Corporate Presentation

NASDAQ: CRIS

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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)”, "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 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 or 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, 2022 and the Company's Quarterly Reports on Form 10-Q for the quarters ended March 31, 2023 and June 30, 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.

Corporate Overview

Leader of IRAK4 Inhibition in Oncology; Developing Emavusertib with Broad Hematological Applications

Investment Thesis	Emavusertib is a highly-active inhibitor with potential to be the cornerstone agent in hematologic malignancies Cash runway into 2025 – Proforma cash of $77.4M as of June 30, 2023
Clinical Path	➢ R/R AML with FLT3 or Spliceosome mutation – Potential fast path to NDA ➢ R/R PCNSL in combination with ibrutinib – Potential fast path to NDA ➢ AML/MDS in combination with azacitidine and/or venetoclax – Potential frontline position
Current Trials	**TakeAim Leukemia:** IRAK4 and FLT3 are the two most prevalent drivers of disease in AML/MDS[1,2] **TakeAim Lymphoma:** emavusertib/ibrutinib enables blockade of both pathways driving NF-κB in NHL
Market Opportunity	There are no drugs approved for IRAK4 inhibition in oncology ➢ AML/MDS: **333K patients**[3] *(current standard of care is HMA)* ➢ NHL/CLL: **1.9M patients**[3] *(current standard of care is BTKi)*
Near Term Milestones	➢ R/R PCNSL data in combination with ibrutinib at ASH 2023 ➢ R/R AML FLT3 and Spliceosome data as a monotherapy ➢ Clarity with FDA on registrational study design for R/R AML FLT3 and Spliceosome ➢ AML/MDS data in combination with azacitidine and venetoclax

1) Smith et al. Nat Cell Biol 2019; 2) Saygin, et al. J Hematol Oncol. 2017 Apr 18; 3) 2022 Prevalence Data DRG Clarivate

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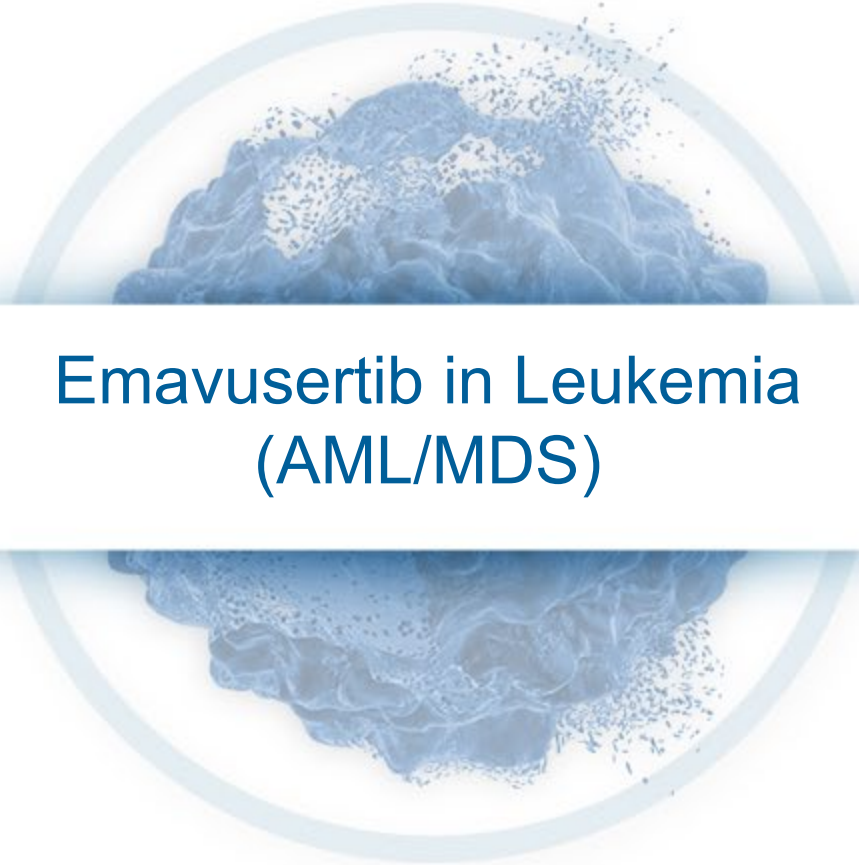


Emavusertib in Leukemia (AML/MDS)

Emavusertib has A Unique Molecular Fingerprint

Targeted design is specifically engineered to be best-in-class

The NCI selected emavusertib for NCI-sponsored research and clinical studies of IRAK4

