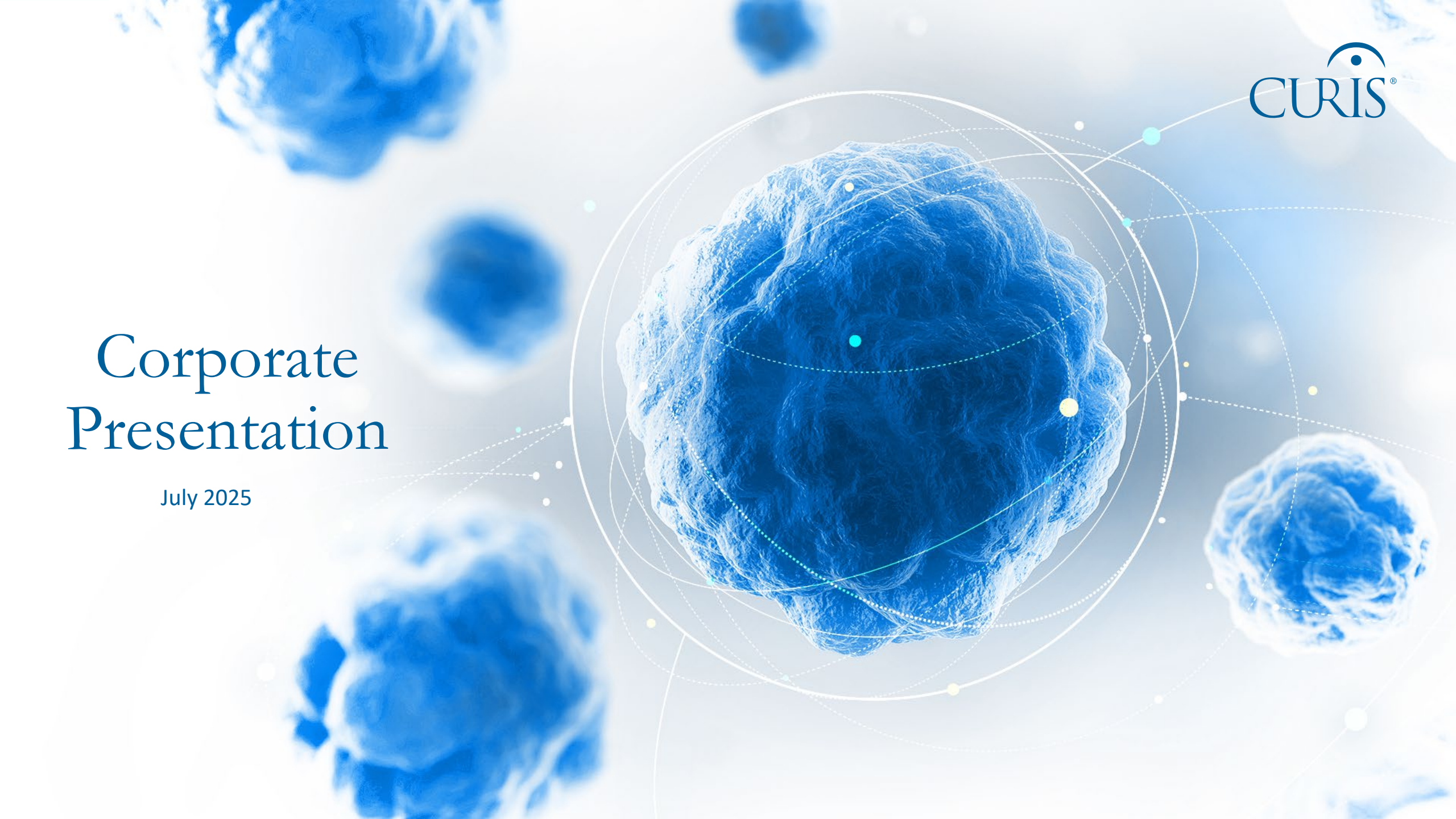


Corporate Presentation

July 2025

Cautionary note regarding forward looking statements and disclaimers

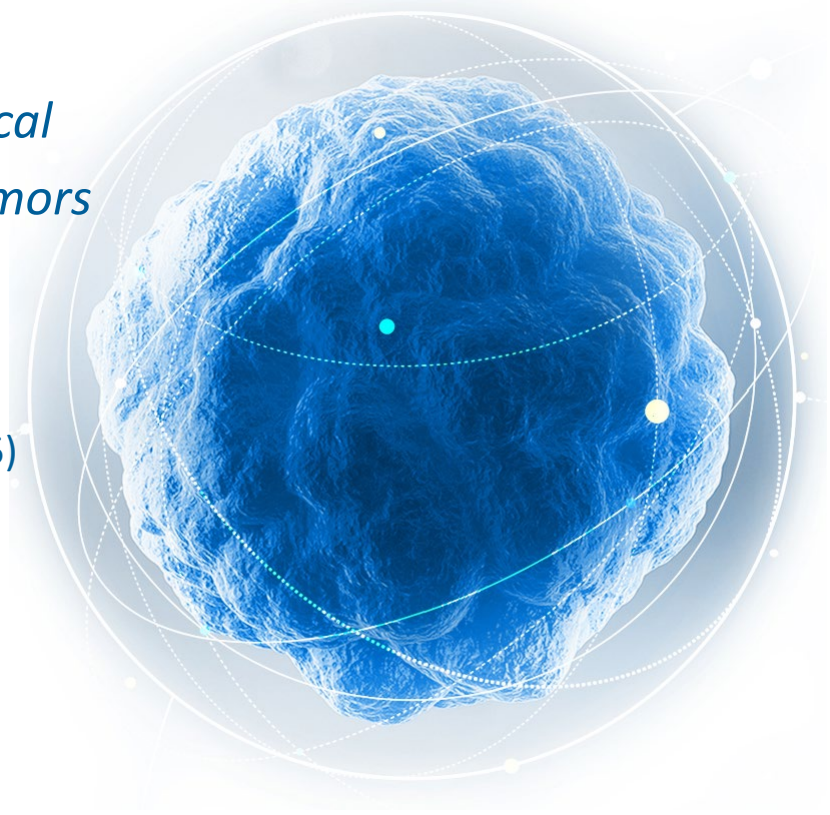
This presentation contains certain forward-looking statements about Curis, Inc. (“we,” “us,” or the “Company”) within the meaning of the Private Securities Litigation Reform Act of 1995, as amended. Words such as “expect(s),” “believe(s),” “will,” “may,” “anticipate(s),” “focus(es),” “plans,” “mission,” “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, therapeutic and market 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, 2024 and the Company's Quarterly Report on Form 10-Q for the quarter ended March 31, 2025 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 into Q4 ‘25*



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)

Curis Leadership Team

Experienced and Accomplished

James Dentzer
President and CEO

Mr. Dentzer is Chief Executive Officer and a member of the Board of Directors of Curis. Mr. Dentzer joined Curis in 2016 and was named CEO in 2018. Prior to joining Curis, Mr. Dentzer held senior leadership positions with Dicerna, Amicus, and Biogen. In 2021, Mr. Dentzer was named a Top 25 CEO in Biotech by The Healthcare Technology Report and currently serves on the Board of Directors of Imunon. Mr. Dentzer holds a B.A. in Philosophy from Boston College and an M.B.A. from the University of Chicago.

Ahmed Hamdy
Chief Medical Officer

Dr. Hamdy is Chief Medical Officer of Curis. Prior to joining Curis, he served as CEO and Chairman of the board of directors of Vincerx Pharma, Inc. Prior to Vincerx, Dr. Hamdy co-founded Acerta Pharma, LLC, and served as its CEO and CMO. Before Acerta, Dr. Hamdy was CMO of Pharmacyclics, Inc. Dr. Hamdy is an Adjunct Professor and a member of the Dean's Council at UC Santa Cruz. Dr. Hamdy received his MBBCH from the KasrAlainy School of Medicine at the University of Cairo, Egypt.

Jonathan Zung
Chief Development Officer

Dr. Zung is Chief Development Officer of Curis, joining the company in May 2023. Prior to joining Curis, Dr. Zung served as Chief Development Officer of Evelo Biosciences where he was responsible for the operational design and execution of Evelo's clinical programs. Dr. Zung held previous leadership roles at WCG, Covance, UCB, BMS, and Pfizer. Dr. Zung also serves on the advisory board of Saama Technologies. Dr. Zung received his Ph.D. in analytical chemistry from Emory University.

Diantha Duvall
Chief Financial Officer

Ms. Duvall is Chief Financial Officer of Curis, joining the company in August 2022. Prior to joining Curis, Ms. Duvall served as CFO of Genocea Biosciences. She was the CAO of Bioverativ and responsible for developing the financial profile. Earlier in her career, she held financial leadership positions of increasing responsibility at Biogen, Merck, and PricewaterhouseCoopers. Ms. Duvall holds a B.A. in economics and public policy from Colby College and an M.S. in accounting and MBA from Northeastern University.

Emavusertib Has Potential to Address Multiple Unmet Needs

In Lymphoma

- NFkB dysregulation in NHL is driven by two pathways: BCR and TLR[1]
- Existing therapies target BTK (in the BCR Pathway); emavusertib targets IRAK4 (in the TLR Pathway) enabling a dual-suppression of NFkB

