Hematology/Oncology Update
ESMO 2023

October 24, 2023

AMGEN

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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. An outbreak of disease or similar public health threat, such as COVID-19, and the public and governmental effort to mitigate against the spread of such disease, could have a significant adverse effect on the supply of materials for our manufacturing activities, the distribution of our products, the commercialization of our product candidates, and our clinical trial operations, and any such events may have a material adverse effect on our product development, product sales, business and results of operations. 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 collaborate with or acquire other companies, products or technology, and to integrate the operations of companies or to support the products or technology we have acquired, may not be successful. There can be no guarantee that we will be able to realize any of the strategic benefits, synergies or opportunities arising from the Horizon acquisition, and such benefits, synergies or opportunities may take longer to realize than expected. We may not be able to successfully integrate Horizon, and such acquisition or integration may take longer, be more difficult or cost more than expected. A breakdown, cyberattack or information security breach of our information technology systems 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 and operations may be negatively affected by the failure, or perceived failure, of achieving our environmental, social and governance objectives. The effects of global climate change and related natural disasters could negatively affect our business and operations. Global economic conditions may magnify certain risks that affect our business. 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, any 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

David Reese, MD
Exec. VP, Research & Development, Amgen
Introduction ● BLINCYTO® ● Concluding Remarks

Taofeek Owinikoko, MD, PhD
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Tarlatamab

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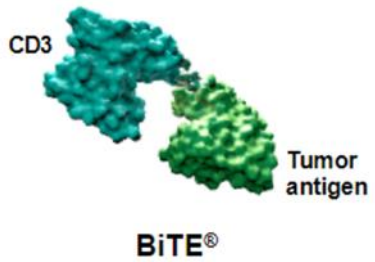
Ch. of the Dept. of Medical Oncology,
Sidney Kimmel Medical Coll., 2
Xaluritamig

Jean-Charles Soria, MD
Sr. VP, Global Development Oncology, Amgen
AMG 193 ● LUMAKRAS® ● Bemarituzumab

ONCOLOGY EFFORTS FOCUSED ON HIGH CONVICTION TARGETS, DIFFERENTIATED THERAPIES AND LARGE EFFECT SIZE

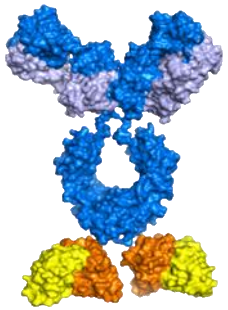
IMMUNO-ONCOLOGY

T-Cell Engagers



Inflame: T-cell mediated killing to eradicate tumors

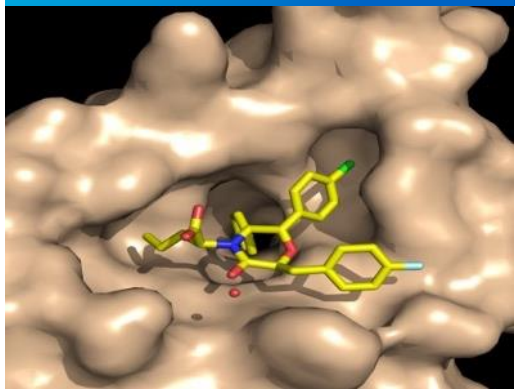
Novel IO



Enhance: Next generation bi-functionals

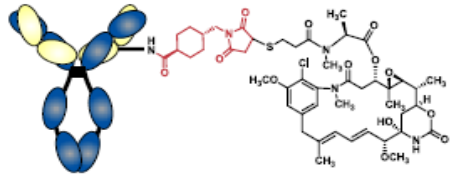
PRECISION ONCOLOGY

Cell Intrinsic Targets



Debulk: Cell-intrinsic therapeutics

Antibody-Drug Conjugates



Debulk: Combines the specificity of mAb with potency of payloads

Combinations required for durable responses in most settings

BiTE® = bispecific T-cell engager; CD3 = cluster of differentiation 3; IO = immuno-oncology; mAb = monoclonal antibody.

AMGEN'S ONCOLOGY PORTFOLIO IS POISED TO POTENTIALLY ALTER THE NATURAL HISTORY OF DISEASE ACROSS MULTIPLE CANCERS

Broad portfolio anchored in first-in-class and best-in-class medicines

- **Bispecific T-cell engagers with promising efficacy results in both hematologic malignancies and solid tumors**
  - Goal is to target the bispecific "sweet spot" of enhanced efficacy and reduced toxicity in early-stage disease with lower tumor burden
  - BLINCYTO® (Approved in B-ALL), tarlatamab (P3 in SCLC), xaluritamig (P1/2 in mCRPC)
- **Precision oncology molecules with potential to treat multiple solid tumors**
  - Goal is to address novel targets in tough-to-treat cancers
  - AMG 193 (P1/2 in solid tumors), LUMAKRAS® (approved in NSCLC, P3 in NSCLC and P3 in CRC), bemarituzumab (P3 in gastric)

B-ALL =B-cell precursor acute lymphoblastic leukemia; P3 = Phase 3; SCLC = small cell lung cancer; P1/2 = Phase 1/2; mCRPC = metastatic castrate-resistant prostate cancer; NSCLC = non-small cell lung cancer; CRC = colorectal cancer

BLINCYTO®

AMGEN

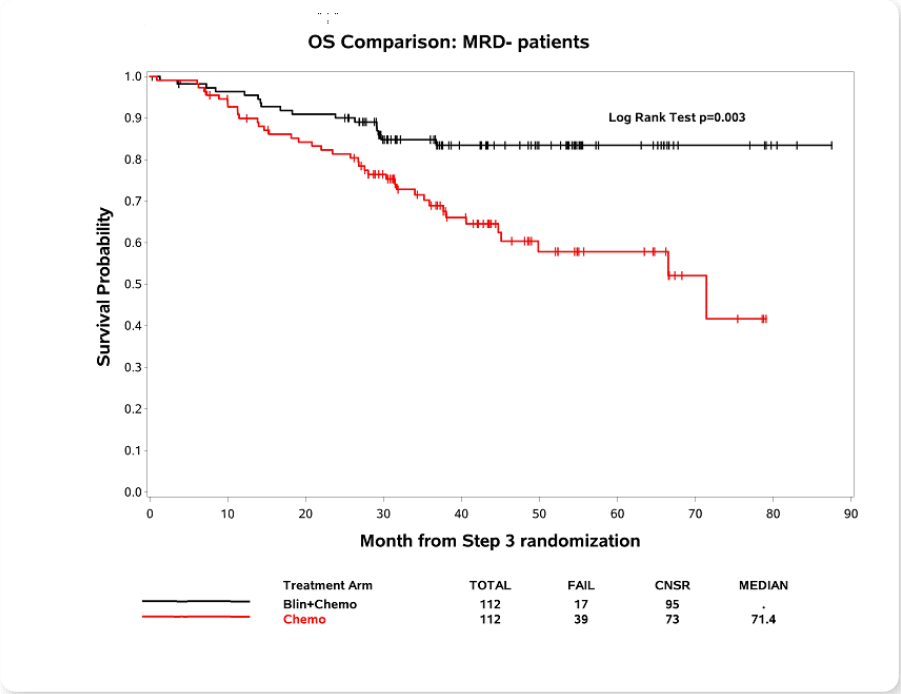
B-CELL PRECURSOR ACUTE LYMPHOBLASTIC LEUKEMIA REMAINS AN AREA OF HIGH UNMET NEED

Disease Progression	Relapsed/Refractory		Adult Minimal Residual Disease Positive	First-Line Adult B-ALL Consolidation (30+)		First-Line Ph- Adult B-ALL (55+) Chemo-sparing
				Ph-	Ph+	
U.S. Drug-Treated Population	~1,600	~850	~600	~1,400	~400	~1,000
BLINCYTO® Status	FDA-Approved[1]	FDA-Approved[1]	FDA-Approved[1]	E1910 Phase 3 study complete, (FDA filing)		Golden Gate Phase 3 study (enrolling)

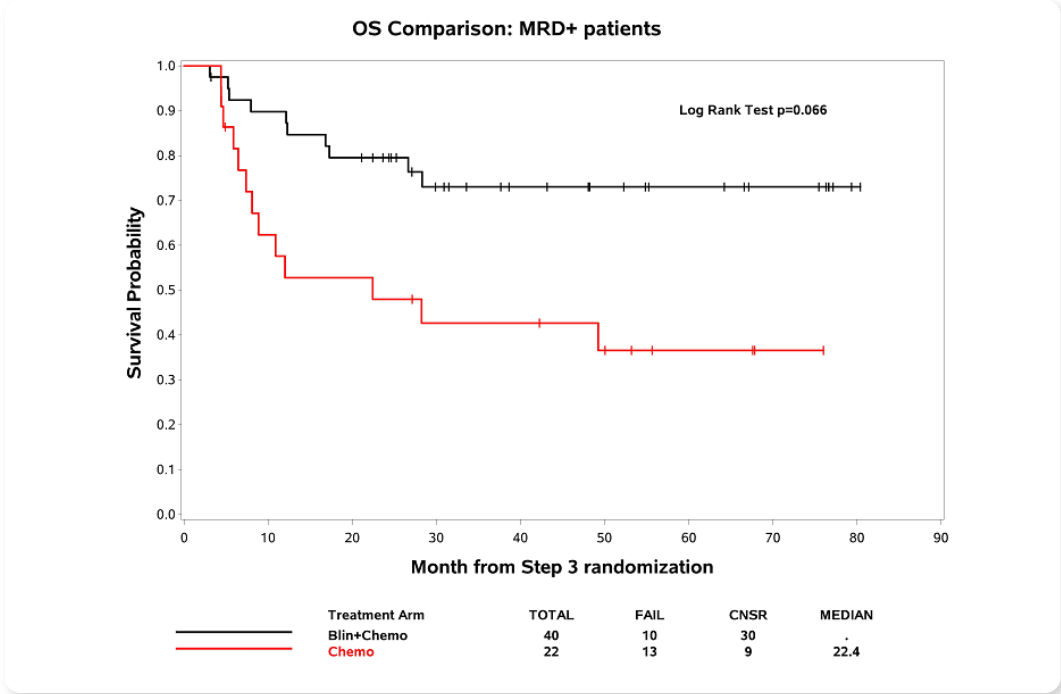
[1] BLINCYTO® USPI
B-ALL = B-cell precursor acute lymphoblastic leukemia; Ph- = Philadelphia chromosome negative; Ph+ = Philadelphia chromosome positive; FDA = U.S. Food and Drug Administration.
Epi source: CfOR Epi forecast model 2022 | Overlap exist in first-line adult patient populations.

BLINCYTO® COMBINED WITH CHEMOTHERAPY DEMONSTRATED AN OVERALL SURVIVAL ADVANTAGE IN FIRST-LINE B-ALL CONSOLIDATION IN THE PHASE 3 ECOG-ACRIN & NCI E1910 TRIAL

Overall Survival Comparison: MRD negative patients



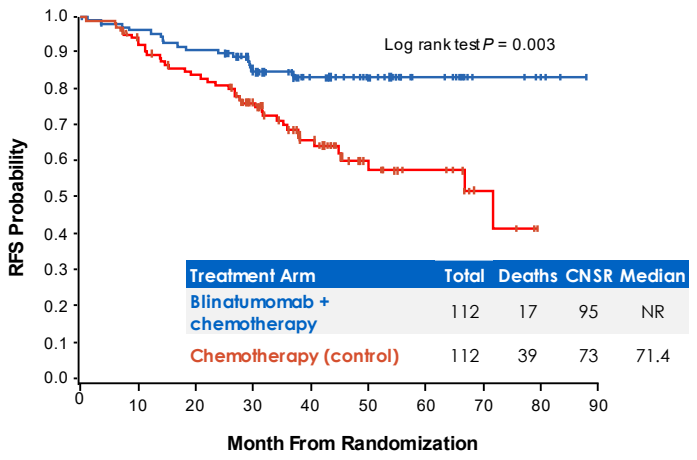
Overall Survival Comparison: MRD positive patients



B-ALL =B-cell precursor acute lymphoblastic leukemia; ECOG-ACRIN = Eastern Cooperative Oncology Group and the American College of Radiology Imaging Network; NCI =National Cancer Institute; MRD = minimal residual disease; OS = overall survival; Blin = BLINCYTO®; CNSR = censor.

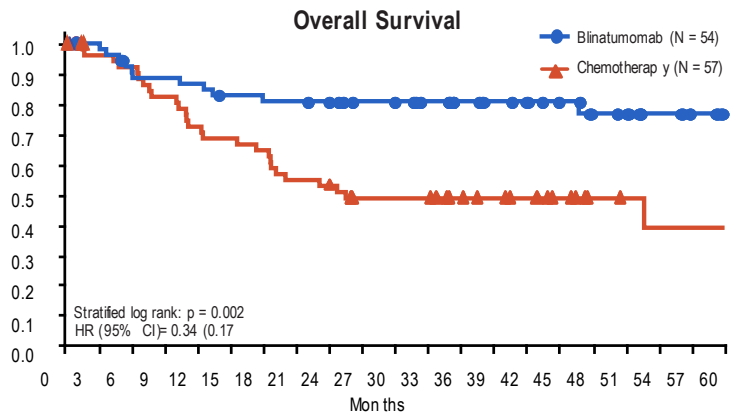
BLINCYTO® DEMONSTRATED PROMISING OVERALL SURVIVAL BENEFIT ACROSS MULTIPLE SUBGROUPS IN FIRST-LINE B-ALL

Adults
ECOG-ACRIN, NCI E1910 Study[1]

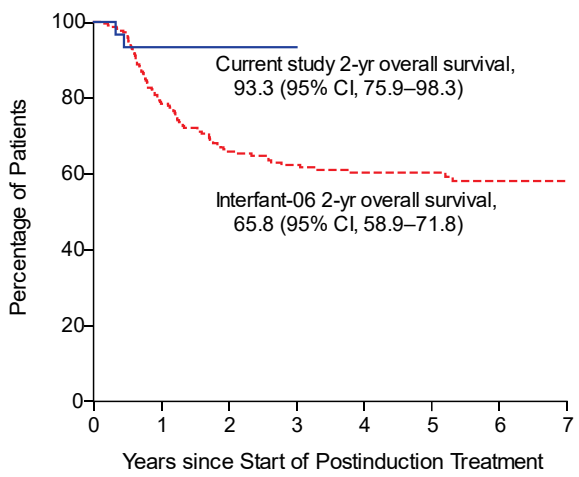


Treatment Arm	Total	Deaths	CNSR	Median
Blinatumomab + chemotherapy	112	17	95	NR
Chemotherapy (control)	112	39	73	71.4

Pediatrics
Amgen 20120215 Study[2]



Infants
Interfant BLINCYTO® Study vs. Interfant-06 Historical Control[3]

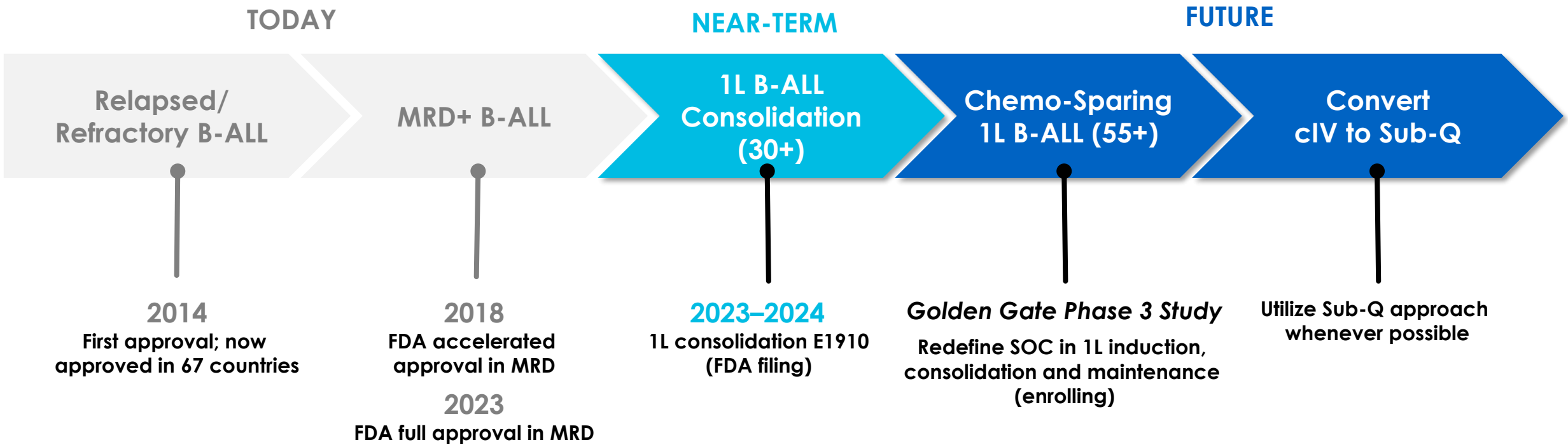


Greater benefit in settings of lower disease burden with potential for improved tolerability

B-ALL =B-cell precursor acute lymphoblastic leukemia; ECOG-ACRIN = Eastern Cooperative Oncology Group and the American College of Radiology Imaging Network; NCI =National Cancer Institute; CNSR = censor.

1. Litzow MR, et al. Oral presented at: American Society of Hematology Annual Meeting; December 10-13, 2022; New Orleans, LA. 2. Locatelli F, et al. Poster presented at: European Hematology Association Congress; June 9-12, 2022; Vienna, Austria. 3. Van der Sluis IM, et al. N Engl J Med. 2023 Apr 27;388(17):1572-1581.