Emavusertib
Kinase Interaction Map

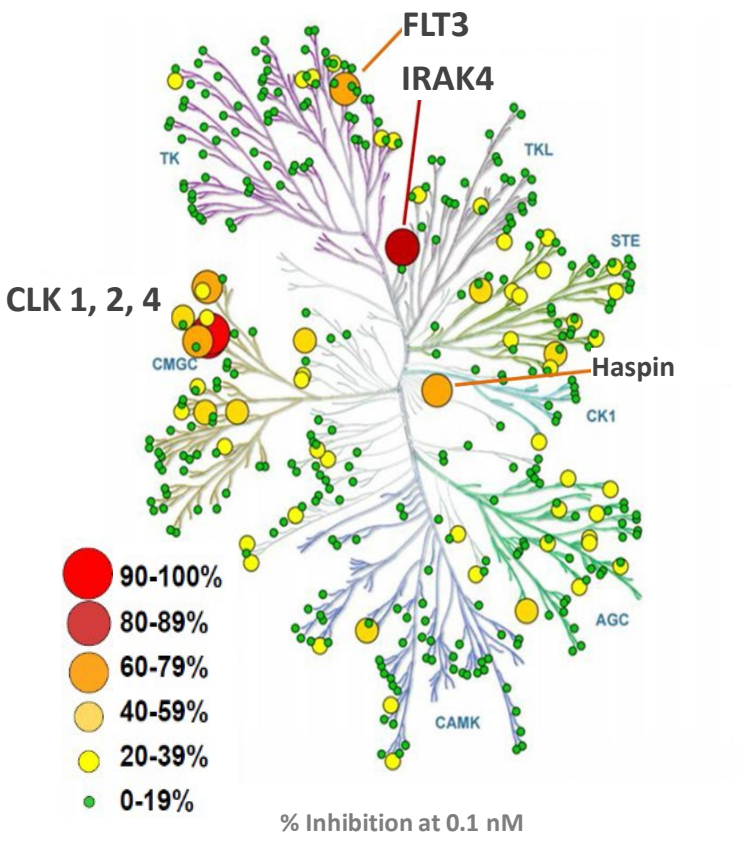


Illustration reproduced courtesy of Cell Signaling Technology

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)

high binding affinity to IRAK4
(>97% inhibition achieved at clinical dose concentrations)

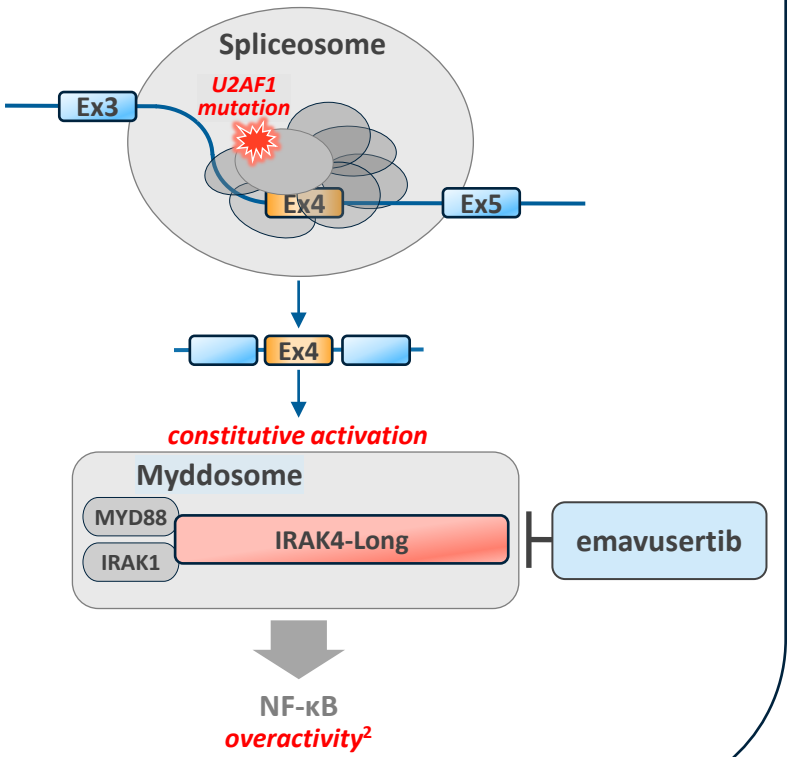
high binding affinity to FLT3
contributes additional anti-cancer activity, differentiating emavusertib from other IRAK4-directed therapies

