AMG 510 UPDATE FROM ESMO 2019

SEPTEMBER 28, 2019

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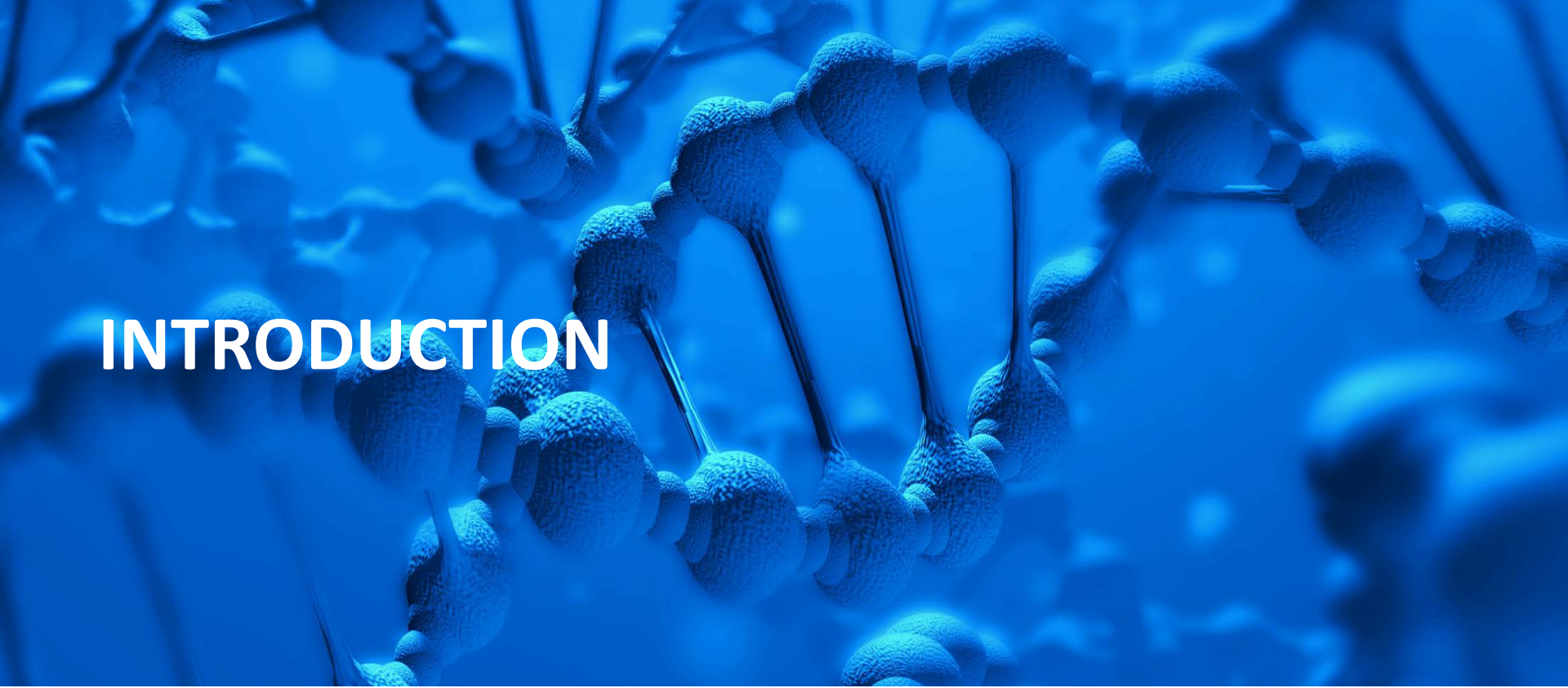
Our results may be affected by our ability to successfully market both new and existing products domestically and internationally, clinical and regulatory developments involving current and future products, sales growth of recently launched products, competition from other products including biosimilars, difficulties or delays in manufacturing our products and global economic conditions. In addition, sales of our products are affected by pricing pressure, political and public scrutiny and reimbursement policies imposed by third-party payers, including governments, private insurance plans and managed care providers and may be affected by regulatory, clinical and guideline developments and domestic and international trends toward managed care and healthcare cost containment. Furthermore, our research, testing, pricing, marketing and other operations are subject to extensive regulation by domestic and foreign government regulatory authorities. Our business may be impacted by government investigations, litigation and product liability claims. In addition, our business may be impacted by the adoption of new tax legislation or exposure to additional tax liabilities. If we fail to meet the compliance obligations in the corporate integrity agreement between us and the U.S. government, we could become subject to significant sanctions. Further, while we routinely obtain patents for our products and technology, the protection offered by our patents and patent applications may be challenged, invalidated or circumvented by our competitors, or we may fail to prevail in present and future intellectual property litigation. We perform a substantial amount of our commercial manufacturing activities at a few key facilities, including in Puerto Rico, and also depend on third parties for a portion of our manufacturing activities, and limits on supply may constrain sales of certain of our current products and product candidate development. We rely on collaborations with third parties for the development of some of our product candidates and for the commercialization and sales of some of our commercial products. In addition, we compete with other companies with respect to many of our marketed products as well as for the discovery and development of new products. Further, some raw materials, medical devices and component parts for our products are supplied by sole third-party suppliers. Certain of our distributors, customers and payers have substantial purchasing leverage in their dealings with us. The discovery of significant problems with a product similar to one of our products that implicate an entire class of products could have a material adverse effect on sales of the affected products and on our business and results of operations. Our efforts to acquire other companies or products and to integrate the operations of companies we have acquired may not be successful. A breakdown, cyberattack or information security breach could compromise the confidentiality, integrity and availability of our systems and our data. Our stock price is volatile and may be affected by a number of events. Our business performance could affect or limit the ability of our Board of Directors to declare a dividend or our ability to pay a dividend or repurchase our common stock. We may not be able to access the capital and credit markets on terms that are favorable to us, or at all.

