BlossomHill Therapeutics, Inc.

Corporate Presentation

August 6, 2026
San Diego, CA

BLOSSOMHILL THERAPEUTICS

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This presentation regarding BlossomHill Therapeutics, Inc. (the "Company," "BlossomHill," "we," "us," or "our") contains forward-looking statements. All statements other than statements of historical facts contained in this presentation, including statements regarding the Company's strategy, future financial condition and future operations, the Company's anticipated cash runway, the Company's expectations with regard to its research and development programs, the design and results of current or planned clinical trials and preclinical studies, including the timing and availability of data from such studies and trials, the Company's expectations regarding the potential benefits, activity, effectiveness and safety of its product candidates vis-à-vis other approved cancer therapies, the Company's preclinical, clinical and regulatory development plans, potential accelerated approval and registration pathways, the timing of regulatory meetings, first-patient dosing and the timing and availability of clinical data for its product candidates, including BH-30643, BH-30236 and BH-501284, the potential for the Company's product candidates to be a first-line treatment either as a mono or combination therapy, the Company's ability to commercialize its product candidates, the potential market size and size of the potential patient populations for current and any future product candidates and the potential benefits of collaborations, prospects, plans and objectives of management are forward-looking statements.

In some cases, you can identify forward-looking statements by terminology such as "aim," "anticipate," "assume," "believe," "contemplate," "continue," "could," "design," "due," "estimate," "expect," "goal," "intend," "may," "objective," "plan," "positioned," "potential," "predict," "seek," "should," "target," "will," "would" and other similar expressions that are predictions of or indicate future events and future trends, or the negative of these terms or other comparable terminology. The Company has based these forward-looking statements largely on the Company's current expectations and projections about future events and financial trends that the Company believes may affect its financial condition, results of operations, business strategy and financial needs. These statements are inherently uncertain and investors are cautioned not to unduly rely upon these statements. Although the Company believes that it has a reasonable basis for these forward-looking statements, these forward-looking statements are subject to a number of known and unknown risks, uncertainties and assumptions and the Company cannot guarantee that the future results, levels of activity, performance or events and circumstances reflected in the forward-looking statements will be achieved or occur at all. The actual results may differ materially from those expressed or implied by these forward-looking statements. Except as required by law, the Company undertakes no obligation to publicly update any forward-looking statements, whether as a result of new information, future events or otherwise.

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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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BLOSSOMHILL THERAPEUTICS

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 naive 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
1H 2027	Phase 1 Data: C797S durability
2H 2027	Phase 1 Data: TKI-naive durability and chemo combo cohort

BH-30236 Macrocyclic CLK Inhibitor

1H 2027	Phase 1 safety and efficacy data

BH-501284 Pseudo-irreversible Pan-KRAS Inhibitor

Q1 2027	IND submission

Financial / Corporate Overview

Ticker	BLSM
Cash (end of Q1 2026)	$116.0M
IPO Date	August 7, 2026
Estimated Cash Runway	Q1 2028
Offices	San Diego, CA

Seasoned Management Team and Board

Executive Team	Experience
 **J. Jean Cui, Ph.D.** Scientific Founder, President & CEO ***Board of Directors***	Turning Point Therapeutics, Pfizer
 **Y. Peter Li, Ph.D., M.B.A.** Co-Founder, Executive Chairman ***Board of Directors***	Turning Point Therapeutics
 **Geoffrey Oxnard, M.D.** Chief Medical Officer and EVP	Lilly, Dana-Farber Cancer Institute, FOUNDATION MEDICINE
 **Jason Keyes, M.B.A.** Chief Financial Officer and EVP	equillium, AMGEN, AMYLIN PHARMACEUTICALS, Baxter
 **Vincent P. Liptak, JD, Ph.D., M.B.A.** General Counsel and EVP	Pfizer

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** *+/- venetoclax*	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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THERAPEUTICS

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

316,000[1]

209,000
Total Stage III-IV (advanced/metastatic)
USA, Europe, Japan, China

125,000
Treated with 3rd Gen TKI[2]: USA ~80%; ex-US ~50-60%

16,000
~13% develop C797S on target resistance mutation[3]

Substantial Market Opportunity in EGFR Mutant NSCLC

(2025 Tagrisso sales: ~$7.3B)[4]

Early launch opportunity:

- ~5,400[5] 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

1) GlobalData for EGFR-mutant NSCLC epidemiology estimates; 2) 3rd generation TKI treatment rates are management estimates; 3) C797S resistance frequency is based on Piotrowska 2026; 4) AstraZeneca Annual Report and Form 20-F Information 2025; 5) Management's estimates

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

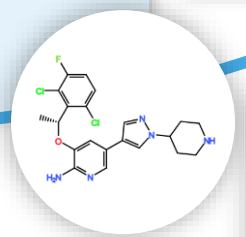
ACHIEVING LONGER PFS AND PRE-EMPTION OF RESISTANCE WITH HIGH-POTENCY

EML4-ALK Inhibitors in *ALK*+ NSCLC

Pfizer

1st Gen: Crizotinib
Cell Potency: 87 nM

ORR: 74%
PFS: 10.9m

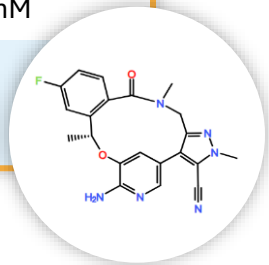


2nd Gen:
Alectinib, Brigatinib

Pfizer

3rd Gen:
Macrocyclic Lorlatinib
Cell Potency: 2.4 nM

ORR: 76%
PFS: >84m

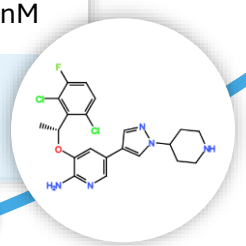


ROS1 Inhibitor in *ROS1*+ NSCLC

Pfizer

1st Gen: Crizotinib
Cell Potency: 12 nM

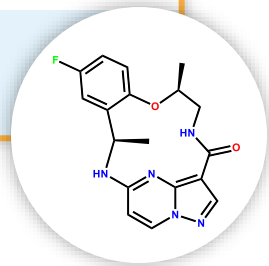
ORR: 66%
PFS: 19.2m



Turning Point Therapeutics

2nd Gen:
Macrocyclic Repotrectinib
Cell Potency: 0.2 nM

ORR: 79%
PFS: 35.7m

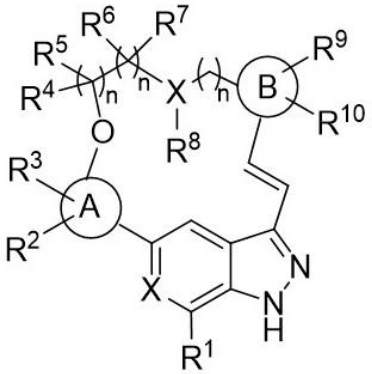


Design & Development Approach

- Macrocyclic design for super-potency and reduced off-target toxicity
- Initial POC in resistant population, crafting a path to first-line development and adoption

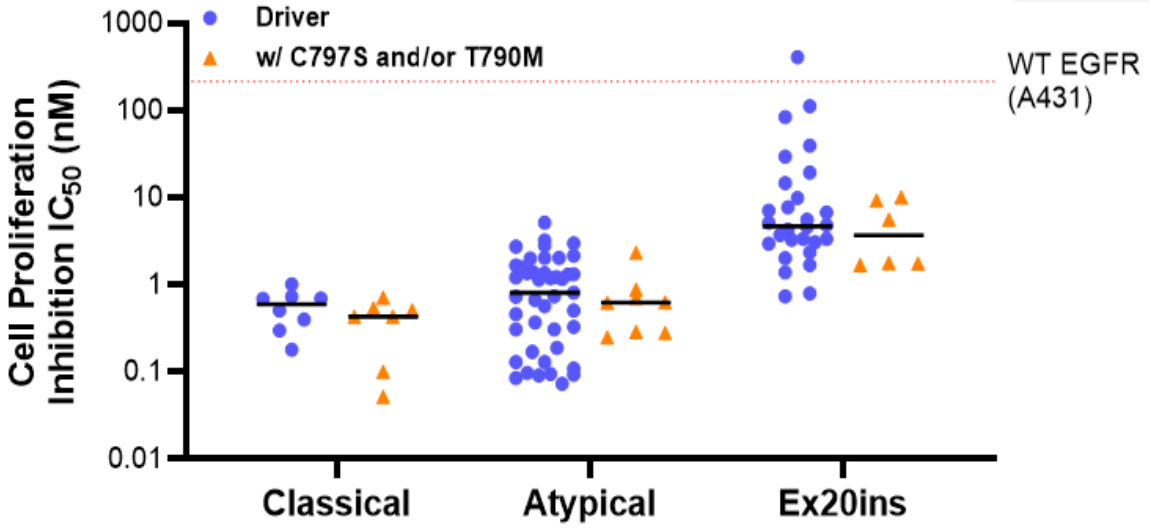
BH-30643: An Intentionally Designed Macrocyclic, Mutant-Selective OMNI-EGFR™ Inhibitor

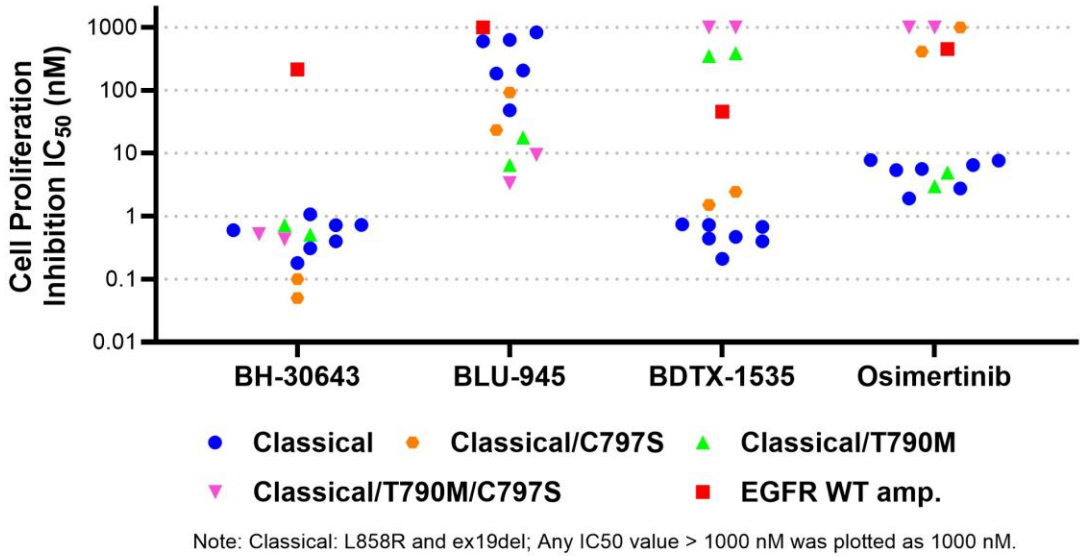
- High cell potency against *EGFR* classical, atypical, and exon20ins
- Equipotent in the setting of on-target resistance mutations



BH-30643

- ~ 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.

