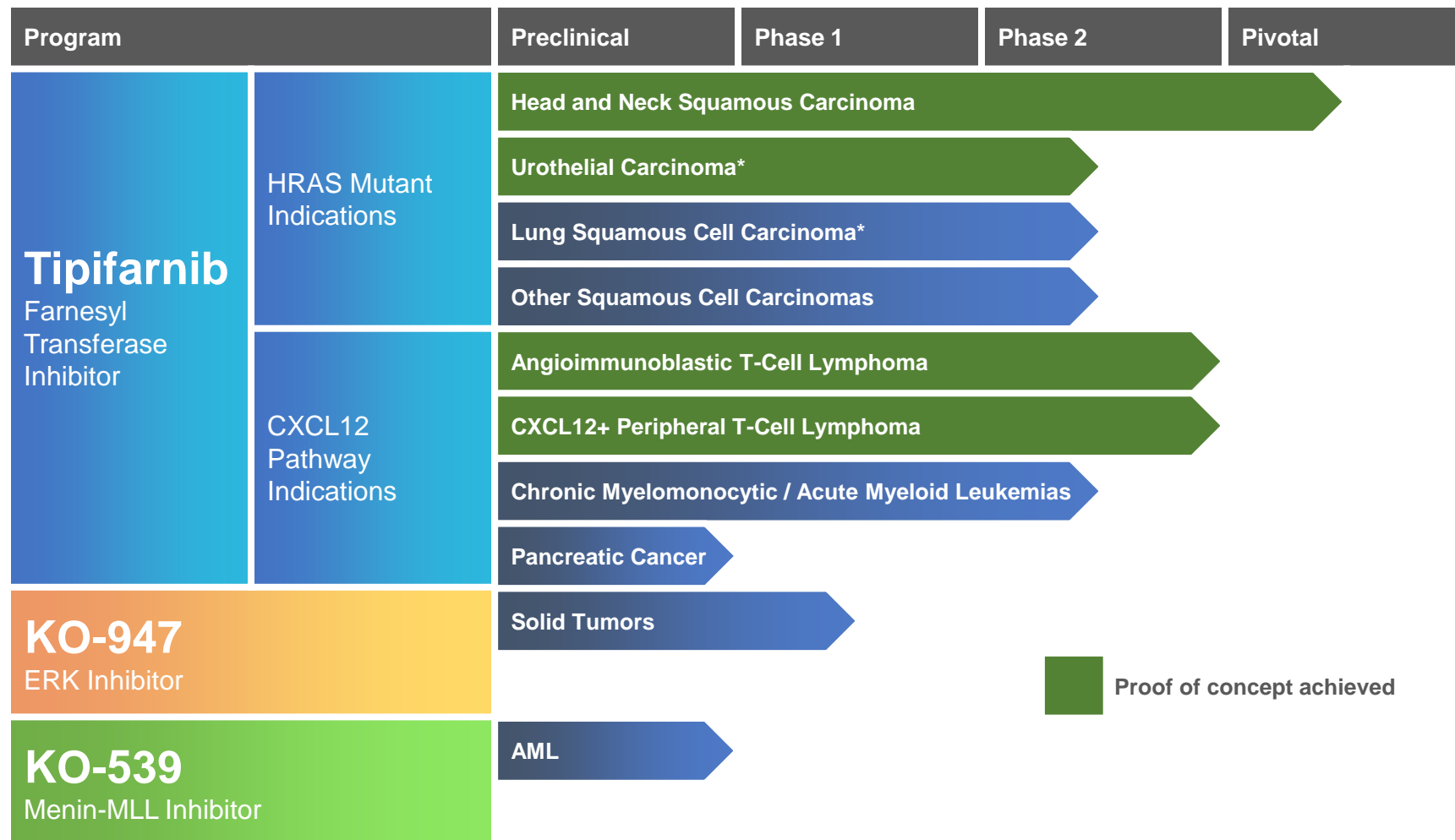
Development Status

- Registration-directed AIM-HN study
- Multiple Phase 2 trials ongoing

- Ongoing Phase 1 dose-escalation trial

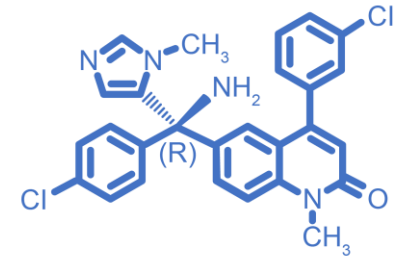
- Phase 1 trial expected to initiate in 2H 2019

Product Candidate Pipeline



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

- Extremely potent and selective inhibitor of farnesyl transferase¹ 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*

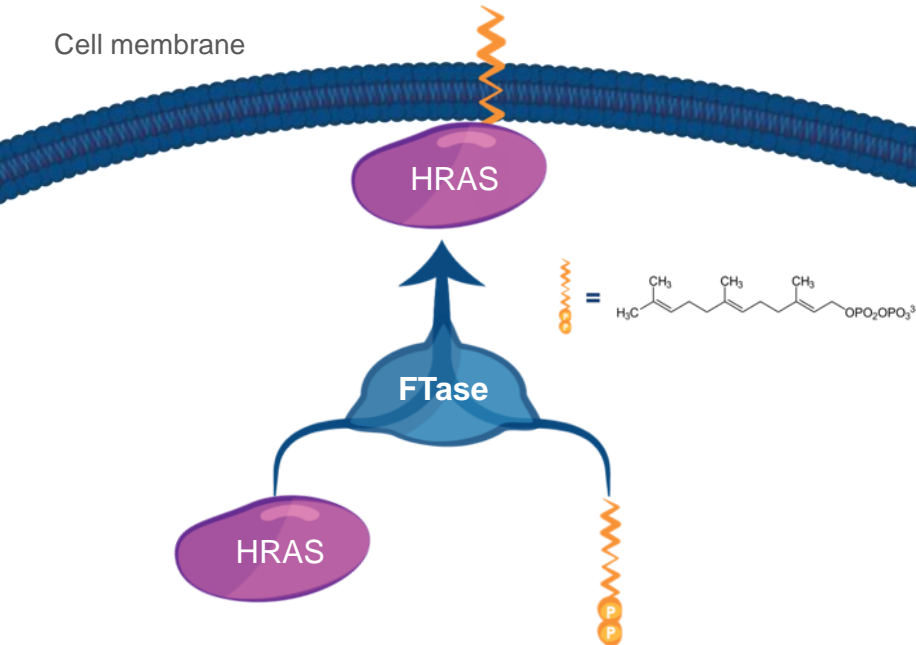
02 *Tipifarnib Using CXCL12 Pathway Biomarkers*

