Corporate Overview

October 2023

NASDAQ: NEXI

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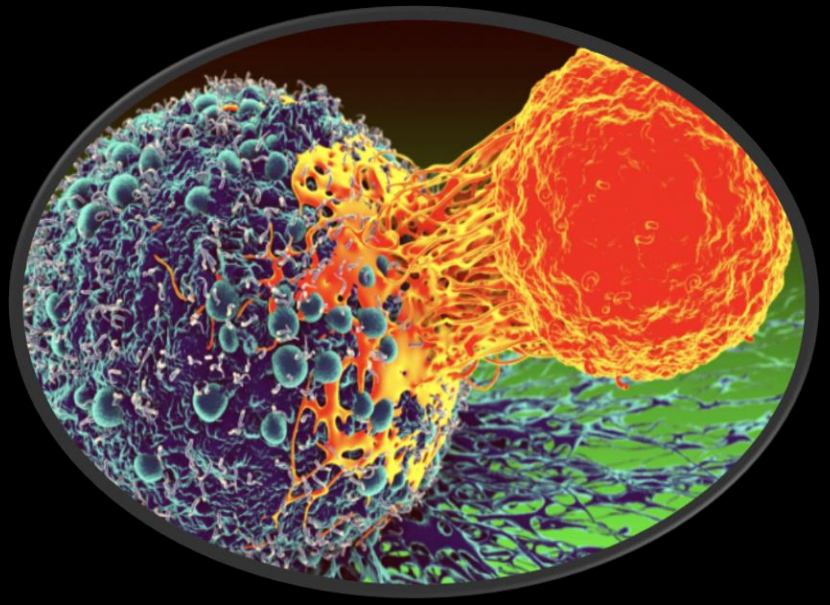
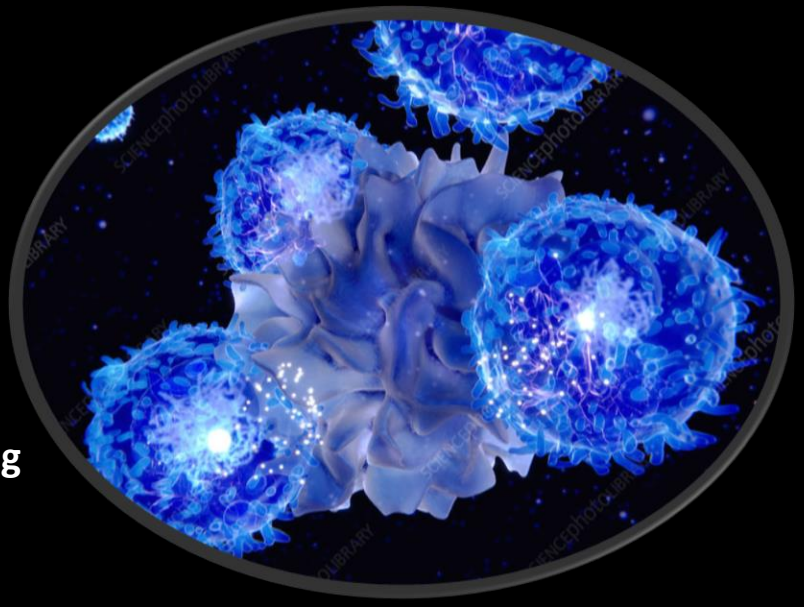


Target-specific killing

"Intensifying" immune response
(Cancer, Infectious Disease)



Target-specific T Cell Tolerance

"Suppressing" or deleting immune response
(Autoimmune Disorders)

AIM™ Platform:

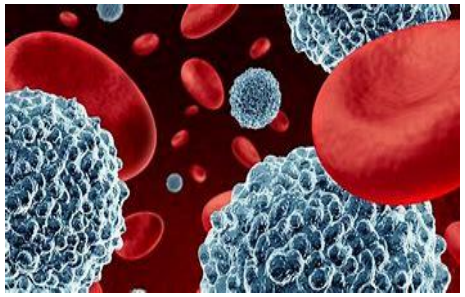
Develop Therapies that Drive Antigen-Specific Cell-Mediated Immune Responses with Curative Potential

Rationally designed synthetic dendritic cell (nanoparticle) based therapeutics

Our Company

Directing antigen-specific responses is one of the top priorities in immunology, though the next wave of breakthroughs has been elusive

Drive multi-antigen T cell mediated durable response

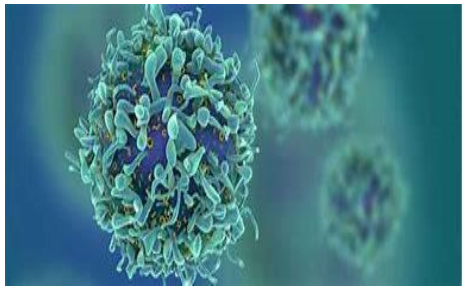


Blood cancers

- Greatest progress and approvals in blood cancers
- Novel approaches (e.g.CAR-T, bispecific TCE*) are limited to a few well-characterized surface targets (e.g. CD19, BCMA)
- **Tumor heterogeneity, T cell persistence** and ACT scale up challenging

Solid tumors

- CPI's well established, combinations offer incremental benefit
- **Increasing activated, tumor-specific T cells in the tumor**, bypassing dysfunctional immune cells in TME is needed – with a scalable solution
- **Multi-antigen targeted therapies and combinations essential to address heterogeneity, drive persistence** and increase durable response



Autoreactive T cells

- Most treatments are non-specific, systemic (e.g., aTNF, aCD20) and globally suppress the immune system
- Emerging opportunity to **target autoreactive, disease-causing T cells directly** and leave healthy tissue alone

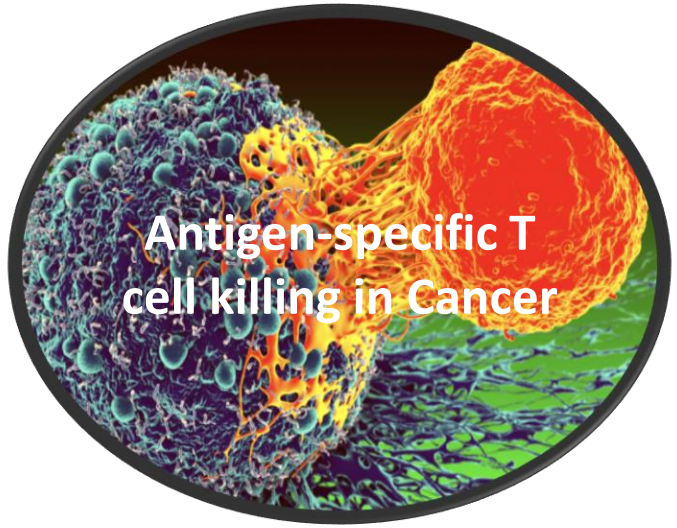
* Bispecific CD3 / Targeted T cell engagers

Summary: AIM™ (Artificial Immune Modulating) Platform and Products

Unique Pharmaceutical Approach to directing ***potent multi-antigen-specific T cell responses***

Transforming Treatment Paradigms

- T cells are the most effective way to specifically target and kill cancer cells
- Therapeutics to *directly* drive durable T cell mediated responses
- Expanding accessible antigen combinations to address heterogeneity



- **Validated MOA:**
  - Early adoptive cell therapy clinical POC
  - Pre-clinical POC in oncology and AI
- **Potential "IND Engine": designed to generate new multi-antigen product INDs with increased speed**
- **Seeking to advance Injectable off-the-shelf IND in 2H2024 (Oncology)**

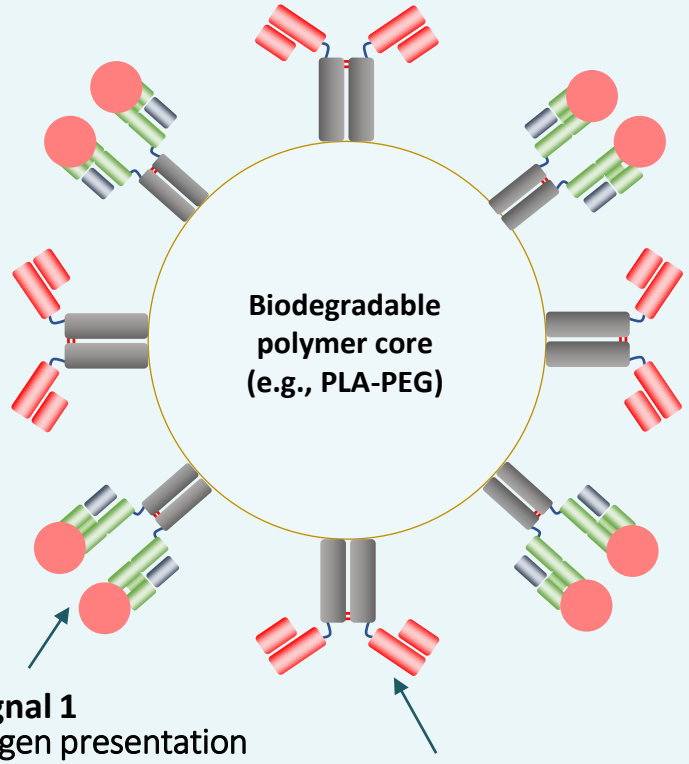
NexImmune's AIM™ platform products have breakthrough potential

Directing ***antigen-specific T cell responses*** across Oncology, Autoimmune and Infectious Diseases

- **AIM "aAPC" nanoparticles designed** to act as synthetic dendritic cells **to deliver precise instructions directly to T cells through natural signaling mechanisms**
- **Multi-antigen specific products designed to generate a T cell mediated response** without impacting healthy tissue or immune function (bypassing host DC)
  - **Break tolerance and drive a durable response in Cancer -** Activate and expand multiple antigen-specific T cell subtypes associated with anti-tumor activity, persistence and establishment of immunologic memory (Tscm, Tcm, Tem)
  - **Establish tolerance in Autoimmune Disorders -** Suppress or delete targeted antigen-specific autoreactive T cells
  - **Direct T cells to kill virally** infected cells **in Infectious Diseases**
- **"Off-the-shelf" injectable products that are scalable**

The AIM™ aAPC nanoparticle

Directs Ag specific T cell specificity and Function through S1/S2 signaling mechanisms



One MOA - Two Therapeutic Modalities

Direct multi-antigen specific T cells to deliver potent, durable response

Establish ACT P1 POC, Advance INJ modality, a scalable, off-the-shelf solution

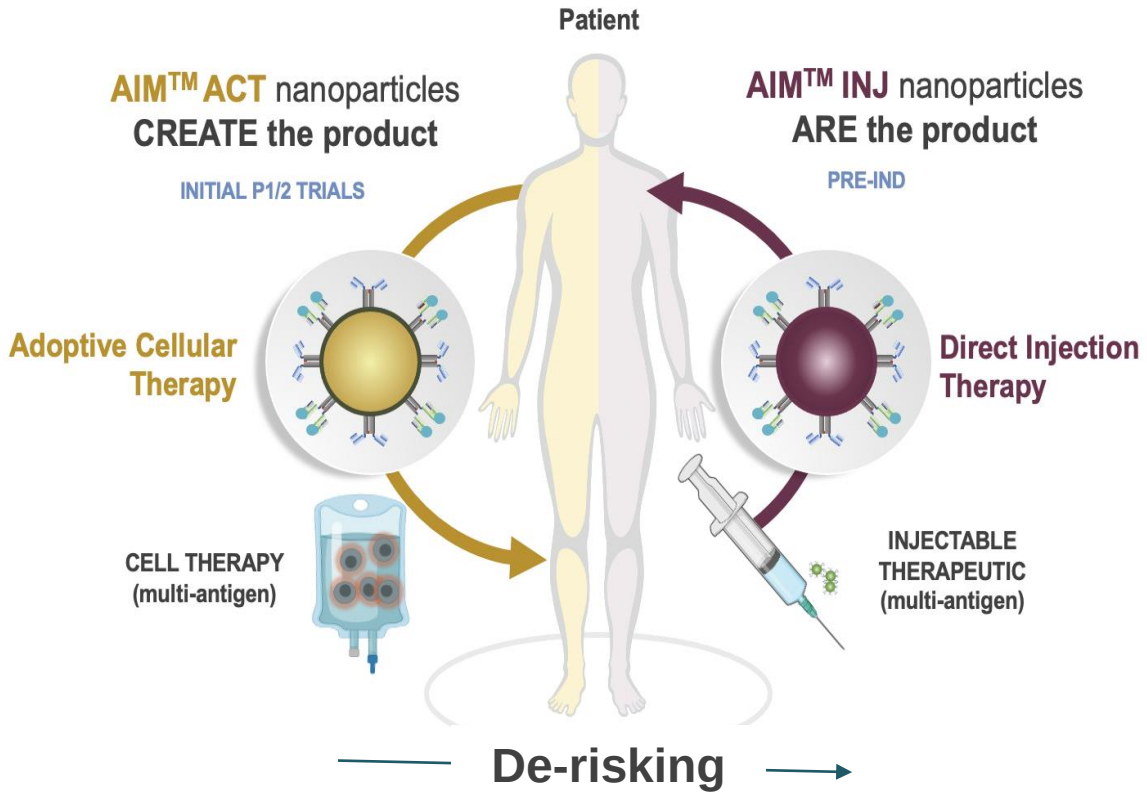
Validated MOA

1. **ACT Early Clinical POC, INJ pre-clinical POC**

2. **Potent, multi-tumor-specific activated T cells that traffic to tumor and persist -** Directly engages the broad TCR repertoire

