KURA ONCOLOGY

Developing Precision Medicines
for the Treatment of Cancer

EHA Data Review

June 14, 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.

Agenda for Today's Call

1) Introduction / Background
2) Data from Positive Phase 2 Trial of Tipifarnib in PTCL – EHA 2019
3) Opportunities to Expand to Additional CXCL12-Driven Indications
4) Upcoming Milestones
5) Q & A

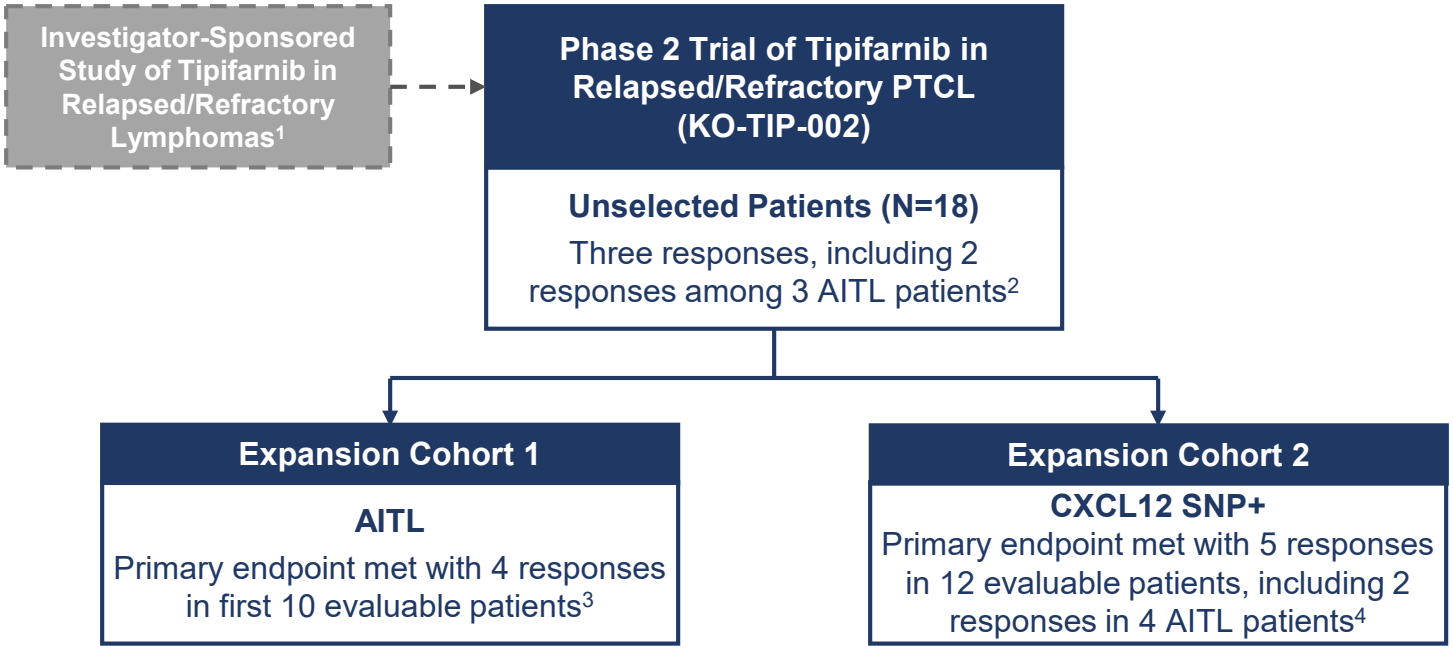
Kura Oncology – Key Themes

- Targeted therapies remain an essential category for new drug development in oncology
  - Enhanced clinical benefit in selected patient populations
  - Displacement of existing therapies and becoming part of the standard of care in a population of high unmet need
- Rationale for understanding “exceptional responders” in the clinic
  - Leverage technology toward comprehensive tumor profiling
  - Identification of biomarkers to enrich for clinical activity
  - Improved understanding of mechanisms of sensitivity and resistance
- A successful precision medicine based approach permits
  - Strategies for accelerated development and registration
  - Label expansion to other biomarker-guided populations
  - Extension to earlier lines of therapy through displacement or combination

Discovery of Tipifarnib as a First-in-Class CXCL12 inhibitor that Achieved Clinical POC

