Fate THERAPEUTICS

Better Cells For Better Therapies™

Programmed Cellular Immunotherapies

Corporate Overview

April 2018

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 Company's advancement of and plans related to the Company's product candidates, clinical studies, and research and development programs, the Company's progress and plans for its clinical investigation of ProTmune™, FATE-NK100 and its induced pluripotent stem cell-derived product candidates, the timing for initiation of the Company's planned clinical trials of its product candidates, the therapeutic potential of the Company's product candidates, the scope and enforceability of the Company's intellectual property portfolio, and the Company's financial condition. 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, including preclinical studies of ProTmune, FATE-NK100 and its induced pluripotent stem cell-derived product candidates, will not be observed in ongoing or future studies involving these product candidates, the risk of a delay in the enrollment or evaluation of subjects in any ongoing clinical studies, the risk that the Company may cease or delay preclinical or clinical development for any of its existing or future product candidates for a variety of reasons (including requirements that may be imposed by regulatory authorities and requirements for regulatory approval, difficulties or delays in subject enrollment in current and planned clinical trials, difficulties in manufacturing and supplying the Company's product candidates for clinical testing, and any adverse events or other negative results that may be observed during preclinical or clinical development), and the risk that the Company's expenditures may exceed current expectations for a variety of reasons. These statements are also subject to other risks and uncertainties as further detailed in the Company's most recently filed Form 10-Q, 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.

Fate THERAPEUTICS

Fate Therapeutics

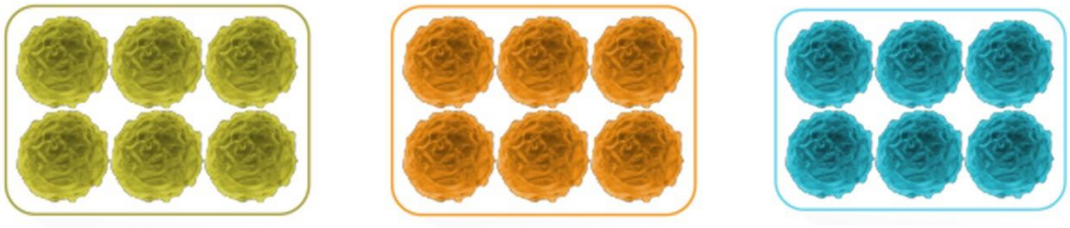
Mission

To develop first-in-class cell-based immunotherapies

for cancer and immune disorders

by programming cell function and fate

T cells | **CD34**$^{+}$ cells | **NK** cells



induced Pluripotent Cell Platform

for off-the-shelf engineered immunotherapies

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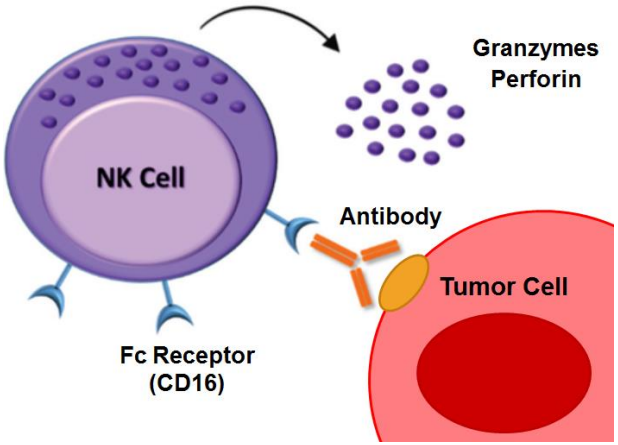
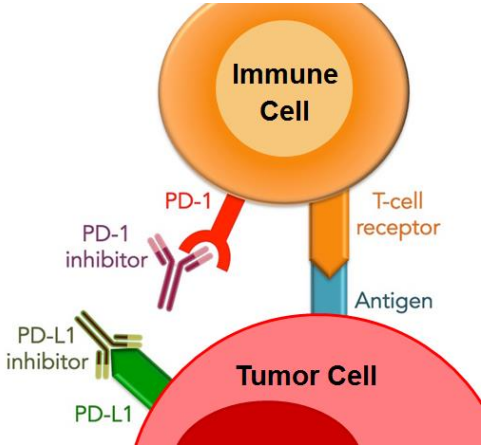
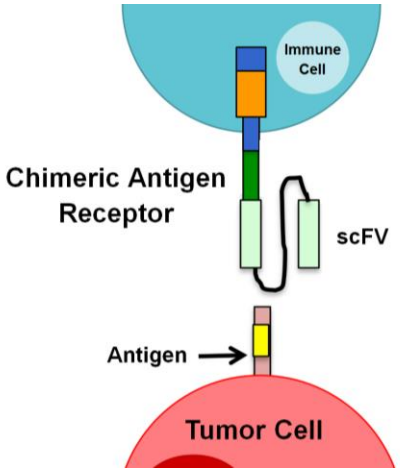
Proven Role of Cells in Cancer Immunotherapy

Antibody-Dependent Cellular Cytotoxicity (ADCC)	Immune Checkpoint Blockade	Targeted / Activated Cell Products







Rituxan Rituximab

Herceptin trastuzumab

ERBITUX CETUXIMAB

DARZALEX

OPDIVO (nivolumab)

KEYTRUDA (pembrolizumab)

JUNO THERAPEUTICS

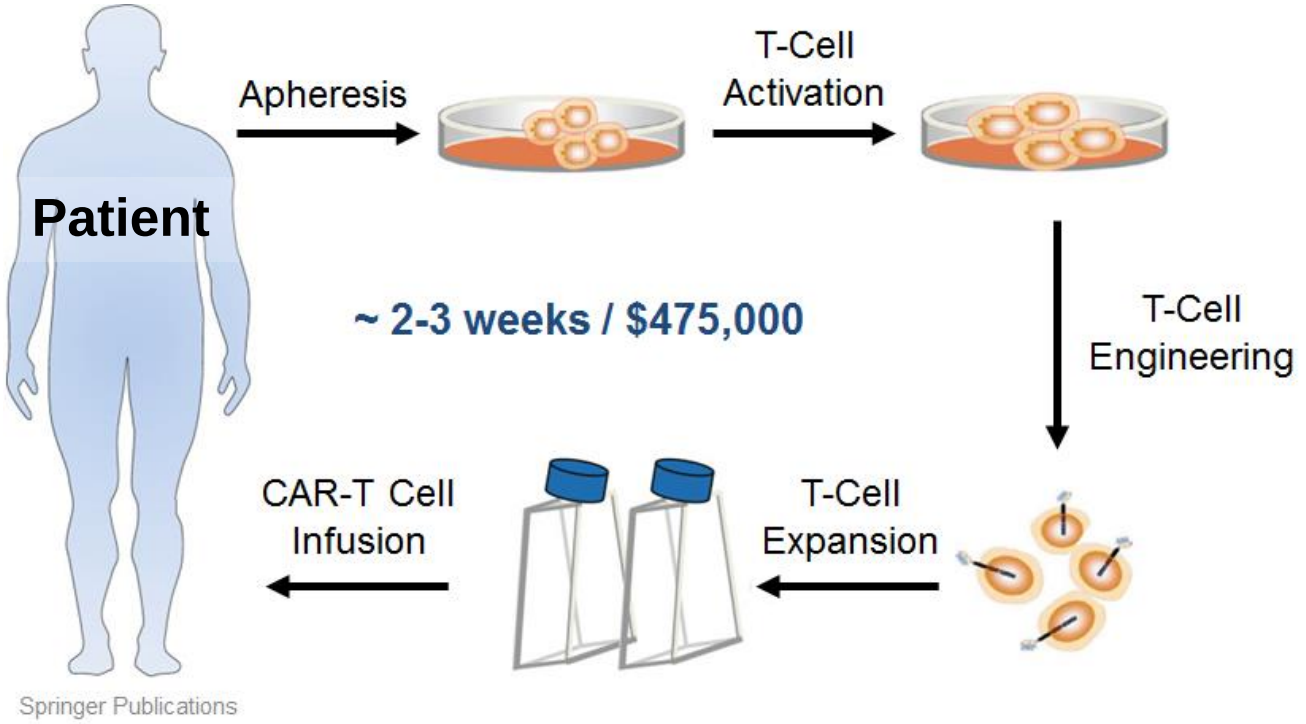
Kite Pharma

NOVARTIS

Fate THERAPEUTICS

Early Innings of Cellular Immunotherapy Development

Patient-derived CAR T Cells



Genetic Engineering
Random & Variable

Cell Composition
Heterogeneous

Manufacturing Yield
Single Patient per Run

How do we Build on Early Successes and Transition From a Personalized Process to the Delivery of an Optimized Cell Product?

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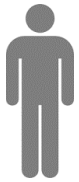
Better Cells for Better Therapies™

Our Approach to Cellular Immunotherapy

Programmed Donor Cell Products

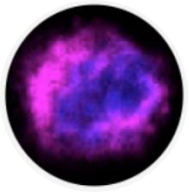


Donors

Cells from healthy donors with selected traits

Molecules

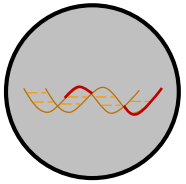
Ex vivo cell modulation to program biological properties



Cell Therapies

Cell products programmed for enhanced therapeutic function

Off-the-Shelf Cell Products

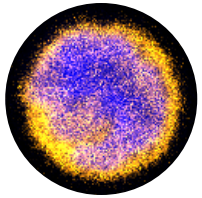


iPSC Line

Renewable pluripotent cell line with engineered functionality

Cell Bank

Ex vivo expansion / differentiation to derive clonal cell populations



Cell Therapies

Off-the-shelf engineered cell products for 1000s of patients

Fate THERAPEUTICS

Fate Therapeutics

First-in-Class Cellular Immunotherapy Pipeline

PROGRAM	PRECLINICAL	CLINICAL	RIGHTS
IMMUNO-ONCOLOGY			
FATE-NK100 – AML		**Phase 1**	Worldwide
FATE-NK100 – Ovarian		**Phase 1**	Worldwide
FATE-NK100 – Solid Tumor mAb Combo		**Phase 1**	Worldwide
FT500 (iNK Cell)	**OTS**	*Checkpoint Inhibitor Combination*	Worldwide
FT516 (Engineered hnCD16 iNK Cell)	**OTS**	*Monoclonal Antibody Combination*	Worldwide
FT538 (Engineered CD38- iNK Cell)	**OTS**	*Daratumumab Combination*	Worldwide
FT819 (Engineered CAR19 iT Cell)	**OTS**		Worldwide
IMMUNO-REGULATION			
ProTmune™ – Graft-versus-Host Disease		**Phase 2**	Worldwide
ToleraCyte™ – Autoimmune Disorders			Worldwide
FT300 (iMDS Cell)	**OTS**		Worldwide

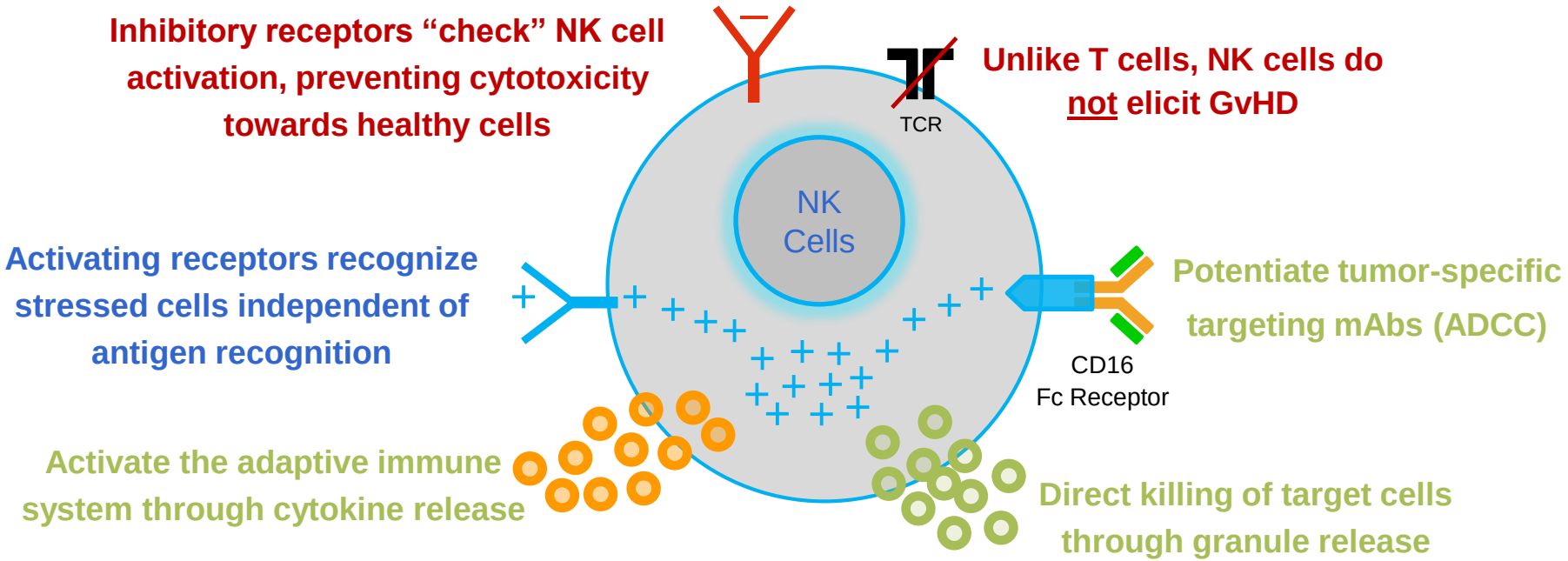
OTS *Off-the-Shelf using Clonal Master Induced Pluripotent Stem Cell (iPSC) Lines*

Immuno-Oncology Programs

Natural Killer Cells

Unique Properties Enable Off-the-Shelf Cancer Immunotherapy

- Effector function is <u>not</u> patient or single-antigen specific
- <u>Multi-faceted</u> effector function against tumor cells
- Use of <u>mismatched</u> cells has been shown to be well-tolerated / low risk of GvHD



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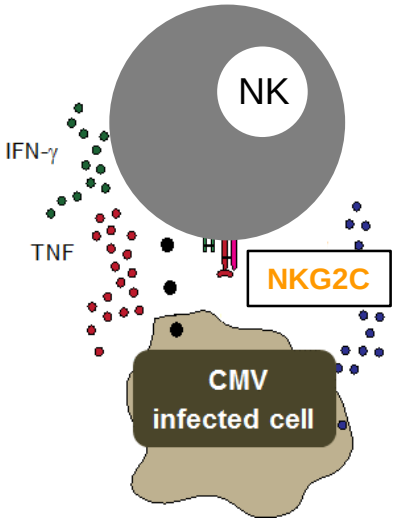
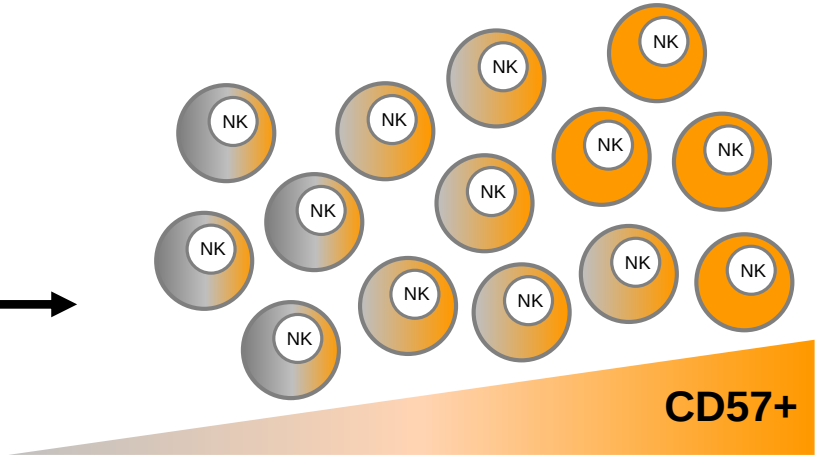
Adaptive Memory NK Cells

A Potent Subset of NK Cells with Unique Anti-tumor Attributes

Jeffrey S. Miller, MD

UNIVERSITY OF MINNESOTA
Driven to Discover℠





Formation of Adaptive Memory NK Cells

Correlated with reduced relapse risk and superior disease-free survival in HCT

Unique Subset of Activated NK Cells

Heightened Effector Function ***Enhance Persistence***

Resistant to Immune Checkpoint Pathways

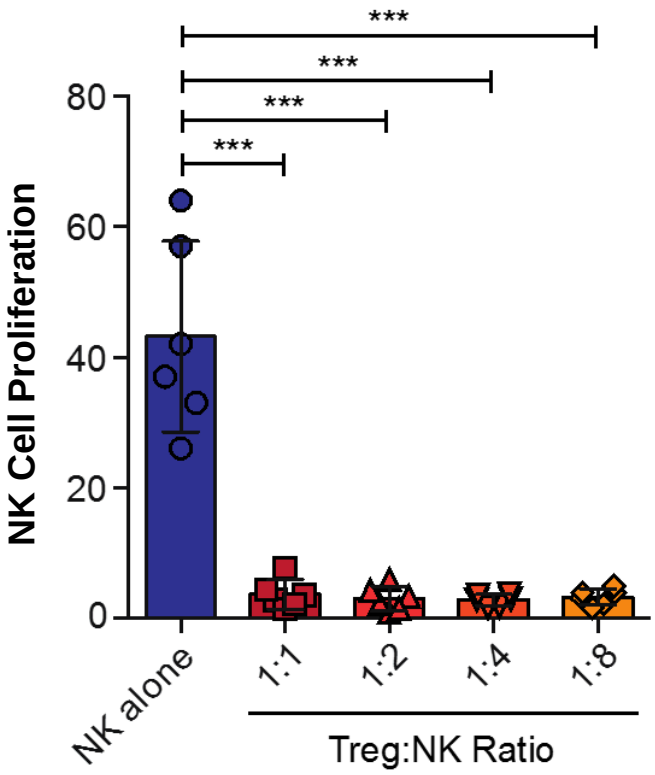
Fate THERAPEUTICS

Adaptive Memory NK Cells

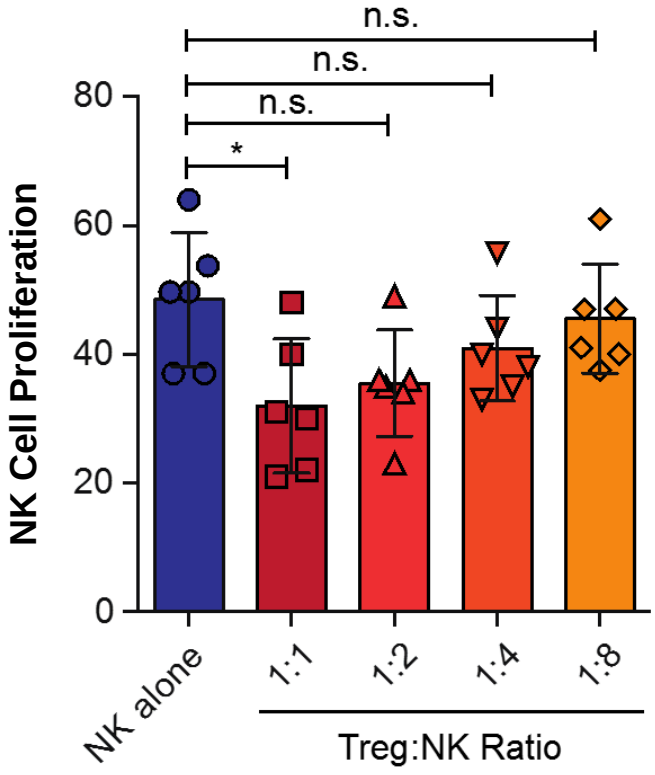
Resistance to Immune Checkpoint Pathways

