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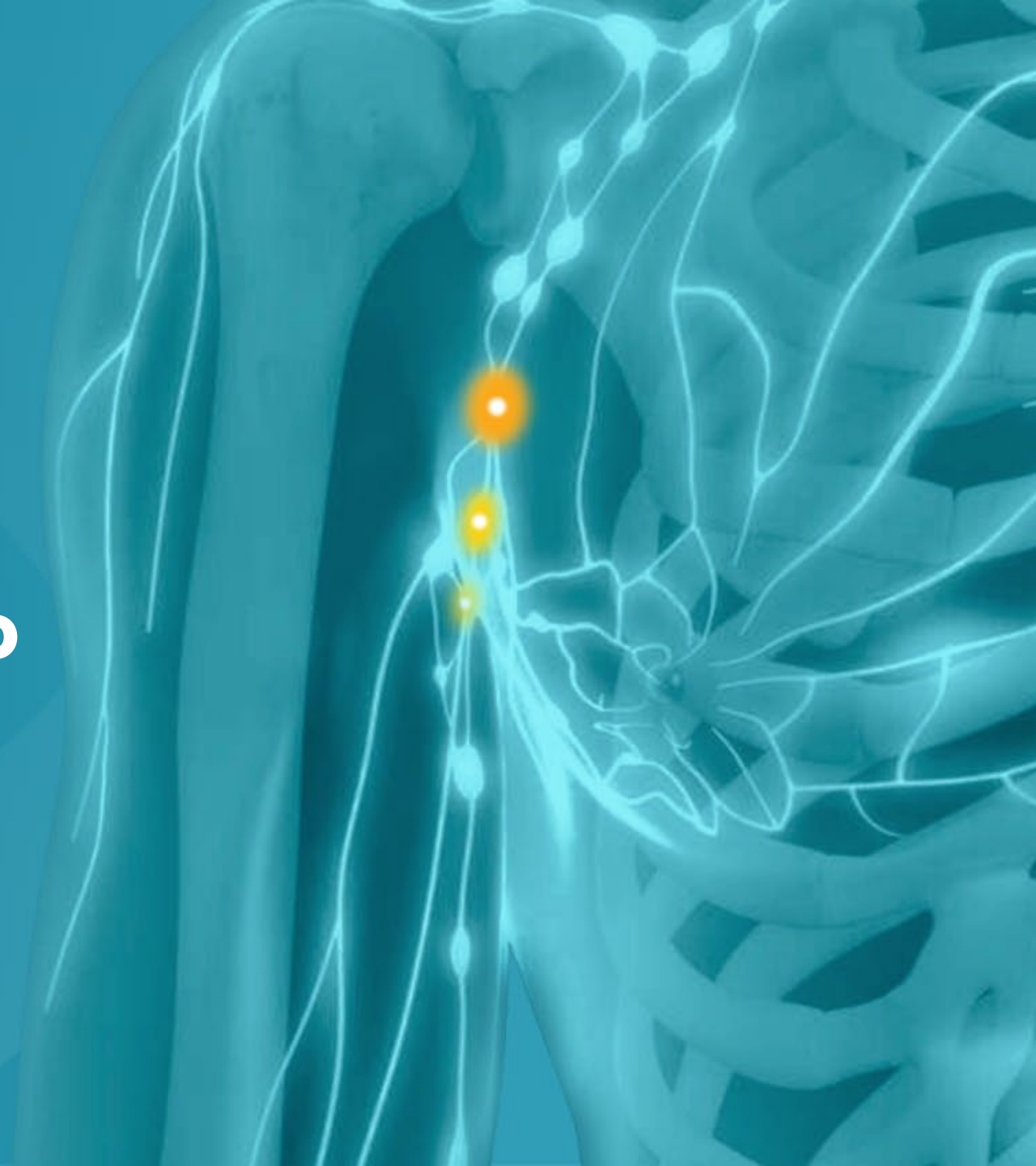
AMPLIFYing Immunity to Defeat Cancer

November 2026

Nasdaq: ELTX



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This presentation contains forward-looking statements as that term is defined in Section 27A of the Securities Act of 1933, as amended, Section 21E of the Securities Exchange Act of 1934, as amended, and the Private Securities Litigation Reform Act of 1995, known as the PSLRA. Statements in this presentation that are not purely historical are forward-looking statements. Such forward-looking statements include, among other things, statements regarding our planned and ongoing clinical programs, including the timing and outcome of planned and ongoing clinical trials, including the planned Phase 1 study evaluating camupepimut (ELI-002 7P) in combination with Rasonque™ with or without nivolumab in metastatic PDAC; our planned end of Phase 2 meeting with the FDA and potential Phase 3 trial design; the potential of our product candidates, including the potential of camupepimut; the extent to which results of subgroup or exploratory analyses are predictive of results in future clinical trials; the potential for the Phase 1 study to provide a rapid development pathway for camupepimut; the potential for the Phase 1 study to inform future metastatic PDAC and Phase 3 adjuvant PDAC development; the potential for ELI-002-induced immune priming to synergize with other therapies to enable tumor eradication; the potential for future expansion of ELI-002 to other mPDAC combinations or indications, including in mKRAS positive cancers beyond PDAC; the potential benefits and effectiveness of off-the-shelf immunotherapy approaches; and other statements regarding management's intentions, plans, beliefs, expectations or forecasts for the future and, therefore, you are cautioned not to place undue reliance on them. No forward-looking statement can be guaranteed and actual results may differ materially from those projected. We undertake no obligation to publicly update any forward-looking statement, whether as a result of new information, future events or otherwise, except to the extent required by law. We use words such as "anticipates," "believes," "plans," "expects," "projects," "future," "intends," "may," "will," "should," "could," "estimates," "predicts," "potential," "continue," "guidance," and similar expressions to identify these forward-looking statements that are intended to be covered by the safe-harbor provisions of the PSLRA. Such forward-looking statements are based on our expectations and involve risks and uncertainties; consequently, actual results may differ materially from those expressed or implied in the statements due to a number of factors, including, but not limited to, our plans to develop and commercialize our product candidates, including camupepimut; the timing of initiation of our planned clinical trials; the extent to which results of subgroup or exploratory analyses are predictive of results in future clinical trials; the timing of the availability of data from our planned clinical trials; the timing of any planned investigational new drug application or new drug application; our plans to research, develop and commercialize our current and future product candidates; and our estimates regarding future revenue, expenses, capital requirements and need for additional financing.

New factors emerge from time to time, and it is not possible for us to predict all such factors, nor can we assess the impact of each such factor on the business or the extent to which any factor, or combination of factors, may cause actual results to differ materially from those contained in any forward-looking statements. These risks are more fully discussed in our Annual Report on Form 10-K filed with the SEC on March 12, 2026, as amended on April 29, 2026, under the heading "Risk Factors", in our Quarterly Report on Form 10-Q for the quarter ended March 31, 2026, filed with the SEC on May 11, 2026, in our Quarterly Report on Form 10-Q for the quarter ended June 30, 2026, filed with the SEC on August 13, 2026, and any subsequent reports and other documents filed from time to time with the SEC. Forward-looking statements included in this Presentation are based on information available to us as of the date of this Presentation. We do not undertake any obligation to update such forward-looking statements to reflect events or circumstances after the date of this Presentation, except to the extent required by law.

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 immunity 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**

Camupepimut: 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 therapy with CPI [2]

Value-creating catalysts and capitalization

- **Metastatic PDAC Phase 1 Initiation:** ORR planned endpoint, Rasonque™ +/- nivolumab
- **Adjuvant PDAC Phase 3 Preparation and Next Steps:** Camupepimut end of Phase 2 FDA meeting
- Investigator-initiated trial of **camupepimut + checkpoint inhibitor in neoadjuvant PDAC**

[1] Elicio Press Release 6/15/2026; [2] Elicio Press Release 6/17/2026

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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

axcella

Flagship Pioneering

PULMATRIX

domantis

BioVeris

Abbott

KOCH INSTITUTE for Integrative Cancer Research at MIT

Massachusetts Institute of Technology

NIH

NOVARTIS

Whitehead Institute

ATARA BIO

AMGEN

Cougar Biotechnology

janssen

UCSF

HARVARD UNIVERSITY

Massachusetts Institute of Technology

Stanford University

CIDARA THERAPEUTICS

brainstorm cell therapeutics

BARCLAYS

cg/Canaccord Genuity

Stanford University

Wharton University of Pennsylvania

tem MOLECULAR TEMPLATES

BLANKROME

RUTGERS

SAIL BIOMEDICINES

BIONTECH

CERULEAN LEADERSHIP IN NANOPHARMACEUTICALS

MILLENNIUM PHARMACEUTICALS, INC

Biogen

Boehringer Ingelheim

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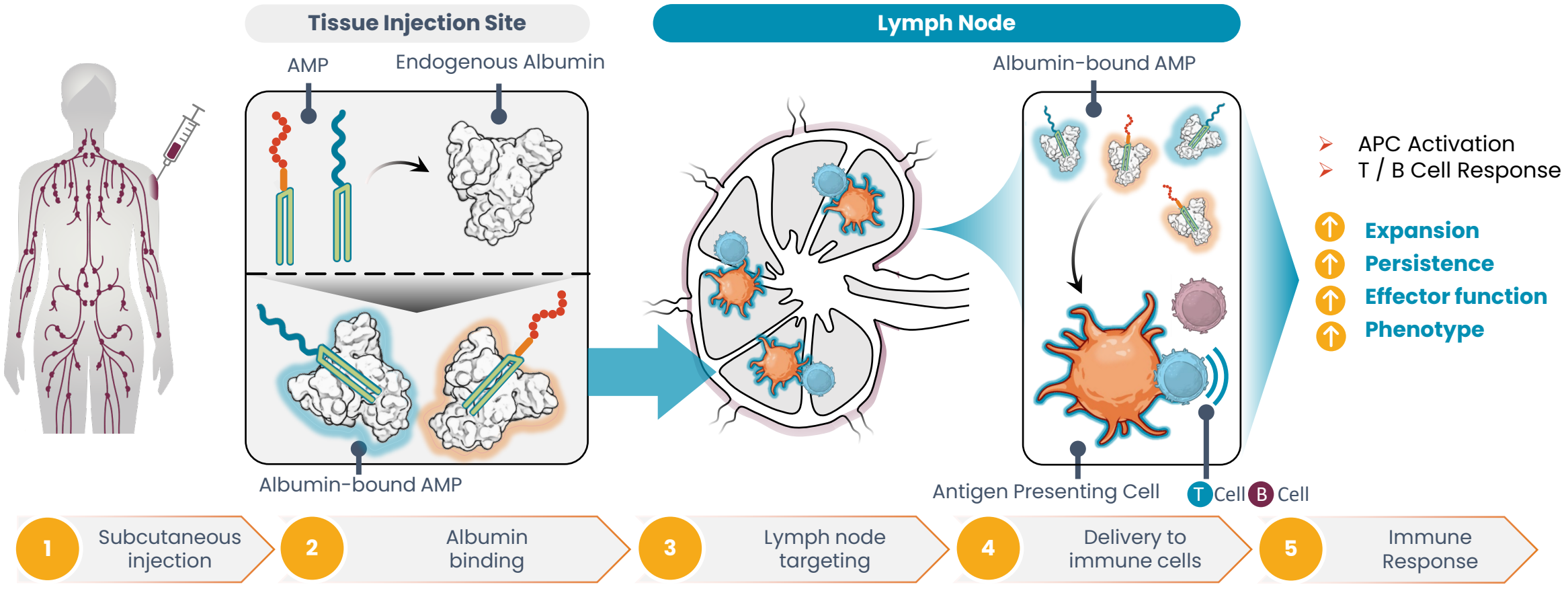
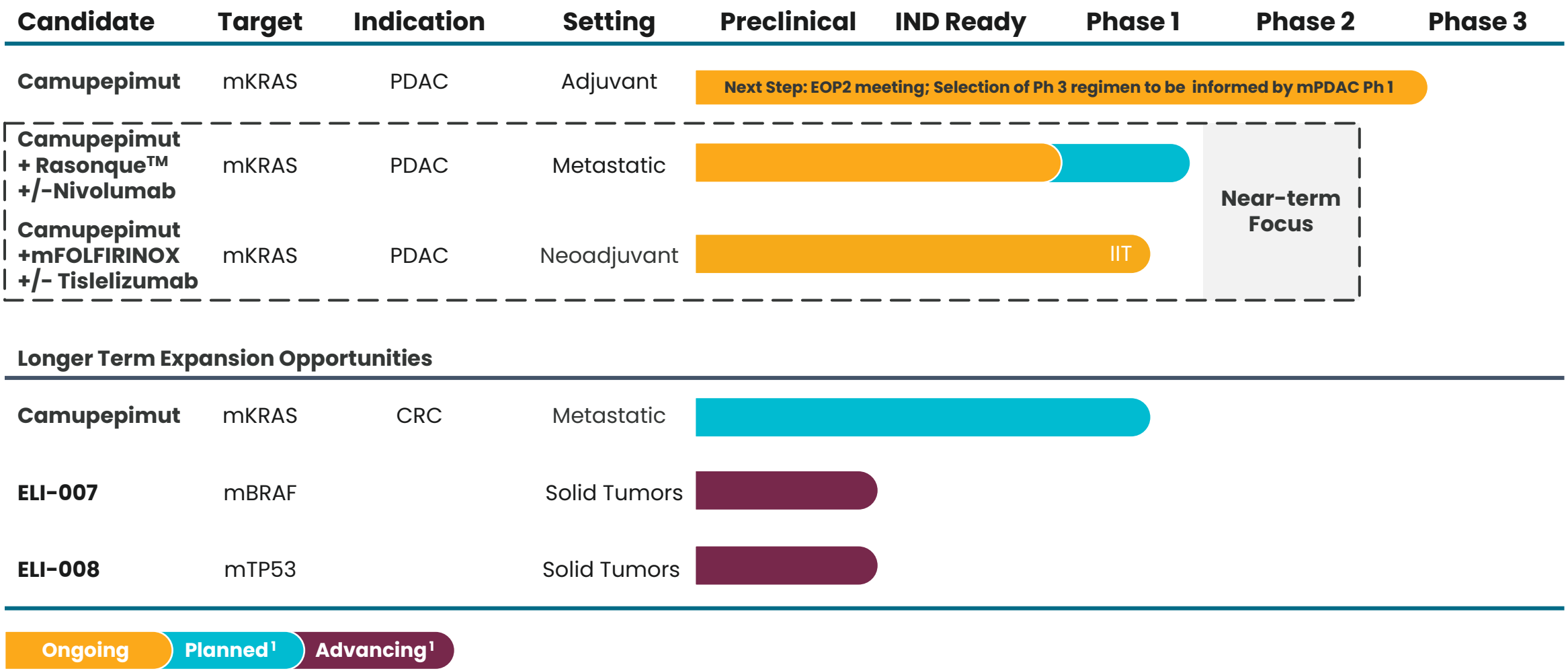
Targeting the Lymph Nodes with AMP to Orchestrate Immunity



