



BBOT

Next-Generation RAS-Pathway Therapeutics

- **Corporate Update**
February 2026



Forward-Looking Statements

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Advancing next generation RAS-pathway targeted small molecules

To accelerate scientific and medical breakthroughs and deliver well-tolerated medicines with greater efficacy and safety to people with the deadliest cancers.

Mission

- **Optimized target coverage** for patients with tumors driven by RAS and PI3K α
- **Synergistic & differentiated portfolio** designed to enable targeted KRAS combinations
- **Multiple** clinical assets with anticipated value inflection points in 2026
- **Financial strength** to fund operations **into 2028**

Differentiated portfolio of precision oncology assets targeting RAS and PI3K α

BBO-8520

First direct inhibitor
of KRAS^{G12C} ON/OFF with
effector blockade function

- ✓ Promising monotherapy data with **65% ORR, 68% 6-mo. PFS, and 83% of patients remaining on treatment** for ≥ 6 mo follow-up; **differentiated tolerability**
- ✓ **Differentiated pembro combination safety** observed at **optimally active dose level** with **favorable liver safety profile compared to pembro alone**
- ✓ Promising early efficacy signals in combination with pembro
- ✓ Encouraging early efficacy signals in patients **with STK11/KEAP1 co-mutations**

BBO-11818

Potent, direct inhibitor
of panKRAS ON/OFF

- ✓ **Partial response (PR) observed in PDAC**
- ✓ Anti-tumor activity across dose levels and tumor types with tumor reductions at higher dose levels
- ✓ **Differentiated safety profile** observed in dose escalation
- ✓ **PK exposure approximately dose proportional**

BBO-10203

Novel RAS:PI3K α Breaker
specifically blocks RAS activation
of PI3K α

- ✓ Potentially **differentiated safety** profile
- ✓ **No observed events of hyperglycemia with no enrollment restrictions on HbA1c and glucose levels**
- ✓ Achieved **target systemic exposure and rapid full target engagement**
- ✓ Recommended dose for expansion has been determined **and combo cohorts are open**

BBOT's portfolio of clinical-stage RAS-pathway inhibitors have extensive commercial opportunities and exciting internal combination potential

BBO-8520
KRAS^{G12C} (ON / OFF)

BBO-11818
panKRAS (ON / OFF)

BBO-10203
RAS:PI3K α Breaker

	BBO-8520 KRAS ^{G12C} (ON / OFF)	BBO-11818 panKRAS (ON / OFF)	BBO-10203 RAS:PI3K α Breaker	
KRAS ^{mut}	NSCLC	KRAS ^{G12C}	KRAS ^{G12X}	KRAS ^{G12C} KRAS ^{G12X}
	CRC		KRAS ^{G12X}	KRAS ^{G12X} KRAS ^{mut}
	PDAC		KRAS ^{G12X}	KRAS ^{G12X}
BrCa			HER2 ^{amp}	HR+ PIK3CA ^{mut}

■ SoC combinations
 ■ Internal BBOT portfolio combinations
 ■ SoC & BBOT portfolio combinations

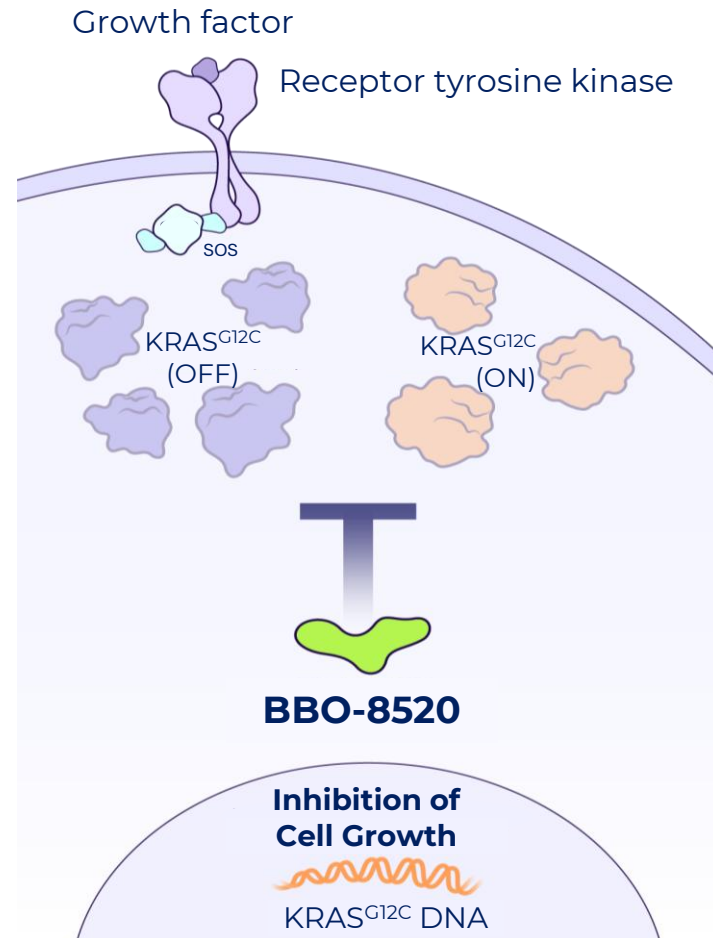
Portfolio designed to enable direct dual inhibition of KRAS ON and OFF states and panRAS inhibition of PI3K α activation, including concurrent inhibition of PI3K α and MAPK through internal combinations

Over 250K annual incident patients in the U.S. across multiple indications

BBO-8520

Dual KRAS^{G12C} ON and OFF Inhibitor

BBO-8520's direct ON/OFF inhibition enables improved Therapeutic Index



- BBO-8520 is uniquely designed to **block effector binding** by inhibiting both the ON and OFF states of KRAS^{G12C}
- Direct ON-state inhibition **drives gains in potency** and **lowers the free drug levels** needed for activity. These attributes may result in:
 - Improved therapeutic index in combination with pembrolizumab in patients with KRAS^{G12C} NSCLC
 - Prevention of adaptive mechanisms of resistance that emerge in response to therapeutic pressure to OFF inhibitors
 - Enable the full benefit of covalent MOA – PK / PD disconnect via sustained pathway inhibition after systemic drug levels decline

ONKORAS-101: Completed Phase 1a dose escalation in monotherapy and PD-1 combination - currently enrolling backfill and expansion cohorts

ONKORAS-101 is enrolling locally advanced and unresectable or metastatic non-small cell lung cancer with a KRAS^{G12C} mutation

Safety profile observed to date has enabled combination with pembrolizumab at an active monotherapy dose

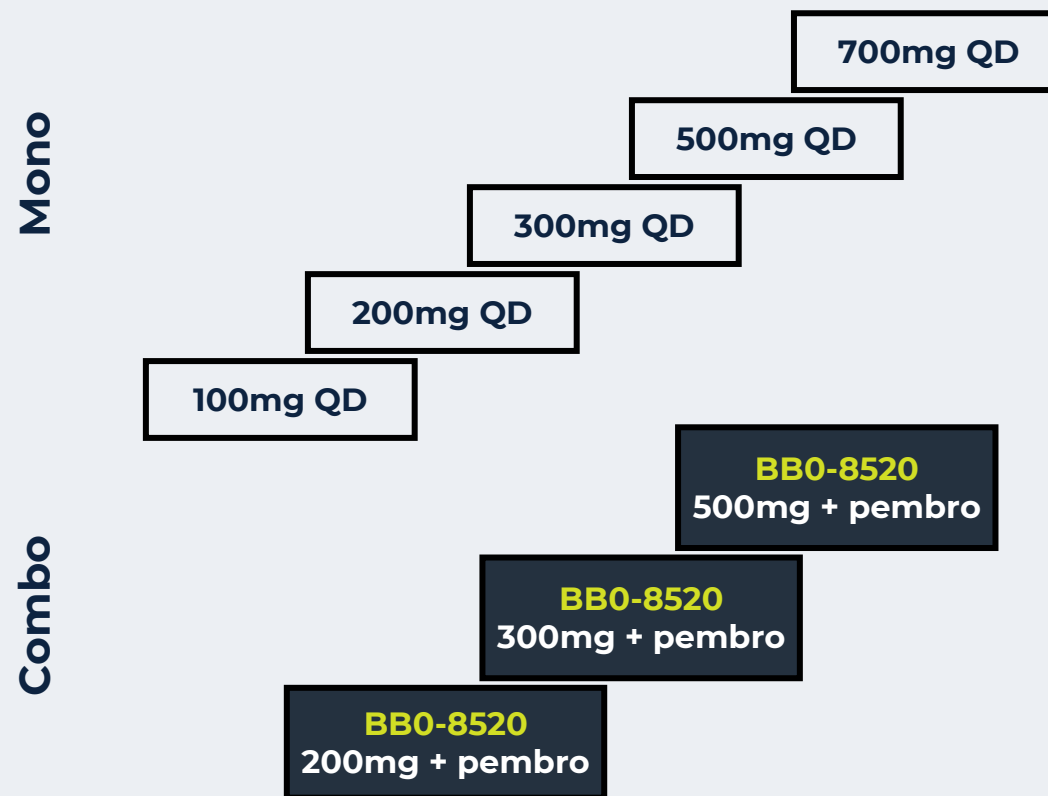
Monotherapy and pembrolizumab combination cohort expansions are currently enrolling

