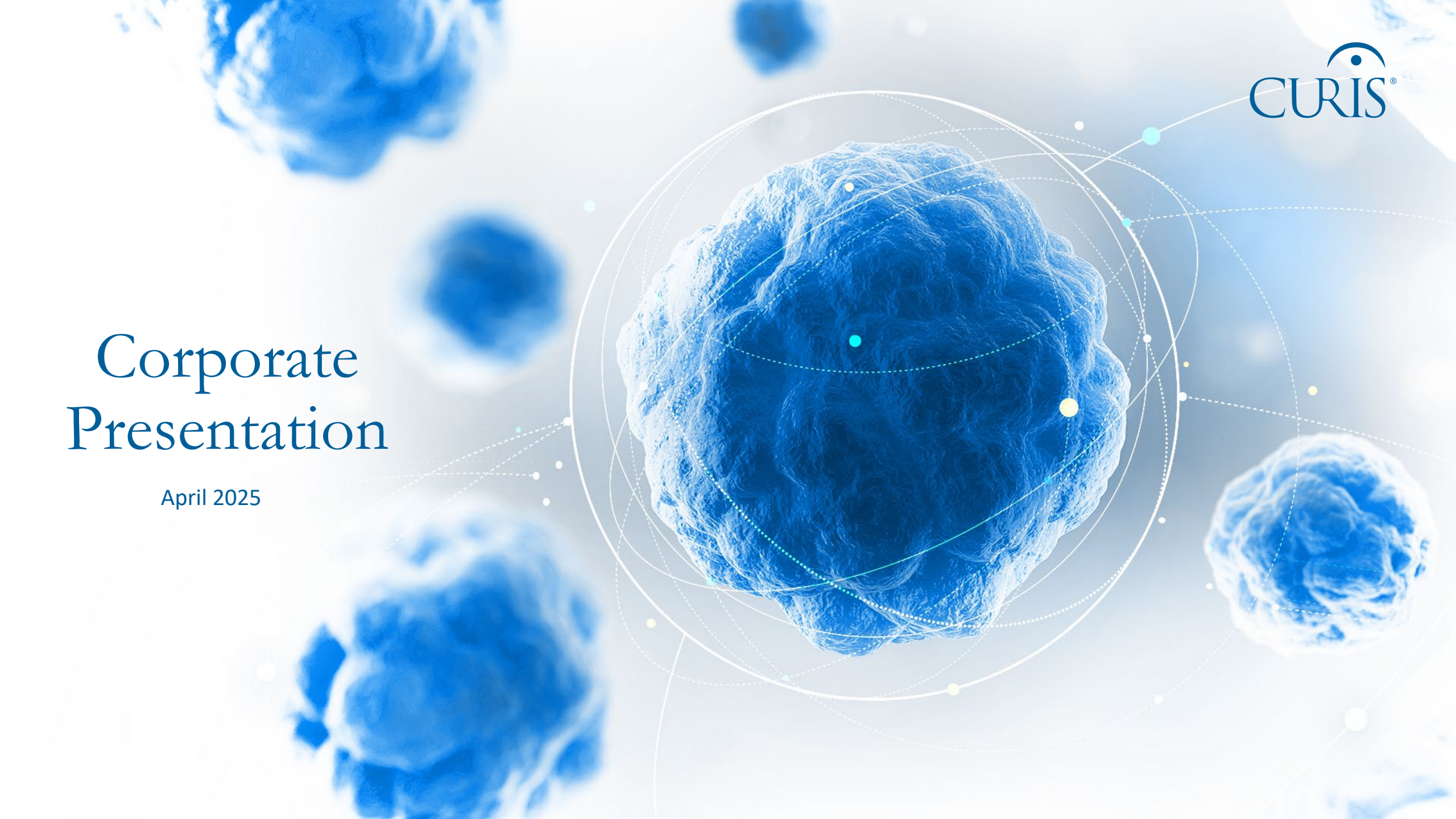


Corporate Presentation

April 2025

Cautionary note regarding forward looking statements and disclaimers

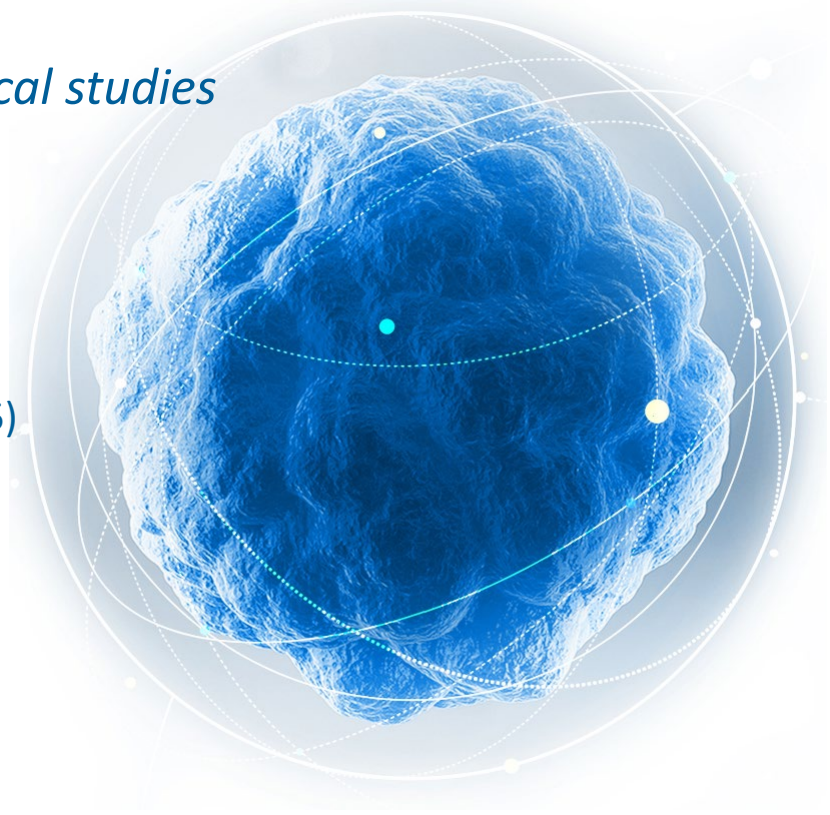
This presentation contains certain forward-looking statements about Curis, Inc. (“we,” “us,” or the “Company”) within the meaning of the Private Securities Litigation Reform Act of 1995, as amended. Words such as “expect(s),” “believe(s),” “will,” “may,” “anticipate(s),” “focus(es),” “plans,” “mission,” “strategy,” “potential,” “estimate(s)”, “opportunity,” "intend," "project," "seek," "should," "would," likelihood,” 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 clinical milestones; ongoing and future clinical trials and the results of these trials; expectations with respect to regulatory objectives; the clinical and therapeutic potential of emavusertib; our cash runway; the focus on emavusertib and management’s ability to successfully achieve its strategies and 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: regulatory action by the U.S. Food and Drug Administration ("FDA") or any equivalent foreign regulatory agency with regard to our trials; whether emavusertib will advance further in the clinical development process and whether and when, if at all, it will receive approval from the FDA or equivalent foreign regulatory agencies; whether historical preclinical and clinical trial results will be predictive of future clinical trial results; whether historical clinical trial results will be predictive of future trial results; whether emavusertib development efforts will be successful; whether emavusertib will be successfully marketed if approved; our ability to achieve the benefits contemplated by our collaboration agreements; management’s ability to successfully achieve its strategies and goals; the sufficiency of our cash resources; our ability to raise necessary 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, June 30, 2024 and September 30, 2024 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 update 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

Potential first-in-class inhibitor of IRAK4

- *Being evaluated in Phase 1/2 clinical studies in NHL, AML, and Solid Tumors*
- *Anticipated 2025 milestones:*
  - Data from 30-35 PCNSL patients (Q4 '25)
- *Expected cash runway, mid-2025*



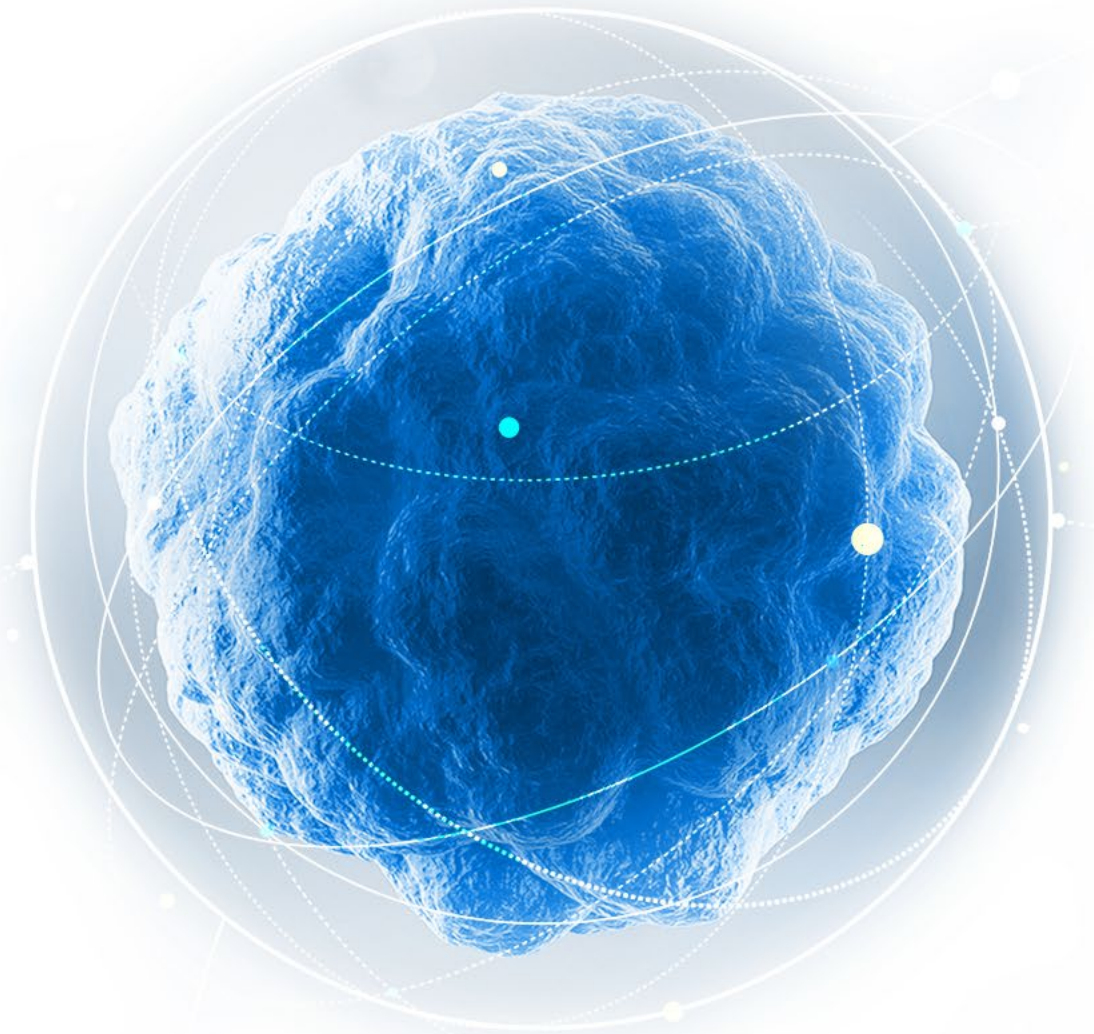
Well tolerated in monotherapy & combination

