



TRANSFORMING THE LIVES OF
PATIENTS WITH AUTOIMMUNE
DISEASES AND CANCER

Making Cell Therapies Accessible to All™

Fate
THERAPEUTICS

Corporate Presentation

February 2026

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Q1 2026 Business Updates & Near-Term Objectives

Competitively positioned to accelerate clinical stage development

	Milestones Achieved	Near Term Next Steps
Autoimmune Development (FT819)	✓ 15 lupus patients enrolled with 7 initial sites, with 16 total clinical sites now activated ¹	Complete Phase 1 SLE cohort enrollment by mid-2026 Continue no conditioning dose escalation and multi-dosing
	✓ Engaged with FDA under RMAT designation to align on pivotal trial and protocol strategy	Gain FDA alignment on registrational protocol and initiate pivotal trial in 2026
	✓ Continued patient treatment expansion beyond SLE with 4 SSc and one IIM patient treated ¹	Conduct dose expansion in IIM, SSc, and ANCA vasculitis. Expand into RA & additional indications ²
Oncology Development (FT825)	✓ Optimized FT825 patient enrollment strategy	Complete dose escalation with a focus on HER2+ patients
Next Gen Development w/ Sword & Shield™ (FT836 & FT839)	✓ Treated 3 patients in FT836 (MICA/B) pan-solid tumor Phase 1 trial ¹	Complete dose escalation cohorts by mid 2026
	✓ Initiated FT836 in multiple myeloma IIT	Enroll first multiple myeloma patient 1H 2026
	✓ Commenced FT839 IND enabling activities	Initiate Phase 1 pan-autoimmune trial in 2026
Cash & Equivalents	✓ \$205 million in cash, cash equivalents, & investments ✓ 30% reduction in operating expenses in 2025 compared to 2024	Projected operating runway through year-end 2027

1. Data as of February 25th

2. ANCA = Anti-neutrophil cytoplasmic auto-antibody associated vasculitis; IIM = Idiopathic inflammatory myositis; RA = Rheumatoid Arthritis; SLE = Systemic Lupus Erythematosus; SSc = Systemic Sclerosis

FDA = Food and Drug Administration

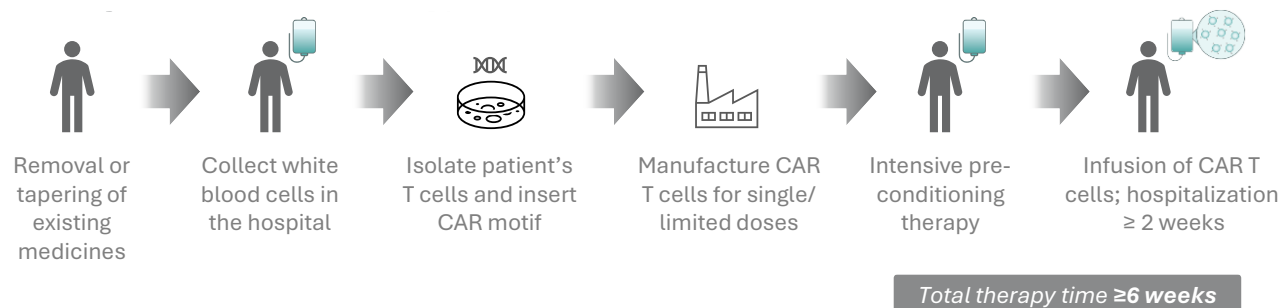
RMAT = Regenerative medicine advanced therapy

Outpatient Treatment Enabled CAR T-cell Therapy Platform

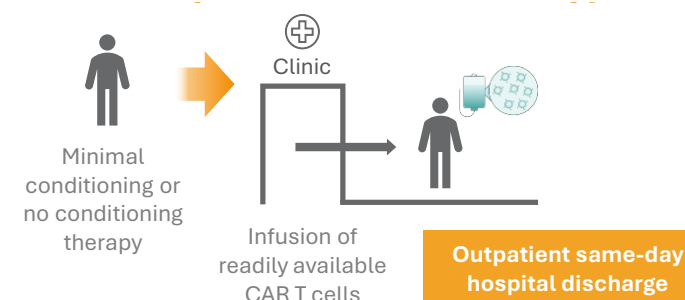
First CAR T-cell therapy in autoimmune without the requirement for hospital stay

- ✓ Successful dosing of FT819 in an outpatient setting as same-day discharge without extended hospitalization and observation
- ✓ Elimination of inpatient care and the reduction/elimination of conditioning chemotherapy combined with on-demand availability facilitates broad patient access
- ✓ Providing off-the-shelf CAR T-cell therapy in community hospital-based treatment settings reduces the burden on healthcare infrastructure, and unlocks a viable, scalable and practical therapeutic paradigm

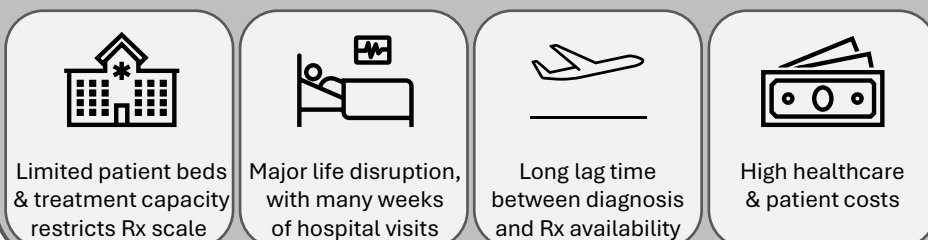
Autologous CAR T-cell Therapy



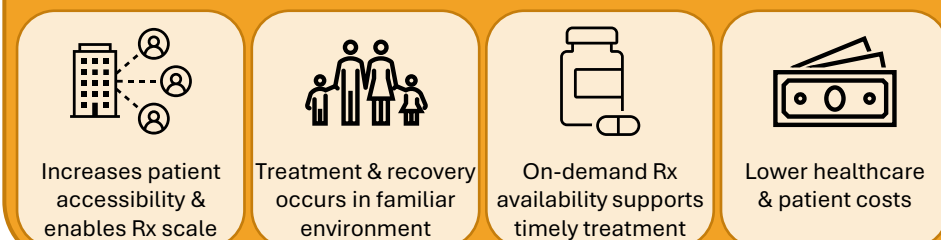
Off-the-Shelf CAR T-cell Therapy

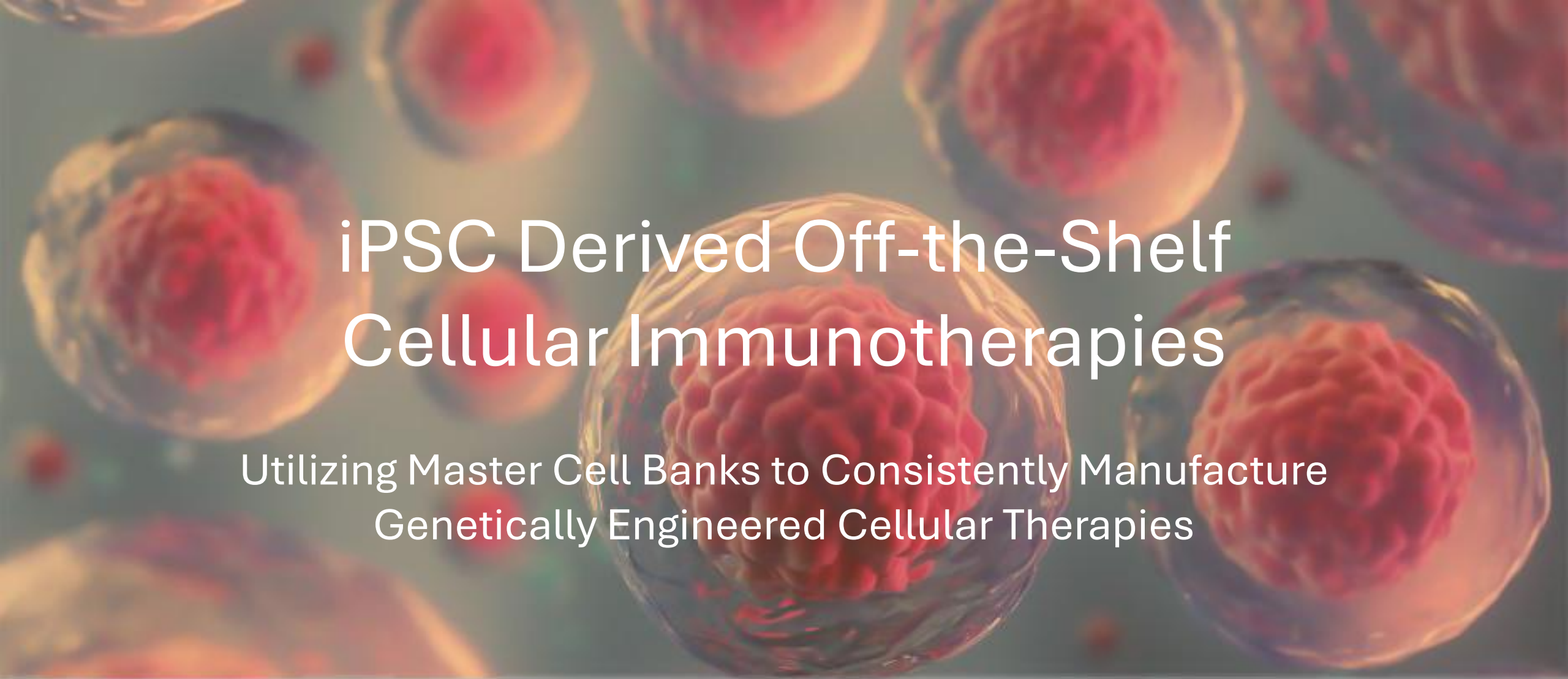


Inpatient Care (Hospital Setting) Existing CAR T-cell therapies in autoimmune



Outpatient Care (Community Setting) Fate Therapeutics CAR T-cell therapies in autoimmune





iPSC Derived Off-the-Shelf Cellular Immunotherapies

Utilizing Master Cell Banks to Consistently Manufacture
Genetically Engineered Cellular Therapies



Making Cell Therapy Accessible to All™

Unique Platform For Delivery Of Off-the-Shelf Cellular Therapies

Mass produced, multiplexed-engineered cell products for on-demand patient treatment

iPSC → **any**
cell in the body

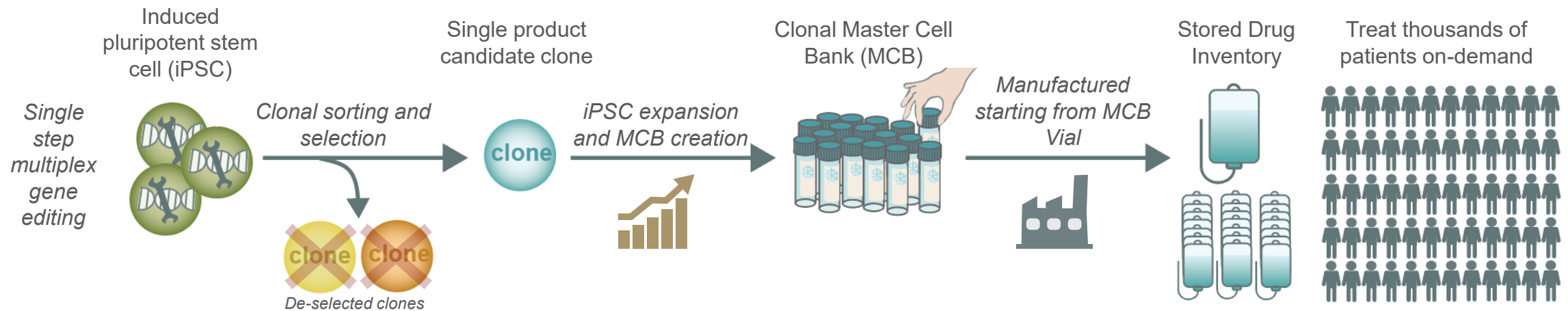
Gene editing is
a **one-time**
event completed
at iPSC stage

Master cell
bank contains
clonally
identical
population

Master cell bank is
starting material
for CAR T-cell
manufacture

Consistent
manufacture
results in uniform
and homogenous
drug product

Off-the-shelf
drug product
supports on-
demand delivery
for broad patient
reach



Mass Production Of Cell Therapy Drug Products

Advanced manufacturing capabilities to provide clinical and early commercial supply

State of the Art GMP facility (San Diego, CA, USA)

- 40,000+ ft² cGMP manufacturing facility co-located with Fate Therapeutics' corporate headquarters
 - Capacity to generate approximately 50,000 doses per year at \$3,000 cost of goods per dose
 - 1 master cell bank with working cell bank intermediates, may produce more than 10 million doses
- End-to-end capabilities and controls
 - Licensed by the State of California, Department of Health Services, Food and Drug Branch
 - Commissioned and qualified with first drug product manufacturing runs completed
 - On-site integration with quality, assay development, and process development
- Supports USA and international clinical development as well as initial commercial launch



Highly Differentiated & Scalable Cell Therapy Paradigm

Fate's platform uniquely delivers off-the-shelf cell therapies on-demand to patients in need

Autologous CAR T Cell



- Profound efficacy in difficult-to-treat diseases
- Impaired starting material
- Random, variable, per-patient T-cell engineering
- Complex logistics
- Single dose paradigm
- Heterogeneous drug product
- Extended hospitalization
- Prohibitively Expensive (\$\$\$\$)

Allogeneic CAR T Cell



- Potential for profound efficacy in difficult-to-treat diseases
- **Healthy** starting material
- Random, variable, **per-batch** T-cell engineering
- Complex logistics
- **Multiple** dose paradigm
- Heterogeneous drug product
- Extended hospitalization
- Expensive (\$\$\$)

