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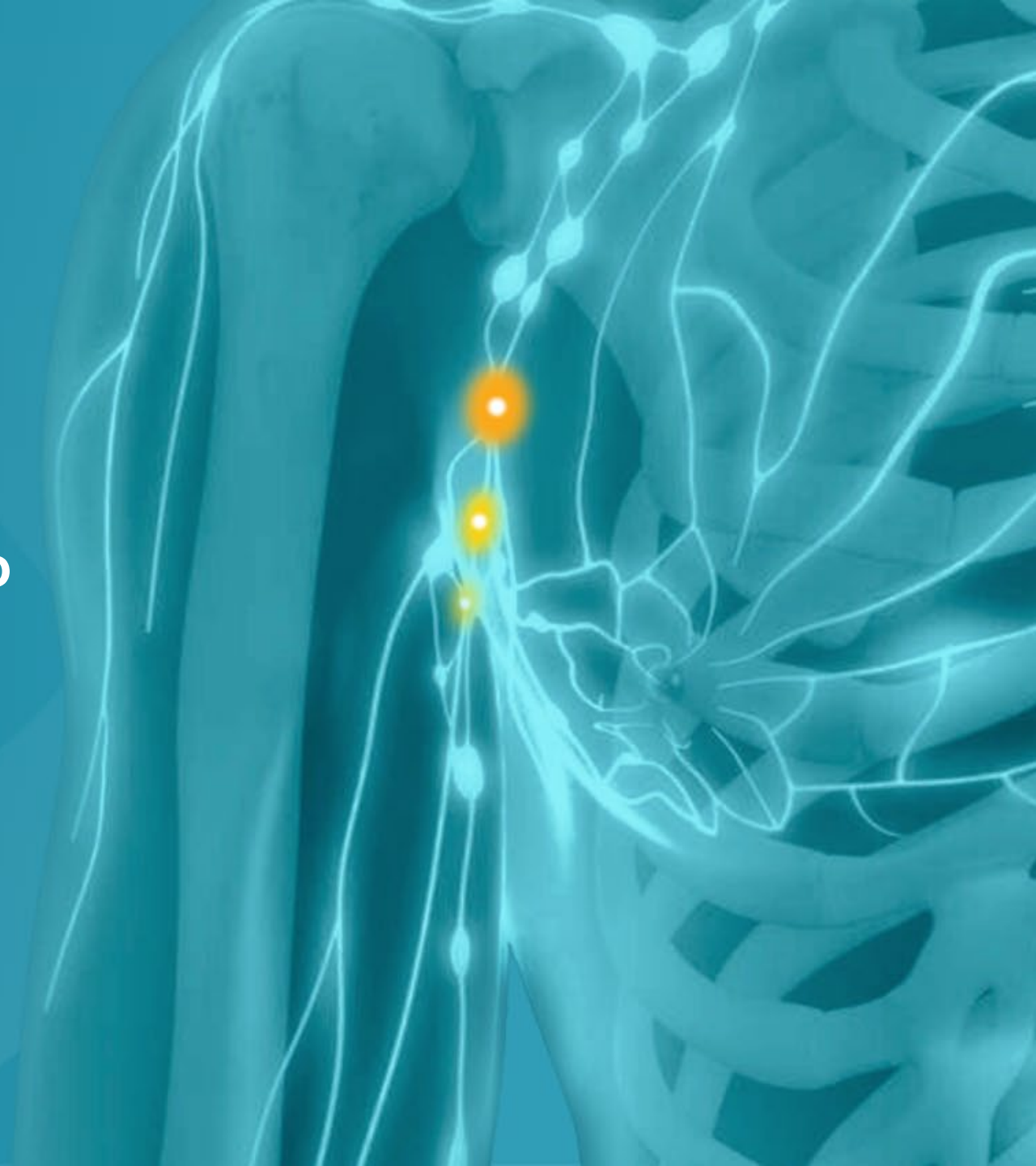
Targeting the Lymph Nodes to AMPlify Immunotherapy

January 2024



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Company Highlights

Clinical-stage biotech pioneering novel lymph node targeted cancer immunotherapies

Novel Approach to Immunotherapy

- Amphiphile or "AMP" platform traffics payloads to lymph nodes to generate robust immune responses

ELI-002: A Unique Lymph Node Targeted Vaccine

- Designed to target 7 KRAS mutations that drive ~25% of solid tumors
- Initial focus is PDAC where ELI-002 could address 88% of incident tumors

Phase 1a Clinical Data

- 84% of patients showed T cell responses with an average 58x increase in T cell numbers
- 84% of patients showed a decline in tumor biomarker, 24% having complete clearance
- Patients achieving large T cell response have an 86% decrease in risk of progression or death

Anticipated Near Term Catalysts

- Ongoing: Two Phase 1 trials with additional data available through 2024-2025
- Initiation of Phase 2 PDAC monotherapy 1Q 2024

Pipeline

Innovative pipeline of cancer immunotherapies addressing critical unmet needs

Candidate	Target	Indications	Preclinical	Phase 1	Phase 2	Phase 3
ELI-002	mKRAS	PDAC, CRC			**PDAC**	
ELI-007	mBRAF	GI Tumors				
ELI-008	mTP53	GI Tumors				

CRC: colorectal carcinoma | PDAC: Pancreatic pancreatic ductal adenocarcinoma
mKRAS: mutant Kirsten rat sarcoma | mBRAF: mutant homolog B of the Rapidly Accelerated Fibrosarcoma | mTP53: mutant tumor protein p53