Wainberg, O'Reilly, et al. ***Nature Medicine****. 2025; Pant, O'Reilly, et al.* ***Nature Medicine****. 2024; Liu, Irvine, et al.* ***Nature*** *2014; Moynihan, Irvine, et al.* ***Nature Medicine*** *2016; Ma, Irvine, et al.* ***Science*** *2019*

Company Pipeline

Innovative Pipeline of Cancer Immunotherapies Addressing Critical Unmet Needs



CRC: colorectal carcinoma | PDAC: pancreatic ductal adenocarcinoma; mKRAS: mutant Kirsten rat sarcoma | mBRAF: mutant homolog B of the Rapidly Accelerated Fibrosarcoma | mTP53: mutant tumor protein p53; IIT: Investigator initiated trial; ***[1] Planned and Advancing programs are subject to funding***

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Camupepimut: A 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

- Complete response rare (0-13%)[1], subject to multiple resistance mechanisms, G12C agents only affect 1 mutation
- 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

elicio THERAPEUTICS — 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 camupepimut, subsequent checkpoint-based regimens
- Potential broad application for ~25% of human solid cancers expressing mKRAS

*[1] O'Reilly **NEJM** 2026; Conroy **NEJM** 2011; Morizane **BJC Rep** 2024; Revolution Medicines April 21 2026 PR; Jameson **JAMA Oncol** 2020; Wainberg, **Clinical Cancer Research**. 2020; Padron, **Nature Medicine**. 2022; CR was not confirmed; Chen, **J Clinical Oncology**. 2022; Xie, **Clinical Cancer Research**. 2020; [2] Elicio Press Release 6/15/2026*

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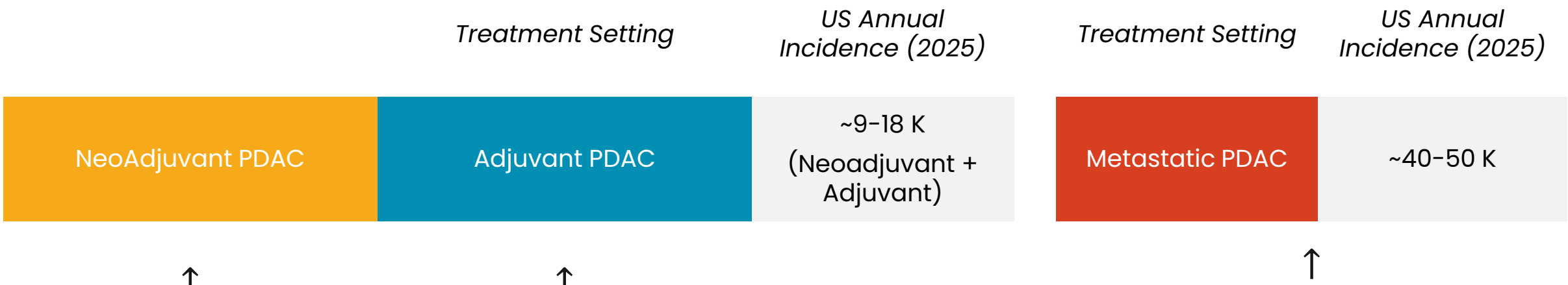
Camupepimut Targets a Large Addressable U.S. Pancreatic Cancer Market Across Multiple Treatment Settings

Approximately 49,000–68,000 annual addressable patients across metastatic, neoadjuvant, and adjuvant PDAC, supporting multiple clinical development opportunities



*American Cancer Society: Cancer Facts & Figures 2026; O'Reilly et al. **JAMA**, 2022; Soweid et. al. 2017; Park et. al. 2021; Auclin et. al. 2021*

Camupepimut:
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)

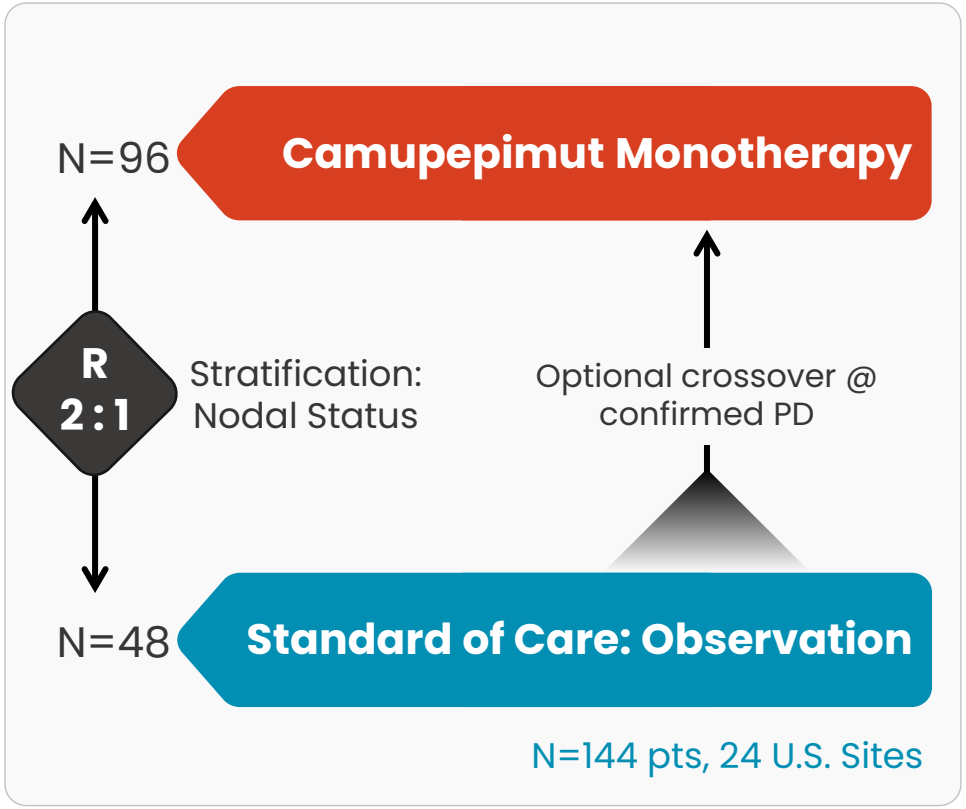
Designed to evaluate whether camupepimut could improve disease-free survival following standard locoregional therapy

Camupepimut 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 < 6 months
- MRD agnostic (biomarker +/-)
- Enrollment Completed 12/2024



Endpoints

Primary Endpoint:
Disease Free Survival (DFS)

Tumor Biomarker Response

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

Baseline Imbalances Favored Observation Despite Balanced Stratification Factor

Stratification balanced nodal status, stage; However, ctDNA positive, R0/R1, prior radiation were imbalanced

	Camupepimut (*n*=96)	Observation (*n*=48)	Overall (*n*=144)	
Age (years)				
Median, range	65 (33-86)	69 (46-81)	66 (33-86)	
Disease stage at screening, *n* (%)				
Stage I	30 (31.3)	16 (33.3)	46 (31.9)	
Stage II	41 (42.7)	20 (41.7)	61 (42.4)	
Stage III	25 (26.0)	12 (25.0)	37 (25.7)	**Allocation Relative to Observation Arm**
Nodal status, *n* (%)				
N0	39 (40.6)	19 (39.6)	58 (40.3)	**Stratification Factor**
N1 or N2	57 (59.4)	29 (60.4)	86 (59.7)	
Resection margin, *n* (%)				
R0	71 (74.0)	39 (81.3)	110 (76.4)	
R1	25 (26.0)	9 (18.8)	34 (23.6)	
ctDNA positive at baseline, *n* (%)				
ctDNA positive	20 (20.8)	6 (12.5)	26 (18.1)	**ctDNA Positive**
Prior anti-cancer therapy, *n* (%) [1]				
Systemic therapy-FOLFIRINOX	83 (86.5)	43 (89.6)	126 (87.5)	
Systemic therapy-GnP	26 (27.1)	9 (18.8)	35 (24.3)	
Surgery/Procedure	96 (100)	48 (100)	144 (100)	
Radiation therapy	23 (24.0)	15 (31.3)	38 (26.4)	
CA19-9 at baseline, *n* (%)				
CA19-9 >37 U/mL	28 (29.2)	14 (29.2)	42 (29.2)	