3. **Address Disease Heterogeneity**
   Broad access to target combinations

Two Modalities
ACT and INJ



Established np and ACT platform clinical manufacturing process

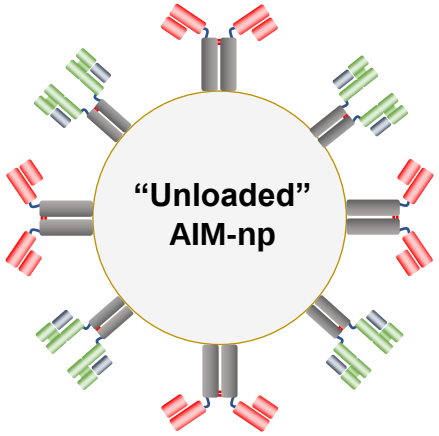
Same proteins, np conjugation chemistry (change in core material)

Speed, Scale and Optionality: Reduced to Practice

Potential for more rapid multi-antigen product development within months - an "IND engine"

HPV product candidate (NEXI-003): From target validation and selection to filing new product IND in ~6 months*

Antigen-peptide "Unloaded" AIM nanoparticle



- *Manufactured in advance and stored in bulk aliquots for future use*
- *3 clinical lots for ACT produced*

Validation and selection of disease specific antigen targets
e.g. HPV example 2 months

ZEPHYR AI

DANA-FARBER CANCER INSTITUTE

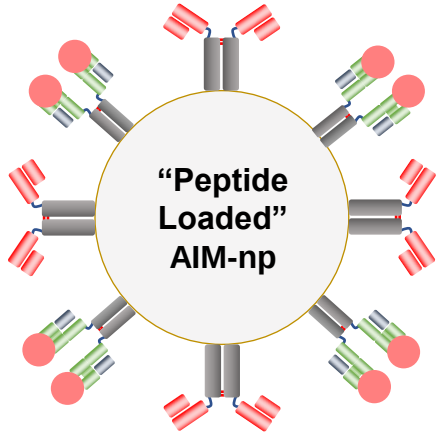
NYU Perlmutter Cancer Center

RUTGERS

NexImmune

- *Partner world-class discovery and AI capabilities combined with NexImmune efforts*
- *Ability to screen and validate T cell responses to antigen targets which informs final selection (HPV example: validation screening of **50 peptides in 8 weeks**)*

Rapid multi-antigen new product development
*e.g. NEXI-003 example 6m**

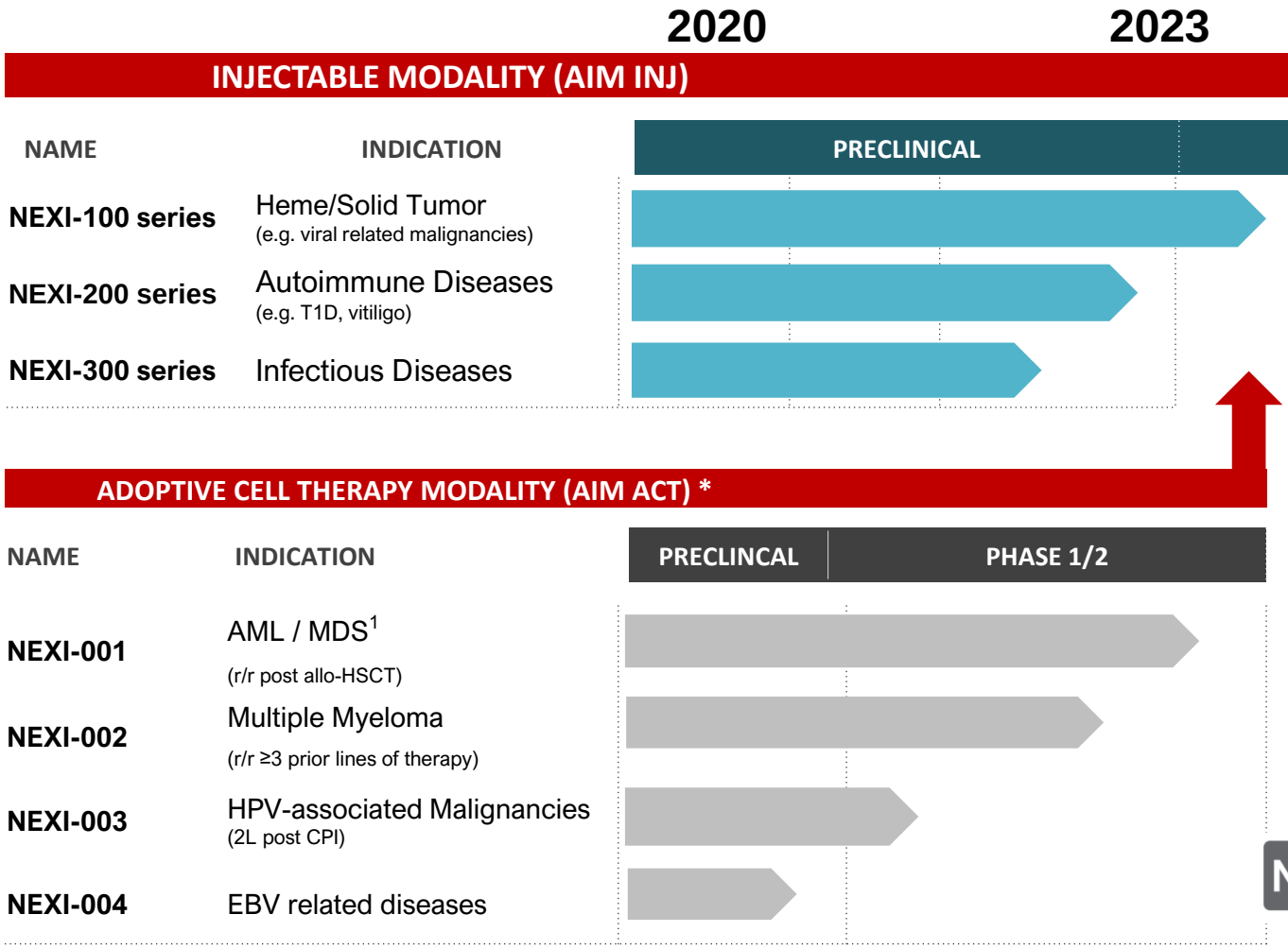


- *Rapid loading of multiple antigens **(hours)***
- *Reduces time from target selection to new product I**ND to months***
- *Nanoparticle is "loaded" with single peptides and combined to make a custom disease specific mix*

*Timing influenced by external CDMO; fill finish and release schedule

Pipeline: Developing the AIM technology in multiple therapeutic areas

Early collaboration with world-class centers, significant opportunity for disease specific partnering



Key Collaborators

NYU Perlmutter Cancer Center
Yale University
Johns Hopkins University
JDRF
NIH National Institutes of Health
Turning Discovery Into Health
City of Hope
The Ohio State University
Memorial Sloan Kettering Cancer Center
NIH National Institute of Neurological Disorders and Stroke
Dana-Farber Cancer Institute
Columbia University

[1]Acute Myeloid Leukemia (AML) or Myelodysplastic Syndrome (MDS) who have relapsed disease after an allogeneic hematopoietic cell transplant (HCT)
Clinical Trial: NCT04284228 https://clinicaltrials.gov/ct2/show/record/NCT04284228
Additional HLA's in development

*AIM ACT programs have currently paused enrollment. NexImmune is actively seeking academic and industry partners and collaborators to continue development of the AIM ACT programs.

Oncology

The Power of Multi-Antigen-Specific T cells

AIM T cell Directing Approach: Combination of Differentiating Attributes

T cells are the most effective mechanism to identify and clear cancer cells and establish immunologic memory

1 Attack Multiple Tumor Targets Simultaneously

- AIM T cell populations that can **attack** multiple tumor-specific antigen targets **simultaneously to address heterogeneity**
- AIM T cells can **attack a broad range of antigen targets** - cell-surface proteins and survival proteins or neoantigens to increase response and limit escape
- AIM T cell are active on tumors bearing non-targeted genetic and epigenetic changes

2 Increase Response and Persistence

- AIM T cell subtypes (stem-cell-like memory T cells, central memory T cells) are associated with **potent killing, self-renewal and long-term immunologic memory**
- Unlike fully differentiated effector T cells, which exhaust themselves in weeks or a few months (leading to tumor relapse), these subtypes **persist for years or decades**
- **Potent** AIM activated specific T cells, **traffic to the tumor site and persist**

3 Natural TCR Safety Profile

- AIM T cells maintain natural target recognition, engagement, activation and killing mechanisms
- Can effectively **distinguish healthy cells from tumor cells** — low potential for on-target/off-tissue toxicity
- Express a **broad range of TCRs** with both high and low affinity

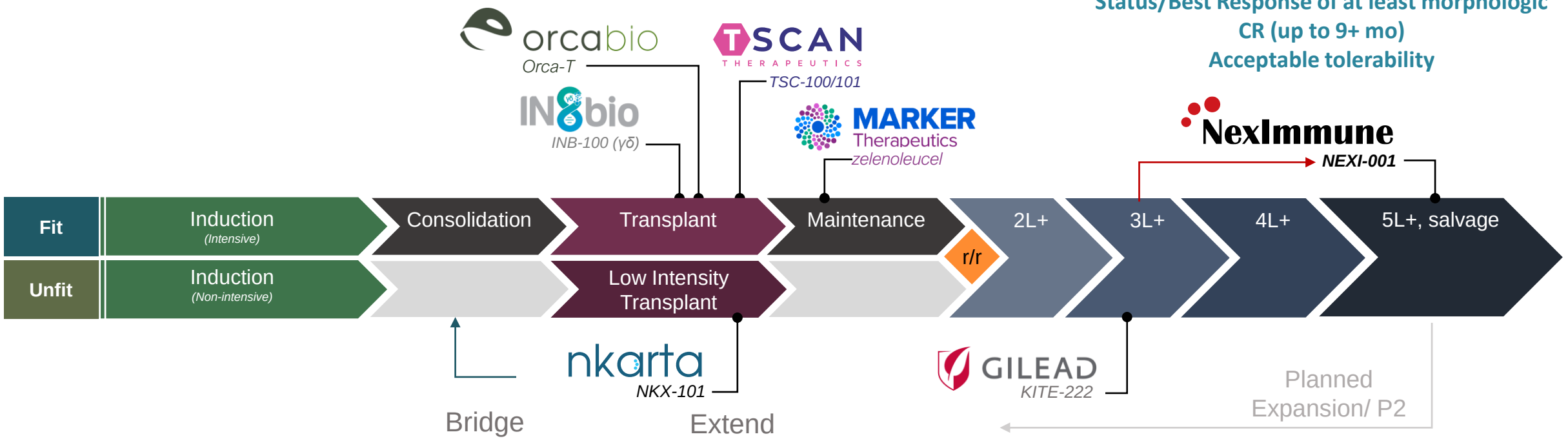
NEXI-001 AML Therapeutic for r/r AML post allo-SCT and salvage therapy

POC for multi-AML antigen-specific T cell approach as cell therapy – **5 antigen peptide targets**

Concentrated clinical activity of cell therapies around transplant window **leaves high unmet need for heavily pretreated patients with no approved options**

- Rapidly progressing, heterogeneous disease where most patients succumb within 1-year and the 2-year survival rate is < 10-15%.
- Infrequent remission rates of limited duration further decline with later lines of therapy, even when combining options

4 of 7 patients in last 2 cohorts achieved Best Status/Best Response of at least morphologic CR (up to 9+ mo)
Acceptable tolerability



Cohort 2: 200M / cycle (1-2 cycles)
Cohort 3: 600M / Cycle (1 cycle)

NEXI-001: Observed Dose Response

Percent of Antigen-specific CD8+ T cells increase and persist in blood and marrow with increasing dose, maintaining important phenotype

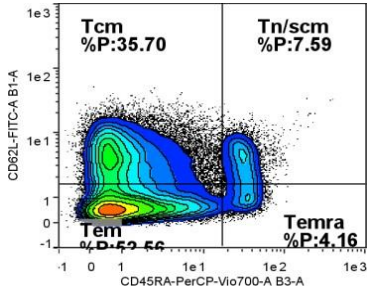
Antigen-specific T cells persisted and expanded in peripheral blood, trafficked to bone marrow and persist

Total Antigen Specificity (% of CD8+ T cells)				
	Cohort 2 Patient 8 (200M)		Cohort 3 Patient 10 (600M)	
	Visit	Total Specificity	Visit	Total Specificity
Blood	M1	5.86	M1	14.54
	M3	9.10	M3	14.66
Bone	M1	33.86	M2	**83.75**
	M3	51.89	M3	**72.23**

