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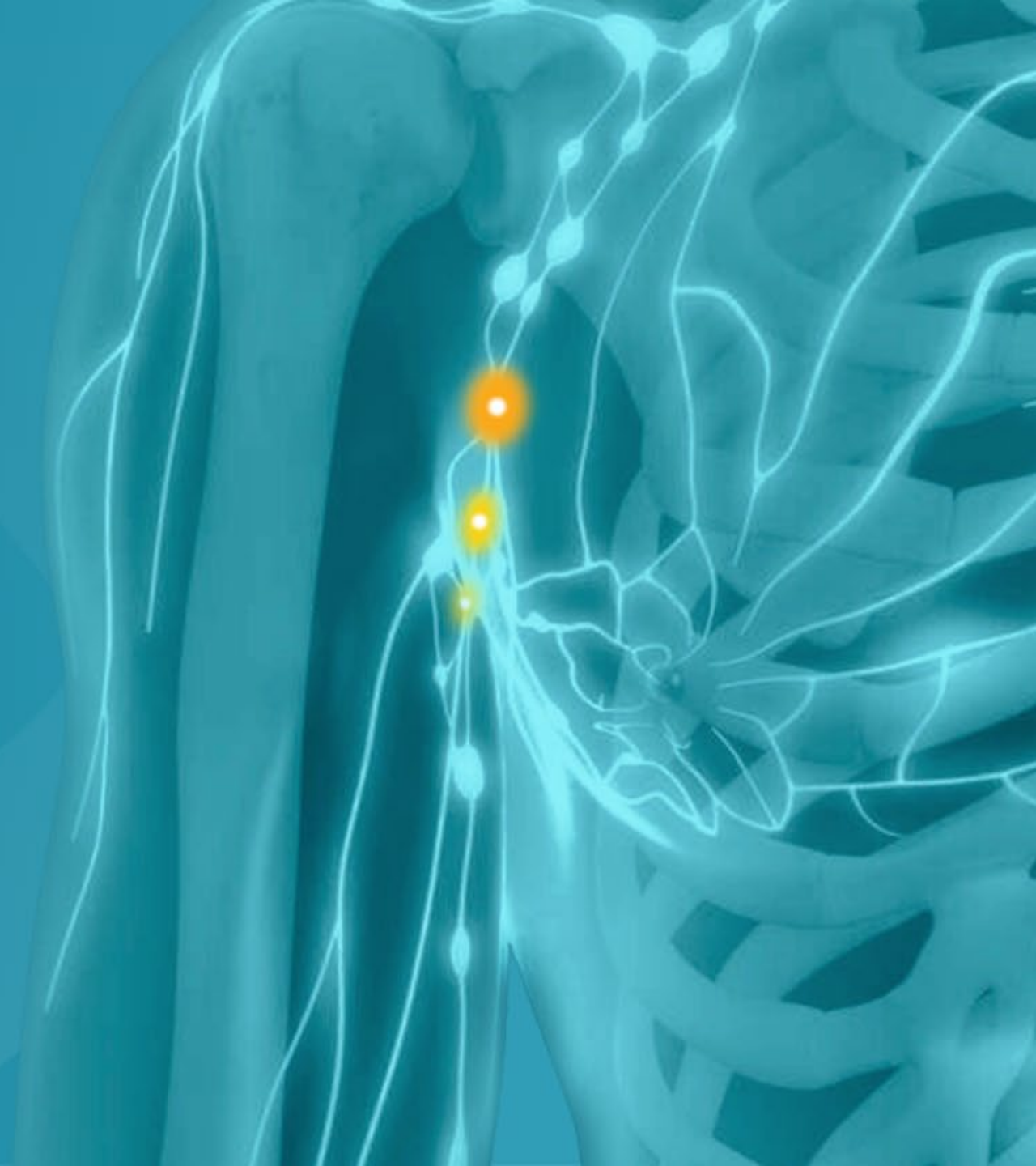
Targeting the Lymph Nodes to AMPlify Immunotherapy

Nasdaq: ELTX

May 2025



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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 the sufficiency of our existing cash to support operations, our planned clinical programs, including planned clinical trials and the potential of our product candidates, including the potential durable clinical benefits and potential broad application 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 “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 financial condition, including our anticipated cash runway; our ability to obtain the funding necessary to advance the development of ELI-002 and any other future product candidates; our ability to continue as a going concern; our plans to develop and commercialize our product candidates, including ELI-002; the timing of initiation of our planned clinical trials, including advancing ELI-007 BRAF and ELI-008 p53 vaccines for Phase 1 readiness and working with investigators to initiate the ELI-002 clinical study in additional KRAS-mutated tumor indications; the timing and initiation of investigator-sponsored trials, including studies of ELI-002 plus checkpoint inhibitors in pancreatic ductal adenocarcinoma (“PDAC”) and colorectal cancer (“CRC”) and other combinations; the outcome of our anticipated ELI-002 7P End of Phase 2 U.S. Food and Drug Administration (“FDA”) meeting; the potential timing and ability to finalize our Phase 3 trial protocol in adjuvant PDAC for ELI-002; the timing of the availability of data from our clinical trials, including the disease-free survival interim analysis from the ELI-002 7P Phase 2 trial; the timing of any planned investigational new drug application or new drug application; our plans to research, develop and commercialize its 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 31, 2025, 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 this release. We do not undertake any obligation to update such forward-looking statements to reflect events or circumstances after the date of this release, except to the extent required by law.

Investment Highlights

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

Leveraging proprietary Amphiphile (“AMP”) Technology

- Our proprietary AMP platform is designed to generate robust, functional, and durable immune responses by targeting lymph nodes
- AMP technology delivers antigen-specific payloads directly to lymph nodes to educate, activate, and expand tumor-eliminating T cell populations and has the potential for broad applicability across cancer immunotherapy
- Proof-of-concept has been demonstrated in two completed Phase 1 trials; a randomized Phase 2 study is expected to read out this year

ELI-002
Lymph Node Targeted mKRAS Cancer Vaccine

- Off-the-shelf cancer vaccine candidate targeting the most common KRAS mutations that drive 25% of solid tumors
- Potential monotherapy adjuvant treatment in high relapse-risk mKRAS^{+} cancers: pancreatic (PDAC), colorectal (CRC)
- Completed two Phase 1 trials and currently in randomized Phase 2 trial in PDAC:
  - ELI-002 2P elicited mKRAS-specific T cell response **~100x** increased over baseline at the Phase 2 dose without any DLTs or SAEs
  - ELI-002 2P Update at ESMO-IO: full cohort (n=25) mOS of **28.9 months**; **16.3 months** mRFS

Value-creating catalysts and capitalization

- ELI-002 7P Phase 2 trial: Disease-free survival interim analysis expected in Q3 2025
- ELI-002 7P End of Phase 2 FDA meeting expected in H2 2025
- Investigator-sponsored trials of ELI-002 + checkpoint inhibitors (CPI) in PDAC and CRC and other combinations
- Cash runway expected to support operations into Q4 2025 beyond anticipated Phase 2 interim data analysis in Q3 2025

Seasoned Management Team

Robert Connelly

Chief Executive Officer

axcella

Flagship Pioneering

PULMATRIX

domantis

BioVeris
THE GLOBAL BIOSECURITY COMPANY™

Abbott

Peter DeMuth, PhD

Chief Scientific Officer

KOCH INSTITUTE for Integrative Cancer Research at MIT

Massachusetts Institute of Technology

NIH

NOVARTIS

Whitehead Institute

Christopher Haqq, MD, PhD

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

ATARA BIO

AMGEN

Cougar Biotechnology

janssen

UCSF

HARVARD UNIVERSITY

Massachusetts Institute of Technology

Stanford University

Preetam Shah, MBA, PhD

Chief Strategy and Financial Officer

CIDARA THERAPEUTICS

brainstorm cell therapeutics

BARCLAYS

cg Canaccord Genuity

Stanford University

Wharton University of Pennsylvania

Megan Filoon, JD

General Counsel, Secretary and Compliance Officer

tem
MOLECULAR TEMPLATES

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RUTGERS

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Company Pipeline

Innovative Pipeline of Cancer Immunotherapies Addressing Critical Unmet Needs

Candidate	Target	Indication	Setting
ELI-002	mKRAS	PDAC	Adjuvant
ELI-002	mKRAS	CRC	Adjuvant
ELI-002	mKRAS	CRC	Metastatic
ELI-002 + CPI	mKRAS	PDAC	Neoadjuvant PDAC
ELI-004 + Radiation		Soft Tissue Sarcoma	Metastatic
ELI-007	mBRAF	GI Tumors	
ELI-008	mTP53	GI Tumors	

Ongoing | Planned [1] | Advancing [1]

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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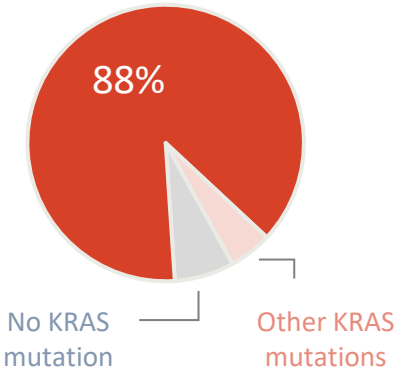
The Addressable KRAS-mutant Market – A Significant Opportunity

ELI-002 Targets the 7 Most Common KRAS Mutations Driving 25% of Solid Tumors

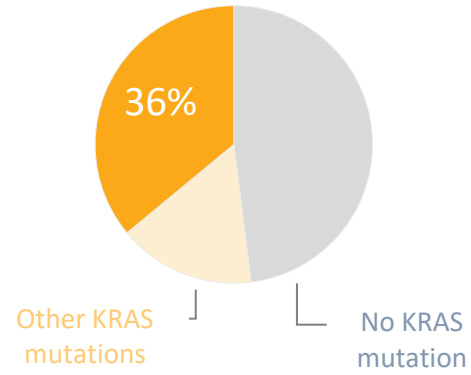
Pancreatic Ductal Adenocarcinoma

ELI-002 addressable
Incidence: ~128k



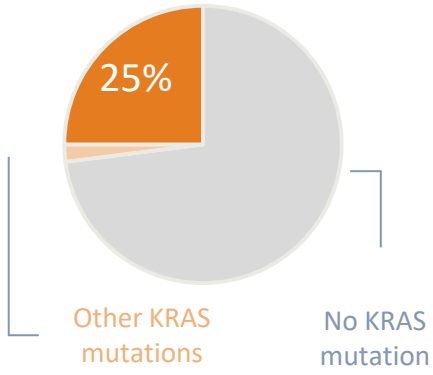
Colorectal Cancer

ELI-002 addressable
Incidence: ~192k



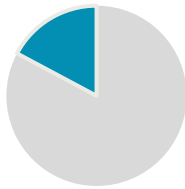
Non-Small Cell Lung Cancer

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

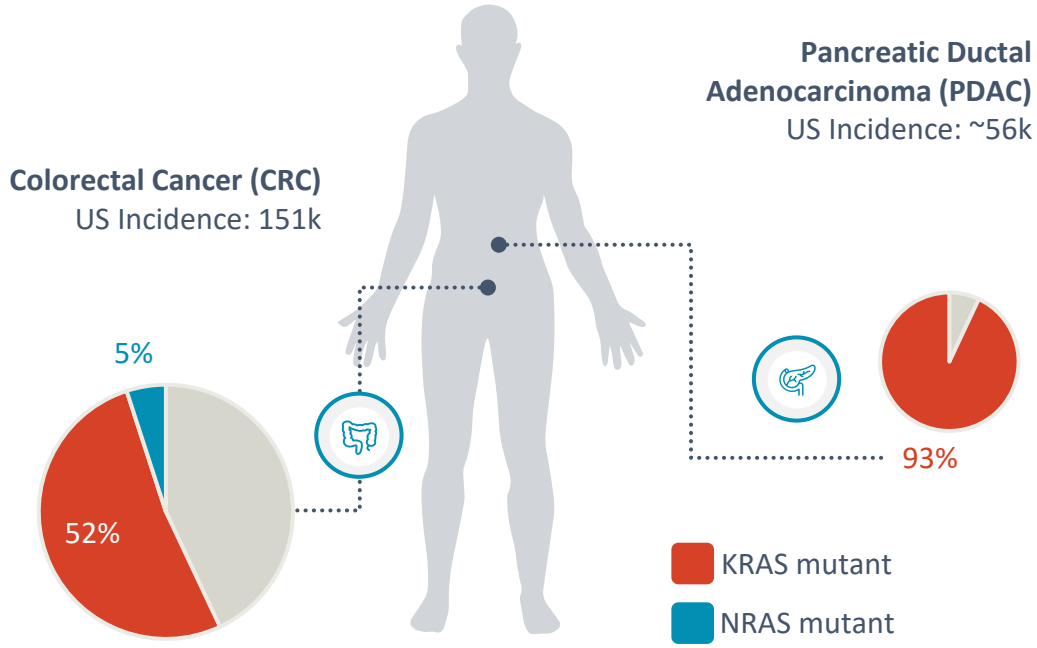
Significant Opportunity to Treat Multiple Common Cancers with KRAS Mutations

Mutant KRAS Drives 25% of Solid Human Cancers

- Prevalent among numerous tumor types
- Overall poor clinical prognosis
- Limited therapeutic options

Mutant KRAS is a Promising Tumor Antigen

- Mutations occur early and expressed uniformly in tumor cells
- Broad HLA presentation
- Multi-targeting potential



KRAS-mutant cancers represent a significant market opportunity for ELI-002, with PDAC offering an especially compelling case due to the high unmet need.

