TRANSFORMING THE LIVES OF PATIENTS WITH AUTOIMMUNE DISEASES AND CANCER

Making Cell Therapies Accessible to All™

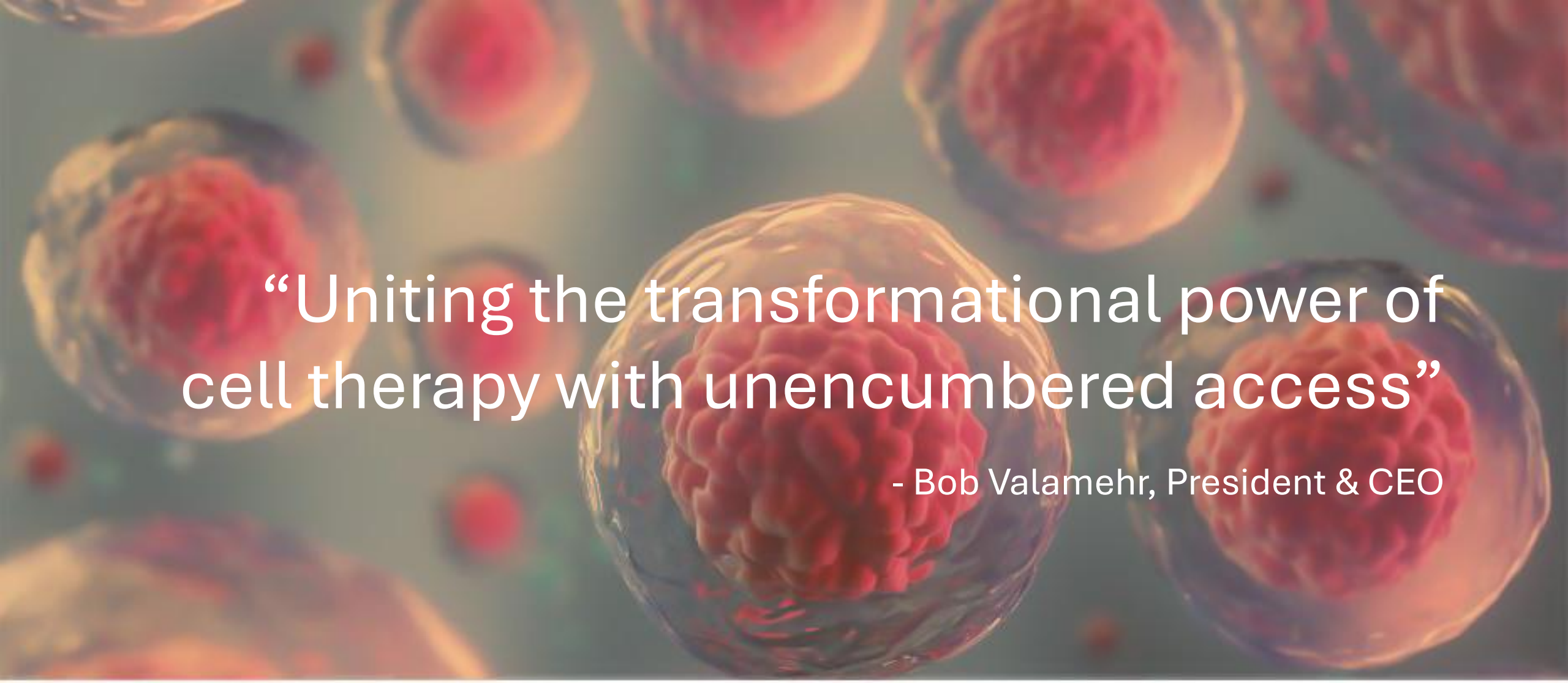
Fate THERAPEUTICS

Corporate Presentation

October 2025

Forward-Looking Statements

This presentation contains "forward-looking statements" within the meaning of the Private Securities Litigation Reform Act of 1995, including statements regarding the safety and therapeutic potential of the Company's product candidates, the advancement of and plans and timelines related to the Company's ongoing and planned clinical studies and the clinical investigation of its product candidates, the timing for the Company's receipt and announcement of data from its clinical trials and preclinical studies, the Company's clinical development and regulatory strategy, and the Company's expectations regarding progress and timelines, and potential payments under its collaboration, and the objectives, plans and goals of its collaboration with Ono Pharmaceutical, Ltd. These and any other forward-looking statements in this presentation are based on management's current expectations of future events and are subject to a number of risks and uncertainties that could cause actual results to differ materially and adversely from those set forth in or implied by such forward- looking statements. These risks and uncertainties include, but are not limited to, the risk that results observed in studies of its product candidates, including interim results and results from earlier studies, may not be predictive of final results or results observed in ongoing or future studies involving these product candidates, the risk of a delay in the initiation of, or in the enrollment or evaluation of subjects in, any clinical studies, and the risk that the Company may cease or delay manufacture, or preclinical or clinical development, of any of its product candidates for a variety of reasons (including regulatory requirements, difficulties in manufacturing or supplying the Company's product candidates, prioritization of other of its product candidates for advancement, and any adverse events or other negative results that may be observed during preclinical or clinical development). These statements are also subject to other risks and uncertainties as further detailed in the Company's most recently filed periodic report, and subsequent periodic reports filed by the Company, under the Securities Exchange Act of 1934, as amended, any of which could cause actual results to differ materially from those contained in or implied by the forward-looking statements in this presentation. The Company is providing the information in this presentation as of the date hereof and does not undertake any obligation to update any forward-looking statements contained in this presentation unless required by applicablelaw.



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

- Bob Valamehr, President & CEO

Fate THERAPEUTICS

Making Cell Therapy Accessible to All™

2025 Corporate Highlights

Competitively positioned to accelerate clinical stage development

	Milestones Achieved	Near Term Next Steps
Autoimmune Clinical Development (FT819)	✓ 10 lupus patients treated (as of Sep 25th 2025) with 4 initial sites. Nine US sites activated	Accelerate patient enrollment with activated US and exUS sites
	✓ Successful FDA interactions under RMAT designation	Gain FDA alignment on registrational design and initiate pivotal trial in 2026
	✓ Enroll basket indications	Conduct dose expansion in IIM, SSc, and ANCA vasculitis. Expand into RA and additional indications*
Next Gen Development w/ Sword & Shield™ (FT836 & FT839)	✓ Initiate FT836 (MICA/B) pan-solid tumor Ph1	Enroll first patient in 4Q 2025
	✓ Initiate FT836 in multiple myeloma IIT	Enroll first patient 1H 2026
	✓ FT839 IND enabling activities	Initiate Ph1 trial

✓ **Cash & Equivalents** ~$249M as of June 30, 2025, with projected operating runway through year-end 2027

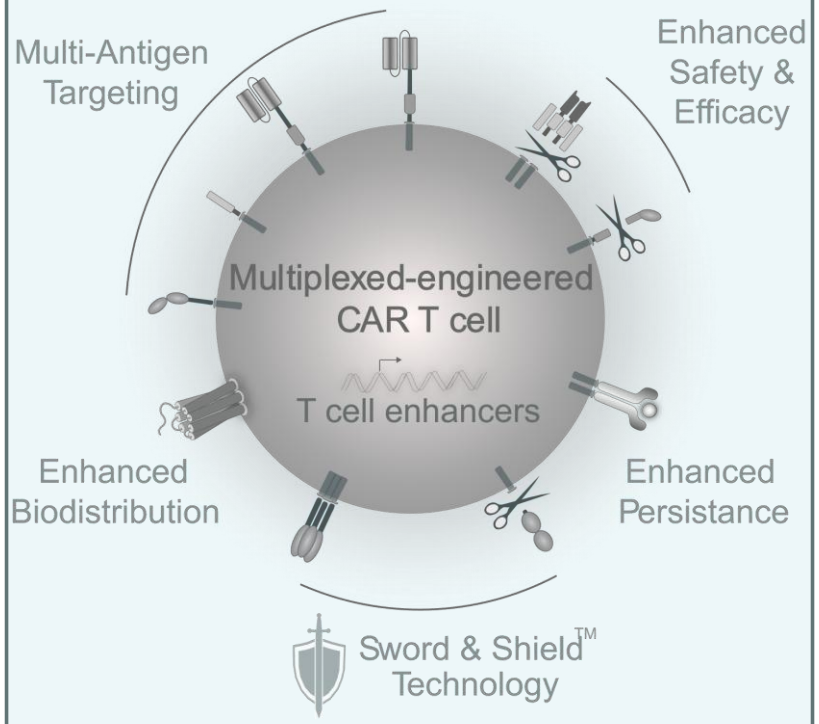
* ANCA = Antineutrophilic cytoplasmic anti-body associated vasculitis; IIM = Idiopathic inflammatory myositis; RA= Rheumatoid arthritis; SSc = Systemic Sclerosis

Vision Statement: Resetting the Paradigm - Not just the Patient

Fate THERAPEUTICS

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

Unique Living Drugs with Broad Disease Targeting Capability



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

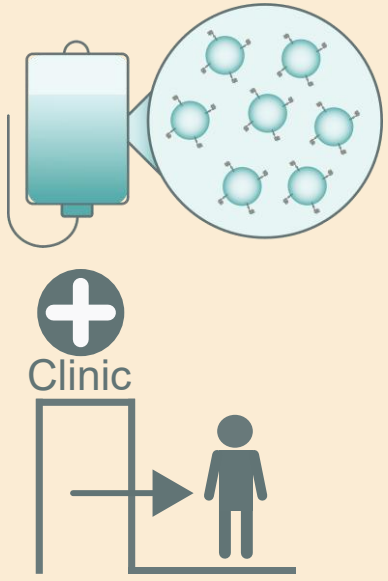
Uniform Product Profiles: consistent identity, purity and potency