Emavusertib Has a Unique Mechanism of Action

The two primary targets of emavusertib are independent drivers of cancer

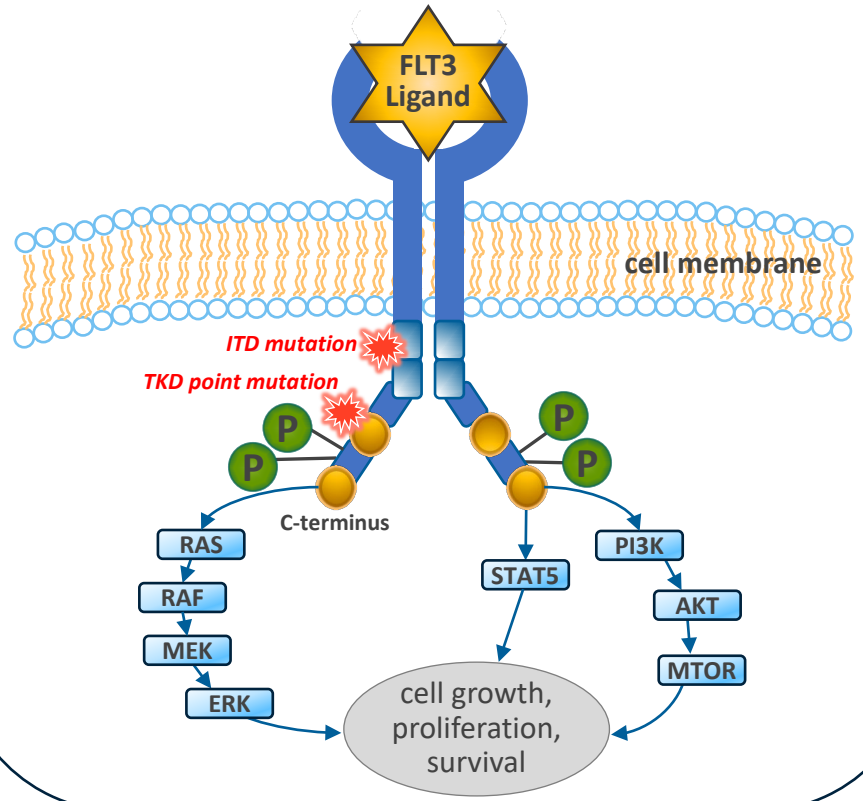
IRAK4

spliceosome mutations drive overexpression of IRAK4-Long, which constitutively activates the myddosome, driving NF-κB overactivity



FLT3

genetic mutations cause constitutive activation of FLT3 and downstream cell growth, proliferation, and survival pathways

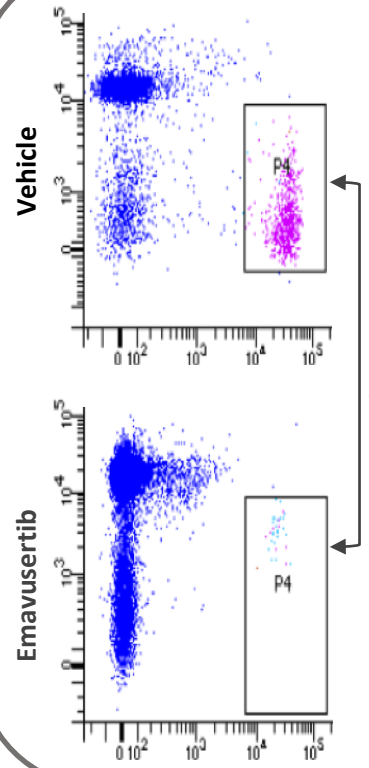


1) Guillamot et al. Nat Cell Biol 2019; 2) Smith et al. Nat Cell Biol 2019; 3) American Cancer Society, Cancer Facts & Figures 2020; 4) Leukemia & Lymphoma Society, Facts and Statistics Overview

Preclinical Data

Rationale for monotherapy and combination with azacitidine/venetoclax

Monotherapy in IRAK4

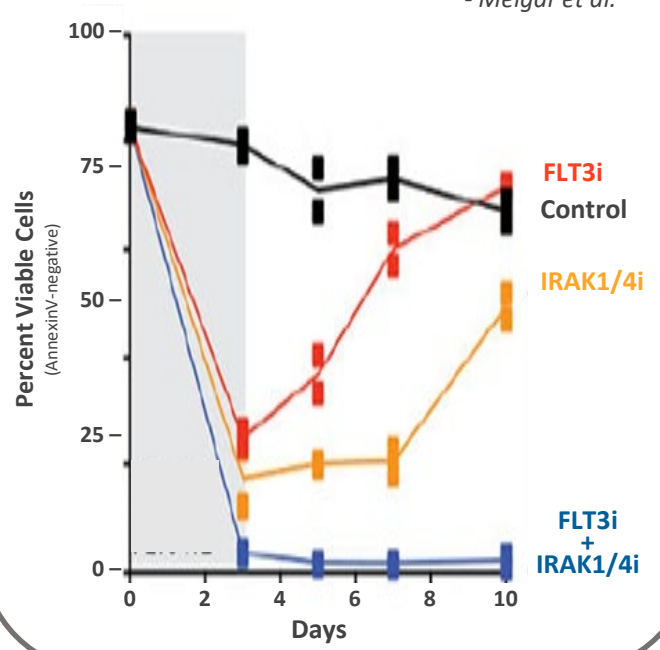


clear reduction of leukemic blasts in models with high IRAK4-L expression

emavusertib demonstrates monotherapy activity in patient-derived xenografts[1]