Witzig *et al.* EHA 2019 #S869	• **AITL and CXCL12 3'UTR SNP cohorts achieved clinical POC** • **Validation of robust clinical markers of sensitivity to tipifarnib** • **Definition of patient subsets with exceptional activity (AITL/KIR3DL2 mutant) and broader target population (CXCL12 3'UTR SNP)**
Gualberto *et al.* EHA 2019 #PS1002	• **Potential farnesylated targets that correlate with CXCL12 expression in AML**
Gualberto *et al.* AACR 2019 #CT191	• Retrospective analysis of patient samples from initial Phase 2 trial • CXCL12 expression associated with clinical activity in DLBCL and CTCL
Gualberto *et al.* ASCO GI 2019 #275	• Retrospective analysis identifies activity of tipifarnib in pancreatic Phase 3 trial using clinical surrogates of CXCL12 expression
Witzig *et al.* ASH 2018 #2937	• Tipifarnib reported to downregulate CXCL12 ex-vivo; AITL and CXCL12 3'UTR SNP cohorts designed to test prospectively the CXCL12 hypothesis • AITL cohort achieved clinical proof-of-concept • Tumor CXCL12 expression enriches for clinical activity in AITL (and PTCL NOS)
Witzig *et al.* ASH 2017 #2788; Gualberto *et al.* ASH 2017 #3957	• Identification of AITL histology, SNP in the 3'-UTR of CXCL12 gene and CXCL12/CXCR4 levels associated with clinical activity in T cell lymphoma • CXCL12 and bone marrow homing define tipifarnib's activity in AML
Witzig *et al.* 2011 *Blood* 118(18):4882	• Exploratory Phase 2 trial in relapsed and refractory lymphomas • No genetic selection but observed activity in PTCL patients

PTCL / AITL Trial Design



- Multi-center, single-arm, open-label Phase 2 trial designed to determine the efficacy, safety and biomarkers of tipifarnib in patients with relapsed or refractory PTCL
- Based upon molecular characterization of first 18 patients, trial amended to include two expansion cohorts:
  1) Patients with AITL, an aggressive form of T-cell lymphoma often characterized by high levels of CXCL12 expression
  2) Patients with PTCL who lack a single nucleotide variation in the 3'-untranslated region of the CXCL12 gene (CXCL12+)
- Expansion cohorts: Tipifarnib 300 mg twice daily (bid) on days 1-21 of 28-day treatment cycles

[1] Witzig *et al.* 2011 Blood 111:4882-89 | [2] Witzig *et al.* ASH 2017 #2788 | [3] Witzig *et al.* ASH 2018 #2937 | [4] Witzig *et al.* EHA 2019 #S869

Tipifarnib in Relapsed or Refractory Angioimmunoblastic T-cell Lymphoma (AITL) and CXCL12+ Peripheral T-cell Lymphoma (PTCL): Preliminary Results from an Open-Label, Phase 2 Study

Thomas Witzig[1], Lubomir Sokol[2], Won Seog Kim[3], Francine Foss[4], Eric Jacobsen[5], Fatima de la Cruz Vicente[6], Dolores Caballero[7], Ranjana Advani[8], Jose Maria Roncero Vidal[9], Ana Marin Niebla[10], Antonia Rodriguez Izquierdo[11], Raquel Oña Navarrete[12], Maria Jose Terol[13], Eva Domingo-Domenech[14], Marta Rodriguez[15], Miguel Piris[15], James Bolognese[16], Matthew R Janes[17], Francis Burrows[18], Linda Kessler[18], Vishnu Mishra[18], Robert Curry[19], Michael Kurman[19], Catherine Scholz[19] and Antonio Gualberto[19]

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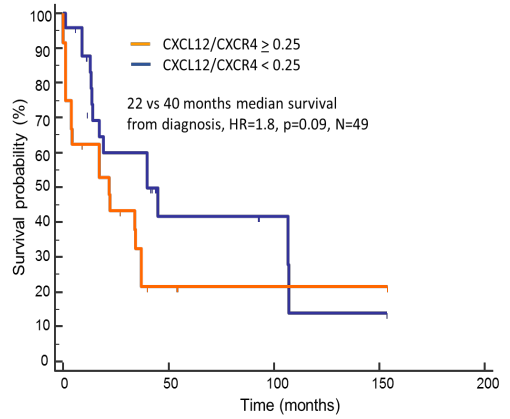
[14] Institut Catala d'Oncologia, Barcelona, Spain
[15] Fundación Jiménez Díaz, Madrid, Spain
[16] Cytel, Cambridge, MA USA
[17] Wellspring Biosciences, Inc., San Diego, CA USA
[18] Kura Oncology, Inc., San Diego, CA USA
[19] Kura Oncology, Inc., Cambridge, MA USA

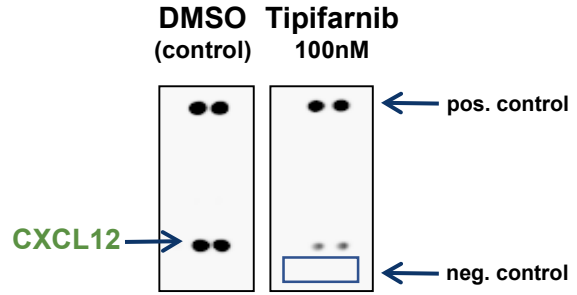
24th Congress of the European Hematology Association, Abstract S869

