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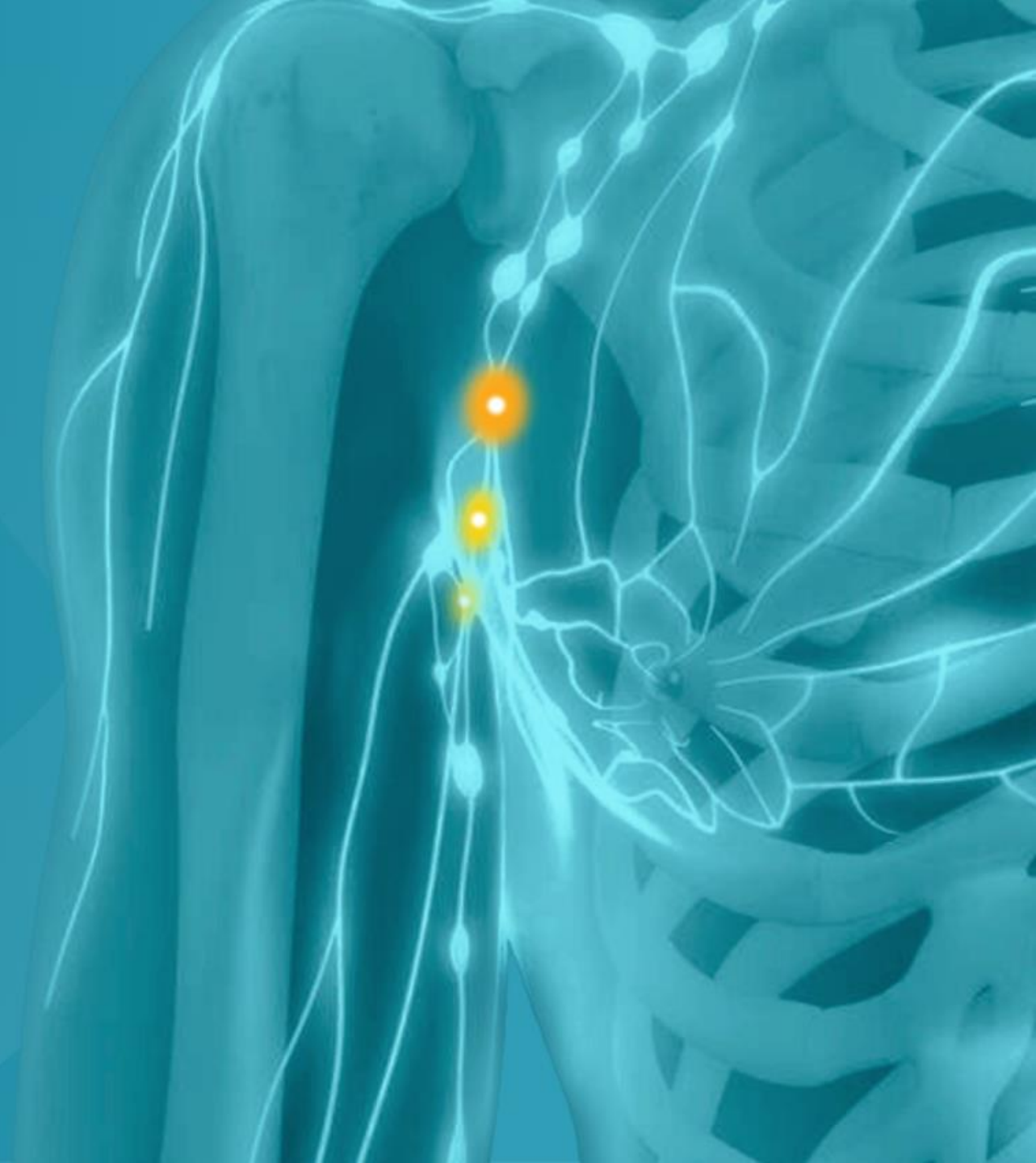
Targeting the Lymph Nodes to AMPlify Immunotherapy
Nasdaq: ELTX

June 2024



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The 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. Certain statements in the Presentation that are not purely historical are forward-looking statements. Such forward-looking statements include, among other things, statements regarding our planned clinical programs, including planned clinical trials and the potential of our product candidates, the unmet need and potential addressable market for our product candidates, the potential clinical utility, potential benefits and market acceptance of our product candidates, the potential advantages of our product candidates over those of existing therapeutics and/or those of our competitors, the expected receipt of clinical data, the timing of initiation of our planned clinical trials, and the advancement of and funding for our developmental programs generally. 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 “aim,” “anticipate,” “assume,” “believe,” “contemplate,” “continue,” “could,” “due,” “estimate,” “expect,” “goal,” “intend,” “may,” “objective,” “plan,” “predict,” “potential,” “positioned,” “seek,” “should,” “target,” “will,” “would,” and other similar expressions that are predictions of or indicate future events and future trends, or the negative of these terms or other comparable terminology 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 financial condition, including our ability to obtain the funding necessary to advance the development of ELI-002 and any other future product candidates, and our ability to continue as a going concern and our cash runway; our plans to develop and commercialize our product candidates, including ELI-002; the timing of initiation of our planned clinical trials, including the initiation of an ELI-002 Phase 1B Colorectal Cancer study; our ability to advance ELI-007 and/or ELI-008 into Phase 1 trials; the timing of the availability of data from our clinical trials, including data anticipated from our Phase 1A trials of ELI-002 2P and ELI-002 7P and the Phase 2 trial of ELI-002 7P; the timing for completion of enrollment in our Phase 2 trial of ELI-002 7P; 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; our ability to successfully collaborate with existing collaborators or enter into new collaborations, and to fulfill our obligations under any such collaboration agreements; the clinical utility, potential benefits and market acceptance of our product candidates; our commercialization, marketing and manufacturing capabilities and strategy; our ability to identify additional products or product candidates with significant commercial potential; our ability to advance ELI-002 outside of pancreatic ductal adenocarcinoma monotherapy and our pipeline programs; developments and projections relating to our competitors and our industry; the impact of government laws and regulations; our ability to protect our intellectual property position covering ELI-002 and other product candidates we may develop, including the extensions of existing patent terms where available; 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 29, 2024, as amended on April 29, 2024, under the heading “Risk Factors”, and any subsequent reports and other documents filed from time to time with the SEC. Forward-looking statements included in this release are based on information available to us as of the date of the Presentation. We do not undertake any obligation to update such forward-looking statements to reflect events or circumstances after the date of the Presentation, except to the extent required by law.

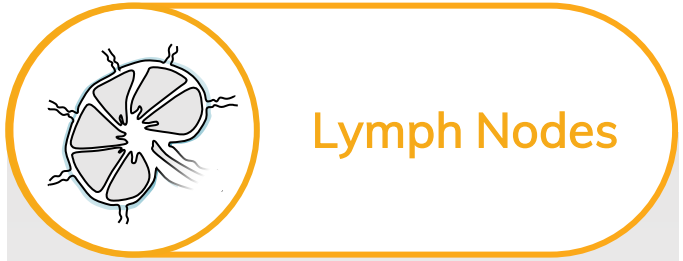
Company Overview

Clinical-stage biotech developing novel lymph node-targeted immunotherapies

Amphiphile ("AMP") Technology	• AMP delivers payloads to lymph nodes eliciting immune responses with increased magnitude, function, and durability • Designed to harness the lymph nodes' natural ability to educate, activate and amplify differentiated T cell responses, which are essential for recognizing and eliminating tumor cells • AMP technology offers broad immunotherapy applications; initially focused on cancer vaccine applications
ELI-002 Lymph Node Targeted mKRAS Cancer Vaccine	• Off-the-shelf cancer vaccine candidate targeting seven most common KRAS mutations that drive 25% of solid tumors • Adjuvant treatment for patients with high relapse risk mKRAS+ pancreatic (PDAC) and colorectal cancer (CRC) • Advantageous lymph node-targeted vaccine design: potent immunogenicity, durable CD4+ and CD8+ T cell responses, increased T cell cytotoxic function, and antigen spreading to target personalized tumor mutations
ELI-002 Clinical Data (39 pts across two Phase 1 a trials)	• Elicits mKRAS specific T cell response ~100x increase over baseline at the phase 2 dose without any DLTs or SAEs • T cell responses include generation of CD4+ helper and CD8+ killer cells, formation of a pool of memory cells, and ability to elicit T cell response to personal tumor neoantigens (antigen spreading) • mKRAS specific T cell response correlates with reductions in tumor biomarker and reduced risk of relapse or death
ELI-002 Next Steps	• '7P Phase 1a preliminary disease-free survival data 3Q 2024 • '2P Phase 1a updated recurrence-free survival and T cell response data 4Q 2024 • '7P Phase 2 trial enrollment anticipated to be completed (135 patients) in 4Q 2024 • '7P Phase 2 trial disease-free survival interim analysis expected in 1Q 2025

Engaging the Lymph Node

A critical component to activating the immune response in the fight against cancer



Lymph Nodes

- The lymph nodes, known as the "command center" of the immune response play a vital role in detecting threats to our health and coordinating the buildup of protective immune responses
- Lymph nodes collect and filter lymphatic fluid for signs of danger, including traces of cancer or infectious pathogens

T Cell Response

- Proper education, multiplication, and activation in the lymph nodes is critical for preparing disease-fighting immune cells to resolve disease
- Numerous key immune cells, including antigen-presenting cells, and T cells are organized in the lymph nodes – the primary site where the immune response is generated to fight cancers

Amphiphile (AMP) Technology

- Elicio is harnessing the natural power of the immune system with AMP technology, to deliver therapeutic payloads directly to the lymph nodes, enhancing the immune system's cancer-fighting capabilities.
- Our targeted-delivery approach generates a robust immune response that amplifies cancer fighting T cells, for the treatment of cancers

Previous cancer vaccine efforts have not generated a sufficient T cell response to be an effective immunotherapy.
We believe accessing the lymph nodes is critical to generate the robust immune response for effective immuno-oncology.

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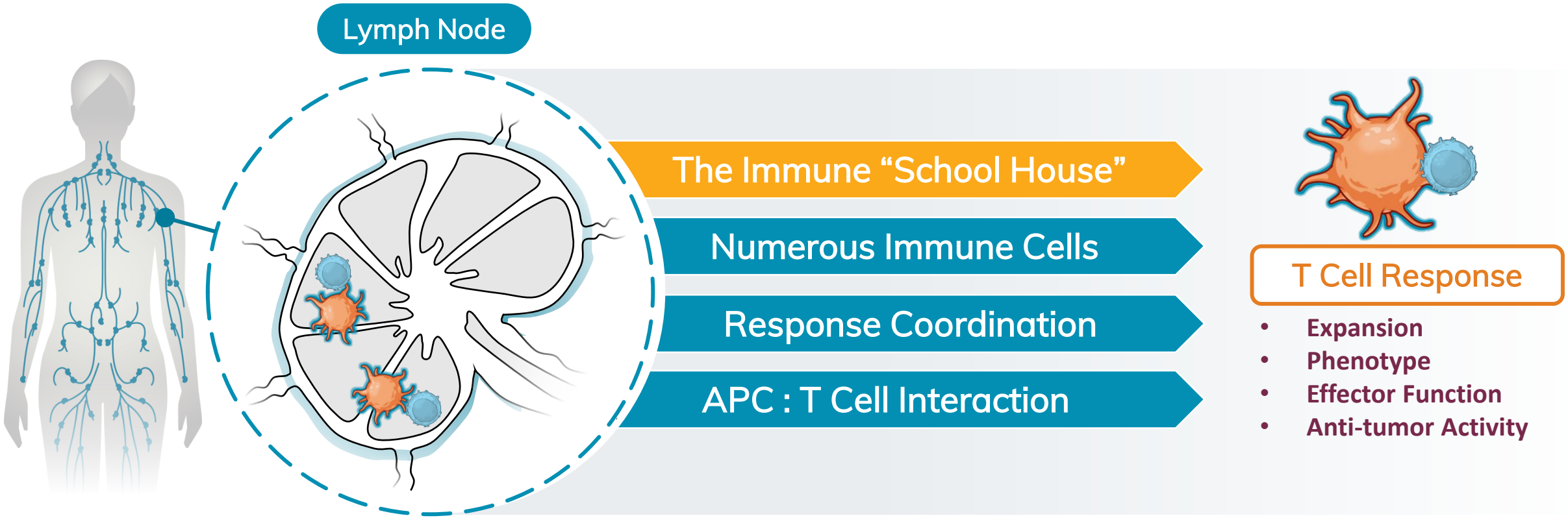
Lymph Nodes: Eliciting a Robust T Cell Response

