

Better Cells For Better Therapies™

Off-the-shelf Cell-based Cancer Immunotherapy

Developing First-of-kind Cell Products using Clonal Master iPSC Lines

November 2019

www.fatetherapeutics.com

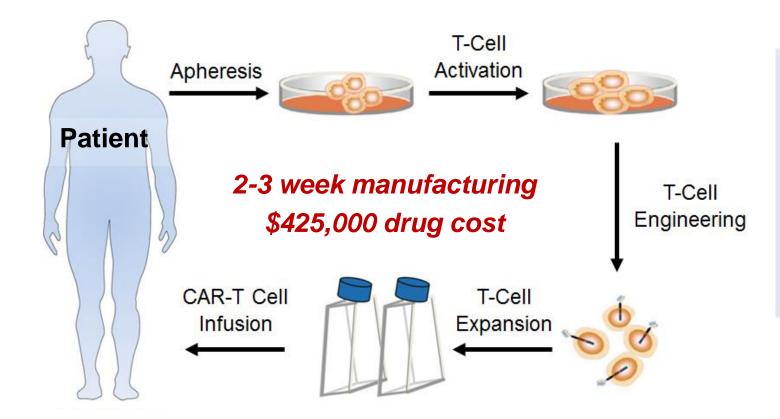


This presentation contains "forward-looking statements" within the meaning of the Private Securities Litigation Reform Act of 1995, including statements regarding the Company's research and development activities and its progress, plans and timelines for its manufacture, preclinical development and clinical investigation of its product candidates, the timing for the Company's receipt of data from its clinical trials and preclinical studies, the Company's clinical development and regulatory strategy, and the therapeutic and market potential of the Company's product candidates. 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 prior studies of its product candidates will not be 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, 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.



First Innings of Cell Therapy Development

Patient-derived CAR-T Cell Immunotherapy



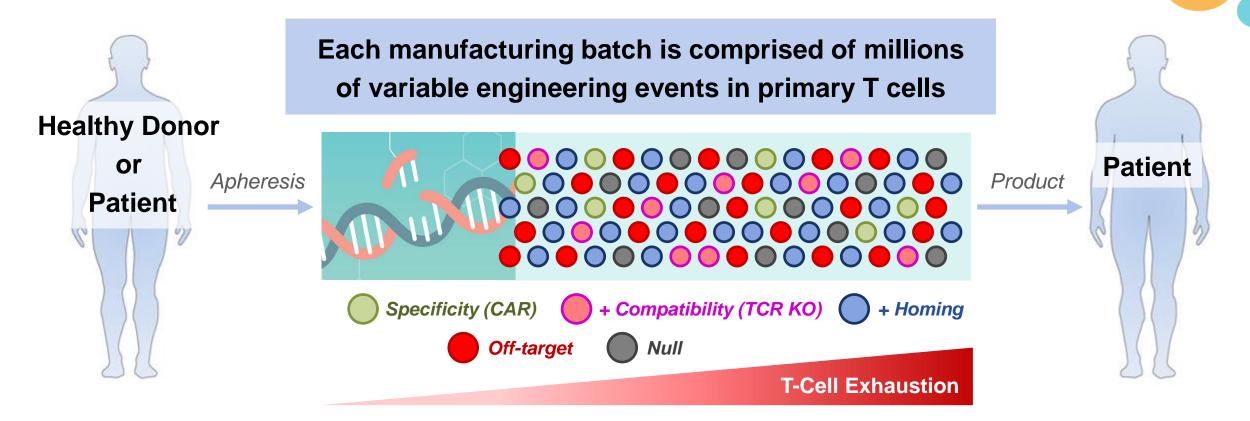
"When factoring in all the costs associated with CAR T-cell therapy, hospitals may charge as much as \$1.5 million or more to avoid losing money." Richard T. Maziarz, MD Professor of Medicine, Oregon Health & Science University's Knight Cancer Institute

Impaired Starting Material | Random & Variable Engineering | Complex Logistics Heterogeneous Drug Product | Expensive | Single-dose Limitation



First Innings of Cell Therapy Development

Batch-to-Batch Engineering is Expensive and Results in Significant Product Heterogeneity



How do we build on early successes and transition from a heterogenous <u>process</u> to the cost-effective delivery of optimized cell <u>products</u>?



First Innings of Cell Therapy Development

Example: Multiplexed CRISPR / Cas9 Gene Editing of Patient T Cells





49 First-in-Human Assessment of Feasibility and Safety of Multiplexed Genetic Engineering of Autologous T Cells Expressing NY-ESO -1 TCR and CRISPR/Cas9 Gene Edited to Eliminate Endogenous TCR and PD-1 (NYCE T cells) in Advanced Multiple Myeloma (MM) and Sarcoma

- Patient-specific Engineered TCR T-cell Therapy (University of Pennsylvania)
 - Mulitplexed CRISPR / Cas9 editing
 - Removal of TCRα (TRAC), TCRβ (TRBC) and PD-1 (PDCD1)
 - Transduced to express NY-ESO-1-specific TCR
- Reported CRISPR / Cas9 Gene Disruption Efficiency
 - TRAC = 44.3 to 49.4%
 - TRBC = 3.61 to 15.7%
 - PDCD1 = 15.6 to 20.2%

Significant inefficiencies and heterogeneity



Changing the Game in Cell-based Cancer Immunotherapy

The Potential to Characterize, Select and Renewably Use a Single Cell







What if we had the opportunity to engineer, characterize, select, and renewably use a <u>single</u> cell?

Changing the Game in Cell-based Cancer Immunotherapy

Universal, Off-the-Shelf Cell Products Derived from Renewable Master Cell Lines

Key Features	Cell Therapy 1.0 and 2.0	Cell Therapy 3.0
Cell Source	Patient and Donor Cells	Renewable Master Cell Line
Genetic Engineering	Random & Variable	Uniform & Complete
Characterization	Imprecise	Well-defined
Product Identity	Heterogeneous	Homogeneous
Manufacturing	Limited Dose Availability	Off-the-Shelf Availability
Cost-per-Dose	High	Low
Dosing	Single Dose	Multiple Doses / Multiple Cycles
Overall Paradigm	Process-centric	Product-centric

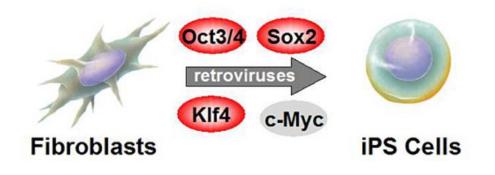


Human Induced Pluripotent Stem Cells (iPSCs)

