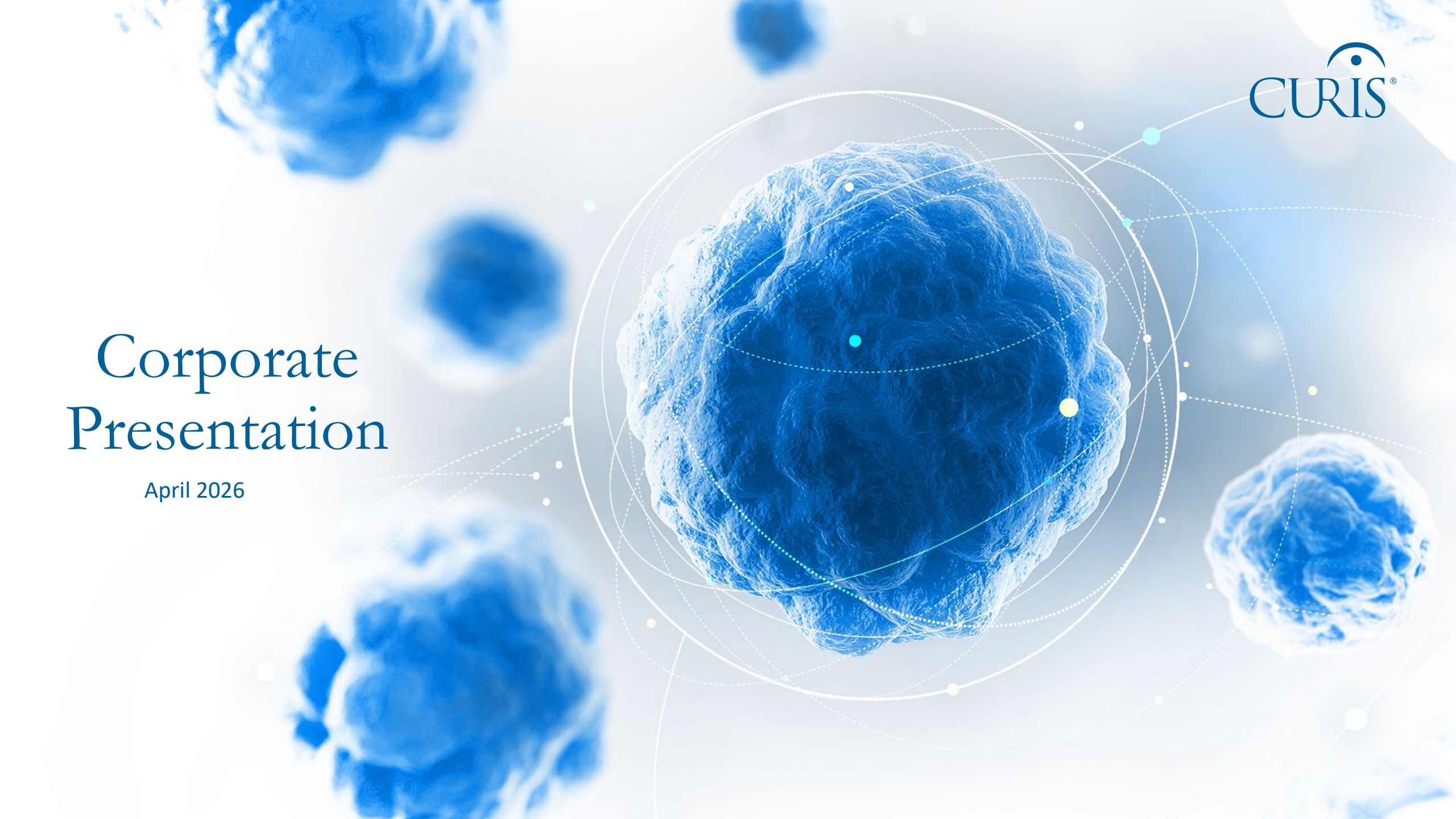


Corporate Presentation

April 2026

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, 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

Novel IRAK4 - FLT3 inhibitor

2 Proof-of Concept datasets demonstrate mechanism

(n=34)

PCNSL

BTKi-experienced *27% ORR (SOC is n/a)[1]*

BTK-naïve *63% ORR (SOC is 39%)[2]*

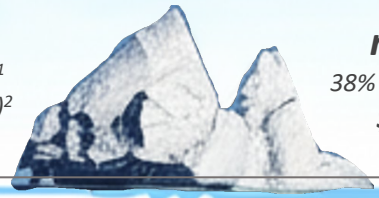
$0.5B market[3]

(n=21)

mFLT3 AML

38% CR/CRh (SOC is 21%)[5]

$0.6B market[4]



	PRE-CLINICAL	CLINICAL		
	Validation of Mechanism	Dose Optimization	Proof of Concept	Registrational
Lymphoma				
PCNSL (ema + BTKi)	*ORR in BTKi-experienced patients*			*ongoing*
CLL (ema + BTKi)	*convert PR to CR / MRD-*			*ongoing*
WM (ema + BTKi)	*convert PR to CR / MRD-*			
MCL (ema + BTKi)	*convert PR to CR / MRD-*			
Leukemia				
mFLT3 AML (monotherapy)	*CR in FLT3i-experienced patients*			
AML (ema-ven-aza)	*CR / MRD-*			
MDS (ema-aza)	*CR / MRD-*			

plus 5 additional ISTs in solid tumors

Additional opportunities in other NHL subtypes and AML

CLL
$9B market[4]

WM and other NHL
$3B market[4]

AML
$2.4B market[4]

Abbreviations: Primary Central Nervous System Lymphoma (PCNSL), Chronic Lymphocytic Leukemia (CLL), Waldenström's Macroglobulinemia (WM), Mantle Cell Lymphoma (MCL), Marginal Zone Lymphoma (MZL), Diffuse Large B-cell Lymphoma (DLBCL), FLT3 mutation (mFLT3), Acute Myeloid Leukemia (AML), B-Cell Receptor (BCR), Toll-Like Receptor (TLR), Non-Hodgkin lymphoma (NHL)

[1] There is no standard of care for PCNSL patients who progress on treatment with a BTKi.; [2] Soussain, Eur J Cancer 2019; [3] management estimate; [4] Citeline 2025 and company reported financial statements; [5] USPI, gilteritinib

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.

2025 Addition

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.

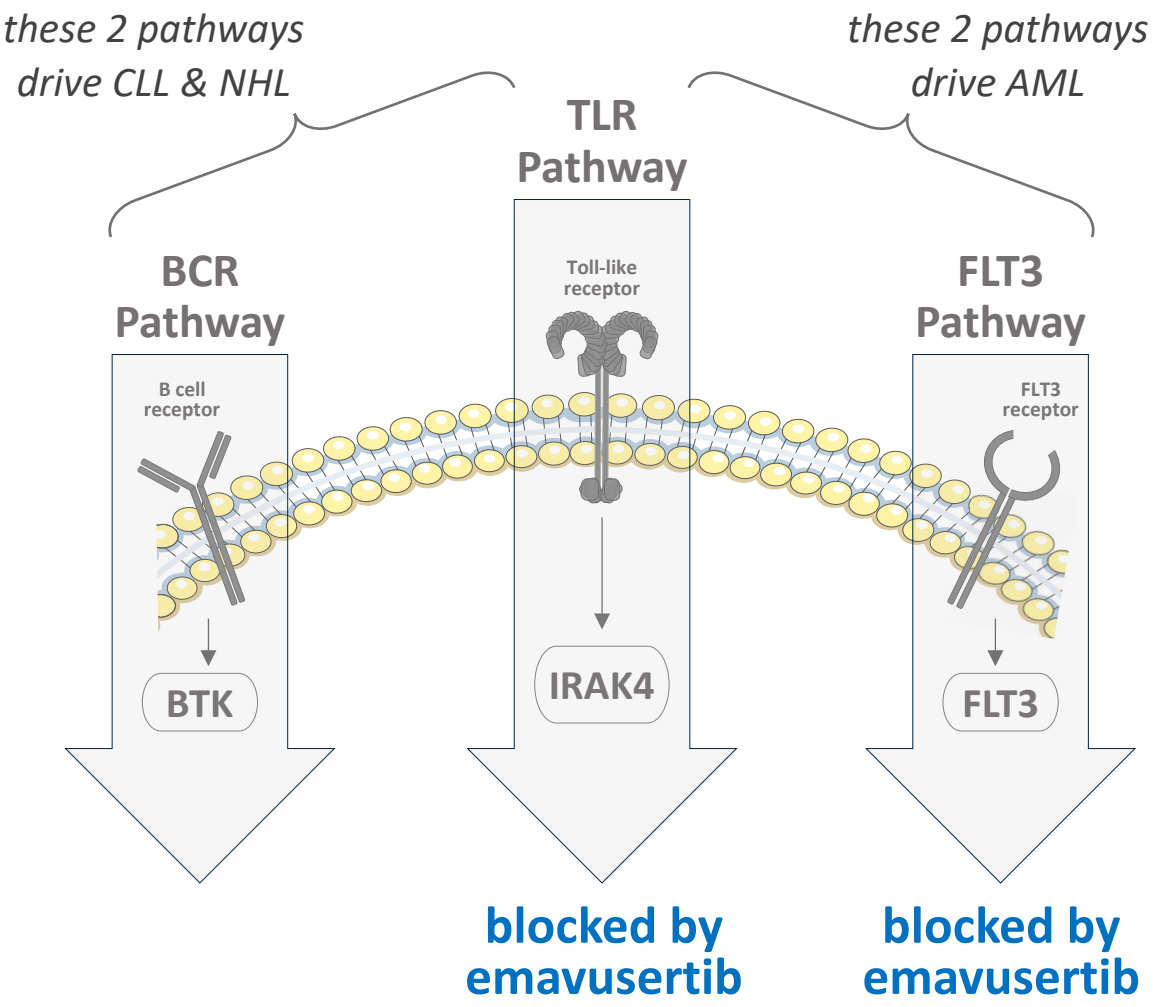
Emavusertib's mechanism targets key signaling pathways

In Lymphoma

- CLL and NHL are driven by NFkB dysregulation, which is in turn driven by two pathways: BCR and TLR[1]
- Current standard of care targets BTK (in the BCR Pathway); emavusertib targets IRAK4 (in the TLR Pathway), combining emavusertib with BTKi enables a dual-blockade of NFkB

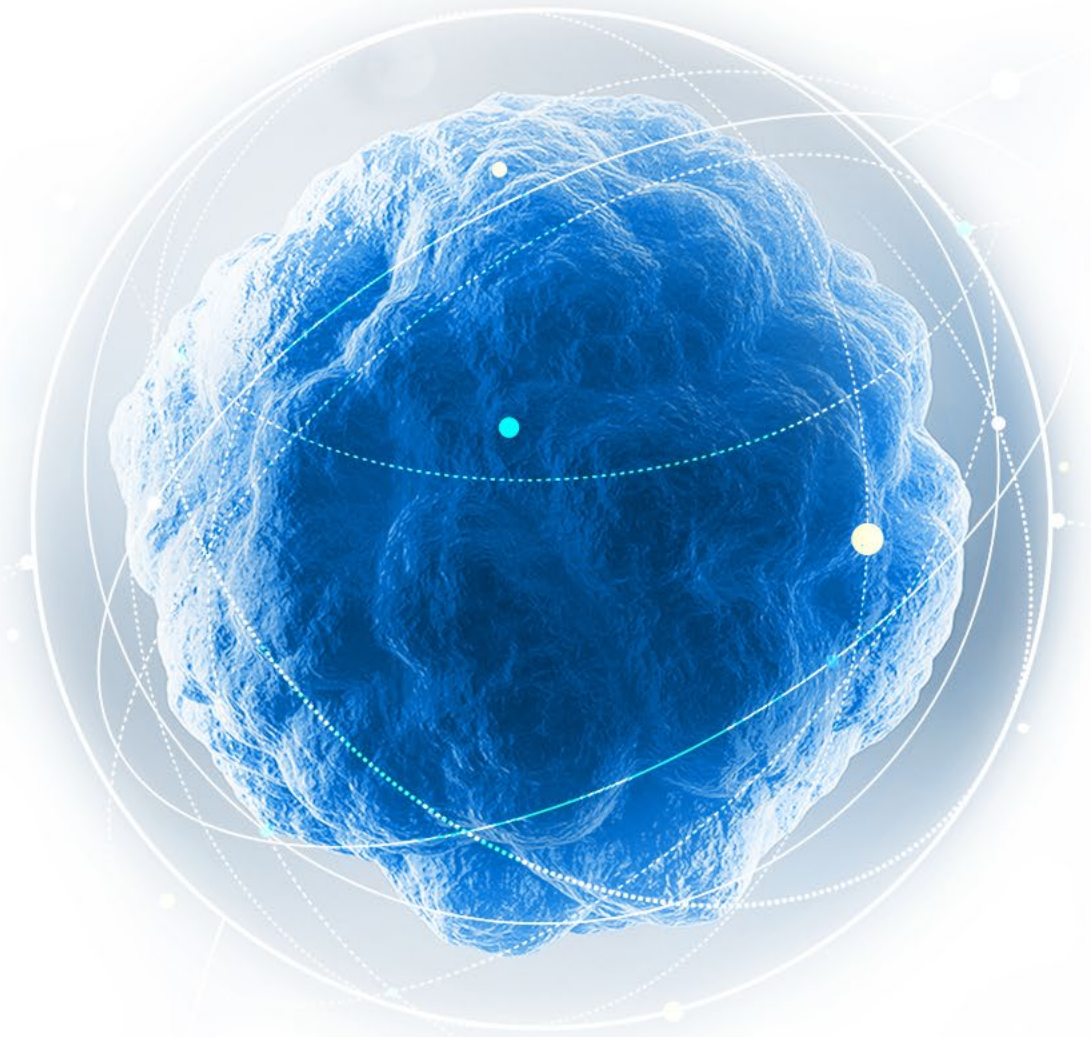
In Leukemia

- TLR signaling via IRAK4 has emerged as the leading driver of 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]



