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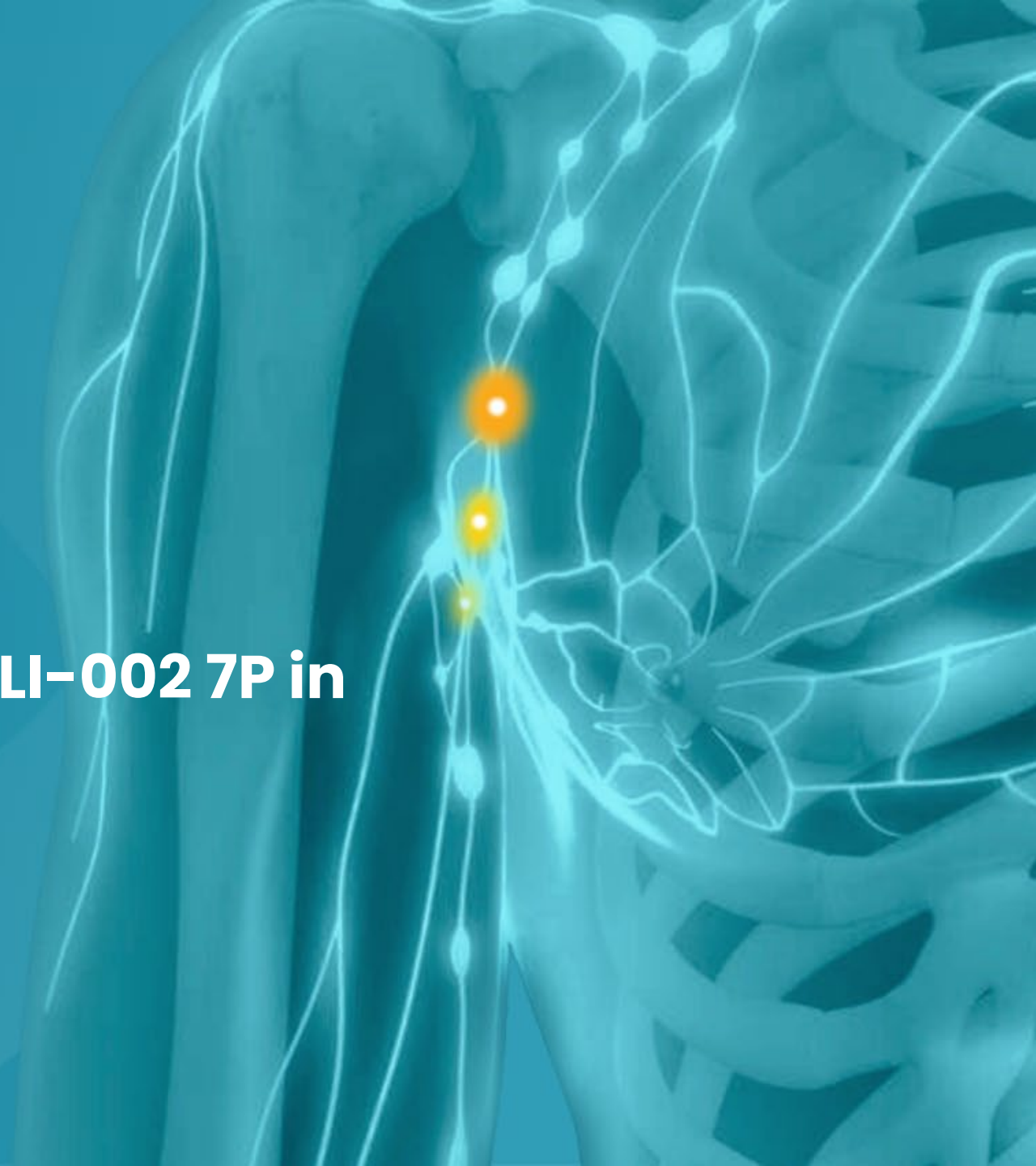
AMPLIFY-7P
Defining the Path Forward for ELI-002 7P in Metastatic mKRAS PDAC

JUNE 24, 2026



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Agenda		Speaker
01	**Introduction**	**Christopher Haqq,** M.D., Ph.D., Executive Vice President, Head of Research and Development and Chief Medical Officer
02	**Review of Historical mPDAC Studies**	**Zev Wainberg,** M.D., Professor of Medicine, UCLA, Co-Director of the UCLA GI Oncology Program
03	**mPDAC Complete Responses**	**Peter Hosein**, M.D., Professor of Clinical Medicine, Sylvester Comprehensive Cancer Center, University of Miami
04	**Translational Medicine Data**	**Christopher Haqq,** M.D., Ph.D.
05	**Phase 1 Strategy**	**Christopher Haqq,** M.D., Ph.D.
06	**Conclusion**	**Christopher Haqq,** M.D., Ph.D.
07	**Discussion and QnA**	**Zev Wainberg,** M.D., **Peter Hosein**, M.D., and **Christopher Haqq,** M.D., Ph.D.

Peter Hosein, M.D.

Zev Wainberg, M.D.

Chris Haqq, M.D., Ph.D.

Christopher Haqq, M.D., Ph.D.,

Executive Vice President, Head of Research and Development and Chief Medical Officer

Multiple Confirmed Complete Responses Observed Following ELI-002 7P Treatment, Subsequent Therapy in metastatic PDAC

Hypothesis: ELI-002 7P primes mKRAS-specific immunity to enhance responsiveness to subsequent treatment

Promising preliminary responses	• **100% confirmed** complete radiographic and metabolic **responses in three patients** who received ELI-002 7P treatment and received subsequent nivolumab-based therapy • Subsequent therapy included chemotherapy, checkpoint inhibition, and radiation • Prospective study needed to confirm these observations
Supports differentiated therapeutic potential	• **Complete responses are rare** in metastatic PDAC, particularly in MSS/MMR-proficient disease where checkpoint inhibitors have shown limited activity • These observations are therefore notable relative to historical experience

Elicio believes these observations provide preliminary clinical support for evaluating ELI-002 7P in combination with checkpoint inhibition in metastatic mKRAS pancreatic cancer.