Tipifarnib is a CXCL12/CXCR4 Pathway Inhibitor

- **Key characteristics of CXCL12**
  - Expressed primarily by immune cells, endothelial cells and stromal fibroblasts that constitute the tumor microenvironment
  - CXCL12 and its receptors (CXCR4, CXCR7) are key factors linking cancer cells with the tumor microenvironment
- **High CXCL12 expression defines poor prognosis in PTCL**
  - 50% of AITL and 35% of PTCL-NOS have high CXCL12 expression
  - Trend for worse prognosis in AITL and PTCL-NOS patients with tumors with high CXCL12 expression[1]
- **Tipifarnib is a CXCL12/CXCR4 pathway inhibitor**
  - Tipifarnib downregulates CXCL12 secretion ex-vivo in stroma cultures
  - Expression of uniquely farnesylated proteins (RHOE and PRICKLE2) is strongly correlated with CXCL12 expression, suggesting potential CXCL12-related tipifarnib targets[2]
  - Resistance to tipifarnib potentially mediated by CXCR2 and its ligands (CXCL1, CXCL5, CXCL8) in myeloid indications[3]



Trend for poor prognosis with high CXCL12 expression (adjusted to CXCR4) in AITL and PTCL NOS pts



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

[1] Witzig 2018 *Blood* 132:2937 | [2] Gualberto EHA 2019 #PS1002 | [3] Gualberto *Blood* 2017 130:3957

Proof of Concept for Tipifarnib in wt CXCL12 3'UTR PTCL

	wt CXCL12 3'UTR Cohort: All pts		wt CXCL12 3'UTR Cohort: AITL pts		wt CXCL12 3'UTR Cohort: PTCL-NOS pts	
Total treated	15		4		11	
Total efficacy evaluable	12		3		9	
Overall Best Response						
Complete Response (CR)	3		2		1	
Partial Response (PR)	2		-		2	
Stable Disease (SD)	6		-		6	
Progressive Disease (PD)	1		1		-	
Not efficacy evaluable (NE)	3		1		2	
	PPS[1]	mITT	PPS[1]	mITT	PPS[1]	mITT
Overall Response Rate[1] (CR + PR)	**41.7%**	**33.3%**	**66.7%**	**50%**	**33.3%**	**27.3%**
95% CI	18.1 – 70.6	14.2 - 60.6	13.5 - 98.3	9.8 - 90.2	9.8 - 68.4	7.9 - 59.9
Clinical Benefit Rate[1] (CR + PR + SD)	**91.7%**	**73.3%**	**66.7%**	**50%**	**100%**	**81.8%**
95% CI	63.4 - 99.6	46.5 - 90.3	13.5 - 98.3	9.8 - 90.2	68.4 - 100.0	50.0 - 96.7

[1] Per protocol set – prespecified primary analysis population includes all pts who received at least 1 dose of tipifarnib and have 1 post-baseline tumor measurement

