DAVA Oncology – 2023 Bermuda Heme Conference

Emavusertib (IRAK4 inhibitor) in Development for Patients with AML/MDS and PCNSL

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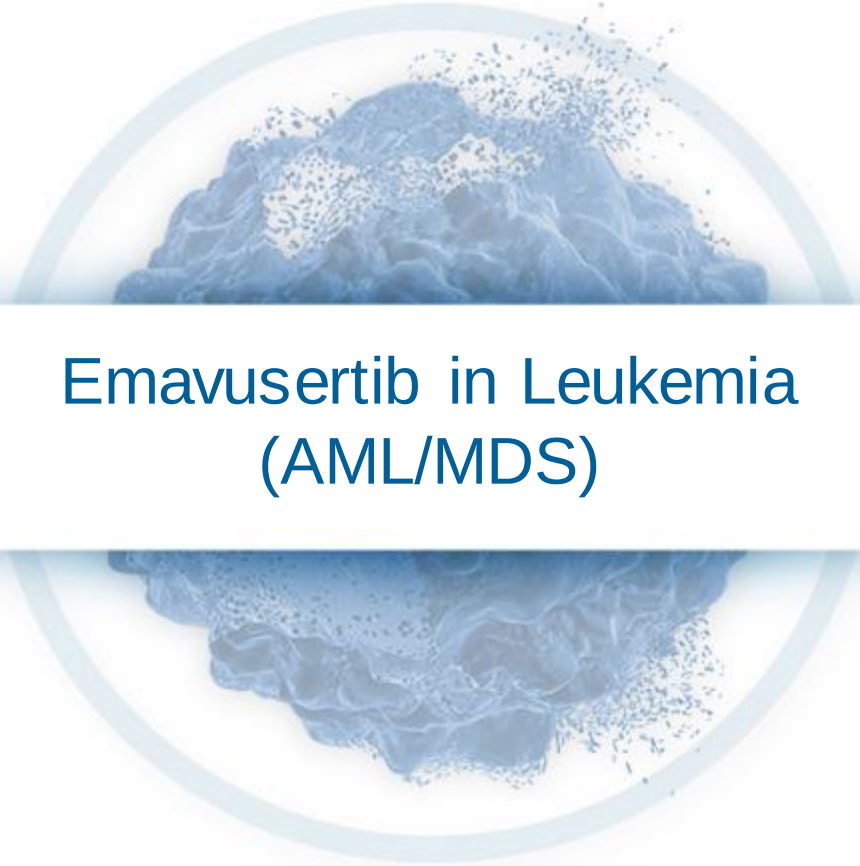


Emavusertib in Leukemia (AML/MDS)

Unique Molecular Fingerprint

Molecular design tailored to be the best-in-class IRAK4 inhibitor

The NCI selected emavusertib for NCI-sponsored research and clinical studies of IRAK4

Emavusertib
Kinase Interaction Map

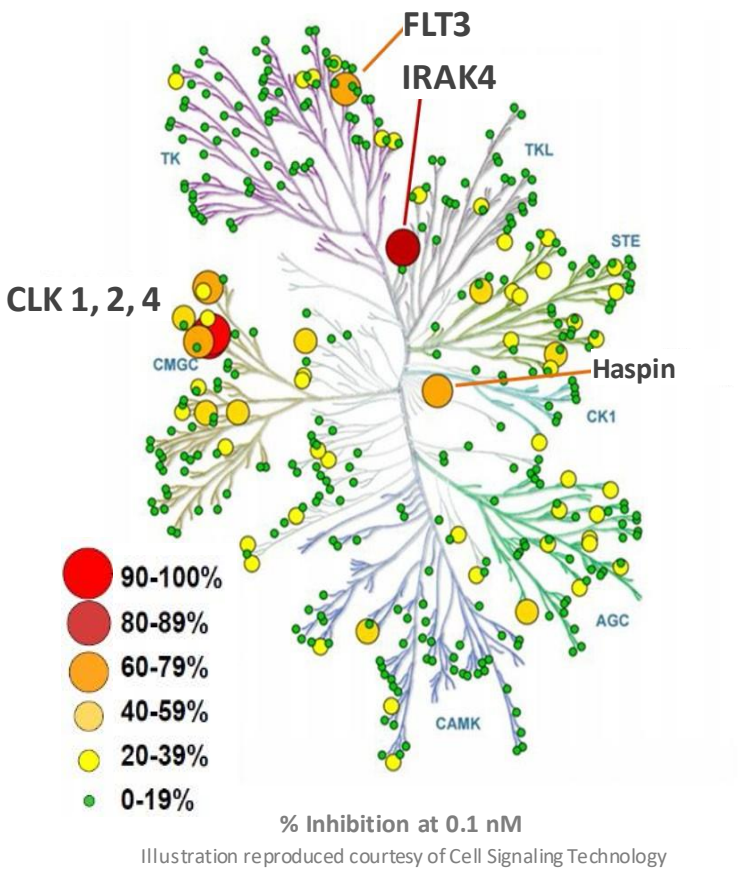


Illustration reproduced courtesy of Cell Signaling Technology

Emavusertib
Binding Affinity

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

DiscoverX Kinase Panel
(378 kinases screened)

high binding affinity to IRAK4
(>97% inhibition achieved at RP2D concentrations)

