AMGEN AT WCLC 2019

SEPTEMBER 8, 2019

AMGEN®

SAFE HARBOR STATEMENT

This presentation contains forward-looking statements that are based on the current expectations and beliefs of Amgen. All statements, other than statements of historical fact, are statements that could be deemed forward-looking statements, including estimates of revenues, operating margins, capital expenditures, cash, other financial metrics, expected legal, arbitration, political, regulatory or clinical results or practices, customer and prescriber patterns or practices, reimbursement activities and outcomes and other such estimates and results. Forward-looking statements involve significant risks and uncertainties, including those discussed below and more fully described in the Securities and Exchange Commission reports filed by Amgen, including our most recent annual report on Form 10-K and any subsequent periodic reports on Form 10-Q and current reports on Form 8-K. Unless otherwise noted, Amgen is providing this information as of the date of this presentation and does not undertake any obligation to update any forward-looking statements contained in this document as a result of new information, future events or otherwise. No forward-looking statement can be guaranteed and actual results may differ materially from those we project. Discovery or identification of new product candidates or development of new indications for existing products cannot be guaranteed and movement from concept to product is uncertain; consequently, there can be no guarantee that any particular product candidate or development of a new indication for an existing product will be successful and become a commercial product. Further, preclinical results do not guarantee safe and effective performance of product candidates in humans. The complexity of the human body cannot be perfectly, or sometimes, even adequately modeled by computer or cell culture systems or animal models. The length of time that it takes for us to complete clinical trials and obtain regulatory approval for product marketing has in the past varied and we expect similar variability in the future. Even when clinical trials are successful, regulatory authorities may question the sufficiency for approval of the trial endpoints we have selected. We develop product candidates internally and through licensing collaborations, partnerships and joint ventures. Product candidates that are derived from relationships may be subject to disputes between the parties or may prove to be not as effective or as safe as we may have believed at the time of entering into such relationship. Also, we or others could identify safety, side effects or manufacturing problems with our products, including our devices, after they are on the market.

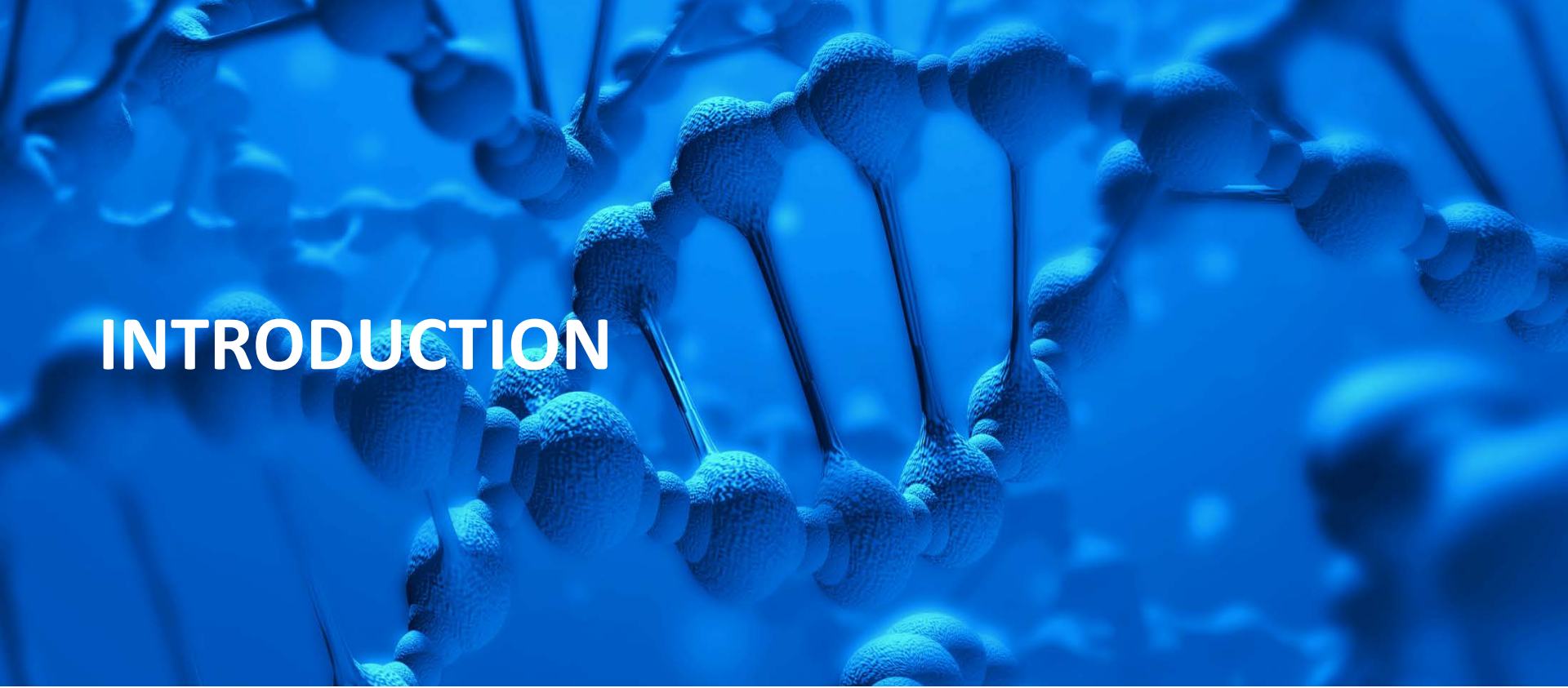
Our results may be affected by our ability to successfully market both new and existing products domestically and internationally, clinical and regulatory developments involving current and future products, sales growth of recently launched products, competition from other products including biosimilars, 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**

