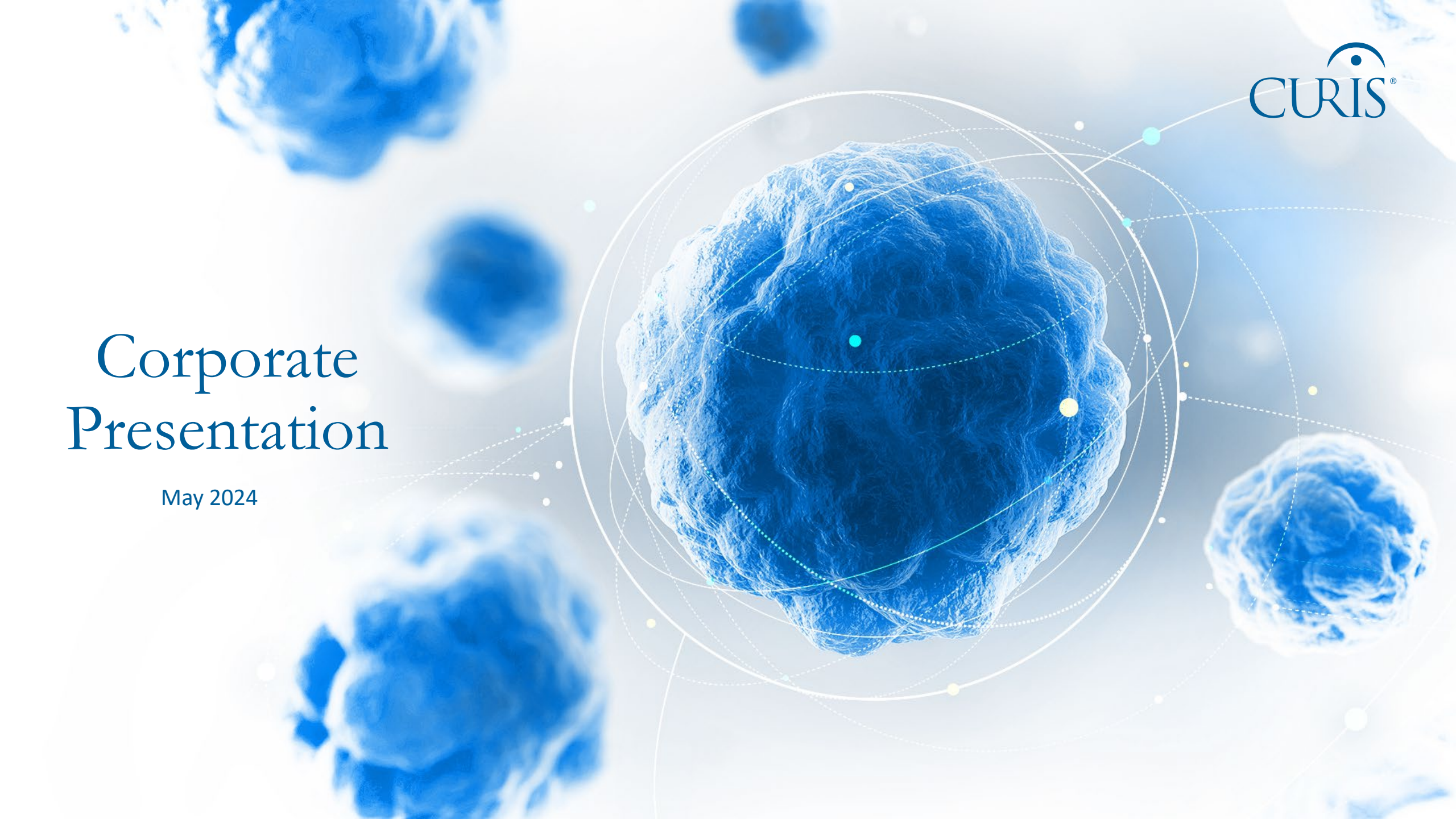


Corporate Presentation

May 2024

Cautionary note regarding forward looking statements and disclaimers

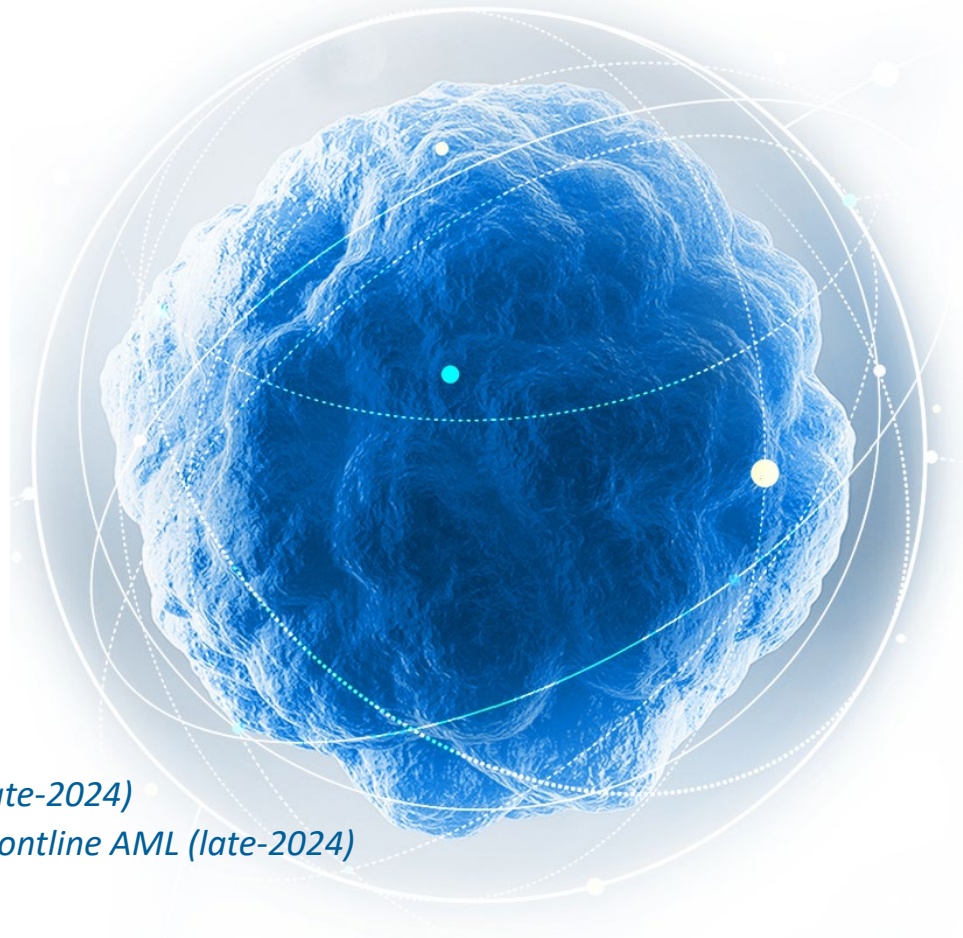
This presentation contains certain forward-looking statements about Curis, Inc. (“we,” “us,” or the “Company”) within the meaning of the Private Securities Litigation Reform Act of 1995, as amended. Words such as “expect(s),” “believe(s),” “will,” “may,” “anticipate(s),” “focus(es),” “plans,” “mission,” “strategy,” “potential,” “estimate(s)”, “opportunity,” "intend," "project," "seek," "should," "would" and similar expressions are intended to identify forward-looking statements. Forward-looking statements are statements that are not historical facts, reflect management’s expectations as of the date of this presentation, and involve important risks and uncertainties. Forward-looking statements herein include, but are not limited to, statements with respect to the timing and results of future clinical and pre-clinical milestones; the timing of future preclinical studies and clinical trials and results of these studies and trials; the clinical and therapeutic potential of our drug candidates; our cash runway; the proposed focus on emavusertib and management’s ability to successfully achieve its goals. These forward-looking statements are based on our current expectations and may differ materially from actual results due to a variety of important factors including, without limitation, risks relating to: whether and when the U.S. Food and Drug Administration (the “FDA”) may take further regulatory action with regard to our trials, whether any of our drug candidates will advance further in the clinical development process and whether and when, if at all, they will receive approval from the FDA or equivalent foreign regulatory agencies; whether historical preclinical results will be predictive of future clinical trial results; whether historical clinical trial results will be predictive of future trial results; whether any of our drug candidate development efforts will be successful; whether any of our drug candidates will be successfully marketed if approved; our ability to achieve the benefits contemplated by our collaboration agreements; management’s ability to successfully achieve its goals; the sufficiency of our cash resources; our ability to raise additional capital to fund our operations on terms acceptable to us and the use of proceeds of any offering of securities or other financing; general economic conditions; competition; and the other risk factors contained in our periodic reports filed with the Securities and Exchange Commission, including the Company's Annual Report on Form 10-K for the fiscal year ended December 31, 2023, which are available on the SEC website at www.sec.gov. You are cautioned not to place undue reliance on these forward-looking statements that speak only as of the date hereof, and we do not undertake any obligation to revise and disseminate forward-looking statements to reflect events or circumstances after the date hereof, or to reflect the occurrence of or non-occurrence of any events, except as required by law.

This presentation includes statistical and other industry and market data that we obtained from industry publications and research, surveys, and studies conducted by third parties as well as our own estimates. All of the market data used in this presentation involves a number of assumptions and limitations, and you are cautioned not to give undue weight to such data. Industry publications and third-party research, surveys, and studies generally indicate that their information has been obtained from sources believed to be reliable, although they do not guarantee the accuracy or completeness of such information. Our estimates of the potential market opportunities for our product candidates include several key assumptions based on our industry knowledge, industry publications, third-party research, and other surveys, which may be based on a small sample size and may fail to accurately reflect market opportunities. While we believe that our internal assumptions are reasonable, no independent source has verified such assumptions.

Curis is developing the first-in-class IRAK4 inhibitor

Curis Overview

- *Originator of the first Hedgehog inhibitor, licensed to Roche/Genentech , marketed as vismodegib (Erivedge®)*
- *Emavusertib, the most advanced IRAK4 inhibitor is in Phase I/II clinical studies in leukemia and lymphoma*
- *Initial Phase I/II clinical data demonstrated single-agent anti-cancer activity in AML and NHL, with potential for broader application in combination with standard of care*
- *Upcoming Anticipated Milestones:*
  - *POC combination data in R/R PCNSL (late-2024)*
  - *Initial data for triplet combination in frontline AML (late-2024)*



Demonstrated single-agent activity and safety in NHL and AML

Demonstrated synergy with BTKi, HMA, BCL2i

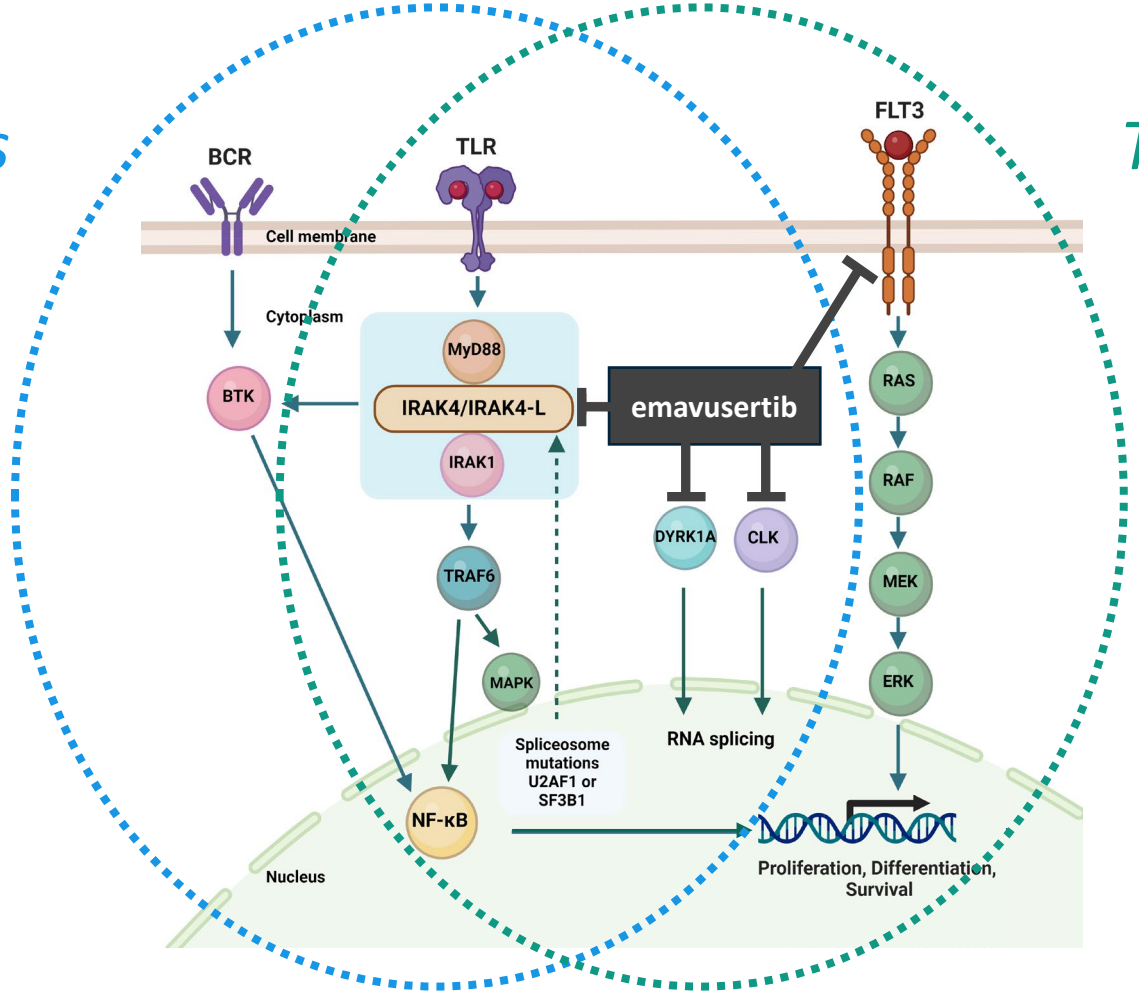
Broad opportunity in heme and solid malignancies

Abbreviations: Interleukin 1 Receptor Associated Kinase 4 (IRAK4), Acute Myeloid Leukemia (AML), Non-Hodgkin Lymphoma (NHL), Primary Central Nervous System Lymphoma (PCNSL), FLT3 mutation (FLT3m), Bruton's Tyrosine Kinase inhibitors (BTKi), Hypomethylating agents (HMA) and B-Cell Lymphoma 2 inhibitor (BCL2i)

BCR, TLR, and FLT3 Pathways are activated in heme malignancies

In NHL, BCR and TLR pathways drive NF-κB (and lymphoma)

emavusertib + BTKi combo binds to IRAK4 and BTK, blocking TLR and BCR pathways



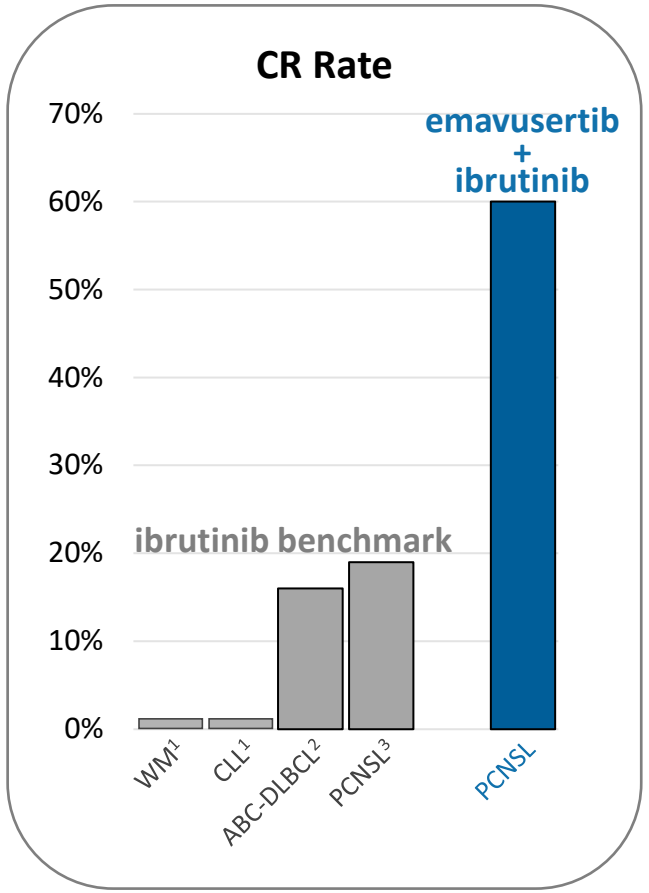
In AML, TLR and FLT3 pathways drive NF-κB (and leukemia)

emavusertib binds to IRAK4 and FLT3, blocking TLR and FLT3 pathways

Bennett Curr Opin Hematol 2022, Grafone Oncol Rev 2012, Kelly J Exp Med 2015, Wang Cancer Cell 2023

