Precision Immunotherapy for Solid Tumors

Non-Confidential Corporate Presentation

March 2025

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This presentation also includes statistical and other industry and market data that we obtained from industry publications and research, surveys and studies conducted by third parties as well as our own estimates of potential market opportunities. All of the market data used in this presentation involves a number of assumptions and limitations, and you are cautioned not to give undue weight to such data. Industry publications and third party research, surveys and studies generally indicate that their information has been obtained from sources believed to be reliable, although they do not guarantee the accuracy or completeness of such information. Our estimates of the potential market opportunities for our product candidates include several key assumptions based on our industry knowledge, industry publications, third-party research and other surveys, which may be based on a small sample size and may fail to accurately reflect market opportunities. While we believe that our internal assumptions are reasonable, no independent source has verified such assumptions.

RIGHT TARGETS. RIGHT CELLS. RIGHT PLACE.

We target oncogenic driver mutations to deliver transformative therapies for patients with solid tumors

- Leader in Precision Immunotherapy - developing a deep pipeline of TCR-based therapies that have first-in-class / best-in-class potential
- Focus on targeting the most frequent oncogenic driver mutations in solid tumors; including KRAS and P53
- Proprietary platform technologies to build potent and persistent T cell therapies and generate bispecific T cell Engagers
- Science-driven team and founders focused on continued innovation to develop novel therapies with curative potential

Development Pipeline Milestones

AFNT-211
A11 KRAS G12V

- Lead KRAS targeting program
- Phase 1a data generation ongoing in 2L+ solid tumor indications
- Dose escalation proceeding on track across ~10 US sites with indication-specific expansions planned

Completion of Dose Escalation anticipated 2H25

AFNT-212
A11 KRAS G12D

- Doubles addressable KRAS population
- Introduces THRIVE non-viral gene-editing platform to enable future product development
- IND-enabling studies complete

IND clearance 1H 2025

AFNT-313
A2 P53 R175H

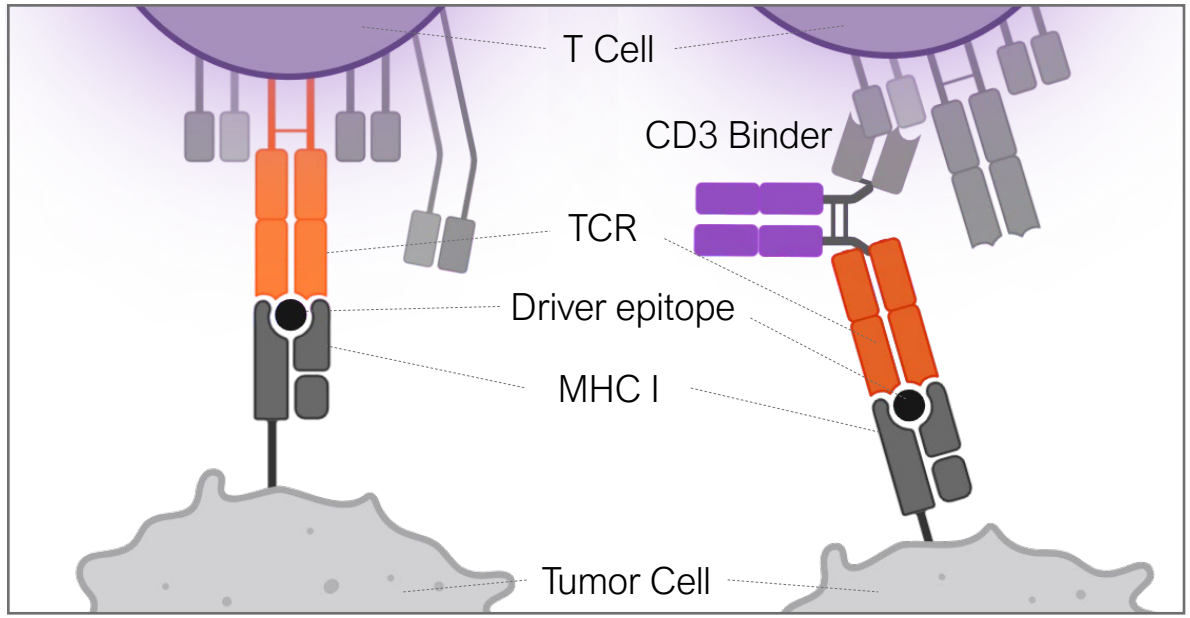
- Expands beyond KRAS to address largest P53 population
- Differentiated development candidate designed to integrate immunostimulatory signals for optimal T-cell activation

Pre-IND planning under way

Affini-T is Developing Two TCR-Based Therapeutic Modalities

TCR-T Cell Therapies

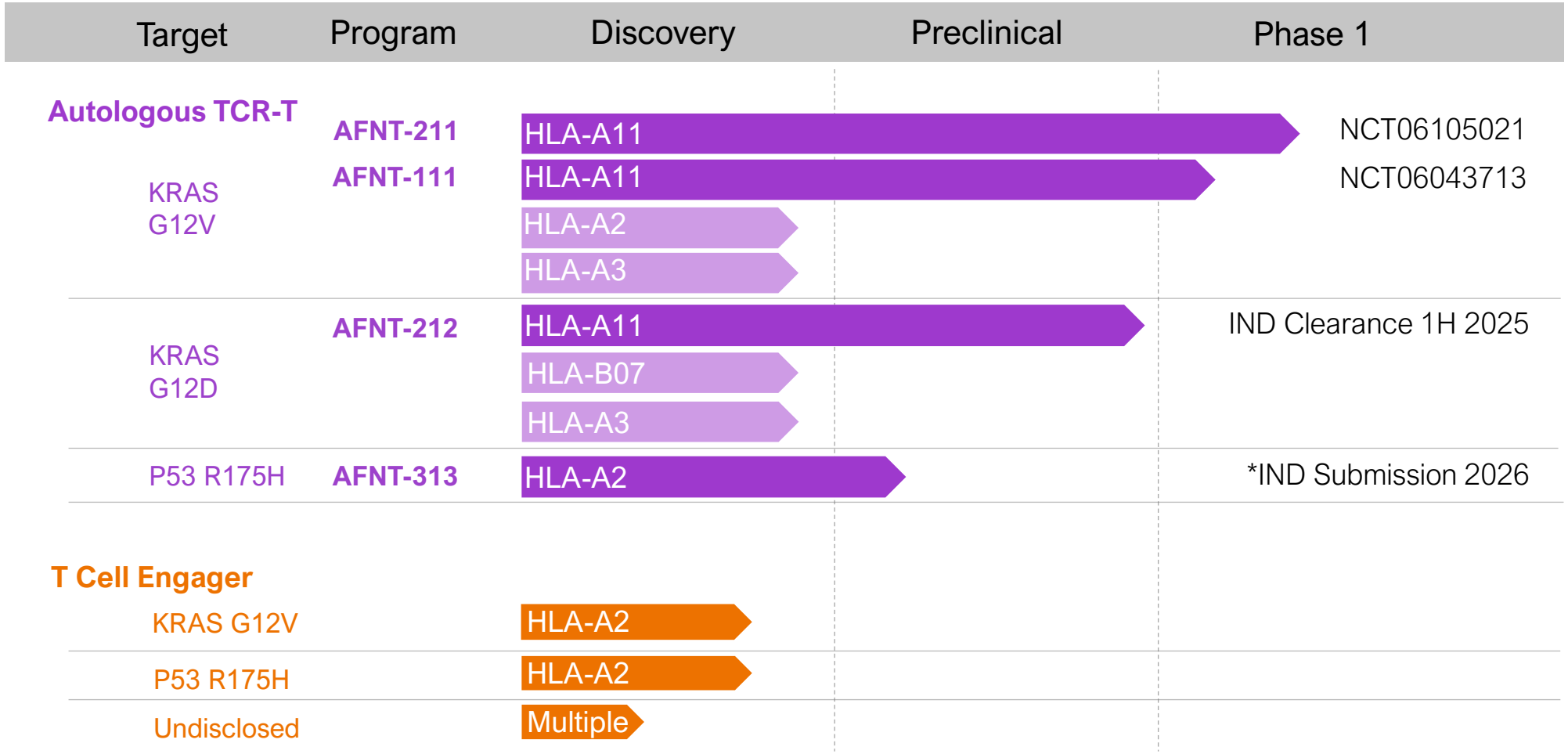
T cells engineered with a transgenic TCR that allows recognition of specific driver mutant epitopes



