



Programmed Cellular Immunotherapies

Corporate Overview

January 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.

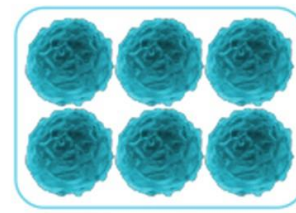
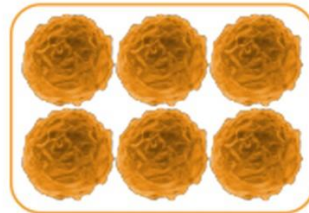
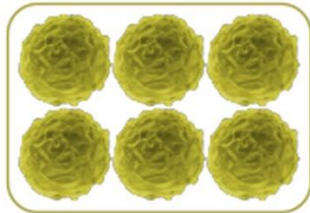
Fate Therapeutics

Mission



*To develop first-in-class cell-based immunotherapies
for cancer and immune disorders
by programming cell function and fate*

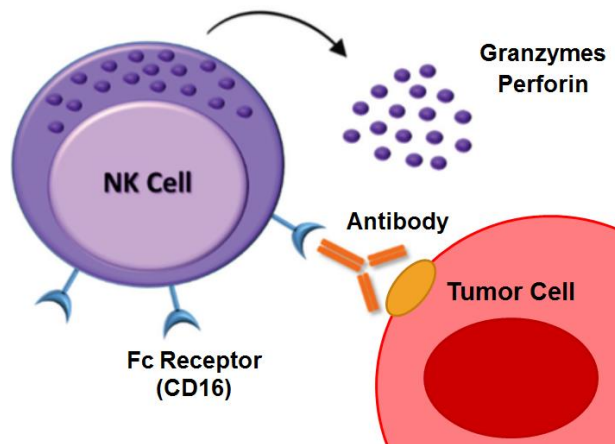
T cells | **CD34⁺** cells | **NK** cells



induced Pluripotent Cell Platform
for off-the-shelf engineered immunotherapies

Proven Role of Cells in Cancer Immunotherapy

Antibody-Dependent Cellular Cytotoxicity (ADCC)



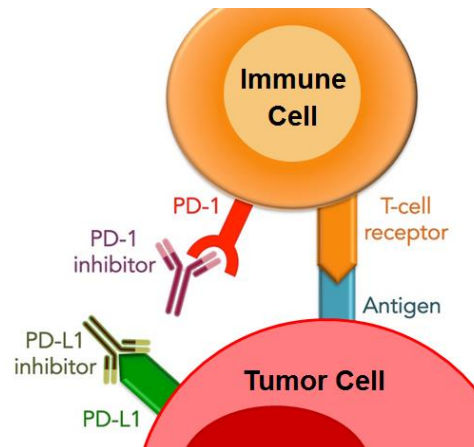
Rituxan
Rituximab

Herceptin
trastuzumab

ERBITUX
CETUXIMAB

DARZALEX

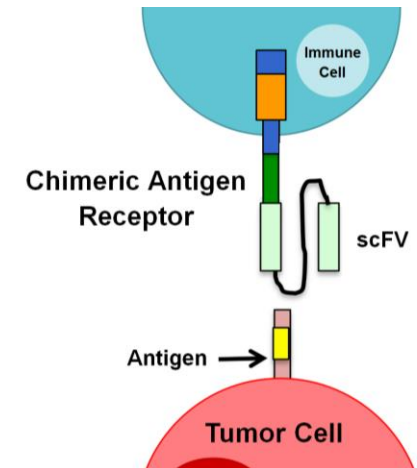
Immune Checkpoint Blockade



OPDIVO
(nivolumab)

KEYTRUDA
(pembrolizumab)

Targeted / Activated Cell Products



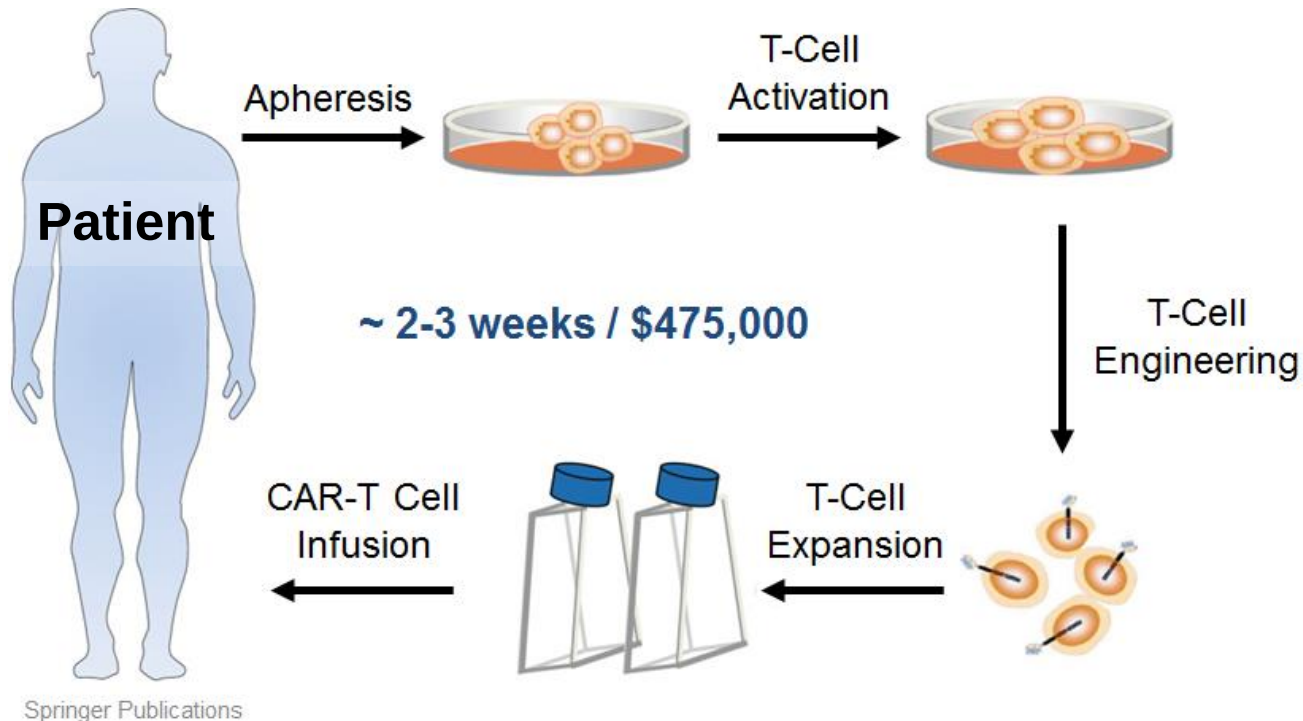
JUNO THERAPEUTICS **Kite** Pharma

NOVARTIS

Fcete
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

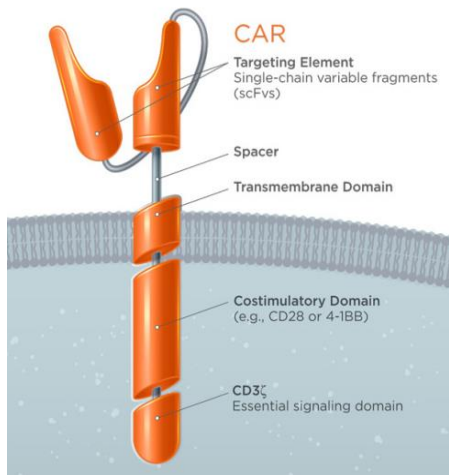
How do we Build on Early Successes and Transition From a Personalized Process to the Delivery of an Optimized Cell Product?

Fate Therapeutics – Juno Therapeutics Collaboration

Patient-derived CAR T Cells



Broad Research Collaboration Combining Fate's Small Molecules with Juno's Industry-leading CAR T-Cell Franchise



- May 2015: Four-year research collaboration to identify small molecule modulators that enhance the properties of T cells
 - Juno to fund all collaboration activities
- Juno has exclusive option to license small molecule modulators for genetically-engineered CAR / TCR T-cell therapies against selected tumor-associated antigen targets
 - Juno option excludes license for all iPSC-derived cell therapies (including CAR / TCR T-cell therapies)
- Significant economic opportunity
 - \$5M upfront payment, plus purchase of 1M shares of FATE c/s at \$8.00 per share
 - ~\$500M in target selection fees / milestones across first five modulated products
 - Single-digit royalties on sales of modulated products

Better Cells for Better Therapies™

Our Approach to Cellular Immunotherapy



Programmed Donor Cell Products



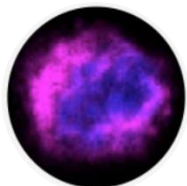
Donors

Cells from healthy donors with selected traits



Molecules

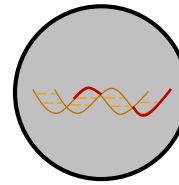
Ex vivo cell modulation to program biological properties



Cell Therapies

Cell products programmed for enhanced therapeutic function

Off-the-Shelf Cell Products



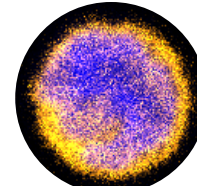
iPSC Line

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

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	<i>Checkpoint Inhibitor Combination</i>	Worldwide
FT516 (Engineered hnCD16 iNK Cell)	OTS	<i>Monoclonal Antibody Combination</i>	Worldwide
FT538 (Engineered CD38- iNK Cell)	OTS	<i>Daratumumab Combination</i>	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



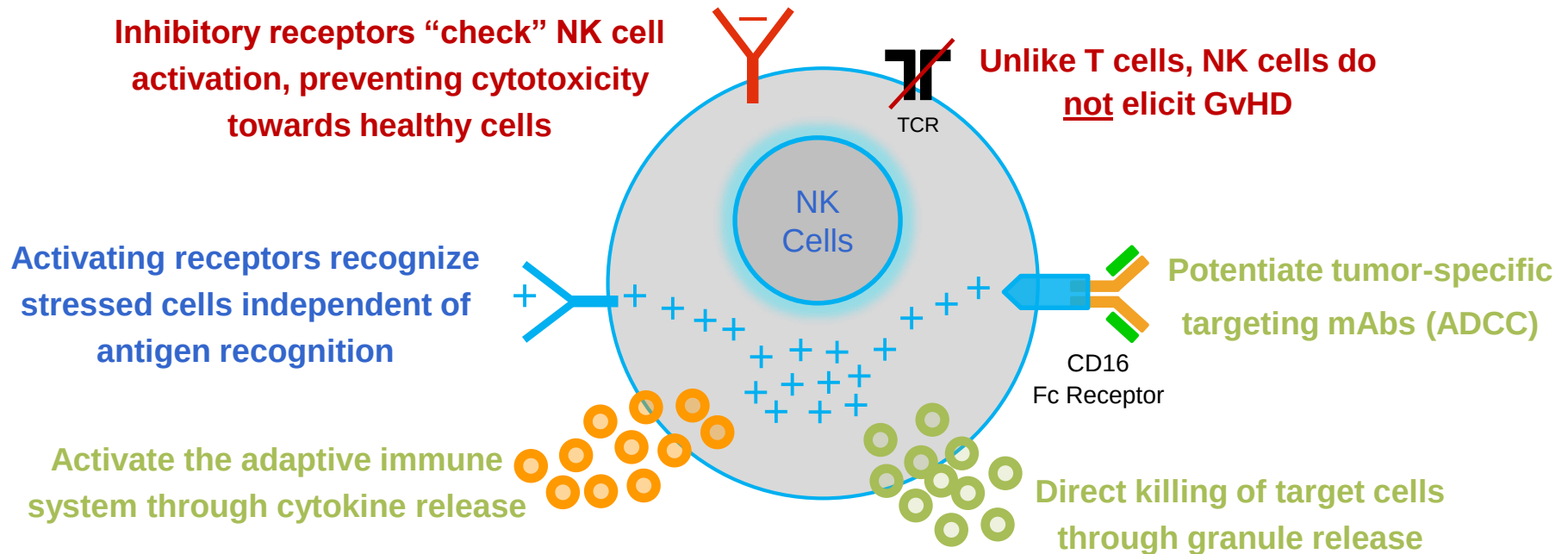
Immuno-Oncology Programs

Natural Killer Cells

Unique Properties Enable Off-the-Shelf Cancer Immunotherapy



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



Adaptive Memory NK Cells

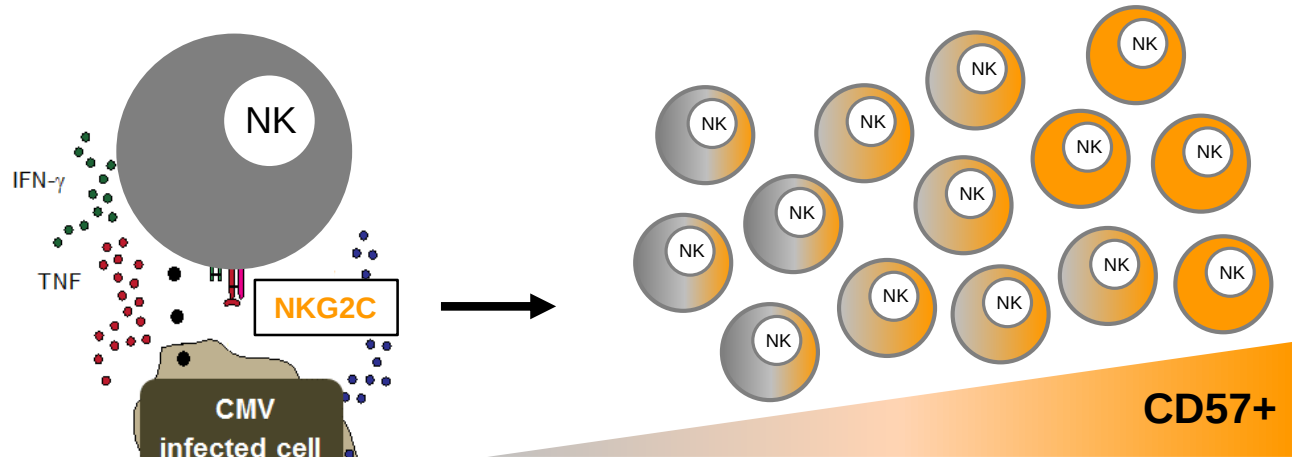
A Potent Subset of NK Cells with Unique Anti-tumor Attributes



