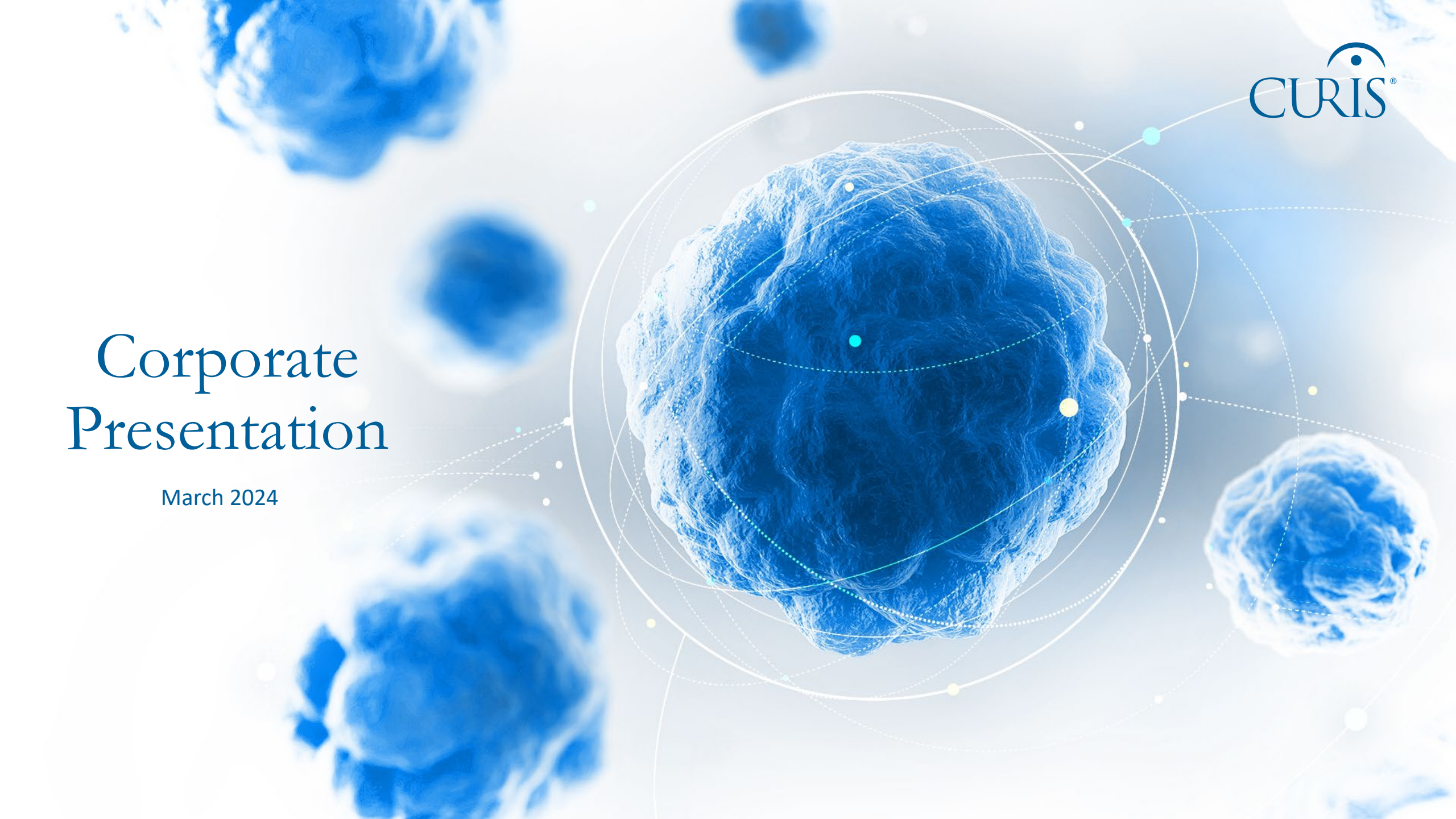


Corporate Presentation

March 2024

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)”, "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, 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 the leader of IRAK4 in oncology