LONG-TERM ASPIRATION IS TO EXPAND BLINCYTO® ACROSS ALL SUBSETS OF B-ALL AND TO UTILIZE SUB-Q DELIVERY WHERE POSSIBLE



B-ALL =B-cell precursor acute lymphoblastic leukemia; Sub-Q = subcutaneous; MRD = minimal residual disease; 1L = first-line; cIV = continuous intravenous infusion; FDA = U.S. Food and Drug Administration; SOC = standard of care.

Tarlatamab

SMALL CELL LUNG CANCER IS ONE OF THE MOST AGGRESSIVE SOLID TUMORS WITH SIGNIFICANT UNMET NEED

Extensive Stage →

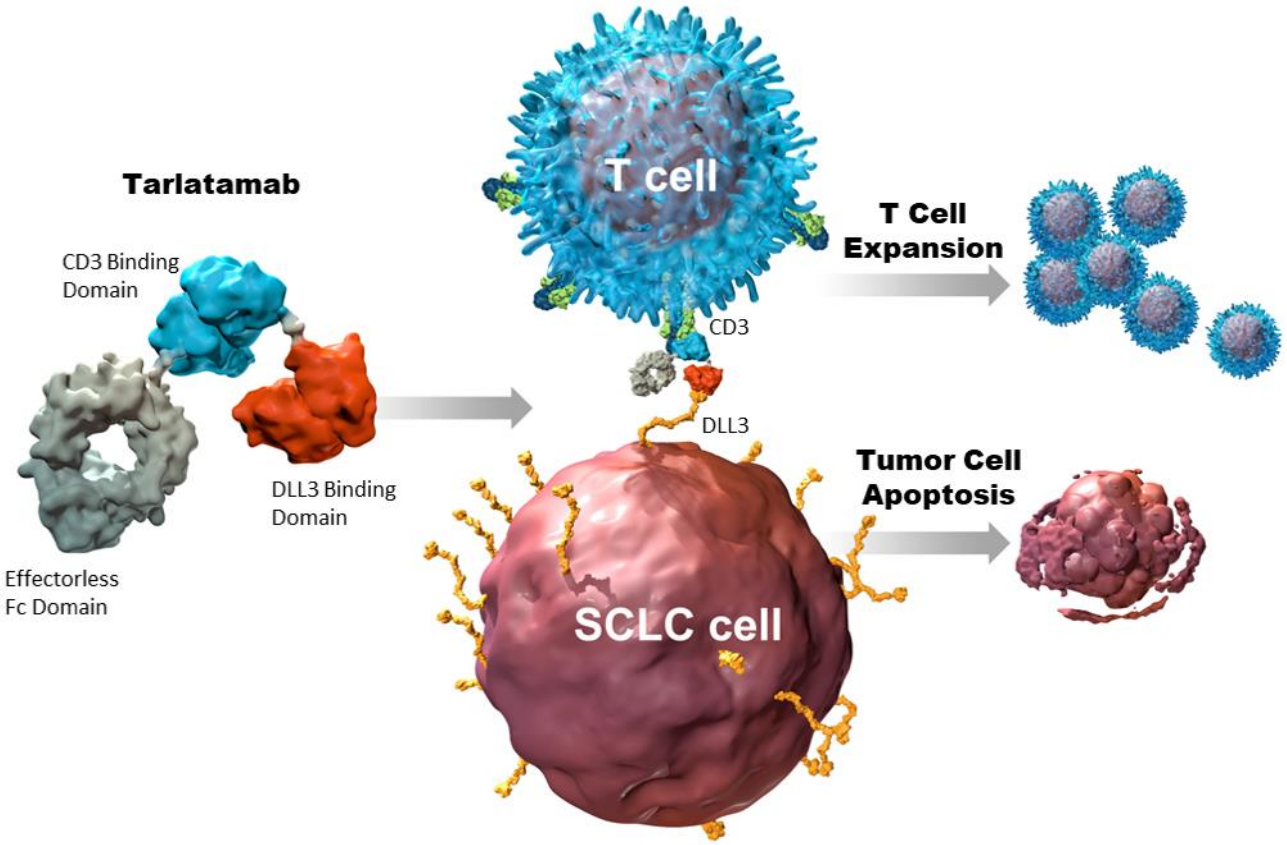
Disease Progression	Third-Line	Second-Line	First-Line Induction and Maintenance	Limited Stage
U.S. Drug-Treated Population	~2.0–3.5K	~8–10K	~21–23K	~5K
Current Efficacy Parameters	No approved SOC	OS: 5.8-9.3m PFS ~3m DOR: 3.3-5.3m	OS: 12.3–12.9m PFS 5.1–5.2m	OS: 25–30m PFS 14.3–15.4m
Clinical Study	DeLLphi-301 Phase 2 Monotherapy			

SOC = standard of care; OS = overall survival; PFS = progression-free survival; DOR = duration of response.

Drug Treated Patient share source: US new patient share based on IQVIA LAAD (Full year 2022); Treated: Enviza. Based on Drug Tx rate ~80% from Cerner (CfOR 2023) – A proportion of patients undergo surgery, radiotherapy. Source: CfOR Epi Forecast for CTRS, 2020 | Tx Rate (2022 → 2030): **1L:** 75% → 85%; **2L:** 75% → 85%; **3L:** 65% → 70%.

* LS-SCLC: Progression based on Cerner Enviza (CancerMPact 2022). A large % of SCLC patients will be diagnosed with ES-SCLC when first diagnosed.
^ Progression applied to exclude deceased and long-term remission patients. These patients get no further therapy.

TARLATAMAB, a BiTE® Immunotherapy Targeting DLL3 in SCLC



- Binds to both DLL3 on SCLC cells and CD3 on T-cells, leading to T- cell mediated cancer cell lysis[1]
- Tarlatamab showed encouraging data in the potentially registrational Phase 2 DeLLphi-301 study in patients with previously treated advanced SCLC[2]

BiTE® = bispecific T-cell engager; DLL3 - delta-like ligand 3; SCLC = small cell lung cancer; CD3 = cluster of differentiation 3; Fc = fragment crystallizable.

[1]Giffin MJ, et al. *Clin Cancer Res*. 2021; 27:1526–1537.
[2] Paz-Ares L, et al. *J Clin Oncol*. 2023; 41:2893–2903.

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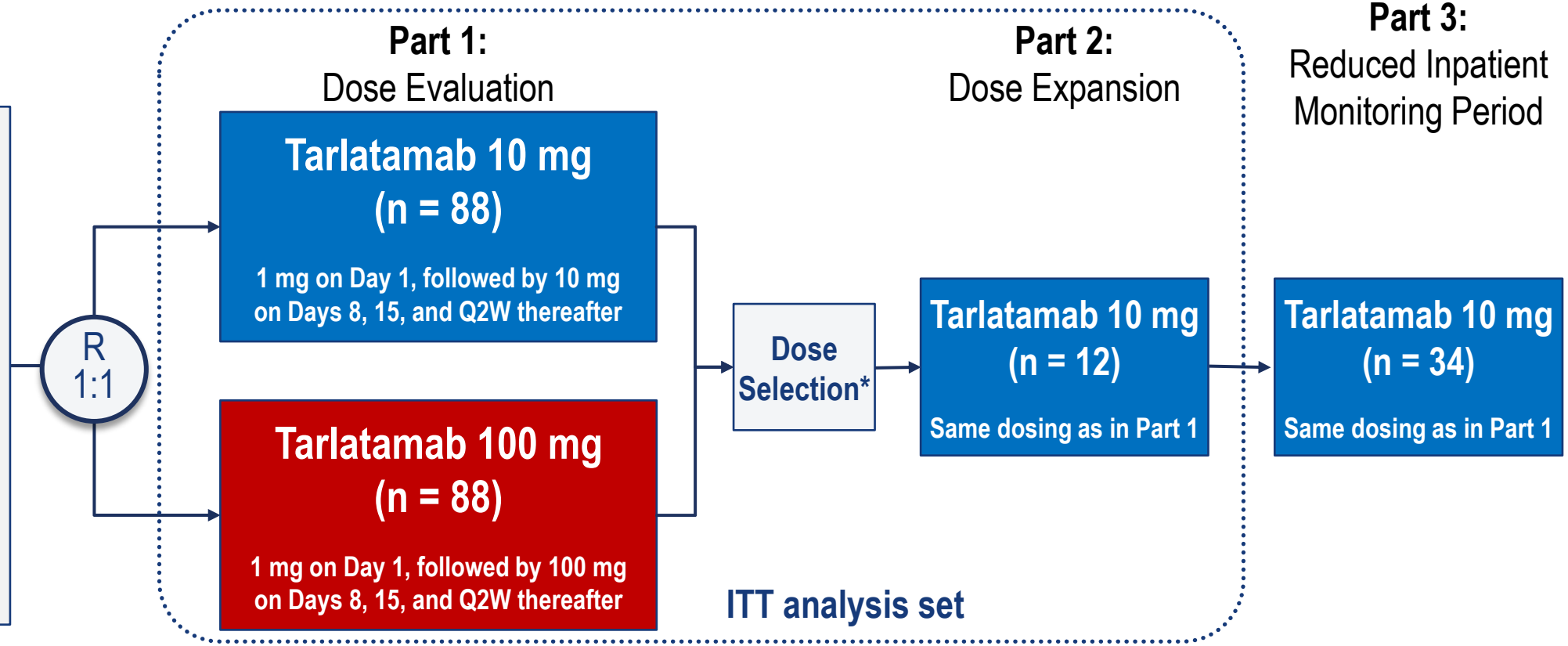
DeLLphi-301 Study Design

Phase 2, open-label study (NCT05060016)

Key Inclusion Criteria

- ES-SCLC
- Previous treatment with ≥ 2 lines (including platinum-doublet)
- ECOG PS 0–1
- Measurable disease
- Treated and stable brain metastases allowed



Primary Endpoint: ORR per RECIST v1.1 by BICR, TEAEs, tarlatamab serum concentrations

Secondary Endpoints Included: DOR, DCR, PFS per RECIST v1.1 by BICR, OS

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*Once 30 patients per dose level had the opportunity to confirm an objective response after the first post-treatment scan or ≥ 13 weeks of follow-up, whichever occurred first.
BICR, blinded independent central review; **DCR**, disease control rate; **DOR**, duration of response; **ECOG PS**, Eastern Cooperative Oncology Group performance status; **ES-SCLC**, extensive stage-small cell lung cancer; **ITT**, intention-to-treat; **ORR**, objective response rate; **OS**, overall survival; **PFS**, progression-free survival; **Q2W**, every 2 weeks; **R**, randomization; **RECIST**, Response Evaluation Criteria in Solid Tumors; **TEAE**, treatment-emergent adverse event.

Baseline Characteristics

	Part 1 + 2 Tarlatamab 10 mg (n = 100)	Part 1 Tarlatamab 100 mg (n = 88)	Part 3 Tarlatamab 10 mg (n = 34)
Median age, years (range)	64 (35–82)	62 (34–80)	66 (49–80)
Male, %	72	70	71
Asian / Black or African American / White,* %	41 / 0 / 58	41 / 0 / 58	6 / 3 / 91
Ever smoker / non-smoker, %	92 / 8	94 / 6	97 / 3
ECOG performance status: 0 / 1, %	26 / 74	27 / 73	29 / 71
Prior lines of therapy, median (range)	2 (1–6)	2 (1–8)	2 (2–6)
2 prior lines of therapy, %	65	55	65
≥ 3 prior lines of therapy, %	33	43	35
Prior anti-PD-(L)1 treatment, %	73	70	82
< 90 days to progression after first-line platinum therapy,† %	28	20	21
Brain / liver metastases, %	23 / 39	36 / 34	12 / 35
DLL3 expression (> 0%), n/N evaluable (%)	80/83 (96)	71/74 (96)	N/A‡

Data cutoff, June 27, 2023. Median follow-up was 10.6 months for tarlatamab 10 mg and 10.3 months for tarlatamab 100 mg.
*No patients of American Indian, Alaska Native, Native Hawaiian, or other Pacific Islander race were enrolled.
†Platinum sensitivity was calculated as end of first-line platinum therapy to date of first progression.
‡DLL3 sample analysis from Part 3 in progress.
DLL3, delta-like ligand 3; **ECOG**, Eastern Cooperative Oncology Group; **N/A**, not available; **PD-(L)1**, programmed death 1 / ligand 1.

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Tarlatamab Anti-Tumor Activity

Outcome	Tarlatamab 10 mg (n = 100)	Tarlatamab 100 mg (n = 88)
Objective response rate, n (%) (97.5% CI)	40 (40) (29, 52)	28 (32) (21, 44)
Complete response	1 (1)	7 (8)
Partial response	39 (39)	21 (24)
Stable disease	30 (30)	27 (31)
Progressive disease	20 (20)	13 (15)
Not evaluable / no post-baseline scan*	10 (10)	20 (23)
Observed duration of response ≥ 6 months, n/N (%)	23/40 (58)	17/28 (61)
Disease control rate, n (%) (95% CI)	70 (70) (60, 79)	55 (62.5) (52, 73)

Tarlatamab 10 mg demonstrated anti-tumor activity in heavily pre-treated SCLC with an objective response rate of 40%

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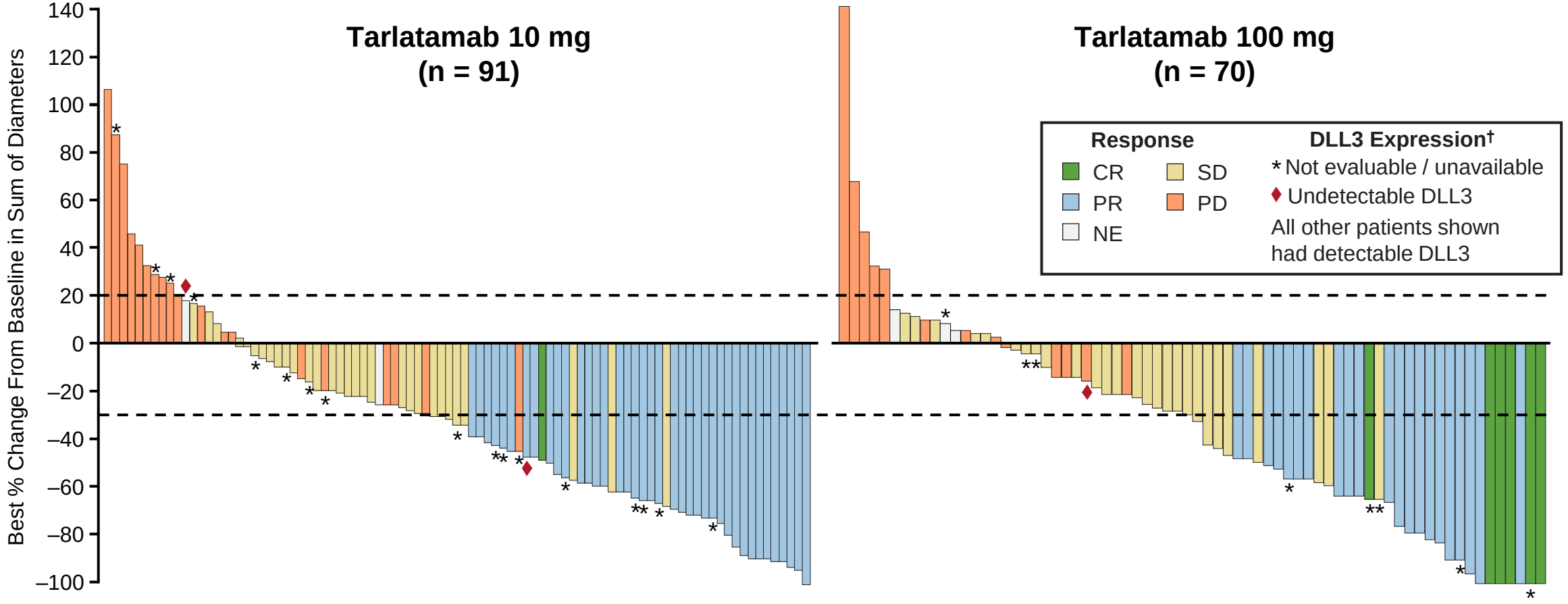
Data cutoff, June 27, 2023. Median follow-up was 10.6 months for tarlatamab 10 mg and 10.3 months for tarlatamab 100 mg. The efficacy analysis set consists of patients in Parts 1 and 2 (N = 188). Part 3 did not have adequate follow-up for response analysis.
*Not evaluable and no post-baseline scan were considered non-responders for response analysis. **SCLC**, small cell lung cancer.

