



AMPLIFY-7P

**Defining the Path Forward for ELI-002 7P in
Metastatic and Adjuvant mKRAS PDAC**

SEPTEMBER 2026

Nasdaq: ELTX

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Investment Highlights

Clinical-stage Biotech Developing Novel Lymph Node-targeted “off the shelf” Cancer Immunotherapies

“AMP” Platform for Lymph Node Targeting

- Designed to generate robust immune responses by **targeting lymph nodes**– the “brain center” of the immune response
- Proof-of-concept has been demonstrated in **two completed Phase 1 trials, Randomized Phase 2 trial**

ELI-002: mKRAS Cancer Immunotherapy

- **Off-the-shelf immunotherapy candidate** targeting KRAS mutations that drive 25% of solid tumors
- **Randomized Phase 2 monotherapy** in pancreatic cancer: findings support a precision medicine Phase 3 ¹
Multiple CRs observed in recurrent mPDAC patients following subsequent CPI combination ²

Value-creating catalysts and capitalization

- **Metastatic PDAC Phase 1 Initiation:** ORR, CR rate planned endpoints, RAS inhibitor +/- checkpoint combination
- **Adjuvant PDAC Phase 3 Preparation and Next Steps:** ELI-002 7P end of Phase 2 FDA meeting
- Investigator-initiated trial of **ELI-002 7P + checkpoint inhibitor in neoadjuvant PDAC**

Seasoned Management Team



Robert Connelly

Chief Executive Officer



Peter DeMuth, PhD

Chief Scientific Officer



Christopher Haqq, MD, PhD

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



Preetam Shah, MBA, PhD

Chief Strategy and
Financial Officer



Megan Filoon, JD

General Counsel,
Secretary and
Compliance Officer

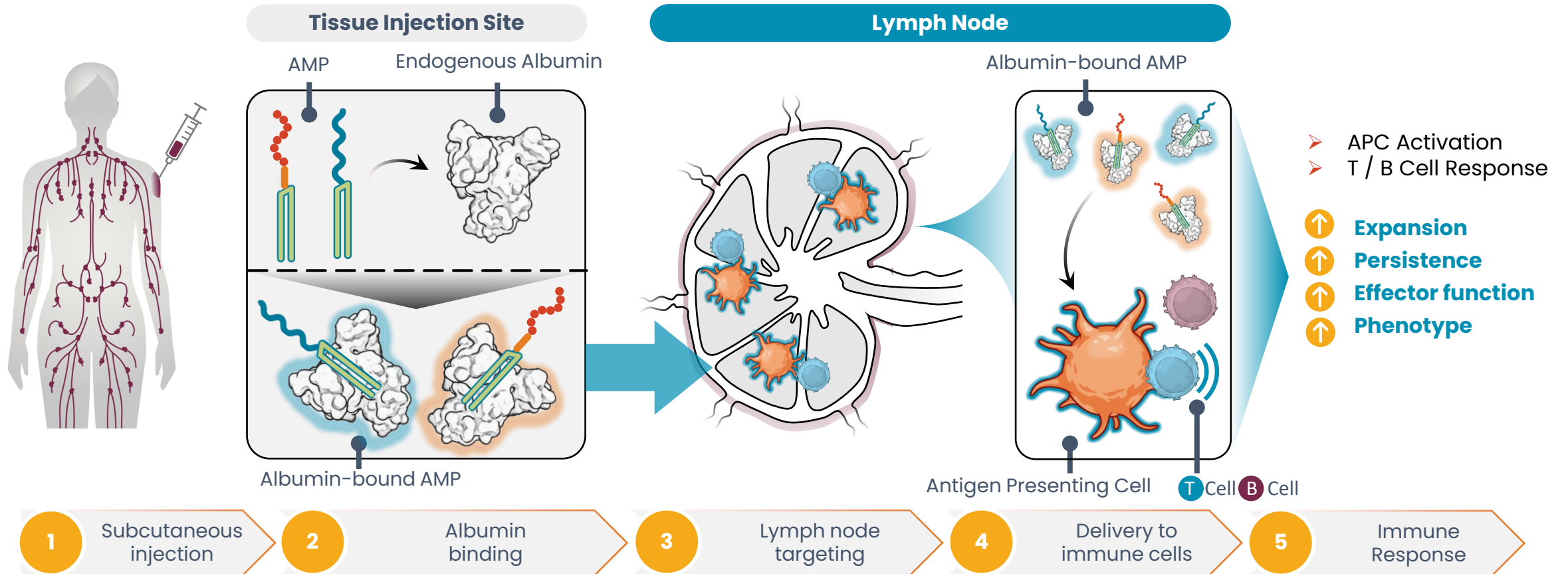


Marc Wolfgang, MS

Chief Technology Officer



Targeting the Lymph Nodes with AMP to Orchestrate Immunity



Company Pipeline

Innovative Pipeline of Cancer Immunotherapies Addressing Critical Unmet Needs

Candidate	Target	Indication	Setting	Preclinical	IND Ready	Phase 1	Phase 2	Phase 3
ELI-002 7P	mKRAS	PDAC	Adjuvant	Next Step: EOP2 meeting; Selection of Phase 3 regimen to be informed by Phase 1				
ELI-002 7P + RASi +/-CPI	mKRAS	PDAC	Metastatic					Near-term Focus
ELI-002 7P + CPI	mKRAS	PDAC	Neoadjuvant	IIT				
Longer Term Expansion Opportunities								
ELI-002 7P	mKRAS	CRC	Metastatic					
ELI-007	mBRAF	Solid Tumors						
ELI-008	mTP53	Solid Tumors						

Ongoing

Planned¹

Advancing¹

ELI-002 7P's Differentiated Approach to mKRAS Therapy

Validated mKRAS Target | Differentiated Immunotherapy Approach | Clinical Stage



Small Molecules Inhibiting Mutant KRAS

- FDA approvals for LUMAKRAS® & KRAZATI® validate target
- Mirati: \$4.8B acquisition by BMS
- Revolution Medicine: ~\$30B Market Cap; Phase 3 data, FDA approval in 2L mPDAC
- BUT
- • G12C agents only affect 1 mutation, complete response rare (~1.4%)¹, subject to multiple resistance mechanisms
- Limited duration of clinical benefit seen to date