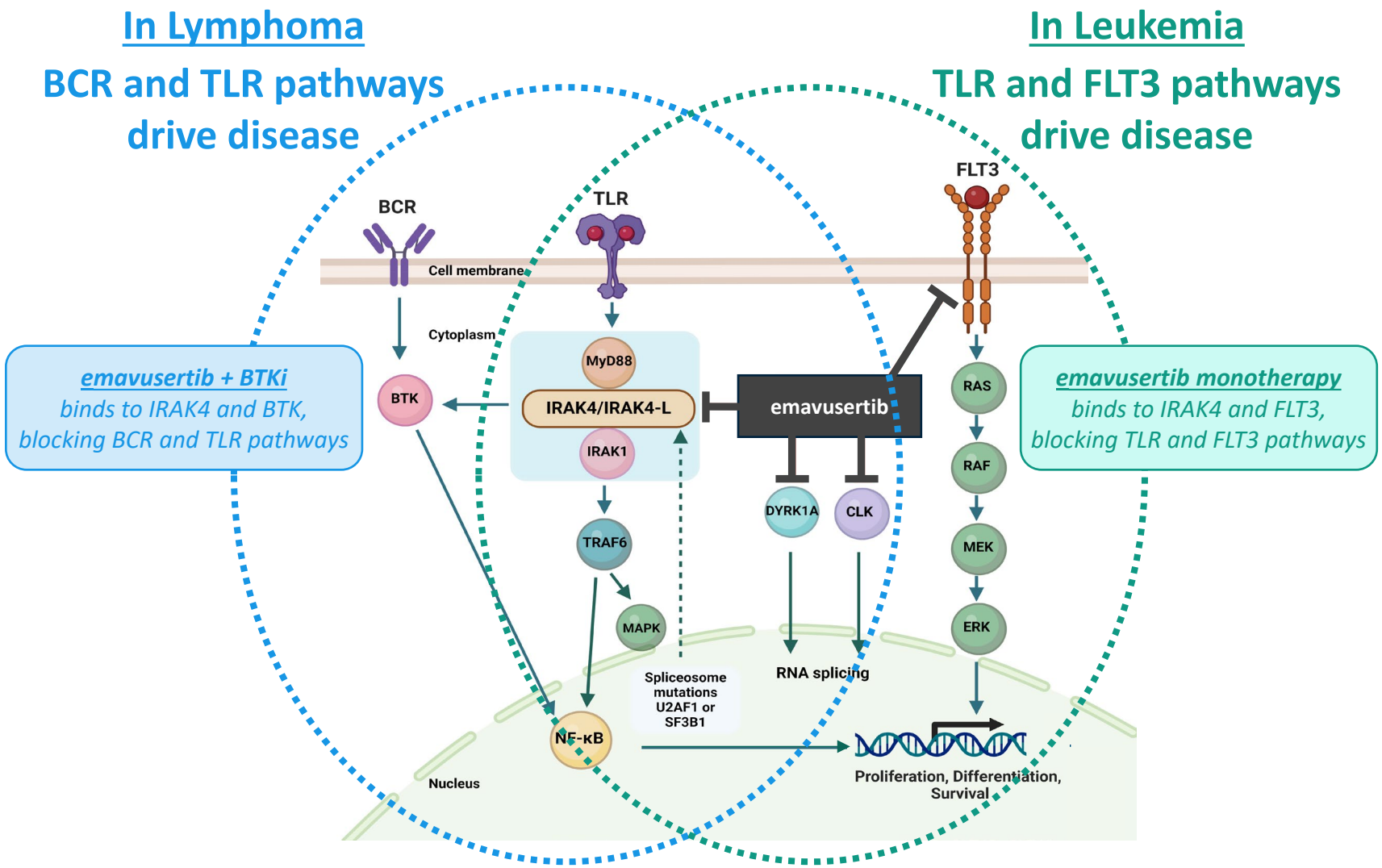
In Leukemia

- IRAK4 has emerged as the leading candidate driving innate immune signaling in AML and MDS[2]
- Concomitant targeting of IRAK4 and FLT3 is the most effective means to overcome the adaptive resistance incurred when targeting FLT3[3]

In Solid Tumors

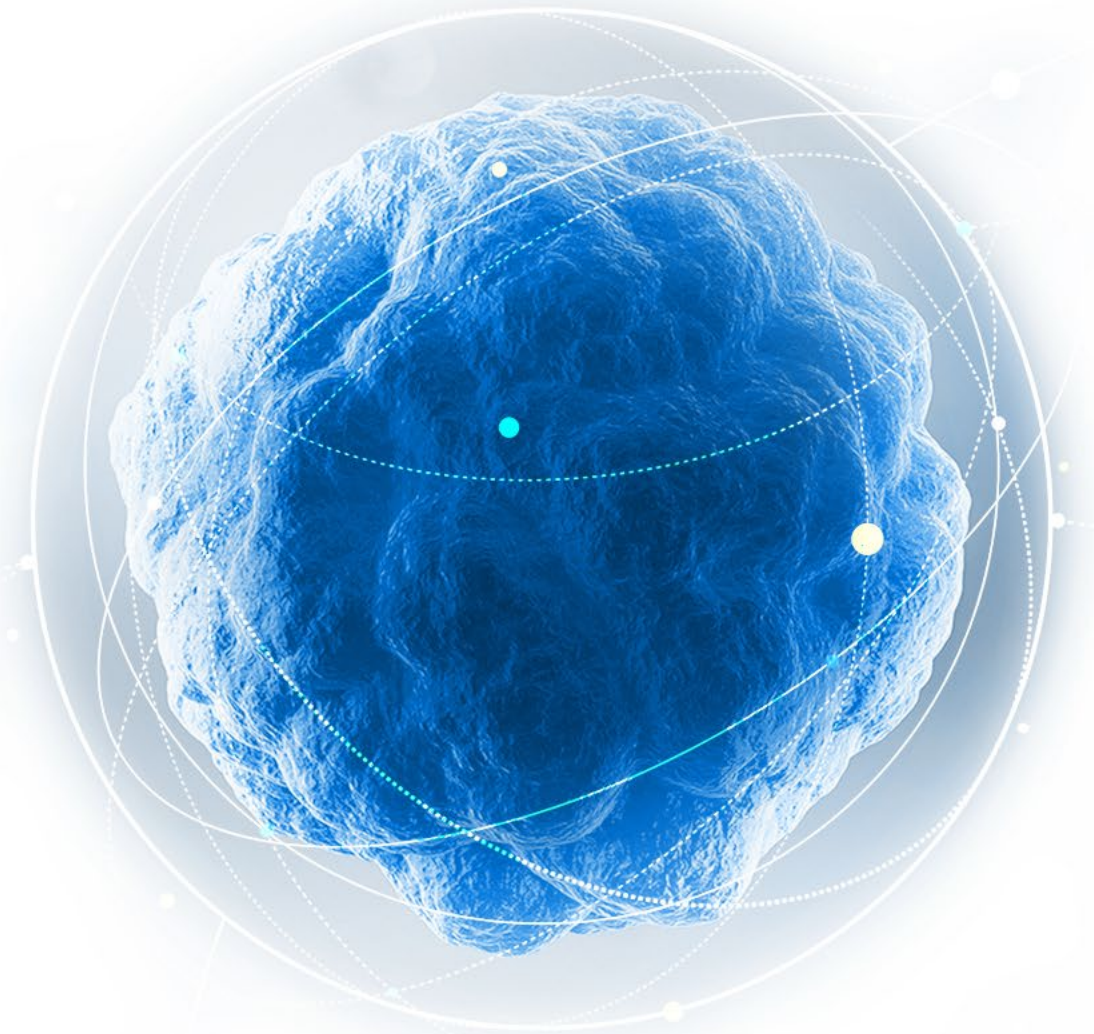
- IRAK4 upregulation is associated with increased phenotypically exhausted TILs, MDSCs, increased CD4+ T regs, and resistance to aPD-1 therapy[4,5]
- Initial data suggest emavusertib potentiates both chemo- and immunotherapies[6,7]

1 – Bennett, Curr Opin Hematol. 2022, Grafone, Oncol Rev. 2012, Kelly, J Exp Med. 2015, Wang, Cancer Cell. 2023; 2 – Smith, Nat Cell Biol. 2019; 3 - Melgar, Sci Transl Med. 2019; 4 - Martin Lasola, Cancer Immunol Res. 2017; 5 - Somani, Gastroenterology. 2022; 6 - Li, JCI Insight. 2019; 7 - 2024 IRAK4 Symposium

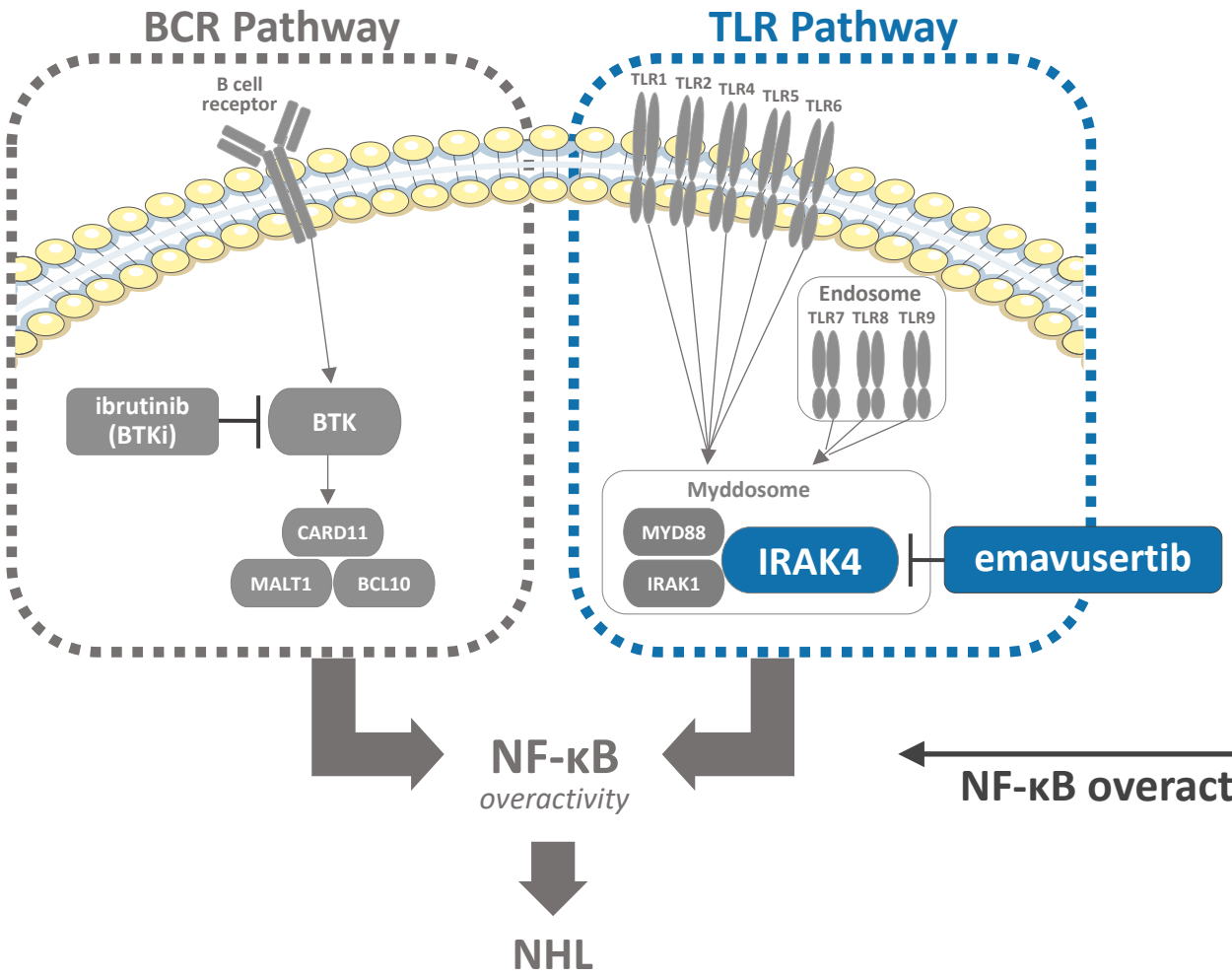


Bennett, Curr Opin Hematol. 2022, Grafone, Oncol Rev. 2012, Kelly, J Exp Med. 2015, Wang, Cancer Cell. 2023

