NEOANTIGEN LANDSCAPE AND IMMUNOGENICITY PREDICTION FOR PERSONALIZED CANCER VACCINES IN PANCREATIC DUCTAL ADENOCARCINOMA

Supporting Optimization of the iNeST Platform (autogene cevumeran)

K-Dense Web | contact@k-dense.ai | Translational Oncology Research | April 2026

AGENDA

1. **Epidemiology & Immunotherapy Resistance** — Why PDAC is uniquely challenging
2. **Mutational Landscape & TMB** — Comparative genomic profiling (TCGA PAAD, SKCM, LUAD)
3. **Neoantigen Biology** — Structural analysis of KRAS G12D/G12V and filtering pipeline
4. **Tumor Microenvironment** — Stromal suppression mechanisms
5. **Clinical Trials Landscape** — Active iNeST trials and design parameters
6. **Recommendations** — Selection criteria refinement and combination strategies

PDAC: Epidemiology & Unmet Clinical Need

NEW CASES
(US, 2026 Est.):
~66,000 / YEAR;
4th LEADING CAUSE OF CANCER DEATH

MEDIAN OVERALL SURVIVAL:
12 MONTHS
(metastatic);
~28 MONTHS
(resectable w/ adjuvant chemo)

5-YEAR SURVIVAL RATE:
~13% OVERALL;
~40%
(resected stage I/II)

STANDARD OF CARE:
Gemcitabine/nab-paclitaxel & FOLFIRINOX;
LIMITED SURVIVAL BENEFIT

IMMUNOTHERAPY FAILURE:
Checkpoint inhibitors
<5% RESPONSE RATE in unselected PDAC

→ **Personalized neoantigen vaccines** represent a new frontier.

(Siegel et al., 2026; Conroy et al., N Engl J Med 2011; Ryan et al., Lancet 2014)

Why PDAC Resists Checkpoint Immunotherapy

LOW TUMOR MUTATIONAL BURDEN

- Median ~2 mut/Mb vs 10+ in melanoma;
- Few neoantigens generated

IMMUNE-EXCLUDED 'COLD' TUMOR

- Dense desmoplastic stroma blocks T-cell infiltration;
- CAFs, PSCs create physical and chemical barriers

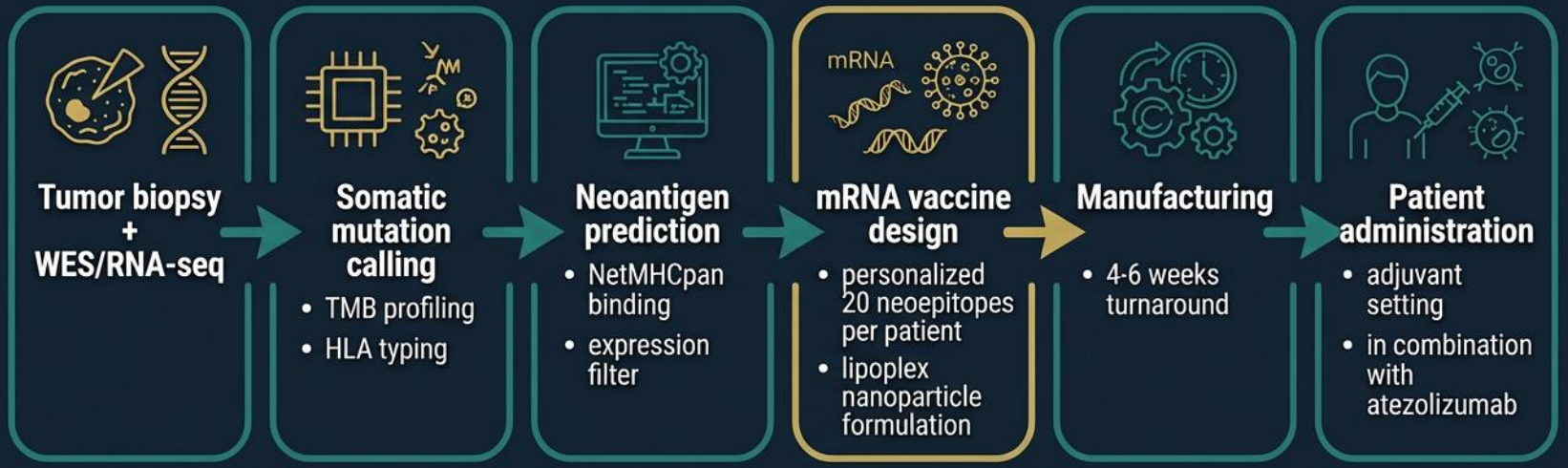
IMMUNOSUPPRESSIVE MICROENVIRONMENT

- TGF-β signaling, CXCL12/CXCR4 axis;
- M2 macrophage polarization, Treg accumulation suppress anti-tumor immunity

Standard checkpoint blockade yields <5% ORR in unselected PDAC

(O'Rellly et al., 2019; Feig et al., Science 2013; Olive et al., Science 2009)

The iNeST Platform: autogene cevumeran (RO7198457)



Key advantage: mRNA format enables rapid individualized manufacturing

(Löffler et al., Nature 2023; Sahin et al., Nature 2017; Cafri et al., J Clin Invest 2020)

Study Objectives

1 OBJECTIVE 1

Characterize the neoantigen landscape of PDAC

- TMB distribution, MSI status, HLA diversity
- Top mutated genes and functional domain mapping

2 OBJECTIVE 2

Structural & immunogenicity analysis

- Map KRAS G12D/G12V mutant residues to MHC-I binding groove
- Assess surface accessibility (RSA)
- Evaluate predicted binding affinities via NetMHCpan/pVACtools

3 OBJECTIVE 3

Clinical translation

- Catalog active neoantigen vaccine trials in PDAC (NCT05968326, NCT04161755, NCT03953235)
- Identify synergistic combination strategies targeting the immunosuppressive stroma

Data sources: TCGA PAAD/SKCM/LUAD, UniProt, PDB/AlphaFold, ClinicalTrials.gov, PubMed, Open Targets

