AMGEN AT ASCO20 VIRTUAL SCIENTIFIC PROGRAM

MAY 29, 2020

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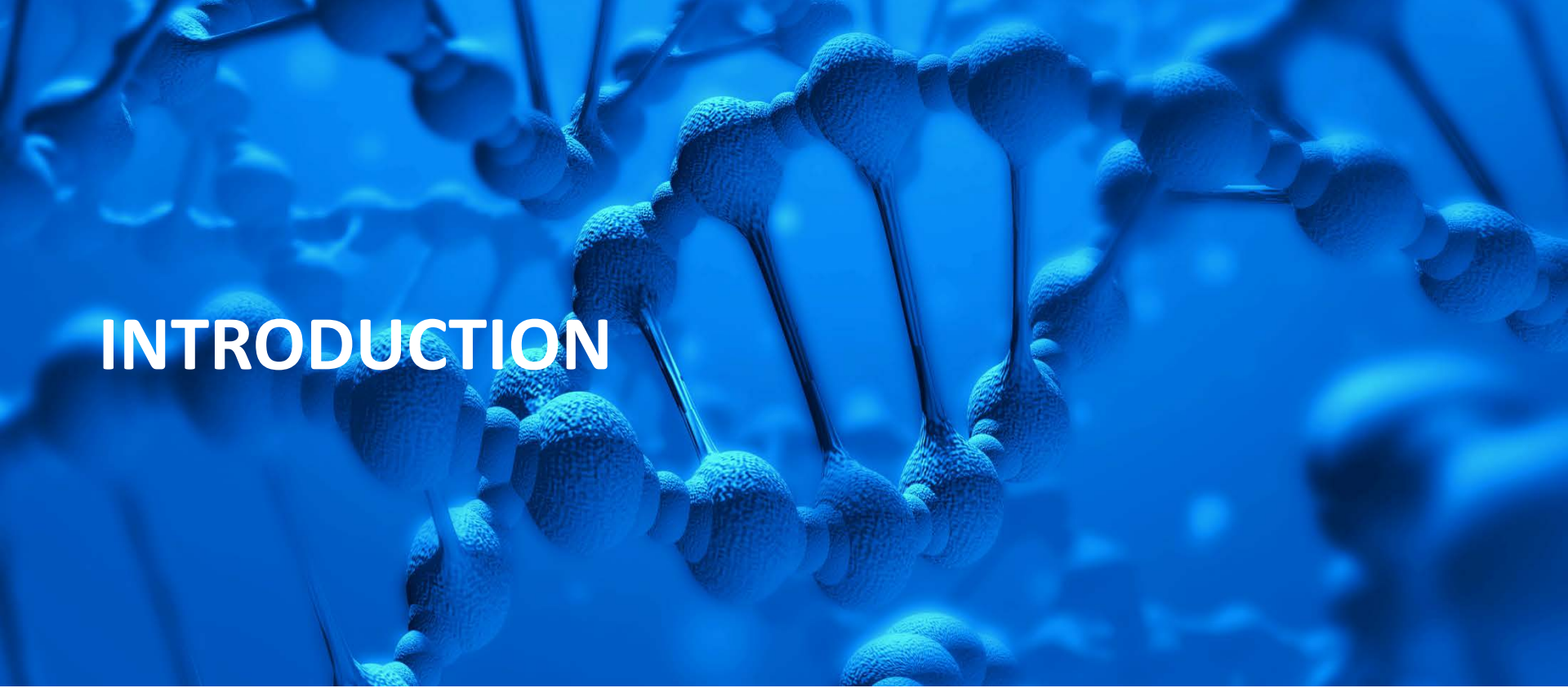
AGENDA

Introduction	**David Reese, M.D.—Executive Vice President, Research and Development**
ASCO Data Overview	**Gregory Friberg, M.D.—Vice President, Global Development and Oncology Therapeutic Area Head**
Q&A	**David Reese, M.D.** **Gregory Friberg, M.D.** **Marwan Fakih, M.D.—Professor, Department of Medical Oncology and Therapeutics Research, City of Hope, Duarte, CA** **David Hong, M.D.—Deputy Chair, Department of Investigational Cancer Therapeutics, Division of Cancer Medicine, The University of Texas MD Anderson Cancer Center, Houston, TX**



INTRODUCTION

DAVID REESE, M.D.

EXECUTIVE VICE PRESIDENT, RESEARCH AND DEVELOPMENT

AMGEN®

AMGEN ONCOLOGY: A BROAD, DIFFERENTIATED PORTFOLIO

- **Built on first-in-class molecules with high potential**
- **Developing combination/sequential therapies against multiple targets in several indications to drive deep, durable responses**
- **Prioritizing high-potential programs for rapid advancement**
- **AMG 510 (sotorasib) data updates in 2020**
  - **Phase 1 NSCLC dose expansion data**
  - **Potentially pivotal NSCLC Phase 2 data (≥ 6 months follow-up after first response)**
- **Half-life extended BiTE® program updates in 2020**
  - **AMG 757 (DLL3) dose escalation data in small cell lung cancer**
  - **AMG 160 (PSMA) dose escalation data in prostate cancer**
  - **AMG 701 (BCMA) dose escalation data in multiple myeloma**

NSCLC = non-small-cell lung cancer; ORR = overall response rate; PFS = progression-free survival; BiTE® = bispecific T-cell engager; DLL3 = delta-like ligand 3; PSMA = prostate-specific membrane antigen; BCMA = B-cell maturation antigen

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

- **Highly selective covalent inhibitor with no dose limiting toxicities**
- **Significant unmet need for patients with KRAS^{G12C} tumors with sub-optimal current standards of care**
- **Most advanced and robust KRAS^{G12C} clinical program**
  - **> 400 patients dosed with 13 different tumor types in CodeBreaK 100 study**
  - **Phase 2 monotherapy studies in NSCLC and CRC fully enrolled**
  - **Enrollment commenced in Phase 3 monotherapy NSCLC study**
  - **Paused studies beginning to enroll patients on site-by-site basis**
  - **Exploring six different combination cohorts in CodeBreaK 101 study with more to come**
  - **ASCO20: data on 42 CRC patients with 7.9 months follow-up; 25 patients with non-NSCLC, non-CRC solid tumors**

KRAS **= Kirsten rat sarcoma viral oncogene homolog; CRC = colorectal cancer**

VIRTUAL ASCO20 HIGHLIGHTS

CodeBreak 100: Activity of AMG 510, a novel small molecule inhibitor of KRAS^{G12C}, in patients with advanced colorectal cancer	**Abstract 4018 (Poster 10)**
CodeBreak 100: Phase 1 study of AMG 510, a novel KRAS^{G12C} inhibitor, in patients (pts) with advanced solid tumors other than non–small-cell lung cancer (NSCLC) and colorectal cancer (CRC)	**Abstract 3511 (Poster 241)**
Updated Results From Phase 1 Dose Escalation Study Of AMG 330, A Bispecific T-Cell Engager Molecule, in Patients With Relapsed/Refractory Acute Myeloid Leukemia (R/R AML)	**Abstract 7508**

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AMG 510 (SOTORASIB) UPDATE

GREGORY FRIBERG, M.D.