Antigen-specific T cells maintain important memory subtypes over time

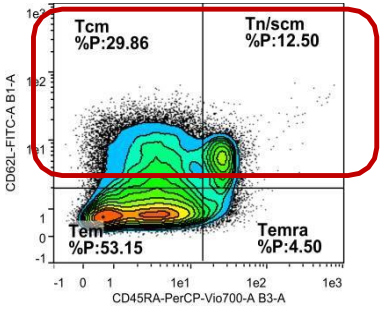
Patient 8

NEXI-001 Product

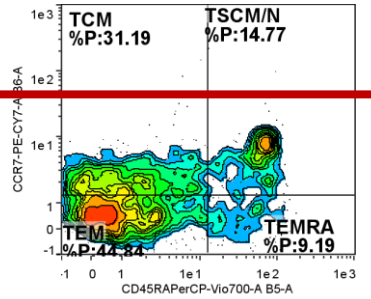


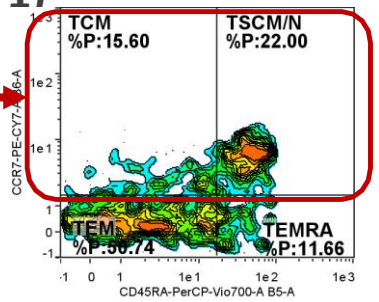
Patient 10

NEXI-001 Product



Month 3 memory of specific cells in bone marrow (WT1)





Optimize anti-tumor potency and durability in heterogeneous tumors

Novel Combination of Bispecific TCE with AIM TAA-specific "Fit" CD8+ T cells

Article

Cancer Cell

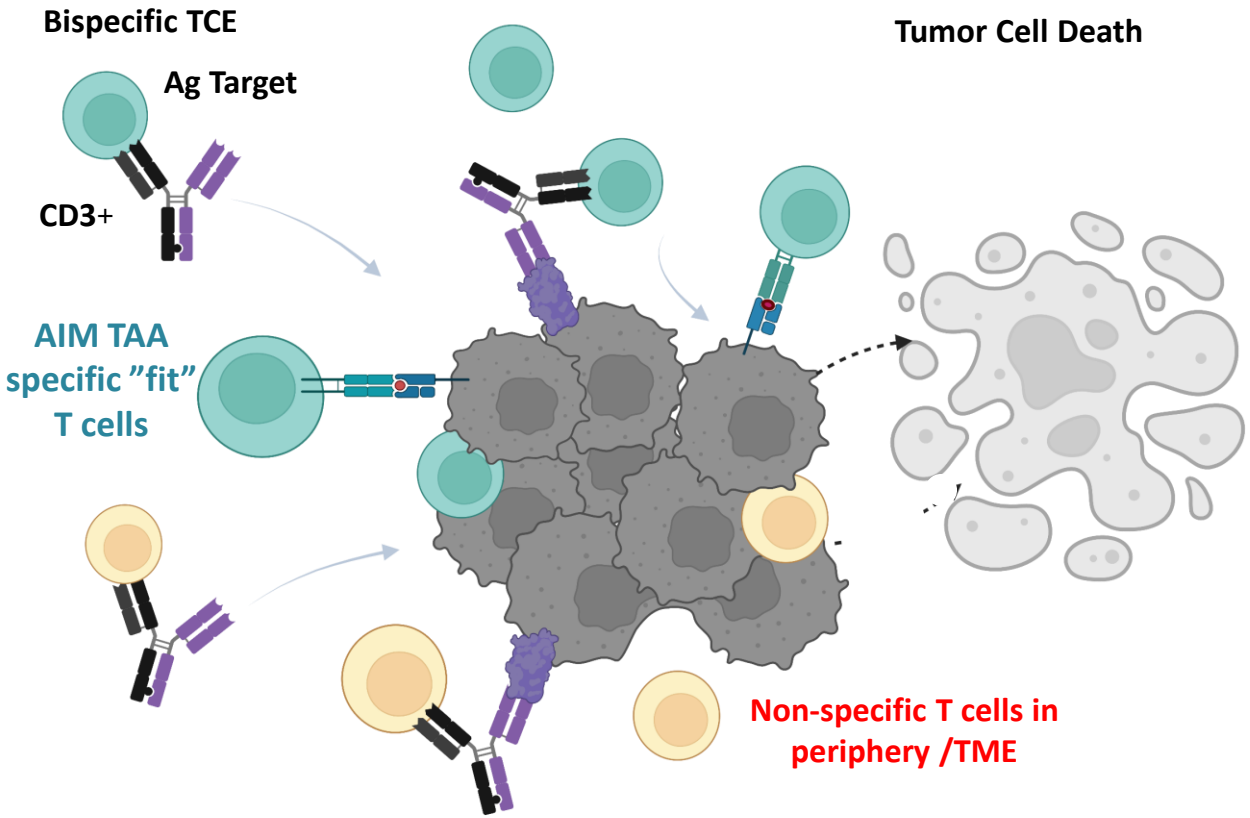
The pre-existing T cell landscape determines the response to bispecific T cell engagers in multiple myeloma patients

1. **Increase potency and address escape**
   - Increase number of tumor targets (intracellular, surface)
   - Increase polyclonal multi-TAA specific T cells in tumor (direct trafficking to the tumor / T cell redirecting to tumor)
   - Combine MHC restricted and unrestricted killing

2. **Increase persistence / durability**

   TAA long lived memory cells, Tscm, Tcm in absence of bispecific

Dual Mechanism of Action

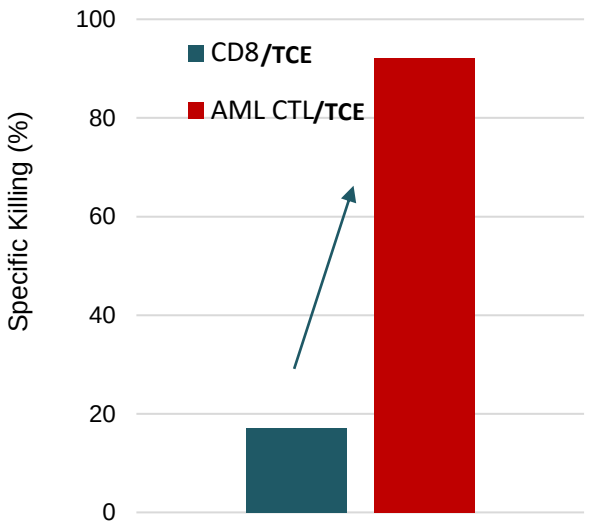


(Mirco J. Friedrich et al, The pre-existing T cell landscape determines the response to bispecific T cell engagers in multiple myeloma patients, Cancer Cell, 2023).

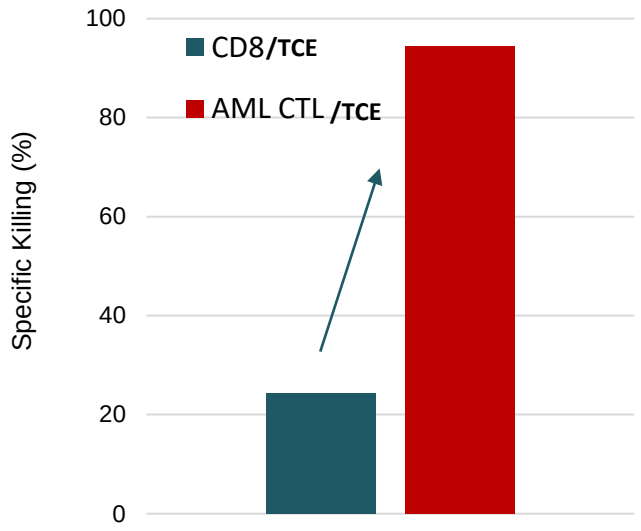
Superior Potency when combining Bispecific TCE[1] combination with AIM multi-targeted T cells across AML and Multiple Myeloma (MM) cell lines *(low doses)*

Combines HLA dependent / independent killing, expands target repertoire and increases specific, fit memory /effector T cells at the site of tumor - demonstrating superior potency compared to bulk $CD8^+$ T cells + TCE (tumor cell lines); low doses of both agents

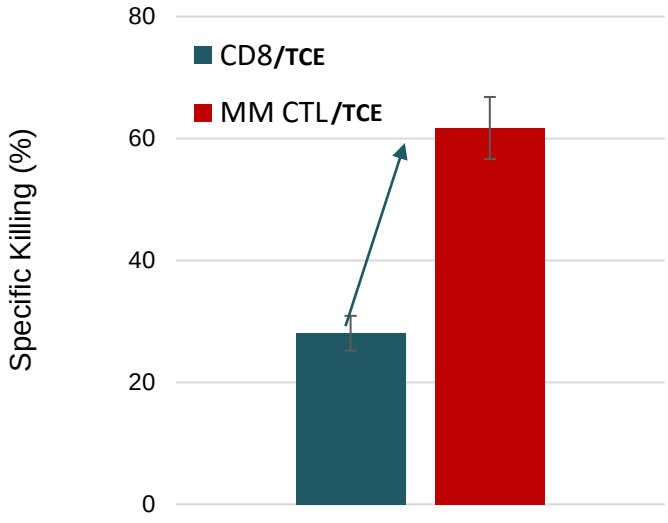
AML specific T cells + FLT3 TCE



AML specific T cells + CD123 TCE



MM specific T Cells + BCMA TCE



AIM AML and MM specific CD8+ T cells are more "fit" defined by high proportions of Tscm, Tcm and Tem vs non-specific bulk CD8+ T cells from HD that were used to mimic TCE monotherapy

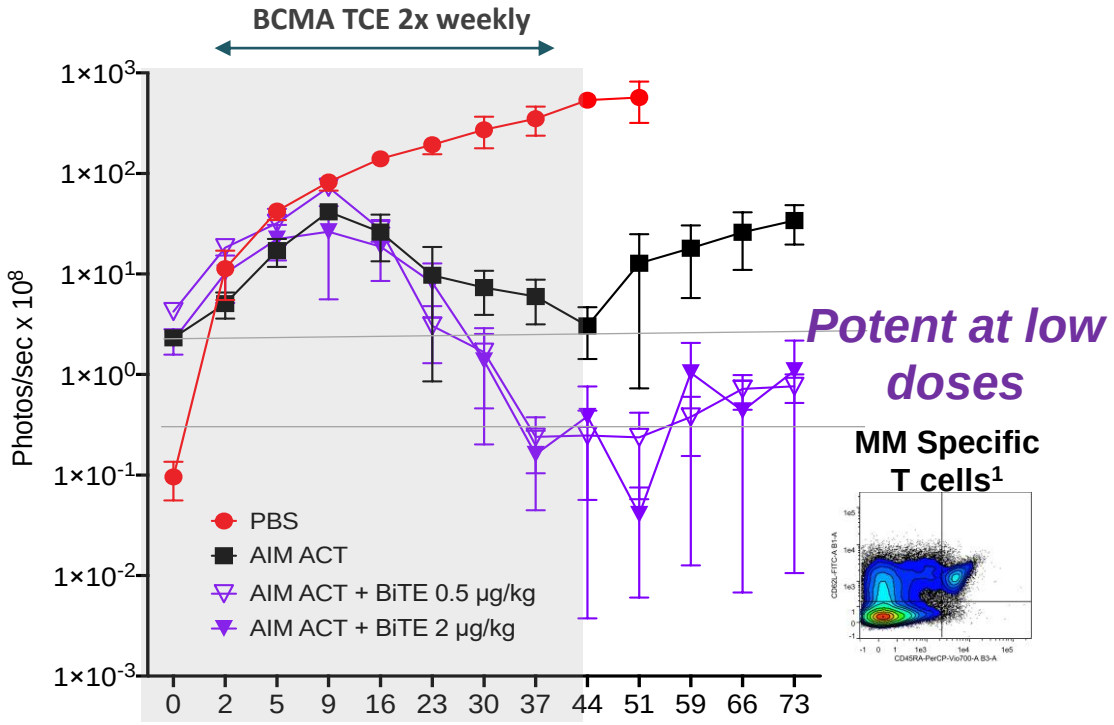
Low E:T ratio + Low dose Bispecific; Antigen specific cells include Tscm, Tcm, Tem memory phenotypes associated with anti-tumor activity and persistence

[1] TCE: aCD3 /Targeted T Cell Engager or BiTE's

Multiple Myeloma: Superior Potency and Enhanced Persistence Combining BCMA TCE with AIM multi-antigen specific CTL[1] *(Low Doses of both)*

aCD3 / BCMA Bispecific + AIM ACT MM T cells provide Superior Killing and Survival