Demonstrated synergy with BTKi, HMA, BCL2i

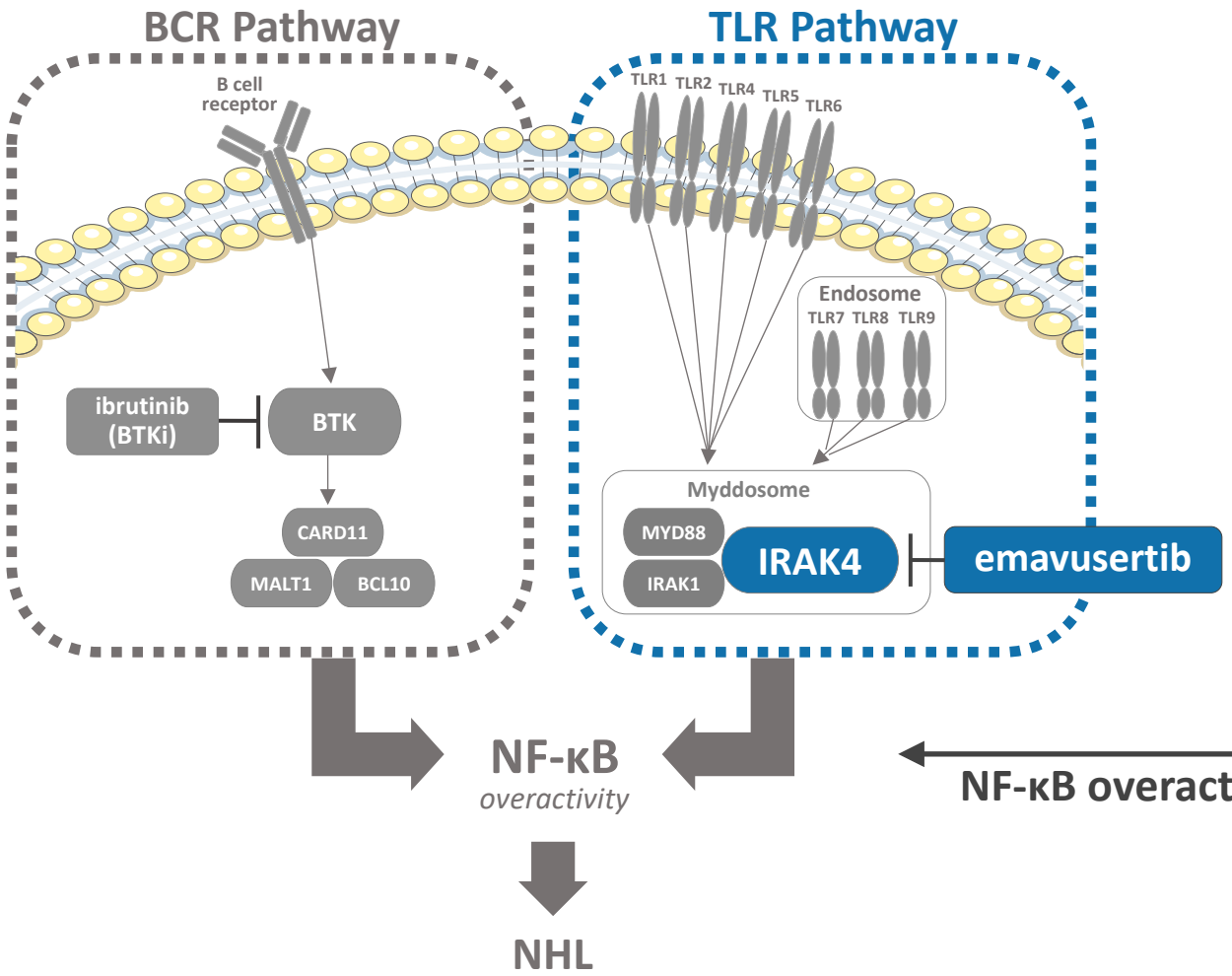
Encouraging clinical data in NHL and AML

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

Emavusertib
in NHL



Emavusertib Mechanism in NHL

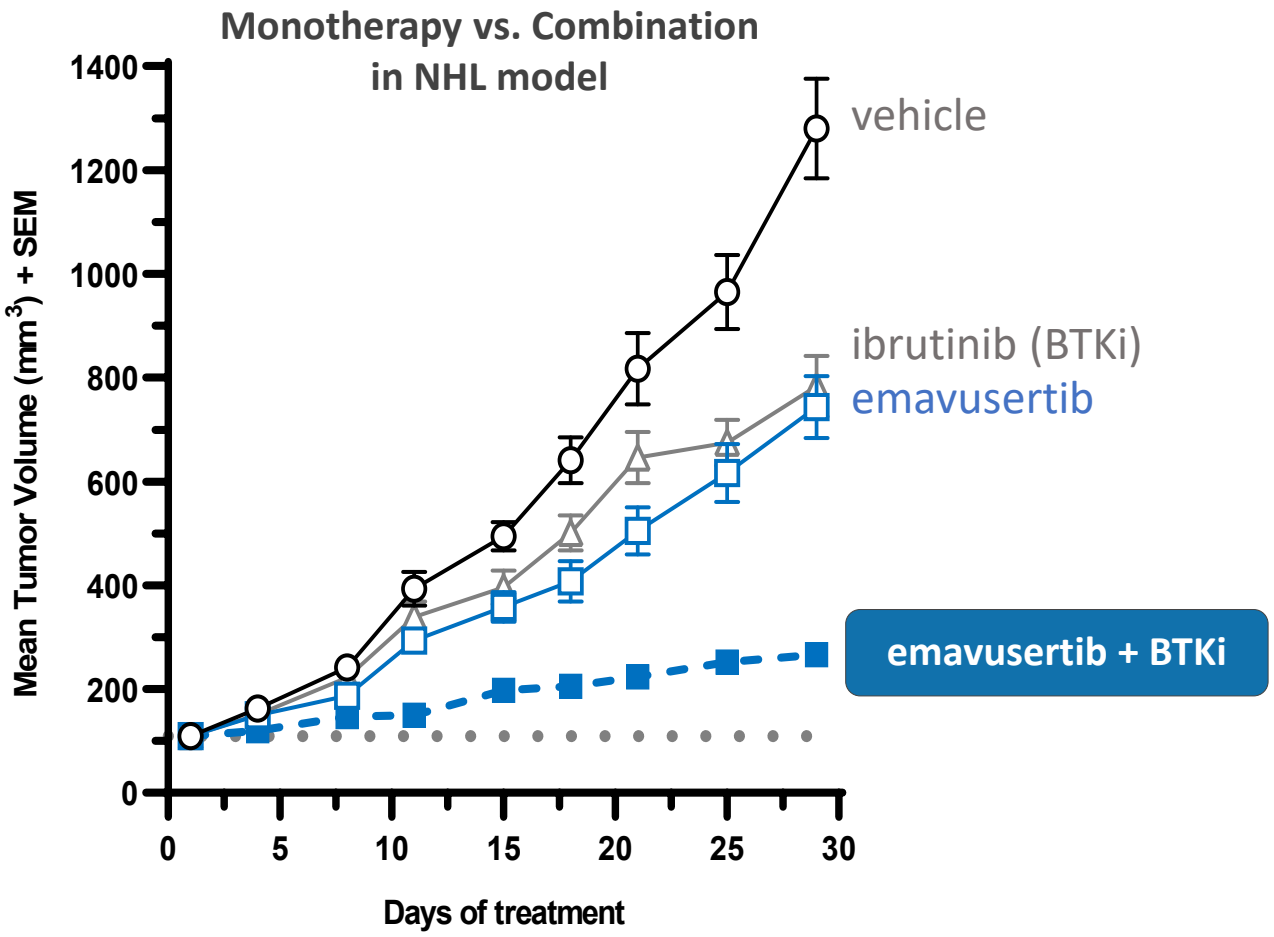


Dual blockade of the TLR and BCR pathways maximizes downregulation of NF-κB

NF-κB overactivity drives disease in NHL

TakeAim Lymphoma Clinical Outcomes, ASH 2023 Poster
Abbreviations: B-Cell Receptor (BCR) and Toll-Like Receptor (TLR)

Mechanism Demonstrated in Preclinical Models



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

Dual blockade
of the TLR and BCR pathways
achieved deeper tumor reduction

Strategy in NHL

1

Demonstrate safety

39 patients[1] treated in TakeAim Lymphoma study, emavusertib was well tolerated with 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 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 clinical studies across multiple NHL subtypes and prepare for potential commercial launch

1 – As of Jan 2, 2025

Safety profile in NHL

- 39 patients treated in NHL
- Shown to be well tolerated
- No dose-limiting myelosuppression has been observed
- Emavusertib crosses the BBB
- No dose-limiting CNS toxicities have been observed

Grade 3+ Treatment-Related Adverse Event Reported in > 1 Patients, n (%)	100 mg BID ema +ibr (n=10)	200 mg BID ema +ibr (n=22)	300 mg BID ema +ibr (n=7)	Total (n=39)
# patients having grade 3+ TRAEs	5 (50)	10 (45)	6 (86)	21 (54)
Neutropenia	4 (40)	1 (4.5)	0	5 (13)
Lipase increased	2 (20)	1 (4.5)	0	3 (8)
Platelet count decreased	0	2 (9)	1 (14)	3 (8)
Alanine aminotransferase increased	0	1 (4.5)	1 (14)	2 (5)
Amylase increased	2 (20)	0	0	2 (5)
Aspartate aminotransferase increased	0	1 (4.5)	1 (14)	2 (5)
Fatigue	0	1 (4.5)	1 (14)	2 (5)
Hyponatraemia	0	2 (9)	0	2 (5)
Leukopenia	2 (20)	0	0	2 (5)
Syncope	0	1 (4.5)	1 (14)	2 (5)

Safety data as of January 02, 2025
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

1

Demonstrate safety

39 patients[1] treated in TakeAim Lymphoma study, emavusertib was well tolerated with no overlapping dose-limiting toxicity with ibrutinib

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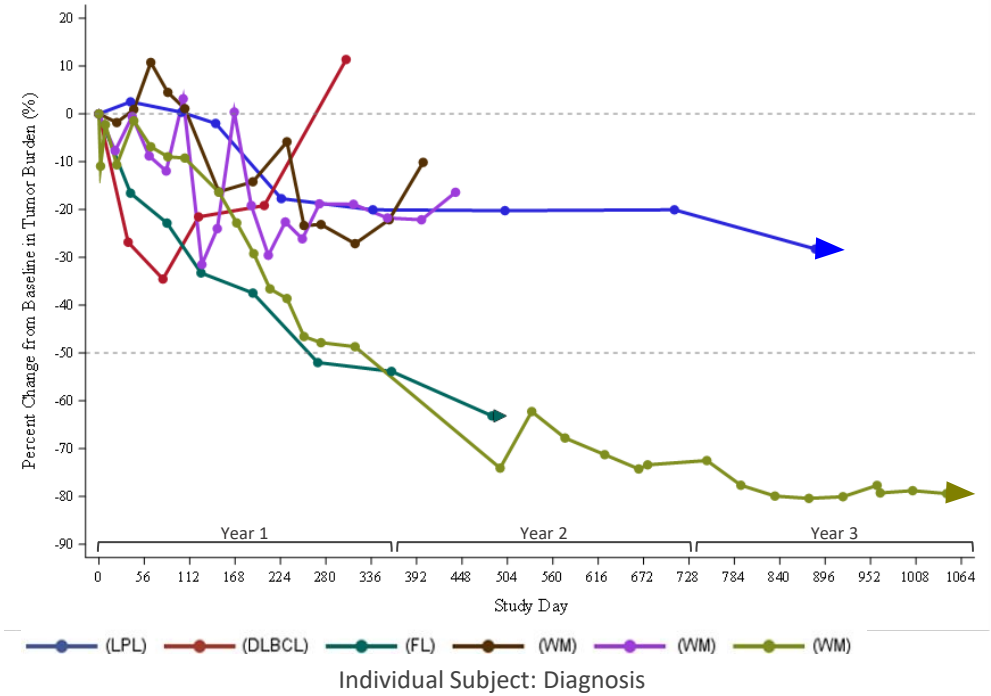
1 – As of Jan 2, 2025

