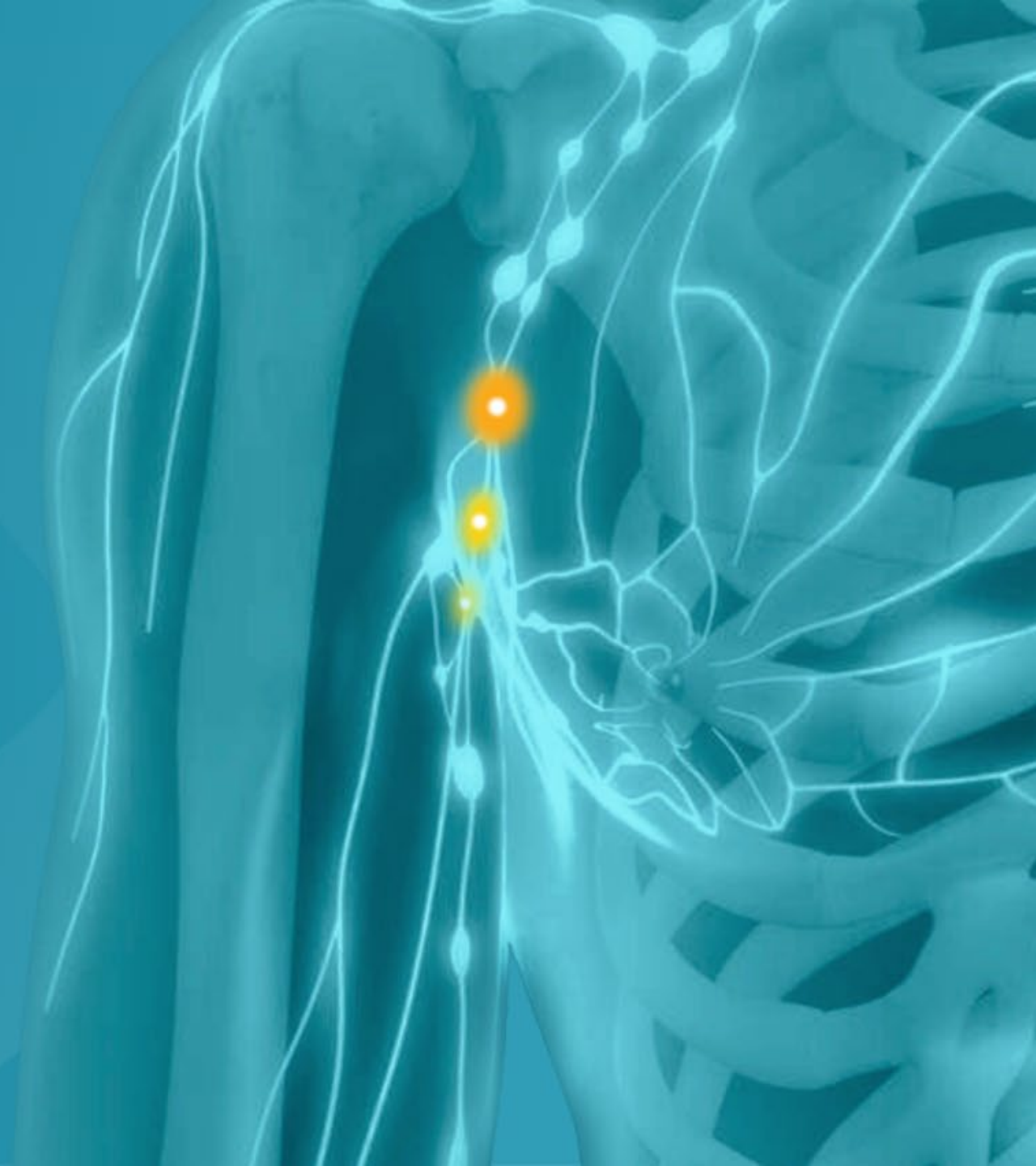




Targeting the Lymph Nodes to AMPLify Immunotherapy

Nasdaq: ELTX

January 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 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 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, including our cash runway funding the Company beyond the anticipated Phase 2 interim analysis; 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 and Checkpoint Inhibitor investigator-sponsored clinical study in metastatic micro-satellite stable CRC in 2025, the initiation of an ELI-002 and Checkpoint Inhibitor investigator-sponsored clinical combination study in neo-adjuvant pancreatic ductal adenocarcinoma (“PDAC”) in 2025, the initiation of an ELI-004 with radiation investigator-sponsored clinical study in 2025-2026, and our anticipated finalization of a Phase 3 trial protocol in the adjuvant PDAC setting; our ability to advance ELI-007 and/or ELI-008 for Phase 1 readiness; the timing of the availability of data from our clinical trials, including the DFS interim analysis anticipated from the Phase 2 trial of ELI-002 7P anticipated in the first half of 2025; 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; the occurrence and timing of an end of Phase 2 meeting for the ELI-002 7P trial with the FDA anticipated in the second half of 2025; 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 PDAC 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 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.

Company Overview

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

Amphiphile (“AMP”) Technology

- AMP delivers payloads to lymph nodes eliciting immune responses with increased magnitude, function, and durability
- Designed to use the lymph nodes to educate, activate and amplify differentiated T cell responses to eliminate tumor cells
- AMP technology offers potential for broad cancer immunotherapy application

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) and colorectal (CRC)
- Driver mutation specificity in combination with T cell responses to personal tumor neoantigens (antigen spreading)

ELI-002 Clinical Data (39 pts, 2 Phase 1 trials)

- Elicited mKRAS specific T cell response ~100x increased over baseline at the Phase 2 dose without any DLTs or SAEs
- Strength of T cell response correlated with tumor biomarker reduction and reduced risk of relapse or death
- ELI-002 2P Update at ESMO-IO: full cohort (n=25) mOS of 28.9 months; 16.3 months mRFS

ELI-002 Next Steps

- ELI-002 7P Phase 2 trial: Disease-free survival interim analysis expected in H1 2025
- ELI-002 7P End of Phase 2 FDA meeting expected in H2 2025
- Investigator-sponsored trials of ELI-002 + CPI in PDAC and CRC

