

ONCOLOGY CLINICAL UPDATE ESMO 2021

SEPTEMBER 16, 2021

AMGEN[®]

SAFE HARBOR STATEMENT

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AGENDA

Introduction

**David Reese, M.D.—Executive Vice President,
Research and Development**

LUMAKRAS™ Update

**P.K. Morrow, M.D.—Vice President, Global
Development**

Q&A

All



INTRODUCTION

DAVID REESE, M.D.

EXECUTIVE VICE PRESIDENT, RESEARCH AND DEVELOPMENT



AMGEN ONCOLOGY-HEMATOLOGY: A BROAD, DIFFERENTIATED PORTFOLIO

- Built on first-in-class molecules directed against high-quality targets in areas of high unmet need
- Developing combination/sequential therapies against multiple targets and indications to drive deep, durable responses
- Prioritizing high-potential programs for rapid advancement
 - First-in-class KRAS^{G12C} inhibitor LUMAKRAS[™] (sotorasib)
 - FGFR2b antibody bemarituzumab for gastric cancer
 - BiTE[®] immuno-oncology platform clinically validated in solid and hematologic tumors

ADVANCING FIRST-IN-CLASS MOLECULES AGAINST HIGH-QUALITY TARGETS FOR BOTH SOLID AND HEMATOLOGIC MALIGNANCIES

Solid Tumors				Hematologic Malignancies			
Tumor Type	Molecule	Target	Modality	Tumor Type	Molecule	Target	Modality
Gastric and Gastroesophageal Junction Cancer	Bemarituzumab	FGFR2b	Monoclonal Ab	Acute Lymphoblastic Leukemia	BLINCYTO® (blinatumomab)	CD19	BiTE® Molecule
	AMG 199	MUC17	HLE-BiTE® Molecule				
	AMG 910	CLDN18.2	HLE-BiTE® Molecule				
Melanoma	IMLYGIC®		Oncolytic Virus	Acute Myeloid Leukemia	AMG 330	CD33	BiTE® Molecule
Non-Small Cell Lung Cancer	Acapatamab (AMG 160)	PSMA	HLE-BiTE® Molecule				
Prostate Cancer	Acapatamab	PSMA	HLE-BiTE® Molecule		AMG 427	FLT3	HLE-BiTE® Molecule
	Tarlatamab (AMG 757)	DLL3	HLE-BiTE® Molecule	Hematologic Malignancies			
	AMG 509	STEAP1	Bivalent T-cell engager XmAb® 2+1 Ab		AMG 176	MCL1	Small Molecule
Small Cell Lung Cancer	Tarlatamab	DLL3	HLE-BiTE® Molecule				
Solid Tumors	LUMAKRAS™	KRAS ^{G12C}	Small Molecule	Multiple Myeloma	Kyprolis	Proteasome	Small Molecule
	AMG 256	PD-1 / IL-21	Bifunctional Fusion Protein				
	AMG 650	KIF18A	Small Molecule		Pavurutamab (AMG 701)	BCMA	HLE-BiTE® Molecule
	AMG 994	Undisclosed	Bifunctional Fusion Protein				

Ab = antibody; BCMA = B-cell maturation antigen; BiTE® = bispecific T-cell engager; CD = cluster of differentiation; CLDN = claudin; DLL3 = delta-like ligand 3; EGFRvIII = epidermal growth factor receptor variant III; FGFR2b = fibroblast growth factor receptor 2b; FLT3 = FMS-like tyrosine kinase 3; HLE = half-life extended; KIF18A = Kinesin Family Member 18A; MUC17 = mucin 17; PSMA = prostate-specific membrane antigen; STEAP1 = six transmembrane epithelial antigen of the prostate 1; Additional ongoing clinical programs can be found at [Amgenpipeline.com](https://www.amgenpipeline.com)

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LUMAKRAS™: THE FIRST AND ONLY APPROVED KRAS^{G12C} INHIBITOR

- **Very positive reception to U.S. launch—increased awareness and testing**
- **Recently approved in Canada and U.K.—multiple ongoing global regulatory reviews, including EU and Japan**
- **Largest and most comprehensive global program with ~ 3,000 patients treated***
- **Broad-based combination approach including triplet therapy**

Exemplifies Amgen's Research, Development, Regulatory, and Commercial excellence in developing innovative medicines

*Includes clinical studies, early access programs and commercial patients
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LUMAKRAS™: ADVANCING THE MOST COMPREHENSIVE GLOBAL KRAS^{G12C} CLINICAL DEVELOPMENT PROGRAM

Phase	Tumor Type	Treatment Regimen
Phase 1	First-line NSCLC	Monotherapy
Phase 1	NSCLC, CRC, other solid tumors	Monotherapy*
Phase 1b/2	NSCLC with active brain metastases	Monotherapy
Phase 1b/2	NSCLC	+ Oral EGFR inhibitor (afatinib) + PDL1 inhibitor (atezolizumab) + Chemotherapy (carboplatin, premetrexed, docetaxel)
Phase 1b/2	CRC	+ EGFR Ab (panitumumab) +/- chemotherapy (FOLFIRI) + VEGF Ab (bevacizumab-awwb) + chemotherapy (FOLFIRI or FOLFOX)
Phase 1b/2	NSCLC, CRC, other solid tumors	+ PD-1 inhibitor (AMG 404) (pembrolizumab) + MEK inhibitor (trametinib) +/- EGFR Ab (panitumumab) + SHP2 inhibitor (RMC-4630) (TNO155) + mTOR inhibitor (everolimus) + CDK inhibitor (palbociclib)
Phase 2	NSCLC, CRC, other solid tumors	Monotherapy
Phase 2	First-line NSCLC with STK11 mutated or PD-L1–tumors	Monotherapy
Phase 3	NSCLC	Monotherapy vs. docetaxel