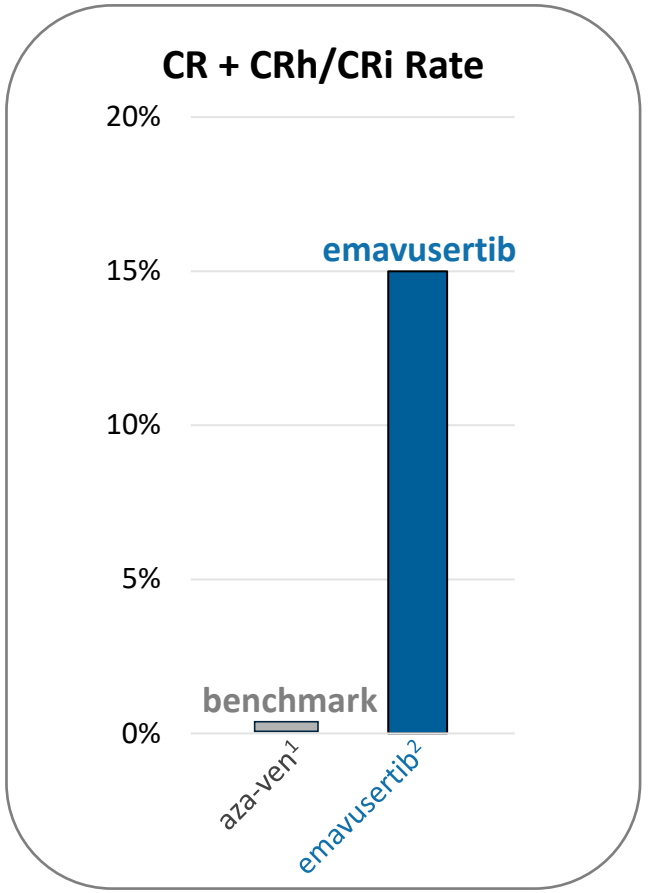
Summary Snapshot of Clinical Data

R/R NHL



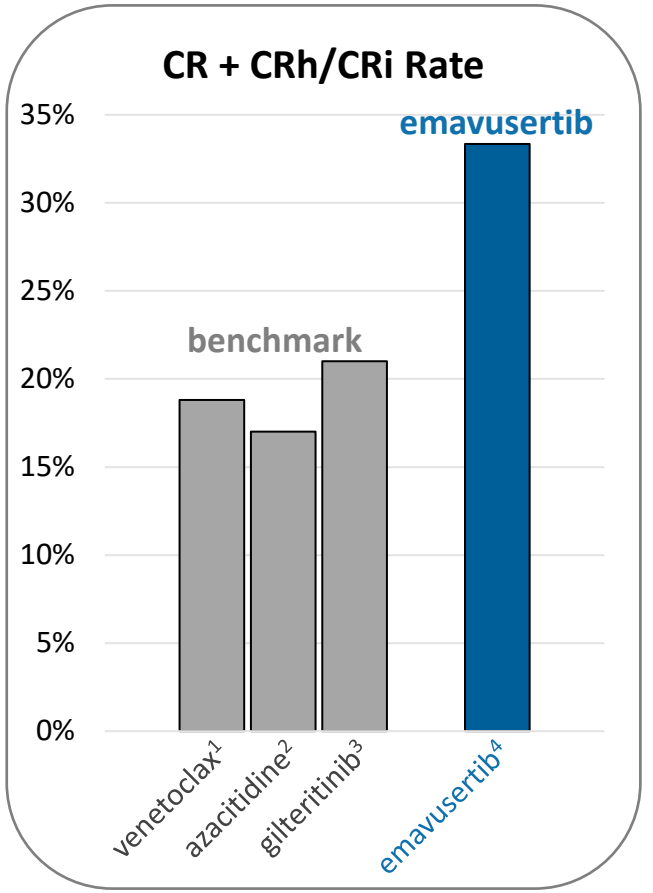
1) ibrutinib USPI, 2) Wilson Nat Med 2015, 3) Soussain Euro J Cancer 2019

R/R SFm AML



1) Aldoss Am J Hem 2019 [CR/CRi]; 2) emavusertib [CR/CRh]

R/R FLT3m AML



1) Konopleva Cancer Discov 2016 [CR/CRi], 2) Itzykson Leuk Res 2015 [CR/CRi], 3) gilteritinib USPI [CR/CRh]; 4) emavusertib [CR/CRh]

The comparisons presented in the figures above represent cross-trial comparisons and do not involve data from a head-to-head clinical trials

Abbreviations: Waldenström's Macroglobulinemia (WM), Chronic lymphocytic leukemia (CLL), Activated B Cell-like - Diffuse Large B-Cell Lymphoma (ABC-DLBCL), azacitidine (aza), venetoclax (ven)

Market Opportunity

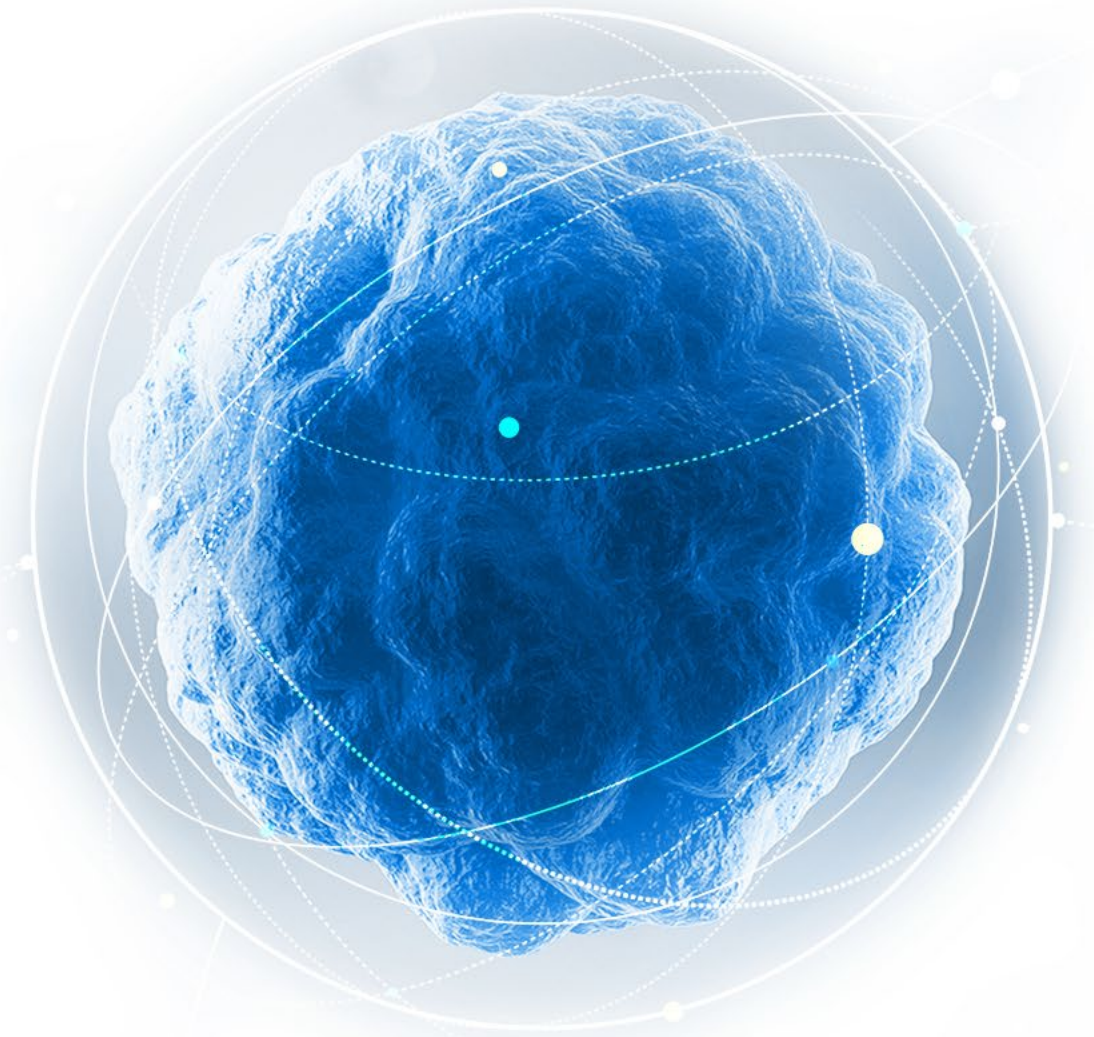
Significant market opportunities in current development programs

	PCNSL	FLT3m	SFm	AML
US Incidence per 100K	**0.5[1]**	**1.3[2]**	**0.6[3]**	**4.2[4]**
	Newly Diagnosed Patients Per Year			
US	1,700[1]	6,000[2]	2,700[3]	20,000[4]
Big 5 Europe/Canada	1,800[1]	5,200[5]	2,300[3]	17,000[5]
Japan/China	7,700[1]	12,700[5]	5,600[3]	41,200[5]
Total	**11,200**	**23,900**	**10,600**	**78,200**

1 – Derived from incident rate in Lv Ther Adv Hematol 2022 and 2022 country population [data.worldbank.org]
2 – Derived from total AML cases (see footnote 4); FLT3m represents 30% of newly diagnosed AML cases [Daver Leukemia 2019]
3 – Derived from total AML cases (see footnote 4); SFm represents 50% of SAML [Lachowiez Blood Adv 2021] & sAML is 27% of AML [Martinez-Cuadrón Blood Adv 2022]
4 – Vakiti Acute Myeloid Leukemia 2023 [www.ncbi.nlm.nih.gov]
5 – Clarivate DRG, March 2024

Abbreviations: Spliceosome mutation (SFm)

NHL Biology
IRAK4 is a novel target



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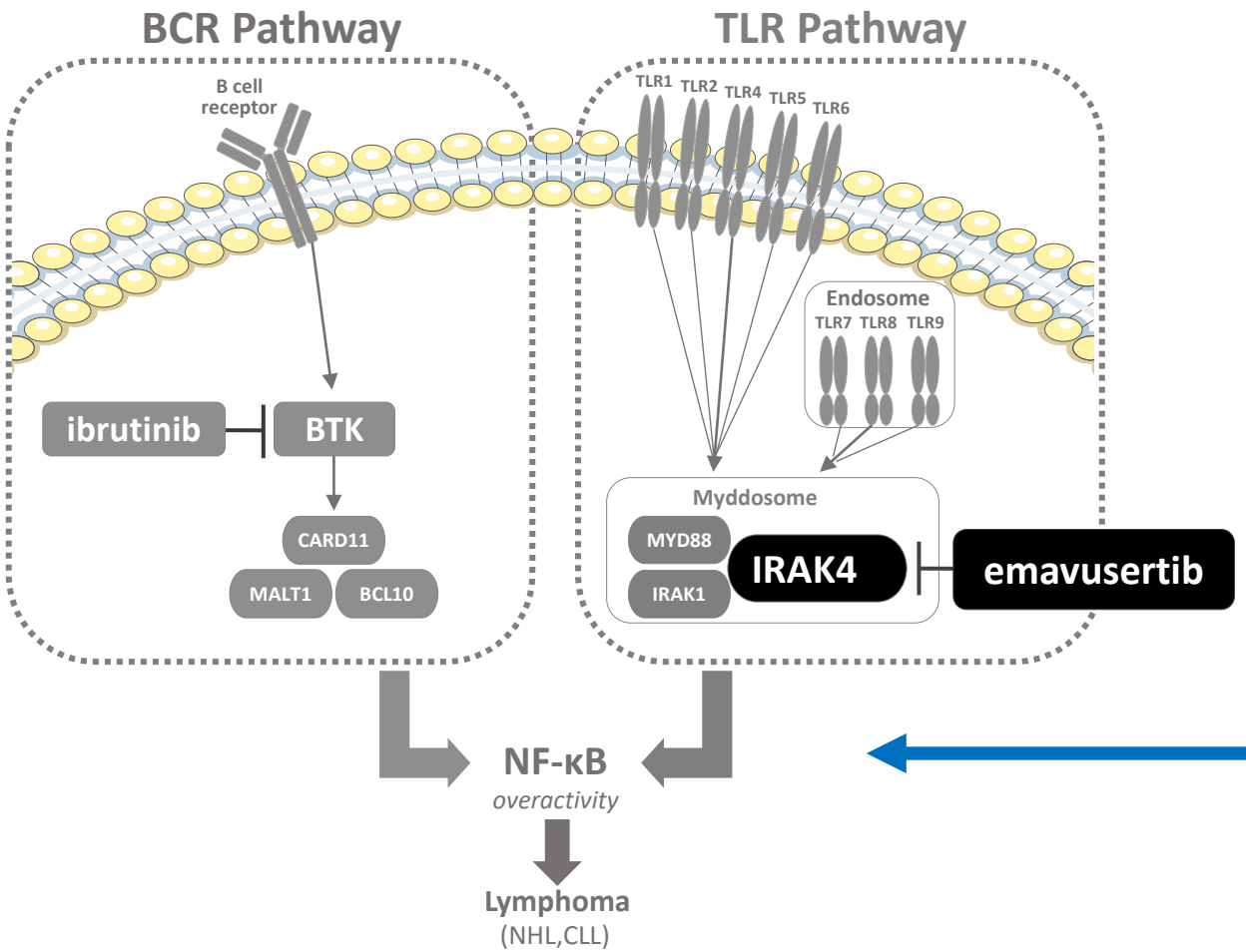
Mechanism in Lymphoma



Two pathways drive NF-κB
(which drives B-cell lymphomas)

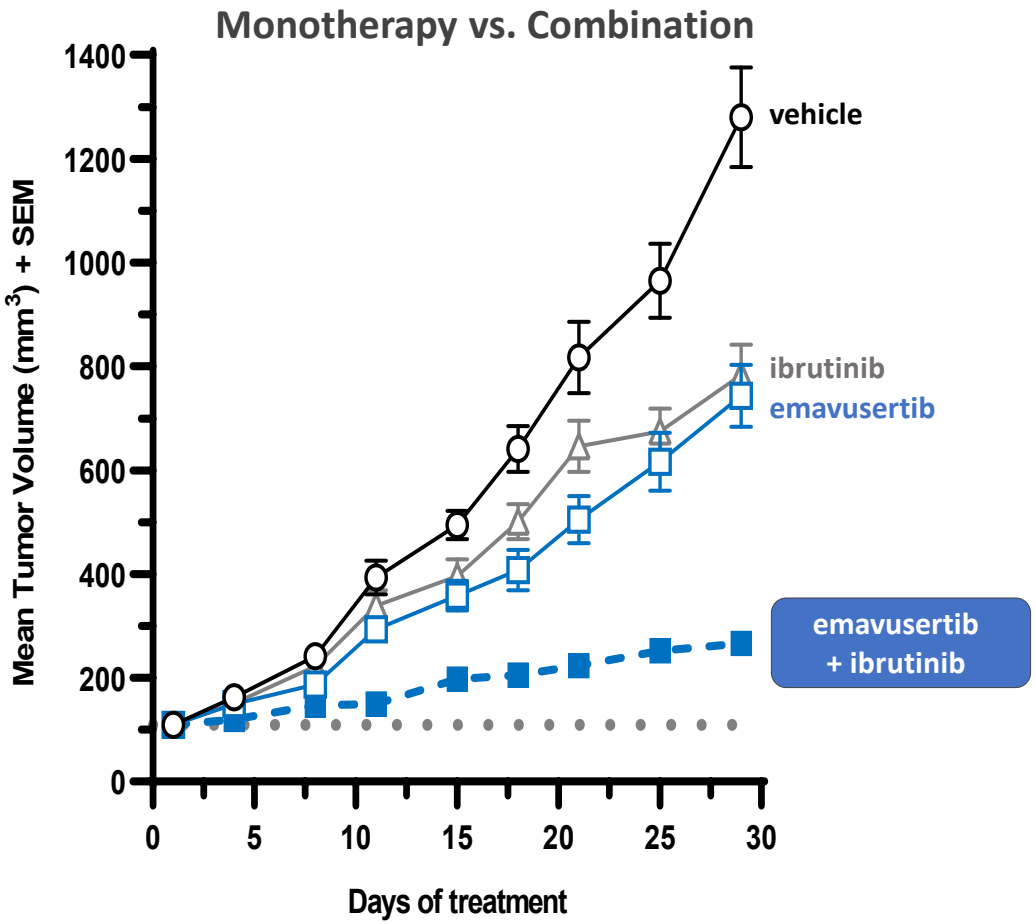
IRAK4i + BTKi
enables dual blockade of NF-κB