Key endpoints include:

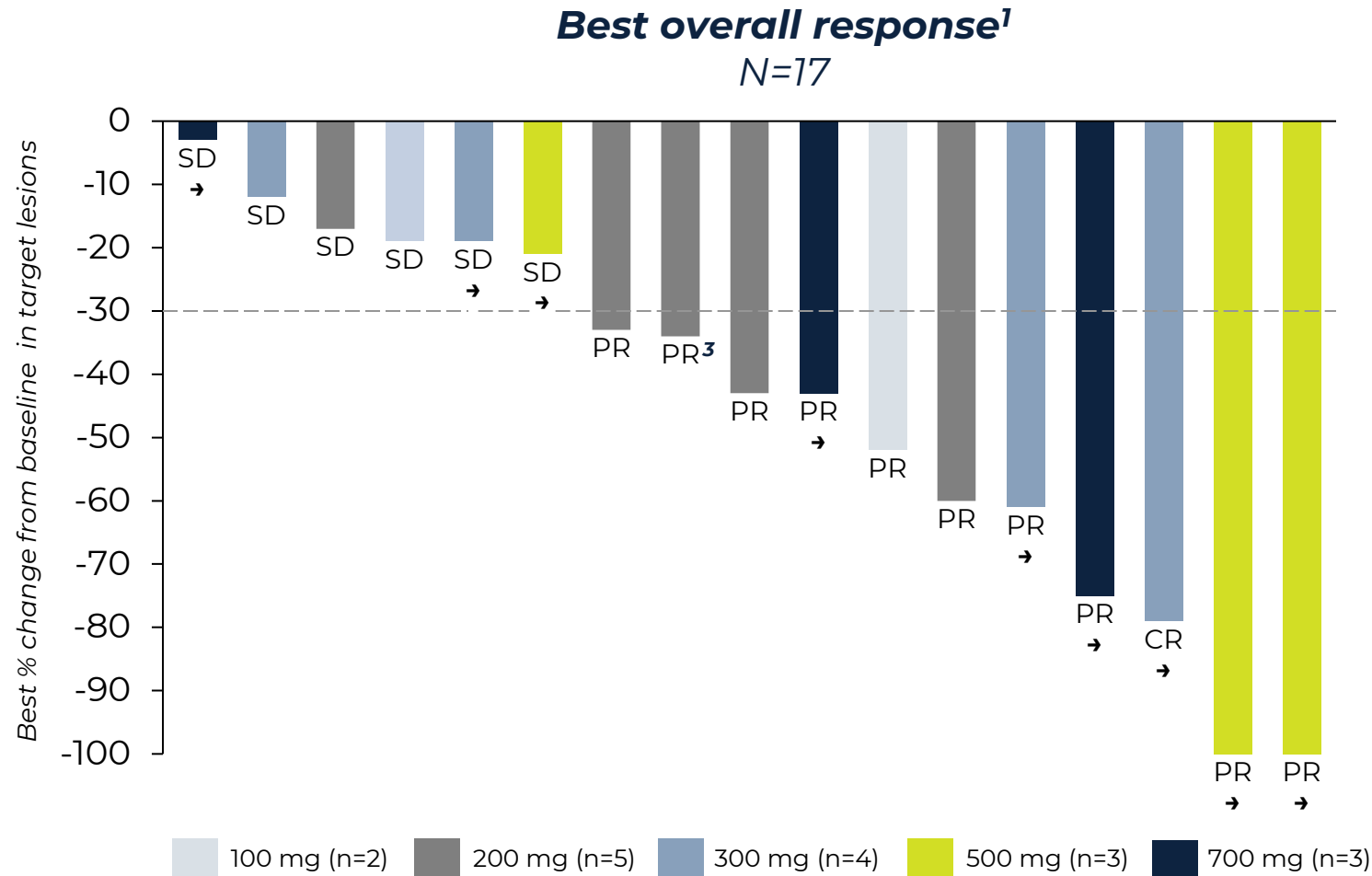
- Safety and tolerability
- Pharmacokinetics
- Anti-tumor activity

ONKORAS-101 Phase 1a Dose Escalation

Monotherapy & combination with pembrolizumab



Robust monotherapy activity across escalation dose levels in previously treated KRAS^{G12C} NSCLC with no prior G12C inhibitor experience