[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

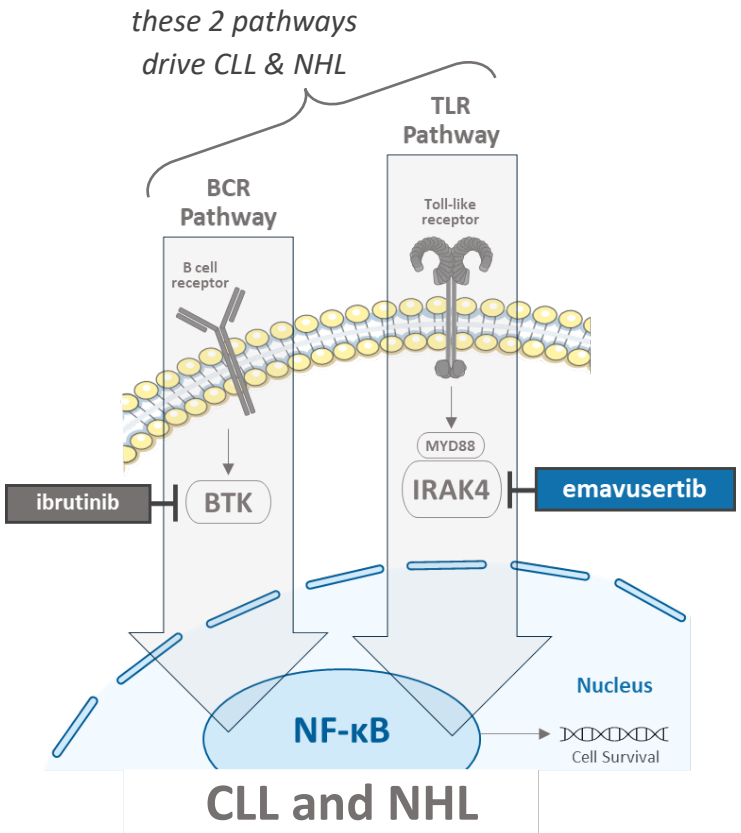
Emavusertib
in CLL and NHL



CURIS®

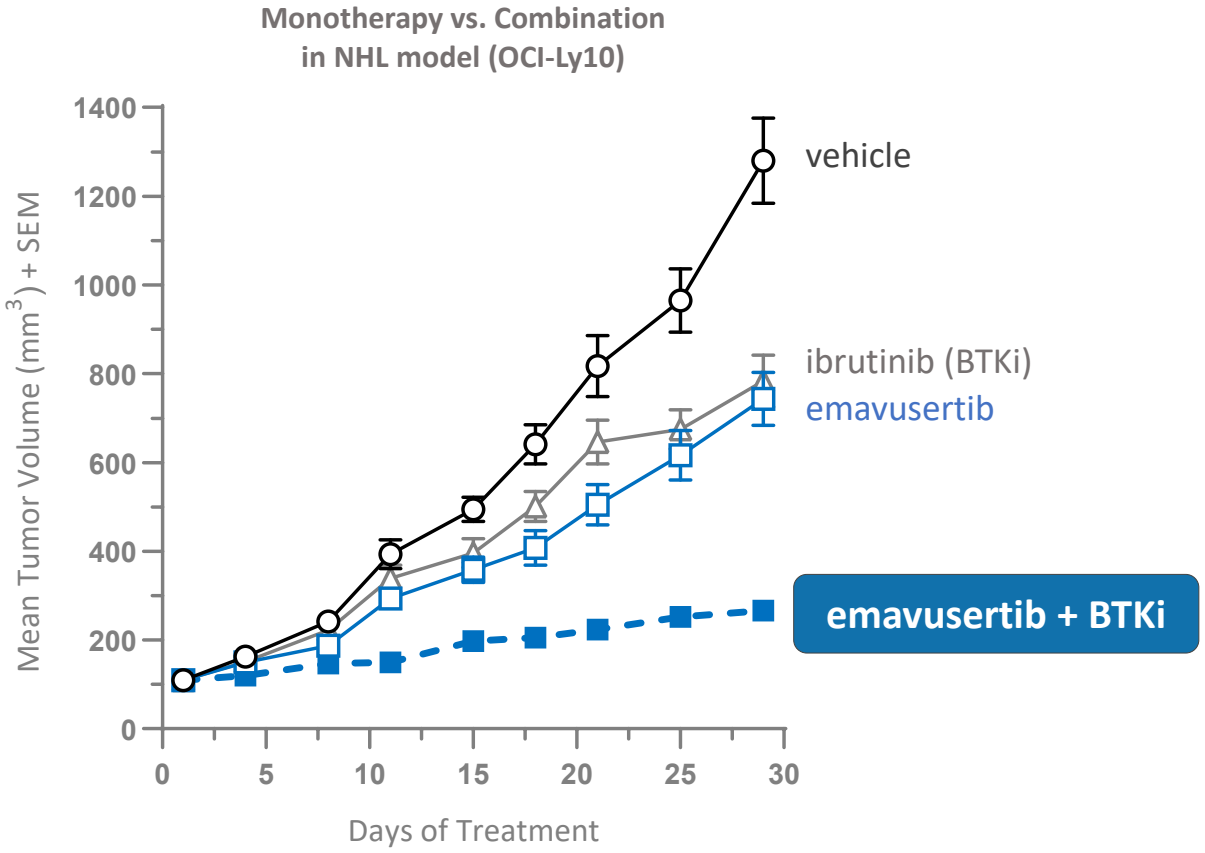
Adding emavusertib to BTKi provides deeper responses

Mechanism of Action



dual blockade of NF-kB enables stronger downregulation of NF-kB and deeper responses

Preclinical Evidence



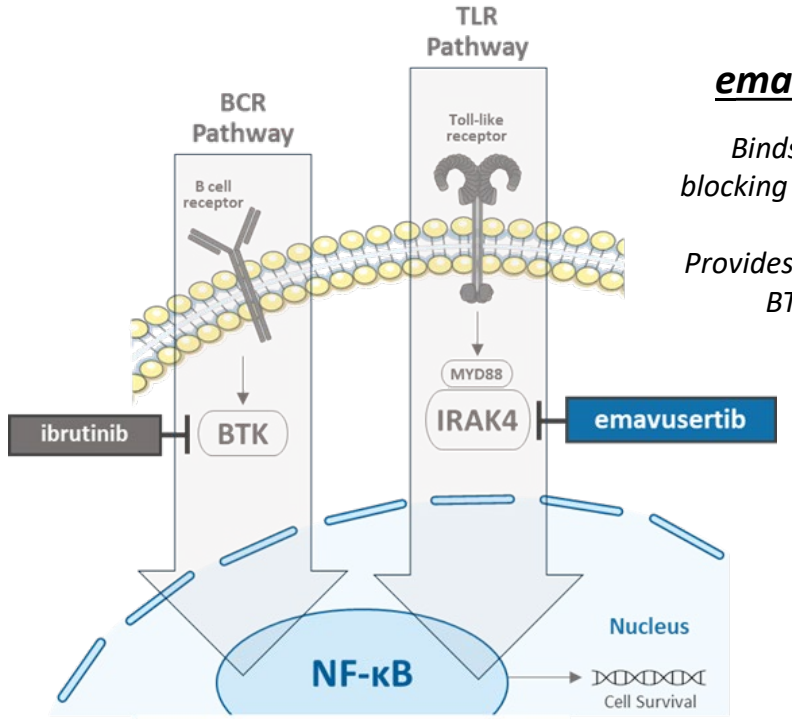
TakeAim Lymphoma Clinical Outcomes, ASH 2023 Poster

The goal in CLL and NHL is fixed duration, oral therapy

Emavusertib offers potential to achieve the "one and done" fixed duration benefit of CAR-T, but with an all oral therapy

2024 Market Leader
$10.8B Revenue

Current Therapies	Unmet Need / Limitation[1]
BTKi	• *Bleeding, bruising, headaches, fatigue, cardiac events* • *Chronic dosing risks development of mutations/resistance* • *Lack of complete remission*
BCL2 - αCD20	• *On-target tox: TLS, IRR, neutropenia, thrombocytopenia* • *Cannot re-treat after progression on BCL2 per label*
CAR-T	• *Requires specialty center (long vein-to-vein time)* • *Requires long-term immunosuppression* • *On-target tox: CRS, neurotoxicity, cytopenias*
Bispecifics	• *On-target tox: CRS, neurotoxicity*
NHL only ADCs	• *Nonspecific linker cleavage leads to off-target toxicities leading to myelosuppression, neuropathy, and eye tox*
Chemotherapy	• *Chemotoxicity*



emavusertib + BTKi

Binds to IRAK4 and BTK, blocking BCR and TLR pathways

Provides deeper responses than BTKi monotherapy

Abbreviations: Tumor Lysis Syndrome (TLS), Infusion-Related Reaction (IRR), anti-CD20 (α-CD20), Cytokine Release Syndrome (CRS), Antibody-Drug Conjugate (ADC)

[1] USPIs for ibrutinib, acalabrutinib, zanubrutinib, pirtobrutinib, venetoclax, and axicabtagene ciloleucel

Clinical Study Design in CLL and NHL

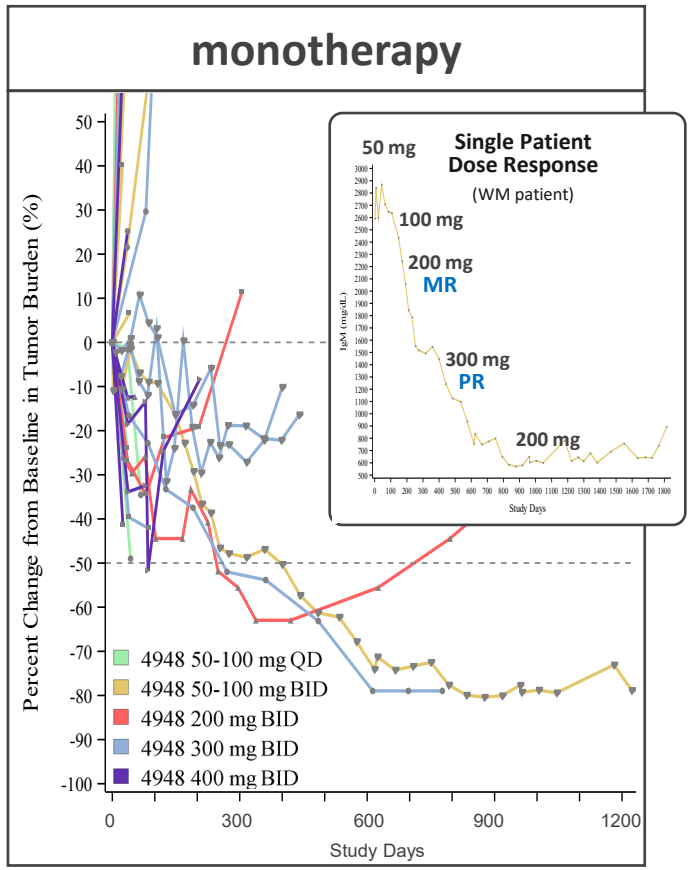
Three-part design

- **Part A: Dose Escalation**
  - 50 mg QD ⟶ 400 mg BID
  - monotherapy and combination
  - multiple NHL subtypes, including PCNSL, CLL, WM, and MCL
- **Part B: Study to support filing for Accelerated Approval**
  - PCNSL only; BTKi-experienced patients
  - single-arm study that adds emavusertib to a patient's BTKi regimen directly after they progress on BTKi monotherapy
  - benchmark: none[2]
- **Part C: Study to support filing for Full Approval**
  - PCNSL only; BTKi-naïve patients
  - randomized study comparing BTKi monotherapy vs. BTKi + emavusertib
  - benchmark: 39%[1] for ibrutinib monotherapy