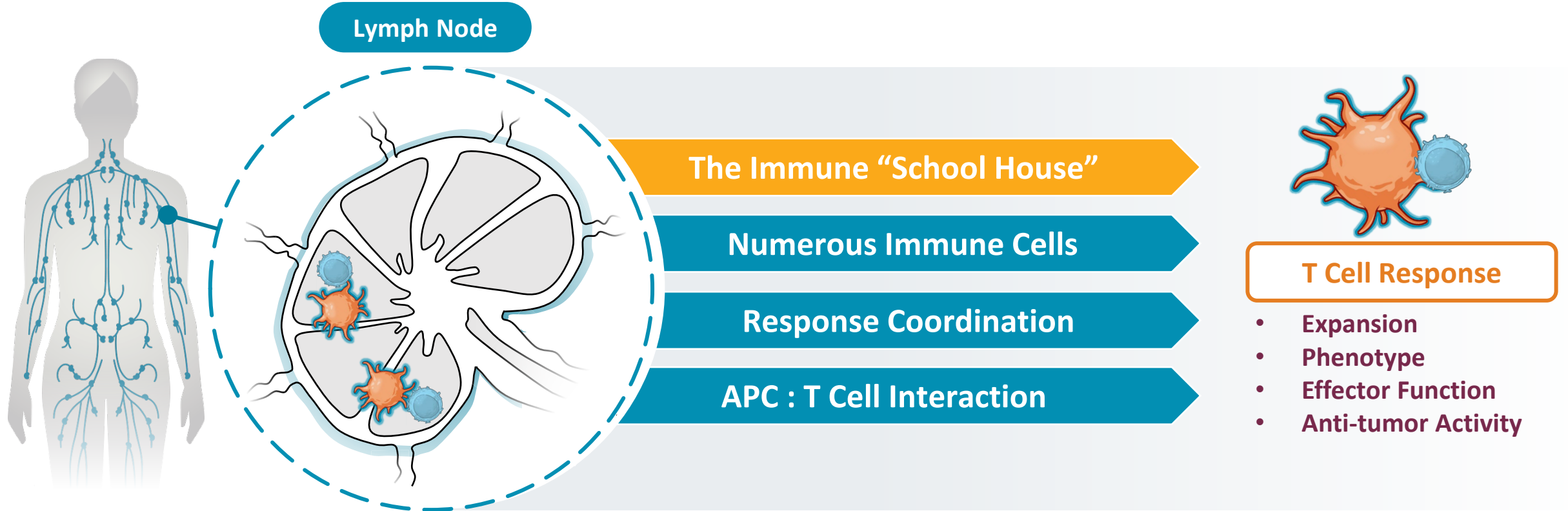
H2 2024 Execution

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

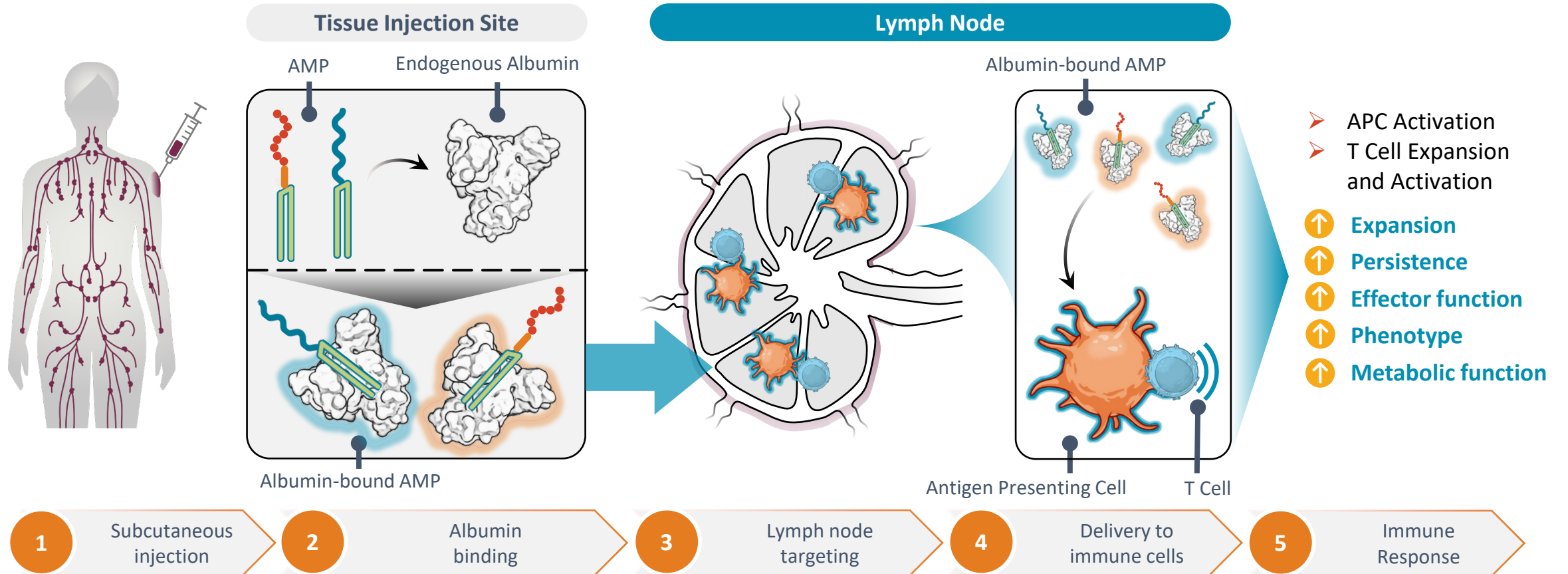
Targeting Lymph Nodes Essential for Eliciting Robust T Cell Responses

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



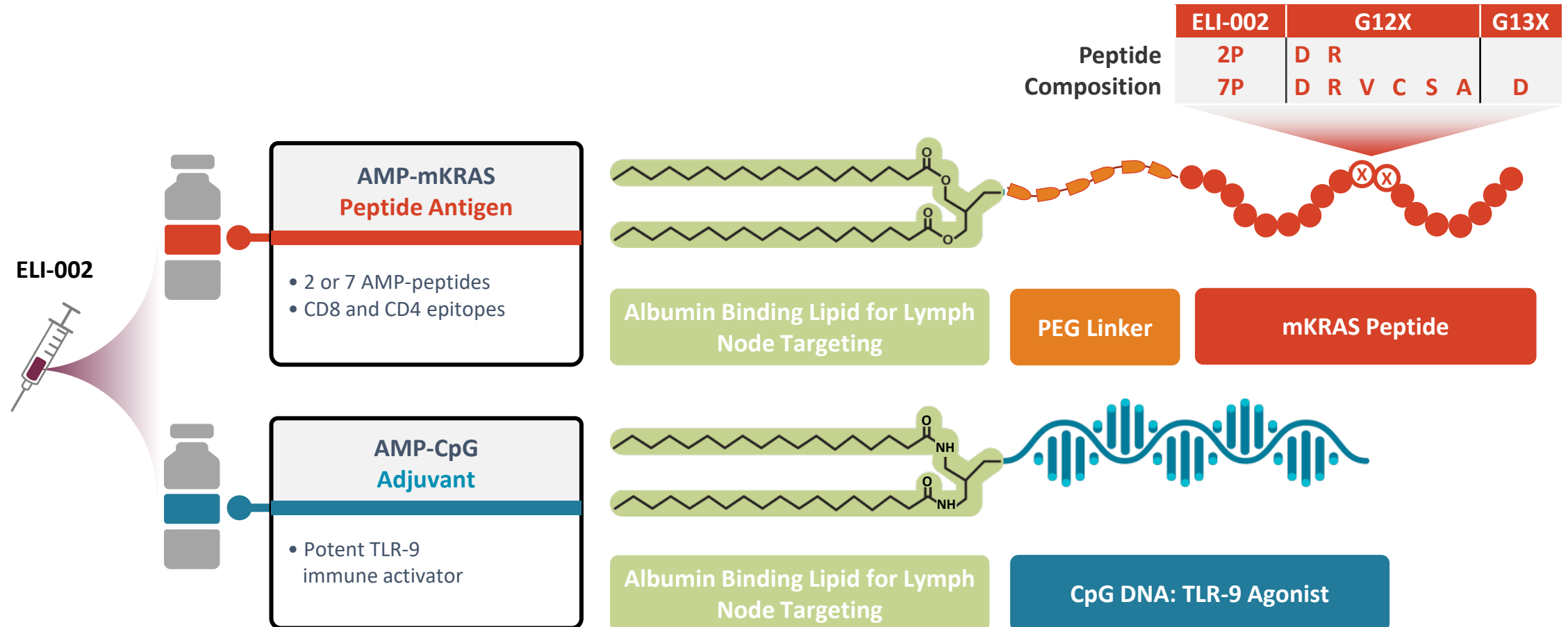
AMP Technology Targets Vaccine Delivery to Lymph Nodes

