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BridgeBio Oncology

Fourth quarter 2022

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BridgeBio Executive Summary

- BridgeBio Oncology is advancing 3 lead programs targeting the two most prominent oncogenes in human cancer (KRAS & PIK3CA): 1) Dual KRAS^{G12C}, 2) PI3Kα:RAS breaker, and 3) pan-KRAS.

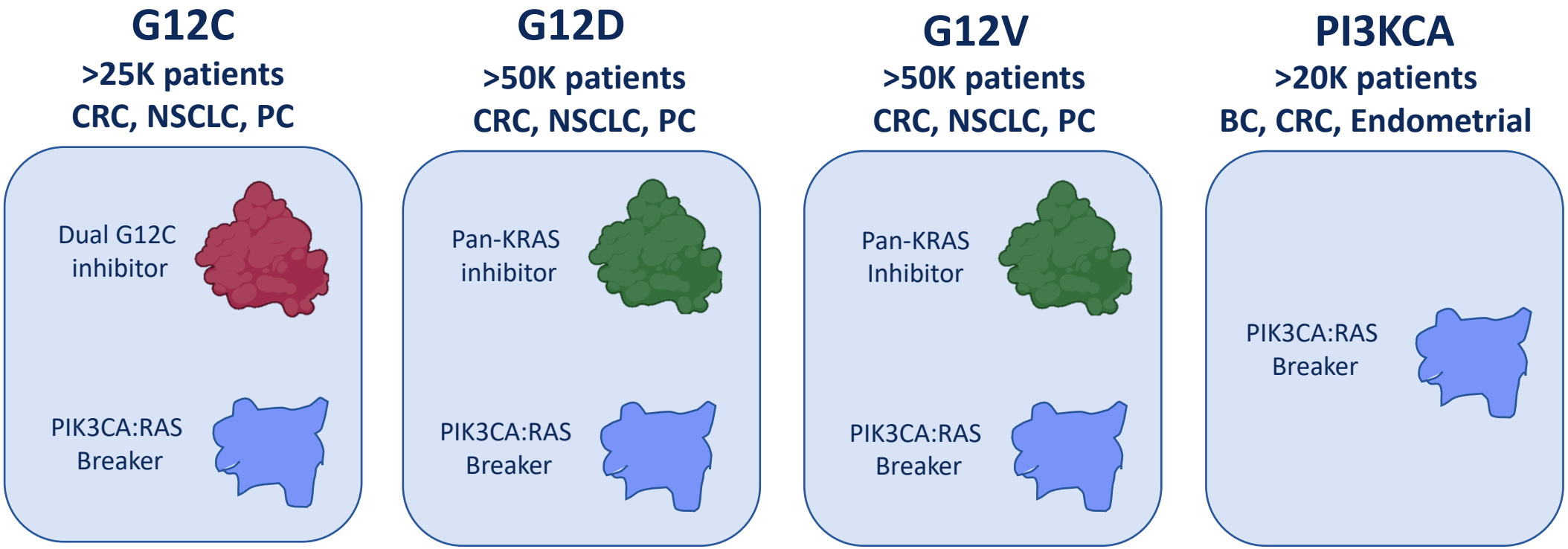
- **Dual KRAS^{G12C}** inhibitor program has selected a Development Candidate (BBO-8520).
- BBO-8520 is a highly potent dual inhibitor of KRAS^{G12C} with 30-300x higher potency than sotorasib *in vitro* and *in vivo* including differentiated activity in a PDX model.
- Complete inhibition of the active and most prominent KRAS^{G12C} form should lead to deeper and longer lasting responses in patients.
- BBO-8520's IND-filling is projected for 2023.

- **PI3Kα:RAS breaker** is highly differentiated in that it is a) tumor selective, b) targets both mutant and wild-type PIK3CA, and c) does not induce hyperglycemia
- Our compounds demonstrate robust efficacy in multiple mouse xenograft models including those with HER2/HER3 dependency, KRAS mutation and PIK3CA helical mutation
- Our latest molecules produce dose-responsive pharmacodynamic data and display promising drug-like properties.
- Biomarker data shows that Her2/Her3 dependency, PIK3CA helical mutations and KRAS^{G12X} mutations may be sensitive tumor types.
- Development Candidate selection in 2023, and IND-filling in 2024

- **pan-KRAS** program targets KRAS^{G12D} and KRAS^{G12V} mutations present in a large percentage of colorectal, pancreatic and NSCLC tumors.
- The Team has achieved *in vivo* target engagement and has identified leads with promising oral bioavailability.
- The Team projects Development Candidate selection in the 2023.

BBIO pipeline attacks Ras mutant tumors from multiple angles

BBIO oncology programs address the 2 most mutated oncogenes in human cancer and potentially enable tolerable and simultaneous inhibition of the MAPK and PI3K signaling pathways



Novel First-In-Class PI3Kα breaker has the potential to address PI3KCA helical mutations, half of $KRAS^{G12X}$ mutations and nearly all HER family driven cancers

We have a world class oncology team and strategic partners driving forward our targeted pipeline

Frank McCormick
Chairman of Oncology
ONYX PHARMACEUTICALS · Cetus

Richard Scheller
Chairman of R&D
Genentech · 23andMe

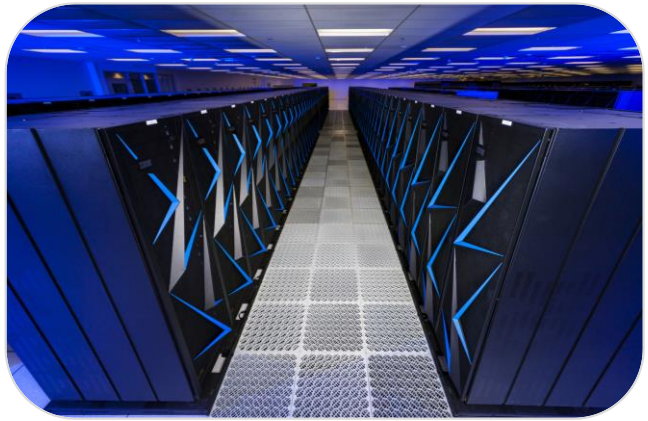
Eli Wallace
CSO, Oncology
ARRAY BIOPHARMA · Peloton Therapeutics

Pedro Beltran
SVP, Oncology
AMGEN · UNITY BIOTECHNOLOGY

NIH NATIONAL CANCER INSTITUTE

- Partnership with the National RAS Initiative, including **60 of the world's foremost academic RAS researchers**
- Cutting edge RAS **structural biology expertise**

Lawrence Livermore National Laboratory



- Home to Sierra: the **world's 3rd fastest computing system**
- Enables **multi-microsecond molecular dynamics simulations** of protein complexes, and highly efficient in silico docking simulations

BridgeBio is progressing multiple approaches against KRAS

- **Dual KRAS^{G12C} inhibitor** program has selected a Development Candidate (BBO-8520) with projected IND-filing 2023
  - Inhibits both KRASG12C GTP (active) and GDP (inactive) states; directly binds KRAS
  - Differentiates from KRASG12C GDP (inactive)-only inhibitors

- **PI3Kα:RAS breaker** is highly differentiated in that it is a) tumor selective, b) targets both mutant and wild-type PIK3CA, and c) does not induce hyperglycemia
- Projected Development Candidate selection in 2023
  - Blocks specific interaction between RAS and PI3Ka
  - RAS driver agnostic
  - Blocks PI3Ka / AKT effector signaling

- **pan-KRA**S program targets multiple KRAS mutants including KRAS^{G12D} and KRAS^{G12V}
- Projected Development Candidate selection in the 2023
  - Potent pan-KRAS inhibitor
  - Directly binds KRAS

