ONCOLOGY CLINICAL UPDATE
ESMO 2021

SEPTEMBER 16, 2021

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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**

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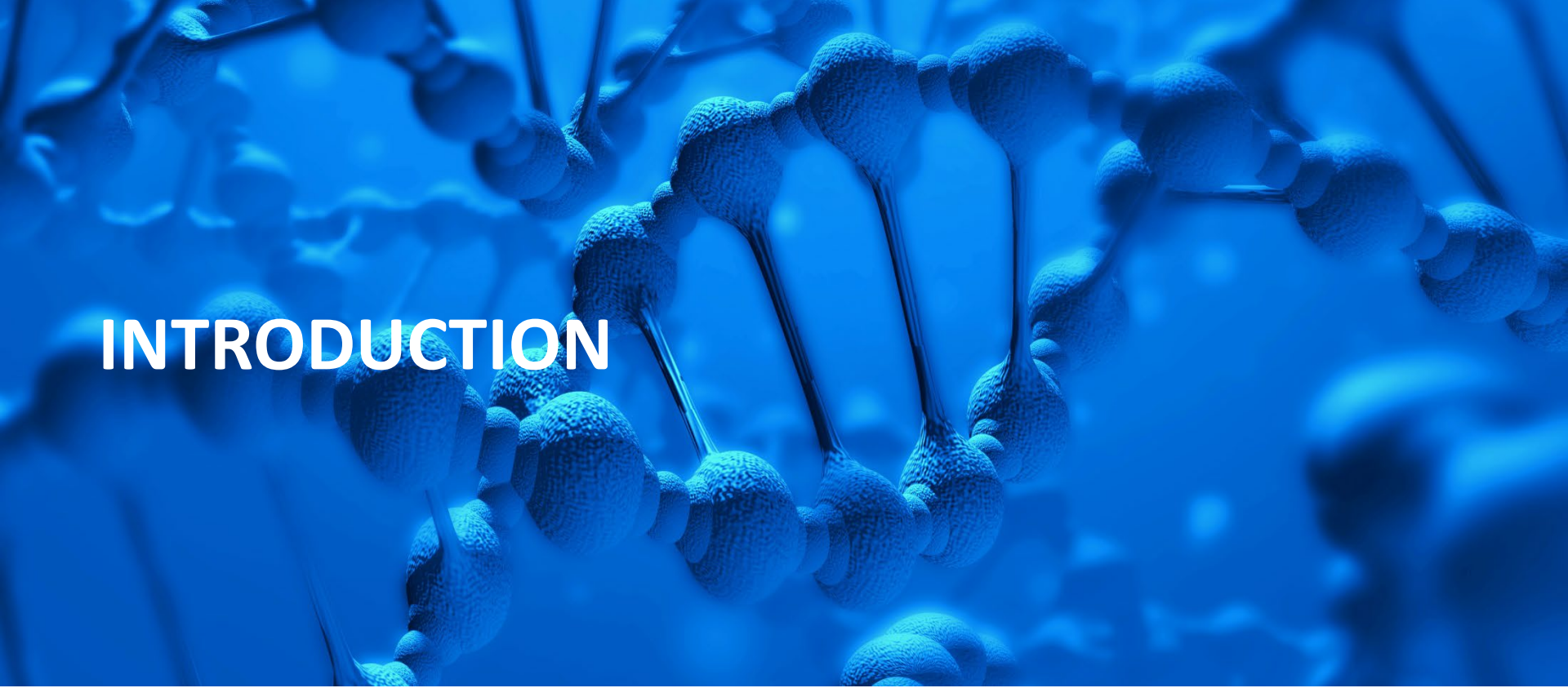

INTRODUCTION

DAVID REESE, M.D.