1. AACR 2025
2. AACR 2026

BH-30643 SOLARA Phase 1/2 Trial

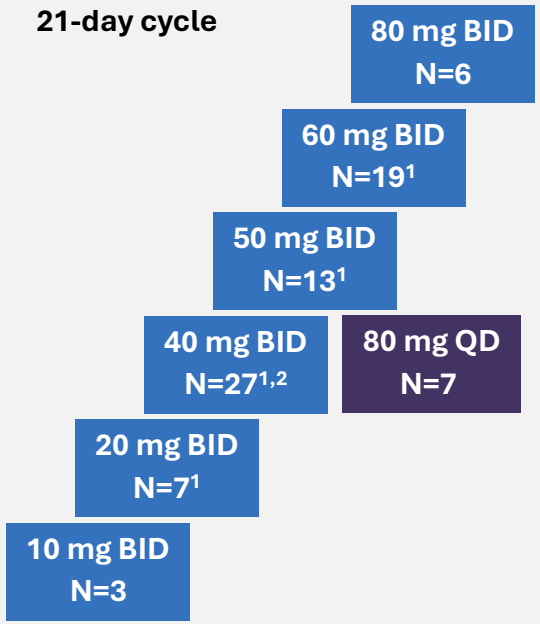
PRELIMINARY DOSE ESCALATION & EXPANSION EXPERIENCE

SOLARA (NCT06706076): Study Design and Patient Characteristics

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

Phase 1 Part 1A (Escalation & backfill)

BOIN design
21-day cycle



Patients enrolled at 29 sites across North America and APAC

Endpoints:
- Primary: Selection of recommended doses for expansion (RDEs) and, if applicable, MTD
- Overall safety and tolerability
- PK characterization
- Preliminary antitumor activity

Key eligibility:
- Adults with advanced *EGFR* and/or *HER2* mutant NSCLC
- ECOG status 0-1
- Measurable disease by RECIST 1.1
- No limit on the number of prior therapy lines
- Untreated asymptomatic brain metastases allowed

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 mutation[4], n (%):**	
Classical *EGFR* (19del or indel, L858R)	54 (66%)
EGFR exon 20 insertion	16 (20%)
Atypical / other *EGFR*	10 (12%)
HER2 mutation	2 (2%)
Multiple *EGFR* mutations[4], n (%):	51 (62%)

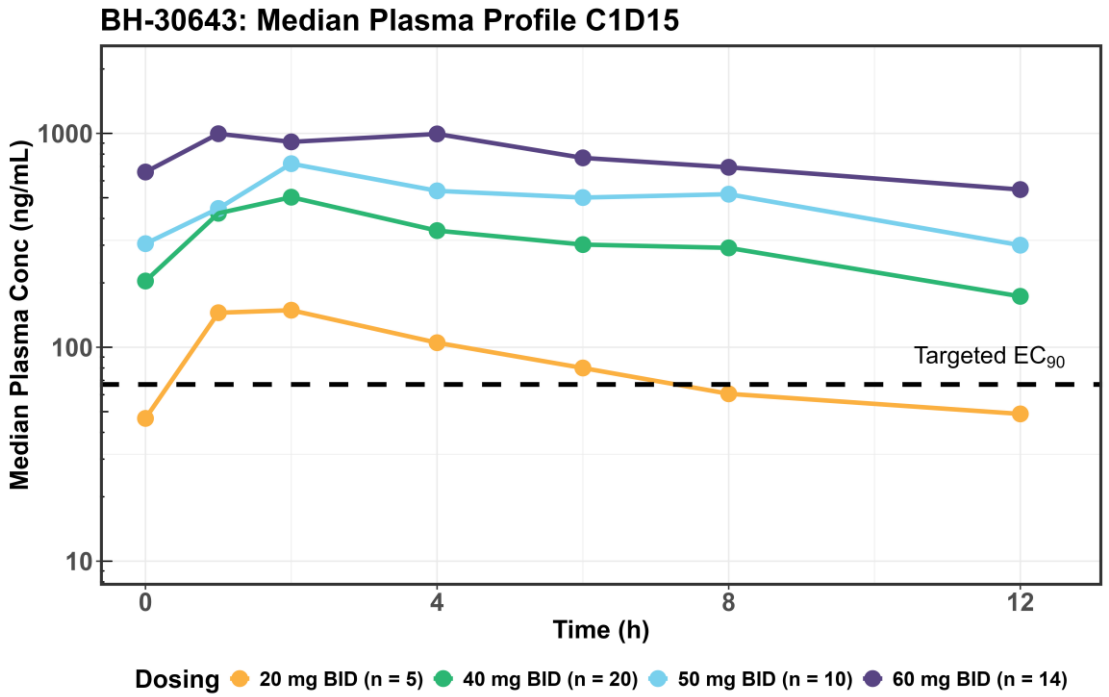
[1]Backfill cohorts enrolled at potentially efficacious doses based on PK and/or anti-tumor activity.
[2]Includes patients enrolled into pilot food effect sub study at 40mg BID

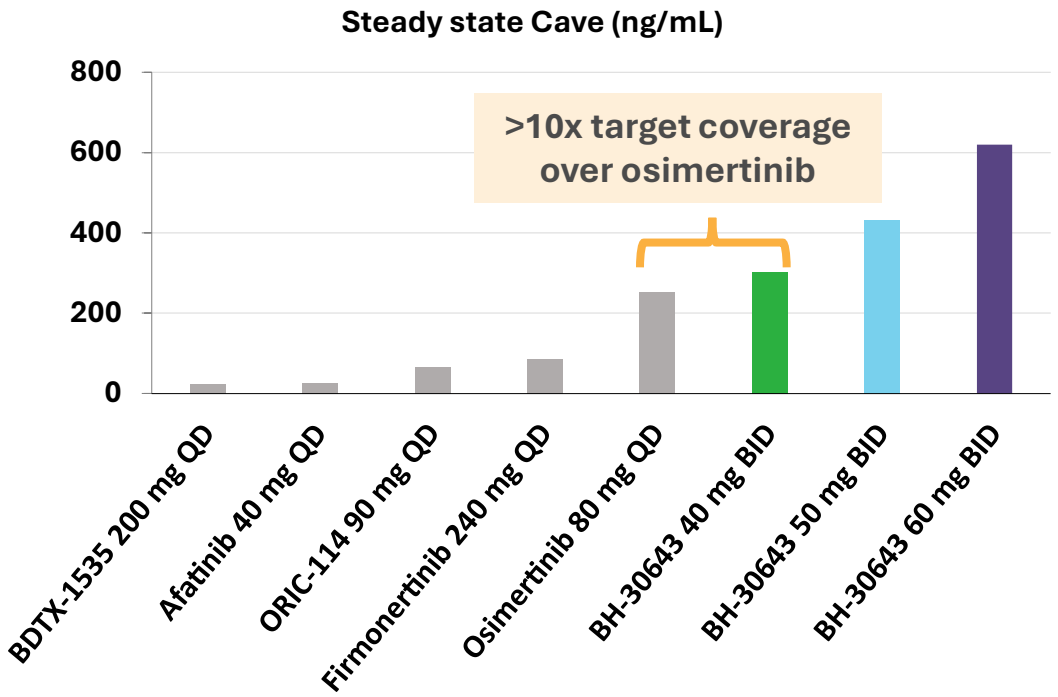
[3]All data as of 2 March 2026 data cut, with efficacy follow-up through 29 April 2026 data cut
[4]Includes historical and baseline molecular results

High PK Exposures Observed in Escalation / Backfill Experience

Median preliminary steady state plasma exposures at ≥40mg 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



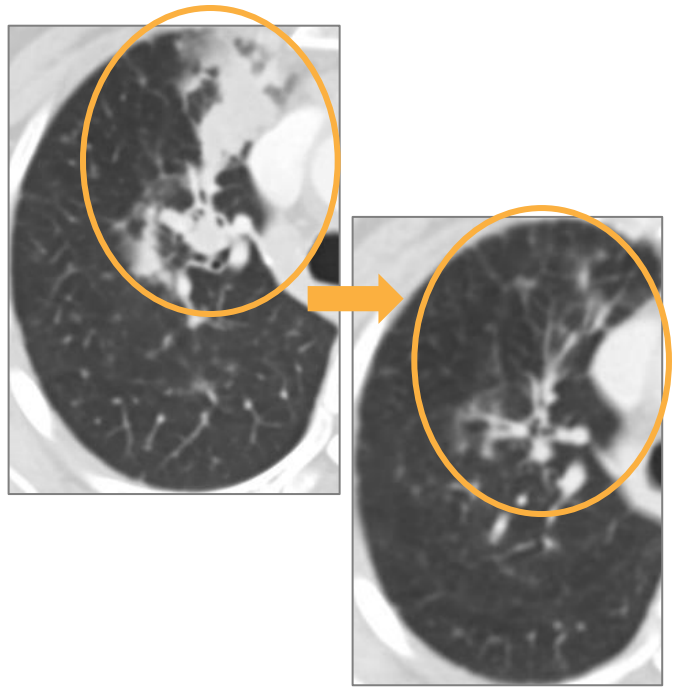


Note: The clinical data presented above are based on cross-study comparisons and are not based on any head-to-head clinical trials. Differences exist between trial designs, patient characteristics and other factors and caution should be exercised in drawing any conclusions from a comparison of the data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