Immune biology in the Lymph Nodes directs the development of anti-tumor immunity

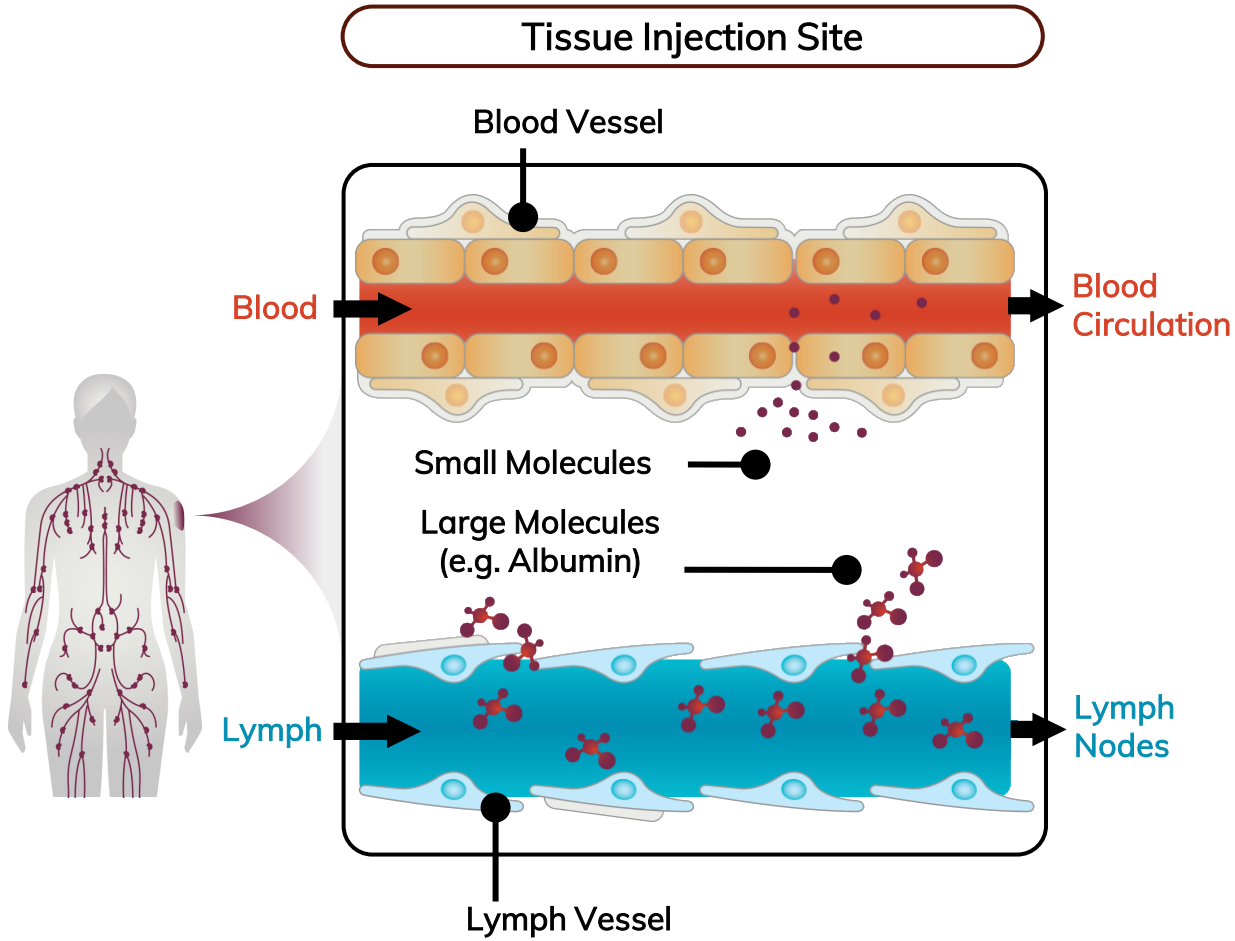


The Amphiphile (AMP) Technology

Harnessing the untapped potential of the lymph nodes for immunotherapy

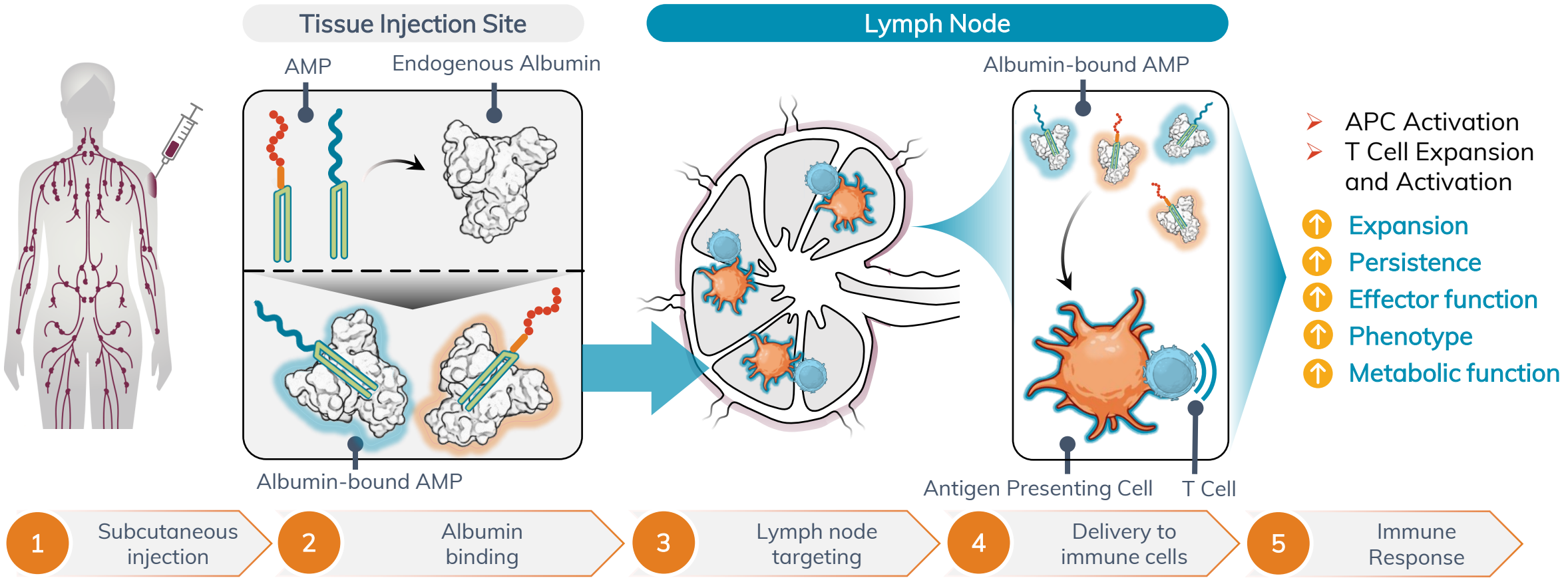
Targeting Immunity in the Lymph Nodes

- 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**



MOA: AMP Locates to Lymph Node Orchestrating Immunity

AMP technology capable of delivering payloads to lymph nodes generating robust immune response

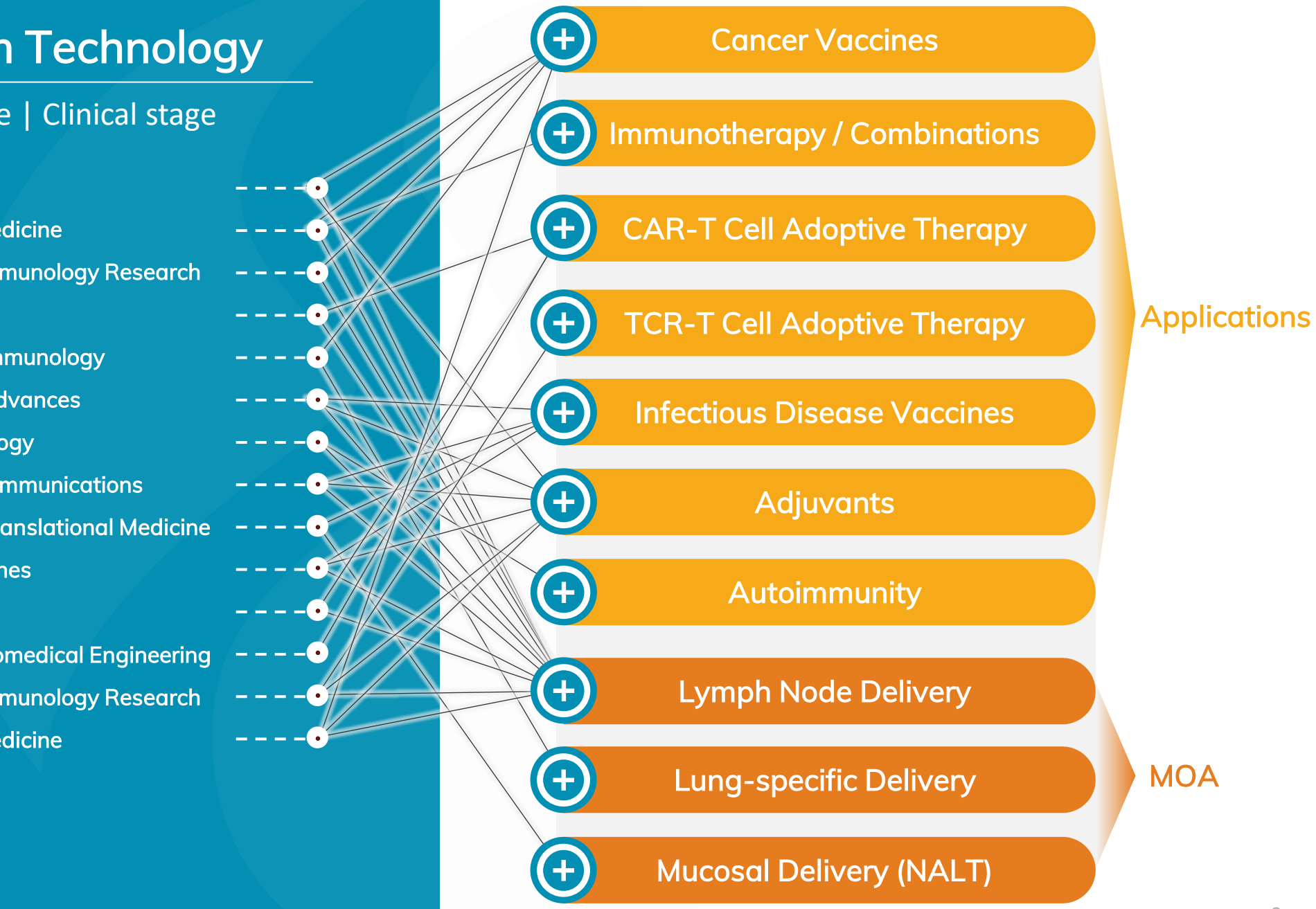


Pant, et al. **Nature** Medicine 2024; Liu, et al. **Nature** 2014; Moynihan, et al. **Nature Medicine** 2016; Ma, et al. **Science** 2019

AMP Platform Technology

Versatile | Simple | Clinical stage

2014	Liu	Nature
2016	Moynihan	Nature Medicine
2018	Moynihan	Cancer Immunology Research
2019	Ma	Science
2021	Rakhra	Science Immunology
2021	Steinbuck	Science Advances
2021	Li	J Immunology
2022	Dasari	Nature Communications
2022	Hartwell	Science Translational Medicine
2022	Seenappa	NPJ Vaccines
2023	Ma	Cell
2023	Zhang	Nature Biomedical Engineering
2024	Drakes	Cancer Immunology Research
2024	Pant	Nature Medicine



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AMP Technology has the potential to address several mutations

Preclinical programs funded by Gastro-Intestinal (GI) Research Foundation

In preclinical models, ELI-007 and ELI-008 have shown strong induction of tumor antigen-specific T cell responses

Preclinical Data presented at AACR 2024. Posters can be found here.

ELI-007 (Mutant BRAF-Peptide Vaccine)

- ELI-007 is an investigational multi-valent lymph node–targeted AMP peptide vaccine comprised of the V600E and V600K mutant antigens, developed to target BRAF gene mutations.
- In preclinical models, AMP-immunization generated robust *in vivo* immune responses yielding strong T cell activation against mBRAF epitopes.
- Lymph node-targeted AMP-vaccination resulted in a 19-fold increase in immune response after only 3 doses and a 42-fold increase after 5 doses when compared to conventional comparator vaccines.

