Revolution Medicines

# On Target to Outsmart Cancer:

## Results from Phase 3 RASolute 302 Study

May 31, 2026

# Legal Disclaimer

This presentation contains forward-looking statements within the meaning of the Private Securities Litigation Reform Act. All statements other than statements of historical facts contained in this presentation, including statements regarding our future results of operations and financial position, business strategy, product candidates, availability of funding, ability to manage existing collaborations and establish new strategic collaborations, licensing or other arrangements, the scope, progress, results and costs of developing our product candidates or any other future product candidates, conducting clinical trials, the potential market size and size of the potential patient populations for our product candidates, commercialization plans and capabilities, the timing and likelihood of success of obtaining product approvals, plans and objectives of management for future operations, future results of anticipated products and the impact of global events and other macroeconomic conditions on our business are forward-looking statements. These statements involve known and unknown risks, uncertainties and other important factors that may cause our actual results, performance or achievements to be materially different from any future results, performance or achievements expressed or implied by the forward-looking statements. Because forward-looking statements are inherently subject to risks and uncertainties, some of which cannot be predicted or quantified and some of which are beyond our control, you should not rely on these forward-looking statements as predictions of future events. The events and circumstances reflected in our forward-looking statements may not be achieved or occur and actual results could differ materially from those projected in the forward-looking statements. The information included in these materials is provided as of May 6, 2026, unless specified elsewhere herein, and is qualified as such. Except as required by applicable law, we undertake no obligation to update any forward-looking statements or other information contained herein, whether as a result of any new information, future events, changed circumstances or otherwise.

For a further description of the risks and uncertainties that could cause actual results to differ from those anticipated in these forward-looking statements, as well as risks relating to the business of Revolution Medicines in general, see Revolution Medicines' Quarterly Report on Form 10-Q filed with the Securities and Exchange Commission on May 6, 2026, and its future periodic reports to be filed with the Securities and Exchange Commission.

This presentation concerns product candidates that are under clinical investigation and which have not yet been approved for marketing by the U.S. Food and Drug Administration (FDA) or any other regulatory authority. These product candidates are currently limited by federal law to investigational use, and no representation is made as to their safety or effectiveness for the purposes for which they are being investigated.

This presentation includes certain information regarding publicly available results from clinical trials by third parties evaluating other product candidates. Such trials were not head-to-head trials with any of Revolution Medicines' product candidates and include differences in study protocols, patient populations and reporting standards, and caution should be exercised when comparing data across trials.

# Growing Impact for Patients

## Major milestone reached

- Unprecedented overall survival benefit demonstrated in RASolute 302, a randomized Phase 3 clinical trial of daraxonrasib (oral, once daily) in patients with previously treated metastatic pancreatic cancer

## Expanding reach with daraxonrasib

- Preparation of NDA for U.S. FDA as first step in global registration
- Ongoing Phase 3 trials across treatment lines and tumor types

## Robust pipeline momentum

- Expansive development programs with multiple pioneering investigational targeted medicines and rich discovery portfolio

# **Our Vision** | Build a Leading Global Targeted Oncology Company Focused on RAS-Addicted Cancers

**Highly Productive Innovation Engine**

Proprietary, clinically-validated discovery platform fuels virtuous cycle of innovation to produce expansive collection of developable RAS(ON) inhibitor candidates

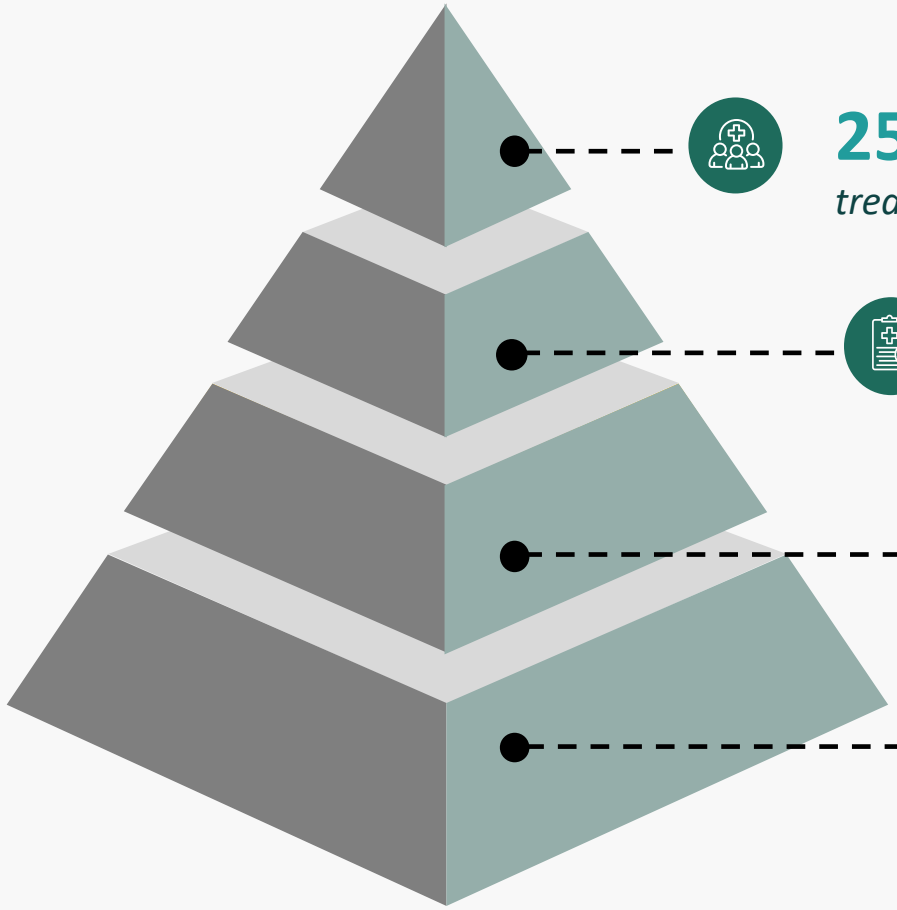
**Deep, Durable Asset Portfolio**

Pioneering assets with differentiated activity against key tumor types and diverse RAS mutations, expanded further by rational combination strategies

**Multiple Groundbreaking Medicines**

Initial wave of product candidates demonstrating strong performance with first registrational study completed; pipeline sustained through a generational product strategy and global commercial capabilities

# **Our Mission** | Revolutionize Treatment Globally for Patients with RAS-Addicted Cancers through the Discovery, Development and Delivery of Innovative, Targeted Medicines



**2500+ patients**

*treated with one or more of our RAS(ON) inhibitors*

**8 randomized Phase 3 registrational trials**

- *completed, active and planned for 2026*

*+ extensive Phase 1/2 trials*

**4 clinical-stage investigational drugs**

- *daraxonrasib | zoldonrasib | elironrasib | RMC-5127*

*+ preclinical pipeline*

**3 common RAS-addicted cancers**

*pancreatic | non-small cell lung | colorectal*



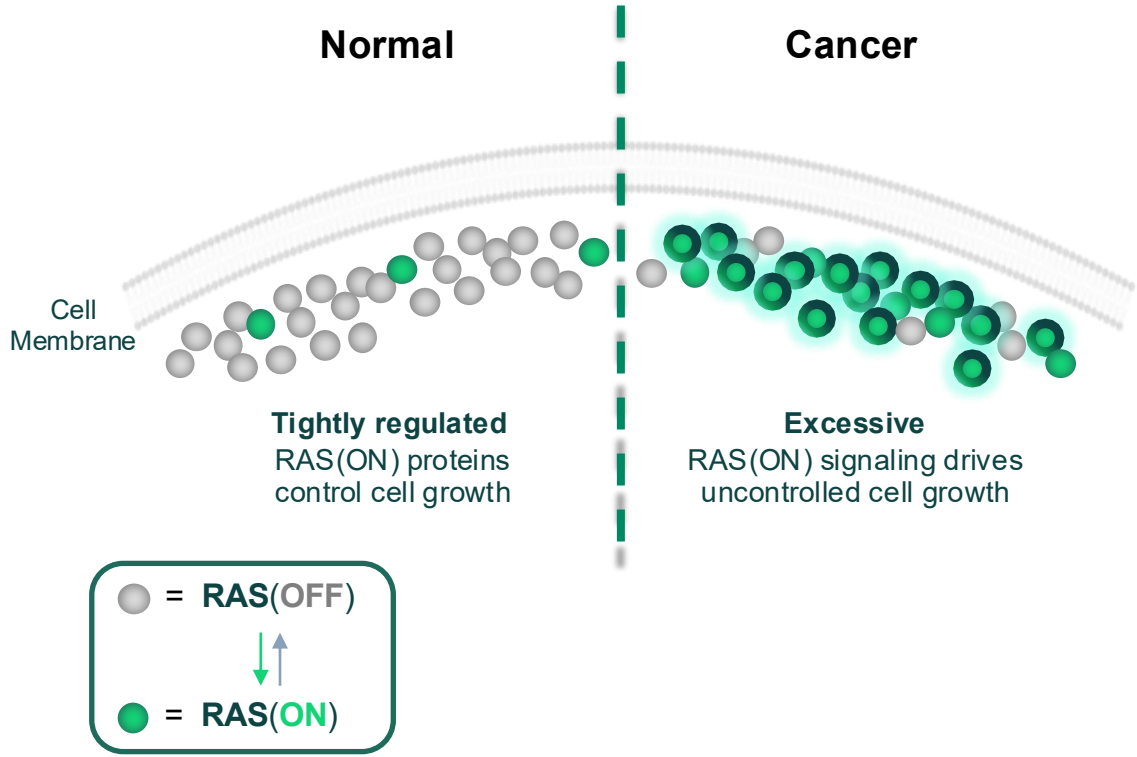
As of January 2026

# RASolute 302 Detailed Results

# Background

- PDAC is among the most challenging and lethal cancers, with limited therapeutic options
- Standard cytotoxic chemotherapy provides limited benefit in previously treated mPDAC: mPFS 3–4 months; mOS 6–7 months[1-3]
- Excessive RAS(ON) signaling drives tumor growth across RAS-mutant and RAS wild-type PDAC
  - >90% of PDAC tumors harbor an oncogenic RAS mutation[4,5]
- No RAS-targeted therapies are approved for PDAC, highlighting a substantial unmet need

