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Virtual KOL Event to Discuss AMP-Powered ELI-002 for the Treatment of mKRAS-driven Pancreatic Cancer

Nasdaq: ELTX

June 25, 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 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.

Agenda

Topic	Participants
Introduction	**Robert Connelly**
Enhancing Cancer Vaccines through Lymph Node Targeting	**Darrell J. Irvine, PhD**
Pancreas Adenocarcinoma (PDAC): Adjuvant & Neoadjuvant Therapy	**Eileen M. O'Reilly, MD, FASCO**
• What are the current standards and treatment paradigms?	
• Biomarker selected therapy in PDAC	
• KRAS and PDAC	
• KRAS Directed & Other Immunotherapy	
• The Future: Opportunities for KRAS Immunotherapy in PDAC	
ELI-002 7P Phase 2 Trial Protocol Design, Upcoming Interim Data, and High-Level Statistical Plan	**Christopher Haqq, MD, PhD**
Closing Remarks	**Robert Connelly**
Q&A Discussion	**All**

Today's Speakers

Robert Connelly

Chief Executive Officer

Christopher Haqq, MD, PhD

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

Darrell J. Irvine, PhD

Professor & Vice-Chair, Scripps Research Institute

Eileen M. O'Reilly, MD, FASCO

Winthrop Rockefeller Endowed Chair in Medical Oncology at Memorial Sloan Kettering (MSK)

Available for Q&A

Peter DeMuth, PhD

Chief Scientific Officer

Preetam Shah, MBA, PhD

Chief Strategy and Financial Officer

Megan Filoon, JD

General Counsel, Secretary and Compliance Officer

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Enhancing Cancer Vaccines Through Lymph Node Targeting

Darrell J. Irvine, PhD

Professor, Department of Immunology & Microbiology, The Scripps Research Institute, Howard Hughes Medical Institute

Enhancing Cancer Vaccines through lymph node targeting

Darrell J. Irvine

Scripps Research

Primary challenges of therapeutic vaccines in cancer

Poor selection of vaccine antigens

- Early trials often targeted antigens restrained by tolerance
- Lack of T cell responses targeting driver mutations

Clinical evaluation using poor trial designs

Lack of lymph node targeting – “the brain center of the immune system”

Insufficient induction of anti-tumor T cell responses

Inability to overcome cancer’s natural immunosuppression

Peptide vaccines in particular have shown weak T cell responses in animal models and human clinical trials

Peptide vaccines for cancer

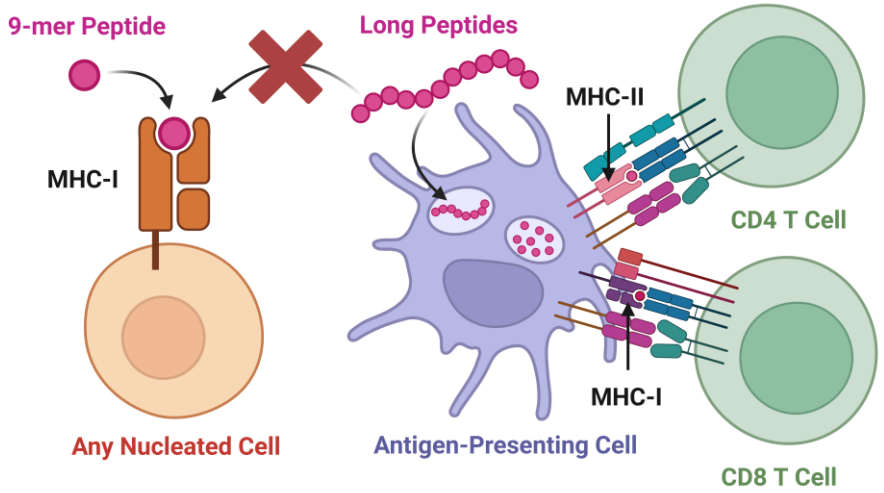
20-30 aa epitopes + adjuvants

Features

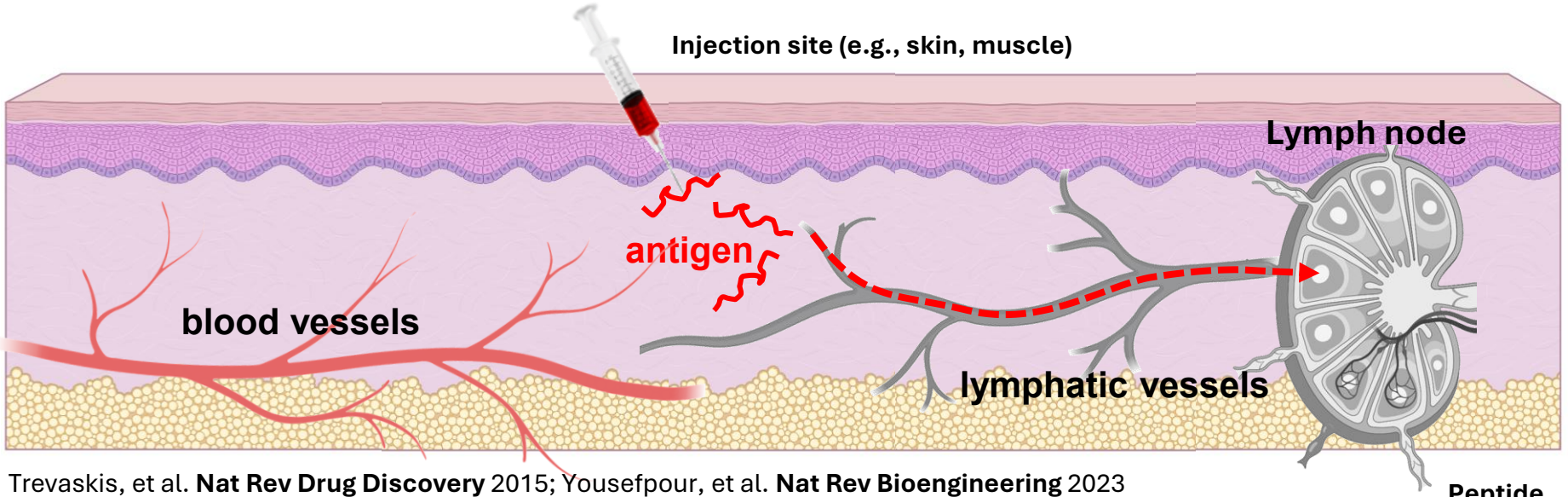
- Safe
- Inexpensive
- Enable targeting of unique antigens (e.g., phosphopeptides)
- Amenable to rapid manufacture (e.g., for neoantigen-based vaccines – Ott et al. *Nature* 2017; Keskin et al. *Nature* 2018; Hilf et al. *Nature* 2019)

Challenges

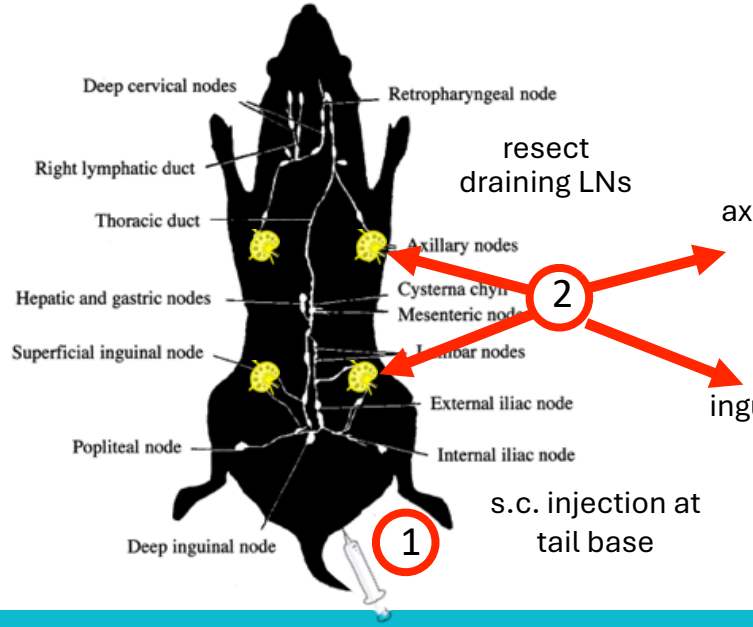
- Challenge of choosing effective adjuvants
- Biology of antigen presentation
- Poor-to-modest potency in humans



Where we started: understanding limitations of peptide vaccines



Trevaskis, et al. **Nat Rev Drug Discovery** 2015; Yousefpour, et al. **Nat Rev Bioengineering** 2023



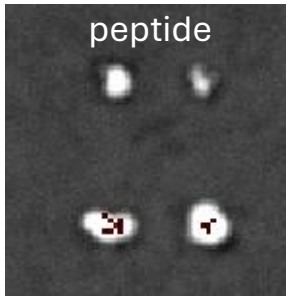
resect draining LNs

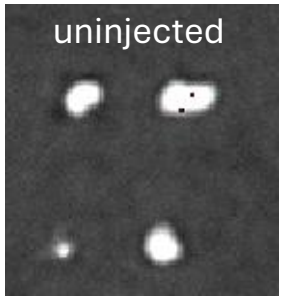
axillary

inguinal

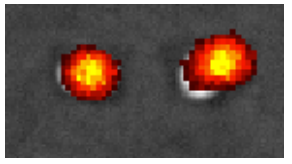
s.c. injection at tail base

Antigen accumulation in draining lymph nodes by whole-tissue imaging:

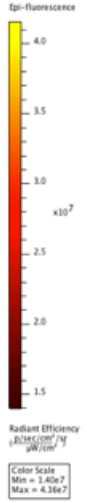




Positive control – direct injection ex vivo:

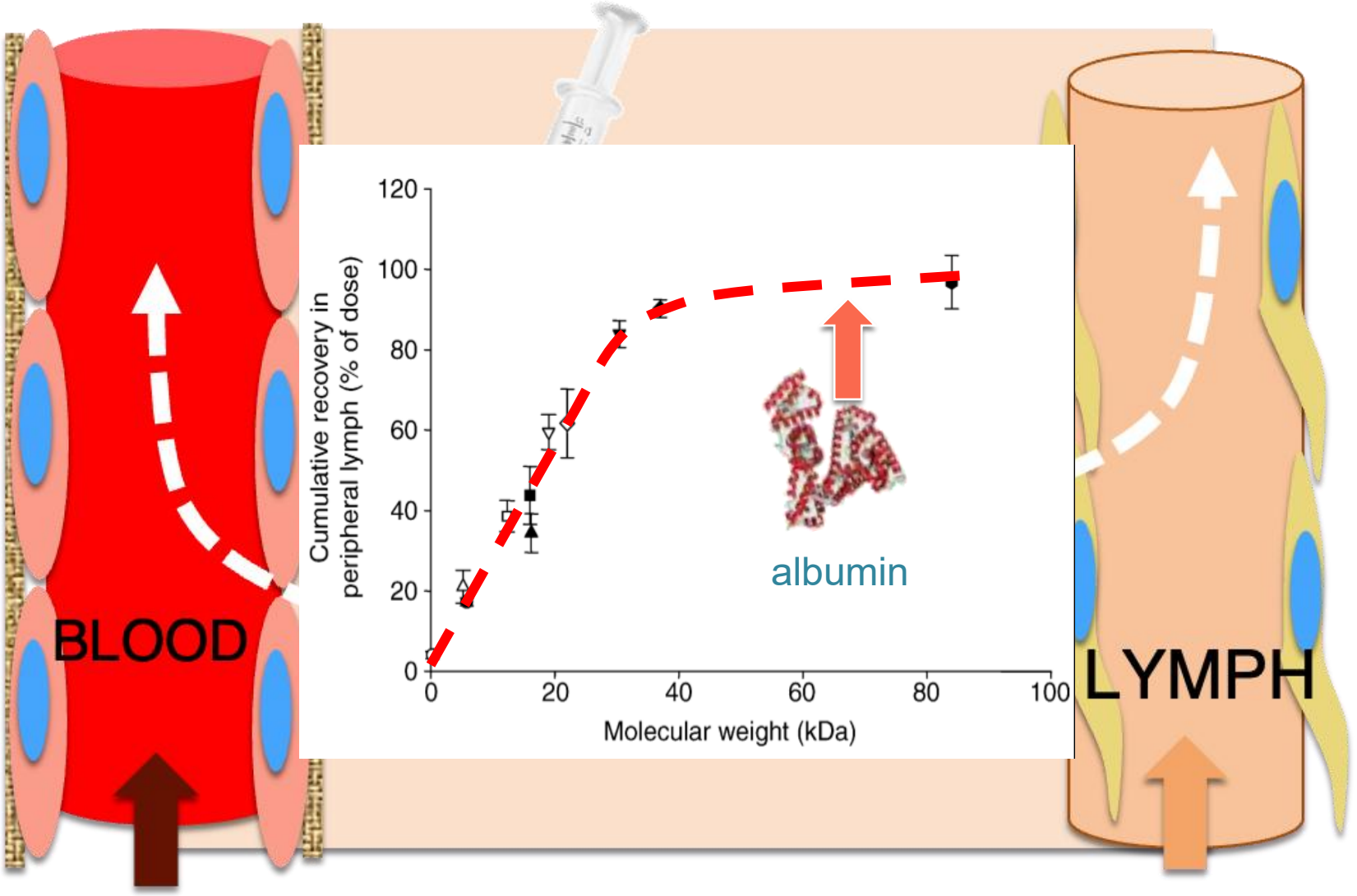


Peptide fluorescence



Trevaskis, et al. Nat Rev Drug Discovery 2015

Physiology of solute transport in tissues

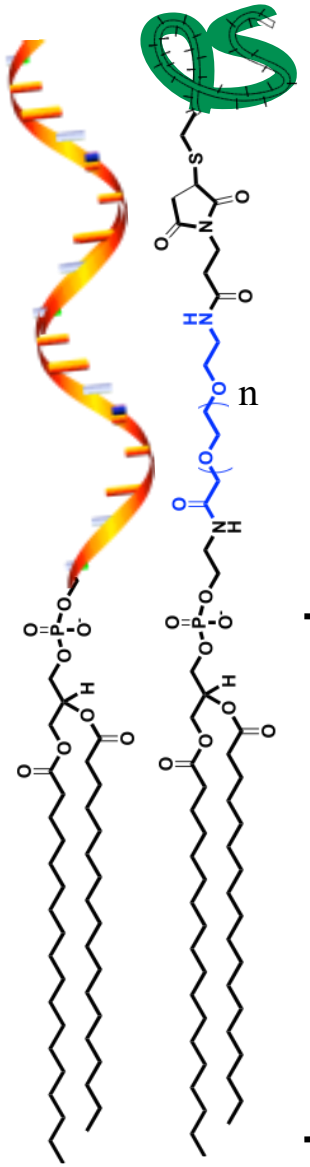


McLennan, D. N., Porter, C. J. H., & Charman, S. A. (2005). *Drug Discovery Today: Technologies*, *2*(1), 89–96

Conceptual basis for albumin as vaccine chaperone

Molecular adjuvants (e.g., CpG DNA)

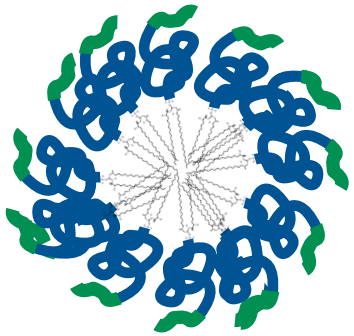
Peptide antigen



polar block - solubility

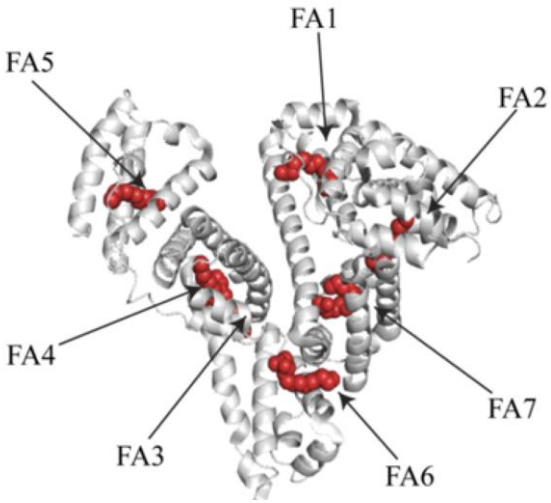
phospholipid tail – albumin binding

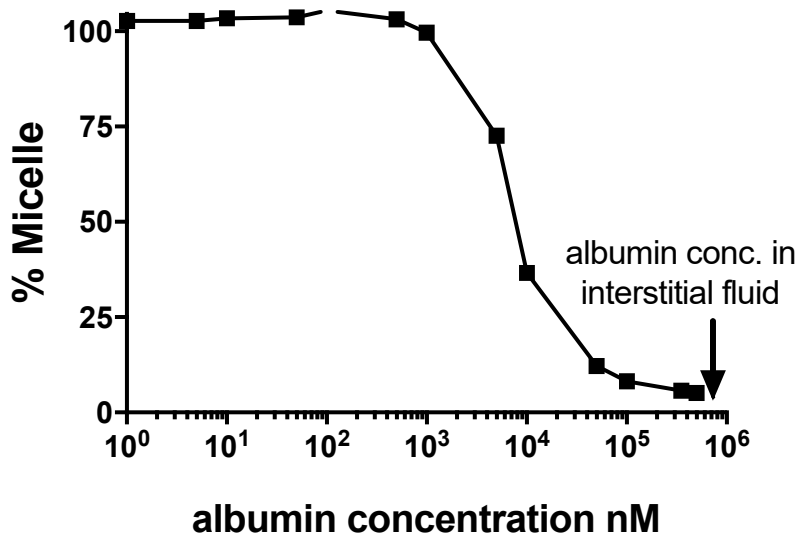
water

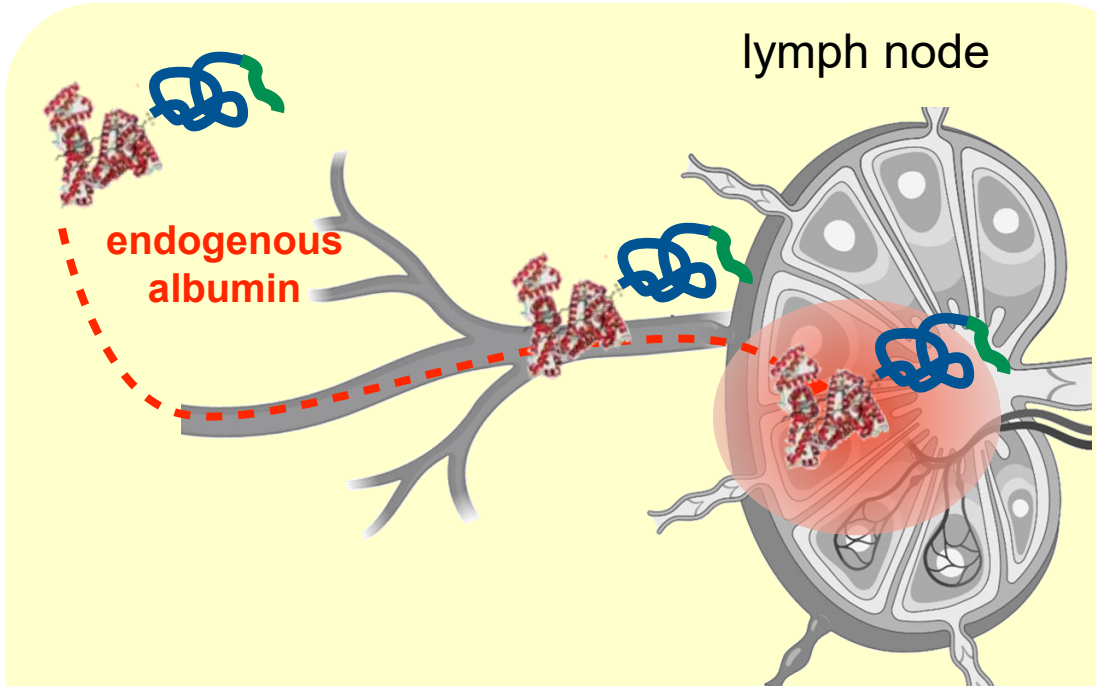


serum

albumin

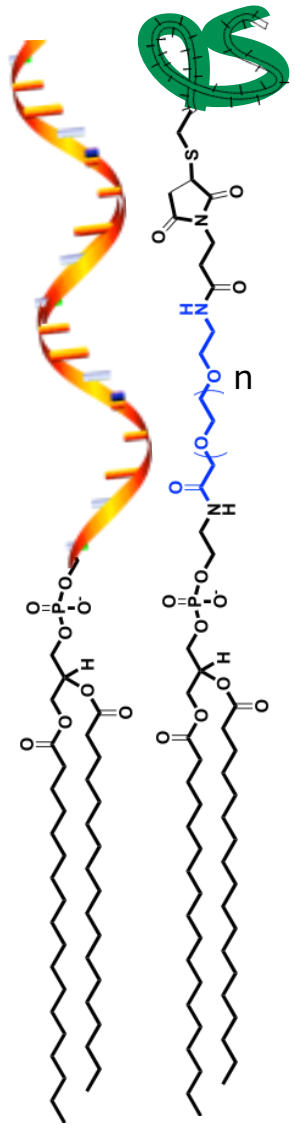






(Mishra et al. *Int. J. Mol. Sci*. 2021)