VICE PRESIDENT, GLOBAL DEVELOPMENT AND ONCOLOGY
THERAPEUTIC AREA HEAD

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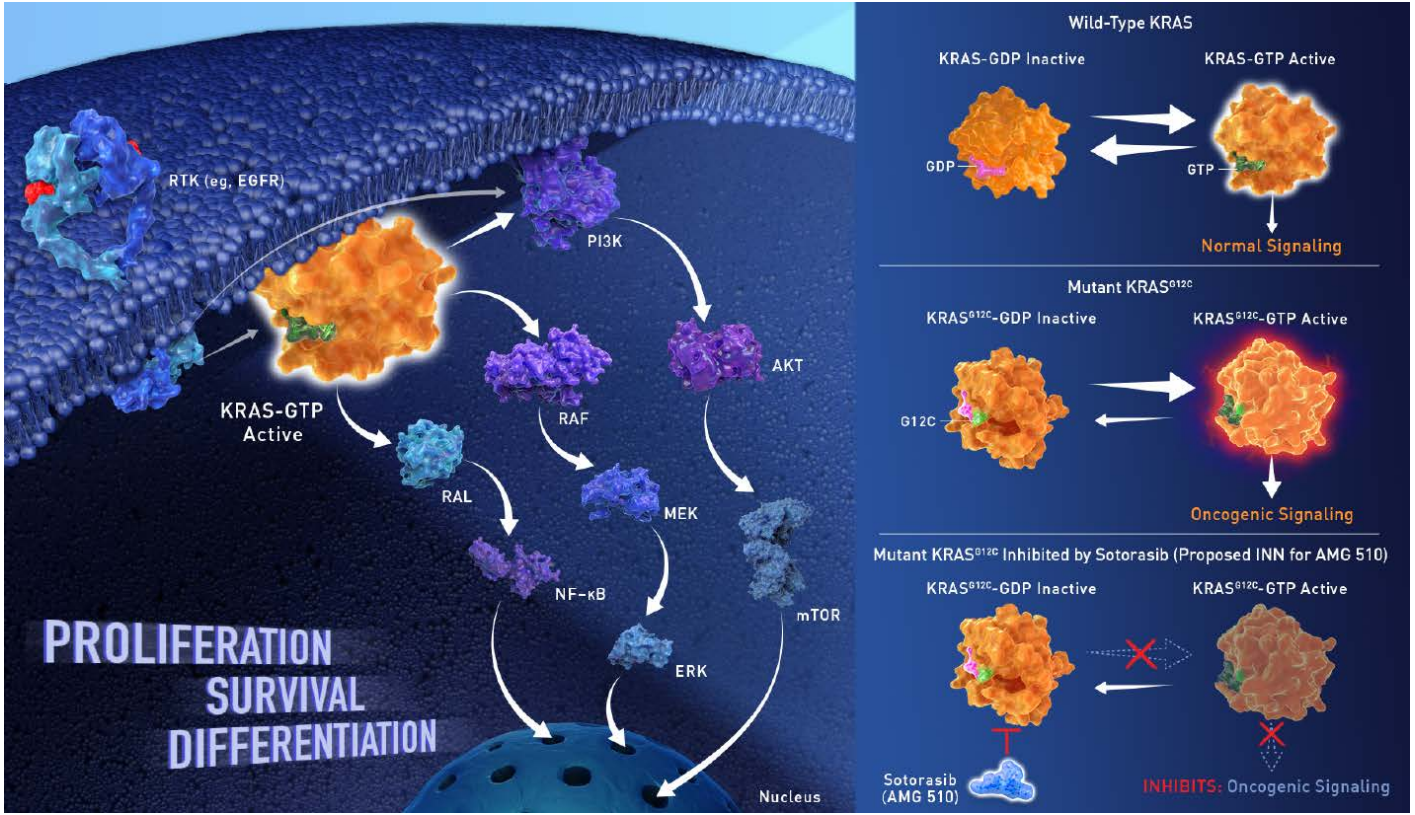
AMG 510 (SOTORASIB) MECHANISM OF ACTION



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CODEBREAK 100: ACTIVITY OF AMG 510, A NOVEL SMALL MOLECULE INHIBITOR OF KRAS^{G12C}, IN PATIENTS WITH ADVANCED COLORECTAL CANCER

Marwan G. Fakih,[1] Jayesh Desai,[2] Yasutoshi Kuboki,[3] John H. Strickler,[4] Timothy J. Price,[5] Gregory A. Durm,[6] Gerald S. Falchook,[7] Crystal S. Denlinger,[8] John C. Krauss,[9] Geoffrey I. Shapiro,[10] Tae Won Kim,[11] Keunchil Park,[12] Andrew L. Coveler,[13] Pamela N. Munster,[14] Bob T. Li,[15] June Kim,[16] Haby Henary,[16] Gataree Ngarmchamnanrith,[16] David S. Hong[17]

