BBOT

Next-Generation RAS-Pathway Therapeutics

Corporate Update

September 2026

BBOT

Forward-Looking Statements

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Advancing next generation RAS-pathway targeted small molecules

Mission	Bring hope to patients with mutant KRAS-driven malignancies by translating innovative science into novel medicines

- **KRAS is ON** enabled by direct effector blockade mechanism
- **Selective targeting** of RAS pathway oncogenic drivers designed to enable superior therapeutic index
- **Unique approach to targeting the MAPK and PI3Ka** signaling pathways designed to enable **superior response and durability potential**
- **Strong research pipeline** focused on breakthrough science

Financial Strength	Flexibility to fund operations into 2028 including full phase 1 development across all three clinical programs

BBOT is developing three Phase 1 programs; all three programs have generated clinical data supporting further development

Program / Target	Previously disclosed data	Clinical achievements
BBO-8520 **$KRAS^{G12C}$ (ON / OFF)**	▪ 65% ORR monotherapy (N=17) in 2L+ G12C inhibitor naïve NSCLC, 68% 6-mo. PFS, and 83% of patients remaining on treatment for >6mo follow-up ▪ Differentiated pembrolizumab combination (N=15) safety data observed at optimally active dose level with favorable liver safety profile	✓ *Compelling monotherapy efficacy profile* ✓ *Differentiated liver toxicity in combination with pembrolizumab at active doses*
BBO-11818 **Pan-KRAS (ON / OFF)**	▪ Anti-tumor activity across dose levels and tumor types with tumor reductions and partial response at higher dose levels ▪ Differentiated safety profile (N=13) observed in dose escalation ▪ PK exposure approximately dose proportional	✓ *Highly differentiated safety profile vs. panRAS* ✓ *Dose-proportional PK & preliminary evidence of clinical actviity*
BBO-10203 **RAS:PI3Kα Breaker**	▪ N=32 patients with no observed events of hyperglycemia and no enrollment restrictions on HbA1c or glucose ▪ Achieved target systemic exposure and rapid full target engagement	✓ *Achieved predicted efficacious exposure & full target engagement at 500 mg QD* ✓ *Differentiated safety profile vs. PI3Kα inhibitors*

Note: NSCLC, non-small cell lung cancer; PK, pharmacokinetics; ORR, objective response rate; PFS, progression-free survival; QD, once daily; 2L+, second line or later; G12Ci, KRAS G12C inhibitor; HbA1c, hemoglobin A1c

Q3 2026 corporate update summary: Strategic prioritization

Allocating capital to opportunities with the highest probability of success, clear development path, and greatest potential patient benefit

Strategic Focus

A **BBO-8520** and pembrolizumab combination in 2L+ G12C inhibitor experienced $KRAS^{G12C}$ NSCLC patients

B **BBO-11818** and **BBO-10203** +/- standard of care in $KRAS^{mut}$ cancers

BBO-8520 in 1L NSCLC and BBO-10203 in breast cancer have been deprioritized based on evolving treatment landscape, competitive crowding, & portfolio dynamics, BBOT has no near-term plans for further investment

BBO-8520 in 2L+ G12C inhibitor experienced patients in combination with pembrolizumab

A

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

NSCLC ~21K

~21,000 incident KRAS^{G12C} NSCLC patients, US 2026[2]

Strong early efficacy signals

In G12C inhibitor experienced patients, BBO-8520 + pembrolizumab has shown 75% ORR at 500mg QD and 53% ORR across dose levels[1]

- **Growing market**
  Potential G12C OFF inhibitor approvals in 1L are expected to change SoC and create a significant G12C inhibitor experienced 2L market
- **Unmet need**
  Docetaxel delivers 9-13% ORR and 3.8-4.5 months[3] PFS in the G12C inhibitor naive setting
- **Competitive whitespace**
  No approved targeted agents and no announced or ongoing registrational trials in this segment

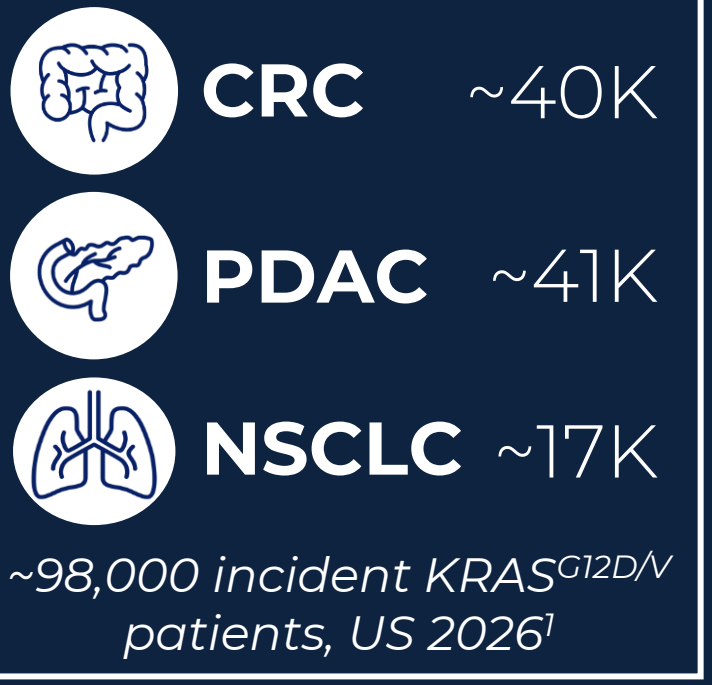
Notes: 1) ONKORAS-101 DCO June 1, 2026, analysis set includes patients with at least 12 weeks of follow-up and at least 1 post-baseline radiographic disease, all patients TPS≥1% and includes confirmed and unconfirmed responses; 2) ACS Cancer Statistics 2026 / KRAS variant prevalence: Lee et al., npj Precis Oncol 6(1):91 (2022); 3) CodeBreaK 200 / KRYSTAL-12 (NCT04685135)

BBO-11818 and BBO-10203 +/- standard of care combinations in KRAS^{mut} cancers

B

BBO-11818 Pan-KRAS(ON/OFF) inhibitor

BBO-10203 RAS:PI3Kα breaker



BBOT is uniquely positioned to fully inhibit MAPK and PI3Ka signaling in KRAS^{mut} tumors

- **Combination enrollment ongoing**
  Multiple combination cohorts are enrolling across CRC & PDAC
- **Clinical data on both molecules**
  Demonstrated potential based on clinical safety, exposure, and early efficacy data
- **Differentiated CRC opportunity**
  Potential to address CRC efficacy in combination with PI3Ka and / or EGFR inhibition based on single-agent toxicity profiles

Source: 1) ACS Cancer Statistics 2026 / KRAS variant prevalence: Lee et al., npj Precis Oncol 6(1):91 (2022)

BBOT is investing in phase 1b cohorts with the greatest potential patient benefit

	Indication	Mutation	Treatment Regimen		Status of Combination Cohorts
A	NSCLC	$KRAS^{G12C}$	BBO-8520	+ pembrolizumab	Enrolling
				Monotherapy	Enrollment Complete
B	CRC	$KRAS^{G12D/V}$	BBO-11818	+ BBO-10203	Enrolling
				+ cetuximab	Enrolling
				+ BBO-10203 + cetuximab	Planned
		$KRAS^{mut}$	BBO-10203	Monotherapy	Enrollment Complete
				+ FOLFOX / bevacizumab	Enrollment Complete
	PDAC	$KRAS^{G12D/V}$	BBO-11818	Monotherapy	Enrolling
				+ BBO-10203	Enrolling

Source: ClinicalTrials.gov NCT06343402 (ONKORAS-101, BBO-8520); NCT06917079 (KONQUER-101, BBO-11818); NCT06625775 (BREAKER-101, BBO-10203)

BBO-11818 and BBO-10203 data catalysts planned for Q4 2026

A Expanded **BBO-8520** + pembrolizumab dataset in 2L+ G12C inhibitor experienced $KRAS^{G12C}$ NSCLC — ***Mid-2027***

B Data update on **BBO-11818** and **BBO-10203** monotherapy — ***Q4 2026***

BBO-11818 and **BBO-10203** internal / SoC combination cohorts in CRC & PDAC — ***Mid-2027***