The scientific information discussed in this presentation related to our product candidates is preliminary and investigative. Such product candidates are not approved by the U.S. Food and Drug Administration, and no conclusions can or should be drawn regarding the safety or effectiveness of the product candidates. Further, the scientific information discussed in this presentation relating to new indications for our products is preliminary and investigative and is not part of the labeling approved by the U.S. Food and Drug Administration for the products. The products are not approved for the investigational use(s) discussed in this presentation, and no conclusions can or should be drawn regarding the safety or effectiveness of the products for these uses.

AGENDA

Introduction	**David Reese, M.D.—Executive Vice President, Research and Development**
AMG 510 Update	**Gregory Friberg, M.D.—Vice President, Global Development and Oncology Therapeutic Area Head**
Q&A	**All**



INTRODUCTION

DAVID REESE, M.D.

EXECUTIVE VICE PRESIDENT, RESEARCH AND DEVELOPMENT

AMGEN®

AMGEN ONCOLOGY: A BROAD, DIFFERENTIATED PORTFOLIO

- **Built on differentiated, first-in-class molecules with high potential**
- **Developing combination/sequential therapies against multiple targets in several indications to drive deep, durable responses**
- **Clinical data over next 12 months will provide key insights**
- **Programs with compelling efficacy may rapidly advance toward registration**
- **Upcoming data presentations from multiple oncology programs**
  - **AMG 596 (EGFRvIII BiTE® molecule) and AMG 673 (CD33 HLE-BiTE® molecule) submitted for presentation in 2019**
  - **AMG 701 (BCMA HLE-BiTE® molecule) data expected in 2020**

EGFRvIII = epidermal growth factor receptor variant III; BiTE® = bispecific T-cell engager; HLE = half-life extended; BCMA = B-cell maturation antigen

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AMG 510: A FIRST-IN-CLASS KRAS^{G12C} INHIBITOR ADVANCING RAPIDLY THROUGH CLINICAL DEVELOPMENT

- **Entered Phase 2 only 12 months after first patient dosed**
- **Responses seen in multiple tumor types in heavily pretreated patients**
- **Exploring combinations to unlock the additional potential of AMG 510**
  - **Anti-PD-1 combination underway in NSCLC, CRC study beginning in Q4**
  - **MEK inhibitor combination study in solid tumors beginning in Q4**
- **Next clinical update will be Phase 2 monotherapy data and Phase 1 combination data in 2020**

KRAS **= Kirsten rat sarcoma viral oncogene homolog; PD-1 = programmed cell death protein 1; NSCLC = non-small-cell lung cancer; CRC = colorectal cancer; MEK = mitogen-activated protein kinase kinase**

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AMG 510 UPDATE

GREGORY FRIBERG, M.D.

VICE PRESIDENT, GLOBAL DEVELOPMENT AND ONCOLOGY
THERAPEUTIC AREA HEAD

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PHASE 1 STUDY OF AMG 510, A NOVEL KRAS^{G12C} INHIBITOR, IN ADVANCED SOLID TUMORS WITH *KRAS P. G12C* MUTATION

Ramaswamy Govindan, MD;[1] **Marwan G Fakih, MD**;[2] Timothy J Price, MBBS, DHlthSci, FRACP;[3] Gerald S Falchook, MD;[4] Jayesh Desai, MBBS, FRACP;[5] James C Kuo, MBBS, FRACP;[6] John H Strickler, MD;[7] John C Krauss, MD;[8] Bob T Li, MD;[9] Crystal S Denlinger, MD;[10] Greg Durm, MD;[11] Jude Ngang, PharmD;[12] Haby Henary, MD;[12] Gataree Ngarmchamnanrith, MD;[12] June Kim, PhD;[12] Phuong Khanh Morrow, MD;[12] David S Hong, MD[13]

[1]Alvin J Siteman Cancer Center at Washington University School of Medicine, St Louis, MO, USA; [2]City of Hope, Duarte, CA, USA; [3]The Queen Elizabeth Hospital, Woodville South, AU; [4]Sarah Cannon Research Institute at HealthONE, Denver, CO, USA; [5]Peter MacCallum Cancer Centre, Melbourne, AU; [6]Scientia Clinical Research, Randwick, AU; [7]Duke University Medical Center, Durham, NC, USA; [8]University of Michigan, Ann Arbor, MI, USA; [9]Memorial Sloan Kettering Cancer Center, New York, NY, USA; [10]Fox Chase Cancer Center, Philadelphia, PA, USA; [11]Indiana University, Simon Cancer Center, Indianapolis, IN, USA; [12]Amgen Inc, Thousand Oaks, CA, USA; [13]MD Anderson Cancer Center, Houston, TX, USA

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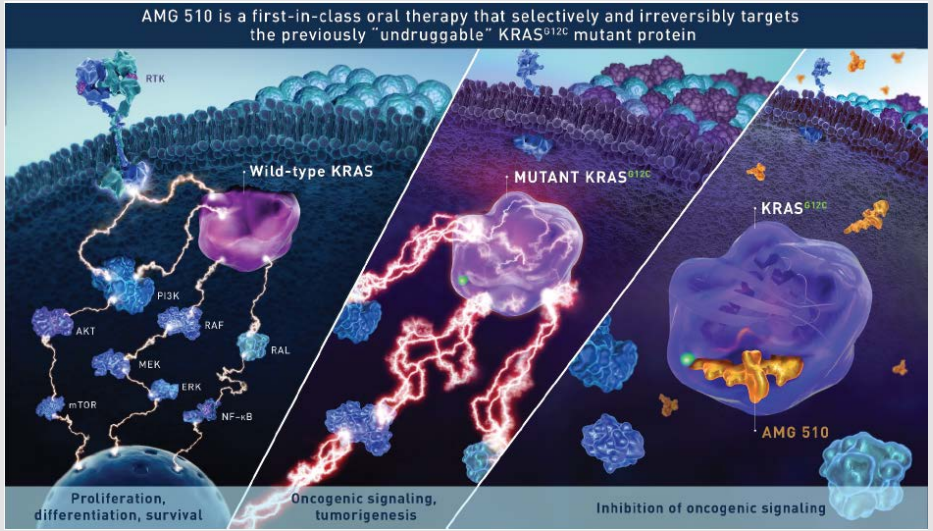
AMG 510 IS A FIRST-IN-CLASS KRAS^{G12C} INHIBITOR

- ***KRAS p. G12C*** **mutation is found in approximately 13% of lung cancer, 3% of colorectal cancer and appendix cancer, and 1%–3% of other solid tumors[1-3]**
- **Currently, there is no approved therapy targeting this mutation**
- **AMG 510 is a novel, first-in-class small molecule that specifically and irreversibly inhibits KRAS^{G12C} by permanently locking it in an inactive GDP-bound state**



GDP = guanosine diphosphate; *KRAS* = Kirsten rat sarcoma viral oncogene homolog; KRAS^{G12C} = KRAS protein with a G12C mutation at the protein level

1. Biernacka A, et al. *Cancer Genet.* 2016;209:195-198. 2. Neumann J, et al. *Pathol Res Pract.* 2009;205:858-862. 3. Zhou L, et al. *Med Oncol.* 2016;33:32.

