Precision Immunotherapy for Oncogenic Driver Mutations

Non-Confidential Corporate Presentation

September 2024

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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.

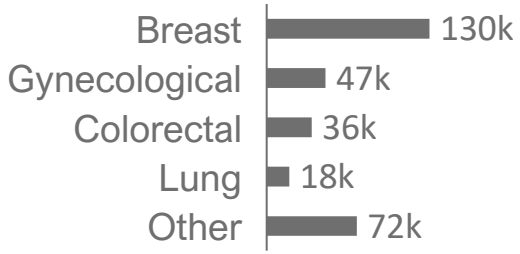
RIGHT TARGETS. RIGHT CELLS. RIGHT PLACE.

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

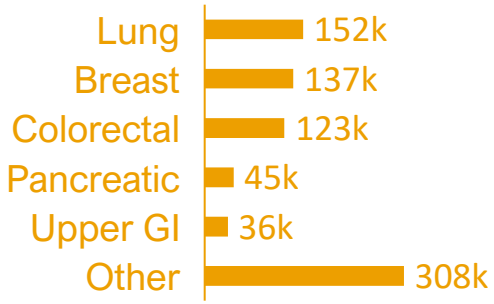
- Leader in Precision Immunotherapy - developing a deep pipeline of TCR-based therapies that have first-in-class / best-in-class potential
- Focus on targeting the most frequent oncogenic driver mutations in solid tumors; including KRAS, NRAS, P53, and PIK3CA
- Proprietary platform technologies to build 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

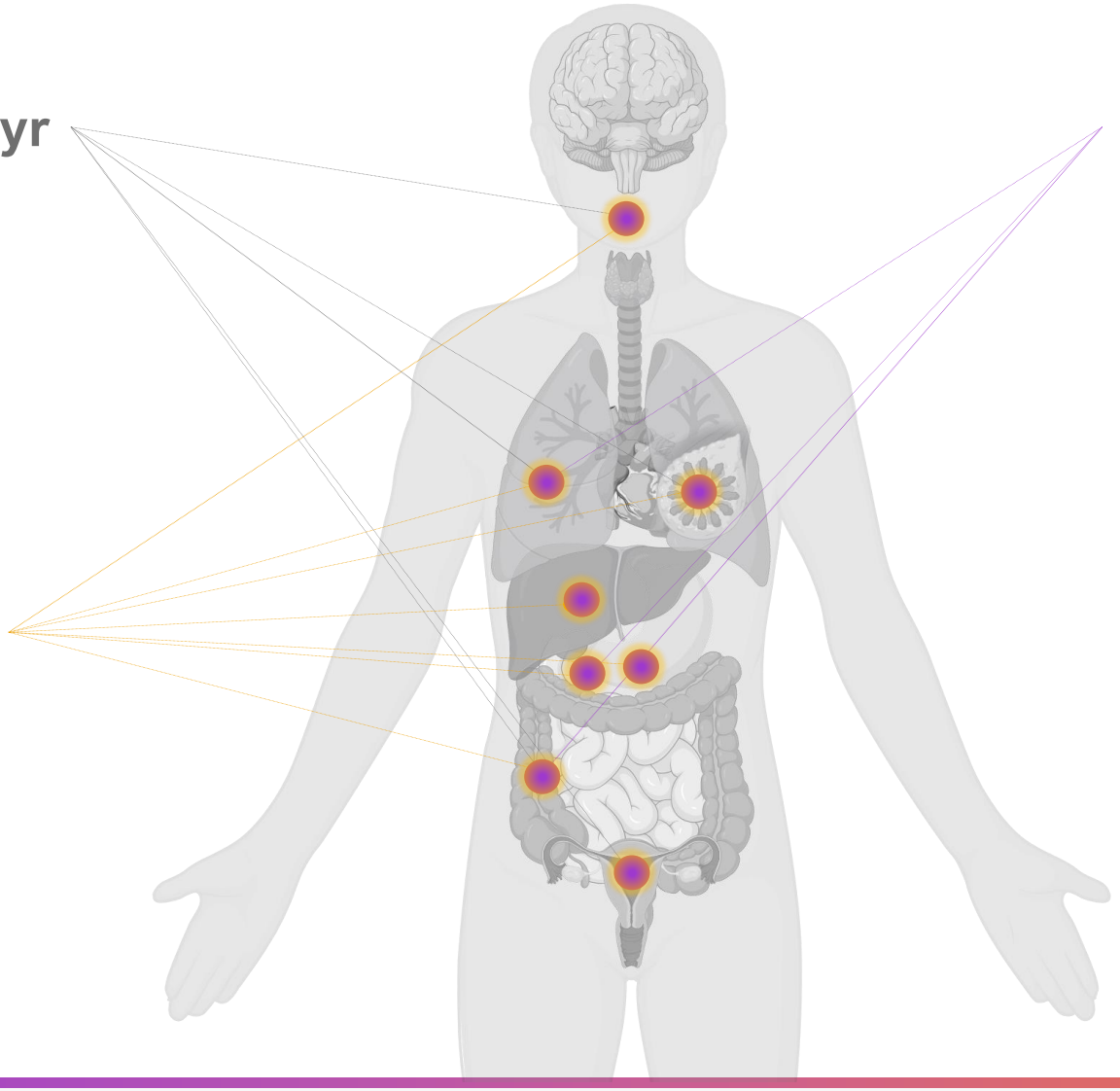
Driver Mutations are Ubiquitous but Underutilized Targets for Treating Solid Tumors

PIK3CA 303k Patients/yr

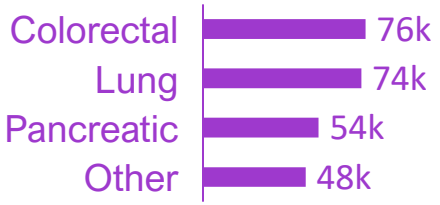


TP53 801k Patients/yr





KRAS 253k Patients/yr



Affini-T targets the most prevalent oncogenic drivers across solid tumors

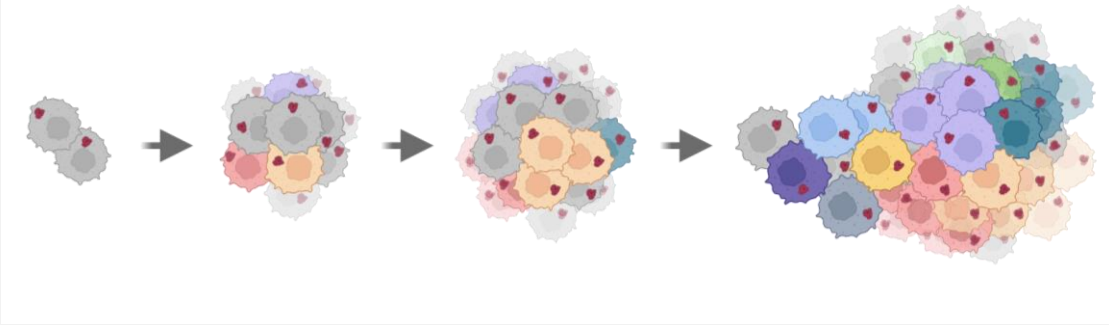
2030 US Incidence. Source: WHO IARC GCO / AACR Genie

Targeting Oncogenic Driver Mutations Like KRAS Strikes at the Core of Tumor Biology

Cancer cells are *dependent* on oncogenic drivers for survival and proliferation

Oncogenic drivers are ubiquitously expressed in heterogeneous tumors

KRAS mutations are present in up to 30% of solid tumor malignancies



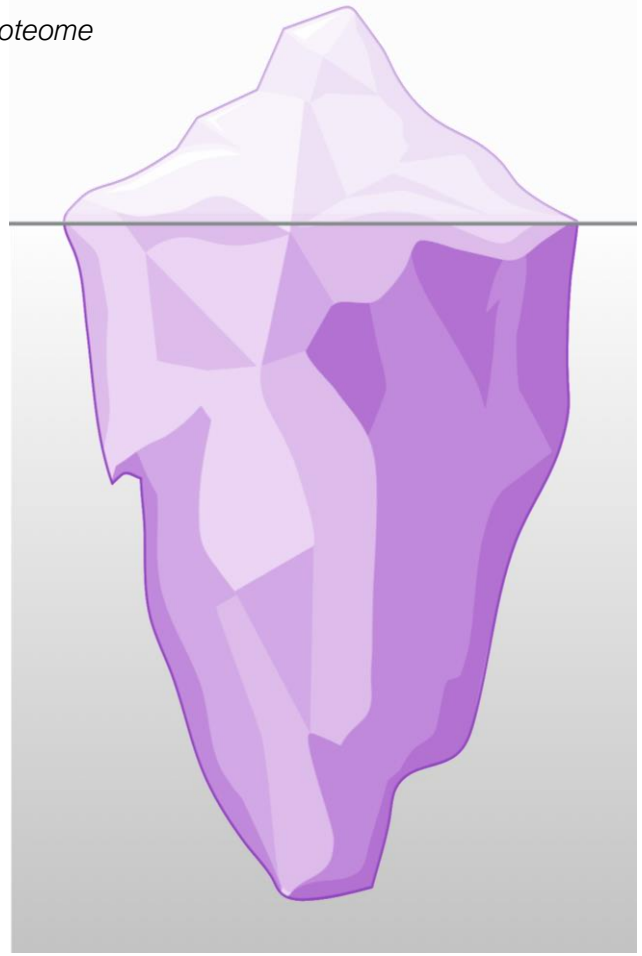
Solid tumors are heterogenous, but oncogenic driver mutations are conserved