$344M cash as of end Q2 2026[1] | **Cash runway projected into 2028**

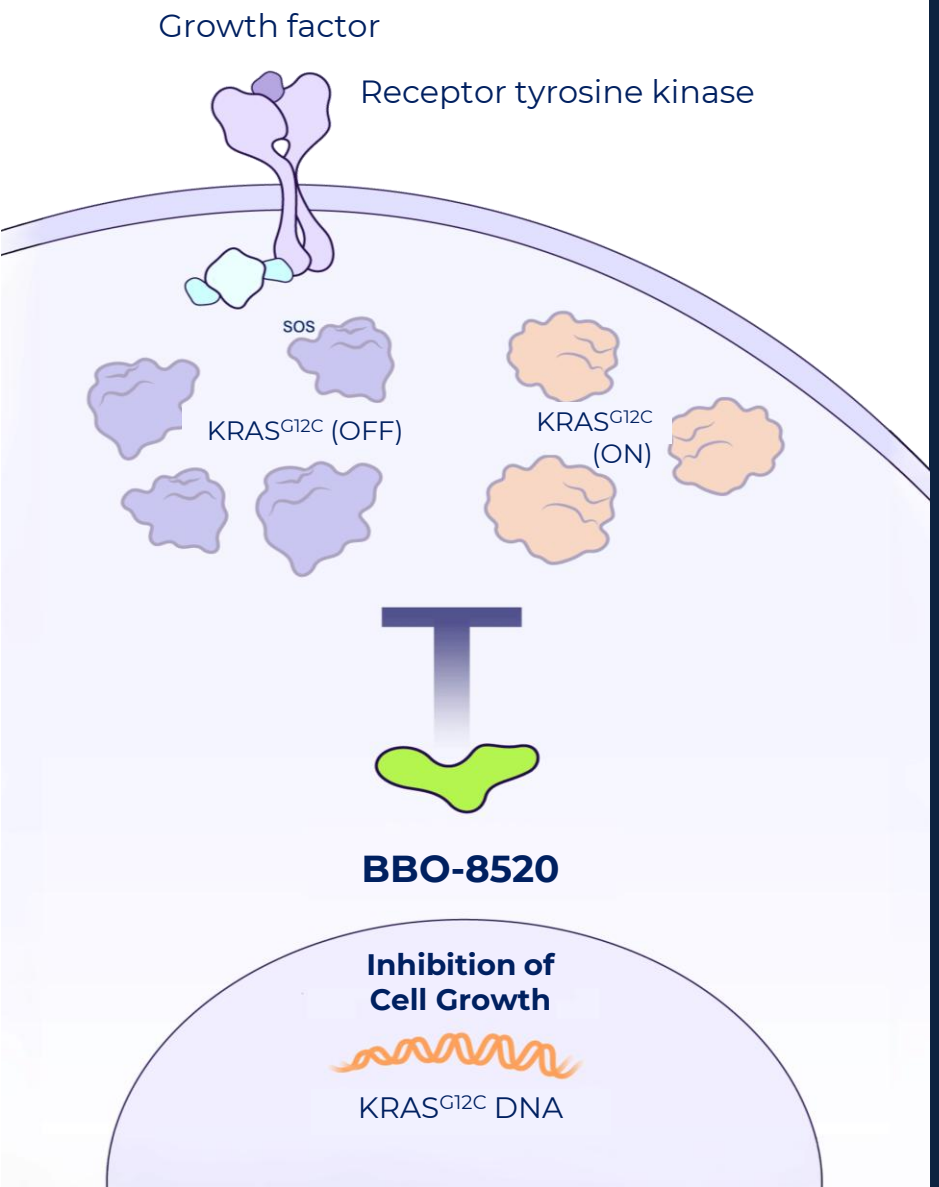
Source: 1) BBOT 10-Q August 11th, 2026

Strategic focus

A **BBO-8520** + pembrolizumab combination in 2L+ G12C inhibitor experienced $KRAS^{G12C}$ NSCLC

B **BBO-11818** and **BBO-10203** +/- standard of care in $KRAS^{mut}$ cancers

BBO-8520's direct ON/OFF inhibition enables improved therapeutic index



- Direct ON-state inhibition **drives gains in potency** and **lowers the free drug levels** needed for activity
- 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 of OFF inhibitors

Source: Awad et al., NEJM 384(25):2382–2393 (2021); Amodio et al., Cancer Discov 10(8):1129–1139 (2020)
BBO-8520: Maciag et al., Cancer Discovery. 2024

2L+ G12C inhibitor experienced KRAS^{G12C} NSCLC opportunity: Differentiated data in a growing market with high unmet need

Differentiated data

In G12C inhibitor experienced patients, BBO-8520 + pembrolizumab has shown **75% ORR at 500mg** QD and **53% ORR across dose levels**[1]

Growing market

Potential G12C OFF inhibitor approvals in 1L are expected to change SoC and create a significant G12Ci experienced 2L market

Unmet need

Docetaxel delivers 9-13% ORR and 3.8-4.5 months[2] PFS in the G12C inhibitor naive setting

Competitive whitespace

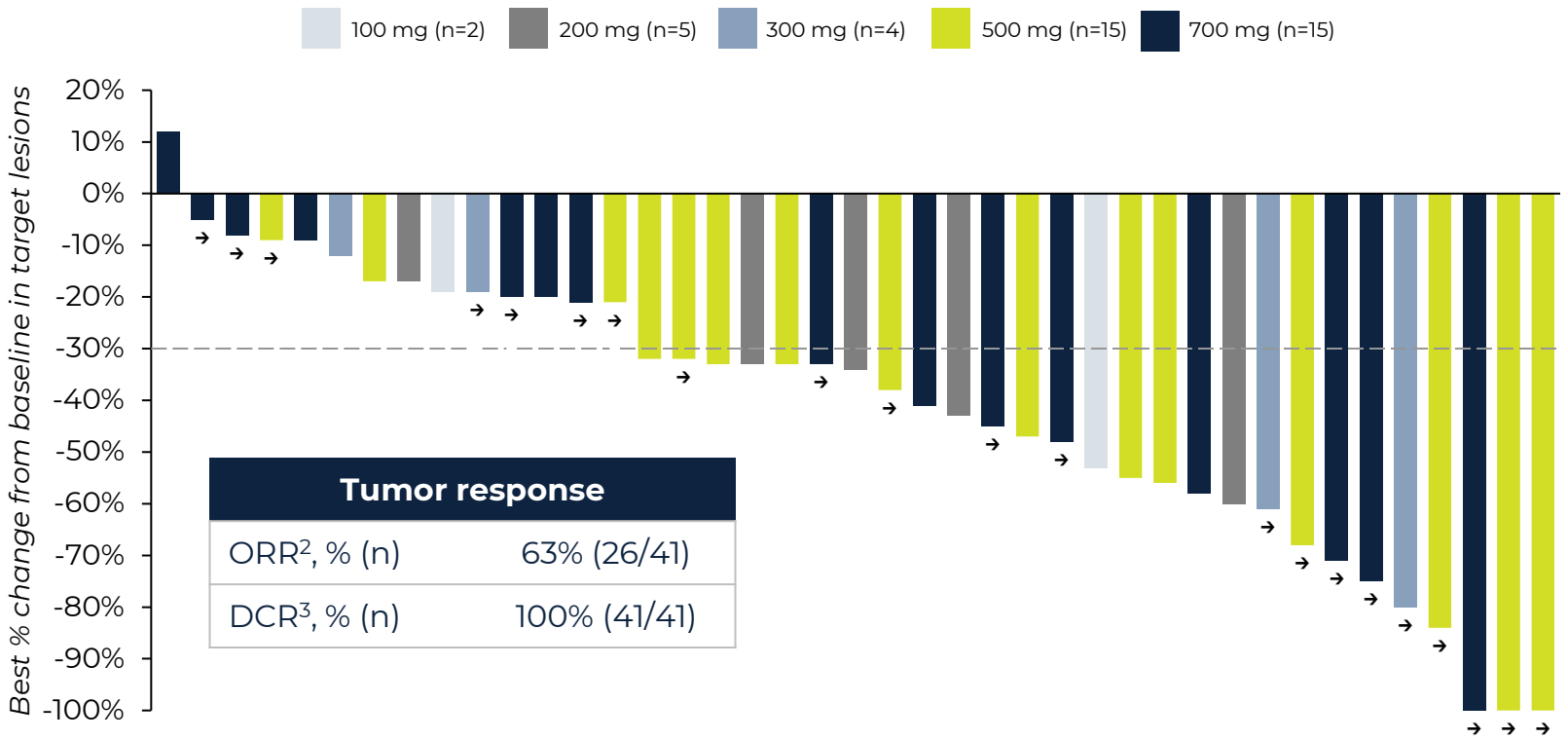
No approved targeted agents and no announced or ongoing registrational trials in this segment

Source: 1) ONKORAS-101 DCO June 1, 2026, analysis set includes patients with at least 12 weeks of follow-up and at least 1 post-baseline radiographic disease, all patients TPS≥1% and includes confirmed and unconfirmed responses; 2) CodeBreaK 200 / KRYSTAL-12 (NCT04685135)

In G12C inhibitor naïve 2L+ monotherapy patients, efficacy and safety data continue to track in line with our prior data disclosure in January 2026, with an ORR of 63%

BBO-8520 efficacy evaluable[1] G12C inhibitor naïve patients

Best overall response (N=41)



Tumor response	
ORR[2], % (n)	63% (26/41)
DCR[3], % (n)	100% (41/41)

Evidence of response durability - *21/28 (75%) of patients eligible for 6-month follow-up remained on treatment greater than 6 months*