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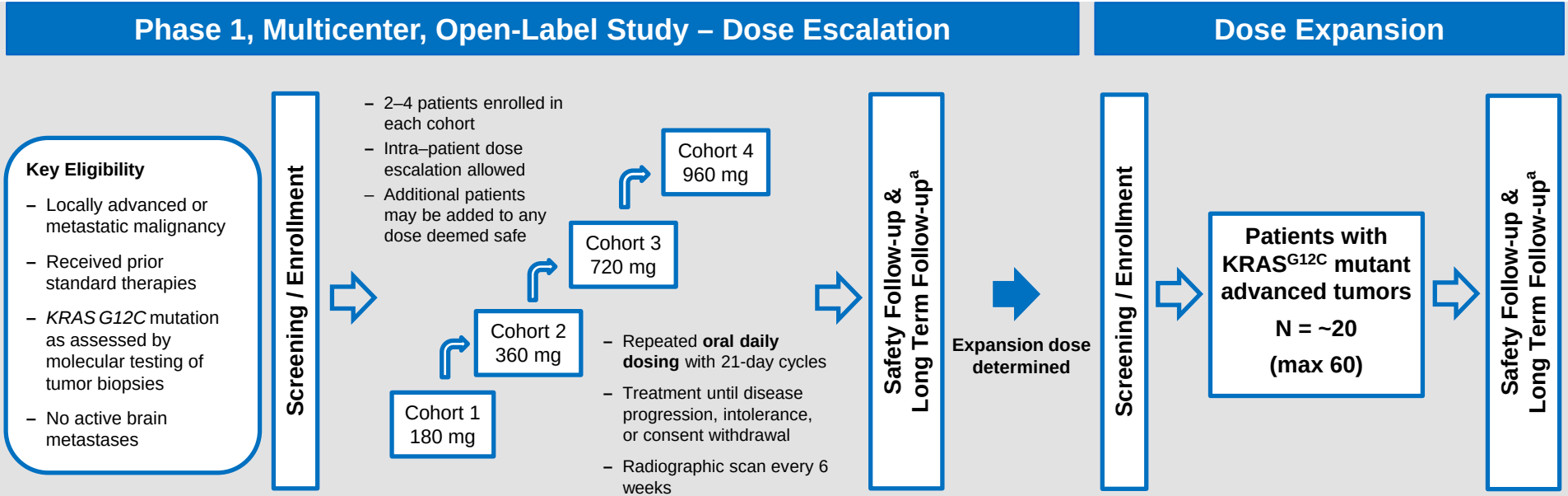
AMG 510 FIRST-IN-HUMAN STUDY DESIGN



Primary endpoints: dose-limiting toxicities (DLTs); safety
Key secondary endpoints: PK; objective response rate; duration of response; disease control rate; PFS; duration of stable disease

[a]30 (+7) days after end of treatment for safety follow-up; every 12 weeks for long-term follow-up
KRAS = Kirsten rat sarcoma viral oncogene homolog; KRAS^{G12C} = KRAS protein with a G12C mutation at the protein level; PFS = progression-free survival; PK = pharmacokinetics

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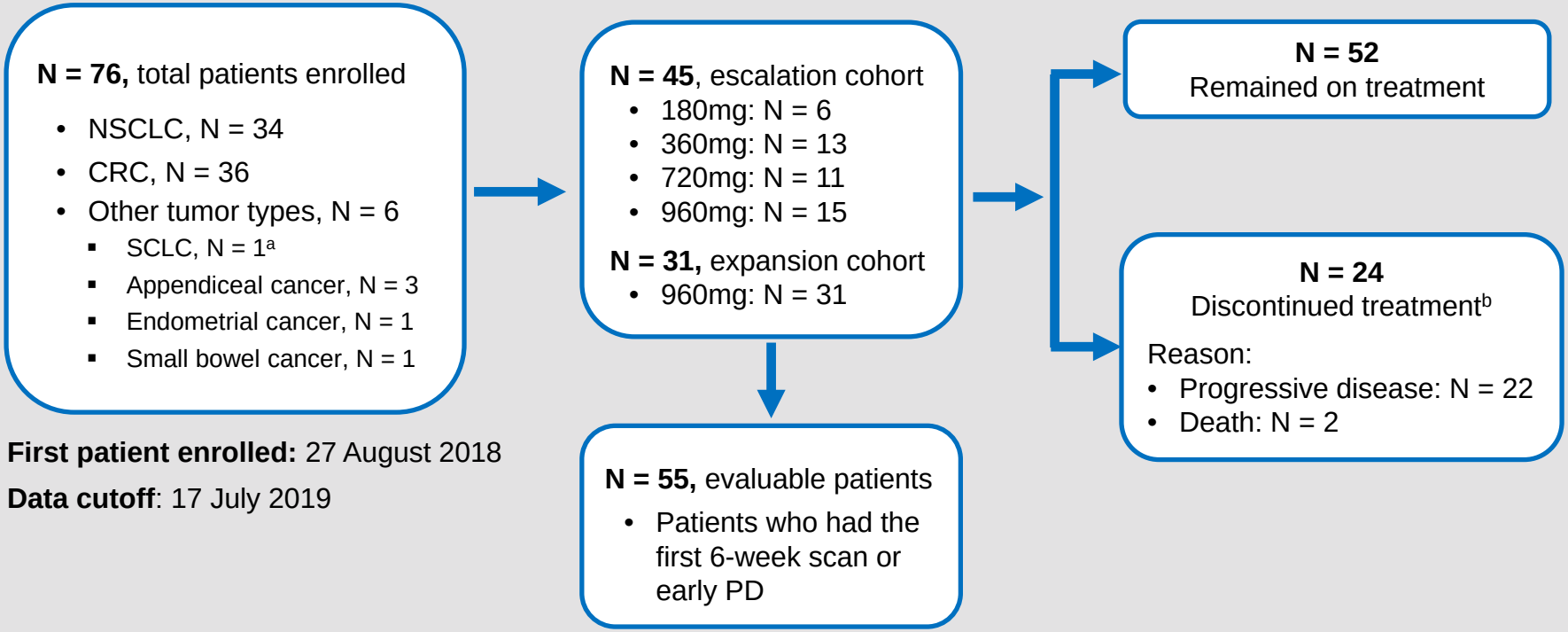
PATIENT DISPOSITION



