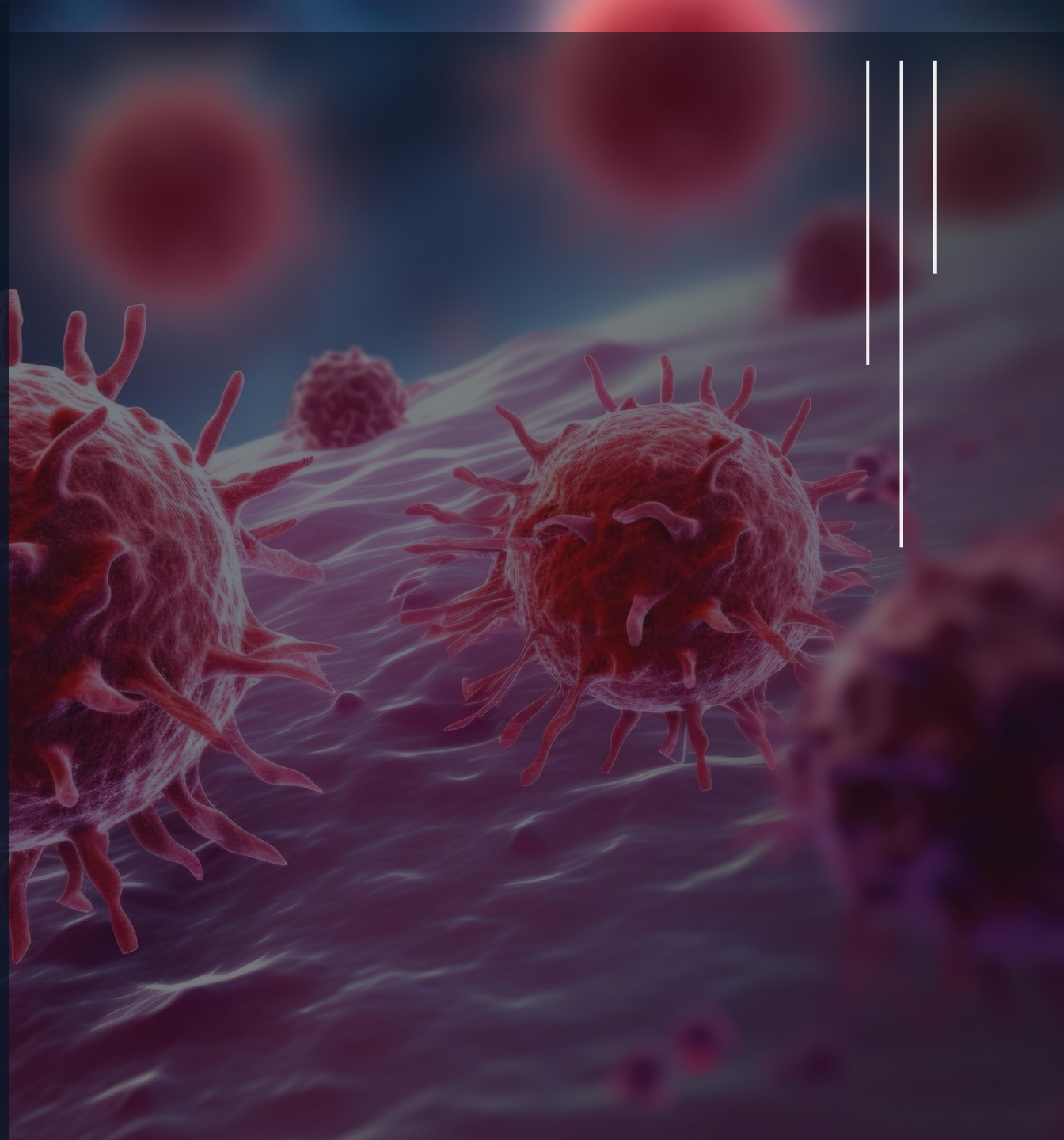
curIOS

Therapeutics

Shared-antigen mRNA immunotherapies for cancer

Unlocking viral and endogenous retroviral targets with programmable T-cell immunity.

Better targets. Better T-cell persistence.



Non-confidential deck | May 2026

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CurIOS builds *in vivo* mRNA cancer immunotherapies against shared driver antigens

We start where cancer immunobiology is the strongest: shared and driver antigens in virus-associated tumors

What CurIOS does

Next-generation mRNA therapies that:

- Target shared and essential viral and endogenous retroviral antigens
- Combine antigen discovery in patient samples with immune engineering
- IP protection filed through Yale on mRNA constructs and immune-enhancement.