Bispecific T Cell Engagers

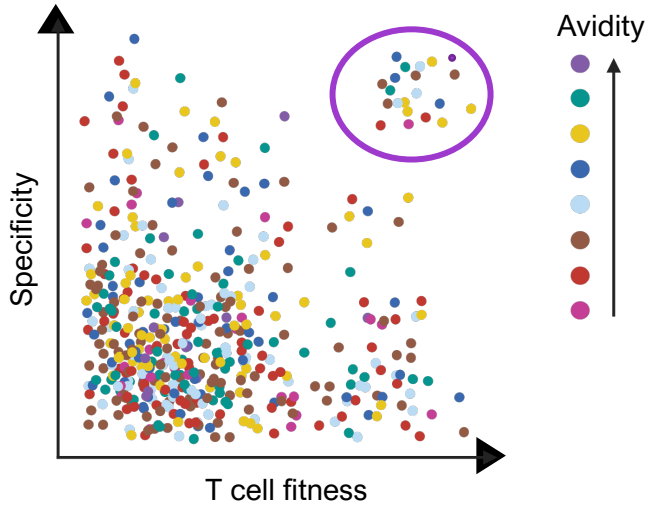
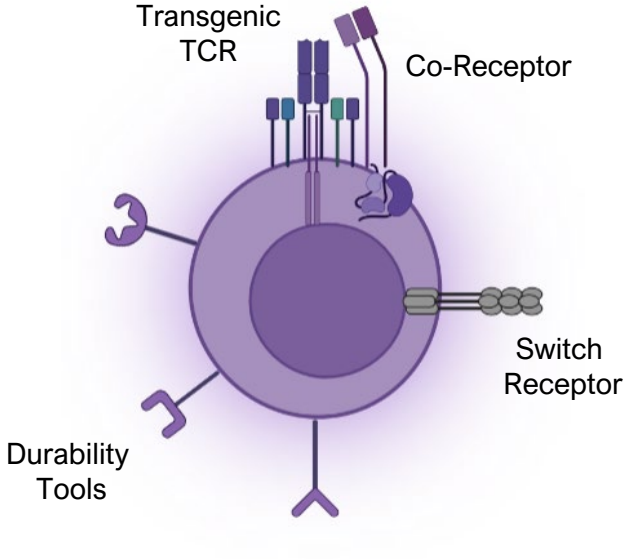
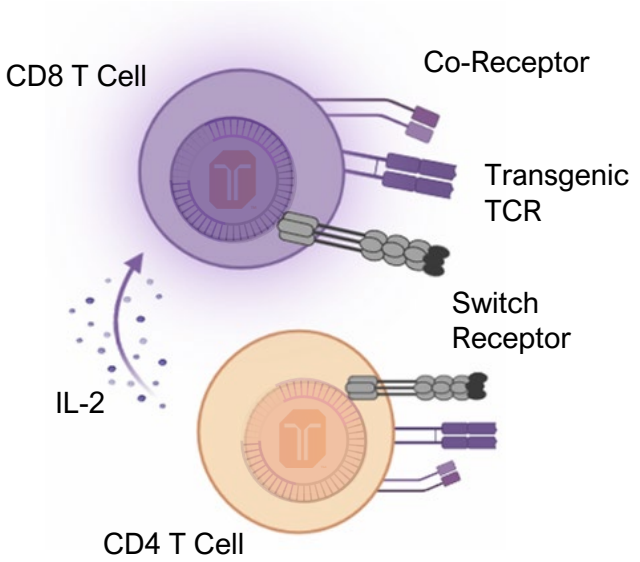
Bispecific biologics combining a TCR moiety to recognize the driver mutant epitope with a CD3 binding moiety to recruit endogenous T Cells

First-In-Class Potential for Multiple Products Targeting Oncogenic Drivers in Solid Tumors



*Planned submission date

Affini-T Platform Technologies Designed To Generate Potent & Tolerable TCR-T Cells

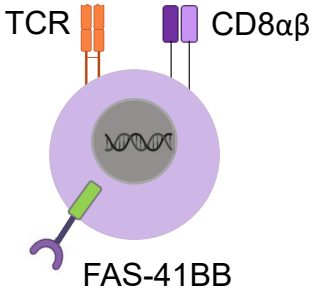
TAILOR™ TCR Discovery	TUNE™ Synthetic Biology	THRIVE™ Engineering and Manufacturing
		
Optimized system that integrates decades of learning with predictive algorithms to identify highly functional & specific TCRs against diverse targets	Program cell persistence in the TME to enhance durability with switch receptors (eg Fas-41BB), co-receptor and other armoring technologies	Scalable manufacturing with gene editing technologies to generate a robust engineered product with high stemness phenotype

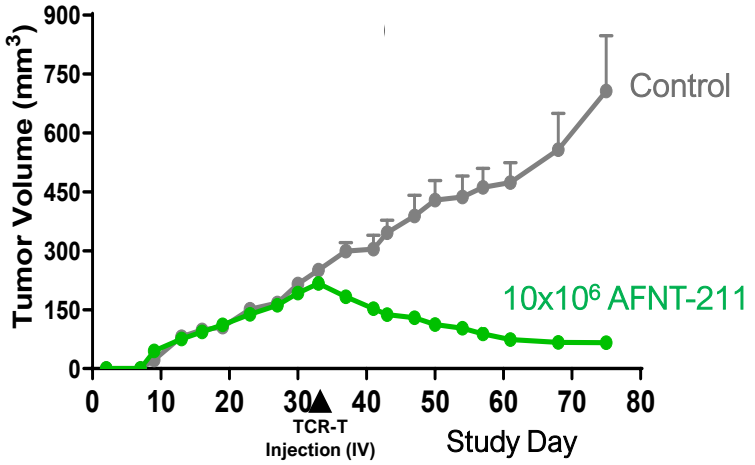
TME: Tumor Microenvironment

Innovative pipeline leverages TAILOR™, TUNE™ & THRIVE™, designed to eradicate difficult-to-treat solid tumors

Enhanced survival in TME

AFNT-211
Integrates
Signal 1+2

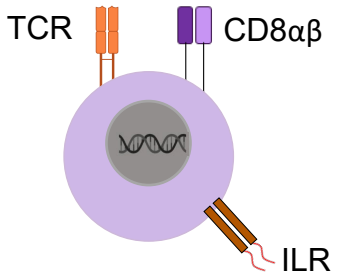


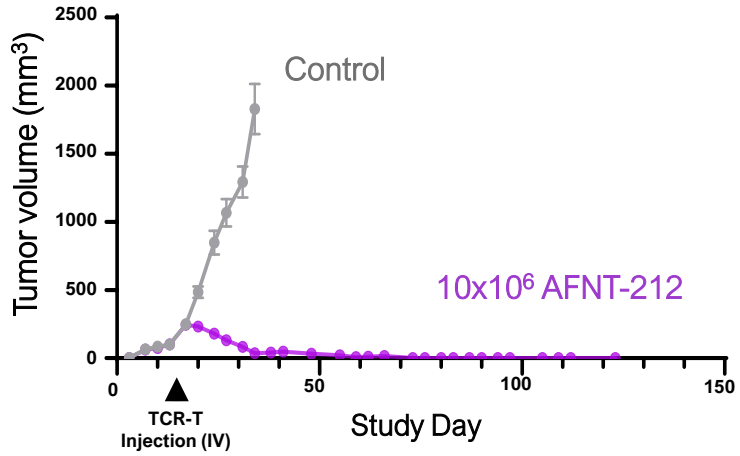


AFNT-211 induced preclinical tumor regression in established Breast (SW527) tumors with mut. KRAS G12V