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



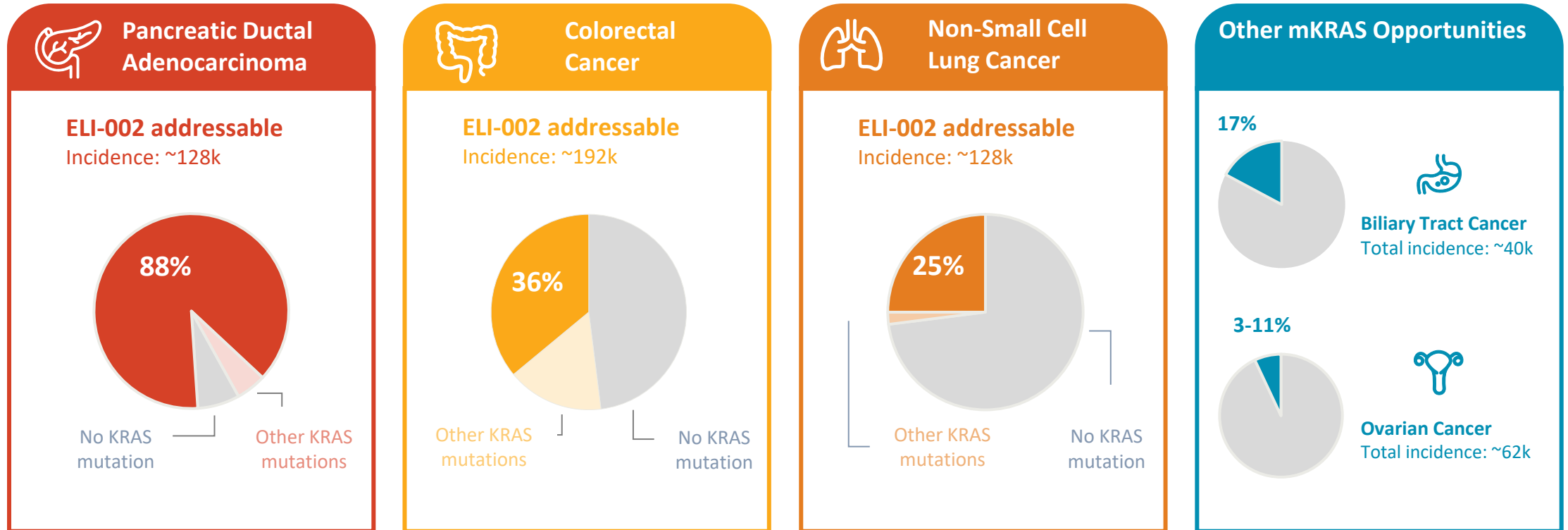
ELI-002 Is a Lymph Node Targeted mKRAS Vaccine

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



The mKRAS Opportunity

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



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

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 Neoantigen Vaccines

- > 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 vs 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