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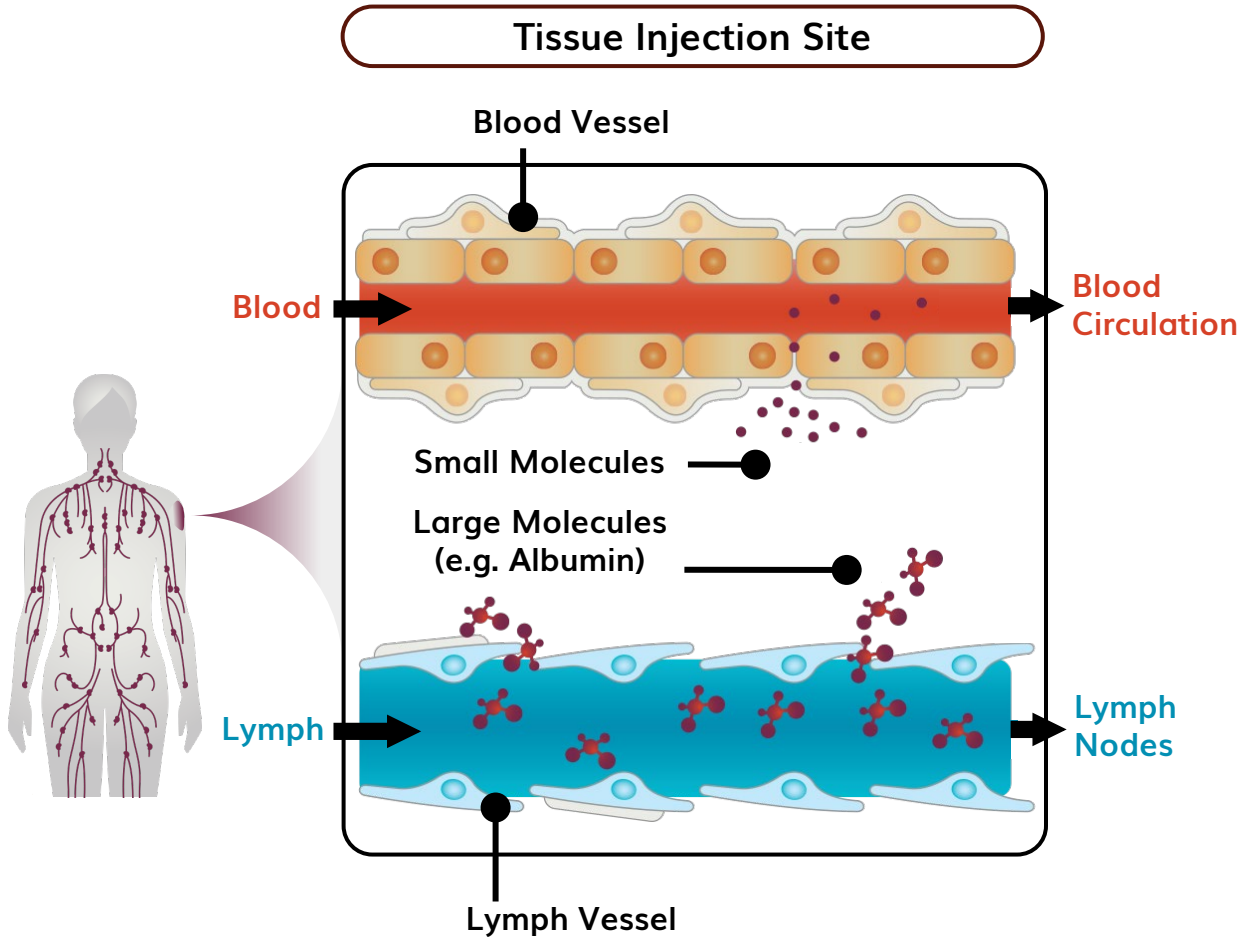
Elicio: Amplifying Immunotherapy

The AMP Platform

Harnessing the untapped potential of the lymph nodes for immunotherapy

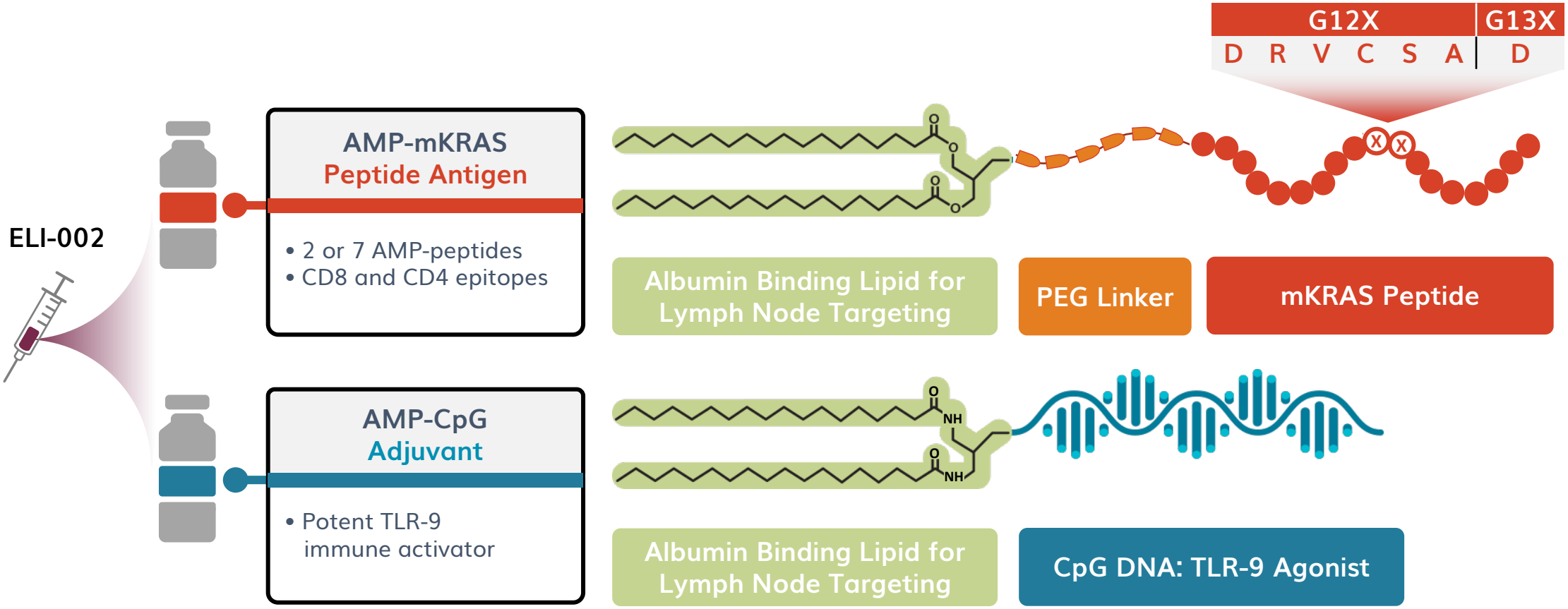
Targeting Immunity in the Lymph Nodes

- Most immune cells are located in lymph nodes, yet these critical sites are not engaged by conventional immunotherapies
- AMP promotes targeted delivery of payloads to the lymph nodes via “albumin hitchhiking”
- **AMP harnesses the unique biology of the lymph nodes to enhance the magnitude, potency, and durability of immune responses**