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

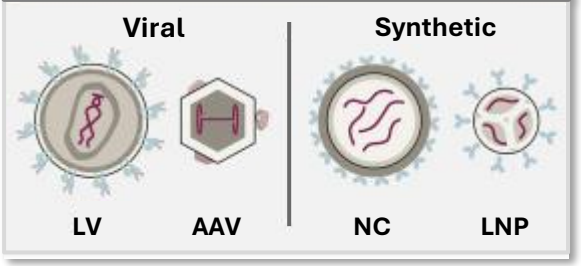
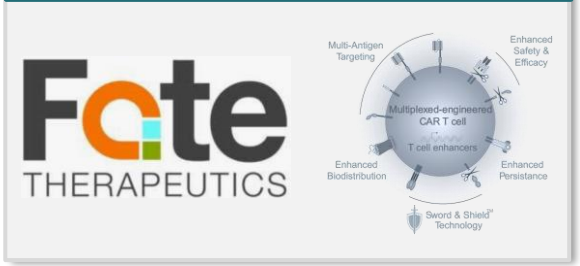
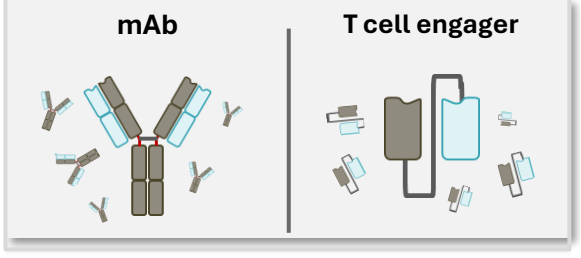
- Potential for profound efficacy in difficult-to-treat diseases
- **Healthy** starting material
- Random, variable, **per-batch** T-cell engineering
- Complex logistics
- **Multiple** dose paradigm
- Heterogeneous drug product
- Extended hospitalization
- Expensive ($$$)

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

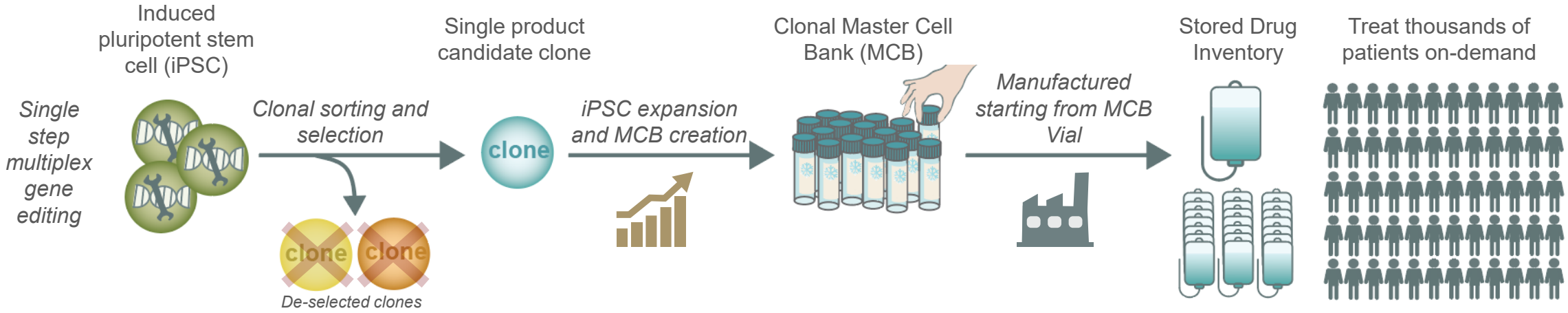
Fate's preprogrammed CAR T cells deliver potency of a living drug with uniformity of traditional biologics

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

Cellular Medicines Pipeline

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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	IND enabling studies			
FT522	CD19/CD20	Pan-Indication w/o LCC	FT522-102			
Oncology						
FT825	HER2/EGFR	Solid Tumor (s)	FT825-101		NCT06241456 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	FT836-101		NCT07216105 Enrolling	
FT839 (NxG)	CD19/CD38/BCMA/GPRC5D	Pan-Indication (Heme) w/o LCC	IND enabling studies			

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

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)

FT819 Program

CD19 Targeting CAR T-Cell Product Candidate

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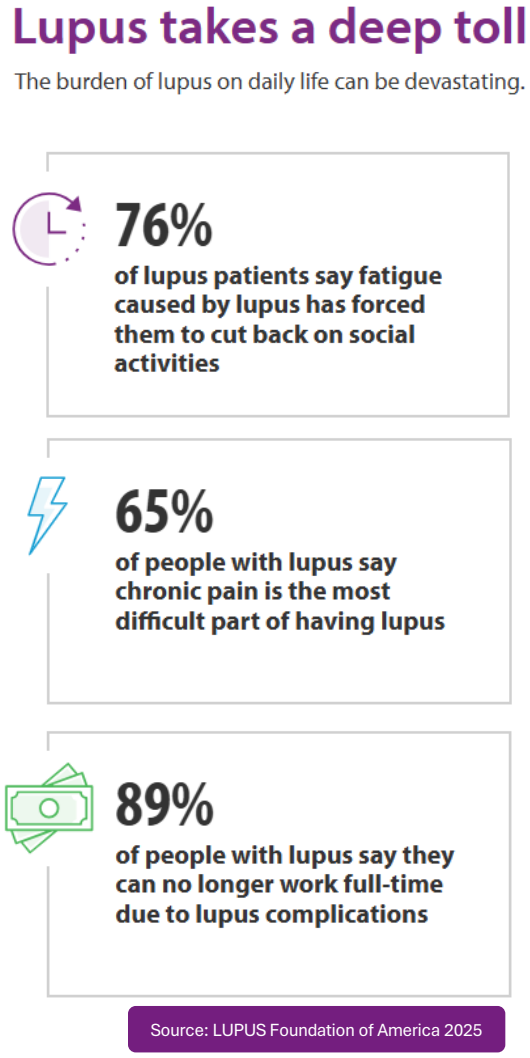
Systemic Lupus Erythematosus (SLE): A Disease of Significant Unmet Need

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]
- Four 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

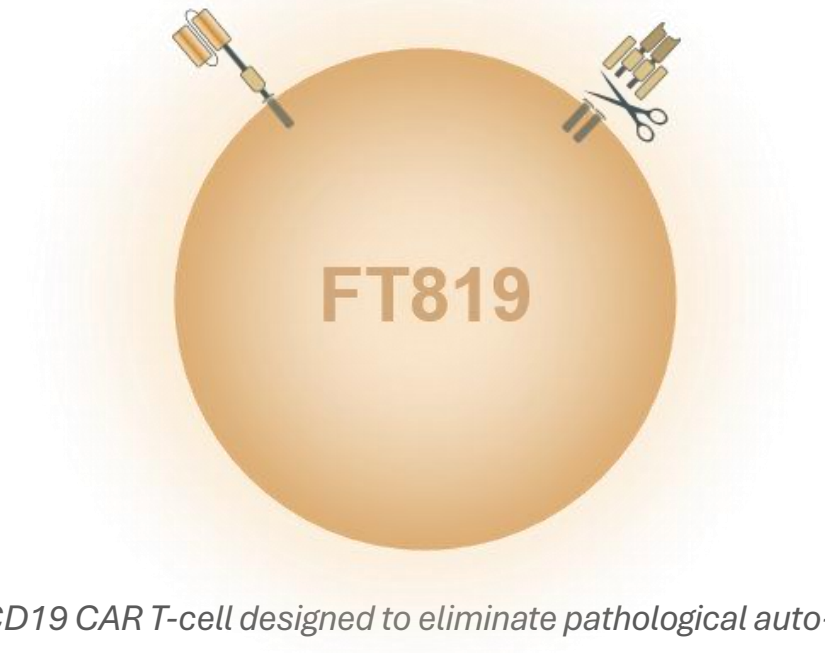
True Off-the-Shelf CAR T-Cell Drug Product

CD19 CAR

State-of-the-art CAR motif and expression control[1]

TCR null

Complete TCR disruption to prevent GvHD in allogeneic settings



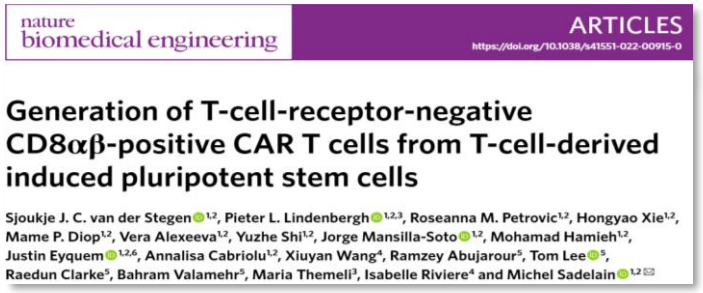
CD19 CAR T-cell designed to eliminate pathological auto-reactive B-cells with balanced efficacy and safety to establish immune reset and clinical remission[2]

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.

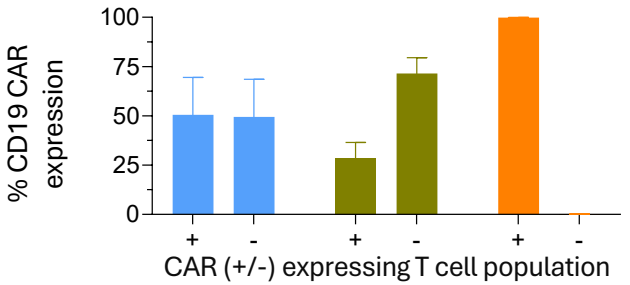
FT819 Provides Rapid and Dose Dependent B Cell Elimination

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

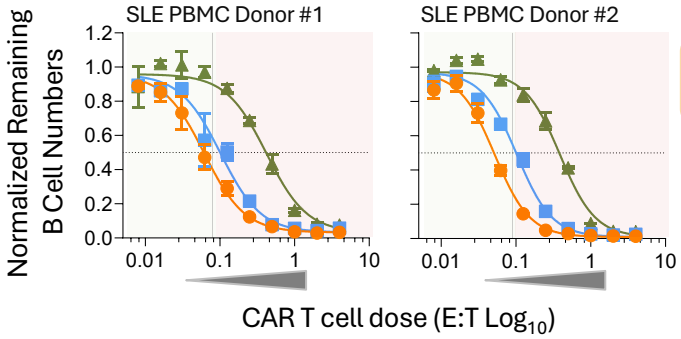
- **Autologous CAR T cell** *surrogate manufacturing process (3-14 days of T cell culture):*
  - Enriched T cells activated and transduced with CD19 CAR lentivirus
  - Standard manufacturing processes & timelines used to generate product
- **Allogeneic CAR T cell** *surrogate manufacturing process (21-35 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