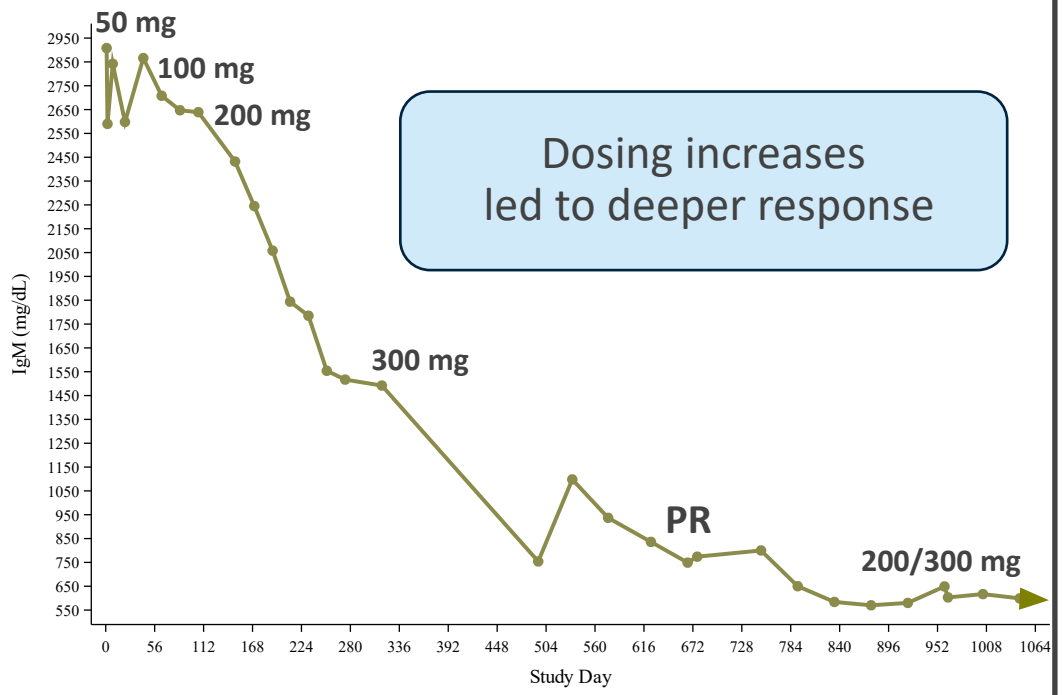
Single-agent activity demonstrated in NHL

Tumor Reduction Sustained up to 3+ Years



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.

Case Study in Dose Response (WM patient)



2022 IWWM Conference Presentation

Abbreviations: Lymphoplasmacytic Lymphoma (LPL), Follicular Lymphoma (FL), Partial Response (PR)

Strategy in NHL

1

Demonstrate safety

39 patients[1] treated in TakeAim Lymphoma study, emavusertib was well tolerated with no overlapping dose-limiting toxicity with ibrutinib

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Pursue partnership to expand across NHL

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

1 – As of Jan 2, 2025

BTKi used in six NHL subtypes

PCNSL selected as 1st NHL indication for emavusertib

NHL Subtype	Incidence in U.S.	Key Targets of Interest	Therapies Used
ABC-DLBCL	2 per 100,000	IRAK4, MYD88, CD79, NF-kB	BTKi, R-CHOP
PCNSL	0.5 per 100,000	IRAK4, MYD88, CD79, NF-kB	BTKi, Chemo, MTX, RT
WM	0.5 per 100,000	IRAK4, MYD88, CD79, NF-kB	BTKi, Chemo
MCL	0.5 per 100,000	BCR and TLR pathways	BTKi, Chemo, αCD20
MZL	1.5 per 100,000	IRAK4, MYD88, CARD11, NF-kB	BTKi, Chemo, αCD20, RT
CLL/SLL	4.5 per 100,000	NF-kB	BTKi, αCD20

Published Studies Support Potential in Multiple NHL Subtypes

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

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

Abbreviations: NF-kB, Nuclear factor-κB, proteasome inhibitors (PI)

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 L256P 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);

Clinical Study Design

Part A in multiple NHL subtypes – Parts B & C in the PCNSL subtype

COMPLETED
Part A

Multiple NHL Subtypes
dose escalation

- emavusertib + ibrutinib
- emavusertib

Parts B & C are currently enrolling

Part B

PCNSL
in BTKi-experienced patients

- emavusertib + ibrutinib

single-arm design intended to support Accelerated Approval

Part C

PCNSL
in BTKi-naïve patients

- emavusertib + ibrutinib
- emavusertib
- ibrutinib

randomized design intended to support Confirmatory Study

Note: Part C is a randomized study comparing the emavusertib + ibrutinib combination versus ibrutinib monotherapy to support full approval; it also includes an arm of emavusertib monotherapy as required for NDA submission; patients who progress on a monotherapy therapy arm are eligible to crossover to the combination therapy arm.

Clinical Data

*Anti-cancer activity observed in **BTKi-naïve** and **BTK-experienced** patients*

In BTKi-naïve patients:

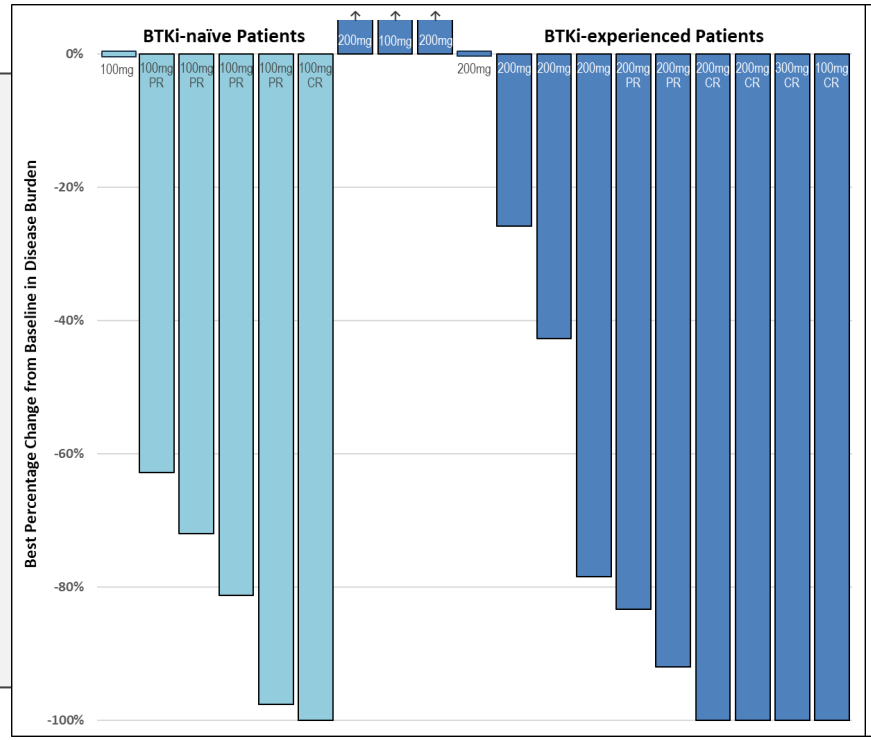
Adding emavusertib to ibrutinib **achieved higher ORR** than published data for ibrutinib alone

- *71% for ema + ibr (5 of 7 patients ITT)*
- *39% for ibr monotherapy[1] (15 of 38 patients ITT)*

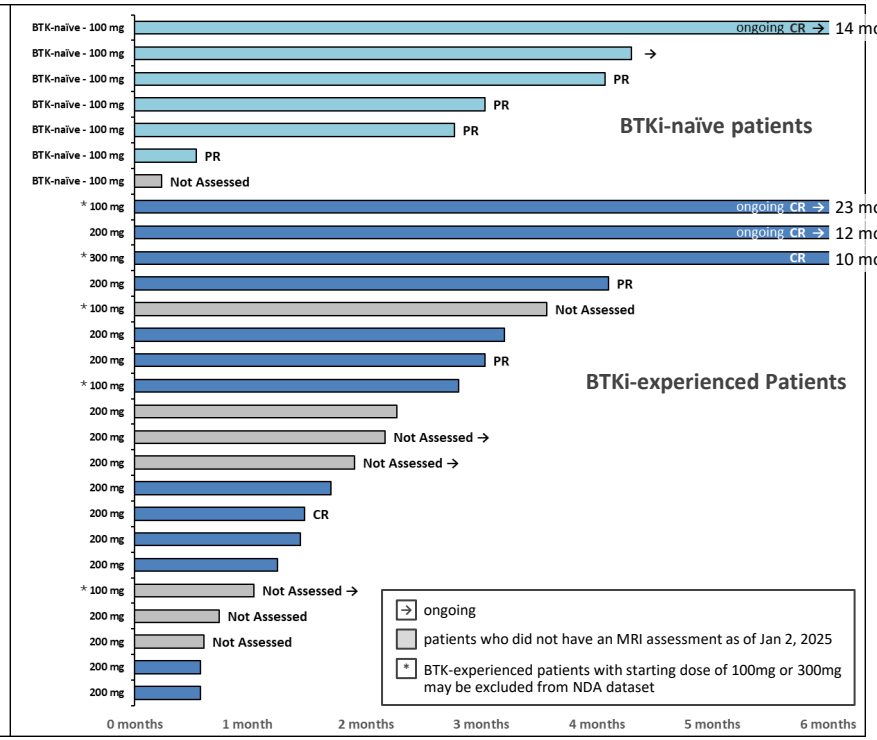
In BTKi-experienced patients:

Adding emavusertib **reversed tumor growth**, with reductions in tumor size in 9 patients, incl 6 responses

Reduction in Tumor Burden



Duration of Treatment



Data include all patients who had an MRI assessment as of Jan 2, 2025

1 additional BTKi-naïve patient and 7 BTKi-experienced patients had not received an MRI assessment as of Jan 2, 2025

Data include all patients (ITT) as of Jan 2, 2025

1 – Soussain, Eur J Cancer 2019
Abbreviation: Intent to Treat (ITT)

PCNSL Case Study

Patient with R/R PCNSL treated with emavusertib + ibrutinib

Male patient, 53 yrs

Diagnosis: PCNSL diagnosed on 30 Jun 2020

Baseline: Depression, elevated LFTs, loss of appetite, cerebral edema, mixed IBS, hiatal hernia, GERD, essential hypertension, and obstructive sleep apnea