[1] Soussain, Eur J Cancer 2019 ; [2] there is no standard of care for PCNSL patients who progress on treatment with a BTK

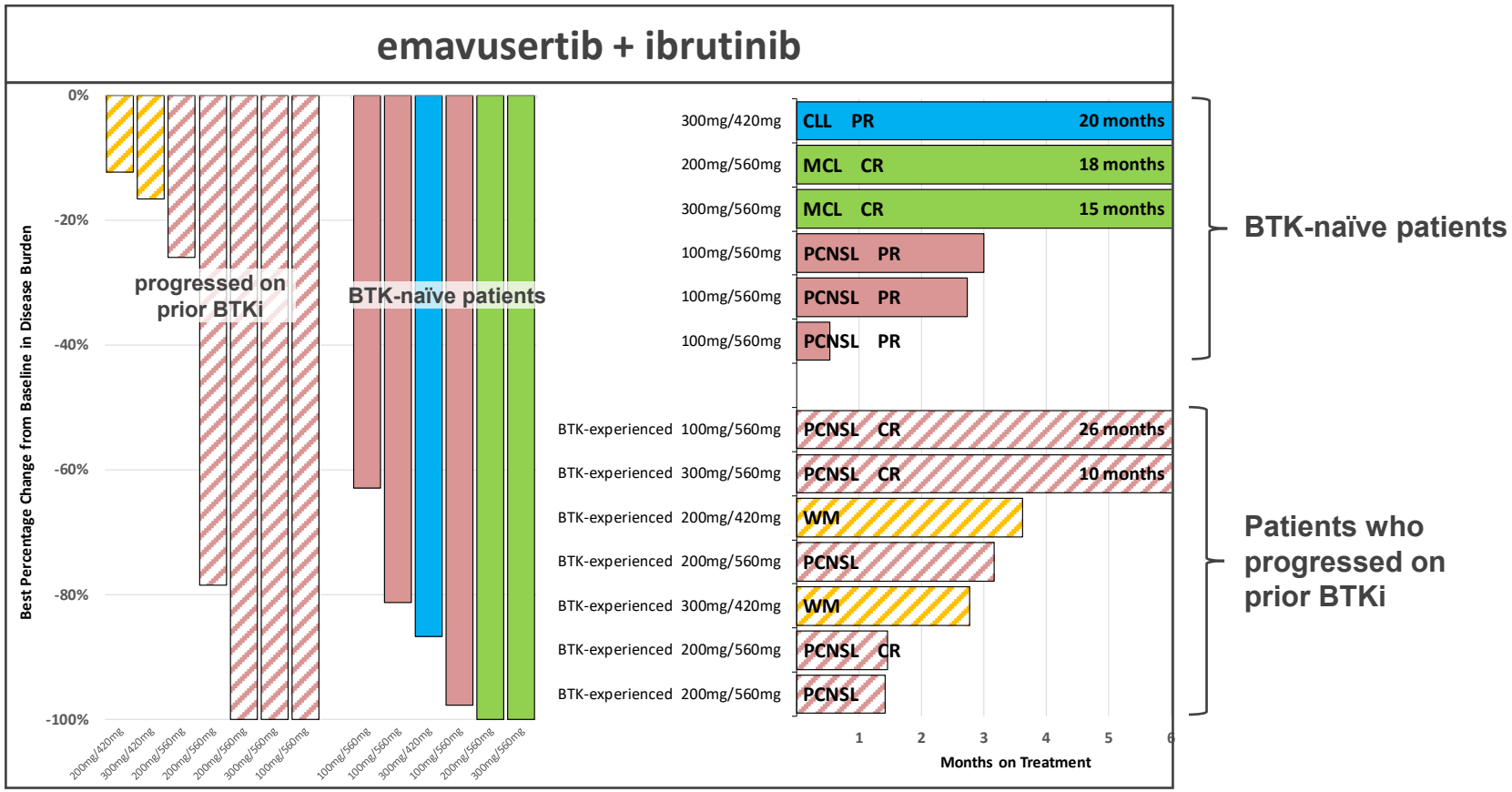
Part A: early evidence of monotherapy and combination activity in dose escalation study with multiple NHL subtypes

dose escalation (50-400mg)



IgM values were used as the measure for tumor burden for WM and LPL patients; Sum of product of diameters of target lesions used for other lymphoma types

dose escalation in CLL, WM, MCL, PCNSL (100-300mg)

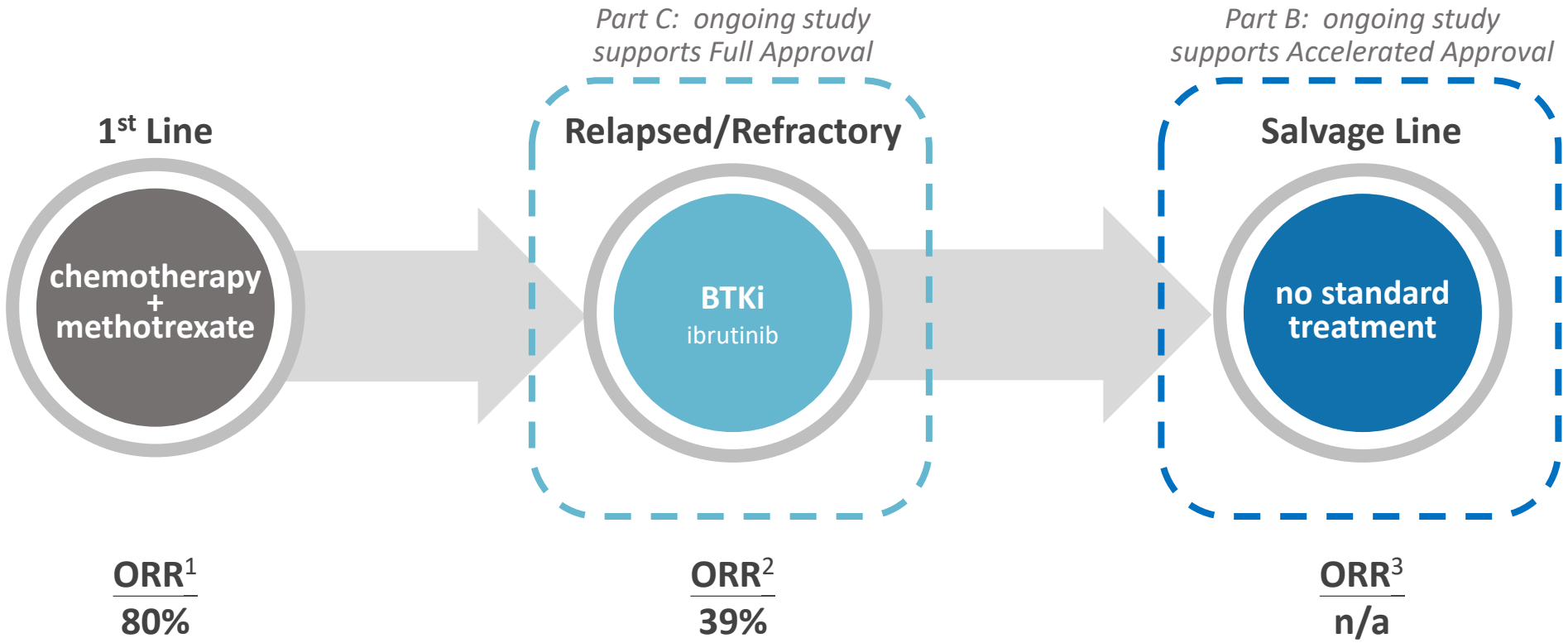


Data above include all patients treated with emavusertib + ibrutinib combination who completed at least 1 cycle and had calculable postbaseline tumor, using the sum of products of diameters of target lesions for MCL and CLL, and IgM levels for WM.

Cutoff date: May 1, 2025

Abbreviations: Minor Response (MR), Partial Response (PR), Complete Remission (CR)

Parts B & C: registrational study design in PCNSL



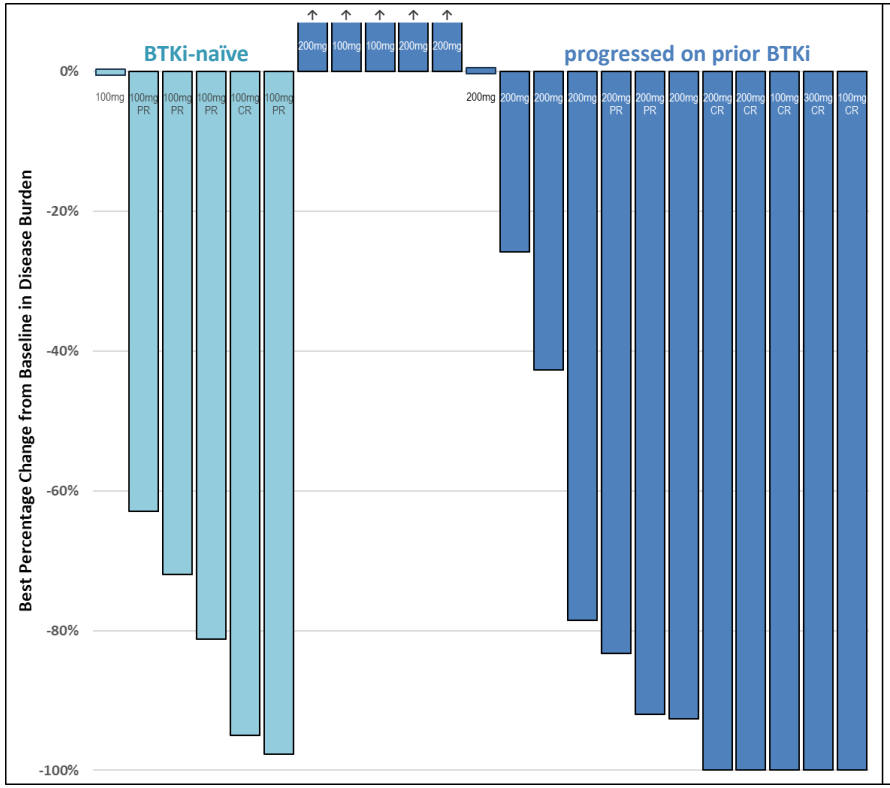
Goal:
Demonstrate that BTKi + emavusertib can achieve deeper responses than BTKi monotherapy

Goal:
Demonstrate that adding emavusertib to a patient's BTKi regimen directly after they progress on BTKi monotherapy can reverse tumor growth and enable patients to achieve objective responses

[1] Shi, Medicine, 2025 ; [2] Soussain, Eur J Cancer 2019; [3] there is no standard of care for PCNSL patients who progress on treatment with a BTK