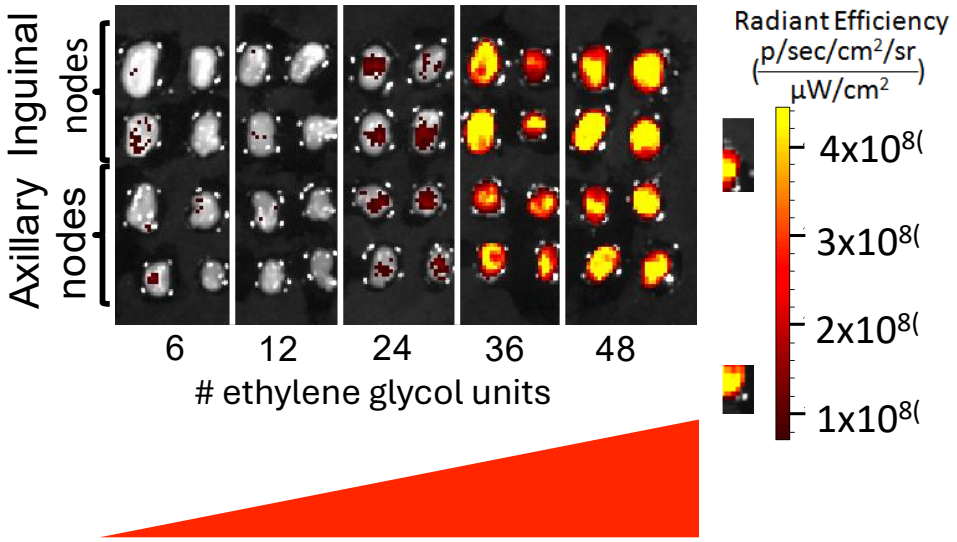
Structural programming of vaccine amphiphile equilibrium state in vivo



polar block

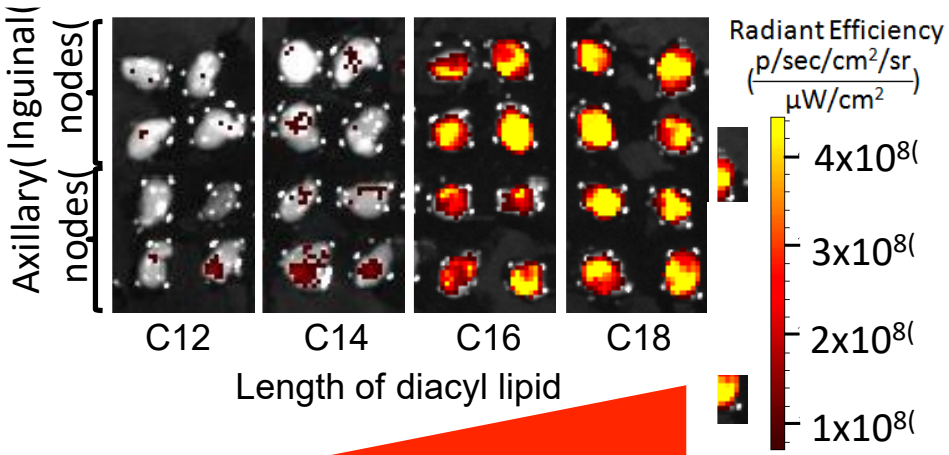
saturated long diacyl tail

Solubility



amphiphile solubility

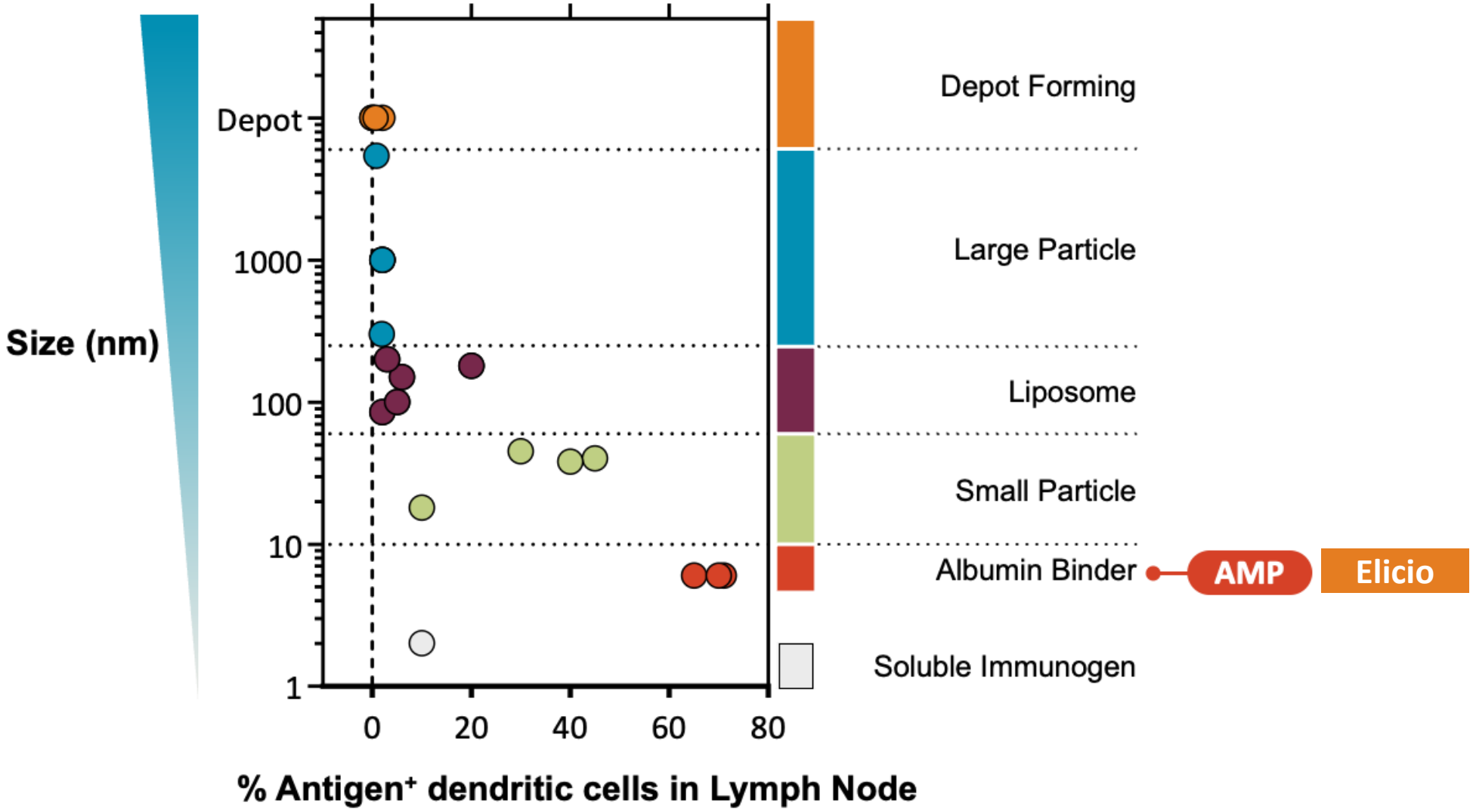
Albumin binding



albumin binding affinity

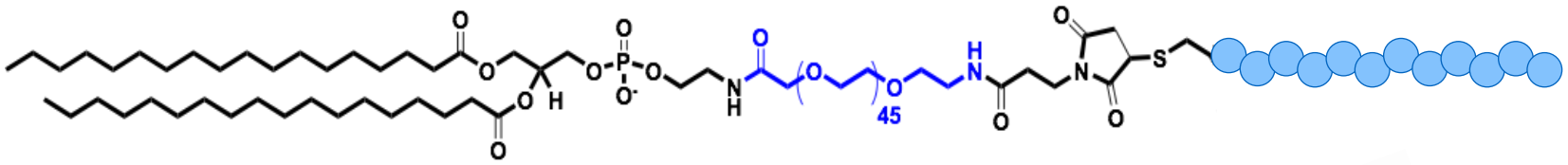
Liu et al. **Nature** 2014

What about alternative strategies for lymph node targeting?

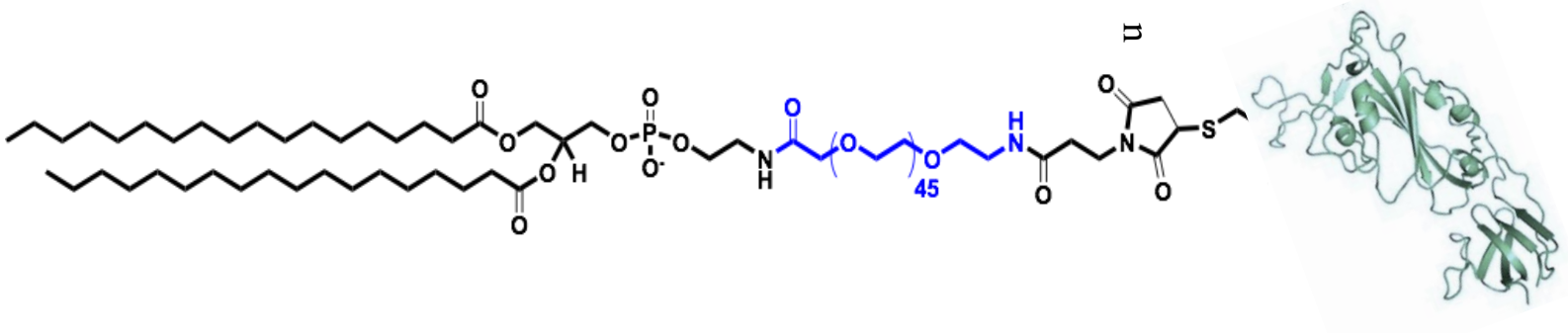


Irvine, et al. **Advanced Drug Delivery Reviews** 2020

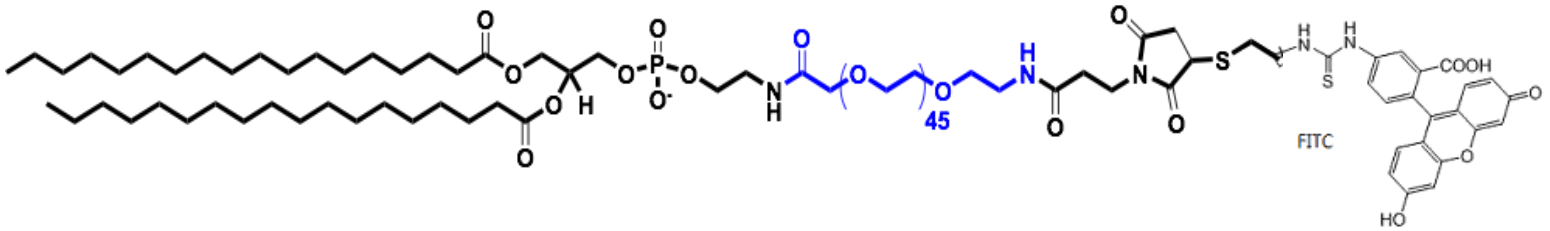
AMP modification can be applied to target diverse molecular payloads to lymph nodes



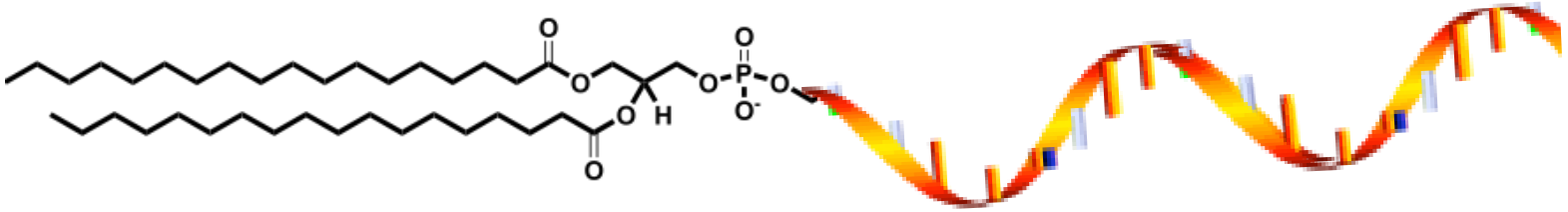
peptides



proteins



small molecules

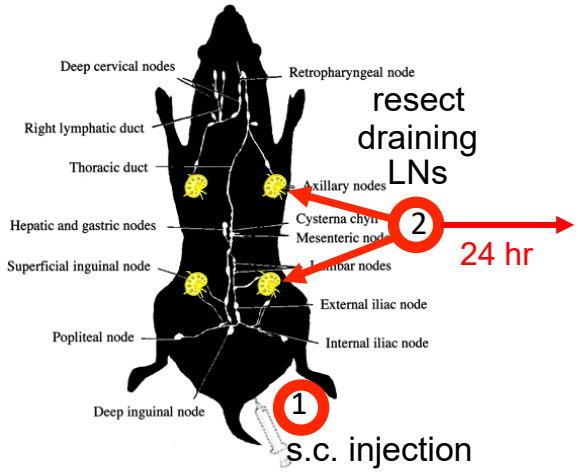


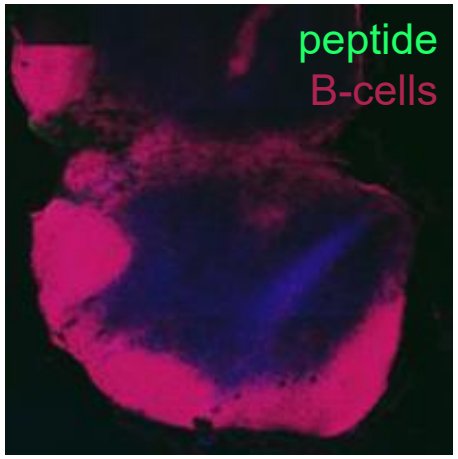
nucleic acids

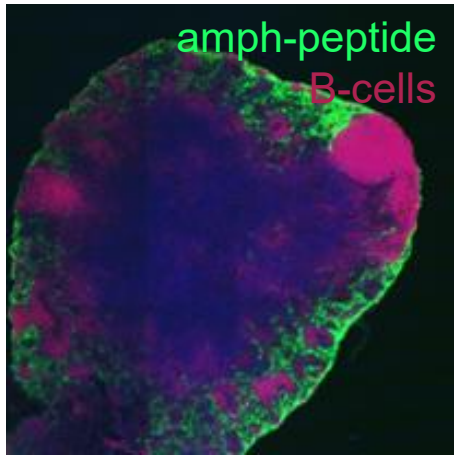
Liu et al. **Nature 2014**; Ma et al. **Science** 2019; Hartwell et al. **Science Translational Medicine** 2022

AMP-vaccines show enhanced uptake in lymph nodes in small and large animal models

In mice:

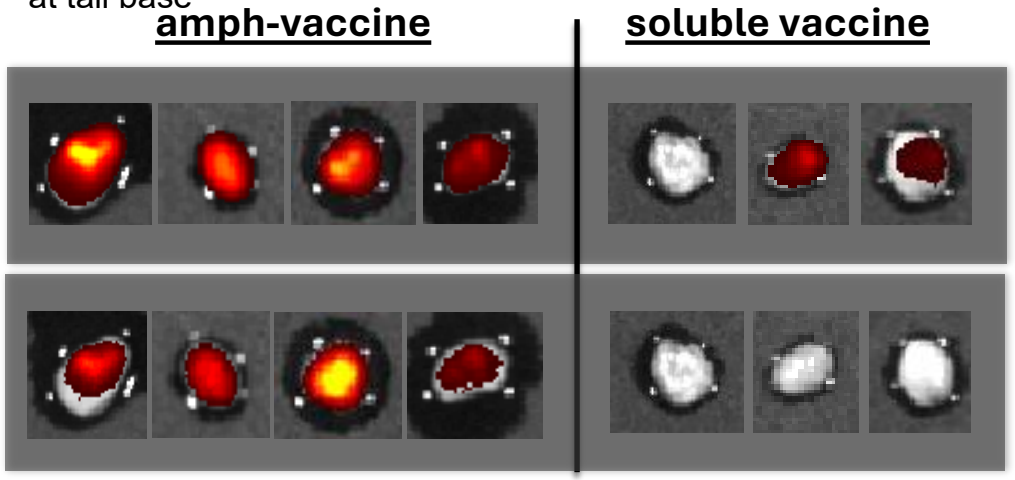


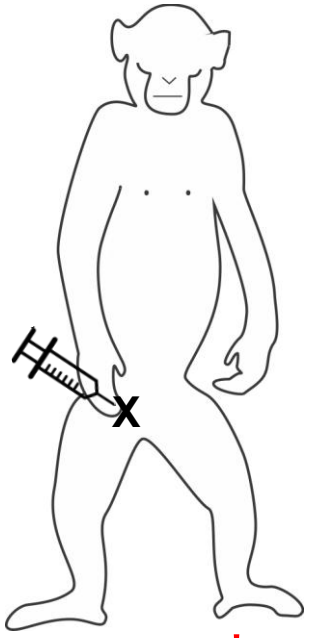


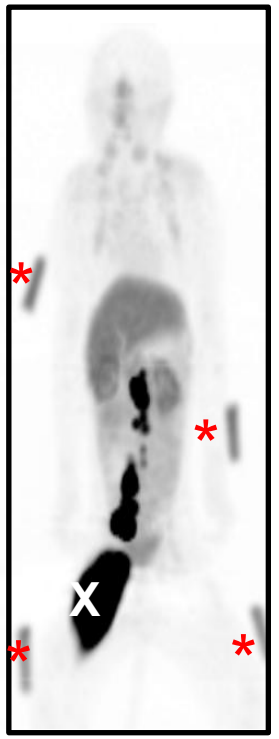


Liu et al. *Nature* 2014

In rhesus macaques:







* ^{64}Cu standards

Martin et al. **Biomaterials** 2021

AMP-vax molecules also protect peptides from premature degradation

Antigen stability assay:

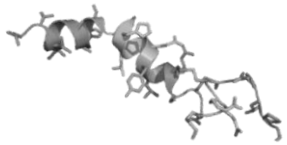
Incubate antigen with serum 37°C 24 hr

↓

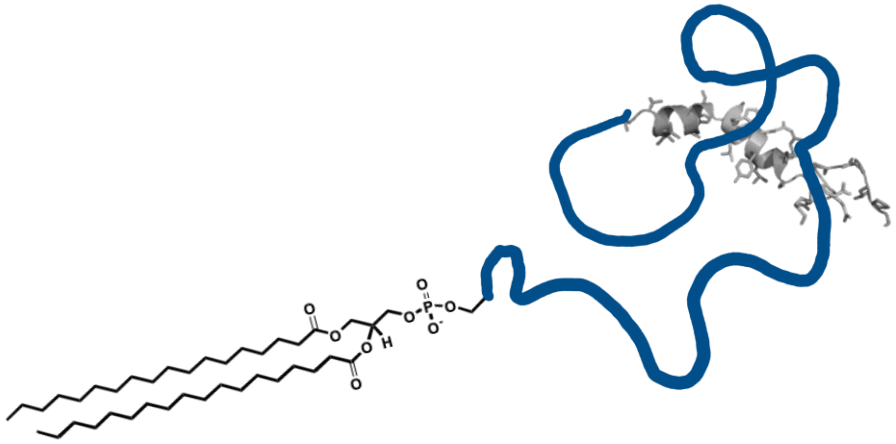
Add to splenocytes from vaccinated mice

↓

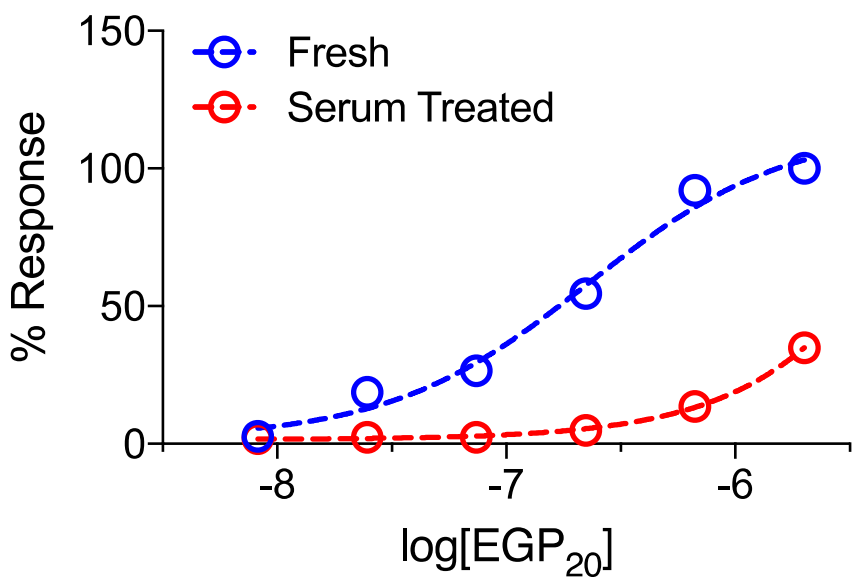
Measure frequency of IFN-γ-producing T-cells by ICS

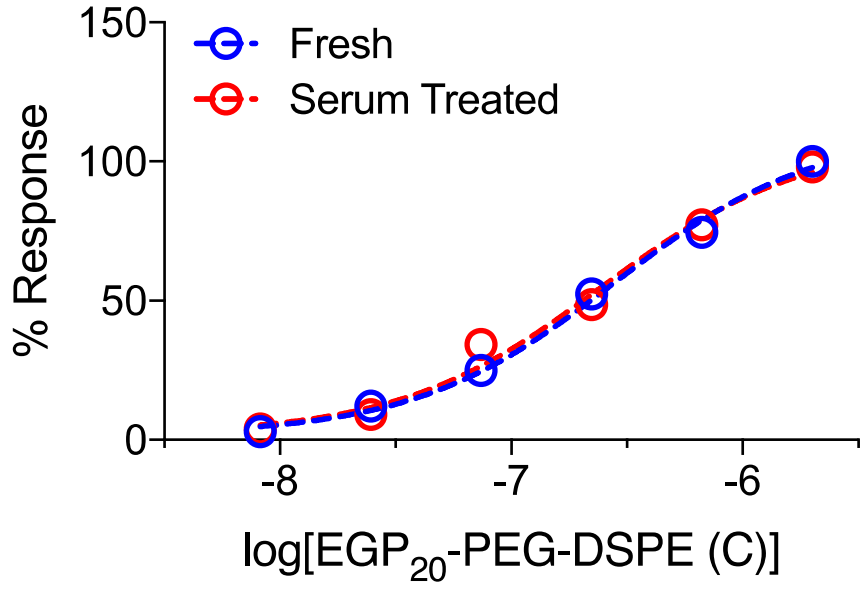


EGP peptide



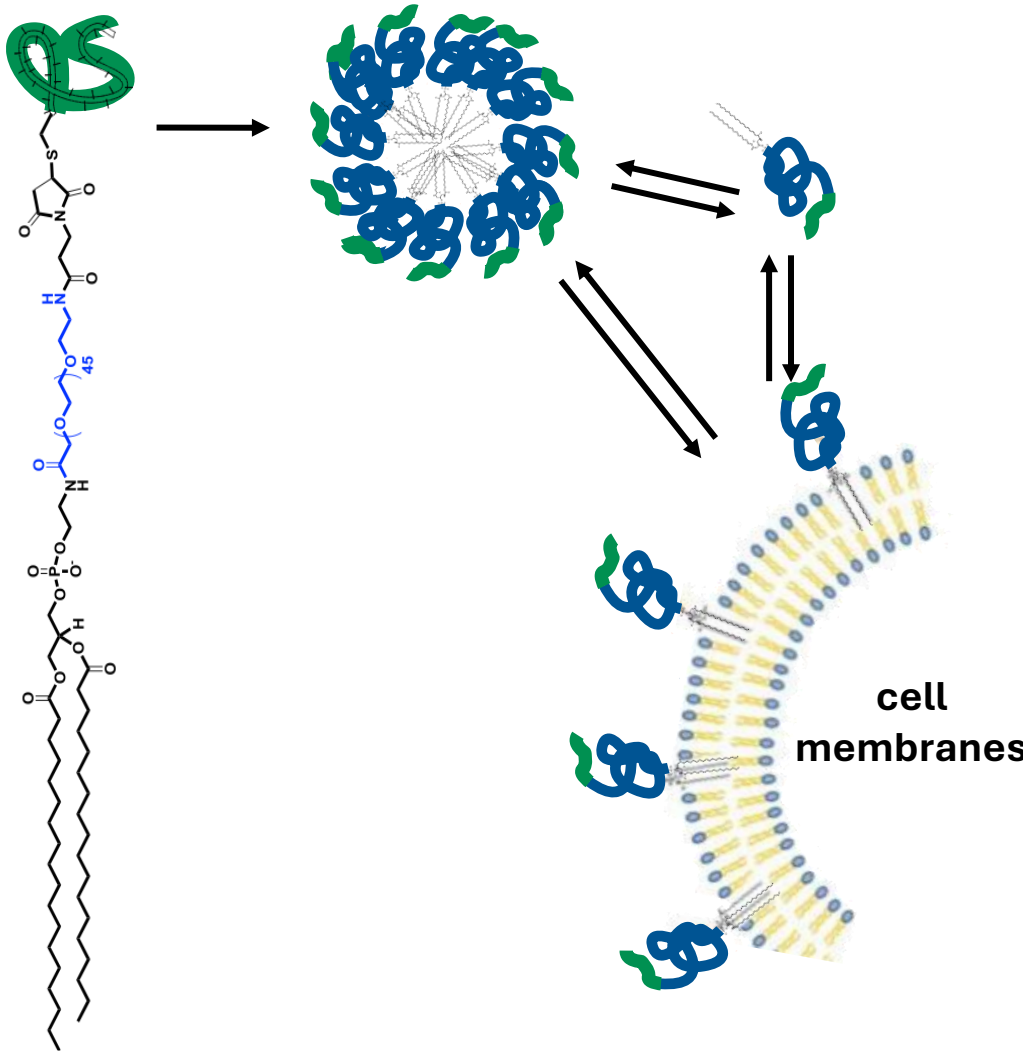
amph-EGP

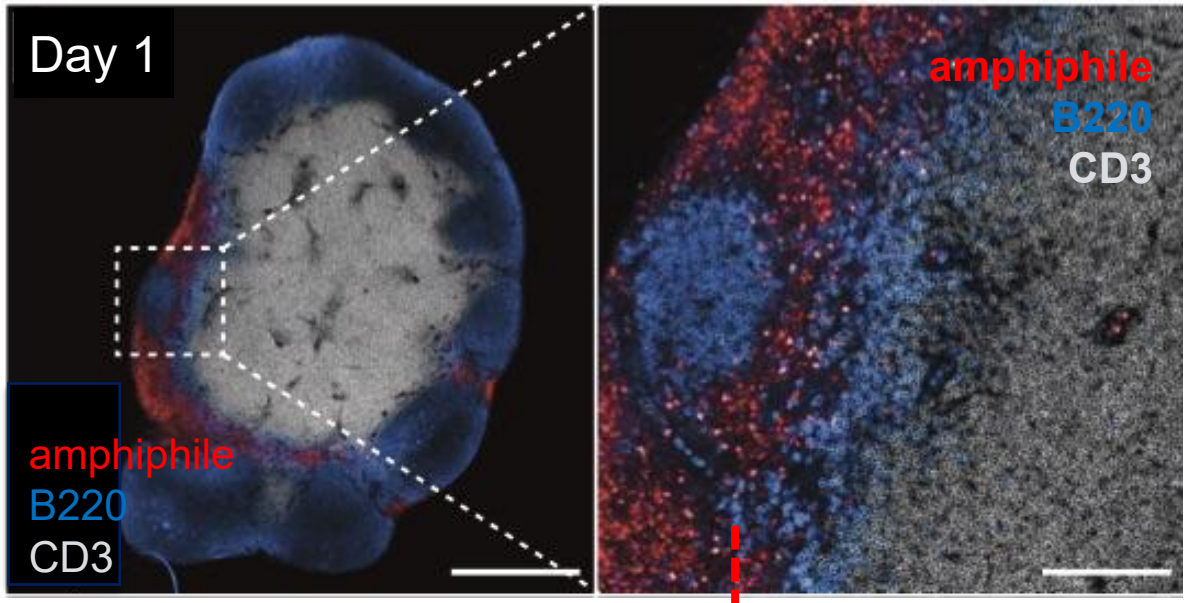


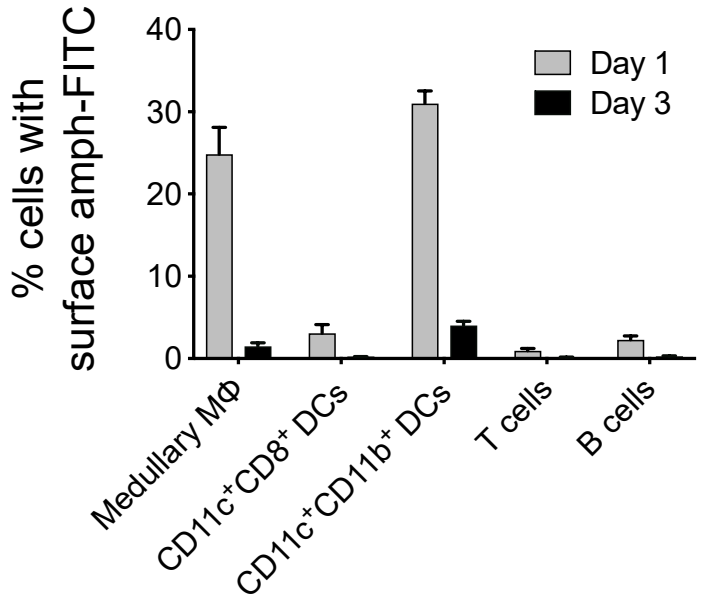


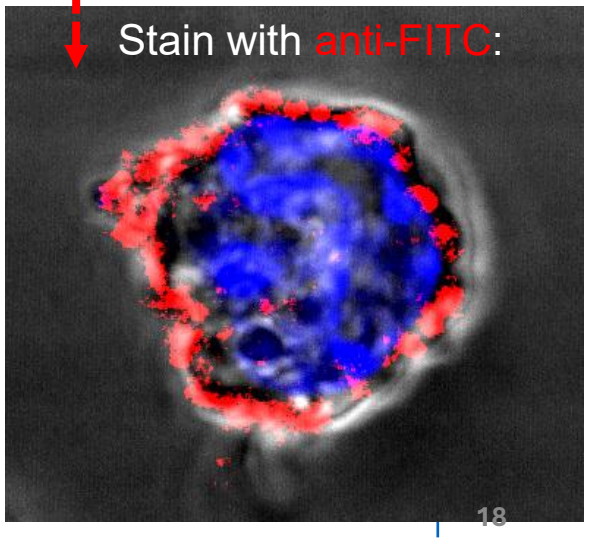
Moynihan et al. **Cancer Immunol. Res**. 2018

A third property of amphiphile-ligands: Cell membrane insertion



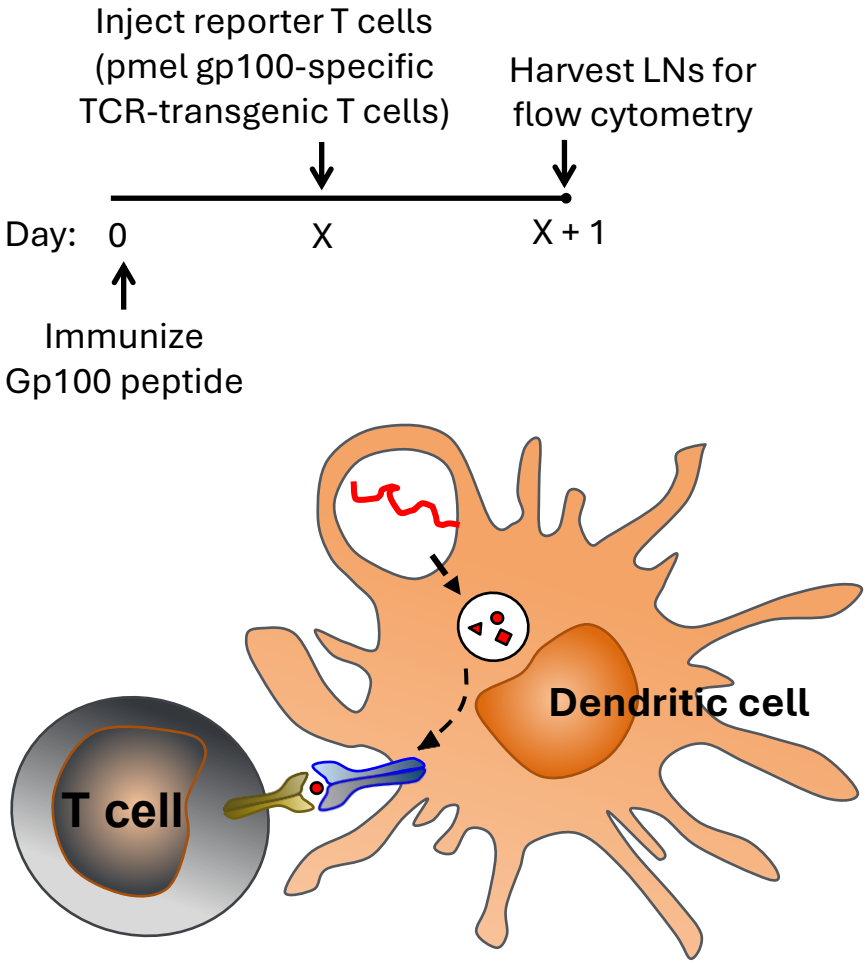


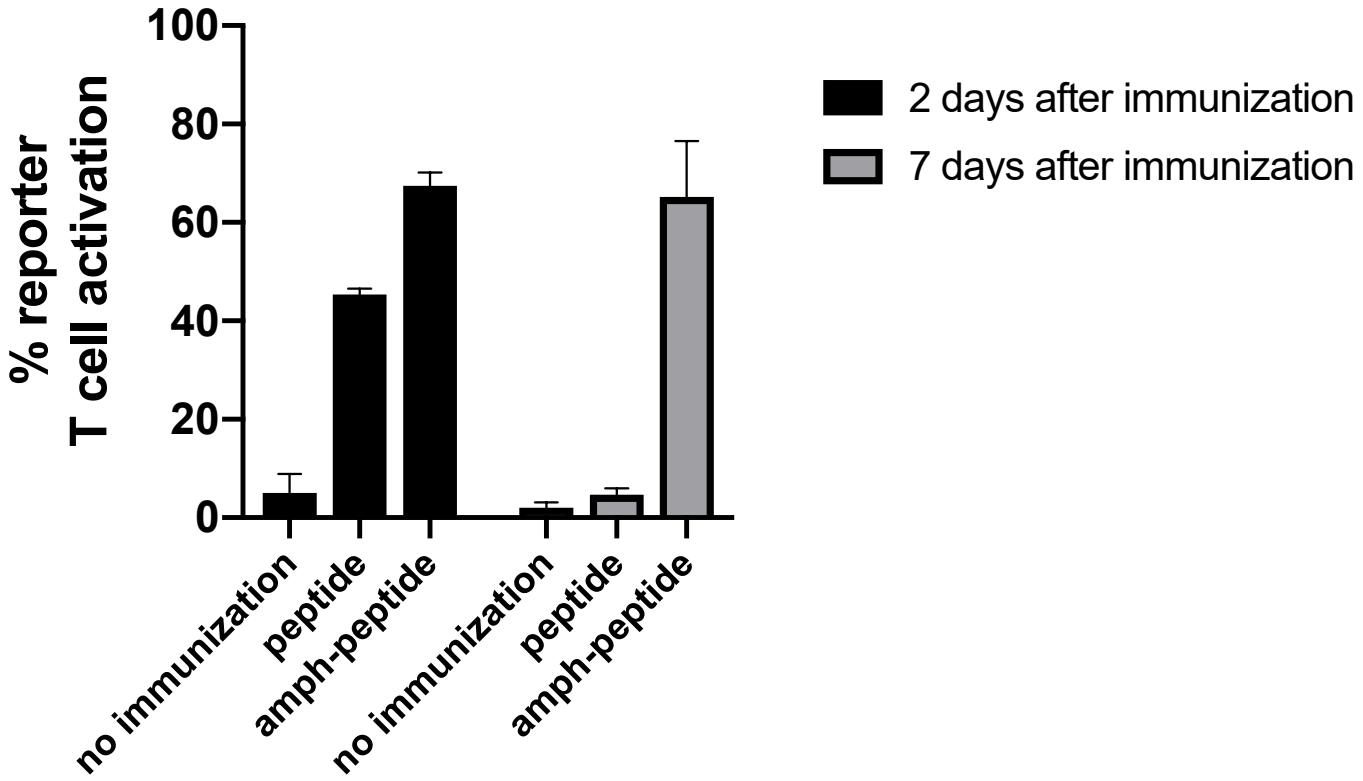




Liu et al. Angew. **Chem. Intl. Ed.** 2011;
Ma et al. **Science** 2019

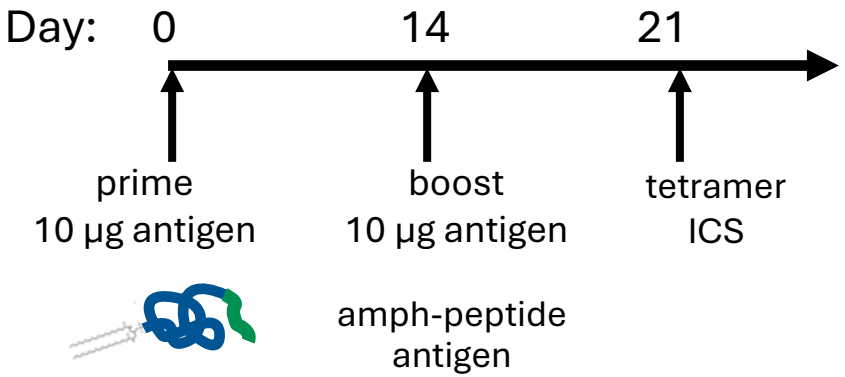
Enhanced lymph node delivery leads to prolonged antigen presentation