Reprogramming Adult Somatic Cells to a Pluripotent State

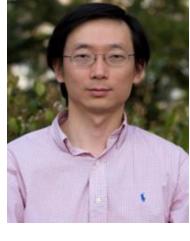
Generation of Human iPSCs

Fate Scientific Founders



Mouse iPS cells reported in 2006 Human iPS cells reported in 2007







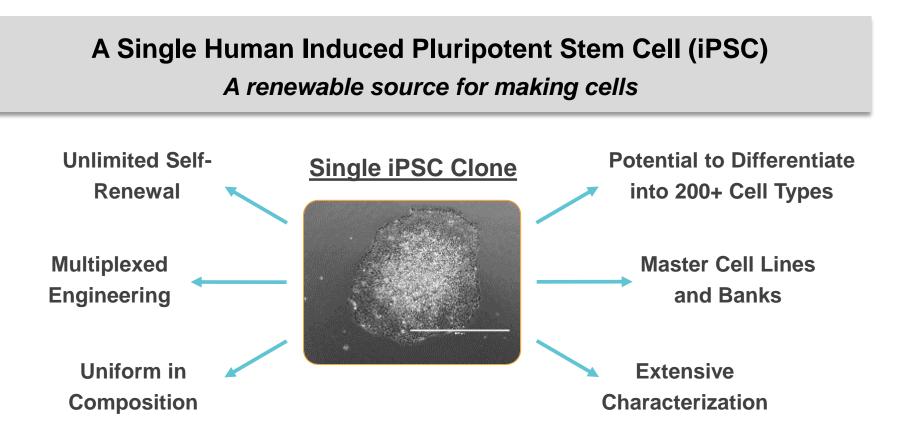






Unique Advantages of Human iPSCs

Isolation, Characterization & Selection of a Single iPSC Clone

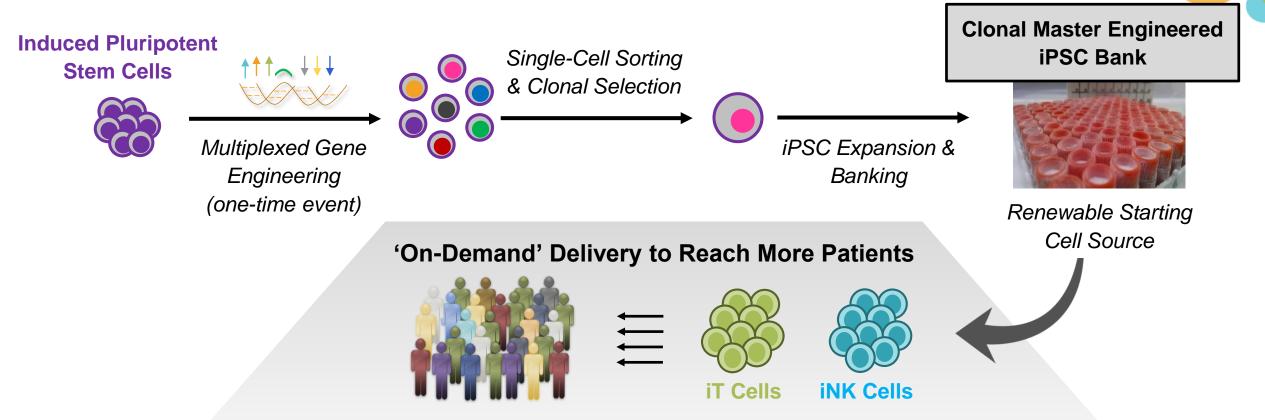


Fate Therapeutics' iPSC product platform is supported by an IP portfolio of 250+ issued patents and 150+ pending patent applications



Off-the-Shelf Cell-based Cancer Immunotherapy

iPSC Product Platform for Mass Production of Universal NK Cell and T-Cell Products



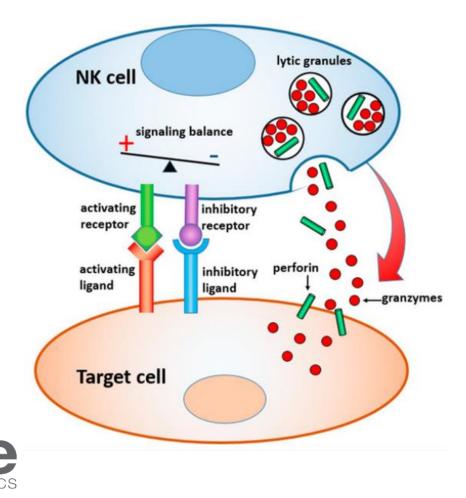
Clonal master iPSC lines are a renewable cell source that can be repeatedly used to mass produce homogeneous, cryopreserved cell product in a cost-effective manner



Natural Killer (NK) Cells

First Line of Defense against Tumors and Diverse Range of Pathogens

Array of Activating and Inhibitory Surface Receptors Mediate NK Cell Activity



Activating receptors convey multi-faceted effector function against tumor cells

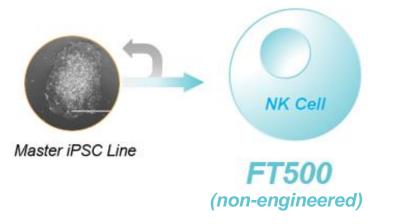
- Unique ability to recognize stressed / transformed cells, leaving healthy cells unharmed
- Engage and lyse antibody-coated tumor cells through CD16 Fc receptor (antibody-dependent cellular cytotoxicity, or ADCC)
- Direct killing through release of cytotoxic granules
- Trigger adaptive immune response through cytokine production

Inhibitory receptors can override activating signals

- KIR receptors balance activation through MHC-I molecule interactions
- Multiple immune checkpoint receptors (e.g., TIGIT, PD-1)

First-ever iPSC-derived Cell Therapy to Advance to Clinical Investigation in the U.S.

FT500 Product Candidate



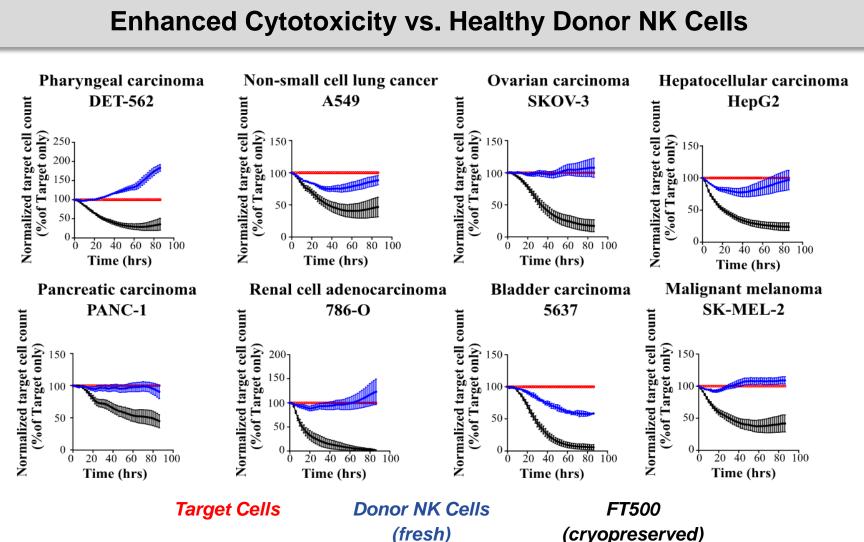
- High levels of expression of potent activating receptors (NKG2D, NKp30/40/46)
- High levels of secretion of cytolytic proteins (perforin and granzyme B)
- Low levels of expression of inhibitory receptors (KIR, TIGIT, PD-1)