[1]Department of Medical Oncology and Experimental Therapeutics, City of Hope Comprehensive Cancer Center, Duarte, California, USA; [2]Royal Melbourne Hospital/Peter MacCallum Cancer Centre, Victoria, VIC, Australia; [3]Department of Experimental Therapeutics, National Cancer Center Hospital East, Kashiwa, Japan; [4]Duke University Medical Center, Durham, North Carolina, USA; [5]The Queen Elizabeth Hospital and University of Adelaide, Woodville South, Australia; [6]Department of Medicine, Division of Hematology/Oncology, Indiana University School of Medicine, Indianapolis, Indiana, USA; [7]Sarah Cannon Research Institute at HealthONE, Denver, Colorado, USA; [8]Fox Chase Cancer Center, Philadelphia, Pennsylvania, USA; [9]University of Michigan, Ann Arbor, Michigan, USA; [10]Dana-Farber Cancer Institute, Harvard Medical School, Boston, Massachusetts, USA; [11]Department of Oncology, Asan Medical Centre, University of Ulsan College of Medicine, Seoul, South Korea; [12]Samsung Medical Center, Sungkyunkwan University School of Medicine, Seoul, South Korea; [13]Department of Medicine, Division of Oncology, University of Washington, Seattle, Washington, USA; [14]Helen Diller Family Comprehensive Cancer Center, San Francisco, California, USA; [15]Memorial Sloan Kettering Cancer, Center, New York, New York, USA; [16]Amgen Inc. Thousand Oaks, California, USA; [17]Department of Investigational Cancer Therapeutics, Phase I Clinical Trials Program, The University of Texas MD Anderson Cancer Center, Houston, Texas, USA

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CRC: BASELINE CHARACTERISTICS

Baseline characteristics	N = 42
Median age (range) – year	57.5 (33–82)
Female – n (%)	21 (50)
ECOG performance at baseline – n (%) 0 1	 17 (40.5) 25 (59.5)
Prior lines of systemic anticancer therapy – n (%) 1 2 3 > 3	 2 (4.8) 11 (26.2) 10 (23.8) 19 (45.2)
Number of prior lines of systemic anticancer therapy – median (range)	3 (1–4)

Median follow-up: 7.9 (range: 4.2–15.9) months

ECOG: Eastern Cooperative Oncology Group

CRC: PATIENT INCIDENCE OF ADVERSE EVENTS

	Treatment-Emergent AEs (TEAEs) N = 42, n (%)	Treatment-related TEAEs N = 42, n (%)
Any grade Grade ≥ 2 Grade ≥ 3 Grade ≥ 4	38 (90.5) 29 (69.0) 13 (31.0) 3 (7.1)	20 (47.6) 9 (21.4) 2 (4.8) 0 (0.0)
Dose-limiting toxicities	0 (0.0)	0 (0.0)
Serious AEs	10 (23.8)	0 (0.0)
Fatal AEs	3 (7.1)	0 (0.0)
AEs leading to treatment discontinuation	2 (4.8)	0 (0.0)

Treatment-related TEAEs of any grade occurring in > 1 patients	N = 42, n (%)
Diarrhea	8 (19.0)
Fatigue	4 (9.5)
Nausea	2 (4.8)
Blood creatine phosphokinase increase	2 (4.8)
Anemia	2 (4.8)
Vomiting	2 (4.8)

- **Grade 3 treatment-related TEAEs**: diarrhea and anemia, occurring in 1 patient each.

AE: adverse event

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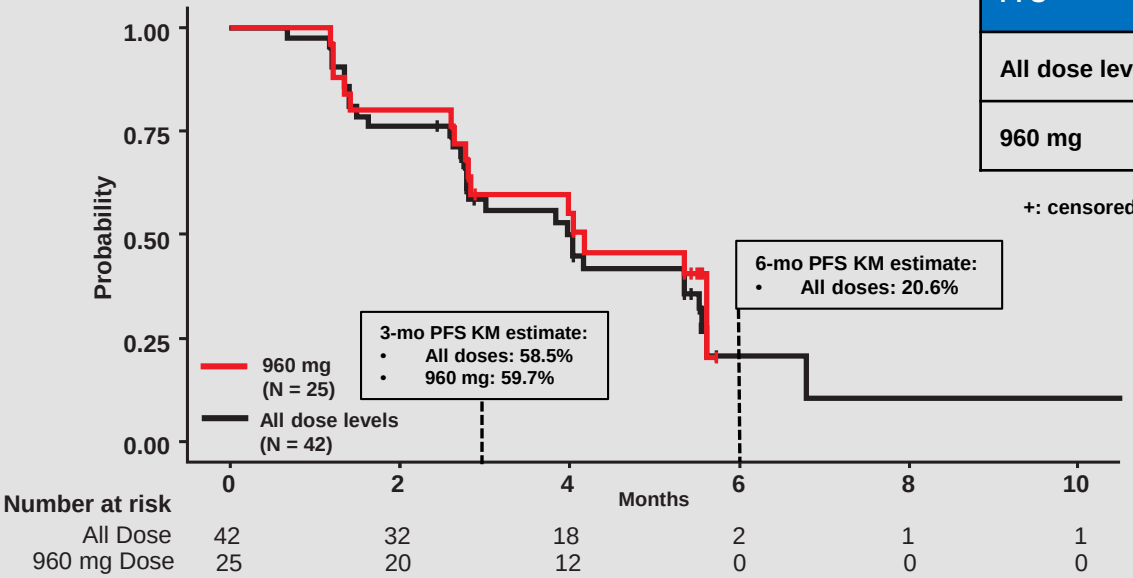
CRC: TUMOR RESPONSE

Efficacy outcomes	All dose levels N = 42, n (%)	960 mg N = 25, n (%)
Best overall response		
Confirmed partial response – n (%)	3 (7.1)	3 (12.0)
Stable disease – n (%)	29 (69.0)	17 (68.0)
Progressive disease – n (%)	9 (21.4)	4 (16.0)
Not done – n (%)[a]	1 (2.4)	1 (4.0)
Objective response rate – % (95% CI)	7.1 (1.50, 19.48)	12.0 (2.55, 31.22)
Disease control rate – % (95% CI)	76.2 (60.55, 87.95)	80.0 (59.30, 93.17)
Duration of response for 3 responders – months	1.4+, 4.2+, 4.3+	1.4+, 4.2+, 4.3+
Duration of stable disease – months Median (min, max)	4.2 (2.5+, 11.0)	4.2 (2.6, 5.7+)

[a]**Patient had clinical progression with no postbaseline measurement. +: censored value**