Jeffrey S. Miller, MD



UNIVERSITY OF MINNESOTA
Driven to DiscoverSM



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

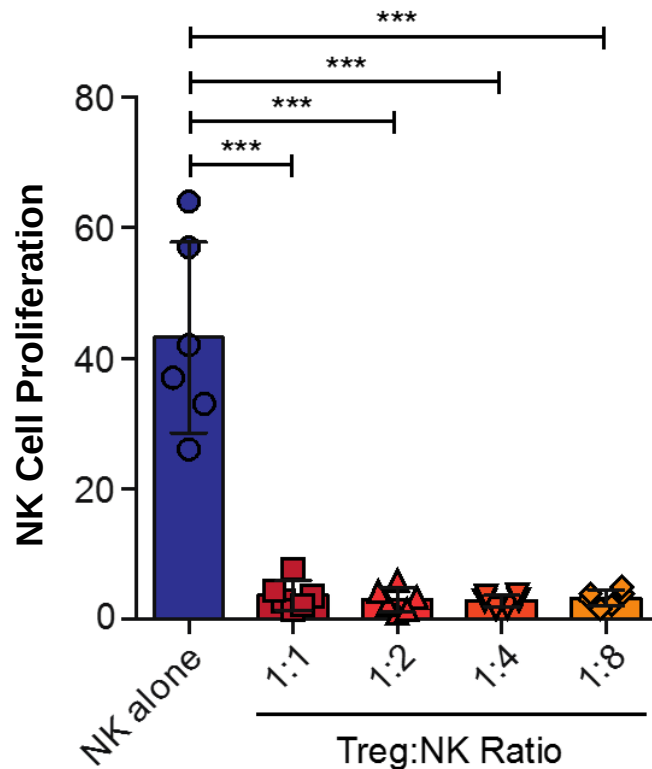
Resistant to Immune Checkpoint Pathways

Adaptive Memory NK Cells

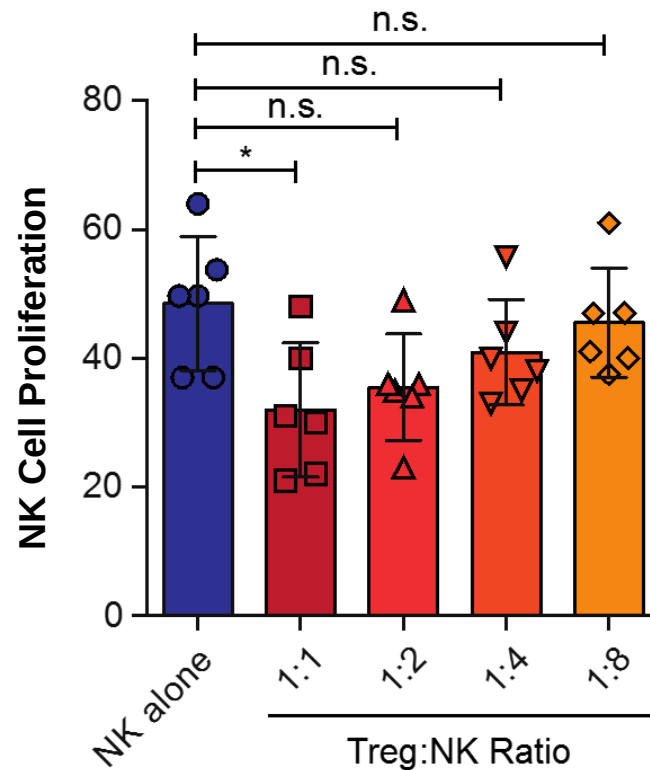
Resistance to Immune Checkpoint Pathways



Retained Proliferation Potential of Adaptive Memory NK Cells



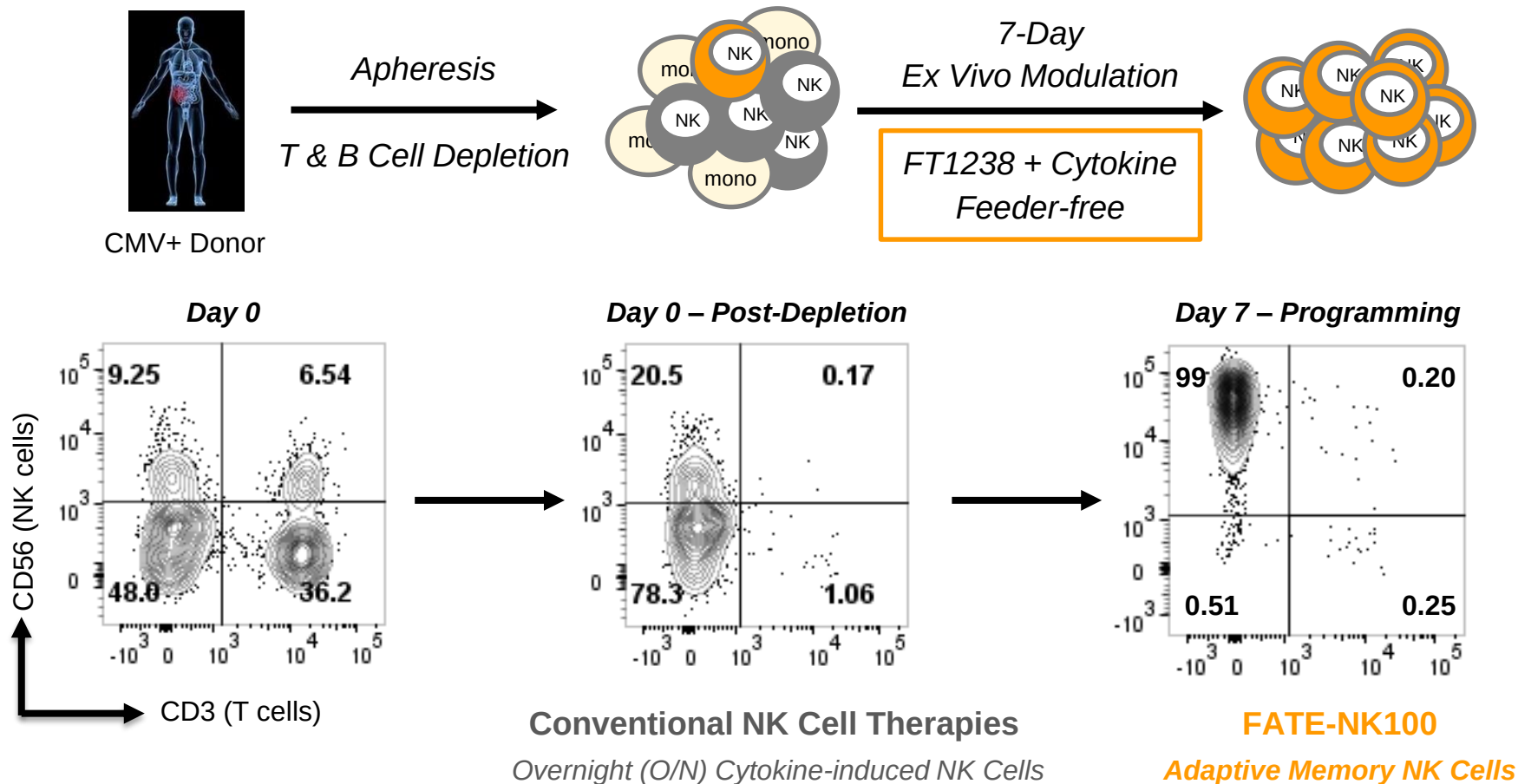
Conventional NK Cells



Adaptive Memory NK Cells

FATE-NK100

Realizing the Potential of Adaptive Memory NK Cells



Cancer Research

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

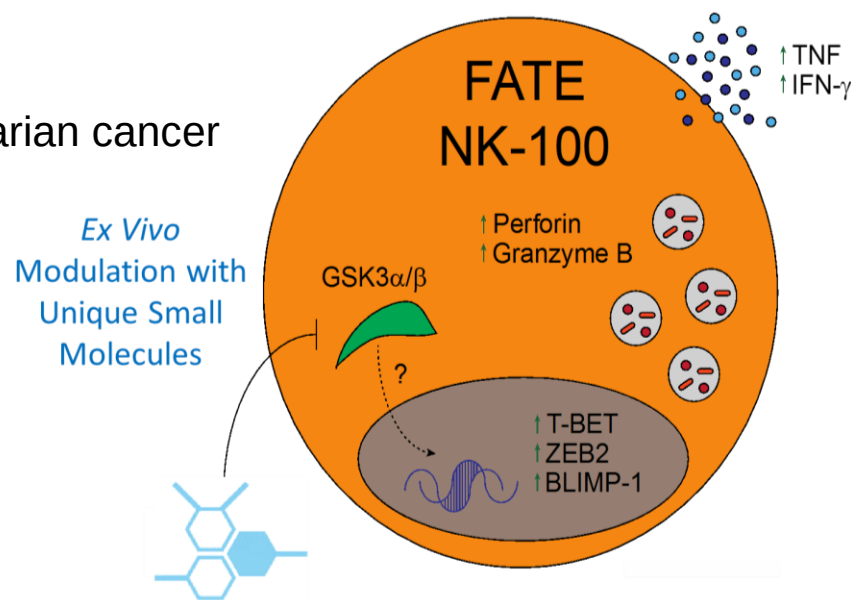
Cichocki et. al. 10.1158/0008-5472.CAN-17-0799

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



FATE-NK100

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 (1×10^7 TNC/kg)	Dose Cohort 2 (2×10^7 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
Day 10 Persistence	76% of PB NK cells were of FATE-NK100 origin	95% of PB NK cells were of FATE-NK100 origin
Day 14 Activity	~50% reduction in leukemic blasts	Morphologic leukemia-free state (mLFS) [†]

Dose Cohort 3 (up to 1×10^8 TNC/kg) currently enrolling

[†] Not sustained following a single IV infusion

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 dose cohort with no DLTs; 2nd subject dosed

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
- Patient screening ongoing

Cell-based Cancer Immunotherapy

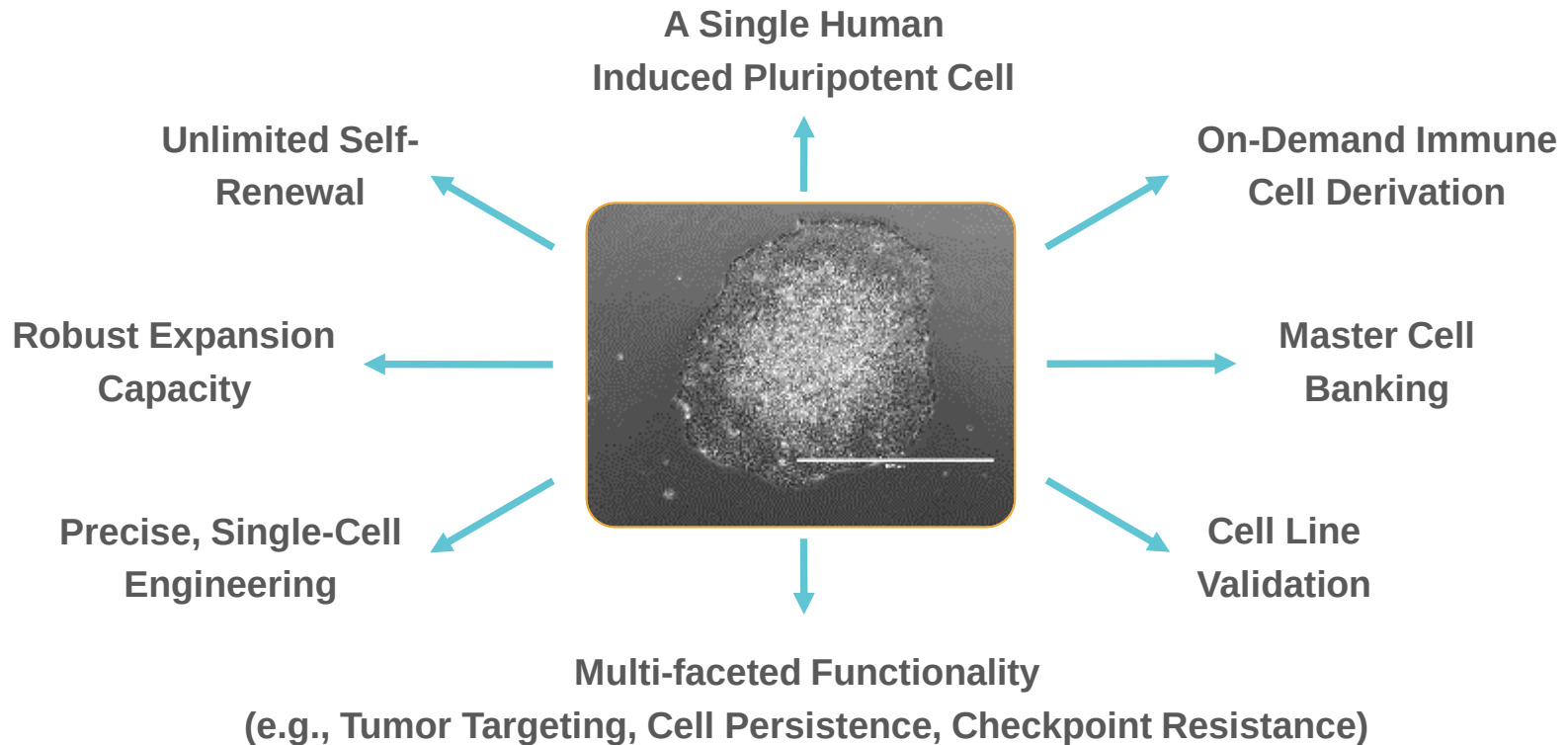
Advantages of an Off-the-Shelf Cell Product Paradigm



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

Human Induced Pluripotent Stem Cells

Renewable Source for Off-the-Shelf Cell Products



Renewable | Engineered

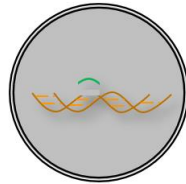
Clonal Cell Lines ---> Clonal Cell Products