EXECUTIVE VICE PRESIDENT, RESEARCH AND DEVELOPMENT

AMGEN®

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**

KRAS^{G12C} = Kirsten rat sarcoma viral oncogene homolog with G12C mutation; FGFR2b = fibroblast growth factor receptor 2b; BiTE® = bispecific T-cell engager

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

Solid Tumors			
Tumor Type	**Molecule**	**Target**	**Modality**
Gastric and Gastroesophageal Junction Cancer	Bemarituzumab	FGFR2b	Monoclonal Ab
	AMG 199	MUC17	HLE-BiTE® Molecule
	AMG 910	CLDN18.2	HLE-BiTE® Molecule
Melanoma	IMLYGIC®		Oncolytic Virus
Non-Small Cell Lung Cancer	Acapatamab (AMG 160)	PSMA	HLE-BiTE® Molecule
Prostate Cancer	Acapatamab	PSMA	HLE-BiTE® Molecule
	Tarlatamab (AMG 757)	DLL3	HLE-BiTE® Molecule
	AMG 509	STEAP1	Bivalent T-cell engager XmAb® 2+1 Ab
Small Cell Lung Cancer	Tarlatamab	DLL3	HLE-BiTE® Molecule
Solid Tumors	LUMAKRAS™	$KRAS^{G12C}$	Small Molecule
	AMG 256	PD-1 / IL-21	Bifunctional Fusion Protein
	AMG 650	KIF18A	Small Molecule
	AMG 994	Undisclosed	Bifunctional Fusion Protein

Hematologic Malignancies			
Tumor Type	**Molecule**	**Target**	**Modality**
Acute Lymphoblastic Leukemia	BLINCYTO® (blinatumomab)	CD19	BiTE® Molecule
Acute Myeloid Leukemia	AMG 330	CD33	BiTE® Molecule
	AMG 427	FLT3	HLE-BiTE® Molecule
Hematologic Malignancies	AMG 176	MCL1	Small Molecule
Multiple Myeloma	Kyprolis	Proteasome	Small Molecule
	Pavurutamab (AMG 701)	BCMA	HLE-BiTE® Molecule

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

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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

***Incudes clinical studies, early access programs and commercial patients**

Provided September 16, 2021, as part of an oral presentation and is qualified by such, contains forward-looking statements, actual results may vary materially; Amgen disclaims any duty to update.

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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, leukovorin, 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

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

1L = first-line; 2L+ = second-line or greater; 3L+ = third-line or greater

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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 ≥ 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

HER2 = human epidermal growth factor receptor 2; mCRPC = metastatic castrate resistant prostate cancer

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LUMAKRAS™ (SOTORASIB) UPDATE

P.K. MORROW, M.D.

VICE PRESIDENT, GLOBAL DEVELOPMENT

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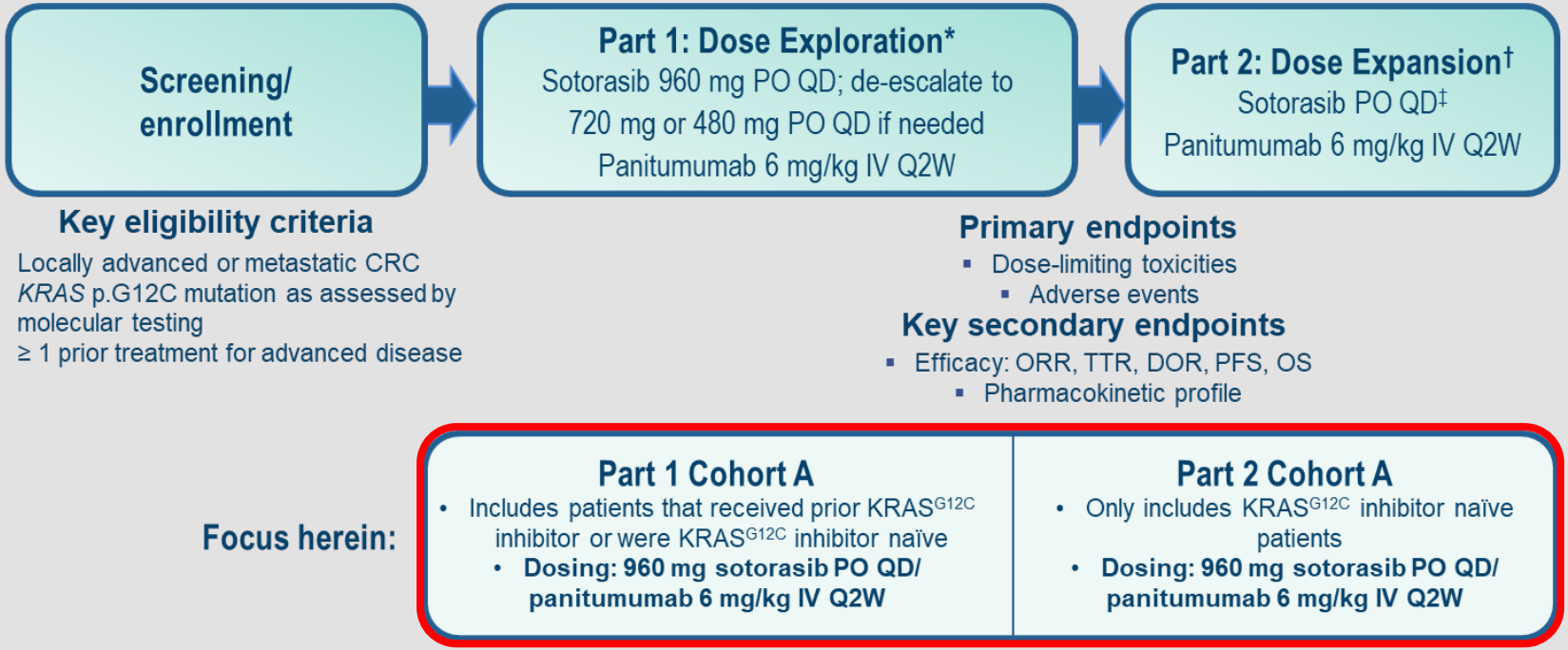
RECENT LUMAKRAS™ DATA PRESENTATIONS