Treatment-related AEs and AEs of Interest

Reported in >20% of monotherapy patients

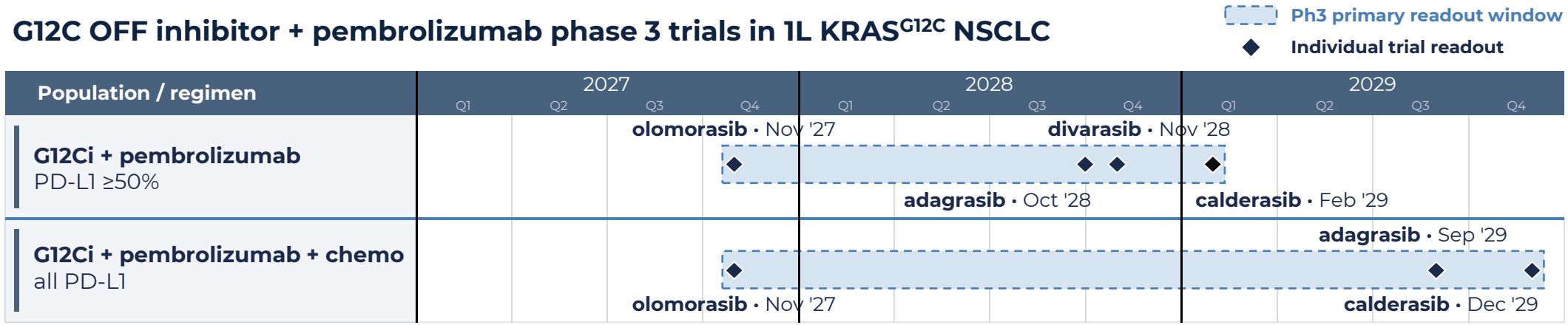
AE term	BBO-8520 Monotherapy N=44	
	All Grades	Grade ≥3
Any TRAE	**41 (93.2)**	**7 (15.9)**
DIARRHEA	33 (75.0)	5 (11.4)
NAUSEA	33 (75.0)	1 (2.3)
VOMITING	23 (52.3)	0
FATIGUE	15 (34.1)	1 (2.3)
DECREASED APPETITE	10 (22.7)	0
AE of Interest		
AST INCREASED	5 (11.4)	0
ALT INCREASED	4 (9.1)	0

Note: → indicates patient is still on study treatment 1) Analysis set includes patients with at least 12 weeks of follow-up and at least 1 post-baseline radiographic disease assessment; 2) ORR includes both confirmed and unconfirmed responses; 3) Disease control rate (DCR) includes complete responses (CR), partial response (PR) and stable disease (SD)

Source: ONKORAS-101 DCO June 1, 2026

2L KRAS^{G12C} NSCLC may see an increasing share of G12C inhibitor experienced patients with significant unmet need

G12C OFF inhibitor + pembrolizumab phase 3 trials in 1L KRAS^{G12C} NSCLC

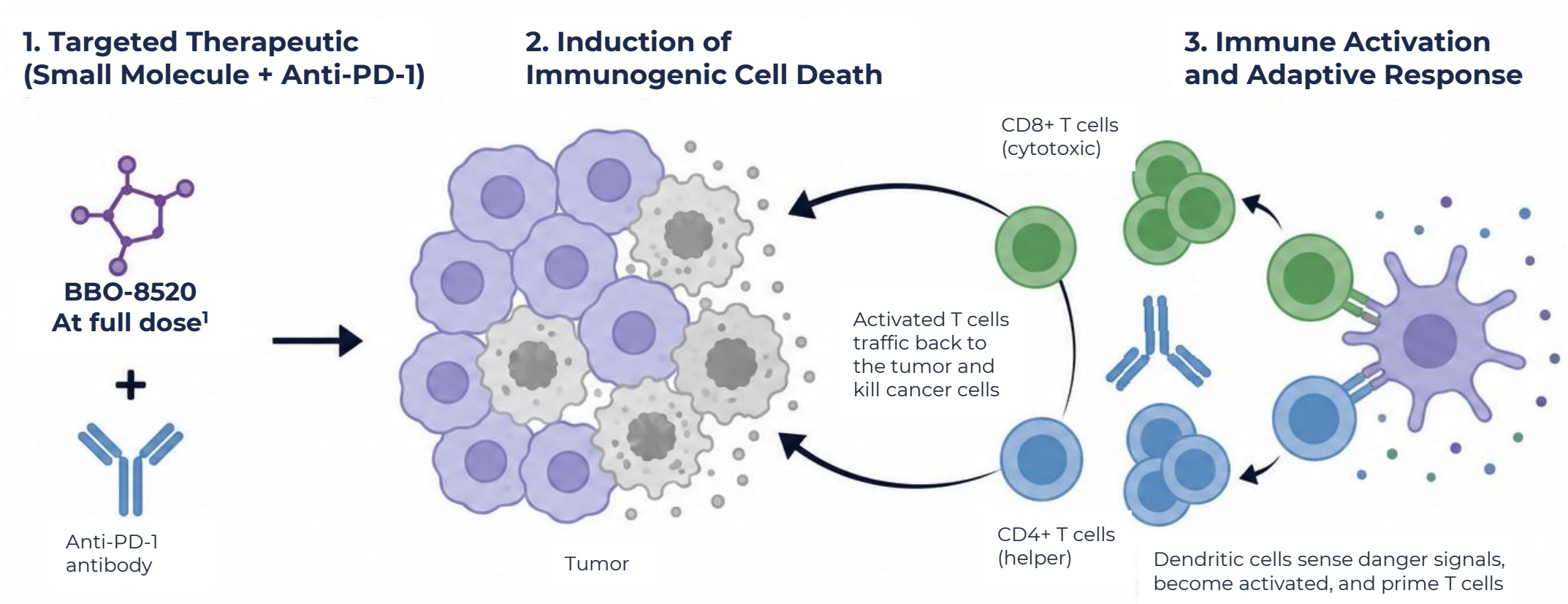


Growing, unaddressed patient population

Six 1L Ph3 G12Ci trials in 1L NSCLC are expected to complete in 2027 - 2029, potentially moving OFFi into the front line and building a growing 2L+ G12Ci experienced population with no targeted therapy available

Notes: 1) Revolution Medicines has guided to a first-line phase 3 with elironrasib but no start date has been specified and this trial would be at least three years behind competitors; 2) 1L NSCLC KRAS inhibitor + pembrolizumab combinations are dosed below mono RP2D/label to limit liver/immune Adverse Events: adagrasib 400 vs 600 mg BID (label); olomorasib tested 50–100 mg BID combination vs 200 mg BID monotherapy; calderasib tested 25–400 mg QD combination vs 25-800 mg combination
Source: ClinicalTrials.gov primary completion dates and N, as accessed August 2026; olomorasib, SUNRAY-01; adagrasib, KRYSTAL-7 (Oct '28) and KRYSTAL-4 (Sep '29); divarasib, Krascendo-2; calderasib, KANDLELIT-004 (Feb '29) and KANDLELIT-007 (Dec '29).

Following a suboptimal dose of a G12C OFF inhibitor in the 1L, BBO-8520 ON/OFF potency could enable new immunogenic cell death in combination with PD-1