FT500 cGMP Manufacture

FT500 Cell Product				
Identity, CD45+	100%			
Identity, CD45+CD56+	98%	77-77		
Viability	80%	al thread		
Residual iPSCs	Not detected			
Packaging	Cryopreserved	Research and a second and a sec		
Availability	On-site	THE L		
Administration	Thaw-and-infuse 'on demand'			
Delivery	Outpatient setting			

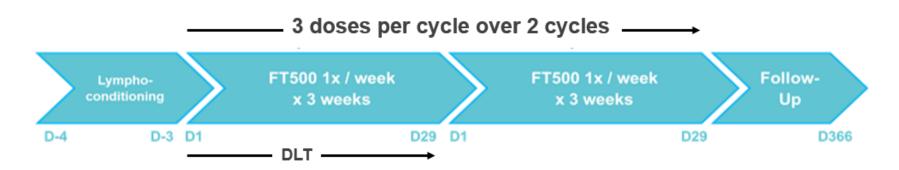
- Homogeneous cell product
- Low-cost per dose cGMP production
- Cryopreserved with high post-thaw viability
- Administered off-the-shelf in outpatient setting

In Vitro Cytotoxicity – Head-to-Head Comparison against Healthy Donor NK Cells



Phase 1 Dose Escalation – Monotherapy and Combination with Checkpoint Inhibitor Therapy

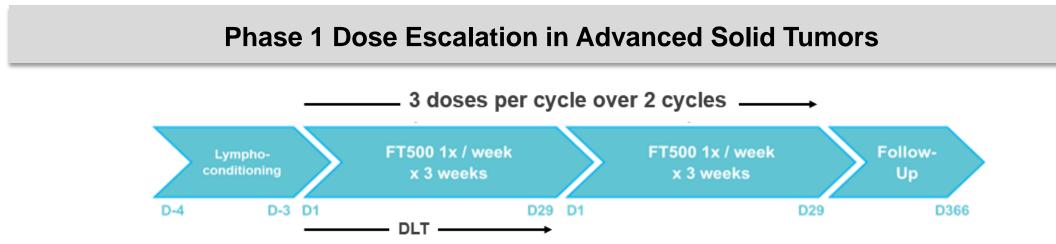
Phase 1 Dose Escalation in Advanced Solid Tumors



- **Regimen A**: Monotherapy
 - Salvage setting with patients having progressed or failed all FDA-approved therapies
- **Regimen B**: Combination with checkpoint blockade therapy (CBT)
 - Tumor types where CBT is approved
 - Salvage setting with patients having progressed or failed CBT
- Two dose levels
 - 100M cells / dose and 300M cells / dose x up to 6 doses



Phase 1 Dose Escalation – Monotherapy and Combination with Checkpoint Inhibitor Therapy



Assess Novel Treatment Paradigm

- First-ever U.S. clinical investigation of iPSC-derived cell
- Universal starting material (e.g., no patient matching)
- Multi-dose, multi-cycle treatment strategy
- One-time, outpatient lympho-conditioning
- No exogenous cytokine support

Key Read-outs: Safety and Tolerability

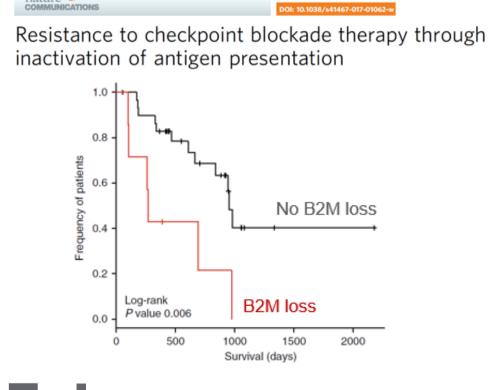
- Clinical
 - No DLTs or FT500-related SAEs (11 subjects)
- Molecular (i.e., patients' immunological response)
 - Immune cell recovery
 - Cytokine levels (e.g. CRS, GvHD)
 - > Anti-cell immunogenicity

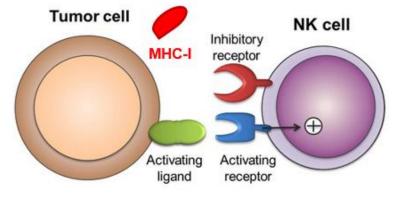
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Phase 1 Dose Expansion – Overcome Resistance to Checkpoint Inhibitor Therapy

Phase 1 Dose Expansion – Target Loss of MHC-I Tumor Escape Mechanism





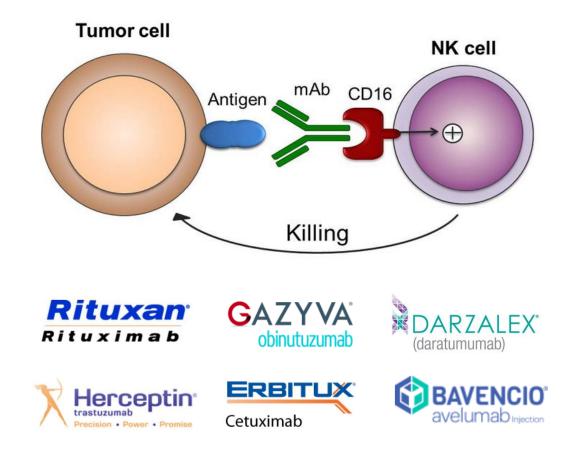
NK cells have the unique ability to recognize and kill cancer cells that have down-regulated MHC Class I, a major tumor escape mechanism

- Loss or down-regulation of MHC Class I is a major tumor escape mechanism
 in patients having progressed / failed checkpoint inhibitor therapy
- Several tumor cell mutations, including in B2M gene, disrupt MHC Class 1 expression
- B2M mutations are enriched in patients who are resistant to checkpoint blockage (~30%) and are associated with poor survival

CD16 Fc Receptor Mediates Antibody-Dependent Cellular Cytotoxicity (ADCC)