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 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 (%) Related to sotorasib Related to panitumumab	23 (74.2) 14 (45.2) 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 Panitumumab	 3 (9.7)† 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 Confirmed and unconfirmed†	 12.5 (0.3, 52.7) 12.5 (0.3, 52.7)	 16.7 (3.6, 41.4) 33.3 (13.3, 59.0)	 15.4 26.9
Partial response, n (%) Confirmed Confirmed and unconfirmed†	 1 (12.5) 1 (12.5)	 3 (16.7) 6 (33.3)	 4 (15.4) 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)

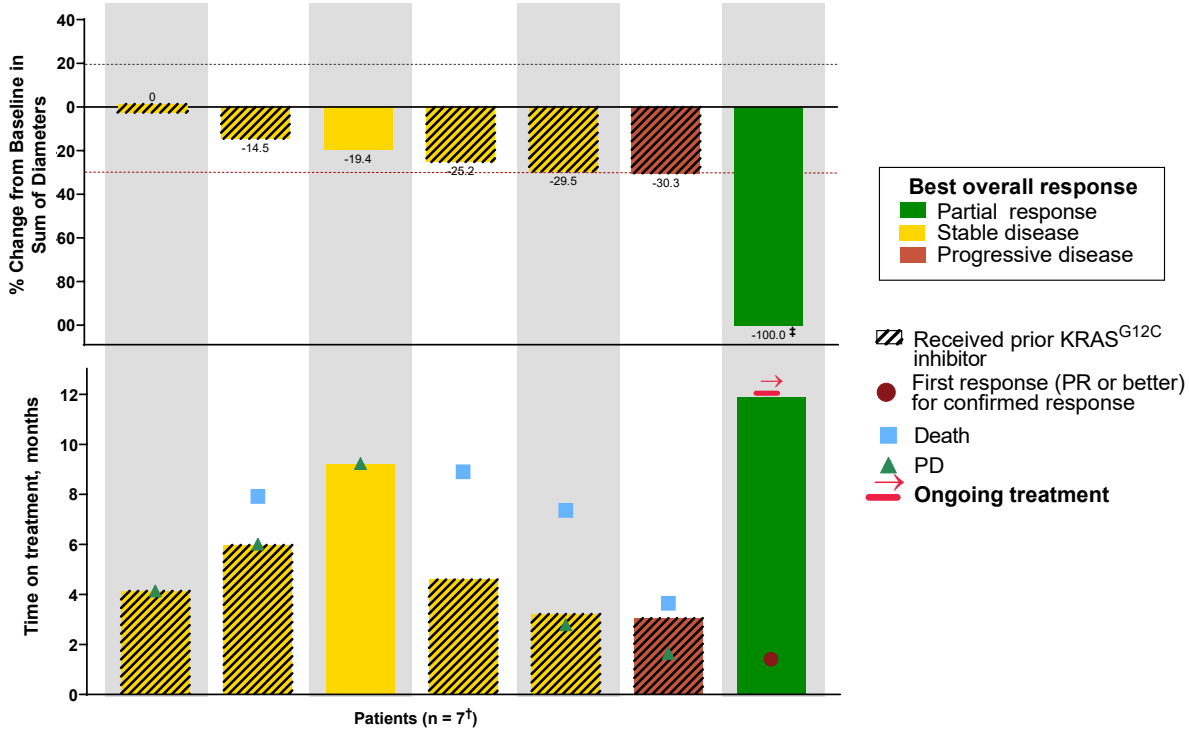
Overall, 27% achieved response (including unconfirmed response awaiting confirmation) and 81% achieved disease control