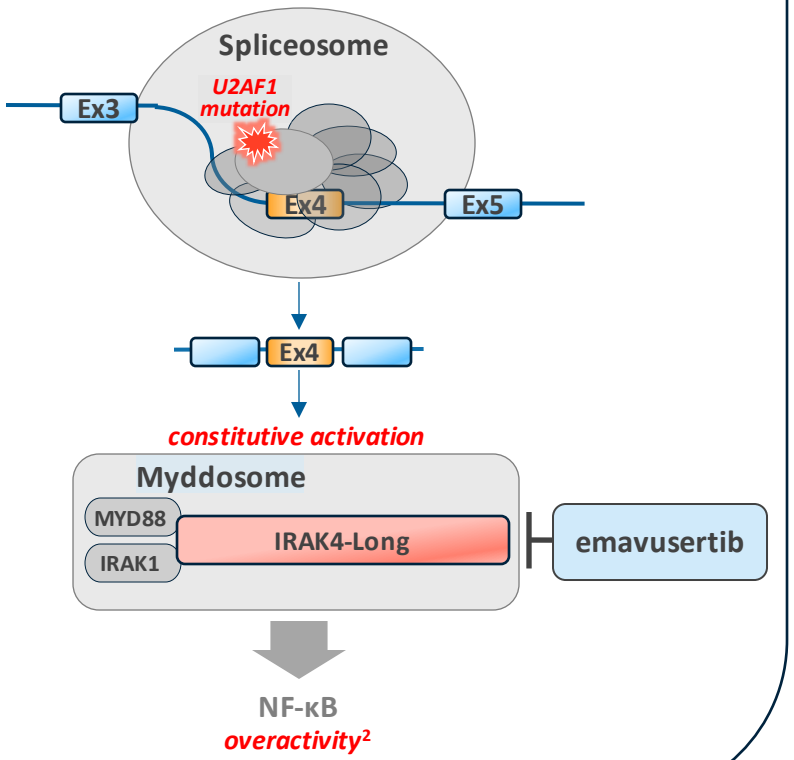
high binding affinity to FLT3
contributes additional anti-cancer activity, differentiating emavusertib from other IRAK4-directed therapies

