

BlossomHill Therapeutics, Inc.

Corporate Presentation

September 2026
San Diego, CA



BLOSSOMHILL
THERAPEUTICS



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Caution should be exercised when interpreting results from separate trials or preclinical studies involving separate products and/or product candidates. Differences exist between clinical trial design and preclinical study design, patient populations used in clinical trials or factors related to a preclinical study, and the products and/or product candidates themselves, which limit the conclusions that can be drawn from comparisons across different trials or preclinical studies, as applicable. In addition, this presentation includes results from head-to-head studies against certain competitor product candidates that have not been approved by the FDA or other regulatory authorities and that were independently synthesized by, or on behalf of, the Company based on publicly available information rather than obtained directly from the originating companies, and such candidates may differ in formulation, manufacturing process, or purity from the version ultimately developed, tested or approved by their originators, such that the comparative data presented herein may not be representative of, and should not be relied upon as predictive of the actual safety, efficacy, or performance of those competitor candidates.

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Intentionally designing novel medicines to address significant unmet medical needs in cancer treatment

Strong leadership team
with proven track record of
designing & developing
FDA-approved cancer
medicines

**Early launch opportunity
targeting EGFR C797S**
with a convenient oral
monotherapy

**Broad EGFR development
ongoing** including TKI
naïve cohorts, potentially
offering substantial upside

**Differentiated wholly-
owned pipeline** spanning
novel mechanisms (CLK)
and novel scaffolds
(pan-KRAS)

Pipeline Assets with Multiple Near-term Anticipated Milestones

BH-30643 OMNI-EGFR™ Inhibitor

Q4 2026	EOP1 Meeting: C797S potential accelerated approval pathway
Q1 2027	First Patient Dosed in anticipated pivotal Phase 2 trial
1H 2027	Phase 1 Update: C797S durability
2H 2027	Phase 1 Update: TKI-naïve durability
2H 2027	Phase 1 Initial Data: chemo combo

BH-30236 Macrocytic CLK Inhibitor

1H 2027	Updated Phase 1 data on safety and anti-leukemic effect
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BH-501284 Pseudo-irreversible Pan-KRAS Inhibitor

Q1 2027	IND submission
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Financial / Corporate Overview

Ticker	BLSM
Cash (end of Q2 2026)	\$95.4M
IPO Net Proceeds (August 2026)	\$151.7M
Shares Outstanding	31.8M
Estimated Cash Runway	Q2 2028



Seasoned Management Team and Board

Executive Team

Experience



J. Jean Cui, Ph.D.

Scientific Founder, President & CEO
Board of Directors



Y. Peter Li, Ph.D., M.B.A.

Co-Founder, Executive Chairman
Board of Directors



Geoffrey Oxnard, M.D.

Chief Medical Officer and EVP



Jason Keyes, M.B.A.

Chief Financial Officer and EVP



Vincent P. Liptak, JD, Ph.D., M.B.A.

General Counsel and EVP



Board of Directors



Bihua Chen, M.B.A.



CORMORANT ASSET MANAGEMENT



Carl L. Gordon, Ph.D.

orbimed



John Schmid, M.B.A.

Independent Board Member



Sheila Gujrathi, M.D.

Independent Board Member



Sundeep Agrawal, M.D.



BRAHMA
CAPITAL



Designing Cancer Medicines the BlossomHill Way



Identify unmet medical need for patients



Structural analysis of existing molecules and their limitations



Precision drug design based on deep understanding of target biology and protein structure dynamics



Iterate and optimize toward specified performance objectives



**Our goal is to develop orally available,
potent precision medicines that maximize patient benefit**



BlossomHill

Product Candidate Pipeline

Target	Candidate	Indication / Therapeutic Area	Design/ Discovery	IND Enabling	Phase 1	Phase 2	Phase 3
EGFR	BH-30643	EGFR-mutant NSCLC					
CLK	BH-30236 <i>+/- venetoclax</i>	R/R AML and HR-MDS					
Pan-KRAS	BH-501284	Solid Tumors					

**Our goal is to develop orally available,
potent precision medicines that maximize patient benefit**



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BH-30643: OMNI-EGFR™ Inhibitor

INVESTIGATIONAL, MACROCYCLIC, NON-COVALENT, BRAIN ACTIVE INHIBITOR
TARGETING MUTANT EGFR ACTIVE CONFORMATION



Targeting *EGFR*-Mutant NSCLC: C797S Resistance And Beyond



Total *EGFR*-mutant NSCLC incidence in 2025
USA, Europe, Japan, China

316,000¹

209,000

Total Stage III-IV (advanced/metastatic)
USA, Europe, Japan, China

125,000

Treated with 3rd Gen TKI²: USA ~80%;
ex-US ~50-60%

16,000

~13% develop C797S
on target resistance mutation³

Substantial Market Opportunity in *EGFR* Mutant NSCLC

(2025 Tagrisso sales: ~\$7.3B)⁴

Early launch opportunity:

- ~5,400⁵ C797S patients annually across planned accelerated approval markets (US: ~3,200, Japan: ~2,200)
- Osimertinib intolerance (~11-13%)⁵ represents an additional near-term unmet medical need
- An effective TKI has potential for premium pricing and prolonged treatment duration compared to current SOC

Growth opportunity:

- Unmet need for a novel TKI that offers prolonged effect in 1L with safety profile that allows combination development

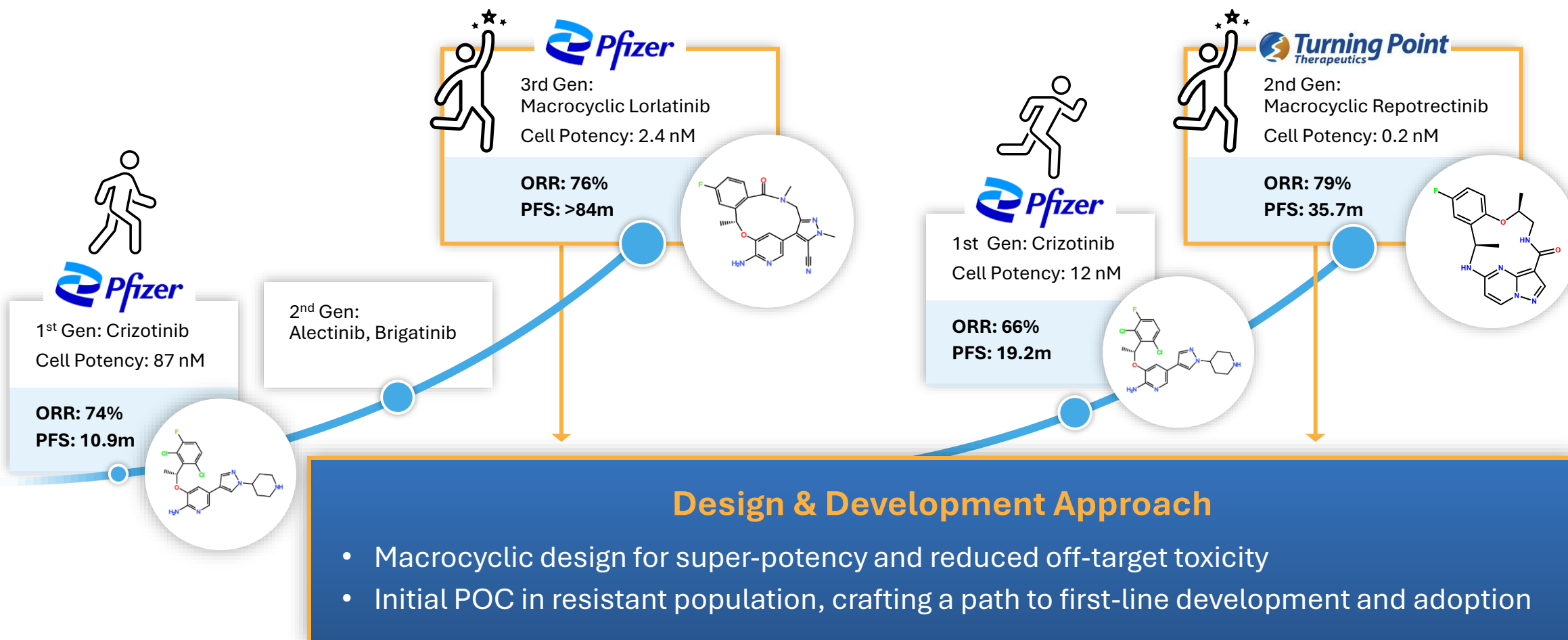


Why a Leap in *EGFR*-mutant NSCLC Is Attainable

ACHIEVING LONGER PFS AND PRE-EMPTION OF RESISTANCE WITH HIGH-POTENCY

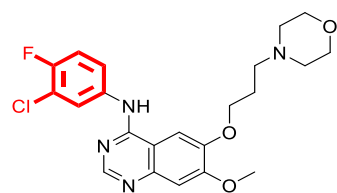
EML4-ALK Inhibitors in *ALK*⁺ NSCLC

ROS1 Inhibitor in *ROS1*⁺ NSCLC

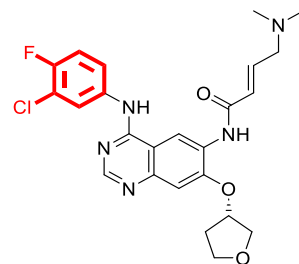


Existing EGFR Tyrosine Kinase Inhibitors Share Structural Families and Design Liabilities

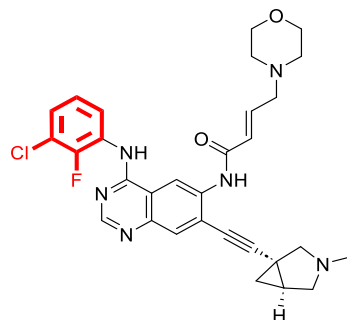
Many TKI designs use a **back-pocket motif** for potency, vulnerable to **T790M** resistance



Gefitinib



Afatinib

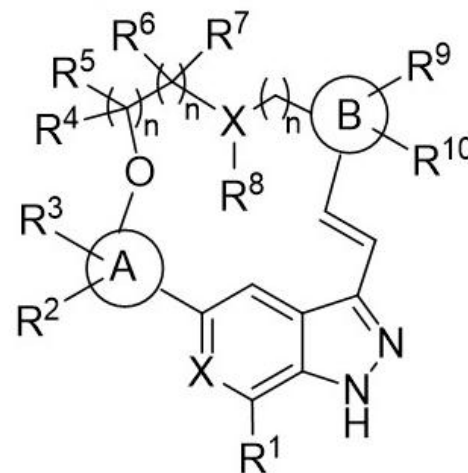


BDTX-1535

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

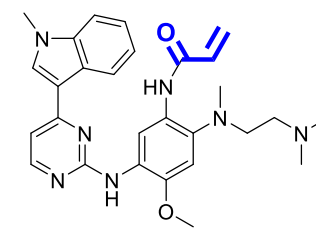
Gefitinib	Afatinib	STX-721
Erlotinib	Dacomitinib	Zipalertinib
Icotinib	BDTX-1535	Enozertinib