*In subjects of Chinese descent; NSCLC = non-small cell lung cancer; CRC = colorectal cancer; EGFR = epidermal growth factor receptor; PD-L1 = programmed death-ligand 1; Ab = antibody; FOLFIRI = fluorouracil, leucovorin, and irinotecan; VEGF = vascular endothelial growth factor; FOLFOX = fluorouracil, leucovorin, and oxaliplatin; PD-1 = programmed cell death protein 1; MEK = mitogen-activated protein kinase kinase; SHP2 = Src homology region 2-containing protein tyrosine phosphatase 2; mTOR = mammalian target of rapamycin; CDK = cyclin-dependent kinase; STK11 = serine/threonine kinase 11

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ANTICIPATED LUMAKRAS™ MILESTONES

H2 2021

Study initiations

- Phase 3 study in combination with Vectibix® in patients with 3L+ CRC
- Phase 2 study in patients with 1L NSCLC and STK11 mutated and/or PDL-1 negative tumors

Combination data

- EGFR (afatinib)
- MEK (trametinib)

H1 2022

Top-line results

- Confirmatory Phase 3 study vs. docetaxel in patients with 2L+ NSCLC
- Phase 2 study in patients with advanced solid tumors other than NSCLC and CRC

Combination data

- PD-1 (pembrolizumab)
- SHP2 (RMC-4630)
- Other cohorts TBD

BEYOND LUMAKRAS™ WE ARE ADVANCING OTHER INNOVATIVE FIRST-IN-CLASS ONCOLOGY PROGRAMS

Bemarituzumab

- Phase 3 program planned to initiate in Q4 '21 for HER2-negative, FGFR2b-positive gastric cancer
- Breakthrough Therapy Designation by FDA for patients with $\geq 10\%$ FGFR2b overexpression and HER2-negative 1L gastric cancer in combination with modified FOLFOX6
- Planning clinical studies in other solid tumors, including squamous cell NSCLC

Acapatamab (AMG 160)

- Dose expansion cohort has completed enrollment of patients with mCRPC; enrollment ongoing in cohorts exploring outpatient administration
- Dose escalation study enrolling patients with PSMA-positive NSCLC
- Studying combinations in earlier-line mCRPC with AMG 404, enzalutamide or abiraterone

Tarlatamab (AMG 757)

- Planning potentially pivotal Phase 2 study with one or more doses in patients with SCLC
- Initiating Phase 1b SCLC combination study with AMG 404
- Phase 1b study enrolling patients with neuroendocrine prostate cancer



LUMAKRAS™ (SOTORASIB) UPDATE

P.K. MORROW, M.D.

VICE PRESIDENT, GLOBAL DEVELOPMENT



RECENT LUMAKRAS™ DATA PRESENTATIONS

CodeBreaK101 subprotocol H: Phase 1b study evaluating the combination of sotorasib, a *KRAS*^{G12C} inhibitor, and panitumumab, an EGFR inhibitor, in advanced *KRAS* p.G12C-mutated colorectal cancer

**ESMO Abstract
3245**

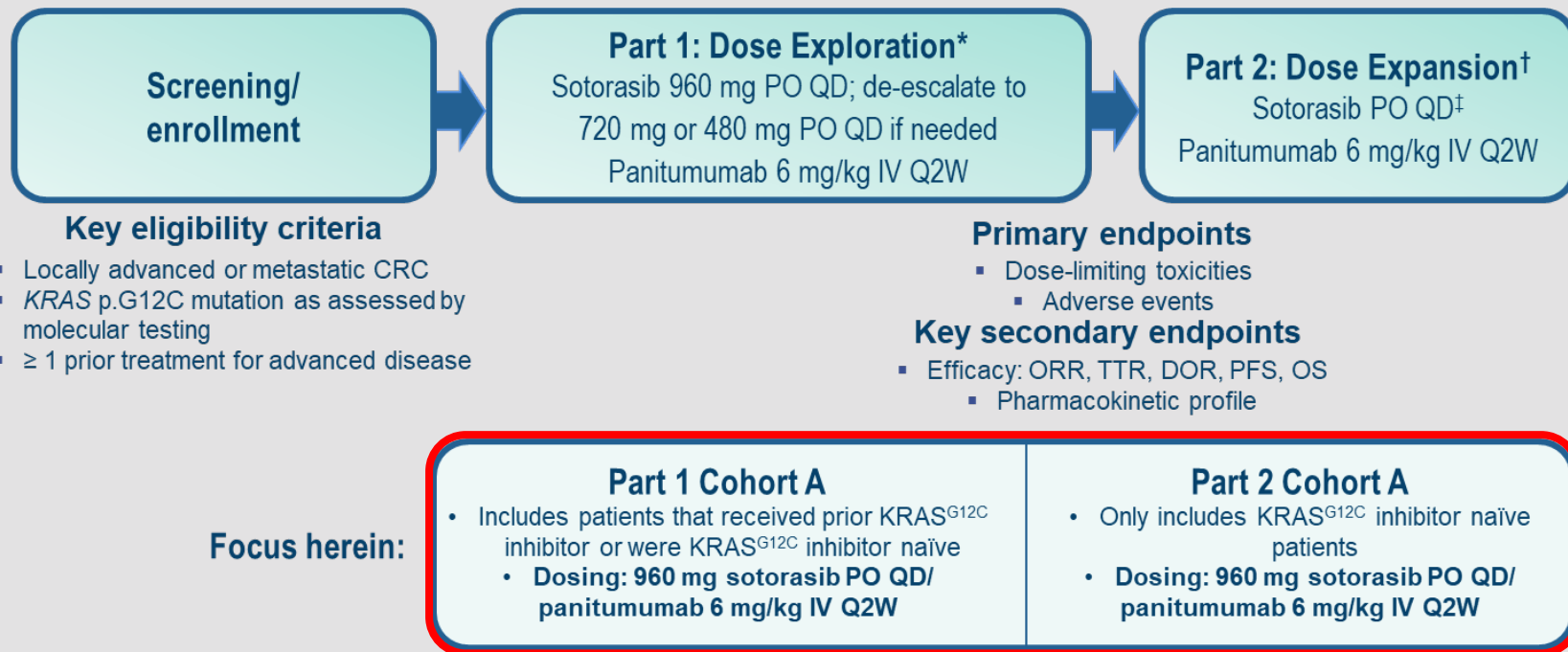
Efficacy of sotorasib in *KRAS* p.G12C-mutated NSCLC with stable brain metastases: a post-hoc analysis of CodeBreaK 100

