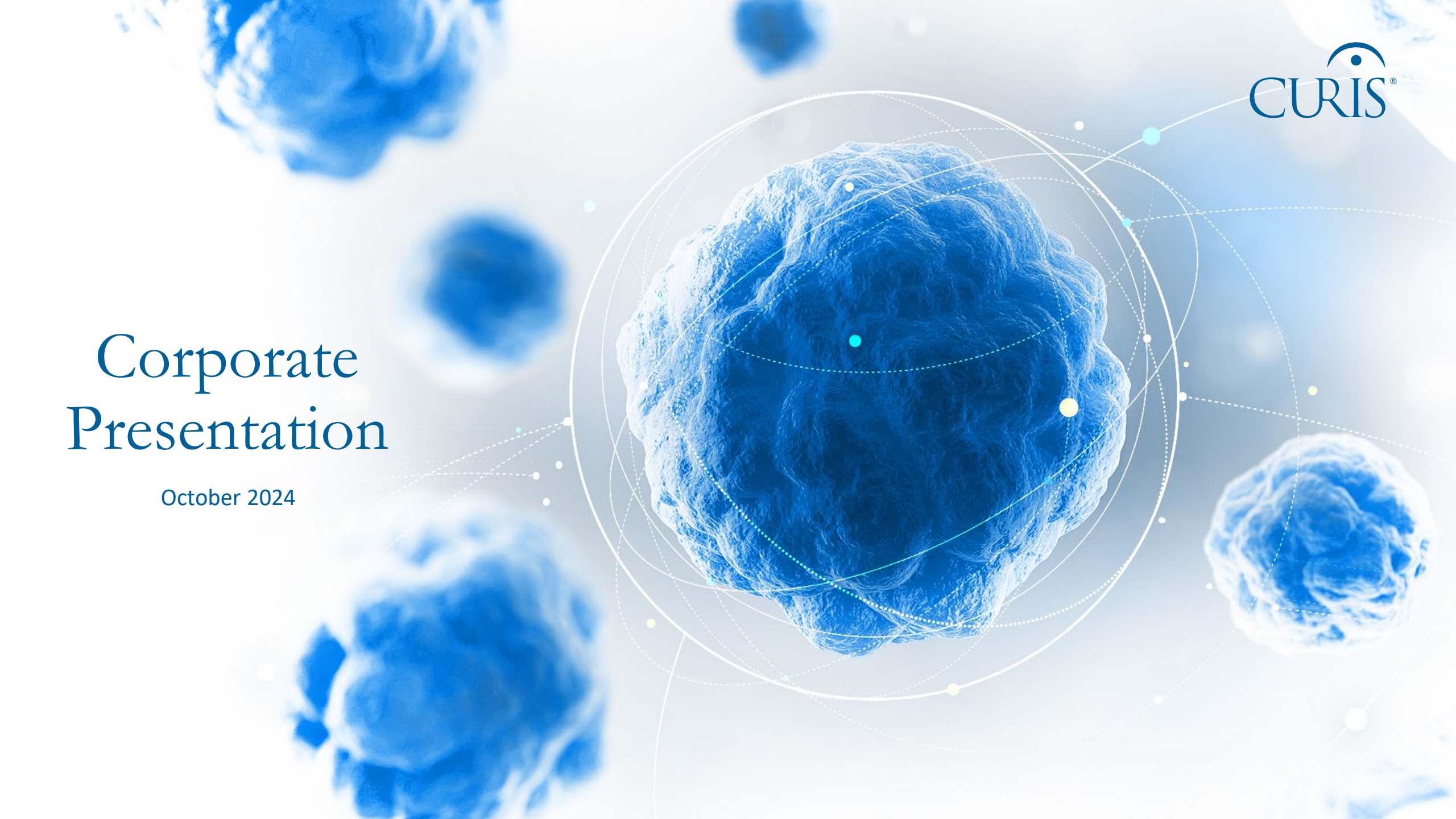


# Corporate Presentation

October 2024



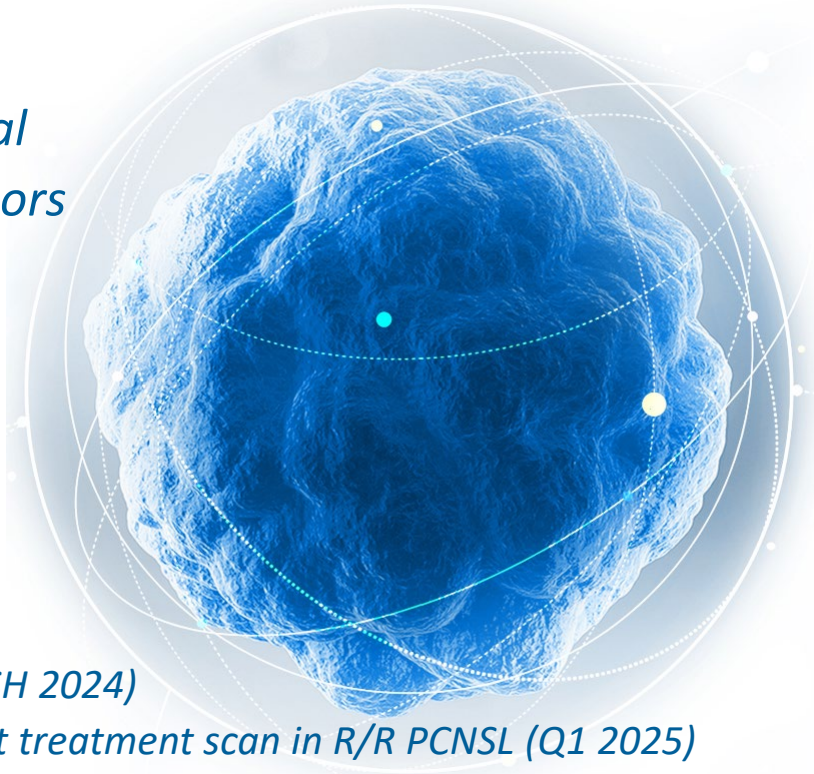
# Cautionary note regarding forward looking statements and disclaimers

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 and the Company's Quarterly Reports on Form 10-Q for the quarters ended March 31, 2024 and June 30, 2024, which are available on the SEC website at [www.sec.gov](http://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.

# Emavusertib is a potential first-to-market IRAK 4 inhibitor

- *Being evaluated in Phase 1/2 clinical studies in NHL, AML, and Solid Tumors*
- *Potential for monotherapy and combination use in NHL, AML and Solid Tumors*
- *Near-Term Milestones*
  - *Data in ~20 pts in R/R FLT3m AML (ASH 2024)*
  - *Data in ~15-20 pts with at least 1 post treatment scan in R/R PCNSL (Q1 2025)*
  - *Initial safety data for triplet combination in frontline AML (Q1 2025)*



***Demonstrated safety and single-agent activity***

***Demonstrated synergy with BTKi, HMA, BCL2i***

***Broad opportunity in NHL, AML, and Solid Tumors***

# Broad Market Opportunity in NHL, AML, and Solid Tumors

## Significant market opportunities in current development programs

	PCNSL	FLT3m	AML
<b>US Incidence per 100K</b>	<b>0.5<sup>1</sup></b>	<b>1.3<sup>2</sup></b>	<b>4.2<sup>3</sup></b>
	<u>Newly Diagnosed Per Year</u>		
US	1,700 <sup>1</sup>	6,000 <sup>2</sup>	20,000 <sup>3</sup>
Big 5 Europe/Canada	1,800 <sup>1</sup>	5,200 <sup>4</sup>	17,000 <sup>4</sup>
Japan/China	<u>7,700<sup>1</sup></u>	<u>12,700<sup>4</sup></u>	<u>41,200<sup>4</sup></u>
<b>Total</b>	<b>11,200</b>	<b>23,900</b>	<b>78,200</b>

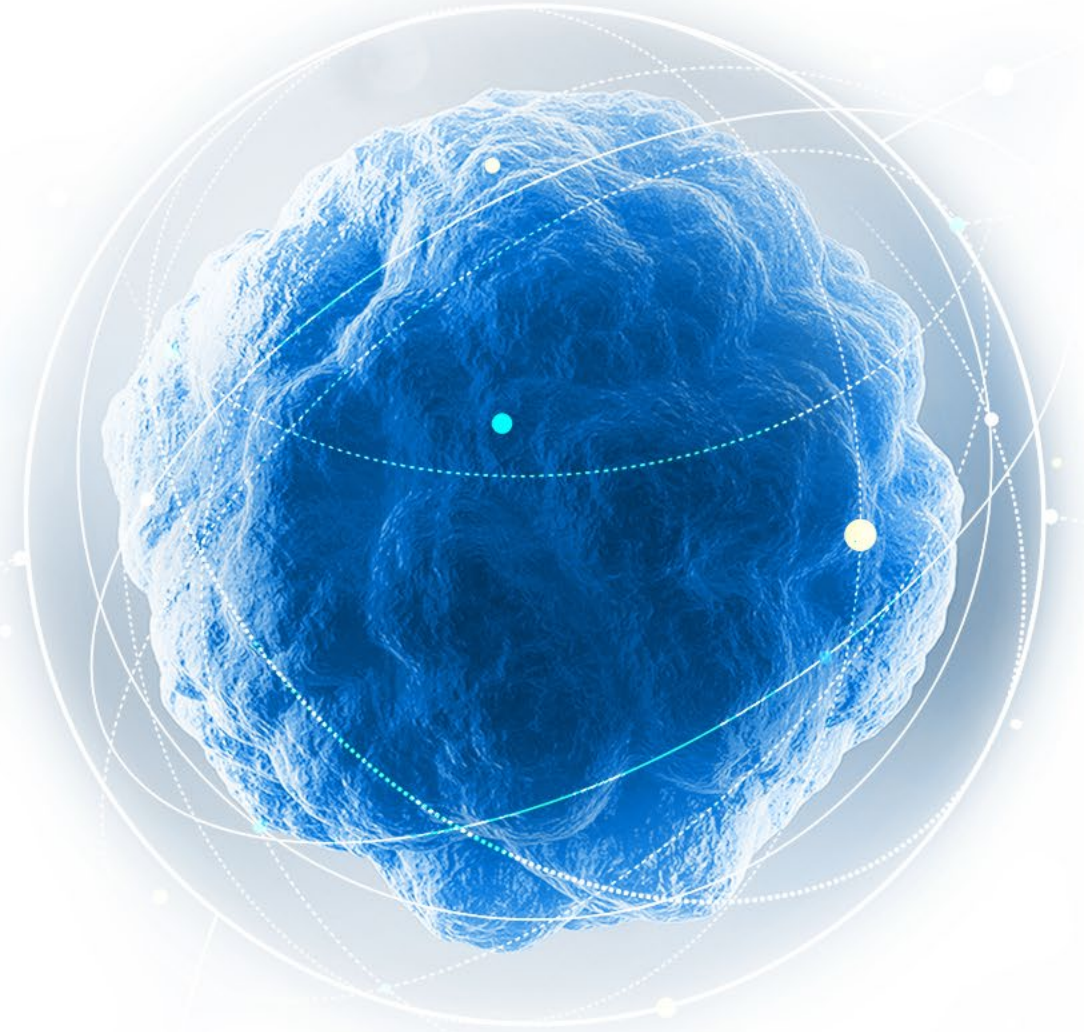
1 – Derived from incident rate in Lv Ther Adv Hematol 2022 and 2022 country population [data.worldbank.org]  
 2 – Derived from total AML cases (see footnote 4); FLT3m represents 30% of newly diagnosed AML cases [Daver Leukemia 2019]  
 3 – Vakiti Acute Myeloid Leukemia 2023 [www.ncbi.nlm.nih.gov]  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