Monotherapy in FLT3

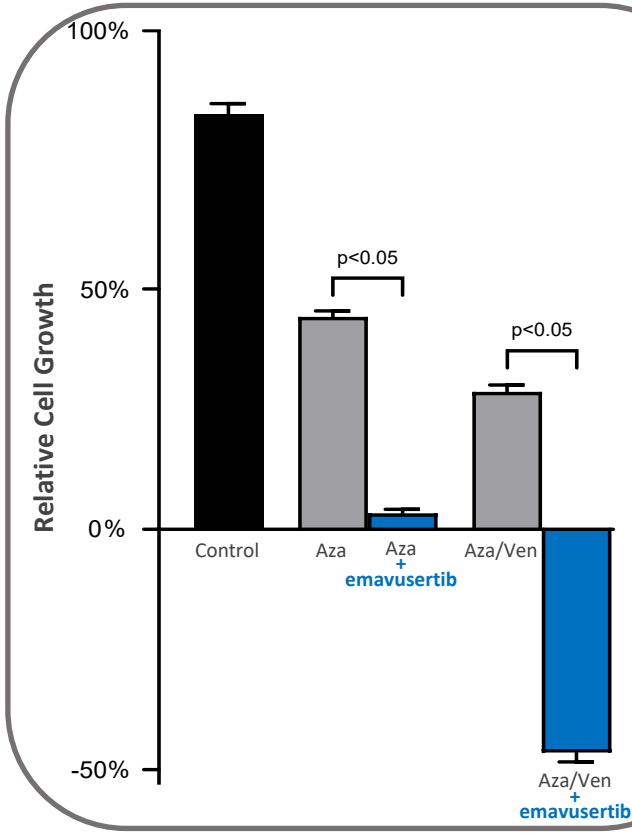
"Concomitant targeting of IRAK1 or IRAK4, alongside FLT3, is the most effective means to overcome the adaptive resistance incurred when targeting FLT3"
- Melgar et al.



IRAK/FLT3 combination demonstrates synergy in pre-clinical studies[2]

FLT3-ITD cells treated for 3 days with DMSO (control), quizartinib (0.5 µM), IRAKi (10 µM), and quizartinib + IRAKi

Combination with Aza/Ven



emavusertib demonstrates synergy with both azacitidine and venetoclax in THP-1 model[3]

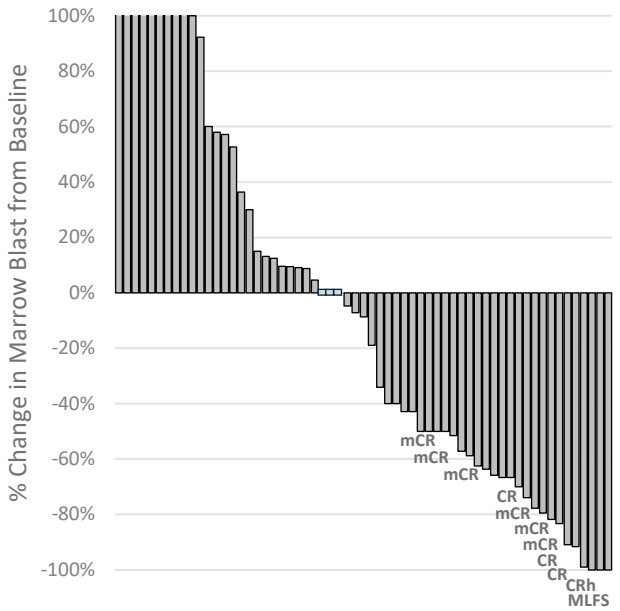
AML cell lines treated for 96 hrs (values presented as mean ± SE)

1) Choudhary et al. AACR 2017; 2) Melgar, Sci Transl Med. 2019; 3) Curis AML MDS poster, EHA 2021

Emavusertib Compelling Initial Clinical Data

Showing clear single agent activity where expected in clinical studies

84 AML/MDS Patients
all comers, all dose levels



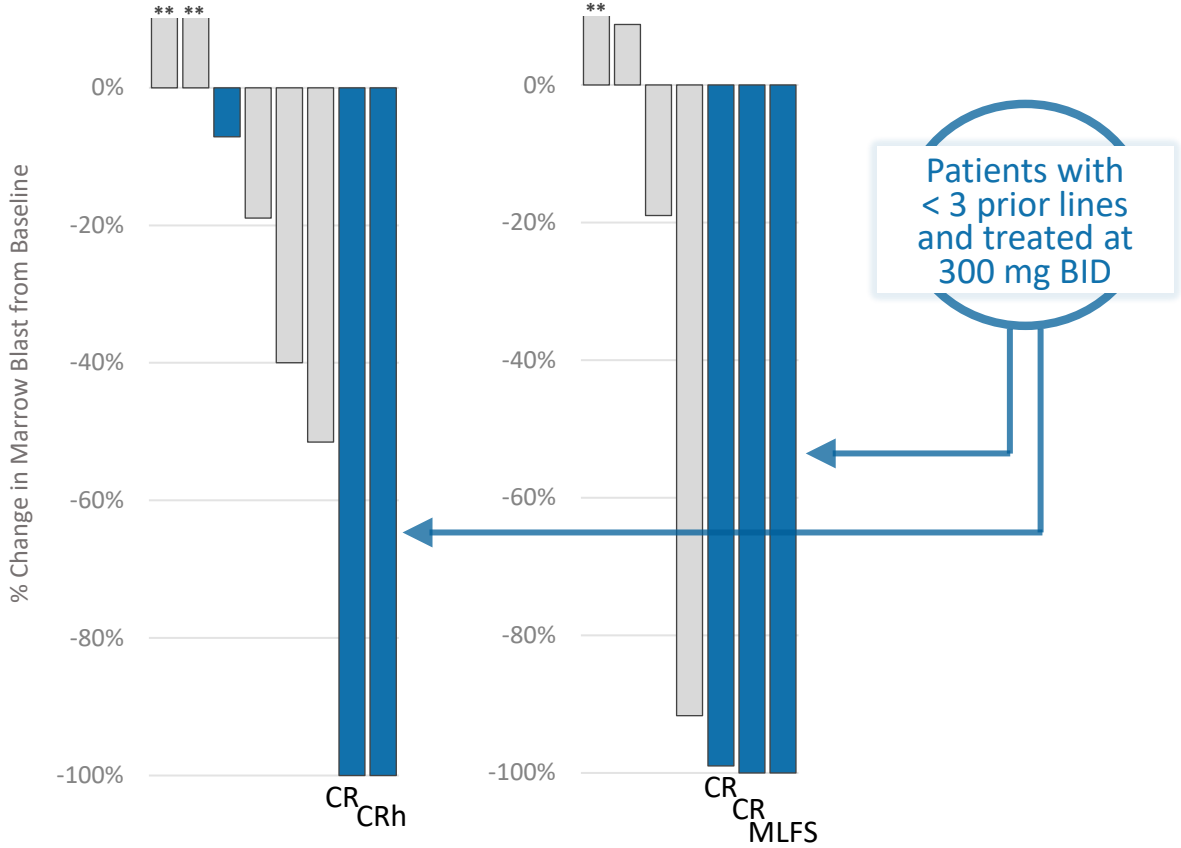
7	AML FLT3*
8	AML Spliceosome*
24	AML Other
17	AML Not Evaluable for Marrow Assessment
12	MDS Spliceosome
13	MDS Other
6	MDS Not Evaluable for Marrow Assessment
84	Total patients enrolled in monotherapy