AMGEN®



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**
- **Initial clinical data from MCL-1 program at the International Myeloma Workshop, September 12-15**
- **BCMA BiTE® program focusing on intermittent dosing**
- **Clinical data from several BiTE® molecules in 2019**
- **Programs with compelling efficacy may rapidly advance toward registration**

MCL-1 = myeloid cell leukemia-1; BCMA = B-cell maturation antigen; BiTE® = bispecific T-cell engager

AMGEN®

AMGEN ONCOLOGY: A BROAD, DIFFERENTIATED PORTFOLIO OF HIGH-POTENTIAL PROGRAMS

Hematology/Hematologic Malignancies

MULTIPLE MYELOMA


AMG 420
(BiTE® molecule targeting BCMA)
PHASE 1

MULTIPLE MYELOMA


Carfilzomib
(proteasome inhibitor)
PHASE 3

NON-HODGKIN'S LYMPHOMA


Blinatumomab
(BiTE® molecule targeting CD19)
PHASE 2

MULTIPLE MYELOMA


AMG 701
(HLE-BiTE® molecule targeting BCMA)
PHASE 1

MULTIPLE MYELOMA


AMG 424
(XmAb® [CD38 x CD3])
PHASE 1

NON-HODGKIN'S LYMPHOMA


AMG 562
(HLE-BiTE® molecule targeting CD19)
PHASE 1

MULTIPLE MYELOMA
ACUTE MYELOID LEUKEMIA


AMG 176
(Intravenous MCL-1 inhibitor)
PHASE 1

ACUTE MYELOID LEUKEMIA


AMG 330
(BiTE® molecule targeting CD33)
PHASE 1

NON-HODGKIN'S LYMPHOMA*


ABP 798
(rituximab biosimilar)
PHASE 3

MULTIPLE MYELOMA
ACUTE MYELOID LEUKEMIA
NON-HODGKIN'S LYMPHOMA


AMG 397
(Oral MCL-1 inhibitor)
PHASE 1

ACUTE MYELOID LEUKEMIA


AMG 673
(HLE-BiTE® molecule targeting CD33)
PHASE 1

PAROXYSMAL NOCTURNAL HEMOGLOBINURIA*


ABP 959
(eculizumab biosimilar)
PHASE 3

ACUTE MYELOID LEUKEMIA


AMG 427
(HLE-BiTE® molecule targeting FLT3)
PHASE 1

Solid Tumors

SOLID TUMORS


AMG 510
(KRAS G12C inhibitor)
PHASE 1

PROSTATE CANCER


AMG 160
(HLE-BiTE® molecule targeting PSMA)
PHASE 1

GLIOBLASTOMA


AMG 596
(BiTE® molecule targeting EGFR vIII)
PHASE 1

SOLID TUMORS
MELANOMA


Talimogene laherparepvec
(oncolytic viral therapy)
PHASE 1
PHASE 3

PROSTATE CANCER


AMG 212
(BiTE® molecule targeting PSMA)
PHASE 1

CHEMOTHERAPY-INDUCED THROMBOCYTOPENIA


Romiplostim
(thrombopoiesis stimulator)
PHASE 3

SOLID TUMORS


AMG 404
(Anti-PD-1 antibody)
PHASE 1

SMALL CELL LUNG CANCER


AMG 757
(HLE-BiTE® molecule targeting DLL3)
PHASE 1

***The regulatory approval pathway for biosimilars requires study of a single indication and permits extrapolation to other reference indications with scientific justification; MCL-1 = myeloid cell leukemia-1; BiTE® = bispecific T-cell engager; BCMA = B-cell maturation antigen; HLE = half-life extended; FLT3 = fms-like tyrosine kinase 3; DLL3 = delta-like 3; PSMA = prostate-specific membrane antigen; EGFR vIII = epidermal growth factor receptor variant III; PD-1 = programmed cell death protein 1**

AMGEN®

AMG 510 UPDATE

GREGORY FRIBERG, M.D.

