Fate THERAPEUTICS

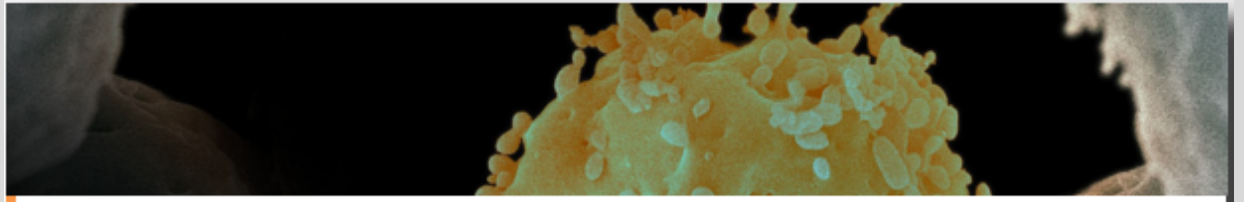
Better Cells For Better Therapies™

Off-the-shelf Cell-based Cancer Immunotherapy

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

2019 ASH Dinner Discussion

December 6, 2019



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Better Cells For Better Therapies™

Join Us for Dinner During the ASH Annual Meeting

Friday, December 6, 2019
7:00 - 9:00pm

Hyatt Regency Orlando
9801 International Drive
Orlando, FL 32819

RSVP by November 29
Michael Horowicz
michael.horowicz@sternir.com
212.362.1200

Initial clinical data of FT500, first-ever iPSC-derived cell therapy to undergo U.S. clinical investigation, to be highlighted

Special Guest Speakers

Jeffrey S. Miller, MD
Deputy Director, Masonic Cancer Center
Director, Cancer Experimental Therapeutics Initiative (CETI), University of Minnesota

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

Eric Smith, MD, PhD
Director of Clinical Translation, Cellular Therapeutics Center, Memorial Sloan Kettering Cancer Center

ASH Oral Presentations

FT538: Preclinical Development of an Off-the-Shelf Adoptive NK Cell Immunotherapy with Targeted Disruption of CD38 to Prevent Anti-CD38 Antibody-Mediated Fratricide and Enhance ADCC in Multiple Myeloma When Combined with Daratumumab
Saturday, December 7, 2019, 9:30 AM, W415A

FT596: Translation of First-of-Kind Multi-Antigen Targeted Off-the-Shelf CAR-NK Cell with Engineered Persistence for the Treatment of B Cell Malignancies
Saturday, December 7, 2019, 4:00 PM, W415A

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 research and development activities and its progress, plans and timelines for its manufacture, preclinical development and clinical investigation of its product candidates, the timing for the Company's receipt of data from its clinical trials and preclinical studies, the Company's clinical development and regulatory strategy, and the therapeutic and market potential of the Company's product candidates. These and any other forward-looking statements in this presentation are based on management's current expectations of future events and are subject to a number of risks and uncertainties that could cause actual results to differ materially and adversely from those set forth in or implied by such forward-looking statements. These risks and uncertainties include, but are not limited to, the risk that results observed in prior studies of its product candidates will not be observed in ongoing or future studies involving these product candidates, the risk of a delay in the initiation of, or in the enrollment or evaluation of subjects in, any clinical studies, and the risk that the Company may cease or delay manufacture, or preclinical or clinical development, of any of its product candidates for a variety of reasons (including regulatory requirements, difficulties in manufacturing or supplying the Company's product candidates, and any adverse events or other negative results that may be observed during preclinical or clinical development). These statements are also subject to other risks and uncertainties as further detailed in the Company's most recently filed periodic report, and subsequent periodic reports filed by the Company, under the Securities Exchange Act of 1934, as amended, any of which could cause actual results to differ materially from those contained in or implied by the forward-looking statements in this presentation. The Company is providing the information in this presentation as of the date hereof and does not undertake any obligation to update any forward-looking statements contained in this presentation unless required by applicable law.

Fate THERAPEUTICS

Introduction

2019 – A Break-through Year for the FATE iPSC Product Platform

Feb 2019 – First-ever Patient Treated with an iPSC-derived Cell Therapy in U.S.

UC San Diego
MOORES CANCER CENTER

FT500

From left: Sandip Patel, MD; Dan Kaufman, MD, PhD; Derek Ruff

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2019 – A Break-through Year for the FATE iPSC Product Platform

Oct 2019 – First-ever Patients Treated with Cell Therapy derived from a Clonal Master Engineered iPSC Line

University of Minnesota opens first-ever U.S. clinical trial of engineered iPSC-derived cell therapy for blood cancers

UNIVERSITY OF MINNESOTA
Driven to Discover℠

MINNEAPOLIS, MN- October 21, 2019 - A new cancer clinical trial has opened at the M Health Fairview University of Minnesota Medical Center that leverages the groundbreaking research on stem cells and natural killer (NK) cells done at the Masonic Cancer Center and applies it to attack acute myeloid leukemia (AML) and B-cell lymphoma. The first-of-its-kind NK cell cancer immunotherapy, called FT516, is manufactured from a human induced pluripotent stem cell (iPSC) that has been genetically engineered to enhance its anti-tumor activity.

Claudio Brunstein, MD, PhD

The first-in-human clinical trial of FT516, sponsored by Fate Therapeutics, will be run locally by Claudio Brunstein, MD, PhD, who is a professor of Medicine at the U of M Medical School, a member of the Masonic Cancer Center, and the medical director of the Adult Blood and Marrow Transplant and Cellular Therapy Program at M Health Fairview.

"We potentially have an unlimited source of very similar, reproducible cancer fighters," said Brunstein. "This is opening a whole new door in cellular therapy. With increased modifications to these NK cells, we can elevate their ability to attack tumors. As we add more functionality to NK cells, we have the potential to bring together multiple anti-tumor mechanisms and more effectively target and kill cancer."

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2019 – A Break-through Year for the FATE iPSC Product Platform

Sept 2019 – Secured FDA Clearance of IND Application for First-ever Cell Therapy Engineered with Three Anti-Tumor Modalities

Fate Therapeutics Announces the Opening of its cGMP Manufacturing Facility Dedicated to iPSC-derived Cell Therapies

State-of-the-Art Facility Designed to use Clonal Master iPSC Lines as Renewable Cell Source for Manufacture of Off-the-Shelf Product Pipeline

San Diego, CA – September 30, 2019 – Fate Therapeutics, Inc. (NASDAQ: FATE), a clinical-stage biopharmaceutical company dedicated to the development of programmed cellular immunotherapies for cancer and immune disorders, announced today that the Company has opened its current Good Manufacturing Process (cGMP) compliant manufacturing facility for the clinical production of its off-the-shelf natural killer (NK) cell and chimeric antigen receptor (CAR) T-cell product candidates. The

- *Completed GMP production of FT596 at FATE facility in November*
- *Single "small-batch" manufacturing campaign yielded ~320 cryopreserved, infusion-ready doses*
- *Estimated actual cost per dose: <$2,500*

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2019 – A Break-through Year for the FATE iPSC Product Platform

Aug 2019 – Issuance of Foundational U.S. Patent Covering iPSC-derived CAR T Cells

United States Patent
Themeli et al.

Patent No.: US 10,370,452 B2
Date of Patent: Aug. 6, 2019

EFFECTIVE GENERATION OF TUMOR-TARGETED T CELLS DERIVED FROM PLURIPOTENT STEM CELLS

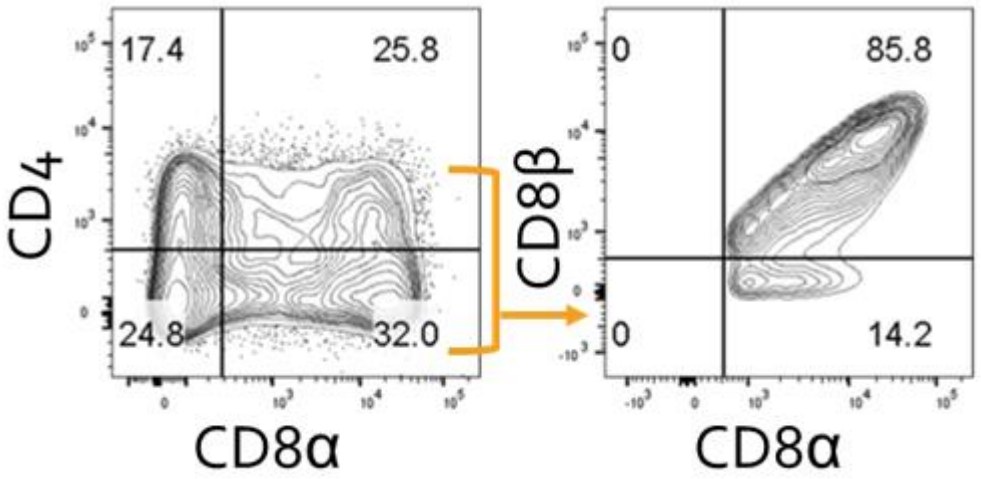
Applicant: **MEMORIAL SLOAN-KETTERING CANCER CENTER**, New York, NY (US)

Inventors: **Maria Themeli**, New York, NY (US); **Michel Sadelain**, New York, NY (US); **Christopher C. Kloss**, New York, NY (US)

Assignee: **MEMORIAL SLOAN-KETTERING CANCER CENTER**, New York, NY (US)

Claim 1. A population of T cells that are produced by in vitro differentiation of a pluripotent stem cell, wherein (i) the pluripotent stem cell expresses a chimeric antigen receptor (CAR), and (ii) the population of T cells comprises a T cell exhibiting a CD45RA+ CD27- CD28- CCR7- CD62L- phenotype.

iPSC-derived CAR T-cell Phenotype



- ***Priority Date = April 3, 2013***
- ***Publication Date = October 9, 2014***

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2019 – A Break-through Year for the FATE iPSC Product Platform

Dec 2019 – No Morphologic Evidence of Leukemia, with Complete Neutrophil Recovery, Observed in First Patient Treated with FT516 Monotherapy for AML

41 year old male diagnosed with AML in January 2019

- Refractory to initial induction therapy and multiple additional lines of therapy

Enrolled in FT516 Study (Oct 2019)

- Early assessment following first three doses of FT516 with IL-2 cytokine support showed:
  - No morphologic evidence of leukemia, with evidence of hematopoietic recovery, in bone marrow
  - No circulating leukemic blasts in peripheral blood
  - Recovery of neutrophils (>1,000 per µL)
  - No observed CRS, neurotoxicity or GvHD
  - FT516 chimerism detected *in the bone marrow* at Day 18 by digital PCR

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iPSC Product Platform

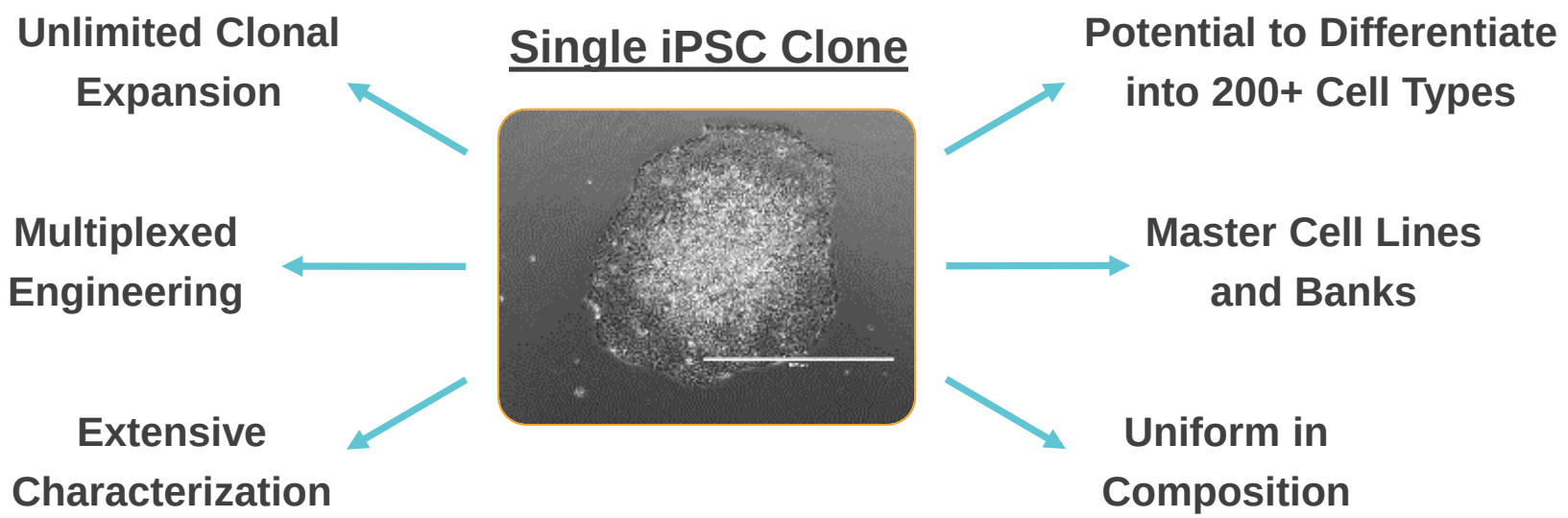
First-ever Patients in U.S. Treated with an iPSC-derived Cell Product!

Unique Advantages of Human iPSCs

Single-cell Isolation, Characterization & Selection

A Single Human Induced Pluripotent Stem Cell (iPSC)

A renewable source for making cell products



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

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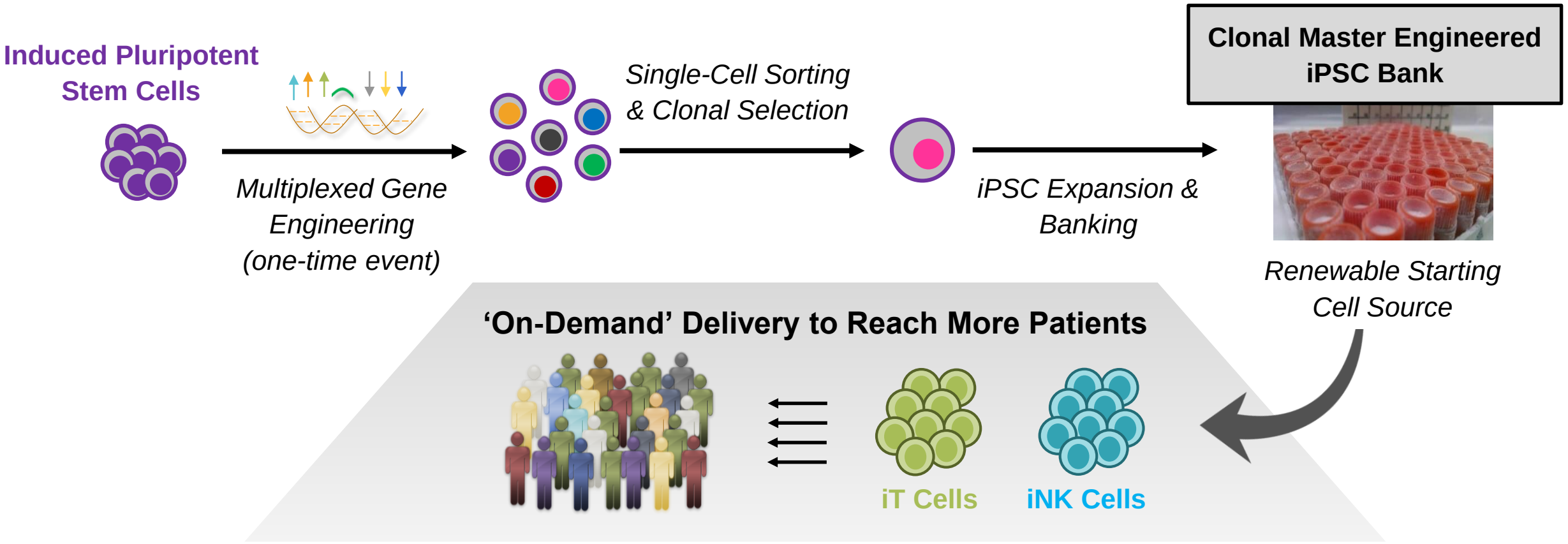
iPSC Product Platform

Disruptive Approach Enabling Mass Production of Universal NK Cell and T-Cell Products



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

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FT500 Off-the-Shelf NK Cell Product Candidate

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

FT500 Product Candidate

FT500

(non-engineered)

- ***IND submitted to FDA in July 2018***
- ***IND cleared by FDA in November 2018***
- ***First patient treated in February 2019***

Key Questions to Address

- Can we make *bona fide* NK cells?
- What is the comparable functionality of iNK cells?
- Can we cost-effectively manufacture iNK cells under GMP conditions?
- What is the clinical safety of an iPSC-derived cell therapy?
- Can multiple doses of an unmatched iPSC-derived cell therapy be tolerated without rejection?

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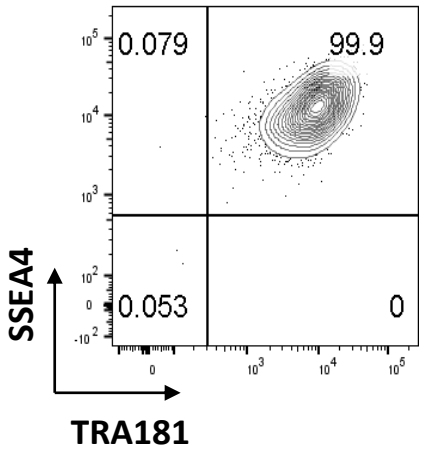
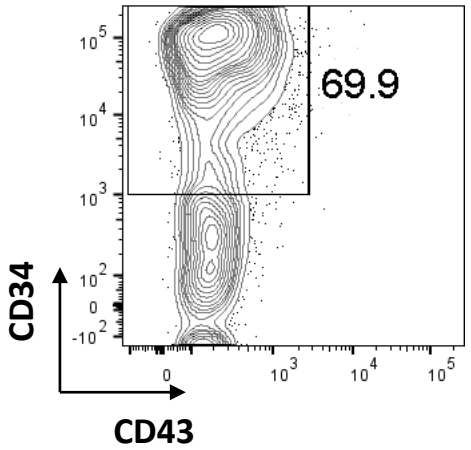
The Making of *Bona Fide* NK Cells from a Master iPSC Bank

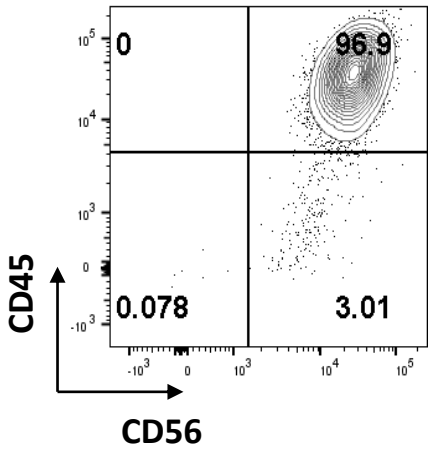
Robust cGMP Process

iPSCs → **iCD34s** → **iNKs**

Day 0 ***Day 10*** ***Day 44***







> 1 million-fold expansion

10^6 iPSCs

> 10^{12} iNKs

100s – 1,000s of doses of cryopreserved, infusion-ready iNK cells

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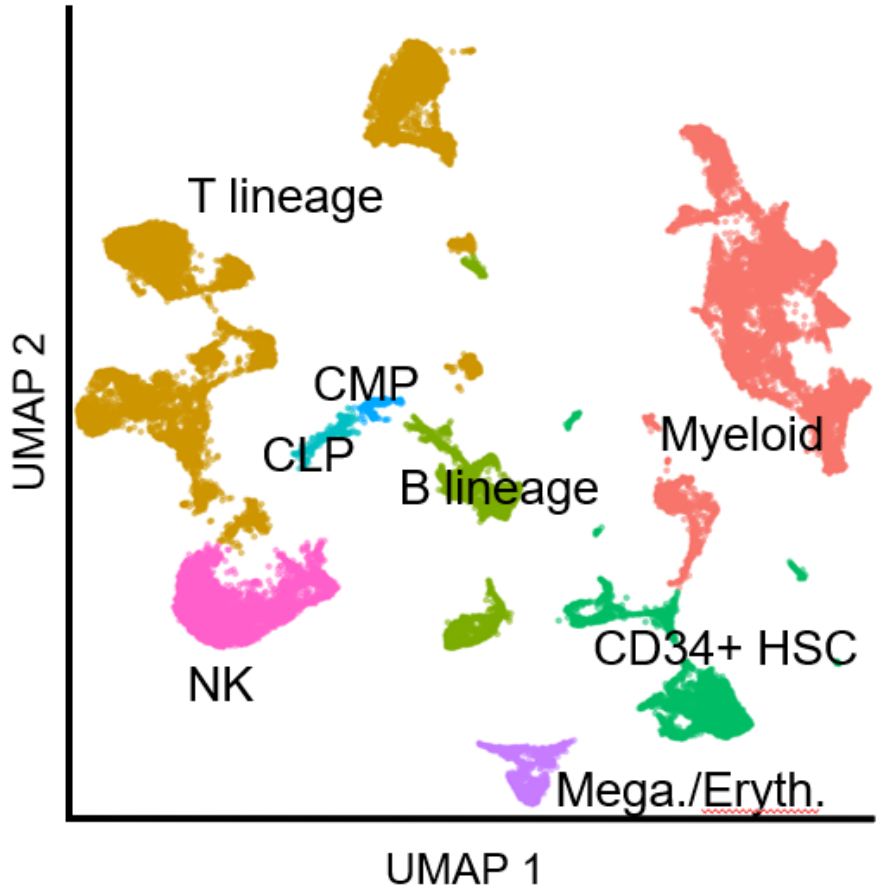
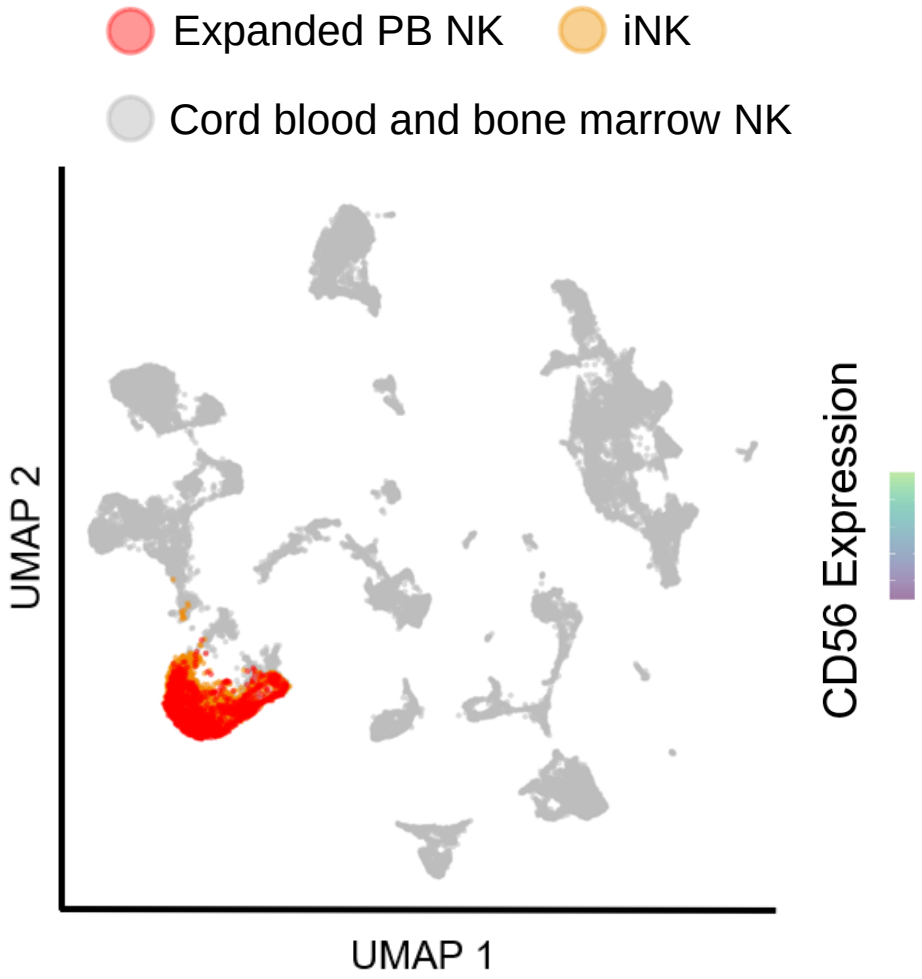
The Making of *Bona Fide* NK Cells from a Master iPSC Bank