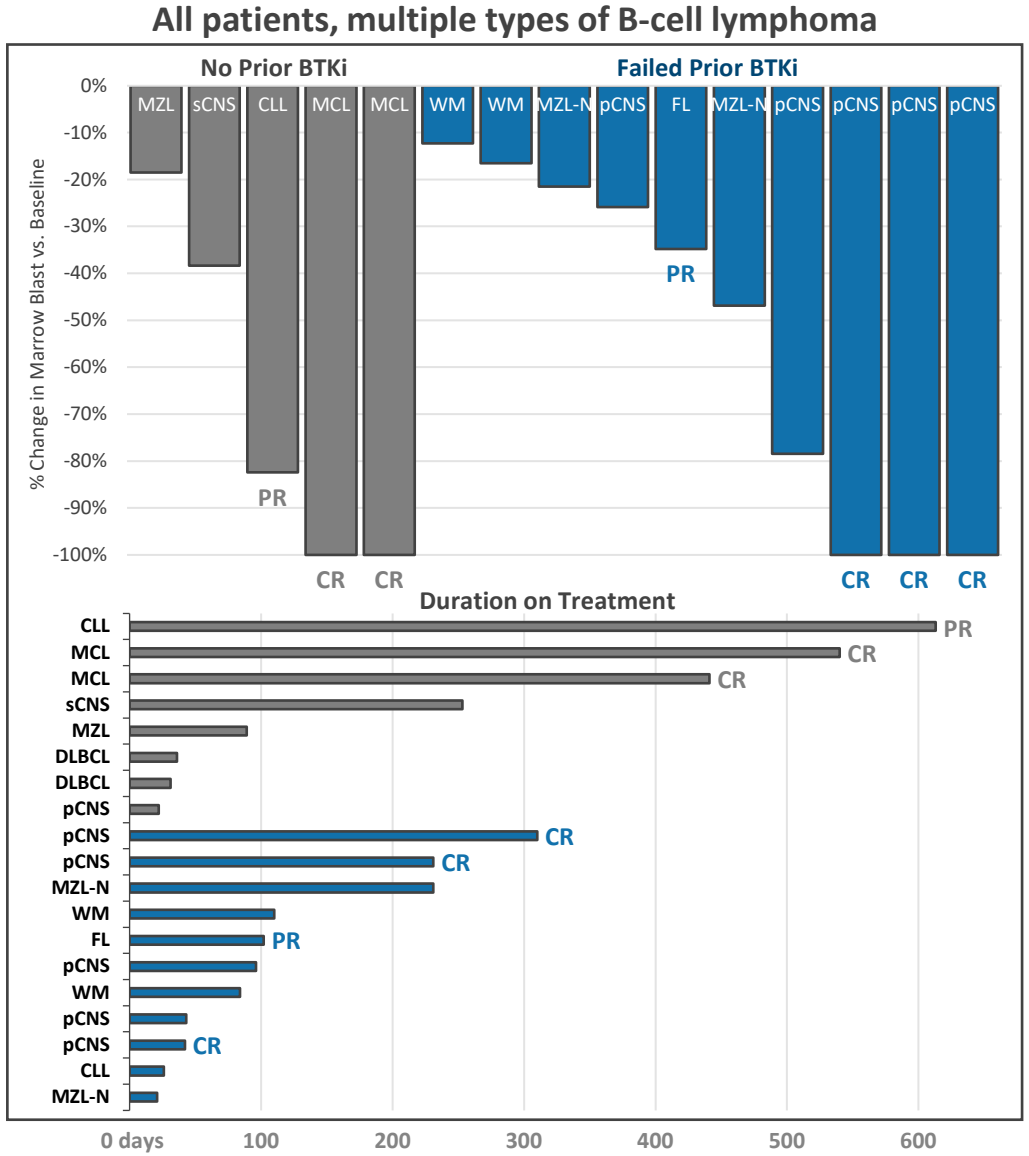
CURIS | Snapshot

Investment Thesis	Emavusertib is a novel, highly-active IRAK4 inhibitor with potential cornerstone utility in heme and solid tumors *Full development program underway in front-line and end-stage AML/MDS; PoC study in PCNSL in combination with BTKi* *Single agent and combination study results near-term (2024); Cash runway into 2025 – $56.3M as of Dec 31, 2023*	
Recent Update	PoC data in PCNSL released at ASH 2023 supports thesis for emavusertib in lymphoma, 3 of 5 patients who progressed on prior BTKi achieved CR when dosed with emavusertib/ibrutinib combination	
Key Indications	TakeAim Leukemia:	Near-term, potentially registration-directed, studies advancing in AML/MDS *Emavusertib inhibits IRAK4 and FLT3, the two most prevalent disease drivers in AML/MDS[1,2]*
	TakeAim Lymphoma:	Near-term, potentially registration-directed, study advancing in PCNSL *Emavusertib facilitates complementary blockade of two key pathways driving NFκB-mediated proliferation in NHL*
	Solid Tumors:	Multiple investigator-sponsored studies underway *Preclinical studies show IRAK4 potentiates chemo- and immunotherapies in combination in solid tumor malignancies*
Market Opportunity	AML/MDS:	**333K patients[3]** *all patients addressable with either front-line combination or salvage-line monotherapy*
	NHL/CLL:	**1.9M patients[3]** *all patients addressable with emavusertib in combination with BTKi*
	Solid Tumors:	**tbd** *opportunities being explored in metastatic melanoma, bladder, colorectal, and others*

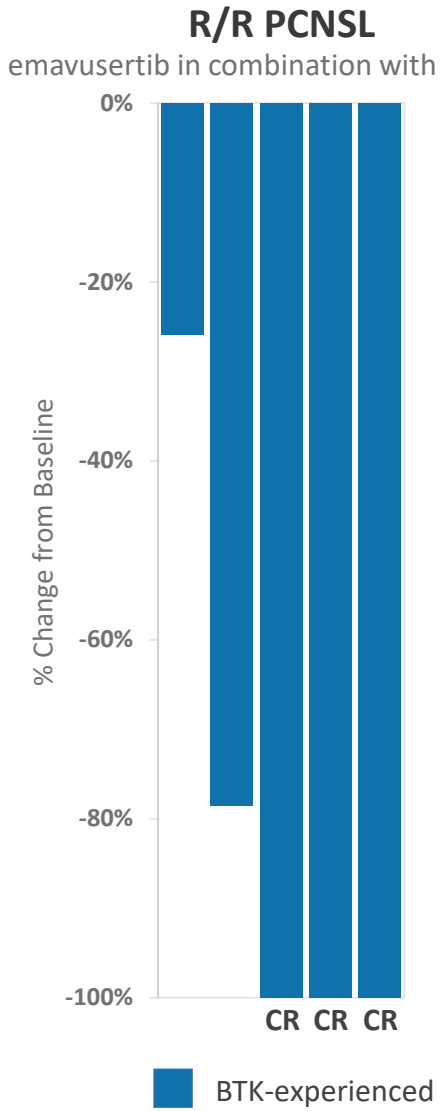
1) Smith et al. Nat Cell Biol 2019; 2) Saygin, et al. J Hematol Oncol. 2017 Apr 18; 3) 2022 Prevalence Data DRG Clarivate

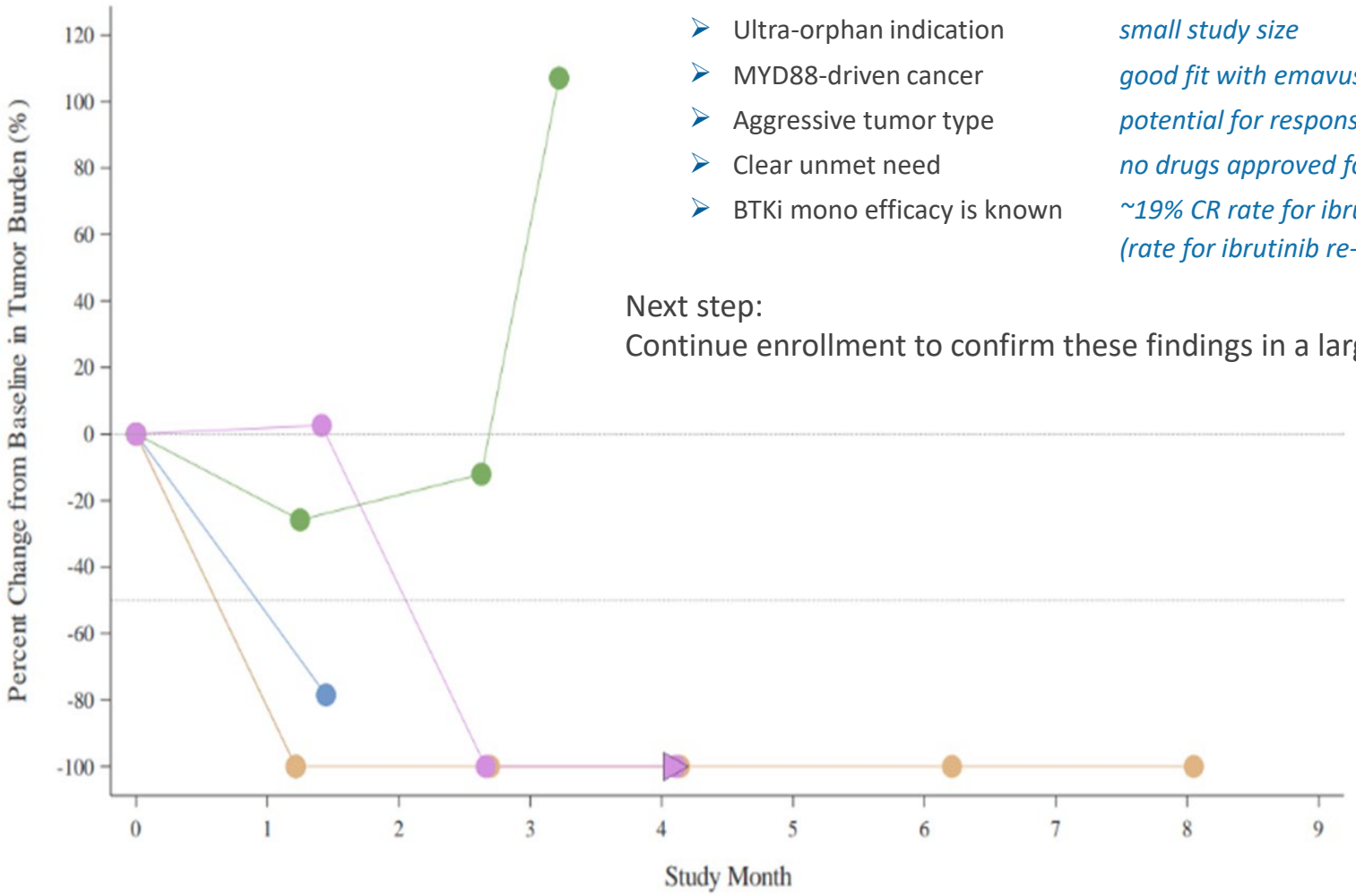
Anti-cancer activity for emavusertib/ibrutinib combination in B-cell lymphoma updated at ASH 2023