UNIVERSITY OF MINNESOTA
Driven to Discover℠

Retained Proliferation Potential of Adaptive Memory NK Cells



Conventional NK Cells



Adaptive Memory NK Cells

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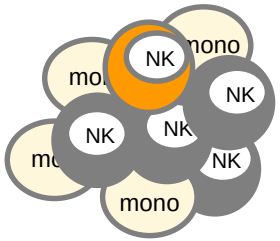
FATE-NK100

Realizing the Potential of Adaptive Memory NK Cells

CMV+ Donor

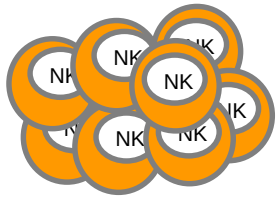
Apheresis

T & B Cell Depletion

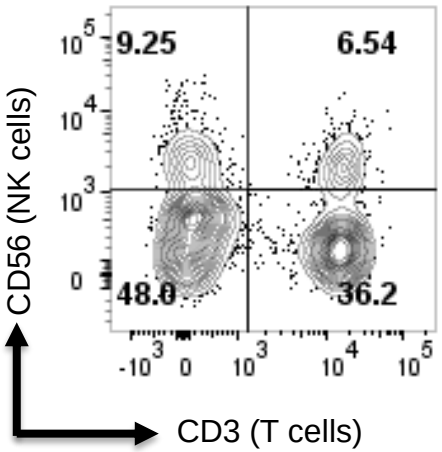


7-Day
Ex Vivo Modulation

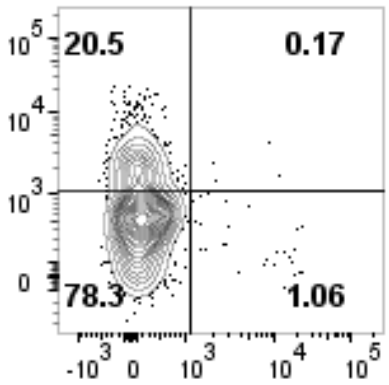
FT1238 + Cytokine
Feeder-free



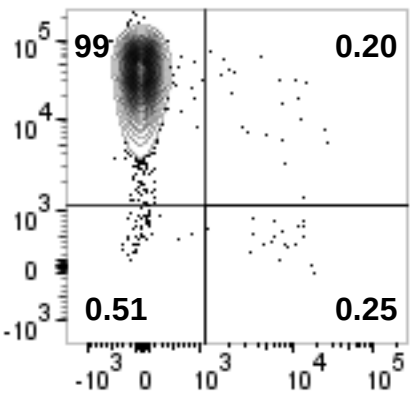
Day 0



Day 0 – Post-Depletion



Day 7 – Programming



Conventional NK Cell Therapies

Overnight (O/N) Cytokine-induced NK Cells

FATE-NK100

Adaptive Memory NK Cells

Cancer Research

GSK 3 inhibition drives maturation of NK cells and enhances their antitumor activity

Cichocki et. al. 10.1158/0008-5472.CAN-17-0799

FATE-NK100

Unique and Differentiated Properties of Adaptive Memory NK Cells

↑ NK cell product purity, potency and consistency

↑ NK cell maturation during product manufacture (↑CD57, ↑KIR, ↓NKG2A, ↓TIGIT)

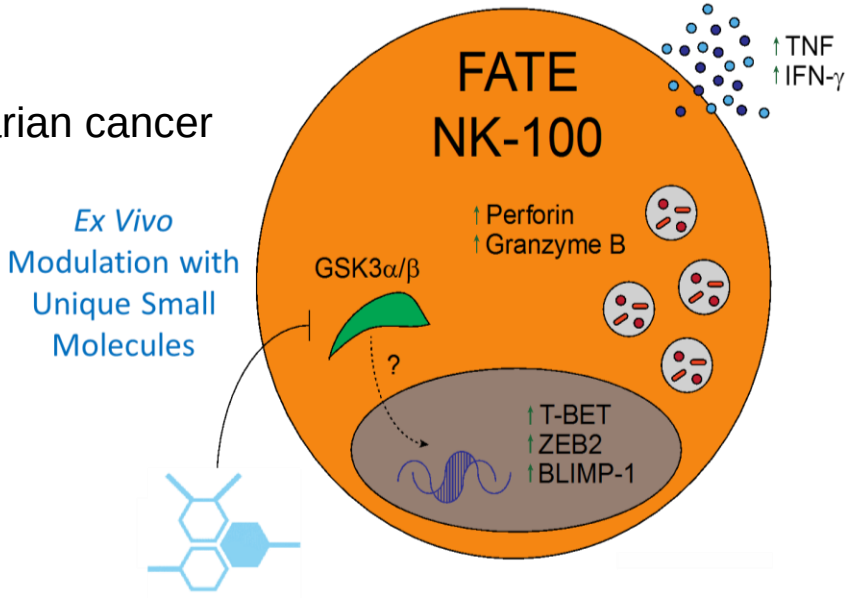
↑ Tumor necrosis factor (TNF) and interferon (IFN)-γ cytokine production

↑ Direct cytotoxicity against tumor targets *in vitro*

↑ ADCC in combination with mAbs against solid tumor targets in *in vivo* models

↑ *In vivo* persistence in preclinical models

↑ Tumor control in a xenogeneic model of ovarian cancer



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FATE-NK100

Launch of Multi-pronged Clinical Development Strategy

VOYAGE
Refractory / Relapsed AML

- Single IV infusion; accelerated dose-escalation; up to 3 dose levels
- 10-patient expansion at MTD
- Key read-outs: NK cell persistence; anti-leukemia activity; CRs
- Advanced through first two dose cohorts with no DLTs

APOLLO
Recurrent Ovarian

- Single IP infusion; accelerated dose-escalation; up to 3 dose levels
- 10-patient expansion at MTD
- Key read-outs: NK cell persistence; ORR by RECIST
- Advanced through first two dose cohorts with no DLTs

DIMENSION
mAb Combination in Solid Tumors

- Single IV infusion; accelerated dose-escalation; up to 3 dose levels
- 3 parallel arms: mono; + trastuzumab; + cetuximab
- Key read-outs: NK cell persistence; ORR by RECIST
- Advanced through first dose cohort (mono) with no DLTs

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FATE-NK100

The VOYAGE Study: Initial Clinical Observations

VOYAGE
Refractory / Relapsed AML

- Single IV infusion; accelerated dose-escalation; up to 3 dose levels
- 10-patient expansion at MTD
- Key read-outs: NK cell persistence; anti-leukemia activity; CRs
- Advanced through first two dose cohorts with no DLTs

	Dose Cohort 1 ($1x10^7$ TNC/kg)	Dose Cohort 2 ($2x10^7$ TNC/kg)
Age / Sex	67 / M	62 / F
History	Primary induction failure	Relapsed; refractory to conventional NK cell therapy
Leukemic Load	87% leukemic blasts in marrow	50% leukemic blasts in marrow
NK100 Persistence	76% of PB NK cells were of FATE-NK100 origin at Day 10	95% of PB NK cells were of FATE-NK100 origin at Day 10
Day 14 Activity	~50% reduction in leukemic blasts	Morphologic leukemia-free state (mLFS) †

Dose Cohort 3 (up to $1x10^8$ TNC/kg) currently enrolling

† Not sustained following a single IV infusion

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FATE-NK100

The APOLLO Study: Initial Clinical Observations

MEDICAL SCHOOL
UNIVERSITY OF MINNESOTA

APOLLO
Recurrent Ovarian

- Single IP infusion; accelerated dose-escalation; up to 3 dose levels
- 10-patient expansion at MTD
- Key read-outs: NK cell persistence; ORR by RECIST
- Advanced through first two dose cohorts with no DLTs

	Dose Cohort 1 ($1x10^7$ TNC/kg)	Dose Cohort 2 ($2x10^7$ TNC/kg)
Age / Sex	63 / F	71 / F
History	Platinum Resistant Ovarian Cancer	Stage IIIC Platinum Resistant Serous Carcinoma of Fallopian Tubes
Prior Therapies	5 prior lines of therapy	5 prior lines of therapy
Persistence	5% of Ascites NK cells were of FATE-NK100 origin (Day 5)	81% of Ascites NK cells were of FATE-NK100 origin (Day 5)
Day 28 Activity	Progressive Disease	Stable Disease with decrease splenic mass

Dose Cohort 3 (up to $1x10^8$ TNC/kg) currently enrolling

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Cell-based Cancer Immunotherapy

Advantages of an Off-the-Shelf Cell Product Paradigm

Key Features	Today	Tomorrow
Cell Source	Patient / Donor Cells	Master Cell Line
Genetic Engineering	Random & Variable	Precise & Complete
Manufacturing	Patient-specific	Off-the-Shelf
Product Consistency	Heterogeneous	Uniform & Well-defined
Therapeutic Functionality	Single MOA	Multiple MOA
Delivery	Delayed & Uncertain	On Demand
Dose-per-Patient	Single	Multiple
Overall Paradigm	**Patient-centric**	**Product-centric**

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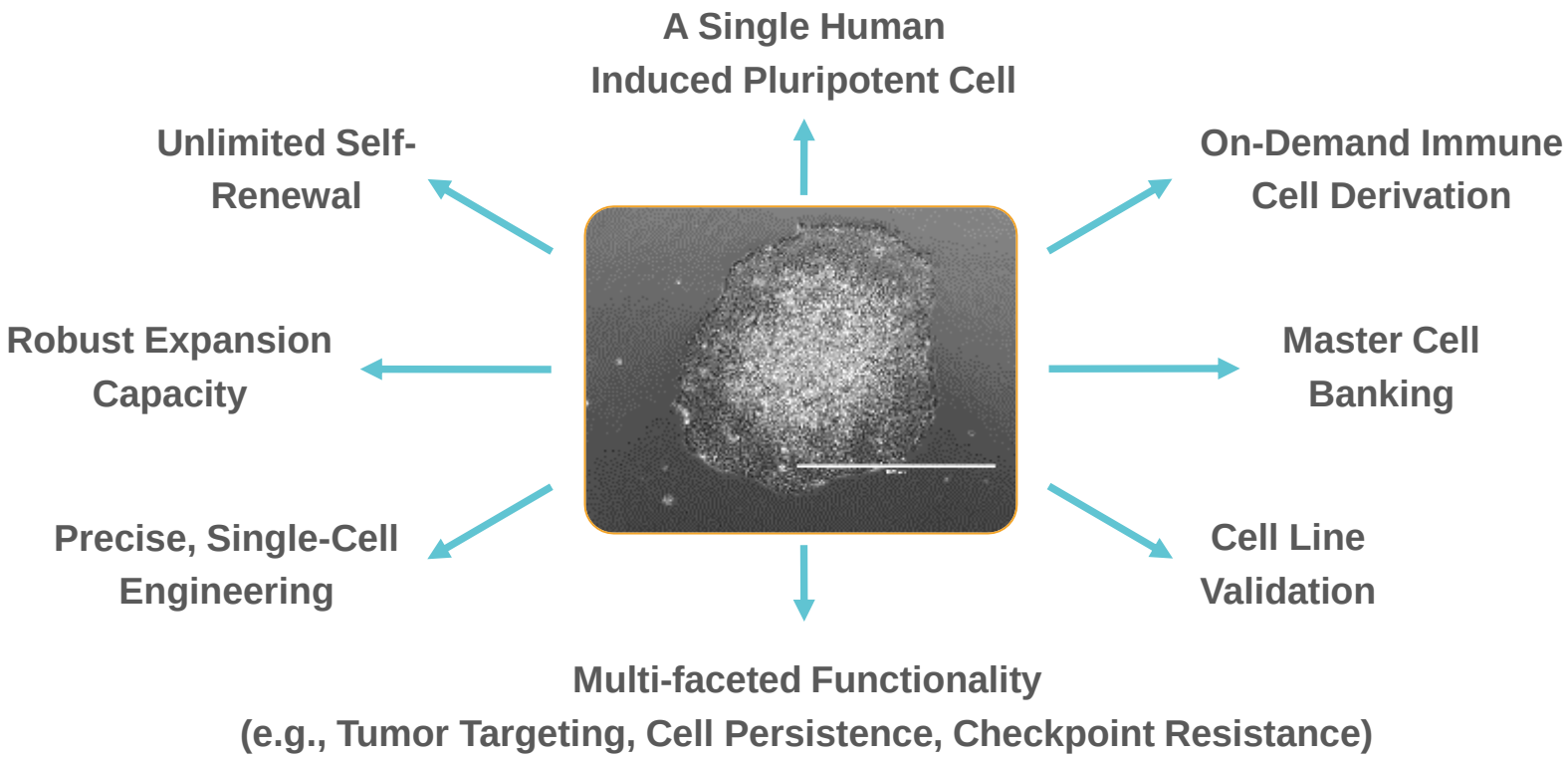
Human Induced Pluripotent Stem Cells

Renewable Source for Off-the-Shelf Cell Products



Renewable | Engineered

Clonal Cell Lines ---> Clonal Cell Products

Fate THERAPEUTICS

iPSC Product Platform

Fate Therapeutics' Transformative Approach to Cancer Immunotherapy

Single iPSC Clone

(Engineered) Single Pluripotent Stem Cell

- *Renewable*
- *Propensity to differentiate into 200+ cell types*

Expansion & Banking

Unlimited Supply of Clonal iPSC Master Cell Lines

Master Cell Bank → Working Cell Banks | Working Cell Banks | Working Cell Banks

Differentiation & Expansion

Thousands of Clonally-derived Doses of Cell Products

T Cell

Off-the-Shelf
Homogeneous | Multi-Dosing
(Engineered) Cell Products

NK Cell

Fate THERAPEUTICS

Off-the-Shelf NK- and T-Cell Products

Collaborations with Top Investigators and Leading Centers

59th American Society of Hematology (ASH) Annual Meeting

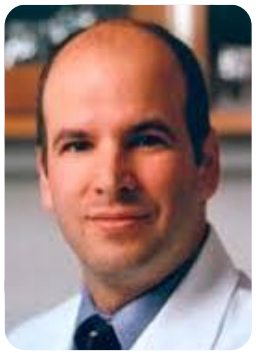
December 9-12, 2017 in Atlanta, Georgia

Clinical Translation of Pluripotent Cell-derived Off-the-Shelf NK Cell Cancer Immunotherapy

Jeffrey S. Miller, MD

University of Minnesota
Driven to Discover℠

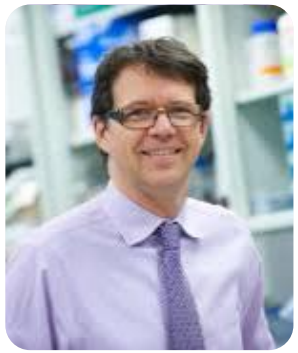
Engineered Human iPSCs with Novel CARs to Generate NK Cell Cancer Immunotherapies with Targeted Anti-Tumor Activity



Dan Kaufman, MD PhD

UC San Diego

Generation of Clonal Antigen Specific CD8ab+ Cytotoxic T-Lymphocytes from Renewable Pluripotent Stem Cells for Off-the-Shelf T Cell Therapeutics



Michel Sadelain, MD, PhD

Memorial Sloan Kettering Cancer Center

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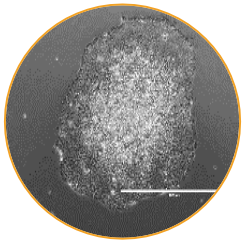
Off-the-Shelf NK Cell Products

Three Product Candidates Undergoing IND-Enabling Activities

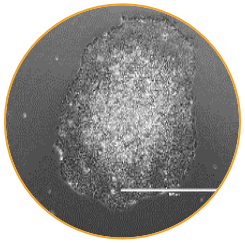


Master iPSC Line

- Clonal master iPSC line
- NK differentiation
- Homogenous cell product

OPDIVO™

KEYTRUDA®



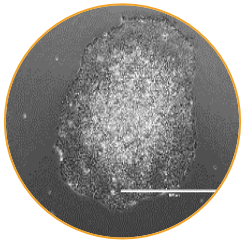
Engineered Master iPSC Line

- hnCD16 insertion
- Clonal master iPSC line
- NK differentiation
- Homogeneous cell product

Herceptin® trastuzumab

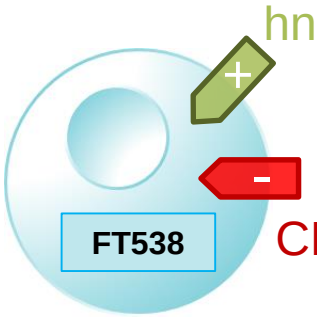
ERBITUX® CETUXIMAB

Rituxan® Rituximab



Engineered Master iPSC Line

- hnCD16 insertion / CD38 KO
- Clonal master iPSC line
- NK differentiation
- Homogenous cell product