BRAF-mutations are highly prevalent in melanoma, colorectal and thyroid cancer existing in 8% of solid tumors representing up to 1.5 million cancer incidences worldwide each year

ELI-008 (p53 Hotspot Mutant-Peptide Vaccine)

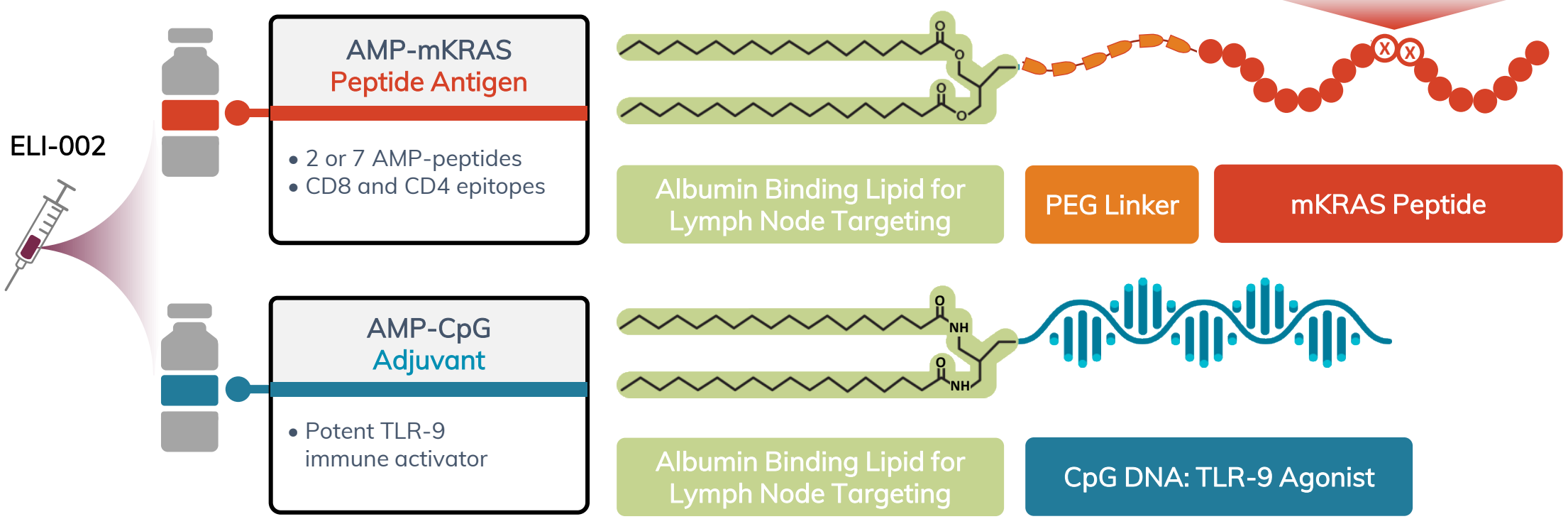
- ELI-008 is an investigational multi-valent lymph node–targeted AMP peptide vaccine comprised of the several p53 mutant antigens, developed to target hotspot p53 gene mutations.
- In preclinical models, AMP-immunization generated robust immune responses yielding strong T cell activation against common p53 hot spot mutations (R248W, R248Q, R175H, R273H, R273C, R282W, G245S, Y220C, C135Y, R158H, H214R).
- For multiple p53 mutations, ELI-008 demonstrated a several-hundred-fold increase over conventional comparator vaccines.

Similar to KRAS, p53 mutations are present in a broad spectrum of cancer types accounting for >30% of solid tumors

ELI-002 Composition

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

	ELI-002	G12X	G13X
Peptide Composition	2P	D R	
	7P	D R V C S A	D

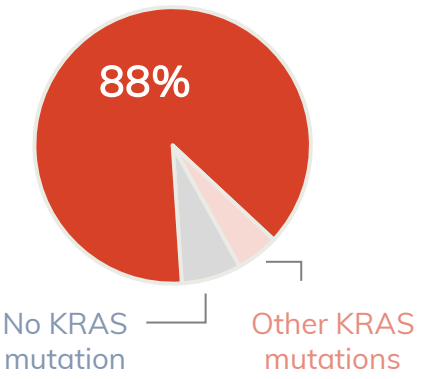


The mKRAS Opportunity

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

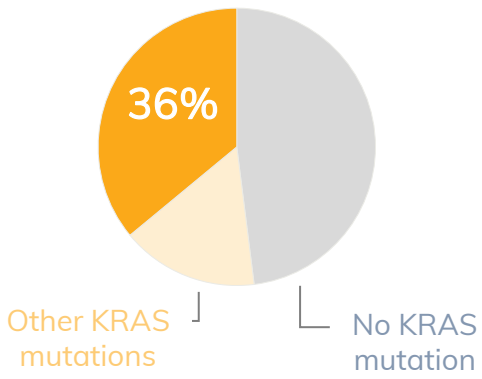
Pancreatic Ductal Adenocarcinoma

ELI-002 addressable
Incidence: ~128k



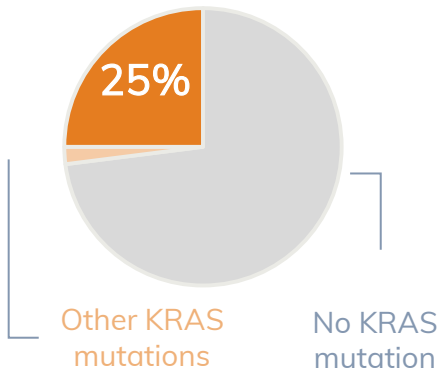
Colorectal Cancer

ELI-002 addressable
Incidence: ~192k

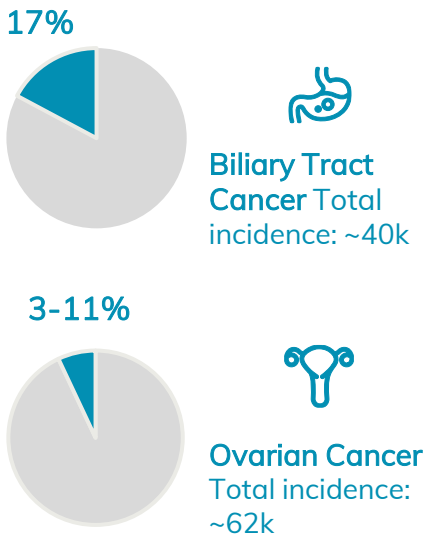


Non-Small Cell Lung Cancer

ELI-002 addressable
Incidence: ~128k



Other mKRAS Opportunities



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 2P: 2-Peptide (2P) Formulation

Phase 1A: Adjuvant Dose Ranging 0.1mg to 10mg doses

First-in-human Study: mKRAS G12D or G12R-expressing, Adjuvant treatment of MRD+ PDAC and CRC

AMPLIFY-201 Study Overview

✻ Published in Nature Medicine | ELI-002 2P | Phase 1A

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

ELI-002 MONOTHERAPY: NCT04853017

Monotherapy (no chemo, CPI combo)

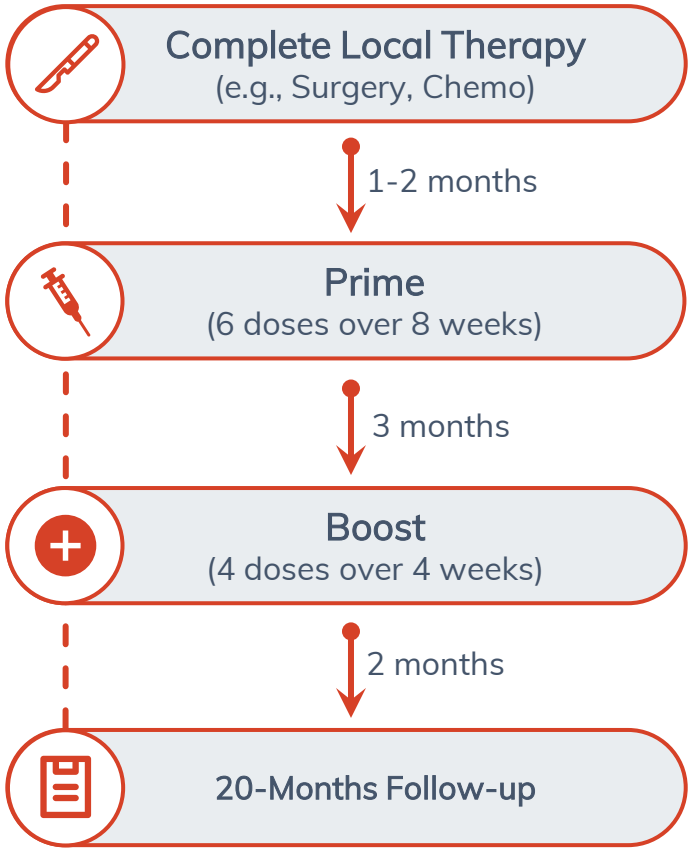
Key Criteria

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

Baseline Characteristics

25 patients enrolled across 5 dose cohorts, 25 evaluable at database cutoff (9/6/2023)

- **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)

✻ **Preliminary Phase 1 (AMPLIFY-201) study of ELI-002 2P published in Nature Medicine January 2024**

Pant, et al. Lymph-node-targeted, mKRAS-specific amphiphile vaccine in pancreatic and colorectal cancer: the phase 1 AMPLIFY-201 trial. Nature Medicine. 2024. https://doi.org/10.1038/s41591-023-02760-3

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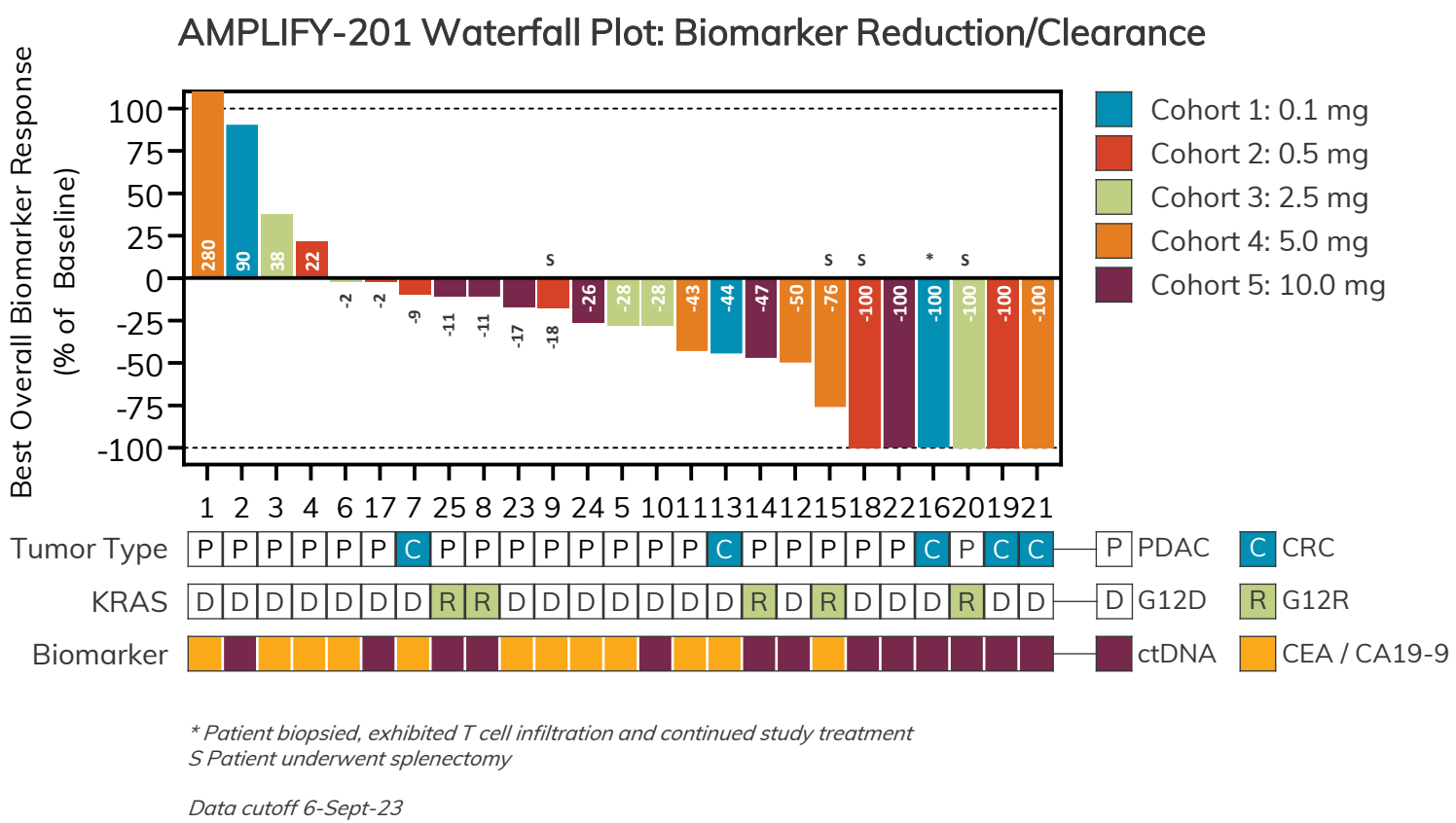
AMPLIFY-201: Tumor Biomarker Response

ELI-002 2P | Phase 1A

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

Tumor Biomarker Responses

- Waterfall displays best response of ctDNA or serum tumor biomarker
- Most patients (84%, 21/25) showed decline from baseline in ctDNA or CEA/CA19-9 levels
- 24% of patients (6/25) showed complete clearance of ctDNA
- Responses observed in PDAC and CRC, mKRAS G12D and G12R
- Responses observed despite prior splenectomy (S annotated)