Bianken, et al. **Nature**. 2012

ELI-002's Differentiated Approach to mKRAS Therapy

Validated mKRAS Target | Differentiated Vaccine Approach | Advanced Clinical Stage

Small Molecules Inhibiting Mutant KRAS

- FDA approvals for LUMAKRAS® & KRAZATI® validate target
- Mirati acquisition: $4.8B by BMS

BUT

- Only affects 1 mutation (G12C), subject to multiple resistance mechanisms
- Limited duration of clinical benefit

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
- Combination with CPI needed

Lymph Node Targeted Vaccine Targeting Mutant KRAS

- Promising P1 data in PDAC and CRC
- Targeting 7 mKRAS driver mutations
- Lymph Node Targeting MoA
- Off-the-shelf simplicity, COGs
- Robust T cell Response (CD4 + CD8)
- Monotherapy activity
- Potential durable clinical benefit
- Potential broad application for ~25% of human solid cancers expressing mKRAS

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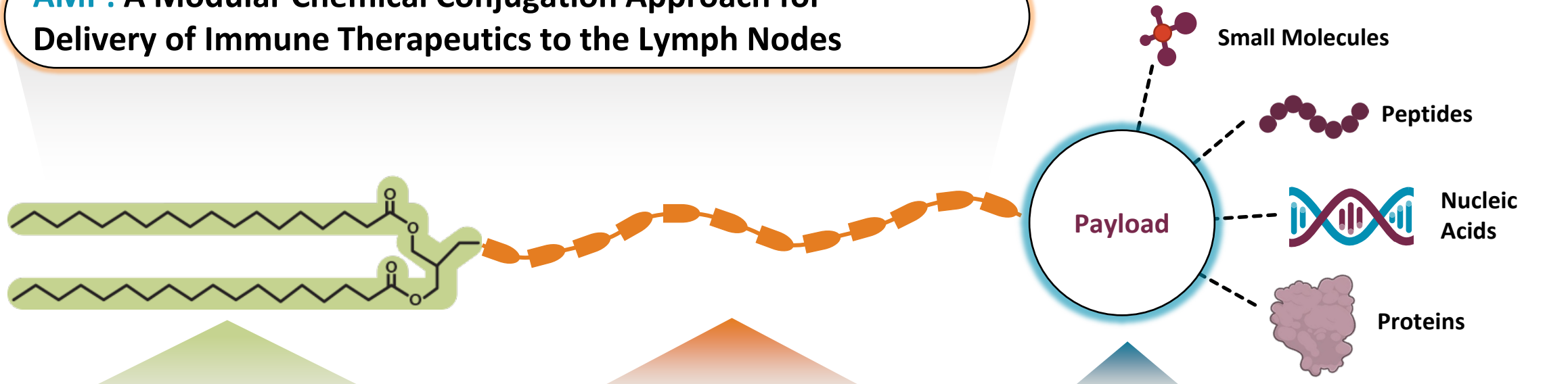
Targeting the Lymph Nodes to Orchestrate Immunity

Amphiphile (AMP) Platform

Amphiphile (AMP) Platform Enables Lymph Node Delivery with Simple and Versatile Application

AMP: A Modular Chemical Conjugation Approach for Delivery of Immune Therapeutics to the Lymph Nodes



1 Albumin Binding Lipid

- ✓ Lymph Node Delivery
- ✓ Lymph Node Retention

2 Polymeric Linker

- ✓ Improved Solubility
- ✓ Controlled Payload Release

3 Payload

- ✓ Antigens
- ✓ Adjuvants

- ➢ *Immune Modulating*
- ➢ *Small Size (<50 kDa)*
- ➢ *Validated Activity*

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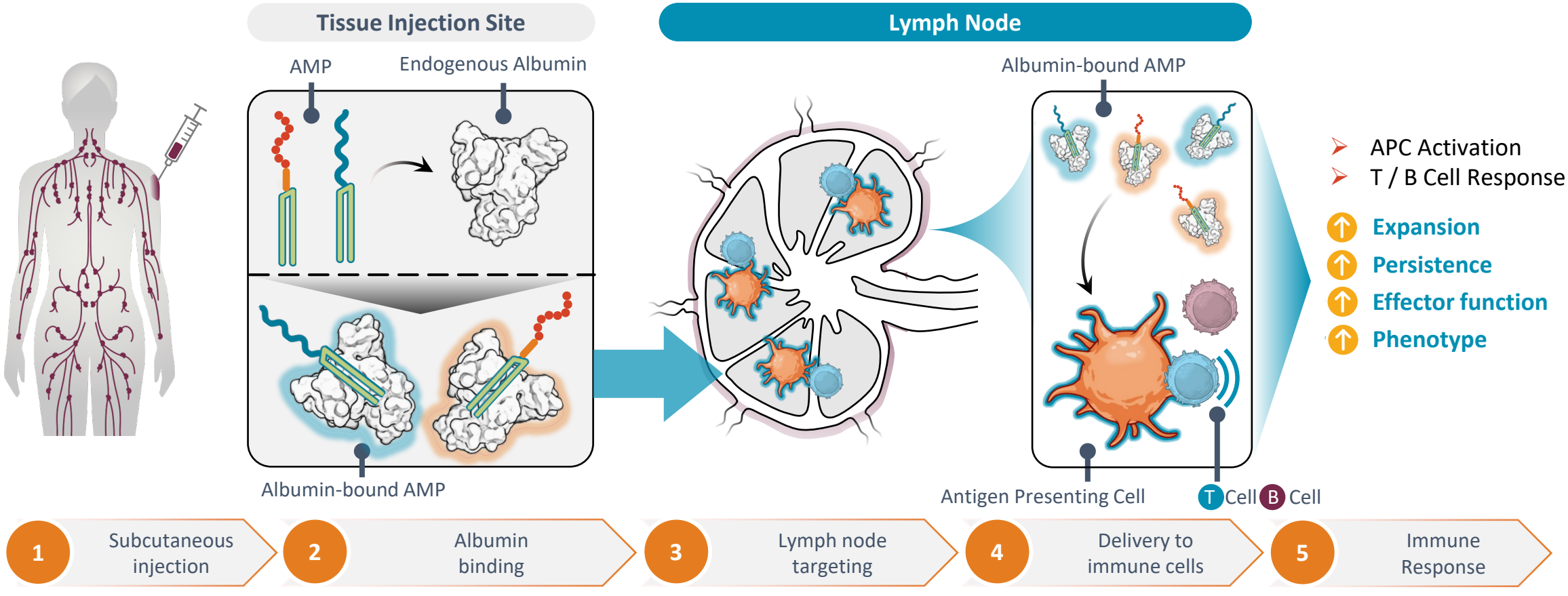
Pant, O'Reilly, et al**. Nature Medicine**. 2024; Liu, et al. **Nature** 2014, Ma, et al. **Science** 2019; Hartwell, et al. **Science Translational Medicine** 2022

MOA: Targeting the Lymph Nodes with AMP to Orchestrate Immunity



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

AMP-vaccination Induces Coordinated Immune Activation in Lymph Nodes

Enhanced Antigen-specific T cell Response Magnitude and Functional Quality

Albumin-based Delivery to Lymph Nodes

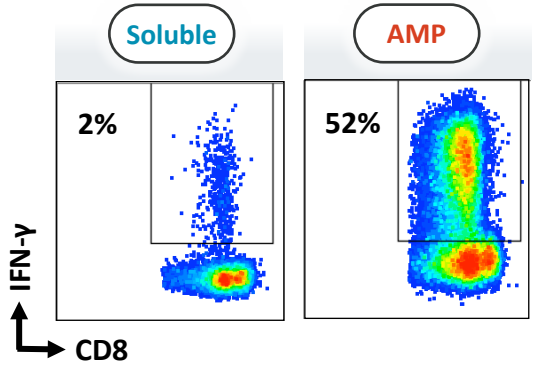
Albumin (65 kDa)

Binding to Tissue Albumin

↑ Enhanced Lymph Node Delivery

↑ T cell Response Magnitude / Function

Blood | IFNγ⁺ T Cell Response | gp100 Antigen



↑ Lymph Node Delivery

↑ T Cell Response

- Expansion
- Persistence
- Effector function
- Phenotype
- Metabolic function

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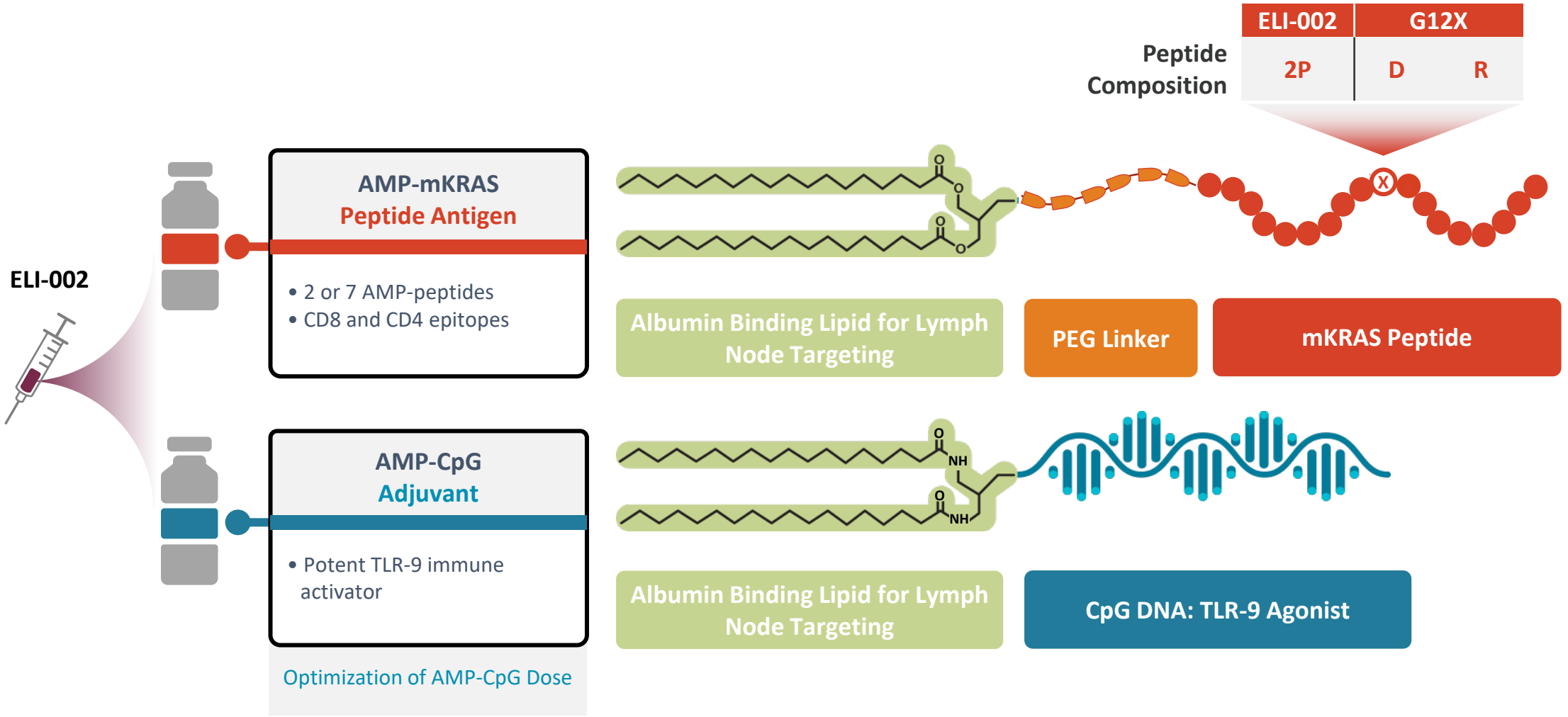
Liu, Irvine, et al. **Nature** 2014; Moynihan, Irvine, et al. **Nature Medicine** 2016

Boosting Endogenous Anti-tumor Immunity Targeting mKRAS

ELI-002 2 Peptide (2P) Formulation

ELI-002 Is a Lymph Node Targeted mKRAS Vaccine

Proprietary AMP-CpG Adjuvant Shown to Enhance Immunogenicity in Early-Stage Trials



ELI-002 2 Peptide Phase 1 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)

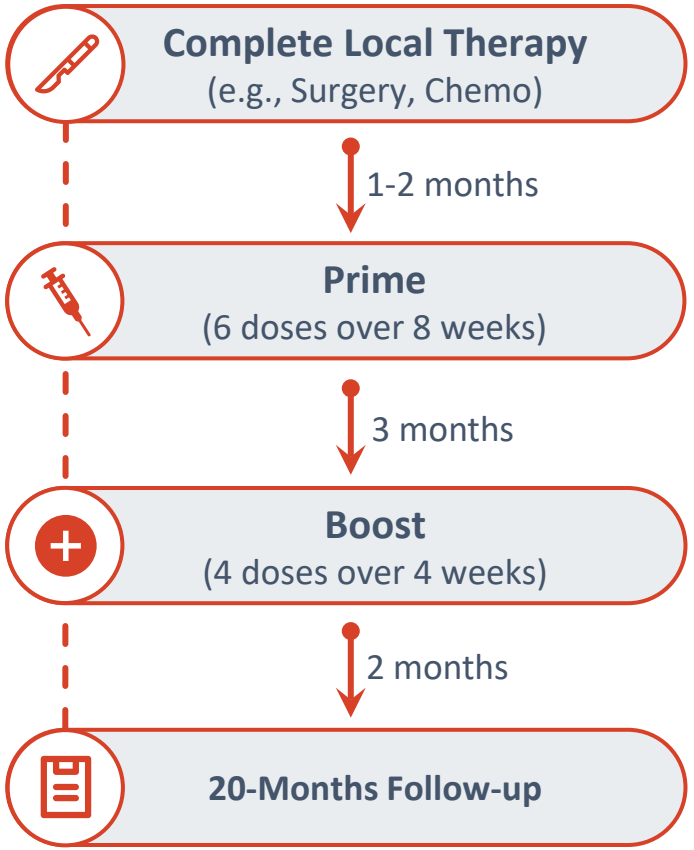
Key Criteria

- ✓ **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

ELI-002 2P Induces Tumor Biomarker Declines in Majority of Phase 1a Patients

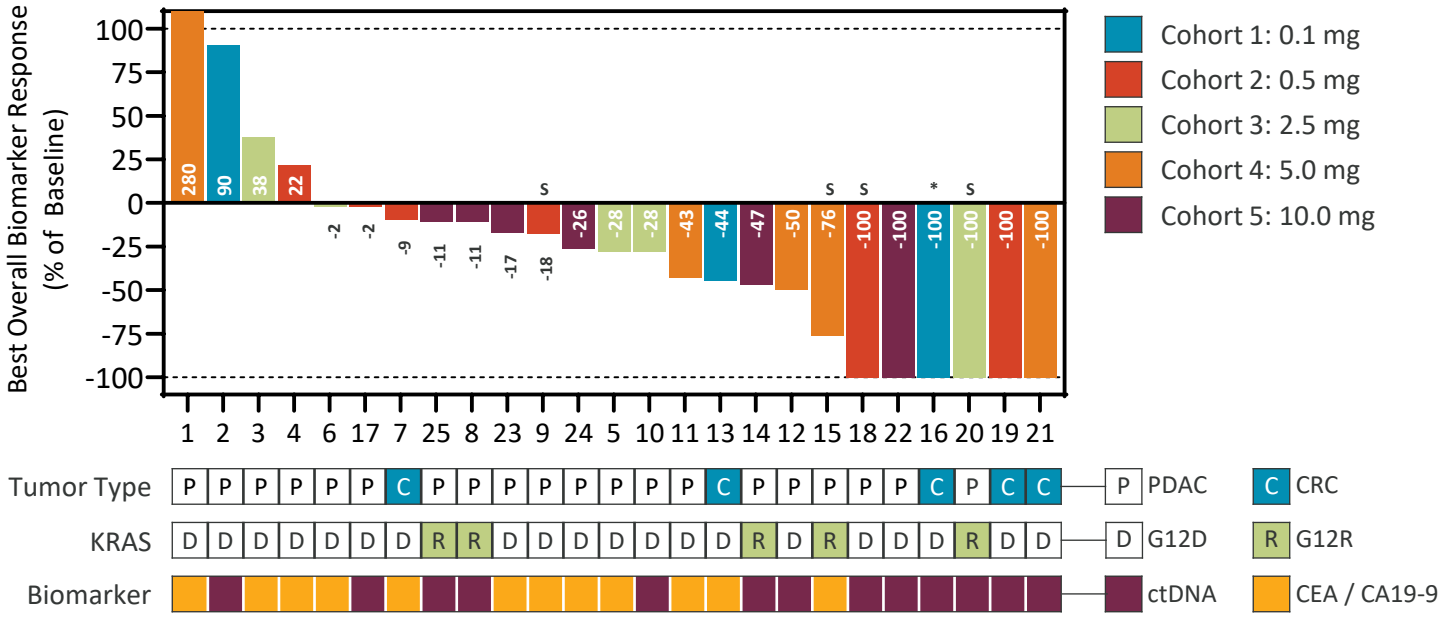
ELI-002 2P | Phase 1A

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)

ELI-002 2P Waterfall Plot: Biomarker Reduction/Clearance



** 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

ELI-002 Elicits Robust Immune Responses in Majority of Phase 1a Patients

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

ELI-002 2P | Phase 1A

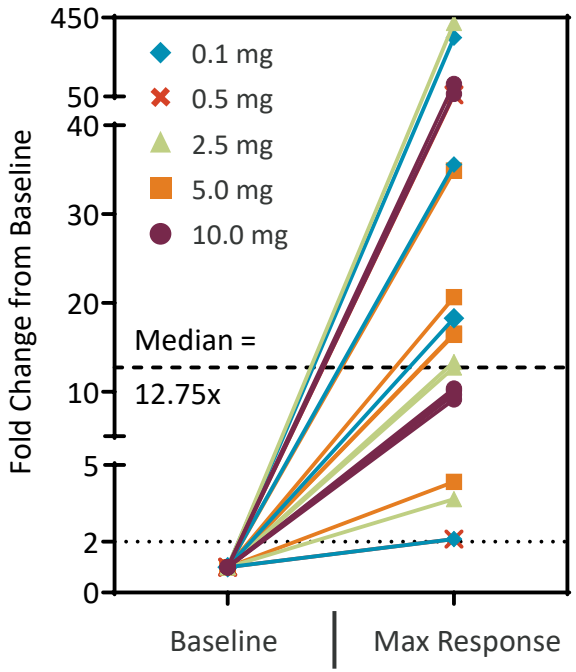
mKRAS T Cell Responses

- 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

Increasing adjuvant concentrations were tested:
10.0 mg adjuvant was identified as the optimal adjuvant concentration.