## Additional opportunities with NHL expansion

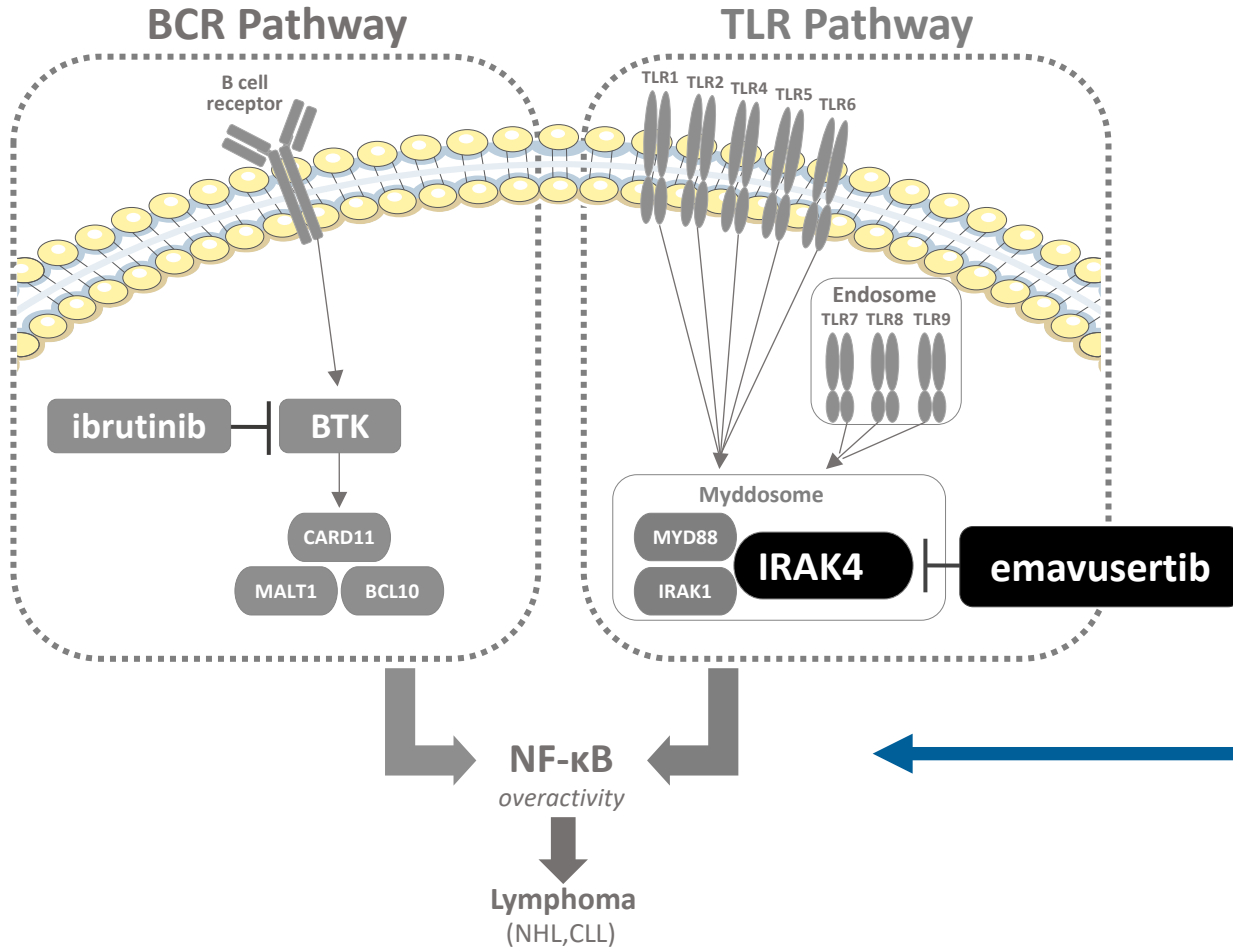
WM	MCL	MZL	ABC-DLBCL
<b>0.5<sup>5</sup></b>	<b>0.5<sup>6</sup></b>	<b>1.5<sup>7</sup></b>	<b>2.0<sup>8</sup></b>
	<u>Newly Diagnosed Per Year</u>		
1,700 <sup>5</sup>	1,700 <sup>6</sup>	5,000 <sup>7</sup>	6,800 <sup>8</sup>
1,800 <sup>5</sup>	1,800 <sup>6</sup>	5,500 <sup>7</sup>	7,500 <sup>8</sup>
<u>7,700<sup>5</sup></u>	<u>7,700<sup>6</sup></u>	<u>23,000<sup>7</sup></u>	<u>31,400<sup>8</sup></u>
<b>11,200</b>	<b>11,200</b>	<b>33,500</b>	<b>45,700</b>

5 – Derived from incident rate in <https://rarediseases.org/rare-diseases/waldenstroms-macroglobulinemia/#affected> and 2022 country population [data.worldbank.org].  
 6 – Derived from incident rate in <https://www.ncbi.nlm.nih.gov/books/NBK536985/> and 2022 country population [data.worldbank.org].  
 7 – Derived from incident rate in Kalashnikov, Blood Cancer Journal, April 2023 and 2022 country population [data.worldbank.org].  
 8 – Derived from incident rates in NHL incident rate of 18.6 per 100,000 (seer.cancer.gov) with DLBCL representing 25% of NHL per <https://www.ncbi.nlm.nih.gov/books/NBK557796/>. ABC represents 44% per letters to the editor, haematologica, 2011, 96 and 2022 country population [data.worldbank.org].

# Emavusertib in NHL



# Emavusertib's Mechanism in NHL



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

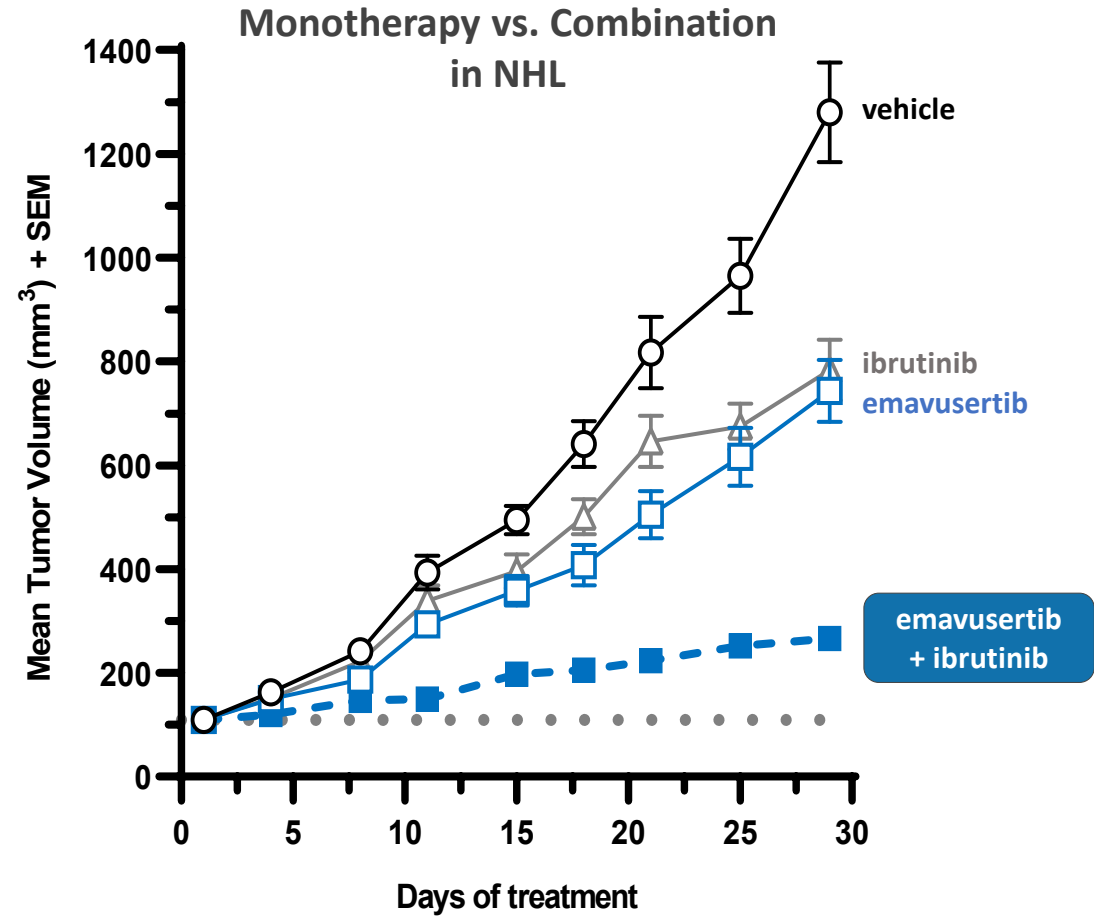
*combining IRAK4i and BTKi  
enables dual blockade of NF-κB*

# Emavusertib is synergistic with BTKi in NHL

## emavusertib + BTKi

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

- IRAK4i synergizes with BTKi to promote killing of **ABC-DLBCL**<sup>1</sup>
- Concurrent treatment with IRAKi and BTKi was significantly more potent in patient **CLL** cells than either drug alone<sup>2</sup>
- Data suggest IRAK4 as a novel treatment target for **CLL**; inhibition of IRAK4 blocks survival and proliferation of CLL cells<sup>3</sup>



Preclinical data for emavusertib and ibrutinib in OCI-Ly10 model (Booher et al., IWWM 2018)

<sup>1</sup> Kelly J Exp Med 2015, <sup>2</sup> Dadashian Ca Res 2019, <sup>3</sup> Giménez Leukemia 2020

# Strategy in NHL

**1****Demonstrate safety**

31 patients<sup>1</sup> treated in TakeAim Lymphoma Ph 1b study, acceptable safety profile established, no overlapping dose-limiting toxicity with ibrutinib

**2****Demonstrate single-agent activity**

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

**3****Pursue fastest path to 1<sup>st</sup> 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



# Emavusertib safety profile in NHL<sup>1</sup>

- 31 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 Ema + Ibr (N=6)	200 mg BID Ema + Ibr (N=18)	300 mg BID Ema + Ibr (N=7)	Total (N=31)
	n (%)	n (%)	n (%)	n (%)
# patients having grade 3+ TRAEs	4 (67)	8 (44)	6 (86)	18 (58)
Lipase increased	2 (33)	1 (6)		3 (10)
Neutropenia	2 (33)	1 (6)		3 (10)
Platelet count decreased		2 (11)	1 (14)	3 (10)
Alanine aminotransferase increased		1 (6)	1 (14)	2 (6.5)
Amylase increased	2 (33)			2 (6.5)
Aspartate aminotransferase increased		1 (6)	1 (14)	2 (6.5)
Fatigue		1 (6)	1 (14)	2 (6.5)
Hyponatraemia		2 (11)		2 (6.5)