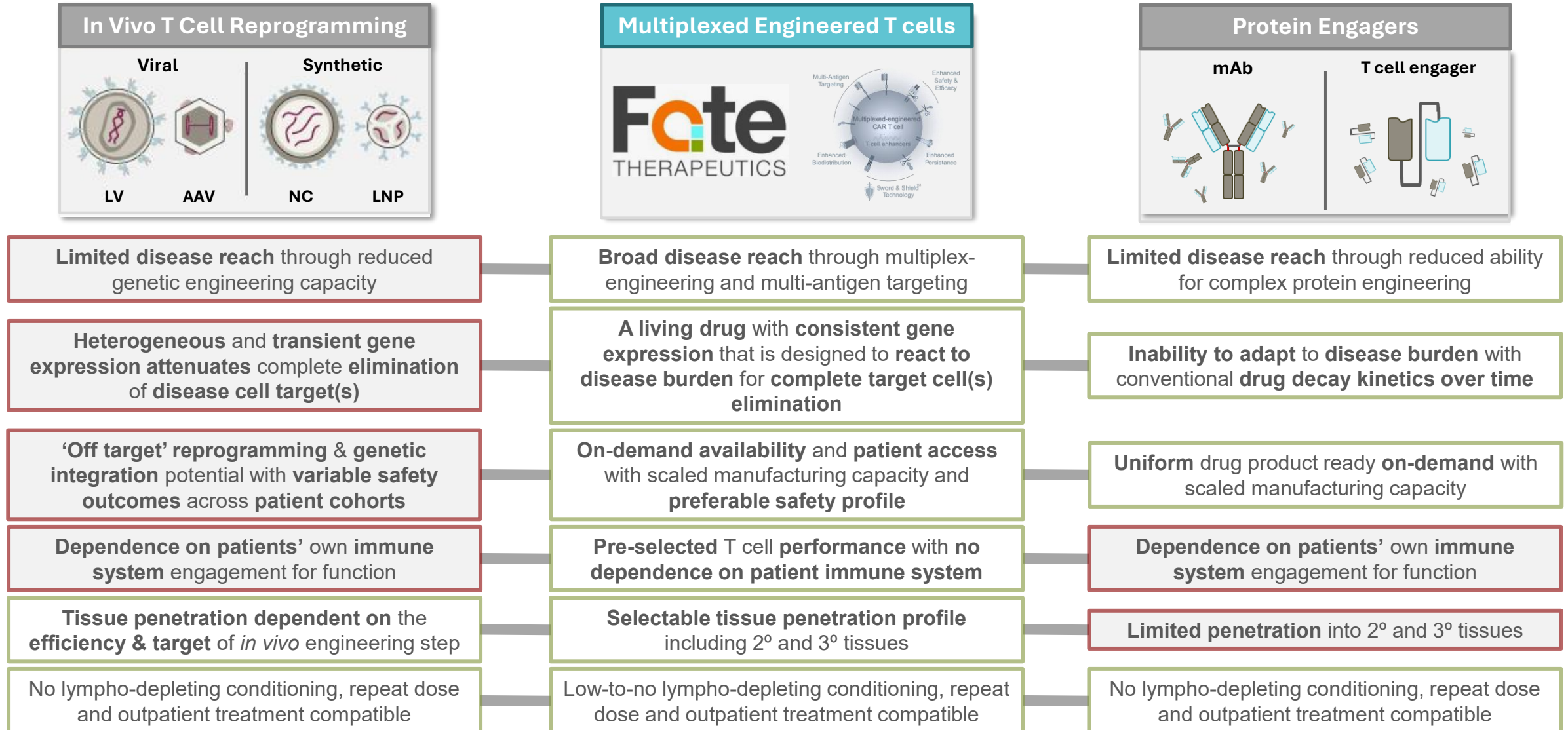
Accessible CAR T Cell



- Potential for profound efficacy in difficult-to-treat diseases
- **Healthy** starting material
- **One-time uniform iPSC precision** engineering event
- **Off-the-shelf** streamlined logistics
- **Multiple** dose paradigm
- **Homogenous** drug product
- **Reduced** hospitalization
- **Cost-Effective (\$)**

Immune Modulation Therapeutic Landscape

Fate's multiplexed engineered CAR T cells deliver the potency of a living drug with uniformity of traditional biologics



Off-the-Shelf Cellular Immunotherapy Product Pipeline

Multiplexed-engineered, iPSC-derived product candidates

Program	CAR/Antigen Target	Indication	Pre Clinical	Phase 1/2	Registrational	Partner
Autoimmunity						
FT819	CD19	Systemic Lupus Erythematosus (SLE) (RMAT)	FT819-102		NCT06308978 Enrolling	CIRM CALIFORNIA INSTITUTE FOR REGENERATIVE MEDICINE
		Systemic Sclerosis (SSc)	FT819-102			
		ANCA associated Vasculitis (AAV)	FT819-102			
		Idiopathic Inflammatory Myopathies (IIM)	FT819-102			
FT839	CD19/CD38 ^{1,2}	Pan-Indication (Autoimmune) w/o LCC	IND enabling studies			
FT522	CD19 ¹	Pan-Indication (Autoimmune) w/o LCC	FT522-102			
Oncology						
FT825	HER2 ¹	Solid Tumor (s)	FT825-101		NCT06241456 Enrolling	ONO ONO PHARMACEUTICAL CO.,LTD.
Undisclosed	Undisclosed	Solid Tumor (s)	Ongoing R&D activity			
FT836	MICA/B ¹	Pan-Indication (Solid Tumor w/o LCC)	FT836-101		NCT07216105 Enrolling	CIRM CALIFORNIA INSTITUTE FOR REGENERATIVE MEDICINE
FT836	MICA/B ¹	Multiple Myeloma (Heme Onc)	FT836 IIT			
FT839	CD19/CD38 ^{1,2}	Pan-Indication (Heme Onc) w/o LCC	IND enabling studies			

1. Product contains hnCD16 that allows combination with licensed monoclonal antibodies, i.e. anti-CD20 to extend antigen targeting capability
2. Product contains CD3 fusion receptor that allows combination with licensed T cell engagers, i.e. anti-BCMA and anti-GPRC5D to extend antigen targeting capability

RMAT: FDA Regenerative Medicine Advanced Therapy (RMAT) received in SLE

LCC: Lympho-conditioning chemotherapy

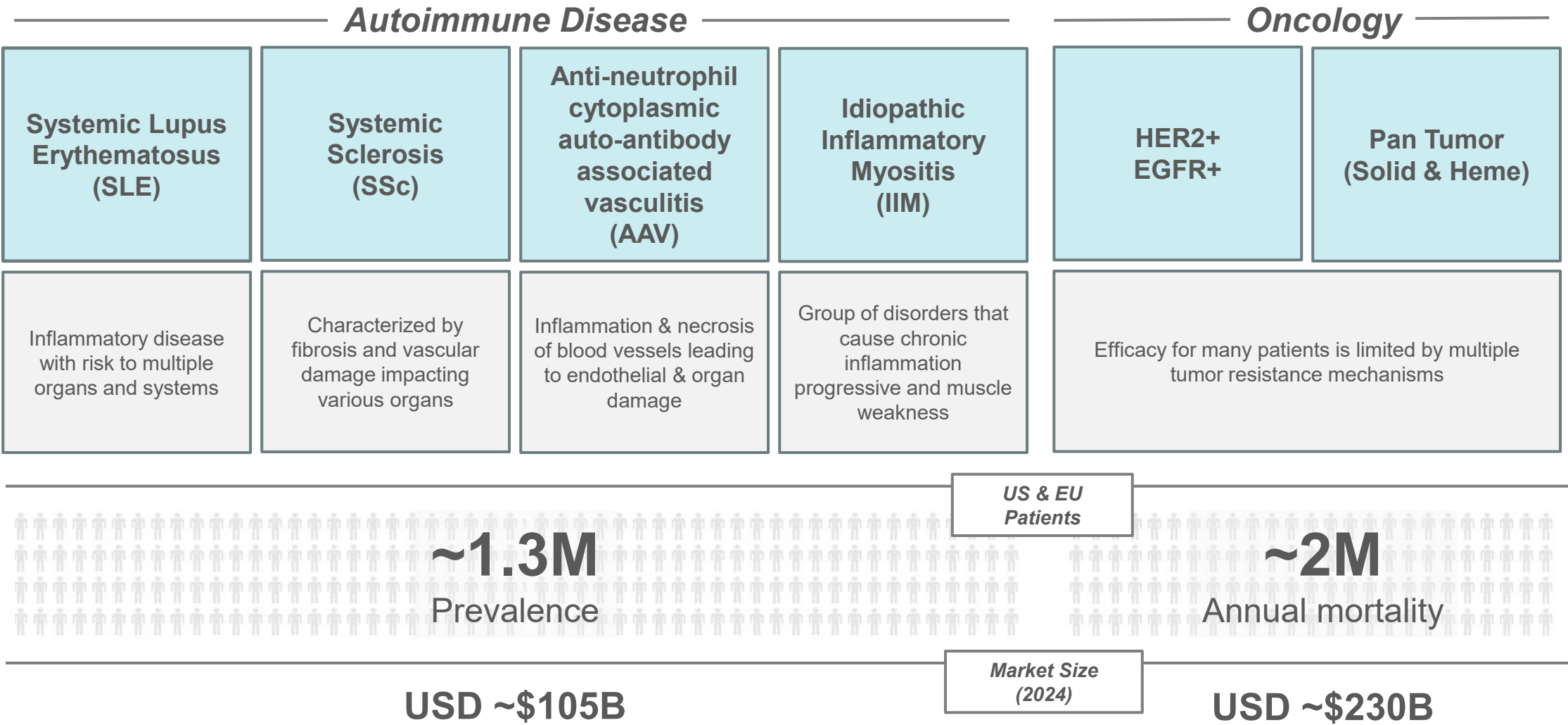
World rights to FT819, FT836, FT839 and FT522

T Cell Asset

NK Cell Asset

Addressing Diseases with Significant Unmet Clinical Need

Reaching patients in their communities with an off-the-shelf treatment without lympho-conditioning chemotherapy



1. Izmirly et al. Arthritis Reum 2021
2. Smoyer-Tomic et al. BMC Musc Disorders 2012
3. Bergamasco et al. Epi of Systemic Sclerosis 2019
4. Khoo et al. Nature 2023. Rare Disease Advisor, Nat'l Scleroderma Foundation
5. SEER, Dyba 2021
6. Market Reports World (Jul2025), BioSpace (Feb 2025)



FT819 Program

CD19 Targeting CAR T-Cell Product Candidate

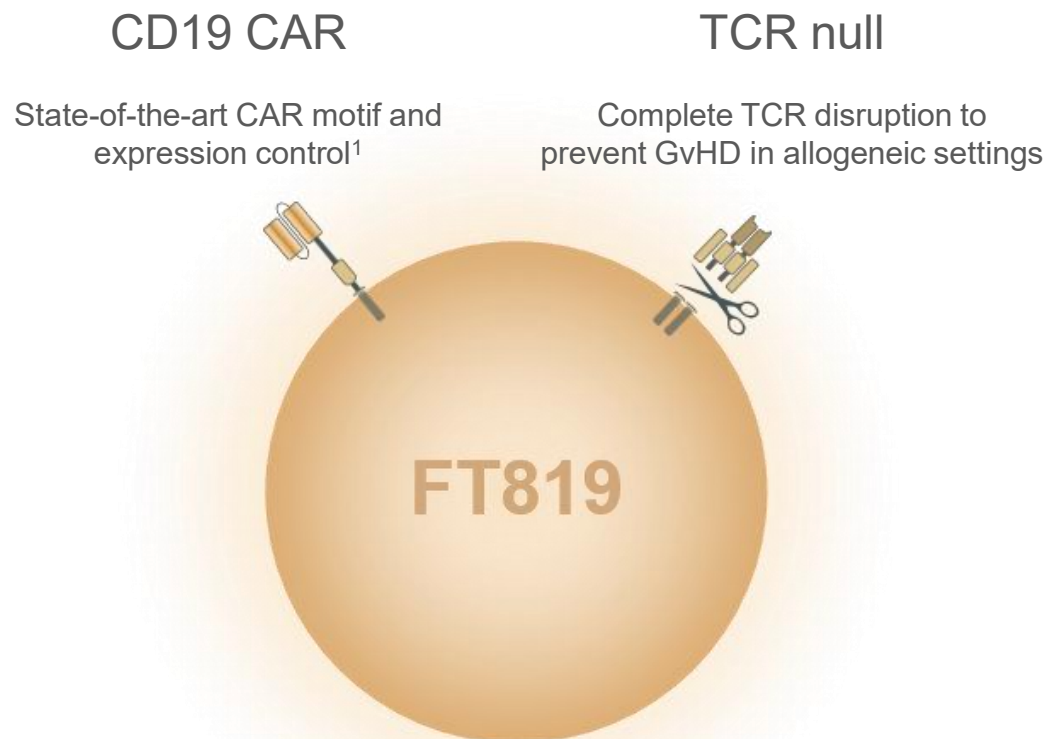


Making Cell Therapy Accessible to All™

FT819: Off-the-Shelf anti-CD19 CAR T-Cell Product Candidate

Safe and effective targeting of CD19+ B cells with broad patient accessibility

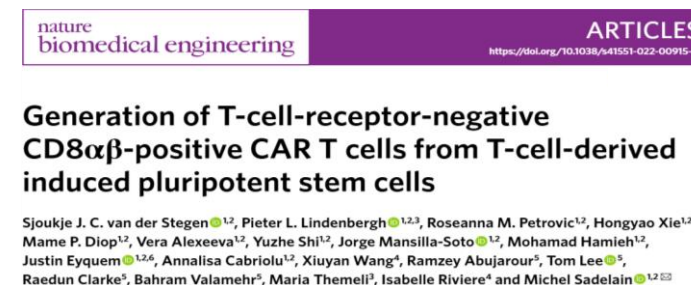
True Off-the-Shelf CAR T-Cell Drug Product



CD19 CAR T-cell designed to eliminate pathological auto-reactive B-cells with balanced efficacy and safety to establish immune reset and clinical remission²

Derived from a defined clonal MCB incorporating unique functional elements to balance safety and efficacy:

- **1XX CAR19:** Novel CAR with CD28 costimulatory and modified CD3z signaling domains for optimal safety and activity
- **TRAC-targeted CAR:** CAR inserted in the T-cell receptor alpha constant (TRAC) locus to reproduce endogenous TCR expression for regulated and optimal function
- **TCR Null:** Complete bi-allelic disruption of TRAC ablates TCR expression and eliminates the possibility of GvHD
- **On-Demand Delivery:** Routinely manufactured at large scale from an engineered MCB that uniquely ensures a uniform, off-the-shelf drug product for broad patient access



1. van der Stegen, S.J.C., et al. Nat. Biomed. Eng 6, 1284–1297 (2022).