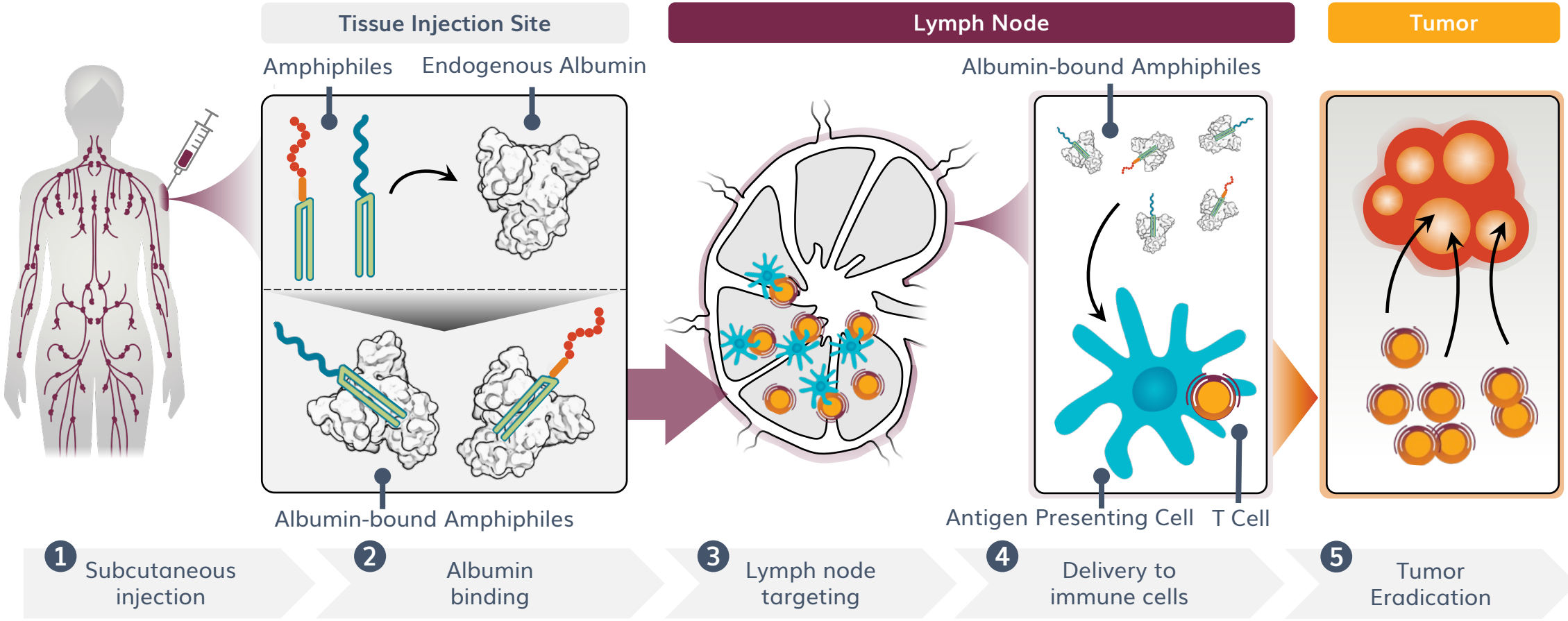
ELI-002 Composition

Lymph node targeted therapeutic vaccine comprised of AMP-peptides and AMP-CpG



Mechanism of Action

AMP immunotherapy generates an anti-tumor immune response via the lymph nodes

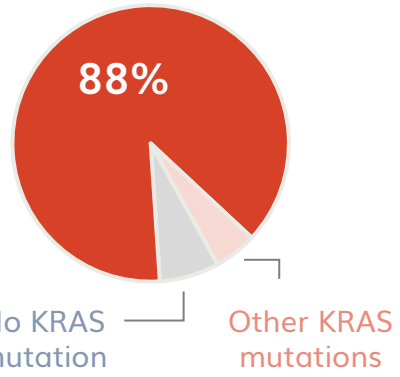


The mKRAS Opportunity

ELI-002 targets the 7 most common KRAS mutations driving 25% of solid tumors

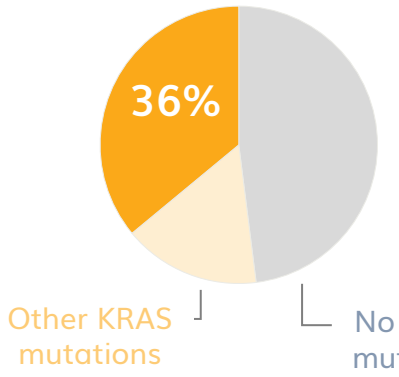
Pancreatic Ductal Adenocarcinoma

ELI-002 addressable
Incidence: ~128k



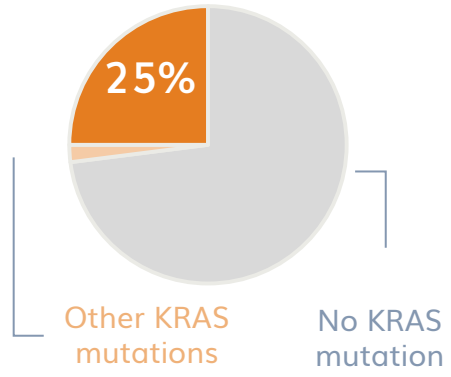
Colorectal Cancer

ELI-002 addressable
Incidence: ~192k



Non-Small Cell Lung Cancer

ELI-002 addressable
Incidence: ~128k



Other mKRAS Opportunities

17%

Biliary Tract Cancer Total incidence: ~40k

3-11%

Ovarian Cancer Total incidence: ~62k

Incidence for the 7 Major Markets (MM): US, France, Germany, Italy, Spain, UK, and Japan
Sources for tumor incidence obtained from GLOBOCAN (2020). PDAC: 90% of pancreatic cancers (O'Reilly, 2021), NSCLC 84.3% of lung cancers (SEER, 2021), BTC: 15% of liver cancers + gallbladder
Sources for KRAS mutation data: Waters & Der, 2018; Ji Luo, 2021, Meng 2021; Hofmann 2022, AACR Project GENIE Registry; Froesch et al, 2022, Gordon et al, 2023

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ELI-002's Differentiated Approach to mKRAS

Early mKRAS-targeting efforts in the clinic, while promising, leave significant white space

Small Molecules Directly Inhibiting Mutant KRAS

- FDA approvals for LUMAKRAS® & KRAZATI® validate target

BUT

- Only affects one mutation (G12C), subject to multiple resistance mechanisms

Small Molecules Indirectly Inhibiting Mutant KRAS

- May affect more than one mutation

BUT

- Unlikely to affect all mutations, still subject to bypass resistance mechanisms

Vaccines Targeting Mutant KRAS

- Validates immune-targeting, and affects multiple mutations

BUT

- Poor lymph node targeting and weaker T-cell activation

elicio THERAPEUTICS — Lymph Node Targeted Vaccine vs Mutant KRAS

- 7 key mutations
- Reduced risk of resistance mechanisms
- Potent activation of immune mechanisms
- Expansion/activation of T cell response
- Promotion of anti-tumor T cell function

ELI-002: Clinical Development Program

ELI-002 At-a-Glance

mKRAS immunotherapy eliciting strong T cell activity leveraging the AMP platform's lymph node targeting design