**WCLC Abstract
P52.03**

Genomic profiles and potential determinants of response and resistance in *KRAS* p.G12C-mutated NSCLC treated with sotorasib

**WCLC Abstract
MA14.03**

CODEBREAK 101 SUBPROTOCOL H: PHASE 1B STUDY EVALUATING THE COMBINATION OF SOTORASIB AND PANITUMUMAB IN ADVANCED *KRAS* P.G12C-MUTATED COLORECTAL CANCER



DOR = duration of response; IV = intravenous; ORR = objective response rate; OS = overall survival; PFS = progression-free survival; PO = oral; Q2W = every 2 weeks; QD = daily; TTR = time to response

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BASELINE CHARACTERISTICS AND TREATMENT EXPOSURE

	Part 1 + Part 2 Combined Cohort A (N = 31) Sotorasib 960 mg PO QD/Panitumumab 6 mg/kg IV Q2W
Baseline characteristic	
Median age, years (range)	58.0 (31–79)
Female, n (%)	21 (67.7)
Median lines of therapy for metastatic disease, n (range)	2.0 (1–10)
Prior sotorasib therapy, n (%)	5 (16.1)
Exposure	
Median treatment duration of combination, week (range)	10.3 (2.1–48.1)

TREATMENT-RELATED ADVERSE EVENTS

- No dose-limiting toxicities (DLT) were observed during the first 28 days (DLT evaluation period)
- The majority of Treatment-Related Adverse Events (TRAEs) were grade 1–2 in severity

Variable	Part 1 + Part 2 Combined Cohort A (N = 31) Sotorasib 960 mg PO QD/Panitumumab 6 mg/kg IV Q2W
TRAE any grade, n (%)	23 (74.2)
Related to sotorasib	14 (45.2)
Related to panitumumab	23 (74.2)
Grade 3 TRAE, n (%)	4 (12.9)*
Grade 4 TRAE, n	0
Fatal TRAE, n	0
TRAE leading to dose modification, n, (%)	
Sotorasib	3 (9.7) [†]
Panitumumab	2 (6.5) [‡]

Sotorasib in combination with panitumumab was well tolerated, with no fatal TRAEs

*One patient experienced grade 3 hypokalemia, hypomagnesemia, dry skin, and rash (panitumumab-related); panitumumab dose modified. One experienced grade 3 dermatitis acneiform and myalgia (panitumumab-related); panitumumab dose modified only for dermatitis acneiform. One experienced grade 3 diarrhea (sotorasib-related); sotorasib dose modified. One experienced grade 3 cellulitis, edema peripheral, and dermatitis acneiform (panitumumab-related); sotorasib and panitumumab dose not changed; [†]One patient had diarrhea, one patient had fatigue and another patient had hypokalemia, resulting in dose modification of sotorasib; [‡]One patient had dermatitis acneiform and another patient had dry skin, rash, hypokalemia, and hypomagnesemia, resulting in dose modification of panitumumab.

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TUMOR RESPONSE FOR SOTORASIB IN COMBINATION WITH PANITUMUMAB

Response assessed by investigator	Part 1 Cohort A (n = 8) Sotorasib 960 mg / Panitumumab 6 mg/kg	Part 2 Cohort A (n = 18) Sotorasib 960 mg / Panitumumab 6 mg/kg	Part 1 + Part 2 Combined Cohort A (N = 26)*
Disease control rate, n (%)	6 (75.0)	15 (83.3)	21 (80.8)
ORR, % (95% CI)			
Confirmed	12.5 (0.3, 52.7)	16.7 (3.6, 41.4)	15.4
Confirmed and unconfirmed†	12.5 (0.3, 52.7)	33.3 (13.3, 59.0)	26.9
Partial response, n (%)			
Confirmed	1 (12.5)	3 (16.7)	4 (15.4)
Confirmed and unconfirmed†	1 (12.5)	6 (33.3)	7 (26.9)
Stable disease, n (%)	5 (62.5)	12 (66.7)	17 (65.4)
Progressive disease, n (%)	1 (12.5)	2 (11.1)	3 (11.5)
Not done, n (%)	1 (12.5)	1 (5.6)	2 (7.7)