1 – As of July 10, 2024

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 NHL

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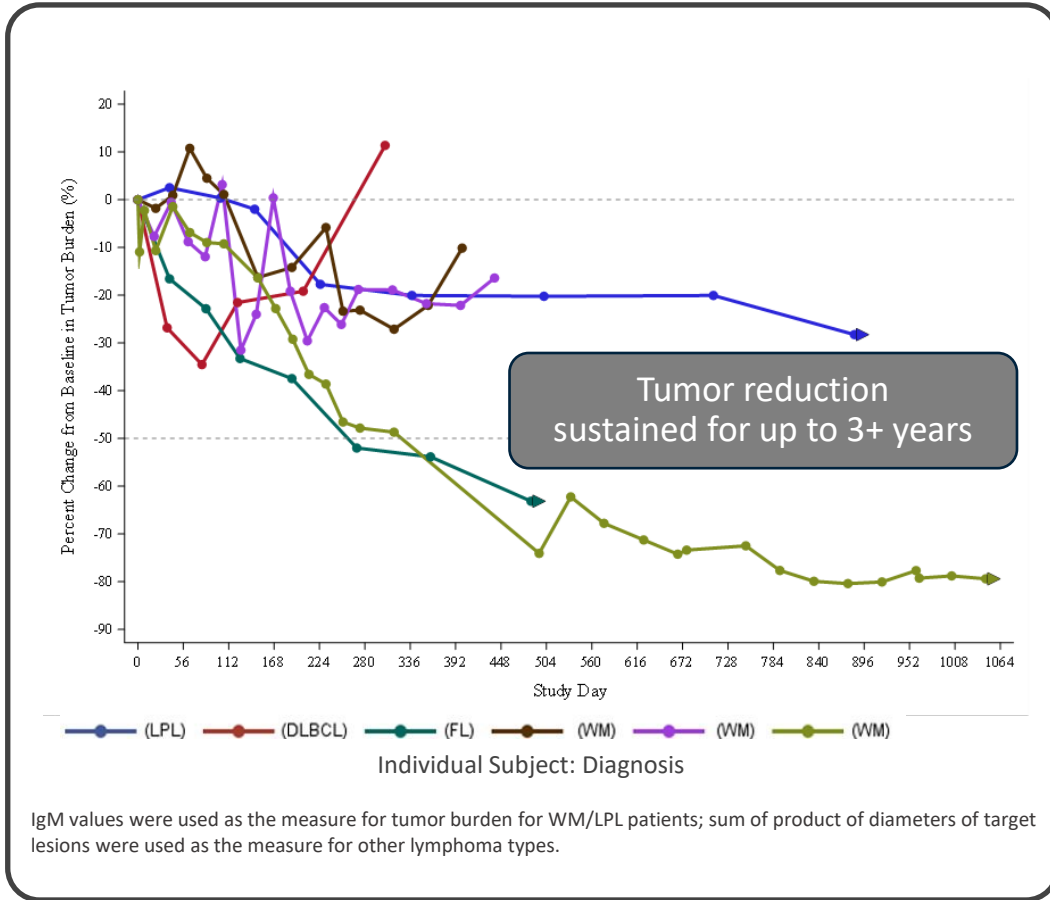
Identify orphan indication with clear unmet need that is addressable with emavusertib's novel mechanism of action

**4****Pursue partnership to expand across NHL**

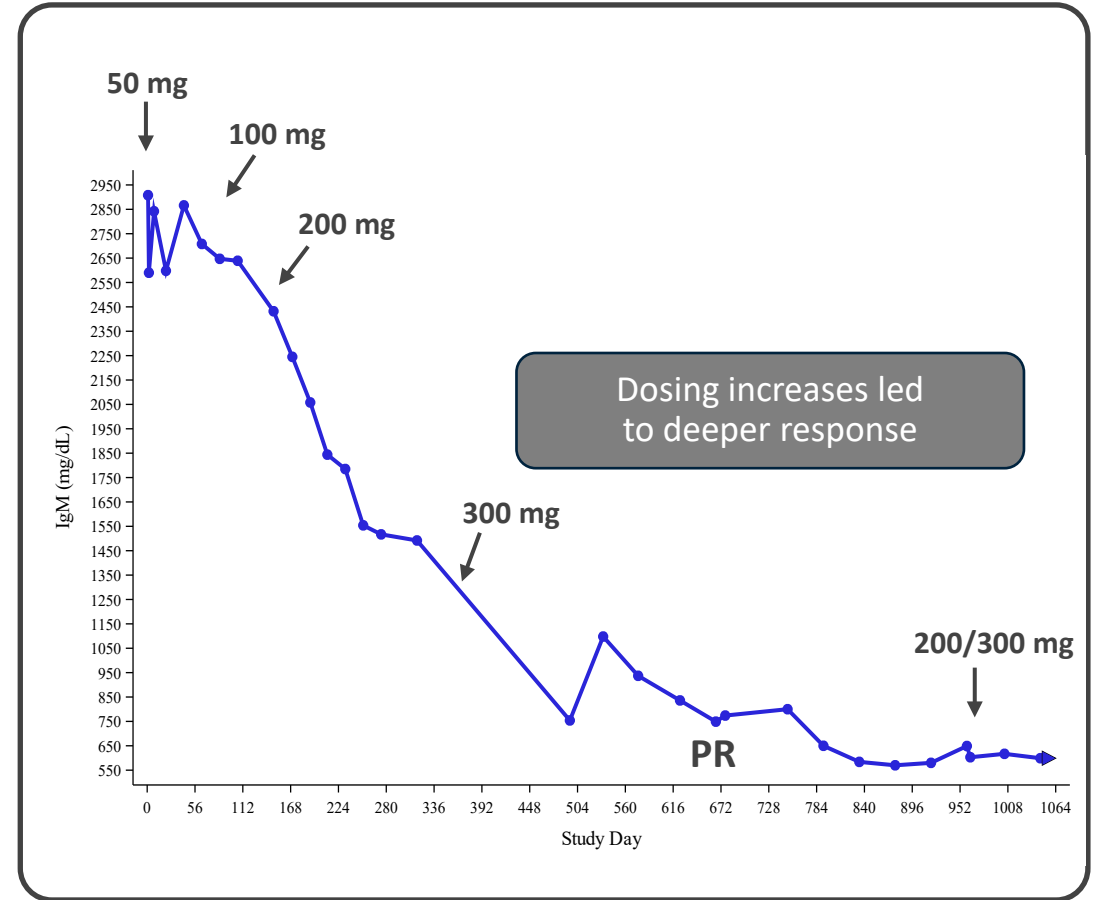
Significant resources will be required to execute large clinical studies across multiple NHL subtypes and prepare for potential commercial launch

# Single-agent activity demonstrated in NHL

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



**Case Study in Dose Response (WM patient)**



2022 IWWW Conference Presentation

Abbreviation: Partial Response (PR)

# Strategy in NHL

**1****Demonstrate safety**

31 patients<sup>1</sup> treated in TakeAim Lymphoma Ph 1b study, acceptable safety profile established, no overlapping dose-limiting toxicity with ibrutinib

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

**3****Pursue fastest path to 1<sup>st</sup> label in R/R line patients**

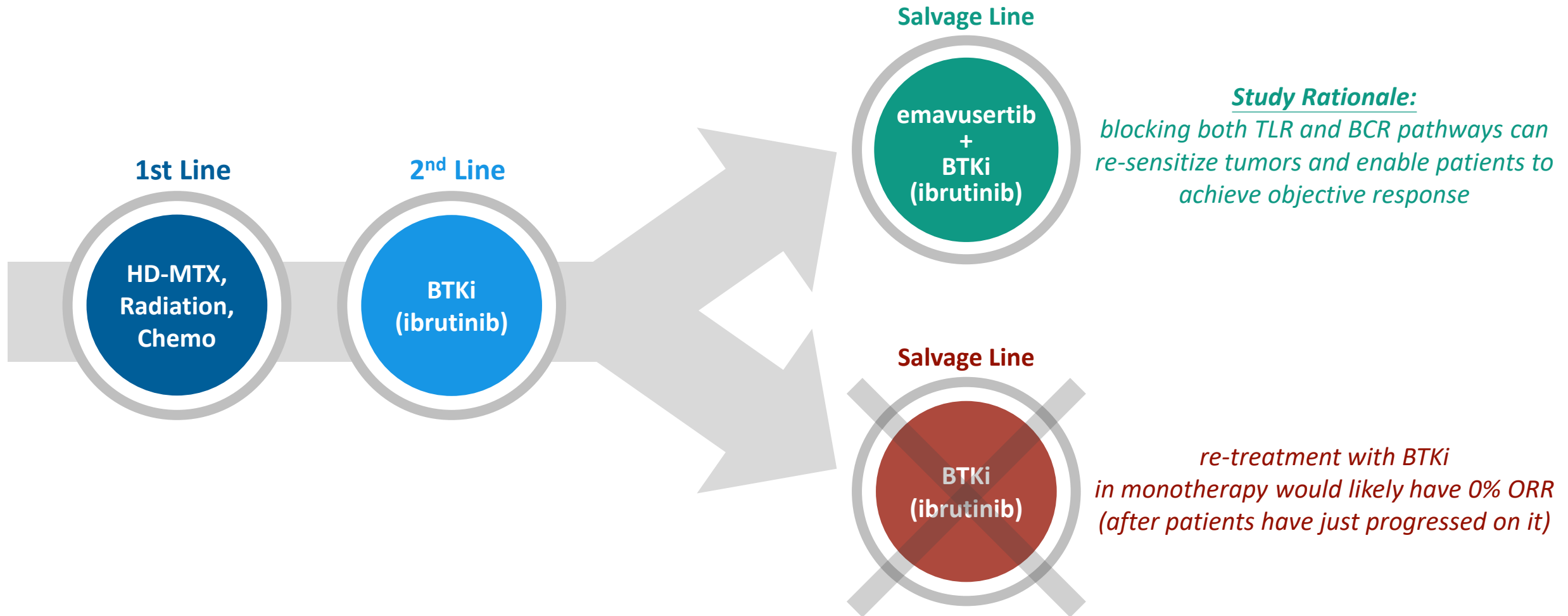
Identify orphan indication with clear unmet need that is addressable with emavusertib's novel mechanism of action

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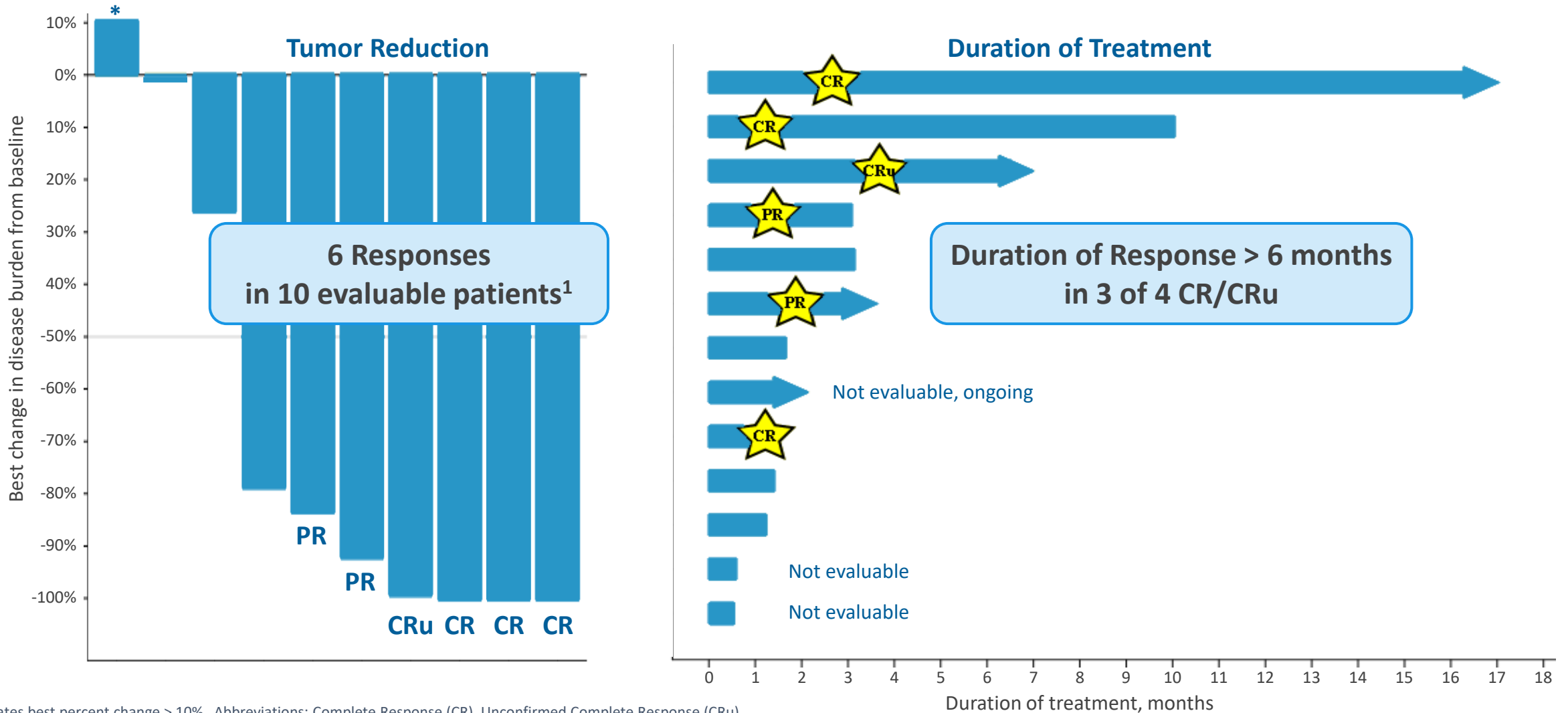
# R/R PCNSL selected for 1<sup>st</sup> NHL indication