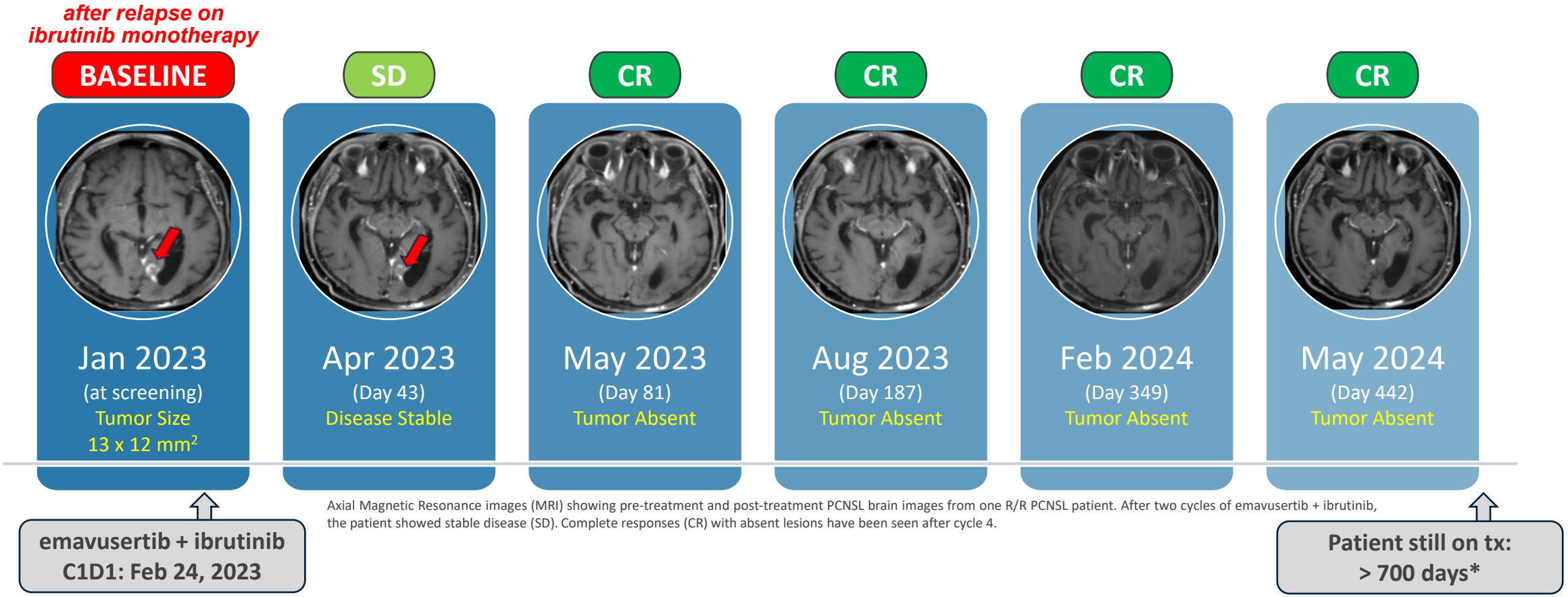
Prior Tx: Line 1: MTX, high-dose BCNU, Ara-C, thiotepa, WBRT, rituximab, and ASCT (PR)
Line 2: ibrutinib (CR)

Relapse: Disease progressed on treatment with ibrutinib on 29 Nov 2022, primary lesion measured 13 x 12 mm

Abbreviations: Liver Function Test (LFT), Irritable Bowel Syndrome (IBS), Gastroesophageal Reflux Disease (GERD), Methotrexate Sodium (MTX), Carmustine (BCNU), Cytosine Arabinoside (Ara-C), Whole Brain Radiation Therapy (WBRT) and Autologous Stem Cell Transplant (ASCT)

PCNSL Case Study

Patient with R/R PCNSL who achieved CR on emavusertib + ibrutinib



Axial Magnetic Resonance images (MRI) showing pre-treatment and post-treatment PCNSL brain images from one R/R PCNSL patient. After two cycles of emavusertib + ibrutinib, the patient showed stable disease (SD). Complete responses (CR) with absent lesions have been seen after cycle 4.

Consistent with previous findings, these data support the hypothesis that emavusertib can re-sensitize patients to BTKi therapy, and demonstrates its potential to significantly advance R/R PCNSL treatment

Abbreviations: Cycle 1, Day 1 (C1D1), Treatment (tx)

* *as of Jan 2, 2025*

Strategy in NHL

1

Demonstrate safety

39 patients[1] treated in TakeAim Lymphoma study, emavusertib was well tolerated with 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

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

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Pursue partnership to expand across NHL

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

1 – As of Jan 2, 2025

Expand across NHL, wherever BTKi monotherapy is used

NHL Subtype	Incidence in U.S.	Key Targets of Interest	Therapies Used
ABC-DLBCL	2 per 100,000	IRAK4, MYD88, CD79, NF-kB	**BTKi**, R-CHOP
PCNSL	0.5 per 100,000	IRAK4, MYD88, CD79, NF-kB	**BTKi**, Chemo, MTX, RT
WM	0.5 per 100,000	IRAK4, MYD88, CD79, NF-kB	**BTKi**, Chemo
MCL	0.5 per 100,000	BCR and TLR pathways	**BTKi**, Chemo, αCD20
MZL	1.5 per 100,000	IRAK4, MYD88, CARD11, NF-kB	**BTKi**, Chemo, αCD20, RT
CLL/SLL	4.5 per 100,000	NF-kB	**BTKi**, αCD20

Published Studies Support Potential in Multiple NHL Subtypes

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

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

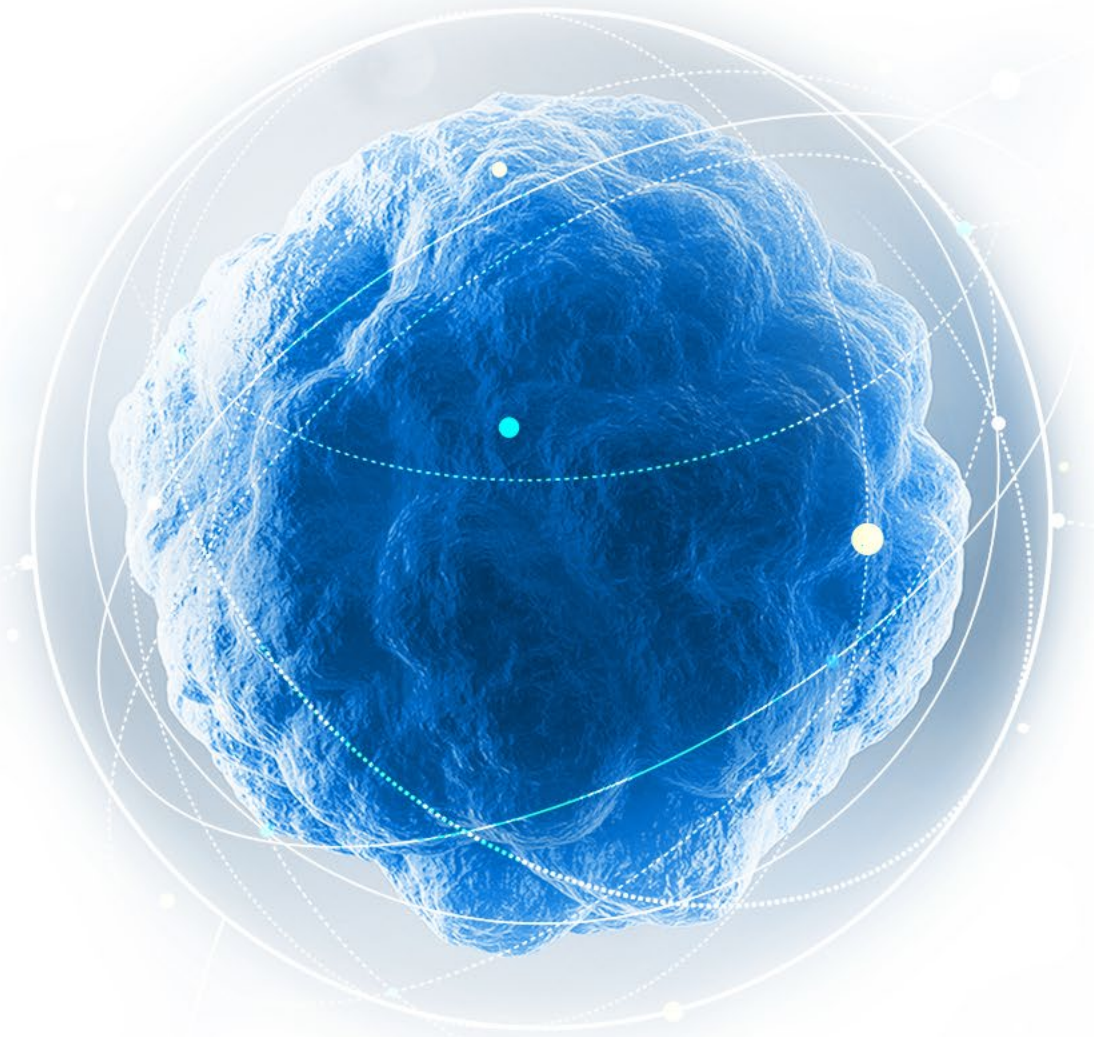
Abbreviations: NF-kB, Nuclear factor-κB, proteasome inhibitors (PI)

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 L256P 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);

Summary in NHL

- Demonstrated anti-cancer activity in PCNSL
- Orphan Drug designation received from both FDA and EMA in PCNSL
- Next steps:
  - Continue enrollment in PCNSL toward potential Conditional Approval and Accelerated Approval
  - Work with EMA and FDA on confirmatory trial design in PCNSL
  - Prioritize the next NHL indications for expansion

Emavusertib
in AML



CURIS®

Emavusertib Hits Multiple Targets of Interest in AML

IRAK4 and FLT3m are important drivers of disease

Emavusertib
Kinase Interaction Map

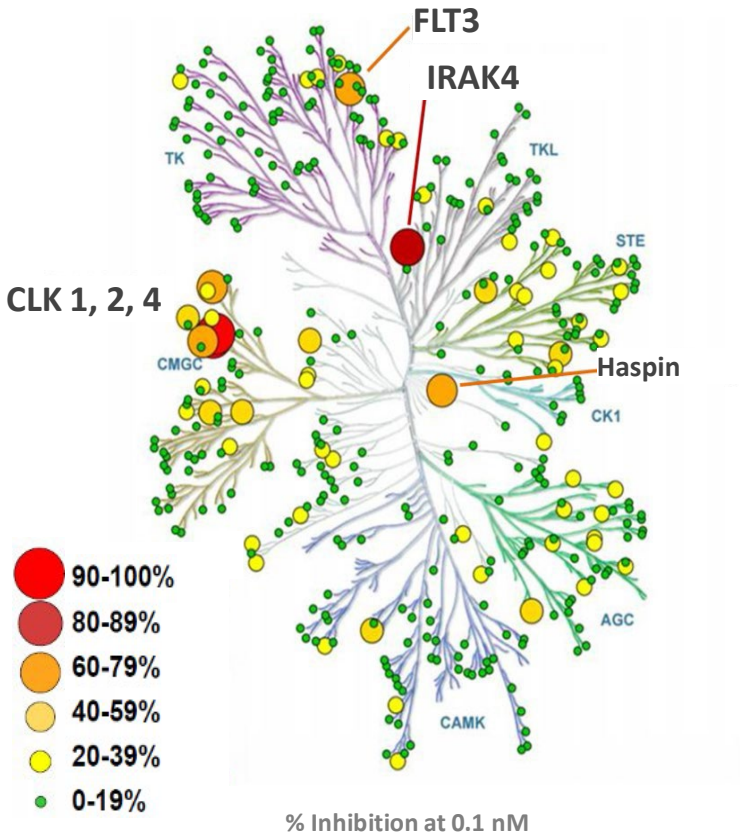


Illustration reproduced courtesy of Cell Signaling Technology

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 (F691L)	**20**
FLT3 (N841I)	**16**
Haspin (GSG2)	32
CLK1	10
CLK2	20
CLK3	>20,000
CLK4	14
TrkA	130