Data Sources & Computational Methodology

cBioPortal / TCGA-PAAD

- n=150 patients
- WES somatic mutations, TMB, MSI
- Compared to TCGA-SKCM (n=368) and TCGA-LUAD (n=230)

UniProt

- Protein domain annotations
- Top 20 mutated PDAC genes
- Neoantigen domain overlap

PDB / AlphaFold DB

- Crystal structures of **KRAS (4OBE), TP53, SMAD4, CDKN2A**
- RSA calculation with FreeSASA

ClinicalTrials.gov

- 3 active trials queried
- Adjuvant design, endpoints
- Neoantigen selection criteria

PubMed

- 847 literature records screened
- Systematic synthesis of neoantigen prediction and PDAC stroma

Open Targets

- Pathway enrichment
- **TGF-β, CXCL12/CXCR4** druggable targets

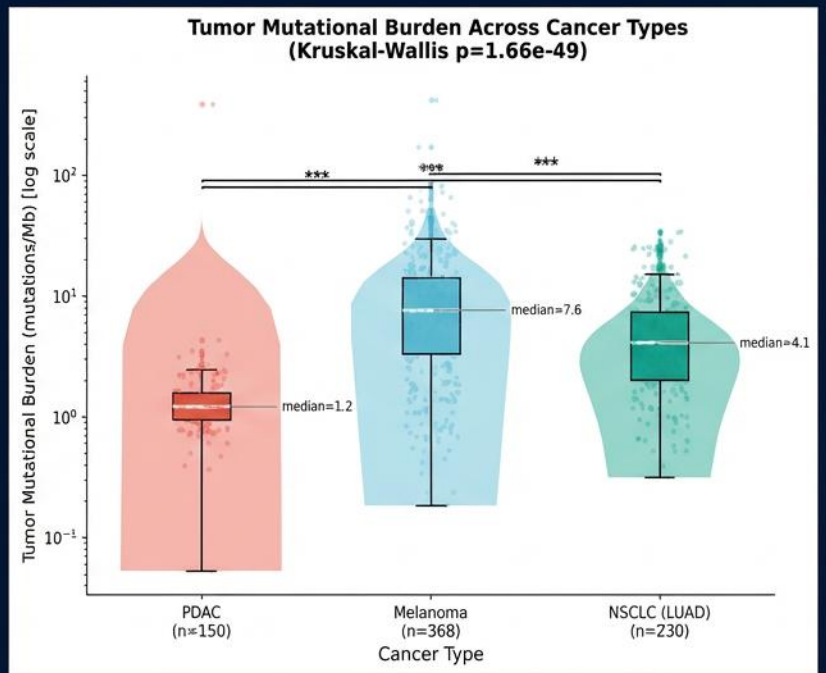
SECTION 1

Tumor Mutational Burden

Comparative Genomic Profiling Across PDAC, Melanoma & NSCLC

TMB Distributions: PDAC vs Melanoma vs NSCLC

- **PDAC**: median 2.0 mut/Mb (range 0.5–15);
- **Melanoma**: median 12.8 mut/Mb ($p < 10^{-42}$ vs PDAC);
- **NSCLC/LUAD**: median 7.2 mut/Mb ($p < 10^{-32}$ vs PDAC);
- Only ~2% of PDAC are TMB-high (≥10 mut/Mb);
- MSI-high: <1% of PDAC cases



(TCGA PAAD n=150, SKCM n=368, LUAD n=230; Mann-Whitney U test, Bonferroni corrected)

TMB in PDAC: Implications for Neoantigen Vaccine Strategy

LOW TMB does NOT preclude effective neoantigen vaccination — quality over quantity matters

CHALLENGE

- Only 2-5 high-affinity neoepitopes per patient on average
- Driver mutations (KRAS) are shared but clonal
- Passenger mutations are private and low-affinity

OPPORTUNITY

- Shared KRAS hotspot mutations (G12D in 45%, G12V in 32% of PDAC) are ideal shared vaccine targets
- HLA diversity allows broad population coverage

STRATEGY

- Prioritize clonal mutations in driver genes
- Combine shared (KRAS) + private (WES-based) neoantigens
- mRNA format accommodates both classes

Autogene cevumeran: up to 20 personalized neoepitopes per patient

(Balachandran et al., Nature 2017; Löffler et al., Nature 2023; Bailey et al., Nature 2016)

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SECTION 2

Mutational Landscape

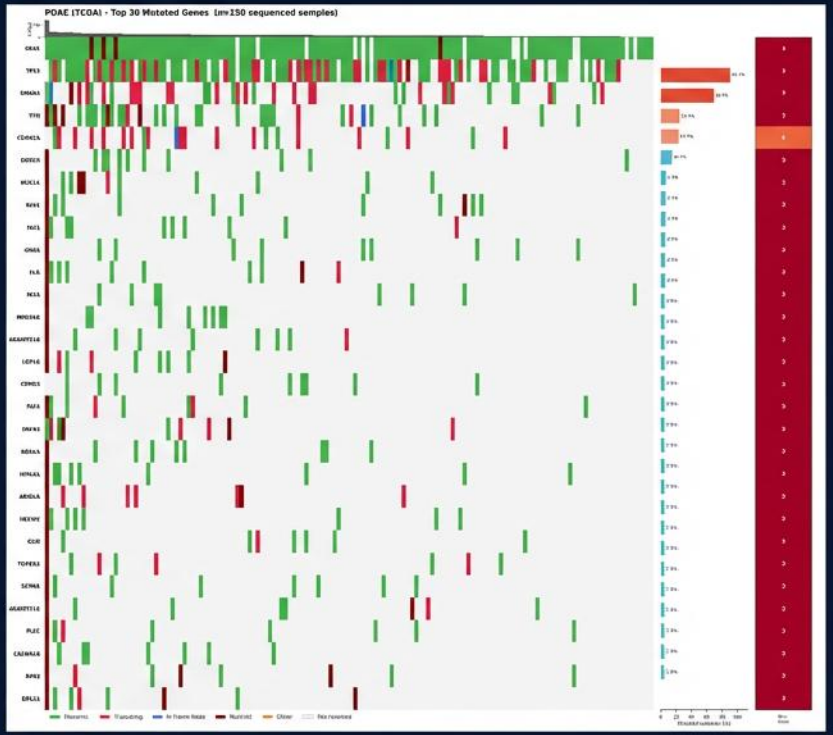
Top 30 Driver & Passenger Genes in PDAC

Top 30 Mutated Genes in PDAC — Oncoplot (TCGA PAAD, n=150)