Preliminary data as of 24 May 2019

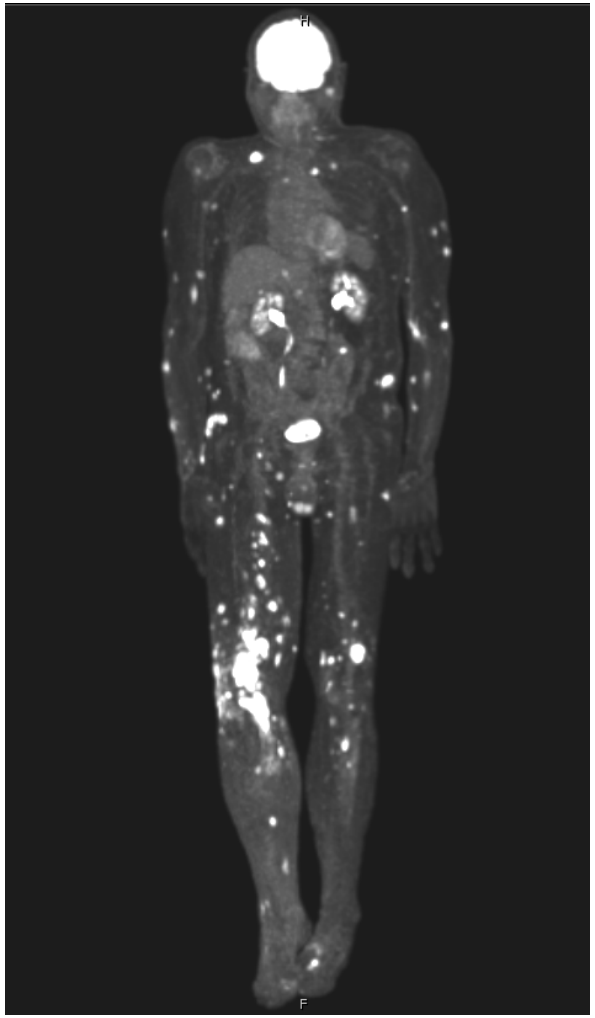
Enrichment by wt CXCL12 3'UTR PTCL-NOS

	All PTCL-NOS wt CXCL12 3'UTR[1]		All PTCL-NOS Variant CXCL12 3'UTR[2]
Total treated	17		6
Total efficacy evaluable	15		6
Overall Best Response			
Complete Response (CR)	1		-
Partial Response (PR)	2		-
Stable Disease (SD)	10		-
Progressive Disease (PD)	2		6
Not efficacy evaluable (NE)	2		-
	PPS[3]	mITT	PPS/mITT
Overall Response Rate (CR + PR)	**20%**	**17.6%**	**0%**
95% CI	5.7 - 46.5	5.0 - 41.7	0 - 40.6
Clinical Benefit Rate (CR + PR + SD)	**86.7%**	**76.5%**	**0%**
95% CI	60.6 - 97.6	51.1 - 91.5	0 - 40.6

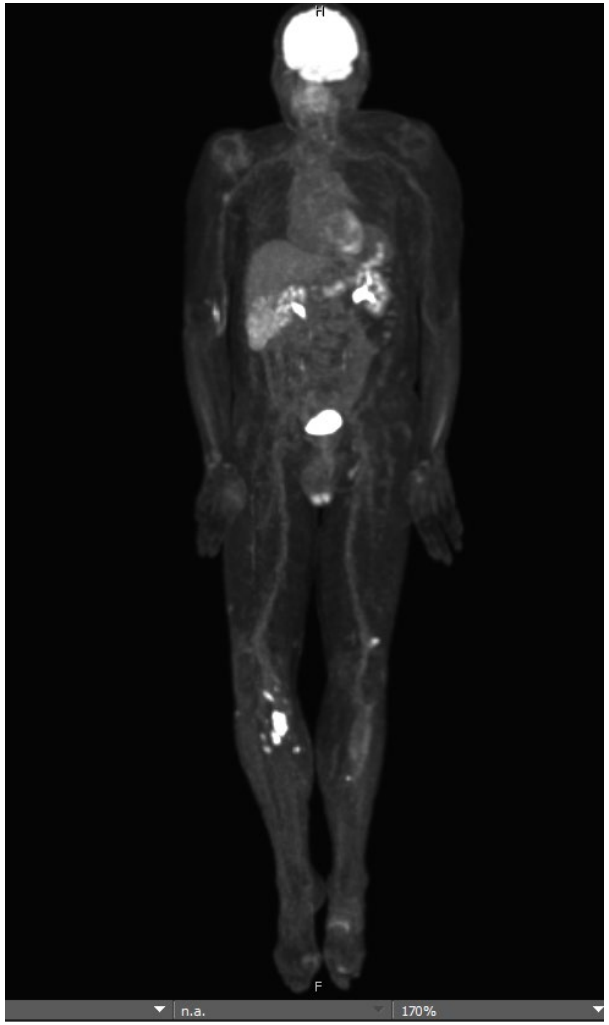
[1] All PTCL-NOS wt CXCL12 3'UTR includes all PTCL-NOS pts with CXCL12 rs2839695 A/A genotype enrolled in all portions of the trial.
[2] Includes PTCL-NOS pts with CXCL12 rs2839695 A/G or G/G genotype (enrolled in the original protocol stages 1 and 2)
[3] Per protocol set – prespecified primary analysis population includes all pts who received at least 1 dose of tipifarnib and have 1 post-baseline tumor measurement

Preliminary data as of 24 May 2019

Tumor Reduction in PTCL-NOS, wt 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

