Fate THERAPEUTICS

Better Cells For Better Therapies®

Programmed Cellular Immunotherapies

Transforming the Treatment of Cancer and Autoimmune Diseases with Off-the-shelf, Multiplexed-engineered, iPSC-derived Cellular Immunotherapy

May 2024

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 applicable law.

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Fate Therapeutics

Pioneering Off-the-Shelf iPSC-derived Cell Therapies

Induced Pluripotent Stem Cell Platform	Highly differentiated approach to cell therapy with unmatched engineering capability, manufacturing scale, and product quality and consistency
Eliminate Conditioning Chemotherapy	Proprietary ADR technology to redefine the cell therapy treatment paradigm: outpatient administration, add-on to standard-of-care therapies, reduced toxicities
Cell Therapies for Autoimmune Diseases	Designed to enable on-demand availability, patient convenience, broad therapeutic reach, and cost-effective utilization
Advanced T-cells for Solid Tumors	Constellation of novel synthetic controls to promote safety, deliver multi-pronged attack, and overcome tumor resistance for clinically meaningful outcomes
Next Generation T-cell Therapies	Highly sophisticated T-cell therapies with direct effector cell function, secretion of immune modulators, and synergy with host immune system

Supported by a strong balance sheet of ~$390 million (as of March 30, 2024)

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Changing the Game of Cell Therapy

Mass Produced, Multiplexed-engineered Cell Products for Off-the-shelf Patient Treatment

Platform Advantages:

- ✓ Single-cell, CRISPR-based, multiplexed engineering
- ✓ Engineered master cell banks selected for genomic stability, differentiation capacity, and product functionality
- ✓ Highly-scalable, cost-effective GMP manufacture with no further engineering
- ✓ Fast, efficient and modular innovation

iPSC-derived Cell Therapy Products:

- ➢ Well-characterized, uniform in composition, consistent production of drug product
- ➢ Low cost of goods and not susceptible to donor-to-donor variability
- ➢ Monoclonal antibody-like treatment: on-demand availability, repeat dosing, ease of combinability
- ➢ Patient convenience and reach: off-the-shelf, reduced toxicities, reduced hospitalization, community setting

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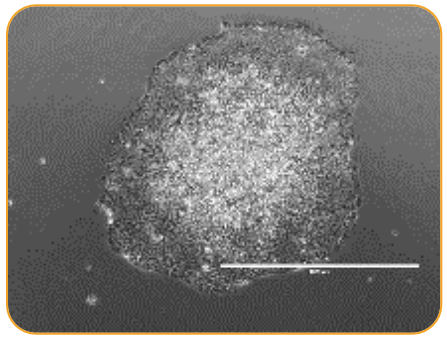
Disruptive iPSC Product Platform

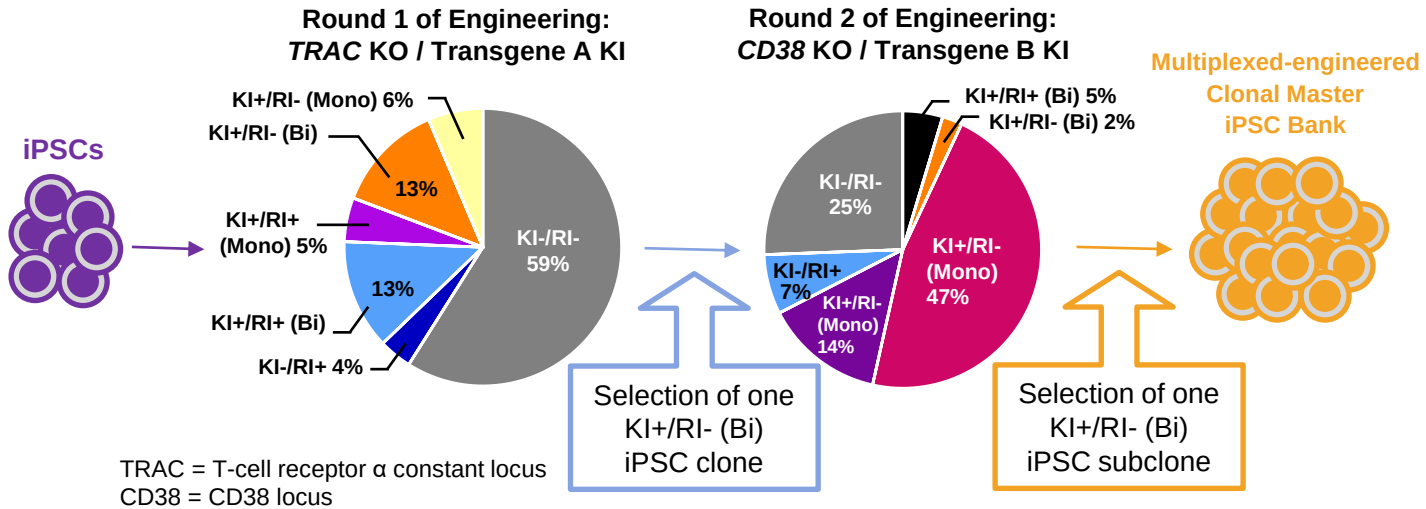
Creating Multiplexed-engineered Clonal Master iPSC Banks

Single iPSC Clone



- Multiplexed Engineering
- Extensive Characterization
- Uniform Composition
- Unlimited Clonal Expansion
- Clonal Master Cell Lines

Unprecedented Resolution in Clonal iPSC Characterization and Selection



TRAC = T-cell receptor α constant locus
CD38 = CD38 locus
KI = Knock-in Mono = mono-allelic
RI=Random integration Bi = bi-allelic
Percentages represents clones within the whole population

Pan et al. 2022 ASGCT Annual Conference

Fate Therapeutics' iPSC product platform is supported by an IP portfolio with 500+ issued patents and 500+ pending patent applications

First-in-class Product Pipeline

Multiplexed-engineered, iPSC-derived CAR NK Cell and CAR T-cell Product Candidates

Program	Indication	CAR Target(s)	Research	Preclinical	Phase 1	Partner
CAR T-cell Product Candidates						
FT819	Systemic Lupus Erythematosus	CD19				
FT825	Solid Tumors	HER2				ONO PHARMACEUTICAL CO.,LTD.
Undisclosed	Solid Tumors	Undisclosed				
FT836	Multiple Tumor Types	MICA/B				
NG iTs	Multiple Therapeutic Areas	Undisclosed				
CAR NK cell Product Candidates						
FT522	B-cell Lymphoma	CD19, 4-1BB				
FT522	Autoimmunity					
NG iNKs	Multiple Therapeutic Areas	Undisclosed				

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Oncology **Autoimmunity**

NG= Next-generation

ADR

Proprietary ADR Technology

Synthetic ADR Receptor Designed to Eliminate Requirement for Cy / Flu Conditioning

nature biotechnology ARTICLES https://doi.org/10.1038/s41587-020-0601-5

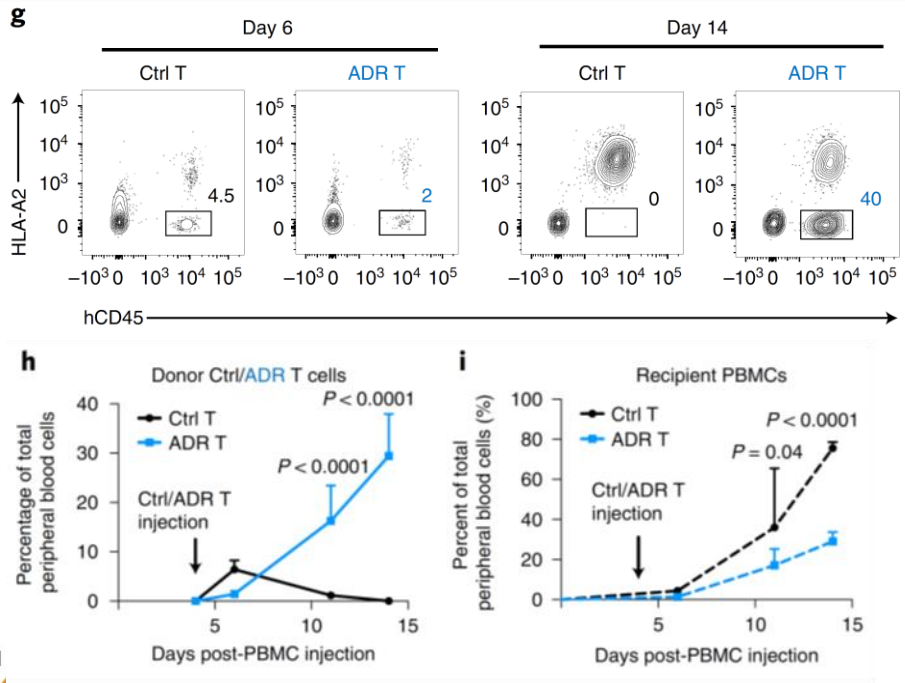
Engineered off-the-shelf therapeutic T cells resist host immune rejection

Feiyan Mo[1,2], Norihiro Watanabe[1], Mary K. McKenna[1], M. John Hicks[3], Madhuwanti Srinivasan[1], Diogo Gomes-Silva[1], Erden Atilla[1], Tyler Smith[1], Pinar Ataca Atilla[1], Royce Ma[1,4], David Quach[1], Helen E. Heslop[1,2], Malcolm K. Brenner[1,2] and Maksim Mamonkin[1,2,3,4]

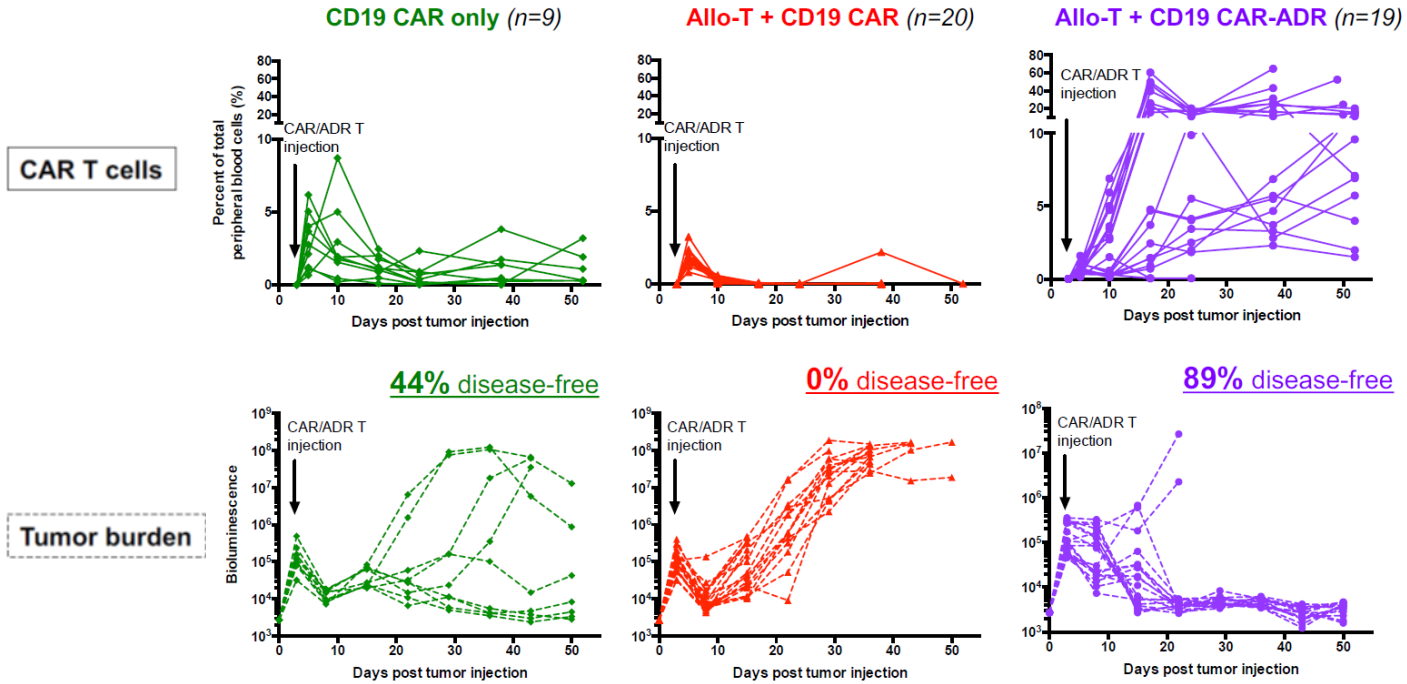
"The best defense is a good offense"

ADR-armed CAR T cells specifically target activated host immune cells expressing 4-1BB, avoid host immune cell rejection, and retain potent and durable anti-tumor activity against CD19+ cells.