ELI-002 2P T Cell Fold-Changes

Direct Ex Vivo T Cell Response



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

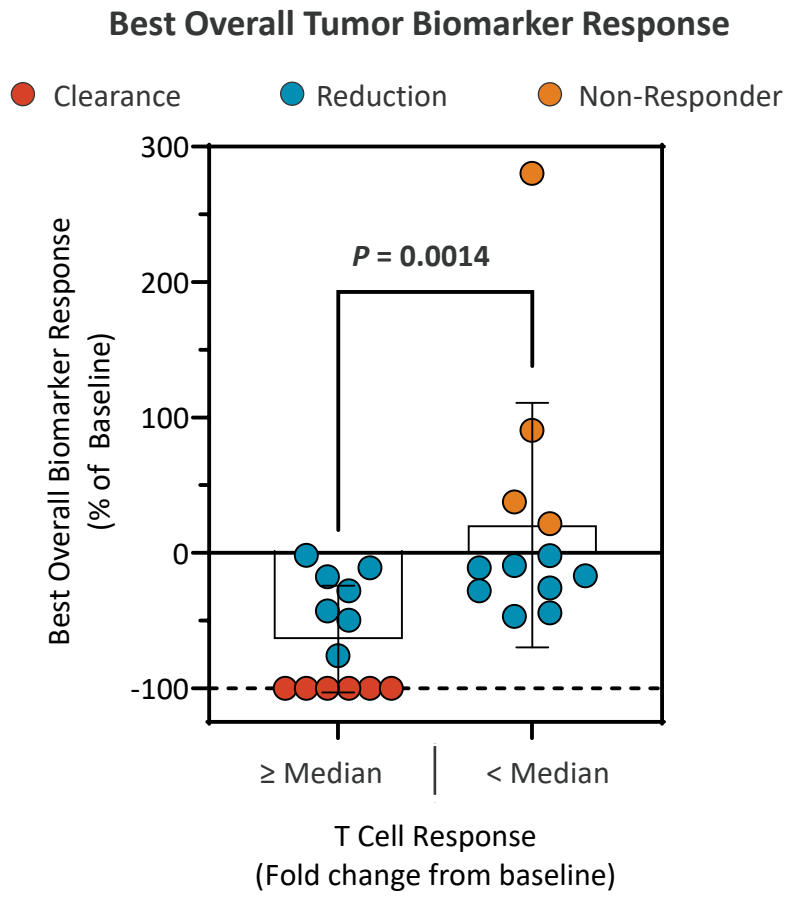
ELI-002 2P: T cell Response Magnitude Correlates with Tumor Biomarker Response

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 patients in the above-median T cell group responded to ELI-002, compared to 67% (8/12) in the below-median group**
- All (100%) of the observed tumor biomarker clearances (6/6) are in the above median T cell group
- Statistically significant per Mann Whitney Test (P < 0.0014)



Data cutoff 6-Sept-23

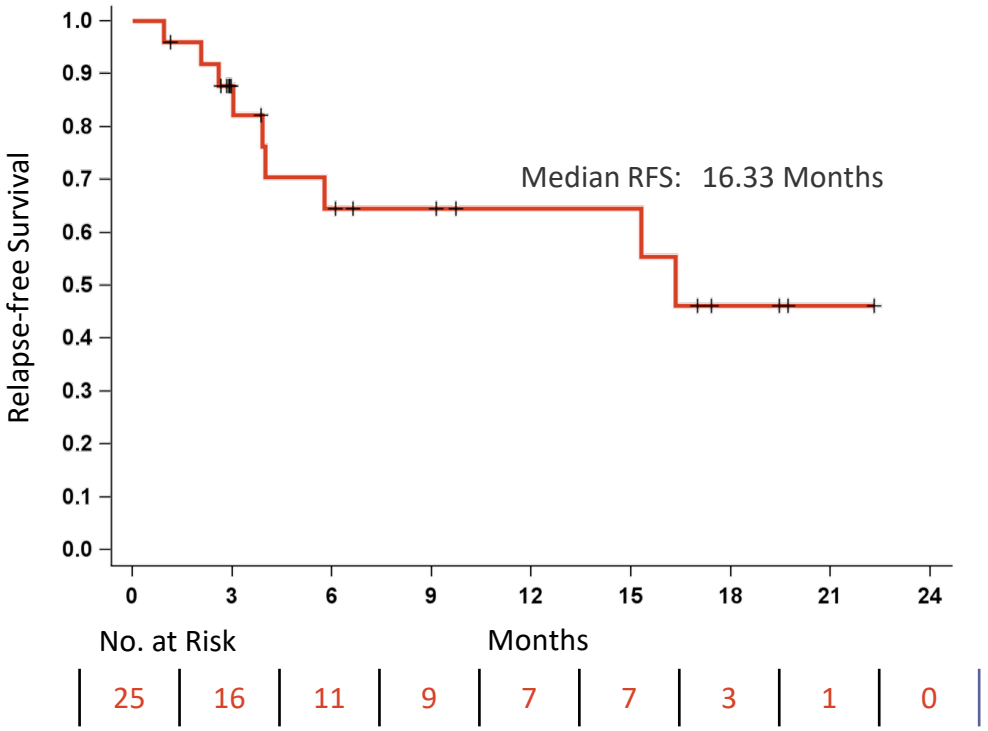
Pant, O'Reilly, et al. **Nature Medicine**. 2024

ELI-002 2P | Phase 1A

ESMO-IO UPDATE: RFS in Full Cohort vs PDAC Subgroup

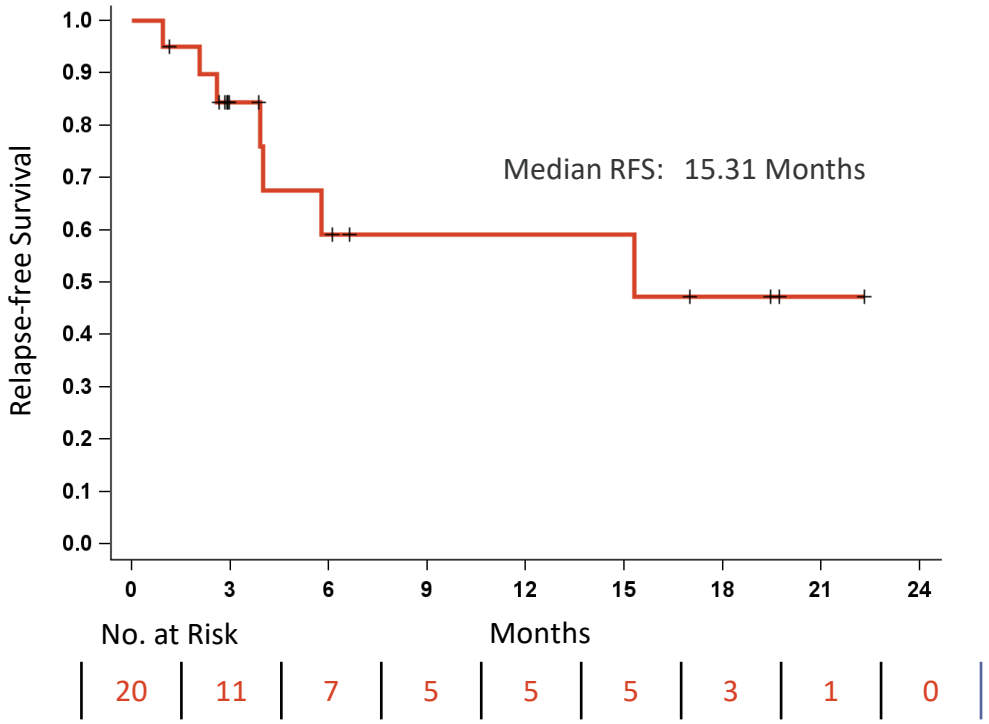
Median follow up has increased to 19.7 months vs 8.5 months in Pant et al., 2024 Nature Med
Median RFS times similar for the full cohort and PDAC subgroup: Data cut-off Sept 24, 2024

Relapse-free Survival: Full Cohort (n=25)



Median follow-up for study: 19.7 months

Relapse-free Survival: PDAC Subgroup (n=20)



Pant, et al. ESMO-IO Annual Meeting. 2024

ELI-002 demonstrated compelling median RFS in the full cohort (~16 months), including the PDAC subgroup (~15 months), which is particularly important given the significant unmet need—only ~10% of patients survive without relapse beyond one year.

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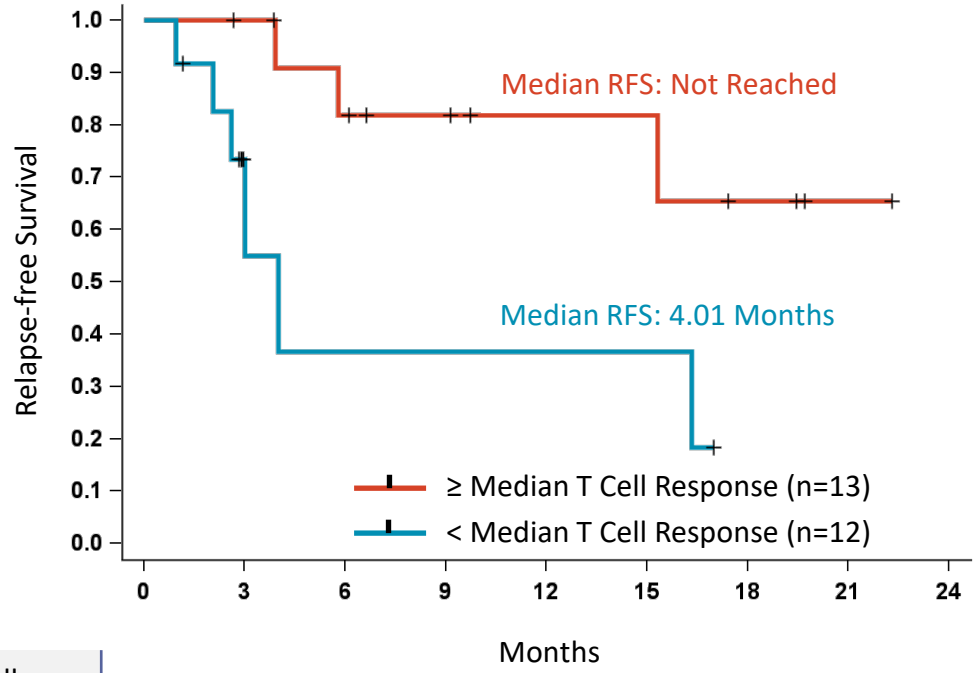
2024 ESMO-IO Update Shows Strong Correlation between RFS and T Cell Response

ELI-002 2P | Phase 1A

RFS Prolonged - no relapse or death in 10/13 (77%) of above median T cell group

Relapse-free Survival: DCO September 24, 2024



T Cell Response	No. at Risk								
≥ Median	13	12	9	7	5	5	3	1	0
< Median	12	4	2	2	2	2	0		

Median follow-up for study: 19.7 months

ELI-002 2P Relapse-free Survival

DCO		06-Sept-2023	24-Sept-2024
Median RFS (Months)	≥ Median T Cell	Not Reached	Not Reached
	< Median T Cell	4.01	4.01
	HR (95% CI)	0.142 (0.0321, 0.6278)	0.226 (0.0552, 0.9277)
	P-value	0.0167	0.0184

Data cut-off (DCO): September 24, 2024

- 10/13 in the above median T cell group have not relapsed or died
- Favorable RFS stratified by T cell response was maintained relative to prior analysis:
  - Median RFS not reached for above median T cell Responders
  - Median RFS 4.01 months for below median T cell Responders
  - HR 0.226, P = 0.0184
- 77% reduction in Risk of Progression or Death due to any cause in above median T cell Responders to ELI-002

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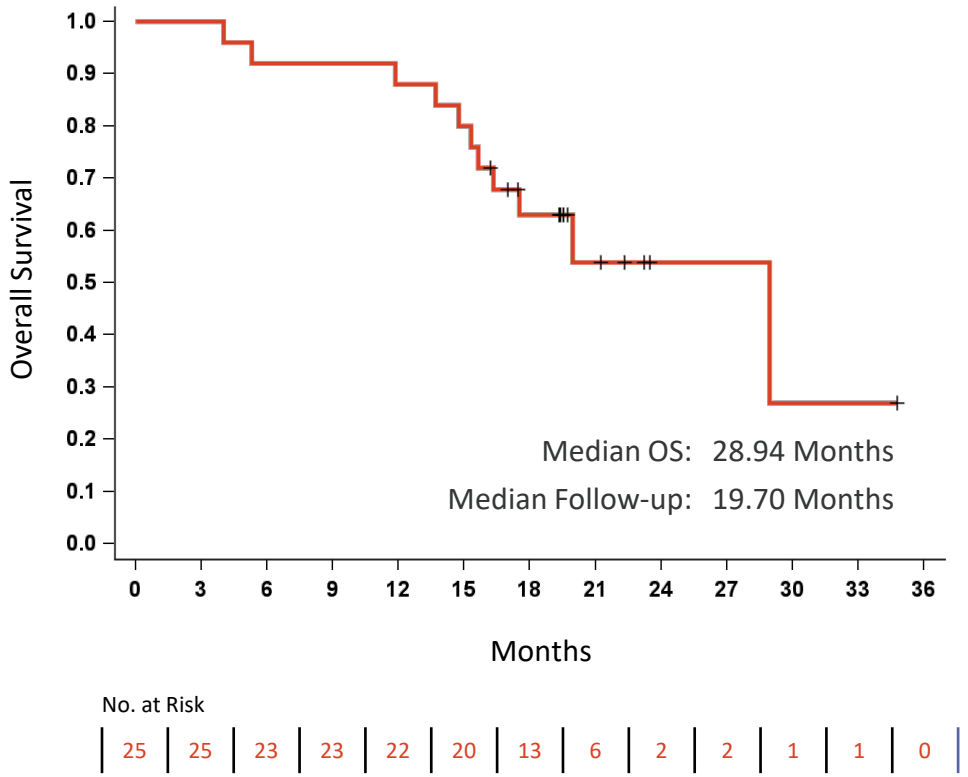
Pant, et al. **ESMO-IO Annual Meeting**. 2024

2024 ESMO-IO Update Shows Early Encouraging Patient Survival

ELI-002 2P | Phase 1A

Full Cohort (n=25) Overall Survival; mOS 28.94 months is longer than historical for PDAC, CRC not yet estimable

Overall Survival: **DCO September 24, 2024**



ELI-002 2P Relapse-free and Overall Survival

Cohort	Full (n=25)	PDAC (n=20)	CRC (n=5)
Median RFS (months)	16.33	15.31	16.33
Median OS (Months)	28.94	28.94	NR
Median Follow-up (Months)	19.7	19.5	23.2