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

Data cutoff 6-Sept-23

Pant, O'Reilly, et al. Nature Medicine. 2024

AMPLIFY-201: Immune Response

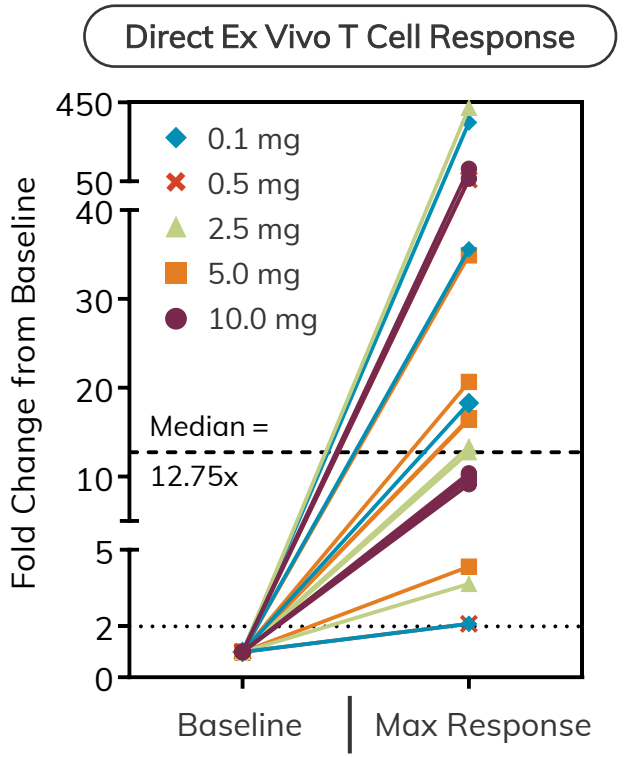
ELI-002 2P | Phase 1A

84% of patients generated mKRAS-specific T cells directly *ex vivo*; 100% at RP2D

mKRAS T Cell Responses

- T cells detectable by standard direct *ex vivo* FluoroSpot and flow cytometry, with no expansion required
- 84% of patients showed T cell responses; 100% at the RP2D (10 mg)
- 58x average fold-change in T cell numbers from baseline (median 12.75; range 2-423x)
- 59% of patient responses included both CD4 and CD8 T cells
- *De novo* T cell priming and memory cell expansion
- Responses were observed across diverse HLA backgrounds

AMPLIFY-201 T Cell Fold-Changes



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

Data cutoff 6-Sept-23

Pant, O'Reilly, et al. Nature Medicine. 2024

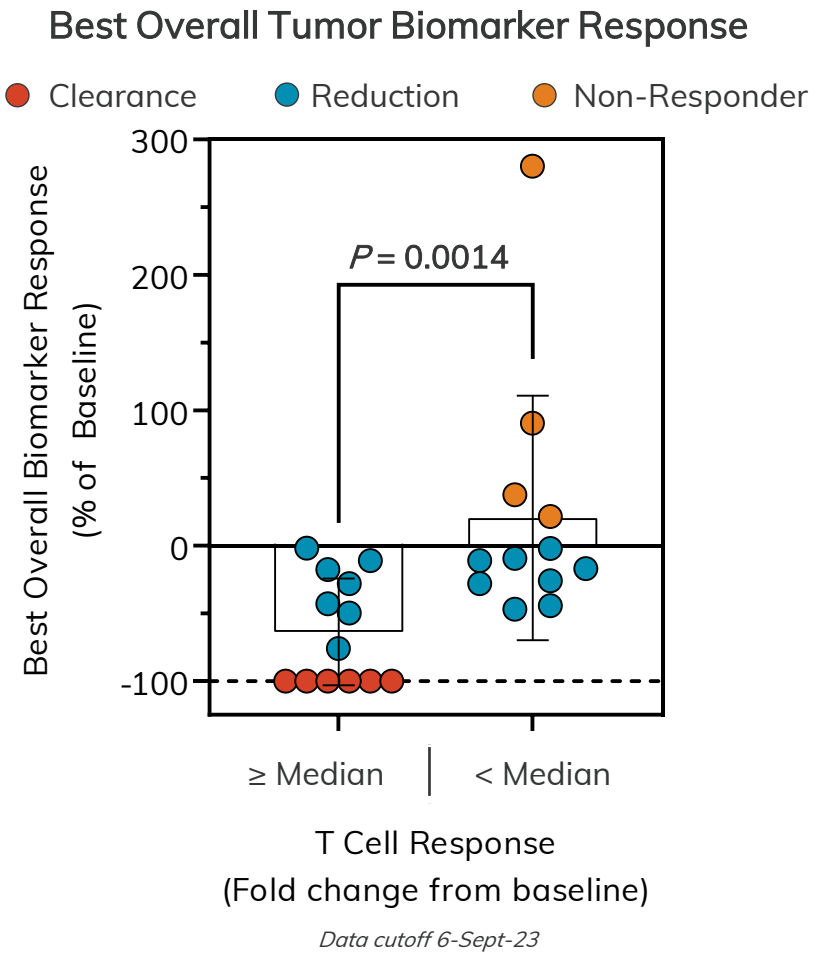
AMPLIFY-201: T Cell Fold-change Predicts Tumor Biomarker Response

ELI-002 2P | Phase 1A

All patients with T cell responses over the 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

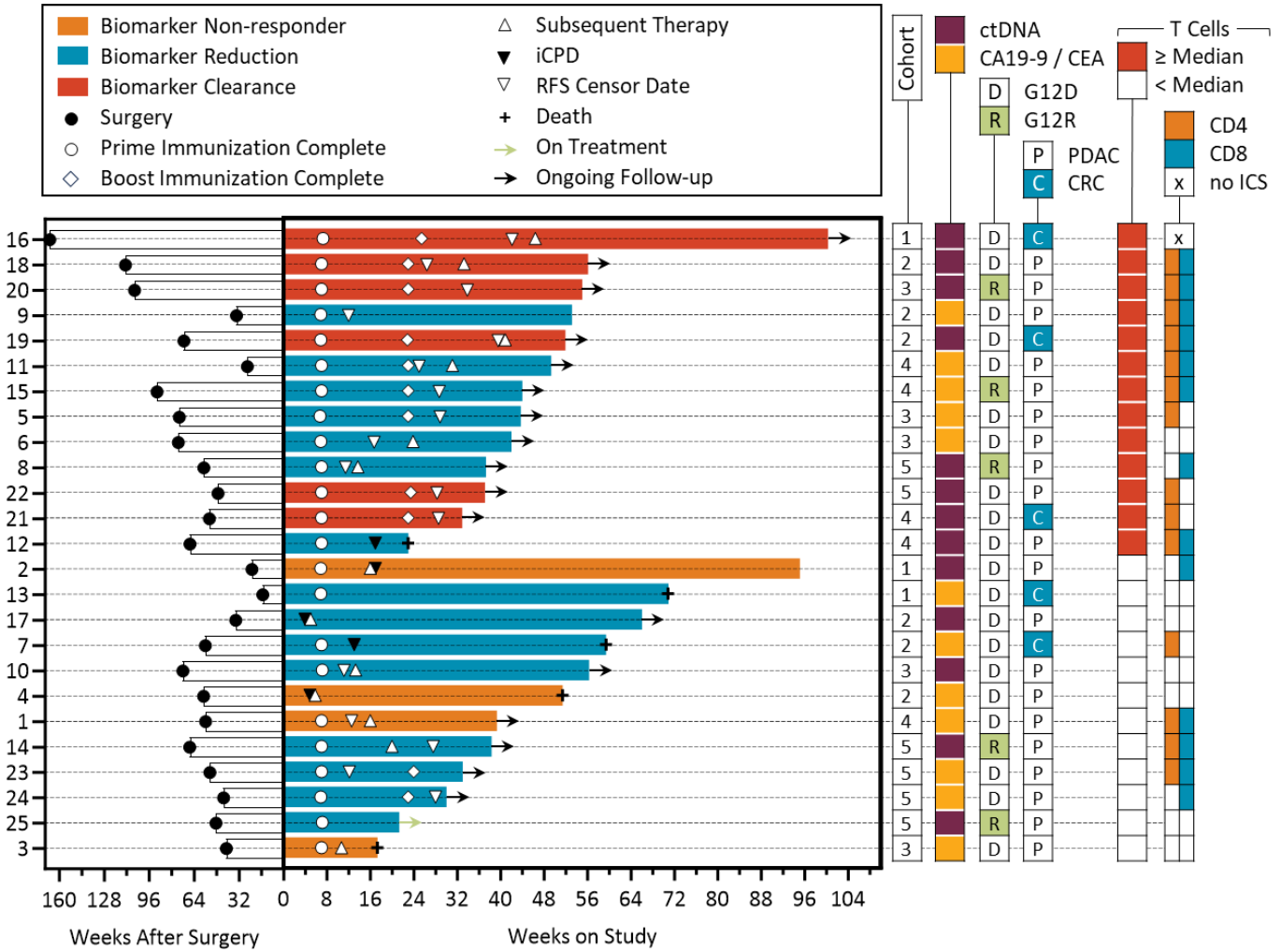
AMPLIFY-201: Majority of Above Median T Cell Responders Include CD4 and CD8 T cells

ELI-002 2P | Phase 1A

Above median mKRAS-specific T cell response correlated 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

ELI-002 2P | Phase 1A

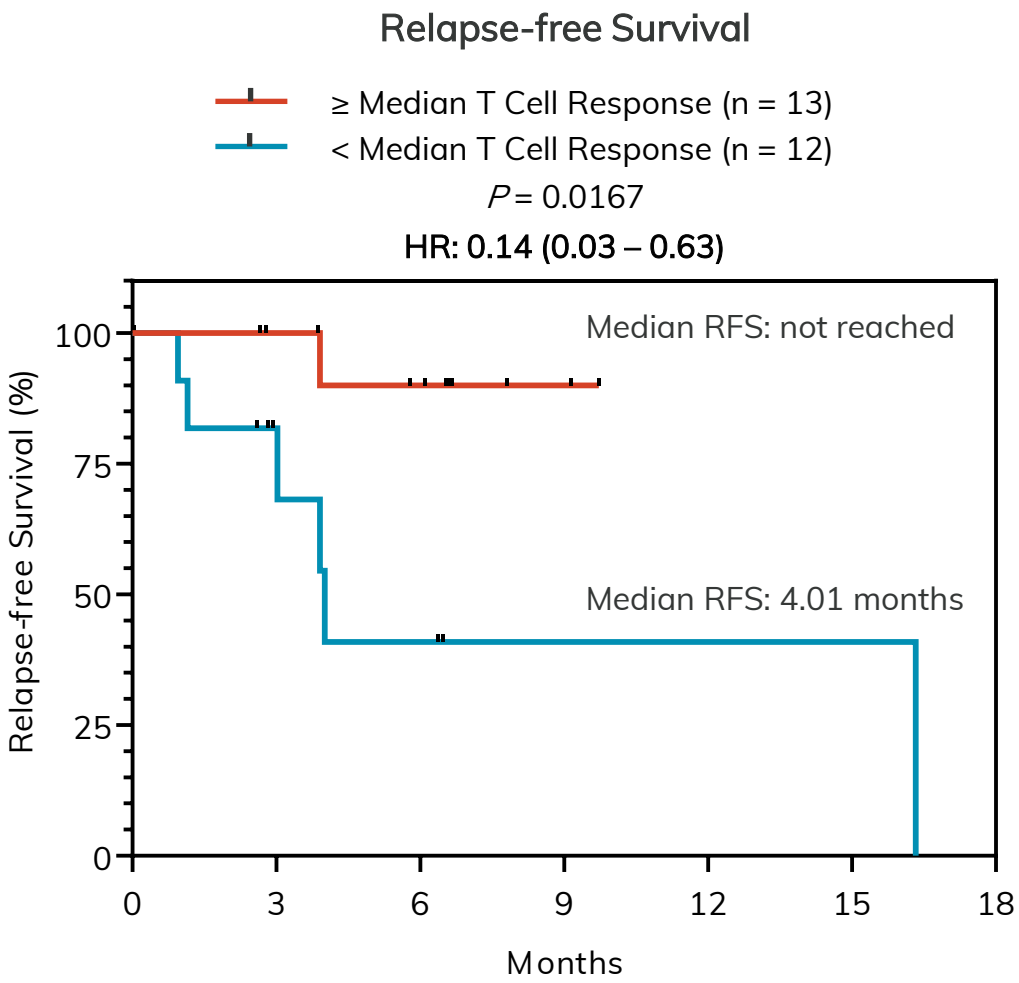
86% reduction in the risk of progression or death in above median T cell responders

