TRANSFORMING THE LIVES OF PATIENTS WITH AUTOIMMUNE DISEASES AND CANCER

Making Cell Therapies Accessible to All™

Fate THERAPEUTICS

Corporate Presentation

August 2025

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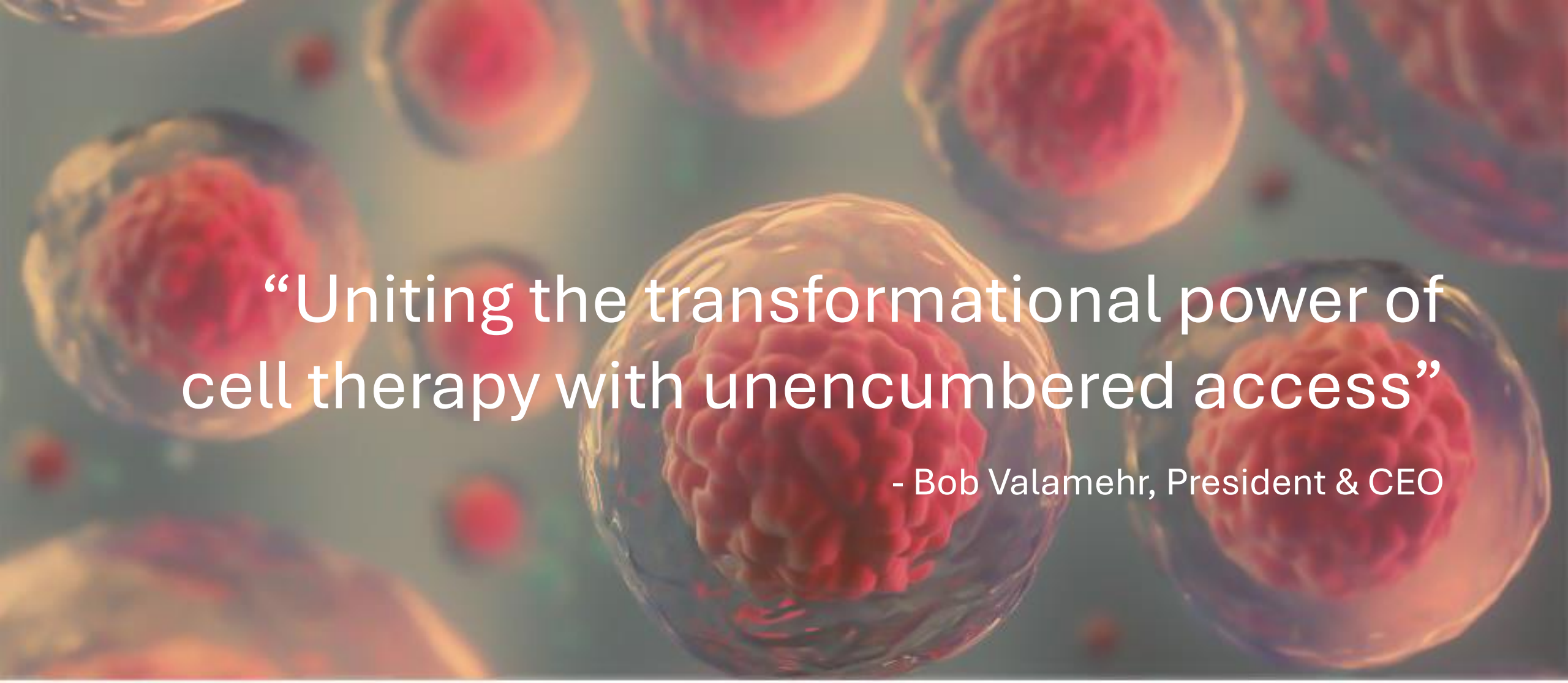


“Uniting the transformational power of cell therapy with unencumbered access”

- Bob Valamehr, President & CEO

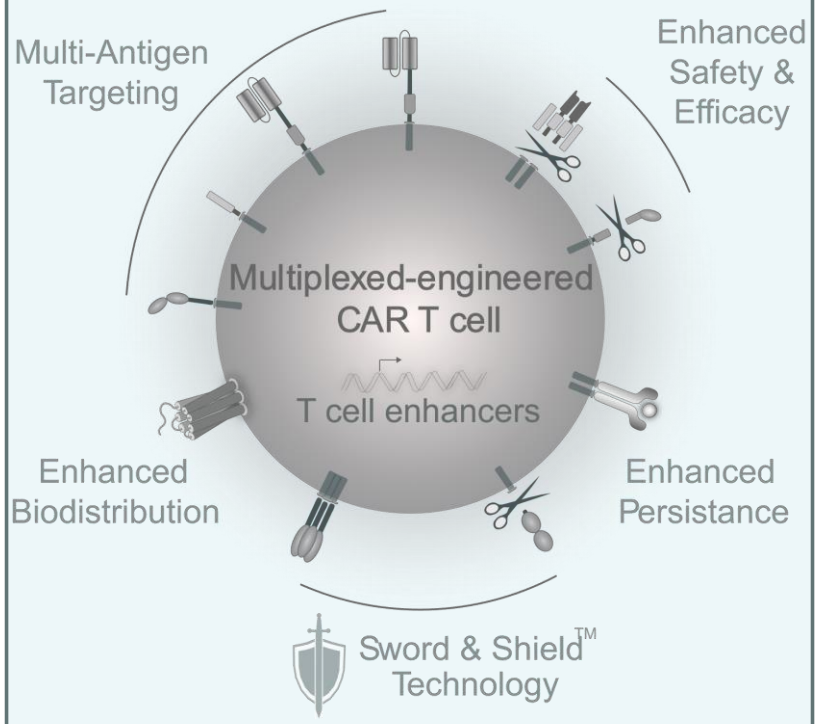
Fate THERAPEUTICS

Making Cell Therapy Accessible to All™

Resetting the Paradigm - Not just the Patient

Pioneering the development of truly accessible cell therapies - available on demand - anywhere

Unique Living Drug with Broad Disease Targeting



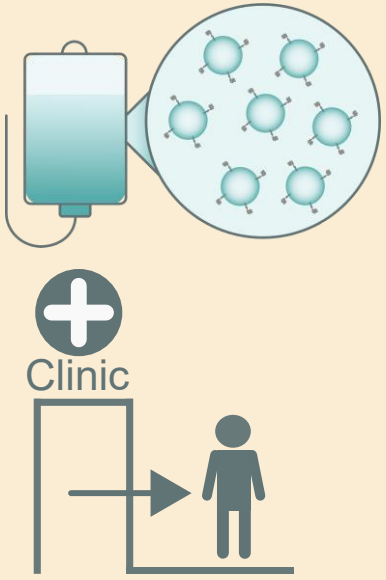
Renewable Manufacturing Process Delivering On-Demand Cell Therapies

- Single-Step Multiplex Gene Engineering: integrates multiple mechanisms of action
- Scalable Manufacturing: high-yield, cost-efficient from a defined MCB
- Uniform Product Profiles: consistent identity, purity and potency
- Off-the-Shelf CAR T cells: Cryopreserved inventory for on-demand use and broad patient reach

Novel Therapy Paradigm

To enable:

- ✓ Manufactured prior to patient need
- ✓ Available as an off-the-shelf therapy
- ✓ Stored for on-demand delivery
- ✓ Outpatient treatment enabled
- ✓ No conditioning chemotherapy
- ✓ Redosing as needed



A Differentiated & Renewable Cell Manufacturing Process

Fate’s platform uniquely delivers cell therapies on-demand to all 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

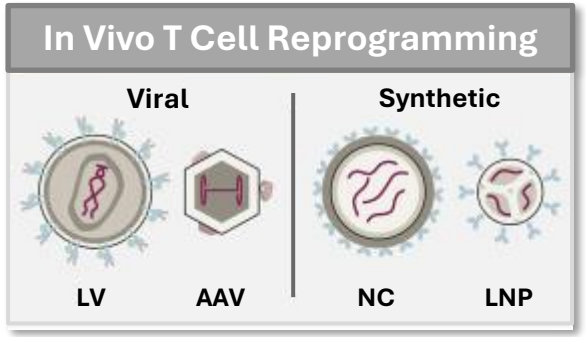
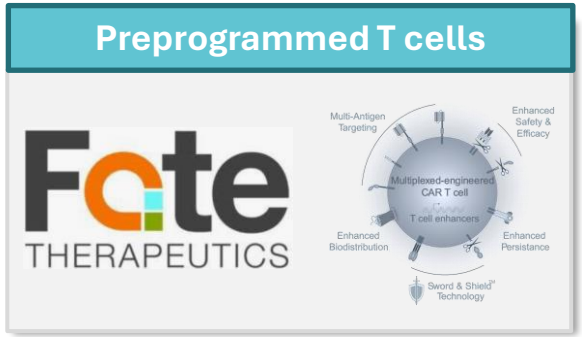
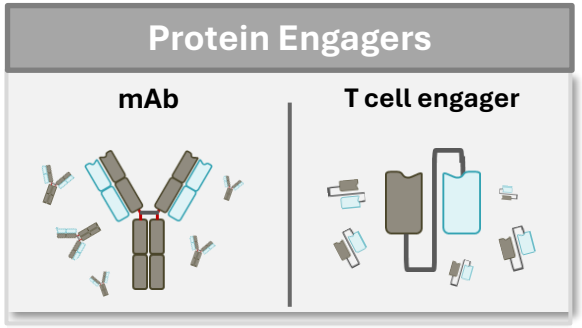
- Potential for profound efficacy in difficult-to-treat diseases
- **Healthy** starting material
- Random, variable, **per-batch** T-cell engineering
- Complex logistics
- **Multiple** dose paradigm
- Heterogeneous drug product
- Extended hospitalization
- Expensive ($$$)

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

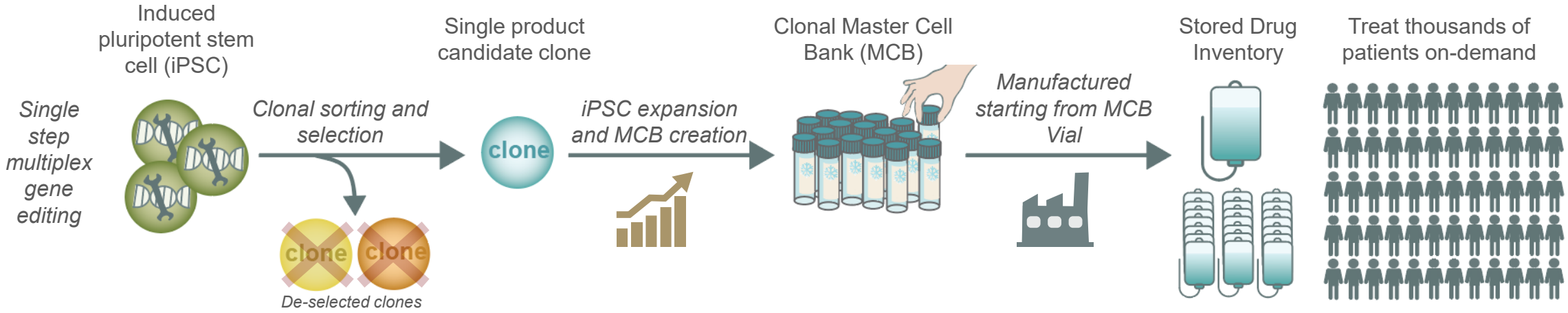
Fate's preprogrammed CAR T cells deliver potency of a living drug with uniformity of traditional biologics

In Vivo T Cell Reprogramming	Preprogrammed T cells	Protein Engagers
		
Limited disease reach through reduced genetic engineering capacity	**Broad** disease reach through multiplex-engineering and multi-antigen targeting	**Limited** disease reach through reduced ability for complex protein engineering
Potential for 'off target' cellular reprogramming & **genetic integration** with unknowable safety outcomes	**Uniform** drug product, ready on demand by large-scale manufacture, provides unique access and safety profile	**Uniform** drug product ready on demand with scaled manufacturing capacity
Dependance on **patients'** own immune system engagement for function	**Pre-selected** T cell performance with no dependance on patient immune system	Dependence on **patients'** own immune system engagement for function
Tissue penetration dependent on the efficiency of *in vivo* engineering step	**Tissue penetration** including 2º and 3º tissues	**Limited** penetration into 2º and 3º tissues
No lympho-depleting conditioning, outpatient treatment	**Low-to-no** lympho-depleting conditioning, outpatient treatment	**No** lympho-depleting conditioning, outpatient treatment

LV: Lentiviral vector; AAV: Adenovirus associated vector; NC: Nanocarriers; LNP: Lipid nanoparticles; mAb: Monoclonal antibodies; TCE: T cell engager

Unique Platform for Delivery of Off-the-Shelf Cellular Therapies

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



Platform Advantages

- ✓ **Defined Clonal MCB**: Single-cell derived, genetically uniform, selected for potency and genomic integrity.
- ✓ **Engineered MCB Starting Material**: One-time edit, highly scalable, donor-independent, and enables consistent high-quality products.
- ✓ **Modular Innovation**: Accelerates development through efficient, multiplexed engineering.

iPSC-derived Cell Therapy Products

- ✓ **Reliable, Scalable Drug Product**: Consistent, well-characterized, >5-year shelf stability; ~50,000-dose GMP-scale capacity at current site.
- ✓ **Cost-Effective & Consistent**: Low COGs (~$3,000/dose), inventory-based economics, and no donor variability.
- ✓ **Patient-Centered Therapy**: Off-the-shelf, antibody-like treatment with repeat dosing, low toxicity, and outpatient-friendly administration.