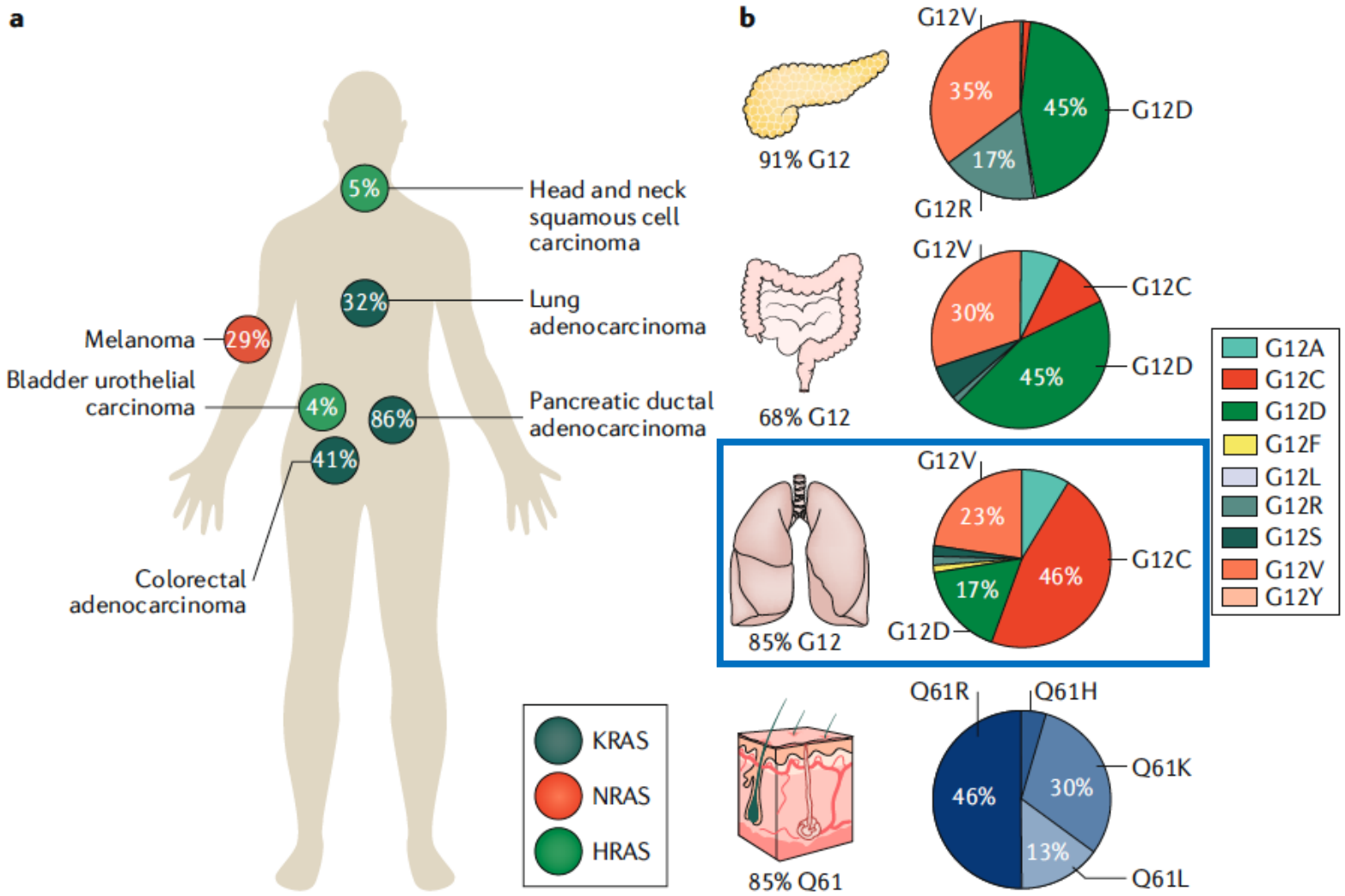
Agenda

BBO-8520: A KRAS^{G12C} Dual Inhibitor

PI3Kα:RAS Breaker

Pan-KRAS program

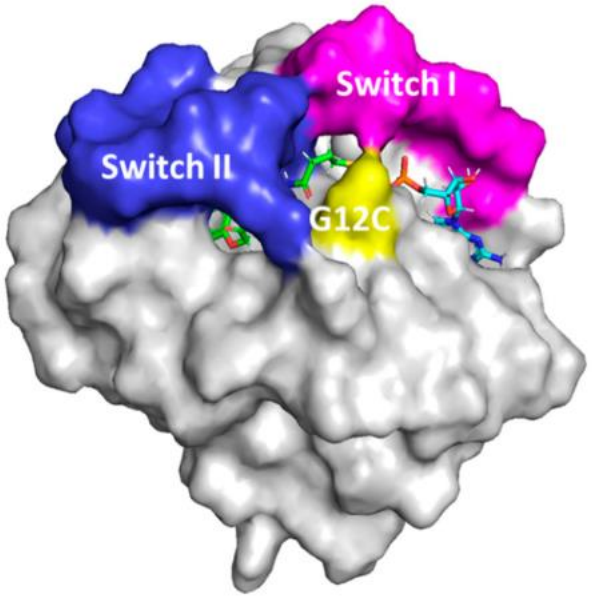
Mutant KRAS is the most common oncogene in cancer – $KRAS^{G12C}$

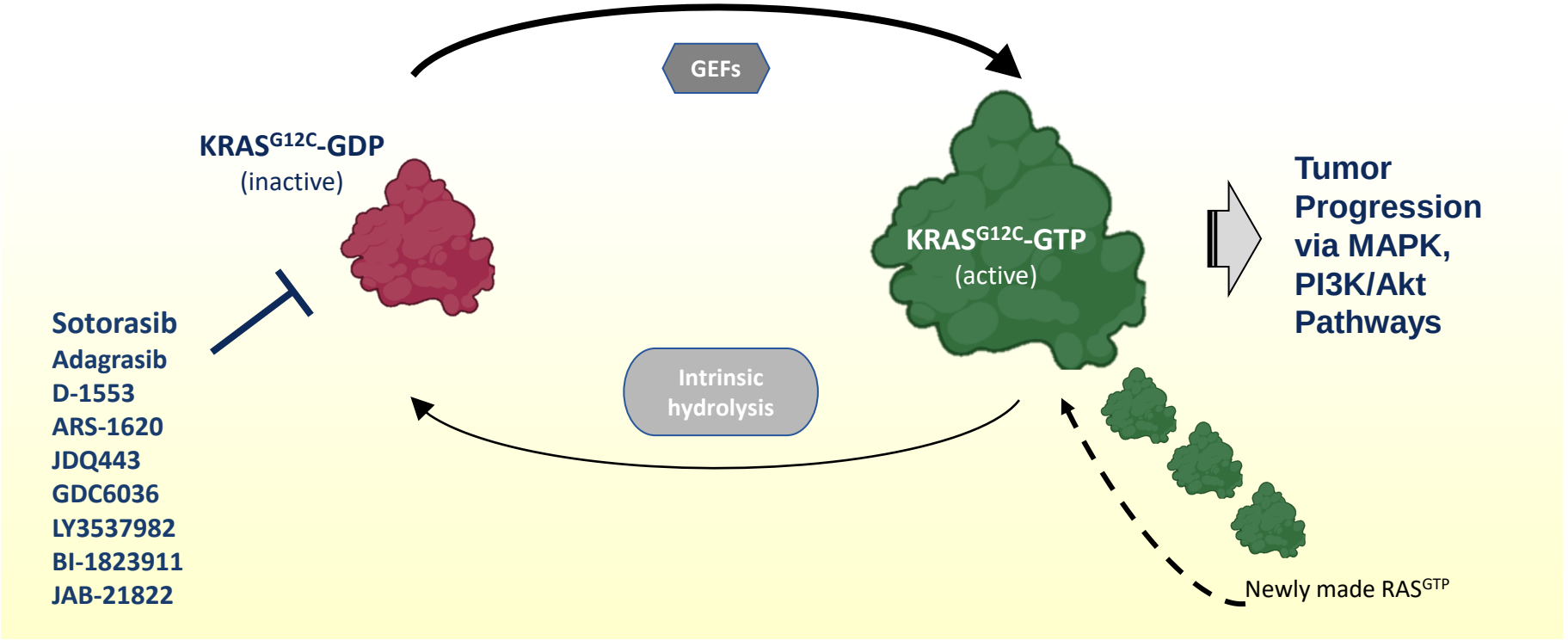


- Lung cancer is the second most common cancer in the US with greater than 235K new cases and 130K deaths a year
- ***$KRAS^{G12C}$ mutant found in ~15% of all NSCLC (~35K pts/yr)***
- Other common human cancers with KRAS mutations are colorectal and pancreatic adenocarcinomas with a combined 168K new cases a year in the US
- $KRAS^{G12D/G12V}$ mutant found in 70% of pancreatic cancers (~43K pts/yr) and 25% of colorectal cancers (~53K pts/yr)

Figure from: Moore, Rosenberg, McCormick, Malek Nat Rev Drug Disc 2020

KRAS^{G12C}-GDP inhibitors do not directly inhibit the active form of KRAS^{G12C} allowing for the emergence of resistance





Shokat's discovery led to an explosion of KRAS^{G12C}-GDP inhibitors; led by sotorasib, these will change the treatment paradigm for people with KRAS^{G12C}-driven cancers

Efficacy of KRAS^{G12C}-GDP inhibitors in the clinic is clearly suboptimal when compared to other driver-targeted therapies in the pathway

	KRAS^{G12C}-GDP inhibitors			RTK targeted agents			
	Sotorasib	Adagrasib	GDC-6036	Selpercatinib	Alectinib	Osimertinib	Capmatinib
	2L+ KRAS G12C NSCLC			2L+ RET Fusion+ NSCLC	1L ALK+ NSCLC	1L EGFR mutant NSCLC	1L cMET exon14 NSCLC
ORR	41%	43%	46%	64%	79%	77%	68%
mPFS (mo.)	6.3	6.5	*tbd*	*tbd*	25.7	18.9	12.4

Phase 3 CODEBREAK 200 – PFS 5.6 months; ORR 28%

ORR, objective response rate; PFS, progression-free survival

Sources: Sotorasib data from registrational Ph2 CODEBREAK 100 & Ph3 CODEBREAK 200 results presented at 2022 EMSO meeting; Adagrasib data from KRYSTAL-1 results presented at 2022 ASCO Meeting; GDC-6036 data from 2022 WCLC meeting; Analog data taken from product labels

Alterations associated with clinical resistance to KRAS^{G12C}-GDP inhibitors

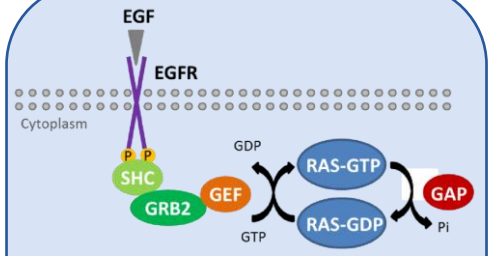
BBO-8520

KRAS^{G12C} amplification

New KRAS^{G12C} is GTP-bound

Fast adaptation

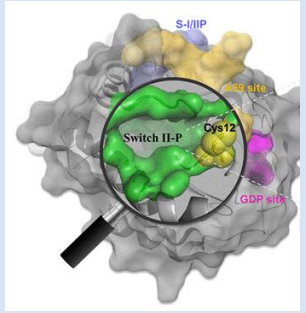
Independent RTK activation



CRC Tx	ORR
G12Ci	22%
G12Ci + EGFRi	45%

Fast adaptation

2^{ry} *in cis* KRAS mutations

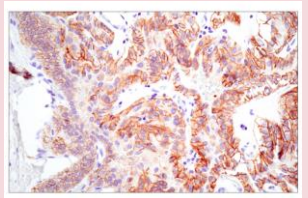


2ry mut	BBIO IC_{50}
Y96D	3 nM
R68	5 nM

Emergent resistance

2^{ry} KRAS mutation & lineage plasticity

G12C ➡ G12D



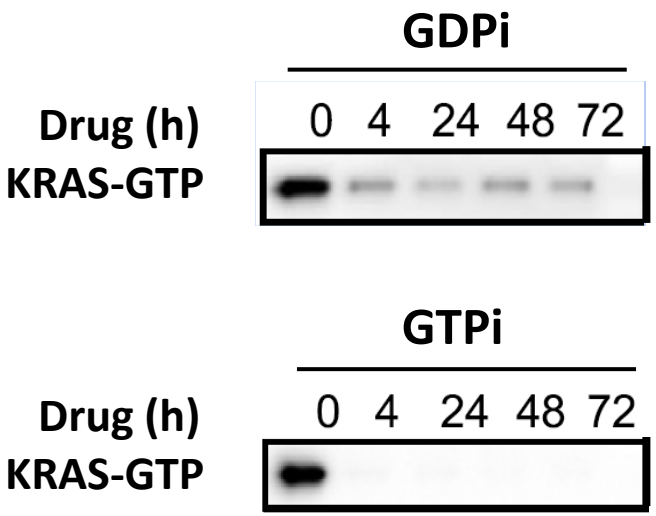
Non-NSCLC Transition

Emergent resistance

BBIO's dual KRAS^{G12C} inhibitor can address 3 of the 4 most prevalent mechanisms of resistance to current clinical KRAS^{G12C} inactive inhibitors