03 *KO-947 (ERK Inhibitor)*

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

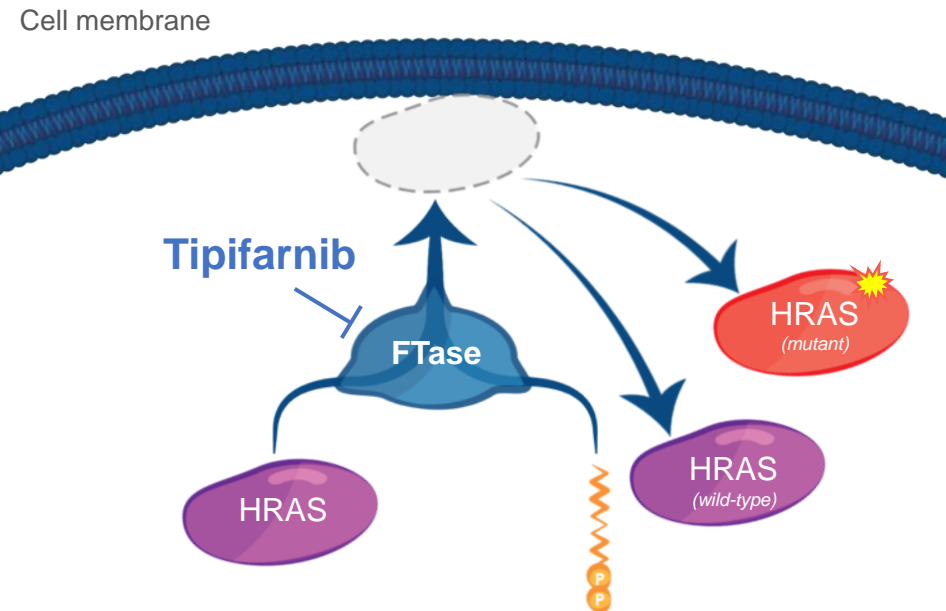
Tipifarnib Inhibits Farnesylation – An Essential Modification Required for HRAS Activity

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



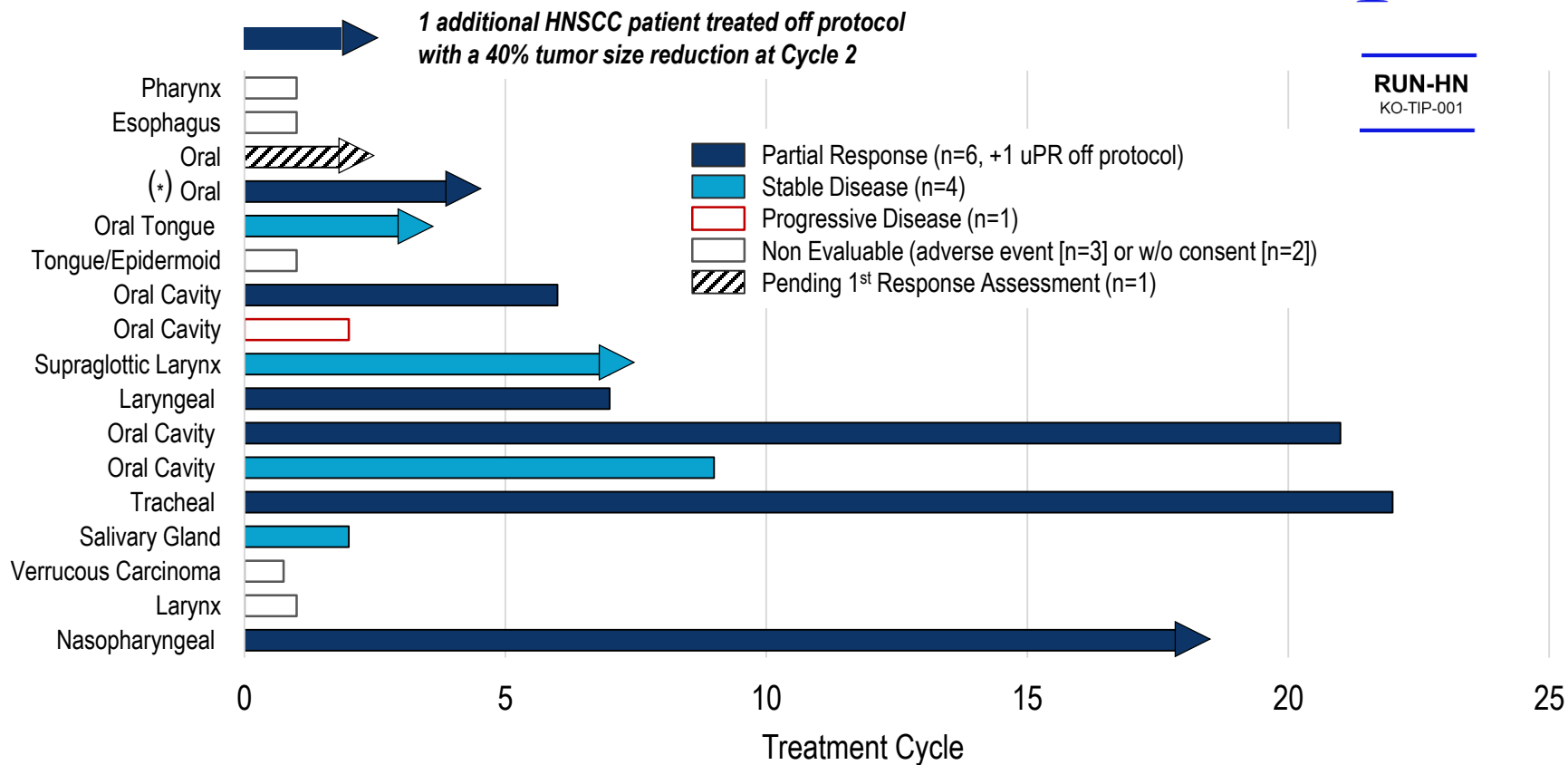
- Blocking farnesylation **prevents wild-type and mutant HRAS membrane localization**
- While NRAS and KRAS can be anchored to the membrane via an alternative pathway, **HRAS is solely dependent on FTase**

Tipifarnib: Phase 2 Proof-of-Concept Study in HRAS Mutant HNSCC

N=17 patients on study + 1 patient treated off protocol



RUN-HN
KO-TIP-001



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

HRAS Mutant HNSCC / SCC Indications

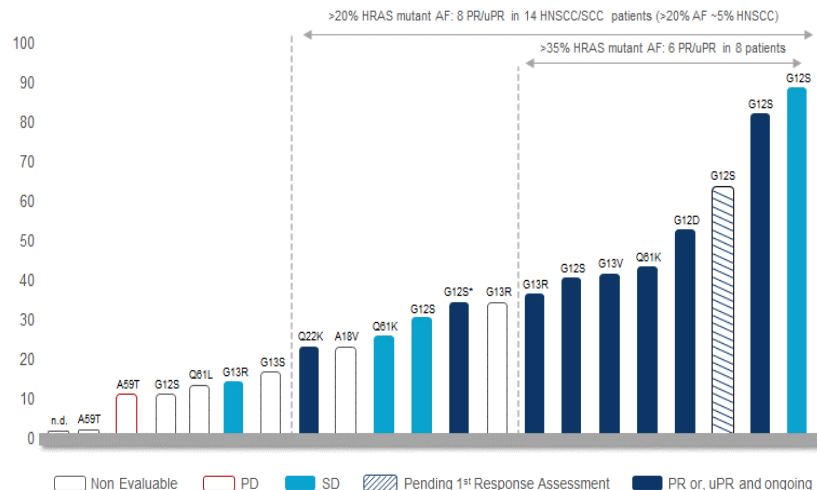


RUN-HN
KO-TIP-001

HRAS Mutant HNSCC/Other SCC



Clinical benefit observed in **HNSCC/SCC** with **> 20% HRAS mutant VAF**



Registration-Directed Trial

HRAS mutant HNSCC N=59

- Population: Recurrent/Metastatic HNSCC after one prior line of platinum therapy
- Statistical assumptions: N=59, 15% ORR (null hypothesis)
- Trial initiated and open for enrollment in November 2018; Two-year accrual
- As designed, AIM-HN may be adequate to support an NDA seeking accelerated approval*



AIM-HN
KO-TIP-007

HRAS Mutant Cancers: Market Opportunity

HNSCC Represents Significant Unmet Need¹

1L

ORR 36%

PFS 5.6 months

OS ~10 months

2L

ORR 13-16%

PFS ~2 months

OS 6-8 months

- Outcome of SOC in unselected populations
- Lower response rate expected in HRAS mutant patients²