- Heavily pre-treated patients (1-10 prior lines)
- Responses achieved in patients who failed prior BTKi
- Deep responses, including 5 CRs
- Ongoing study with median treatment duration of 96 days (range 21-613 days)



PCNSL selected as lead indication for POC in lymphoma





Rationale for selection of R/R PCNSL as lead POC study in lymphoma

- Ultra-orphan indication — *small study size*
- MYD88-driven cancer — *good fit with emavusertib mechanism*
- Aggressive tumor type — *potential for responses in < 3 months*
- Clear unmet need — *no drugs approved for R/R PCNSL*
- BTKi mono efficacy is known — *~19% CR rate for ibrutinib monotherapy (rate for ibrutinib re-challenge is presumably lower)*

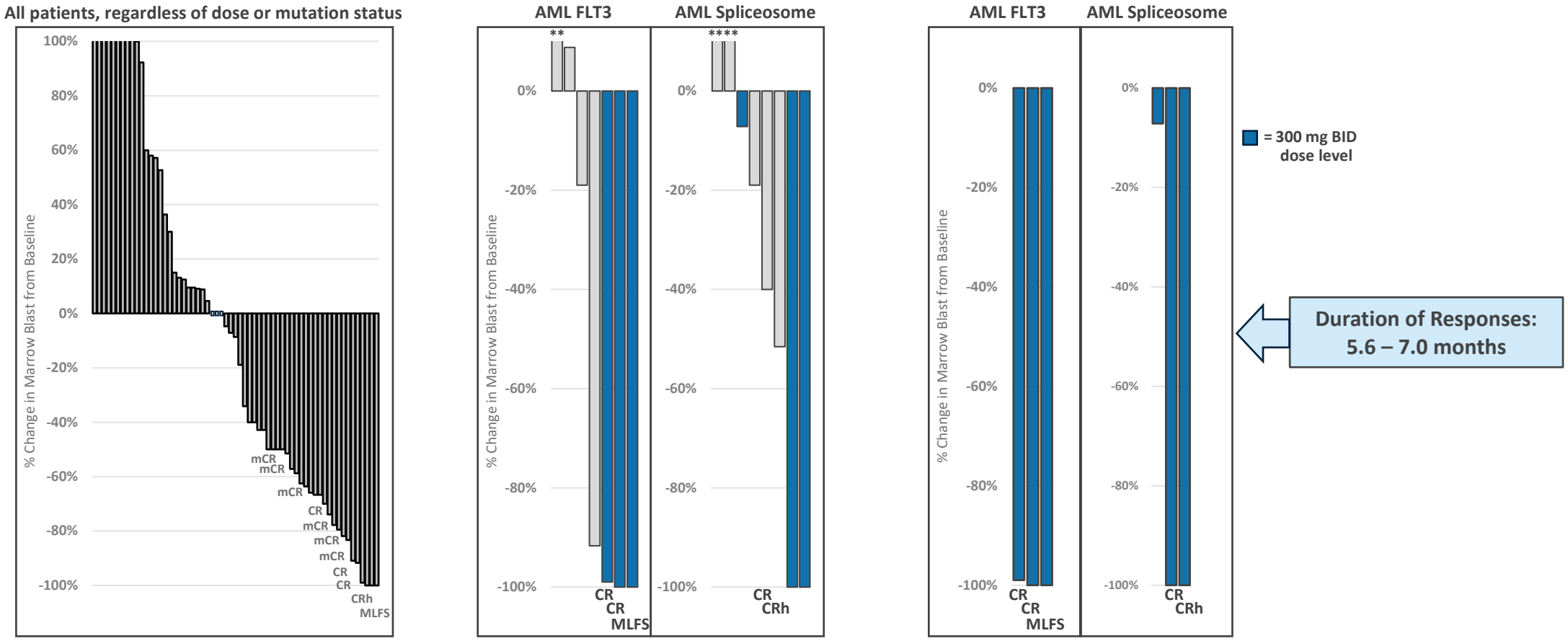
Next step:
Continue enrollment to confirm these findings in a larger number of patients

Anti-cancer activity for emavusertib monotherapy in leukemia

Demonstrate single-agent activity → **Enrich for genetically-defined population** → **Optimize for dose**

Next Step:

Continue enrollment to confirm these findings in a larger number of patients

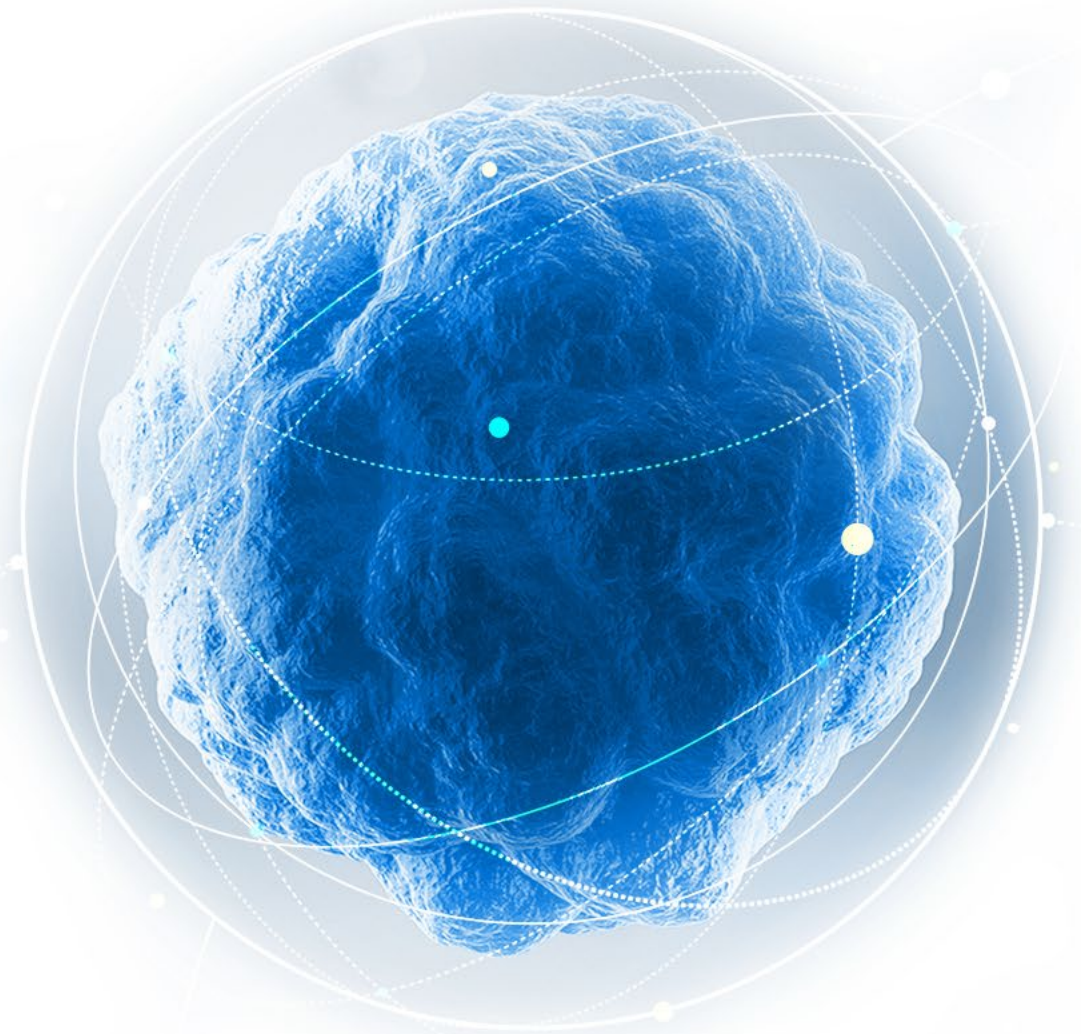


Note: 84 total R/R AML/MDS patients enrolled in monotherapy as of Feb 9, 2023 with data cutoff as of Mar 17, 2023;

* Data include all evaluable R/R patients with FLT3 and/or Spliceosome mutation and < 3 prior lines of therapy who were treated with 300 mg BID and were determined to be evaluable for objective response using baseline and post-treatment marrow assessments

** Denotes blast percent increase > 10%

Emavusertib Molecular Design



CURIS®

Emavusertib molecule specifically designed to hit multiple targets relevant in oncology

CURIS | Emavusertib

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

Emavusertib
Kinase Interaction Map

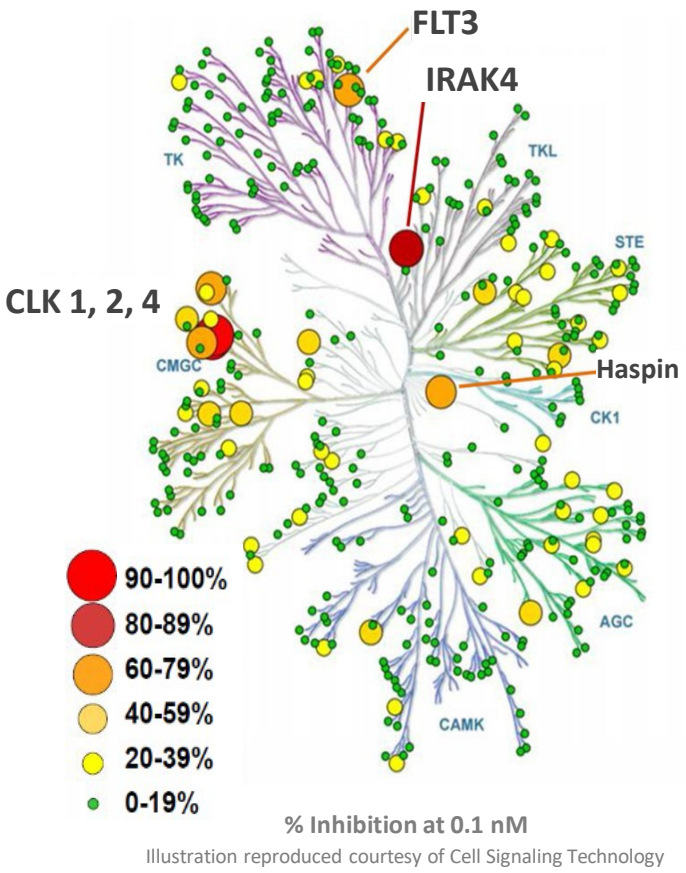


Illustration reproduced courtesy of Cell Signaling Technology

Emavusertib
Binding Affinity

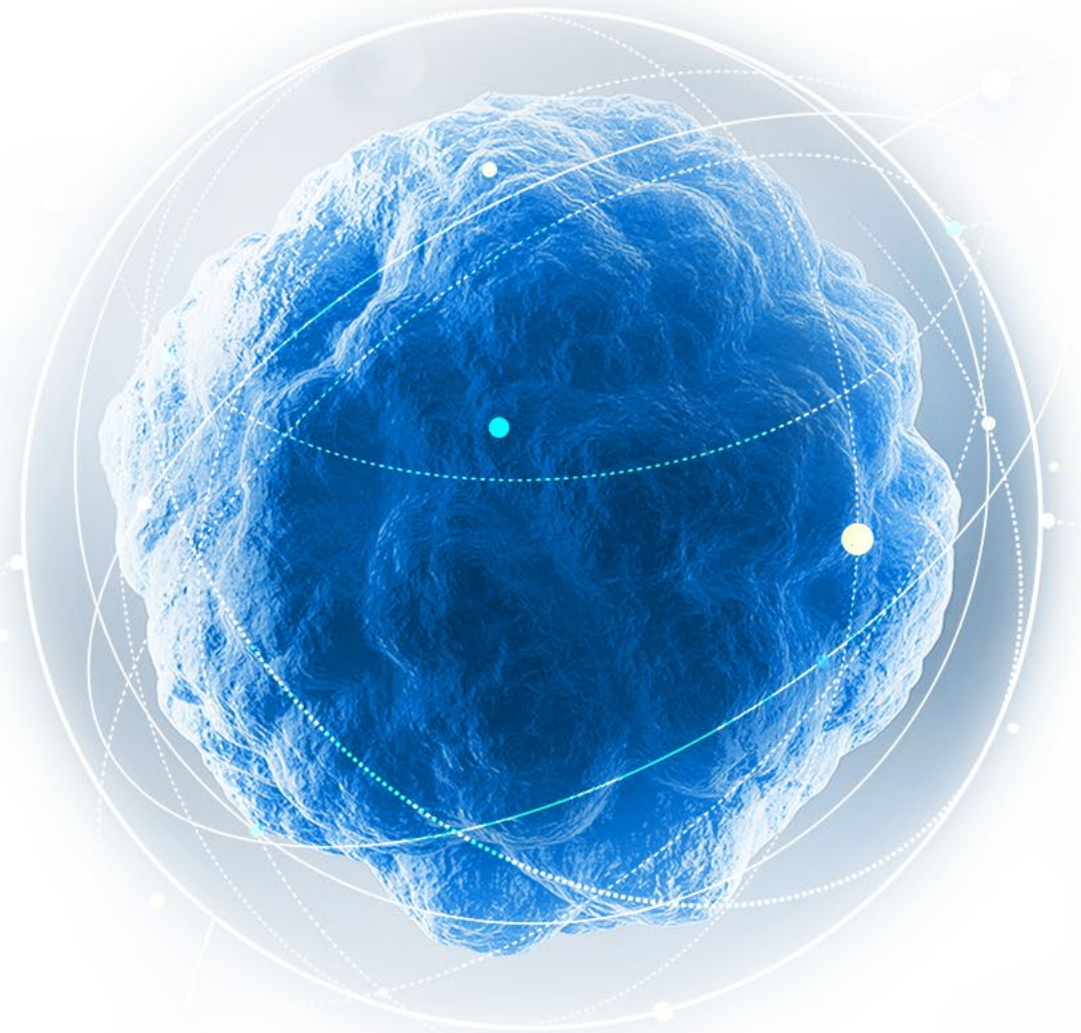
Target	K_d nM
IRAK1	12,000
IRAK2	>20,000
IRAK3	8,500
IRAK4	**23**
DYRK1A	25
FLT3 WT	**31**
FLT3 (D835H)	**5**
FLT3 (D835V)	**44**
FLT3 (D835Y)	**3**
FLT3 (ITD)	**8**
FLT3 (K663Q)	**47**
FLT3 (N841I)	**16**
Haspin (GSG2)	32
CLK1	10
CLK2	20
CLK3	>20,000
CLK4	14
TrkA	130