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+ ft^2 Fate cGMP manufacturing facility co-located with corporate headquarters
- 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 US and international clinical development as well as initial commercial launch

Addressing Diseases with Significant Unmet Clinical Need

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

Autoimmune Disease

Systemic Lupus Erythematosus (SLE)	Systemic Sclerosis (SSc)	Antineutrophilic cytoplasmic anti-body associated vasculitis (AAV)	Idiopathic Inflammatory Myositis (IIM)
Inflammatory disease with risk to multiple organs and systems	Characterized by fibrosis and vascular damage impacting various organs	Inflammation & necrosis of blood vessels leading to endothelial & organ damage	Group of disorders that cause chronic inflammation progressive and muscle weakness

Oncology

HER2+ EGFR+	Pan Tumor (Solid & Heme)
Efficacy for many patients is limited by multiple tumor resistance mechanisms	

US & EU Patients

~1.3M
Prevalence

~2M
Annual mortality

Market Size (2024)

USD ~$105B

USD ~$230B

1. Izmirly et al. Arthritis Reum 2021
2. Smoyer-Tomic et al. BMC Musc Dissorders 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)

First-in-Class Product Pipeline

Multiplexed-engineered, iPSC-derived product candidates

Program	CAR/Antigen Target	Indication	Research	Preclinical	Phase 1 (NCT#)	Partner
Autoimmunity						
FT819 (RMAT)	CD19	Systemic Lupus Erythematosus (SLE)	FT819-102		NCT06308978 Enrolling	CIRM
		Systemic Sclerosis (SSc)	FT819-102			
		ANCA associated Vasculitis (AAV)	FT819-102			
		Idiopathic Inflammatory Myopathies (IIM)	FT819-102			
FT839 (NxG)	CD19/CD38/CD20	Pan-Indication w/o LCC				
FT522	CD19/CD20	Pan-Indication w/o LCC	FT522-102			
Oncology						
FT825	HER2/EGFR	Solid Tumor (s)	FT825-101		NCT05950334 Enrolling	ONO PHARMACEUTICAL CO.,LTD.
Undisclosed	Undisclosed	Solid Tumor (s)	Research extended thru June 2026			
FT836 (NxG)	MICA/B/EGFR/HER2	Pan-Indication (Solid/Heme) w/o LCC	IND Cleared July 2025			CIRM
FT839 (NxG)	CD19/CD38/BCMA/GPRC5D	Pan-Indication (Heme) w/o LCC				

T Cell NK Cell

RMAT: FDA Regenerative Medicine Advanced Therapy (RMAT) received in SLE
LCC: Lympho-conditioning chemotherapy
NxG= Next-generation CAR T cell product
US rights to all products

FT819 Program

CD19 Targeting CAR T-Cell Product Candidate

Fate THERAPEUTICS

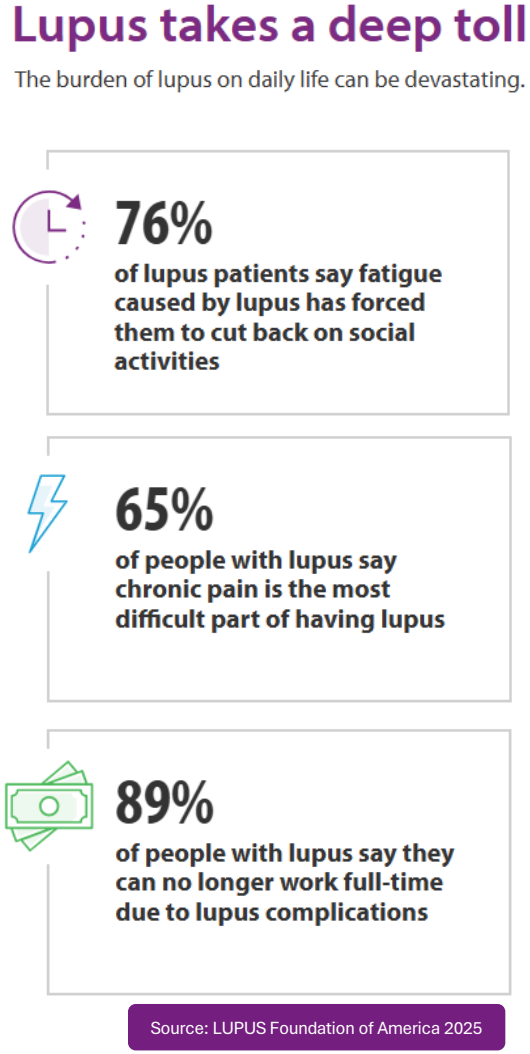
Making Cell Therapy Accessible to All™

Systemic Lupus Erythematosus (SLE): A Disease of Significant Unmet Need

Chronic disease burden, multi-organ involvement and increased morbidity & mortality

High disease burden, disability & organ damage

- Typical patient is a women of childbearing age presenting with fatigue, joint pain, rash and systemic inflammation affecting kidneys, CNS, lungs or heart
  - 40-60% patients exhibit moderate to severe multi-organ functional impairment[1]
  - Chronic fatigue, cognitive dysfunction & photosensitivity significantly limit quality of life
  - Renal involvement (lupus nephritis) occurs in ~40% of cases
  - High risk of end stage renal disease (ESRD)[2]
- Mortality risk is 3 x higher than the general population due to cumulative organ damage, infection and treatment complications[3]
- Approximately 20% of patients experience irreversible organ damage with in 5 years despite current therapy treatment[4]
- Only three FDA approved therapies, with limited therapeutic benefit



1. Petri et al., Lupus. 2012; 21(5): 499–503.
2. Almaani et al., Nat Rev Nephrol. 2017;13(3):170-183.
3. Yurkovich et al., Arthritis Care Res (Hoboken). 2014;66(4):608-616.
4. Bruce IN. Lupus. 2005;14(1):5-10

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

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

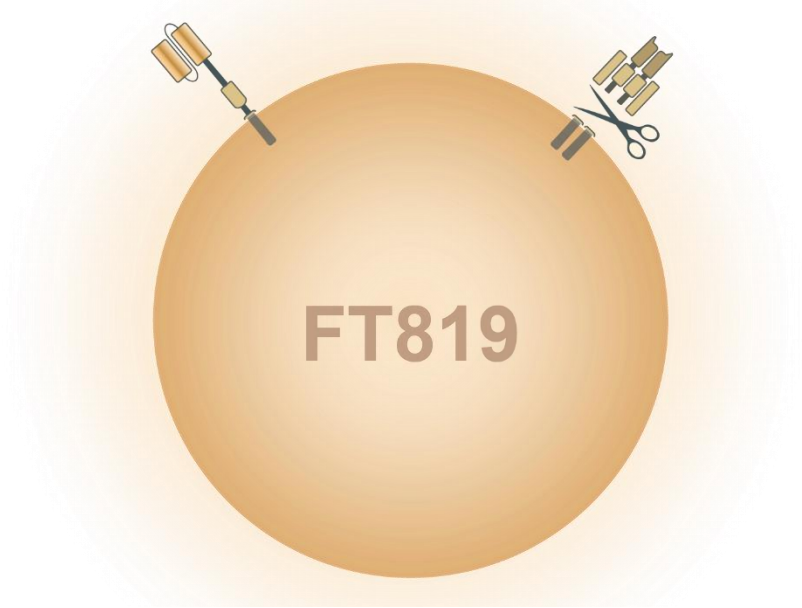
True Off-the-Shelf CAR T-Cell Drug Product

CAR19

State-of-the-art CAR motif and expression control

TCR null

Complete TCR knock-out to prevent GvHD in allogeneic settings



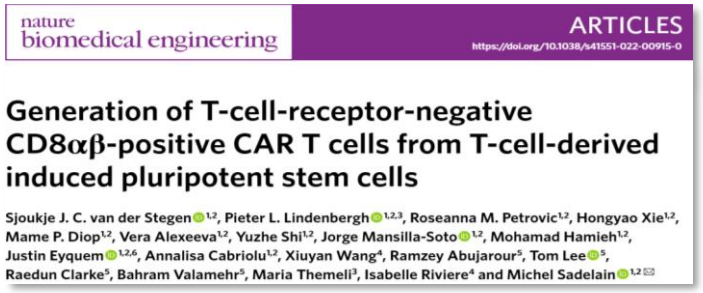
CD19 CAR T-cell designed to eliminate pathological auto-reactive B-cells with balanced efficacy and safety to establish immune reset and clinical remission

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

Memorial Sloan Kettering Cancer Center

CIRM CALIFORNIA INSTITUTE FOR REGENERATIVE MEDICINE


nature biomedical engineering ARTICLES https://doi.org/10.1038/s41551-022-00915-0

Generation of T-cell-receptor-negative CD8αβ-positive CAR T cells from T-cell-derived induced pluripotent stem cells

Sjoukje J. C. van der Stegen[1,2], Pieter L. Lindenbergh[1,2,3], Roseanna M. Petrovic[1,2], Hongyao Xie[1,2], Mame P. Diop[1,2], Vera Alexeeva[1,2], Yuzhe Shi[1,2], Jorge Mansilla-Soto[1,2], Mohamad Hamieh[1,2], Justin Eyquem[1,2,6], Annalisa Cabriolu[1,2], Xiuyan Wang[4], Ramzey Abujarour[5], Tom Lee[5], Raedun Clarke[5], Bahram Valamehr[5], Maria Themeli[3], Isabelle Riviere[4] and Michel Sadelain[1,2]

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.