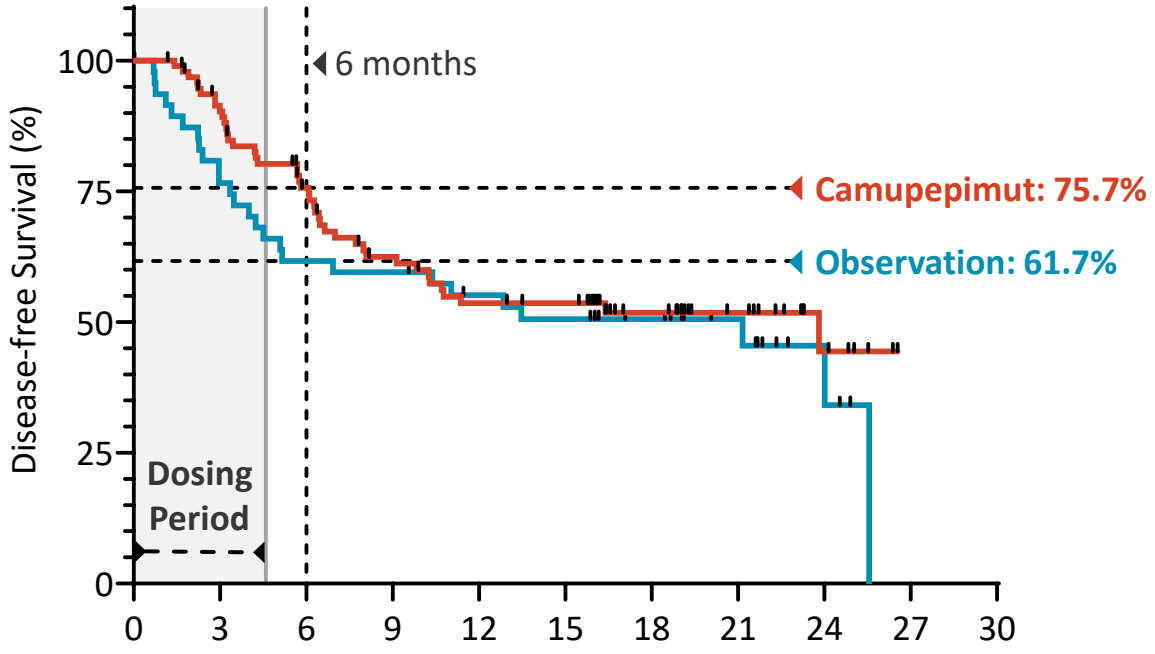
George, O'Reilly, et al. ***Nature Medicine****. 2026; O'Reilly, et al.* ***Annals of Oncology.*** *2026*

Primary DFS Endpoint Not Met in ITT Population

Early separation favored camupepimut, with a 14% higher DFS rate at 6 months (75.7% vs. 61.7%)

***n*=144**

Intention-to-Treat:



At risk										
Camupepimut	96	82	64	50	42	40	24	15	6	0
Observation	48	36	29	28	24	22	16	10	4	0

Camupepimut (*n*=96)	Median 23.8 months
Observation (*n*=48)	Median 21.2 months
HR: 0.85 (0.52-1.41)	
***P*=0.5329**	

		6 months
DFS Rate	**Camupepimut**	75.7%
	Observation	61.7%
	DFS Rate Difference	14.0%
	P-value	0.0557

HR from t_0 to 6 months	0.55

George, O'Reilly, et al. ***Nature Medicine***. 2026; O'Reilly, et al. ***Annals of Oncology.*** 2026

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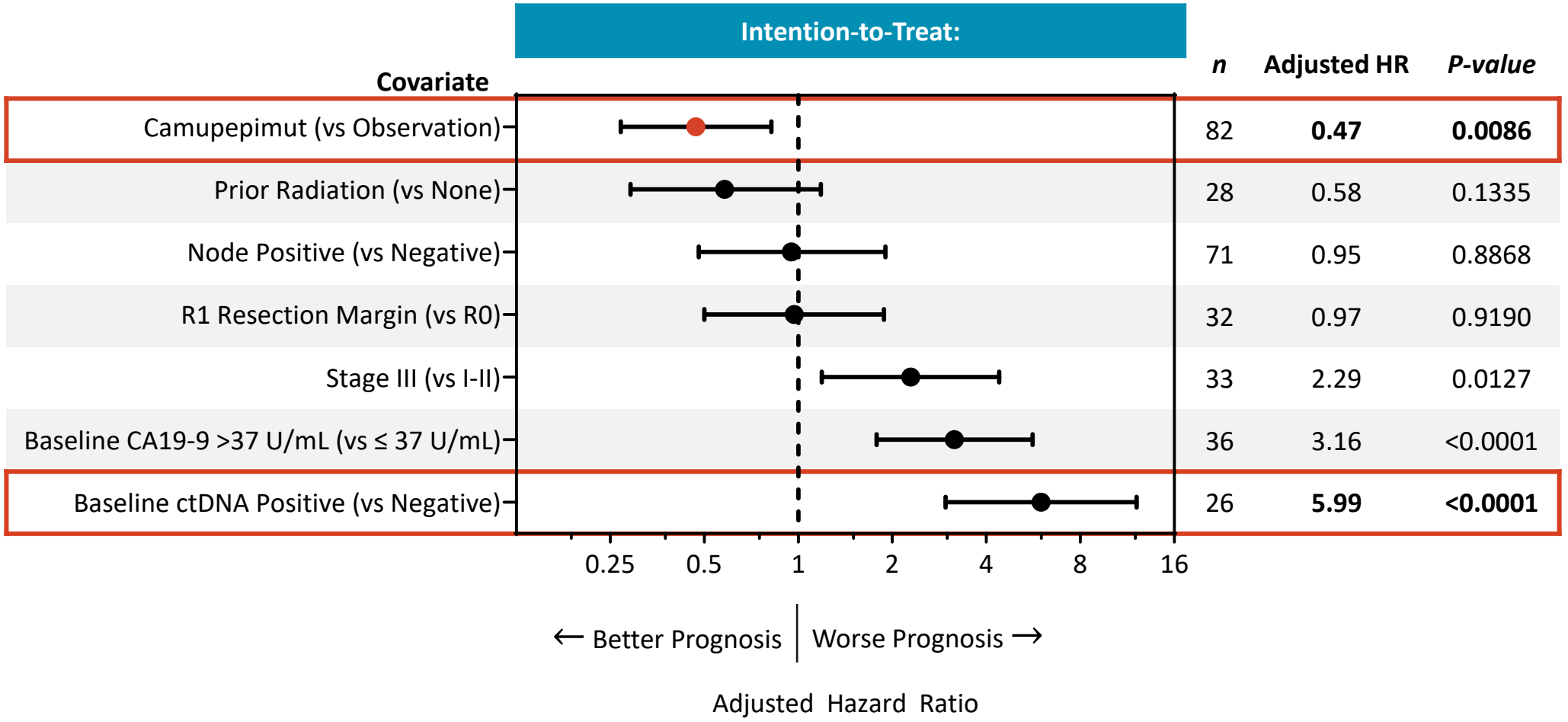
Multivariate Analysis Identifies ctDNA as a Strong Prognostic Factor for DFS

After adjustment for baseline prognostic factors, camupepimut was associated with a 53% reduction in risk of recurrence or death (HR 0.47; p=0.0086)



George, O'Reilly, et al. ***Nature Medicine***. 2026; O'Reilly, et al. ***Annals of Oncology.*** 2026

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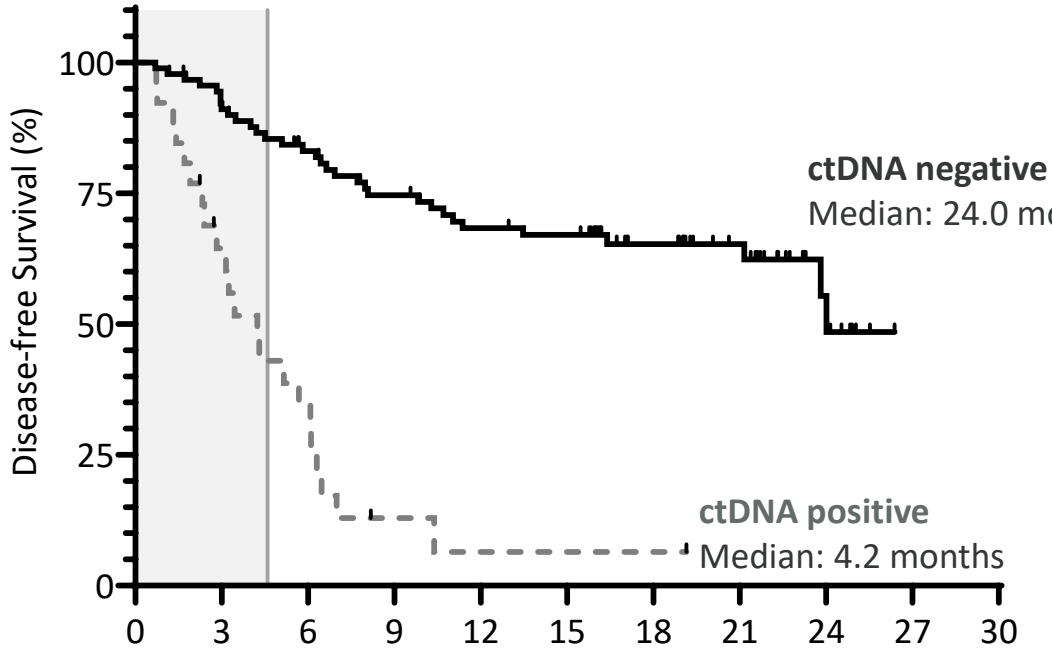
Baseline ctDNA Positivity Observed to Strongly Correlate with Disease Recurrence

Pre-specified analysis showed ctDNA-positive patients had a 6.56-fold higher risk of recurrence or death

n=119 | **Prespecified:** Pooled ctDNA Positive vs Negative



At risk										
ctDNA–	93	81	70	61	54	51	33	22	8	0
ctDNA+	26	15	8	2	1	1	1	0	0	0

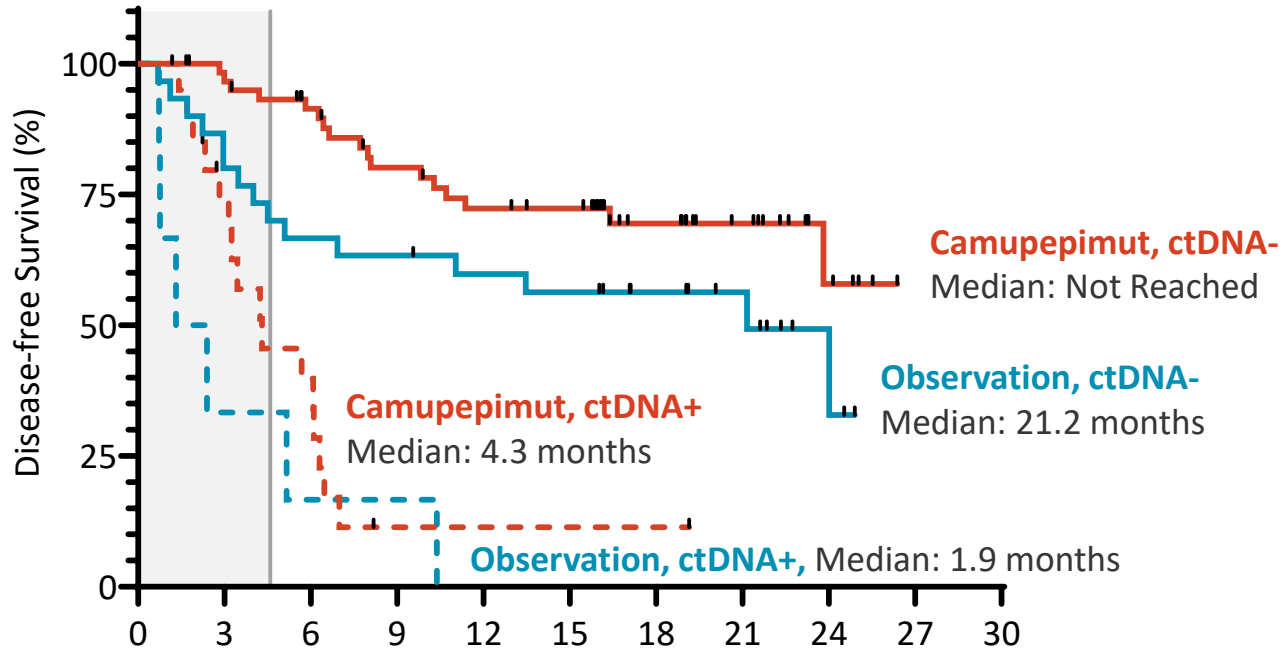
ctDNA– (*n*=93)	Median 24.0 months
ctDNA+ (*n*=26)	Median 4.2 months
HR: 6.56 (3.63-11.83)	
***P*<0.000001**	

George, O'Reilly, et al. ***Nature Medicine****. 2026; O'Reilly, et al.* ***Annals of Oncology.*** *2026*