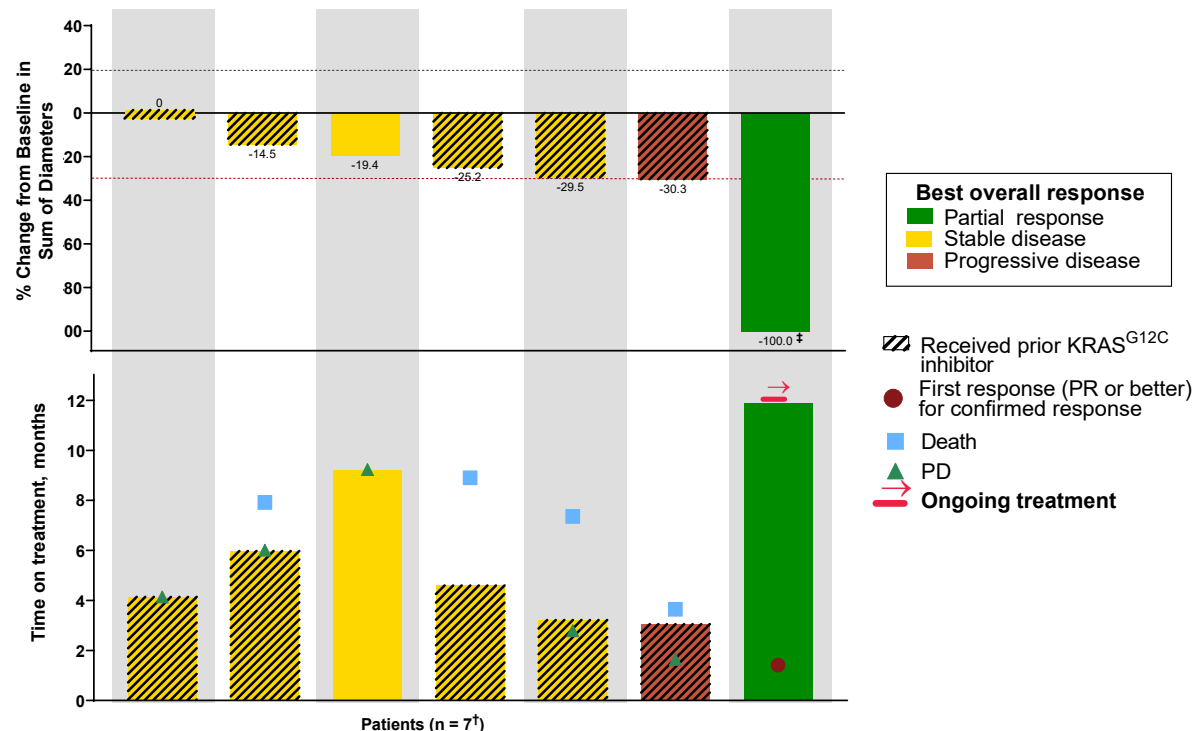
**Overall, 27% achieved response (including unconfirmed response awaiting confirmation)
and 81% achieved disease control**

*Efficacy analysis set includes all patients who received ≥ 1 dose of investigational products, have ≥ 1 measurable lesions at baseline assessed using RECIST 1.1, and have the opportunity to be followed for ≥ 7 weeks starting from day 1; †Includes patients with unconfirmed partial response, awaiting confirmatory scan
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PART 1 COHORT A:

TUMOR RESPONSE AND TREATMENT DURATION

- Part 1 Cohort A (n = 8)
 - Sotorasib 960 mg/panitumumab 6 mg/kg
 - Five patients had received prior KRAS^{G12C} inhibitor
 - Three patients were KRAS^{G12C} inhibitor naïve
- Four out of five patients who had prior KRAS^{G12C} treatment showed tumor shrinkage from 15%–30%
- Two patients who did not have prior KRAS^{G12C} treatment had 19%–100% tumor shrinkage
- The majority of the patients (80%) with prior KRAS^{G12C} inhibitor exposure had a best response of stable disease



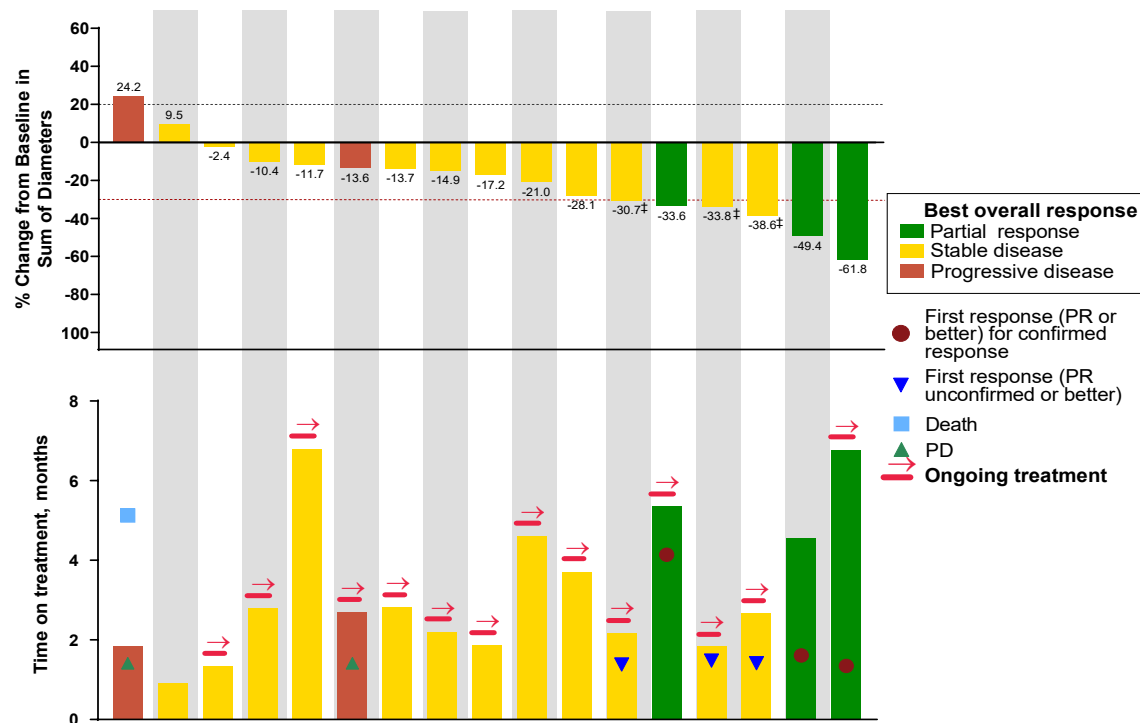
*Only considers tumor assessments prior to and including the first assessment where timepoint response is progressive disease, and prior to the start of the next anti-cancer therapy. †Includes patients who received ≥ 1 dose of investigational product, have ≥ 1 RECIST 1.1 measurable lesions at baseline, and have been followed for ≥ 7 weeks from the day one; 1 patient was not able to be included here as withdraw from study before post baseline tumor assessment. ‡This patient had 100% reduction in target lesion size, but still had non-target lesions present.