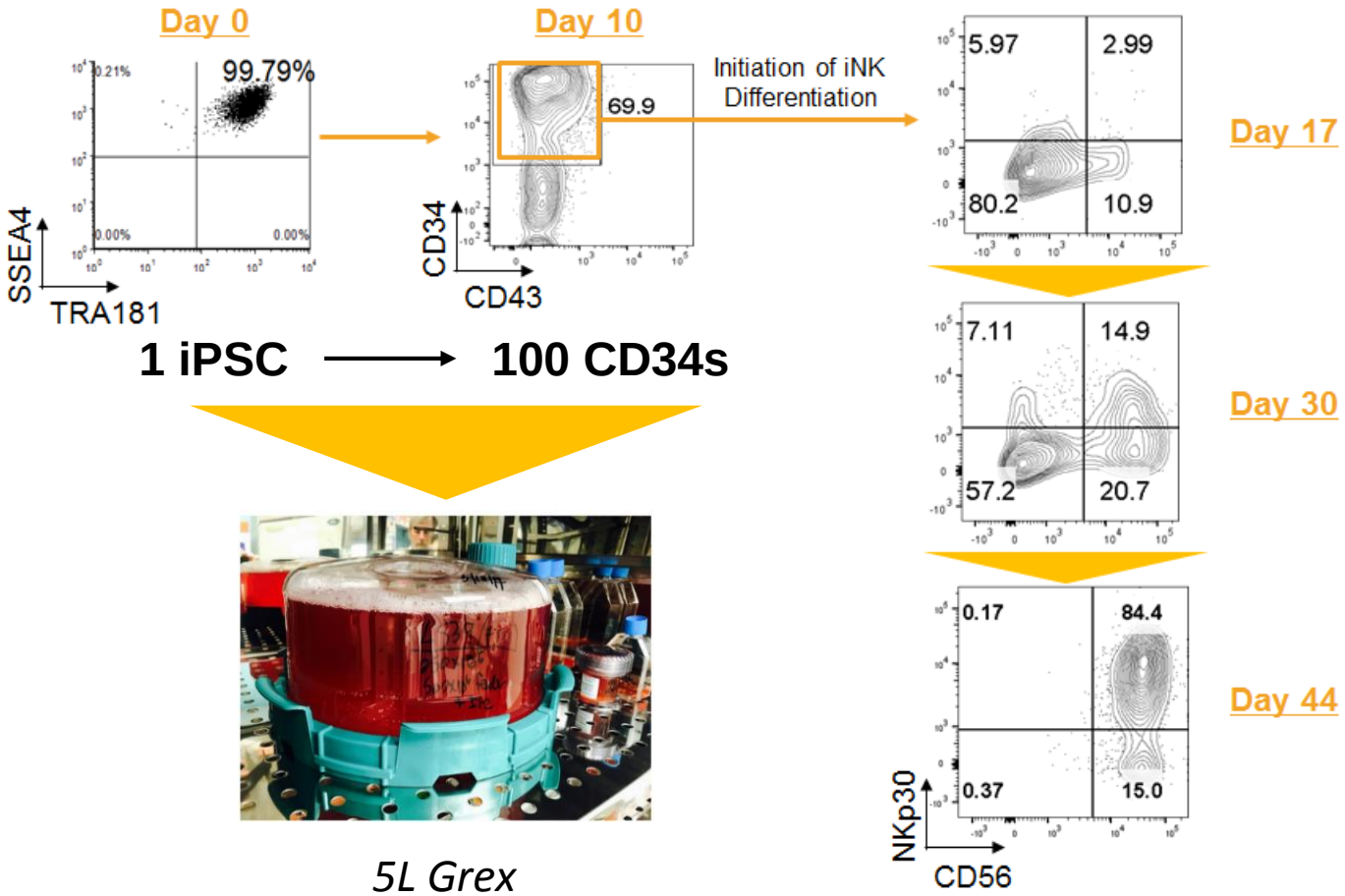


DARZALEX®

Fate THERAPEUTICS

FT500 Manufacturing

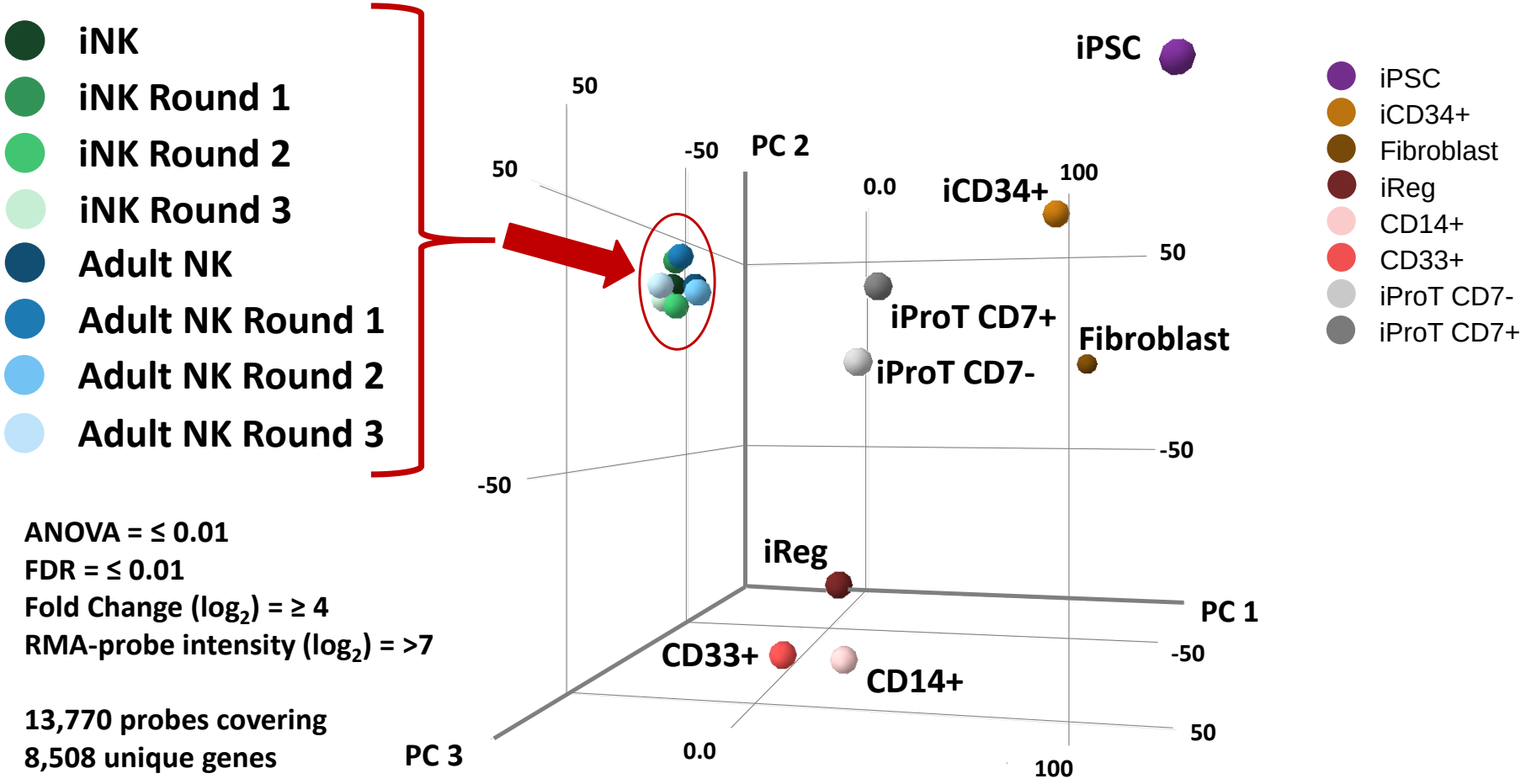
Robust Differentiation Protocol for iPSC-derived NK Cell Production



Large clonal population of NK cells in a single production run capable of yielding hundreds of doses of homogeneous drug product for off-the-shelf delivery to patients

FT500 Biological Properties

Comparative Gene Expression Analysis vs. Peripheral Blood NKs



ANOVA = ≤ 0.01
FDR = ≤ 0.01
Fold Change (log_2) = ≥ 4
RMA-probe intensity (log_2) = >7

13,770 probes covering
8,508 unique genes

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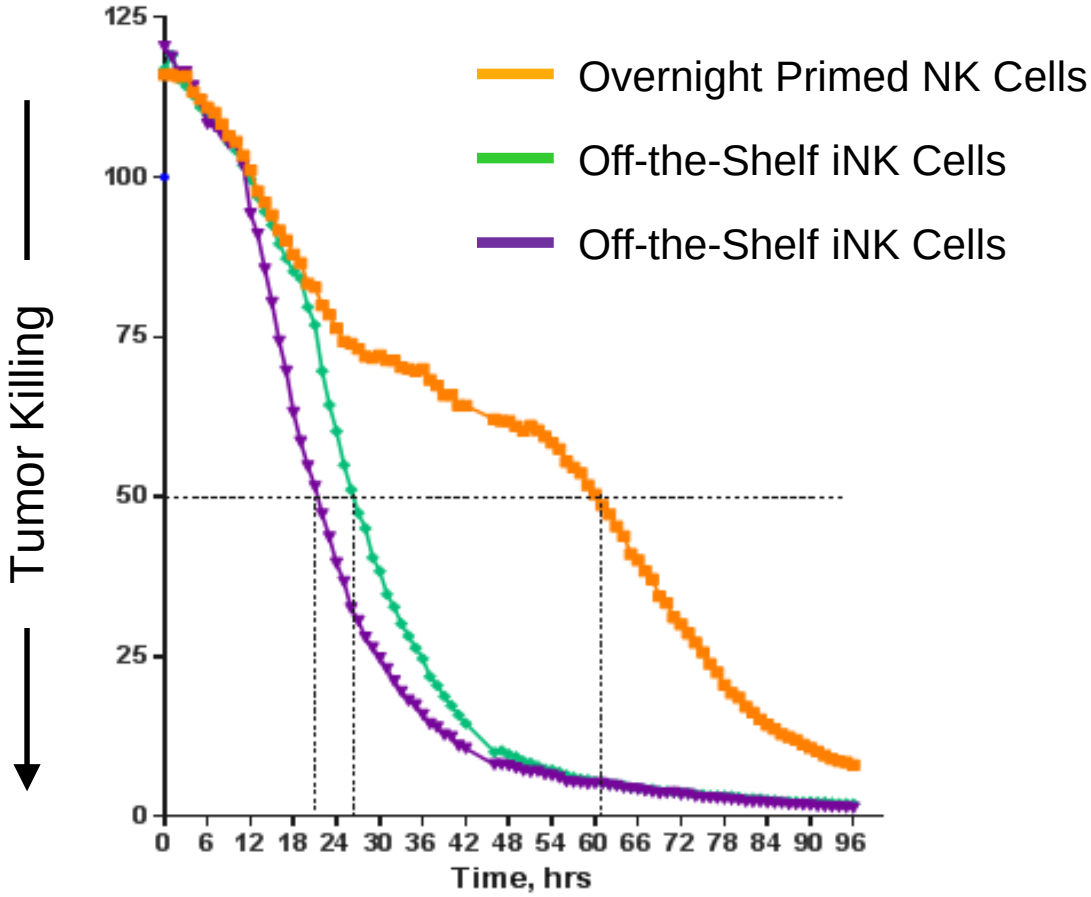
FT500 Cell Potency

iPSC-derived vs. Peripheral Blood NK Cells

SKOV3 (Ovarian Cancer) Killing Assay



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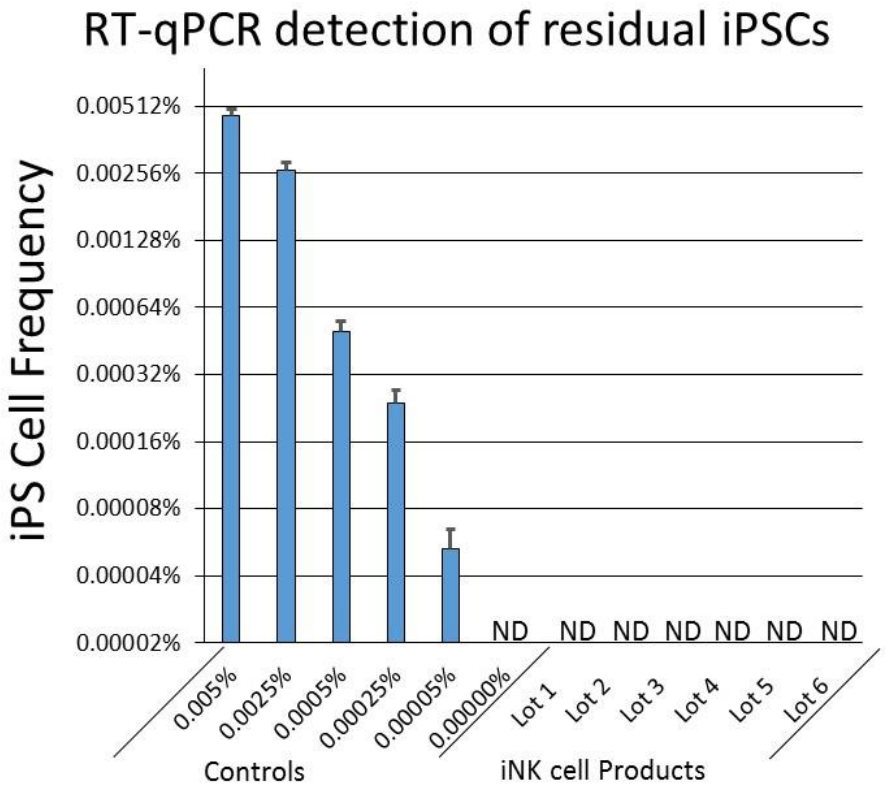
FT500 Purity

No Residual iPSCs in FT500 Cell Product



- Determination made analyzing a set of master pluripotency genes (NANOG, OCT4, SOX2, REX1) highly expressed in iPSCs but not in background of NK cells

No iPSCs detected at the resolution of 1 in 2 million during multiple iNK cell manufacturing runs

Fate THERAPEUTICS

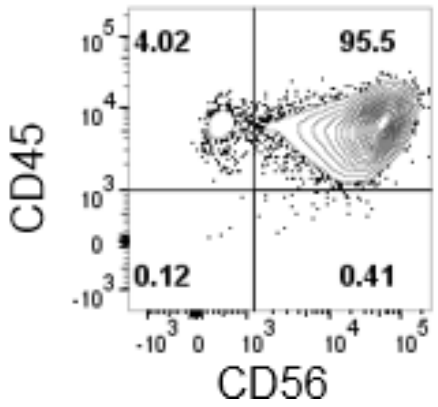
FT500 Production

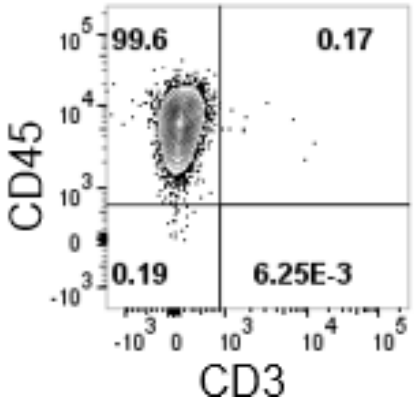
Technology Transfer to MCT cGMP Facility

1x10^6 iPSCs delivers 1x10^{12} NK cells during 44 day manufacturing process

Molecular and Cellular Therapeutics

33,000 sf, free-standing, state-of-the-art GMP facility





FT500

1x10^9 Cryopreserved Cells

Fate THERAPEUTICS

FT500 in Combination with Checkpoint Inhibitors

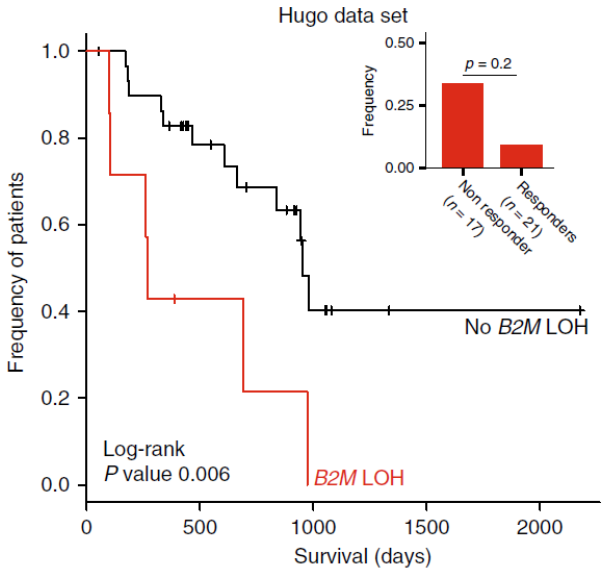
Synergy with T Cells to Infiltrate and Destroy 3D Tumor Mass

nature COMMUNICATIONS

Resistance to checkpoint blockade therapy through inactivation of antigen presentation

Moshe Sade-Feldman[1,2], Yunxin J. Jiao[2,3], Jonathan H. Chen[2,4], Michael S. Rooney[2], Michal Barzily-Rokni[1], Jean-Pierre Eliane[4], Stacey L. Bjorgaard[1,2], Marc R. Hammond[1], Hans Vitzthum[1], Shauna M. Blackmon[1], Dennie T. Frederick[1], Mehlika Hazar-Rethinam[1], Brandon A. Nadres[1], Emily E. Van Seventer[1], Sachet A. Shukla[2,5], Keren Yizhak[2], John P. Ray[2], Daniel Rosebrock[2], Dimitri Livitz[2], Viktor Adalsteinsson[2], Gad Getz[2,4], Lyn M. Duncan[4], Bo Li[6], Ryan B. Corcoran[1], Donald P. Lawrence[1], Anat Stemmer-Rachamimov[4], Genevieve M. Boland[7], Dan A. Landau[2,8,9], Keith T. Flaherty[1], Ryan J. Sullivan[1] & Nir Hacohen[1,2]