Gene Expression Analysis Confirms iNK Cells Cluster with Peripheral Blood NK Cells

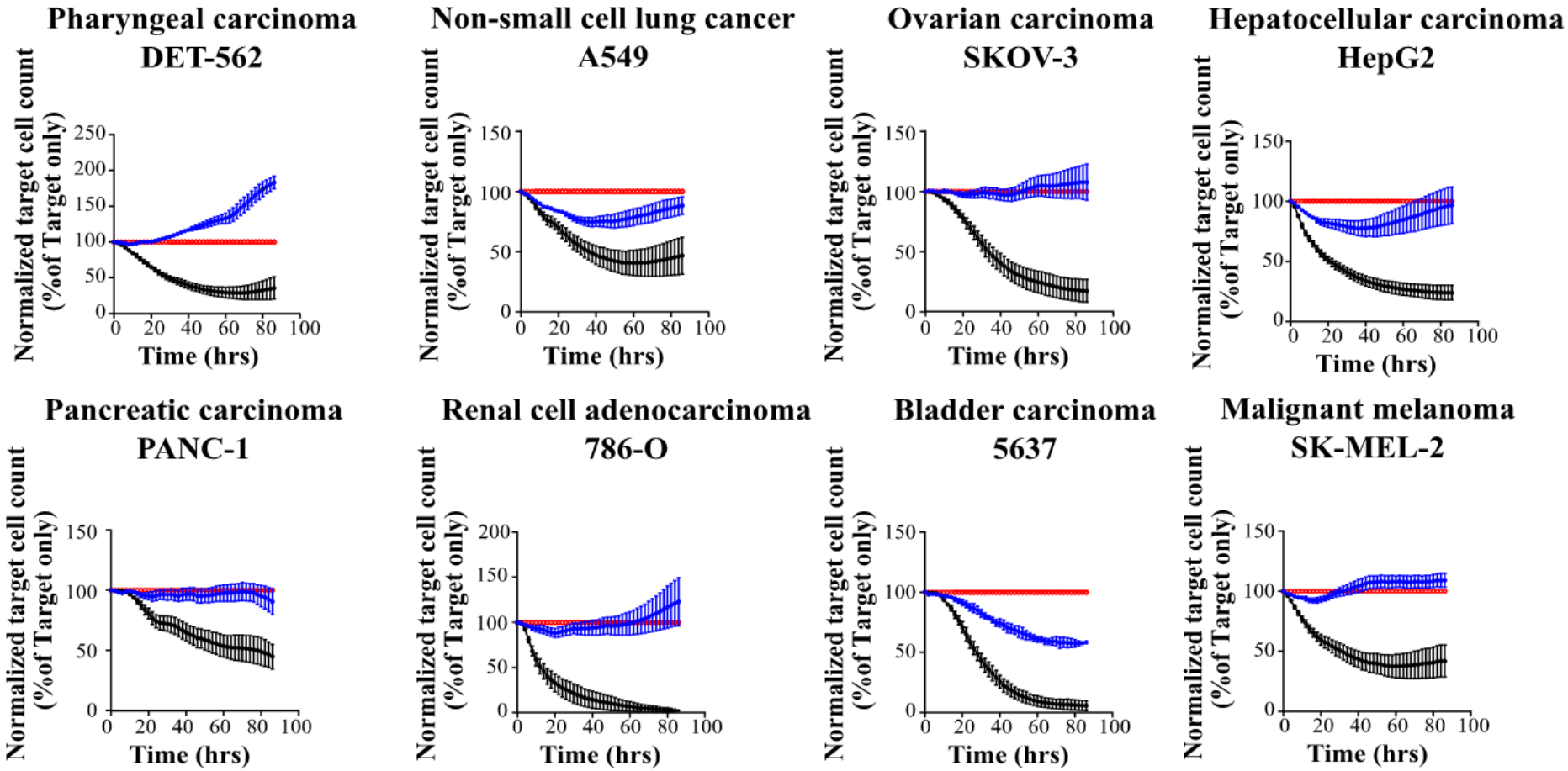
Immune Cell Atlas
+ Adult NK Cells + FATE iNKs





Enhanced Functionality of iNK Cells

Increased In Vitro Cytotoxicity vs. Healthy Donor Peripheral Blood NK Cells



Target Cells ***Donor NK Cells (fresh)*** ***iNK Cells (cryopreserved)***

Cost-Effective GMP Manufacture of FT500 iPSC-derived NK Cells

Unprecedented Purity and Post-thaw Viability

cGMP Manufacture Run Specifications

FT500 Cell Product	
Identity, CD45+	100%
Identity, CD45+CD56+	98%
Post-thaw Viability	80%
Residual iPSCs	Not detected
Packaging	Cryopreserved
Storage	Clinical sites
Administration	Thaw-and-infuse 'on demand'
Delivery	Outpatient setting

Yield & Cost for Small-scale GMP Campaign

Estimated Costs for MCT FT500 Clinical Campaign	
Supplies & Reagents	$367,292
Fairview MCT Labor & Services	54,389
QC Testing & QA Validation	70,300
MCT GMP Suite Occupancy	177,763
Sub-total	$669,744
Overhead	171,836
Total Cost	$841,580
Approx. Unit Cost	**$3,000**

One 44-day GMP campaign yielded ~300 doses at a cost of ~$3,000 per dose

- *Homogeneous cell product*
- *Cryopreserved in infusion media*
- *High post-thaw viability*

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FT500 Phase 1 – First-ever U.S. Clinical Study of iPSC-derived Cell Product

Phase 1 Dose Escalation Clinical Objectives

Assessment of Safety & Tolerability as Monotherapy and in Combination with Checkpoint Inhibitor

Assess Novel Paradigm

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

Key Clinical Read-outs

- FT500 safety and tolerability (DLTs, AEs)
- Immune-mediated toxicities (GvHD, CRS)

Key Molecular Read-outs

- Immune cell recovery
- Endogenous cytokine response (GvHD, CRS)
- Anti-product immunogenicity

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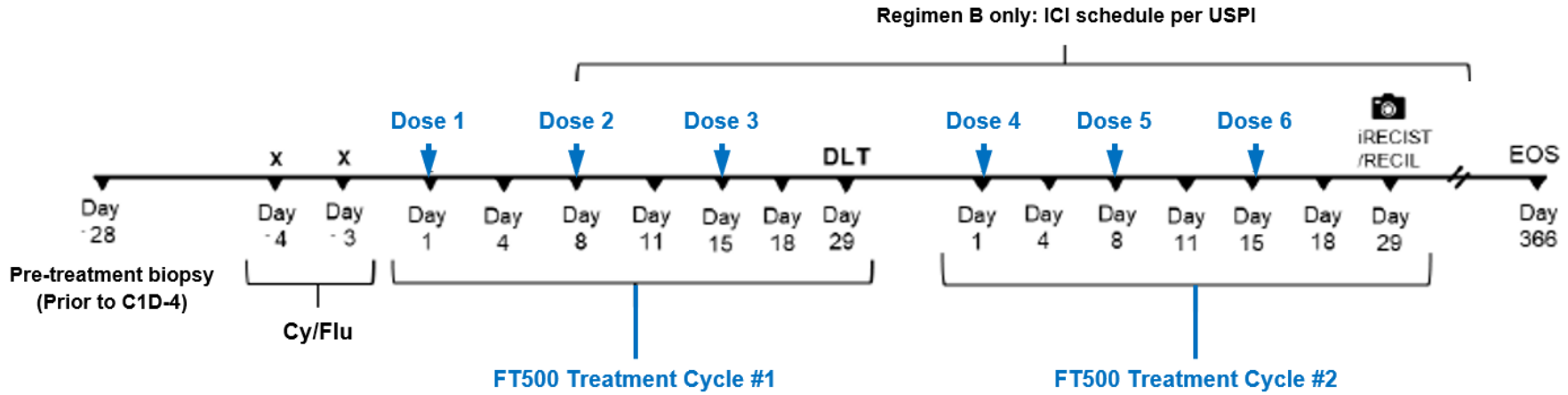
FT500 Phase 1 Dose Escalation – Study Schema

Designed to Assess Safety and Tolerability of a Multi-dose, Multi-cycle Treatment

Advanced Solid Tumors as a Monotherapy and in Combination with ICI Therapy



Treatment Paradigm	Regimen A	Regimen B
➢ Mild lympho-conditioning: Flu (25 mg/m^2), Cy (300 mg/m^2) x 2 days prior to Cycle 1 only	➢ Salvage setting	➢ Salvage setting
➢ Up to 6 doses in outpatient setting	➢ Relapsed/refractory disease	➢ Relapse/refractory from ICI therapy
➢ **No cytokine support**	➢ No available approved therapies	➢ Combine with prior ICI therapy

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FT500 Phase 1 Dose Escalation – Patient Baseline Characteristics

Heavily Pretreated Patients with Refractory Disease

Cohort / Cell Dose	Age / Sex	Tumor Type	# Lines of Prior Therapy	Refractory to Last Prior Therapy
A1 100M cells / dose	54 / M	Colon	3	Yes
	57 / M	Metastatic salivary gland carcinoma	2	No
	61 / F	Ovary	6	Yes
A2 300M cells / dose	43 / M	Colon	4	No
	52 / F	Colorectal	1	No
	57 / M	Squamous cell carcinoma, left tonsil	2	Yes
	62 / M	Floor of mouth cancer	4	No
	53 / F	Pancreas	4	Yes
B1 100M cells / dose	59 / F	Non-small cell lung cancer	7	Yes
	54 / F	Non-small cell lung cancer	4	Yes
	61 / M	Hepatocellular carcinoma	2	Yes
B2 300M cells / dose	71 / F	Primary peritoneal mesothelioma	2	Yes

As of 28 November 2019 data cutoff. Database is not locked and final data are subject to change.

FT500 Phase 1 Dose Escalation – Key Clinical Read-outs

Regimen A Monotherapy – Safety, Tolerability, Best ORR, and Disposition

Cohort / Cell Dose	Subject #	# Lines of Prior Therapy	FT500 Doses Received	Safety			Best Overall Response *	Disposition	
				Dose Limiting Toxicities	Related Grade ≥ 3 AEs	Related SAEs		Days on Study	Reason for Study Discontinuation
A1 100M cells / dose	1	3	6	None	None	None	SD	94	Clinical Progression
	2	2	6	None	None	None	iUPD	94	iCPD
	3	6	6	None	None	None	SD	83	iUPD
A2 300M cells / dose	1	4	6	None	None	None	SD	70	iUPD
	2	1	5	None	None	None	SD	55	Clinical Progression
	3	2	3	None	None	None	iUPD	33	iUPD
	4	4	6	None	None	None	iUPD	72	Clinical Progression
	5	4	6	None	None	None	iUPD	90	iCPD

* Per iRECIST SD = stable disease iUPD = immune unconfirmed progressive disease iCPD = immune confirmed progressive disease

As of 28 November 2019 data cutoff. Database is not locked and final data are subject to change.

FT500 Phase 1 Dose Escalation – Key Clinical Read-outs

Regimen B ICI Combination – Safety, Tolerability, Best ORR, and Disposition

Cohort / Cell Dose	Subject #	# Lines of Prior Therapy	FT500 Doses Received	Safety			Best Overall Response *	Disposition	
				Dose Limiting Toxicities	Related Grade ≥ 3 AEs	Related SAEs		Days on Study	Reason for Study Discontinuation
B1 100M cells / dose	1	7	3	None	None	None	SD	76	Patient decision
	2	4	6	None	None	None	SD	98	iUPD
	3	2	6	None	None	None	iUPD	85	iCPD
B2 300M cells / dose	1	2	3 (ongoing)	None	None	None	Pending	On-study	

* Per iRECIST SD = stable disease iUPD = immune unconfirmed progressive disease iCPD = immune confirmed progressive disease

As of 28 November 2019 data cutoff. Database is not locked and final data are subject to change.

FT500 Phase 1 Dose Escalation – Key Clinical Read-outs

Multi-dose, Multi-cycle Regimen: Favorable Safety, Well-tolerated Treatment

As of a November 28, 2019 data cutoff:

- All patients received ≥3 doses of FT500 in outpatient setting
- No DLTs
- No FT500-related SAEs or Grade ≥3 AEs
- No immune-related AEs (e.g., CRS, neurotoxicity, or GVHD)
- No treatment discontinuations due to AEs

Currently enrolling 300M cells / dose in combination with immune checkpoint inhibitors

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As of 28 November 2019 data cutoff. Database is not locked and final data are subject to change.

Translational Research Objectives for First-ever iPSC-Derived Therapy in U.S.

Assess Patient's Immunological Response to Multiple Doses of Off-the-Shelf Cell Therapy

FT500 is a Universal, Off-the-Shelf NK Cell Cancer Immunotherapy administered with Multiple Doses to Patients without Matching

- Immune cell recovery
  - Do iNK cells negatively impact hematopoietic recovery following lympho-conditioning?
- Endogenous cytokine response
  - Is there molecular evidence of immunotoxicity (e.g., CRS, neurotoxicity and/or GvHD)?
- Anti-product immunogenicity
  - T-cell mediated: Do anti-product T-cell clones expand and become dominant?
  - B-cell mediated: Are anti-product antibodies raised?

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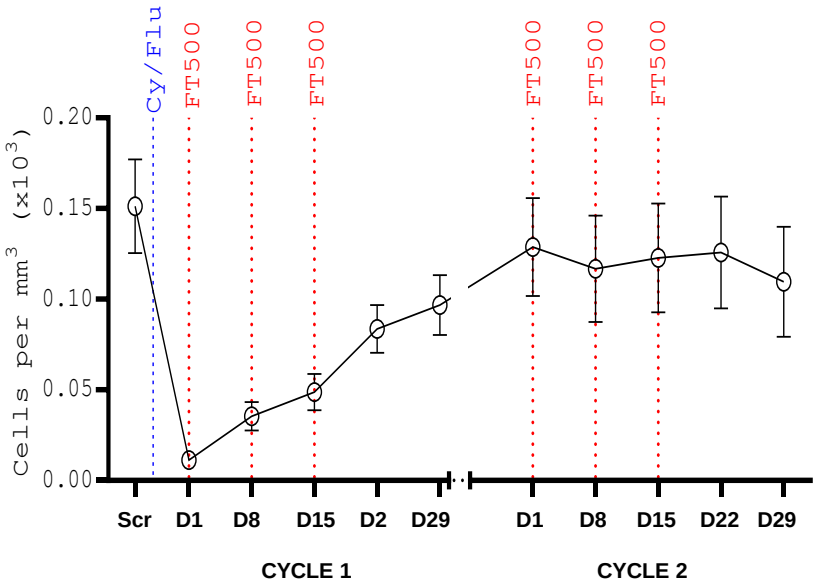
FT500 Phase 1: Clinical Translation

Immune Cell Recovery

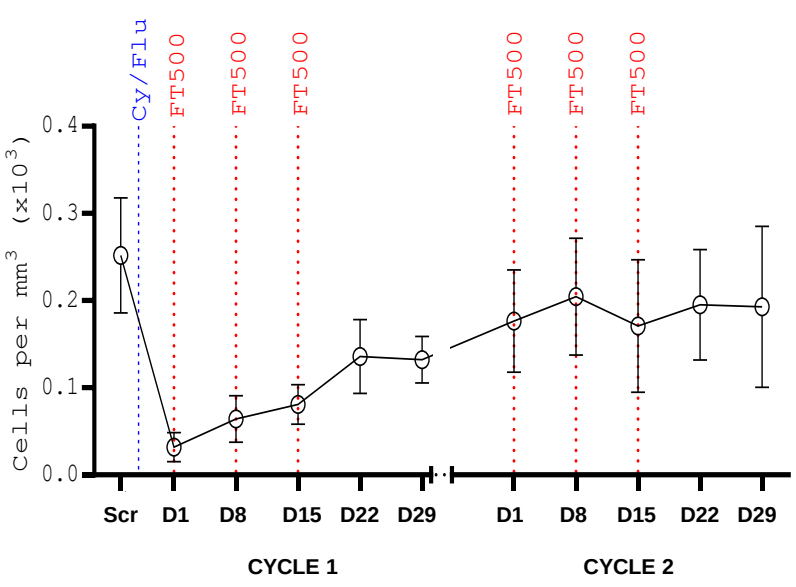
Healthy Immune Reconstitution following Multi-dose FT500 Treatment

Outpatient lympho-conditioning regimen: Cy (300 mg/m^2) x Flu (25 mg/m^2) x 2 days prior to Cycle 1 <u>only</u>

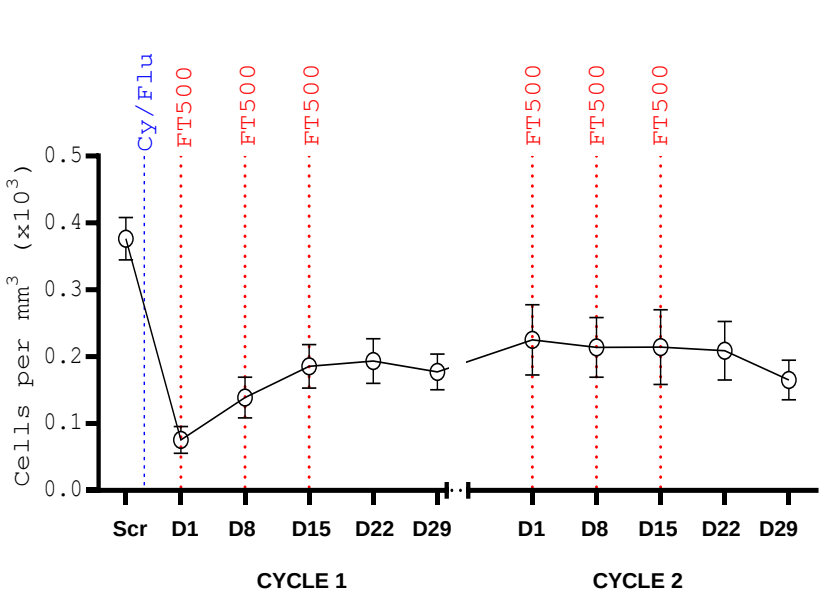
NK Cells



CD8+ T Cells



CD4+ T Cells



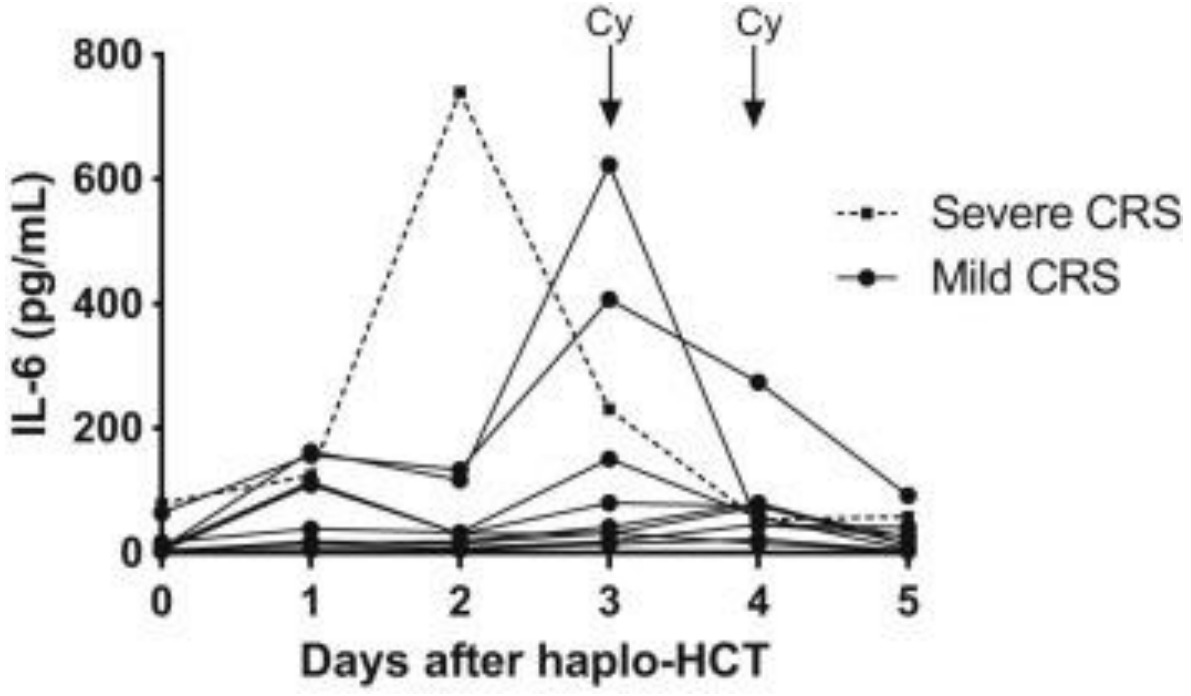
Mean +/- SD; N = 11

FT500 Phase 1: Clinical Translation

Endogenous Cytokine Response

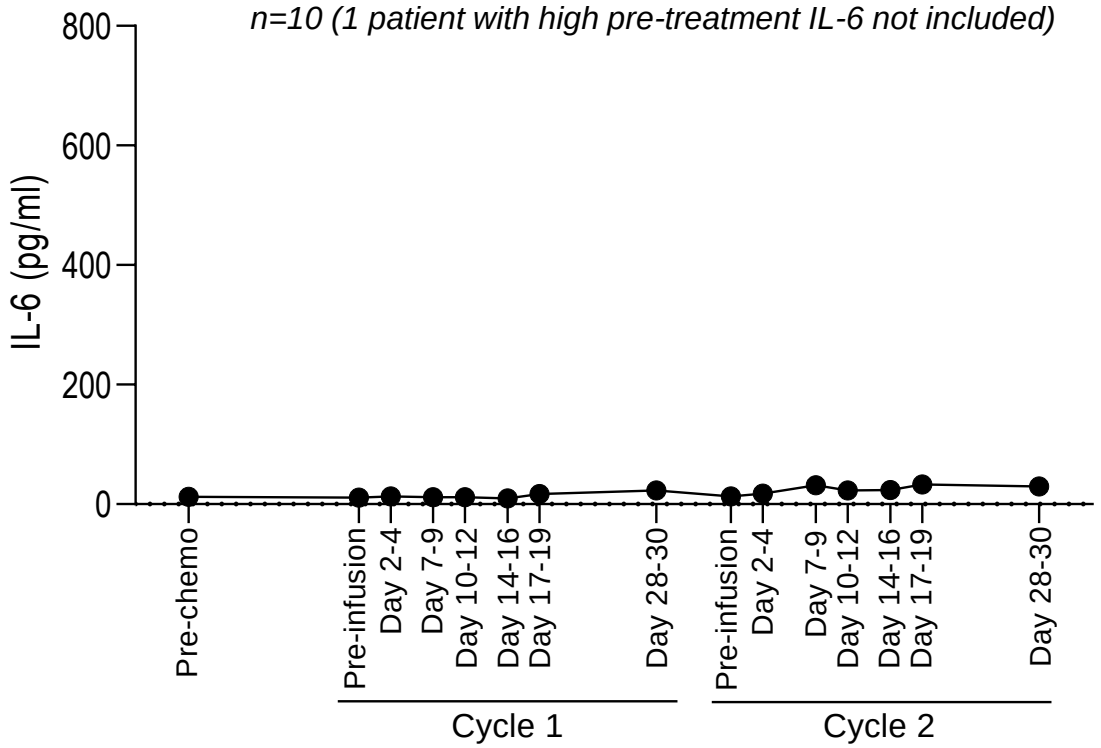
No Biomarker Evidence of Subclinical CRS, neurotoxicity, or GvHD