IRAK4i is synergistic with BTKi (ibrutinib)

emavusertib + ibrutinib

blocking both BCR and TLR pathways has been demonstrated to be better than blocking either one alone

- IRAK4 inhibition synergizes with BTK inhibition to promote killing of **ABC-DLBCL**[1]
- Concurrent treatment with IRAKi and ibrutinib was significantly more potent in patient **CLL** cells than either drug alone[2]
- "Our data suggest IRAK4 as a novel treatment target for **CLL**; inhibition of IRAK4 blocks survival and proliferation of CLL cells" [3]



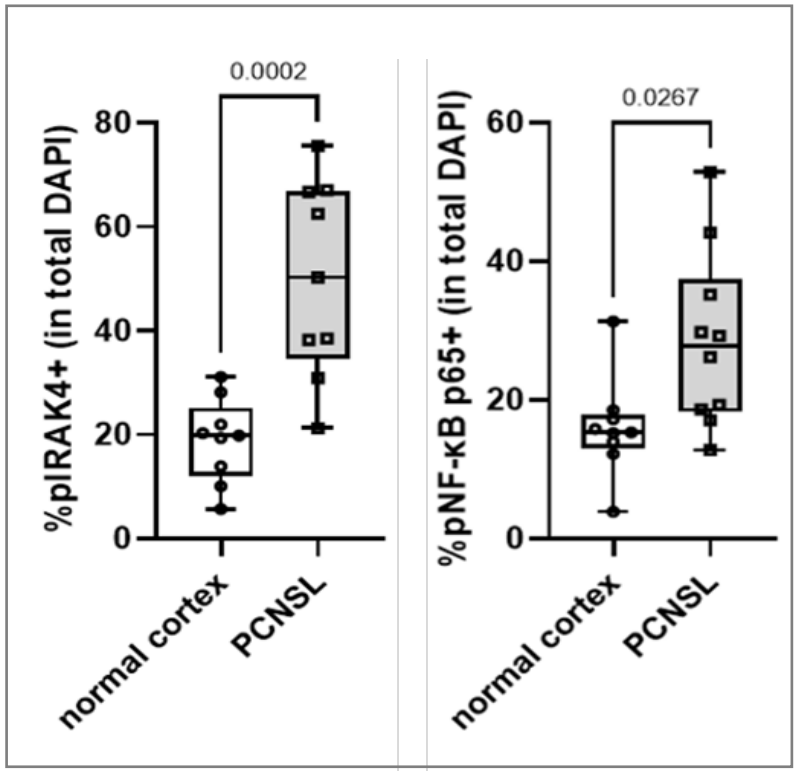
Preclinical data in OCI-Ly10 model from Booher et al. **Waldenström** Roadmap Symposium 2019

1) Kelly J Exp Med 2015, 2) Dadashian Ca Res 2019, 3) Giménez Leukemia 2020

Preclinical support for PCNSL

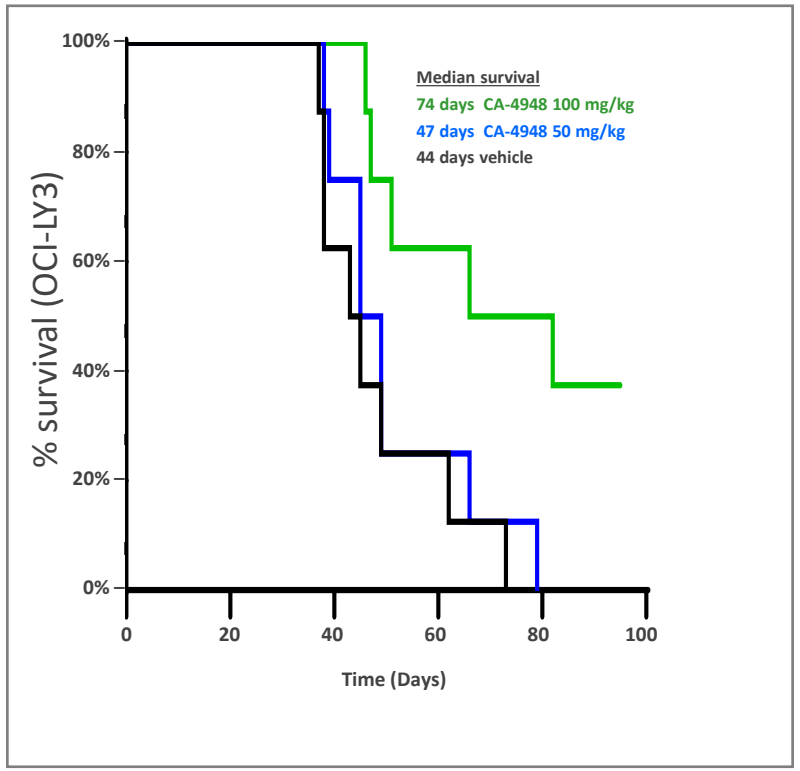
IRAK4 is highly expressed in PCNSL

IRAK4 Expression in PCNSL



emavusertib demonstrated extended survival in PCNSL models

% survival in PCNSL

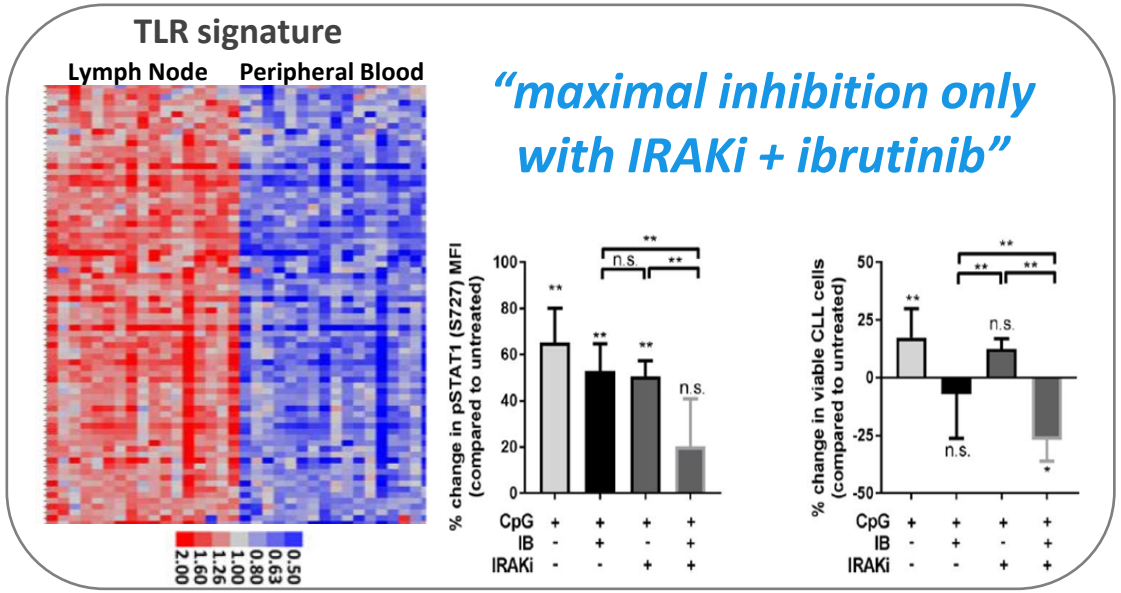


Figures from von Roemeling NHCC poster 2023

Additional NHL indications of interest

CLL

- TLR signaling is highly activated in lymph node–resident CLL cells[1]



Waldenström's Macroglobulinemia

- MYD88 and CXCR4 mutations activate NF-κB through the TLR pathway
- Recurring mutations in innate immune signaling and TLR/MYD88 pathway regulators are characteristic of ibrutinib-resistant WM patients[2]

ABC-DLBCL

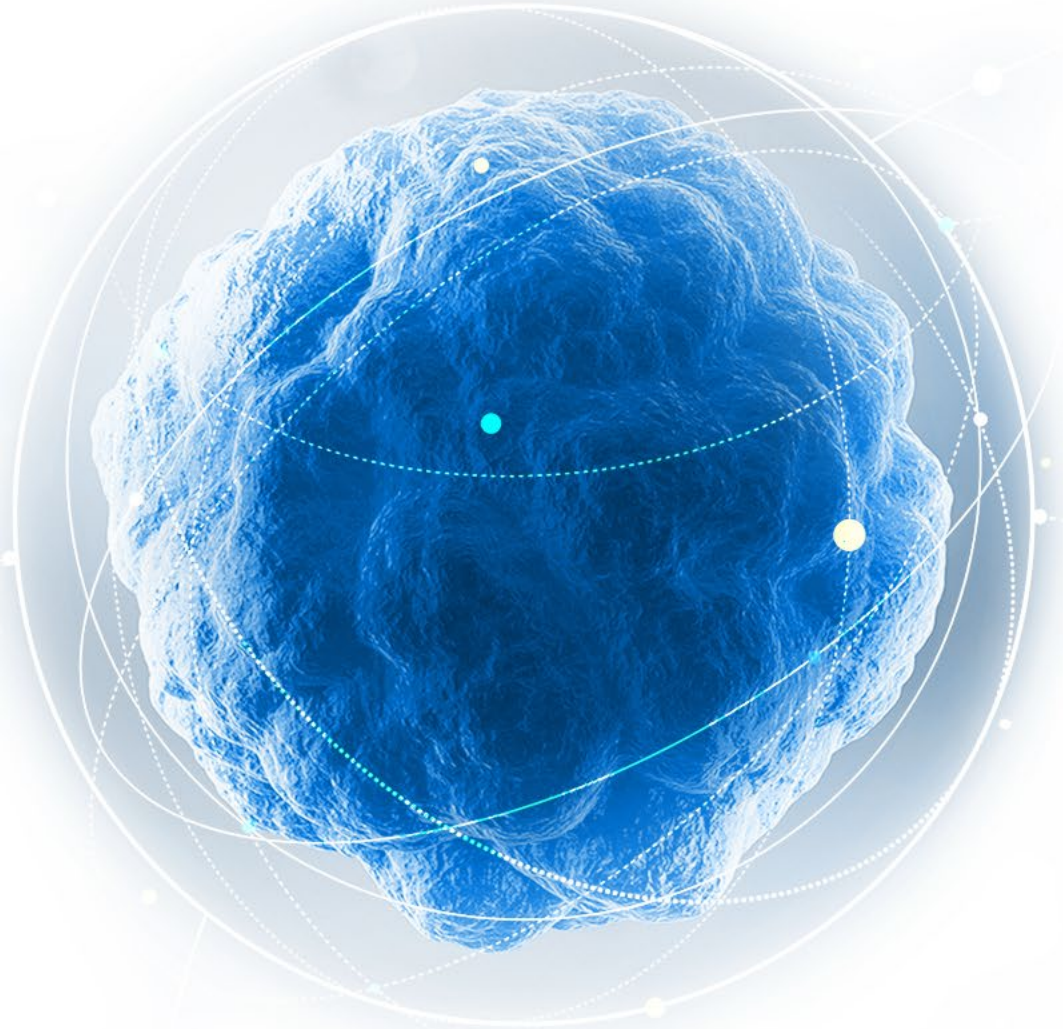
- Mutations in MYD88 activate NF-κB through the TLR pathway

MCL

- TLR signaling is highly active in MCL, inducing proliferation and immune evasion in a MYD88-dependent fashion[3]

1) Dadashian Tumor Biol and Immunol 2019, 2) Jiménez Brit Jour Haem 2020, 3) Wang Cancer 2013

Emavusertib
in Lymphoma



CURIS®

Strategy in Lymphoma

1

Demonstrate safety

19 patients[1] treated in TakeAim Lymphoma Ph 1b study, acceptable safety profile established, no overlapping dose-limiting toxicity with ibrutinibn

2

Demonstrate single-agent activity

Single-agent activity demonstrated, with patients remaining on study up to 4 years

3

Pursue fastest path to 1st label in R/R patients

Identify orphan indication with clear unmet need that is addressable with emavusertib's novel mechanism of action

4

Pursue partnership to expand across NHL

Significant resources will be required to execute large clinical studies across multiple NHL subtypes and prepare for potential commercial launch

1 – Data cutoff October 12, 2023

Emavusertib safety profile in Lymphoma[1]

- 19 patients treated with emavusertib in combination with ibrutinib in multiple NHL subtypes
- Shown to be well tolerated with an acceptable safety profile
  - No DLTs observed at 100mg or 200mg
  - 2 reversible DLTs observed at 300mg (stomatitis and syncope)
- Emavusertib crosses the BBB and no dose-limiting CNS toxicities have been observed
- No dose-limiting myelosuppression has been observed

Grade 3+ TRAE in >1 Patient	100 mg BID+IBR (N=2)	200 mg BID+IBR (N=10)	300 mg BID+IBR (N=7)	Total (N=19)
	n (%)	n (%)	n (%)	n (%)
# patients having grade 3+ TRAEs	1 (50)	7 (70)	6 (86)	14 (74)
Platelet count decreased		2 (20)	1 (14)	3 (16)
Alanine aminotransferase increased		1 (10)	1 (14)	2 (11)
Aspartate aminotransferase increased		1 (10)	1 (14)	2 (11)
Fatigue		1 (10)	1 (14)	2 (11)
Hyponatraemia		2 (20)		2 (11)
Lipase increased	1 (50)	1 (10)		2 (11)

1 – Curis Emavusertib TakeAim Lymphoma ASH 2023 poster

Abbreviation: Treatment Related Adverse Event (TRAE), ibrutinib (IBR), Dose Limiting Toxicity (DLT), Blood Brain Barrier (BBB), Central Nervous System (CNS), twice daily (BID)

Strategy in Lymphoma

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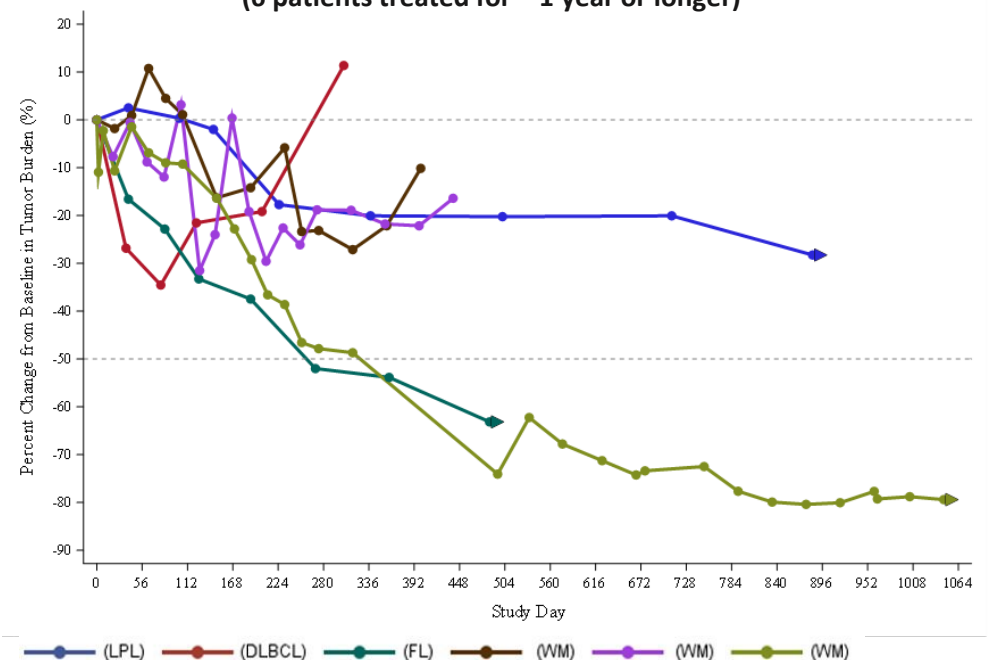
Significant resources will be required to execute large clinical studies across multiple NHL subtypes and prepare for potential commercial launch