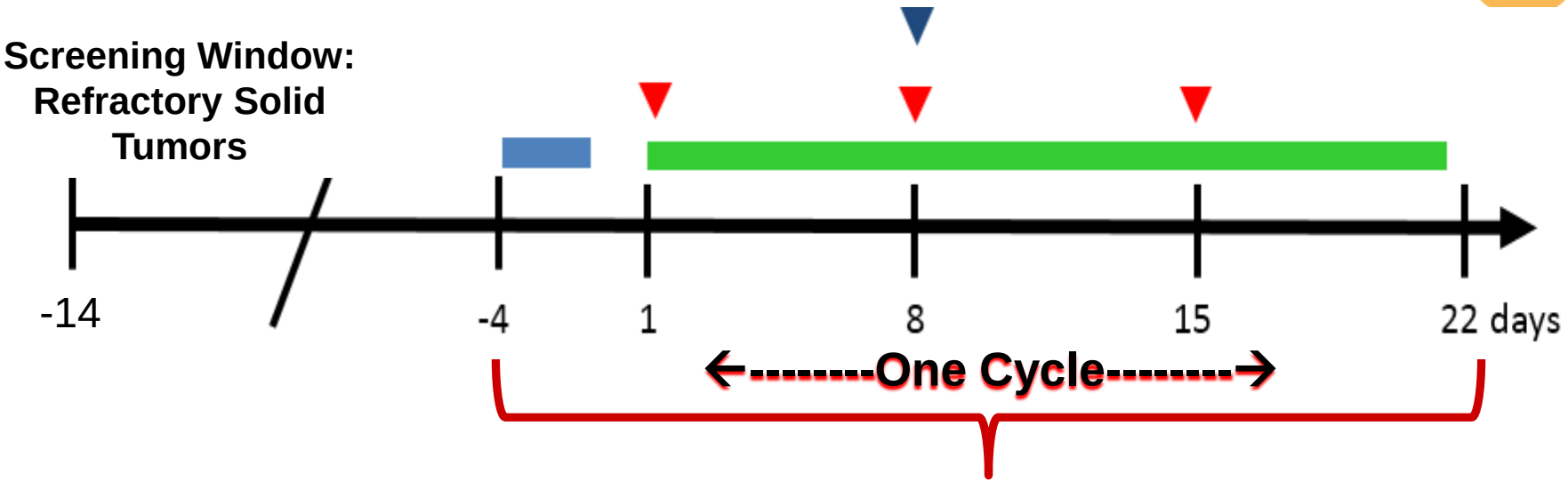
Treatment with immune checkpoint blockade (CPB) therapies often leads to prolonged responses in patients with metastatic melanoma, but the common mechanisms of primary and acquired resistance to these agents remain incompletely characterized and have yet to be validated in large cohorts. By analyzing longitudinal tumor biopsies from 17 metastatic melanoma patients treated with CPB therapies, we observed point mutations, deletions or loss of heterozygosity (LOH) in beta-2-microglobulin (*B2M*), an essential component of MHC class I antigen presentation, in 29.4% of patients with progressing disease. In two independent cohorts of melanoma patients treated with anti-CTLA4 and anti-PD1, respectively, we find that *B2M* LOH is enriched threefold in non-responders (~30%) compared to responders (~10%) and associated with poorer overall survival. Loss of both copies of *B2M* is found only in non-responders. *B2M* loss is likely a common mechanism of resistance to therapies targeting CTLA4 or PD1.



Fate THERAPEUTICS

FT500 Proposed FIH Study

Multiple Cycles of FT500 + Checkpoint Inhibitor Dosed Weekly



Conditioning Cyclophosphamide:300 mg/m^2 intravenous (IV) on Day-4 (or Day -3)
Fludarabine 25mg/m^2 IV on Days -4, -3, (or Day -3 and -2)

IL2 (2x/week) IL-2: 3 million units SC twice weekly from Day1 thru Day21

FT500 3 doses at 1 week intervals*

Checkpoint Inhibitor Given per SOC dose and frequency until disease progression

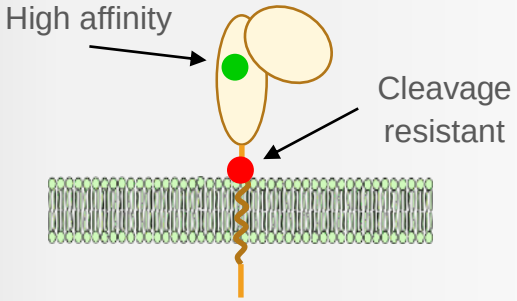
Fate THERAPEUTICS

FT516 Engineered hnCD16+ NK Cell Product Candidate

For Combination with Monoclonal Antibody Therapy

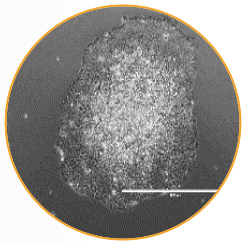
Engineered high-Affinity non-Cleavable CD16 Fc Receptor

Modified form of CD16a IgG antibody-binding receptor resists shedding upon activation

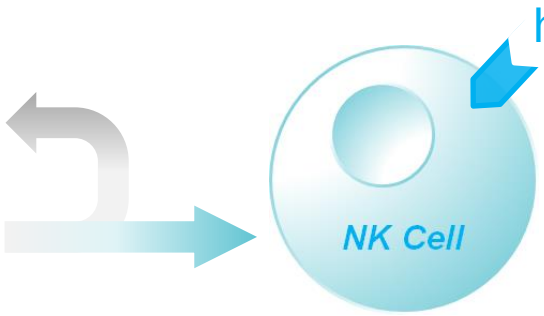


FDA-approved Monoclonal Antibodies

Rituxan Rituximab · Herceptin trastuzumab · ERBITUX CETUXIMAB · DARZALEX



Renewable Engineered Pluripotent Cell Line



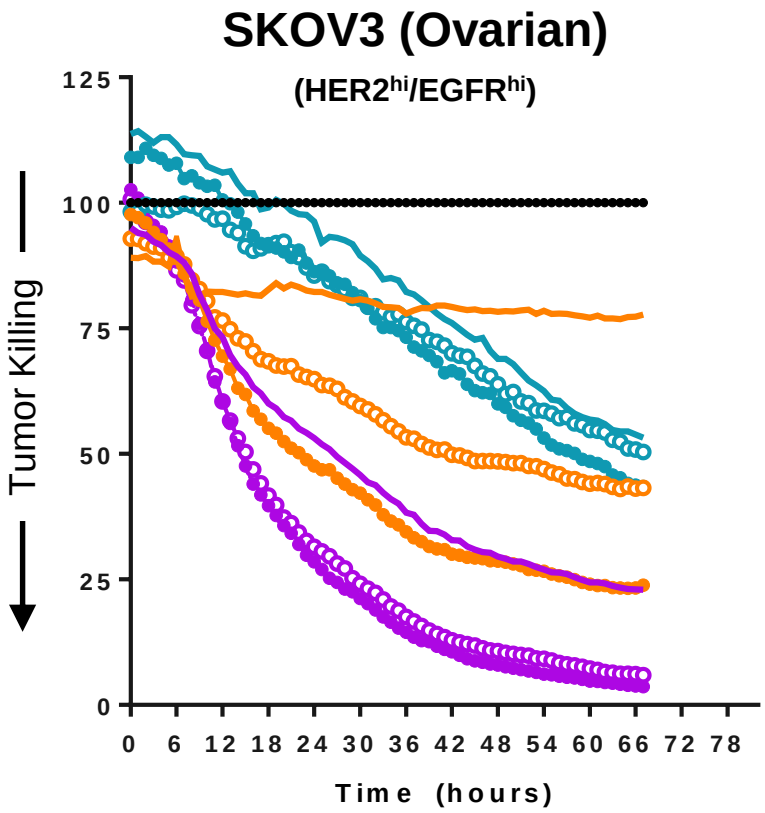
Bi- / Tri- Specific Engagers

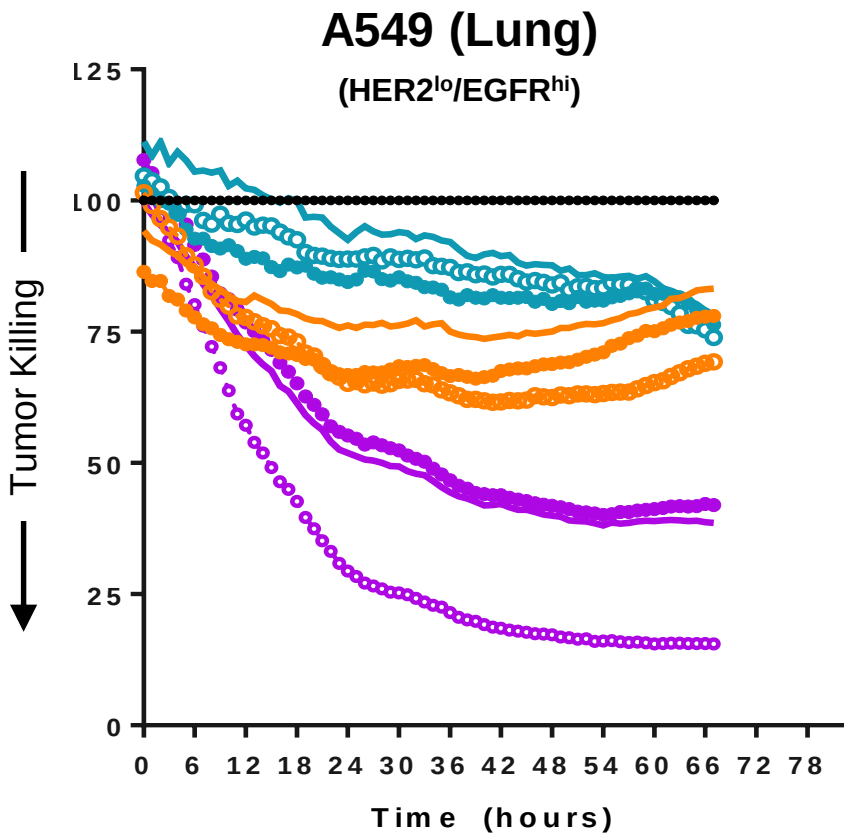
Engineered hnCD16 iNK Cells for ADCC

Fate THERAPEUTICS

FT516 Engineered hnCD16+ NK Cell Product Candidate

Augmented In Vitro ADCC for Solid Tumors





Target cells only
pbNK (n=3)
Cord blood NK (n=1)
hnCD16 iNK (n=3)

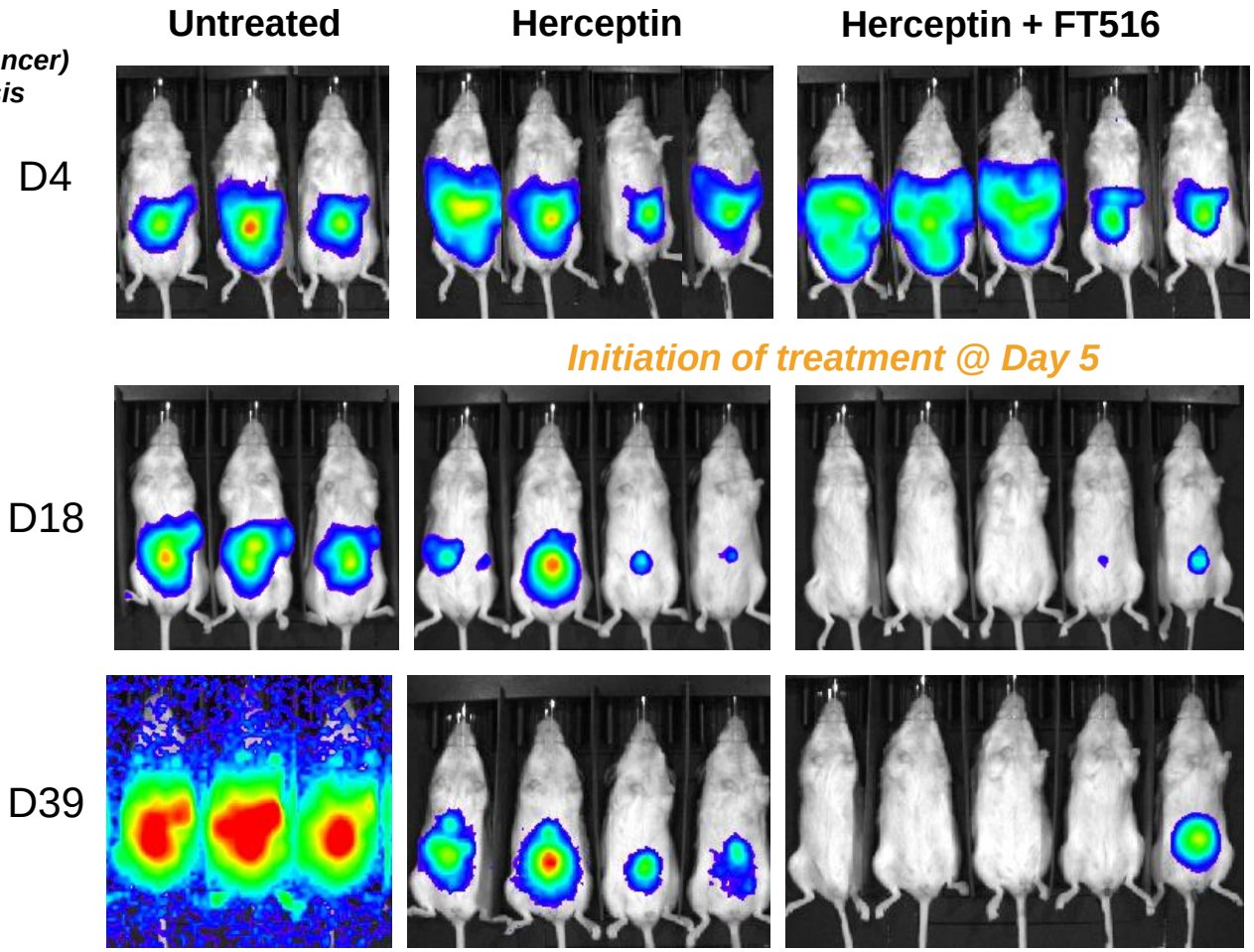
No antibody
Anti-Her2
Anti-EGFR

Fate THERAPEUTICS

FT516 Engineered hnCD16+ NK Cell Product Candidate

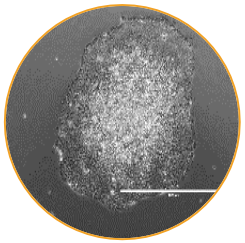
In Vivo POC

Luc-SKOV3 (Ovarian Cancer) Tumor Image Analysis



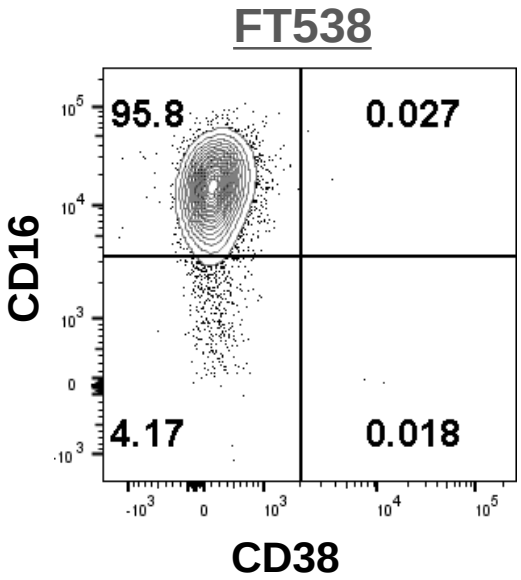
FT538 Engineered hnCD16+ / CD38-null NK Cell Product

For Combination with Darzalex for Multiple Myeloma



Renewable Engineered Master Pluripotent Cell Line

- **hnCD16 insertion**
- **CD38 knock-out**
- **Single cell selected**
- **Master iPSC line generated**
- **NK differentiation**



- CD38 is expressed at high levels on myeloma cells
- As an IgG1 antibody, daratumumab is an ideal mediator of ADCC against CD38+ tumor cells
- NK cells, which are critical to ADCC-induced lysis of tumor cells, also express CD38
- Clinical studies have shown that peripheral blood NK cell counts are reduced rapidly following daratumumab administration and remain low over the course of treatment

Fate THERAPEUTICS

Off-the-Shelf T Cell Products

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

CellPress

New Cell Sources for T Cell Engineering and Adoptive Immunotherapy

Fate Therapeutics and Memorial Sloan Kettering Cancer Center Launch Partnership for Development of Off-the-Shelf T-Cell Immunotherapies

Unite Cellular Immunotherapy Expertise to Accelerate Clinical Translation of Off-the-Shelf Products Offering Broad Patient Access

Collaboration to Use Engineered Pluripotent Cell Lines to Renewably Generate T-Cell Product Candidates

Foundational Intellectual Property Covering Pluripotent Cell-derived Engineered T Cells Exclusively Licensed to Fate Therapeutics

Fate THERAPEUTICS

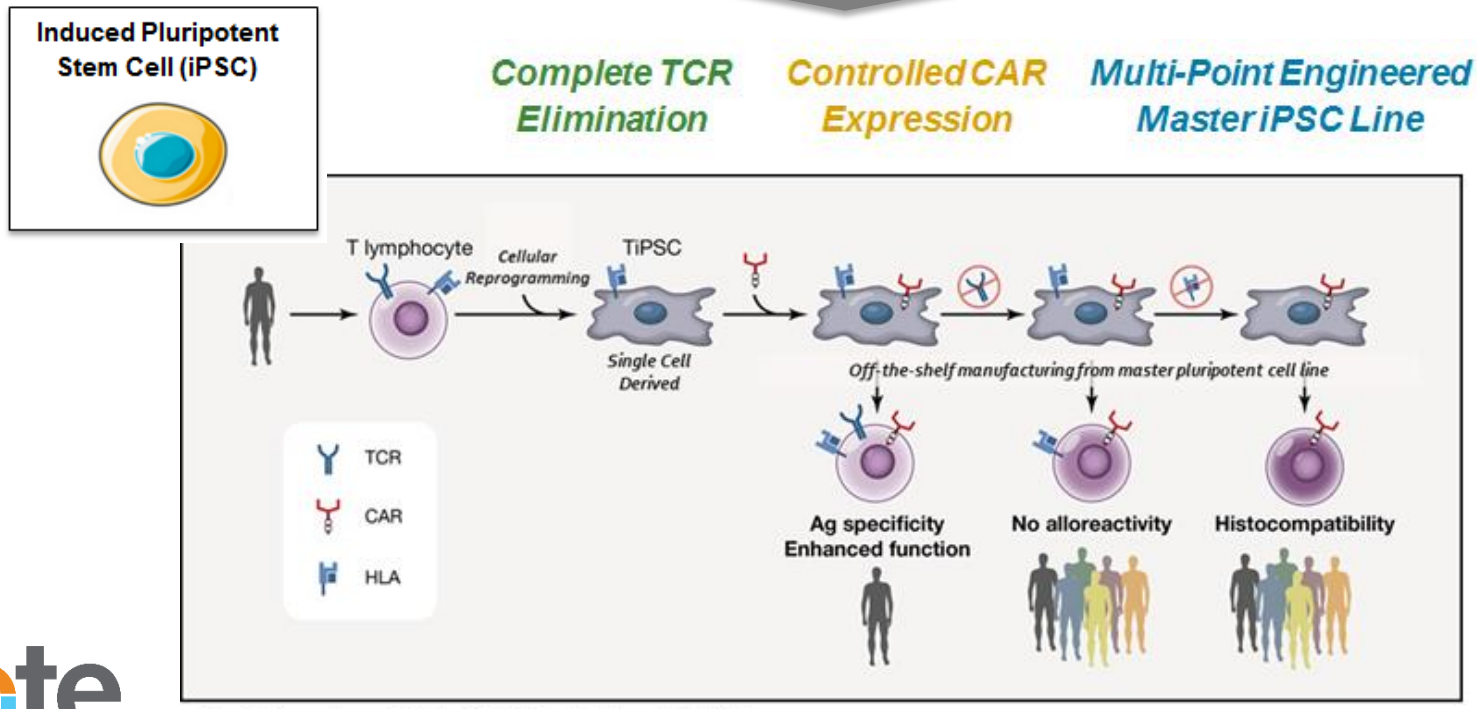
Off-the-Shelf T Cell Products

The Sadelain Roadmap

"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."