*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

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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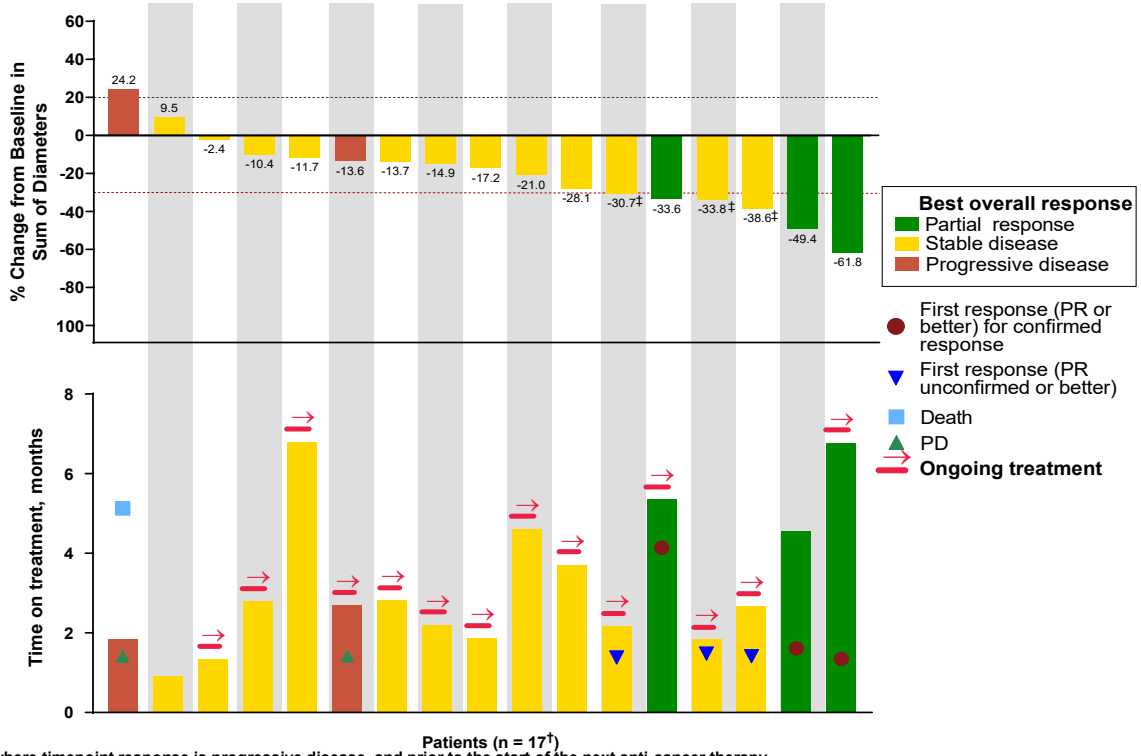
PART 2 COHORT A: TUMOR RESPONSE AND TREATMENT DURATION

- **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. ‡Patient has partial response which is not confirmed by a second scan; therefore, currently stable disease.