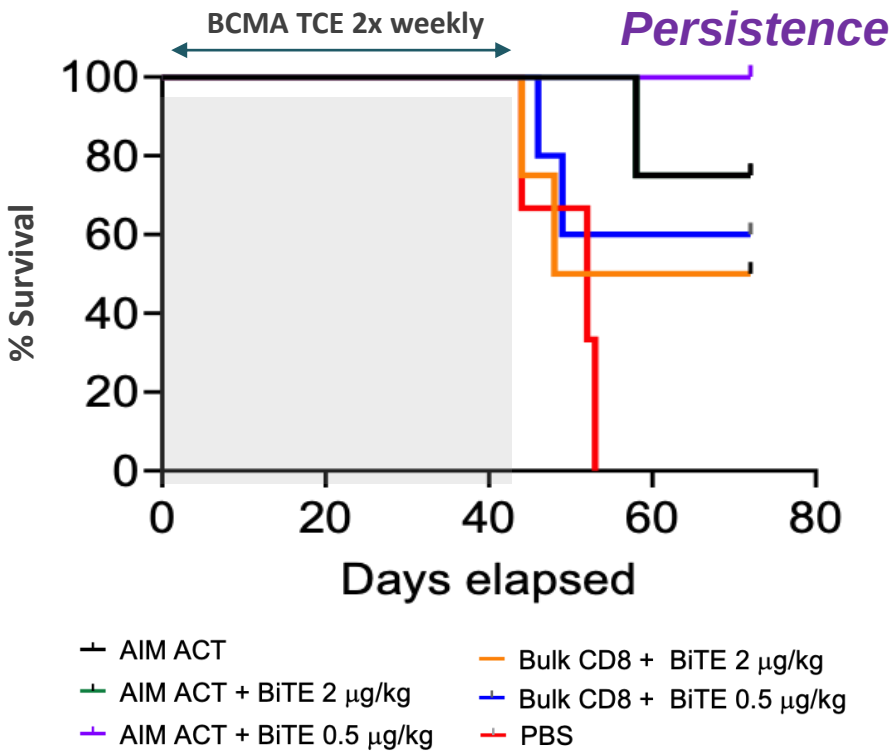
Durable superior killing



Potent at low doses

MM Specific T cells[1]

Increased Survival

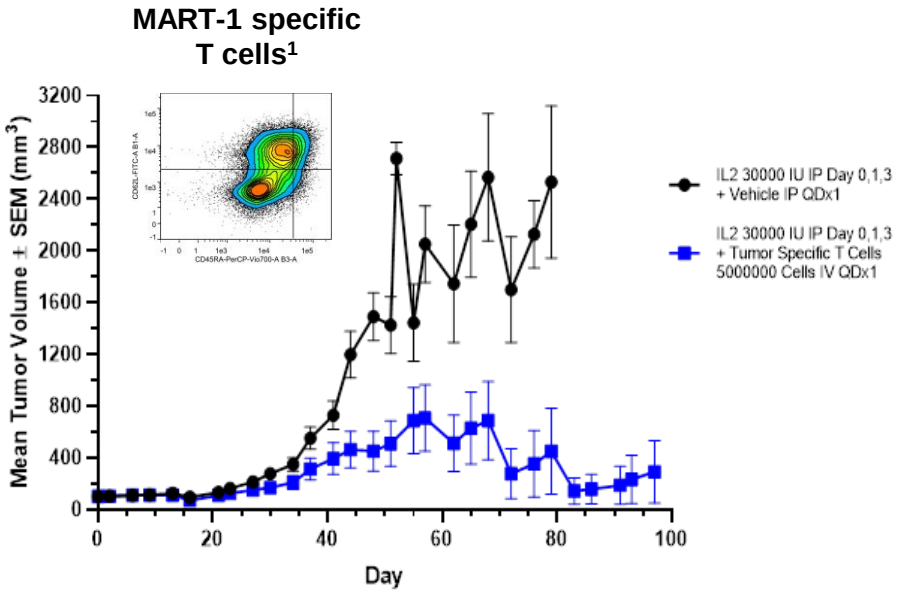


Hackensack Meridian *Health*

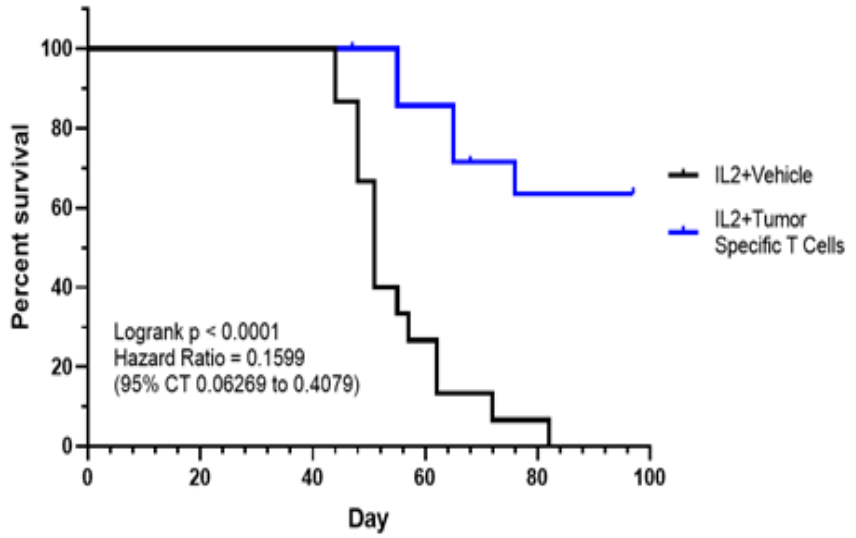
Data from Hackensack Lab; Day 0 tumor established, treatment started; Lower dose of T cells and low dose of BCMA bispecific agents

Melanoma: Superior anti-tumor effect and increased survival
NEXI MART-1-specific T cell treated mice

Superior Reduction in Mean Tumor Volumes Over Time
Human Melanoma Model (PDX)



Superior Survival - Kaplan-Meier Survival Plot of Time to Tumor Volume ≥ 2000 mm³
Human Melanoma Model



Persistence of MART-1 CD3+CD8+ T Cells (TIL) Over Course of Study (D97 last day measured)

In the treatment group, 8 /15 mice (53%) survived to end-of-study (D97), with ORR = 6CR, 1PR, 1SD. All vehicle-treated mice expired on study.

[1] MART-1 specific T cells primarily Tscm, Tcm and Tem phenotypes associated with anti-tumor killing and persistence

Melanoma: AIM expanded CD8+ T cells possess superior anti-tumor activity compared to those expanded by mature peptide-pulsed DCs

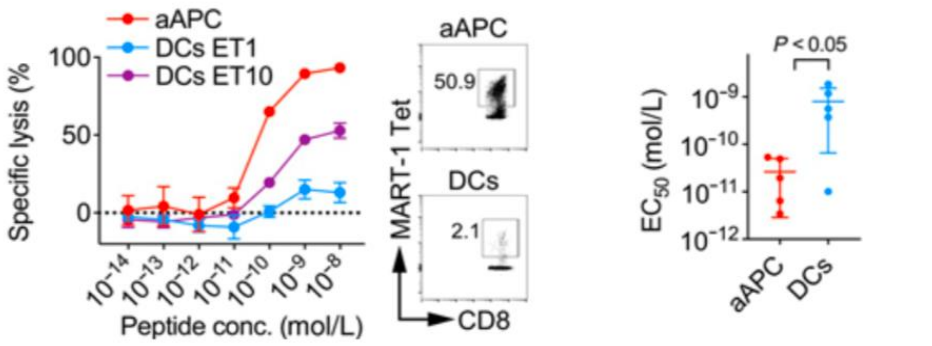
(Melanoma patients n=3, PBMC)

Study Conclusions

- Demonstrated consistent expansion of MART-1 from HD and Melanoma patient (PBMC)
- Significantly greater killing with AIM expanded MART-1 T cells vs DC expanded T cells
- AIM expanded MART-1 T cells were equally polyclonal (TCR diversity) with greater affinity vs DC expanded T cells

Rapid Expansion of Highly Functional Antigen-Specific T Cells from Patients with Melanoma by Nanoscale Artificial Antigen-Presenting Cells

T cell Affinity | **Functional Avidity**



NYU Perlmutter Cancer Center

Current Project:

Evaluate ability of AIM nanoparticles to expand **neoantigen-specific T cells in previously treated melanoma patients**

The AIM INJ nanoparticle directly expands multi-antigen specific T-cells designed to address heterogeneity and increase durable efficacy

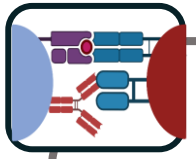
Early success with modalities focused on increasing antigen specific CD8⁺ T killing

Cancer Therapeutic Vaccines
(rely on host DC to activate T cells)

moderna messenger therapeutics · BIONTECH · gritstone bio

Bi- (Tri-) specific TCE Antibodies

AMGEN · IMMUNOCORE · Bristol Myers Squibb™ · REGENERON® · janssen PHARMACEUTICAL COMPANIES OF Johnson&Johnson · sanofi



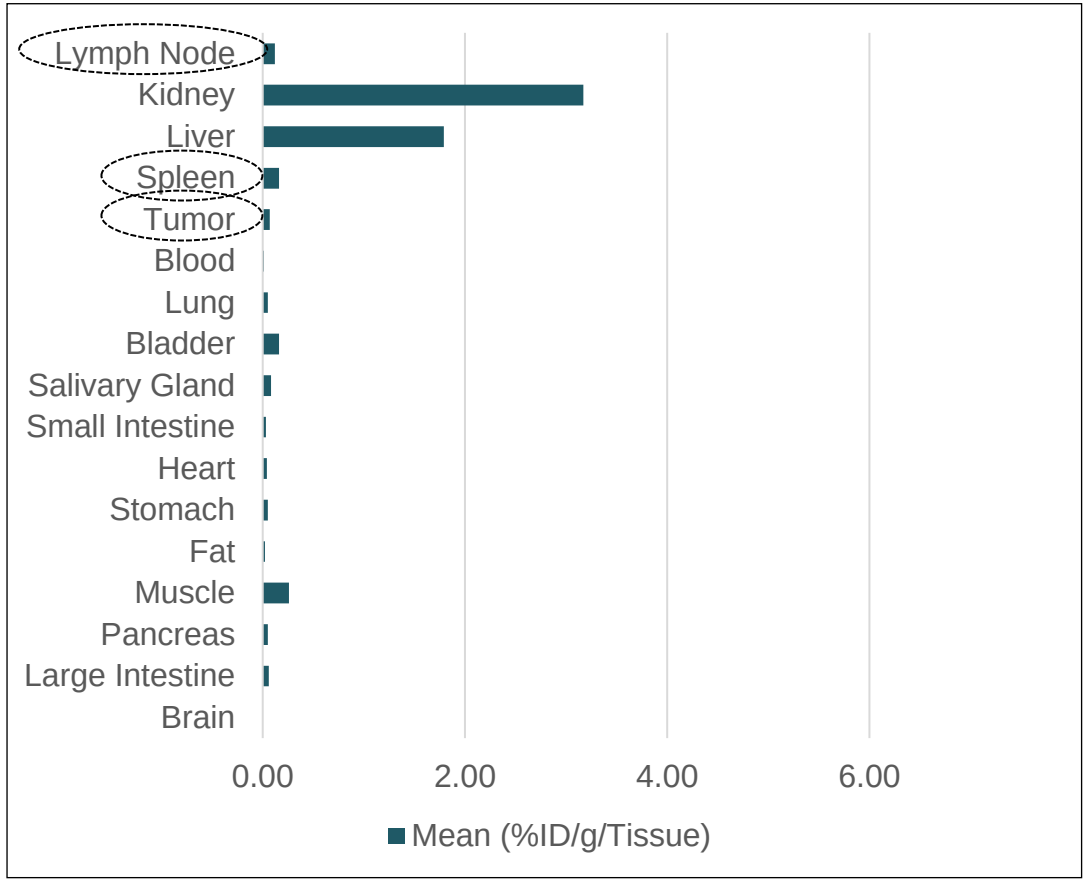
NexImmune

- **Direct activation and expansion of antigen-specific T cells,** bypassing dendritic cells – which frequently become dysfunctional in cancer
- **Broad accessible targets (surface, intracellular)**
- **Simultaneous activation of "fit" multi-tumor specific CD8+ T cells (heterogeneity) for increased efficacy and durability**
- **Scalable, Off-the-shelf -simplify logistics, access**