CRC: PROGRESSION-FREE SURVIVAL



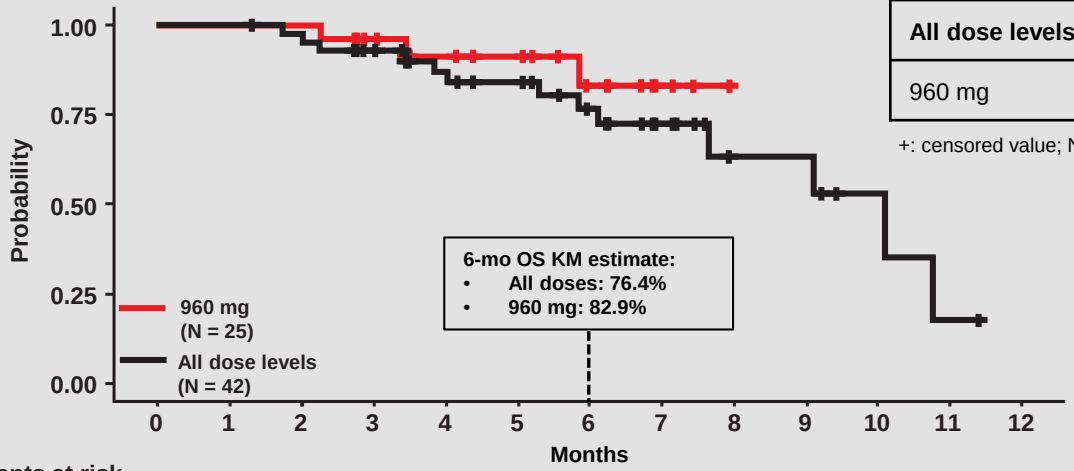
PFS	Median (min, max) - months
All dose levels	4.0 (0.7, 11.0)
960 mg	4.2 (1.2, 5.7+)

+: censored value.

CRC: OVERALL SURVIVAL

OS	Median (min, max) - months
All dose levels	**10.1 (1.3+, 11.4+)**
960 mg	NE (2.3, 8.0+)

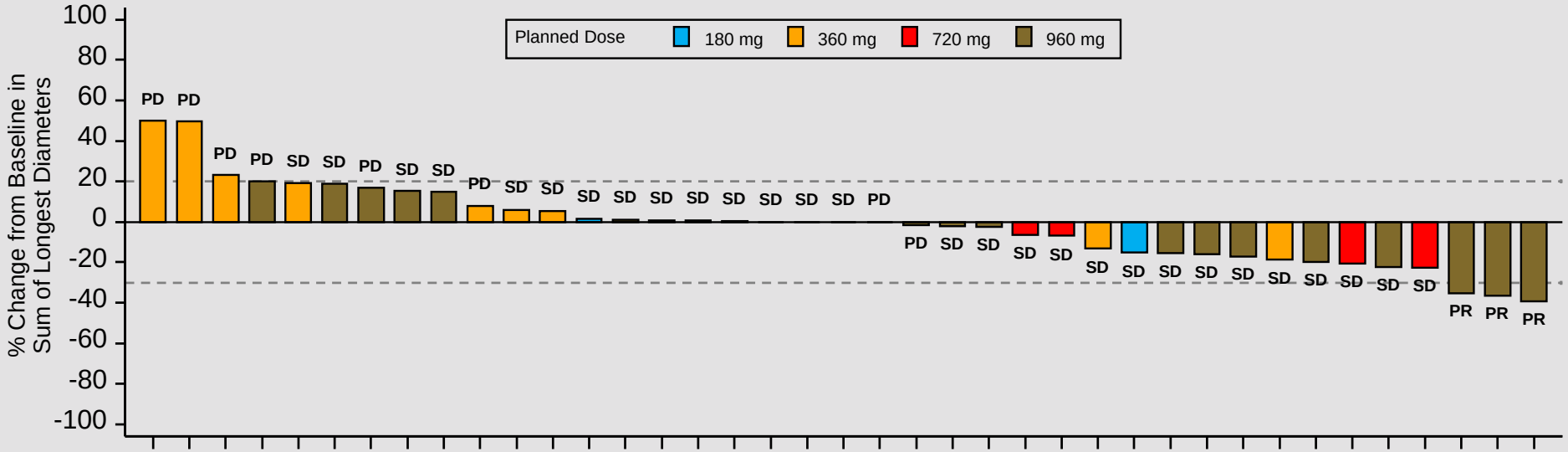
+: censored value; NE: not evaluable.



Number of patients at risk

All Dose	42	40	29	19	6	3	0
960 mg	25	25	17	9	0		

CRC: TUMOR BURDEN CHANGE FROM BASELINE



Three patients are not included in the graph due to missing postbaseline tumor data (1 PD, 1 SD, 1 not done with clinical progression)