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PART 2 COHORT A:

TUMOR RESPONSE AND TREATMENT DURATION

- Part 2 Cohort A (n = 18)
 - Sotorasib 960 mg/panitumumab 6 mg/kg
 - Only includes KRAS^{G12C} inhibitor naïve patients
- Decrease in target lesion size was observed in the majority (15/17) of this chemotherapy refractory mCRC population treated in dose expansion.
- Among these 15 patients, 14 remain on treatment with two patients remaining on treatment after 6 months.
- Median time for progression free survival can not yet be estimated.



*Only considers tumor assessments prior to and including the first assessment where timepoint response is progressive disease, and prior to the start of the next anti-cancer therapy. †Includes patients who received ≥ 1 dose of investigational product, have ≥ 1 RECIST 1.1 measurable lesions at baseline, and have been followed for ≥ 7 weeks from the day one; 1 patient was not able to be included here as withdraw from study before post baseline tumor assessment. ‡This patient had 100% reduction in target lesion size, but still had non-target lesions present.

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RECENT LUMAKRAS™ DATA PRESENTATIONS

CodeBreaK101 subprotocol H: Phase 1b study evaluating the combination of sotorasib, a *KRAS*^{G12C} inhibitor, and panitumumab, an EGFR inhibitor, in advanced *KRAS* p.G12C-mutated colorectal cancer

**ESMO Abstract
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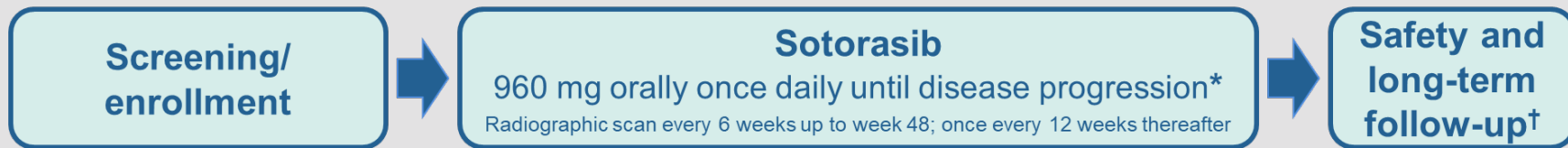
Efficacy of sotorasib in *KRAS* p.G12C-mutated NSCLC with stable brain metastases: a post-hoc analysis of CodeBreaK 100

**WCLC Abstract
P52.03**

Genomic profiles and potential determinants of response and resistance in *KRAS* p.G12C-mutated NSCLC treated with sotorasib

**WCLC Abstract
MA14.03**

EFFICACY OF SOTORASIB IN KRAS P.G12C-MUTATED NSCLC WITH STABLE BRAIN METASTASES: A POST-HOC ANALYSIS OF CODEBREAK 100



Key eligibility criteria

- Locally advanced or metastatic NSCLC
- KRAS p.G12C mutation as assessed by central testing of tumor biopsies
- Progressed on prior standard therapies‡
- **No active brain metastases, as determined by investigators§**

Primary endpoint

- Overall response rate (RECIST 1.1) by BICR

Key secondary endpoints

- Duration of response
- Disease control rate
- Time to response
- Progression-free survival
- Overall survival
- Safety

Post-hoc exploratory endpoint

- Retrospectively evaluate response to sotorasib in target and non-target stable brain metastases, where
 - **Target lesion:** measurable lesion suitable for accurate repeated measurements
 - **Non-target lesion:** lesion too small for accurate repeated measurements

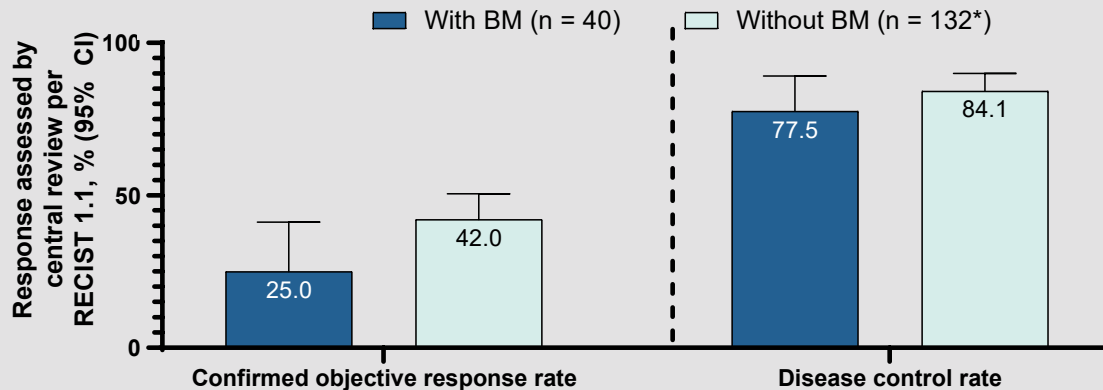
*Treatment beyond disease progression was allowed if certain criteria were met; †Safety follow-up occurred 30 (+7) days after the last dose of sotorasib; long-term follow-up occurred every 12 (±2) weeks for up to 3 years; ‡≤ 3 prior lines of therapies were allowed; §Active brain metastases from non-brain tumors were excluded. Patients who had BM resected or had received radiation therapy ending ≥ 4 weeks before study day 1 were eligible if they met the following criteria: a) residual neurological symptoms grade ≤ 2; b) on stable doses of dexamethasone, if applicable; and c) follow-up MRI performed within 30 days showed no new lesions appearing.