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

ELI-002 2P: Phase 1a Trial of ELI-002 2-Peptide Formulation

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

ELI-002 2P Study Overview

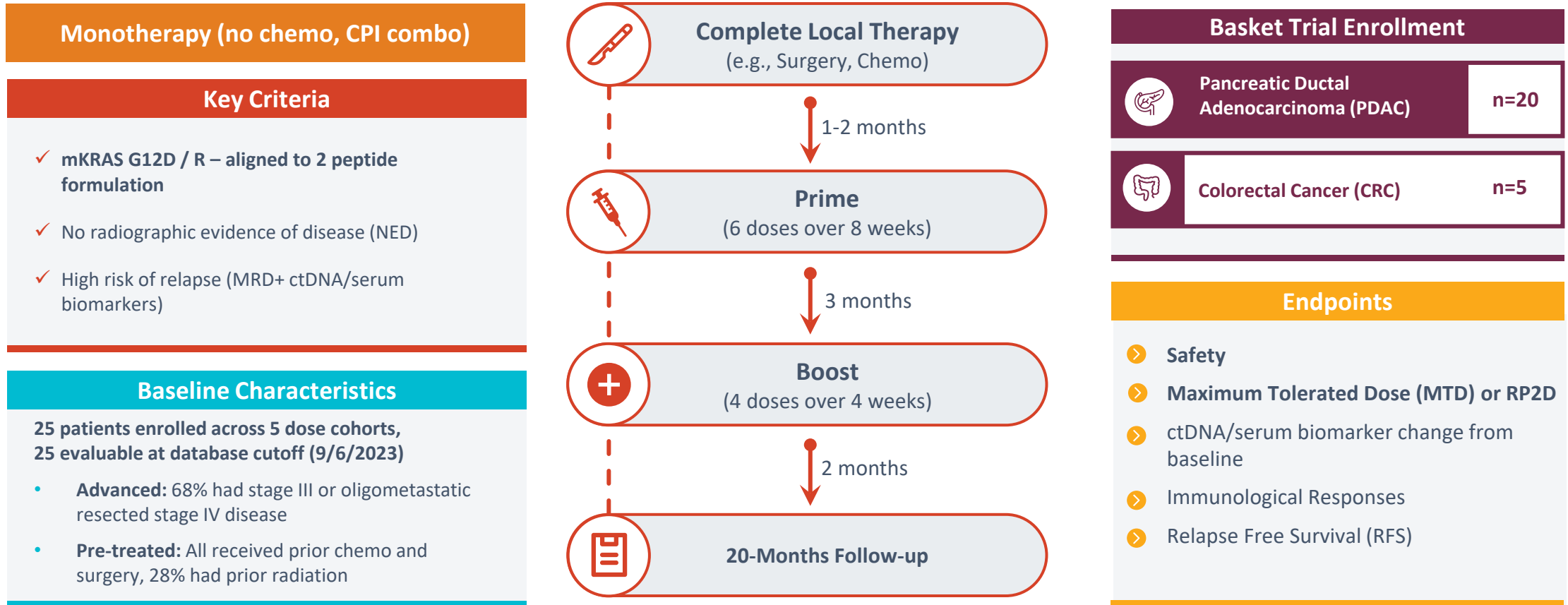
* Published in Nature Medicine

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

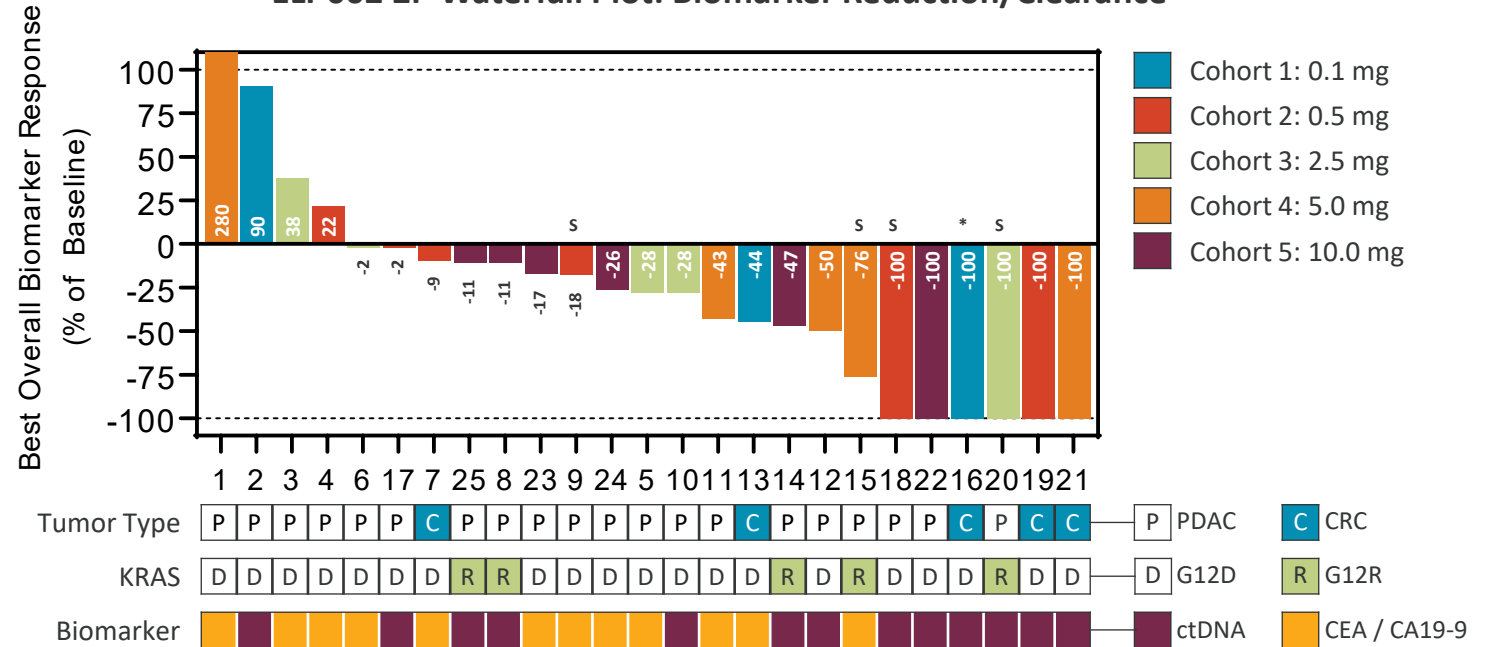


Robust responses observed across tumor types and KRAS mutations with ELI-002 monotherapy

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

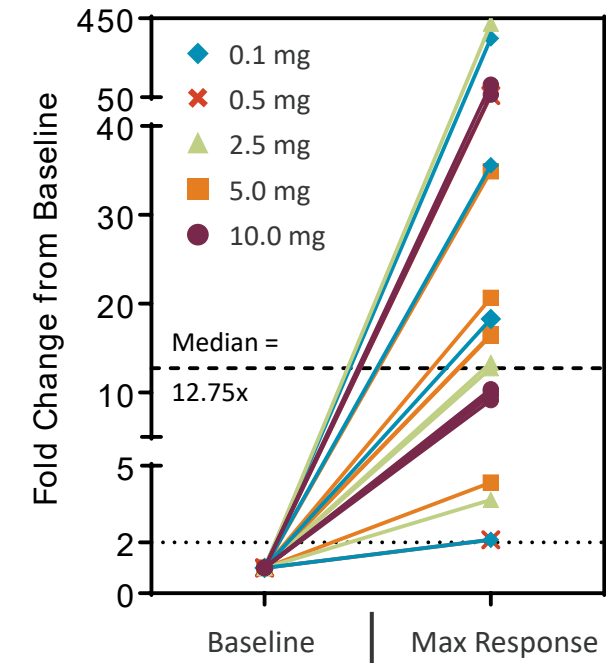
84% of patients generated mKRAS-specific T cells directly *ex vivo*; 100% at RP2D

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

ELI-002 2P T Cell Fold-Changes

Direct Ex Vivo T Cell Response

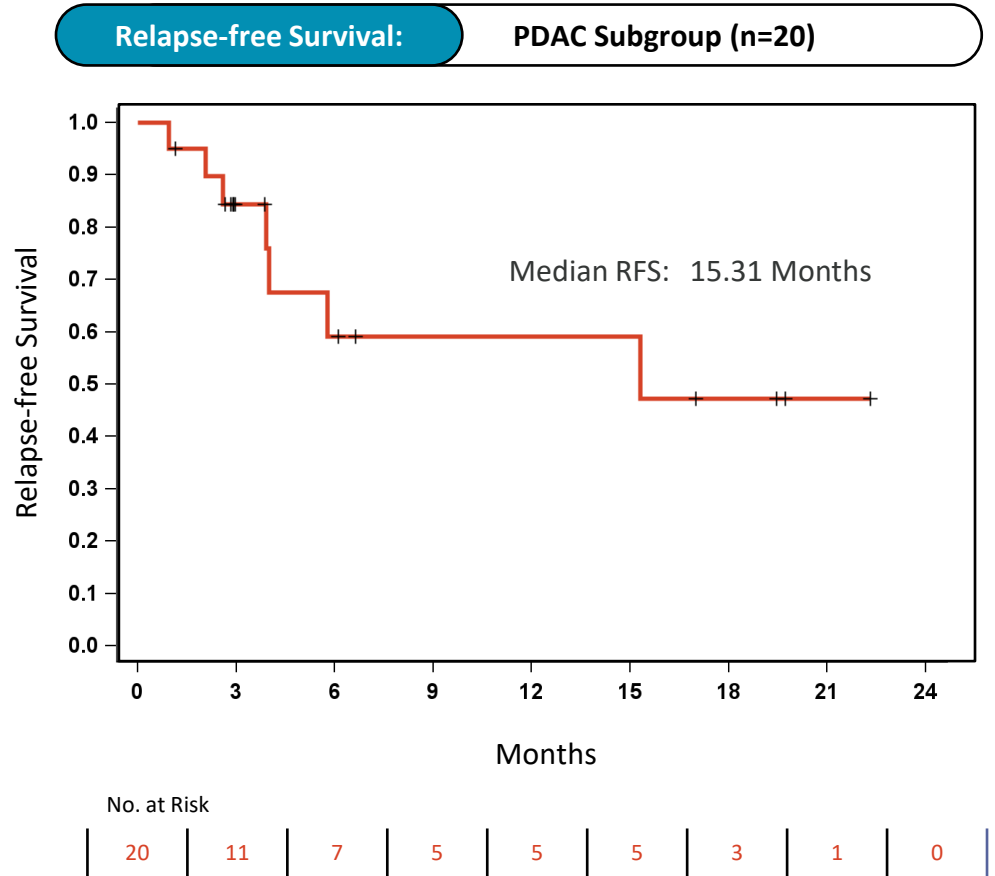
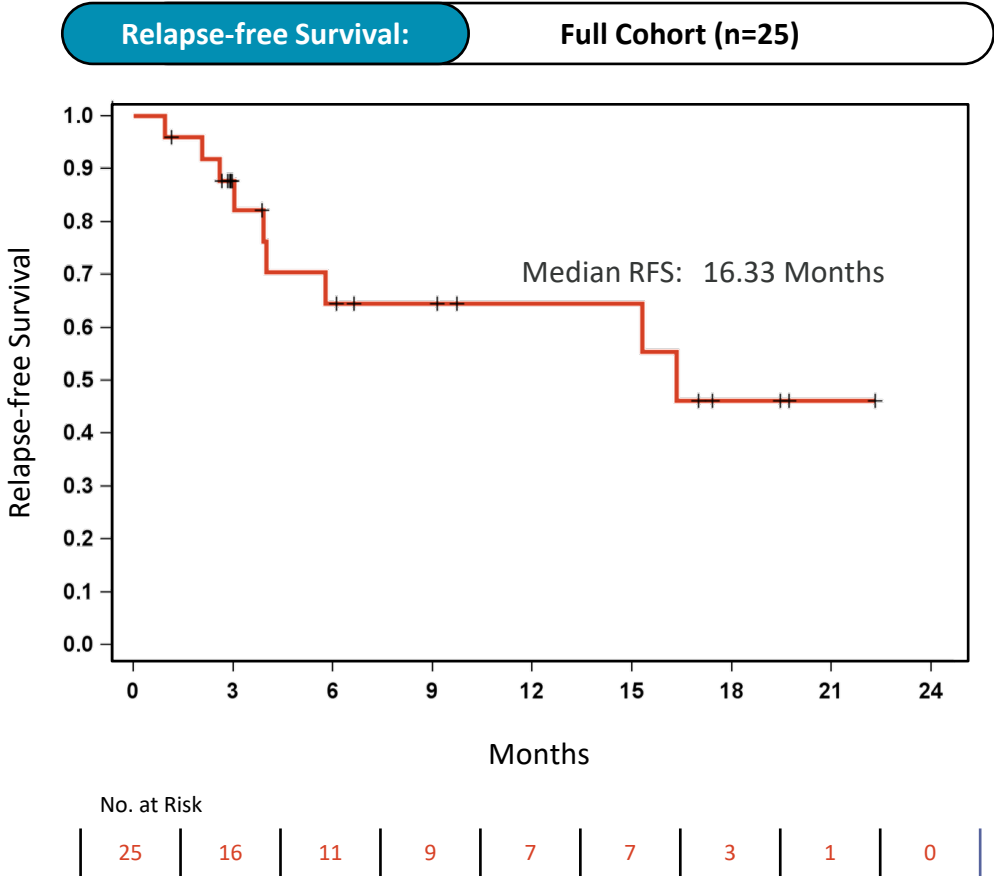