Anti-tumor Activity



Responses were observed regardless of DLL3 expression, as well as in patients without evaluable tumor tissue

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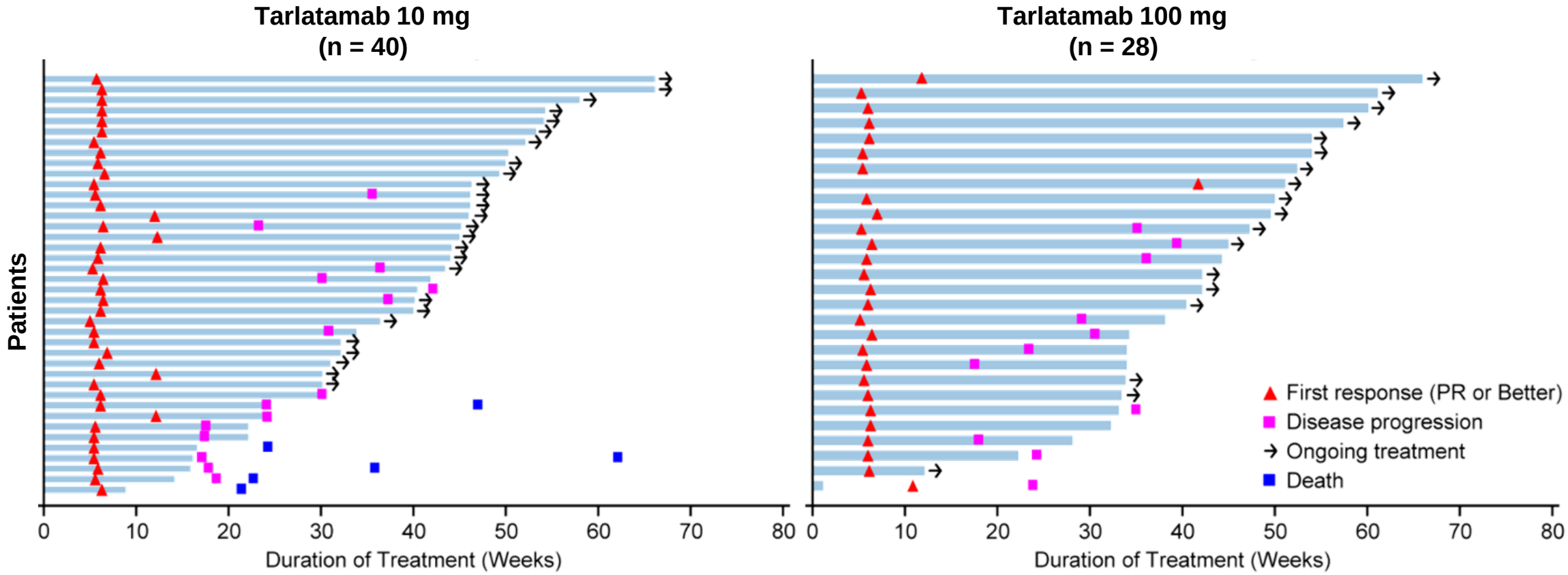
Shown are 91 of 100 patients (tarlatamab 10 mg) and 70 of 88 patients (tarlatamab 100 mg) who had available post-baseline measurements of target lesions.
†DLL3 expression was assessed by immunohistochemistry of tumor tissue samples.
CR, complete response; **DLL3,** delta-like ligand 3; **NE,** not evaluable; **PD,** progressive disease, **PR,** partial response; **SD,** stable disease.

Duration of Response and Treatment



- Median TTR was 1.4 months (range, 1.1–9.6 months), and median DOR was not reached
- Of the 68 responders, the DOR was ≥ 6 months in 40 patients (59%)
- 56% of the responses were ongoing at data cutoff

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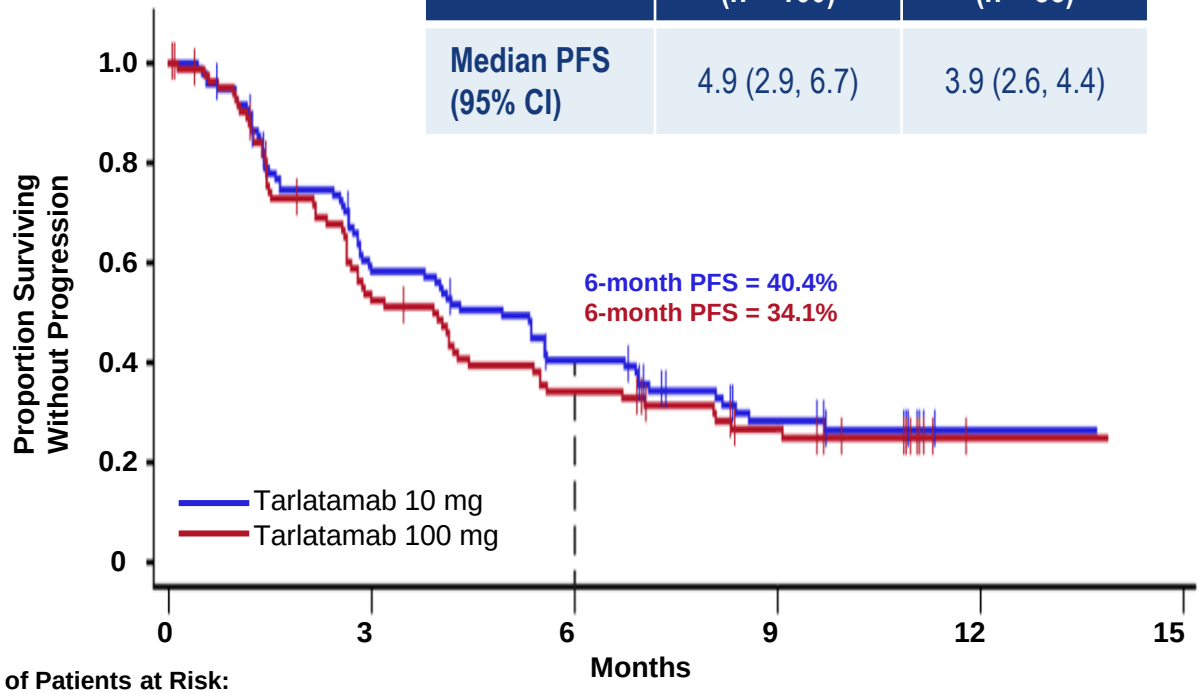
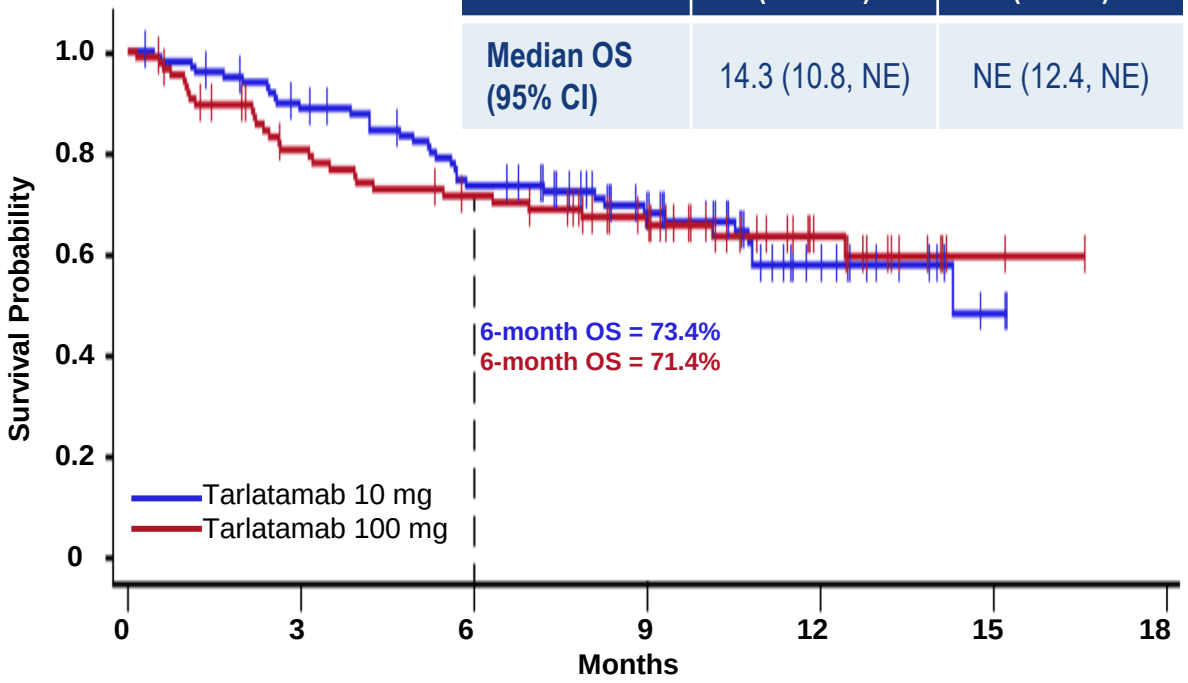
Median follow-up time for DOR, 9.5 months (95% CI; 8.3, 9.7 months).
DOR, duration of response; **PR**, partial response; **TTR**, time to objective response.

PFS and OS

Outcome	Tarlatamab 10 mg (n = 100)	Tarlatamab 100 mg (n = 88)
Median PFS (95% CI)	4.9 (2.9, 6.7)	3.9 (2.6, 4.4)



Outcome	Tarlatamab 10 mg (n = 100)	Tarlatamab 100 mg (n = 88)
Median OS (95% CI)	14.3 (10.8, NE)	NE (12.4, NE)



Number of Patients at Risk:

Tarlatamab 10 mg	100	53	35	18	2	0	100	84	67	44	17	3	0
Tarlatamab 100 mg	88	41	26	15	3	0	88	62	53	39	16	2	0

OS data is not yet mature; at the last follow-up, 57% of patients in the tarlatamab 10 mg group and 51% of patients in the tarlatamab 100 mg group were still alive

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Median follow-up was 10.6 months for tarlatamab 10 mg and 10.3 months for tarlatamab 100 mg.
NE, not estimable; **OS**, overall survival; **PFS**, progression-free survival.

Summary of Adverse Events*

TEAEs, n (%)	Part 1 + 2 Tarlatamab 10 mg (n = 99)	Part 1 Tarlatamab 100 mg (n = 87)	Part 3 Tarlatamab 10 mg (n = 34)
Any grade	96 (97)	87 (100)	34 (100)
≥ Grade 3	57 (58)	56 (64)	22 (65)
Related to tarlatamab, any grade	89 (90)	81 (93)	29 (85)
≥ Grade 3	29 (29)	29 (33)	5 (15)
Fatal	0	0	1 (3)†
Leading to dose interruption/reduction	14 (14)	25 (29)	3 (9)
Leading to discontinuation	4 (4)	3 (3)	0

Most Common TEAEs in ≥ 20% of Patients, n (%)	Part 1 + 2 Tarlatamab 10 mg (n = 99)	Part 1 Tarlatamab 100 mg (n = 87)	Part 3 Tarlatamab 10 mg (n = 34)
CRS	49 (49)	53 (61)	19 (56)
Grade 1–2	49 (49)	48 (55)	18 (53)
≥ Grade 3	0	5 (6)	1 (3)
Decreased appetite	25 (25)	38 (44)	13 (38)
Pyrexia	38 (38)	29 (33)	8 (24)
Constipation	28 (28)	22 (25)	8 (24)
Anemia	26 (26)	22 (25)	9 (26)
Asthenia	20 (20)	21 (24)	10 (29)
Dysgeusia	24 (24)	12 (14)	14 (41)
Fatigue	21 (21)	17 (20)	9 (26)

- **Tarlatamab demonstrated a favorable safety profile, with a low rate of discontinuations due to treatment-related adverse events (TRAEs)**
- **Shorter inpatient monitoring (Part 3) did not alter the safety profile**

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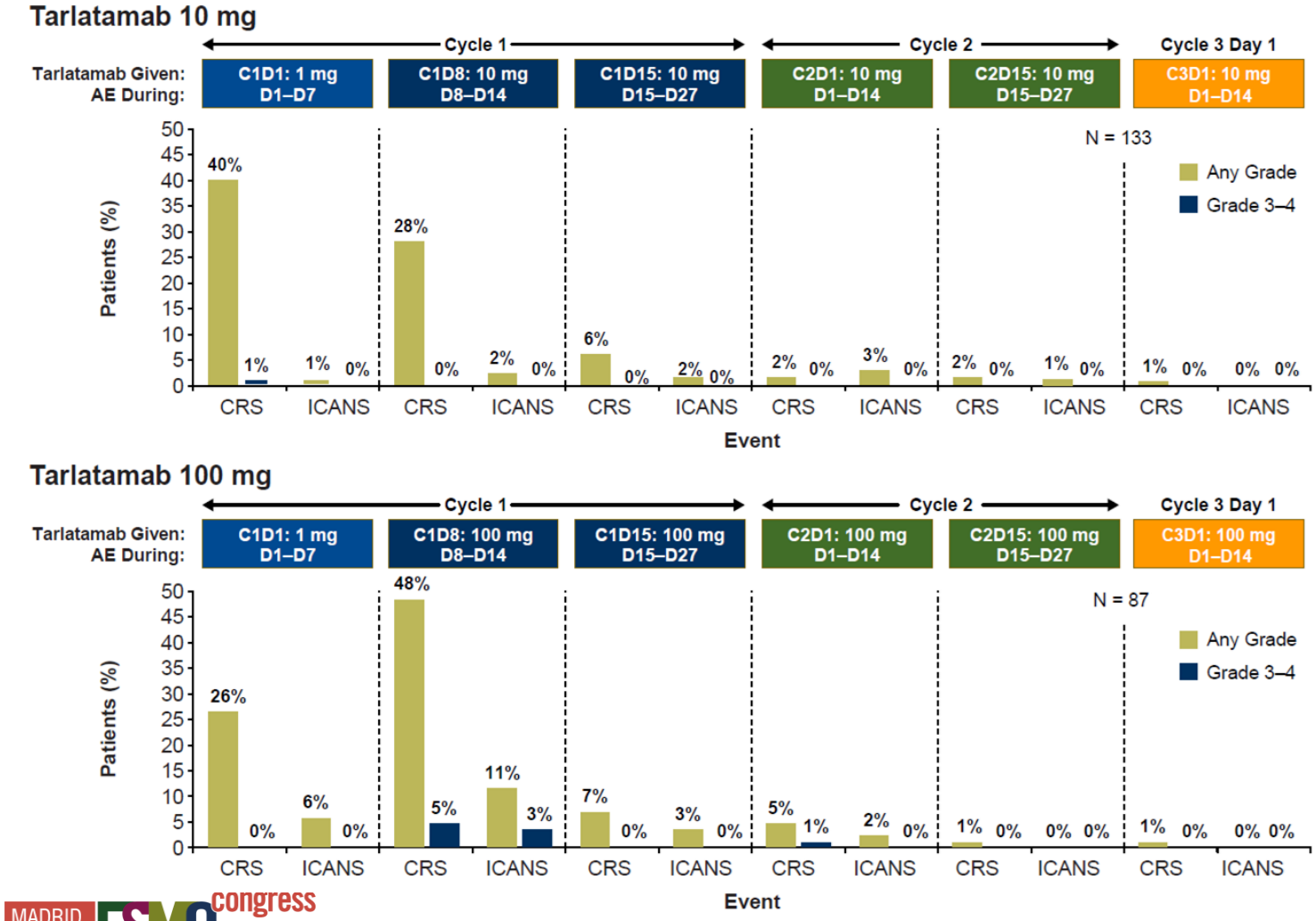
*The safety analysis set includes all patients in Part 1, Part 2, and Part 3 who received at least one dose of tarlatamab (N = 220). †Fatal TRAE was respiratory failure.
CRS, cytokine release syndrome; **TEAE**, treatment-emergent adverse event.

CRS and Immune Effector Cell-Associated Neurotoxicity Syndrome (ICANS)*



- CRS was largely confined to the first or second dose, primarily grade 1–2
- ICANS* occurred infrequently overall and was predominantly observed with tarlatamab 100 mg

Additional Interventions for CRS:

Patients receiving tarlatamab, n (%)	10 mg (n = 133)	100 mg (n = 87)
Tocilizumab	7 (5)	9 (10)
Supplemental oxygen	11 (8)	8 (9)
Vasopressor support	1 (1)	1 (1)

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*ICANS includes associated neurologic events based on a broad search using 61 selected preferred terms from MedDRA version 26.0.
AE, adverse event; **C**, cycle; **CRS**, cytokine release syndrome; **D**, day; **ICANS**, immune effector cell-associated neurotoxicity syndrome.

DeLLphi-301 Conclusions

- Tarlatamab 10 mg demonstrated durable anticancer activity and manageable safety
  - ORR was 40%, observed DOR was ≥ 6 months in 58% of responders, 6-month PFS was 40%, and 6-month OS was 73%
  - The most common TEAE was CRS which primarily occurred in cycle 1, was mostly grade 1–2 and was generally manageable with supportive care
  - Discontinuation of tarlatamab due to TRAEs was low (3%)
  - Shorter inpatient monitoring (Part 3) did not alter the safety profile
  - Tarlatamab 10 mg was the selected dose for further development
- **Rapidly progressing tarlatamab into earlier lines of therapy**

Results support the use of tarlatamab in patients with previously treated SCLC

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CRS, cytokine release syndrome; **DOR**, duration of response; **ORR**, objective response rate; **OS**, overall survival; **PFS**, progression-free survival; **Q2W**, every 2 weeks; **SCLC**, small cell lung cancer; **TEAE**, treatment-emergent adverse event; **TRAE**, treatment-related adverse event.

ADVANCING TARLATAMAB INTO EARLIER SCLC TREATMENT LINES WHERE THERE IS UNMET NEED AND OPPORTUNITY TO SERVE PATIENTS ACROSS THE CONTINUUM

Extensive Stage →

Disease Progression	Third-Line	Second-Line	First-Line Induction and Maintenance	Limited Stage
U.S. Drug-Treated Population	~2.0–3.5K	~8–10K	~21–23K	~5K
Current Efficacy Parameters	No approved SOC	OS: 5.8–9.3m PFS ~3m DOR: 3.3–5.3m	OS: 12.3–12.9m PFS 5.1–5.2m	OS: 25–30m PFS 14.3–15.4m
Tarlatamab Opportunity	No current SOC; potential for transformative DOR	Potential to deliver transformative OS	Largest SCLC population; Potential for transformative OS in combination with SOC	Aspiration of curative Intent
Clinical Study	DeLLphi-301 P2 – Monotherapy (FDA submission in progress)	DeLLphi-304 P3 – Monotherapy vs. SOC (Enrolling)	Studies planned	

SCLC = small cell lung cancer; SOC = standard of care; OS = overall survival; PFS = progression-free survival; DOR = duration of response; P2 = Phase 2; P3 = Phase 3; 1L = first-line.