VICE PRESIDENT, GLOBAL DEVELOPMENT AND ONCOLOGY
THERAPEUTIC AREA HEAD

AMGEN®

Phase 1 Study of Safety, Tolerability, Pharmacokinetics, and Efficacy of AMG 510, a Novel KRAS^{G12C} Inhibitor, in Non-Small Cell Lung Cancer

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, Australia; [4]Sarah Cannon Research Institute at HealthONE, Denver, CO, USA; [5]Peter MacCallum Cancer Centre, Melbourne, Australia; [6]Scientia Clinical Research, Randwick, Australia; [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

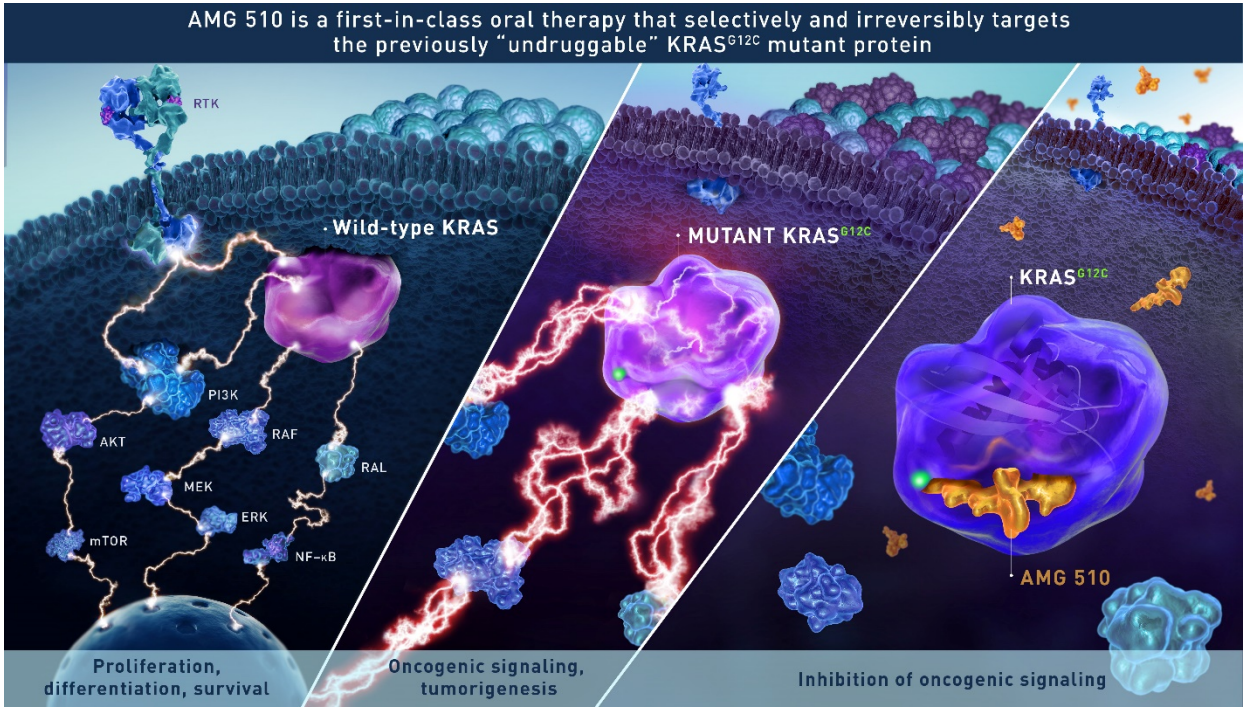
DISCLOSURES

Dr. Ramaswamy Govindan, MD

Commercial Interest	Relationship(s)
AbbVie Inc., Adaptimmune Ltd, AstraZeneca LP, Celgene Corporation, Ignyta Inc, Inivata Ltd, Merck & Co, NEKTAR, Pfizer Pharmaceuticals, Roche	Consulting or advisory role

AMG 510 is a First-in-Class KRAS^{G12C} Inhibitor

- *KRAS G12C* mutation is found in approximately **13% of lung cancer**,[1] **3% of colorectal cancer**[2] and appendix cancer, and **1%–3% of other solid tumors**[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



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.

AMG 510 First-in-Human Study Design

Phase 1, Multicenter, Open-label Study – Dose Escalation

Key Eligibility

- Locally advanced or metastatic malignancy
- Received prior standard therapies
- *KRAS G12C* mutation as assessed by molecular testing of tumor biopsies
- No active brain metastases

Screening / Enrollment

- 2–4 patients enrolled in each cohort
- Intra-patient dose escalation allowed
- Additional patients may be added to any dose deemed safe

Cohort 1 180 mg → Cohort 2 360 mg → Cohort 3 720 mg → Cohort 4 960 mg

- Repeated **oral daily dosing** with 21-day cycles
- Treatment until disease progression, intolerance, or consent withdrawal
- Radiographic scan every 6 weeks

Safety Follow-up & Long-term Follow-up[a]

Expansion dose determined

Dose Expansion

Screening / Enrollment

Patients with $KRAS^{G12C}$ mutant advanced tumors

N = ~20

(max 60)

Safety Follow-up & Long-term Follow-up[a]

Primary endpoints: dose-limiting toxicities; 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. PK: pharmacokinetics; PFS: progression-free survival.