BH-30643

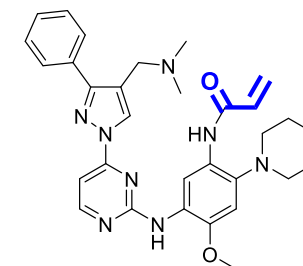


BH-30643 leverages an entirely novel macrocyclic scaffold to uniquely address both T790M and C797S resistance

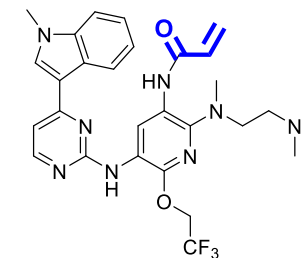
Other TKI designs use a **covalent bond** for potency, vulnerable to **C797S** resistance



Osimertinib



Lazertinib



Firmonertinib

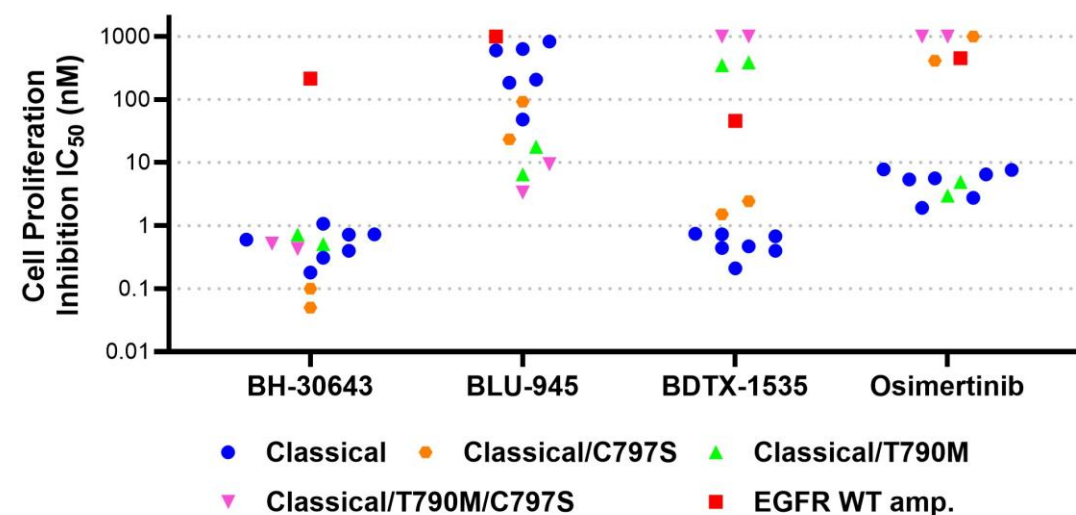
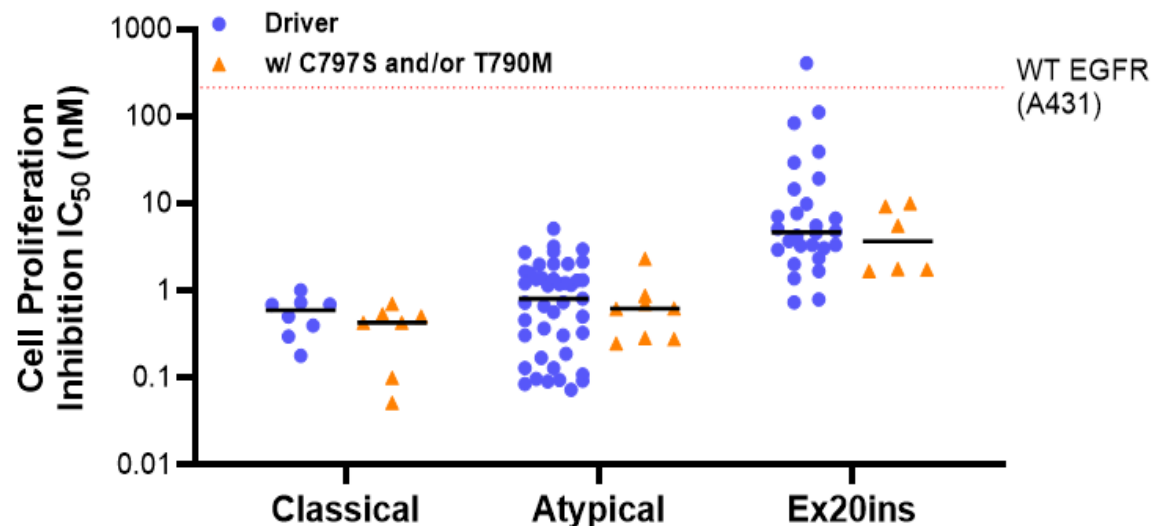
Other TKIs **covalently bind** for potency

Osimertinib	Almonertinib	Mobocertinib
Rociletinib	Aumolertinib	Sunvozertinib
Lazertinib	Olmotinib	Firmonertinib

BH-30643: An Intentionally Designed Macrocyclic, Mutant-Selective OMNI-EGFR™ Inhibitor



- High cell potency against *EGFR* classical, atypical, and exon20ins
- Equipotent in the setting of on-target resistance mutations
- ~ 10 times more potent than osimertinib for L858R / 19del
- Avoided potency limitations of recent TKIs designed to target resistance



Note: Classical: L858R and ex19del; Any IC₅₀ value > 1000 nM was plotted as 1000 nM.



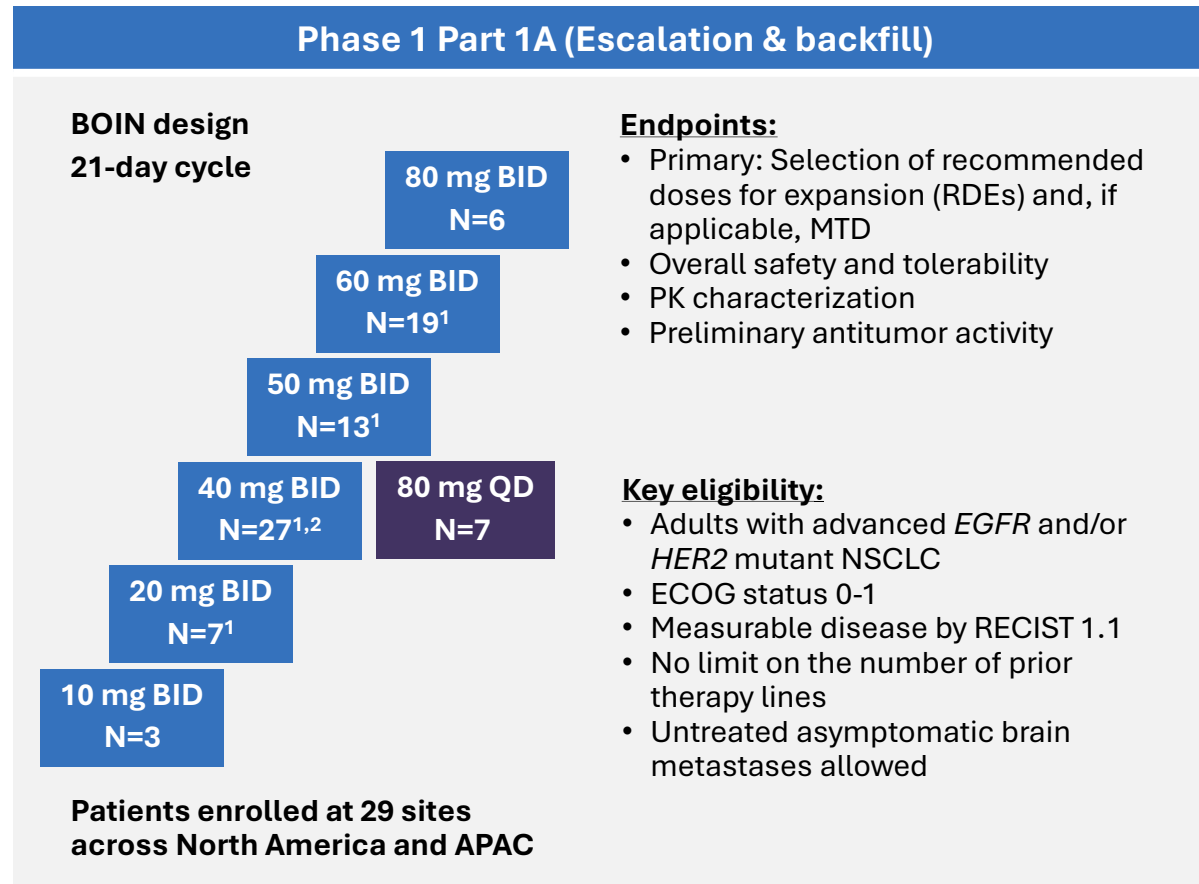
BH-30643 SOLARA Phase 1/2 Trial

PRELIMINARY DOSE ESCALATION & EXPANSION EXPERIENCE

SOLARA (NCT06706076):

Study Design and Patient Characteristics for Dose Escalation

Phase 1 escalation / backfill enrolled a heavily pretreated and heterogeneous patient population, with a median of 3 prior lines of therapy, 66% with history of brain metastases, and 71% ECOG=1



Patients Baseline Characteristics	N=82 ³
Age (years), median (min, max)	61 (31, 81)
ECOG = 1, n (%)	58 (71%)
History of brain metastases, n (%)	54 (66%)
Prior lines of therapy, median (min, max)	3 (1, 12)
Prior 3rd Gen EGFR TKI	63 (79%)
Other prior targeted therapy	17 (21%)
Prior chemotherapy and/or ADC	58 (71%)
Prior immunotherapy	14 (17%)
EGFR or HER2 driver mutation⁴, n (%):	
Classical <i>EGFR</i> (19del or indel, L858R)	54 (66%)
<i>EGFR</i> exon 20 insertion	16 (20%)
Atypical / other <i>EGFR</i>	10 (12%)
<i>HER2</i> mutation	2 (2%)
Multiple <i>EGFR</i> mutations⁴, n (%):	51 (62%)

¹Backfill cohorts enrolled at potentially efficacious doses based on PK and/or anti-tumor activity.