MSS: Micro-satellite stable; MMR: Mismatch Repair

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Zev Wainberg, M.D.,

Professor of Medicine, UCLA, Co-Director of the UCLA GI Oncology Program

Complete Responses Remain Rare in Metastatic Pancreatic Cancer Across Therapeutic Modalities

Published studies evaluating chemotherapy, checkpoint inhibitors, and targeted therapies generally report complete response rates ranging from 0–8%

Regimen / Study	Population	CR signal
FOLFIRINOX	First Line (1L) mPDAC	**0.6% (1 / 171)** [1]
Nivo + mFOLFIRINOX	First Line (1L) mPDAC	**0% (0 / 31)** [2]
Daraxonrasib + GnP	First Line (1L) RAS-mutant mPDAC	**2.5% (1 / 40)** [3]
nP + Cisplatin + Gemcitabine	First Line (1L) stage IV mPDAC	**8% (2 / 24)** [4]

Multiple complete responses remain uncommon even among the most active regimens evaluated in metastatic PDAC.

*[1] Conroy **NEJM** 2011; [2] Morizane **BJC Rep** 2024; [3] Revolution Medicines April 21 2026 PR; [4] Jameson **JAMA Oncol** 2020*
GnP: Gemcitabine Nab-paclitaxel

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Immunotherapy with Chemotherapy or Radiation Has Not Been Effective

Historical rates of complete response: **2-6%** for Nivo + GnP, **0%** for Nivo + SBRT

Regimen / Study	Population	CR Signal
Nivo + GnP (PRINCE)	First Line (1L) mPDAC	**2% (1 / 50)** [1]
Nivo + GnP	First Line (1L) mPDAC	**3% (1 / 34)** [2]
Nivo + SBRT	Second Line (2L) mPDAC	**0% (0 / 41)** [3]
Durva + SBRT	Second Line (2L) mPDAC	**0% (0 / 16)** [4]

Multiple complete responses remain uncommon with immunotherapy combinations.

*[1] Wainberg, **Clinical Cancer Research**. 2020; [2] Padron, **Nature Medicine**. 2022; CR was not confirmed [3] Chen, **J Clinical Oncology**. 2022; [4] Xie, **Clinical Cancer Research**. 2020*

GnP: Gemcitabine Nab-paclitaxel; SBRT: Stereotactic Body Radiation Therapy; CR: Complete Response

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Peter Hosein, M.D.,

Professor of Clinical Medicine, Sylvester Comprehensive Cancer Center, University of Miami

Complete Responses Observed Following ELI-002 7P and Subsequent Checkpoint Inhibition

Three patients who recurred following ELI-002 7P treatment subsequently achieved complete radiographic response, complete metabolic response, and biomarker normalization after nivolumab-based therapy

- 3 / 3 Patients (100%) Achieved Complete Responses (CR + CMR)
- All 3 Patients Were MSS / MMR-Proficient
- Persistence of mKRAS-specific T cells observed post-chemo and CPI
- mKRAS-Specific CD4+ and CD8+ T Cell Responses
- 3 / 3 Patients with Antigen Spreading Observed
- 2 / 3 Patients with Complete Responses Maintained ≥9 Months

These observations provide preliminary clinical support for evaluating ELI-002 7P in combination with checkpoint inhibition in non-liver metastatic mKRAS pancreatic cancer.

CR: Complete Response; CMR: Complete Metabolic Response; MSS: Micro-satellite stable; MMR: Mismatch Repair; CPI: Checkpoint Inhibition

Complete Responses Were Confirmed and Durable[1]

Durable complete responses observed following subsequent nivolumab-based therapy in three patients previously treated with ELI-002 7P

Patient	Tumor mKRAS	Nivo start → PR / CR	DOR [1]	Current Status
Patient 1	G12D	**6.1 mo (CR)**	**2.2 mo (8.3 mo PFS)**	PD, SBRT, FOLFOX-Nivo, Ipi/Nivo, ERAS-0015
Patient 2	G12V	**1.5 mo (PR) 8.9 mo (CR)**	**11.6 mo (13.4 mo PFS)**	PD, GnP-Nivo
Patient 3	G12D	**2.5 mo (CR)**	**9.2 mo (ongoing)**	NED