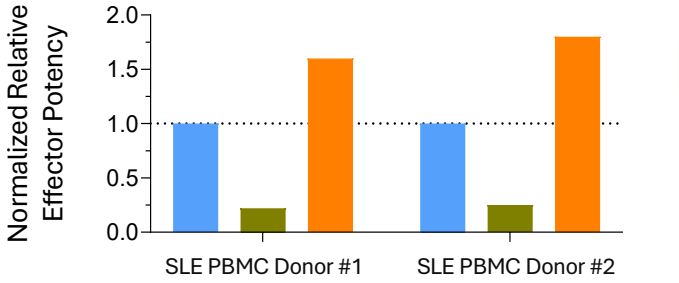


FT819 demonstrates superior CD19 CAR expression uniformity compared to autologous and allogeneic CAR T cells derived from patient or doner cells

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



Left shifted curves indicates greater potency



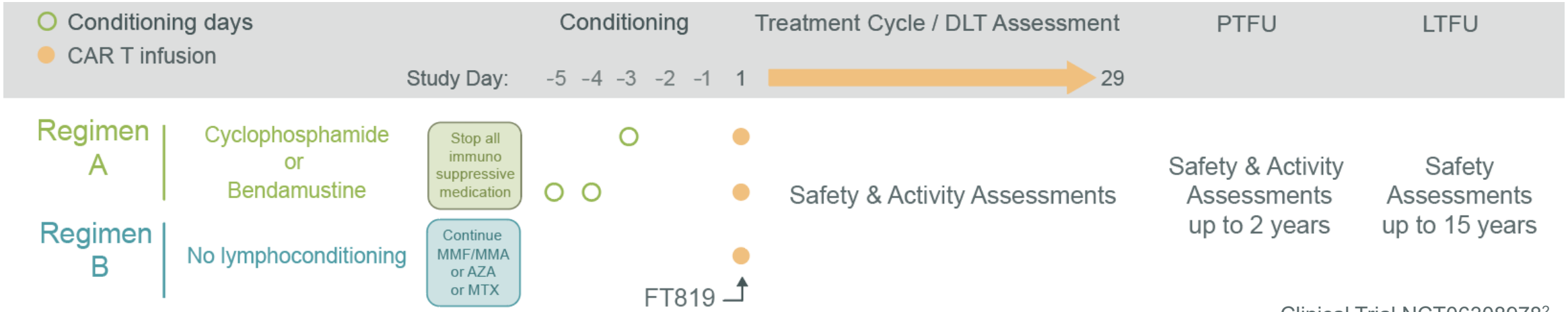
Relative potency >1 suggest greater potency

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[1,2]

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
- Protocol authorized autoimmune diseases 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 Update: Ongoing Phase 1 Study of FT819 in B-cell Mediated Autoimmune Diseases[1,2]

SLE patient baseline characteristics treated with FT819 (N=10) as of data cut off date

Essential Patient Features

- Ten patients with treatment refractory SLE enrolled & treated[1]
  - Eight with fludarabine-free, less-intensive conditioning chemotherapy (Regimen A)
  - Two without conditioning chemotherapy (on background therapy) (Regimen B)
  - Six lupus nephritis across both regimens
- All patients exhibit high disease burden at baseline
  - Median disease duration of 10 years
  - Up to 10 prior treatment failures
  - Mean baseline SLEDAI-2K of 14.4

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

	Regimen A								Regimen B	
Patient	**A1-DL1**	**A2-DL1**	**A3-DL1**	**A4-DL1[3]**	**A5-DL1[3]**	**A1-DL2**	**A2-DL2**	**A3-DL2**	**B1-DL1**	**B2-DL1**
Age, sex	28 F	22 F	29 F	57 F	32 F	28 F	19 F	41 F	23 F	23 F
BILAG domain for inclusion	**Renal**	**Renal**	Heme, **Renal**	MSK, Mucocut	**Renal**	MSK, Mucocut	Cardioresp, MSK, Mucocut	Constitut, Mucocut, **Renal**	Cardioresp	**Renal**
LN classification	III	IV	IV	NA	IV	NA	NA	III + V	NA	IV
Disease duration	~11 y	~4 y	~24 y	~34 y	~4 y	~9 y	~1 y	~19 y	~5 y	~16 y
Baseline SLEDAI-2K	20	20	14	14	8	18	16	9	8	17
Concomitant SLE therapies	GC, HCQ	HCQ	GC, HCQ	GC	GC, HCQ	HCQ	GC, HCQ	HCQ	GC, HCQ, MMF	GC, HCQ, MMF
Prior Therapies	7	8	8	4	7	10	3	7	5	8
*B-cell targeted therapy **bolded**	AZA, **BEL**, GC, HCQ, MMF, **RTX**, TAC	ANI, **BEL**, CY, GC, HCQ, MMF, MTX, **RTX**	AZA, **BEL**, CY, GC, HCQ, MMF, MTX, **RTX**	**BEL**, GC, HCQ, MMF	CY,GC, HCQ, MMF, **OBI**, **RTX**, TAC	ANI, AZA, **BEL**, CY, GC, HCQ, IVIG, MMF, MTX, UST	ANI, GC, HCQ	AZA, **BEL**, CY, GC, HCQ, IVIG, MTX	CY, GC, HCQ, MMF, **RTX**	AZA, **BEL**, CY, GC, HCQ, MMF, **OBI**, VOC
Conditioning	Benda	CY	CY	Benda	CY	CY	Benda	CY	None	None

ANI = anifrolumab; AZA = azathioprine; BEL = belimumab; Benda = bendamustine; BILAG = British Isles Lupus Assessment Group; Cardioresp = cardiorespiratory; Constitut = constitutional; CY = cyclophosphamide; DL = dose level; F = female; GC = glucocorticoids; HCQ = hydroxychloroquine; Heme = hematological; IVIG = intravenous immunoglobulin; LN = lupus nephritis; MMF = mycophenolate mofetil; MSK = musculoskeletal; MTX = methotrexate; Mucocut = mucocutaneous; NA = not applicable; OBI = obinutuzumab; RTX = rituximab; SLE = systemic lupus erythematosus; SLEDAI-2K = Systemic Lupus Erythematosus Disease Activity Index 2000; TAC = tacrolimus; UST = ustekinumab; VOC = voclosporin.

1. *Fazeli P, et al. Arthritis Rheumatol.* 2025; 77 (suppl 9).
2. *https://clinicaltrials.gov/study/NCT06308978 (ClinicalTrials.gov)*
3. *Data not reported for patients A4-DL1 and A5-DL1 as have < 1 month follow up at time of 25th Sep 2025 data cutoff*

FT819-102 Update: Ongoing Phase 1 Study of FT819 in B-cell Mediated Autoimmune Diseases[1,2]

Continuation of differentiated safety data – select adverse events of interest (N=8)

Continued absence of high-grade CRS, No ICANS, and No DLTs observed in FT819 treated patients with at least 1 month follow up[3]

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

	Regimen A						Regimen B	
Patient (conditioning)	**A1-DL1 (Benda)**	**A2-DL1 (CY)**	**A3-DL1 (CY)**	**A1-DL2 (CY)**	**A2-DL2 (Benda)**	**A3-DL2 (CY)**	**B1-DL1 (none)**	**B2-DL1 (none)**
CRS	-	-	-	Grade 2	Grade 1	Grade 1	-	-
Cytopenia*	Grade 2	Grade 4	-	Grade 4	-	-	Grade 2	-
Infection Grade ≥3†	-	-	-	Grade 3	-	-	Grade 3	-

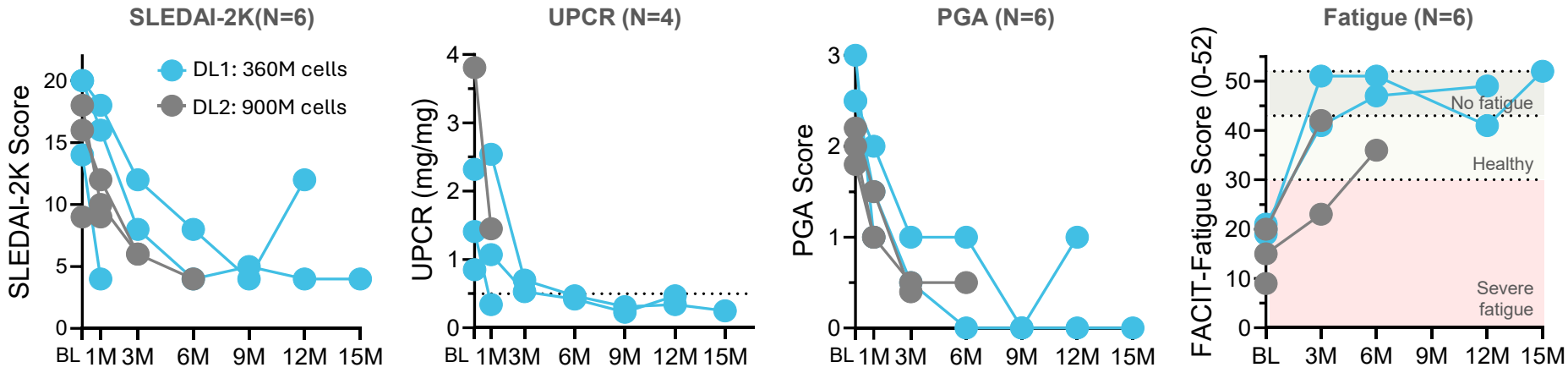
Benda = bendamustine; CRS = cytokine release syndrome; CY = cyclophosphamide; DLT = dose-limiting toxicity; GvHD = graft-versus-host-disease; ICANS = immune effector cell-associated neurotoxicity syndrome.
Patients having more than one AE within a preferred term (PT) are counted only once for that PT at the maximum severity.
* Cytopenia includes any PTs of anaemia, leukopenia, neutropenia, neutrophil count decreased, lymphopenia, pancytopenia, and thrombocytopenia.
† Infection includes influenza and urinary tract infection
Regimen A = Conditioning prior to treatment with either Benda or Cy; Regimen B = no conditioning chemotherapy.
DL = Dose level; DL1 = 360M cells; DL2 = 900M cells.
Adverse events graded per CTCAEv5.