## Uncontrolled RAS(ON) Signaling Drives Cells to an Oncogenic State[6-8]



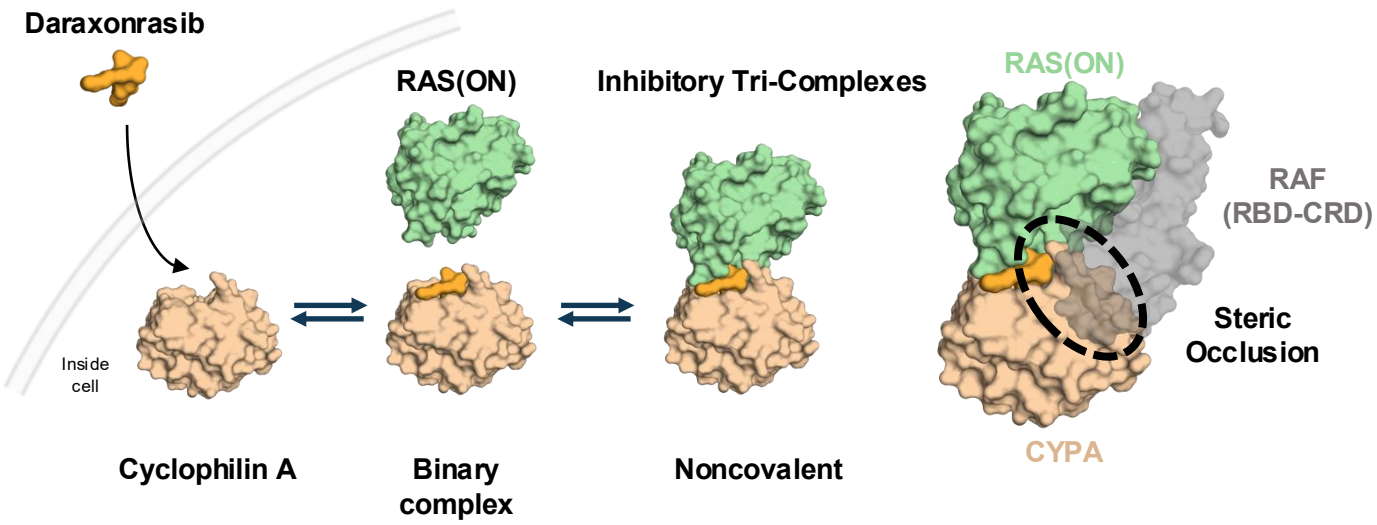

m, median; mPDAC, metastatic pancreatic adenocarcinoma; OS, overall survival; PDAC, pancreatic adenocarcinoma; PFS, progression-free survival.

1. Park W, et al. JAMA. 2021;326:851-62; 2. Petrelli F, et al. BMC Gastroenterol. 2023;23:212; 3. Dayyani F, et al. Cancer Treat Rev. 2023;113:102502; 4. Pant S, et al. J Clin Oncol. 2025;43:777; 5. Cancer Genome Atlas Research Network. Cancer Cell. 2017;32:185-203.e13; 6. Holderfield M, et al. Nature. 2024;629:919-26; 7. Lokhandwala J, et al. Front Oncol. 2024;14:1394702; 8. Jiang J, et al. Cancer Discov. 2024;14:994-1017.

# Daraxonrasib Mechanism of Action

## Daraxonrasib is an oral RAS(ON) multi-selective inhibitor

- Daraxonrasib is an oral, potent, RAS(ON), multi-selective, tri-complex inhibitor of GTP-bound mutant RAS (including variants with G12, G13, and Q61 mutations) and wild-type RAS[1-4]
- In a prior Phase 1/2 trial, daraxonrasib showed clinical activity with a manageable safety profile in patients with previously treated advanced PDAC[5]




Daraxonrasib binds to intracellular cyclophilin A to form a binary complex that engages RAS(ON) and suppresses downstream signaling[1-4]

CRD, cysteine rich domain; GTP, guanosine triphosphate; PDAC, pancreatic adenocarcinoma; RBD, RAS-binding domain.

1. Holderfield M, et al. Nature. 2024;629:919-26; 2. Cregg J, et al. J Med Chem. 2025;68:6064-83; 3. Lokhandwala J, et al. Front Oncol. 2024;14:1394702; 4. Jiang J, et al. Cancer Discov. 2024;14:994-1017; 5. Wolpin B, et al. N Engl J Med. 2026;394:1790-1802.

# Study Design

**Key eligibility criteria:**

- Adults with mPDAC
- One prior fluoropyrimidine- or gemcitabine-based regimen in the metastatic setting
- ECOG PS 0-1
- Documented tumor RAS mutational status[a] by local testing

R
1:1

**Daraxonrasib**
300 mg, PO QD

**Investigator's choice chemotherapy (1 of 4)**
GnP, mFOLFIRINOX, Nal-IRI + 5-FU/LV, FOLFOX

**Dual primary endpoints**

- OS and PFS[b] (RAS G12 population)

**Key secondary endpoints**

- OS and PFS[b] (overall population[c])
- ORR[b] (RAS G12 and overall populations)
- TTD in PROs[d] (RAS G12 and overall populations)

**Stratification factors**

- ECOG PS (0 vs 1)
- Metastatic disease at diagnosis (yes vs no)
- Liver metastases at baseline (yes vs no)
- Tumor RAS mutational status (RAS G12D/V vs other RAS G12 vs RAS G13 or Q61 mutation or no RAS mutation identified)

**Global enrollment:** 59 sites across 6 countries

**NCT06625320.**

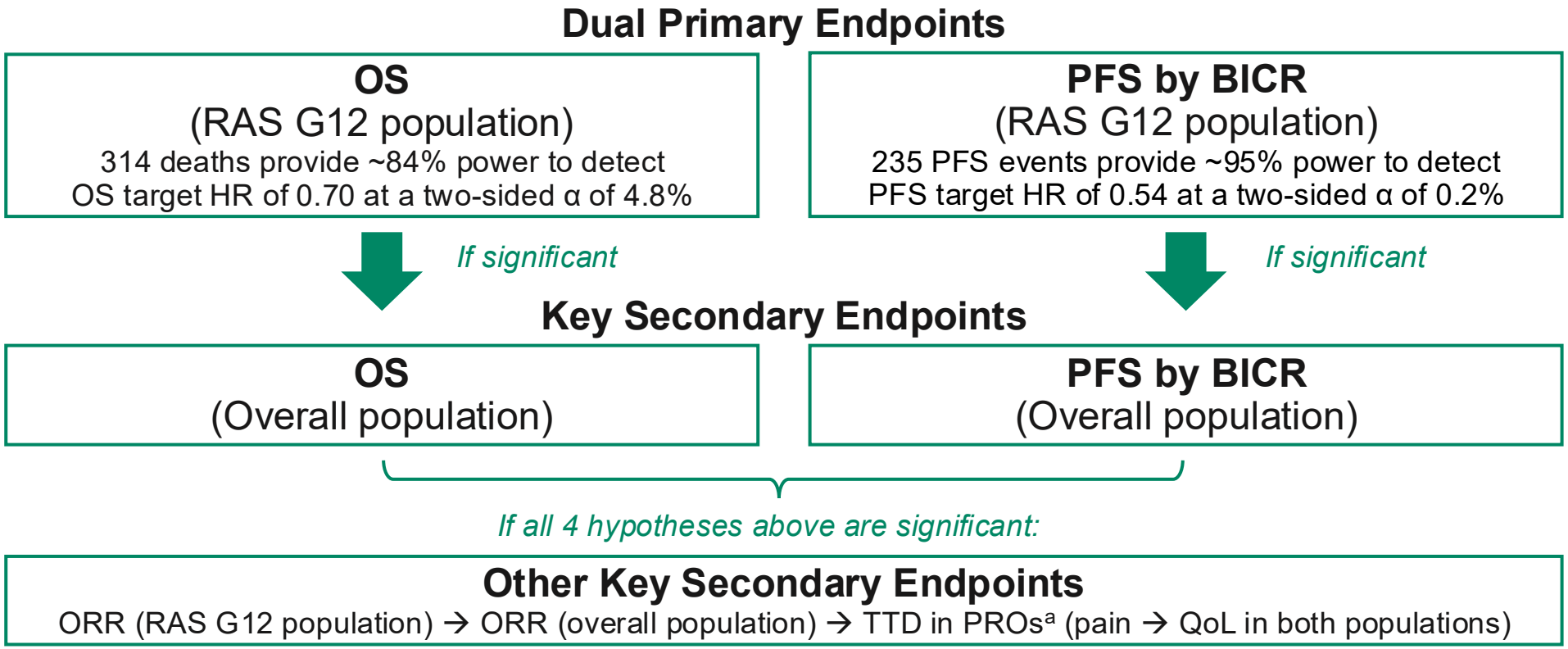
5-FU, 5-fluorouracil; BICR, blinded independent central review; ECOG Eastern Cooperative Oncology Group; FOLFOX, leucovorin, 5-FU, and oxaliplatin; GnP, gemcitabine and nab-paclitaxel; LV, leucovorin; mFOLFIRINOX, modified 5-FU, irinotecan, leucovorin, and oxaliplatin; mPDAC, metastatic pancreatic adenocarcinoma; Nal-IRI, nanoliposomal irinotecan; ORR, objective response rate; OS, overall survival; PFS, progression-free survival; PO, oral administration; PRO, patient-reported outcome; PS, performance status; QD, once daily; RECIST, Response Evaluation Criteria in Solid Tumors; TTD, time to deterioration.