2. V. Sandhu, et al. Annals of the Rheumatic Diseases, Volume 84, Supplement 1, 2025, Pages 29-30.

Forte
THERAPEUTICS

The diagram illustrates the timeline for the clinical trial, starting from Study Day -5 to Day 29. Key events include conditioning days (green circles), CAR T infusion (orange circle), and treatment cycle / DLT assessment (orange arrow). Regimen A involves cyclophosphamide or bendamustine, while Regimen B involves no lymphoconditioning. Both regimens include safety and activity assessments up to 2 years and up to 15 years. The diagram also shows the continuation of MMF/MPA or AZA or MTX for Regimen B.

Study Day	Conditioning	Treatment Cycle / DLT Assessment	PTFU	LTFU
-5	○			
-4	○			
-3	○			
-2	○			
-1	○			
1	○	→		
29				

Regimen A: Cyclophosphamide or Bendamustine. Stop all immuno suppressive medication. Safety & Activity Assessments up to 2 years. Safety Assessments up to 15 years.

Regimen B: No lymphoconditioning. Continue MMF/MPA or AZA or MTX. FT819. Safety & Activity Assessments up to 2 years. Safety Assessments up to 15 years.

- 14 -

FT819-102 Patient Cohort Representative of Real-World Patient Characteristics

Essential Patient Features
➤ Thirteen patients enrolled & treated¹ • Ten SLE with fludarabine-free, less-intensive conditioning chemotherapy (Regimen A) • Two SLE without conditioning chemotherapy (on background therapy) (Regimen B) • One SSc with fludarabine-free, less-intensive conditioning chemotherapy (Regimen A)
➤ All SLE patients exhibit high disease burden at baseline • Median disease duration of 8.7 years • Up to 10 prior treatment failures • Median baseline SLEDAI-2K of 14

Baseline Parameter	Overall (N=13) ⁴ Regimen A, n=11; Regimen B, n=2
SSc, n	1
SLE, n – Clinical Characteristics	12
Age, y, mean (range)	30.8 (19-57)
Female, n (%)	11 (92)
SLE duration, y, median (range)	8.7 (1-34)
Prior therapies, n, median (range)	7 (3-10)
Follow-up, month, median (range)	4 (0-19.5)
SLEDAI-2K, median (range)	14 (8-20)
Lupus nephritis, n (%)	7 (58)
UPCr, mean ± SD ³	2.6 ± 1.8
PGA, mean ± SD	2.3 ± 0.4
FACIT-Fatigue score, mean ± SD	23 ± 13

SSc = Systemic Sclerosis; SLE = Systemic Lupus Erythematosus; SLEDAI-2K = Systemic Lupus Erythematosus Disease Activity Index 2000; UPCR = urine protein-to-creatinine ratio; PGA = Physician's Global Assessment; FACIT = Functional Assessment of Chronic Illness Therapy

1. Jelcic, et al. Blood. Vol 146, Sup 1, 2025

2. <https://clinicaltrials.gov/study/NCT06308978> (ClinicalTrials.gov)

3. Applicable to lupus nephritis patients only

4. Nov 25, 2025 data cut off date

Continued absence of high-grade CRS, No ICANS, and No DLTs observed in FT819 treated patients with at least 1 month follow up³

Safety data in line with FT819-101 in B cell malignancies (NCT04629729)

Safety Parameter	Incidence, n (%) Among Patients with ≥1 Month Follow-up (N=10)
CRS, Grade 1-2	3 (30)
CRS, Grade ≥3	0
ICANS/neurotoxicity	0
GvHD	0
DLT	0
Grade ≥3 AEs (any)	3 (30)
Infection, Grade ≥3*	2 (20)
Cytopenia, Grade ≥3 [†]	2 (20)
Hypogammaglobulinemia	0

*Infection includes influenza and urinary tract infection.

[†] Cytopenia includes any PTs of anaemia, leukopenia, neutropenia, neutrophil count decreased, lymphopenia, pancytopenia, and thrombocytopenia.

AE = adverse event; CRS = cytokine release syndrome; DLT = dose-limiting toxicity; GvHD = graft-versus-host-disease;

ICANS = immune effector cell-associated neurotoxicity syndrome;

FT819 Treatment Demonstrates Early and Sustained Improvement in SLE disease

Regimen A: One-time FT819 treatment with less intensive conditioning chemotherapy^{1,2,3}

Durable (≥18 month) SLE disease remission and complete renal response in LN patients with single dose of FT819 with less intensive conditioning^{1,3}

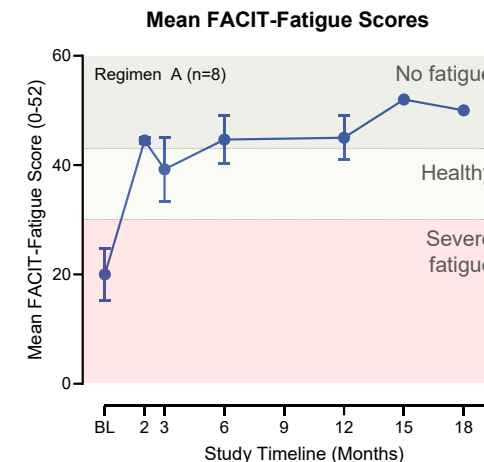
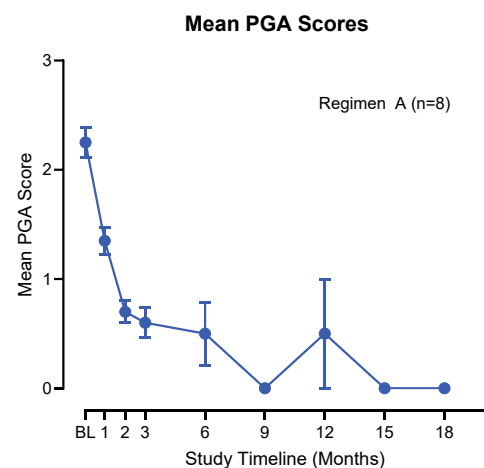
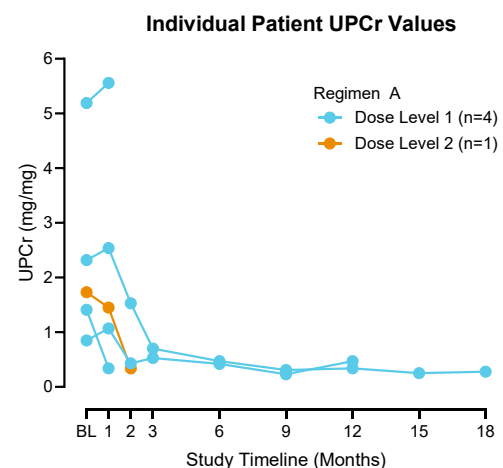
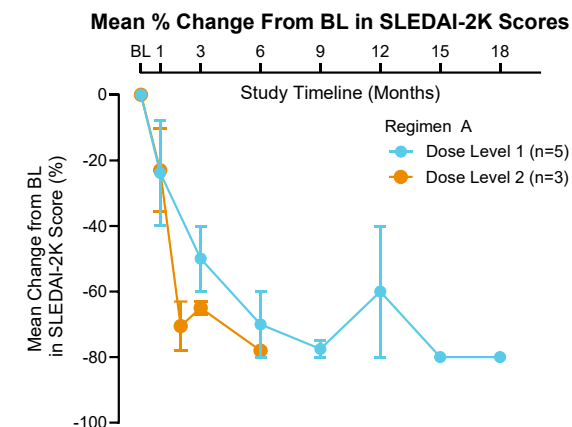
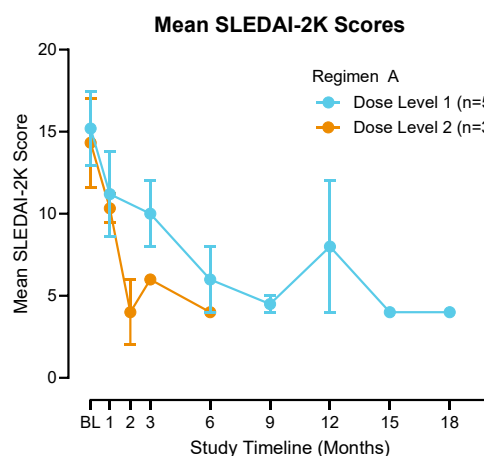
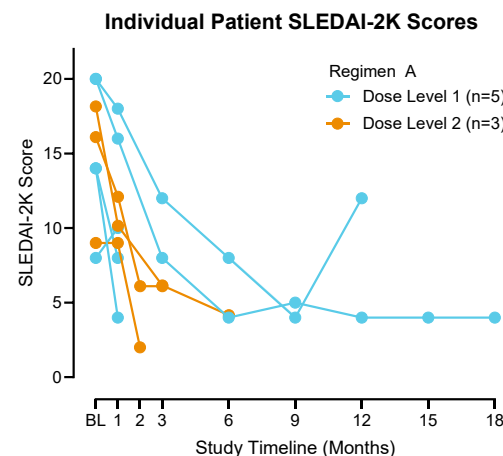
✓ Decrease in disease burden with less intensive conditioning chemotherapy is uniquely demonstrated

✓ Completed dose escalation and now enrolling in DL1 & DL2 expansion

✓ No DLT, CRS, GvHD or ICANS across 2 dose levels

✓ Reduced hospitalization, increased patient accessibility during early dose escalation and expansion³

✓ On-demand CAR T-cell delivery is enabled by scalable manufacturing from a master cell bank



1. Jelcic, et al. Blood. Vol 146, Sup 1, 2025

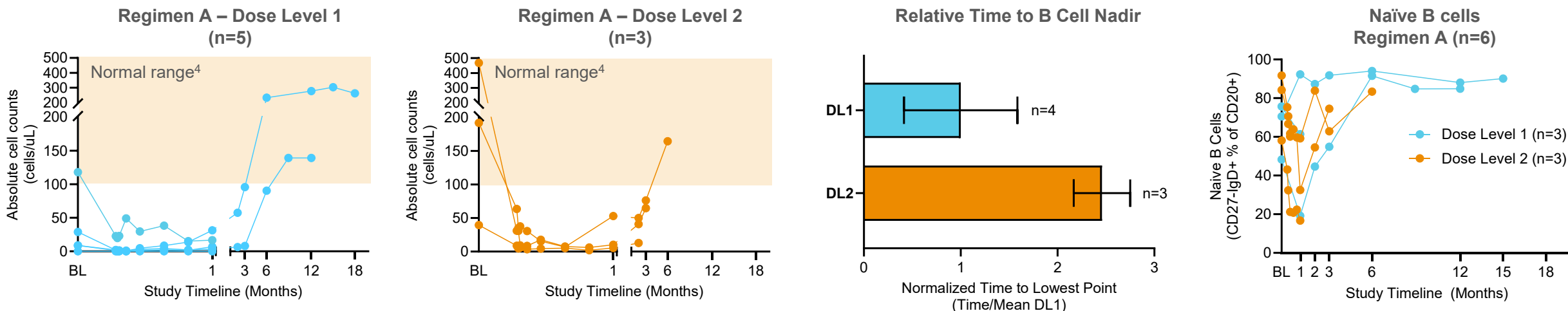
2. <https://clinicaltrials.gov/study/NCT06308978> (ClinicalTrials.gov)

3. Data cutoff 22nd Oct 2025; Error graphed as SEM where appropriate

FT819 Elicits Rapid and Deep Depletion of B Cells and Immune Remodeling

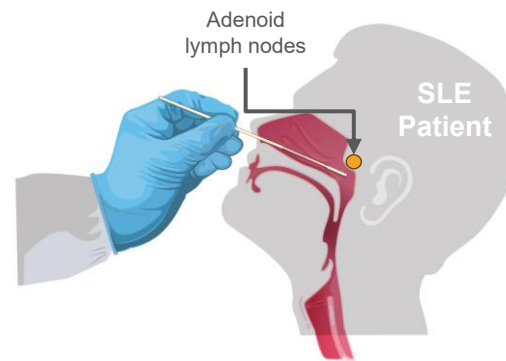
Elimination of B cells in peripheral blood and tissue results in the repopulation of naïve B cell subsets

Effective depletion of pathogenic B cell populations & replacement with naïve subsets to normal levels¹⁻³