Targeting KRAS has been clinically de-risked by approved G12C therapies, but depth and duration of response fall short and unmet need remains high

Prior et al. Cancer Res (2020)

TCRs Enable Targeting of Intracellular & Hard-to-Drug Oncogenic Drivers

Surface-associated Proteins
~ 27% of the proteome



Intracellular Proteins
~73% of the proteome

Conventional CAR cellular therapies & ADCs are limited to targeting surface proteins

TCR-based therapies enable precise targeting of intracellular proteins presented as epitopes on the cell surface

This allows direct targeting of hard-to-drug oncogenic drivers

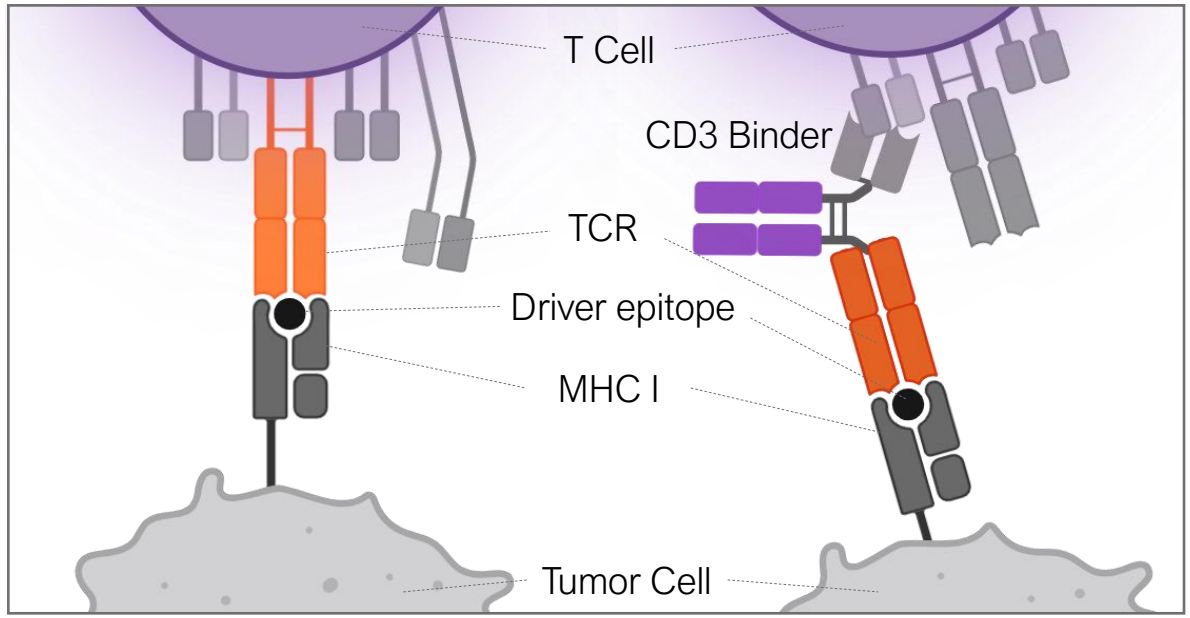
Klebanoff & Chandran: Immunological Reviews, Volume: 290, Issue: 1, Pages: 127-147, First published: 29 July 2019, DOI: (10.1111/imr.12772)

CAR: Chimeric Antigen Receptor
ADC: Antibody Drug Conjugate

Affini-T is Developing Two TCR-Based Therapeutic Modalities

TCR-T Cell Therapies

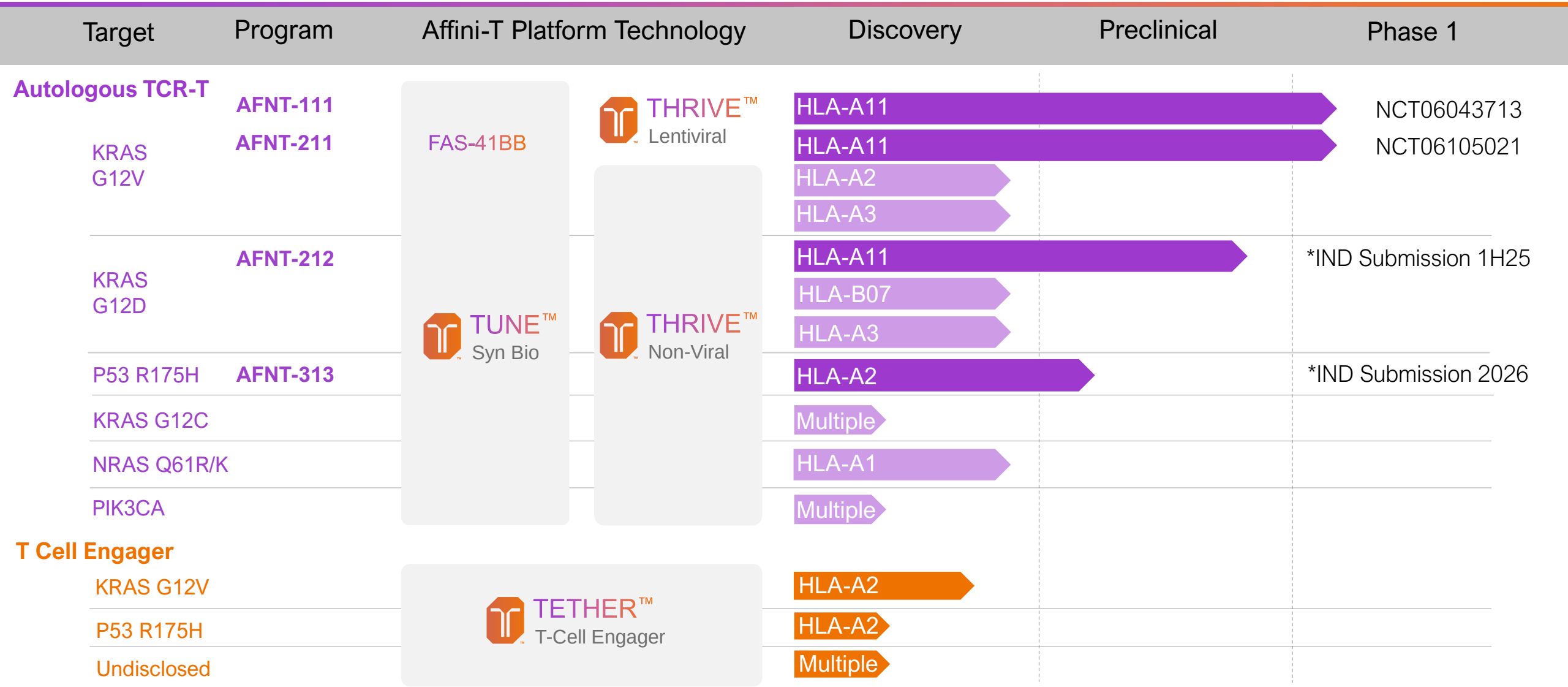
T cells engineered with a transgenic TCR that allows recognition of specific driver mutant epitopes



Bispecific T Cell Engagers

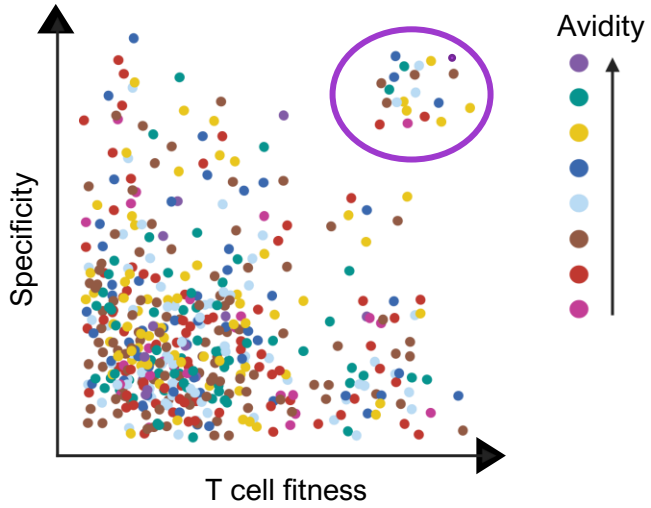
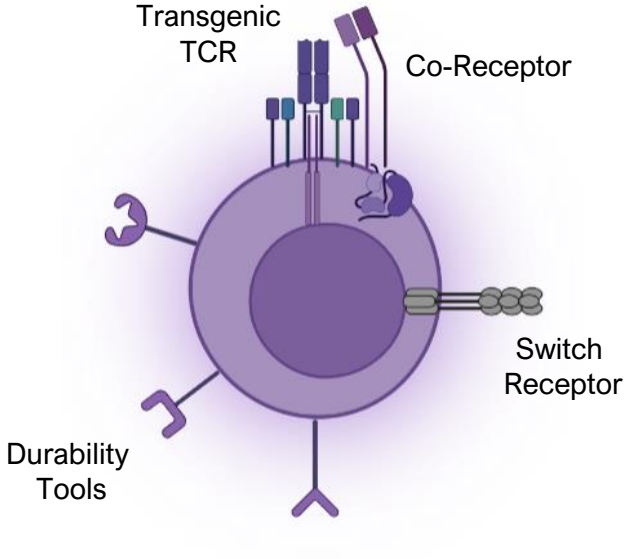
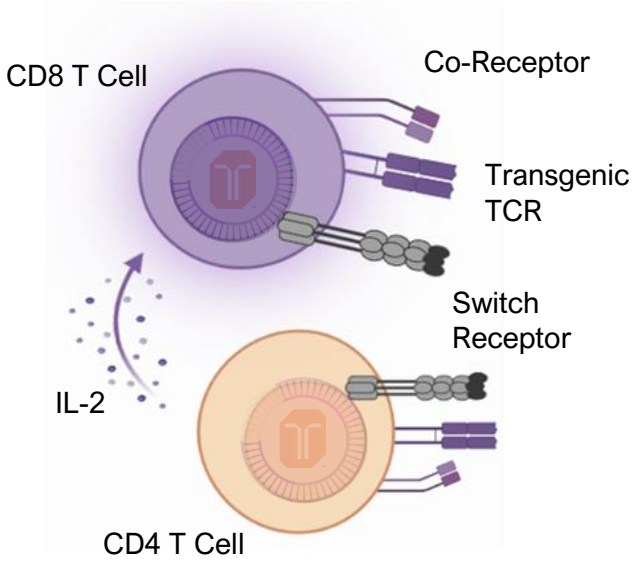
Bispecific biologics combining a TCR moiety to recognize the driver mutant epitope with a CD3 binding moiety to recruit endogenous T Cells