• CD16 is an activating receptor expressed on NK cells

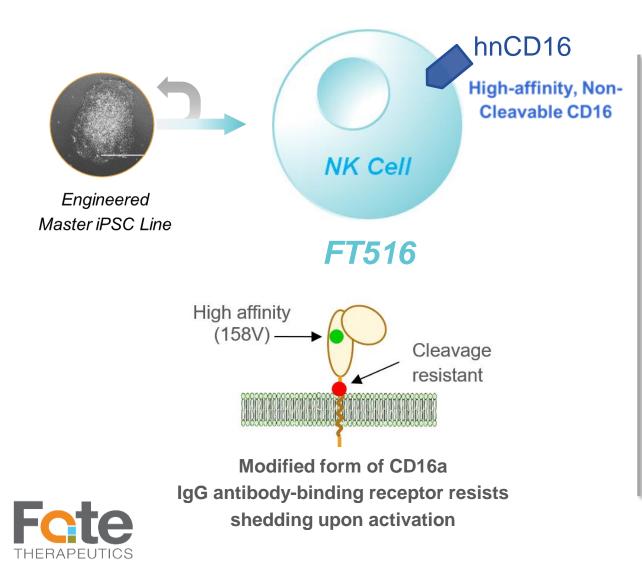
- Mediates antibody-dependent cellular cytotoxicity (ADCC), a potent anti-tumor mechanism by which NK cells recognize, bind and kill antibody-coated cancer cells
- CD16 occurs in two variants: high (158V) or low (158F) affinity for the Fc domain of IgG1 antibodies
 - Only ~15% of patients are homozygous for 158V
 - Numerous clinical studies with FDA-approved tumortargeting antibodies have demonstrated that patients homozygous for 158V have improved clinical outcomes
- CD16 shedding in the tumor microenvironment can significantly limit NK cell anti-tumor activity

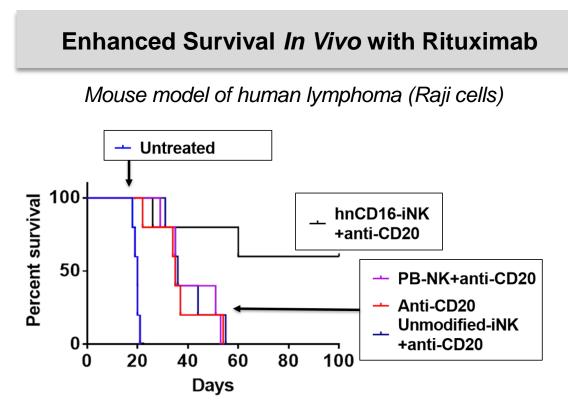




How to bring the 158V CD16 NK cell experience to <u>all</u> patients?

High-Affinity 158V, Non-Cleavable CD16 Fc Receptor for Enhanced ADCC

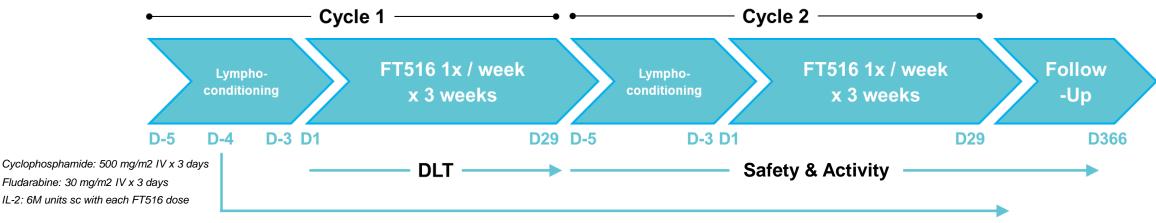




Median survival time for FT516 + anti-CD20 was not reached at Day 100

Phase 1 Study Design: Multiple Doses over Multiple Cycles for AML & Lymphoma





Regimen B: Rituximab 375 mg/m² IV

<u>Regimen A</u> – Monotherapy

- Relapsed / refractory AML
- Dose Escalation: 90M, 300M, 900M cells per dose
- Dose Expansion: up to 15 subjects

Regimen B – Rituximab Combination Rituxan

Rituximab

- Relapsed / refractory B-cell lymphoma
- Dose Escalation: 30M, 90M, 300M, 900M cells per dose + mAb
- Dose Expansion: up to 15 subjects



First Patients Treated in October 2019

Supported by Clinical POC in Hematologic Malignancies and Solid Tumors

- Recipient 🛨 Donor

100-

80

60-

40.

20

Donor NK Cell Therapy for AML

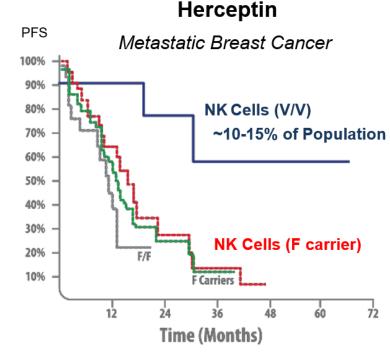
Phase 1 clinical trial in relapsed / refractory AML

- Cytokine-primed, donor-derived NK cell therapy
- Single-dose administration (0.5M, 1.0M, 10.0M per kg)
- 5 of 9 patients had clinical responses (4 CRs)
 - > No DLTs / GvHD

UPN	Dose level	Number of previous therapies	Pretreatment BM blast (%)	IWG response	DLT	GVHD
001	1	2	16	TF-PD	No	No
006	1	3	28	TF-PD	No	No
007	1	1	47	CR	No	No
800	2	3	17	TF-PD	No	No
009	2	3	80	MLFS	No	No
012	2	3	15	CR	No	No
017	3	3	69	TF-PD	No	No
019	3	4	15	CR	No	No
020	3	1	13	CRi	No	No



Monoclonal Antibody for Solid Tumors



Musolino et al, J. Clin Oncol, 26, 1789, 2008



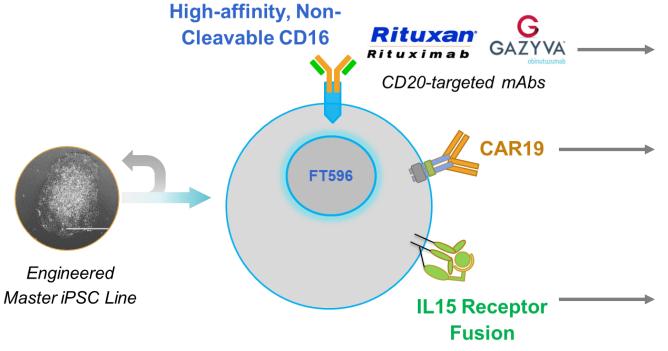
*** FATE is not affiliated with product candidate or clinical study ***