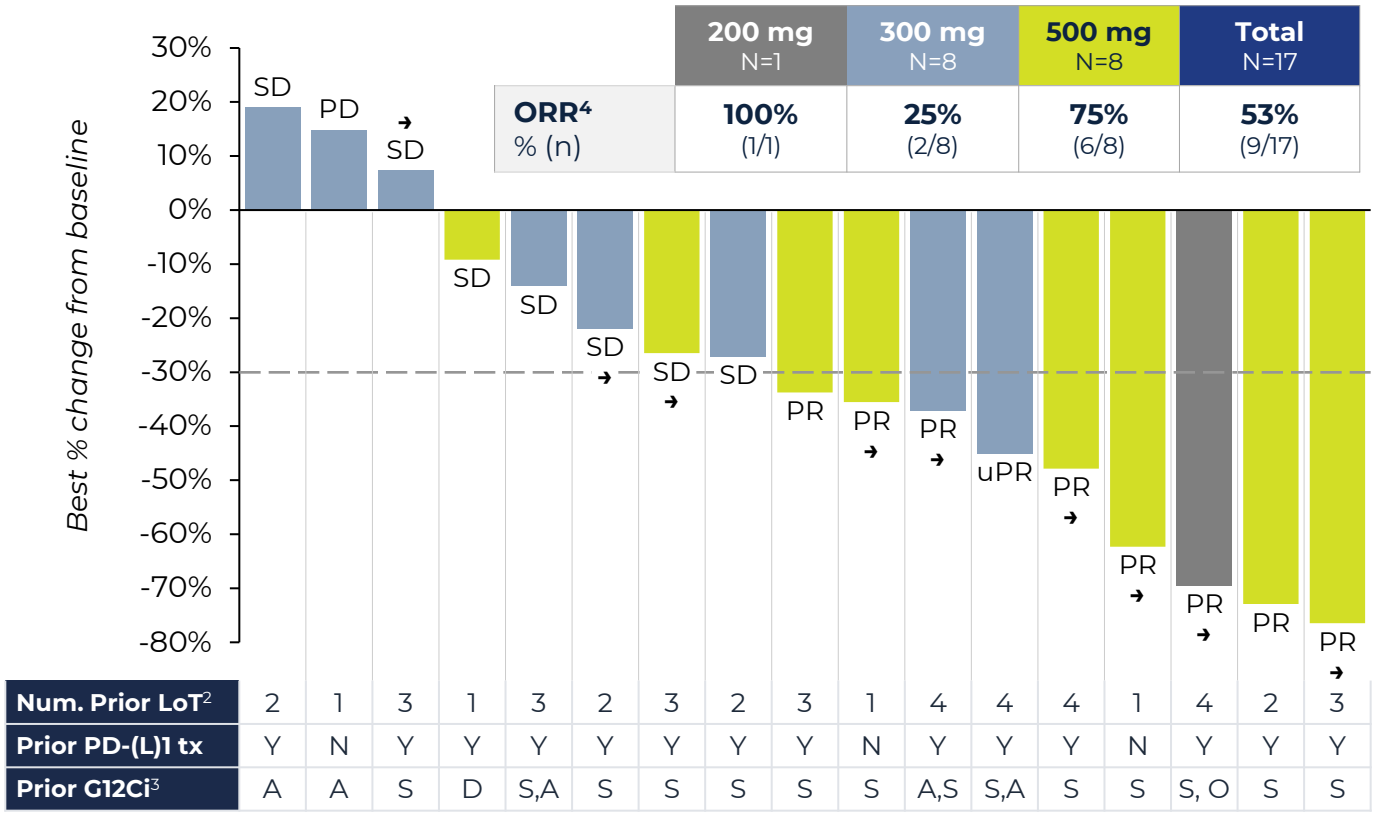


Notes: 1) Full dose refers to the dose shown to be efficacious as monotherapy. BBOT Phase 1 data indicate BBO-8520 can be combined with pembrolizumab at this dose, unlike most G12C(OFF) inhibitors.
Source: Figure adapted from Zhai et al., Frontiers in Pharmacology, 2023

In the G12C inhibitor experienced setting, BBO-8520 + pembrolizumab has shown 75% ORR at 500mg QD and 53% ORR across dose levels in 2nd to 5th line patients

BBO-8520 in efficacy evaluable G12C inhibitor experienced patients[1]

Best overall response (N=17)

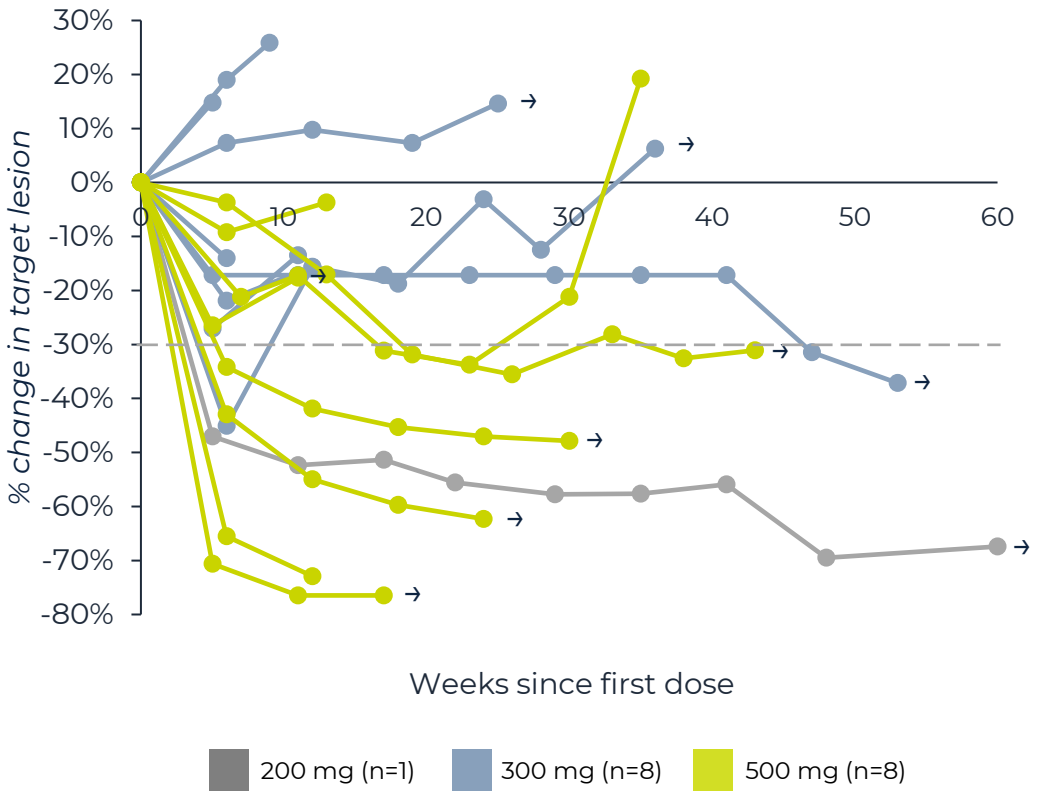


ORR for PD-(L)1 experienced patients is 50%, in line with overall ORR

Patients have 1-4 prior lines of therapy in the metastatic setting

Change in tumor burden over time[1,4]

N=17

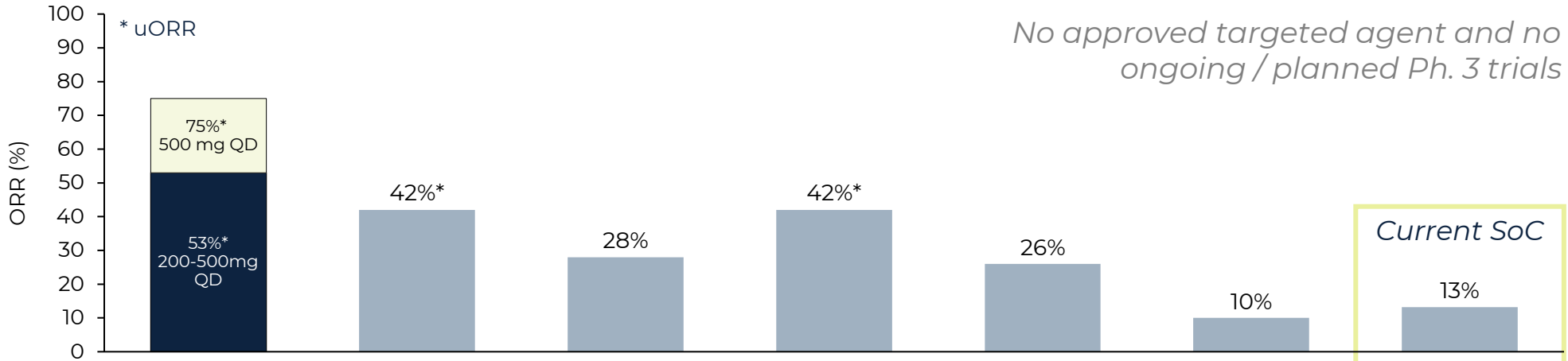


Note: → indicates patient is still on study treatment; LoT, Lines of Therapy 1) ONKORAS-101 DCO June 1, 2026, analysis set includes patients with at least 12 weeks of follow-up and at least 1 post-baseline radiographic disease, all patients TPS≥1% 2) In the advanced or metastatic setting; 3) Prior G12Ci treatment: D=Divarasib, A=Adagrasib, S=Sotorasib, O=Olomorasib; 4) Includes one unconfirmed PR at 300mg

BBO-8520 + pembrolizumab ORR reported to date is above phase 1 clinical benchmarks and standard of care in G12Ci pretreated patients

BBO-8520 + pembrolizumab vs. third-party trials of other KRAS^{G12C} inhibitors[1]

2L+ G12C inhibitor experienced ORR (%)



No approved targeted agent and no ongoing / planned Ph. 3 trials

Regimen	BBO-8520[1] + pembro	elironrasib[2]	olomorasib + pembro[3]	olomorasib[4]	elisrasib[5]	divarasib + Atezo[6]	docetaxel[7]
MoA	ON/OFF	ON	2nd gen OFF				N/A
Trial	ONKORAS-101	NCT06128551	LOXO-RAS-20001		NCT05410145	NCT04449874	CodeBreak 200
Dose	200-500 mg QD	200 mg BID	50-100 mg BID	150 mg BID	600 mg QD	400 mg QD	N/A
mPFS	N/A	6.2 mo	4.3 mo	8.2 mo	8.1 mo	N/A	3.8-4.5 mo
N *efficacy eval.*	17	24	18	38	31	10	300+

Notes: The trials shown on this slide involved different patient populations and trial designs, and no direct comparisons can be drawn, no head-to-head trials of the BBO-8520 + pembrolizumab regimen versus other therapies have been conducted; uORR= unconfirmed Objective Response Rate; 1) ONKORAS-101 DCO June 1, 2026; 2) Jänne PA, et al. AACR-NCI-EORTC 2025 (RMC-6291-001), 200 mg BID; Revolution Medicines Q3 2025 Corporate Update; elironrasib 200 mg BID + daraxonrasib (100-200 mg QD) has 62% uORR in n=26 pts but 43% Gr3+ TRAE; 3) Olomorasib + pembro: Burns TF, et al. J Thorac Oncol 2026 (LOXO-RAS-20001, n=18); 4) Olomorasib mono post-G12Ci: Koyama T, et al. Nat Commun 2026;17:3834 (LOXO-RAS-20001, n=38); 5) D3S-001 post-G12Ci: AACR 2026 (n=31); 6) 1 PR out of 10 G12Ci pretreated patients, WCLC 2024 (400 mg QD); 7) CodeBreak 200 and KRYSTAL-12; G12C inhibitor naïve setting