1 – Data cutoff October 12, 2023

Single-agent activity demonstrated with emavusertib

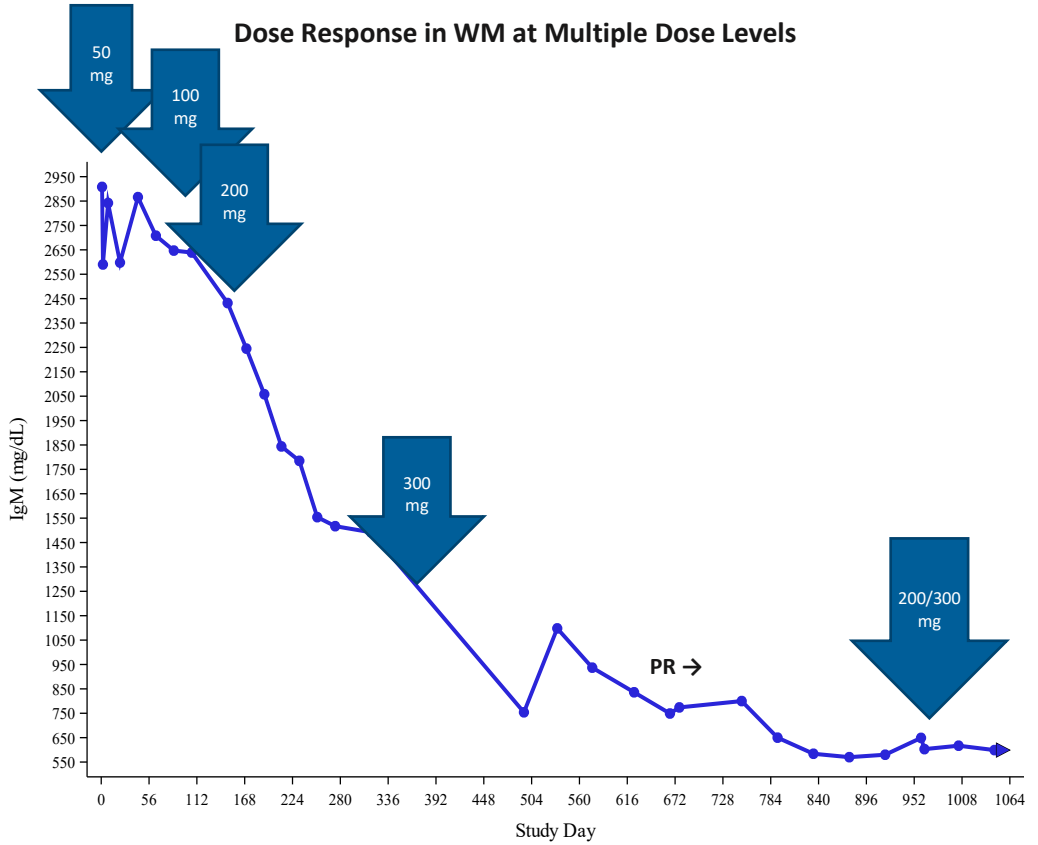
Percent Change in Tumor Burden from Baseline
(6 patients treated for ~ 1 year or longer)



Individual Subject: Diagnosis

IgM values were used as the measure for tumor burden for WM/LPL patients; sum of product of diameters of target lesions were used as the measure for other lymphoma types.

Dose Response in WM at Multiple Dose Levels



2022 IWWM Conference Presentation

Strategy in Lymphoma

1

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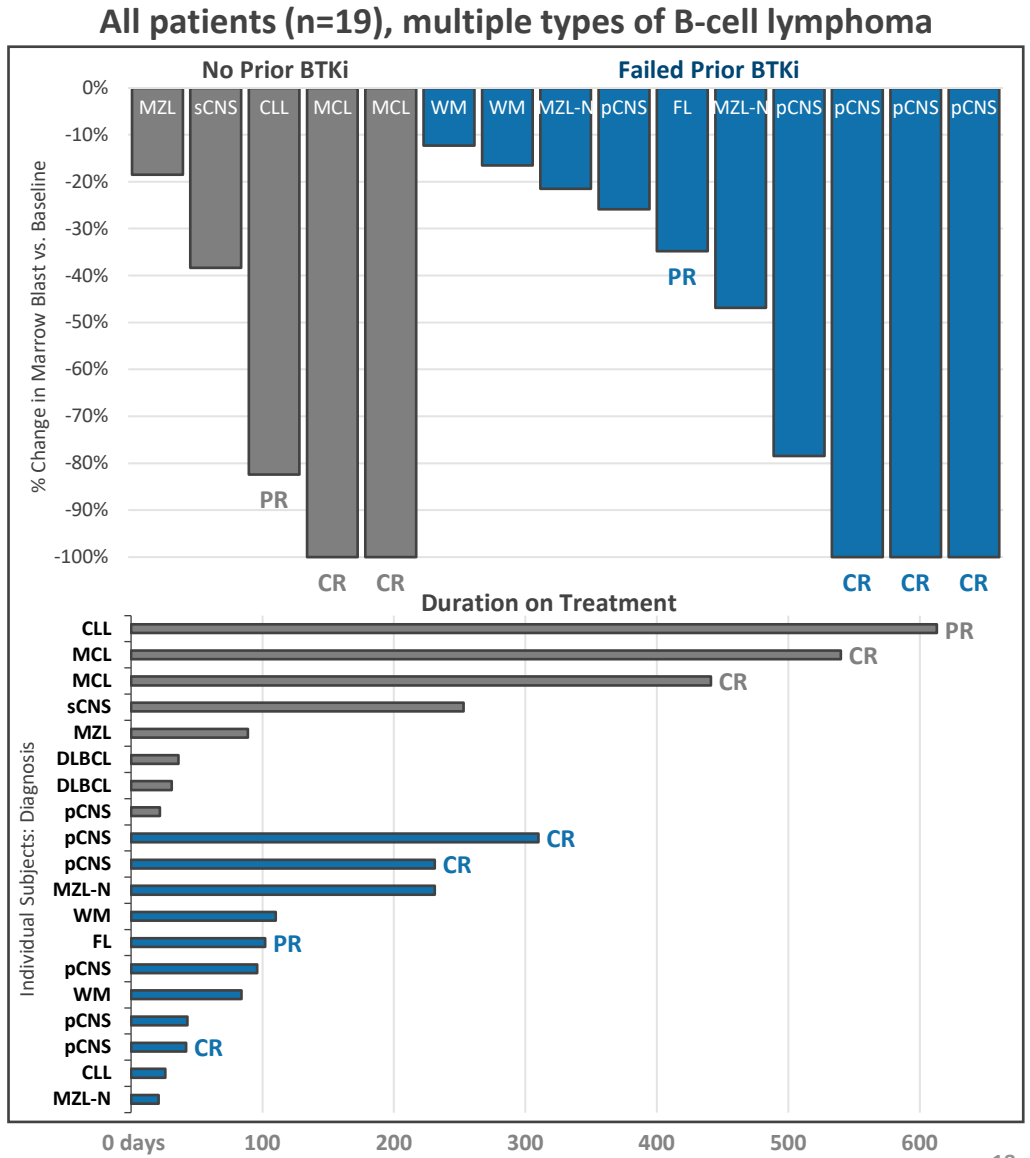
Pursue partnership to expand across NHL

Significant resources will be required to execute large clinical studies across multiple NHL subtypes and prepare for potential commercial launch

1 – Data cutoff October 12, 2023

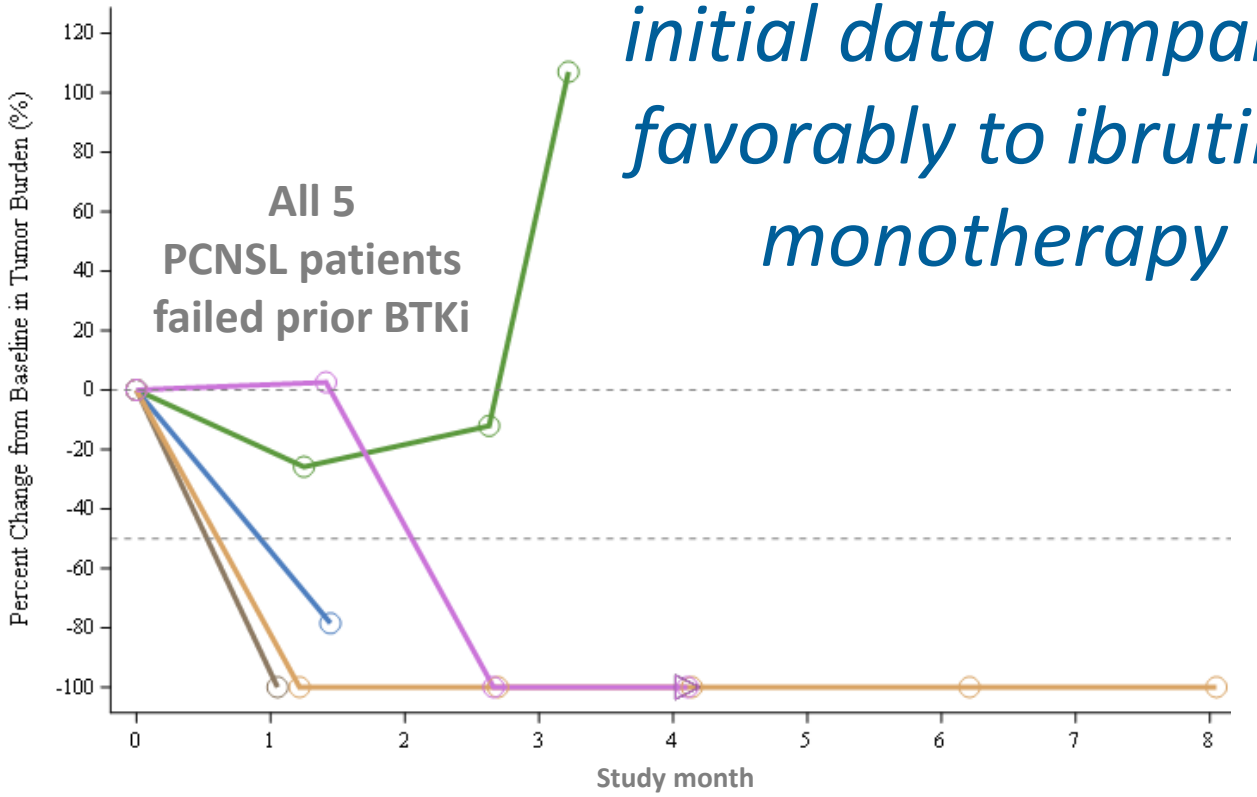
Combination data released at ASH 2023

- Heavily pre-treated patients (**1-10 prior lines**)
- Responses achieved in patients **who failed prior BTKi**
- 15 of 19 patients saw a reduction in tumor burden, including 5 CRs
- Ongoing study with median treatment duration of 96 days (range 21-613 days)

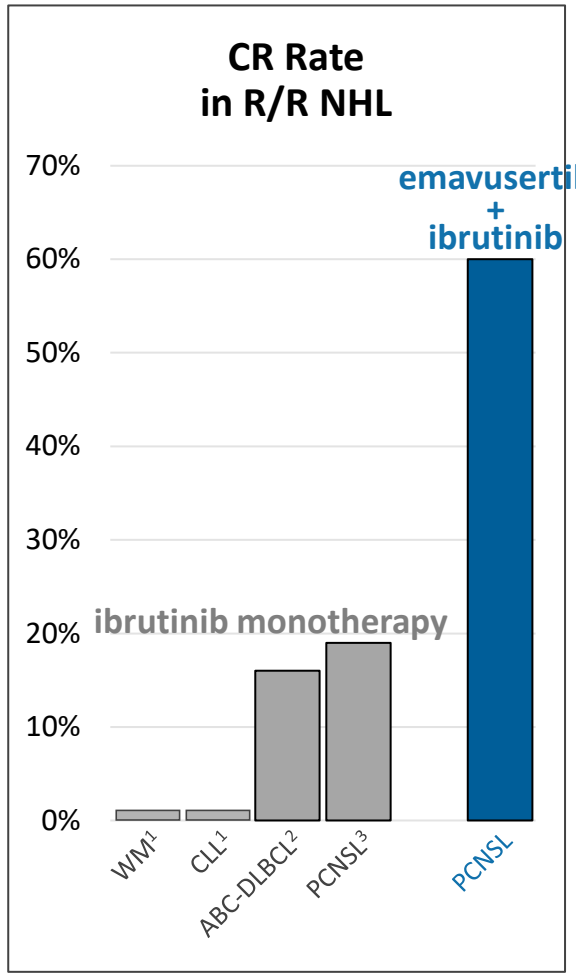


Abbreviations: Complete Response (CR), Partial Response (PR)

PCNSL selected for lead indication in NHL



initial data compared favorably to ibrutinib monotherapy

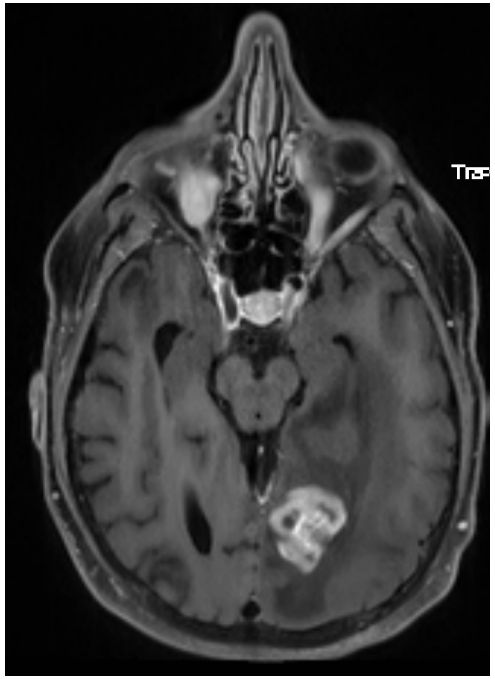


PCNSL
CR Rate
3 of 5 (60%)

1) ibrutinib USPI, 2) Wilson Nat Med 2015, 3) Soussain Euro J Cancer 2019

The comparisons presented in the figure above represent cross-trial comparisons and do not involve data from a head-to-head clinical trials