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RECENT LUMAKRAS™ DATA PRESENTATIONS

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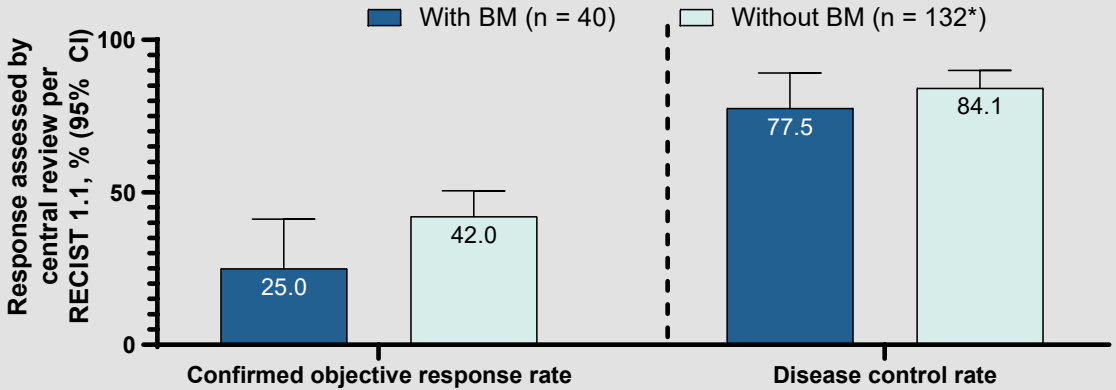
EFFICACY OF SOTORASIB IN KRAS P.G12C-MUTATED NSCLC WITH STABLE BRAIN METASTASES (BM): A POST-HOC ANALYSIS OF CODEBREAK 100

- **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**

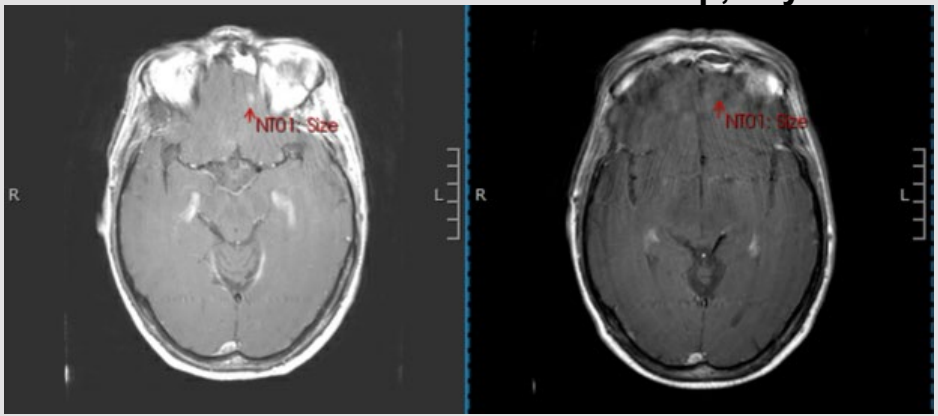
*40 patients were identified by investigator as having BM; 16 patients with evaluable BM were identified per central review; †Nine patients had 1 lesion; two had 4 lesions; five had ≥ 5 lesions; CNS = central nervous system

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 metatases

Complete Response in CNS (brain metastasis)