ADR facilitates robust persistence in an allogenic setting



ADR+ CAR+ T cells avoid rejection and exhibit durable response in the presence of allogeneic T cells



Synthetic ADR Receptor

Unique Attributes in Comparison to Other Immune Evasion Strategies

Strategy	Combination with Intense Conditioning Chemotherapy	Knockout of HLA-I & -II	Knockout of HLA-I & -II + HLA-E & -G	Knockout of HLA-I & -II + CD47	**Fate's Approach** ADR Expression CD58 Knockout
Avoidance of rejection by host CD8 T cells	+	+	+	+	+++
Avoidance of rejection by host CD4 T cells	+	+	+	+	+++
Avoidance of rejection by host NK cells	+	-	+/-	+/-	+++
Avoidance of suppression by host Tregs	+	-	-	-	+++
Induction of proliferation signal	+	-	-	-	+++
Creation of endogenous space	+	-	-	-	+++
Avoidance of toxicity associated with immunosuppression	X	✓	✓	✓	✓

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FT819 Program

Off-the-shelf, CD19-targeted CAR T-cell Product Candidate

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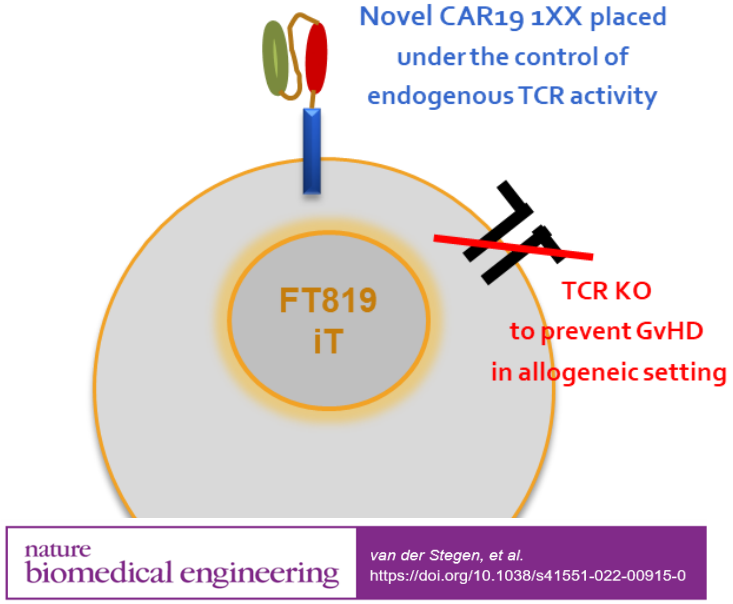
FT819 iPSC-derived, CD19-targeted CAR T-Cell Product Candidate

Product Design

Derived from Clonal Master Engineered iPSC Line Incorporating A Novel 1XX CAR Targeting CD19 and a TCR Knock-out



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

1XX CAR19: Novel chimeric antigen receptor consisting of CD28 costimulatory domain and modified CD3z signaling domain for optimal effector cell safety and activity

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TRAC targeted CAR: Chimeric antigen receptor integrated into the T Cell Receptor Alpha Constant region to be regulated by endogenous control of TCR expression for optimal CAR function

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TCR null: Bi-allelic disruption of TRAC at the clonal level for complete removal of TCR expression and the elimination for the possibility of GvHD in allogeneic setting

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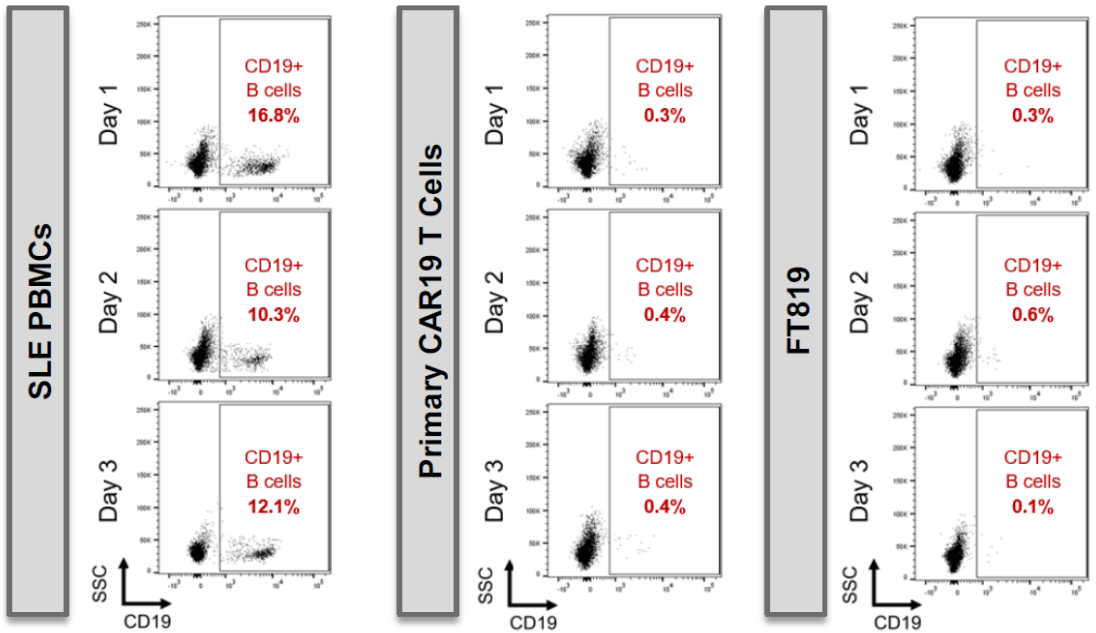
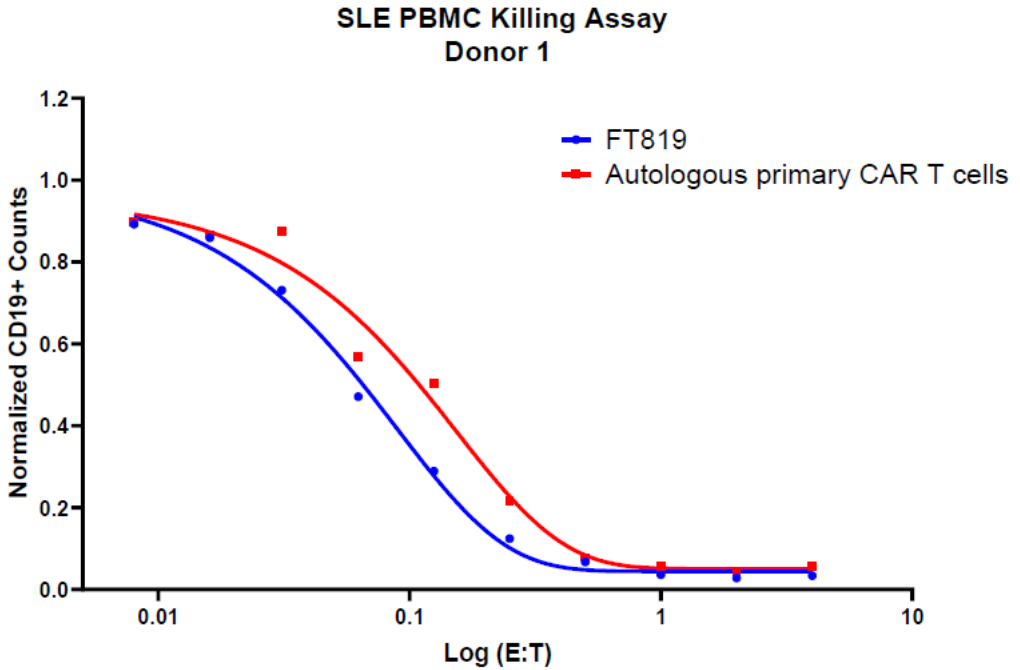
Ex Vivo B-cell Depletion

Preclinical Data in B Cell-mediated Autoimmune Disease

*FT819 demonstrates rapid, deep, and dose-dependent depletion of CD19+ B cells sourced from the peripheral blood of unmatched SLE donors in preclinical studies**





* Rapid and deep depletion of CD19+ B cells was observed in a 72-hour killing assay against peripheral blood mononuclear cells (PBMCs) sourced from a blood sample of an unmatched SLE donor (E:T ratio = 1:1). FT819 displays dose-dependent CD19+ B cell depletion against PBMCs sourced from a blood sample of an unmatched SLE donor in a manner comparable to autologous-manufactured CAR T cells.

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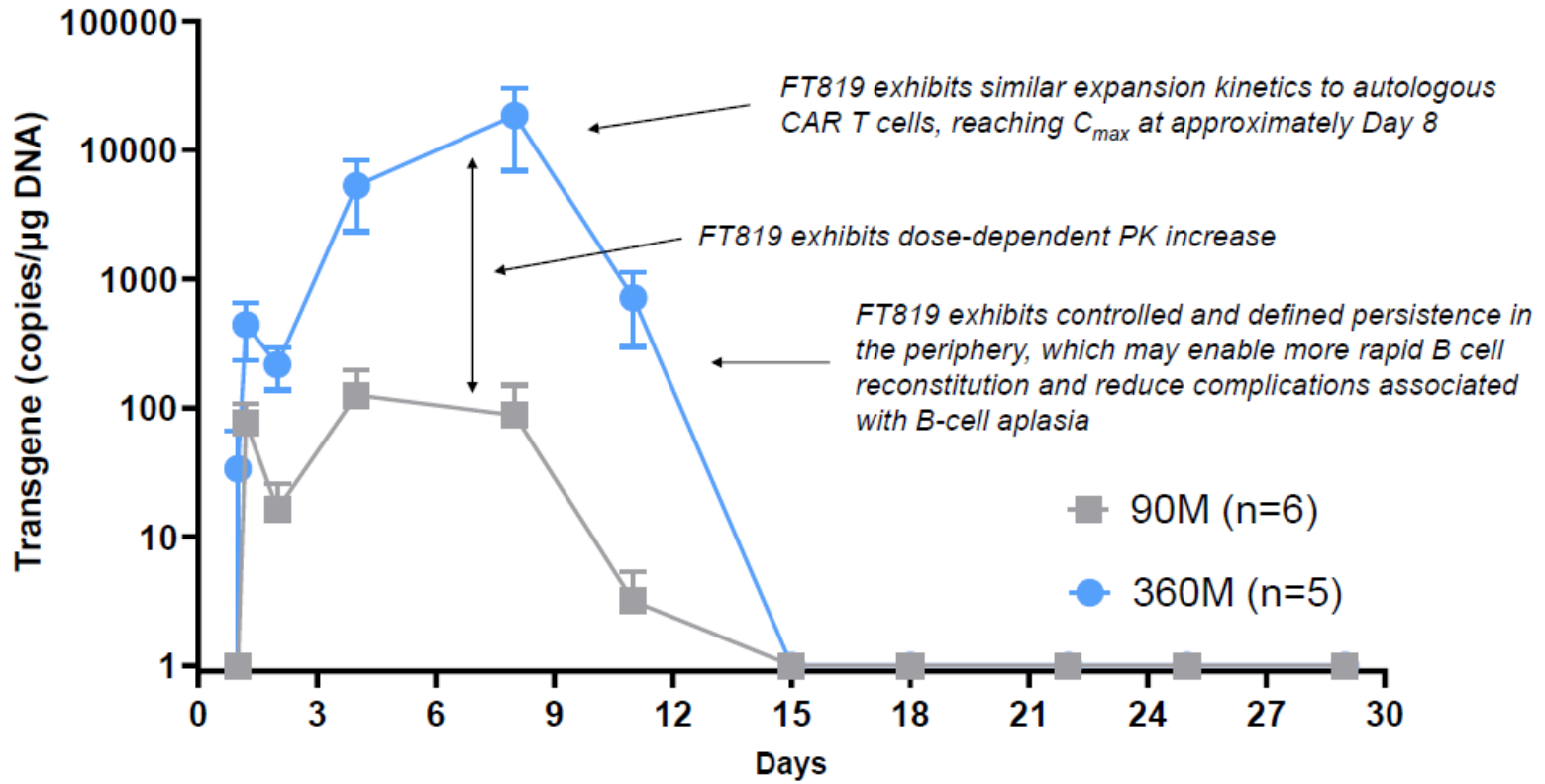
Pharmacokinetics

Phase 1 Translational Data

*FT819 demonstrates dose-dependent CAR T-cell expansion and exposure**



* FT819 PK (mean ± SEM) at 90 and 360 million cells in patients with r/r B-cell lymphoma (BCL) (n=11). *In vivo* CAR T-cell expansion and persistence were measured by a ddPCR assay according to the number of CAR transgene copies per microgram of genomic DNA in blood samples.

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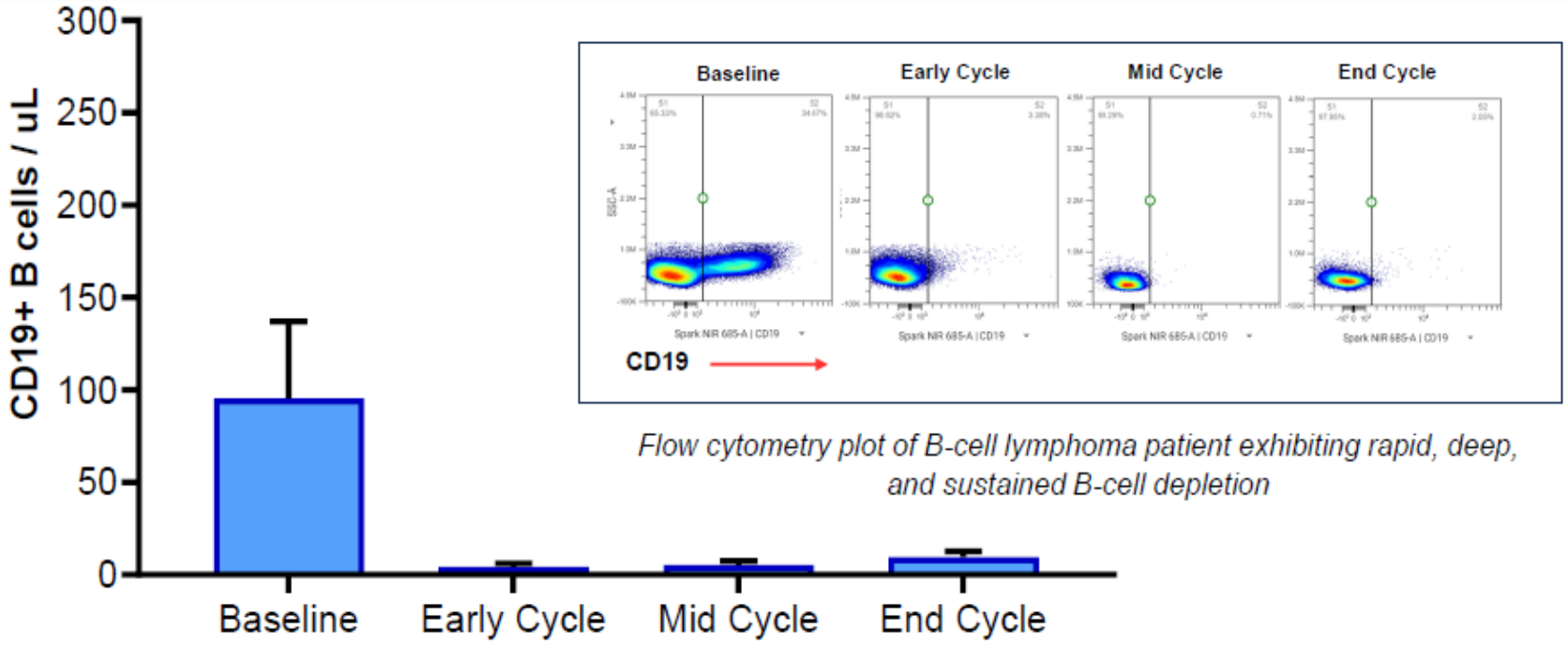
B-cell Depletion in the Periphery

Phase 1 Translational Data

*FT819 demonstrates rapid, deep, and sustained CD19+ B cell depletion during 30-day treatment cycle**



Flow cytometry plot of B-cell lymphoma patient exhibiting rapid, deep, and sustained B-cell depletion

* Rapid and deep B cell depletion, with sustained suppression of B cells, in the peripheral blood during FT819 treatment cycle in patients with r/r BCL (n=23; includes patients with baseline B cell counts in the range of normal (up to 1,000 cells/µL)). Baseline represents B cell counts in the peripheral blood prior to treatment with conditioning chemotherapy and single-dose FT819. Data represent the mean ± SEM.