Proof of Concept for Tipifarnib in AITL

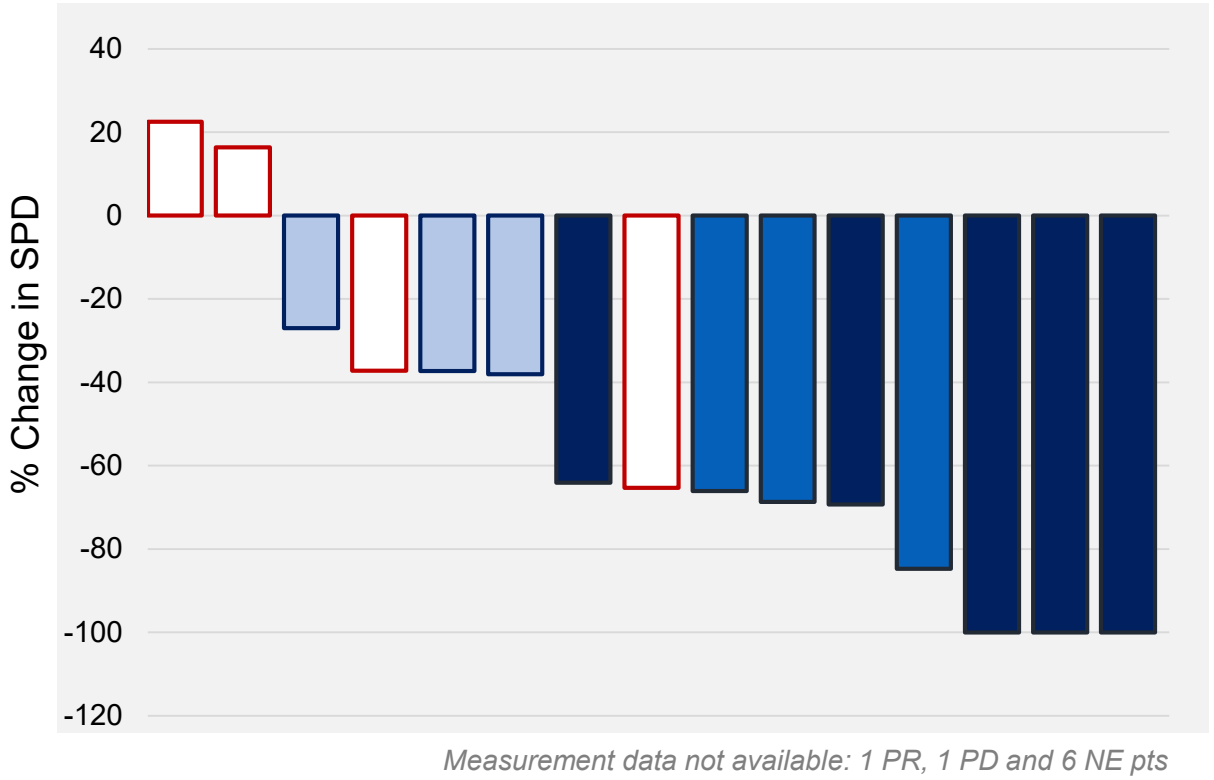
	AITL Cohort		All AITL [1]	
Total treated	16		23	
Total efficacy evaluable	11		17	
Overall Best Response				
Complete Response (CR)	3		5	
Partial Response (PR)	2		4	
Stable Disease (SD)	3		3	
Progressive Disease (PD)	3		5	
Not efficacy evaluable (NE)	5		6	
	PPS[2]	mITT	PPS[2]	mITT
Overall Response Rate (CR + PR)	**45.4%**	**31.3%**	**52.9%**	**39.1%**
95% CI	20.0 - 74.4	13.2 - 56.6	28.2 - 74.7	20.7 - 61.3
Clinical Benefit Rate (CR + PR + SD)	**72.7%**	**50.0%**	**70.6%**	**52.2%**
95% CI	40.1 - 92.1	27.2 - 72.8	45.6 - 87.6	32.0 - 72.6

[1] All AITL includes all AITL pts enrolled in all portions of the trial: original protocol (stages 1 and 2), AITL cohort and wt CXCL12 3'UTR cohort.
[2] Per protocol set – prespecified primary analysis population includes all pts who received at least 1 dose of tipifarnib and have 1 post-baseline tumor measurement.

Preliminary data as of 24 May 2019

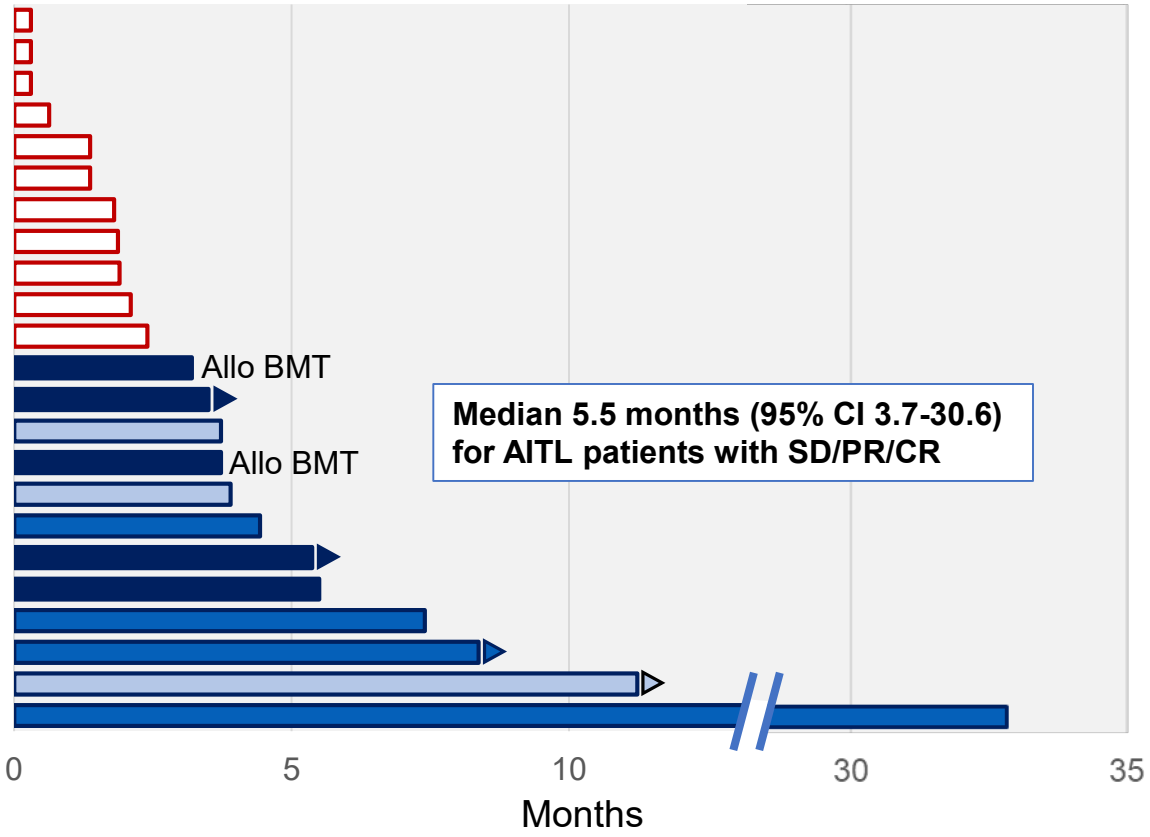
AITL: Tipifarnib treatment resulted in durable clinical responses and enabled subsequent transplant in patients achieving a CR