2 Peptide (2P) & 7 Peptide (7P) Formulations

- 2P used in Phase 1 AMPLIFY-201 for clinical proof-of-concept while 7P CMC finalized
- Program now switches to full 7P formulation, to maximize efficacy and opportunity

AMPLIFY Clinical Program Underway

- Phase 1 AMPLIFY-201 fully enrolled with positive tumor and mechanism of action (T cell) biomarker responses correlating with clinical outcome reported
- Phase 1/2 AMPLIFY-7P enrollment underway, randomized Phase 2 in PDAC expected to start in 2024

Distinct Clinical & Operational Advantages

- Vs comparable vaccines including targeting driver mutations and "off-the-shelf"

AMPLIFY-201 Study Overview

Phase 1 dose-ranging study to assess safety and efficacy of ELI-002 2P as adjuvant treatment in patients who completed standard therapy and have molecular disease

CLINICAL PROGRAM OVERVIEW: NCT04853017

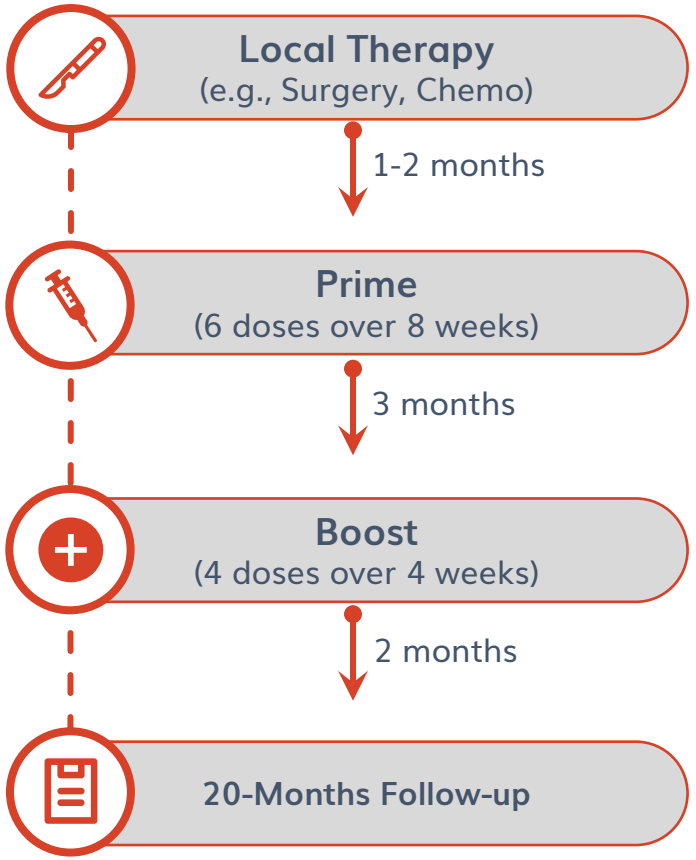
Key Criteria

- ✓ mKRAS G12D / R – aligned to 2 peptide formulation
- ✓ No metastatic disease after locoregional treatment
- ✓ No radiographic evidence of disease (NED)
- ✓ High risk of relapse (MRD+ ctDNA/serum biomarkers)

Baseline Characteristics

25 patients enrolled across 5 dose cohorts, 23 evaluable at database cutoff (4/25/2023)

- **Advanced:** 68% had stage III or oligometastatic resected stage IV disease
- **Pre-treated:** All received prior chemo and surgery, 28% had prior radiation



Basket Trial Enrollment

Pancreatic Ductal Adenocarcinoma (PDAC)	n=20
Colorectal Cancer (CRC)	n=5

Endpoints

- **Safety**
- **Maximum Tolerated Dose (MTD) or RP2D**
- ctDNA/serum biomarker change from baseline
- Immunological Responses
- Relapse Free Survival (RFS)

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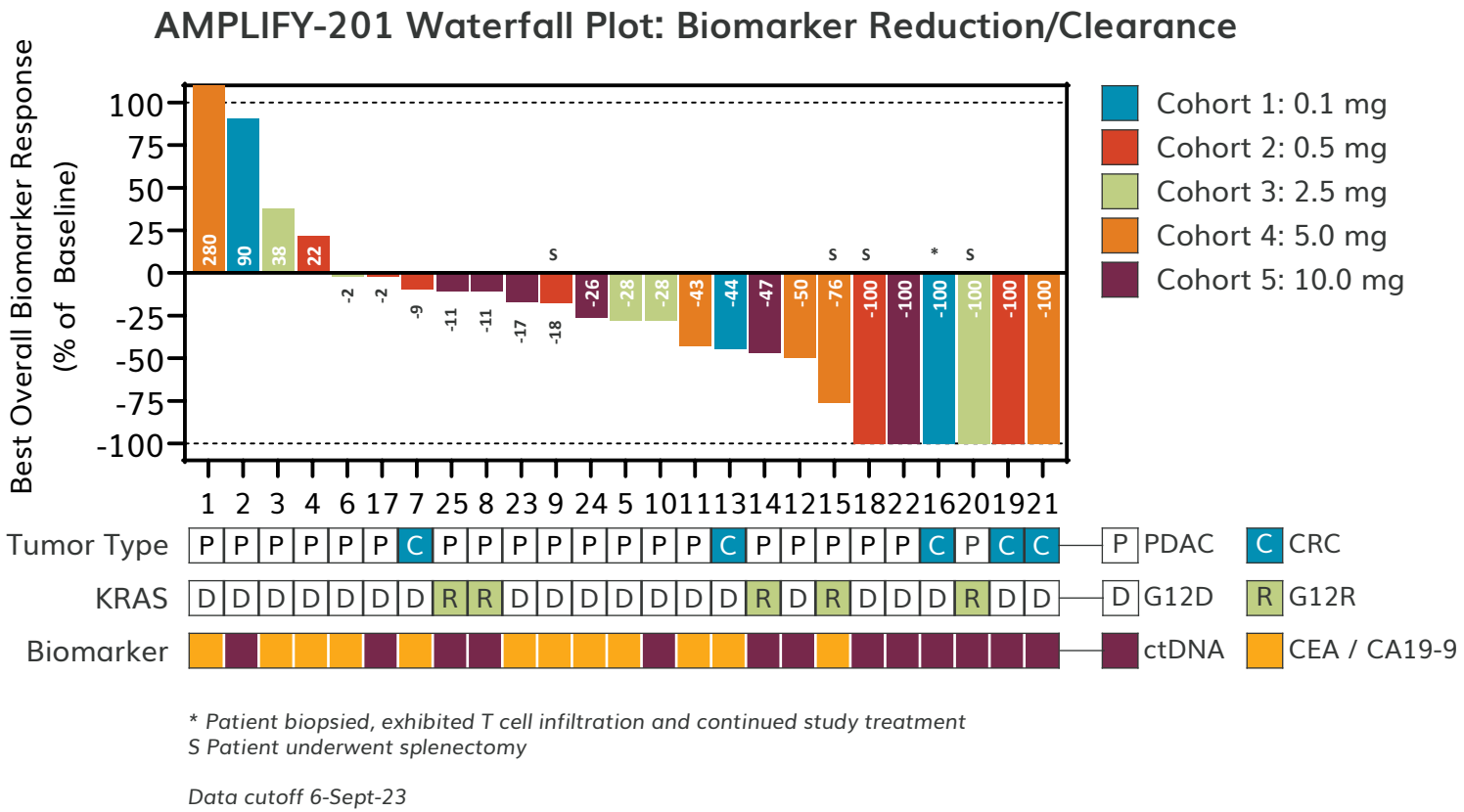
AMPLIFY-201: Tumor Biomarker Responses