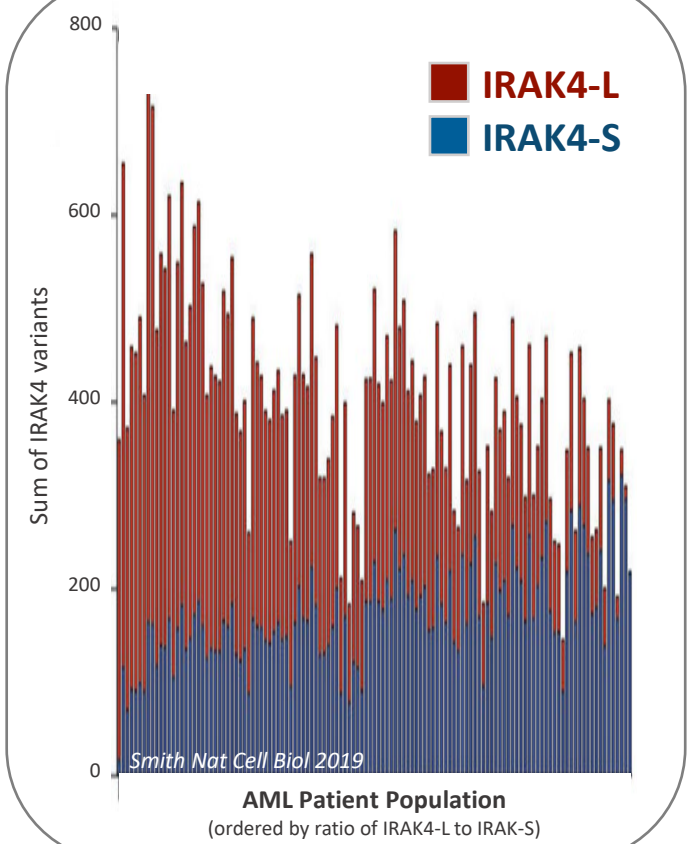
DiscoverX Kinase Panel
(378 kinases screened)

Binds tightly to IRAK4

Binds tightly to FLT3

IRAK4 is a disease driver in almost 100% of AML

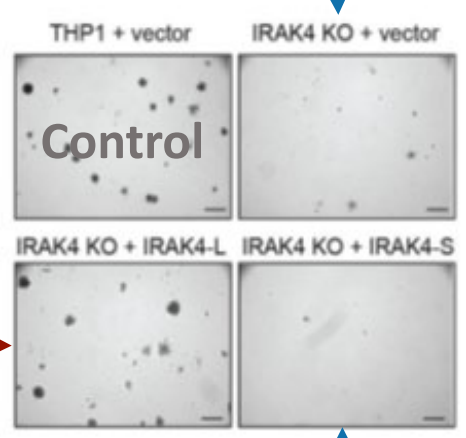
IRAK4-L is expressed in nearly all AML patients

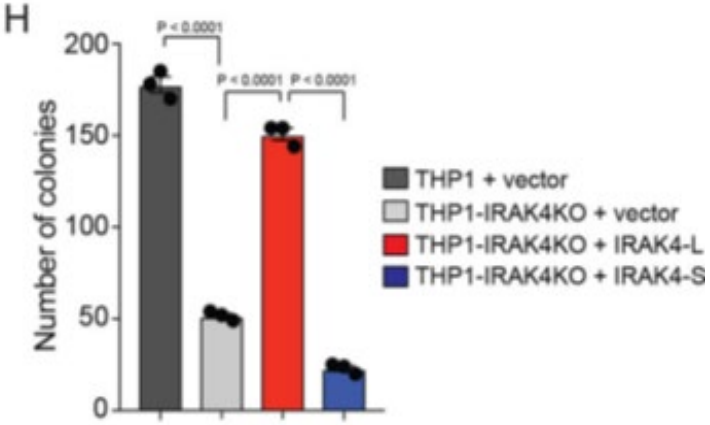


IRAK4-L is oncogenic in AML

Knocking out IRAK4 stops leukemic activity

(similar findings using CRISPR/CAS9)





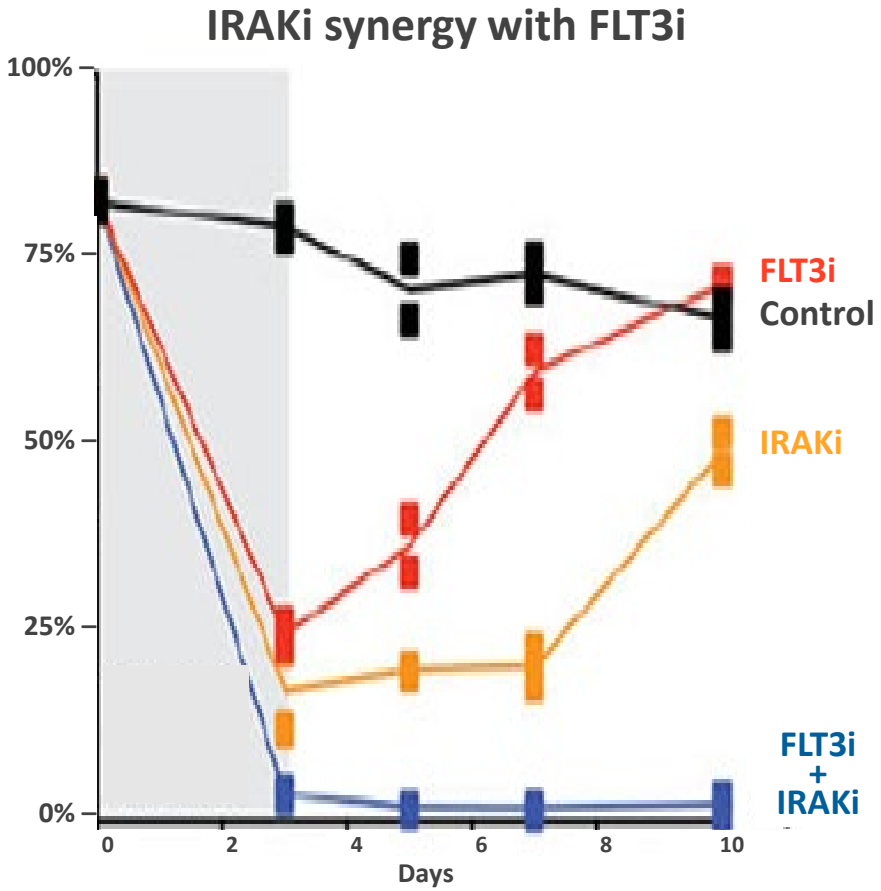
adding back IRAK4-L restarts activity

adding back IRAK4-S has no effect

Smith et al. Nat Cell Biol 2019

FLT3 is a disease driver in 33% of AML

And FLT3i is synergistic with IRAK4i



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

[1] *Melgar Sci Transl Med 2019*

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[1]

Strategy in AML

1

Demonstrate safety

102 AML patients[1] treated in TakeAim Leukemia Ph 1/2 study, emavusertib was well tolerated

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

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

1 – As of October 31, 2024

Safety profile in AML

- 102 patients treated in AML
- Shown to be well tolerated
- No dose-limiting myelosuppression has been observed

Grade 3+ Treatment-Related Adverse Event Reported in > 1 Patients, n (%)	200 mg BID (n=17)	300 mg BID (n=75)	400 mg BID (n=8)	500 mg BID (n=2)	Total (n=102)
# patients having grade 3+ TRAEs	1 (5.9)	29 (38.7)	3 (37.5)	1 (50.0)	34 (33.3)
Blood creatine phosphokinase increased	0	6 (8.0)	0	0	6 (5.9)
Neutropenia	0	5 (6.7)	1 (12.5)	0	6 (5.9)
Anaemia	0	5 (6.7)	0	0	5 (4.9)
Platelet count decreased	0	3 (4.0)	0	0	3 (2.9)
Rhabdomyolysis*	0	2 (2.7)	1 (12.5)	0	3 (2.9)
Syncope	0	1 (1.3)	1 (12.5)	1 (50.0)	3 (2.9)
Aspartate aminotransferase increased	0	2 (2.7)	0	0	2 (2.0)
Febrile neutropenia	0	1 (1.3)	1 (12.5)	0	2 (2.0)
Leukopenia	0	2 (2.7)	0	0	2 (2.0)
Orthostatic hypotension	0	2 (2.7)	0	0	2 (2.0)
Thrombocytopenia	0	2 (2,7)	0	0	2 (2.0)

Source: TakeAim Leukemia FLT3 Clinical Presentation ASH 2024. Data as of October 31, 2024

* Three events of rhabdomyolysis were investigator-reported, 1/3 met laboratory defined criteria for rhabdomyolysis (CPK >10 x ULN and SCr ≥ 1.5 x ULN).

Abbreviations: Treatment Related Adverse Event (TRAE) and Upper Limit Normal (ULN)

Strategy in AML

1

Demonstrate safety

102 AML patients[1] treated in TakeAim Leukemia Ph 1/2 study, emavusertib was well tolerated

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

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

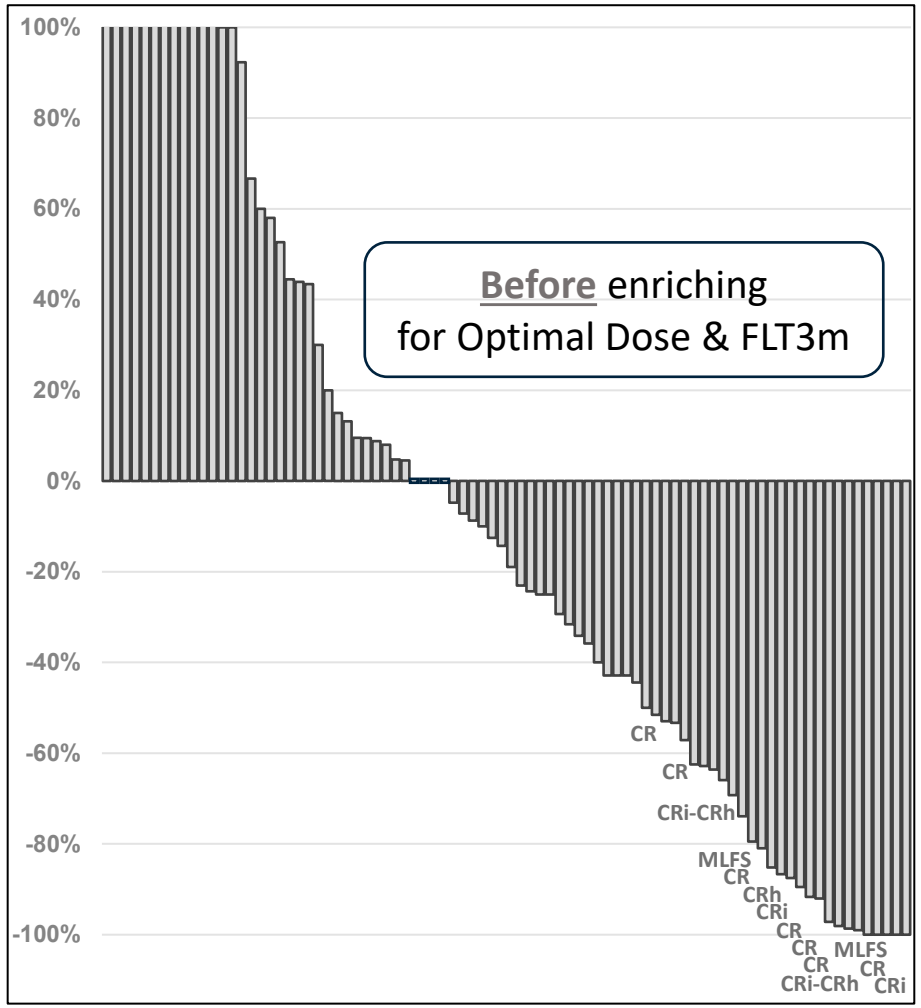
Pursue partnership to maximize potential commercial opportunity