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*



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



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

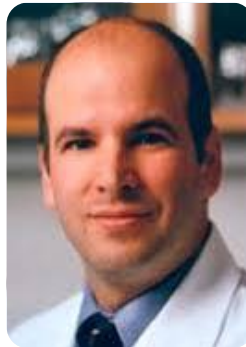


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Driven to DiscoverSM

Fote
THERAPEUTICS

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

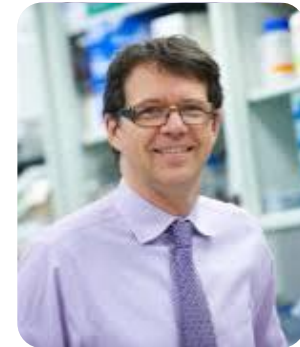


Dan Kaufman, MD PhD



UC San Diego

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



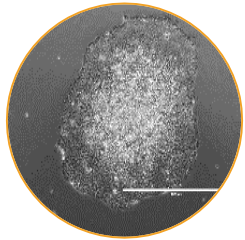
Michel Sadelain, MD, PhD



**Memorial Sloan Kettering
Cancer CenterTM**

Off-the-Shelf NK Cell Products

Targeting Three 2018 INDs for Combination Therapy

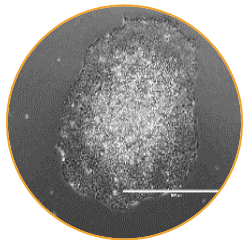


Master iPSC Line

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



OPDIVO™
KEYTRUDA®



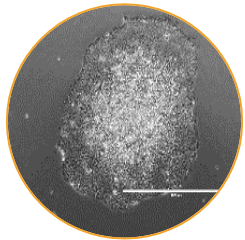
Engineered Master iPSC Line

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



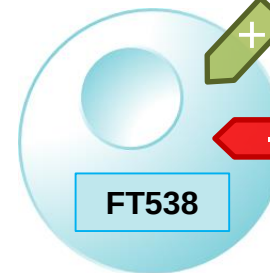
hnCD16

Herceptin® **ERBITUX®**
trastuzumab CETUXIMAB
Rituxan®
Rituximab



Engineered Master iPSC Line

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

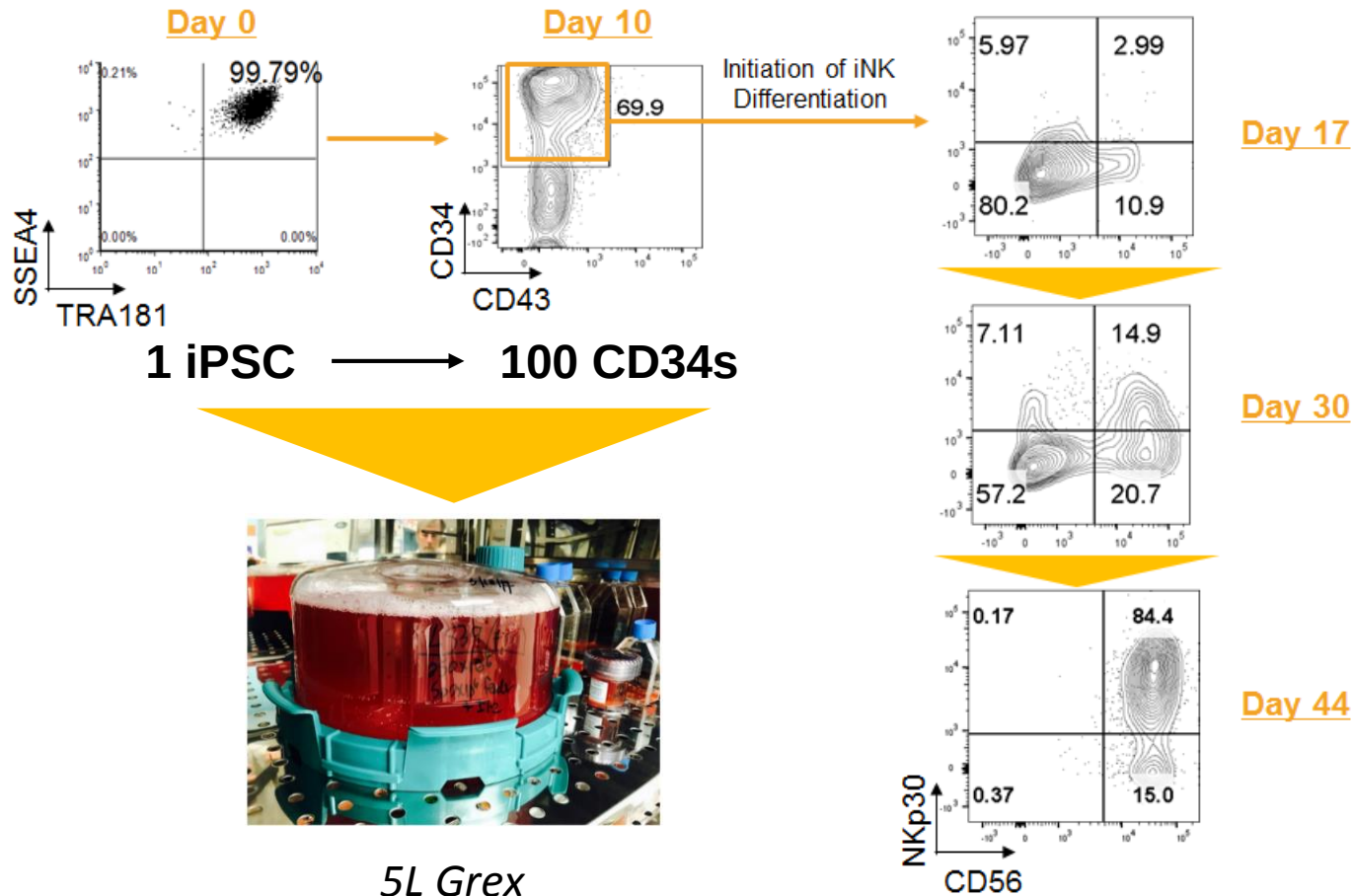


hnCD16

DARZALEX®
CD38

FT500 Manufacturing

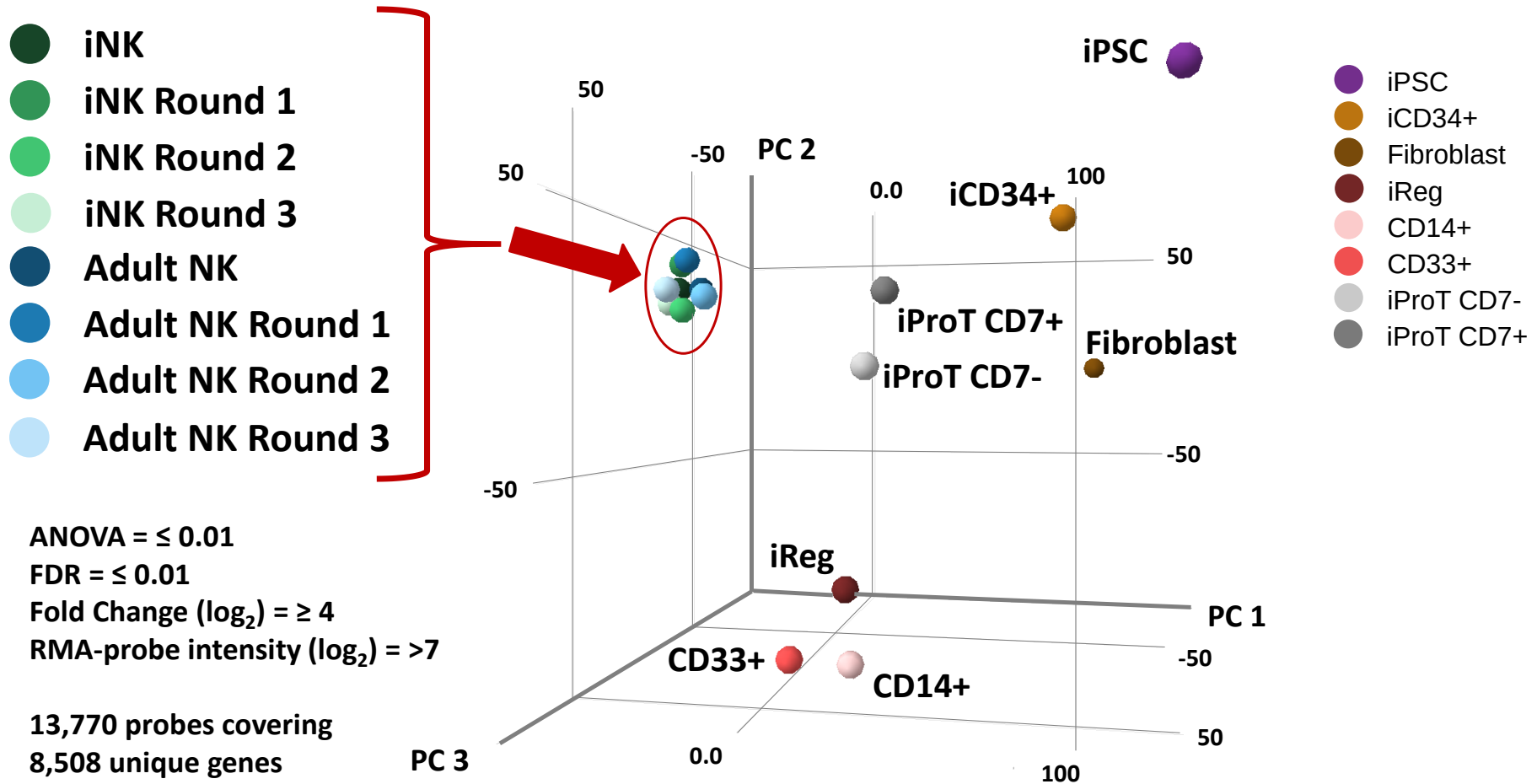
Robust Differentiation Protocol for iPSC-derived NK Cell Production



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

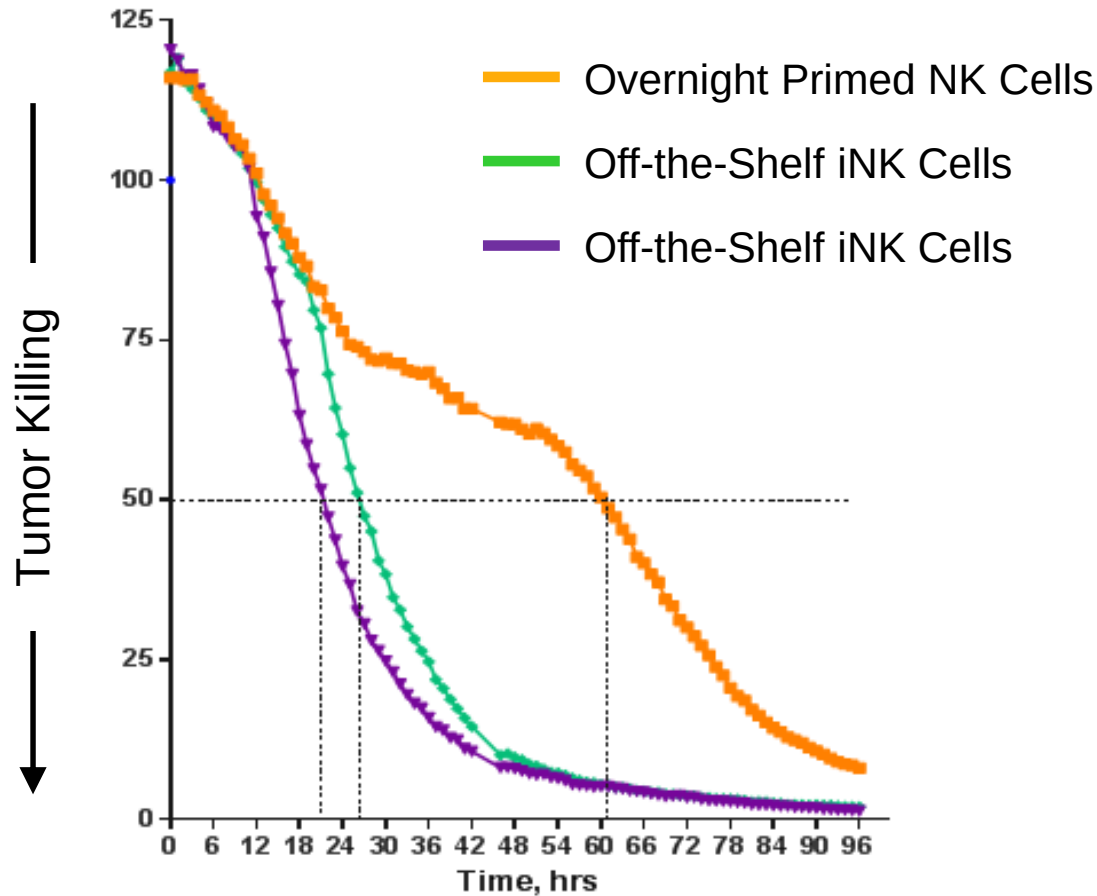


FT500 Cell Potency

iPSC-derived vs. Peripheral Blood NK Cells



SKOV3 (Ovarian Cancer) Killing Assay

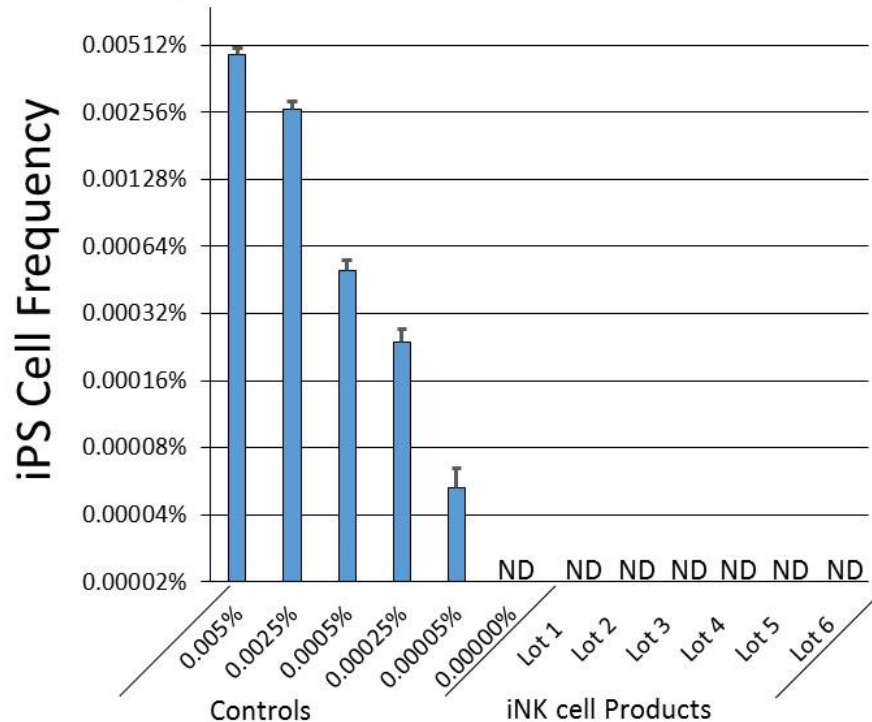


FT500 Purity

No Residual iPSCs in FT500 Cell Product



RT-qPCR detection of residual iPSCs



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

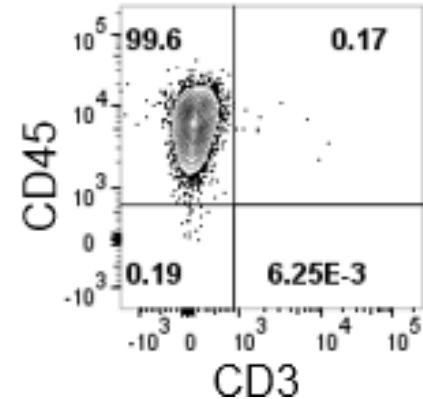
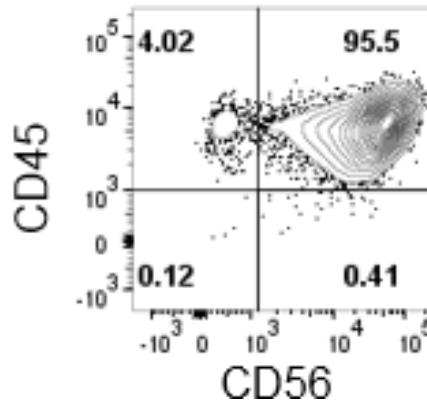
No iPSCs detected at the resolution of 1 in 2 million during multiple iNK cell manufacturing runs

FT500 Production

Technology Transfer to MCT cGMP Facility



1×10^6 iPSCs delivers 1×10^{12} NK cells during 44 day manufacturing process



Molecular and Cellular
Therapeutics

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



FT500

1×10^9 Cryopreserved Cells

FT500 in Combination with Checkpoint Inhibitors

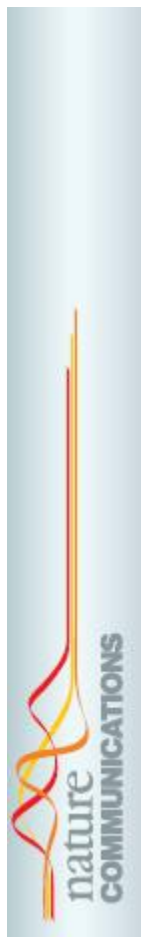
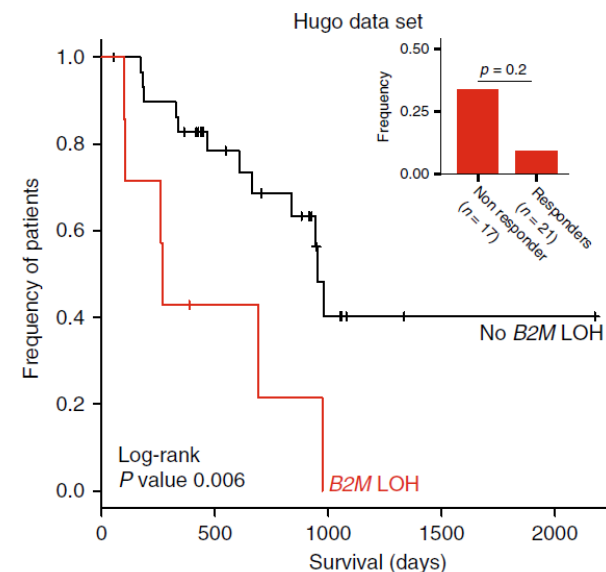
Synergy with T Cells to Infiltrate and Destroy 3D Tumor Mass



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. Rooney², Michal Barzily-Rokni¹, Jean-Pierre Eliaie⁴, Stacey L. Bjorgaard^{1,2}, Marc R. Hammond¹, Hans Vitzthum¹, Shauna M. Blackmon¹, Dennie T. Frederick¹, Mehlika Hazar-Rethinam¹, Brandon A. Nadres¹, Emily E. Van Seventer¹, Sachet A. Shukla^{2,5}, Keren Yizhak², John P. Ray², Daniel Rosebrock², Dimitri Livitz², Viktor Adalsteinsson², Gad Getz^{2,4}, Lyn M. Duncan⁴, Bo Li⁶, Ryan B. Corcoran¹, Donald P. Lawrence¹, Anat Stemmer-Rachamimov⁴, Genevieve M. Boland⁷, Dan A. Landau^{2,8,9}, Keith T. Flaherty¹, Ryan J. Sullivan¹ & 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.