Emavusertib
in NHL



Emavusertib Mechanism in NHL



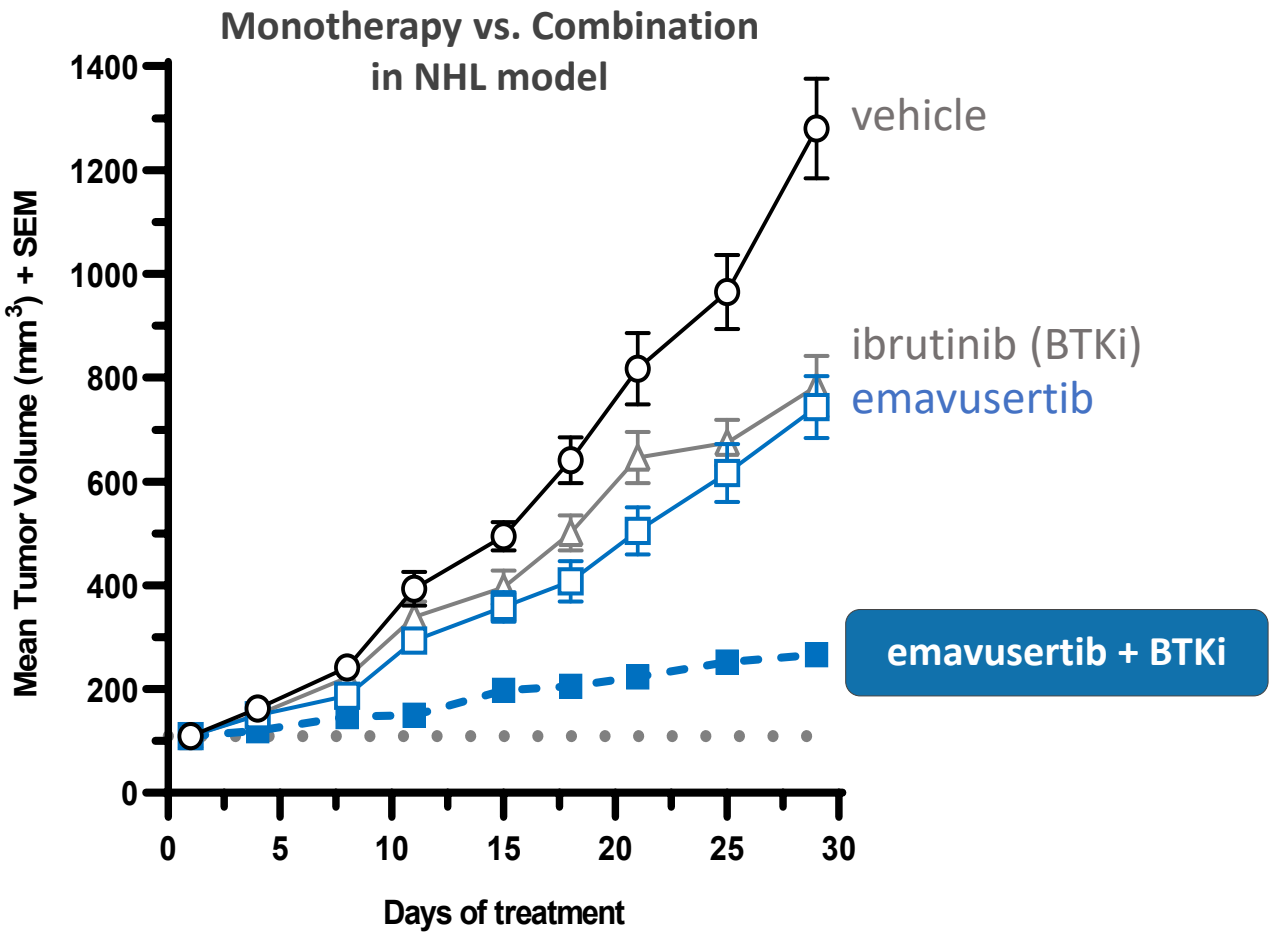
Dual blockade of the TLR and BCR pathways maximizes downregulation of NF-κB[1]

NF-κB overactivity drives disease in NHL

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

1 - Dadashian, Ca Res. 2019

Mechanism Demonstrated in Preclinical NHLModels



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

Safety profile in NHL

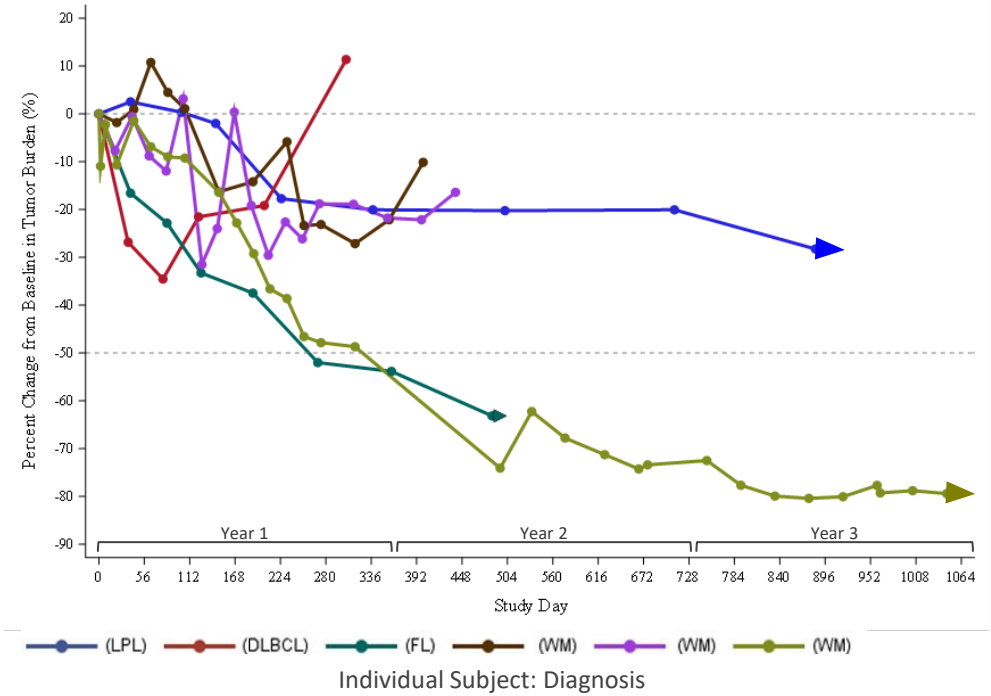
- 47 patients treated in NHL
- Shown to be well tolerated
- No dose-limiting myelosuppression has been observed
- Emavusertib crosses the BBB
- No dose-limiting CNS toxicities have been observed

Grade 3+ Treatment-Related Adverse Event Reported in > 1 Patients, n (%)	100 mg BID ema +ibr (n=13)	200 mg BID ema +ibr (n=27)	300 mg BID ema +ibr (n=7)	Total (n=47)
# patients having grade 3+ TRAEs	5 (38)	11 (41)	6 (86)	22 (47)
Neutropenia	4 (31)	1 (4)	0	5 (11)
Lipase increased	2 (15)	1 (4)	0	3 (6)
Platelet count decreased	0	2 (7)	1 (14)	3 (6)
Alanine aminotransferase increased	0	1 (4)	1 (14)	2 (4)
Amylase increased	2 (15)	0	0	2 (4)
Aspartate aminotransferase increased	0	1 (4)	1 (14)	2 (4)
Fatigue	0	1 (4)	1 (14)	2 (4)
Hyponatraemia	0	2 (7)	0	2 (4)
Leukopenia	2 (15)	0	0	2 (4)
Syncope	0	1 (4)	1 (14)	2 (4)

Safety data as of May 1, 2025
Abbreviation: Treatment Related Adverse Event (TRAE), ibrutinib (IBR), Dose Limiting Toxicity (DLT), Blood Brain Barrier (BBB), Central Nervous System (CNS), twice daily (BID)

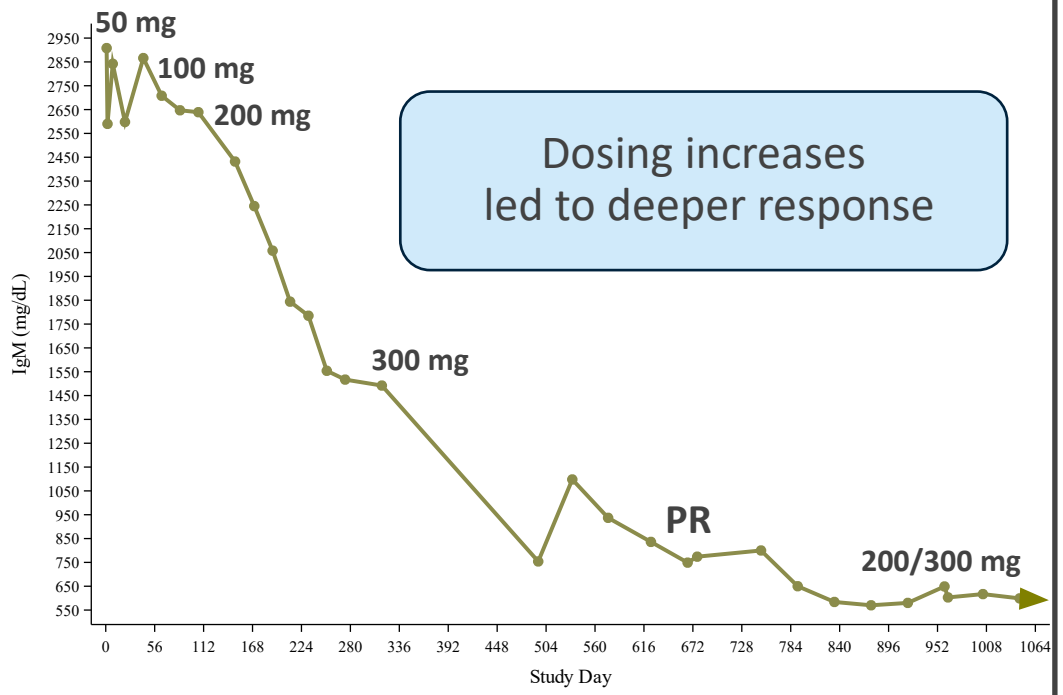
Single-agent activity demonstrated in NHL