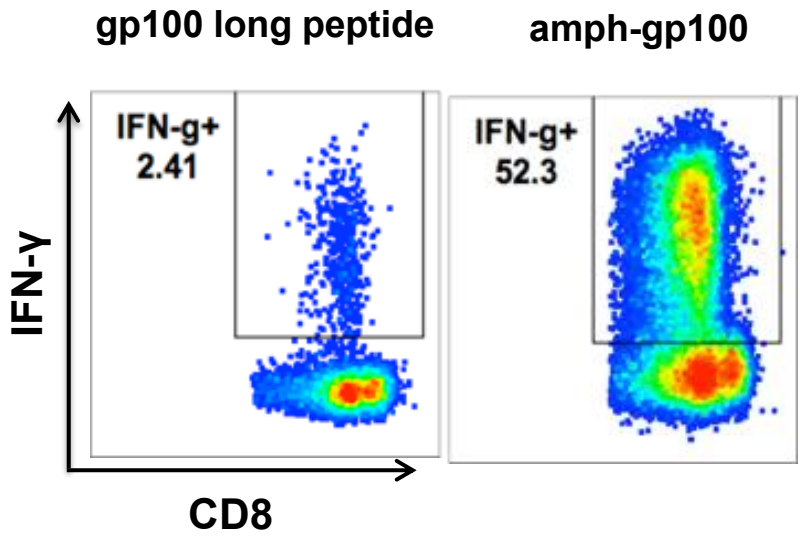


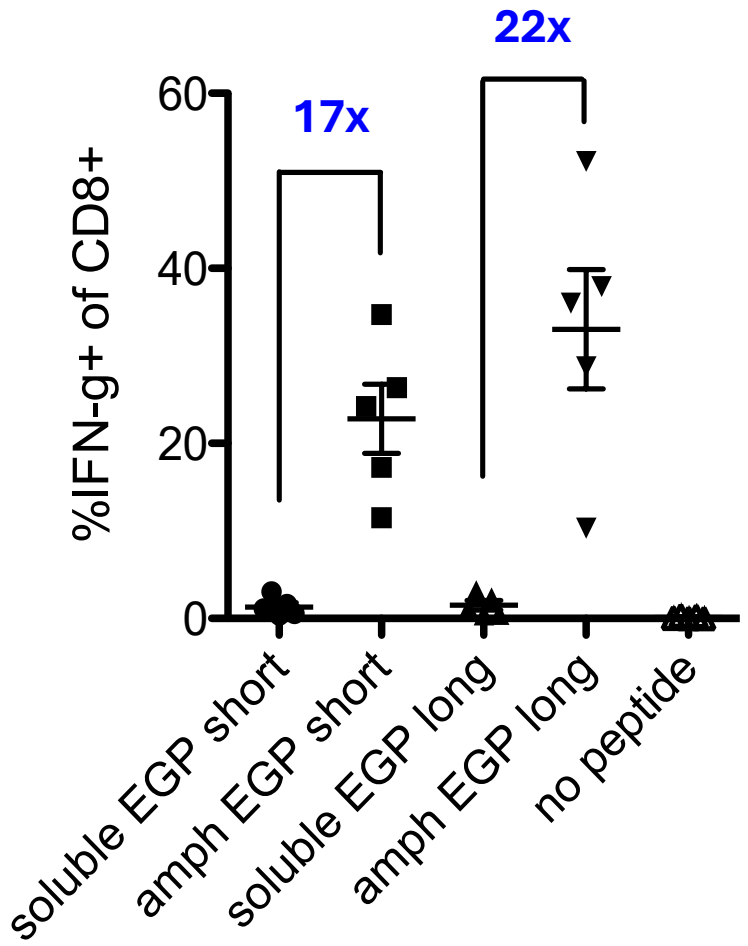


Moynihan et al. **Cancer** Immunol. Res. 2018

What is the impact of optimizing these features of a vaccine?

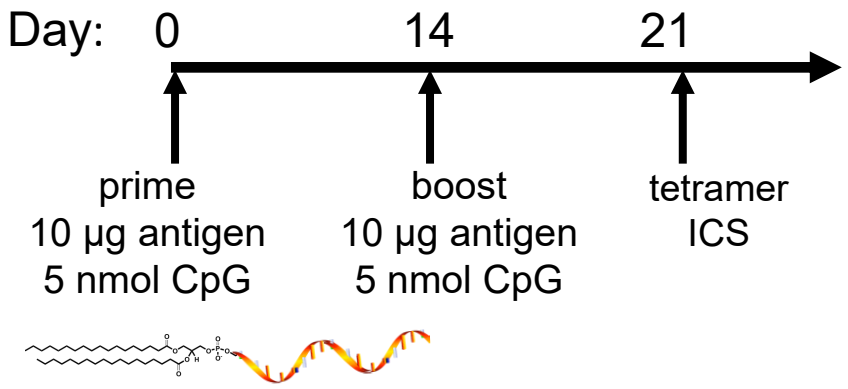


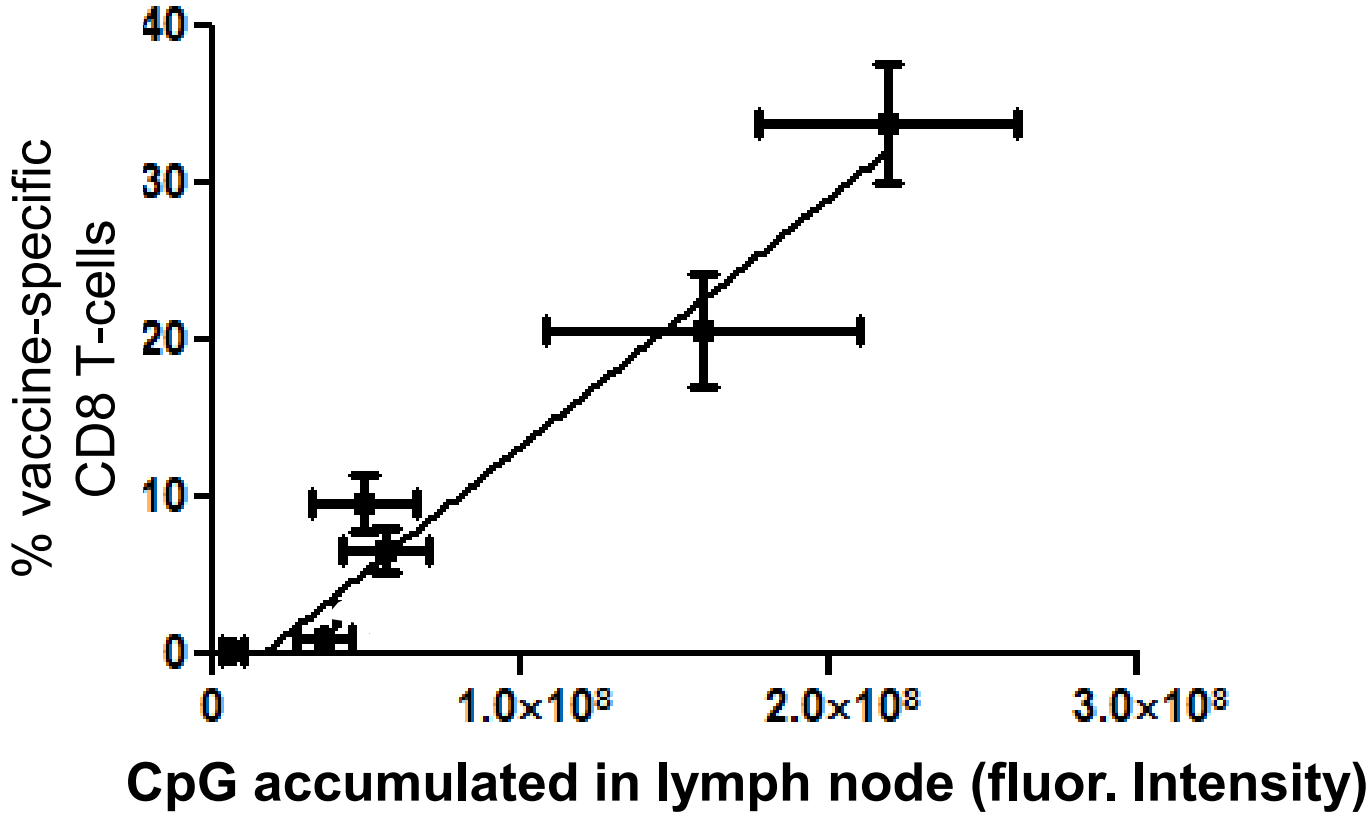




Liu et al. **Nature** 2014; Moynihan et al. **Cancer Immunol. Res.** 2018

Targeting the adjuvant to lymph nodes is also critical for optimal T cell priming

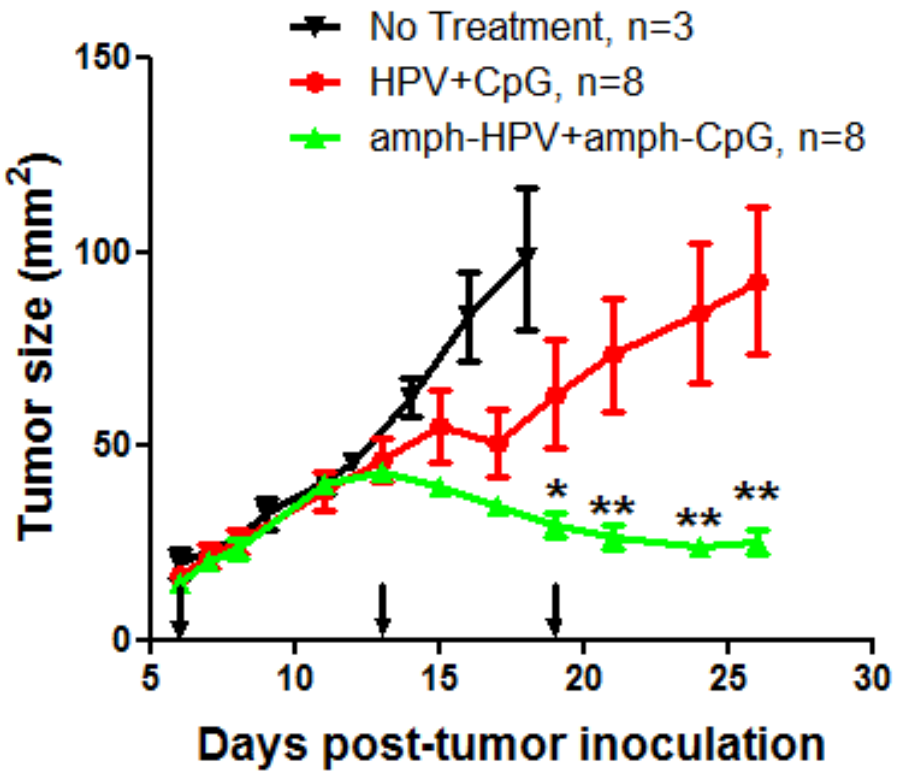




Liu et al. **Nature** (2014);
Moynihan et al. **Cancer Immunol. Res.** (2018)

Albumin-mediated LN-targeting of both antigen and adjuvant maximizes immune response

therapeutic anti-tumor vaccination–
TC-1 cervical cancer model:



Liu et al. **Nature** 2014

Conclusions

- “albumin hitchhiking” enables AMP-vaccines to concentrate in lymph nodes via 3 intertwined mechanisms:
  - Efficient entry into lymphatics
  - Protection of cargo from premature degradation
  - Transfer of AMP molecules to antigen presenting cells in the lymph node via membrane insertion
- …this change in vaccine PK dramatically amplifies T cell priming in preclinical mouse models
- **Eileen** and **Chris** will discuss today how Elicio has been able to harness the advantages of AMP technology to dramatically improve T-cell response as evidenced in Elicio’s two Phase 1 trials

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Esther Welkowsky

Funding:

Pancreas Adenocarcinoma: Adjuvant & Neoadjuvant Therapy

Eileen M. O'Reilly, MD, FASCO

Winthrop Rockefeller Endowed Chair in Medical Oncology at Memorial Sloan Kettering (MSK)

Pancreas Adenocarcinoma: Adjuvant & Neoadjuvant Therapy

Eileen M. O'Reilly, MD, FASCO

Winthrop Rockefeller Endowed Chair, Memorial Sloan Kettering Cancer Center

Chair, Human Research Protection Program and IRB

Professor of Medicine, Weill Cornell Medicine

June 25th , 2025

Memorial Sloan Kettering Cancer Center

Disclosures: O’Reilly

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Consulting/DSMB/Steering Committees

Arcus, Alligator, Agenus, Astellas, BioNTech, Ipsen, Merck, Novartis, Leap Therapeutics, Astellas, BMS, Fibrogen, Revolution Medicine, Merus, Moma Therapeutics, Regeneron, Ikena

Agenda

Current standards

Biomarker selected therapy

KRAS and PDAC

Immunotherapy

The future

Setting the Scene: Pancreas Cancer 2025

New diagnoses – US 2025 67,040; Global (2023) 510,992

Mortality – US 2025: 51,980

8th–10th most common cancer (3% new cancers)

1.2% increase/year → 2nd cause-related deaths by 2030

5-year survival (all stages) ~13% (Localized 44%; Regional 16%; Metastatic 3%)

8% of all cancer mortality

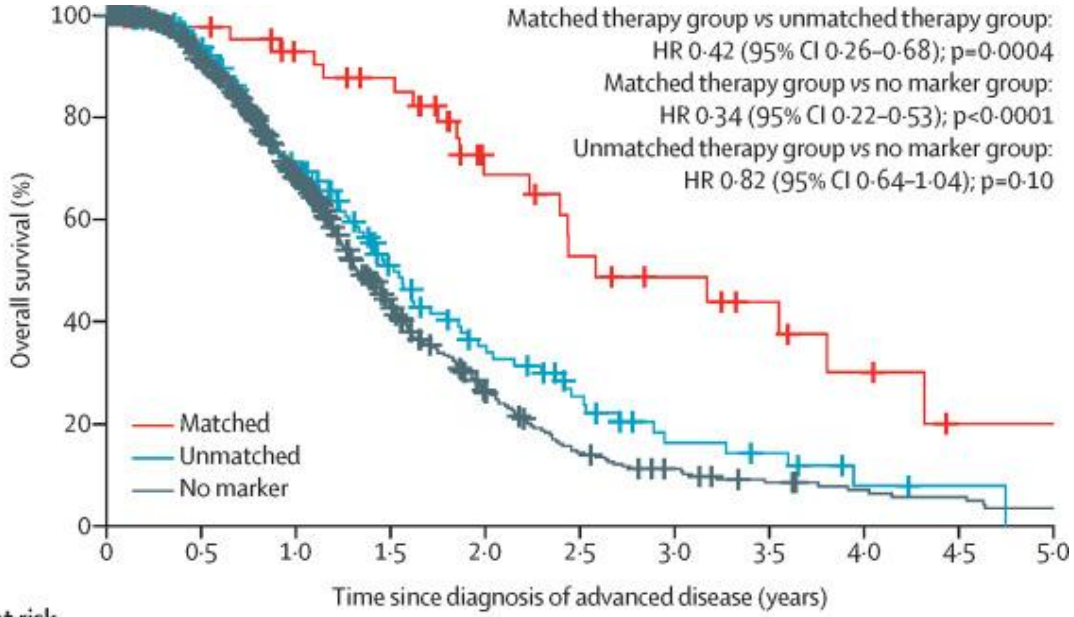
Current State of Therapy Advanced PDAC 2025

First & Second-Line Practice-Changing Phase III Trials for Unselected Disease

	N	Median OS	Median PFS	Response Rate	Reference
First-Line					
mFOLFIRINOX (vs Gem) PRODIGE/ ACCORD 11	171	**11.1 m**	6.4 m	31.6%	2011
Gemcitabine, nab-Paclitaxel (vs Gem) MPACT	431	**8.5 m**	5.5 m	23%	2013
NALIRIFOX (vs Gem/Nab-P) NAPOLI-3	383	**11.1m**	7.4 m	41.8%	2024
Second-Line					
Liposomal irinotecan/5-FU vs 5-FU NAPOLI-1	117	**6.1 m**	3.1 m	16%	2016

Conroy T. N Engl J Med. 2011
Von Hoff, D. NEJM, 2013
Wainberg, Z. Lancet, 2023
Wang-Gillam, A. Lancet Oncol, 2016

Targeted Therapeutics Improve Outcomes in PDAC



Number at risk (number censored)											
Matched therapy	46	42	36	32	18	13	10	7	4	1	1
	(0)	(3)	(4)	(2)	(8)	(1)	(2)	(2)	(1)	(2)	(0)
Unmatched therapy	143	116	78	44	27	16	8	6	2	1	0
	(0)	(19)	(11)	(15)	(4)	(4)	(3)	(1)	(2)	(1)	(0)
No marker	488	384	241	124	63	33	22	14	10	8	5
	(0)	(66)	(55)	(39)	(15)	(4)	(4)	(3)	(2)	(0)	(0)

- **Genomic alterations and matched therapy improves outcome**
  (retrospective, non-randomized, pre-RAS)