Data cut-off (DCO): September 24, 2024; NR= not reached

- Median RFS for full cohort and PDAC, CRC subgroups are similar
- Median OS for full cohort and PDAC, CRC subgroups are identical
- mOS longer than MRD+ PDAC e.g. 17 mo from resection, Groot et al., 2019. Clin Cancer Res 25:4973

Pant, et al. **ESMO-IO Annual Meeting**. 2024

ELI-002 Summary of Phase 1 Clinical Trials

39 patients treated in two Phase 1A trials: ELI-002 2P and ELI-002 7P

- MRD+ PDAC (n=33) and CRC (n=6) patients treated after local surgery and chemotherapy (adjuvant setting)
- Phase 1 trials included dose-ranging for both peptide and adjuvant components of ELI-002
- Data from both trials have shown:
  - ELI-002 was well tolerated at all dose levels, with no DLTs or SAEs observed
  - RP2D established: 10 mg AMP-CpG with 4.9 mg AMP-peptide mix (elicited median 113-fold T cell increase)
  - ELI-002 elicited a robust mKRAS-specific T cell response (CD4+ and CD8+) in a majority of patients
  - ELI-002 elicited T cell response correlating with a reduction in tumor biomarker levels
  - Evidence of Antigen Spreading at RP2D with immune response targeting personal tumor neoantigens
  - **Strength of ELI-002 T cell response correlates with a reduction in the risk of progression or death**
- Preliminary Phase 1 study of ELI-002 2P including RFS outcome published in Nature Medicine January 2024
- Long-Term Follow-up Phase 1 (ELI-002 2P) data highlighting durable RFS and OS presented at ESMO-IO December 2024

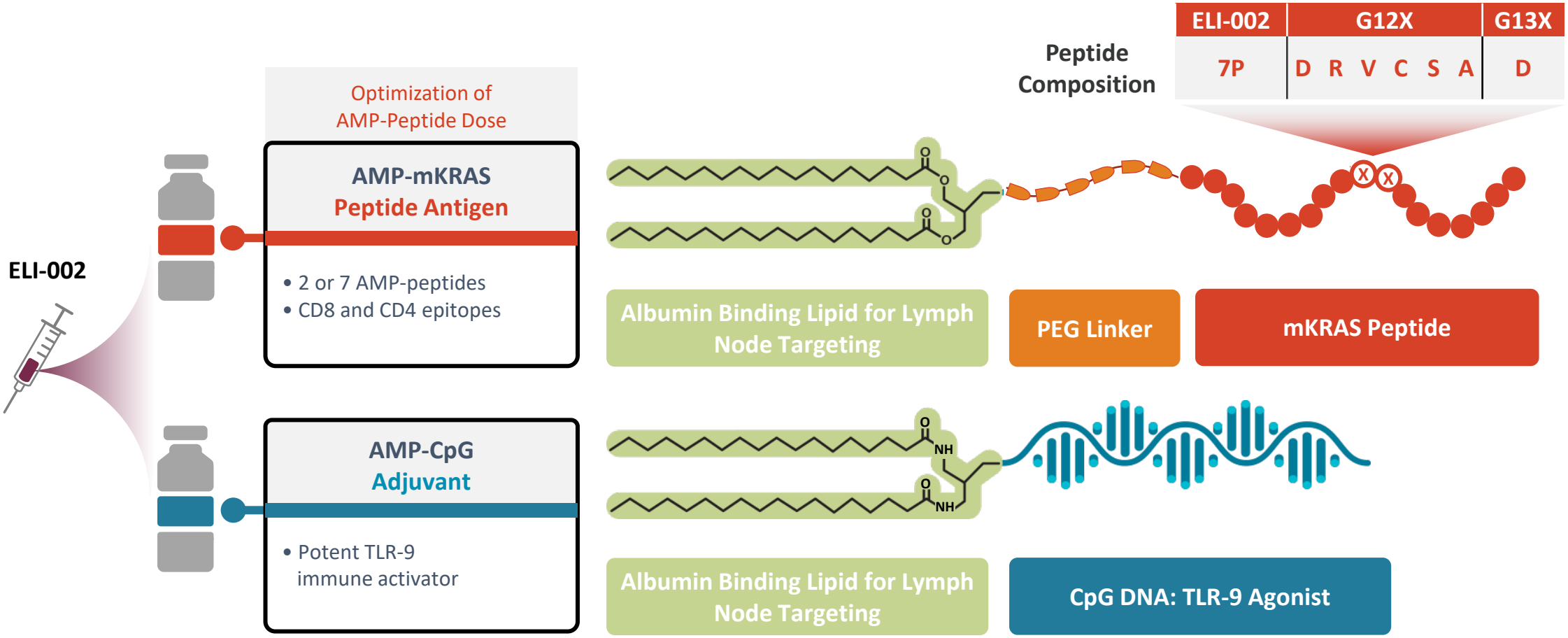
Enhancing Endogenous Anti-tumor Immunity by Using Additional Peptides Targeting mKRAS

ELI-002 7P:
Phase 1a Trial of ELI-002 7-Peptide Formulation

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

ELI-002 Is a Lymph Node Targeted mKRAS Vaccine

Proprietary AMP-CpG Adjuvant Shown to Enhance Immunogenicity in Early-Stage Trials



ELI-002 7P Phase 1A Study Overview

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)

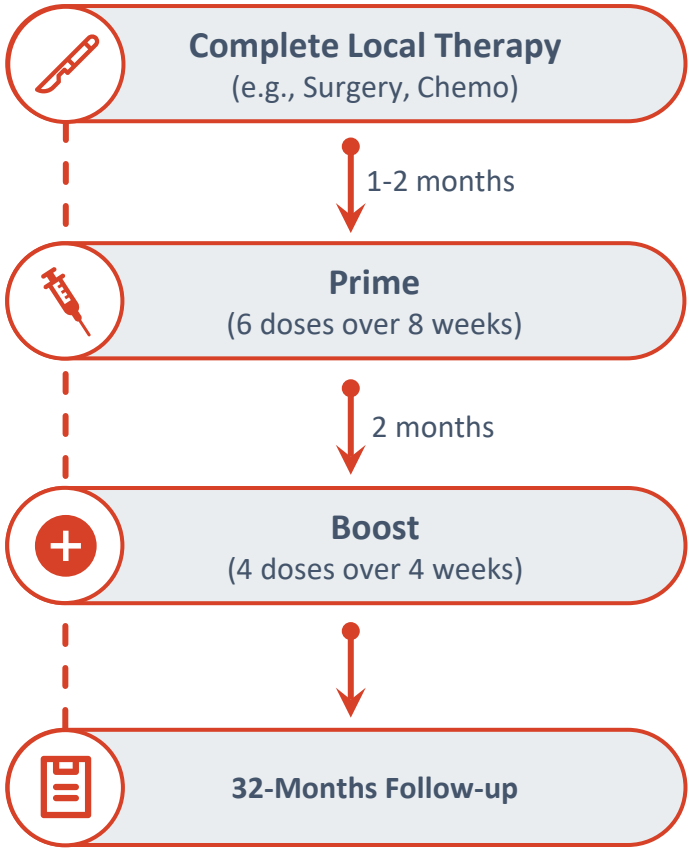
Key Criteria

- ✓ **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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ELI-002 7P Elicits Immune Responses in all Phase 1a Patients

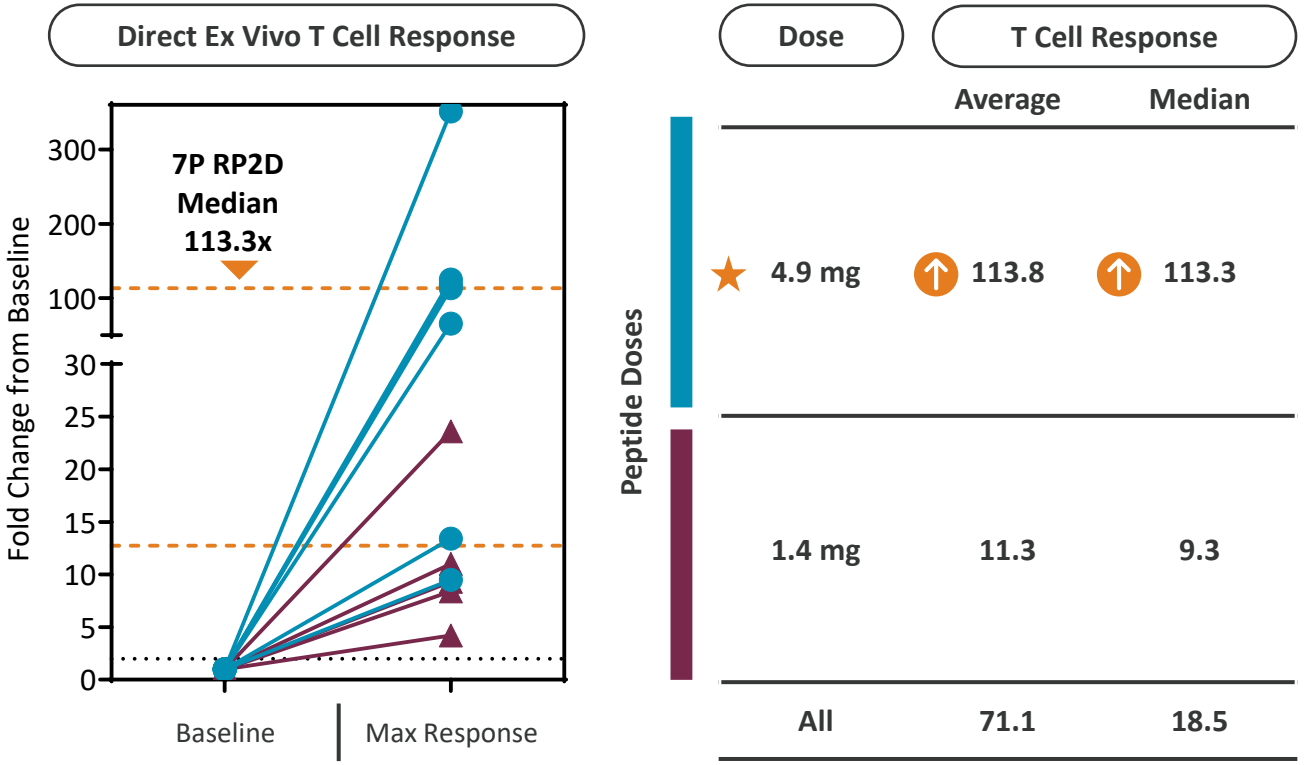
100% of patients with robust T cell response

ELI-002 7P | Phase 1A

mKRAS T Cell Responses

- **100% of patients showed T cell responses**
- 4.9 mg dose group selected for Phase 2
  - Median fold change = **113.3x**
  - 85.7% with CD4 and CD8 T cells
- T cells detectable by standard direct *ex vivo* FluoroSpot and flow cytometry, with no expansion required

ELI-002 7P T Cell Fold-Changes by Dose Level



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

ELI-002 7P: Data cutoff 24-Sep-24

★ = Phase 2 Dose

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ELI-002 7P: T Cell Response Magnitude Correlates with Dose

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=12 Patients (1.4 mg, n=5; 4.9 mg, n=7)
- 100% T cell Response Rate (n=12)
- 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	18.5	↑ **113.3**
CD4 + CD8 T cells	59%	75.0%	↑ **85.7%**
Response to 7 mKRAS Antigens	52.4%	50.0%	↑ **71.4%**
Response to Tumor Antigen	81%	83.3%	↑ **100%**
			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 24-Sep-24

★ = Phase 2 Dose

The ELI-002 7P formulation demonstrated robust T-cell activation in patients.

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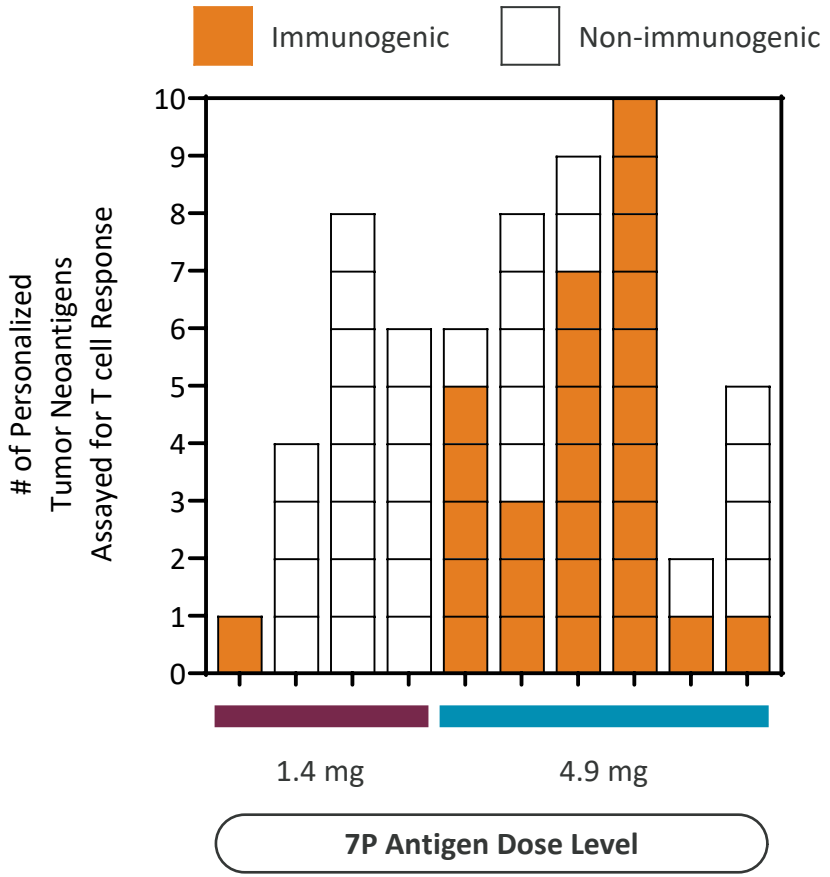
ELI-002 7P: ELI-002 Stimulates Antigen Spreading

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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ELI-002 7P Induces Tumor Biomarker Declines in Majority of Phase 1A Patients