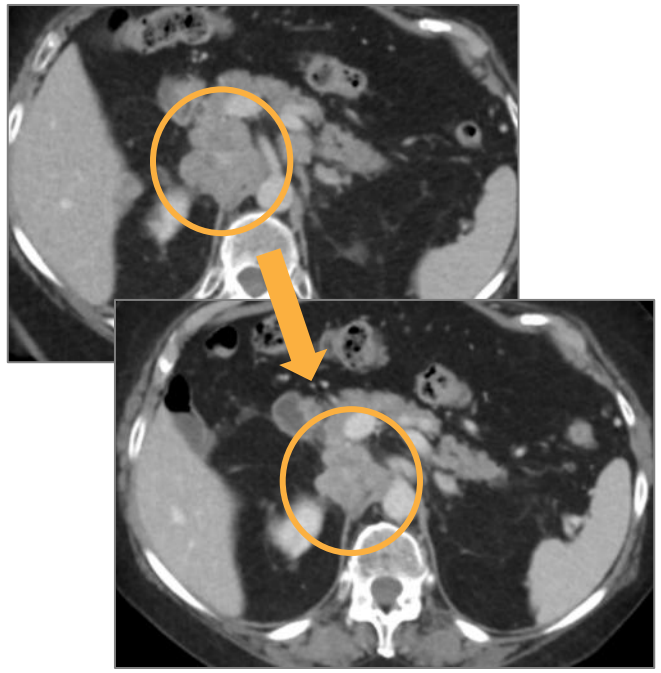
All data as of March 2, 2026 data cut, with efficacy follow-up through April 29, 2026 data cut

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

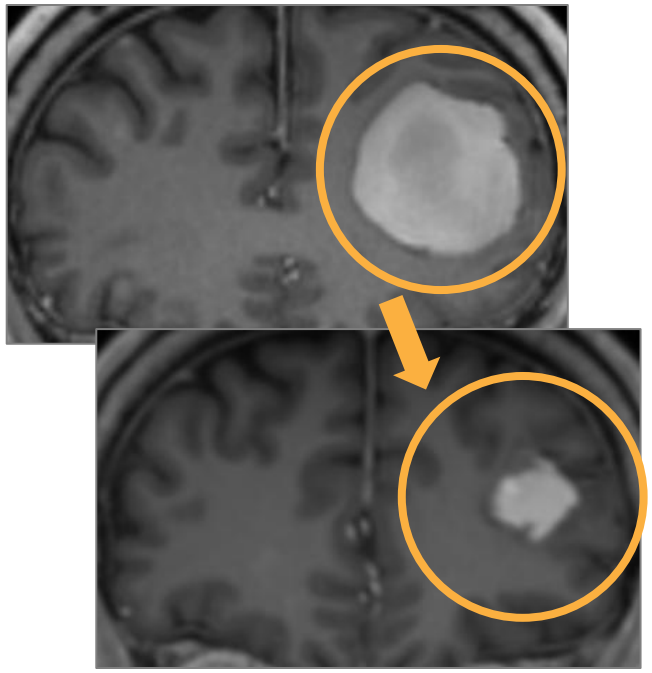
- 44 yo female, enrolled at 40mg BID
- EGFR **exon 19 deletion & C797S** detected after prior osimertinib, amivantamab and immunotherapy
- >11 months on treatment



- 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



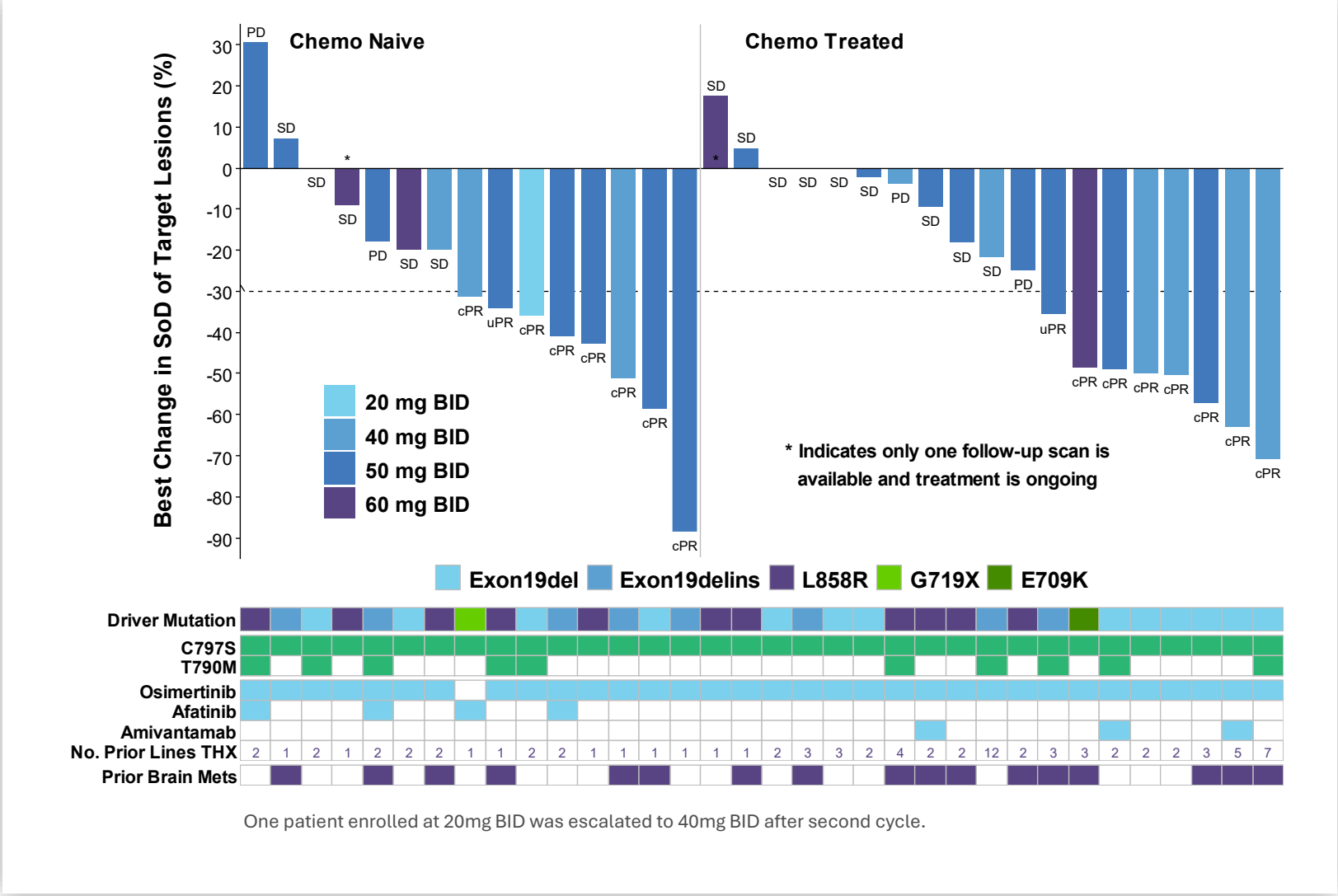
All data as of March 2, 2026 data cut, with efficacy follow-up through April 29, 2026 data cut

BH-30643 Experience in C797S +/- T790M Resistance

PRELIMINARY DOSE ESCALATION / BACKFILL AND EARLY EXPANSION EXPERIENCE

C797S Efficacy Results: Confirmed Responses Observed Across Dose Levels

- 34 patients from target C797S population were response evaluable across escalation/backfill and expansion cohorts
- Responses (confirmed or unconfirmed and ongoing) seen both without prior chemo (53% ORR) and with prior chemo (42% ORR)
  - ➢ 2 patients ongoing with first uPR pending confirmatory imaging
- Responses observed with or without concurrent T790M, with or without prior history of brain metastases, and in atypical/C797S resistance

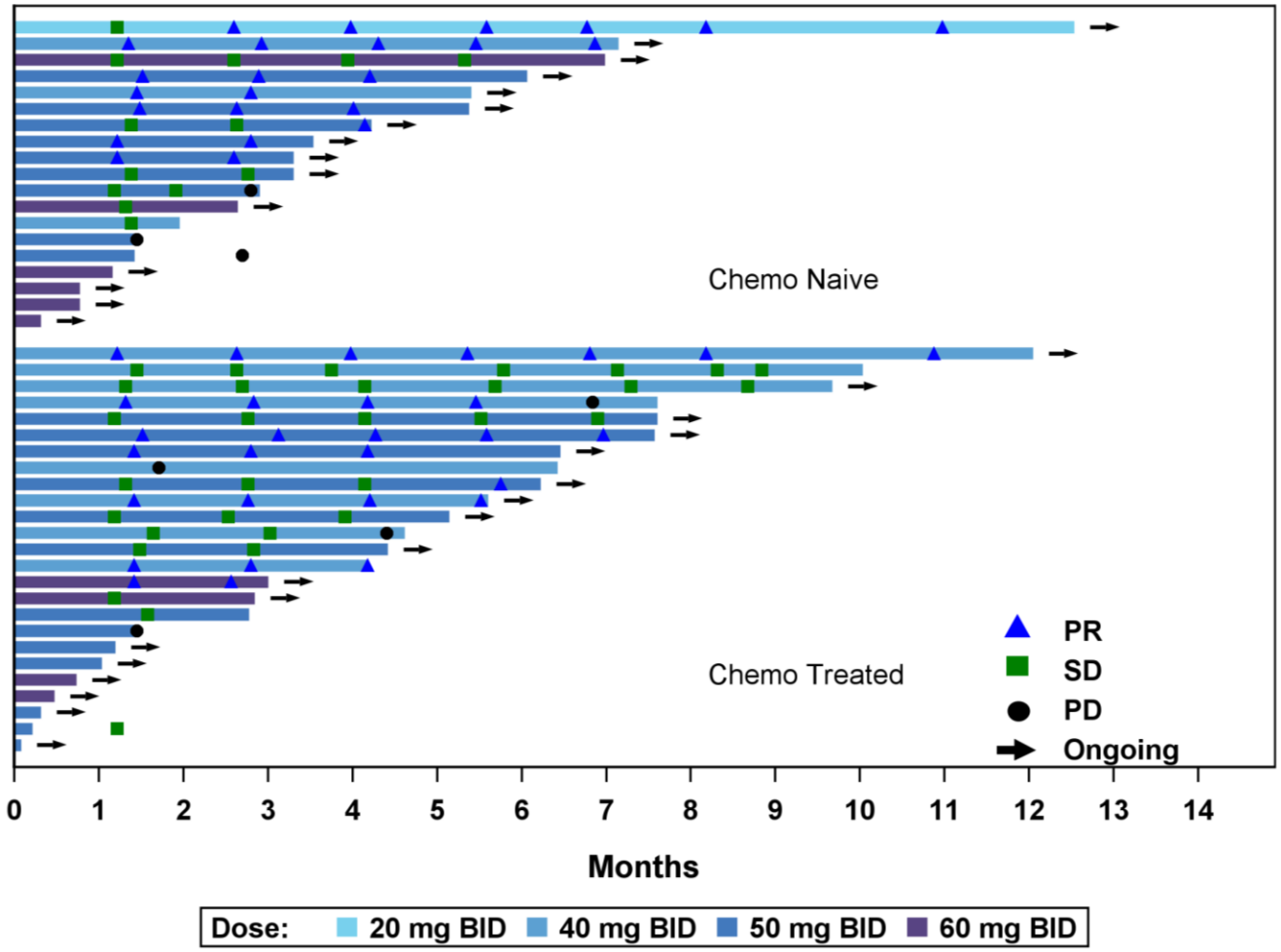


One patient enrolled at 20mg BID was escalated to 40mg BID after second cycle.