FT819 Provides Rapid and Dose Dependent B Cell Elimination

Relative CD19 CAR T cell activity against SLE patient derived PBMCs show FT819 to be highly potent

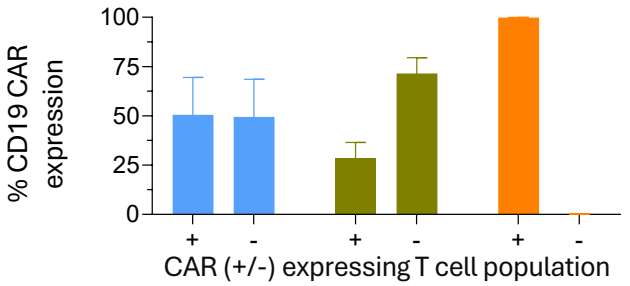
Manufacturing & Engineering Process Influences Product Uniformity and Potency

- ***Autologous CAR T cell*** *surrogate manufacturing process (3-14 days of T cell culture):*
  - Enriched T cells activated and transduced with CD19 CAR lentivirus
  - Standard manufacturing processes & timelines used to generate product

- ***Allogeneic CAR T cell*** *surrogate manufacturing process (21-35 days of T cell culture):*
  - Enriched T cells CRISPR engineered to eliminate TCR expression (TCR KO)
  - T cells subsequently transduced with CD19 CAR lentivirus using extended manufacturing processes & timelines to establish a multi-dose product

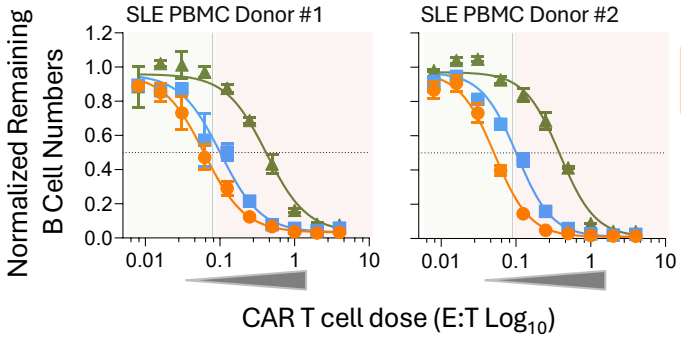
- ***iPSC derived CAR T cell (FT819)*** *manufacturing process (7 days of T cell culture):*
  - Reprogrammed iPSCs engineered one-time to insert CAR into TRAC locus & clonally selected on engineering fidelity, genomic stability, and functional performance
  - Selected clonal master iPSC cells manufactured to reproducibly generate large CAR T-cell product inventory with limited expansion at T-cell stage

CAR T-cell Product Uniformity

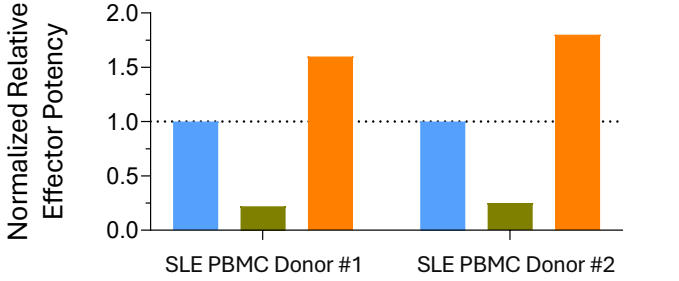


FT819 demonstrates superior CD19 CAR expression uniformity compared to autologous and allogeneic CAR T cells derived from patient or doner cells

FT819 Exhibits Significant Product Potency, Comparable to Autologous CAR T cells[1]



Left shifted curves indicates greater potency



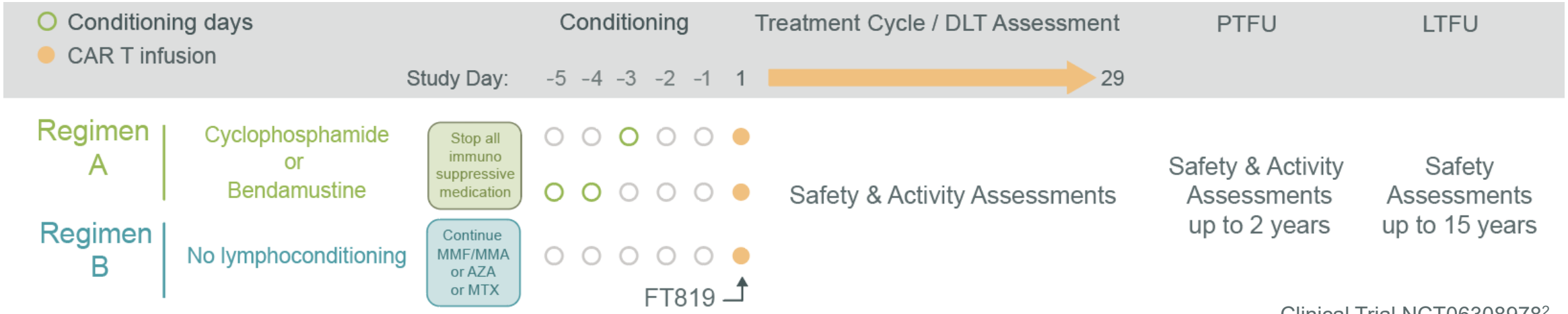
Relative potency >1 suggest greater potency

1. Wong L, et al. Molecular Therapy Vol 32 No 4S1, April 2024

CRISPR: Clustered Regularly Interspaced Short Palindromic Repeats; E:T: Effector to target ratio; iPSC: induced pluripotent stem cells; KO: knock out; PBMC: peripheral blood mononuclear cell: TCR: T cell receptor; TRAC: T cell receptor alpha chain

FT819-102: Phase 1 Study of FT819 in B-cell Mediated Autoimmune Diseases[1]

Uniquely administered with fludarabine-free conditioning or maintenance therapy in the absence of chemotherapy conditioning



Clinical Trial NCT06308978[2]

Highly-Differentiated Therapeutic Approach

Available on-demand with:

- No patient apheresis
- Less-intensive or no conditioning chemotherapy regimens
  - No discontinuation of maintenance therapy (Regimen B)
- Shortened hospitalization requirement (3 days)
- Ability to redose in inadequate response or relapse
- Autoimmune diseases in the protocol include: Systemic lupus erythematosus (SLE), ANCA-associated vasculitis (AAV), Idiopathic inflammatory myopathy (IIM), Systemic sclerosis (SSc)

1. *V. Sandhu, et al. Annals of the Rheumatic Diseases, Volume 84, Supplement 1, 2025, Pages 29-30*
2. *https://clinicaltrials.gov/study/NCT06308978 (ClinicalTrials.gov)*

FT819-102: Phase 1 Study of FT819 in B-cell Mediated Autoimmune Diseases[1]

SLE patient disease characteristics

Patient Characteristics					
	Regimen A				Regimen B
Patient #	A1-DL1	A2-DL1	A3-DL1	A1-DL2	B1-DL1
Age, Gender	28 F	22 F	29 F	28 F	23 F
BILAG domain for inclusion	Renal	Renal	Renal	MSK, Mucocutaneous	Cardiorespiratory
Disease Duration	~11 years	~4 years	~24 years	~9 years	~5 years
Baseline SLEDAI-2K	20	20	14	18	8
Concomitant SLE Therapies	GC, HCQ	HCQ	GC, HCQ	HCQ	GC, HCQ, MMF
Prior Therapies *B-cell targeted therapy bolded	7 AZA, **BEL**, GC, HCQ, MMF, **RTX**, TAC	8 ANI, **BEL**, CY, GC, HCQ, MMF, MTX, **RTX**	8 AZA, **BEL**, CY, GC, HCQ, MMF, MTX, **RTX**	6 ANI, **BEL**, CY, HCQ, GC, MTX	5 CY, GC, HCQ, MMF, **RTX**
Conditioning	Bendamustine	CY	CY	CY	None

ANI = anifrolumab; AZA = azathioprine; BEL = belimumab; CY = cyclophosphamide; GC = glucocorticoids; HCQ = hydroxychloroquine; MMF = mycophenolate mofetil; MSK = musculoskeletal; MTX = methotrexate; RTX = rituximab; TAC = tacrolimus

Data cut off 15th May 2025

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

FT819-102: Phase 1 Study of FT819 in B-cell Mediated Autoimmune Diseases[1]

Preliminary clinical safety data

No high-grade CRS, No ICANS, and No DLTs observed

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

Selected Adverse Events, Highest Grade Reported

	Regimen A				Regimen B
Patient (conditioning)	A1-DL1 (Benda)	A2-DL1 (Cy)	A3-DL1 (Cy)	A1-DL2 (Cy)	B1-DL1 (none)
Cytokine Release Syndrome	-	-	-	Grade 2	-
ICANS	-	-	-	-	-
Graft vs. Host Disease	-	-	-	-	-
Grade ≥ 3 infection	-	-	-	UTI (Grade 3)	Influenza (Grade 3)

Data cut off 15th May 2025

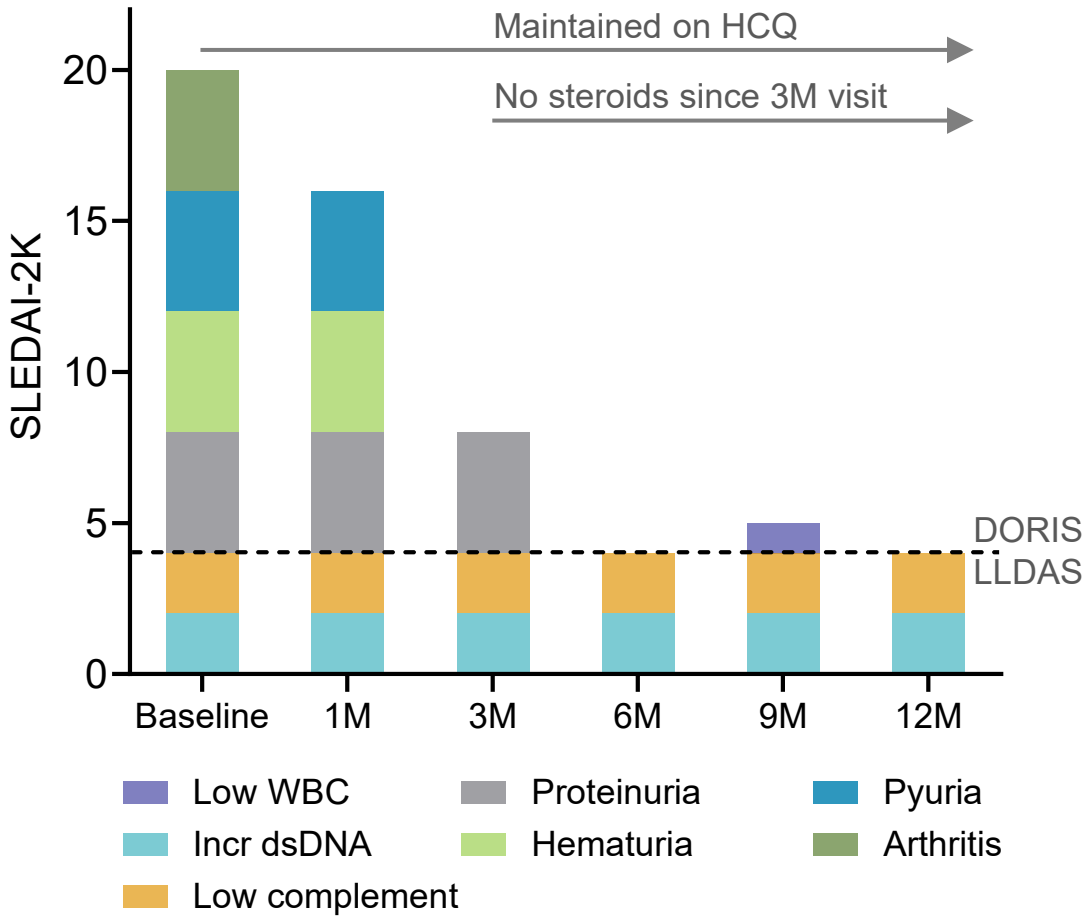
Regimen A = Conditioning prior to treatment with either bendamustine (Benda) or cyclophosphamide (Cy); Regimen B = no conditioning chemotherapy.
DL = Dose level; DL1 = 360M cells; DL2 = 900M cells.
Adverse events graded per CTCAEv5.
ICANS = Immune effector Cell Associated Neurotoxicity Syndrome; UTI = urinary tract infection. Grade ≤ 2 Headache reported in 4 participants and Grade ≤ 2 nausea and vomiting reported in 3 participants.