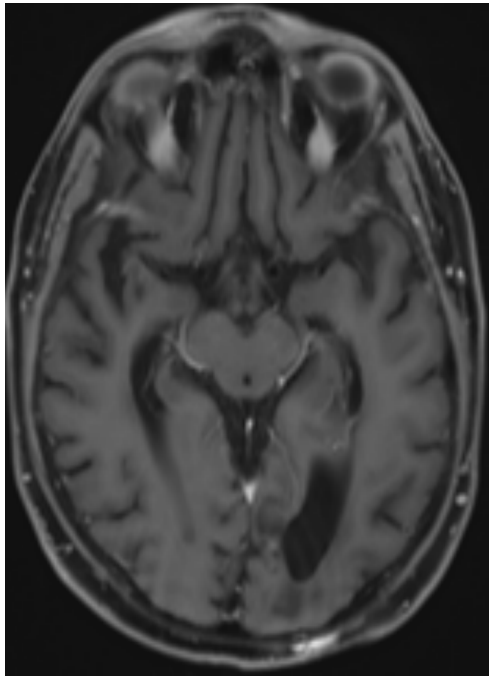
PCNSL patient who achieved CR



Nov 2022
relapsed on ibrutinib

Feb 2023
enrolled in study

May 2023
achieved CR



June 2023
CR

patient remains in CR as of April 2024

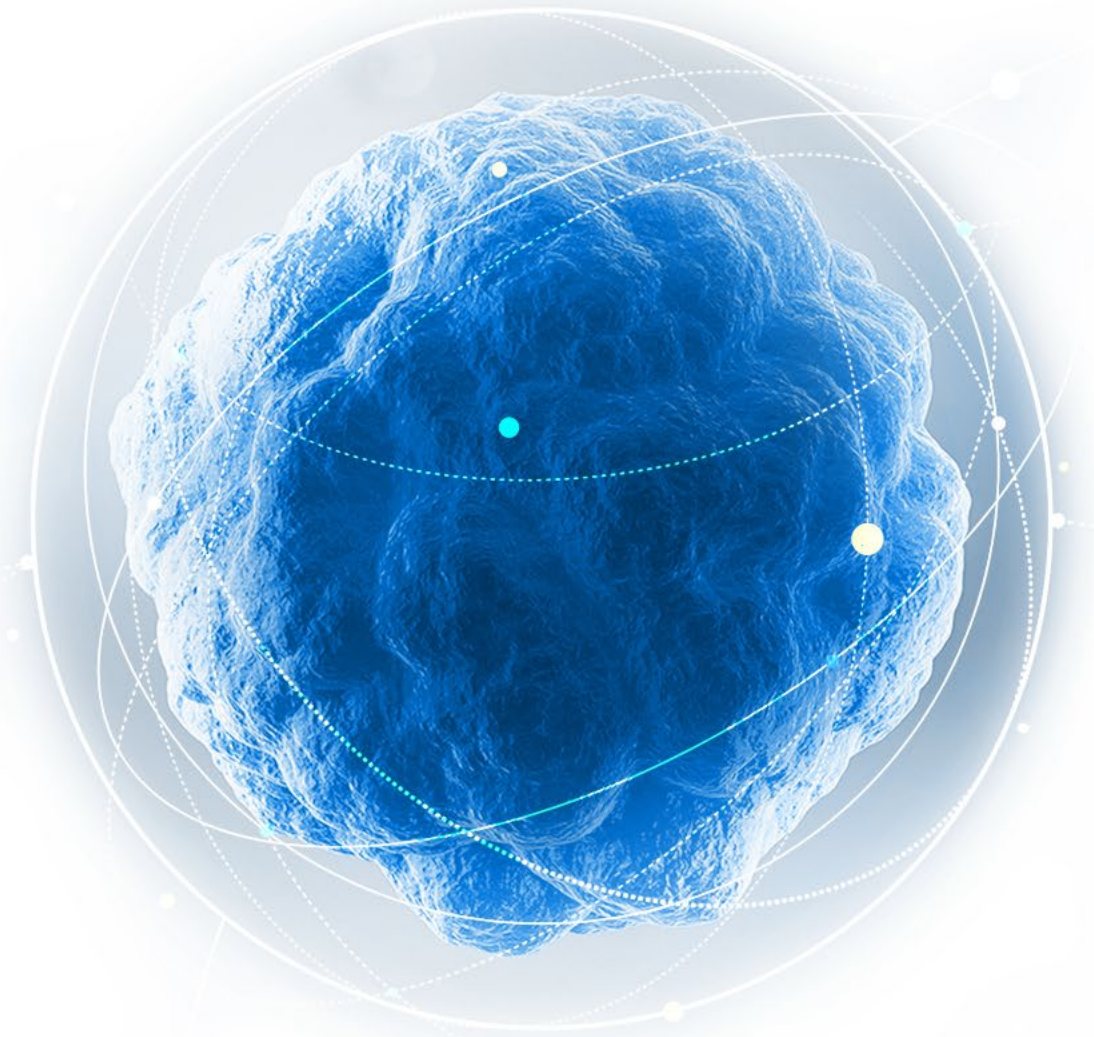
Previous treatments:
MATRIX, HD BCNU/thiotepa-ASCT, whole brain radiation, ibrutinib

Summary in Lymphoma

TakeAim Lymphoma

- Anti-cancer activity in R/R PCNSL exceeded benchmark response criteria with potential for best-in-class therapy
- Continue enrolling in combination study of emavusertib + ibrutinib in R/R PCNSL
- Prioritize additional lymphoma indications that could benefit from dual-blockade of NF-κB (blocking TLR pathway with emavusertib and blocking BCR pathway with BTKi)

AML Biology
IRAK4 is a novel target



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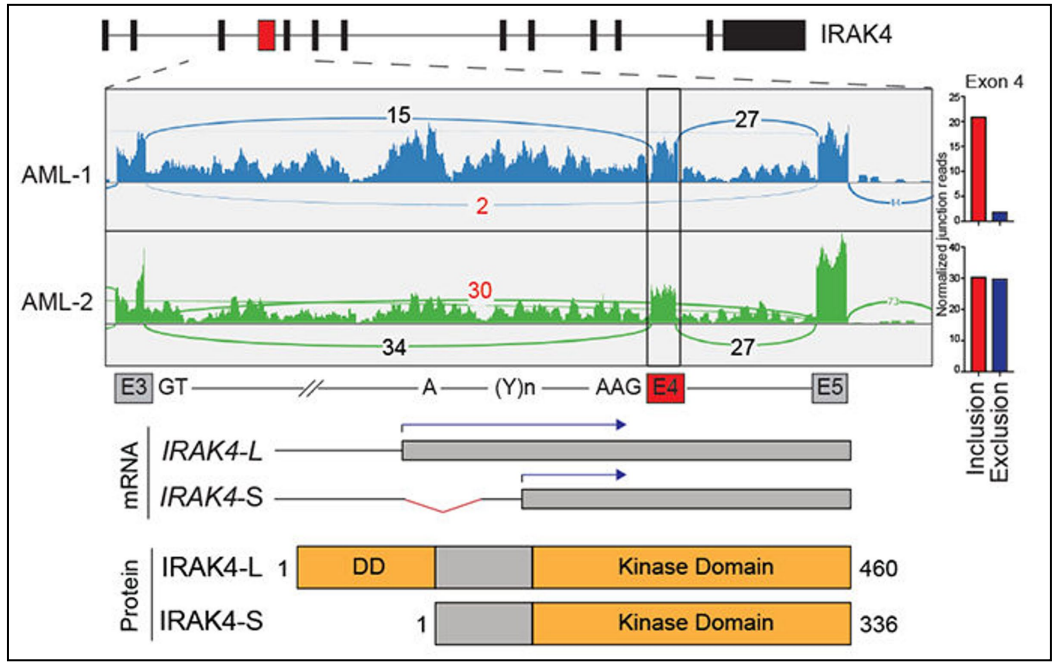
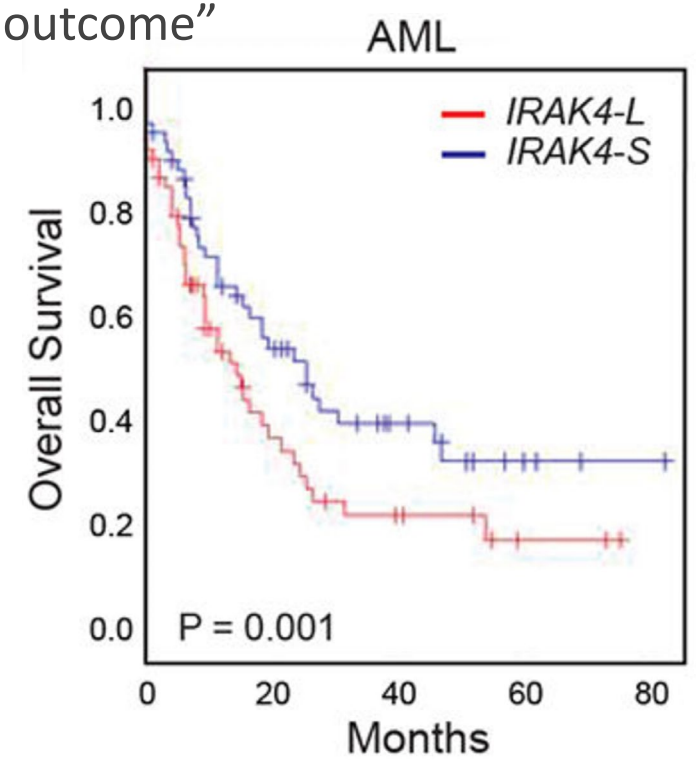
Among innate immune and NF-κB signaling genes driving AML, IRAK-L is highly significant



Exon architecture of IRAK4 and protein domains.
Sashimi plots represent junction reads in representative AML samples.

"The magnitude of **IRAK4 isoform switching** was **highly significant** among AML samples and the inclusion of exon 4 alone correlated with worse outcome"

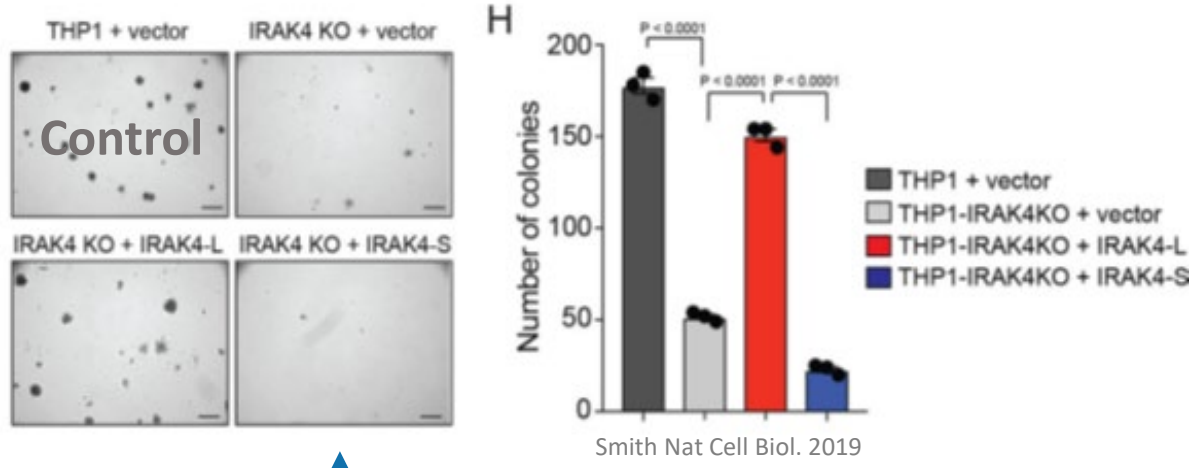


Smith Nat Cell Biol. 2019

Preclinical experiments demonstrated IRAK4-L to be a powerful driver of AML

Knocking out IRAK4 stops leukemic activity

(similar findings using CRISPR/CAS9)



Smith Nat Cell Biol. 2019

adding back IRAK4-L restarts activity

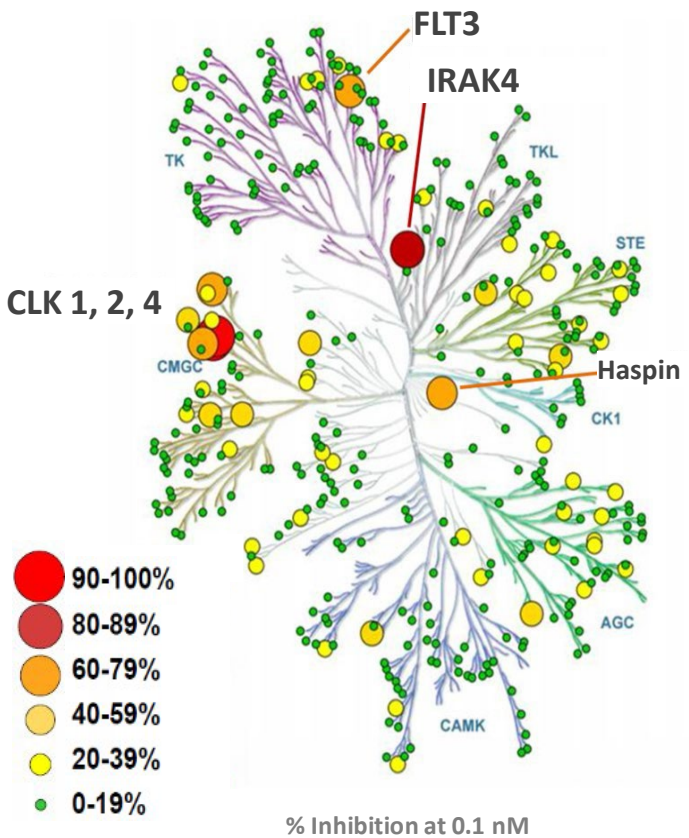
adding back IRAK4-S has no effect

Smith Nat Cell Biol. 2019

Emavusertib specific design

emavusertib was selected by NCI for its sponsored research and clinical studies of IRAK4

Emavusertib
Kinase Interaction Map



% Inhibition at 0.1 nM

Illustration reproduced courtesy of Cell Signaling Technology

Emavusertib
Binding Affinity

Target	K_d nM
IRAK1	12,000
IRAK2	>20,000
IRAK3	8,500
IRAK4	**23**
DYRK1A	25
FLT3 WT	**31**
FLT3 (D835H)	**5**
FLT3 (D835V)	**44**
FLT3 (D835Y)	**3**
FLT3 (ITD)	**8**
FLT3 (K663Q)	**47**
FLT3 (N841I)	**16**
Haspin (GSG2)	32
CLK1	10
CLK2	20
CLK3	>20,000
CLK4	14
TrkA	130

DiscoverX Kinase Panel
(378 kinases screened)

binds tightly to IRAK4

engineered to include multiple other targets, including FLT3

Strategy in AML

1

Demonstrate safety

123 patients[1] treated in TakeAim Leukemia Ph 1/2 study, acceptable safety profile established

2

Demonstrate single-agent activity

Single-agent activity observed; next step is to confirm these initial findings in a larger number of patients

3

Pursue fastest path to 1st label in R/R patients

Address genetically-defined AML population with emavusertib's novel mechanism of action

4

Pursue frontline opportunity with combination

IRAK4-L is expressed in nearly all AML patients; preclinical "all comer" models suggest emavusertib is synergistic with azacitidine and venetoclax

5

Pursue partnership to maximize potential commercial opportunity

Significant resources will be required to execute a large clinical study and prepare for potential commercial launch

1 – Data cutoff February 26, 2024

Emavusertib safety profile in Leukemia[1]

- 123 patients treated with emavusertib in AML
- Shown to be well tolerated with an acceptable safety profile
- No dose-limiting myelosuppression has been observed

Grade 3+ TRAE > 1 patients	200 mg BID (N = 27)	300 mg BID (N = 78)	400 mg BID (N = 15)	500 mg BID (N = 3)	Total (N=123)
	n (%)	n (%)	n (%)	n (%)	n (%)
Number of patients having grade 3+ TRAEs	4 (14.8)	21 (26.9)	7 (46.7)	2 (66.7)	27 (27.6)
Blood creatine phosphokinase increased	0	6 (7.7)	0	0	6 (4.9)
Platelet count decreased	1 (3.7)	3 (3.8)	2 (13.3)	0	6 (4.9)
Rhabdomyolysis[2]	0	2 (2.6)	1 (6.7)	1 (33.3)	4 (3.3)
Anaemia	0	3 (3.8)	0	0	3 (2.4)
Aspartate aminotransferase increased	1 (3.7)	2 (2.6)	0	0	3 (2.4)

1 – Data as of February 26, 2024

Abbreviation: Treatment Related Adverse Event (TRAE), Upper Limit Normal (ULN)

2 - One patient with an event of Rhabdomyolysis met laboratory-defined criteria, defined as creatine phosphokinase > 10 × ULN with concurrent serum creatinine ≥ 1.5 × ULN. The remaining 3 patients experienced investigator-reported events of Rhabdomyolysis that did not meet laboratory-defined criteria

Strategy in AML

1

Demonstrate safety

123 patients[1] treated in TakeAim Leukemia Ph 1/2 study, acceptable safety profile established

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Single-agent activity observed; next step is to confirm these initial findings in a larger number of patients

3

Pursue fastest path to 1st label in R/R patients

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IRAK4-L is expressed in nearly all AML patients; preclinical "all comer" models suggest emavusertib is synergistic with azacitidine and venetoclax