²Includes patients enrolled into pilot food effect sub study at 40mg BID

³All data as of 2 March 2026 data cut, with efficacy follow-up through 29 April 2026 data cut

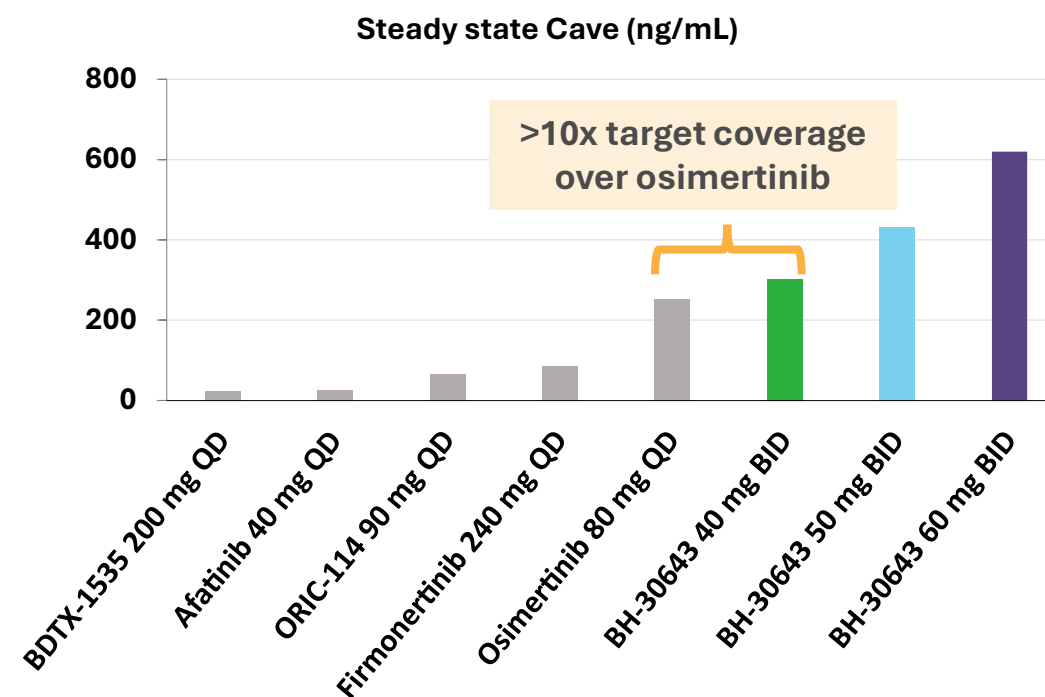
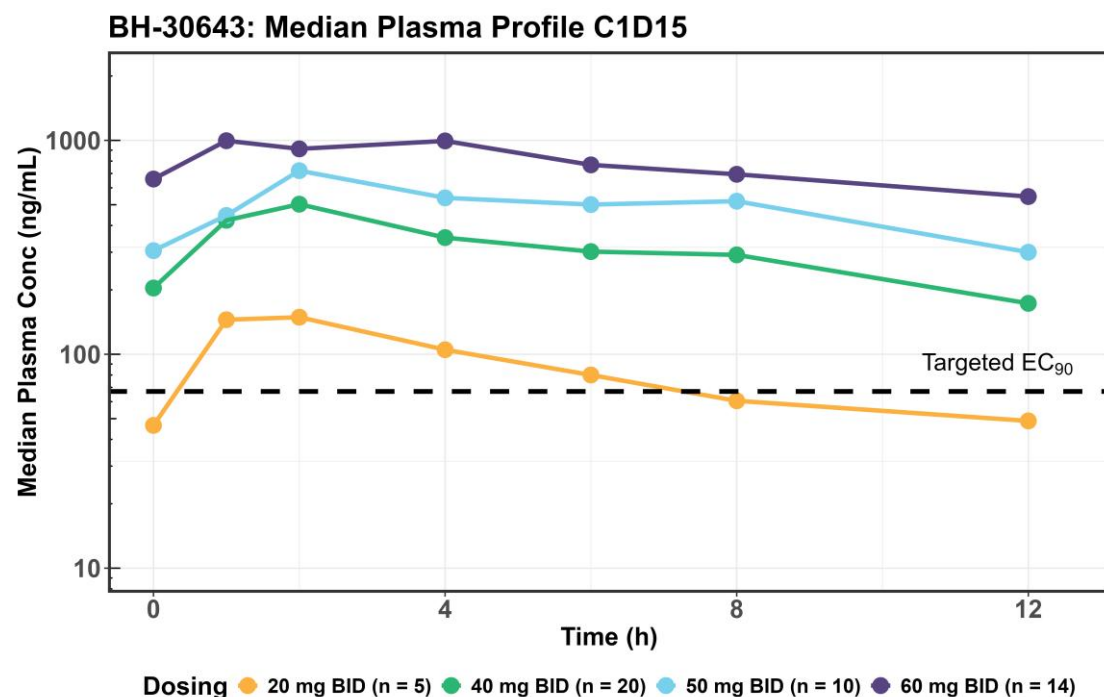
⁴Includes historical and baseline molecular results



High PK Exposures Observed in Escalation / Backfill Experience

Median preliminary steady state plasma exposures at ≥ 40 mg BID achieved high coverage over target EC_{90} and exceeded reported exposures of many contemporary EGFR TKIs

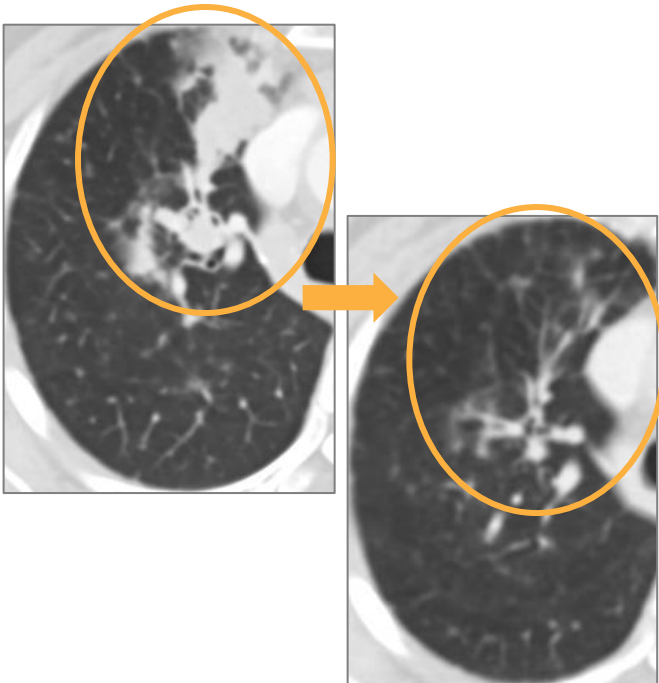
High exposure and high potency combined with $K_{p,uu}$ of 27% (CSF, primate) supports the observed preclinical and clinical CNS activity



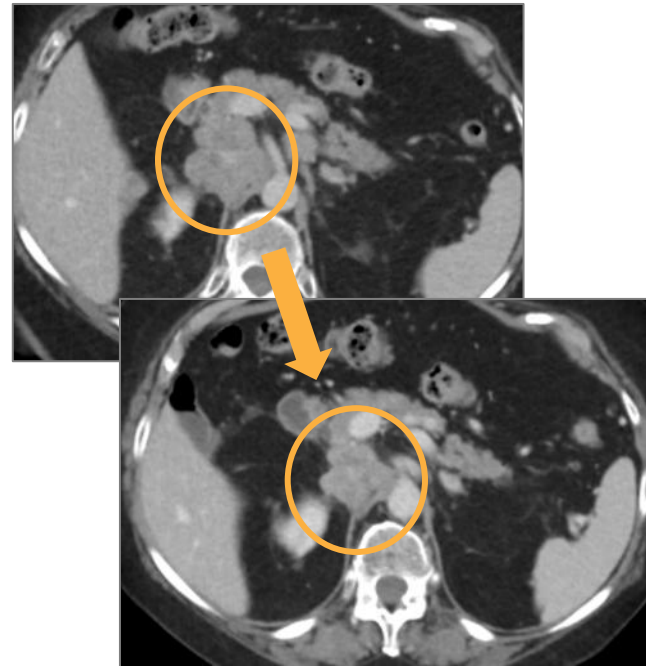
Note: The clinical data presented above are based on cross-study comparisons and are not based on any head-to-head clinical trials. Differences exist between trial designs, patient characteristics and other factors and caution should be exercised in drawing any conclusions from a comparison of the data across studies as cross study comparisons are inherently limited and such data may not be directly comparable.

Responses in Diverse *EGFR*-mutant Subtypes May Support Multiple Paths for Future Clinical Development

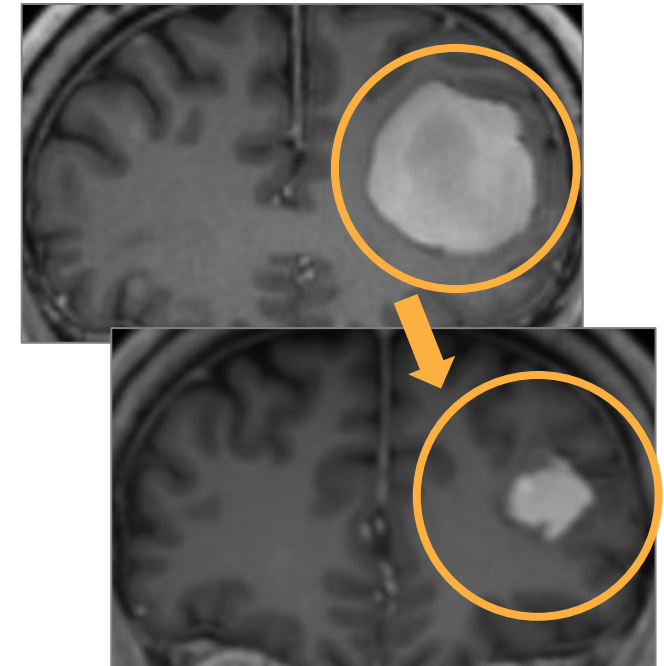
- 44 yo female, enrolled at 40mg BID
- **EGFR exon 19 deletion & C797S** detected after prior osimertinib, amivantamab and immunotherapy
- >11 months on treatment



- 68 yo female enrolled at 80mg QD
- **EGFR L861Q** previously treated with chemotherapy, erlotinib, osimertinib, mobocertinib
- 9 months on treatment



- 51 yo male enrolled at 80mg BID
- **EGFR exon 20 insertion** with 3 prior lines of chemotherapy; no brain radiation or steroids
- 8 months on treatment