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Davs Post-Infusion

Potential Best-in-Class Cell-based Cancer Immunotherapy for B-cell Malignancies

First-ever Cell Therapy Engineered with <u>Three</u> Active Anti-tumor Modalities Cleared for U.S. Clinical Investigation



hnCD16: High-affinity 158V, non-cleavable CD16 Fc receptor that has been modified to augment antibody-dependent cellular cytotoxicity by preventing CD16 down-regulation and enhancing CD16 binding to tumor-targeting antibodies

CAR19: Chimeric antigen receptor optimized for NK cell biology, which contains a NKG2D transmembrane domain, a 2B4 co-stimulatory domain and a CD3-zeta signaling domain, that targets B-cell antigen CD19

IL-15RF: Interleukin-15 receptor fusion, a potent cytokine complex that promotes survival, proliferation and transactivation of NK cells and CD8 T cells

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IND Application Cleared for Clinical Investigation by FDA

Fate Therapeutics Announces FDA Clearance of IND Application for FT596 Offthe-Shelf, iPSC-derived CAR NK Cell Cancer Immunotherapy

FT596 Product Candidate Derived from Clonal Master iPSC Line Engineered with Three Anti-Tumor Functional Components

Designed to Overcome CD19 Antigen Escape and Improve Durability of Response by Targeting Multiple Tumor-associated Antigens

Off-the-Shelf Availability of FT596 Enables Rapid Time-to-Patient Treatment and Broader Patient Access

San Diego, CA – September 3, 2019 – Fate Therapeutics, Inc. (NASDAQ: FATE), a clinical-stage biopharmaceutical company dedicated to the development of programmed cellular immunotherapies for cancer and immune disorders, announced today that the U.S. Food and Drug Administration (FDA) has cleared the Company's Investigational New Drug (IND) application for FT596, the Company's first off-the-shelf chimeric antigen receptor (CAR) natural killer (NK) cell cancer immunotherapy which targets multiple tumor-associated antigens. FT596 is derived from a clonal master induced pluripotent

Clonal Master iPSC Line

Renewable source One-time iPSC engineering Scalable, cost-effective manufacture

Off-the-Shelf

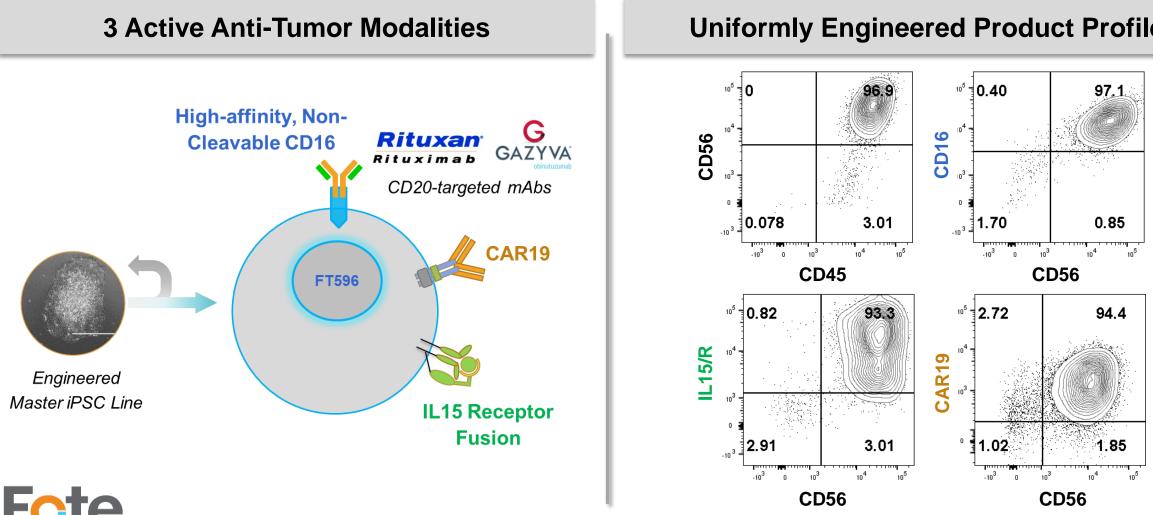
Rapid time-to-patient treatment Broader patient access Multi-cycle availability

Best-in-Class Profile

3 anti-tumor modalities Multi-antigen targeted Address antigen escape



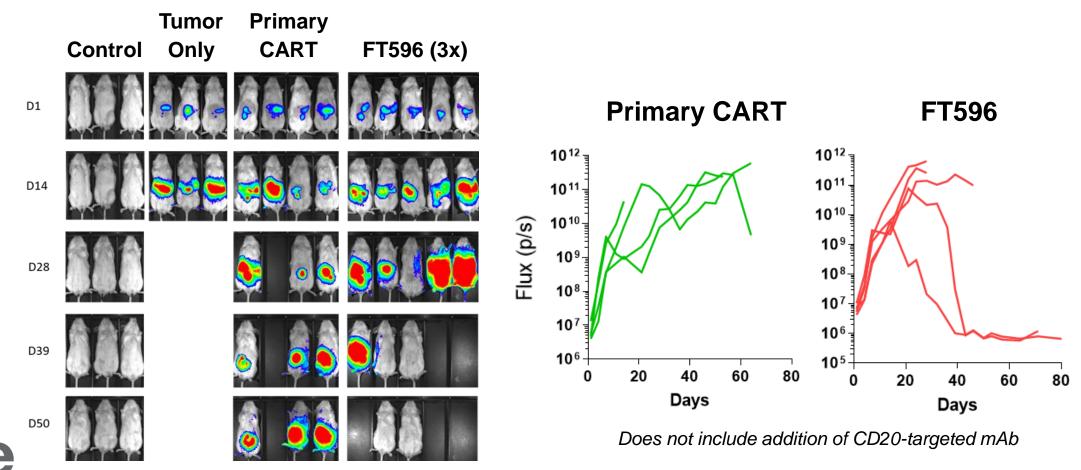
Uniformly Engineered with Three Active Anti-Tumor Functional Components