Strongest monotherapy signal observed where expected

(Spliceosome/FLT3 patients)

8 AML Patients
Spliceosome Mutation*

7 AML Patients
with FLT3 Mutation*



Duration of Responses: 5.6 – 7.0 months

Note: 84 total AML/MDS patients enrolled in monotherapy as of Feb 9, 2023 with data cut-off as of Mar 17, 2023;
* Three AML patients had both FLT3 and Spliceosome mutation and are counted in both populations; evaluable patients include all patients whose disease was determined to be evaluable for objective response with baseline and post-treatment marrow assessments
** Denotes blast percent increase > 10%

Potential Cornerstone Treatment for Hematologic Malignancies

Emavusertib inhibits both IRAK4 and FLT3 – key disease drivers in leukemia and lymphoma

Leukemia

Monotherapy targets the two largest **genetically-defined** patient populations in AML/MDS[a,6-8]

Emavusertib synergizes with existing treatments[5] as **IRAK4-L is expressed in nearly all AML/MDS patients**

AML/MDS opportunity: 333K patients[3]

Emavusertib has demonstrated clinical activity across both targeted and non-targeted populations[1,4,5]

Lymphoma

Initial clinical data show potential to **overcome BTKi resistance** in patients with NHL/CLL[1]

Combination with BTKi **blocks both BCR and TLR signaling**, maximizing downregulation of NF-kB[2]

NHL/CLL opportunity: 1.9M patients[3]

[a] Patients with *FLT3* mutation or IRAK4-L overexpression. 1) Joffe, et al. Hemasphere. 2022. June 6(Suppl 3):1011-1012; 2) Guidetti, et al. AACR Mol Cancer Ther. 2021;20(Suppl 12):P073; 3) 2022 Prevalence Data DRG Clarivate; 4) Garcia-Manero, et al. Hemasphere. 2022. June 6(Suppl 3):30-31; 5) Curis press release. https://investors.curis.com/2022-12-12-Curis-Announces-Additional-Encouraging-Clinical-Data-from-TakeAim-Leukemia-Study-of-emavusertib-CA-4948-in-Monotherapy-R-R-AML-and-hrMDS, accessed May 19, 2023; 6) Smith, et al. Nat Cell Biol. 2019; 7) Choudhary, et al. eLife. 2022;11:378136; 8) Saygin, et al. J Hematol Oncol. 2017 Apr 18.

Emavusertib in Leukemia (AML/MDS)

The ideal candidate to address the two largest genetically-defined populations in AML/MDS

Emavusertib addresses the two largest genetically-defined populations in AML/MDS:

1) IRAK4-L (>50% of population)[1]
2) FLT3 (>25% of population)[2]

- Clear single-agent activity
- Genetically-defined patient population
- Dual targeting of IRAK4 and FLT3 may prevent adaptive resistance observed with current FLT3 inhibitors[3]

TakeAim

Next Steps

TakeAim Leukemia Study

- *Monotherapy: R/R AML with FLT3*
  R/R AML with Spliceosome
- *Combination: AML/MDS in combination with azacitidine/venetoclax*

1) Smith et al. Nat Cell Biol 2019; 2) Saygin, et al. J Hematol Oncol. 2017 Apr 18; 3) Melgar, Sci Transl Med. 2019