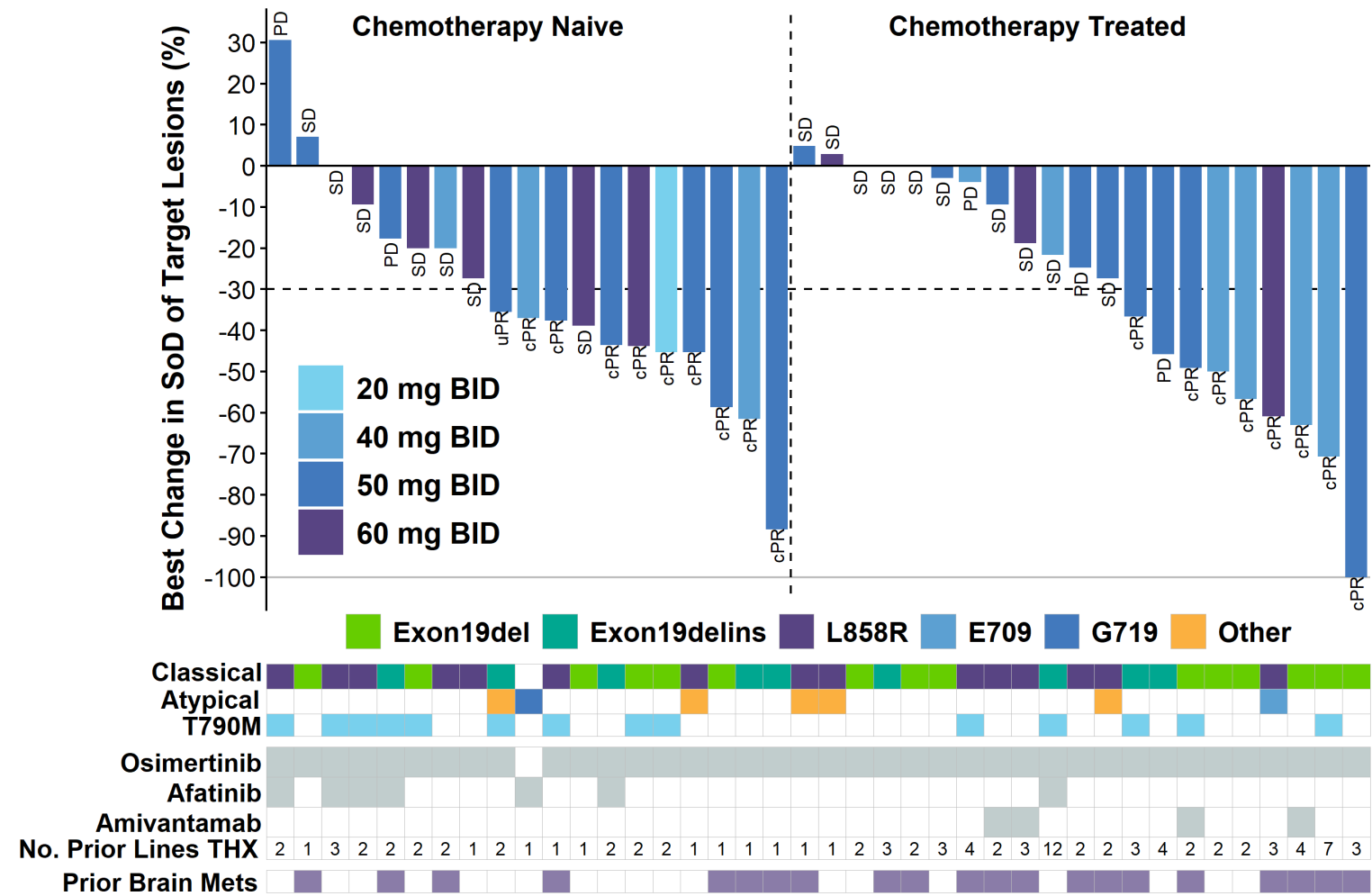
BH-30643 Experience in C797S +/- T790M Resistance

PRELIMINARY DOSE ESCALATION / BACKFILL AND EARLY EXPANSION EXPERIENCE



C797S Efficacy Results: Confirmed Responses Observed Across Dose Levels in Patients with C797S

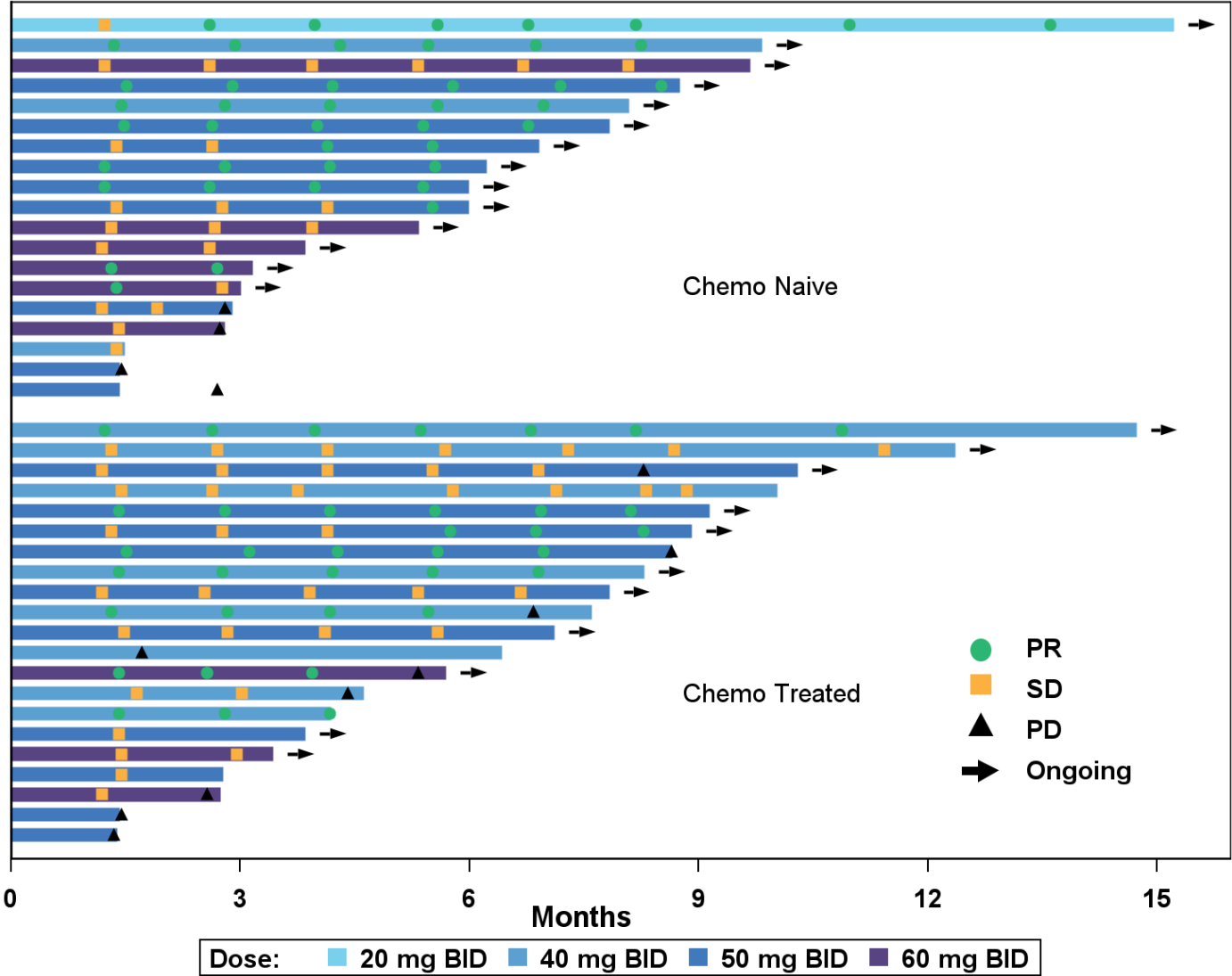
- 40 patients from the target C797S population (19 chemo naïve and 21 chemo treated) were response evaluable across escalation and expansion cohorts
- Responses (45% ORR with 17 confirmed and 1 unconfirmed and ongoing) seen both without prior chemo (53% ORR) and with prior chemo (38% ORR)
- Responses observed with or without concurrent T790M and with or without prior history of brain metastases



One patient enrolled at 20mg BID was escalated to 40mg BID after second cycle. Target C797S population excludes patients with concurrent driver alterations or who previously received EGFR TKI specifically targeting a known C797S. One patient discontinued treatment due to AE on day 6 and is not included in efficacy analysis set

C797S Efficacy Results: Preliminary Durability

- Studied 40 patients in the target C797S population enrolled as of the data cutoff
 - Excludes patients with concurrent driver alterations or who previously received EGFR TKI specifically targeting a known C797S
- Median follow-up was 6.9 months
- 25 patients (63%) remain on therapy with 3 patients exceeding 12-month treatment and on-going



Updated analysis using 5/12/26 formal data cut obtained for regulatory purposes, with efficacy follow-up through 8/10/26. Compared to the 5/20/26 data snapshot, this excludes one patient who had not yet enrolled, one patient who discontinued treatment due to AE on day 6 and is not included in efficacy analysis set, and two patients found to have concurrent driver alterations.

Maturing Safety Data Supports RP2D Selection

Treatment-related adverse events (TRAE) in $\geq 10\%$ patients¹

Preferred Term, N (%)	40 mg, 50mg, 60 mg BID N=174			
	All Grade	Grade 1	Grade 2	Grade 3/4 ³
Any TRAEs	155 (89)	65 (37)	52 (30)	38 (22)
Bilirubin elevation²	78 (45)	25 (14)	40 (23)	13 (7)
Rash²	75 (43)	60 (34)	11 (6)	4 (2)
Diarrhea	67 (39)	61 (35)	4 (2)	2 (1)
Dry skin	38 (22)	37 (21)	1 (1)	0 (0)
Stomatitis²	37 (21)	28 (16)	6 (3)	3 (2)
ALT increased	30 (17)	11 (6)	6 (3)	13 (7)
AST increased	29 (17)	11 (6)	10 (6)	8 (5)
Fatigue	25 (14)	19 (11)	6 (3)	0 (0)
Nausea	20 (11)	13 (7)	7 (4)	0 (0)
Paronychia	20 (11)	8 (5)	11 (6)	1 (1)

- Dose reduction (9%) and treatment discontinuation (3%) due to TRAE are uncommon
- EGFR-wildtype associated TRAEs are mostly Grade 1
- Most common TRAE is bilirubin elevation:
 - “Gilbert’s-like” pattern: usually asymptomatic and predominately unconjugated due to UGT1A1 inhibition by BH-30643
 - Exposure dependent: Reversible with dose modification or often tolerated without dose adjustment
- TRAE of Grade 3+ ALT / AST increase in 7% / 5% of patients, respectively, with no cases of Hy’s law
- Rare pneumonitis (~1%)
- No clinically significant treatment-related cardiac effects or QTc prolongation