BICR = blinded independent central review; RECIST = Response Evaluation Criteria in Solid Tumors.

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EFFICACY OF SOTORASIB IN KRAS P.G12C-MUTATED NSCLC WITH STABLE BRAIN METASTASES (BM): A POST-HOC ANALYSIS OF CODEBREAK 100

- Per RECIST 1.1, sotorasib was associated with a disease control rate of 77.5% in patients with NSCLC and BM vs 84.1% in patients without BM (Figure)
 - Median (95% CI) PFS: 5.3 (2.7, 9.3) vs 6.7 (5.3, 8.2) months, respectively
 - Median (95% CI) OS: 8.3 (7.3, 12.5) vs 13.6 (10.0, NE), respectively



- 8/40 patients (20%) with BM and 26/134[†] (19%) without BM reported grade 3 TRAEs
- No patients with BM and 2/134 (1.5%) without BM reported grade 4 TRAEs; no fatal TRAEs occurred

*For patients with NSCLC without BM, 132 patients were included in the efficacy set. [†]For patients with NSCLC without BM, 134 patients were included in the safety set
NE = not evaluable; OS = overall survival; PFS = progression-free survival; TRAE = treatment-related adverse event.
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DESCRIPTIVE ANALYSIS USING RESPONSE ASSESSMENT IN NEURO-ONCOLOGY (RANO)

- **Per central RANO brain metastasis review, 16/174 (9.2%) patients had baseline and ≥ 1 on-treatment evaluable scans*:**
 - **Nine patients had 1 lesion; two had 4 lesions; five had ≥ 5 lesions**

Best Response by RANO, n (%)	Patients with Target and Non-target CNS Lesions Sotorasib 960 mg (n = 3)	Patients with only Non-target CNS Lesions Sotorasib 960 mg (n = 13)	All Patients with Evaluable Brain Metastases Sotorasib 960 mg (N = 16)[†]
Complete response	0	2 (15)	2 (13)
Stable disease	1 (33)	11 (85)	12 (75)
Progressive disease	2 (67)	0	2 (13)

- **Overall, intracranial disease control was achieved in 14/16 patients (88%) with evaluable brain metastases**

*40 patients were identified by investigator as having BM; 16 patients with evaluable BM were identified per central review; [†]Nine patients had 1 lesion; two had 4 lesions; five had ≥ 5 lesions; CNS = central nervous system
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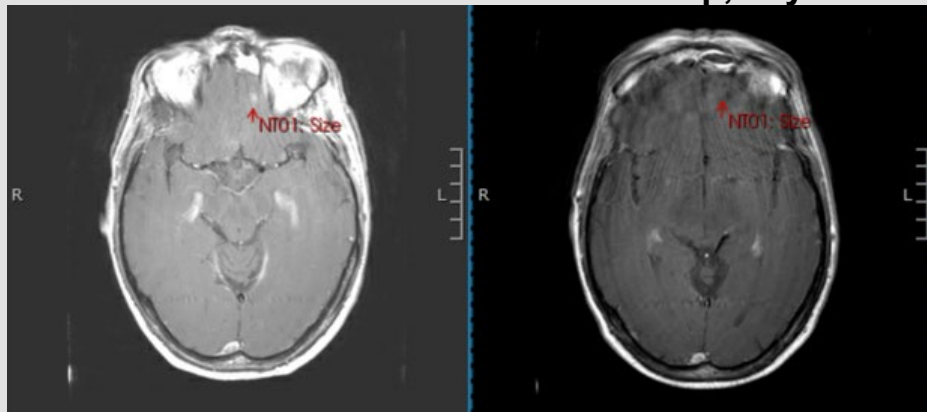
RANO BRAIN METASTASES MRI SCANS: PATIENT CASE

- 63 year old male with stage IV *KRAS* p.G12C mutant metastatic NSCLC
- Treatment history:
 - Progressed on two prior lines of therapy
 - Platinum chemotherapy, anti-PD-1/PD-L1
 - No prior radiotherapy for brain metastases

Complete Response in CNS (brain metastasis)