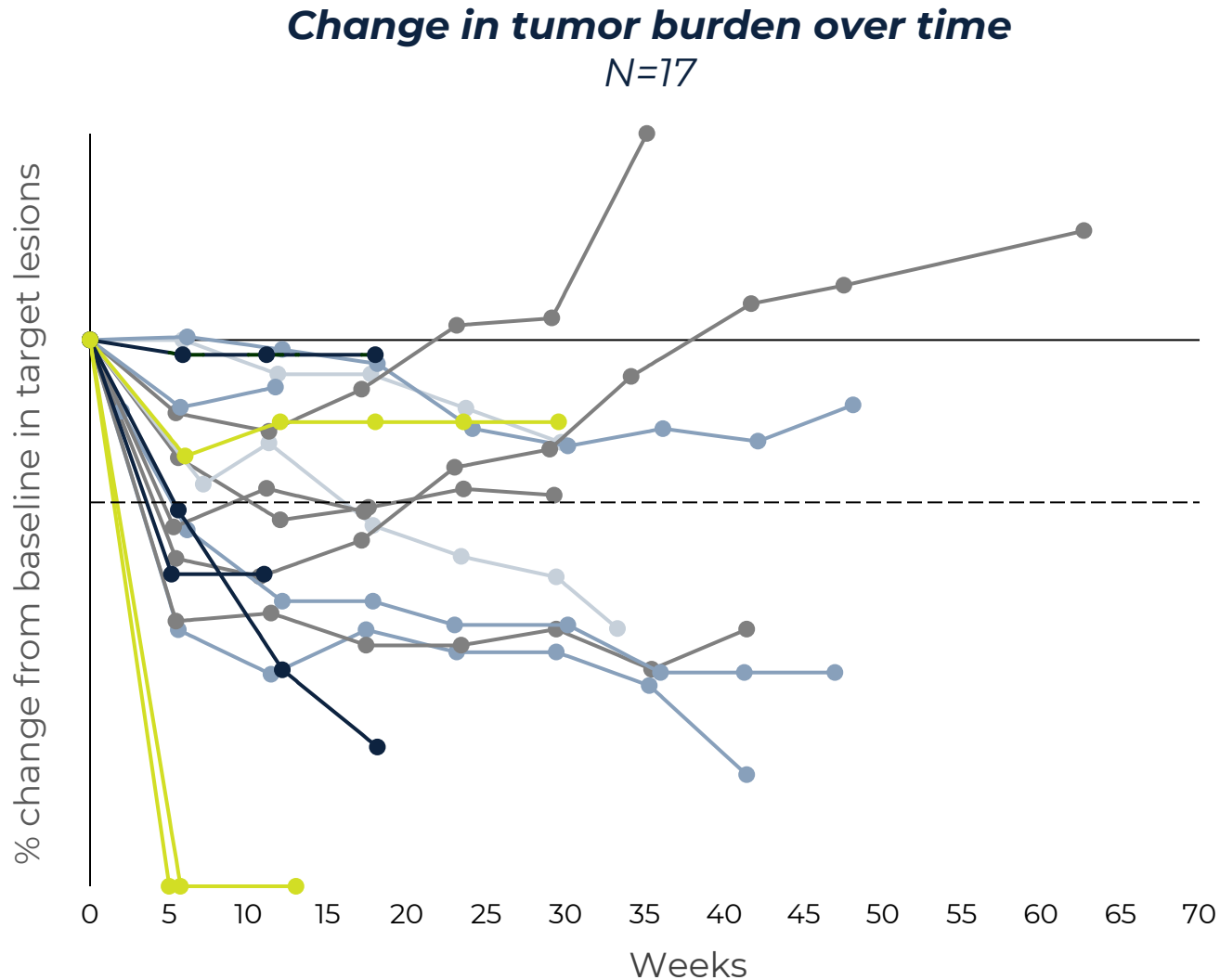


Efficacy data summary

Tumor response	
ORR, % (n)	65% (11/17)
DCR ² , % (n)	100% (17/17)

Note: → indicates patient is still on study treatment 1) Efficacy evaluable defined as at least two on treatment scans, 2) Disease control rate (DCR) includes complete responses (CR), partial response (PR) and stable disease (SD); 3) Patient with partial response not confirmed but treated beyond radiographic progression for additional 4 months due to clinical benefit
Source: ONKORAS-101 DCO Nov 15, 2025

Responses appear durable with 83% of patients eligible for 6-month follow up remaining on treatment for at least 6 months



Evidence of response durability

- 10/12 (83%) of patients with at least 6-month follow-up remained on treatment greater than 6 months
- 6-months PFS 68%

BBO-8520 has shown a generally differentiated safety profile with no Grade 3 or higher liver toxicity

TRAEs reported in >15% patients and TRAEs of interest

N=37

AE term	All Grades	Grade ≥3
Any TRAE	31 (84%)	6 (16%)
NAUSEA	23(62%)	1 (3%)
DIARRHEA	19 (51%)	3 (8%)
VOMITING	13 (35%)	0
FATIGUE	13 (35%)	1 (3%)
ANOREXIA	6 (16%)	0
AE of Interest		
AST INCREASED	3 (8%)	0
ALT INCREASED	2 (5%)	0

Monotherapy safety profile summary

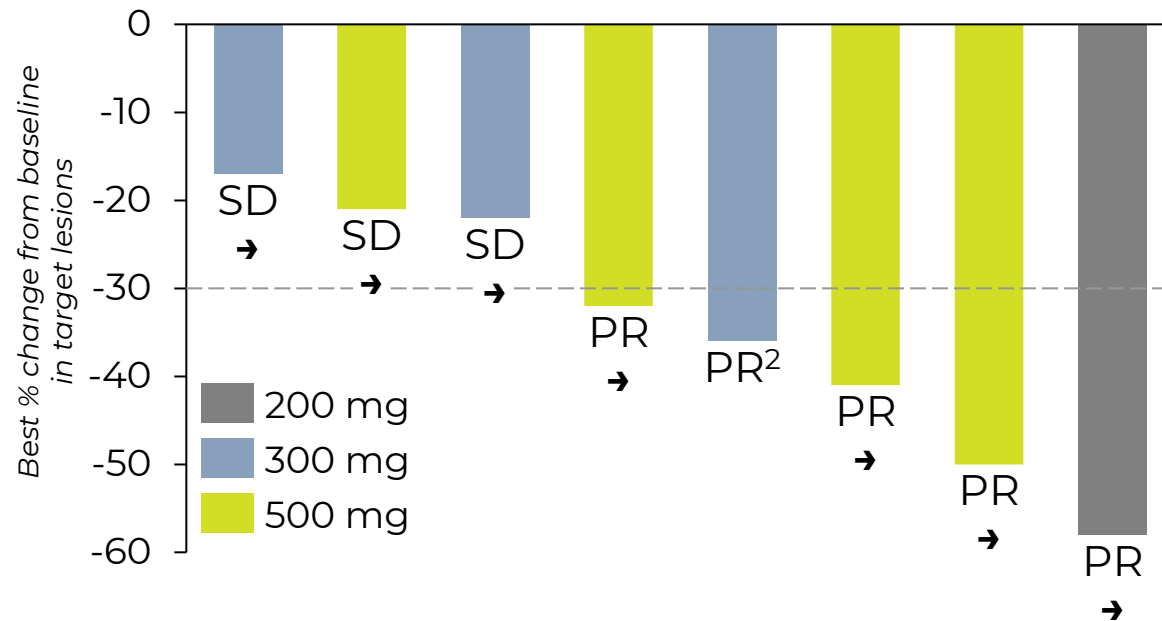
- BBO-8520 safety profile as monotherapy appears generally tolerable and manageable, with a potentially differentiated liver toxicity profile
- **No DLTs; no Grade ≥4 AEs; no treatment-related SAEs**
- Nausea, vomiting, and diarrhea were the most common treatment-related AEs
 - Instances of G3 diarrhea were mostly associated with suboptimal management based on investigator assessment
- **AST/ALT elevations occurred at low frequency and were low grade, transient, and clinically asymptomatic**

Promising data in combination with pembrolizumab in patient population largely pretreated with both ICI and G12Ci

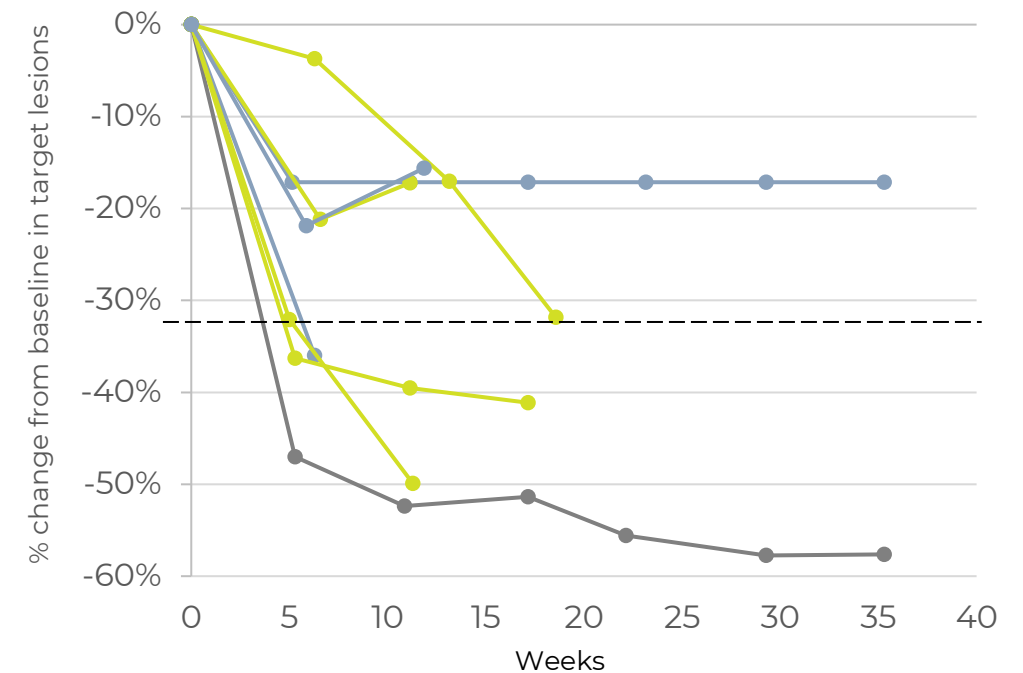
ONKORAS-101 efficacy evaluable¹ patients in combination with pembrolizumab