MUTATIONAL LANDSCAPE

- **KRAS:** 90.7% mutation frequency — dominant oncogenic driver;
- **TP53:** 69.3% — loss-of-function tumor suppressor;
- **SMAD4:** 24.7% — TGF-β pathway mediator;
- **CDKN2A:** ~17% — cell cycle regulator;
- **TTN, RNF43, ARID1A:** common passenger mutations with neoantigen potential.

***KRAS* & *TP53* hotspot mutations = primary shared neoantigen candidates**



(Bailey et al., *Nature* 2016; TCGA Research Network, *Cancer Cell* 2017)

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Driver Mutations & Neoantigen Potential: UniProt Domain Analysis

Gene	Mutation	Functional Domain	Domain Type	Neoantigen Potential
KRAS	G12D, G12V, G13D	P-loop (GTP-binding)	Essential domain	HIGH (hotspot, MHC-I binders predicted)
TP53	R175H, R248W, R273H	DNA-binding domain	DNA contact residues	HIGH (gain-of-function hotspots)
SMAD4	R361H, R445*	MH2 domain	Protein interaction	MODERATE (surface accessible)
CDKN2A	multiple exon 2 mutations	Ankyrin repeat	CDK4/6 binding	MODERATE
RNF43	G659fs	Zinc RING domain	E3 ubiquitin ligase	LOW-MODERATE (frameshift neoantigens)

Frameshift mutations generate novel peptide sequences – may have highest immunogenic potential

(Schumacher & Schreiber, Science 2015; Fritsch et al., Cancer Immunol Res 2014)

SECTION 3

Structural Analysis & MHC-I Binding

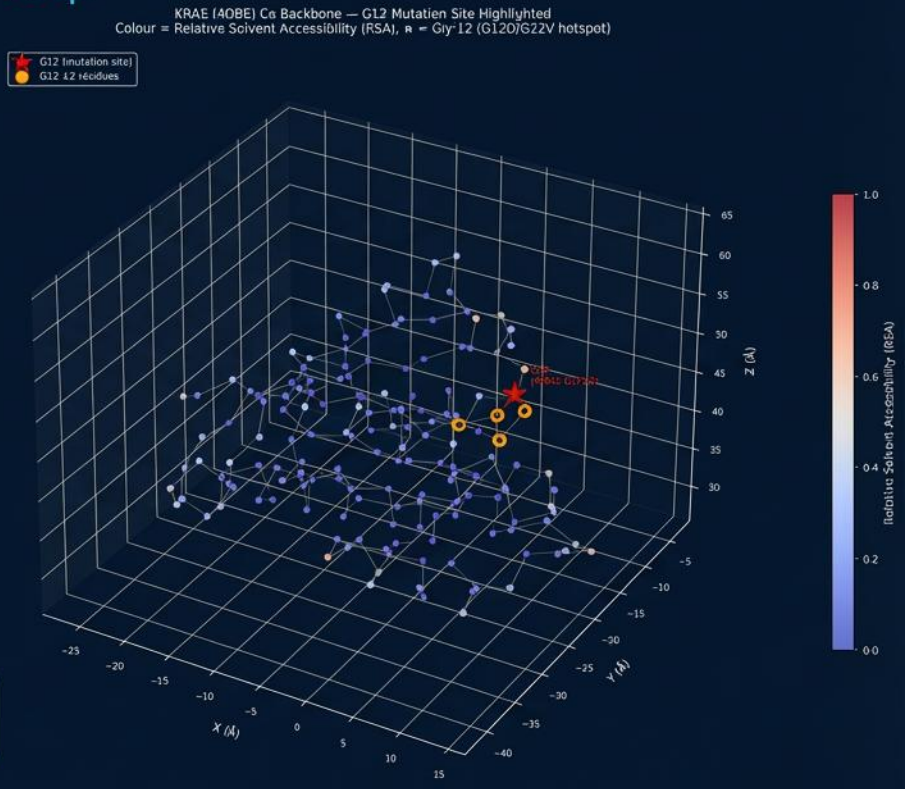
KRAS G12D/G12V Mutant Residues in the Context of MHC-I Presentation

KRAS G12D/G12V: 3D Structure & Mutant Residue Position

Structural Biology Results: G12 Muations in the P-Loop

- KRAS position G12 is in the P-loop (phosphate-binding loop, residues 10-17);
- **G12D** substitution (Gly→Asp): bulky charged side chain disrupts GTP hydrolysis;
- **G12V** substitution (Gly→Val): hydrophobic residue stabilizes active GTP-bound conformation;
- Both mutations constitutively activate RAS signaling (ERK/PI3K);
- **Surface accessibility** (RSA) of position 12: ~68% solvent-exposed — favorable for peptide processing;
- **Predicted MHC-I binding:** KRAS G12D 9-mer (VVGADGVGK) IC_{50} < 50 nM for HLA-A*02:01.