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



*Planned submission dates

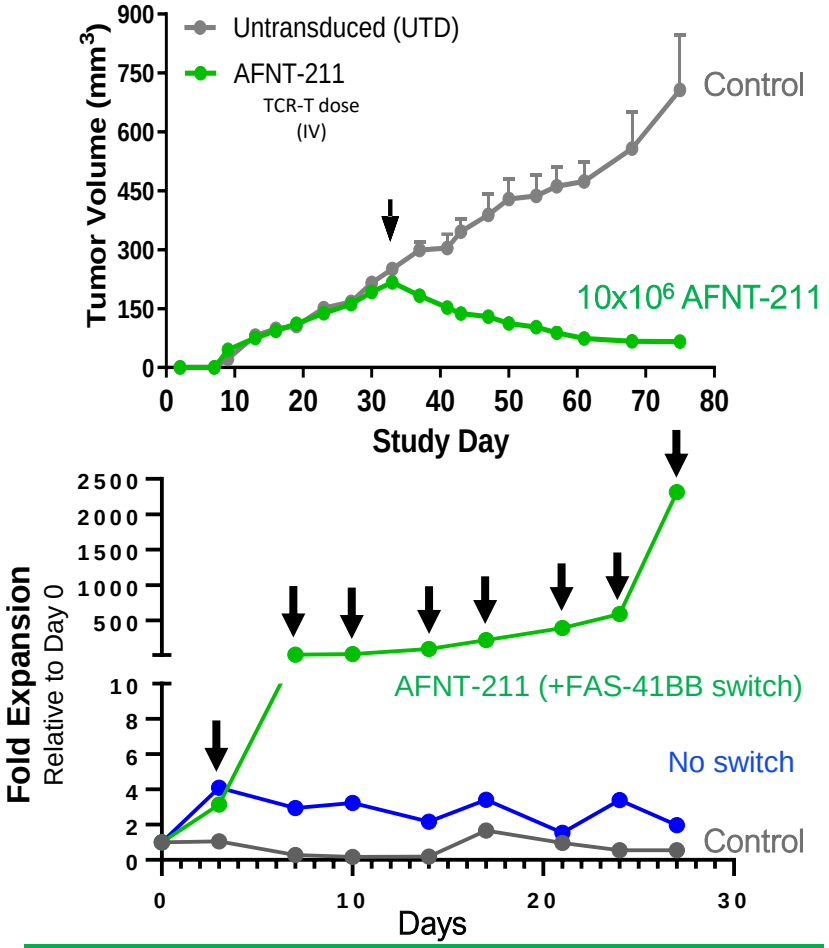
Affini-T Platform Technologies Enable the Generation of Potent & Tolerable TCR-T Cells

TAILOR™ TCR Discovery	TUNE™ Synthetic Biology	THRIVE™ Engineering and Manufacturing
		
Predictive algorithms & machine learning identify highly functional and 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 > 95% central memory / stemness phenotype

TME: Tumor Microenvironment

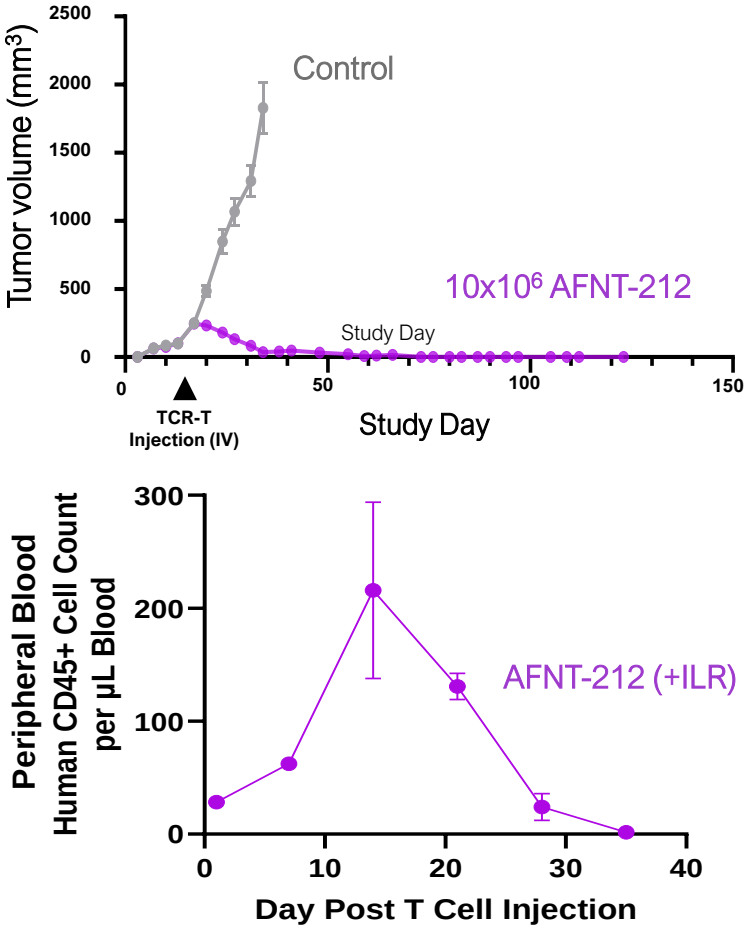
Innovative pipeline leverages TAILOR™, TUNE™ and THRIVE™ to eradicate difficult-to-treat solid tumors