N=8

Best overall response



Change in tumor burden over time



All patients remain on study treatment, except for one patient who withdrew consent despite PR

G12C-treated	Y	Y	Y	Y	N	N	N	Y
ICI treated	Y	Y	Y	N	N	N	N	Y
Prior LoT³	4	2	2	1	0	0	0	4
TPS	80%	1%	7%	30%	1%	2%	98%	>90%

Note: → indicates patient is still on drug as of November 15, 2025; 1) Efficacy evaluable defined as at least two on treatment scans; 2) Patient decision to come off study despite of PR; 3) Number of prior lines of therapy in the metastatic setting
Source: ONKORAS-101 DCO Nov 15, 2025

BBO-8520 has a potential best-in-class safety profile in combination with pembrolizumab

TRAEs reported in >1 patient and TRAEs of interest

N=15

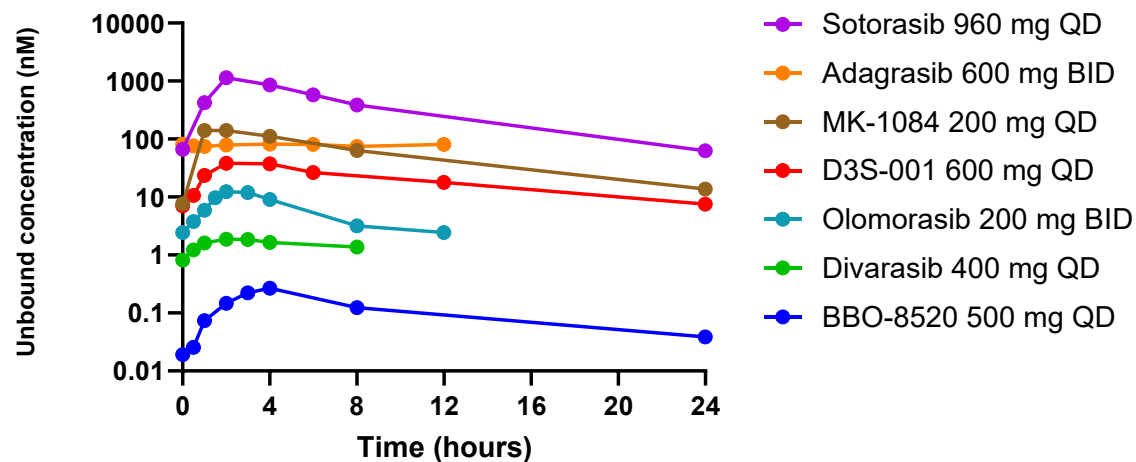
AE term	All Grades	Grade ≥3
Any TRAE	7 (47%)	2 (13%)
NAUSEA	6 (40%)	0
DIARRHEA	5 (33%)	1 (7%)
VOMITING	4 (27%)	0
FATIGUE	3 (20%)	0
AE of Interest		
AST INCREASED	1 (7%)	1 (7%)
ALT INCREASED	1 (7%)	1 (7%)

Combination safety profile summary

- BBO-8520 safety profile in combination with pembrolizumab appears generally tolerable and manageable, with potentially differentiated liver toxicity profile
- The only G3 AST/ALT was considered by PI to be mainly due to co-medications and LFT increases do not seem to be dose dependent
 - This patient was previously treated with both sotorasib and olomorasib in addition to pembrolizumab, achieved confirmed PR, and has now been on study for >36 weeks with AST/ALT resolved

BBO-8520's ON state inhibition enables efficacy with relatively lower exposure resulting in superior therapeutic index compared to OFF inhibitors...

Relative exposure for BBO-8520 vs. KRAS^{G12C} OFF inhibitors



Relative AUC fold over BBO-8520 500 mg QD dose

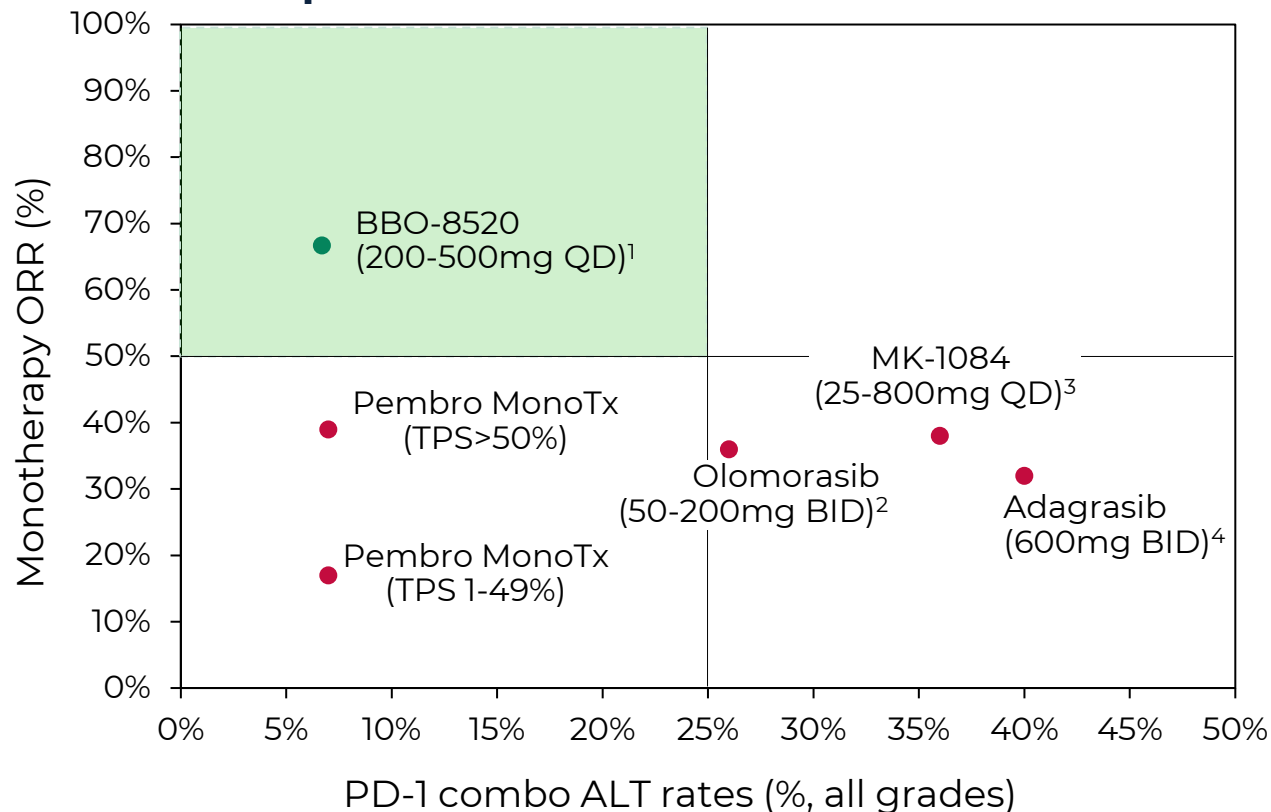
Compound	AUC fold over BBO-8520 500 mg QD dose
Sotorasib	2700
Adagrasib	742
MK-1084	424
D3S-001	191
Olomorasib	58
Divarasib	13

- BBO-8520 exposure is approximately dose proportional achieving predicted efficacious levels at 200 mg and above
- Compared with systemic exposure of BBO-8520 500mg QD, exposures of adagrasib/sotorasib are >700 fold higher, MK1084/D3S-001 are >150 fold higher and olomorasib/divarasib are >10 fold higher

BBO-8520's significantly lower exposure than OFF inhibitors may be due to the ON state mechanism creating a PK/PD disconnect

... which may enable its combination with pembrolizumab at active dose levels to differentiate in earlier settings