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

1 – As of October 31, 2024

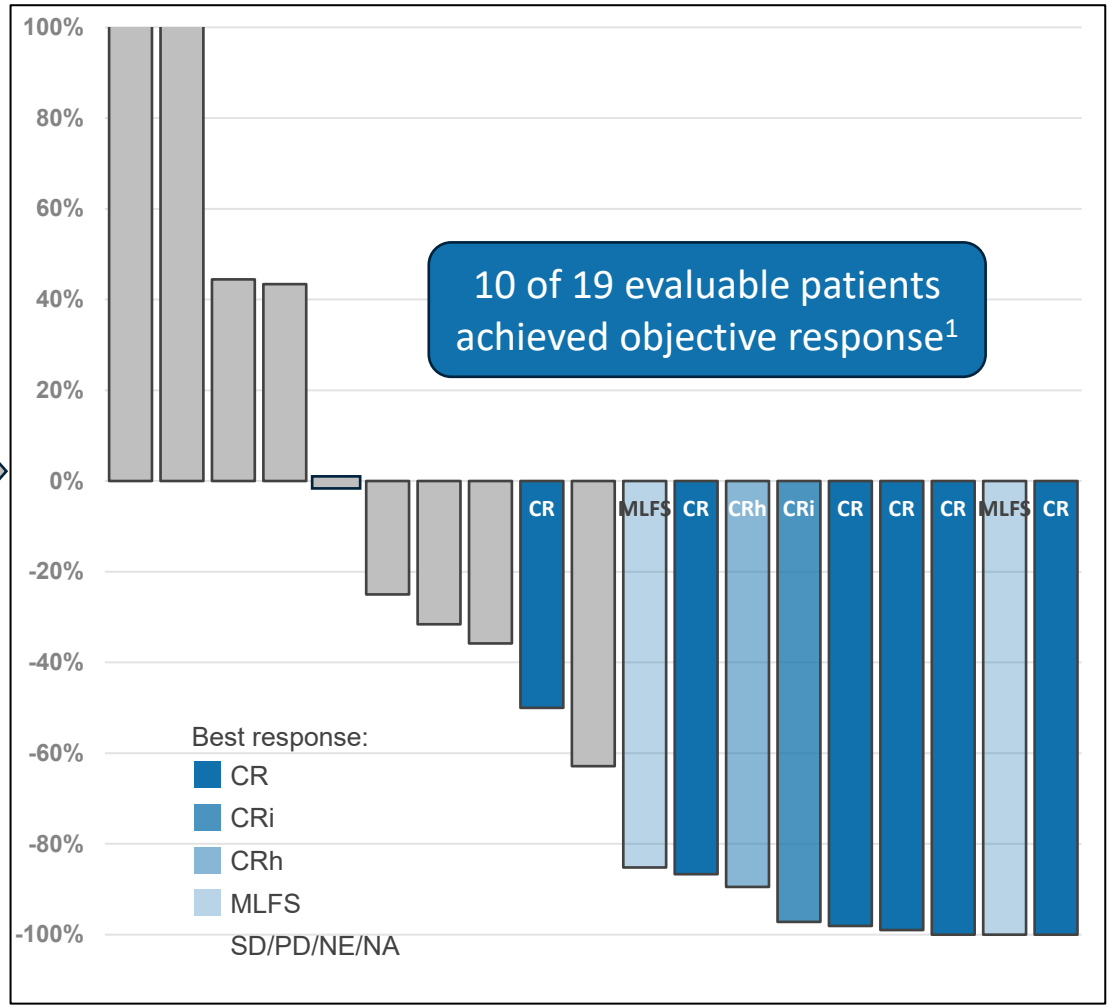
Single-agent activity demonstrated in AML

All Patients, All Dose Levels



Enrich for Dose & Mutation Status

Patients with < 3 lines of prior therapy with FLT3 mutation and treated at 300mg BID



Data include all R/R AML patients determined to be evaluable for objective response using baseline and post-treatment marrow assessments as of Oct 31, 2024.
Abbreviations: Complete Remission with incomplete count recovery (CRi), Complete Remission with partial hematological recovery (CRh), Morphologic Leukemia-Free State (MLFS), Stable Disease (SD); Progressive Disease (PD), Not Evaluable (NE) and Not Assessed (NA)

Source: TakeAim Leukemia FLT3 Clinical Presentation ASH 2024. Data as of October 31, 2024

1 - 2 of 21 patients were treated, but discontinued treatment prior to first disease response assessment (death occurred at Day 8 and Day 13, respectively), and were not included as evaluable.

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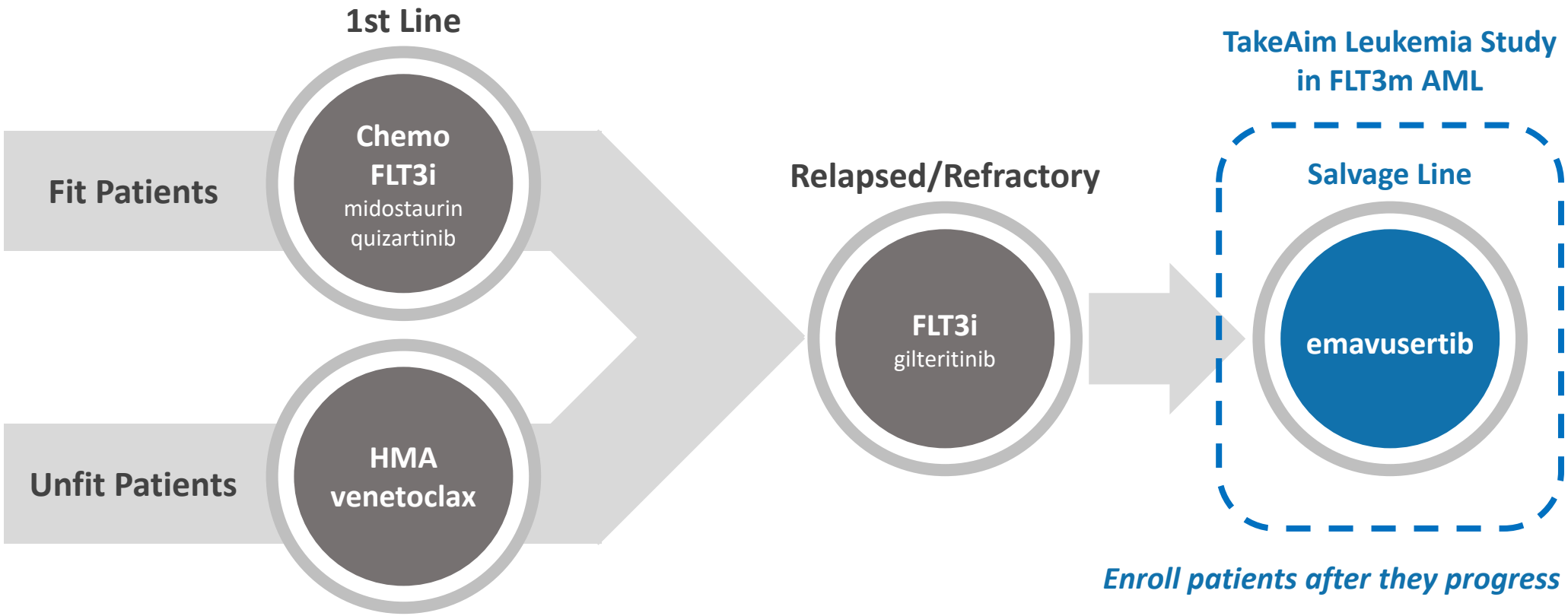
1 – As of October 31, 2024

TakeAim Leukemia

Study design



Enroll patients after they progress on FLT3i

Objective is to demonstrate that by blocking both FLT3 and IRAK4, salvage line patients can achieve an objective response (IRAK4i overcomes adaptive resistance to FLT3i)

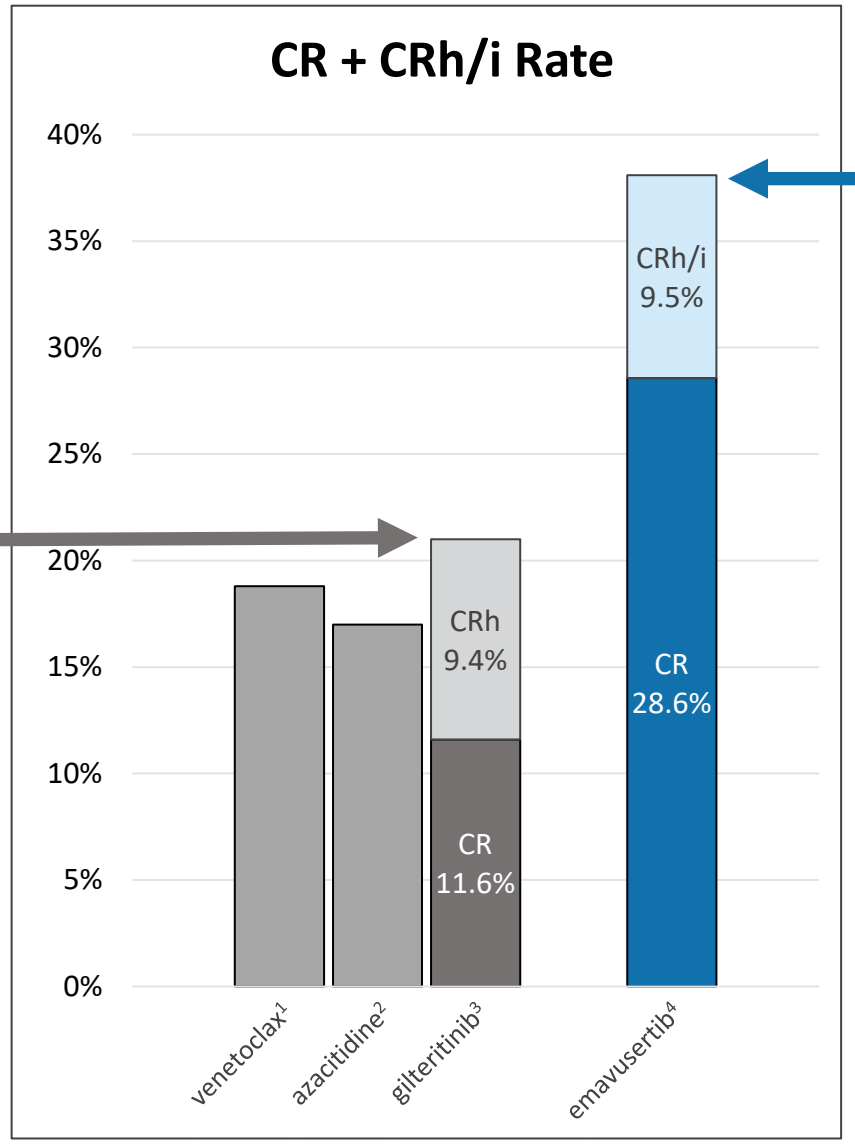
Updated Clinical Data in FLT3m AML

FLT3i-naïve

Benchmark is 21%

13% of patients were previously treated with a FLT3i

(source: FDA label)



Salvage Line

emavusertib is 38%

81% of patients were previously treated with a FLT3i

(n=21)