Lead wedge: viral Merkel cell carcinoma
Expansion path: Renal Cancers / ERV and common solid tumors (breast, lung, CRC, prostate)

Why CurIOS can win:

Shared driver antigens + durable T-cell memory enable more effective mRNA cancer immunotherapies

Right target

Shared driver antigens are conserved, biologically relevant and difficult for tumors to escape.

FUSION^2 improves antigen selection and vaccine design

Right immune response

Co-encoded immune signals are designed to deepen and extend CD8$^+$ T-cell expansion.

COMET platform programs durable immunity

Fast path to value

MCC offers a focused path to human proof-of-concept; ERVs in RCC and solid tumors expands the opportunity to larger markets.

NIH NATIONAL CANCER INSTITUTE | **NCI NeXT selected**
Non-dilutive support to IND

Efficient, NCI-supported MCC proof-of-concept with a large and scalable upside in ERV driven solid tumors

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Built by clinician-scientists and operators with mRNA, disease and immuno-oncology depth.

Domain-expertise in RNA therapies and immuno-oncology; Disease and clinical development expertise in MCC, RCC and solid tumors; Company build expertise

Jeffrey Ishizuka, MD, DPhil

Medical oncology; Yale immuno-oncology; mRNA therapies and MCC

David Braun, MD, PhD

Medical oncology; cancer vaccines, RCC / ERV biology, and clinical trials.

Maggie Cook, MD

Experienced biotech CEO and investor; launched Renovacor and led into the public markets.

Gene Griffin, DVM, MS

mRNA and rare-disease development executive; prior CureVac and Alexion experience.

Kelly Olino, MD, FACS

Surgical oncology; clinical-translational expertise across MCC and skin cancers.

Alex Frey, MD

Surgical oncology; co-inventor on CurIOS technologies.

Core advantage: team combines disease insight, translational access, development experience and executive leadership track record

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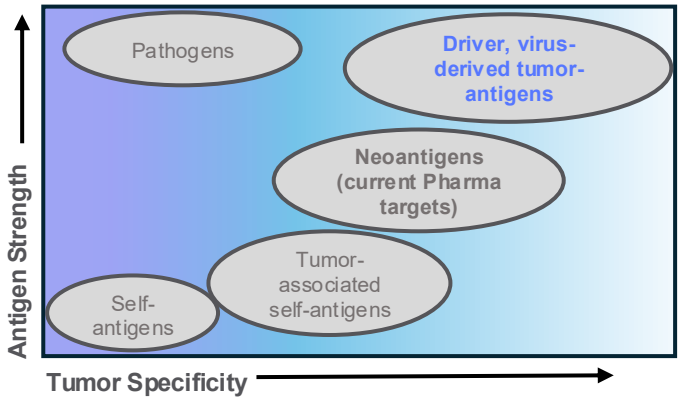
Most antigen-targeted cancer immunotherapies underperform for two structural reasons.

CurIOS solves for target antigen quality and T-cell durability

1. Weak or escapable targets

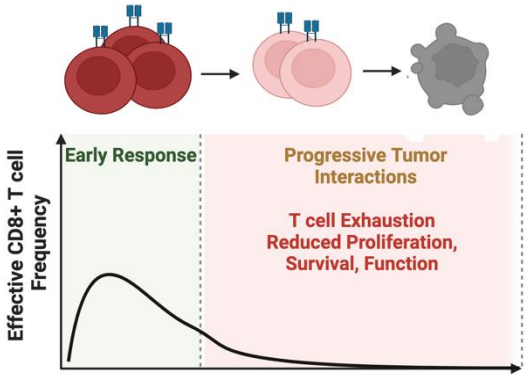
Most antigen-targeting immunotherapies center on neoantigens, which are often weakly antigenic, patient-specific, and non-essential for tumor survival.



Driver, virus-associated antigens sit in a stronger region of the target landscape: more tumor-specific than self-antigens, more immunogenic than neoantigens.

2. Short-lived T-cell responses

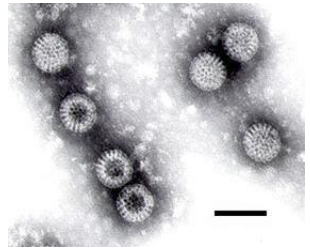
Even when vaccines generate initial activity, sustained expansion of tumor-reactive $CD8^+$ T cells is hard to achieve. Durable response biology is a key bottleneck.