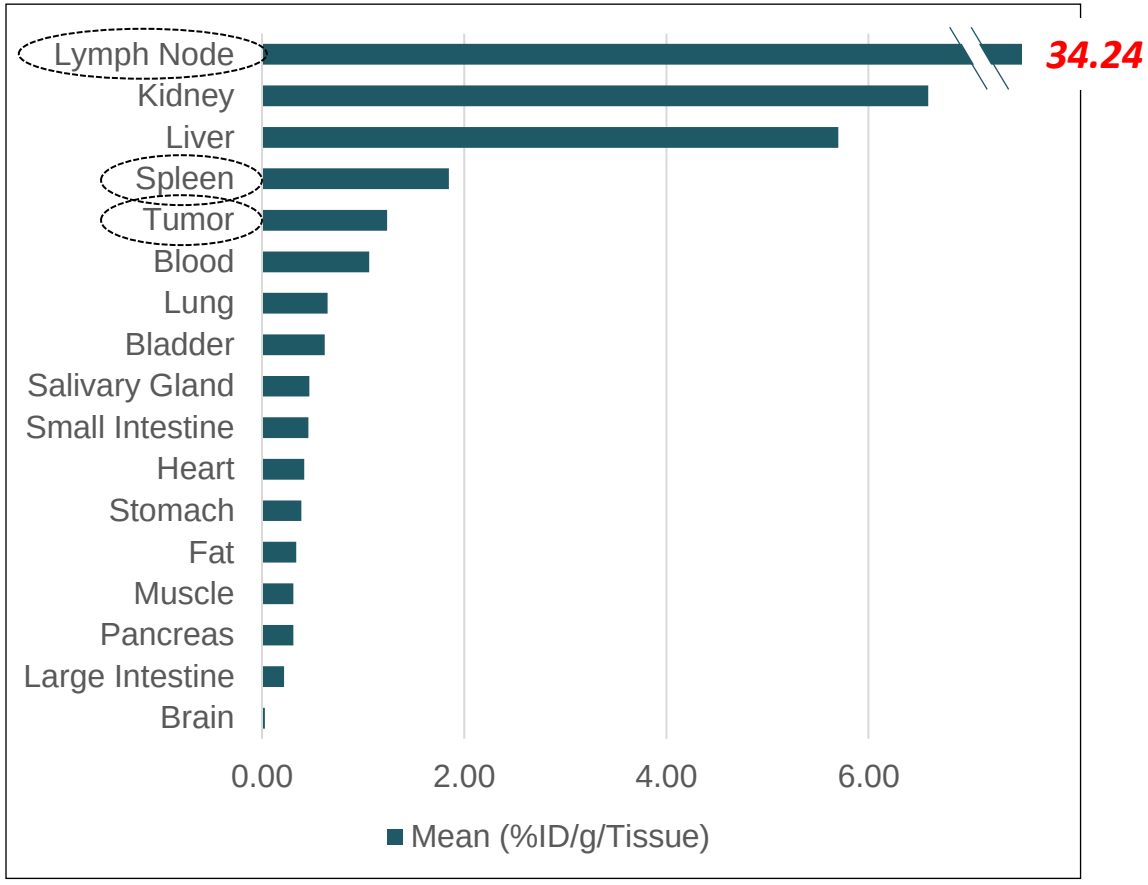
“Antigen-peptide Loaded” AIM INJ nanoparticles traffic to lymph nodes, spleen and tumor compared to “naked” nanoparticles

Studies done in an oncology model illustrate how AIM nanoparticles (e.g., loaded with target antigen) that are administered systemically travel naturally to critical immune sites (96 hrs)

“Naked” nanoparticle without proteins or peptide targets in tumor-bearing mice

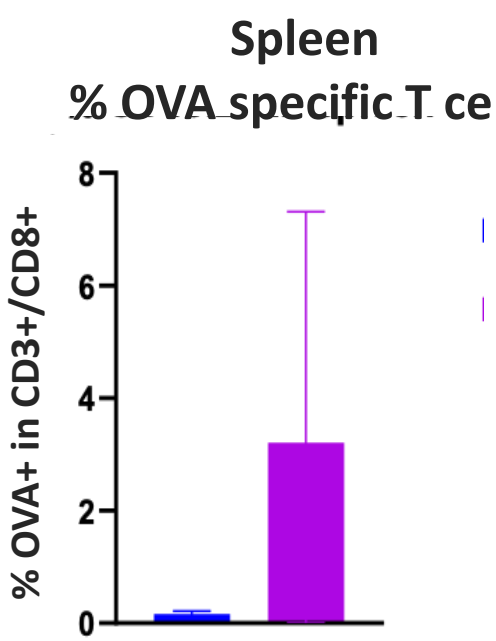


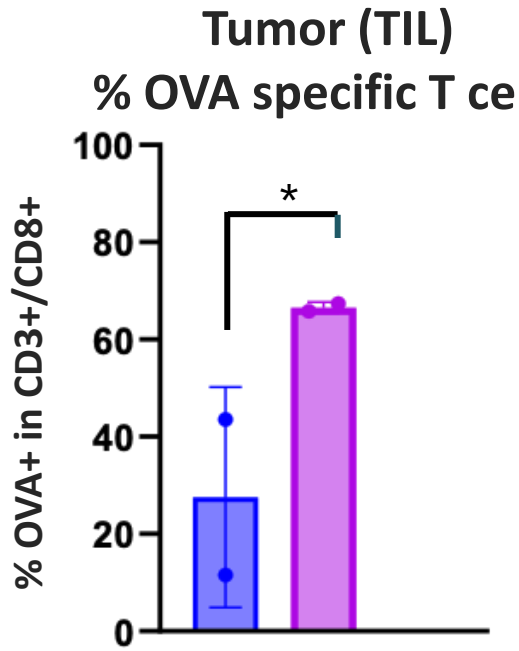
AIM nanoparticle with peptide-loaded proteins in tumor-bearing mice

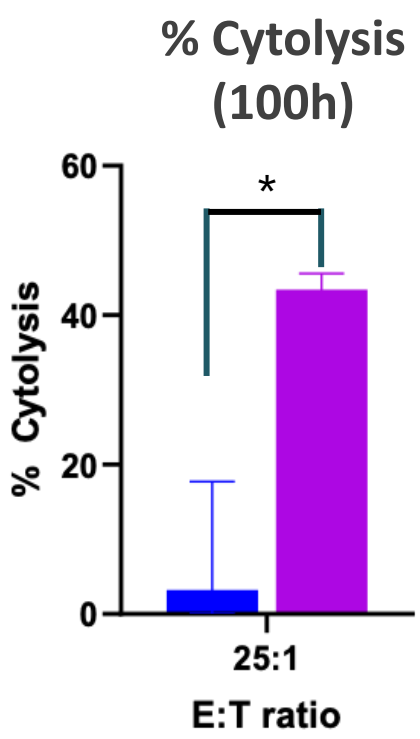


AIM INJ NPs significantly increased antigen-specific T cells in spleen and tumor with greater killing when compared to peptide in CFA

B16-OVA (implanted OT-1 T cells); OVA loaded NP compared to OVA peptide in CFA







In vitro killing assay with splenocytes harvested on day 22 (n=2)

N=2 / arm; D22

OVA peptide CFA

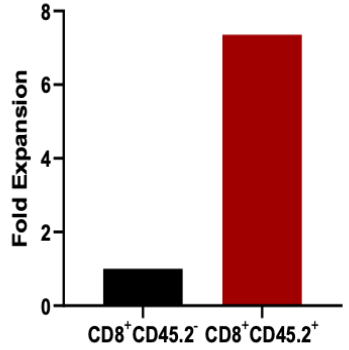
AIM NP OVA 2 mg

Reproducible: 7-16-fold increase of antigen-specific activated T cells using “Peptide loaded” INJ nanoparticles (3 models, multiple targets)

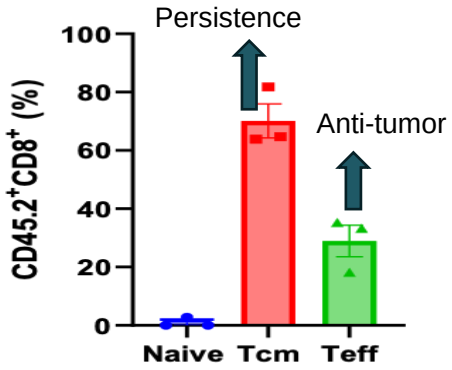
The activated antigen-specific T cells are polyfunctional, maintaining effector and memory functions with no exhausted phenotypes

7-fold increase in OVA specific CD8+ T cells in LN

(non-disease, MOA study, day 7)

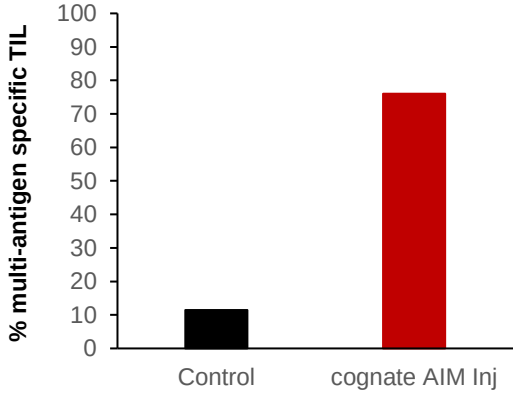


…which maintain effector and memory functions

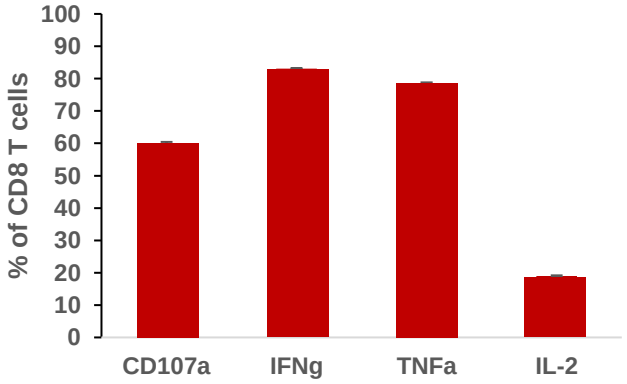


7-fold increase in multi-antigen specific CD8+ T cells in tumors

(melanoma, B16F10)



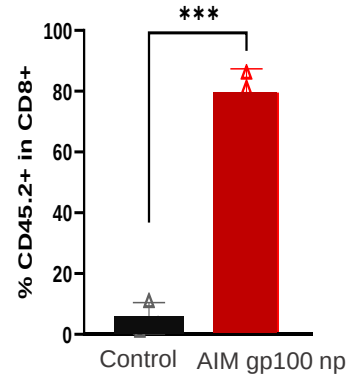
…which are polyfunctional with 63% of CD3+/CD8+ T cells having 3+ effector functions



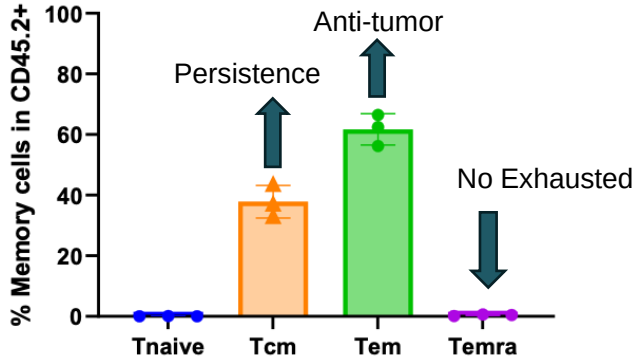
16-fold increase in gp100 specific CD8+ in tumors

(melanoma B16F10, D13)

CD8+ TIL CD45.2+ (gp100)



..which maintain effector and memory functions with continued exposure to tumor

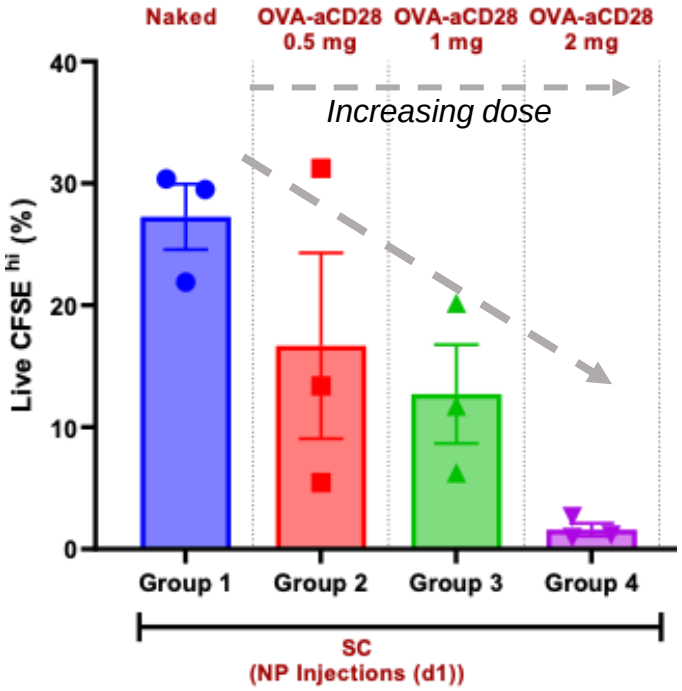


Significant AIM np single ascending dose dependent *in vivo* killing of antigen pulsed target cells in Spleen and LN