Uniformly Engineered Product Profile

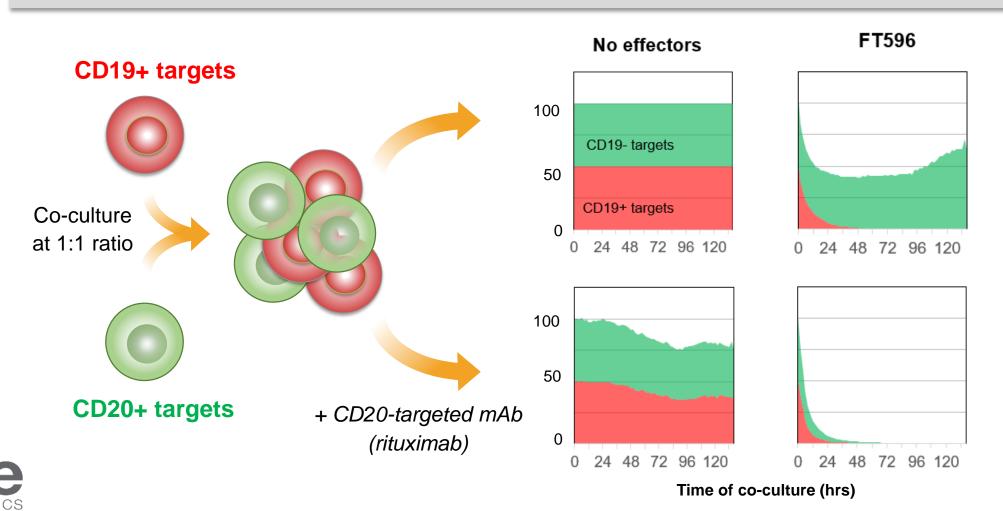
In Vivo Anti-Tumor Activity – Head-to-Head Comparison against Primary CAR T Cells

Single-Antigen Targeting of CD19 in Humanized Mouse Model of Lymphoma



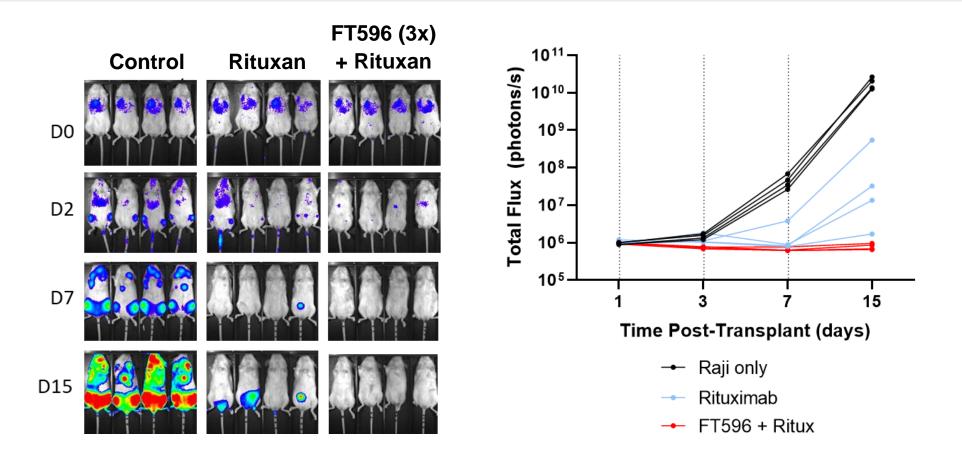
Leveraging CAR + hnCD16 to Overcome Tumor Heterogeneity and Antigen Escape

Targeting Multiple Tumor-Associated Antigens



Leveraging CAR + hnCD16 to Overcome Tumor Heterogeneity and Antigen Escape

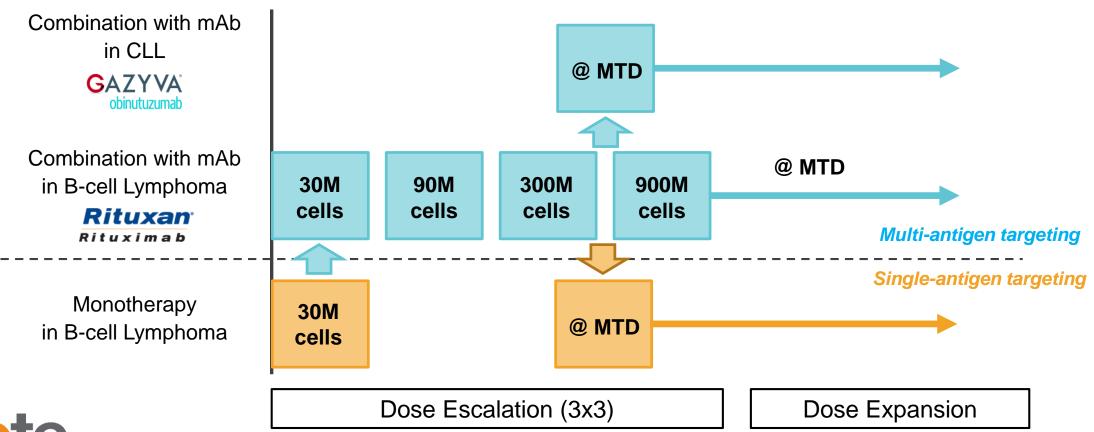
Dual-Antigen Targeting of CD19 and CD20 in a Xenograft Model of Lymphoma





Phase 1 Study Design in Relapsed / Refractory B-cell Lymphoma and CLL

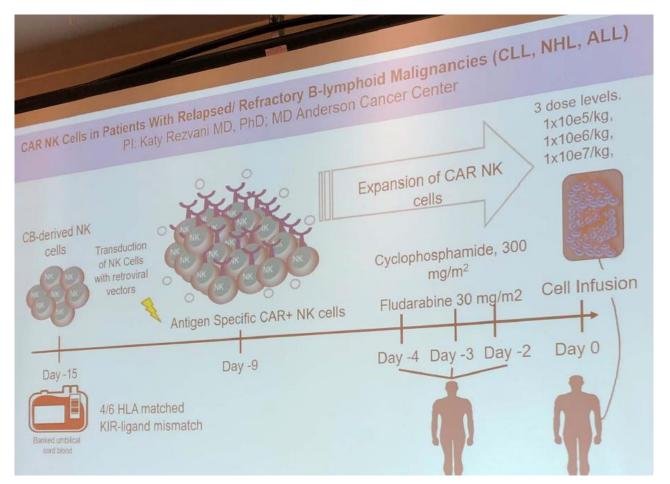






FT596 Supported by Clinical POC with Donor-derived CAR19 NK Cells

M.D. Anderson Cancer Center, Katy Rezvani, M.D., Ph.D. (NCT03056339)



- First-in-human clinical trial testing the safety and efficacy of donor-derived CAR NK cell therapy
 - Cord blood derived
 - Transduced with CAR19 (28z) / IL15 (secreted) / iCas9 (suicide)
- Treated 11 patients with r/r B-cell malignancies
 - r/r DLBCL (4); r/r CLL (5); r/r Follicular (2)
 - 3 dose levels (0.1M, 1.0M, 10M cells / kg)
- CR in 8/11 patients
 - CRs observed at all dose levels
 - CRs observed across all disease sub-types
- No CRS / neurotoxicity