*PCNSL is an orphan NHL indication, where no drugs are approved in salvage-line therapy*



# Encouraging clinical data in R/R PCNSL

Results for patients treated with emavusertib + ibrutinib, after they have progressed on prior BTKi



\*Indicates best percent change > 10%. Abbreviations: Complete Response (CR), Unconfirmed Complete Response (CRu)

<sup>1</sup>Evaluable patients are those who have completed at least one cycle of treatment and received at least one post-treatment assessment.

# Strategy in NHL

**1****Demonstrate safety**

31 patients<sup>1</sup> treated in TakeAim Lymphoma Ph 1b study, acceptable safety profile established, no overlapping dose-limiting toxicity with ibrutinib

**2****Demonstrate single-agent activity**

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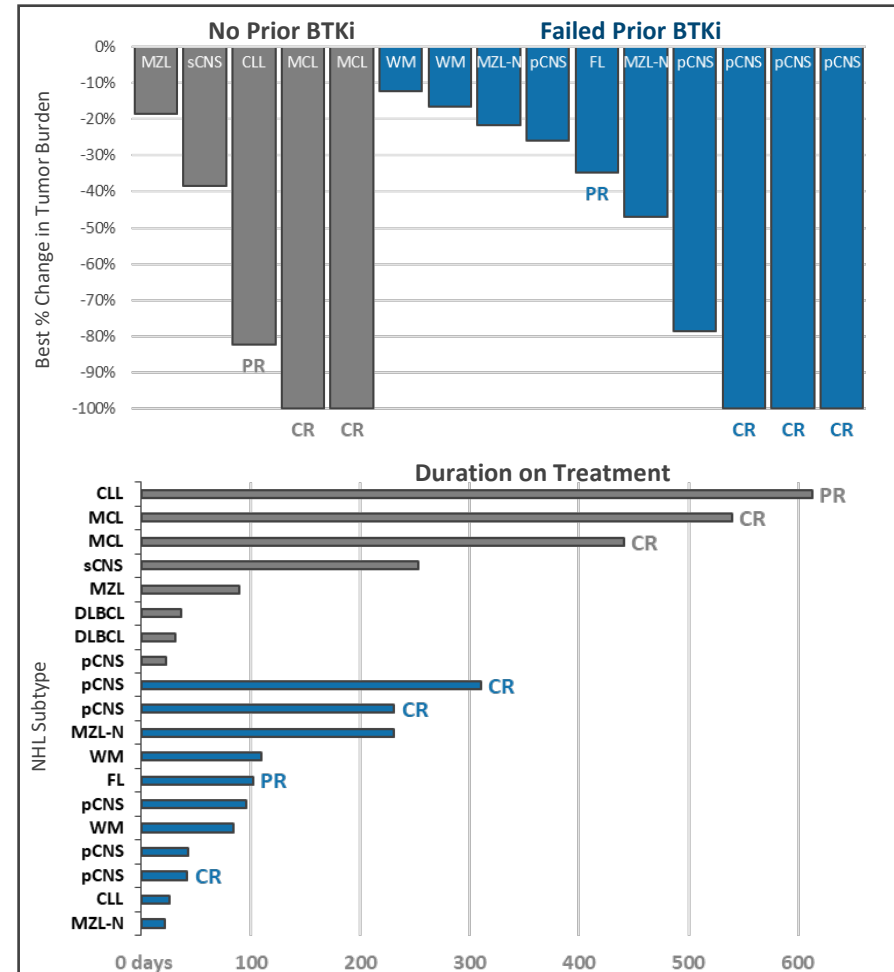
Significant resources will be required to execute clinical studies across multiple NHL subtypes and prepare for potential commercial launch

# Anti-cancer activity shown across multiple NHL subtypes

*Supports the mechanism of emavusertib + BTKi combination in additional NHL subtypes*

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

All patients (n=19), multiple NHL subtypes



Data from TakeAim Lymphoma Clinical Outcomes ASH 2023 poster



# Opportunities in additional NHL subtypes

*Emavusertib's inhibition of NF-κB via IRAK4, enables broad therapeutic potential in NHL*

Tumor	(US only) Incidence	Key Targets of Interest	SOC
<b>ABC-DLBCL</b>	2 per 100,000 ~ 6,800 patients	IRAK4, MYD88, CD79, NF-κB	R-CHOP
<b>PCNSL</b>	0.5 per 100,000 ~ 1,700 patients	IRAK4, MYD88, CD79, NF-κB	Chemotherapy, HDMTX
<b>WM</b>	0.5 per 100,000 ~ 1,700 patients	IRAK4, MYD88, CD79, NF-κB	Chemotherapy, αCD20
<b>MCL</b>	0.5 per 100,000 ~ 1,700 patients	BCR and TLR pathway activation	BTKi, αCD20
<b>MZL</b>	1.5 per 100,000 ~ 5,000 patients	IRAK4, MYD88, CARD11, NF-κB	Chemotherapy, αCD20, RT
<b>CLL</b>	5 per 100,000 ~ 18,700 patients	NF-κB	BTKi, αCD20

**Emavusertib's inhibition of IRAK4 suppresses NF-κB pathway activity**, offering a therapeutic strategy for other NHL indications driven by NF-κB overactivity

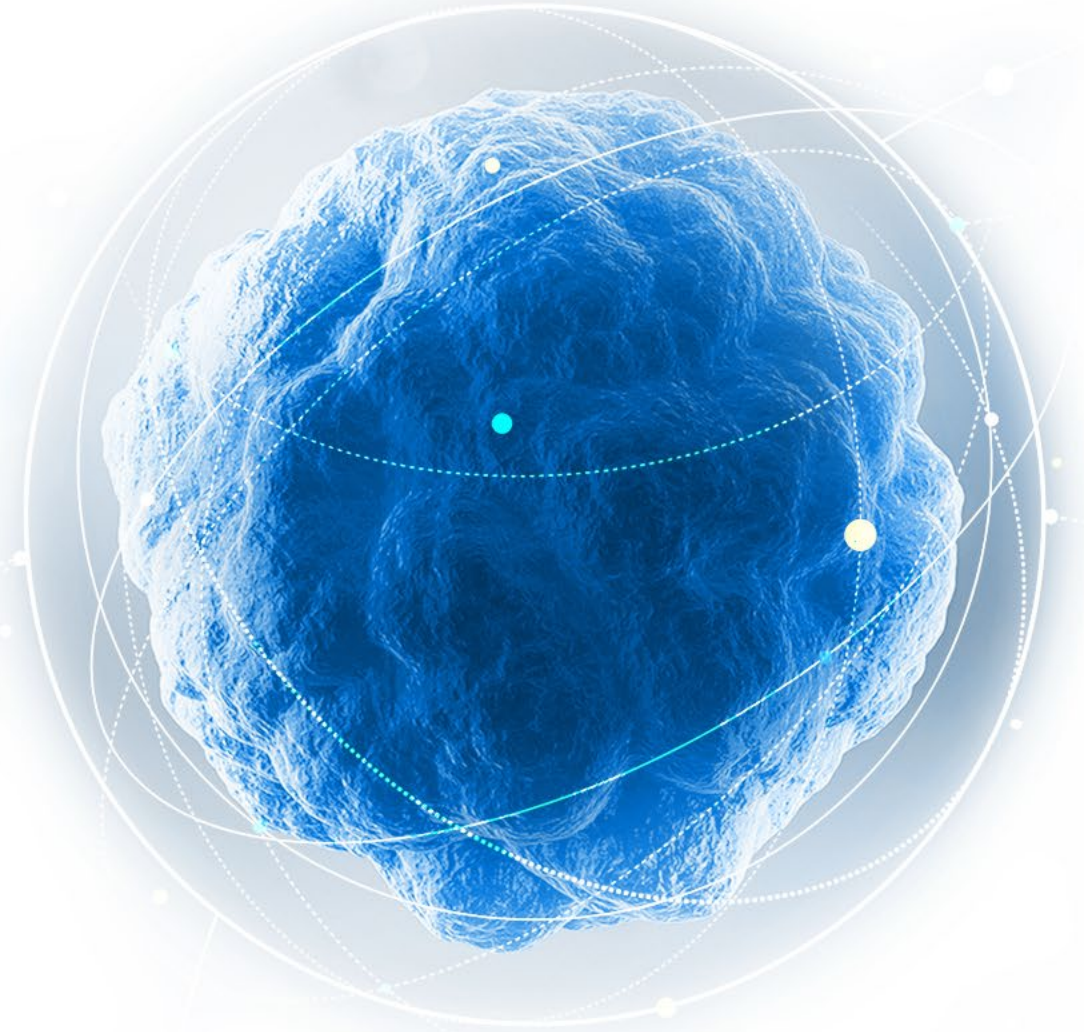
Abbreviations: NF-κB, Nuclear factor-κB; IR, CLL, Chronic lymphocytic leukemia; NHL  
 Sources: 1. Vermaat, J. S., et al. (2019). MYD88 mutations identify a molecular subgroup of diffuse large B-cell lymphoma with an unfavorable prognosis. *Haematologica*, 105(2), 424–434 ([Link](#)); 2. Zhou, Y., et al (2018). Analysis of genomic alteration in primary central nervous system lymphoma and the expression of some related genes. *Neoplasia*, 20(10), 1059–1069 ([Link](#)); 3. Alcoceba, M., et al (2022). MYD88 mutations: Transforming the landscape of IGM monoclonal gammopathies. *International Journal of Molecular Sciences*, 23(10), 5570. ([Link](#)); 4. Shekhar, R., et al. (2021). Frequency of MYD88 L265P mutation and its correlation with clinico-hematological profile in mature B-cell neoplasm. *Hematology/Oncology and Stem Cell Therapy*, 14(3), 231–239 ([Link](#)); 5. Insuasti-Beltran, G., et al. (2015). Significance of MYD88 L265P mutation status in the subclassification of Low-Grade B-Cell Lymphoma/Leukemia. *Archives of Pathology & Laboratory Medicine*, 139(8), 1035–1041 ([Link](#)); 6. Shuai, W., et al. (2020). Clinicopathological characterization of chronic lymphocytic leukemia with MYD88 mutations: L265P and non-L265P mutations are associated with different features. *Blood Cancer Journal*, 10(8) ([Link](#)); NHL incident rate of 18.6 per 100,000 (seer.cancer.gov) with DLBCL representing 25% of NHL per <https://www.ncbi.nlm.nih.gov/books/NBK557796/>. ABC represents 44% per letters to the editor, *haematologica*, 2011; Lv Ther Adv Hematol 2022; <https://rarediseases.org/rare-diseases/waldenstroms-macroglobulinemia/#affected>; <https://www.ncbi.nlm.nih.gov/books/NBK536985/>; Kalashnikov, *Blood Cancer Journal*, April 2023; <https://my.clevelandclinic.org/health/diseases/6210-chronic-lymphocytic-leukemia>