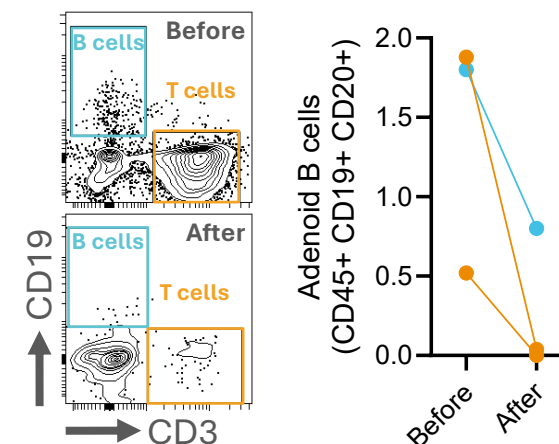


- ✓ Deep and enduring elimination of peripheral blood B cell subsets and plasmablasts populations
- ✓ Clinical demonstration of B cell compartment remodeling followed by the re-emergence of naïve B cell states from bone marrow¹
- ✓ Complete B cell elimination in nasopharyngeal lymphoid tissues within 2 weeks following DL2 treatment with FT819 (N=2)

Nasopharyngeal adenoid B cell collection technique



Lymphoid tissue residing B cell elimination post FT819 treatment



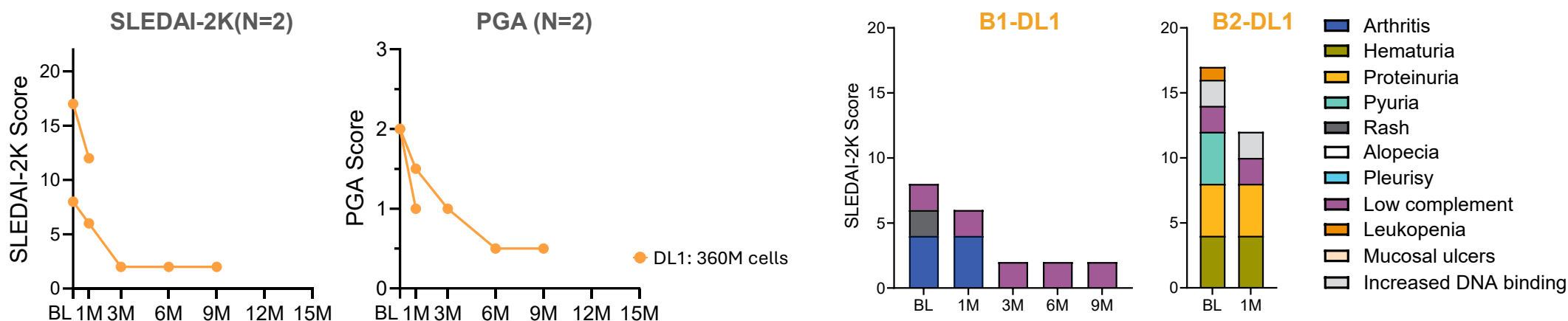
1. Jelcic, et al. *Blood*. Vol 146, Sup 1, 2025
2. <https://clinicaltrials.gov/study/NCT06308978> (ClinicalTrials.gov)

3. Data cutoff 22nd Oct 2025
4. Morbach H et al. Reference values for B cell subpopulations from infancy to adulthood. *Clin Exp Immunol*. 2010 Nov;162(2):271-9.

Sustained Response in SLE Achieved Without Lympho-Conditioning

Regimen B: FT819 (DL1) as add-on to maintenance therapy exhibit improvement in disease activity

SLEDAI-2K composition scores per patient at baseline (pre-FT819) and at specified study time points¹⁻⁴



BL = baseline; D = Day; DL= dose level; M = month; SLEDAI-2K = Systemic Lupus Erythematosus Disease Activity Index 2000. PGA = Physician global assessment. Data cutoff date 25th September 2025

- ✓ Two patients treated with a one-time dose of FT819 (DL1; 360M) as an add on therapy show disease activity improvement absent of any conditioning chemotherapy
- ✓ B cell elimination and clonal repertoire remodeling in the absence of lympho-conditioning
- ✓ No DLT, CRS, GvHD or ICANS as of data cutoff.

Preliminary Efficacy in SSc Patient Following FT819 Treatment

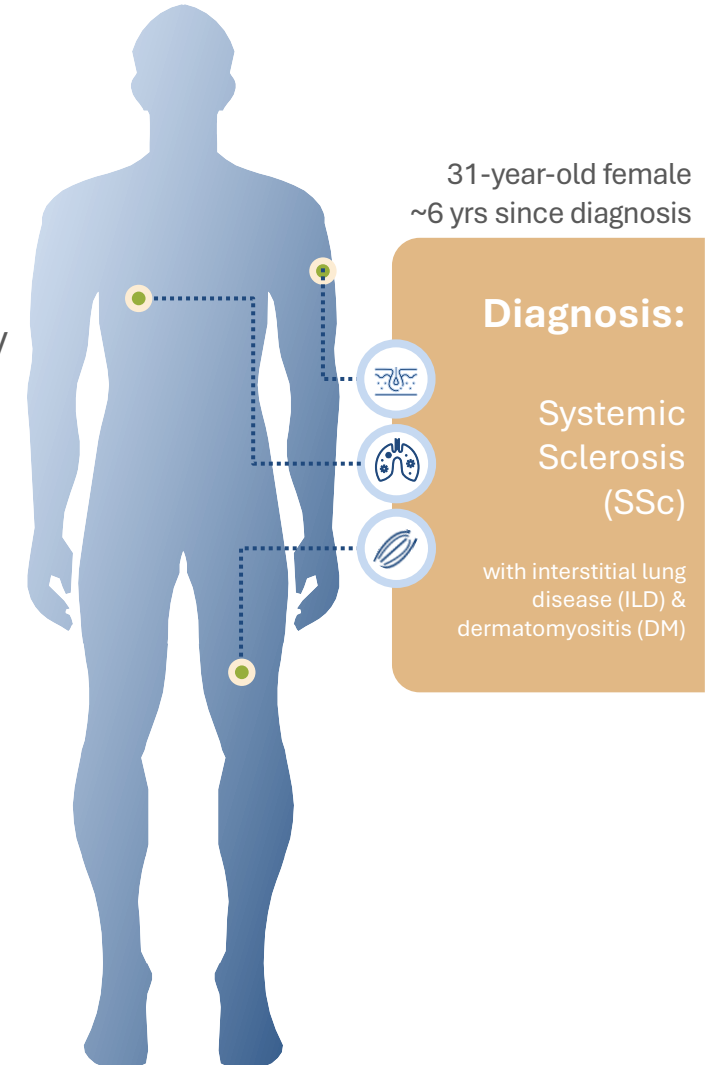
First SSc patient with 3-month follow-up shows meaningful disease improvement using less-intensive conditioning chemotherapy

Systemic Sclerosis (SSc) Disease

- Patients with severe systemic sclerosis (SSc) can face high morbidity, early mortality, and limited treatment options¹
- No current FDA licensed therapy, except Tocilizumab and Nintedanib in SSc associated ILD¹
- Immunosuppressant refractory patients represent a well-defined, high-risk subgroup suitable for transformative therapy
- Novel therapies that provide deep clinical responses could support accelerated pathways
- Case-level efficacy signals can justify rapid transition from proof-of-concept to registrational strategy

Patient Case Study²

- 31Y/F with interstitial lung disease (ILD) and dermatomyositis (DM)
- 6Y since original diagnosis
- Refractory to 7 prior therapies
- ✓ Received one time dose of FT819 with less-intense fludarabine- free conditioning
- ✓ No CRS, GvHD, or ICANS observed
- ✓ mRSS = 22 at baseline, dropping to 15 at 3 months evaluation time point
- ✓ Patient also experienced meaningful improvements in overall disease severity vs baseline (CGA 6 → 2; PtGA 5 → 0)
- ✓ Meaningful real-world benefits noted in physical function and daily activities at 3M evaluation time point (SHAQ score 0)



mRSS= Modified Rodnan Skin Score; CGA = Clinical Global Assessment; PtGA = Patient Global Assessment; SHAQ = Scleroderma Health Assessment Questionnaire

1.C. Denton et al. The 2024 British Society for Rheumatology guideline for management of systemic sclerosis, *Rheumatology*, Volume 63, Issue 11, November 2024, Pages 2956–2975

2. Data cut-off 9th Feb 2026

FT819 Product Candidate Executive Summary & Next Steps

Strategic highlights and value drivers

Differentiated Off-the-Shelf CAR T cell

- True off-the-shelf CAR T cell, consistently manufactured from a precisely engineered master cell bank, that addresses the key challenges faced by autologous, allogenic and in vivo re-programming approaches.

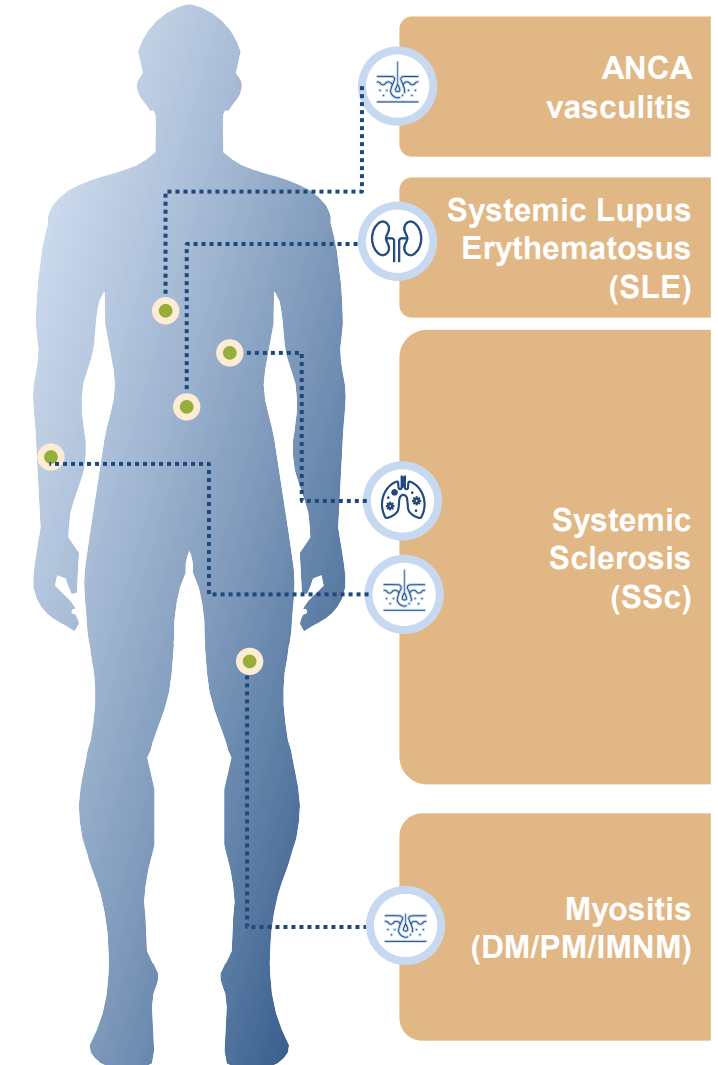
Clinical & Regulatory Advancement

- Recent reported data¹ from 12 SLE patients treated with FT819 showed progressive and durable reductions in disease activity, including mean SLEDAI-2K score reductions of up to 78% by six months from the dose level 2 (DL2) cohort, complete renal responses in lupus nephritis, and sustained B-cell depletion with immune remodeling.
- > 60 patients treated across FT819-101 (B cell malignancies) & FT819-102 (autoimmune) programs; Favorable safety with no ICANs, GvHD or CRS > Grade 2, underscoring a differentiated safety profile supporting same day outpatient administration.

Regulatory & Operational Development

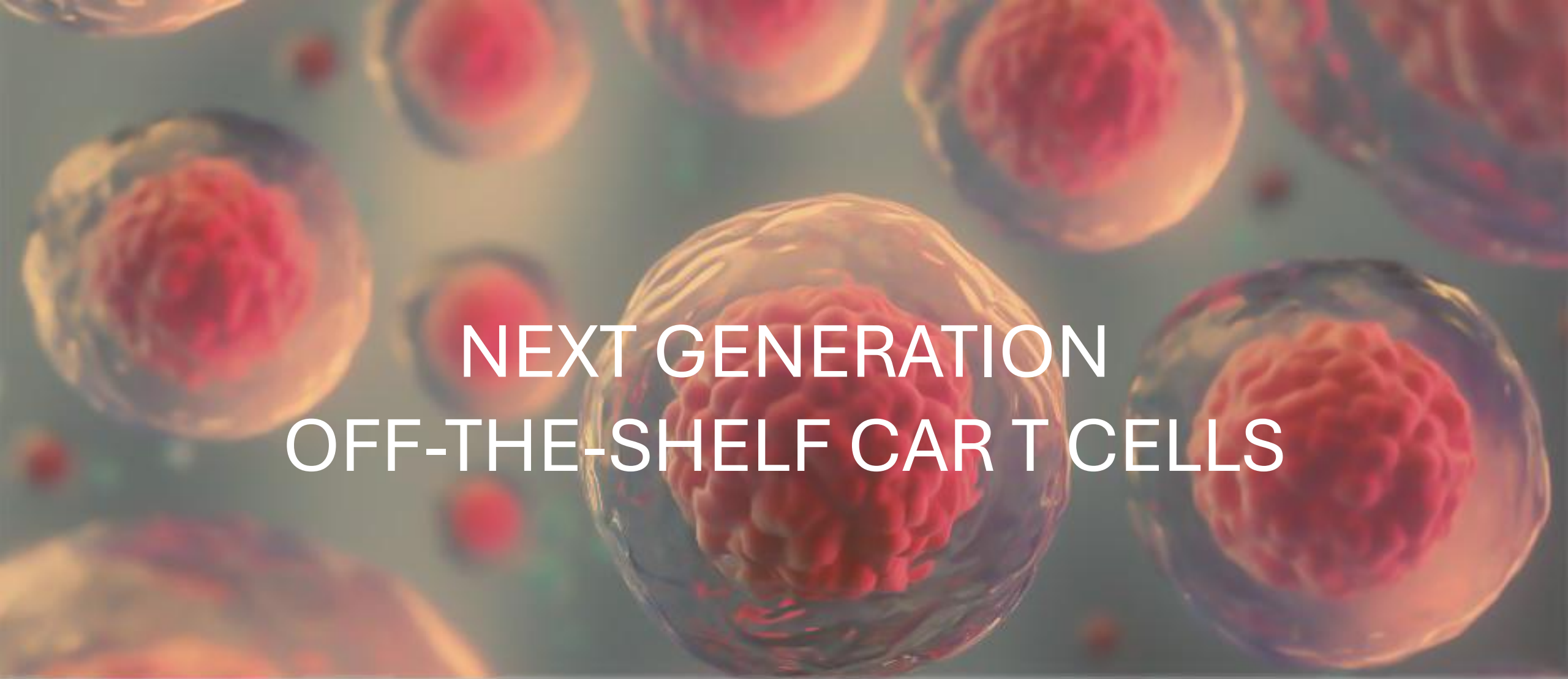
- Ongoing Ph1 trial in ANCA vasculitis, myositis, SLE, and SSc (ages 12-70 years); allowing redosing after relapse or inadequate response.
- 15 SLE patients treated across 7 initial sites, 4 SSc and 1 IIM patients treated as part of cohort expansion².
- 16 total clinical sites now activated (academic and community sites)².
- Engaged with FDA under RMAT designation to align on SLE LN pivotal trial and protocol strategy to commence in 2H 2026.