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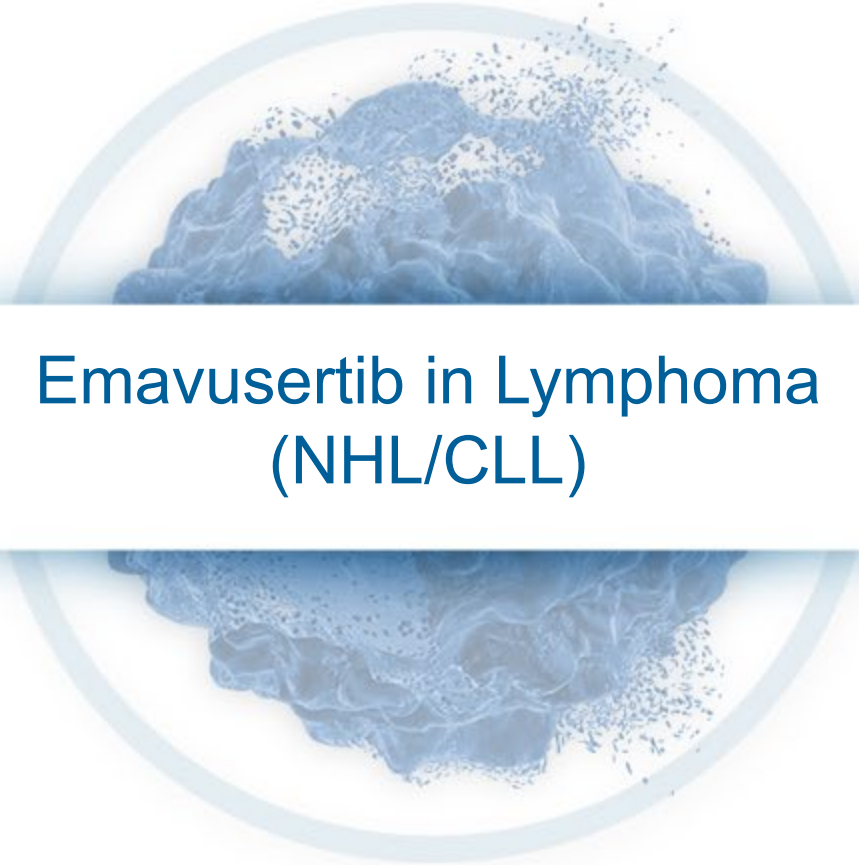


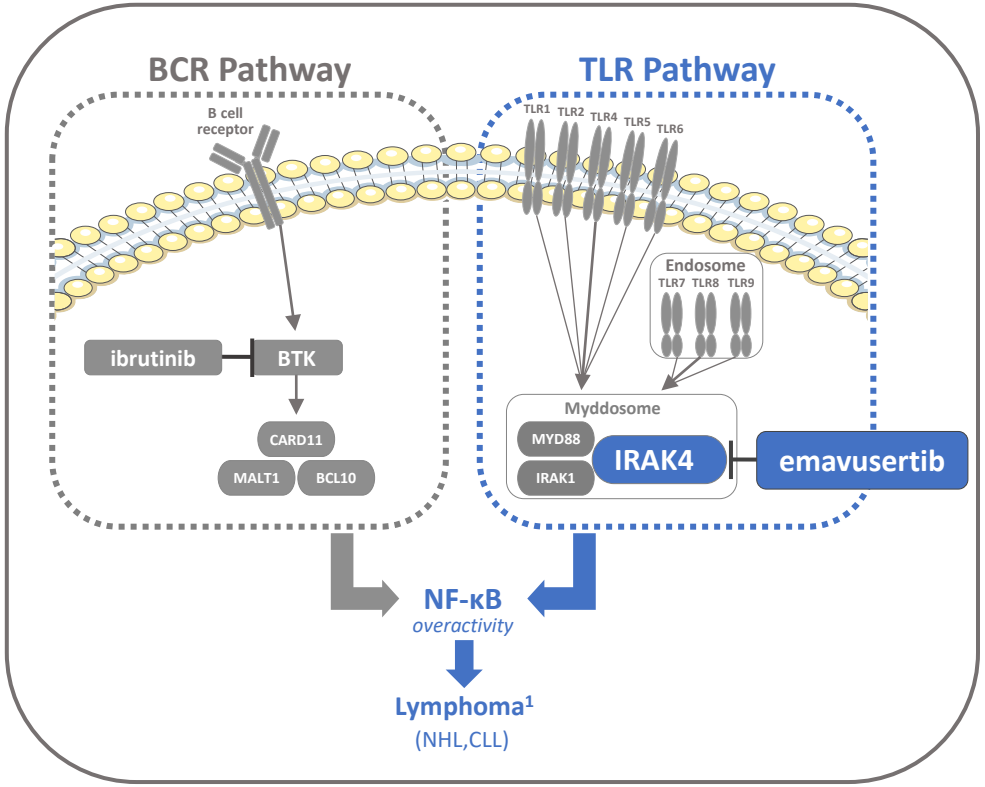
Emavusertib in Lymphoma (NHL/CLL)

Emavusertib in Lymphoma

Combination therapy provides complimentary inhibition of two pathways that drive NF-κB