Enhanced proliferation in periphery

AFNT-212
Integrates
Signal 1+3

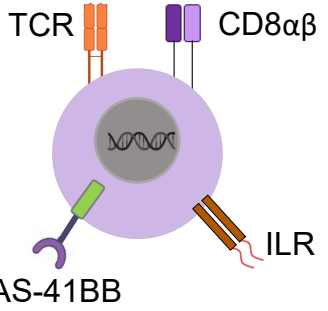


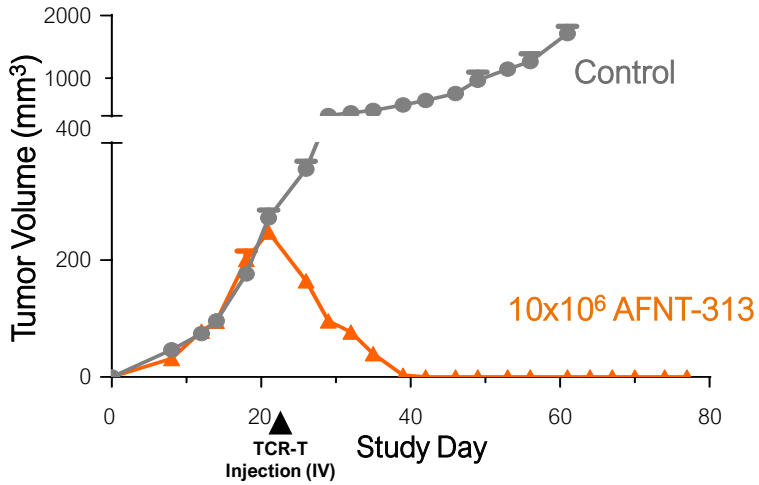


AFNT-212 induced preclinical tumor eradication in established Colorectal (CL40) tumors with mut. KRAS G12D

Support in TME & Periphery

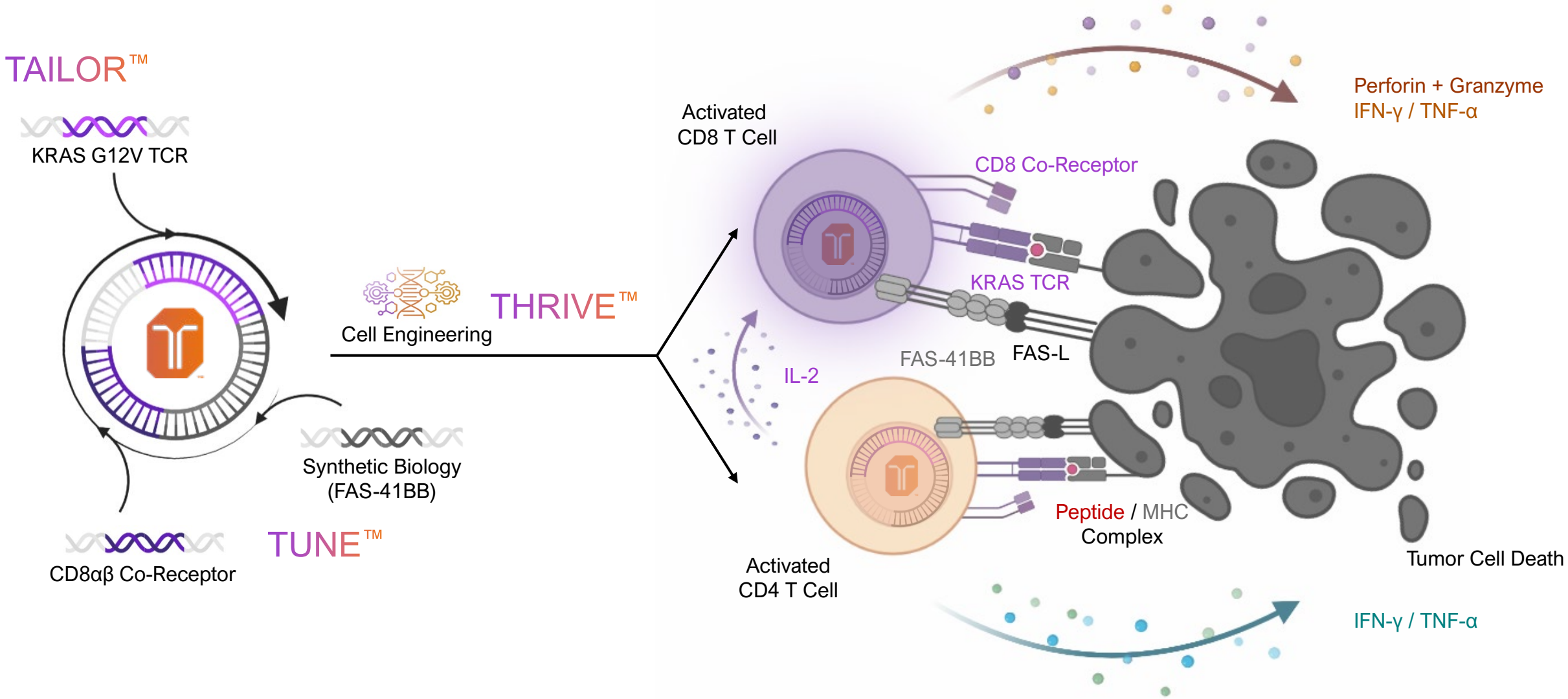
AFNT-313
Integrates
Signal 1+2+3





AFNT-313 induced preclinical tumor eradication in established Ovarian (TYK-nu) tumors with mut. p53 R175H

AFNT-211: A11 KRAS G12V TCR Engineered T Cells + FAS-41BB Durability Switch Receptor



AFNT-211: Patient Journey

Pre-screen

Leukapheresis

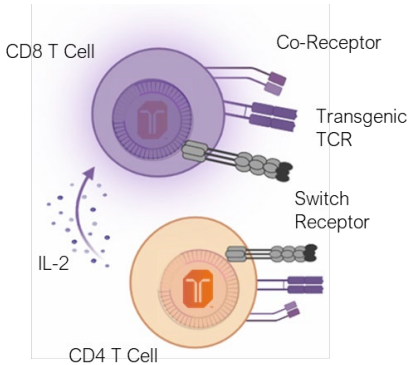
Lymphodepletion

AFNT-211 product infusion

Transition to long term follow up

Patient Monitoring

THRIVE™ Manufacturing



elevatebio

~10-day manufacturing
~6-day QC testing

Flu / Cy**

KRAS G12V
HLA-A*11:01

Targeting ~22-day vein-to-vein time

**Lymphodepleting chemotherapy (LDC) with cyclophosphamide 500mg/m2/day and fludarabine 30mg/m2/day intravenously (I.V.) on Days -6 to -3, (4 days),

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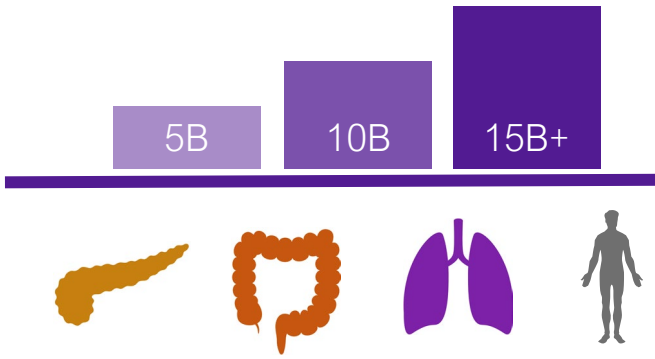
AFNT-211: Clinical Development Plan