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



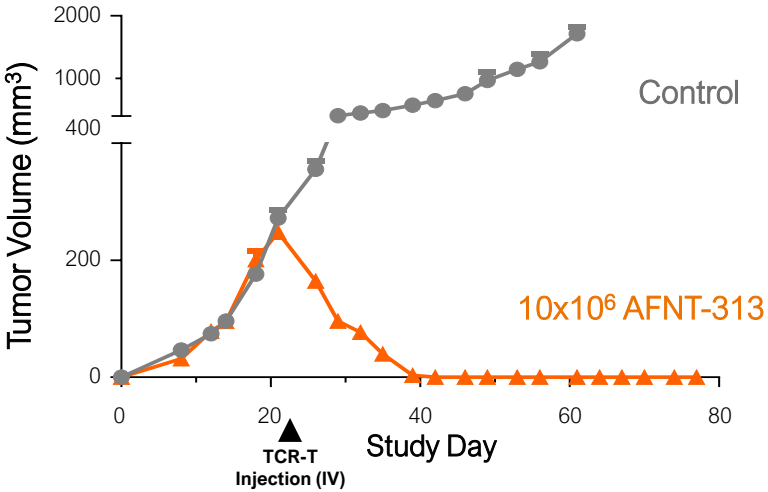
Signal 1+2: Enhanced survival in TME

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



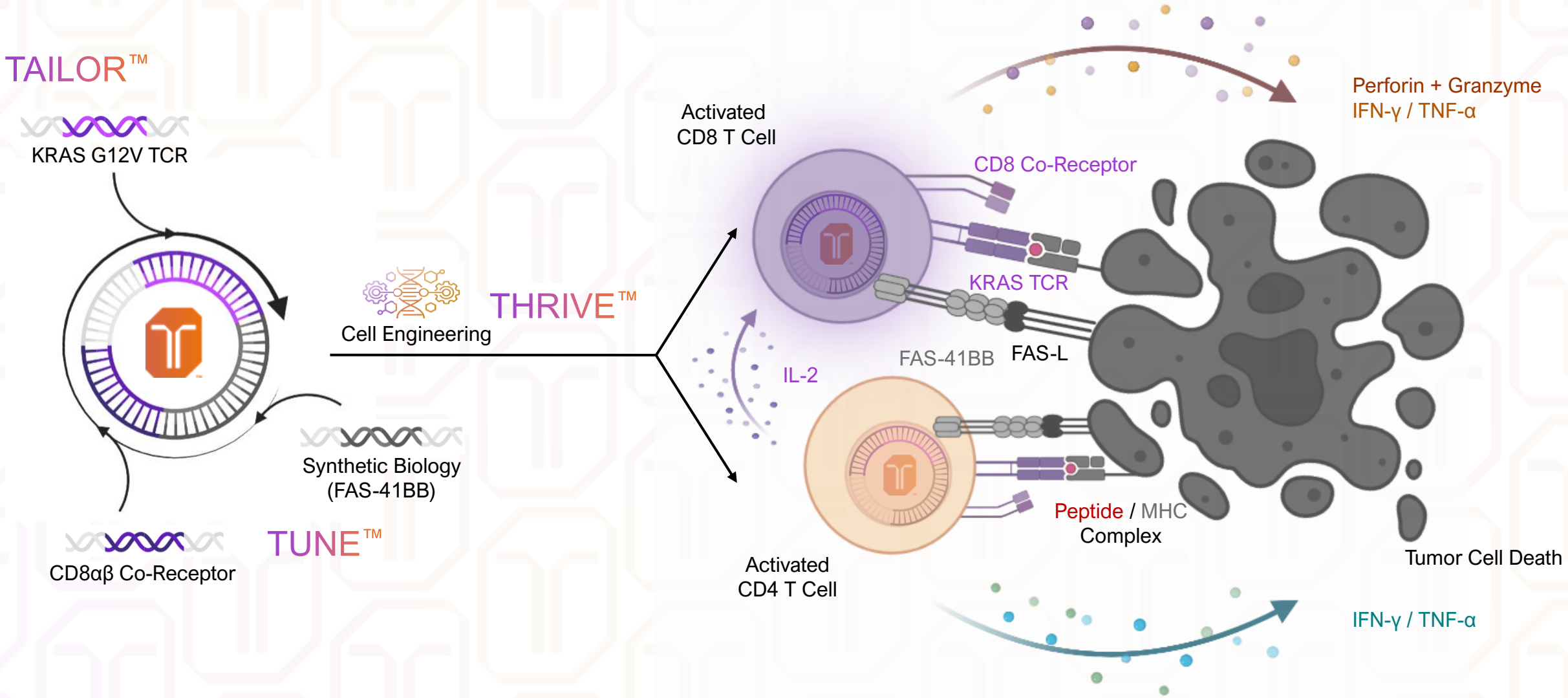
Signal 1+3: Enhanced proliferation in periphery

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



Signal 1+2+3: Support in TME + periphery

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



AFNT-211: Clinical Development Plan

Phase 1a Basket Trial Dose Finding
Sample size N=15-20
~10 US clinical trial sites

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

Non-small-cell lung (NSCLC) | Colorectal (CRC) | Pancreatic (PDAC) | Tissue Agnostic

Optimal Biological Dose / Proof of Clinical Concept

Phase 1b/2 Expansion Cohorts
Sample size up to N=20 per indication

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

Interim Analysis

Registration Study
Expand trial sites to 35-40 in US/EU5/CAN

- Continued FDA interactions for single arm study design
- Aim for approval based on ORR & DoR data
- Target sample size N=~80 for potential indication

ORR & DoR

**Excluding primary brain tumors*

ORR – Overall Response Rate
DoR – Duration of Response

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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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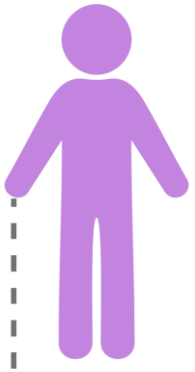
AFNT-211: Patient Journey

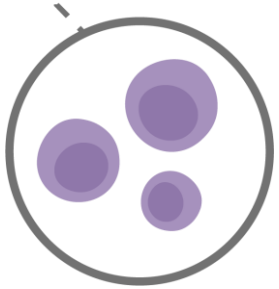
Pre-screen

- *KRAS G12V*
- *HLA-A*11:01*

Eligibility Screen

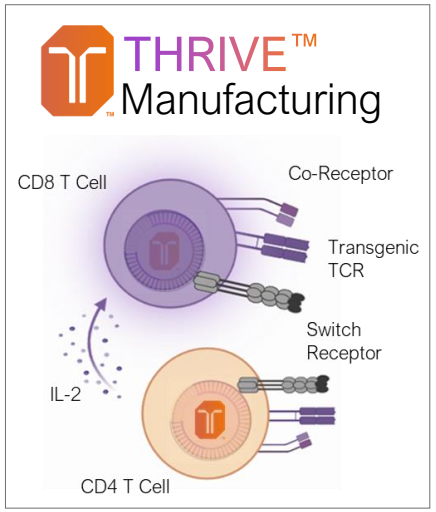
Leukapheresis





elevatebio

~10-day manufacturing process +
~6-day QC testing



Lymphodepletion**

6 Day LDC

Ship to clinic

AFNT-211 product infusion

Patient Monitoring

Transition to long term follow up

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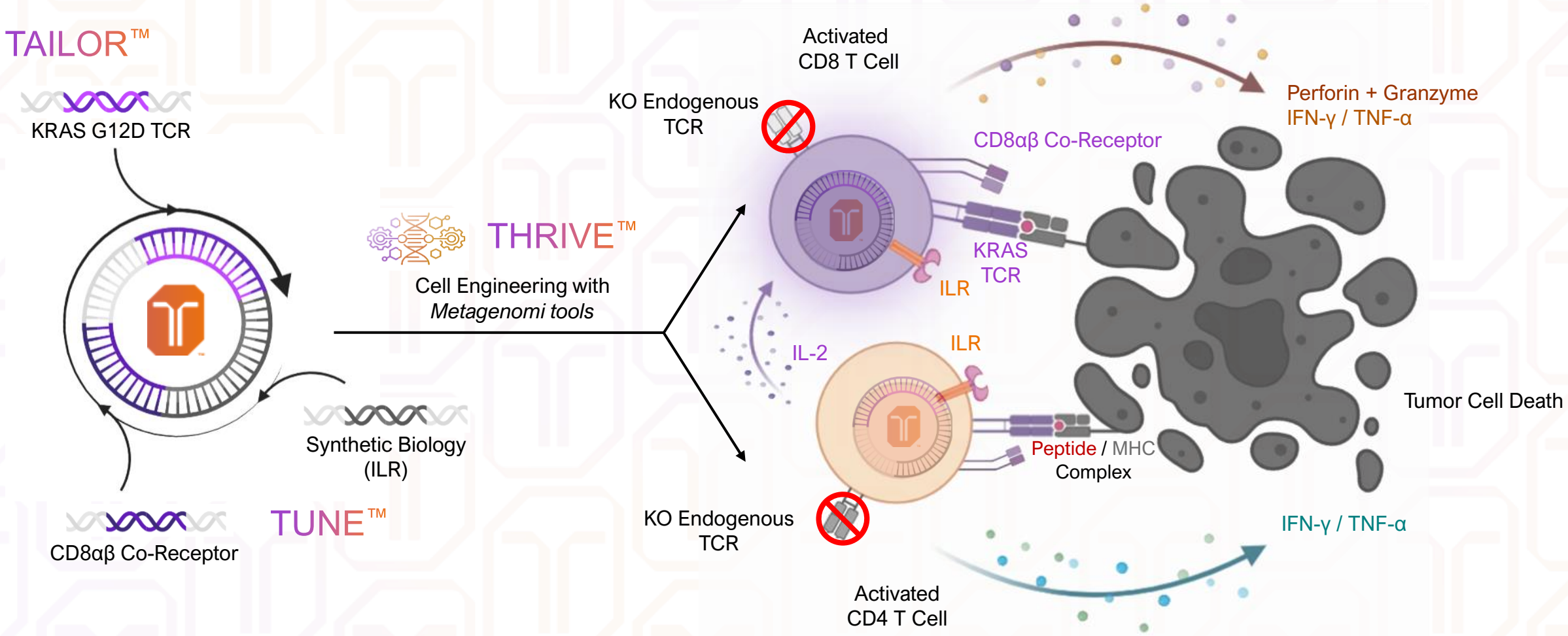
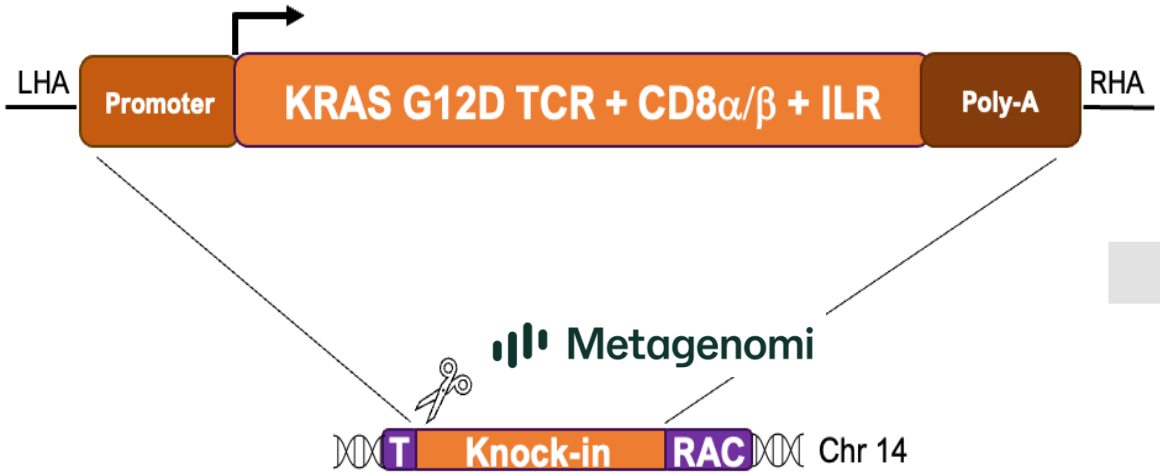
AFNT-212: A11 KRAS G12D TCR Engineered T Cells + Durability Switch Receptor + Gene Editing