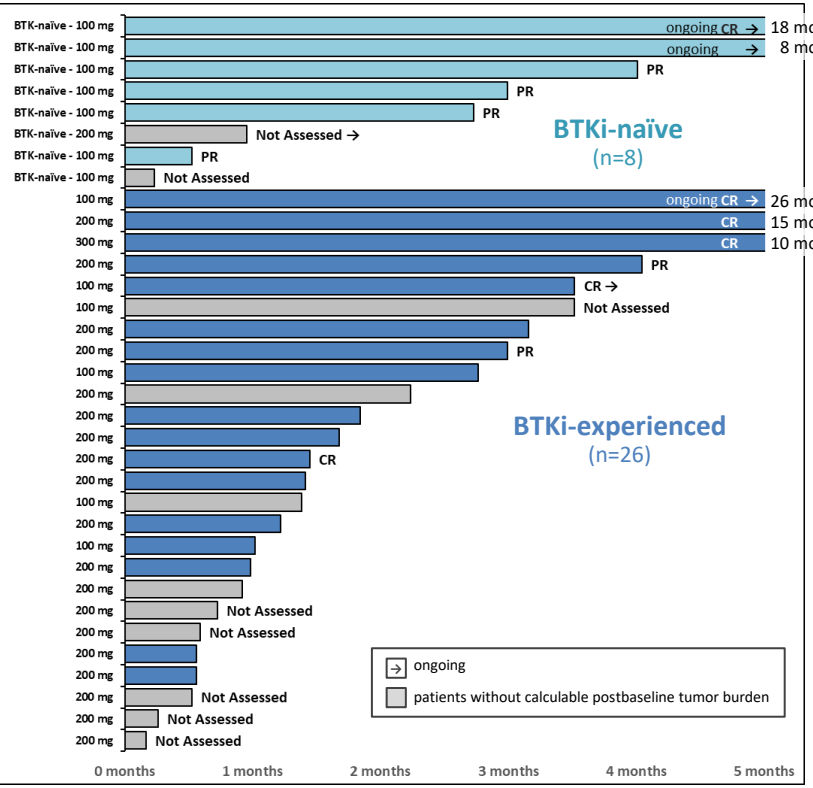
Ongoing registrational study in PCNSL demonstrates that adding emavusertib to ibrutinib provides deep and durable responses

Reduction in Tumor Burden



Data include all PCNSL patients treated with emavusertib in combination with BTKi in Parts A, B, and C with calculable postbaseline tumor burden at cutoff date

Time on Treatment



Data include all PCNSL patients treated with emavusertib in combination with BTKi in Parts A, B, and C as of the cutoff date

BTKi-naïve
63% ORR (5 of 8)

Benchmark: 39% [1]

BTKi-experienced
27% ORR (7 of 26)

Benchmark: n/a [2]

Cutoff date: May 1, 2025
Abbreviations: Intent to Treat (ITT) , overall response rate (ORR)

[1] Soussain, Eur J Cancer 2019 ; [2] there is no standard of care for PCNSL patients who progress on treatment with a BTK

Well tolerated safety profile with duration > 1-2 years

emavusertib monotherapy

Grade 3+ TRAEs Reported in > 1 Patient n (%)	50-100 mg QD (N=9)	50-100 mg BID (N=8)	200 mg BID (N=3)	300 mg BID (N=6)	400 mg BID (N=8)	Total (N=34)
# patients w/ Gr3+ TRAEs	4 (44)	2 (25)	1 (33)	4 (67)	4 (50)	15 (44)
Neutrophil count decr	2 (22)	0	1 (33)	2 (33)	0	5 (15)
Blood CPK incr	0	0	0	3 (50)	1 (13)	4 (12)
Hypophosphataemia	0	1 (13)	0	1 (17)	2 (25)	4 (12)
Amylase incr	1 (11)	1 (13)	0	0	1 (13)	3 (9)
Anaemia	0	1 (13)	0	1 (17)	1 (13)	3 (9)
Neutropenia	1 (11)	0	0	1 (17)	1 (13)	3 (9)
Lipase incr	1 (11)	1 (13)	0	0	0	2 (6)
Rhabdomyolysis	0	0	0	0	2 (25)	2 (6)
Thrombocytopenia	0	0	0	1 (17)	1 (13)	2 (6)

Safety data for patients treated in Part A

emavusertib + ibrutinib

Grade 3+ TRAEs Reported in > 1 Patient n (%)	100 mg BID (n=13)	200 mg BID (n=27)	300 mg BID (n=7)	Total (n=47)
# patients w/ Gr3+ TRAEs	5 (38)	11 (41)	6 (86)	22 (47)
Neutropenia	4 (31)	1 (4)	0	5 (11)
Lipase incr	2 (15)	1 (4)	0	3 (6)
Platelet count decr	0	2 (7)	1 (14)	3 (6)
ALT incr	0	1 (4)	1 (14)	2 (4)
Amylase incr	2 (15)	0	0	2 (4)
AST incr	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)

- Well tolerated
- Durable safety profile > 1-2 years
- Emavusertib crosses the BBB
- No dose-limiting myelosuppression or CNS toxicities
- 2 DLTs in monotherapy at 400 mg BID (CPK increase and rhabdomyolysis)
- 2 DLTs in combination at 300 mg BID (syncope and stomatitis)

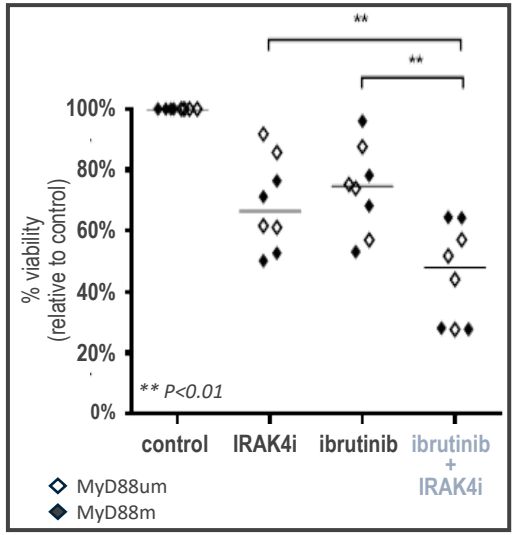
Safety data as of May 1, 2025

Abbreviations: Treatment Related Adverse Event (TRAE), Blood Brain Barrier (BBB), Central Nervous System (CNS) , twice daily (BID) , once daily (QD); creatine phosphokinase (CPK); Alanine aminotransferase (ALT); Aspartate aminotransferase (AST)

Preclinical evidence for IRAK4 in CLL

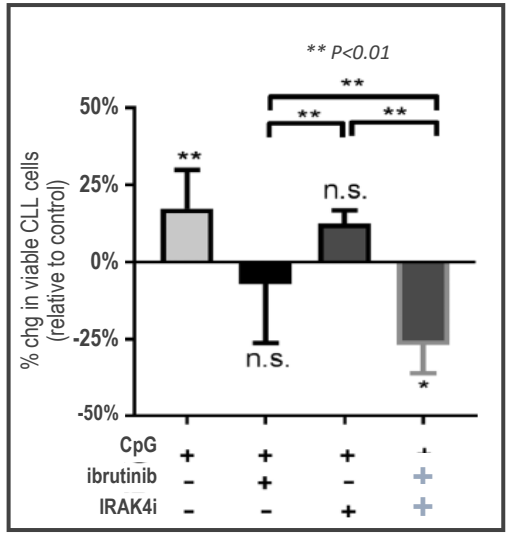
- Inhibition of IRAK4 blocks survival and proliferation of CLL cells[1]
- Dual inhibition of IRAK4 and BTK is significantly more potent in patient CLL cells than either drug alone[2]
- Differences in TLR function represent an independent and earlier event than acquired mutations in CLL[3] and TLR activation generates de novo resistance to ibrutinib in CLL and MCL[4]
- IRAK4 inhibition decreases the viability and proliferation in patient-derived CLL cells, including those without MYD88 mutations, suggesting IRAK4 promotes tumorigenesis independent of MYD88 mutation[5]

IRAK4i + BTKi reduces patient CLL cell viability, regardless of MYD88 status



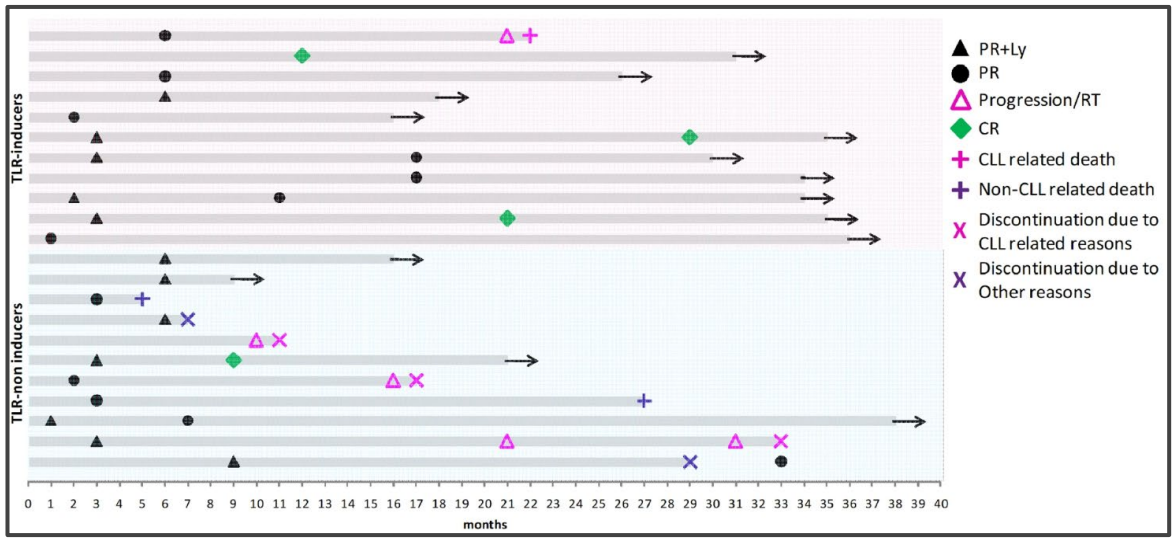
Giménez, Leukemia. 2020

Dual targeting with IRAK4i + BTKi is superior to single agents in CLL



Dadashian, Cancer Res. 2019

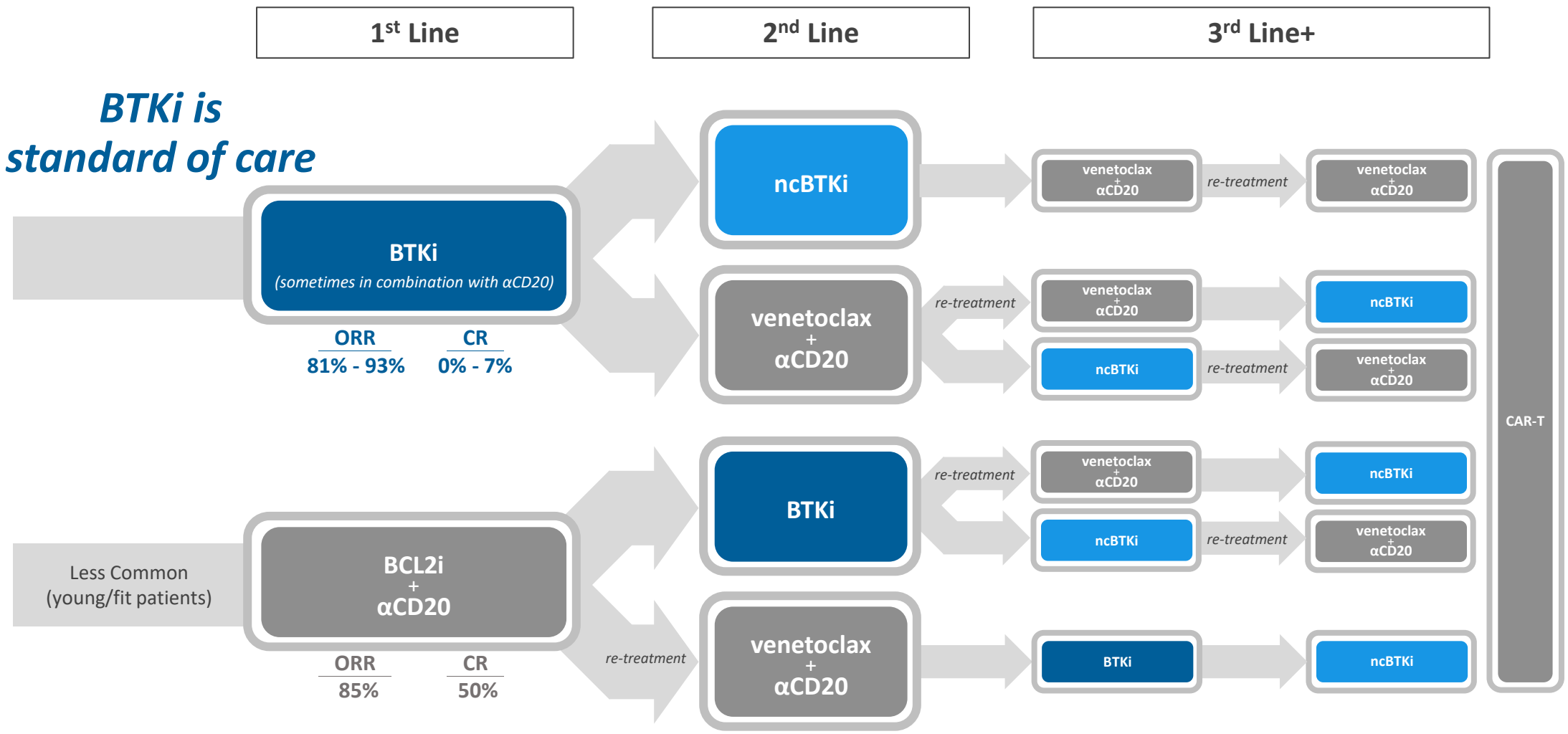
Ibrutinib-treated CLL survival signaling is sustained via TLR pathways