¹12 May 2026 cut-off. Median duration of exposure of 3 months

²Grouped term

³No Grade 5 TRAE



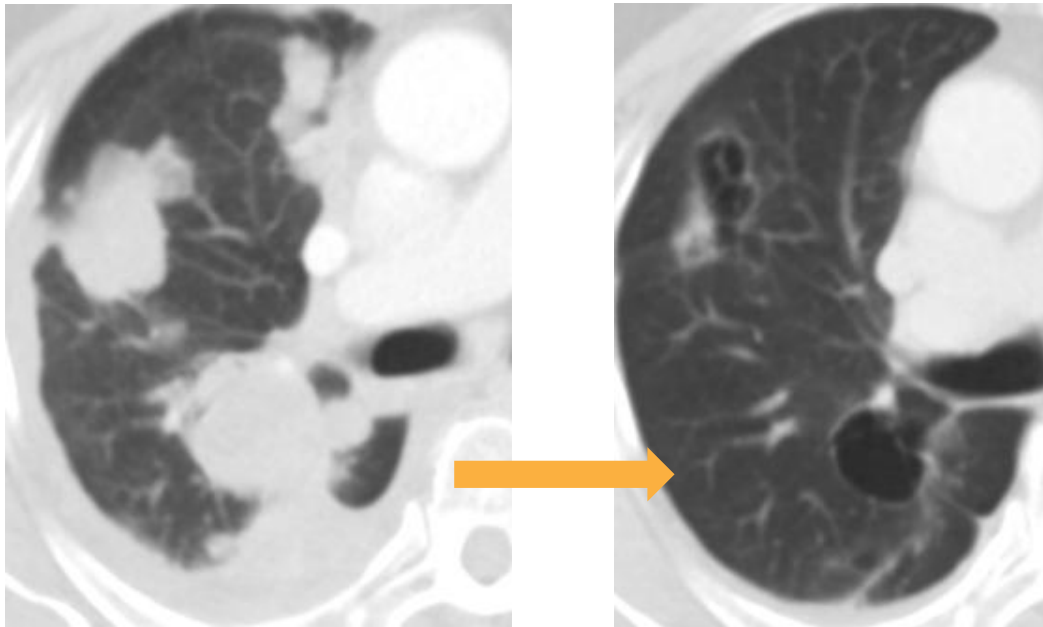
BH-30643 Experience in EGFR TKI Naive Classical Mutation

PRELIMINARY EARLY EXPANSION EXPERIENCE

First-line Activity Against Challenging EGFR Mutations

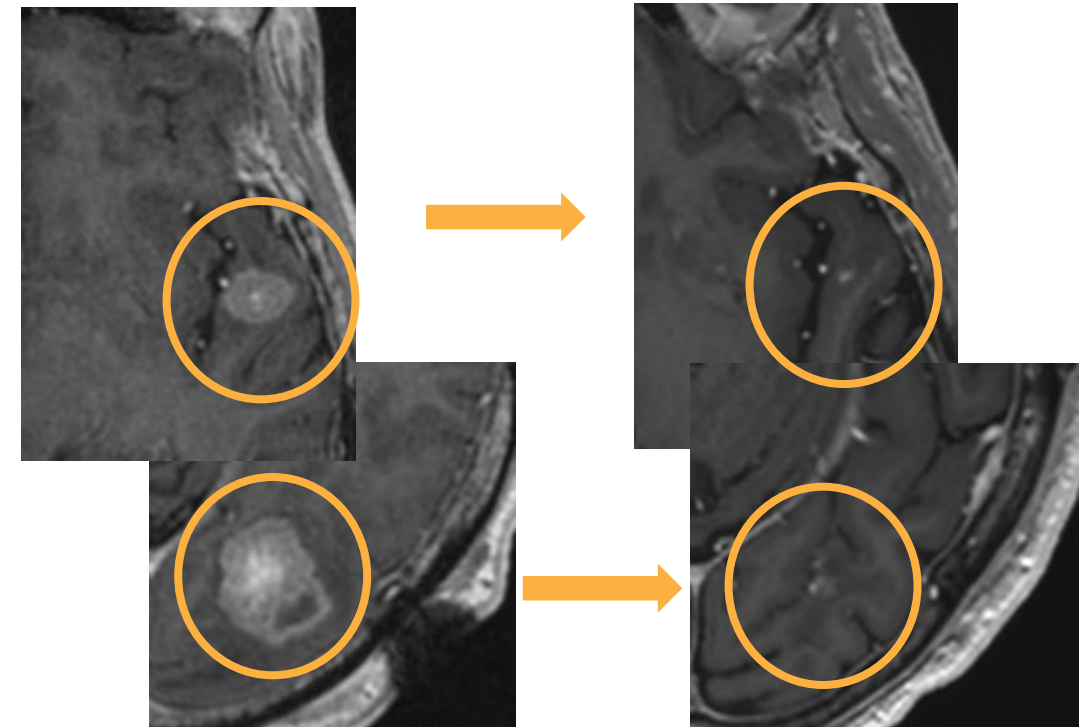
L858R

- Treatment naive, treated at 40mg BID, with treatment ongoing
- Partial response confirmed on second scan (-86%)



Exon19del & H870R

- TKI naive, presented with multiple brain mets
- Treated at 40mg BID with systemic and CNS PR, in the absence of radiation / steroids, with therapy ongoing

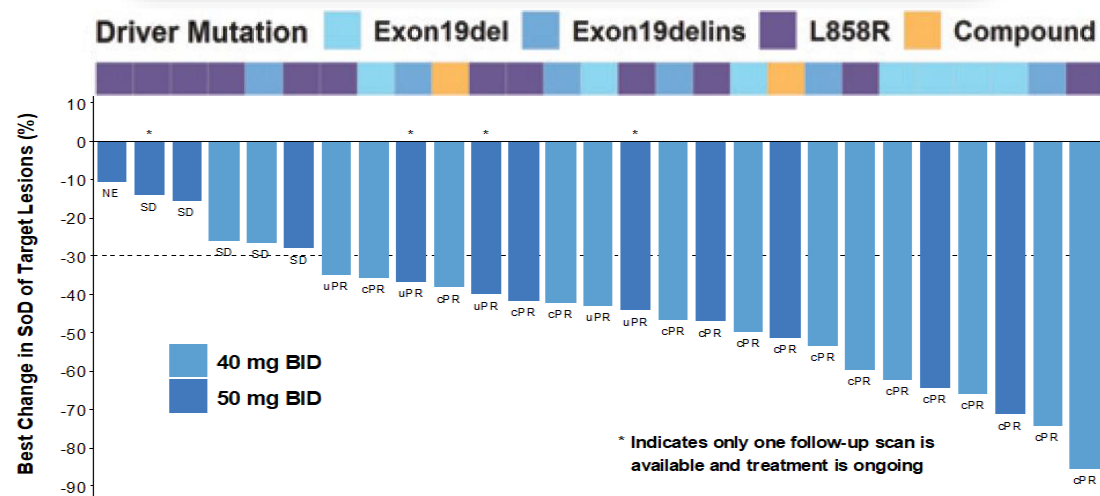




Early Clinical Activity of BH-30643 in Classical TKI-naïve Cohort

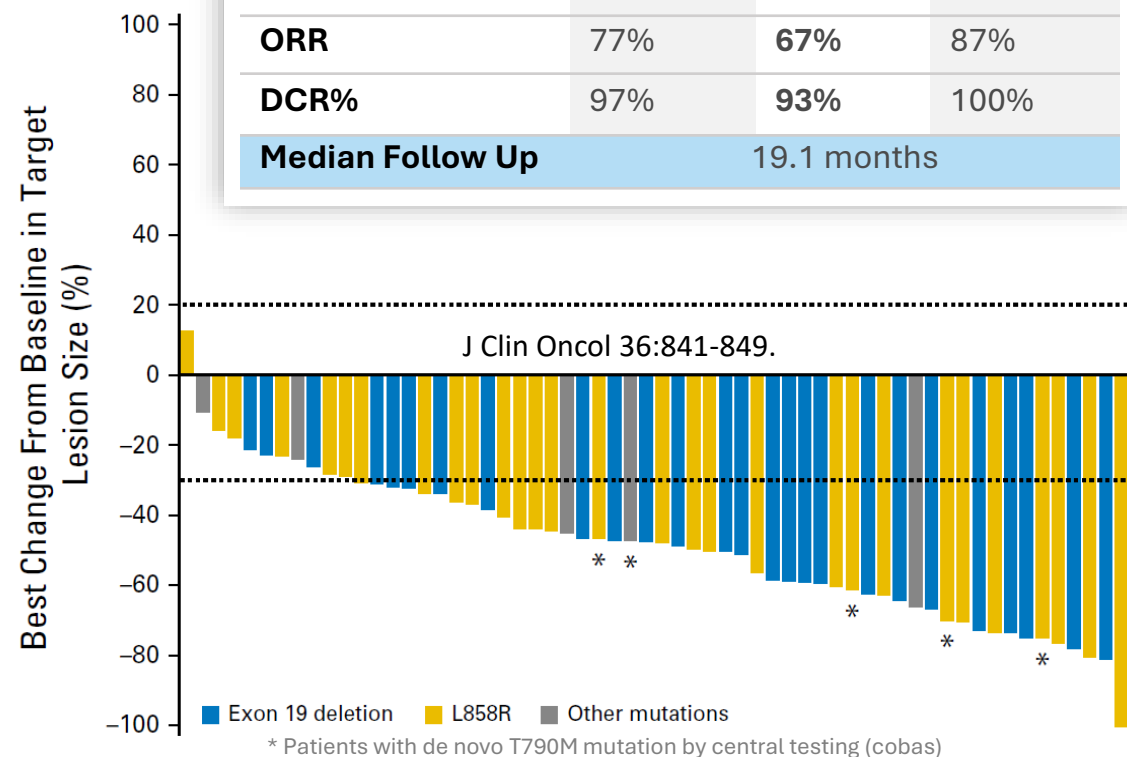
BH-30643 TKI-naïve

Total (40mg+50mg BID)	
# Patients	27
Brain mets n (%)	13 (48%)
ORR	78%
DCR%	96%
Median Follow Up	4.2 months



Osimertinib Phase 1 Treatment-naïve

	Total	80 mg	160 mg
# Patients	60	30	30
Brain mets n (%)	15 (25%)	7 (23%)	8 (27%)
ORR	77%	67%	87%
DCR%	97%	93%	100%
Median Follow Up	19.1 months		





SOLARA Expansion Cohorts Now Enrolling Globally

Expansion cohorts to inform Phase 2/3 development

**Escalation
and backfill**



TKI resistance

- C797S detected after most recent therapy
- Atypical resistance mutations (L718X, G724X, etc.)