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 RFS was 16.3 months

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

Database cutoff 6-Sept-23



Pant, O'Reilly, et al. Nature Medicine. 2024

AMPLIFY-201: ELI-002 Amplifies Cytotoxic mKRAS-specific CD4+ T cells

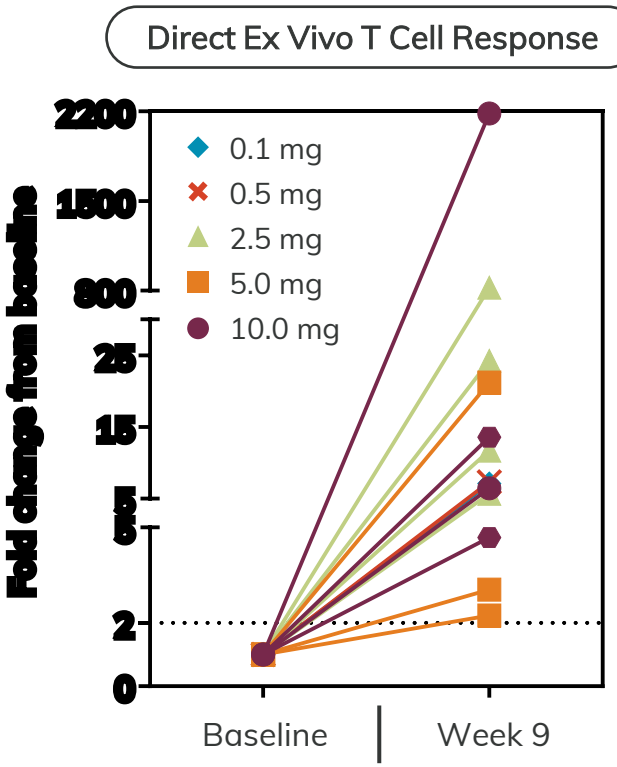
ELI-002 2P | Phase 1A

Induced mKRAS-specific CD4+ T cells develop enhanced cytotoxic function, memory phenotype

mKRAS Cytotoxic CD4+ T Cell Response

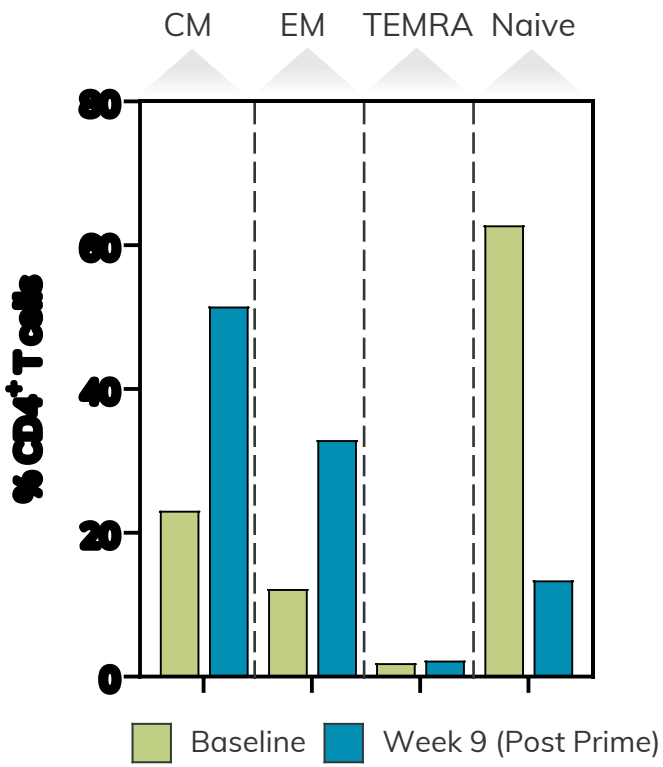
- T cells detectable by standard direct *ex vivo* FluoroSpot and flow cytometry, with no expansion required
- 68% of evaluated patients (13/19) developed increased cytotoxic CD4+ T cell responses targeting mKRAS
- ELI-002 vaccination induces development of central and effector memory phenotype

Cytotoxic CD4+ T Cell Fold-Changes



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

CD4+ T cell Memory



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AMPLIFY-201: ELI-002 Amplifies Cytotoxic mKRAS-specific CD8+ T cells

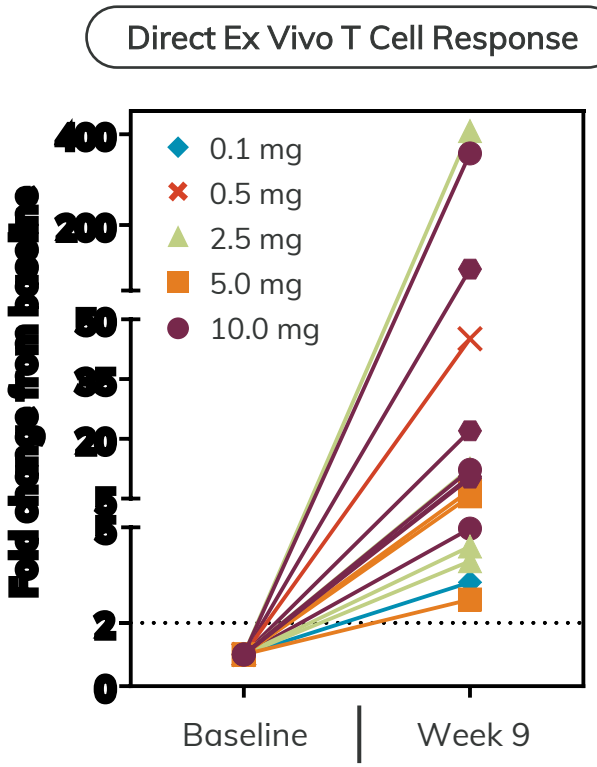
ELI-002 2P | Phase 1A

Induced mKRAS-specific CD8+ T cells develop enhanced cytotoxic function, memory phenotype

mKRAS Cytotoxic CD8+ T Cell Response

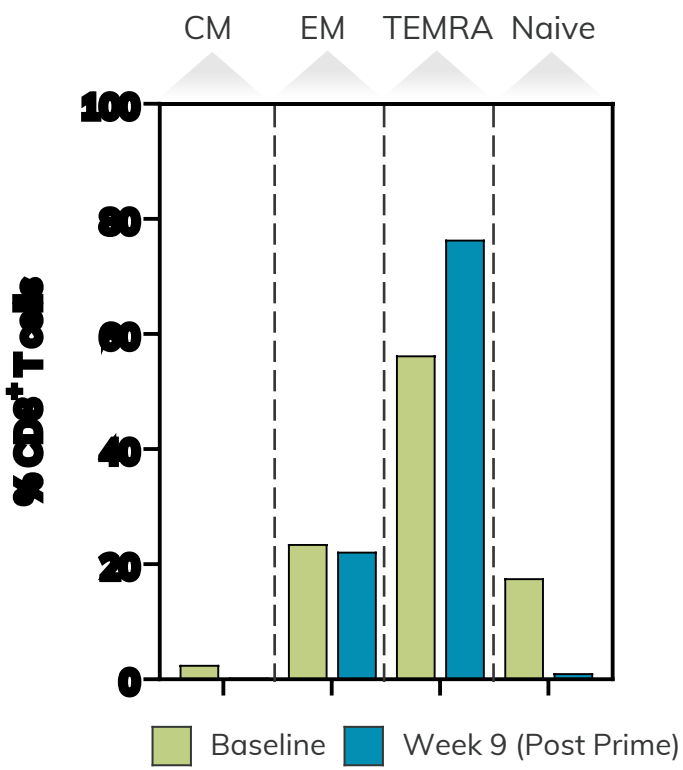
- T cells detectable by standard direct *ex vivo* FluoroSpot and flow cytometry, with no expansion required
- 84% of evaluated patients (16/19) developed increased cytotoxic CD8+ T cell responses targeting mKRAS
- ELI-002 vaccination induces development of TEMRA phenotype

Cytotoxic CD8+ T Cell Fold-Changes



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

CD8+ T cell Memory



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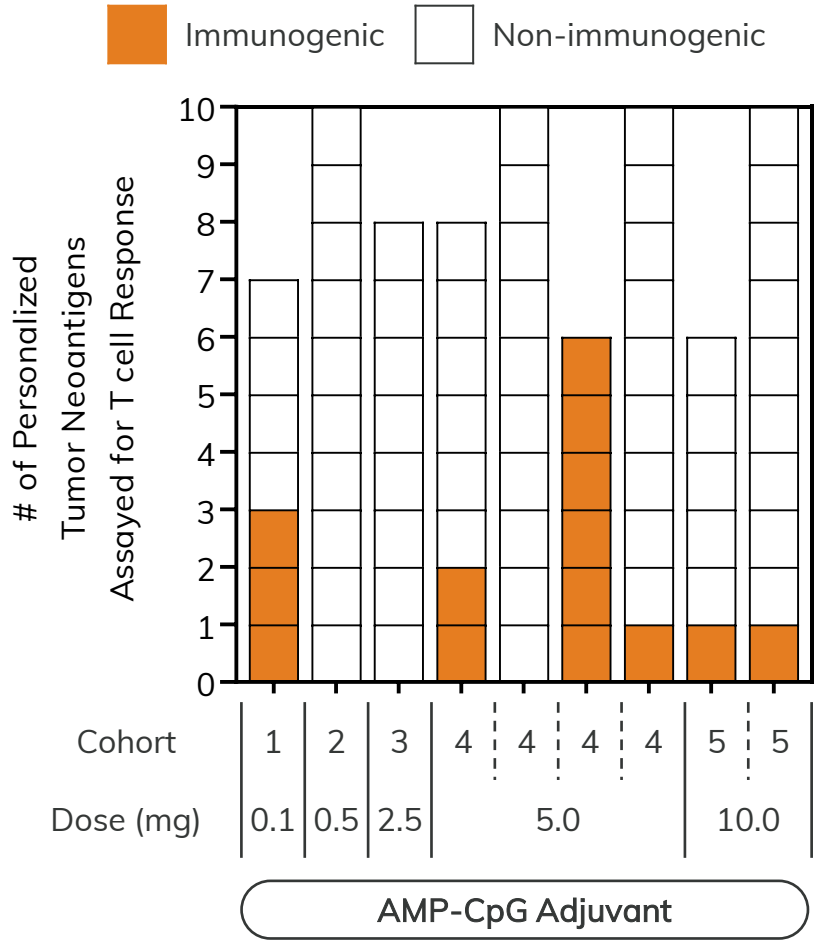
AMPLIFY-201: ELI-002 Stimulates Antigen Spreading

ELI-002 2P | Phase 1A

Expansion of T cells specific to personalized tumor antigens not targeted by vaccination



Antigen Spreading to Personal Tumor Neoantigens

- ELI-002 2P vaccination led to expansion of T cell responses targeting passenger mutations alongside mKRAS driver mutations in a majority of evaluable patients
- T cells detectable by standard direct *ex vivo* FluoroSpot and flow cytometry, with no expansion required
- 67% of evaluated patients (6/9) developed increased T cell responses targeting personalized tumor neoantigens
  - 100% at RP2D 10.0 mg CpG adjuvant dose
- Polyfunctional CD4 and CD8 T cells

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AMPLIFY-201: Safety & Tolerability

ELI-002 2P | Phase 1A

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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ELI-002 7P: 7-Peptide (7P) Formulation

Phase 1A: Peptide Dose Ranging 1.4mg or 4.9mg doses

First-in-human Study: mKRAS G12x or G13D-expressing, Adjuvant treatment of MRD+ PDAC and CRC

AMPLIFY-7P Phase 1A Study Overview

ELI-002 7P | Phase 1A

Phase 1 peptide dose-ranging study to assess safety and efficacy of ELI-002 7P in patients who completed standard therapy and have minimal residual disease

ELI-002 MONOTHERAPY: NCT05726864

Monotherapy (no chemo, CPI combo)

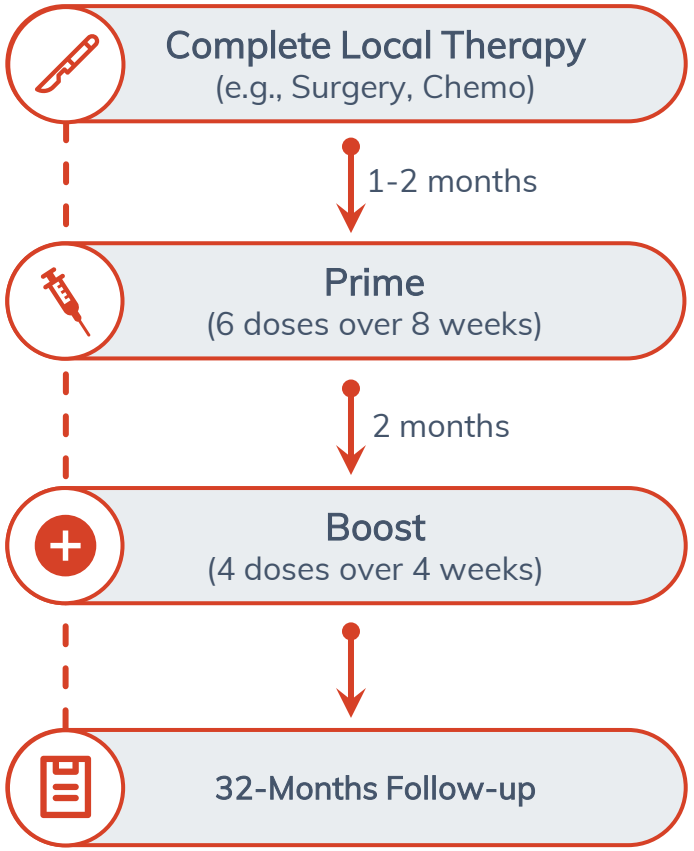
Key Criteria

- ✓ **Includes: mKRAS G12D/R/V/C/A/S/G13D**
- ✓ No radiographic evidence of disease (NED)
- ✓ High risk of relapse (MRD+ ctDNA/serum biomarkers)