Camupepimut Demonstrated Consistent DFS Benefit Across ctDNA Subgroups

Favorable treatment effects were observed in both ctDNA- (HR 0.51), ctDNA+ (HR 0.57) patients in pre-specified analysis

n=119 | **Prespecified:** ctDNA Positive vs Negative



At risk	ctDNA									
Camupepimut	–	62	57	50	42	37	35	21	14	5	0
Observation	–	31	24	20	19	17	16	12	8	3	0
Camupepimut	+	20	13	7	1	1	1	1	0	0	0
Observation	+	6	2	1	1	0	0	0	0	0	0

ctDNA– Subgroup

Camupepimut (*n*=62)	Median Not Reached
Observation (*n*=31)	Median 21.2 months

HR: 0.51 (0.25-1.02)

***P*=0.0280**

ctDNA+ Subgroup

Camupepimut (*n*=20)	Median 4.3 months
Observation (*n*=6)	Median 1.9 months

HR: 0.57 (0.22-1.48)

***P*=0.0489**

George, O'Reilly, et al. ***Nature Medicine****. 2026; O'Reilly, et al.* ***Annals of Oncology.*** *2026*

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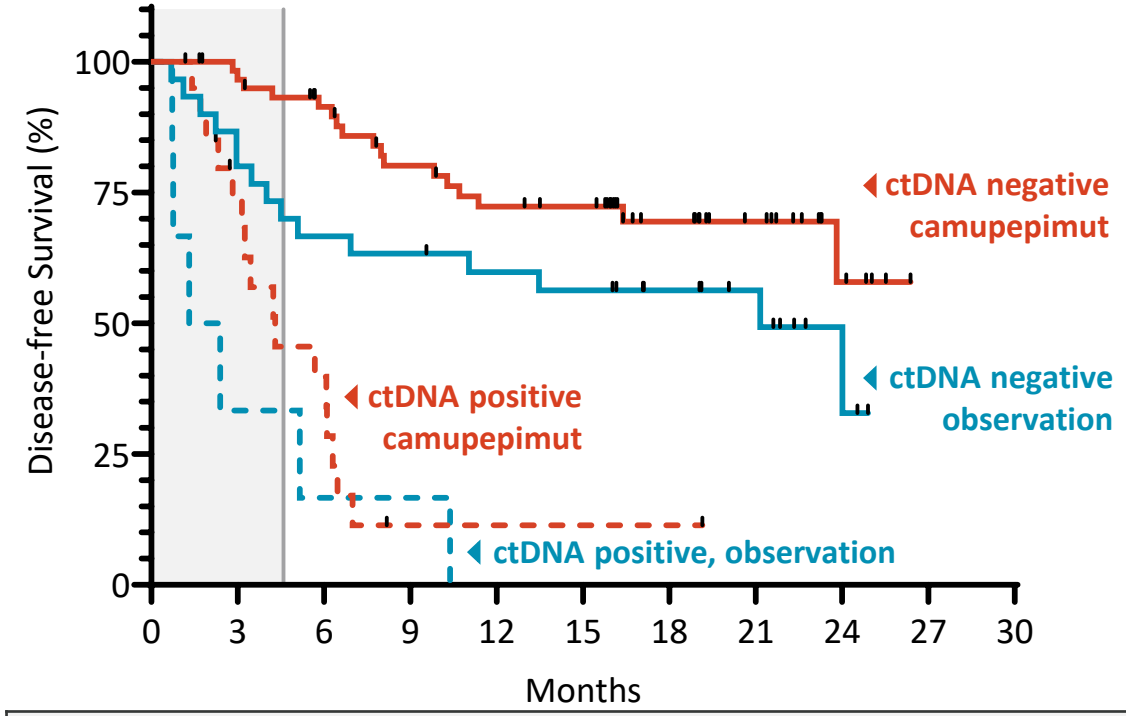
ctDNA-Stratified Outcomes Showed Similar Patterns Across PDAC and Melanoma

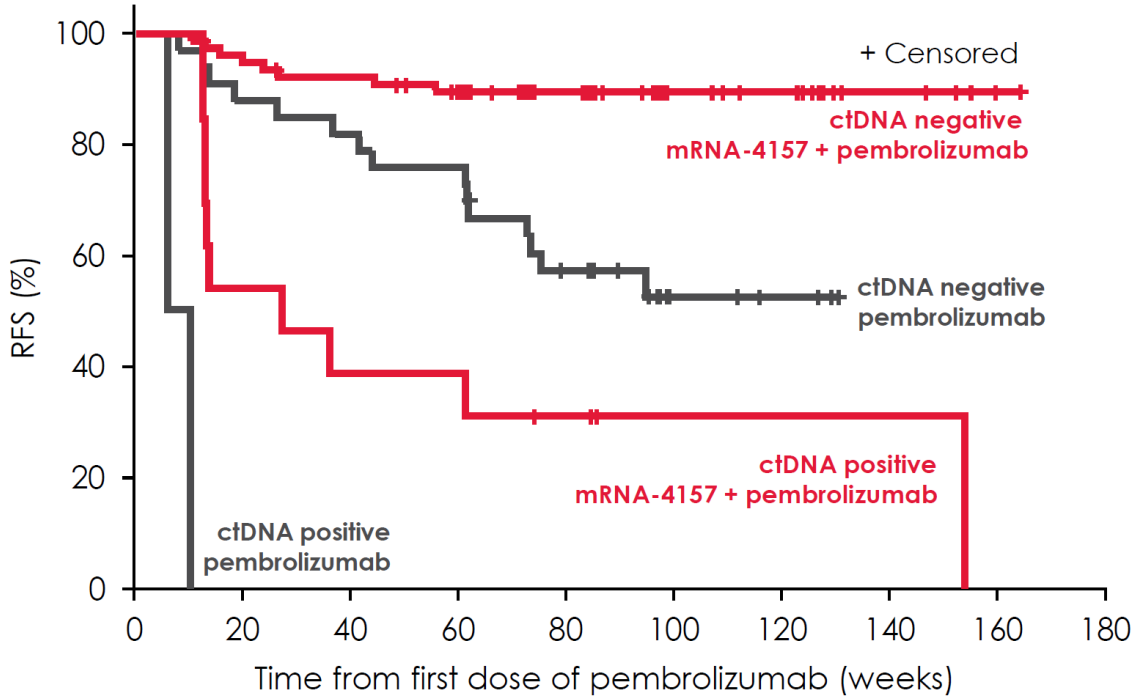
AMPLIFY-7P findings consistent with Phase 2 melanoma observations in baseline ctDNA +/- subgroups

Adjuvant PDAC | **Camupepimut Monotherapy vs Observation**



	Camupepimut Events n/N (%)	Observation Events n/N (%)	Camupepimut vs Observation HR (95% CI)	*P-value*
ctDNA positive	16/20 (80.0)	6/6 (100)	0.57 (0.22-1.48)	0.0489
ctDNA negative	17/62 (27.4)	15/31 (48.4)	0.51 (0.25-1.02)	0.0280

Adjuvant Melanoma | **mRNA-4157 + Pembro vs Pembro**



	mRNA-4157 (V940) + pembrolizumab Events, n/N (%)	pembrolizumab Events, n/N (%)	mRNA-4157 (V940) + pembrolizumab versus pembrolizumab HR (95% CI)
ctDNA positive	10/13 (76.9)	2/2 (100)	NE
ctDNA negative	8/77 (10.4)	15/33 (45.5)	**0.225** (0.095–0.531)

George, O'Reilly, et al. **Nature Medicine**. 2026; O'Reilly, et al. **Annals of Oncology**. 2026; Weber JS, *et al*. **Annals of Oncology**. 2023; Weber JS, *et al*. **The Lancet**. 2024; Comparisons based on historical published data; no head-to-head studies have been conducted and cross-trial comparisons may not be reliable due to differences in trial design and patient population and characteristics.

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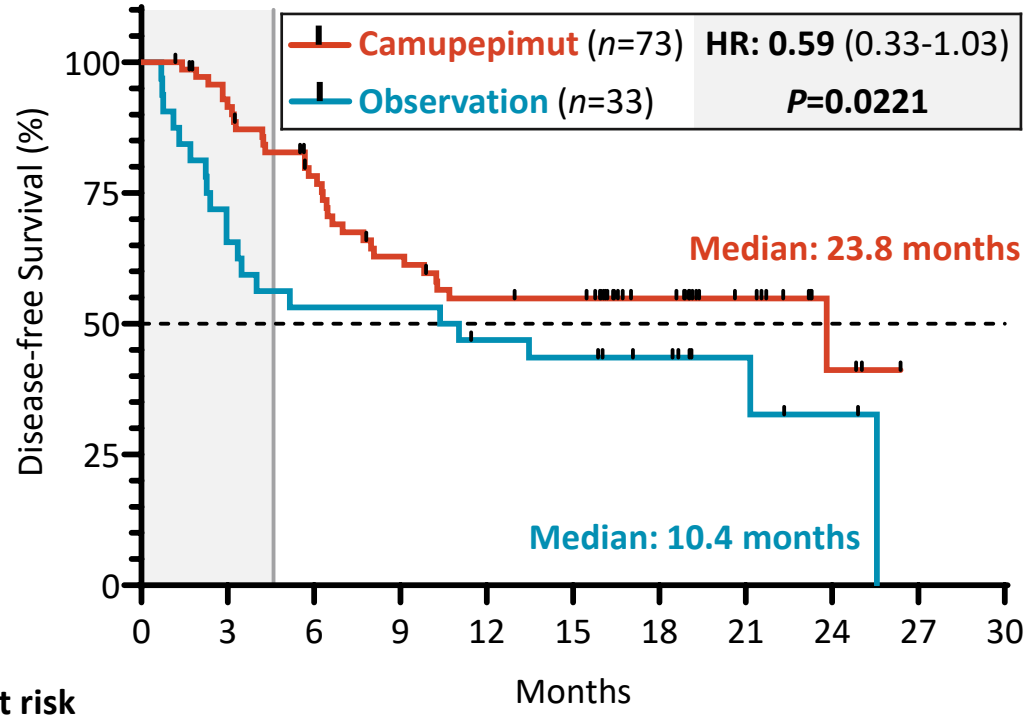
Stronger DFS Signal Observed with Camupepimut in Patients Without Prior Radiation

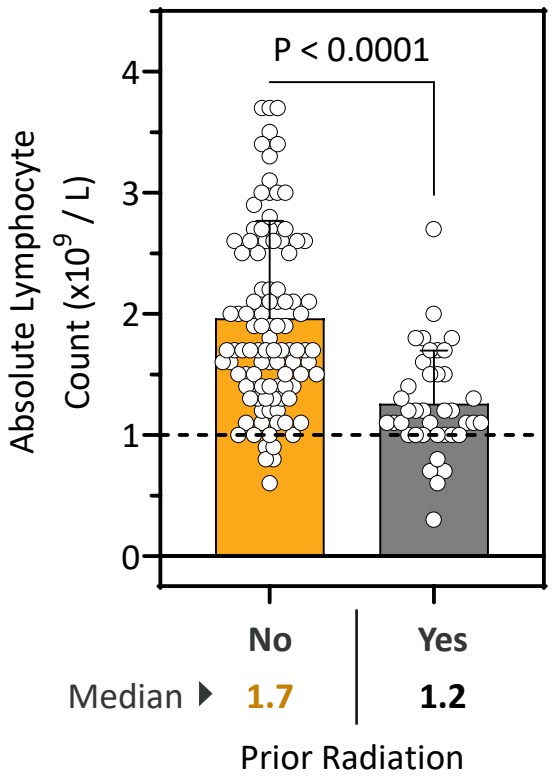
Prior radiation was associated with response to camupepimut; Treatment effect observed in no prior radiation

n=106 | **Prespecified:** | **No Prior Radiation**

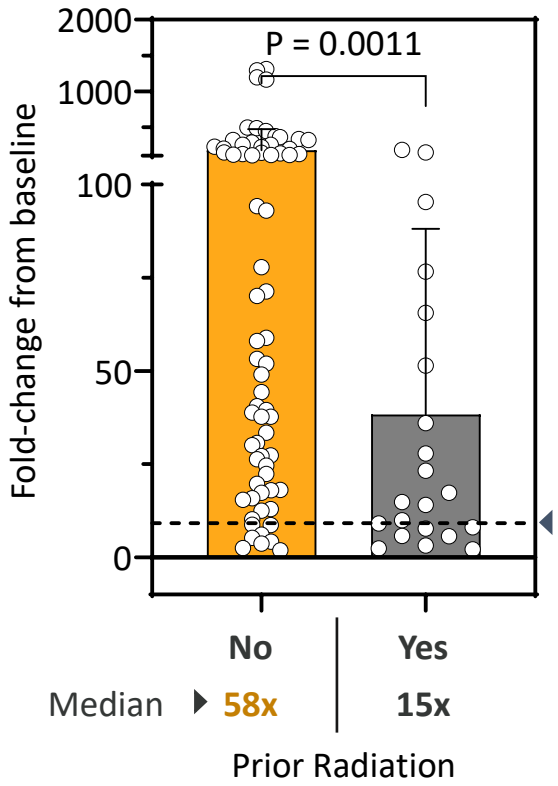


At risk										
Camupepimut	73	64	51	40	34	33	20	11	3	0
Observation	33	21	17	17	14	13	8	4	2	0