Xaluritamig

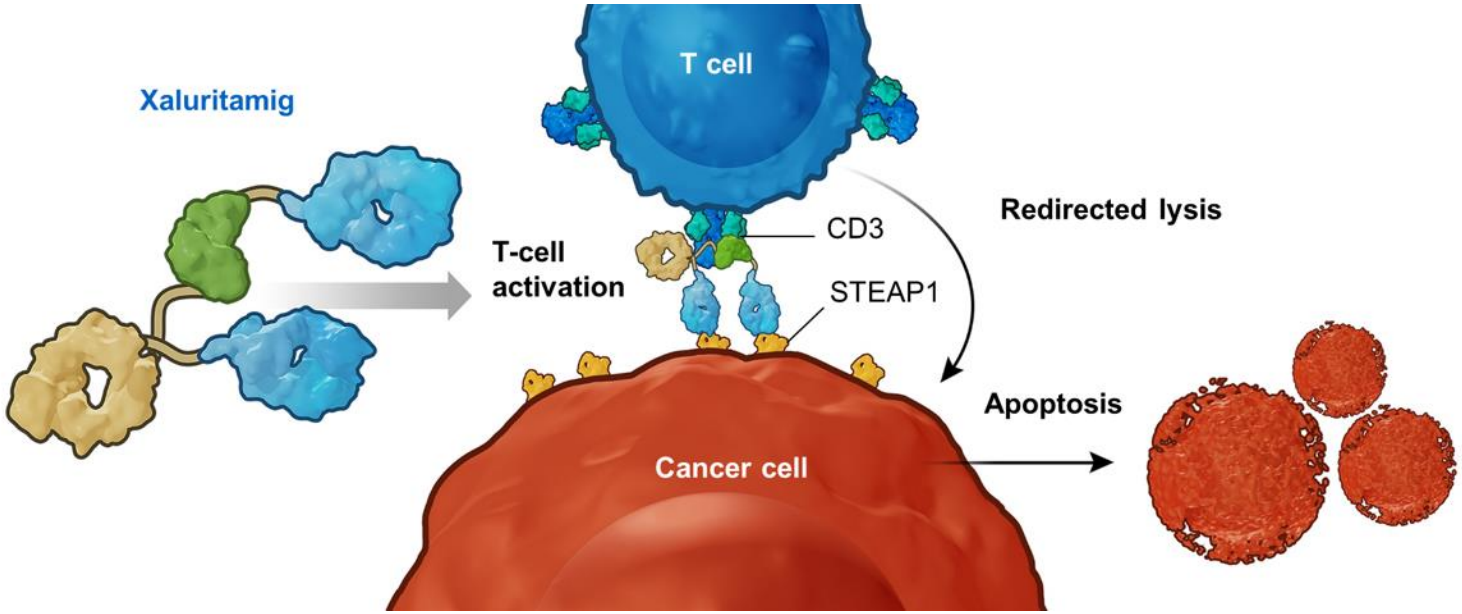
AMGEN

DESPITE RECENT TREATMENT BREAKTHROUGHS IN mCRPC WITH ACCELERATION INTO EARLIER DISEASE, HIGH UNMET NEED REMAINS

Disease Progression	Chemo Refractory (metastatic CRPC)	Chemo Naïve (metastatic CRPC)	Hormonal Therapy (non-metastatic, mCSPC)	Interventional Therapy (non-metastatic, mCSPC)
U.S. Drug-Treated Population	~8K	~38K	~150K	~260K
Current Efficacy Parameters	OS: 11–19m | PFS: 4–8m	OS: 19–24m | PFS: 8–10m	OS: ~50+m | PFS: ~33+m	*Potentially curable with surgery/radiation and hormonal therapy (ADT) for patients at higher risk for recurrence*

mCRPC = metastatic castrate-resistant prostate cancer; mCSPC = metastatic castrate-sensitive prostate cancer; OS = overall survival; PFS = progression-free survival; m = months; ADT = androgen directed therapy.

Xaluritamig is a STEAP1-targeted T-cell engager being evaluated for the treatment of prostate cancer



Xaluritamig is an XmAb® 2+1 T-cell engager designed to facilitate T-cell–mediated lysis of STEAP1-expressing cells[2,3]

- **STEAP1 is a cell surface antigen highly expressed in prostate cancer and associated with poor survival[1,2]**
- **In preclinical studies, xaluritamig showed broad anti-cancer effects in prostate cancer xenograft models[2]**

mAb, monoclonal antibody; mCRPC, metastatic castration-resistant prostate cancer; STEAP1, six transmembrane epithelial antigen of the prostate; CD3 = cluster of differentiation 3.

1. Xu M, et al. Cancers (Basel). 2022;14:4034.
2. Nolan-Stevaux O, et al. Cancer Res. 2020;80(16 suppl):DDT02-03.
3. Li C, et al. J ImmunoTher Cancer. 2020;8:718.

XmAb® is a registered trademark of Xencor, Inc.
Xaluritamig is being developed pursuant to a research collaboration with Xencor, Inc.

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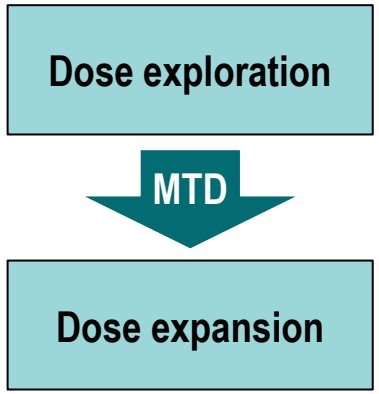
Study design, key eligibility, and baseline demographics

Primary objectives: Safety and tolerability, MTD
Secondary objectives: PK, preliminary anti-tumor activity
Exploratory objectives: PD, immunogenicity

Key inclusion criteria:
- mCRPC refractory to prior novel hormonal therapy and 1–2 taxane regimens*
- ECOG PS 0–1
- Adequate organ function

Key exclusion criteria:
- Histology other than adenocarcinoma
- Active autoimmune disease

Part 1: FIH Monotherapy



A global, first-in-human, open-label study in patients with advanced prostate cancer (NCT04221542)

Patient Characteristics	All cohorts, Part 1 (N = 97)
Age, median (range), years	67 (40, 86)
Race,† n (%)	
White	59 (61)
Asian	32 (33)
Black / African American	5 (5)
ECOG PS 0 / 1, n (%)	45 (46) / 52 (54)
Number of prior lines of therapy,‡ median (range)	4 (1, 9)
≥ 5, n (%)	27 (28)
Prior taxane, n (%)	82 (85)
Prior PSMA-targeting radioligand therapy, n (%)	4 (4)
Baseline PSA, ng/mL, median (range)	113.0 (0.2, 5808.9)
Visceral metastases, n (%)	51 (53)
Liver	19 (37)
Median (range) duration of follow-up, months	8.1 (0.5, 29.2)

Data cutoff, March 28, 2023.
*Patients not eligible or who refused taxanes were allowed without prior taxane treatment.
†One patient (1%) declined to answer. ‡Number of prior lines of therapy do not include androgen deprivation therapy or first-generation androgen receptor deprivation therapy.
ECOG PS, Eastern Cooperative Oncology Group performance status; FIH, first-in-human; mCRPC, metastatic castration-resistant prostate cancer; MTD, maximum tolerated dose; PD, pharmacodynamics; PK, pharmacokinetics; PSA, prostate-specific antigen; PSMA, prostate-specific membrane antigen.

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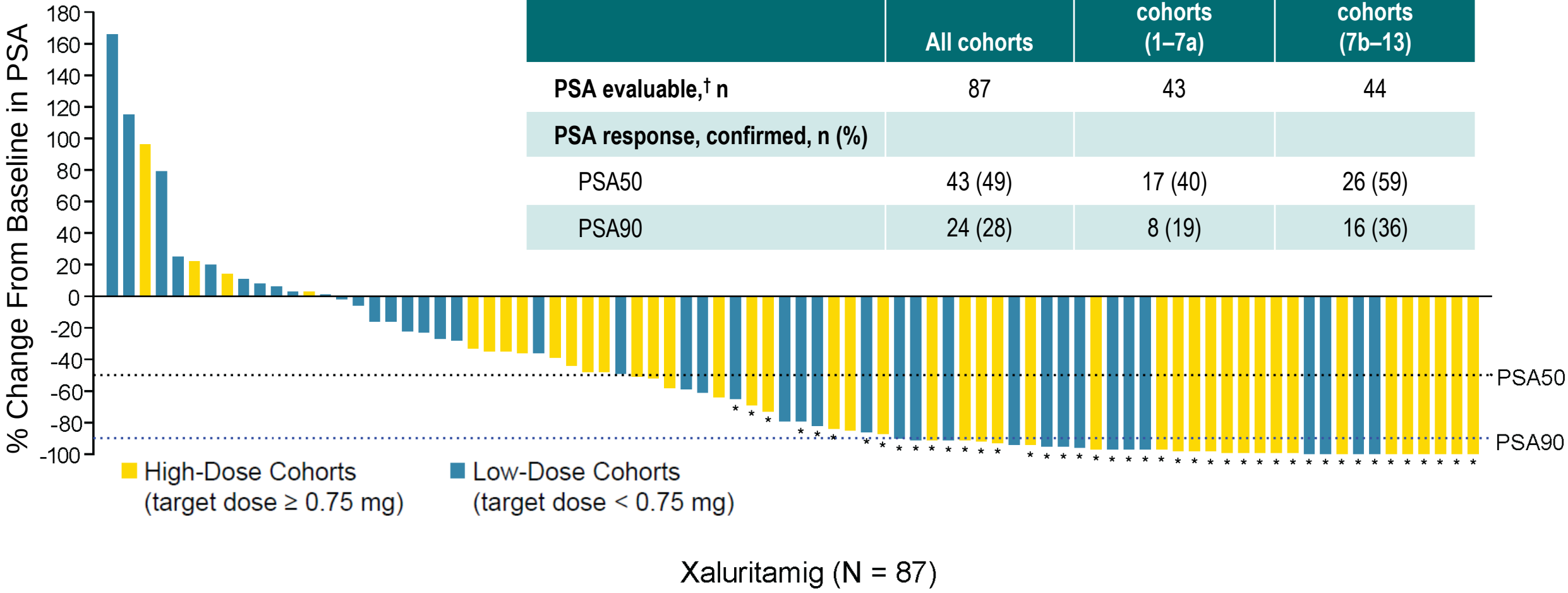
Confirmed PSA responses were observed across cohorts

	All cohorts	Low-dose cohorts (1–7a)	High-dose cohorts (7b–13)
PSA evaluable,† n	87	43	44
PSA response, confirmed, n (%)			
PSA50	43 (49)	17 (40)	26 (59)
PSA90	24 (28)	8 (19)	16 (36)



*Confirmed PSA responders of PSA50 or better.
†10 patients were not PSA evaluable: 6 patients were missing baseline PSA values, and 4 patients did not have sufficient follow-up duration.
PSA, prostate-specific antigen.
Xaluritamig is being developed pursuant to a research collaboration with Xencor, Inc.

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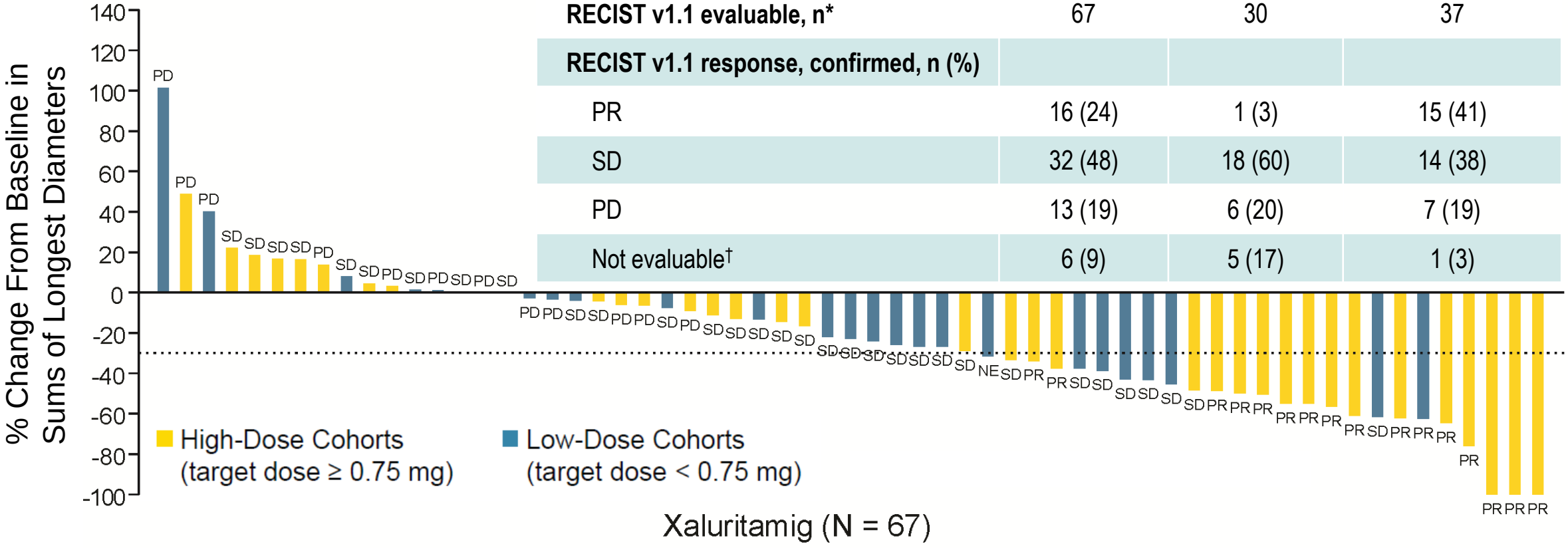
Confirmed RECIST responses occurred more often in high-dose cohorts

	All cohorts	Low-dose cohorts (1–7a)	High-dose cohorts (7b–13)
RECIST v1.1 evaluable, n*	67	30	37
RECIST v1.1 response, confirmed, n (%)			
PR	16 (24)	1 (3)	15 (41)
SD	32 (48)	18 (60)	14 (38)
PD	13 (19)	6 (20)	7 (19)
Not evaluable†	6 (9)	5 (17)	1 (3)



Dashed line indicates 30% reduction in tumor sum of longest diameters from baseline. *Historically, ~40% of mCRPC patients have RECIST-measurable disease.[1,2] †BOR of NE includes 5 patients without post-baseline scans and 1 patient without sufficient follow-up duration prior to post-baseline assessment.

BOR, best overall response; mCRPC, metastatic castration-resistant prostate cancer; NE, not evaluable; PD, progressive disease; PR, partial response; PSA, prostate-specific antigen; RECIST; Response Evaluation Criteria in Solid Tumors; SD, stable disease.