NFκB Biology:
Two Pathways Drive NHL/CLL

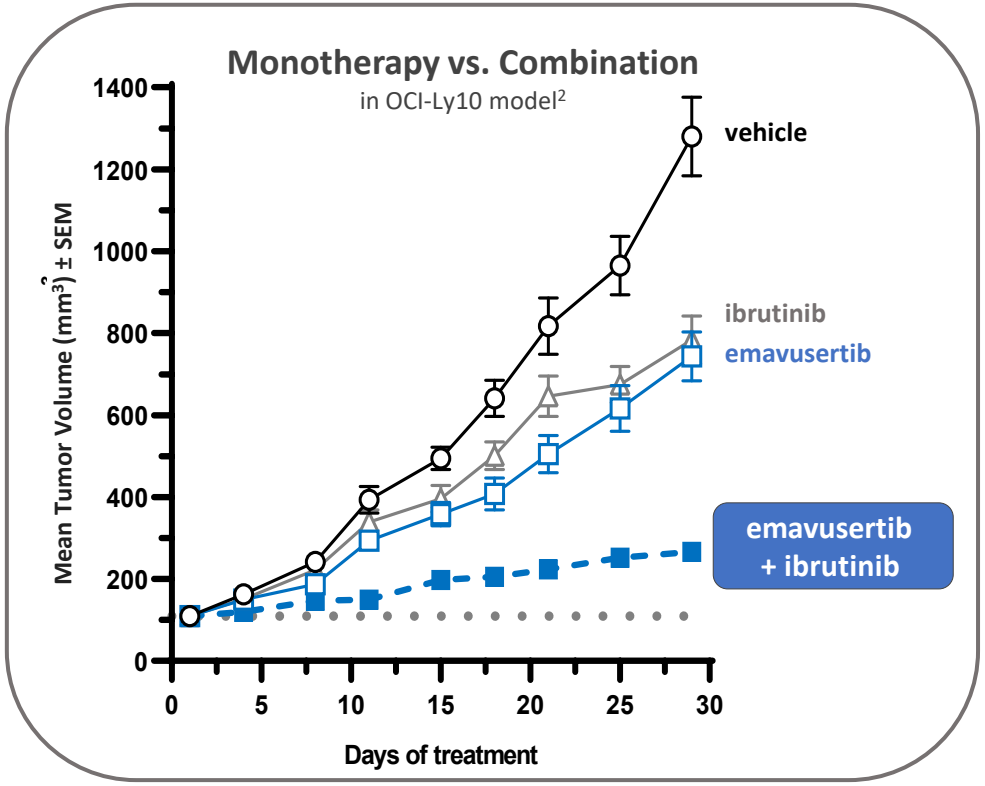
BCR and TLR Pathways independently drive NF-κB overactivity (and NF-κB drives NHL/CLL)



1) IMBRUVICA Package Insert. Rev 08/2018

Clinical Strategy:
Block both pathways with Combination Therapy

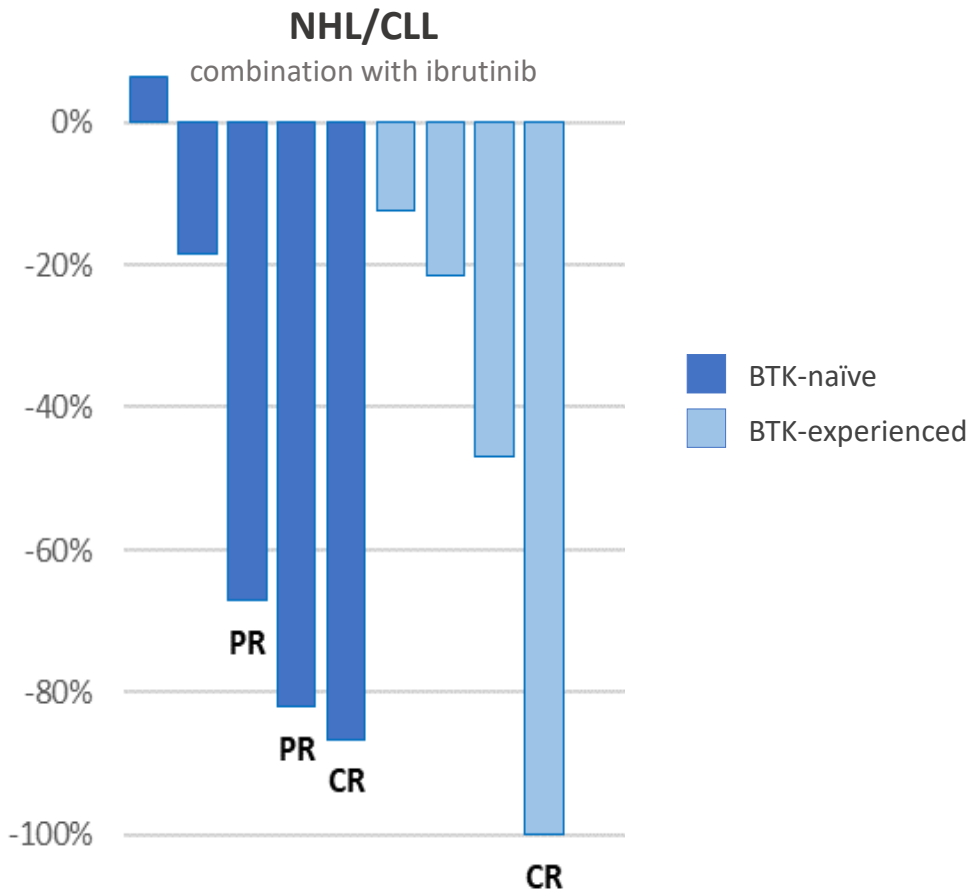
In preclinical testing, blocking both IRAK4 and BTK drove tumor reduction better than blocking either one alone