1. *Fazeli P, et al. Arthritis Rheumatol.* 2025; 77 (suppl 9).
2. *https://clinicaltrials.gov/study/NCT06308978 (ClinicalTrials.gov)*
3. *Data cutoff 25th Sep 2025*

FT819 Treatment Demonstrates Early and Sustained Improvement in SLE disease

Regimen A: One-time FT819 treatment with less intensive conditioning chemotherapy[1,2]

Durable (≥15 month) SLE disease remission and complete renal response in LN patients with single dose of FT819 with less intensive conditioning[1,3]



BL = baseline; DL= dose level; FACIT = Functional Assessment of Chronic Illness Therapy; M = month; PGA = Physician Global Assessment; SLEDAI-2K = Systemic Lupus Erythematosus Disease Activity Index 2000; UPCR = urine protein-to-creatinine ratio.

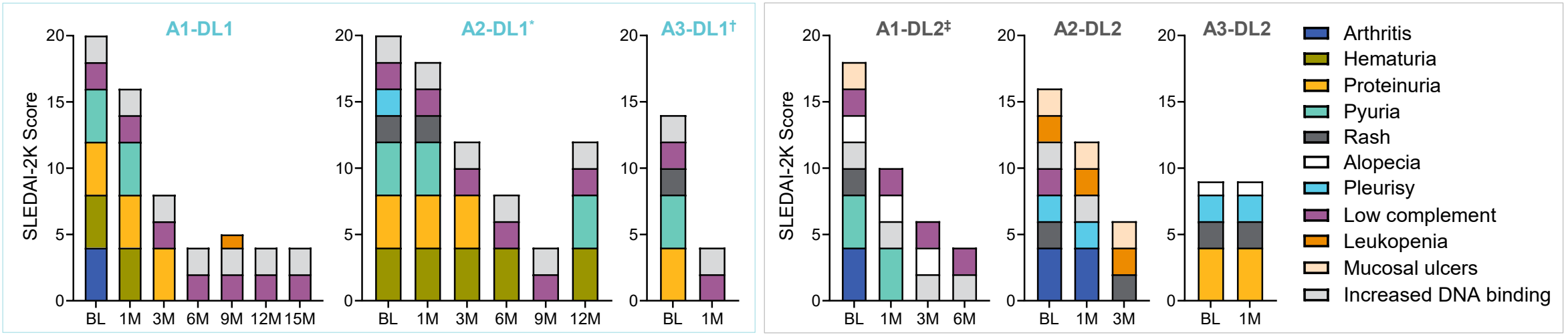
- ✓ Decrease in disease burden with less intensive conditioning chemotherapy is uniquely demonstrated
- ✓ Completed dose escalation and now enrolling in DL1 & DL2 expansion
- ✓ No DLT, CRS, GvHD or ICANS across 2 dose levels
- ✓ Reduced hospitalization, increasing patient accessibility
- ✓ On-demand CAR T-cell delivery is enabled by scalable manufacturing from a master cell bank

1. *Fazeli P, et al. Arthritis Rheumatol.* 2025; 77 (suppl 9).
2. *https://clinicaltrials.gov/study/NCT06308978 (ClinicalTrials.gov)*
3. *Data cutoff 25th Sep 2025*

FT819 Treatment Demonstrates Early and Sustained Improvement in SLE disease

Regimen A: One-time FT819 treatment with less intensive conditioning chemotherapy[1,2]

SLEDAI-2K composition scores per patient at baseline (pre-FT819) and at specified study time points[1,3]



* A2-DL1 resumed mycophenolate at ~7.5 months having previously been on this therapy for >4 years prior to receiving CAR T-cell therapy. †A3-DL1 discontinued after the 1-month visit due to inability to meet study requirements.

‡ A1-DL2 resumed anifrolumab at ~2 months having previously been on this therapy for 3 years prior to receiving CAR T-cell therapy.BL = baseline; DL= dose level; M = month; SLEDAI-2K = Systemic Lupus Erythematosus Disease Activity Index 2000. Data cutoff date 25th September 2025

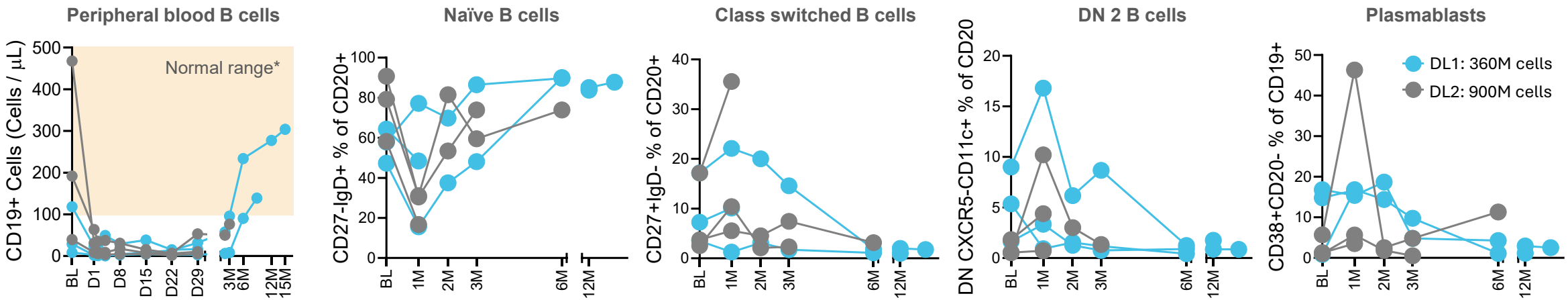
- ✓ Of the 8 patients who received FT819 with or without fludarabine-free conditioning and had at least 1 month of follow-up, early improvements were observed in disease activity (SLEDAI-2K, PGA).
- ✓ Patients with lupus nephritis (LN) showed reductions in urine protein-to-creatinine ratio (UPCR), and all patients experienced meaningful reductions in fatigue.

1. *Fazeli P, et al. Arthritis Rheumatol.* 2025; 77 (suppl 9).
2. *https://clinicaltrials.gov/study/NCT06308978 (ClinicalTrials.gov)*
3. *Data cutoff 25th Sep 2025*

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

Peripheral blood and tissue residing B cell elimination results in the repopulation of naïve B cell subsets

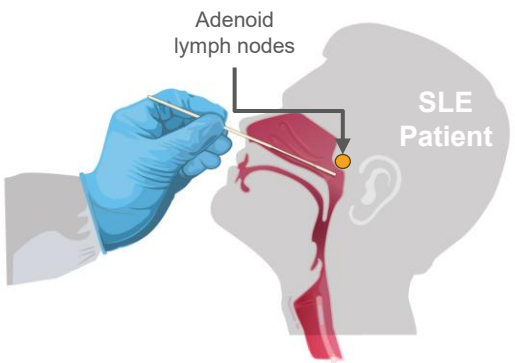
Effective depletion of pathogenic B cell populations & replacement with naïve subsets to normal levels[1-3]



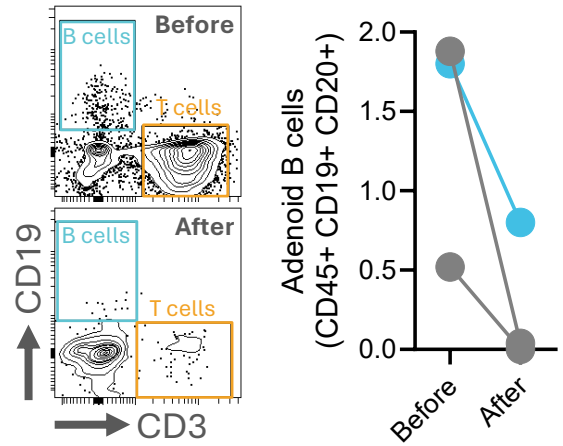
Graphs show measurements across study timepoints, BL = baseline, M=month

- ✓ 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 marrow[1]
- ✓ 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

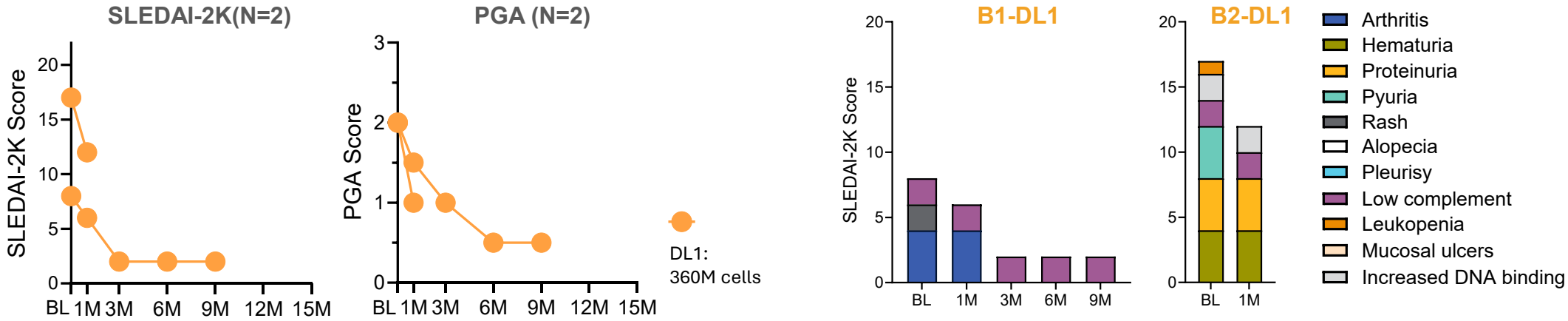


1. *Tuncel J, at al. Arthritis Rheumatol.* 2025; 77 (suppl 9).
2. *https://clinicaltrials.gov/study/NCT06308978 (ClinicalTrials.gov)*
3. *Data cutoff 25th Sep 2025*