From Autologous to Off-the-Shelf T Cell Therapy

Induced Pluripotent Stem Cell (iPSC)

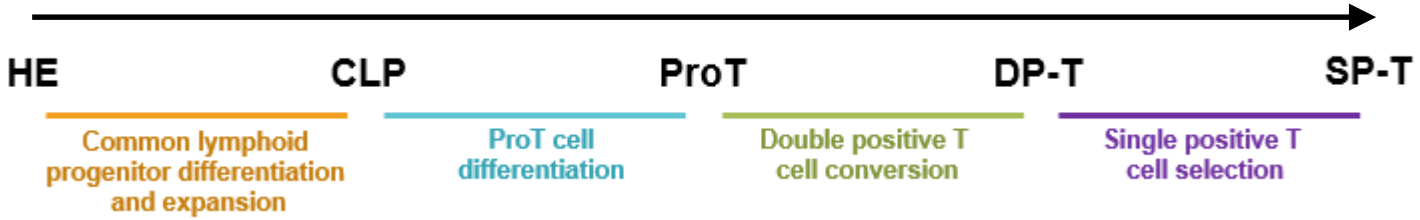


Adapted from: Themeli, Riviere & Sadelain, *Cell Stem Cells*, 2015

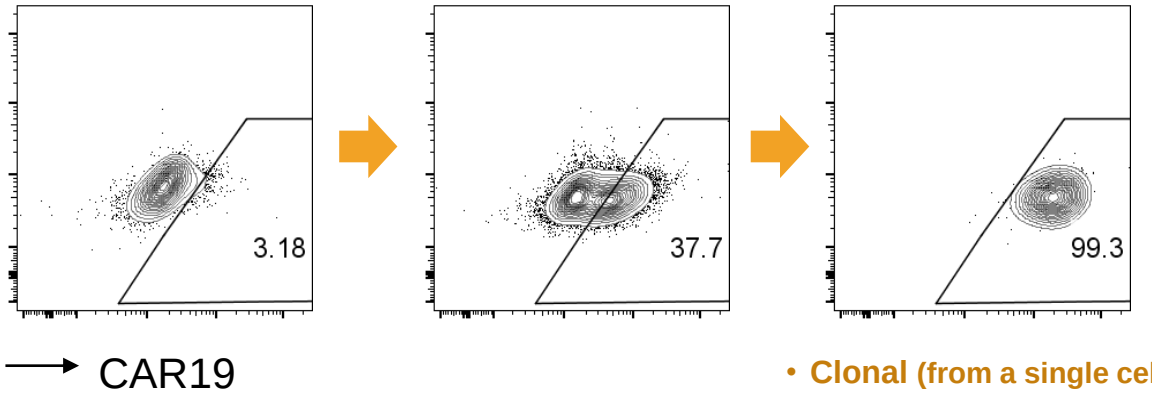
Fate THERAPEUTICS

Ex Vivo Generation of CAR19+ CD8αβ+ T Cells

Generation from Clonal Engineered Master iPSC Line

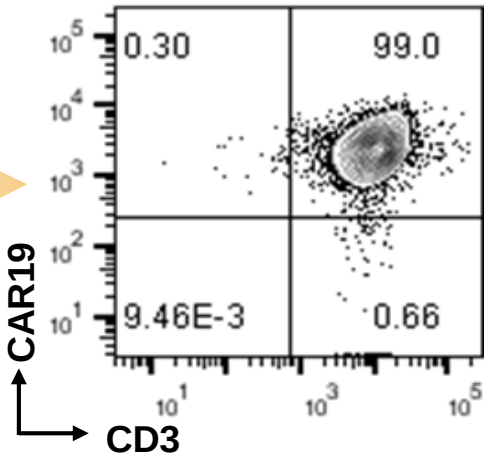


Clonal CAR19 Master hiPSC Line



- **Clonal (from a single cell)**
- **Pluripotent**
- **Renewable (unlimited source)**

CAR19-iT cells

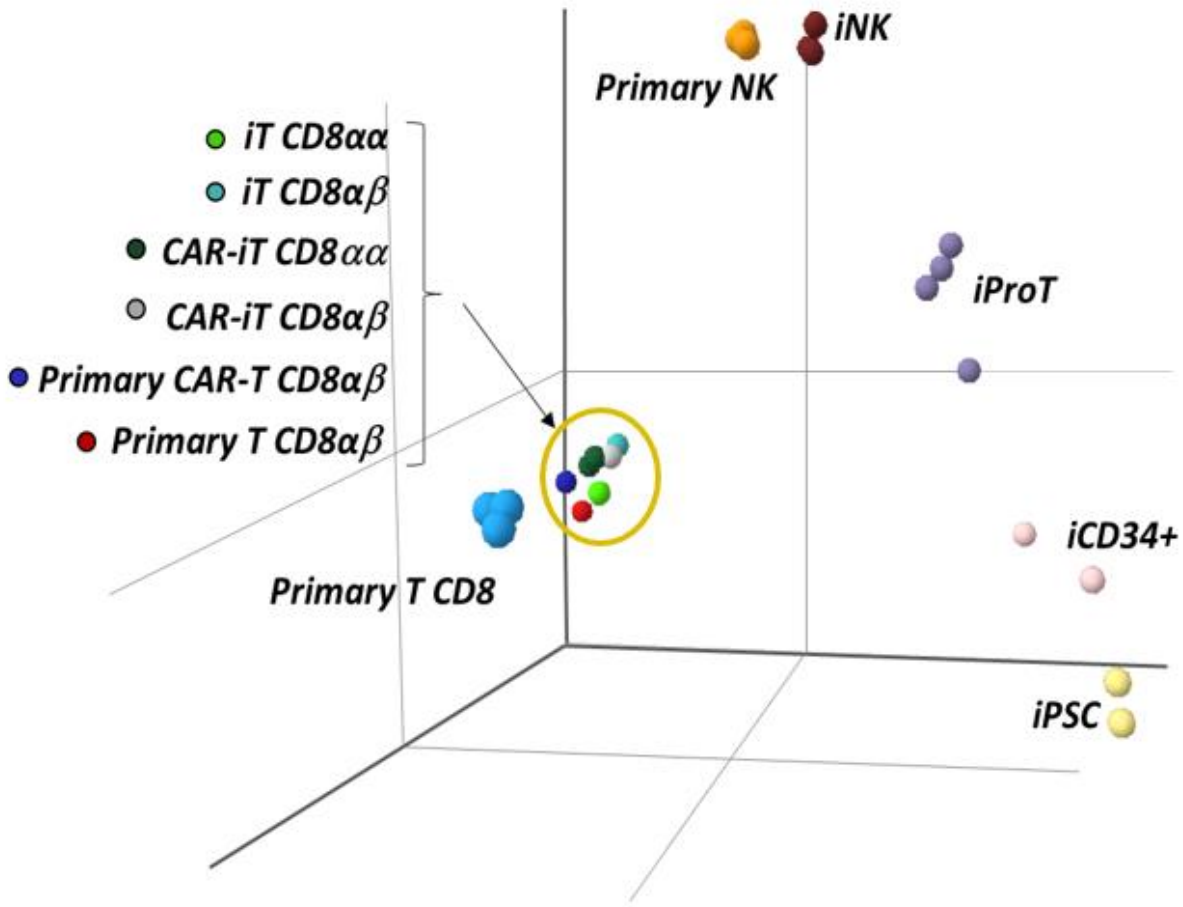


- **Homogenous population**
- **Reproducible**
- **Well-defined**

Fate THERAPEUTICS

iPSC-Derived CAR19+ CD8αβ+ T Cells

Global Gene Expression Comparison vs. Primary T Cells



Fate THERAPEUTICS

FT819 CAR19 TCR-null T Cell Product Candidate

Generated from Clonal Engineered Master iPSC Line

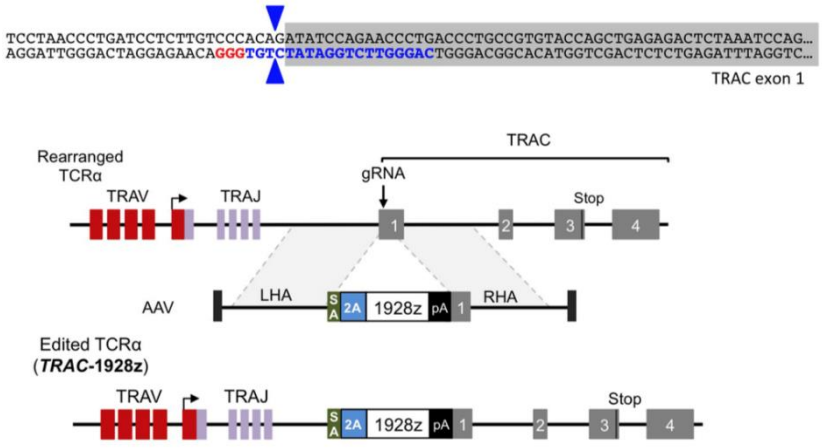
LETTER

doi:10.1038/nature21405

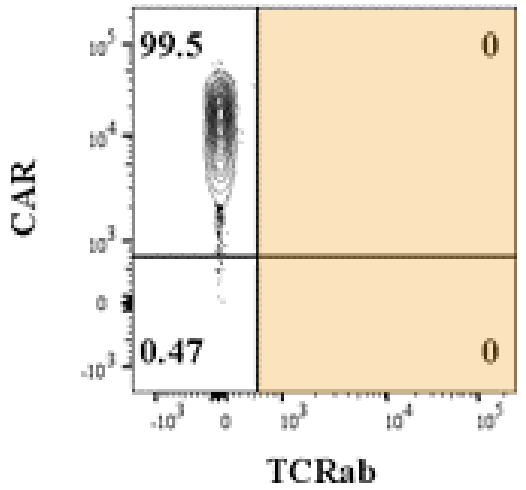
Targeting a CAR to the *TRAC* locus with CRISPR/Cas9 enhances tumour rejection

Justin Eyquem[1]*, Jorge Mansilla-Soto[1]*, Theodoros Giavridis[1], Sjoukje J. C. van der Stegen[1], Mohamad Hamieh[1], Kristen M. Cunanan[2], Ashlesha Odak[1], Mithat Gönen[2] & Michel Sadelain[1]

TCR Disruption



TRAC-encoded CAR Expression

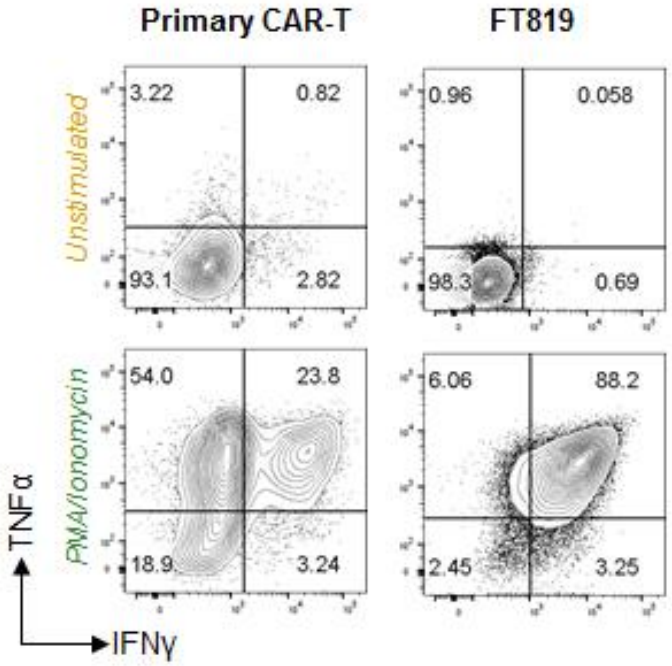


Complete Elimination of TCR

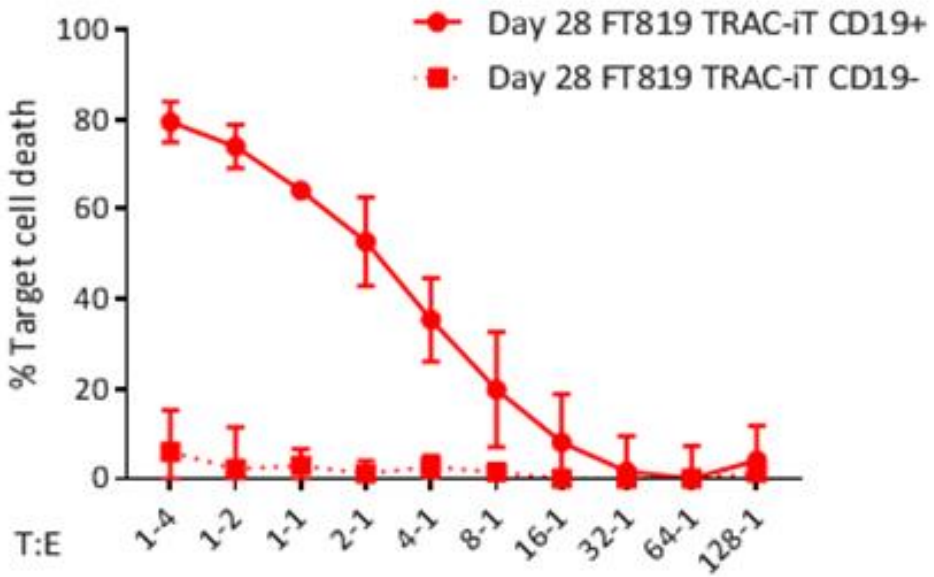
FT819 CAR19 TCR-null T Cell Product Candidate

In Vitro CAR19 Cytokine Production and Antigen Specificity

Cytokine Production



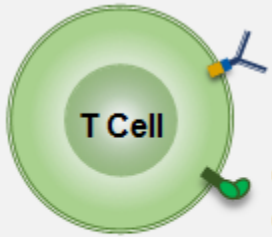
Antigen-specific Cytotoxicity



Fate THERAPEUTICS

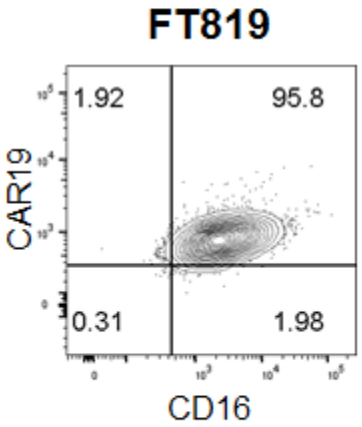
FT819 CAR19 TCR-null T Cell Product Candidate

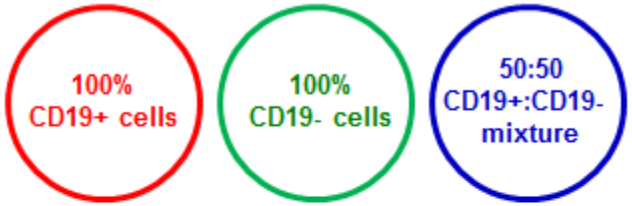
In Vitro CD16 ADCC Activity to Overcome Antigen Escape



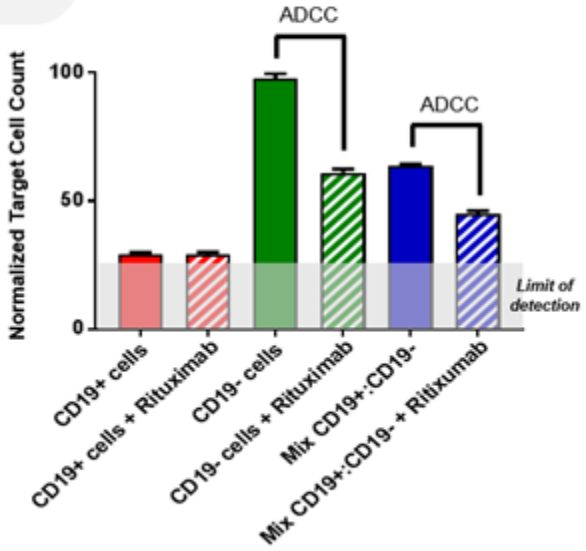
CAR19 – CD19 antigen targeting

CD16 – universal engager for antibody-dependent cellular cytotoxicity (ADCC)