All data as of updated May 20, 2026 efficacy cutoff

C797S Efficacy Results: Preliminary Durability

- A total of 44 patients were enrolled for target C797S population with 10 pending initial scan results
  - ➢ Excluded patients with concurrent driver alterations or who previously received EGFR TKI specifically targeting a known C797S
- 32 (73%) remained on therapy; with 2 responders on therapy for over 12 months
- Median follow-up was 4.2 months, too immature for PFS estimation at this time



All data as of updated May 20, 2026 efficacy cutoff

Maturing Safety Data Supports RP2D Selection

TEAE reported in >10% of patients, with a median exposure of 2.8 months

TEAEs, Starting Dose and Grade	All doses (N = 196)		40 mg BID (N = 85)		50 mg BID (N=60)		60 mg BID (N = 28)	
N (%), by preferred term	Any Grade	Grade ≥3	Any Grade	Grade ≥3	Any Grade	Grade ≥3	Any Grade	Grade ≥3
Any TEAEs	189 (96.4)	75 (38.3)	84 (98.8)	32 (37.6)	57 (95.0)	18 (30.0)	25 (89.3)	12 (42.9)
Bilirubin Elevation*	93 (47.4)	21 (10.7)	43 (50.6)	7 (8.2)	29 (48.3)	4 (6.7)	11 (39.3)	4 (14.3)
Rash*	93 (47.4)	6 (3.1)	42 (49.4)	3 (3.5)	25 (41.7)	0	15 (53.6)	1 (3.6)
Diarrhea	87 (44.4)	4 (2.0)	41 (48.2)	0	21 (35.0)	2 (3.3)	14 (50.0)	1 (3.6)
Dry skin	45 (23.0)	0	22 (25.9)	0	14 (23.3)	0	8 (28.6)	0
Stomatitis*	45 (23.0)	4 (2.0)	19 (22.4)	1 (1.2)	12 (20.0)	0	8 (28.6)	2 (7.1)
AST increased	38 (19.4)	9 (4.6)	22 (25.9)	6 (7.1)	9 (15.0)	2 (3.3)	2 (7.1)	0
Fatigue	38 (19.4)	3 (1.5)	19 (22.4)	2 (2.4)	8 (13.3)	0	6 (21.4)	0
ALT increased	37 (18.9)	17 (8.7)	22 (25.9)	10 (11.8)	10 (16.7)	4 (6.7)	1 (3.6)	0
Nausea	30 (15.3)	1 (0.5)	17 (20.0)	1 (1.2)	3 (5.0)	0	5 (17.9)	0
Paronychia	29 (14.8)	1 (0.5)	17 (20.0)	0	5 (8.3)	0	3 (10.7)	1 (3.6)
Decreased appetite	26 (13.3)	2 (1.0)	10 (11.8)	2 (2.4)	6 (10.0)	0	5 (17.9)	0
Anaemia	25 (12.8)	6 (3.1)	13 (15.3)	4 (4.7)	4 (6.7)	0	3 (10.7)	1 (3.6)
Headache	25 (12.8)	1 (0.5)	12 (14.1)	1 (1.2)	5 (8.3)	0	6 (21.4)	0
Vomiting	23 (11.7)	0	10 (11.8)	0	4 (6.7)	0	7 (25.0)	0
Constipation	21 (10.7)	0	11 (12.9)	0	4 (6.7)	0	5 (17.9)	0
Cough	20 (10.2)	0	9 (10.6)	0	3 (5.0)	0	3 (10.7)	0

- Bilirubin elevation is predominantly asymptomatic and unconjugated due to UGT1A1 inhibition
- Grade 3/4 treatment-related transaminitis in 14 patients (7.1%) with no cases meeting Hy's law criteria
- Pneumonitis reported in 2 patients overall (1%: one Grade 3 & one Grade 4)
- No clinically significant QTc effects or other cardiac effects; no Grade 5 TRAE

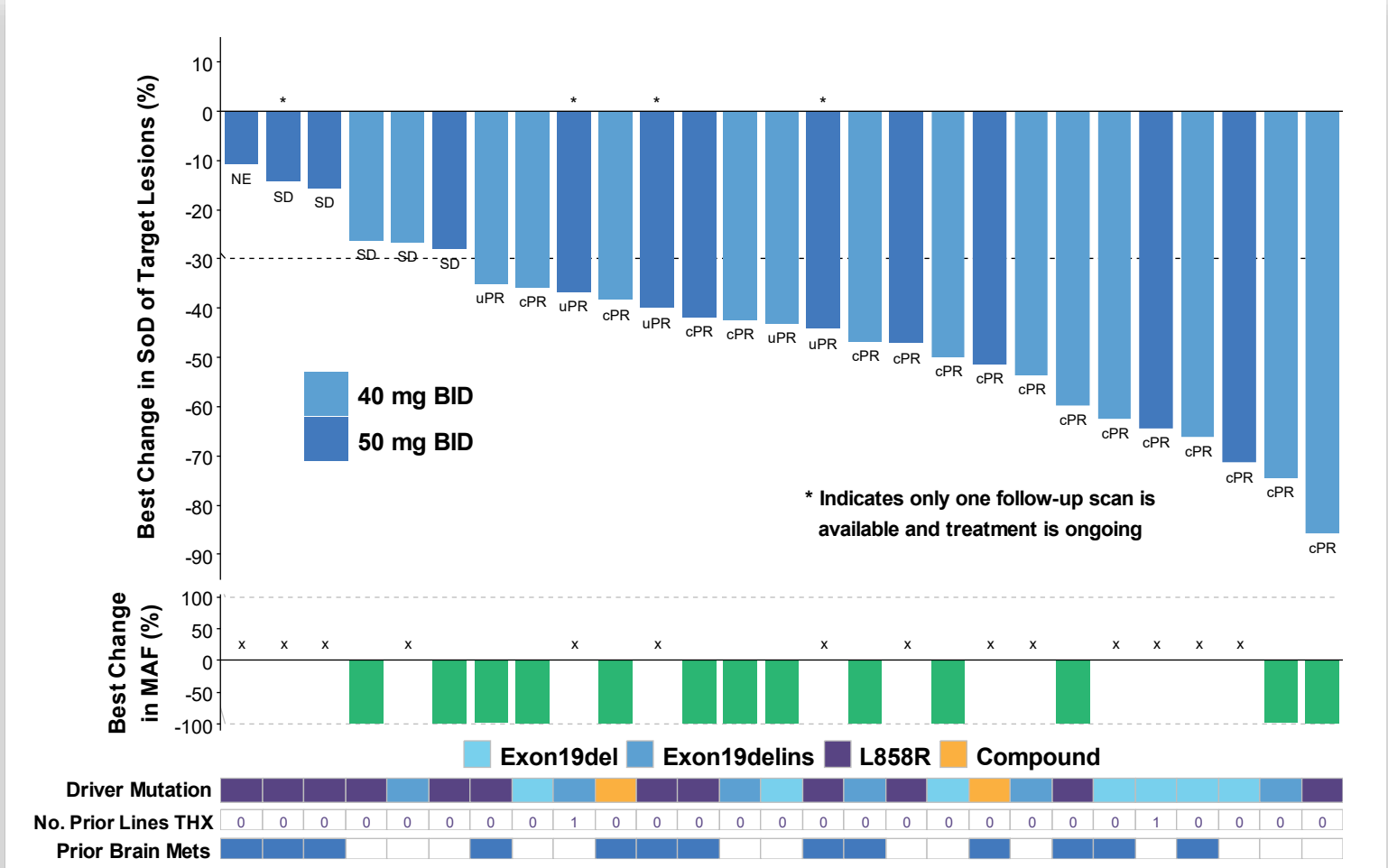
* Grouped terms; All data as of May 12, 2026, safety data cut

BH-30643 Experience in EGFR TKI Naive Classical Mutation

PRELIMINARY EARLY EXPANSION EXPERIENCE

Compelling Early Activity Observed in the Classical TKI-naive Setting

- Response (confirmed or unconfirmed and ongoing) in 21 of 27 patients (78%) with on-treatment imaging; all had tumor reduction
- Enriched for brain mets (48%) and higher risk genotypes (30% exon19 indels or H870R)
- Median follow-up 4.2 months
- 11/13 pts (85%) showed 100% clearance of *EGFR* mutations in ctDNA and two had >98% reduction



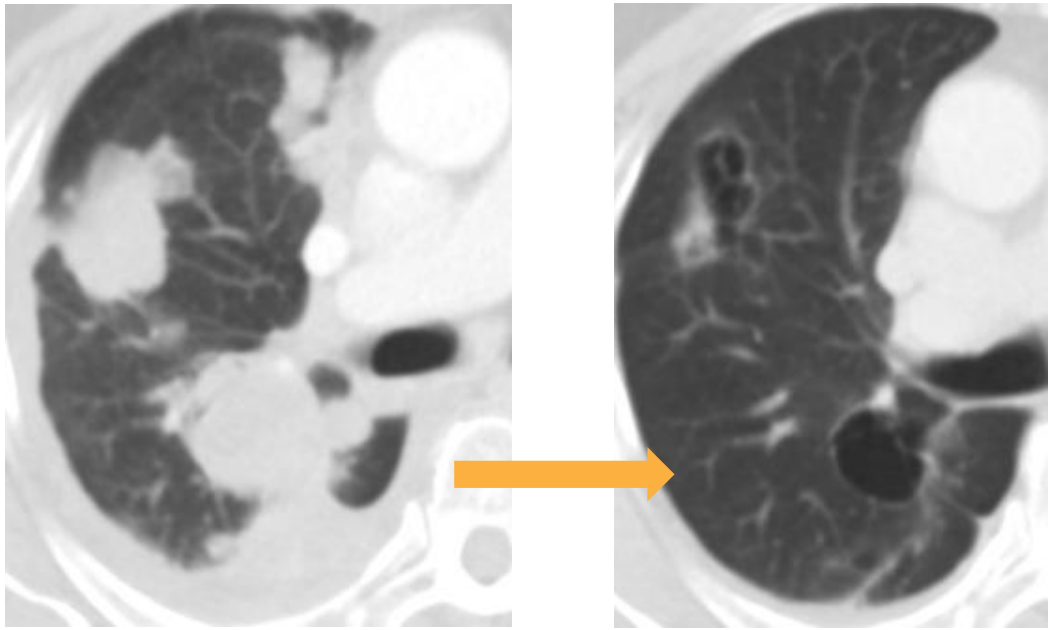
x Currently, ctDNA data is not available for these patients.