BBO-8520 + pembrolizumab has shown a generally differentiated safety profile in 2L+ G12C inhibitor experienced patients

TRAEs reported in >20% patients & TRAEs of interest[1]

N=17

AE term	All Grades	Grade ≥3
Any TRAE	**17 (100%)**	**7 (41.2%)**
DIARRHEA	13 (76.5%)	5 (29.4%)
NAUSEA	12 (70.6%)	1 (5.9%)
VOMITING	9 (52.9%)	0
FATIGUE	7 (41.2%)	0
AE of Interest		
AST INCREASED	2 (11.8%)	1 (5.9%)
ALT INCREASED	1 (5.9%)	1 (5.9%)

Dose modifications due to TRAEs[1,2]

N=17

Dose modification by agent	BBO-8520	pembro
Dose discontinuation, n (%)	0 (0.0%)	1 (5.9%)
Dose interruption, n (%)	9 (52.9%)	7 (41.2%)
Dose reduction, n (%)	7 (41.2%)[2]	N/A

Combination safety profile summary

- BBO-8520 + pembrolizumab safety profile appears generally tolerable and manageable
- TRAEs are mostly GI-related with a potentially differentiated liver toxicity profile as of the data cutoff date
- ALT/AST safety profile enabled PD-1 combination
  - The only G3 ALT/AST was considered by the PI to be mainly due to co-medications and LFT increases do not seem to be dose dependent
  - Other G12C inhibitors see ~10-15% Gr3+ ALT/AST in combination with pembrolizumab[3]

Notes: 1) ONKORAS-101 DCO June 1, 2026, analysis set includes patients with at least 12 weeks of follow-up, all patients TPS≥1%; 2) TRAEs leading to dose reduction were diarrhea (n=5), anemia (n=1), and vomiting (n=1); 3) G12Ci + pembrolizumab in 1L NSCLC: adagrasib 400 mg BID, KRYSTAL-7 (11% / 14%, ASCO 2025); divarasib 400 mg QD, Krascendo-170 (23% / 18%, ASCO 2026); olomorasib 50–100 mg BID, LOXO-RAS-20001 (18% / 15%, ASCO 2026); MK-1084 25–400 mg QD, KANDLELIT-001 (8% / 10%, ASCO 2025), No head-to-head studies have been conducted to evaluate the BBO-8520 + pembrolizumab regimen versus other G12C inhibitors, and no direct comparisons can be drawn

BBO-8520 + pembrolizumab has the potential to address a $1-2B US opportunity

Patient flow to our target population

KRAS^{G12C} metastatic NSCLC, US

Today's SoC

1L
pembro +/- chemo

2L
KRAS G12C OFF inhibitor
Lumakras + Krazati: ~$400M US, FY2025
Divarasib: ~$1.2 to 2.5B peak, WW[1]

3L
docetaxel

SoC 2028+

1L
G12C OFFi + pembro +/- chemo

2L+ G12Ci-experienced
BBO-8520 + PD-1

$1-2B US TAM

Notes: SoC = standard of care; TAM, total addressable market; WW, worldwide; 3L, third line; pembro, pembrolizumab; patient flow and TAM are a BBOT projection
Source: 1) Amgen and BMS FY2025 filings; Roche Q1 2026 earnings presentation

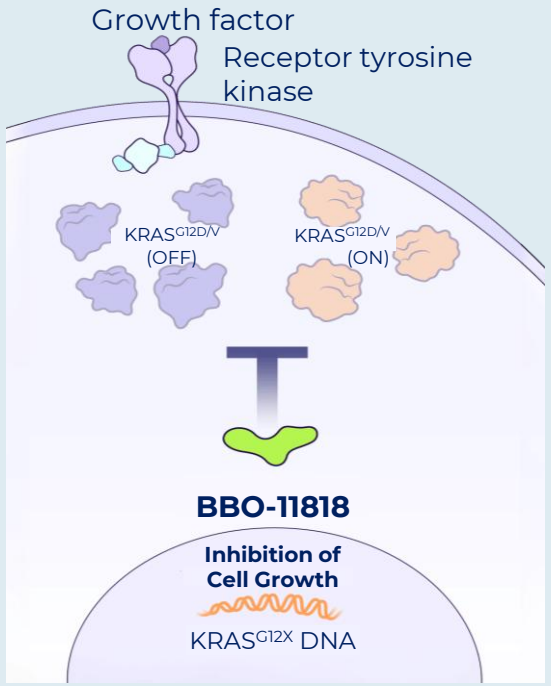
Strategic focus

A **BBO-8520** + pembrolizumab combination in 2L+ G12C inhibitor experienced $KRAS^{G12C}$ NSCLC

B **BBO-11818** and **BBO-10203** +/- standard of care in $KRAS^{mut}$ cancers

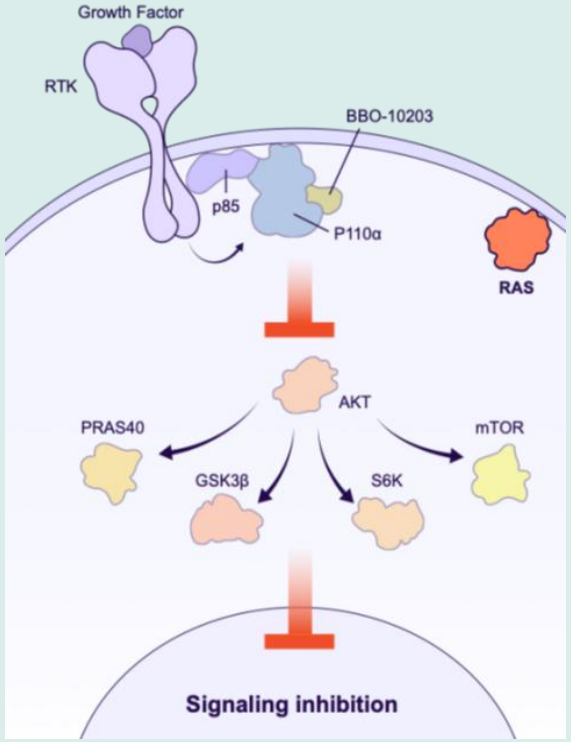
Mechanism of action for BBO-11818, Pan-KRAS (ON/OFF) inhibitor & BBO-10203, RAS:PI3Kα breaker

BBO-11818 Pan-KRAS(ON/OFF) inhibitor
Mechanism of action



- BBO-11818 is an orally bioavailable, reversible pan-KRAS inhibitor with activity in both the ON and OFF states
- Highly selective (>500x) for KRAS, spares H- and N-RAS
- Single-digit nM activity with no shift in potency between pERK and 3D viability

BBO-10203 RAS:PI3Kα breaker
Mechanism of action



- Blocks binding of K-, H-, and N-RAS to PI3Kα
- Agnostic to the mutational status of either partner
- Does not inhibit the kinase activity of PI3Kα

Source: BBO-10203: Simanshu et al. Science, 2025; BBO-11818: Stahlhut et al. Cancer Discovery, 2026;16(4):740-59

BBO-11818 and BBO-10203 were each designed to improve therapeutic index and enable unique combinations relative to competitor approaches

BBO-11818
Pan-KRAS(ON/OFF) inhibitor

- Improved tolerability and no significant skin toxicity observed to date[1], potentially due to >500x selectivity over N-, H-RAS relative to ***panRAS inhibitors***
- Broader mutant allele coverage may increase market opportunity relative to ***mutant-selective KRAS inhibitors***

BBO-10203
RAS:PI3Kα breaker

- Designed to overcome safety limitations, including high rates of hyperglycemia of ***WT PI3Kα inhibitors***
- Designed to inhibit RAS-driven PI3K***α*** signaling in >90% KRASmut tumors that are PI3K***α*** WT, which are not addressable for ***mutant selective PI3Kα inhibitors***

Notes: 1) <10% of patients experienced grade 1 rash TRAE, no higher-grade skin toxicity observed as of September 1st, 2026

Each program has been independently clinically evaluated through monotherapy dose escalation

BBO-8520 | BBO-11818 | BBO-10203

	BBO-11818 Pan-KRAS(ON/OFF) inhibitor	BBO-10203 RAS:PI3Kα breaker
Dose selection	☑ Escalation through top dose of 800mg BID complete	☑ 500mg QD selected as RDE
Safety profile	☑ 2/13 (15%) Gr 3 TRAEs; No significant skin toxicity observed	☑ No hyperglycemia; overall grade 3 TRAEs <5%

KONQUER-101 for **BBO-11818** is currently enrolling monotherapy backfill and combination dose escalation