[a]Either mutant, defined as nonsynonymous mutations in KRAS, NRAS, or HRAS at codons 12, 13, or 61 (G12, G13, or Q61), or no RAS mutation identified. [b]By BICR per RECIST 1.1. [c]Including patients whose tumors harbor RAS G12, G13, or Q61 mutations, or had no RAS mutation identified. [d]TTD in symptom of pain and in global health status/quality of life.

# Statistical Analysis Overview

Two interim OS analyses were planned before the final analysis in the RAS G12 population

- First OS interim analysis (IA1): planned after enrollment completion and ≥166 deaths; final analysis for PFS
- Results reported are based on IA1



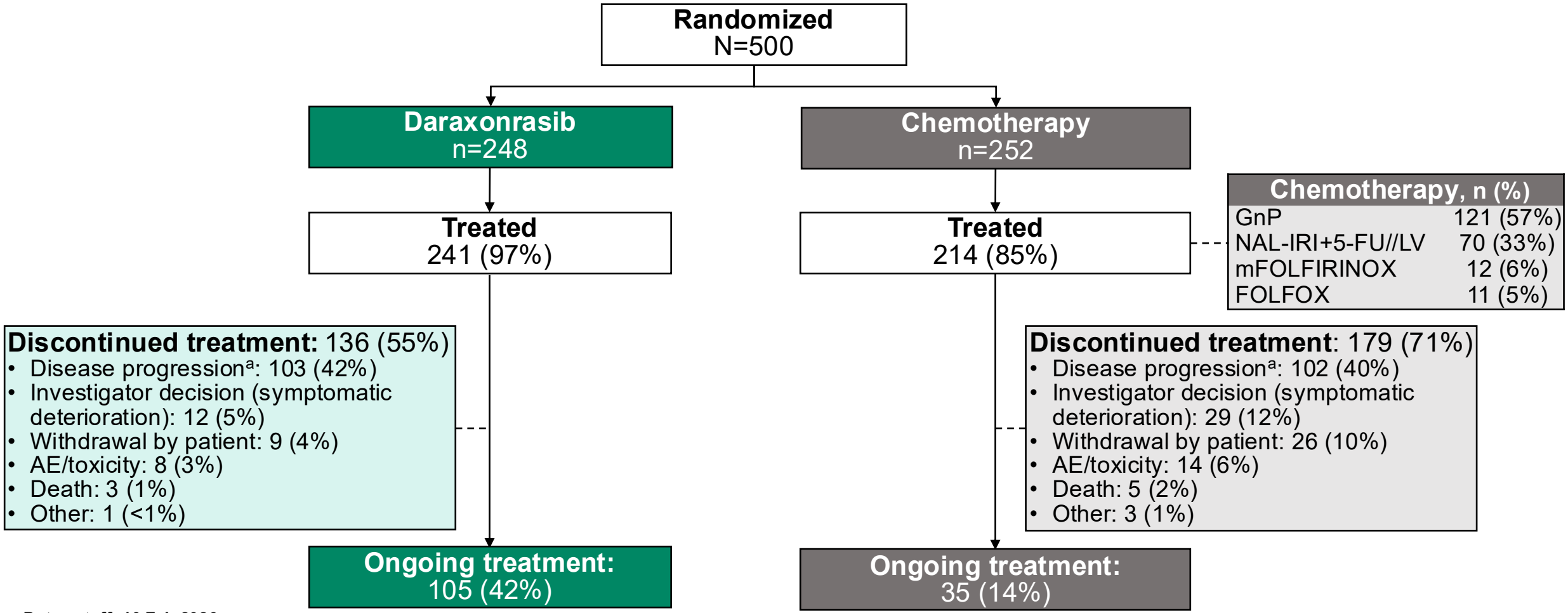

**NCT06625320.**

BICR, blinded independent central review; EORTC QLQ-C30, European Organisation for the Research and Treatment of Cancer Quality of Life Questionnaire; EORTC QLQ-PAN26, European Organisation for the Research and Treatment of Cancer Quality of Life Questionnaire – Pancreatic Cancer Module; GHS, global health status; ORR, objective response rate; OS, overall survival; PFS, progression-free survival; PRO, patient-reported outcome; QoL, quality of life; TTD, time to deterioration.

[a]TTD in symptom of pain and in GHS/QoL, defined as a change of at least 10 points from baseline or death (whichever occurs first) in the EORTC QLQ-PAN26 pain scale and in the EORTC QLQ-C30 GHS/QoL scale, respectively.

# Patient Disposition

Enrollment period: October 16, 2024 – November 7, 2025




**Data cutoff: 10 Feb 2026.**

5-FU, 5-fluorouracil; AE, adverse event; FOLFOX, leucovorin, 5-FU, and oxaliplatin; GnP, gemcitabine and nab-paclitaxel; LV, leucovorin; mFOLFIRINOX, modified 5-FU, irinotecan, leucovorin, and oxaliplatin; Nal-IRI, nanoliposomal irinotecan; RECIST, Response Evaluation Criteria for Solid Tumors.

[a]Documented based on RECIST 1.1.