Targeted therapy naive

- Classical mutations
- Atypical mutations (G719X, L861Q, S768I)
- Exon 20 insertions

Combination

- With platinum/pemetrexed following progression on 3rd-gen EGFR TKI



**Potentially
registrational
Phase 2**



BH-30643

Key Takeaways

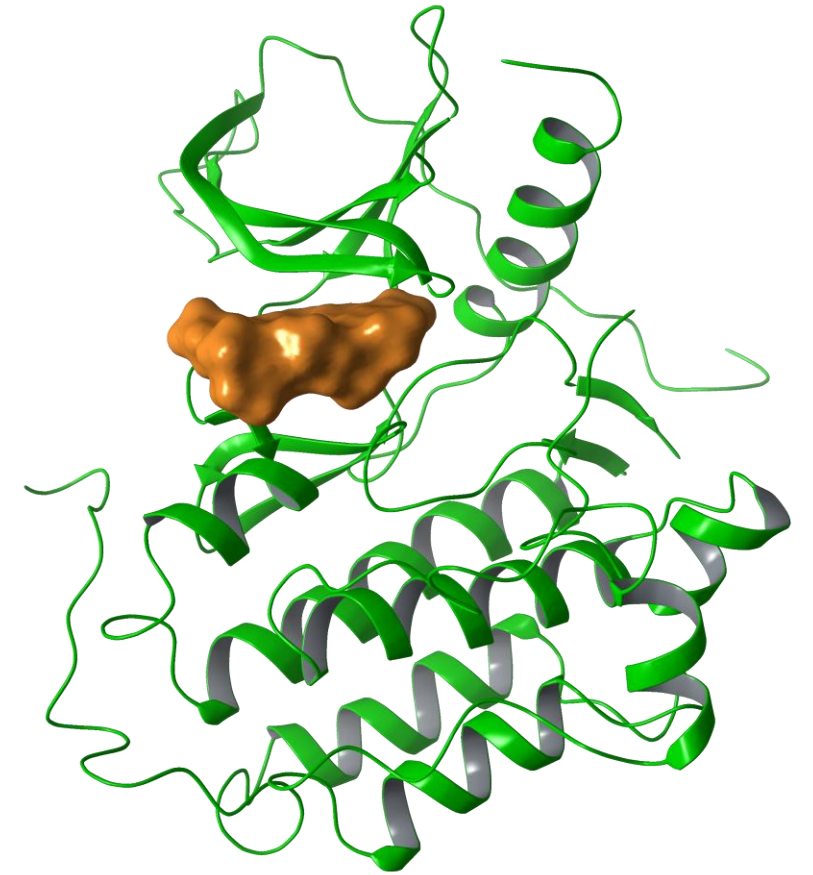
Compelling activity in patients with C797S with potential path to accelerated approval

Early activity in TKI naive patients with potential to expand 1L development

Differentiated safety profile mitigating many of the idiopathic toxicities seen with osimertinib

EOP1 meeting planned for Q4 2026

to enable seamless transition to potentially registrational Phase 2 trial in C797S-positive resistance, with first patient expected to be dosed in Q1 2027





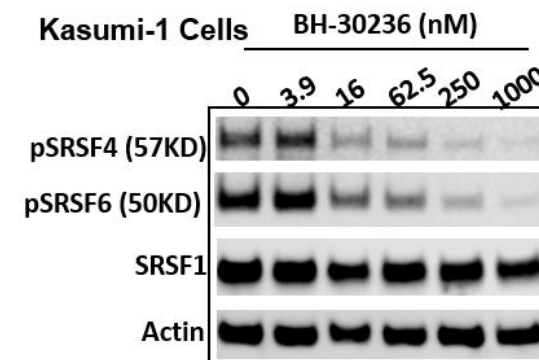
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BH-30236: CLK Inhibitor

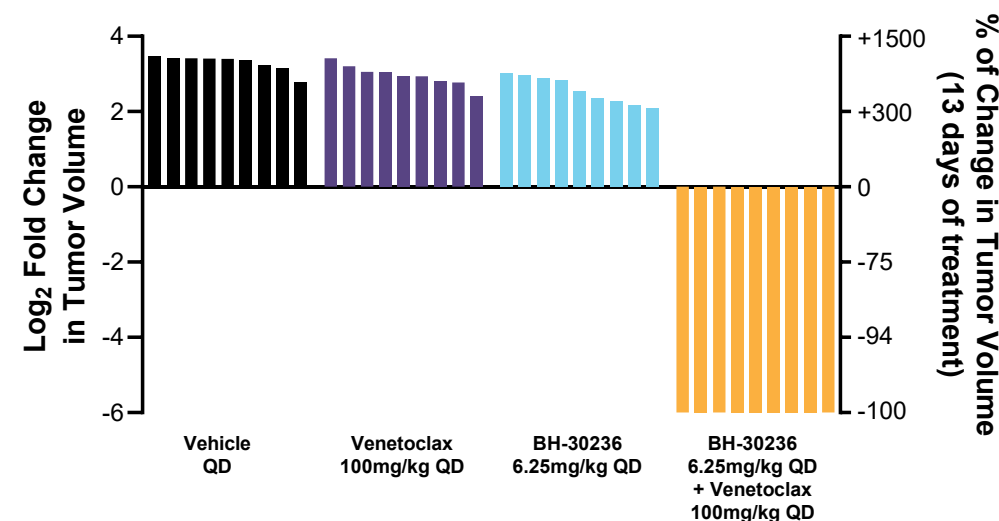
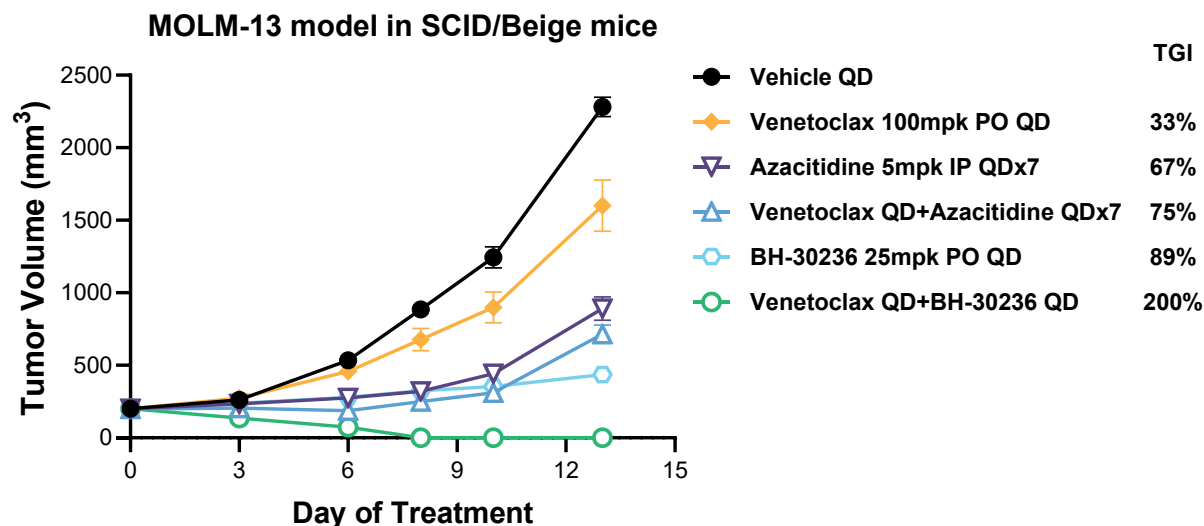
INVESTIGATIONAL, MACROCYCLIC, NON-COVALENT INHIBITOR
TARGETING ABERRANT ALTERNATIVE SPLICING

BH-30236: Potent CLK inhibitor Modulating Aberrant Alternative Splicing

- Upregulation and mutations of serine/arginine rich splicing factors (SRSFs) have been reported in hematological malignancies and solid tumors
- CDC-like kinases (CLKs) can modulate aberrant splicing via phosphorylation of SRSF proteins
- BH-30643 is a potent CLK inhibitor to inhibit pSRSF leading to modulate aberrant alternative splicing and demonstrated strong synergy with venetoclax in AML models



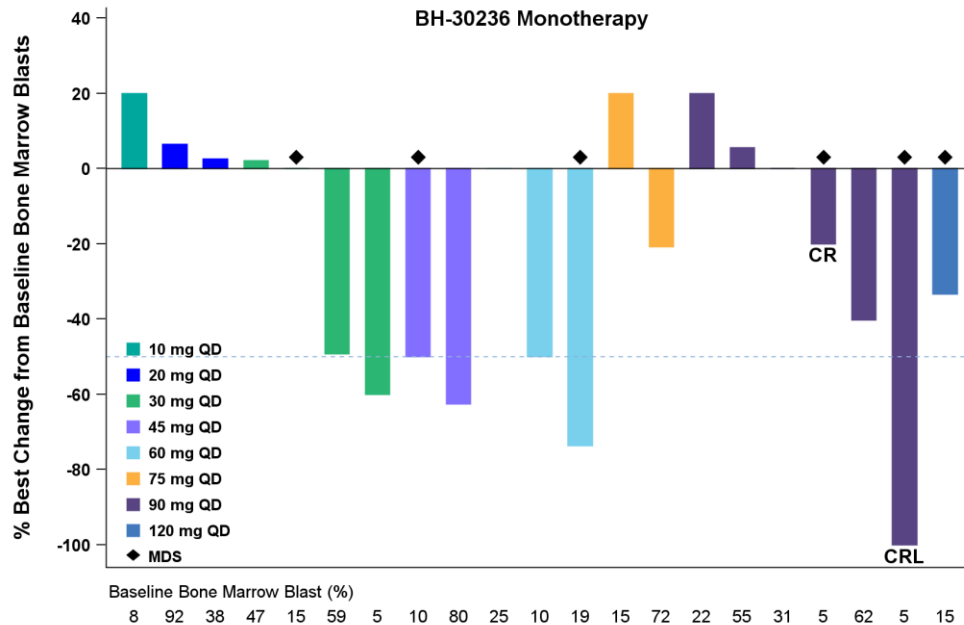
Anti-tumor Activity of BH-30236 in Combination with Venetoclax in MOLM-13 CDX Tumors



BH-30236 Monotherapy Anti-leukemic Activity at ≥ 30 mg QD

- 6/21 (28.6%) patients with post-baseline marrow assessment had $\geq 50\%$ blast count reduction on therapy
- Of 6 evaluable patients with previously treated HR-MDS, 2 (33%) had a CR on monotherapy