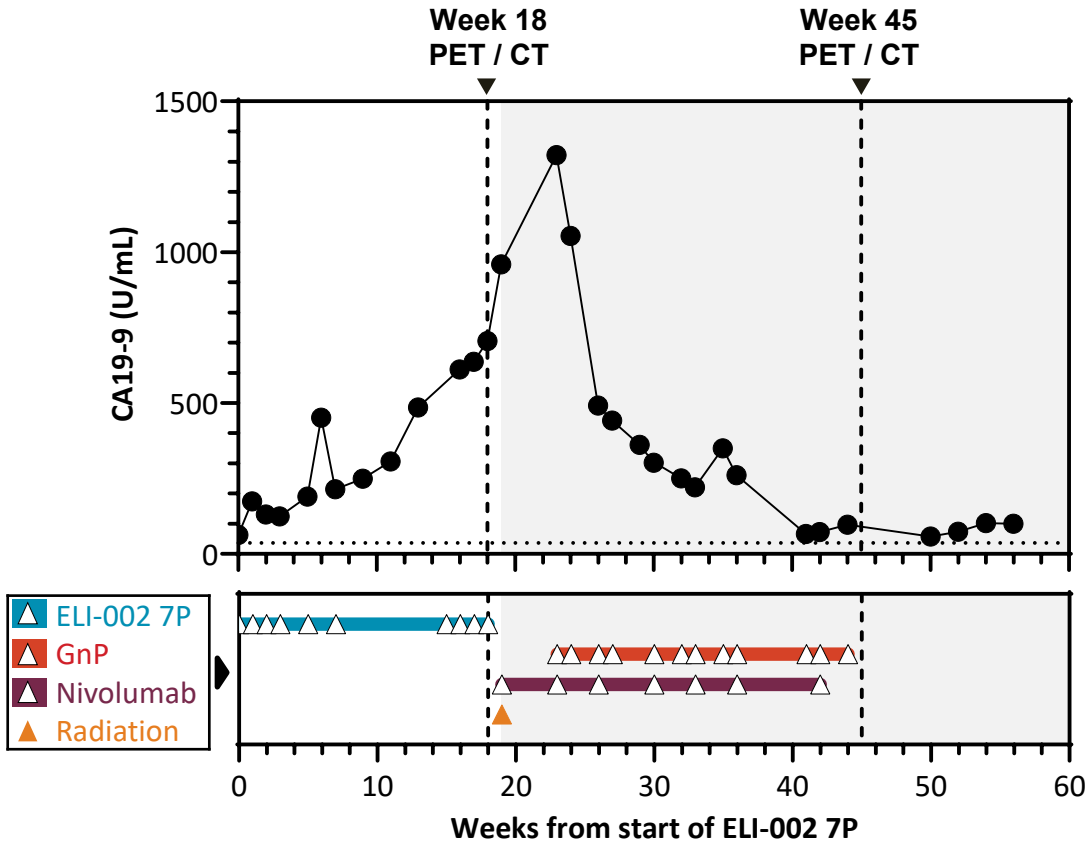
These observations support further investigation of the relationship between ELI-002 7P treatment and subsequent clinical outcomes.

[1] June 21, 2026; GnP: Gemcitabine Nab-paclitaxel; CR: Complete Response; PR: Partial Response; PD: Progressive Disease; SBRT: Stereotactic Body Radiation Therapy; NED: No evidence of disease; PFS: Progression-free survival; DOR: Duration of response

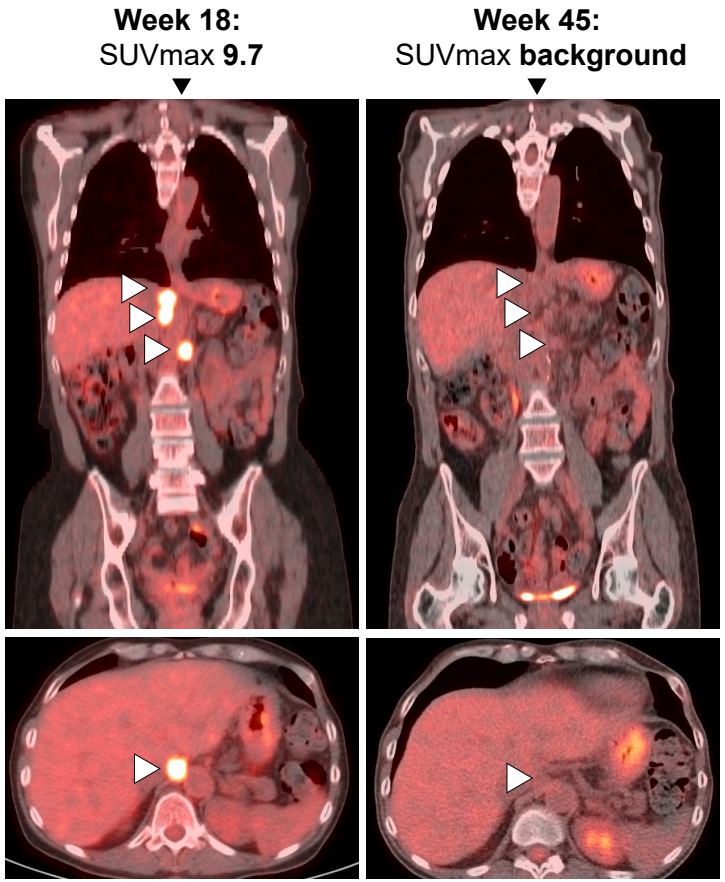
Patient 1: Complete Response Following ELI-002 7P and Subsequent Nivolumab

Complete Radiographic, Metabolic, and Biomarker Resolution of Multifocal Para-esophageal and Mesenteric Nodes

Patient 1: CA19-9 Tumor Biomarker Values, Treatment Timeline



Patient 1: PET / CT



Para-esophageal and mesenteric nodes

RECIST: Complete Response (CR)

PERCIST: Complete Metabolic Response (CMR)