All data as of updated 20 May 2026 efficacy cutoff

First-line Activity Against Challenging EGFR Mutations

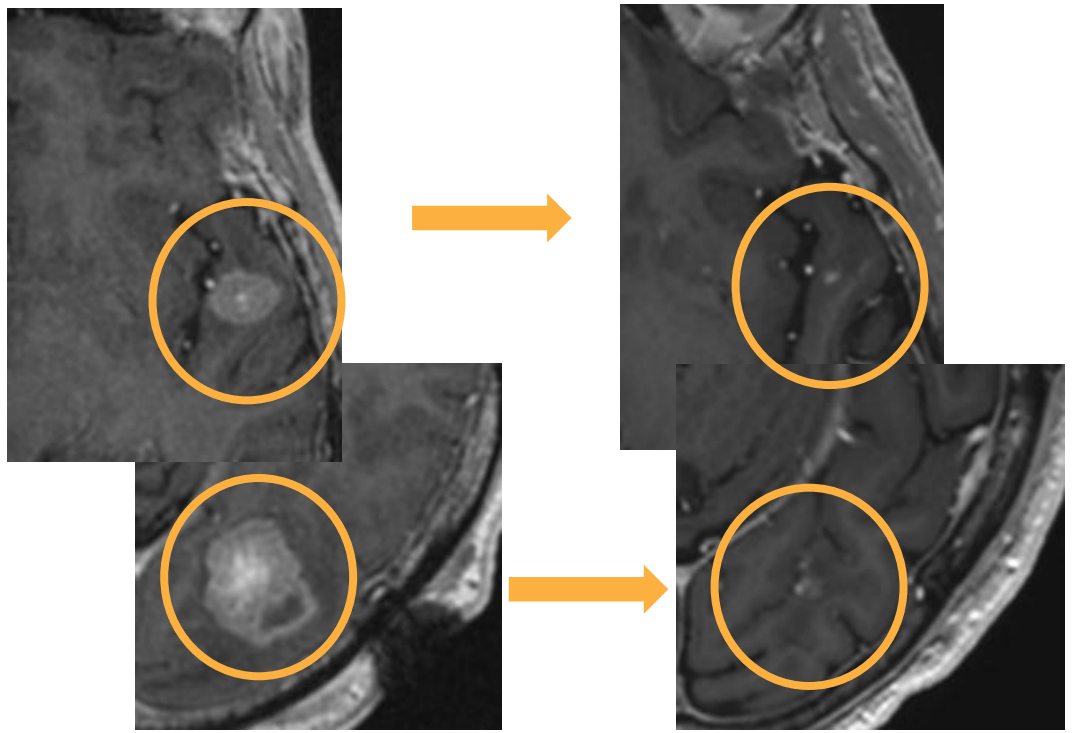
L858R

- 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

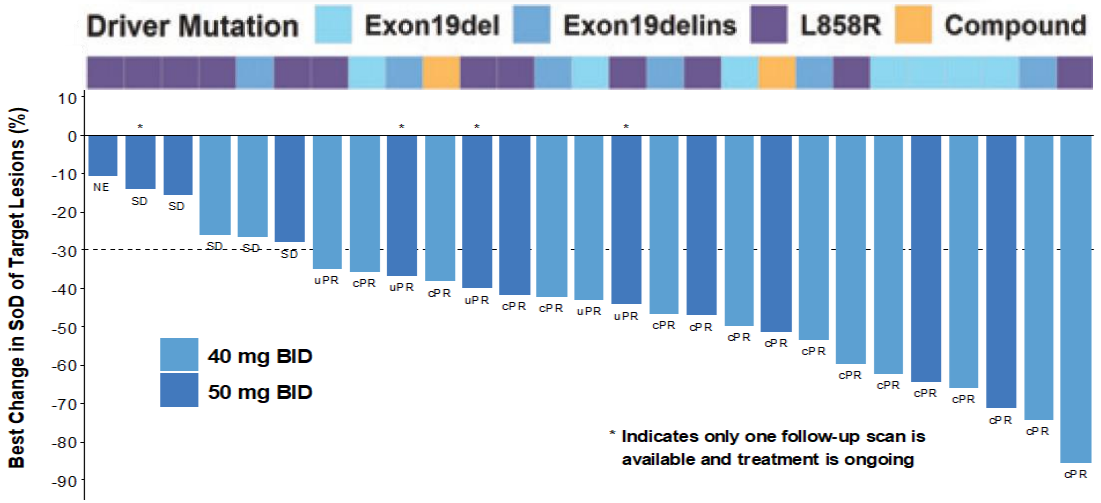


Interim data as of April 29, 2026

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

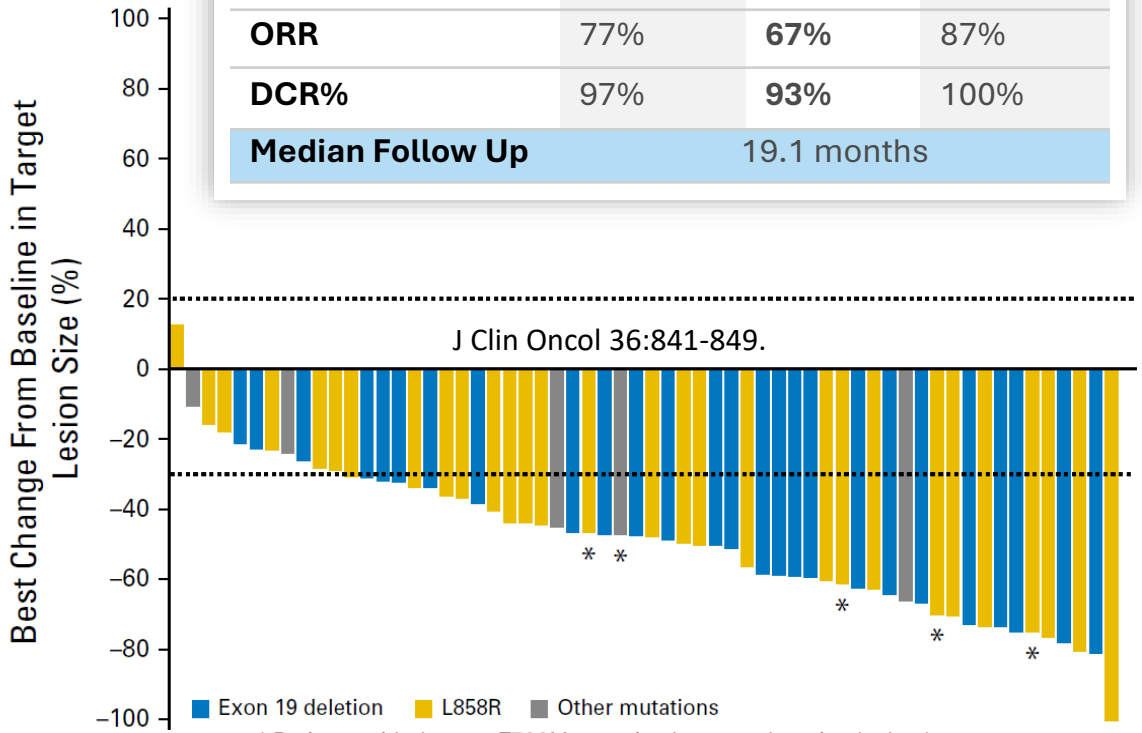
BH-30643 TKI-naive

	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-naive

	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		



All data as of updated May 20, 2026 efficacy cutoff

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.

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

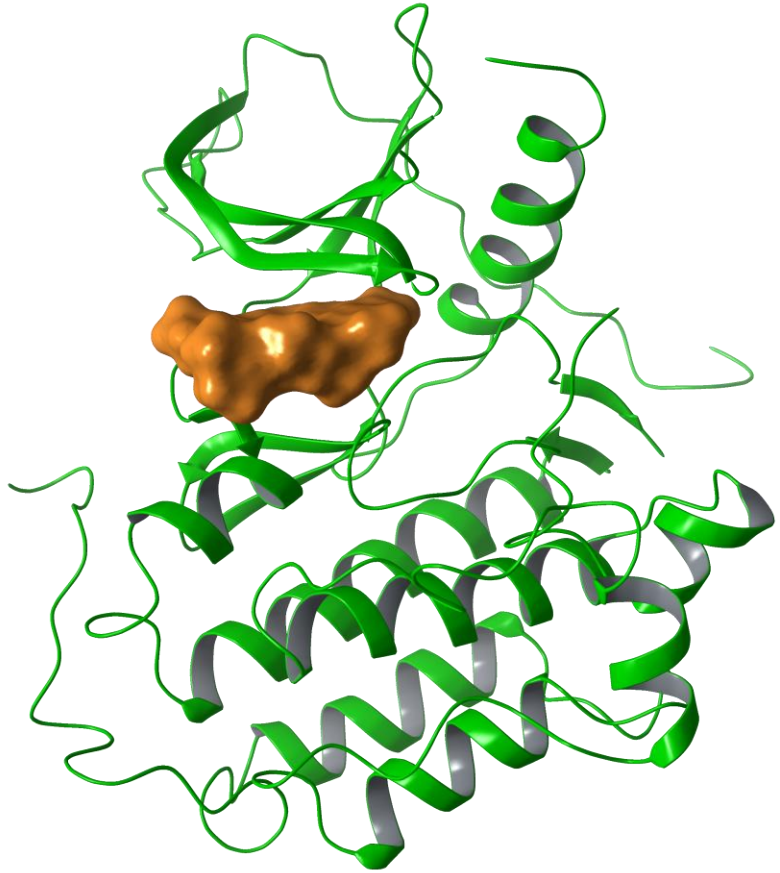
Compelling activity in patients with C797S with potential path to accelerated approval