Monotherapy activity vs. ALT elevations in combination with pembrolizumab



OFF inhibitor / pembrolizumab combinations

- All OFF inhibitors have had to **significantly reduce their dose to sub-optimal levels** when combined with pembrolizumab **due to liver toxicity**
 - This suggests that monotherapy efficacy for their combination dose level **may be lower than the benchmarks shown**
- Even at the sub-optimal dose levels, patients **experienced grade 3 toxicities** ranging from 30% to 70%

BBO-8520's superior safety profile positions it well to potentially achieve best-in-class status, by serving as the decisive factor for patient enrollment and physician adoption

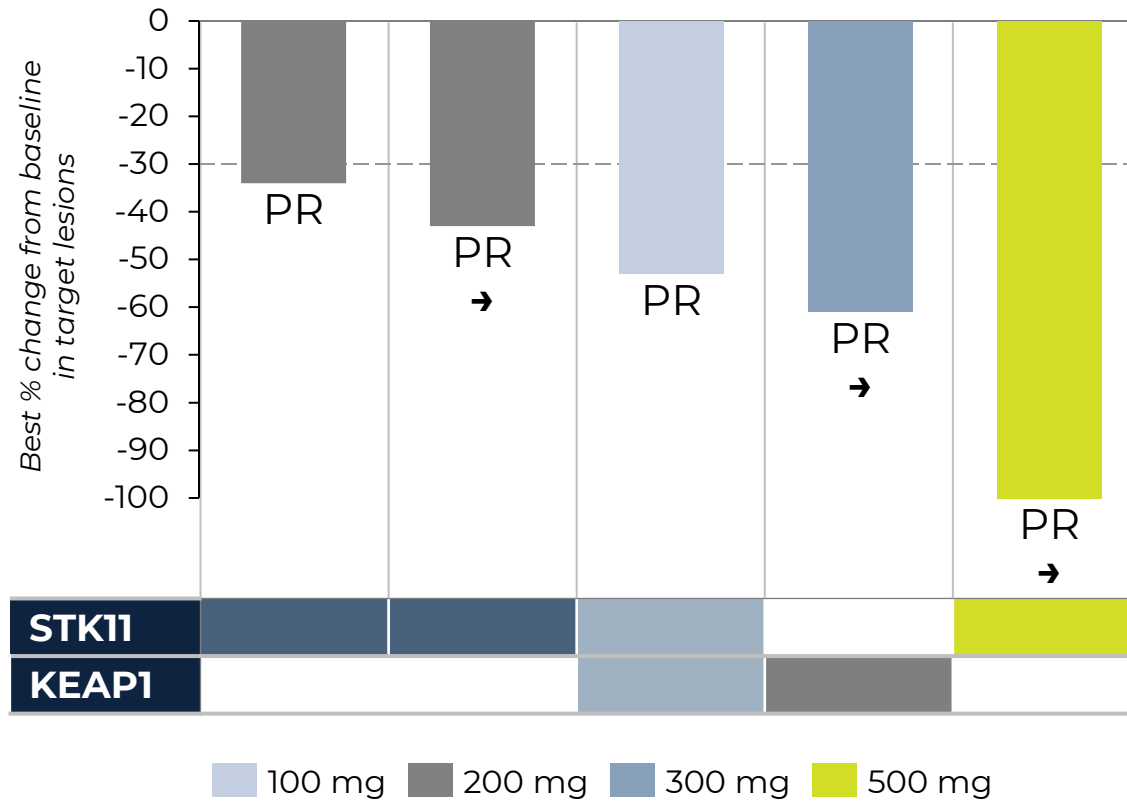
Note: 1) BBO-8520 dose levels tested in combination with pembrolizumab as of November 15, 2025; 2) Dose ranges listed for monotherapy efficacy data, safety data is derived from 50-100mg BID; 3) Dose ranges listed for monotherapy efficacy data, safety data derived from 25-400mg QD; 4) 600mg BID listed for monotherapy efficacy data, safety data derived from 400mg BID

Source: De Castro et al., 2022 JOCS; Procedure No. EMEA/H/C/003820/II/0057; Burns et al., 2024 ASCO; Johnson et al., 2025 WCLC; Sacher et al., 2025 ASCO; Cobb et al., 2024 ESMO AoO; Negrao et al., 2025 WCLC; Barlesi et al., Lancet 2025; Jänne et al., ASCO 2025; ONKORAS-101 DCO Nov 15, 2025

Early responses in STK11 / KEAP1 mutants – a key underserved portion of NSCLC patients

Best overall response by KEAP1 / STK11 mutation status

N=5

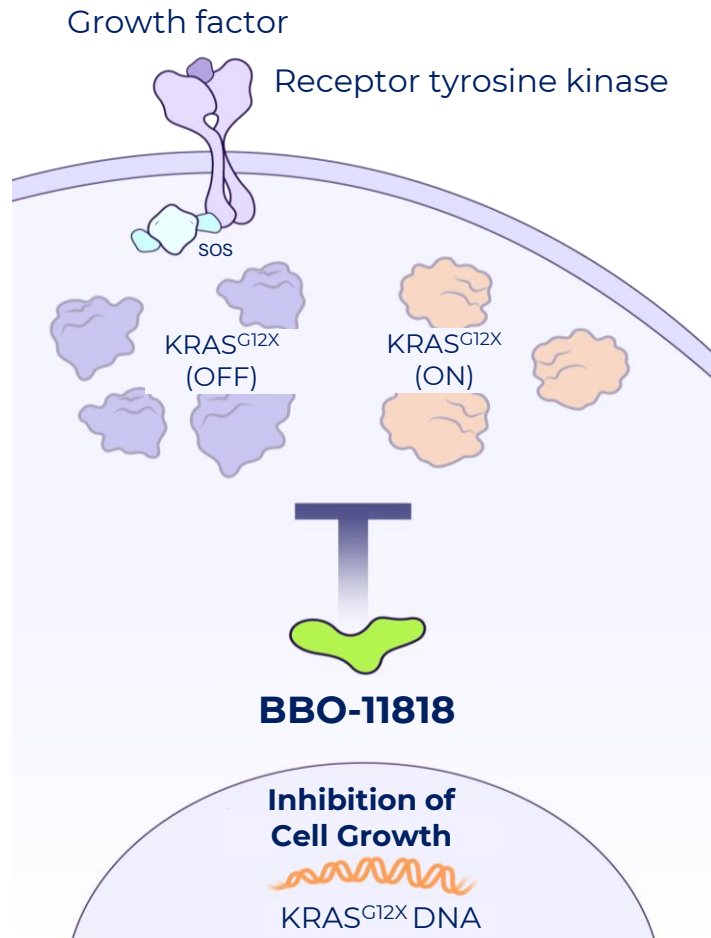


- **Early efficacy signals seen in resistant STK11/KEAP1 co-mutants;** all five initial patients showing a response
- **STK11 / KEAP1 mutations** are highly prevalent in KRAS^{G12C} NSCLC, **occurring in 25-35% of patients**
- Patients with STK11 / KEAP1 co-mutations show intrinsic resistance to ICI, leading to inferior clinical outcomes across TPS scores and regimens
- KRAS^{G12C} OFF inhibitors perform poorly in these patients with divarasisib showing ORR of 25-33% compared to ~60% in WT patients

BBO-11818

Dual panKRAS ON and OFF inhibitor

BBO-11818's direct ON/OFF inhibition enables effector binding blockade



- BBO-11818 is an orally bioavailable, reversible panKRAS inhibitor with activity in both the ON and OFF states
- Structure based design built upon BBO-8520 innovation
 - Direct ON-state inhibition enables effector binding blockade
 - Highly selective (>500x) for KRAS, spares H- and N-RAS
 - Single-digit nM activity with no shift in potency between pERK and 3D viability
- Strong monotherapy activity and promising combination potential with anti-EGFR mAb and BBOT's RAS:PI3K α Breaker BBO-10203 in mouse models

KONQUER-101 phase 1a monotherapy dose escalation ongoing; expansion and combination cohorts are planned for 2026