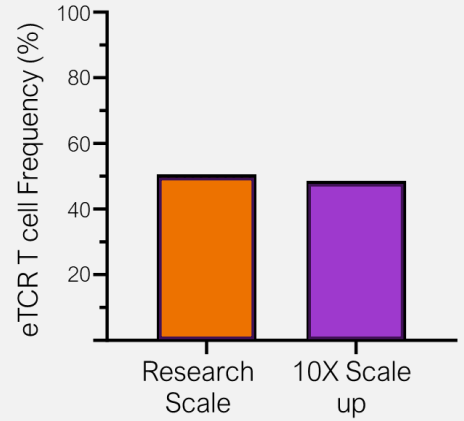
THRIVE™ High Efficiency Non-viral Delivery of Large Transgenes at cGMP Scale



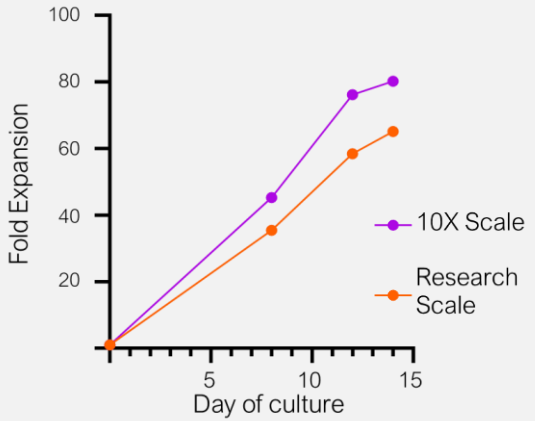
Transgenes inserted within the endogenous TRAC gene via CRISPR/Cas driven homology mediated repair

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

Transgene Integration Frequency



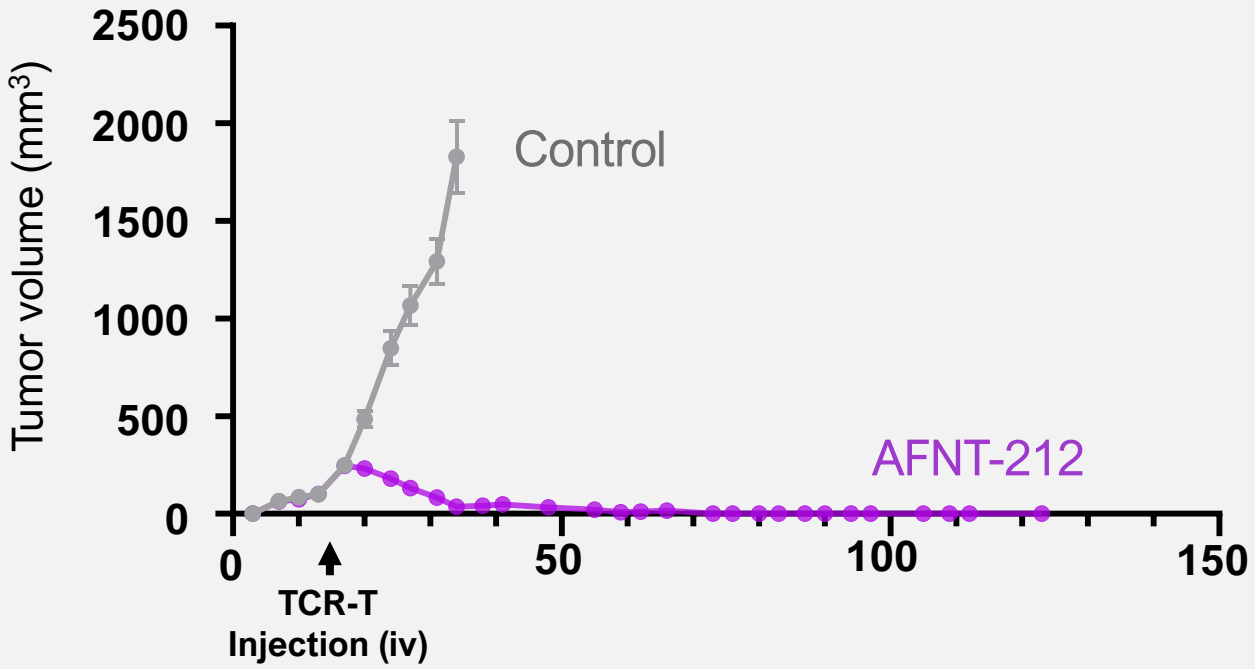
Expansion Kinetics



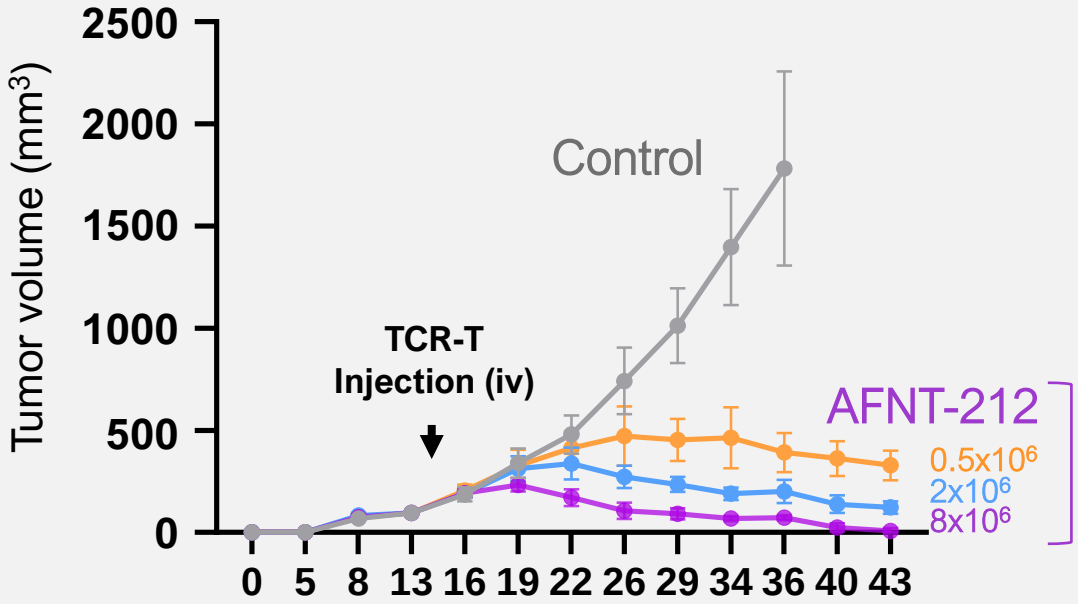
KI = Knock-In

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

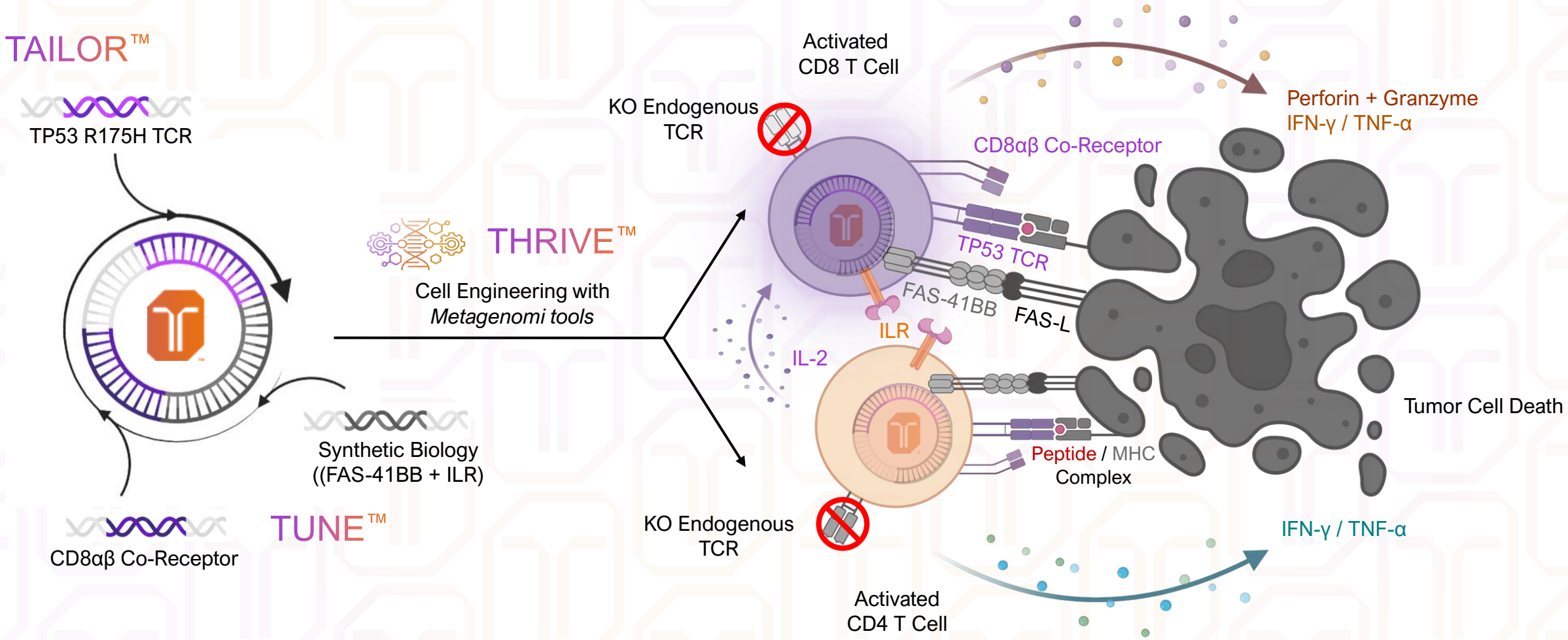


AFNT-212 T cells drove a dose-dependent anti-tumor response



(CL-40 colorectal model)