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)

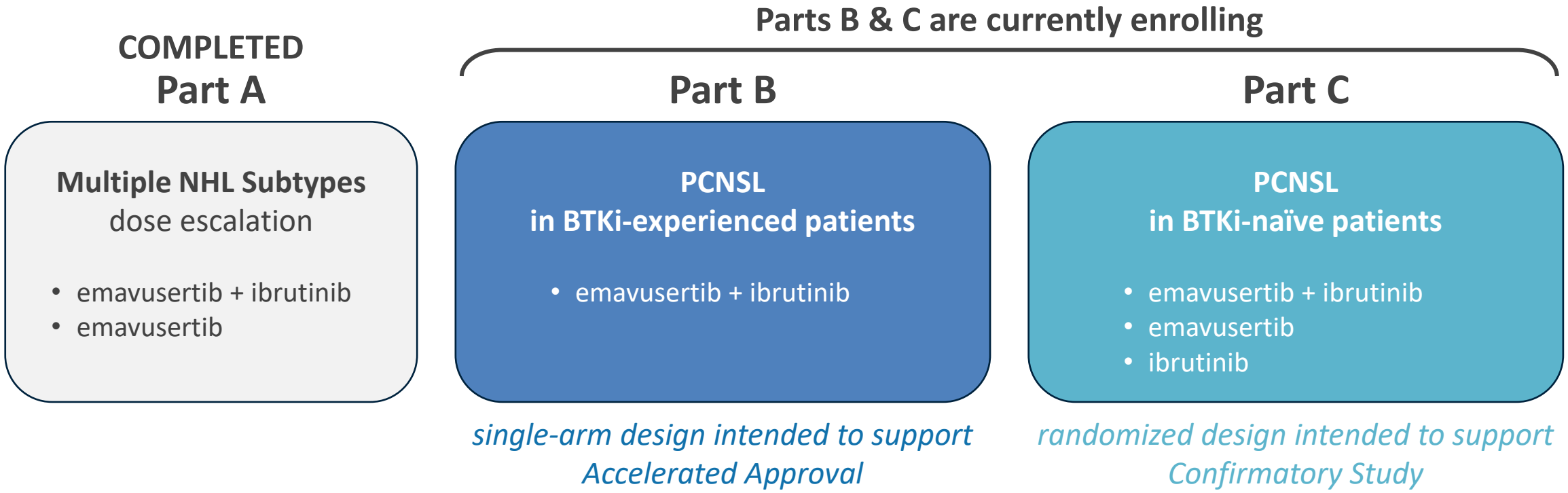


2022 IWWM Conference Presentation

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

Clinical Study Design

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



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.

PCNSL Clinical Data

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

In BTKi-naïve patients:

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

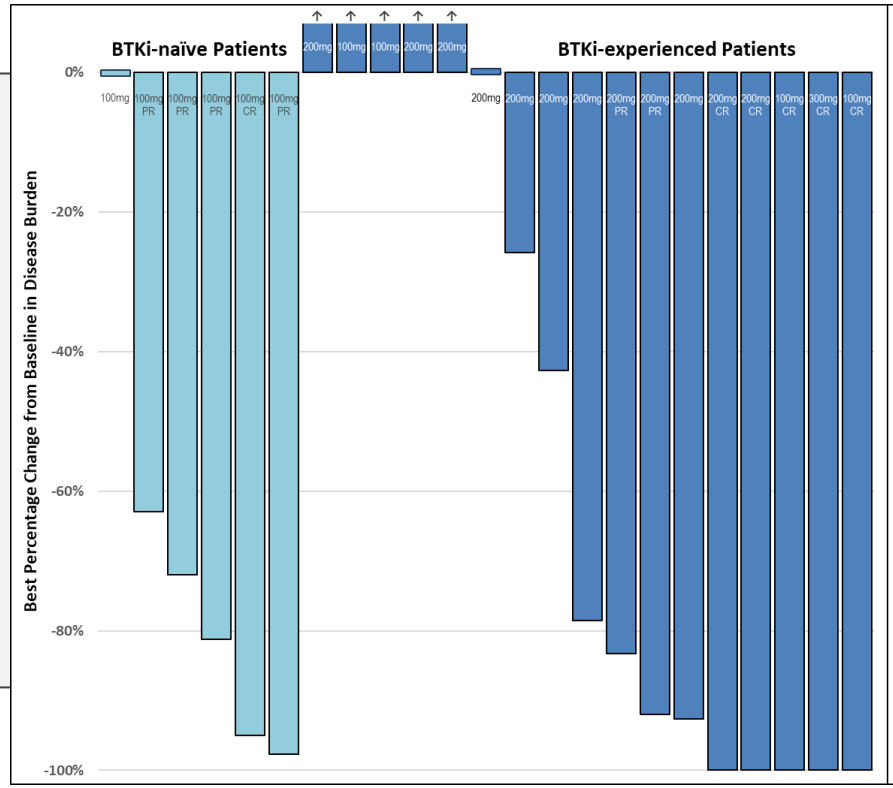
- *63% for ibr + ema (5 of 8 patients ITT)*
- *39% for ibr monotherapy[1] (15 of 38 patients ITT)*

In BTKi-experienced patients:

Adding emavusertib **reversed tumor growth**, with reductions in tumor size in 11 patients, incl 7 responses

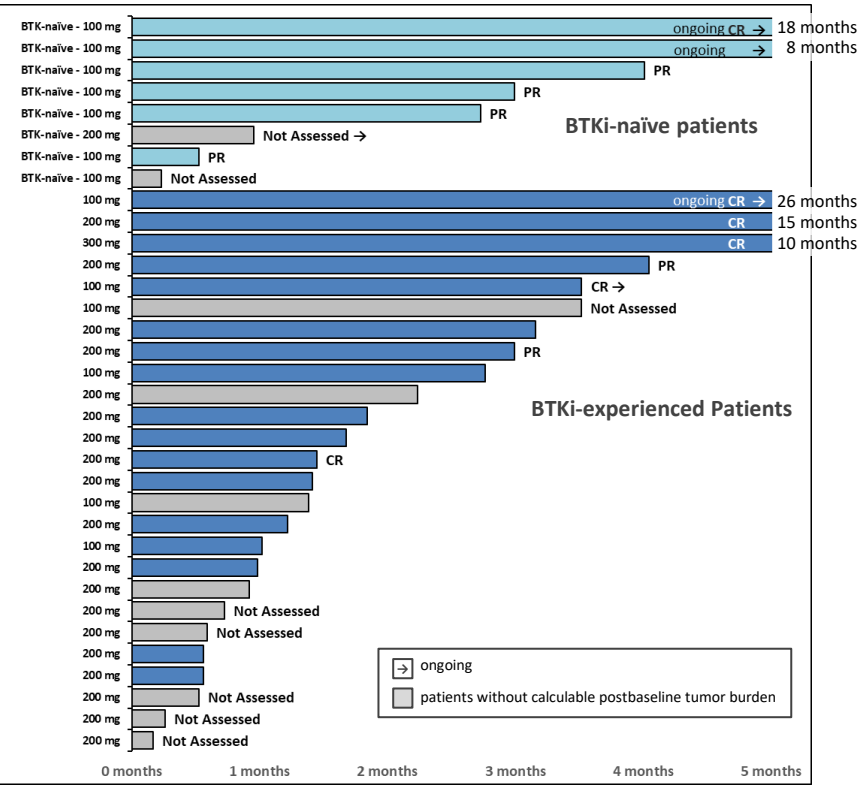
1 – Soussain, Eur J Cancer 2019
Abbreviation: Intent to Treat (ITT)

Reduction in Tumor Burden



Data include all patients with calculable postbaseline tumor burden at cutoff date

Duration of Treatment



Data include all patients treated as of cutoff date

Clinical data cutoff: May 1, 2025

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

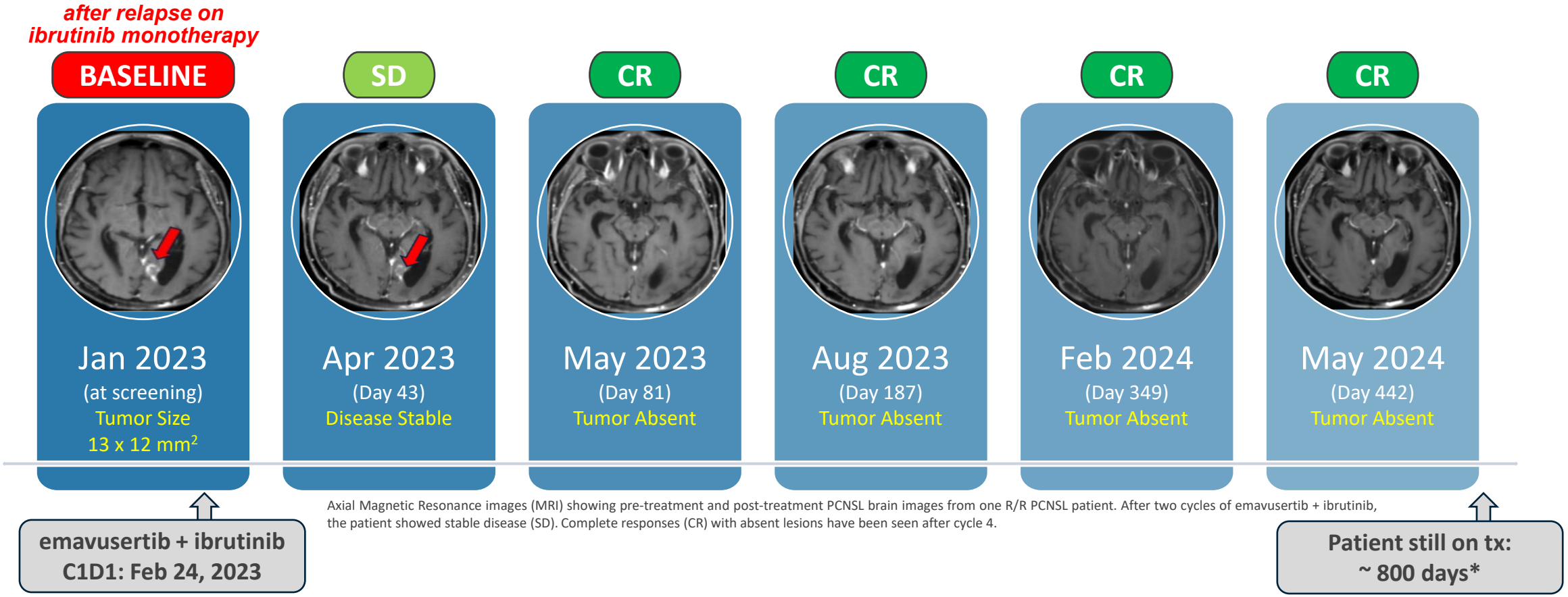
Prior Tx: Line 1: MTX, high-dose BCNU, Ara-C, thiotepa, WBRT, rituximab, and ASCT (PR)
Line 2: ibrutinib (CR)