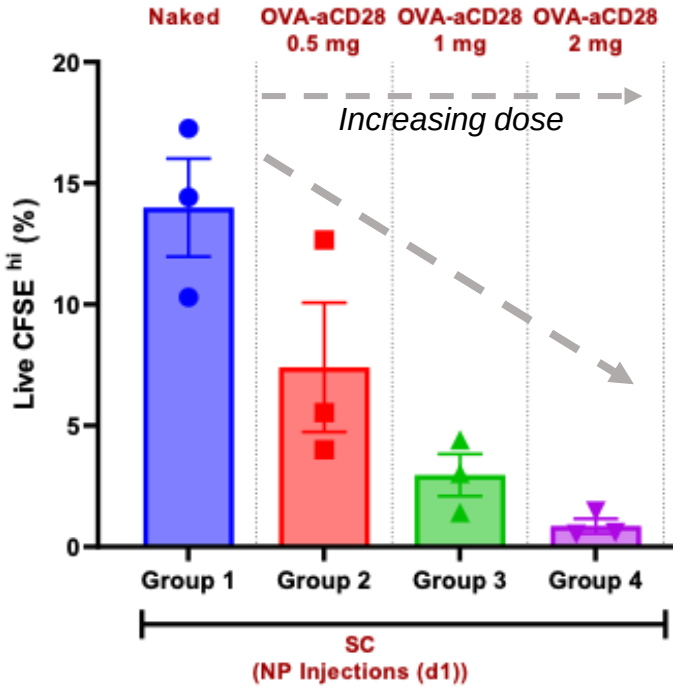
(OVA-loaded AIM INJ activated T cells)

Mice were injected with OT-1 cells (D0); single, ascending sc dose of np (D1); CSFE labeled, peptide pulsed targets (splenocytes) were administered i.v. (D9); and killing of target cells was measured by flow (D10)

Dose Dependent Specific Killing Spleen ($CFSE^{hi}$)



Dose Dependent Specific Killing Lymph Node (LN) ($CFSE^{hi}$)



N=3 mice/group; CFSE fluorescence cell mediated killing assay

NEXI-101 AIM INJ Product: HPV+ HNSCC Lead indication

Multi-antigen targeted approach has broad potential in HPV+ Cancers

- **HPV+ cancers represent ~5% of all cancers globally** ~300,000 new cases / year globally, and **>45,000 new cases per year in the US (ref)**
- **HPV vaccines are not approved to treat related malignancies** and with marginal vaccination rates, many remain vulnerable
- Treatments include chemo and checkpoint inhibitors (CPI) yet **responses remain low**
- **Estimated 79%** of cervical, vulvar, penile, vaginal, anal, and oropharyngeal cancers attributed to HPV, **predominantly high-risk HPV 16 and 18 strains.**
- **Oncoproteins E6 and E7 are important for cancer survival; and DC impairment in directing T cells to tumor targets** has been observed in cervical and HN cancers

NEXI-101 HPV+ Solid Tumor Vision

Direct simultaneous T cell responses against both high-risk strains and cancer survival proteins

Targets: HPV-16 (E6, E7) and HPV-18 (E6, E7), survivin

1. **ESTABLISH monotherapy (Head and Neck cancer)**

   Dose escalation (RP2D) and expansion phase

2. **COMBINE with current CPI SOC (RP2D)**
3. **EXPAND into basket trial of HPV+ cancers and additional HLA's when available**

RP2D, Recommended Phase 2 Dose; PD, pharmacodynamics; CPI, Checkpoint Inhibitor(s) approved for the indication

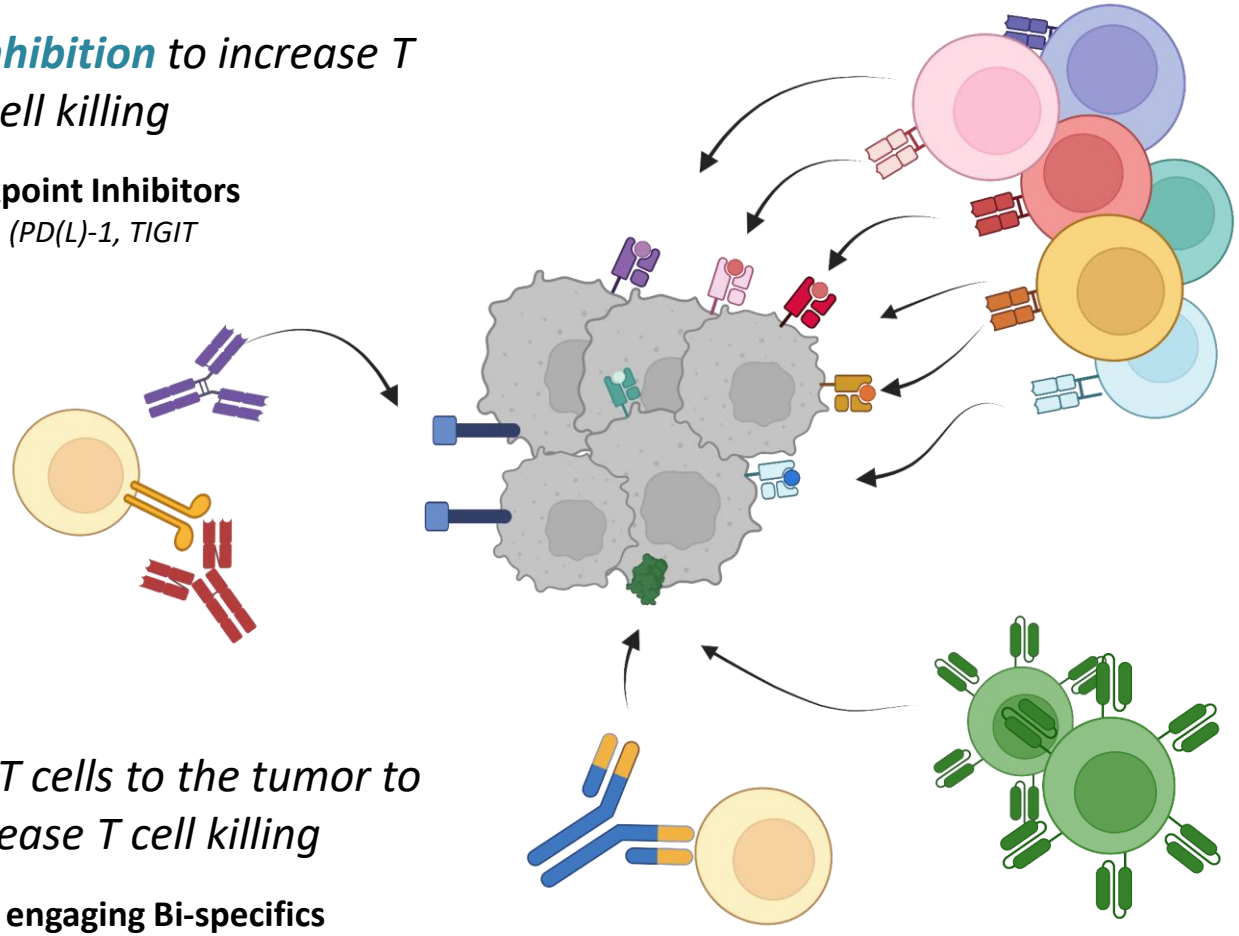
IO/IO treatment paradigm shift:

Combining multi-antigen T cells with current and emerging approaches to limit escape and increase response

Block T cell inhibition *to increase T cell killing*

Checkpoint Inhibitors

e.g. (PD(L)-1, TIGIT



Increase multi-TAA T cell killing *to limit escape and increase persistence*

NexImmune

Multi-TAA products

Redirect *T cells to the tumor to increase T cell killing*

T cell engaging Bi-specifics

Increase CAR-T cell killing

CD19, BMCA, other

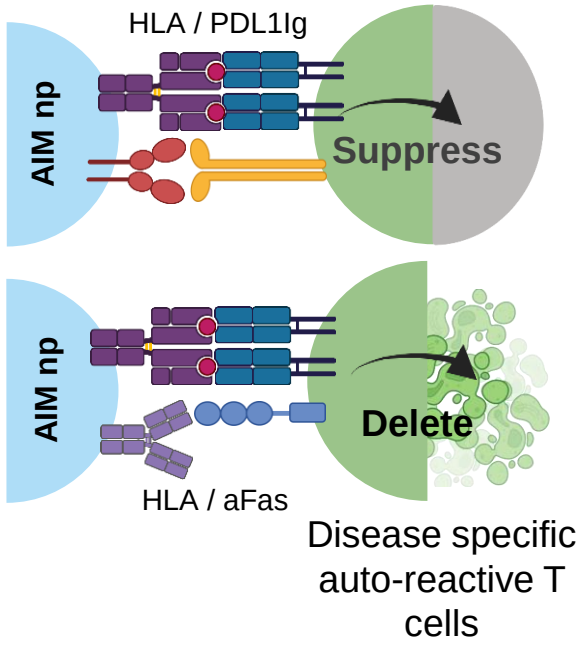
Beyond Oncology

Autoimmune Disorders and Virally Driven Diseases

Autoimmune Disorders: AIM nanoparticles designed to target and suppress, or delete disease causing antigen specific T cells

Antigen-targeting therapies are emerging focus in autoimmune disorders; the AIM™ platform has the potential to address multiple areas of unmet need

MHC Class I
(CD8+ T cells)

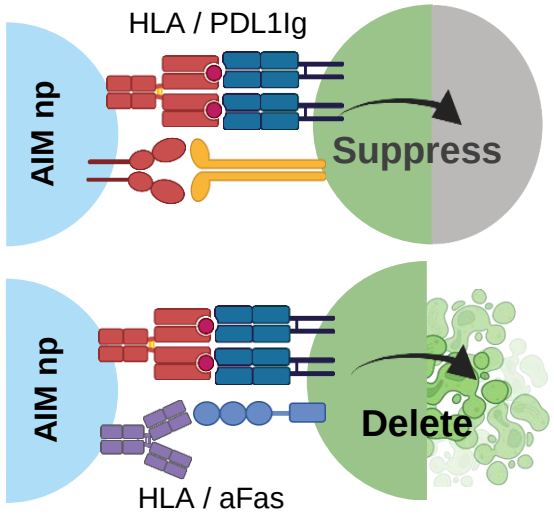


Disease specific auto-reactive T cells

Illustration of T-cell Mediated Diseases
> 80 Diseases with 24 Million US Affected[1]

Alopecia	**Rheumatoid Arthritis**
Ankylosing Spondylitis	**Scleroderma**
Celiac Disease	**Sjogren's Disease**
Crohn's Disease	**Systemic Lupus (SLE)**
HAM/TSP	**Type 1 Diabetes**
Multiple Sclerosis	**Vitiligo**

MHC Class II
(CD4+ T cells)



Disease specific auto-reactive T cells

Leading experts guide T1D program: Yale | JDRF

Recent publications in Type 1 diabetes suggest targeting stem like $CD8^+$ autoreactive cells could emerge as a novel, powerful intervention

- Collaboration with Dr. Kevan Herold and Yale for murine model research
- Dr. Gerry Nepom (Director, Immune Tolerance Network) as Chair of NexImmune's Autoimmune SAB
- Based on early findings, received significant grant from Juvenile Diabetes Research Foundation (JDRF) to support and accelerate work
- Data continues to be encouraging and supports our technology as potentially breakthrough and disruptive therapeutic in T1D

NexImmune: Autoimmune SAB Members

Kevan Herold, MD, C.N.H. Long Professor of Immunology, Yale University

Gerry Nepom, M.D., PhD Director, ITN; Founder of Benaroya Research Institute

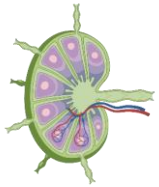
[1]Nature Immunology | VOL 21 | 578 May 2020 | 578–587 | www.nature.com/natureimmunology
NATURE https://doi.org/10.1038/s41586-021-04248-x (2021

AIM INJ significantly reduces and inhibits T1D disease causing T-cells in lymph node and pancreas delaying onset of T1D (Antigen presentation, signal 1 only)

T1D specific T cells in the pancreas of 13-week-old NOD mice are significantly less activated than in control mice; Signal 1 (Antigen presentation)

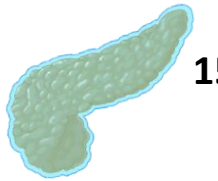
T1D specific T cells in pre-diabetic NOD mice

Pancreatic Lymph Node (LN)