- Escalation through 800mg BID fasted with no DLTs
- No significant skin toxicity observed at any dose level[1]
- Safety profile is manageable and tolerable

BREAKER-101 for **BBO-10203** has completed monotherapy dose escalation and is currently enrolling combinations

- 500mg QD selected as RDE
- Full steady state target engagement achieved

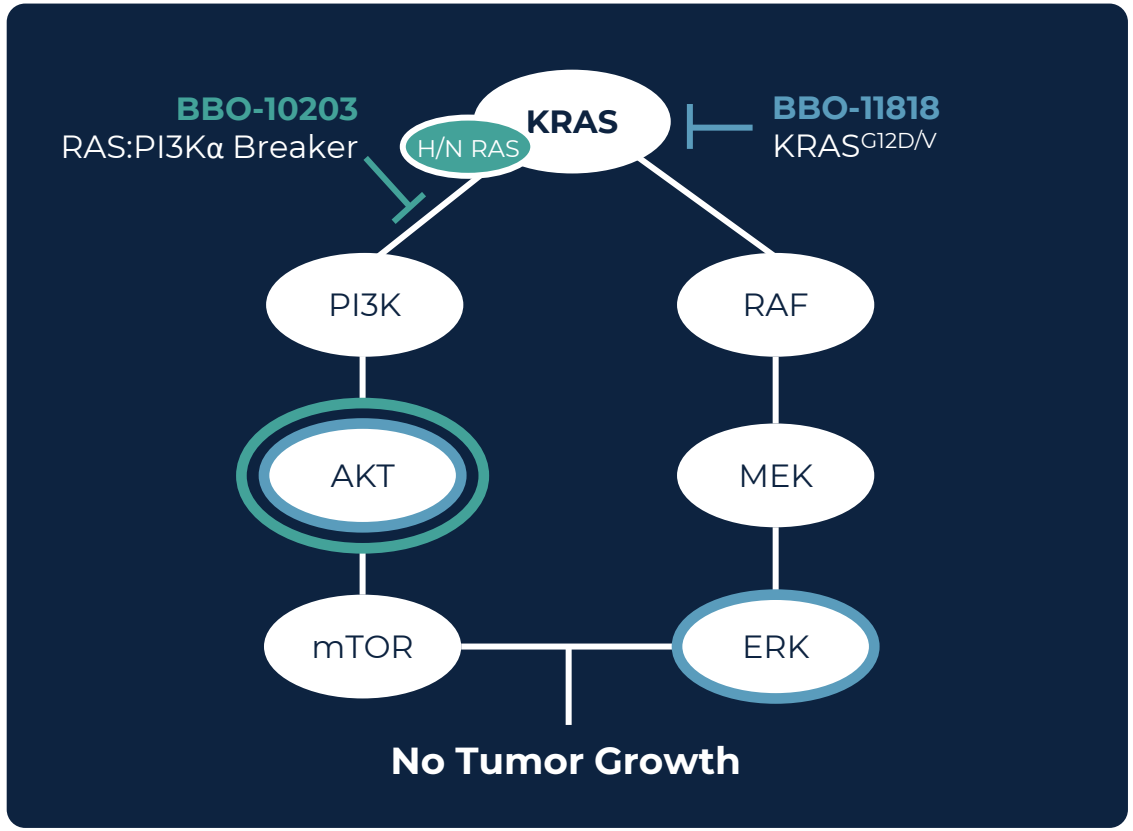
KONQUER-101 & BREAKER-101

Monotherapy & combination – active cohorts

PDAC	$KRAS^{G12D/V}$	BBO-11818
	$KRAS^{G12D/V}$	BBO-11818 + BB0-10203
CRC	$KRAS^{G12D/V}$	BBO-11818 + cetuximab
	$KRAS^{G12D/V}$	BBO-11818 + BBO-10203
	$KRAS^{mut}$	BBO-10203 + FOLFOX + bevacizumab

Notes: 1) <10% of patients experienced grade 1 rash TRAE, no higher-grade skin toxicity observed as of September 1st, 2026
Source: NCT06917079; NCT06625775

BBO-11818 and BBO-10203 were designed for internal combination to enable superior activity in KRAS$^{G12D/V}$ tumors



- Mutant KRAS strongly drives both the MAPK and AKT pathways
- In response to MAPK pathway inhibition, WT RAS activates the AKT pathway for survival
- BBO-10203's pan-RAS activity enables inhibition of RAS-driven AKT pathway activation by any RAS
- Simultaneous inhibition of both the MAPK and AKT pathways has the potential to stop proliferation and induce apoptosis leading to strong tumor regressions

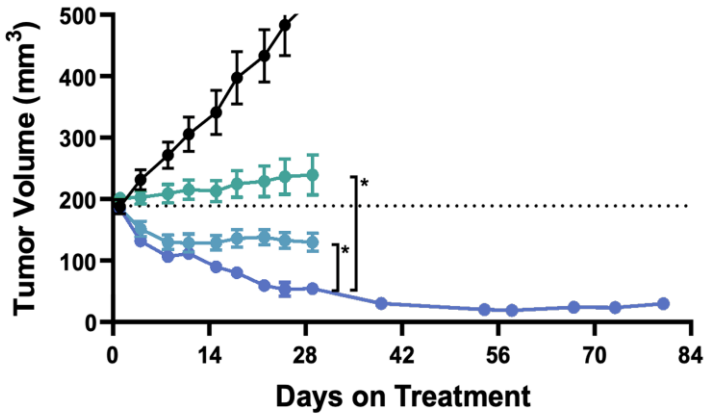
BBOT has initiated enrollment of BBO-10203 combinations with BBO-11818 in CRC and PDAC in order to test this hypothesis in the clinic

Source: Left hand side visual demonstration adapted from Moore et al. 2020

BBO-11818 in combination with BBO-10203 drives deeper tumor regression than single-agents in preclinical models

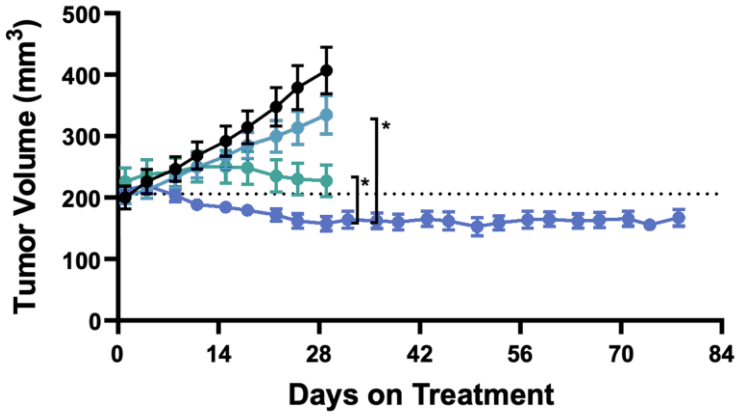
BBO-8520 | BBO-11818 | BBO-10203

C1157 PDX
CRC - KRAS^{G12D}



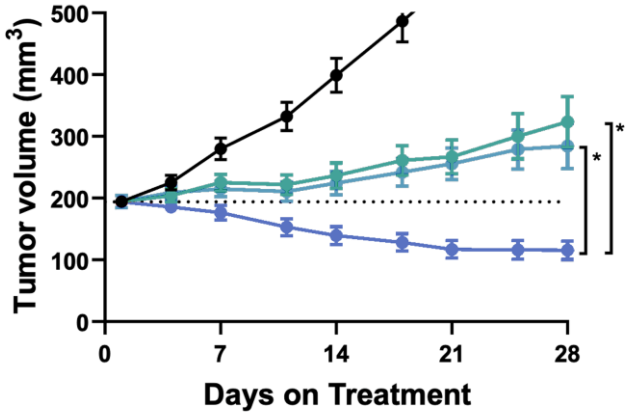
- Vehicle (BID)
- **BBO-10203** (100 mg/kg, QD)
- **BBO-11818** (100 mg/kg, BID)
- **BBO-11818** + **BBO-10203**

C1329 PDX
CRC - KRAS^{G12V}



- Vehicle (BID)
- **BBO-10203** (100 mg/kg, QD)
- **BBO-11818** (100 mg/kg, BID)
- **BBO-11818** + **BBO-10203**