Populations Based on Annual U.S. Incidence

HRAS Mutant
HNSCC

2,900-4,700
patients*

HRAS Mutant
All SCCs

7,500+
patients

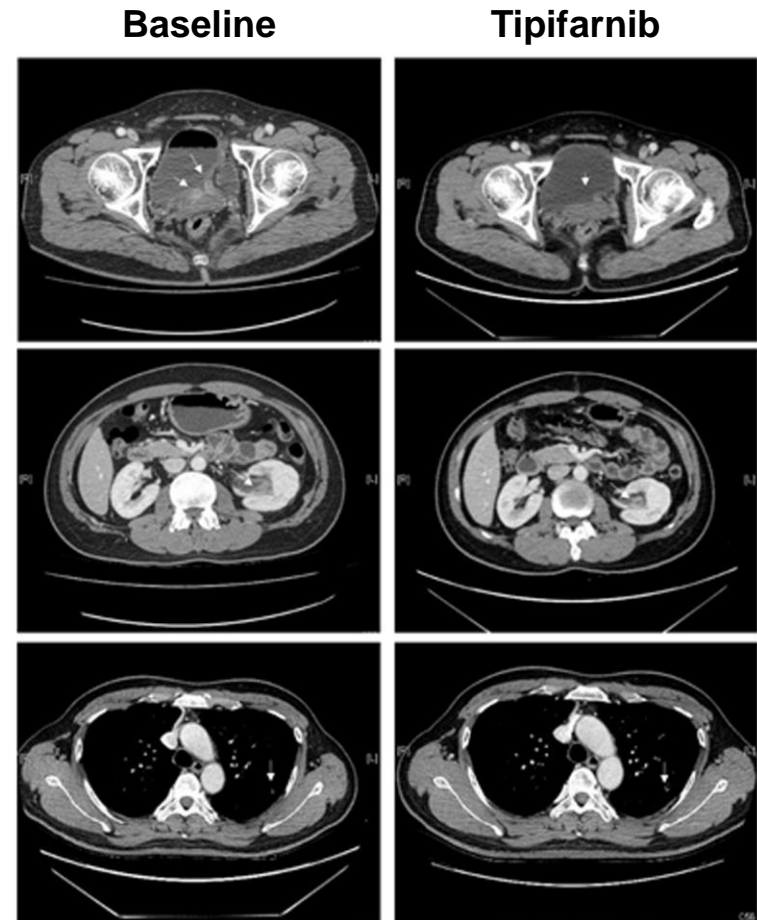
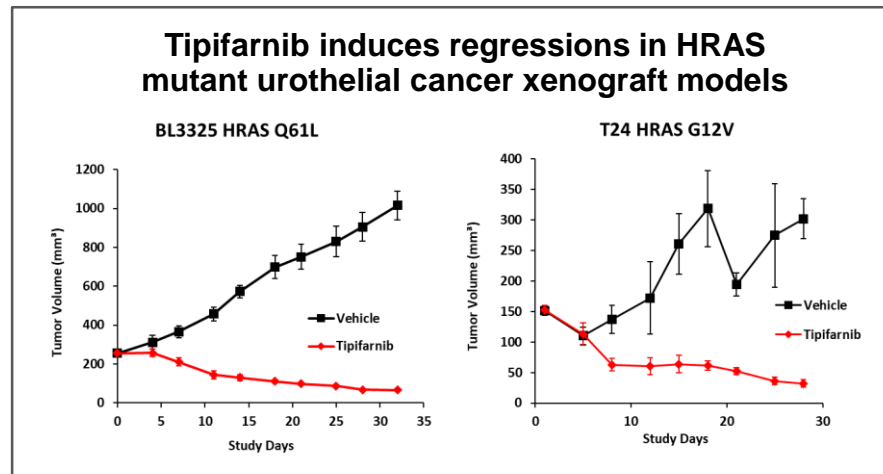
¹ *N Engl J Med.* 2008 Sep 11;359(11):1116-27 | Keytruda & Opdivo package inserts | *J Clin Oncol.* 2007 Jun 1;25(16):2171-7

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)

Tipifarnib: Proof-of-Concept Achieved in HRAS Mutant Urothelial Carcinoma

- 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



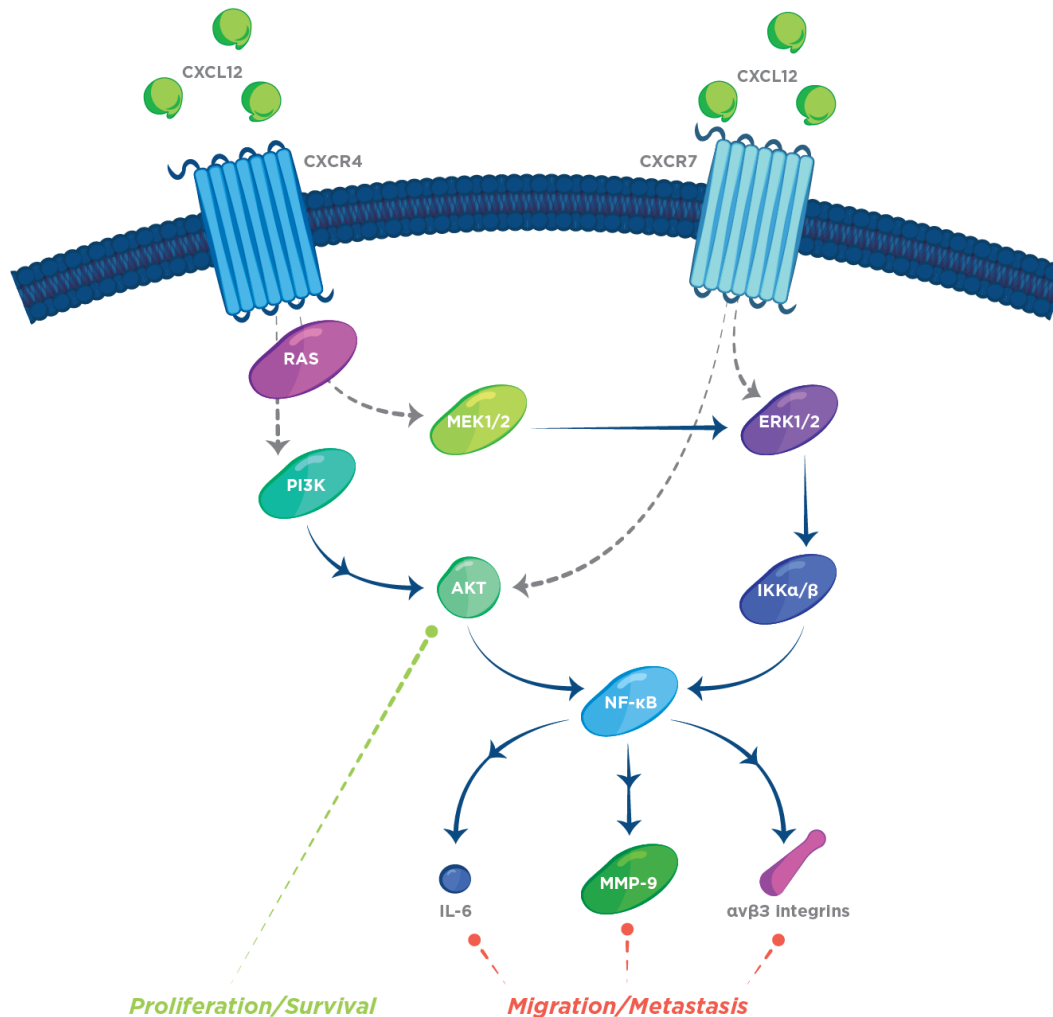
01 | *Tipifarnib in HRAS Mutant Solid Tumors*