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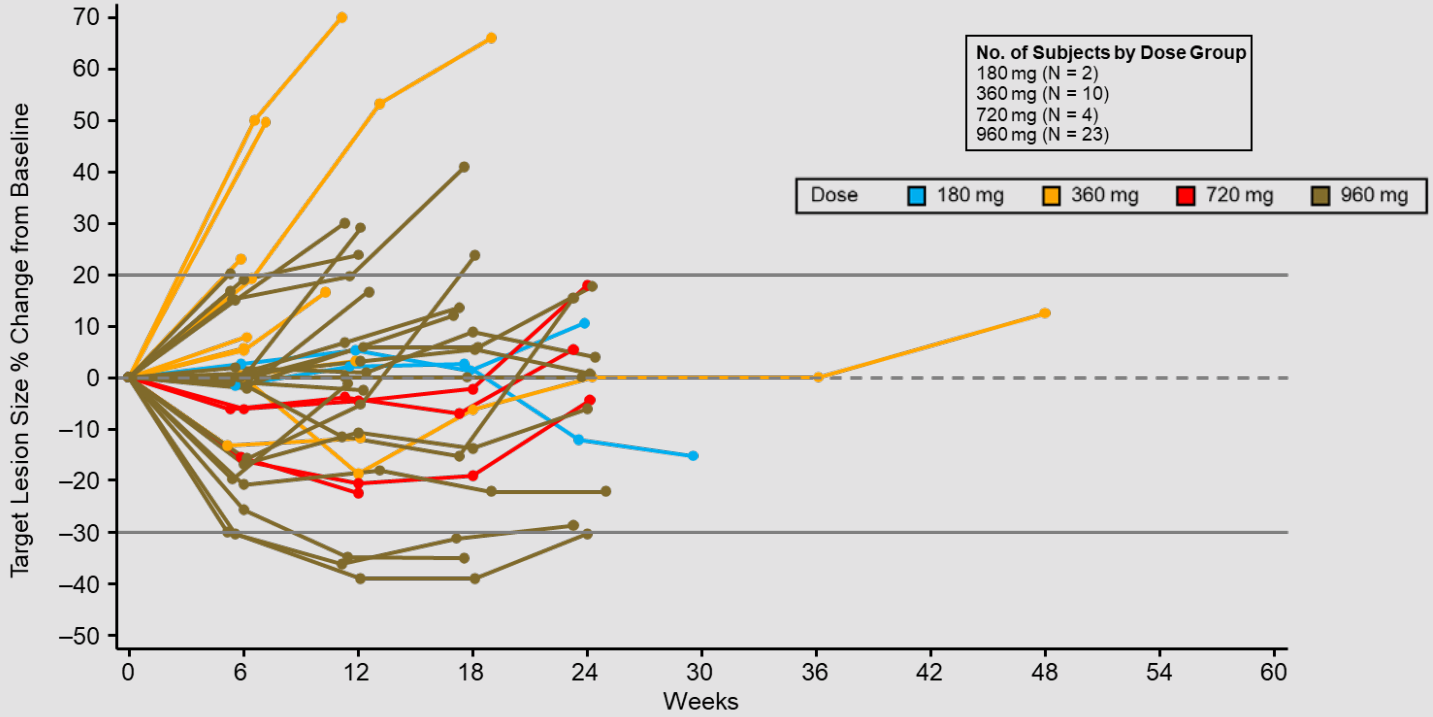
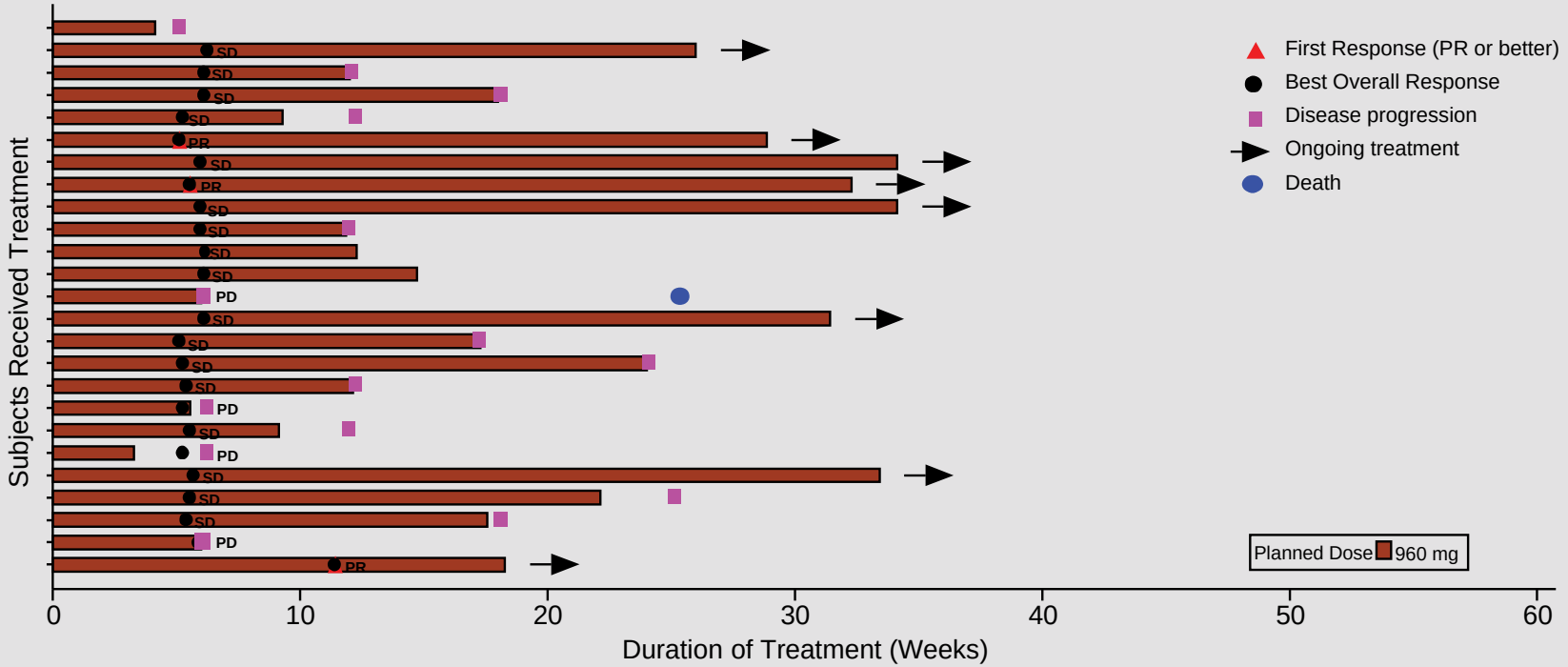
CRC: TUMOR BURDEN CHANGE FROM BASELINE OVER TIME



Three patients are not included in the graph due to missing postbaseline tumor data (1 PD, 1 SD, 1 not done with clinical progression)

CRC: TIME TO RESPONSE AND TREATMENT OVER TIME



CODEBREAK 100: PHASE 1 STUDY OF AMG 510, A NOVEL KRAS^{G12C} INHIBITOR, IN PATIENTS WITH ADVANCED SOLID TUMORS OTHER THAN NON–SMALL-CELL LUNG CANCER (NSCLC) AND COLORECTAL CANCER (CRC)

David S. Hong, MD[1], James C. Kuo, MBBS, FRACP[2], Adrian Sacher, MD[3], Fabrice Barlesi, MD, PhD[4], Benjamin Besse, MD, PhD[5], Yasutoshi Kuboki, MD[6], Grace K. Dy, MD [7], Vikas Dembla, MD [8], John C. Krauss, MD[9], Timothy F. Burns, MD, PhD [10], June Kim, PhD[11], Haby Henary, MD[11], Gataree Ngarmchamnanrith, MD[11], Bob T. Li, MD, PhD[12]

[1]MD Anderson Cancer Center, Houston, TX, USA; [2]Scientia Clinical Research, Randwick, AU; [3]Princess Margaret Cancer Centre, University of Toronto, Ontario, Canada; [4]Aix Marseille University, France; [5]Gustave Roussy Institute, Villejuif, France; [6]National Cancer Center Hospital East, Kashiwa, Japan; [7]Roswell Park Comprehensive Cancer Center, Buffalo, NY, USA; [8]Gibbs Cancer Center, Greer, SC, USA; [9]University of Michigan, Ann Arbor, MI, USA; [10]University of Pittsburgh Medical Center (UPMC) Hillman Cancer Center, Pittsburgh, PA, USA; [11]Amgen Inc, Thousand Oaks, CA, USA; [12]Memorial Sloan Kettering Cancer Center, New York, NY, USA