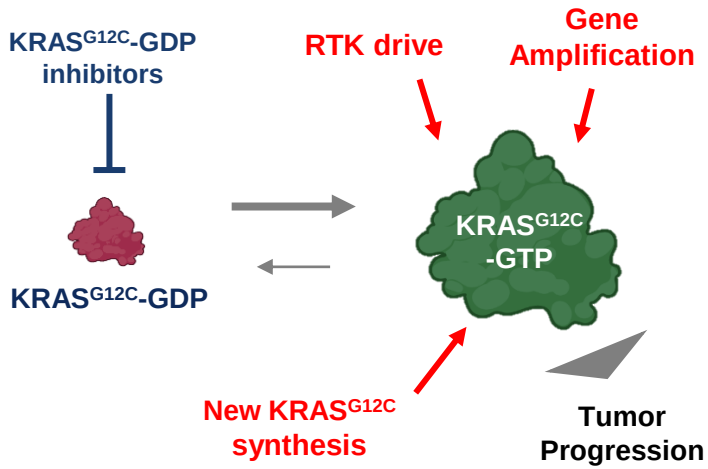
A compound that inhibits both GTP (active) and GDP (inactive) forms of KRAS^{G12C} will be superior to one that only inhibits the latter

GDPi does not achieve complete inhibition of "active" KRAS^{G12C}*



Growth factors render GDPi completely inactive

KRAS^{G12C} prefers the GTP state: GTP levels are 10x GDP levels



- A mechanism of resistance was not identified for most NSCLC patients that became refractory to sotorasib**
- Among patients with identified resistance mechanisms to sotorasib, the majority were driven by RTK re-activation**

We believe efficacy of targeting of KRAS^{G12C} can be improved by targeting the oncogenic active GTP form

*Ryan et al. Cell Reports 2022; **Li BT, et al. Journal of Clinical Oncology 40, no. 16_suppl (June 01, 2022) 102-102

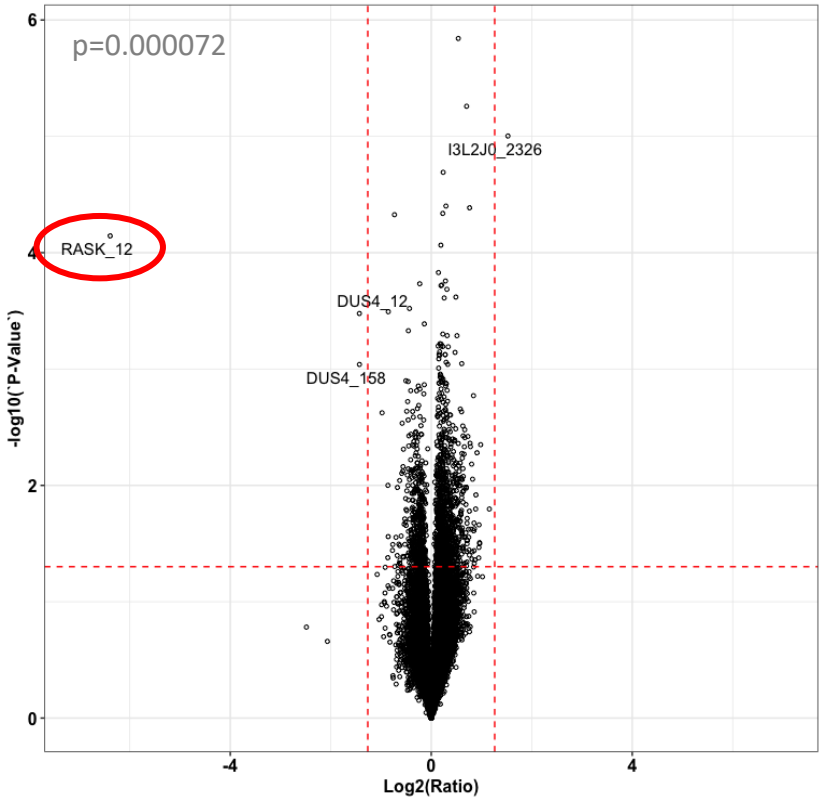
BBO-8520 completely modifies both GTP (active) and GDP (inactive) forms of $KRAS^{G12C}$ and is exceptionally potent

			BBO-8520	Sotorasib	Adagrasib	GDC-6036
% modified	$KRAS^{G12C}$ GTP (active)	15’	100	0	0	0
		60’	100	0	0	0
	$KRAS^{G12C}$ GDP (inactive)	15’	91	80	73	77
		60’	100	82	84	84
$KRAS^{G12C}$: RAF1 Effector Binding IC_{50} (nM)			33	>100,000	20,000	4,200
H358 pERK IC_{50} @ 30’ (nM)			4	50	310	8
H358 kinact/Ki (M*s)-1			43,000	776	1064	27,000

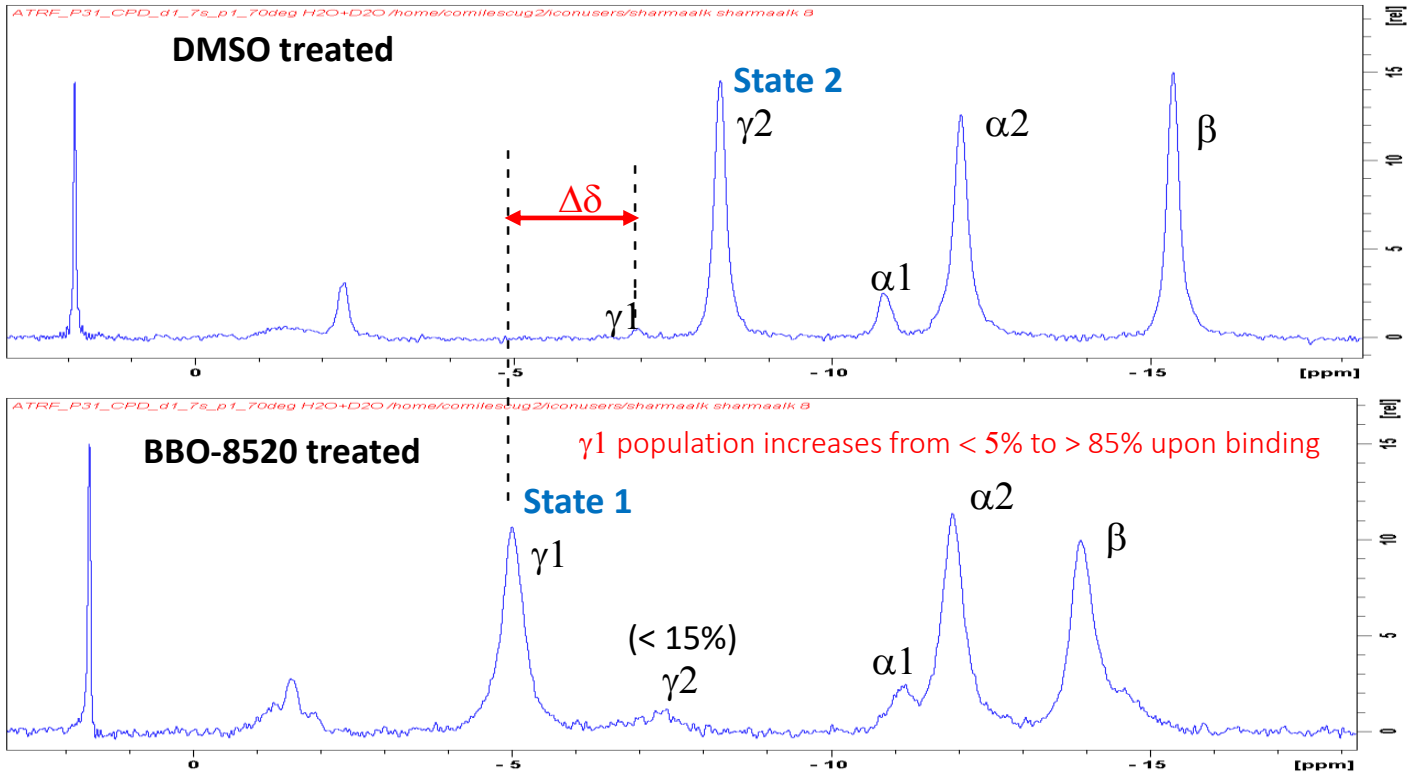
- High degree of protein modification supports high affinity binding to GTP state
- Potent inhibition of effector binding and oncogenic signaling
- Superior kinact/Ki