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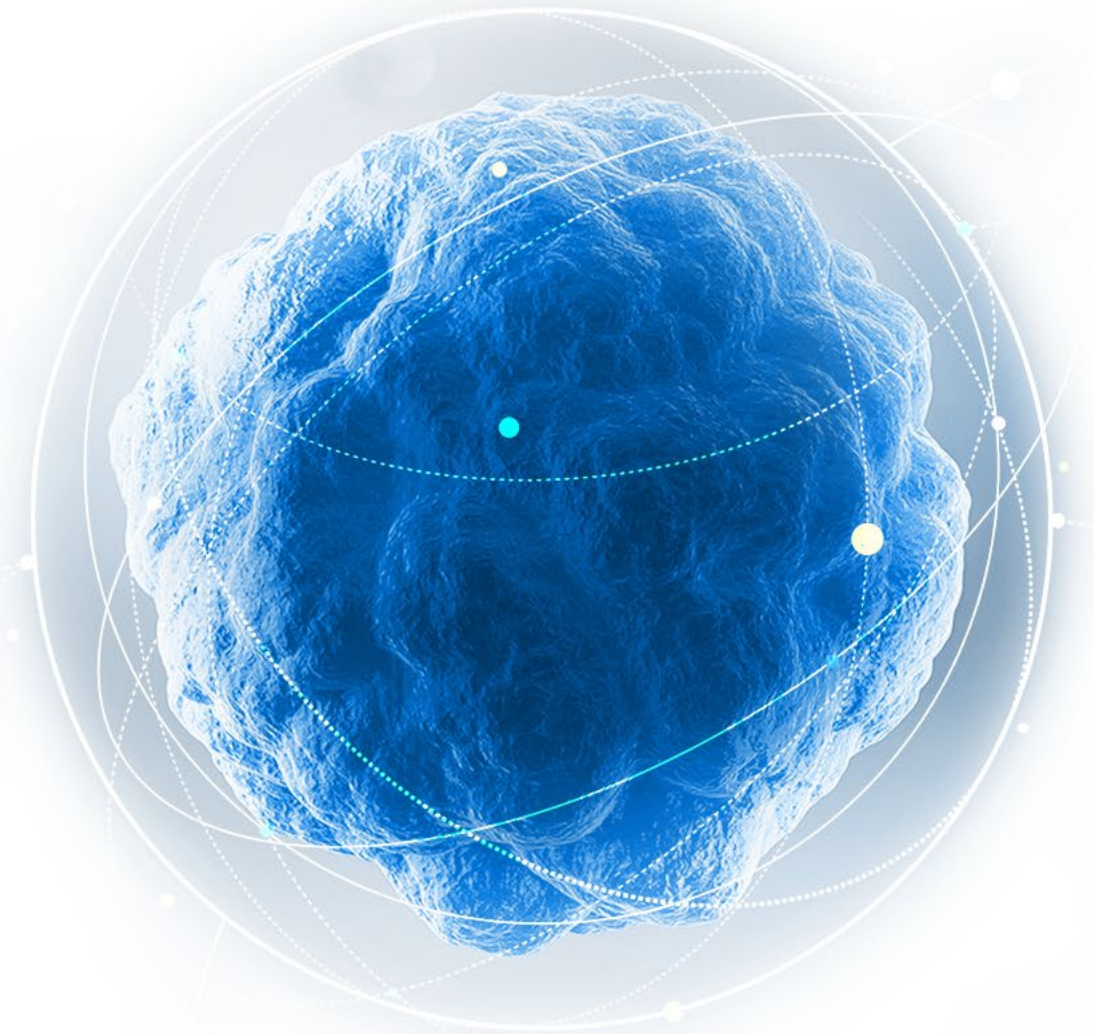
Significant resources will be required to execute a large clinical study and prepare for potential commercial launch

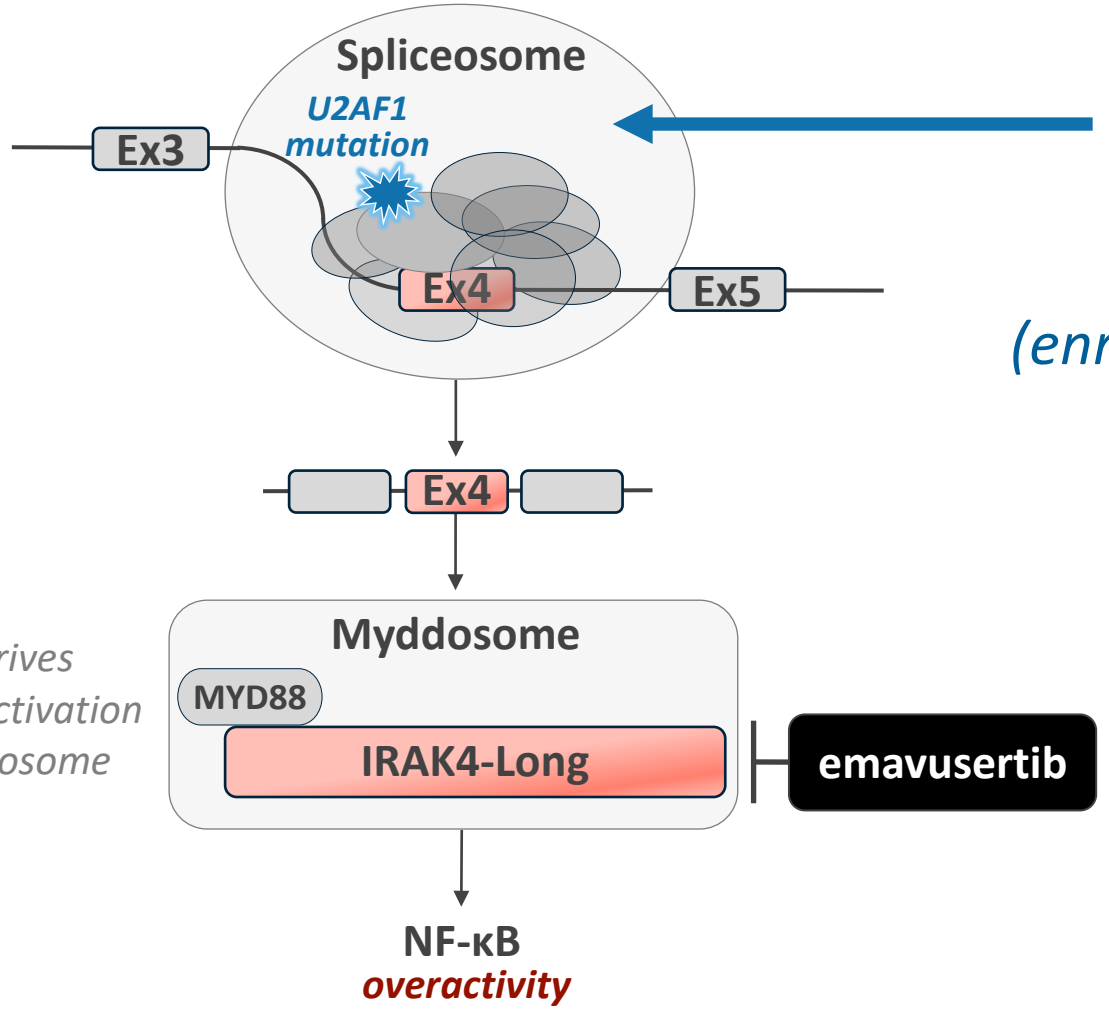
1 – Data cutoff February 26, 2024

Emavusertib
in SFm AML



CURIS®

Mechanism in SFm AML



IRAK4-L drives constitutive activation of the myddosome

splicing mutations are a driver of IRAK4-L

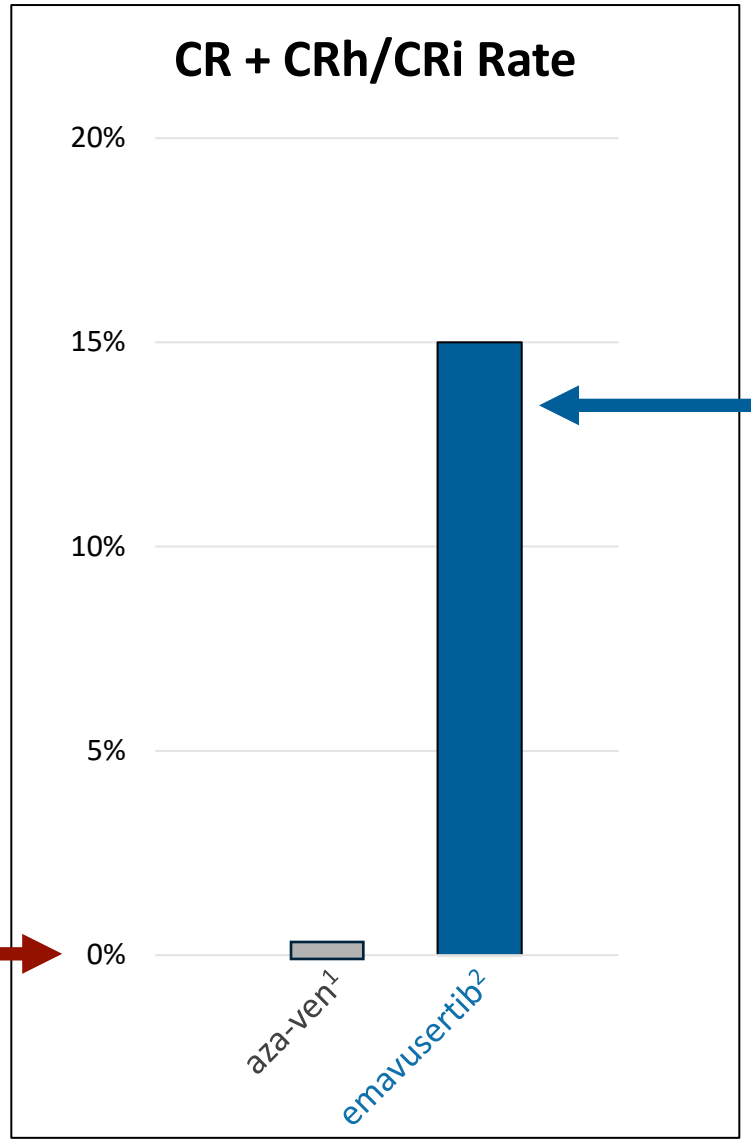
(enriching for SFm, enriches for high IRAK4-L)

Smith et al. Nat Cell Biol. 2019

R/R SFm AML

Benchmark for SFm AML
0% CR/CRi

R/R sAML with **aza+ven**[1]	CR/CRi
- Tet2	86%
- Favorable risk	75%
- Intermediate risk	65%
- Adverse-risk	34%
- U2AF1	0%



anti-cancer activity supports combination approach

1) Aldoss Am J Hem 2019 [CR/CRi]; 2) emavusertib [CR/CRh]

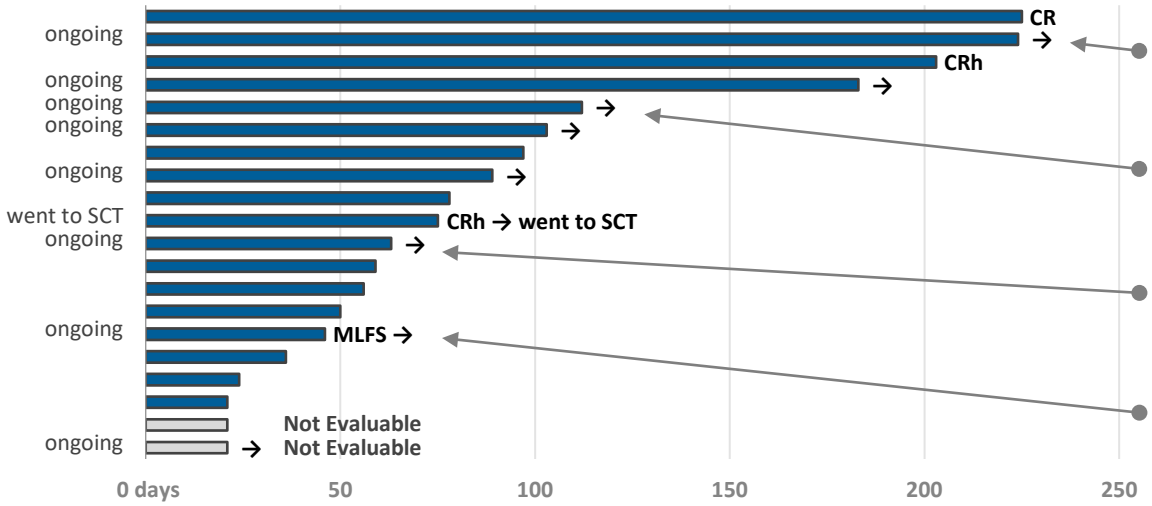
Data include all patients in target population (R/R AML patients with SFm and < 3 prior lines of therapy) treated with 300 mg BID as of Feb 26, 2024; 1 patient w/CR had dual FLT3m and SFm
The comparisons presented in the figures above represent cross-trial comparisons and do not involve data from a head-to-head clinical trials

R/R SFm AML

anti-cancer activity supports combination approach

8 patients ongoing

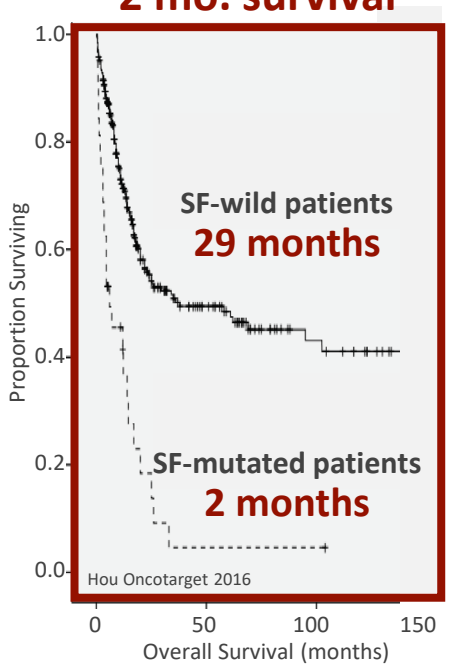
Duration on Treatment



Clinical Observations in Ongoing Patients who have not yet achieved CR/CRh

- *neutrophils up 49%, platelets > 100,000* ***(meets ANC response criteria)***
- *neutrophils up from 130 to 1,780, blasts to 15%* ***(meets ANC response criteria)***
- *neutrophils up 500%* ***(meets ANC response criteria)***
- *neutrophils up 170%, platelets up 61%, blasts to 4%*

Benchmark for SFm
2 mo. survival



Data include all patients in target population (R/R AML patients with SFm and < 3 prior lines of therapy) treated with 300 mg BID as of Feb 26, 2024; 1 patient w/CR and 1 patient w/MLFS had dual FLT3m and SFm
→ Denotes ongoing with treatment

Abbreviations: Stem Cell Transplant (SCT) and Morphologic Leukemia-Free State (MLFS)

