

# Company Overview

J.P. Morgan 2026 Healthcare Conference

THE NEXT LEAP FORWARD IN PRECISION MEDICINE





**Intelligently designed small molecules  
to address the challenges of cancer treatment resistance with the  
potential for deeper, longer response**

**BLOSSOMHILL  
THERAPEUTICS**  
Founded in  
San Diego, CA

**\$71M**  
Preferred Series A  
Cormorant Asset Management  
**orbimed** VIVO CAPITAL  
HERCULES BioVentures

**\$100M**  
Preferred Series B  
COLT VENTURES  
PLAISANCE CAPITAL MANAGEMENT  
Cormorant Asset Management  
VIVO CAPITAL  
**orbimed**

**CLK**  
Inhibitor (BH-30236)  
First in human

**OMNI-EGFR™**  
Inhibitor (BH-30643)  
First in human

**\$84M**  
Preferred Series B+  
Janus Henderson INVESTORS  
Brahma Capital  
Cormorant Asset Management  
PLAISANCE CAPITAL MANAGEMENT  
VIVO CAPITAL  
**orbimed** BioTrack Capital

Jun 2020

Mar 2021

Feb 2024

Jun 2024

Jan 2025

Dec 2025



# Experienced Leadership, Proven Drug Design Expertise

## J. Jean Cui, PhD

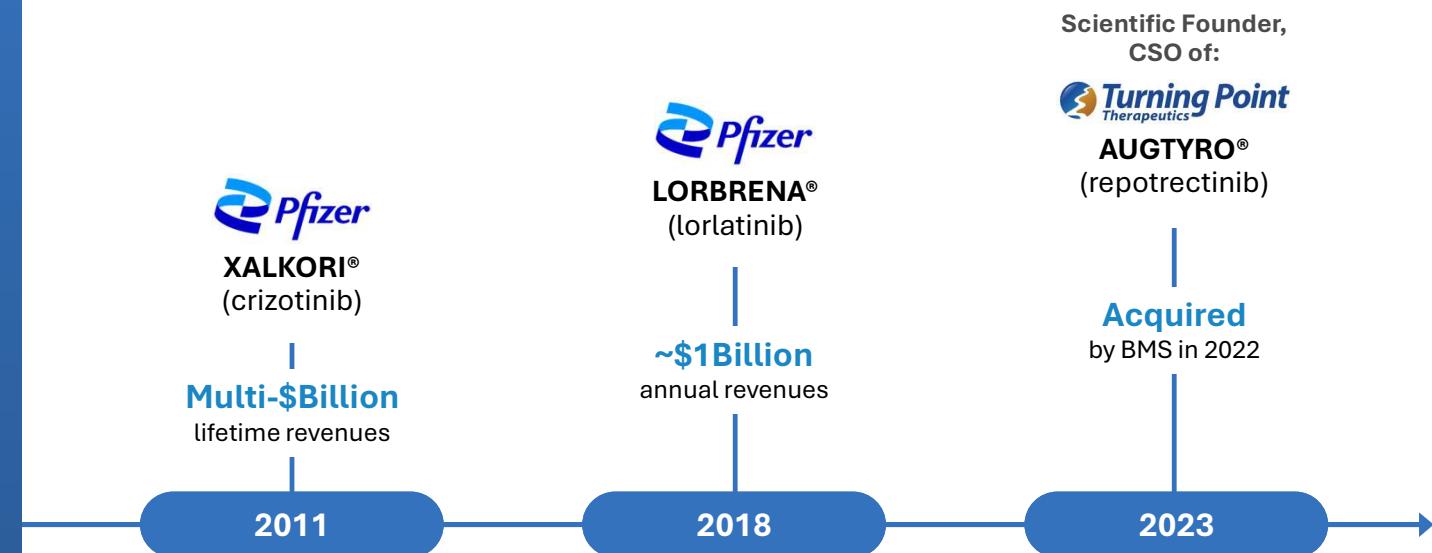
Scientific Founder, President and CEO



### Sustained Recognition

- Elected to the National Academy of Engineering .....2024
- American Chemical Society  
Heroes of Chemistry .....2013 & 2021
- 38<sup>th</sup> Annual Inventor of the Year .....2011

## 3 FDA-Approved Cancer Therapeutics



## 30 Years of Drug Design and Development



## Our Differentiated, Global, Wholly-Owned Pipeline

Target	Candidate	Indication/Therapeutic Area	Differentiation/Opportunity	Discovery	IND Enabling	Phase 1 Dose Escalation	Phase 1 Dose Expansion
EGFR	<b>BH-30643</b>	EGFR-mutant NSCLC	<ul style="list-style-type: none"><li>Mutant-selective, non-covalent, macrocyclic OMNI-EGFR inhibitor</li><li>Potent against classical, atypical, resistance mutations</li></ul>	<div style="width: 100%;"><div style="width: 100%; background-color: #0072BD; height: 10px; margin-bottom: 5px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div></div>	<div style="width: 100%;"><div style="width: 100%; background-color: #D9E1F2; height: 10px; margin-bottom: 5px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div></div>	<div style="width: 100%;"><div style="width: 100%; background-color: #D9E1F2; height: 10px; margin-bottom: 5px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div></div>	<div style="width: 100%;"><div style="width: 100%; background-color: #D9E1F2; height: 10px; margin-bottom: 5px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div></div>
CLK	<b>BH-30236</b>	R/R AML and HR-MDS	<ul style="list-style-type: none"><li>First-in-class opportunity</li></ul>	<div style="width: 100%;"><div style="width: 100%; background-color: #0072BD; height: 10px; margin-bottom: 5px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div></div>	<div style="width: 100%;"><div style="width: 100%; background-color: #D9E1F2; height: 10px; margin-bottom: 5px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div></div>	<div style="width: 100%;"><div style="width: 100%; background-color: #D9E1F2; height: 10px; margin-bottom: 5px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div></div>	<div style="width: 100%;"><div style="width: 100%; background-color: #D9E1F2; height: 10px; margin-bottom: 5px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div></div>
KRAS	<b>Undisclosed</b>	Oncology	<ul style="list-style-type: none"><li>Novel chemical scaffold</li></ul>	<div style="width: 100%;"><div style="width: 100%; background-color: #0072BD; height: 10px; margin-bottom: 5px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div></div>	<div style="width: 100%;"><div style="width: 100%; background-color: #D9E1F2; height: 10px; margin-bottom: 5px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div></div>	<div style="width: 100%;"><div style="width: 100%; background-color: #D9E1F2; height: 10px; margin-bottom: 5px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div></div>	<div style="width: 100%;"><div style="width: 100%; background-color: #D9E1F2; height: 10px; margin-bottom: 5px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div></div>
Undisclosed Validated Targets	<b>Discovery</b>	Oncology, metabolic and autoimmune diseases	<ul style="list-style-type: none"><li>Highly selective, macrocycle molecules</li></ul>	<div style="width: 100%;"><div style="width: 100%; background-color: #0072BD; height: 10px; margin-bottom: 5px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div></div>	<div style="width: 100%;"><div style="width: 100%; background-color: #D9E1F2; height: 10px; margin-bottom: 5px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div></div>	<div style="width: 100%;"><div style="width: 100%; background-color: #D9E1F2; height: 10px; margin-bottom: 5px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div></div>	<div style="width: 100%;"><div style="width: 100%; background-color: #D9E1F2; height: 10px; margin-bottom: 5px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div><div style="width: 100%; background-color: #D9E1F2; height: 10px;"></div></div>