Current products drive effector responses without necessarily inducing memory. CurIOS engineers response durability by design.

Where CurIOS starts

Virus-associated cancers offer a cleaner biology: strong antigens, tumor-dependence and high-unmet need



Exogenous virus-associated cancers represent 10% of the global oncology market; Targeting Endogenous Retroviruses (ERVs) opens even larger markets

CurIOS overcomes key bottlenecks limiting existing therapeutic cancer vaccine approaches

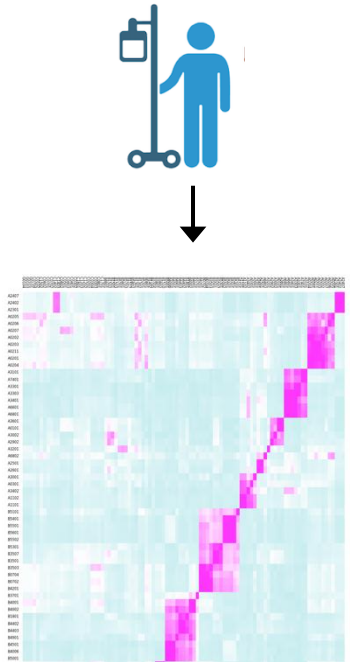
CurIOS platforms solve for target and immune response

Platforms give us an advantage in: i) modeling antigen strength and vaccine-immunogenicity; and ii) memory response engineering

FUSION²

Selecting the right shared antigens directly from patient tumors

- Novel antigens from endogenous retroviruses (ERVs): viral antigens with *shared expression* in *common cancers.*
- Discovery directly in patient samples
- Ranks and prioritizes shared driver antigens from viral and ERV biology across HLA types.
- Integrated computational / experimental platform designs and assembles more immune-activating constructs

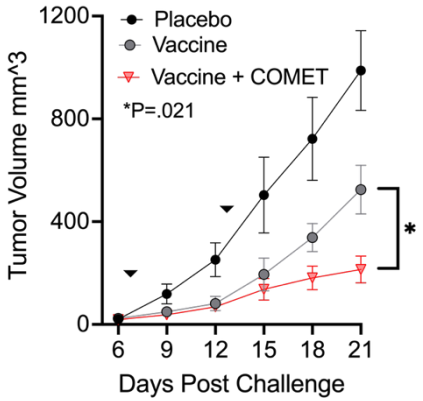


Target selection + optimized construct design derived from patient data

COMET

Engineering durable T-cell responses

- **CO**ordinated **M**ultisignal **E**xpansion of **T** cells
- Co-encodes immune-modulating signals with the antigen cassette.
- Boosts and fine-tunes T cell memory– the key to long-lived control of cancers.
- Permits immune engineering beyond ‘antigen+adjuvant’ frameworks.



Co-encoded immune programs improve and sustain T-cell responses

Better target selection and T-cell response durability

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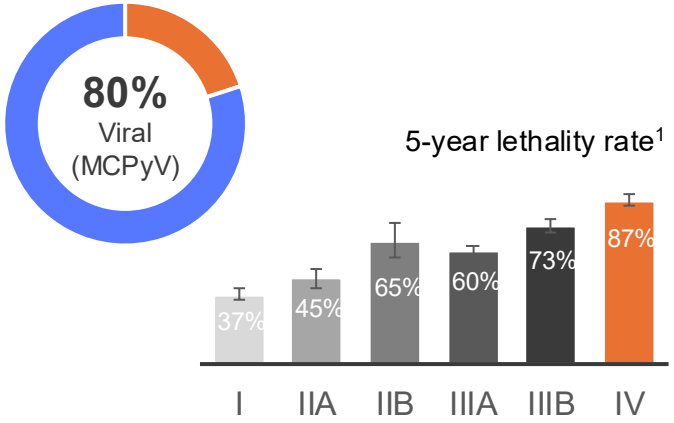
Merkel cell carcinoma is a fast path, non-dilutive path to clinical POC

MCC combines a strong rationale, high unmet need and an externally validated capital-efficient development path.