DiscoverX Kinase Panel
(378 kinases screened)

high binding affinity to IRAK4
>97% inhibition achieved at Ph2 dose concentrations

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

Emavusertib in Leukemia

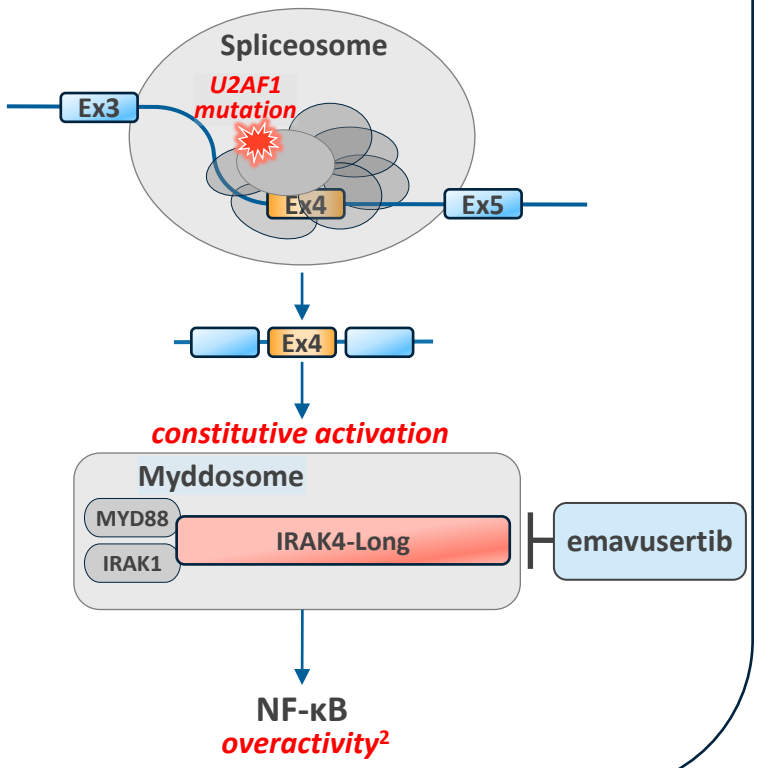


CURIS®

The two primary targets of emavusertib are independent drivers of cancer

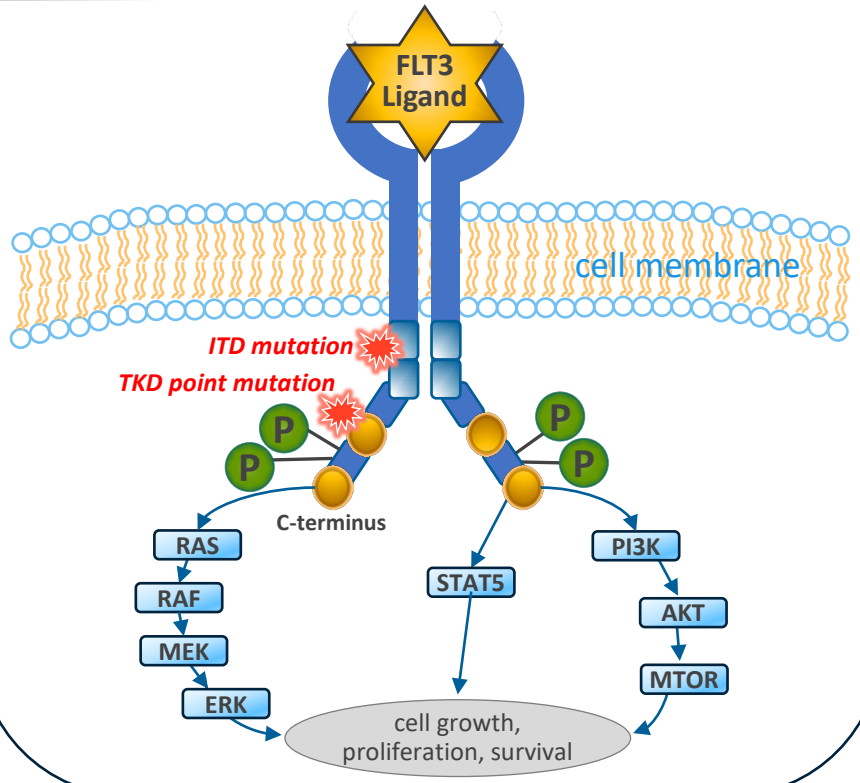
IRAK4

spliceosome mutations drive overexpression of IRAK4-L, 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

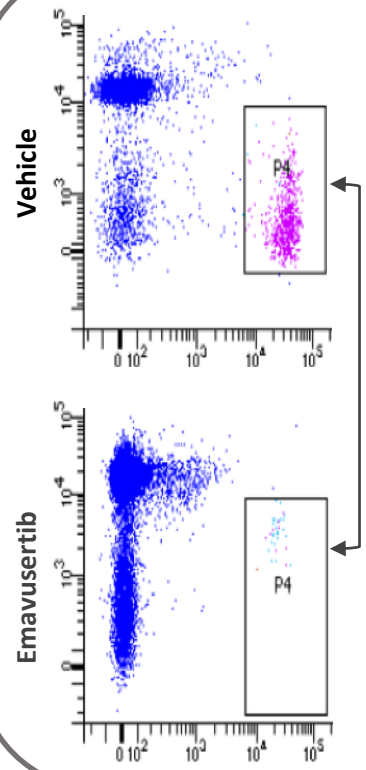


1) Guillamot et al. Nat Cell Biol 2019; 2) Smith et al. Nat Cell Biol 2019; 3) American Cancer Society, Cancer Facts & Figures 2020; 4) Leukemia & Lymphoma Society, Facts and Statistics Overview

Rationale for monotherapy vs. combination

Targeted Population

Monotherapy in IRAK4



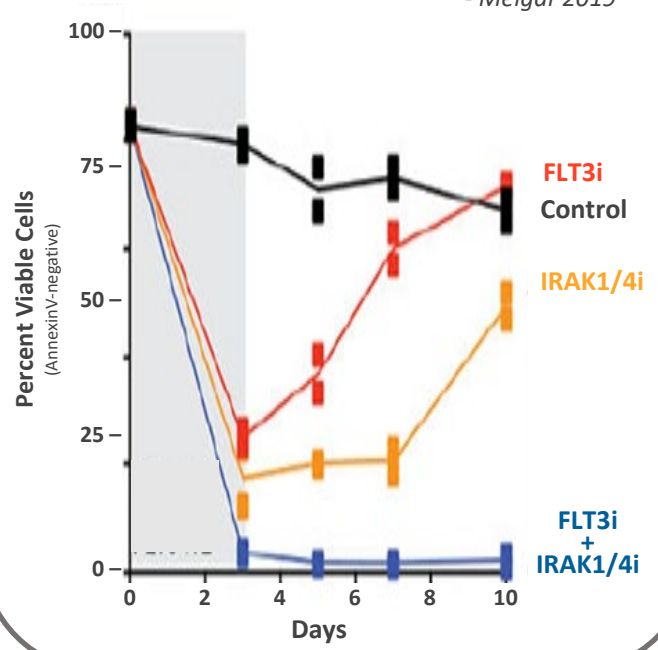
clear reduction of leukemic blasts in models with high IRAK4-L expression