1-6% T1D NRPV7+ T cells in LN

Pancreas

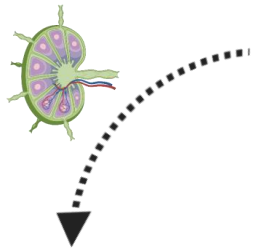


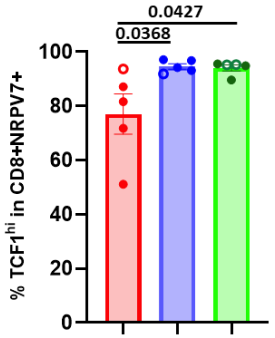
15-30% T1D NRPV7+ T cells in pancreas

After 7 weeks, S1 only

~20% Reduction of T1D disease renewing cells

Tscm, lymph node



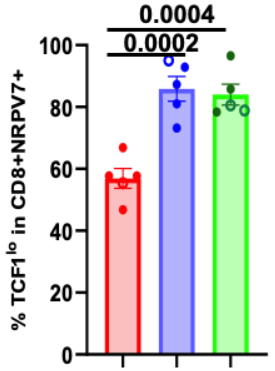


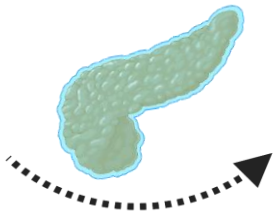
nature

An autoimmune stem-like CD8+ T cell population drives Type 1 diabetes*

~25% Reduction of TID T cells

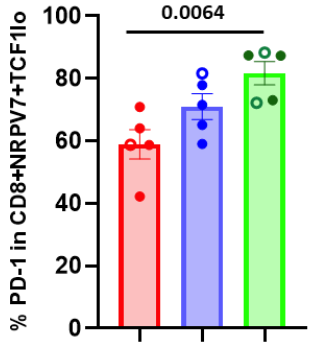
Tem, Pancreas





~30% Reduction of T1D T cell activation

Tem, Pancreas



AIM np

Control

Untreated

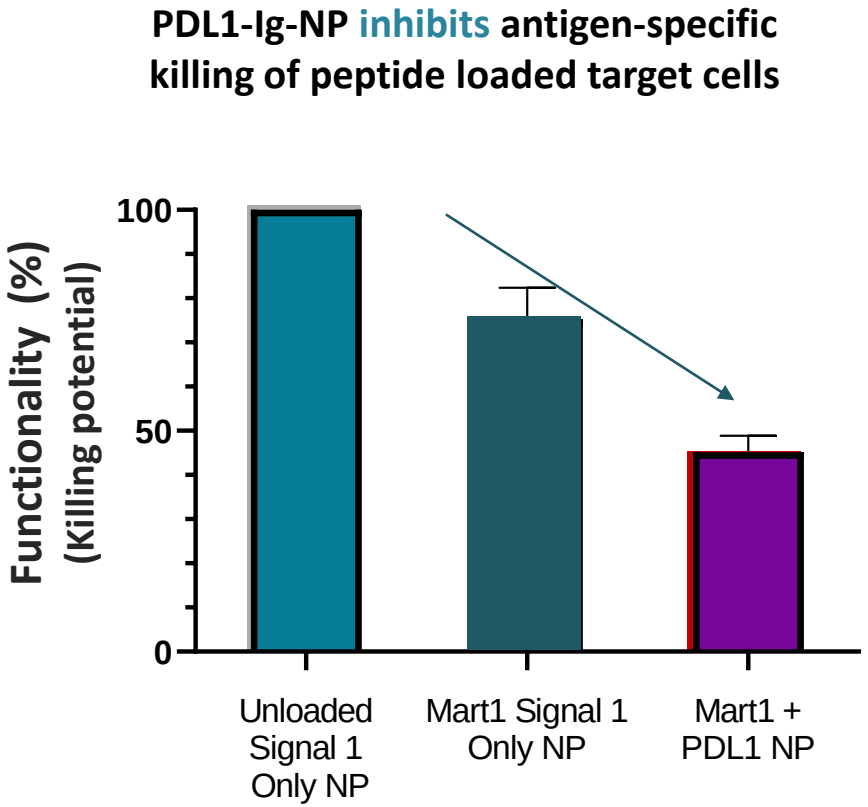
Yale

*Gearty, S.V., Dündar, F., Zumbo, P. *et al.* An autoimmune stem-like CD8 T cell population drives type 1 diabetes. *Nature* **602**, 156–161 (2022)

Open symbols indicate diabetic; solid symbols indicate pre-diabetic

S1+S2 POM: HLA/PD-L1-Ig inhibits antigen-specific CD8+ T cell function (cytotoxic activity) – Yale initiated in vivo study

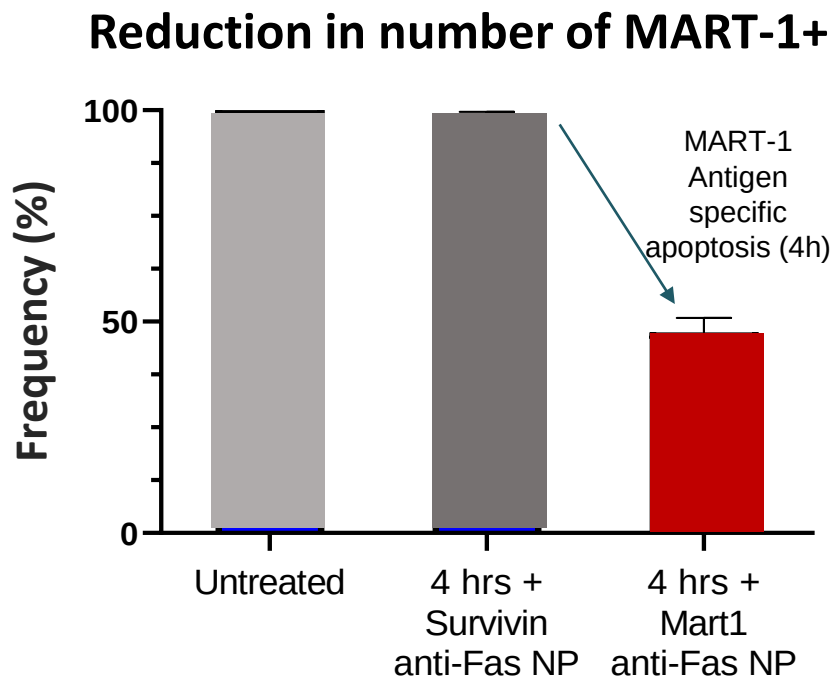
Signal 2 options in development to enable antigen-specific suppression (PDL1-Ig) or elimination (anti-Fas) in autoimmune diseases



Mart-1 cells were incubated for 90 minutes with 50 ug/ml nanoparticles. After washing cells were incubated with peptide loaded targets for 4 hours and antigen specific killing was assessed by caspase 3/7 assay.

S1+S2 POM: Combined HLA / a-FAS nanoparticles demonstrate initial antigen-specific CD8+ apoptosis within 4 hours leaving non-target cells alone

HLA / anti-Fas np eliminates 50% of MART-1 antigen-specific CD8+ T cells



- Dose dependent effect observed
- Time course evaluation planned

Novel functional validation:
3rd dimension can enhance target discovery and screening

Well understood that immune cells can become dysfunctional in the face of cancer and certain other diseases

Multiple approaches to target discovery

- Tissue, tumor, TCR
- Genomic, proteomic, immunopeptidomes and transcriptomes

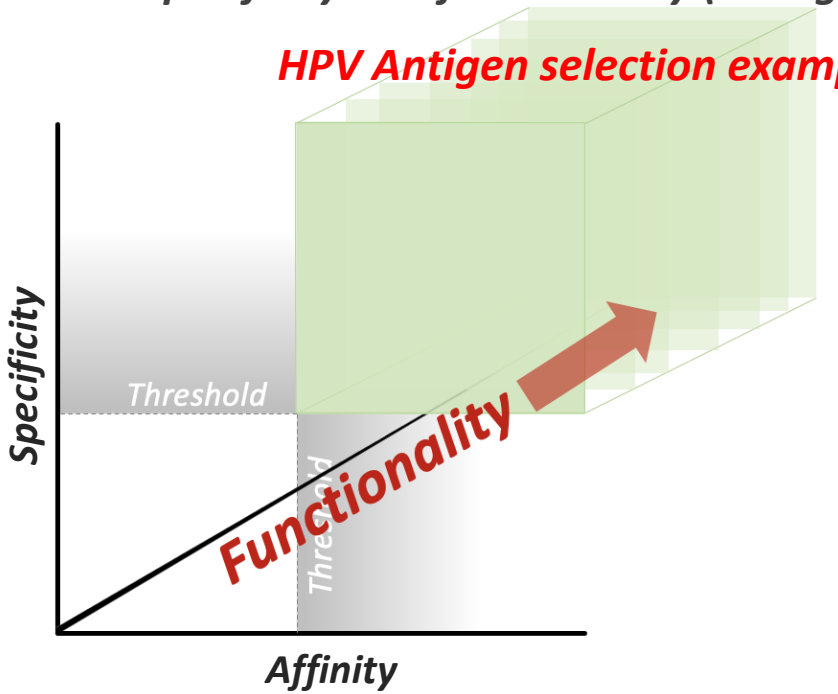
Screening and algorithms primarily focus on:

- High affinity TCR's (assumes target on tumor)
- Prevalence of target (on tumor cells)
- Affinity enhanced TCR's
- Absence /low detection on healthy tissue

3rd Dimension: Functional T cell response

Multiplex interrogation of antigen T cell responses - Specificity and functionality (killing capacity)

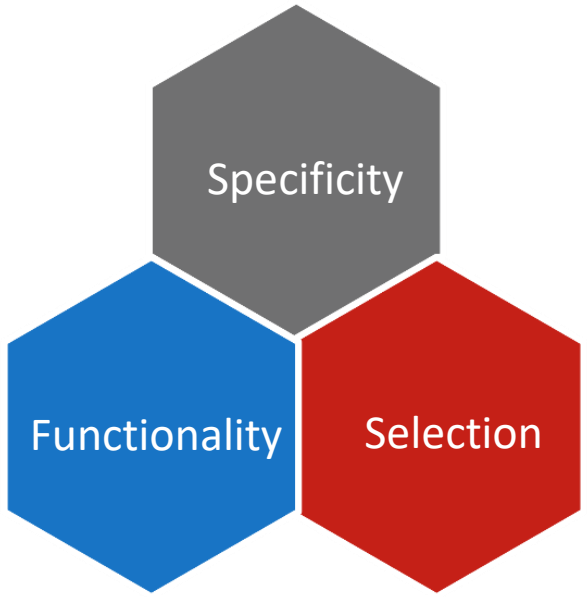
HPV Antigen selection example



***Affinity** is an important factor in selecting targets or TCR's, however, high affinity does not always correlate with best response*

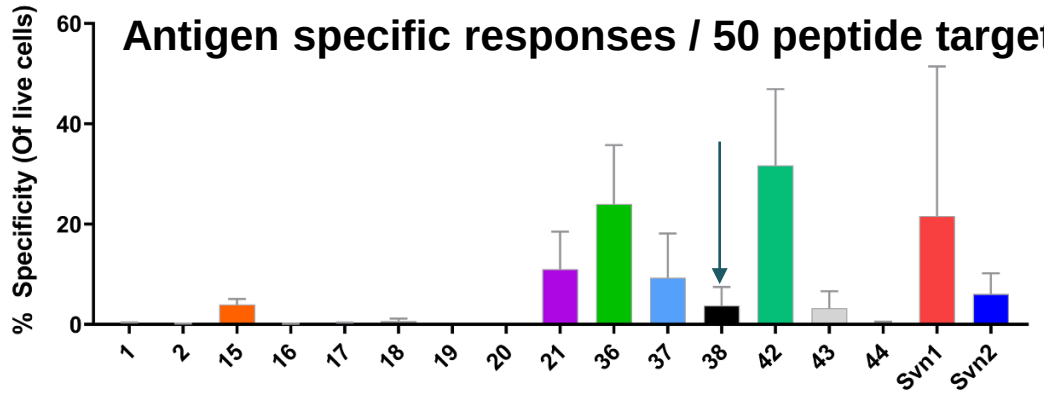
HPV Example: Rapid T-cell based screening and functional validation of targets

Screened 50 targets for HPV+ cancer(s) product mix selection



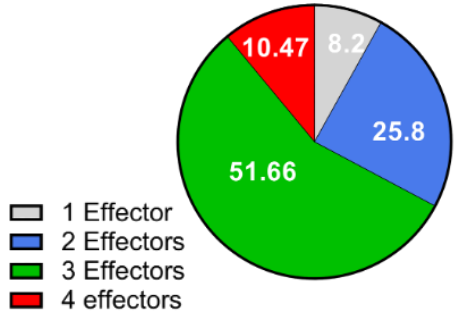
Selection of targets and Disease-specific Product Mix

Rapid Screening for Antigen-Specific T cell Response

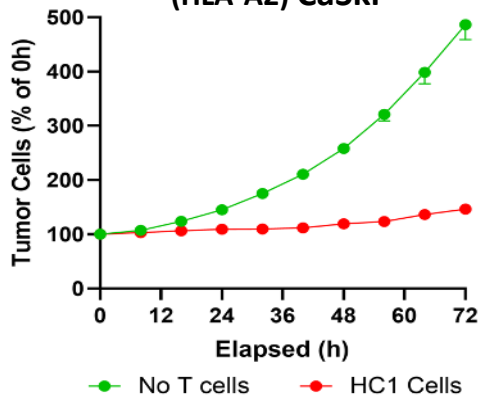


Functional Validation of T cells (e.g. ICS, killing)

76% of Specific T cells Expressed ≥3 Effector Functions



Potent Antigen-specific Killing (HLA-A2) CaSki



*Cells stimulated non-specific (ICS); A2 + Killing of tumor target / A2- not shown