Severe CRS associated with IL-6 levels > 200 pg/ml



Abboud, Romey et al. BBMT 2016

FT500 IL-6 Cytokine Response



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FT500 Phase 1: Clinical Translation

Yellow Fever Vaccine: T-Cell Response following Successful Immunization

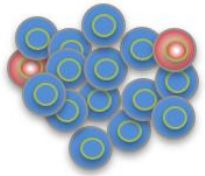
Dynamics of the Cytotoxic T Cell Response to a Model of Acute Viral Infection

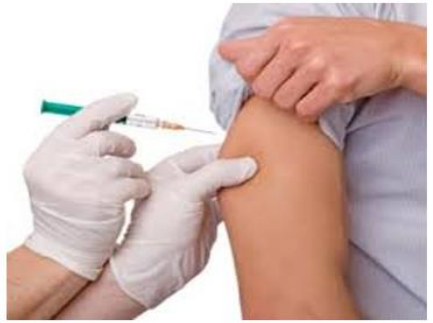
Journal of Virology April 2015 Volume 89 Number 8

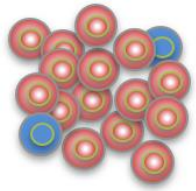
William S. DeWitt,[a] Ryan O. Emerson,[a] Paul Lindau,[b,c] Marissa Vignali,[a] Thomas M. Snyder,[a] Cindy Desmarais,[a] Catherine Sanders,[a] Heidi Utsugi,[b] Edus H. Warren,[b] Juliana McElrath,[b] Karen W. Makar,[b] Anna Wald,[c] Harlan S. Robins[a,b]

Adaptive Biotechnologies, Seattle, Washington, USA[a]; Fred Hutchinson Cancer Research Center, Seattle, Washington, USA[b]; University of Washington, Seattle, Washington, USA[c]

PBMCs







Expanded PBMCs
(Enriched for T cells with YFV-specific TCR)

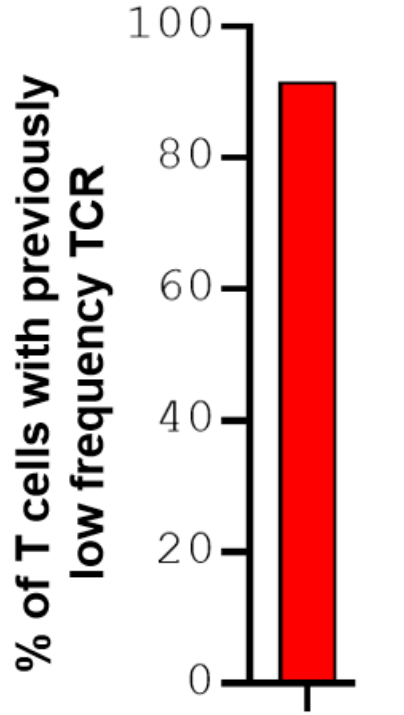
TABLE 2 Number of YFV-induced clones[a]

Presence or absence in T_{M-0}	No. of YFV-induced clones in subject no.: 1	2	3	4	5	6	7	8	9	Avg	Total (%)
+	139	241	36	139	426	163	57	181	256	182	1,638 (8.5)
−	2,303	2,126	3,804	2,010	1,618	1,764	1,538	1,653	757	1,953	17,573 (91.5)
Total (% absent)	2,442 (94.3)	2,367 (89.8)	3,840 (99.1)	2,149 (93.5)	2,044 (79.2)	1,927 (91.5)	1,595 (96.4)	1,834 (90.1)	1,013 (74.7)	2,135 (91.5)	21,346 (82.3)

[a] For each subject, the table shows the number of YFV-induced clones present (+) or absent (−) in the memory compartment on day 0 before vaccination (T_{M-0}), as well as the total number of YFV-induced clones identified and the percentage of those that were absent from T_{M-0}. The last two columns correspond to the aggregated data (average, total, and percentage) from all 9 subjects.

YFV Immunization

TCR Repertoire Shift



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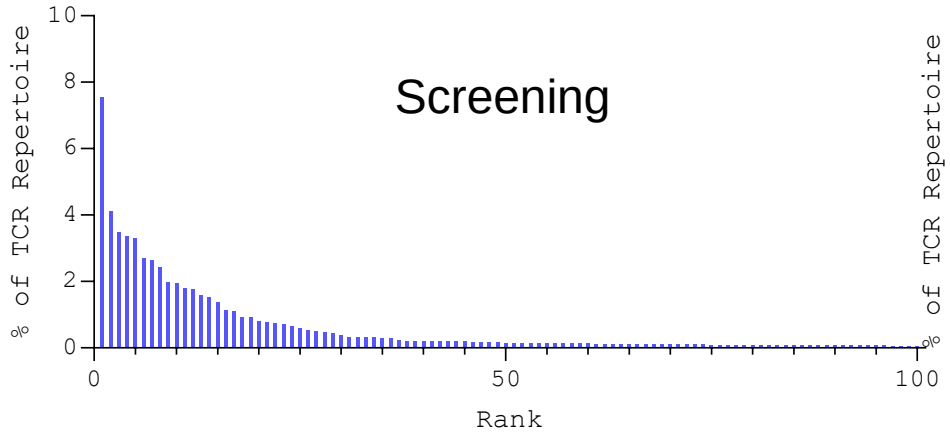
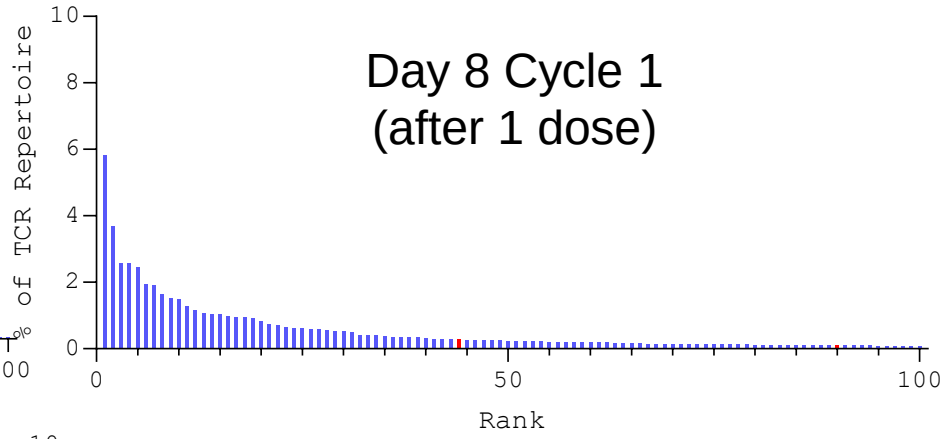
FT500 Phase 1: Clinical Translation

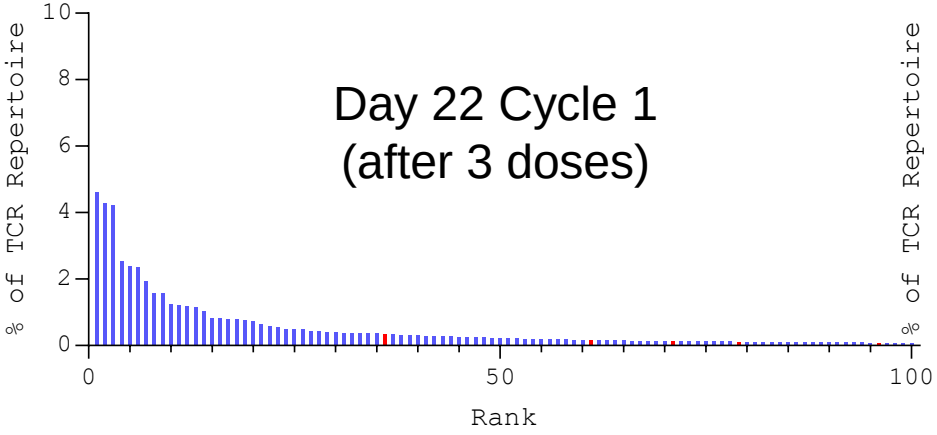
Patient 3: Assessment of Patient T-cell Repertoire with FT500 Multi-dose Treatment

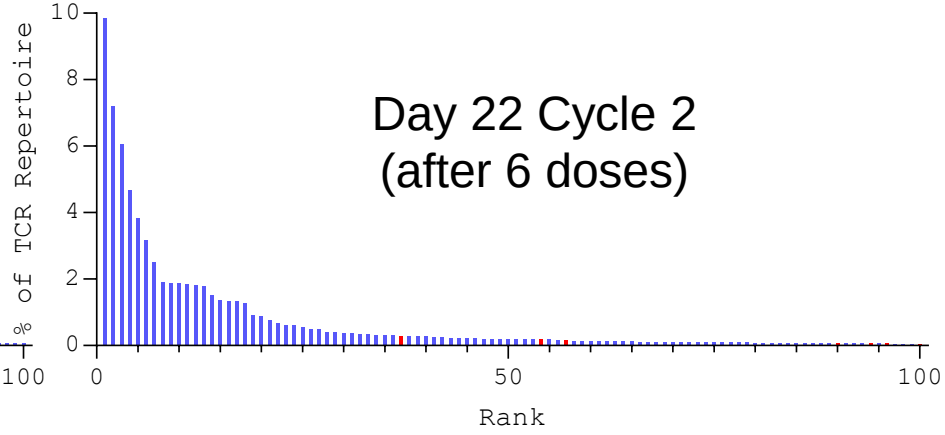
T-cell clones – Existing at Baseline (prior to FT500 treatment)

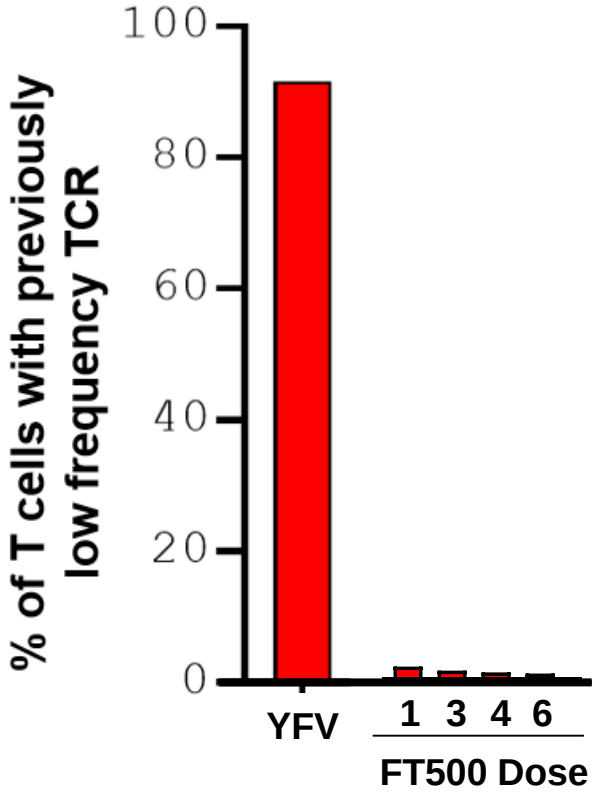
T-cell clones – Emerging post-FT500 treatment







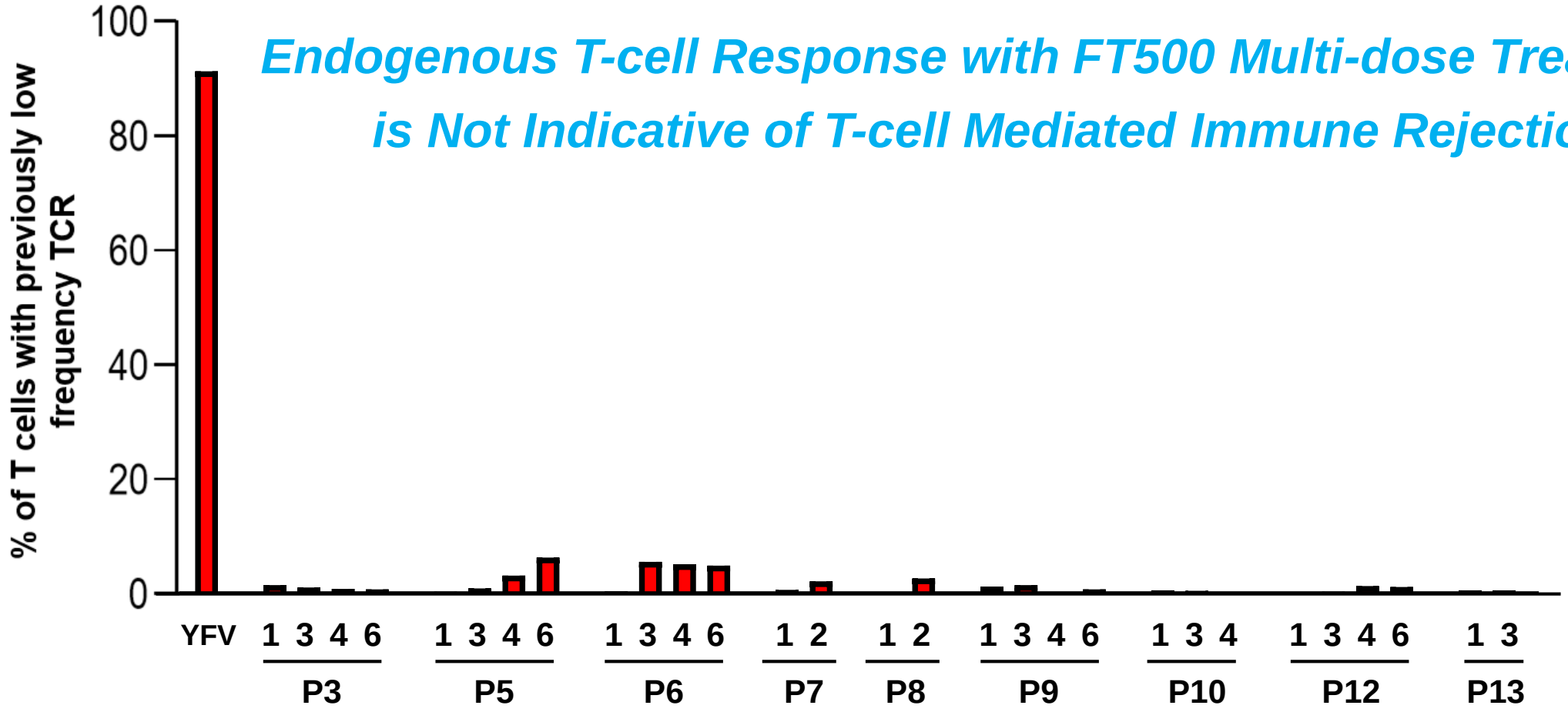




FT500 Phase 1: Clinical Translation

T-cell Mediated Anti-FT500 Immunogenicity

Endogenous T-cell Response with FT500 Multi-dose Treatment is Not Indicative of T-cell Mediated Immune Rejection



By patient by dose

Outpatient lympho-conditioning regimen: Cy (300 mg/m^2) x Flu (25 mg/m^2) x 2 days prior to Cycle 1 only

FT500 Phase 1: Clinical Translation

Pre-existing B-cell Mediated Anti-FT500 Immunogenicity

Pre-existing anti-FT500 Antibodies are NOT Increased with FT500 Multi-dose Treatment

MFI levels > 5,000 are correlated with increased risk of primary graft failure in HSCT (9% vs. 54%)

EBMT Guidelines on Donor Specific Antibodies; Ciuria et al, BMT, 2018

Method: Panel Reactive Antibody Test (LabCorp)

- Detection of anti-HLA antibodies against an HLA antigen panel, including 6 Class I HLA types expressed by FT500
- Positive results confirmed by single antigen bead assay with MFI

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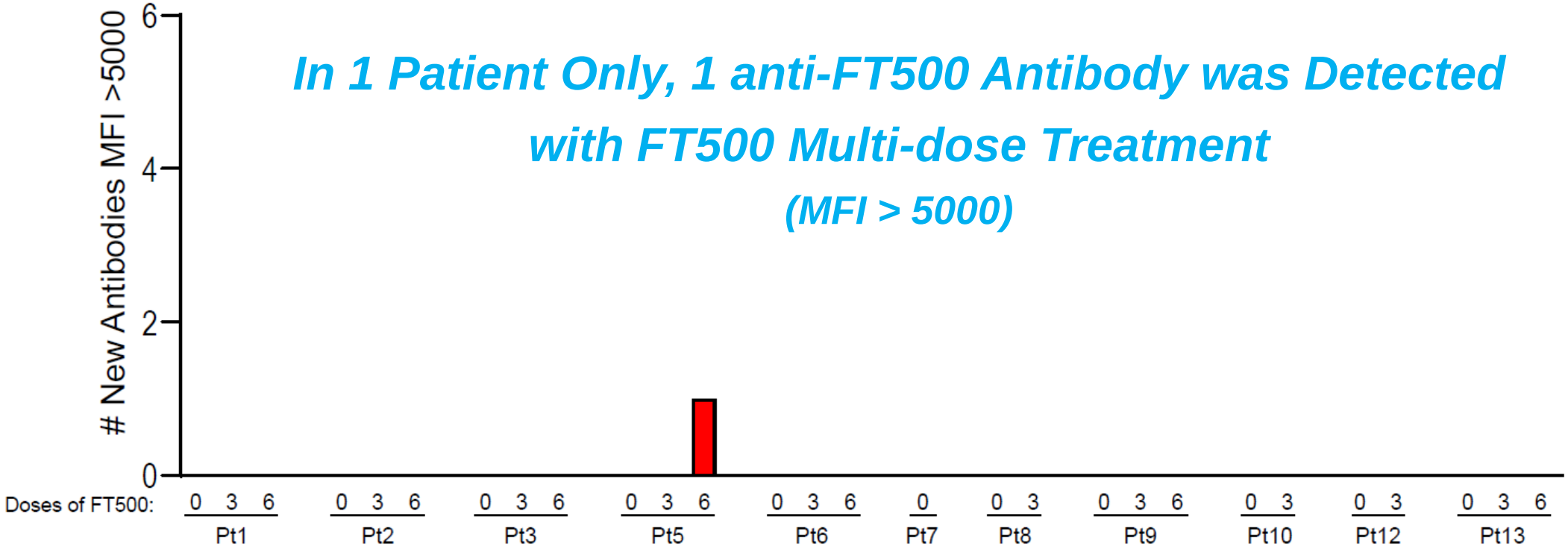
FT500 Phase 1: Clinical Translation

De Novo B-cell Mediated Anti-FT500 Immunogenicity



In 1 Patient Only, 1 anti-FT500 Antibody was Detected with FT500 Multi-dose Treatment (MFI > 5000)

By patient by dose

Outpatient lympho-conditioning regimen: Cy (300 mg/m^2) x Flu (25 mg/m^2) x 2 days prior to Cycle 1 only

MFI levels > 5,000 are correlated with increased risk of primary graft failure in HSCT (9% vs. 54%)

EBMT Guidelines on Donor Specific Antibodies; Ciuria et al, BMT, 2018

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FT500 Phase 1: Clinical Translational

Summary Findings

Multi-dose, Multi-cycle Treatment with iPSC-derived NK Cell Product is Safe and Well Tolerated without Eliciting Host Immune Rejection

- Healthy endogenous immune cell recovery following multi-dose FT500 treatment
- No biomarker evidence of sub-clinical CRS, neurotoxicity, or GvHD
- Endogenous T-cell response to FT500 is not indicative of T-cell mediated immune rejection
- Anti-FT500 antibody assessment is not indicative of B-cell mediated immune rejection

Outpatient lympho-conditioning regimen: Cy (300 mg/m^2) x Flu (25 mg/m^2) x 2 days prior to Cycle 1 only

Fate THERAPEUTICS

Off-the-shelf NK Cell Cancer Immunotherapy

iPSC-Derived NK Cell Therapies: Off-the-Shelf, Multi-Dosing Strategies

Jeffrey S. Miller, MD
Deputy Director, Masonic Cancer Center
Director, NK Cell Program
Minneapolis, MN

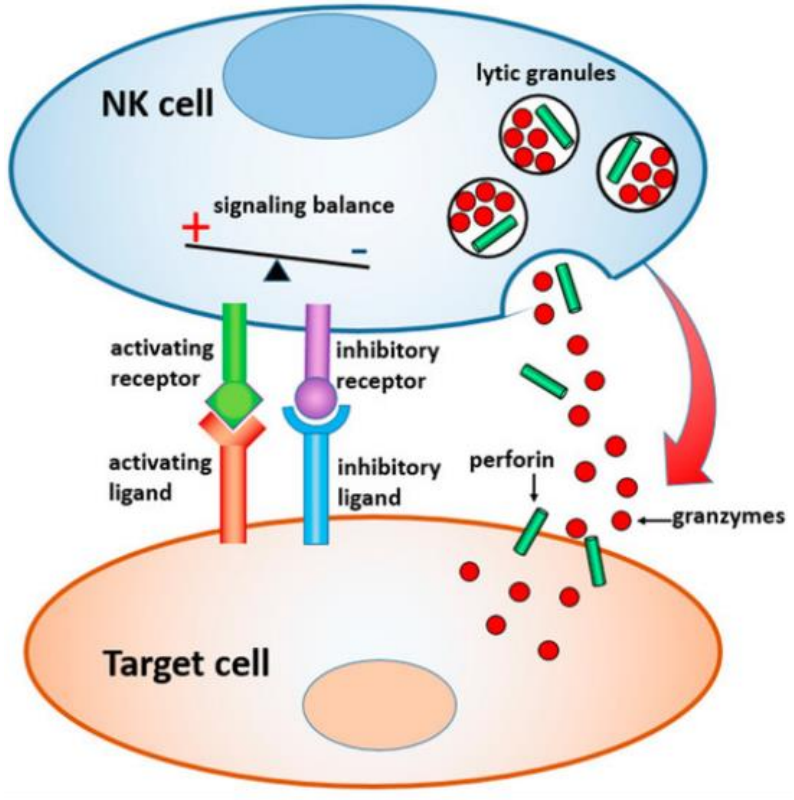
Disclosures

- Fate Therapeutics – Research Support, Consulting
- GT BioPharma – AB, Research Support, Consulting
- OnkImmune – SAB
- Dr. Reddy's Laboratory – SAB