Robust responses observed across tumor types and KRAS mutations with ELI-002 monotherapy

Tumor Biomarker Responses

- **24% of patients (6/25) showed complete clearance of ctDNA**
- Most patients (84%, 21/25) showed decline from baseline in ctDNA or CEA/CA19-9 levels
- 44% (11/25) showed a >30% reduction in biomarker levels
- Waterfall displays best response of ctDNA or serum tumor biomarker



** Patient biopsied, exhibited T cell infiltration and continued study treatment*
S Patient underwent splenectomy

Data cutoff 6-Sept-23

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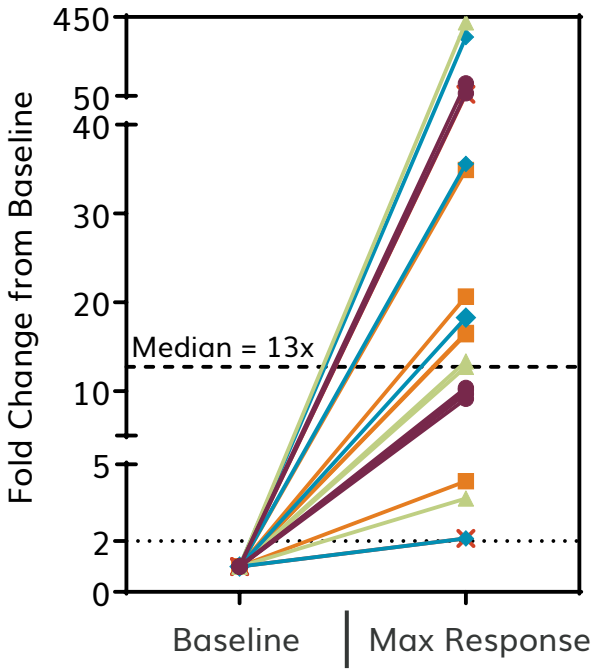
AMPLIFY-201: 84% patients generated mKRAS specific T Cells

mKRAS T Cell Responses

- **84% of patients showed T cell responses**
- 100% in two highest dose cohorts, including at the RP2D (10 mg)
- 58x average fold-change in T cell numbers from baseline (median 13x; range 2-423x)
- T cells detectable by standard direct *ex vivo* FluoroSpot and flow cytometry, with no expansion required

AMPLIFY-201 T Cell Fold-Changes by Dose Level

Direct Ex Vivo T Cell Response



Response per Dose Level

AMP-CpG Dose Level	*ex vivo* T cell response (n, %)	Average fold-change
0.1 mg	2/3 (67%)	30
0.5 mg	4/6 (67%)	101
2.5 mg	4/5 (80%)	113
5.0 mg	5/5 (100%)	19
10.0 mg	6/6 (100%)	36
Total	21/25 (84%)	58

Responses shown are best overall responses vs baseline for each patient at any timepoint during the assessment period.

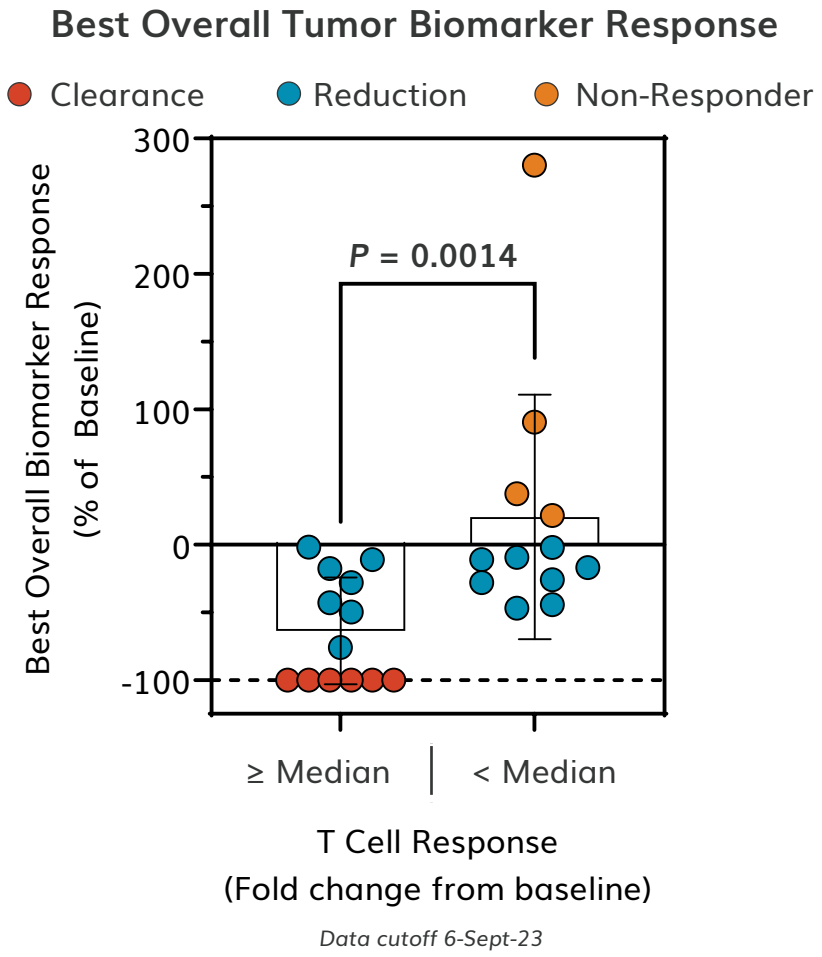
Data cutoff 6-Sept-23

AMPLIFY-201: T Cell Fold-Change Predicts Tumor Biomarker Response

All patients with T cell responses over median showed tumor biomarker response

mKRAS T Cell Response ➤ Tumor Biomarker Response

- **Strength of T cell response to ELI-002 is strongly correlated to tumor biomarker response**
- 100% of the above median T cell group respond to ELI-002; in the below median group 67% (8/12) respond to ELI-002
- All (100%) of the observed tumor biomarker clearances (6/6) are in the above median T cell group
- Statistically significant, p-value per Mann Whitney Test ($P < 0.0014$)