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 May 1, 2025*

Next Steps

Expand across NHL, to 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

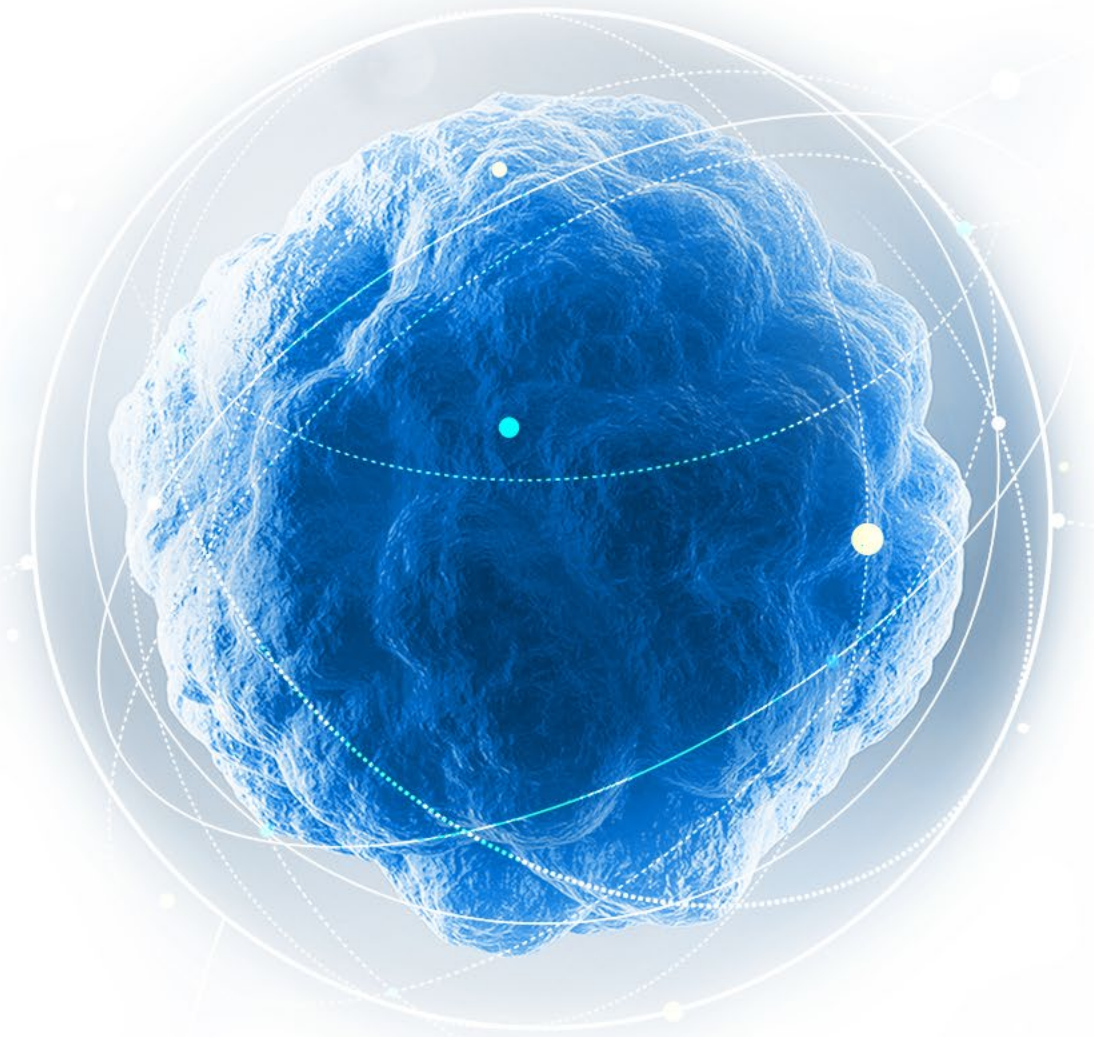
Abbreviations: Nuclear factor-κB (NF-kB), proteasome inhibitors (PI)

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-L and FLT3m are important drivers of disease

Emavusertib
Kinase Interaction Map

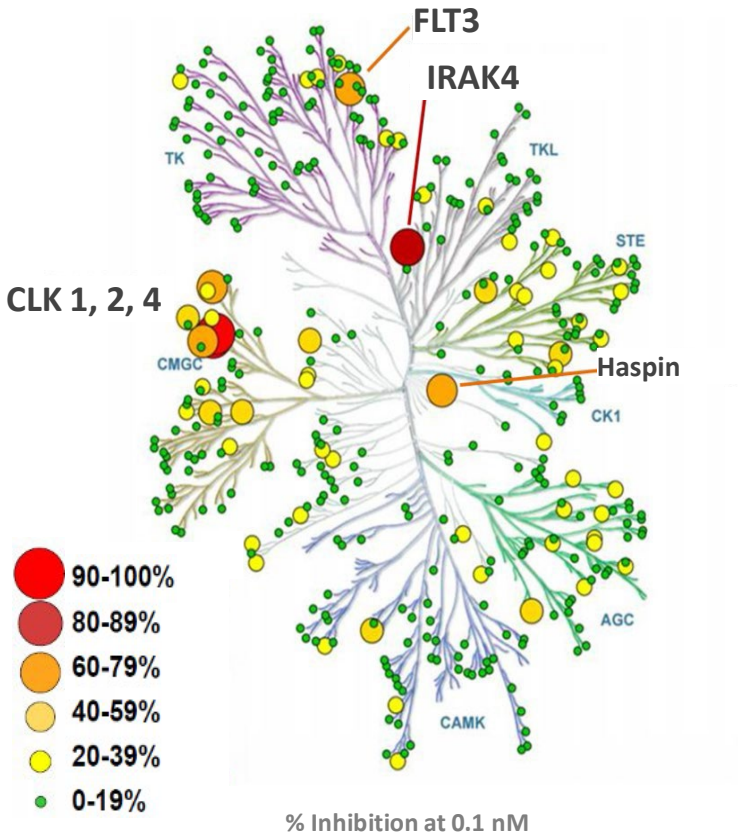


Illustration reproduced courtesy of Cell Signaling Technology

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

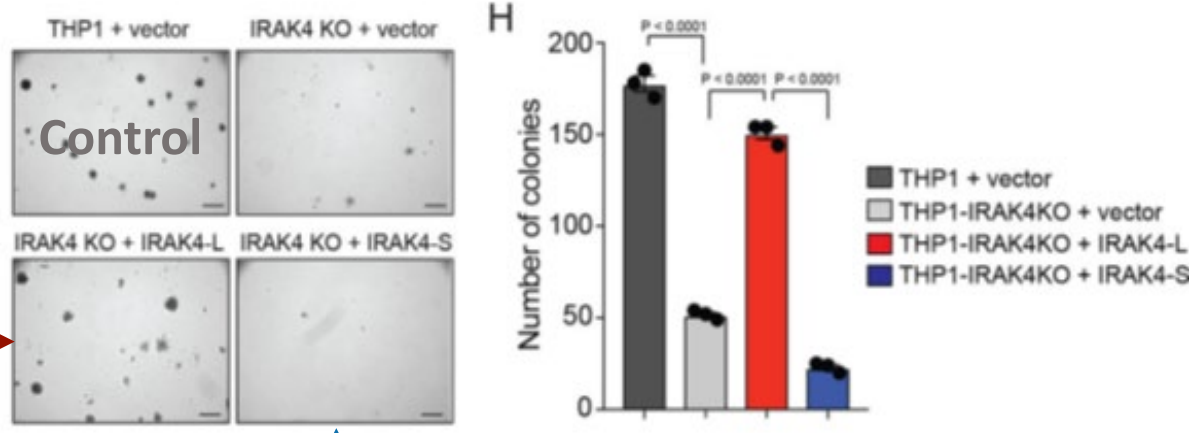
Binds tightly to FLT3

Abbreviation: FLT3 mutations (FLTm)

IRAK4-L is a disease driver in nearly all AML patients

IRAK4-L is oncogenic

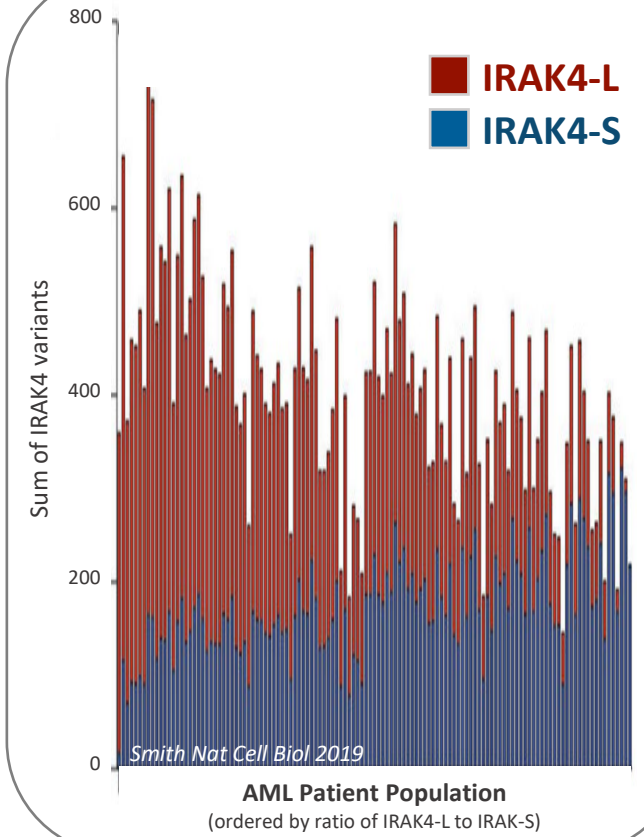
Knocking out IRAK4 stops leukemic activity



adding back IRAK4-L restarts activity

adding back IRAK4-S has no effect

IRAK4-L is expressed in nearly all AML patients

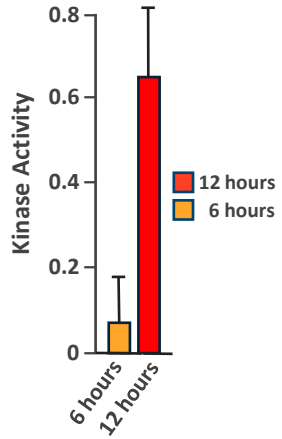


Smith et al. Nat Cell Biol 2019

FLT3m is a disease driver in ~1/3 of newly diagnosed AML patients[1]