MCC has high unmet need

- The most lethal skin cancer (stage-for-stage)
- Rising incidence (3,000 US cases predicted to rise to 5,000 by 2030)
- Offers a quicker, rare disease development path



Enables CurIOS to rapidly test whether its platforms and technologies improve patient immune responses.

Current therapies fall short

~50%
aPD-1 response in MCC

- Checkpoint blockade leaves substantial room to improve on response depth and durability
- There is no established second-line standard of care
- Chemotherapy is used, but has a low response rate and duration

The case for MCC development

Shared viral target antigens are required for tumor cell survival.

Boosting patient immunity is the key to better control.

There is a feasible neoadjuvant path to pathologic response in Ph1.

Focused, rare disease development path with non-dilutive support offers a low-cost path to commercialization

NIH NATIONAL CANCER INSTITUTE | **NCI NeXT selected**
Non-dilutive support to IND

Why we can execute: patient access, translational assays, neoadjuvant clinical development path powered by NCI support

MCC is an optimal first indication with high probability of success and the potential to derisk future programs

[1] Jing et al., Cancer Immunol Res, 2020

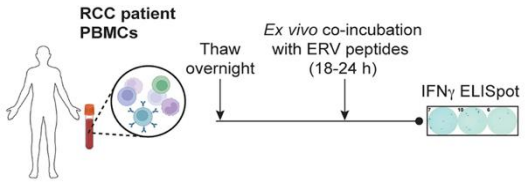
EXPANSION OPPORTUNITY

ERV targeting has a large upside potential in solid tumors

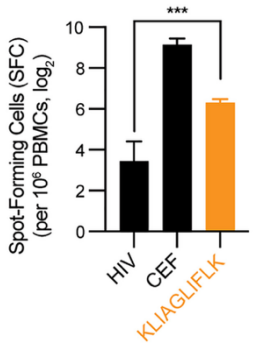
Endogenous retroviruses are prevalent and immunogenic but underexplored as cancer vaccine targets.

ERVs are a compelling target class in RCC

- RCC: a large indication where tumor-associated endogenous retroviruses can create a tractable shared-antigen opportunity.
- Immune responses against ERVs are associated with RCC remission[1]
- Immunogenic ERVs are driven by aberrant HIF-signaling in RCC and have a credible case to be primary targets of T- cell immunity[2].



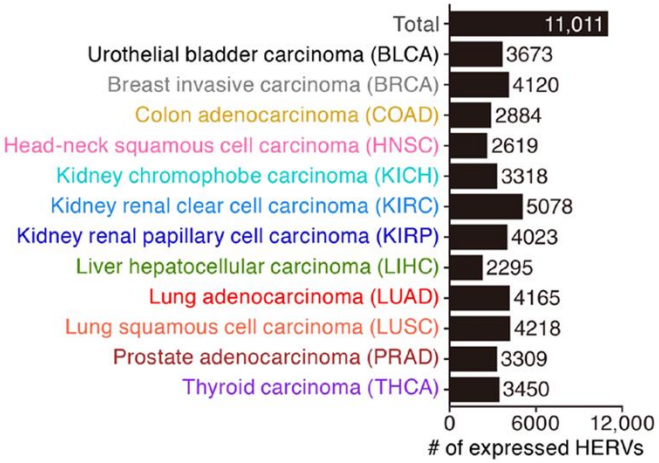
ERV immune responses are observed in patients



RCC / ERV extends the CurIOS pipeline into a larger market

ERV expression across common cancers

Follow-on opportunity set is significant



Most solid tumors express a significant quantity of shared ERV targets[3]

ERVs provide shared, immunogenic vaccine targets across human cancers

CurIOS identified immunogenic, RCC-associated ERVs and developed unique HLA-humanized mouse models to enable validation.

Why CurIOS can win in ERV-targeting:

- RCC and ERV expertise
- Better antigen selection and vaccine design
- Clinical / translational access, pipelines and experience

Expansion from MCC to RCC enables the product thesis to scale to a larger business.

[1]Takahashi et al. HCI, 2008; [2] Jiang, Braun, et al., Cell, 2025; [3] Ito et al., Sci Advances, 2020.