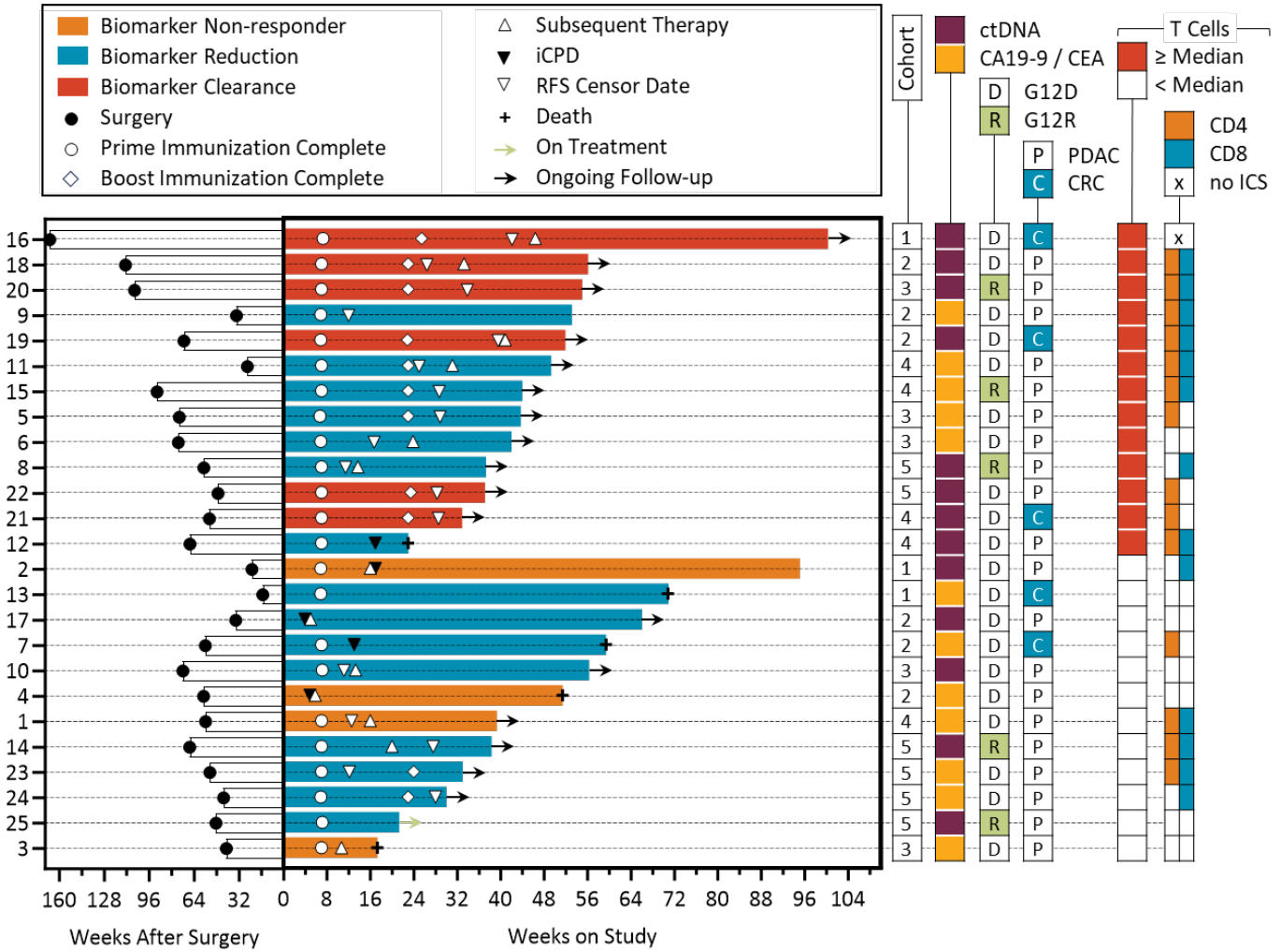
Data cutoff 6-Sept-23

AMPLIFY-201: Majority of Above Median T Cell Responders Include CD4+ and CD8+

Above median mKRAS-specific T cell response correlates to improved clinical outcome

mKRAS T Cell Response ➤ Clinical Response

- **Strength of T cell response to ELI-002 is correlated to tumor response and duration of ELI-002 administration**
- Patients with both CD4 and CD8 T cell responses have favorable clinical outcomes



Data cutoff 6-Sept-23

AMPLIFY-201: Above Median T Cell Responders Median RFS Significantly Prolonged

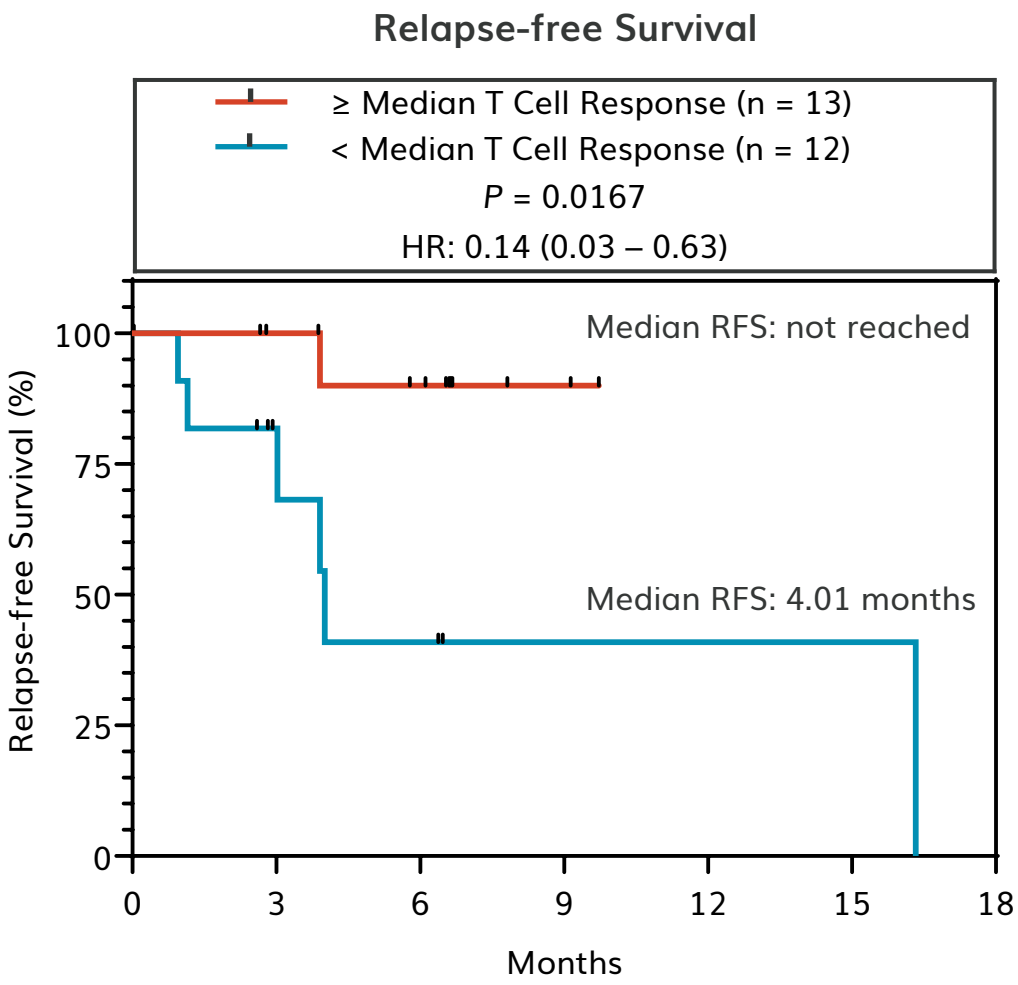
86% reduction in the risk of progression or death in above median T cell responders