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



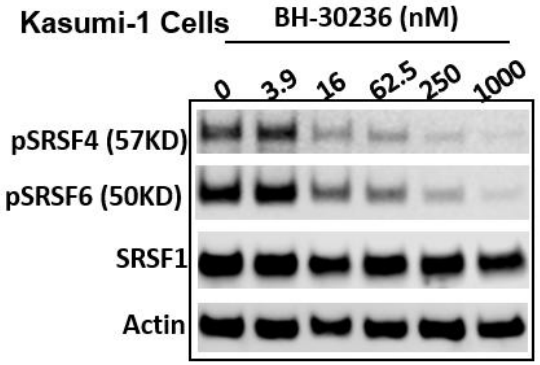
BLOSSOMHILL
THERAPEUTICS

BH-30236: CLK Inhibitor

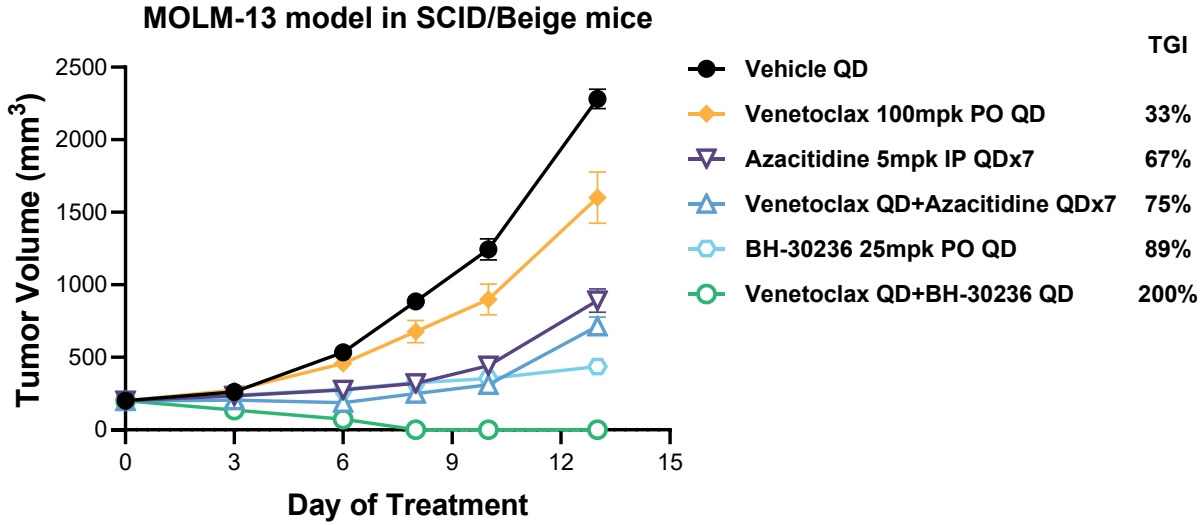
INVESTIGATIONAL, MACROCYCLIC, NON-COVALENT INHIBITOR
TARGETING ABERRANT ALTERNATIVE SPLICING

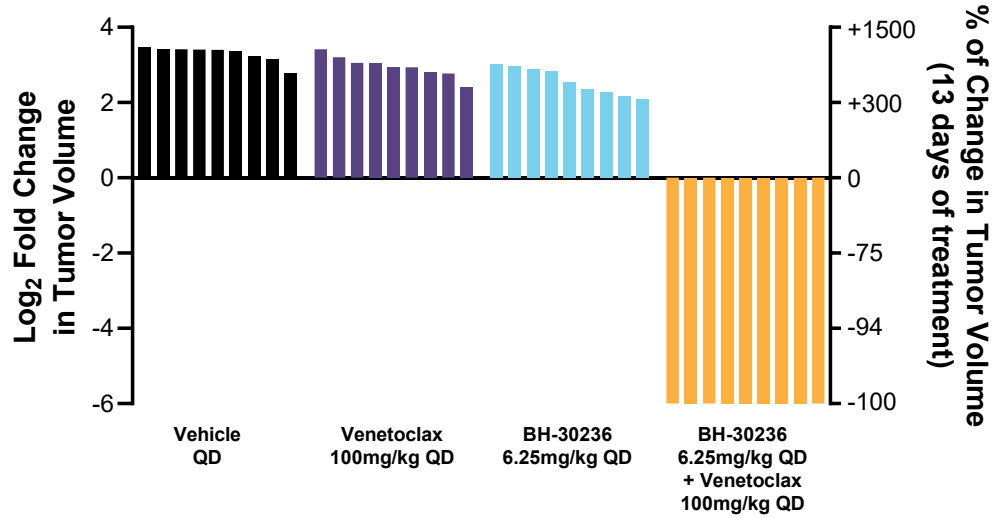
BH-30236: Potent CLK inhibitor Modulating Aberrant Alternative Splicing

- 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

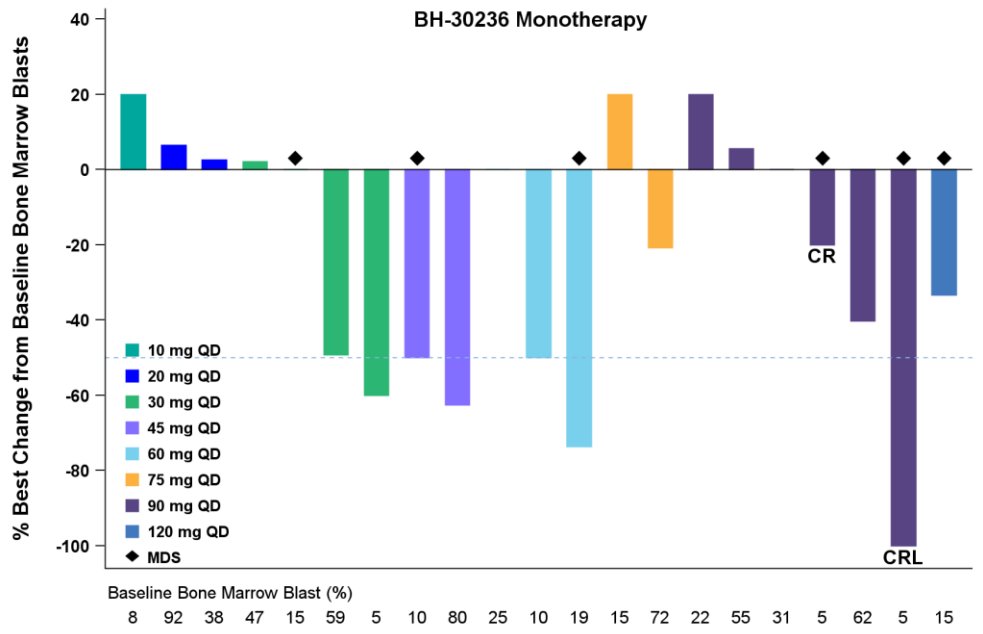




BH-30236 Monotherapy Anti-leukemic Activity at ≥ 30mg QD

- 6/21 (28.6%) patients with post-baseline marrow assessment had ≥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

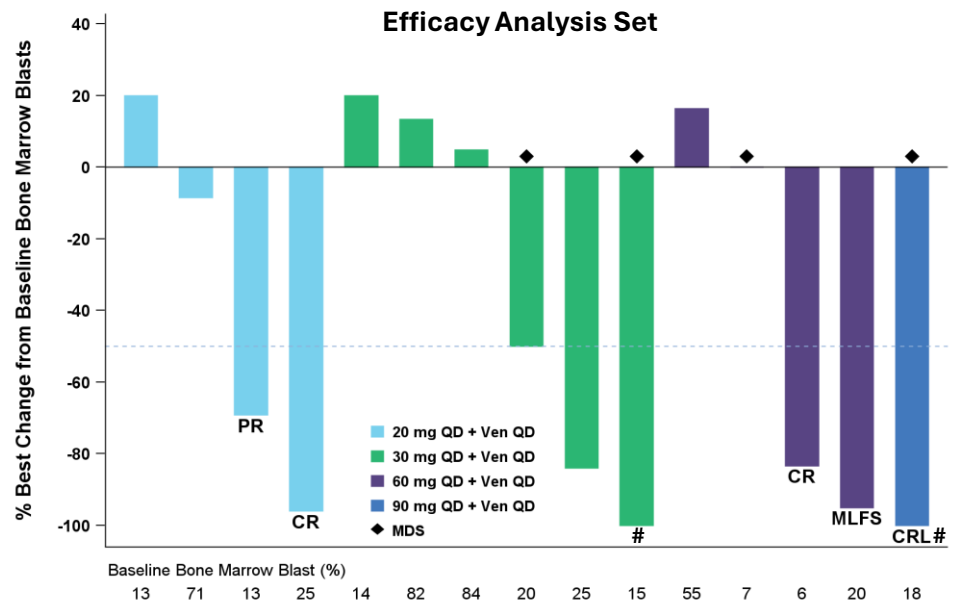
Dose	Patient / Disease	Baseline Mutations	Prior Therapy	Key Blood and Marrow Parameters (pre → post)	Best Response / Outcome
60 mg QD	81 yo M Relapsed HR-MDS	ASXL1 DDX41 ETV6, SETBP1 SRSF2 PAX5	1. Aza + SL-172154/ Aza → CR	BM blasts: 19% → 5% ANC: 0.3 → 1.35 PLTs: 25 → 28	NR* Continuing on treatment 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

Interim study data as of May 20, 2026

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

- 13/15 patients with prior venetoclax exposure
- ≥ 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