Current **FT819** Disease Indications



1. Jelcic, et al. Blood. Vol 146, Sup 1, 2025 (ASH December 2025 Presentation)

2. As of data cut-off 25th February



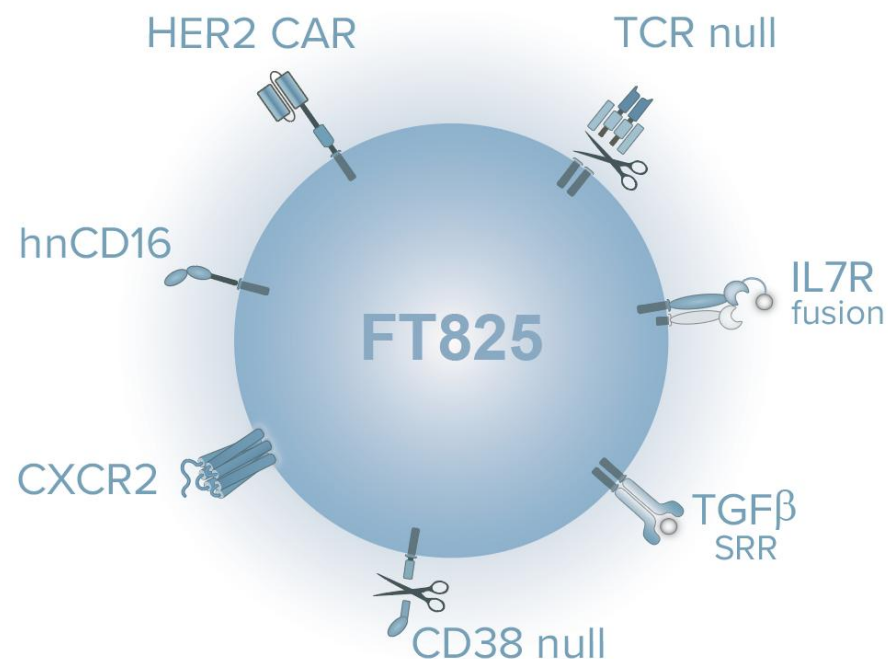
NEXT GENERATION OFF-THE-SHELF CAR T CELLS



Making Cell Therapy Accessible to All™

Seven-Point Edited HER2-Directed CAR T-Cell Therapy Designed for Enhanced Solid Tumor Efficacy

FT825/ONO-8250: Off-the-shelf anti-HER2 CAR T-cell product candidate



HER2-targeted CAR T-cell designed to overcome tumor heterogeneity, improve cell trafficking, and resist tumor microenvironment mediated immune suppression

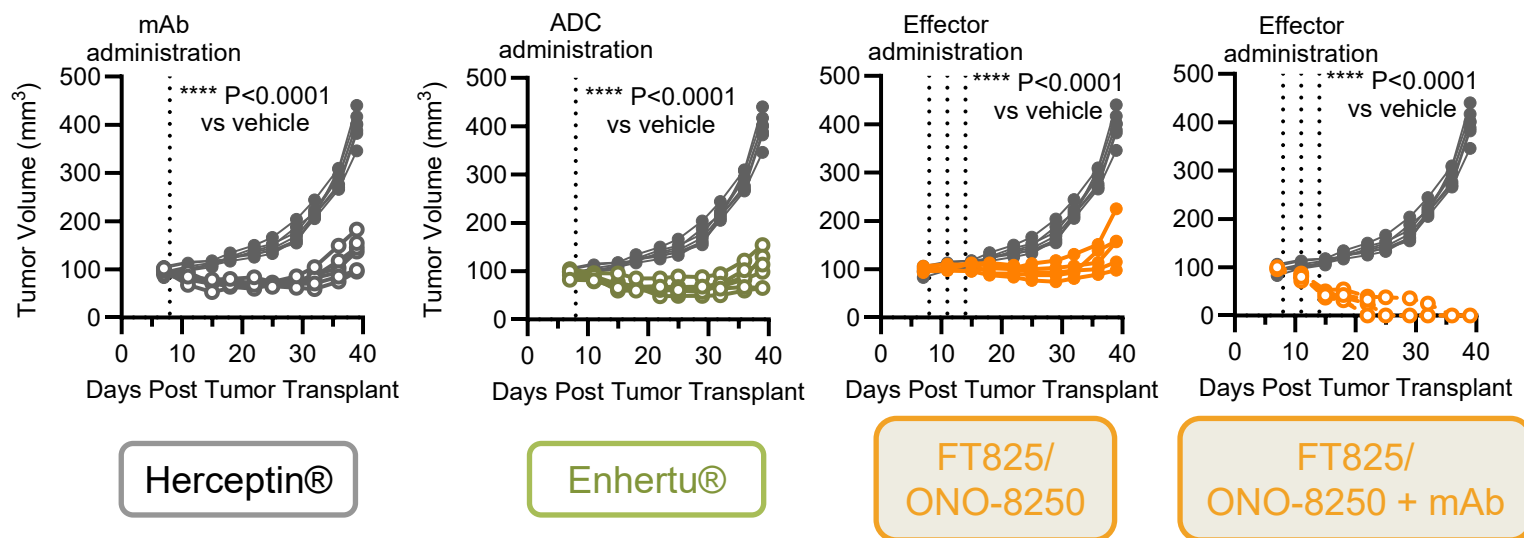
Overcoming the Challenges in Solid Tumors

- **TCR Null**: Complete bi-allelic disruption of TRAC ablates TCR expression and eliminates the possibility of GvHD
- **Novel HER2-Directed CAR**: Potent and preferential targeting of tumor cells expressing HER2 with H₂CasMab-2 CAR expression and optimized for enhanced activity
- **hnCD16**: Enables ADCC in combination with therapeutic monoclonal antibodies to complement CAR to overcome tumor heterogeneity through multi-antigen targeting
- **TGFβ-SRR**: TGFβ signal redirection receptor provides resistance to TGFβ-mediated suppression commonly found in TME of solid tumors
- **CXCR2**: Enhancement of migration into solid tumors
- **IL7RF**: Enhances CAR T cell, potency, persistence and self-renewal
- **CD38 KO**: Potential to enhance metabolic cell fitness

Novel Cancer-Specific CAR Binder Limits Off Tumor Toxicity

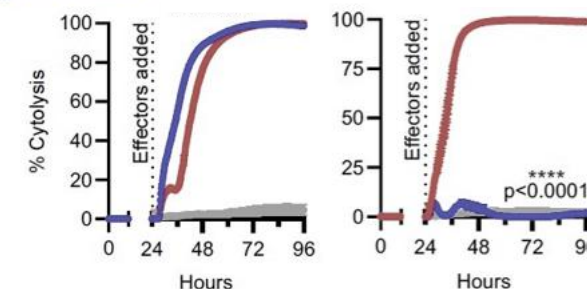
FT825/ONO-8250 designed for preferential and multi-antigen targeting

- Novel binder (H₂CasMab-2) preferentially targets HER2 expressed on tumor cells with limited on-target off-tumor toxicity
- FT825/ONO-8250 shows flexible multi-antigen targeting via enhanced antibody-directed cellular cytotoxicity (ADCC)



HER2⁺⁺⁺ Tumor Tissue HER2⁺ Normal Tissue

Herceptin®
H₂CasMab-2

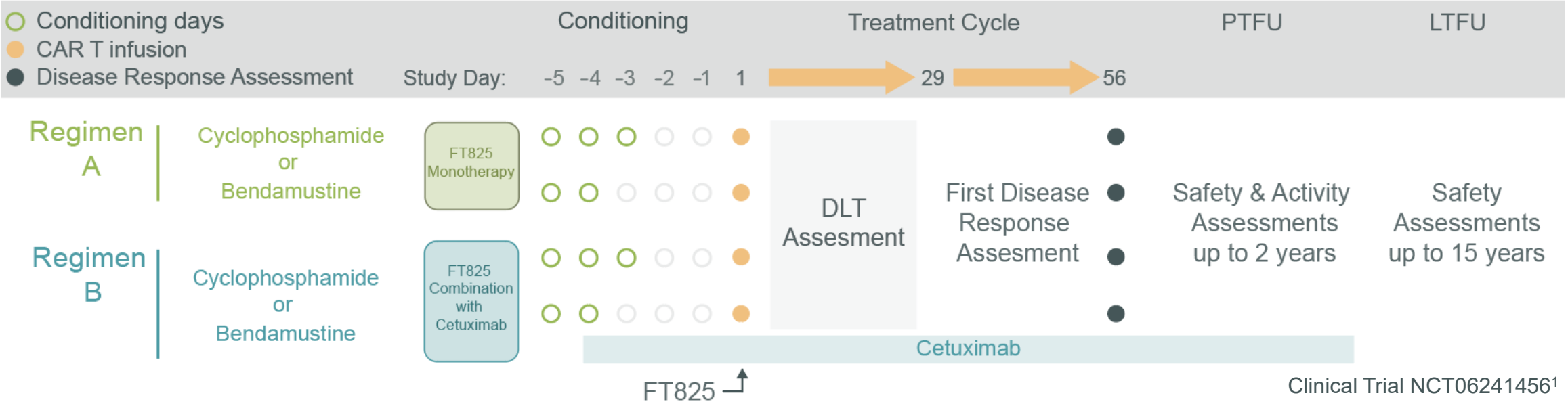


	Herceptin®	H ₂ CasMab-2
On Target On Tumor	✓	✓
On Target Off Tumor ²	✗	✓

In contrast to Herceptin® H₂CasMab-2 shows limited On Target-Off Tumor toxicity

FT825/ONO-8250-101: A Phase 1 Study of FT825 in Advanced Solid Tumor

Phase 1 Study: FT825/Ono0825 with/without monoclonal antibody combination (NCT06241456)



Regimen A: FT825 Monotherapy	- HER2⁺ Breast, Gastric, GEJ, HER2^{Mut} NSCLC - HER2⁺ salivary, endometrial, other cancers	Early Clinical Observations
		- Dose levels 1 and 2 have been evaluated. Enrollment is ongoing at Dose Level 3 - No dose-limiting toxicities to date - Preliminary observations at the lower doses indicate clinical activity, including stable disease (per RECIST v1.1)
Regimen B: FT825 Combination with Cetuximab	- Colorectal (KRAS WT or BRAF V600E) - NSCLC (EGFR^{Mut}) - HNSCC	

Engineering a Portfolio of Attributes to Unlock Multi-Disease Therapy Potential

Integrating modular attribute cell systems to operate & synergize with the patients' immune system

Overcoming Multiple Challenges Across Diverse Tumors Indications

Problem Statement

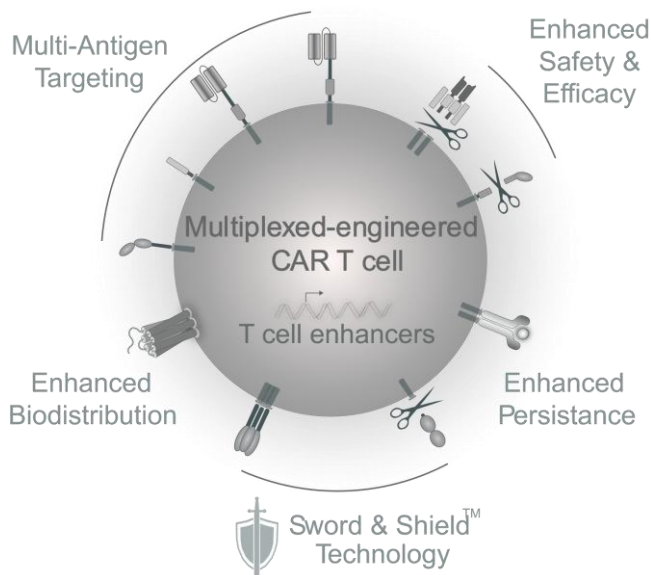
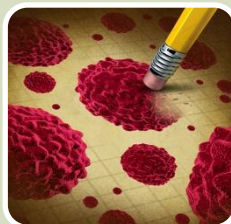
- Most tumors lack distinct lineage markers, making it difficult to distinguish tumor from healthy tissue.
- Single mechanism therapies often drive immune escape, enabling resistant/refractory tumor variants.
- Tumor microenvironments suppress immune function and impede cell access, creating zones of immune exclusion.