Baseline Characteristics

14 patients enrolled across 2 dose cohorts, 12 biomarker evaluable at database cutoff (Dec 18, 2023)

- **Advanced:** 7 (50%) had stage III
- **Pre-treated:** All received prior chemo and surgery, 29% had prior radiation



Basket Trial Enrollment

Pancreatic Ductal Adenocarcinoma (PDAC)	n=13
Colorectal Cancer (CRC)	n=1

Endpoints

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

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AMPLIFY-7P: T Cell Responses

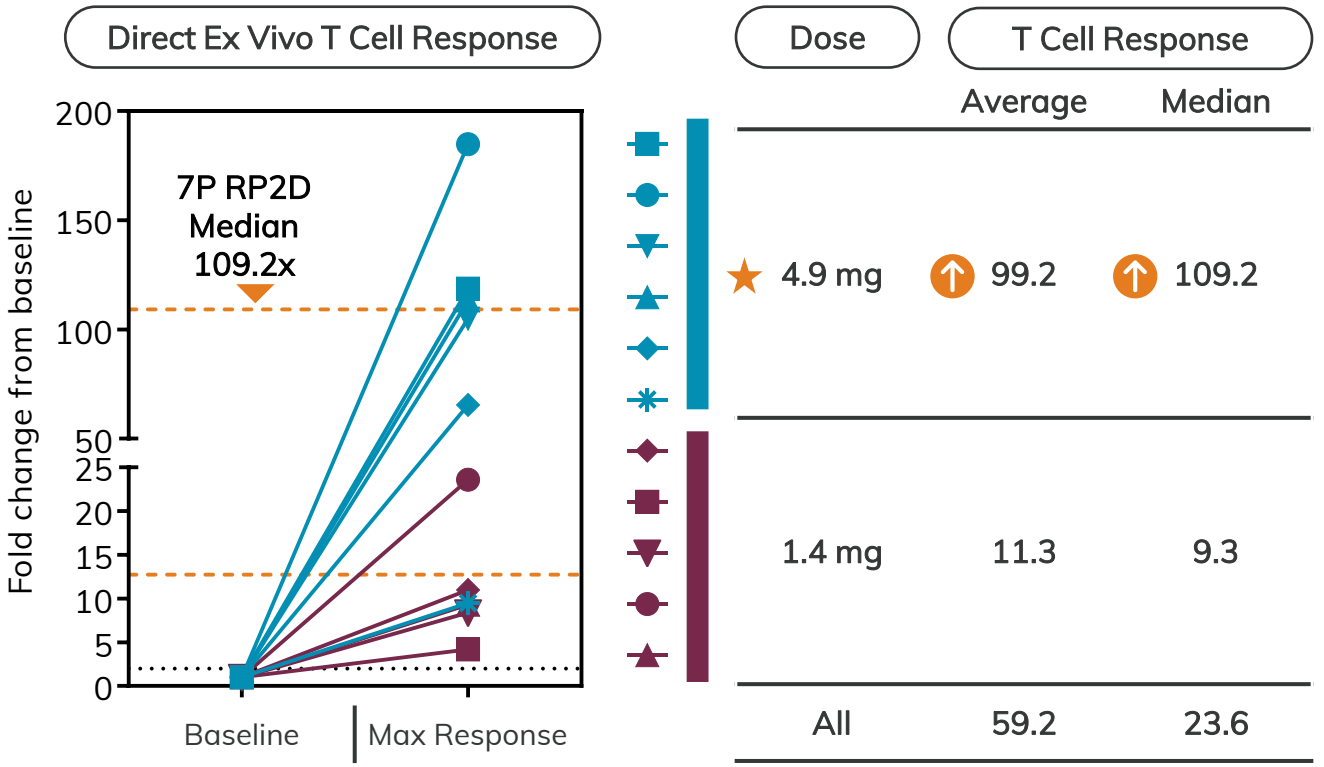
ELI-002 7P | Phase 1A

100% of patients with robust T cell response

mKRAS T Cell Responses

- **100% of patients showed T cell responses**
- 4.9 mg dose group selected for Phase 2
  - Median fold change = **109.2x**
  - 66.7% with CD4 and CD8 T cells
- 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



Dose	T Cell Response Average	T Cell Response Median
★ 4.9 mg	↑ 99.2	↑ 109.2
1.4 mg	11.3	9.3
All	59.2	23.6

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

ELI-002 7P: Data cutoff 18-Dec-23

★ = Phase 2 Dose

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AMPLIFY-7P: T Cell Responses

ELI-002 7P | Phase 1A

Phase 2 Dose generates higher immune response than seen with ELI-002 2P

ELI-002 2P vs ELI-002 7P 4.9 mg

- ELI-002 7P data based on n=11 Patients (1.4 mg, n=5; 4.9 mg, n=6)
- 100% T cell Response Rate (n=11)
- ELI-002 7P 4.9 mg shows increased:
  - ↑ Median Fold Change
  - ↑ CD4 + CD8 Response Rate
  - ↑ Response Rate for all 7 mKRAS Antigens
  - ↑ Response Rate to Patient Tumor Antigen

	ELI-002 2P (Nat Med)	ELI-002 7P (All)	ELI-002 7P (4.9 mg) ★
Response Rate	84%	100%	**100%**
Median Fold Change	12.8	23.6	↑ **109.2**
% CD4 + CD8 T cells	59%	63.6%	↑ **66.7%**
Response to 7 mKRAS Antigens	52.4%	45.5%	↑ **66.7%**
Response to Tumor Antigen	81%	72.7%	↑ **83.3%**
			Phase 2 Dose

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

ELI-002 2P: Data cutoff 6-Sept-23
ELI-002 7P: Data cutoff 18-Dec-23

★ = Phase 2 Dose

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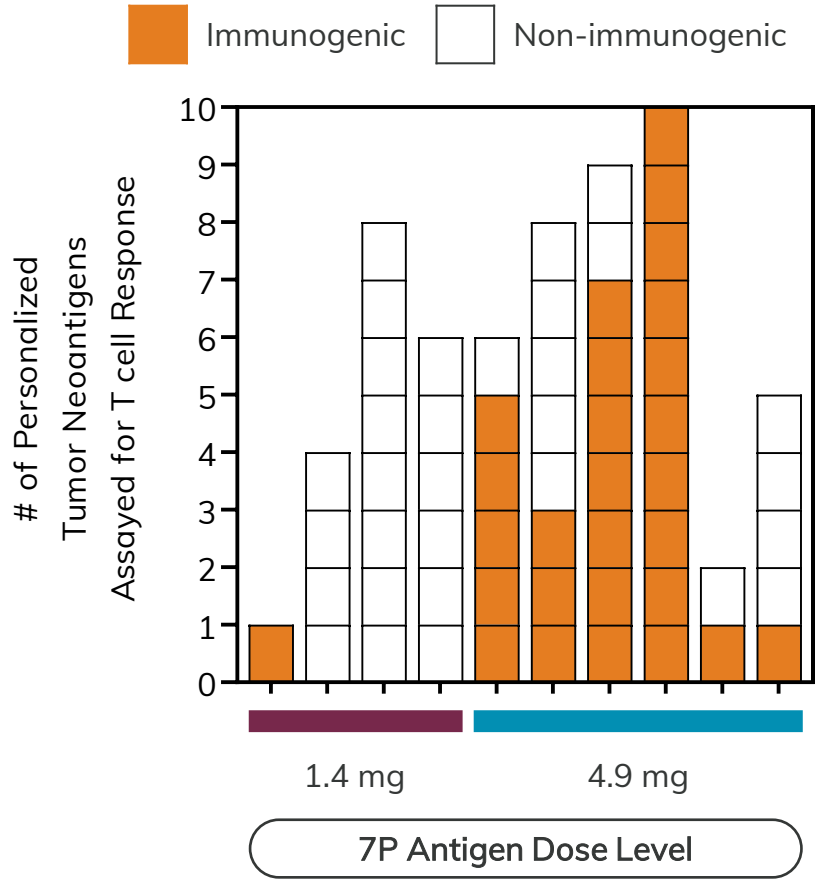
AMPLIFY-7P: ELI-002 Stimulates Antigen Spreading

ELI-002 7P | Phase 1A

Expansion of T cells specific to personalized tumor antigens not targeted by vaccination



Antigen Spreading to Personal Tumor Neoantigens

- ELI-002 7P vaccination led to expansion of T cell responses targeting passenger mutations alongside mKRAS driver mutations in a majority of evaluable patients
- T cells detectable by standard direct *ex vivo* FluoroSpot and flow cytometry, with no expansion required
- 70% of evaluated patients (7/10) developed increased T cell responses targeting personalized tumor neoantigens
  - 100% at RP2D 4.9 mg peptide antigen dose
- Polyfunctional CD4 and CD8 T cells

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AMPLIFY-7P: Tumor Biomarker Responses

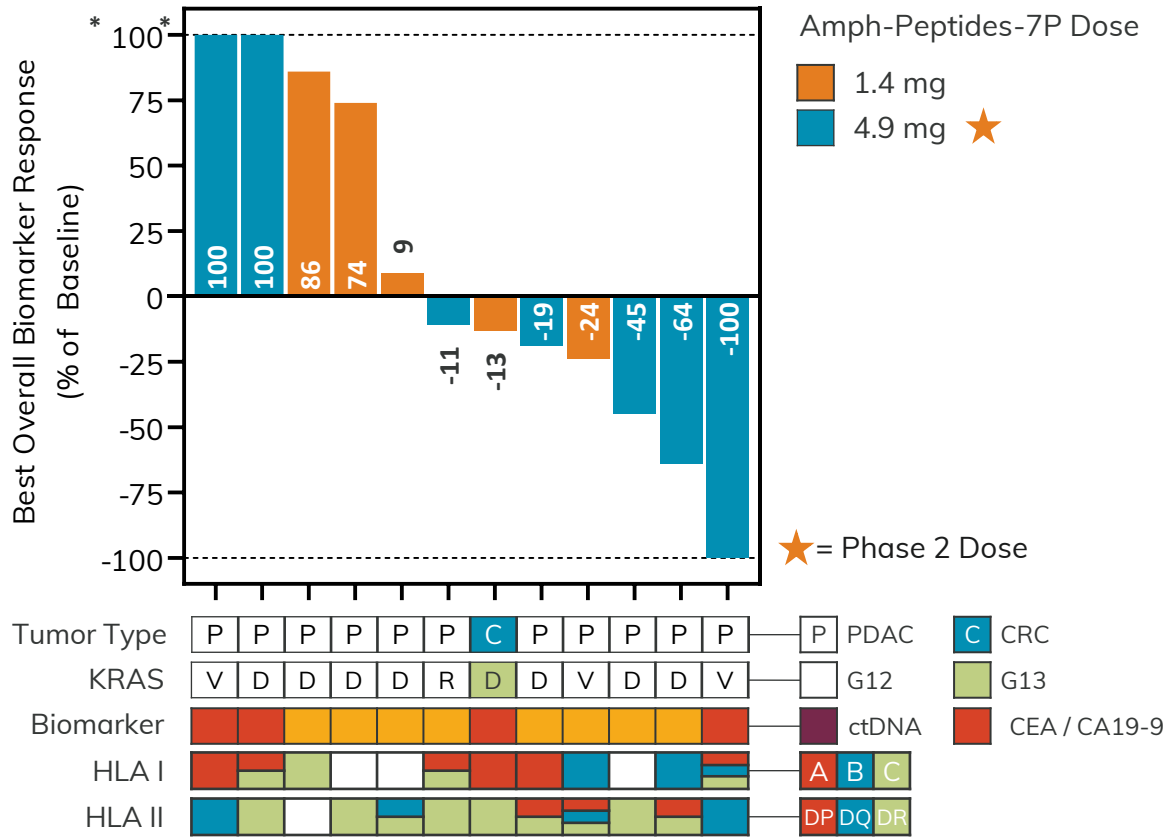
ELI-002 7P | Phase 1A

Waterfall reflects superiority of 4.9 mg AMP-Peptide 7P dose level

Tumor Biomarker Responses

- 71% (5/7) of patients in the 4.9 mg dose had biomarker decline
- 40% (2/5) of patients in the 1.4 mg dose had biomarker decline
- 14% (1/7) PDAC patients at 4.9 mg dose had complete clearance
- Response may deepen over time (some patients not yet finished boosters)

AMPLIFY-201 Waterfall Plot: Biomarker Reduction / Clearance



Data cutoff 18-Dec-23

** Represents percent change > 100%; data display at maximum 100%*

Two (2) pts not included in this analysis. Pt 111-002 had insufficient post-baseline biomarker data; pt 107-002 d/c treatment early due to non-treatment related AE

KRAS variant post-analysis: 107001 G12D, 106001 G12V, 110004 G12D, 117001 G12D

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AMPLIFY-7P: T Cell Response Drives Tumor Biomarker Response