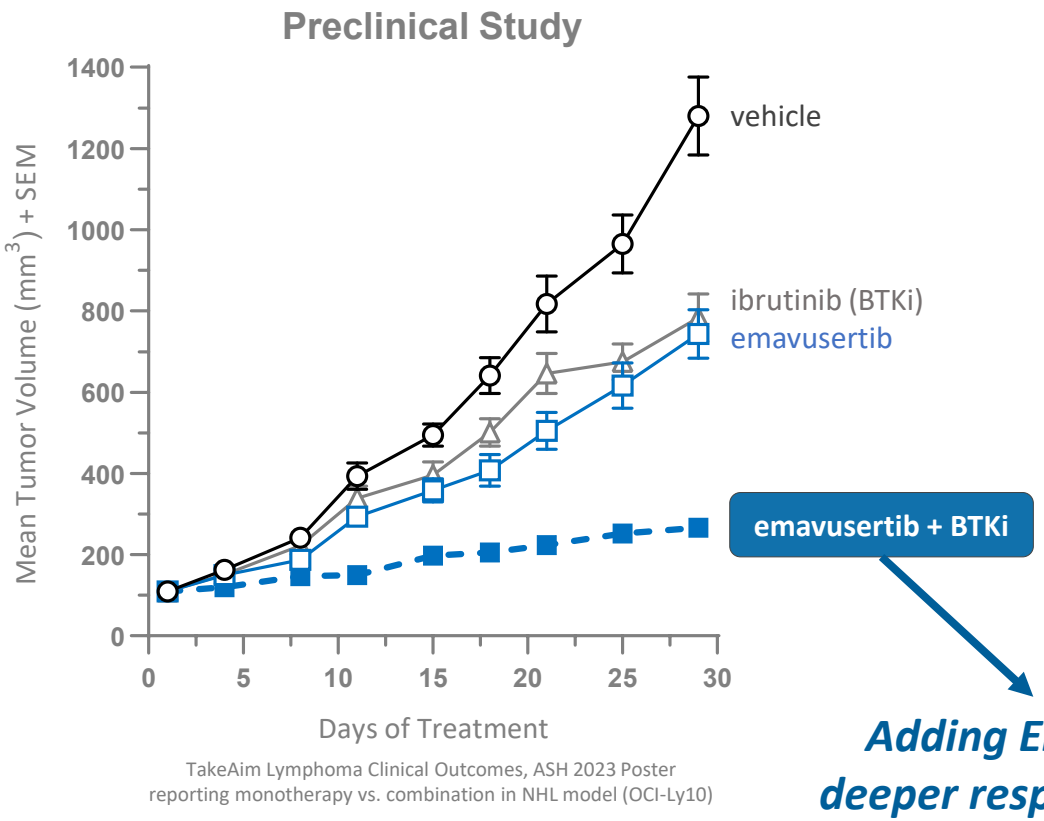
Gounari, Leukemia. 2019

[1] Giménez, Leukemia. 2020; [2] Dadashian, Ca Res. 2019 ; [3] Gounari, Leukemia. 2019; [4] Jayappa, Leukemia. 2017 ; [5] Pirrondo, Front Immunol. 2023

Current Treatment Paradigm in CLL



Ongoing Ph2 study in CLL



TakeAim Lymphoma Clinical Outcomes, ASH 2023 Poster reporting monotherapy vs. combination in NHL model (OCI-Ly10)

Adding Ema to BTKi provided deeper responses than BTKi alone

Goal:

Replicate Preclinical Findings in a Ph2 Study

CLL Clinical Study Design

Study Design:	single-arm
Dosing & Admin:	200 mg BID, orally in combination with BTKi
Primary Endpoint:	uMRD
Secondary Endpoints:	CR, DOR, PFS
Study Population:	Patients on a BTKi who have achieved PR but remain MRD^+
Study Size:	n=40

Will adding Ema to BTKi enable deeper PRs or CR/MRD^-?

MRD emerging as new primary endpoint in CLL

Regulatory Support for MRD

WEEK IN REVIEW:
Dr. Brian G.M. Durie

A Historic Turning Point: ODAC Unanimously Votes in Favor of MRD Testing as an Early Endpoint in Myeloma Clinical Trials to Support Accelerated Approvals of New Treatments

Post date: April 18, 2024

WEEK IN REVIEW

ELSEVIER

Journal of Cancer Policy

Available online 1 May 2025, 100588

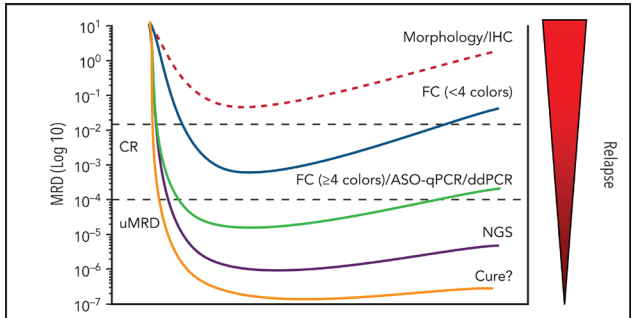
In Press, Corrected Proof What's this?

Minimal residual disease negative: A novel endpoint for accelerated approval; What providers should know

Sruthi Ranganatha[a], Vinay Prasad[b]

Approved Assay for MRD

MRD in CLL: some answers, "Assay is key"



IHC: Inmunohistochemistry; FC: Flow cytometry; CR: Complete response; PCR: Polymerase chain reaction; ASO-qPCR: allele-specific oligonucleotide quantitative PCR; ddPCR: droplet digital PCR; uMRD: undetectable measurable residual disease; NGS: Next generation sequencing.

clonoSEQ®

THE FIRST & ONLY FDA-CLEARED ASSAY FOR MRD DETECTION

In bone marrow from patients with multiple myeloma and B-cell acute lymphoblastic leukemia (ALL) and blood or bone marrow from patients with chronic lymphocytic leukemia (CLL)

MRD: A powerful way to assess response and predict patient outcomes

Measurable (or minimal) residual disease (MRD) refers to the small number of cancer cells that may remain in a patient's body during and after treatment. Clinical practice guidelines recognize that MRD status is a reliable indicator of clinical outcome and response to therapy in myeloma, ALL and CLL patients.[1,2,3,4]

The clonoSEQ® Assay is an MRD assessment tool powered by next-generation sequencing (NGS) technology and differentiated from other NGS assays by groundbreaking advances in chemistry and proprietary bioinformatics.[5,6]

Clinicians who leverage the latest advances in personalized medicine use clonoSEQ to:[5]

- ✓ Predict long-term outcomes
- ✓ Assess treatment response
- ✓ Monitor disease burden
- ✓ Detect potential relapse

Clinical Trials using MRD in CLL

- **Venetoclax-Obinutuzumab +/- Acalabrutinib in R/R CLL Phase3** NCT04560322
- **Mosunetuzumab for CLL MRD Clearance Phase 1/2** NCT07052695
- **MRD Guided Sonrotoclax and Zanubrutinib in Newly Diagnosed CLL/SLL Phase3** NCT06367374
- **Pirtobrutinib (LOXO-305) Consolidation for MRD Eradication in Patients With CLL/SLL Treated With Venetoclax Phase 1/2** NCT05317936
- **Ibrutinib in Combination With Obinutuzumab Versus Chlorambucil in Combination With Obinutuzumab in Patients With Treatment naïve CLL/SLL Phase 3** NCT02264574

Summary in CLL and NHL

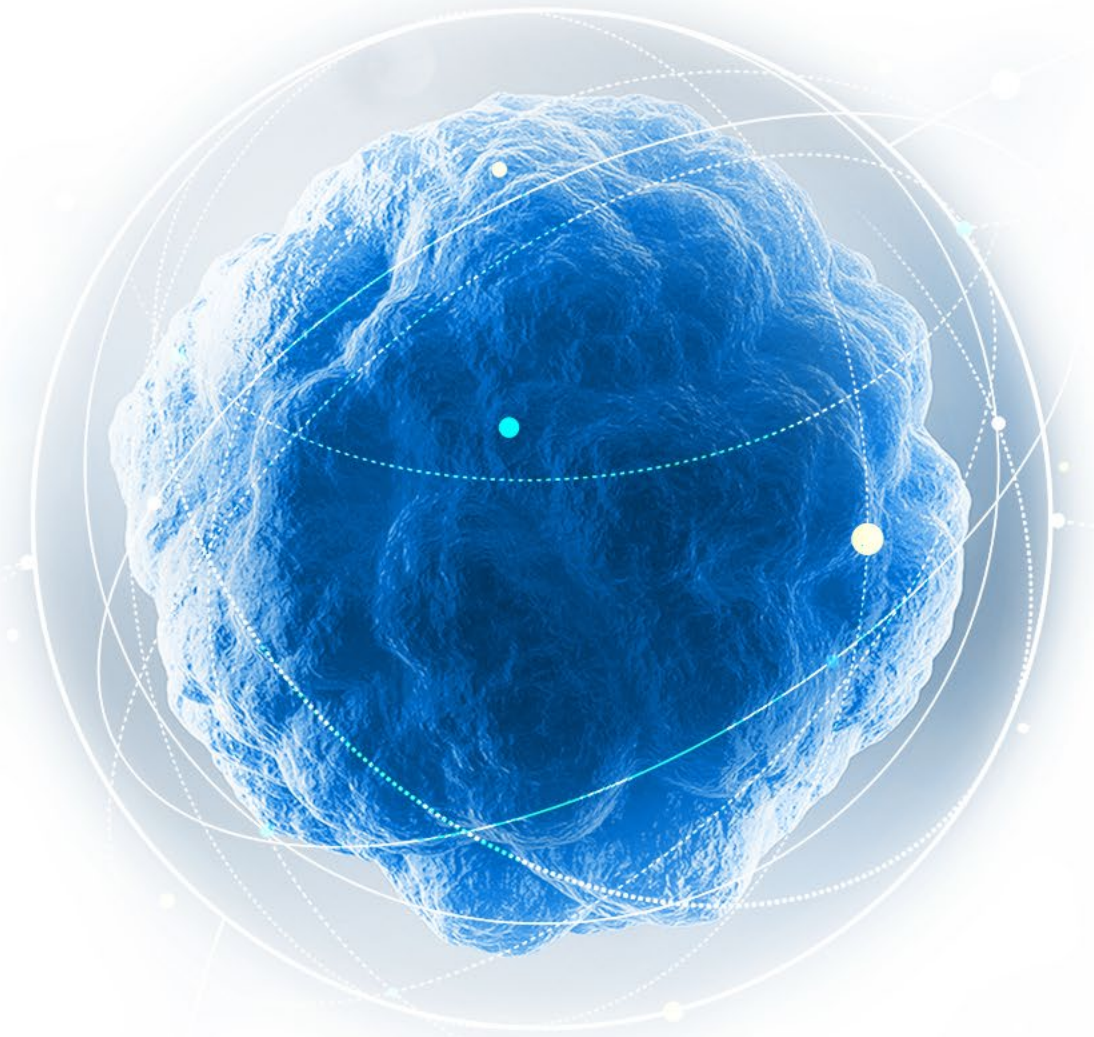
- Adding emavusertib to BTKi provides deep and durable responses in PCNSL

in patients who progressed on BTKi:	**in BTKi-naïve patients:**
27% ORR *vs. n/a*[1]	***63% ORR*** *vs. 39% for ibrutinib*[2]

- Initiating Phase 2 in larger subtypes, starting with CLL
  - Early data in CLL, WM, and MCL show consistent pattern of activity
  - Emavusertib offers potential to achieve the fixed duration benefit of CAR-T, but with an oral-oral therapy

[1] There is no standard of care for PCNSL patients who progress on treatment with a BTKi.; [2] Soussain, Eur J Cancer 2019