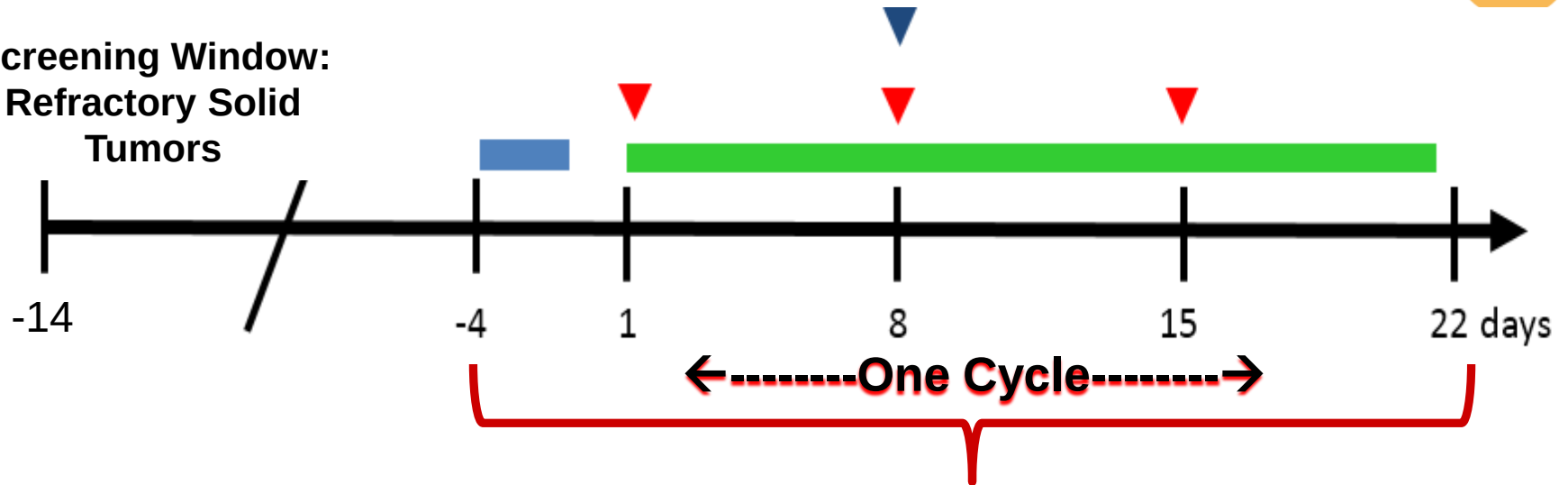


FT500 Proposed FIH Study

Multiple Cycles of FT500 + Checkpoint Inhibitor Dosed Weekly



Screening Window:
Refractory Solid
Tumors



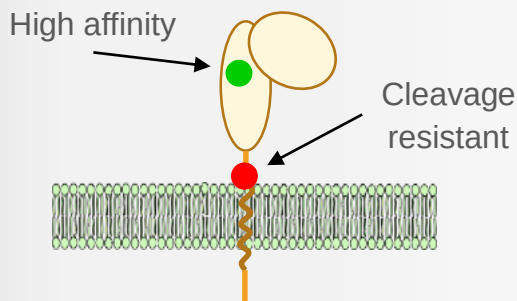
- Conditioning** Cyclophosphamide: 300 mg/m^2 intravenous (IV) on Day-4 (or Day -3)
Fludarabine 25 mg/m^2 IV on Days -4, -3, (or Day -3 and -2)
- IL2 (2x/week)** IL-2: 3 million units SC twice weekly from Day1 thru Day21
- FT500** 3 doses at 1 week intervals*
- Checkpoint Inhibitor** Given per SOC dose and frequency until disease progression

FT516 Engineered hnCD16+ NK Cell Product Candidate

Mid-2018 IND Filing Expected for Monoclonal Antibody Combo

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

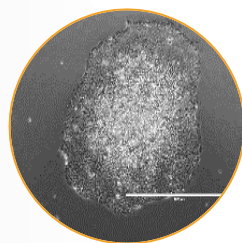
Herceptin
trastuzumab



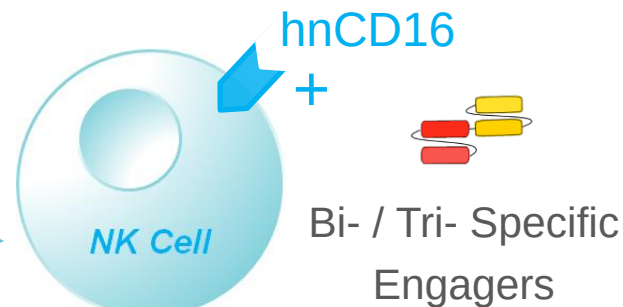
ERBITUX
CETUXIMAB

DARZALEX

Renewable Engineered
Pluripotent Cell Line



Engineered hnCD16 iNK
Cells for ADCC



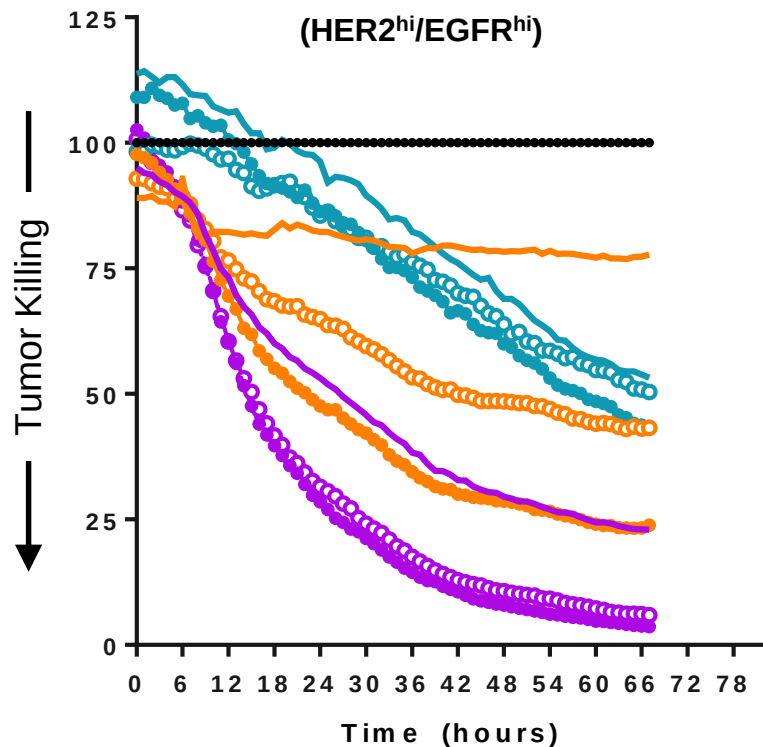
FT516 Engineered hnCD16+ NK Cell Product Candidate

In Vitro ADCC for Solid Tumors



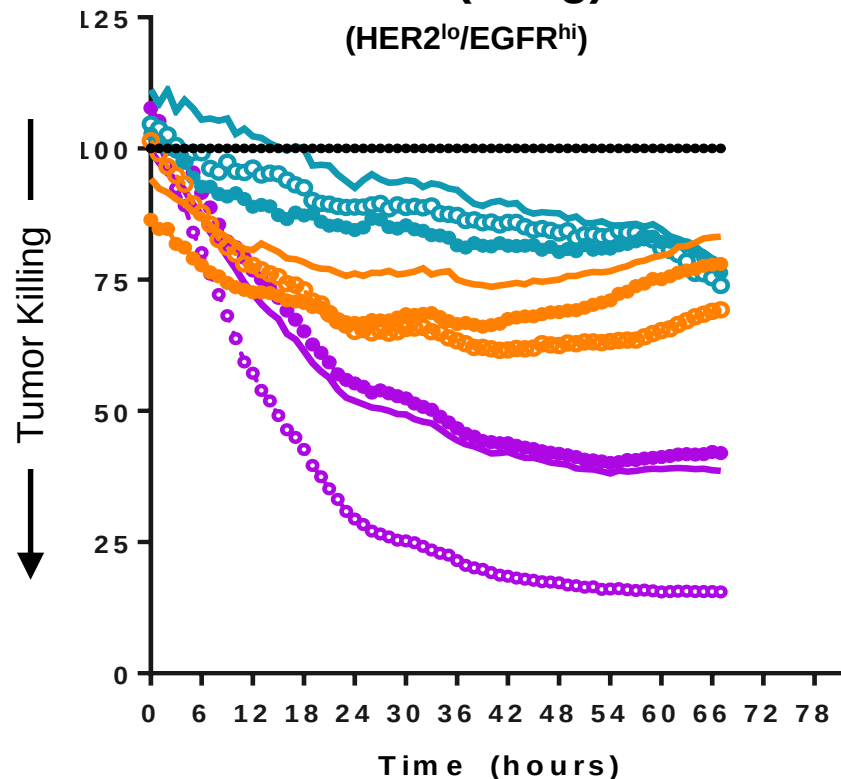
SKOV3 (Ovarian)

(HER2^{hi}/EGFR^{hi})



A549 (Lung)

(HER2^{lo}/EGFR^{hi})



Target cells only

pbNK (n=3)

Cord blood NK (n=1)

hnCD16 iNK (n=3)

No antibody

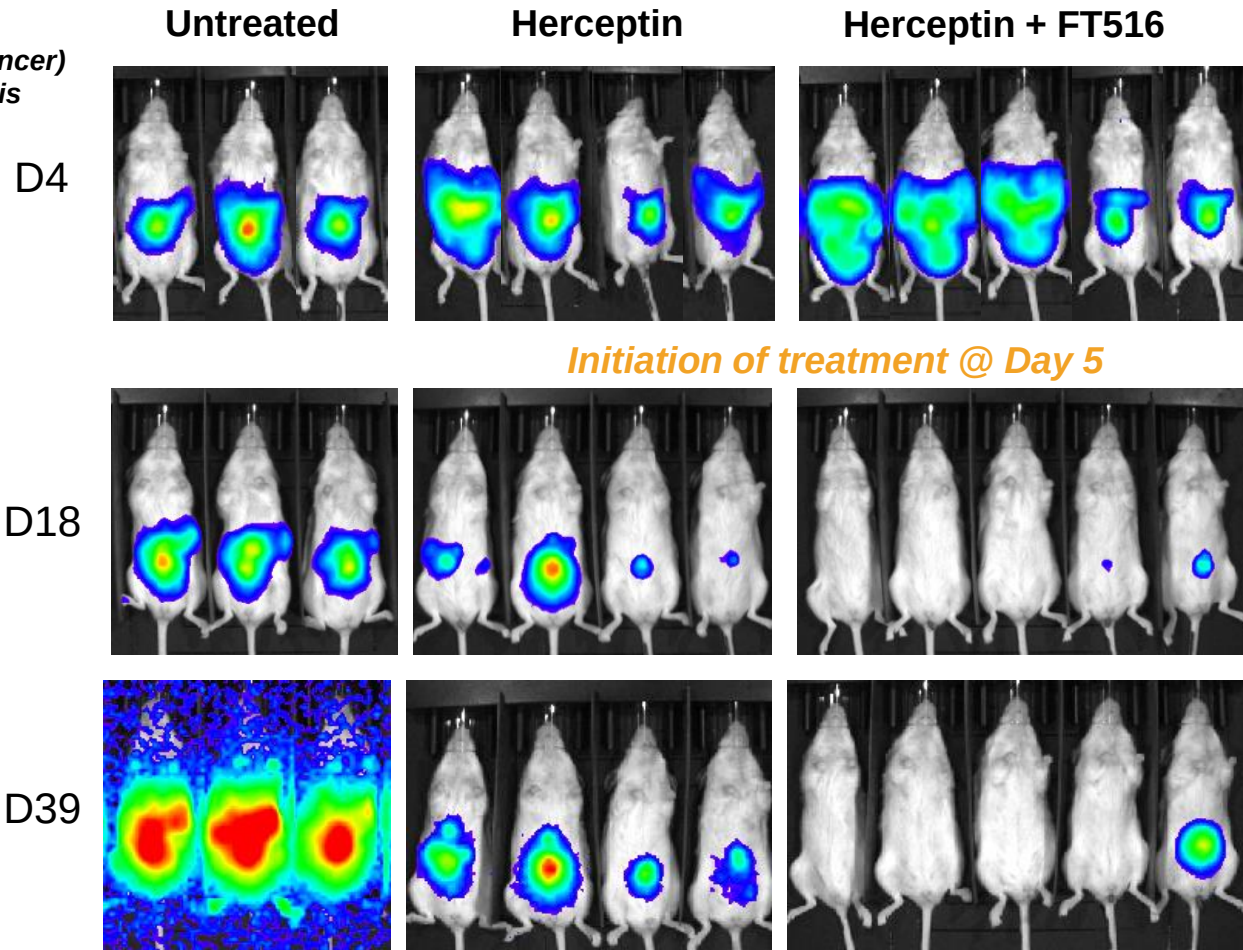
Anti-Her2

Anti-EGFR

FT516 Engineered hnCD16+ NK Cell Product Candidate

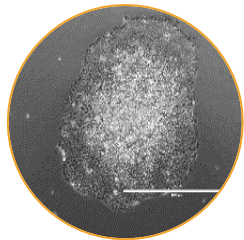
In Vivo POC

Luc-SKOV3 (Ovarian Cancer)
Tumor Image Analysis



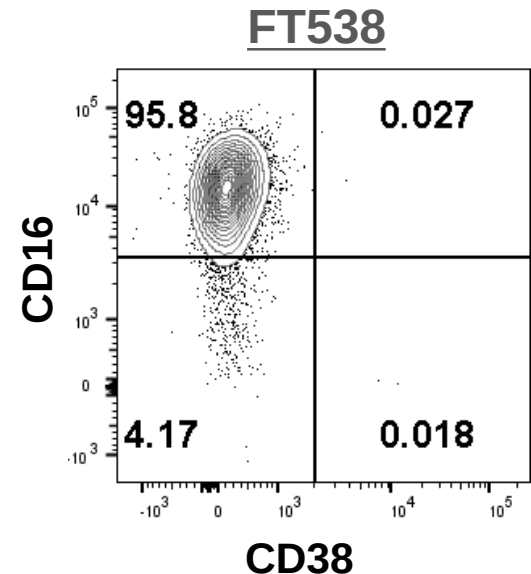
FT538 Engineered hnCD16+ / CD38-null NK Cell Product

2H18 IND Filing to Complement Darzalex Therapy



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

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



**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*

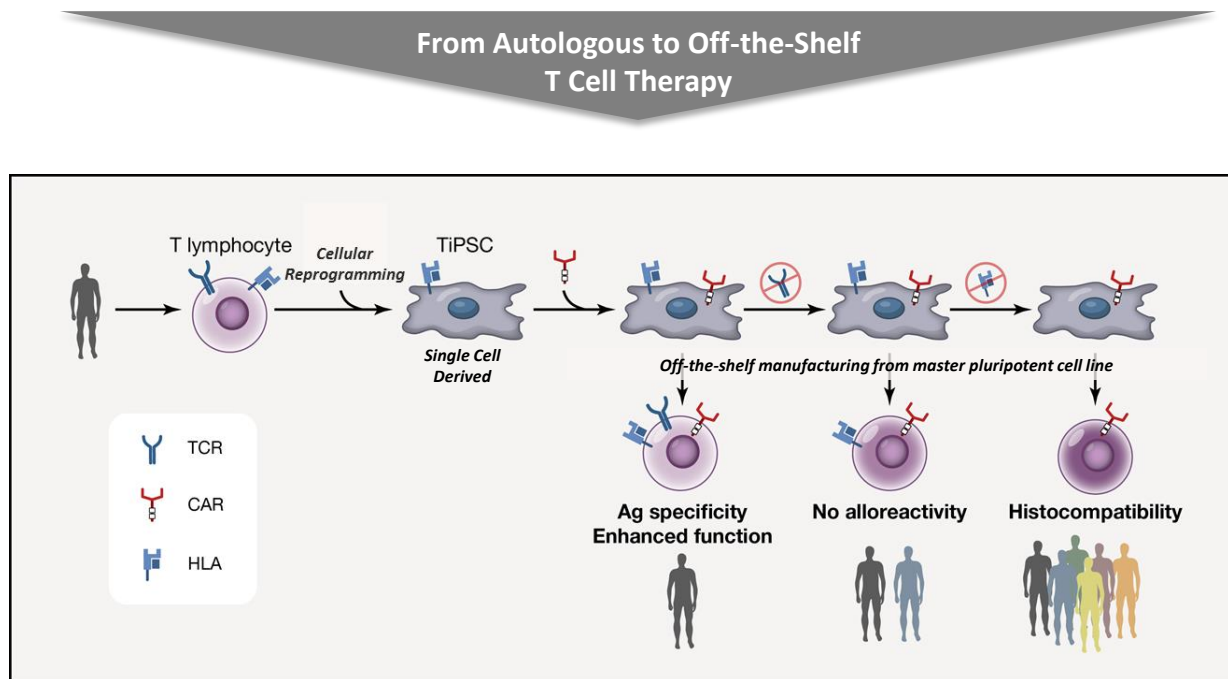
*Foundational Intellectual Property Covering Pluripotent Cell-derived Engineered T Cells
Exclusively Licensed to Fate Therapeutics*