Phase 1a Basket Trial Dose Finding | Phase 1b/2 Expansion Cohorts | Registration Study

KRAS G12V-mutated tumors
& HLA-A*11:01 allele
2nd+ Line



Sample size N=15-20
~10 US clinical trial sites

- PDAC → 2nd/3rd line
- CRC → 2nd/3rd line
- NSCLC → 2nd/3rd line
- Tissue-agnostic → 2nd/3rd line

Sample size up to N=20 per indication

Registrational trial design based on data and FDA interaction (e.g. single-arm)

Target N=~80 for potential indication
Expand to 35-40 sites US/EU5/CAN

Optimal Biological Dose / Proof of Clinical Concept

Interim Analysis

Aim for approval on ORR & DoR

**Excluding primary brain tumors*

ORR – Overall Response Rate
DoR – Duration of Response

AFNT-211: Patient Selection & Biomarker Strategy

I. Patient Selection

- **KRAS G12V** mutation – routinely reported by PCR, NGS, and CGP; by tumor or liquid biopsy (ctDNA)
- **HLA A*11:01** – via standard typing assays (Histogenetics – ASHI accredited) or CGP
- **2L+,** Upside: frontline consolidation

II. Monitoring - Peripheral Blood

- **PK:** TCR-T expansion (VCN and/or CK), C_{max}, T_{last}, AUC
- **PD:** TBNK depletion and reconstitution; cytokines, e.g. IL7, IL15, IFNγ
- **MRD:** ctDNA
- **TCR-T phenotyping:** TCR-T cell differentiation, activation, and exhaustion
- **Safety:** Replication-competent lentivirus, insertion site analysis

III. Phenotyping - Tumor

- **RECIST:** Imaging response assessment
- **TME:** AFNT-211 TCR-T cell infiltration and phenotyping, Host immune infiltration (including CD4 and CD8)
- **Tumor characterization:** TMB, MSI, PD1, FasL, IFNγ and APM

ctDNA – circulating tumor DNA; NGS – next generation sequencing; ASHI – American Society for Histocompatibility and Immunogenetics; LOH – loss of heterozygosity; CGP – comprehensive genomic profiling, PK – pharmacokinetics; PD – pharmacodynamics; VCN – vectory copy number

CK – cellular kinetics; TBNK – T, B, and natural killer cells; MRD – minimal residual disease; TME – tumor microenvironment; TMB – tumor mutational burden; MSI – microsatellite instability; APM – antigen presenting machinery

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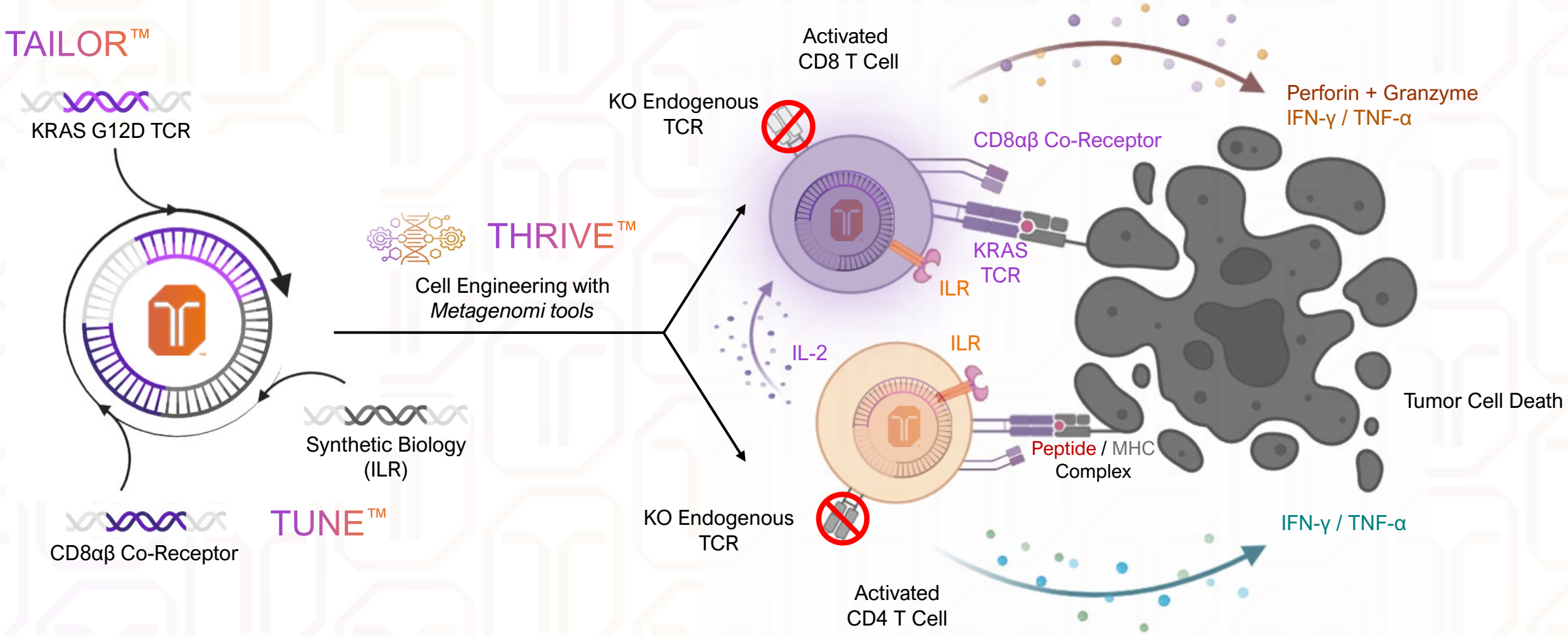
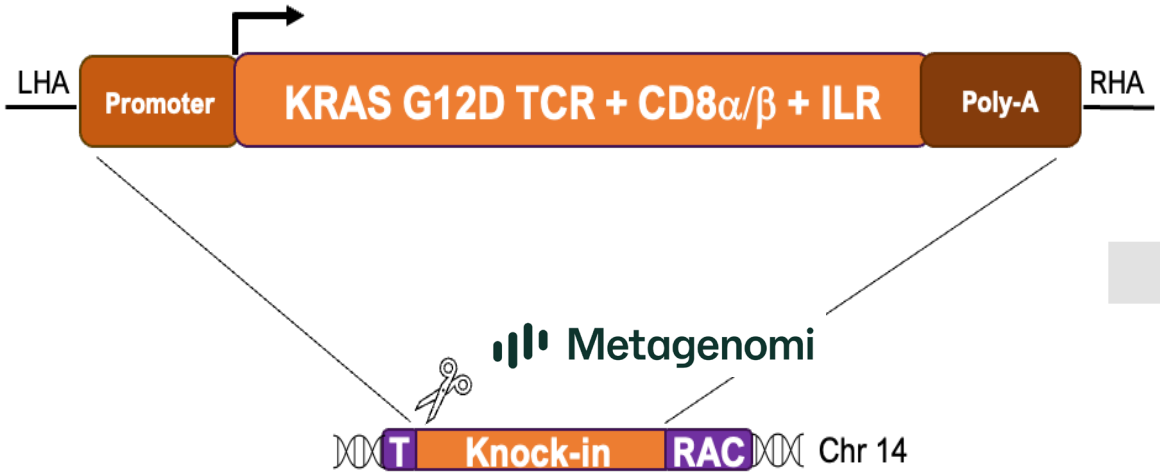
AFNT-212: A11 KRAS G12D TCR Engineered T Cells + Durability Switch Receptor + Gene Editing



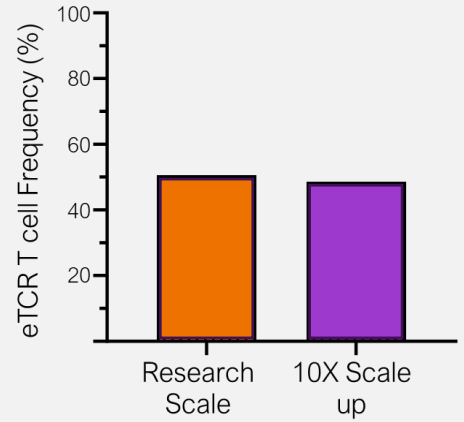
THRIVE™ High Efficiency Non-viral Delivery of Large Transgenes at cGMP Scale