ELI-002 7P | Phase 1A

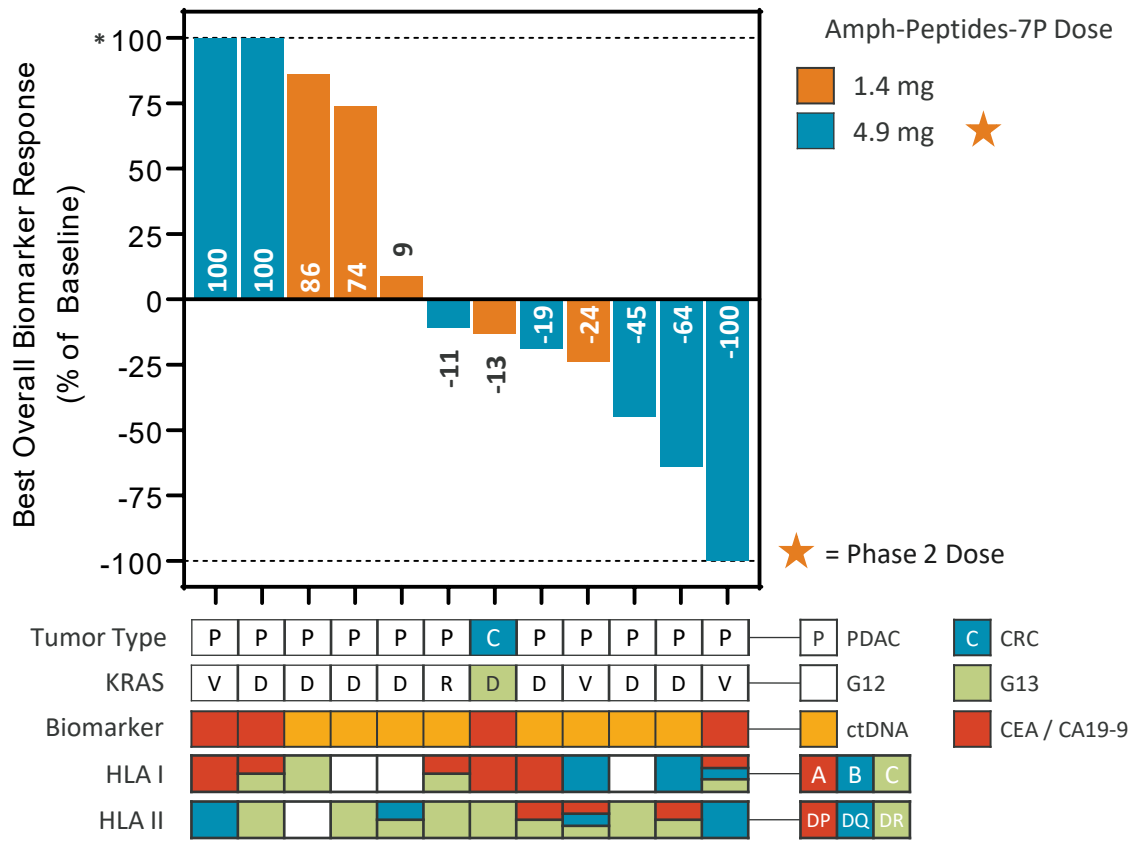
Waterfall reflects superiority of 4.9 mg AMP-Peptide 7P dose level

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)

Data cutoff 18-Dec-23

ELI-002 7P Waterfall Plot: Biomarker Reduction / Clearance



* 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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ELI-002 7P: T Cell Response Drives Tumor Biomarker Response

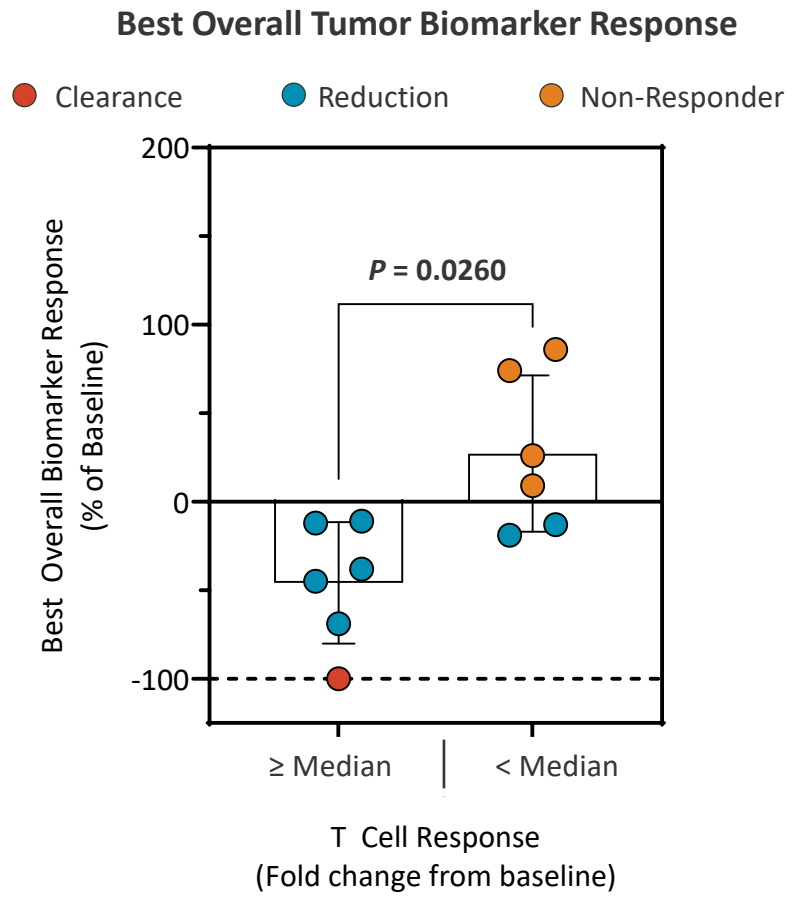
ELI-002 7P | Phase 1A

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% (6/6) of the above median T cell group respond to ELI-002; in the below median group 33% (2/6) respond to ELI-002*
- 71.4% (5/7) of the 4.9 mg dose cohort are in the above median T cell group, including a complete responder
- Statistically significant per Mann Whitney Test (P = 0.0260)

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



Tumor Biomarker data cutoff 18-Dec-23;
T cell biomarker data cutoff 24-Sep-24

ELI-002 7P: DFS Strongly Correlates with T Cell Response

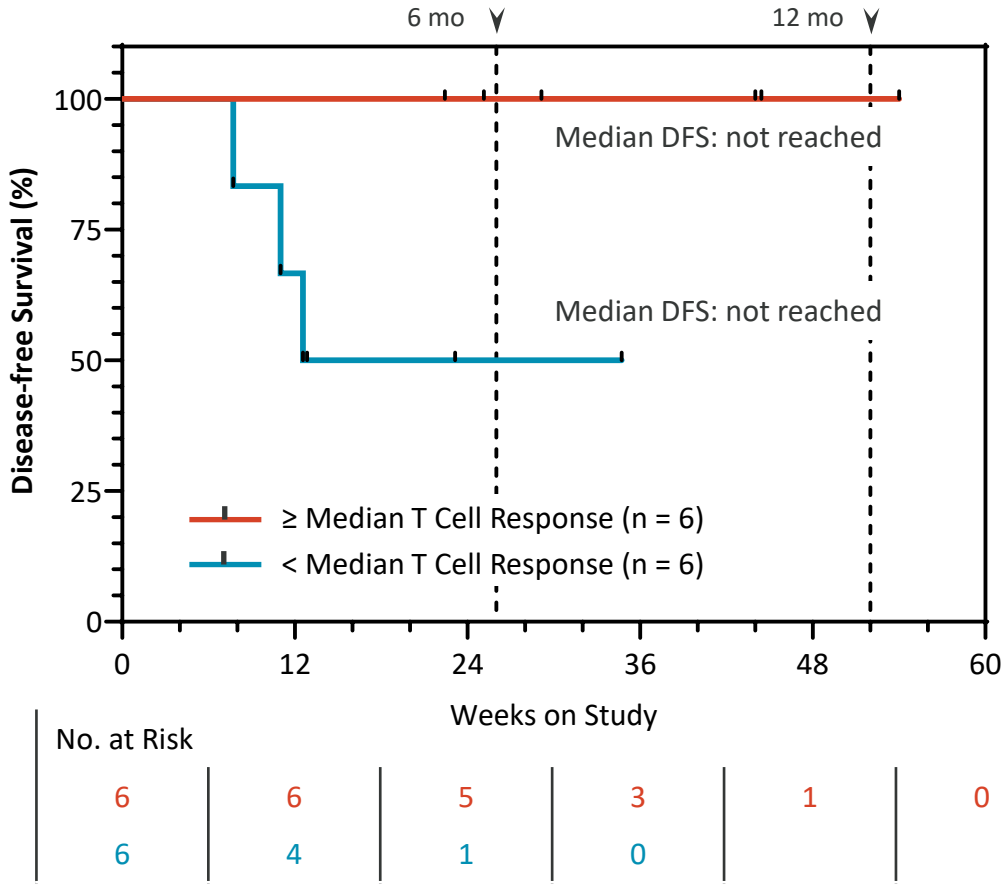
Improved DFS associated with above median T cell response

ELI-002 7P | Phase 1A

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 were free from disease progression as of the data cutoff date

Supervised by Median T cell Fold Change



Data cutoff 23-May-24

ELI-002 7P: Safety

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

ELI-002 7P | Phase 1A

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

ELI-002 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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Enrollment Complete for ELI-002 Randomized Phase 2 PDAC

ELI-002 7P | Phase 2

Event Driven Interim DFS Analysis Expected Q3 2025 for 2:1 Randomized, Open Label Study

CLINICAL STUDY OVERVIEW: NCT05726864

Monotherapy (no chemo, CPI combo)

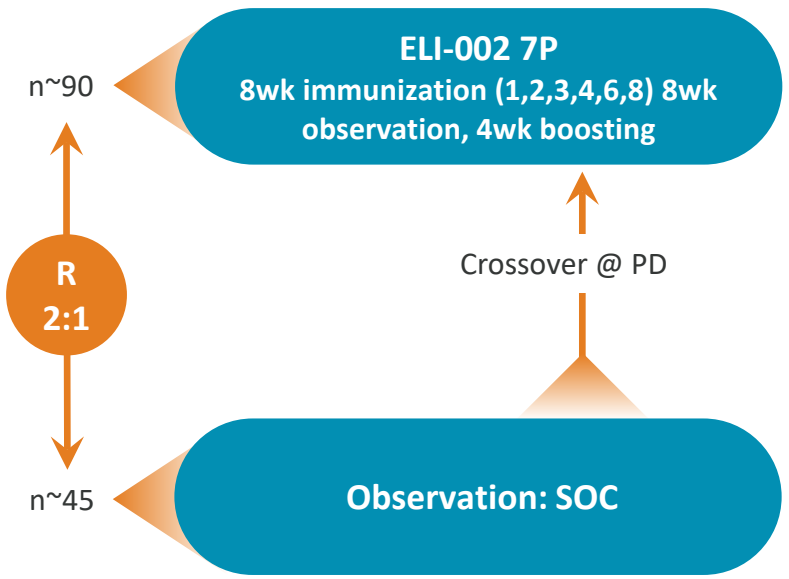
- ✓ mKRAS: Expanded Antigen Coverage

  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



N=144 pts

Endpoints

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

ELI-002 7P Phase 2 trial in PDAC patients: Disease-free survival interim analysis expected in Q3 2025

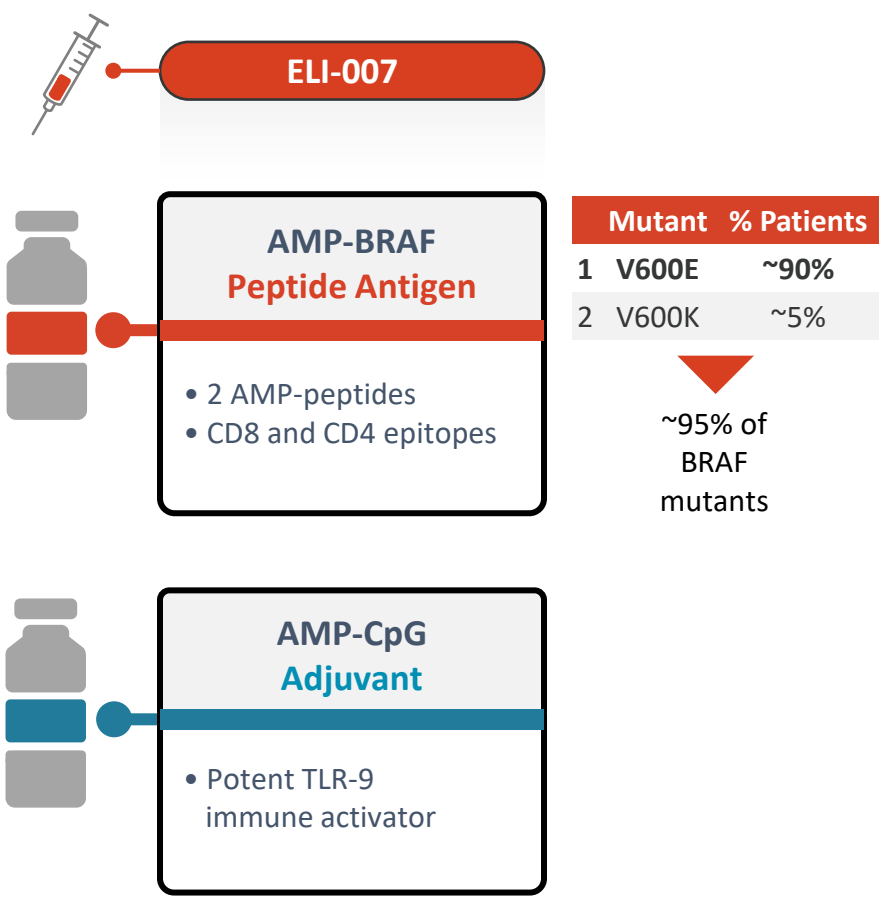
Phase 3 Design aligned in FDA meeting

Randomized, blinded trial; primary endpoint investigator assessed DFS using modified RECIST (new lesions confirmed by biopsy/imaging)

Other Pipeline Assets

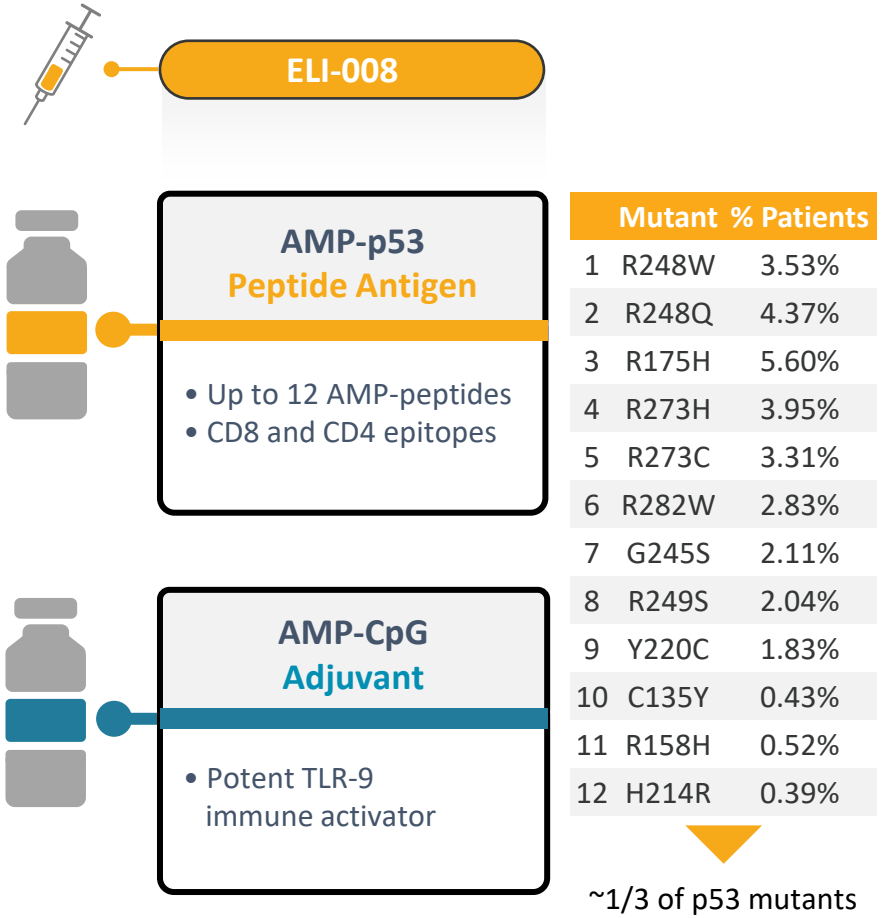
ELI-007 (BRAF) and ELI-008 (p53)

Designing an AMP-Vaccine Targeting Mutant BRAF and p53



	Mutant	% Patients
1	V600E	~90%
2	V600K	~5%

~95% of BRAF mutants



	Mutant	% Patients
1	R248W	3.53%
2	R248Q	4.37%
3	R175H	5.60%
4	R273H	3.95%
5	R273C	3.31%
6	R282W	2.83%
7	G245S	2.11%
8	R249S	2.04%
9	Y220C	1.83%
10	C135Y	0.43%
11	R158H	0.52%
12	H214R	0.39%

~1/3 of p53 mutants

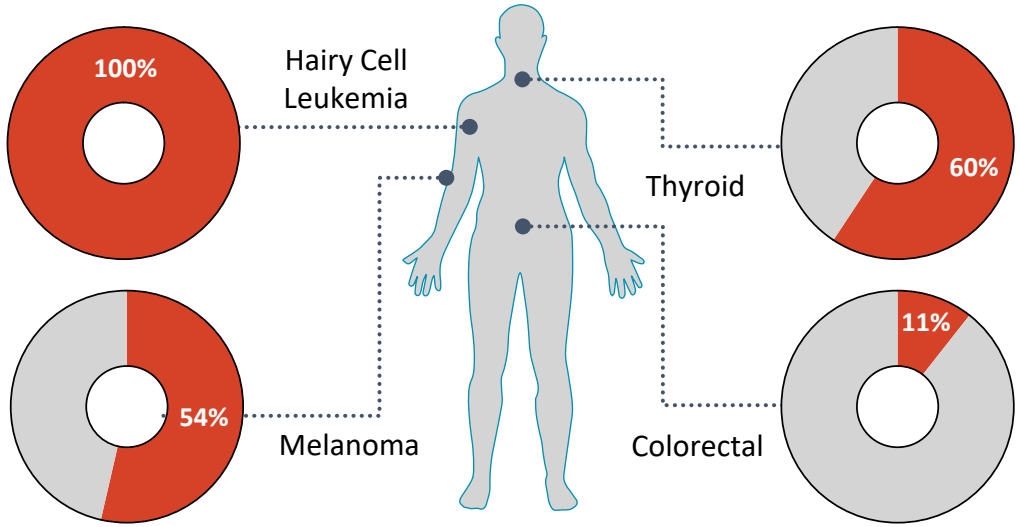
COSMIC Database. 2024; Baugh EH, **Cell Death Differ**. 2018;

ELI-007: Why Target mutant BRAF with Vaccine Therapy?