Maximum Change in Tumor Burden



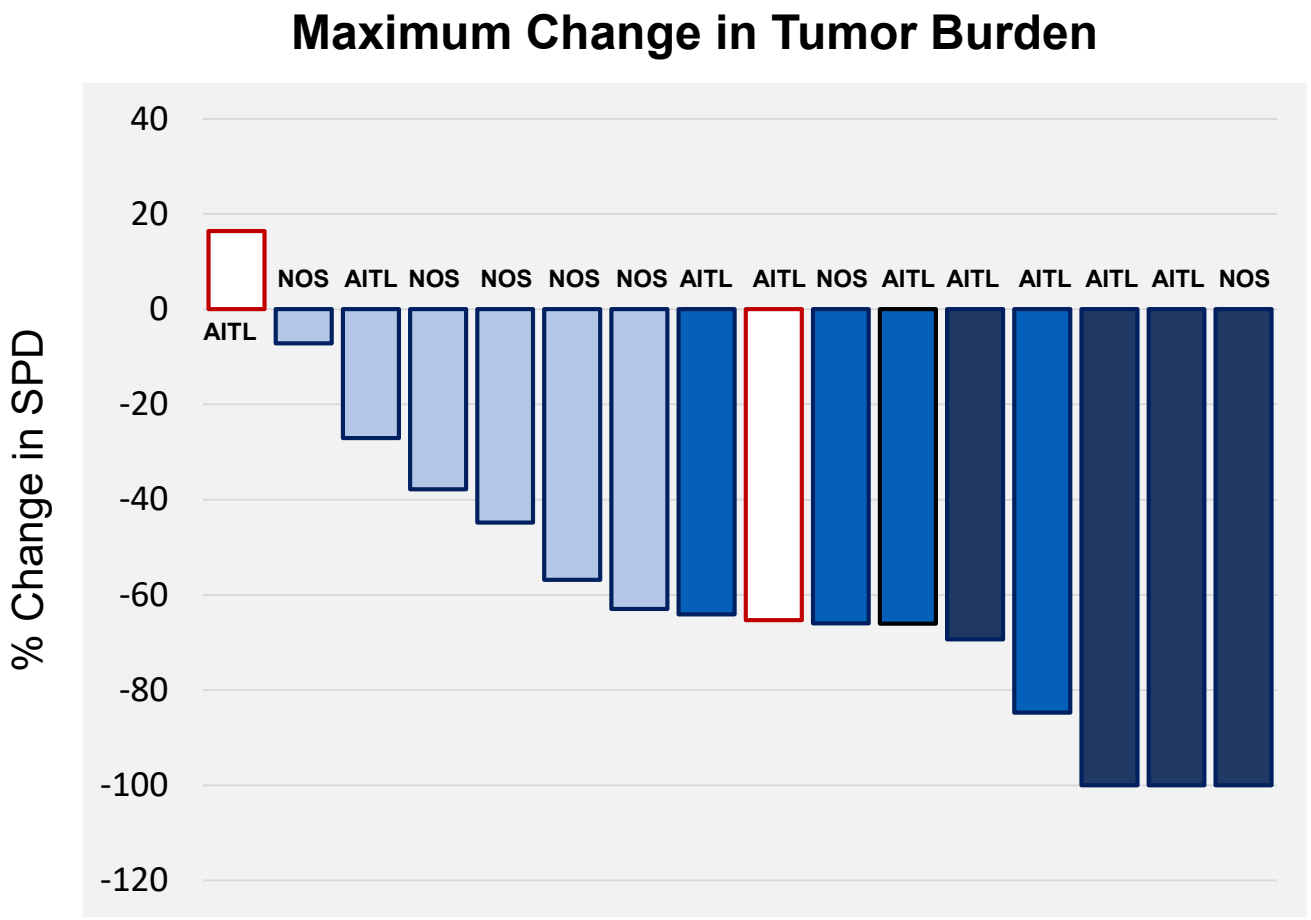
Measurement data not available: 1 PR, 1 PD and 6 NE pts

Time on Treatment



Preliminary data as of 24 May 2019

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.