Mechanism of Action

The two primary targets of emavusertib are independent drivers of cancer

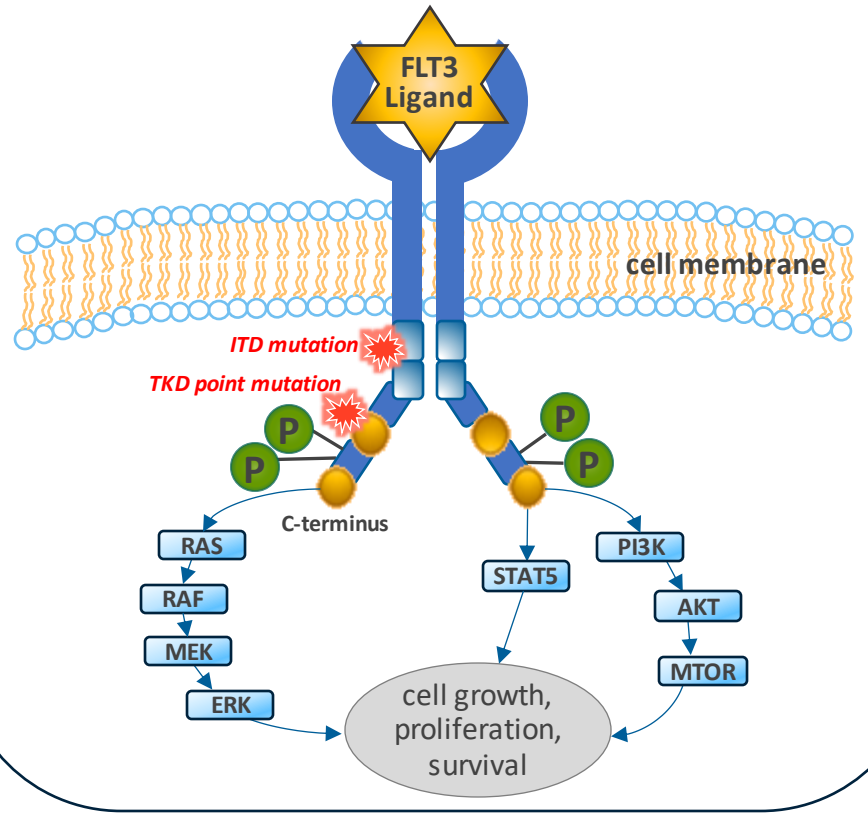
IRAK4

spliceosome mutations drive overexpression of IRAK4-Long, which constitutively activates the myddosome, driving NF-κB overactivity



FLT3

genetic mutations cause constitutive activation of FLT3 and downstream cell growth, proliferation, and survival pathways

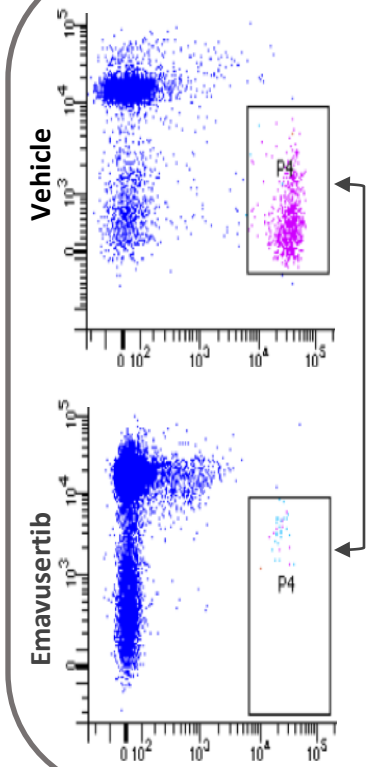


1) Guillamot et al. Nat Cell Biol 2019; 2) Smith et al. Nat Cell Biol 2019; 3) American Cancer Society, Cancer Facts & Figures 2020; 4) Leukemia & Lymphoma Society, Facts and Statistics Overview