Baseline

Follow-up, day 53



MRI = magnetic resonance imaging

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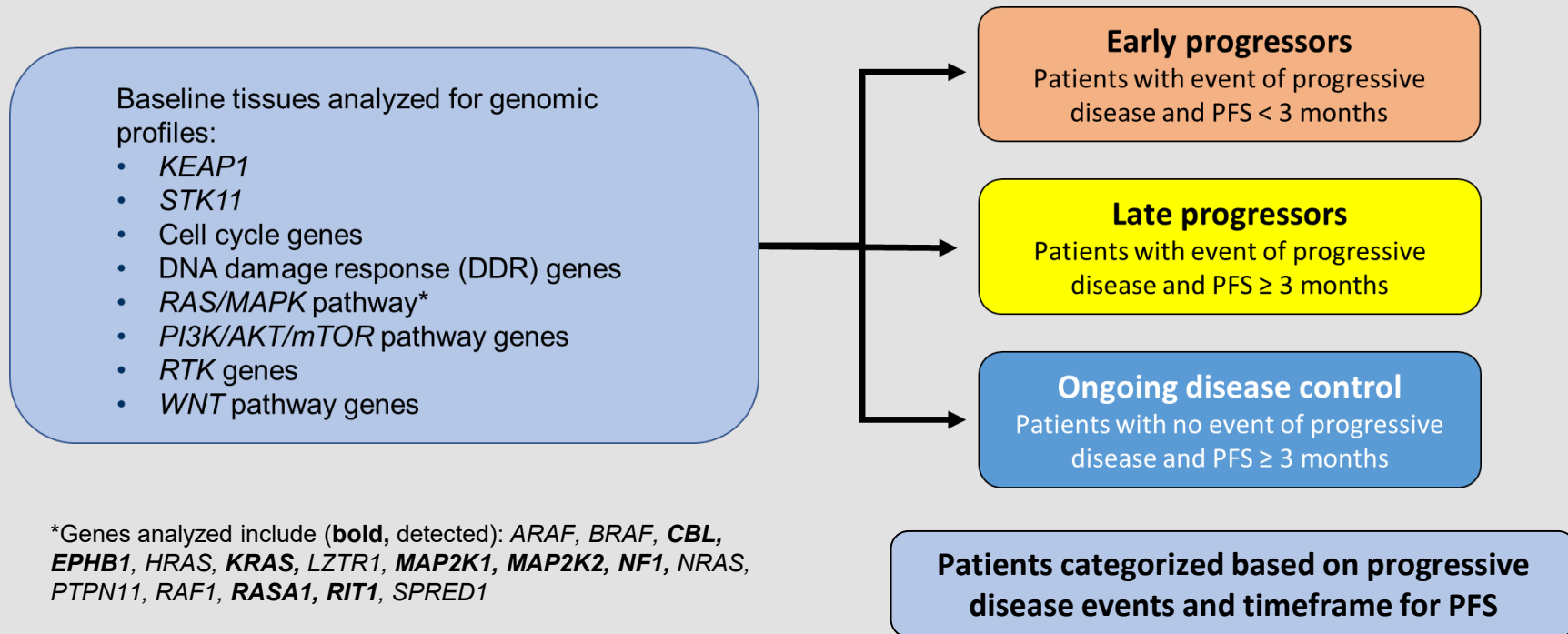
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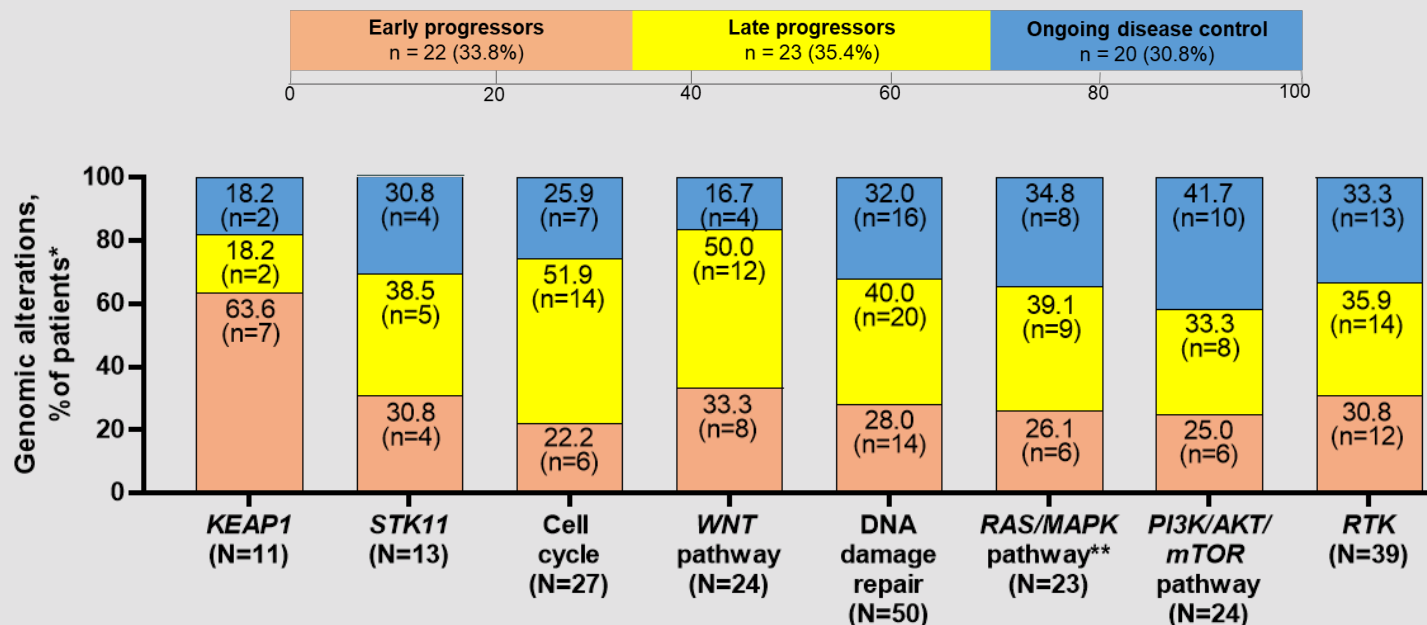
**WCLC Abstract
MA14.03**

GENOMIC PROFILES AND POTENTIAL DETERMINANTS OF RESPONSE AND RESISTANCE IN *KRAS* P.G12C-MUTATED NSCLC TREATED WITH SOTORASIB