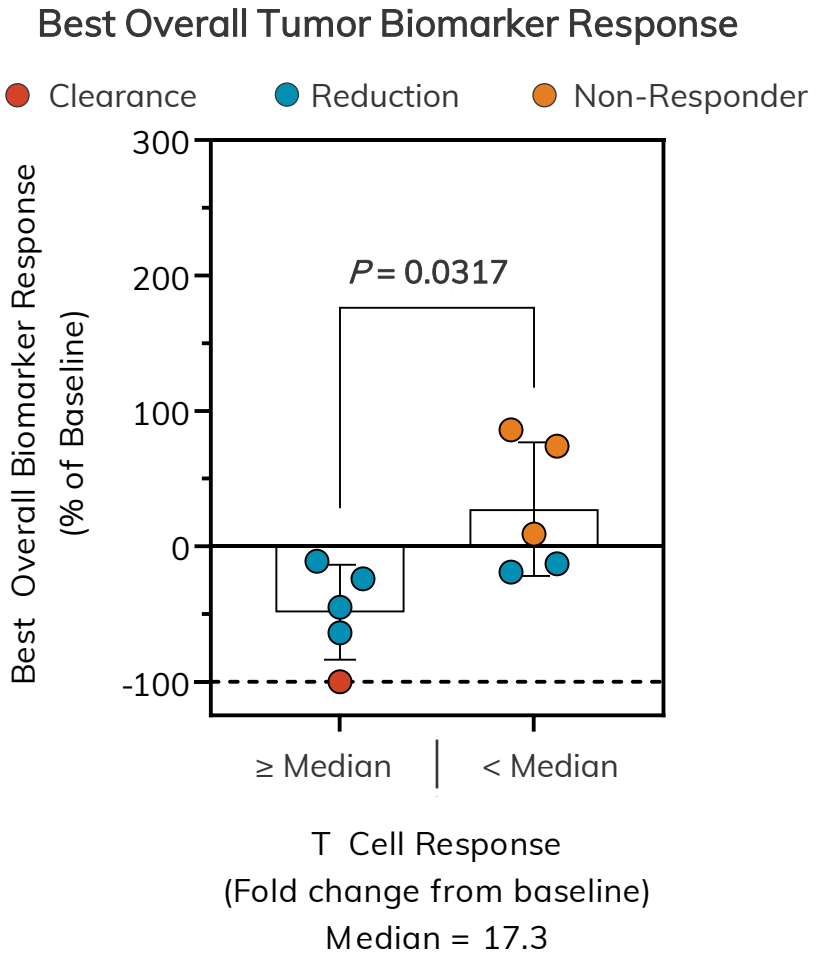
ELI-002 7P | Phase 1A

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

mKRAS T Cell Response ➤ Tumor Biomarker Response

- **Strength of T cell response to ELI-002 is correlated to tumor biomarker response**
- 100% (5/5) of the above median T cell group respond to ELI-002; in the below median group 40% (2/5) respond to ELI-002*
- 80% (4/5) of the 4.9 mg dose cohort are in the above median T cell group, including a complete responder
- Statistically significant, p-value per Mann Whitney Test (*P* = 0.0317)

**10 patients had both immunogenicity and biomarker data available at data cutoff.*



Data cutoff 18-Dec-23

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AMPLIFY-7P: Safety

ELI-002 7P | Phase 1A

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

ELI-002 7P Safety / Tolerability

- No DLT observed, No CRS or T cell Toxicities
- Most common TRAE (>20%) were Fatigue (28.6%; all Gr1) and Malaise (21.4%; all Gr1)
- One (1) pt had SAE (107-002) 1.4 mg dose non-treatment related intestinal obstruction resulted in hospitalization and w/d from treatment
- No dose modification
- No TRAE leading to death

AMP-Peptide 7P Dose	1.4 mg n=6	4.9 mg n=8	Overall n=14
Adverse Event Term [a]			
Patients with Any Related TEAE, n (%)	5 (83.3)	6 (75.0)	11 (78.6)
Fatigue	3 (50.0)	3 (37.5)	6 (42.9)
Malaise	1 (16.7)	2 (25.0)	3 (21.4)
Diarrhea	1 (16.7)	2 (25.0)	3 (21.4)
Abdominal Distension	2 (33.3)	0	2 (14.3)
Abdominal Pain	1 (16.7)	1 (12.5)	2 (14.3)
Patient Summary			
KRAS Mutation	DDDDV 13D	DDDDRVVV	
Dose Limiting Toxicity	0	0	0
Biomarker Reduction / Clearance	2 / 5 (40)	5 / 7 (71)	7 / 12 (58) [b]
T cell Response	6 / 6 (100)	5 / 5 (100)	11 / 11 (100) [c]

TEAE: Treatment Emergent Adverse Event
[a] Preferred terms per the Medical Dictionary for Regulatory Activities, version 25.0
[b] Measured among 12 evaluable patients as of the data cut off: December 18, 2023
[c] Measured among 11 evaluable patients as of the data cut off: December 18, 2023

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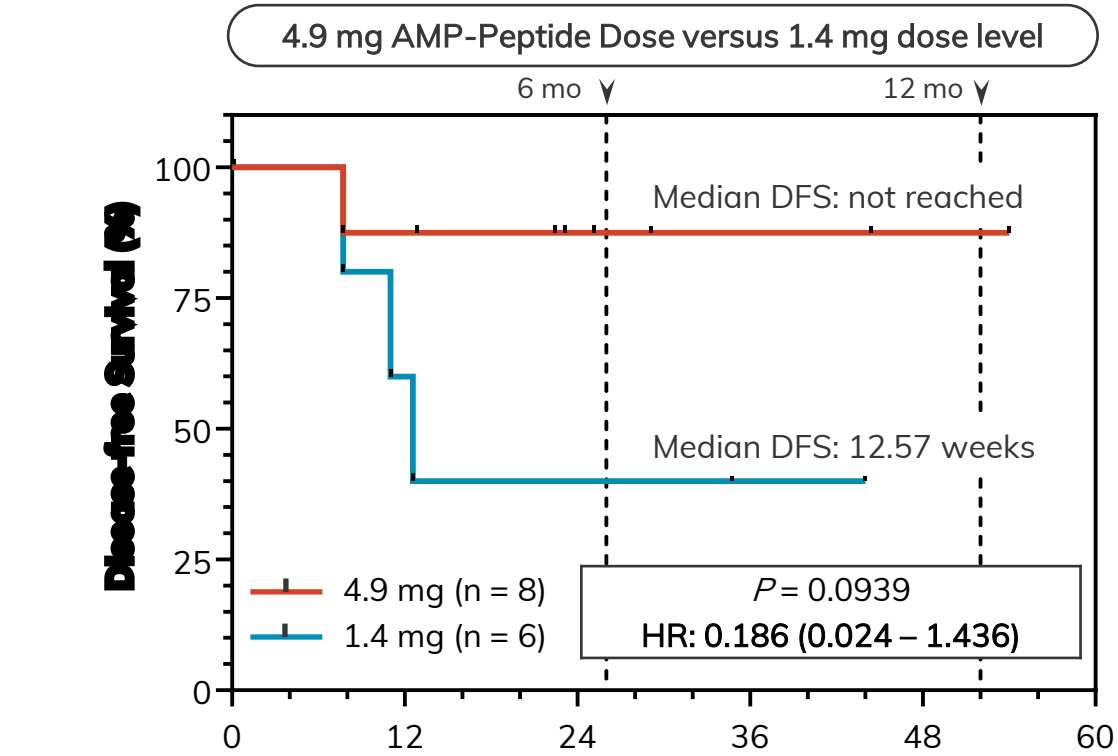
AMPLIFY-7P: Preliminary Phase 1 Disease-free Survival

ELI-002 7P | Phase 1A

Improved DFS observed at 4.9mg Phase 2 dose versus 1.4 mg dose

ELI-002 7P Disease-free Survival

- AMP-Peptide-7P 4.9 mg dose:
  - Median DFS not reached
  - Associated with 81% decreased risk of disease progression and death compared to 1.4 mg *(P = 0.0939)*



No. at Risk						
1.4 mg	6	3	2	1	0	
4.9 mg	8	7	4	2	1	0

Data cutoff 24-May-24

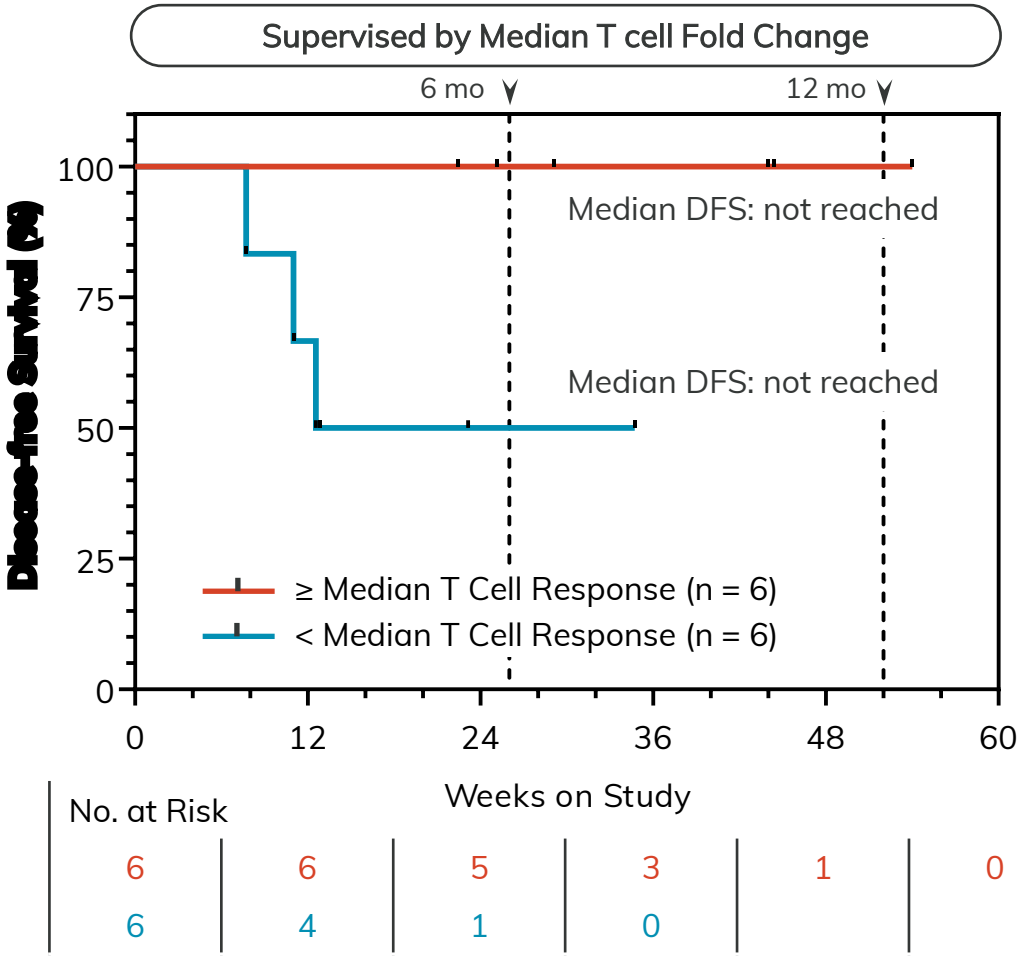
AMPLIFY-7P: Preliminary Phase 1 Disease-free Survival

ELI-002 7P | Phase 1A

Improved DFS associated with above median T cell response

ELI-002 7P Disease-free Survival

- Induction of above median mKRAS-specific T cell responses by ELI-002 7P associated with decreased risk of disease progression and death compared to below median T cell response
- All patients with above median T cell responses are free from disease progression