DOR: 2.2 months

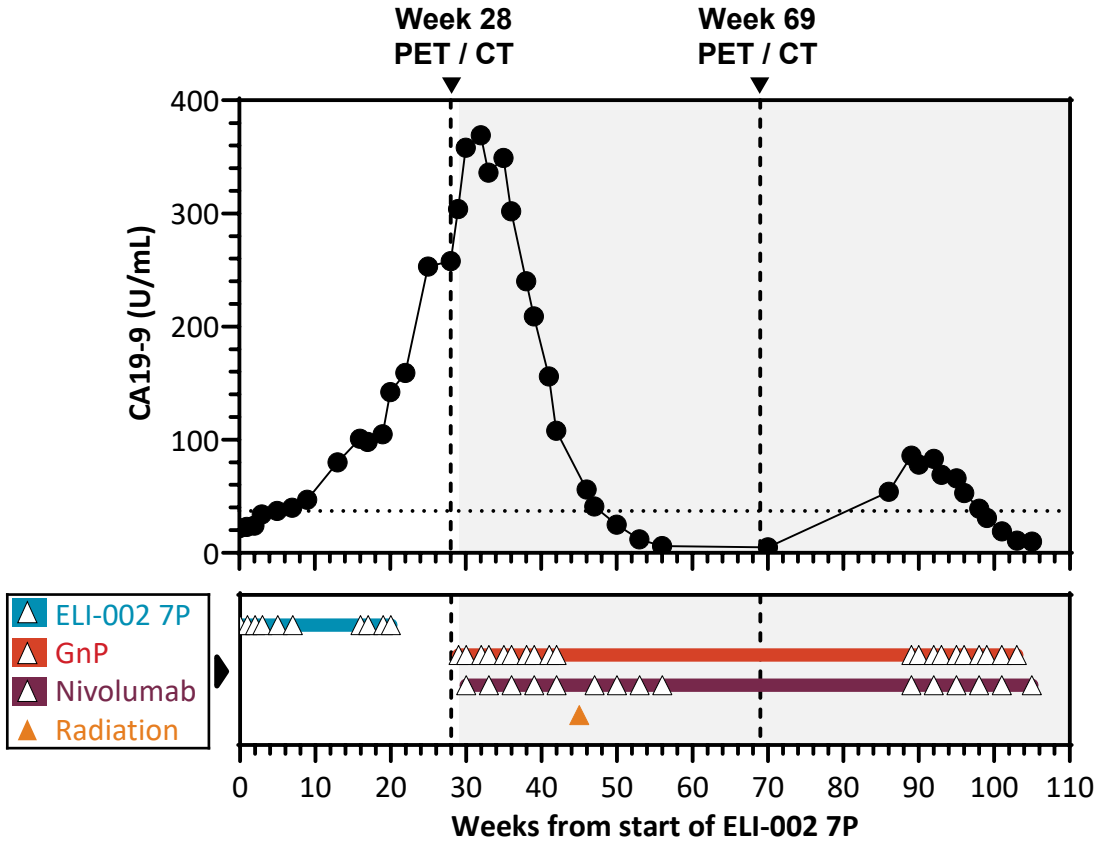
PFS: 8.3 months

GnP: Gemcitabine Nab-paclitaxel; PFS: Progression-free survival; DOR: Duration of response; CT: Computed tomography; PET: Positron emission tomography; SUVmax: Standardized uptake value

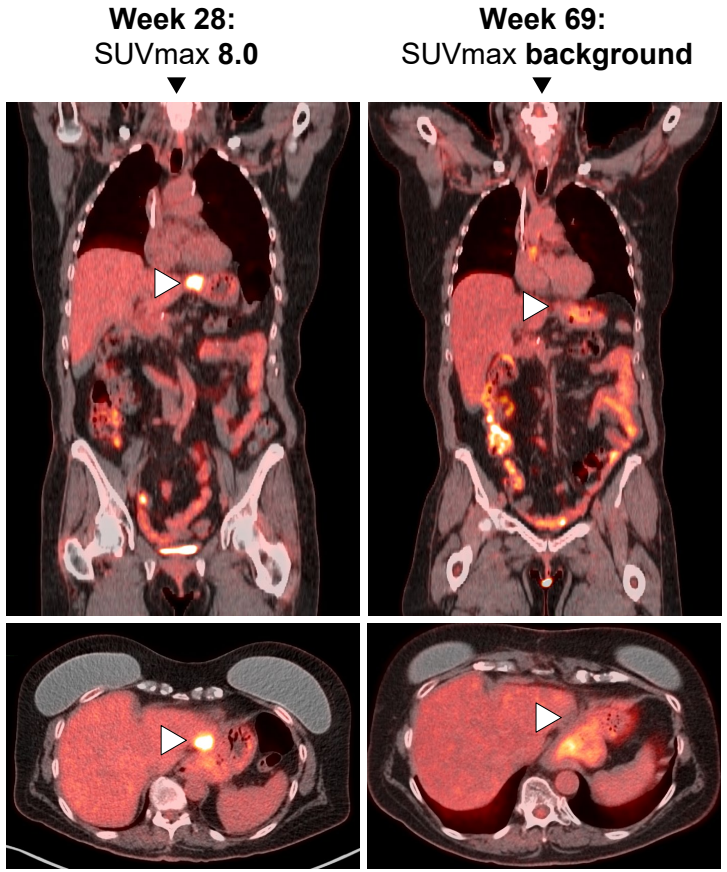
Patient 2: Complete Response Following ELI-002 7P and Subsequent Nivolumab

Complete Radiographic, Metabolic, and Biomarker Resolution of Gastro-hepatic Ligament Nodes

Patient 2: CA19-9 Tumor Biomarker Values, Treatment Timeline



Patient 2: PET / CT



Gastro-hepatic Ligament Node

RECIST: Complete Response (CR)

PERCIST: Complete Metabolic Response (CMR)

DOR: 11.6 months

PFS: 13.4 months

GnP: Gemcitabine Nab-paclitaxel; PFS: Progression-free survival; DOR: Duration of response; CT: Computed tomography; PET: Positron emission tomography; SUVmax: Standardized uptake value