Mutant BRAF is a Common Cancer Driver

- **Prevalent** among numerous tumor types
- Overall **poor clinical prognosis**
- **Limited therapeutic options**

Most Frequently BRAF-Mutated Cancer Types



Mutant BRAF is a Promising Tumor Antigen

- **Truncal**: mutations occur early, expressed uniformly in tumor cells
- **Driver**: mBRAF signaling is required for tumor growth and survival
- **Highly prevalent**: frequently mutated across tumor types
- **Public neoantigen**: not centrally tolerized, cognate TCRs present in naïve repertoire
- **Broad HLA presentation**: potential off-the-shelf use in diverse patient population
- **Demonstrated Clinical MOA**: mBRAF-specific T cells known to mediate anti-tumor efficacy
- **Multi-targeting potential**: broad recognition of mBRAF variants to prevent escape

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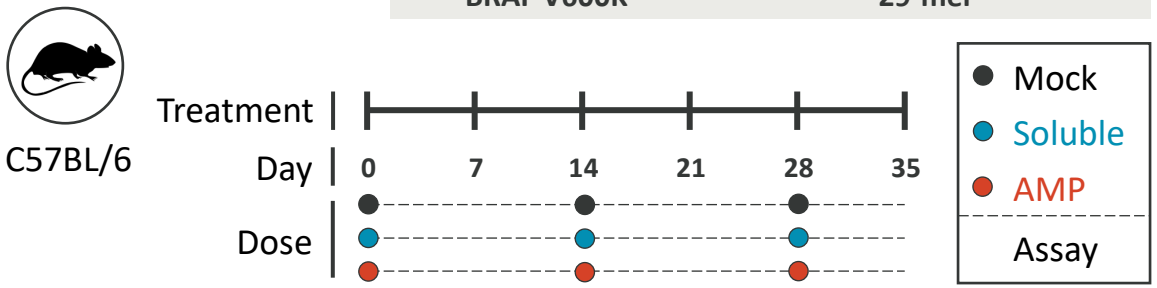
COSMIC Database. 2024; Ahmadzadeh A, **Oncol Rev**. 2014; Agrawal N, **Cell**. 2014; Ihle MA, **BMC Cancer**. 2014; Chu J, **Clin Cancer Res**. 2020

ELI-007 Is a Lymph Node Targeted mBRAF Vaccine

AMP-modification enhances BRAF V600E-specific T cell responses

Experimental Schema:

Mutant	Peptide
BRAF V600E	29-mer
BRAF V600K	29-mer

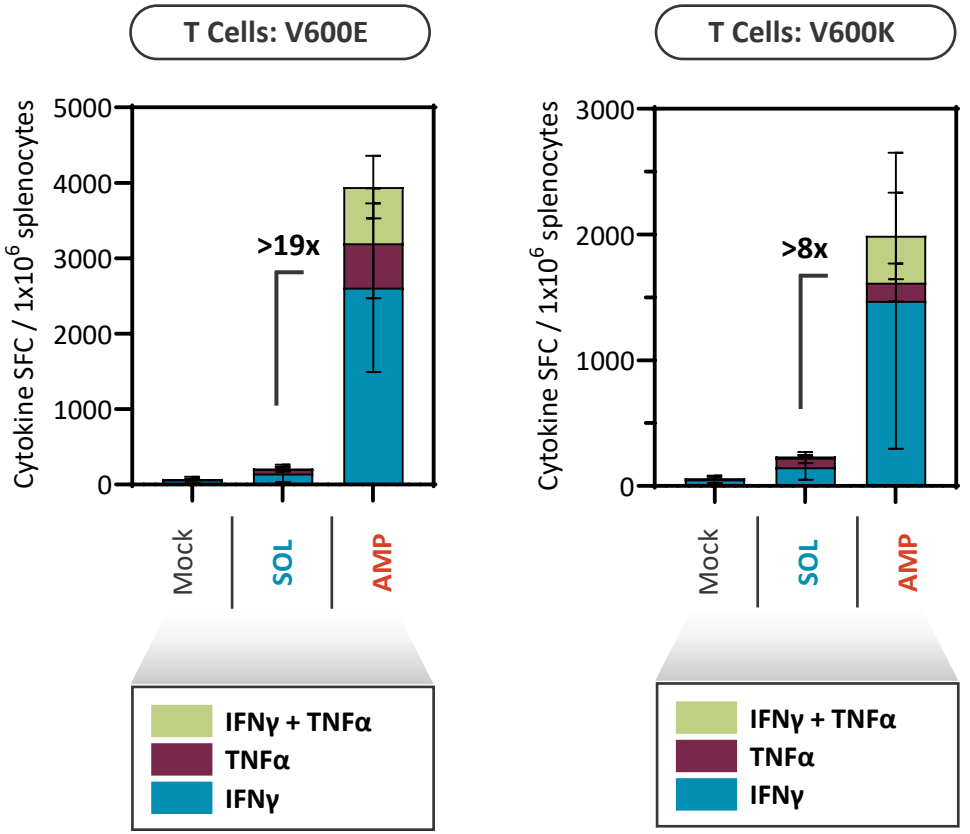


AMP Generates Potent Polyfunctional T Cell Response

- Soluble peptide + CpG is completely inactive
- AMP-vaccine generates potent functional T cell response
- Responses are specific to V600E and V600K mutations
- T cells exhibit polyfunctional effector phenotype
- Cytolytic granzyme production

T Cell Response: BRAF V600 Spleen Day 35

Stim: V600E or V600K Peptide Pool

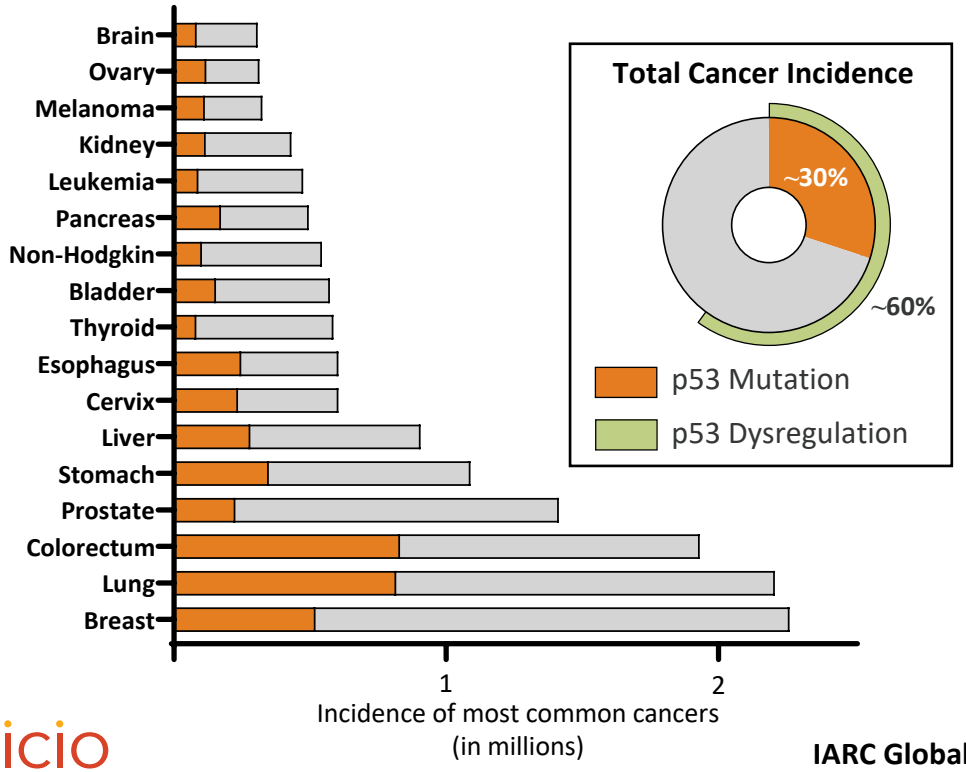


ELI-008: Why Target mutant p53 with Vaccine Therapy?

Mutant p53 Drives ~30% of Human Cancers

- **Prevalent** among numerous tumor types
- **Limited therapeutic options**

Estimated Worldwide Annual Incidence



Mutant p53 is a Promising Tumor Antigen

- **Truncal**: mutations occur early, expressed uniformly in tumor cells
- **Driver**: mp53 signaling is required for tumor growth and survival
- **Highly prevalent**: frequently mutated across tumor types
- **Public neoantigen**: not centrally tolerized, cognate TCRs present in naïve repertoire
- **Broad HLA presentation**: potential off-the-shelf use in diverse patient population
- **Demonstrated Clinical MOA**: mp53-specific T cells known to mediate anti-tumor efficacy
- **Multi-targeting potential**: broad recognition of mp53 variants to prevent escape

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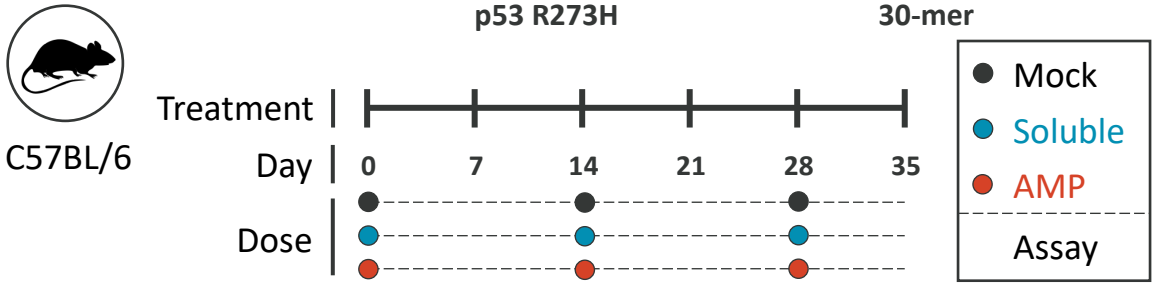
IARC Global Cancer Observatory. gco.iarc.fr. 2023; **The TP53 Database**. tp53.isb-cgc.org. 2023

ELI-008 Is a Lymph Node Targeted mp53 Vaccine

AMP-modification enhances p53 hot-spot mutant-specific T cell responses

Experimental Schema:

Mutant	Peptide
p53 R248Q	30-mer
p53 R20175H	30-mer
p53 R273H	30-mer

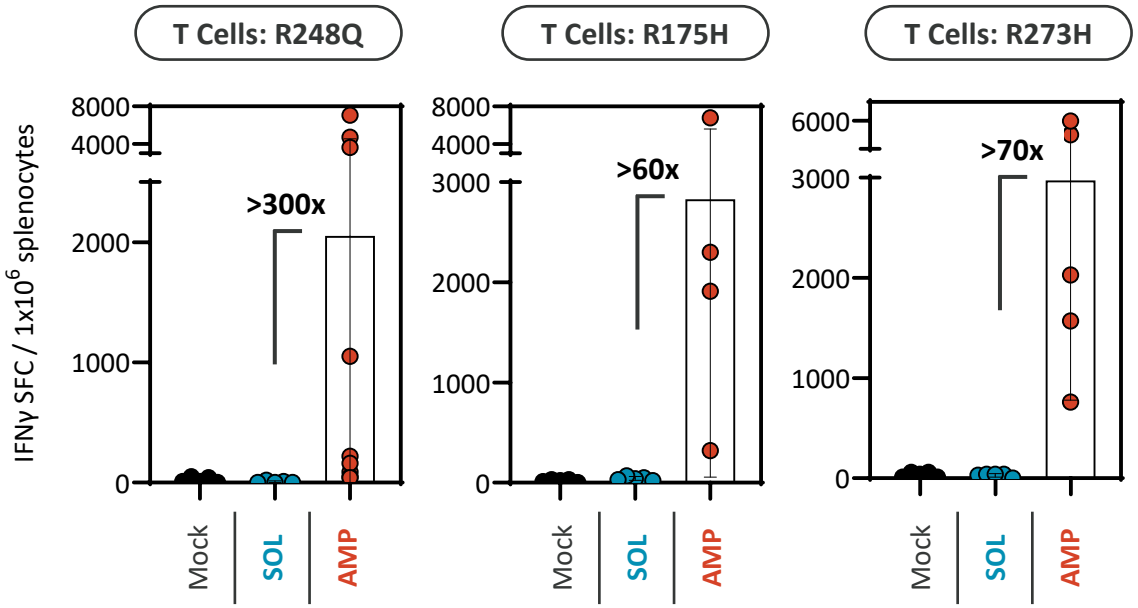


AMP Generates Potent Polyfunctional T Cell Response

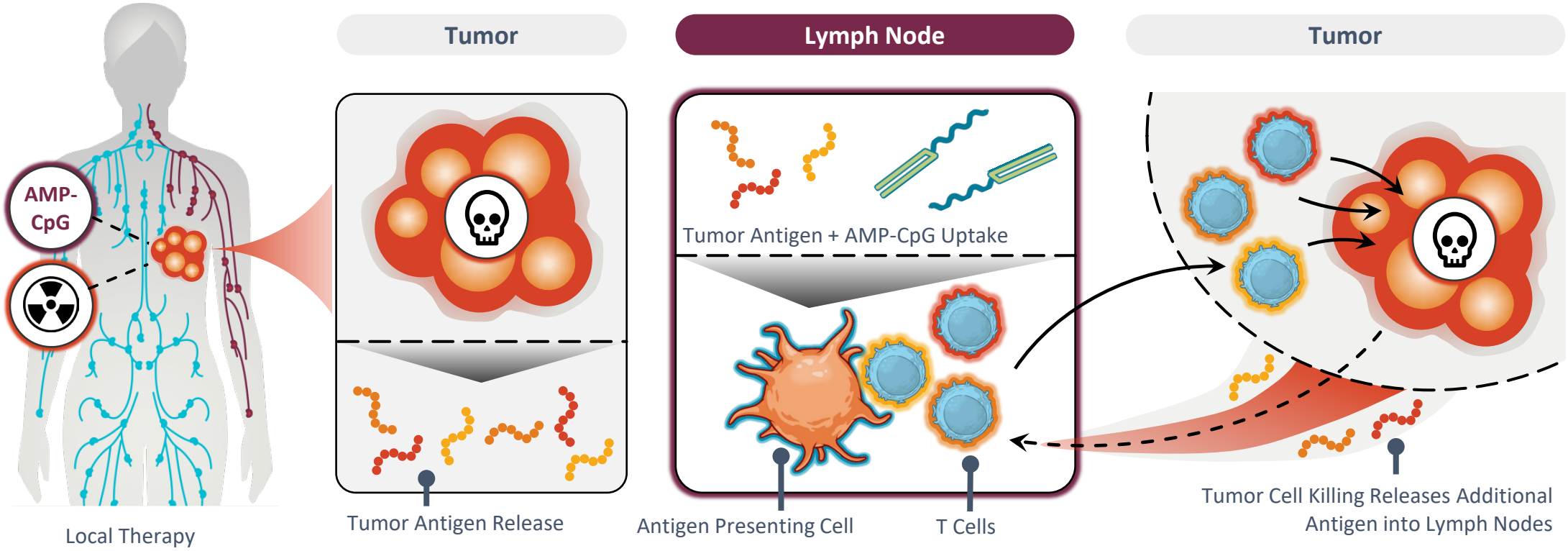
- Soluble peptide + CpG is inactive
- AMP-vaccine generates potent functional T cell response
- Polyfunctional effector cytokine secretion
- Cytolytic granzyme production