Natural Killer Cells

First Line of Defense against Tumors and Diverse Range of Pathogens

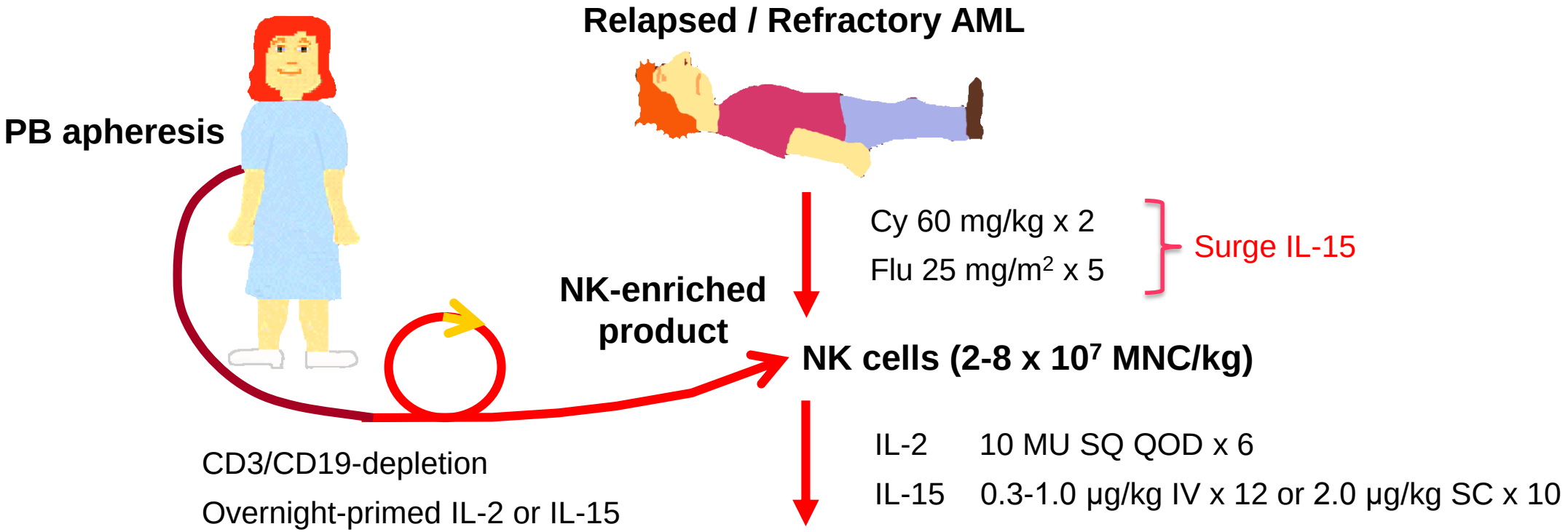
Array of Activating and Inhibitory Surface Receptors Mediate NK Cell Activity



- **Activating receptors convey multi-faceted effector function against tumor cells**
  - Unique ability to recognize stressed / transformed cells, leaving healthy cells unharmed
  - Engage and lyse antibody-coated tumor cells through CD16 Fc receptor (antibody-dependent cellular cytotoxicity, or ADCC)
  - Direct killing through release of cytotoxic granules
  - Trigger adaptive immune response through cytokine production
- **Inhibitory receptors can override activating signals**
  - KIR receptors balance activation through MHC-I molecule interactions
  - Multiple immune checkpoint receptors (e.g., TIGIT, PD-1)

Donor-derived NK Cell Therapy

Clinical Precedent for Anti-Cancer Activity in Relapsed / Refractory AML



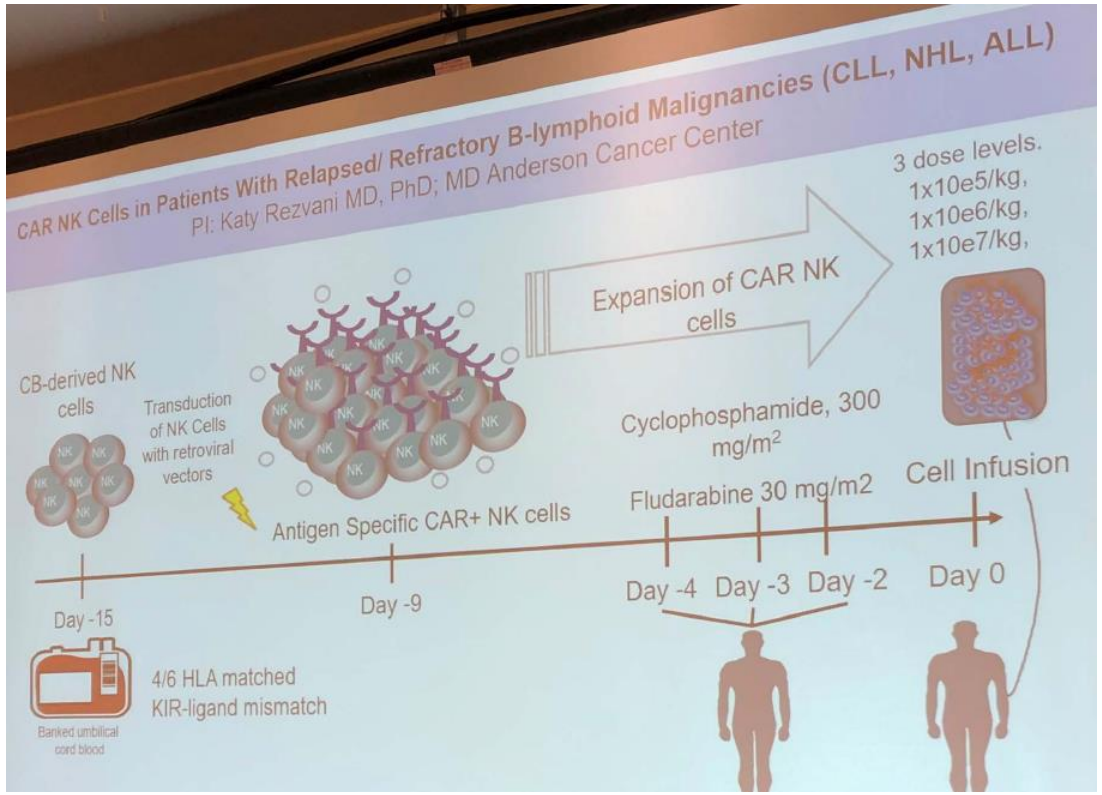
- **Donor NK cell persistence**
  - **10% >100 cells/mL D14 with IL-2[1,2]**
  - **35% >100 cells/mL D14 with IL-15[3]**
- **Response rates**
  - **CR: 26% IL-2[1] or ~35% IL-15[2]**

[1]Miller et al, Blood, 2005 [2]Cooley et al, Blood Advances 2019

Donor-derived CAR NK Cell Therapy

Clinical Precedent for Anti-Cancer Activity in Relapsed / Refractory Lymphoma

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



As reported at ASGCT 2019

- First-in-human clinical trial of donor-derived CAR19 NK cells
  - Cord blood derived
  - Transduced with CAR19 (28z) / IL15 (secreted) / iCas9 (suicide)
- Treated 11 patients with r/r B-cell malignancies
  - 3 dose levels (0.1M, 1.0M, 10M cells / kg)
- CR in 8/11 patients
  - CRs observed at all dose levels
  - CRs observed across all disease sub-types
- No CRS / neurotoxicity

Changing the Game in Cell-based Cancer Immunotherapy

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

Key Features	Patient / Donor Cell Therapy	iPSC-derived Cell Therapy
Genetic Engineering	Random & Variable	Uniform & Complete
Characterization	Imprecise	Well-defined
Product Identity	Heterogeneous	Homogeneous
Manufacturing	Limited Dose Availability	Off-the-Shelf Availability
Cost-per-Dose	High	Low
Dosing	Single Dose	Multiple Doses / Multiple Cycles
Overall Paradigm	**Process-centric**	**Product-centric**

Fate THERAPEUTICS

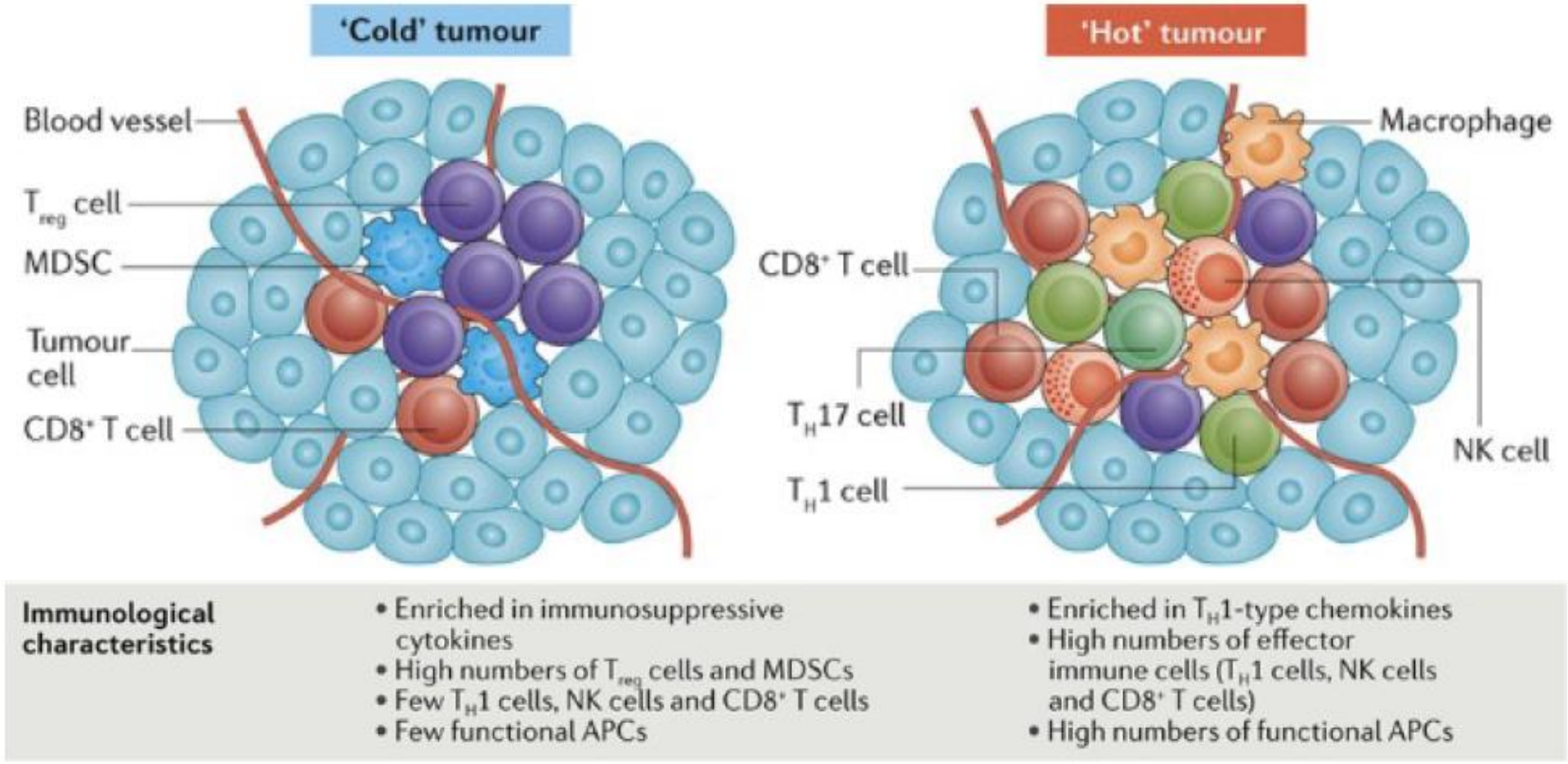
FT500: Off-the-Shelf, iPSC-derived NK cells

Promising Observations from Initial Phase 1 Clinical Data

- ✓ iPSC-derived NK cells can be generated that are of high purity, are phenotypically comparable to healthy donor NK cells, and are functionally potent *in vitro* and *in vivo*
- ✓ iPSC-derived NK cells can be mass-produced in a cGMP process, cryopreserved in an infusion-ready media, and have high post-thaw viability and activity
- ✓ iPSC-derived NK cells can be given in a multi-dose manner from an off-the-shelf source
  - ✓ Operational milestone – 100s of doses can be efficiently distributed to multiple clinical sites
  - ✓ Treatment milestone – off-the-shelf, multi-dose administration in outpatient setting
  - ✓ Safety milestone – No evidence of safety concerns or clinically-meaningful immune rejection
- ✓ Game-changing for cell therapy; converting to mAb-like drug product paradigm

Potential for NK Cell Therapy in Advanced Solid Tumors

iPSC-derived NK Cells can turn a "cold" tumor "hot"



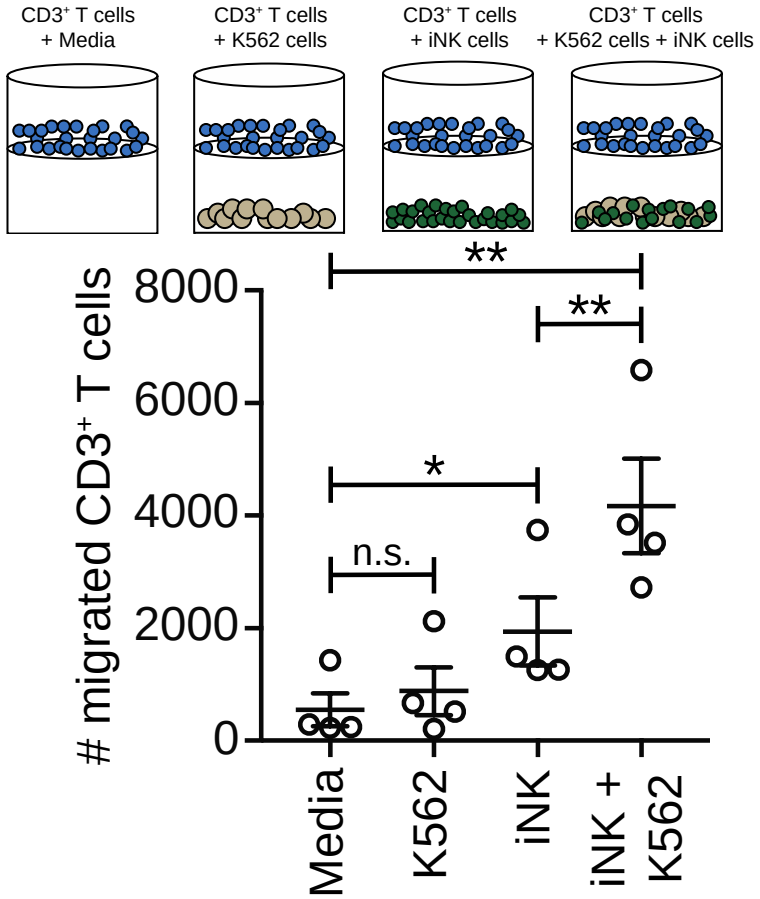
Nagarsheth et al, Nature Reviews Immunology, 2017

ASH Abstract #1933: iPSC-derived NK Cells Synergize with T Cells and anti-PD-1 Antibody to Mediate Durable Anti-tumor Responses In Vivo (Miller et al)

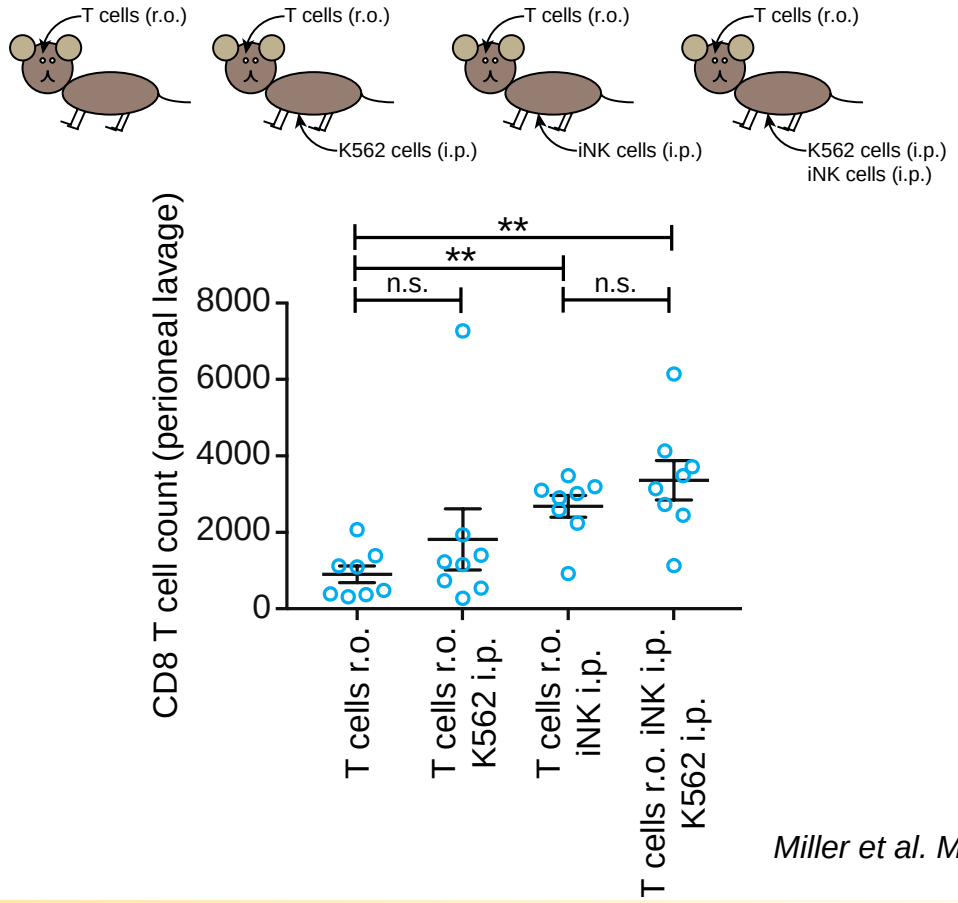
iPSC-derived NK Cells Promote T-cell Recruitment

In Vitro and In Vivo Migration and Trafficking Assays

In Vitro T-cell Recruitment



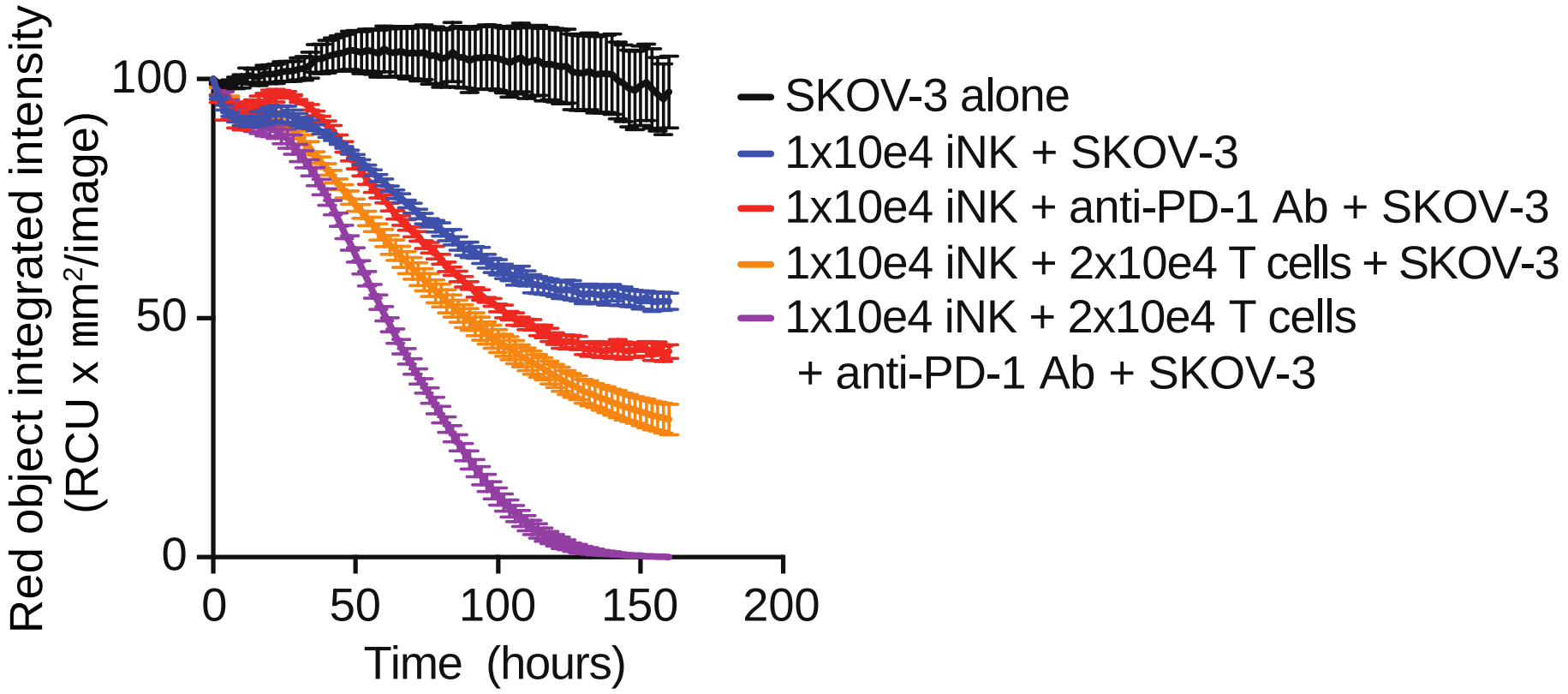
In Vivo T-cell Recruitment



Miller et al. Manuscript under review

iPSC-derived NK Cells Synergize with T Cells and anti-PD1 Blockade

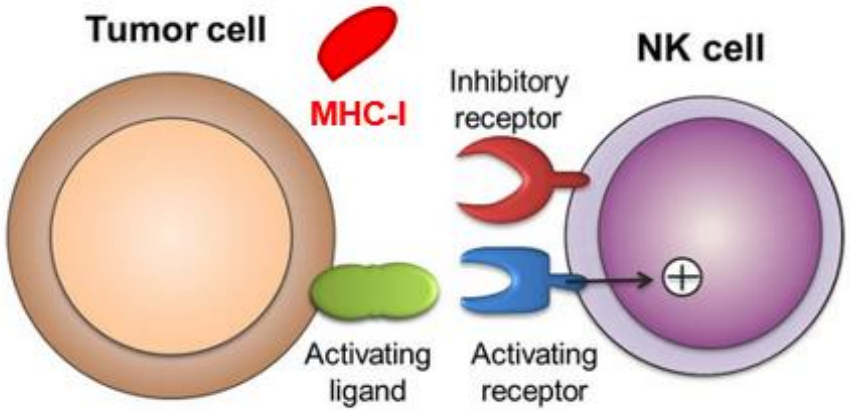
Solid Tumor Spheroid Model of Ovarian Cancer



Miller et al. Manuscript under review

iPSC-derived NK Cells Synergize with T Cells and anti-PD1 Blockade

Potential Clinical Strategy to Overcome Resistance to Checkpoint Inhibitor Therapy



NK cells have the unique ability to recognize and kill cancer cells that have down-regulated MHC Class I

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

FT500 iPSC-derived NK Cell Product Candidate

Plan to Initiate Phase 1 Dose Expansion to Assess FT500 Anti-Tumor Activity

Patient who progressed on prior ICI

FT500 + ICI

KEYTRUDA® (pembrolizumab) Injection 100 mg
OPDIVO® (nivolumab)
TECENTRIQ® atezolizumab