Responses shown are best overall responses vs baseline for each patient at any timepoint during the assessment period.

Data cutoff 6-Sept-23

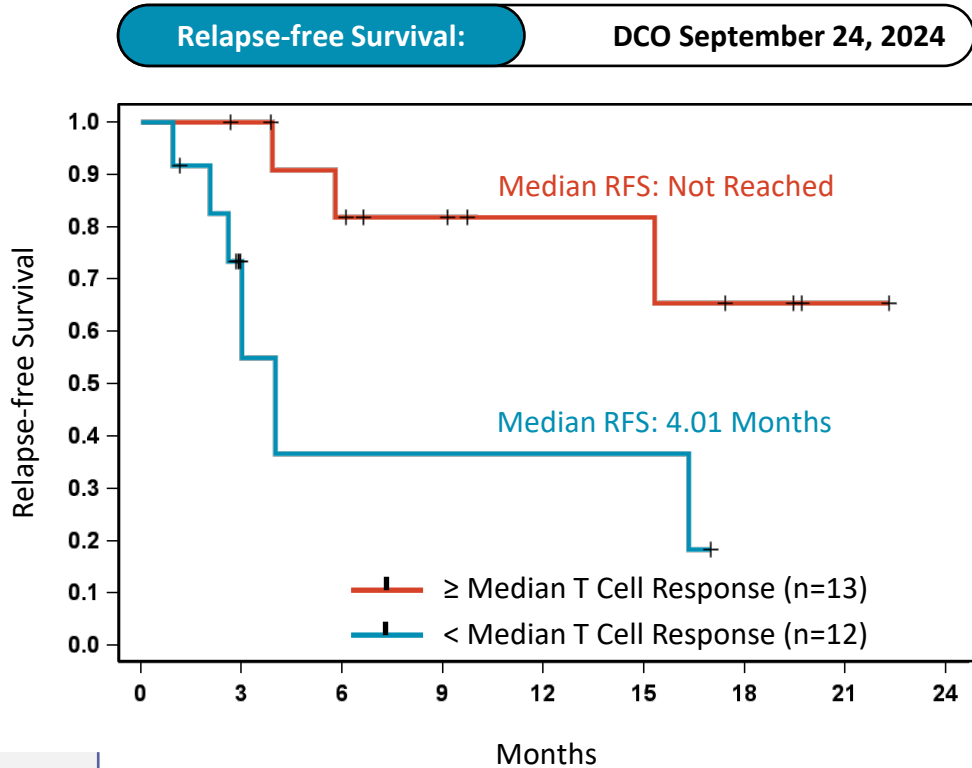
ESMO-IO UPDATE: RFS in Full Cohort vs PDAC Subgroup

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



Median follow-up for study: 19.7 months

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



| 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

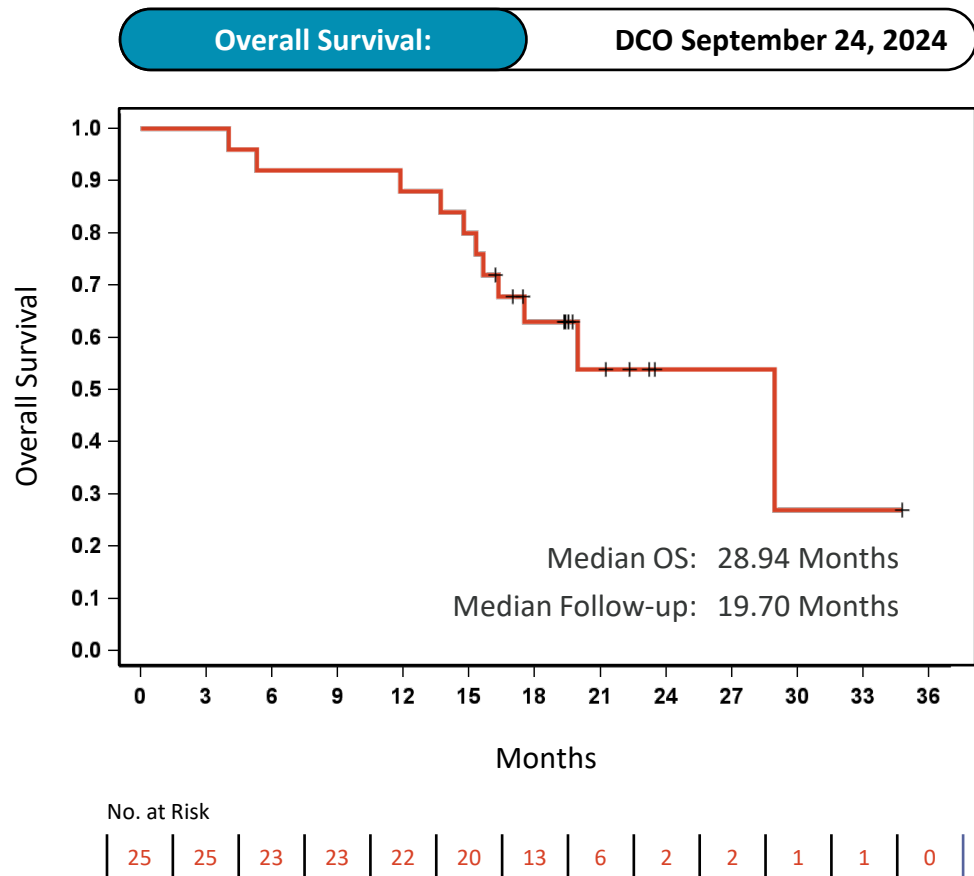
| | DCO | 06-Sept-2023 | 24-Sept-2024 |
|---------------------|------------------------------------|---------------------------|---------------------------|
| Median RFS (Months) | ≥ Median T Cell < Median T Cell | Not Reached 4.01 | Not Reached 4.01 |
| HR (95% CI) | | 0.142 (0.0321, 0.6278) | 0.226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