Patient Disposition

Overall Trial Accrual

N = 76
Total patients enrolled
- NSCLC, N = 34
- CRC, N = 36
- SCLC, N = 1[a]
- Appendiceal cancer, N = 3
- Endometrial cancer, N = 1
- Small bowel cancer, N = 1

N = 34
NSCLC patients enrolled

N = 19, escalation cohort
- 180 mg: N = 3
- 360 mg: N = 2
- 720 mg: N = 6
- 960 mg: N = 8

N = 15, expansion cohort
- 960 mg: N = 15

N = 23, evaluable
- Patients who had the first 6-week scan or early progressive disease

N = 30
Remained on study

N = 4
Discontinued study[b]
- None was related to treatment

First patient enrolled: 27 August 2018

Data cutoff: 17 July 2019

[a]The tumor type of this patient was recorded as SCLC as of the data cutoff and changed to NSCLC by the participating site after cutoff. [b]As of the data cutoff, in addition to four patients who discontinued study, three patients discontinued treatment due to progressive disease. NSCLC: non-small cell lung cancer; CRC: colorectal cancer; SCLC: small cell lung cancer.

Baseline Characteristics

Baseline Characteristics	N = 34
Median age (range) – years	67.5 (49.0–77.0)
Female – n (%)	18 (52.9)
ECOG performance status score – n (%) 0 1 2	 5 (14.7) 26 (76.5) 3 (8.8)
Prior lines of systemic anticancer therapy – n (%) 1 2 > 2	 2 (5.9) 3 (8.8) 29 (85.3)
No. of prior systemic anticancer therapy – median (range)	3.5 (1–8)

ECOG: Eastern Cooperative Oncology Group.

Patient Incidence of Adverse Events (AEs): Summary

	All AEs N = 34 n (%)	All treatment-related AEs N = 34 n (%)
Any grade Grade ≥ 2 Grade ≥ 3 Grade ≥ 4	26 (76.5) 20 (58.8) 11 (32.4) 5 (14.7)	**12 (35.3)** **8 (23.5)** **3 (8.8)** **0 (0)**
Dose-limiting toxicity	0 (0)	**0 (0)**
Serious AEs	8 (23.5)	**0 (0)**[b]
Fatal AEs	4 (11.8)[a]	**0 (0)**
AEs leading to treatment discontinuation	0 (0)	**0 (0)**

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

- **960 mg oral daily dose** was identified as the expansion dose and recommended phase 2 dose

[a]Four patients had the following fatal AEs: dyspnea, aspiration, lung cancer metastatic, and spinal compression fracture; none was related to treatment. [b]One 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.

Patient Incidence of Treatment-Related Adverse Events (AEs)

All Treatment-Related AEs	Any Grade N = 34, n (%)	Grade 3 N = 34, n (%)
Any treatment-related AEs	12 (35.3)	3 (8.8)
Diarrhea	4 (11.8)	2 (5.9)
Nausea	2 (5.9)	0 (0)
Dry mouth	1 (2.9)	0 (0)
Vomiting	1 (2.9)	0 (0)
ALT increased	2 (5.9)	0 (0)
AST increased	2 (5.9)	0 (0)
Blood alkaline phosphate increased	1 (2.9)	0 (0)
Lymphocyte count decreased	1 (2.9)	0 (0)
White blood cell count decreased	1 (2.9)	0 (0)