Cysteine proteome selectivity and mechanism of action

Global cysteine proteomics shows high degree of selectivity for G12C



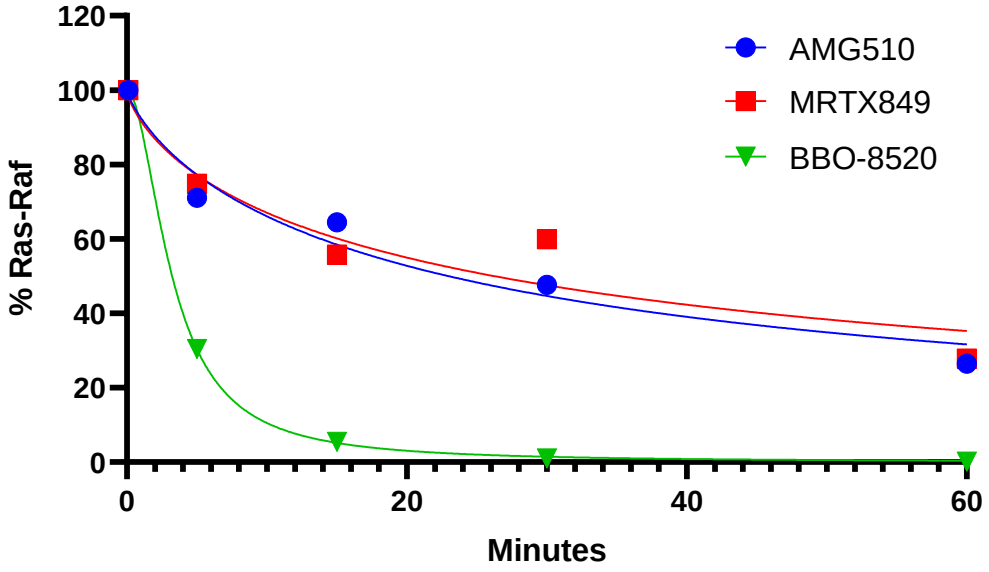
^{31}P NMR peak shifts suggest that BBO-8520 stabilizes State 1 of active GTP-bound KRAS, which disrupts effector protein binding



^{31}P NMR spectrum @ 5 °C

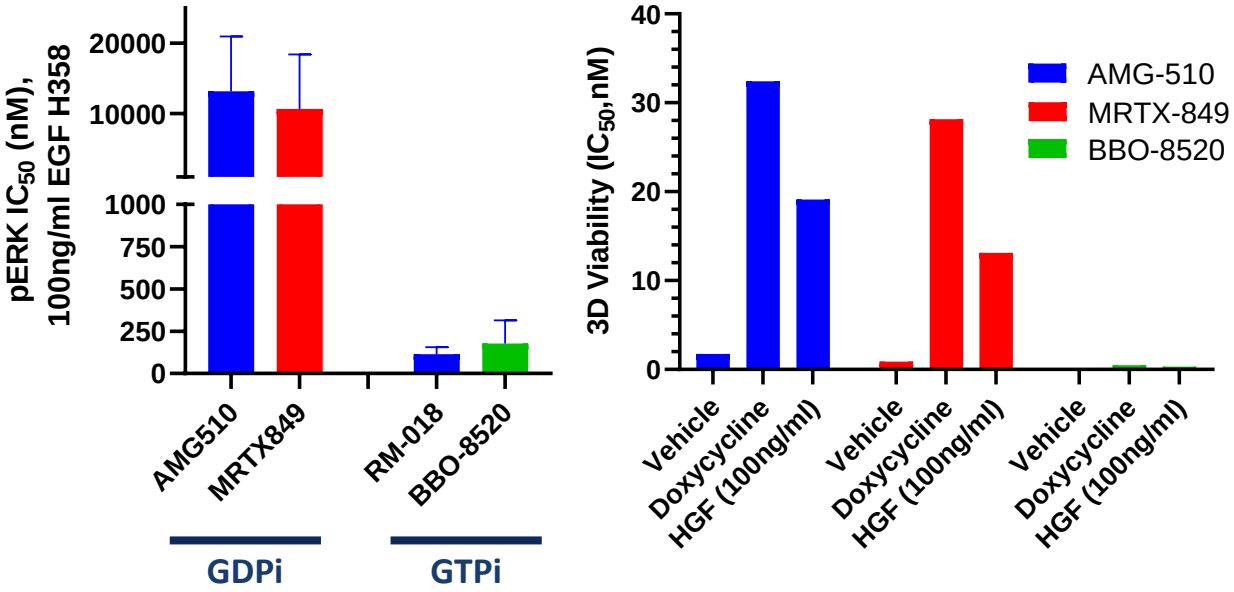
Targeting KRAS^{G12C}-GTP activity allows for rapid signal inhibition and overcomes RTK drive

Rapid and complete inhibition of KRAS^{G12C}-GTP



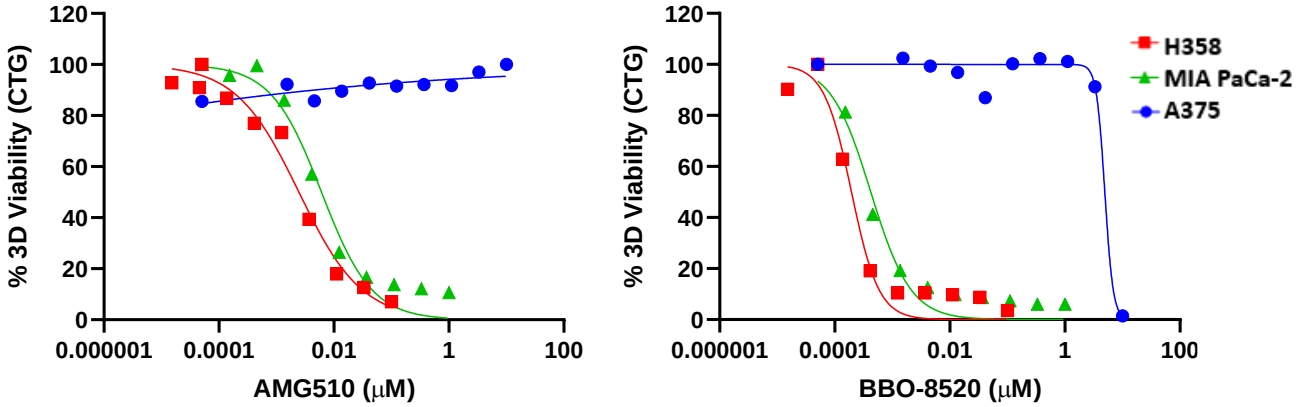
Compound	MALDI-TOF% GTP, 5min	Time (min) to IC_{50}	% of AMG510 Time to IC_{50}
AMG510	0	22	100
MRTX849	0	26	118
BBO-8520	**94**	**3.0**	**14**

GFs abundantly present in human tissues render GDP inhibitors inactive



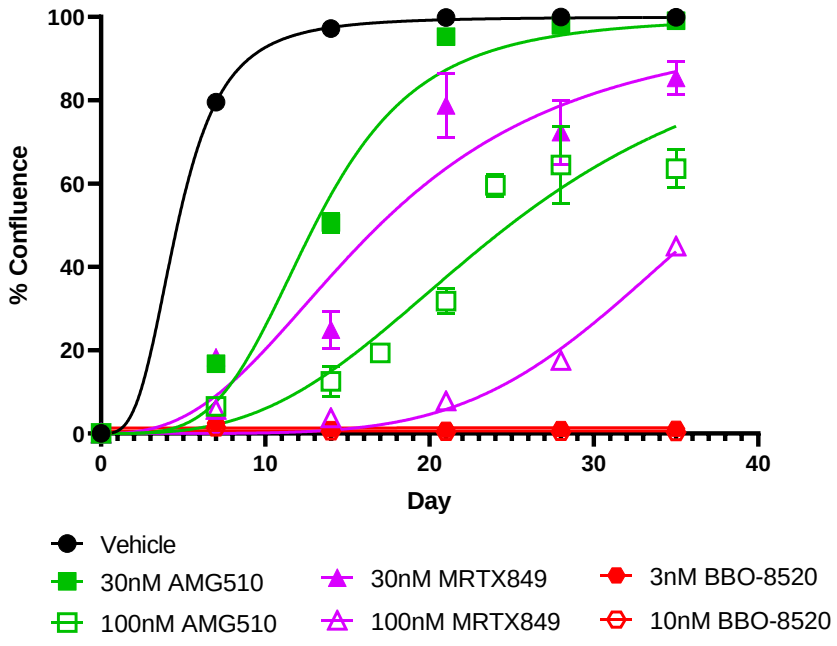
Cellular data support hypothesis that targeting the GTP form yields greater potency and deeper responses

10x increased potency is observed in viability assays



Compound	IC_{50} (nM)	
	H358	MIAPaCa-2
AMG510	2	5
BBO-8520	0.2	0.3

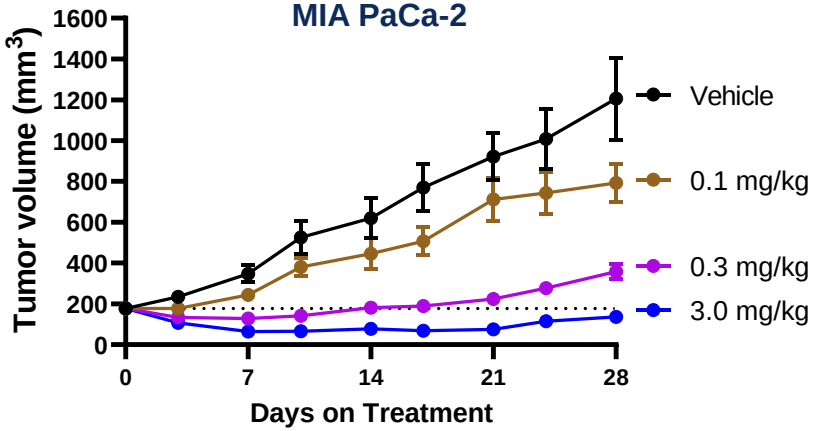
H358 Clonogenic Assay suggests GTPi may reduce development of resistance