2024 ESMO-IO Update Shows Early Encouraging Patient Survival

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



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

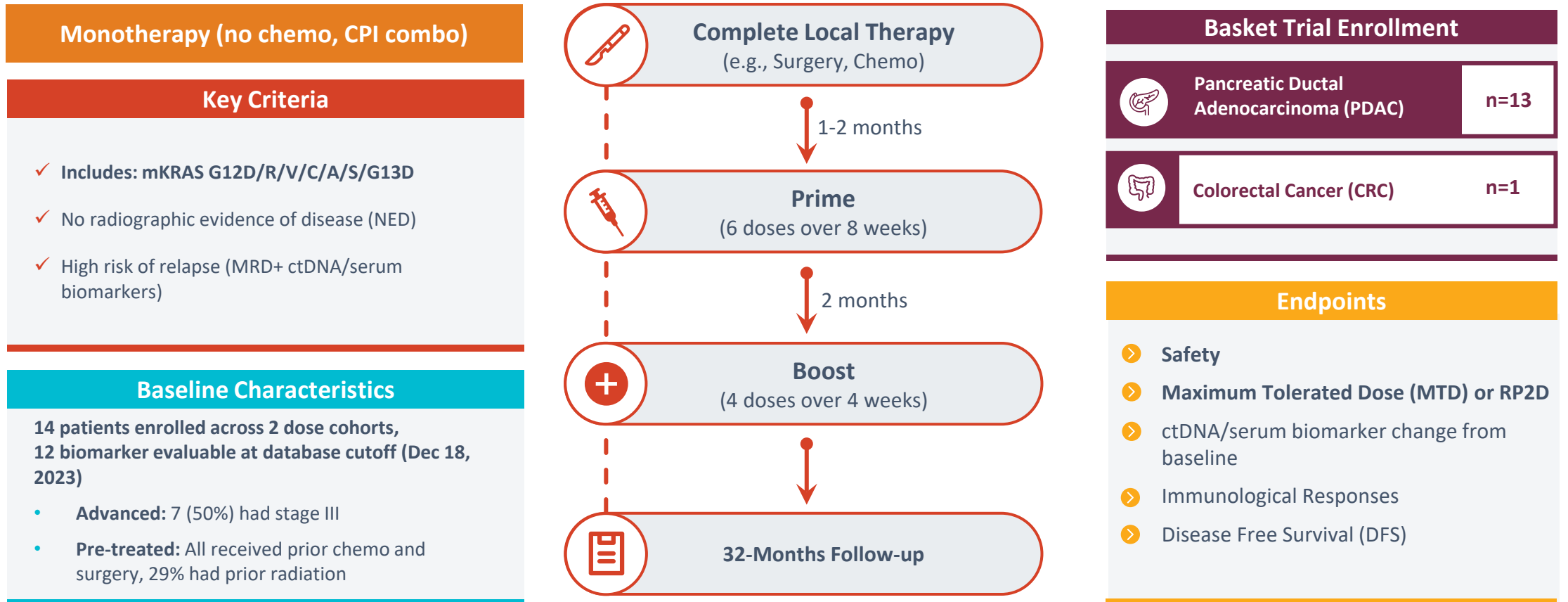
- Median RFS for full cohort and PDAC, CRC subgroups are similar
- Median OS for full cohort and PDAC, CRC subgroups are identical
- mOS longer than MRD+ PDAC e.g. 17 mo from resection, Groot et al., 2019. Clin Cancer Res 25:4973

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

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

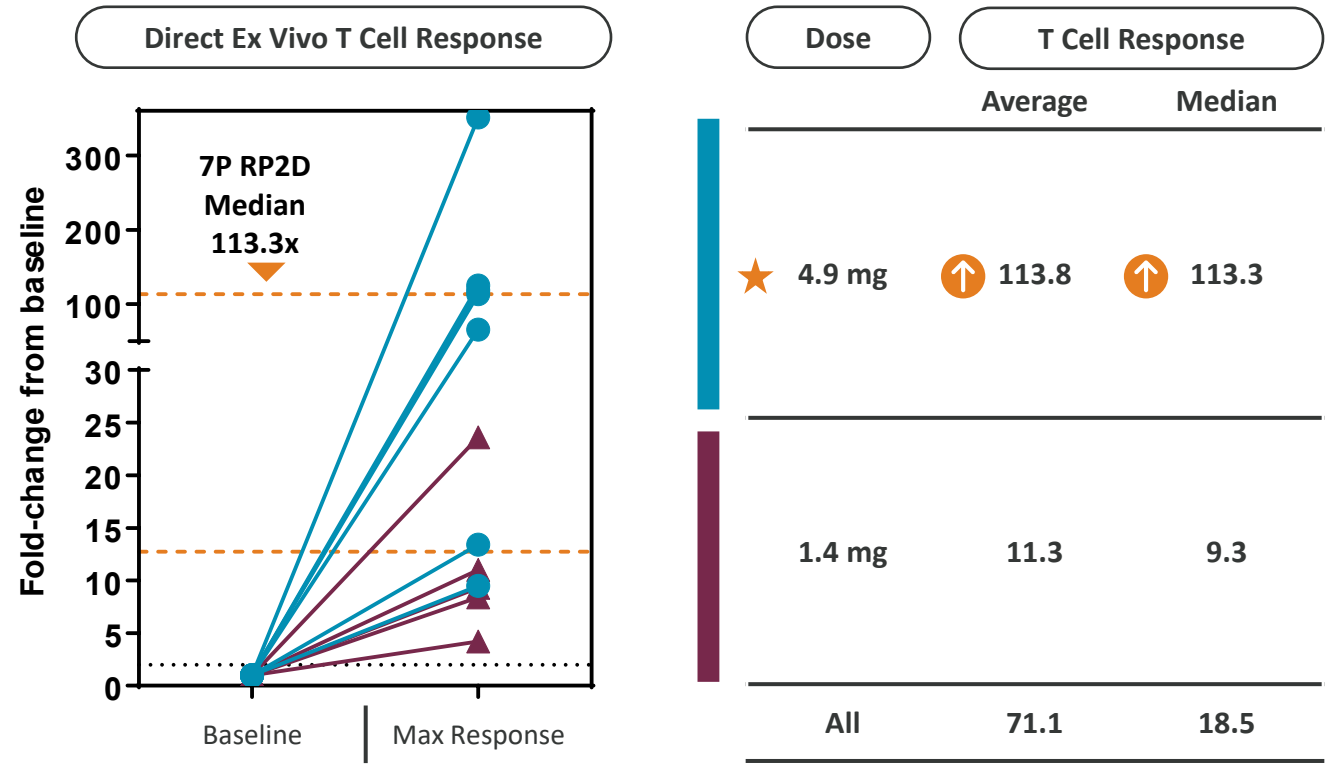


100% of patients with robust T cell response

mKRAS T Cell Responses

- 100% of patients showed T cell responses
- 4.9 mg dose group selected for Phase 2
 - Median fold change = **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