1. Scher HI, et al. *Clin Cancer Res.* 2005;11(14):5223-5232. 2. Lorente D, et al. *Eur Urol Focus.* 2018;4(2):235-244.

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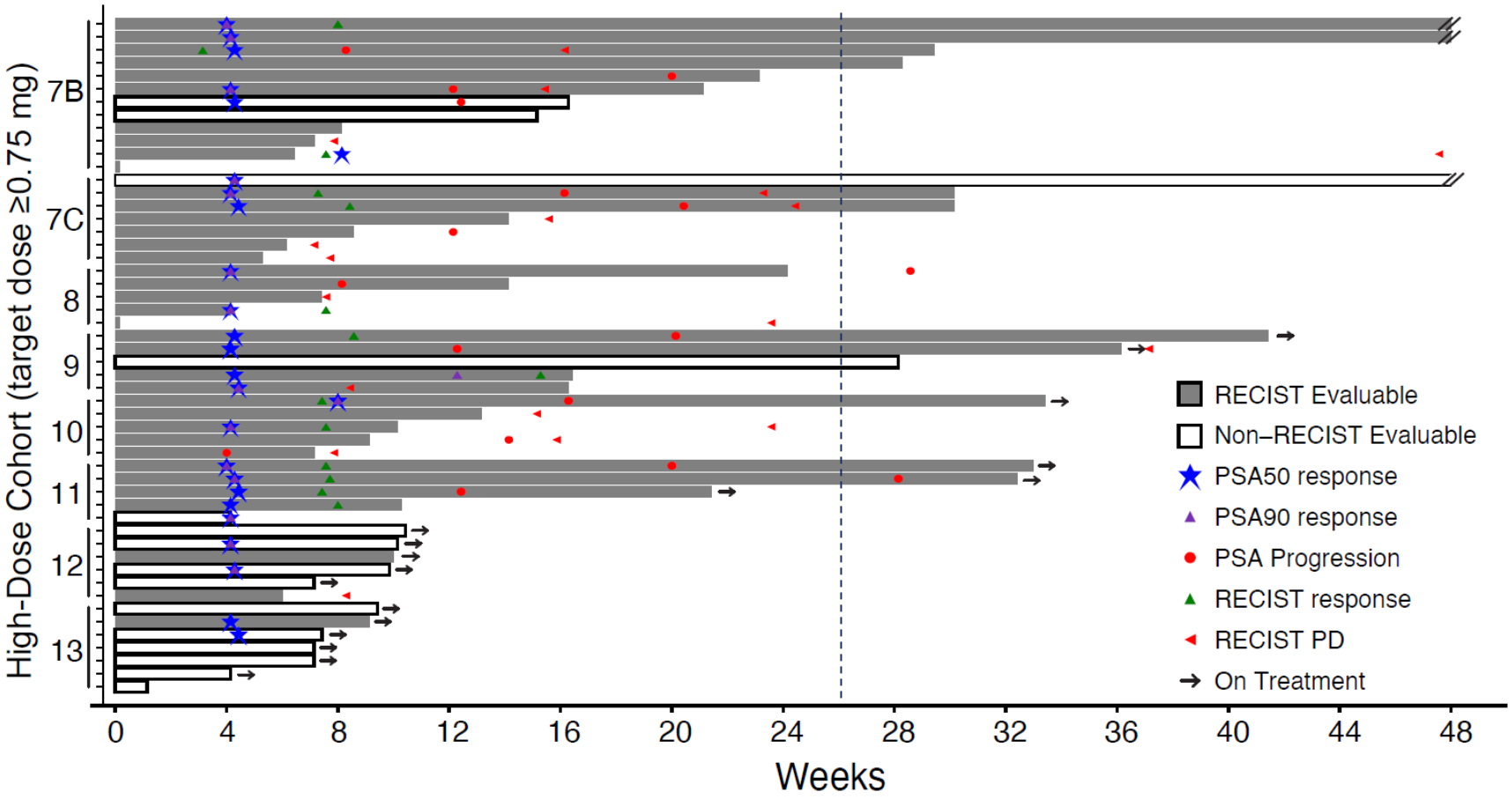
Responses were rapid; preliminary durability encouraging but immature



- Nineteen patients from high-dose cohorts (n = 52) remained on treatment at data cutoff
- Of those, 13 patients from high-dose cohorts remained on treatment for > 6 months

Duration of response*

Median, 9.2
(range, 1.9+ to 17.7+) months

n = 16 (confirmed PR)
10/16 still in response

+ Indicates censored value. *Includes 15 patients from high-dose cohort and 1 patient from low-dose cohort. Duration of response was measured from first evidence of PR/CR to disease progression or death due to any cause, whichever was earlier. Patients whose treatment was ongoing are noted by an arrowhead. Double parallel lines (//) represent patients who have extended beyond 48 weeks: 1 patient is ongoing treatment at 90 weeks, 1 patient is ongoing treatment at 84 weeks, and 1 patient ended treatment at 58 weeks.
CR, complete response; PD, progressive disease; PR, partial response; PSA, prostate-specific antigen; RECIST, Response Evaluation Criteria in Solid Tumors.

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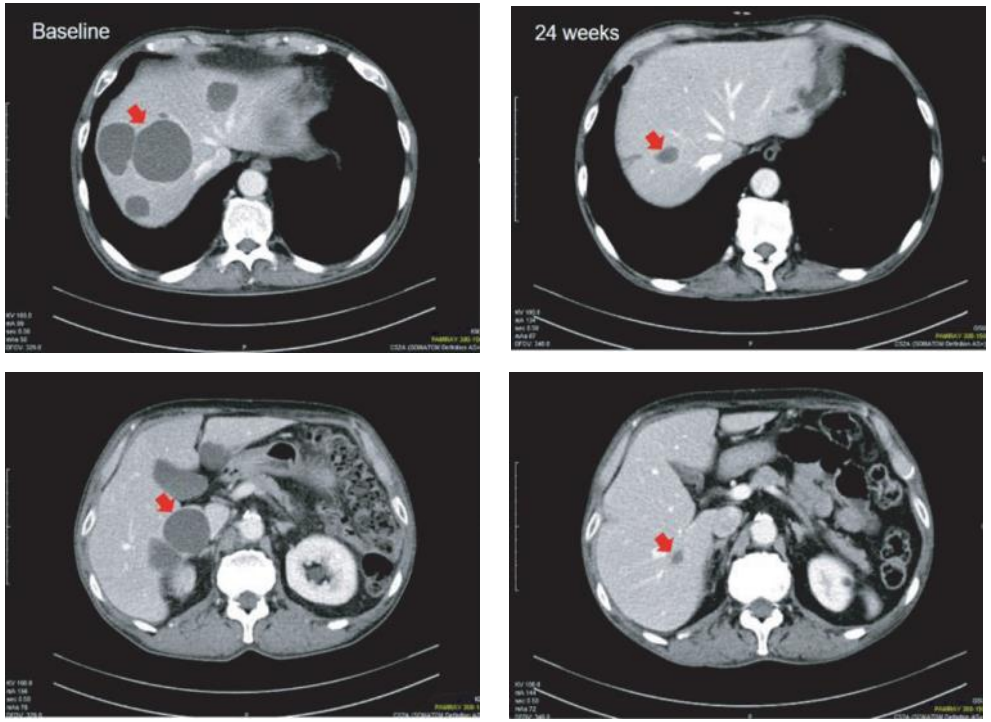
Anti-tumor activity has been observed against both soft tissue and bone disease

CT Scan

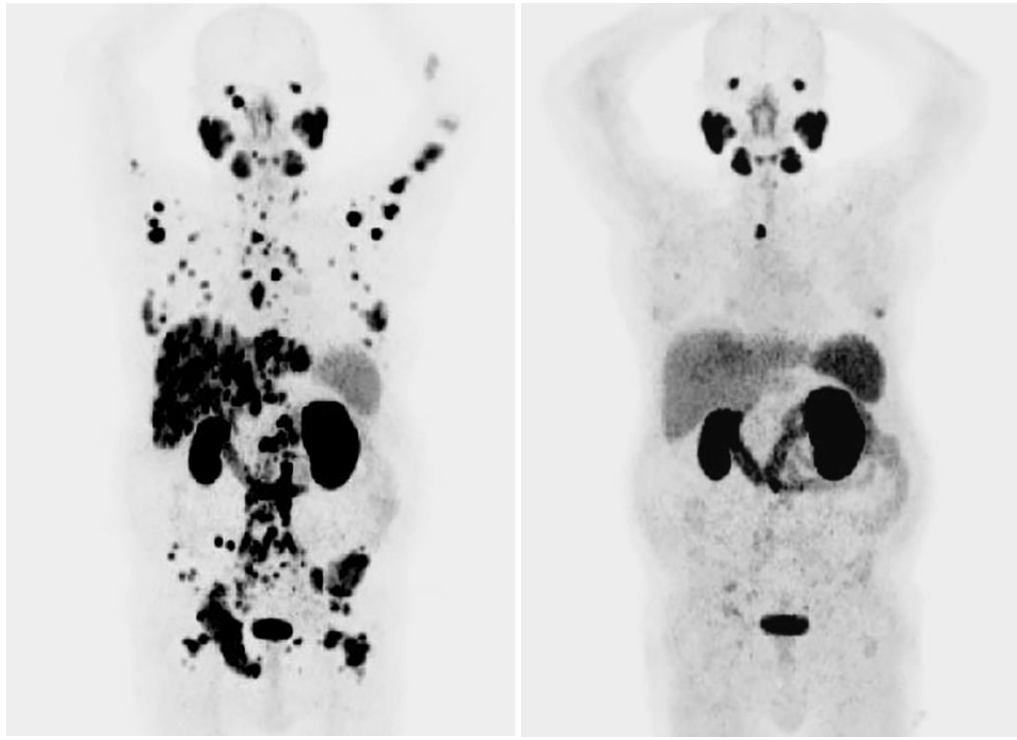
Baseline	Week 24



65-year-old heavily pre-treated patient with mCRPC. Patient was enrolled in cohort 11 and achieved a confirmed RECIST and PSA90 response.

PSMA PET Imaging

Baseline	Week 12



56-year-old heavily pre-treated patient with mCRPC. Patient was enrolled in cohort 12 and achieved a confirmed PSA90 response (not RECIST evaluable).

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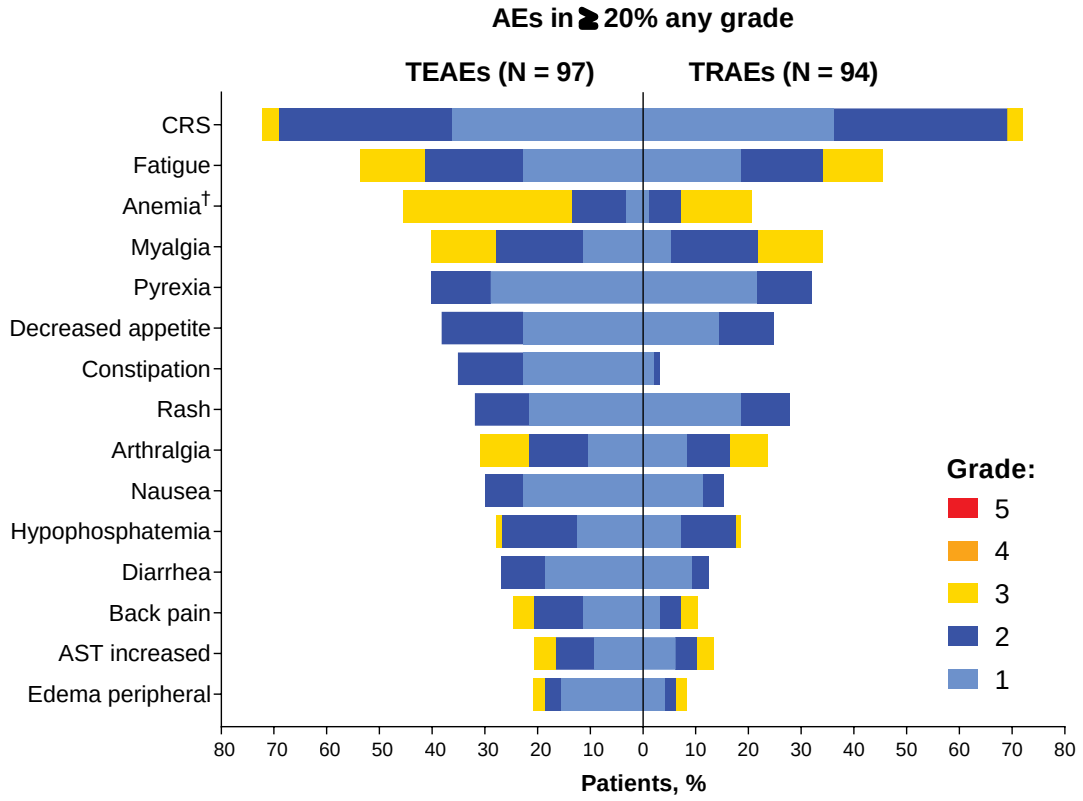
CT, computerized tomography; mCRPC, metastatic castration-resistant prostate cancer; PSA, prostate-specific antigen; PSMA PET, prostate-specific membrane antigen positron emission tomography; RECIST, Response Evaluation Criteria in Solid Tumors.

The safety profile during dose exploration was generally manageable with majority of CRS grade 1/2

Patient Incidence of TEAEs, n (%)	All cohorts (N = 97)
Any TEAE	97 (100)
Grade ≥ 3	74 (76)
Serious	55 (57)
Related to xaluritamig	94 (97)
Grade ≥ 3	53 (55)
Leading to discontinuation from xaluritamig	18 (19)
Leading to xaluritamig dose interruption (missed doses)	46 (47)
Leading to xaluritamig dose reduction	7 (7)
Serious	38 (39)
DLT-evaluable patients, N	**82**
DLTs, n (%)*	20 (24)



- AEs were generally consistent with the MOA and patient population, with no grade 4 / 5 events
- TRAEs of musculoskeletal and connective tissue disorders were reported, with 14% being serious

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*All DLTs were grade 3 and included myalgia (n=3); back pain (n=2); and performance status decrease, encephalopathy, CRS, hypotension, hypoalbuminemia, ALT increase, AST increase, fatigue, stomatitis, arthralgia, fasciitis, pharyngitis, QT prolongation, atrial fibrillation, and oropharyngeal pain (n=1 each). † Twenty-four patients required red blood cell transfusion on study.
AE, adverse event; ALT, alanine aminotransferase; AST, aspartate aminotransferase; CRS, cytokine release syndrome; DLT, dose-limiting toxicity; MOA, mechanism of action; TEAE, treatment-emergent adverse event TRAE, treatment-related adverse event.

Conclusions

- Xaluritamig is the first clinical T-cell engager targeting STEAP1
- MTD was established utilizing step-dosing and pre-medication
  - 1.5 mg IV QW (3-step, D1 0.1 mg / D8 0.3 mg / D15 1.0 mg / D22+ 1.5 mg)
- The safety profile was clinically manageable, with CRS that was generally low grade and primarily in cycle 1
- Observed encouraging anti-tumor activity in heavily pre-treated patients with mCRPC
  - PSA50 response: 49% (Total) 59% (High-dose)
  - PSA90 response: 28% (Total) 36% (High-dose)
  - RECIST ORR: 24% (Total) 41% (High-dose)
- Dose expansion and optimization are currently ongoing to advance further development of xaluritamig as both a monotherapy and in combination

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CRS, cytokine release syndrome; D, day; IV, intravenous; mCRPC, metastatic castration-resistant prostate cancer; MTD, maximum tolerated dose; ORR, objective response rate; PSA, prostate-specific antigen; QW, weekly; RECIST; Response Evaluation Criteria in Solid Tumors; STEAP1, six transmembrane epithelial antigen of the prostate 1
Xaluritamig is being developed pursuant to a research collaboration with Xencor, Inc..

ADVANCING XALURITAMIG INTO EARLIER TREATMENT LINES WHERE THERE IS UNMET NEED AND OPPORTUNITY TO SERVE PATIENTS ACROSS THE CONTINUUM

Extensive Stage →

Disease Progression	Chemo Refractory (metastatic CRPC)	Chemo Naïve (metastatic CRPC)	Hormonal Therapy (non-metastatic, mCSPC)	Interventional Therapy (non-metastatic, mCSPC)
U.S. Drug-Treated Population	~8K	~38K	~150K	~260K
Current Efficacy Parameters	OS: 11–19m | PFS: 4–8m	OS: 19–24m | PFS: 8–10m	OS: ~50+m | PFS: ~33+m	*Potentially curable with surgery/radiation and hormonal therapy (ADT) for patients at higher risk for recurrence*
Xaluritamig Opportunity	Potential to deliver transformative OS with QoL improvement			Aspiration of curative Intent
Clinical Study	Phase 3 studies planned		P1 –Biochemical Recurrent Setting – Xaluritamig	P1 – Neoadjuvant Xaluritamig
Ongoing	P1/1b FIH – Monotherapy dose escalation, expansion and combination with SOC			

CRPC = metastatic castrate-resistant prostate cancer; mCSPC = metastatic castrate-sensitive prostate cancer; OS = overall survival; PFS = progression free survival; m = months; QoL = quality of life; P3 = Phase 3; ARDT = androgen receptor-directed therapy; P1 = Phase 1; FIH = first-in-human; SOC = standard of care.
Xaluritamig is being developed pursuant to a research collaboration with Xencor, Inc.