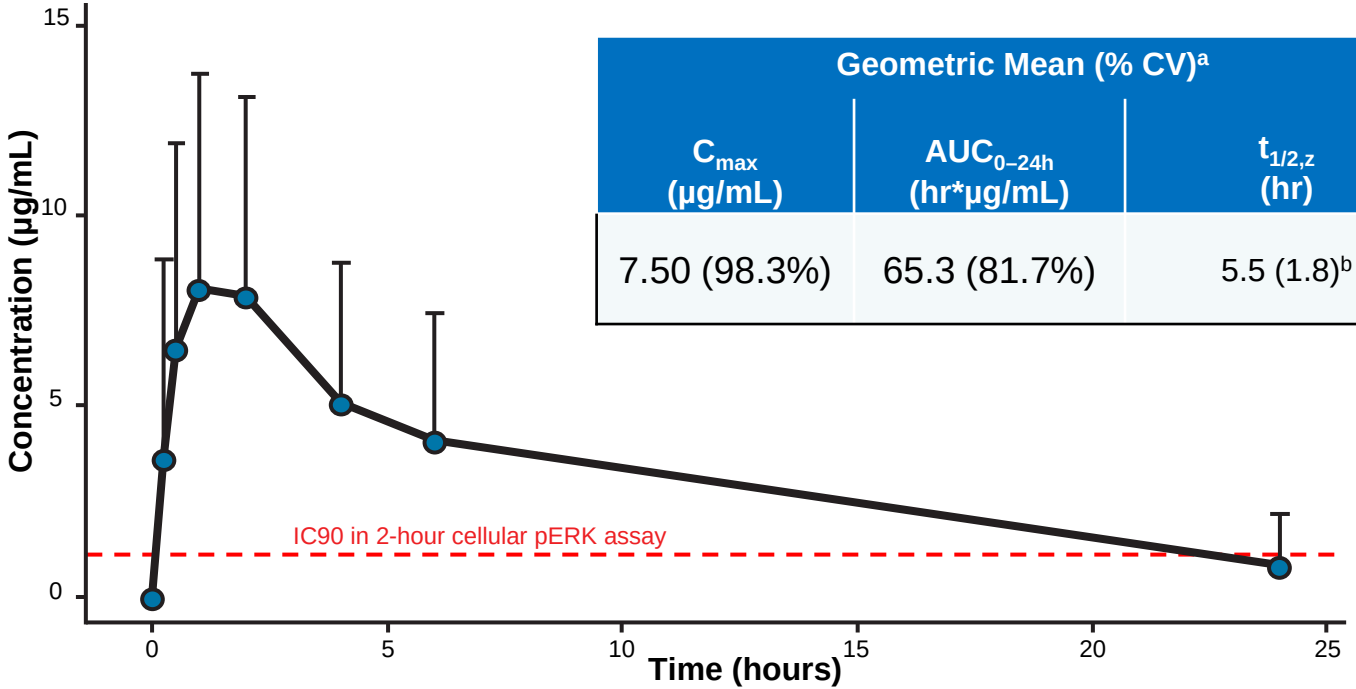
Cont.

All Treatment-Related AEs	Any Grade N = 34, n (%)	Grade 3 N = 34, n (%)
Decreased appetite	1 (2.9)	0 (0)
Hyperkalemia	1 (2.9)	0 (0)
Hypokalemia	1 (2.9)	0 (0)
Anemia	1 (2.9)	1 (2.9)
Leukopenia	1 (2.9)	0 (0)
Dysgeusia	1 (2.9)	0 (0)
Neuropathy peripheral	1 (2.9)	0 (0)
Proteinuria	1 (2.9)	0 (0)

- 12 of 34 patients **(35.3%)** reported treatment-related AEs; most were grade 1 or 2
- 3 of 34 patients **(8.8%)** reported two grade 3 treatment-related AEs: diarrhea and anemia
- There were no grade 4 or higher treatment-related AEs.

ALT: alanine aminotransferase; AST: aspartate aminotransferase; AE: adverse event.