[1] N = 34 tumors with available CXCL12 expression data.

Preliminary data as of 24 May 2019

High Activity of Tipifarnib in AITL with KIR3DL2 mutations

- CXCL12 and CXCL5 drive, respectively, 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

KIR data analyses to be presented at 15-ICML: Gualberto et. al. Abstract 156-P

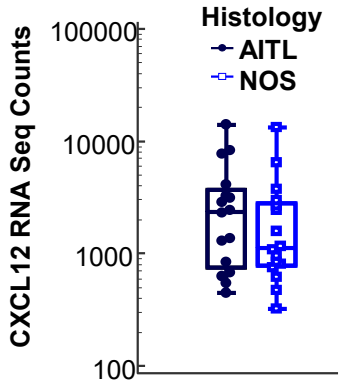
Preliminary data as of 24 May 2019

Drivers of Tipifarnib's Activity in AITL

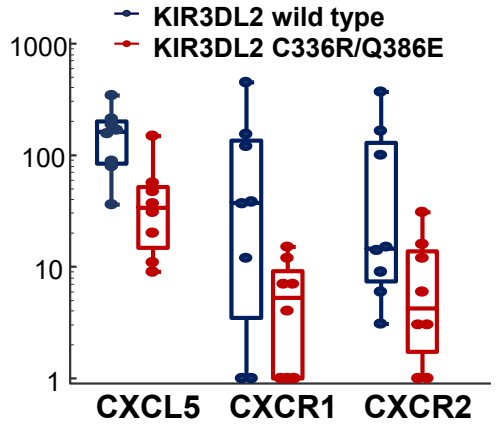
Overall high CXCL12 expression in AITL, Low CXCL5 expression in KIR3DL2 mutant AITL

Genetics

High CXCL12 expression in AITL histology

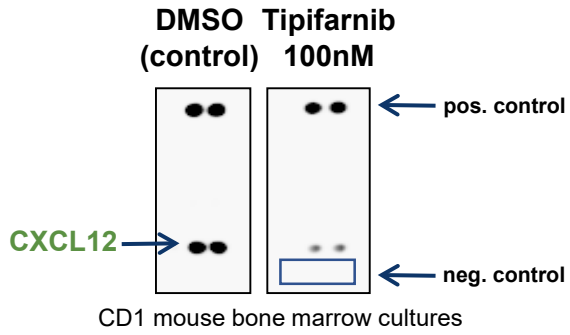


Low CXCL5 expression in KIR3DL2 mutant AITL

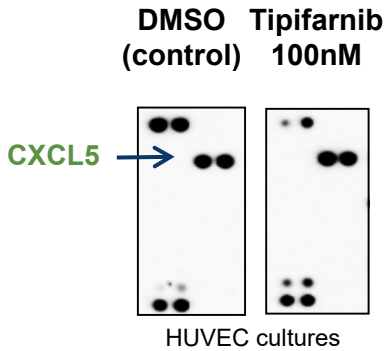


Mechanism of Action

Tipifarnib downregulates CXCL12 secretion



Tipifarnib does not inhibit CXCL5 secretion



Activity

	AITL KIR3DL2 Mutant
N	8
Overall Best Response	
Complete Response (CR)	4
Partial Response (PR)	2
Stable Disease (SD)	2
Progressive Disease (PD)	-
Not evaluable (NE)	-
Overall Response Rate	**75%**
Clinical Benefit Rate	**100%**

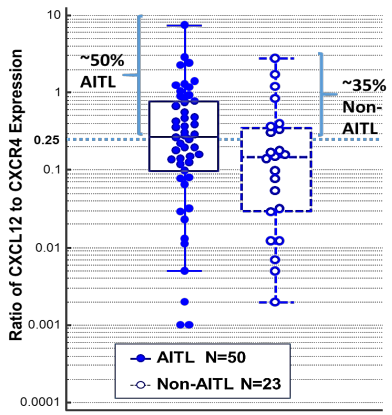
N= 32 AITL/NOS cases with response, NGS and RNA Seq data