BBO-8520 retains single-digit nM activity against reported GDP-inhibitor active-site mutants, including G12C/R68S, G12C/Y96D, G12C/G13D, G12C/Q61H, and G12C/A59G

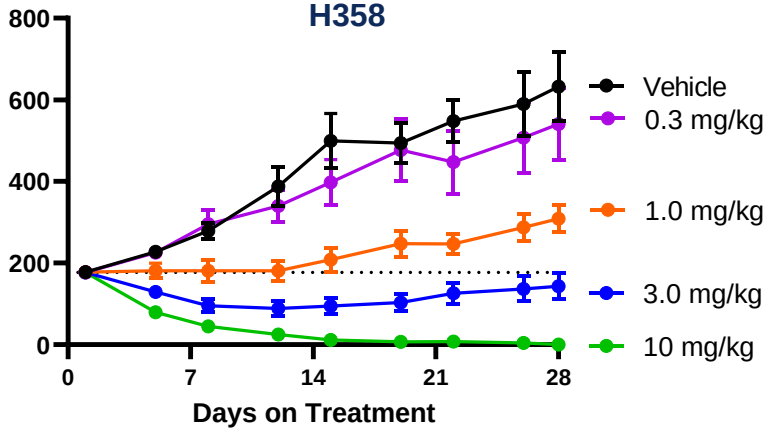
BBO-8520 exhibits strong efficacy in KRAS^{G12C} models

High Potency



ED_{50}	ED_{90}
0.13 mg/kg	0.40 mg/kg
EC_{50}	EC_{90}
4.6 nM	9.9 nM

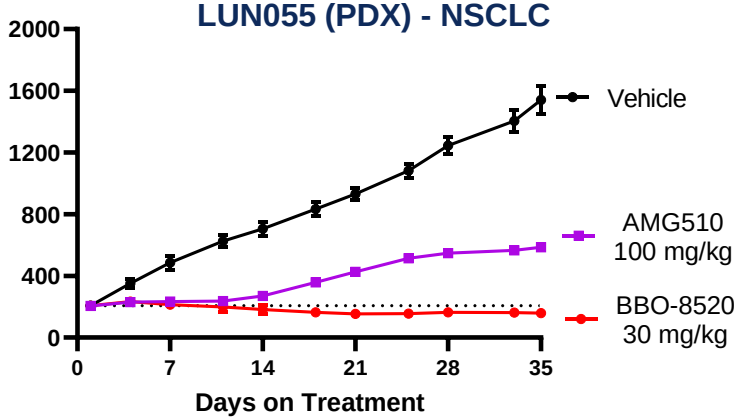
Deep Efficacy



10/10 CRs at 10 mg/kg

ED_{50}	ED_{90}
0.61 mg/kg	1.6 mg/kg
EC_{50}	EC_{90}
14 nM	34 nM

Differentiated



Group (n=10)	Day 35		
	TGI	Regression	FF AUC_{0-24} (ng*hr/ml)
BBO-8520	100%	23% (7/10)	59
AMG510	71%	- (1/10)	1563

BBO-8520 is efficacious in cell line and PDX models with high potency, deep efficacy, and differentiated activity

BBO-8520: G12C Dual Inhibitor Development Candidate

- BBO-8520 is a potential “first-in-class” direct $KRAS^{G12C}$ dual inhibitor
  - Completely modifies both GTP (active) and GDP (inactive) forms of $KRAS^{G12C}$
  - Exceptionally potent and selective with superior kinact/Ki
  - Binding stabilizes GTP-bound $KRAS^{G12C}$ in state 1 which cannot bind effectors
  - Overcomes RTK drive
  - Strong efficacy in $KRAS^{G12C}$ models
- IND projected 2023
- Therapeutic opportunity in $KRAS^{G12C}$ mutant NSCLC, CRC and other GI tumors in both GDP-$KRAS^{G12C}$ inhibitor naïve and experienced patients

Agenda

BBO-8520: A KRASG12C Dual Inhibitor

PI3Kα:RAS Breaker

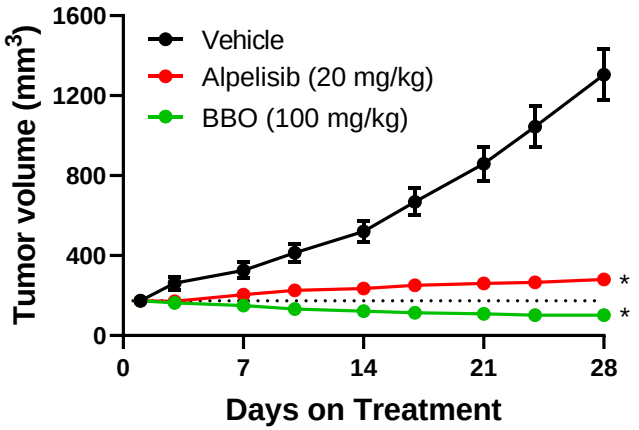
Pan-KRAS program

PI3Kα:RAS Breaker is a novel, potent and differentiated therapeutic approach that can deliver efficacy in multiple common tumor genotypes as monotherapy or in combination

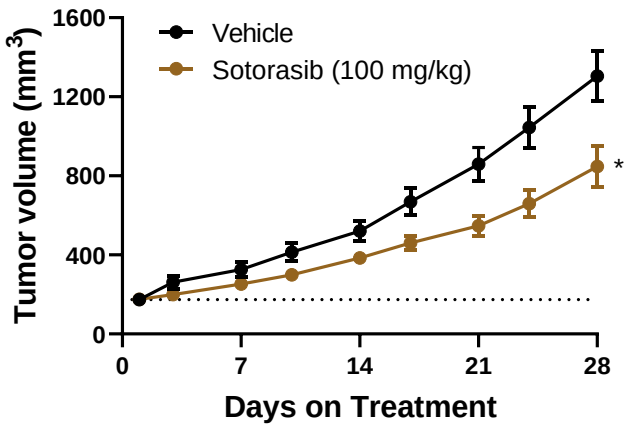
- First-in-class molecular mechanism targeting the second most mutated oncogene in human cancer
- Molecular mechanism provides tumor selectivity and prevents well-known target liability (hyperglycemia)
- Proven activity in common tumor genotypes ($KRAS^{G12x}$, PIK3CA helical and HER2/HER3)
- Differentiated activity from Her2 and KRAS approved inhibitors (tucatinib, sotorasib, etc)

KYSE-410 CDX esophageal carcinoma - $HER2^{amp}$ $KRAS^{G12C}$

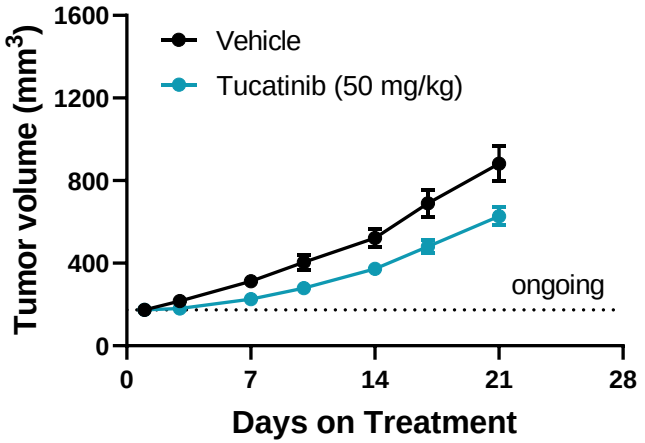
PIK3CA inhibition



$KRAS^{G12C}$ inhibition

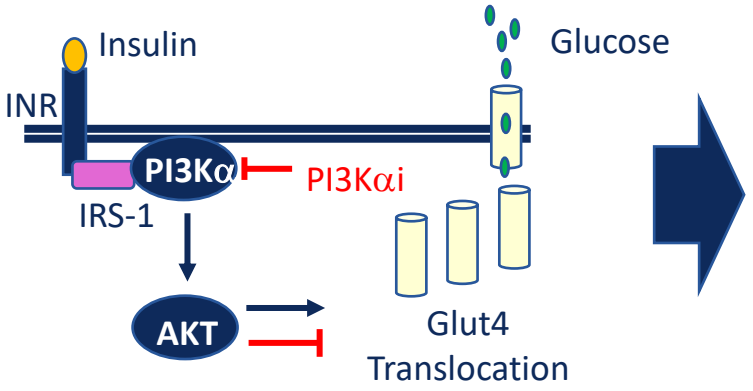


Her2 inhibition

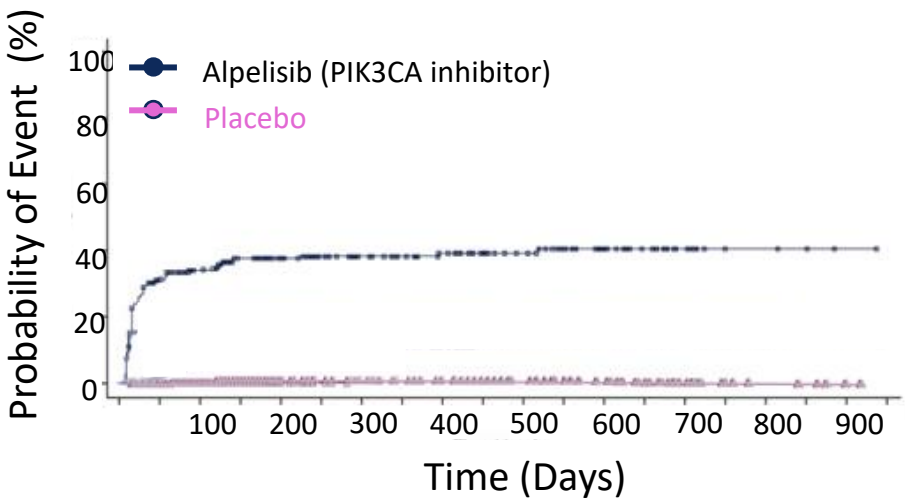


Tucatinib is a HER2 inhibitor, sotorasib is a KRAS-G12C inhibitor, alpelisib is a PI3Kα inhibitor. All groups dosed PO, QD, *p<0.05 RM ANOVA vs vehicle