Objective Response: FT500 re-establishes sensitivity of tumors to ICI

Dose Expansion Strategy	Rationale
Add IL-2 Support	• IL-2 known to enhance NK cell function and persistence
Add Cycle 2 Outpatient Lympho-conditioning	• Established safety and tolerability of outpatient conditioning in Cycle 1 • Facilitates NK cell proliferation due to IL-15 surge
Tumor Enrichment • NSCLC • Melanoma • Bladder (urothelial carcinoma)	• High % of low MHC Class I expression (amenable to NK cell activity) • NK cell trafficking to lung (NSCLC) • Inflamed tumor types • Accessible tumor biopsies • Higher prevalence of somatic mutations (amenable to T-cell activity)

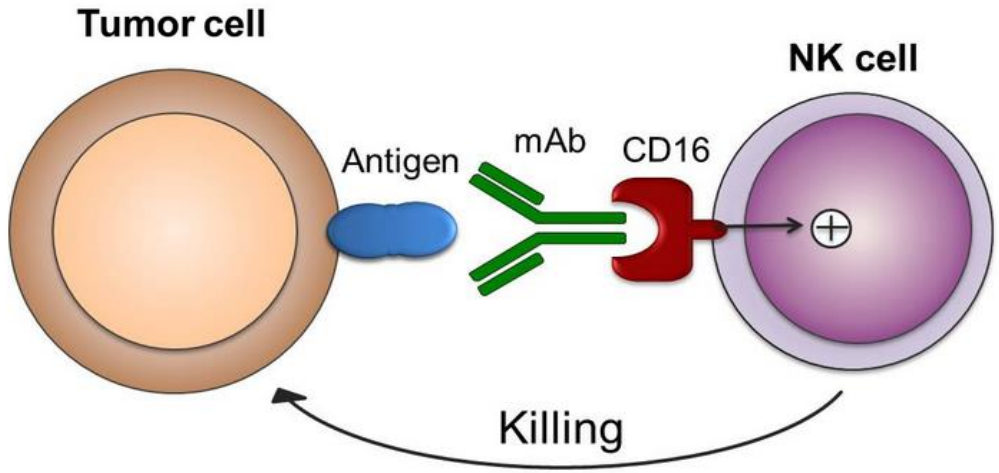
Phase 1 Dose Expansion of Regimen B at 300M cells / dose, subject to MTD determination in ongoing Phase 1 Dose Escalation

Fate THERAPEUTICS

Potential for NK Cell Therapy in Combination with mAb Therapy

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

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



Rituxan Rituximab

GAZYVA obinutuzumab

DARZALEX (daratumumab)

Herceptin trastuzumab Precision • Power • Promise

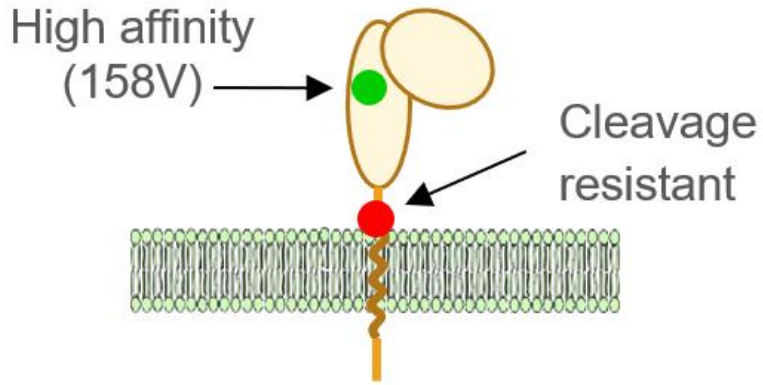
ERBITUX Cetuximab

BAVENCIO avelumab Injection

iPSC-derived NK Cells with Engineered High-affinity, Non-cleavable CD16 Fc Receptor

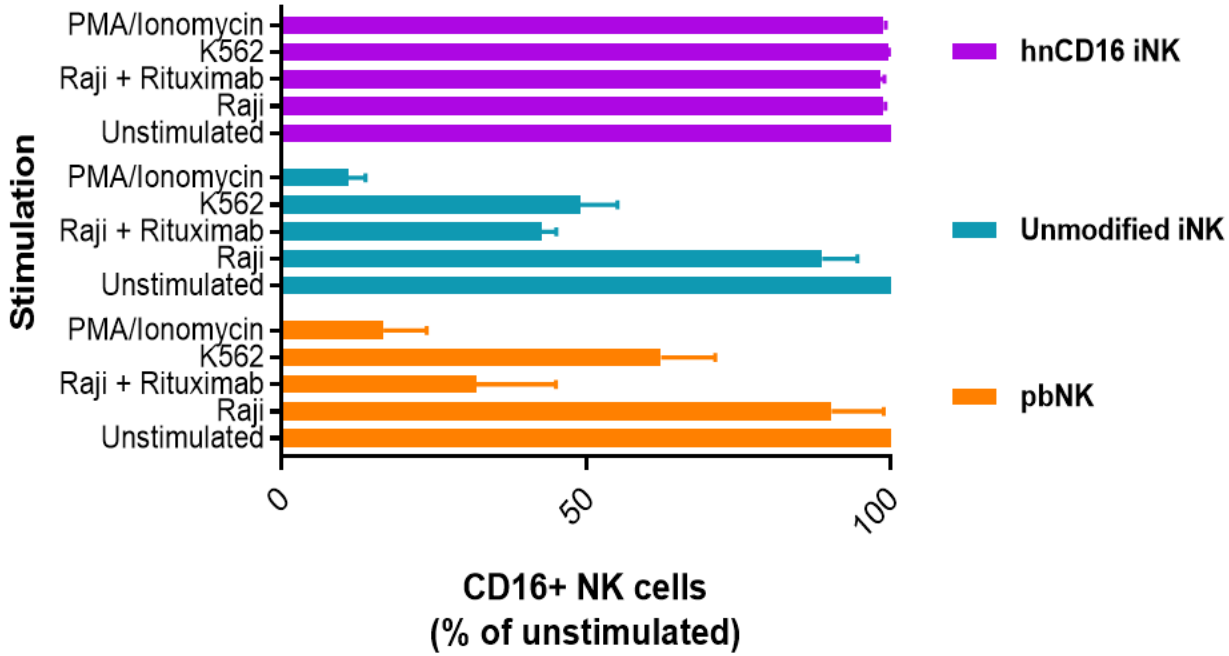
Designed to Overcome Deficiencies in Endogenous NK Cell Numbers, CD16 Expression, and mAb Affinity

Novel Engineered CD16 for Optimized ADCC



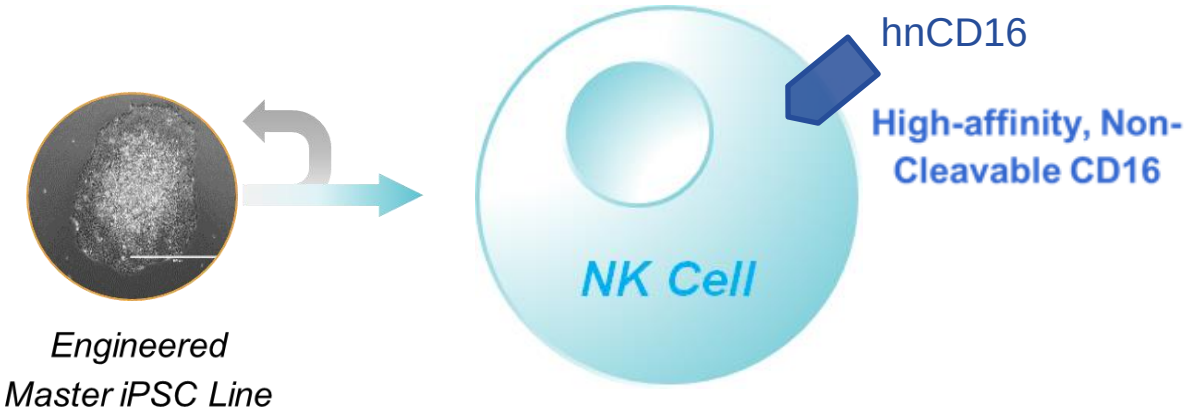
Modified form of CD16a IgG antibody-binding receptor resists shedding upon activation

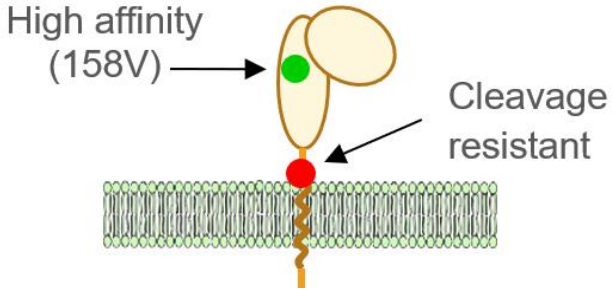
Maintenance of Engineered CD16 Expression



FT516 Engineered hnCD16 NK Cell Product Candidate

High-Affinity 158V Binding to Monoclonal Antibody for Enhanced ADCC

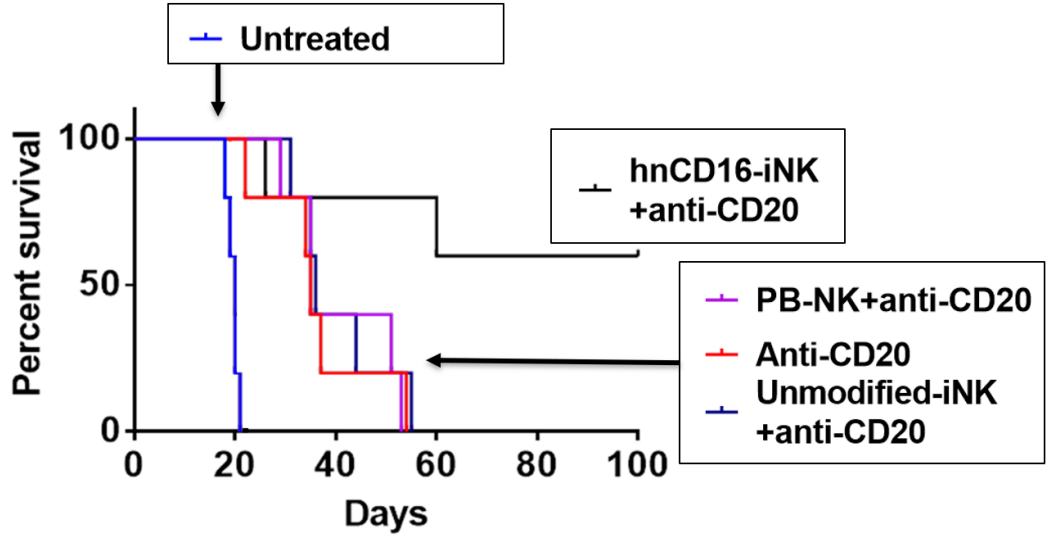




Modified form of CD16a IgG antibody-binding receptor resists shedding upon activation

Enhanced Survival *In Vivo* with Rituximab

Mouse model of human lymphoma (Raji cells)



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

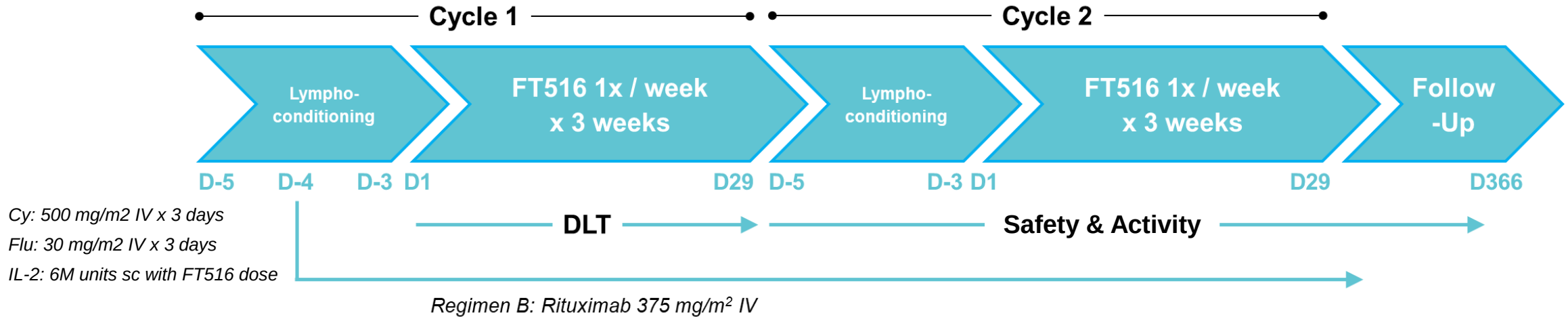
Kaufman et al.
Manuscript accepted by Blood

Fate THERAPEUTICS

FT516 Engineered hnCD16 NK Cell Product Candidate

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

First-ever Clinical Trial in World of Engineered iPSC-derived Cell Therapy



Regimen A – Monotherapy

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

Regimen B – Rituximab Combination Rituxan Rituximab

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

First Patients Treated in October 2019

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FT516 Patient 1 in R/R AML as Monotherapy

No response to Induction, Refractory to Three Prior Therapies

41 year old male diagnosed with AML

- Diagnosis: 40% blasts with marked megakaryocytic dysplasia; normal karyotype; ETV6, RBM15 and MLLT10 translocations; NRAS mutated by NGS; FLT3, NPM1, IDH1/2 not detected
- No response to initial 7 + 3 induction
- No response to MEC (mitoxantrone, etoposide, Ara-C) re-induction
- No response to venetoclax and decitabine
- ***Enrolled in FT516 Study – Regimen A Monotherapy***
  - After one cycle of three weekly doses of FT516 with IL2 support:
    - No observed dose-limiting toxicities
    - No observed Grade ≥3 adverse events or related serious adverse events
    - No observed CRS, neurotoxicity or GvHD
  - By Day 42:
    - No morphologic evidence of leukemia with evidence of hematopoietic recovery in bone marrow
    - No circulating leukemic blasts detected
    - Recovery of peripheral neutrophil count to >1,000 per µL without growth factor support
    - Detectable FT516 in bone marrow aspirate at Day 18

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FT516 Patient 1 in R/R Lymphoma in Combination with Rituximab

High-risk DLBCL, Early Relapse following CD19 CAR-T Cell Therapy

66 year old female diagnosed with double-hit DLBCL (high-risk DLBCL variant)

- Initial treatment with DA-EPOCH-R with intrathecal (IT) methotrexate for prophylaxis against CNS disease
  - Achieved remission; relapsed 1 year later
- Salvage immunochemotherapy (R-ICE x 2 cycles with IT methotrexate prophylaxis) resulting in complete response
  - High-dose chemotherapy (BEAM) followed by autologous HSCT
  - Relapsed disease in scalp area treated with salvage radiation
- Received lympho-depleting chemotherapy followed by Kymriah (autologous CD19 CAR-T)
  - Achieved remission; relapsed 60 days post-Kymriah with multi-focal sites of disease
- ***Enrolled in FT516 Study – Regimen B Combination with Rituximab***
  - Received full Cycle 1 treatment: three once-weekly doses of FT516 + rituximab
    - Expected cytopenias observed following lympho-conditioning
    - No observed Grade ≥3 adverse events or related serious adverse events
    - No observed CRS, neurotoxicity, or GvHD
  - Initiated Cycle 2 treatment of FT516 + rituximab
  - Initial response assessment pending at the end of Cycle 2

Fate THERAPEUTICS

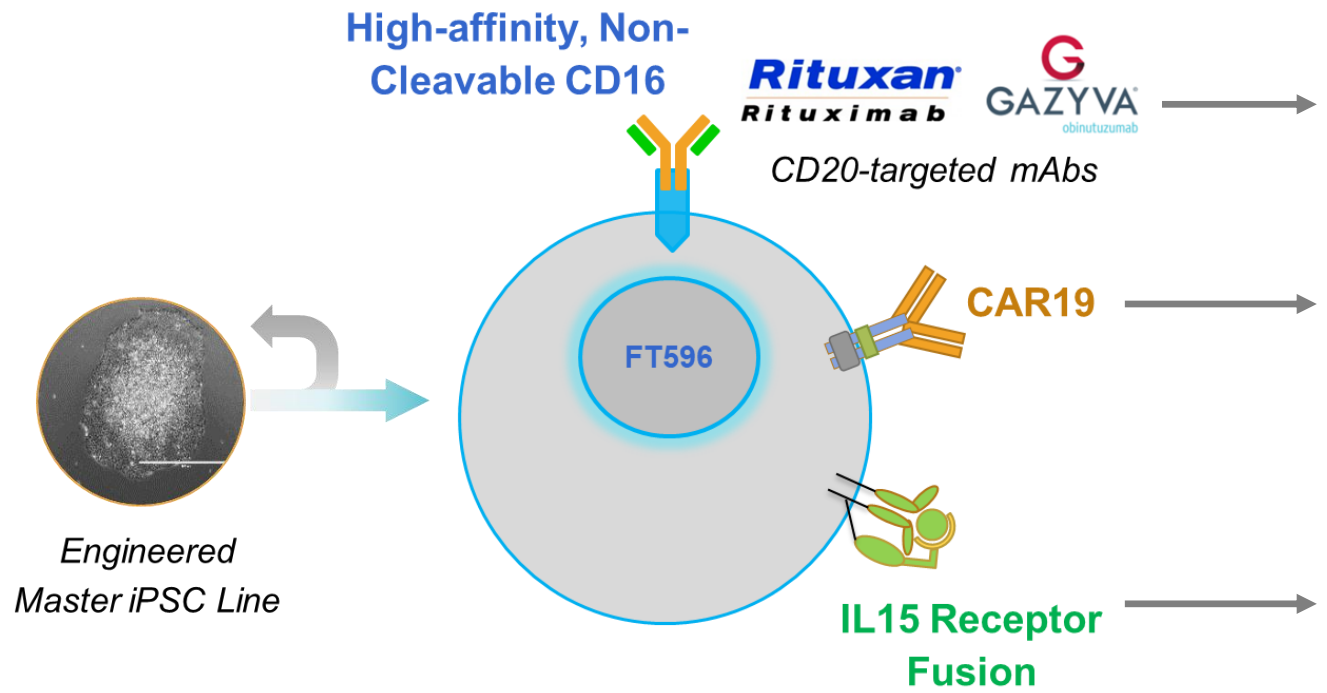
Multi-Antigen Targeting

Best-in-Class Therapeutic Strategy for Lymphoma

FT596 Off-the-Shelf Multi-Targeted CAR19 NK Cell Product Candidate

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

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



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

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

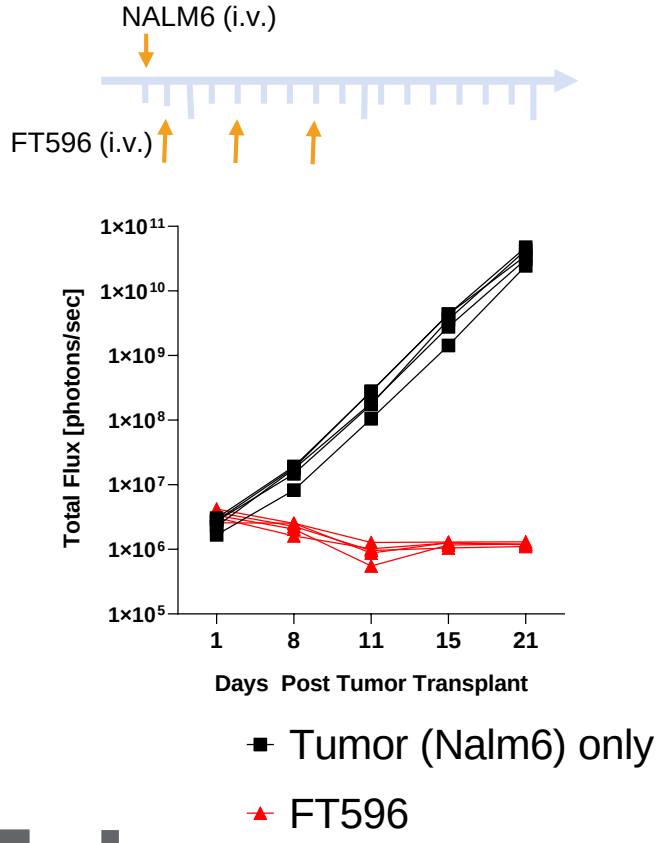
IL-15RF: Interleukin-15 receptor fusion, a potent cytokine complex that promotes survival, proliferation and trans-activation of NK cells and CD8 T cells

ASH Abstract #301: Translation of First-of-Kind Multi-Antigen Targeted Off-the-Shelf CAR-NK Cell with Engineered Persistence for the Treatment of B Cell Malignancies

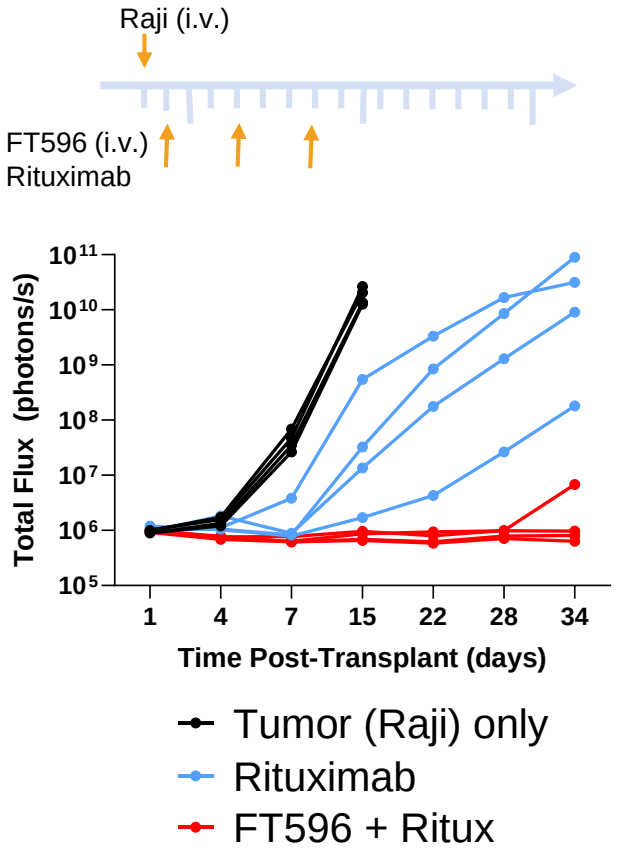
FT596 Demonstration of Efficacious Anti-tumor Activity *In Vivo*

Durable anti-Leukemia and anti-Lymphoma Efficacy in Various Xenograft Mouse Models