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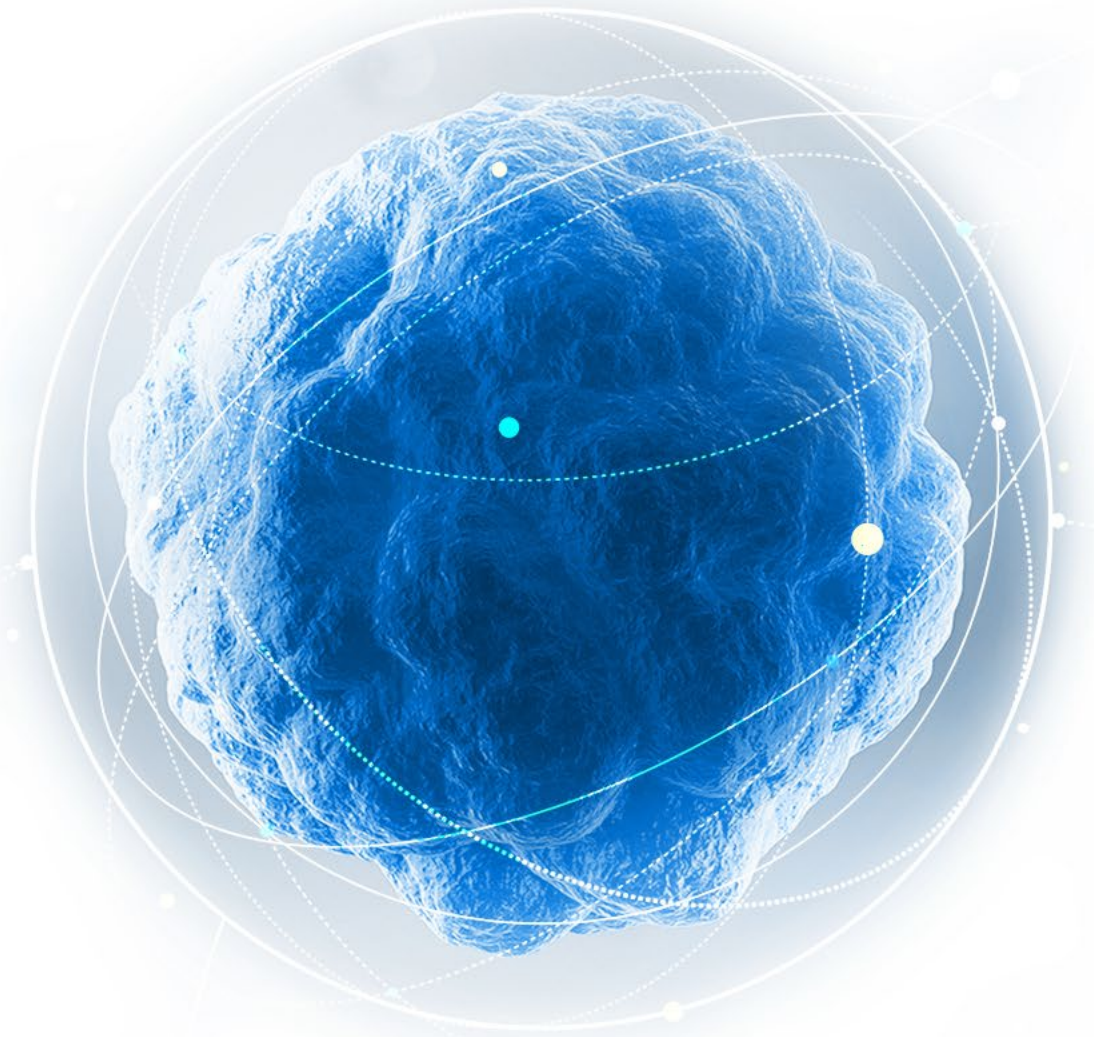
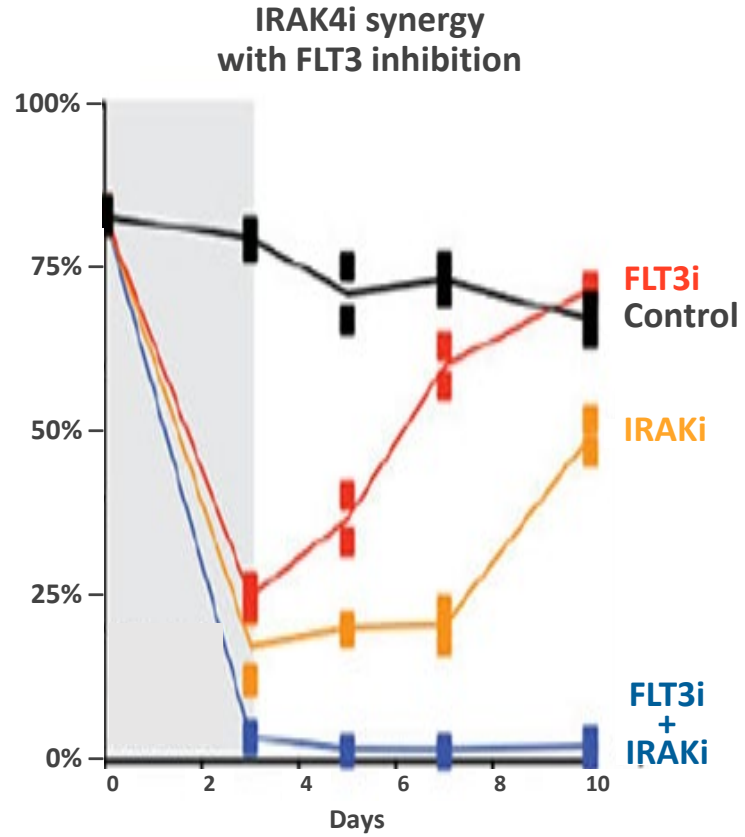
Emavusertib
in FLT3m AML



CURIS®

Emavusertib's dual-targeting of IRAK4 and FLT3 offers opportunity in FLT3m AML

IRAK4 inhibition overcomes adaptive resistance to FLT3i

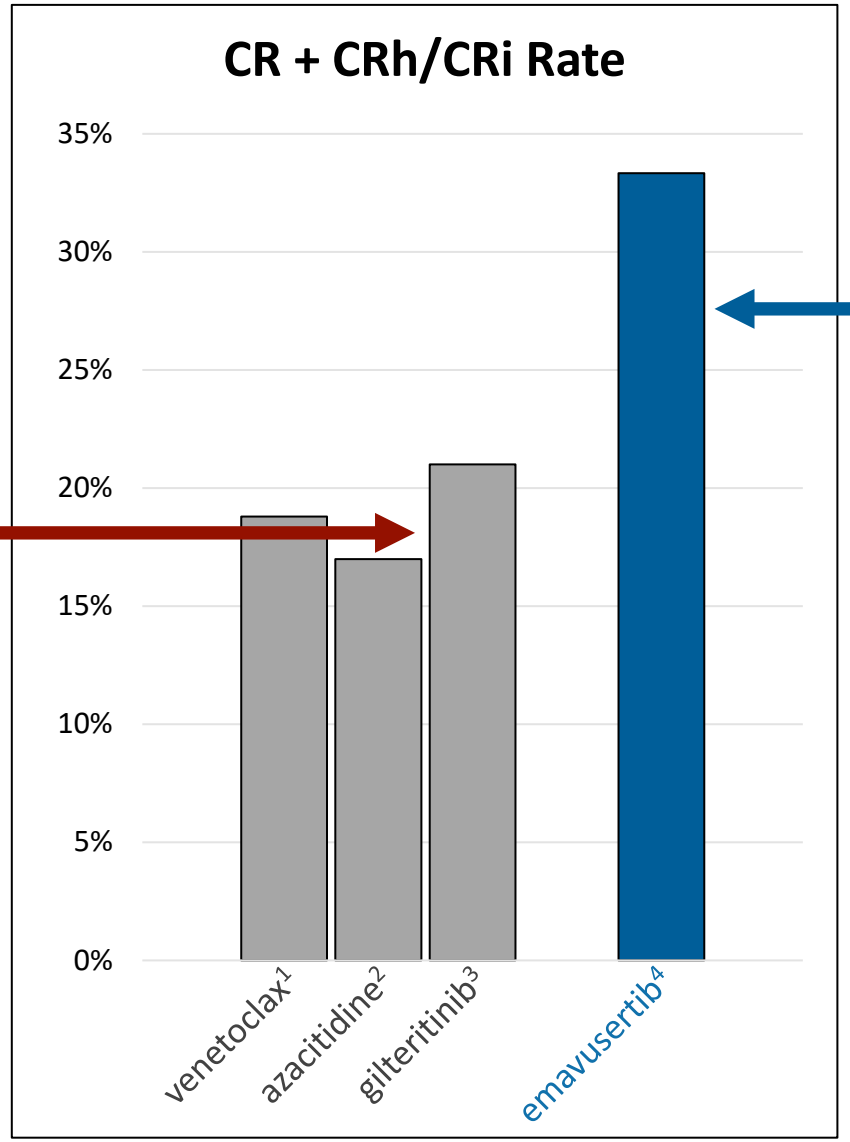


Percent viable cells in preclinical AML cell lines (FLT3-ITD) treated for 72 hrs.

"Concomitant targeting of IRAK1 or IRAK4, alongside FLT3, is the most effective means to overcome the adaptive resistance incurred when targeting FLT3"

Melgar Sci Transl Med 2019

R/R FLT3m AML



Benchmark for FLT3m AML
21% CR/CRh

87% of patients in benchmark study were naïve to FLT3i[3]

Potential Best-in-class

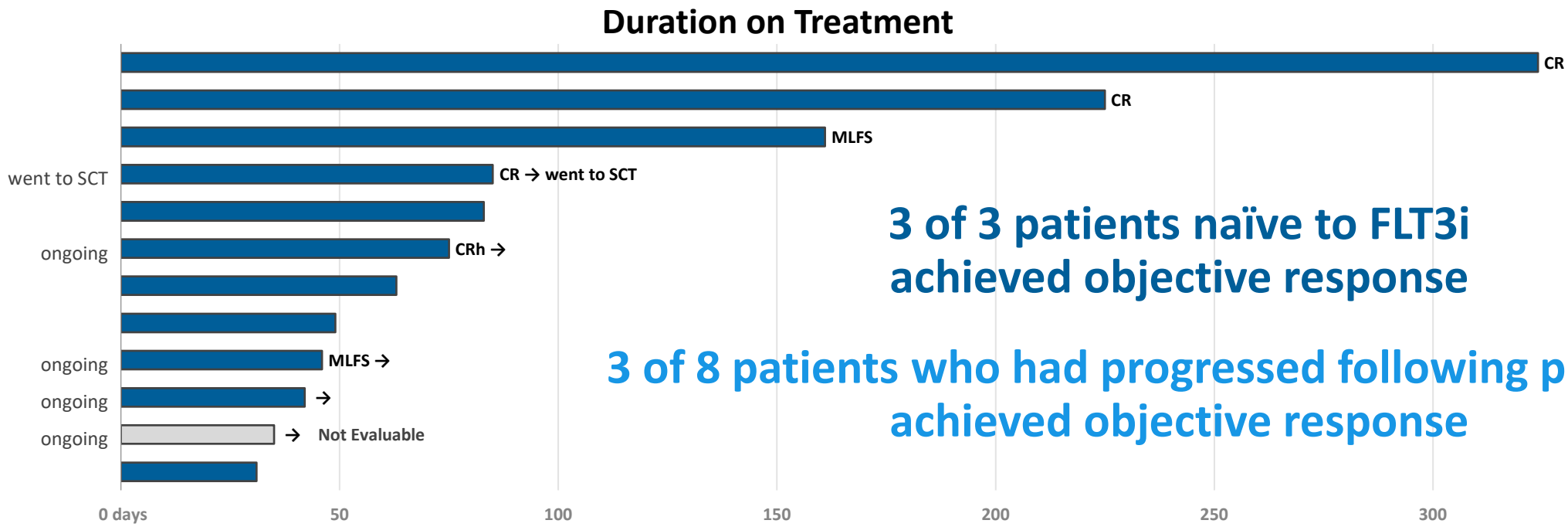
9 of 12 patients had progressed following prior FLT3i

1) Konopleva Cancer Discov 2016 [CR/CRi], 2) Itzykson Leuk Res 2015 [CR/CRi], 3) gilteritinib USPI [CR/CRh]; 4) emavusertib [CR/CRh]

The comparisons presented in the figures above represent cross-trial comparisons and do not involve data from a head-to-head clinical trials

R/R FLT3m AML

Potential Best-in-class



3 of 3 patients naïve to FLT3i achieved objective response

3 of 8 patients who had progressed following prior FLT3i achieved objective response

Data include all patients in target population (R/R AML patients with FLT3 mutation and < 3 prior lines of therapy) treated with 300 mg BID as of Feb 26, 2024; 1 patient w/CR and 1 patient w/MLFS had dual FLT3 and SF mutation

→ *Denotes ongoing with treatment*

Strategy in AML

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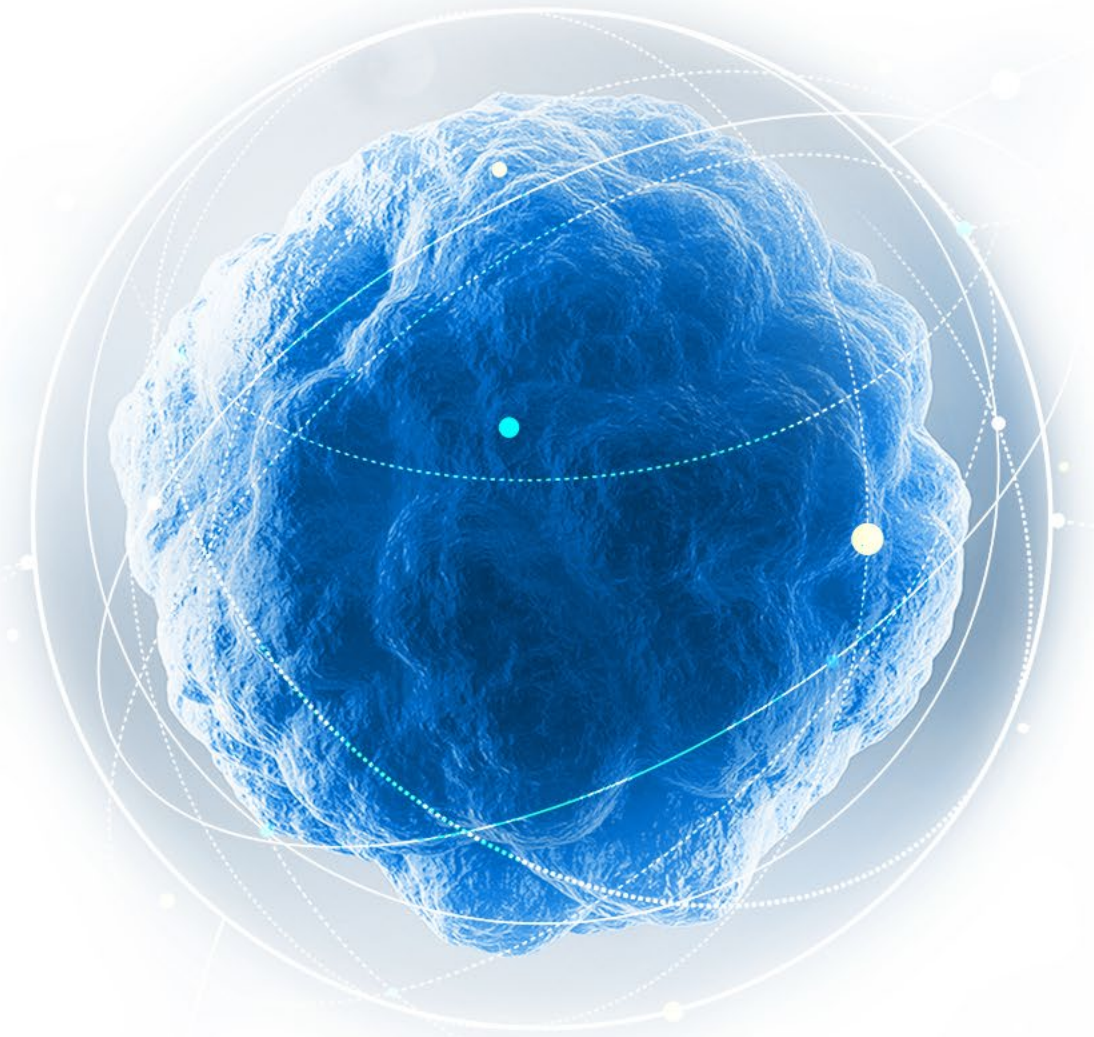
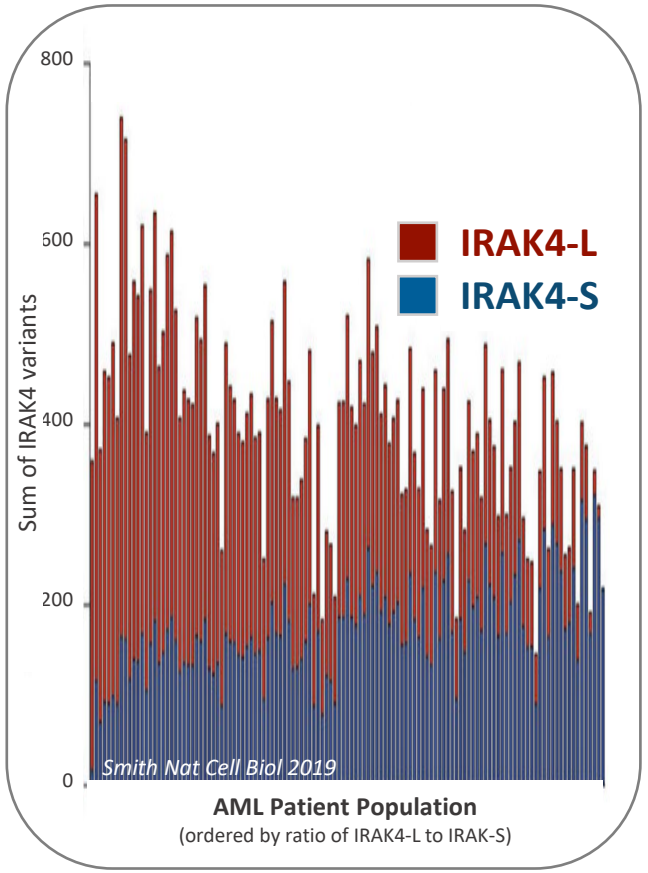
Emavusertib
in All Comers