CLINICAL RESPONSE PATTERNS VARIED IN CO-MUTATIONAL SUBGROUPS

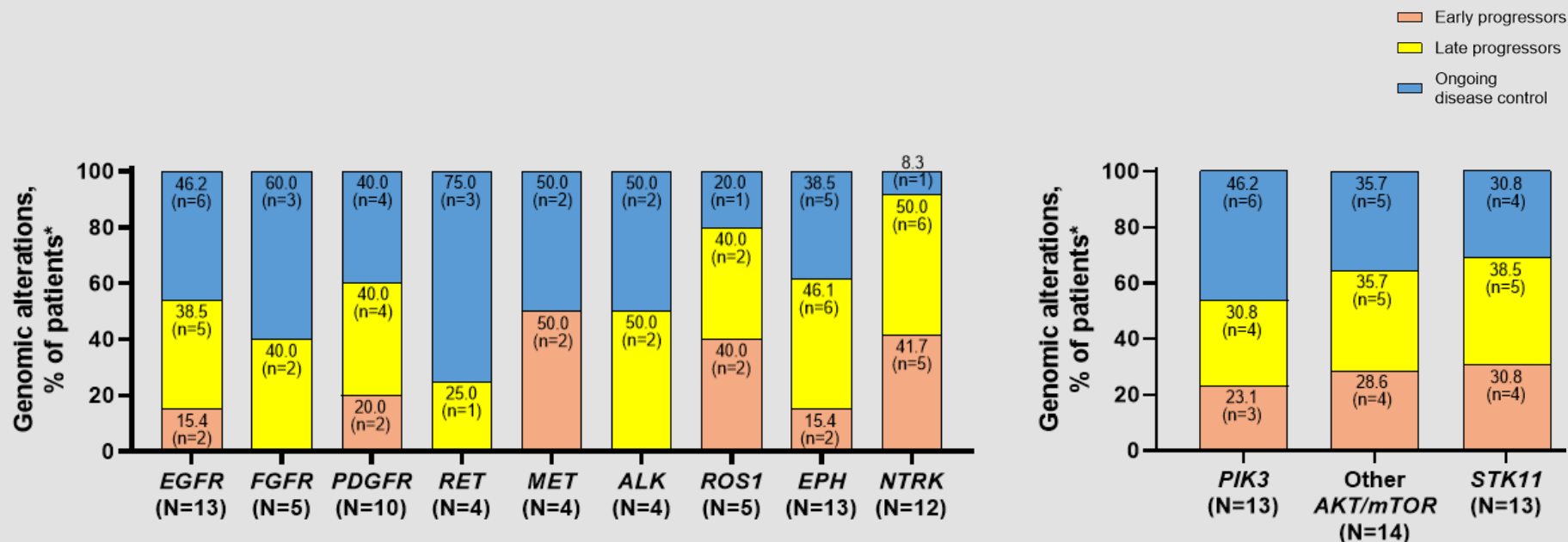
- Of the 126 patients enrolled, 65 had baseline tissue samples and at least 3 months follow-up:



*Percentage of patients with one or more relevant alterations in any pathway gene based on total number of genomic alterations detected in baseline tissue samples; **At least one genomic alteration in the RAS/MAPK pathway in addition to KRAS p.G12C; N = total patients with genomic alterations in pathway gene; n = patients in each subgroup with genomic alteration.

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CLINICAL RESPONSE PATTERNS VARIED ACROSS SUBGROUPS BASED ON SPECIFIC GENES WITH GENOMIC ALTERATIONS



*Percentage of patients with one or more relevant alterations in any pathway gene based on total number of genomic alterations detected in baseline tissue samples; N = total patients with genomic alteration; n = patients in each subgroup with genomic alteration.

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LUMAKRAS™ DATA SUMMARY

- **The combination of LUMAKRAS™ + Vectibix® was safe and tolerable in chemorefractory patients with *KRAS* p.G12C-mutated CRC**
 - Adverse events were consistent with known adverse events for LUMAKRAS™ and Vectibix®
 - 26.9% ORR; 15.4% confirmed ORR across cohorts, including patients with prior LUMAKRAS™ therapy
 - 33.3% ORR; 16.6% confirmed ORR in LUMAKRAS™ naïve patient cohort
 - Phase 3 study in patients with 3L+ CRC planned for Q4 '21
- **LUMAKRAS™ demonstrated systemic durable anticancer activity in patients with NSCLC with stable brain metastases previously treated with radiation or surgery**
 - Intracranial complete responses observed—continued intracranial stabilization in majority of patients
 - Enrolling a cohort of patients with 2L+ NSCLC with active untreated brain metastases
- **No single genetic signature predicted LUMAKRAS™ responses in patients with NSCLC**
 - A large and expanding master protocol allows for a broad based combination approach

LUMAKRAS™: LEVERAGING AMGEN'S LEADERSHIP IN ONCOLOGY TO SERVE PATIENTS


- **First and only approved KRAS^{G12C} inhibitor**
- **Unparalleled development speed—rapidly advancing the largest and most comprehensive clinical program with nearly 3,000 global patients***
- **Proven commercial success in Oncology—established and trusted relationships with customers and payers around the globe**
- **Strong ongoing commitment supporting patients with NSCLC**
- **Uniquely positioned for success in CRC with established portfolio**
 - **Pursuing triplet combinations with chemotherapy + Vectibix® or MVASI® in CodeBreak 101 master protocol**
 - **Phase 3 Vectibix® combination in 3L+ CRC planned to initiate in Q4**

*Includes clinical studies, early access programs and commercial patients
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Q&A

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SEPTEMBER 16, 2021

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