Personalized Cancer Vaccines Targeting Private Tumor Neoantigens

- Significant progress and investment in vaccine product development:
 - Moderna / Merck: KeyNote-942
 - BioNTech / Roche: Autogene Cevumeran
- Validates clinical utility of cancer vaccines in adjuvant settings
- BUT
- • Manufacturing is long + costly
- Targets non-essential mutations
- No monotherapy activity

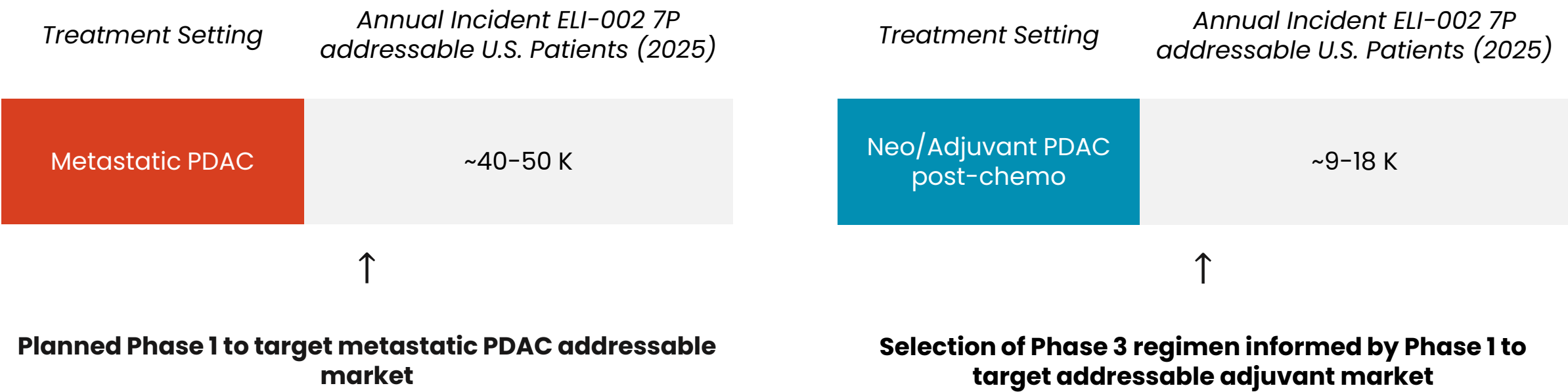


Lymph Node Targeted Immunotherapy Targeting Mutant KRAS

- ✔ Promising P1 data in PDAC and CRC
- ✔ Randomized P2 informs P3 strategy
- ✔ Off-the-shelf simplicity, COGs
- ✔ Robust T cell Response (CD4 + CD8)
- ✔ CRs observed in metastatic PDAC in sequence of ELI-002 7P, subsequent checkpoint-based regimens
- ✔ Potential broad application for ~25% of human solid cancers expressing mKRAS

Annual U.S. PDAC Addressable Market

Significant Addressable Market in Metastatic and Neo/Adjuvant PDAC



ELI-002 7P: Boosting Anti-tumor Immunity Targeting
mKRAS

Metastatic PDAC: Complete Response Observations

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.

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.

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.

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

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

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

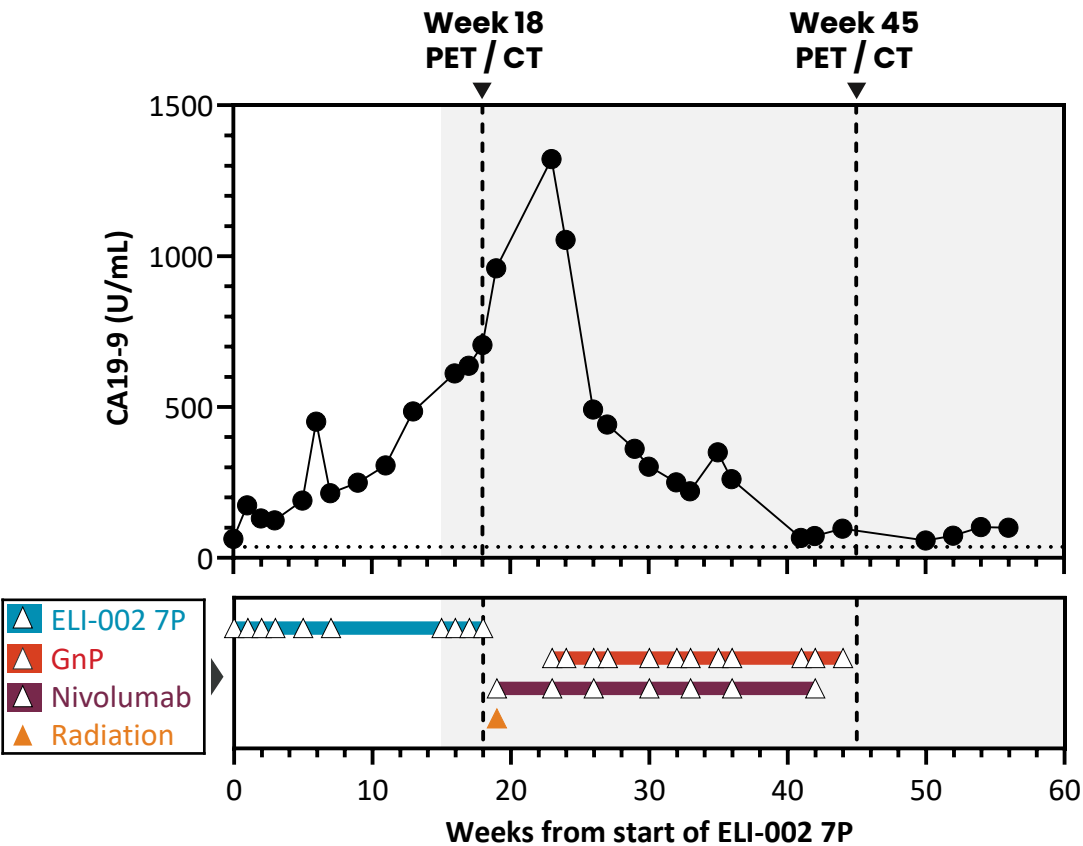
Elicio believes these observations support further investigation of the relationship between ELI-002 7P treatment and subsequent clinical outcomes.