First patient enrolled: 27 August 2018

Data cutoff: 17 July 2019

[a]The tumor type of this patient was recorded as SCLC ("other tumor type" category) as of the data cutoff and changed to NSCLC by the participating site after cutoff.
[b]None of the discontinuations was caused by treatment-related adverse events. NSCLC = non-small cell lung cancer; CRC = colorectal cancer; SCLC = small cell lung cancer; PD = progressive disease

BASELINE CHARACTERISTICS

Baseline Characteristics	N = 76
Median age (range) – year	59.0 (33.0–78.0)
Female – n (%)	40 (52.6)
Primary tumor type – n (%) NSCLC CRC SCLC[a] Appendiceal cancer Endometrial cancer Small bowel cancer	 34 (44.7) 36 (47.4) 1 (1.3) 3 (3.9) 1 (1.3) 1 (1.3)
ECOG performance at baseline – n (%) 0 1 2	 20 (26.3) 53 (69.7) 3 (3.9)
Prior lines of systemic anticancer therapy – n (%) 1 2 > 2	 5 (6.6) 9 (11.8) 62 (81.6)
Number of prior systemic anticancer therapy – median (range)	4.0 (1–10)

[a]The tumor type of this patient was recorded as SCLC (other tumor types) by the data cutoff; the participating site updated the tumor type to NSCLC after cutoff.
NSCLC = non-small-cell lung cancer; CRC = colorectal cancer; SCLC = small-cell lung cancer; ECOG = Eastern Cooperative Oncology Group

PATIENT INCIDENCE OF ADVERSE EVENTS (AES): SUMMARY

	All AEs N = 76 n (%)	All treatment-related AEs N = 76 n (%)
Any grade Grade ≥ 2 Grade ≥ 3 Grade ≥ 4	57 (75.0) 44 (57.9) 24 (31.6) 8 (10.5)	**26 (34.2)** **14 (18.4)** **6 (7.9)** **0 (0.0)**
Dose limiting toxicity	0 (0)	**0 (0)**
Serious adverse events	17 (22.4)	**0 (0)**[c]
Fatal adverse events	7 (9.2)[a]	**0 (0)**
AEs leading to treatment discontinuation	2 (2.6)[b]	**0 (0)**

- **No dose-limiting toxicities were reported**
- **No treatment-related serious or fatal AEs were reported**
- **There were no treatment-related AEs leading to treatment discontinuation**

960 mg oral daily dose was identified as the expansion dose and recommended Phase 2 dose

[a]Seven patients had the following fatal AEs: dyspnea, aspiration, lung cancer metastatic, colorectal cancer metastatic, and spinal compression fracture; none was related to treatment. [b]Two patients with CRC discontinued treatment due to AE of colorectal cancer metastatic. [c]One NSCLC patient had respiratory infection, which was initially reported as a treatment-related serious AE in the snapshot; after snapshot, the study site confirmed that it was not attributed to treatment but the underlying disease. CRC = colorectal cancer; NSCLC = non-small-cell lung cancer

PATIENT INCIDENCE OF TREATMENT-RELATED ADVERSE EVENTS (AES)

All Treatment-Related AEs	Any Grade N = 76 n (%)	Grade 3 N = 76 n (%)
Any treatment-related AEs	26 (34.2)	6 (7.9)
Diarrhea	11 (14.5)	4 (5.3)
Nausea	3 (3.9)	0 (0)
Dry mouth	2 (2.6)	0 (0)
Abdominal pain	1 (1.3)	0 (0)
Cheilitis	1 (1.3)	0 (0)
Eructation	1 (1.3)	0 (0)
Flatulence	1 (1.3)	0 (0)
Vomiting	1 (1.3)	0 (0)
ALT increased	2 (2.6)	0 (0)
AST increased	2 (2.6)	0 (0)
Blood alkaline phosphate increased	2 (2.6)	0 (0)
Blood creatine phosphokinase increased	2 (2.6)	0 (0)
Alanine aminotransferase	1 (1.3)	0 (0)
Aspartate aminotransferase	1 (1.3)	0 (0)

All Treatment-Related AEs	Any Grade N = 76 n (%)	Grade 3 N = 76 n (%)
Lymphocyte count decreased	1 (1.3)	0 (0)
White blood cell count decreased	1 (1.3)	0 (0)
Anemia	3 (3.9)	3 (3.9)
Leukopenia	1 (1.3)	0 (0)
Decreased appetite	2 (2.6)	0 (0)
Hyperkalemia	1 (1.3)	0 (0)
Hypokalemia	1 (1.3)	0 (0)
Fatigue	2 (2.6)	0 (0)
Dysgeusia	1 (1.3)	0 (0)
Neuropathy peripheral	1 (1.3)	0 (0)
Arthralgia	1 (1.3)	0 (0)
Proteinuria	1 (1.3)	0 (0)
Epistaxis	1 (1.3)	0 (0)
Rash	1 (1.3)	0 (0)
Hot flush	1 (1.3)	0 (0)