02 • *Tipifarnib Using CXCL12 Pathway Biomarkers*

03 | *KO-947 (ERK Inhibitor)*

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

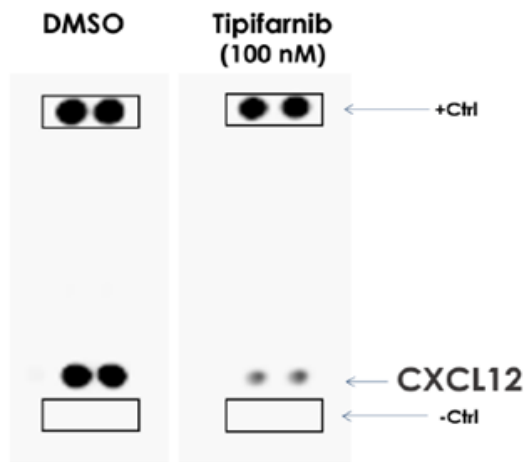
CXCL12 and CXCR4/7 Signaling Drive Multiple Downstream Pathways Critical to Cancer



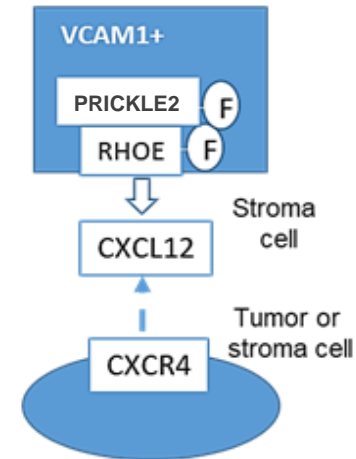
Key Features:

- 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

Tipifarnib is a CXCL12 Pathway Inhibitor



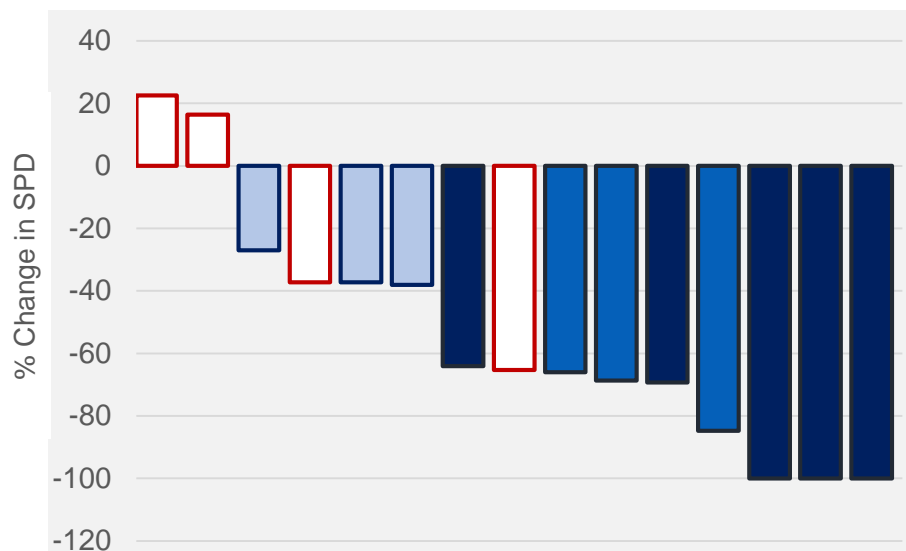
Tipifarnib downregulates CXCL12 secretion ex-vivo in CD1 mouse bone marrow stroma cultures



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 targets¹

Tipifarnib: Phase 2 Proof-of-Concept Study in Angioimmunoblastic T-Cell Lymphoma (AITL)

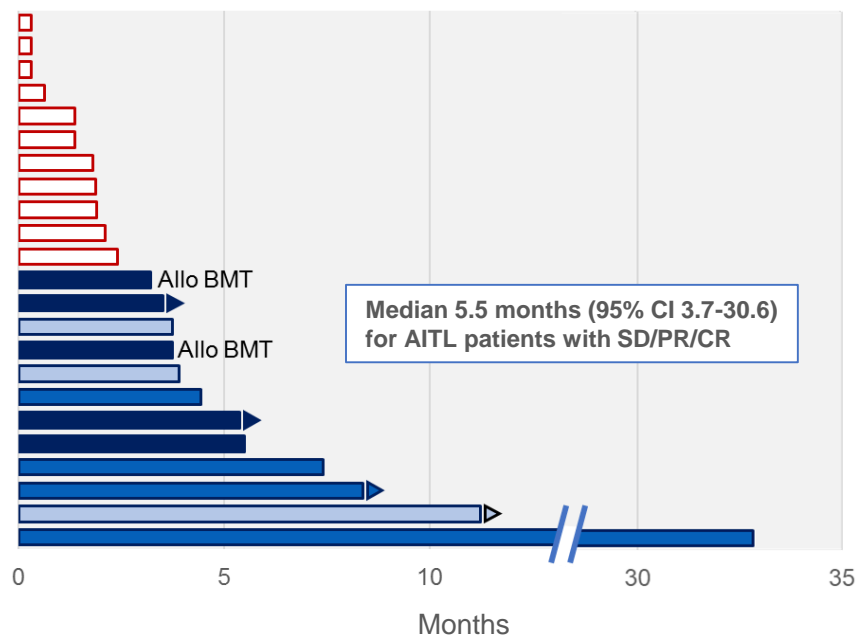
Maximum Change in Tumor Burden



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



Time on Treatment

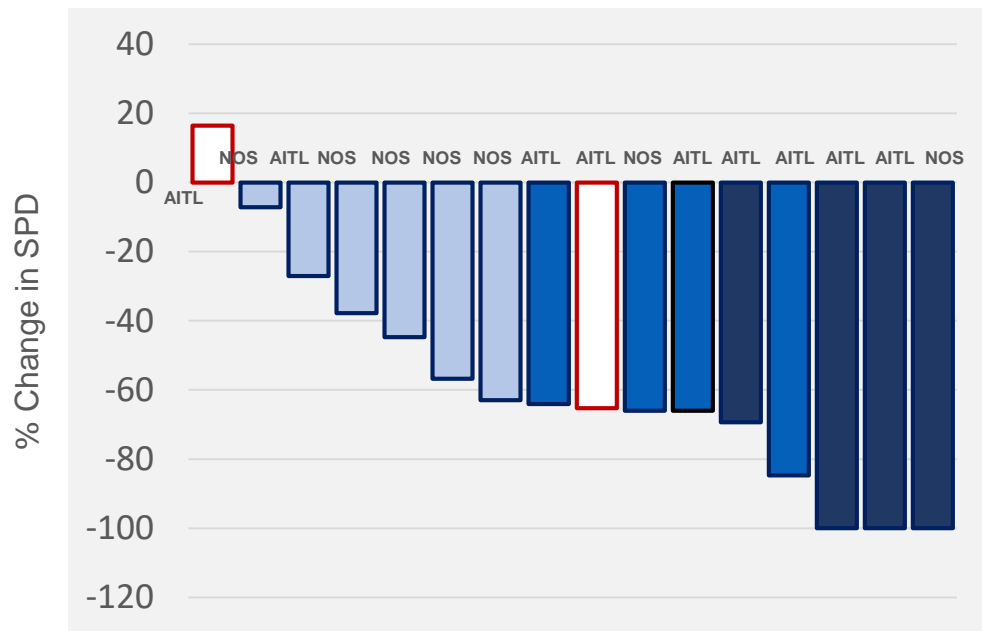


Preliminary data as of May 24, 2019

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

Maximum Change in Tumor Burden



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	2.3
36	NOS	SD	1.3
17	NOS	SD	0.85
32	AITL	PD/NE	0.8
37	NOS	SD	0.6
1	AITL	CR	0.48
30	AITL	PR	0.4
7	AITL	PD/NE	0.3
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	0.13
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	0.03
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 expression¹.