Baseline Lymphocytes



mKRAS T Cell Response



mKRAS T cell Response >9.17x

OR: 0.21 (0.07-0.64)

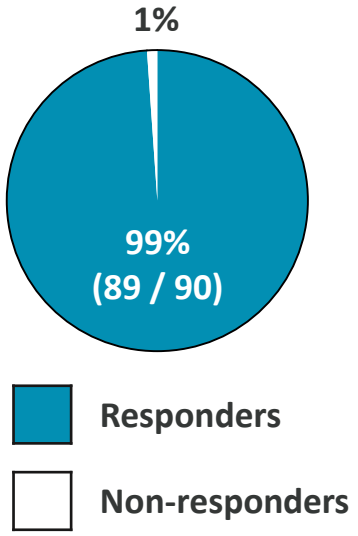
***P*=0.005**

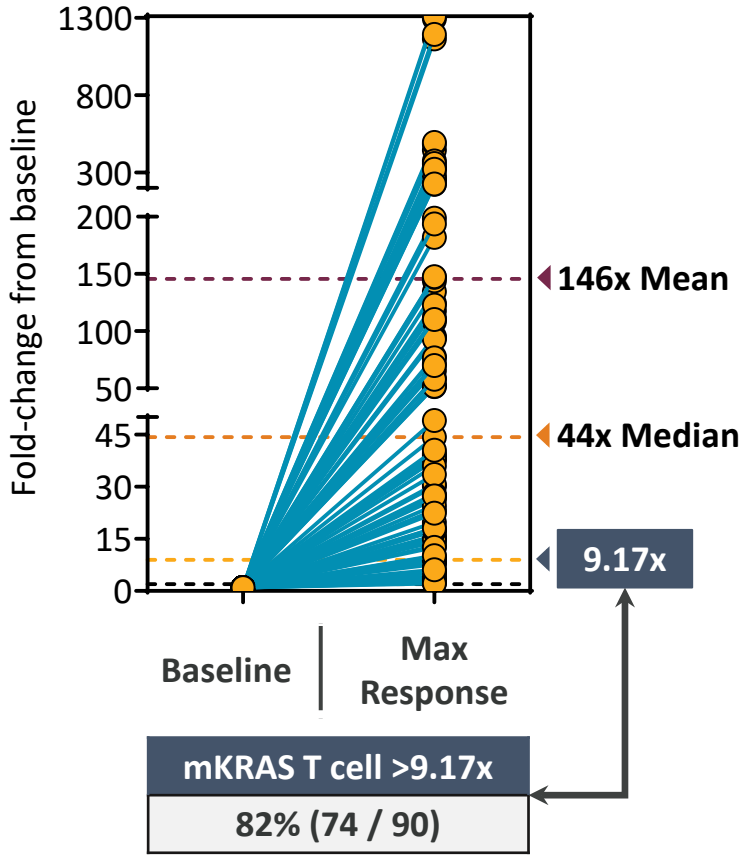
George, O'Reilly, et al. ***Nature Medicine***. 2026; O'Reilly, et al. ***Annals of Oncology***. 2026

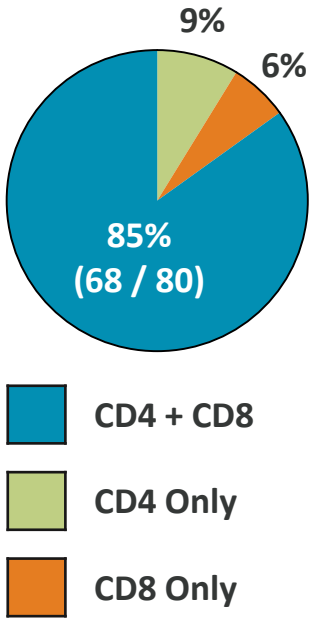
Camupepimut Induced Potent mKRAS-Specific T-Cell Responses in 99% of Patients

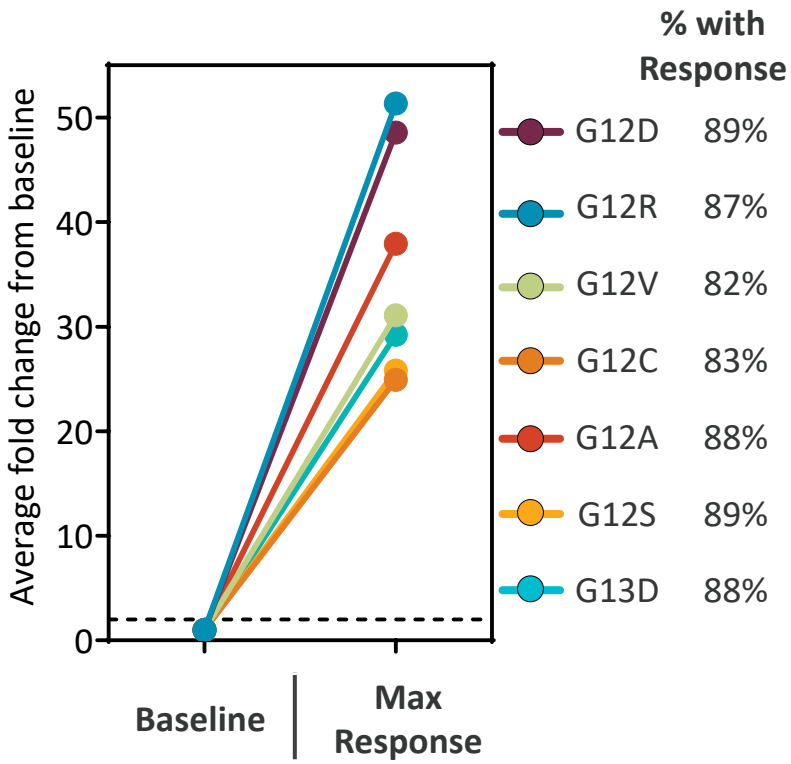
Robust CD4+/CD8+ responses across KRAS variants, 82% of patients achieving >9.17-fold T-cell expansion

T Cell Response | T cell Fold Change Over Baseline | CD4 / CD8 | mKRAS Variant Specificity









George, O'Reilly, et al. ***Nature Medicine****. 2026; O'Reilly, et al.* ***Annals of Oncology.*** *2026*

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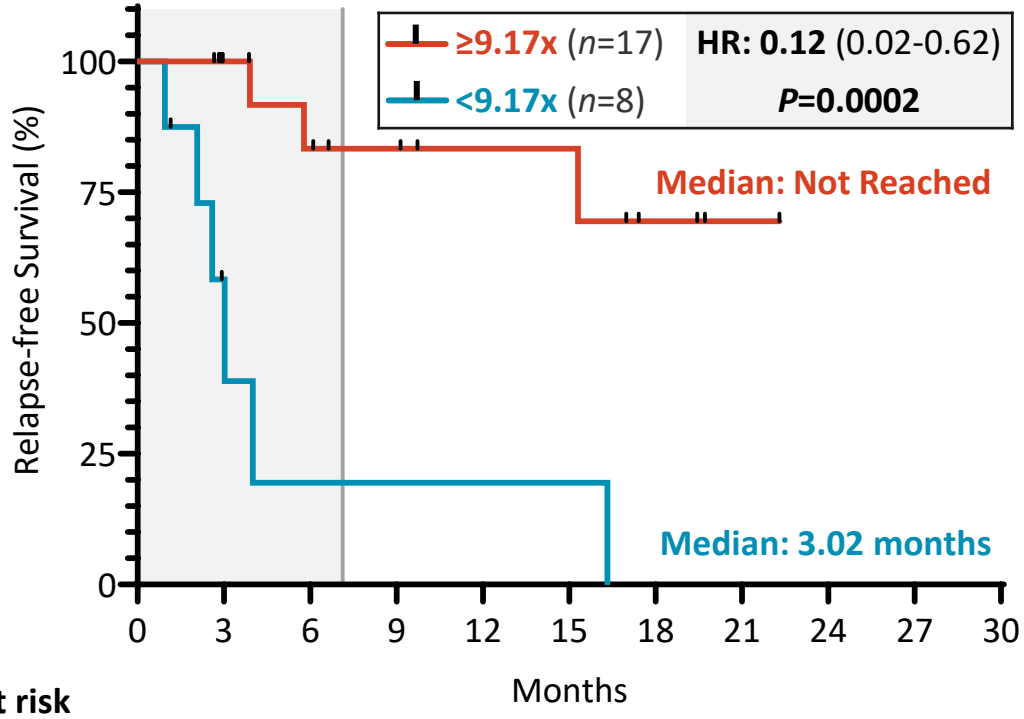
mKRAS-Specific T-Cell Responses Consistently Associated with Improved Clinical Outcomes

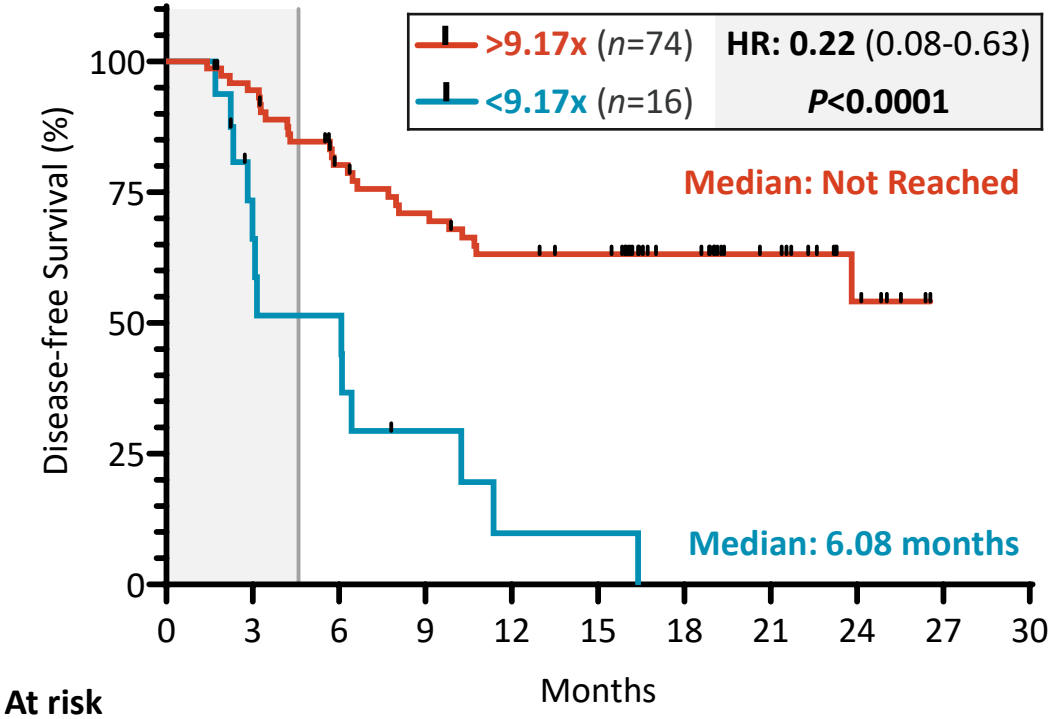
Phase 1 and 2 analyses demonstrate a strong association between T-cell response and improved RFS/DFS

n=25 | **Phase 1:** | **RFS Supervised by T cell Response**



At risk									
>9.17x	17	13	10	8	6	6	3	1	0
<9.17x	8	3	1	1	1	1	0		

n=90 | **Phase 2:** | **DFS Supervised by T cell Response**



At risk										
>9.17x	74	68	53	46	40	38	24	15	6	0
<9.17x	16	9	7	3	1	1	0			

Wainberg, O'Reilly, et al. ***Nature Medicine****. 2025; George, O'Reilly, et al.* ***Nature Medicine****. 2026; O'Reilly, et al.* ***Annals of Oncology.*** *2026*

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Favorable Safety Profile Observed with Camupepimut

Low rates of serious and Grade ≥3 adverse events, with no treatment-related deaths, support further evaluation in combination with RAS inhibitors and checkpoint inhibitors

Category	Camupepimut (*n*=96)	Observation (*n*=48)
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 [1]	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)

[1] No dose reductions; all dose modifications were dose delays

Proportionally Fewer Adverse Events Observed for Camupepimut vs. Observation SOC

- Proportionally fewer camupepimut patients had adverse events (AEs), Grade ≥3 AEs, and Serious AEs
- 0% stopped camupepimut for toxicity; no treatment-related mortality
- AE event terms were similar across groups (e.g. diarrhea, fatigue in both arms reflected prior Whipple surgery)

George, O'Reilly, et al. ***Nature Medicine****. 2026; O'Reilly, et al.* ***Annals of Oncology.*** *2026*

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Phase 2 Insights Define a Precision Medicine Strategy for Pivotal Phase 3

Refined patient selection, extended treatment duration, and potential checkpoint inhibitor combinations aim to maximize clinical benefit in adjuvant PDAC

Refine Patient Eligibility	Focus on large patient subgroups with the greatest potential to benefit from treatment based on subgroup analyses
Extend Treatment Duration	Robust early efficacy during the dosing period with DFS separation maintained through 9 months
Potential Evaluation of ICB Combination	Observation of complete responses in camupepimut 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.