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

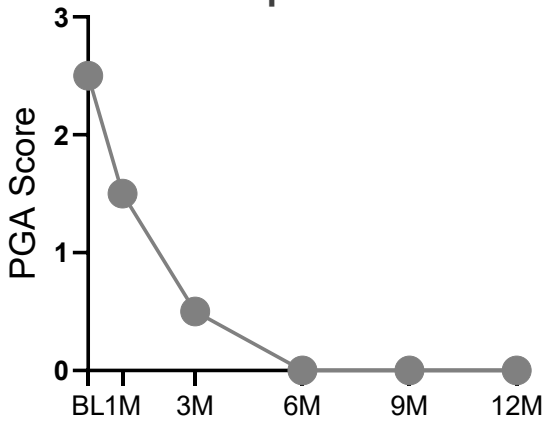
FT819-102 Activity in SLE: DORIS Remission >1 Year Indicates Durable Response

Patient 1 SLE case (A1-DL1)

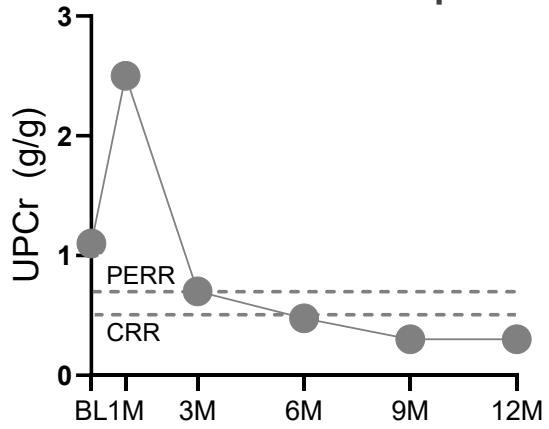
Remission in SLE without use of steroids



Sustained improvement in overall patient status



Durable renal response



- ✓ No leukapheresis
- ✓ Fludarabine-free lympho-conditioning
- ✓ On-demand CAR T-cell delivery
- ✓ No DLT, CRS, GvHD or ICANS
- ✓ Reduced hospitalization (3 days)

CRR: Complete Renal Response
DORIS: Definition Of Remission In SLE
HCQ: Hydroxychloroquine
LLDAS: Low Lupus Disease Activity State
SLEDAI: SLE Disease Activity Index
PERR: Primary Efficacy Renal Response
PGA: Physician Global Assessment

Data cut off 15th May 2025

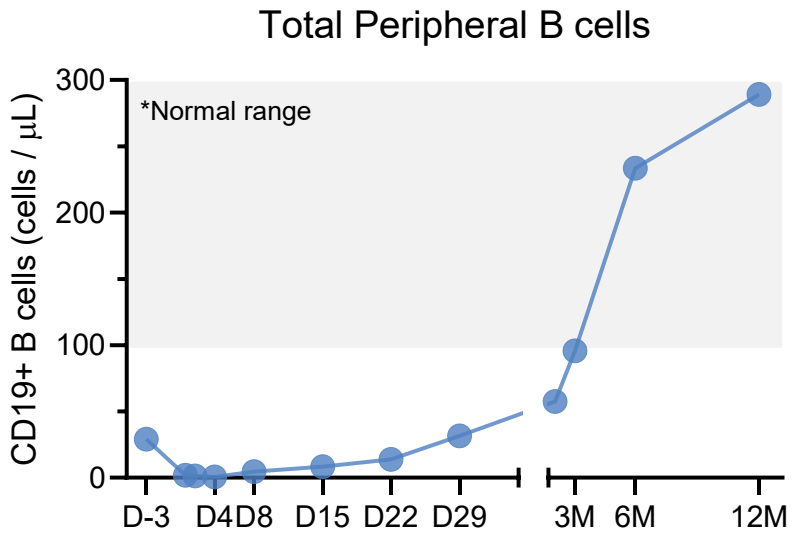
Graphs show measurements across study timepoints, BL = baseline, M=month

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

FT819-102 Durability of Remission is Supported by B-cell Immunological Reset

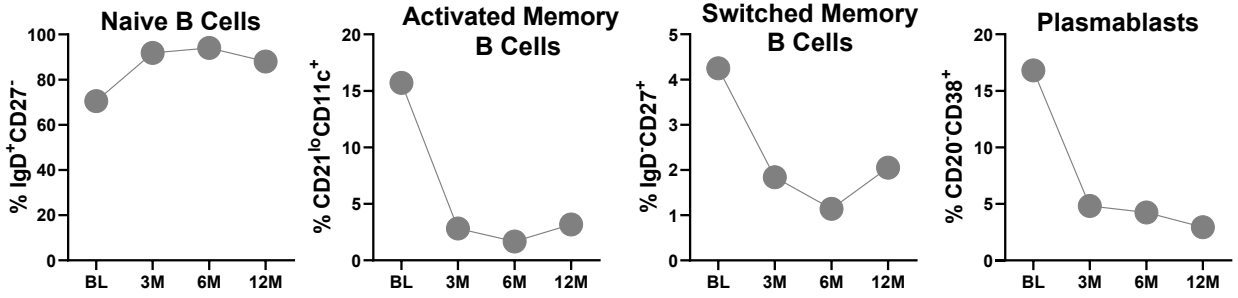
Patient 1 SLE case (A1-DL1)

Effective B cell depletion & repopulation to normal levels*

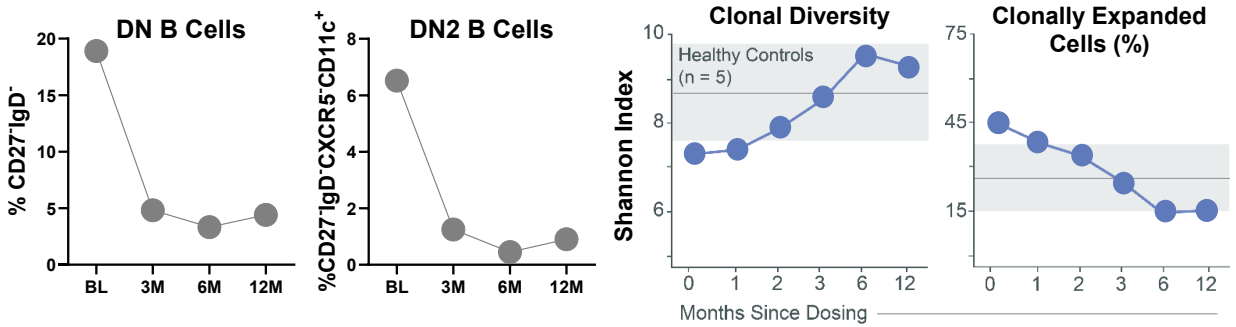


- ✓ No leukapheresis
- ✓ Fludarabine-free lympho-conditioning
- ✓ On-demand CAR T-cell delivery
- ✓ No DLT, CRS, GvHD or ICANS
- ✓ Reduced hospitalization (3 days)

Reconstituting B cells are predominantly naïve, minimal switched memory, and low plasmablasts indicative of immune reset



Persistent DN pathogenic subset clone depletion and diversification of the B cell repertoire observed post-FT819 treatment



Data cut off 15th May 2025

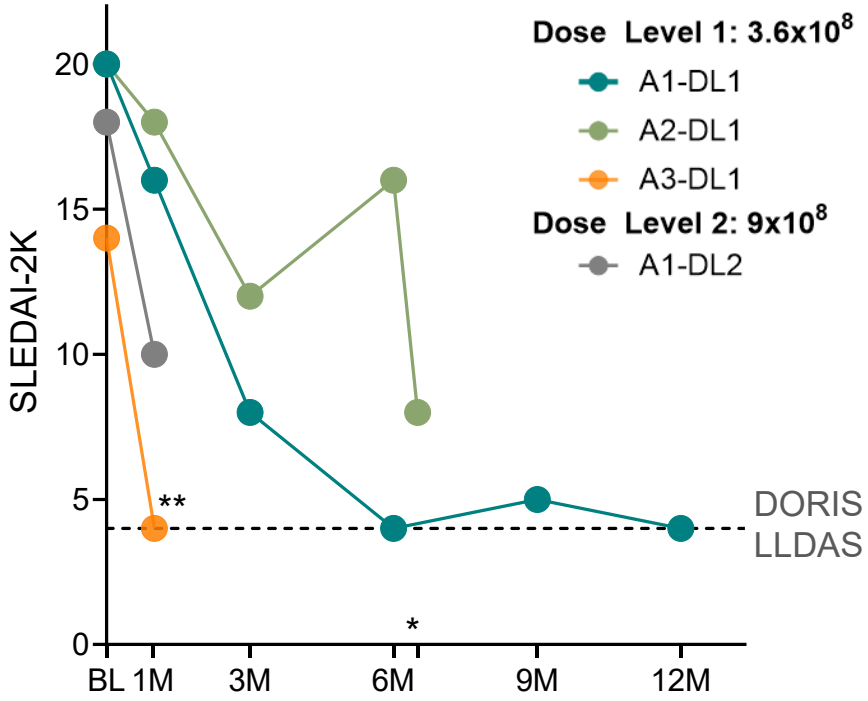
*Graphs show measurements across study timepoints, BL = baseline, D=day, M=month. *Normal B cell levels defined as 100-700 cells/µL*

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

FT819-102 Collective Reduction in Disease with Fludarabine-Free Conditioning

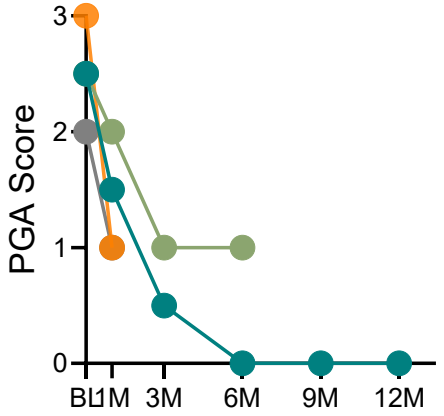
Patients 1 - 4 in Regimen A *(Cyclophosphamide or Bendamustine monotherapy lympho-conditioning)*

Improved disease activity across all patients evaluated up to data cut off (n4)

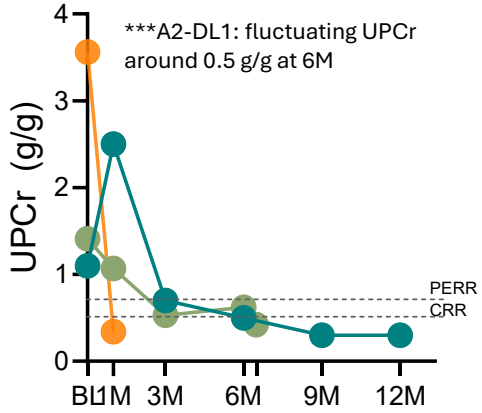


*A2-DL1: fluctuating UPCr around 0.5 g/g at 6M
**A3-DL1: Patient discontinued due to noncompliance after 1M visit

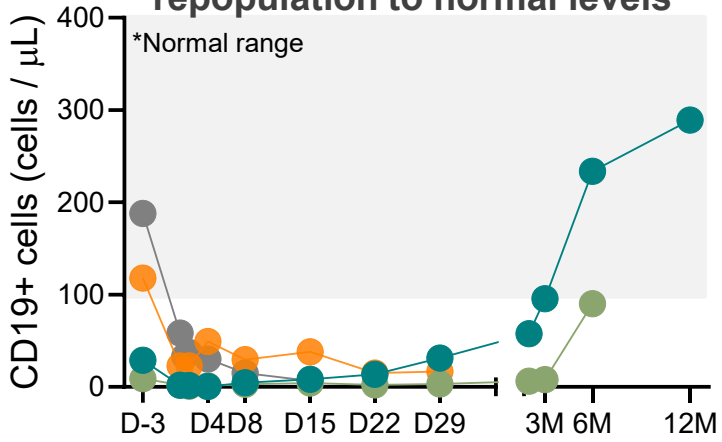
Persistent improvement in overall patient status (n4)



Renal responses achieved in all LN patients (n3)



Effective B cell depletion & repopulation to normal levels*



Data cut off 15th May 2025

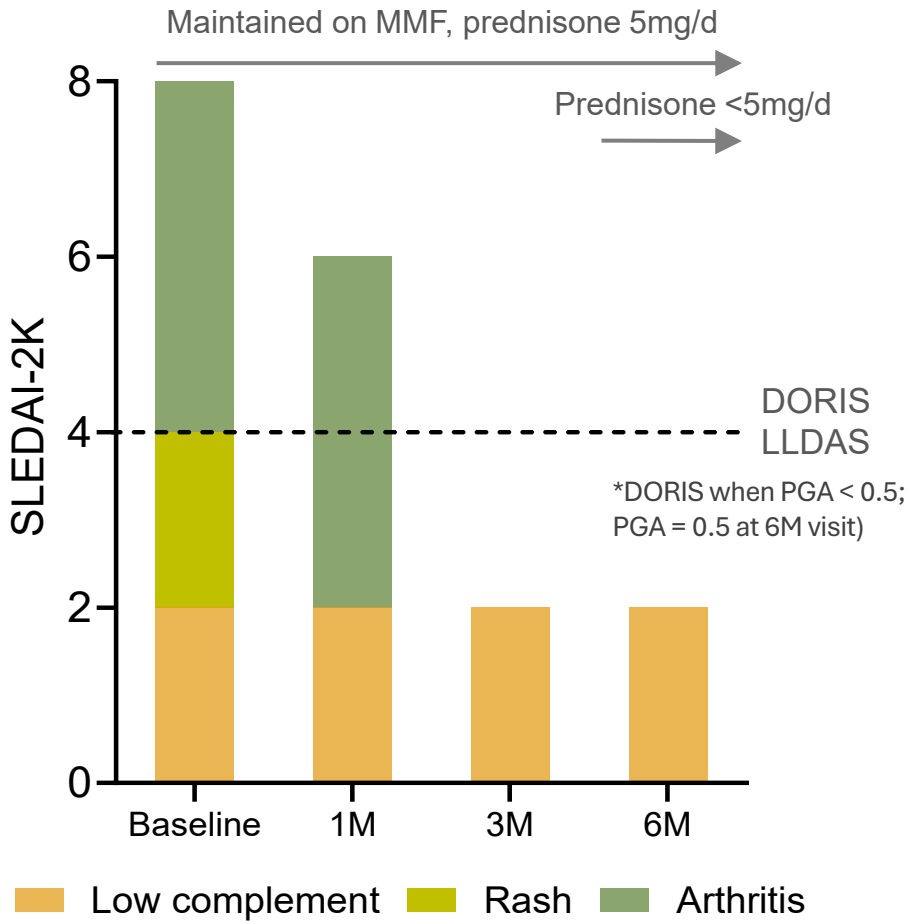
*Graphs show measurements across study timepoints, BL = baseline, D=day, M=month. *Normal B cell levels defined as 100-700 cells/µL*