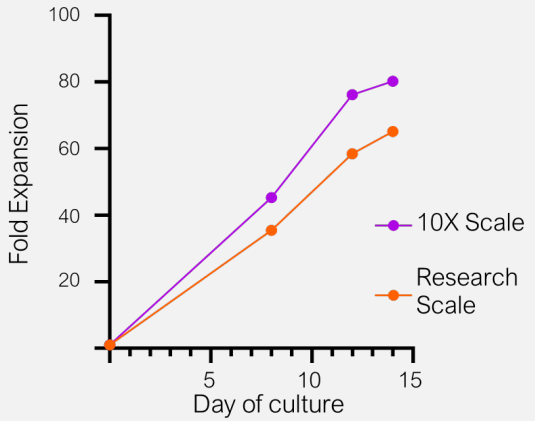
Transgenes inserted within the endogenous TRAC gene via CRISPR/Cas driven homology mediated repair

TRAC-inserted knock-in of 6.3kb 5 gene cassette

Transgene Integration Frequency



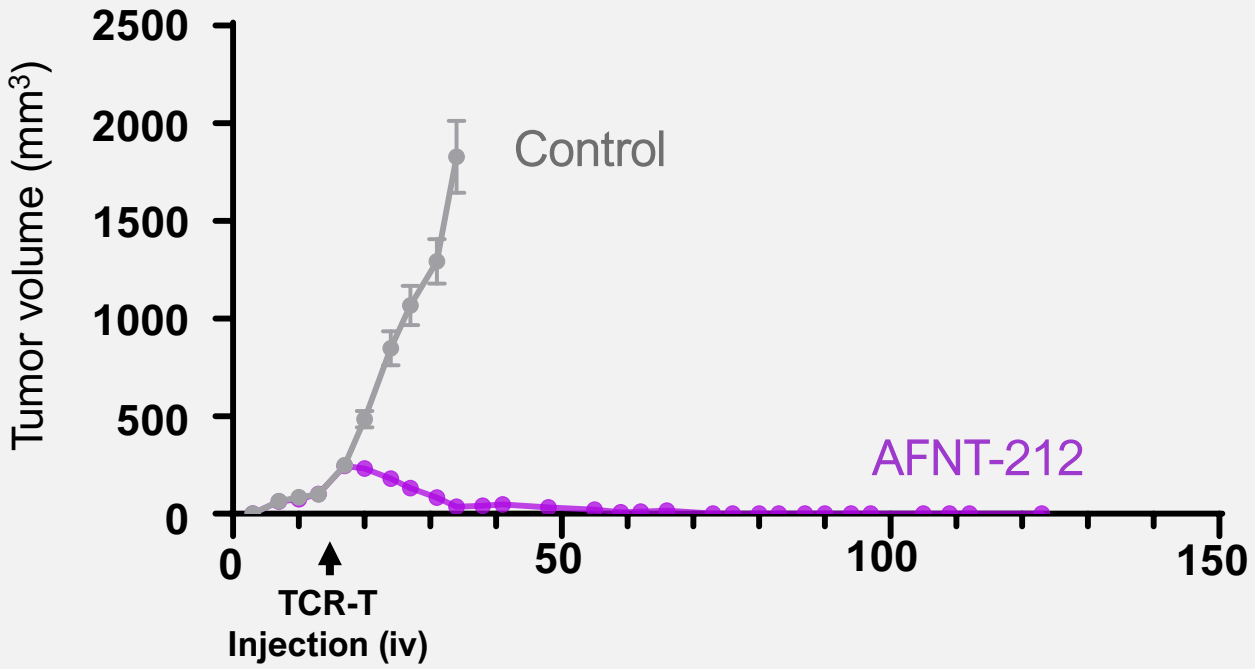
Expansion Kinetics



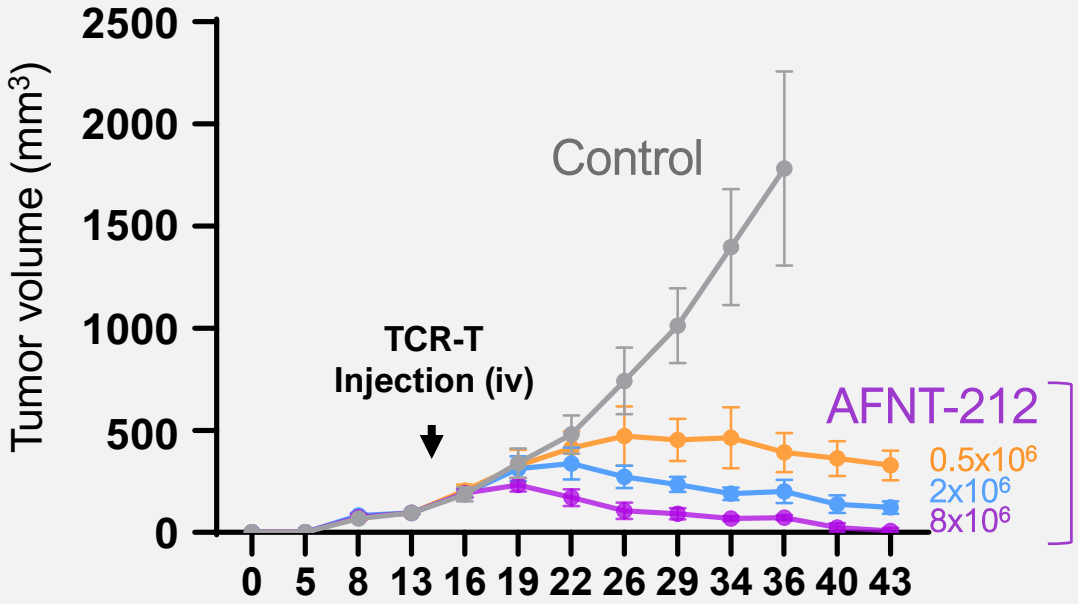
KI = Knock-In

AFNT-212 Showed Robust Anti-tumor Activity in Established Tumor Mouse Models *in vivo*