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

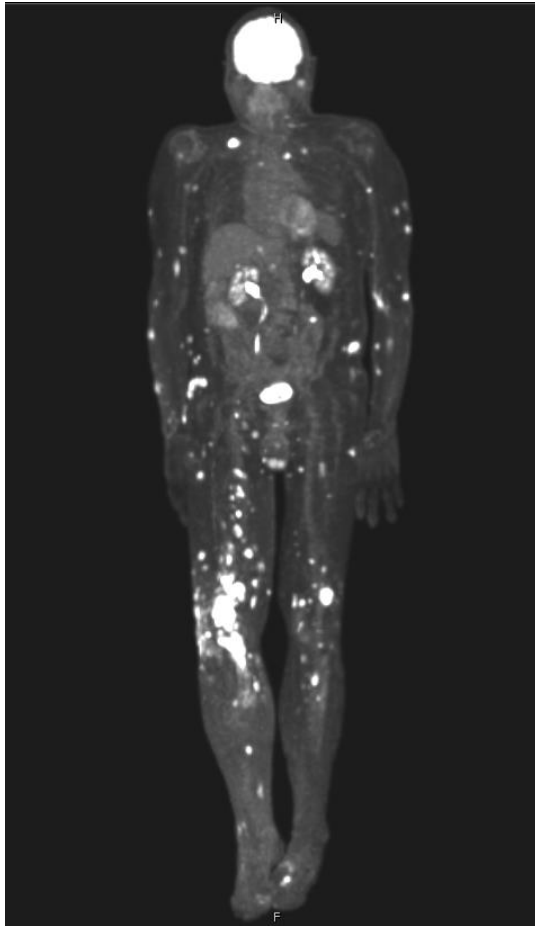
- CXCL12 and CXCL5, respectively, drive sensitivity and resistance to tipifarnib
- AITL expresses high levels of CXCL12 and is sensitive to tipifarnib
- AITL also expresses CXCL5; however, ~50% of AITL carry mutations of KIR3DL2, express low levels of CXCL5 and are highly sensitive to tipifarnib (50% 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)

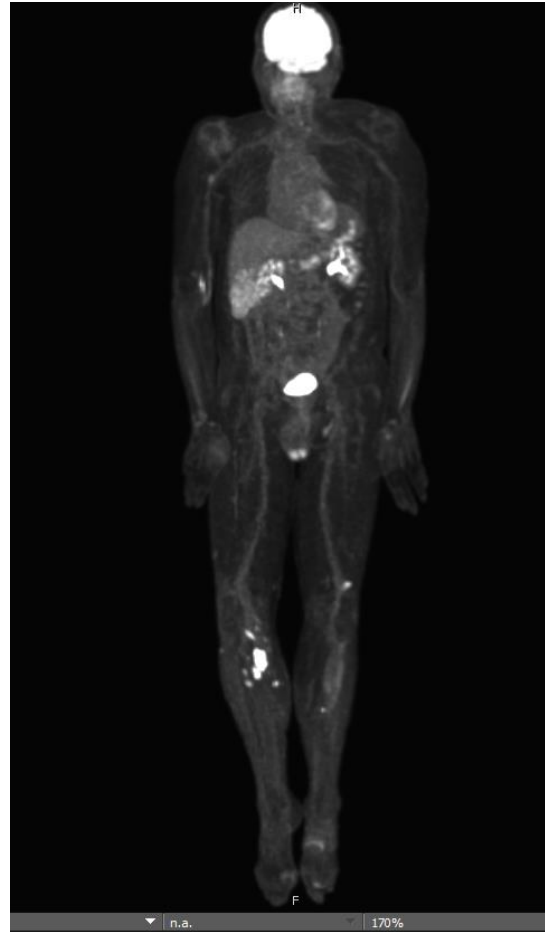
KIR3DL2 Mutant		KIR3DL2 Wild Type
N	8	8
Overall Best Response		
Complete Response (CR)	4	-
Partial Response (PR)	2	2
Stable Disease (SD)	2	-
Progressive Disease (PD)	-	6
Not evaluable (NE)	-	-
		25%
Overall Response Rate (CR + PR)	75%	4.6 - 64.1
95% CI	35.9 - 95.4	
		25%
Clinical Benefit Rate (CR + PR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

Preliminary data as of May 24, 2019

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



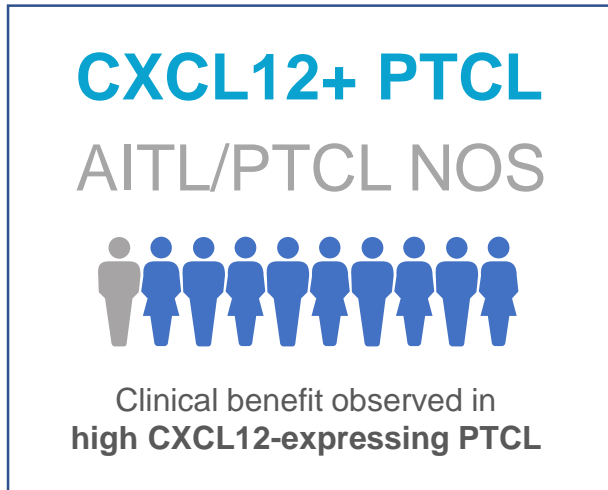
Baseline



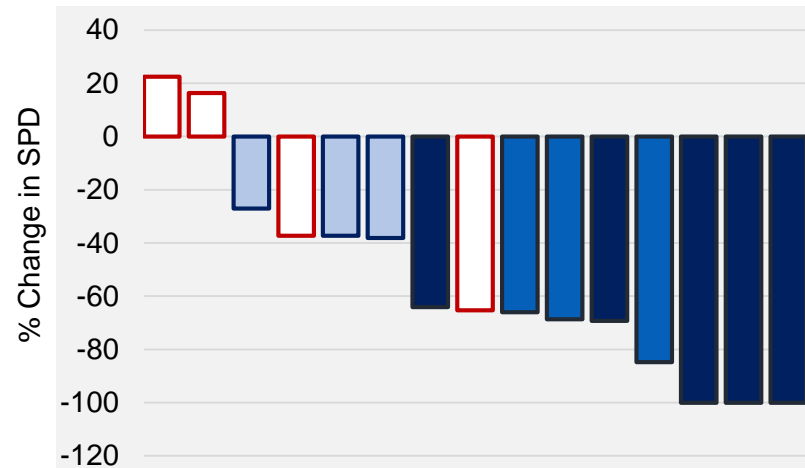
End of Cycle 2

- 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



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 Arm¹ N=120	Single Arm² N=130	Single Arm³ N=109	Single Arm⁴ 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%
- Folutyn, Beleodaq and Istodax are not approved in EU

¹ Beleodaq® package insert

² Istodax® package insert

³ Folutyn® package insert

⁴ Adcetris® package insert

⁵ 1,000 mg/m² administered over 30 mins by IV infusion once daily on days 1-5 of a 21-day cycle