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

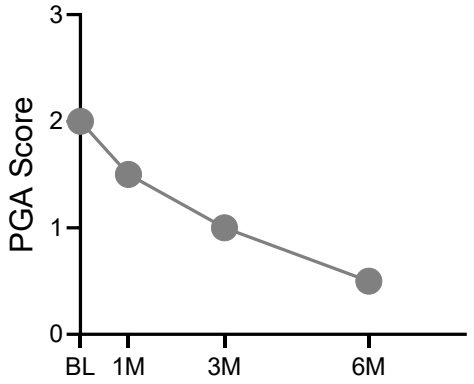
FT819-102: Remission in SLE Achieved Without Lympho-Conditioning

First patient (B1-DL1) in Regimen B *(FT819 as add-on to maintenance therapy)*

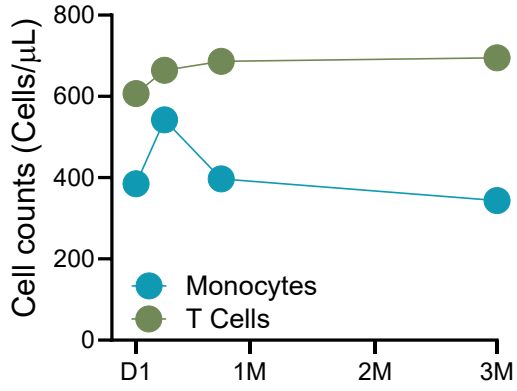
Remission in SLE without lympho-conditioning



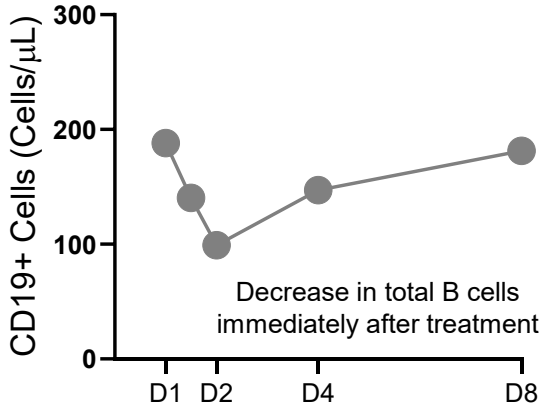
Persistent improvement in overall patient status



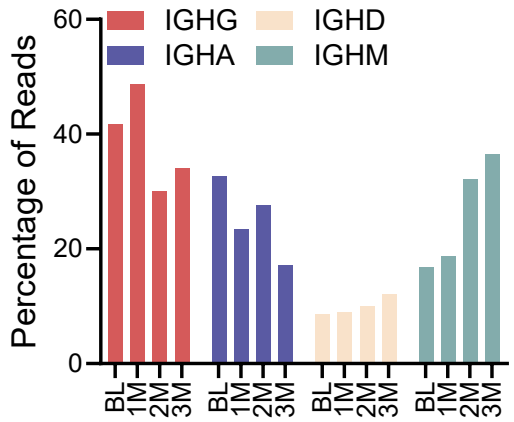
Preservation of baseline immune cells



Initial B cell reduction resolves within one week



B cell remodeling favors naïve, non-switched populations



Data cut off 15th May 2025

*Graphs show measurements across study timepoints, BL = baseline, D=day, M=month. *Normal B cell levels defined as 100-700 cells/µL*

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

FT819-102 Summary and Next Steps

- FT819 provides a true off-the-shelf CAR T cell therapeutic option that overcomes many of the challenges seen in autologous and allogenic cellular therapies
- Preliminary clinical data suggests FT819 can support durable clinical activity with less intensive conditioning or in combination with maintenance therapy without conditioning
- Complemented by the initial clinical trial in lymphoma (> 50 patients), FT819 exhibits a differentiated safety profile with no reported ICANS, GvHD or CRS > Grade 2
- Trial open to treat ANCA vasculitis, Myositis (DM/PM/IMNM), SLE, and Systemic Sclerosis aged 12-70 years; treatment option includes redosing after relapse or inadequate response
- Current data generated from 2 clinical sites (University of Nebraska and University of Minnesota)
  - 8-10 sites projected to be active by summer 2025, in addition to ongoing efforts to expand to multiple outside of US sites in 2025
- Upcoming regulatory discussions
  - RMAT designation granted with goal to have pivotal trial design reviewed with the FDA by YE2025
  - Removal of hospitalization requirement

FT825 Program

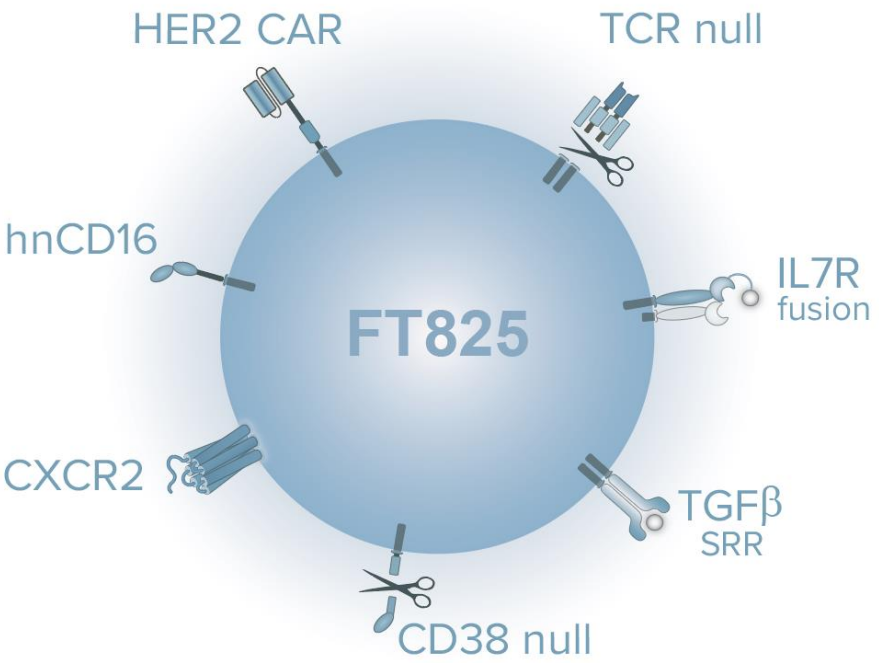
HER2 Targeting CAR T-Cell Product Candidate

Fate THERAPEUTICS

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 **H_2CasMab-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**: *Resistance to TGF*β-mediated suppression commonly found in TME of solid tumors
- **CXCR2**: Enhancement of *migration into solid tumors*
- **IL7RF**: Enhances CAR iT *persistence* and self-renewal
- **CD38 KO**: Potential to enhance metabolic *cell fitness*

ONO PHARMACEUTICAL CO.,LTD.

Article

Cell Stem Cell

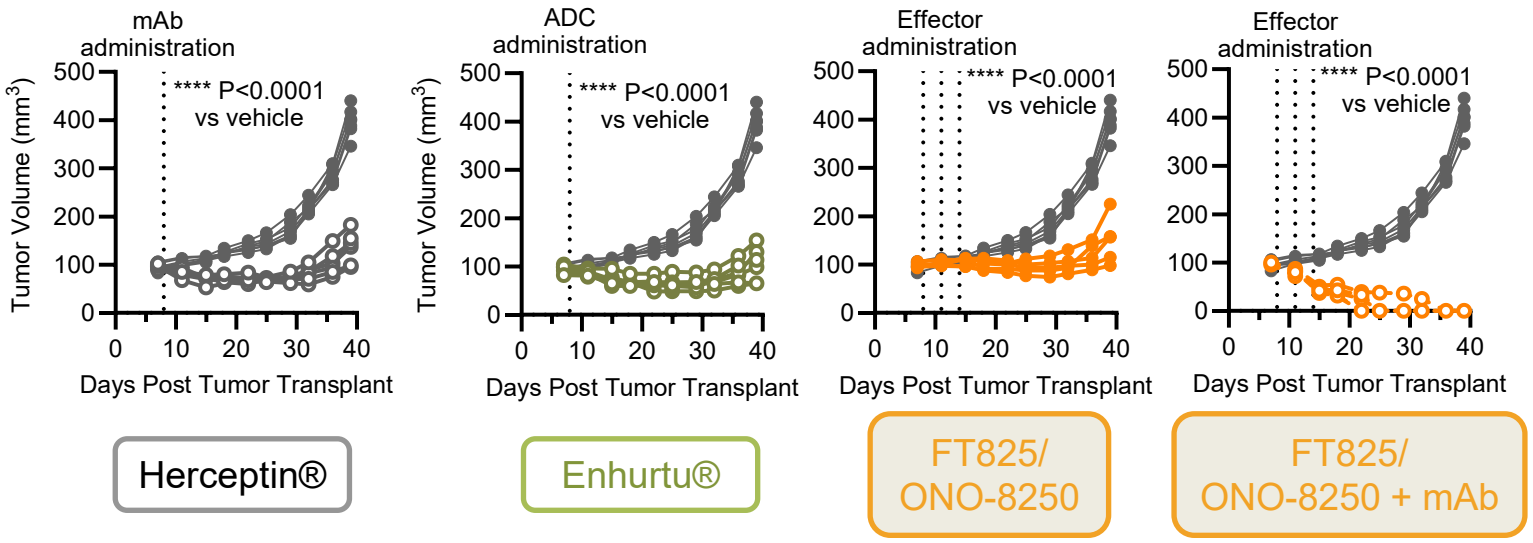
Preferential tumor targeting of HER2 by iPSC-derived CAR T cells engineered to overcome multiple barriers to solid tumor efficacy

1. Hosking MP et al. Cell Stem Cell. 2025 May 30:S1934-5909(25)00187-0.
2. https://ir.fatetherapeutics.com/news-releases/news-release-details/fate-therapeutics-highlights-cancer-selective-her2-targeting

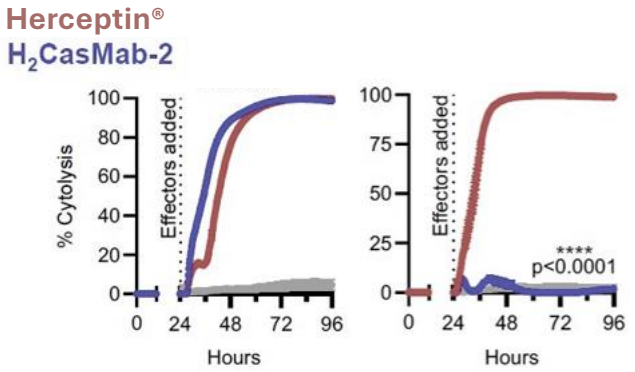
Novel Cancer-Specific CAR Binder Limits Off Tumor Toxicity