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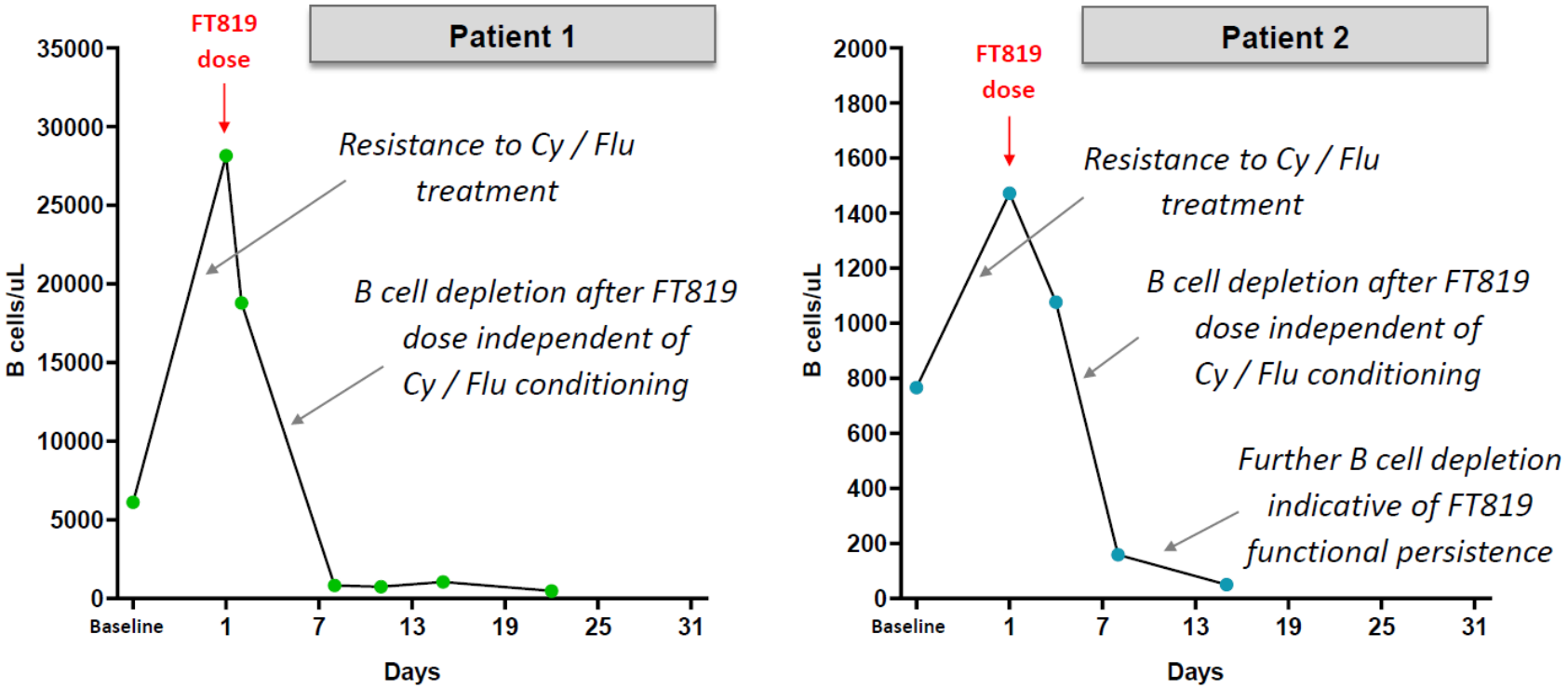
B-cell Depletion by FT819 Independent of Cy / Flu Conditioning

Phase 1 Translational Data

Cy / Flu conditioning did not result in B-cell depletion in the periphery
*Single dose of FT819 achieved rapid and deep depletion of supraphysiological B-cell burden**



* Case study of two patients with r/r chronic lymphocytic leukemia, where B cell depletion was resistant to Cy / Flu conditioning chemotherapy. Rapid and deep B cell depletion was observed following a single dose of FT819 at 180 million cells.

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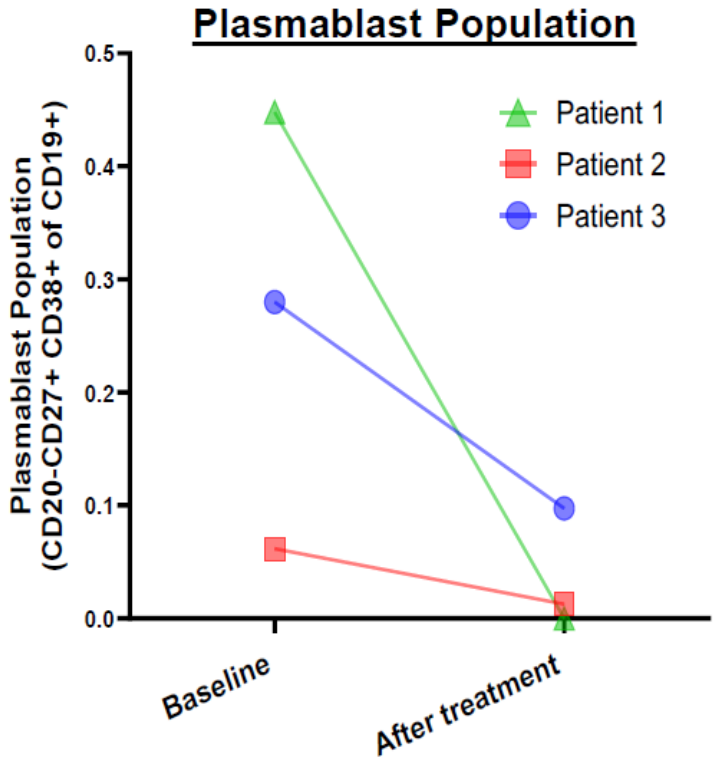
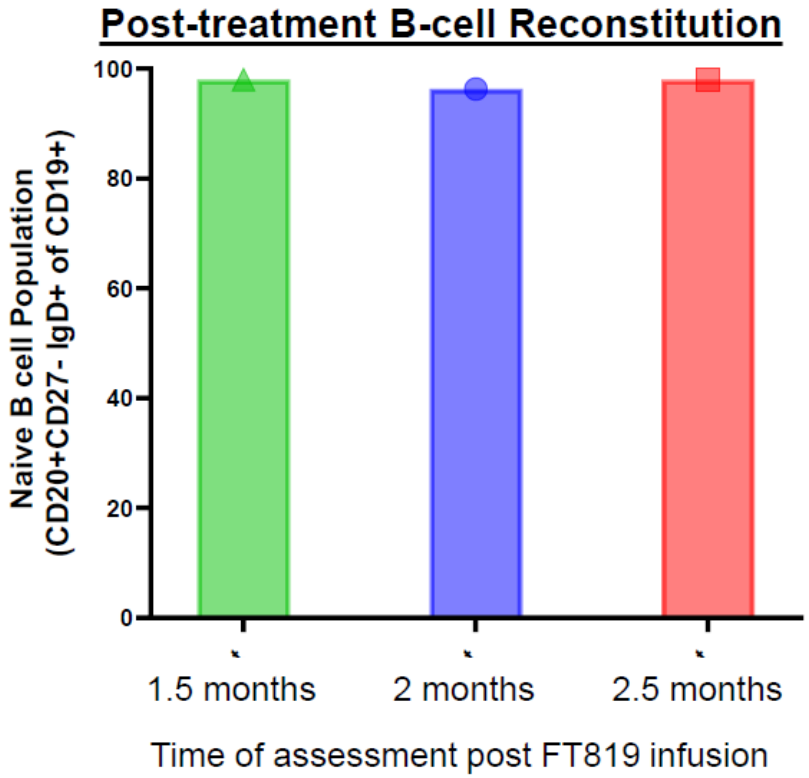
Reconstitution of Naïve B Cells after FT819 Treatment

Phase 1 Translational Data

FT819 demonstrated depletion of low-frequency CD19+ plasmablast population
*Post-treatment reconstitution comprised of naïve B cells**





* Case study of plasma cell depletion and B cell reconstitution in 3 patients with diverse B-cell repertoire at baseline, with reconstitution population comprised of naïve B cells in 6-10 weeks following single-dose FT819.

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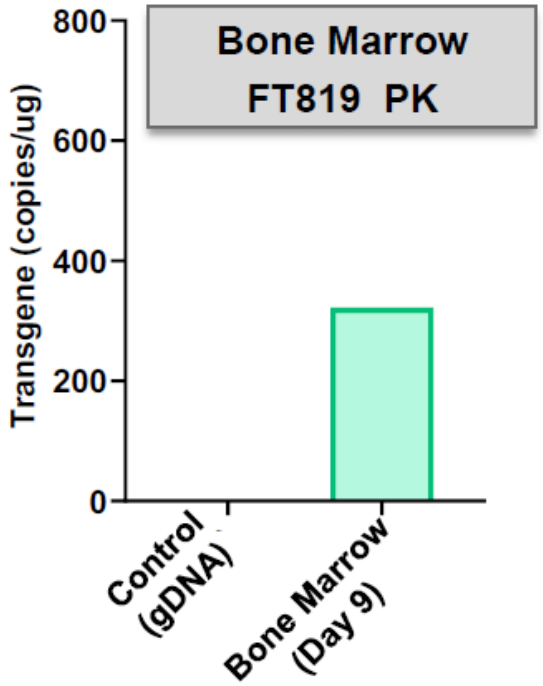
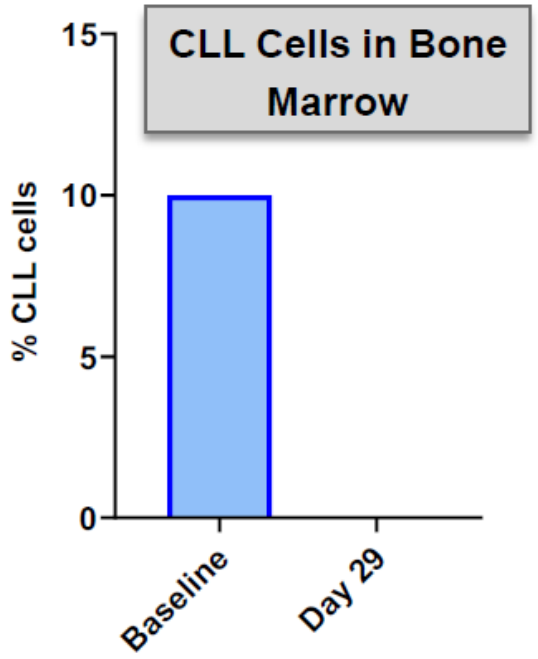
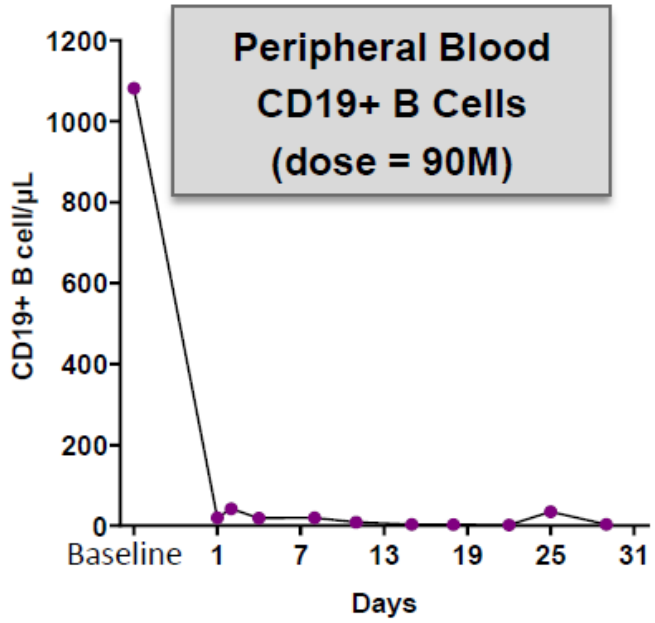
Tissue Homing, Infiltration & B Cell Clearance

Phase 1 Translational Data

*FT819 was detected in bone marrow at Day 9, with complete elimination of CD19+ cells in the bone marrow at the end of 30-day treatment cycle**







* Case study of chronic lymphocytic leukemia patient treated with conditioning chemotherapy followed by a single dose of FT819 at 90 million cells. FT819 was detected and persisted in the bone marrow at Day 9, with complete elimination of CD19+ cells in the bone marrow and peripheral blood at the end of treatment cycle.