Data cutoff 24-May-24

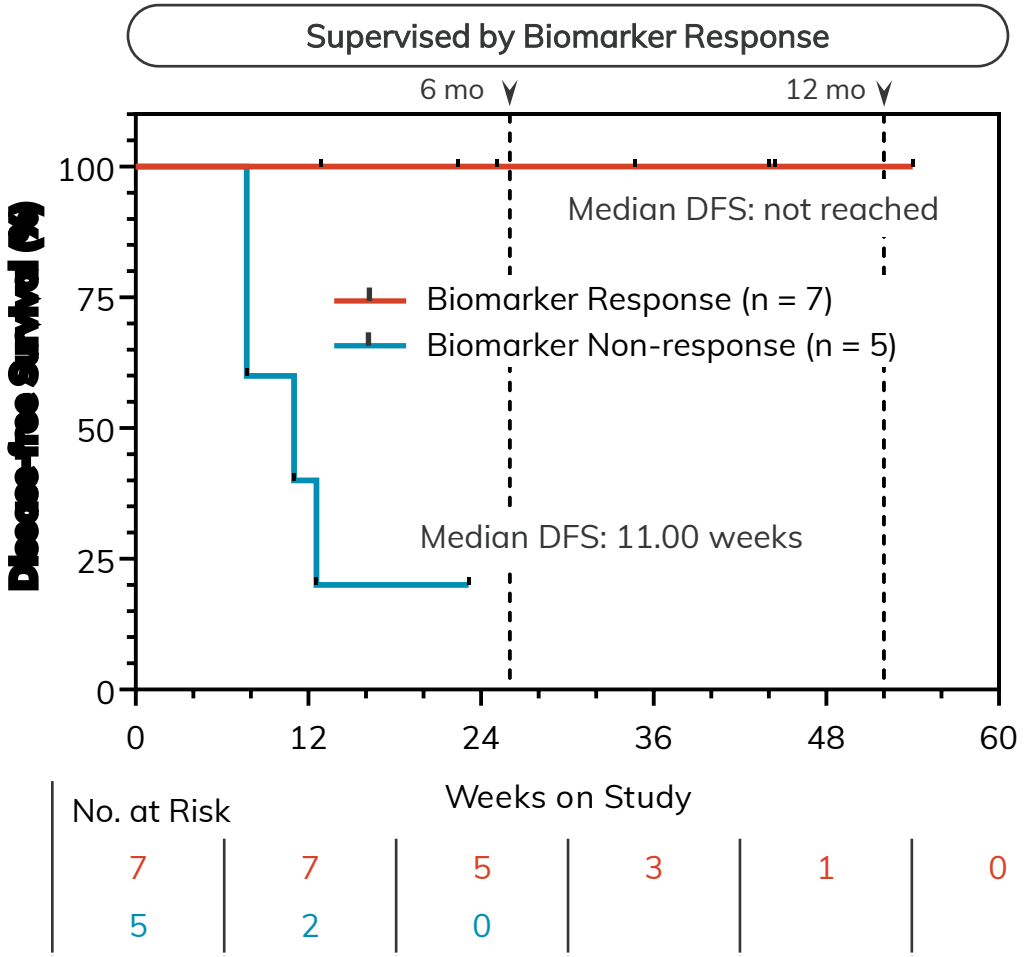
AMPLIFY-7P: Phase 1 Disease-free Survival

ELI-002 7P | Phase 1A

Improved DFS associated with biomarker response

ELI-002 7P Disease-free Survival

- Tumor biomarker reduction or clearance after ELI-002 7P treatment associated with decreased risk of disease progression and death compared to below median T cell response
- All patients with tumor biomarker reduction or clearance are free from disease progression



Data cutoff 24-May-24

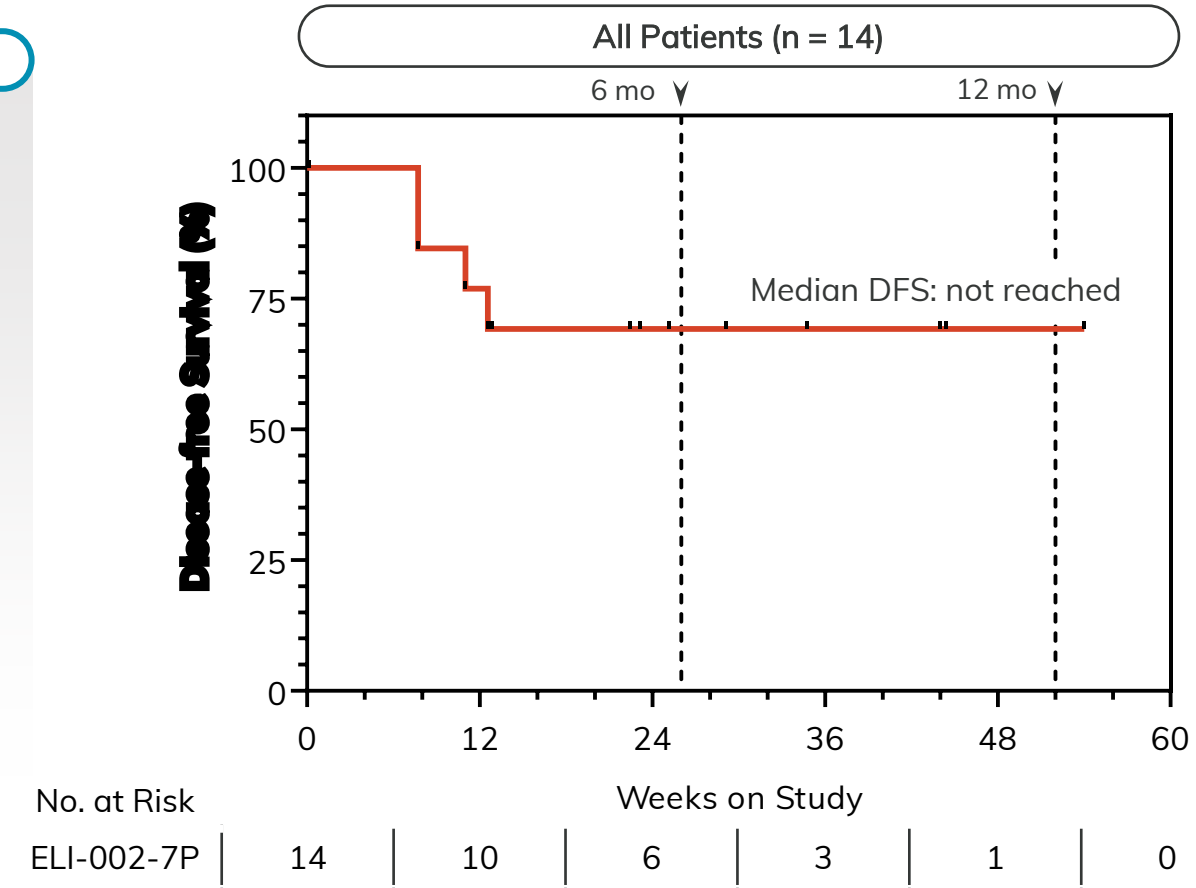
AMPLIFY-7P: Preliminary Phase 1 Disease-free Survival

ELI-002 7P | Phase 1A

Favorable overall DFS observed

ELI-002 7P Disease-free Survival

- Full Phase 1 Cohort:
  - Median DFS not reached



Data cutoff 24-May-24

ELI-002 7P: 7-Peptide (7P) Formulation

Phase 2: 135 PDAC Patients Randomized 2:1

ELI-002 Randomized Phase 2 PDAC, n=135 patients

ELI-002 7P | Phase 2

2:1 Randomized, Open Label Study with 1^0 DFS endpoint

CLINICAL STUDY OVERVIEW: NCT05726864

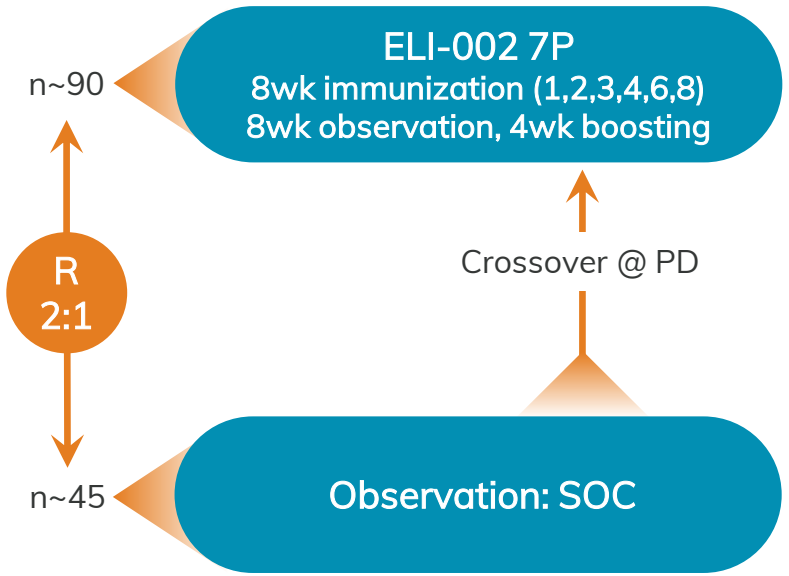
Monotherapy (no chemo, CPI combo)

- ✓ mKRAS: Expanded Antigen Coverage
  G12D / R / V / C / A / S / G13D

Phase 2: Key Criteria

- ✓ Includes: mKRAS G12D/R/V/C/A/S/G13D
- ✓ Up front resectable Stage I, II or III disease (PDAC)
- ✓ Complete R0/R1 resection
- ✓ Radiographic NED status within 6 months following completion of locoregional treatment
- ✓ MRD agnostic (biomarker +/- included)

Schema



Endpoints

- Primary Endpoint: Disease Free Survival
- Tumor Biomarker Response (biomarker subset)
- Overall Survival, Safety, iRECIST Overall Response Rate
- Exploratory: Immunogenicity

Phase 2 interim analysis expected in 1Q 2025

Key Milestones and Growth Initiatives 2024-2025

ELI-002 2P Phase 1 Study

- ✓ Published Preliminary Phase 1a Data in **Nature Medicine**
- ✓ Antigen spreading and expanded immunogenicity data (AACR)
- ❑ Updated RFS and T Cell response data (4Q 2024)

ELI-002 7P Phase 1/2 Study

- ✓ Preliminary Phase 1 T Cell and biomarker response (ASCO)
- ❑ Complete 135 patient Phase 2 enrollment (4Q 2024)
- ❑ Phase 2 DFS Interim Analysis (1Q 2025)

Growth Initiatives

- ❑ Initiate ELI-002 Phase 1B Colorectal Cancer study
- ❑ Advance ELI-007 / ELI-008 into Phase 1

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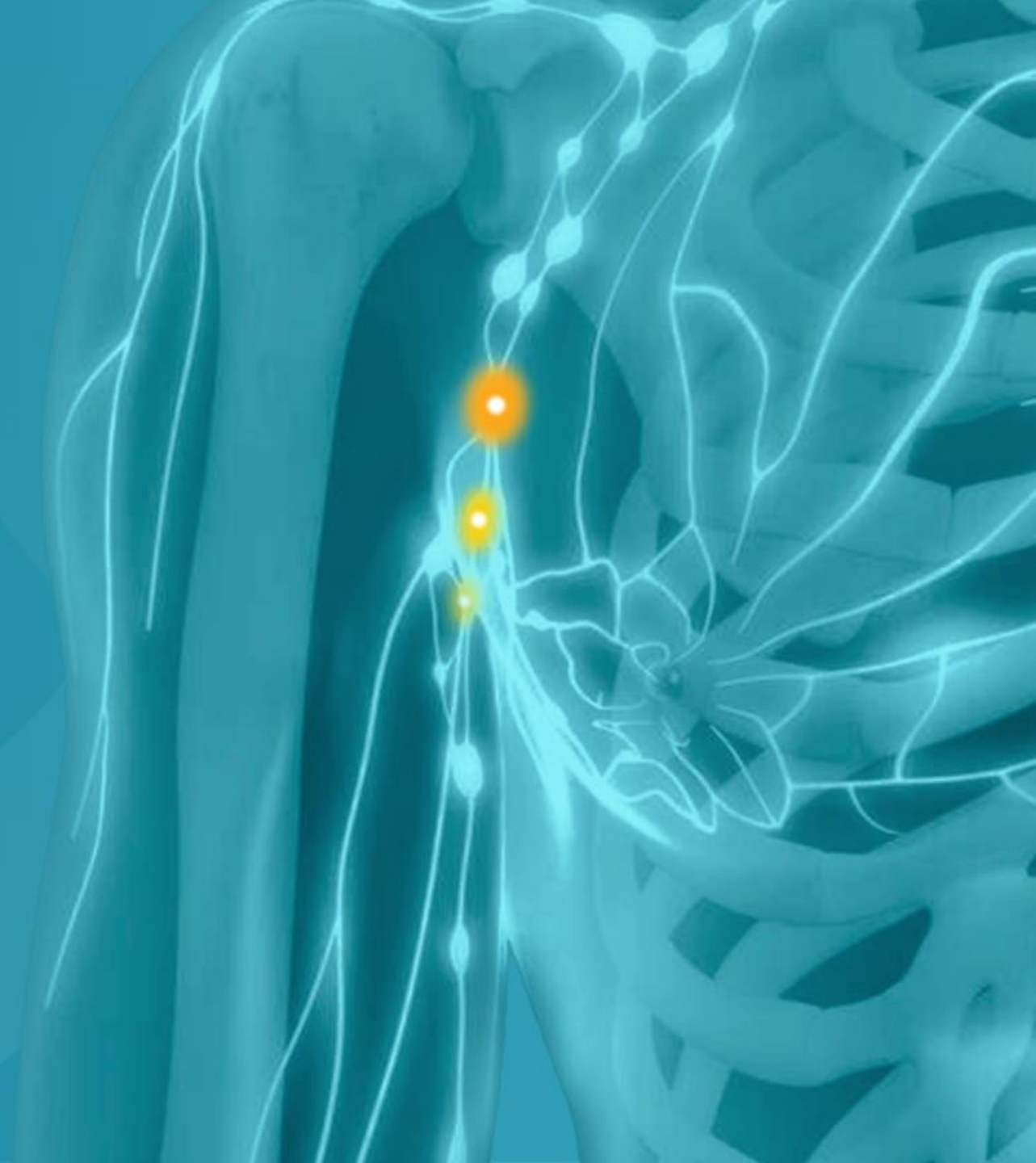
Targeting the Lymph Nodes to AMPlify Immunotherapy
Nasdaq: ELTX

June 2024