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

- Novel binder (H_2CasMab-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



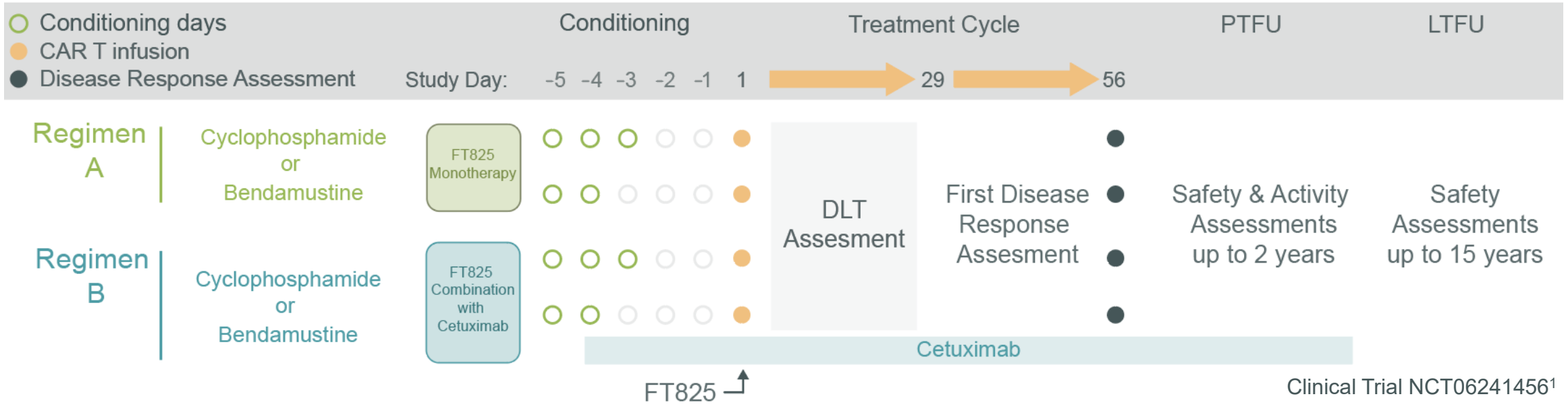
	Herceptin®	H_2CasMab-2
On Target **On** Tumor	✓	✓
On Target **Off** Tumor[2]	✗	✓

In contrast to Herceptin® H_2CasMab-2 shows limited On Target-Off Tumor toxicity

1. *Hosking MP et al. Cell Stem Cell. 2025 May 30:S1934-5909(25)00187-0.*
2. Moja Let al. Cochrane Database Syst Rev. 2012 Apr 18;2012(4)

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

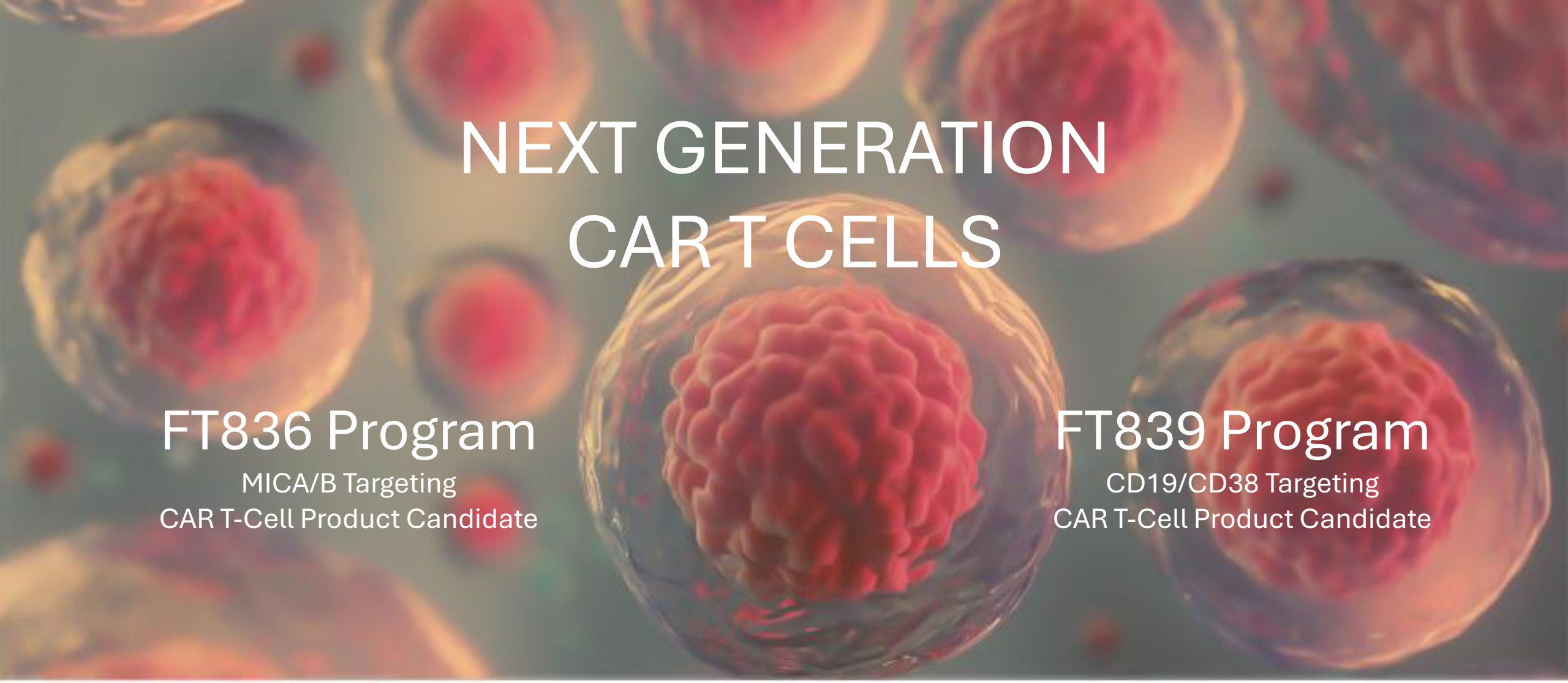
Phase 1 Study: FT825/Ono0825 with/without monoclonal antibody combination (NCT06241456)



Clinical Trial NCT06241456[1]

Regimen A: FT825 Monotherapy	• HER2⁺ Breast, Gastric, GEJ, HER2^{Mut} NSCLC • HER2⁺ salivary, endometrial, other cancers	**Early Clinical Observations** • Two Dose Levels (100M and 300M cells) have been evaluated. Enrollment is ongoing at Dose Level 3 (900M cells) • 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	

1. *https://clinicaltrials.gov/study/NCT06241456 (ClinicalTrials.gov).*



NEXT GENERATION CAR T CELLS

FT836 Program

MICA/B Targeting
CAR T-Cell Product Candidate

FT839 Program

CD19/CD38 Targeting
CAR T-Cell Product Candidate

Fate THERAPEUTICS

Making Cell Therapy Accessible to All™

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

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

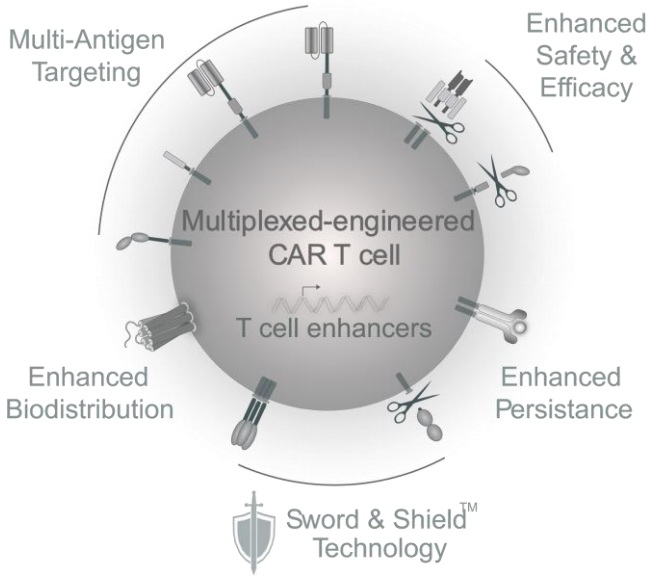
Overcoming Multiple Tumor Challenges Across Diverse Tumors Indications

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.



Broad Elimination of Pathological Immune Cell Subsets & Compartments

Problem Statement

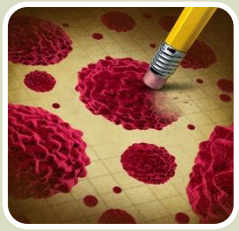
- Autoimmune, hematological malignancies & inflammatory diseases arise from dysregulated T, B and myeloid cell function across secondary and tertiary immune sites.
- Current therapies offer broad immune suppression or narrowly target specific cells, frequently falling short of effective immune control.

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 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

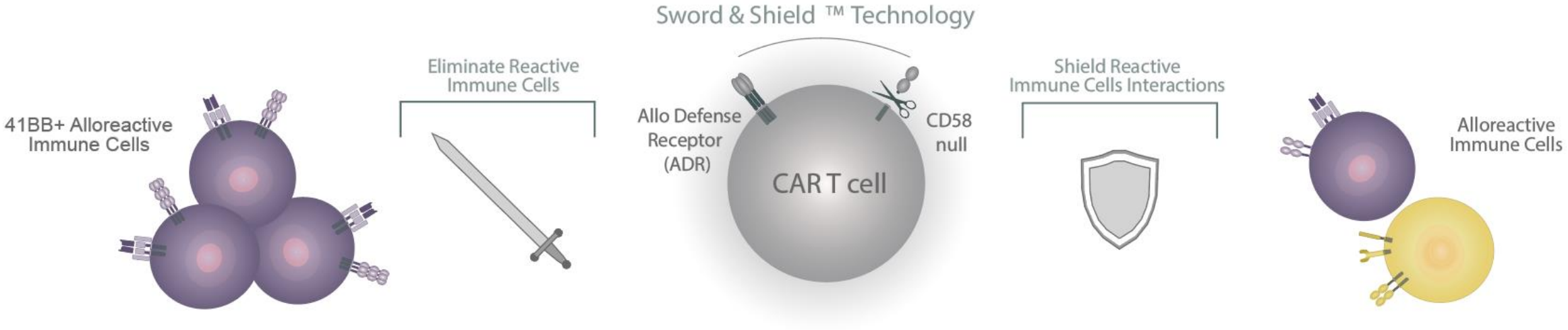


Engineered Attribute System(s):

- ✓ 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



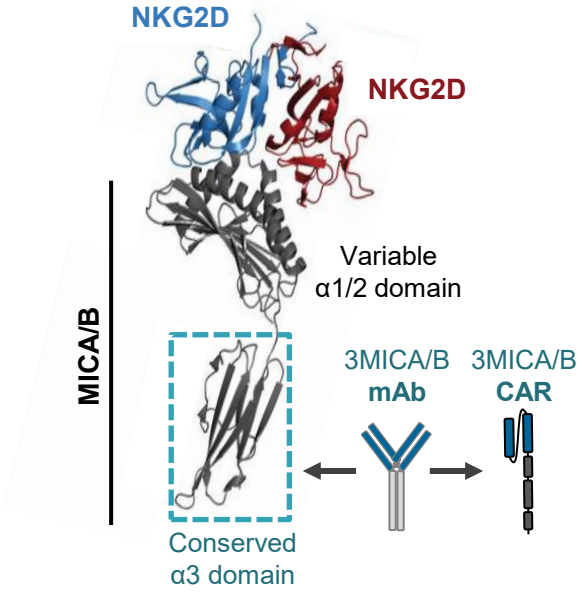
Strategy	Combination with Intense CCT	HLA-I & HLA-II Knockout	HLA-I & HLA-II Knockout + HLA-E[1]	HLA-I & HLA-II Knockout + CD47[2,3]	**Sword & Shield™ ADR[4] + CD58 Knockout[5]**
Avoid host CD8 T cells	+	+	+	+	**+++**
Avoid host CD4 T cells	+	+	+	+	**+++**
Avoid host NK cells	+	-	+/-	+/-	**+++**
Avoid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

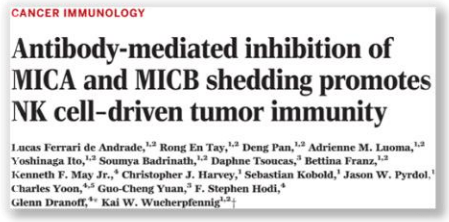
1. Li W et al. Front Immunol. 2022 Dec 2;13:1052717.
2. *Hu, X., et al. Nat Biotechnol 42, 413–423 (2024).*
3. *Hu X, et al. Nat Commun. 2023 Apr 10;14(1).*
4. *Mo F et al, Nat Biotechnol. 2021 Jan; 39(1):56-63.*
5. *Hamer Q et al. Cell Stem Cell. 2024 Sept 5;31(9):1376-1386.e8.*