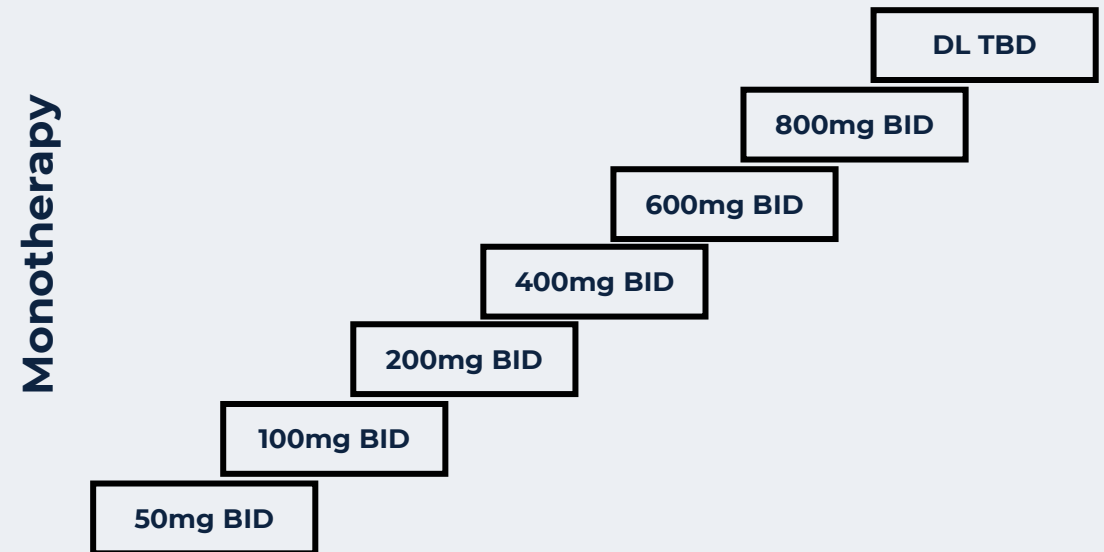
Monotherapy dose escalation is ongoing

BBO-11818 will be evaluated in combination with pembrolizumab, cetuximab and chemo in KRAS mutant NSCLC, CRC, and PDAC

Key endpoints include:

- Safety and tolerability
- Anti-tumor activity
- Pharmacokinetics

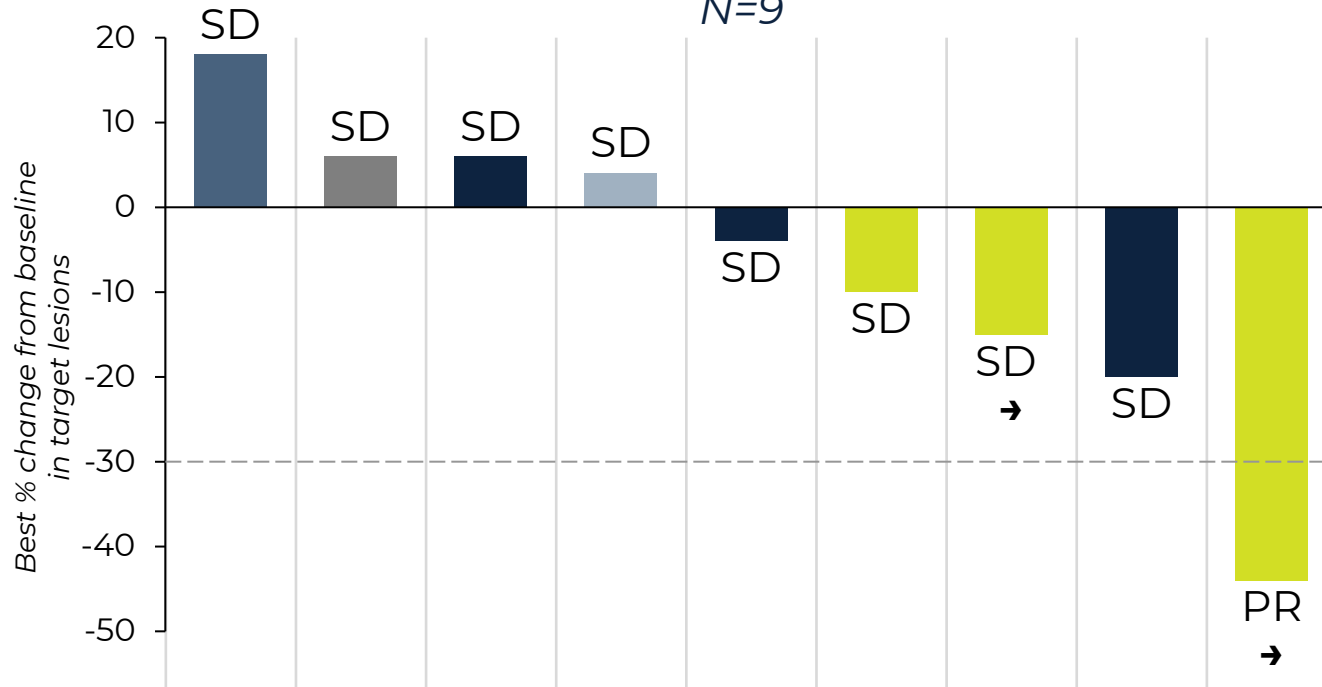
KONQUER-101 Phase 1a Dose Escalation Monotherapy



Initial cohorts demonstrate anti-tumor activity at predicted efficacious dose levels across tumor types

Best overall response

N=9



Tumor type	PDAC	PDAC	PDAC	Rectum	PDAC	PDAC	NSCLC	NSCLC	PDAC
Mutation	G12V	G12D	G12V	G12D	G12D	G12D	G12D	G12V	G12D
Prior LoT ¹	1	3	3	3	4	3	4	7	2

- BBO-11818 has demonstrated encouraging anti-tumor activity in heavily pretreated PDAC and NSCLC patients
- 1 confirmed² PR in a patient with PDAC with a 56% tumor reduction at 600mg BID (n=3)

- 50 mg (n=1)
- 100 mg (n=1)
- 200 mg (n=1)
- 400 mg (n=3)
- 600 mg (n=3)

Note: → indicates patient is still on drug as of December 10, 2025; 1) Number of prior lines of therapy in the metastatic setting ; 2) PR was unconfirmed at the time of data cutoff but was subsequently confirmed.

Source: KONQUER-101 DCO Dec 10, 2025

BBO-11818 appears generally tolerable and manageable

TRAEs reported in >1 patient

N=13

AE term	All Grades	Grade 1/2	Grade 3
Nausea	8	7	1
Diarrhea	6	5	1
Vomiting	5	5	-
Dry Mouth	3	3	-
Fatigue	5	5	-
Anorexia	3	3	-
Hypomagnesemia	2	2	-
Dysgeusia	2	2	-