AFNT-212 T cells induced complete remission for over 120 days

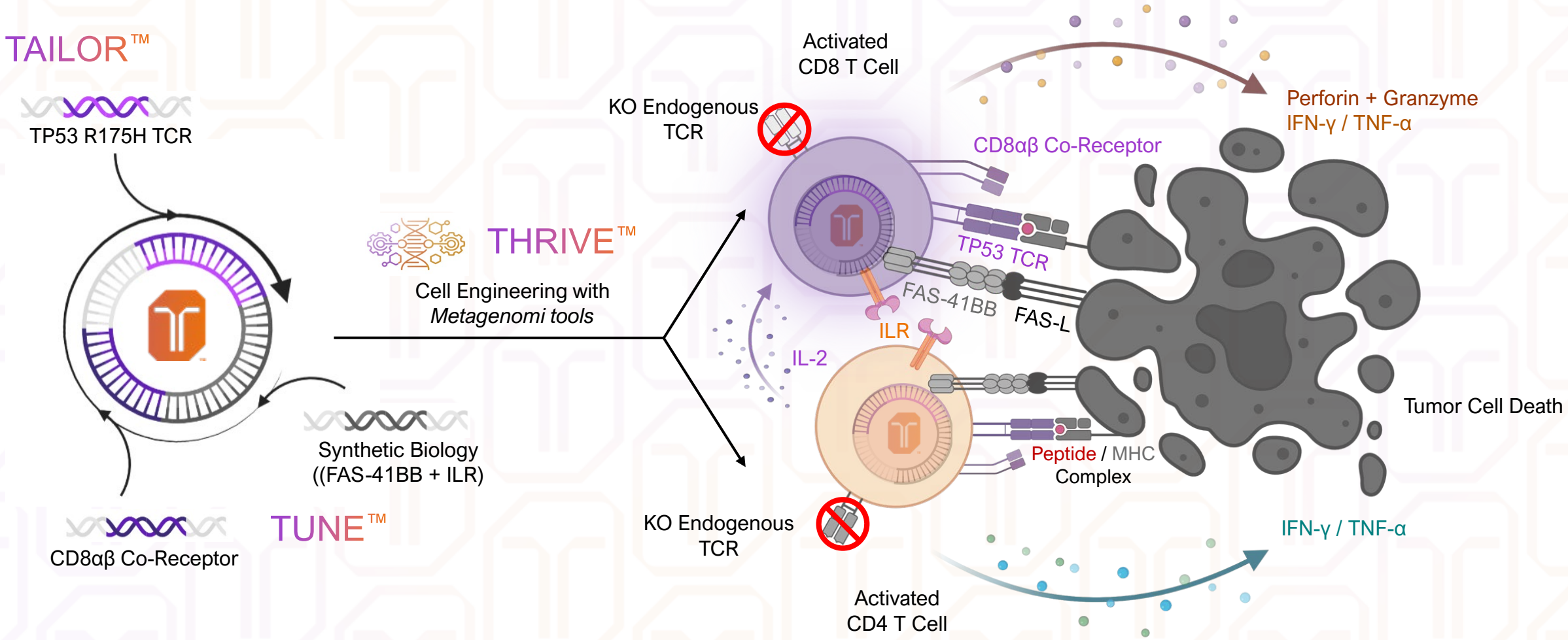


AFNT-212 T cells drove a dose-dependent anti-tumor response



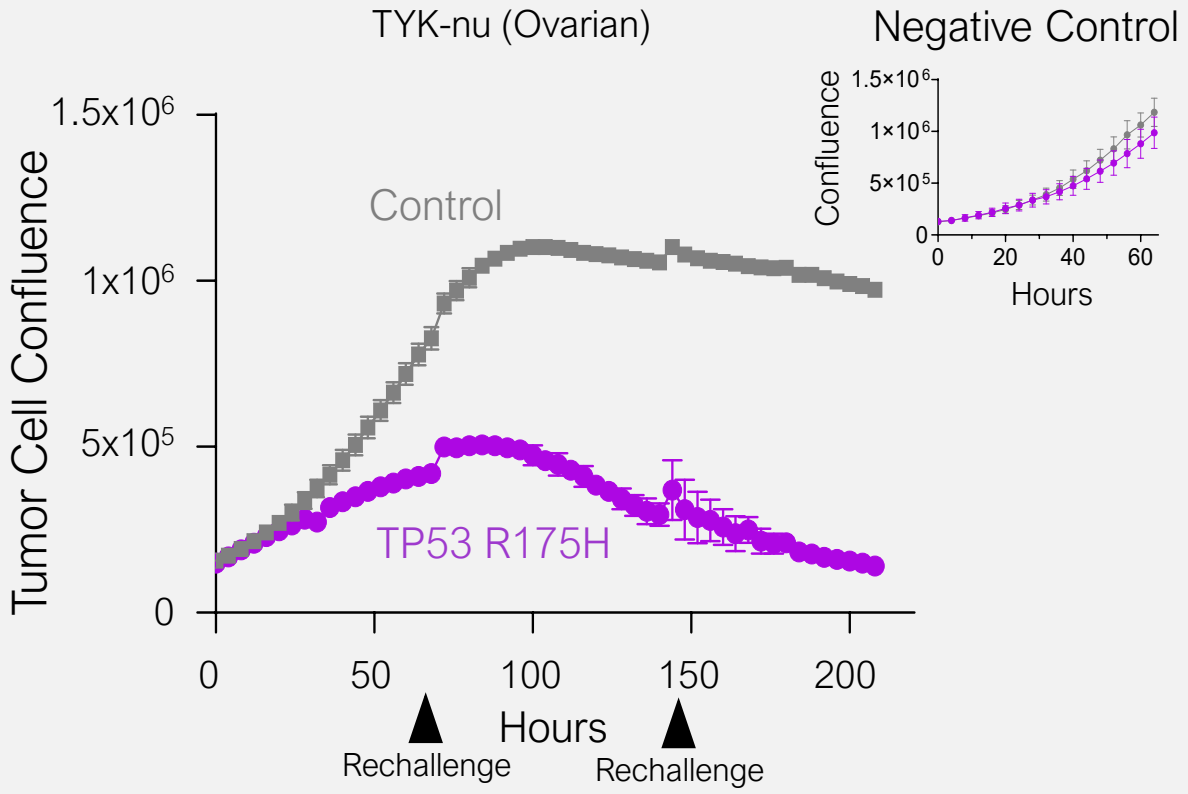
(CL-40 colorectal model)

AFNT-313: A2 TP53 R175H TCR Engineered T Cells + 2 Durability Switch Receptors + Gene Editing



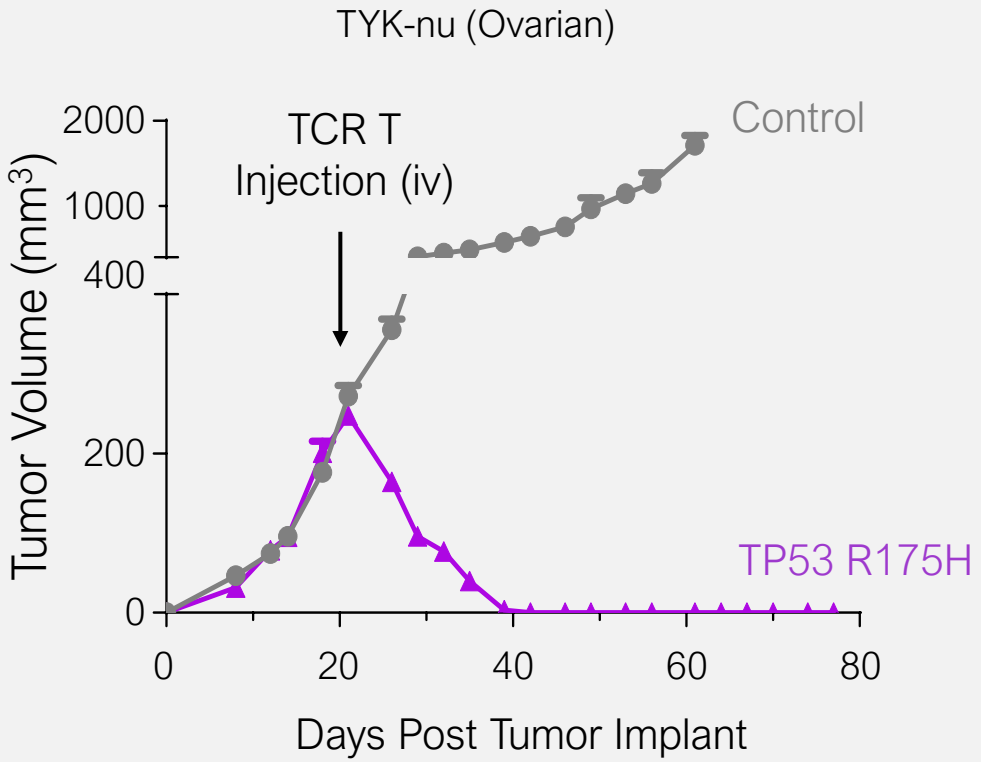
AFNT-313 TCR-T Showed Robust Preclinical Tumor Cell Control *In Vitro* and *In Vivo*