As reported at ASGCT 2019

*** FATE is not affiliated with product candidate or clinical study ***

Off-the-Shelf NK Cell Cancer Immunotherapy Franchise

Systematic Build of Industry-Leading iPSC-derived NK Cell Product Pipeline

Universal, Off-the-Shelf NK Cell Cancer Immunotherapy Pipeline

Clonal Master iPSC Line	Synthetic Biology	FT500	FT516	FT596	FT538	FT576
Multi-faceted Innate Immunity		1	\	1	1	
+ High-Affinity, Non-cleavable CD16	Augment mAb therapy		1	1	1	1
+ IL-15 Receptor Fusion	Enhance NK cell function			1	 Image: A second s	 Image: A second s
+ CAR Insertion	Target tumor-associated antigen			CD19		BCMA
+ CD38 Knock-out	Resist CD38-mediated fratricide				1	 Image: A second s
	Total # of Synthetic Elements	0	1	3	3	4



IND Submissions for FT538 and FT576 in Multiple Myeloma Planned for 2020

Off-the-Shelf CAR T-Cell Cancer Immunotherapy

Memorial Sloan Kettering Collaboration





Dr. Michel Sadelain, MD, PhD Director, Center for Cell Engineering Memorial Sloan Kettering Cancer Center

LETTERS

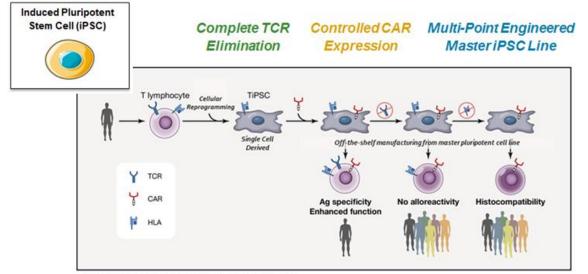
nature biotechnology

Generation of tumor-targeted human T lymphocytes from induced pluripotent stem cells for cancer therapy

Cell Stem Cell
Perspective

New Cell Sources for T Cell Engineering and Adoptive Immunotherapy

"Engineering therapeutic attributes into pluripotent cell lines is a breakthrough approach to renewably generate potent T-cell immunotherapies. This unique approach offers the prospect for off-the-shelf delivery of T-cell therapies with enhanced safety and therapeutic potential at the scale necessary to serve significant numbers of patients."





FT819 TRAC-encoded CAR 1XX Expression

Engineering Primary T Cells vs. Single iPSC Clone for TCR Elimination

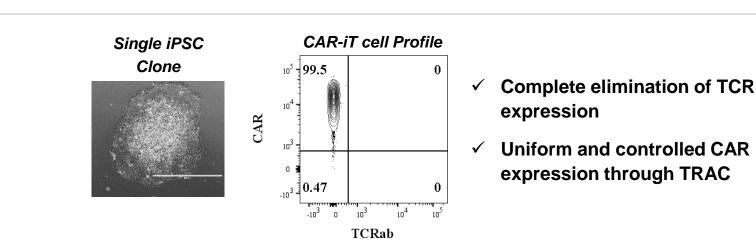
0

CAR

10³ 10⁴ 10⁵ 0

CRISPR Engineering: TCR Disruption + TRAC-encoded CAR Expression Cas9 + gRNA Primary T Cell AAV6 MOI 1×10^{6} 1×10^{6} 0 **Batch** 0.13 29.9 96.9 20.9 0.049 2.82 \checkmark 20% of T cells express 105 allo-reactive TCR 104 10³ Only 45% of T cells have TCR 2.97 0.029 69.9 0.14 30.7 45.6

TCR KO + CAR expression



10³ 10⁴ 10⁵ 0

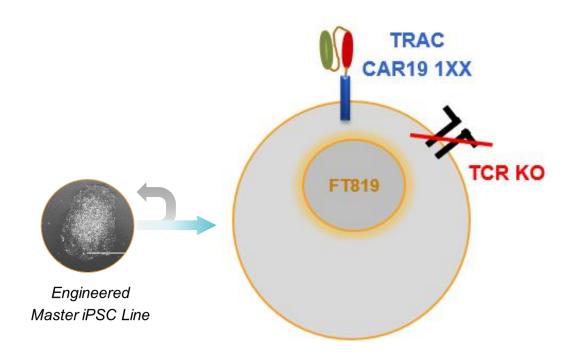
10³ 10⁴ 10⁵



FT819 Off-the-Shelf CAR19 T-Cell Product

Novel CAR19 Targeted to the TRAC Locus for Improved Safety and Efficacy

First-of-Kind Off-the-Shelf CAR T-cell Therapy Derived from Renewable Master iPSC Line Engineered to Uniformly Lack TCR Expression and Express Novel 1XX CAR19



1XX CAR19: Novel chimeric antigen receptor consisting of CD28 costimulatory domain and modified CD3z signaling domain for optimal effector cell persistence and anti-tumor potency

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 performance

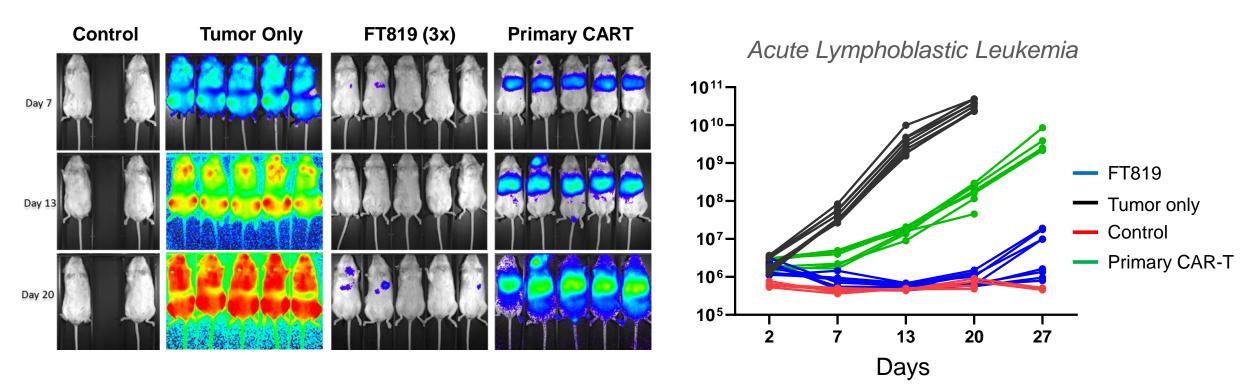
TCR-null: Bi-allelic disruption of TRAC at the clonal level for complete removal of TCR expression and the elimination for the possibility of GvHD in allogeneic setting