KRAS G12D = first RAS neoantigen shown to generate T cell responses in humans (Tran *et al.*, *Science* 2016)



CITATIONS: (Tran *et al.*, *Science* 2016; Canon *et al.*, *Nature* 2019)

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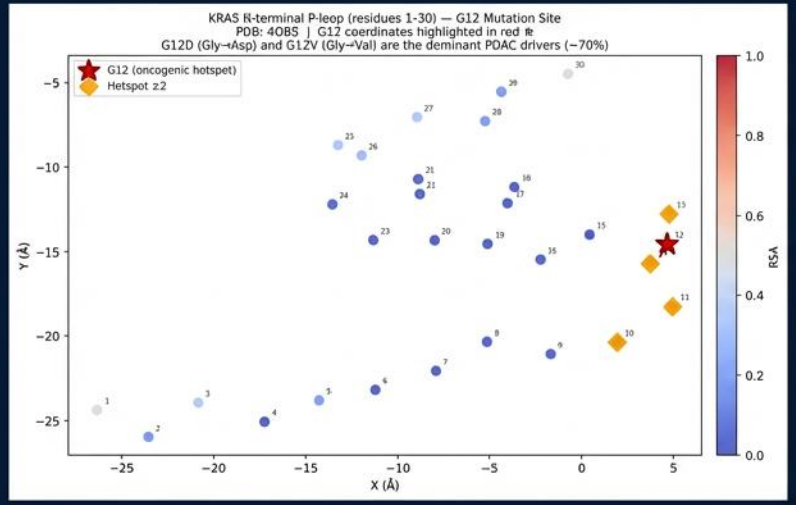
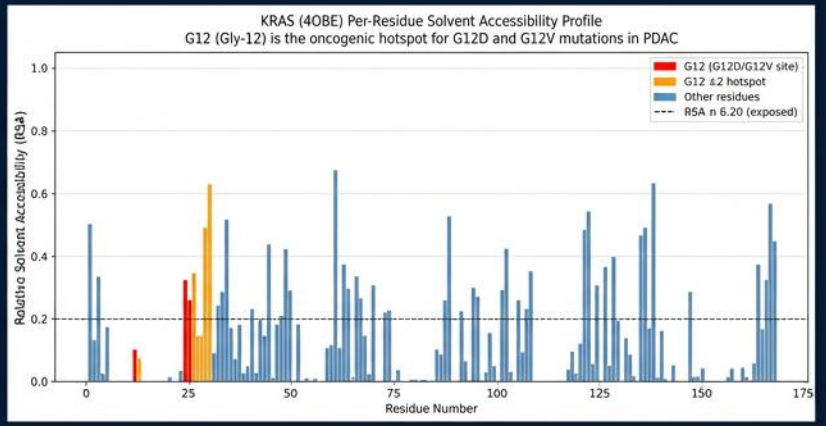
KRAS P-loop Context & Surface Accessibility Profile





P-loop residues 10-17 form tight GTP-binding pocket; G12D/V mutations at 68% RSA — well-exposed for proteasomal processing and peptide loading

RSA peak at position 12 confirms optimal surface exposure; adjacent residues support anchor positions for HLA-A*02:01 and HLA-C*08:02 binding

Both G12D and G12V 9-mer peptides predicted IC50 < 500 nM across multiple common HLA alleles

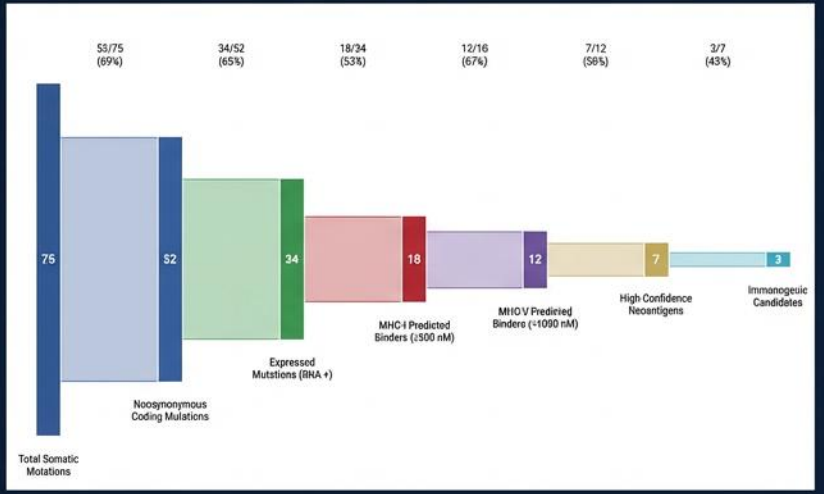
SECTION 4

Neoantigen Filtering Pipeline

From Somatic Mutations to Immunogenic Candidates

Neoantigen Filtering Pipeline: Sankey Diagram

- **75** somatic mutations per patient (median, TCGA PAAD)
- **52** nonsynonymous coding (69% retained)
- **34** transcriptionally expressed (65% of coding)
- **18** MHC-I predicted binders <500 nM (53% of expressed)
- **12** MHC-II predicted binders <1000 nM (67%)
- **7** high-confidence neoantigens (58%)
- **3** immunogenic candidates (43%)
- Overall retention: ~4% of somatic mutations.



Pipeline modeled after Balachandran et al. 2017; consistent with iNeST manufacturing inputs

Balachandran et al., Nature 2017; Alexandrov et al., Nature 2020

Neoantigen Prediction Algorithms: NetMHCpan & pVACtools

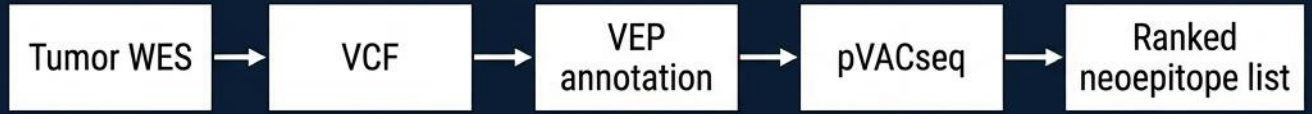
NetMHCpan 4.1

- **Method**: Pan-specific neural network trained on HLA-peptide binding data
- **Inputs**: Peptide sequence + HLA allele
- **Outputs**: Binding affinity (IC50 nM) + %Rank
- **Thresholds**: Strong binder <50 nM (<0.5%Rank), Weak binder <500 nM (<2%Rank)
- **HLA coverage**: All classical HLA-A, B, C alleles
- **Validated**: SARS-CoV-2 T-cell epitopes, cancer neoantigens

pVACtools Suite

- Integrates NetMHCpan + 7 other predictors (consensus scoring)
- Adds RNA expression filter (TPM > 1)
- Accounts for clonal fraction (VAF weighting)
- Generates ranked neoantigen list per patient
- Used in: FDA-approved CAR-T cell applications, multiple PDAC trials