Pishvaian, M. Lancet Oncology, 2020
Reissig, TM.…Siveke, JT. ESMO Open, 2023

Pancreas Cancer: Genomic Testing

Genomic Subsets for Therapeutic Actionability: Today

Increasing Biomarker Selected Options for PDAC

KRAS^{MUT} PDAC (~95%)	*KRAS*^{WT} PDAC (~5%)	HRD, MSI-H	Other Targets, Descriptors
Chemotherapy mFOLFIRINOX NALIRIFOX Gemcitabine, nab-Paclitaxel Gemcitabine, **Erlotinib**	**Fusions** RET* ALK, ROS, NTRK FGFR2/3, MET, NRG-1, MET, RAF1 Selperactinib, Entrectinib, Larotrectinib, Zenocutuzumab	**Mismatch Repair Deficiency** TMB >10 Nivolumab, Pembrolizumab, Dostarlimab	**Surface Tropisms** Claudin 18.2 Tissue factor
***KRAS* Therapies** G12C Sotorasib, Adagrasib + other alleles: Pending	***BRAF* V600E^{MUT}** Dabrafenib, Encorafenib ***KRAS*^{WT}** Erlotinib	**BRCA1/2, PALB2** Platinum therapy PARPi: Olaparib, Rucaparib Acinar cancer	**Classical, Basal** GATA6 GEMPRED
BRCA1/2, PALB2 Platinum therapy PARPi: Olaparib, Rucaparib Immunotherapy	**HER2/ERBB2(+)** Trastuzumab deruxtecan	Germline (multigene), Somatic (+/-ctDNA) testing	

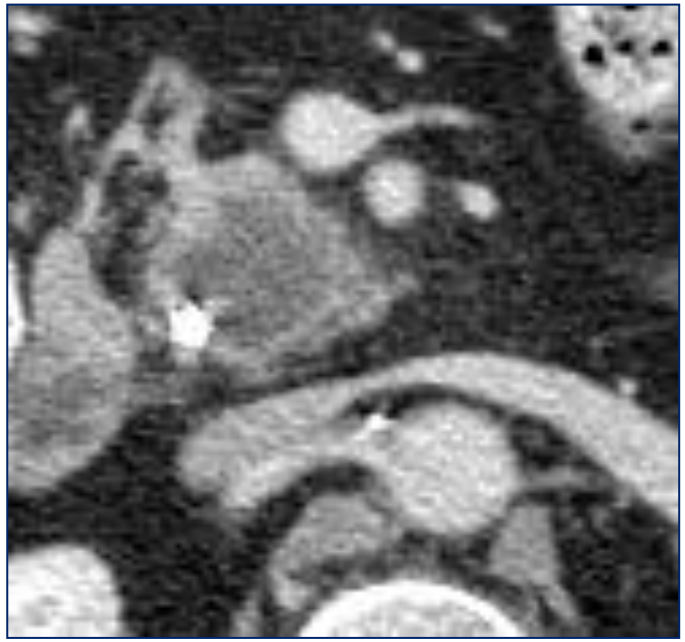
All referenced drugs either FDA approved, guideline endorsed

Localized PDAC

Pancreas Cancer: Early-Stage Disease

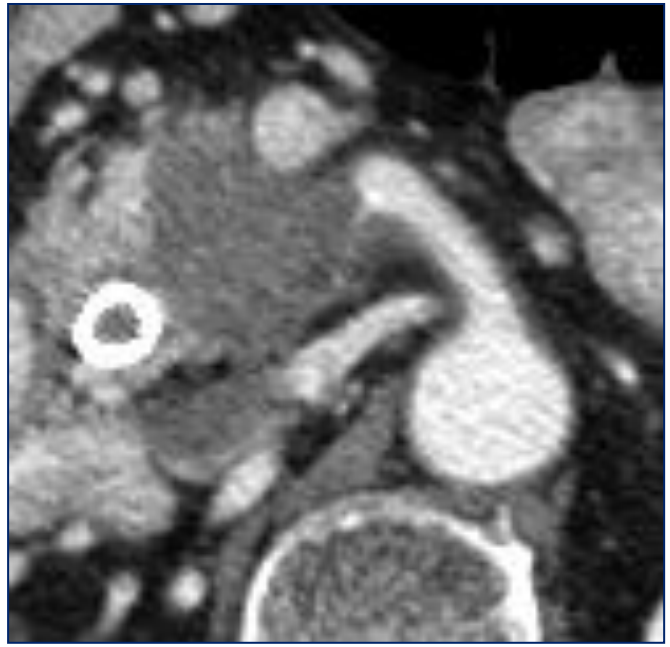
Spectrum of Localized PDAC

Resectable (15%)



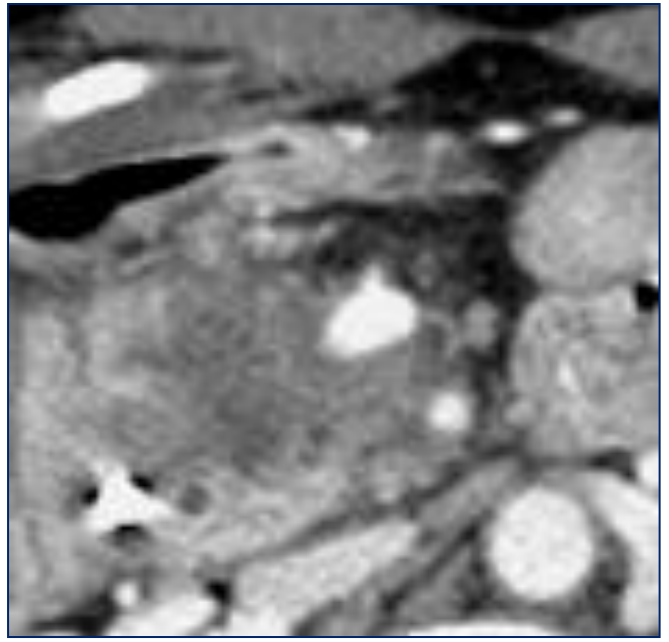
Upfront surgery
R0 resection

Borderline (15%)



High probability
R1 resection

Locally Advanced (25%)

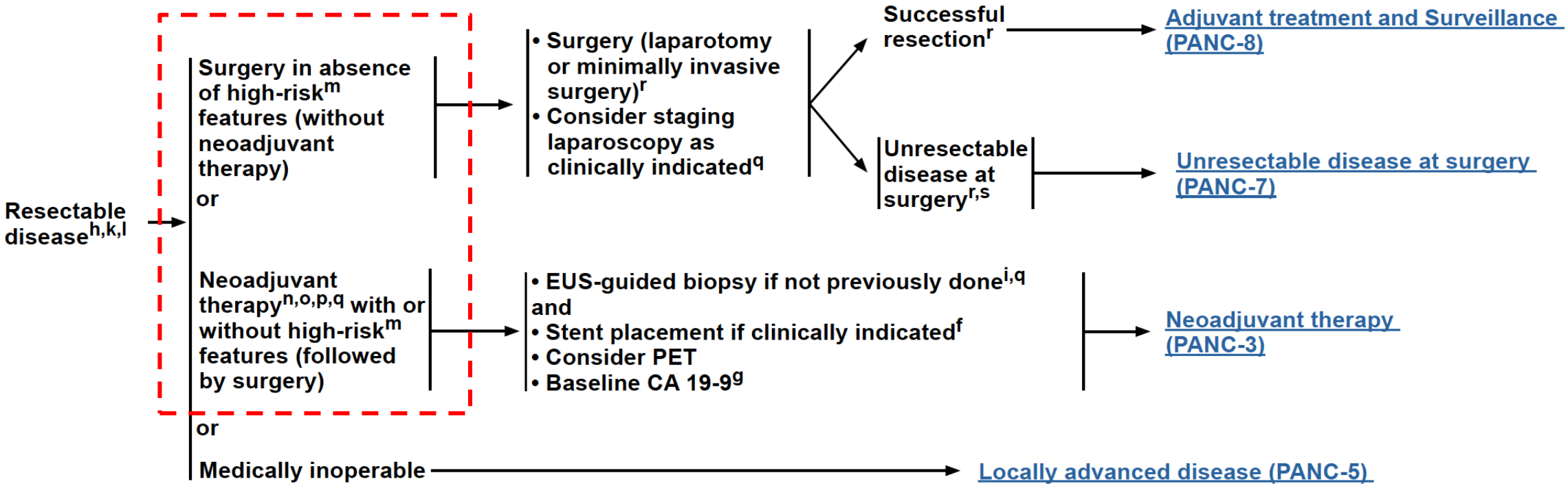


Inoperable
R2 (resection)

Adapted with thanks to @MattKatz

NCCN Guidelines Version 2.2025 PDAC: PANC-2

What Do the Guidelines Say for Resectable Disease?



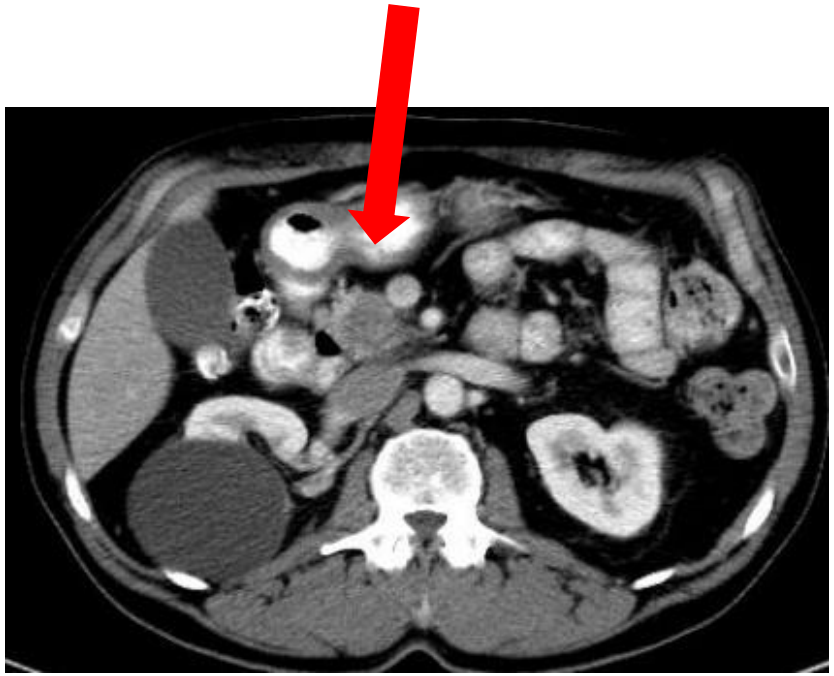
Neoadjuvant vs Adjuvant for Resectable PDAC

Level 1 evidence supports surgery followed by adjuvant mFOLFIRINOX;

Neoadjuvant therapy results in tumor shrinkage, N0, R0, less fistula, OS benefit in some studies; More randomized trials awaited
Current signal promising

Optimal neoadjuvant regimen, chemotherapy, chemoRT, both?
Remain to be defined

Key issue is **patient selection**

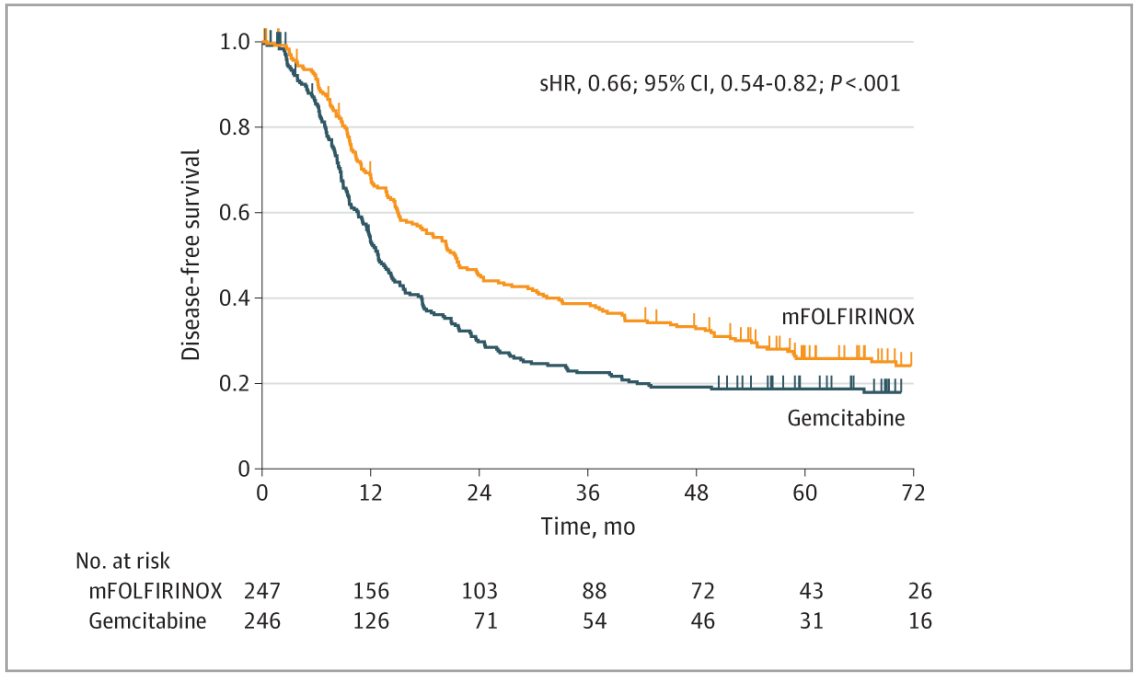


Summary Adjuvant Therapy: Improving Survival

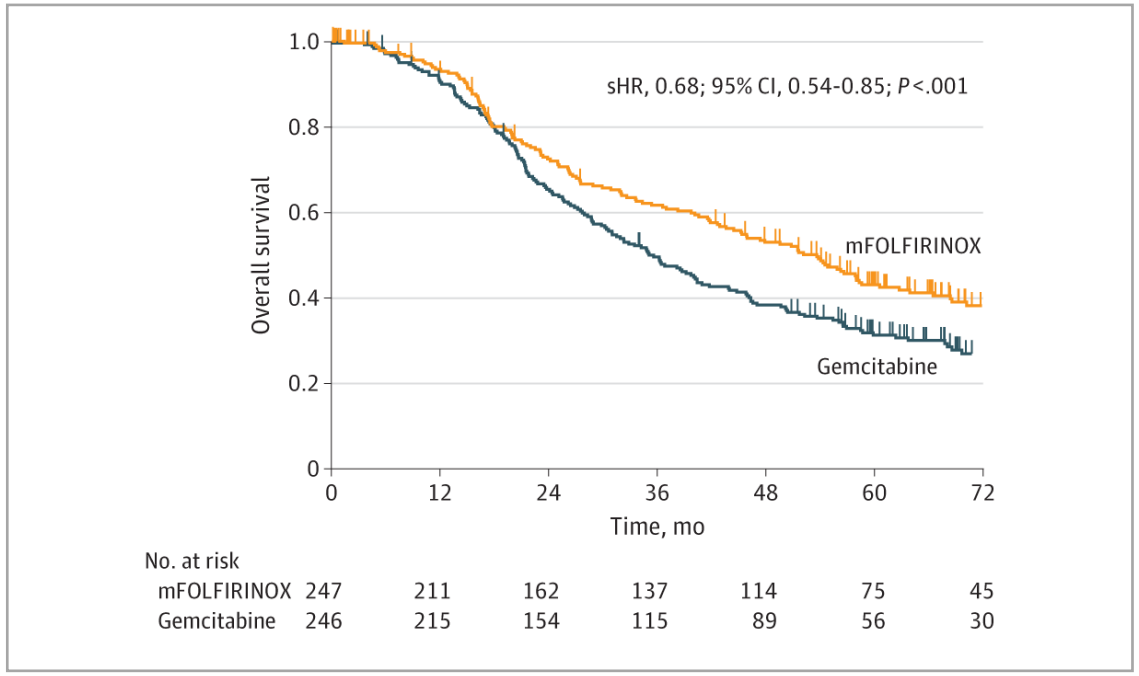
Study	N	Treatment Arms	Primary Endpoint	Result (months)
GITSG, 1985	43	5-FU/RT x 2 years Surgery	OS	20 vs. 11 p=0.03
EORTC, 1999	218	5-FU/RT Surgery	OS	24.5 vs. 19 HR 0.8, p=0.21
ESPAC-1, 2004	289	4 arms, 2x2 design: 5-FU vs. No comparison	OS	20.1 vs. 15.5 HR 0.71, p=0.009
ESPAC-1, 2004	289	4 arms, 2x2 design: RT vs. No comparison	OS	15.9 vs. 17.9 HR 1.28, p=0.05
CONKO-001, 2007	368	Gemcitabine Surgery	DFS	13.4 vs. 6.7 HR 0.55, p<0.001
RTOG 9704, 2008	451	5-FU + 5-FU/RT Gem + 5-FU/RT	OS	NA HR 0.93, p=0.51
ESPAC-4, 2017, **2024**	**732**	**Gemcitabine + Capecitabine** Gemcitabine	**OS**	**31.6 vs. 28.4** **HR 0.83, p=0.031**
PRODIGE 24, 2018	**493**	**mFOLFIRINOX** Gemcitabine	**DFS (2^0 OS)**	**21.6 vs 12.8 (54.4 vs 35)** **HR 0.58, p <0.0001**

Adjuvant: mFOLFIRINOX vs Gemcitabine 5-Year Outcome

Disease-Free Survival



Overall Survival



	N	Disease-Free Survival	Med Overall Survival	Five-Year OS
mFOLFIRINOX	247	**21.4 m** (17.5- 26.7)	**53.5 m** (43.5- 58.4)	**43.3%** (36.5%- 49.7%)
Gemcitabine	246	12.8 m (11.6- 15.2)	35.5 m (30.1- 40.3)	31.4% (25.5- 37.5%)
Hazard, P-value		0.66 (0.54- 0.82); p< 0.001	0.68 (0.54- 0.85); p= 0.001	

Conroy, T. New Engl J Med, 2018, Conroy, T. JAMA Oncology, 2022

Key Neoadjuvant/Adjuvant Trials: Resectable PDAC

Study	Inclusion	N	Treatment Arms	Endpoint
PREOPANC-3 Phase III NCT04927780	Resectable	378	Neoadjuvant FOLFIRINOX (8 pre-op) Surgery, Adjuvant FOLFIRINOX	OS **Accruing/ Completes 2025**
A021806 Phase III NCT04340141	Resectable	342	Neoadjuvant FOLFIRINOX (8 pre-op) Surgery, Adjuvant FOLFIRINOX	OS **Accruing/ Completes 2025**

Summary: Resectable/Resected PDAC 2025
No clearly Superior Approach….