# Summary in NHL



- Emavusertib continues to demonstrate clear anti-cancer activity in R/R PCNSL
- Next steps:
  - Work with FDA to explore the potential for an accelerated approval path
  - Prioritize next NHL indications (after PCNSL) that could benefit from the dual-blockade of NF- $\kappa$ B (blocking the TLR pathway with emavusertib and blocking the BCR pathway with a BTKi)

# Emavusertib in AML



# Emavusertib binds to IRAK4 and FLT3, blocking both the TLR and FLT3 pathways

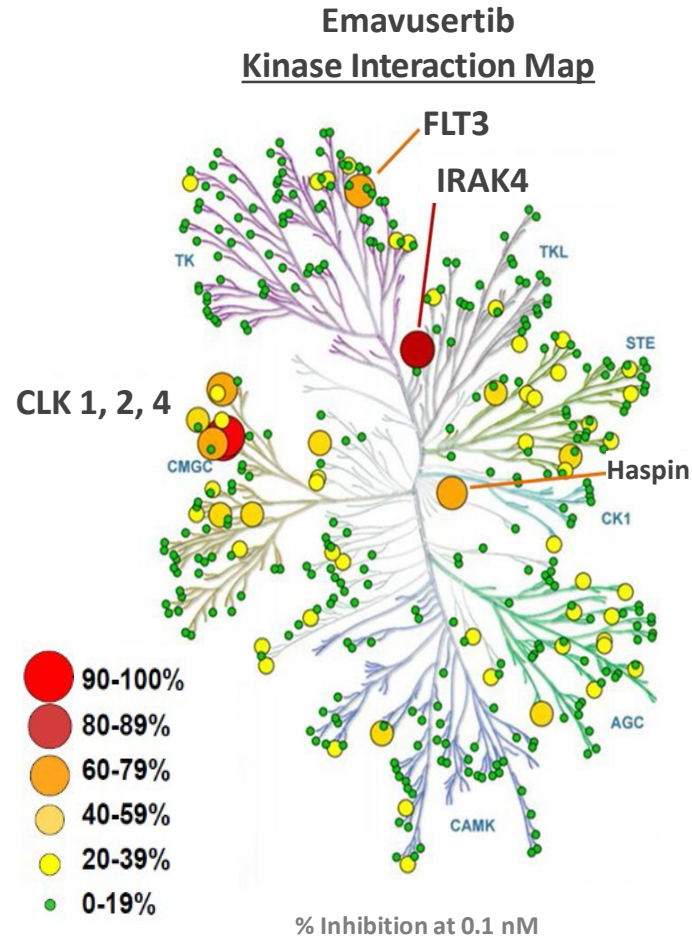


Illustration reproduced courtesy of Cell Signaling Technology

**Emavusertib Binding Affinity**

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

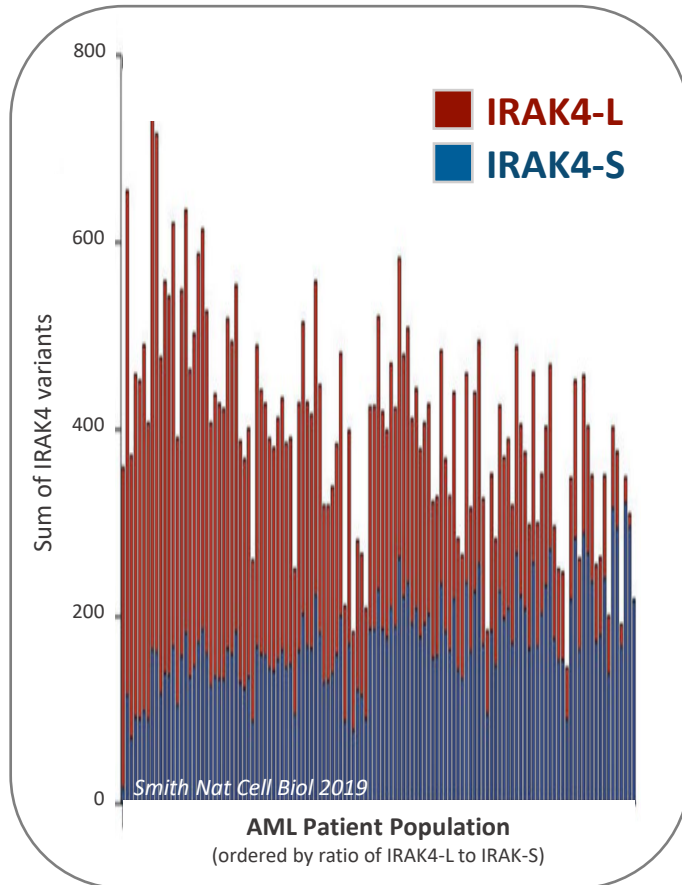
DiscoverX Kinase Panel (378 kinases screened)

**Binds tightly to IRAK4**

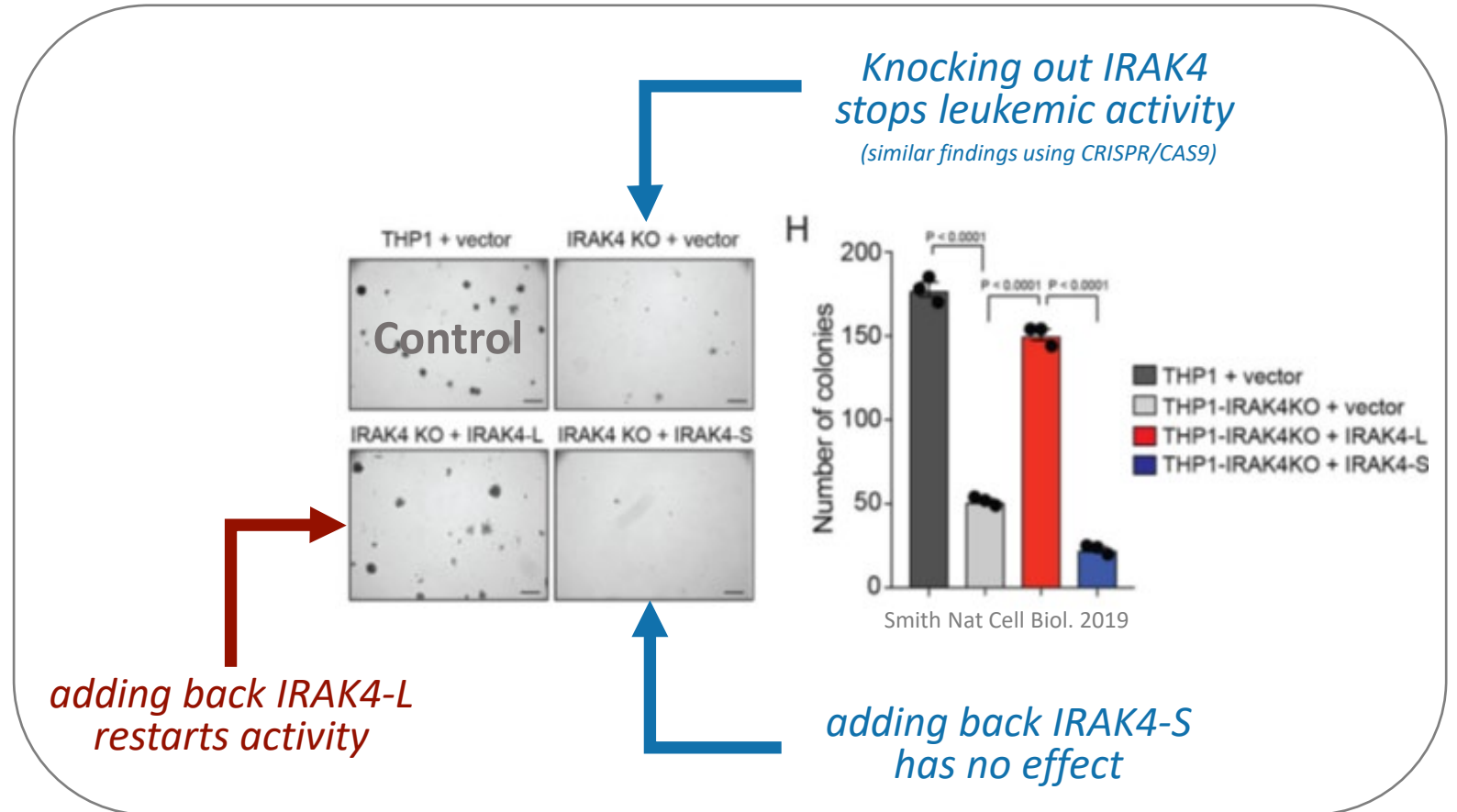
**Engineered to hit multiple targets of interest in oncology, including FLT3**

# IRAK4-L is an independent and powerful driver of disease in AML

**IRAK4-L is expressed in nearly all AML patients**



**IRAK4-L is oncogenic in AML**



# Strategy in AML

1

## Demonstrate safety

123 patients<sup>1</sup> treated in TakeAim AML 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 1<sup>st</sup> label in R/R patients

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

4

## Explore 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

# Safety Profile of Emavusertib as Monotherapy in AML<sup>1</sup>

- 123 patients treated with emavusertib in TakeAim Leukemia Study
- 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 <sup>2</sup>	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

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<sup>1</sup> treated in TakeAim AML 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