Targeting MICA/B Inhibits Tumor Resistance & Unlocks Pan-Tumor Potential

FT836: Off-the-shelf anti-MICA/B CAR T-cell product candidate

Novel recognition of MICA/B α3 domain unlocks pan-tumor targeting[1,2]

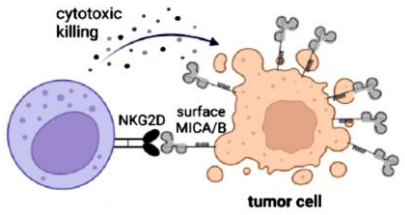


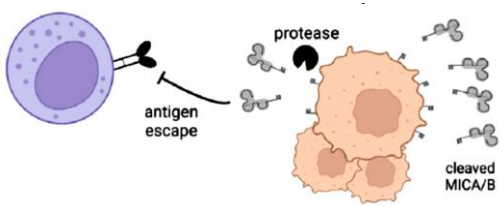

CANCER IMMUNOLOGY

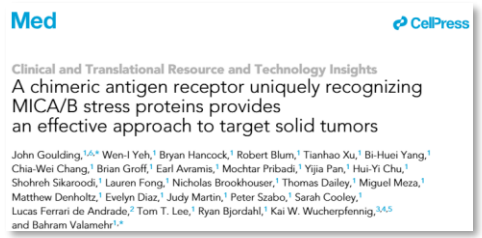
Antibody-mediated inhibition of MICA and MICB shedding promotes NK cell-driven tumor immunity

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,[3] Bettina Franz,[1,2] Kenneth F. May Jr.,[3] Christopher J. Harvey,[3] Sebastian Kobold,[3] Jason W. Pyrdol,[1] Charles Yoon,[4,5] Guo-Cheng Yuan,[2] F. Stephen Hodi,[4] Glenn Dranoff,[4,*] Kai W. Wucherpfennig[1,2,†]

MICA/B shedding is a common immune escape mechanism in cancer[3,4]






Med

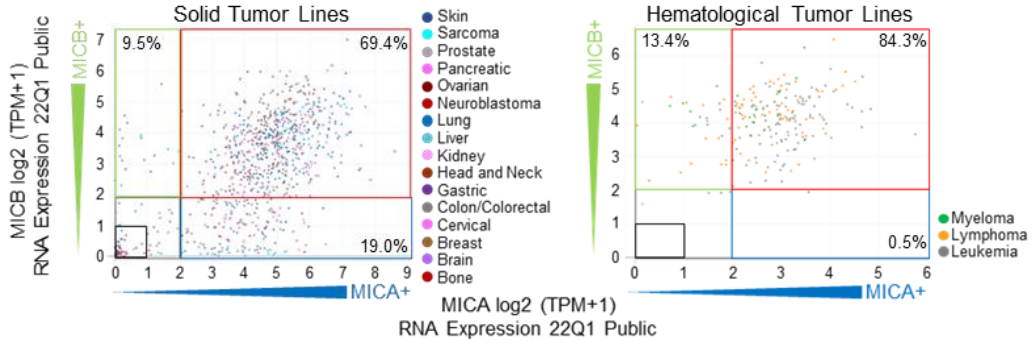
CellPress

Clinical and Translational Resource and Technology Insights

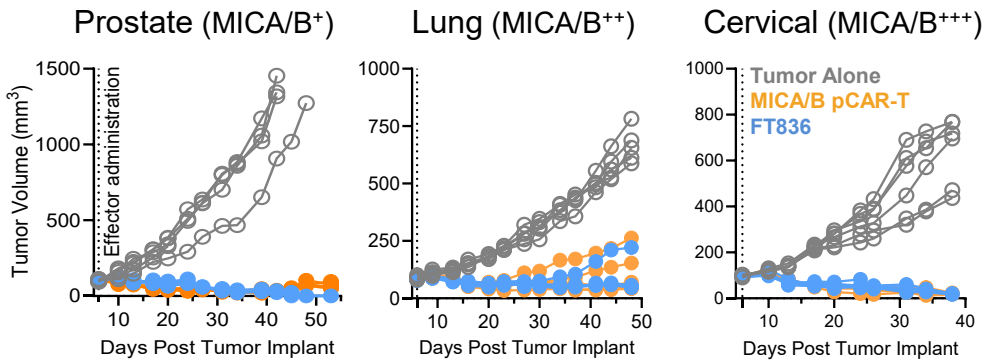
A chimeric antigen receptor uniquely recognizing MICA/B stress proteins provides an effective approach to target solid tumors

John Goulding,[1,6,*] Wen-I Yeh,[1] Bryan Hancock,[1] Robert Blum,[1] Tianhao Xu,[1] Bi-Huei Yang,[1] Chia-Wei Chang,[1] Brian Groff,[1] Earl Avramis,[1] Mochtar Pribadi,[1] Yijia Pan,[1] Hui-Yi Chu,[1] Shohreh Sikaroodi,[1] Lauren Fong,[1] Nicholas Brookhouser,[1] Thomas Dailey,[1] Miguel Meza,[1] Matthew Denholtz,[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,[3,4,5] and Bahram Valamehr[1,*]

MICA/B is widely expressed across multiple cancer indications[5]



FT836 shows broad activity across diverse xenograft tumor models



- 3MICA/B CAR activity is greater than similar NKG2D CARs
- 3MICA/B CAR is resistant to soluble cleaved MICA/B, in contrast to NKG2D
- 3MICA/B CAR provides specific tumor reactivity across cancer indications

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*

CD19/CD38 Co-Targeting Delivers Potent, Multi-Compartment Immune Ablation

FT839: Off-the-shelf anti-CD19/CD38 dual CAR T-Cell product candidate

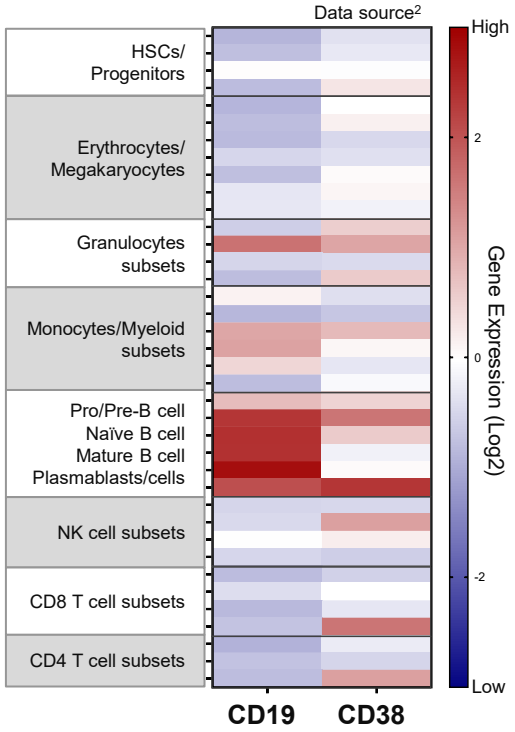
B cell lineage and activated immune cell state antigen targeting

Target Biology:

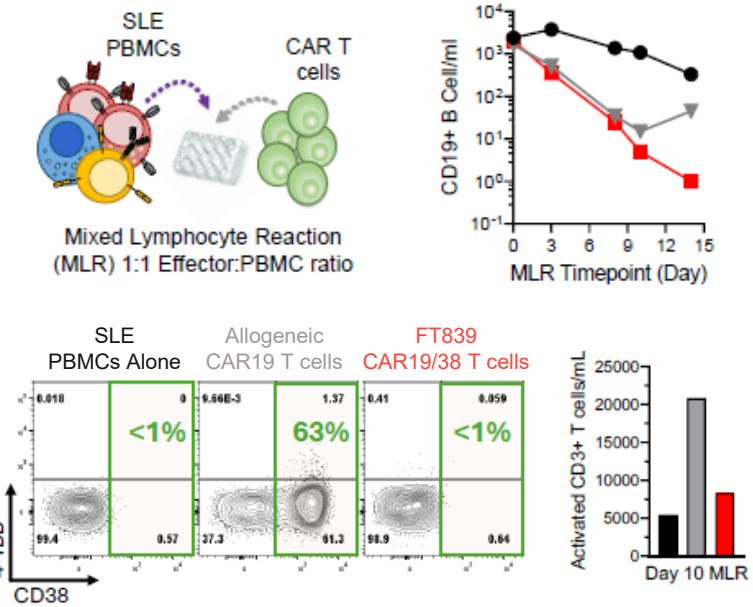
- **CD19** is a co-receptor that amplifies B-cell receptor (BCR) signaling. It plays a critical role in B cell development, activation, and survival by regulating BCR signaling.
- **CD38** is an ectoenzyme with NADase activity. It is involved in cell adhesion, signal transduction, and calcium mobilization. It also regulates metabolism and is upregulated during cell activation and differentiation.

Target Clinical Validation:

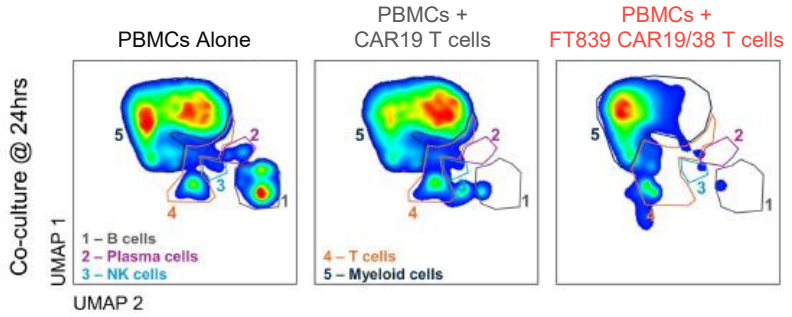
- CD19 is a common target in B cell malignancies and autoimmune diseases for CD19 directed CAR T-cell therapies.
- CD38, the target of daratumumab in multiple myeloma, is increasingly implicated in autoimmunity as a marker of pathogenic plasma cells and dysregulated T cells[1].



FT839 eliminates B cells & allo-reactive immune cells[3]



FT839 simultaneously eliminates B cells, plasma cells and activated immune cell subsets[3]



- Individual and combined specific CD19 and CD38 CAR activity provides broad and potent B and activated immune cell subset targeting capability.
- Sword & Shield™ technology, combined with CD38 CAR, enables selective elimination of activated immune cell states - T cell and myeloid - for enhanced immune reset precision

1. Yan-Ruide Li, et al. Trends in Pharmacological Sciences. Volume 45, Issue 9,2024.
2. Novershtern, Noa et al. Cell, Volume 144, Issue 2, 296 – 309. 2011.
3. J. Goulding, Annals of the Rheumatic Diseases, Volume 84, Supplement 1, 2025.