# Baseline Demographics and Disease Characteristics

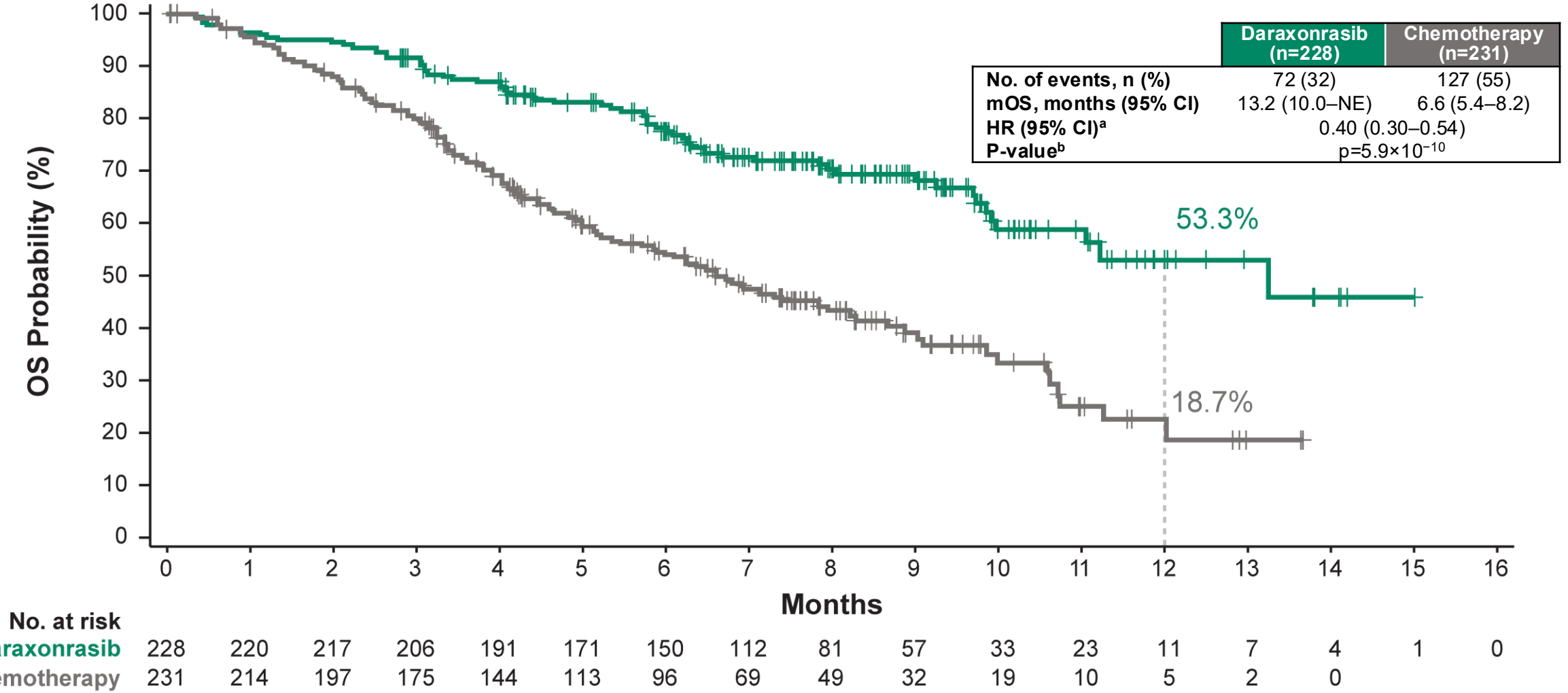
| | Daraxonrasib (n=248) | Chemotherapy (n=252) |
|---|---|---|
| **Age, median (range), years** | 66 (30-88) | 65 (36-86) |
| **Female, n (%)** | 117 (47) | 108 (43) |
| **Region, n (%):** North America \| Europe \| APAC | 145 (58) \| 87 (35) \| 16 (6) | 158 (63) \| 74 (29) \| 20 (8) |
| **ECOG PS, n (%):** 0 \| 1[a] | 129 (52) \| 119 (48) | 118 (47) \| 134 (53) |
| **Metastatic disease at initial diagnosis, n (%)** | 154 (62) | 154 (61) |
| **Prior chemotherapy regimens in metastatic setting, n (%)** | | |
| (m)FOLFIRINOX | 98 (40) | 105 (42) |
| Gemcitabine + nab-paclitaxel | 94 (38) | 94 (37) |
| Other | 56 (23) | 53 (21) |
| **Prior pancreatectomy, n (%)** | 83 (33) | 92 (37) |
| **Liver metastases at baseline, n (%)** | 174 (70) | 176 (70) |
| **Tumor RAS mutational status, n (%)** | | |
| RAS G12 D/V | 197 (79) | 197 (78) |
| Other RAS G12 | 31 (13) | 34 (13) |
| RAS G13 or Q61, or no RAS mutation identified | 20 (8) | 21 (8) |

**Data cutoff: 10 Feb 2026.**

5-FU, 5-fluorouracil; APAC, Asia-Pacific; ECOG, Eastern Cooperative Oncology Group; mFOLFIRINOX, modified 5-FU, irinotecan, leucovorin, oxaliplatin; PS, performance status.

[a]Three patients (including 2 patients randomized to the daraxonrasib arm and 1 patient randomized to the chemotherapy arm) had ECOG PS >1 at baseline. These patients were not dosed due to no longer meeting trial eligibility criteria.

# Primary Endpoint: Overall Survival in the RAS G12 Population



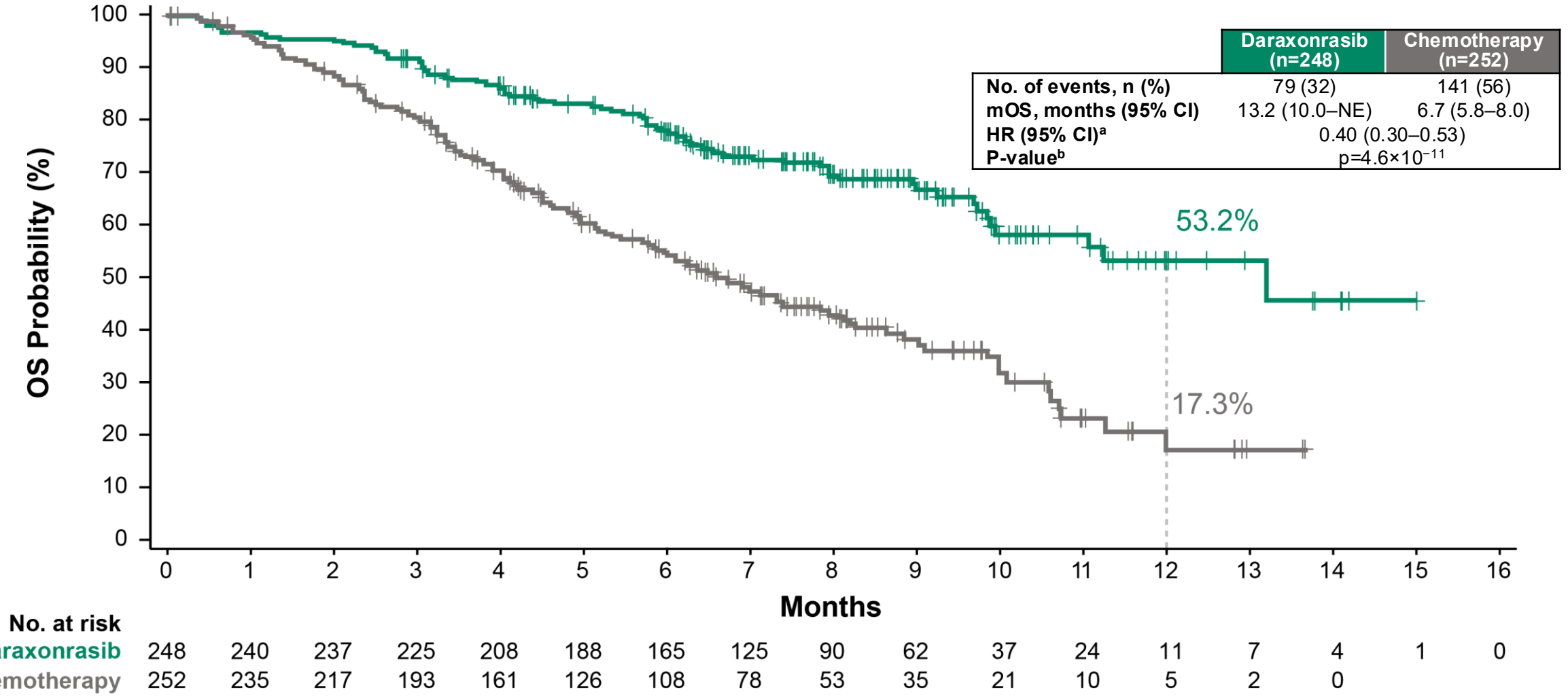

| | Daraxonrasib (n=228) | Chemotherapy (n=231) |
|---|---|---|
| **No. of events, n (%)** | 72 (32) | 127 (55) |
| **mOS, months (95% CI)** | 13.2 (10.0–NE) | 6.6 (5.4–8.2) |
| **HR (95% CI)[a]** | 0.40 (0.30–0.54) | |
| **P-value[b]** | $p=5.9\times10^{-10}$ | |

**Data cutoff: 10 Feb 2026.** Median (range) follow-up time was 8.5 (3.2−15.9) months.

m, median; NE, not estimable; OS, overall survival.

[a]HRs and 95% CIs were based on the stratified Cox model with Efron's method of tie handling. [b]P-values were calculated using the stratified log-rank test.

# Key Secondary Endpoint: Overall Survival in the Overall Population




| | Daraxonrasib (n=248) | Chemotherapy (n=252) |
|---|---|---|
| **No. of events, n (%)** | 79 (32) | 141 (56) |
| **mOS, months (95% CI)** | 13.2 (10.0–NE) | 6.7 (5.8–8.0) |
| **HR (95% CI)[a]** | 0.40 (0.30–0.53) | |
| **P-value[b]** | $p=4.6\times10^{-11}$ | |

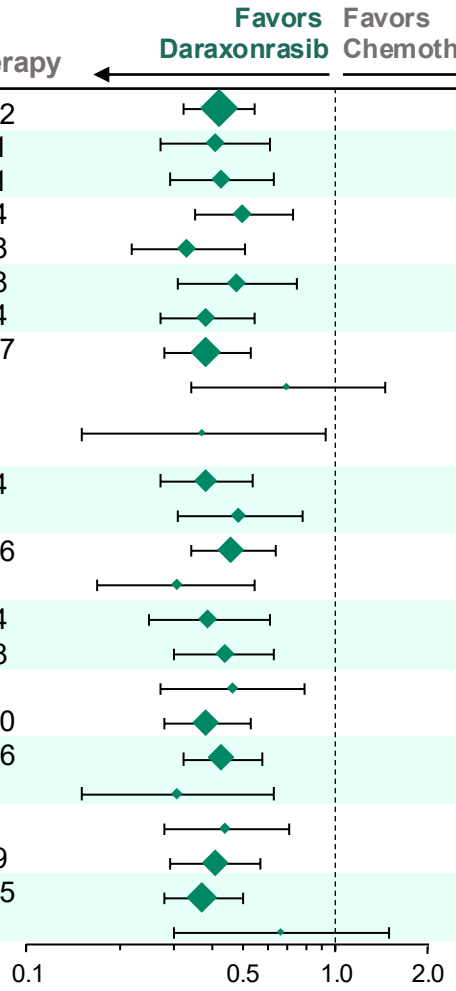
**Data cutoff: 10 Feb 2026.** Median (range) follow-up time was 8.5 (3.2−15.9) months.

m, median; NE, not estimable; OS, overall survival.