AMG 510 Pharmacokinetic Profile – 960 mg Oral Daily Dose

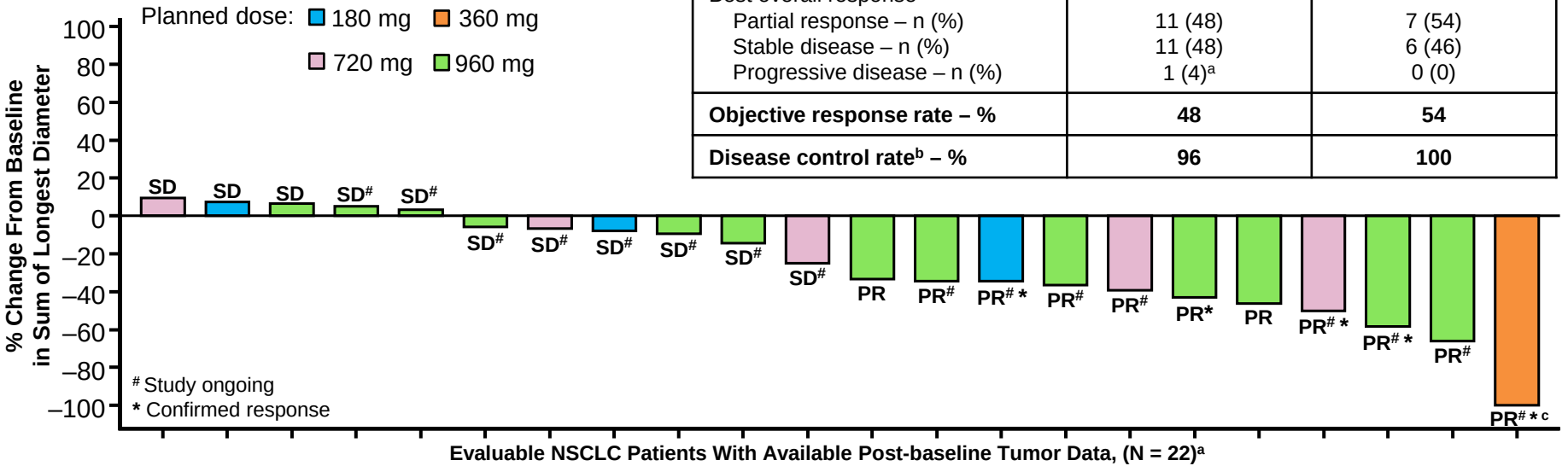


Geometric Mean (% CV)[a]		
C_{max} (µg/mL)	AUC_{0-24h} (hr*µg/mL)	$t_{1/2,z}$ (hr)
7.50 (98.3%)	65.3 (81.7%)	5.5 (1.8)[b]

[a]PK data cutoff: 24 July 2019; N = 32 including patients with CRC. [b]$t_{1/2}$ is presented as mean (standard deviation). Only the top error bars are shown for clarify on a semi-logarithmic scale (non-symmetric scale).
PK: pharmacokinetics; C_{max}: maximum serum concentration; $t_{1/2}$: elimination half life; CV: coefficient of variation; AUC: area under the curve; IC90: 90% inhibitory concentration in vitro; pERK: phosphorylated extracellular signal-regulated kinase.

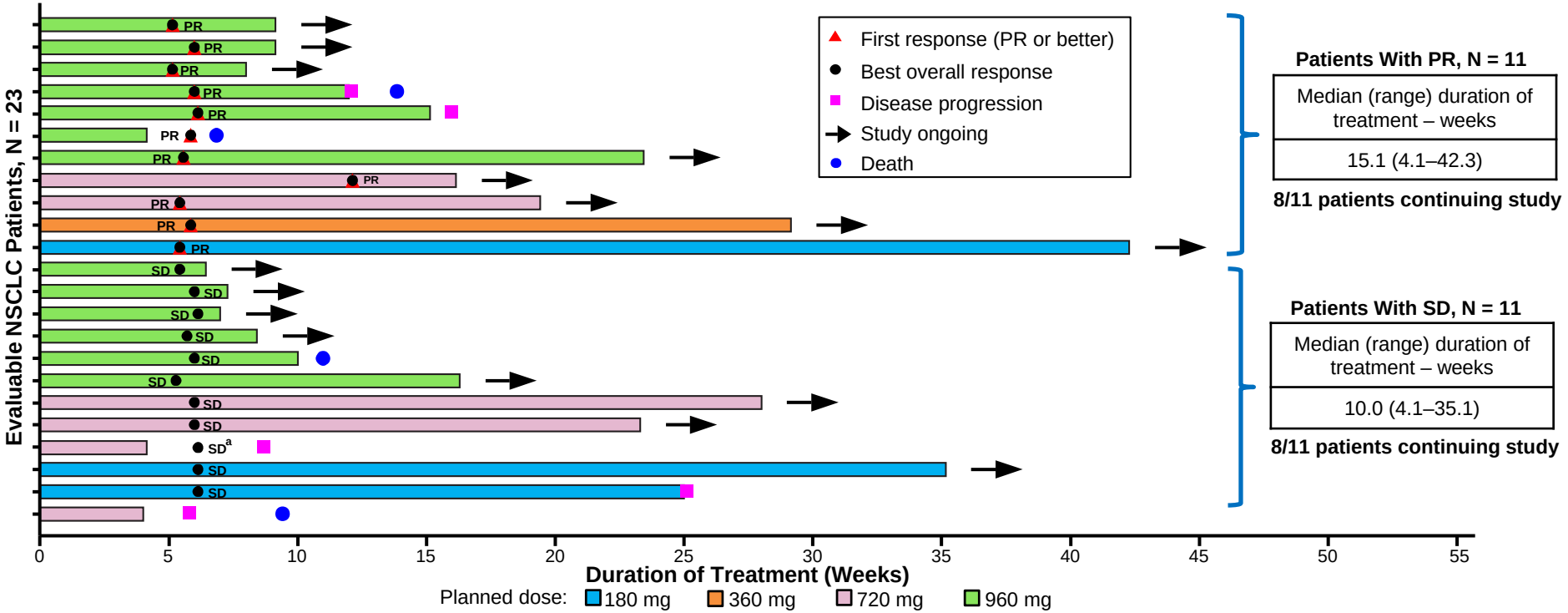
Best Tumor Response and Change in Tumor Burden From Baseline

Efficacy outcomes	All evaluable patients N = 23	Evaluable patients treated with 960mg N = 13
Best overall response		
Partial response – n (%)	11 (48)	7 (54)
Stable disease – n (%)	11 (48)	6 (46)
Progressive disease – n (%)	1 (4)[a]	0 (0)
Objective response rate – %	**48**	**54**
Disease control rate[b] – %	**96**	**100**



[a]One patient discontinued study due to PD prior to the 1st assessment, and the post-baseline tumor burden data are missing. [b]PR or SD at week 6. [c]Patient had complete response to the target lesions. Evaluable patients: patients who had the first 6-week scan or early PD; NSCLC: non-small cell lung cancer; PR: partial response; SD: stable disease; PD: progressive disease.