AMGEN

AMG 193

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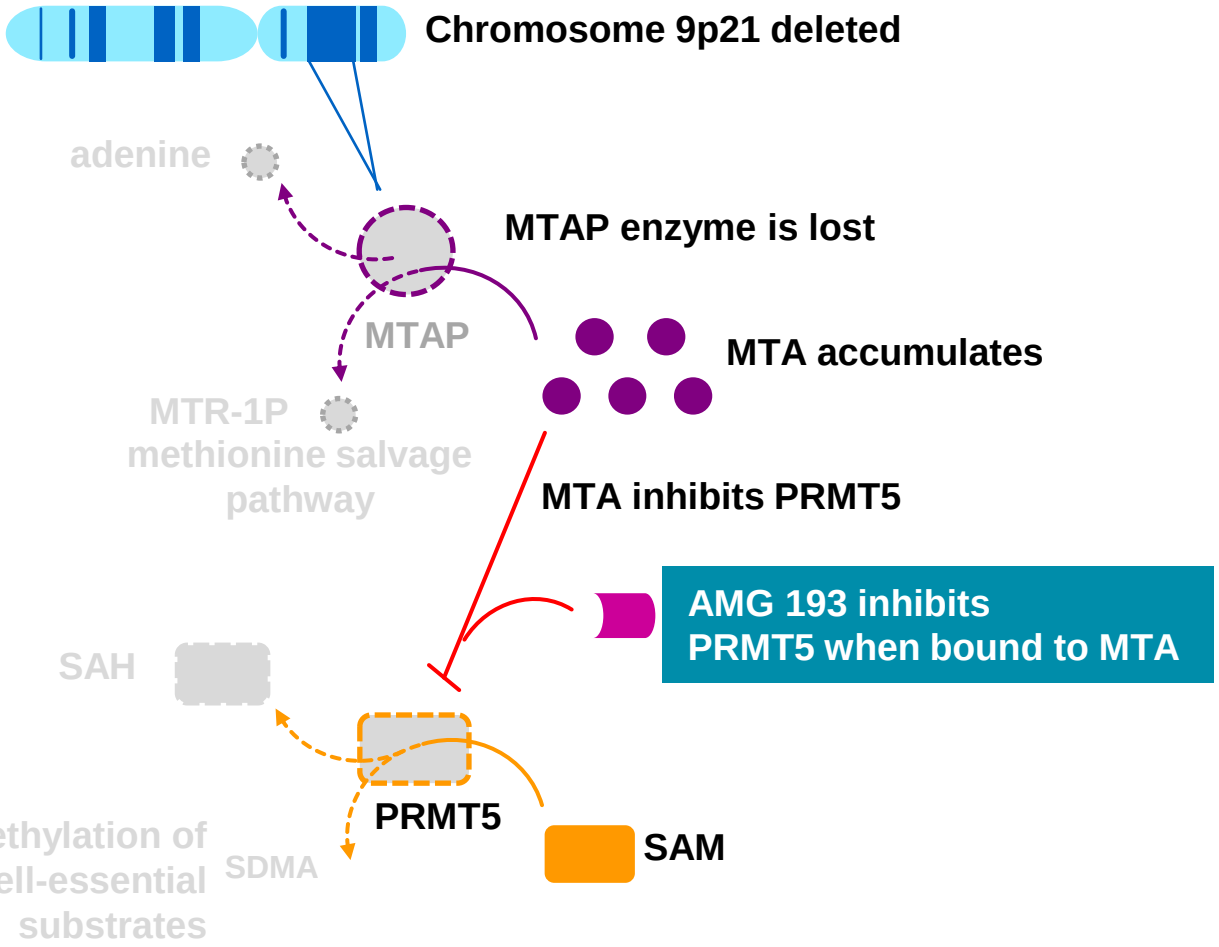
AMG 193, AN MTA–COOPERATIVE PRMT5 INHIBITOR THAT DEMONSTRATES SYNTHETIC LETHALITY IN MTAP-DELETED CANCERS

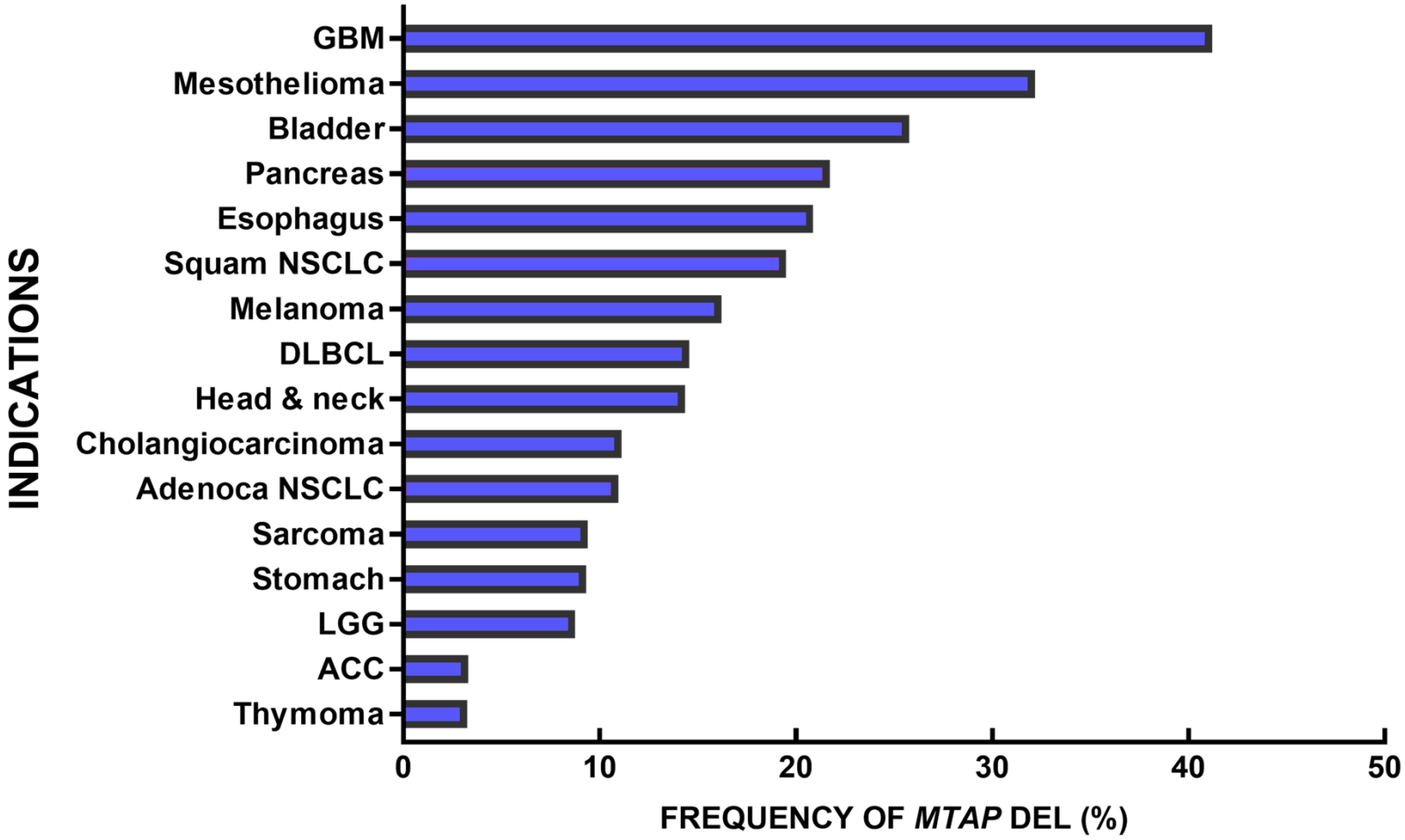


- **PRMT5** modifies proteins dysregulated in malignancy; implicated in cancer development and progression
- **MTAP** gene deletion results in accumulation of **MTA**, which competes with **SAM** binding to **PRMT5** resulting in partial loss of **PRMT5** activity
- **AMG 193** binds to and inhibits the **PRMT5-MTA** complex, resulting in the selective growth arrest of **MTAP**-deleted cells

Adapted from Marjon K, Cell Reports 2016

MTA = methylthioadenosine; PRMT5 = protein arginine methyltransferase 5; MTAP = methylthioadenosinephosphorylase; SAM = S-adenosylmethionine.

MTAP DELETION IS PREVALENT IN ~15% OF CANCERS



- >100k annual MTAP-null patient incidence in the U.S.
- Equivalent opportunity in Western Europe
- Top 5 tumors contribute ~70% of overall MTAP-null patient incidence
- MTAP deletion is detected through NGS, and routinely reported with Foundation One and Tempus results

Adapted from Marjon K, Cell Reports 2016

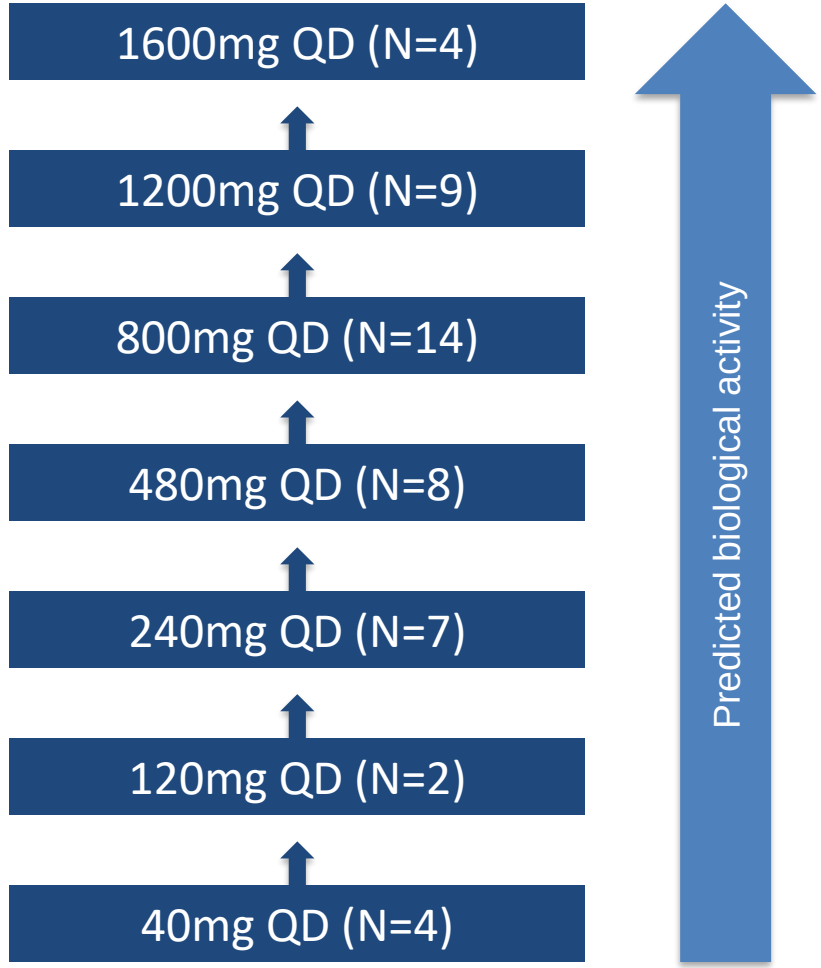
MTAP = methylthioadenosinephosphorylase; GBM = glioblastoma; NSCLC = non-small cell lung cancer; DLBCL = diffuse large B-cell lymphoma; LGG = low-grade gliomas; ACC = adenoid cyctic carcinoma; ; DEL = deletion; NGS = next-generation sequencing.

Phase 1, first-in-human, dose-escalation study of AMG 193 in advanced solid tumors selected for MTAP/CDKN2A loss

Key Inclusion Criteria
- Local NGS with homozygous MTAP-del or CDKN2A-del
- Central IHC MTAP null
- Measurable disease
- ECOG PS ≤ 1
- ANC ≥ 1.5 x 10^9 /L
- PLT ≥ 100 x 10^9 /L
- HB > 9 g/dL

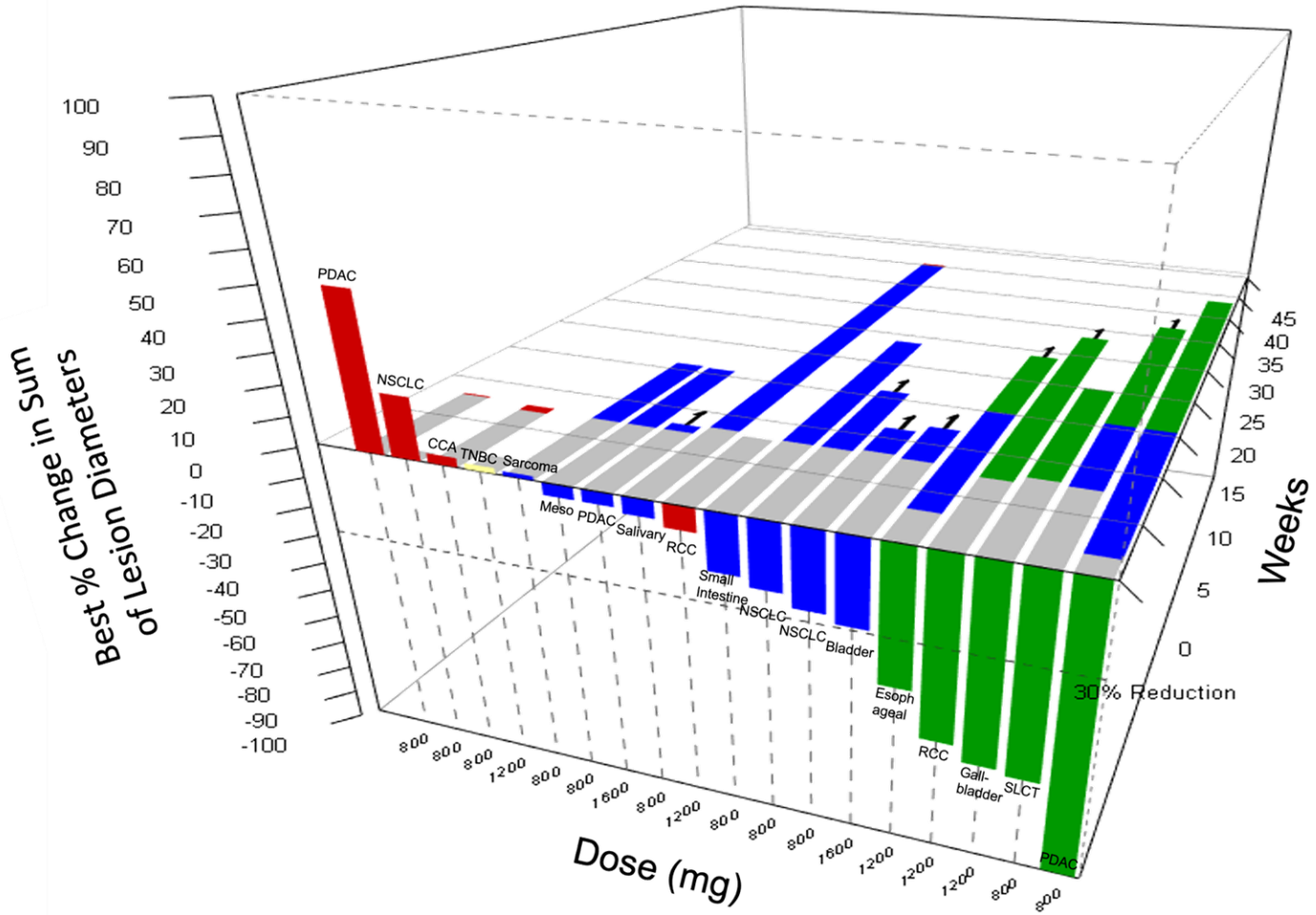
Key Exclusion Criteria
- Spinal cord compression, untreated brain metastases or leptomeningeal disease
- History of other malignancy within 2 years of study
- Prior PRMT5 or MAT2A inhibitor

Objectives/Endpoints
- Primary: Safety, tolerability, MTD/RP2D
- Secondary: PK, anti-tumor activity
- Exploratory: PD and correlative biomarkers



CDKN2A, cyclin dependent kinase inhibitor 2A; ECOG PS, Eastern Cooperative Oncology Group performance status; ANC, absolute neutrophil count; HB, hemoglobin; IHC, immunohistochemistry; MAT2A, methionine adenosyltransferase 2a; MTAP, methylthioadenosine phosphorylase; MTD, maximum tolerated dose; NGS, next-generation sequencing; PD, pharmacodynamics; PK, pharmacokinetics; PLT, platelets; PRMT5, protein arginine methyltransferase 5; RP2D, recommended phase 2 dose; QD, once daily.

AMG 193 demonstrates responses across solid tumors at ≥ 800 mg



Subset receiving ≥ 800 mg AMG 193 (N=18)

Presented on October 13 at AACR-NCI-EORTC International Conference: Molecular Targets and Cancer Therapeutics

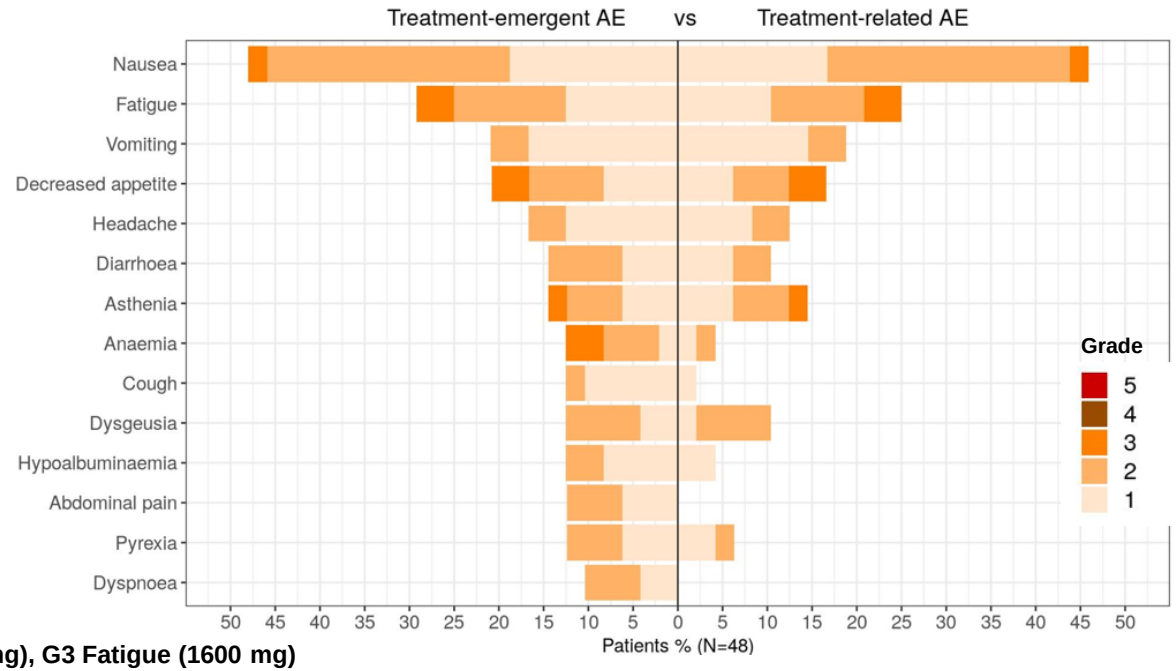
Data cut-off 01SEP23

CCA, cholangiocarcinoma; MESO, mesothelioma; NSCLC, non-small cell lung cancer; PDAC, pancreatic ductal adenocarcinoma; RCC, renal cell carcinoma; SLCT, Sertoli-Leydig cell tumor; SCLC, small cell lung cancer; TNBC, triple negative breast cancer. Response assessments conducted by investigator/local radiology according to RECIST v1.1

AMG 193 Preliminary Safety Profile

	All cohorts (N = 48)
DLTs, n	5
TEAEs, n (%)	44 (91.7)
Grade ≥ 3	15 (31.3)
Serious AEs	10 (20.8)
TRAEs, n (%)	39 (81.3)
Grade ≥ 3	8 (16.7)
Serious AEs	4 (8.3)
Leading to IP interruption, n (%)	10 (20.8)
Leading to IP reduction, n (%)	10 (20.8)
Leading to IP discontinuation, n (%)	3 (6.3)

TEAEs with incidence ≥ 10%



DLTs: G3 Hypersensitivity (240 mg), G1 Palpitation (800 mg), G3 Hypokalemia (1200 mg), G3 Nausea (1600 mg), G3 Fatigue (1600 mg)

N = number of patients in the safety analysis set who received at least one dose of AMG 193 ; n = number of patients with observed data;
AEs, adverse events; IP, investigational product; DLTs, dose-limiting toxicities; TEAEs, treatment-emergent adverse events; TRAEs, treatment-related adverse events.