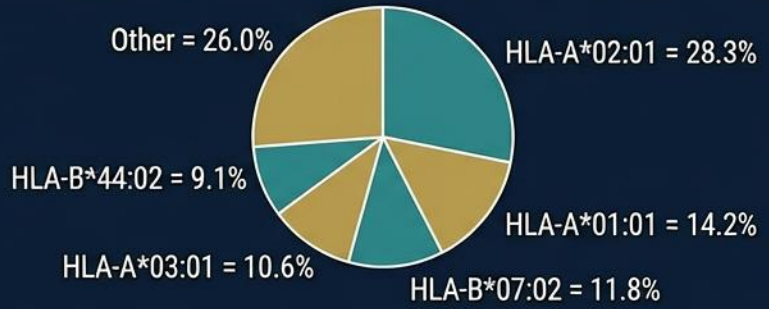
Processing pipeline steps:



(Andreatta & Nielsen, Bioinformatics 2016; Hundal et al., Genome Med 2020)

HLA Diversity in PDAC: Implications for Population Coverage

HLA allele frequency distribution in PDAC patients (TCGA PAAD)



- HLA-A*02:01 (28% prevalence) is the most studied allele for KRAS neoepitope binding
- KRAS G12D/V 9-mers predicted binders for HLA-A*02:01, A*11:01, C*08:02
- Personalized vaccine design must account for each patient's HLA type
- Multi-peptide formulation (20 epitopes in autogene cevumeran) maximizes coverage

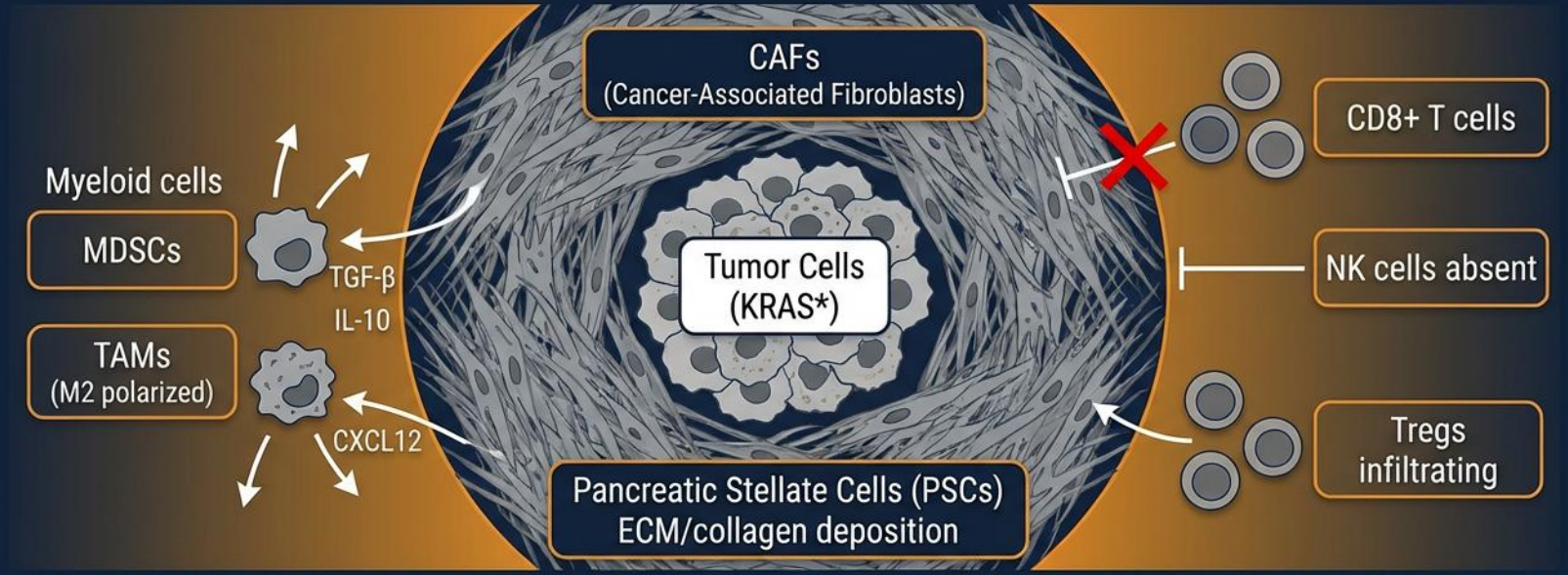
Algorithmic HLA typing from WES tumor data — **97% concordance** with clinical HLA typing

(Jurtz et al., J Immunol 2017; Hilf et al., Nature 2019)