Emavusertib
in AML



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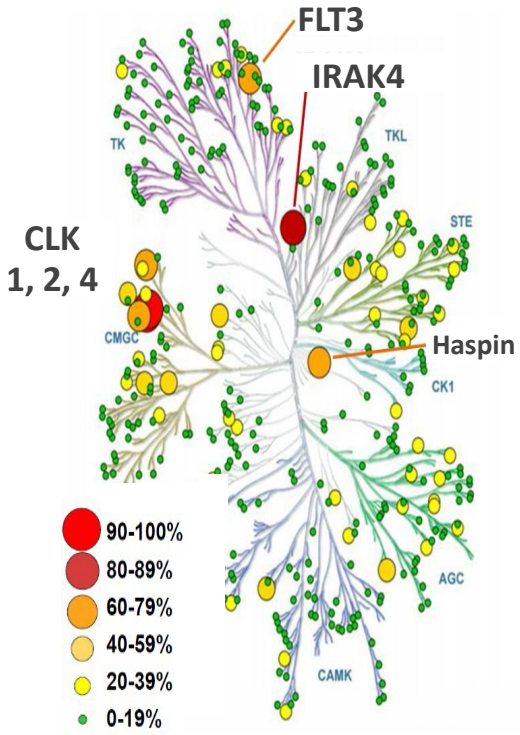
Emavusertib hits two important targets in AML

Emavusertib
Kinase Interaction Map



% Inhibition at 0.1 nM

illustration reproduced courtesy of
Cell Signaling Technology

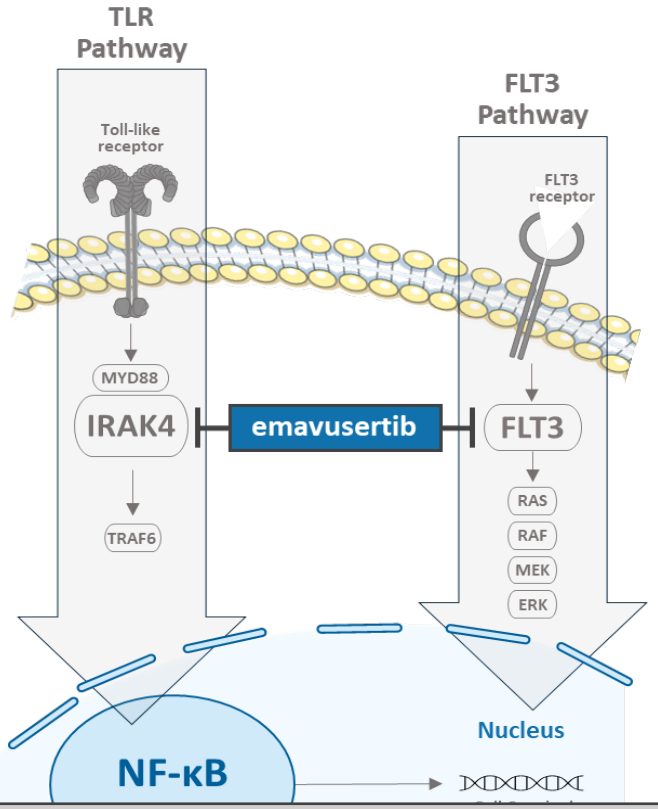
Emavusertib
Binding Affinity

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)

TLR and FLT3 pathways drive disease in AML

blocking both pathways provides deeper responses

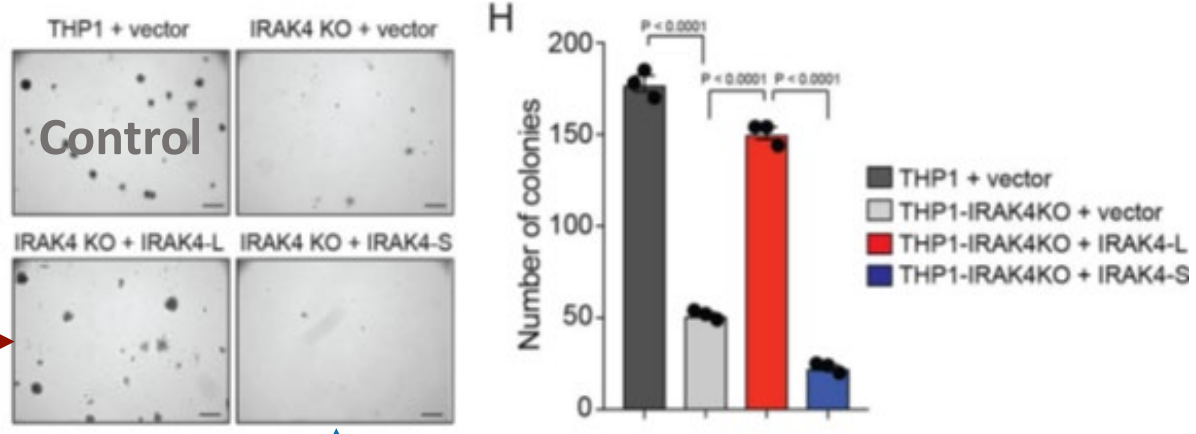


	Current Therapies	Unmet Need / Limitation
fit	(1st Line) Chemo + FLT3i	• *Low 5yr OS (22%), myelosuppression* • *FLT3i resistance (driven by IRAK4)*
unfit	(1st Line) HMA + Ven	
	(2nd Line) FLT3i	• *Low response rate (21% composite CR)* • *FLT3i resistance (driven by IRAK4)*
	HSCT	• *Patient must be in remission* • *Risk of rejection, GVHD*

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

IRAK4-L is oncogenic

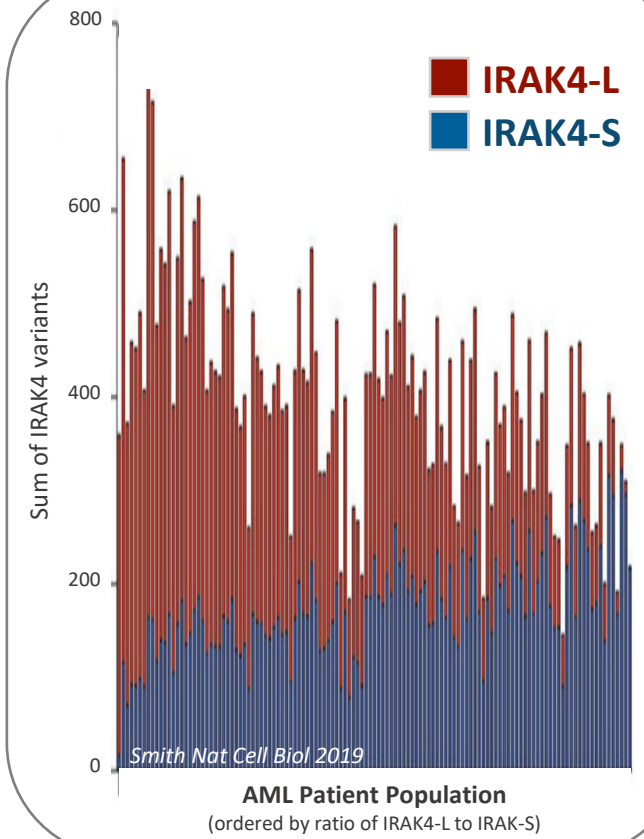
Knocking out IRAK4 stops leukemic activity



adding back IRAK4-L restarts activity

adding back IRAK4-S has no effect

IRAK4-L is expressed in nearly all AML patients

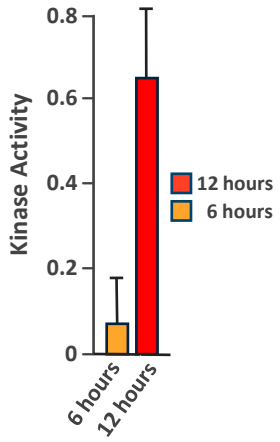


Smith et al. Nat Cell Biol 2019

Emavusertib's dual blockade of IRAK4 and FLT3 has the potential to outperform approved FLT3 inhibitors

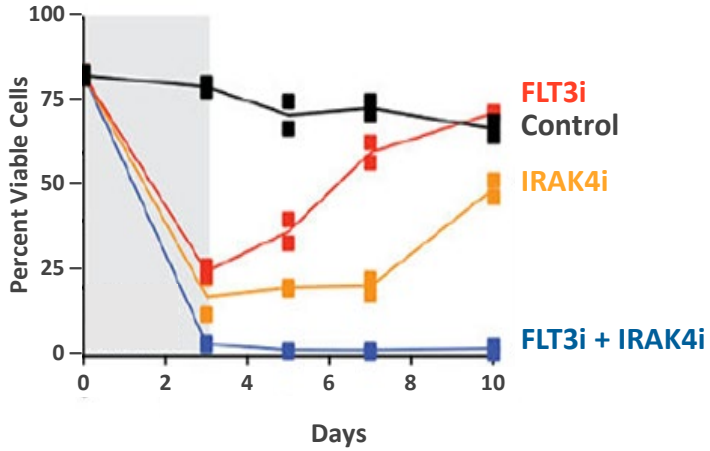
Because IRAK4 drives adaptive resistance to FLT3i

IRAK4 activity increased after treatment with FLT3i



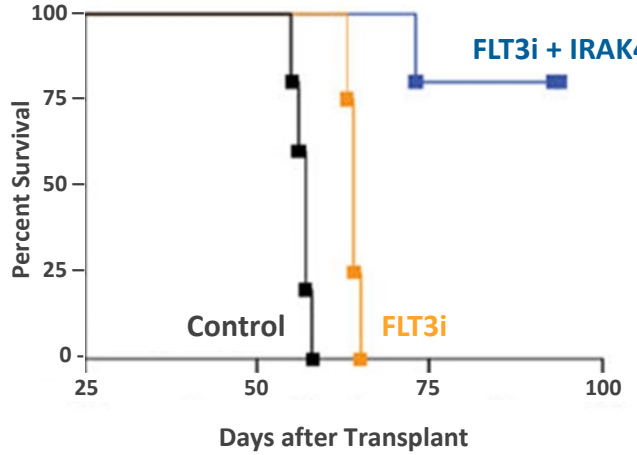
relative phospho-peptide intensity of IRAK4

IRAK4i + FLT3i were synergistically cytotoxic



Viability of MLL-AF9; FLT3-ITD cells treated for 3 days with DMSO (control), quizartinib (0.5 µM), IRAK4i (10 µM), and quizartinib + IRAK4i

IRAK4i + FLT3i significantly extended leukemia-free survival

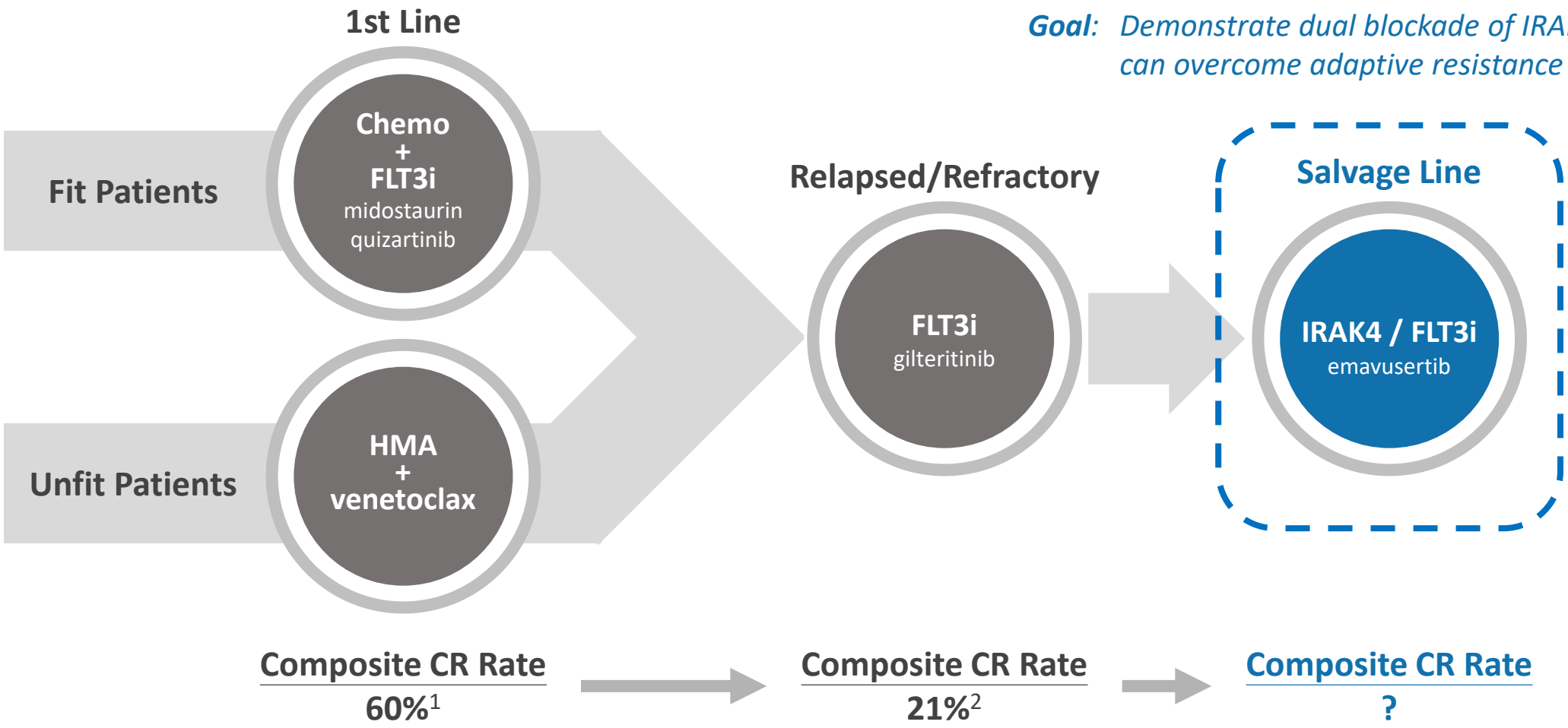


Leukemia-free survival of NRGS mice xenografted with AML-019 patient cells and treated with quizartinib