Emavusertib is a potential best-in-class therapy in FLT3 AML

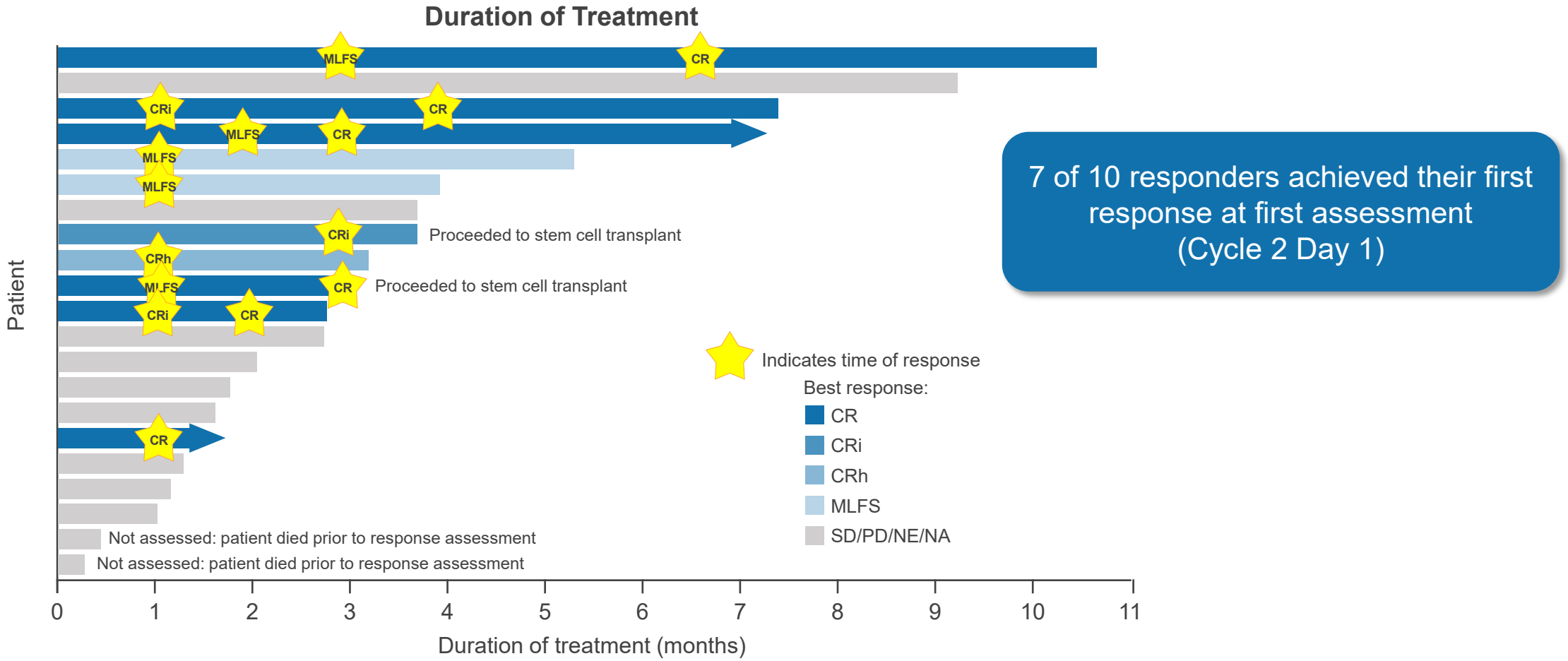
(as monotherapy)

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

Emavusertib data include 21 patients < 3 lines of prior therapy treated with emavusertib monotherapy at 300mg BID

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

Updated Clinical Data in FLT3m AML



7 of 10 responders achieved their first response at first assessment (Cycle 2 Day 1)

Presented at ASH 2024, data as of October 31, 2024
Includes 21 patients < 3 lines of prior therapy treated with emavusertib monotherapy at 300mg BID

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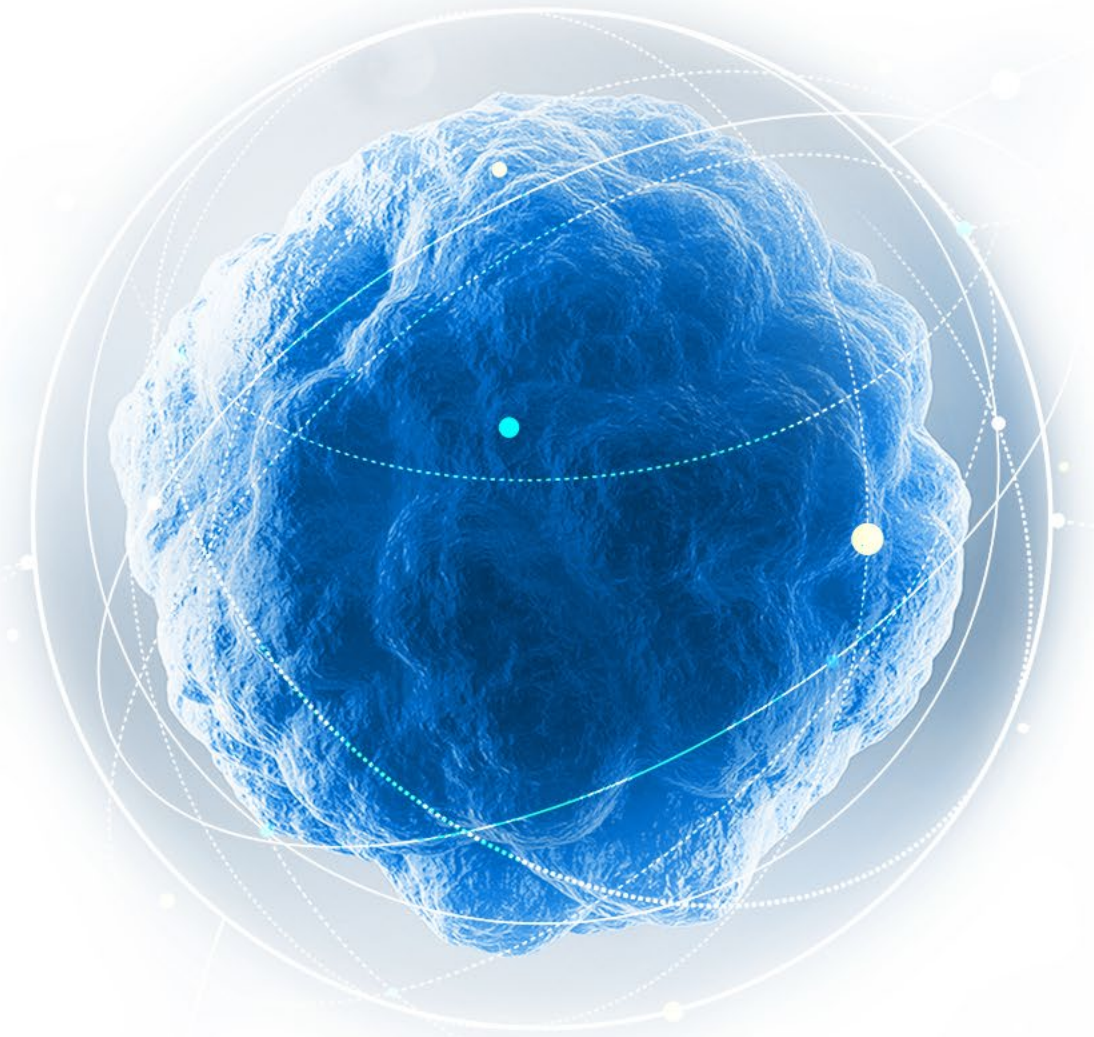
1 – As of October 31, 2024