# BH-30643: OMNI-EGFR™ Inhibitor

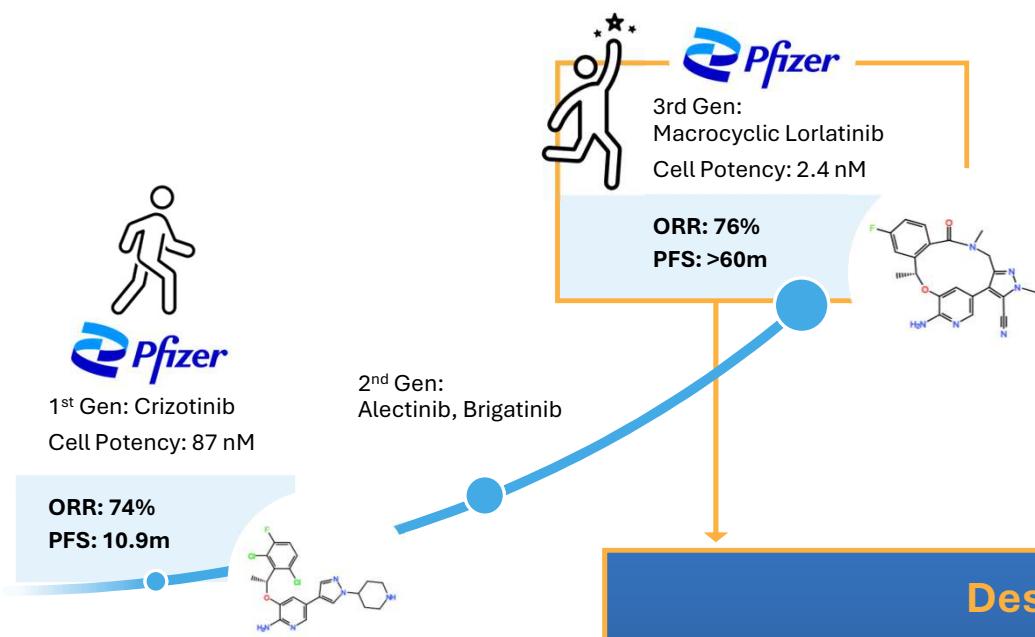
Novel macrocyclic, non-covalent, targeting mutant EGFR active conformation



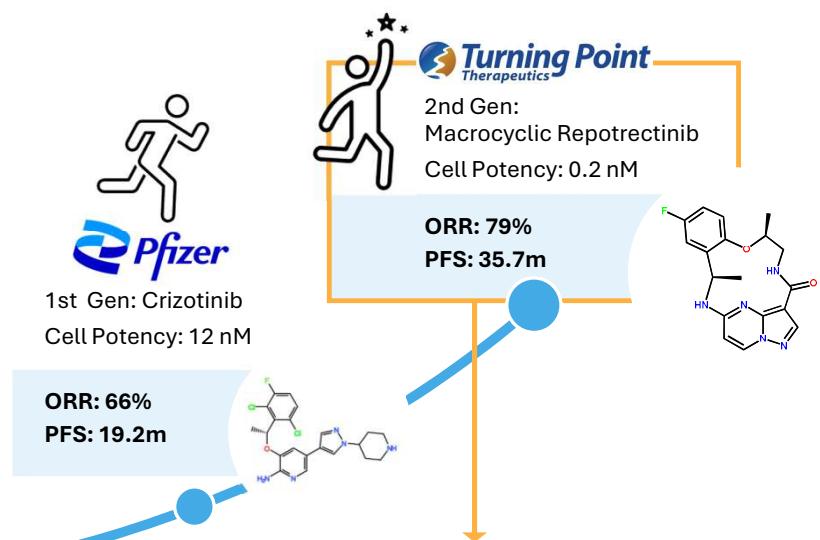
# Making a Leap in Patient Treatment Outcomes

## ACHIEVING LONGER PFS AND PRE-EMPTION OF RESISTANCE WITH SUPER-POTENCY

### EML4-ALK Inhibitors in *ALK*<sup>+</sup> NSCLC



### ROS1 Inhibitor in *ROS1*<sup>+</sup> NSCLC



### Design Approach and Attributes

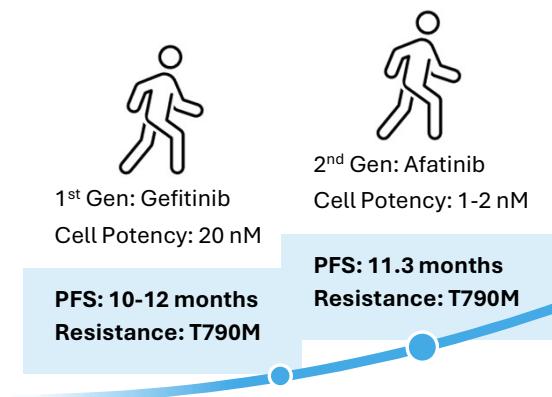
- Macrocyclic design for conformationally restricted chemotype and precision interactions with target
- Super-potency against both primary and secondary mutations
- Better drug-like properties



# Our Goal: Making the Leap in *EGFR*-mutant NSCLC

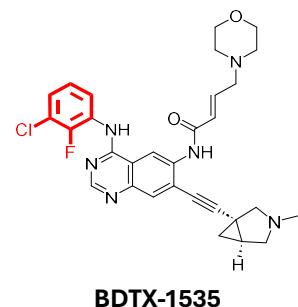
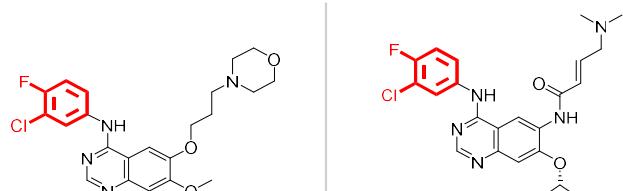
## Limitations of Existing *EGFR* TKIs:

- Suboptimal efficacy constrained by wildtype *EGFR*/HER2 toxicity
- Liable to on-target resistance (e.g. T790M, C797S, etc.)
- Targeting only a subset of *EGFR* mutations (eg, classical, PACC, exon 20 insertion, etc.)

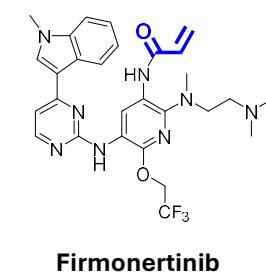
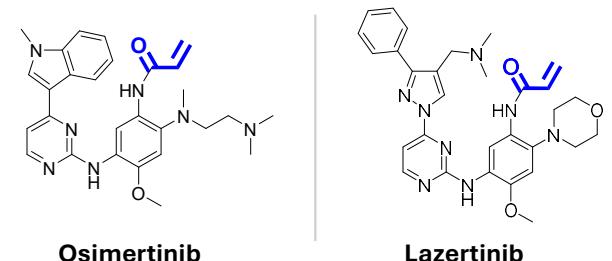


# Existing EGFR Tyrosine Kinase Inhibitors Share Structure Similarities and Common Functional Liabilities

## T790M-resistance



## C797S-resistance



Newer EGFR inhibitors commonly iterate on prior designs, **inheriting the same limitations**