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



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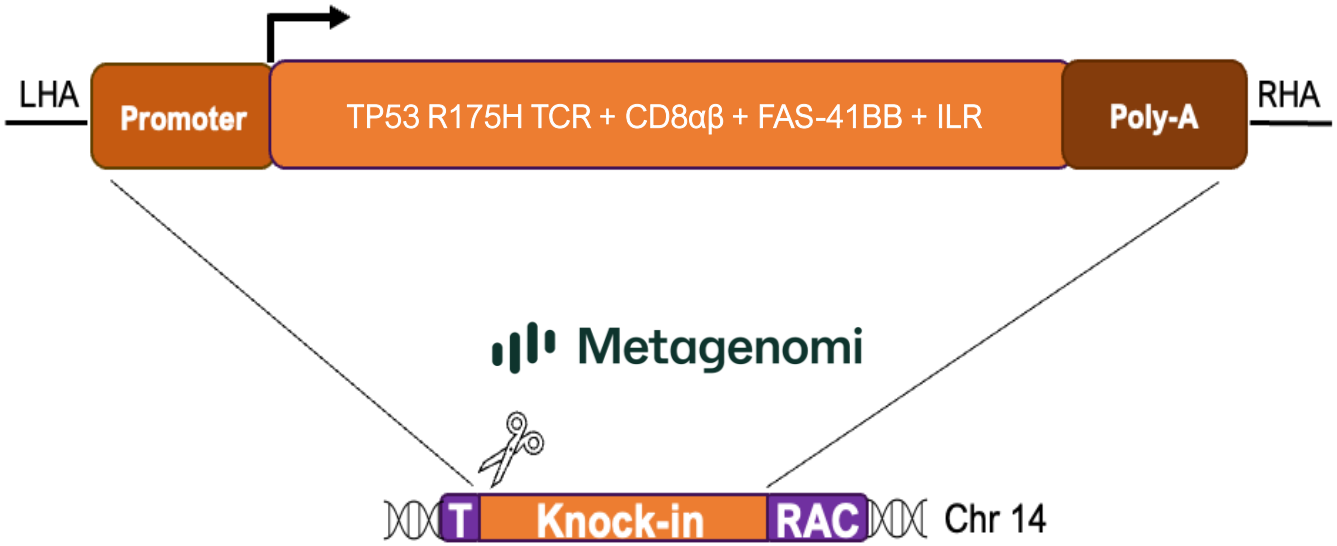
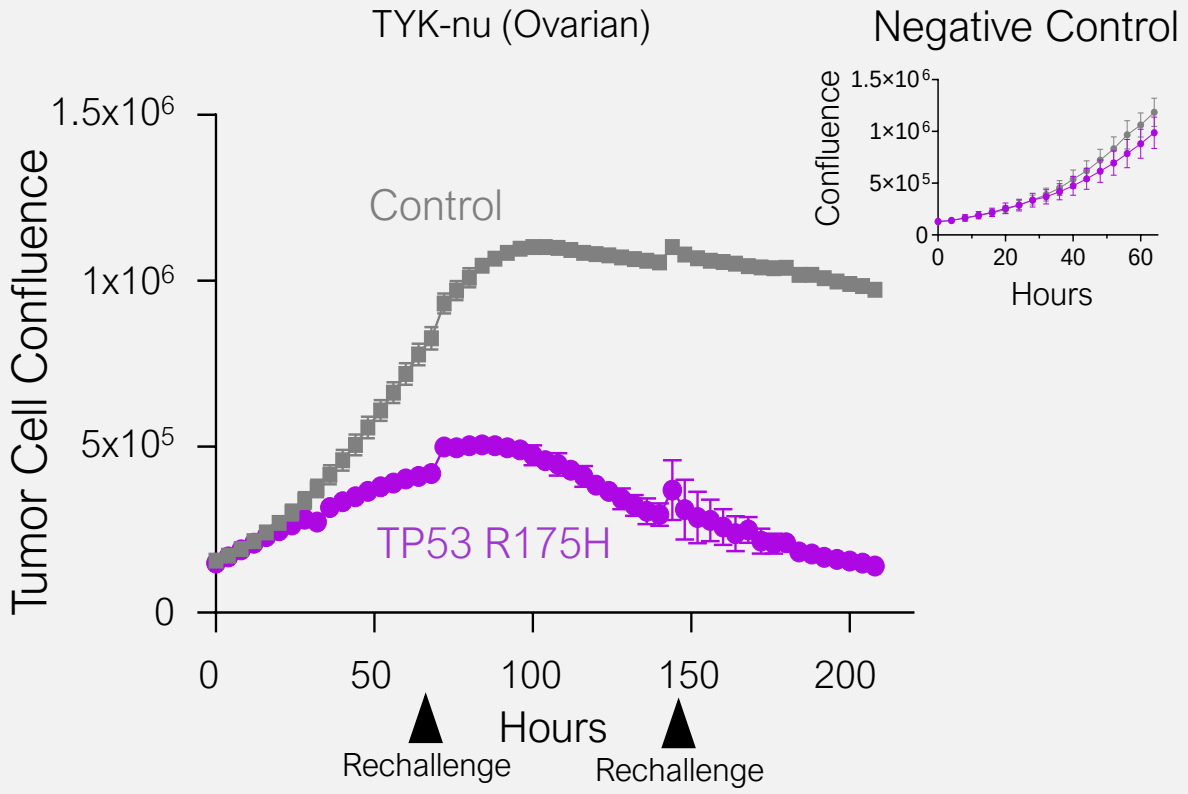


Transgenes inserted within the endogenous TRAC gene via CRISPR/Cas driven homology mediated repair

TRAC-inserted knock-in of 7 kb 6 gene cassette

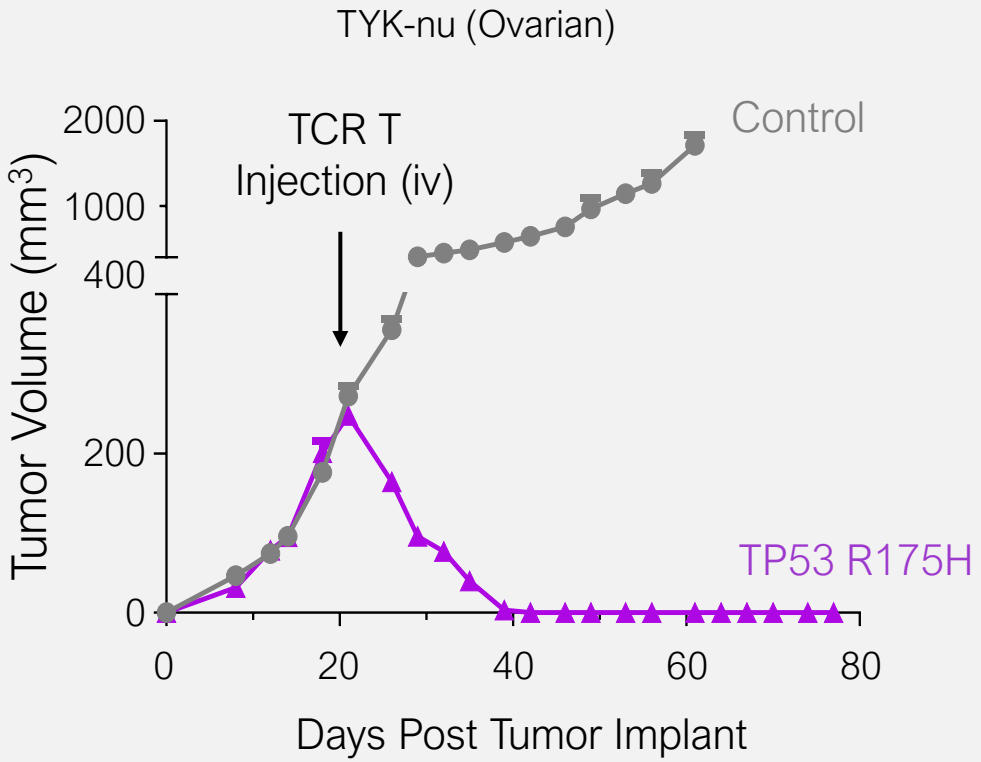
AFNT-313 TCR-T Showed Robust Preclinical Tumor Cell Control *In Vitro* and *In Vivo*