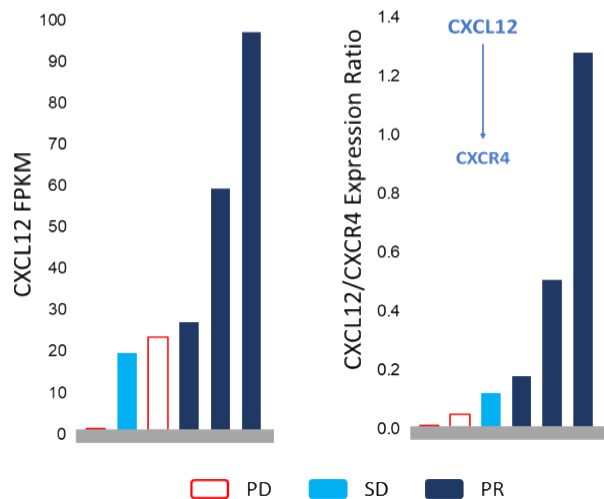
⁶ 14 mg/m² administered over a 4-hour period by IV on days 1, 8 and 15 of a 28-day cycle

⁷ 30 mg/m² administered over 3-5 mins as an IV push once weekly for 6 weeks in 7-week cycles

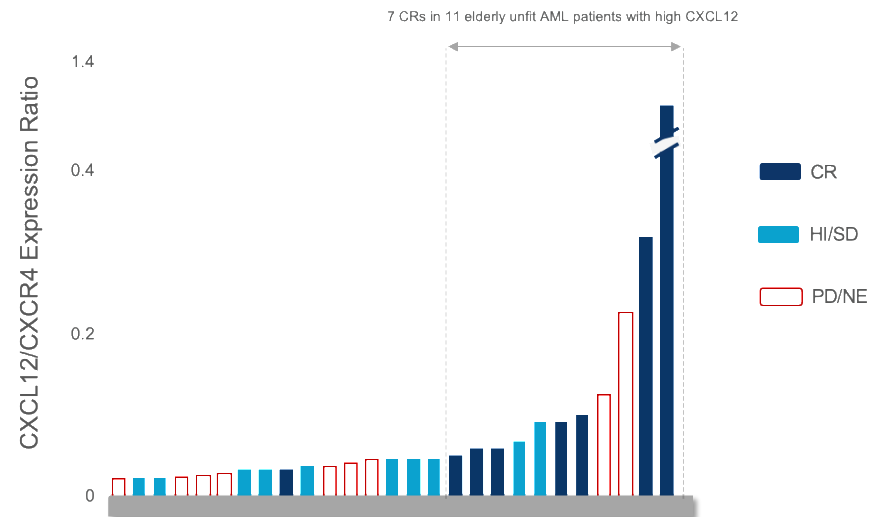
⁸ 1.8 mg/kg administered intravenously over 30 minutes every 3 weeks for up to 16 cycles

Prior Data Supports Expansion of CXCL12 Pathway Inhibition Beyond AITL/PTCL-NOS¹

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



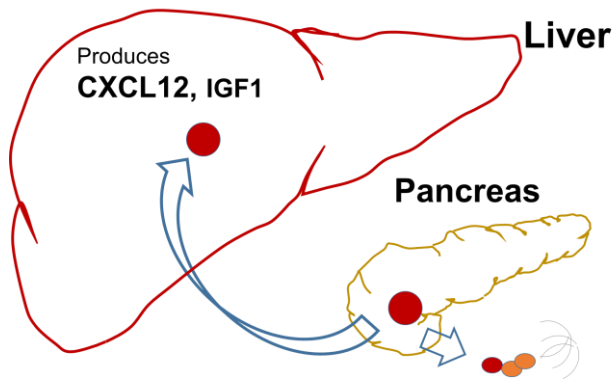
Association of High CXCL12 with Activity of Tipifarnib in Elderly Unfit AML



- 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

Inhibition of CXCL12 May Drive Clinical Benefit in Pancreatic Cancer

Disease Model for CXCL12 Expressing Sites



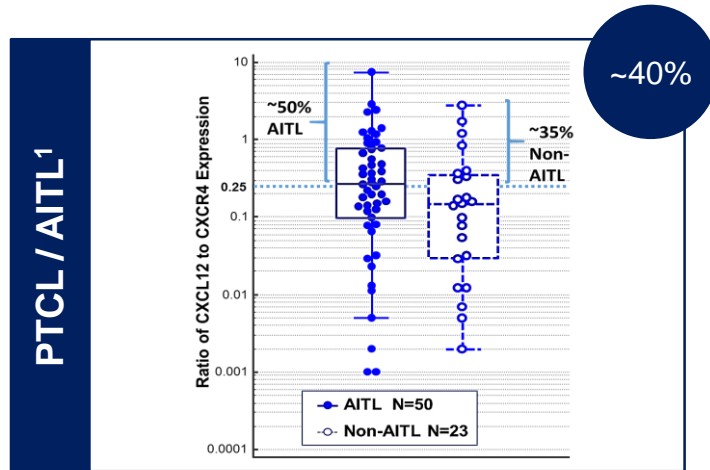
Rationale:

- CXCL12/IGF1 is known to induce tumor homing to liver and lymph nodes¹
- 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
 - $\leq 5\%$ KRAS mutant allele frequency

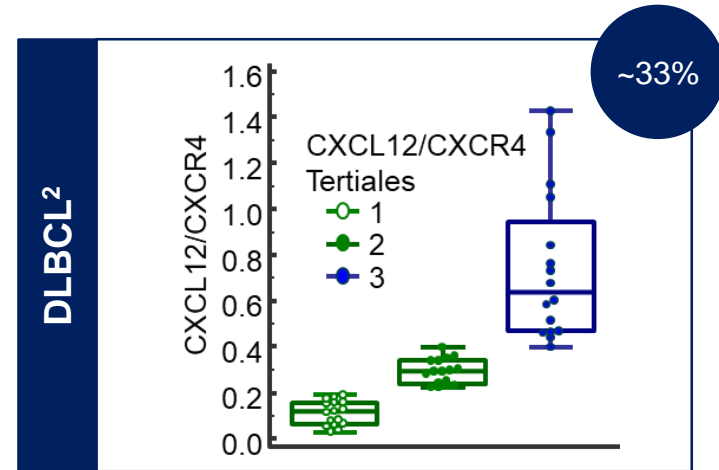
Next Steps:

- 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

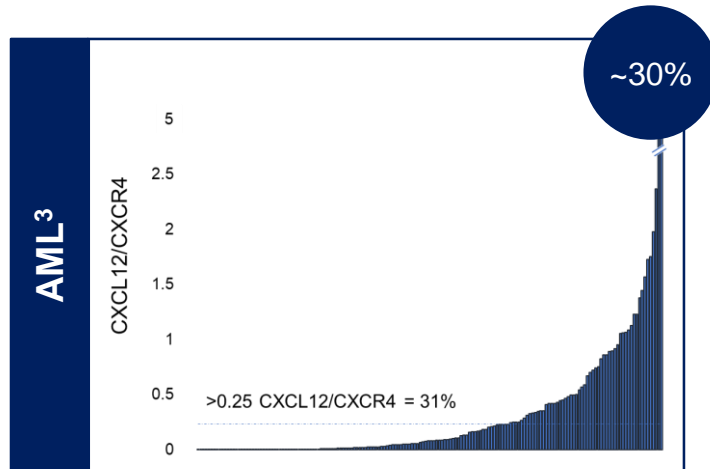
Tipifarnib Has Potential to Expand to Additional CXCL12-High Populations