[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

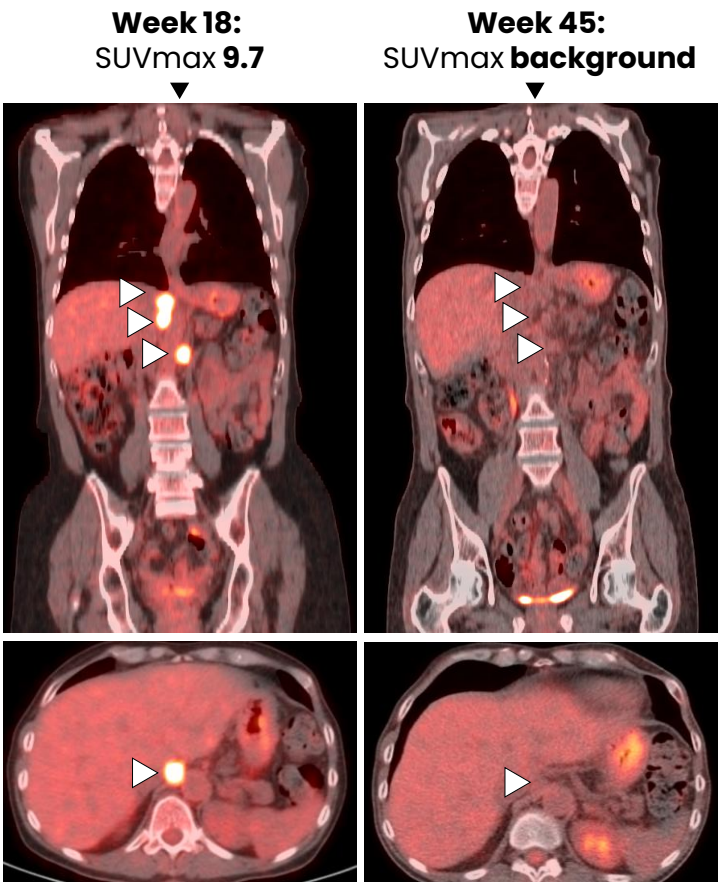
Patient 1: Complete Response Following ELI-002 7P and Subsequent Nivolumab

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



RECIST: Complete Response (CR)

PERCIST: Complete Metabolic Response (CMR)

DOR: 2.2 months

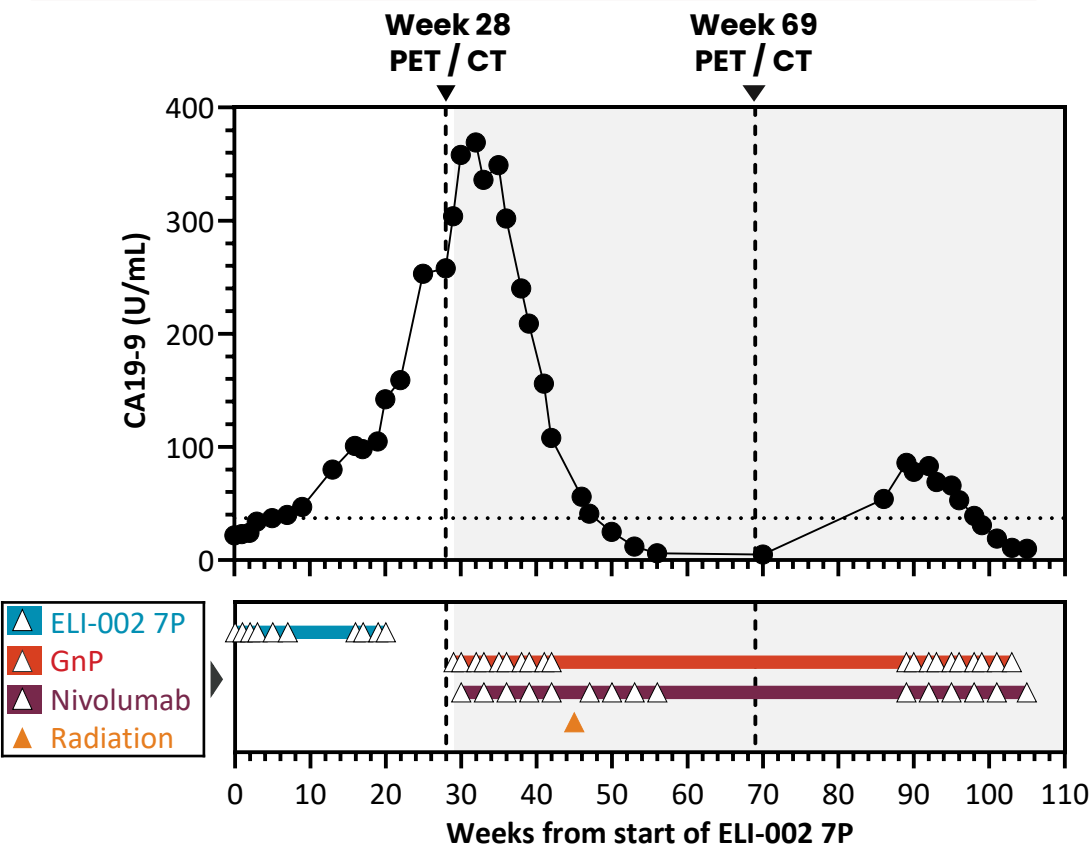
PFS: 8.3 months

Para-esophageal and mesenteric nodes

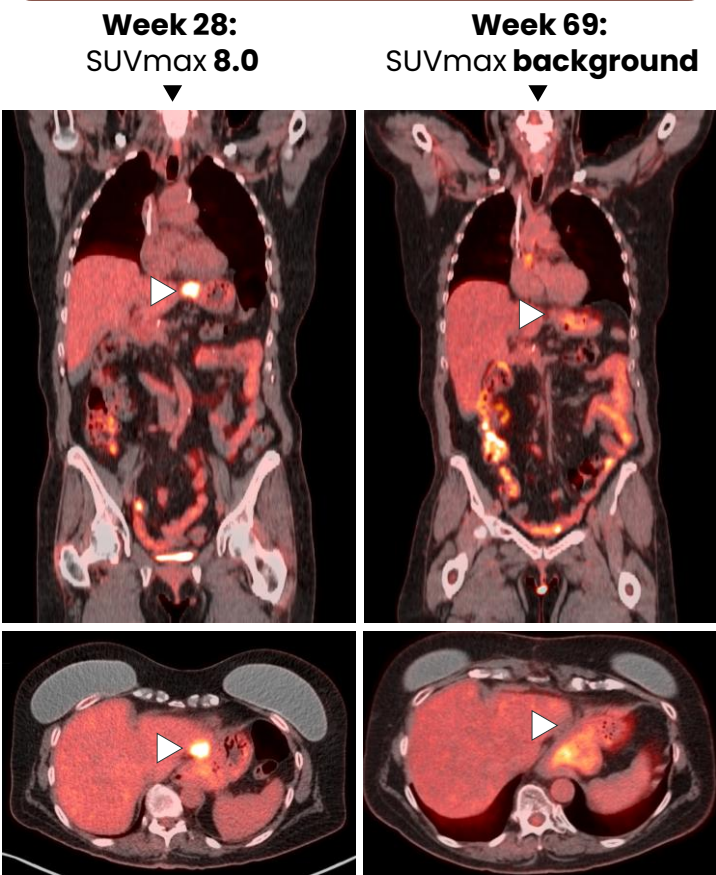
Patient 2: Complete Response Following ELI-002 7P and Subsequent Nivolumab