Sustained Response in SLE Achieved Without Lympho-Conditioning

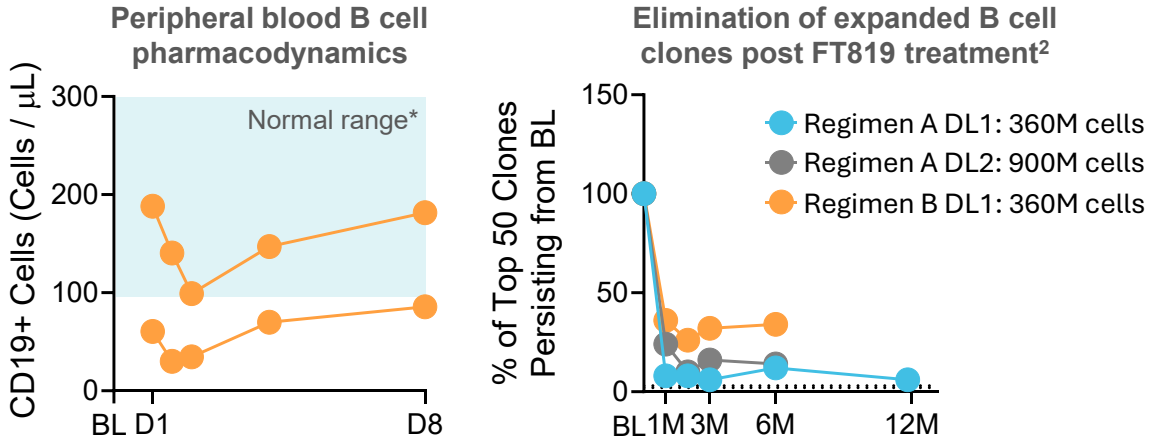
Regimen B: FT819 (DL1) as add-on to maintenance therapy exhibit improvement in disease activity

SLEDAI-2K composition scores per patient at baseline (pre-FT819) and at specified study time points[1-4]



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.



1. *Fazeli P, et al. Arthritis Rheumatol.* 2025; 77 (suppl 9).
2. *Tuncel J, at al. Arthritis Rheumatol.* 2025; 77 (suppl 9).
3. *https://clinicaltrials.gov/study/NCT06308978 (ClinicalTrials.gov)*
4. *Data cutoff 25th Sep 2025*

FT819-102 Summary and Next Steps

- True off-the-shelf CAR T that addresses the key challenges faced by autologous and allogenic cellular therapies.
- Preliminary data supports durable clinical activity with reduced or no conditioning and potential for combination with maintenance therapy without conditioning.
- > 60 patients treated across FT819-101 & FT819-102 programs; no ICANs, GvHD or CRS > Grade 2, underscoring a differentiated safety profile.
- Ongoing trial in ANCA vasculitis, Myositis (DM/PM/IMNM), SLE, and Systemic Sclerosis (ages 12-70 years); allows redosing after relapse or inadequate response.
- To date 10 patients treated across 4 initial sites as of data cutoff 25th September; 9 US sites currently activated, with expansion into academic & community sites; and ex US activation by YE 2025.
- RMAT-designated, with pivotal trial design discussion planned with FDA by YE 2025.
- Favorable safety with no dose limiting toxicities, supporting same day outpatient administration.

ANCA = Antineutrophilic cytoplasmic anti-body associated vasculitis; DM = Dermatomyositis; PM = polymyositis; IMNM = Immune mediated necrotizing myopathy; SLE = Systemic Lupus Erythematosus; SSc = Systemic Sclerosis

FT825 Program

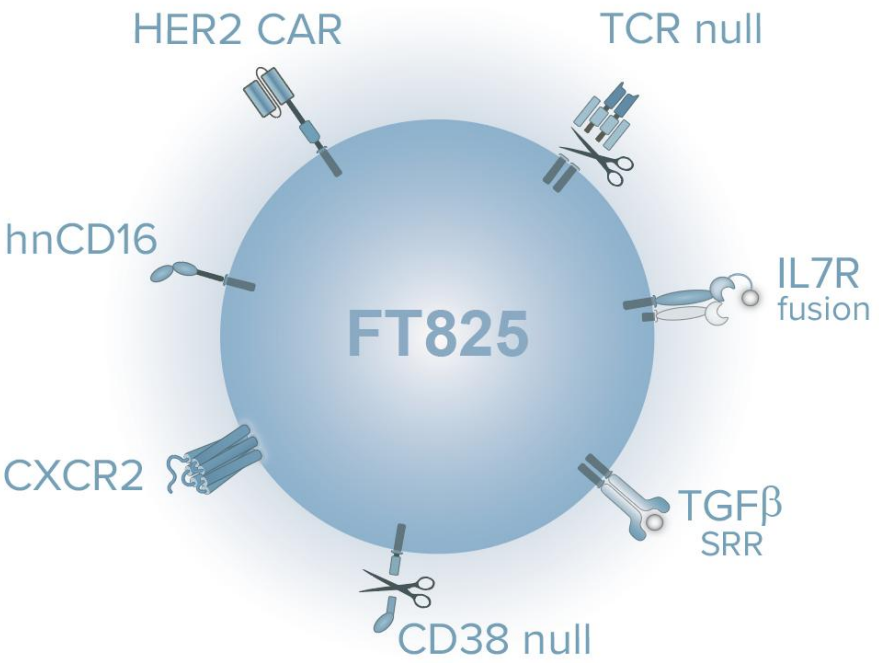
HER2 Targeting CAR T-Cell Product Candidate

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

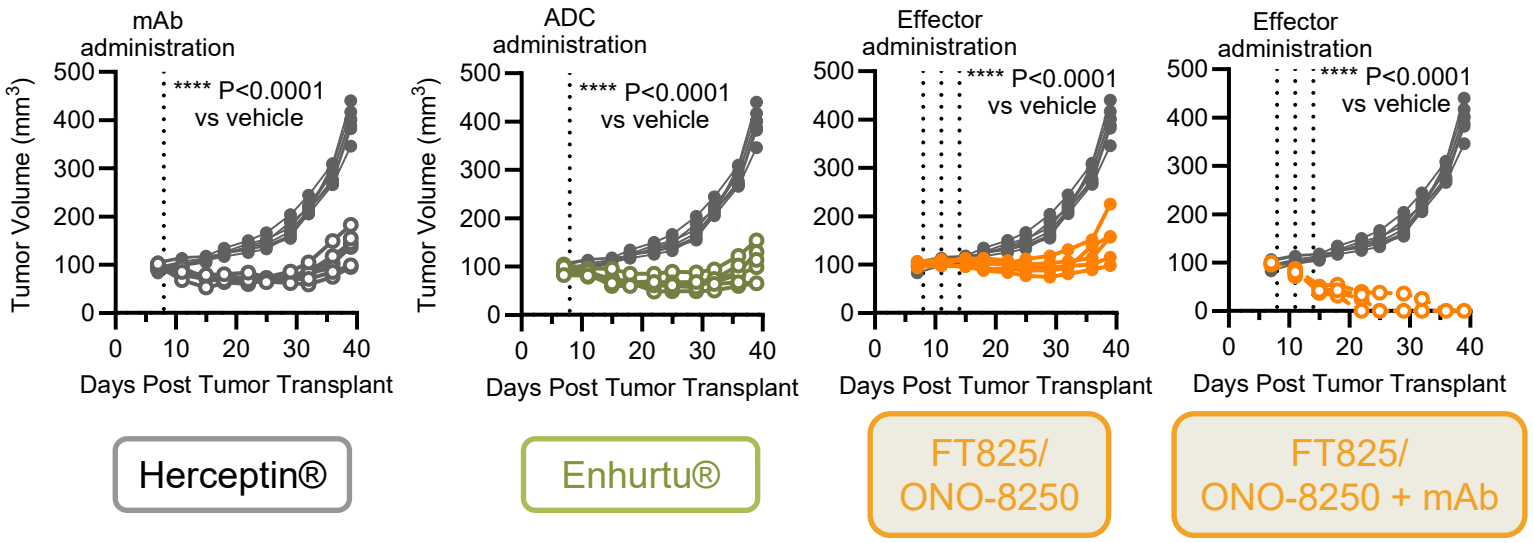
Preferential tumor targeting of HER2 by iPSC-derived CAR T cells engineered to overcome multiple barriers to solid tumor efficacy

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

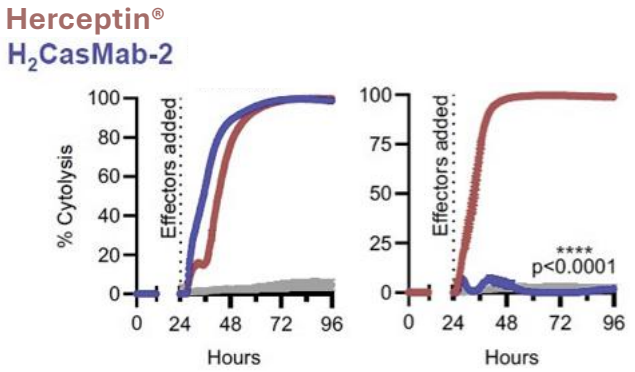
Novel Cancer-Specific CAR Binder Limits Off Tumor Toxicity

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



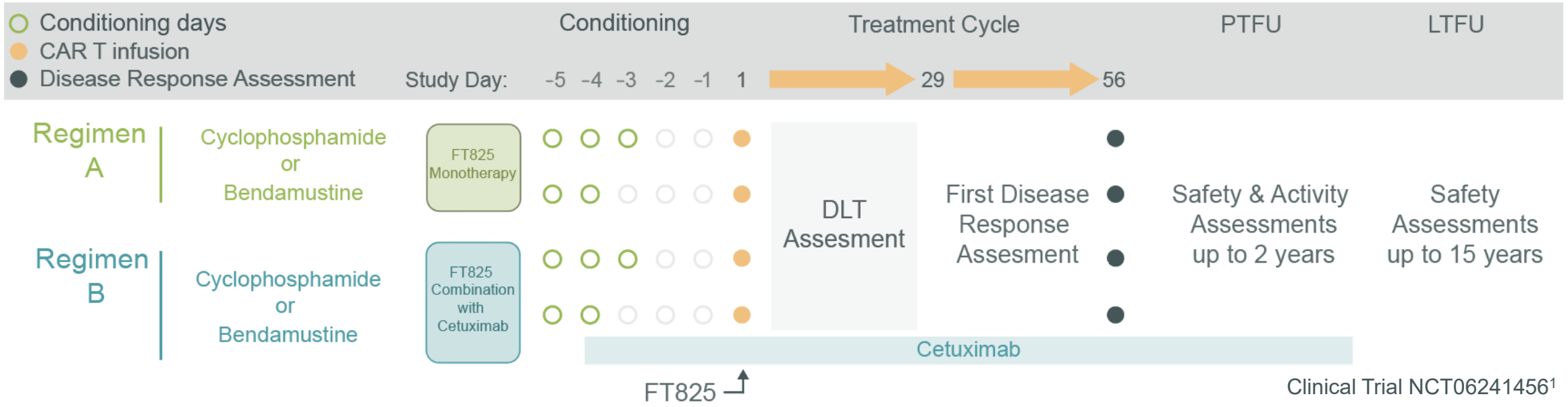
	Herceptin®	H_2CasMab-2
On Target **On** Tumor	✓	✓
On Target **Off** Tumor[2]	✗	✓