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CurIOS offers a capital-efficient path to human POC in mRNA shared-antigen immunotherapies

$10–15M Series A Financing

Value inflection
Capital deployed across workstreams with incremental value inflections over 24 months

Capital-efficient path
Series A funds staged milestones that compound across clinical programs

Platform leverage
Each milestone expands IP, improves construct design and strengthens the next IND path

Platform Build

- FUSION^2 : Target discovery and construct design across common cancers
  Optimal RCC target product; translational preclinical efficacy; ERV solid tumor landscape; IP moat
- COMET: Immune Programming for Durable T-cell immunity
  AI-guided directed evolution of bespoke immune combinations with IP protection

Clinical Development

- MCC: preclinical development NCI funded to IND acceptance ~ 18-24 months
  RCC: clinical lead in IND-enabling studies; IP moat
  Additional solid tumor candidates designed with preclinical validation and IP protection

Inflection points / potential exit

Milestone path	9-12 months	24 months	36 months	36-48 months	48-60 months
Start	MCC Preclinical, COMET combo IP 3rd Indication Lead	MCC IND, RCC preclinical, FUSION^2 Data Moat	RCC / ERV IND	MCC Clinical POC RCC / ERV Ph1 start 3rd Indication IND	MCC Ph2 complete RCC / ERV Clinical POC 3rd Indication Ph1 Start

CurIOS is defining the next generation of cancer immunotherapies by pairing the right antigens with durable T-cell immunity

Extra Slides

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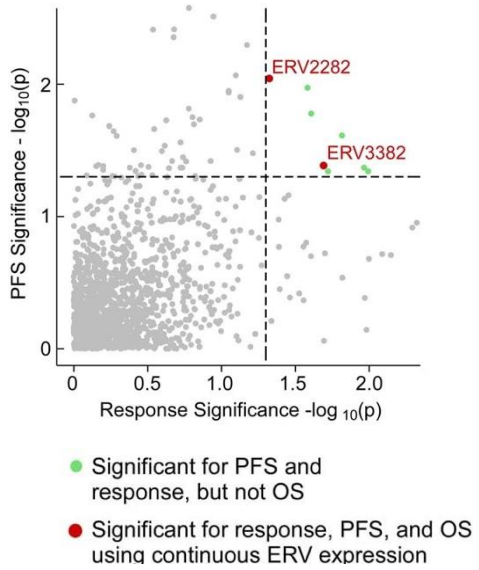
FUSION² helps CurIOS pick fewer, better shared ERV antigens

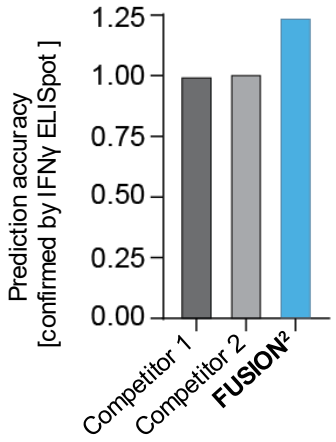
Integrated antigen detection, epitope prioritization and construct design improve vaccine quality.

1. Detect Shared Driver Antigens

- Antigen detection approach combines transcriptional, protein and in silico modeling with functional data from patients



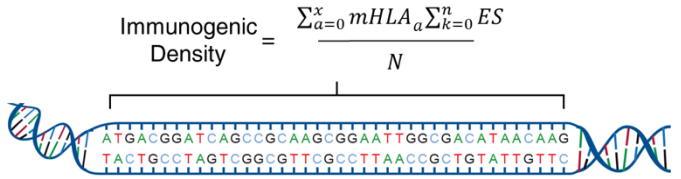
2. Prioritize the strongest Epitopes



Clinical and patient-sample data improve epitope selection

FUSION² improves epitope prediction vs. state-of-the-art approaches

3. Design optimized multi-epitope constructs



Construct design integrates epitope density and population-level MHC diversity.

Target selection and construct design are one workflow.

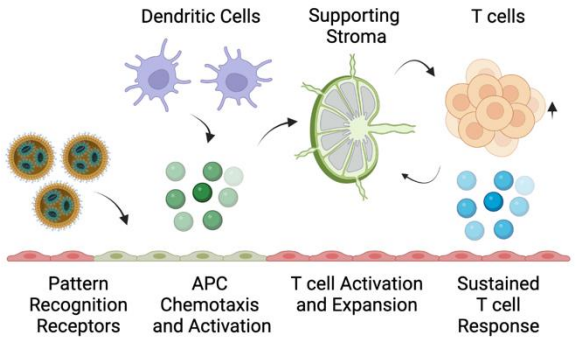
Integrated computational-experimental workflows for improved models and vaccines.