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

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



Patient 2: PET / CT



RECIST: Complete Response (CR)

PERCIST: Complete Metabolic Response (CMR)

DOR: 11.6 months

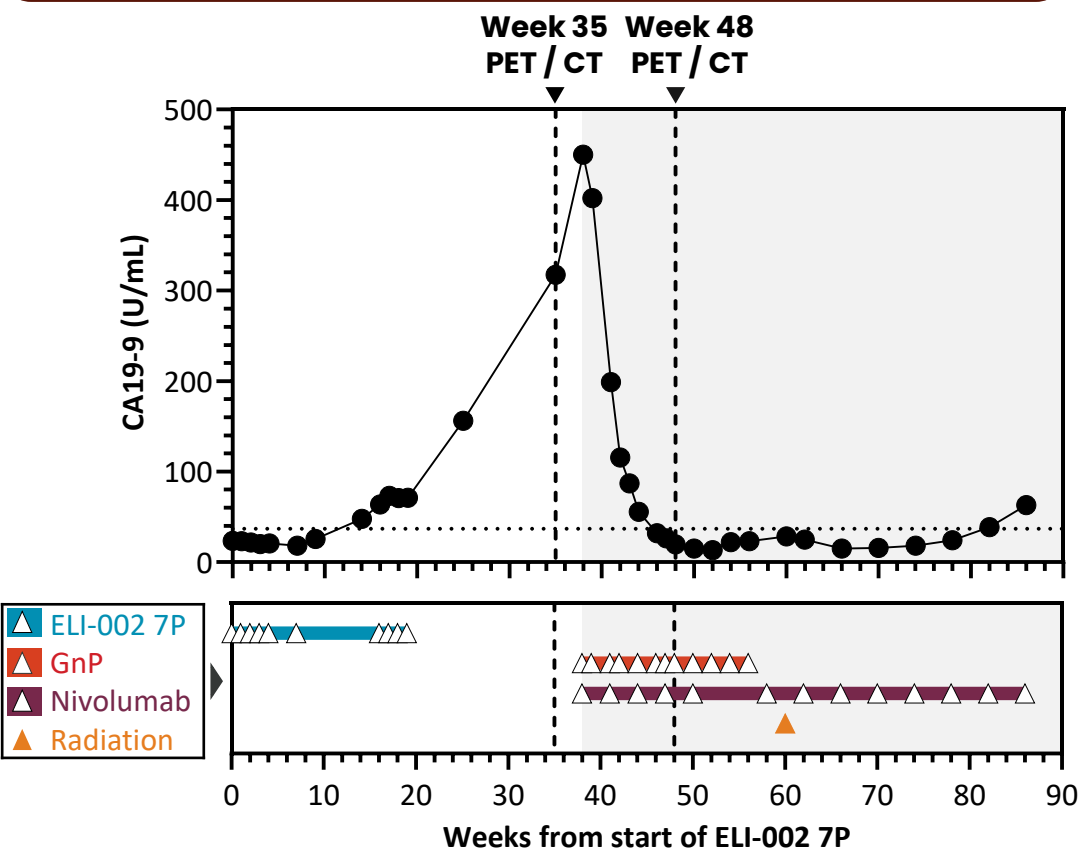
PFS: 13.4 months

Gastro-hepatic Ligament Node

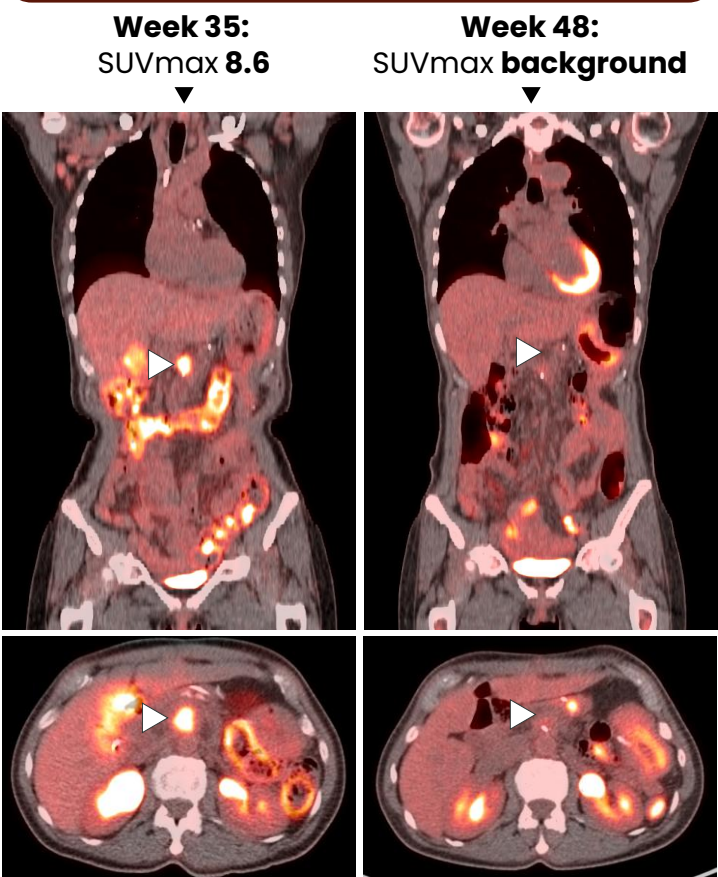
Patient 3: Complete Response Following ELI-002 7P and Subsequent Nivolumab