T Cell Response: p53 Spleen Day 35

Stim: p53 R248Q, R175H, or R273H OLPs



ELI-004: AMP-CpG Combination with Radiation to Induce Tumor-specific Immunity *In Situ*



1 Radiation + AMP-CpG

1. Debulk tumor
2. Activate tumor-resident immune cells
3. Release tumor antigens into local lymph nodes

2 Tumor Cell Death

3 Immune Activation + T Cell Induction

1. Concurrent delivery of tumor antigen and adjuvant to lymph nodes
2. Tumor-specific T cell activation

4 T Cell Infiltration and Elimination of Tumor

1. T cells infiltrate treated site and distal lesions (abscopal effect)
2. Reinforcing cycle of tumor killing and further support of immune therapy

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Key Milestones Achieved and Growth Initiatives for 2025

ELI-002 Clinical Development

- ✓ Preliminary Phase 1 T Cell and biomarker response (ASCO)
- ✓ T cell and Antigen Spreading (SITC)
- ✓ Complete Phase 2 enrollment with 144 patients enrolled (4Q 2024)
- ✓ FDA Type B Meeting (4Q 2024)
- ❑ Phase 2 DFS Interim Analysis (expected Q3 2025)
- ❑ End of Phase 2 FDA Meeting (expected H2 2025)

2025 Growth Initiatives

- ❑ Initiate ELI-002 + CPI investigator-sponsored clinical combination study in neo-adjuvant PDAC and potential other ELI-002 combination studies
- ❑ Select Sponsor and potentially initiate ELI-004 + radiation investigator-sponsored clinical study, subject to funding
- ❑ Select Sponsor and potentially initiate ELI-002 + CPI investigator-sponsored clinical study in metastatic micro-satellite stable CRC, subject to funding
- ❑ Advance ELI-007 BRAF and ELI-008 p53 vaccines for Phase 1 readiness
- ❑ Finalize Phase 3 trial protocol in adjuvant PDAC setting for ELI-002

Investment Highlights

Clinical-stage biotech developing novel lymph node-targeted “off the shelf” cancer immunotherapies

Leveraging proprietary Amphiphile (“AMP”) Technology

- Our proprietary AMP platform is designed to generate robust, functional, and durable immune responses by targeting lymph nodes
- AMP technology delivers antigen-specific payloads directly to lymph nodes to educate, activate, and expand tumor-eliminating T cell populations and has the potential for broad applicability across cancer immunotherapy
- Proof-of-concept has been demonstrated in two completed Phase 1 trials; a randomized Phase 2 study is expected to read out this year

ELI-002
Lymph Node Targeted mKRAS Cancer Vaccine

- Off-the-shelf cancer vaccine candidate targeting the most common KRAS mutations that drive 25% of solid tumors
- Potential monotherapy adjuvant treatment in high relapse-risk mKRAS^{+} cancers: pancreatic (PDAC), colorectal (CRC)
- Completed two Phase 1 trials and currently in randomized Phase 2 trial in PDAC:
  - ELI-002 2P elicited mKRAS-specific T cell response **~100x** increased over baseline at the Phase 2 dose without any DLTs or SAEs
  - ELI-002 2P Update at ESMO-IO: full cohort (n=25) mOS of **28.9 months**; **16.3 months** mRFS

Value-creating catalysts and capitalization

- ELI-002 7P Phase 2 trial: Disease-free survival interim analysis expected in Q3 2025
- ELI-002 7P End of Phase 2 FDA meeting expected in H2 2025
- Investigator-sponsored trials of ELI-002 + checkpoint inhibitors (CPI) in PDAC and CRC and other combinations
- Cash runway expected to support operations into Q4 2025 beyond anticipated Phase 2 interim data analysis in Q3 2025

Appendix

H2 2024 Execution

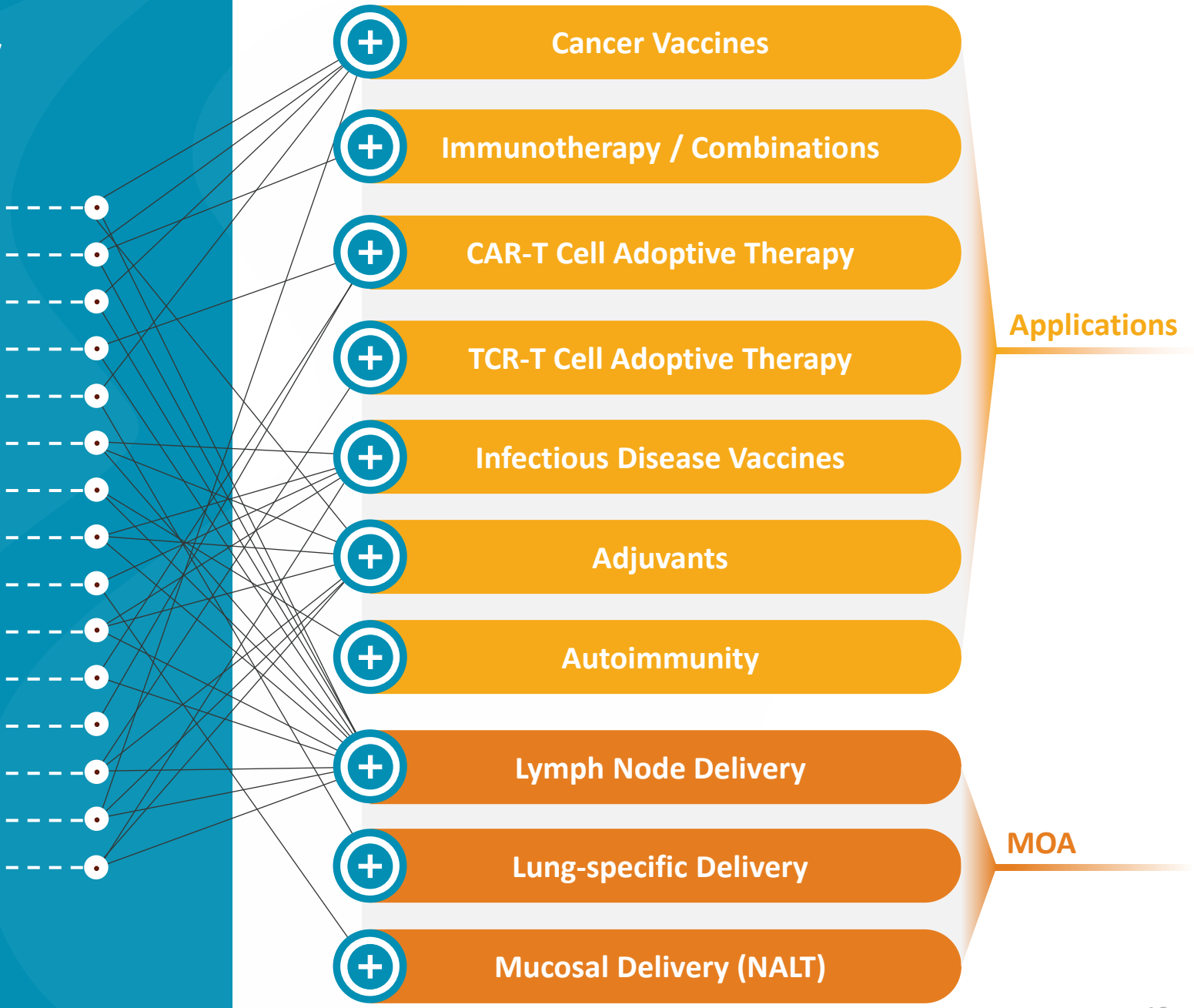
Phase 2 trial fully enrolled – 144 patients enrolled in 11 months in 27 clinical sites

- $33M in new capital raised – expected to fund the Company into Q2 2025 beyond the anticipated Phase 2 interim analysis
- ELI-002 7P updated Phase 1a data presented at AACR-Special Conference-Pancreatic Cancer
- ELI-002 7P updated Phase 1a data presented at SITC
- ELI-002 Phase 3 trial study design alignment from November FDA Type-B meeting
- ELI-002 2P updated Phase 1a data presented at ESMO-IO

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
2023	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
2024	Steinbuck	BioRxIV



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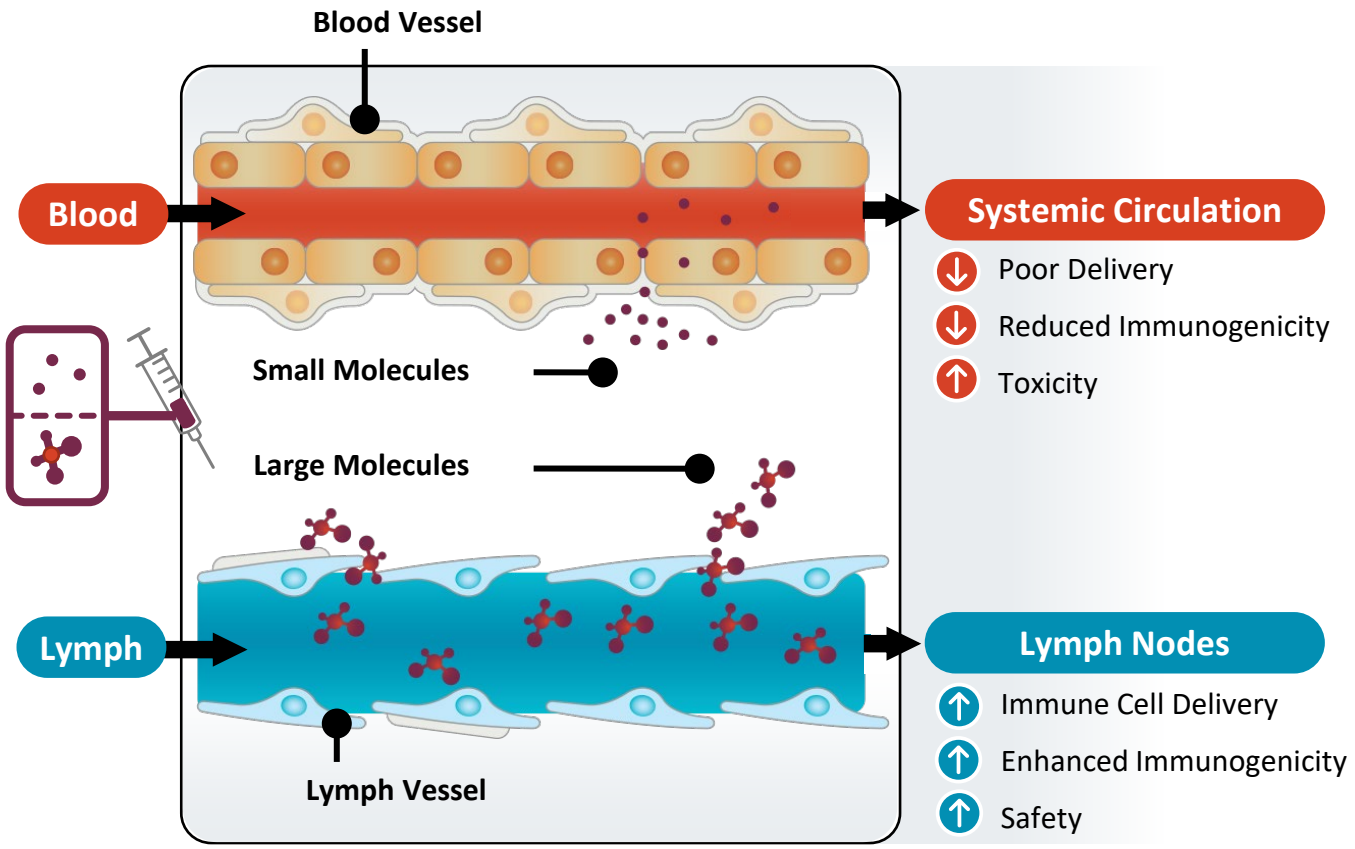
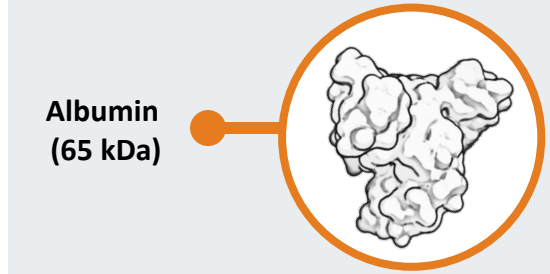
“Albumin-hitchhiking” Reprograms Delivery of Vaccines and Immunotherapy to Target Lymph Nodes