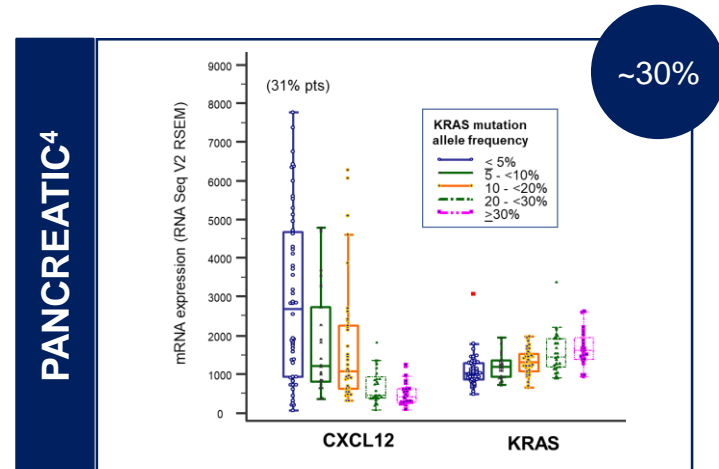
Est. Annual U.S. Incidence: 3,950⁵



Est. Annual U.S. Incidence: 27,650⁵



Est. Annual U.S. Incidence: 21,450⁶

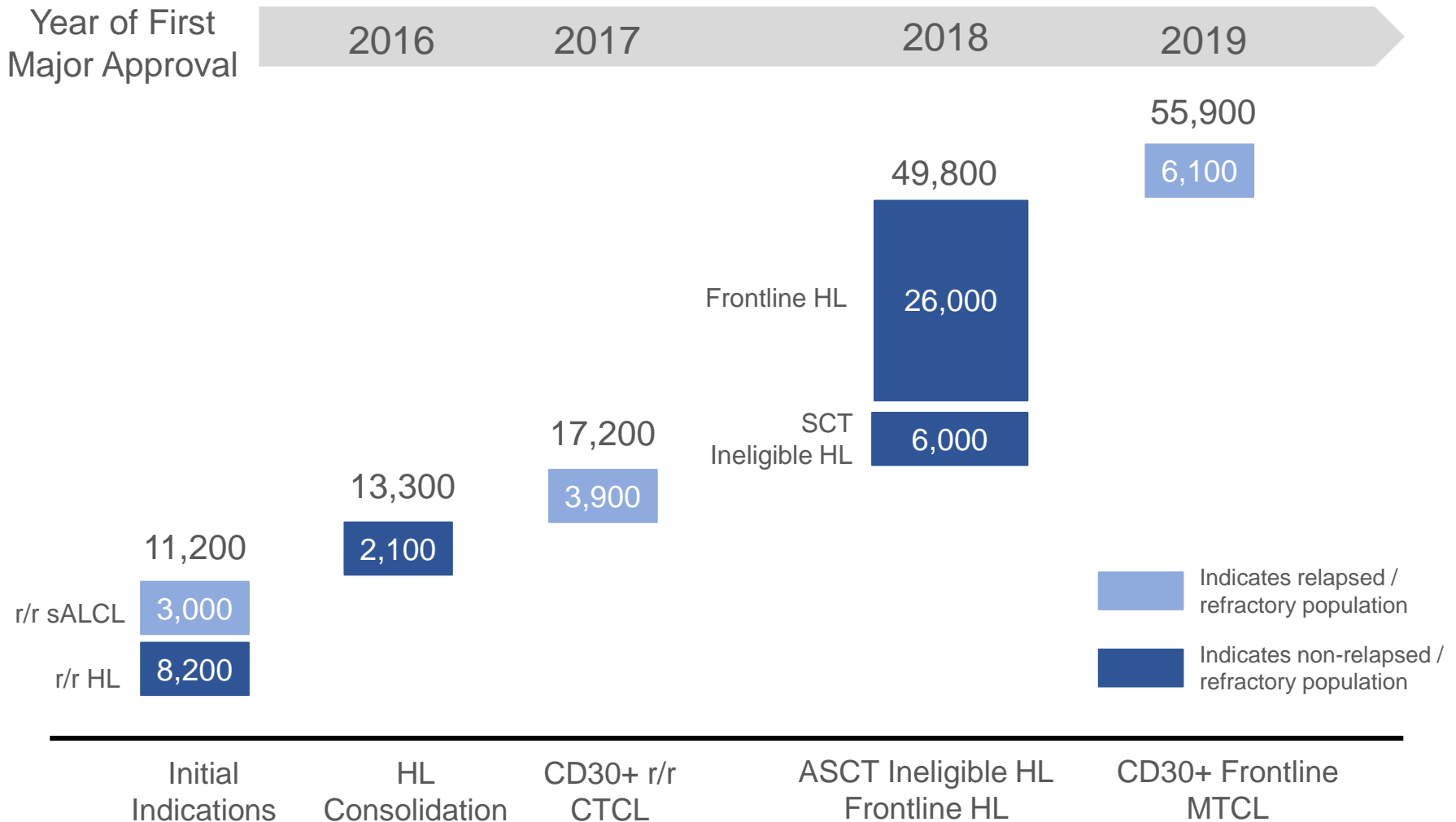


Est. Annual U.S. Incidence: 56,770⁶

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

CD30+ Lymphomas: An Illustrative Case Study of Biomarker-Driven Label Expansion

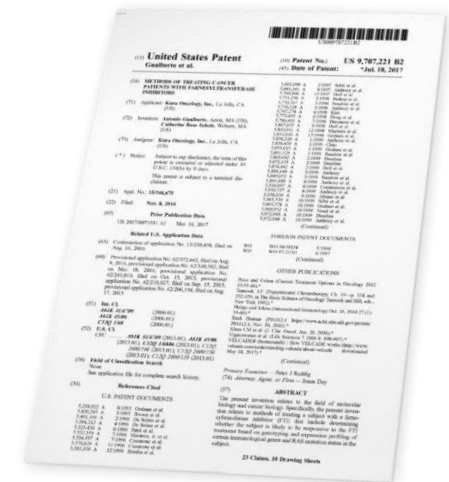


Source: Takeda, Seattle Genetics financial reports; Adcetris package insert

Note: Eligible populations are based on worldwide estimates; Market potential for tipifarnib may differ from that indicated by historical progress for CD30+ lymphomas

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
- First U.S. patent issued covering "any farnesyl transferase inhibitor" for treatment of HRAS mutant HNSCC
- 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



01 | *Tipifarnib in HRAS Mutant Solid Tumors*

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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

- **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 RP2D / MTD by year end 2019

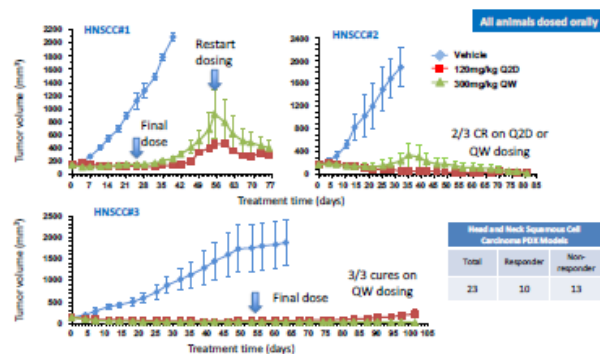


KO-947

ERK inhibitor

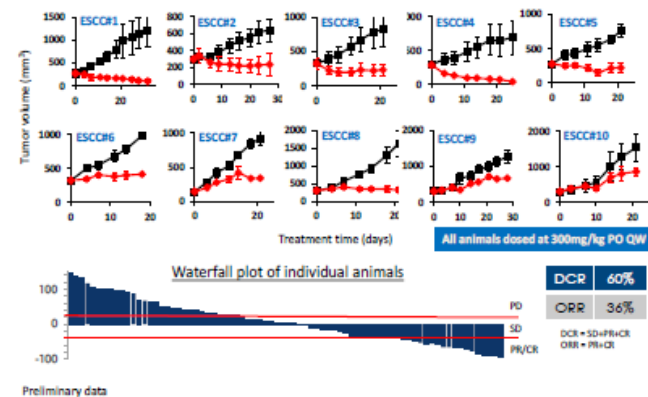
KO-947 Demonstrates Robust Single-Agent Activity in Preclinical Studies