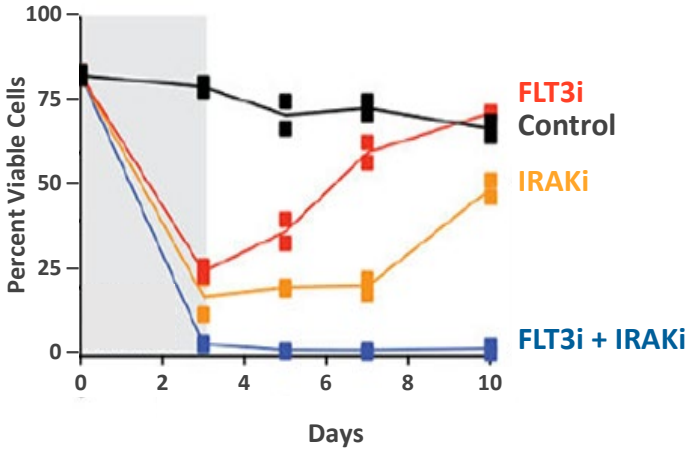
IRAK4 inhibition overcomes adaptive resistance to FLT3i

IRAK4 activity increased after treatment with FLT3i



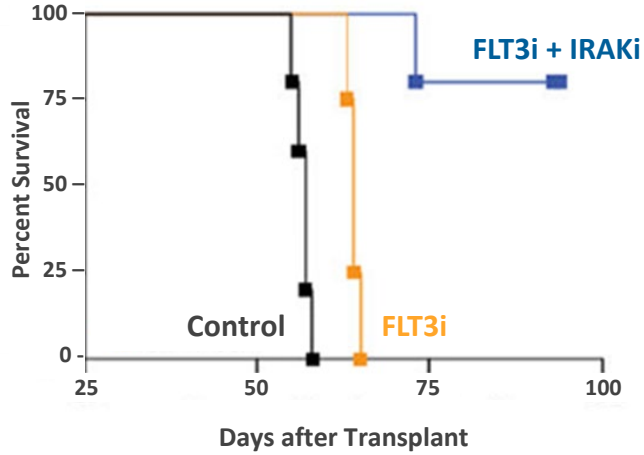
relative phospho-peptide intensity of IRAK4

IRAKi + FLT3i were synergistically cytotoxic



Viability of MLL-AF9; FLT3-ITD cells treated for 3 days with DMSO (control), quizartinib (0.5 µM), IRAKi (10 µM), and quizartinib + IRAKi

IRAKi + FLT3i significantly extended leukemia-free survival



Leukemia-free survival of NRGS mice xenografted with AML-019 patient cells and treated with quizartinib

Concomitant targeting of IRAK1 or IRAK4, alongside FLT3, is the most effective means to overcome the adaptive resistance incurred when targeting FLT3[2]

[1] *Kennedy, Front Oncol. 2020*
[2] *Melgar, Sci Transl Med. 2019*

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)

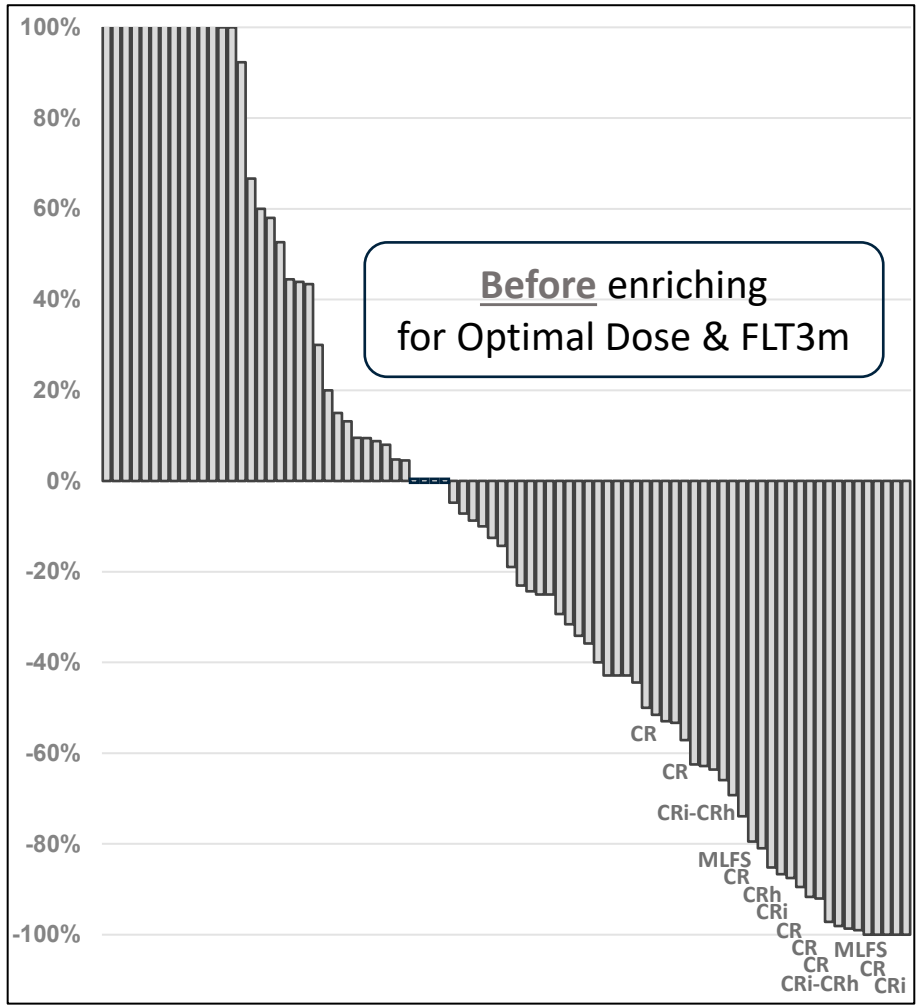
Source: TakeAim Leukemia FLT3 Clinical Presentation ASH 2024. Data as of October 31, 2024

* 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)

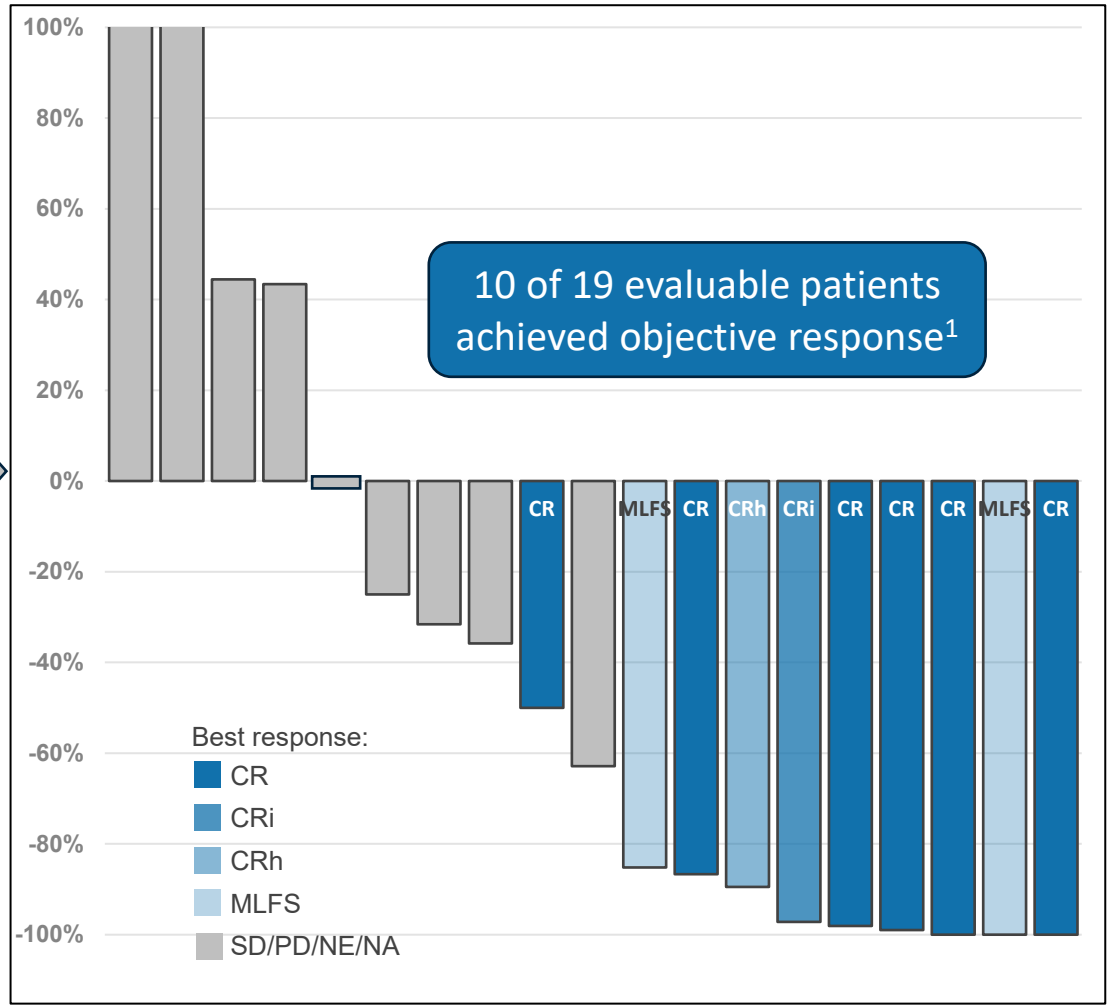
Single-agent activity demonstrated in AML