- **26 of 76 patients (34.2%) reported treatment-related AEs; most were grade 1 or 2**
- **6 of 76 patients (7.9%) reported 1 or more grade 3 treatment-related adverse events: diarrhea and anemia**
- **There were no grade 4 or higher treatment-related adverse events**

ALT = alanine aminotransferase; AST = aspartate aminotransferase

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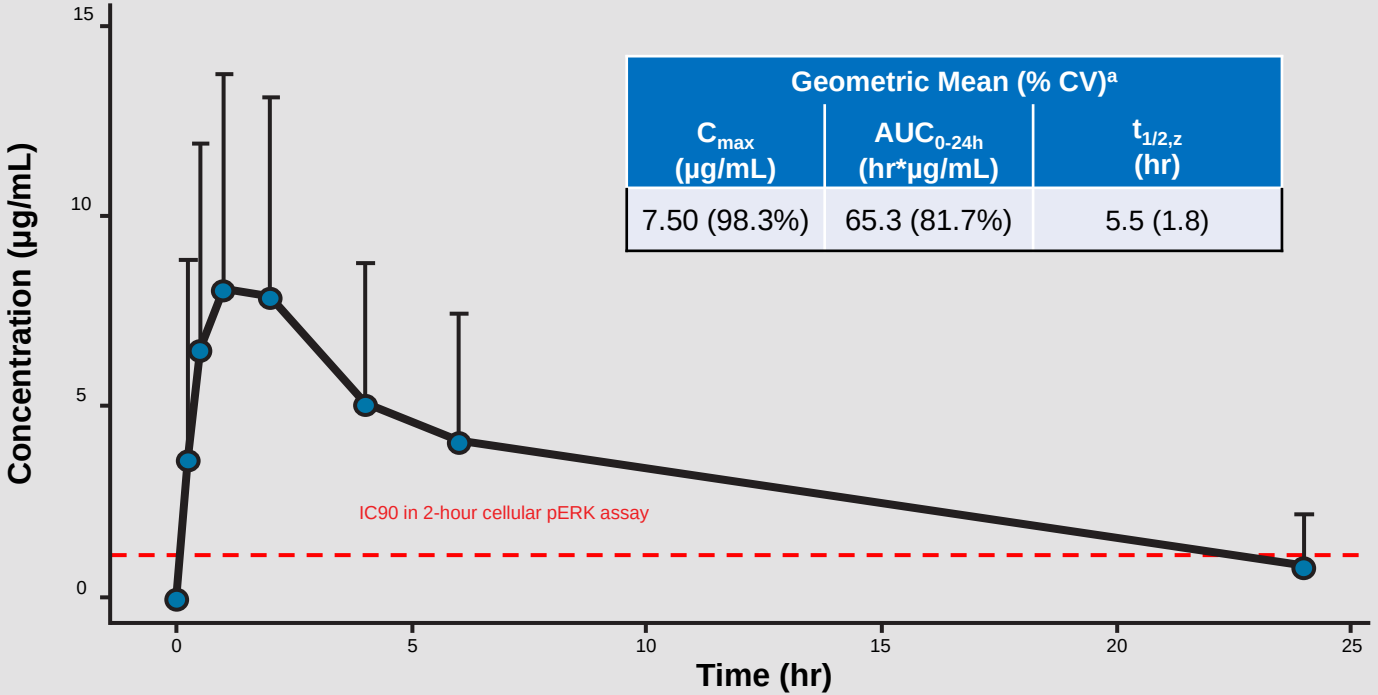
AMG 510 PK PROFILE – 960 MG ORAL DAILY DOSE



Geometric Mean (% CV)[a]		
C_{max} (µg/mL)	AUC_{0-24h} (hr*µg/mL)	$t_{1/2,z}$ (hr)
7.50 (98.3%)	65.3 (81.7%)	5.5 (1.8)

[a]PK data cutoff: 24 July 2019; N = 32 including patients with NSCLC and CRC; $t_{1/2}$ is presented as mean standard deviation. Only the top error bars are shown for clarity.
PK = pharmacokinetics; CV = coefficient of variation; C_{max} = maximum serum concentration; AUC = area under the curve; $t_{1/2}$ = elimination half life
IC_{90} = 90% inhibitory concentration in vitro; pERK = phosphorylated extracellular signal-regulated kinase

BEST TUMOR RESPONSE WITH ALL DOSE LEVELS, ALL TUMOR TYPES

Efficacy outcomes	NSCLC, evaluable patients N = 23	CRC, evaluable patients N = 29	Other tumor types, evaluable patients N = 3
Best overall response			
PR – n (%)	11 (48)	1 (3)	1 (33)[c]
SD – n (%)	11 (48)	22 (76)	1 (33)[d]
PD – n (%)	1 (4)	6 (21)	1 (33)[e]
Objective response rate[a]	**48%**	**3%**	**N/A**
Disease control rate[b]	**96%**	**79%**	**N/A**

[a]Evaluation of response is based on modified RECIST 1.1 criteria. [b]PR or SD at week 6. [c]Appendiceal cancer. [d]Appendiceal cancer. [e]The tumor type of this patient was recorded as small-cell lung cancer (other tumor types) by the data cutoff, and the participating site updated the tumor type to NSCLC after cutoff. Evaluable patients = patients who had the first 6-week scan or early PD; NSCLC = non-small-cell lung cancer; CRC = colorectal cancer; PR = partial response; SD = stable disease; PD = progressive disease