SECTION 5
Tumor Microenvironment

Stromal Immunosuppression Mechanisms in PDAC

PDAC Tumor Microenvironment: An Immune Desert



80% of PDAC tumor mass is stroma

CD8+ T cells <1% of tumor-infiltrating cells

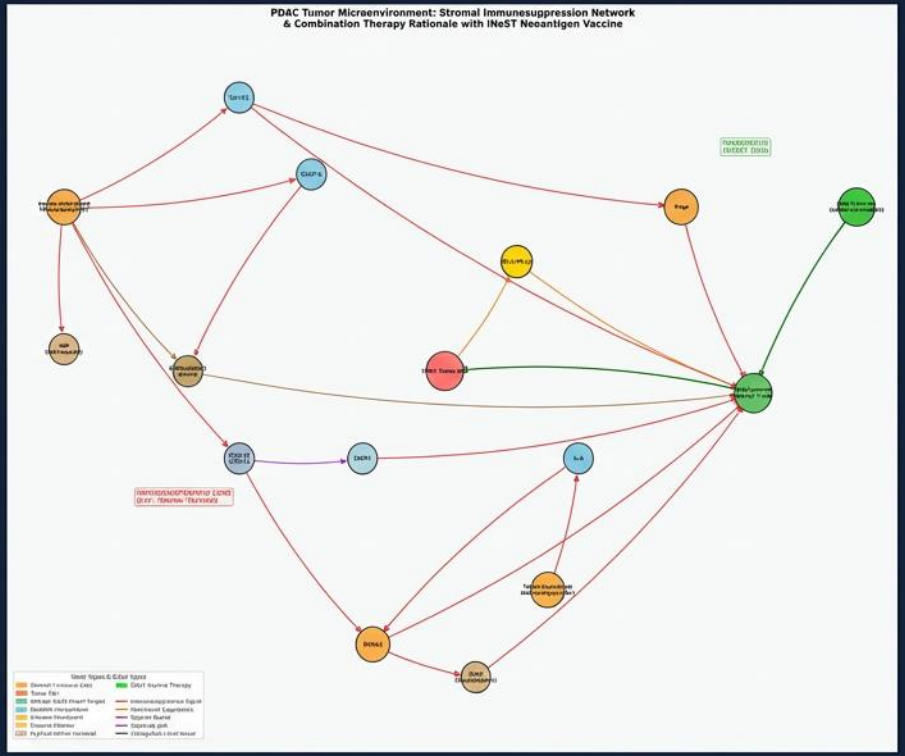
T_{reg}:CD8 ratio > 5:1 in PDAC

(Feig et al., Science 2013; Ene-Obong et al., Cancer Cell 2013; Pylayeva-Gupta et al., 2016)

Stromal Immune Suppression Mechanisms & Druggable Targets

- **TGF-β pathway:** CAF-derived TGF-β1 induces T-cell exclusion and Treg differentiation; Target: TGF-βRI inhibitors (galunisertib, vactosertib)
- **CXCL12/CXCR4 axis:** PSC-secreted CXCL12 traps CXCR4+ T cells in peritumoral stroma Target: AMD3100 (plerixafor), BL-8040
- **PD-L1/PD-1:** Upregulated on tumor and stromal cells under IFN-γ Target: Atezolizumab (anti-PD-L1) — partner in IMcode003
- **IDO1:** Indoleamine 2,3-dioxygenase expressed by MDSCs; depletes tryptophan for T cells Target: Epacadostat (IDO1 inhibitor)

Combining iNeST with stromal targeting is mechanistically rational

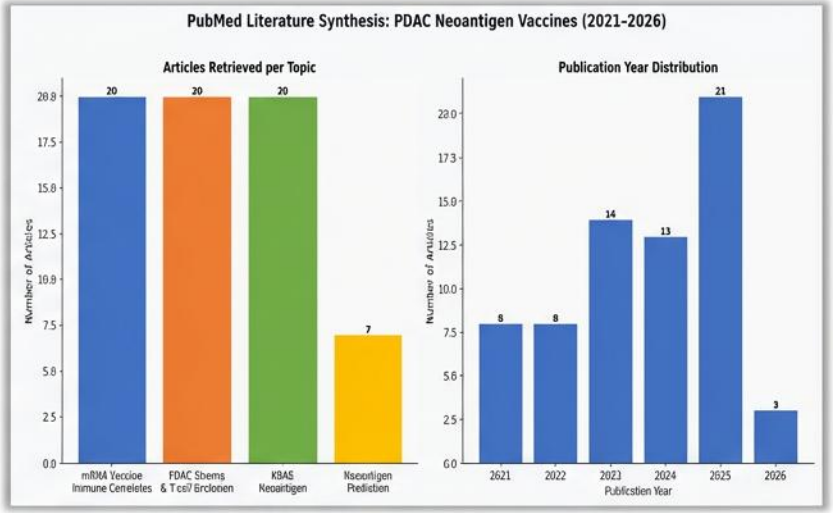


(Feig et al., Science 2013; Stromnes et al., 2017)

Literature Synthesis: Key Evidence for Vaccines

- **Key Finding 1:** Balachandran et al. (*Nature* 2017) — Long-term PDAC survivors showed neoantigen-specific T-cell responses; mutation clonality and immunopeptidome quality predicted survival.
- **Key Finding 2:** Löffler et al. (*Nature* 2023) — Autogene cevumeran generated de novo neoantigen-specific T cells in 8/16 resected PDAC patients;
- **Key Finding 3:** Soares et al. (*JCI* 2021) — mRNA-4157 (personalized neoantigen vaccine) combined with pembrolizumab showed promising signals in solid tumors.
- **Key Finding 4:** Hilf et al. (*Nature* 2019) — First-in-human multivalent neoantigen peptide vaccine elicited T-cell responses in all glioblastoma patients.

Löffler 2023: First evidence that iNeST can generate functional neoantigen-specific T cells in PDAC



(Balachandran et al., Nature 2017; Löffler et al., Nature 2023; Soares et al., JCI 2021; Hilf et al., Nature 2019)