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

Targeted Population

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 2019

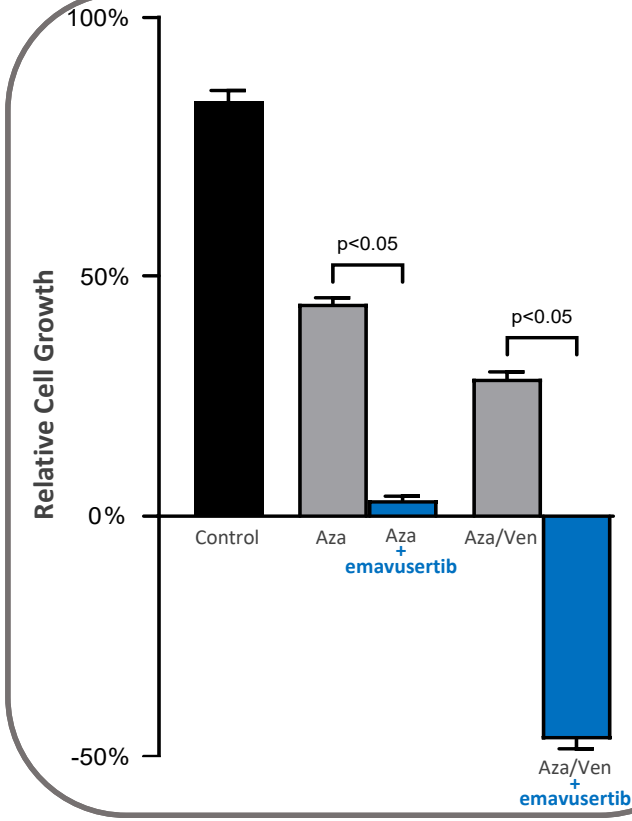


IRAK/FLT3 combination demonstrates synergy in pre-clinical studies[2]

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

All Comers

Combination with Aza/Ven



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

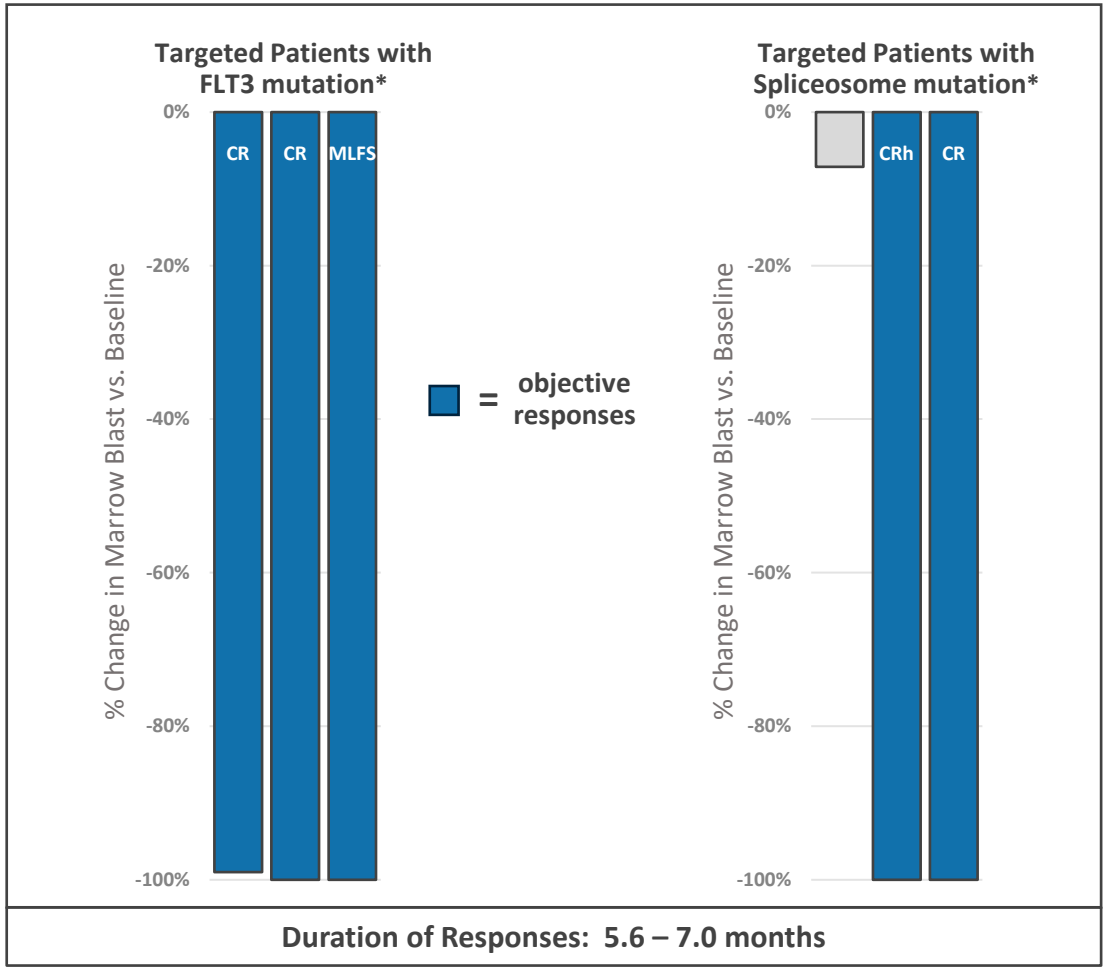
AML cell lines treated for 96 hrs (values presented as mean ± SE)

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

Anti-cancer activity for emavusertib monotherapy in R/R AML

- Safety profile established in dose escalation study of emavusertib monotherapy in 84 patients
- Strong single-agent anti-cancer activity observed
- Strongest signal observed where expected (in patients with FLT3 and/or Spliceosome mutation)
- RP2D established at 300 mg BID
- Next step:
  Continue enrollment to confirm these findings in a larger number of patients

Reduction in Marrow Blast
(for target populations dosed at 300 mg BID RP2D)



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Emavusertib is a novel molecule demonstrating single-agent and combination anti-cancer activity

- IRAK4 is a novel and important target in AML
  - Primary driver of disease in > 50% of AML patients[1]
  - Secondary or tertiary driver in nearly all patients

- As a single agent:
  Emavusertib addresses the two largest genetically-defined populations in AML: patients with IRAK4-L and FLT3 mutation[2]

- In combination with standard of care:
  Emavusertib addresses all comers with its novel blockade of MYD88 and the TLR Pathway[3]