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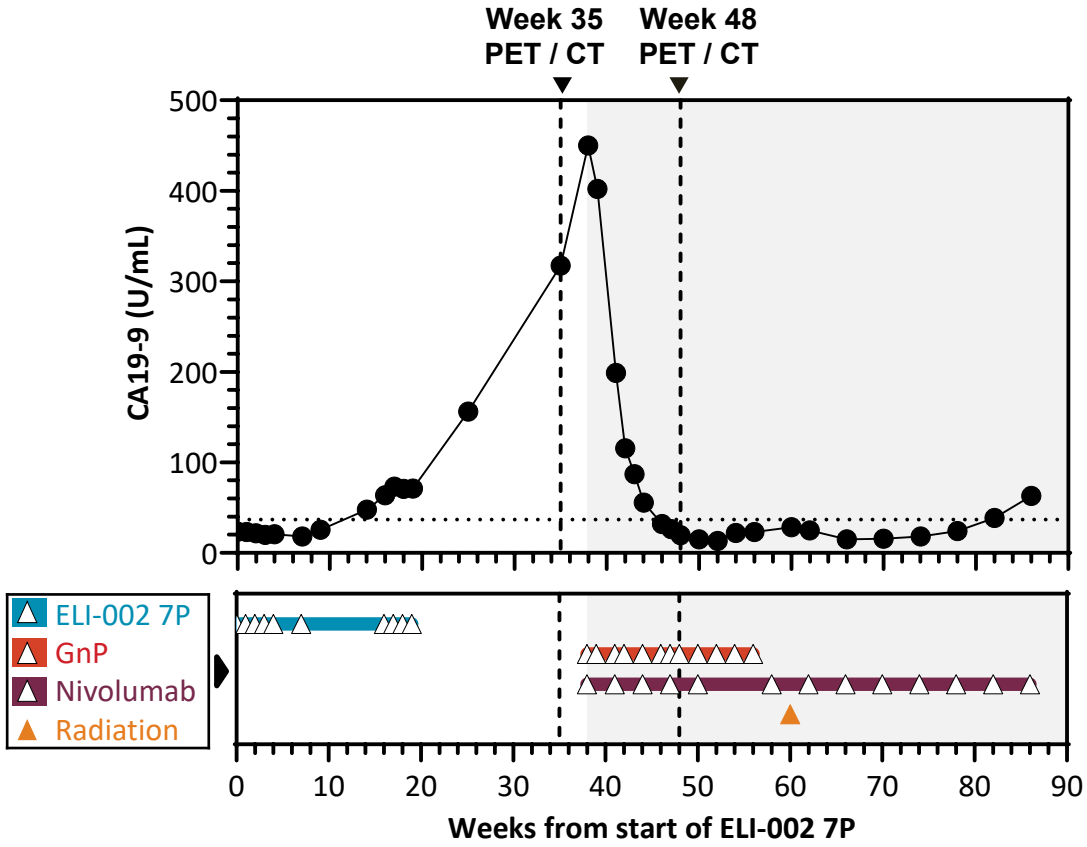
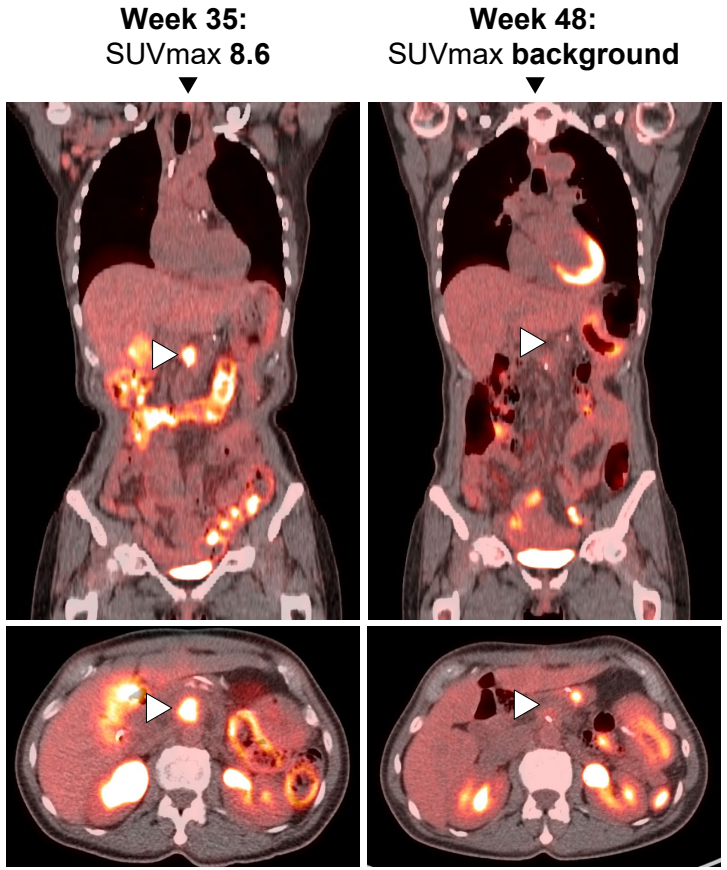
Patient 3: Complete Response Following ELI-002 7P and Subsequent Nivolumab

Complete Metabolic, and Biomarker Resolution of Mesenteric Local Recurrence

Patient 3: CA19-9 Tumor Biomarker Values, Treatment Timeline



Patient 3: PET / CT



Mesenteric Local Recurrence

RECIST: Complete Response (CR)

PERCIST: Complete Metabolic Response (CMR)

DOR: 9.2 months (ongoing)

GnP: Gemcitabine Nab-paclitaxel; PFS: Progression-free survival; DOR: Duration of response; CT: Computed tomography; PET: Positron emission tomography; SUVmax: Standardized uptake value

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Multiple Confirmed Complete Responses Observed Following ELI-002 7P Treatment and Subsequent Therapy in metastatic PDAC Warranting Further Evaluation

Complete responses following ELI-002 7P treatment and subsequent nivolumab-based therapy warrant further prospective evaluation

Executive Summary

- Three patients achieved complete radiographic, metabolic, and/or biomarker responses following nivolumab-based therapy after prior ELI-002 7P treatment
- All three patients had MSS/MMR-proficient disease, a population rarely responsive to checkpoint inhibition
- Two of three complete responses were maintained for ≥9 months, including one ongoing response
- These observations are notable relative to historical experience in metastatic PDAC
- While hypothesis-generating, the findings suggest prior ELI-002 7P treatment may influence responsiveness to subsequent therapy

These findings support prospective evaluation of ELI-002 7P in combination with checkpoint inhibition in metastatic mKRAS pancreatic cancer.