FT819 + / - Rituximab



Immuno-Regulatory Programs

ProTmune™

Transforming the Curative Potential of Allogeneic HCT

A Next-Generation Hematopoietic Cell Graft to Prevent Acute Graft-versus-Host Disease

ProTmune™

Small molecule programmed mobilized peripheral blood graft

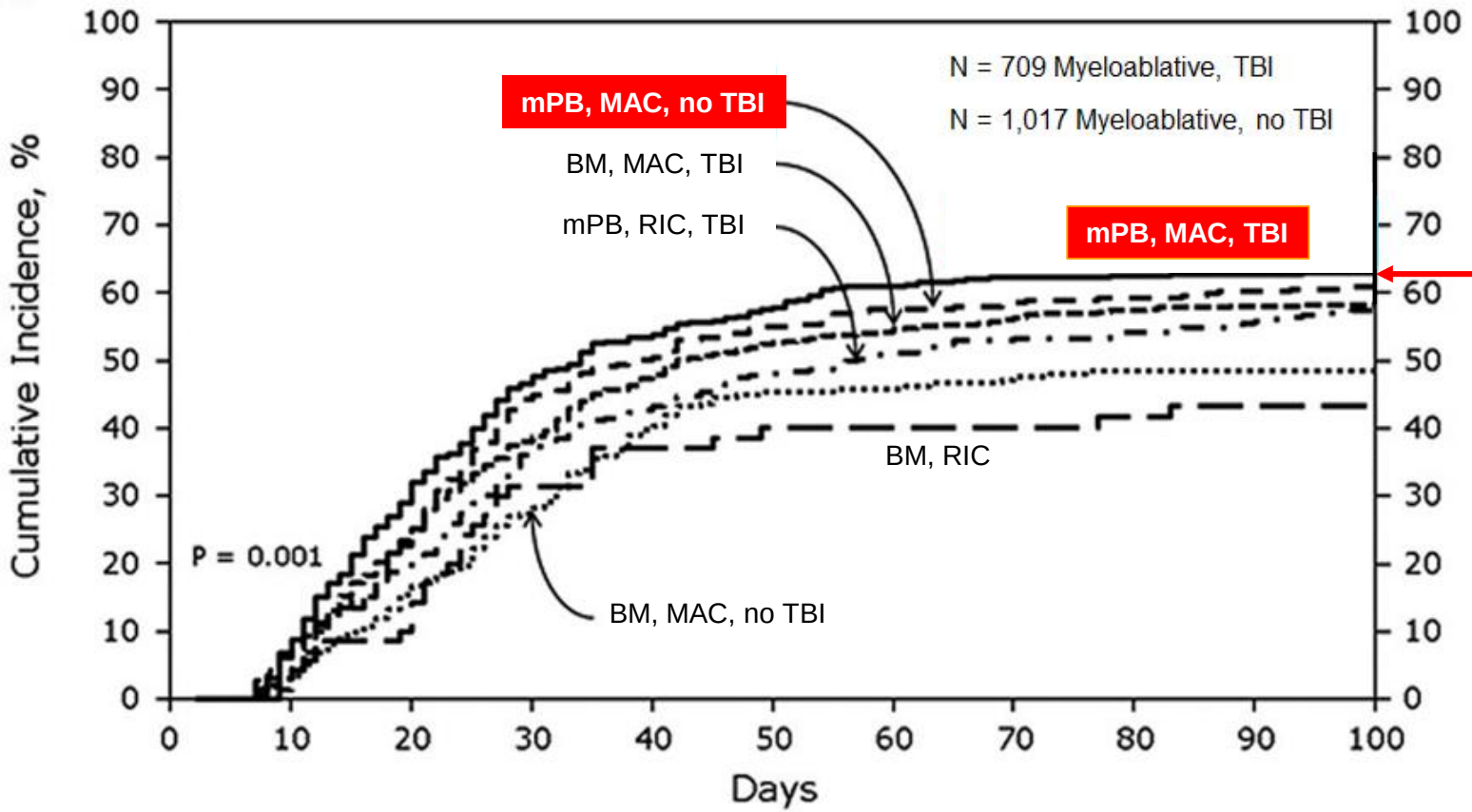
FT1050 + FT4145

- **Allogeneic HCT performed with curative intent**
  - Orphan hematologic malignancies (*e.g.*, AML, ALL, MDS)
  - Rare genetic disorders (*e.g.*, β-thalassemia, sickle cell)
- **Attractive market opportunity**
  - ~30,000 allogeneic HCT procedures performed annually
  - Conducted at concentrated number of centers of excellence
- **Significant unmet medical need**
  - Acute GvHD is leading cause of early morbidity and mortality
  - 40-80% of patients experience Grades 2-4 acute GvHD
- **No FDA approved therapies for prevention**
  - Immunosuppressive treatment can lead to infections / relapse

Fate THERAPEUTICS

Acute Graft-vs-Host Disease

High Incidence Rates during First 100 Days post-HCT



Jagasia et al., Blood, 2011

87 CIBMTR centers

~ 60% Historical Incidence Rate (Grade 2–4)

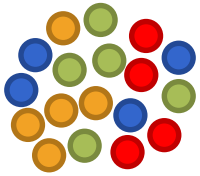
Fate THERAPEUTICS

Pathophysiology of Acute GvHD

Conditioned Patient

Tissue Damage

Cytokine Storm

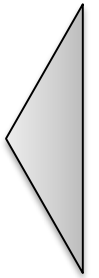


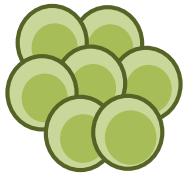
IL-6

IL-1β

TNF-α

IFN-γ





T Cells

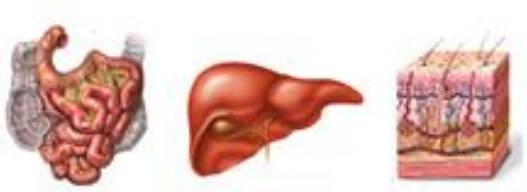
Donor

Donor Allo-reactive T-cell Activation

Host APCs

IL-12

Assault on Patient Tissue

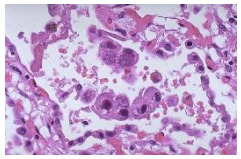


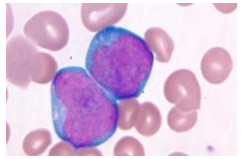
Acute GvHD (gut, liver, skin)

~40-80% D100 cumulative incidence

~10-20% early mortality

Immunosuppressive Agents





Severe Infections

~70% D100 cumulative incidence

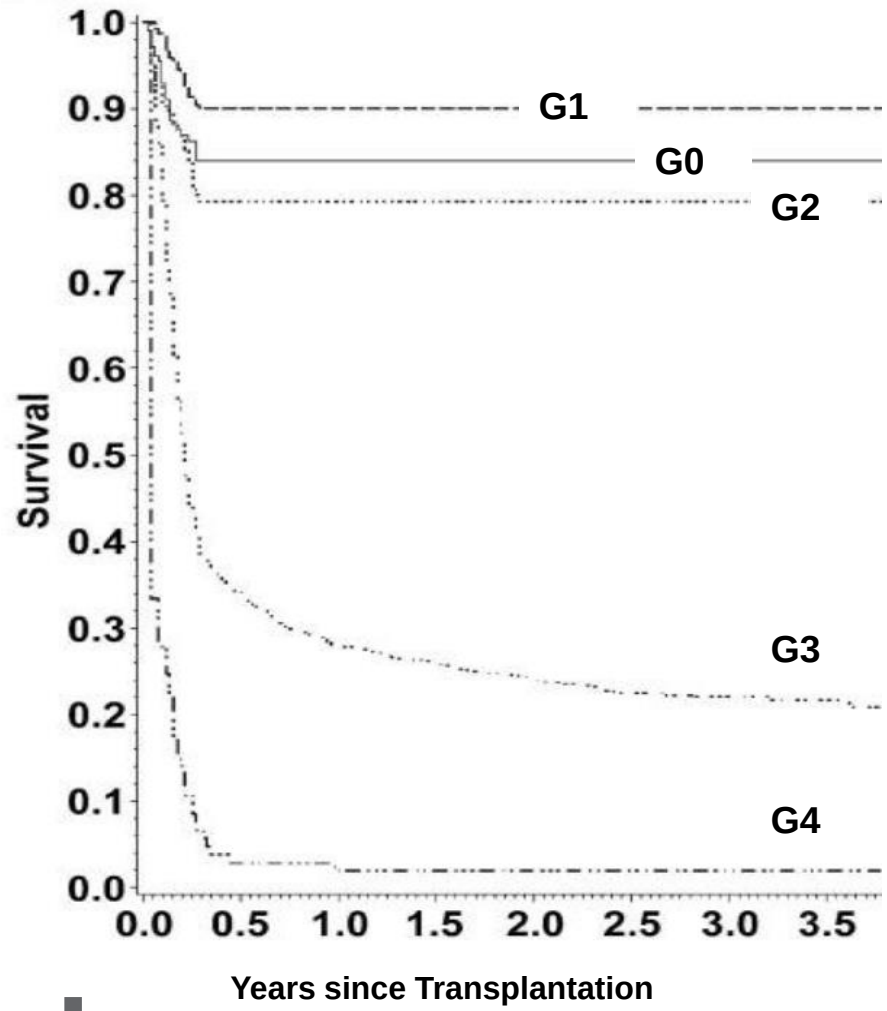
Relapse

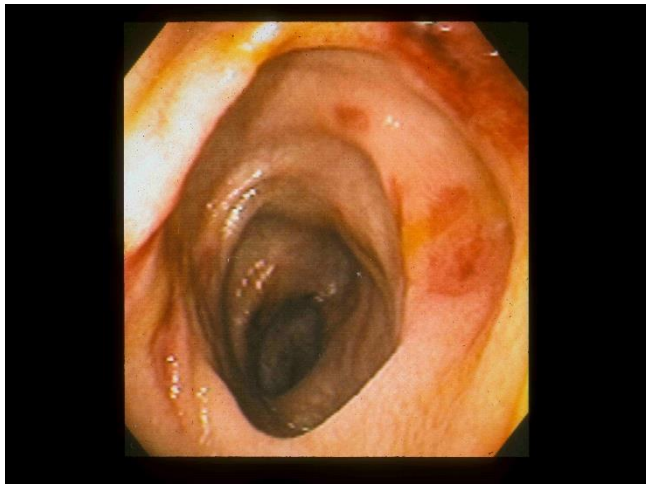
~35% 1YR cumulative incidence

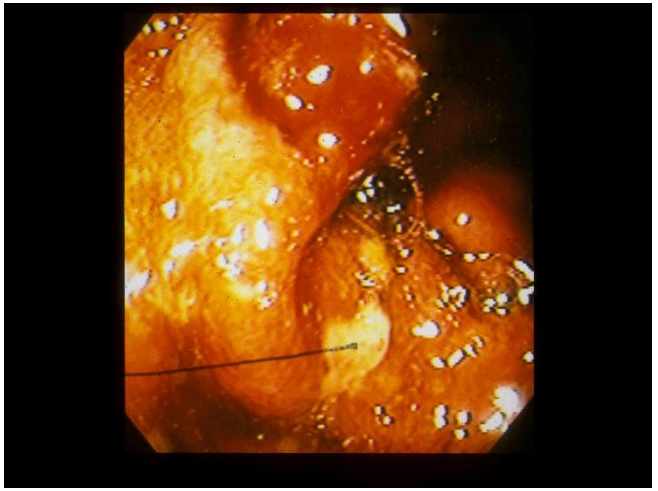
Fate THERAPEUTICS

Acute Graft-vs-Host Disease

Severe Acute GvHD Causes Mortality

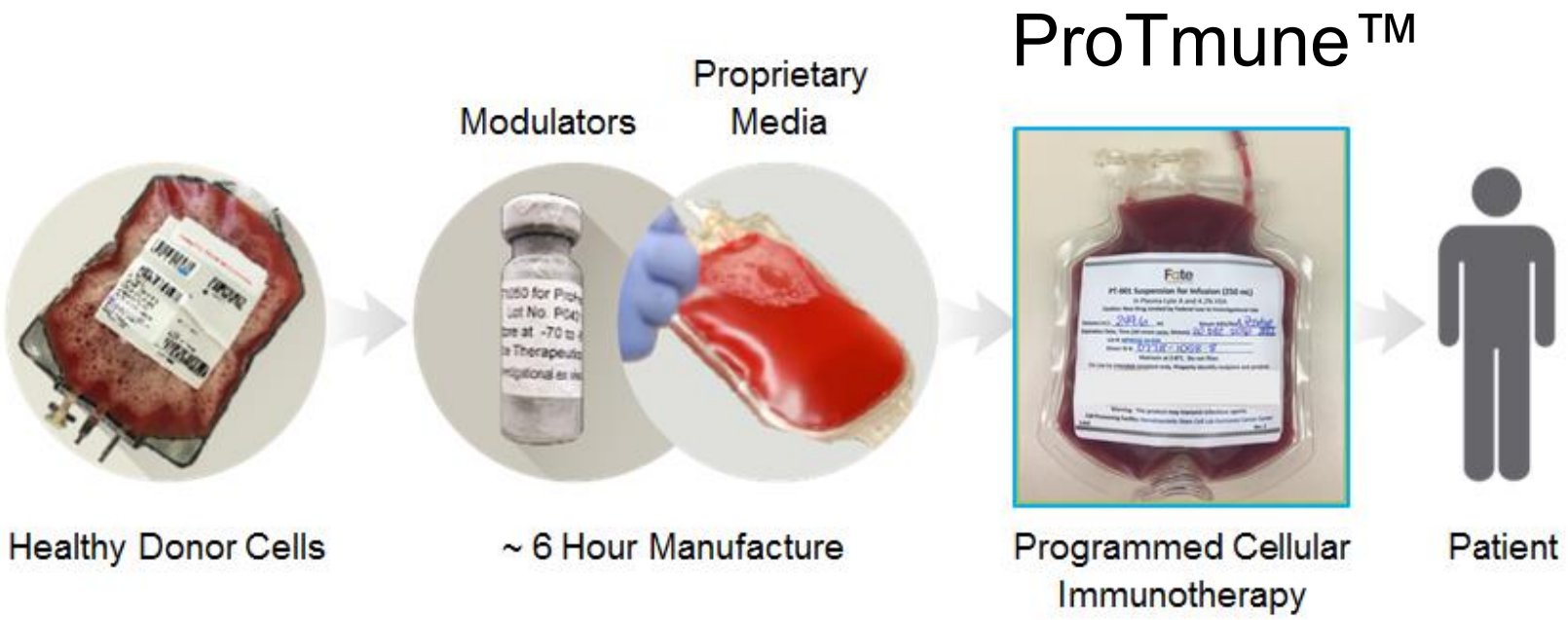






ProTmune™

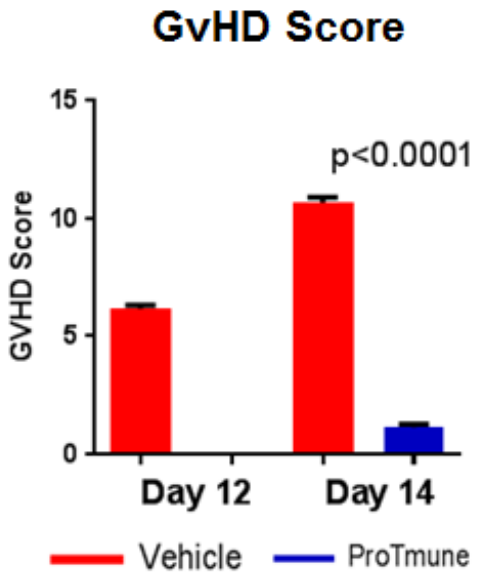
The Next-Generation Hematopoietic Cell Graft



ProTmune™

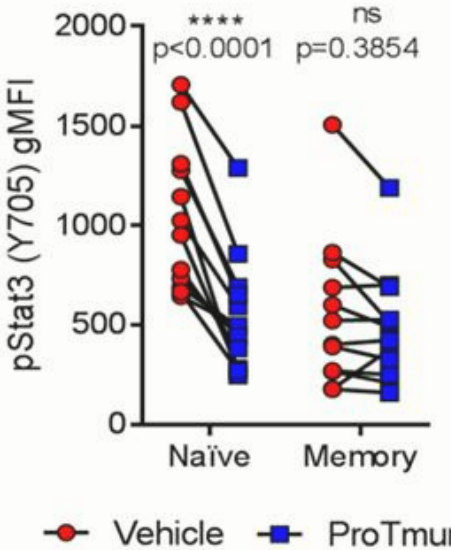
Ex Vivo Small Molecule Modulation of Donor T Cells

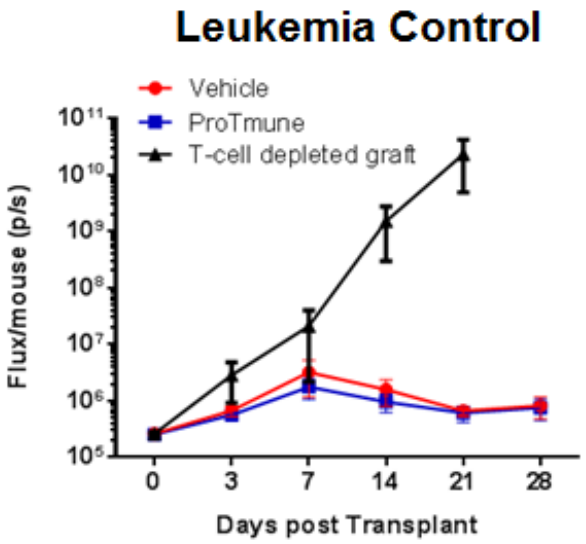
Striking the Balance between GvHD and GvL



Naïve T Cells

Attenuated T-Cell Response to Cytokine Signaling (IL-6)





Memory T Cells

Fate THERAPEUTICS

ProTmune™

PROTECT Phase 1/2 Study for Prevention of Acute GvHD

Phase 1 Stage: Day 28 Safety Assessment

Allogeneic HCT Setting

- Matched unrelated donor (MUD) mPB HCT with myeloablative conditioning
- Hematologic malignancies include ALL, AML & MDS
- Standard-of-care GvHD prophylactic (Methotrexate / Tacrolimus)
- Seven subjects received ProTmune