[a]HRs and 95% CIs were based on the stratified Cox model with Efron's method of tie handling. [b]P-values were calculated using the stratified log-rank test.

# Overall Survival Subgroup Analysis – Overall Population

| Population | | Events/N Daraxonrasib | Events/N Chemotherapy | HR (95% CI)[a] |
|---|---|---|---|---|
| **Overall** | | 79/248 | 141/252 | 0.42 (0.32–0.55) |
| **Age, years** | <65 | 37/114 | 68/121 | 0.41 (0.27–0.61) |
| | ≥65 | 42/134 | 73/131 | 0.43 (0.29–0.63) |
| **Sex** | Male | 45/131 | 77/144 | 0.50 (0.35–0.73) |
| | Female | 34/117 | 64/108 | 0.33 (0.22–0.51) |
| **ECOG PS** | 0 | 33/129 | 51/118 | 0.48 (0.31–0.75) |
| | 1[b] | 46/119 | 90/134 | 0.38 (0.27–0.55) |
| **Tumor RAS mutational status** | RAS G12D/V | 59/197 | 110/197 | 0.38 (0.28–0.53) |
| | Other RAS G12 | 13/31 | 17/34 | 0.70 (0.34–1.44) |
| | RAS G13 or Q61, or no RAS mutation identified | 7/20 | 14/21 | 0.37 (0.15–0.93) |
| **Metastatic disease at diagnosis** | Yes | 51/154 | 94/154 | 0.38 (0.27–0.54) |
| | No | 28/94 | 47/98 | 0.49 (0.31–0.78) |
| **Prior 5-FU-based regimen** | Yes | 62/181 | 103/186 | 0.46 (0.34–0.64) |
| | No | 17/67 | 38/66 | 0.31 (0.17–0.55) |
| **Prior gemcitabine-based regimen** | Yes | 31/110 | 61/114 | 0.39 (0.25–0.61) |
| | No | 48/138 | 80/138 | 0.44 (0.30–0.63) |
| **Prior pancreatectomy** | Yes | 21/83 | 40/92 | 0.47 (0.27–0.79) |
| | No | 58/165 | 101/160 | 0.38 (0.28–0.53) |
| **Liver metastases at baseline** | Yes | 68/174 | 113/176 | 0.43 (0.32–0.58) |
| | No | 11/74 | 28/76 | 0.31 (0.15–0.63) |
| **Peritoneal metastases at baseline** | Yes | 28/76 | 48/83 | 0.44 (0.28–0.71) |
| | No | 51/172 | 93/169 | 0.41 (0.29–0.57) |
| **CA19-9 level at baseline** | ≥40 U/mL | 70/211 | 121/195 | 0.37 (0.28–0.50) |
| | <40 U/mL | 9/36 | 19/56 | 0.67 (0.30–1.49) |




**Data cutoff: 10 Feb 2026.**

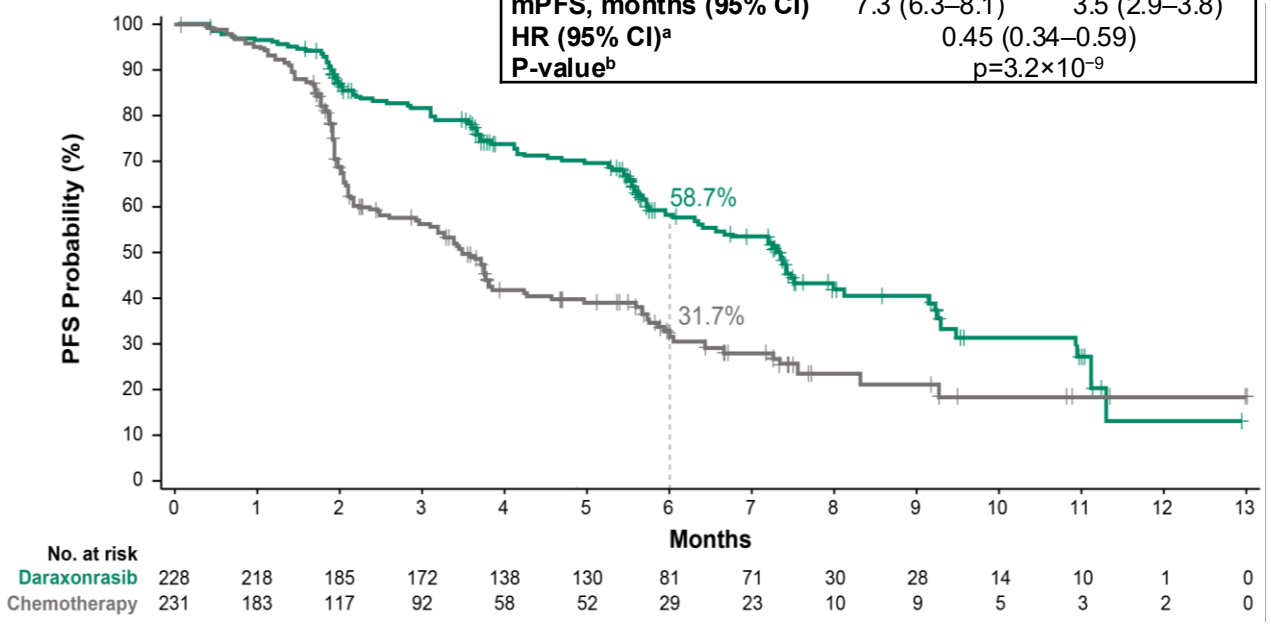
5-FU, 5-fluorouracil; ECOG, Eastern Cooperative Oncology Group; PS, performance status.

[a]The size of diamonds is relative to the size of the population in each subgroup. HRs and 95% CIs were based on the unstratified Cox model with Efron's method of tie handling. [b]Three patients (including 2 patients randomized to the daraxonrasib arm and 1 patient randomized to the chemotherapy arm) had ECOG PS >1 at baseline. These patients were not dosed due to no longer meeting trial eligibility criteria.

# Progression-Free Survival by BICR

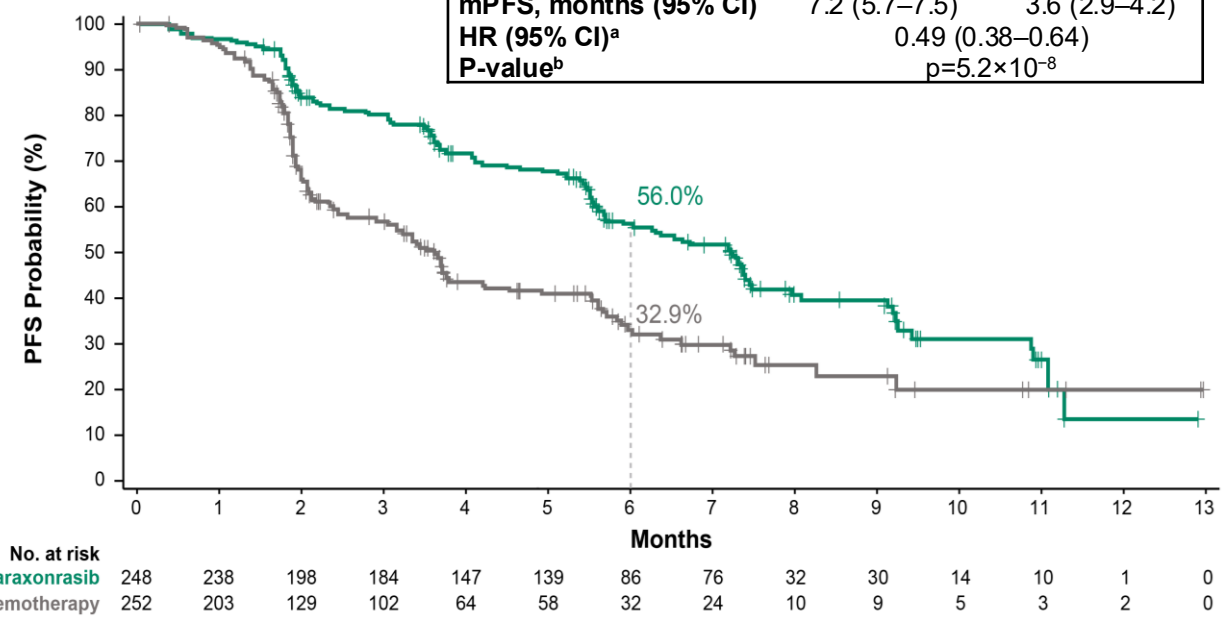
## RAS G12 Population

| | Daraxonrasib (n=228) | Chemotherapy (n=231) |
|---|---|---|
| **No. of events, n (%)** | 112 (49) | 121 (52) |
| **mPFS, months (95% CI)** | 7.3 (6.3–8.1) | 3.5 (2.9–3.8) |
| **HR (95% CI)[a]** | 0.45 (0.34–0.59) | |
| **P-value[b]** | $p=3.2\times10^{-9}$ | |