Many TKIs use a **back-pocket motif** for potency

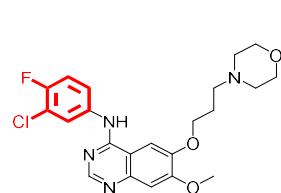
Gefitinib	Afatinib	STX-721
Erlotinib	Dacomitinib	Zipalertinib
Icotinib	BDTX-1535	Enozertinib

Other TKIs **covalently bind** for potency

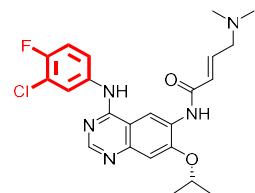
Osimertinib	Almonertinib	Mobocertinib
Rociletinib	Aumolertinib	Sunvozertinib
Lazertinib	Olmutinib	Firmonertinib

# Existing EGFR Tyrosine Kinase Inhibitors Share Structure Similarities and Common Functional Liabilities

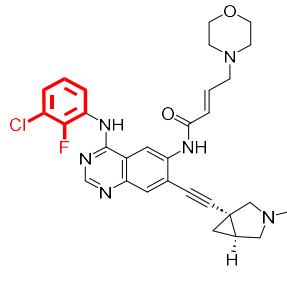
## T790M-resistance



Gefitinib

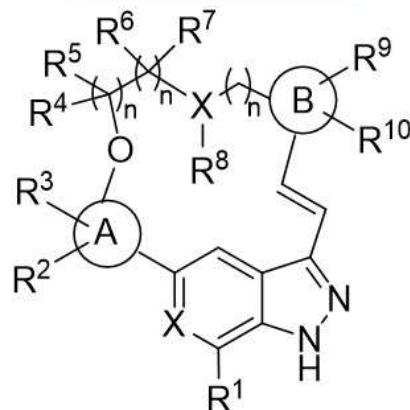


Afatinib



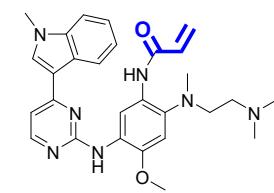
BDTX-1535

## BH-30643

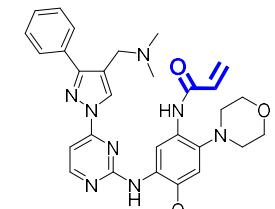


BH-30643 leverages an entirely novel macrocyclic scaffold to uniquely address both T790M and C797S resistance

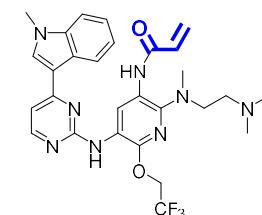
## C797S-resistance



Osimertinib



Lazertinib



Firmonertinib

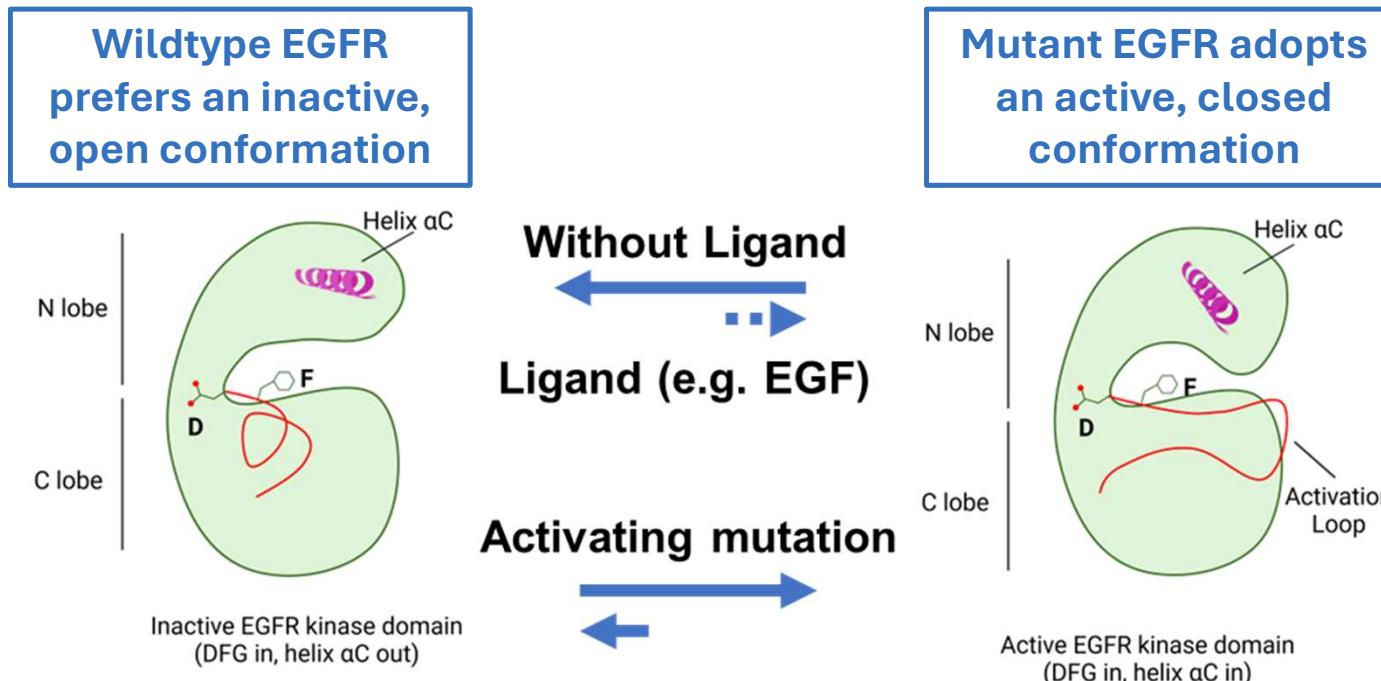
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Other TKIs **covalently bind** for potency

Osimertinib	Almonertinib	Mobocertinib
Rociletinib	Aumolertinib	Sunvozertinib
Lazertinib	Olmutinib	Firmonertinib

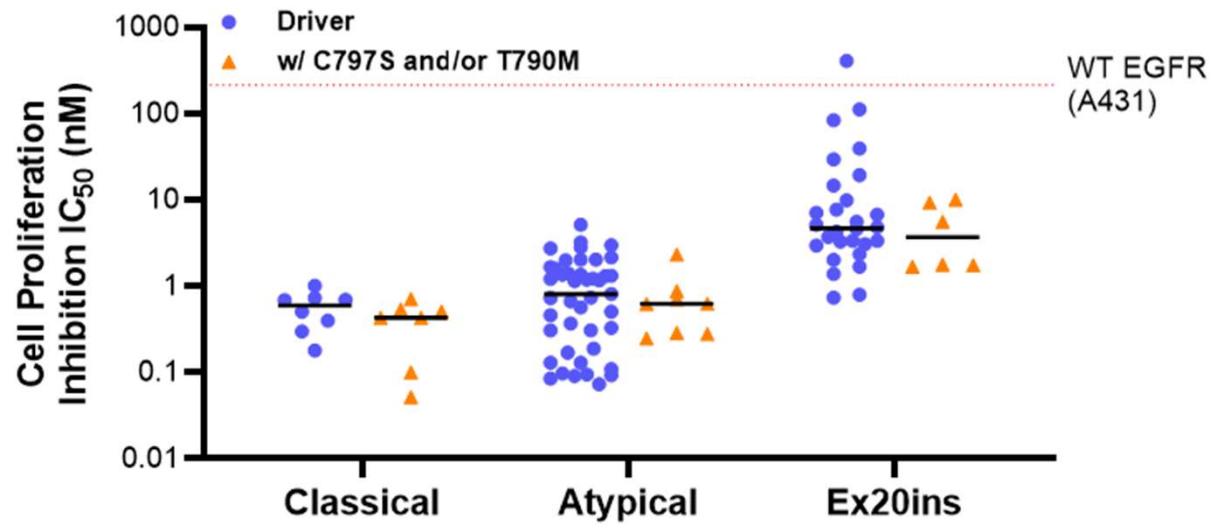
## Design Principle: Targeting the Mutant EGFR Active Conformation