Resectable PDAC

- Level 1 evidence surgery first
- Guidelines – Neoadjuvant option (ongoing randomized trials)
- mFOLFIRINOX (most studied)

Resected PDAC

- Adjuvant mFOLFIRINOX
- Adjuvant Gemcitabine +/- capecitabine
- Clinical trials

KRAS Directed Therapy

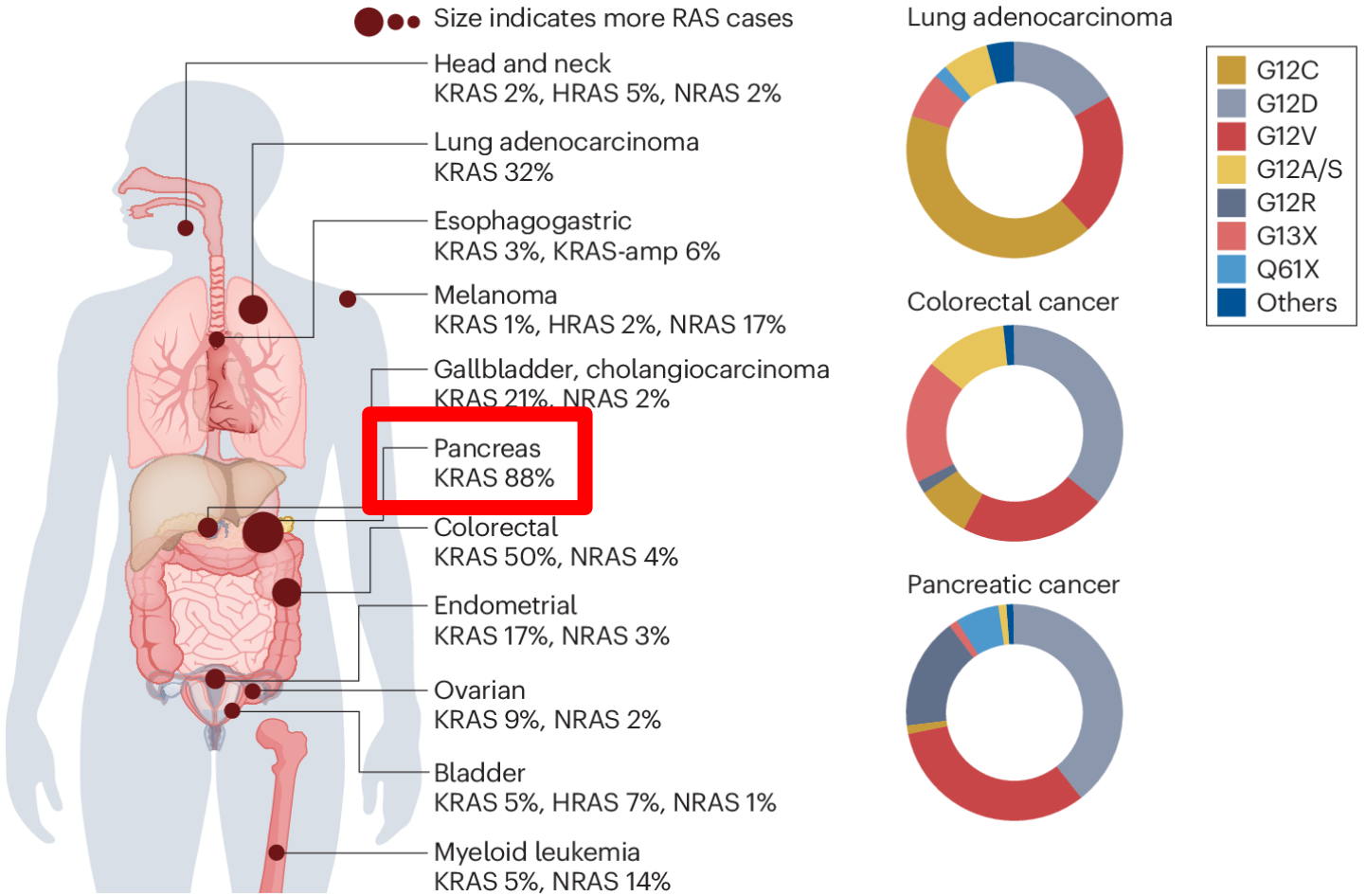
Pancreas Cancer: Genomics

Distribution of *RAS* Mutations in Cancer and Allele Frequency

KRAS mutations

~20% of cancers *RAS* mutation

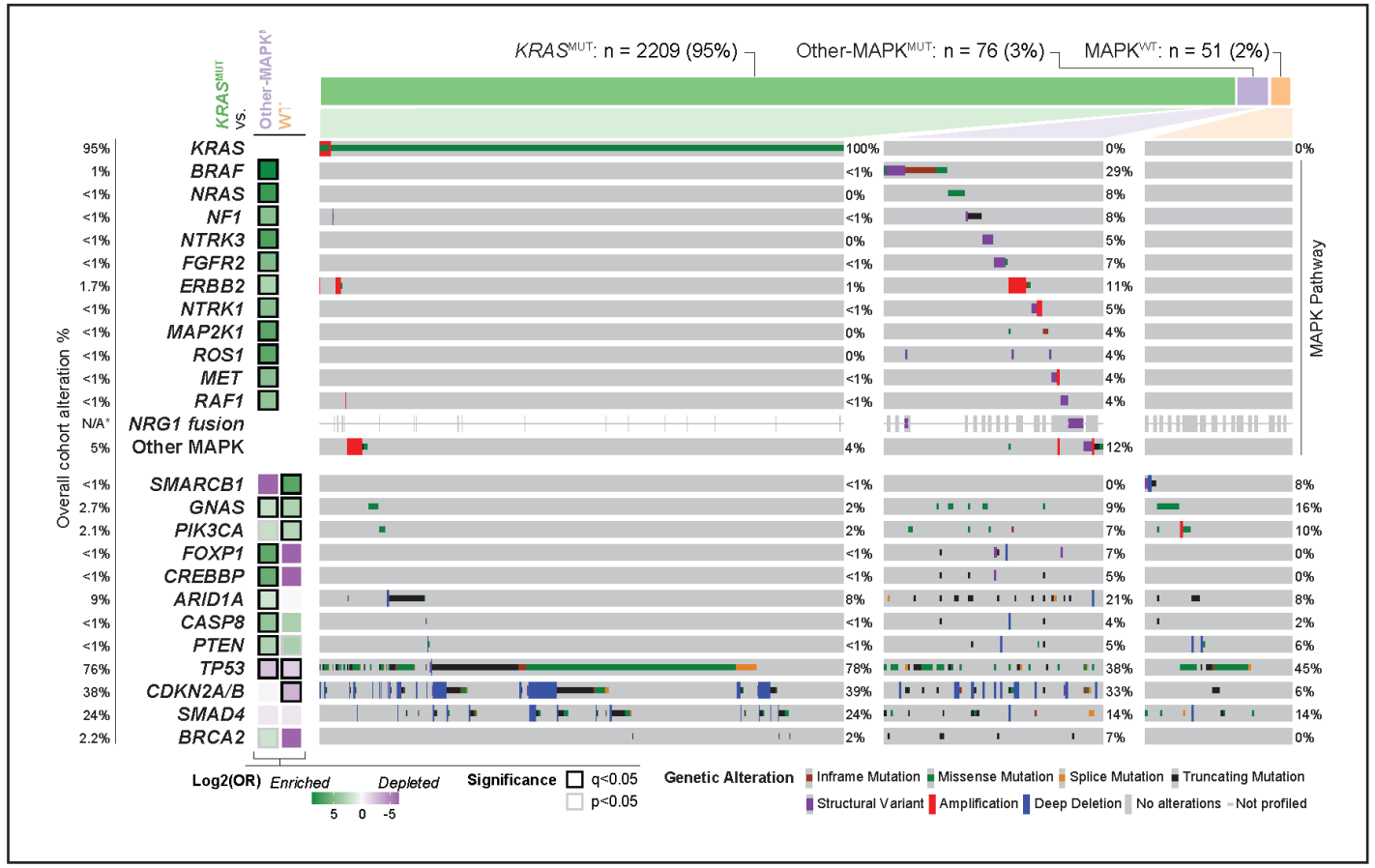
~75% of all RAS mutations in *KRAS*



Singhal, A, Li, B, O'Reilly, EM. Nat Med, 2024

Somatic Alteration Landscape in PDAC (N= 2,336)

Re-emphasizing the Importance of *KRAS*



Genomic Classifiers of PDAC

1. ***KRAS*^{MUT}(+) 95%**
2. ***KRAS*^{WT}, MAPK^{MUT}(+) 3% (Other-MAPK^{MUT})**
   - Enriched other MAPK genes
3. ***KRAS*^{WT} , MAPK^{WT}: 2%**
   - MAPK alterations (15%) (RNA-seq)
   - Enriched *SMARCB1* (7%)
   - Enriched g*ATM* (15%)
   - Enriched early onset
   - Enriched MSI-H, TMB-H

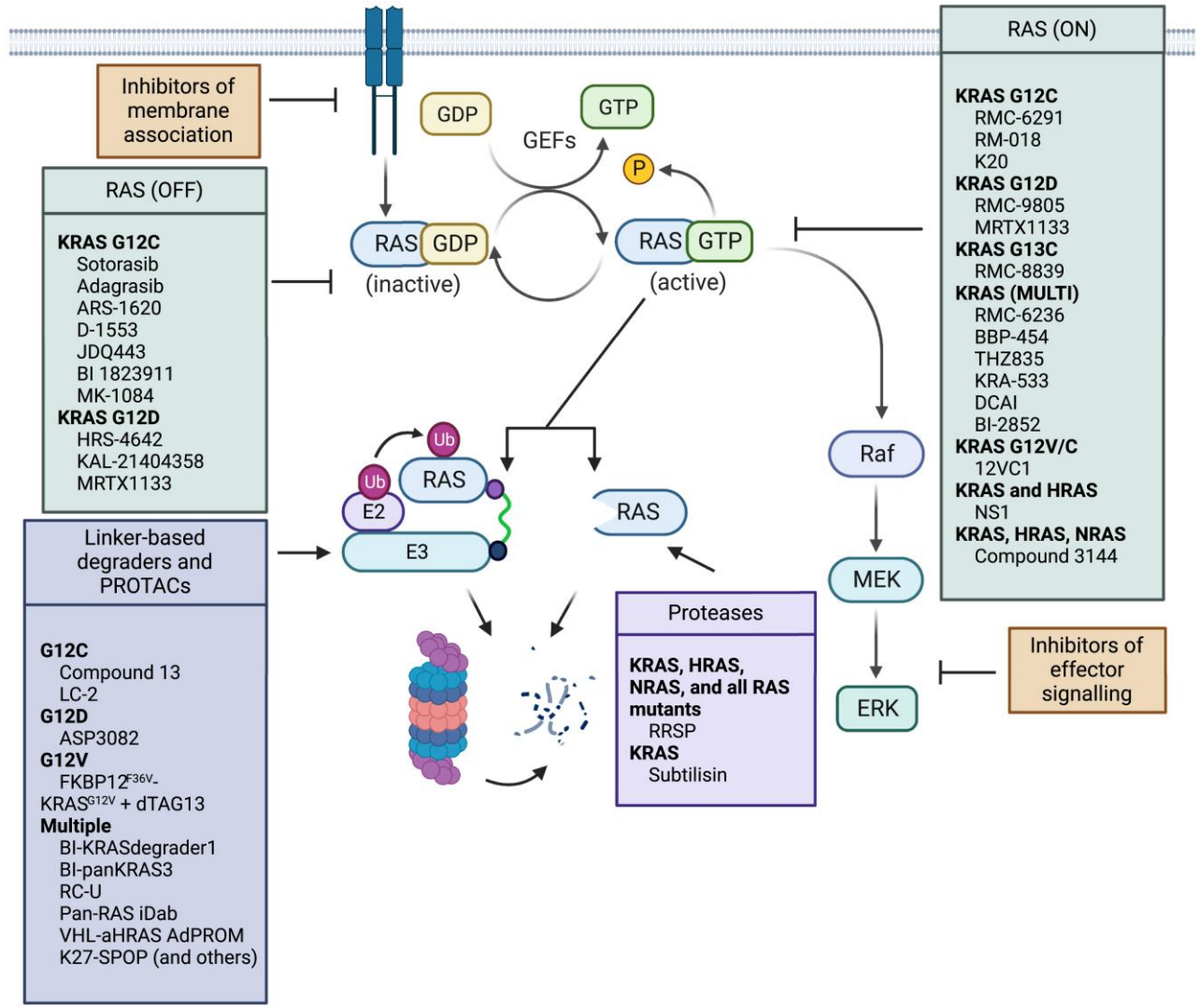
All *KRAS*^{MUT} are not alike

Varghese, A, Perry, M…Schultz, N, Berger, MF, Iacobuzio-Donahue, C, Bandlamudi, C, O'Reilly, EM. Nat Med, 2025

KRAS Targeting

KRAS Therapeutics

- Inhibitors RAS ‘off’ vs ‘on’
- Linker-based degraders PROTAC’s
- Proteases
- Indirect downstream inhibitors
- Immunotherapy



Esacher, TE, Sactchell, KJF. Mol Therapy, 2023

KRAS Directed & Other Immunotherapy

Many Challenges for Immunotherapy in PDAC

Immune suppressive environment

- MDSCs, tumor associated macrophages (TAM's); M2 phenotype, CAF's
- Cytokines (IL6,8,10 TGFβ, CSF, VEGF..,), chemokines (CXCL12)

Lack of effector CD8+ T cells (5-fold fewer vs 'hot' tumors)

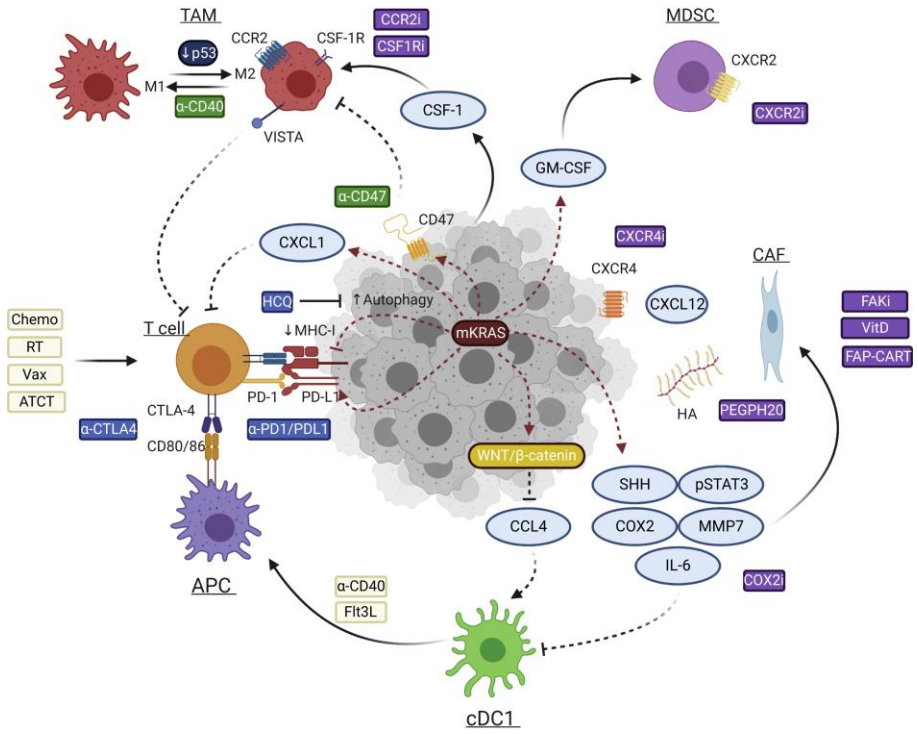
Predominant CD4+ Foxp3+ T cells (Tregs); Th2 T cells

Spatial distribution of cytotoxic T cells relative to cancer cells

Low TMB ~3.5 mut/Mb; low neoepitopes

Low rates of MMR/ MSI-H deficiency (1%)

PD-L1 (low, stroma) in TME suppresses TIL's



Bowers, JS. Oncol Rev, 2019
Carstens, JL. Nat Comm, 2017
Bear, AS. Cancer Cell, 2020
Blando, J. PNAS, 2019
Timmer, FEF. Cancers, 2021
Carpenter, E. J Surg Oncol, 2020

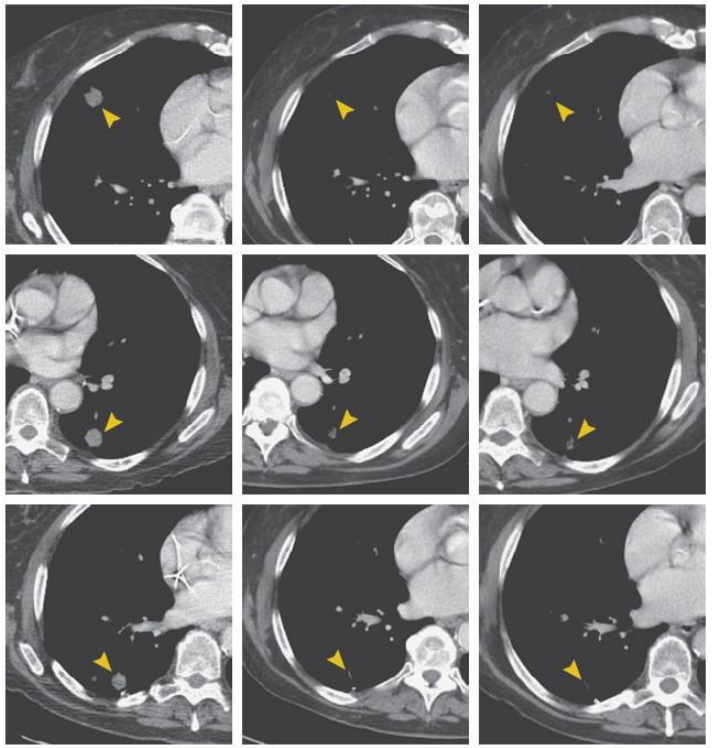
Pancreas Cancer: KRAS TCR Therapy

Targeting m*KRAS* in PDAC with T Cell Receptor Therapies

- Mutant *KRAS* promising public neoantigen target in PDAC
- Mutant KRAS peptides presented by MHC-1, recognized cytotoxic CD8+ T cells

- HLA C*08:02 restricted
  G12D *KRAS* TCR → PR in PDAC, CRC
  G12V

- Adoptive therapy challenges:
  - Select HLA's (e.g., HLA-C*08:02, A*11.01)
  - Select mutations
  - Logistics
  - Potential CRS
  - Cost, resources, time



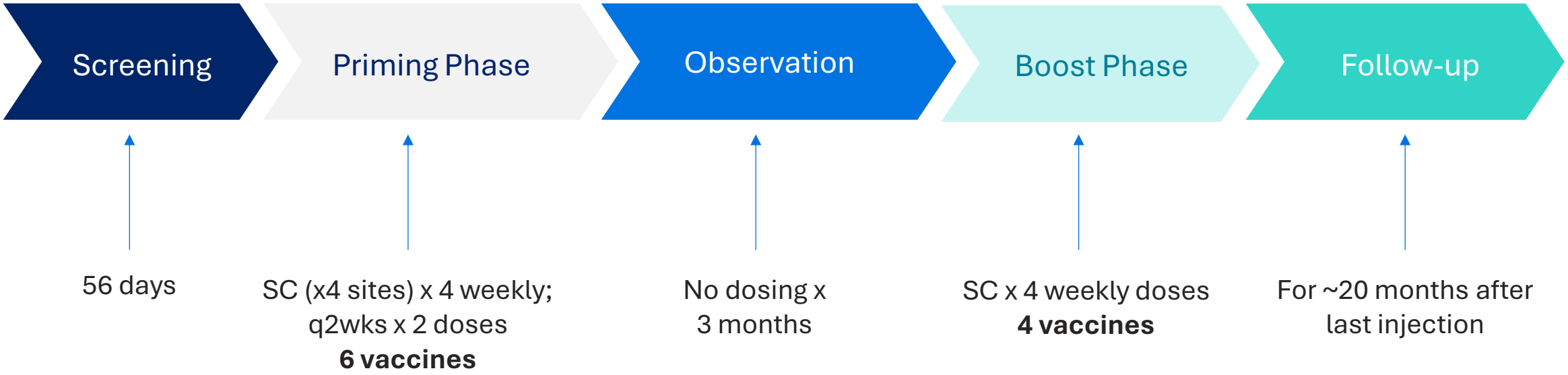
Leidner R. N Engl J Med, 2022
Tran E. N Engl J Med, 2016
Lu, D. Nat Commun, 2023
Poole, A. Nat Commun, 2022
Al, Q, Front Immunol, 2023
NCT03190941
NCT03704532
NCT06105021