## Overall Population

| | Daraxonrasib (n=248) | Chemotherapy (n=252) |
|---|---|---|
| **No. of events, n (%)** | 127 (51) | 130 (52) |
| **mPFS, months (95% CI)** | 7.2 (5.7–7.5) | 3.6 (2.9–4.2) |
| **HR (95% CI)[a]** | 0.49 (0.38–0.64) | |
| **P-value[b]** | $p=5.2\times10^{-8}$ | |

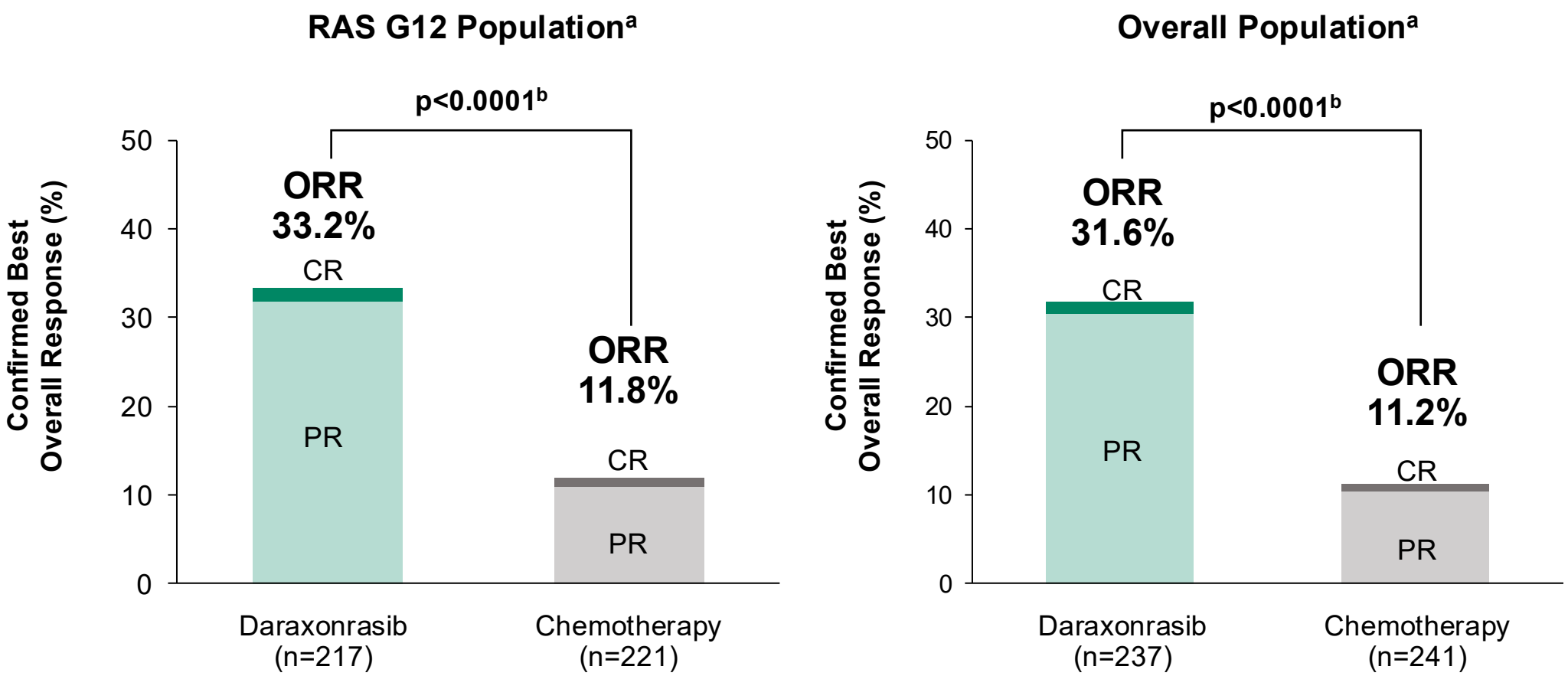



**Data cutoff: 10 Feb 2026.** Median (range) follow-up time was 8.5 (3.2−15.9) months.

BICR, blinded independent central review; m, median; PFS, progression-free survival.

[a]HRs and 95% CIs were based on the stratified Cox model with Efron's method of tie handling. [b]P-values were calculated using the stratified log-rank test.

# Confirmed Objective Response Rate by BICR




**Data cutoff: 10 Feb 2026.**

BICR, blinded independent central review; CR, complete response; ORR, objective response rate; PR, partial response.

[a]Only patients with measurable disease at baseline per BICR were included in the ORR analysis. [b]P-values were calculated using stratified Cochran-Mantel-Haenszel chi-square test.

# Safety Overview

| | Daraxonrasib (n=241) | Chemotherapy (n=214) |
|---|---|---|
| **Median time on treatment, months (range)** | 6.2 (0.03-14.1) | 1.5-3.2[a] (0.03-12.9) |
| **Any TEAEs, n (%)** | 241 (100) | 209 (97.7) |
| **Any TRAEs, n (%)** | 236 (97.9) | 200 (93.5) |
| TRAEs leading to dose interruption | 135 (56.0) | 118 (55.1) |
| TRAEs leading to dose reduction | 87 (36.1) | 123 (57.5) |
| TRAEs leading to discontinuation | 3 (1.2)[b] | 24 (11.2) |
| Grade ≥3 TRAEs | 105 (43.6) | 123 (57.5) |
| Serious TRAEs | 26 (10.8) | 40 (18.7) |
| Grade 5 TRAEs | 1 (0.4)[c] | 0 |

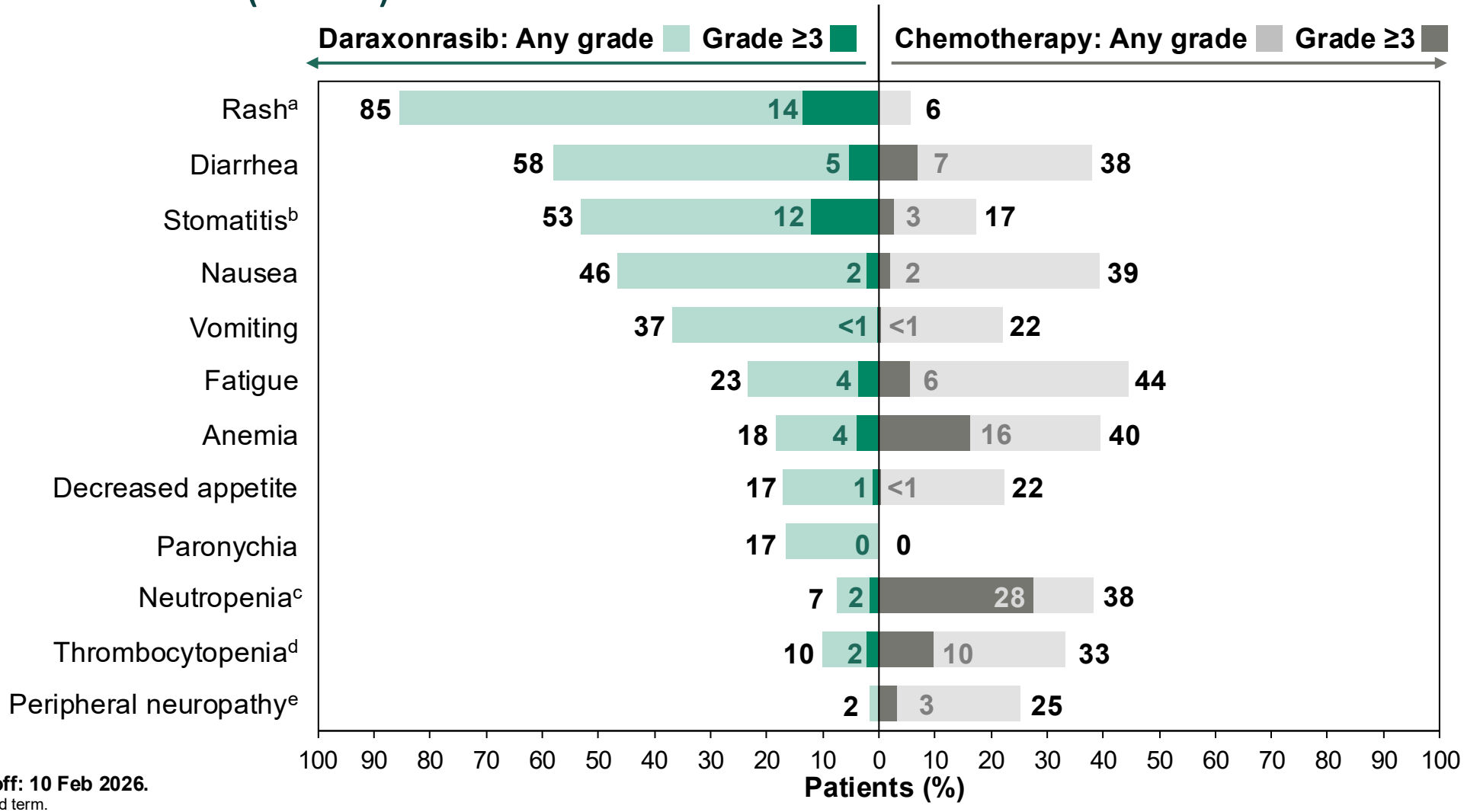
- Median dose intensity was 93.1% with daraxonrasib and 65.3-95.0%[a] across chemotherapy regimens
- Most common (≥5%) TRAEs leading to dose reduction
  - Daraxonrasib: rash (17.4%) and stomatitis (6.6%)
  - Chemotherapy: neutropenia (16.8%), thrombocytopenia (13.6%), fatigue (12.6%), diarrhea (10.3%), and peripheral neuropathy (7.9%)
- TRAEs leading to treatment discontinuation
  - Daraxonrasib (1.2%): 2 patients discontinued due to maculo-papular rash and 1 due to elevated liver function enzymes
  - Chemotherapy (11.2%): the most frequent TRAE leading to discontinuation was peripheral neuropathy (4.7%)