Tissue Injection Site: Molecular Size Determines Distribution



Albumin-based Delivery to Lymph Nodes



Binding to Tissue Albumin

Enhanced Lymph Node Delivery

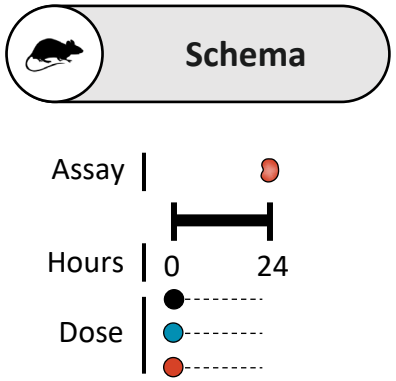
T cell Response Magnitude / Function

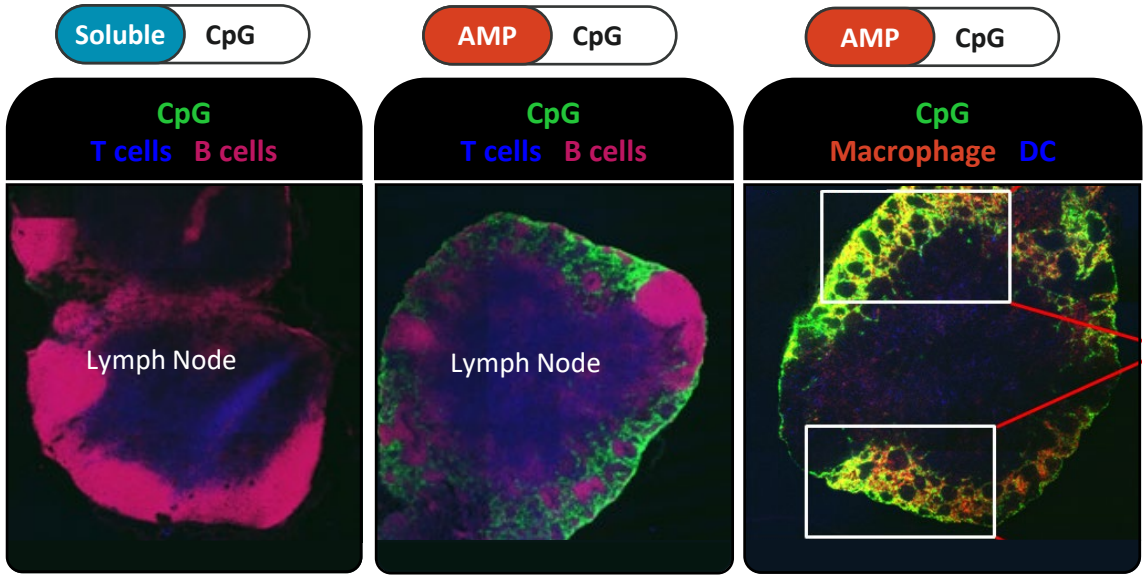
Liu, Irvine, et al. **Nature** 2014; Trevaskis, et al. **Nat Rev Drug Discovery** 2015; Yousefpour, et al. **Nat Rev Bioengineering** 2023

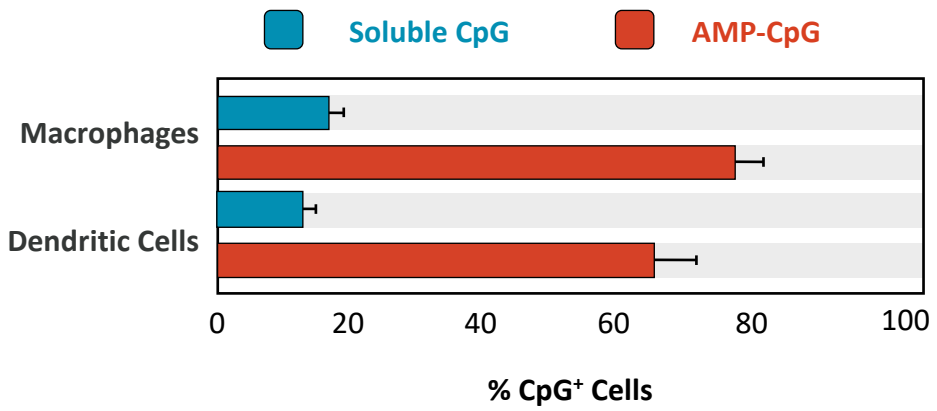
Designing a system to target vaccines to Lymph Nodes:

The AMP Platform Technology

AMP-vaccines Target the Lymph Nodes for Efficient Uptake by Resident Antigen Presenting Cells







Lymph Node Delivery

- Lymph Node Uptake
- Lymph Node Retention
- Colocalization with APCs

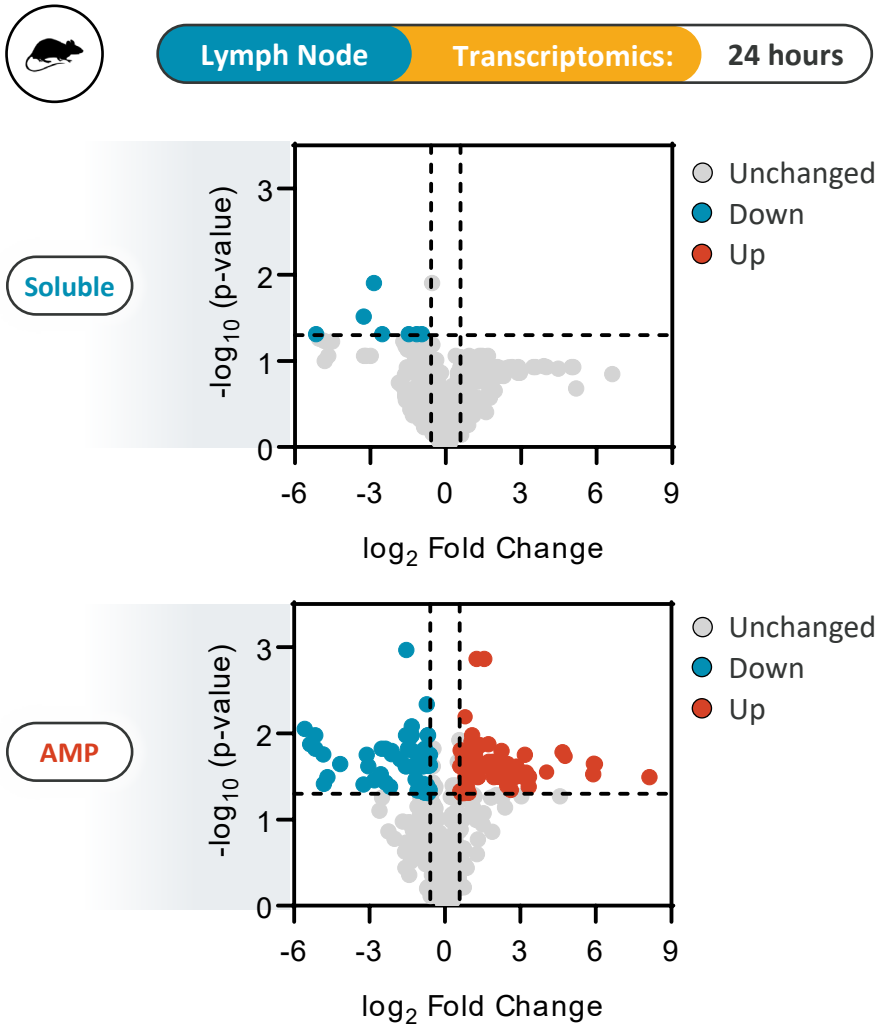
Uptake in APCs

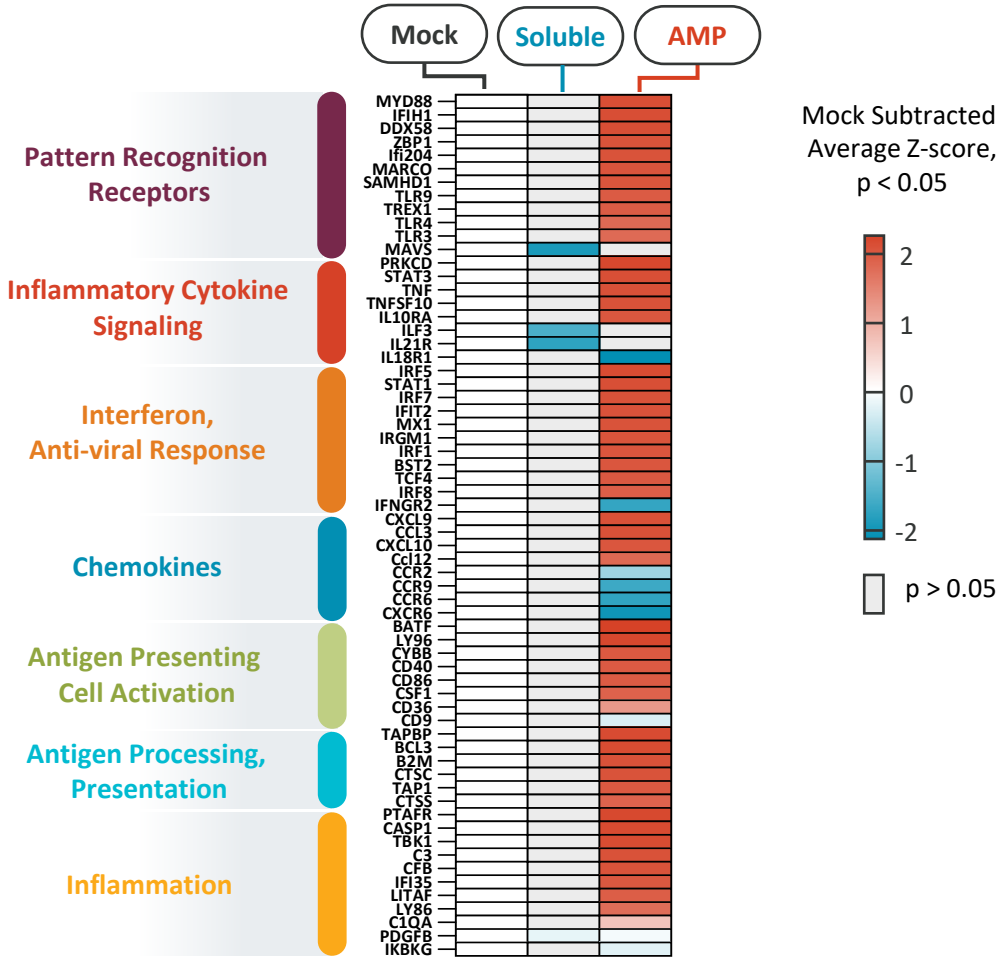
- >75% Macrophage Uptake
- >60% Dendritic Cell Uptake

Liu, Irvine, et al. **Nature** 2014

AMP-vaccination Induces Coordinated Immune Activation in Lymph Nodes

Comprehensive inflammatory transcriptional reprogramming

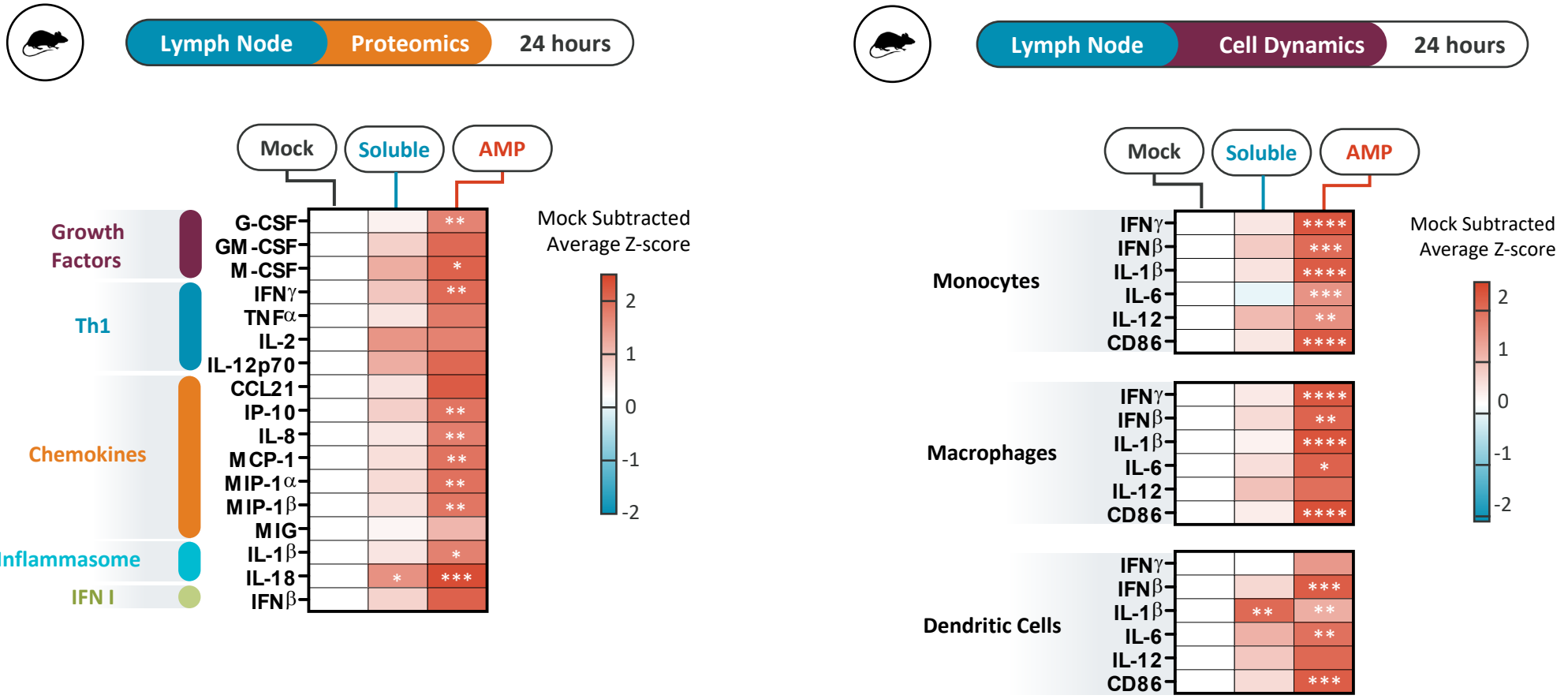




Steinbuck, DeMuth, et al. **Science Advances**. 2021; Seenappa, DeMuth, et al. **NJP Vaccines**. 2022

AMP-vaccination Induces Coordinated Immune Activation in Lymph Nodes

Inflammatory proteomic signatures and coordinated innate cell recruitment and activation



Steinbuck, DeMuth, et al. **Science Advances**. 2021; Seenappa, DeMuth, et al. **NJP Vaccines**. 2022

AMP-vaccination Induces Coordinated Immune Activation in Lymph Nodes

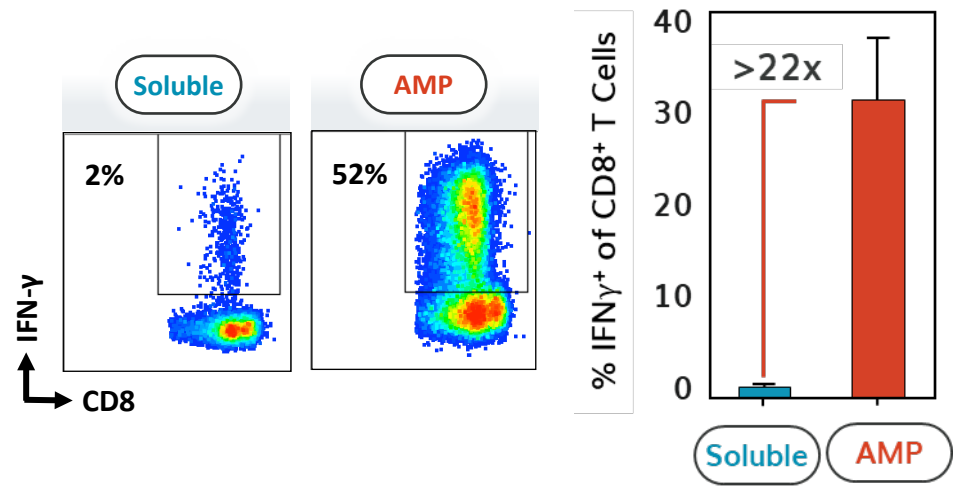
Enhanced antigen-specific T cell response magnitude and functional quality

AMP Vaccine Platform

Enhanced Lymph Node Delivery and Retention

- Increased Uptake into APCs
- Potent APC Activation
- Inflammatory Transcriptional Programming
- Robust Cytokine/Chemokine Milieu

Blood | **IFNγ⁺ T Cell Response** | **gp100 Antigen**



Lymph Node Delivery → **T Cell Response**

- **Expansion**
- **Persistence**
- **Effector function**
- **Phenotype**
- **Metabolic function**

Liu, Irvine, et al. **Nature** 2014; Moynihan, Irvine, et al. **Nature Medicine** 2016