CAPAN-2 CDX
PDAC - KRAS^{G12V}

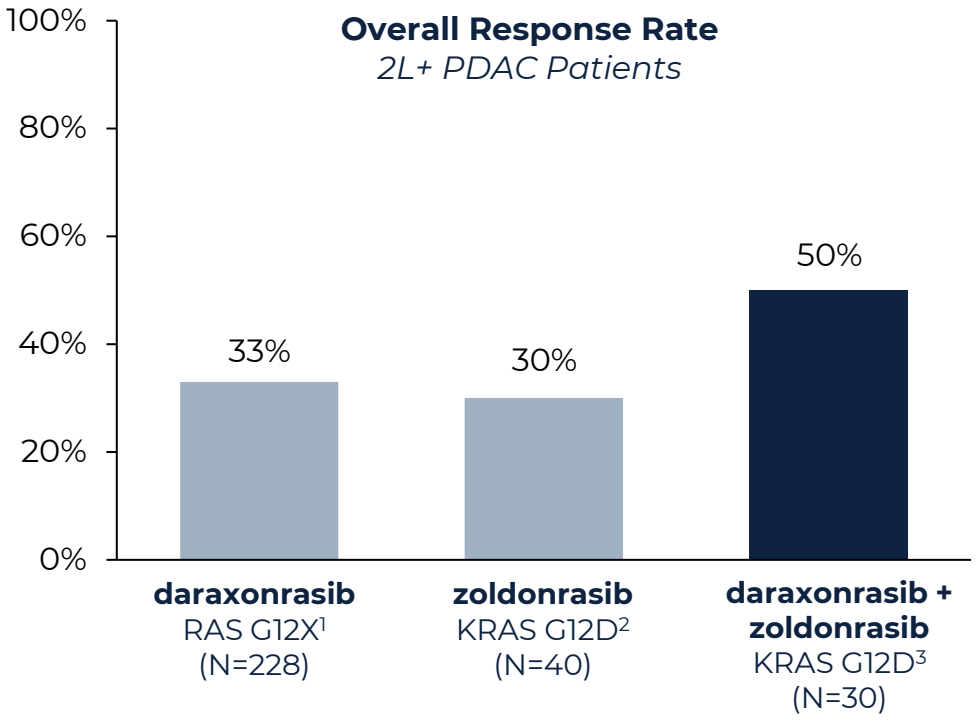


- Vehicle (BID)
- **BBO-10203** (100 mg/kg, QD)
- **BBO-11818** (100 mg/kg, BID)
- **BBO-11818** + **BBO-10203**

Notes: Kopetz lab collaboration; the C1157 and C1329 PDX models were derived from tumors after patient progression on bevacizumab, 5-FU, and oxaliplatin, and on bevacizumab, 5-FU, irinotecan, and oxaliplatin, respectively; Statistical analyses: two-way repeated measures ANOVA from day 4 to 29 *p<0.05 ; PDX, patient-derived xenograft; CDX, cell line-derived xenograft

Competitor approach: Adding selective KRAS^{G12D} inhibition to pan-RAS results in improved efficacy due to deeper target coverage, but with high toxicity

G12Di improves pan-RAS efficacy via target coverage…



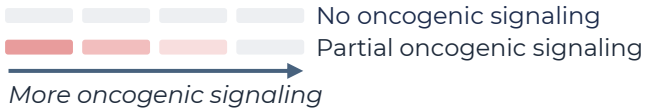
… but the combination still sees high rates of toxicity

Combo Safety Data (N=60)[3]	**All Grades**	**Grade ≥3**
Any TRAE	**97%**	**35%**
Rash	90%	12%
Diarrhea	63%	5%
Nausea	57%	2%
Stomatitis/Mucositis	53%	7%
Anemia	20%	10%
	Daraxonrasib	**Zoldonrasib**
Dose Interruption	57%	40%
Dose Reduction	30%	8%

Source: 1) RASolute 302 study, NEJM 2026; 300mg QD dose level, n=228 RAS G12 patients treated with Daraxonrasib; 2) RVMD Zoldonrasib ENA Presentation 2024, 1200mg QD or 600mg BID dose levels; 3) RVMD ESMO GI Presentation 2026; 300mg QD Daraxonrasib and 1200 mg QD Zoldonrasib dose level

BBOT approach: Enabling robust, concurrent, inhibition of RAS-driven PI3Ka and MAPK signaling using an orthogonal approach to competitors

BBO-8520 | BBO-11818 | BBO-10203



Competitor approach

Monotherapy

Pan-RAS

Rash and GI toxicity limit target coverage

MAPK — Partial inhibition

PI3Kα — Partial inhibition

Combination

Pan-RAS + G12Di

G12Di adds mutant-driven MAPK inhibition

MAPK — **Full inhibition**

PI3Kα — Partial inhibition

BBOT approach

Monotherapy

Pan-KRAS

Better TI provides MAPK coverage, WT RAS reactivates PI3Kα

MAPK — **Full inhibition**

PI3Kα — Partial inhibition

Combination

Pan-KRAS + Breaker

Breaker safety may enable deeper target coverage of both pathways

MAPK — **Full inhibition**

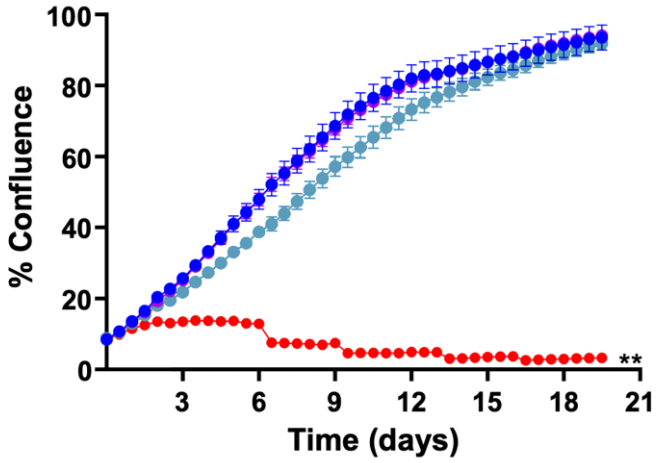
PI3Kα — **Full inhibition**

Notes: No head-to-head trials have not been conducted between BBOT's product candidates and panRAS therapies; Schematic representation of BBOT's mechanistic hypothesis; depth of pathway inhibition is illustrative and not to scale. Pan-RAS tolerability reflects rash and GI adverse events reported for pan-RAS(ON) inhibitors in clinical development; BBO-11818 and BBO-10203 profiles reflect Phase 1 data to date
Source: Choi et al., Cell Reports, 2026; Ge et al., Cancer Research, 2026.

BBOT's agents could enable combination with cetuximab without overlapping skin toxicity, which drives deep regressions in preclinical models

BBO-8520 | BBO-11818 | BBO-10203

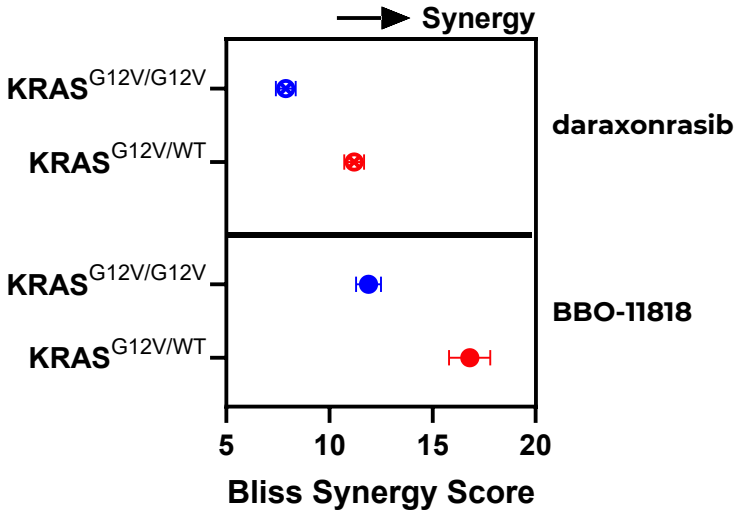
In vitro assay
CRC - KRAS^{G12V}



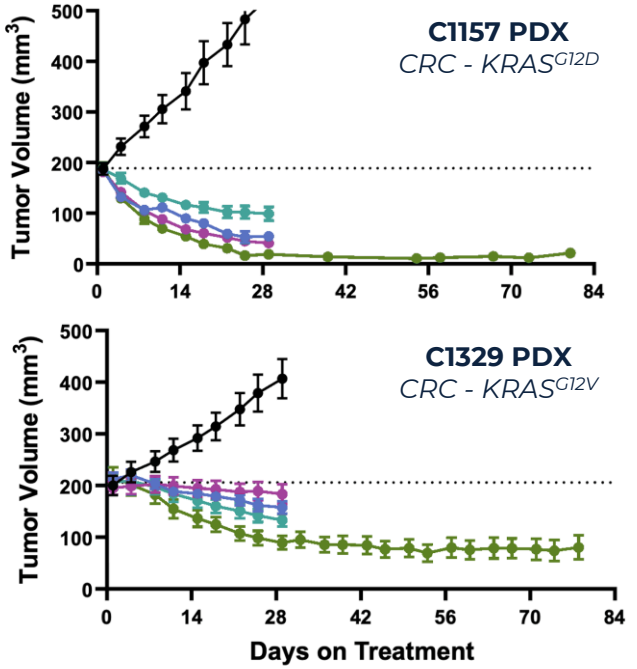
**p<0.0001 vs monotherapy groups

- Vehicle
- 1 µg/mL Cetuximab
- 4 nM BBO-11818
- 4 nM BBO-11818 + 1 µg/mL Cetuximab

Synergy summary
BBO-11818 vs. daraxonrasib



In vivo efficacy data
CRC - KRAS$^{G12D/V}$



- Vehicle (BID)
- BBO-10203 + Cetuximab[1,3]
- BBO-11818 + Cetuximab[2,3]
- BBO-11818 + BBO-10203[1,2]
- BBO-11818 + BBO-10203 +Cetuximab[1,2,3]