RAS Immunotherapy

First in Human Phase I ELI-002 2P *KRAS* G12D/R

Stage II-IV NED Pancreas, Colon with MRD (+ctDNA or +CEA/Ca 19-9)

- Determine MTD or RP2D, safety, ctDNA clearance, Immunogenicity, RFS
- 'Adjuvant' trial – resected 'NED', +biomarkers
- Phase IA: Dose-escalation adjuvant 0.1 mg → 10 mg



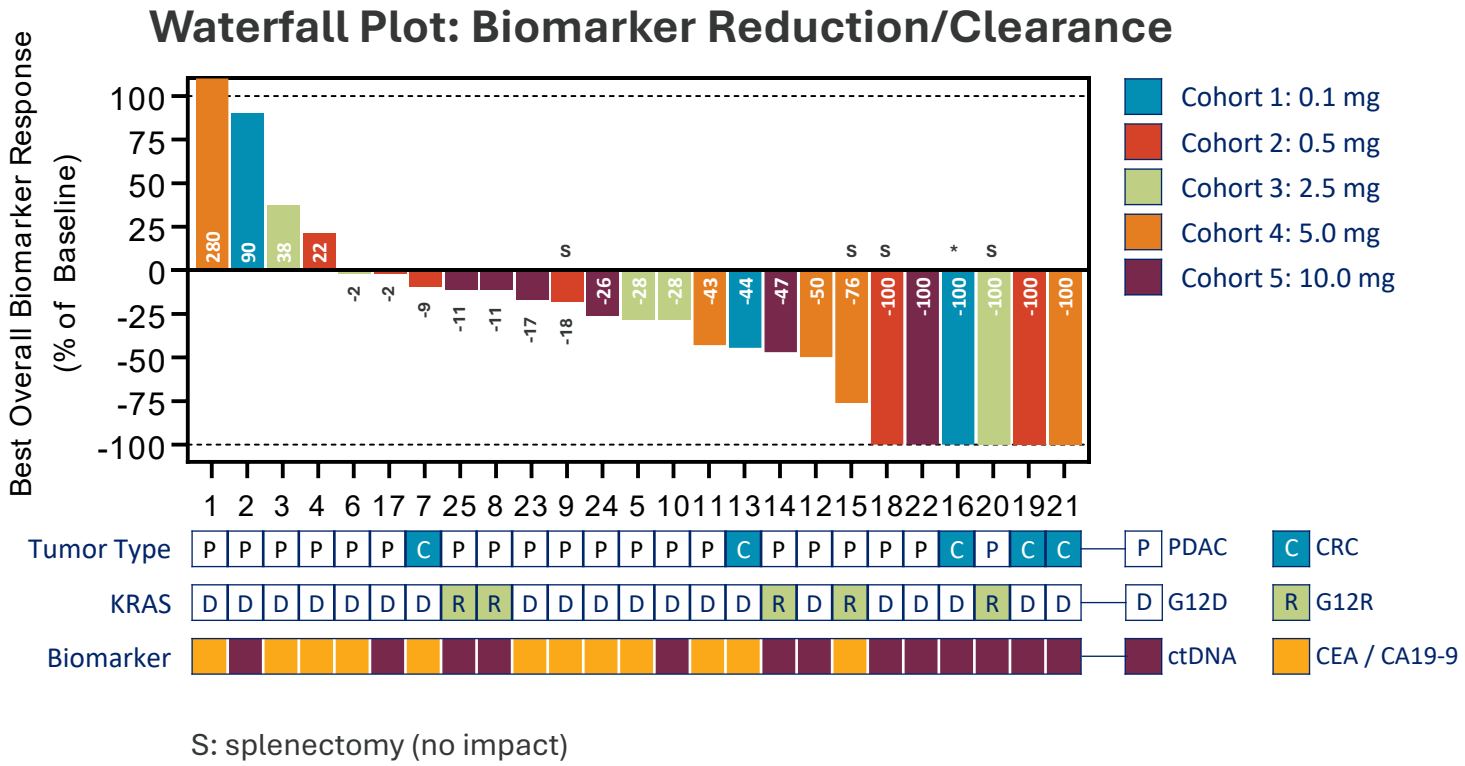
Pant S…O'Reilly EM et al. ASCO 2022. Abstract TPS2701. NCT04853017
O'Reilly EM…Pant S et al. ASCO 2023. Abstract 2528

RAS Immunotherapy

Phase IA: Results ELI 002 2P Pancreas, Colon

Immune and Clinical Responses in Pancreas, Colon, *KRAS* G12D and G12R

- N= 25 enrolled; 5 cohorts
- N= 20 PDAC; N= 5 CRC
- All pretreated; stage III, IV NED
- 21/25 (84%) decline ctDNA or CEA/Ca 19-9 from baseline
- 6/25 (24%) ctDNA clearance
- No DLT; skin reactogenicity, fatigue



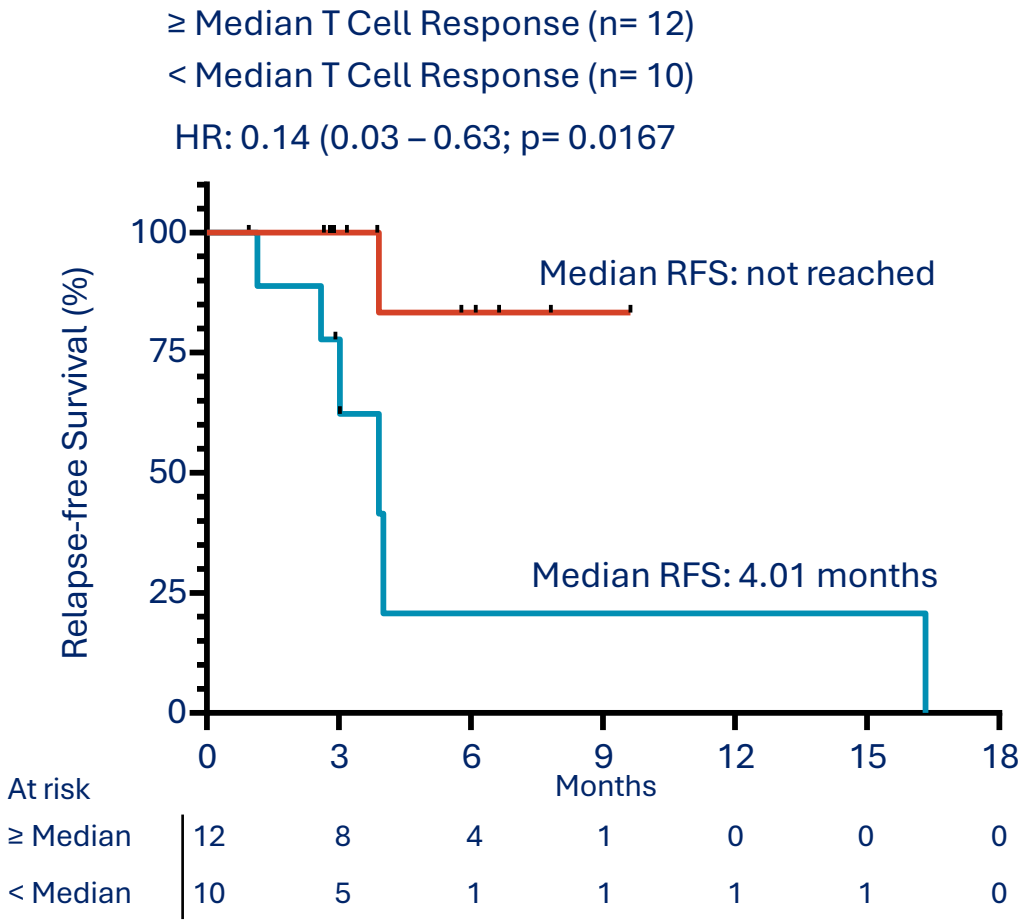
S: splenectomy (no impact)

O'Reilly EM…Pant S et al. Abstract 2528, ASCO, 2023
Wainberg, Z. AACR Pancreas, 2023
Pant, S…Haqq, C, O'Reilly, EM.. Nature Med, 2024

RFS > Median T cell Response Correlates with Outcome

Immunogenicity Signal Correlates with Outcome to ELI-002 2P

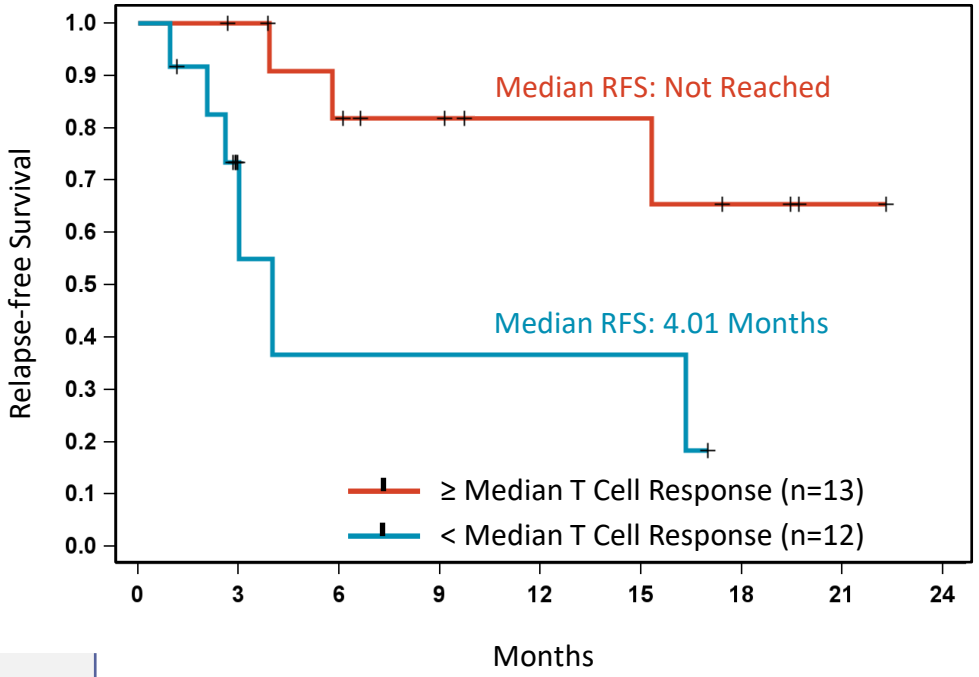
- Strength T cell response to ELI-002 2P strongly correlated with RFS/death
- At median f/up 8.5 m:
  For ≥ median T cell response: Not reached
  For < median T cell response: med RFS 4.01 m
  HR 0.14 (0.03 – 0.63)
- Median Relapse free survival: 16.3 m
- Extended data with ELI-002 7P, higher peptide dose
  More immunogenic
  Preliminary correlation with DFS



Pant, S…Haqq, C, O'Reilly, EM.. Nature Med, 2024
Unpublished, 2024

Phase I: Immune Response 2P: Relapse Free Survival

Strong Signal Persisting with Time – 1 Year Later



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

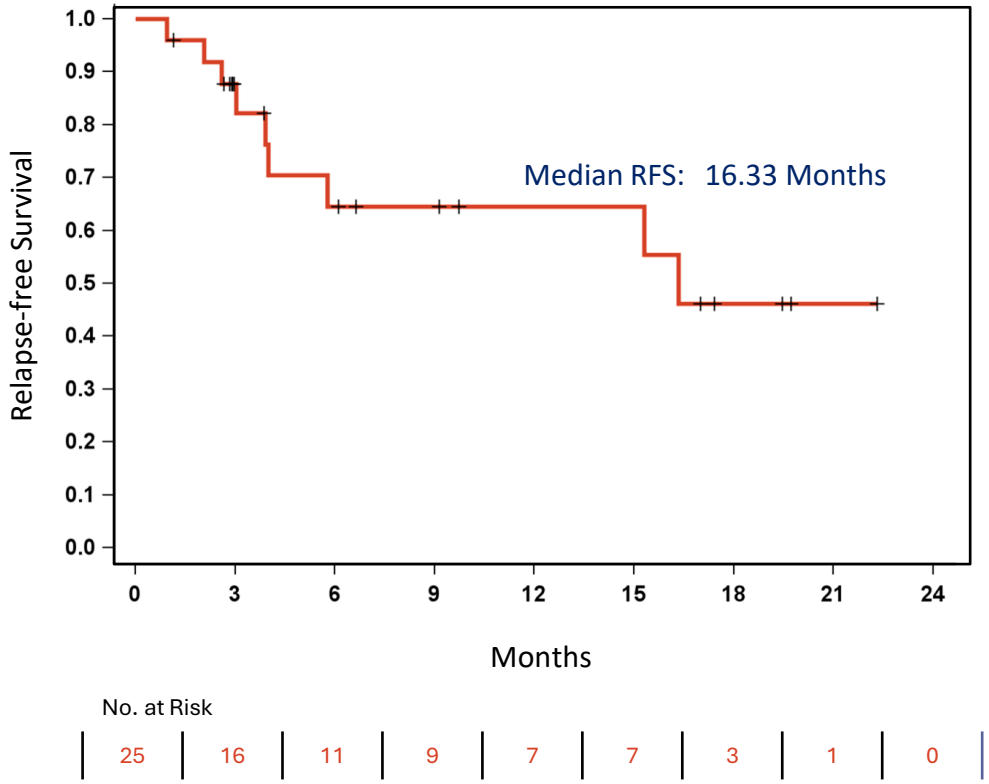
	Data Cut off	Sept 2023	Sept 2024
Median RFS (Months)	≥ Median T Cell	Not Reached	Not Reached
	< Median T Cell	4.01	4.01
	HR (95% CI)	0.142 (0.0321, 0.6278)	0.226 (0.0552, 0.9277)
	P-value	0.0167	0.0184

Pant, S. ESMO-IO, 2024

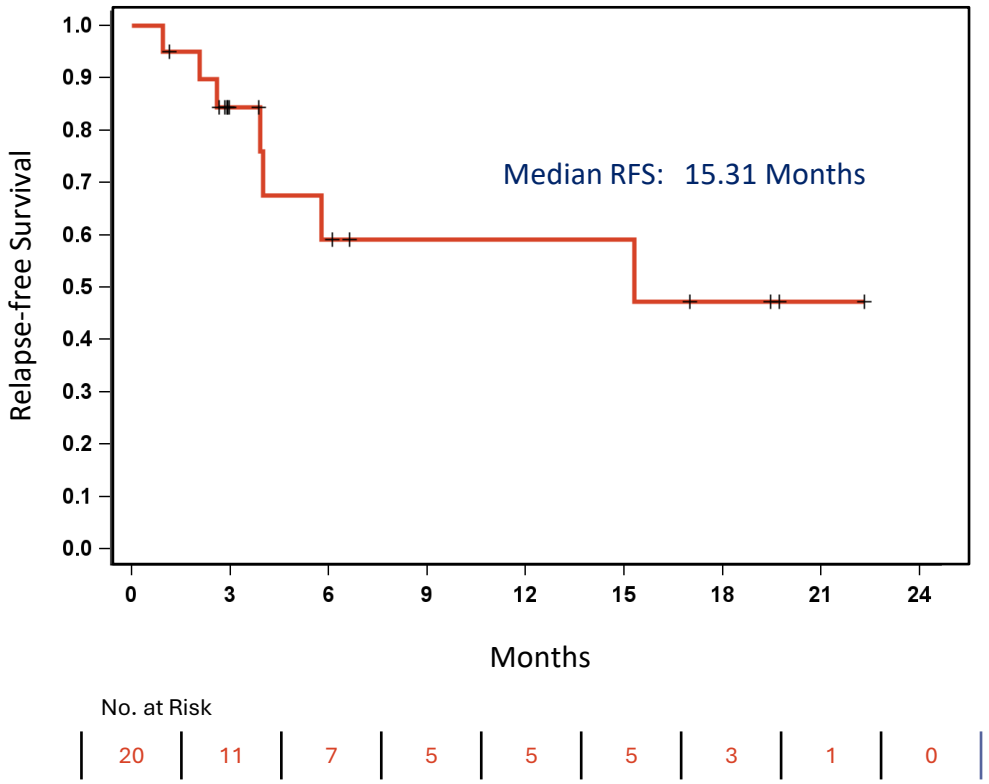
Relapse Free Survival 2P: All Patients; PDAC Subgroup

Median Follow-up: ~20 months

All Patients (N= 25)



PDAC Subgroup (N= 20)



Median OS All Cohort/PDAC: 29 m

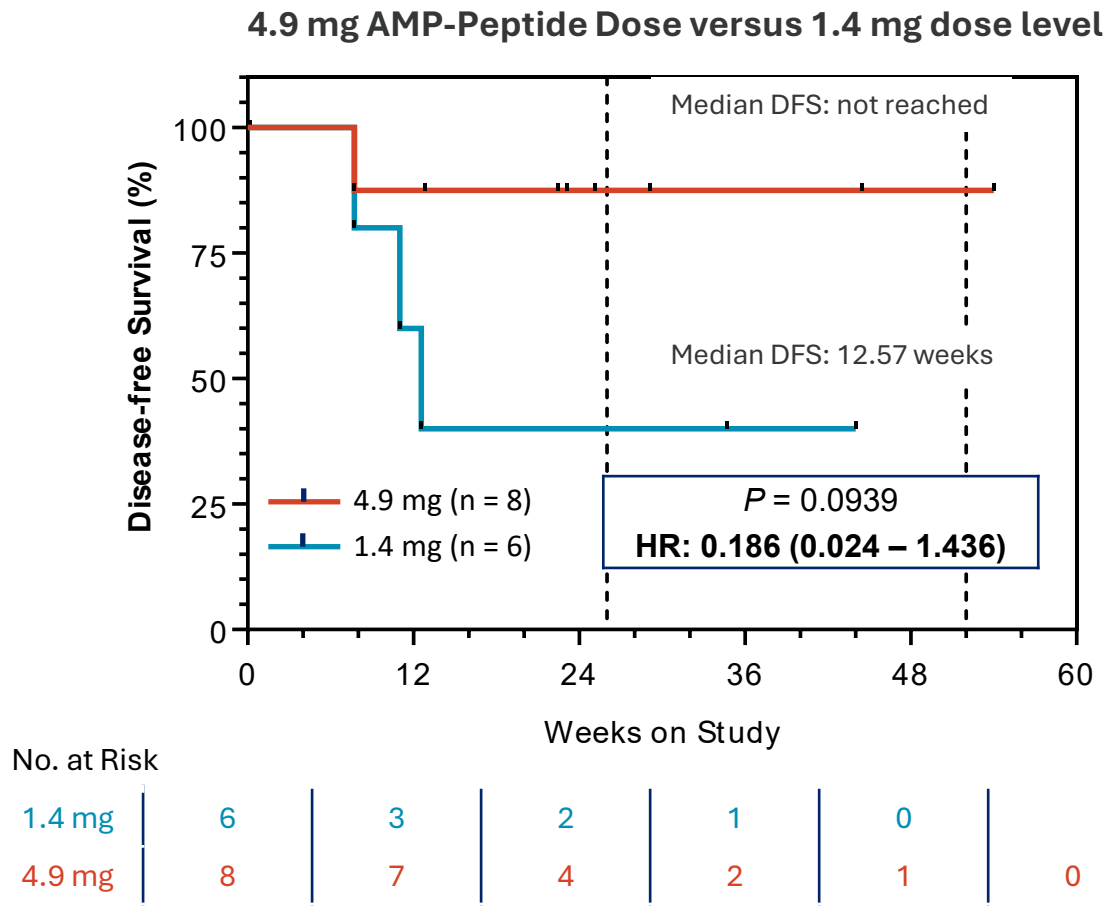
Pant, S. ESMO-IO, 2024

RAS Immunotherapy

Phase IA: ELI-002 7P Disease-Free Survival (Preliminary)