All Patients, All Dose Levels



Enrich for Dose & Mutation Status

Patients with < 3 lines of prior therapy with FLT3m and treated at 300mg BID



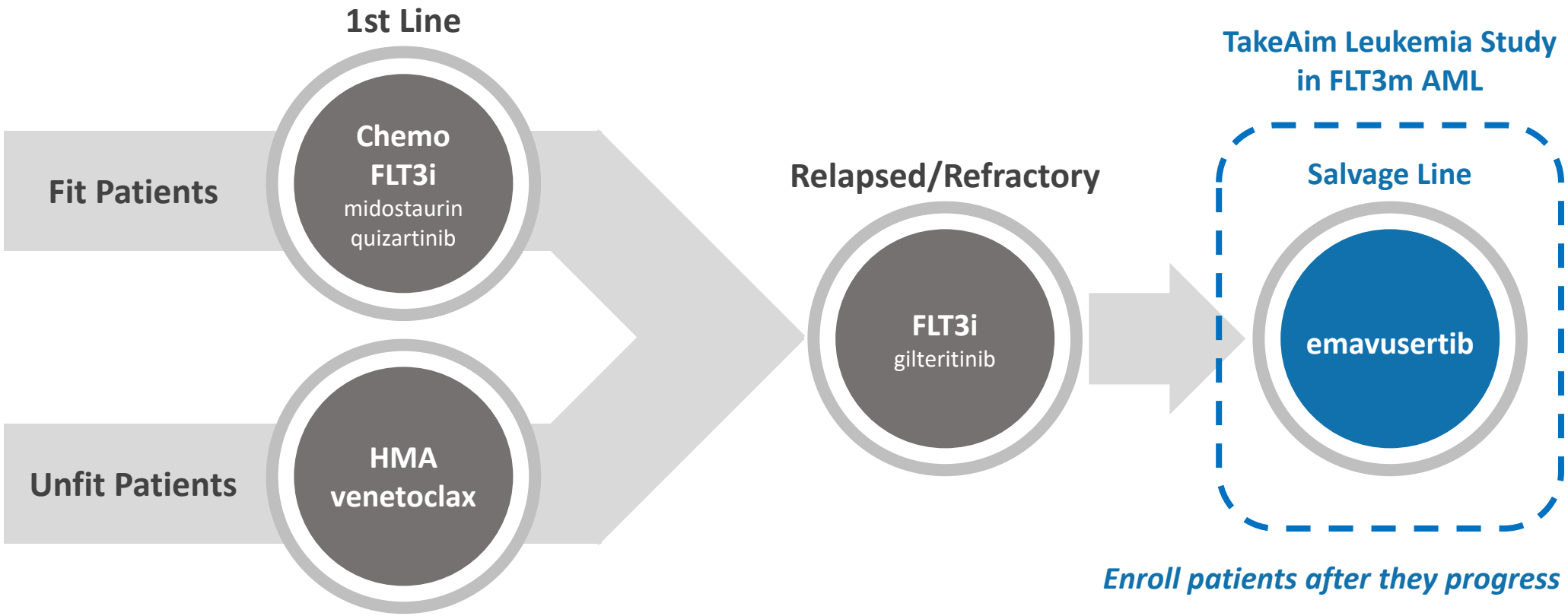
Data include all R/R AML patients determined to be evaluable for objective response using baseline and post-treatment marrow assessments as of Oct 31, 2024.
Abbreviations: Complete Remission with incomplete count recovery (CRi), Complete Remission with partial hematological recovery (CRh), Morphologic 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.

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)

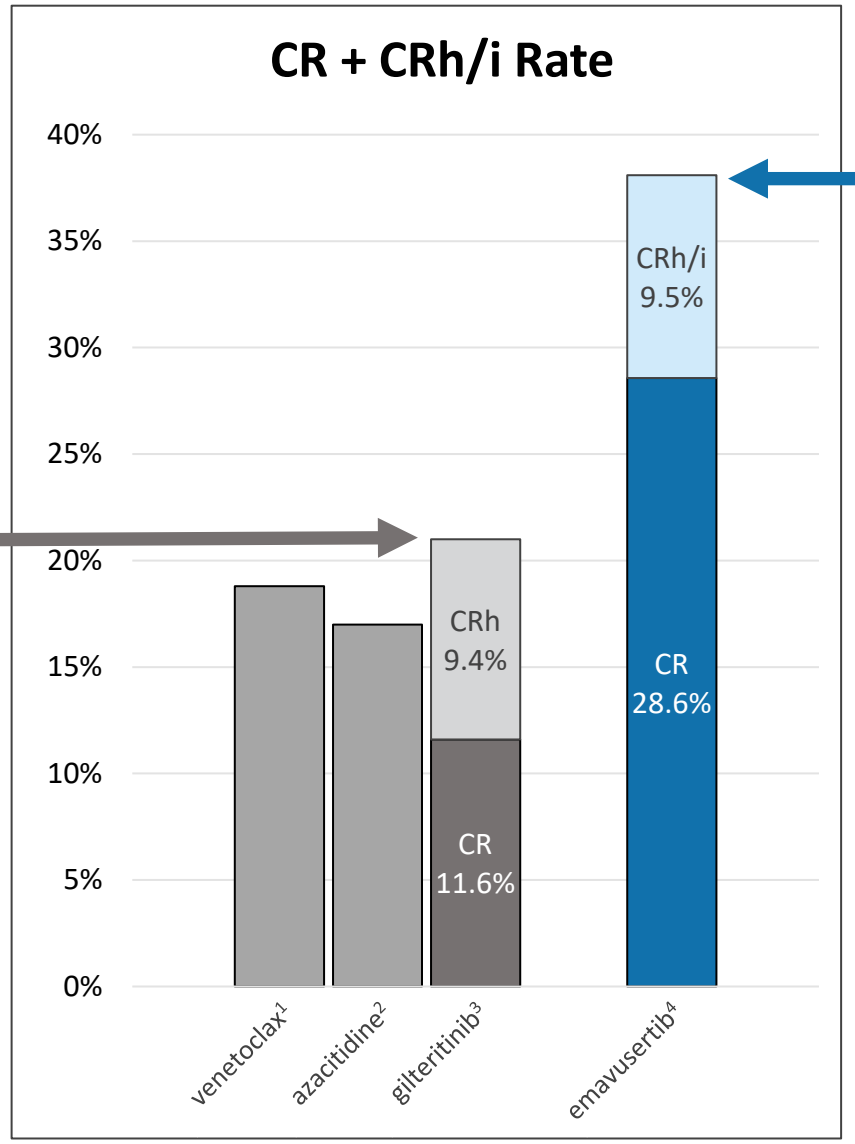
Clinical Data in FLT3m AML

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 FLT3m AML

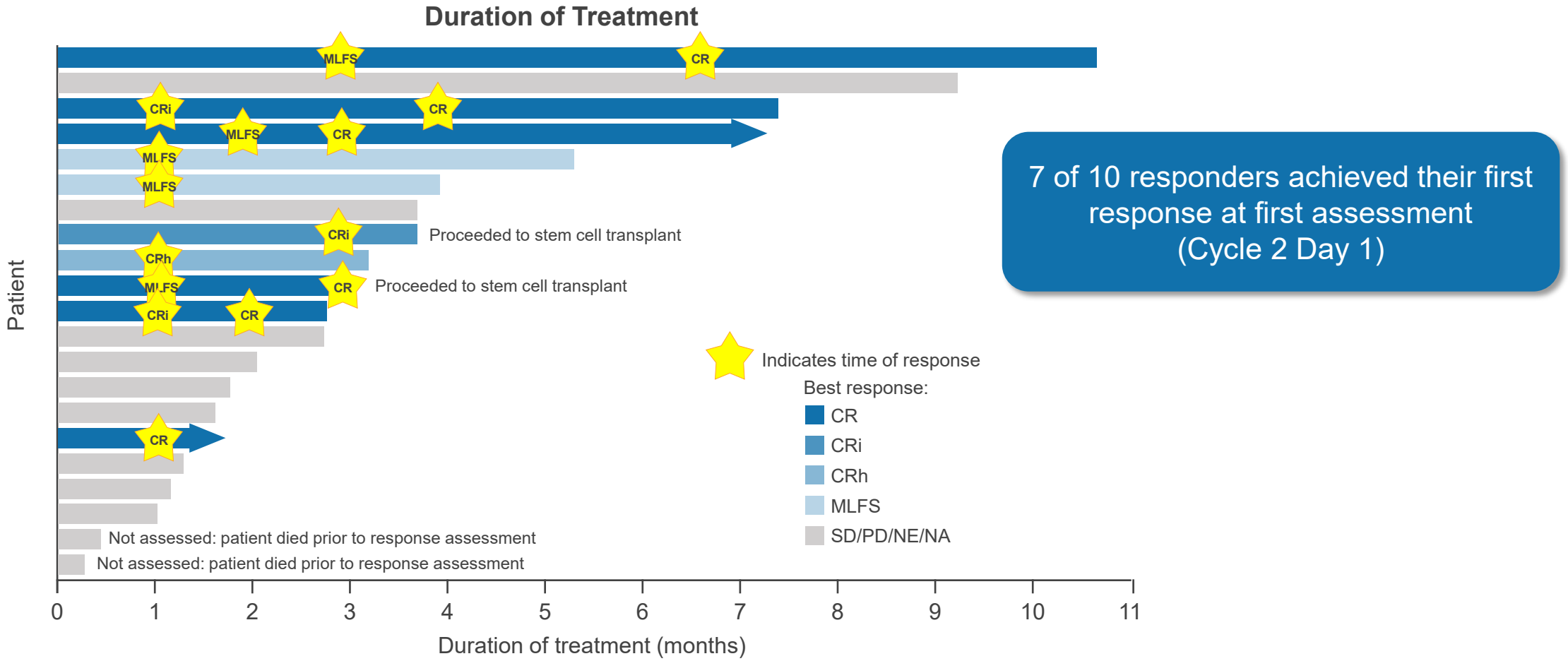
(as monotherapy)