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Camupepimut:
Boosting Anti-tumor Immunity Targeting mKRAS

Metastatic PDAC: Complete Response Observations

Multiple Confirmed Complete Responses Observed Following Camupepimut Treatment, Subsequent Therapy in metastatic PDAC

Hypothesis: Camupepimut 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 camupepimut 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 camupepimut in combination with checkpoint inhibition in metastatic mKRAS pancreatic cancer.

MSS: Micro-satellite stable; MMR: Mismatch Repair

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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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Complete Responses Observed Following Camupepimut and Subsequent Checkpoint Inhibition

Three patients who recurred following camupepimut 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 camupepimut 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

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Complete Responses Were Confirmed and Durable [1]

Durable complete responses observed following subsequent nivolumab-based therapy in three patients previously treated with camupepimut

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 camupepimut 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

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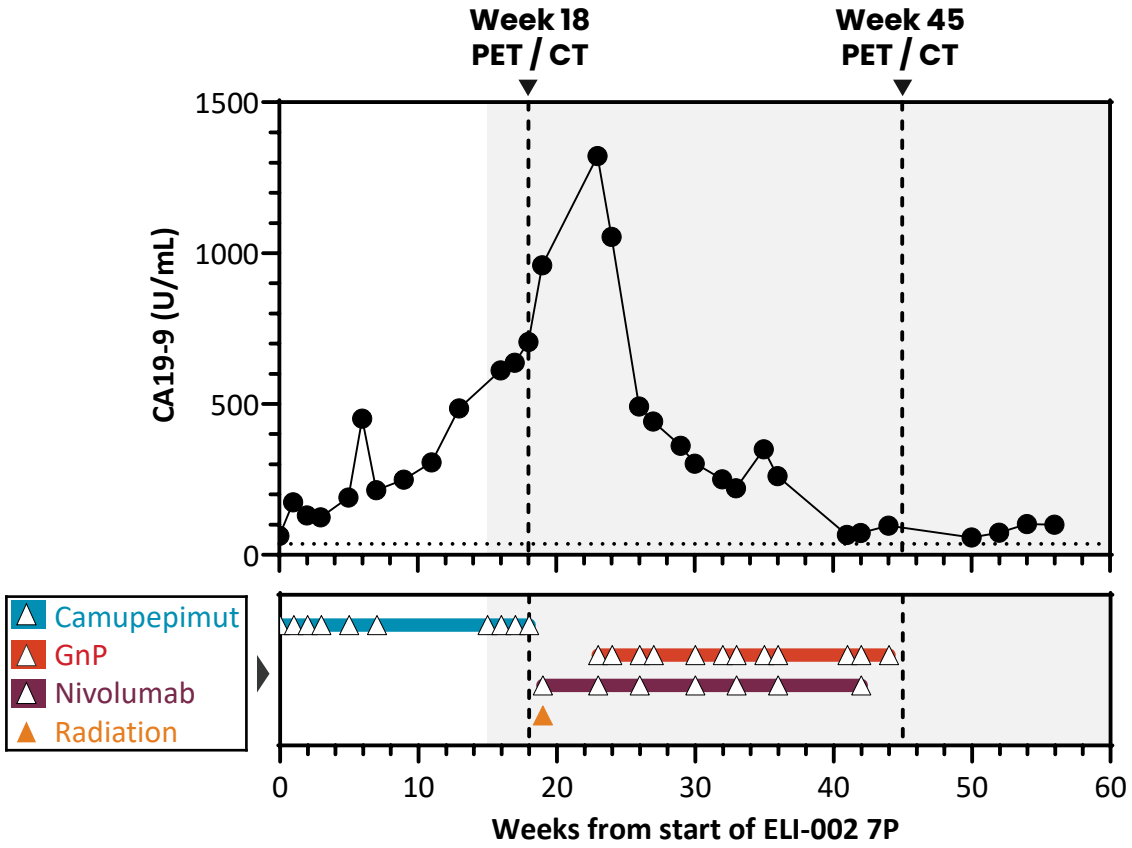
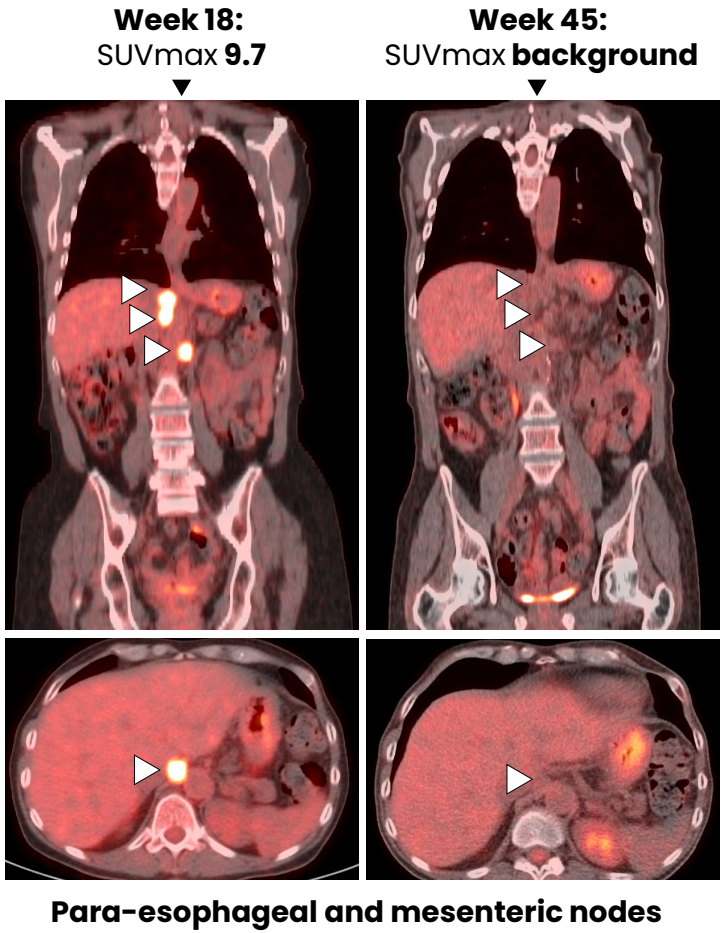
Patient 1: Complete Response Following Camupepimut and Subsequent Nivolumab

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

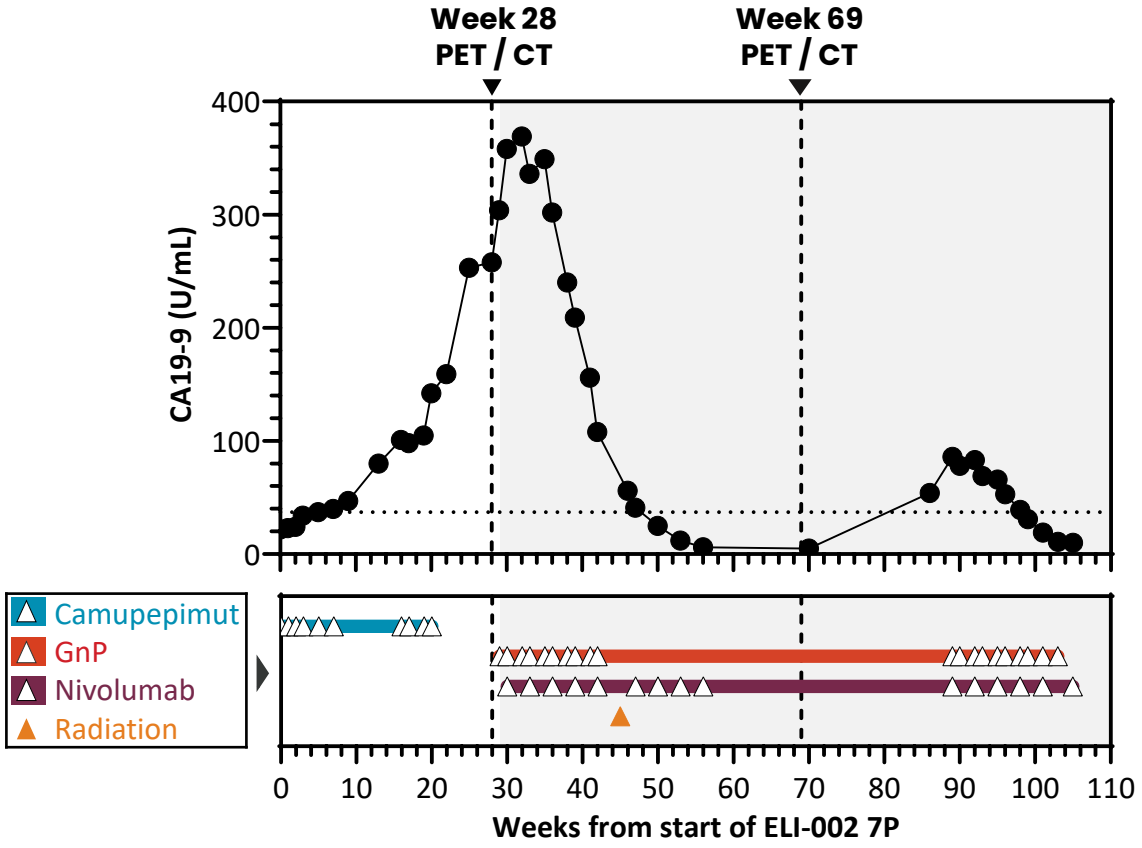
PFS: 8.3 months

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

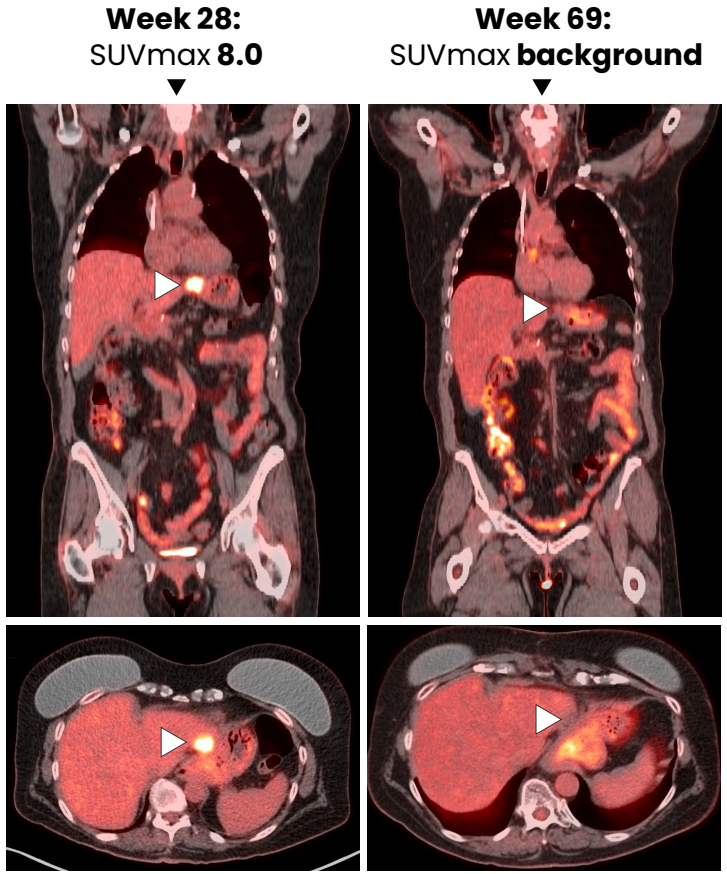
Patient 2: Complete Response Following Camupepimut and Subsequent Nivolumab