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

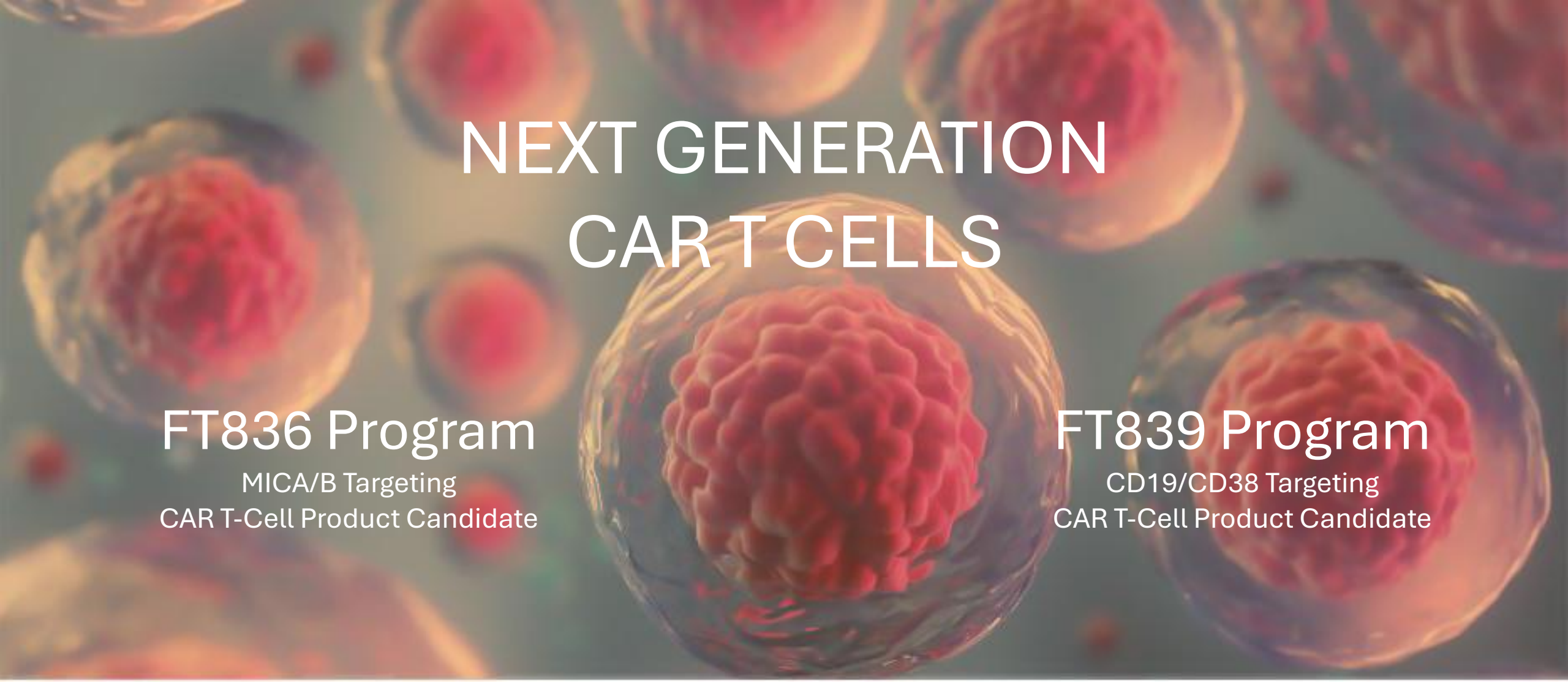
Phase 1 Study: FT825/Ono0825 with/without monoclonal antibody combination (NCT06241456)



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

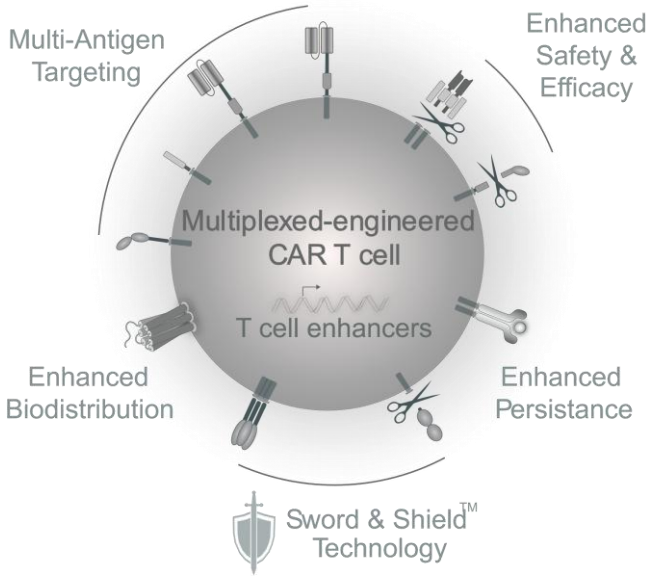
Overcoming Multiple Tumor Challenges Across Diverse Tumors Indications

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

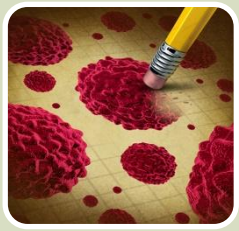
- Autoimmune, hematological malignancies & inflammatory diseases arise from dysregulated T, B and myeloid cell function across secondary and tertiary immune sites.
- Current therapies offer broad immune suppression or narrowly target specific cells, frequently falling short of effective immune control.

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

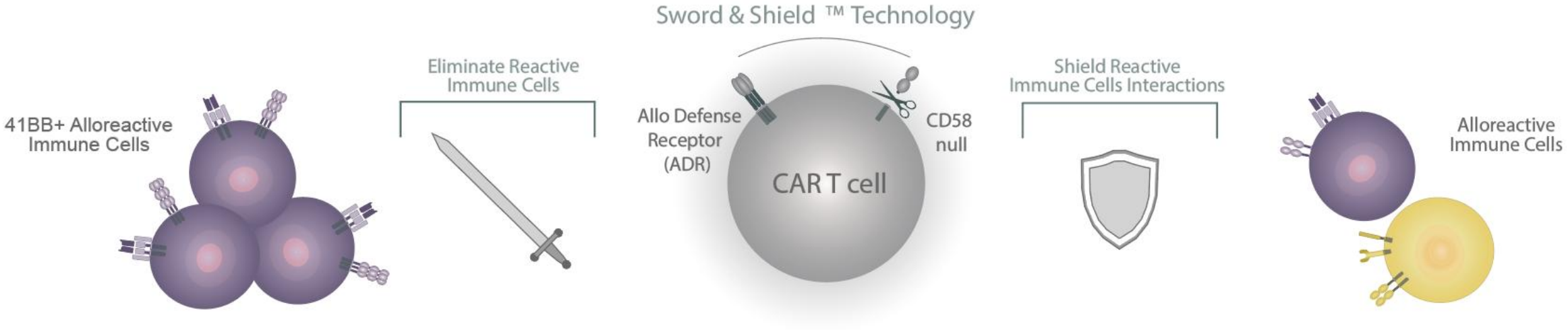


Engineered Attribute System(s):

- ✓ 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



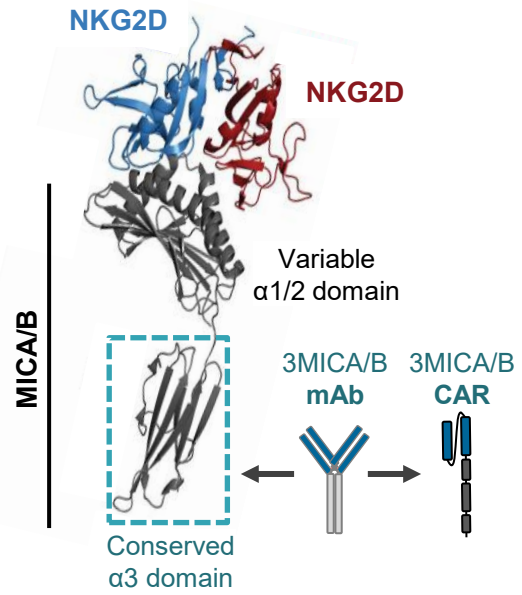
Strategy	Combination with Intense CCT	HLA-I & HLA-II Knockout	HLA-I & HLA-II Knockout + HLA-E[1]	HLA-I & HLA-II Knockout + CD47[2,3]	**Sword & Shield™ ADR[4] + CD58 Knockout[5]**
Avoid host CD8 T cells	+	+	+	+	**+++**
Avoid host CD4 T cells	+	+	+	+	**+++**
Avoid host NK cells	+	-	+/-	+/-	**+++**
Avoid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

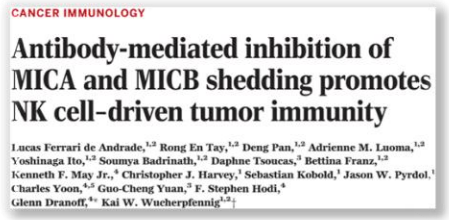
1. Li W et al. Front Immunol. 2022 Dec 2;13:1052717.
2. *Hu, X., et al. Nat Biotechnol 42, 413–423 (2024).*
3. *Hu X, et al. Nat Commun. 2023 Apr 10;14(1).*
4. *Mo F et al, Nat Biotechnol. 2021 Jan; 39(1):56-63.*
5. *Hamer Q et al. Cell Stem Cell. 2024 Sept 5;31(9):1376-1386.e8.*