Inhibiting the 2nd most mutated oncogene (PIK3CA) in human cancer has been limited by side effects of glucose metabolism



Solar-1 study – Hyperglycemia & Efficacy*



	PFS (mo)	ORR (%)
Fulvestrand	5.7	12.8
Alpelisib + Fulvestrand	11	26.6

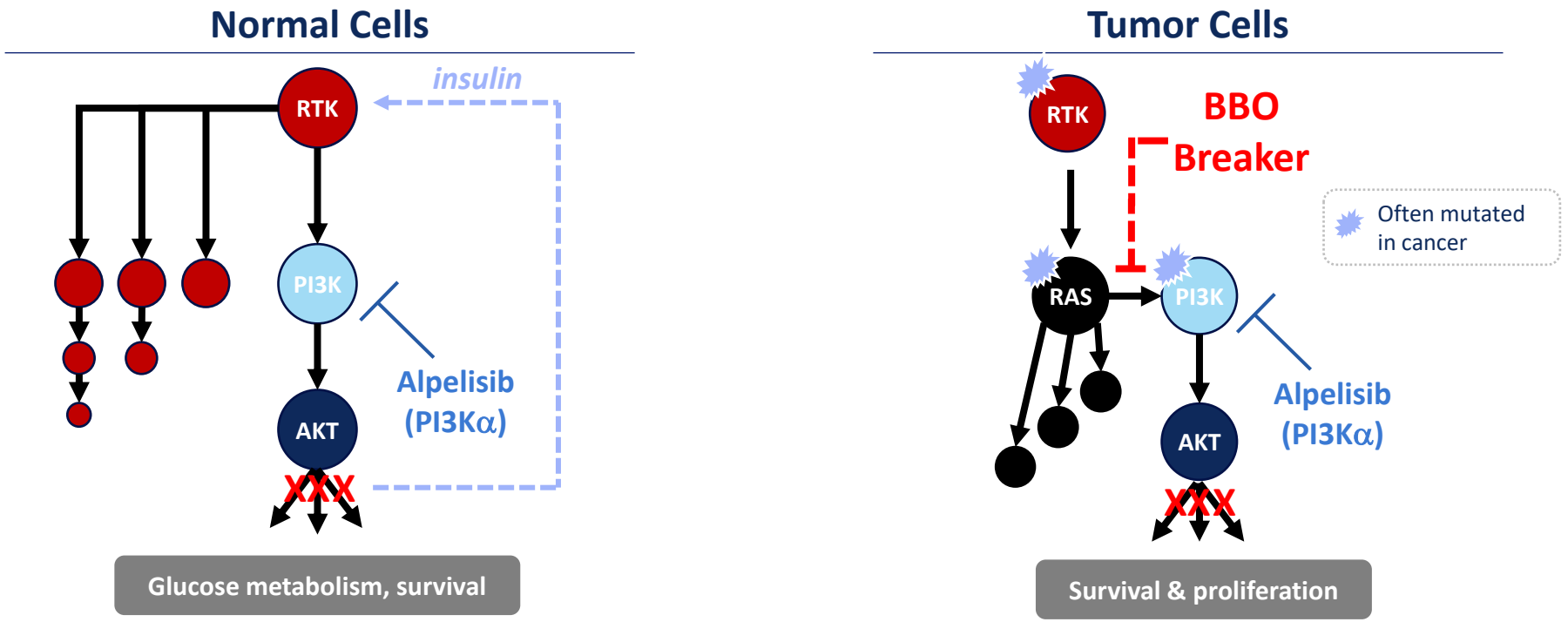
- **High rate of dose modifications and interruptions (>30%) does not allow effective target coverage**
- **Adverse events are not conducive to combination studies**
- **Increased insulin secretion leads to increased pathway signaling and resistance**

- **Dose interruptions occurred in 66% versus 21% in placebo**
- **Dose reductions due to adverse events occurred in 55% versus 4.5% in placebo**
- **The most common adverse reactions were hyperglycemia (65%), diarrhea (58%), and rash (52%)**

*Adapted from Rugo et al. Annals of Oncology 2020

Inhibiting PI3Kα activity by preventing its interaction with RAS provides a "tumor selective" mechanism that spares glucose metabolism

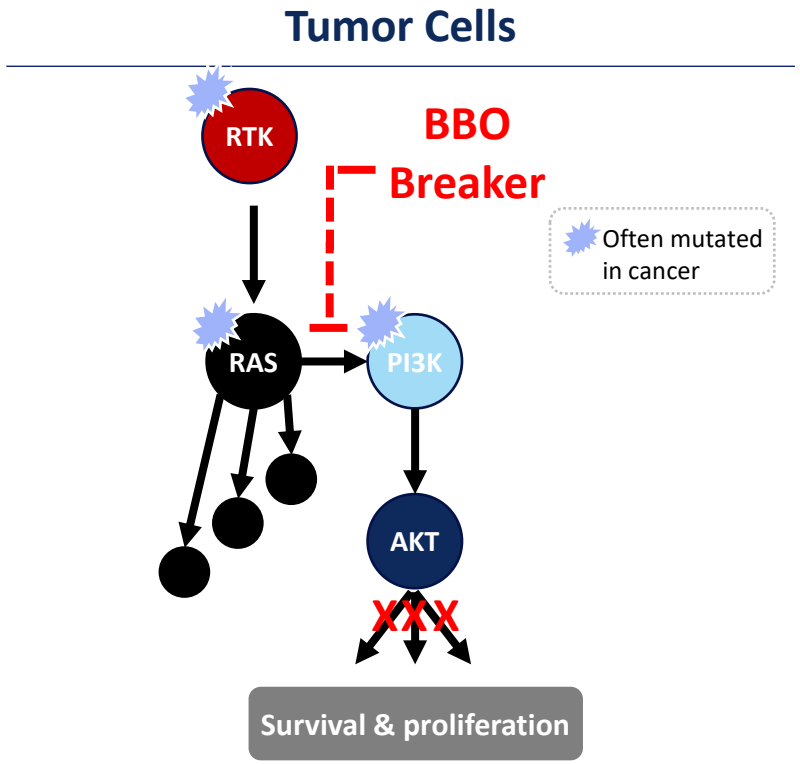
- PI3Kα kinase inhibitors ***block normal cell signaling*** resulting in ***dose-limiting hyperglycemia and insulin-driven resistance***
- Inhibiting PI3Kα:RAS PPI with a ***"PI3Kα Breaker"*** should avoid hyperglycemia and insulin-driven resistance by specifically targeting tumor cells and may provide multiple therapeutic opportunities
- Mice with mutations in the RBD that impair the PI3Kα:RAS interaction block oncogene-driven NSCLC tumor growth *in vivo* and have no effect on glucose metabolism*



PPI, protein-protein interactions; RBD, RAS-binding domain

*Gupta et al. Cell 2007; Castellano et al. Cancer Cell 2013; Murillo et al., Cell Reports 2018

BridgeBio has designed potent and selective PI3Kα:RAS breakers

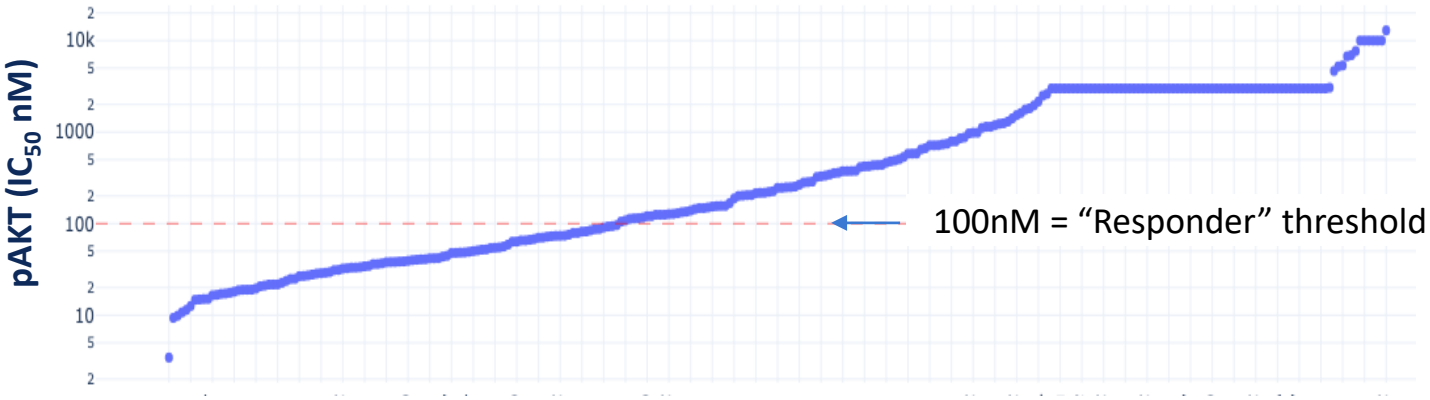


- PI3Kα:RAS breakers selectively bind to PI3Kα
- By ITC and SPR we observe
  - RAS binds to PI3Kα with ~10 μM affinity
  - Breakers binding to PI3Kα blocks its interaction with RAS
  - No binding affinity to RAS
- PI3Kα:RAS breakers do not affect kinase activity of PI3Kα