Time to Response and Duration of Treatment for All Dose Levels



Patients With PR, N = 11

Median (range) duration of treatment – weeks
15.1 (4.1–42.3)

8/11 patients continuing study

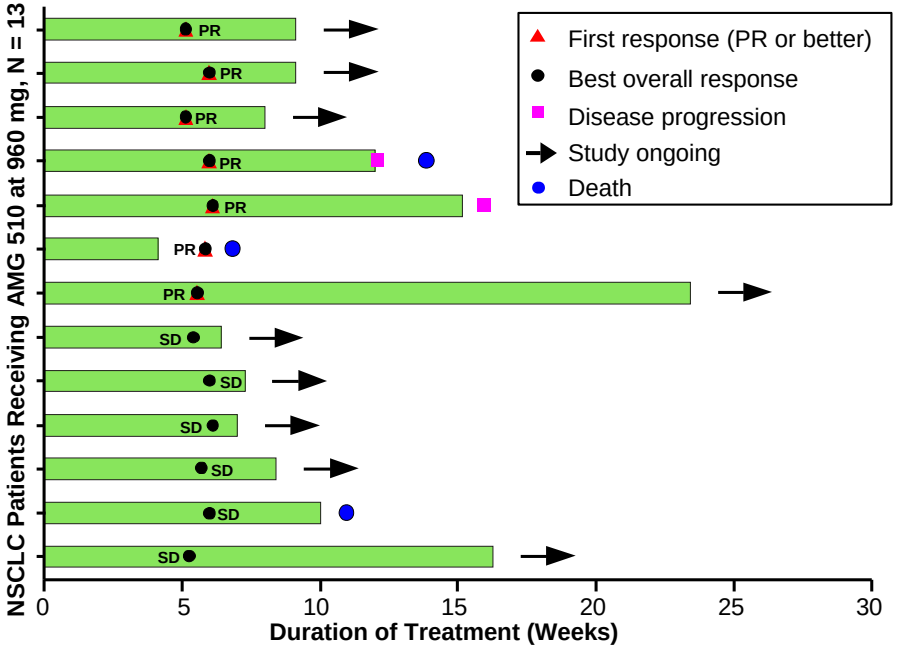
Patients With SD, N = 11

Median (range) duration of treatment – weeks
10.0 (4.1–35.1)

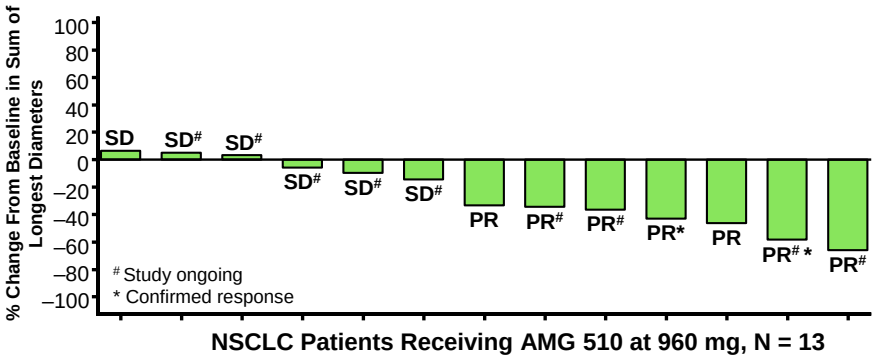
8/11 patients continuing study

[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. PR: partial response; SD: stable disease.

Efficacy of AMG 510 Administered at 960 mg, the Recommended Phase 2 Dose



Efficacy with 960 mg	Evaluable NSCLC patients receiving 960 mg, N = 13
Best overall response Partial response – n (%) Stable disease – n (%) Progressive disease – n (%)	 7 (54) 6 (46) 0 (0)
Objective response rate – %	**54**
Disease control rate[a] – %	**100**



[a]PR or SD at week 6. Evaluable patients: patients who had the first 6-week scan or early 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 DLTs have been observed; and no cumulative toxicities were noted with extended treatment
- AMG 510 demonstrated early promising antitumor activity in patients with advanced NSCLC harboring *KRAS G12C* mutation
- Enrollment is ongoing for phase 1 (in combination) and phase 2 monotherapy

DLTs: dose-limiting toxicities; NSCLC: non-small cell lung cancer.