**Targeting the active conformation unlocks the potential for OMNI-EGFR activity**



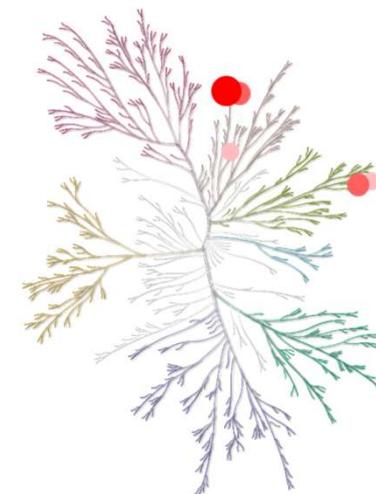
## BH-30643 Represents a First-in-class OMNI-EGFR Kinase Inhibitor



$IC_{50}$ (nM): Median (N)	Classical (N=15)	Atypical (N=51)	Ex20ins (N=34)
<b>Driver</b>	0.60 (8)	0.81 (43)	4.73 (28)
<b>w/ C797S and/or T790M</b>	0.43 (7)	0.63 (8)	3.69 (6)

Note: Classical mutations include ex19del, L858R; Atypical mutations with prevalence  $\geq 0.1\%$  (Sisoudiya et al, 2024; *NPJ Precision Oncology*) include genetic alterations at G719, L861, S768, E709, L747, V834, L833, V769 and any compounds of these mutations with one another or with classical mutations; Ex20ins mutations include any in-dels in ex20; Resistance mutations include C797S, T790M. The bar in each group indicates median value.

Selectivity over 371 human WT kinases



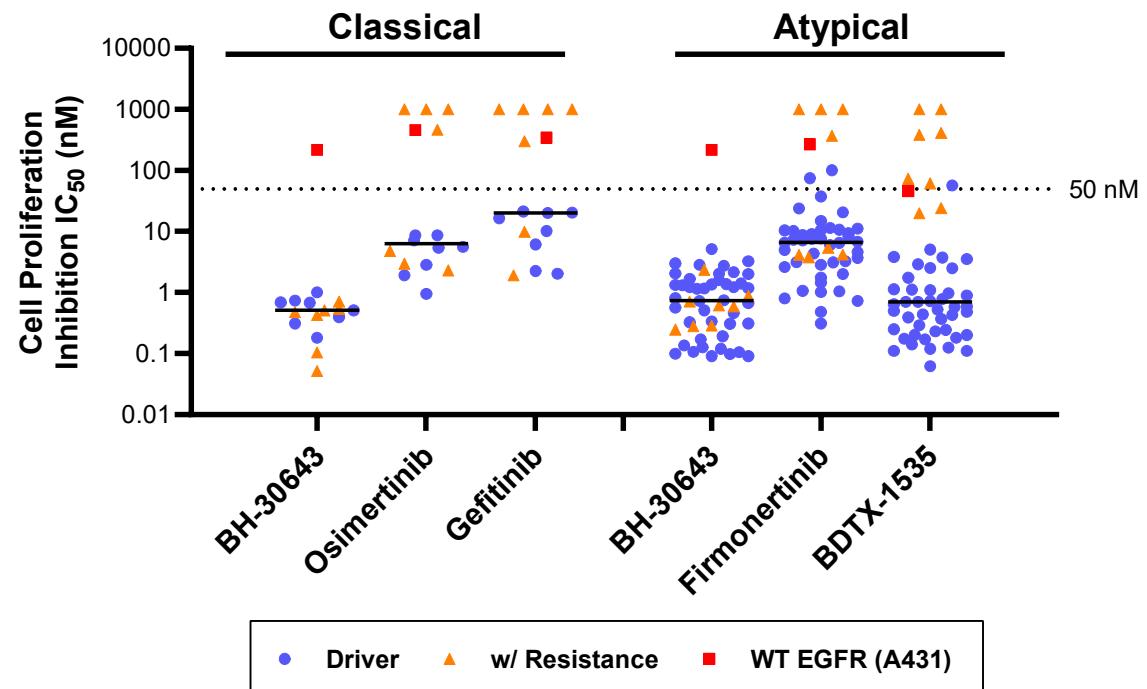
EGFR	1	●
HER2, TNIK	1-5 x	●
MINK, HGK, LRRK2	5-20 x	●



# Unique Design Overcomes the Limitations of Contemporary EGFR TKIs

## BH-30643 has 3 distinguishing features

1. Sub-nM potency against classical EGFR mutations: 10x more potent than osimertinib
2. Wide therapeutic window, avoiding wildtype inhibition and allowing tolerability at high exposures
3. The only molecule which can address both C797S **and** T790M resistance



Note: Classical mutations include ex19del, L858R; Atypical mutations include genetic alterations at G719, L861, S768, E709, L747, V834, L833, V769 and any compounds of these mutations with one another or with classical mutations; Resistance mutations include C797S, T790M. The bar in each group indicates grand median.