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

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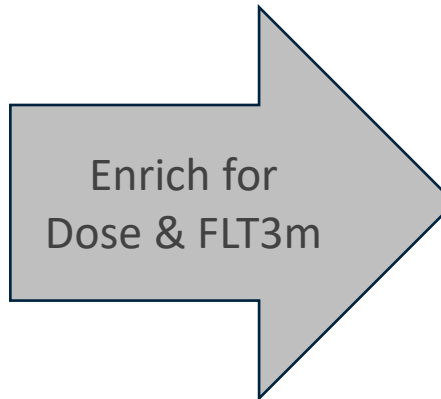
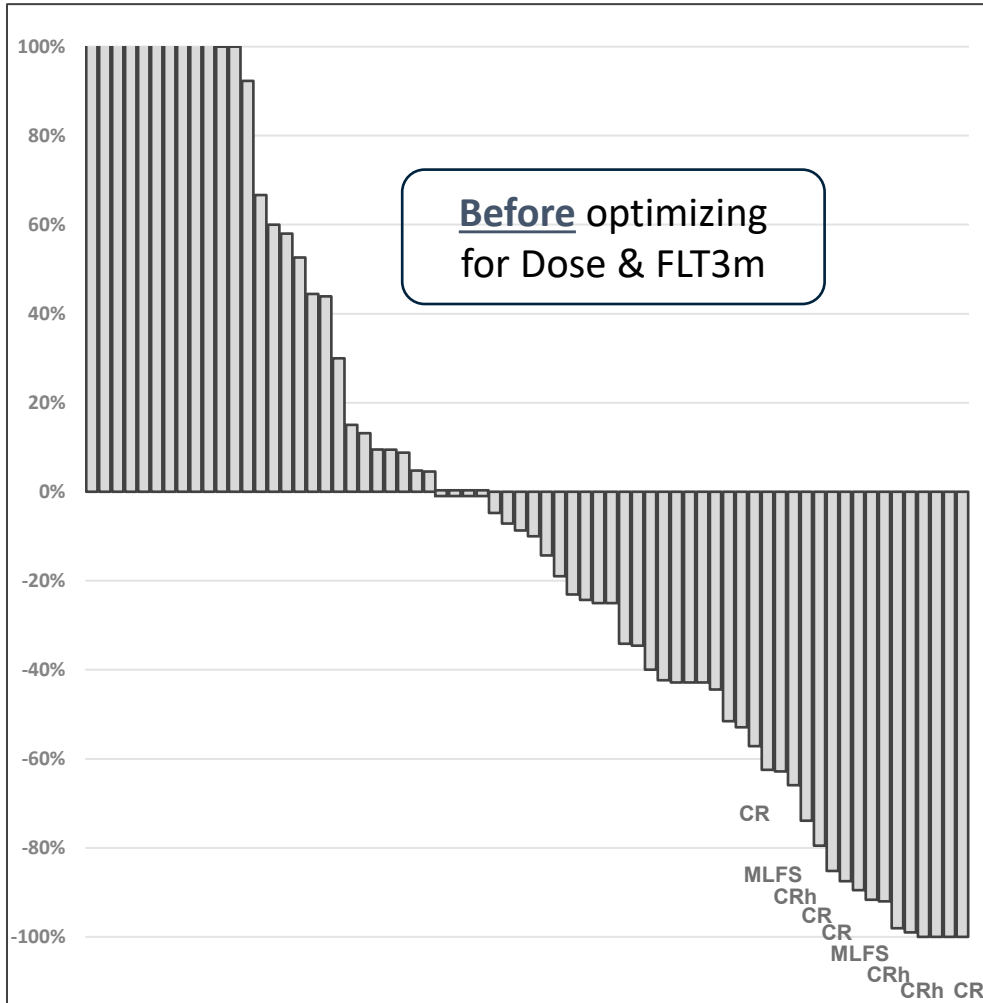
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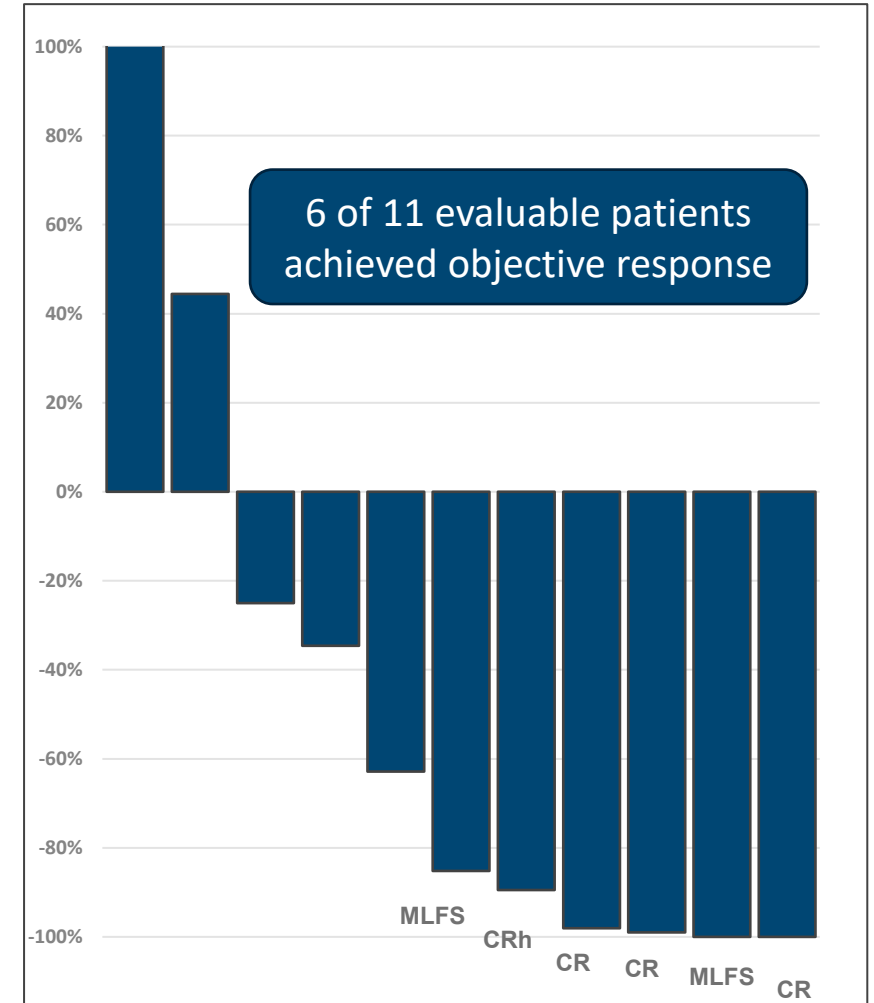


# Single-agent activity demonstrated in AML

All Patients, All Dose Levels



Patients treated at 300mg BID who also have FLT3 mutation



Data include all R/R AML patients determined to be evaluable for objective response using baseline and post-treatment marrow assessments as of Feb 26, 2024

# Strategy in AML

1

## Demonstrate safety

123 patients<sup>1</sup> treated in TakeAim AML 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

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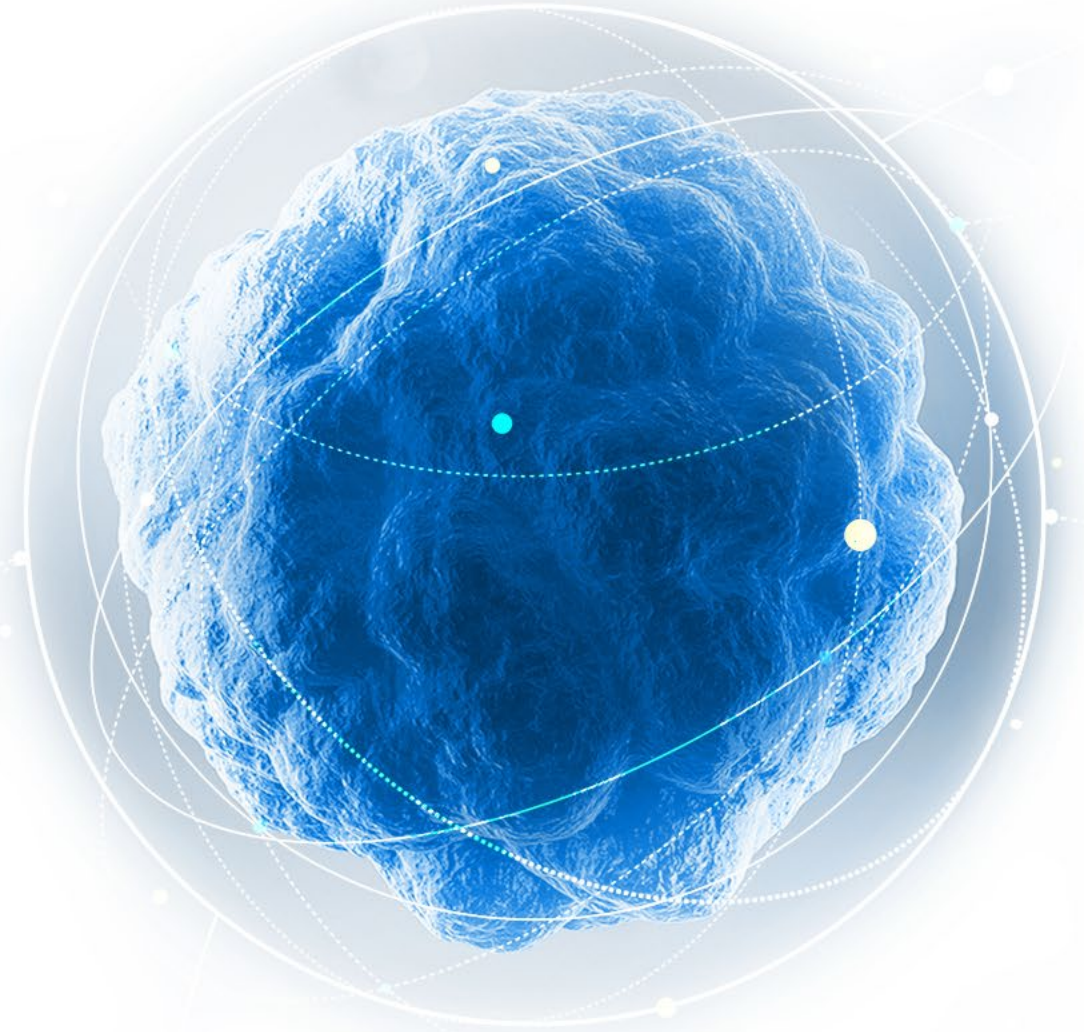
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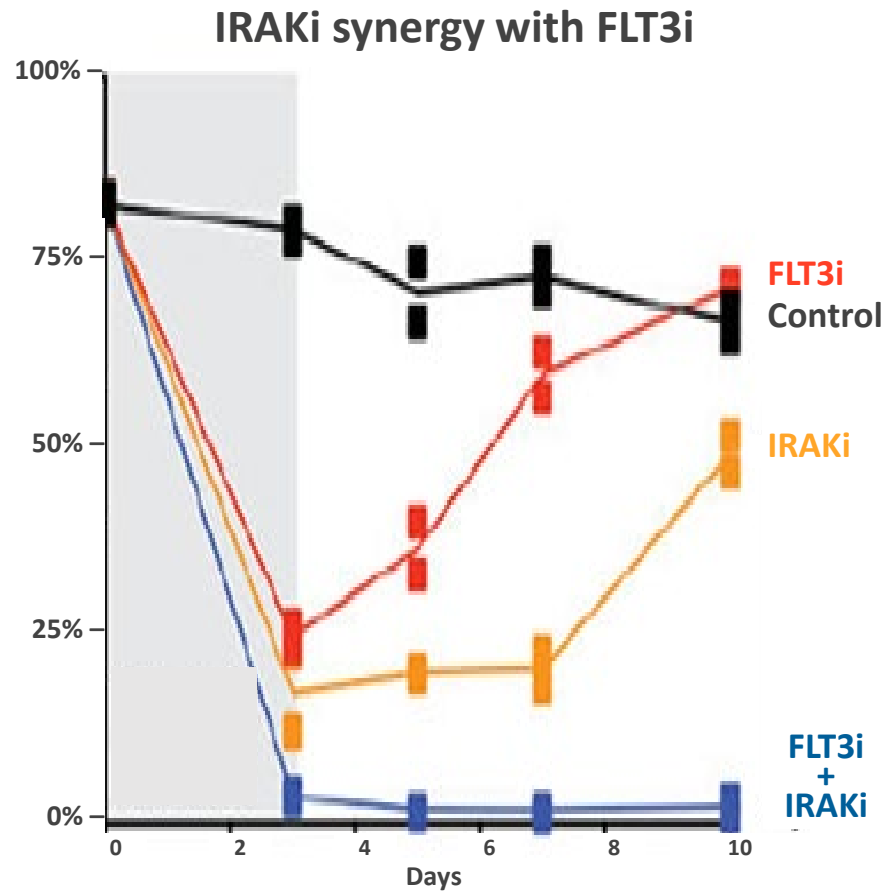
## Pursue partnership to maximize potential commercial opportunity