mKRAS T Cell Response ➤ Clinical Response

- At a median follow up of 8.5 months, median RFS was not reached for above median T cell responders[1] compared to 4.01 months among below median T cell responders (**HR 0.14**, 95% CI 0.03-0.63, P=0.0167)
- **86% Reduction in Risk of Progression or Death in T cell responders to ELI-002**
- Median overall survival was not reached for either group

[1] Above median T cell responder: T cell response ≥ median increase of 12.75

Database cutoff 6-Sept-23



AMPLIFY-201: Safety & Tolerability

ELI-002 was well tolerated at all dose levels, with no DLTs or SAEs

ELI-002 Safety / Tolerability

- **No Grade 3/4 TEAEs, no CRS, no DLTs at time of data cutoff (6-Sept-2023)**
- 12/25 (48%) had Grade 1 or 2 AEs
- 4/25 (16%) had injection site reactions
- 10 mg dose selected as RP2D for Phase 1/2 AMPLIFY-7P study

TEAE: Treatment Emergent Adverse Event | CRS: Cytokine release syndrome | DLT: Dose-limiting toxicity | SAE: Serious adverse event | RP2D: Recommended Phase 2 Dose

	Cohort 1 (0.1 mg) n = 3	Cohort 2 (0.5 mg) n = 6	Cohort 3 (2.5 mg) n = 5	Cohort 4 (5.0 mg) n = 5	Cohort 5 (10.0 mg) n = 6	Overall n = 25
Adverse Event Term[a]						
Patients with Any Related TEAE, n (%)	1 (33.3)	3 (50.0)	2 (40.0)	2 (40.0)	4 (66.7)	12 (48.0)
Fatigue	0	2 (33.3)	2 (40.0)	1 (20.0)	1 (16.7)	6 (24.0)
Injection site reaction*	1 (33.3)	1 (16.7)	0	2 (40.0)	0	4 (16.0)
Myalgia	0	0	0	1 (20.0)	2 (33.3)	3 (12.0)
Anemia	1 (33.3)	0	1 (20.0)	0	0	2 (8.0)
Headache	1 (33.3)	1 (16.7)	0	0	0	2 (8.0)
Hot flush	0	1 (16.7)	0	0	1 (16.7)	2 (8.0)
Nasal congestion	0	1 (16.7)	0	1 (20.0)	0	2 (8.0)
Nausea	1 (33.3)	0	0	1 (20.0)	0	2 (8.0)

TEAE: Treatment Emergent Adverse Events with incidence ≥ 5%; data cutoff 6-Sept-2023
[a] Preferred terms per the Medical Dictionary for Regulatory Activities, version 25.0
*Injection Site Reaction = Injection Site Erythema, Injection Site Induration, Injection Site Swelling, Contusion, Pruritis

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AMPLIFY-201: T Cell Tumor Infiltration

Preliminary clinical evidence shows dense T cell tumor infiltration following ELI-002 therapy

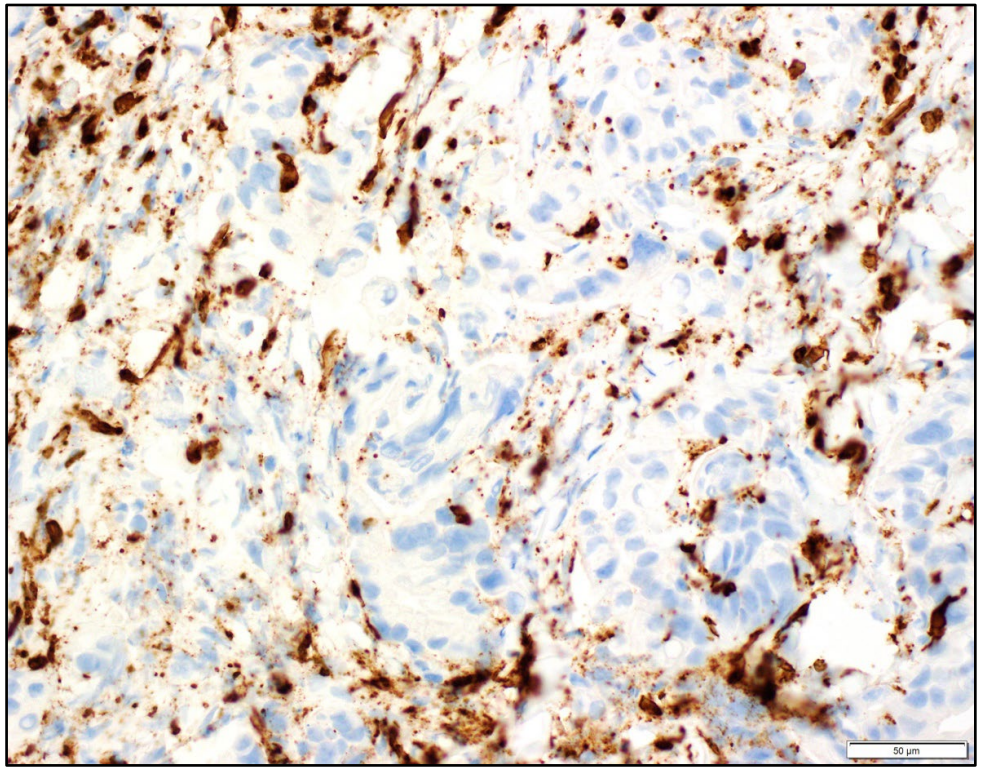
T Cell Tumor Infiltration

- **76 T cells/hpf at time of progression, 29x the expected 2-3 T cells / hpf in PDAC** [1]
- T cell tumor infiltration was associated with complete ctDNA clearance in this patient
- T cell tumor infiltration has been associated with increased survival in pancreatic cancer [2]