Proposed Solution(s)

- ✓ Target disease or altered self markers to enable cell specific killing across diverse tumor types/pathological settings.
- ✓ Deploy multiplex targeting to apply simultaneous immune pressure via distinct mechanisms of action.
- ✓ Navigate immune suppressive niches and convert inhibitory cues into immune activating signals.

Engineered Attribute System(s):

- ✓ Single and/or multi-CAR systems targeting MICA/B, B7-H3 & others
- ✓ High affinity non cleavable CD16 (hnCD16)
- ✓ TGFβ signal redirect receptor (TGFβ SRR)
- ✓ Synthetic CXCR2 & endogenous trafficking receptors
- ✓ Allo-Defense Receptor (ADR) & CD58 KO Synapse Engineering
- ✓ T Cell Enhancers



Broad Elimination of Pathological Immune Cell Subsets & Compartments

Problem Statement

- Autoimmune, inflammatory diseases & hematological malignancies arise from dysregulated T, B and myeloid cell function across secondary and tertiary immune sites.
- Current therapies offer broad immune suppression or narrowly target specific cells, frequently falling short of effective immune control.

Proposed Solution(s)

- ✓ Multiplex targeting of lineage and/or activation markers enables selective elimination of pathogenic cells whilst minimizing broad immune suppression and its associated risks.
- ✓ Deploy multiplex targeting to apply simultaneous immune pressure via distinct mechanisms of action.
- ✓ Navigate immune suppressive niches and convert inhibitory cues into immune activating signals.

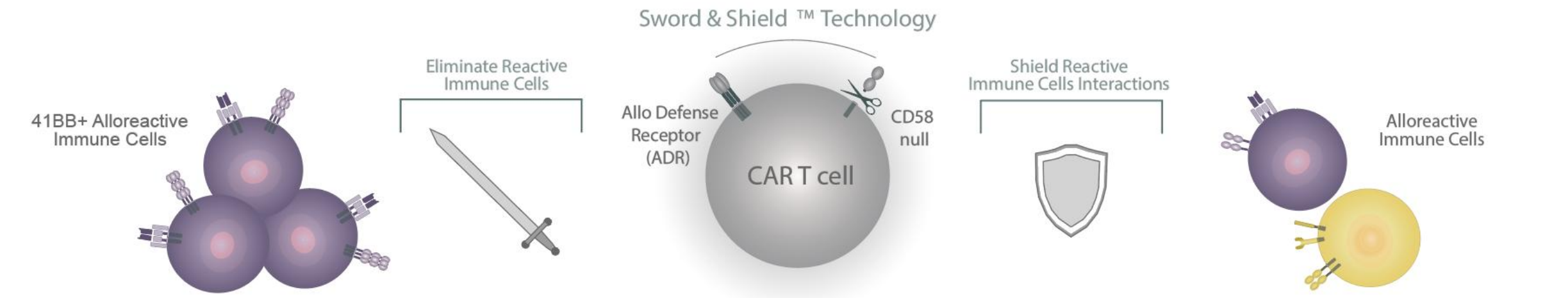
Engineered Attribute System(s):

- ✓ Single and/or multi-CAR systems targeting CD19, BCMA & CD38
- ✓ High affinity non cleavable CD16 (hnCD16) & CD3 Fusion Receptor
- ✓ TGFβ signal redirect receptor (TGFβ SRR)
- ✓ Synthetic CXCR2 & endogenous trafficking receptors
- ✓ Allo-Defense Receptor (ADR) & CD58 KO Synapse Engineering
- ✓ T Cell Enhancers



Sword & Shield™ Technology Shields from Rejection & Drives Persistence

Best-in-class allo-immune evasion system to enhance persistence & eliminate the need for lympho-conditioning



Strategy	Combination with Intense CCT	HLA-I & HLA-II Knockout	HLA-I & HLA-II Knockout + HLA-E ¹	HLA-I & HLA-II Knockout + CD47 ^{2,3}	Sword & Shield™ ADR ⁴ + CD58 Knockout ⁵
Avoid host CD8 T cells	+	+	+	+	+++
Avoid host CD4 T cells	+	+	+	+	+++
Avoid host NK cells	+	-	+/-	+/-	+++
Avoid host Treg suppression	+	-	-	-	+++
Induce proliferation	+	-	-	-	+++
Lymphodepletion	+	-	-	-	+++
Avoid toxicity associated immunosuppression	X	✓	✓	✓	✓

1. Li W et al. *Front Immunol.* 2022 Dec 2;13:1052717.

2. Hu, X., et al. *Nat Biotechnol* 42, 413–423 (2024).

3. Hu X, et al. *Nat Commun.* 2023 Apr 10;14(1).

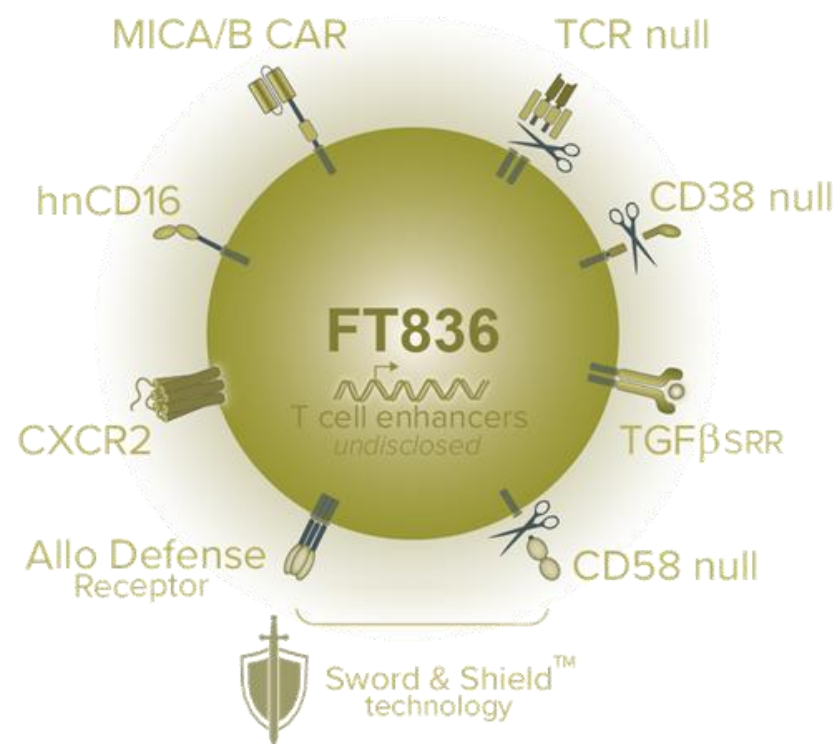
4. Mo F et al, *Nat Biotechnol.* 2021 Jan; 39(1):56-63.

5. Hamer Q et al. *Cell Stem Cell.* 2024 Sept 5;31(9):1376-1386.e8.

FT836: Off-the-Shelf Anti-MICA/B CAR T Cell Product Candidate

Next-gen ADR-armed CAR T cell targets stress ligands MICA/B to eliminate a broad spectrum of tumors

True Off-the-Shelf Next Generation
Multi-Antigen Targeting CAR T Cell Drug Product



Multi-antigen targeting via MICA/B CAR and hnCD16,
with ADR technology designed to reduce/eliminate
need for conditioning chemotherapy

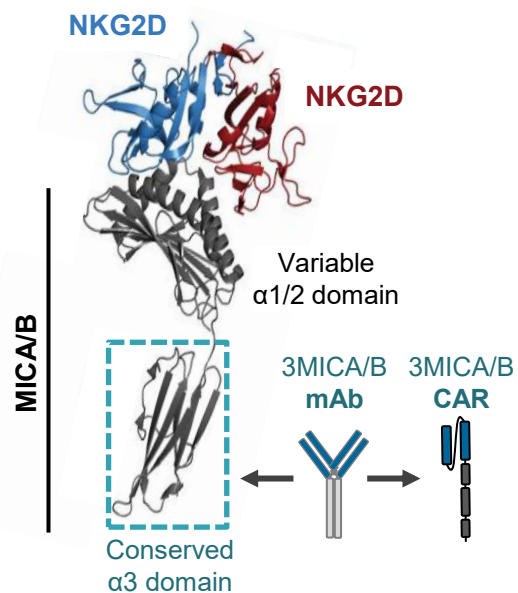
Product Candidate Engineered Attribute Systems

- **MICA/B CAR:** An innovative CAR targeting strategy that uniquely distinguishes tumors from healthy tissue
- **hnCD16:** Enables ADCC when combined with therapeutic monoclonal antibodies to complement CAR specificity to provide additional antigen / target cell coverage
- **Sword & Shield™ (ADR/CD58 null):** An Allo-Defense Receptor that targets activated allo-reactive T-cells; CD58 null to impede allo-reactive T cell recognition & binding
- **T Cell Enhancer(s):** Optimized regulation of cell persistence & potency
- **Enhanced biodistribution & microenvironment performance:** Engineered chemokine receptor surface profile to augment tissue trafficking; enhanced performance in inhibitory microenvironments via TGFβ signal redirection receptor (SRR)
- **TCR Null:** Complete bi-allelic disruption of TRAC ablates TCR expression and eliminates the possibility of GvHD
- **CD38 Null:** Potential to enhance metabolic cell fitness and allow combination with CD38 targeting mAbs

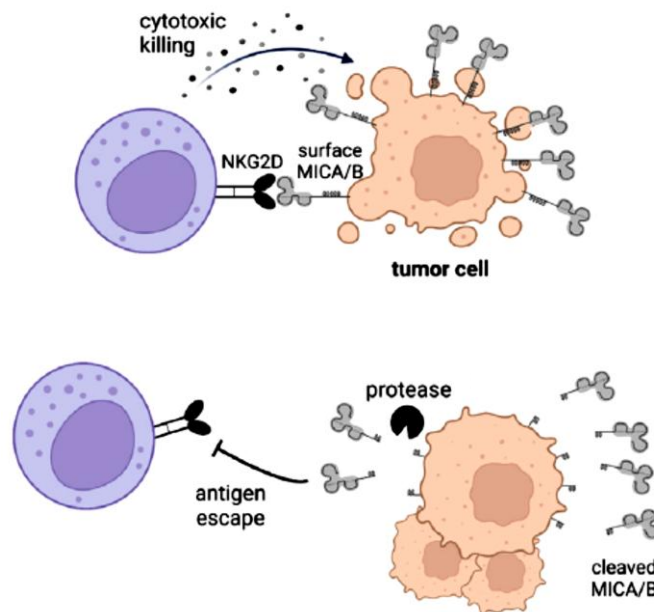
Targeting MICA/B Overcomes Tumor Evasion & Unlocks Pan-Tumor Potential

Stress-ligand recognition of diseased cell states permits indication-agnostic therapy

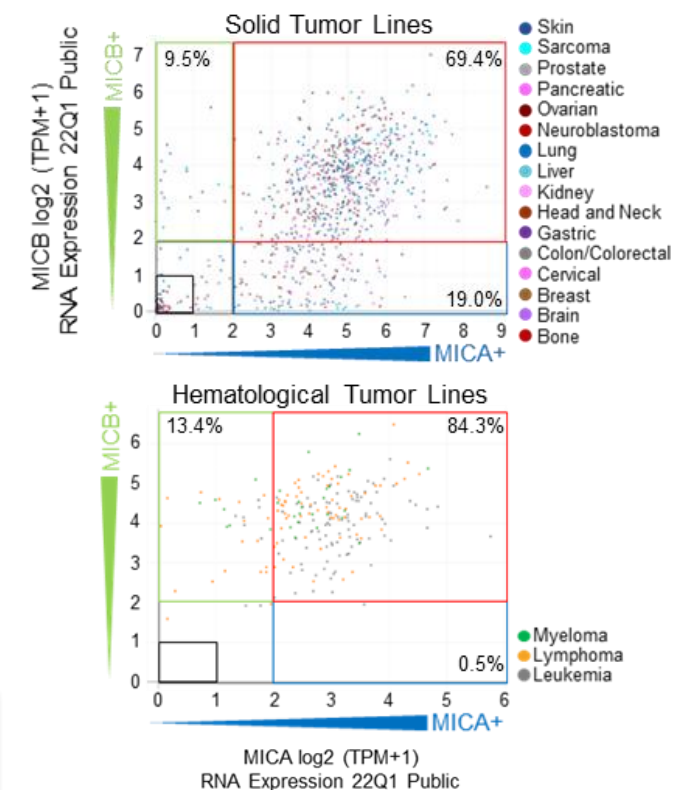
Novel recognition of MICA/B $\alpha 3$ domain unlocks pan-tumor recognition^{1,2}



MICA/B shedding is a common immune evasion mechanism utilized by cancer^{3,4}



MICA/B is widely expressed cross multiple cancer indications⁵



CANCER IMMUNOLOGY

Antibody-mediated inhibition of MICA and MICB shedding promotes NK cell-driven tumor immunity