Notes: 1) BBO-10203 (100 mg/kg, QD); 2) BBO-11818 (100 mg/kg, BID); 3) Cetuximab (15 mg/kg, BIW)
Source: Internal BBOT data. Long-term cellular confluence assay: SK-CO-1 cells (KRASG12V/WT CRC) were treated as indicated and cellular proliferation was measured daily over 20 days. Treatments were refreshed twice weekly. Synergy assay: the cellular synergy of BBO-11818 or RMC-6236 with cetuximab was determined using a 5-day cellular proliferation assay in SK-CO-1 cells (KRASG12V/WT) or engineered SK-CO-1 cells (KRASG12V/G12V) Synergy was determined using the Bliss method

BBO-11818 and BBO-10203 combinations have the potential to address significant unmet medical need for patients with $KRAS^{mut}$ cancers

We are uniquely positioned to robustly inhibit MAPK and PI3Kα signaling in $KRAS^{mut}$ tumors in an orthogonal approach to pan-RAS + G12Di

Potential to address CRC in combination with PI3Kα and EGFR inhibition based on single-agent toxicity profiles

Clinical evaluation of each molecule independently & combination cohort enrollment initiated

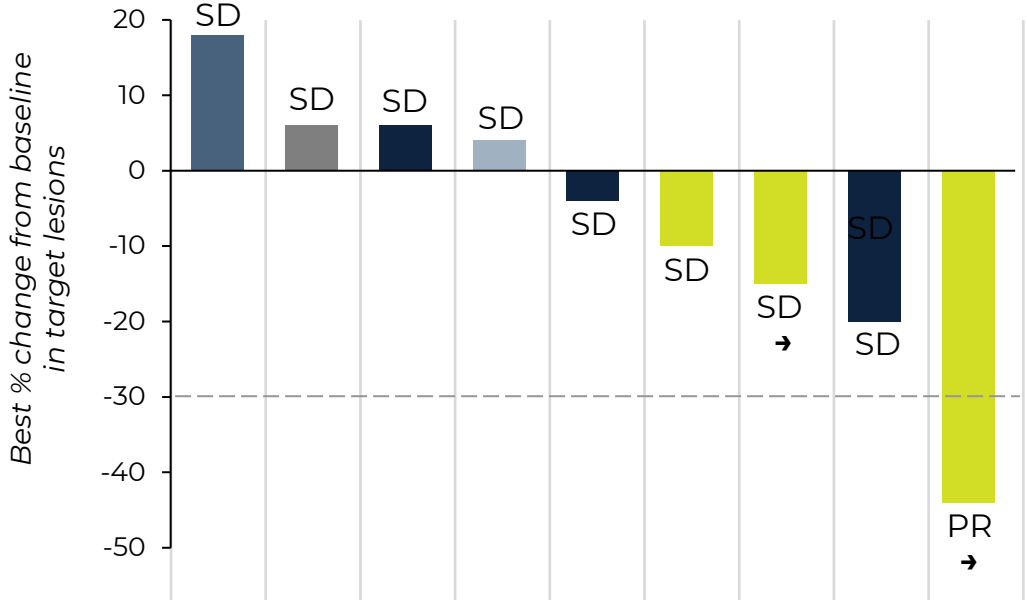
APPENDIX

BBO-11818 & BBO-10203 previously disclosed clinical data

Initial cohorts demonstrate anti-tumor activity at predicted efficacious dose levels across tumor types and appears generally tolerable and manageable

Best overall response

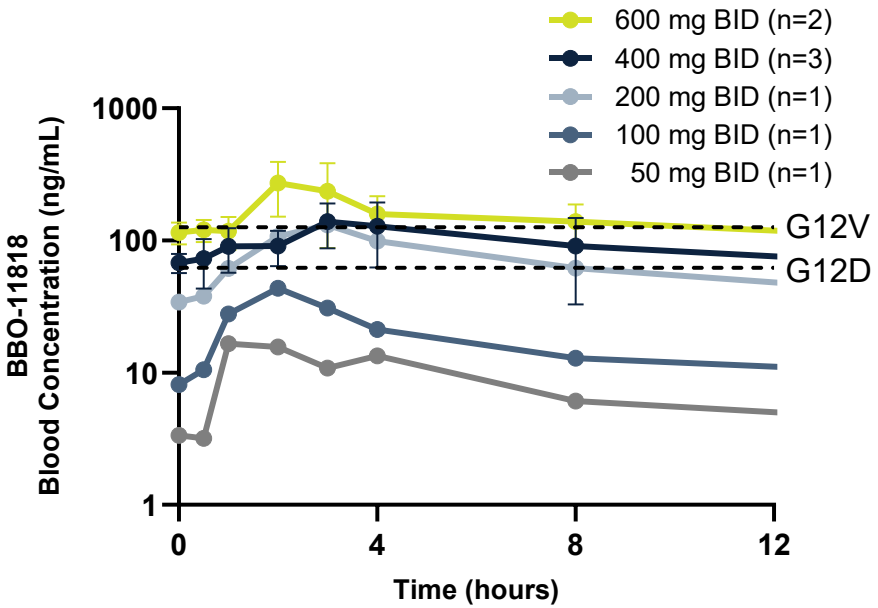
N=9



Tumor type	PDAC	PDAC	PDAC	CRC	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

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

C1D15 (Steady State) in patients



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

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; 3) 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
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	-

Source: KONQUER-101 DCO Dec 10, 2025

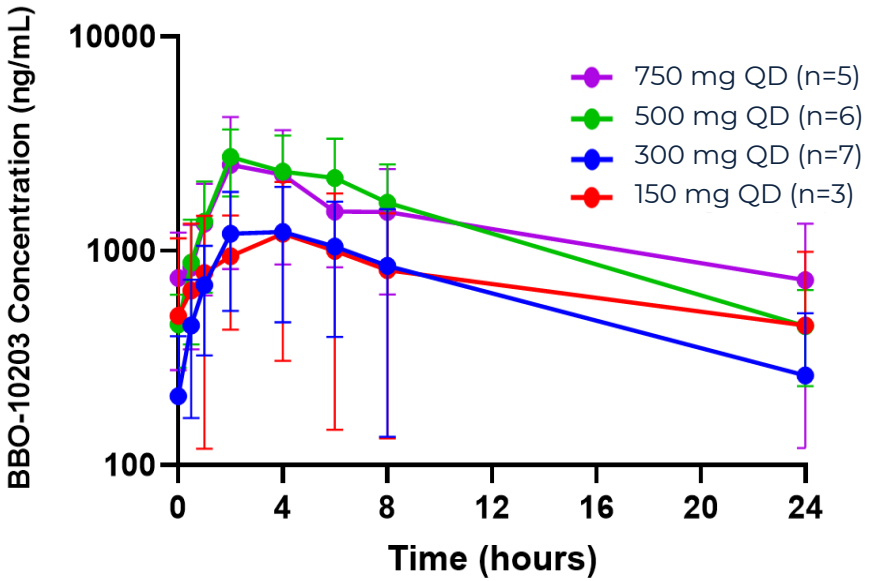
Monotherapy safety data

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

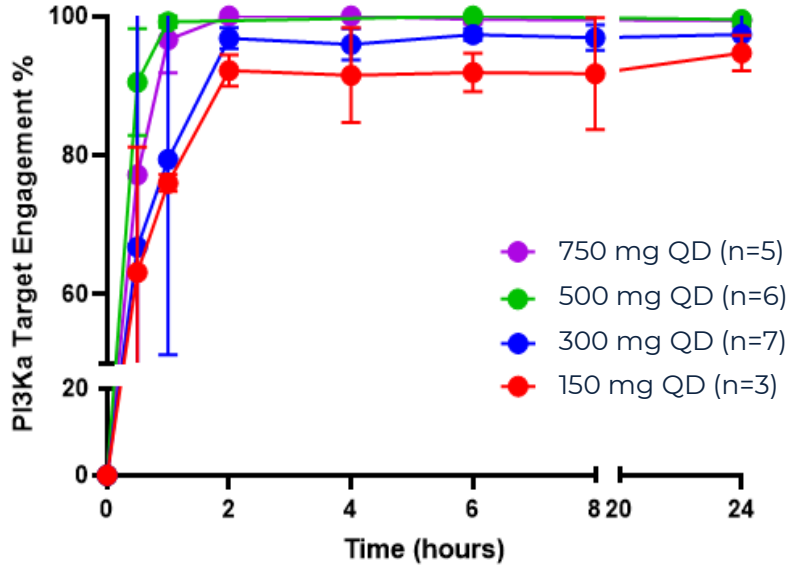
- No DLTs
- TRAEs mainly GI related
- 2 Gr 3 GI events (diarrhea and nausea) in patients with pre-existing GI conditions

BBO-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

Source: BBOT internal data

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

TRAEs by Grade in >10% patients

N=32

	Monotherapy N=24		Trastuzumab Combination N=4		FOLFOX/Bev Combination N=4	
AE term	**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 PI3Kα-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