Upcoming Milestones for NxG CAR T Cell Candidates

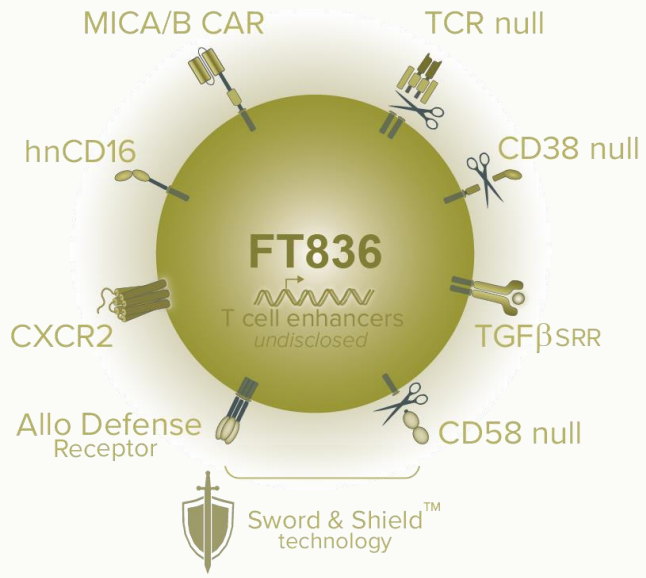
Phase 1 clinical evaluation ready in 2025

FT836 Product Candidate

Attribute Systems:

- Targeting -
  - 3MICA/B CAR
  - hnCD16 (mAb combination)
  - ADR
- Sword & Shield™ Technology
- Enhanced Biodistribution
- Enhanced Persistence

- **IND cleared July 2025**
- Planning for Phase 1 Trials:
  - Multiple tumor indications
  - Monotherapy & mAb combination
  - No lympho-conditioning
- Targeting FPI by YE 2025

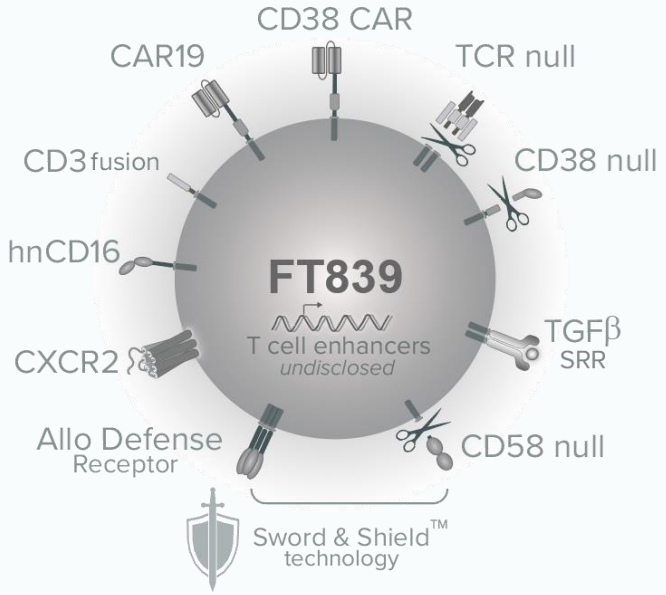


FT839 Product Candidate

Attribute Systems:

- Targeting -
  - CD19 & CD38 CAR
  - hnCD16 & CD3FR
  - ADR
- Sword & Shield™ Technology
- Enhanced Biodistribution
- Enhanced Persistence

- Anticipated IND filing by YE 2025
- Planning for Phase 1 Trials:
  - Multiple autoimmune indication
  - Hematological malignancies
  - Monotherapy & mAb combination
  - No lympho-conditioning
- Targeting FPI Q1 2026



FT522 Program

CD-19 CAR NK Cell Product Candidate

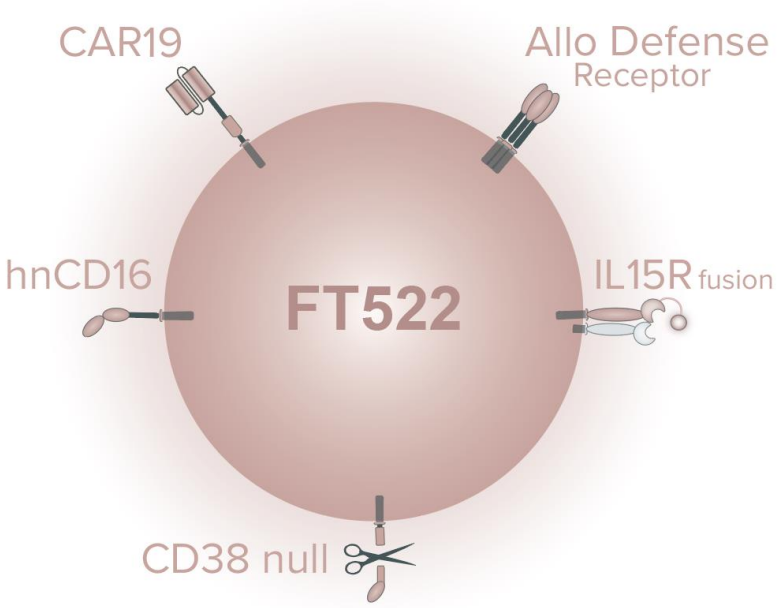
Fate THERAPEUTICS

Making Cell Therapy Accessible to All™

Multi-Antigen Targeting CAR NK Cell Armed with ADR to Avoid the Need for Conditioning Chemotherapy

FT522: Off-the-shelf anti-CD19 CAR NK cell product candidate

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



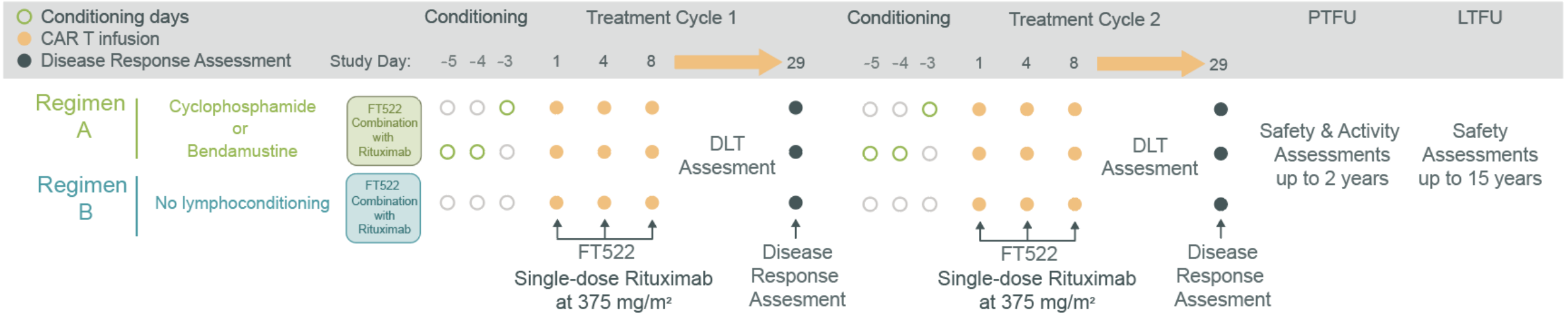
Multi antigen targeting via CD19 CAR and hnCD16, with ADR technology designed to reduce/eliminate need for conditioning chemotherapy

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*

FT522-101 Program for B-cell Lymphoma

Phase 1 Clinical Trial Design



Clinical Trial NCT05950334[1]

Proof-of-concept for Therapeutic Approach

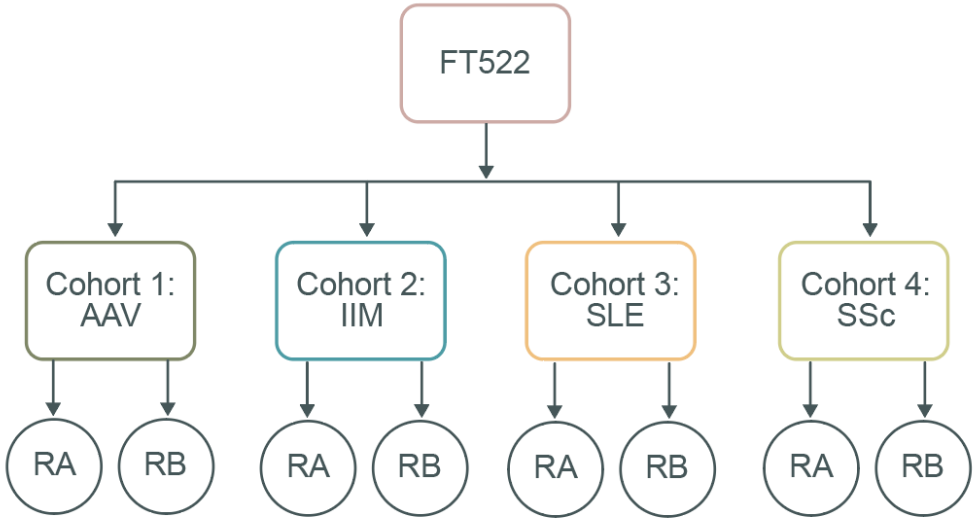
- FT522 demonstrated activity and tolerability in B cell lymphoma
- Pharmacokinetics suggest improved long term FT522 persistence without chemo-conditioning, with no observed host mediated alloreactivity
- FT522 mediated B cell depletion and reduction in disease associated antibodies
- Demonstrated proof-of-concept paves the way to expand program into auto-immune diseases

1. *https://clinicaltrials.gov/study/NCT05950334 (ClinicalTrials.gov).*

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

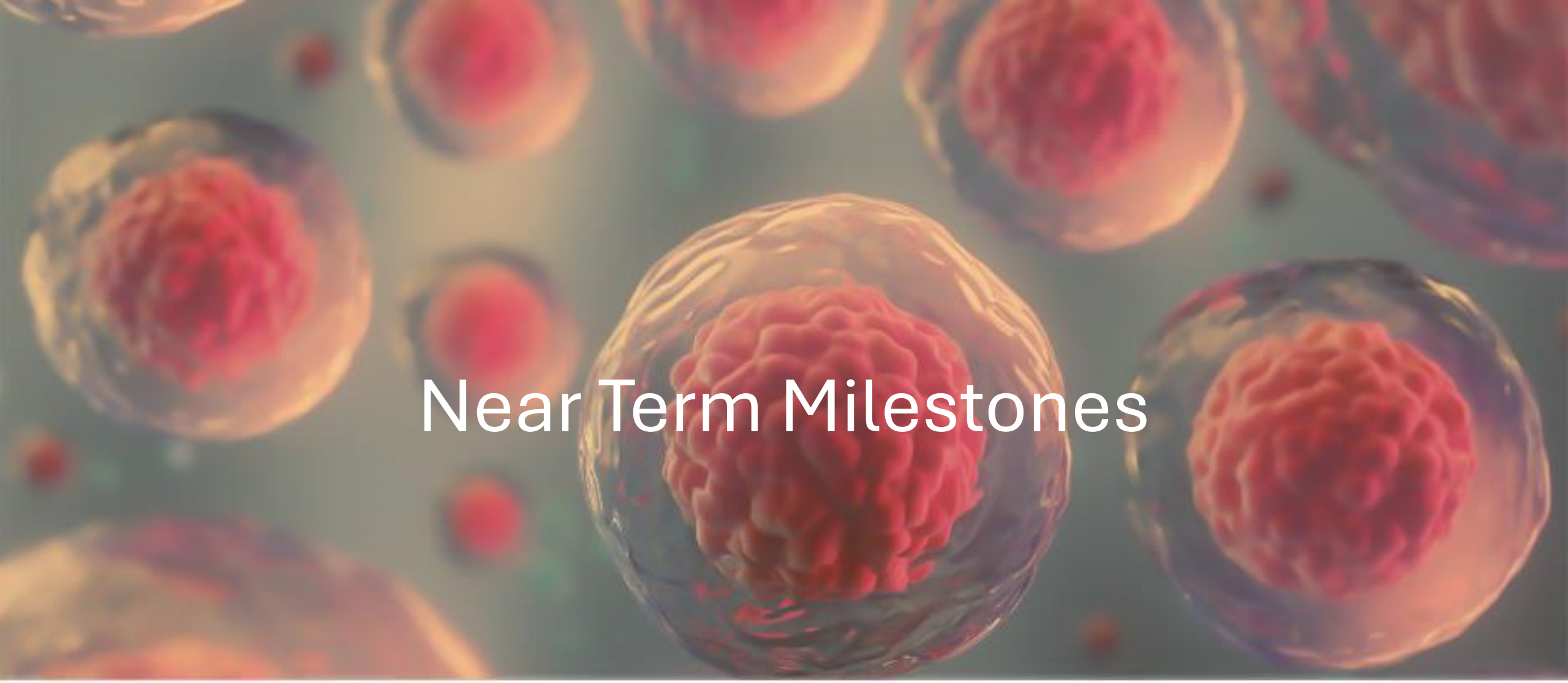
SLE = Systemic lupus erythematosus

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



Near Term Milestones

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2025 Corporate Highlights

Competitively positioned to accelerate next stage of the company

Near Term (within 6-9 months) Anticipated Milestones:

- Removal of hospitalization requirement for FT819
- Alignment with FDA on FT819 pivotal trial design in SLE
- FT819 study site expansion, including into Europe
- Enrollment of auto-immune basket indications in FT819 Phase 1
- FT825 continuing dose escalation in Reg A and Reg B across multiple solid tumors
- Enrollment of first patients in FT836 in pan tumors (IND cleared July 2025)
- Enrollment of first patients in FT839 in auto-immune and hematological malignancies (Anticipated IND filing YE2025)

Cash & Cash Equivalents **~$249M** (as of June 30, 2025)
with Projected Operating Runway through Year-End 2027

TRANSFORMING THE LIVES OF PATIENTS WITH AUTOIMMUNE DISEASES AND CANCER

Making Cell Therapies Accessible to All™

July 2025

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