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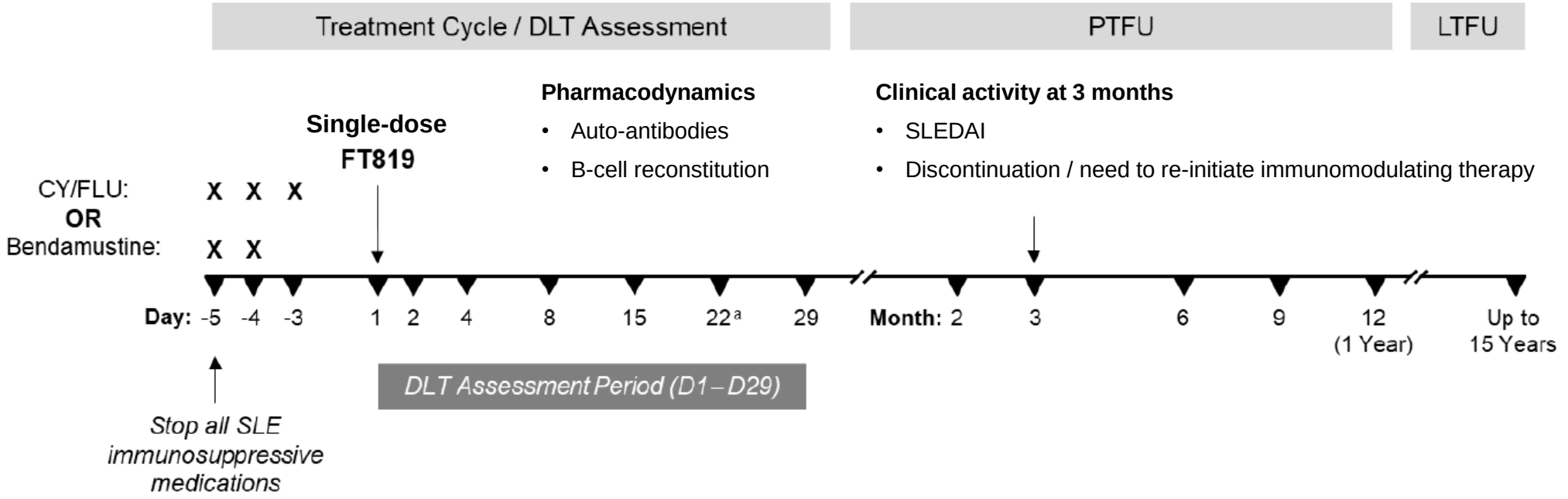
FT819 for B Cell-mediated Autoimmune Diseases

Phase 1 Study for System Lupus Erythematosus: Design

First SLE Patient Treated



Single-arm, 3+3 design initiated at DL1 = 360M cells; potential to escalate up to 3x dose and to expand each dose level up to 10 patients

Includes patients with active lupus nephritis or with active SLE without renal involvement, with disease severity of SLEDAI-2K ≥8

Patient conditioning includes 500 mg/m^2 Cy and 30 mg/m^2 Flu on Days -5, -4 and -3; or 90 mg/m^2 Bendamustine on Days -5 and -4

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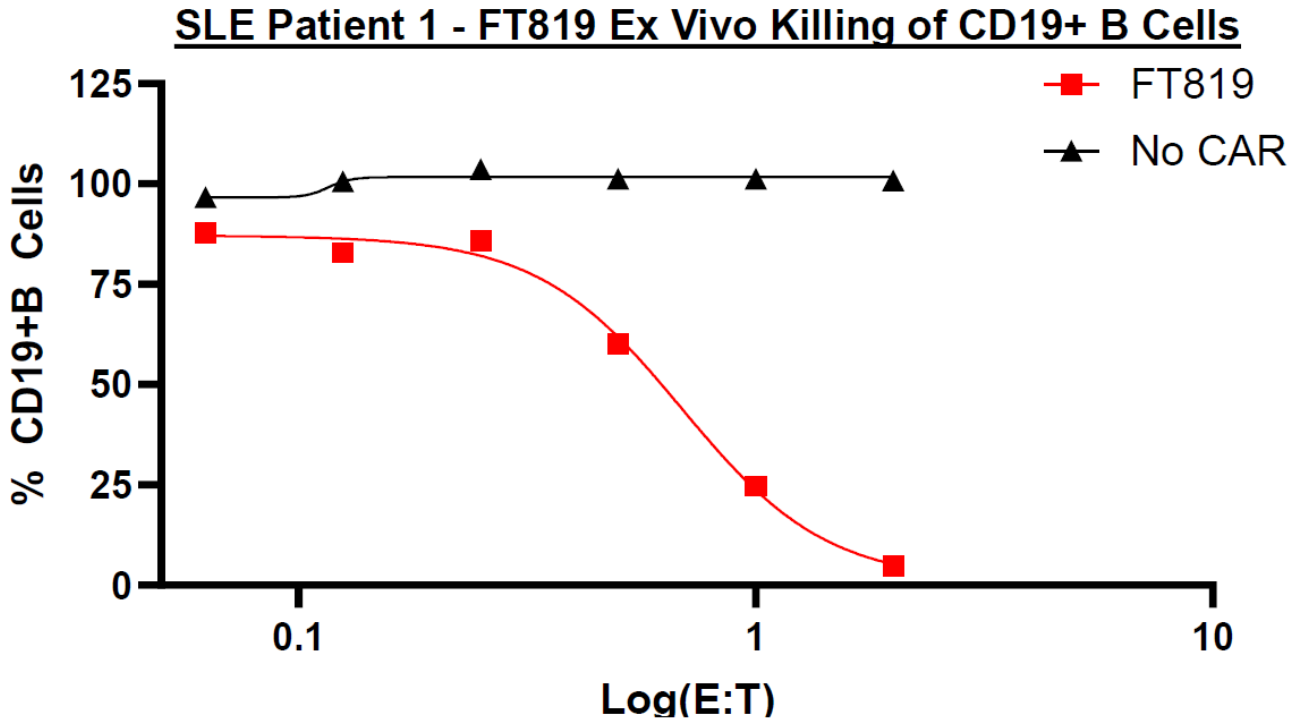
FT819 for B Cell-mediated Autoimmune Diseases

Ex Vivo Cytotoxicity Assay Using Pre-treatment Blood Sample from First SLE Patient

*Potent CD19+ B-cell depletion with FT819 using pre-treatment sample of first patient's peripheral blood in ex vivo killing assay**



* Using a pretreatment sample of the first patient's peripheral blood obtained prior to clinical administration of conditioning chemotherapy and FT819, a 24-hour cytotoxicity assay showed deep depletion of the patient's CD19+ B cells upon exposure to FT819 in a "first-of-kind" *ex vivo* translational assessment. The *ex vivo* translational assay did not include the use of cytokines to support the assay kinetics.

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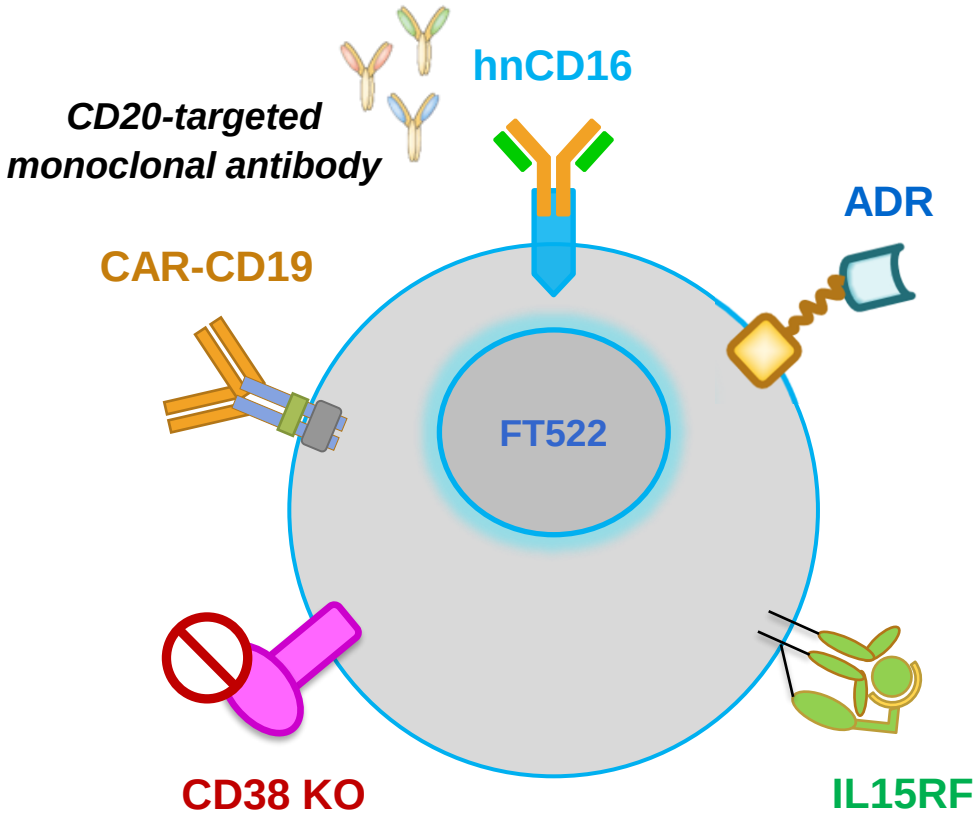
FT522 Program

Off-the-shelf, CD19-targeted CAR NK Cell Product Candidate

FT522 iPSC-derived, CD19-targeted CAR NK Cell Product Candidate

Product Design



hnCD16 *= high affinity, non-cleavable CD16 Fc receptor*
IL15-RF *= IL15 receptor fusion*
CD38-KO *= CD38 knock-out*
CAR-CD19 *= chimeric antigen receptor*
ADR *= allo-defense receptor targeting 4-1BB*

Multi-antigen targeting

- **CD19**: CAR construct targeting CD19
- **CD20**: proprietary hnCD16 receptor designed to augment antibody-dependent cellular cytotoxicity in combination with CD20-targeted mAb

Allo-defense technology

- **ADR:** novel synthetic receptor designed to: 1) selectively deplete host NK and T cells to mitigate rejection; and 2) potentiate cell activation through CD3-zeta signaling

Functional persistence

- **IL15RF:** promotes cell survival and proliferation to extend functional persistence
- **CD38KO:** enhances metabolic fitness

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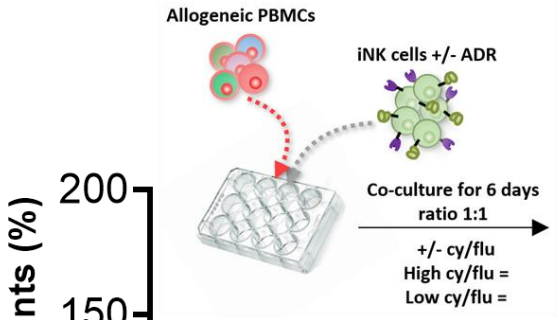
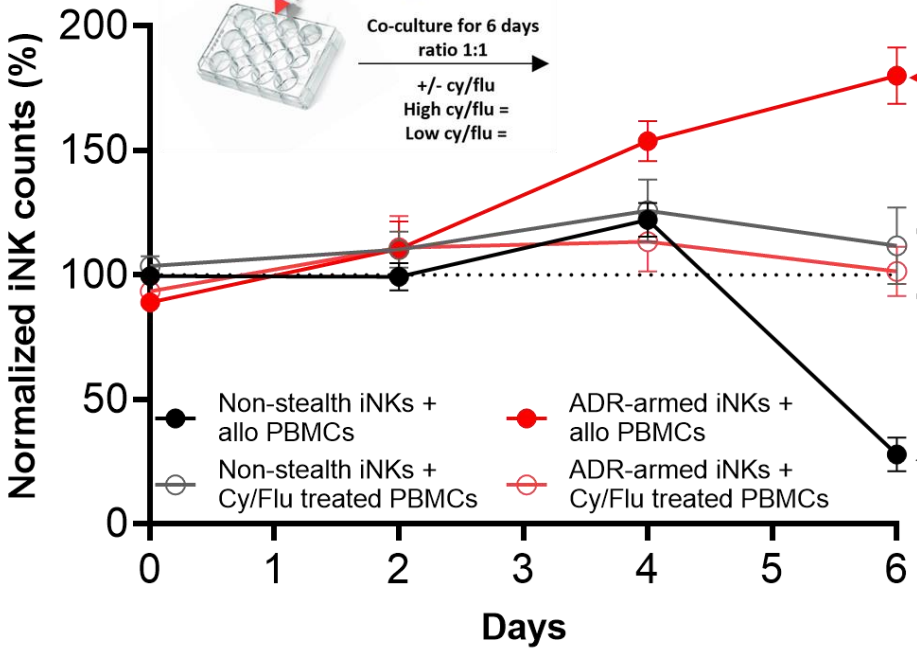
ADR-armed NK Cells Uniquely Proliferate and Persist

Preclinical Data in Ex Vivo Allogeneic System



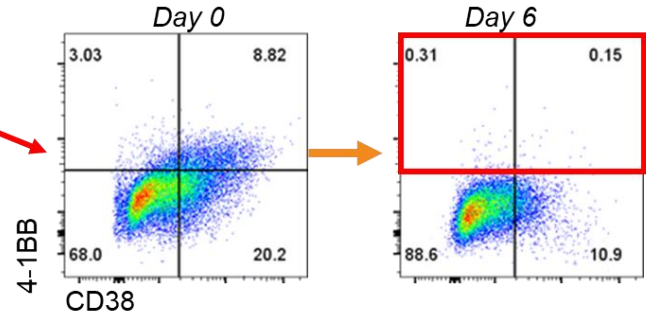


ADR-armed iNK cells proliferate, eliminate alloreactive immune cells, and persist in the presence of host PBMCs

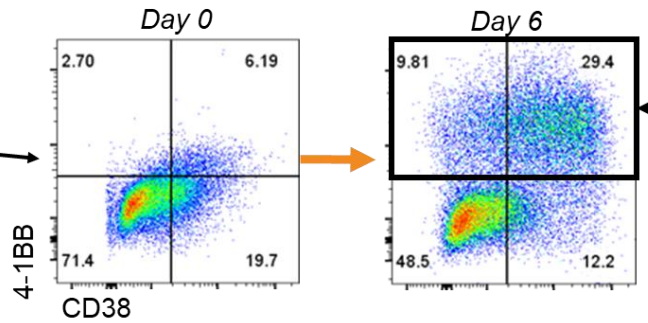
Both ADR-armed and ADR-null iNK cells maintain persistence in the presence of host PBMCs pre-treated with conditioning chemotherapy

ADR-null iNK cells are eliminated in the presence of host PBMCs

ADR-armed iNK cells (FT522) + host PBMCs



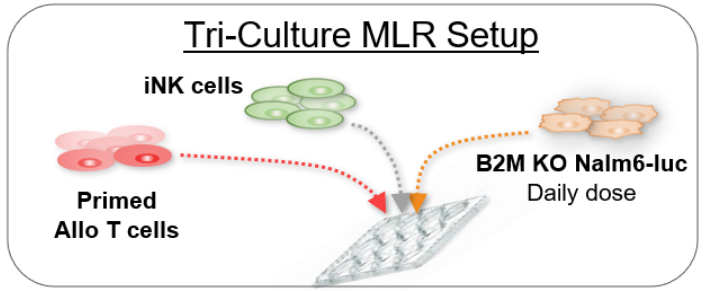
ADR-null iNK cells + host PBMCs



ADR-armed iNK cells eliminate alloreactive immune cells expressing either 4-1BB or CD38, enabling iNK cell potentiation and survival in an allogeneic system