SUPPORTING DATA

COMET platform enables T-cell memory expansion and phenotype engineering

CurIOS is programming a stronger T-cell memory biology

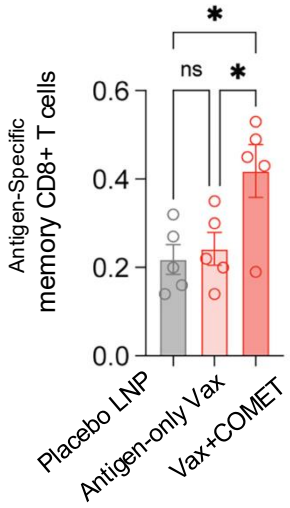
Mechanism view



Co-encoded immune signals shape antigen presentation, T-cell activation, and sustained T-cell response in one construct.
PCT IP protection filed

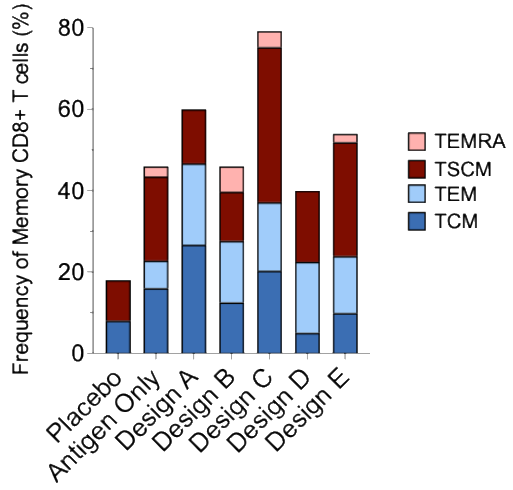
Goal: stronger, more durable CD8+ T-cell response

T-cell memory expansion



Antigen-specific CD8+ T-cell expansion

Memory Phenotype control



T-cell memory states are programmable

COMET enables fine-tuned control of the magnitude and phenotype of immune expansion following vaccination

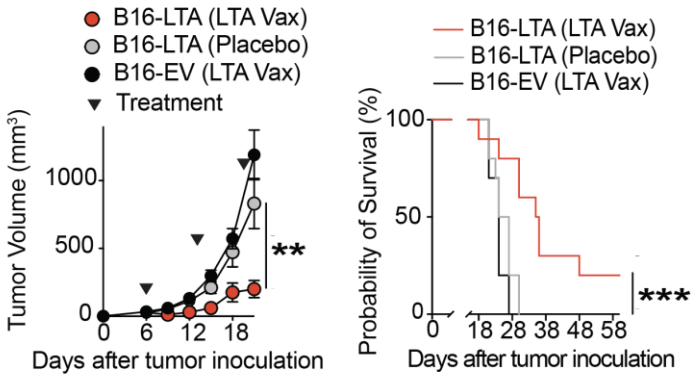
SUPPORTING DATA

CurIOS has preclinical validation of MCC program *in vivo* and in patient samples

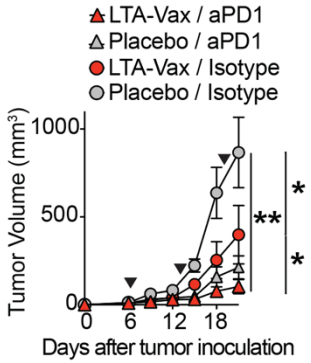
Mouse efficacy supports mechanism and immunogenicity while patient validation derisks translation to the clinic

Mouse efficacy

Monotherapy Efficacy

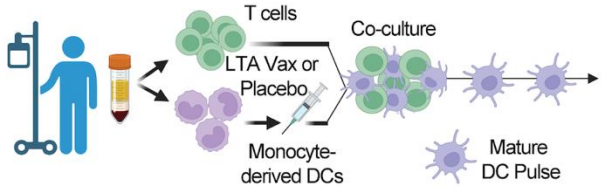


Combination with anti-PD-1

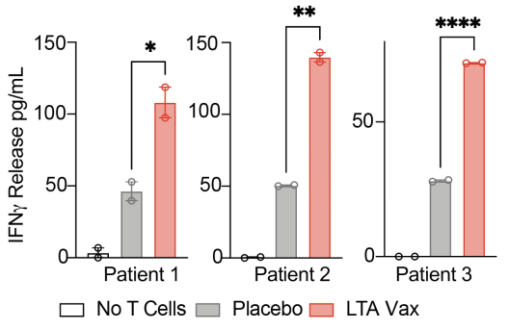


Takeaway: the vaccine shows therapeutic activity in mouse models.