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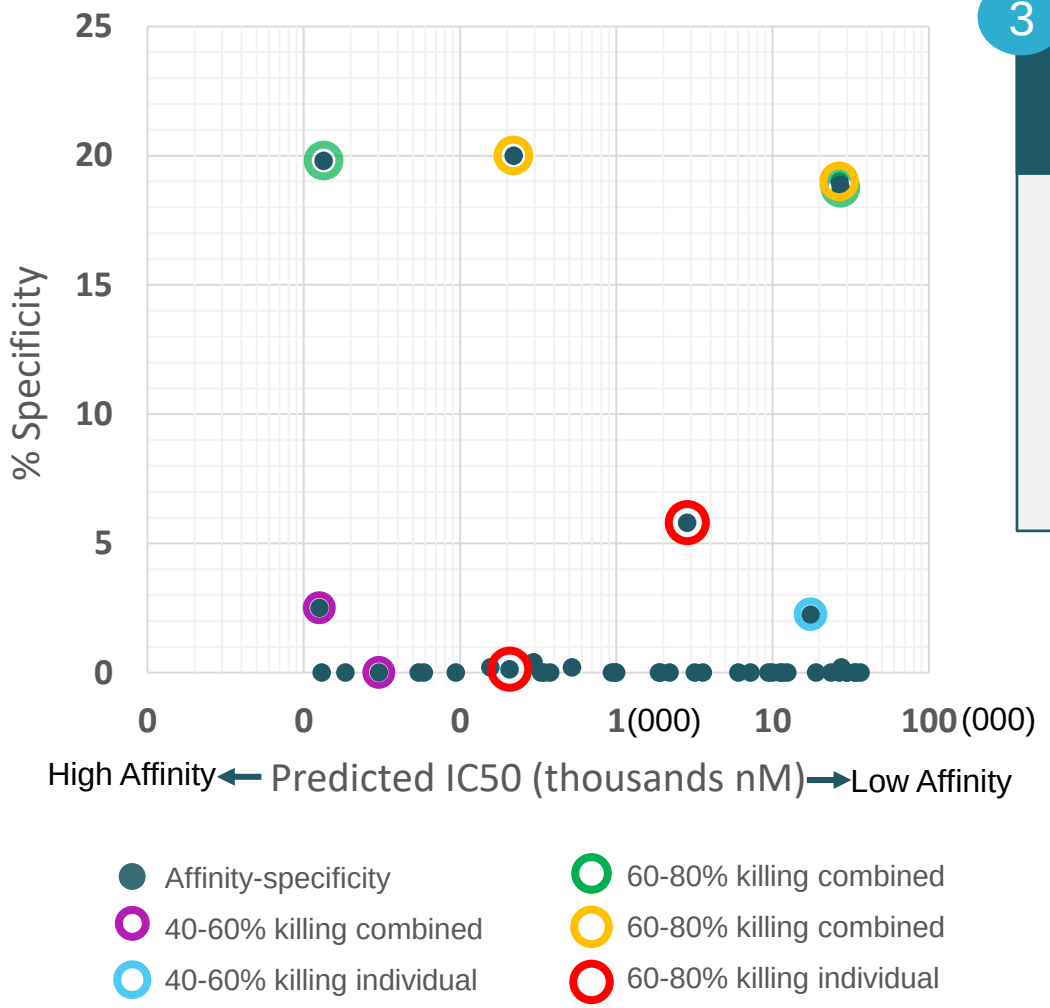
HPV Illustration: AIM enables multiplex evaluation of peptide/MHC and TCR adding a functional dimension to screening and prediction of antigen targets

1 Initial Peptide Screen

- KOL's and literature
- Reports of efficacy and safety
- Peptide scouting and discovery
- *In silico* investigation and triage

2 Prediction Evaluation

No single factor predicts function, no single peptide provides broadest response



3 Multiplex cell based Functional Evaluation

Targeting a broad range of TCR's:

- Specificity
- Range of affinities
- Functional (e.g. killing) capacity

EBV/MS Example: AIM based Functional Validation reveals functional defect in MS patients to EBV specific peptide(s)

Antigen-specific T-cell approach represent a disruptive clinical strategy over current MS treatments

National Institutes of Health
Turning Discovery Into Health

Collaboration with Steve Jacobson's Lab

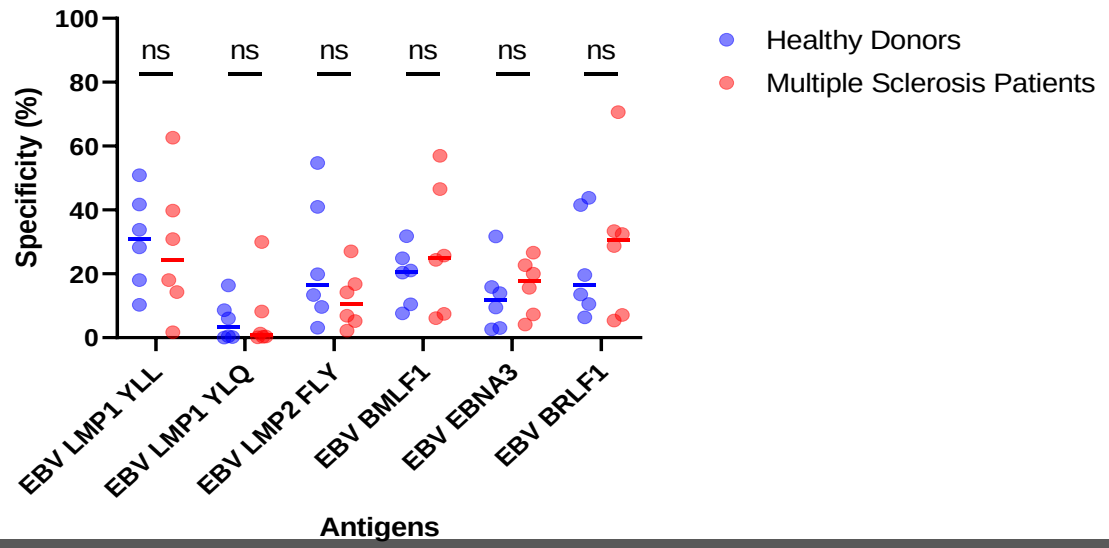
- Expanded EBV **specific T cells similar between HD and MS** PBMC
- However, **specific T cell defects against known EBV targets were identified in treated patients**

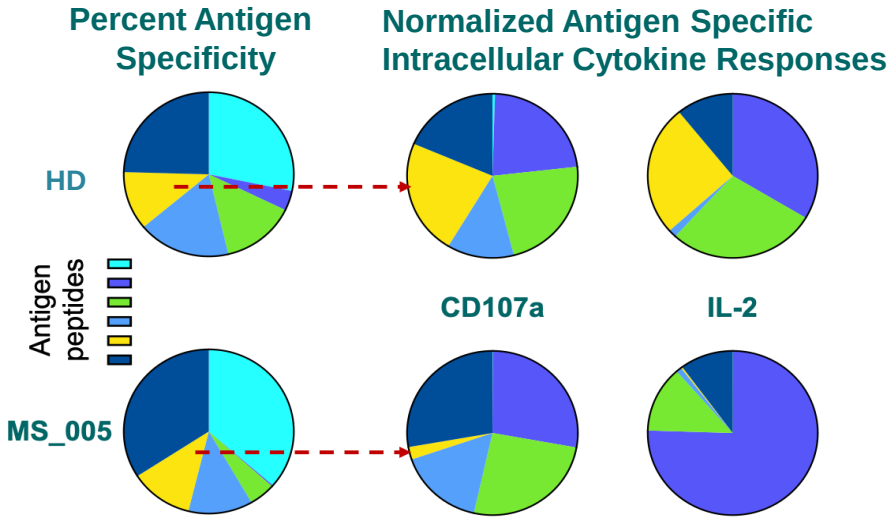
Yale

Collaboration with David Hafler's Lab

- Evaluate functional status of T cell responses to specific EBV targets in **MS patients at initial diagnosis** (blood and tissue)
- Inform strategies for multi-antigen therapies to improve outcomes

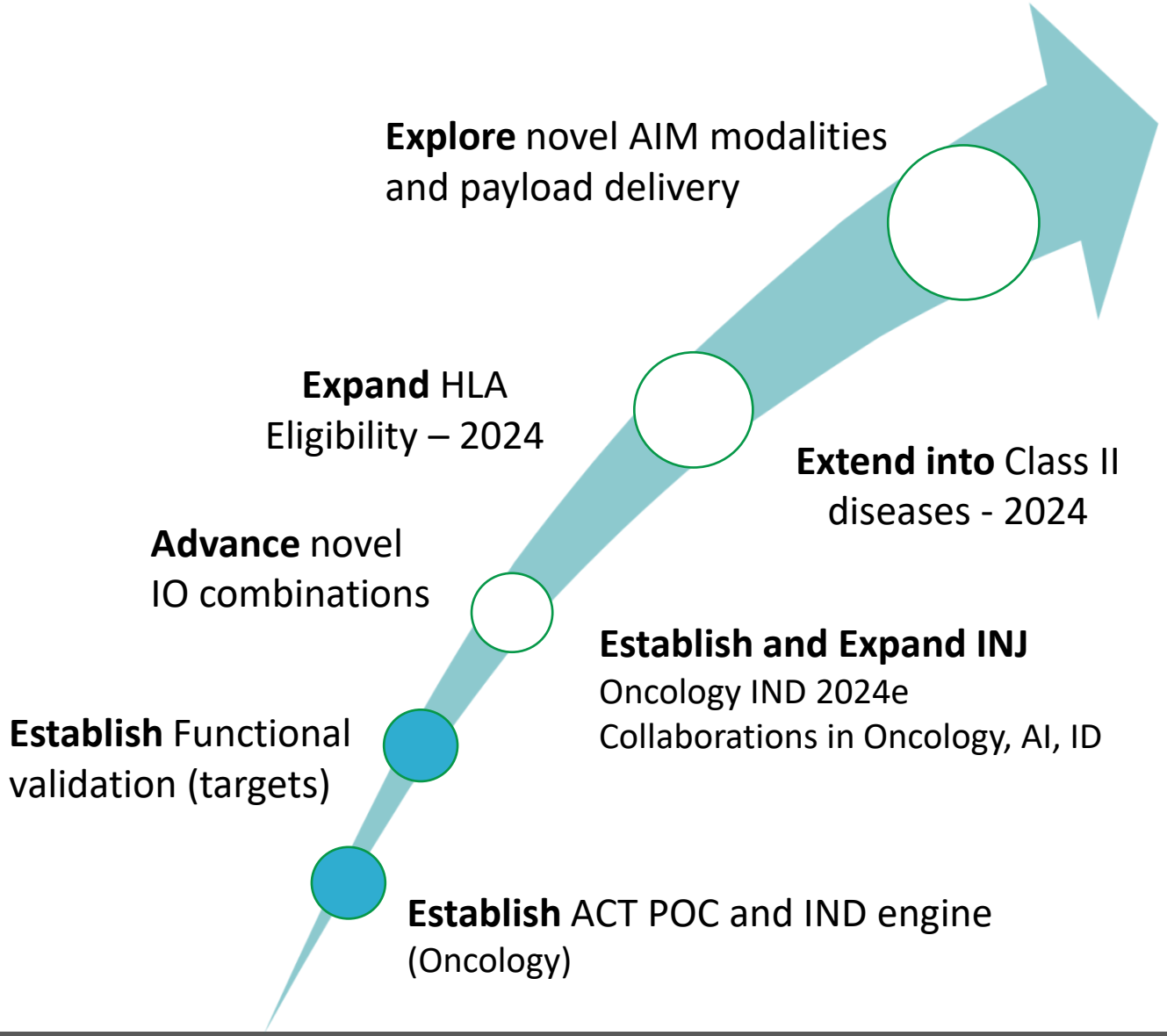
EBV antigen specificity from healthy donors & MS patients





Advance Transformative Paradigms with multi-antigen specific therapeutics

Collaborative target selection, product development and combinations



- **Novel multi-antigen specific products with potential to direct T cell function**
- **Synergistic IO/IO combinations**
- **Collaborative approach to advance new indications and combinations**
- **Functional validation dimension to enhance target prediction**
- **Platform-based IND engine**
- **Initial manufacturing platforms established**
- **Broad IP**

Potential to Transform Treatment Paradigms:
Designed to deliver durable antigen-specific T cell responses

- **Validated Oncology MOA: Early Clinical POC; consistent pre-clinical data across indications, ACT and INJ**
    - Address heterogeneity to increase response and persistence
    - "Fit" tumor specific T cells ideal for synergistic Immunotherapy combinations
- **Validated Autoimmune MOA: Eliminate or inhibit autoreactive, diseases-causing T cells (T1D*, pre-clinical)**
- **Reproducible data** across multiple collaborations and KOL's – demonstrate broad potential
- **Potential "IND Engine"** designed to rapidly generate new multi-antigen product INDs
- **Advancing T-cell based functional validation of targets designed to enhance target discovery and improve success**
- **Unlocking significant expansion opportunities with INJ and next generation approaches (e.g. Class II, bifunctional signaling)**

*AI pre-clinical data generated via external collaborators

NexImmune

Thank You

NASDAQ: NEXI