Off-the-Shelf T Cell Products

The Sadelain Roadmap



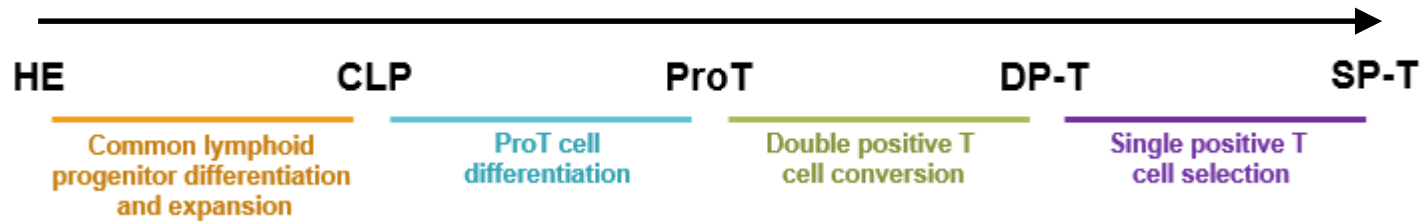
“Engineering therapeutic attributes into pluripotent cell lines is a breakthrough approach to renewably generate potent T-cell immunotherapies. This unique approach offers the prospect for off-the-shelf delivery of T-cell therapies with enhanced safety and therapeutic potential at the scale necessary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



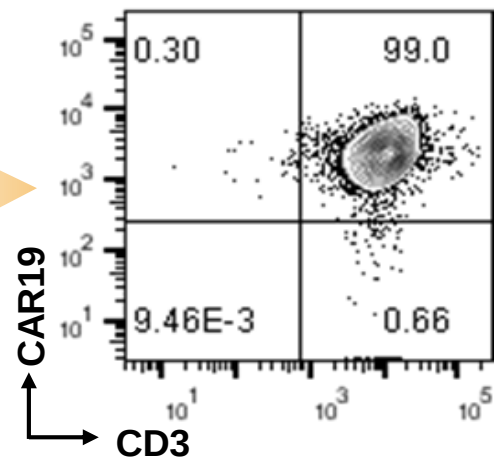
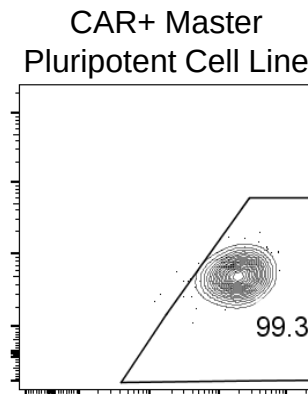
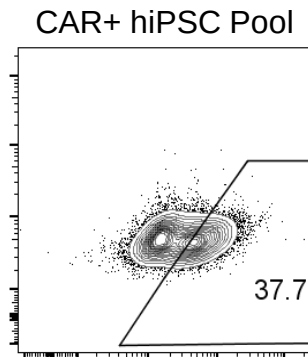
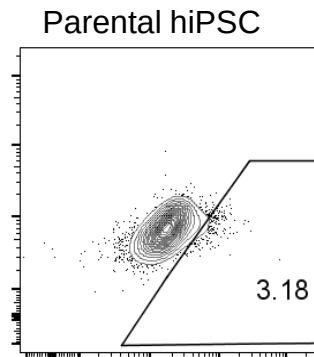
Adapted from: Themeli, Riviere & Sadelain, *Cell Stem Cells*, 2015

Ex Vivo Generation of CAR19+ CD8αβ+ T Cells

Generation from Clonal Engineered Master iPSC Line



Clonal CAR19 Master hiPSC Line

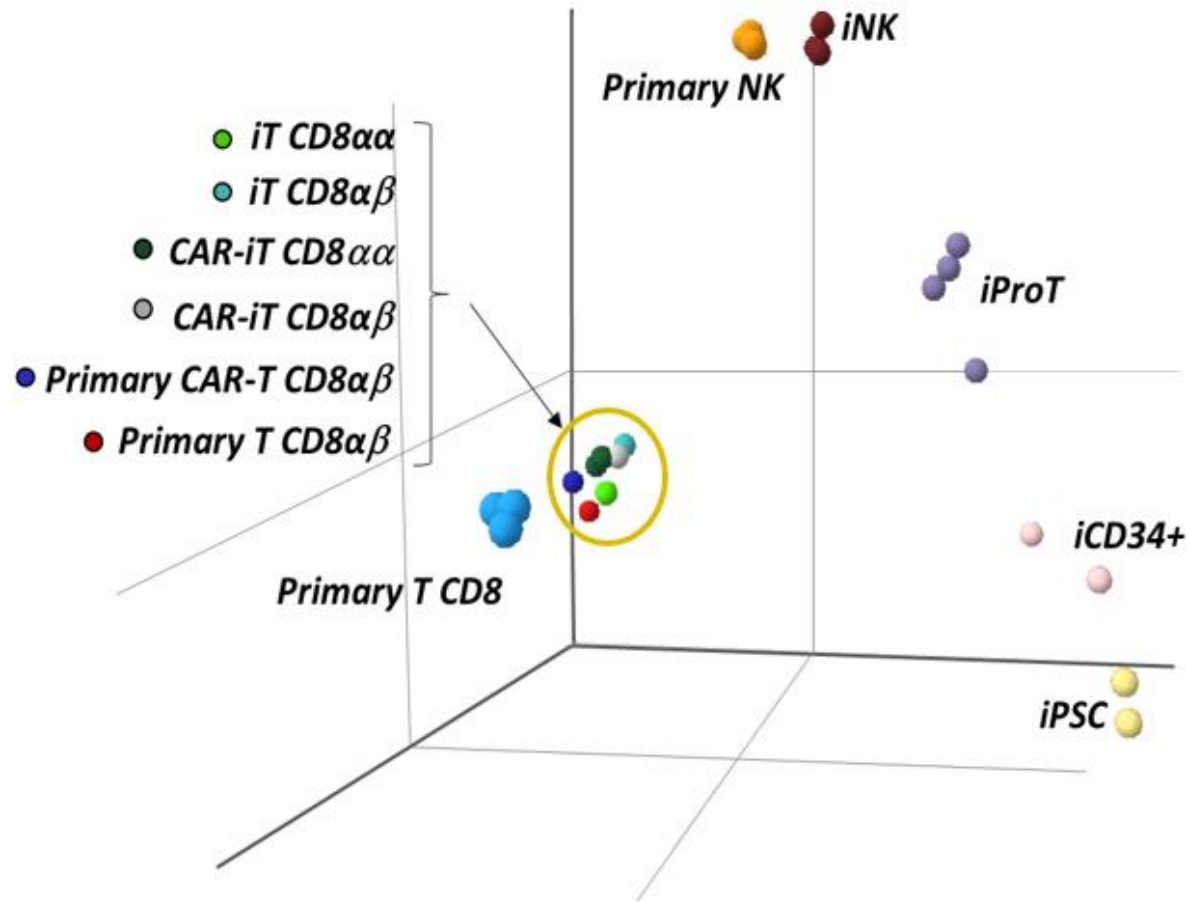


- Clonal (from a single cell)
- Pluripotent
- Renewable (unlimited source)

- Homogenous population
- Reproducible
- Well-defined

iPSC-Derived CAR19+ CD8 $\alpha\beta$ + T Cells

Global Gene Expression Comparison vs. Primary T Cells



FT819 CAR19 TCR-null T Cell Product Candidate

Generated from Clonal Engineered Master iPSC Line



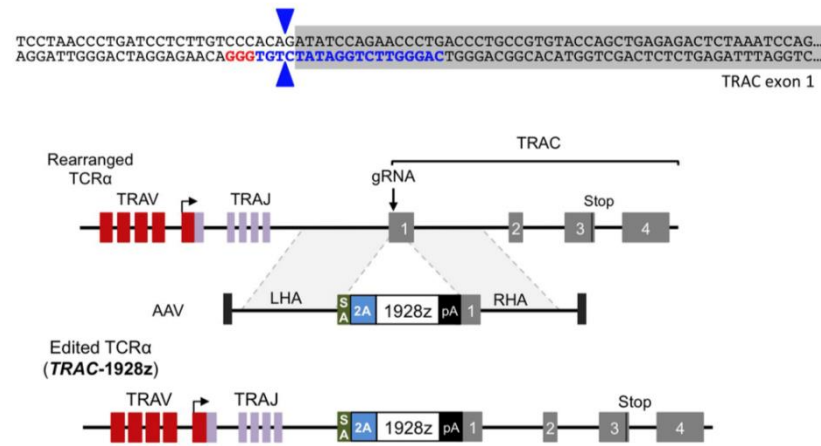
LETTER

doi:10.1038/nature21405

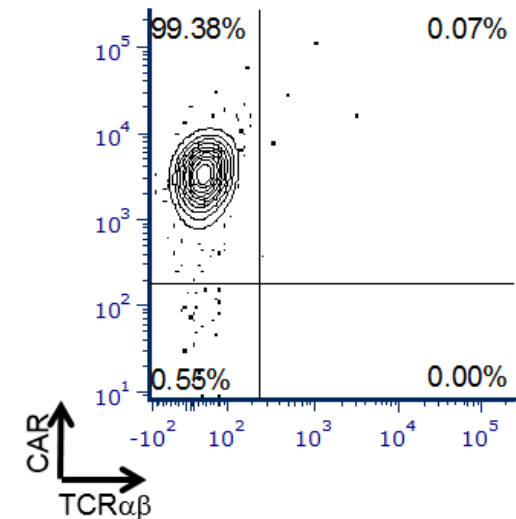
Targeting a CAR to the *TRAC* locus with CRISPR/Cas9 enhances tumour rejection

Justin Eyquem^{1*}, Jorge Mansilla-Soto^{1*}, Theodoros Giavridis¹, Sjoukje J. C. van der Stegen¹, Mohamad Hamieh¹, Kristen M. Cunanan², Ashlesha Odak¹, Mithat Gönen² & Michel Sadelain¹

TCR Disruption

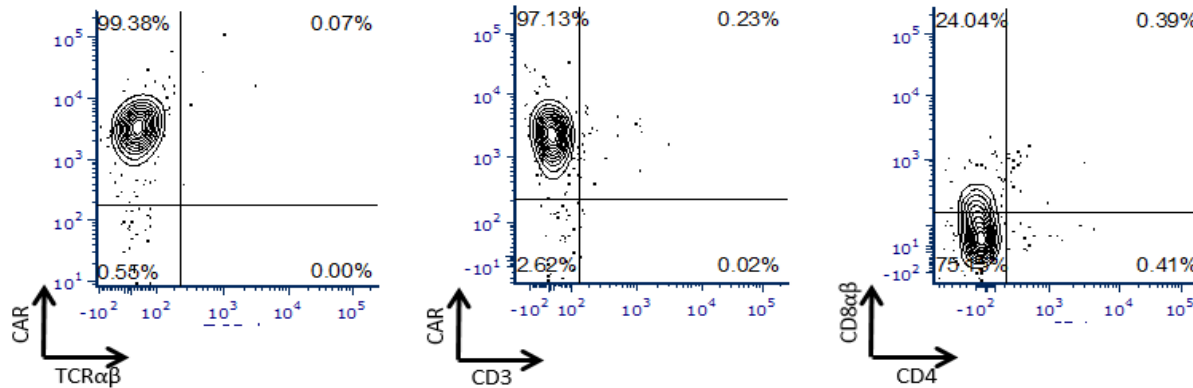


TRAC-encoded CAR Expression

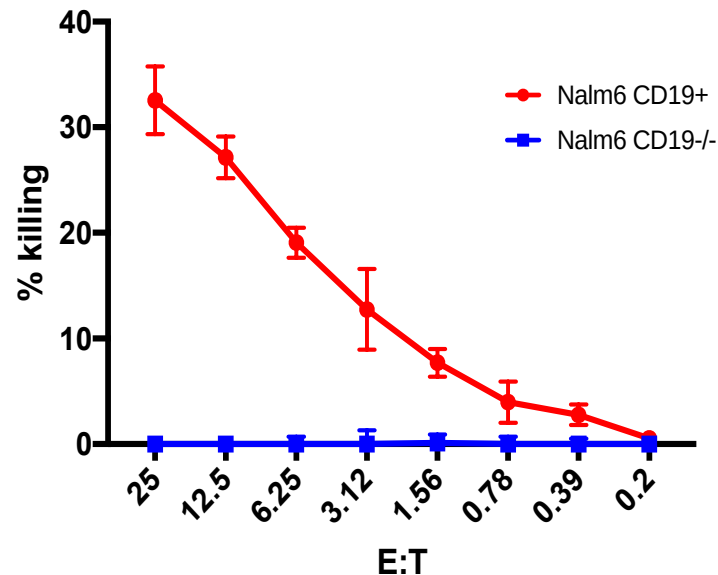


FT819 CAR19 TCR-null T Cell Product Candidate

Generation of iPSC-derived CD8 $\alpha\beta$ ⁺ T Cells with Antigen Specificity



Specific Cytotoxicity





Immuno-Regulatory Programs



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

ProTmune™

*Small molecule programmed
mobilized peripheral blood graft*



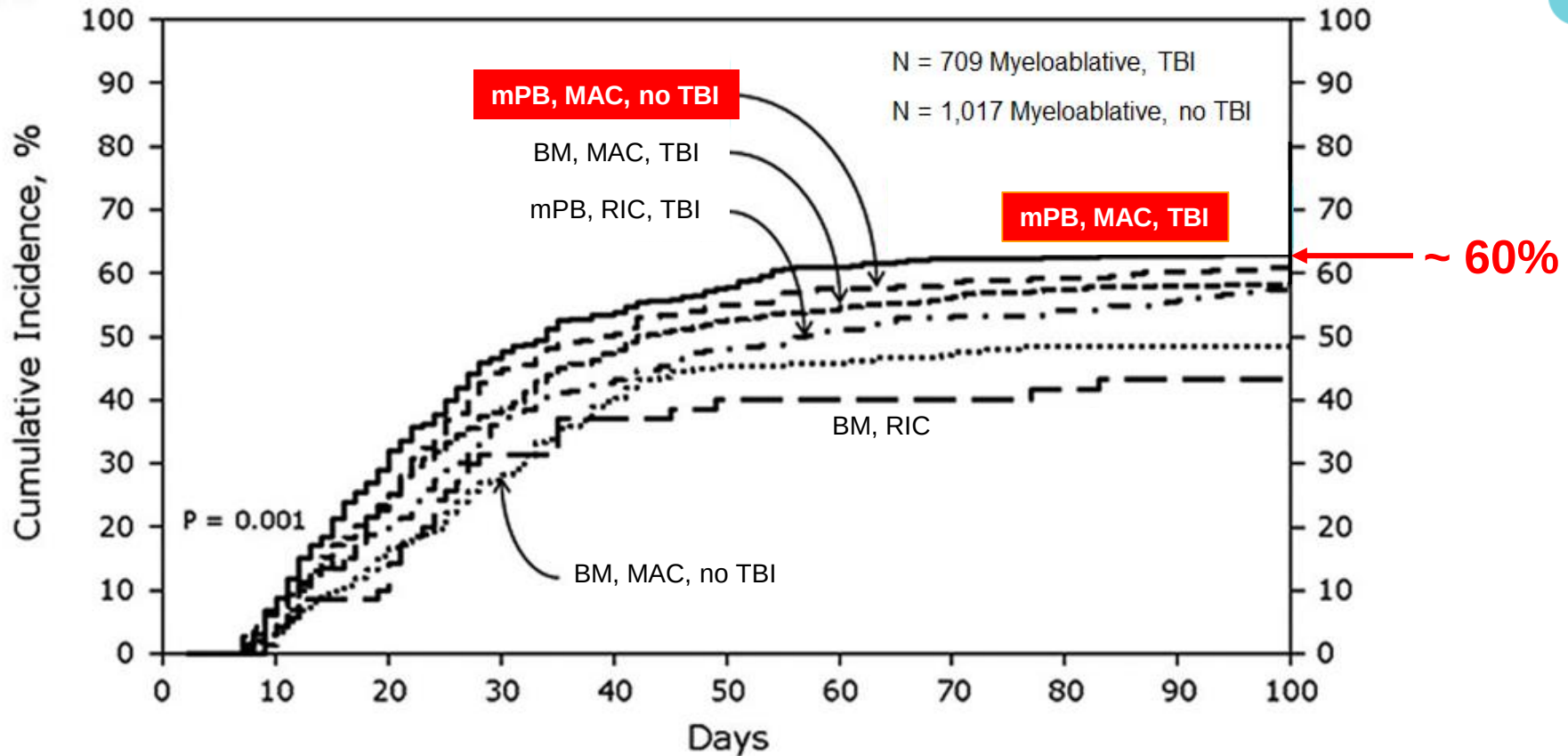
FT1050 + FT4145



- **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

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)