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

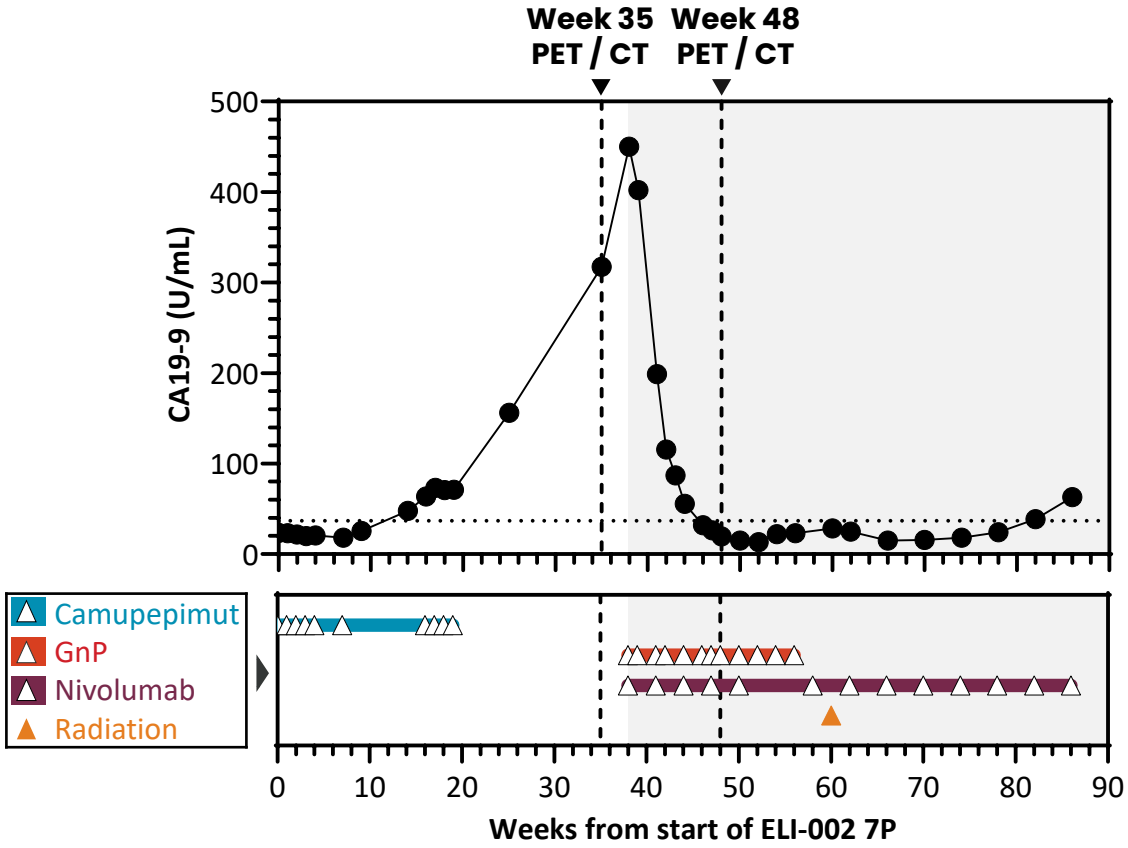
PFS: 13.4 months

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

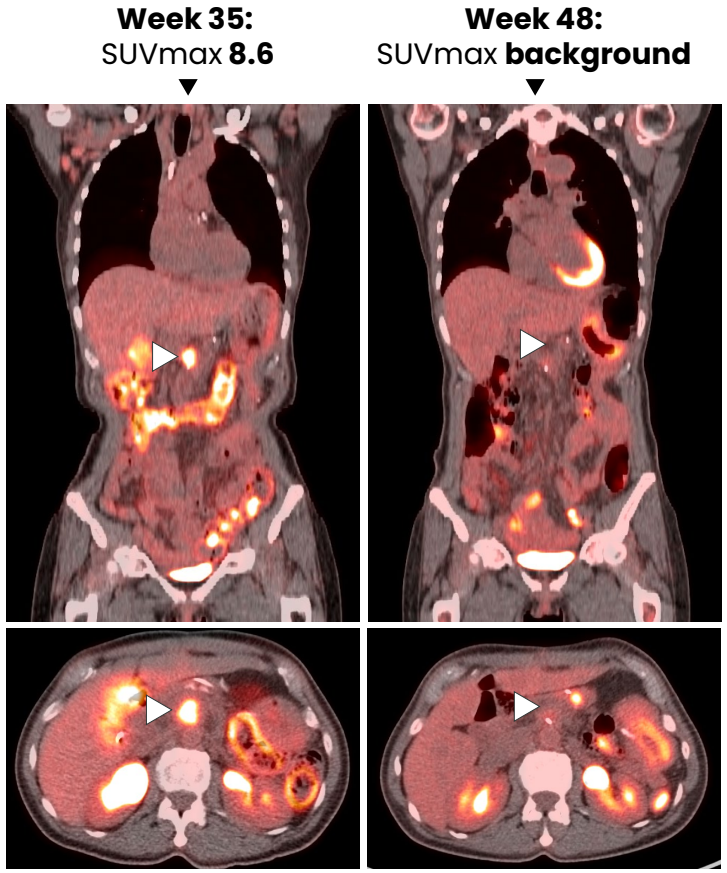
Patient 3: Complete Response Following Camupepimut and Subsequent Nivolumab

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

Hypothesized Mechanism for Complete Responses Following Camupepimut and Subsequent Therapy

Camupepimut 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 Camupepimut 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 camupepimut treatment and subsequent nivolumab-based therapy.

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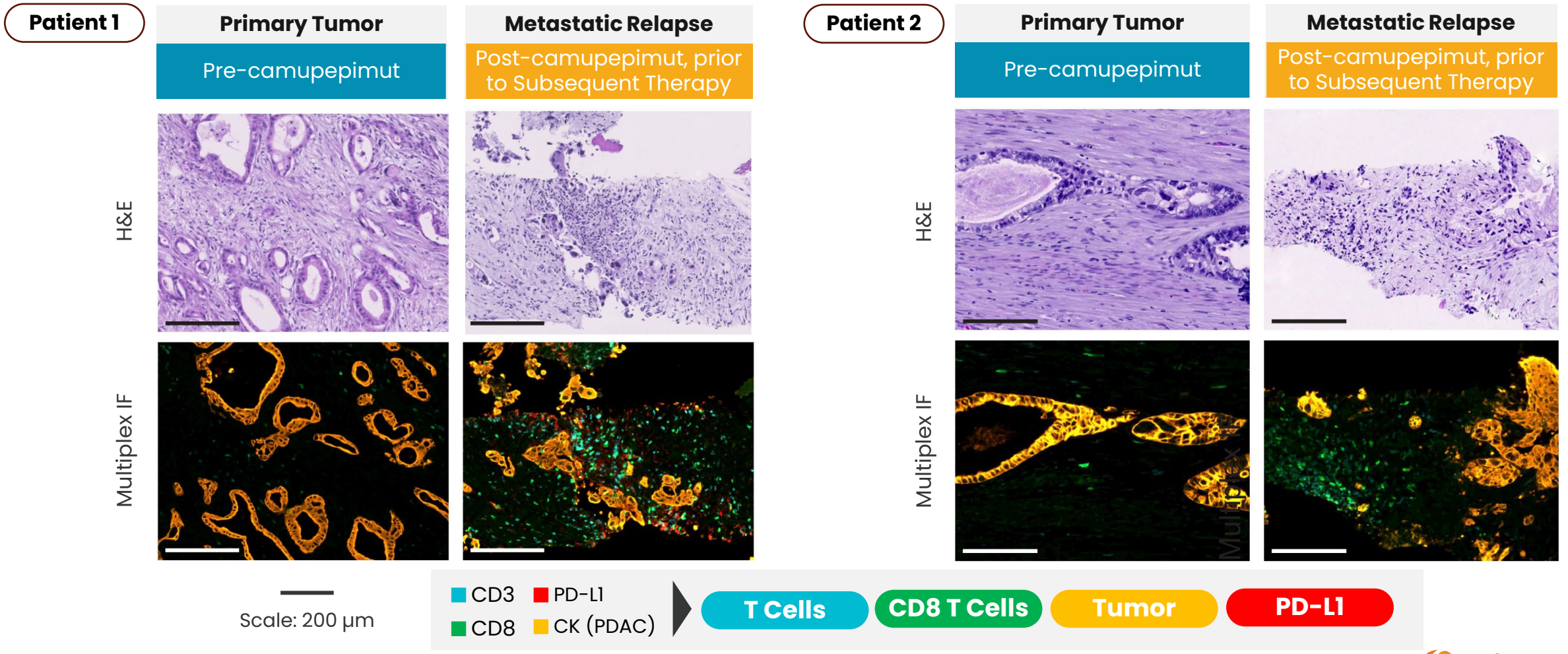
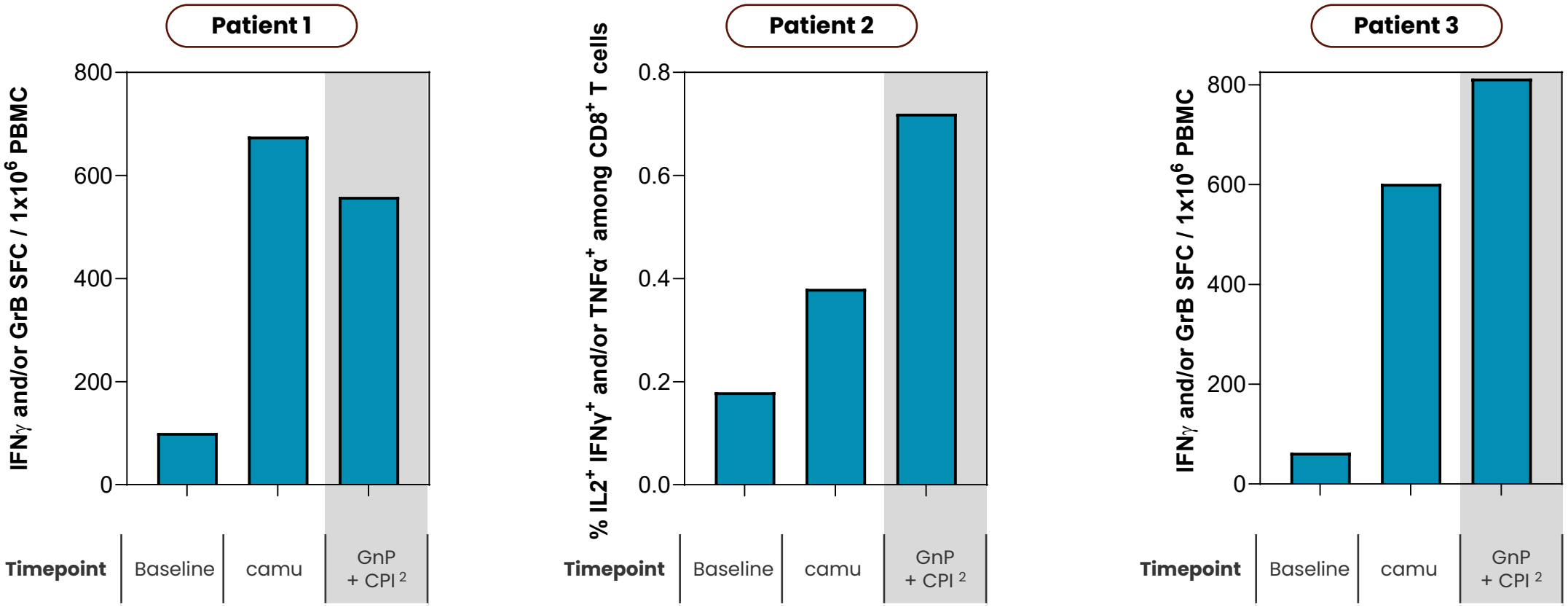
T cell Infiltration after Camupepimut Preceded Complete Responses