Baseline | **Follow-up, day 53**



MRI = magnetic resonance imaging

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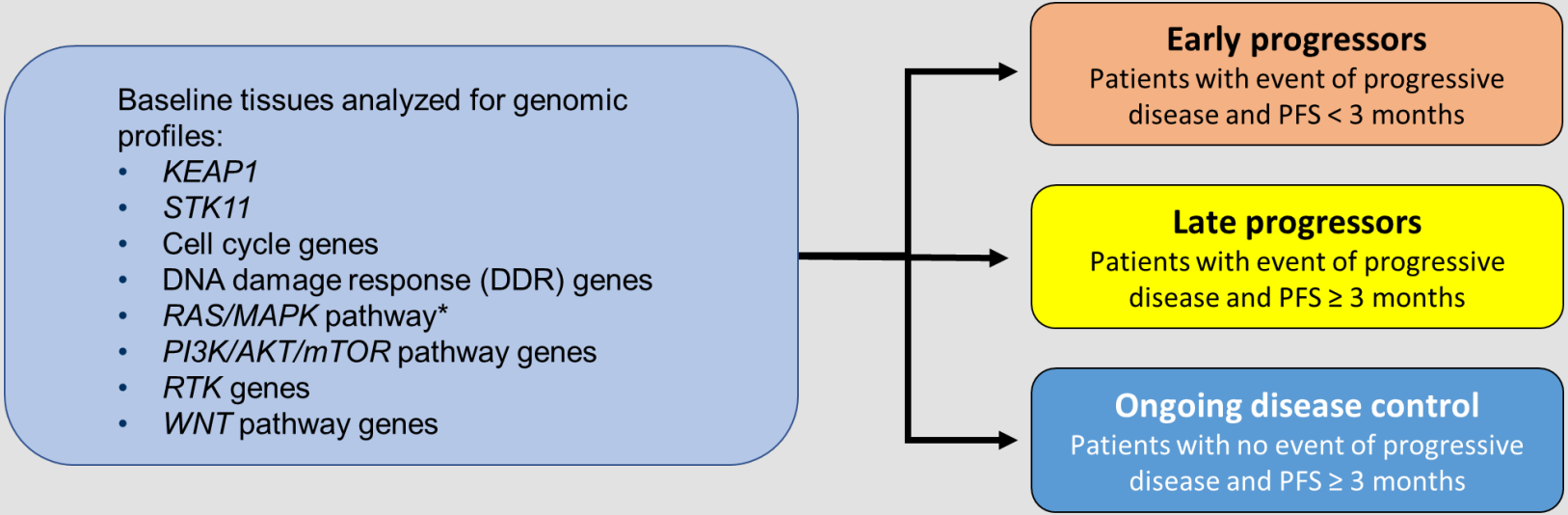
GENOMIC PROFILES AND POTENTIAL DETERMINANTS OF RESPONSE AND RESISTANCE IN *KRAS* P.G12C-MUTATED NSCLC TREATED WITH SOTORASIB



*Genes analyzed include (**bold,** detected): *ARAF, BRAF,* ***CBL, EPHB1****, HRAS,* ***KRAS,*** *LZTR1,* ***MAP2K1, MAP2K2, NF1,*** *NRAS, PTPN11, RAF1,* ***RASA1, RIT1****, SPRED1*