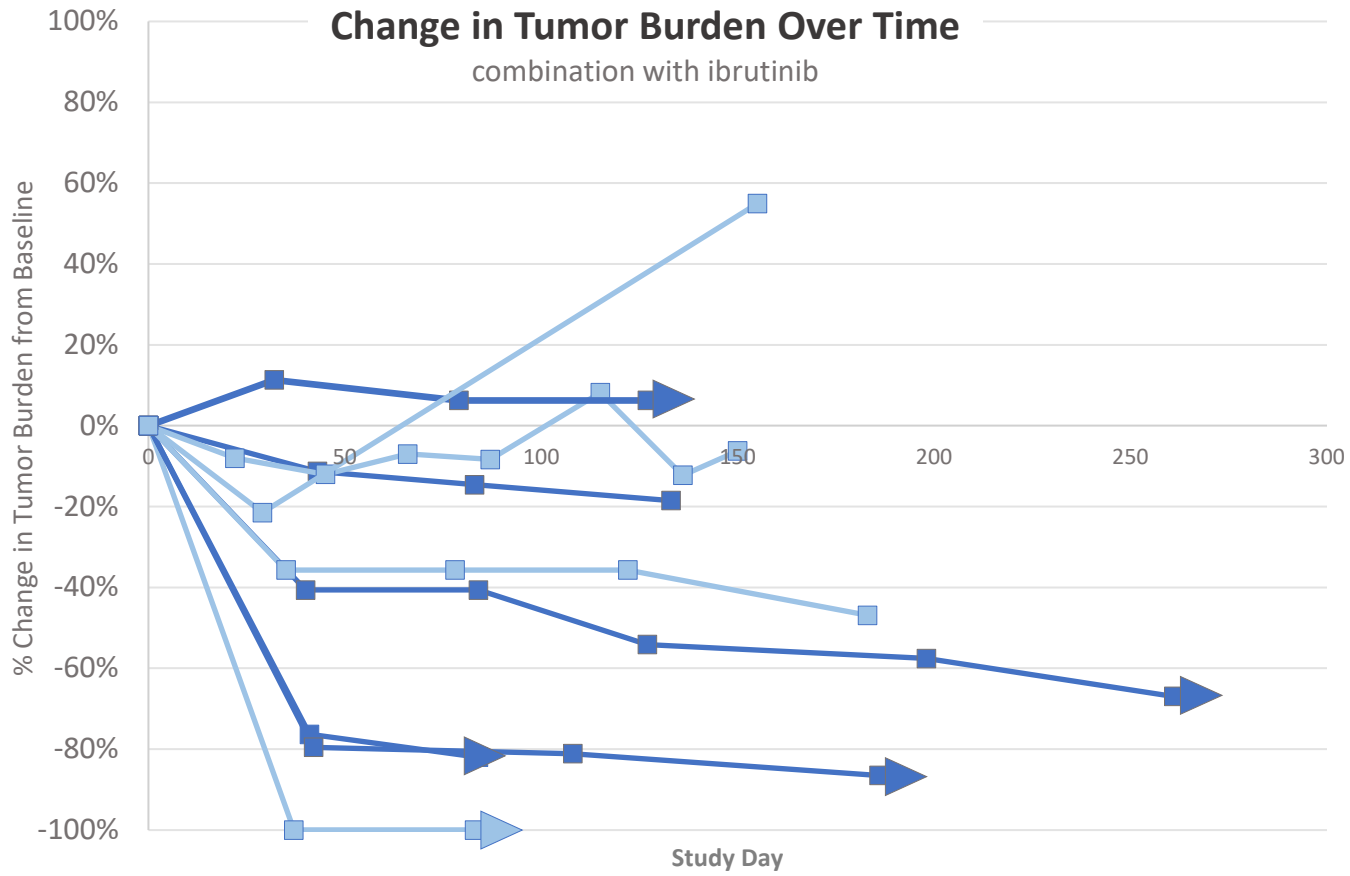


2) Booher et al. Waldenström Roadmap Symposium 2019

Initial Clinical Data in Lymphoma

Majority of patients achieved decreases in tumor burden, including complete responses





Note: Data from ASCO 2022 poster presentation
Response evaluable patients with baseline and post-treatment disease assessment at data cutoff

Clinical Strategy in Lymphoma

The ideal candidate to combine with BTKi to maximize downregulation of NF-κB

- Patients are currently treated with BTKi because it downregulates NF-κB
- Two pathways drive NF-κB:
  1) BCR Pathway: *addressed by blocking BTK*
  2) TLR Pathway: *addressed by blocking IRAK4*
- Initial clinical data suggest blocking both pathways may overcome resistance to ibrutinib

TakeAim

Next Steps

TakeAim Lymphoma Study

- *Combination with BTKi: R/R PCNSL*

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End of Corporate Presentation

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