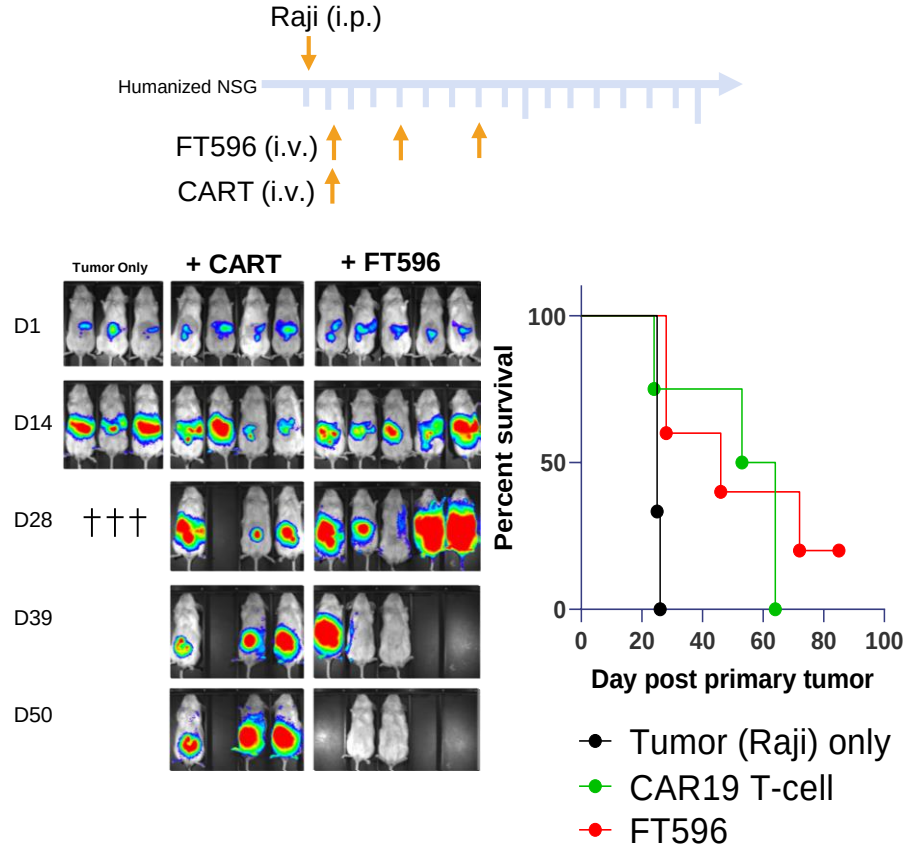
Monotherapy
Leukemia xenograft NSG immunodeficient mouse model



Combination
Lymphoma xenograft NSG immunodeficient mouse model



Allogeneic
Lymphoma xenograft CD34 engrafted humanized NSG mouse model



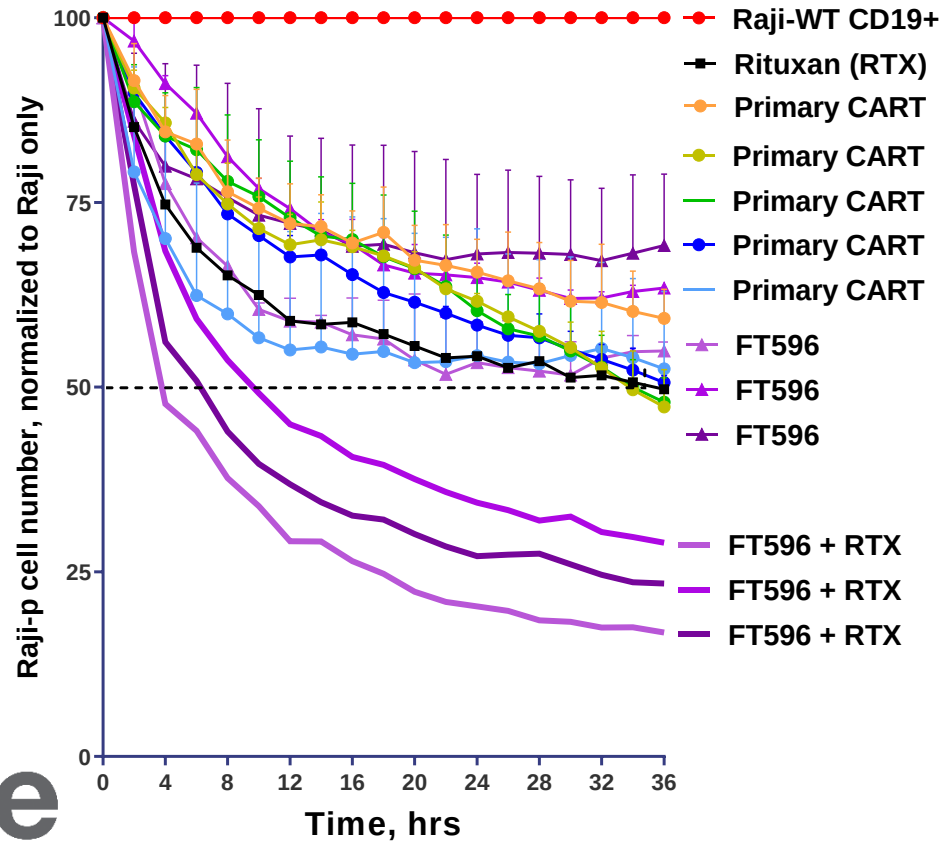
FT596 Off-the-Shelf Multi-Targeted CAR19 NK Cell Product Candidate

Synergistic Anti-tumor Activity of hnCD16 + CAR19 In Vitro

Deeper Response in Combination

in vitro stress test using low effector:target ratio (0.3:1) to determine durable efficacy during antigen availability

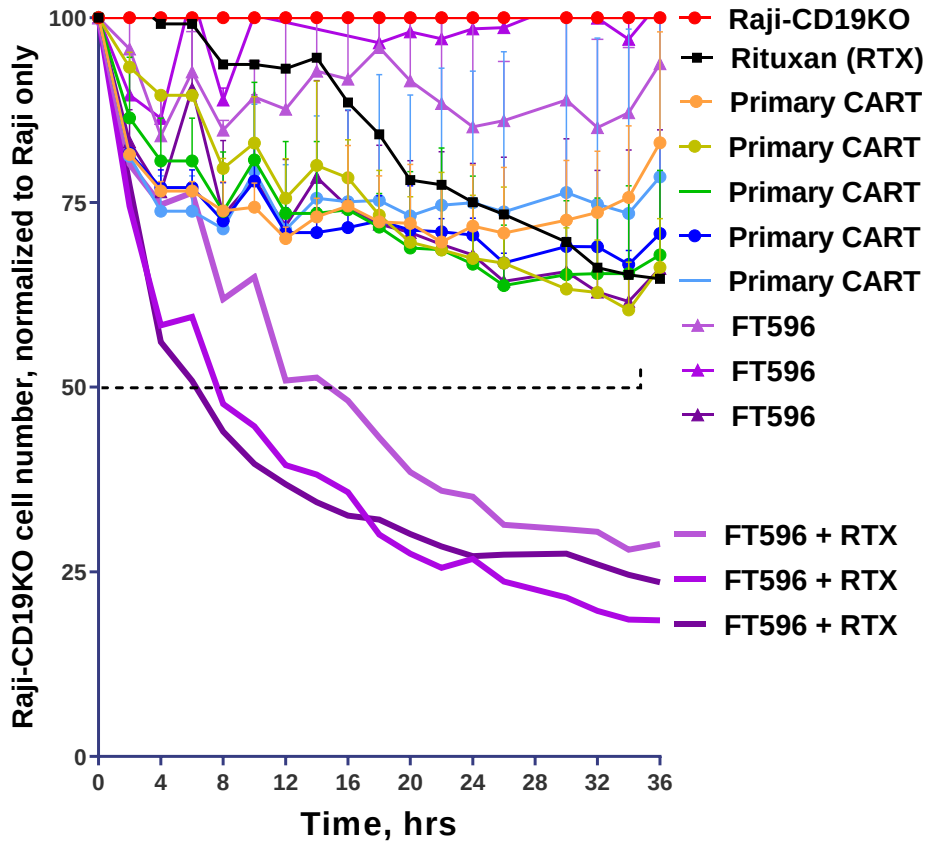
Raji lymphoma line CD19+ CD20+ | CAR19 | rituximab



Prevention of Antigen Escape in Combination

in vitro high-capacity test using high effector:target ratio (3:1) to maximize response in absence of primary antigen availability

Raji lymphoma line CD19- CD20+ | CAR19 | rituximab

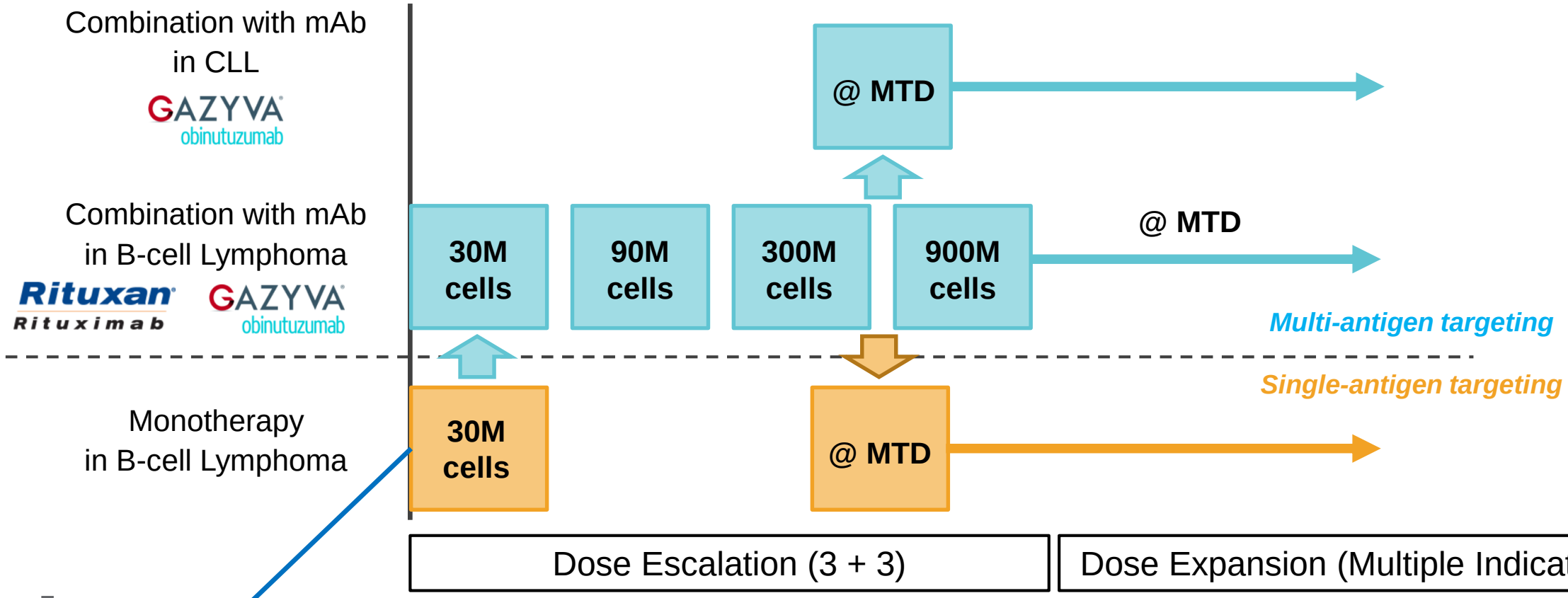


Fate THERAPEUTICS

FT596 Off-the-Shelf Multi-Targeted CAR19 NK Cell Product Candidate

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

Phase 1 Dose Escalation – Monotherapy and mAb Combination



2/3 subjects achieved CR at ~10M cells per dose in MDA cord blood-derived CAR19 NK cell study

Fate THERAPEUTICS

FT596 Off-the-Shelf Multi-Targeted CAR19 NK Cell Product Candidate

Clinical Trial Start-up

- Site selection complete (Memorial Sloan-Kettering; University of Minnesota; City of Hope; MD Anderson; Swedish Medical Center; Washington University)
- Start-up activities ongoing
- Current protocol addresses key FDA clinical questions

Questions	How Addressed in Protocol
• Unknown risk / benefit profile due to lack of safety, cell persistence and efficacy data	• Initiate study with single-dose escalation
• Safety and timing of multi-cycle dosing	• FDA review of emerging data to determine case-by-case feasibility of Cycle 2 dosing for individual patients • Data supporting clinical protocol amendment to include multi-cycle dosing to be shared with FDA
• Concurrent dose escalation of monotherapy and combination regimens	• Conduct initial monotherapy dose cohort (n=3) • Conduct combination regimen dose escalation (3+3) • Conduct monotherapy dose cohort at MTD

Fate THERAPEUTICS

Targeting Early 2020 for First Patient Treatment

Memorial Sloan Kettering Cancer Center

iNK Cell Therapies for Multiple Myeloma

Fate Therapeutics Investor Dinner

December 6, 2019

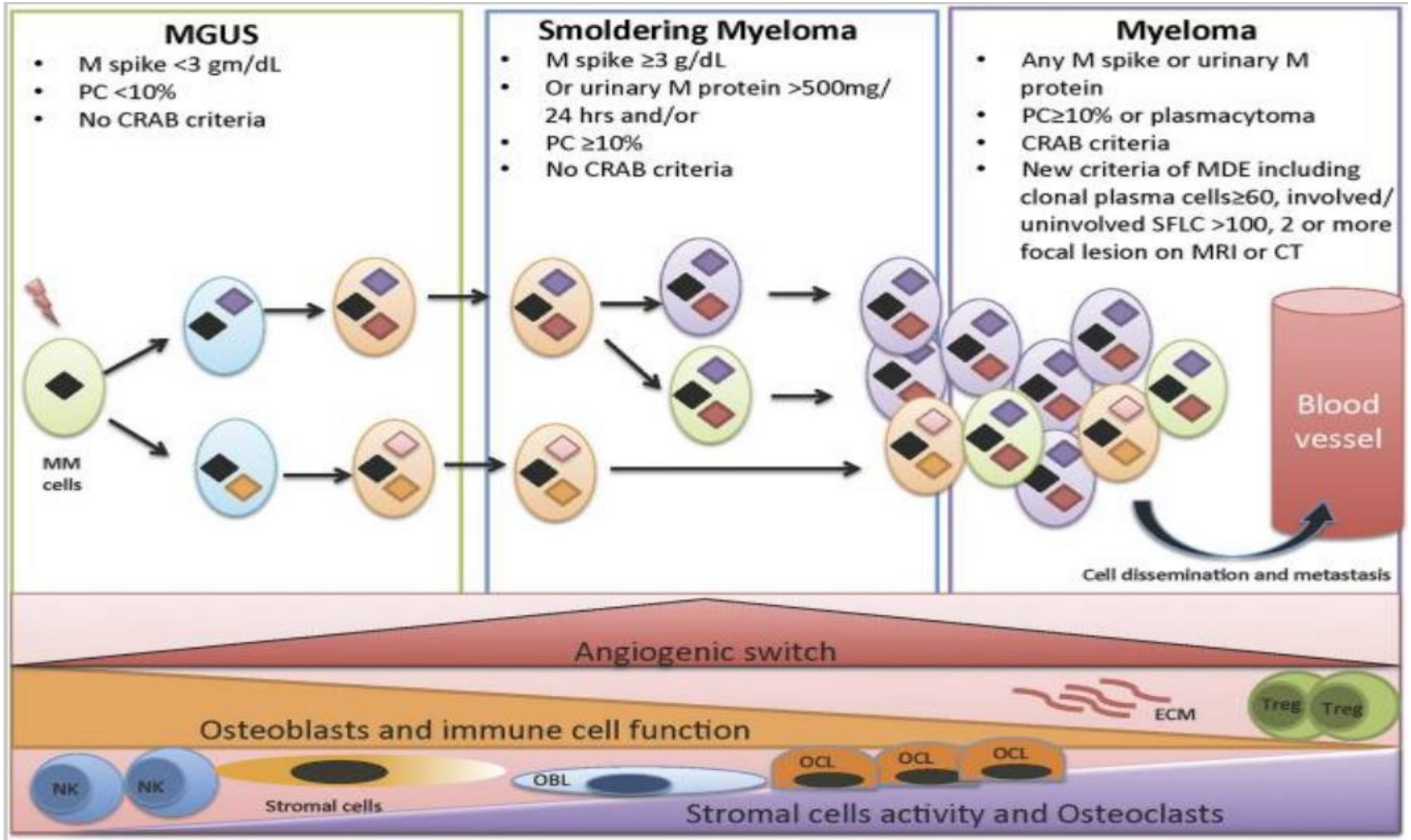
Eric L Smith MD, PhD
Director of Translational Development - Cellular Therapeutics Center
Assistant Member - Center for Cell Engineering
Assistant Attending - Myeloma Service
Memorial Sloan Kettering Cancer Center

Memorial Sloan Kettering Cancer Center

Disclosures

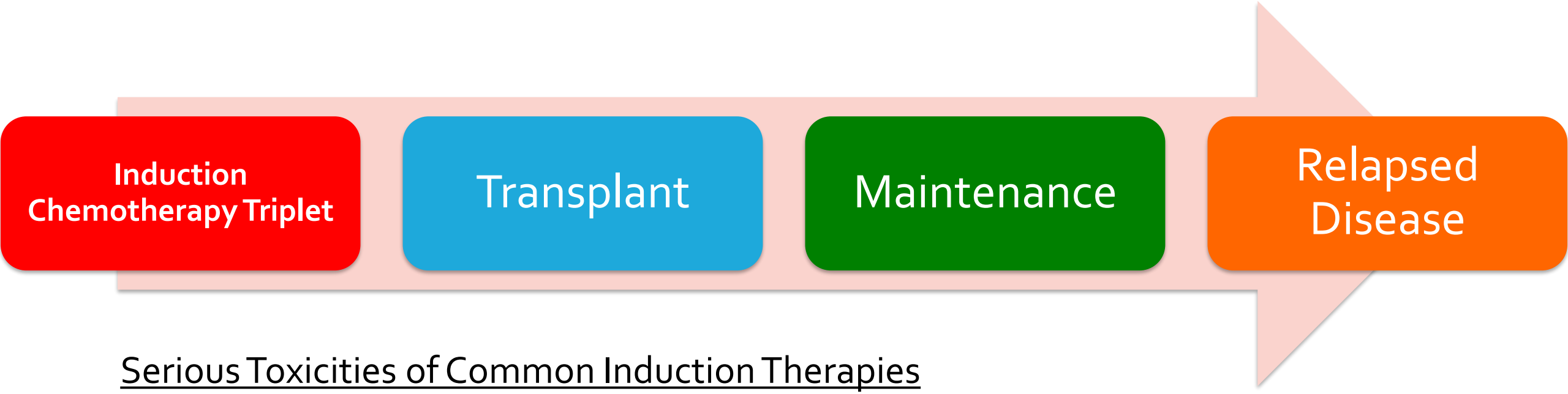
Commercial Interest(s)	Nature of Relationship
BMS	Licensed patents / royalties: CAR T cells to treat MM
BMS	Research Funding
BMS	Consultant
Fate Therapeutics	Consultant
Precision Biosciences	Consultant
Unlicensed patents	Antibodies / BiTEs to treat MM

Evolution of Multiple Myeloma



Ghobrial I and Landgren O. Blood. 2014.

Frontline Therapy



<u>Serious Toxicities of Common Induction Therapies</u>

Thrombosis / Pulmonary embolism

Infection

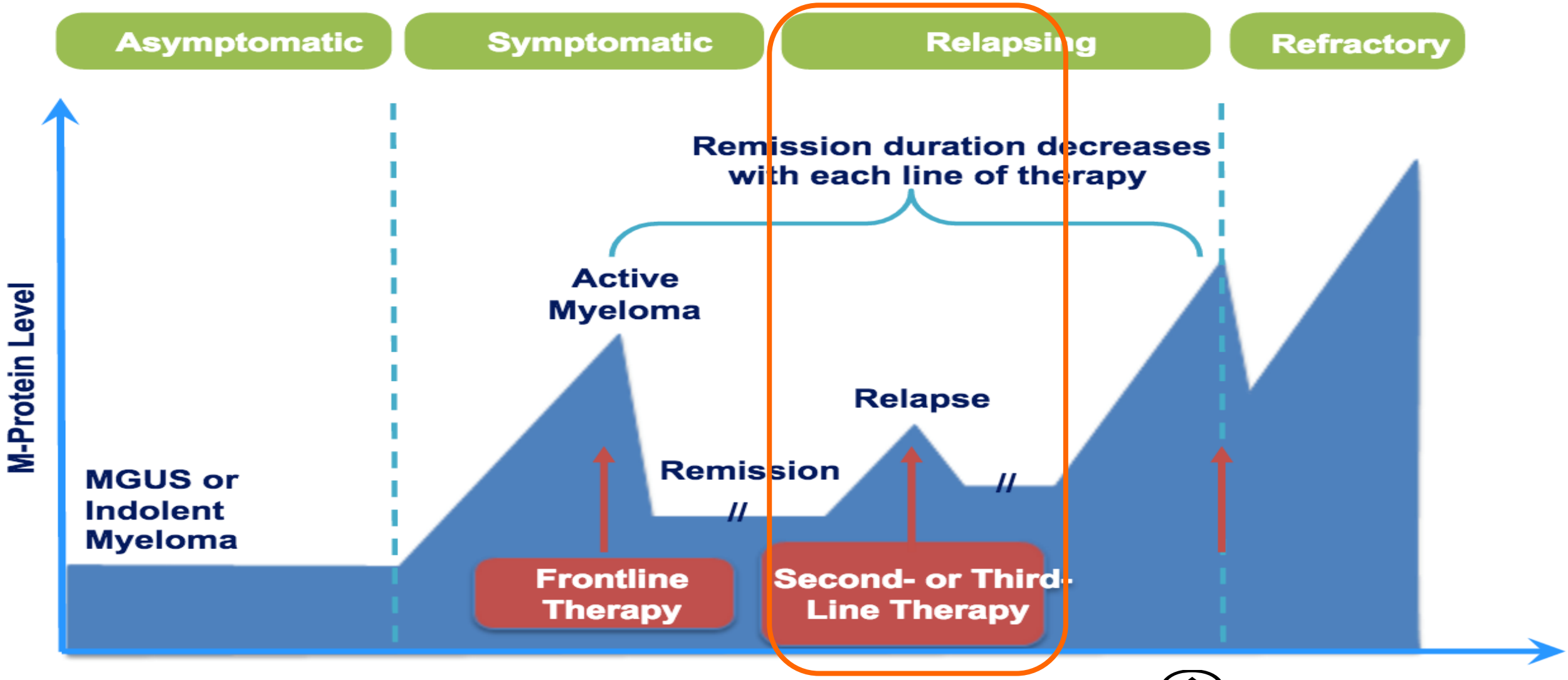
Neuropathy

Cytopenias

Heart failure

Secondary malignancies

Natural History of MM



Durie B; IMF. Concise review of the disease and treatment options: MM. 2011/2012.
Kumar SK et al. Mayo Clin Proc. 2004.