**Best Change in Baseline Marrow Blast %
(N=21)**



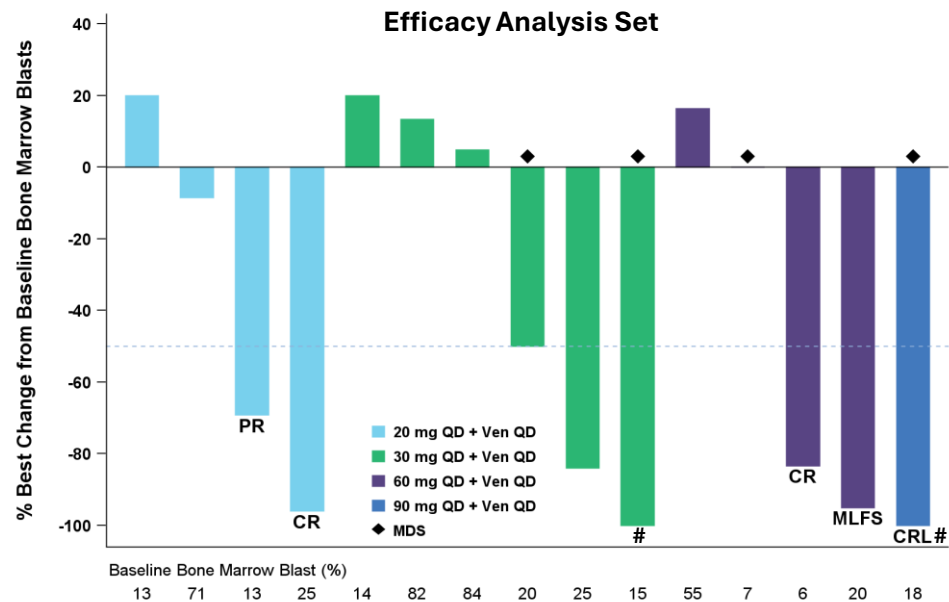
**Anti-Leukemic Activity of BH-30236 Monotherapy
in Relapsed HR-MDS**

Dose	Patient / Disease	Baseline Mutations	Prior Therapy	Key Blood and Marrow Parameters (pre → post)	Best Response / Outcome
60 mg QD	81 yo M Relapsed HR-MDS	ASXL1 DDX41 ETV6, SETBP1 SRSF2 PAX5	1. Aza + SL-172154/ Aza → CR	BM blasts: 19% → 5% ANC: 0.3 → 1.35 PLTs: 25 → 28	NR* Continuing on treatment more than 1yr (Cycle 13+)
90 mg QD	66 yo M Relapsed HR-MDS	ASXL1 NF1 SETBP1 SRSF2	1. Aza + Ven → CR 2. allo-HSCT → CR	BM blasts: 5% → 0% ANC: 0.16 → 1.31 PLTs: 20 → 24	CR _L (MRD -) D/C for allogeneic HSCT

Venetoclax Combo: Anti-leukemic Activity Observed in Ven Pre-treated Patients Across Dose Levels

- 13/15 patients with prior venetoclax exposure
- $\geq 50\%$ blast count reduction in 8/15 (53%) patients with post-baseline marrow assessments
- In 11 evaluable patients with R/R AML, overall response rate was 36% (4/11)

Best Change in Baseline Marrow Blast % (N=15)



Clinical Responses to BH-30236 + Ven with Prior Ven-Refractory AML

Dose	Patient / Disease	Baseline Mutations	Prior Therapy	Key Blood and Marrow Parameters (pre → post)	Best Response / Outcome
20 mg QD	60 yo M AML 2° to HR-MDS	DDX41 SETD2	1. AZA for MDS 2. 7+3 chemo → NR 3. HiDAC → NR 4. FLAG-IDA + Ven → NR	BM blasts: 25% → 1% ANC: 0.86 → 5.86 PLTs: 59 → 158	CR (MRD-) D/C for allo-HSCT
60 mg QD	78 yo M R/R AML	DNMT3A RAD21 RUNX1 SF3B1 TET2	1. Dec + Ven → CR (~12 mo) 2. Clad + LDAC + Ven → PR	BM blasts: 6% → 1% ANC: 1.26 → 2.35 PLTs: 227 → 158	CR Treatment ongoing awaiting allo-HSCT Loss of RUNX1 & SF3B1 mutations in BM



BH-30236

Key Takeaways

Novel approach to target aberrant alternative splicing, an emergent driver of cancer progression and resistance

Tolerable with once daily dosing, both as monotherapy and in combination with venetoclax

Preliminary anti-leukemic effect
motivating additional investigation in myeloid malignancies,
with potential for future investigation in solid tumors





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BH-501284: pan-KRAS Inhibitor

DUAL-STATE, PSEUDO-IRREVERSIBLE, PAN-KRAS INHIBITOR

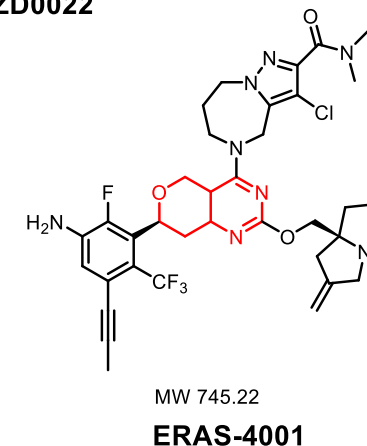
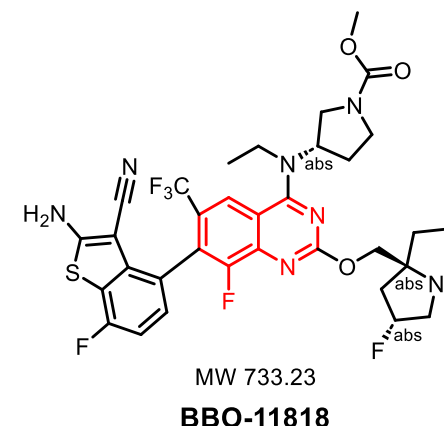
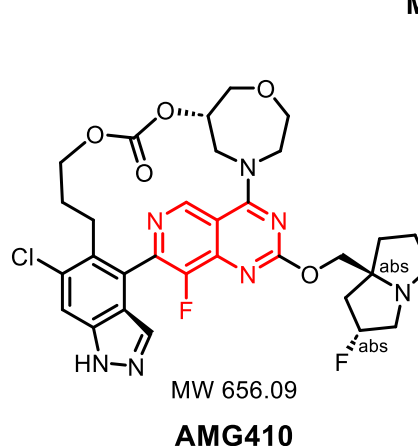
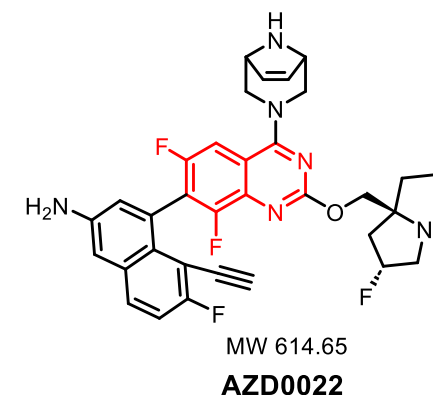
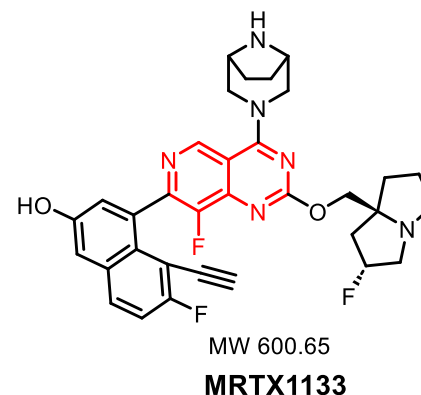
A Fresh Approach to Pan-KRAS Inhibition

Recent progress against KRAS comes with limitations:

- Covalent KRAS inhibition is limited to G12C alleles
- Tricomplex pan-RAS inhibitors have burdensome toxicity that challenges combinations

Non-covalent Switch-II KRAS inhibitors offer the potential for tolerability and broad activity:

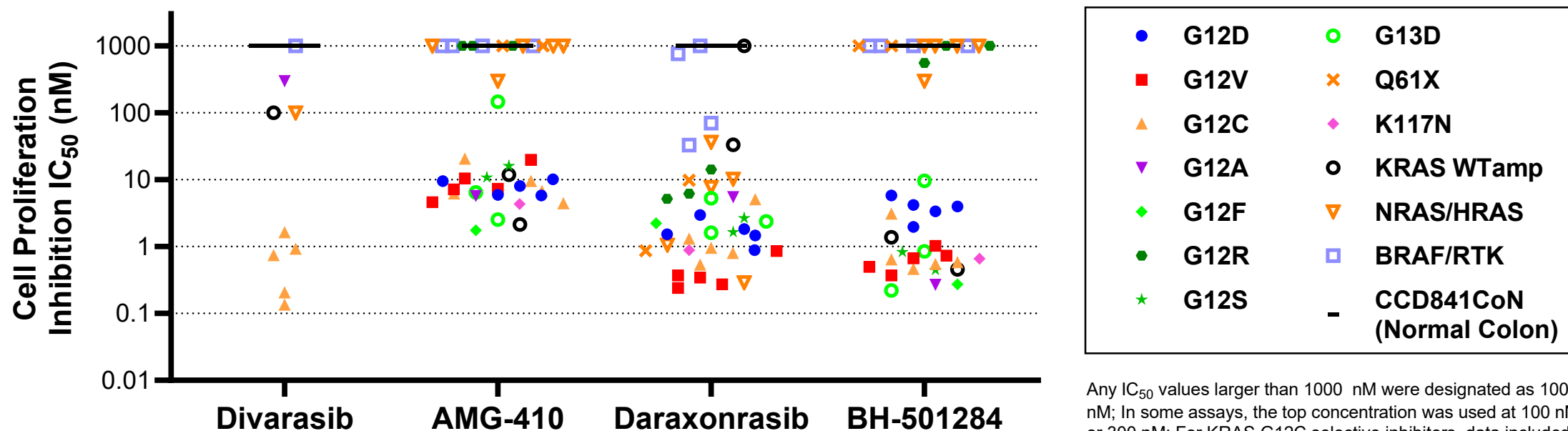
- Current clinical efforts are limited by low bioavailability leading to toxicity from high oral doses with limited efficacy



We believe a fresh chemical scaffold will be needed to deliver a pan-KRAS Switch-II inhibitor which can achieve both high cell potency and marked oral efficacy in clinic

Cell Potency of BH-501284 Versus Clinical Stage KRAS Inhibitors

- Cellular potency mirrored divarasisb in G12C with a non-covalent inhibitor and provided potency against diverse KRAS driver mutations and amplification that exceeded the potency of AMG-410
- Cellular potency matched daraxonrasib, but with a broader therapeutic window against HRAS/NRAS and other RTK-driven cancers



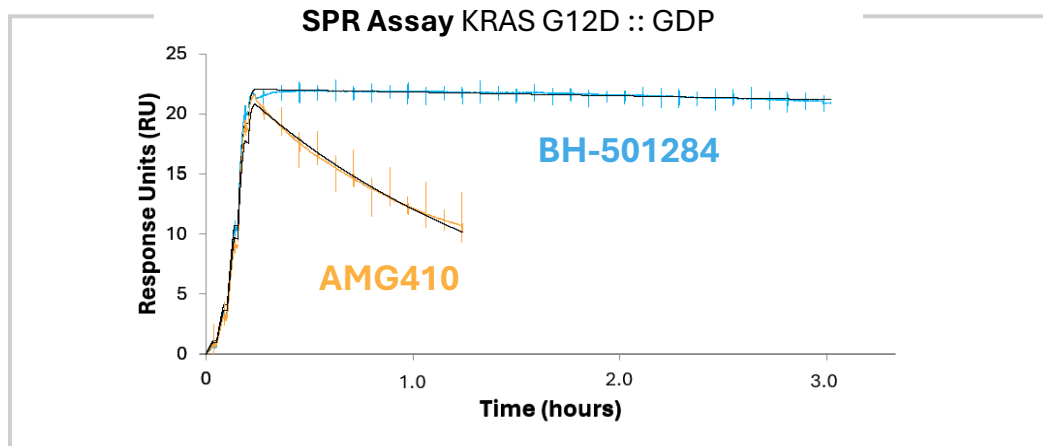
Any IC₅₀ values larger than 1000 nM were designated as 1000 nM; In some assays, the top concentration was used at 100 nM or 300 nM; For KRAS G12C selective inhibitors, data included was restricted to KRAS G12C mutated cells; Data cutoff: 04JUN2026