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 (Amgen Inc.) for graphic assistance, and Nitish Upadhyay (IQVIA) for biostatistics support

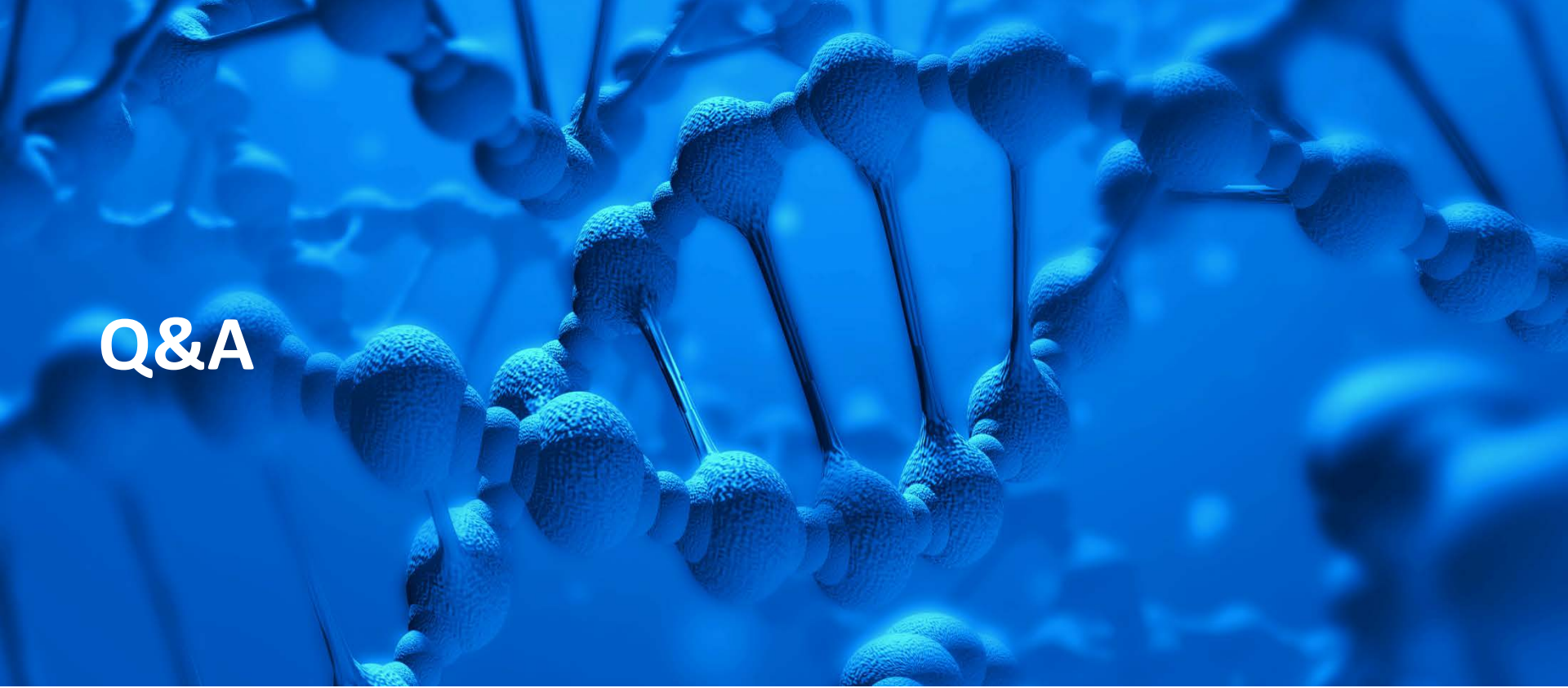
- This study was funded by Amgen Inc (ClinicalTrials.gov identifier: NCT03600883)

SUMMARY

- **AMG 510 clinical program is advancing rapidly**
  - **Next clinical update at European Society for Medical Oncology (ESMO) Congress, September 28**
  - **Completed enrollment of 960 mg dose expansion cohort**
  - **Enrolling NSCLC patients in anti-PD-1 combination cohort**
  - **Enrolling patients in potentially pivotal Phase 2 monotherapy study**
- **Upcoming data presentations from multiple oncology programs**
  - **AMG 420 (BCMA BiTE® molecule) and AMG 176 (MCL-1 inhibitor) at International Myeloma Workshop, September 12—15**
  - **AMG 596 (EGFR vIII BiTE® molecule) and AMG 673 (CD33 HLE-BiTE® molecule) submitted for presentation in 2019**

NSCLC = non-small-cell lung cancer

AMGEN®


Q&A

AMGEN AT WCLC 2019

SEPTEMBER 8, 2019

AMGEN®