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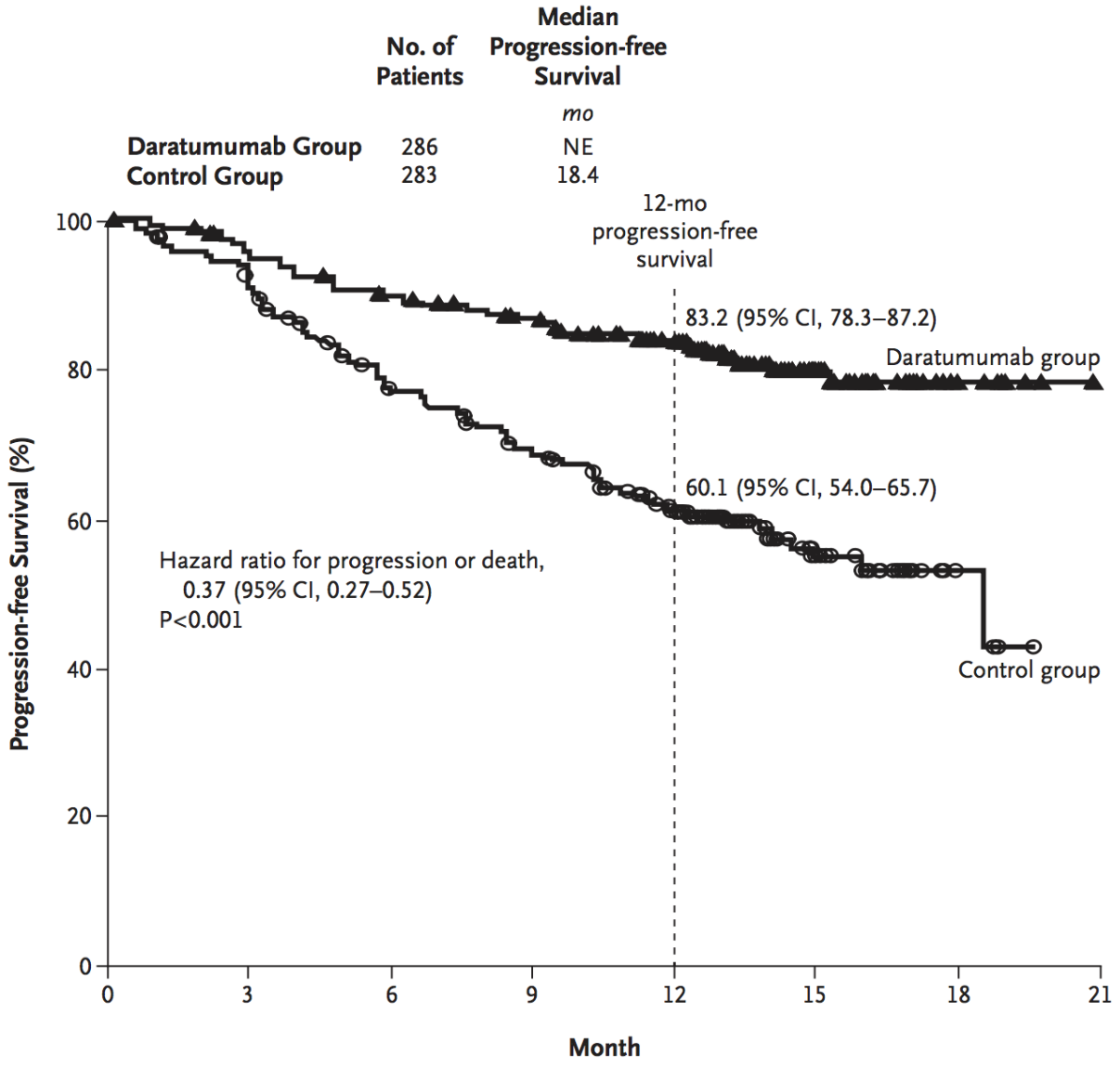
Daratumumab for Relapsed MM (median 1 prior line)

The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 OCTOBER 6, 2016 VOL. 375 NO. 14

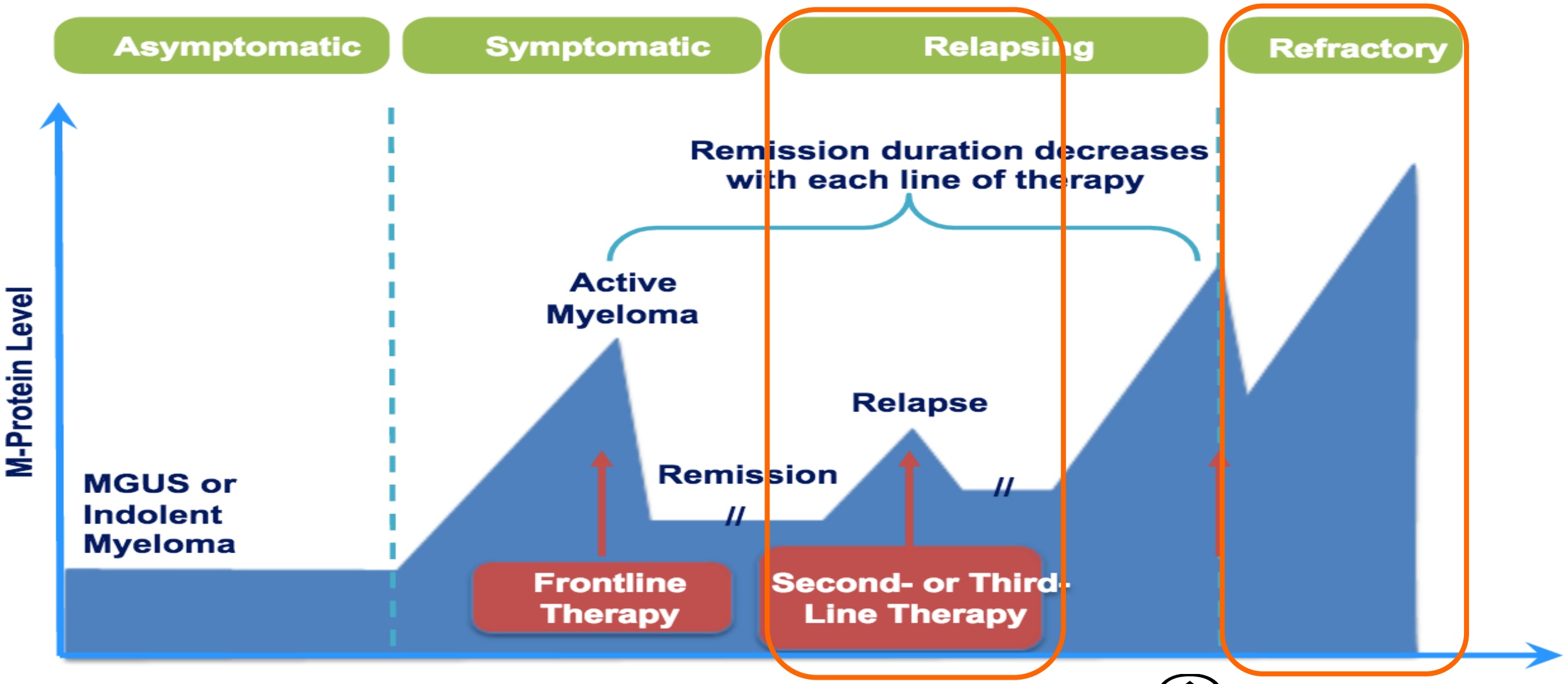
Daratumumab, Lenalidomide, and Dexamethasone for Multiple Myeloma

M.A. Dimopoulos, A. Oriol, H. Nahi, J. San-Miguel, N.J. Bahlis, S.Z. Usmani, N. Rabin, R.Z. Orlowski, M. Komarnicki, K. Suzuki, T. Plesner, S.-S. Yoon, D. Ben Yehuda, P.G. Richardson, H. Goldschmidt, D. Reece, S. Lisby, N.Z. Khokhar, L. O'Rourke, C. Chiu, X. Qin, M. Guckert, T. Ahmadi, and P. Moreau, for the POLLUX Investigators*



See also
CASTOR: Palumbo et al *NEJM* 2016
CANDOR: Usmani et al *ASH* 2019 Late Breaking Abst-6

Natural History of MM



Durie B; IMF. Concise review of the disease and treatment options: MM. 2011/2012.
Kumar SK et al. Mayo Clin Proc. 2004.

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Prognosis in Daratumumab Refractory Patients is Measured in Months

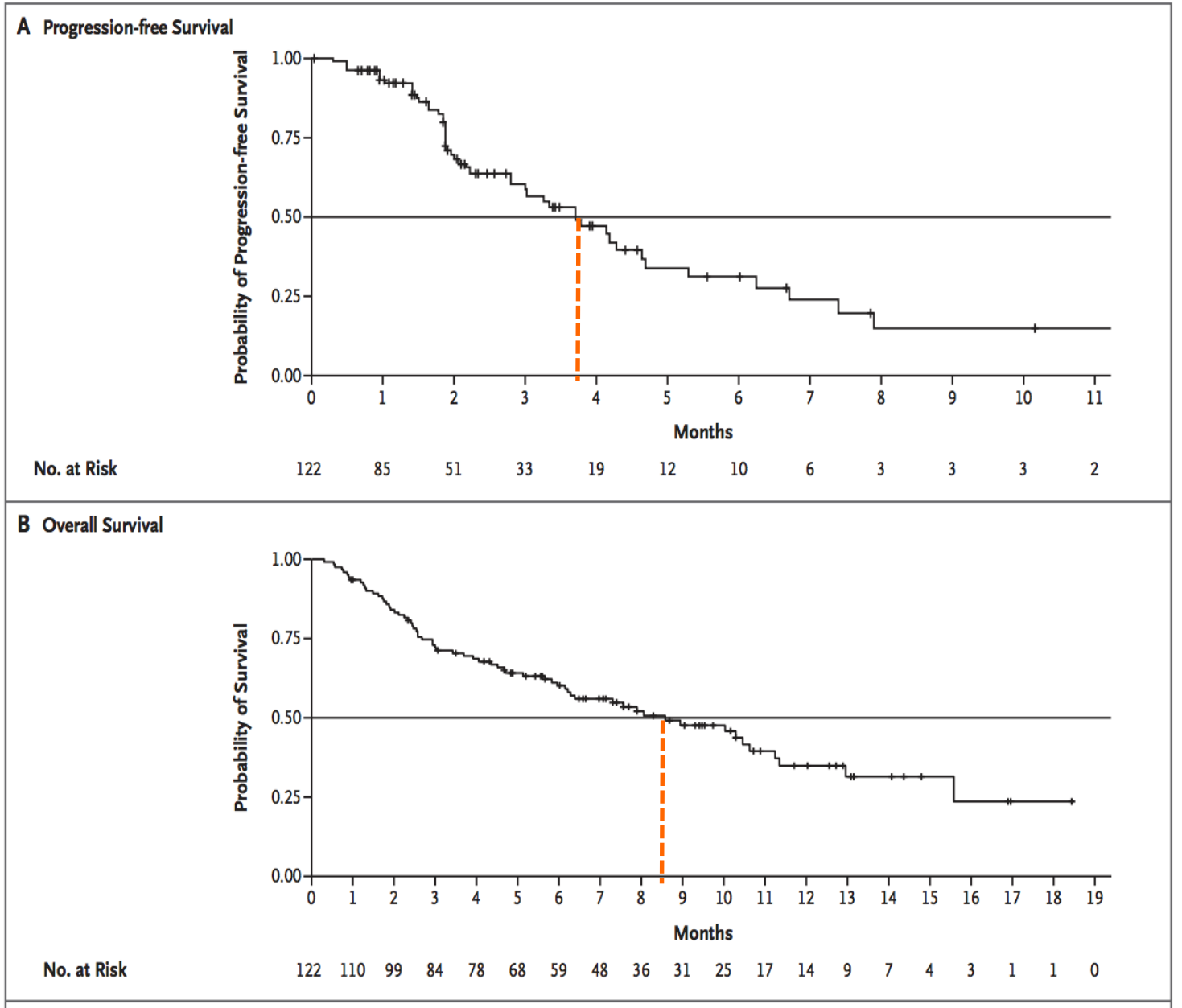
The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

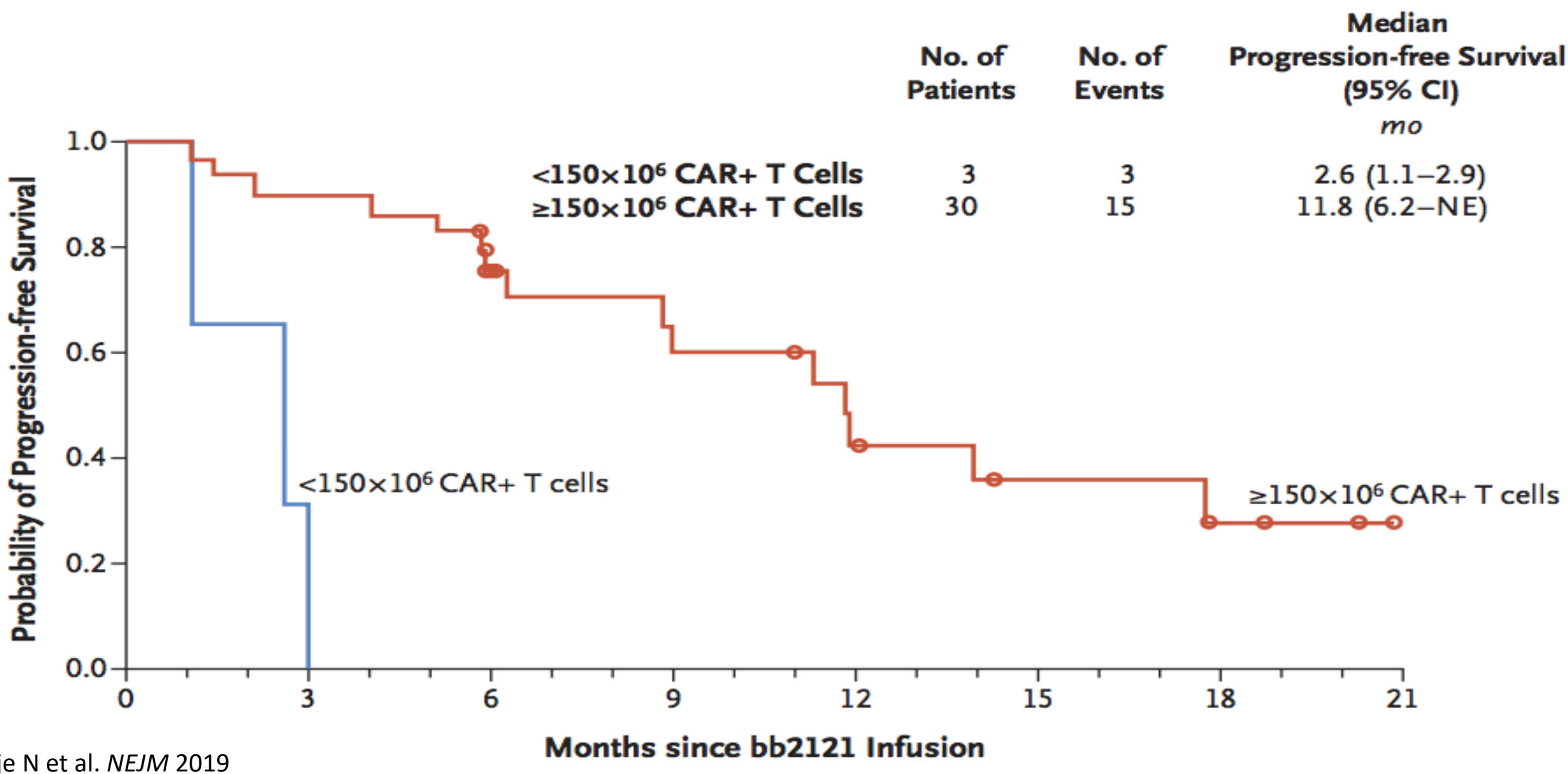
Oral Selinexor–Dexamethasone for Triple-Class Refractory Multiple Myeloma

A. Chari, D.T. Vogl, M. Gavriatopoulou, A.K. Nooka, A.J. Yee, C.A. Huff, et al

N ENGL J MED 381;8 NEJM.ORG AUGUST 22, 2019

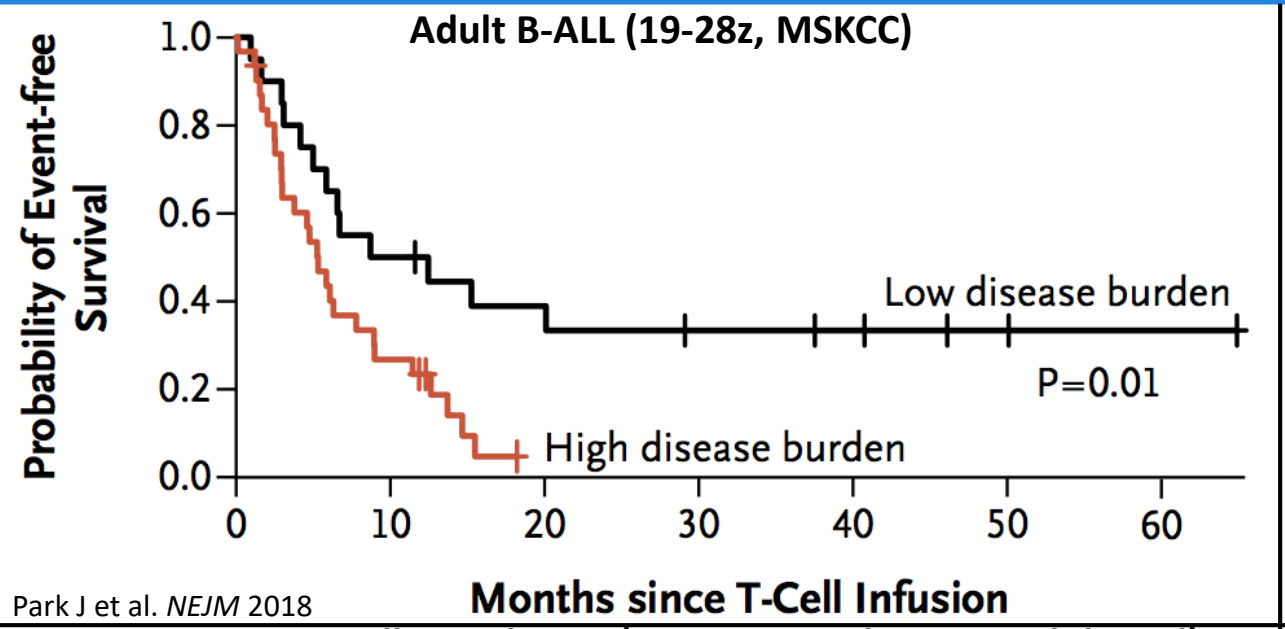


CAR T Cells have high ORRs, but relapses occur commonly in MM

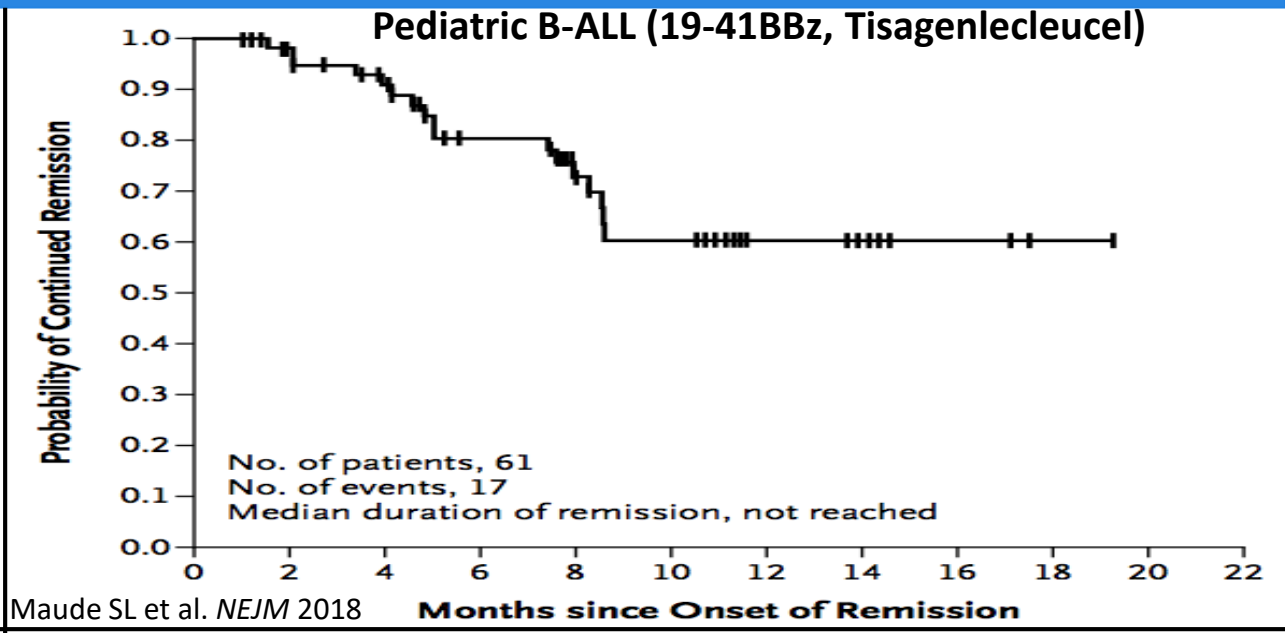


Raje N et al. *NEJM* 2019

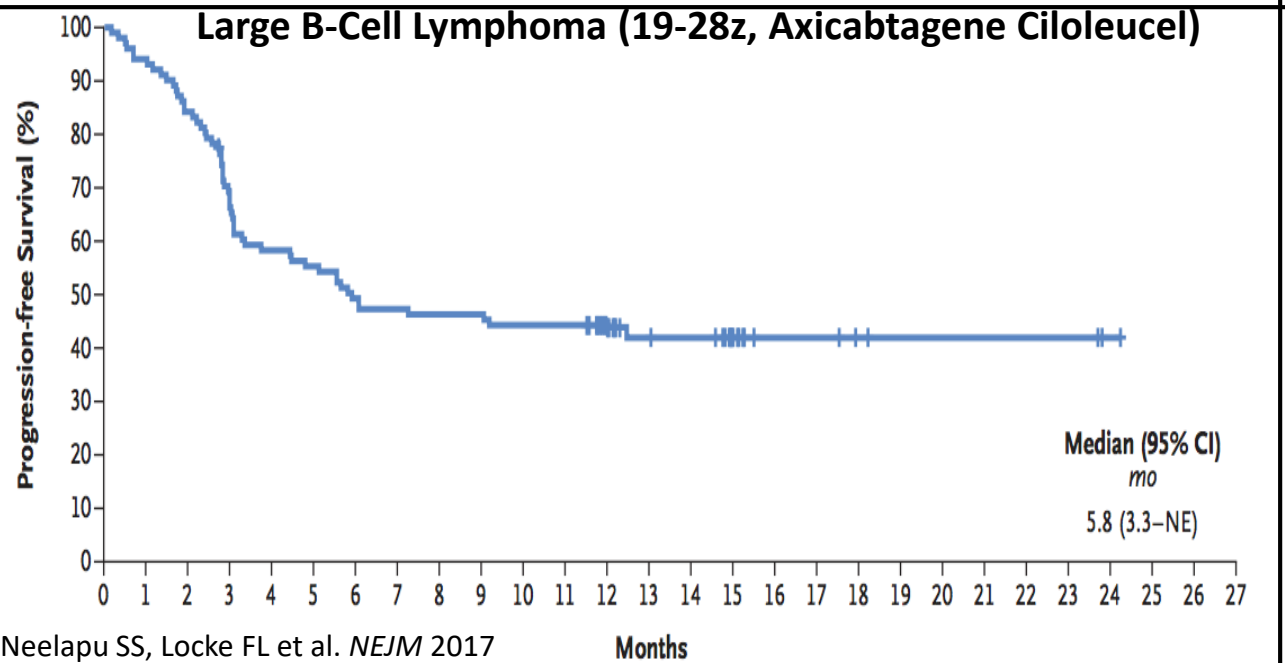
CAR T Cells have high ORRs, but relapses occur commonly in MM