Targeting MICA/B Overcomes Tumor Evasion & 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 recognition[1,2]

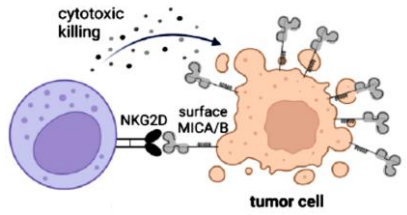


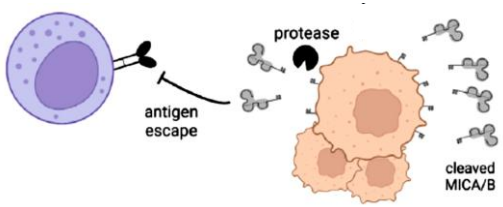

CANCER IMMUNOLOGY

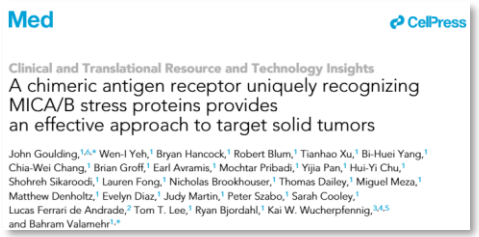
Antibody-mediated inhibition of MICA and MICB shedding promotes NK cell–driven tumor immunity

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

MICA/B shedding is a common immune evasion mechanism utilized by cancer[3,4]






Med

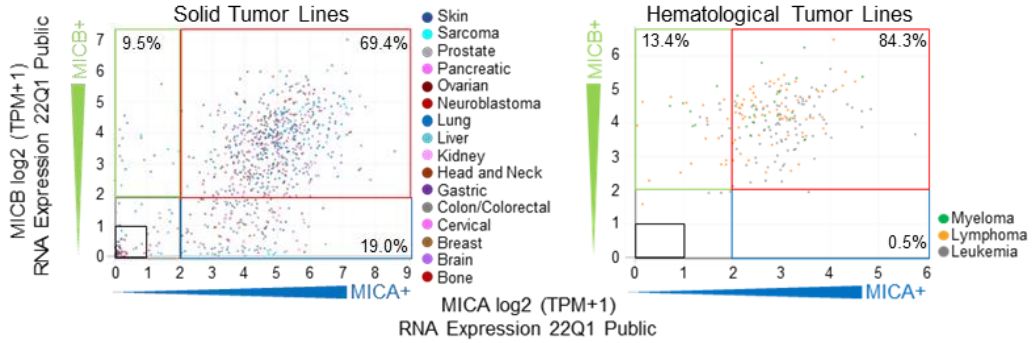
CellPress

Clinical and Translational Resource and Technology Insights

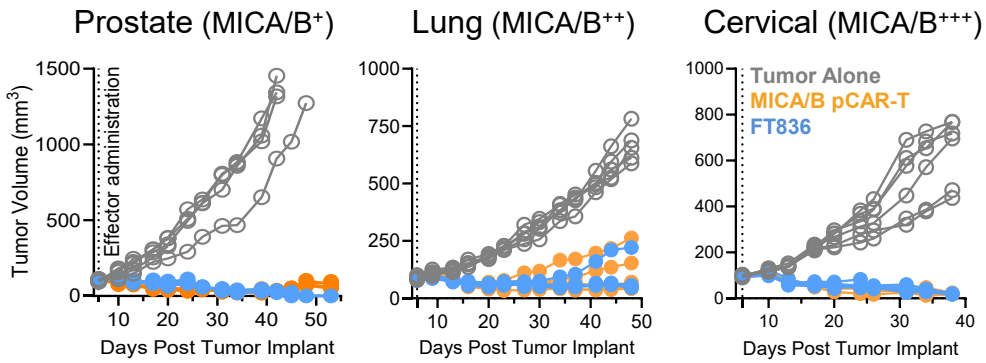
A chimeric antigen receptor uniquely recognizing MICA/B stress proteins provides an effective approach to target solid tumors

John Goulding, Wen-I Yeh, Bryan Hancock, Robert Blum, Tianhao Xu, Bi-Huei Yang, Chia-Wei Chang, Brian Groff, Earl Avramis, Mochtar Pribadi, Yijia Pan, Hui-Yi Chu, Shohreh Sikaroodi, Lauren Fong, Nicholas Brookhouser, Thomas Dailey, Miguel Meza, Matthew Denholtz, Evelyn Diaz, Judy Martin, Peter Szabo, Sarah Cooley, Lucas Ferrari de Andrade, Tom T. Lee, Ryan Bjordahl, Kai W. Wucherpfennig, and Bahram Valamehr

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



FT836 shows broad activity across diverse xenograft tumor models



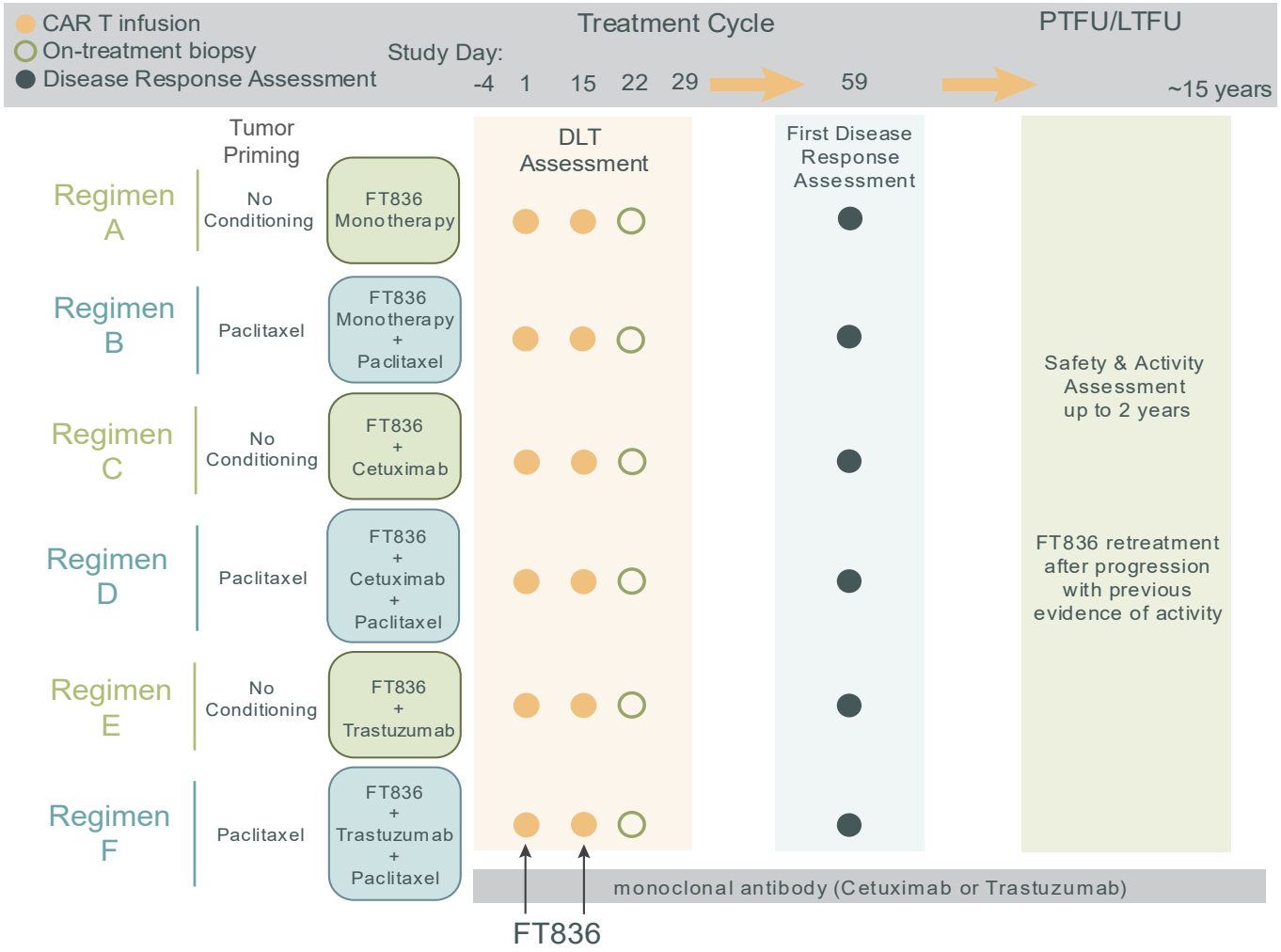
- 3MICA/B CAR activity is greater than similar NKG2D CARs
- 3MICA/B CAR is resistant to soluble cleaved MICA/B, in contrast to NKG2D
- 3MICA/B CAR provides specific tumor reactivity across cancer indications

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*

Phase 1 FT836-101 Study in Pan Solid Tumor Open to Enrollment[1]

Evaluation of FT836 with/without monoclonal antibody combination with optional Paclitaxel priming (NCT07216105)

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 pre-programmed 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 following inadequate response or relapse.
- Protocol authorized solid tumors include;
  - Breast (BC); Colorectal (CRC); Ovarian (OVC); Head & Neck (HNSCC); Endometrial (EC); Gastric (GEJ) & Lung (NSCLC).