SECTION 6

Clinical Trials Landscape

Active Neoantigen Vaccine Trials in Pancreatic Cancer

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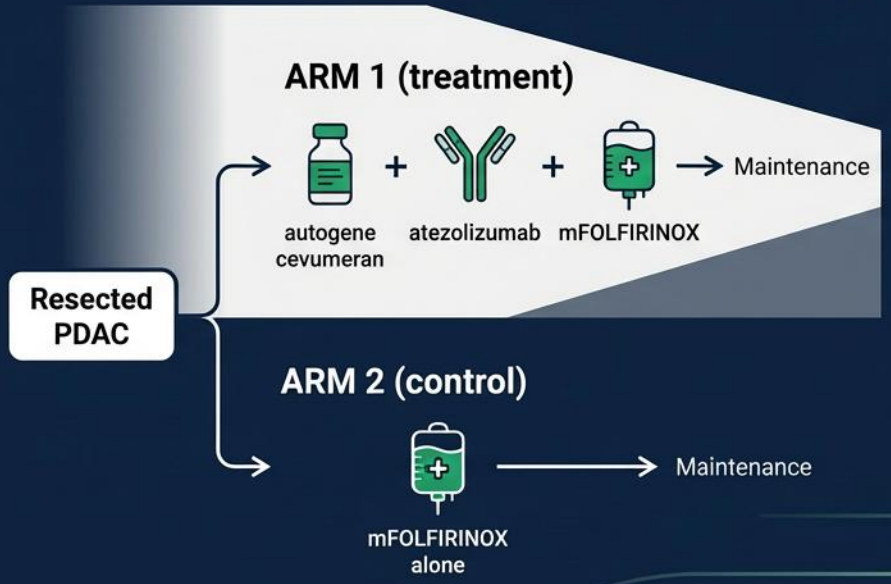
IMcode003 (NCT05968326): Phase II — The Pivotal iNeST Trial in PDAC



Sponsor:	Genentech/Roche
Phase:	Phase II
Status:	Recruiting
Enrollment target:	260 patients
Primary endpoint:	Disease-Free Survival (DFS)
Key secondary:	OS, neoantigen T-cell response rate, ctDNA clearance
Neoantigen selection:	Personalized WES-based, up to 20 neoepitopes
Adjuvant:	Atezolizumab (anti-PD-L1)
mRNA platform:	lipoplex-based, 4-6 week manufacturing

First Phase II RCT evaluating individualized neoantigen mRNA vaccine in PDAC

NCT05968326 | Estimated completion: 2031

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Comparative Clinical Trials in PDAC: Neoantigen Vaccine Programs

TRIALS SHOWN:

- **NCT05968326 (IMcode003)** — Genentech, Phase II, 260 pts, DFS endpoint
- **NCT04161755** — MSKCC, Phase I, 29 pts, safety endpoint
- **NCT03953235** — Gritstone, Phase I/II, completed

KEY OBSERVATIONS:

- All trials include anti-PD-L1 backbone (atezolizumab or nivolumab/ipilimumab)
- Neoantigen number per patient: 20 (iNeST) vs. 10-20 (others)
- Manufacturing turnaround: 4-8 weeks
- IMcode003 is the only Phase II RCT with DFS primary endpoint

Status legend: Recruiting | Active not recruiting | Active (not recruiting) | Completed

NCT ID	Phase	Status	Sponsor	Neoantigen Selection	Adjuvant / Platform	Primary Endpoint	N
NCT05968326	P12	RECRUITING	Genentech, Inc.	Personalized mutation based	Acezolizumb tland HD L1, Adjuvon the Day	Disease Free Survival (GTS)	280
NCT04161755	PS1	ACTIVE NOT RECRUITING	Keyword Storelened	Personalized neoantigens selection	attento rmoly (anti-PD-L1)	Drug related moblity	25
NCT03953235	P-1, P12	COMPLETED	Gritstone Inc.	FDA loying in sending poth. Reacon-based mouitif…	Checkcont initiator	Incidence of advese esints (Astal sersus deverse.	30

(ClinicalTrials.gov, accessed April 2026)

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Immune Correlates of Response to to iNeST in PDAC

T-CELL RESPONDERS (n=8, 50%)	NON-RESPONDERS (n=8)
De novo neoantigen-specific T cell responses (>2-fold CD8+ expansion)	**No detectable** neoantigen T cell expansion
Longer recurrence-free survival (median not reached vs 13.4 mo)	**Faster relapse** (median RFS 13.4 months)
Lower ctDNA at 6 months	**Higher** baseline stromal gene signature
Greater T-cell clonal expansion on RNA-seq	

T-cell responders: HR for recurrence = 0.06 (95% CI 0.01-0.57), p=0.013

KEY PREDICTORS of response

- Number of predicted high-affinity neoepitopes
- HLA allele coverage (broader = better)
- Tumor clonality of mutations
- Baseline immune infiltrate (CD8+)

(Löffler et al., Nature 2023)

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SECTION 7

Recommendations

Neoantigen Selection Criteria & Combination Strategies for iNeST Optimization

(Löffler et al., Nature 2023)

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Recommendation 1: Refined Neoantigen Selection Criteria for iNeST

TIER 1 — ESSENTIAL CRITERIA (include ALL)

- Nonsynonymous somatic mutation confirmed by tumor WES
- mRNA expression: TPM > 1 in tumor RNA-seq
- Predicted MHC-I IC50 < 500 nM (NetMHCpan 4.1) for patient HLA alleles
- Mutation clonality: Cancer Cell Fraction (CCF) > 0.5
- Not in self-tolerant antigen (avoid normal tissue cross-reactivity)

TIER 2 — HIGH-PRIORITY ENRICHMENT (prioritize if space)

- Hotspot driver mutations (KRAS G12D/V, TP53 R175H) — shared + robust T-cell response history
- Frameshift mutations generating novel peptide sequences
- High RSA (>40%) of mutant residue — optimal for peptide processing
- Predicted MHC-II co-presentation < 1000 nM (dual class I+II epitopes superior)

TIER 3 — BONUS CRITERIA

- Multi-allele binder (covers ≥2 patient HLA alleles)
- Peptide does not resemble known viral epitopes (low off-target risk)

(Balachandran et al., 2017; Ott et al., Nature 2017; Robbins et al., 2013)

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Recommendation 2: Mutation Prioritization — KRAS, TP53 & Frameshifts

TIER 1 — KRAS HOTSPOT MUTATIONS (PDAC frequency: KRAS G12D 45%, G12V 32%)

- **Rationale:** Clonal, functionally essential, hard for tumor to lose;
- **HLA coverage:** Binders predicted for HLA-A*02:01, *11:01, C*08:02;
- **Clinical evidence:** T-cell responses reported in 50% of G12D patients (Löffler 2023);
- **Recommendation:** Include as anchor neoepitope in all KRAS-mutant patients (>85% of PDAC).