FT819 Off-the-Shelf CAR19 T-Cell Product

Novel CAR19 Targeted to the TRAC Locus for Improved Safety and Efficacy

In Vivo Head-to-Head Tumor Clearance vs. Primary CAR T Cells



IND Submission Planned for 1H20

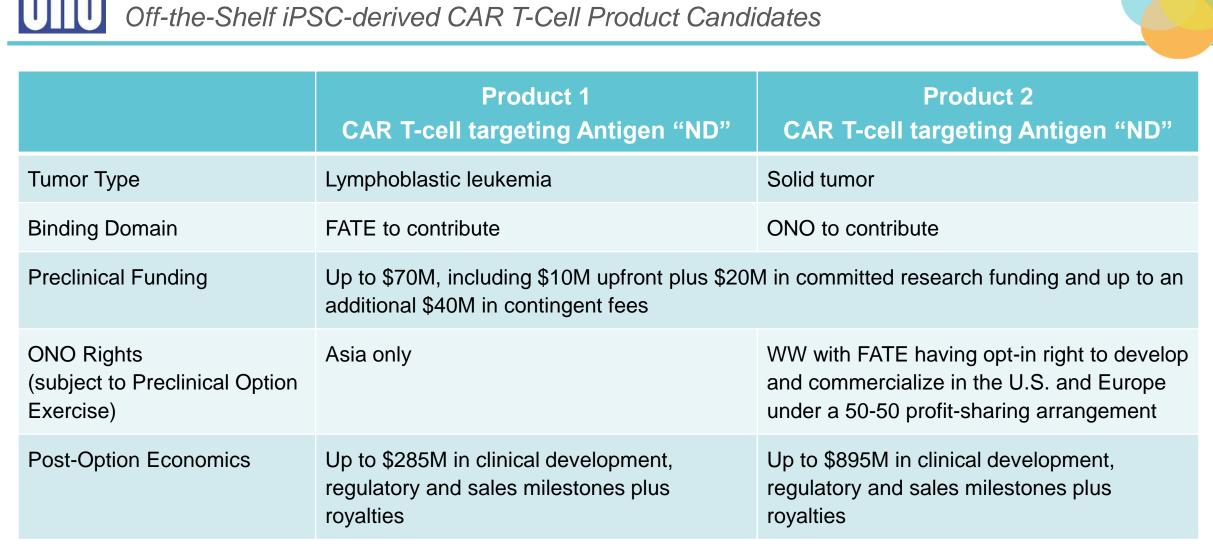


Off-the-Shelf CAR T-Cell Franchise

Foundational IP – Recently Issued Composition of Matter Patents

- U.S. Patent Number 10,287,606 entitled "Genomic Engineering of Pluripotent Cells"
 - Issued May 2019 (FATE owned)
 - A cell or population thereof, wherein (i) the cell is an induced pluripotent stem cell (iPSC), a clonal iPSC, or an iPSC line cell; (ii) the cell comprises a polynucleotide encoding at least one chimeric antigen receptor (CAR) introduced into a T cell receptor (TCR) alpha locus; (iii) an endogenous TCR alpha gene is knocked out; and (iv) expression of the polynucleotide encoding at least one CAR is under control of an endogenous TCR promoter of the TCR alpha locus
- U.S. Patent Number 10,370,452 entitled "Effective Generation of Tumor-targeted T cells derived from Pluripotent Stem Cells"
 - Issued August 2019 (MSK owned; licensed exclusively to FATE for all human therapeutic uses)
 - A population of T cells that are produced by *in vitro* differentiation of a pluripotent stem cell, wherein (i) the pluripotent stem cell expresses a chimeric antigen receptor (CAR), and (ii) the population of T cells comprises a T cell exhibiting a CD45RA+ CD27- CD28- CCR7- CD62L- phenotype





ONO Pharmaceutical Collaboration

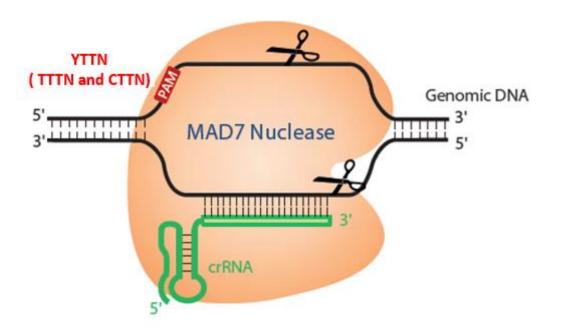




Next-Generation CRISPR Editing Technologies

MAD7 CRISPR Nuclease

- Patent-protected, RNA-guided, Class 2 Type V CRISPR nuclease isolated from *Eubacterium rectale*
- Improved features over commonly-used CRISPR-Cas9 nucleases:
 - Different PAM recognition sequences and cut efficiencies
 - Reduced sizes and differing enzyme kinetics
- MAD7 validated in FATE iPSC Product Platform
 - ~60% cleavage efficiency against CD38KO in iPSCs

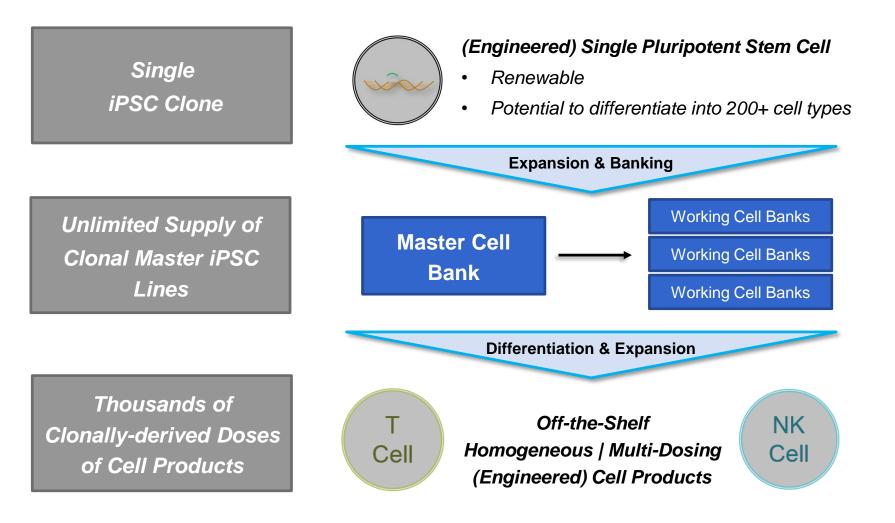


FATE secured royalty-free license to make and use MAD7 for research, development and commercialization of iPSC-derived cell products



iPSC Product Platform

Clonal Master iPSC Lines for Off-the-Shelf Cell Products





"to reach more patients in need"

Financial Summary

As of September 30, 2019



Three Months Ended September 30, 2019				
Revenue	\$2.4M			
Operating Expense, Adjusted ¹	\$25.0M			
Cash & Cash Equivalents	\$303.0M			
Employees	159			
Total Shares Outstanding ²	89.6M			

[1] Excludes non-cash stock-based compensation expense of approximately \$4.6M.

[2] Includes 14.0M shares of common stock from conversion of non-voting preferred stock.



Feite Therapeutics

Better Cells For Better Therapies™