		BBO	Alpelisib
BT474	pAKT (IC_{50}, nM)	34	169
	Cell Viability (nM)	67	744

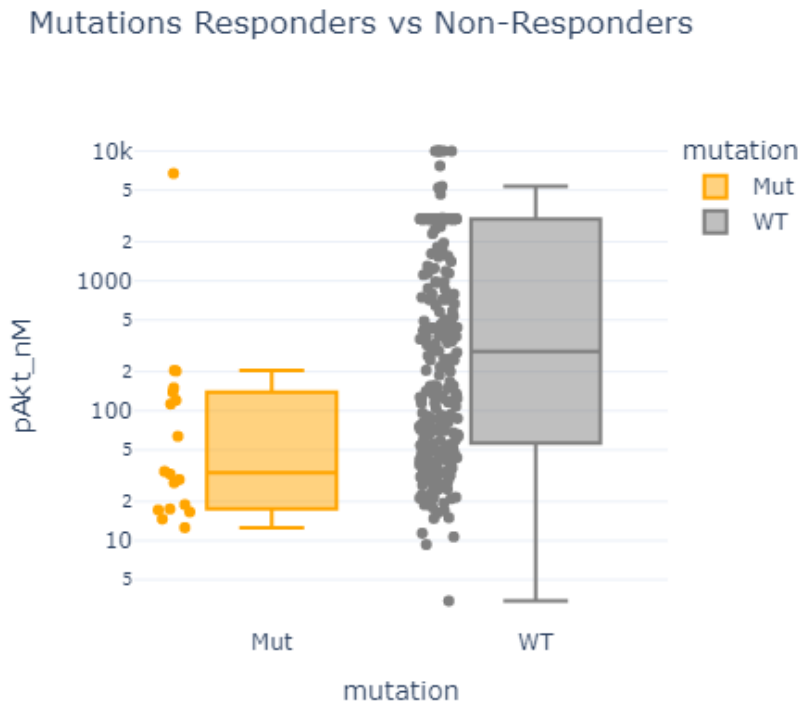
One third of all cancer cell lines depend on PI3Kα:RAS interaction for activation of AKT signaling

pAKT cell line screen



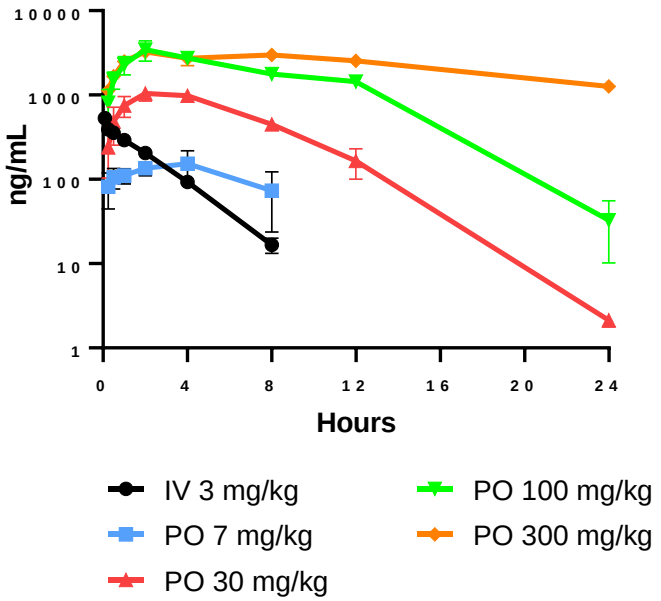
- 105/282 (37%) of screened cell lines are responders
- Responders include
  - 29/50 (58%) KRAS^{G12X} mutant
  - 18/19 (94%) PIKCA helical mutant
  - 16/21 (76%) HER2 amp

PIK3CA helical mutants are highly sensitive

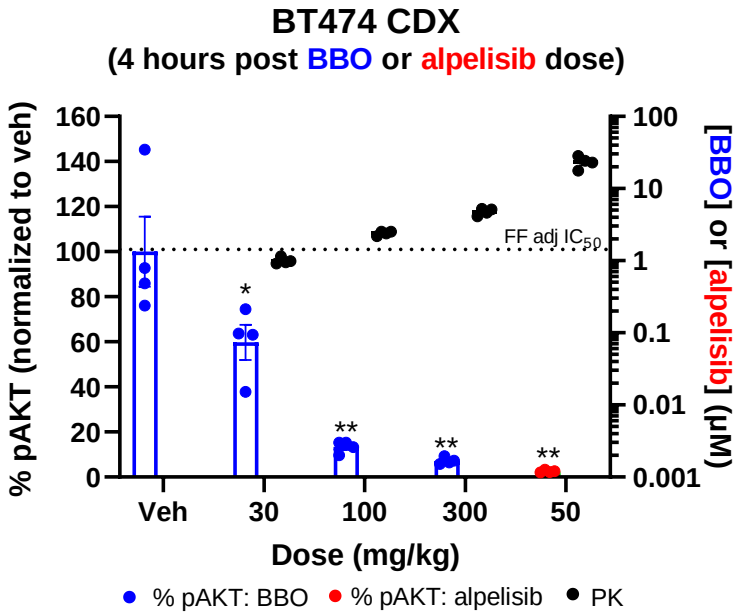


BBO is orally bioavailable and achieves near complete inhibition of signaling in tumors at 100 mg/kg without risk of hyperglycemia

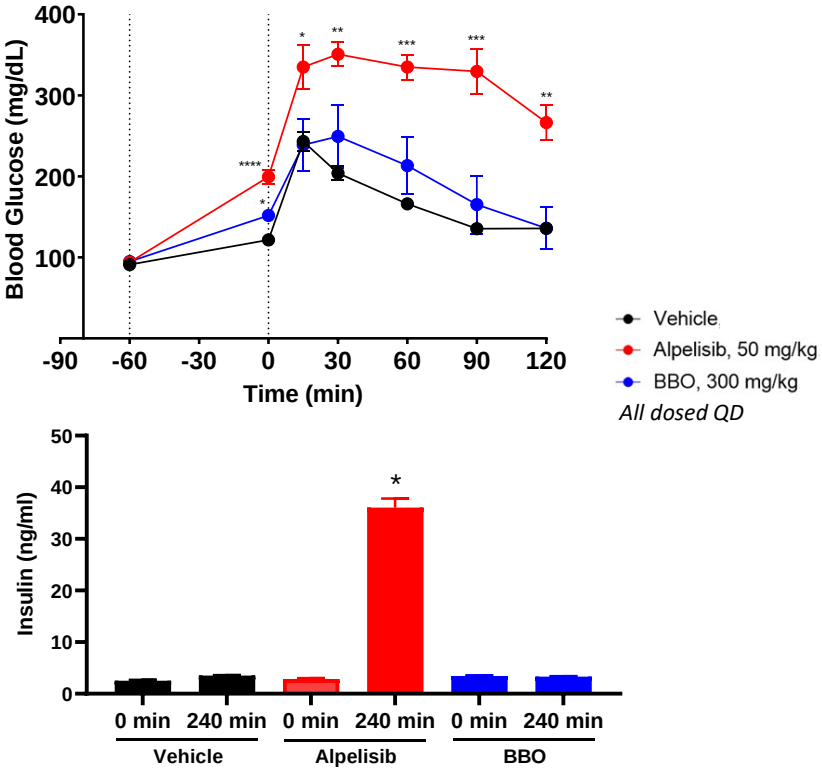
BBO Dose Ranging Mouse PK



BBO Dose Response PD[1]
Full target inhibition achieved at 100 mg/kg



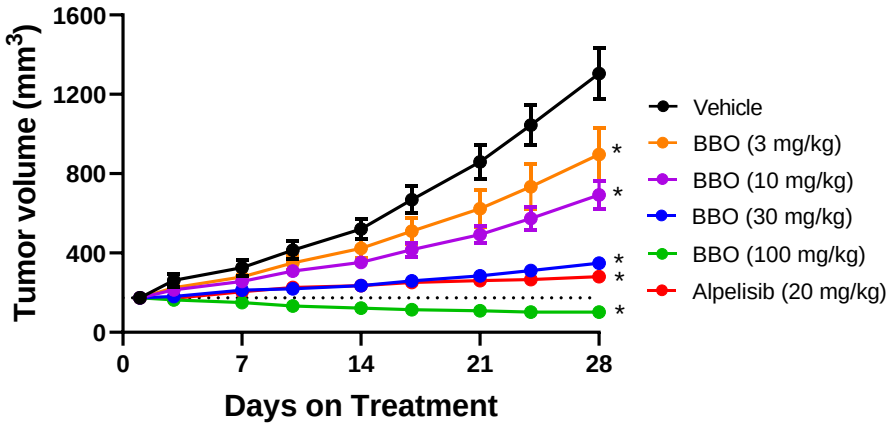
Unlike alpelisib, Breaker MOA does not affect glucose metabolism[2]



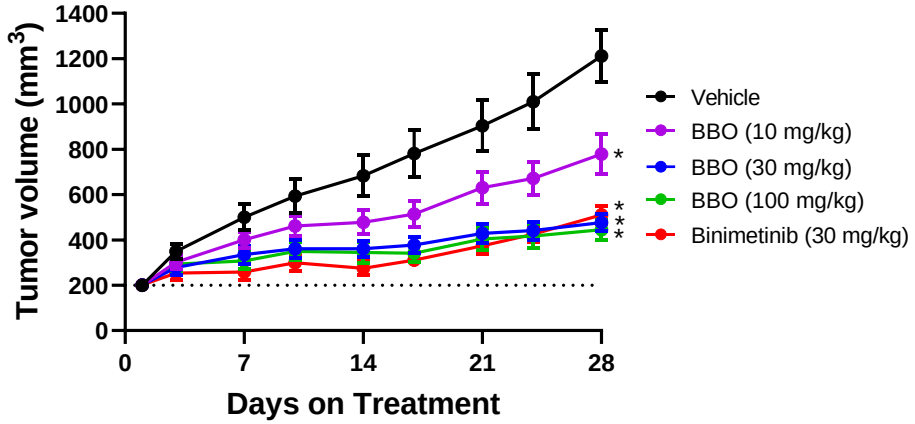
1. One-way ANOVA with Dunnett's test vs vehicle; *p<0.01, **p<0.0001
2. Top: One-way ANOVA with Dunnett's test vs vehicle, *p<0.05, **p<0.01, ***p<0.001, ****p<0.0001, Bottom: One-way ANOVA with Tukey's multiple comparisons test vs all other groups : *p<0.0001