MSS: Micro-satellite stable; MMR: Mismatch Repair

Hypothesized Mechanism for Complete Responses Following ELI-002 7P and Subsequent Therapy

ELI-002 7P may prime durable mKRAS-specific immunity important for responses to subsequent therapy

Historical Observations:

Subsequent Therapy (Chemo, PD-1 Inhibition and / or Radiation) → Modulation of Tumor Microenvironment → × Deep Tumor Responses are Rare

1 ELI-002 7P Treatment → 2 mKRAS-Specific T Cell Expansion → 3 Persistent Immune Memory

4 Subsequent Therapy (Chemo, PD-1 Inhibition and / or Radiation) → 5 Modulation of Tumor Microenvironment → + Deep Tumor Responses

Observations from presented patients suggest a hypothetical mechanism for anti-tumor T cell responses and tumor modulation following ELI-002 7P treatment and subsequent nivolumab-based therapy.

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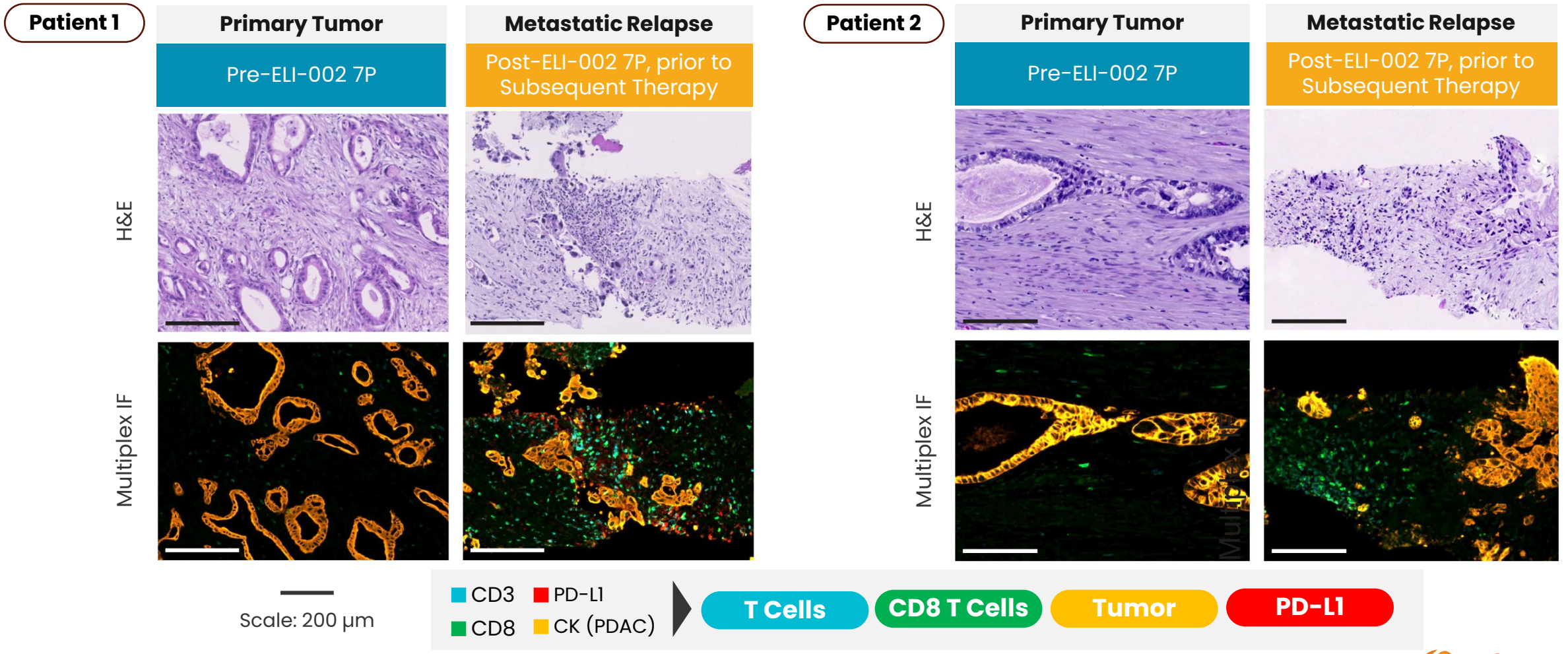
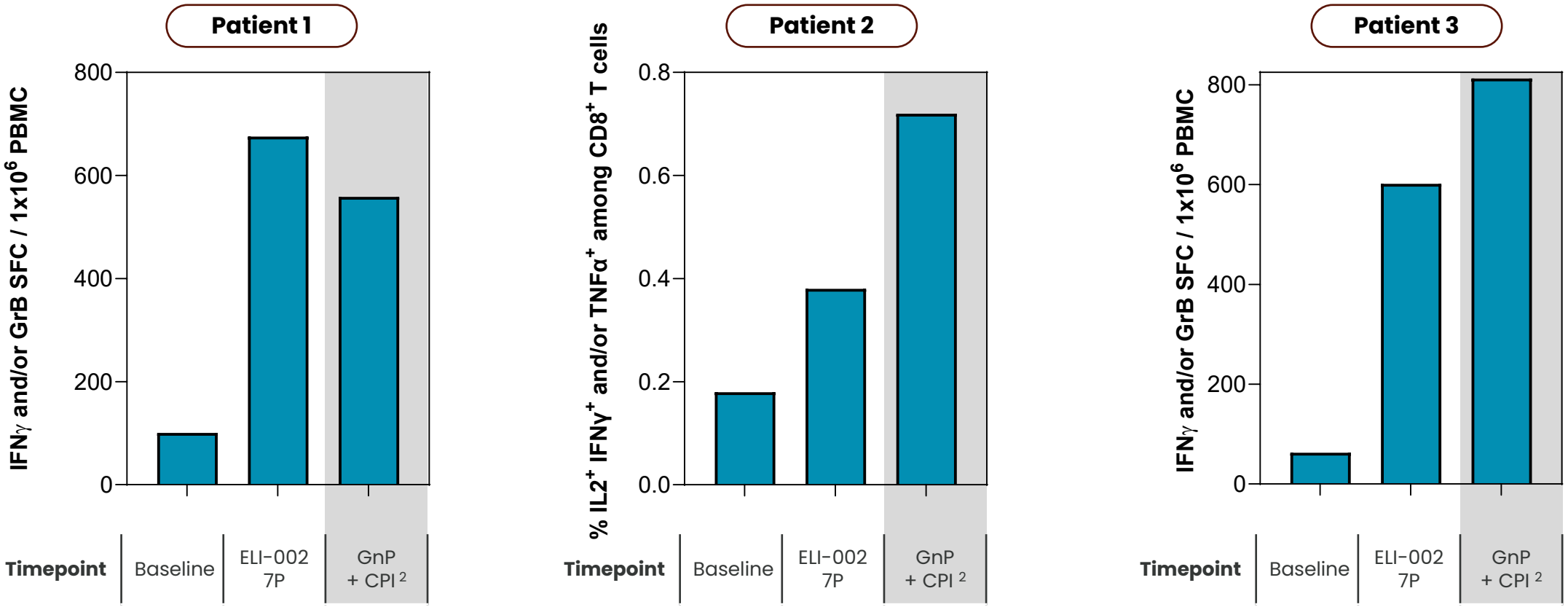
T cell Infiltration after ELI-002 7P Preceded Complete Responses