Clinical Trial NCT07216105[1]

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

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

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

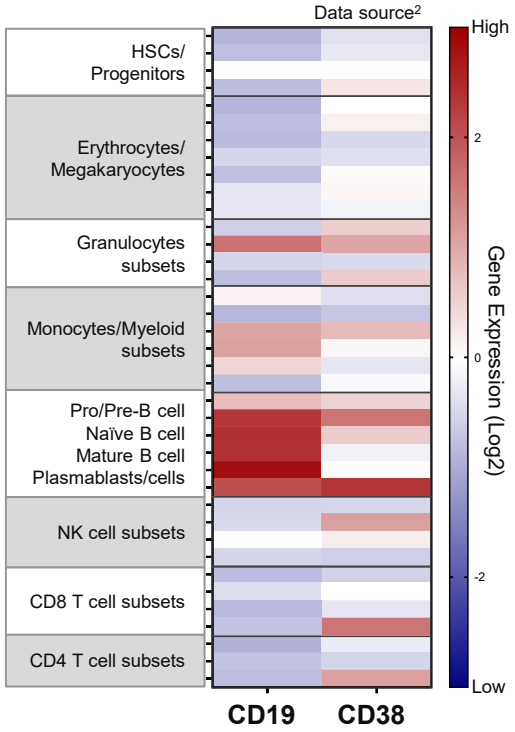
B cell lineage and activated immune cell state antigen targeting

Target Biology:

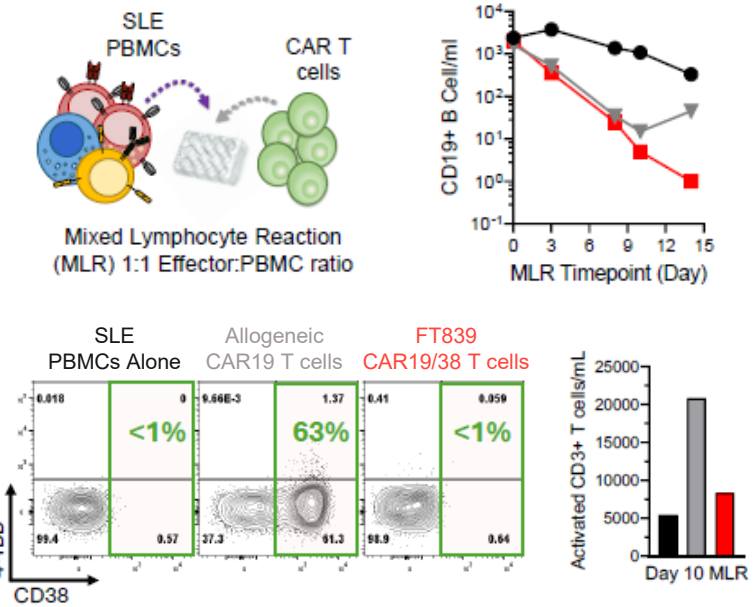
- **CD19** is a co-receptor that amplifies B-cell receptor (BCR) signaling. It plays a critical role in B cell development, activation, and survival by regulating BCR signaling.
- **CD38** is an ectoenzyme with NADase activity. It is involved in cell adhesion, signal transduction, and calcium mobilization.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

Target Clinical Validation:

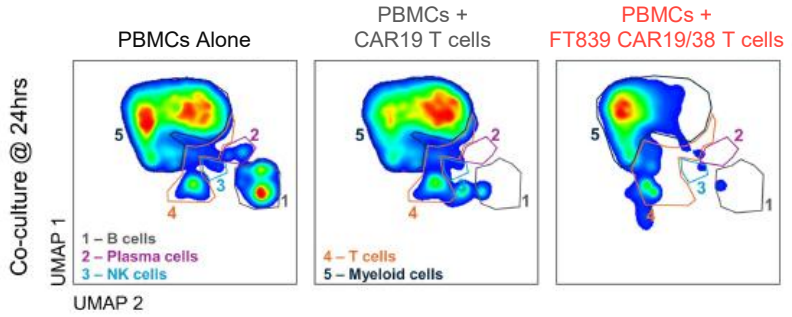
- CD19 is a common target in B cell malignancies and autoimmune diseases for CD19 directed CAR T-cell therapies.
- CD38, the target of daratumumab in multiple myeloma, is increasingly implicated in autoimmunity as a marker of pathogenic plasma cells and dysregulated T cells[1].



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

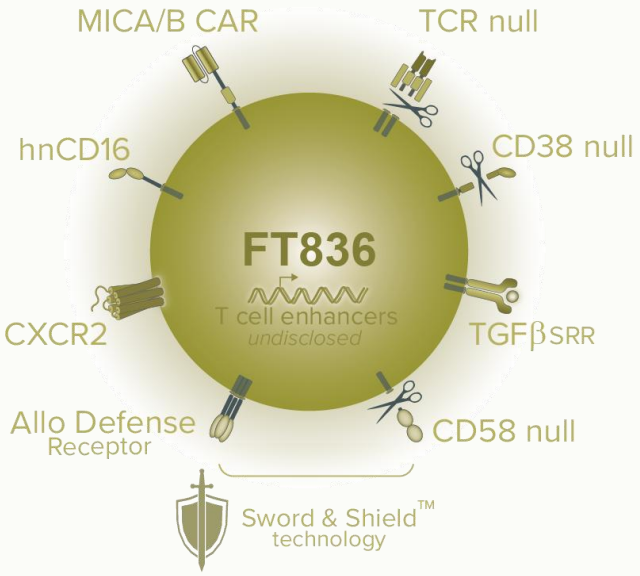
Phase 1 clinical evaluation ready in 2025

FT836 Product Candidate

Attribute Systems:

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

- FPI anticipated by YE 2025
- Phase 1 trial open enrollment:
  - Multiple tumor indications
  - Monotherapy & mAb combination
  - No lympho-conditioning
- Targeting FPI by YE 2025

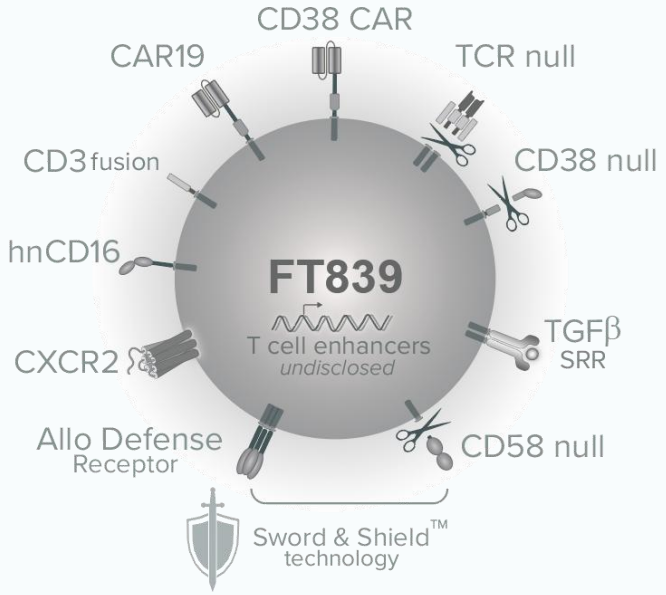


FT839 Product Candidate

Attribute Systems:

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

- Anticipated IND filing in 2026
- Planning for Phase 1 Trials:
  - Multiple autoimmune indication
  - Hematological malignancies
  - Monotherapy & mAb combination
  - No lympho-conditioning



FT522 Program

CD19 CAR NK Cell Product Candidate

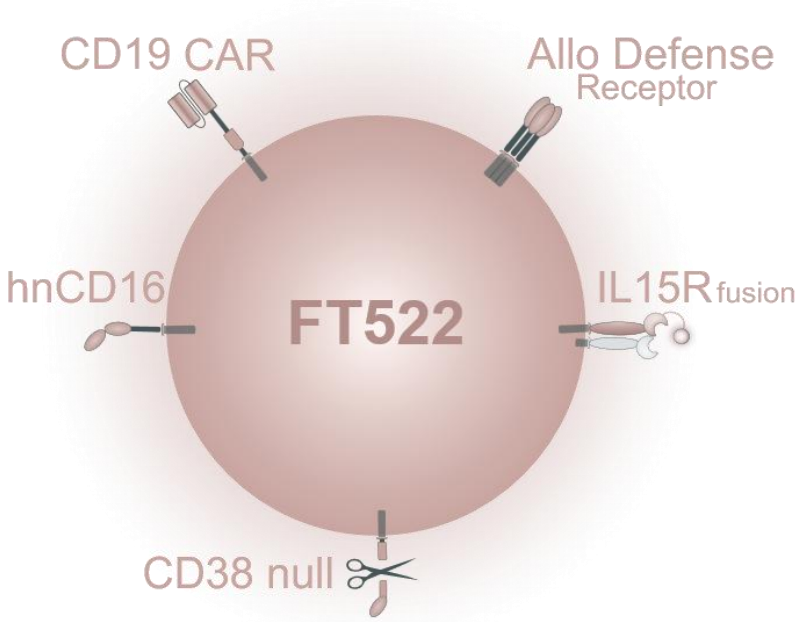
Fate THERAPEUTICS

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

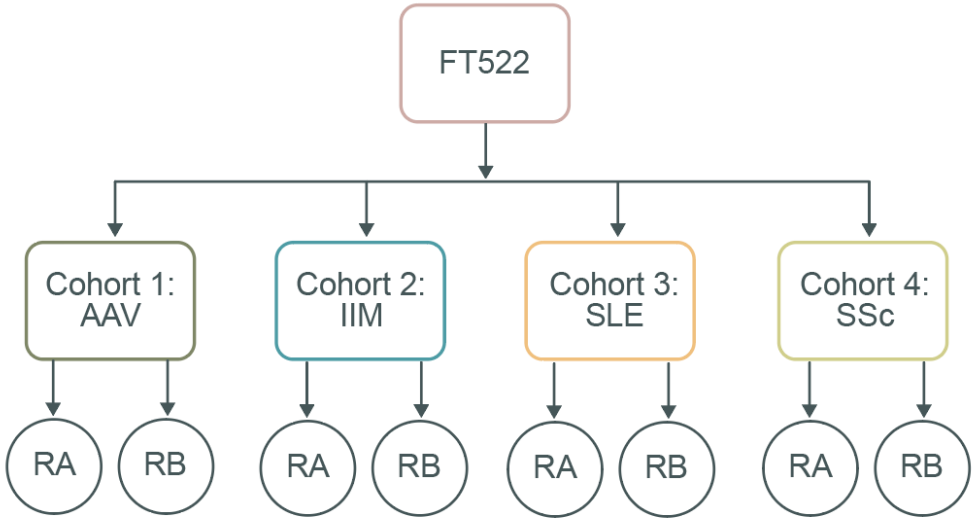
ADR armed NK Cells Uniquely Able to Proliferate and Persist

- **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 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 PATIENTS WITH AUTOIMMUNE DISEASES AND CANCER

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October 2025

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