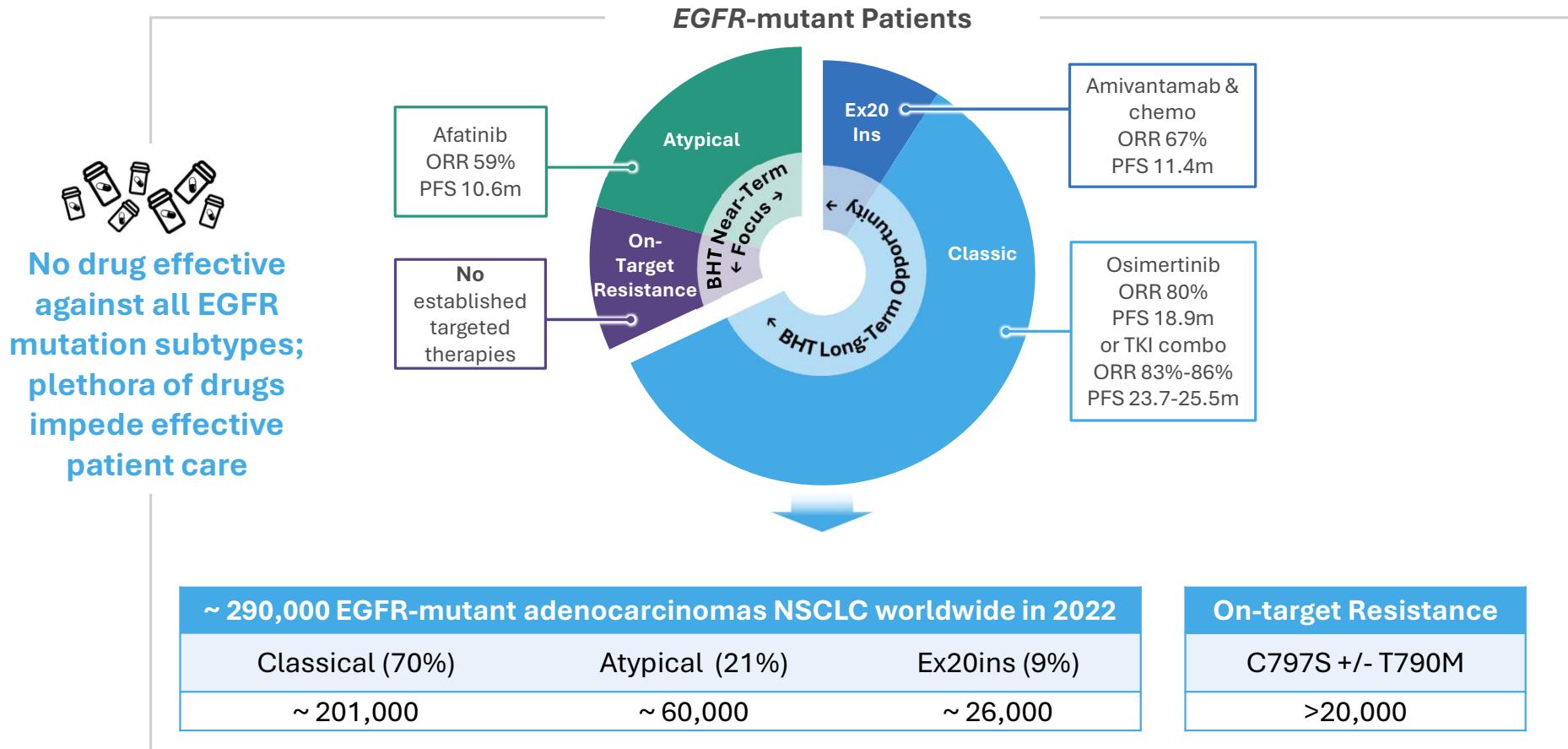


# **BH-30643: SOLARA Clinical Trial**

**Global Phase 1/2 study**



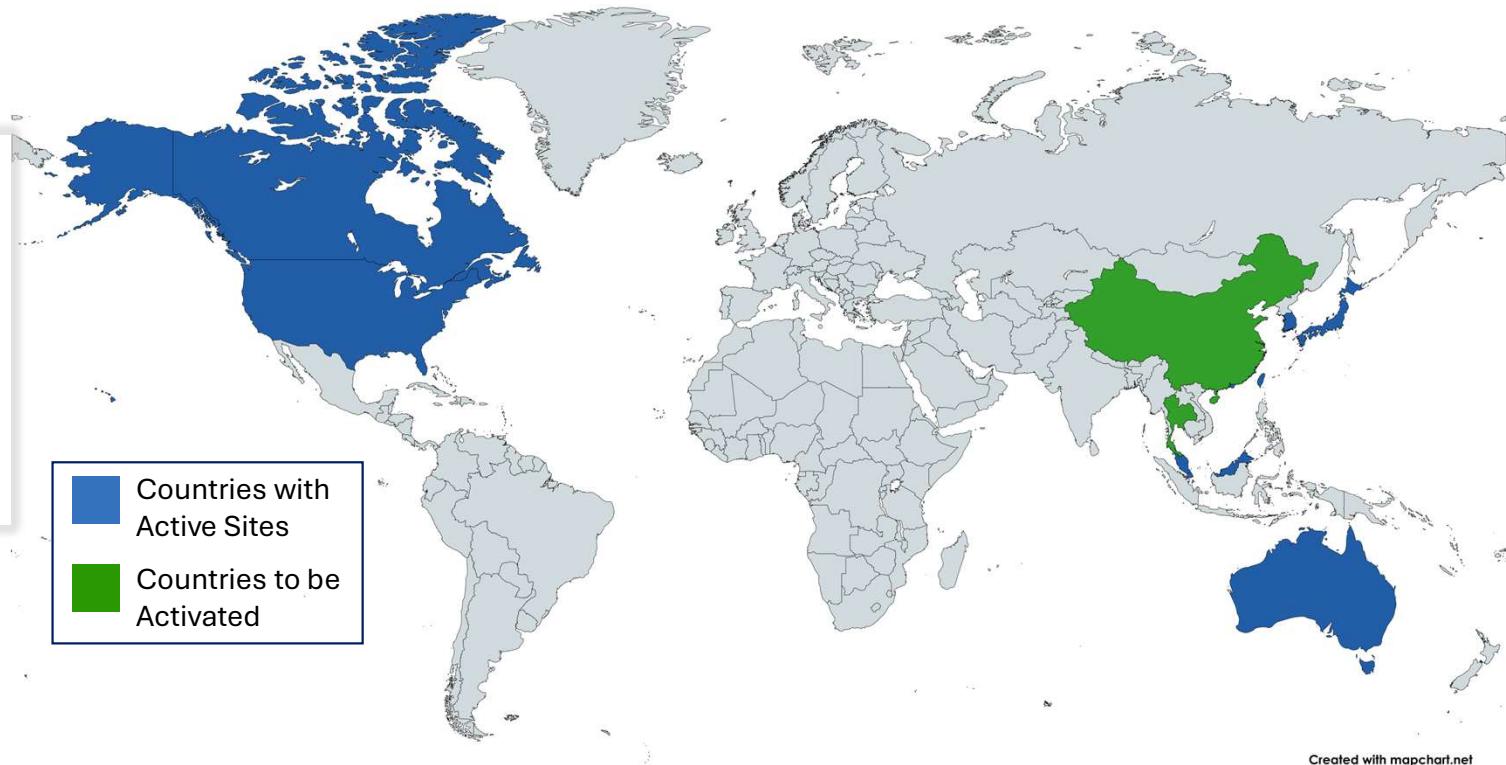
## EGFR-Mutant NSCLC Is a Prevalent Global Disease





## BH-30643-01 (SOLARA) Global Phase 1/2 Clinical Trial

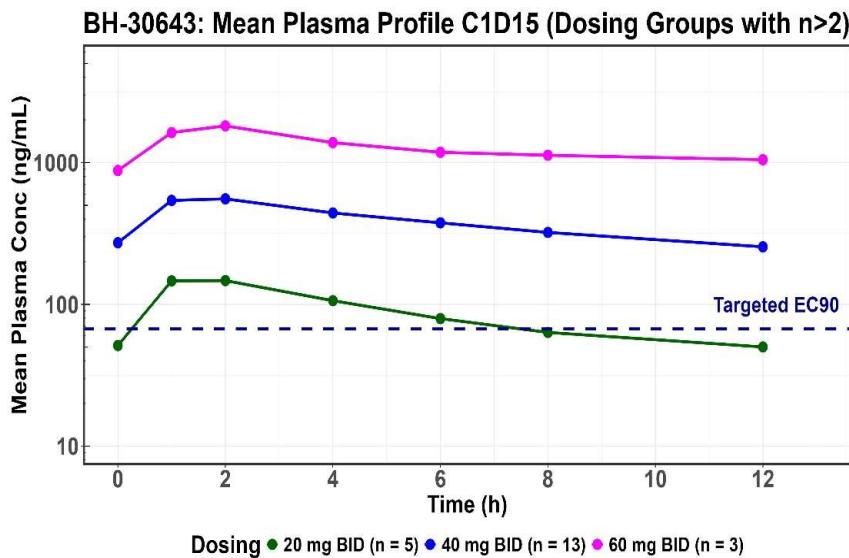
- Global Phase 1/2 trial
- Enrollment initiated January 2025
- Now enrolling at >30 sites in 9 countries