Observations of increased T cell tumor infiltration, and modulation of tumor PD-L1 following recurrence after ELI-002 7P



H&E: Hematoxylin and eosin; IF: Immunofluorescence; Patient 3 biopsy samples were not evaluable by multiplex immunofluorescence

mKRAS-specific T cell responses[1] persisted through subsequent chemotherapy and nivolumab treatment

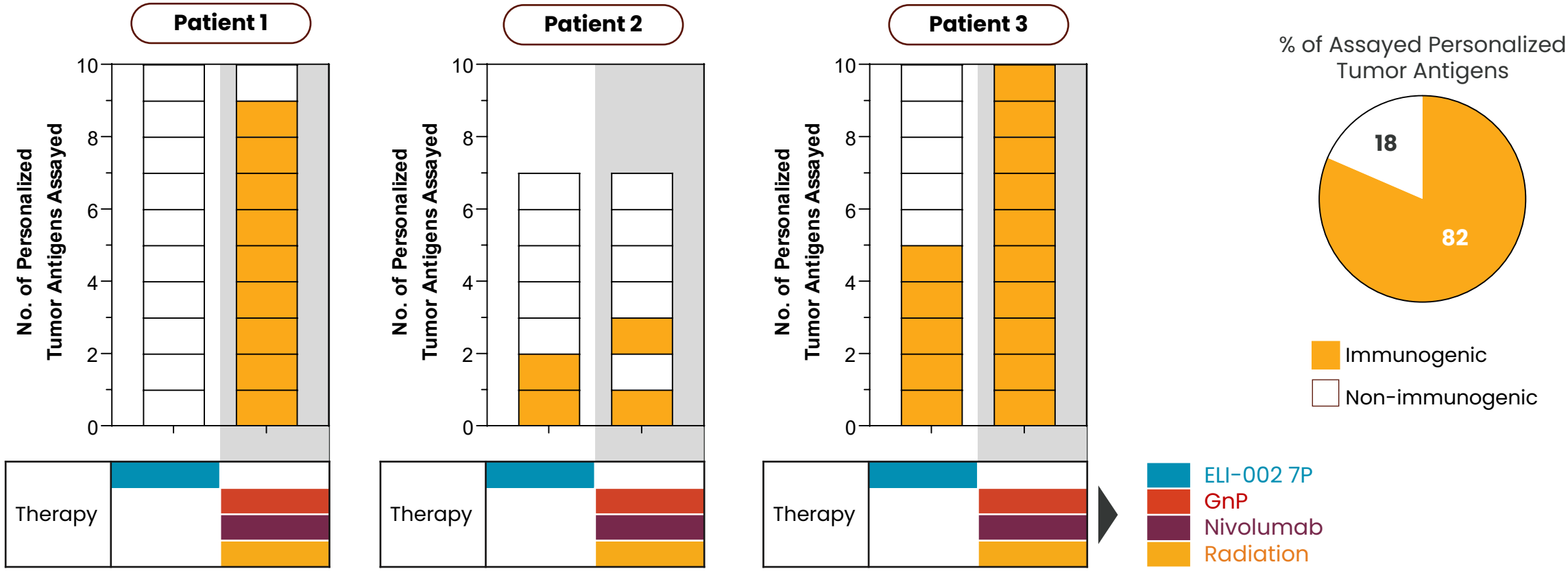


Complete responses associate with mKRAS-specific, polyfunctional, CD4+ and CD8+ T cell responses including IFNγ, TNFα, IL2, and Granzyme B

[1] Maximum ex vivo mKRAS responses determined by IFNγ/GrB Fluorospot or ICS assay at baseline, week 10 (post ELI-002 7P priming) and final post subsequent therapy timepoints [2] Subsequent therapy included GnP, nivolumab, and SBRT; GnP: Gemcitabine Nab-paclitaxel; CPI: Checkpoint Inhibition

Complete Responses Associated with Personalized T Cell Responses

3 / 3 complete responses were associated with modulation of antigen spreading T cell responses



These findings suggest ELI-002 7P-induced immunity may extend beyond targeted KRAS mutations and promote broader anti-tumor immune responses

GnP: Gemcitabine Nab-paclitaxel

Metastatic PDAC Phase 1 Development Strategy

Plans for a staged development pathway designed to evaluate ELI-002 7P + CPI across metastatic PDAC settings

Recurrent metastatic PDAC (CPI combination)

Treatment Naïve metastatic PDAC (CPI and/or RASi combinations)

→

Inform Future metastatic &
Adjuvant Phase 3 Development

Cohort expansion to be informed by clinical, biomarker, and immune-response data

Rapid Readouts:	Open label, initial efficacy signals expected within 3–6 months of study start
Biomarker Response Early Indicator	Tumor biomarker, immune response analyses may provide early evidence of biological activity
Capital Efficient:	Small cohorts designed to generate actionable data with limited capital deployment
Development Impact:	Findings may inform future metastatic combination strategies and Phase 3 design

A focused Phase 1 study has the potential to generate an early assessment of clinical activity in a setting where complete responses are rarely observed.