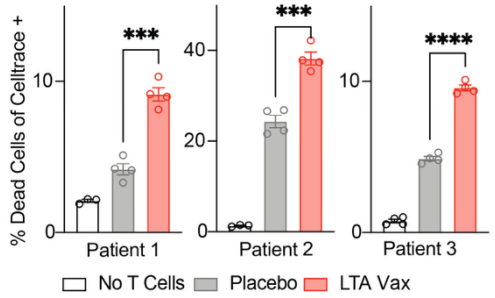
Human MCC validation



IFNγ Release



Tumor cell killing



Takeaway: human sample assays show stronger IFNγ release and tumor-cell killing.

Combined evidence supports vaccine potency and potential to impact MCC patient tumor immunity

EXIT POTENTIAL

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Exit potential for programmable immuno-oncology platforms

Pharma appetite for immune programming

Six major acquisitions in 24 months signal demand for in-vivo immune activation.

RNA + cell engineering convergence

Buyers are pairing programmable RNA control with in-vivo cell programming as one strategic theme.

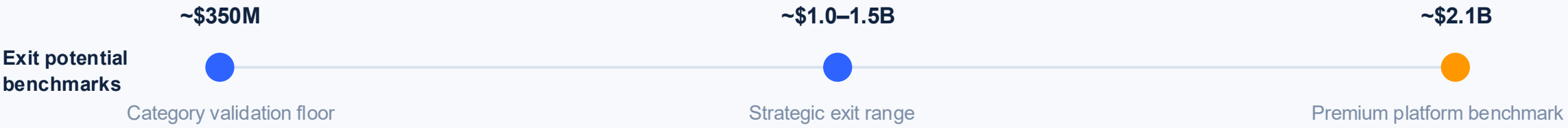
Premium exit benchmarks

Validated valuation band spans ~$350M floor to ~$2.1B for category-defining platforms.

Strategic transaction landscape (2024–2026)

AbbVie	**~$2.1B**	**Capstan Therapeutics**	*In-vivo CAR-T / immune programming; Lead in Phase I*
Bristol Myers Squibb	**~$1.5B**	**Orbital Therapeutics**	*Circular + linear RNA therapeutics; Preclinical Stage*
Roche	**~$1.5B**	**Poseida Therapeutics**	*Cell & immune engineering; Lead in Phase I*
AstraZeneca	**~$1.0B**	**EsoBiotec**	*In-vivo cell programming; Lead in Phase I*
Eli Lilly	**Undisclosed**	**oRNA Therapeutics**	*Circular RNA modalities; Preclinical Stage/ IND-ready*
Kite / Gilead	**~$350M**	**Interius BioTherapeutics**	*In-vivo CAR-T programming; Lead in Phase I*

Programmable immuno-oncology platforms sit at the intersection of two validated acquisition themes: in-vivo cell programming and next-generation RNA control.



$3M vs $5M seed: leverage non-dilutive sources and scale platforms / pipeline

NCI-supported clinical wedge and academic resources enable CurIOS development flexibility

$3M Seed — Lean company formation + platform

MCC
NCI IND path supported; composition-of-matter IP protected.

RCC / ERV
Leverage academic funding to nominate RCC/ERV lead construct.

THIRD INDICATION
Select high-value ERV indication; computational + early validation data of lead antigen set.

FUSION^2 + COMET
FUSION^2 Data Moat in 2 cancers; benchmark natural + 2-5 bespoke COMET signals; IP on 1-2 synthetic designs

TEAM
Part-time CEO, Computational antigen lead + 1 wet-lab scientist/associate; Yale SRAs for access-heavy work.

$5M Seed — Accelerated development

MCC
Clinical-ready plan with NCI + Ph1 trial funds secured.

RCC / ERV
Accelerated path to validated RCC lead candidate with human samples and relevant preclinical model data.

THIRD INDICATION
3rd Indication construct designed, preclinical validation in mouse models + ex vivo patient samples

FUSION^2 + COMET
FUSION^2 solid tumor landscape completed; 3+ hyperfunctional designed COMET variants in ex vivo patient samples functionally validated + IP

TEAM
CEO/BD, Computational lead + 2 wet-lab FTEs
Regulatory and CMC support; Yale SRAs