Lucas Ferrari de Andrade,^{1,2} Rong En Tay,^{1,2} Deng Pan,^{1,2} Adrienne M. Luoma,^{1,2} Yoshinaga Ito,^{1,2} Soumya Badrinath,^{1,2} Daphne Tsoucas,² Bettina Franz,^{1,2} Kenneth F. May Jr.,⁴ Christopher J. Harvey,⁵ Sebastian Kobold,¹ Jason W. Pyrdol,¹ Charles Yoon,^{2,4} Guo-Cheng Yuan,² F. Stephen Hodi,⁴ Glenn Dranoff,^{1,4} Kai W. Wucherpfennig^{1,2,4}

Med

Clinical and Translational Resource and Technology Insights

A chimeric antigen receptor uniquely recognizing MICA/B stress proteins provides an effective approach to target solid tumors

John Goulding,^{1,4,5} Wen-I Yeh,¹ Bryan Hancock,¹ Robert Blum,¹ Tianhao Xu,¹ Bi-Hui Yang,¹ Chia-Wei Chang,¹ Brian Grotz,¹ Earl Avramis,¹ Mochtar Pihlbad,¹ Yijia Pan,¹ Hui-Yi Chu,¹ Shih-Hsin Sitaronski,¹ Lauren Fong,¹ Nicholas Brookhouse,¹ Thomas Dailly,¹ Miguel Meza,¹ Matthew Dornholtz,¹ Evelyn Diaz,¹ Judy Martin,¹ Peter Szabo,¹ Sarah Cooley,¹ Lucas Ferrari de Andrade,² Tom T. Lee,¹ Ryan Bjordahl,¹ Kai W. Wucherpfennig,^{1,4,5} and Bahram Valamehr^{1,4}

CellPress

Viewpoint

Can't drop the MIC(A/B): Preventing stress-ligand shedding to enhance pan-cancer targeting

Nora Lakes,^{1,2,3,5} Laura M. Canaday,^{1,2,5} and Stephen N. Waggoner^{1,2,3,4,*}

Journal of Cancer Biology

Turn up the MIC(A/B): Amplifying stress-ligand recognition to target iPSC derived immune cell therapies against tumors and diseased cell states

John Goulding,¹ Alejandro J. Garcia,¹ Martin R. Hosking,¹ Peter Szabo,¹ Bahram Valamehr¹

1. Ferrari de Andrade, L. *Science*. 2018 Mar 30;359(6383):1537-1542.

2. Goulding J et al. *Cell Med*. 2023 Jul 14;4(7):457-477.

3. Lakes, N. et al *Cell Med*. 2023 Jul 14;4(7):398-400

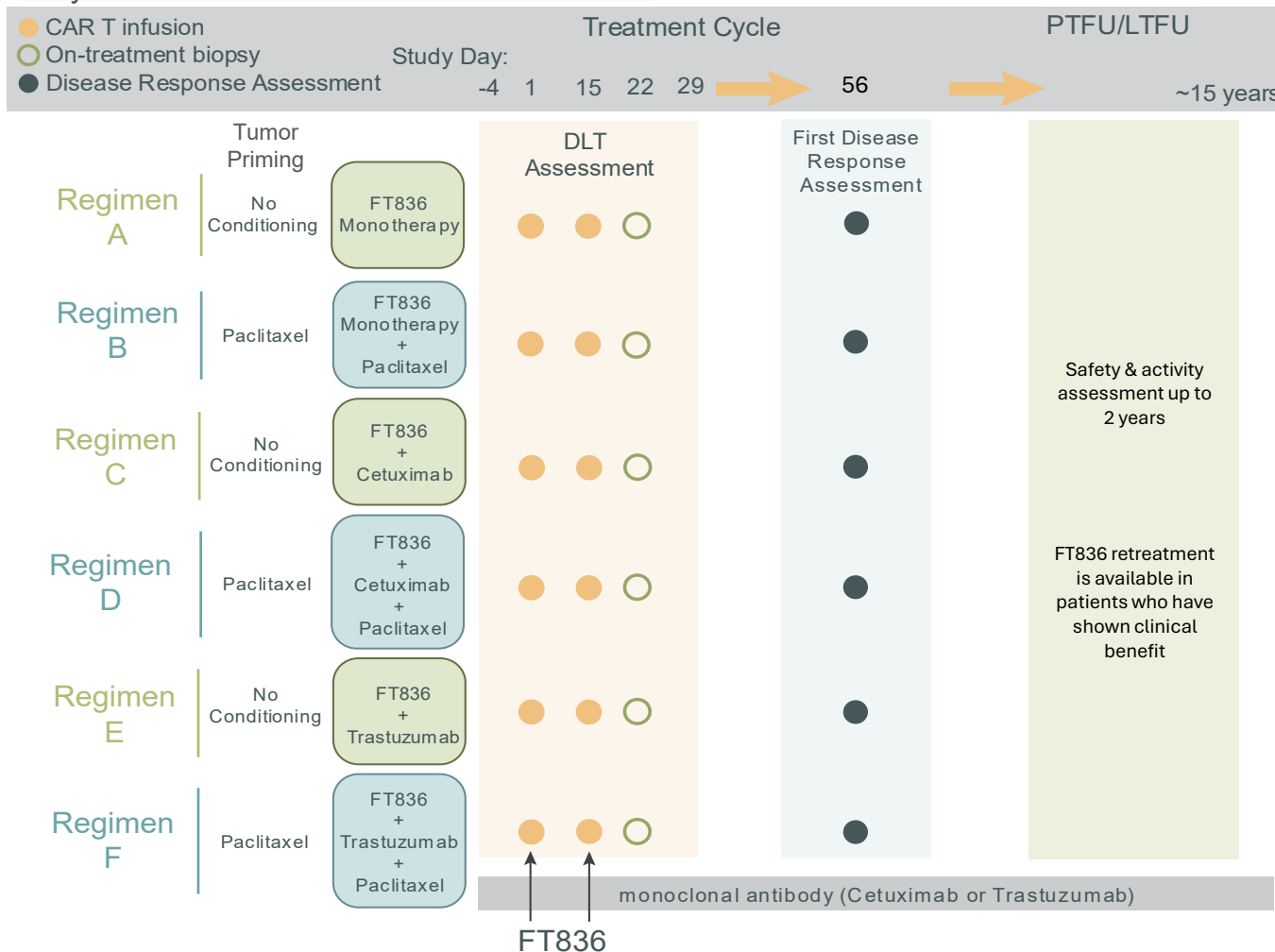
4. Goulding J et al. *J Cancer Biol*. 2023;4(2):49-53.

5. Dhar P et al. *Curr Opin Immunol*. 2018 Apr;51:55-61

Phase 1 FT836-101 Study in Pan Solid Tumor Open to Enrollment¹

Evaluation of FT836 with/without monoclonal antibody combination with optional tumor priming therapy

FT836 Clinical Study Schema



FT836: A Highly-Differentiated Therapeutic Approach

- Novel cancer antigen targeting system that uniquely distinguishes tumor from healthy tissue.
- Combining with HER2 and EGFR targeting mAbs augments activity & allows heterogenous tumor targeting to minimize tumor escape.
- Contains multiplex engineered cellular systems to enhance persistence and cancer killing capability; traffic to and operate within suppressive tumor environments; whilst simultaneously avoiding T cell exhaustion.
- Requires no patient apheresis and no lympho-conditioning chemotherapy.
- Delivered with a shortened hospitalization requirement and can be re-dosed in patients who have shown clinical benefit
- Protocol authorized solid tumors include;
 - Breast (BC); Colorectal (CRC); Ovarian (OVC); Head & Neck (HNSCC); Endometrial (EC); Gastric (GEJ) & Lung (NSCLC).

Clinical Trial NCT07216105¹

1. <https://clinicaltrials.gov/study/NCT07216105> (ClinicalTrials.gov).

FT836: Next Generation Pan Tumor CAR T-cell Therapy

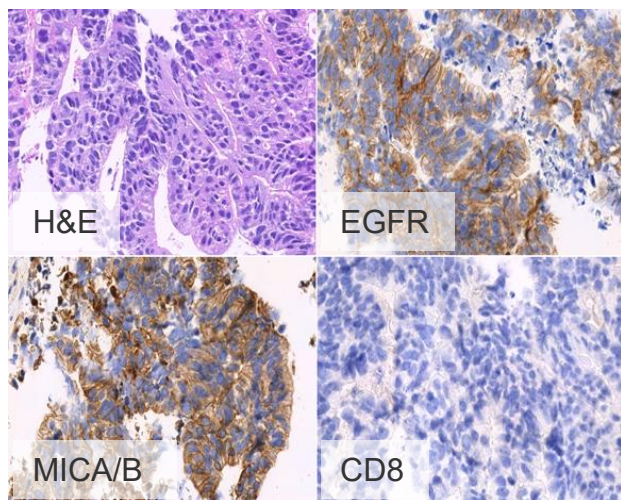
Early evidence of potential anti-tumor activity

Patient Case Study¹:

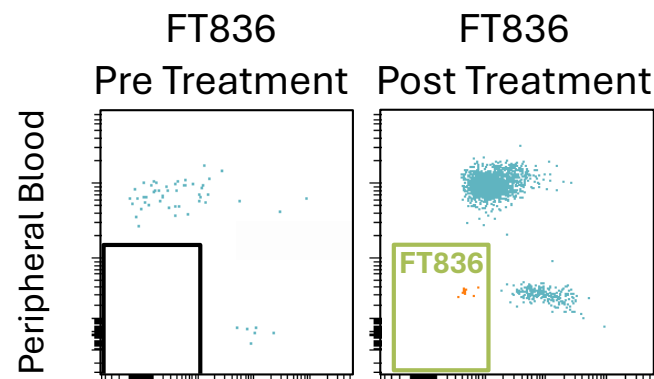
Heavily pretreated 45/M with stage IV refractory metastatic colorectal carcinoma (CRC) with prior cetuximab treatment failure, was dosed with 1 cycle of FT836 in combination with cetuximab without receiving any lympho pre-conditioning chemotherapy

- ✓ No CRS, ICANS or DLTs observed
- ✓ Confirmed MICA/B and EGFR expression in tumor bearing lymph node pre-FT836 treatment
- ✓ FT836 drug product detected in blood post FT836 treatment
- ✓ Demonstrated decrease in CEA and LDH correlated with total mass reduction in all target lesions by ~20% by week 7

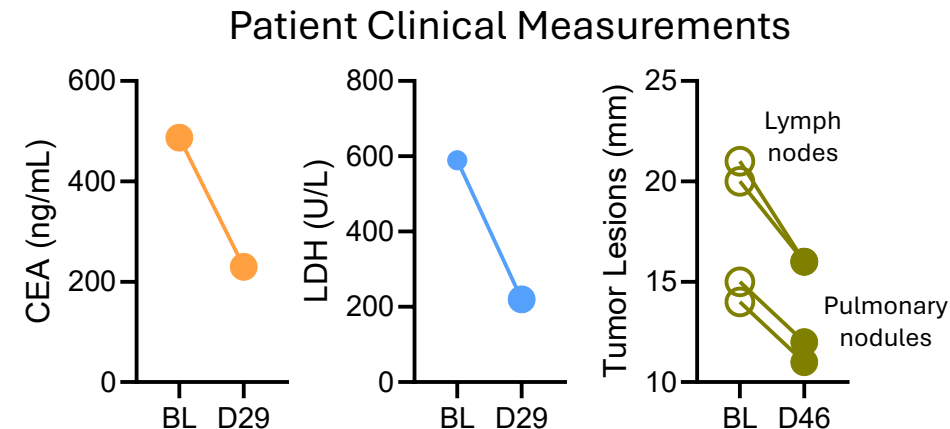
Patient Tumor Antigen Expression at Screen



Live FT836 Cells Detected in Peripheral Blood



Early Evidence of FT836 Anti-Tumor Activity



FT836 Product Candidate Executive Summary & Next Steps

Strategic highlights and value drivers

Novel CAR targeting system

- **MICA/B** is a broadly expressed stress antigen relevant to multiple tumor types and other diseases
- **Targeting MICA/B α3 domain** uniquely overcomes tumor evasion mechanisms of escape

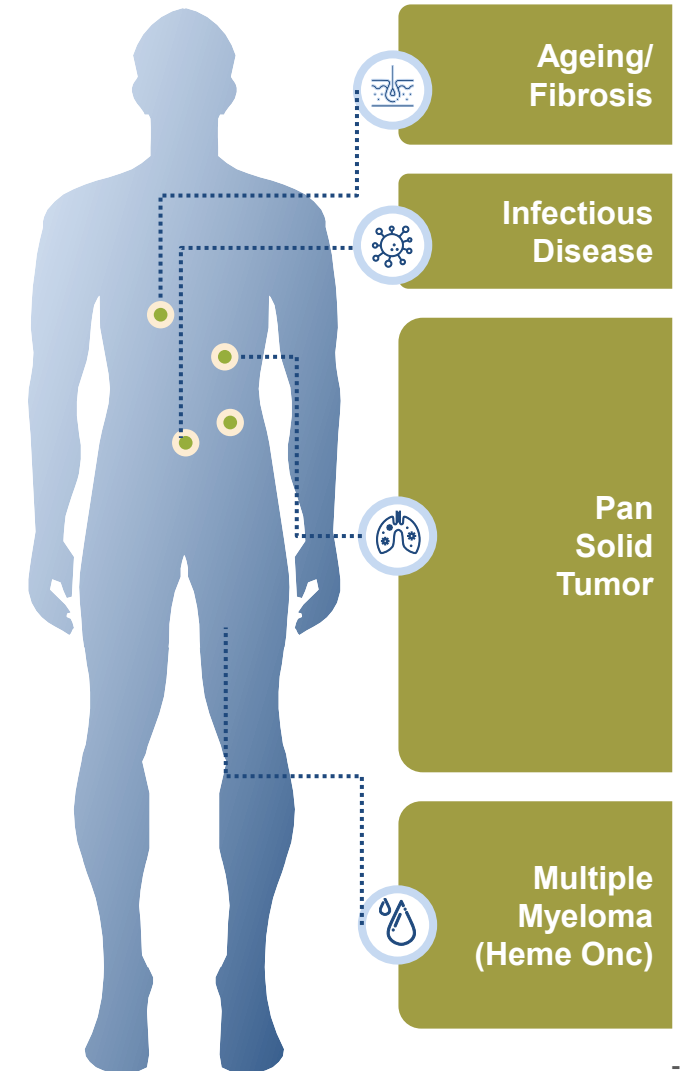
Next Gen CAR T-Cell

- **Multiple functional** edits to enhance potency, persistence, trafficking and multi-antigen targeting
- **Sword and Shield™** technology to provide allo-protection