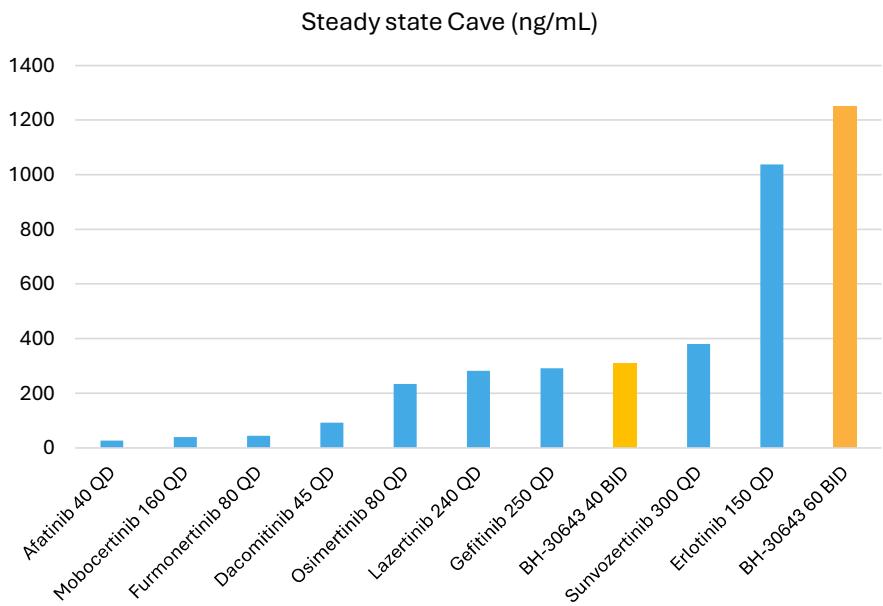


## BH-30643 PK Profile: High and Sustained Exposure

Exposures at doses  $\geq 40$  mg BID well exceed the target EC<sub>90</sub>



Exposures at candidate dose levels exceed those of many contemporary EGFR TKIs

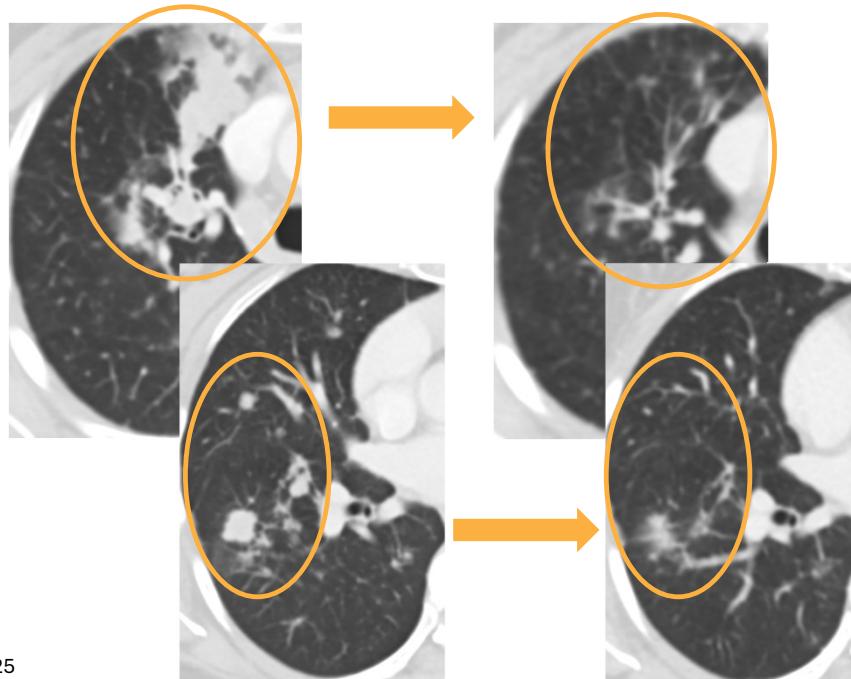


Paired with super-potency, high exposures could permit maximal EGFR inhibition

## Overcomes C797S Resistance Even in the Presence of T790M

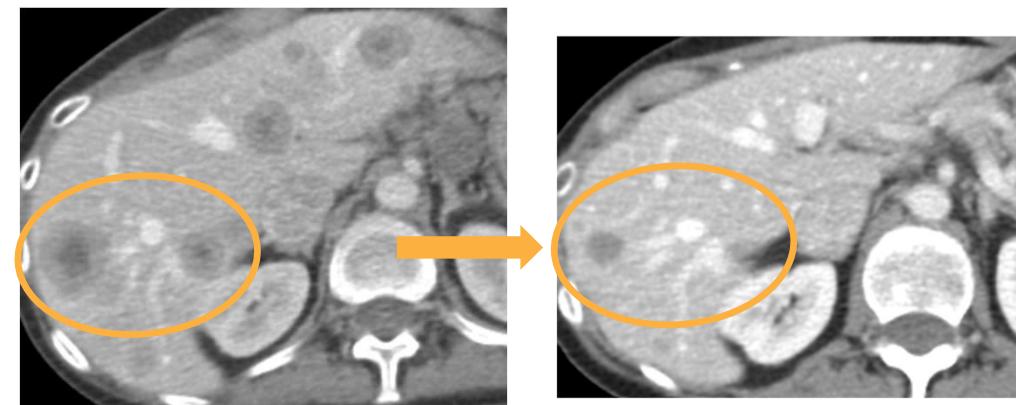
### C797S & exon 19 deletion

- 5 prior lines of therapy including osimertinib, amivantamab, and IO
- Partial response sustained on multiple scans, with therapy ongoing



### C797S & T790M & exon 19 deletion

- 8 prior lines of therapy including osimertinib, investigational TKI, EGFR/met ab, ADC, etc
- Partial response sustained on multiple scans, with therapy ongoing