In Vitro



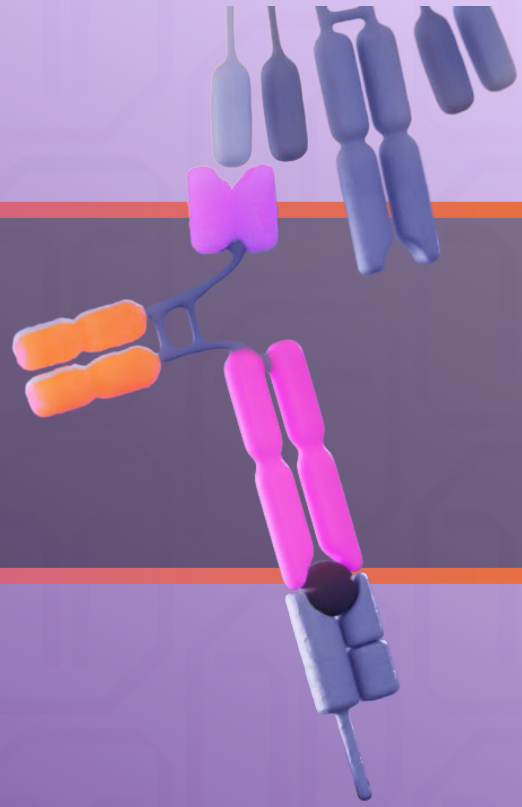
TP53 R175H TCR-T cells controlled tumor proliferation even following multiple re-challenges

In Vivo



TP53 R175H TCR-T drove complete responses even against large established tumors

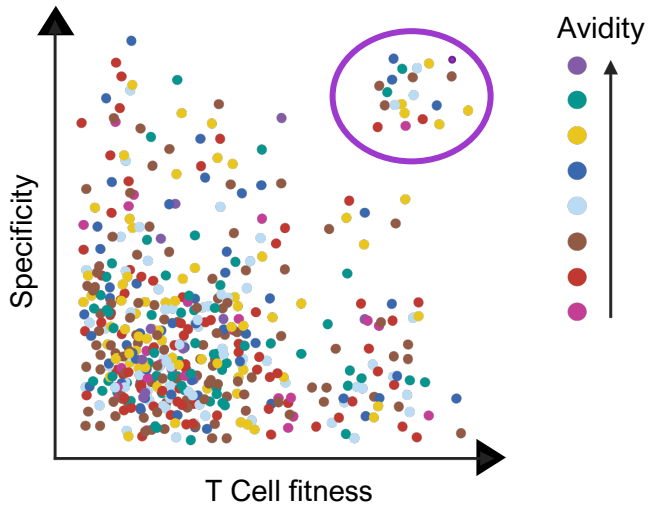
TETHER™ T Cell Engagers Highlights



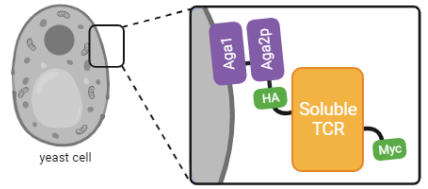
Affini-T Platform Technologies Designed to Generate Highly Specific & Active T Cell Engagers

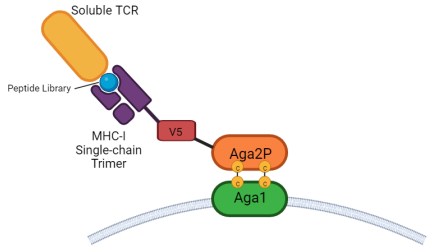
1 TAILOR™ TCR Discovery

- High throughput screening, predictive algorithms, and decades of learning
- Generate highly functional and tolerable TCRs against diverse targets



2 Affinity Maturation



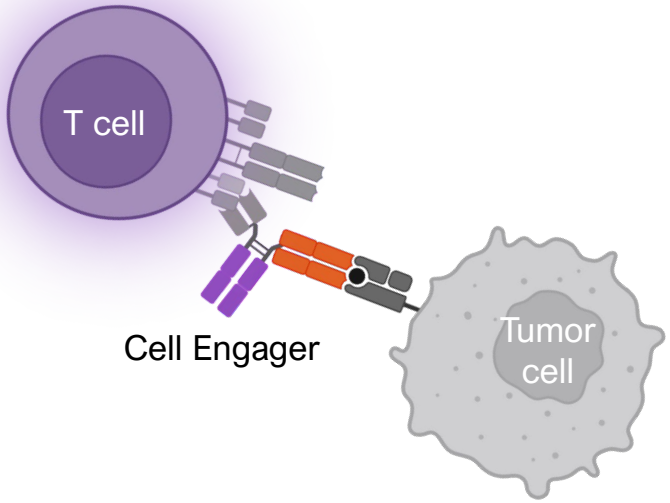


Yeast Display Modalities

- Libraries to identify high affinity TCRs
- Libraries for specificity screenings

3 TETHER™ T Cell Engagers

- Affinity matured TAILOR™ TCRs with high specificity and affinity
- Balanced CD3 binders for optimal T cell engagement
- Bispecific T cell engager format with long half-life



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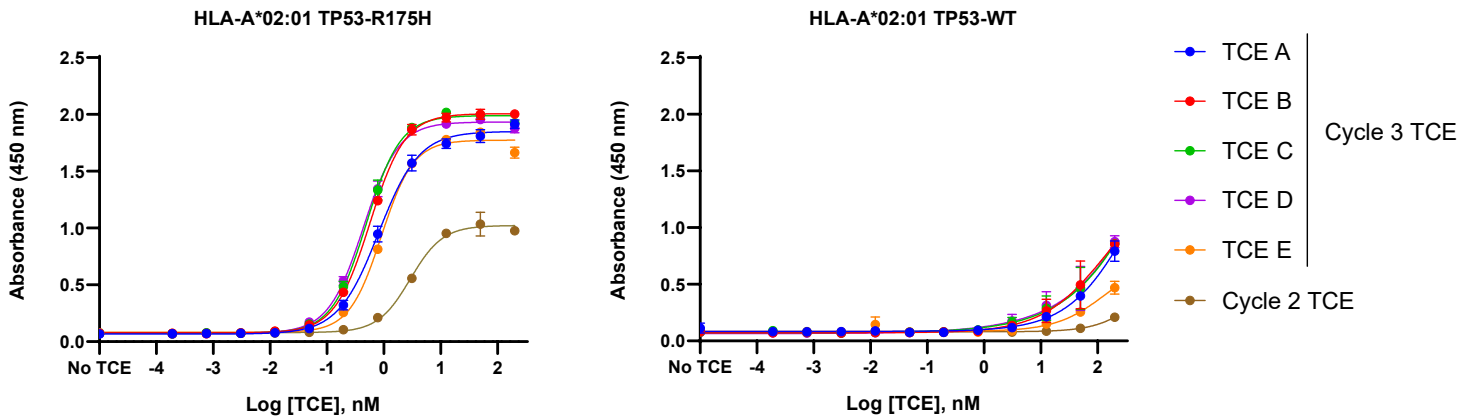
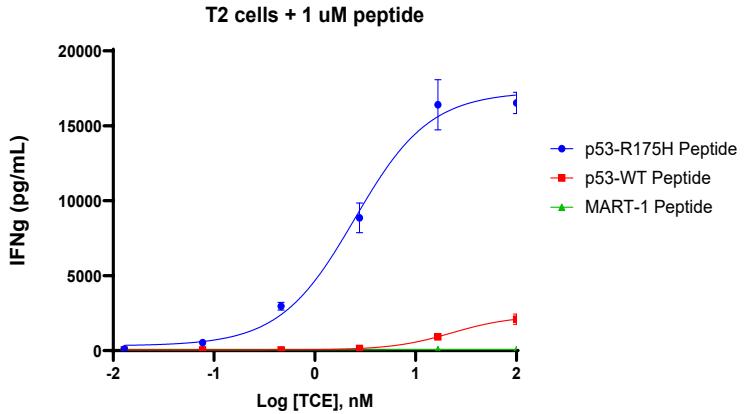
A2 TP53 R175H **TETHER** TCEs Showed Robust & Specific Tumor Killing & T Cell Activation *In Vitro*