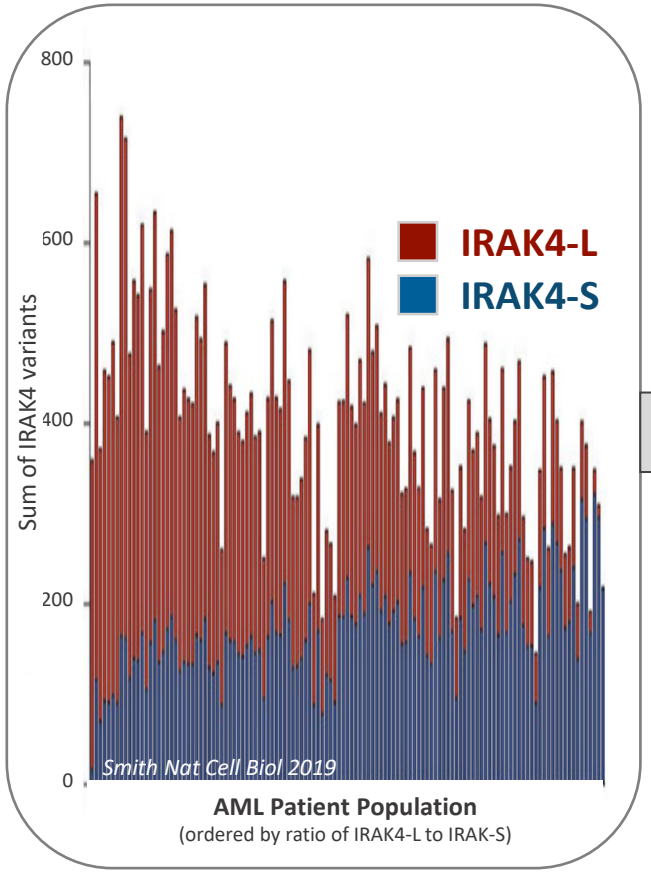
Emavusertib
in All Comers



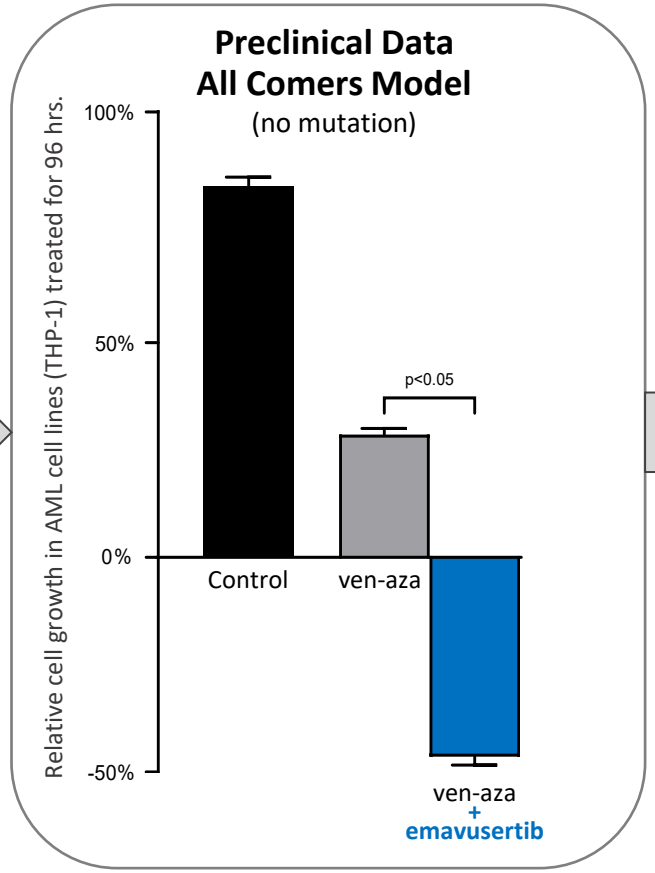
CURIS®

Ema-Ven-Aza triplet targets all comers in frontline AML

oncogenic IRAK4-L is expressed in nearly all AML patients



emavusertib is synergistic with ven-aza in preclinical studies



Curis AML MDS poster, EHA 2021

ema-ven-aza triplet combination

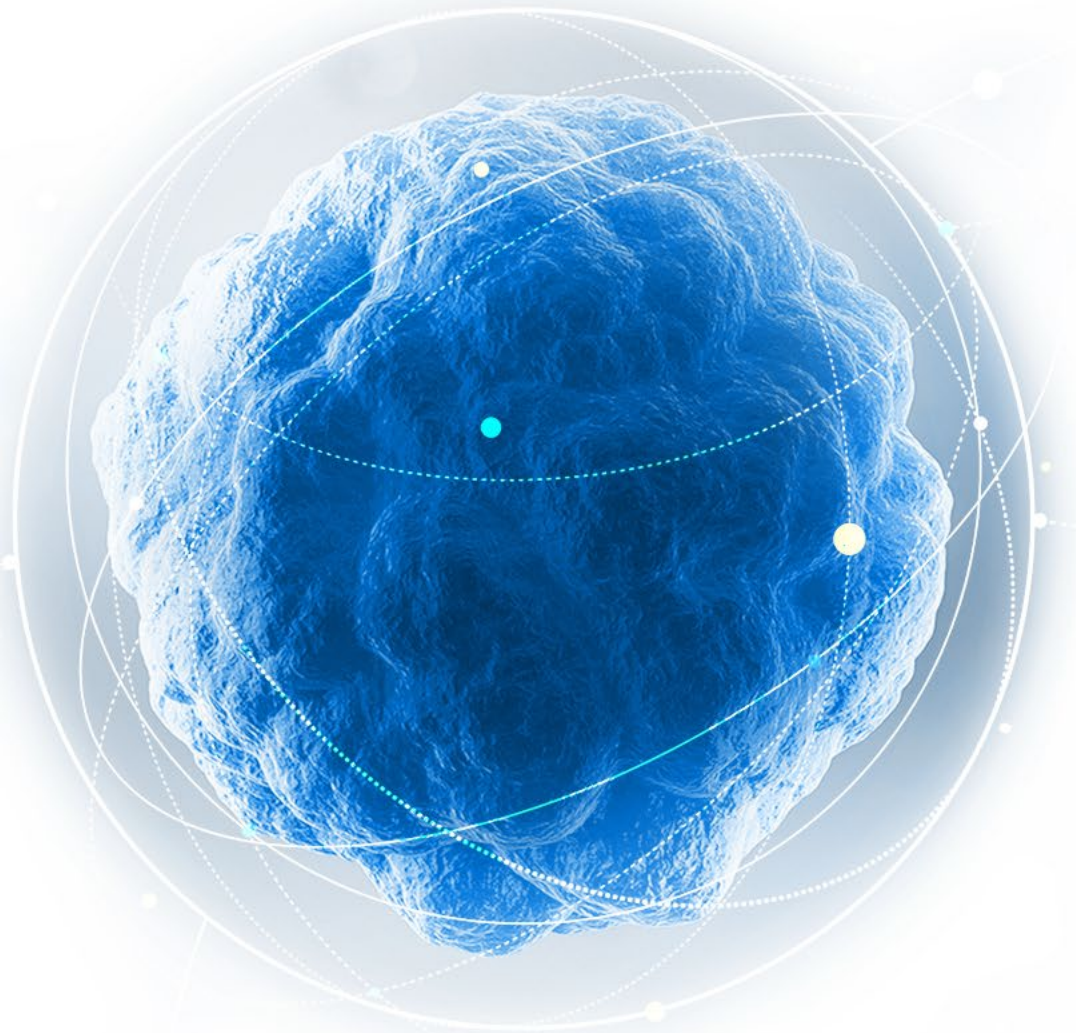
Ph 1 study initiated 1H2024

initial safety data expected Q1 2025

Summary in AML

- Emavusertib, as monotherapy, has the potential to be the best-in-class therapy in FLT3m AML
- Emavusertib, in combination with ven-aza, has the potential to establish a new standard of care in frontline AML for all comers, regardless of mutation

Solid Tumors



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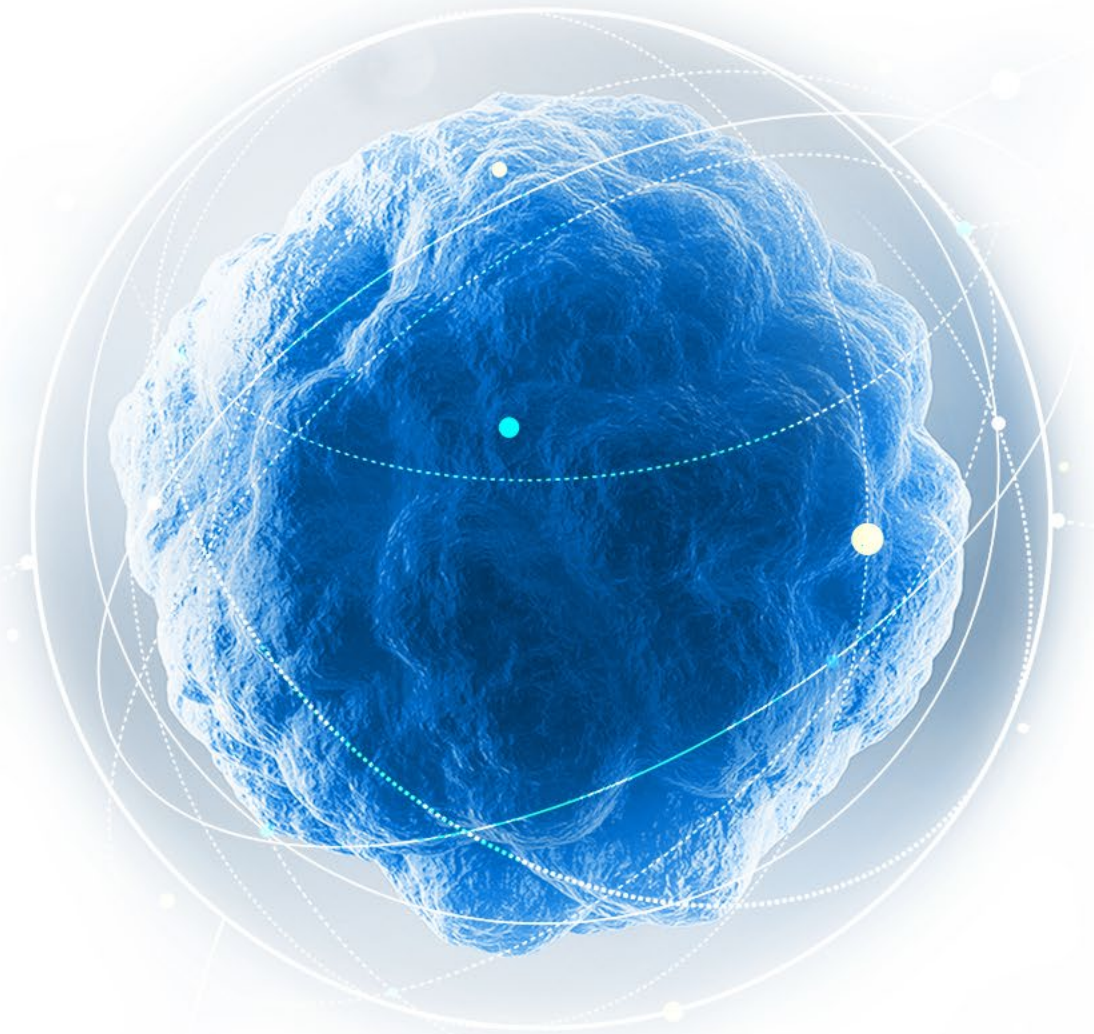
Ongoing studies (ISTs) of emavusertib in Solid Tumors

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

Abbreviation: Investigator Sponsored Trial (IST)

Other Information



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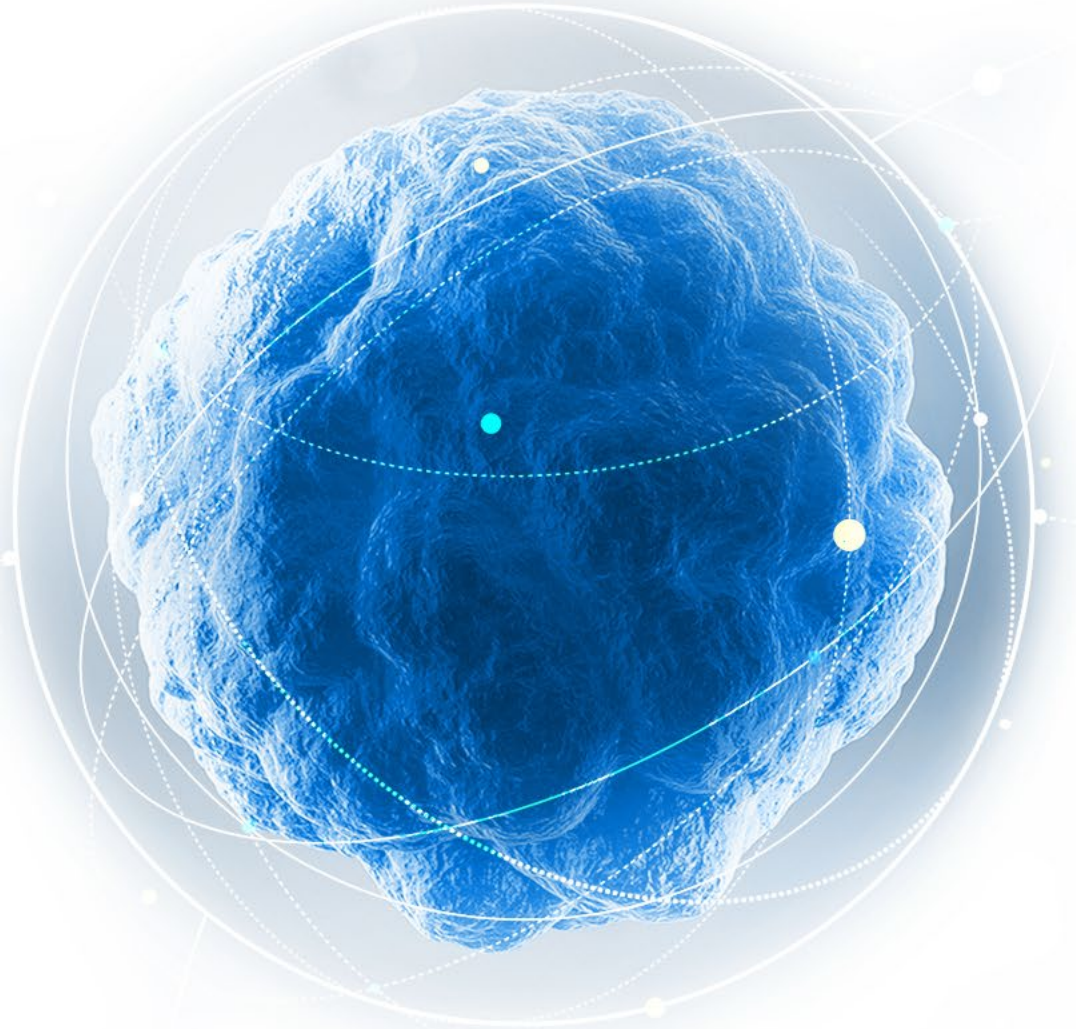
Financials and IP

	December 31, 2024[1]
$20.0M	Cash
8.5M	Shares Outstanding
12.0M	Fully Diluted Shares
2035	Composition of Matter IP on emavusertib (before potential extension)

	March 2025 Financing
~$9.0M	Net Proceeds
2.0M	Common Shares Issued
12.5M	Fully Diluted Shares Issued

[1] Excludes the impact of the March 2025 financing, which extends cash runway to Q4 2025

End of Presentation



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