**Data cutoff: 10 Feb 2026.**

ALT, alanine aminotransferase; AST, aspartate aminotransferase; TEAE, treatment-emergent adverse event; TRAE, treatment-related adverse event.

[a]Medians across each component of the chemotherapy regimens. [b]Two patients with grade 3 maculo-papular rash, and 1 patient with grade 3 ALT increased and grade 4 AST increased. [c]One patient in the daraxonrasib arm died from treatment-related pneumonitis.

# Most Common (>15%) Treatment-Related Adverse Events

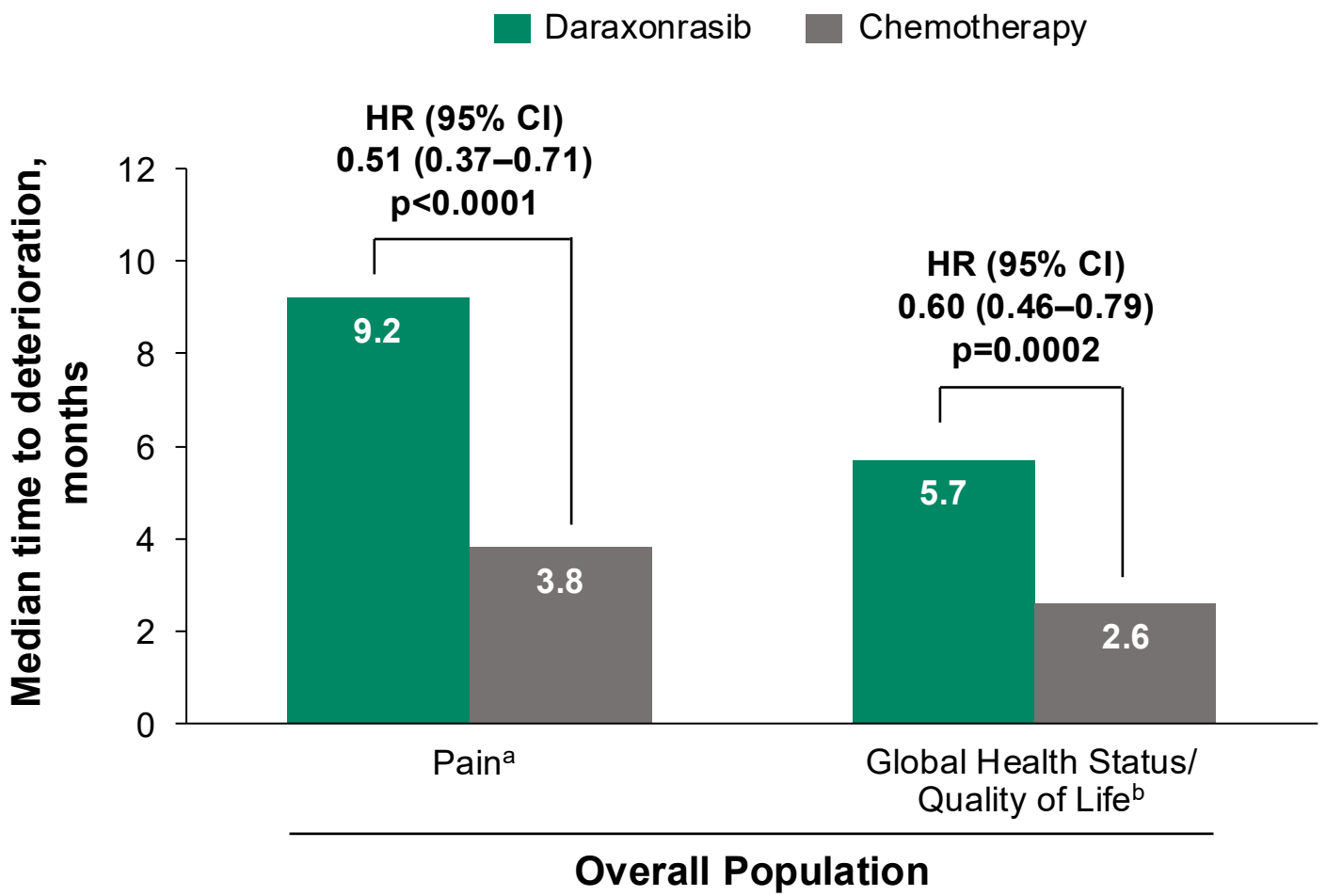



**Data cutoff: 10 Feb 2026.**

PT, preferred term.

[a]Includes PTs of acne, butterfly rash, dermatitis, dermatitis acneiform, eczema, hand dermatitis, rash, rash erythematous, rash macular, rash maculo-papular, rash papular, rash pruritic, and skin ulcer. [b]Includes PTs of aphthous ulcer, mucosal inflammation, mucosal ulceration, and stomatitis. [c]Includes PTs of febrile neutropenia, leukopenia, neutropenia, neutrophil count decreased, and white blood cell count decreased. [d]Includes PTs of platelet count decreased and thrombocytopenia. [e]Includes PTs of neuralgia, neuropathy peripheral, peripheral motor neuropathy, peripheral sensory neuropathy, and polyneuropathy.

# Patient-Reported Outcomes




- EORTC QLQ-PAN26 and EORTC QLQ-C30 questionnaires were administered on day 1 of each cycle and at end of treatment visit
- Daraxonrasib significantly delayed time to deterioration in both symptom of pain and global health status/quality of life compared with chemotherapy