Based on data cut-off: 01SEP2023

- **Consistent with tumor-selective MOA, there was no clinically significant myelosuppression**
- **1600 mg was intolerable due to DLTs of G3 nausea and G3 fatigue; evaluation of 1200 mg is ongoing**
- **Dosing alterations were typically due to GI events with patients reporting difficulty with pill burden**

Conclusions

- **AMG 193, an MTA-cooperative PRMT5i, targets MTAP-null solid tumors**
- **Encouraging monotherapy activity across five solid tumors**
- **Dose-limiting adverse events and treatment discontinuations were typically due to clinically manageable GI events**
- **Favorable therapeutic window and an opportunity for combination therapy**
- **Ongoing dose expansion cohorts will inform future development strategy**

MTA = methylthioadenosine; PRMT5i = protein arginine methyltransferase 5 inhibitor; MTAP = methylthioadenosinephosphorylase; GI = gastrointestinal.

LUMAKRAS®

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ENCOURAGING LUMAKRAS® DATA IN CRC AND NSCLC PROVIDES POTENTIAL TO REACH ADDITIONAL PATIENTS

Colorectal cancer

- CodeBreaK 300: Positive Phase 3 data of LUMAKRAS® + Vectibix® in 3L CRC
- Encouraging Phase 1b data of LUMAKRAS® + Vectibix® + FOLFIRI in previously treated KRAS G12C–mutated metastatic colorectal cancer

Non-small cell lung cancer

- Promising Phase 1b data of LUMAKRAS® + chemo in KRAS G12C–mutated advanced NSCLC

CRC= colorectal cancer; NSCLC= non-small cell lung cancer; 3L= third line; FOLFIRI = leucovorin calcium (folinic acid), fluorouracil, and irinotecan hydrochloride; KRAS = Kirsten rat sarcoma.

CodeBreaK 300 Phase 3 Study Design

Global, randomized, open-label, active-controlled study of sotorasib + panitumumab in mCRC (NCT05198934)

Key eligibility criteria

- ≥ 18 years of age
- *KRAS* G12C–mutated mCRC, identified through central molecular testing
- ≥ 1 prior line of therapy for mCRC; progressed on or after fluoropyrimidine, irinotecan, and oxaliplatin*
- ECOG ≤ 2
- Measurable disease per RECIST 1.1
- No prior $KRAS^{G12C}$ inhibitor†

Stratified by: prior anti-angiogenic therapy (yes / no), **time from diagnosis of mCRC** (≥18 mo / <18 mo), **ECOG status** (0 or 1 / 2)

Randomization 1:1:1 (N = 160)

Sotorasib 960 mg daily + panitumumab 6 mg/kg 2QW (n = 53)

Sotorasib 240 mg daily + panitumumab 6 mg/kg 2QW (n = 53)

Investigator's choice: trifluridine/tipiracil or regorafenib (n = 54)

Treat until disease progression, start of another anti-cancer treatment, withdrawal of consent , or intolerance of treatment

Primary endpoint: PFS by BICR (measured by CT / MRI and assessed by RECIST v1.1)
Key secondary endpoints: OS, ORR

*Patients deemed by the investigator not to be candidates for fluoropyrimidine, irinotecan, or oxaliplatin may still be eligible if ≥ 1 prior line of therapy was received for metastatic disease and trifluridine and tipiracil and/or regorafenib were deemed appropriate next line of therapy. †Patients with prior treatment with trifluridine and tipiracil and with regorafenib were excluded, where the investigator's choice would be these agents.

2QW, every 2 weeks; BICR, blinded independent central review; CT, computed tomography; ECOG, Eastern Cooperative Oncology Group; KRAS, Kirsten rat sarcoma; mCRC, metastatic colorectal cancer; MRI, magnetic resonance imaging; OS, overall survival; ORR, objective response rate; PFS, progression-free survival; RECIST, Response Evaluation Criteria in Solid Tumors.

Baseline Characteristics

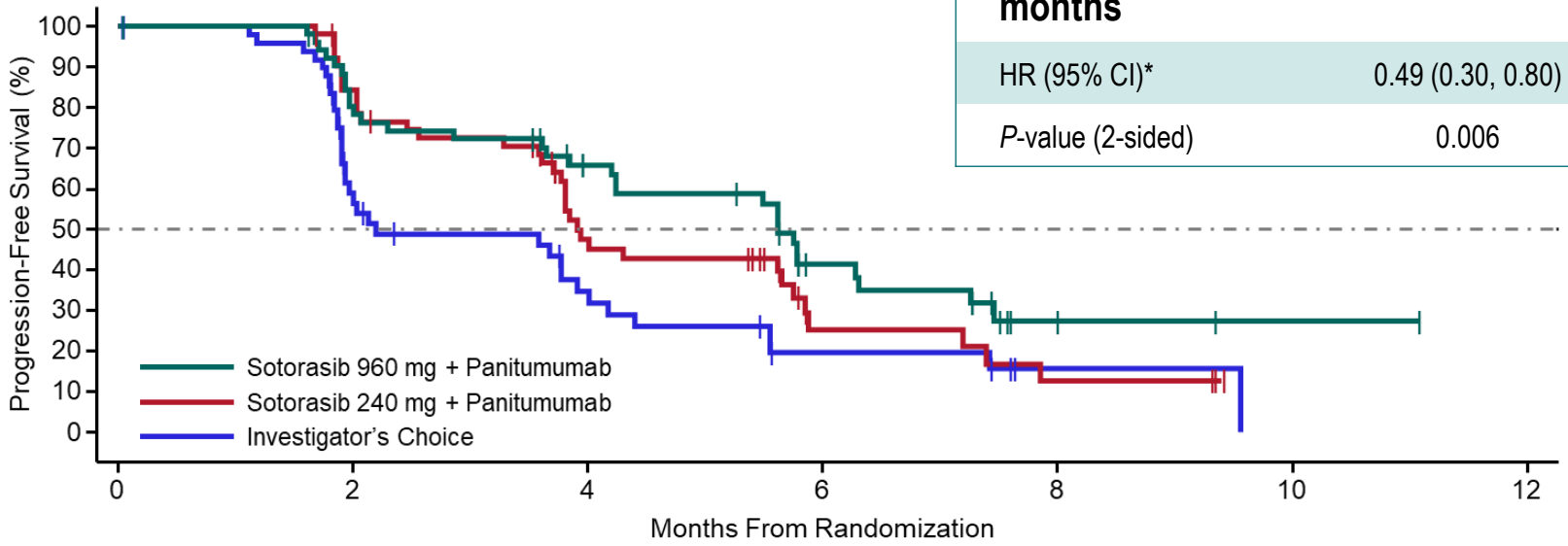
Characteristic	Sotorasib 960 mg + Panitumumab (n = 53)	Sotorasib 240 mg + Panitumumab (n = 53)	Investigator's Choice (n = 54)
Age, median (range), years	63.0 (37–79)	58.0 (35–82)	64.5 (34–81)
Male, n (%)	29 (55)	26 (49)	24 (44)
North America / Europe / Asia / Rest of world, %	9 / 77 / 11 / 2	9 / 53 / 36 / 2	13 / 67 / 20 / 0
ECOG performance status 0 / 1 / 2, %	60 / 36 / 4	55 / 42 / 4	65 / 33 / 2
Tumor sidedness, left / right / unknown, %	53 / 45 / 2	68 / 32 / 0	69 / 30 / 2
Prior lines of therapy, n (%)			
1	7 (13)	8 (15)	9 (17)
≥ 2	46 (87)	45 (85)	45 (83)
Prior oxaliplatin and irinotecan and fluoropyrimidine, n (%)	49 (92)	50 (94)	51 (94)
Prior anti-angiogenic therapy, n (%)	45 (85)	47 (89)	48 (89)
Prior trifluridine and tipiracil, n (%)	7 (13)	7 (13)	6 (11)
Prior regorafenib, n (%)	4 (8)	1 (2)	2 (4)

Demographics and baseline characteristics were generally balanced across arms

Data cutoff, 19 June 2023. ECOG, Eastern Cooperative Oncology Group.

Primary Endpoint: PFS in Intent-to-Treat Population

	Sotorasib 960 mg + Panitumumab (n = 53)	Sotorasib 240 mg + Panitumumab (n = 53)	Investigator's Choice (n = 54)
Median PFS, months	**5.6**	**3.9**	**2.2**
HR (95% CI)*	0.49 (0.30, 0.80)	0.58 (0.36, 0.93)	–
P-value (2-sided)	0.006	0.030	–



Number of Patients at Risk:

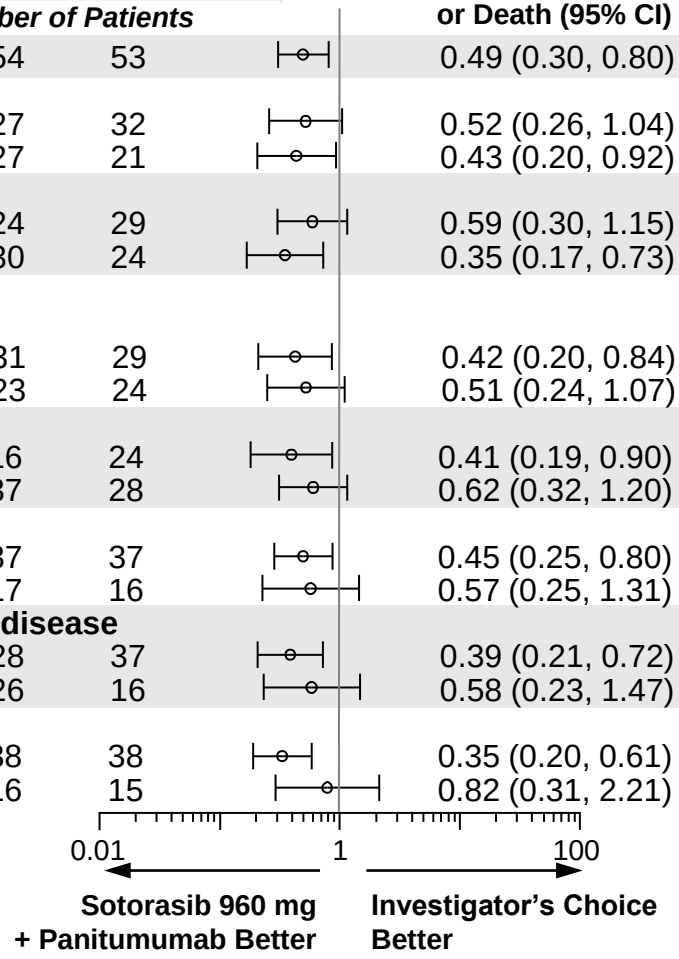
Sotorasib 960 mg + Panitumumab	53	40	28	13	2	1	0
Sotorasib 240 mg + Panitumumab	53	43	20	6	3	0	
Investigator's Choice	54	24	12	5	1	0	

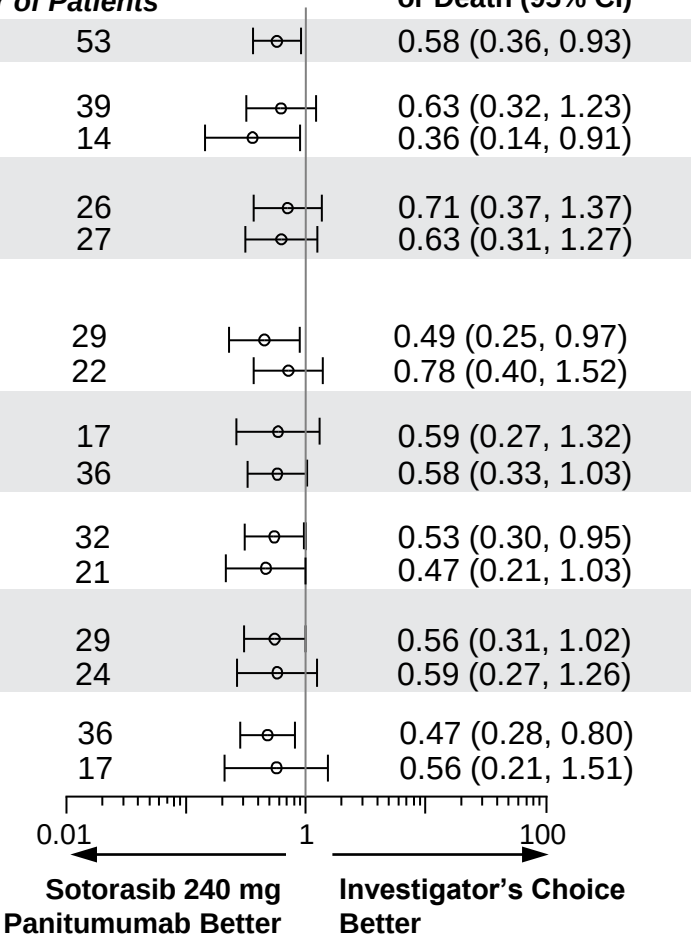
After a median follow-up of 7.8 months, sotorasib (960 mg and 240 mg) in combination with panitumumab significantly improved PFS by BICR versus investigator's choice

PFS was tested using stratified log-rank test. *HR is sotorasib 960 mg + panitumumab / investigator's choice therapy, or sotorasib 240 mg + panitumumab / investigator's choice therapy.
BICR, blinded independent central review; HR, hazard ratio; PFS, progression-free survival.

PFS Across Subgroups

Subgroup	Investigator's Choice (Number of Patients)	Sotorasib 960 mg + Panitumumab (Number of Patients)	Hazard Ratio for Disease Progression or Death (95% CI)	Investigator's Choice (Number of Patients)	Sotorasib 240 mg + Panitumumab (Number of Patients)	Hazard Ratio for Disease Progression or Death (95% CI)
All randomized patients	54	53	0.49 (0.30, 0.80)	54	53	0.58 (0.36, 0.93)
Age (years)						
< 65	27	32	0.52 (0.26, 1.04)	27	39	0.63 (0.32, 1.23)
≥ 65	27	21	0.43 (0.20, 0.92)	27	14	0.36 (0.14, 0.91)
Sex						
Male	24	29	0.59 (0.30, 1.15)	24	26	0.71 (0.37, 1.37)
Female	30	24	0.35 (0.17, 0.73)	30	27	0.63 (0.31, 1.27)
Time from initial diagnosis of metastatic disease to randomization						
≥ 18 months	31	29	0.42 (0.20, 0.84)	31	29	0.49 (0.25, 0.97)
< 18 months	23	24	0.51 (0.24, 1.07)	23	22	0.78 (0.40, 1.52)
Sidedness						
Right sided	16	24	0.41 (0.19, 0.90)	16	17	0.59 (0.27, 1.32)
Left sided	37	28	0.62 (0.32, 1.20)	37	36	0.58 (0.33, 1.03)
Primary tumor location						
Colon	37	37	0.45 (0.25, 0.80)	37	32	0.53 (0.30, 0.95)
Rectum	17	16	0.57 (0.25, 1.31)	17	21	0.47 (0.21, 1.03)
Number of prior therapy lines for metastatic disease						
1-2	28	37	0.39 (0.21, 0.72)	28	29	0.56 (0.31, 1.02)
≥ 3	26	16	0.58 (0.23, 1.47)	26	24	0.59 (0.27, 1.26)
Liver metastasis						
Yes	38	38	0.35 (0.20, 0.61)	38	36	0.47 (0.28, 0.80)
No	16	15	0.82 (0.31, 2.21)	16	17	0.56 (0.21, 1.51)





PFS by BICR favored sotorasib + panitumumab across key patient subgroups

No analysis performed for subgroups with n<10 patients in either arm. HRs (95% CI) were estimated using a Cox proportional hazards model stratified by the randomization stratification factors.
BICR, blinded independent central review; ECOG, Eastern Cooperative Oncology Group; HR, hazard ratio; PFS, progression-free survival.