In Vitro



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

In Vivo



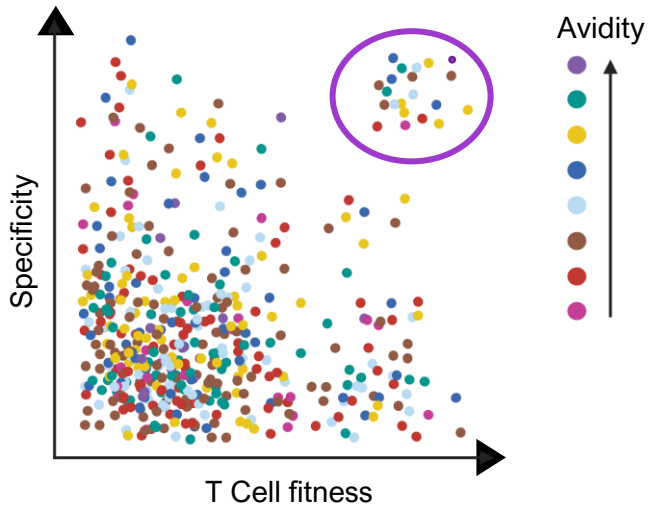
TP53 R175H TCR-T drove complete responses even against large established tumors

TETHER™ T cell engager Highlights

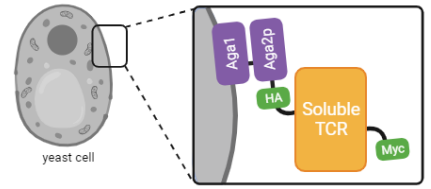
Affini-T Platform Technologies Enable the Generation of Highly Specific & Active T Cell Engagers

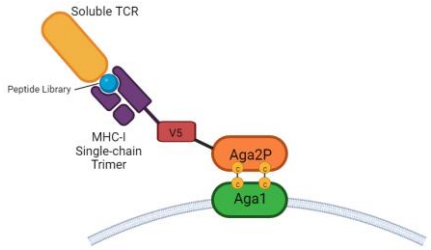
1 TAILOR™ TCR Discovery

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



2 Affinity Maturation



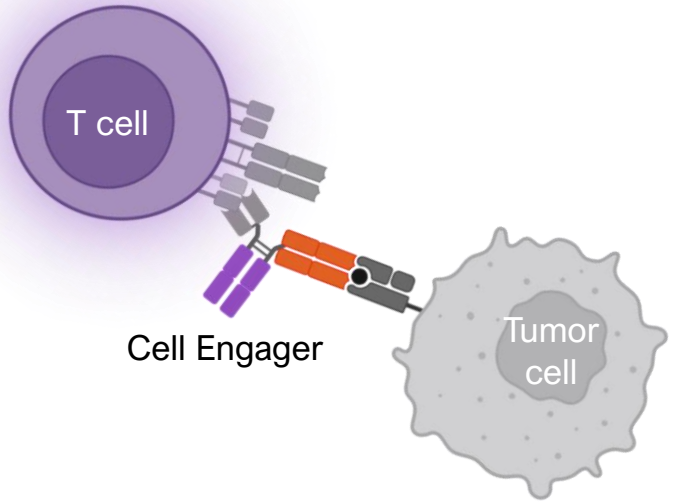


Yeast Display Modalities

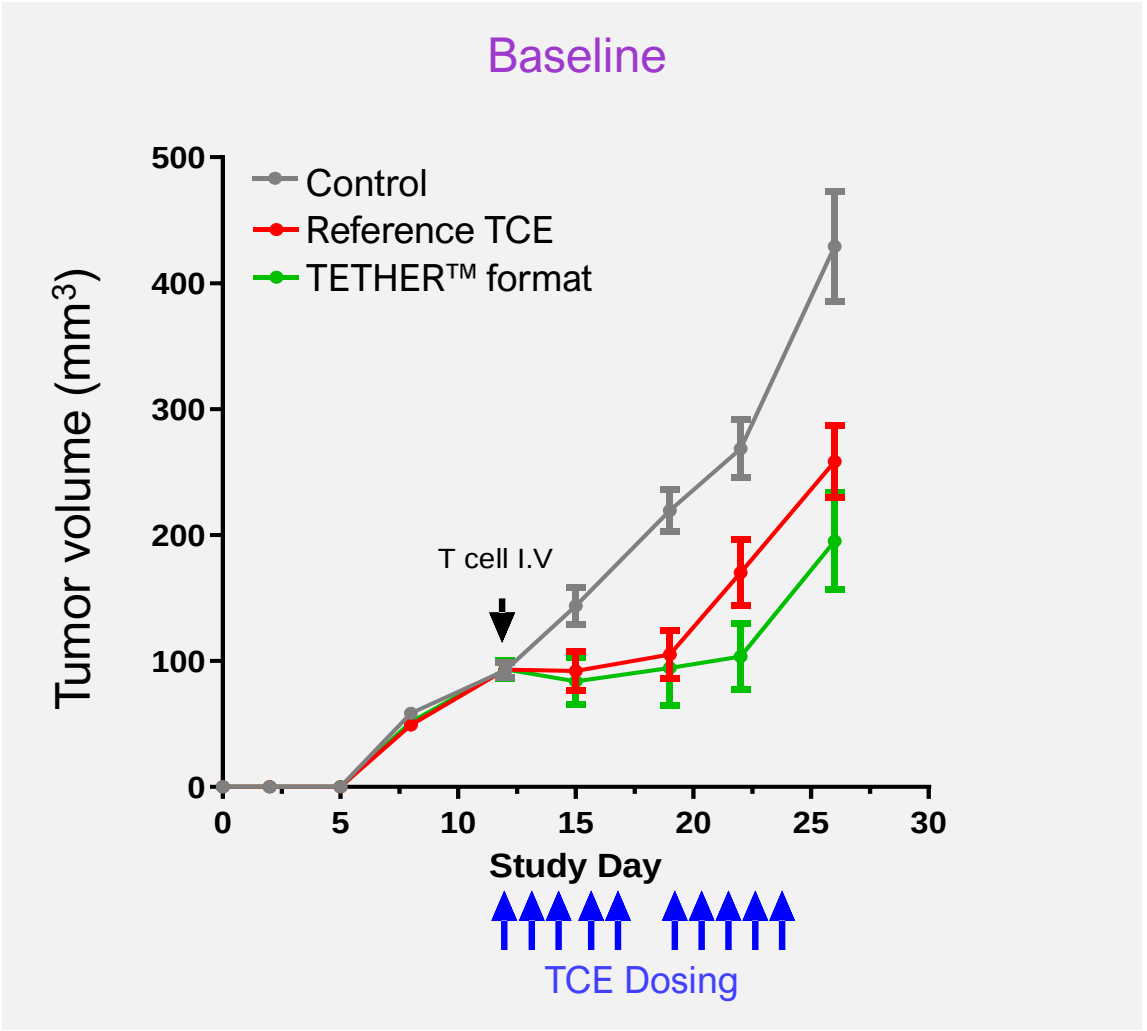
- Libraries to identify high affinity TCRs
- Libraries for specificity screenings