Pathophysiology of Acute GvHD

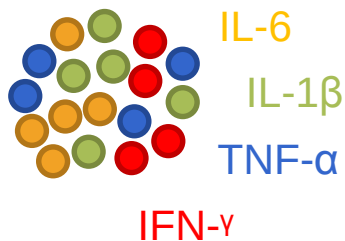


Conditioned
Patient

Tissue Damage



Cytokine Storm



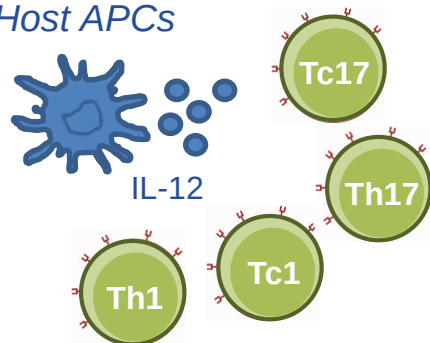
T Cells



Donor

Donor Allo-reactive T-cell Activation

Host APCs

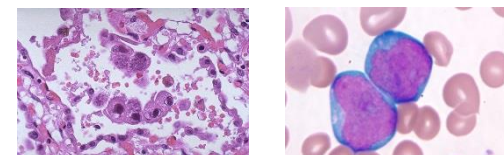


Assault on Patient Tissue



Acute GvHD (gut, liver, skin)
~40-80% D100 cumulative incidence
~10-20% early mortality

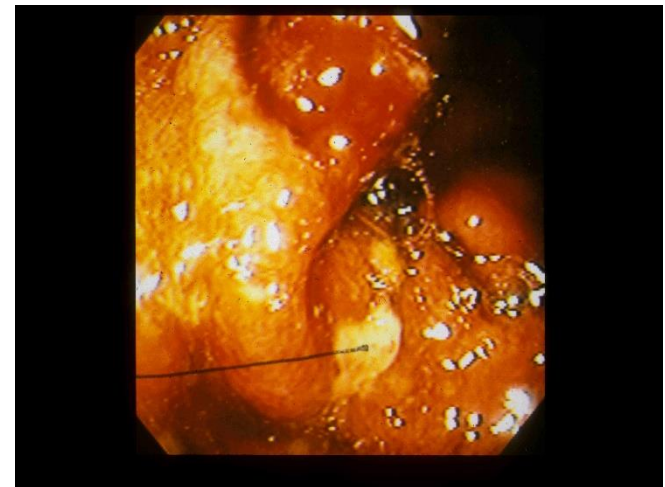
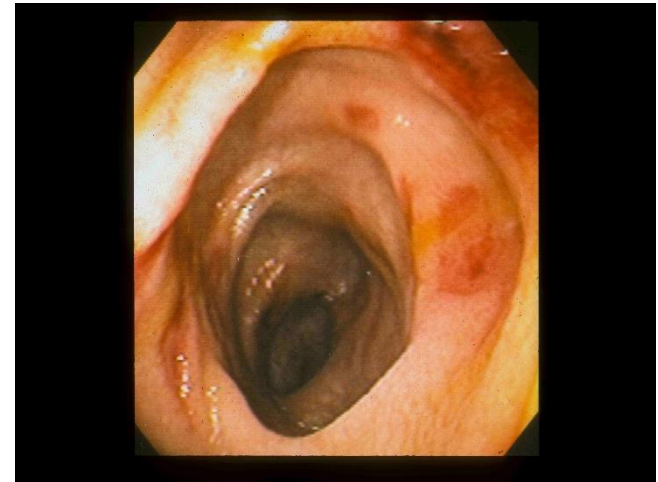
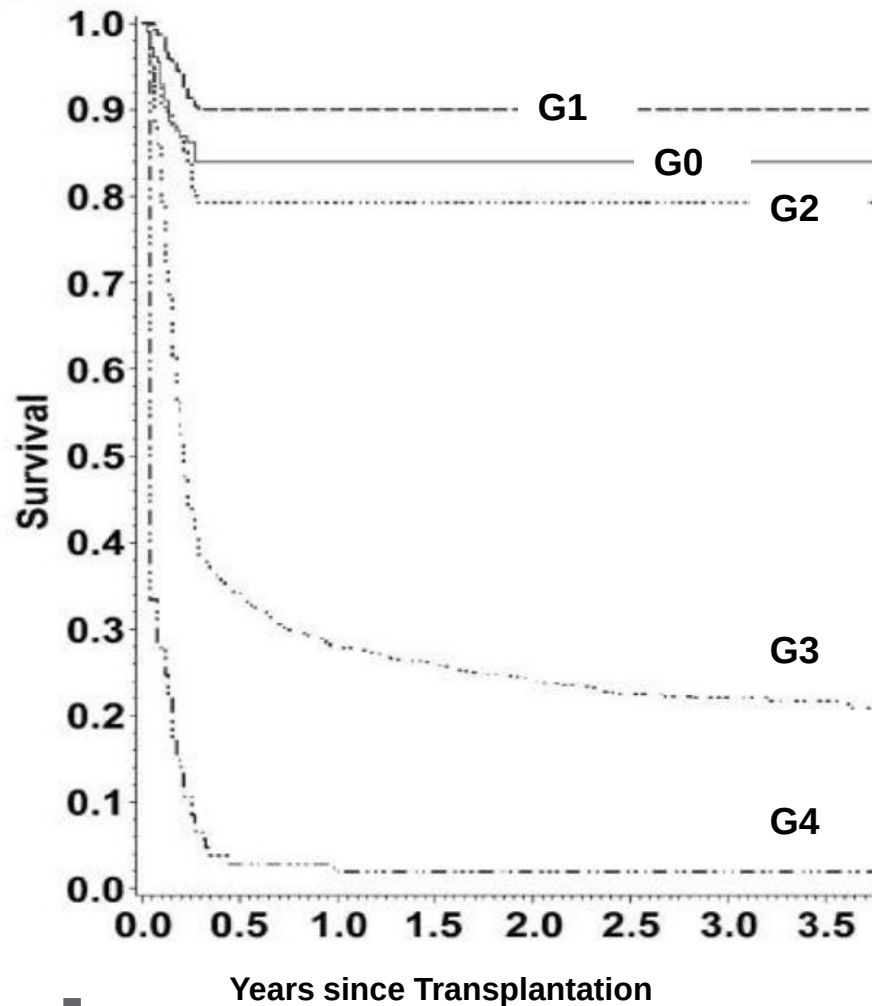
Immunosuppressive Agents



Severe Infections
~70% D100 cumulative incidence
Relapse
~35% 1YR cumulative incidence

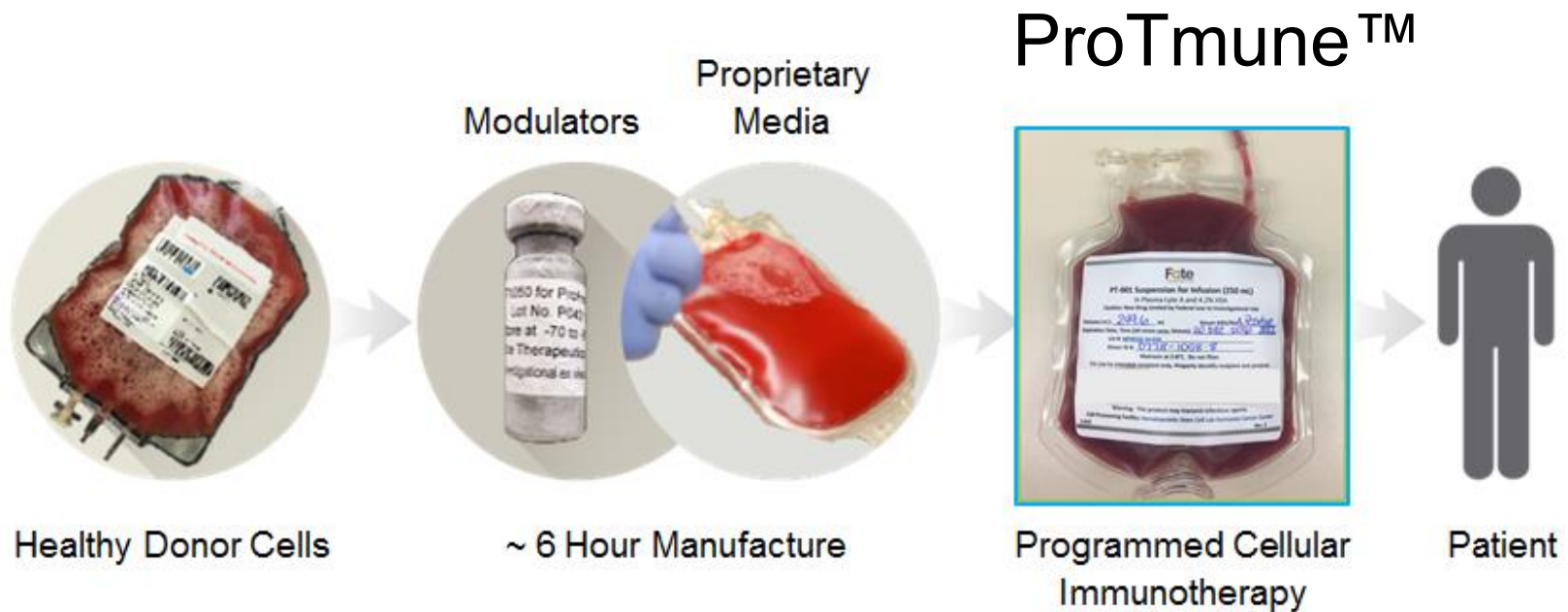
Acute Graft-vs-Host Disease

Severe Acute GvHD Causes Mortality



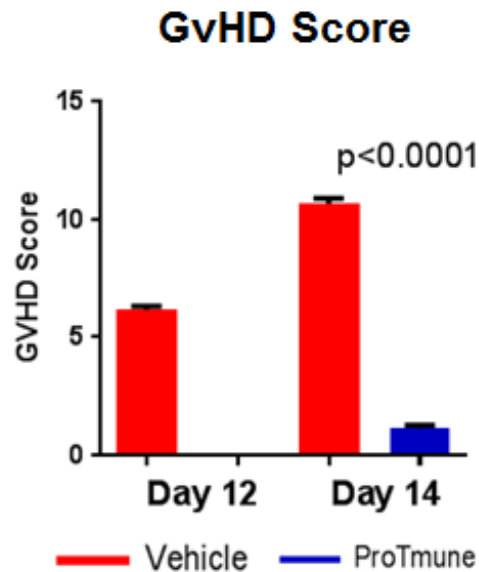
ProTmune™

The Next-Generation Hematopoietic Cell Graft

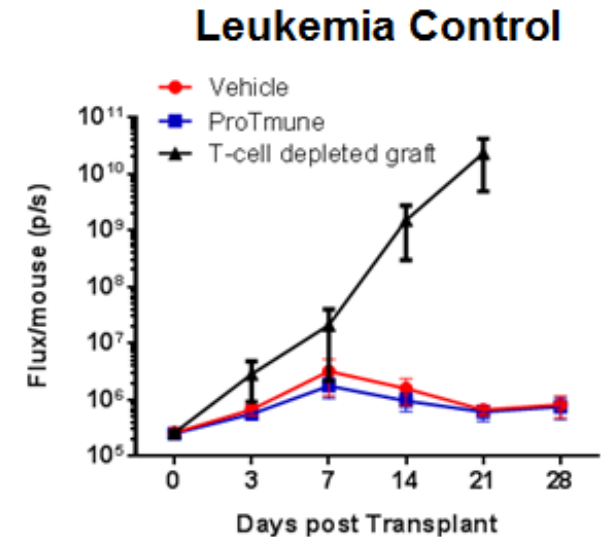
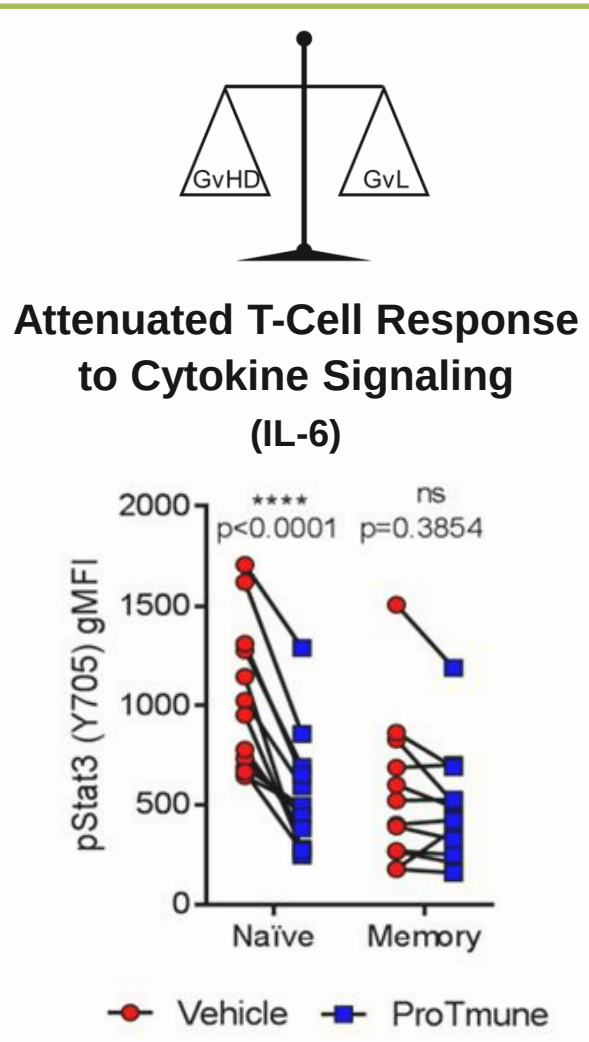