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

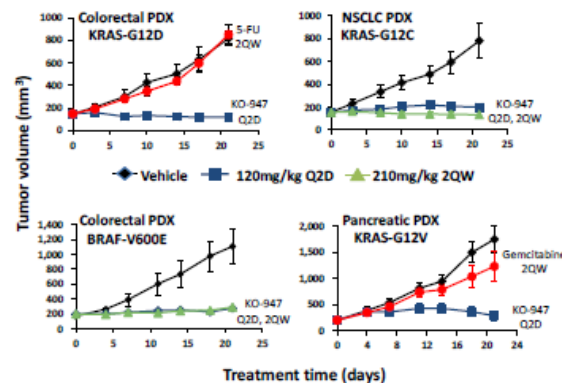


Head and Neck Squamous Cell Carcinoma PDX Models		
Total	Responder	Non-responder
23	10	13

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



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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
- **Menin-MLL inhibitors have the potential to treat approximately 50% of acute leukemias**

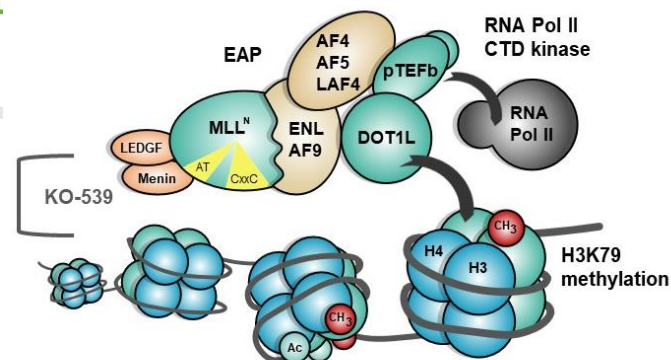
- **Status**

- IND application cleared in Q1 2019
- Initiation of Phase 1 study anticipated in 2H 2019



KO-539

Menin-MLL inhibitor

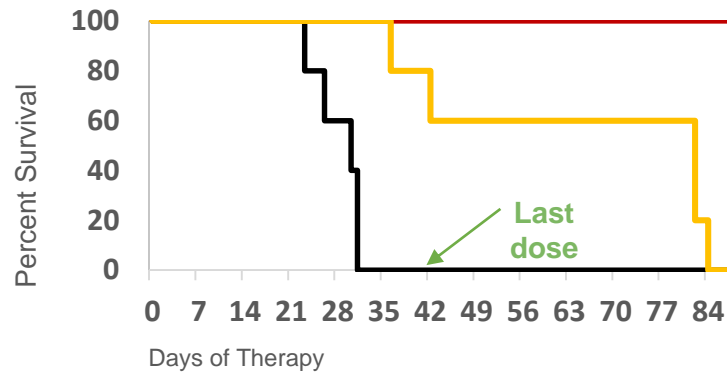


The menin-MLL complex appears to be a central node in epigenetic dysregulation driven by several distinct oncogenic driver mutations important in diverse leukemias and myeloproliferative disorders

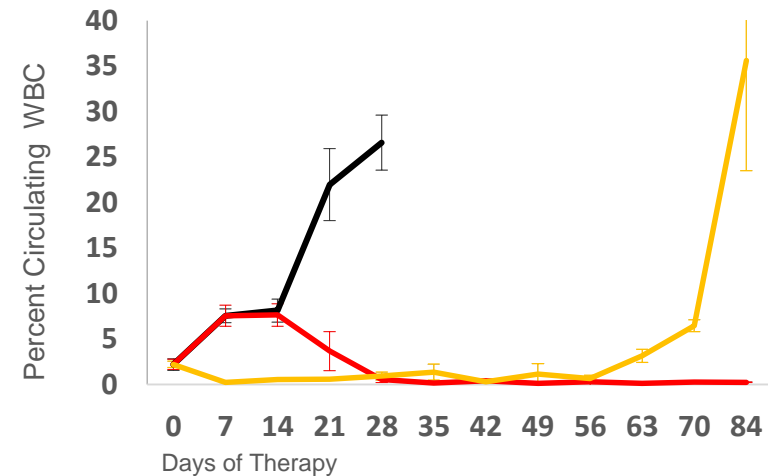
KO-539 Produces Lasting Complete Remissions in a NPM1/DNMT3A/IDH2/FLT3-Mutant AML Model

AM7577 Model

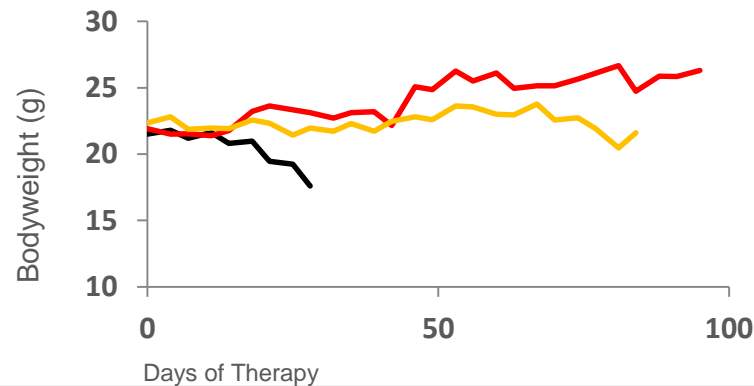
Overall survival



CD45+ human AML blasts



Tolerability



Vehicle

KO-539 100 mg/kg QD

Quizartinib 3 mg/kg QD

Anticipated Milestones & Financial Highlights

Program		Milestone	Status
Tipifarnib Farnesyl Transferase Inhibitor	HRAS Mutant Indications	Initiation of registration-directed trial in HNSCC	✓
		Data from Phase 2 trial in HNSCC and other SCCs	Q4 2019
		Data from Phase 2 trial in urothelial carcinoma	2020
	CXCL12 Pathway Indications	Proof of concept in AITL and CXCL12+ PTCL	✓
		Data from AITL expansion cohort in Phase 2 trial	Q4 2019
		Data from Phase 2 trial in CMML	1H 2020
		Initiation of proof-of-concept study in pancreatic cancer	1H 2020
	KO-947 ERK Inhibitor		Potential biomarker of activity
		Completion of dose escalation in Phase 1 trial	Q4 2019
KO-539 Menin-MLL Inhibitor		FDA clearance of IND application	✓
		Initiation of Phase 1 trial	2H 2019
Financial Highlights		Nasdaq: KURA	
		Shares outstanding: 45.2M basic, 4.1M options*	
		Cash, cash equivalents and short-term investments: \$261.4M*	

The background of the slide is a composite image. On the right side, there is a profile of an elderly man with a grey beard, looking towards the left. Overlaid on his face and the background are several translucent hexagonal shapes. A large, glowing green DNA double helix structure is prominent on the right side, partially overlapping the man's head. On the left side, there is a pattern of white dots of varying sizes arranged in a grid-like fashion, with some dots missing, creating a digital or data-like appearance. The overall color palette is dominated by light blues, greens, and greys, with a soft, ethereal glow.

**Developing Precision Medicines
for the Treatment of Cancer**