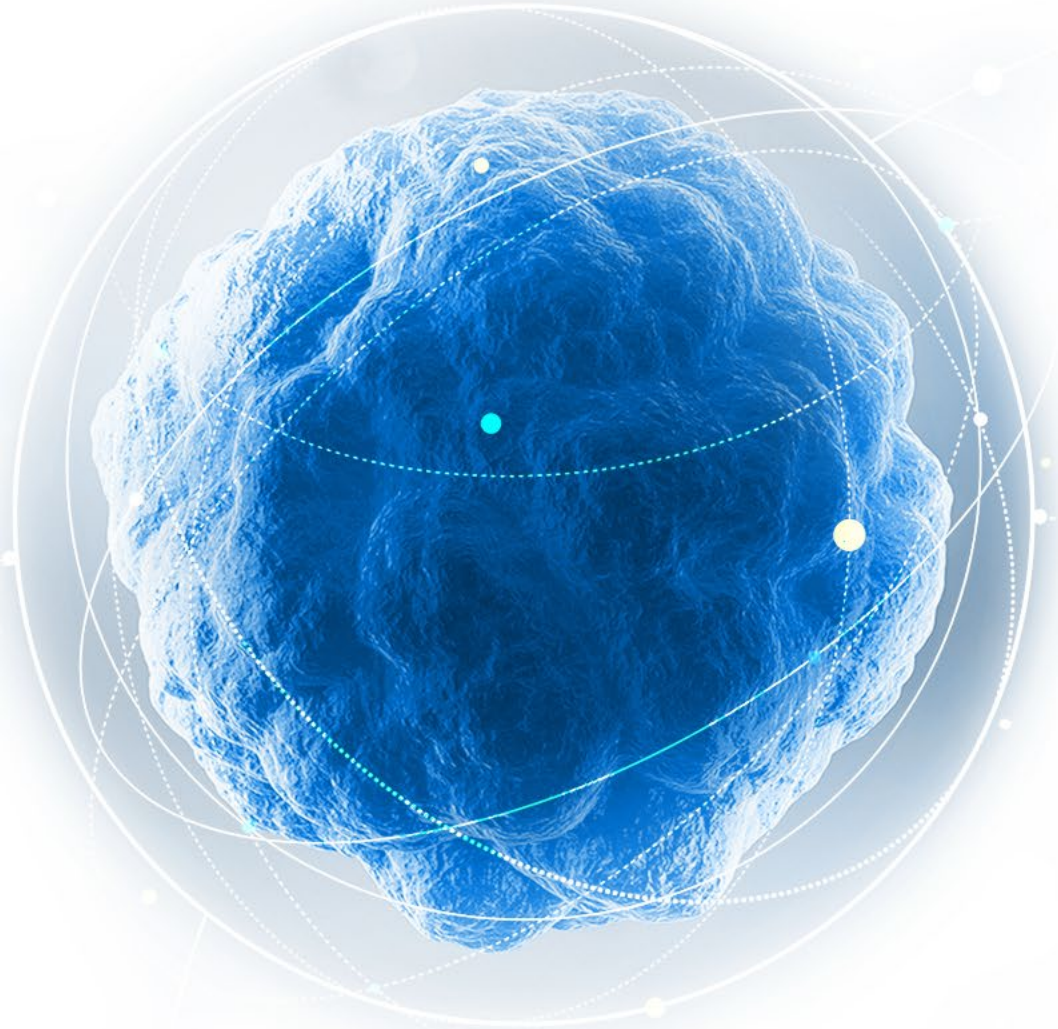
TakeAim Leukemia Study

Next Step: Continue enrolling in

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

1) Smith et al. Nat Cell Biol 2019; 2) Saygin, et al. J Hematol Oncol. 2017 Apr 18; 3) Melgar, Sci Transl Med. 2019; *3) Curis AML MDS poster, EHA 2021*

Emavusertib in Lymphoma



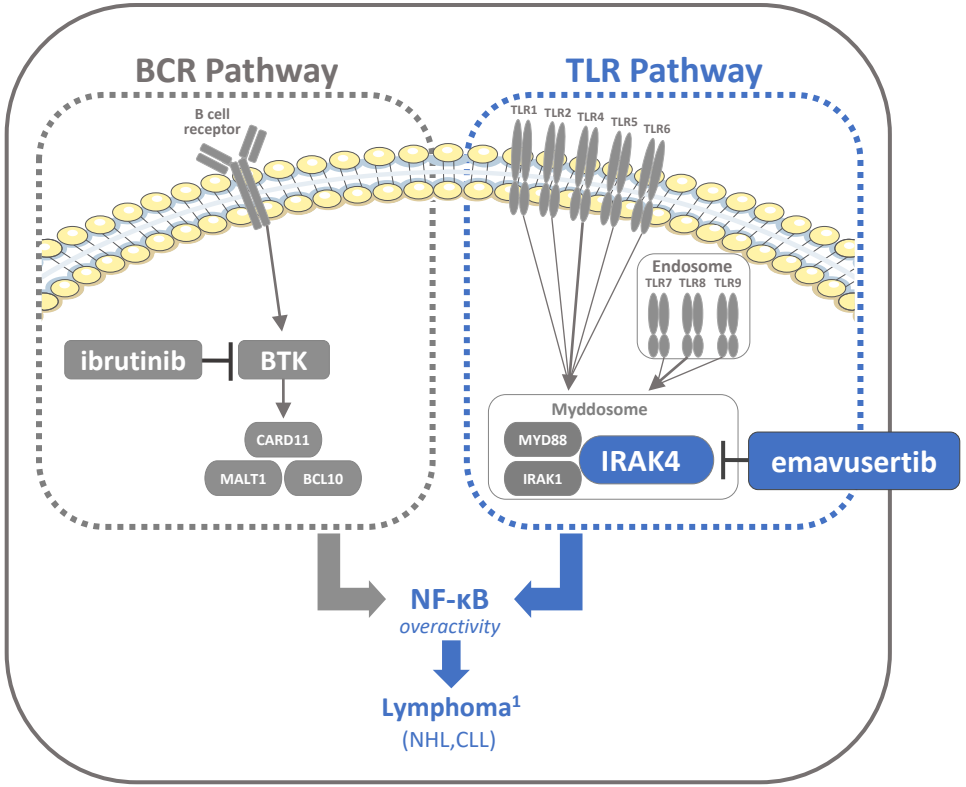
CURIS®

Combination therapy provides complementary inhibition of two pathways driving NF-κB