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% |

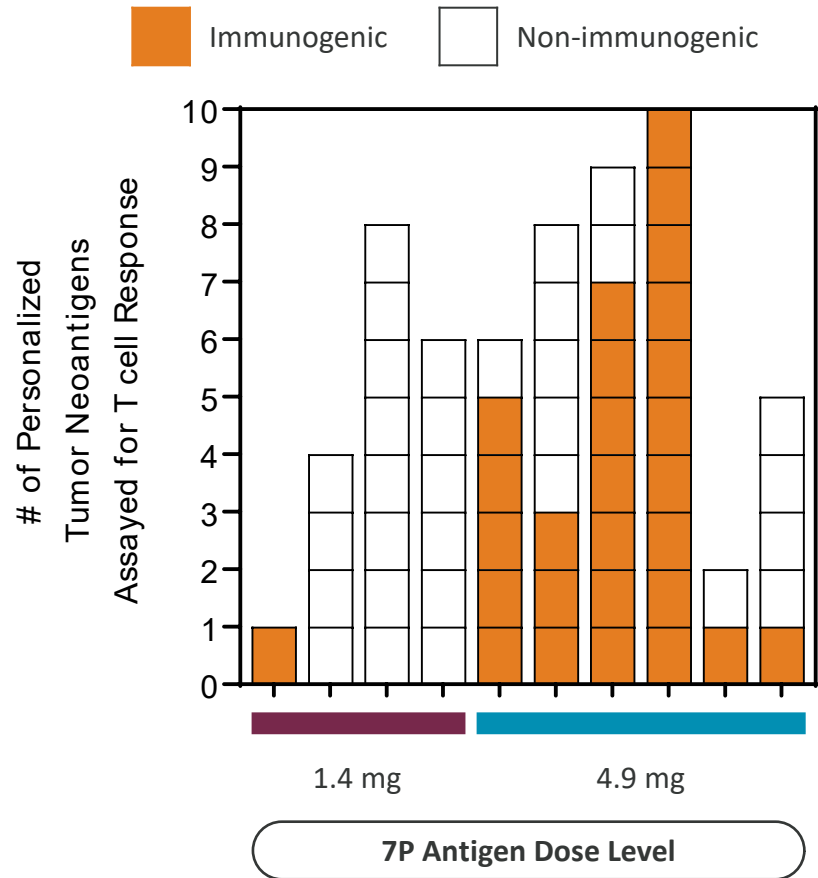
Responses shown are best overall responses vs baseline for each patient at any timepoint during the assessment period.

ELI-002 2P: Data cutoff 6-Sept-23
 ELI-002 7P: Data cutoff 24-Sep-24

★ = Phase 2 Dose

**Phase 2
Dose**

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



Antigen Spreading to Personal Tumor Neoantigens

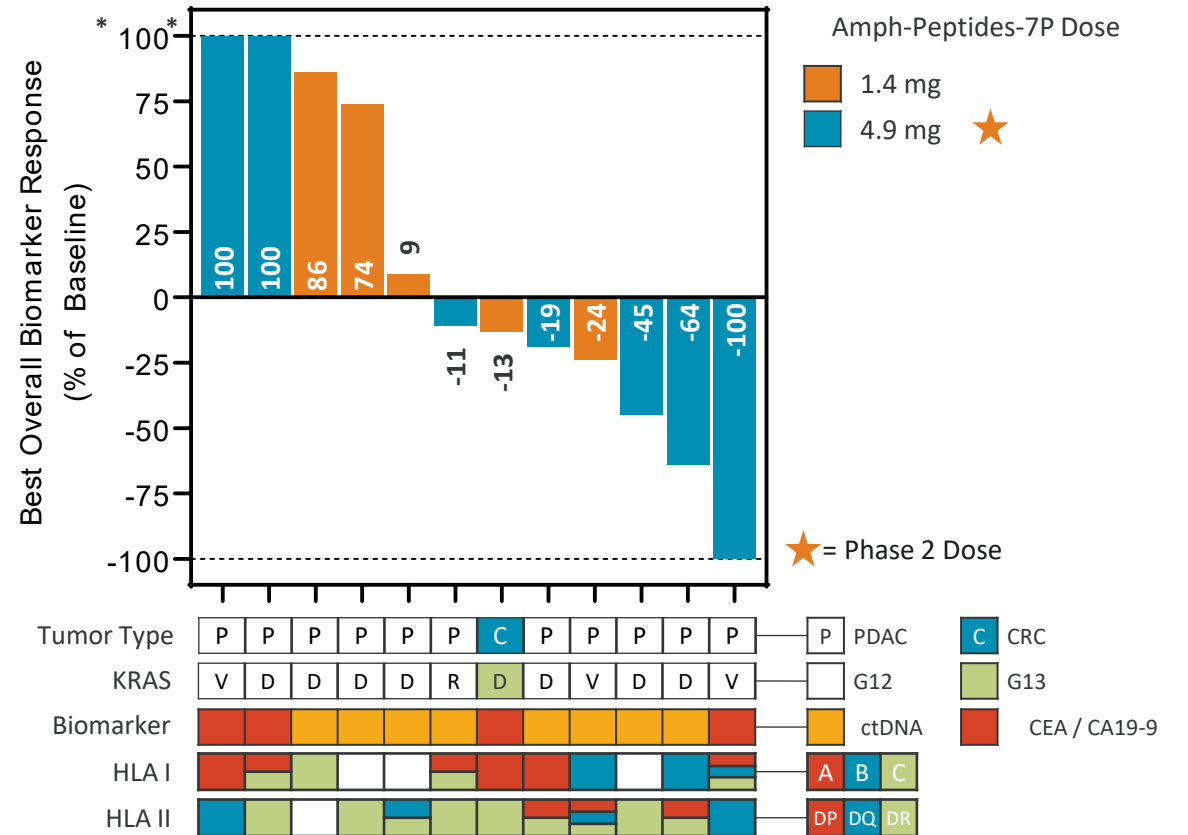
- ELI-002 7P vaccination led to expansion of T cell responses targeting passenger mutations alongside mKRAS driver mutations in a majority of evaluable patients
- T cells detectable by standard direct *ex vivo* FluoroSpot and flow cytometry, with no expansion required
- 70% of evaluated patients (7/10)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

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)

ELI-002 7P 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

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

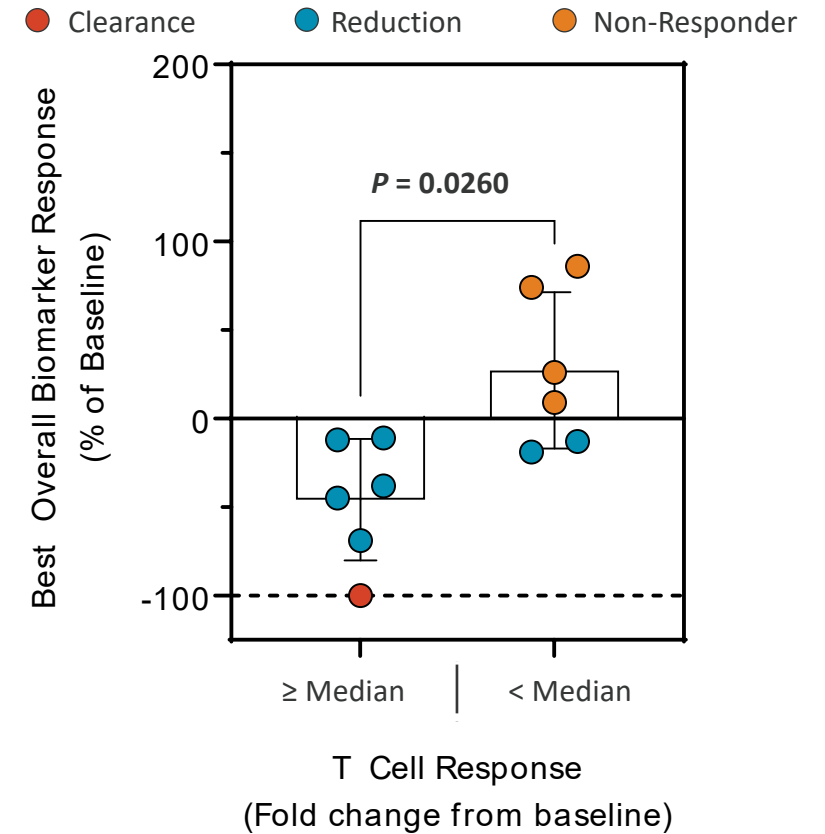
All patients with T cell responses above median showed tumor biomarker response

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.