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OTHER TUMORS: BASELINE CHARACTERISTICS

Baseline Characteristics	N = 25
Median age (range) – year	60.0 (40–75)
Female – n (%)	9 (36.0)
ECOG performance at baseline – n (%) 0 1 2	 7 (28.0) 14 (56.0) 4 (16.0)
Prior lines of systemic anticancer therapy – n (%) 1 2 3 > 3 Missing	 4 (16.0) 5 (20.0) 6 (24.0) 9 (36.0) 1 (4.0)
Number of prior lines of anticancer therapy – median (range)	3 (1–4)

Median follow-up: 4.3 (range: 0.1–12.6) months

ECOG: Eastern Cooperative Oncology Group

OTHER TUMORS: PATIENT INCIDENCE OF ADVERSE EVENTS

	All treatment-emergent AEs (TEAEs) N = 25, n (%)	All treatment-related TEAEs N = 25, n (%)
Any grade Grade ≥ 2 Grade ≥ 3 Grade ≥ 4	20 (80.0) 17 (68.0) 15 (60.0) 4 (16.0)	9 (36.0) 4 (16.0) 2 (8.0) 0 (0.0)
Dose limiting toxicity	0 (0.0)	0 (0.0)
Serious AEs	13 (52.0)	1 (4.0)
Fatal AEs	4 (16.0)	0 (0.0)
AEs leading to treatment discontinuation	3 (12.0)	0 (0.0)

- **Treatment-related TEAEs reported in > 1 patients**
  - **Diarrhea (2/25)**
  - **Fatigue (2/25)**
- **Grade 3 treatment-related TEAEs**
  - **Diarrhea (1/25)**
  - **Pneumonia (1/25)**

AE: adverse events

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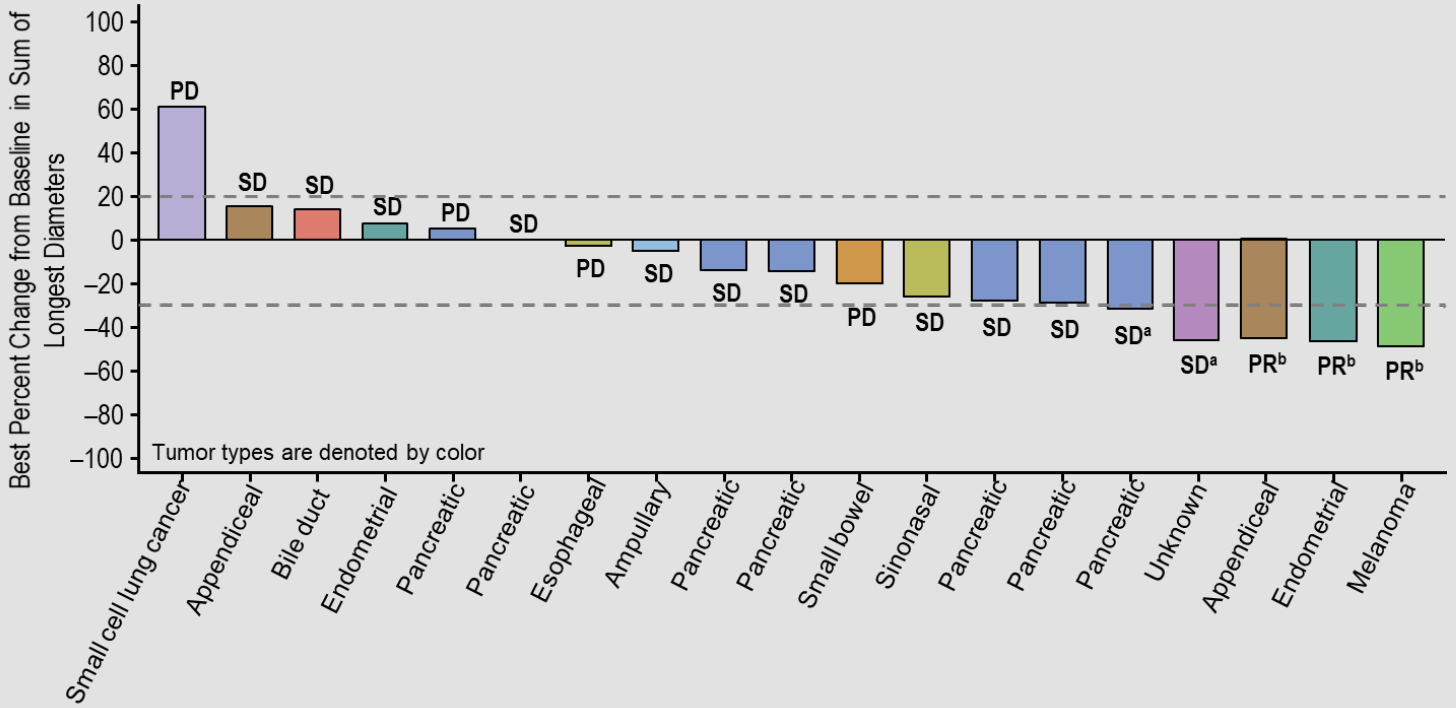
OTHER TUMORS: TUMOR BURDEN CHANGE FROM BASELINE



3 patients are not included due to missing postbaseline tumor data: 2 patients with appendiceal cancer(1 PD, 1 SD) and 1 with pancreatic cancer (PD)
[a]Patients had unconfirmed PR. [b]Of 3 patients with confirmed PR, 1 with appendiceal cancer received 720mg and the other 2 received 960mg.