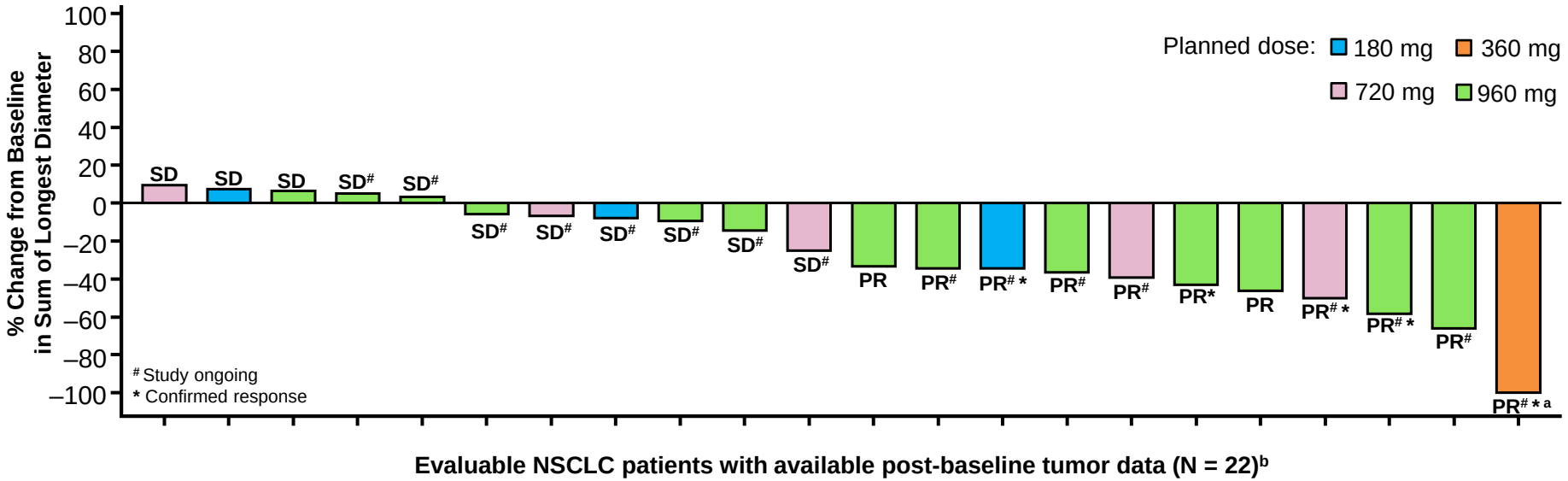
BEST TUMOR RESPONSE WITH 960 MG DOSE, ALL TUMOR TYPES

Efficacy outcomes	NSCLC, evaluable patients receiving 960mg N = 13	CRC, evaluable patients receiving 960mg N = 12	Other tumor types, evaluable patients receiving 960mg N = 1
Best overall response			
PR – n (%)	7 (54)	1 (8)	0 (0)
SD – n (%)	6 (46)	10 (83)	0 (0)
PD – n (%)	0 (0)	1 (8)	1 (100)[c]
Objective response rate[a]	**54%**	**8%**	**N/A**
Disease control rate[b]	**100%**	**92%**	**N/A**

[a]Evaluation of response is based on modified RECIST 1.1 criteria. [b]PR or SD at week 6. [c]The tumor type of this patient was recorded as small cell lung cancer ("other tumor types" category) by the data cutoff, and the participating site updated the tumor type to NSCLC after cutoff. Evaluable patients = patients who had the first 6-week scan or early PD; NSCLC = non-small-cell lung cancer; CRC = colorectal cancer; PR = partial response; SD = stable disease; PD = progressive disease

EFFICACY IN NSCLC

Change in Tumor Burden From Baseline



[a]Patient had complete response to the target lesion; [b]1 patient discontinued study due to PD prior to the 1st assessment without available post-baseline tumor burden data, and therefore is not shown on the graph.
Evaluable patients: patients who have been followed up for at least 6 weeks; NSCLC = non-small-cell lung cancer; PD = progressive disease; PR = partial response; SD = stable disease

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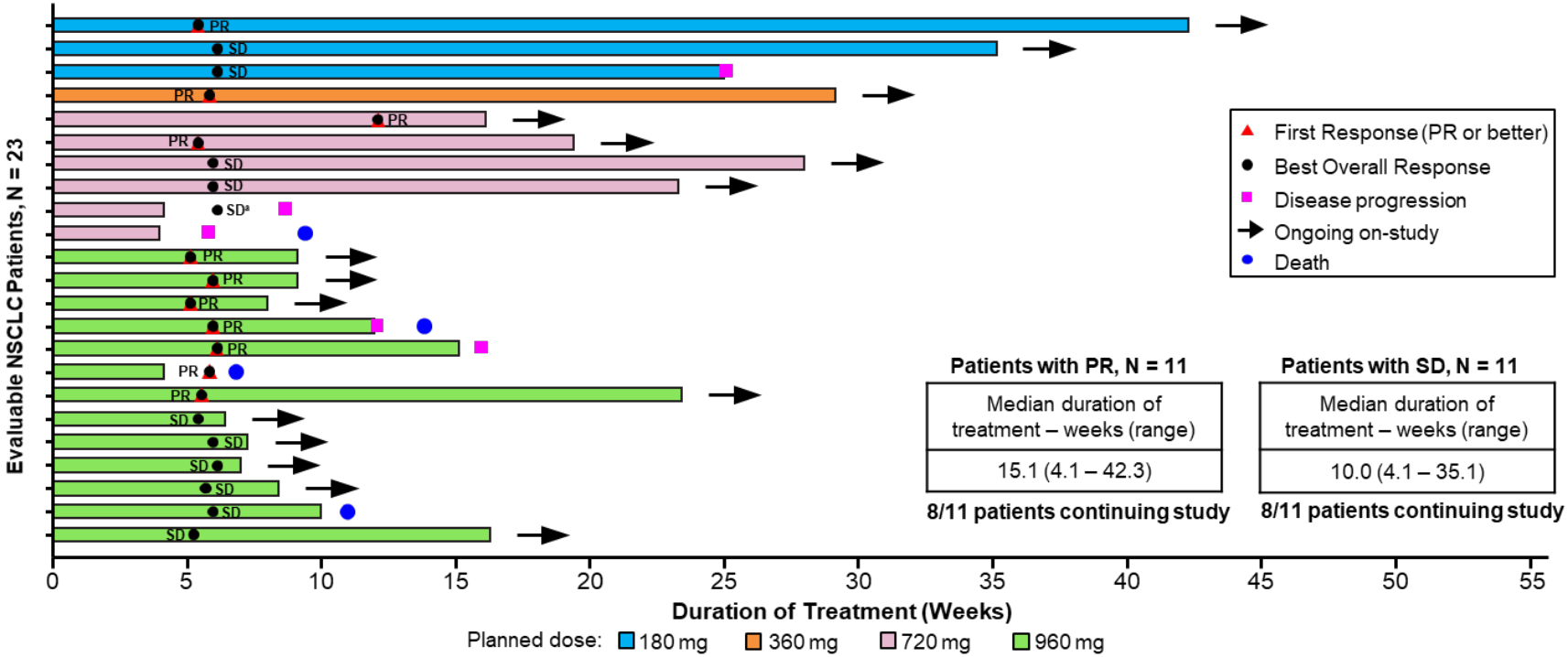
EFFICACY IN NSCLC