Activity Outcomes

Response by BICR	Sotorasib 960 mg + Panitumumab (n = 53)	Sotorasib 240 mg + Panitumumab (n = 53)	Investigator's Choice (n = 54)
ORR, % (95% CI)*†	26 (15.3–40.3)	6 (1.2–15.7)	0 (0–6.6)
Complete response, n (%)	1 (2)	0	0
Partial response, n (%)	13 (25)	3 (6)	0
Stable disease, n (%)	24 (45)	33 (62)	25 (46)
Progressive disease, n (%)	12 (23)	13 (25)	17 (31)
Not evaluable / not done, n (%)	3 (6)	2 (4)	11 (20)
DCR, % (95% CI)*	72 (57.7–83.2)	68 (53.7–80.1)	46 (32.6–60.4)

ORR and DCR by BICR were higher with sotorasib (960 mg and 240 mg) + panitumumab versus investigator's choice

The intention-to-treat analysis set included all patients who underwent randomization.
*95% CIs were estimated using the Clopper-Pearson method. BICR, blinded independent central review; DCR, disease control rate; ORR, objective response rate
†Two patients (4%) in the 240 mg arm and 1 patient (2%) in the investigator's choice arm had non-complete response/non-progressive disease; these patients had BICR assessed non-target disease only

Safety Profile

	Sotorasib 960 mg + Panitumumab (n = 53)	Sotorasib 240 mg + Panitumumab (n = 53)	Investigator's Choice (n = 51)
Duration of treatment, median (range), months	5.8 (1.0–13.2)	4.1 (0.9–10.1)	2.2 (0.8–10.3)
TRAEs, n (%)	50 (94)	51 (96)	42 (82)
Grade ≥3	19 (36)	16 (30)	22 (43)
Serious	3 (6)	0	4 (8)
Leading to discontinuation of any IP	2 (4)	1 (2)	1 (2)
Leading to dose reduction of sotorasib	3 (6)	–*	–
Leading to dose reduction of panitumumab	7 (13)	9 (17)	–

Both sotorasib doses + panitumumab were tolerable, with no new safety signals and no fatal TRAEs

The safety analysis set included all randomized patients who received at least one dose of their assigned treatment.
*Sotorasib dose reduction was not allowed for the 240 mg group. Patients could resume the same dose after interruption or permanently discontinue.
IP, investigational product; TRAE, treatment-related adverse event.

Conclusions

- CodeBreaK 300 met its primary endpoint for superior PFS versus investigator's choice therapy in mCRC
- Sotorasib (960 mg and 240 mg) showed statistically significant improvements in PFS, **with the 960 mg dose demonstrating a more clinically meaningful benefit**
  - Median PFS was 5.6 months and 3.9 months (sotorasib + panitumumab) versus 2.2 months (investigator's choice)
  - PFS favored sotorasib + panitumumab across subgroups
- Higher ORR and DCR were observed, OS was immature at data cutoff
- No new safety concerns were observed
- **These results, along with previous data from NSCLC, support sotorasib 960 mg as the sotorasib dose for use in mCRC**
- **Sotorasib 960 mg plus panitumumab is a potential new standard-of-care therapy for patients with chemorefractory *KRAS* G12C–mutated mCRC**

DCR, disease control rate; KRAS, Kirsten rat sarcoma; mCRC, metastatic colorectal cancer; NSCLC, non-small cell lung cancer; ORR, objective response rate; OS, overall survival; PFS, progression-free survival.

MADRID 2023 ESMO congress

CONTINUE TO ADVANCE COMBINATION-FOCUSED LUMAKRAS® CLINICAL PROGRAM

Growth driven by moving into earlier treatment lines & additional tumor types

Colorectal cancer

- Discussions with Regulators ongoing for CodeBreaK 300 in 3L CRC
- Planning to initiate a Phase 3 study of LUMAKRAS® + Vectibix® + FOLFRI in 1L CRC
- KRAS G12C mutation present in ~3%–5% of CRC cases

Non-small cell lung cancer

- Initiated a Phase 3 study of LUMAKRAS® + chemo in 1L PD-L1 negative KRAS G12C mutated NSCLC
- ~13K KRAS G12C U.S. patients in 1L NSCLC (approximately 1/3 are PD-L1 negative)

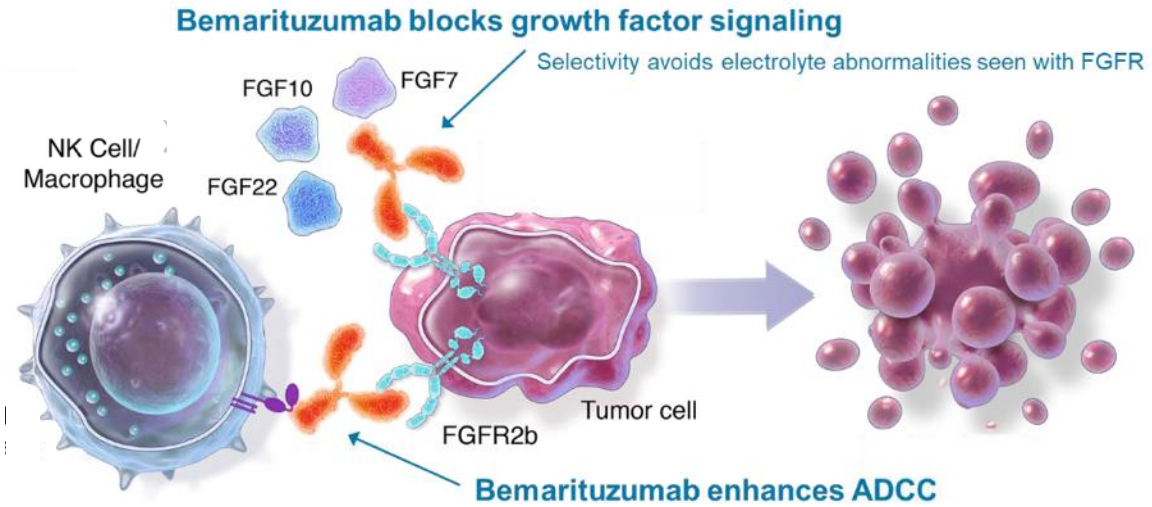
CRC= colorectal cancer; NSCLC= non-small cell lung cancer; 3L= third line; FOLFIRI = leucovorin calcium (folinic acid), fluorouracil, and irinotecan hydrochloride; KRAS = Kirsten rat sarcoma; PD-L1 = programmed cell death protein ligand 1.

Bemarituzumab

IMPROVED BIOLOGICAL UNDERSTANDING IS CREATING AN OPPORTUNITY IN GASTRIC CANCER

- Gastric cancer is the fifth most common cancer worldwide with over 1 million new cases globally[2] and 25k U.S.[3] each year
- New therapeutics options needed in this disease
- Emerging biomarker targeted therapies could provide important treatment options
- Addressable patient population of ~7,000 in the U.S. and ~250K worldwide. Significant need in Southeast Asian region

Bemarituzumab is a first-in-class monoclonal antibody targeting FGFR2b

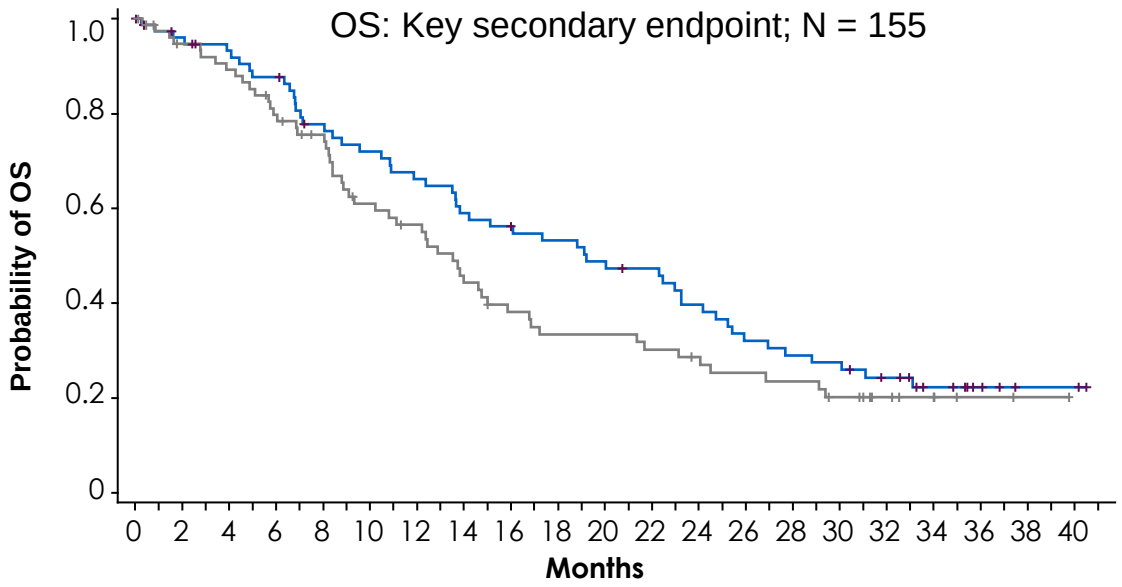


FGFR2b+ represents **~30%**[1] of gastric cancer patients

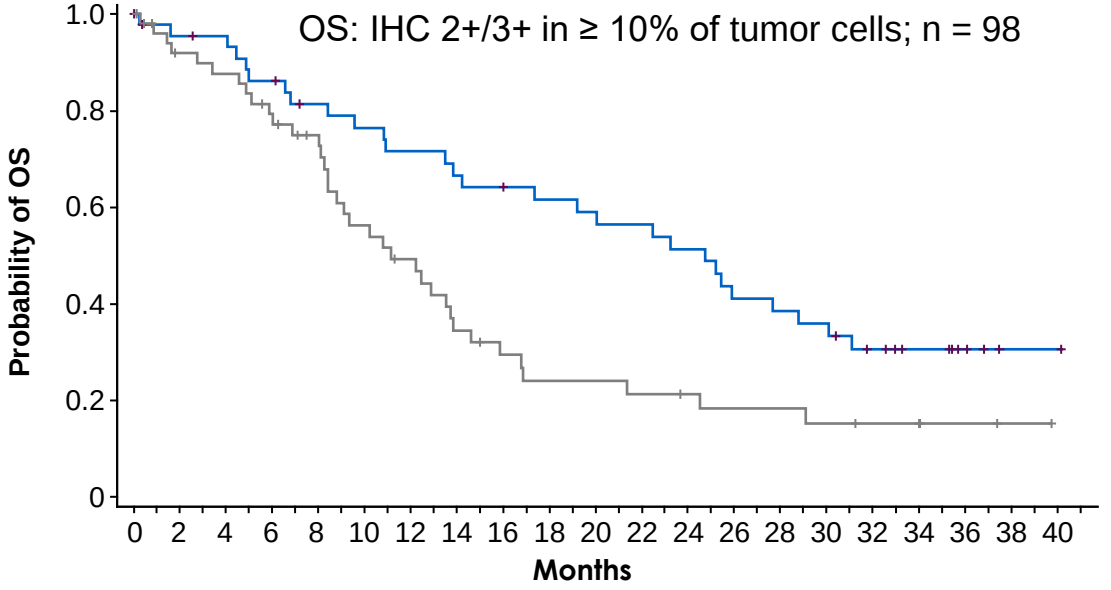
FGFR= fibroblast growth factor receptor; FGFR2b= fibroblast growth factor receptor 2b; FGF7= fibroblast growth factor 7; FGF10= fibroblast growth factor 10; FGF22= fibroblast growth factor 22; NK= natural killer; ADCC= antibody-dependent cellular cytotoxicity

1. FGFR2b prevalence in Five Prime's non-HER2+ Phase 2 FIGHT study; 2. Bray et al, 2018; 3. American Cancer Society Key Statistics, 2022

A PHASE 2 STUDY OF BEMARITUZUMAB PLUS CHEMO IN FIRST-LINE ADVANCED FGFR2B+ GASTRIC CANCER DEMONSTRATED ENCOURAGING OVERALL SURVIVAL



	Bema (N = 77)	Pbo (N = 78)
Median follow-up, months (95% CI)[a]	**34.8 (32.6, 36.1)**	**31.4 (29.5, 34.0)**
mOS, months (95% CI)	**19.2 (13.6–24.2)**	**13.5 (9.3–15.9)**
HR (95% CI)	**0.77 (0.52–1.14)**	



	Bema (n = 46)	Pbo (n = 52)
mOS, months (95% CI)	**24.7 (14.2–30.1)**	**11.1 (8.4–13.8)**
HR (95% CI)	**0.52 (0.31–0.85)**	

Treatment benefit more pronounced in the subgroup with ≥ 10% FGFR2b expression

[a] Median follow-up is estimated by using the reverse Kaplan-Meier approach.

FGFR2b= fibroblast growth factor receptor 2b;, OS = overall survival; IHC = immunohistochemistry; CI, confidence interval; mOS = median overall survival; HR, hazard ratio.

PROGRESSING PHASE 3 STUDIES OF BEMARITUZUMAB IN 1L GASTRIC CANCER

CLINICAL TRIAL	TREATMENTS	ADVANCED CANCERS	PHASE 1b	PHASE 2	PHASE 3
FORTITUDE 101	Bemarituzumab + mFOLFOX6 vs. Placebo + mFOLFOX6	1L G/GEJ Cancer			
FORTITUDE 102	Bemarituzumab + Chemo*+ Nivolumab vs. Placebo + Chemo* + Nivolumab	1L G/GEJ Cancer 1L G/GEJ Cancer			

1L = first-line; mFOLFOX6 = oxaliplatin plus leucovorin plus 5-fluorouracil; G/GEJ = gastric or gastroesophageal junction; Chemo* = fluoropyrimidine- and platinum-containing chemotherapy, specifically mFOLFOX6 or CAPOX (oxaliplatin plus capecitabine).

Concluding Remarks

AMGEN'S ONCOLOGY PORTFOLIO IS POISED TO POTENTIALLY ALTER THE NATURAL HISTORY OF DISEASE ACROSS MULTIPLE CANCERS

BLINCYTO®
Long-term aspiration is to expand across all subsets of B-ALL and to utilize sub-Q delivery where possible.

Tarlatamab
Encouraging data in 3L SCLC with regulatory submission underway. Rapidly advancing into earlier treatment lines.

Xaluritamig
Promising anti-tumor activity in heavily pre-treated patients with mCRPC; advancing into earlier treatment lines.

AMG 193
Initial activity in multiple solid tumors, dose expansion will help to define future development strategy.

LUMAKRAS®
Advancing combination therapy programs in colorectal cancer and non-small cell lung cancer.

Bemarituzumab
Progressing Phase 3 studies in 1L gastric cancer.

B-ALL =B-cell precursor acute lymphoblastic leukemia; Sub-Q = subcutaneous; 3L = third-line; SCLC = small cell lung cancer; mCRPC = metastatic castrate resistant prostate cancer; 1L = first-line.

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Tarlatamab for Patients with Previously Treated Small-Cell Lung Cancer

Myung-Ju Ahn, M.D., Ph.D., Byoung Chul Cho, M.D., Ph.D., Enriqueta Felip, M.D., Ph.D., Ippokratis Korantzis, M.D., Kadoaki Ohashi, M.D., Ph.D., Margarita Majem, M.D., Ph.D., Oscar Juan-Vidal, M.D., Ph.D., Sabin Handzhiev, M.D., Hiroki Izumi, M.D., Ph.D., Jong-Seok Lee, M.D., Ph.D., Rafal Dziadziuszko, M.D., Ph.D., Jürgen Wolf, M.D., Fiona Blackhall, M.D., Ph.D., Martin Reck, M.D., Ph.D., Jean Bustamante Alvarez, M.D., M.S., Horst-Dieter Hummel, M.D., Anne-Marie C. Dingemans, M.D., Ph.D., Jacob Sands, M.D., Hiroaki Akamatsu, M.D., Ph.D., Taofeek K. Owonikoko, M.D., Ph.D., Suresh S. Ramalingam, M.D., Hossein Borghaei, D.O., Melissa L. Johnson, M.D., Shuang Huang, Ph.D., Sujoy Mukherjee, M.D., Mukul Minocha, Ph.D., Tony Jiang, Ph.D., Pablo Martinez, M.D., Ph.D., M.Sc., Erik S. Anderson, M.D., Ph.D., and Luis Paz-Ares, M.D., Ph.D., for the DeLLphi-301 Investigators*

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Sotorasib plus Panitumumab in Refractory Colorectal Cancer with Mutated *KRAS* G12C

Marwan G. Fakih, M.D., Lisa Salvatore, M.D., Taito Esaki, M.D., Dominik P. Modest, M.D., David P. Lopez-Bravo, M.D., Julien Taieb, M.D., Michalis V. Karamouzis, M.D., Erika Ruiz-Garcia, M.D., Tae-Won Kim, M.D., Yasutoshi Kuboki, M.D., Fausto Meriggi, M.D., David Cunningham, M.D., Kun-Huei Yeh, M.D., Emily Chan, M.D., Ph.D., Joseph Chao, M.D., Yaneth Saportas, Ph.D., Qui Tran, Ph.D., Chiara Cremolini, M.D., and Filippo Pietrantonio, M.D.

RESEARCH ARTICLE

Xaluritamig, a STEAP1 × CD3 XmAb 2+1 Immune Therapy for Metastatic Castration-Resistant Prostate Cancer: Results from Dose Exploration in a First-in-Human Study

William K. Kelly[1,2], Daniel C. Danila[3,4], Chia-Chi Lin[5], Jae-Lyun Lee[6], Nobuaki Matsubara[7], Patrick J. Ward[2,8], Andrew J. Armstrong[9], David Pook[10], Miso Kim[11], Tanya B. Dorff[12], Stefanie Fischer[13], Yung-Chang Lin[14], Lisa G. Horvath[15], Christopher Sumey[16], Zhao Yang[17], Gabor Jurida[17], Kristen M. Smith[18], Jamie N. Connarn[18], Hweixian L. Penny[17], Julia Stieglmaier[19], and Leonard J. Appleman[20]

Q&A