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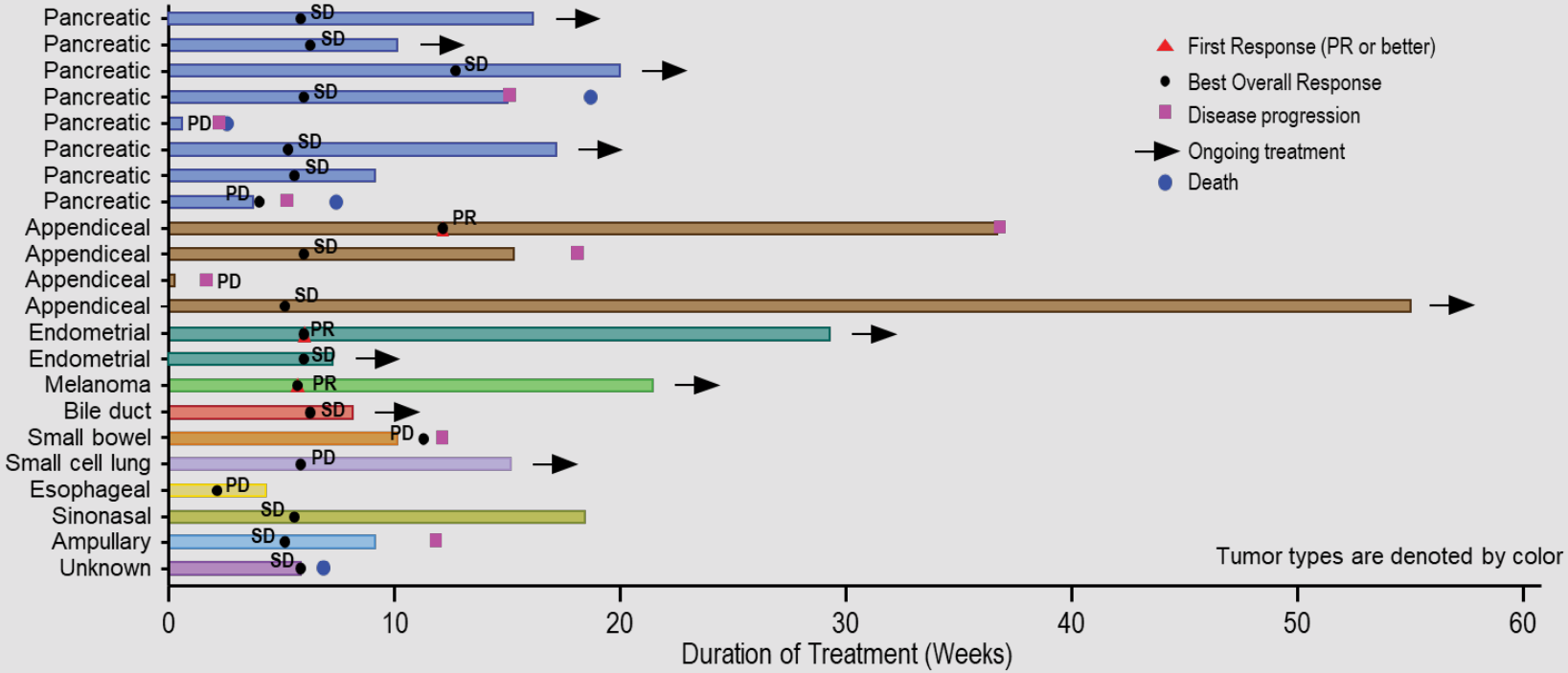
OTHER TUMORS: TIME TO RESPONSE AND TREATMENT OVER TIME



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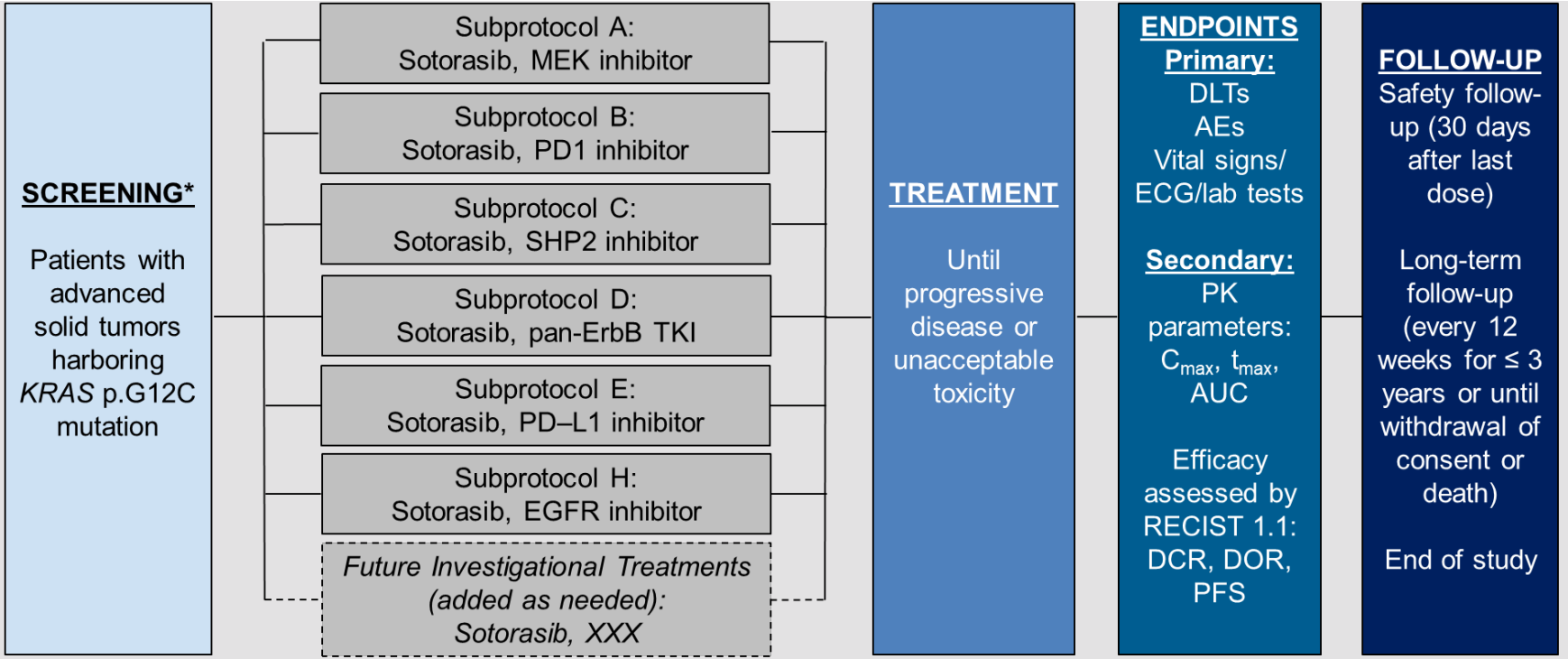
  - **ASCO20: data on 42 CRC patients with 7.9 months follow-up; 25 patients with non-NSCLC, non-CRC solid tumors**