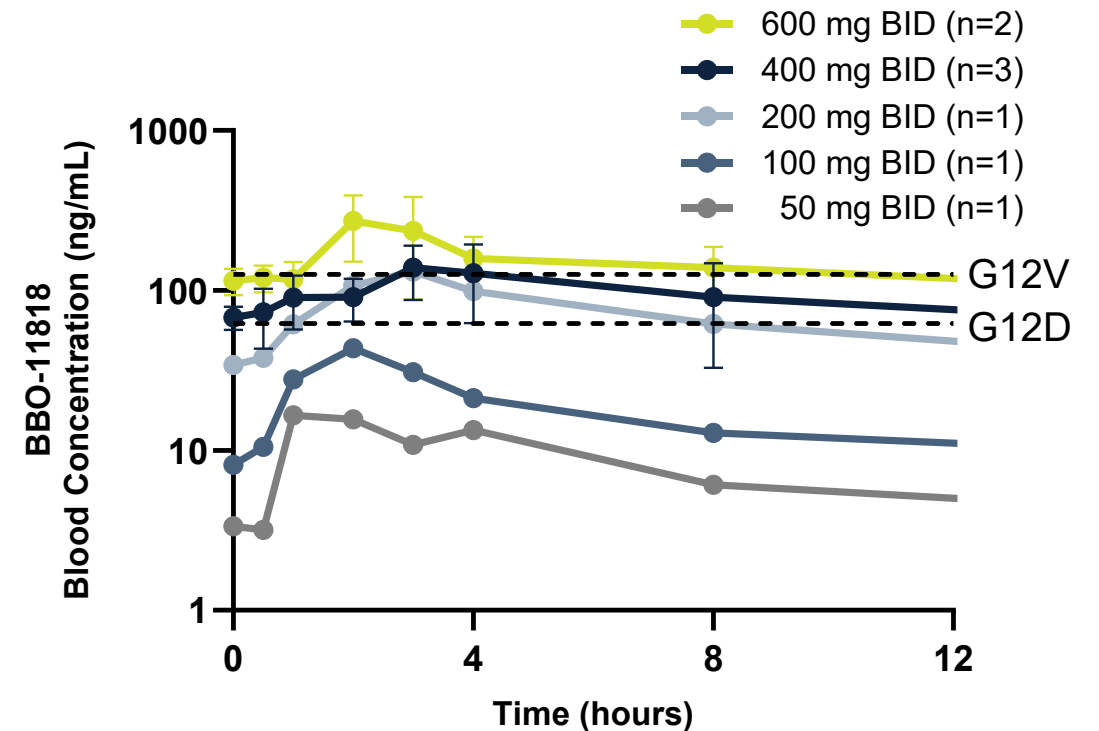
Monotherapy safety data

BBO-11818 monotherapy (N=13) appears generally tolerable and manageable

- No DLTs
- TRAEs mainly GI related
- 2 Gr 3 GI events (diarrhea and nausea) in patients with pre-existing GI conditions

BBO-11818 PK exposure was approximately dose proportional, with 600 mg BID covering G12D and G12V mutant alleles

C1D15 (Steady State) in patients



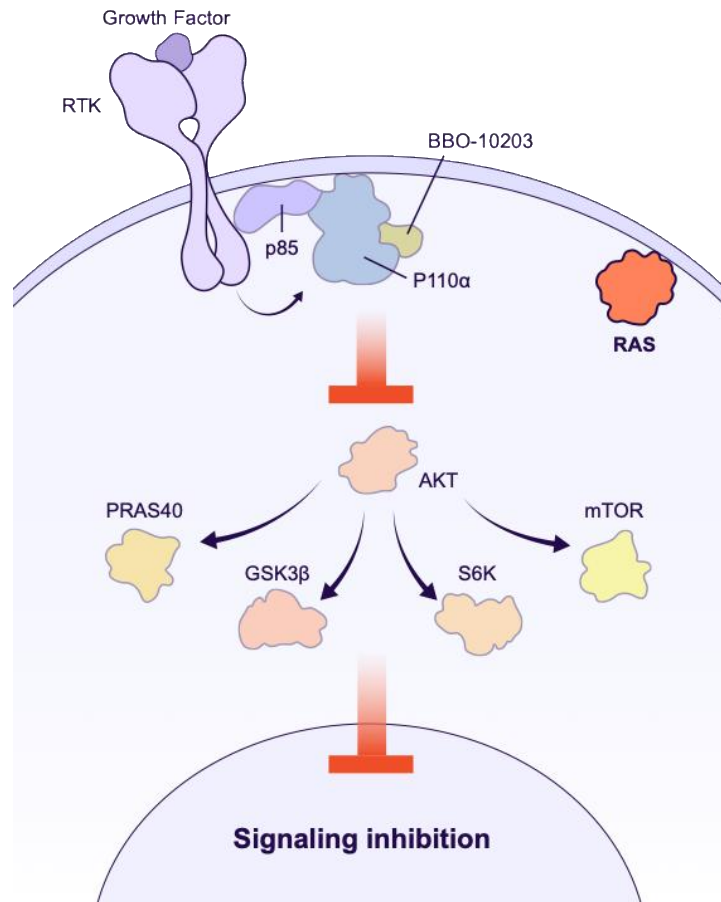
Note: In G12D and G12V CDX mouse models the target concentrations correspond to tumor regression following daily oral BBO-11818 treatment as estimated using a Simeoni PK-TGI model

BBO-10203

Breaker of the RAS:PI3K α interaction

BBO-10203 utilizes a novel MOA that is designed to inhibit the physical interaction between RAS and PI3K α , disrupting RAS-driven PI3K α /AKT signaling

BBO-10203 MoA



BBO-10203's novel MoA leverages a mutation agnostic approach

- Binds specifically to the RBD of PI3K α
- Does not inhibit the kinase activity of PI3K α
- Blocks binding of K-, H-, and N-RAS to PI3K α
- Agnostic to mutational status of either partner
- Excellent brain penetration in mice ($K_{p,uu} = 0.468$)
- **Preclinically no hyperglycemia observed at 3x efficacious doses**
- Consistent with all inhibitors of PI3K α -AKT signaling, development path will be as combination agent

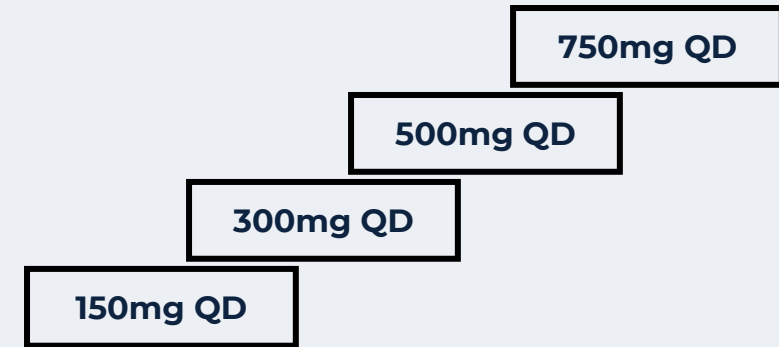
BREAKER-101 has completed Phase 1a dose escalation in monotherapy

- Monotherapy dose escalation is complete
- Key endpoints include:
 - Safety and tolerability
 - Pharmacokinetics
 - Anti-tumor activity
- Combination cohorts with fulvestrant + ribociclib in HR+ HER2- PIK3CA mutant BrCa and with both BBO-8520 and BBO-11818 in KRAS mutant tumors are planned