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

# Emavusertib in FLT3m AML



# Emavusertib's dual-targeting of IRAK4 and FLT3 enables monotherapy opportunity in FLT3m AML



**IRAK4 inhibition  
overcomes adaptive resistance  
to FLT3i**

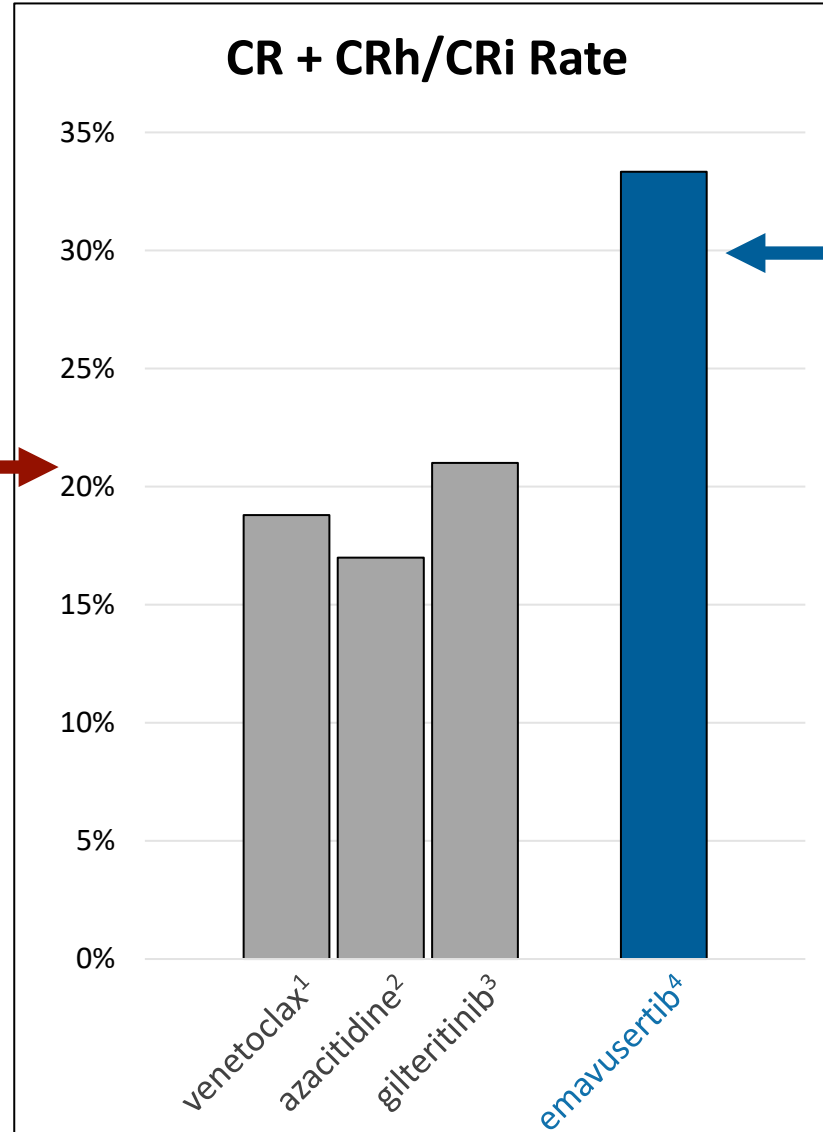
*Concomitant targeting of IRAK1 or IRAK4, alongside FLT3, is the most effective means to overcome the adaptive resistance incurred when targeting FLT3<sup>1</sup>*

Percent viable cells in preclinical AML cell lines (FLT3-ITD) treated for 72 hrs  
<sup>1</sup> Melgar Sci Transl Med 2019

# Emavusertib is a potential best-in-class therapy in FLT3m AML

Benchmark in FLT3i-naïve patients is **21% CR/CRh rate**

*87% of patients in the benchmark study were FLT3i naïve<sup>3</sup>*



Salvage Line Patients treated with emavusertib achieved **> 30% CR/CRh rate**

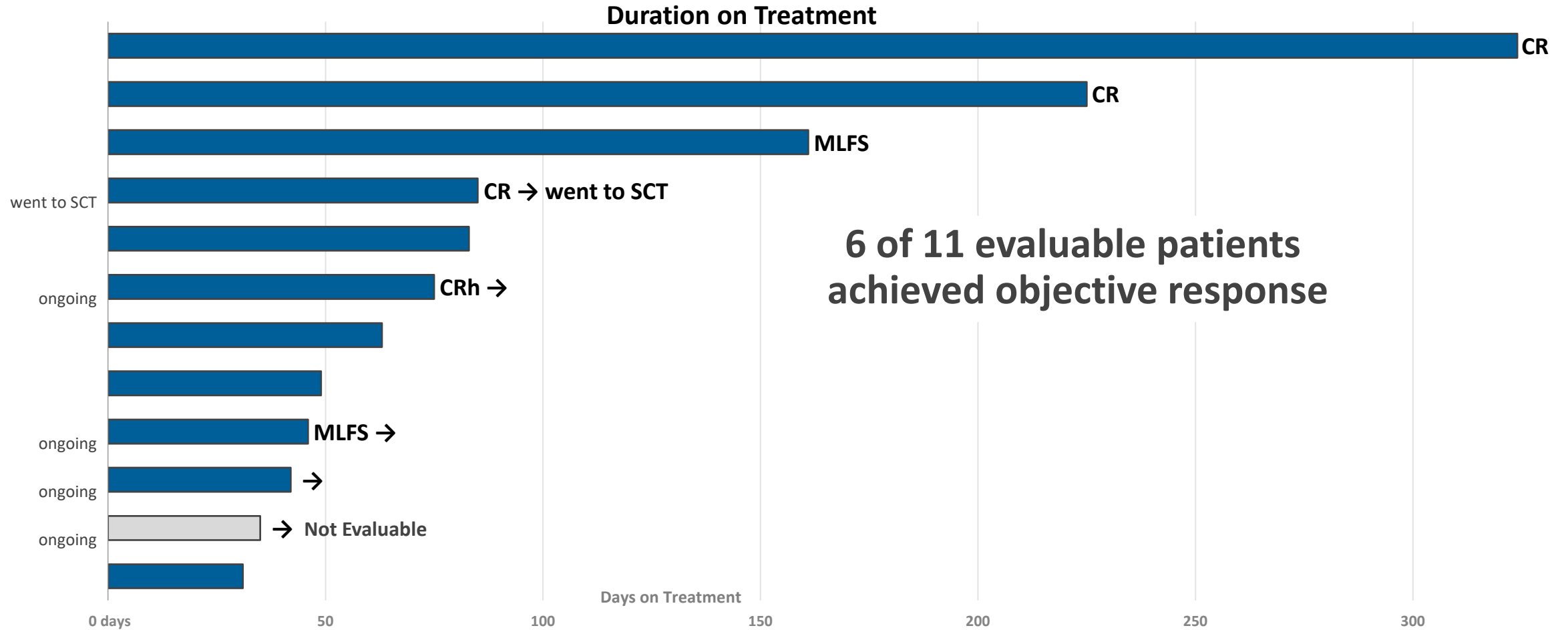
~1.5X greater than the benchmark for FLT3i-naïve patients

*9 of 12 patients treated with emavusertib were FLT3i experienced (they had progressed on 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]

# Encouraging updated data in FLT3m AML

presented at ASCO/EHA 2024



*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

1

## Demonstrate safety

123 patients<sup>1</sup> treated in TakeAim AML 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 1<sup>st</sup> label in R/R patients

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

4

## Explore frontline opportunity with combination

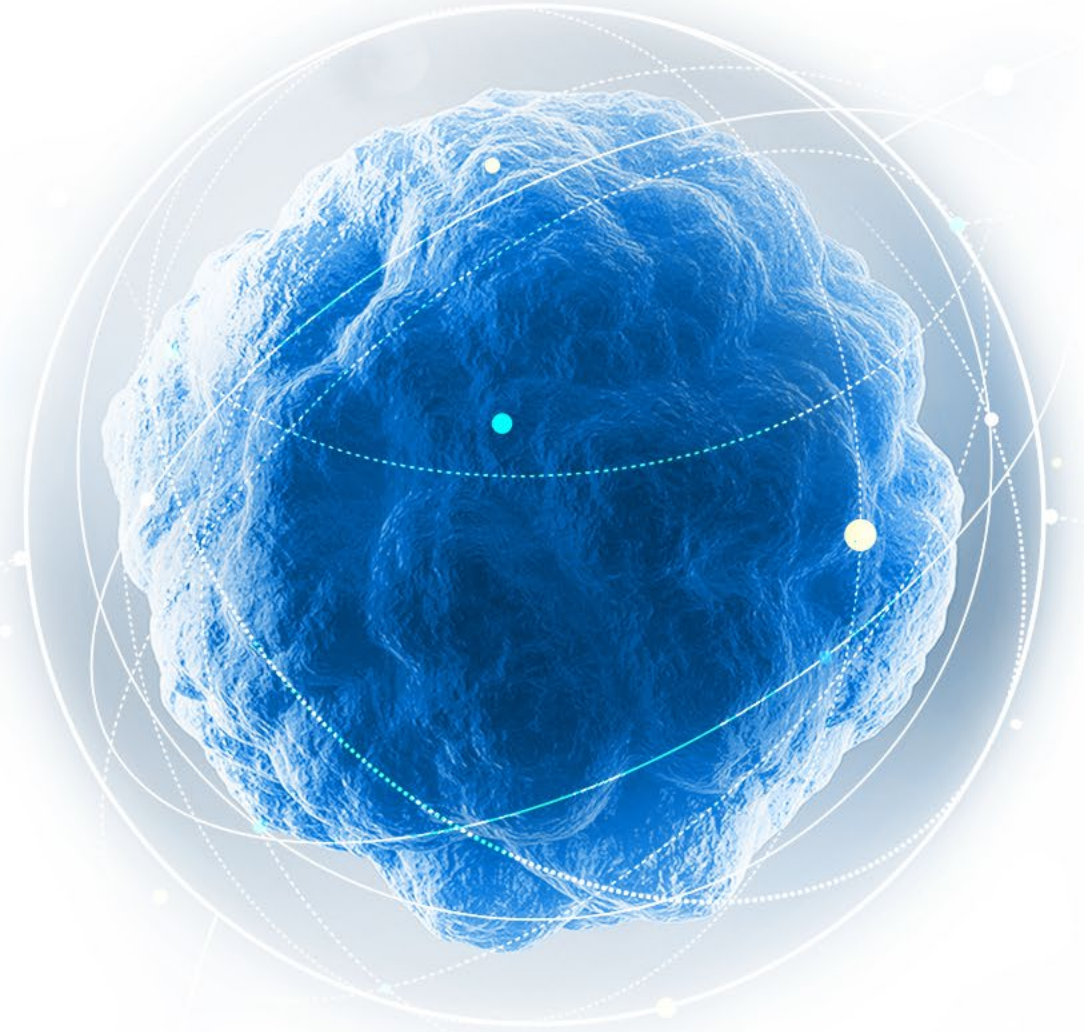
IRAK4-L is expressed in nearly all AML patients; preclinical "all comer" models suggest emavusertib is synergistic with azacitidine and venetoclax