Striking the Balance between GvHD and GvL



Naïve T Cells



Memory T Cells



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



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

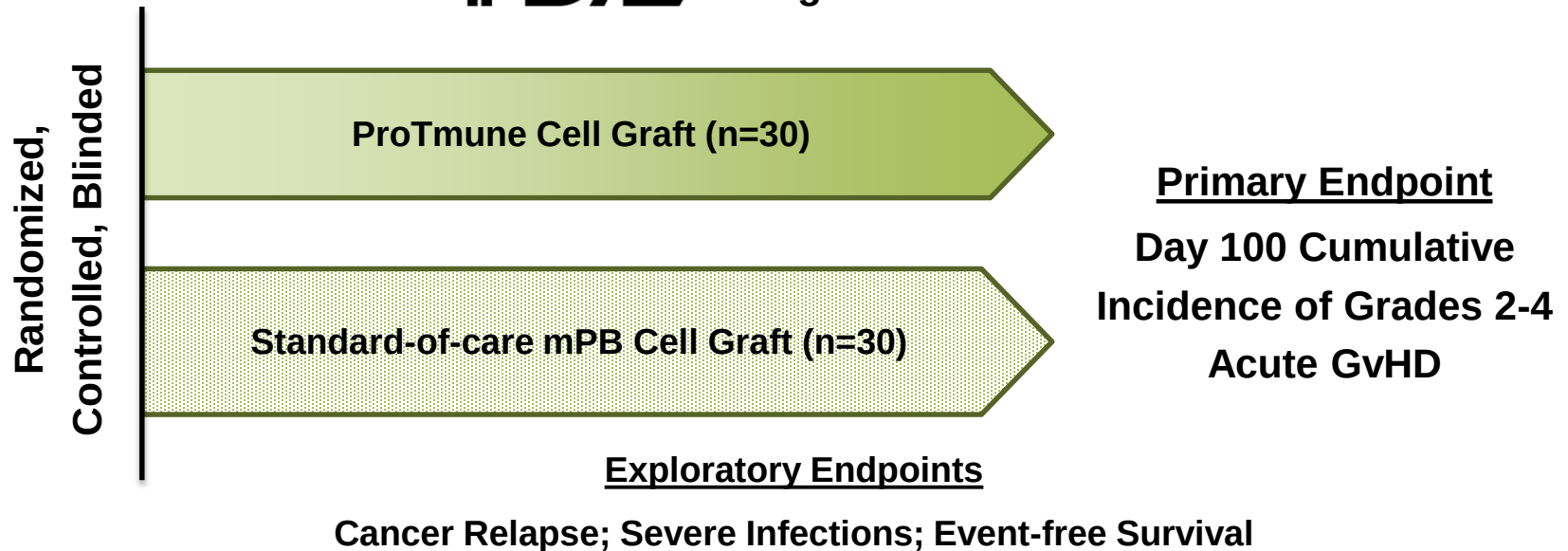


Phase 2 Stage: 60 Subjects, Double-blinded, Randomized, Controlled

Currently Enrolling at 14 U.S. Centers



**Fast Track
Designation**

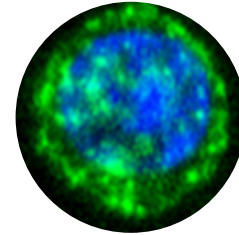




- **Preventive approach to address leading cause of early morbidity and mortality**
 - 40 to 80% of patients undergoing allogeneic HCT experience acute GvHD
 - Death directly attributable to acute GvHD or its treatment occurs in 10 to 20% of patients
 - No approved preventive therapies in the U.S.
- **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

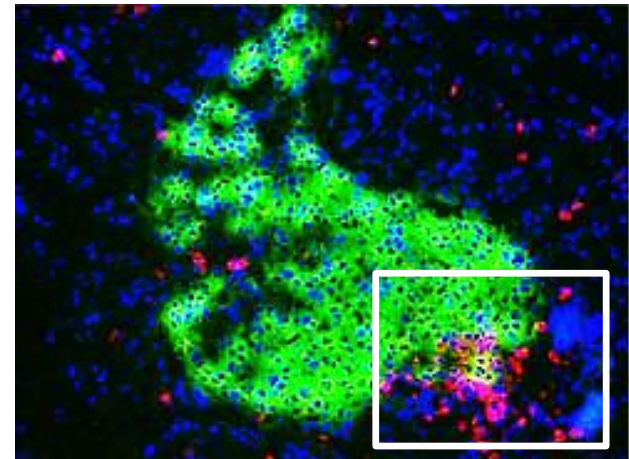


A First-in-Class Immunoregulatory CD34⁺ Cell Product Candidate to Induce Immune Tolerance



ToleraCyte™
Small molecule programmed
CD34⁺ cells

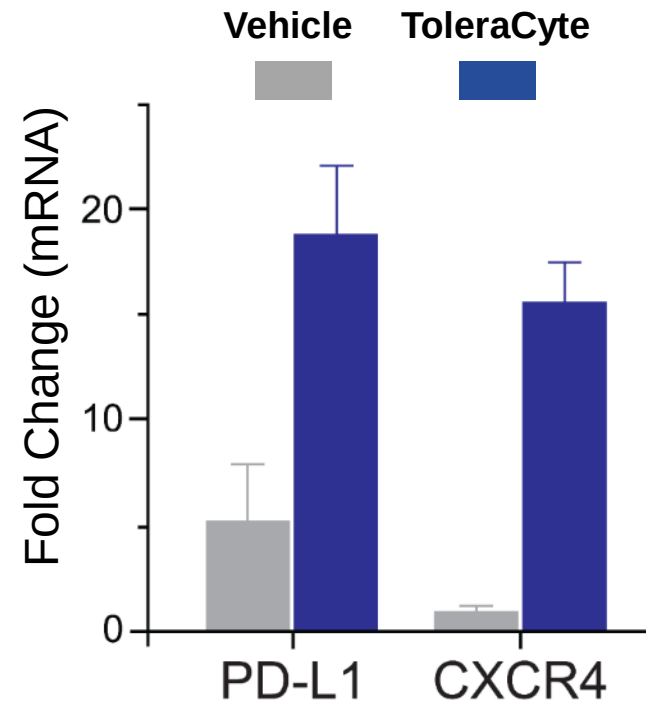
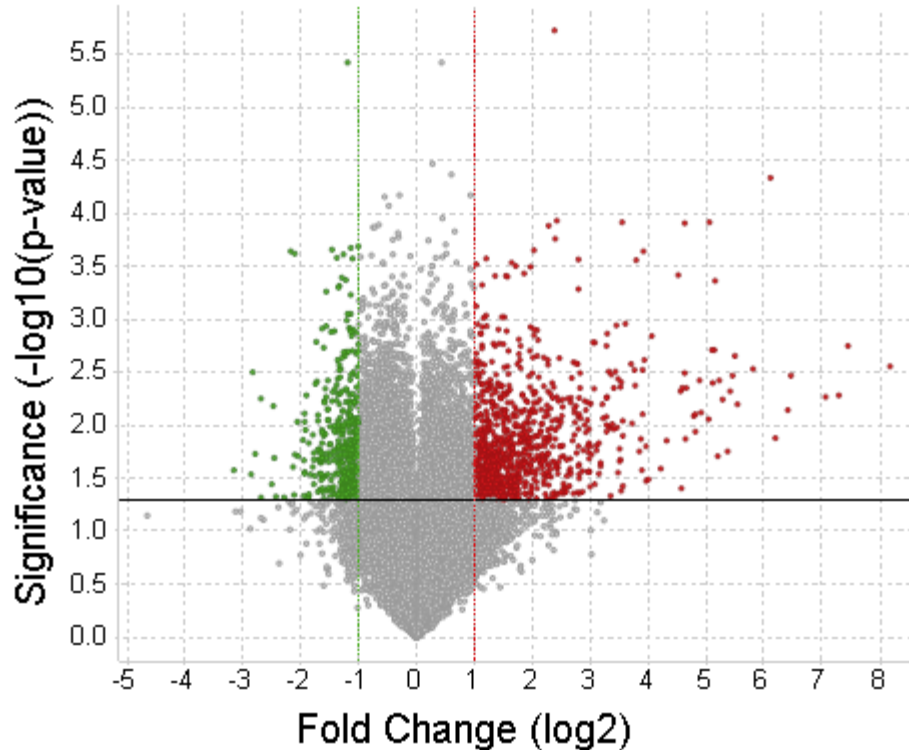
- **Autoimmune disorders result from malfunction of the body's natural defense systems**
 - Adaptive immune system (e.g., autoreactive T cells) mistakenly recognizes healthy cells as foreign and attacks and destroys the body's own tissue
 - 80+ autoimmune disorders estimated to affect ~50M in U.S.
 - 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)



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



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*

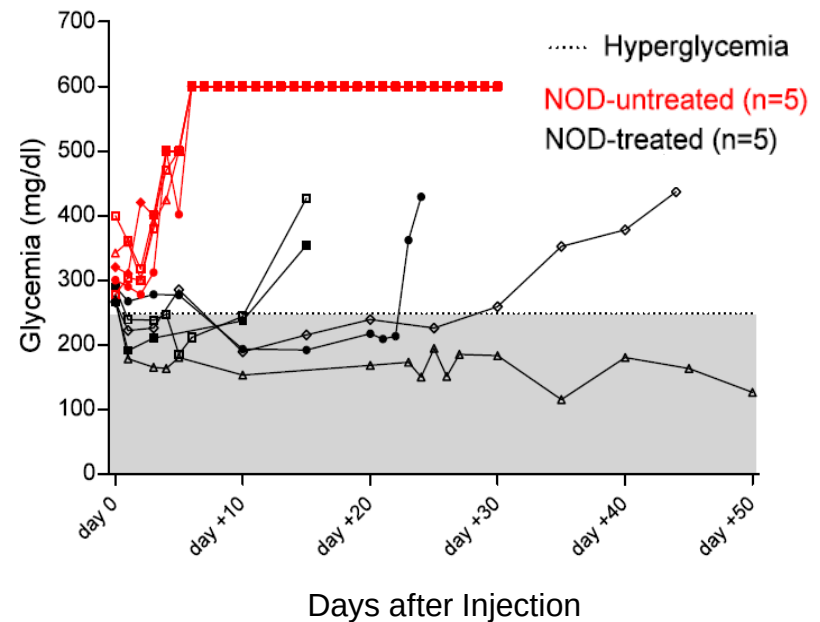


Boston Children's Hospital
Until every child is well™

- 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

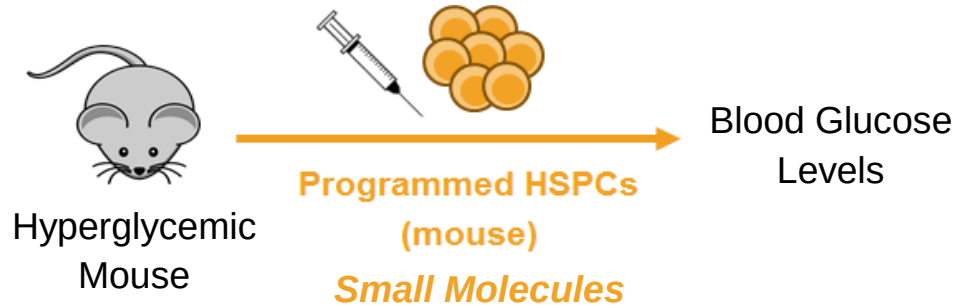


PD-L1 genetic overexpression or pharmacological restoration in hematopoietic stem and progenitor cells reverses autoimmune diabetes

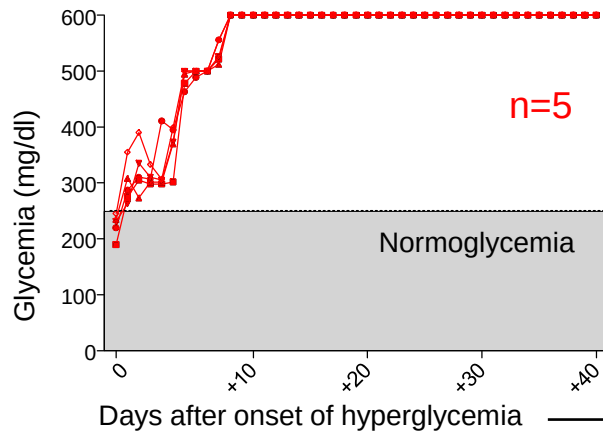
BY MOUFIDA BEN NASR, SARA TEZZA, FRANCESCA D'ADDIO, CHIARA MAMELI, VERA USUELLI, ANNA MAESTRONI, DOMENICO CORRADI, SILVANA BELLETTI, LUCA ALBARELLO, GABRIELLA BECCHI, GIAN PAOLO FADINI, CHRISTIAN SCHUETZ, JAMES MARKMANN, CLIVE WASSERFALL, LEONARD ZON, GIAN VINCENZO ZUCCOTTI, PAOLO FIORINA
SCIENCE TRANSLATIONAL MEDICINE | 15 NOV 2017 |

Immuno-Regulatory CD34⁺ Cell Therapy

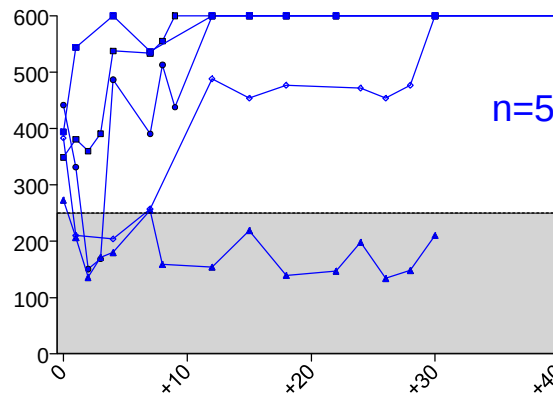
Durable Disease Correction in T1D Mouse Model



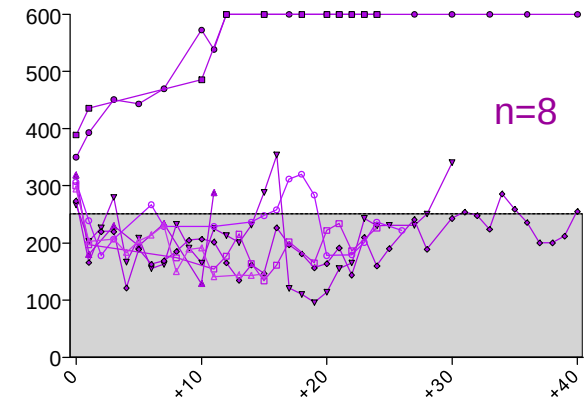
No Treatment



Vehicle-Treated HSPCs



Programmed HSPCs



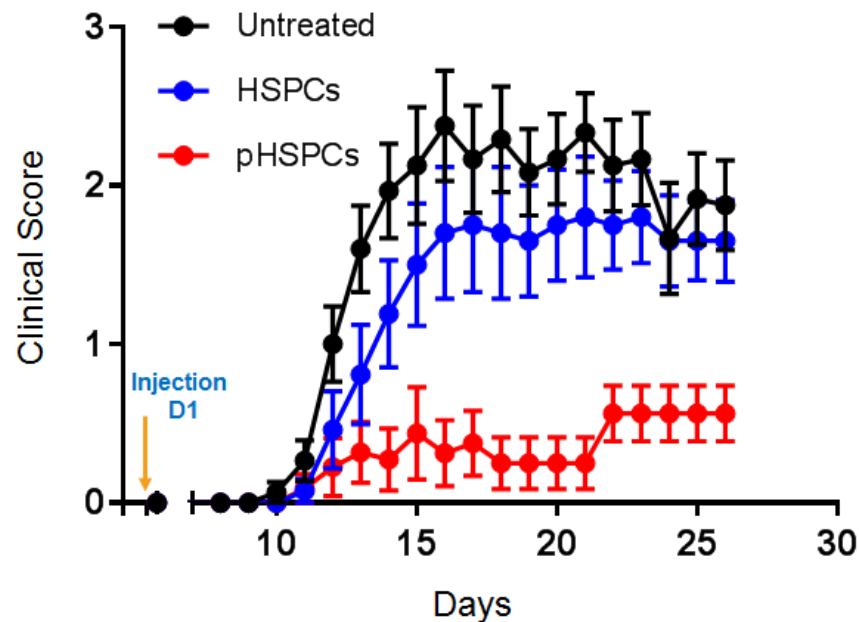
Immuno-Regulatory CD34⁺ Cell Therapy

Disease Attenuation in EAE Mice (Multiple Sclerosis)