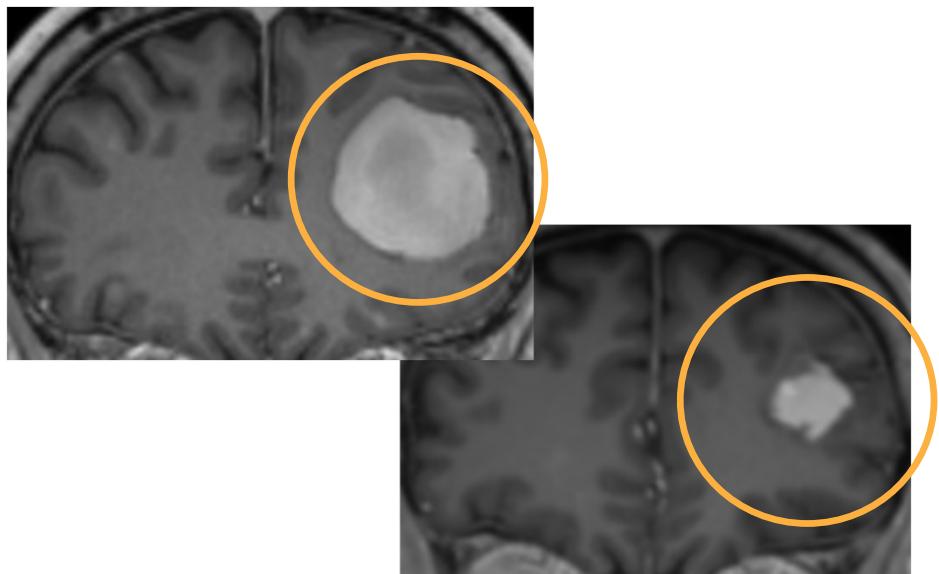




## Breadth of Activity Includes Brain Mets and Atypical EGFR Mutations

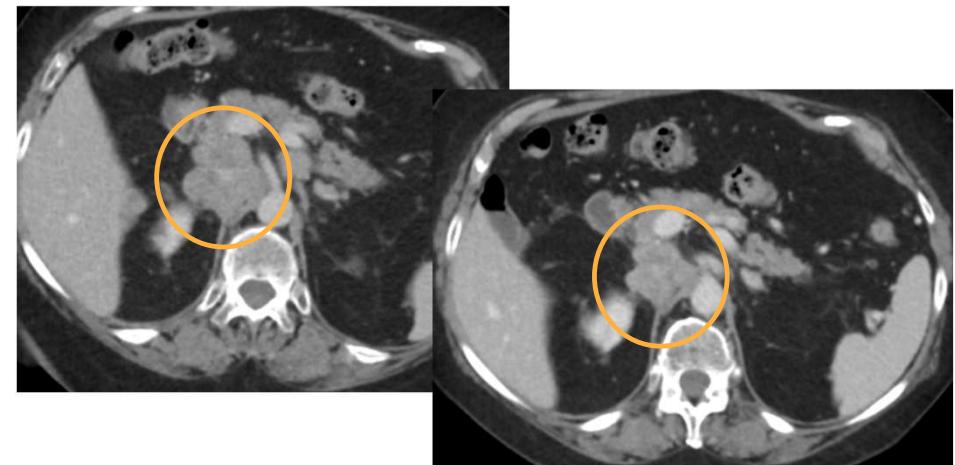
### Exon 20 insertion with brain metastases

- 3 prior lines of chemotherapy
- Partial response sustained on multiple scans, with therapy ongoing
- Sustained CNS improvement in the absence of radiation or steroids



### L861Q (atypical) after multiple TKIs

- 4 prior lines including platinum, erlotinib, osimertinib, and chemo/mobocertinib
- Partial response sustained on multiple scans, with therapy ongoing





## SOLARA Expansion Cohorts: Now Enrolling Across Multiple Dose Levels

Escalation &  
backfill



### 6 Expansion Cohorts (n ~20-40 each):

#### TKI Pretreated Cohorts

- 1) Classical mutation with C797S resistance
- 2) Atypical mutations after one prior TKI

#### TKI-Naive Cohorts

- 3) Classical mutations
- 4) Atypical mutations

#### Additional Cohorts

- 5) EGFR exon 20 insertions, up to 2 prior lines
- 6) HER2 mutations, up to 2 prior lines



Potentially  
registrational  
Phase 2

**Each cohort may study multiple doses**



# BH-30236: Macrocylic CLK Inhibitor

Novel macrocyclic, non-covalent, targeting aberrant alternative splicing



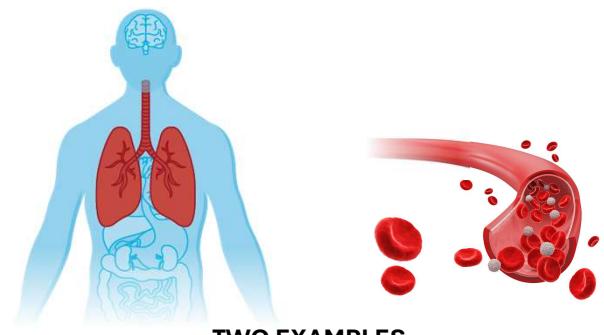
# Targeting Aberrant Alternative Splicing

## Importance of Alternative Splicing



- A single gene can produce multiple forms of a protein via alternative splicing
- Cancer cells can take advantage of this to make proteins that help them grow uncontrollably
- When alternative splicing becomes **dysregulated**, it can **lead to cancer progression and therapeutic resistance**

## Addressing Aberrant Alternative Splicing Extends the Targetable Proteome



### Lung Cancer

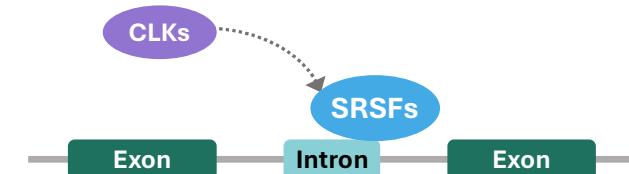
**SRSF1, SRSF6, NUMB, RBM5, RBM6, RBM10, U2AF1**, EGFR, MET, VEGFR, S6K1, MKNK2, AIMP2, BCL2L11, ENHA, KLK8, **DHX9**

### AML/Blood Cancers

**SF3B1, HNRNPK, p53, p21, Cebpa, CebpB, U2AF1, SRSF2, ZRSR2, PRPF8, SFPQ/PSF, DDX41, CD22, IKZF1, WT1, SMC1A**

**Red** = Spliceosome genes  
(mutated/aberrantly expressed)

## Importance of CLK



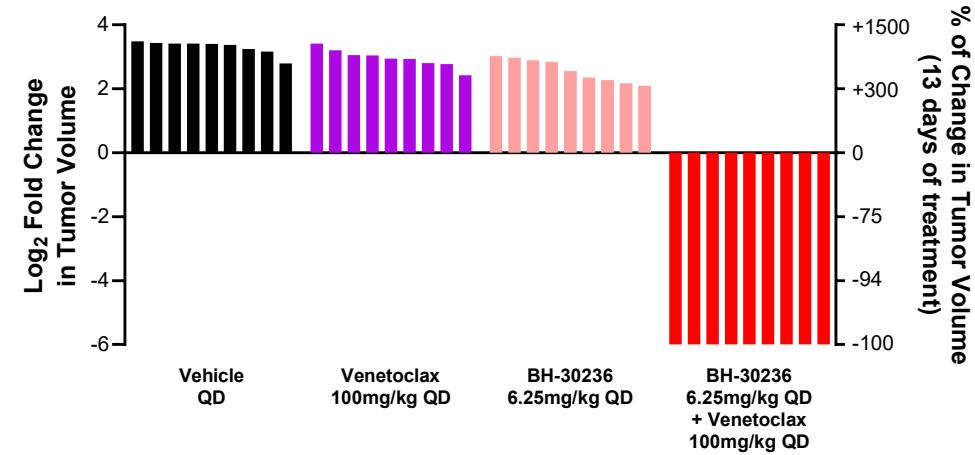
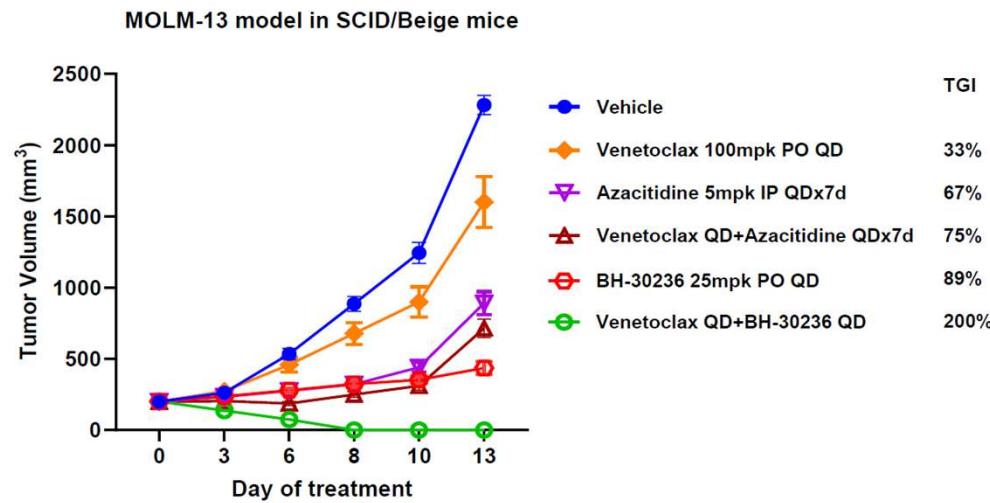
- CDC-like kinases (CLKs) can modulate aberrant splicing via phosphorylation of SRSF proteins
- Restoring normal splicing function is key to overcome off-target resistance in cancer
- Initial CLK inhibitors (eg. CTX-712) have demonstrated proof of concept