**Data cutoff: 10 Feb 2026.**

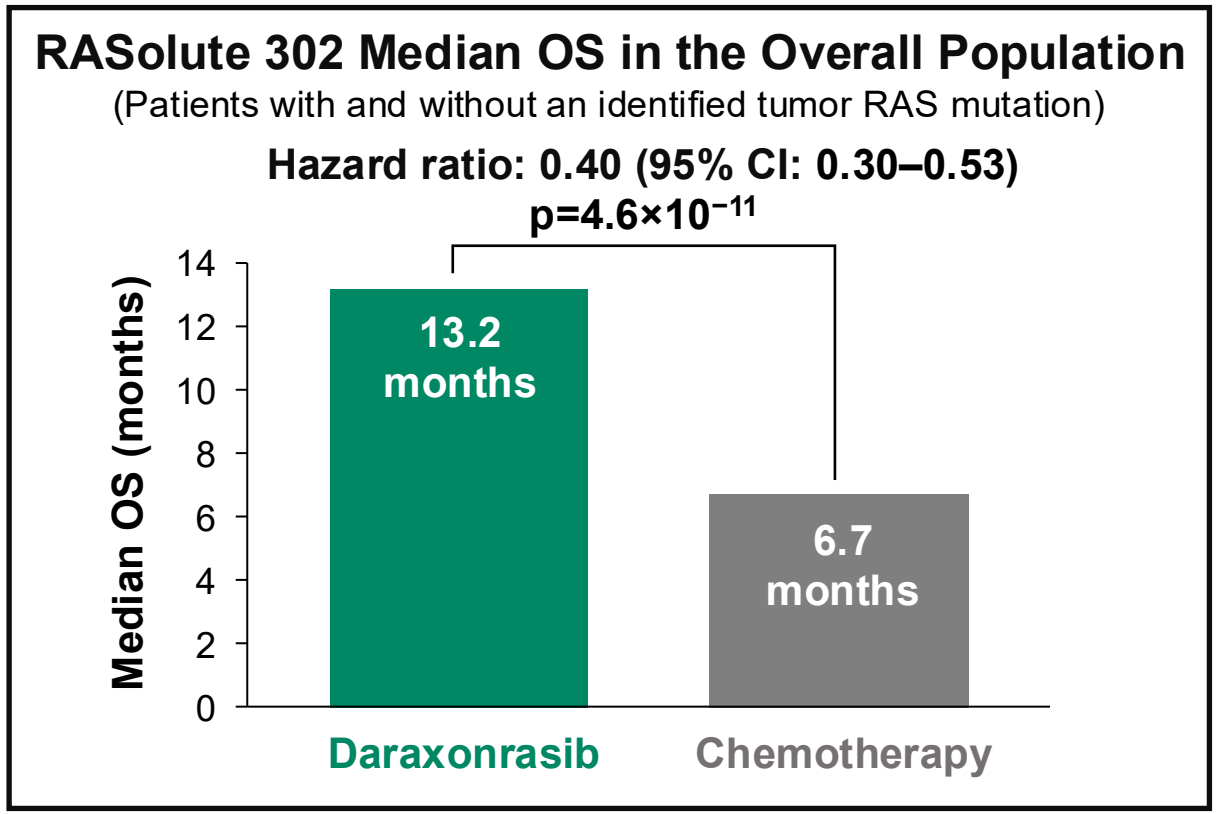
EORTC QLQ-C30, European Organisation for the Research and Treatment of Cancer Quality of Life Questionnaire; EORTC QLQ-PAN26, European Organisation for the Research and Treatment of Cancer Quality of Life Questionnaire – Pancreatic Cancer Module.

[a]Time to deterioration in clinically relevant symptom of pain was defined as the time from randomization to an increase of at least 10 points from baseline in pain, or death, whichever occurs first, in the EORTC QLQ-PAN26 pain scale. [b]Time to deterioration in global health status/quality of life was defined as the time from randomization to a decrease of at least 10 points from baseline in global health status/quality of life, or death, whichever occurs first, in EORTC QLQ-C30.

# Conclusions

**Daraxonrasib (300 mg PO QD):**

- Demonstrated statistically significant and clinically meaningful improvements in OS, PFS, and ORR vs standard cytotoxic chemotherapy
- Showed a consistent treatment effect in patients with and without an identified tumor RAS mutation
- Exhibited a manageable safety profile with no unexpected safety findings[1,2]
- Significantly delayed time to deterioration for the symptom of pain and global health status/quality of life




**Results support daraxonrasib as the new standard of care for patients with previously treated metastatic PDAC**

ORR, objective response rate; OS, overall survival; PDAC, pancreatic adenocarcinoma; PFS, progression-free survival; PO, oral administration; QD, once daily.

1. Garrido-Laguna I, et al. J Clin Oncol. 2025;43:722; 2. Wolpin B, et al. N Engl J Med. 2026;394:1790-1802.

# Implications and Next Steps

# Why RASolute 302 Matters

- **First targeted therapy to demonstrate significant clinical benefit** in patients with metastatic PDAC, with unprecedented impact in OS, PFS, ORR and QoL
- Compelling basis for a potential **new standard of care** in previously treated PDAC
- Clinical proof of **RAS as the primary driver** of PDAC
- **Clinical validation** of targeted RAS(ON) inhibition as a therapeutic strategy in PDAC with important implications for RAS-addicted tumors broadly
- Establishes innovative tri-complex inhibitor pipeline as **foundation for global franchise** in targeted medicines for patients living with RAS cancers



OS, overall survival; PFS, progression-free survival; ORR, objective response rate; QoL, quality of life; PDAC, pancreatic ductal adenocarcinoma.

# Advancing with Urgency to Bring Daraxonrasib to Patients

## Working urgently to make daraxonrasib available to eligible patients with previously treated metastatic PDAC

- Treatment crossover offered to patients in RASolute 302
- FDA-cleared Early Access Program operational and accessible to U.S.-licensed physicians on behalf of eligible patients

## Working aggressively toward global regulatory submissions, beginning with the FDA under CNPV

- Advancing multiple components of NDA through a rolling submission
- Sequential filings planned to international regulatory authorities

## State-of-the-art organization and capabilities positioned to drive successful commercialization

- Core capabilities established in the U.S. with expansion underway in priority international markets
- Experienced team with strong track record of success across range of oncology builds and launches



PDAC, pancreatic ductal adenocarcinoma; CNPV, Commissioner's National Priority Voucher; NDA, New Drug Application.

# Building an Industry-Leading Franchise for RAS-Addicted Cancers

# Pipeline Led by Four Pioneering, Clinical-Stage, RAS(ON) Inhibitors



| COMPOUND | FOCUS | STUDY DETAILS | EARLY CLIN. DEVELOPMENT | REGISTRATIONAL TRIAL |
|---|---|---|---|---|
| **Daraxonrasib (MULTI)** | PDAC | RASolute 302 — 2L metastatic | | |
| | | RASolute 303 — 1L metastatic | | |
| | | RASolute 304 — Adjuvant in resectable | | |
| | NSCLC | RASolve 301 — 2L/3L metastatic | | |
| | | 1L metastatic | | *Planning ongoing* |
| | Solid tumors | + SOC, RAS(ON) inhibitor doublets or other investigational agents | | |
| **Zoldonrasib (G12D)** | PDAC | RASolute 305 — 1L metastatic | | |
| | | RASolute 309 — 1L metastatic | | *Phase 3 initiation planned* |
| | NSCLC | RASolve 308 — 1L metastatic | | *Initiation pending* |
| | Solid tumors | + SOC, RAS(ON) inhibitor doublets or other investigational agents | | |
| **Elironrasib (G12C)** | Solid tumors | Monotherapy | | |
| | | + SOC, RAS(ON) inhibitor doublets or other investigational agents | | |
| **RMC-5127 (G12V)** | Solid tumors | Monotherapy | | |

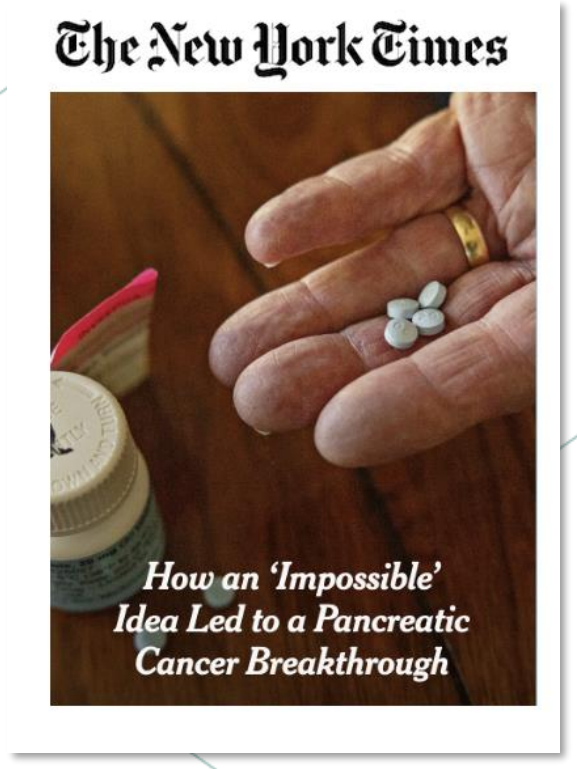
Additional clinical development opportunities include RAS(ON) mutant-selective inhibitors RMC-0708 (Q61H) and RMC-8839 (G13C) and additional novel targeted approaches for patients with RAS-addicted cancers.

# **Industry-Leading Capabilities** | Advancing Targeted Treatment Regimens for Patients with RAS-Addicted Cancers

- Proprietary tri-complex discovery platform targeting the oncogenic (or “ON”) state of RAS
- Robust clinical development programs and expertise to maximize impact for patients
- Expanding commercialization and operational capabilities to ensure delivery of successful launches and change global standards of care


Virtuous cycle of innovation driven by bench, bedside and commercial insights

# **Our Why** | Building on Strong Pillars for Growing Transformative Patient Impact




*Published: May 12, 2026* [1]


*Published: April 21, 2026* [2]


*Published: April 14, 2026*


*Published: April 15, 2026*

RAS-addicted cancers are among the **most common and difficult-to-treat** cancers in need of new targeted medicines

Extensive clinical evidence has shown that RAS(ON) inhibitors **have the potential to improve outcomes** and **change global standards of care** for patients living with such cancers

**Compelling opportunities** for further advancement through drug combinations and continuing **product innovation**

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Image Credits: [1] KIM RAFF/The New York Times/Redux; [2] Photo by JEAN-FRANCOIS MONIER/AFP via Getty Images

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# On Target to Outsmart Cancer®