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

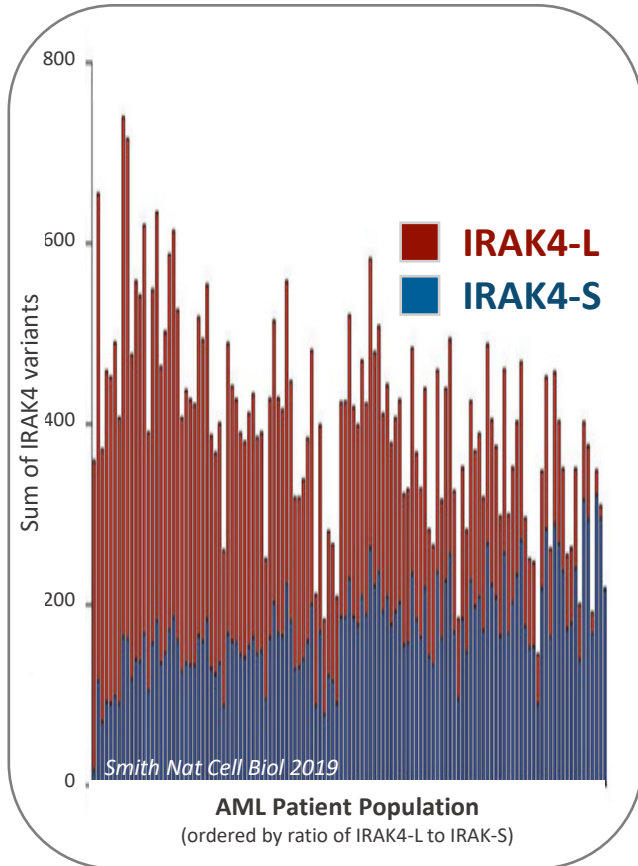
# Emavusertib in All Comers



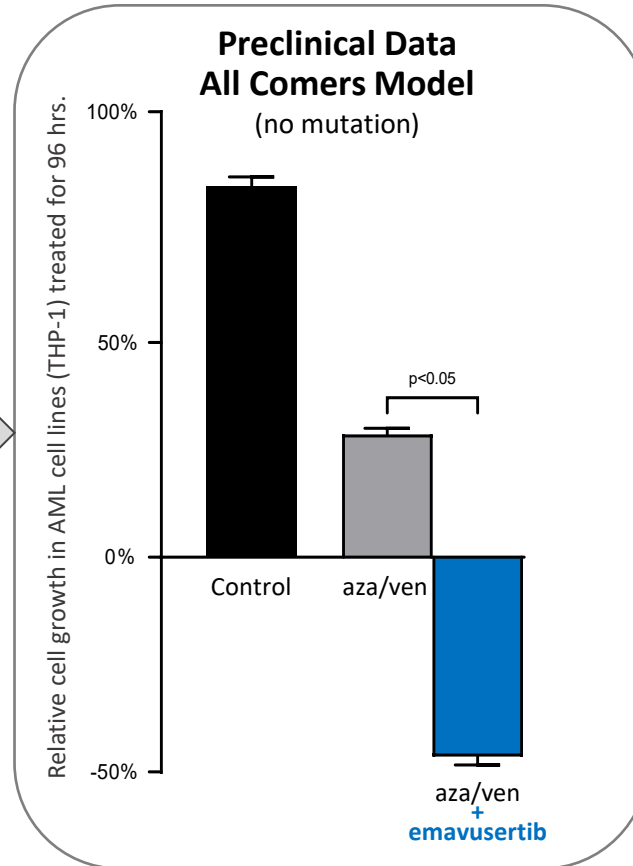


# Emavusertib combination with aza/ven targets all comers in frontline AML

oncogenic IRAK4-L is expressed in nearly all AML patients



emavusertib synergy with aza/ven in preclinical studies



ema/aza/ven triplet combination

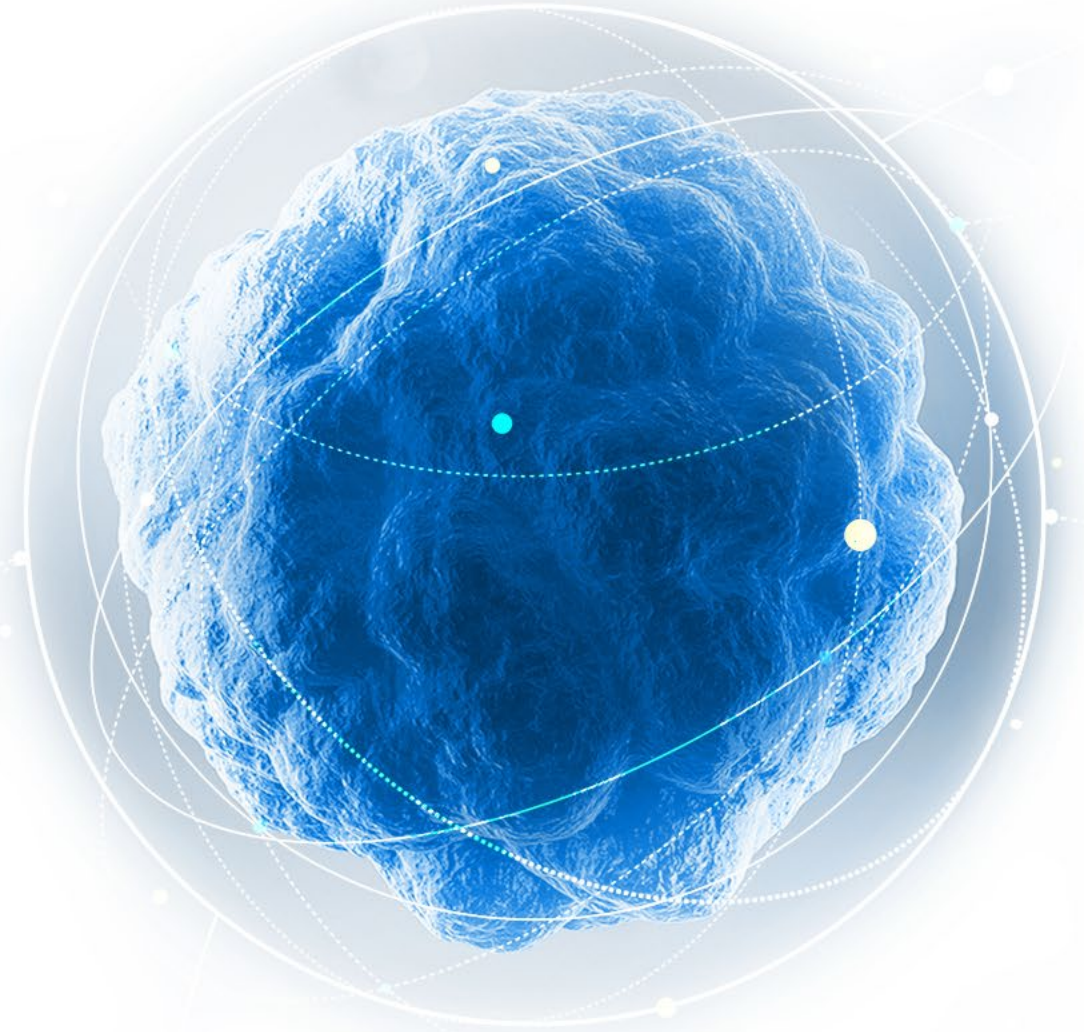
*Ph 1 study initiated 1H2024*  
*initial safety data expected Q1 2025*

# Summary in AML



- Emavusertib targets both FLT3 and IRAK4
- Emavusertib offers potential for best-in-class therapeutic in FLT3m AML (a genetically-defined population)
- Oncogenic IRAK4 is expressed in nearly all AML patients and is not addressed by current standard-of-care (azacitidine and venetoclax)
- Emavusertib, in combination with azacitidine and venetoclax, offers potential for broad commercial opportunity in frontline AML

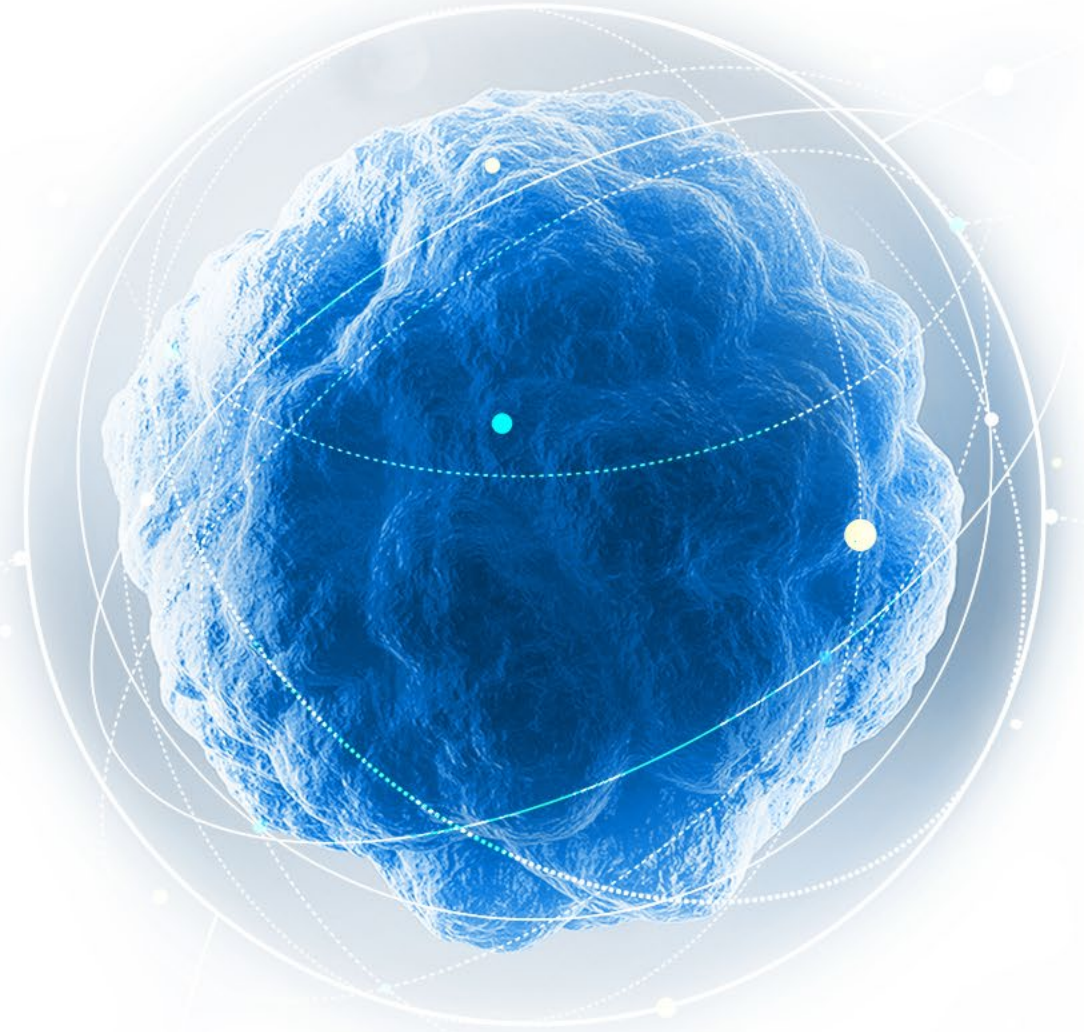
# Solid Tumors



# Ongoing studies (ISTs) of emavusertib in Solid Tumors

Tumor Type	Institution (Investigator)	Emavusertib Combination Partner
<b>Pancreatic</b>	CRADA Washington University (Grierson) Washington University (Lim)	gemcitabine, ( <i>nab</i> )-paclitaxel
<b>Colorectal</b>	CRADA Oklahoma University (Ulahannan) Washington University (Lim)	FOLFOX + bevacizumab
<b>Gastro/Esophageal</b>	Washington University (Grierson)	FOLFOX/PD1 +/- trastuzumab
<b>Melanoma</b>	University of Florida (Doonan)	pembrolizumab
<b>Urothelial</b>	CRADA Mount Sinai (Galsky)	pembrolizumab

# Other Information



# Financials and IP

**As of June 30, 2024**

\$28.4M Cash and investments<sup>1</sup>  
 ~5.9M Shares Outstanding  
 ~7.0M Shares Fully Diluted

2035 Composition of Matter IP on emavusertib  
 (before extension)

***We believe cash is sufficient to achieve anticipated near-term milestones***

- *Updated PCNSL data 15-20 patients (4Q24/1Q25)*
- *AML triplet safety data (4Q24/1Q25)*

<sup>1</sup> estimated cash runway into 2025