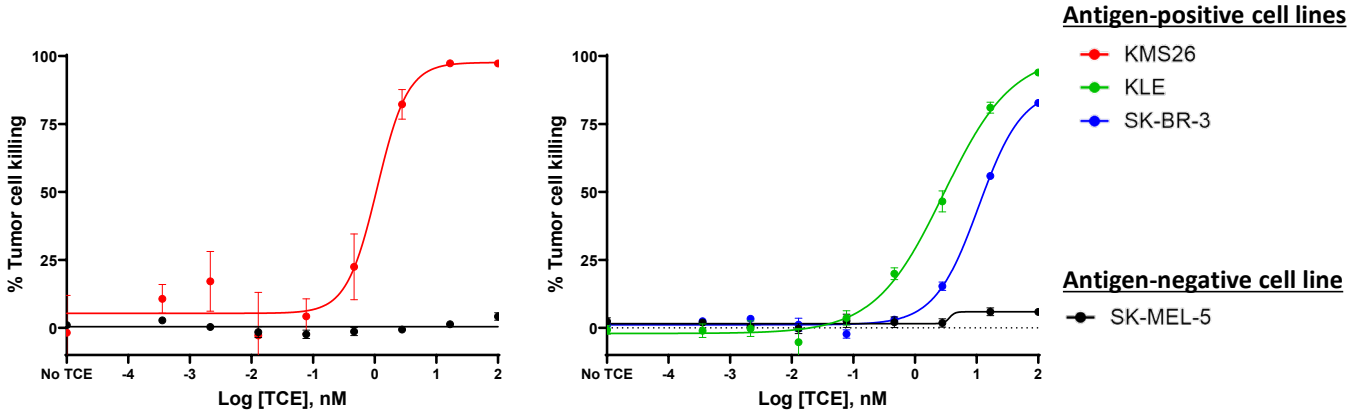
Specific binding



Robust activation

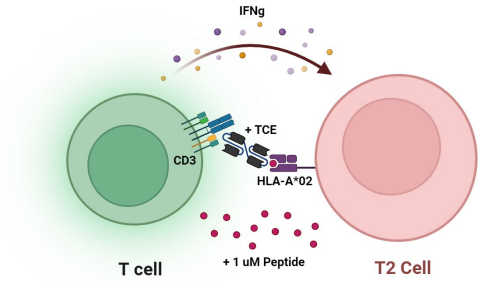


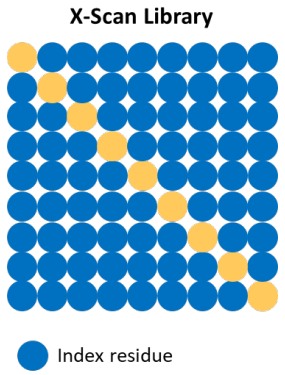
Potent and specific tumor cell killing

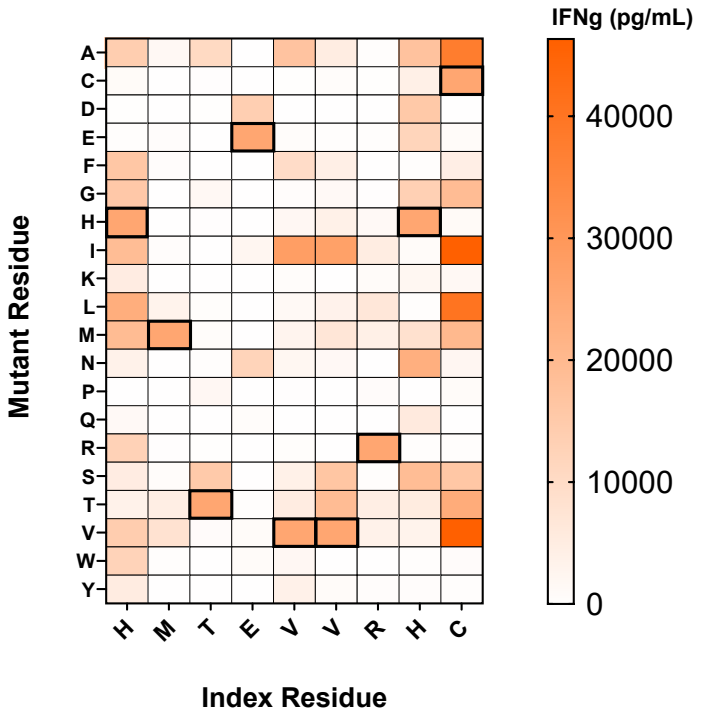


Preliminary Data for A2 TP53 R175H TCE Suggested Favorable Tolerability & Specificity Profiles

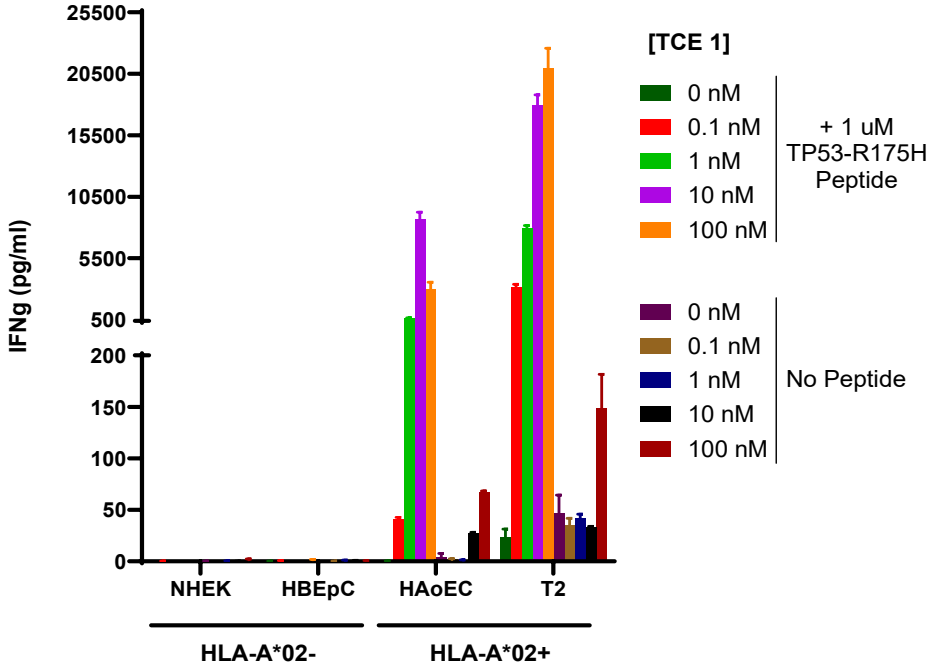
X-scan off-target binding profile







Did not mediate activation toward select normal tissues



Normal Tissue	HLA-A Alleles
Normal Human Epidermal Keratinocytes (NHEK)	A*11:01
Human Bronchial Epithelial Cells (HBEpC)	A*11:01
Human Aortic Endothelial Cells (HAoEC)	A*02:01/A*11:01
T2 Cells (Positive Control)	A*02:01

Experienced Management Team Supported by Blue-Chip Investor Syndicate

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Chief Technical Officer

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Memorial Sloan Kettering Cancer Center

elevatebio

Metagenomi

ADIMAB

Upcoming Catalysts

AFNT-211
A11 KRAS G12V

- Phase 1a data generation ongoing in 2L+ solid tumor indications
- Completion of dose escalation anticipated 2H25

AFNT-212
A11 KRAS G12D

- IND enabling studies complete
- IND clearance 1H 2025
- THRIVE™ non-viral gene-edited FiH

AFNT-313
A2 P53 R175H

- IND enabling studies underway
- Pre-IND feedback anticipated 2025
- 2026 IND

Precision Immunotherapy targeting oncogenic driver mutations to develop potentially curative therapies for patients with solid tumors

* All future catalysts and milestones planned but not guaranteed

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