Safety Criteria

- Day 28 Engraftment without Graft Failure
- Day 28 Survival

Day 28 Safety Assessment

- All subjects met the Day 28 safety objectives of neutrophil engraftment and survival
- All subjects reached Day 28 without any events of graft failure or SAEs related to ProTmune
- DMC unanimously recommended advancement into Phase 2 efficacy stage

Fate THERAPEUTICS

ProTmune™

PROTECT Phase 1 Day 100 Efficacy Data

Subject	Disease	Age / Sex	MAC Regimen	Acute GvHD (CIBMTR) #	Organs Involved	Duration (d) Max Grade	Steroid Responsive	Leukemia Free	Overall Survival
1	MDS	66 / F	FluBu	None	---	---	---	Yes	Yes
2	AML	56 / F	BuCy	None	---	---	---	Yes	Yes
3	AML	66 / F	FluMel	Grade 2	Skin	7	Yes	Yes	Yes
4	ALL	34 / F	CyTBI	None	---	---	---	Yes	Yes
5	ALL	48 / M	CyTBI	Grade 2	Skin	8	Yes	Yes	Yes
6	ALL	56 / M	FluMel	Grade 3	Skin / Gut	5	Yes	Yes	Yes
7	AML	69 / F	FluMel	None	---	---	---	Yes	Yes

 # maximum grade GvHD

Fate THERAPEUTICS

ProTmune™

PROTECT Phase 1/2 Study for Prevention of Acute GvHD

Phase 2 Stage: *60 Subjects, Double-blinded, Randomized, Controlled*

Currently Enrolling at 14 U.S. Centers

Fast Track Designation

Randomized, Controlled, Blinded

ProTmune Cell Graft (n=30)

Standard-of-care mPB Cell Graft (n=30)

Primary Endpoint

Day 100 Cumulative Incidence of Grades 2-4 Acute GvHD

Exploratory Endpoints

Cancer Relapse; Severe Infections; Event-free Survival

Fate THERAPEUTICS

ProTmune™

Next-Generation Graft to Prevent Acute GvHD

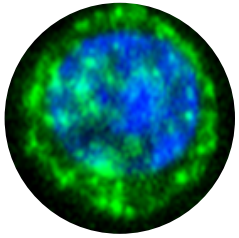
- **Preventive approach to address leading cause of early morbidity and mortality**
  - 40 to 80% of patients undergoing allogeneic HCT experience acute GvHD
  - Death directly attributable to acute GvHD or its treatment occurs in 10 to 20% of patients
  - No approved preventive therapies in the U.S.
- **Highly-differentiated therapeutic paradigm**
  - Optimize biological properties of donor hematopoietic cells *ex vivo* using small molecules
  - On-site manufacture integrates into current clinical practice
  - Avoids costly and time-consuming measures (e.g., genetic engineering, cell expansion, cell separation)
- **Strong commercial positioning targeting significant market opportunity**
  - Matched unrelated donor (MUD) for hematologic malignancies is predominant HCT setting
  - Composition of matter patents extending through 2032
  - Secured Fast Track in US and broad Orphan Drug Designations in US and EU

Fate THERAPEUTICS

ToleraCyte™

Tolerizing the Immune System for Autoimmune Diseases

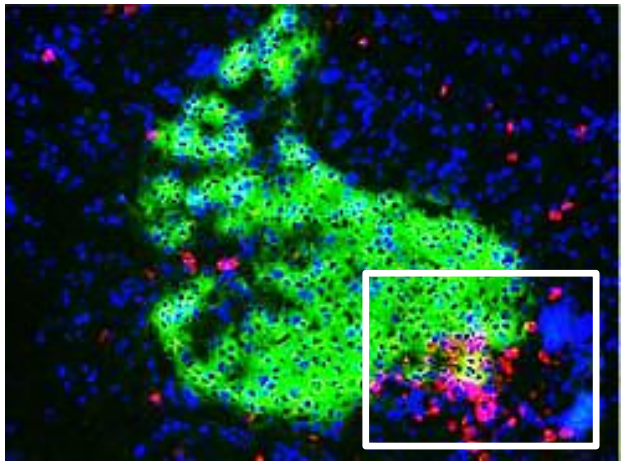
A First-in-Class Immunoregulatory CD34⁺ Cell Product Candidate to Induce Immune Tolerance



ToleraCyte™

Small molecule programmed CD34⁺ cells

- **Autoimmune disorders result from malfunction of the body's natural defense systems**
  - Adaptive immune system (e.g., autoreactive T cells) mistakenly recognizes healthy cells as foreign and attacks and destroys the body's own tissue
  - 80+ autoimmune disorders estimated to affect ~50M in U.S.
  - Most common disorders include rheumatoid arthritis, lupus, inflammatory bowel disease, multiple sclerosis and type 1 diabetes



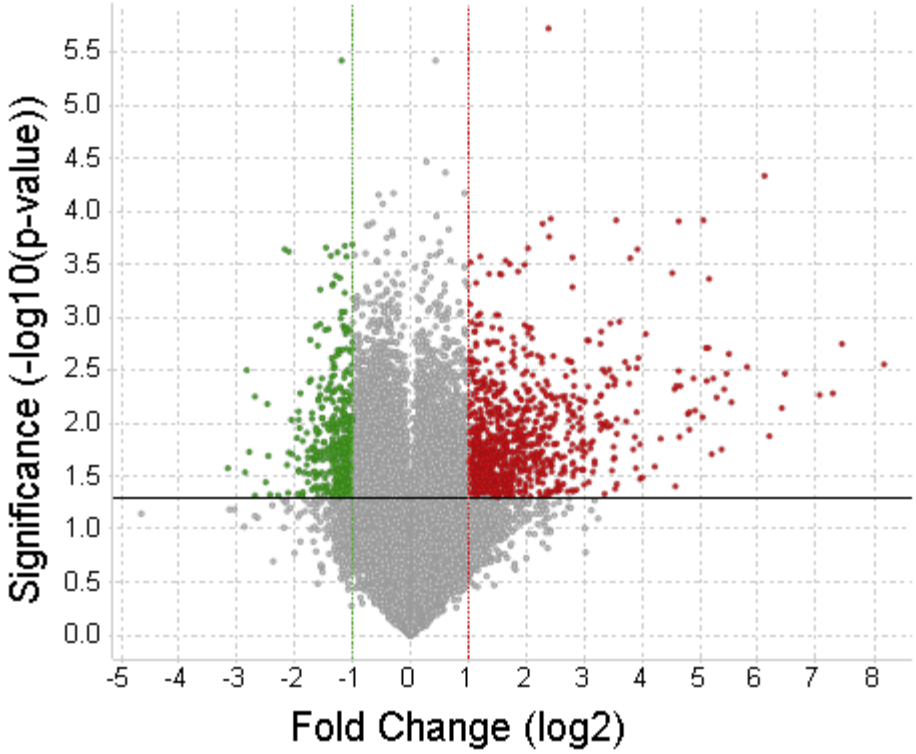
CD8+ T cells (red) attacking pancreatic beta cells (green)

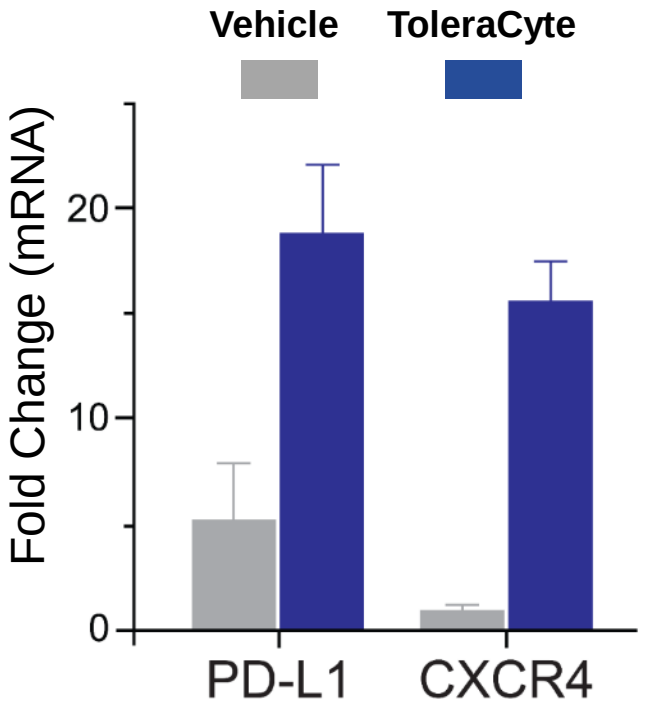
Fate THERAPEUTICS

ToleraCyte™

Small Molecule Programmed CD34+ Cell Product

Ex vivo modulation of CD34^{+} cells with two small molecules induces the expression of genes involved in cell migration and immune regulation





Fate THERAPEUTICS

Immuno-Regulatory CD34+ Cell Therapy

Collaborator Established Proof-of-Principle in Type 1 Diabetes

Paolo Fiorina, MD, PhD

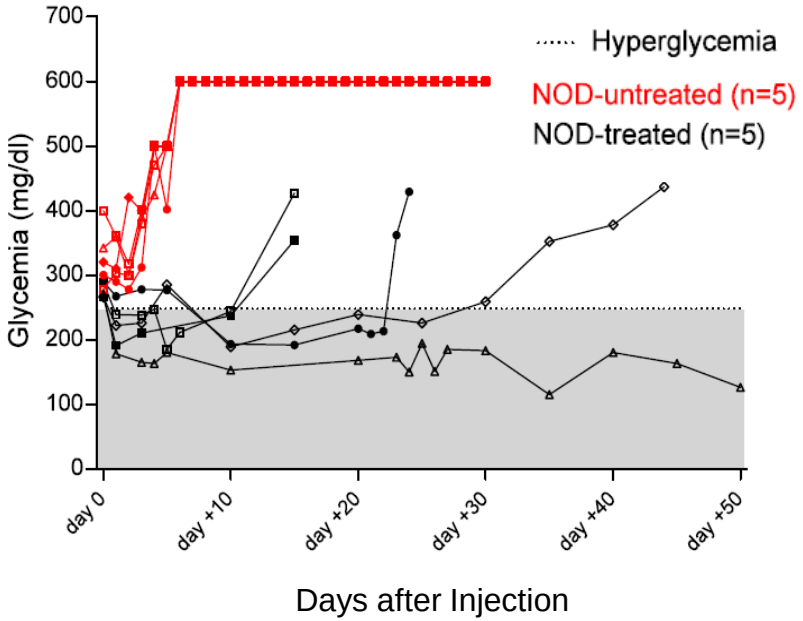
Assistant Professor of Pediatrics, Harvard Medical School

Boston Children's Hospital
Until every child is well™

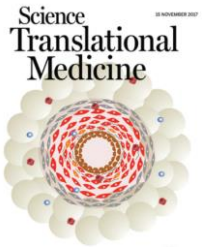
- Extensive investigation into T-cell destruction of pancreatic beta cells
- Engineered PD-L1 hematopoietic cells to assess potential to exploit checkpoint axis
- Demonstrated that single administration of PD-L1+ cells revert hyperglycemia in preclinical model of T1D

Hyperglycemic Mice

Adoptive Transfer of PD-L1+ Hematopoietic Cells



Days after Injection



PD-L1 genetic overexpression or pharmacological restoration in hematopoietic stem and progenitor cells reverses autoimmune diabetes

BY MOUFIDA BEN NASR, SARA TEZZA, FRANCESCA D'ADDIO, CHIARA MAMELI, VERA USUELLI, ANNA MAESTRONI, DOMENICO CORRADI, SILVANA BELLETTI, LUCA ALBARELLO, GABRIELLA BECCHI, GIAN PAOLO FADINI, CHRISTIAN SCHUETZ, JAMES MARKMANN, CLIVE WASSERFALL, LEONARD ZON, GIAN VINCENZO ZUCCOTTI, PAOLO FIORINA

SCIENCE TRANSLATIONAL MEDICINE | 15 NOV 2017 |

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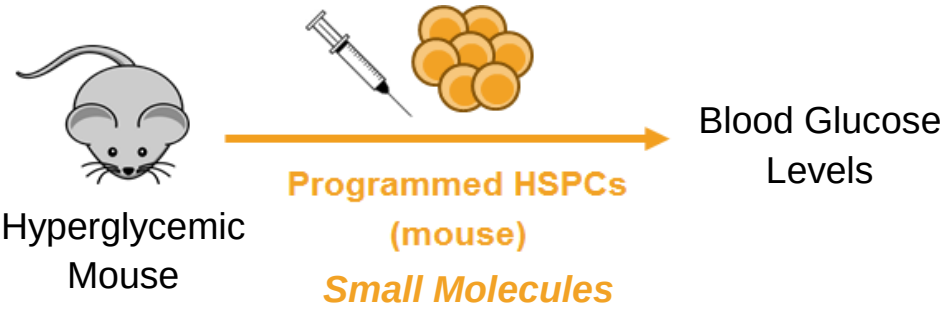
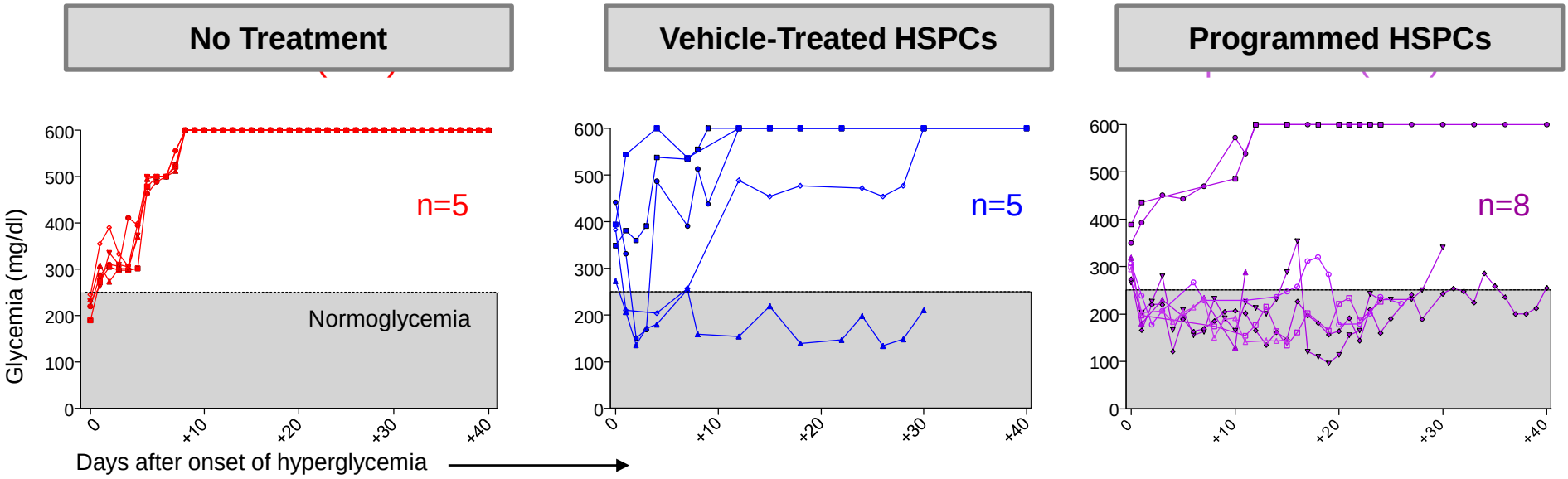
Immuno-Regulatory CD34+ Cell Therapy

Durable Disease Correction in T1D Mouse Model





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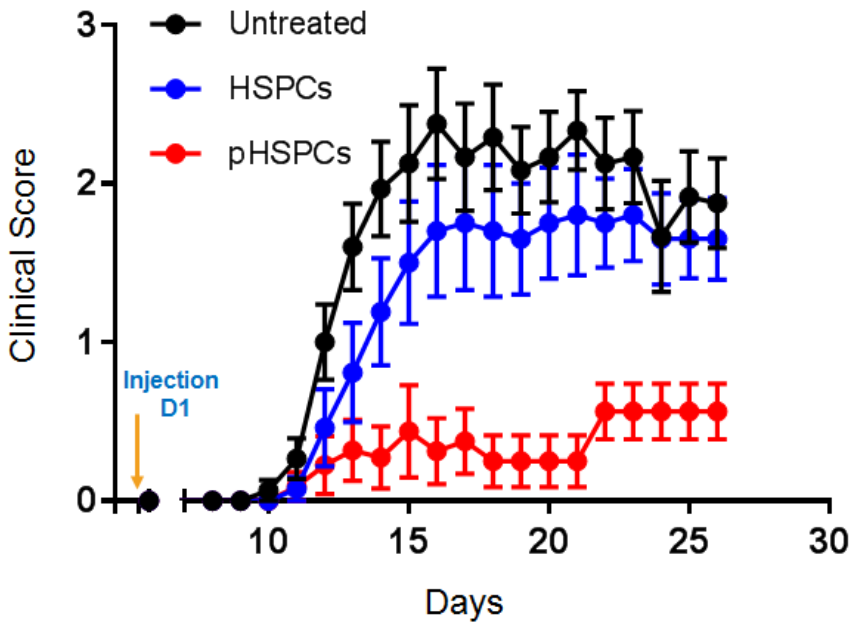
Boston Children's Hospital
Until every child is well™

Immuno-Regulatory CD34+ Cell Therapy

Disease Attenuation in EAE Mice (Multiple Sclerosis)