Preclinical Data

Rationale for monotherapy and combination with azacitidine/venetoclax

Monotherapy in IRAK4

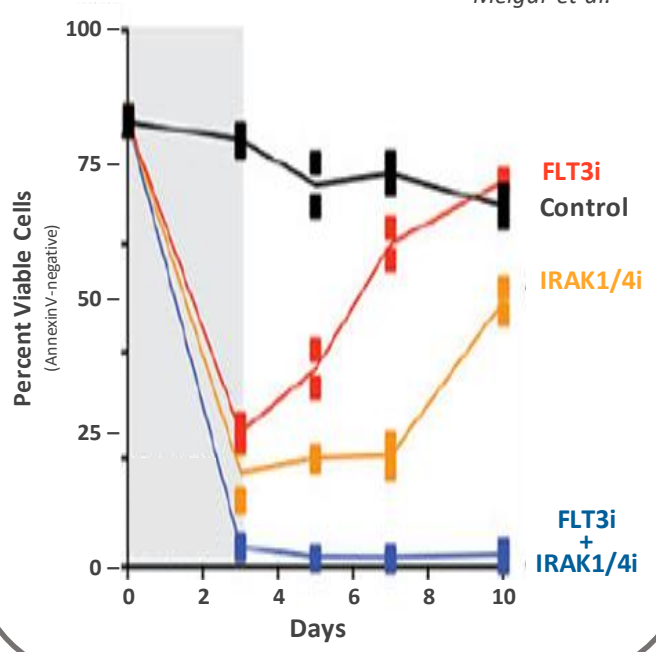


clear reduction of leukemic blasts in models with high IRAK4-L expression

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

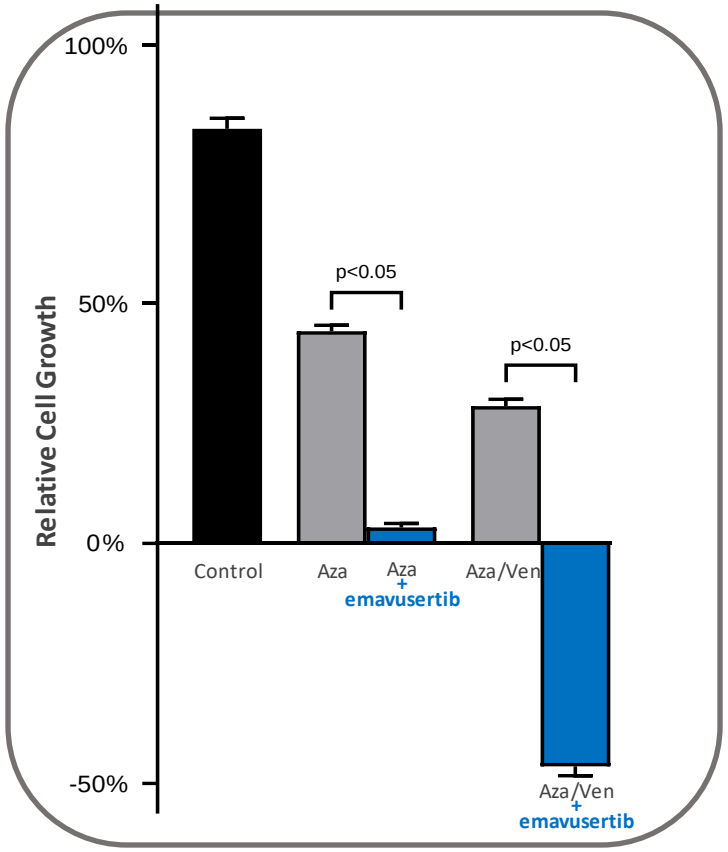
Monotherapy in FLT3

"Concomitant targeting of IRAK1 or IRAK4, alongside FLT3, is the most effective means to overcome the adaptive resistance incurred when targeting FLT3"
- Melgar et al.



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

Combination with Aza/Ven



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

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

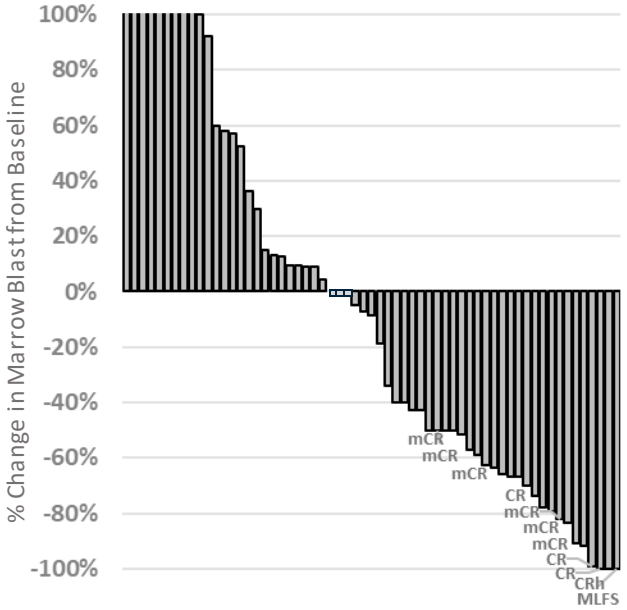
FLT3-ITD cells treated for 3 days with DMSO (control), quizartinib (0.5 µM), IRAKi (10 µM), and quizartinib + IRAKi

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

Initial Clinical Data

Emavusertib is showing clear single agent activity where expected in clinical studies

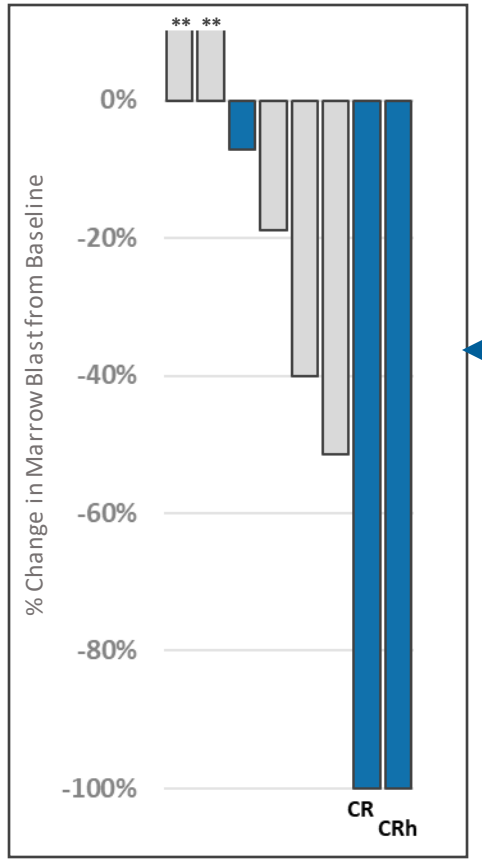
84 AML Patients
all comers, all dose levels