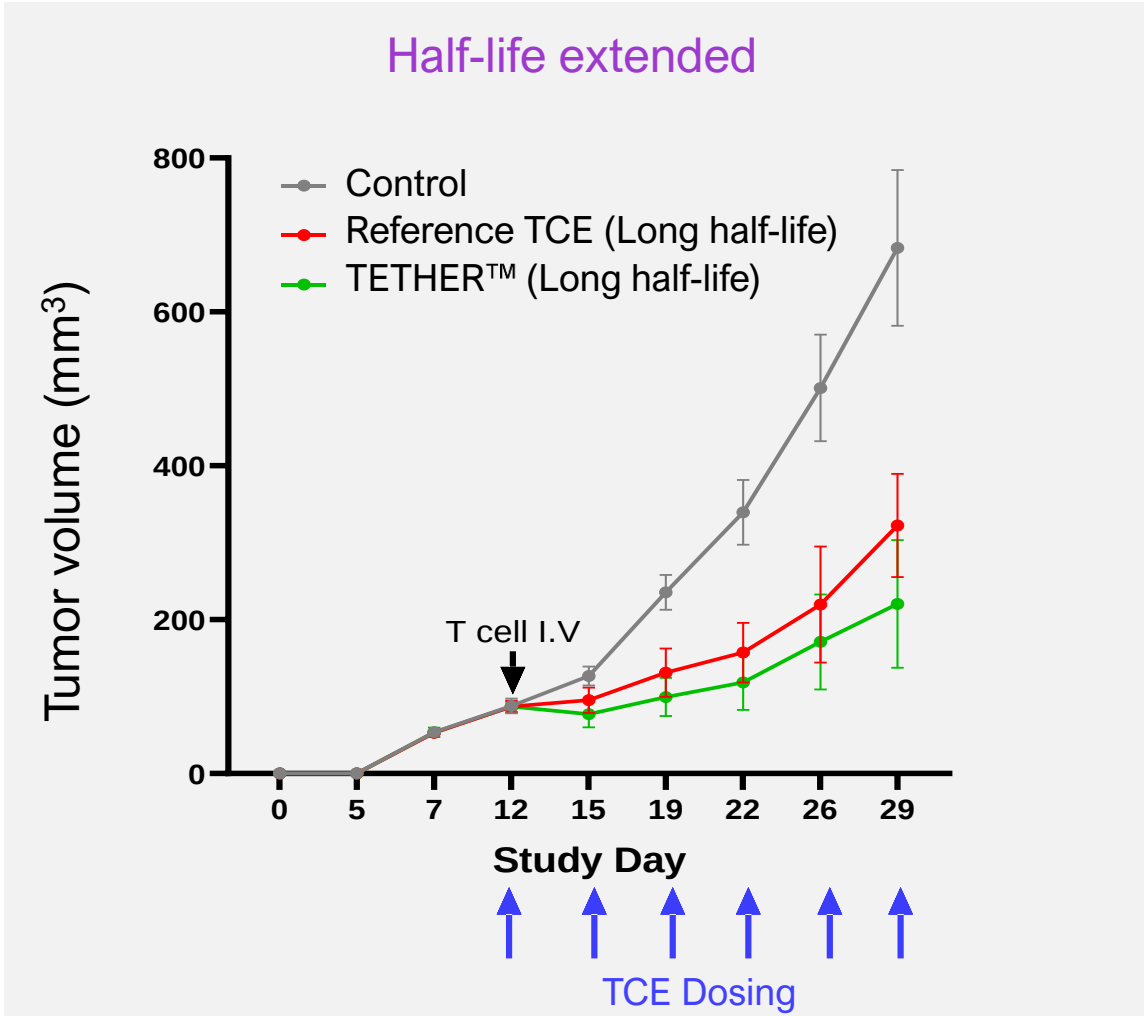
3 TETHER™ T Cell Engagers

- 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



TETHER™ T Cell Engagers Outperformed Reference Product Format *in vivo*

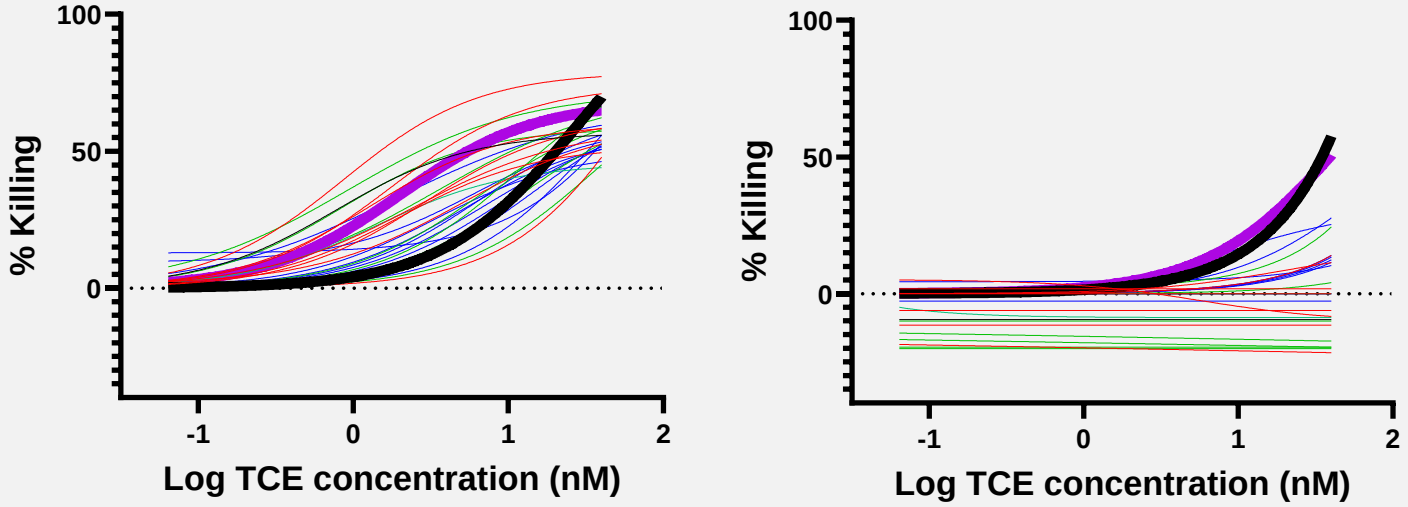




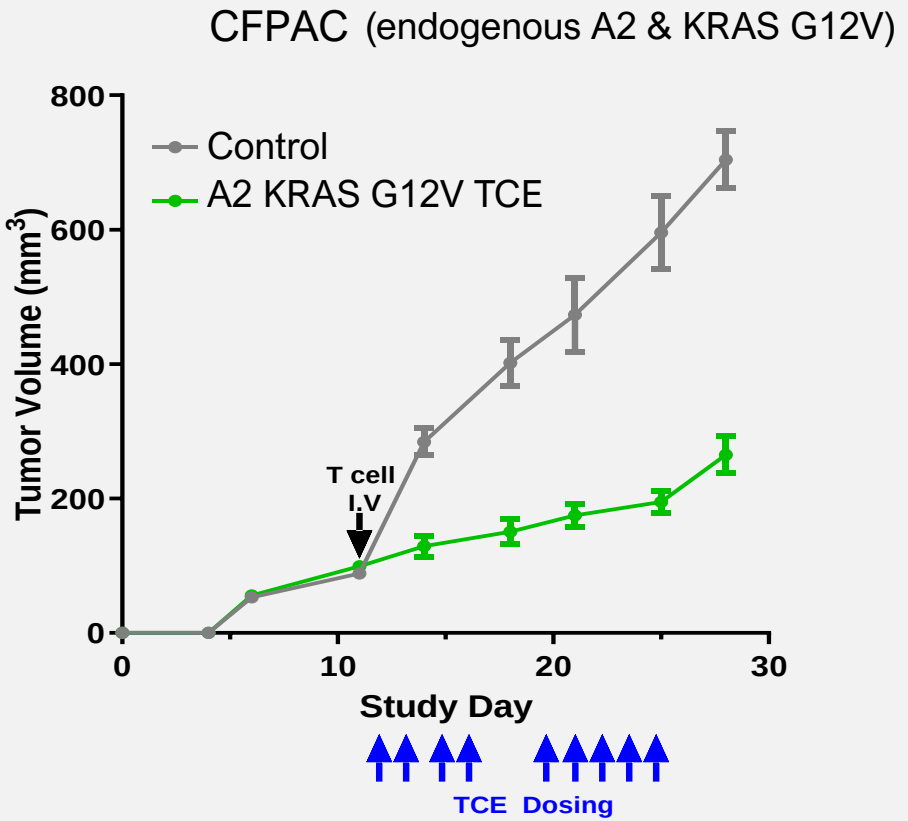
TCE: T Cell Engager

A2 KRAS G12V T Cell Engagers Displayed Functional Activity in *in vitro & in vivo* Preclinical Studies

In vitro activity



In vivo activity



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VIDA ventures · humboldt · InnoPinnacle · AGENT CAPITAL · ALEXANDRIA · leaps by Bayer · PARKER INSTITUTE FOR CANCER IMMUNOTHERAPY · CATALIO CAPITAL MANAGEMENT · ERASCA Ventures

Exceptional Scientific Co-Founders & SAB Specialized in T Cell Biology and Immunology

Co-Founders

Phil Greenberg, MD
Scientific Co-Founder

Aude Chapuis, MD
Scientific Co-Founder

Tom Schmitt, PhD
Scientific Co-Founder

Chris Klebanoff, MD
Scientific Co-Founder

Scientific Advisors

Jim Allison, PhD

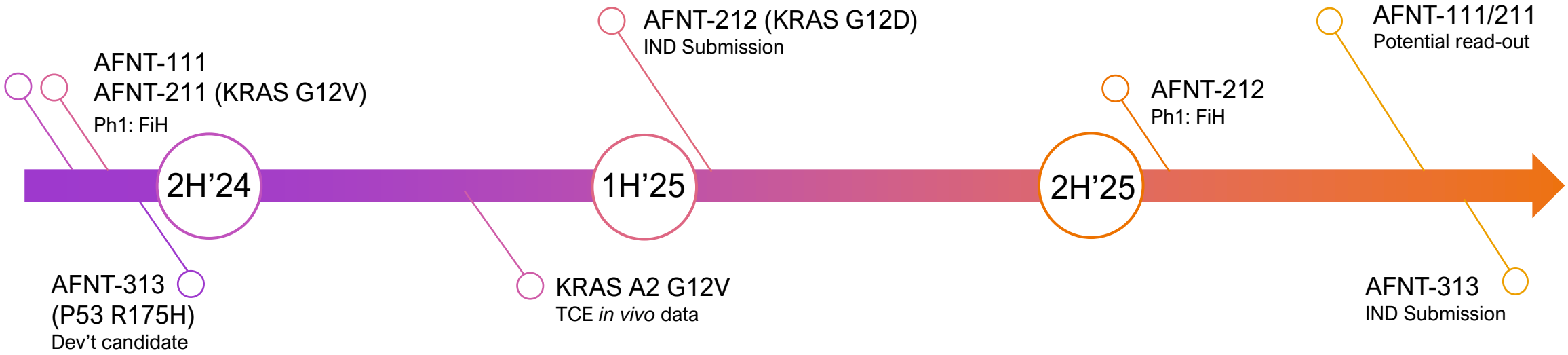
Pam Sharma, MD

Rafi Ahmed, PhD

David Kranz, PhD

Sue Kaech, PhD

Current Status & Key Clinical Catalysts



Affini-T is the premier Precision Immunotherapy company targeting oncogenic driver mutations to develop curative therapies for patients with solid tumors

Partnership Opportunities

TAILOR™
TCR Library for Oncology + I&I

THRIVE™
Engineering & Manufacturing

TUNE™
SynBio Armoring Technology

TETHER™
Bi-specific T Cell Engagers

Strategic Partners

Fred Hutch Cancer Center
Memorial Sloan Kettering Cancer Center
elevatebio
Metagenomi
ADIMAB

* All future catalysts and milestones planned but not guaranteed

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