Programmed HSPCs attenuate loss of motor function in EAE Mice

Loss of Motor Function



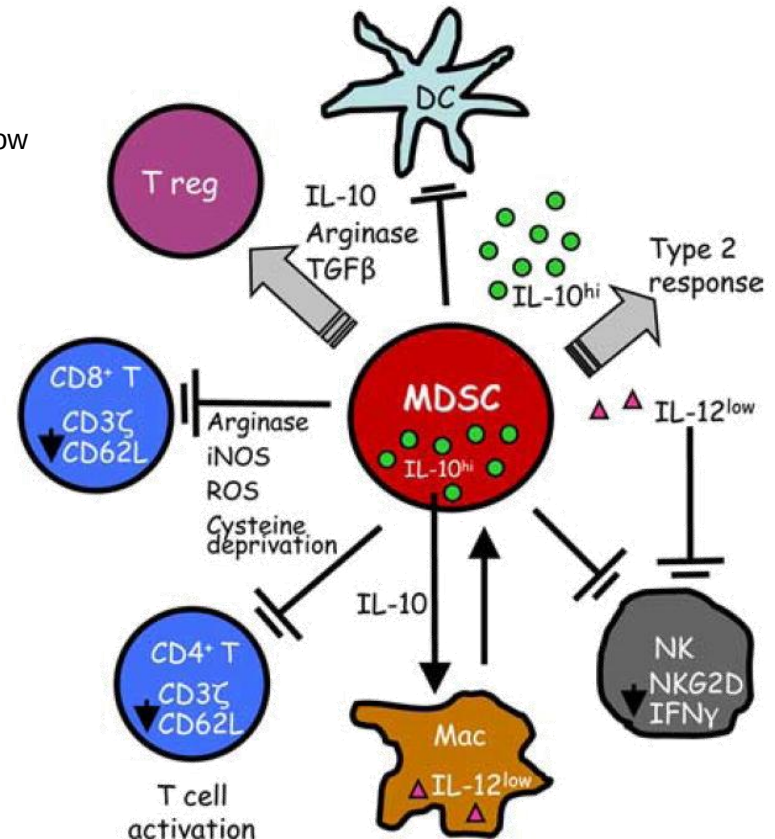


- **Builds on clinical precedent for CD34⁺ cell therapy**
 - Use of patient- and donor-sourced CD34⁺ cells has well-established safety record
- **Unique immuno-regulatory mechanism of action**
 - T-cell targeting approach through enhanced homing of programmed CD34⁺ cells to sites of inflammation
 - Robust 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

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



FT300

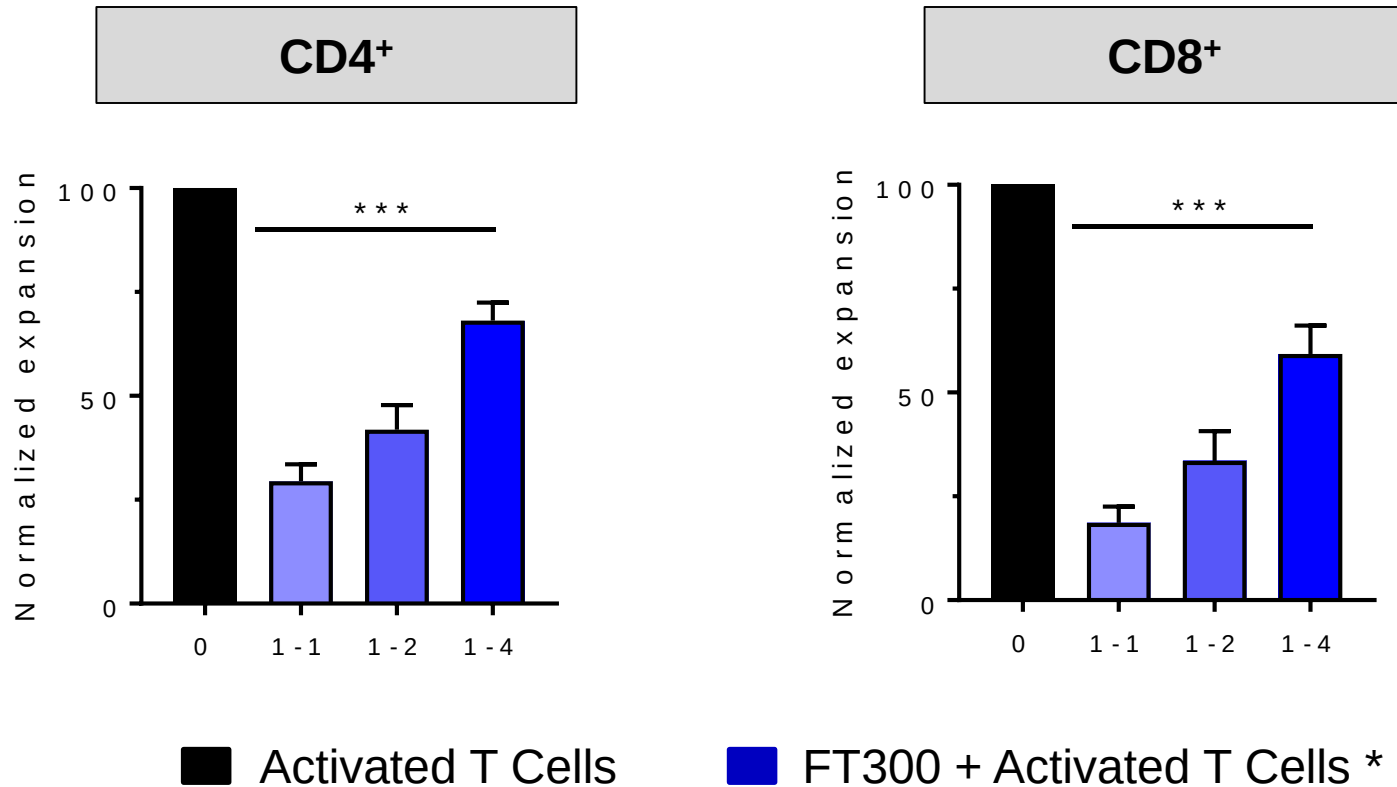
iPSC-derived MDSC Product



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

FT300 *In Vitro* Activity

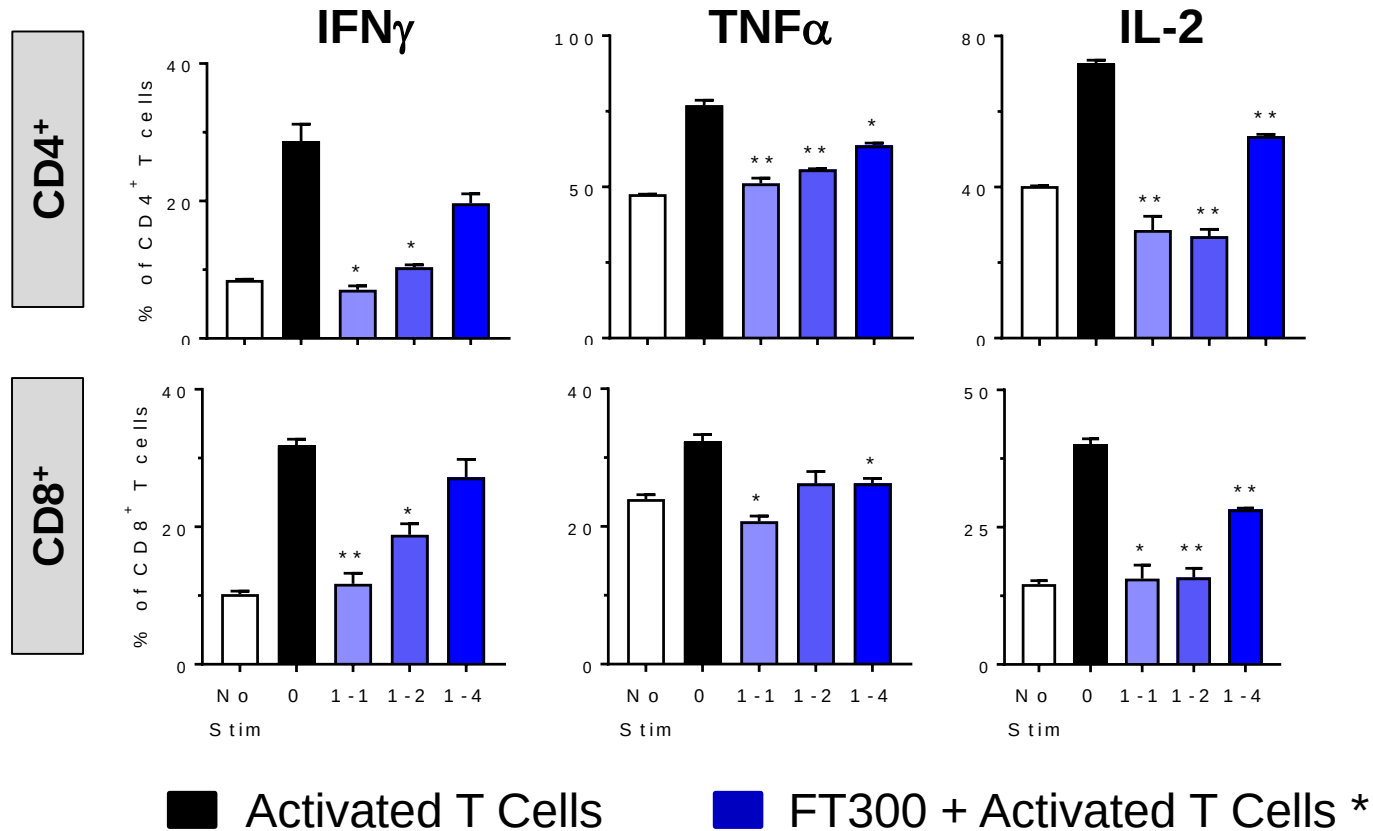
Potent Suppressor of T-Cell Proliferation Across HLA Barriers



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

FT300 *In Vitro* Activity

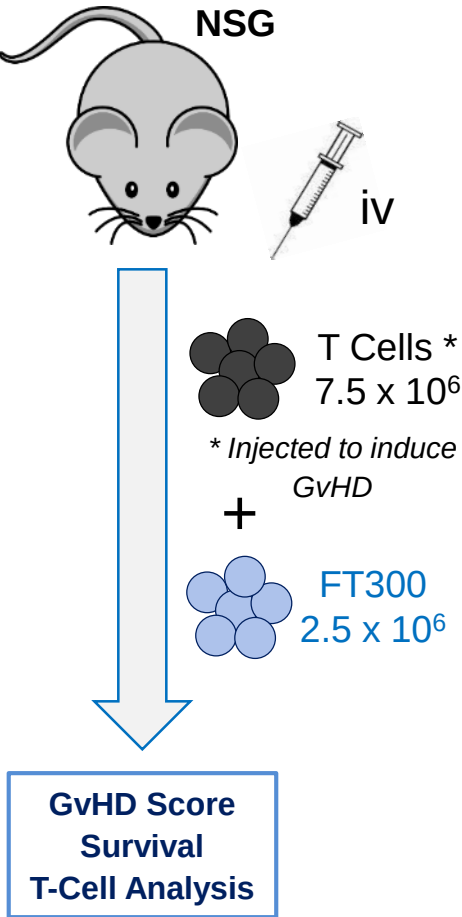
Potent Suppressor of T-Cell Cytokine Release Across HLA Barriers



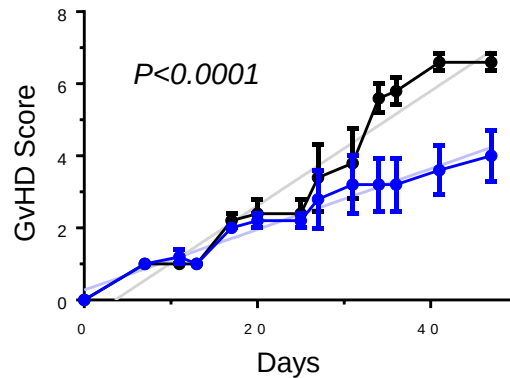
* Segmented by ratio of FT300 : Activated T Cells (5 independent donors for each ratio)

FT300 In Vivo Functionality

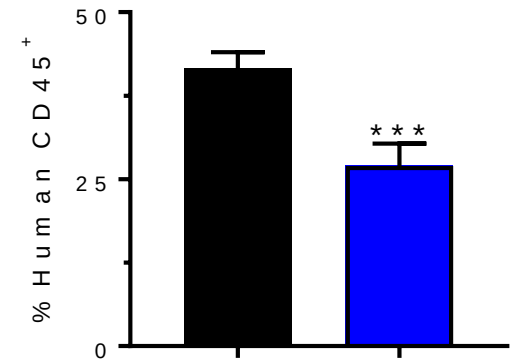
Suppression of T Cells and Disease Activity in GvHD Model



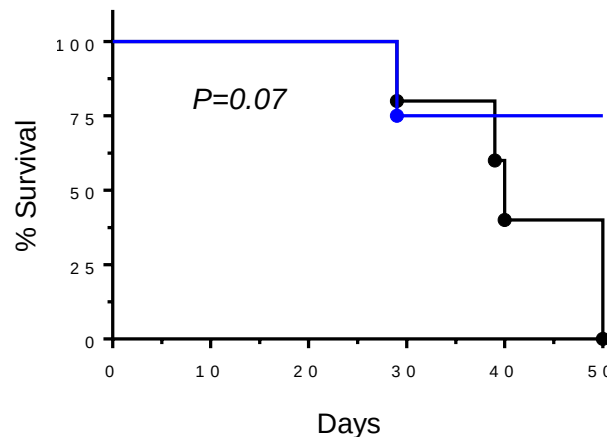
Disease Severity



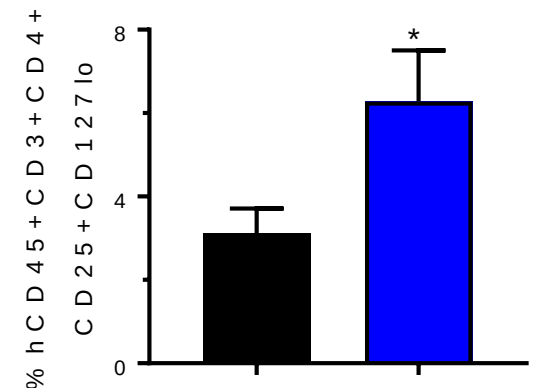
% T Cells



Survival



% Regulatory T Cells



■ T Cells

■ T Cells + FT300

FT300 Path to IND

Next Steps



- Conduct Pre-IND meeting with FDA
 - Already completed with oncology division for FT500 and FT516 (iPSC-derived NK cell products)
- Complete GMP manufacturing at Fate CMO
 - Already underway for FT500 and FT516
- Conduct GLP IND-enabling studies
- File IND
 - iPSC product platform to be de-risked through filing of INDs for FT500 and FT516



Financial Summary

Fate Therapeutics

Financial Summary



Three Months Ended September 30, 2017 ¹	
Revenue	\$1.0M
R&D Expense	\$8.6M
G&A Expense	\$2.8M
Operating Expense, Adjusted ²	\$10.0M
Cash & Cash Equivalents	\$112.2M
Employees	76
Total Shares Outstanding ³	66.7M

[1] Pro forma for Dec 2017 common stock financing.

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

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

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	<i>Checkpoint Inhibitor Combination</i>	Worldwide
FT516 (Engineered hnCD16 iNK Cell)	OTS	<i>Monoclonal Antibody Combination</i>	Worldwide
FT538 (Engineered CD38- iNK Cell)	OTS	<i>Daratumumab Combination</i>	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



Better Cells For Better Therapies™