Concomitant targeting of IRAK4, alongside FLT3, is the most effective means to overcome the adaptive resistance incurred when targeting FLT3[1]

[1] Melgar, Sci Transl Med. 2019

Ph 1/2 study design in AML



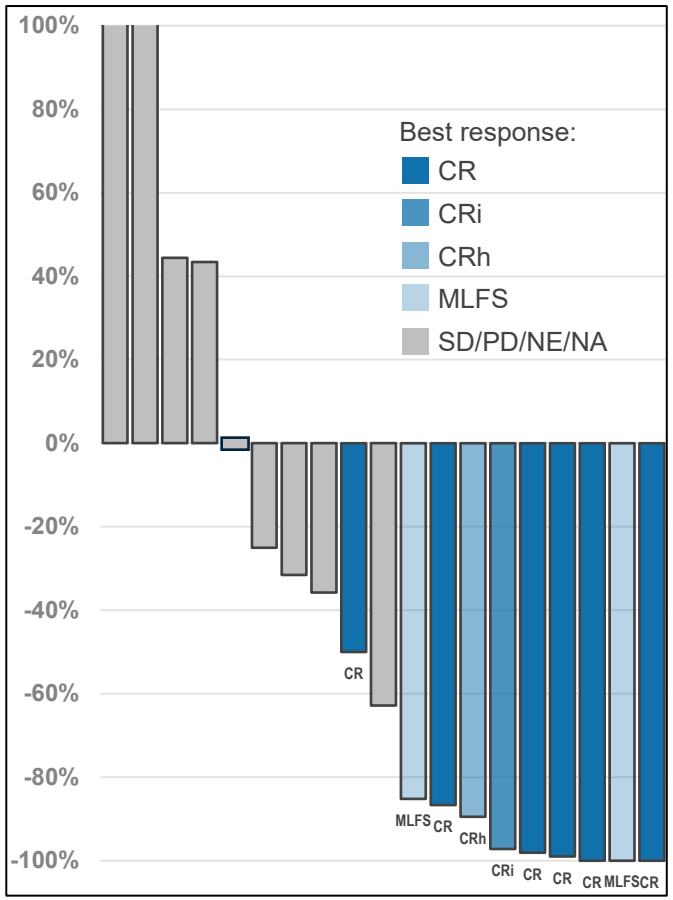
***Goal**: Demonstrate dual blockade of IRAK4 and FLT3 can overcome adaptive resistance to FLT3i*

Composite CR Rate = CR + CRh/i

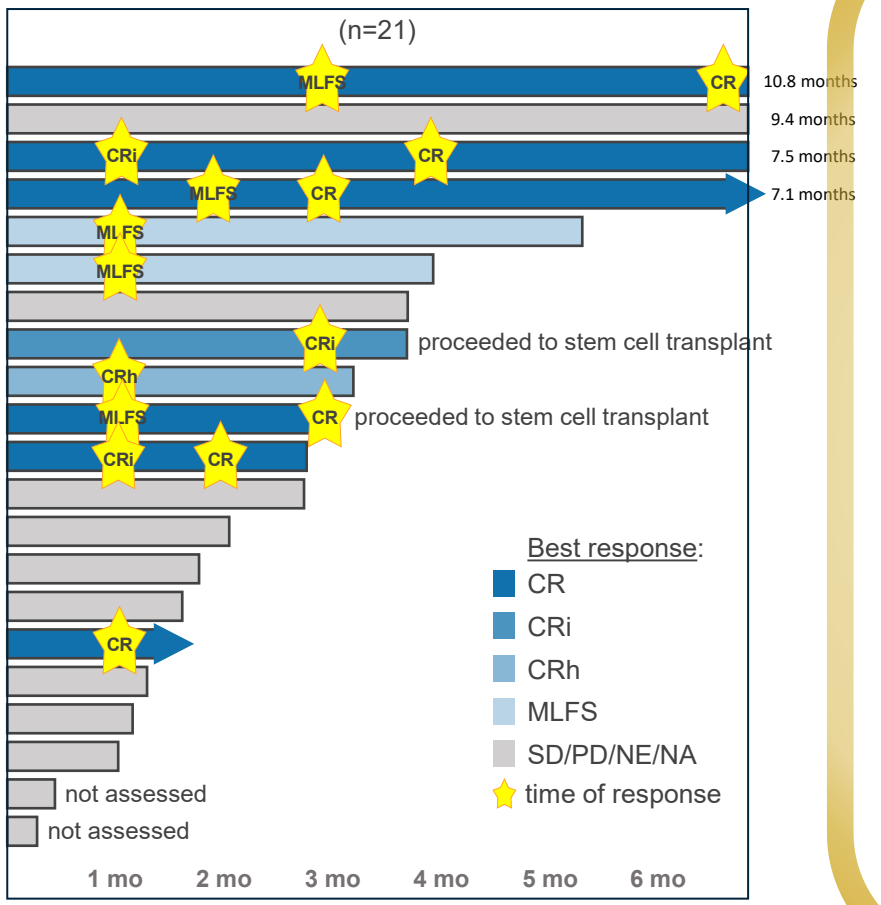
[1] USPI, midostaurin; USPI, quizartinib; USPI, venetoclax; Stone, N Engl J Med. 2015; [2] USPI, gilteritinib

Emavusertib demonstrates the potential to replace gilteritinib as the best-in-class FLT3i

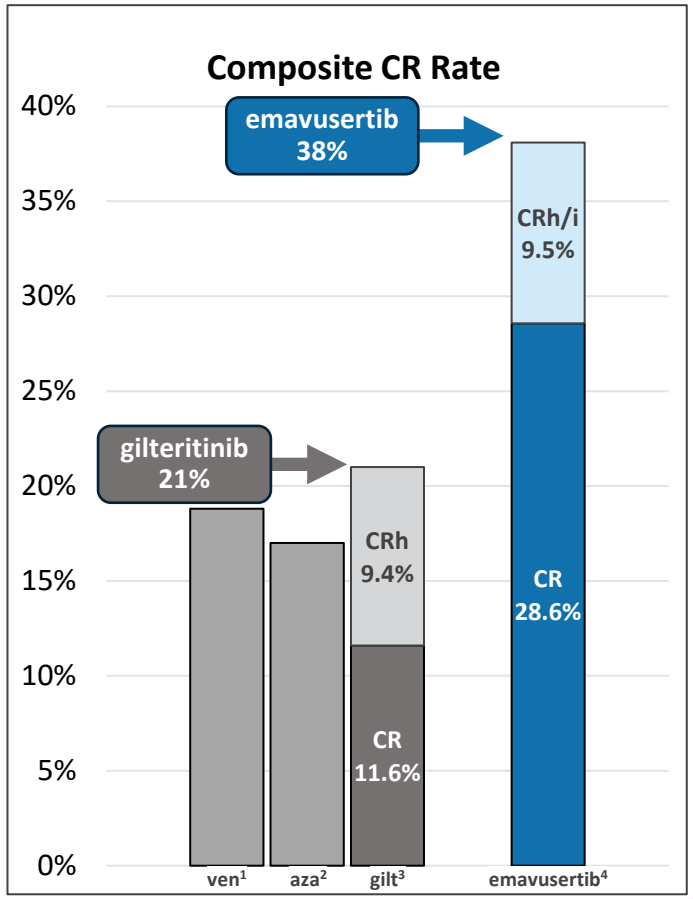
Reduction in Tumor Burden



Time on Treatment



Comparison to Standard of Care



Data include all patients with calculable postbaseline tumor burden at cutoff date. Two additional patients discontinued treatment prior to first disease response assessment.

* 81% of patients had been previously treated with a FLT3 inhibitor

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

Well tolerated safety profile in 102 patients with AML

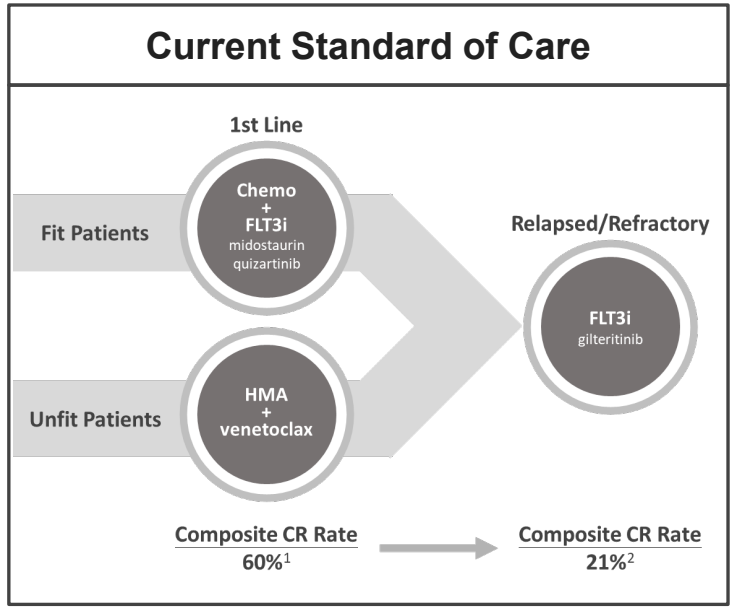
- 102 patients treated in AML
- Well tolerated
- No dose-limiting myelosuppression has been observed

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)
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; however, only 1 of 3 events met laboratory-defined criteria for rhabdomyolysis (CPK >10 x ULN and SCr ≥ 1.5 x ULN) so it is not reported on this table.