Observations of increased T cell tumor infiltration, and modulation of tumor PD-L1 following recurrence after camupepimut



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 camupepimut priming) and final post subsequent therapy timepoints; camu: camupepimut [2] Subsequent therapy included GnP, nivolumab, and SBRT; GnP: Gemcitabine Nab-paclitaxel; CPI: Checkpoint Inhibition

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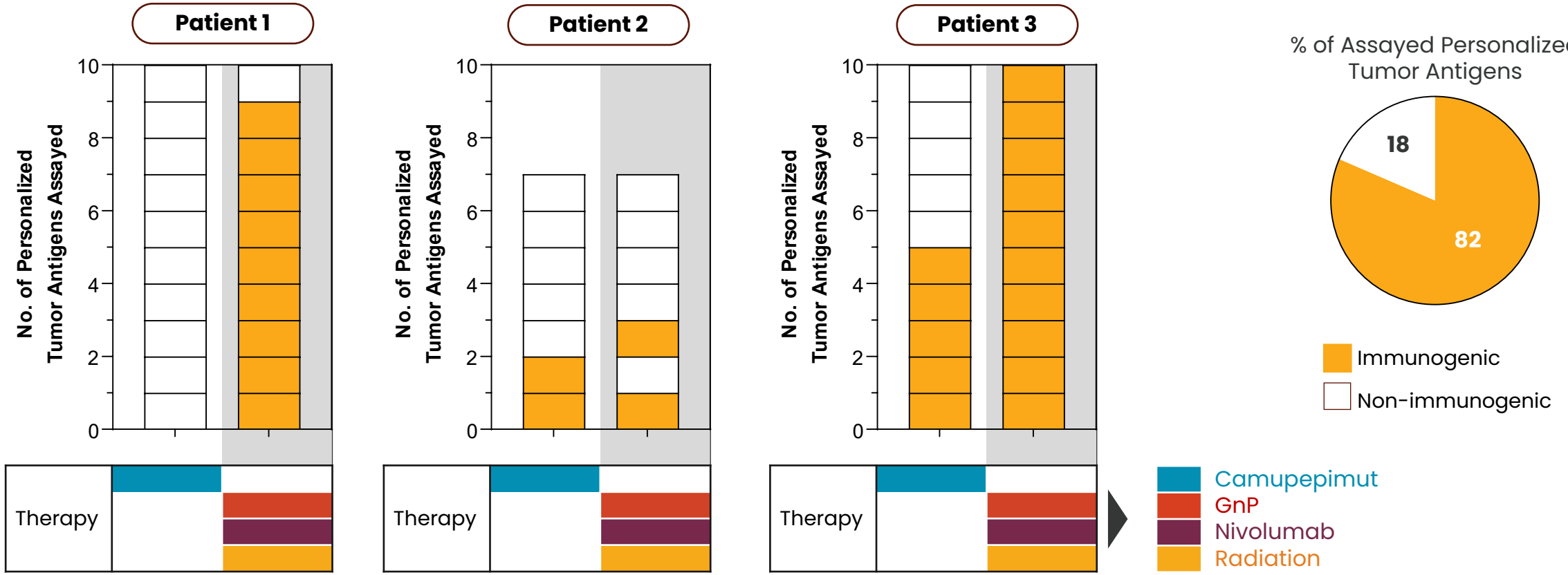
Complete Responses Associated with Personalized T Cell Responses

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



These findings suggest camupepimut-induced immunity may extend beyond targeted KRAS mutations and promote broader anti-tumor immune responses

GnP: Gemcitabine Nab-paclitaxel

AMPLIFY-103: Open Label, Phase 1 Trial in Metastatic mKRAS+ PDAC

CLINICAL STUDY: Initiation expected Q4 2026

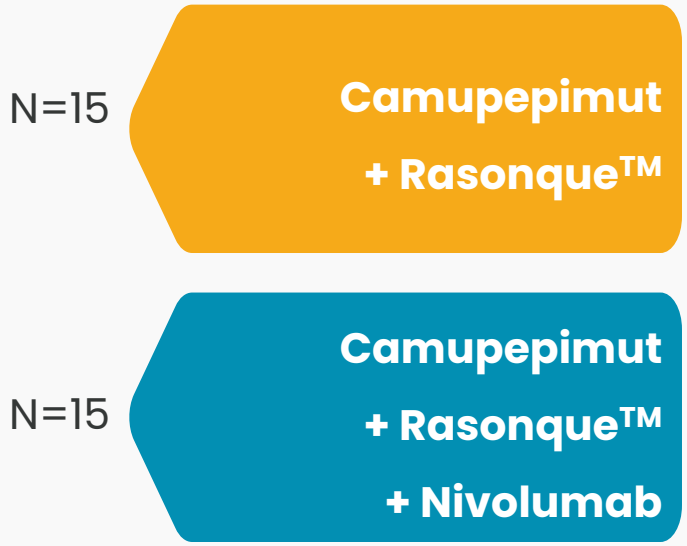
Designed to evaluate whether camupepimut could improve ORR in combination with RAS inhibition +/- immune checkpoint blockade in metastatic PDAC

Camupepimut Combination

- Doublet (+ Rasonque™) and
- Triplet (+ Rasonque™ + Nivolumab)

Phase 1: Key Criteria

- Includes: mKRAS G12D/R/V/C/A/S/G13D
- Previously treated metastatic PDAC
- Prior 1L SOC by NCCN guidelines (e.g. FOLFIRINOX, GnP)
- Measurable disease by RECIST 1.1
- Excludes prior radiation



Endpoints

Primary Endpoints:

- **ORR (assessed by RECIST 1.1)**
- **Safety**

Secondary Endpoints:

- ORR (assessed by PERCIST)
- Progression-free Survival
- ctDNA reduction or clearance from baseline
- CA19-9 / CEA reduction or clearance from baseline
- Immunogenicity

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Camupepimut:
Boosting Anti-tumor Immunity Targeting mKRAS

Neoadjuvant PDAC: Phase 1 Investigator Initiated Trial

Open Label, Phase 1 Trial in Resectable mKRAS+ PDAC

CLINICAL STUDY OVERVIEW: NCT07671339

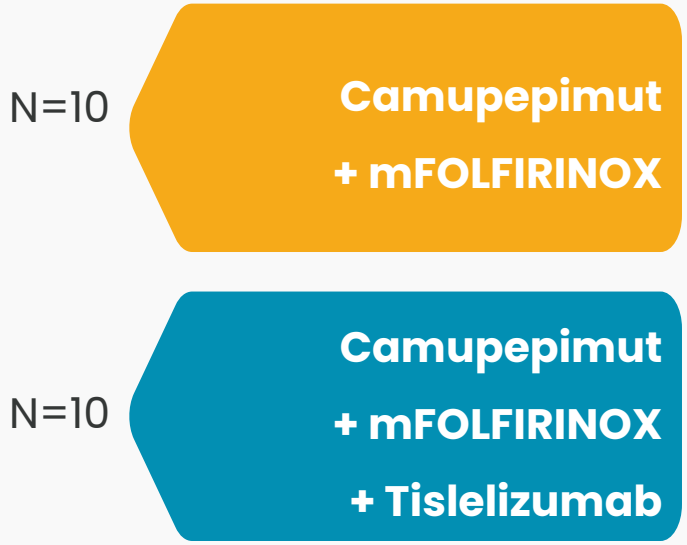
Designed to evaluate safety of camupepimut in combination with chemotherapy and immune checkpoint blockade in resectable, or borderline resectable PDAC

Camupepimut Combination

- Doublet (+ mFOLFIRINOX) and
- Triplet (+ mFOLFIRINOX + Tislelizumab)

Phase 1: Key Criteria

- Includes: mKRAS G12D/R/V/C/A/S/G13D
- Resectable, borderline resectable PDAC
- Imaging negative for metastatic disease



Endpoints

Primary Endpoint:

- **Safety**

Next Steps: Advancing Clinical Development of Camupepimut in Metastatic PDAC in Combination with Rasonque™ and Nivolumab

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

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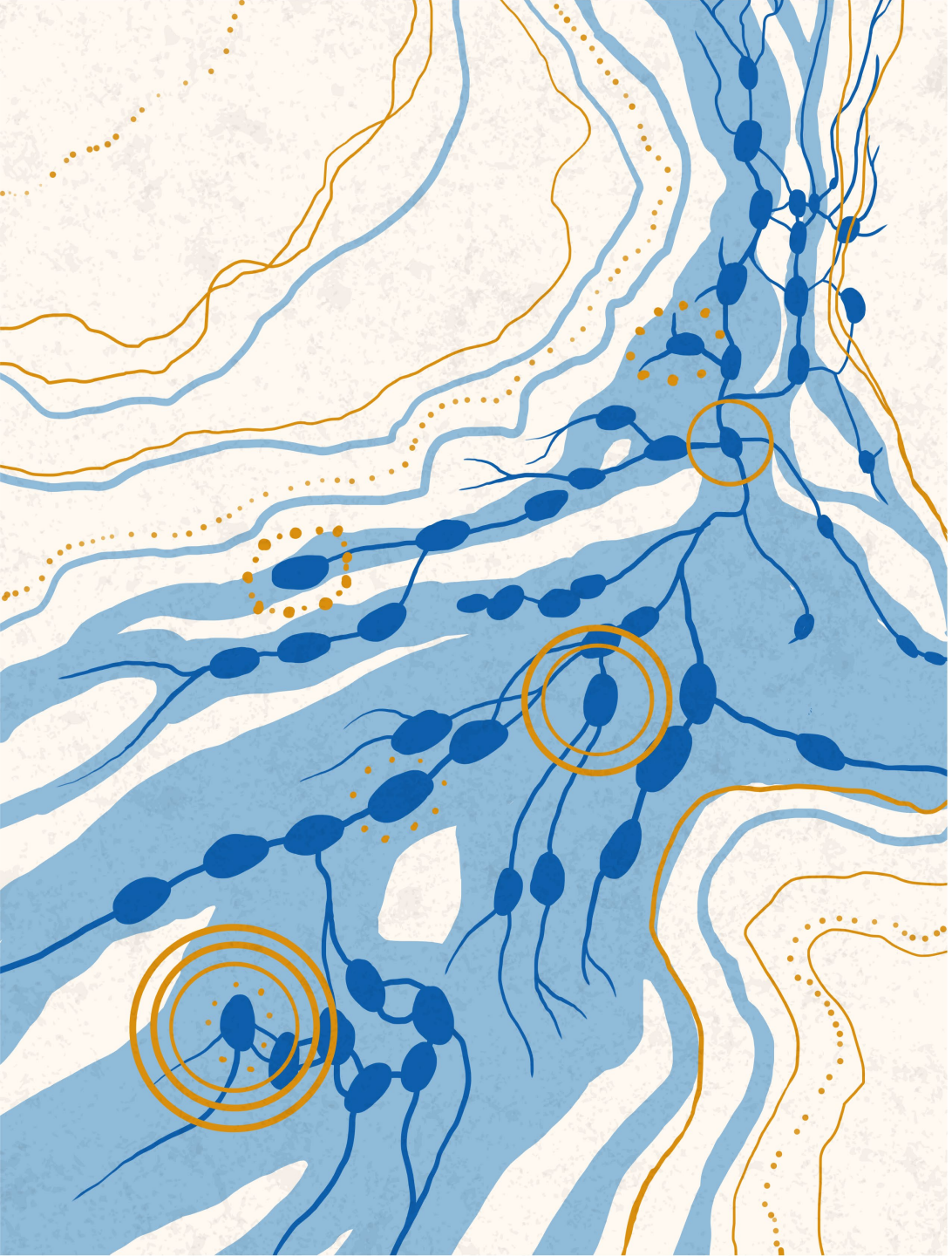

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