Complete Metabolic, and Biomarker Resolution of Mesenteric Local Recurrence

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



Patient 3: PET / CT



RECIST: Complete Response (CR)

PERCIST: Complete Metabolic Response (CMR)

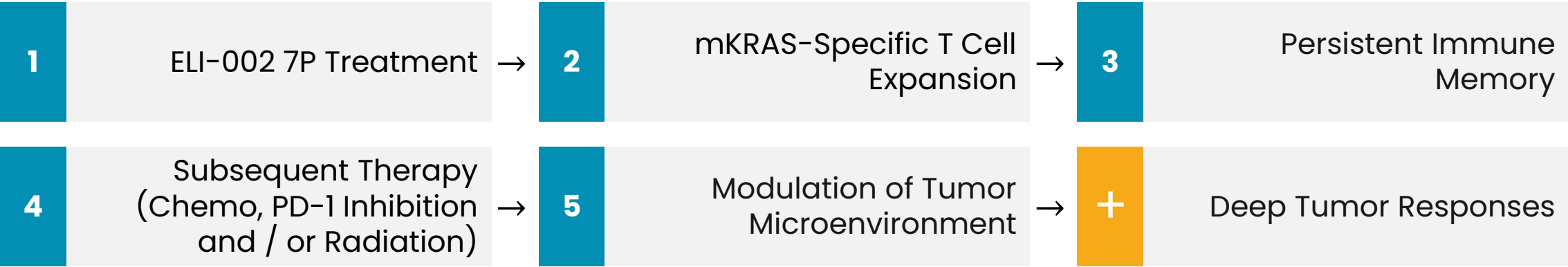
DOR: 9.2 months (ongoing)

Mesenteric Local Recurrence

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

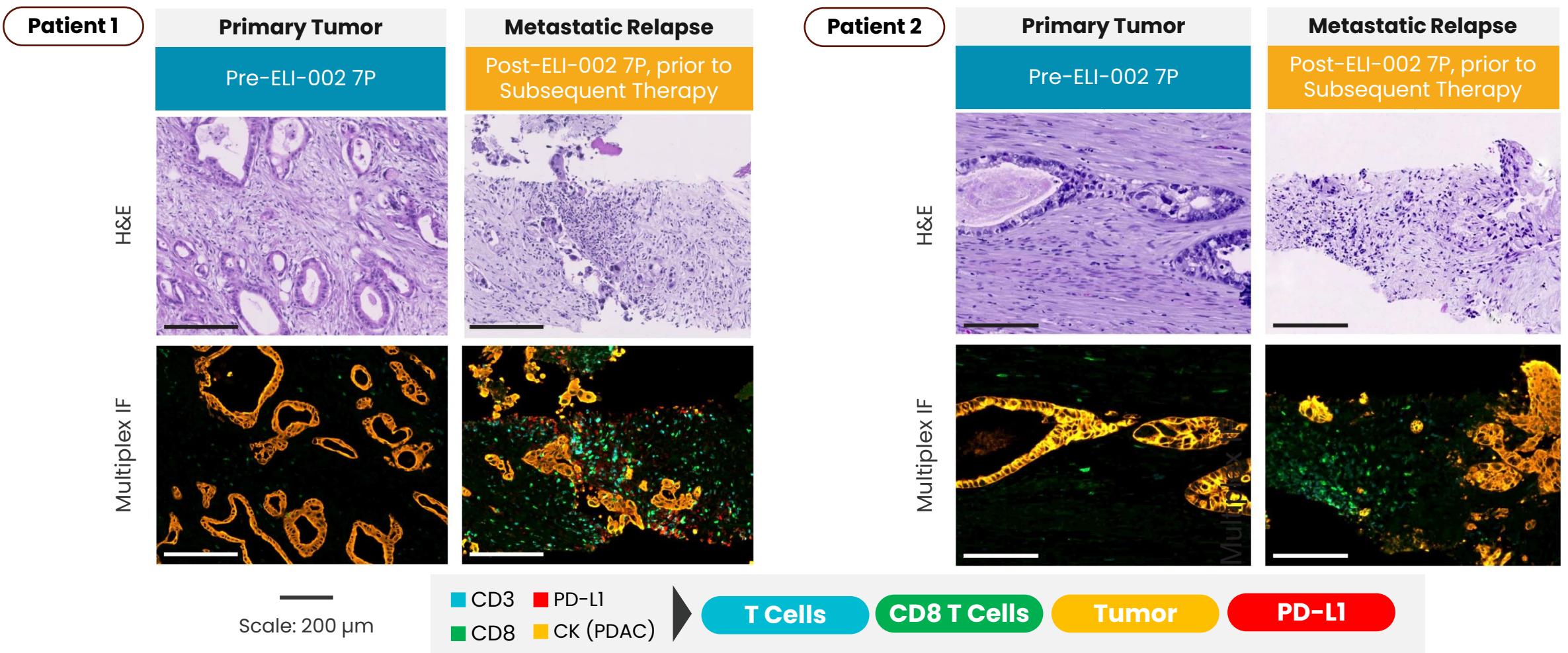
Historical Observations:



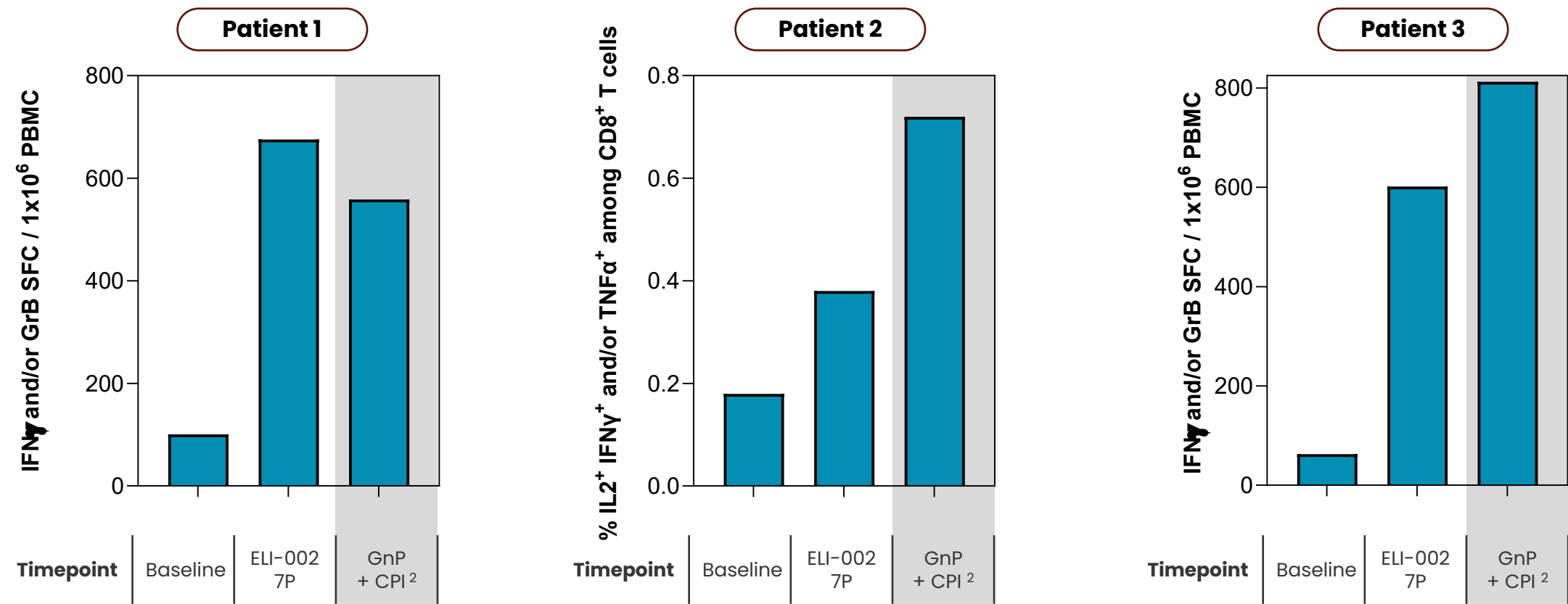
Observations from presented patients suggest a hypothetical mechanism for anti-tumor T cell responses and tumor modulation following ELI-002 7P treatment and subsequent nivolumab-based therapy.

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



mKRAS-specific T cell responses¹ persisted through subsequent chemotherapy and nivolumab treatment

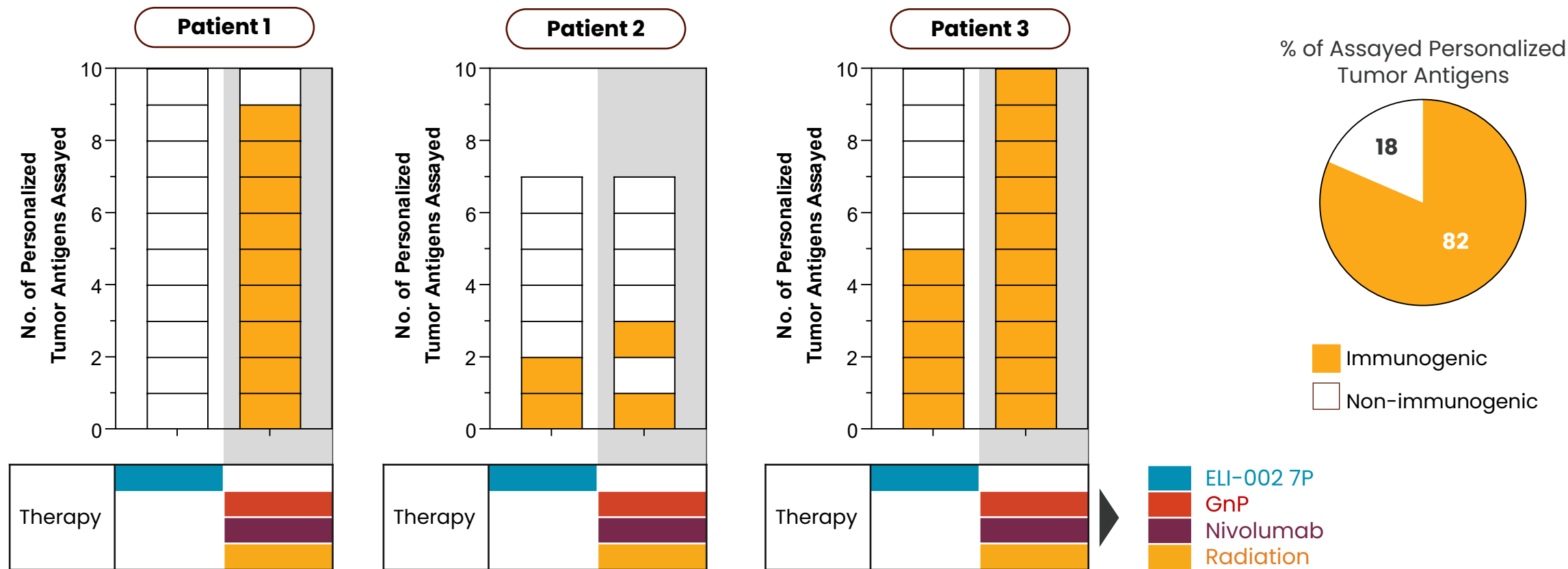


Complete responses associate with mKRAS-specific, polyfunctional, CD4+ and CD8+ T cell responses including IFN γ , TNF α , IL2, and Granzyme B

[1] Maximum ex vivo mKRAS responses determined by IFN γ /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

Metastatic PDAC Phase 1 Development Strategy

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



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.

ELI-002 7P: Boosting Anti-tumor Immunity Targeting
mKRAS

Adjuvant PDAC: AMPLIFY Phase 2 Analysis

AMPLIFY-7P: Randomized Phase 2 Trial in Adjuvant mKRAS+ PDAC

CLINICAL STUDY OVERVIEW: NCT05726864 (2:1 Randomized)