Study design for head-to-head vs. gilteritinib



Opportunity with additional funding

Proposed Pivotal Study

Study Design:	randomized vs. gilteritinib in 2nd Line
Dosing & Admin:	300 mg BID, orally
Primary Endpoint:	CR
Secondary Endpoints:	Duration of response (DOR), OS
Study Population:	mFLT3 AML patients who have failed ≤ 2 lines
Study Size:	n=300-400

Approval	BTK Inhibitor	Trial	Treatment Arms	Study Population	Study Size	Median OS (months)	OS HR	ORR%	Median Duration
2017	midostaurin	RATIFY[1]	midostaurin + chemo vs chemo	1st Line	717	74.7 vs 25.6	0.77	CR: 59% vs 54%	
2017	gilteritinib	ADMIRAL[2] interim analysis	gilteritinib	R/R with ITD, D835, I836	138			CR: 11.6% CRh: 9.4%	4.6 mo
		ADMIRAL[2] final analysis	gilteritinib vs. chemotherapy	R/R with ITD, D835, I836	371	9.3 vs 5.6	0.64	CR 14.2% CRh: 8.9%	7.4 mo
2023	quizartinib	QuANTUM-First[3]	quizartinib + chemo vs chemo	1st Line	539	31.9 vs 15.1	0.78	CR: 54.9% vs 55.4%	38.6 vs 12.4

[1] USPI midostaurin and Stone, Blood. 2015; [2] USPI gilteritinib and Pulte, Clin Cancer Res. 2021; [3] USPI quizartinib and Erba, Lancet. 2023

Summary in AML

- Emavusertib monotherapy has potential to be best-in-class in mFLT3 AML
  - ***38% CR/CRh(i)*** *vs. 21% for gilteritinib*[1]

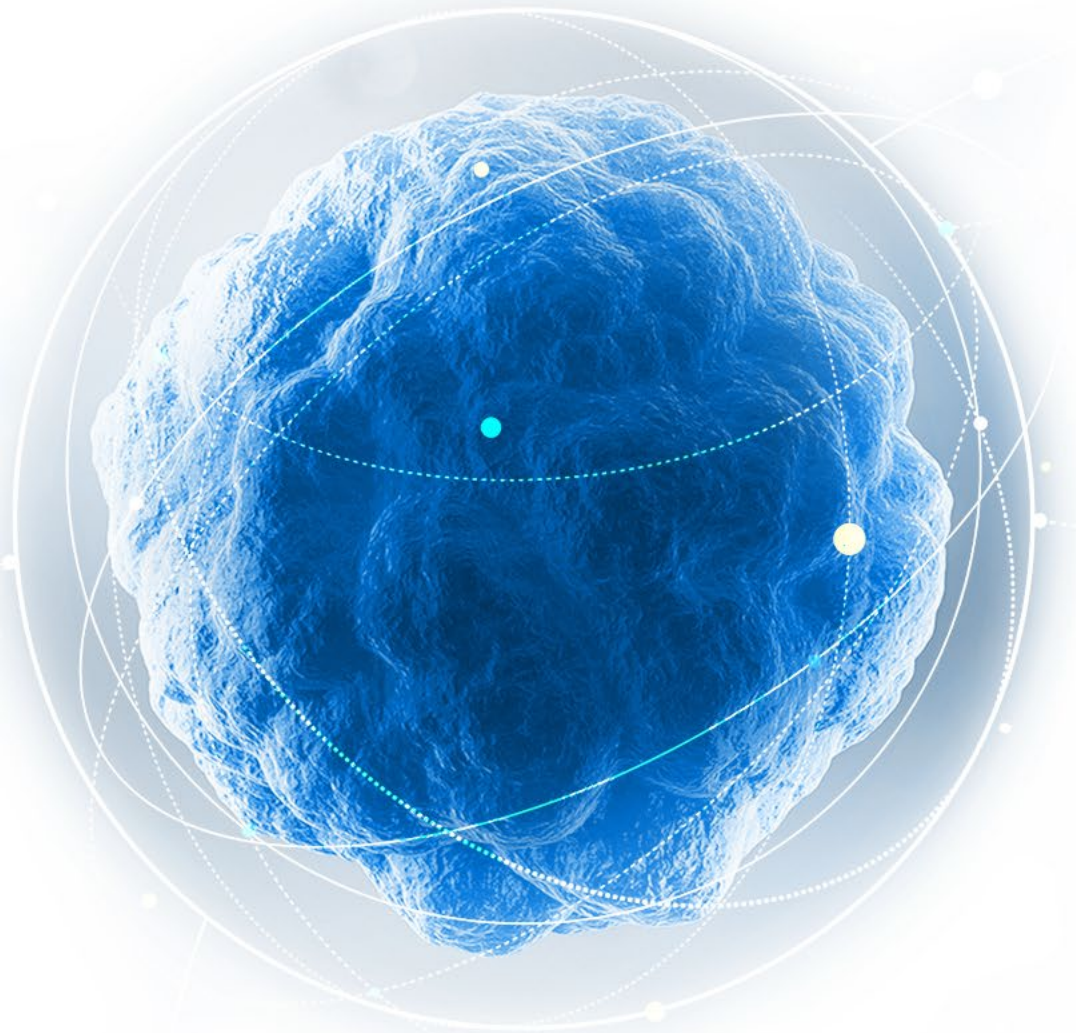
- Planning a registrational study vs. gilteritinib in 2nd line mFLT3 AML

  Goal: *Repeat experience from Ph 1/2*

  Replace gilteritinib as standard of care in R/R mFLT3 AML

[1] USPI gilteritinib

Solid Tumors



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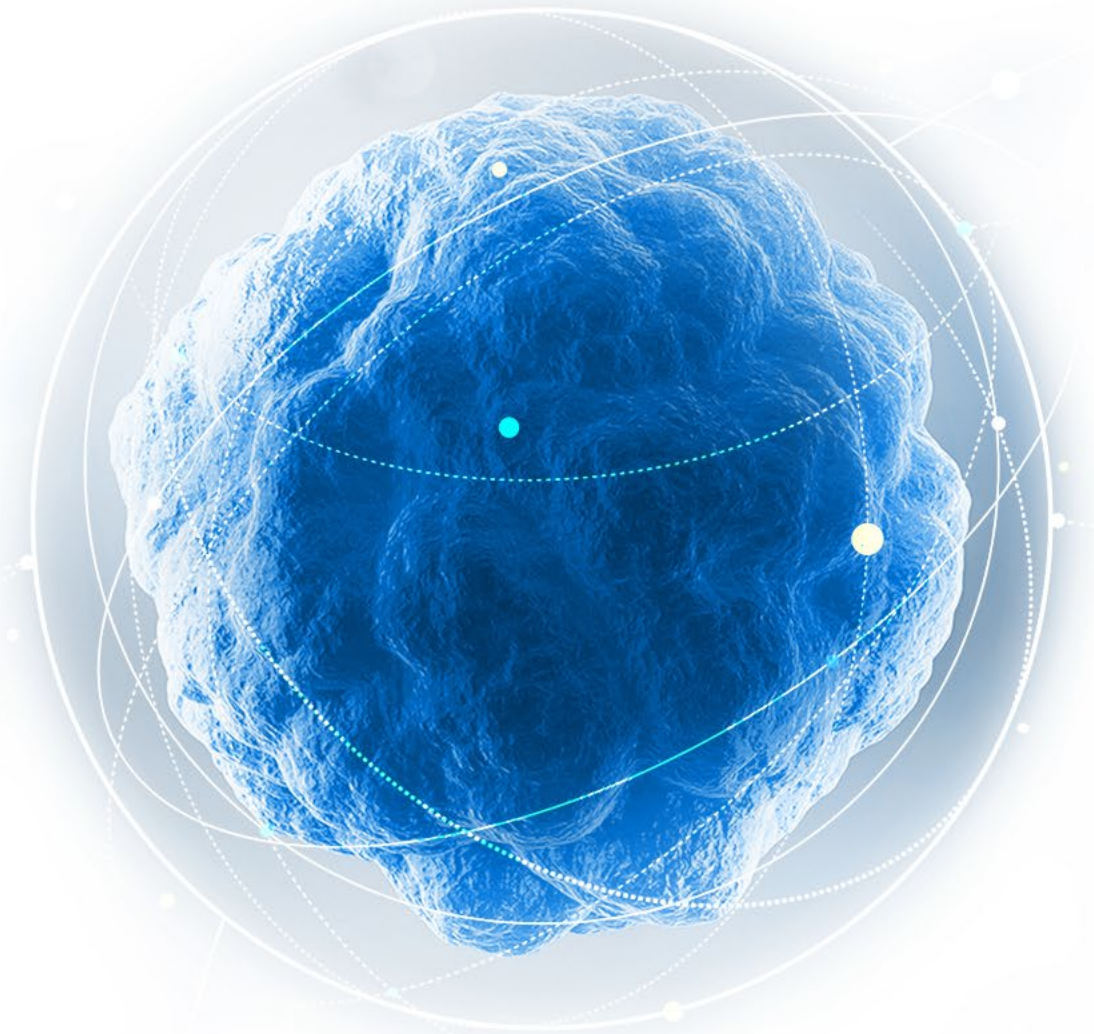
ISTs of emavusertib in Solid Tumors

Tumor Type	Institution (Investigator)	Emavusertib Combination
Pancreatic	NCI CRADA* Washington University (Grierson) *NCT05685602*	gemcitabine, nab-paclitaxel
Colorectal	NCI CRADA* Oklahoma University (Ulahannan) *NCT06696768*	FOLFOX, bevacizumab
Gastro/Esophageal	Washington University (Grierson) *NCT05187182*	FOLFOX, PD1 +/- trastuzumab
Biliary Tract	Washington University (Aranha) *NCT07107750*	cisplatin, gemcitabine, durvalumab
Urothelial	NCI CRADA* Mount Sinai (Galsky) *NCT06439836*	pembrolizumab

Abbreviations: Investigator Sponsored Trial (IST), Cooperative Research and Development Agreement with the National Cancer Institute (NCI CRADA*)

Other Information



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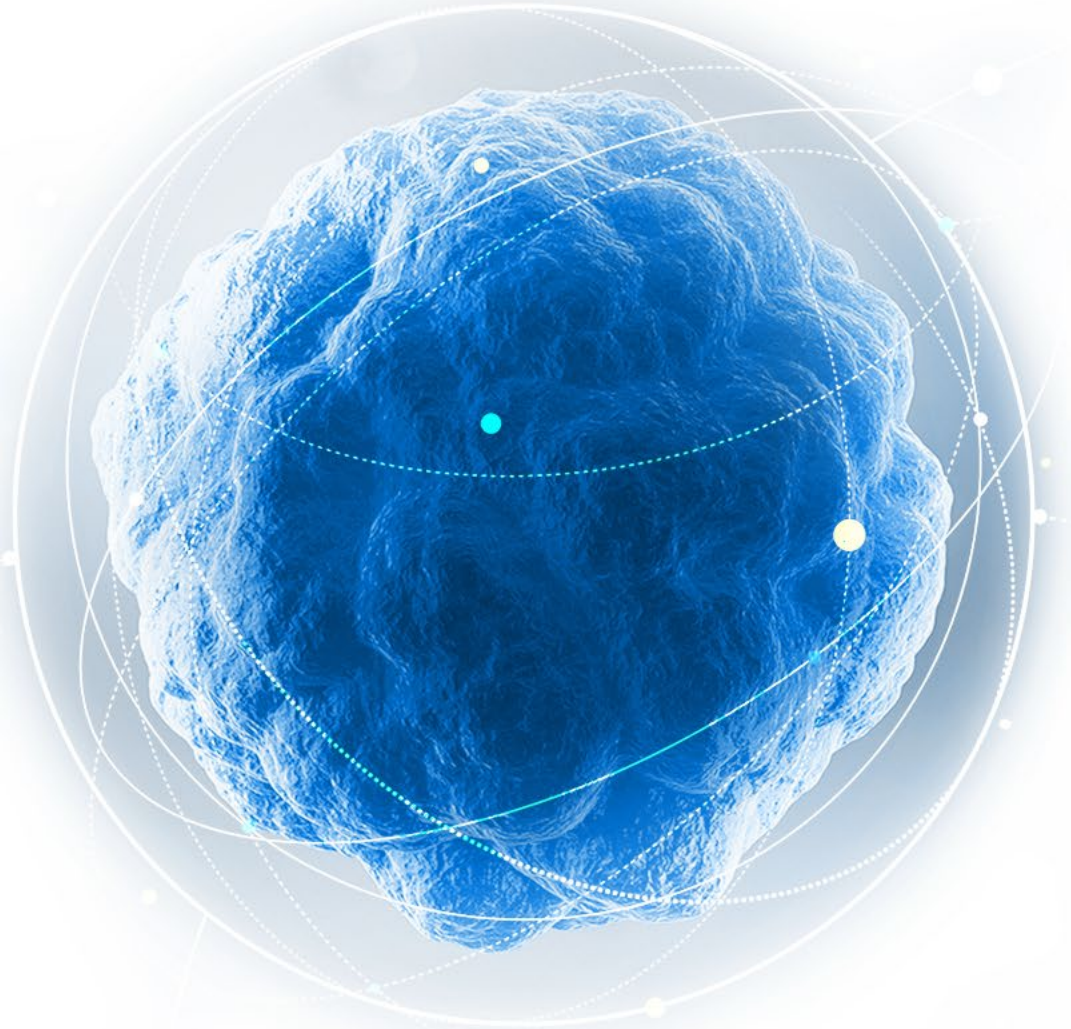
Financials and IP

	Dec 31, 2025	Post Jan 2026 ~$80M PIPE
Cash and Investments	$5.1M	
Shares Outstanding	13M	40M
Pre-Funded Warrants	2.7M	2.7M
Common Stock Warrants	13.8M	94.6M
Employee awards	2.7M	2.7M
Fully Diluted	32M	~140M

Composition of Matter IP on emavusertib extends to 2035 (before potential extension)

End of Presentation



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