Patients categorized based on progressive disease events and timeframe for PFS

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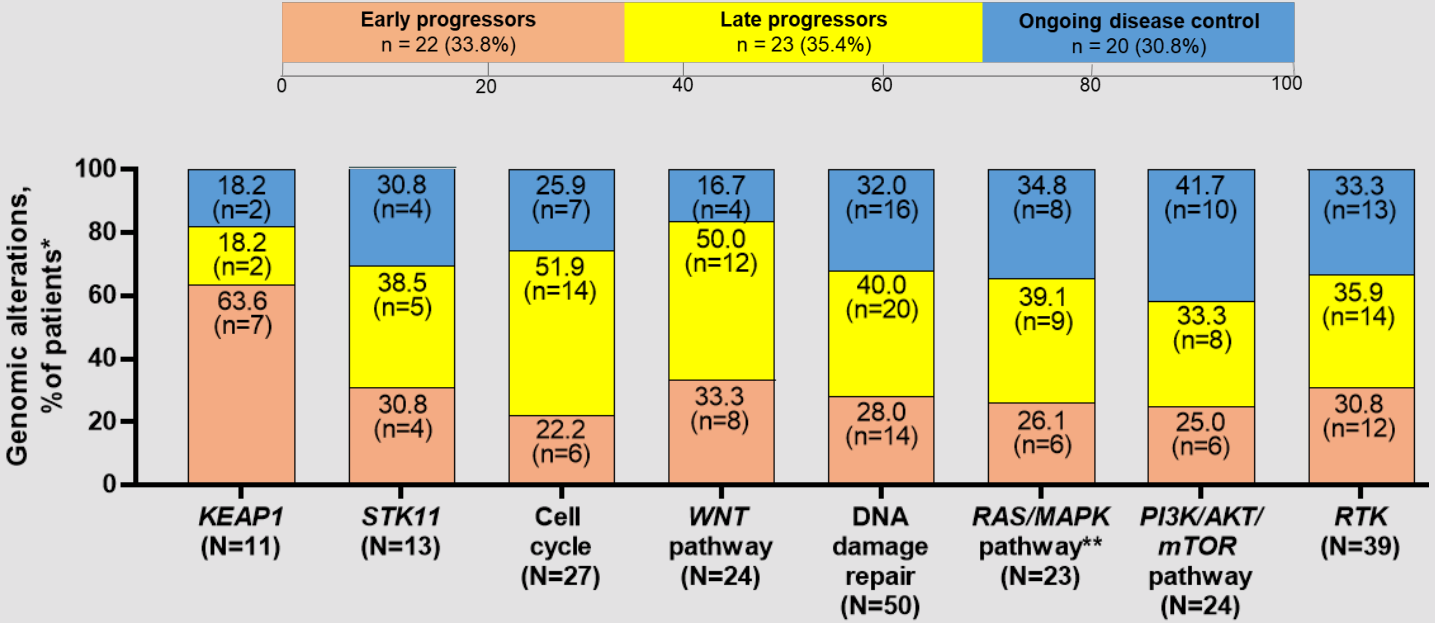
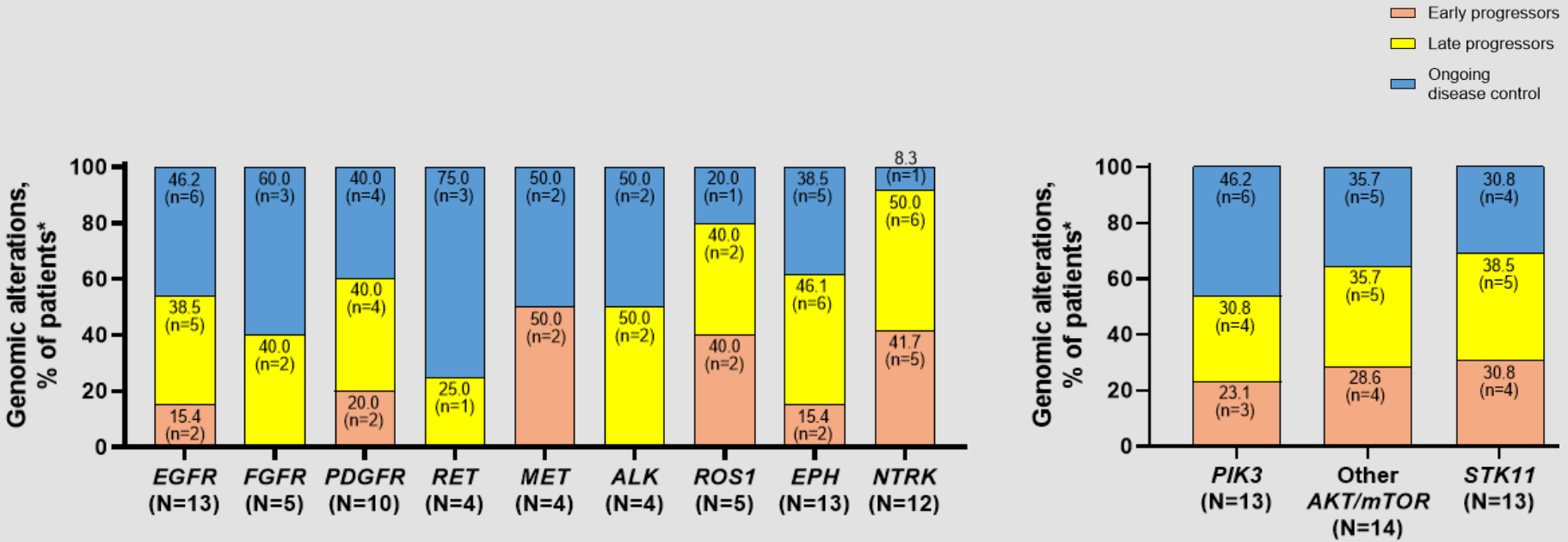
CLINICAL RESPONSE PATTERNS VARIED IN CO-MUTATIONAL SUBGROUPS

- **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.