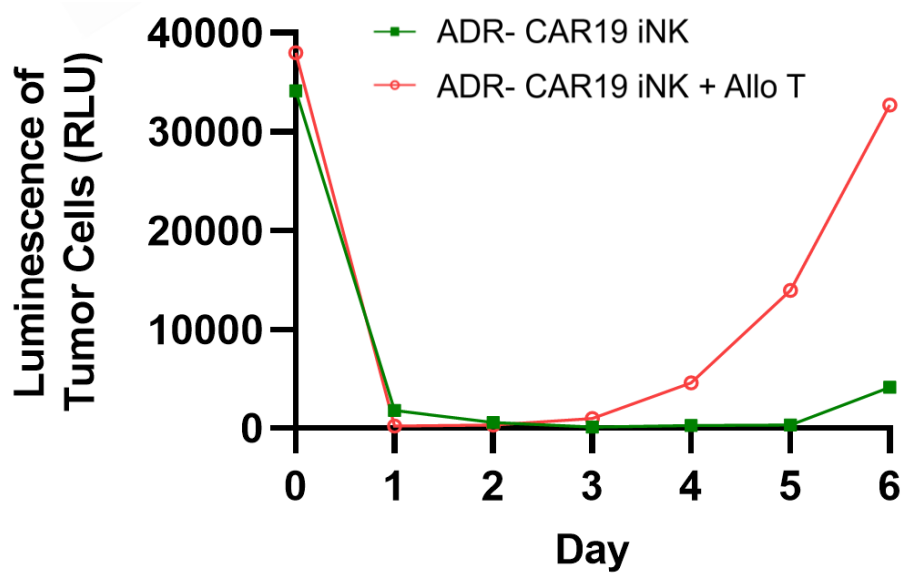
ADR-armed NK Cells Demonstrate Potent Anti-tumor Activity

Preclinical Data in Ex Vivo Alloreactive T-cell System

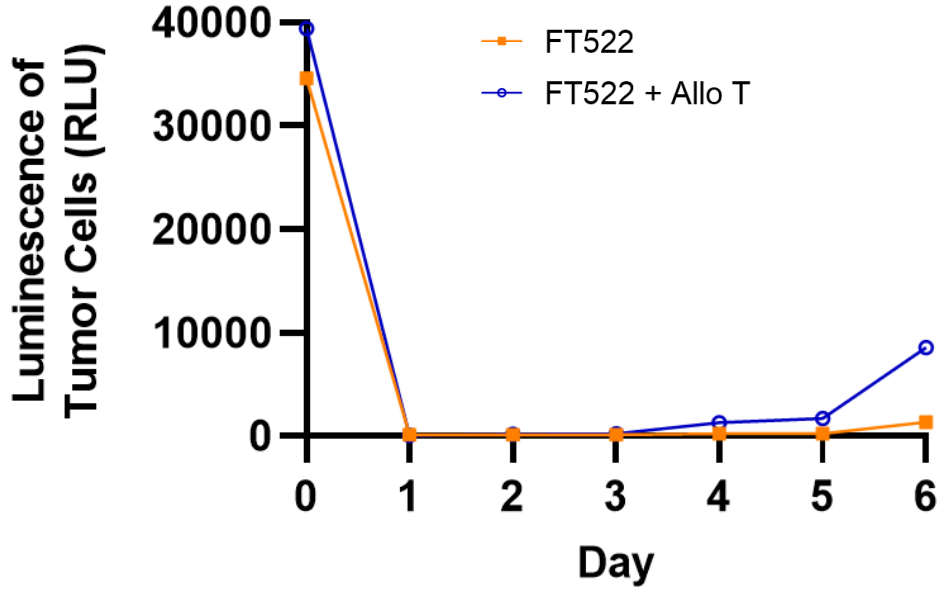


In a tumor cell restimulation assay, FT522 maintains potent CAR activity alone and in the presence of allogeneic T cells

ADR-null CAR19 iNK ± Allo T Cells



ADR-armed CAR19 iNK ± Allo T Cells



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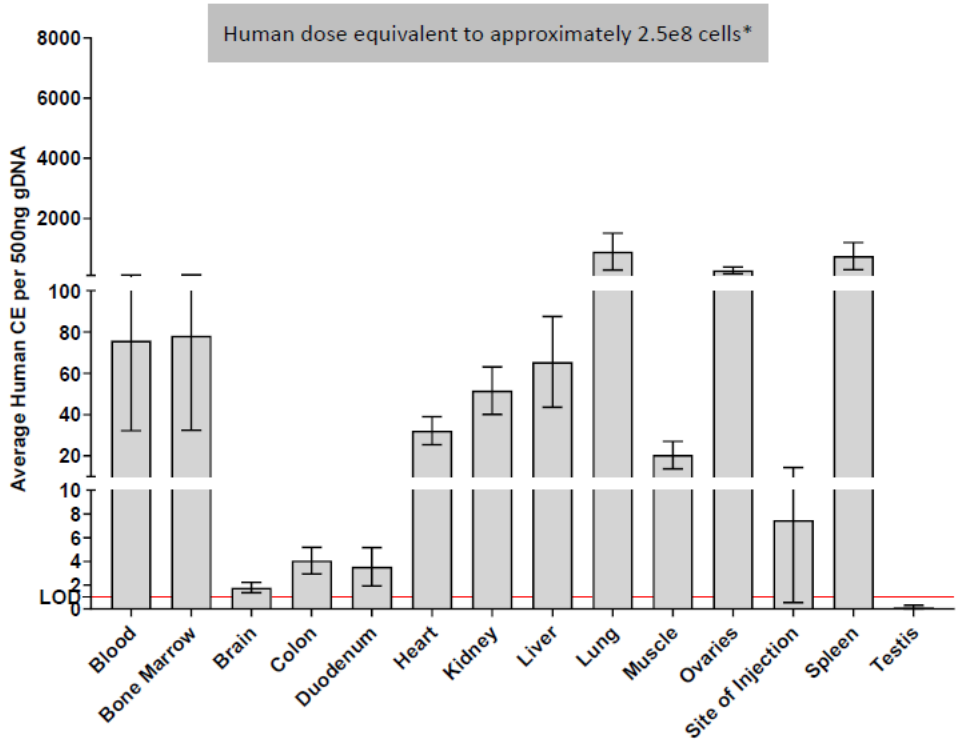
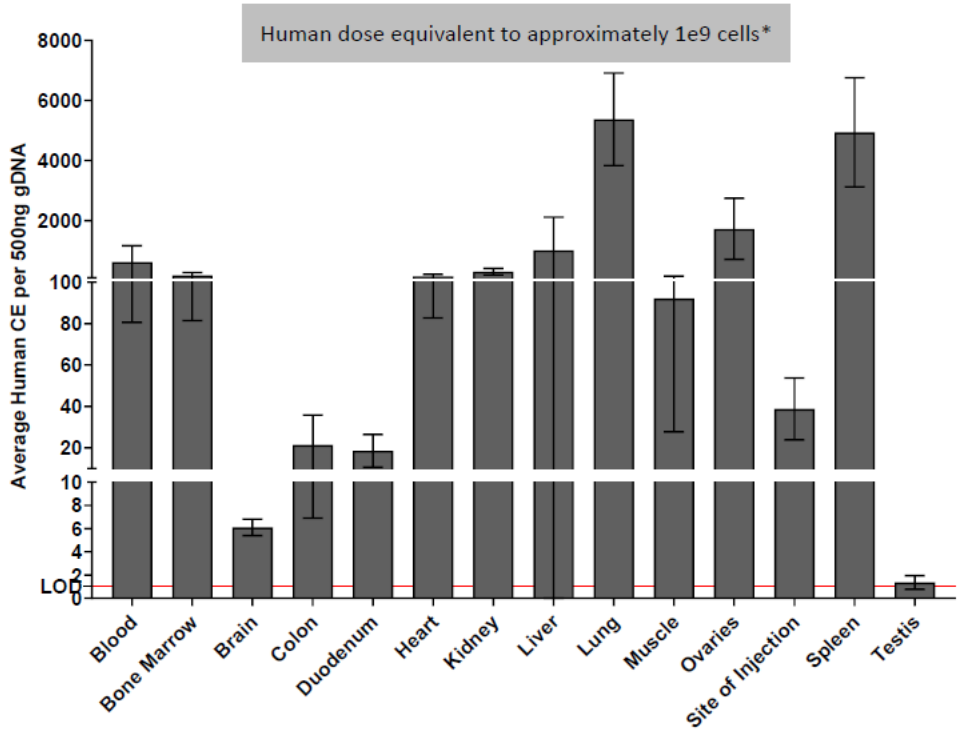
FT522 In Vivo Trafficking and Tissue Residency

Preclinical Data Show Broad Distribution across Primary, Secondary and Tertiary Tissues

*Dose-dependent trafficking, infiltration, & residency in primary, secondary & tertiary tissues without cytokine support at human dose equivalency levels of 250 million & 1 billion cells per dose**





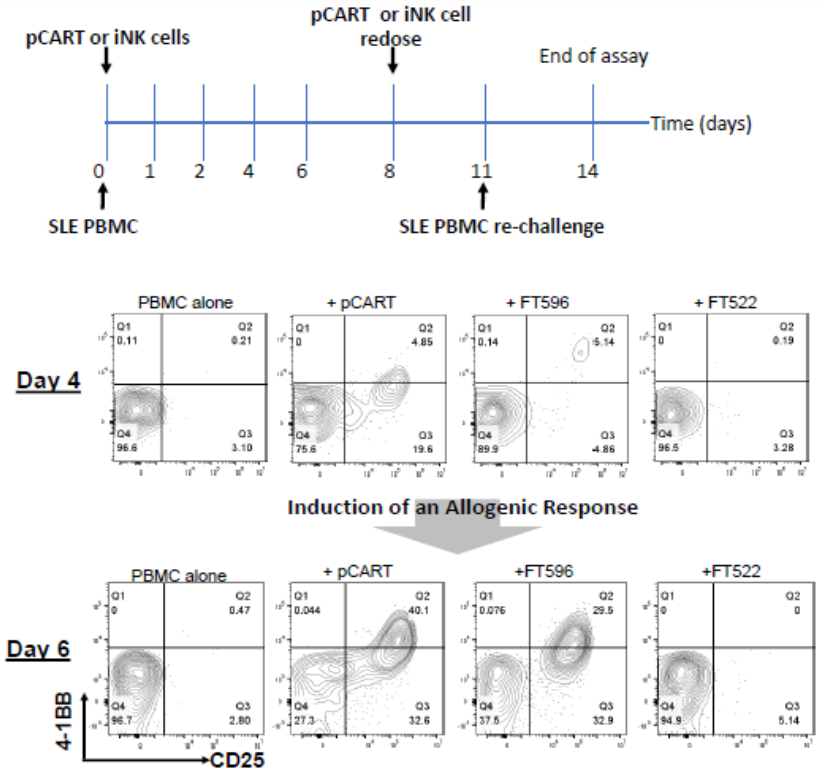
* NSG mice dosed with a human equivalency of 3 x 250 million FT522 cells and 3 x 1 billion FT522 cells over 15 days and analyzed for biodistribution the day after the last dose. No cytokine support or target cells expressing CD19 antigen were provided in this study. Human dose equivalency was calculated based on allometric conversion between a 20g mouse and 65Kg human.

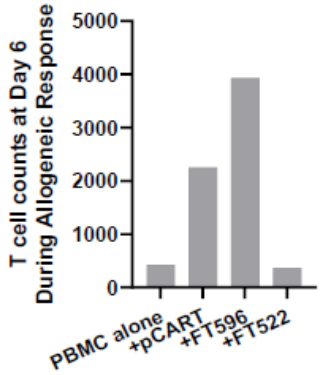
Witty et al. 2024 ASGCT Annual Conference

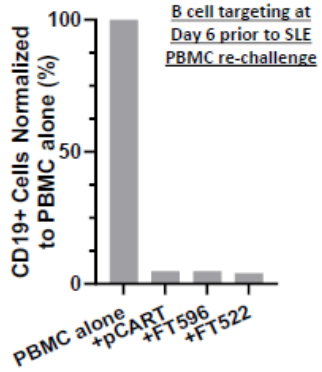
FT522 Unique Functional Profile in Unmatched Donor SLE System

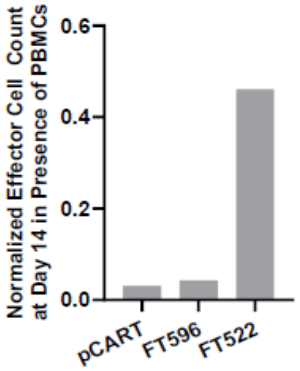
Preclinical Data Show B-cell Depletion, Alloreactive T Cell Elimination, and Functional Persistence

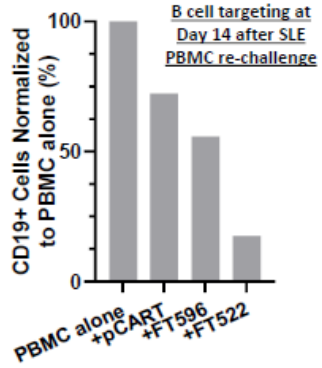
*In vitro activity in unmatched donor SLE PBMCs suggest unique functional profile in the presence of an unmatched host immune system**











Witty et al.
2024 ASGCT Annual Conference

* *In vitro* allogenic re-challenge assay. Effector cell population is co-cultured with unmatched SLE donor PBMCs for 8 days, followed by re-dosing of effector cell population and re-challenge with unmatched SLE donor PBMCs in co-culture for a total of 14 days. Flow cytometry of unmatched SLE donor CD3+ T cells on Day 6 demonstrates T-cell activation and expansion with primary CAR-T and FT596 cells, but not to FT522 cells. Upon re-challenge, primary CAR-T and FT596 cells are depleted, whereas FT522 cells continue to persist and kill CD19+ B cells.