Time to Response and Duration of Treatment



[a]The graph was plotted based on the data received from the participating sites as of the data cutoff; duration of treatment data for this patient might be missing from the study site.
Evaluable patients: patients who had the first 6-week scan or early progressive disease; NSCLC = non-small cell lung cancer; PR = partial response; SD = stable disease

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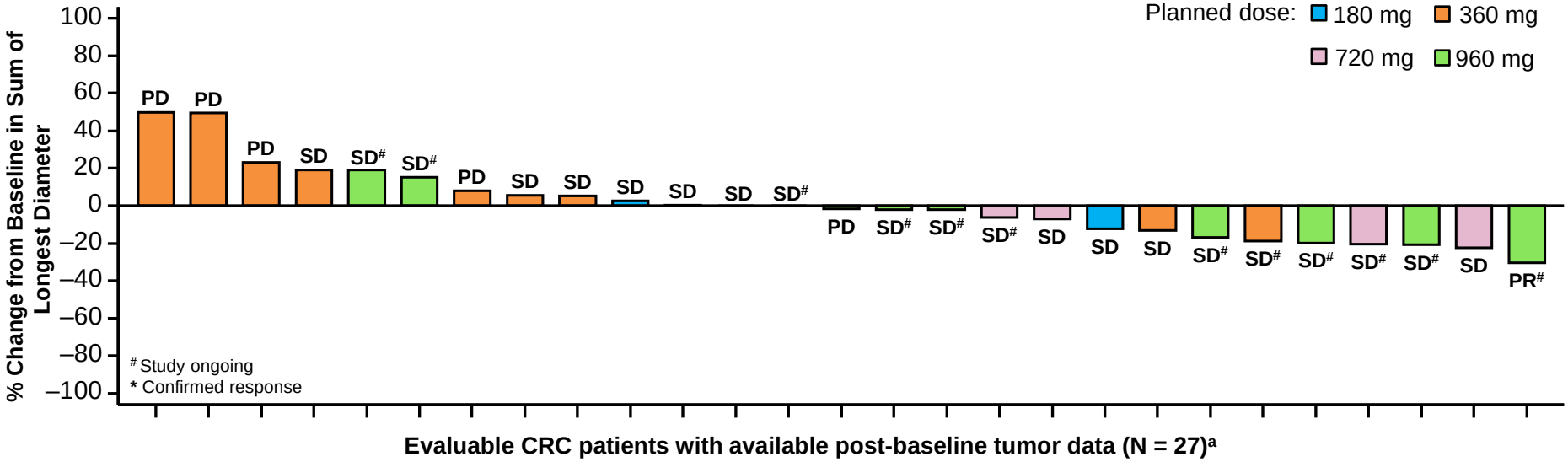
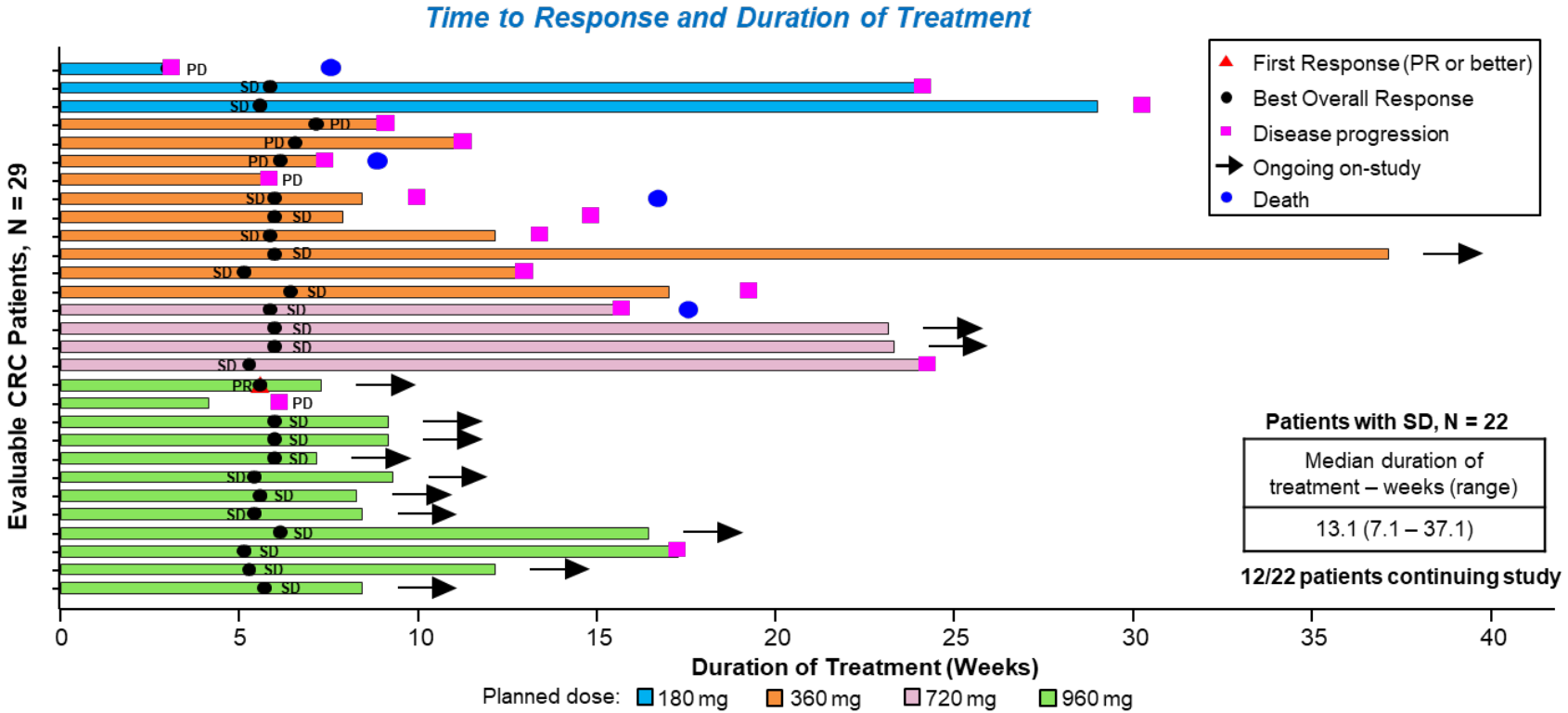
EFFICACY IN CRC