Programmed HSPCs attenuate loss of motor function in EAE Mice

Loss of Motor Function



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Fate THERAPEUTICS

Boston Children's Hospital
Until every child is well™

ToleraCyte™

Small Molecule Programmed CD34⁺ Cell Product for Autoimmunity

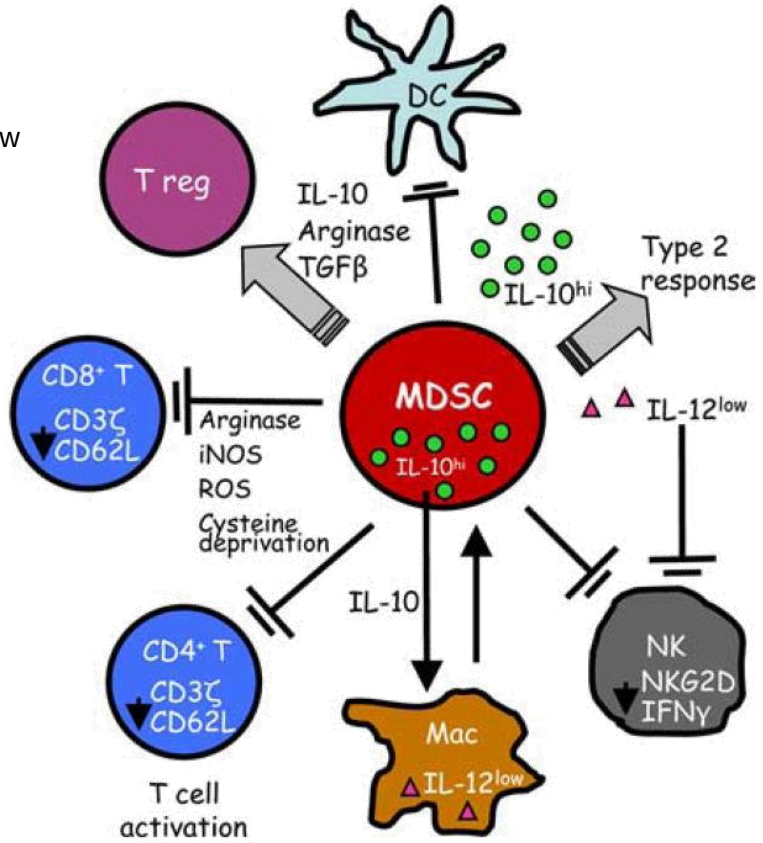
- **Builds on clinical precedent for CD34+ cell therapy**
  - Use of patient- and donor-sourced CD34+ cells has well-established safety record
- **Unique immuno-regulatory mechanism of action**
  - T-cell targeting approach through enhanced homing of programmed CD34⁺ cells to sites of inflammation
  - Robust suppression of T cells through immune checkpoint pathways (e.g., PD-L1, IDO1)
  - Induction of immune tolerance (T-cell anergy)
- **Durable disease correction demonstrated in multiple models of immune disorders**
  - Single administration attenuates disease in murine model of type 1 diabetes
  - Single administration attenuates disease in murine model of multiple sclerosis
- **Successful pre-IND meeting supports clinical investigation**
  - Defined clear path to first-in-human testing in adult patients with T1D
  - Scientific and clinical rationale for testing ToleraCyte in multiple immune indications

Fate THERAPEUTICS

Myeloid-Derived Suppressor Cells (MDSCs)

Immuno-Regulatory Cells With Unique Properties

- Defined as immature myeloid cells with potent suppressive activity
  - CD33+/CD11b+, CD14+/CD66+, HLA-DR^{low}
- Suppressive activity occurs through diverse mechanisms of action
  - ARG, iNOS, IL10, Gal9, CD73, TGFb
  - Inhibition of T, NK, DC cells
  - Activation of regulatory T and B cells
- Activity triggered by pro-inflammatory environment (antigen independent)
- Challenging to produce commercially-viable product using patient- or donor-sourced cells



Myeloid-Derived Suppressor Cells (MDSCs)

Advantages Relative to Other Immuno-Regulatory Cell Types

Cell Feature	Cell Type			
	T Regs	MSCs	HSCs	MDSCs
Multiple anti-inflammatory mechanisms	●	●	●	●
Able to induce long-term tolerance	●	●	●	●
Antigen agnostic activation	-	●	●	●
Homing to site of inflammation (systemic delivery)	●	-	●	●
Scalable manufacturing	-	●	-	-

Enabled using iPSC Product Platform

Fate THERAPEUTICS

FT300

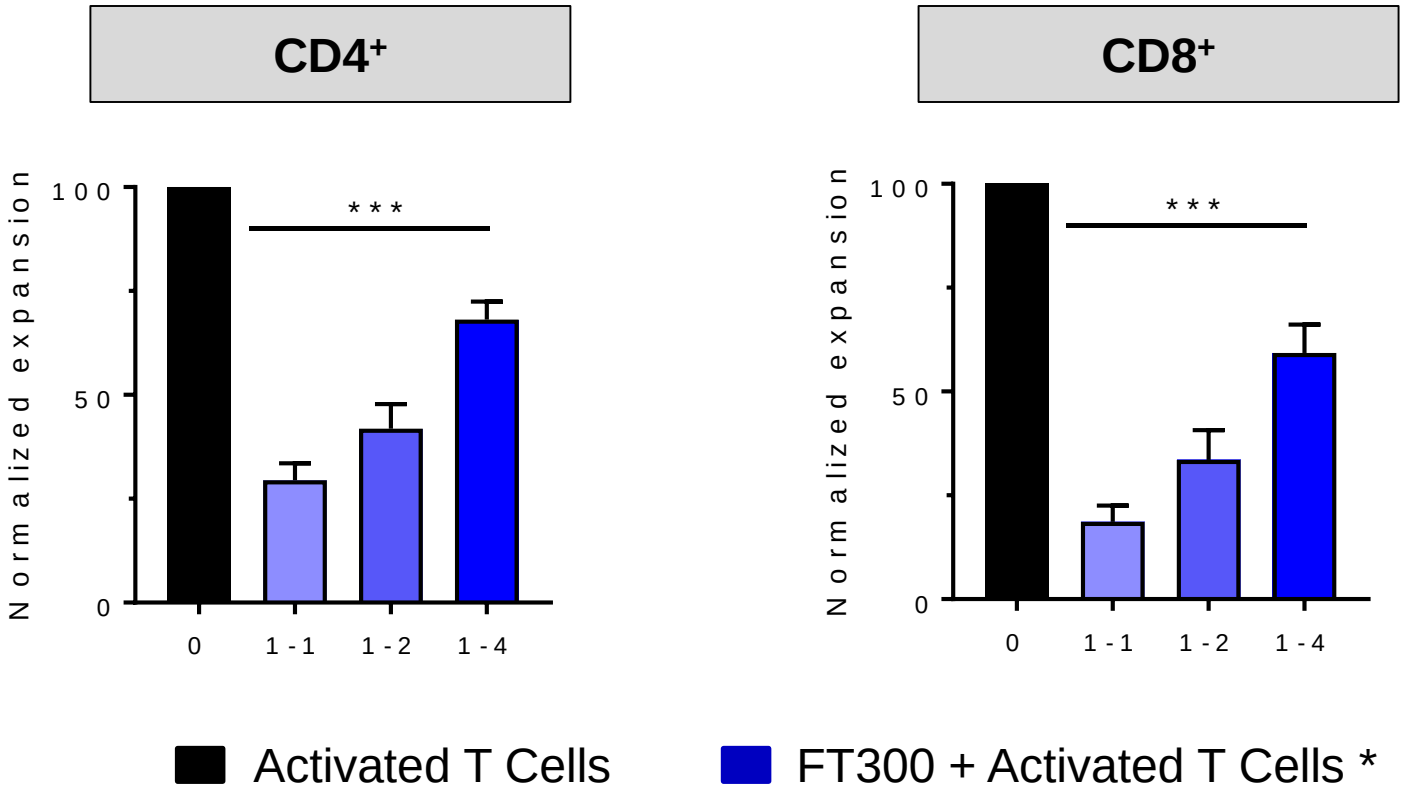
iPSC-derived MDSC Product

Feature	Benefit
Anti-inflammatory activity triggered by local inflammatory environment (antigen independent)	Efficacy across a variety of autoimmune diseases (antigen independent MOA)
MDSC's capable of homing to site of inflammation	Systemic "IV" delivery with efficacy in the local inflammatory environment
HLA matching not required for anti-inflammatory activity or cell persistence	Enables "off-the-shelf" product (one drug for all patients)
Scalable process for manufacturing drug product	Cost-effective therapy available for large patient populations; ability to repeat dose
Cryopreserved drug product	Centralized manufacturing; drug product available "on-demand" at site of care
Homogenous drug product	Predictable safety and efficacy profile with high-quality drug product

Fate THERAPEUTICS

FT300 *In Vitro* Activity

Potent Suppressor of T-Cell Proliferation Across HLA Barriers

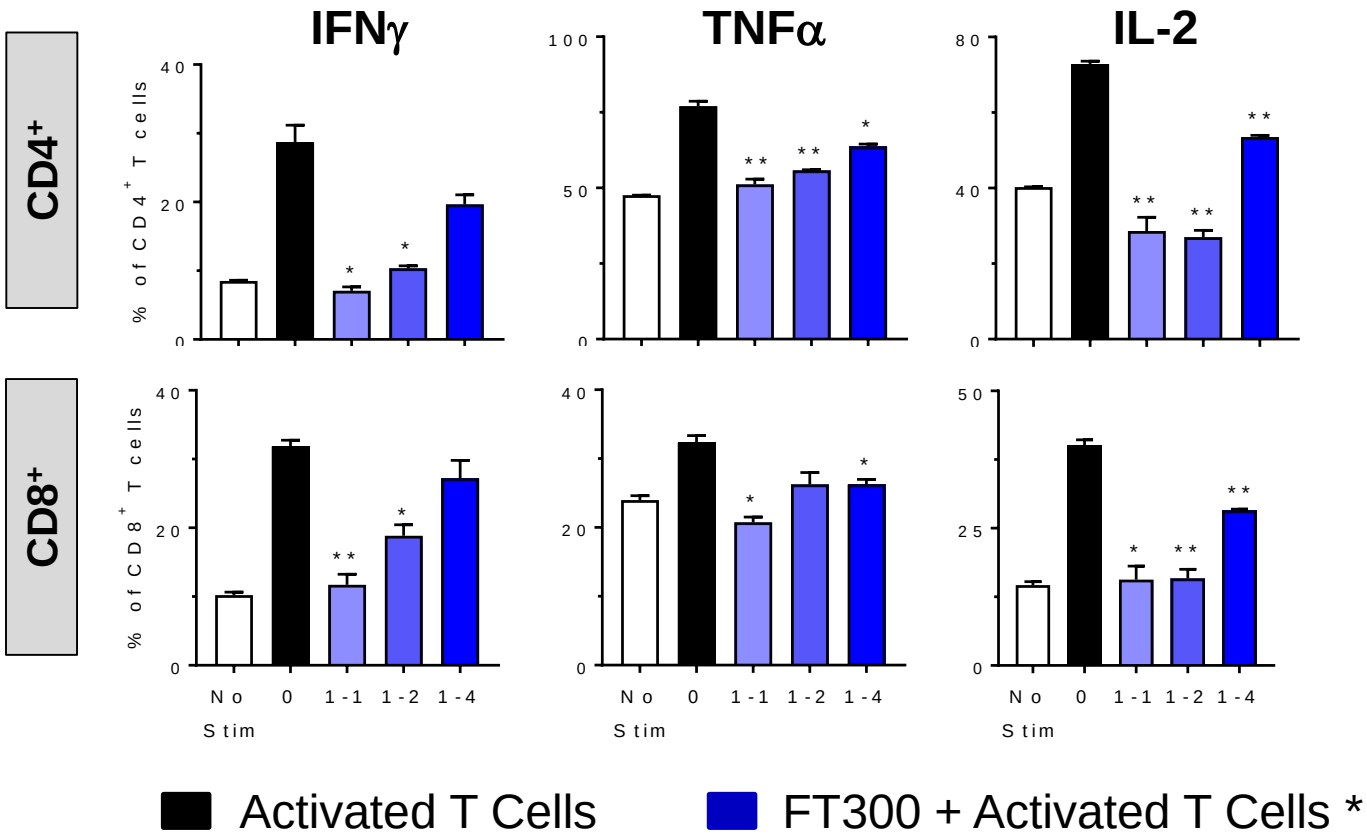


** Segmented by ratio of FT300 : Activated T Cells (5 independent donors for each ratio)*

Fate THERAPEUTICS

FT300 *In Vitro* Activity

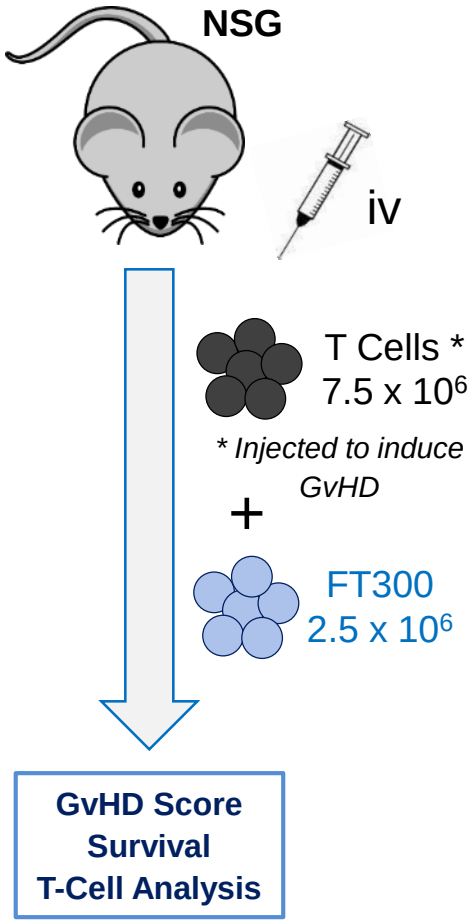
Potent Suppressor of T-Cell Cytokine Release Across HLA Barriers



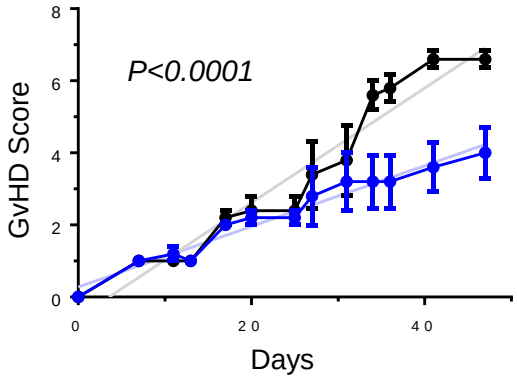
** Segmented by ratio of FT300 : Activated T Cells (5 independent donors for each ratio)*

FT300 *In Vivo* Functionality

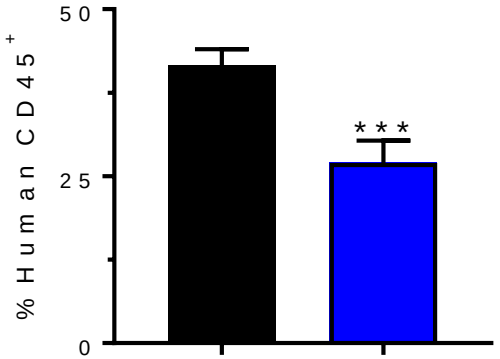
Suppression of T Cells and Disease Activity in GvHD Model



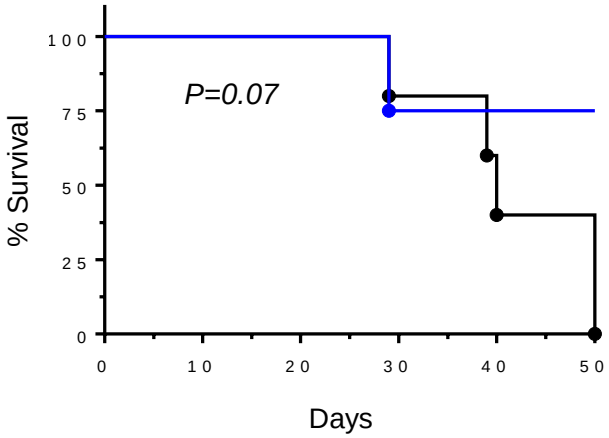
Disease Severity



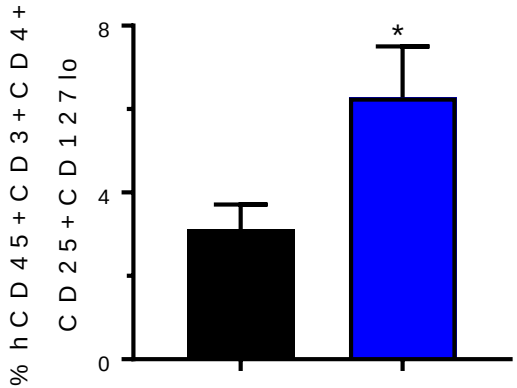
% T Cells



Survival



% Regulatory T Cells



T Cells T Cells + FT300

Fate THERAPEUTICS

Financial Summary

Fate Therapeutics

Financial Summary

Three Months Ended December 31, 2017	
Revenue	$1.0M
R&D Expense	$9.9M
G&A Expense	$3.4M
Operating Expense, Adjusted [1]	$11.9M
Cash & Cash Equivalents	$101.0M
Employees	80
Total Shares Outstanding [2]	66.9M

[1] Excludes $0.9M in stock-based compensation expense and $0.5M in Juno-related research expense.

[2] Includes 14.1M shares of common stock from conversion of non-voting preferred stock.

Fate THERAPEUTICS

Fate Therapeutics

First-in-Class Cellular Immunotherapy Pipeline

PROGRAM	PRECLINICAL	CLINICAL	RIGHTS
IMMUNO-ONCOLOGY			
FATE-NK100 – AML		**Phase 1**	Worldwide
FATE-NK100 – Ovarian		**Phase 1**	Worldwide
FATE-NK100 – Solid Tumor mAb Combo		**Phase 1**	Worldwide
FT500 (iNK Cell)	**OTS**	*Checkpoint Inhibitor Combination*	Worldwide
FT516 (Engineered hnCD16 iNK Cell)	**OTS**	*Monoclonal Antibody Combination*	Worldwide
FT538 (Engineered CD38- iNK Cell)	**OTS**	*Daratumumab Combination*	Worldwide
FT819 (Engineered CAR19 iT Cell)	**OTS**		Worldwide
IMMUNO-REGULATION			
ProTmune™ – Graft-versus-Host Disease		**Phase 2**	Worldwide
ToleraCyte™ – Autoimmune Disorders			Worldwide
FT300 (iMDS Cell)	**OTS**		Worldwide

Fate THERAPEUTICS

OTS *Off-the-Shelf using Clonal Master Induced Pluripotent Stem Cell (iPSC) Lines*

Better Cells For Better Therapies™