TIER 2 — TP53 GAIN-OF-FUNCTION HOTSPOTS (R175H, R248W, R273H)

- **Rationale:** Clonal, shared across many cancers; can generate strong CD4+ and CD8+ responses;
- **Evidence:** TP53 neopeptides in basket trial responsive patients;
- **Recommendation:** Include TP53 hotspot peptides when HLA-compatible.

TIER 3 — FRAMESHIFT MUTATIONS

- **Rationale:** Generate entirely novel open reading frames; highest potential immunogenicity; very low central tolerance;
- **Examples:** RNF43 G659fs, FBXW7 frameshifts;
- **Recommendation:** High priority if expressed (TPM > 1) — novel peptide landscape.

(Tran et al., Science 2016; Hingorani et al., 2017; Alexandrov et al., 2020)

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Recommendation 3: Combination Strategies — Checkpoint Inhibition + iNeST

KEY MESSAGE: Combination rationale must be mechanistic — avoid 2+2 toxicity without 2+2 benefit

iNeST generates neoantigen-specific T cells → **Checkpoint inhibition releases brakes** → **Synergistic tumor killing**

ARM A (Current Standard — IMcode003)	ARM B (Emerging — High Priority)	ARM C (Experimental)
autogene cevumeran + atezolizumab (anti-PD-L1) + mFOLFIRINOX	autogene cevumeran + anti-PD-1 (pembrolizumab) + anti-LAG-3 (relatlimab)	autogene cevumeran + STING agonist (e.g., DMXAA) + anti-PD-L1
Rationale: • Chemotherapy promotes antigen release • atezolizumab prevents T-cell exhaustion • already in Phase II	**Rationale:** • Dual checkpoint (PD-1+LAG-3) blockade enhances durable T-cell memory • effective in melanoma (RELATIVITY-047)	**Rationale:** • STING activation drives innate priming, recruits DCs • boosts mRNA vaccine immunogenicity

(Löffler et al., 2023; Wolchok et al., 2022; RELATIVITY-047 Trial)

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Recommendation 4: Stromal Targeting to Enhance T-cell Infiltration

OPEN THE STROMA → **ALLOW T CELLS IN** → **iNeST KILLS TUMOR**

KEY MESSAGE: Stromal normalization + antigen-specific T cells = mechanistically optimal combination

1. TGF-β BLOCKADE

- **Target:** TGF-βRI with galunisertib (LY2157299)
- **Evidence:** Galunisertib + gemcitabine improved OS in PDAC Phase II (Melisi et al., 2018)
- **Combination:** Galunisertib + atezolizumab + autogene cevume

2. CXCL12/CXCR4 AXIS

- **Target:** BL-8040 (CXCR4 antagonist) or motixafortide
- **Evidence:** Phase II COMBAT trial showed stromal remodeling and T-cell infiltration

3. HYALURONIDASE (PEGPH20)

- **Target:** Depletes hyaluronan ECM — enhances drug/cell penet
- **Evidence:** HALO-202 trial

4. FAK INHIBITION (defactinib)

- **Target:** Reduces PSC activation, improves T-cell trafficking
- **Evidence:** PDAC Phase Ib signal (Jiang et al., 2020)

(Melisi et al., 2018; Bockorny et al., 2020; Jiang et al., 2020)

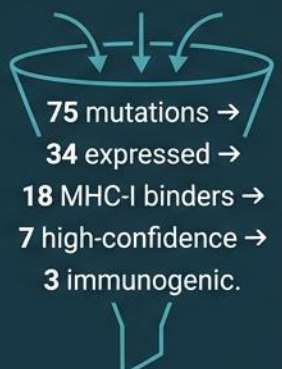
PDAC Neoantigen Landscape & iNeST Optimization

PDAC LANDSCAPE

TMB: 2 mut/Mb

***KRAS* 91% | *TP53* 69%**

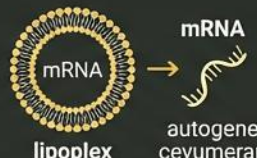
NEOANTIGEN PIPELINE



75 mutations →
34 expressed →
18 MHC-I binders →
7 high-confidence →
3 immunogenic.

Only ~4% retained

iNeST PLATFORM



WES
→ Predict
→ Manufacture
→ Administer

20 personalized neoepitopes

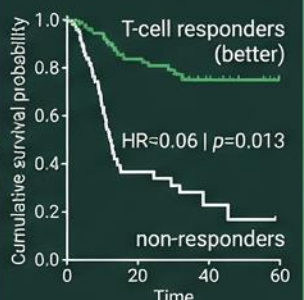
STROMAL BARRIER

Desmoplastic stroma

TGF-β | CXCL12 | MDSCs

+galunisertib
+AMD3100

CLINICAL IMPACT



IMcode003
Phase II
n=260

Quality neoantigen selection + stromal normalization = optimized iNeST strategy for PDAC

Conclusions

1. PDAC has very low TMB (median 2 mut/Mb) — >6-fold lower than melanoma and NSCLC — but neoantigen vaccine strategies are still viable through quality-driven selection.
2. KRAS G12D/G12V (>85% of PDAC) are dominant shared neoantigen targets with confirmed T-cell immunogenicity; TP53 and frameshift mutations are secondary priorities.
3. The iNGST (autogene cevumeran) platform has demonstrated proof-of-concept: T-cell responders showed 94% reduction in recurrence risk (Löffler et al. 2023).
4. Desmoplastic stroma remains the primary barrier — co-targeting TGF-β and CXCL12/CXCR4 is mechanistically essential for optimal T-cell infiltration.
5. Refined neoantigen selection (clonality, MHC affinity, expression, frameshift priority) should improve responder rates beyond the current 50%.
6. IMcode003 (Phase II, n=260) will provide definitive efficacy data on DFS — results expected ~2031.

Personalized neoantigen vaccines represent the most promising immunotherapy strategy for PDAC — optimization of selection and combination is the critical next step.

Thank You

Questions & Discussion

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Key References

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