Change in Tumor Burden From Baseline



[a]2 patients–1 with SD, and 1 with PD–didn't have post-baseline tumor burden data available and therefore, are not shown on the graph. Evaluable patients = patients who had the first 6-week scan or early PD; CRC = colorectal cancer; PD = progressive disease; PR = partial response; SD = stable disease

EFFICACY IN CRC



Evaluable patients: patients who had the first 6-week scan or early PD; PD = progressive disease; PR = partial response; SD = stable disease

CONCLUSIONS

- **AMG 510 is a novel, first-in-class and irreversible inhibitor of KRAS^{G12C}**
- **AMG 510 has been found to have a favorable safety profile at the dose levels tested; no dose-limiting toxicities have been observed; and no cumulative toxicities were noted with extended treatment**
- **AMG 510 demonstrated early promising antitumor activity in patients with advanced solid tumors harboring *KRAS p. G12C* mutation**
- **Enrollment is ongoing for Phase 1 (in combination) and Phase 2 monotherapy**

***KRAS* = Kirsten rat sarcoma viral oncogene homolog; KRAS^{G12C} = KRAS protein with a G12C mutation at the protein level**

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ACKNOWLEDGMENTS

- **Patients, physicians and study teams at all participating centers**
- **Yang Li (Amgen Inc.) for medical writing support, Melissa Farley (Amgen Inc.) for study management, Bob Dawson for graphic assistance**
- **This study was funded by Amgen Inc. (ClinicalTrials.gov identifier: NCT03600883)**

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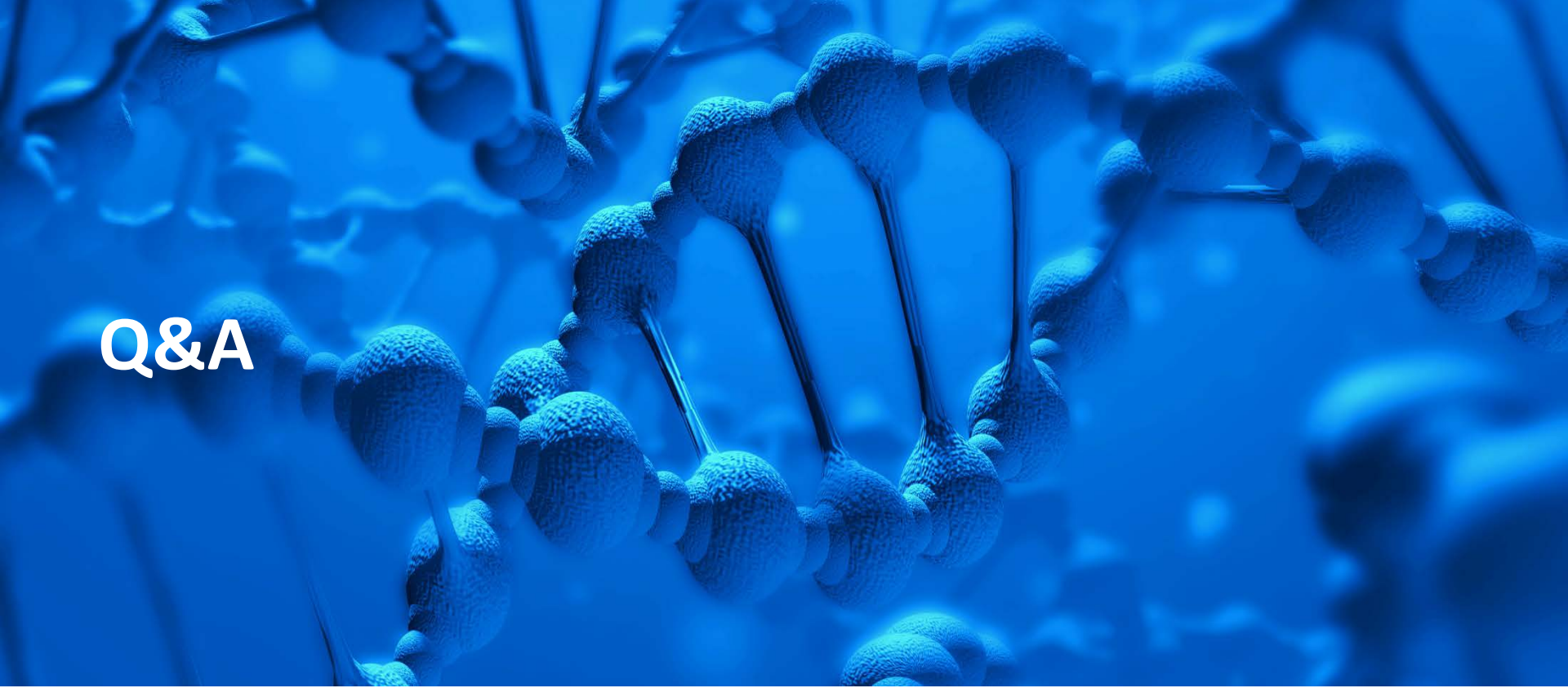
Q&A

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