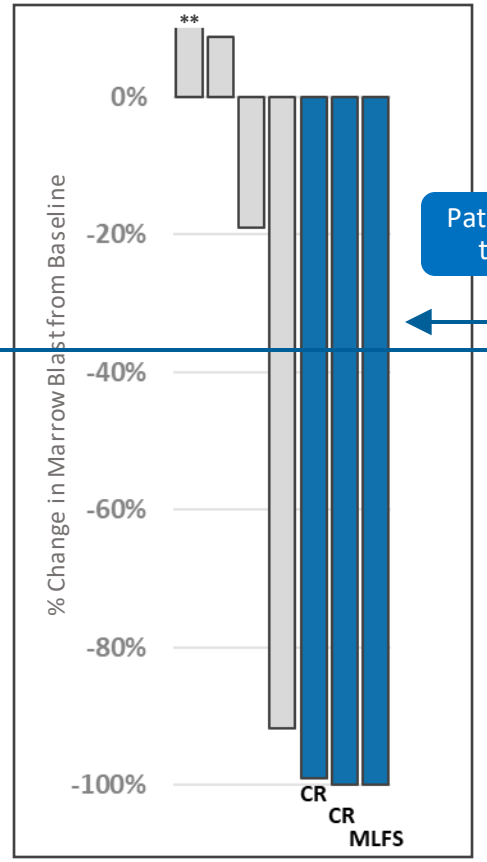
7	AML FLT3*
8	AML Spliceosome*
24	AML Other
17	AML Not Evaluable for Marrow Assessment
12	MDS Spliceosome
13	MDS Other
6	MDS Not Evaluable for Marrow Assessment
84	Total Patients enrolled in monotherapy

strongest monotherapy signal observed where expected
(Spliceosome/FLT3 patients)

8 AML Patients
Spliceosome Mutation*



7 AML Patients
with FLT3 Mutation*



Patients with ≤ 2 prior lines treated at 300 mg BID

Duration of Responses: 5.6 – 7.0 months

Note: 84 total patients enrolled as of Feb 9, 2023 with data cut-off as of Mar 17, 2023;
* Three AML patients had both FLT3 and Spliceosome mutation and are counted in both populations; evaluable patients include all patients whose disease was determined to be evaluable for objective response with baseline and post-treatment marrow assessments
** Denotes blast percent increase > 10%

Clinical Strategy in Leukemia

TakeAim Leukemia Strategy for Monotherapy and Combination

Potential for fast path to NDA

Monotherapy

Enroll 20 Patients (2L/3L)
in genetically-defined populations

- R/R AML with FLT3
- R/R AML with Spliceosome

For each indication, collect clinical data in ~20 patients to facilitate pivotal design discussions with regulatory agencies, including potential for accelerated development

Potential for high value front-line position

Combination

Enroll 20 Patients (1L)
all comers

- 1st line AML combination with aza/ven
- 1st line MDS combination TBD

For each indication, collect clinical data in ~20 patients to establish safety and anti-cancer activity to support discussions with regulatory agencies in front-line opportunity

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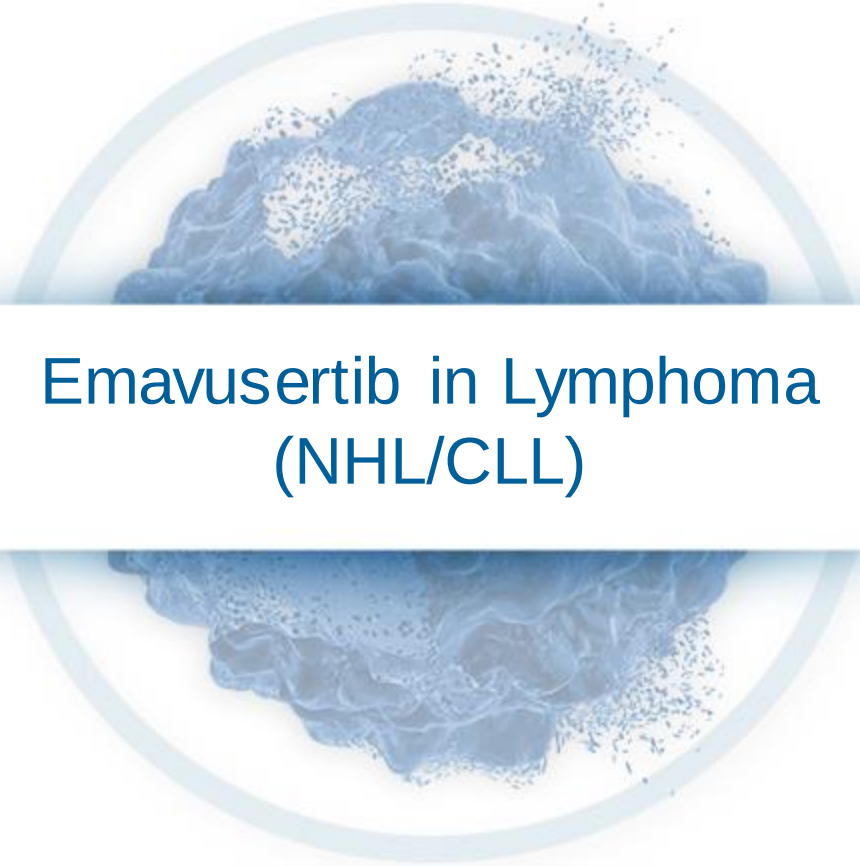