FT522 Phase 1 Study in Relapsed / Refractory B-cell Lymphoma

Two-arm Study Designed to Assess Safety & Activity with and without Cy / Flu Conditioning

Potential to demonstrate early clinical POC of FT522
without administration of conditioning chemotherapy to patients

Conditioning Arm

- 500 mg/m^2 Cy x 3 days + 30 mg/m^2 Flu x 3 days; or
  90 mg/m^2 Bendamustine x 2 days
- Single-dose CD20-targeted mAb on Day -4
- FT522 x 3 doses on Days 1, 4 and 8
- Day 29 DLT / Response assessment

DL1 = 3x300M cells / dose
(up to 2 cycles)
n=3

Dose escalation at up to 3x prior dose level

No Conditioning Arm

- No conditioning
- Single-dose CD20-targeted mAb on Day -4
- FT522 x 3 doses on Days 1, 4 and 8
- Day 29 DLT / Response assessment

DL1 = 3x300M cells / dose
(up to 2 cycles)
n=3

Dose escalation at up to 3x prior dose level

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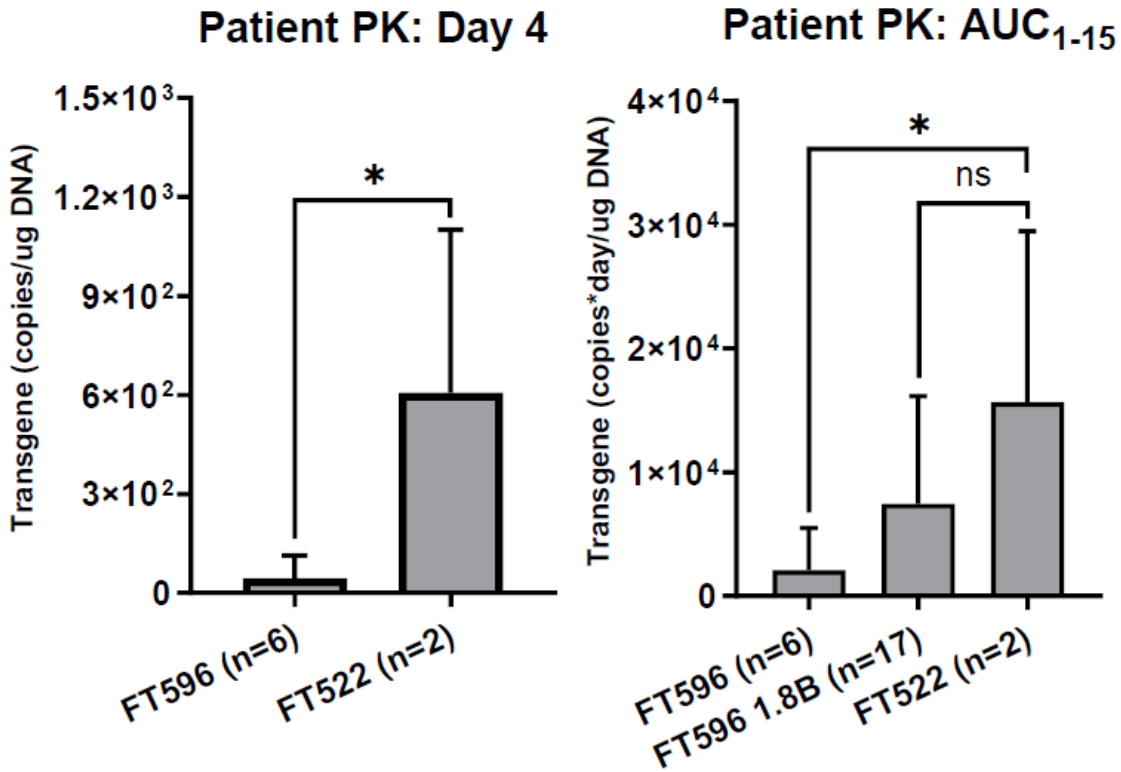
FT522 Phase 1 Study in Relapsed / Refractory B-cell Lymphoma

Initial Clinical Data Indicate Enhanced Product PK in the Periphery

*Clinical data from first two patients in Conditioning Arm show improved PK and AUC with FT522 compared to prior-generation CAR NK cell program**



*** Phase 1 study in B-cell lymphoma (BCL).** FT522 Day 1 dose demonstrates higher PK at Day 4 in comparison to FT596 Day 1 dose (NCT04245722; Phase 1 study in BCL) at an equivalent dose of 300 million cells. Three doses of FT522 at 300 million cells / dose demonstrate higher overall AUC during treatment cycle as compared to three doses of FT596 at 1.8 billion cells / dose.

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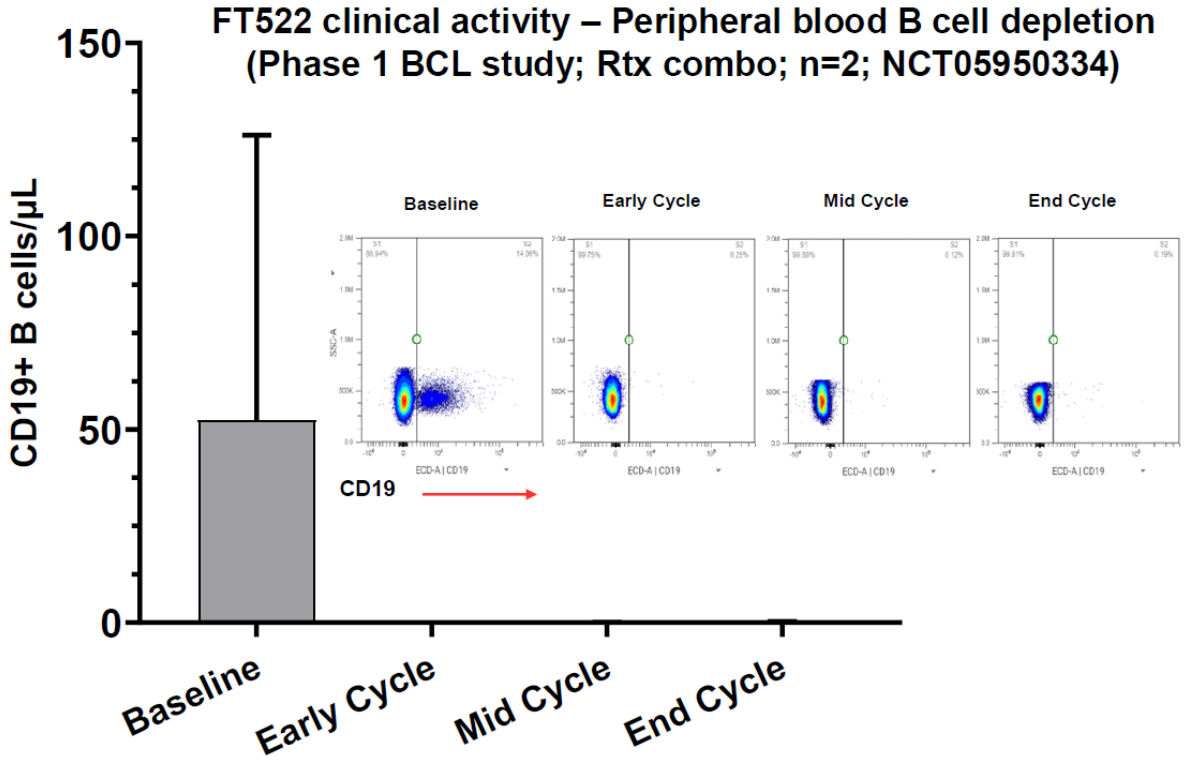
FT522 Phase 1 Study in Relapsed / Refractory B-cell Lymphoma

Initial Clinical Data Show B-cell Depletion in the Periphery

*Clinical data from first two patients in Conditioning Arm show rapid, deep, and sustained CD19+ B cell depletion in the periphery**



* Regimen A (FT522 combination with rituximab; n=2; NCT05950334). Baseline represents B-cell counts in the peripheral blood prior to treatment with standard conditioning chemotherapy. 30-day treatment cycle broken into stages

Witty et al. 2024 ASGCT Annual Conference

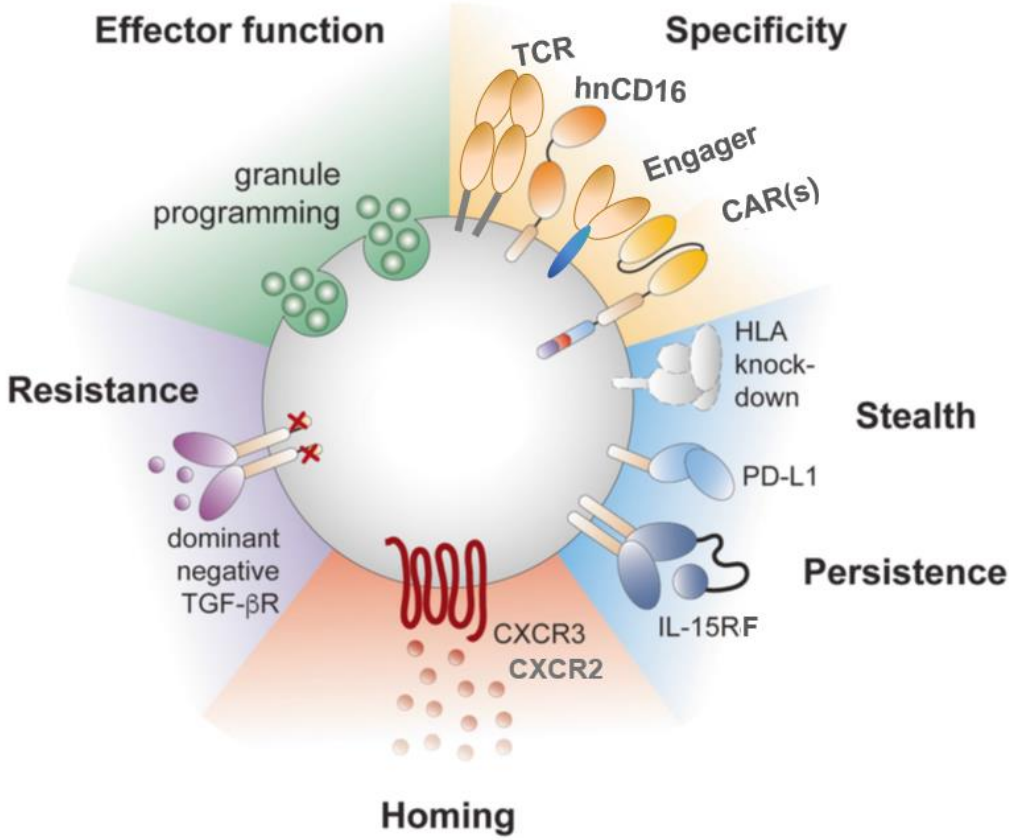
FT825 Program

Off-the-shelf, HER2-targeted CAR T-cell Product Candidate

Off-the-shelf Cell-based Cancer Immunotherapies for Solid Tumors

Developing Multiplexed-engineered, iPSC-derived Synthetic Effector Cells for Solid Tumors

- Next-generation cancer immunotherapies must address numerous challenges that limit the effectiveness of today's agents in treating solid tumors:
  - Depleted / dysfunctional immune cells
  - Immuno-suppressive microenvironment
  - Tumor heterogeneity and escape
- Cell-based cancer immunotherapies have the unique potential to bring rejuvenated immune cells to the fight against cancer.
  - May address deficiencies in patients' host immune system, mount multi-pronged attack, and synergize with complementary agents
- Fate Therapeutics has built a robust pipeline of off-the-shelf, multiplexed-engineered cell therapies for solid tumors.
  - Incorporate synthetic features specifically designed to exploit novel MOAs, synergize with approved agents, and overcome mechanisms of resistance



Modified after Saetersmoen et al. Seminars in Immunopathology 2019

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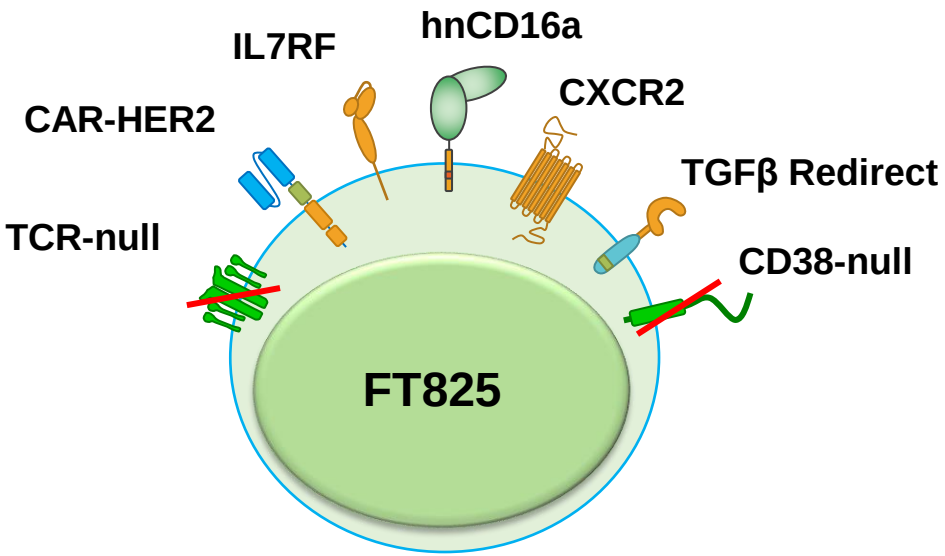
FT825 iPSC-derived, HER2-targeted CAR T-Cell Product Candidate

Incorporates Seven Novel Synthetic Controls of Cell Function



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

First patient treated in 2Q24

CAR-HER2: Novel 1XX CAR targeting HER2; controlled by TRAC locus

TCR KO: Complete loss of TCR surface expression to eliminate potential of GvHD in allogeneic setting

hnCD16: High-affinity 158V, non-cleavable CD16 Fc receptor to maximize ADCC

IL-7RF: Interleukin-7 receptor fusion to support stemness properties and increase persistence

CD38 KO: Resistance to anti-CD38 mAb-mediated fratricide opens new opportunities in conditioning; enhanced effector cell metabolic fitness and persistence.