Clinical Advancement

- **3 patients** enrolled in FT836-101 Ph1 pan-solid-tumor trial
- **Multiple myeloma** IIT initiated, with FPI anticipated in Q1 2026

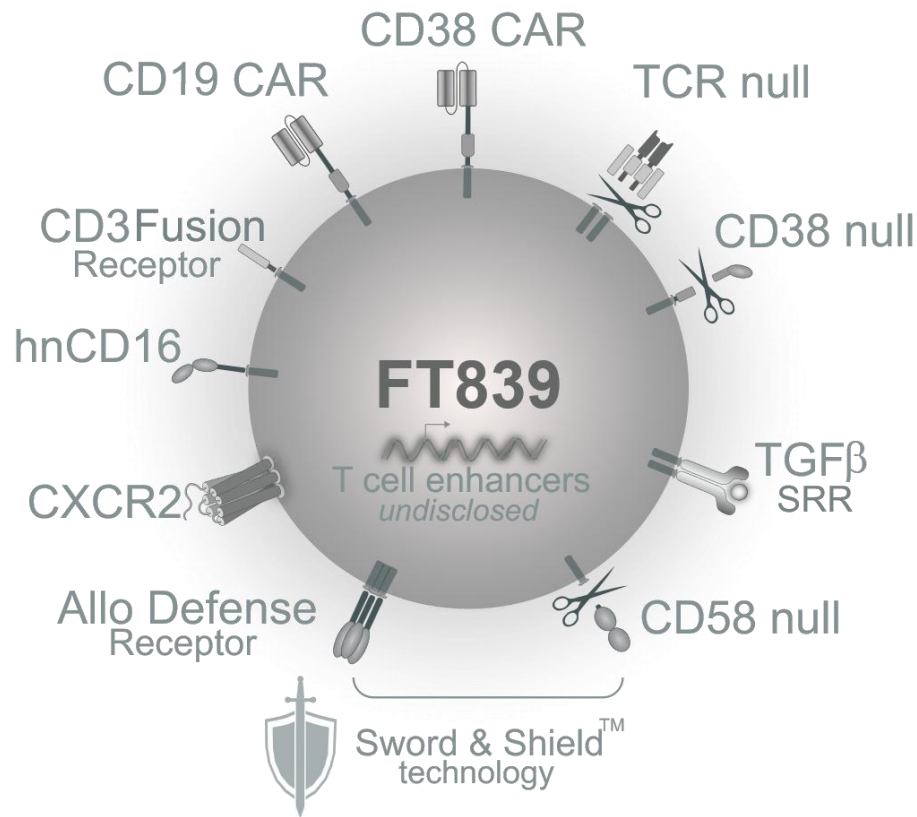
Possible FT836 Disease Indications



FT839: Off-the-Shelf Anti-CD19 & Anti-CD38 CAR T Cell Product Candidate

Next-gen ADR-armed CAR T cell targets select immune cell subsets to unlock multi-disease therapy potential

True Off-the-Shelf Next Generation Multi-Antigen Targeting CAR T Cell Drug Product



Multi-antigen targeting via CD19 CAR, CD38 CAR, hnCD16 and CD3FR, with ADR technology designed to reduce/eliminate need for conditioning chemotherapy

Product Candidate Engineered Attribute Systems

- **1XX CD19 CAR:** Novel CAR with CD28 costimulatory and modified CD3ζ signaling domains for optimal safety and activity
- **CD38 CAR:** A unique targeting strategy to eliminate activated and/or senescent immune cell subsets
- **Sword & Shield™ (ADR/CD58 null):** An Allo-Defense Receptor that targets activated allo-reactive T-cells; CD58 null to impede allo-reactive T cell recognition & binding
- **hnCD16:** Enables ADCC when combined with therapeutic monoclonal antibodies to complement CAR specificity to provide additional antigen / target cell coverage
- **CD3 Fusion Receptor:** Provides additional antigen / target cell subset targeting in combination with T cell engagers
- **T Cell Enhancer(s):** Optimized regulation of cell persistence & potency
- **Enhanced biodistribution & microenvironment performance:** Engineered chemokine receptor surface profile to augment tissue trafficking; enhanced performance in inhibitory microenvironments via TGFβ signal redirection receptor (SRR)
- **TCR Null:** Complete bi-allelic disruption of TRAC ablates TCR expression and eliminates the possibility of GvHD
- **CD38 Null:** Potential to enhance metabolic cell fitness and allow combination with CD38 targeting mAbs

FT839 Product Candidate Executive Summary & Next Steps

Strategic highlights and value drivers

Novel multi-antigen targeting system

- **CD19 + CD38** comprehensively targets B-cell, plasma cell lineage and activated immune cells, including aberrant T cell, NK cell and myeloid cells
- **Ability to synergize** with mAbs and T cell engagers utilizing built in hnCD16 and CD3 fusion receptor

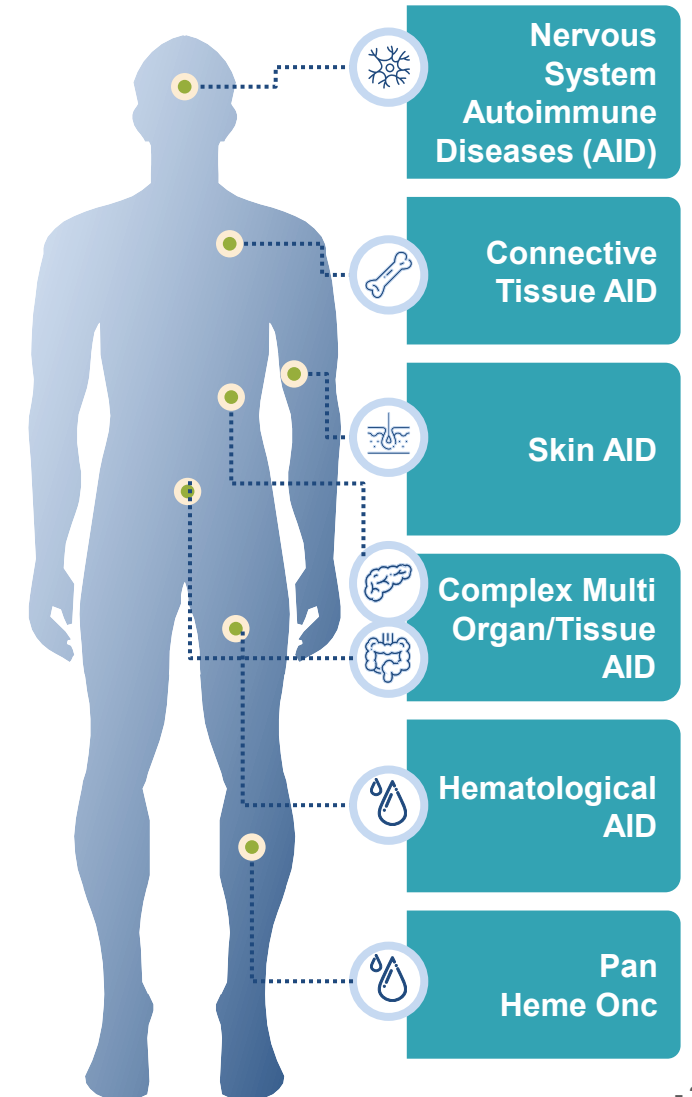
Next Gen CAR T-Cell

- **Multiple functional** edits that enhance potency, persistence, trafficking, and multi-antigen targeting
- **Sword and Shield™** technology to provide allo-protection

Clinical Advancement

- **Anticipated IND filing 2H 2026**
- Future pan autoimmune and hematologic malignancy evaluation leading to **broad indication expansion**

Possible **FT839** Disease Indications



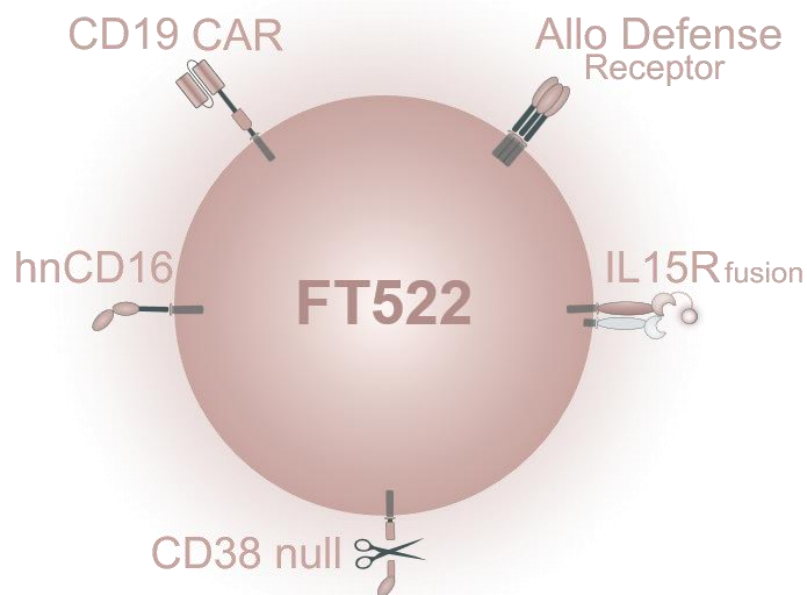
A microscopic view of several CAR NK cells. Each cell is spherical with a translucent, textured outer membrane and a bright red, bumpy nucleus. The cells are scattered across the frame, with some in sharp focus and others blurred in the background.

NEXT GENERATION OFF-THE-SHELF CAR NK CELLS



Making Cell Therapy Accessible to All™

True Off-the-Shelf Next Gen CAR NK cell Drug Product



Multi antigen targeting via CD19 CAR and hnCD16, with ADR technology designed to reduce/eliminate need for conditioning chemotherapy

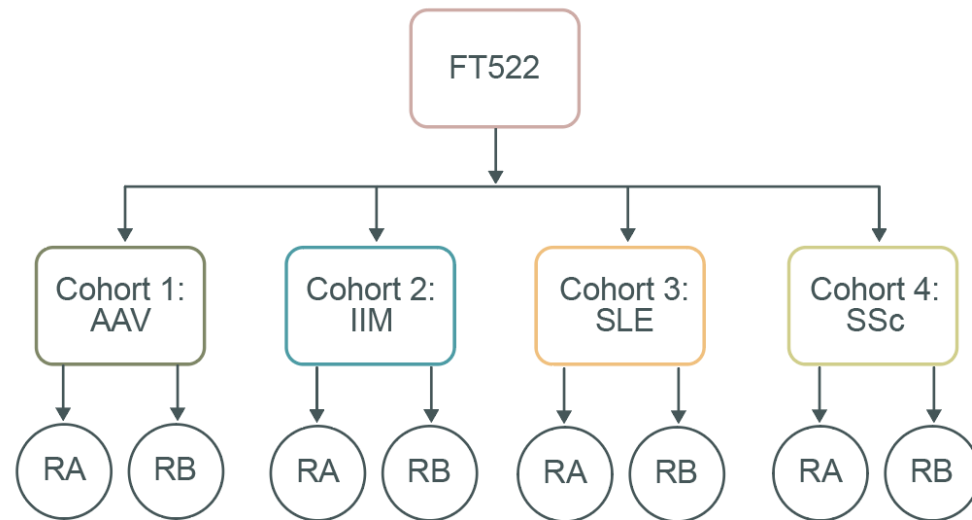
ADR armed NK Cells Uniquely Able to Proliferate and Persist

- **1XX CAR19:** Novel CAR with CD28 costimulatory and modified CD3z signaling domains for optimal safety and activity
- **ADR:** 4-1BB CAR targeting allo-reactive T-cells
- **IL15R Fusion:** Cell potentiation without cytokine support
- **CD38 Null:** Potential to enhance metabolic *cell fitness* and allow *combination with CD38 targeting mAbs*
- **hnCD16:** Enables ADCC when combined with therapeutic monoclonal antibodies to complement CAR to overcome tumor heterogeneity through *multi-antigen targeting*

FT522 Phase 1 Basket Study in Autoimmunity

IND cleared: Clinical development strategic planning ongoing

No Conditioning; Multiple Indications; Induction and Maintenance Regimens



All cohorts and regimens cleared to open in parallel and escalate independently

Basket Trial Design

AAV = Antineutrophilic cytoplasmic antibody-associated vasculitis

IIM = Idiopathic inflammatory myositis

SLE = Systemic lupus erythematosus

SSc = Systemic sclerosis

Regimen A (RA): treatment of participants with FT522 as add-on to Rituximab induction regimen

Regimen B (RB): treatment of participants, who are currently on background maintenance therapy and have been at a stable dose for at least 3 months, with FT522 and Rituximab

- Depending on participant population, background maintenance therapies include MMF, AZA, LEF, MTX, and avacopan



TRANSFORMING THE LIVES OF
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