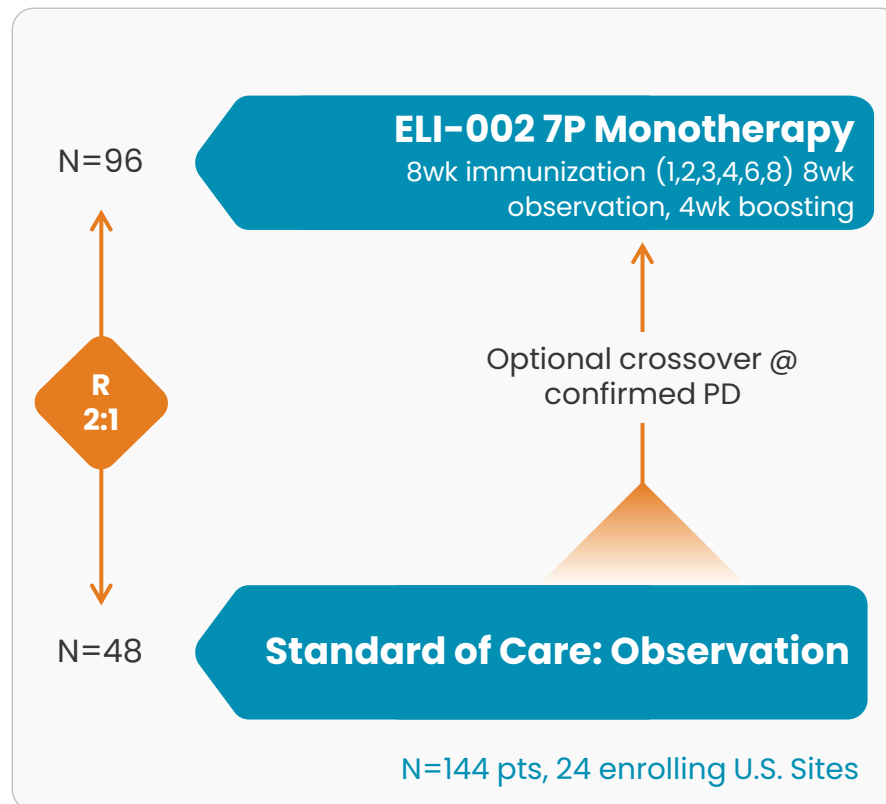
Designed to evaluate whether ELI-002 7P could improve disease-free survival following standard locoregional therapy

ELI-002 7P Monotherapy

- 7 mKRAS peptide antigens

Phase 2: Key Criteria

- Includes: mKRAS G12D/R/V/C/A/S/G13D
- Resected Stage I–III mKRAS PDAC
- Complete R0/R1 resection
- Radiographic NED status within 6 months
- MRD agnostic (biomarker +/- included)
- **Enrollment Completed December 2024**
- 144 patients randomized across 24 U.S. Sites



Endpoints

Primary Endpoint:
Disease Free Survival (DFS)

Tumor Biomarker Response
(biomarker subset)

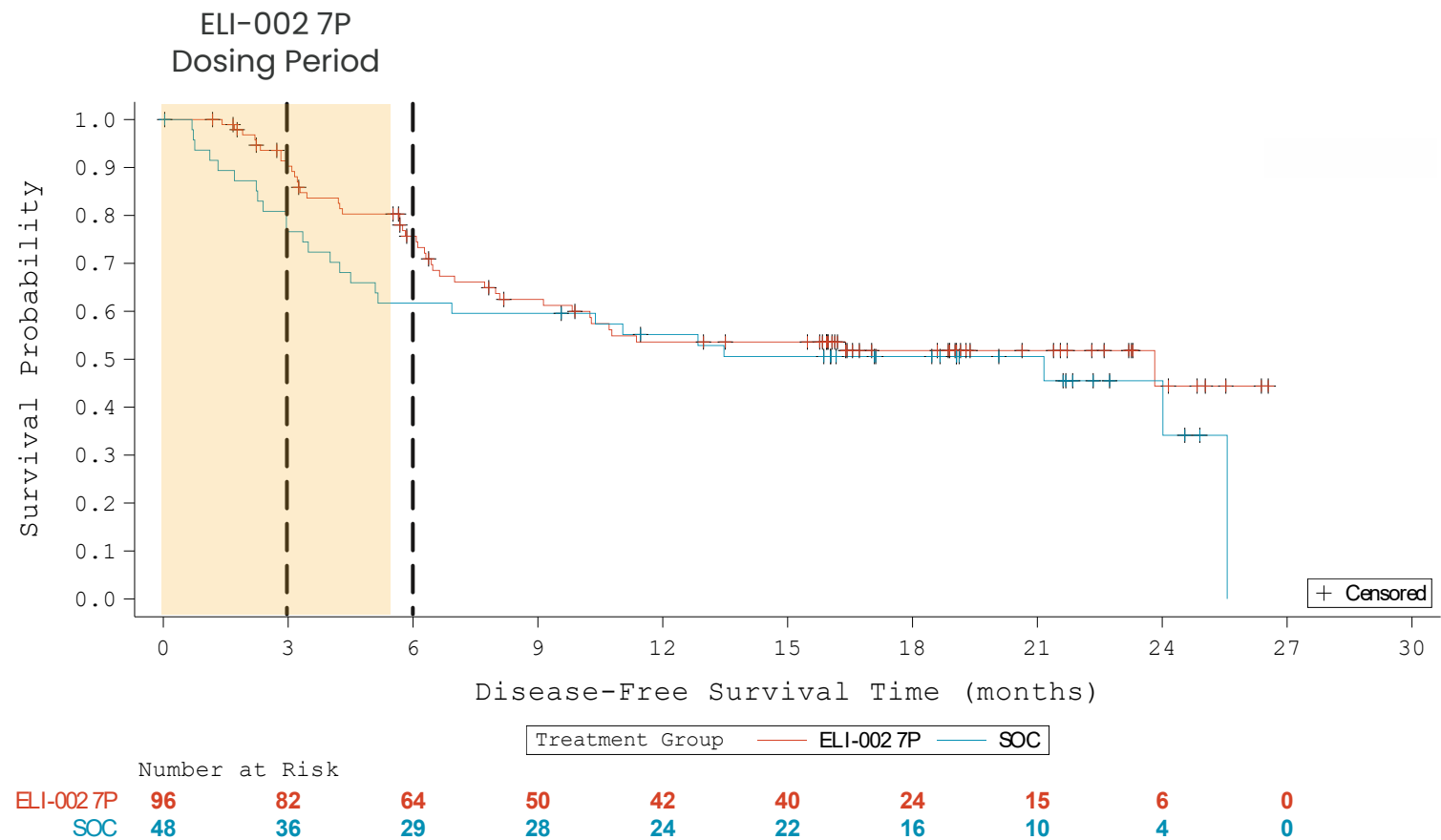
Overall Survival, Safety

Exploratory: Immunogenicity

Note: DFS assessment by iRECIST (new lesions confirmed by biopsy/imaging); R0/R1: Absence or presence of microscopic residual disease; NED: No Evidence of Disease. MRD: Minimal Residual Disease. PD: Progressive Disease