Best Overall Tumor Biomarker Response



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

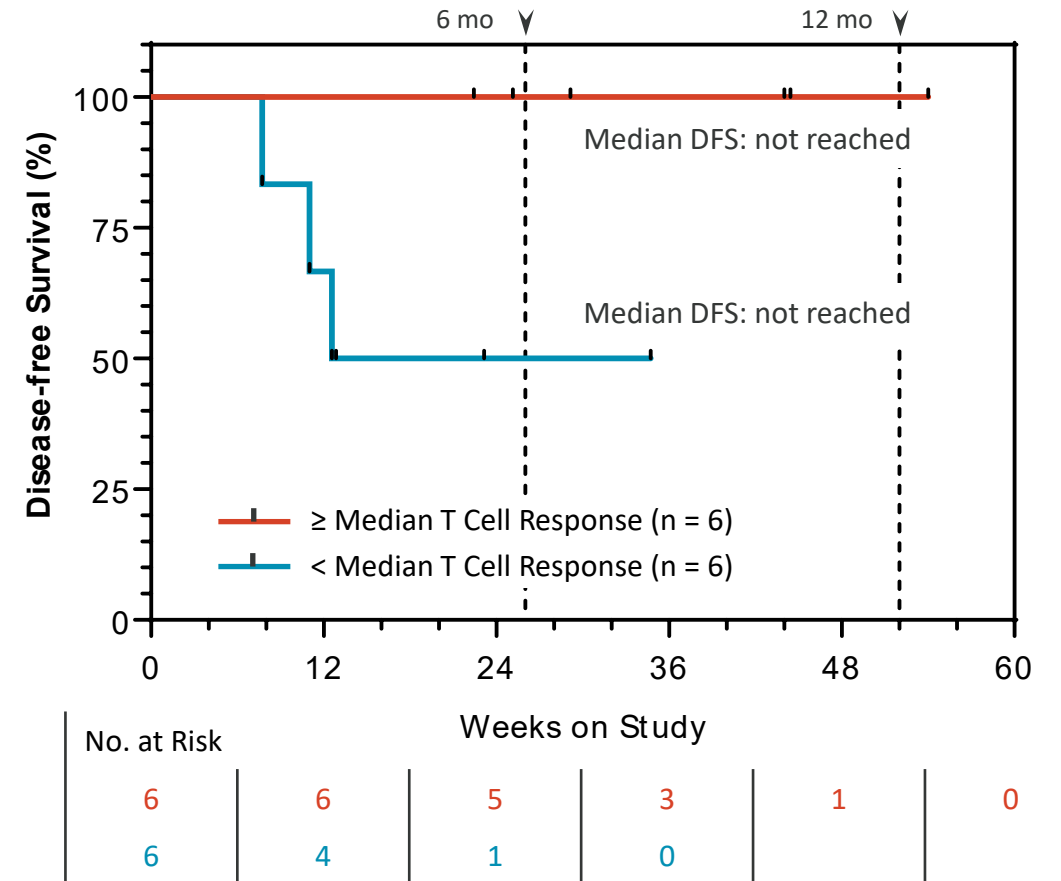
ELI-002 7P: DFS Strongly Correlates with T Cell Response

Improved DFS associated with above median T cell response

ELI-002 7P 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 was well tolerated at all dose levels with no DLTs observed

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 | 4.9 mg | Overall |
|--|-------------|-------------|----------------------------|
| | n=6 | n=8 | 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 | DDDDR VVV | |
| 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

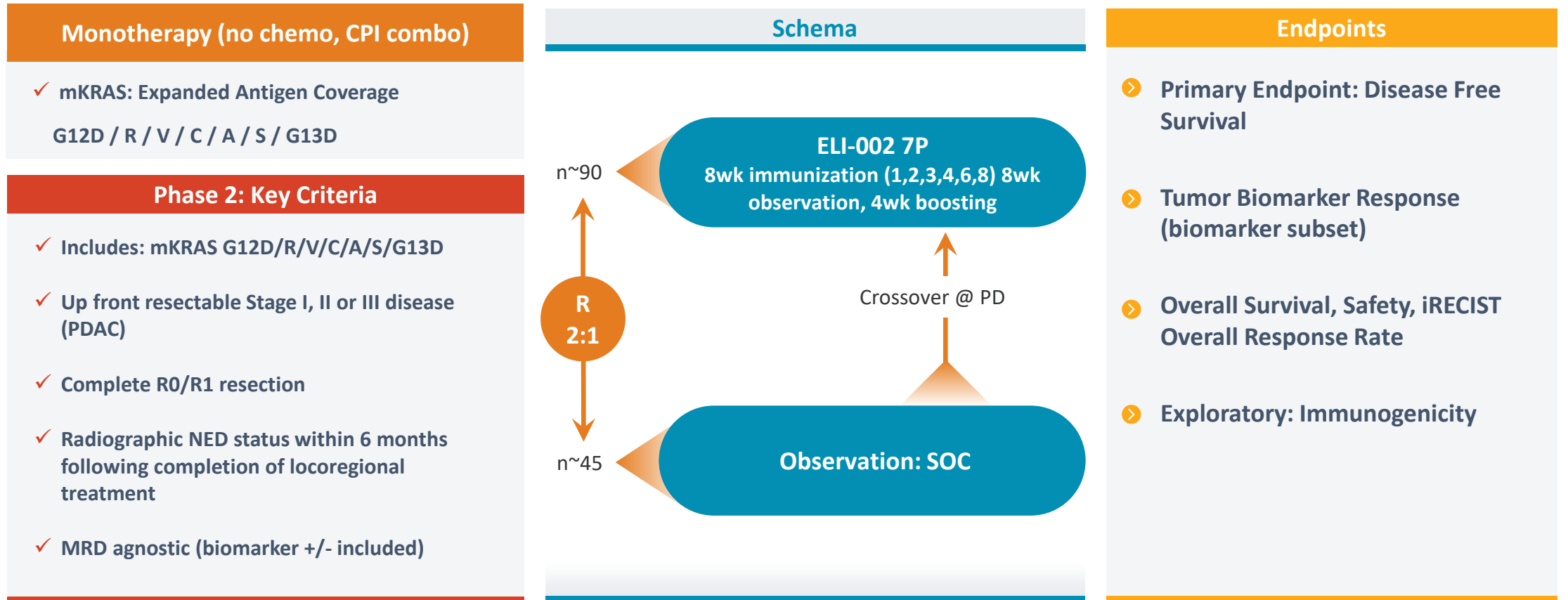
^a Preferred terms per the Medical Dictionary for Regulatory Activities, version 25.0

^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

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

CLINICAL STUDY OVERVIEW: NCT05726864



Key Milestones Achieved and Growth Initiatives for 2025-2026

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 H1 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
- Initiate ELI-002 + CPI investigator-sponsored clinical study in metastatic micro-satellite stable CRC
- Advance ELI-007 BRAF and ELI-008 p53 vaccines for Phase 1 readiness
- Initiate ELI-004 + radiation investigator-sponsored clinical study
- Finalize Phase 3 trial protocol in adjuvant PDAC setting



Targeting the Lymph Nodes to AMPLify Immunotherapy

Nasdaq: ELTX

January 2025