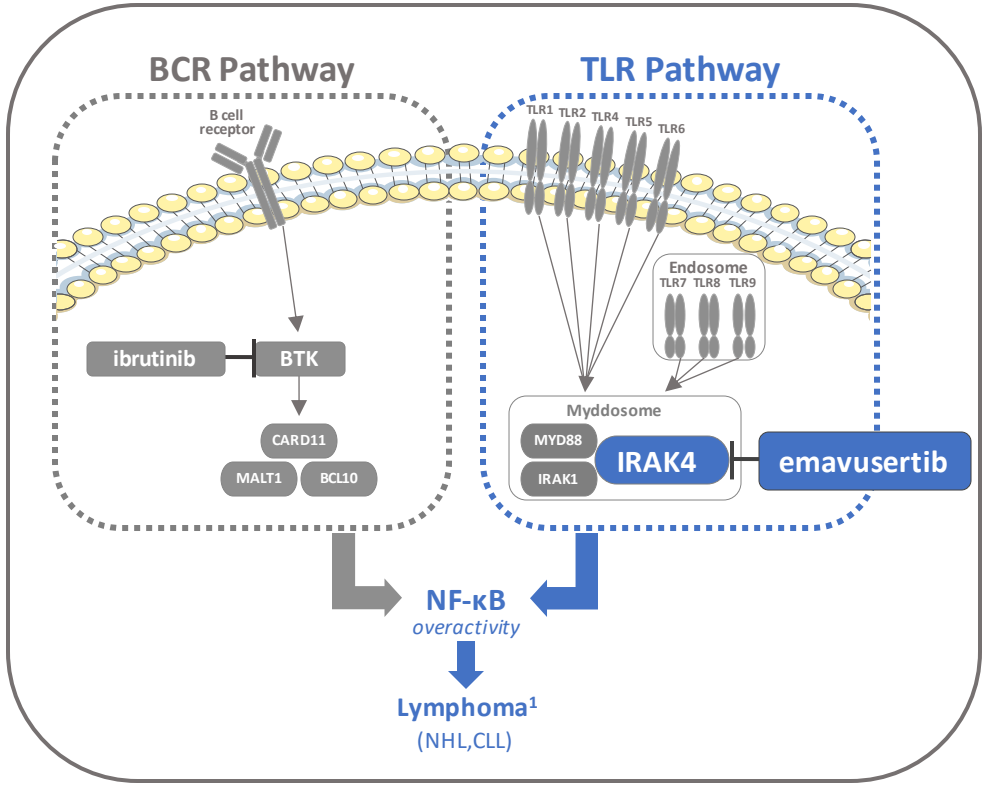
Emavusertib in Lymphoma (NHL/CLL)

Emavusertib in Lymphoma

Combination therapy provides complimentary inhibition of two pathways that drive NF-κB