Conclusions

- The AITL and wt CXCL12 3'UTR cohorts met pre-specified statistical hypotheses supporting proof-of-concept for tipifarnib in PTCL.
- Tipifarnib is active in AITL pts and in PTCL-NOS pts with wt CXCL12 3'UTR
  - AITL: 53% ORR (all subjects, PPS)
  - PTCL-NOS with wt CXCL12 3'UTR: 20% ORR (all subjects, PPS).
- KIR3DL2 and CXCL12 genotype provide robust tools for the selection/stratification of patients:
  - CXCL12 genotype may enrich for CXCL12 expression and tipifarnib activity, particularly in PTCL-NOS (86.7% Clinical Benefit Rate for PTCL-NOS patients with wt CXCL12 3'UTR).
  - KIR3DL2 C336R/Q383E mutations may enrich for low CXCL5 expression and anti-tumor activity in AITL (75% ORR, 50% CR rate).
  - Approximately 50% of AITL carry KIR3DL2 mutations and 70% of PTCL carry reference (wild type) CXCL12 3'UTR rs2839695 sequences.
- TEAEs were consistent with the known safety profile of tipifarnib.
  - Treatment with tipifarnib 300 mg bid days 1-21 every 28-days was generally well tolerated. The majority of Grade ≥ 3 TEAEs were hematological events managed with best supportive care.
- These results suggest that further evaluation of tipifarnib in biomarker defined subsets of PTCL and CTCL would be of interest.

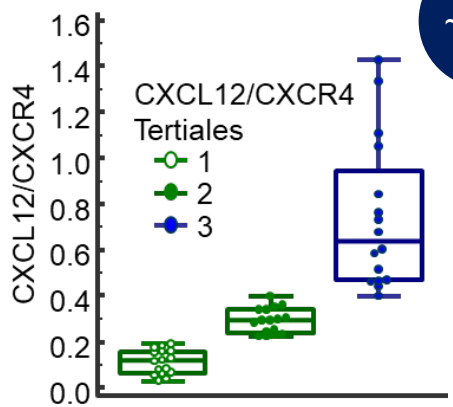
Tipifarnib Has Potential to Expand to Additional CXCL12-High Populations

PTCL / AITL[1] ~40%



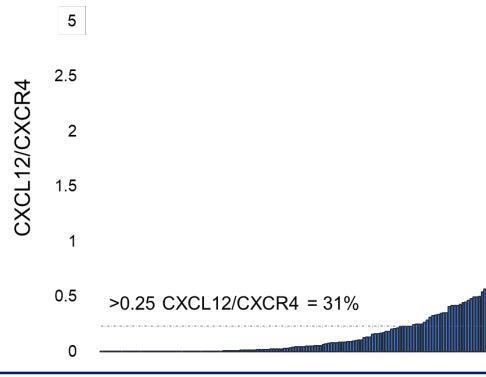
Est. Annual U.S. Incidence: 3,950[5]

DLBCL[2] ~33%



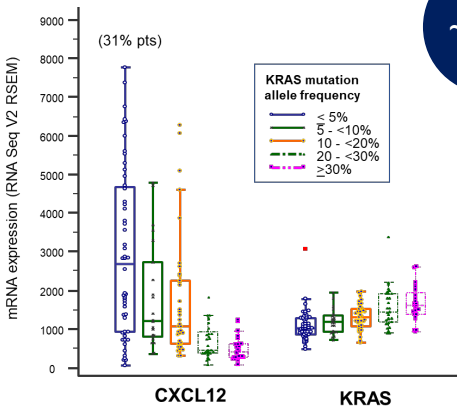
Est. Annual U.S. Incidence: 27,650[5]

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 | Incidience not adjusted for CXCL12-high subset

Key Takeaways

- POC achieved in AITL and wt CXCL12 3'UTR extension cohorts
- AITL and related lymphomas represent approximately one-third of PTCL cases
- KIR3DL2 and CXCL12 genotype provide additional robust tools for the selection/stratification of PTCL patients
- Approximately 50% of AITL carries KIR3DL2 mutations and 70% of PTCL carries reference (wild type) CXCL12 3'UTR rs2839695 sequences
- Company believes results support multiple potential pathways to registration in AITL/PTCL and plans to seek regulatory feedback
- Potential for CXCL12 variations/mutations to predict clinical activity in additional indications, including DLBCL, AML and pancreatic cancer

Anticipated Milestones & Financial Highlights

Program		Milestones	Status
Tipifarnib Farnesyl Transferase Inhibitor	HRAS Mutant Indications	Initiation of registration-direct1ed trial in HNSCC	✓
		Additional data from Phase 2 trial in HNSCC and other SCCs	2H 2019
	CXCL12 Pathway Indications	Patents for tipifarnib in AITL and CXCL12+ PTCL/AML	✓
		Proof-of-concept in AITL	✓
		Positive Phase 2 trial in PTCL	✓
		Additional data from Phase 2 trial in CMML	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		FDA clearance of IND application	✓
		Initiation of Phase 1 trial	Mid-2019

Financial Highlights	
	Nasdaq: KURA
	Shares outstanding: 38.2M basic, 4.3M options*
	Cash, cash equivalents and short-term investments: $165.5M*

* As of March 31, 2019

KURA ONCOLOGY

Developing Precision Medicines for the Treatment of Cancer