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

Clinical Data in FLT3m AML



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

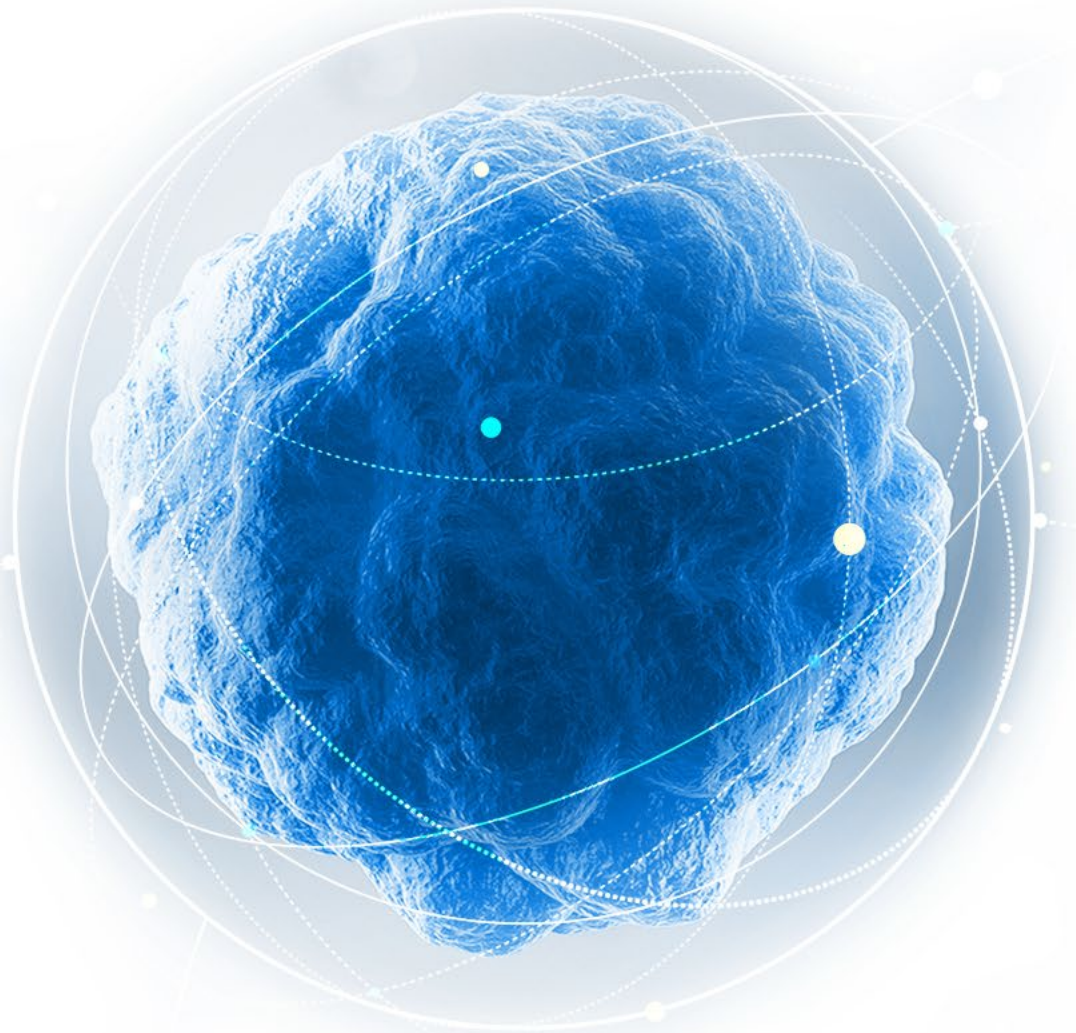
Presented at ASH 2024, data as of October 31, 2024
Includes 21 patients < 3 lines of prior therapy treated with emavusertib monotherapy at 300mg BID

Summary in AML

TakeAim Leukemia

- Emavusertib, as monotherapy, has the potential to be the best-in-class therapy in FLT3m AML
- With additional funding, next step is a registrational head-to-head study vs. gilteritinib

Solid Tumors



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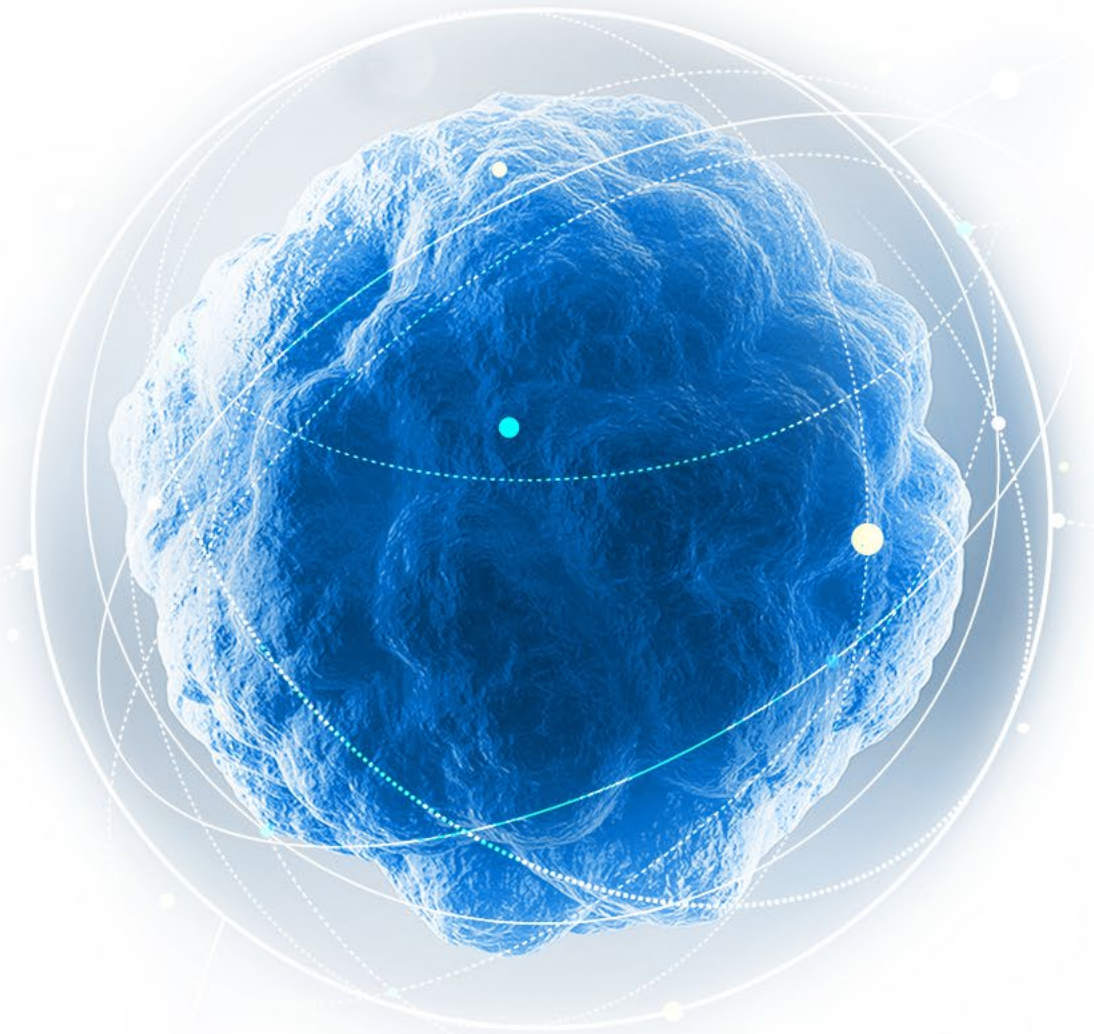
Ongoing studies (ISTs) of emavusertib in Solid Tumors

Tumor Type	Institution (Investigator)	Emavusertib Combination Partner
Pancreatic	CRADA Washington University (Grierson)	gemcitabine, nab-paclitaxel
Colorectal	CRADA Oklahoma University (Ulahannan)	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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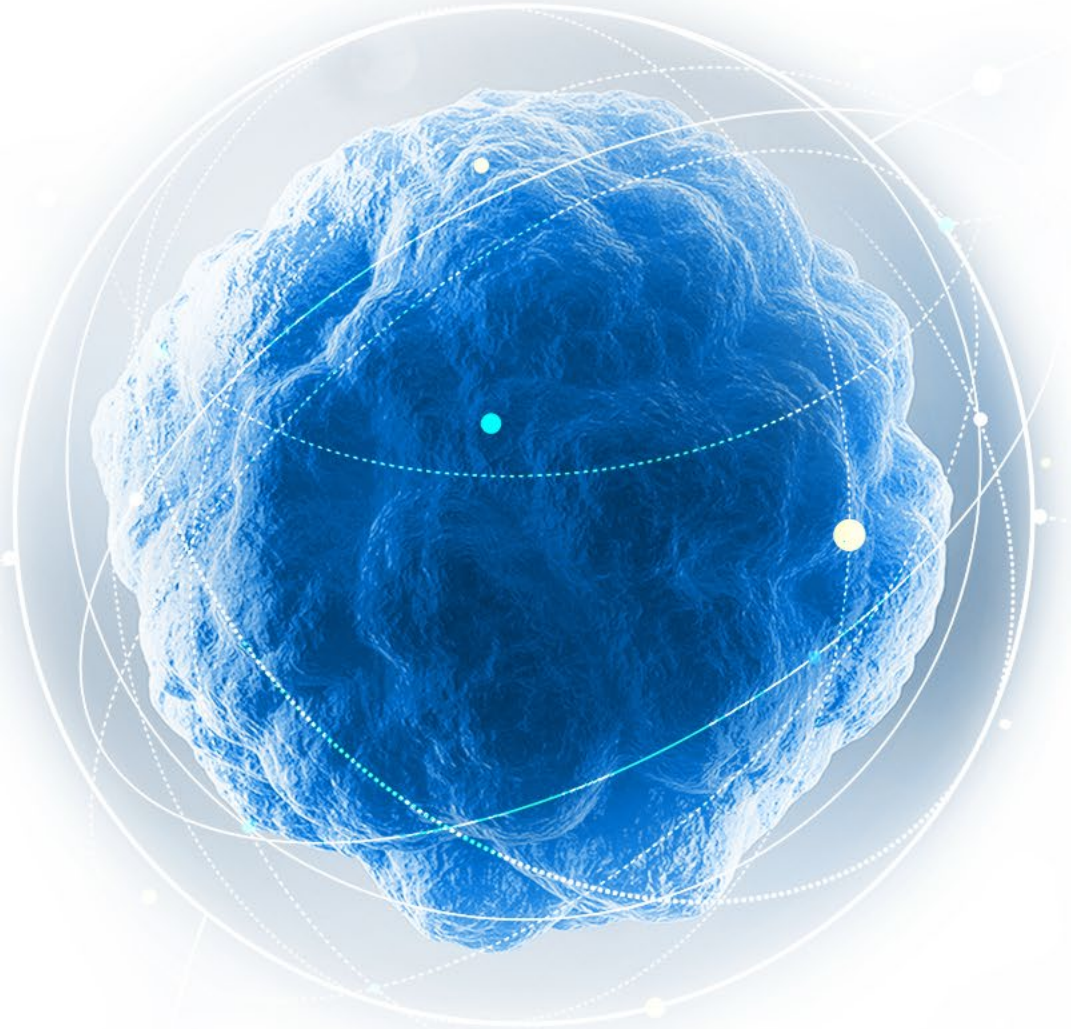
Financials and IP

	March 31, 2025
$20.3M	Cash
10.5M	Common Shares Outstanding
26.0M	Fully Diluted Shares Outstanding
2035	Composition of Matter IP on emavusertib (before potential extension)

End of Presentation



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