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Planned Phase 1 Study Evaluating ELI-002 7P in Combination with Checkpoint Inhibition in 1L mPDAC

Building on complete response observations to identify rapid clinical insights and inform future metastatic and Phase 3 development

ELIGIBILITY

- Recurrent and treatment-naïve metastatic PDAC
- ≥1 of 7 mKRAS alleles
- Measurable disease (RECIST 1.1, PERCIST)
- ECOG 0–1

STUDY REGIMEN — triplet combination

SOC chemotherapy backbone
Gem/Nab-paclitaxel

ELI-002 7P (RP2D)
Prime 6 SC doses → 8-wk rest → 4 boosters → Additional ELI-002 7P dosing until progression

Checkpoint Inhibitor (Q3 Weeks)
Until progression or toxicity

KEY PRIMARY

- **ORR** (CR + PR, RECIST 1.1)
- **CR rate**
- **DOR**
- **Safety**

KEY SECONDARY

- **mORR** (PERCIST 1.0)
- **PFS, OS**
- **Immune Responses**

Phase 1 Study Plan:

Verify signal in CPI combination setting → **Expand** into additional mPDAC combinations (e.g., RASi) and settings

SOC: Standard of care; RP2D: Recommended Phase 2 Dose; SC: Subcutaneous; ORR: Overall response rate; CR: Complete Response; PR: Partial Response

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Next Steps: Advancing Clinical Development of ELI-002 7P in Metastatic PDAC in Combination with Checkpoint Inhibition and Other Therapies

01 **Finalize Study Design**

02 **Initiate Phase 1 Study in mPDAC,** subject to funding

03 **Inform future development plans** in metastatic PDAC, and the adjuvant PDAC Phase 3 trial

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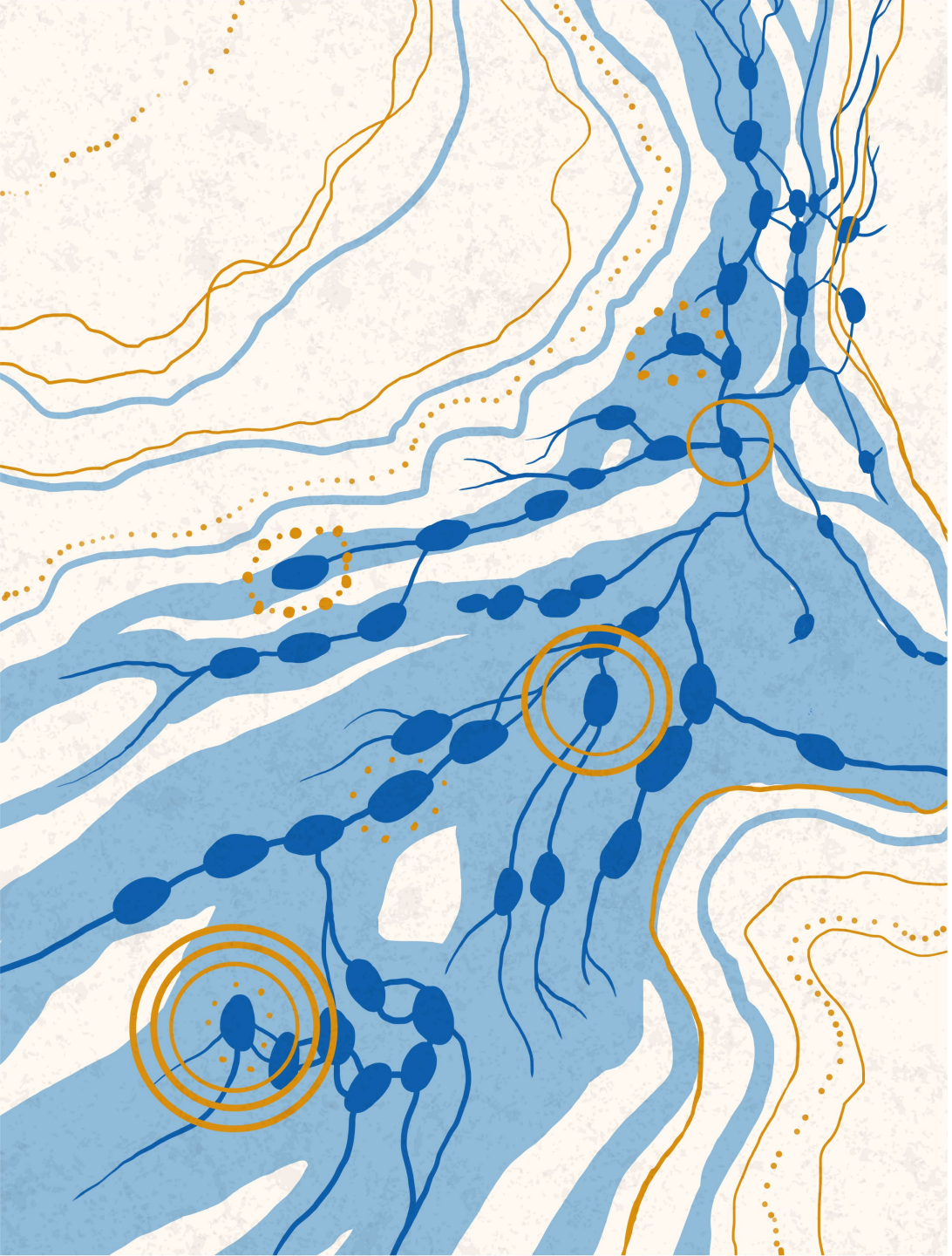

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