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

*Incudes clinical studies, early access programs and commercial patients

Provided September 16, 2021, as part of an oral presentation and is qualified by such, contains forward-looking statements, actual results may vary materially; Amgen disclaims any duty to update.

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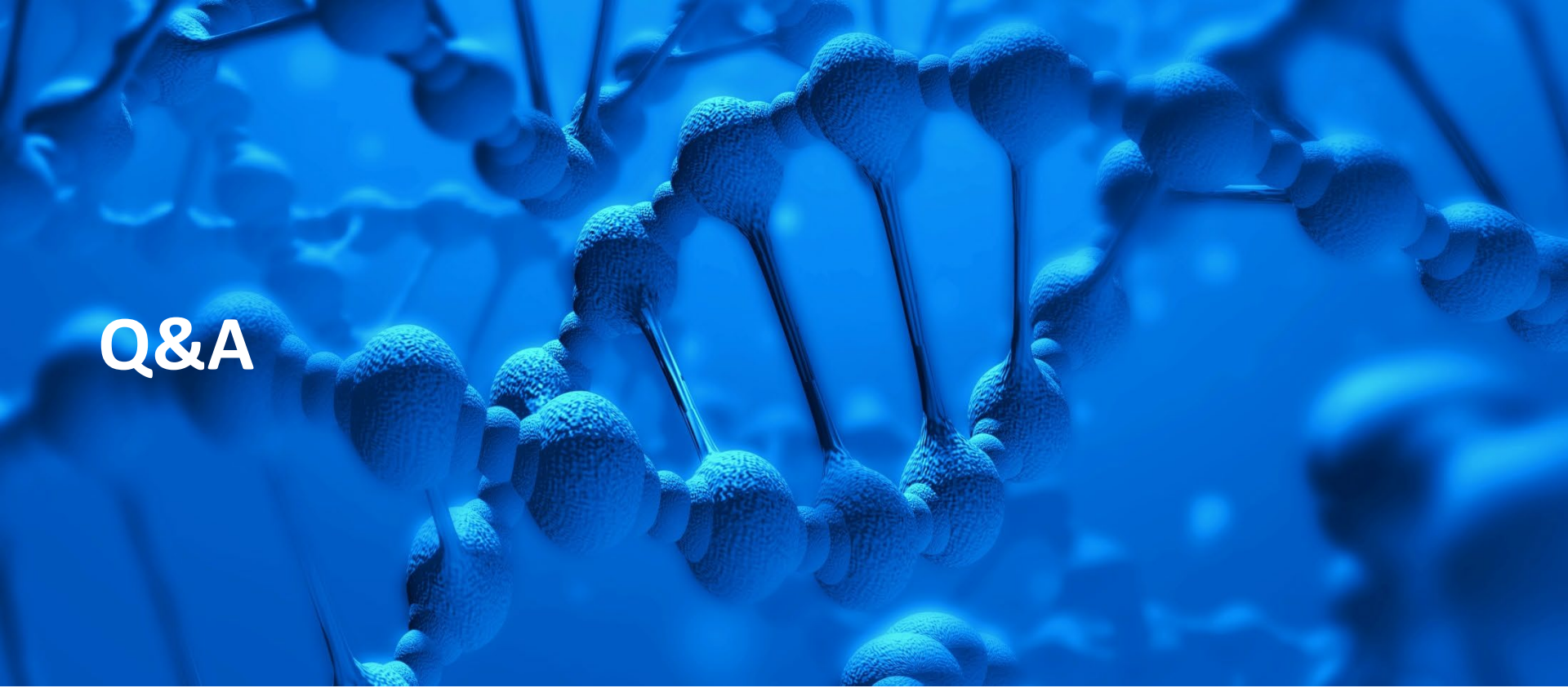
Q&A

ONCOLOGY CLINICAL UPDATE
ESMO 2021

SEPTEMBER 16, 2021

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