## Synergistic Effect of CLK Inhibition in AML Models

- Hematologic malignancies such as AML are especially dependent upon aberrant alternative splicing
- In AML models, CLK inhibition with BH-30236 could overcome venetoclax resistance, even when dosed at low dose levels (6.25 mg QD of BH-30236, equivalent to 30 mg QD in human)

Anti-tumor Activity of BH-30236 in Combination with Venetoclax in MOLM-13 CDX Tumors

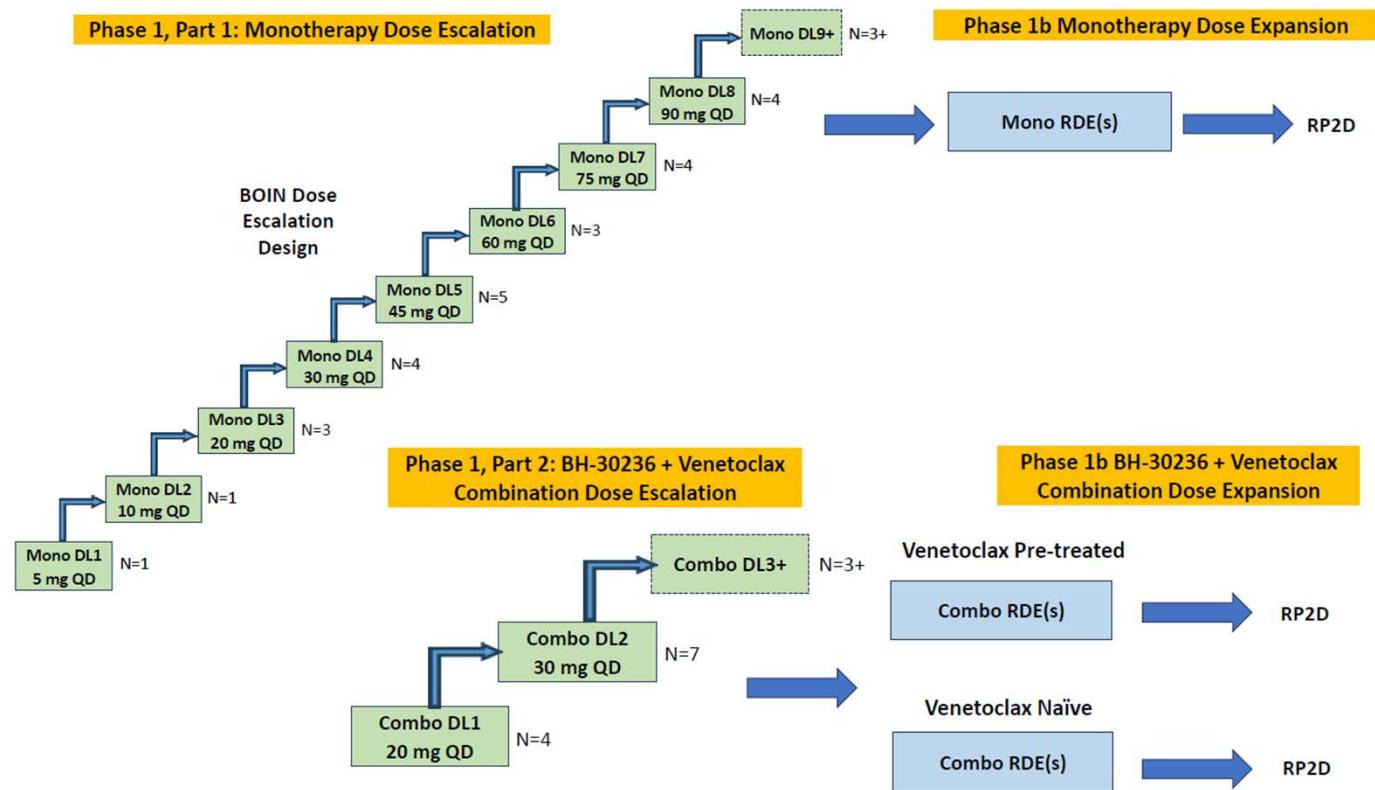




## BH-30236: On-going Phase 1 FIH Study in AML/HR-MDS

Objective: Evaluate safety & tolerability, identify doses for expansion

- Monotherapy dose escalation on-going across multiple US sites
- Now studying dose level 9, with no safety limitations seen with continuous daily dosing
- Combination dose escalation with venetoclax (target dose 400 mg daily) has now initiated



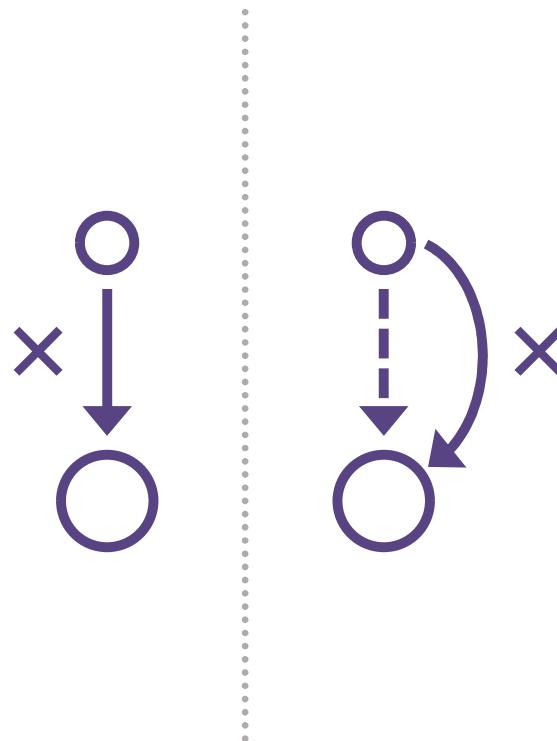


# Making a Transformational Leap in Cancer Therapy

# Intelligently Designed Molecules to Address the Challenges of Cancer Treatment Resistance

## BH-30643

- Novel, non-covalent, brain-active, macrocyclic OMNI-EGFR inhibitor
- Super-potency across broad spectrum of EGFR mutations with good selectivity over wild-type
- Favorable PK & safety profile
- Anti-tumor activity in resistant cancers including CNS activity
- SOLARA trial expansion cohorts are enrolling well in TKI-naïve and TKI-pretreated NSCLC



## BH-30236

- Potent macrocyclic CLK inhibitor
- Modulating splicing, DNA damage repair and apoptosis pathways
- Strong synergy with venetoclax in preclinical models
- No safety limitation with continuous daily dosing
- Combination study with venetoclax is actively enrolling

Clinical trial data readouts anticipated in 2026