KRAS **= Kirsten rat sarcoma viral oncogene homolog; CRC = colorectal cancer**

PHASE 1B STUDY OF AMG 510 (SOTORASIB) IN COMBINATION WITH OTHER ANTICANCER THERAPIES (CODEBREAK 101)



*If a patient is eligible for multiple subprotocols, the investigator and the patient will determine which subprotocol to enroll the patient in. AUC, area under the plasma concentration-time curve; AE, adverse event; C_{max}, maximum plasma concentration; DCR, disease control rate; DLT, dose limiting toxicity; DOR, duration of response; ECG, electrocardiogram; EGFR; epidermal growth factor receptor; MEK, mitogen-activated protein kinase; PD1, programmed death protein-1; PD–L1; programmed death protein ligand 1; PK, pharmacokinetics; PFS, progression-free survival; RECIST 1.1, Response Evaluation Criteria in Solid Tumors Version 1.1; SHP2, Src homology region 2-containing protein tyrosine phosphatase 2; TKI, tyrosine kinase inhibitor; t_{max}, time reach to C_{max}

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UPDATE ON PRELIMINARY RESULTS FROM PHASE 1 FIRST-IN-HUMAN DOSE ESCALATION STUDY OF AMG 330 IN PATIENTS WITH RELAPSED/REFRACTORY ACUTE MYELOID LEUKEMIA (R/R AML)

Farhad Ravandi, MD[1], Roland Walter, MD, PhD, MS[2], Marion Subklewe, MD[3], Veit Buecklein, MD[3], Mojca Jongen-Lavrencic[4], Peter Paschka[5], Gert J. Ossenkoppele[6], Hagop M. Kantarjian[1], Antreas Hindoyan, PhD[7], Suresh Agarwal, PhD[8], Tian Dai, PhD[7], Sophia Khaldoyanidi, MD, PhD[7], Anthony Stein, MD[9]

[1]Department of Leukemia, The University of Texas MD Anderson Cancer Center, Houston, TX, USA; [2]Clinical Research Division, Fred Hutchinson Cancer Research Center, Seattle, WA, USA; [3]Department of Internal Medicine III, University Hospital, LMU Munich, Munich, Germany; [4]Erasmus University Medical Center Rotterdam, Rotterdam, The Netherlands; [5]Ulm University Medical Center, Ulm, Germany; [6]VU University Medical Center, Amsterdam, The Netherlands; [7]Amgen Inc., Thousand Oaks, CA, USA; [8]Amgen Inc., South San Francisco, CA, USA; [9]Gehr Family Center for Leukemia Research, City of Hope, Duarte, CA, USA

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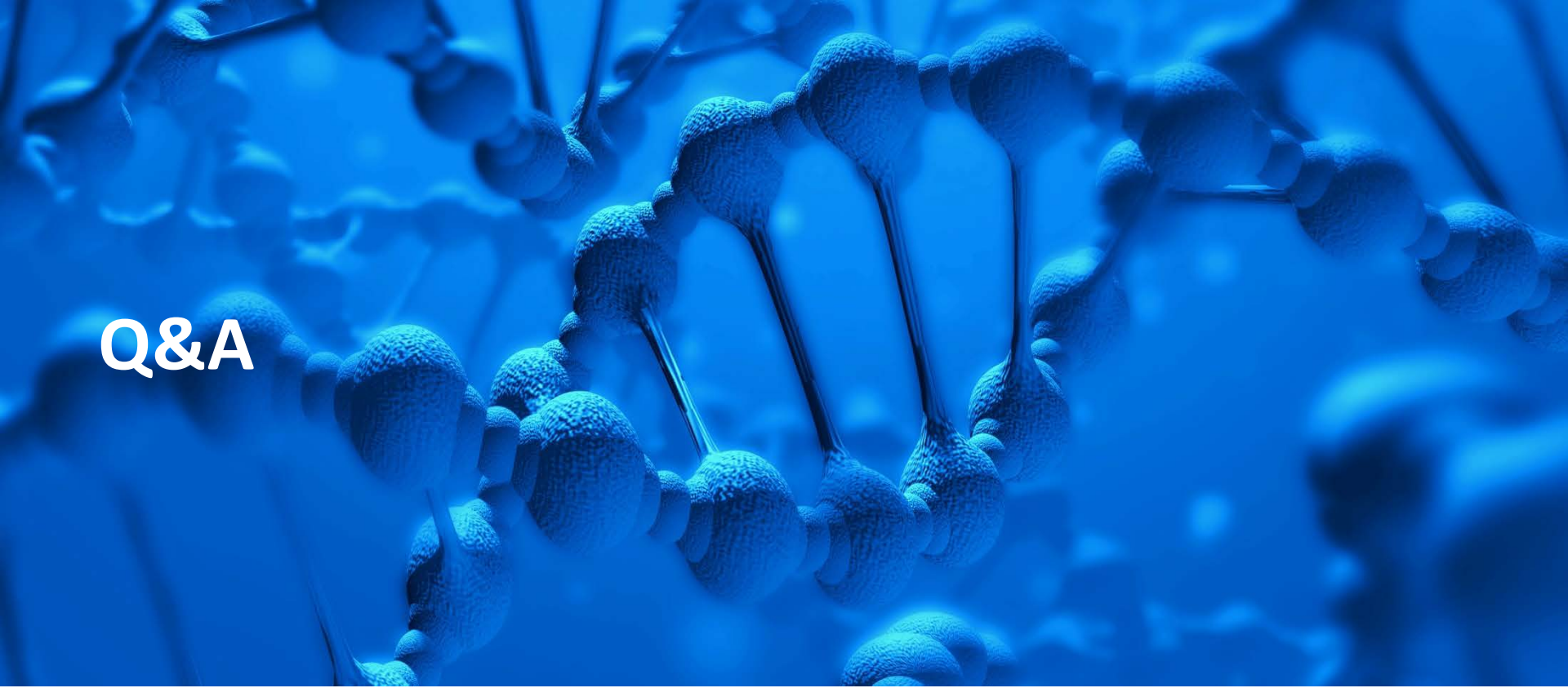

Q&A

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