NFκB Biology:
Two Pathways Drive NHL/CLL

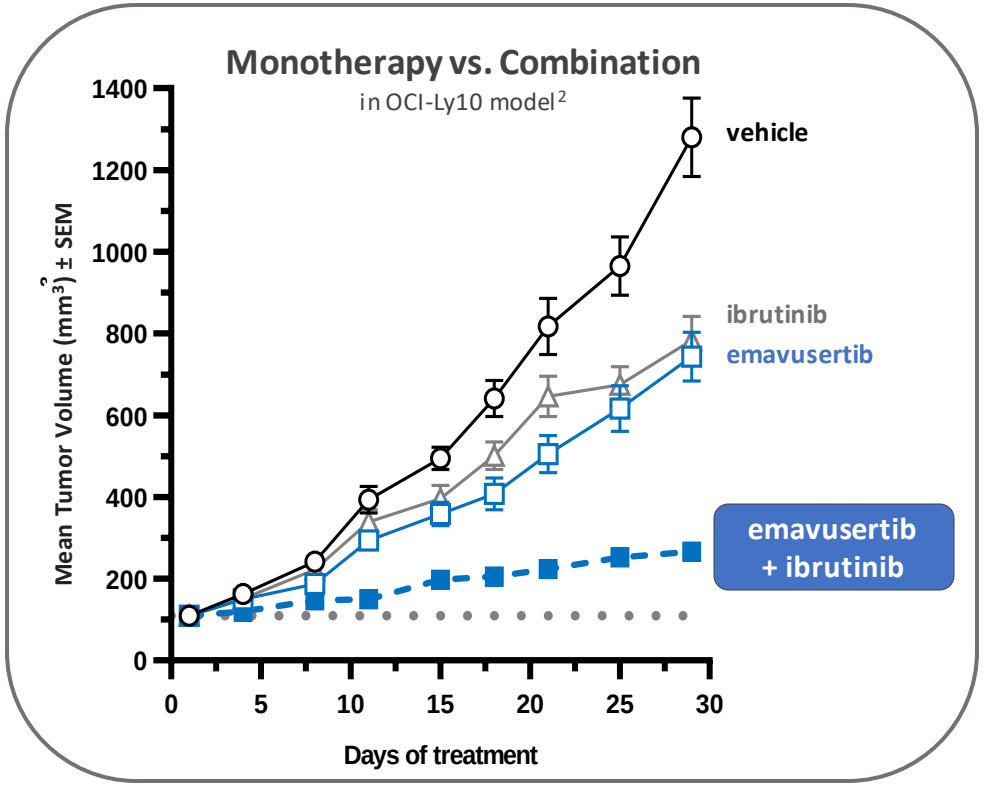
BCR and TLR Pathways independently drive NF-κB overactivity (and NF-κB drives NHL/CLL)



1) IMBRUVICA Package Insert. Rev 08/2018

Clinical Strategy:
Block both pathways with Combination Therapy

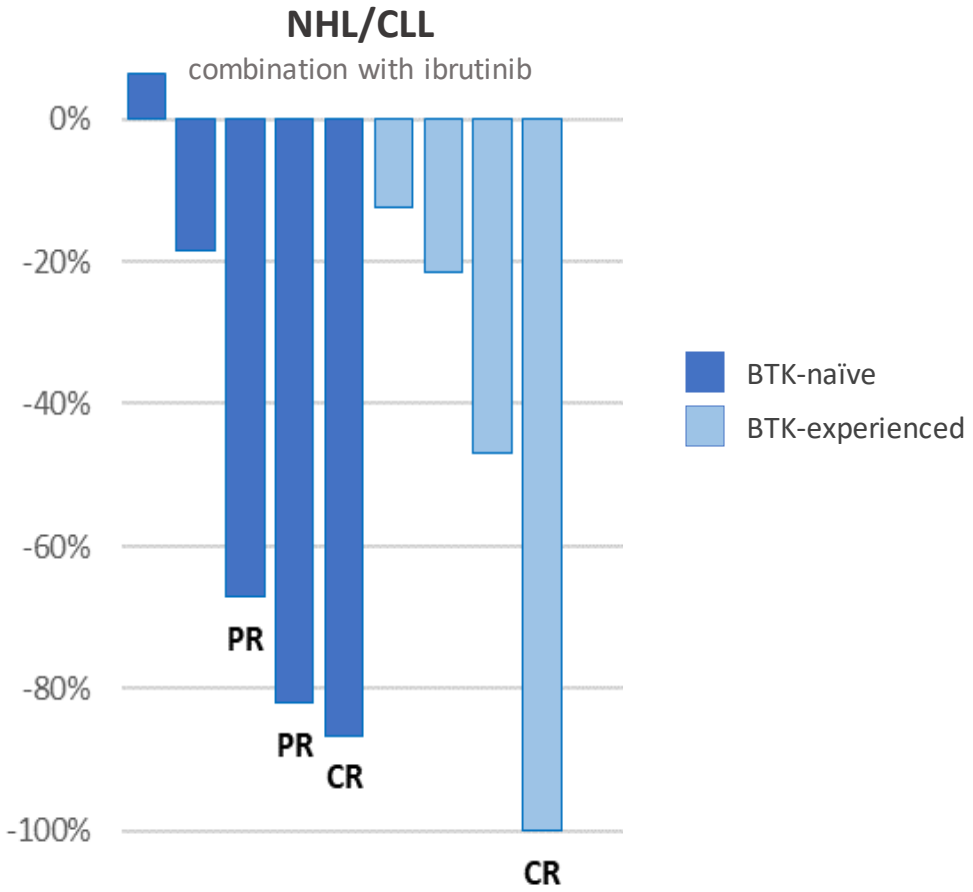
In preclinical testing, blocking both IRAK4 and BTK drove tumor reduction better than blocking either one alone