TGFβ Redirect: Redirect receptor with the unique ability to overcome TME suppression mediated by TGFb signaling

CXCR2: Expression of synthetic chemokine receptor to promote chemotaxis to sites of inflammation and tumor

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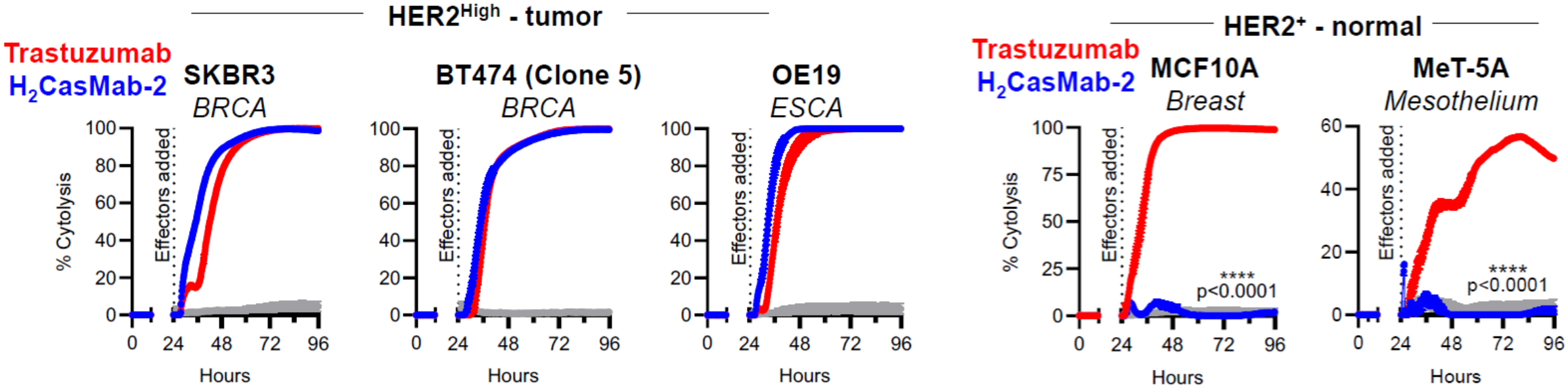
FT825 iPSC-derived, HER2-targeted CAR T-Cell Product Candidate

Novel Cancer-specific Antigen Binder Preferentially Targets HER2 on Tumor Cells

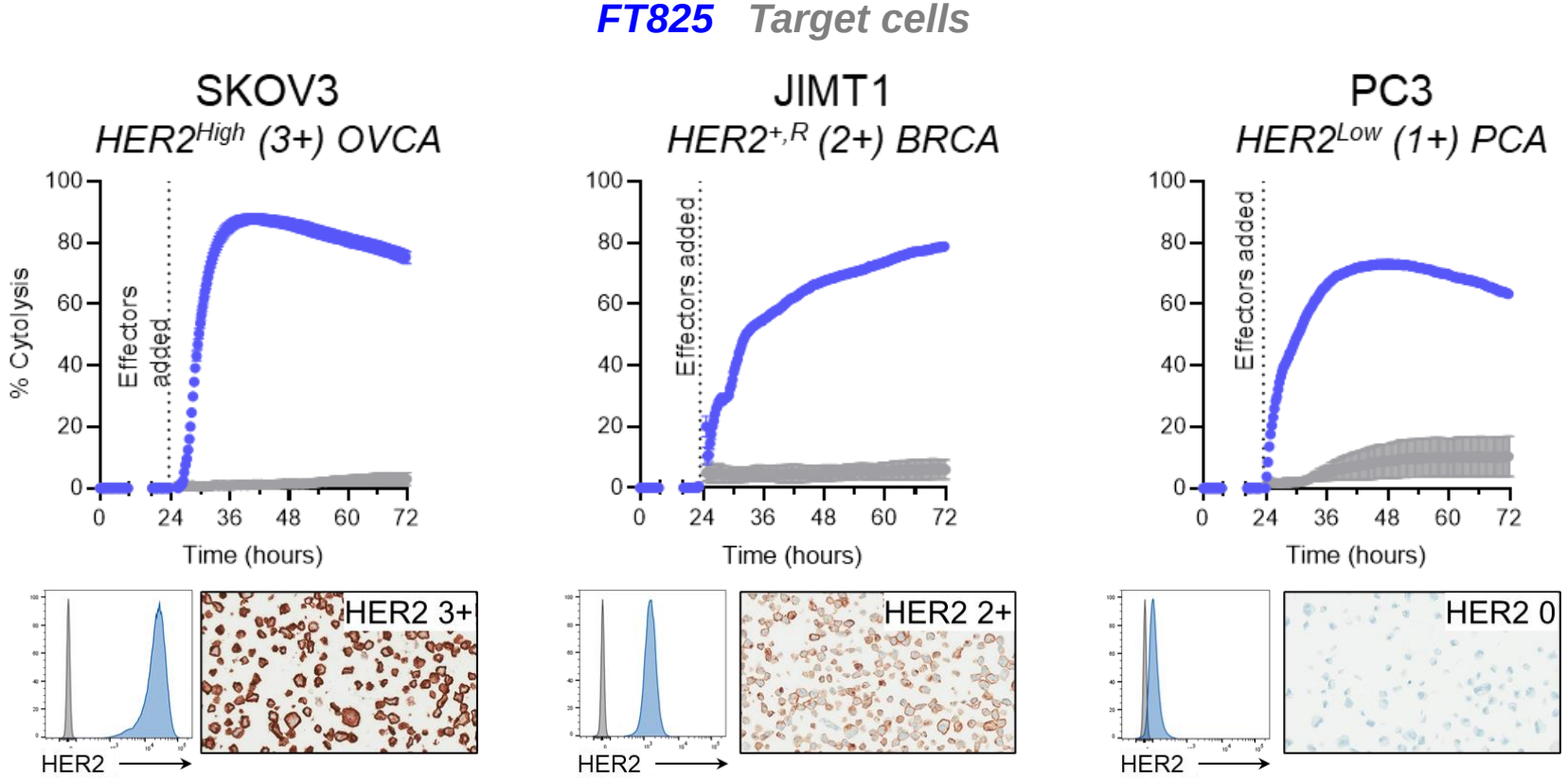
Novel cancer-specific antigen binder preferentially targets HER2 on tumor cells with limited recognition of HER2 on healthy cells



FT825 iPSC-derived, HER2-targeted CAR T-Cell Product Candidate

Novel Cancer-specific Antigen Binder Exhibits Activity Across HER2 Expression Density

Robust antigen-dependent targeting across HER2 expression levels, including HER2-low tumor cell lines



ONO Pharmaceutical Collaboration

FT825 Plus Second Undisclosed Antigen Target for Solid Tumors

Established September 2018 and expanded June 2022

Innovation for Solid Tumors

- Multiplexed-engineered, CAR-targeted product candidates
- Novel antigen binding domains contributed by Ono
- Multiple mechanisms of action to address solid tumor biology

Strategic Collaboration

- FATE leads preclinical development to pre-IND milestone
- Ono has options to WW development & commercialization
- FATE opt-in rights to 50-50 co-dev / co-comm in US and Europe

Financial Terms

- $10m upfront
- 50-50 cost sharing to pre-IND milestone
- Up to $840m in milestones, mid-single to low-double-digit royalties

Progress

- ✓ $12.5m Ono option exercise for FT825 (2022)
- ✓ Fate opt-in to US & EU co-co for FT825 (2022)
- ✓ FT825 IND clearance
- ✓ FT825 P1 initiation
- ✓ $5m milestone for FT825 first patient treated (2Q24)
- ○ Pre-IND opt-in decision for 2nd CAR-T product candidate
- ○ IND filing for undisclosed 2nd solid tumor CAR-T product candidate

T-cell Platform Innovation

Developing a Portfolio of Engineered Features

Proprietary Functional Elements for Enhanced Cell Functionality & Synergizing with IO mAbs

Direct Multi-Antigen Targeting

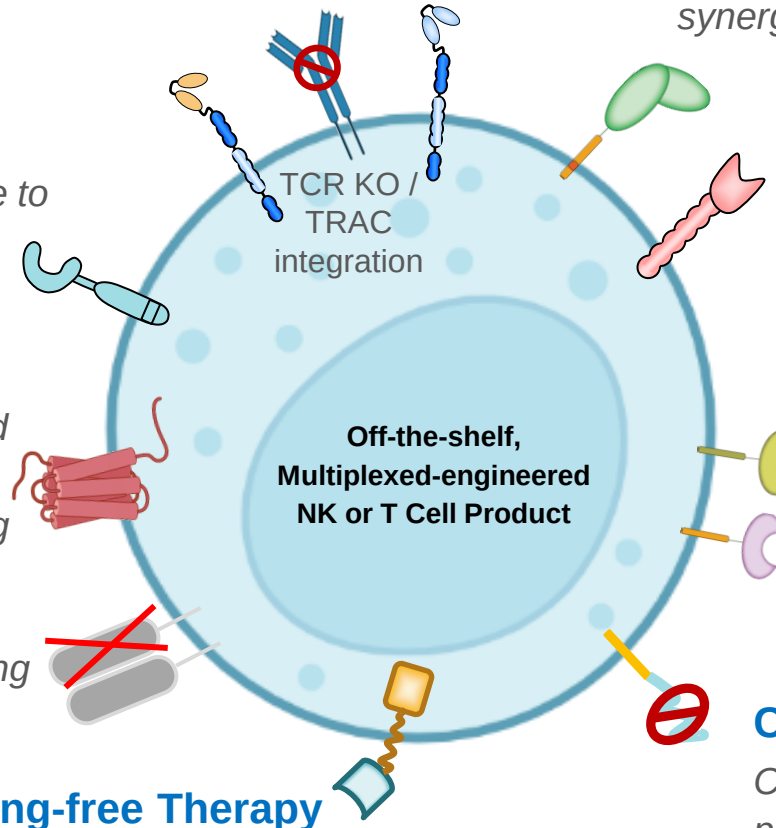
CARs directed to CD19, BCMA, MICA/B, B7H3, others

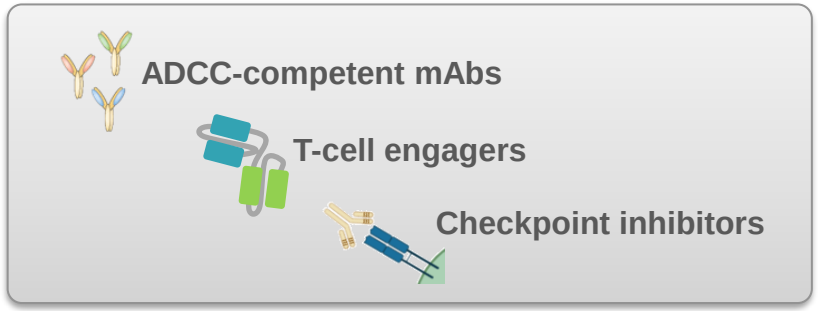
Combinations with mAbs / Engagers

hnCD16 and synthetic CD3 receptors to uniquely synergize with mAbs and NK cell / T-cell engagers

Immunosuppressive Resistance

Synthetic TGFB redirector to promote activation in response to immuno-suppressive TME





Cell Homing

Synthetic CXCR2 receptor coupled with natural CXCR3 and CXCR4 receptors to promote cell trafficking

Cytokine Support

IL15RF, IL7RF and others for cell potentiation

Stealth

Slippery receptor engineering to prevent rejection

Checkpoint KO

CD38 knock-out to promote metabolic fitness and prevent fratricide

Conditioning-free Therapy

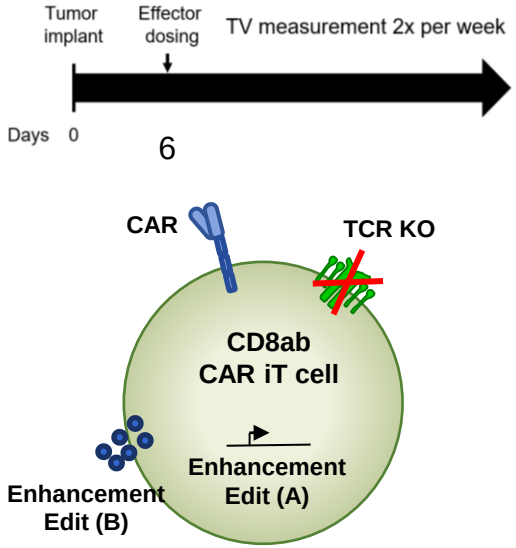
Allo-defense receptor (ADR) to redirect host immune cell alloreactivity and promote activation