CURIS Lymphoma

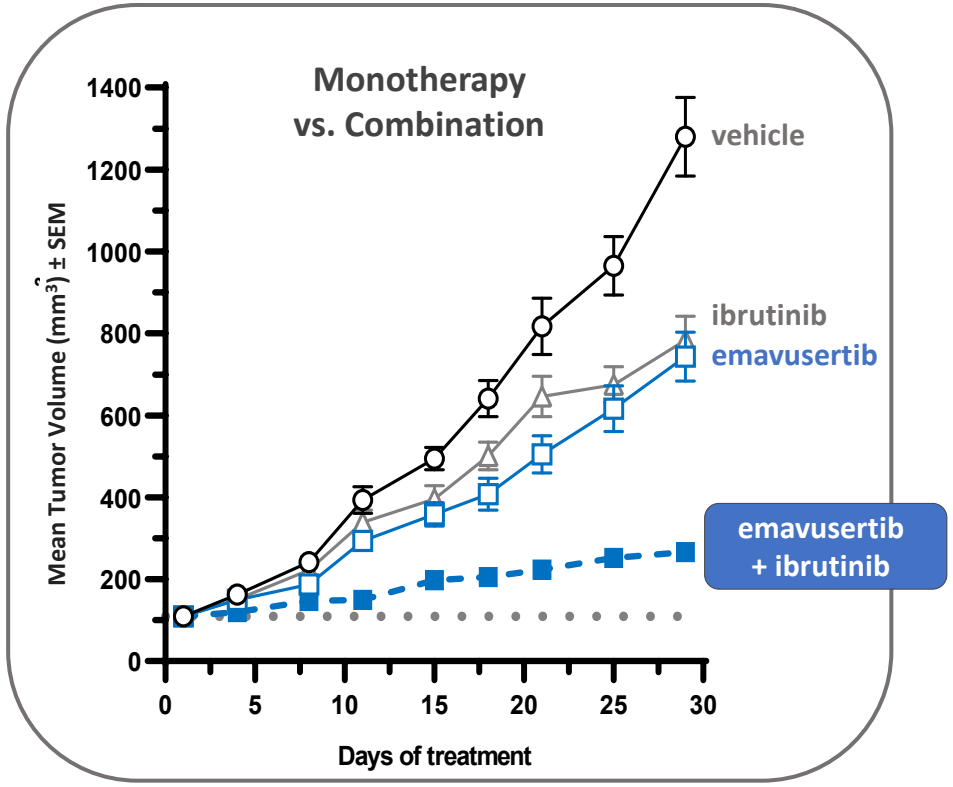
Two Pathways Drive Lymphoma

BCR and TLR Pathways independently drive NF-κB overactivity (and NF-κB overactivity drives Lymphoma)



Clinical Strategy: Block Both Pathways

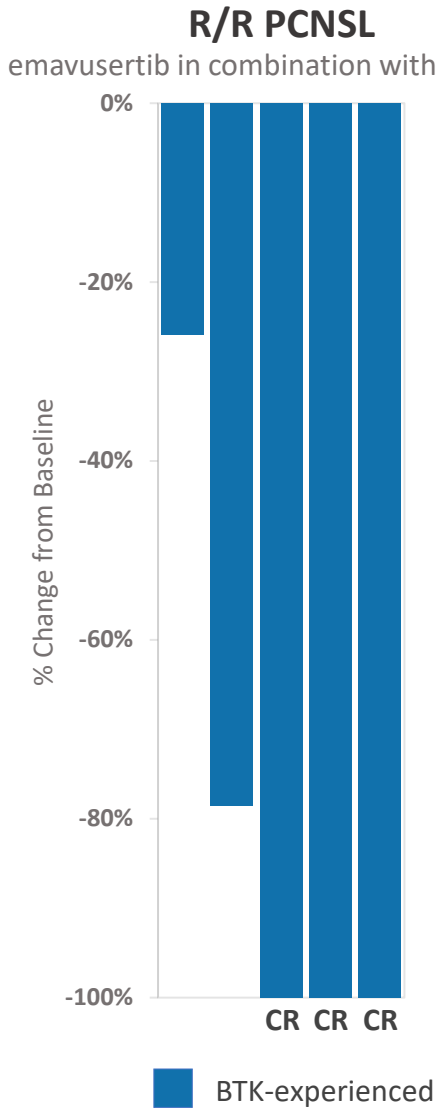
Blocking both the TLR (with IRAK4) and BCR (with BTK) Pathways drives tumor reduction better than blocking either one alone

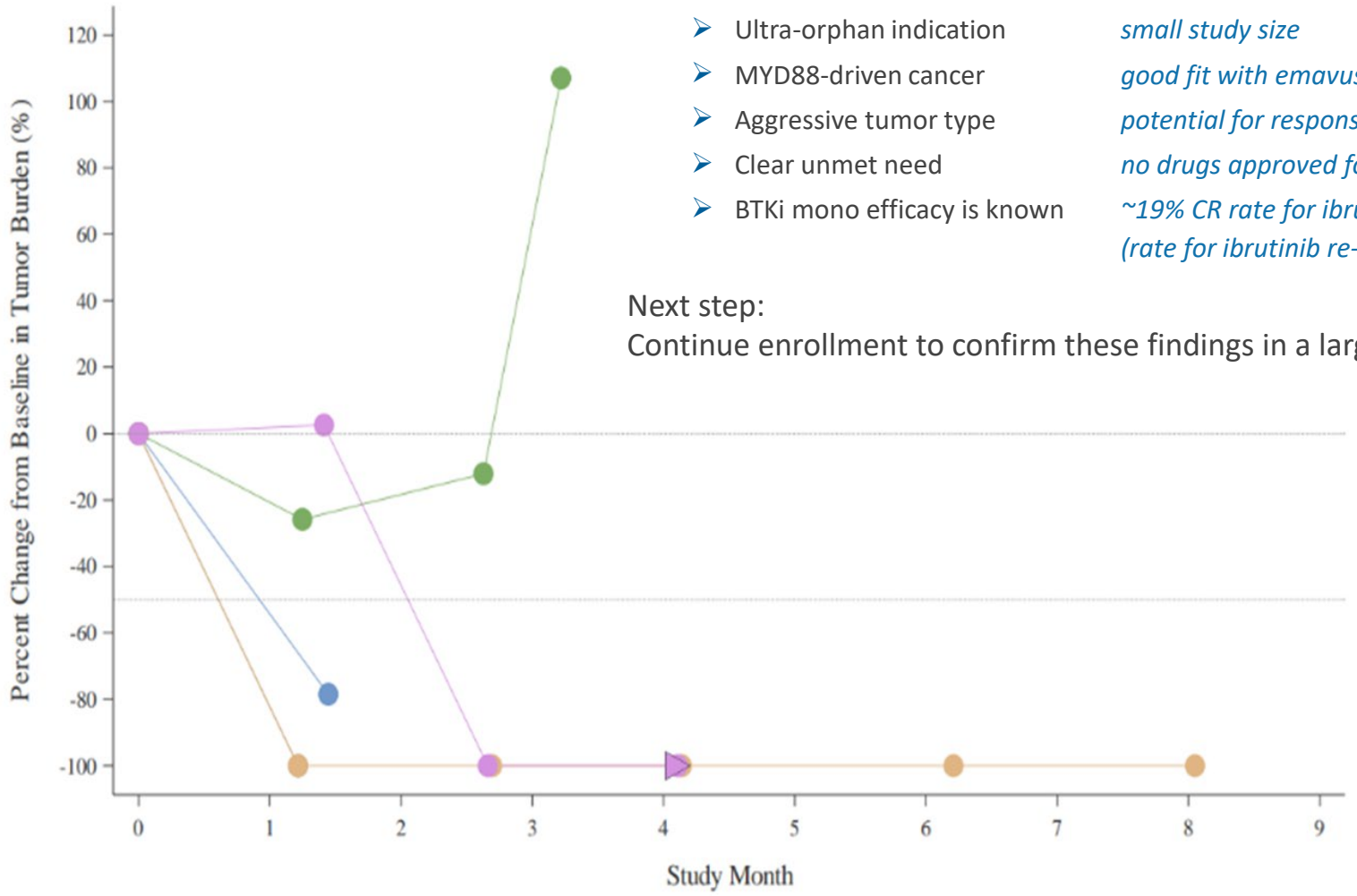


1) IMBRUVICA Package Insert. Rev 08/2018

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

PCNSL selected as lead indication for POC in lymphoma





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CURIS Lymphoma

- 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 Lymphoma Study

Next Step: Continue enrolling in

- *Combination study of emavusertib with ibrutinib in R/R PCNSL*

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End of Corporate Presentation