PI3Kα breakers are efficacious in xenograft models

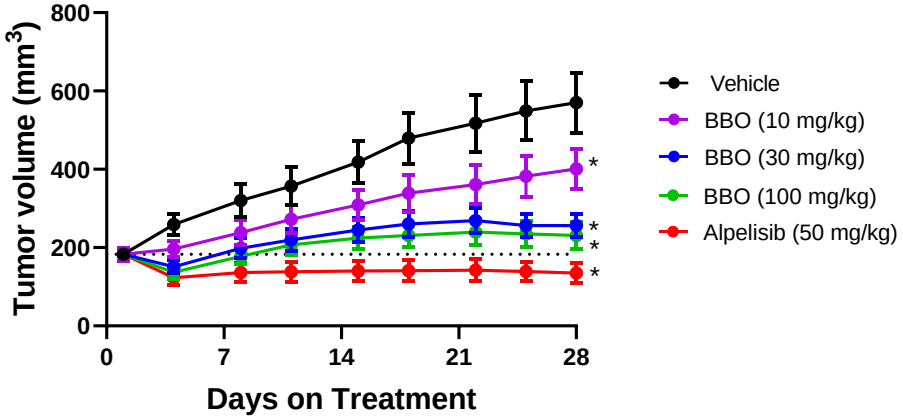
KYSE-410 CDX
- $KRAS^{G12C}$
- $HER2^{amp}$



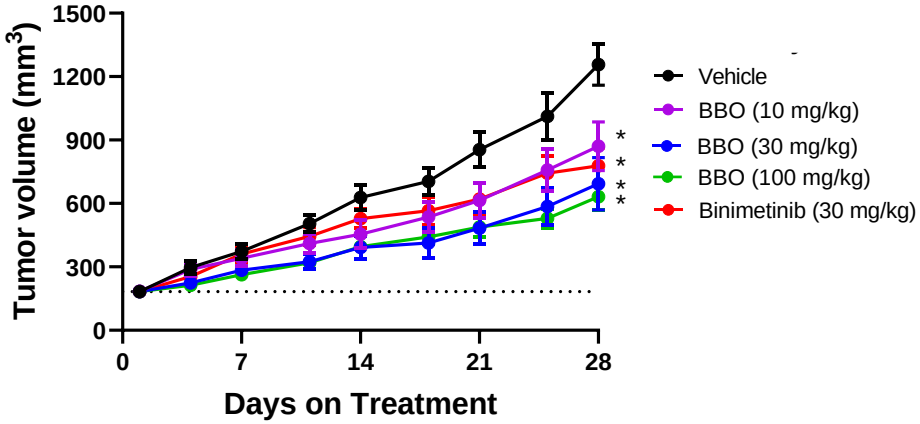
GP2d CDX
- $KRAS^{G12D}$
- $PIK3CA^{H1047L}$



SNU-601 CDX
- $KRAS^{G12D}$
- $PIK3CA^{E542K}$



SNU-16 CDX
- $KRAS^{G12D}$



Efficacy is observed in models with $KRAS^{G12X}$ mutations, with or without PIK3CA mutation

All groups dosed PO, QD, *p<0.0005 RM ANOVA vs vehicle

BridgeBio has designed potent and selective PI3Kα:RAS breakers

- Potential first-in-class opportunity
  - Novel mechanism of action: PI3Kα breakers selectively block RAS activation of PI3Kα
  - Exhibits potent inhibition of AKT activation in $KRAS^{G12x}$, PIK3CA helical mutations and HER family driven populations
  - Potent efficacy in multiple models without hyperglycemia
- Development candidate projected 2023
- Multiple opportunities as monotherapy and in combination in large patient populations

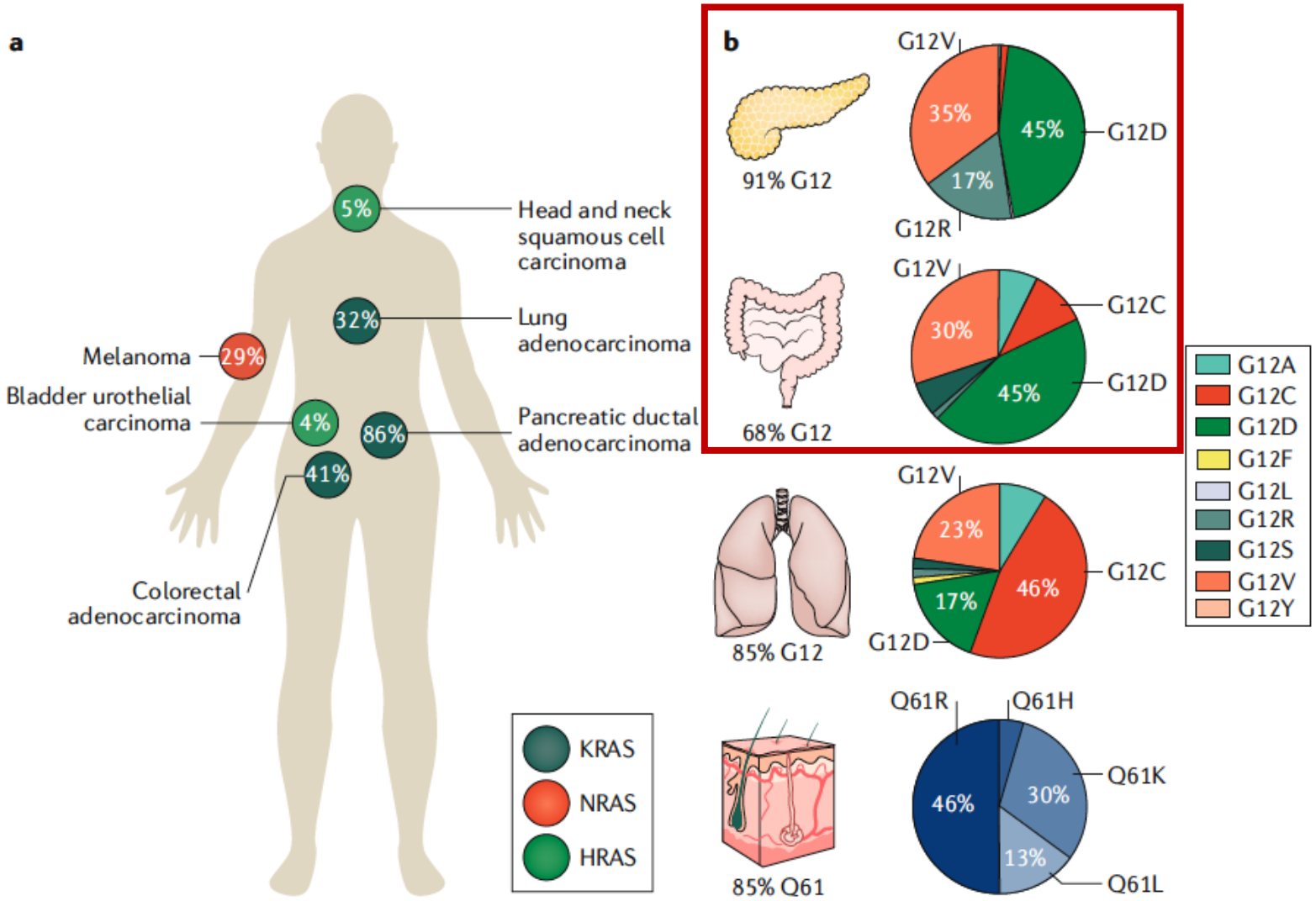
Agenda

BBO-8520: A KRASG12C Dual Inhibitor

PI3Kα:RAS Breaker

Pan-KRAS program

Mutant KRAS is the most common oncogene in cancer – pan KRAS



- Lung cancer is the second most common cancer in the US with greater than 235K new cases and 130K deaths a year
- $KRAS^{G12C}$ mutant found in ~15% of all NSCLC (~35K pts/yr)
- Other common human cancers with KRAS mutations are colorectal and pancreatic adenocarcinomas with a combined 168K new cases a year in the US
- ***$KRAS^{G12D/G12V}$ mutant found in 70% of pancreatic cancers (~43K pts/yr) and 25% of colorectal cancers (~53K pts/yr)***

Figure from: Moore, Rosenberg, McCormick, Malek Nat Rev Drug Disc 2020

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Pan-KRAS program: current lead molecules

- **Recent progress has identified molecules with the right potency and bioavailability**

		BBO-a	BBO-b
PPI: KRAS/RAF1 effector IC_{50} (nM)	G12D	110	100
	G12V	430	270
pERK: HTRF IC_{50} (nM)	GP2D (G12D) @ 1h	4	7
	SW620 (G12V) @ 4 h	10	13
Mouse PK (IV ER % / PO %F)		64 / 6.9 (10 mpk)	**44 / 34 (10 mpk)**

Potent inhibitory activity against multiple KRAS-mutant models *in vitro*

KRAS variant	BBO-b EC_{50} (nM)	
	pERK	Viability
G12D	6.7	10.3
G12V	2.7	165
G12C	11.0	11.2
G12S	126	203
G13D	37.1	326
G12A	406	86.1
$BRAF^{V600E}$	>10 uM	>3.5 uM

We have identified leads with strong *in vivo* target engagement which are progressing into efficacy studies

Pan-KRAS: Lead optimization progressing towards a development candidate

- Potent activity against multiple KRAS mutants
- Selective for KRAS over H- and NRAS
- Potent PD and good mouse oral bioavailability
- Development candidate projected 2023

BridgeBio is progressing multiple approaches against KRAS

- **Dual KRAS^{G12C} inhibitor** program has selected a Development Candidate (BBO-8520) with projected IND-filing 2023

- **PI3Kα:RAS breaker** is highly differentiated in that it is a) tumor selective, b) targets both mutant and wild-type PIK3CA, and c) does not induce hyperglycemia
- Projected Development Candidate selection in 2023

- **pan-KRA**S program targets multiple KRAS mutants including KRAS^{G12D} and KRAS^{G12V}
- Projected Development Candidate selection in the 2023