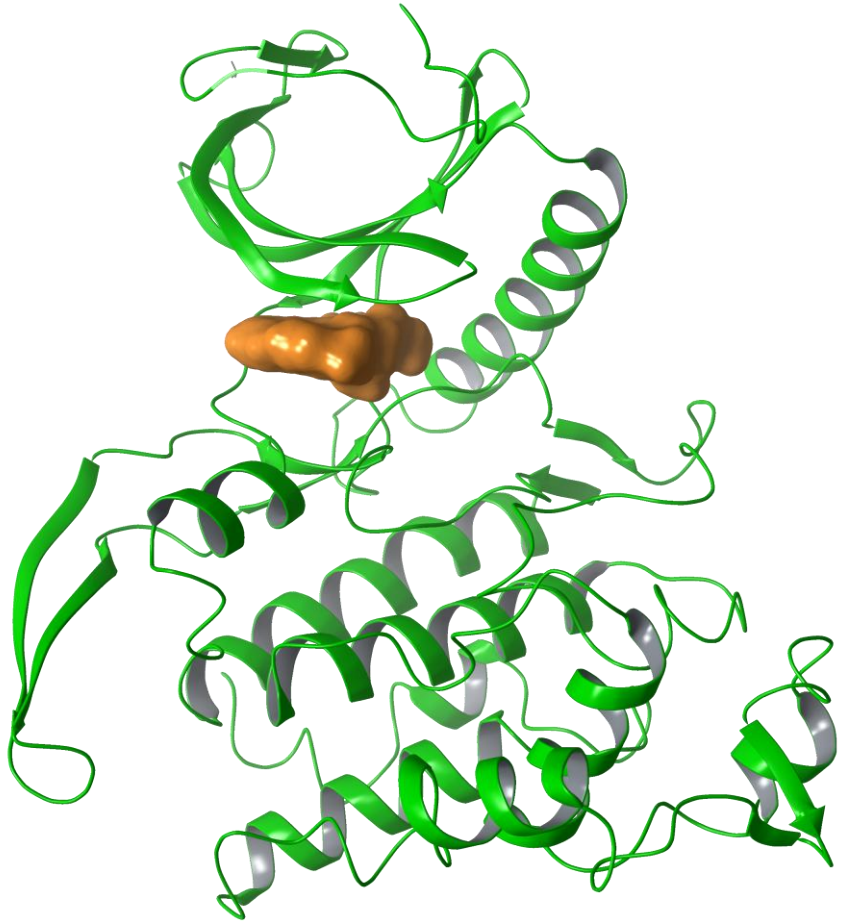
Ongoing study data as of May 20, 2026

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



BLOSSOMHILL THERAPEUTICS

BH-501284: pan-KRAS Inhibitor

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

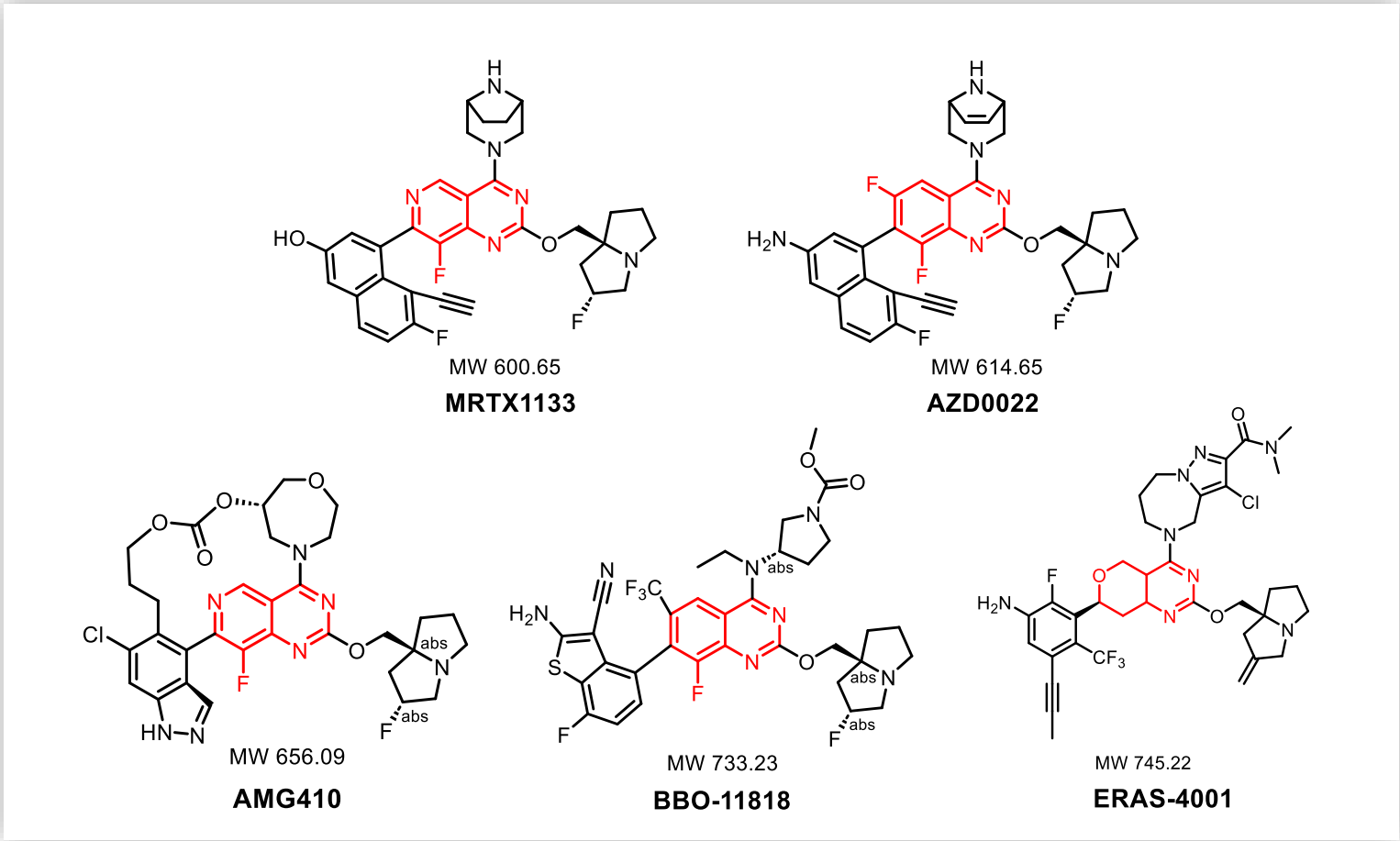
A Fresh Approach to Pan-KRAS Inhibition

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:

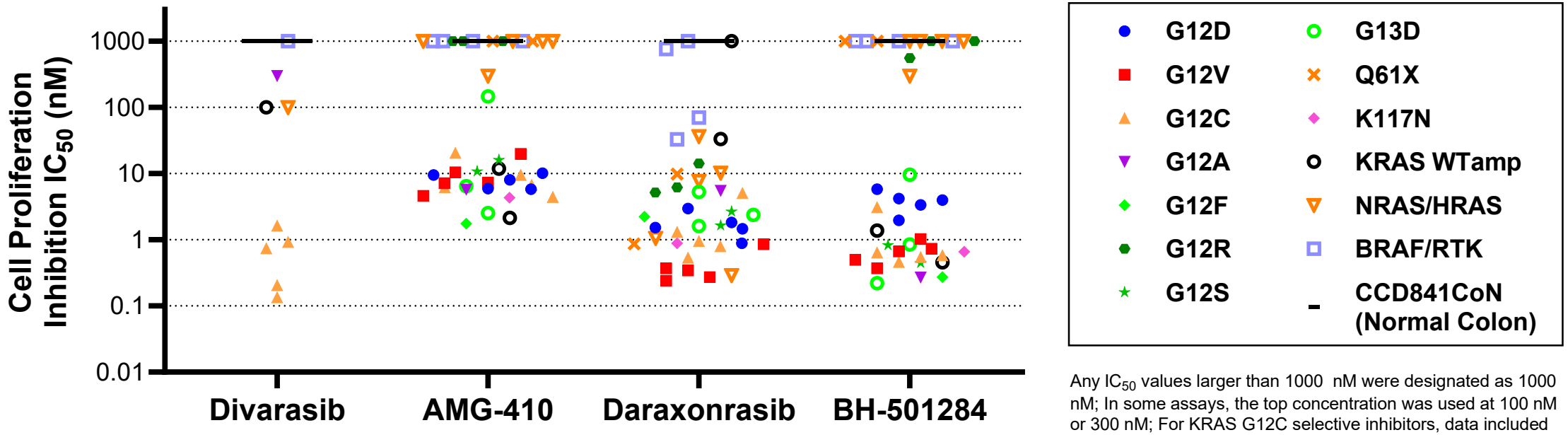
- Current clinical efforts are limited by low bioavailability leading to toxicity from high oral doses with limited efficacy



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 divarasib 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

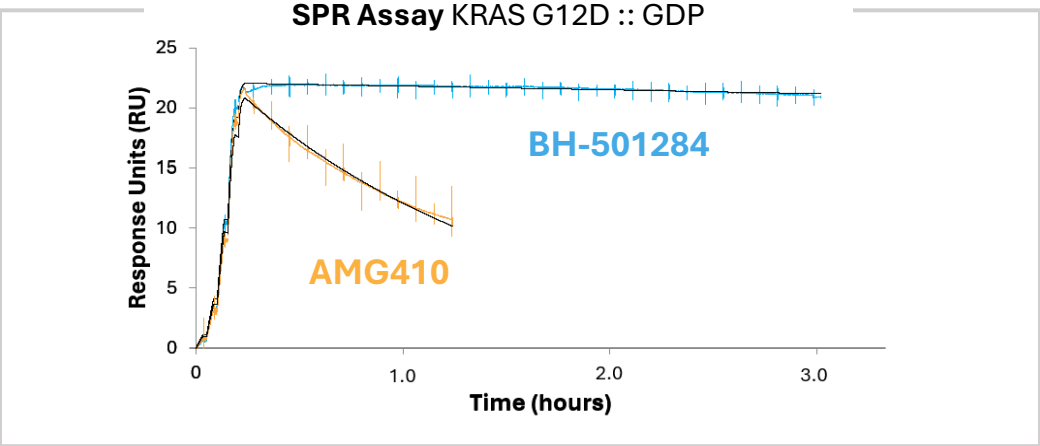


Any IC_{50} values larger than 1000 nM were designated as 1000 nM; In some assays, the top concentration was used at 100 nM or 300 nM; For KRAS G12C selective inhibitors, data included was restricted to KRAS G12C mutated cells; Data cutoff: 04JUN2026