500mg QD has been selected as RDE and 3 combination cohorts have been initiated

BREAKER-101 Phase 1a Dose Escalation *Monotherapy & combination*

Monotherapy

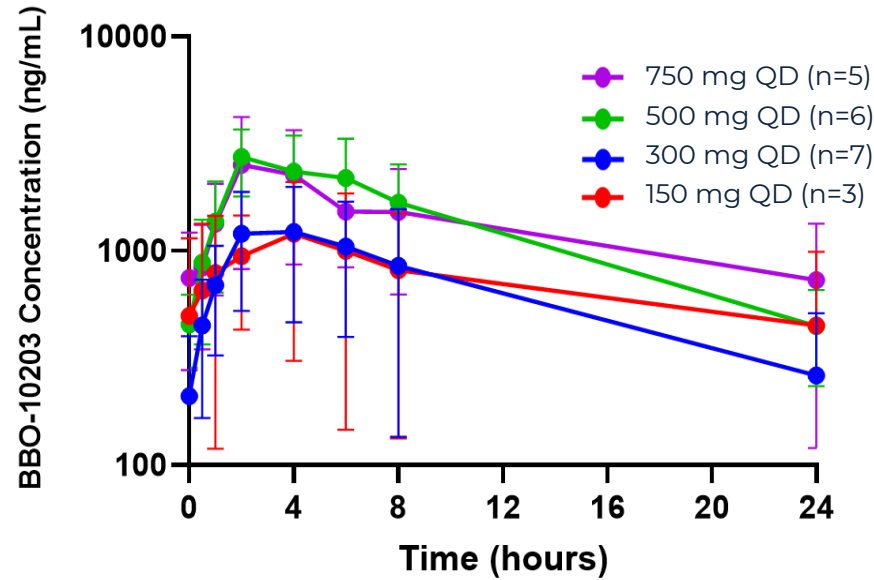


Open combination cohorts

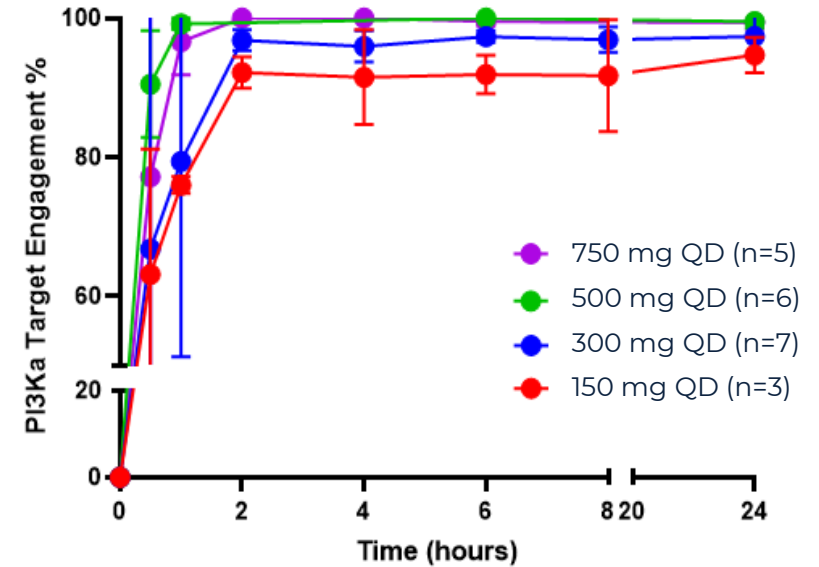
BrCa HER2+	BB0-10203 + trastuzumab
BrCa HR+ / HER2- PIK3CA ^{mut}	BB0-10203 + fulvestrant
CRC KRAS ^{mut}	BB0-10203 + FOLFOX + bevacizumab

BBO-10203 exposure achieved predicted efficacious levels with complete target engagement across all dose levels at steady-state

C1D15 (Steady State) PK in patients



Day 1 target engagement by dose



- Predicted efficacious exposure achieved in patients at 500 mg QD
- Rapid target engagement observed across all dose levels with complete target engagement at steady-state

BBO-10203 demonstrated a potentially differentiated safety profile in heavily pretreated patients

TRAEs by Grade in >10% patients

N=32

AE term	Monotherapy N=24		Trastuzumab Combination N=4		FOLFOX/Bev Combination N=4	
	All Grades	Grade ≥3	All Grades	Grade ≥3	All Grades	Grade ≥3
Any TRAE	17 (71%)	0	4	0	3	0
Diarrhea	7 (29%)	0	1 (25%)	0	0	0
Fatigue	6 (25%)	0	1 (25%)	0	0	0
Nausea	6 (25%)	0	1 (25%)	0	2 (50%)	0
Decreased Appetite / Anorexia	5 (21%)	0	0	0	0	0
Vomiting	3 (13%)	0	1 (25%)	0	1 (25%)	0
Rash / Dermatitis Acneiform	3 (13%)	0	0	0	0	0

Monotherapy and combination safety profile has potential to be a **key differentiator compared to other PI3K α -targeting agents**

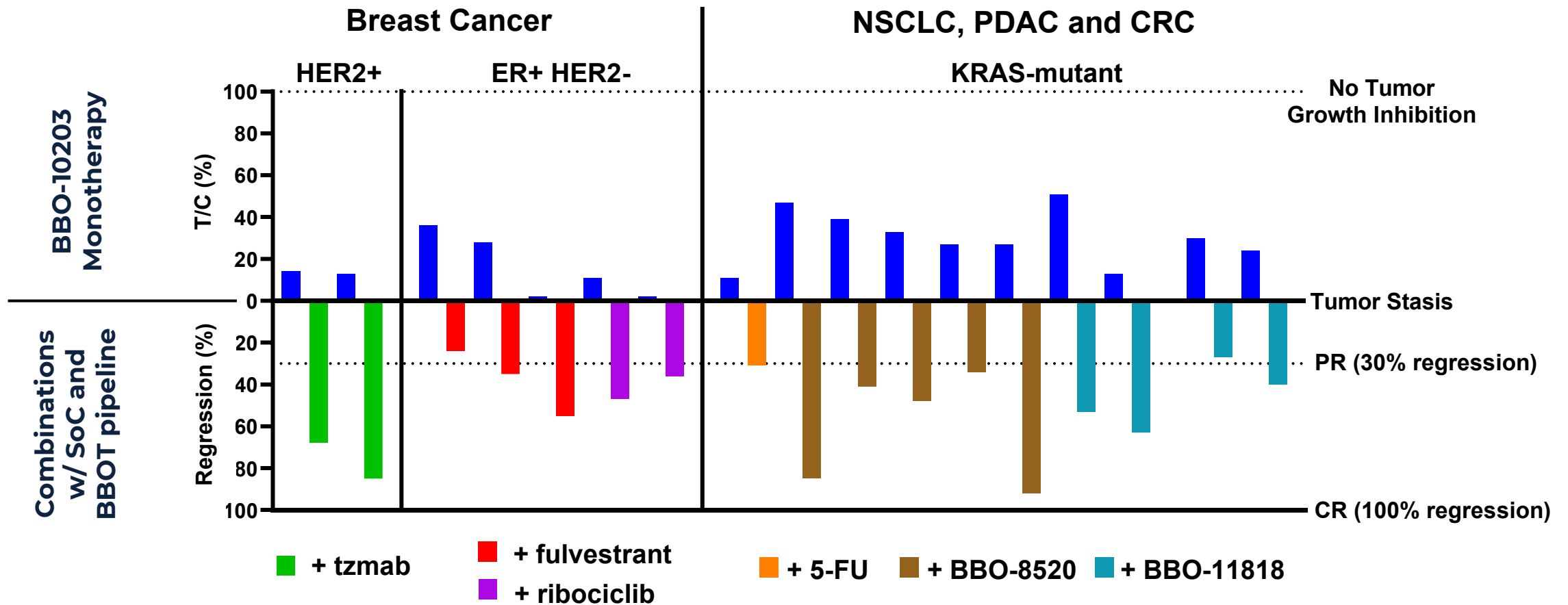
- **No** DLTs and treatment-related SAEs
- **No** hyperglycemia
- **No** Grade ≥3 TRAEs except for 1 incidence of asymptomatic hypokalemia (lab abnormality)
- **No** dose reductions
- Early combination data with trastuzumab and FOLFOX/Bev appears generally tolerable with **no grade 3+ TRAE**

Efficacy data

- Clinical benefit was observed in patients with CRC (>80% 3L+) and HR+ BC who were previously heavily treated and tumor reductions observed in some patients
- Monotherapy DCR: 62% (13/21)¹

Note: 1) Disease control rate (DCR) includes complete responses (CR), partial response (PR) and stable disease (SD) in efficacy evaluable patients defined as at least one on-treatment scan
Source: BREAKER-101 DCO Dec 10, 2025

BBO-10203 has shown robust tumor regression in combination across a panel of breast cancer, and KRAS mutant CRC, NSCLC, and PDAC models



Summary and Upcoming Catalysts

BBO-8520

- ✓ Encouraging efficacy, safety and early durability data
- ✓ Combination with pembrolizumab at an active dose with early efficacy data and a distinct tolerable safety profile
- ✓ Encouraging efficacy signals in resistant STK11/KEAP1 co-mutants

BBO-11818

- ✓ Demonstrated a partial response (PR) in pancreatic cancer; anti-tumor activity across dose levels and tumor types with tumor reductions at higher dose levels
- ✓ Differentiated safety profile observed in dose escalation

BBO-10203

- ✓ Demonstrated a potentially differentiated safety profile without any observed events of hyperglycemia and without any enrollment restrictions on HbA1c levels

Upcoming
Catalysts
2H 2026

Additional pembrolizumab combination efficacy and safety data

Additional monotherapy and combination efficacy and safety data

Combination data in HER2+ BC, HR+/HER2- PIK3CA^{mut} BC and KRAS^{mut} CRC

Strong Balance Sheet with Cash Runway Projected into 2028

Thank You