Early DFS Separation Observed Through 9 Months¹

DFS analysis at 3- and 6-months indicate early clinical activity, with treatment-arm separation persisting through 9 months



ELI-002 7P DFS: ITT Stratified

DCO	20-APR-2026
Median DFS (Months)	ELI-002 7P 23.8 (9.8, NE)
	SOC (Observation) 21.2 (5.1, NE)
HR (95% CI)	0.85 (0.52 – 1.41)
P-value	0.5329

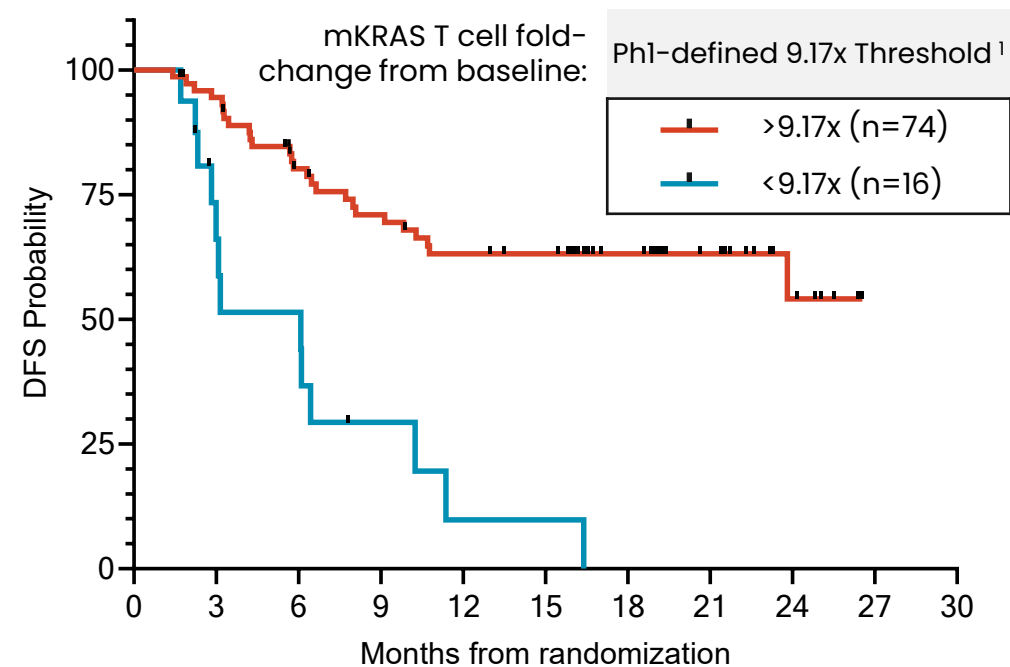
ELI-002 7P DFS: Post-hoc Landmark Analysis

Landmark	3 mo	6 mo
DFS Rate	ELI-002 7P 90.3%	75.7%
	SOC (Observation) 76.6%	61.7%
DFS Rate Difference	13.7%	14.0%
P-value	0.0222	0.0557
HR from t ₀ to Landmark	0.37	0.55

¹Primary DFS analyses used specified stratified Logrank test; DCO: Data cut-off

mKRAS-Specific T Cell Responses Were Strongly Associated with Improved DFS

Greater mKRAS-specific T cell responses were associated with longer DFS following ELI-002 7P treatment



ELI-002 7P DFS: ITT Supervised by mKRAS-specific T cell Response ²

DCO	20-APR-2026
Median DFS (Months)	>9.17xNR <9.17x6.08
HR (95% CI)	0.22 (0.08 – 0.63)
P-value	<0.0001

Immune response to ELI-002 7P was observed to be a strong predictor of DFS benefit.

¹Wainberg, O'Reilly, et al. *Nature Medicine*. 2025; ² mKRAS T cell fold-change assessed among n=90 evaluable patients

ELI-002 7P Demonstrated a Favorable Safety and Tolerability Profile

Safety profile supports extended treatment and combination strategies

Category	ELI-002 7P n (%)	SOC n (%)
Any TEAE	70 (72.9)	37 (77.1)
Treatment-related TEAE	23 (24.0)	0 (0.0)
Grade ≥ 3 TEAE	10 (10.4)	7 (14.6)
Serious TEAE	6 (6.3)	4 (8.3)
TEAE leading to dose modification	5 (5.2)	0 (0.0)
TEAE leading to withdrawal	0 (0.0)	0 (0.0)
TEAE leading to death	0 (0.0)	0 (0.0)

Proportionally Fewer Adverse Events Observed vs. SOC

- Proportionally fewer ELI-002 7P patients had adverse events (AEs), Grade ≥3 AEs, and Serious AEs
- 5% had ELI-002 7P dose delays
- 0% stopped ELI-002 7P for toxicity
- 0% treatment-related deaths
- AE event terms were similar across groups (e.g. diarrhea, fatigue in both arms reflected prior Whipple surgery)

A Precision Medicine Roadmap to Adjuvant PDAC Pivotal Phase 3

Key Phase 2 findings inform the Phase 3 development strategy

Refine Patient Eligibility

Focus on patients with the greatest potential to benefit from treatment based on subgroup analyses

Extend ELI-002 7P Treatment Duration

Robust early efficacy during the dosing period with DFS separation maintained through 9 months

Potential Evaluation of CPI Combination

Observation of complete responses in ELI-002 7P treated mPDAC patients after subsequent combination with checkpoint inhibition provides support for prospective evaluation

Phase 2 findings support a precision medicine Phase 3 strategy with refined patient eligibility, additional dosing, and potential CPI combination.

A Substantial Post-Surgical Treatment Gap Remains in Adjuvant PDAC

Observation remains standard of care despite substantial recurrence risk after surgery and chemotherapy

Current treatment course leaves a major post-surgical recurrence gap



No approved treatment following locoregional therapy

ELI-002 7P is designed to delay recurrence or prevent progression or death after standard locoregional therapy

>50%



Recurrence within 12-months ¹

~70–80%



Recurrence within 24-months ^{2,3}

0



Approved immunotherapies for adjuvant PDAC ⁴

13%



5-year overall survival all stages ⁵

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**, anticipated Q4 2026 with readouts anticipated 3–6 months after study start

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