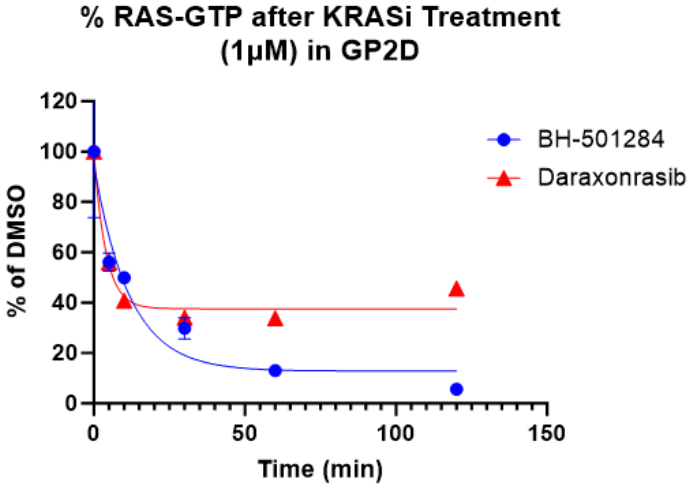
BH-501284 Demonstrated Long Residence Time, Deep Blocking of RAS (GTP)-cRAF Interaction and Sustained Tumor Regression

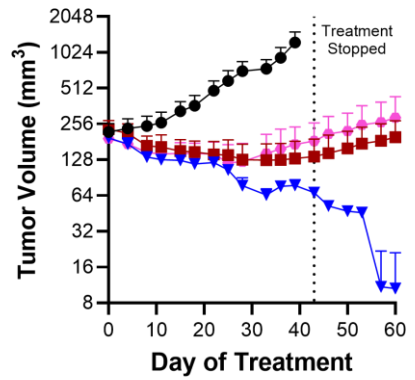
- BH-501284 showed pseudo-irreversible characteristics with long residence time
- BH-501284 showed greater max % KRAS inhibition than daraxonrasib, suggesting more complete elimination of GTP-bound RAS
- BH-501284 demonstrated greater efficacy with deeper and more sustained tumor regression in PANC04.03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

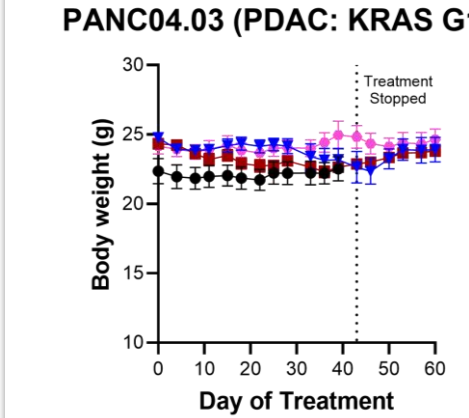
		BH-501284	AMG410
SPR Assay : "Off" state KRAS G12D :: GDP	K_D (nM)	<0.0148	0.119
	T_residence (hr)	>54.2	1.40



Cellular Kinetic Profile



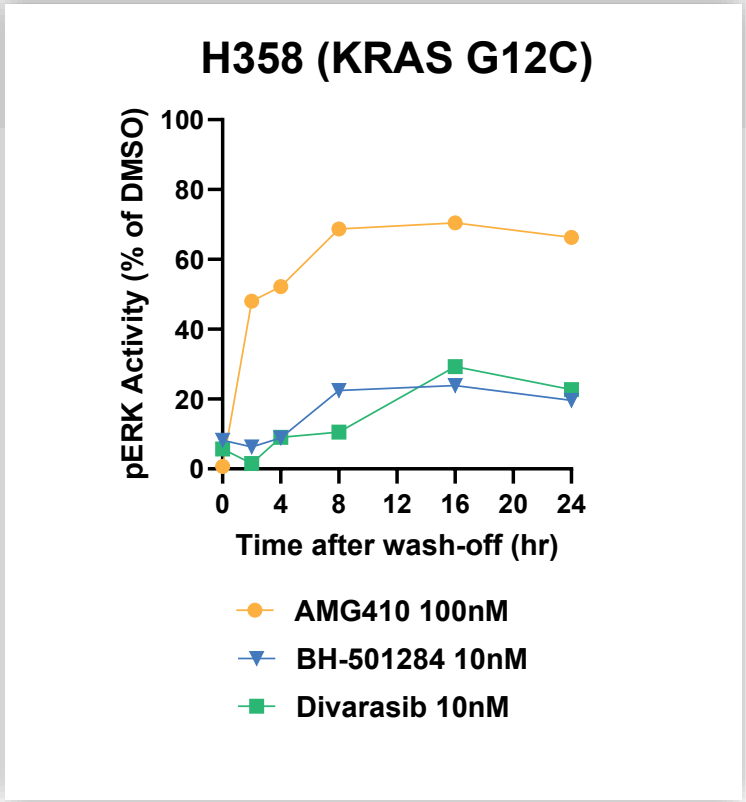


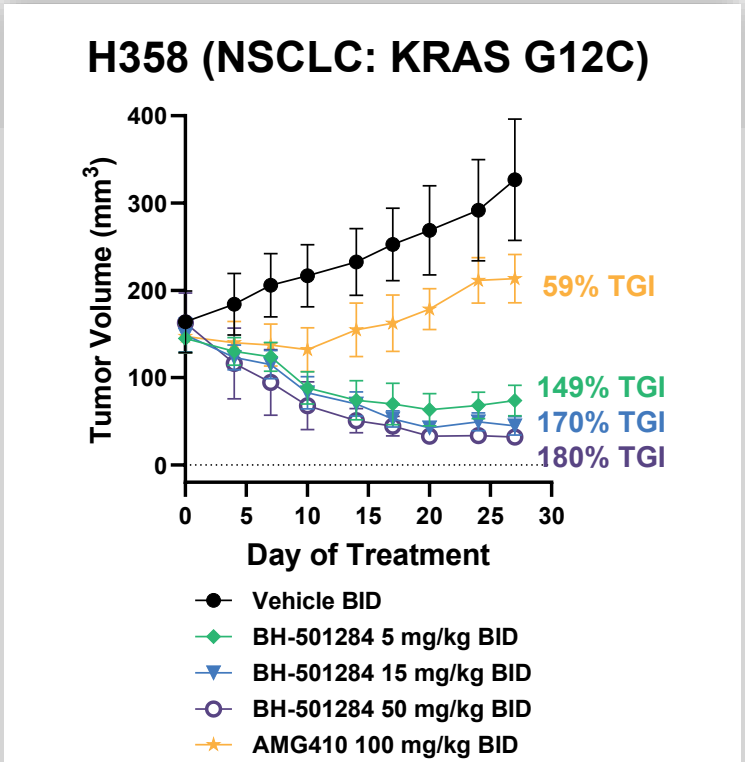


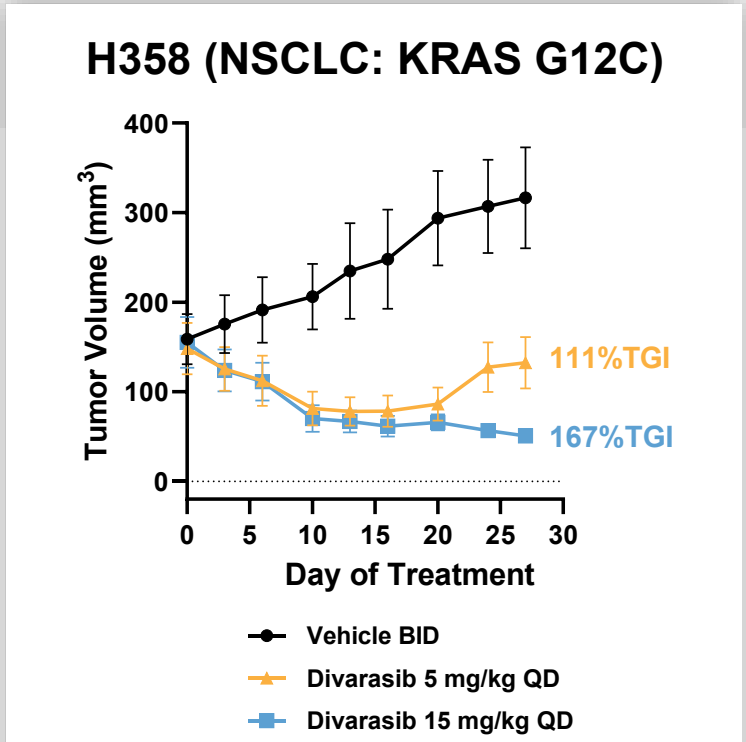
Note: SPR assay was performed at Reaction Biology

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

- In wash-off experiment, BH-501284 sustained pERK inhibition as effectively as divarasib, 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 divarasib, while AMG410 showed limited TGI at high dose







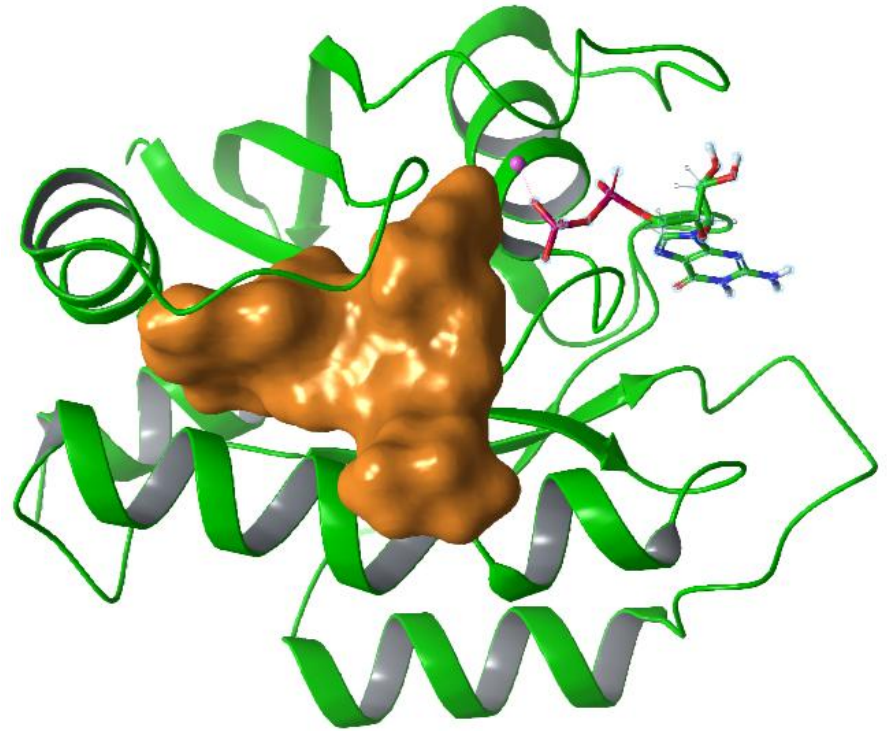
BH-501284
Key Takeaways

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

BLOSSOMHILL THERAPEUTICS