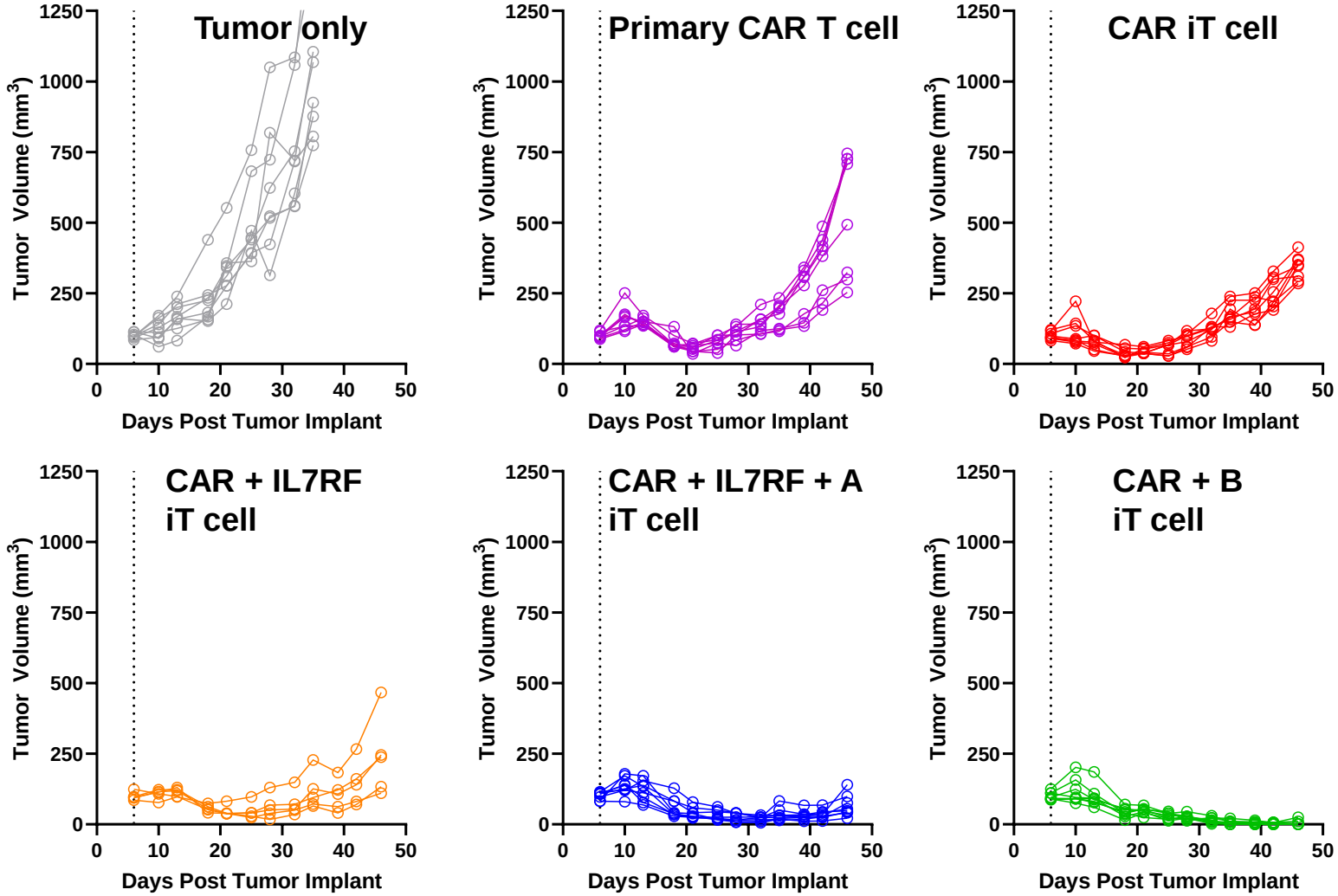
Fate THERAPEUTICS

Next-generation iPSC-derived CD8αβ CAR T cells

Creating Differentiated iPSC-derived CAR T Cells

Subcutaneous model of prostate cancer





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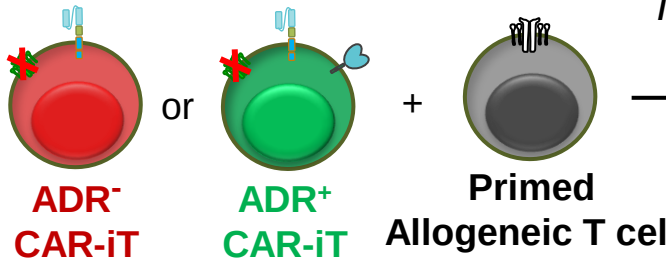
Valamehr et al. 2022 ASGCT Spotlight on IO 2023

Next-generation iPSC-derived CD8αβ CAR T cells

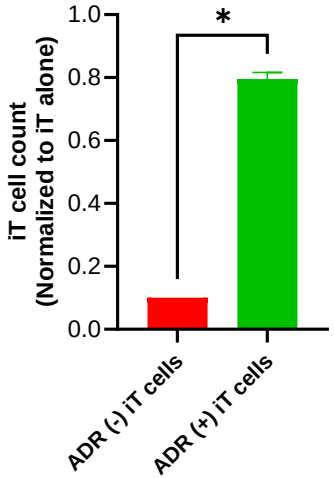
Integration of ADR Technology Enables Resistance to Allogeneic Immune Cells

In vitro allo-resistance and function

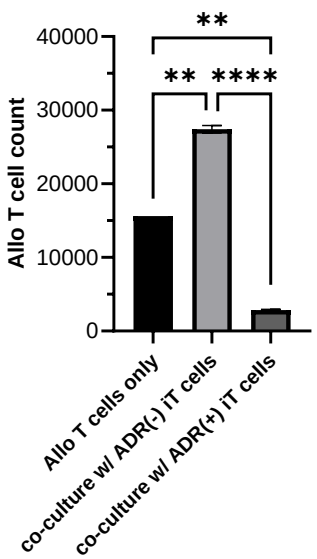


In vivo allo-resistance and function

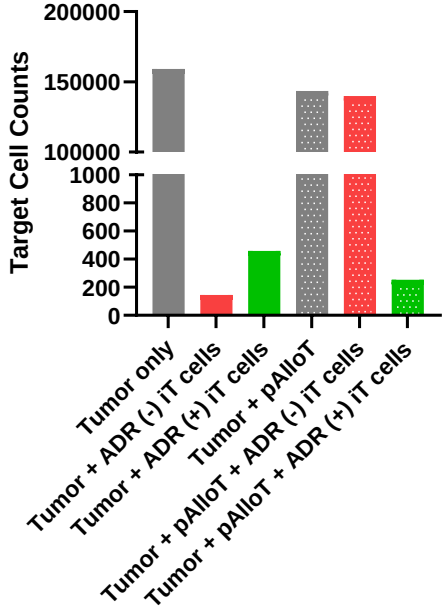
CAR-iT cell count (72 hrs)



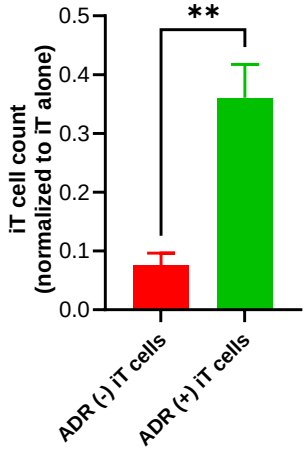
Allo T cell count (72 hrs)



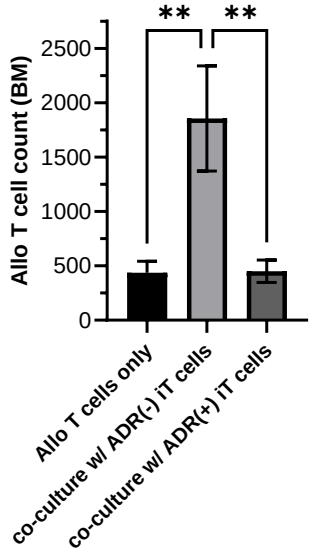
Target cell elimination in allogeneic setting (8 Days)



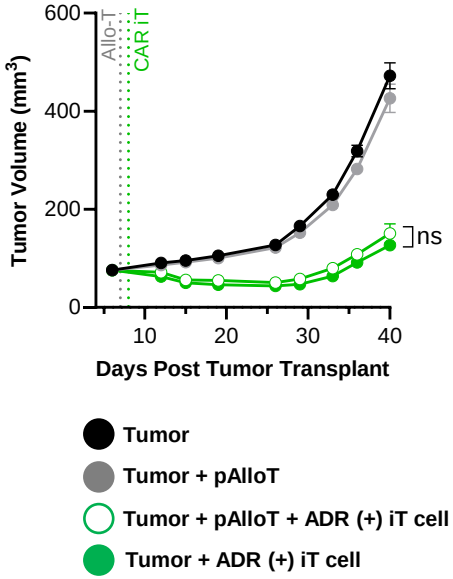
CAR-iT cell count in BM (14 days)



Allo T count in BM (14 days)



Tumor control in allogeneic setting (40 days)

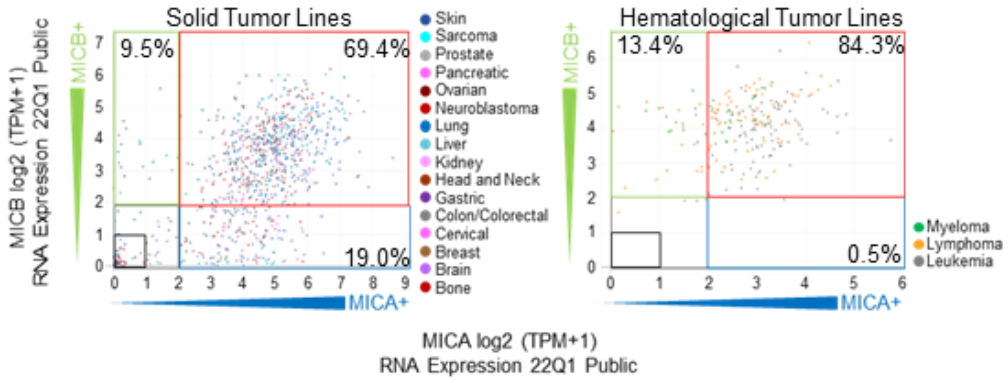


Unpublished data of Fate Therapeutics

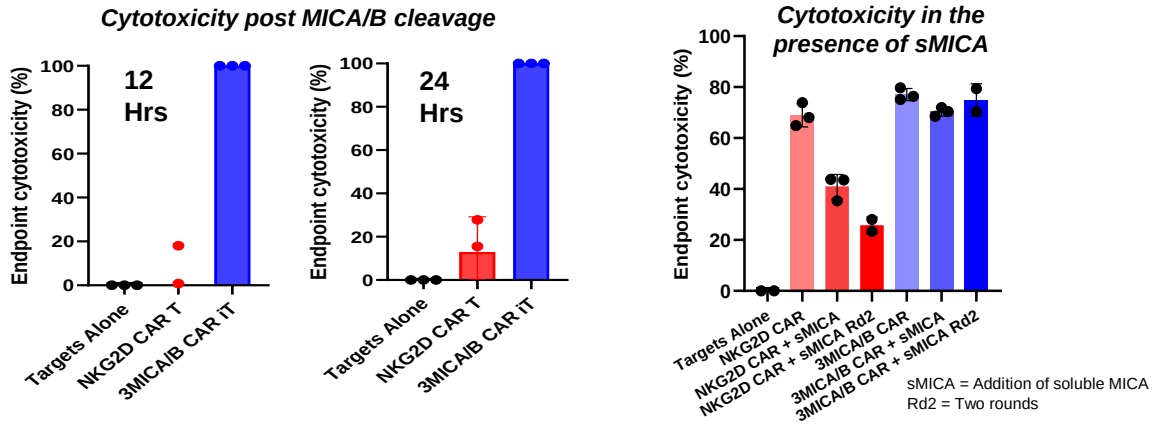
MICA/B-targeted CAR T Cells

α3 Domain Targeting Uniquely Eliminates Broad Array of Tumors in Preclinical Studies

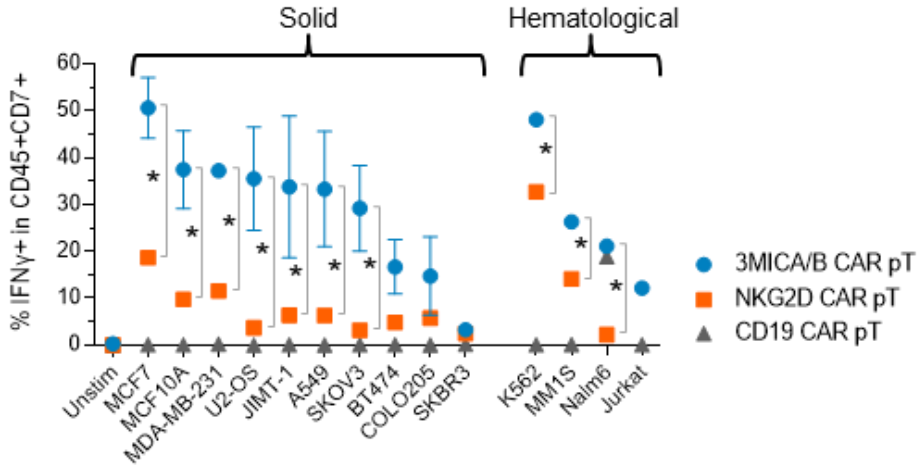
MICA and MICB are stress ligands expressed on many cancers



CAR targeting the alpha 3 domain is uniquely resistant to shedding and interference by soluble MICA/B



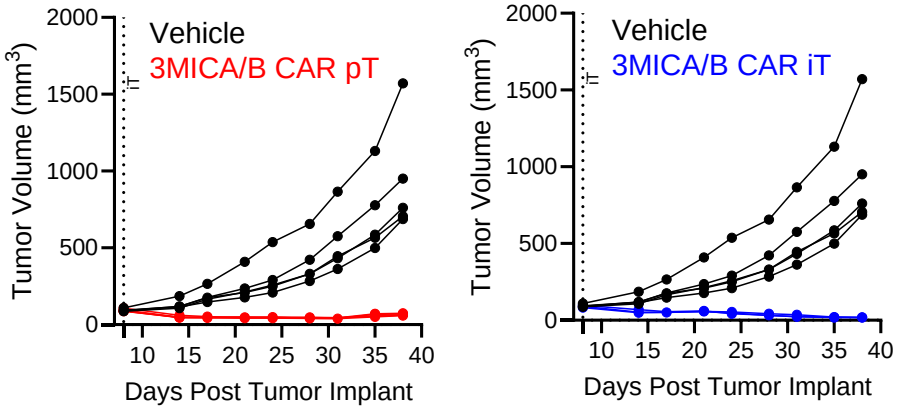
CAR targeting the alpha 3 domain demonstrates pan tumor recognition with enhanced activity over NKG2D CAR



Goulding, J. et al Cell Med. 2023 Jul 14;4(7):455-477

Development of Next Gen CAR iT cells targeting the alpha 3 domain of MICA/B

Prostate Cancer Subcutaneous Model
(PC3, single dose effector, no cytokine support)



Black = Tumor only
Blue = iPSC-derived CAR T cells
Red = primary CAR T cells

Garcia et al. 2024 ASGCT Annual Conference

2024 Milestones

Upcoming 2024 Key Milestones

Autoimmunity	• Present initial clinical data for FT819 in SLE • Dose first SLE patient as add-on to commonly-used treatment regimen • Clear multi-indication IND for FT522 and initiate enrollment without conditioning chemotherapy
Oncology	• Present clinical POC data for FT522 without conditioning chemotherapy in B-cell lymphoma • Present clinical POC data for FT825 supporting multiple MOAs for solid tumors
Platform	• Disclose new target(s) and product configurations for at least 2 next-generation cell product candidates
Platform	• Year-end cash, cash equivalents and investments > $270M • GAAP Operating Expenses between $215-230M

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Better Cells For Better Therapies®