7P Similar to 2P – But More Potent Immunogenicity

- Improved DFS 4.9 mg vs 1.4 mg peptide dose
- Median DFS not reached for 4.9 mg dose
- Improved DFS also associated with
  - Median T cell fold change
  - Biomarker response



Data cut-off May 2024

Pancreas Cancer: RAS Immunotherapy

AMPLIFY-201 7P: Randomized Phase II Trial Resected PDAC

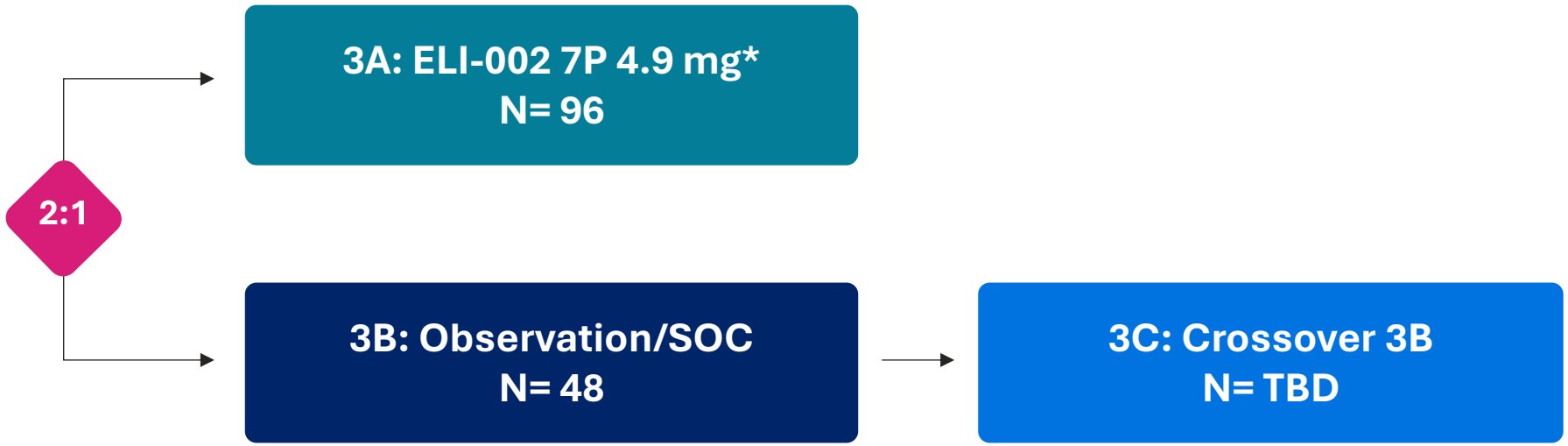
Accrued Q4 2024

Resected PDAC; completed standard (stage I- III)

***KRAS/NRAS*(+) G12/G13**

ECOG 0-1

ALC ≥ 1.0



Primary endpoint: Disease-free survival (investigator); 80% power

Secondary: Biomarker reduction & clearance, 1-year DFS, median OS, safety, ORR (crossover)

Exploratory: Immunogenicity ELI-002 7P to baseline

Stratification: N0 vs N1

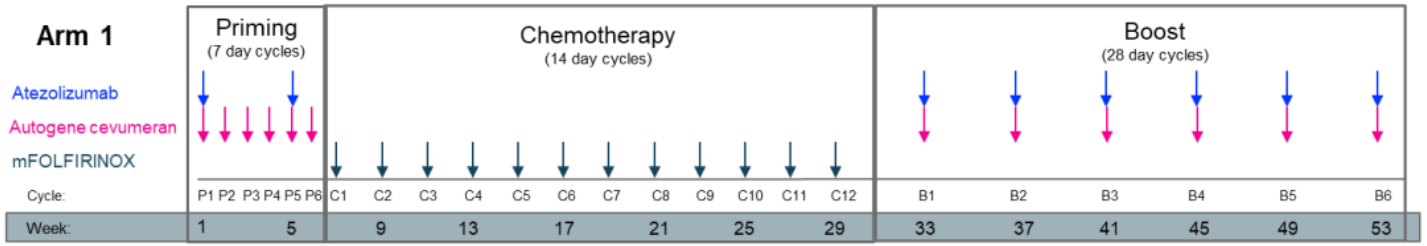
*7-Peptide: G12D, G12V, G12R, G12C, G12A, G12S, G13D

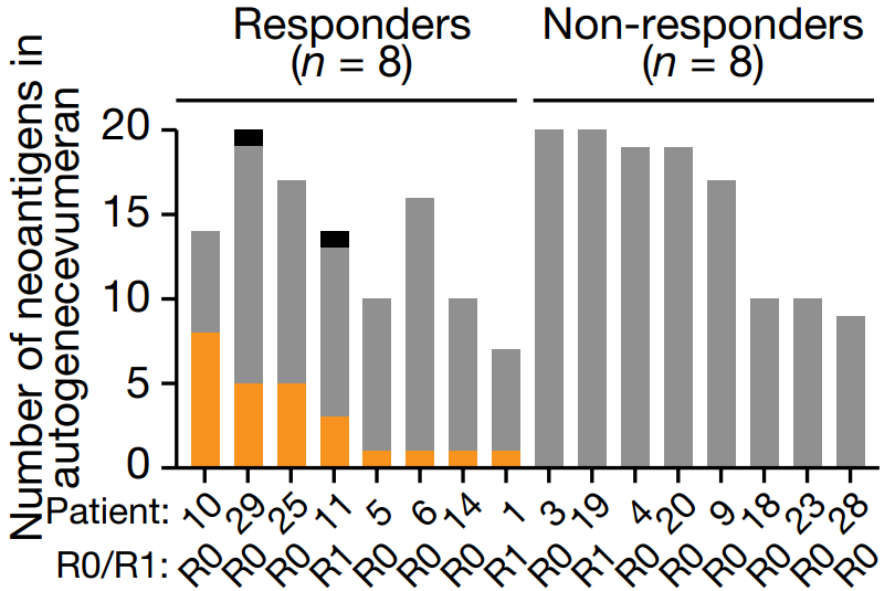
Wainberg, Z...O'Reilly, EM. Trials In Progress, Gastrointestinal Cancers Symposium, 2024
NCT05726864

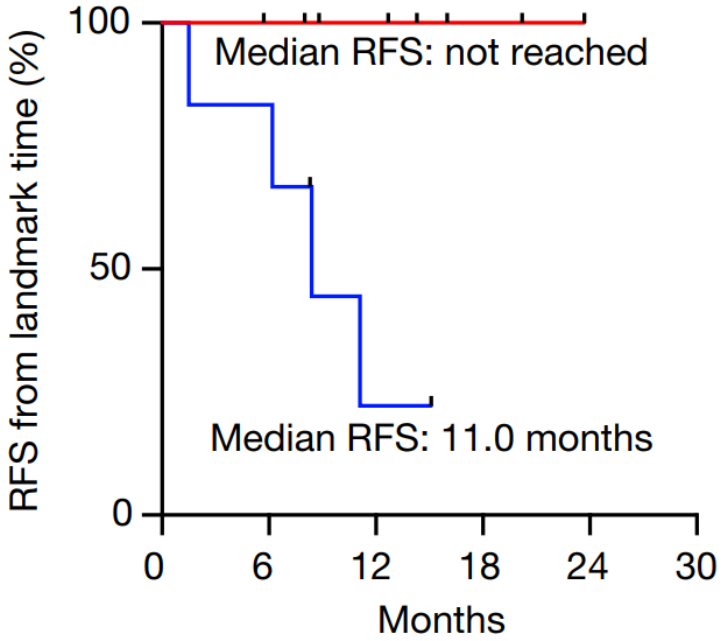
Phase I Autogene Cevumaren, Atezolizumab, mFFX

Resected Pancreas Cancer: Immune Response Correlates with RFS

N= 16 treated (28 screened)
50% immune responders







Rojas, L..Balachandran, V. Nature, 2023

Pancreas Cancer: Adjuvant Immunotherapy

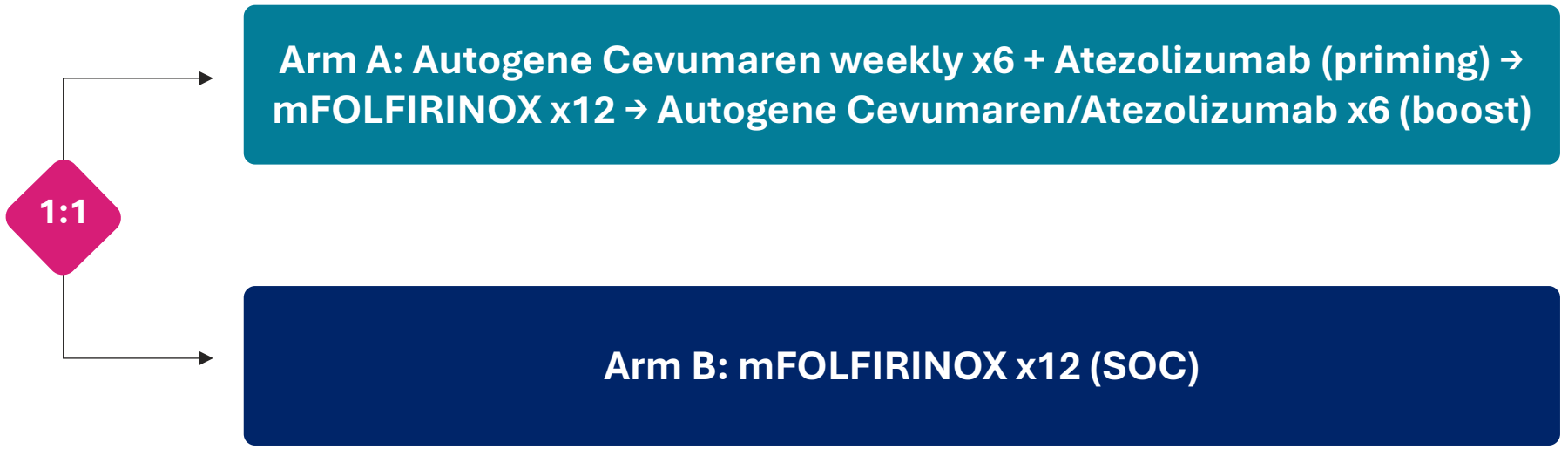
IMCODE003: Randomized Phase II: mFOLFIRINOX +/- Personalized Neoantigen Vaccine (mRNA) + Atezolizumab (ongoing)

Resected PDAC
2-part screening

ECOG 0-1
Ca 19-9 ≤ 180 U/ml

1:1 Randomization

N= 260



Primary endpoint: Disease-free survival (investigator)

Secondary: DFS @12, 24, 26 m; OS, OS @3, 5 years; Safety

Exploratory: QoL; QLQ-C30, EORTC PAN-26, PRO-CTCAE; PK; Immunogenicity

Stratification: R0 vs R1, N0 vs N1

G044479
NCT05968326

KRAS Directed Immunotherapy in PDAC

PDAC is an Immune Responsive Disease

Promising early phase I data for KRAS ELI-002 2P, 7P peptide vaccines (N= 39)
Safe, no DLT's, RP2D identified

Potent lymph node targeting, CD4+, CD8+ T cell responses,
T cell cytotoxicity, antigen spreading (tumor specific mutations)

mKRAS specific T cell response correlates with reduction in biomarkers,
reduced risk of relapse/death

Randomized phase II in PDAC accrued (ELI-002 7P)
Planned: Neoadjuvant trial resectable PDAC; +chemo, +/-ICB

Opportunities for KRAS Immunotherapy in PDAC

Adjuvant/ Neoadjuvant

Neoadjuvant therapy

Await randomized phase II

Combination data with standard of care therapy

Locally Advanced/ Metastatic Disease

Maintenance therapy after ‘debulking’

Stage IV NED/ oligometastatic disease setting

The Future

Targeting multiple epitopes

Combination with multiple emerging therapeutics

Challenge – prioritizing rationale combinations

ELI-002 7P Phase 2 Trial Protocol Design, Upcoming Interim Data, and High-Level Statistical Plan

Christopher Haqq, MD, PhD

Chief Medical Officer

ELI-002 highlights based on Phase 1 data

- ELI-002 is a Lymph Node Targeted mKRAS immunotherapy comprised of 7 KRAS targeted peptides and proprietary AMP-CpG Adjuvant
- Preliminary ELI-002 data suggests it may have the potential to change the treatment paradigm in the PDAC adjuvant setting
- Phase 1 trials included dose-ranging for both peptide and adjuvant components of ELI-002
- Data from both Phase 1 trials have shown:
  - ELI-002 was well tolerated at all dose levels, with **no DLTs and no treatment-related SAEs observed**
  - **Phase 2 dose 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 Phase 2 dose with immune response targeting personal tumor neoantigens i.e., expansion of T cells specific to personalized tumor antigens not targeted by immunotherapy
  - **Strength of ELI-002 T cell response correlates with a reduction in the risk of progression or death**

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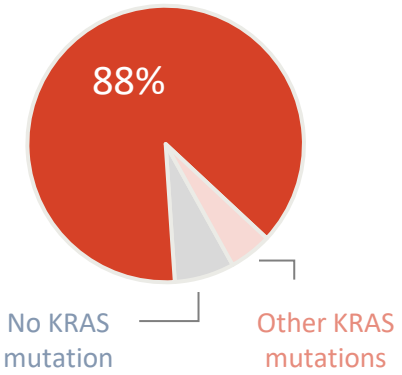
Pant, et al 2024. "Lymph-node-targeted, mKRAS-specific amphiphile vaccine in pancreatic and colorectal cancer: the phase 1 AMPLIFY-201 trial." Nature Medicine 30(2): 531-542.
Craig E. Devoe et al. 2024.. ASCO Annual Meeting Abstract 2636. Journal of Clinical Oncology 42, 2636-2636.

The Addressable KRAS-mutant Market – A Significant Opportunity

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

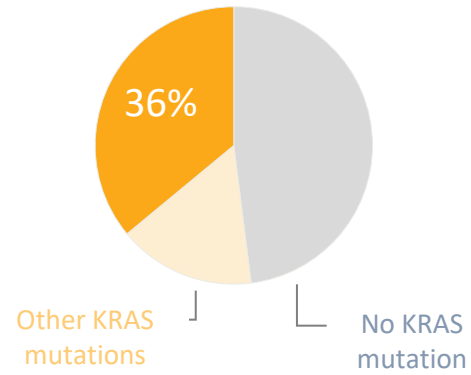
Pancreatic Ductal Adenocarcinoma

ELI-002 addressable
Incidence: ~128k



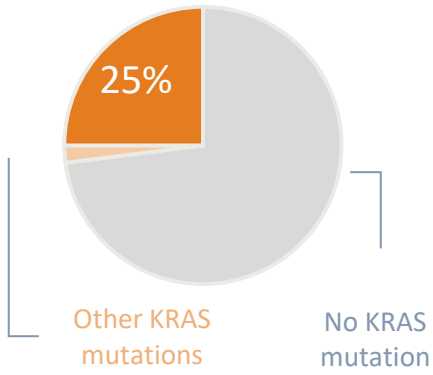
Colorectal Cancer

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

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 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

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- 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 7P | Phase 2

ELI-002 Randomized Phase 2 PDAC Trial Design

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

CLINICAL STUDY OVERVIEW: NCT05726864

Monotherapy (no chemo, CPI combo)

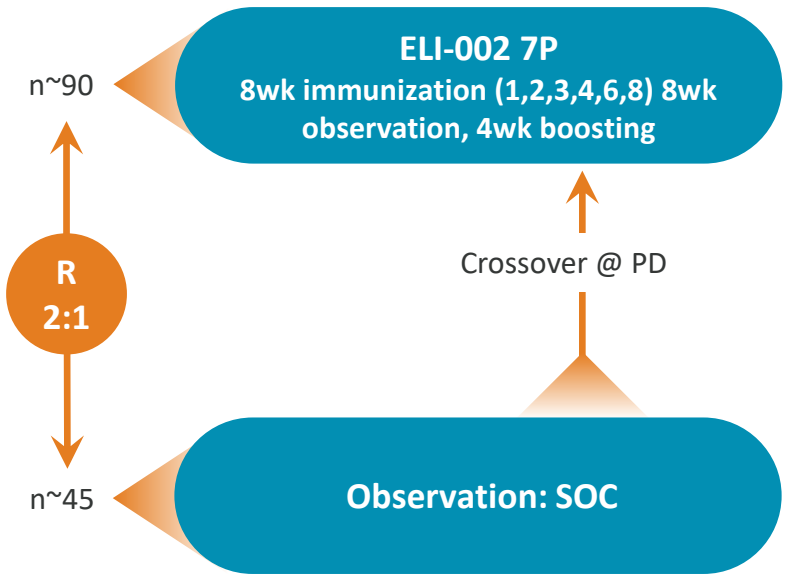
- ✓ mKRAS: Expanded Antigen Coverage

  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, 24 US sites

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)

High Level ELI-002 Phase 2 Statistical Plan

ELI-002 7P | Phase 2

Event driven interim analysis designed to provide an interim look at efficacy to potentially accelerate development

- In phase 2, n=144 pts were randomized 2:1 to ELI-002 7P Vs. standard of care (observation)
- Trial designed to **reduce risk** by using:
  - Stratification by nodal status (node negative versus node positive) avoids imbalance in prognostic factors
  - A weighted average of MRD positive and MRD negative was used for control arm median Disease-Free Survival (DFS)*
  - 80% power for primary endpoint DFS

- **Primary endpoint:** Disease Free Survival (DFS)
- **Secondary endpoints:**
  - Overall Survival (OS), (crossover permitted)
  - 1-year DFS rate
  - Biomarker response rate

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**Groot et al. Clin Cancer Res. 2019 25(16):4973-4984; Botta et al., Oncologist. 2024 29(10):859-869. Sohal et al. JAMA Oncol. 2021 7(3):421-427; Tempero J Clin Oncol. 2023 41(11):2007-2019*

Q&A Discussion

Closing Remarks

Robert Connelly, CEO