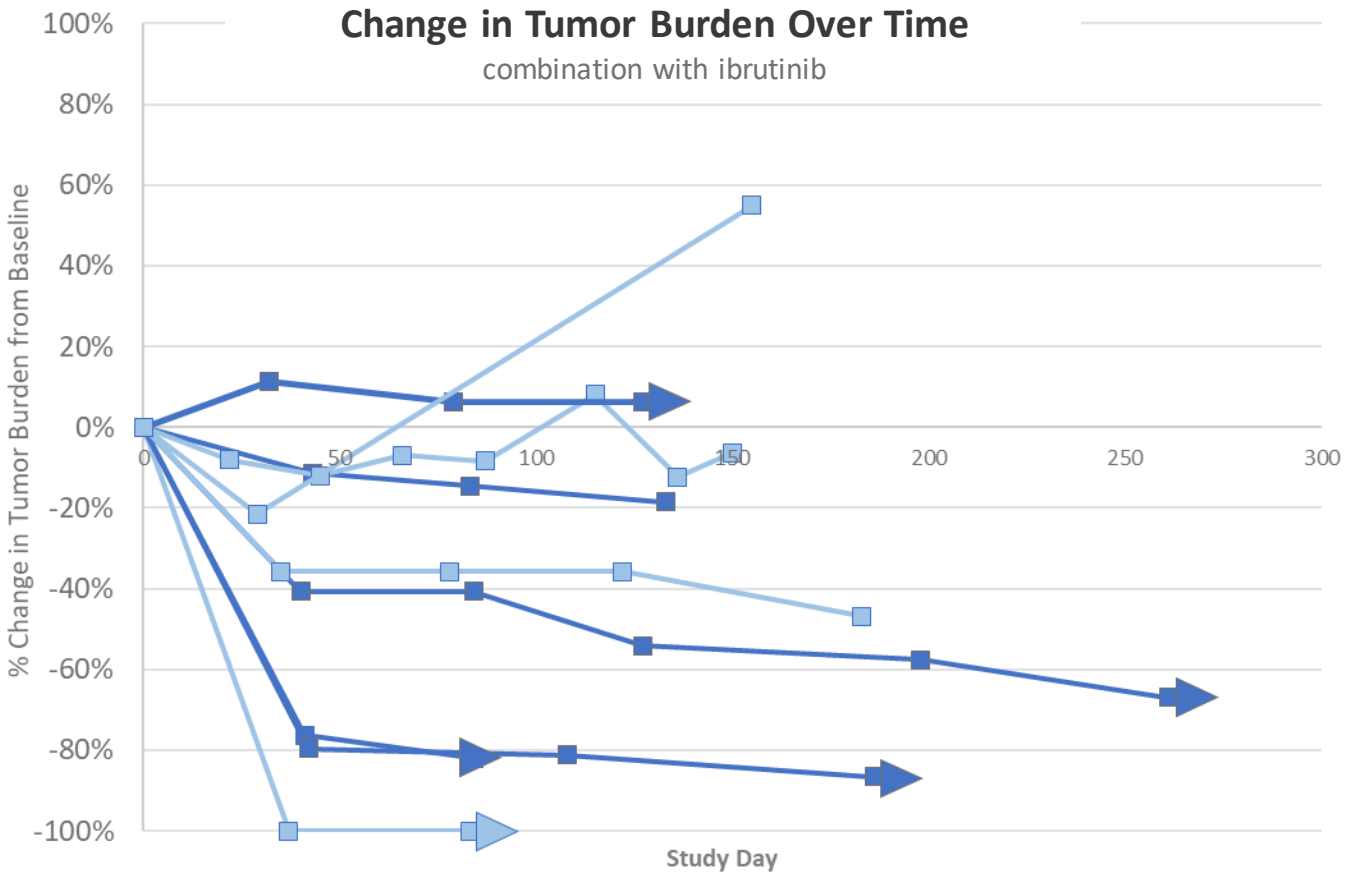


2) Booher et al. Waldenström Roadmap Symposium 2019

Initial Clinical Data in Lymphoma

Majority of patients achieved decreases in tumor burden, including complete responses





Note: Data from ASCO 2022 poster presentation
Response evaluable patients with baseline and post-treatment disease assessment at data cutoff

Clinical Strategy in Lymphoma

TakeAim Lymphoma Strategy for Combination

Potential for front-line position

Monotherapy

not being pursued in NHL

because blocking both the TLR and BCR Pathways
(with IRAK4i and BTKi, respectively)
is better than blocking either one alone

Combination

Enroll 20 Patients (2L/3L)

- R/R PCNSL combination with ibrutinib

Collect clinical data in ~20 patients
to facilitate pivotal design discussions with regulatory agencies,
including potential for accelerated development

End of Presentation