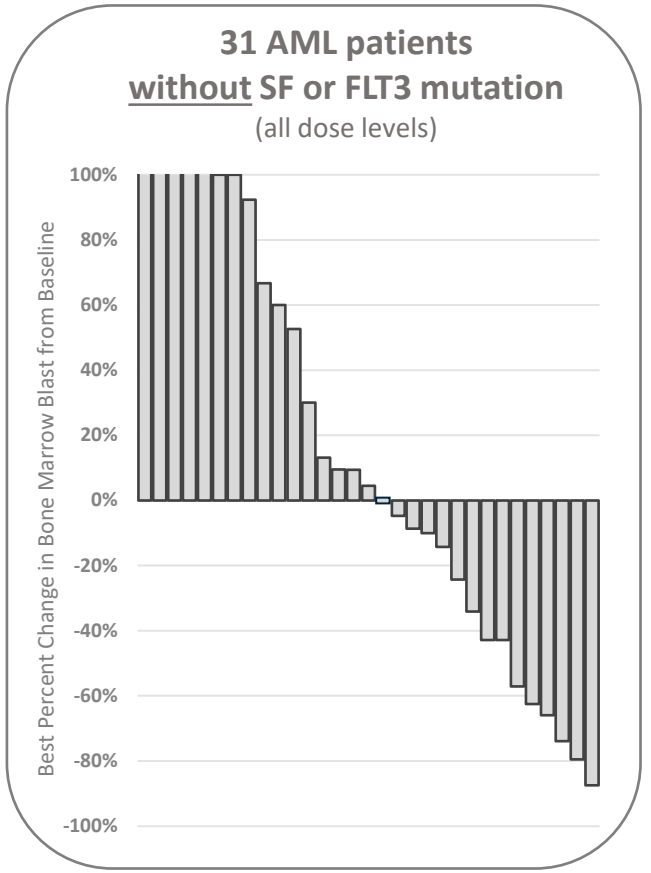
CURIS®

Combination approach to target all comers in AML

IRAK4-L is expressed in nearly all AML patients

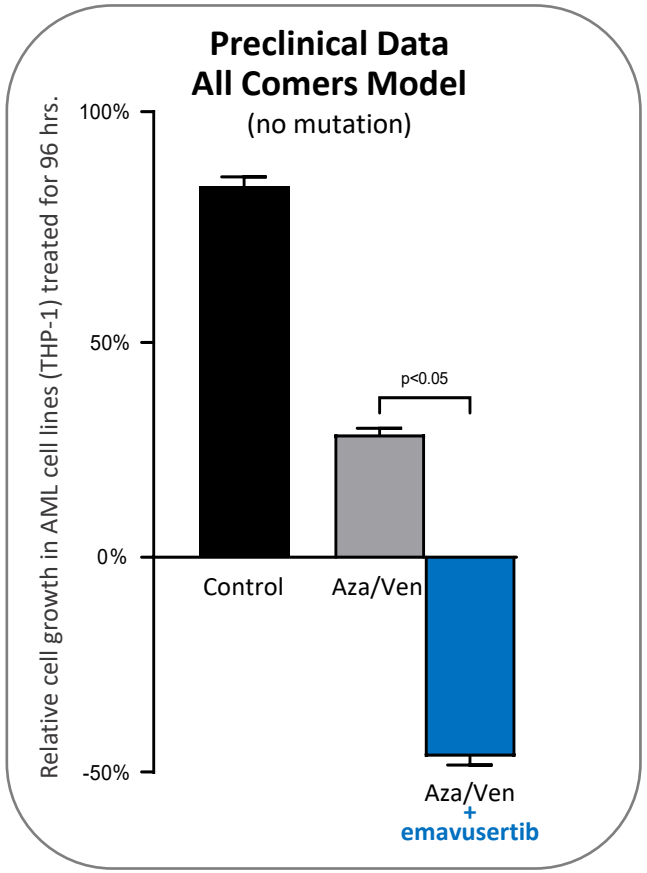


emavusertib was active even in non-targeted patients



Waterfall data include all evaluable R/R patients without FLT3 or Spliceosome mutation who were determined to be evaluable for objective response using baseline and post-treatment marrow assessments

emavusertib's novel MOA was synergistic with aza/ven



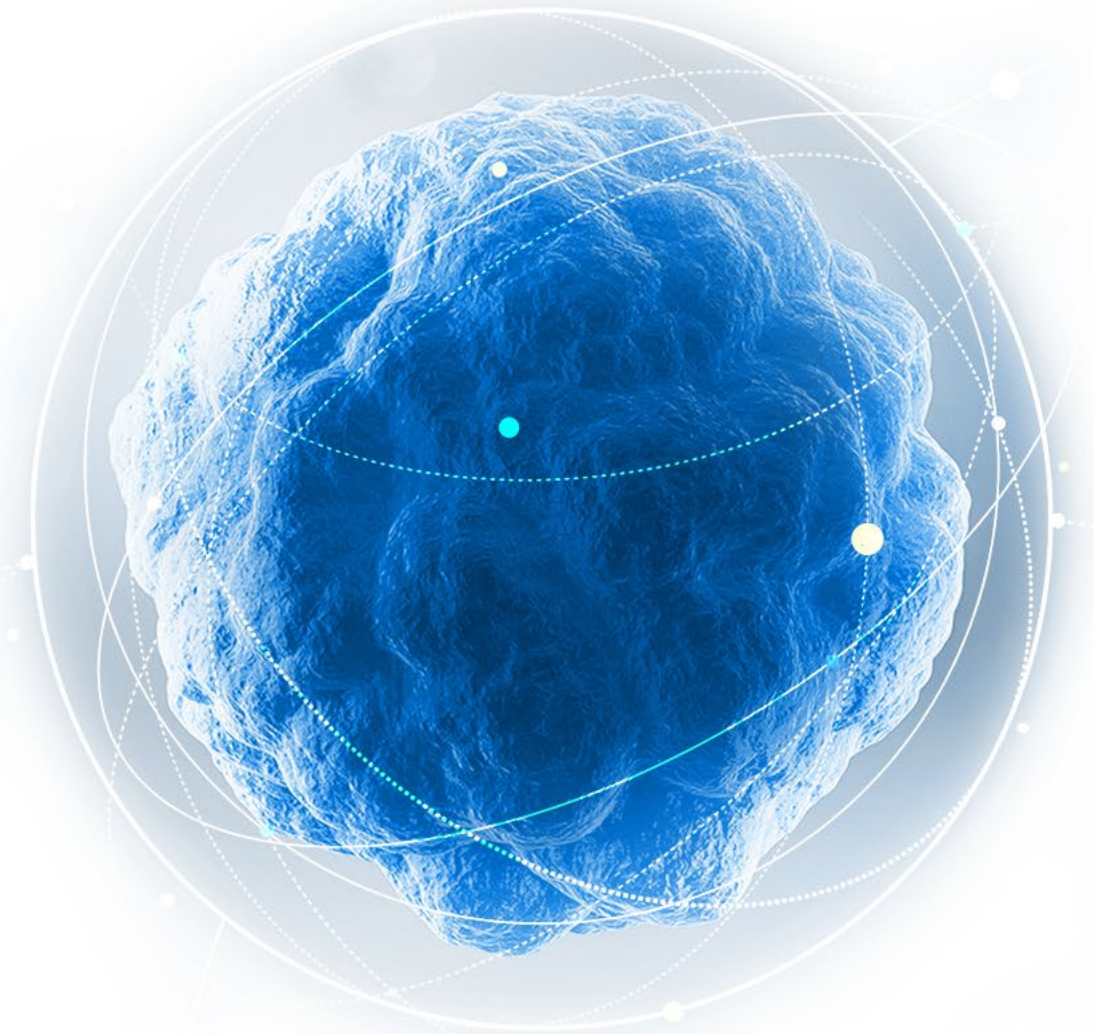
Curis AML MDS poster, EHA 2021

Summary in AML

TakeAim Leukemia

- Single-agent activity in FLT3m meets CR/CRh criteria reported for approved therapies, shows potential for best-in-class therapy
- Observed anti-cancer activity suggests potential for add-on to combination therapy (SFm)
- Potential for rapid path to 1st label in R/R AML for a genetically-defined population
- Potential for broad commercial opportunity in frontline setting with combination therapy

Other Information



CURIS®

Financials and IP

	As of March 31, 2024	
$40.7M	Cash and investments	(Estimated runway into 2025)
~5.9M	Shares Outstanding	
~7.0M	Shares Fully Diluted	
2035	Composition of Matter IP on emavusertib	

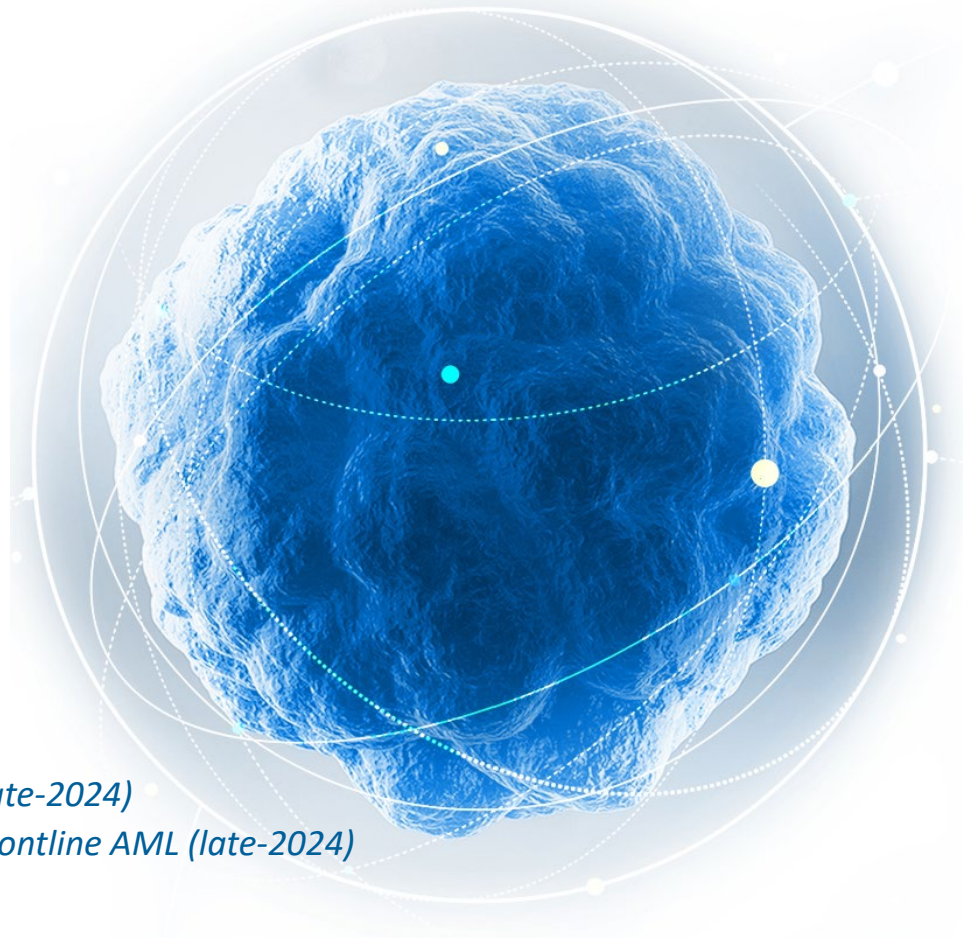
We believe cash is sufficient to achieve anticipated milestones

- *Lymphoma data late-2024*
- *AML triplet data late-2024*

Curis is developing the first-in-class IRAK4 inhibitor

Curis Overview

- *Originator of the first Hedgehog inhibitor, licensed to Roche/Genentech , marketed as vismodegib (Erivedge®)*
- *Emavusertib, the most advanced IRAK4 inhibitor is in Phase I/II clinical studies in leukemia and lymphoma*
- *Initial Phase I/II clinical data demonstrated single-agent anti-cancer activity in AML and NHL, with potential for broader application in combination with standard of care*
- *Upcoming Anticipated Milestones:*
  - *POC combination data in R/R PCNSL (late-2024)*
  - *Initial data for triplet combination in frontline AML (late-2024)*

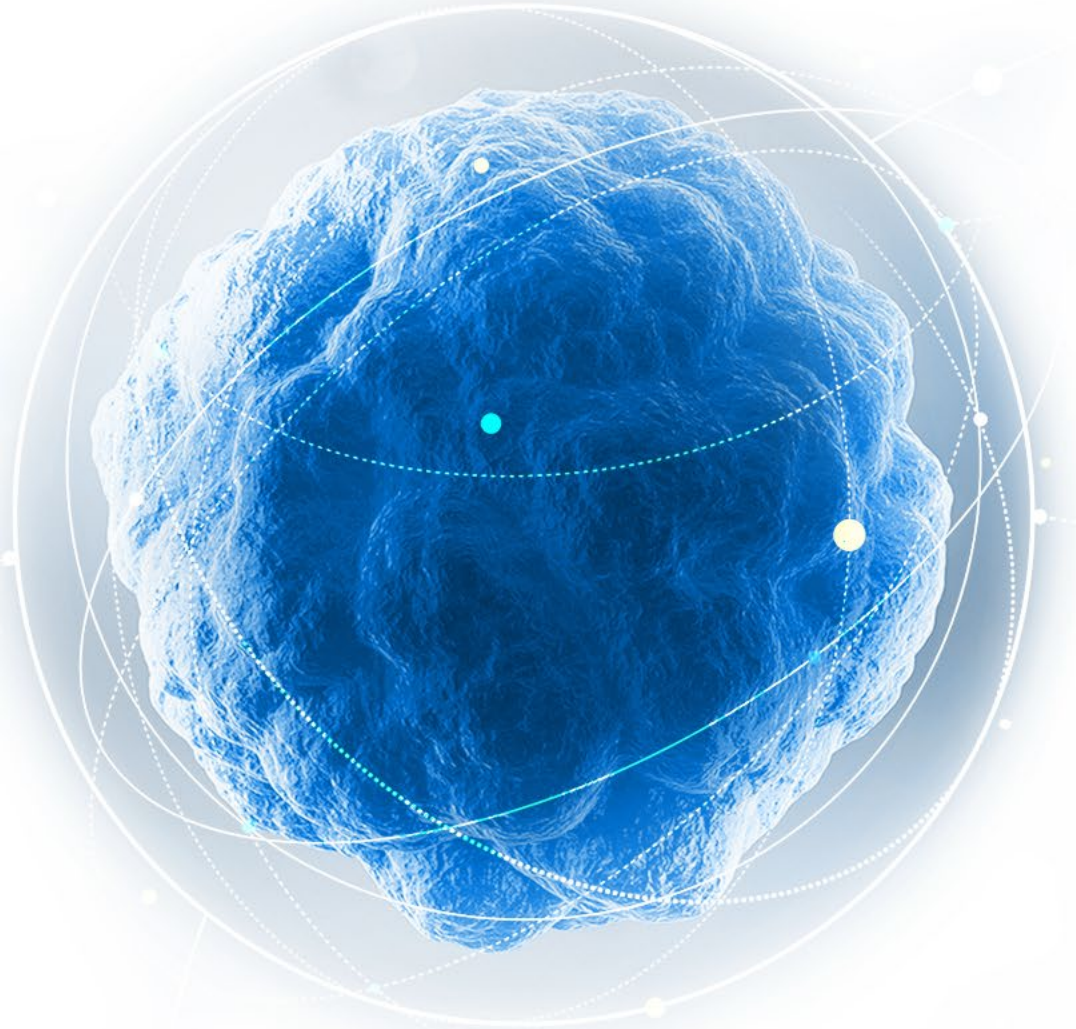


Demonstrated single-agent activity and safety in NHL and AML

Demonstrated synergy with BTKi, HMA, BCL2i

Broad opportunity in heme and solid malignancies

End of Presentation



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