BH-501284 Demonstrated Long Residence Time, Deep Blocking of RAS (GTP)-cRAF Interaction and Sustained Tumor Regression



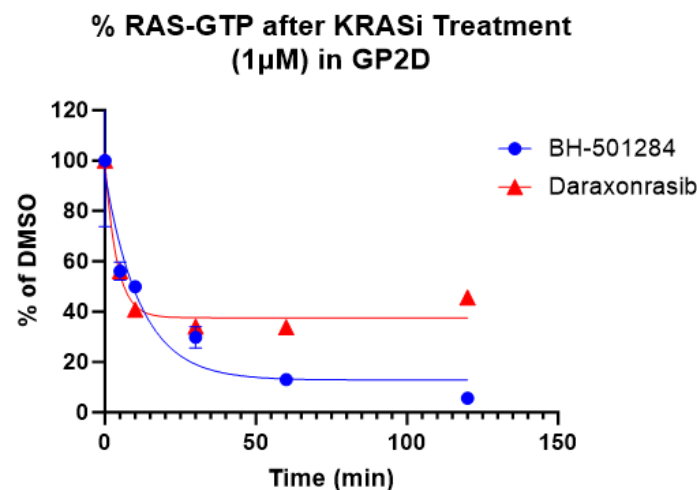
- BH-501284 showed pseudo-irreversible characteristics with long residence time
- BH-501284 showed greater max % KRAS inhibition than daraxonrasib, suggesting more complete elimination of GTP-bound RAS
- BH-501284 demonstrated greater efficacy with deeper and more sustained tumor regression in PANC04.03 CDX tumor model with KRAS G12D mutation than daraxonrasib and zoldonrasib

		BH-501284	AMG410
SPR Assay : "Off" state KRAS G12D :: GDP	K_D (nM)	<0.0148	0.119
	$T_{\text{residence}}$ (hr)	>54.2	1.40

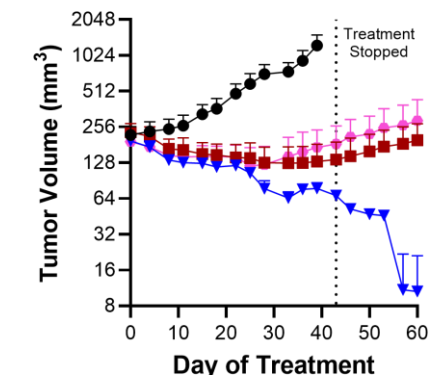


Note: SPR assay was performed at Reaction Biology

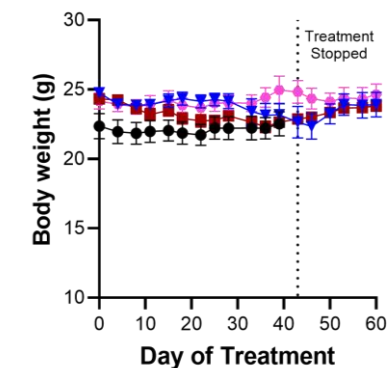
Cellular Kinetic Profile



PANC04.03 (PDAC: KRAS G12D)



PANC04.03 (PDAC: KRAS G12D)

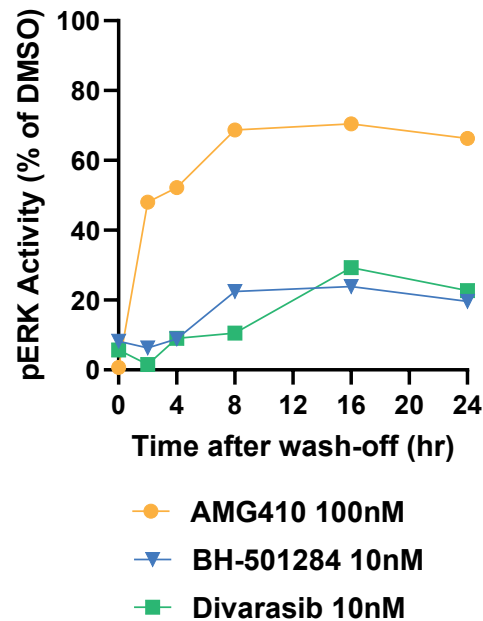




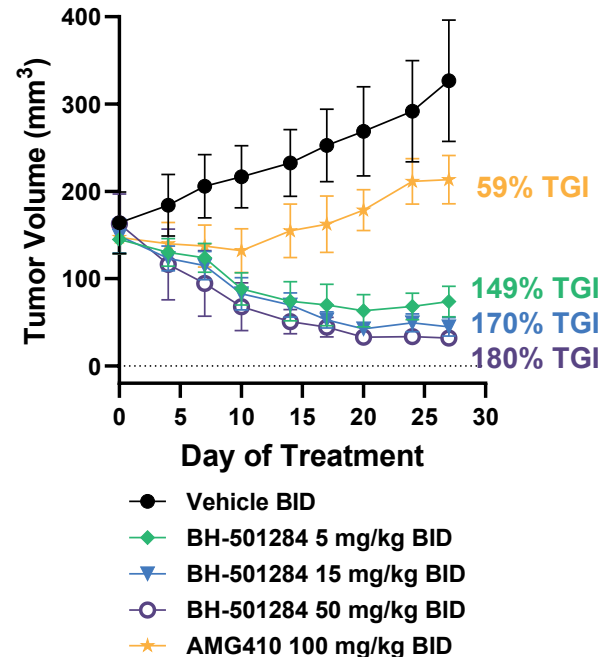
BH-501284 Achieved Sustained Blocking of KRAS Signaling & Marked Efficacy, Comparable to Covalent KRAS G12Ci Divarasilb

- In wash-off experiment, BH-501284 sustained pERK inhibition as effectively as divarasilb, while AMG410 lost pERK inhibition
- In H358 KRAS G12C tumor model, BH-501284 induced tumor regression at a low dose level (5 mg/kg BID 149% TGI), comparable to divarasilb, while AMG410 showed limited TGI at high dose

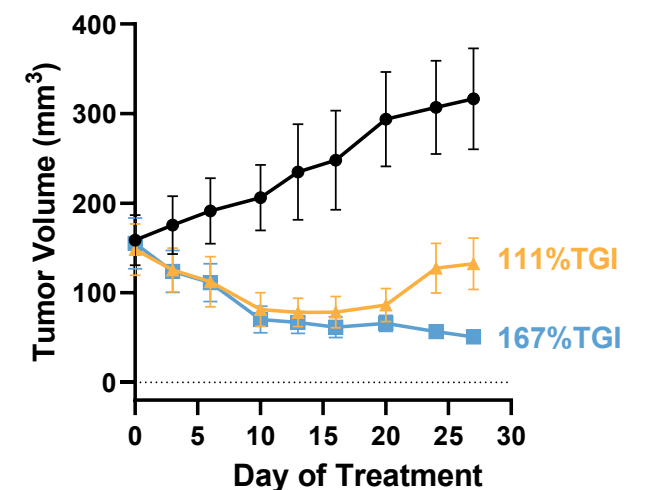
H358 (KRAS G12C)



H358 (NSCLC: KRAS G12C)



H358 (NSCLC: KRAS G12C)





BH-501284

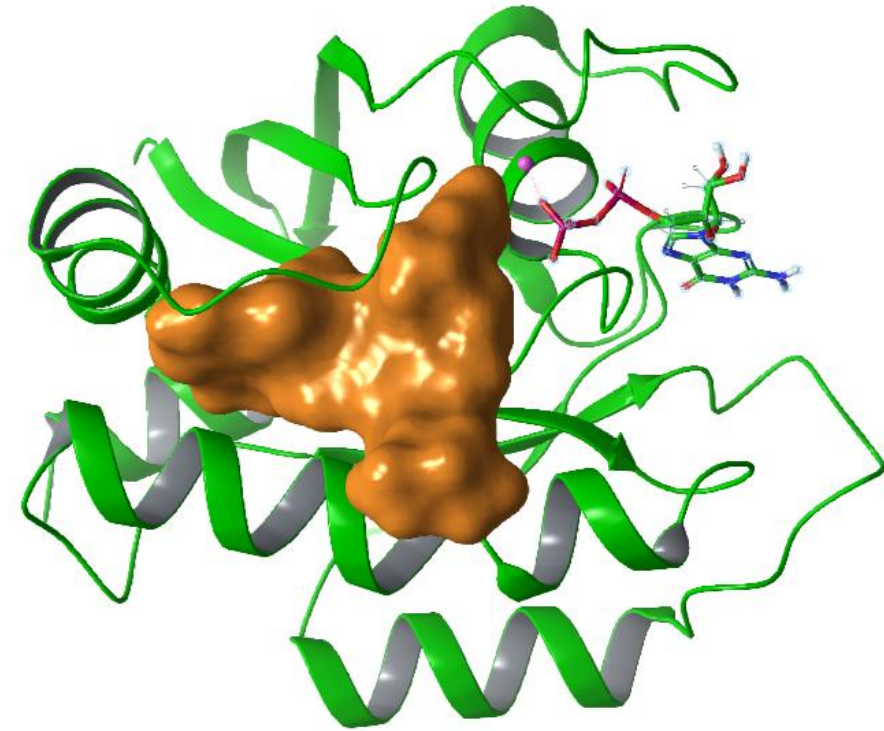
Key Takeaways

Designed for potent pan-KRAS inhibition while sparing HRAS/NRAS, to reduce toxicity seen with tricomplex inhibitors

Unique pseudo-irreversible binding (>54-hour residence time) through use of a novel non-covalent Switch-II chemical scaffold, can lead to prolonged blocking of KRAS signaling

Marked *in vivo* pan-KRAS activity at low oral dose levels across multiple *KRAS*-mutated mouse CDX tumor models

Improved oral bioavailability across species (mouse 30%; rat 27%; dog 13%; 12% bama miniature pig) positions the molecule for clinical impact, with IND-enabling studies ongoing



Thank you

THE NEXT LEAP FORWARD IN PRECISION MEDICINE



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