Hpf: High-powered field

[1] *Ademmer 1988 Clin Exp Immunol 112:21*
[2] *Ino, Y., Yamazaki-Itoh, R., Shimada, K. et al. Immune cell infiltration as an indicator of the immune microenvironment of pancreatic cancer. Br J Cancer 108, 914–923 (2013). https://doi.org/10.1038/bjc.2013.32*

Tumor Biopsy: CD3 Immunohistochemistry



Tumor Biopsy CD3 Immunohistochemistry: T Cell Receptor (brown)
Pancreatic tumor, 2.5 mg dose level

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AMPLIFY-201: Clinical Data Summary

ELI-002 monotherapy generates robust immune response that correlates with clinical benefit

Well Tolerated No Dose Limiting Toxicity

- No Grade 3/4 TEAEs, no CRS, No DLTs; 11/25 (44%) had Grade 1 or 2 AEs

Promising Preliminary Data

- Significant reduction in risk of progression or death with large T cell response
- Tumor biomarker reductions, clearance across different tumor types and KRAS mutations
- T cell response strongly correlates with tumor biomarker reduction/clearance and relapse free survival benefit

Robust mKRAS T Cell Responses

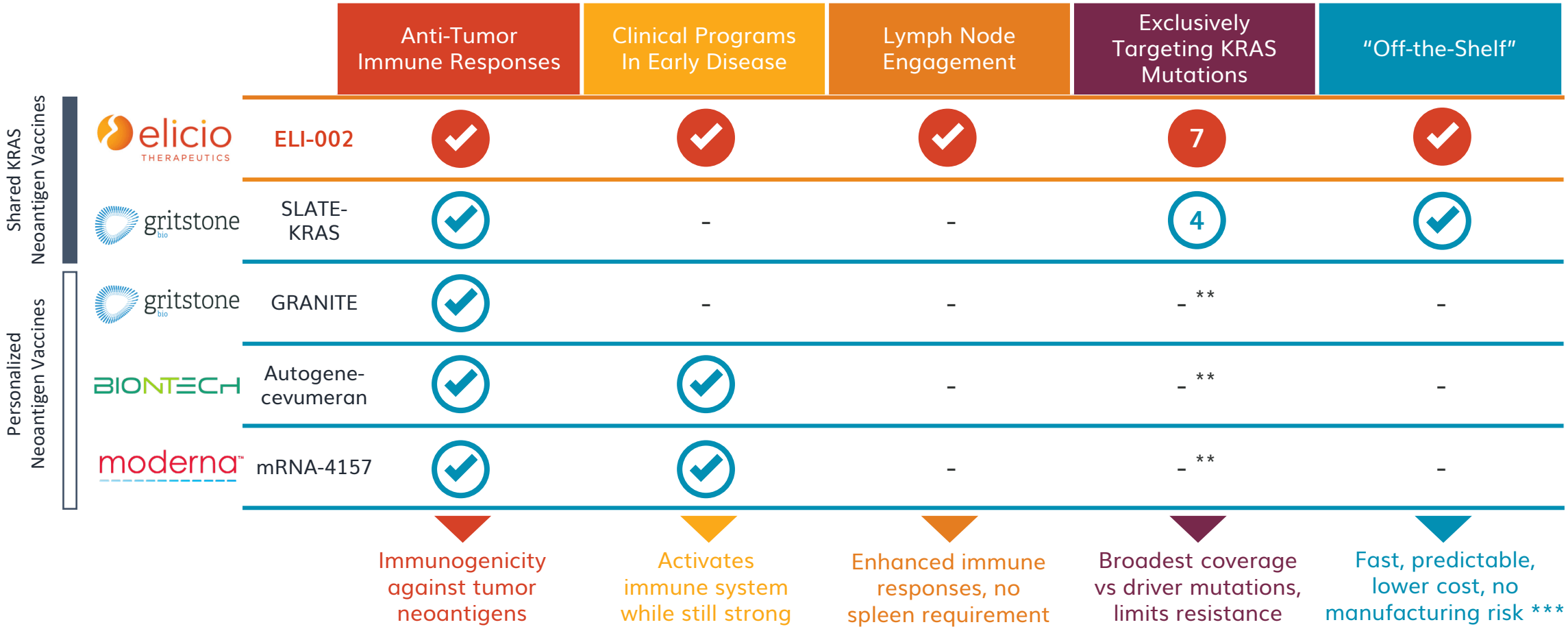
- T cell response and tumor infiltration observed in AMPLIFY-201 historically associated with survival in PDAC
- Able to generate KRAS-specific CD4+ and CD8+ response in majority of patients with large T cell response
- Among evaluable patients 100% of patients maintained elevated KRAS-specific T cell response post-boost
- 90% of patients developed T cell responses to two or more KRAS antigens

RP2D Selected

- AMPLIFY-7P IDMC has recommended the phase 2 dose of ELI-002 7P
- Randomized PDAC Phase 2 next portion of study to open (Q1 2024)

ELI-002 Differentiation*

Distinct clinical and operational advantages over comparable cancer vaccines



** Based on publicly available information*
*** Personalized neoantigen vaccines encode for multiple neoantigens which may or may not include KRAS neoantigens, but do not exclusively target KRAS driver mutations*
**** No risk associated with 'just-in-time' manufacturing that impacts availability; Product candidates not evaluated in a head-to-head study; comparisons based on public information*
KRAS mutations targeted: Elicio 7P: G12D, G12R, G12V, G12C, G12A, G12S, G13D, SLATE: G12C, G12D, G12V, Q61H

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Continuing Execution Momentum into 2024

2023 Accomplishments

- ✓ AMPLIFY-201 Completed Phase 1a enrollment
- ✓ AMPLIFY-7P Initiated Phase 1a study
- ✓ AMPLIFY-201 Presented Preliminary Safety, Immune and Biomarker Response data from Phase 1a study (ASCO)
- ✓ Received second GIRF grant to fund p53/ BRAF program
- ✓ AMPLIFY-201 Presented T Cell response and Relapse Free Survival data (AACR Special Conference Pancreas)
- ✓ Presented positive preclinical data for p53 / BRAF program (SITC)
- ✓ Presented AMPLIFY-201 Immune Response durability data (SITC)

2024 Anticipated Milestones

✓ AMPLIFY-201 Updated Clinical data	(1Q-2024)
❑ AMPLIFY-7P PDAC Phase 2 Initiation	(1Q-2024)
❑ AMPLIFY-7P Phase 1a data	(1H-2024)
❑ AMPLIFY-201 Clinical Immune Response follow up	(1H-2024)

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Targeting the Lymph Nodes to AMPlify Immunotherapy

January 2024