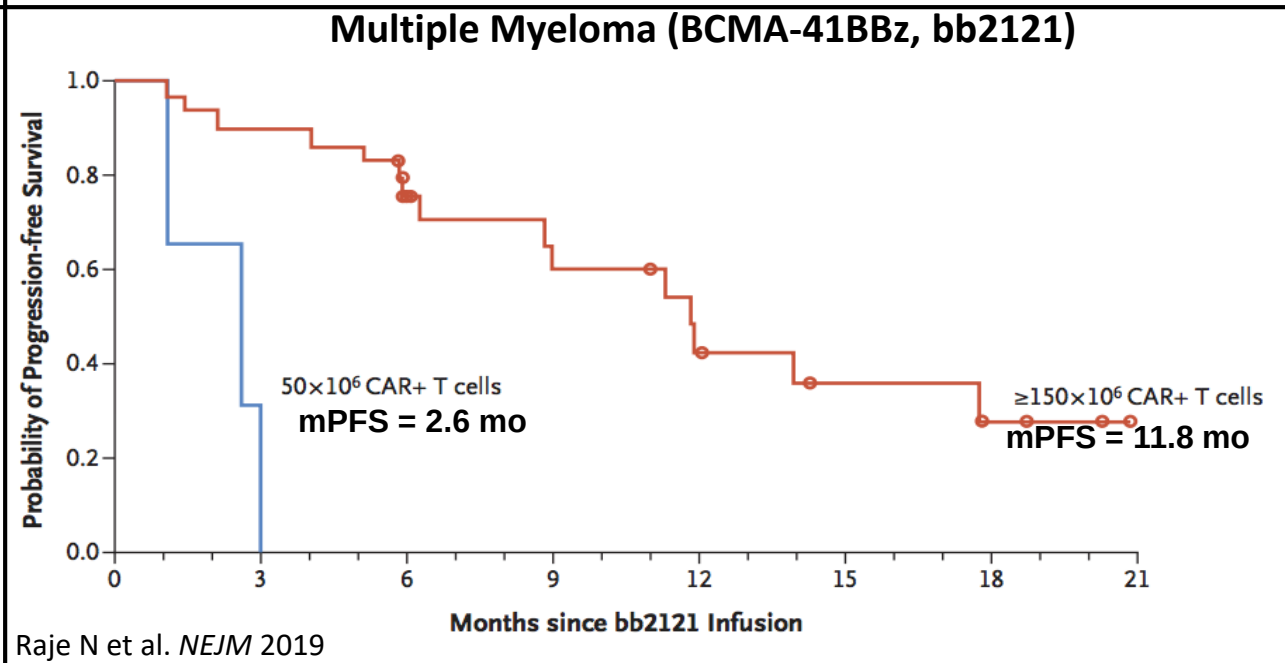
Park J et al. *NEJM* 2018



Maude SL et al. *NEJM* 2018



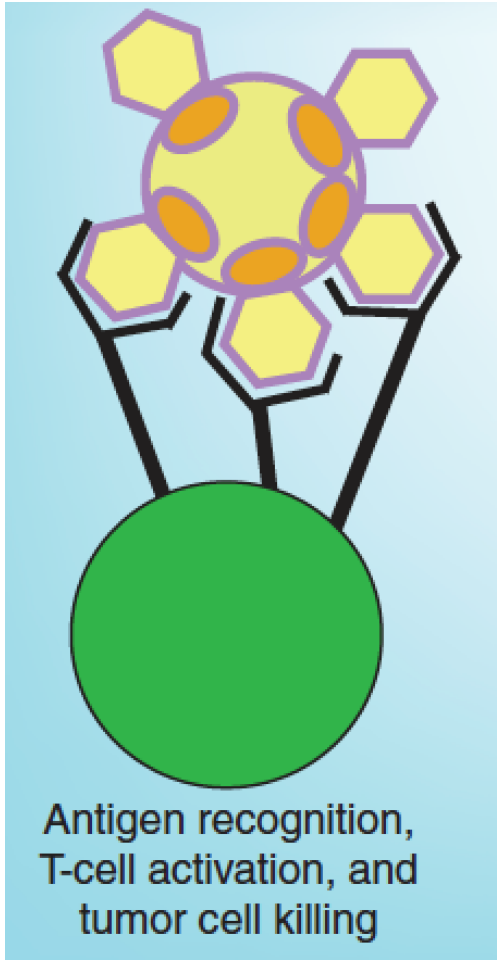
Neelapu SS, Locke FL et al. *NEJM* 2017



Raje N et al. *NEJM* 2019

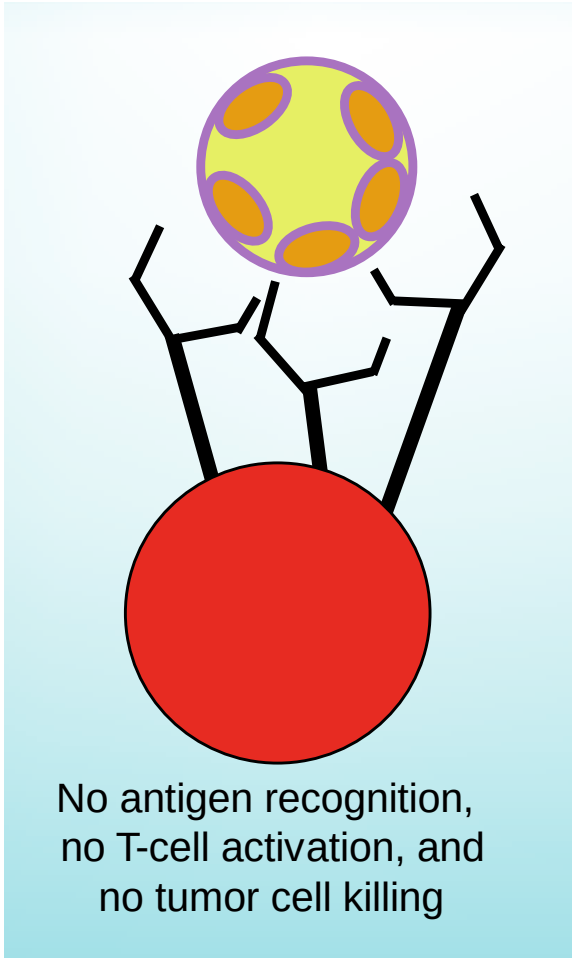
Antigen Escape Mediated Relapse

Activated CAR T cell



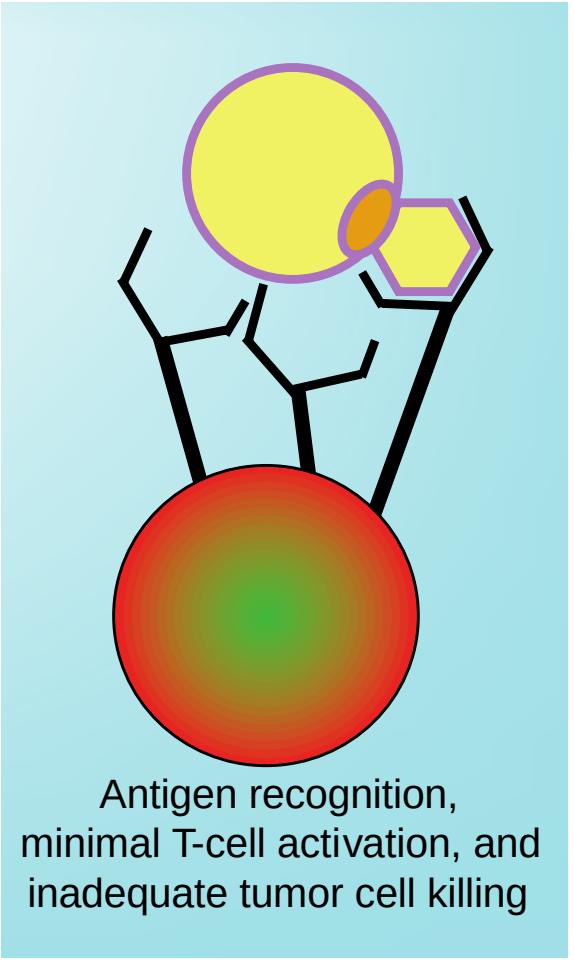
Antigen recognition, T-cell activation, and tumor cell killing

Antigen Negative



No antigen recognition, no T-cell activation, and no tumor cell killing

Antigen Downregulation



Antigen recognition, minimal T-cell activation, and inadequate tumor cell killing

Adapted from Majzner RG and Mackall CL. *Cancer Discovery* 2018.

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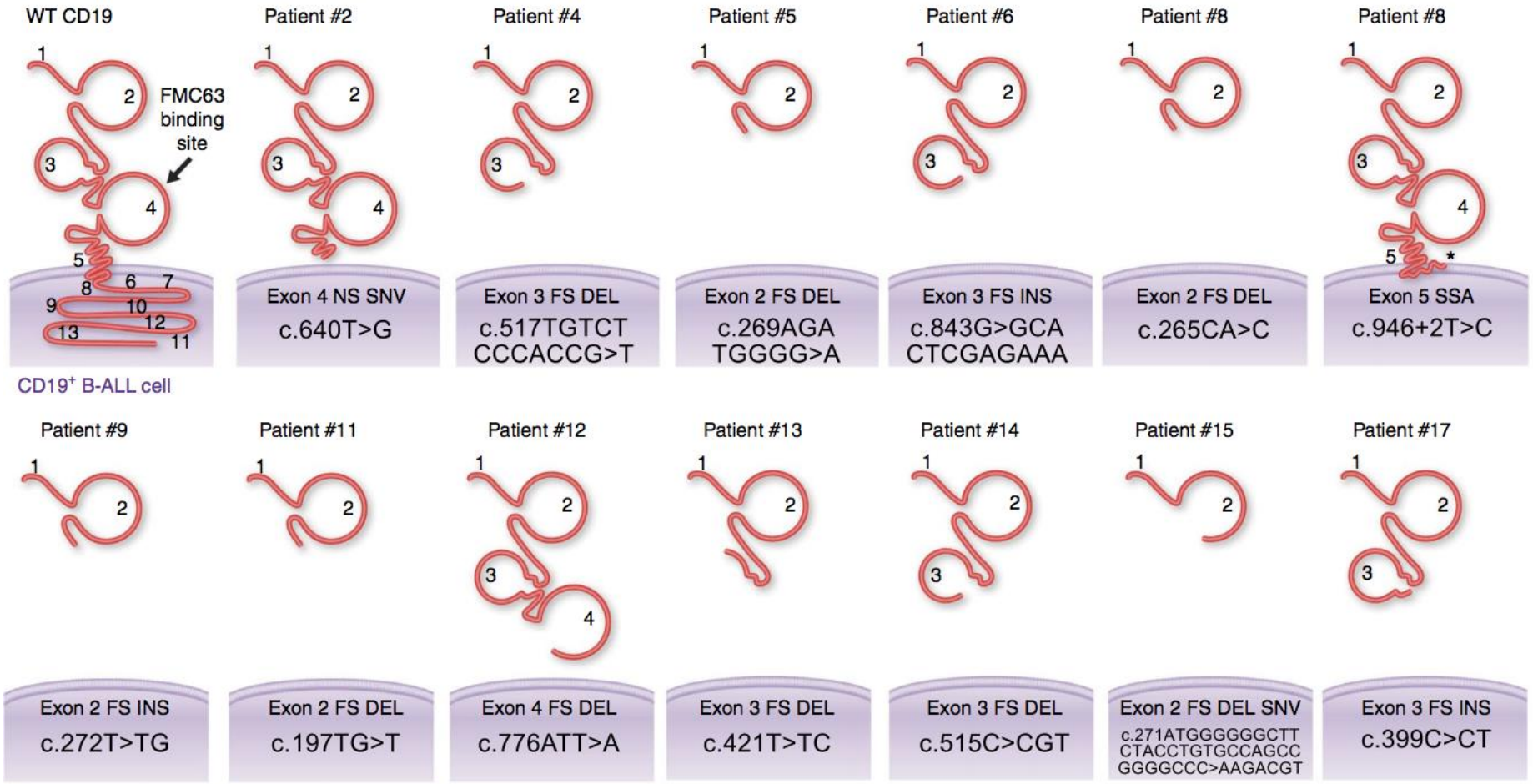
Antigen Escape: CD19-targeted CAR T cells for ALL

Trial	CAR	% relapses CD19 neg	Reference
CHOP	FMC63/4-1BBz	13/20 (65%)	Maude NEJM 2014, Maude JCO 2016
ELIANA (Novartis ph II)	FMC63/4-1BBz	15/16 (94%); 6 pts unknown CD19 status	Maude NEJM 2018
Seattle Children's	FMC63/4-1BBz	7/18 (39%)	Gardner Blood 2016
NCI	FMC63/CD28z	5/8 (63%)	Lee Lancet 2015, Lee Blood 2016
MSKCC	SJ25C1/CD28z	4/25 (16%)	Park NEJM 2018
Fred Hutch	FMC63/4-1BBz	2/9 (22%)	Turtle JCI 2016

Adapted from Majzner RG and Mackall CL. *Cancer Discovery*. 2018.

Antigen Escape: CD19-targeted CAR T cells for ALL



Orlando EJ et al. *Nat Med*. 2018

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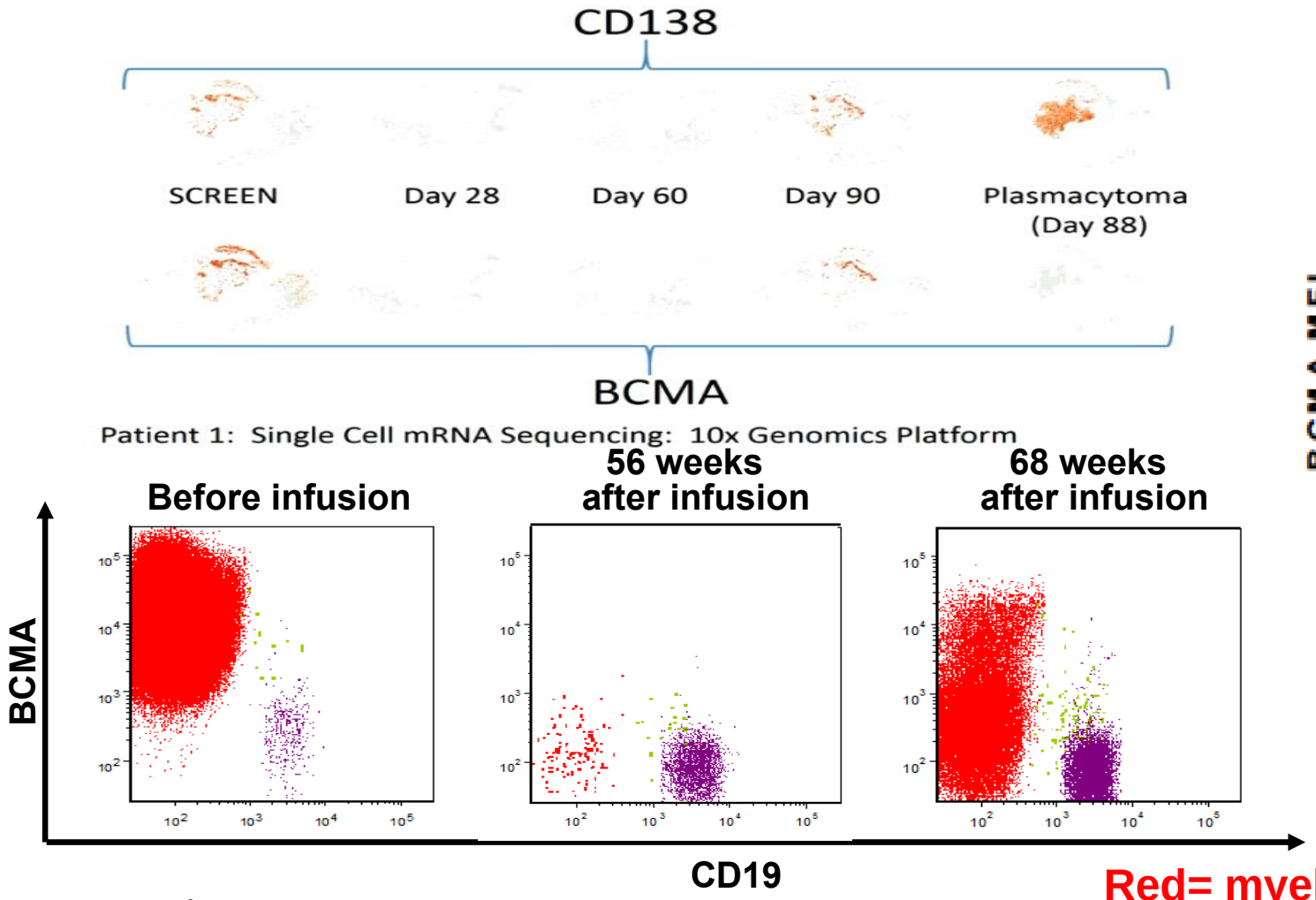
FCARH143 Bone Marrow Response Assessment

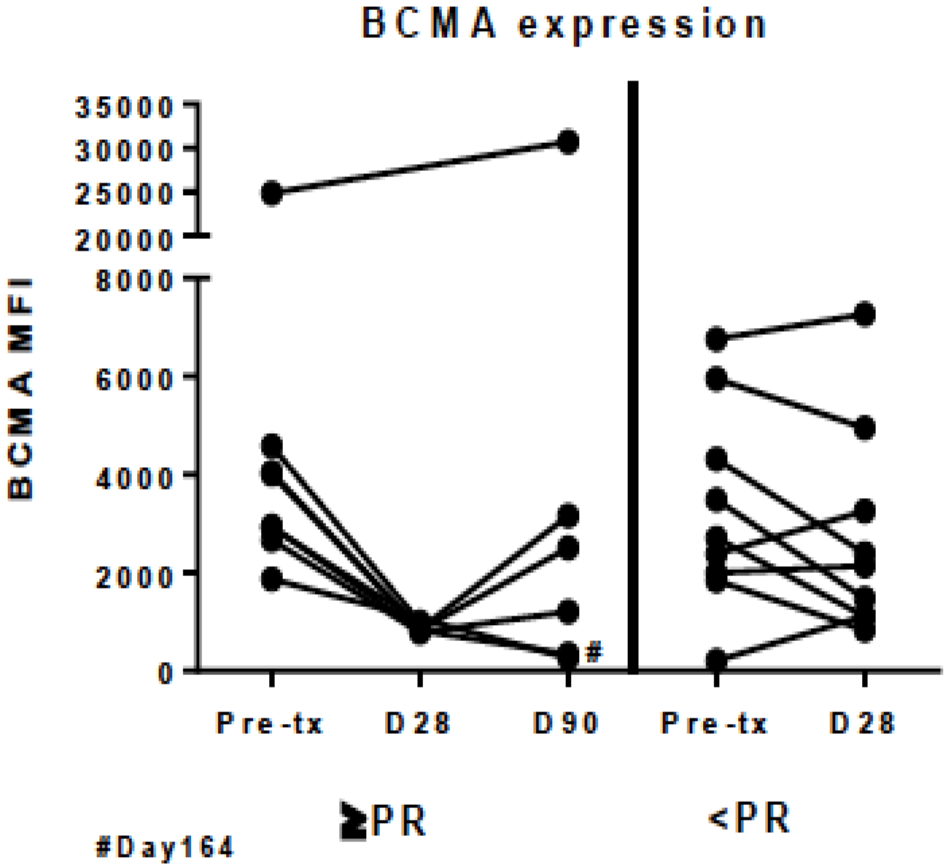
	50x10^6 CAR+							150x10^6 CAR+			
Patient	1	2*	3	4	5*	6*	7	8*	9	10	11
Pre	20%	70%	75%	60%	20%	20%	10-15%	50%	50%	50%	50%
D14	0%	<5%	30-40%	60%	0%	0%	0%	0%	0%	0%	0%
D28	**0%**	**0%**	**0%**	**0%**	**0%**	**0%**	**0%**	**0%**	**0%**	**0%**	**0%**
D60	20%	--	0%	0%	0%	0%	0%	0%	0%	0%	0%
Best Response	**PR D28**	**VGPR D60**	**sCR D28**	**CR D180**	**sCR D90**	**sCR D180**	**CR D28**	**VGPR D60**	**VGPR D60**	**VGPR D60**	**sCR D90**
Days on Study	79 RELAPSE	180 RELAPSE	356	329	217	182	132	219 RELAPSE	258	231	146

*CD4:CD8 ≠ 50:50 ± 15%

Updated from Green D et al. ASH 2018; do not post

Antigen Escape: BCMA-targeted CAR T cells for MM





Green D et al. *ASH*. 2018.
Brudno J et al. *JCO*. 2018.
Cohen A et al. *JCI*. 2019.

Red= myeloma plasma cells
Green=normal plasma cells
Purple=B cells

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Limitations of CAR T Cell Therapies (2019)

- Product made for each patient
  - Expensive manufacturing
  - Long wait times require bridging therapy; potential for rapid progression
  - Suboptimal cell fitness from heavily pre-treated patients
- Single effector gene (2nd generation CAR) does not address
  - Antigen escape
  - Immune suppressive TME
  - Recruitment of endogenous immune effectors

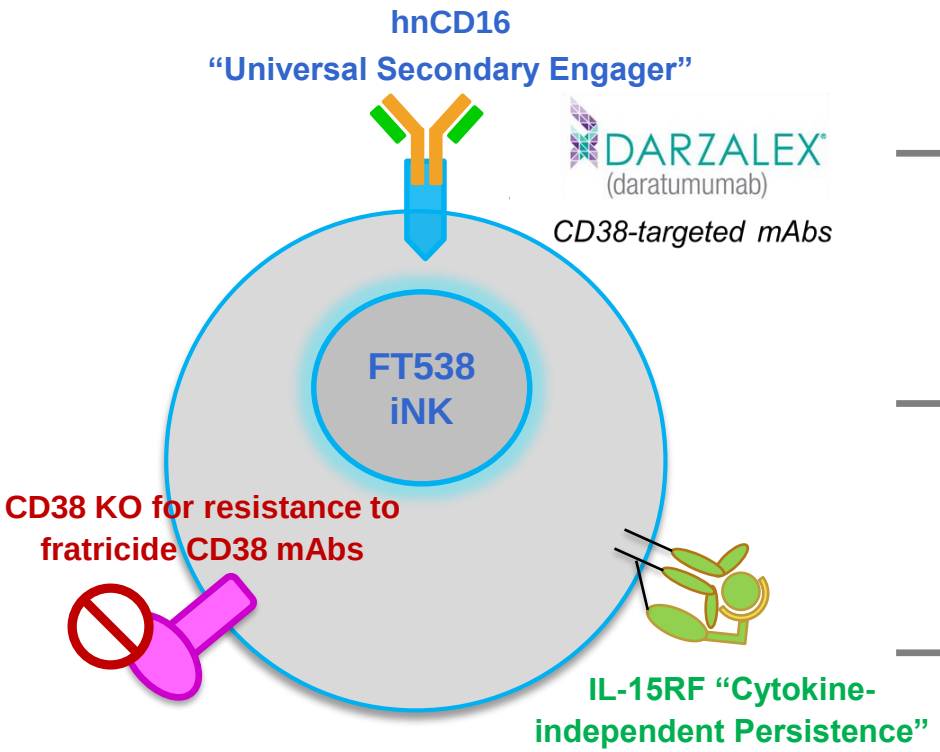
Multi-Antigen Targeting

Best-in-Class Therapeutic Strategy for Myeloma

FT538 Off-the-Shelf hnCD16 CD38- NK Cell Product Candidate

Combination with anti-CD38 mAb for Multiple Myeloma

Overcome Endogenous NK Cell Deficiencies for Optimized ant-CD38 Activity in Myeloma



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

---

CD38 KO: Deletion of CD38 to eliminate anti-CD38 antibody mediated NK cell fratricide. Also shown to improve NK cell biology and potency through optimization of metabolic signaling

---

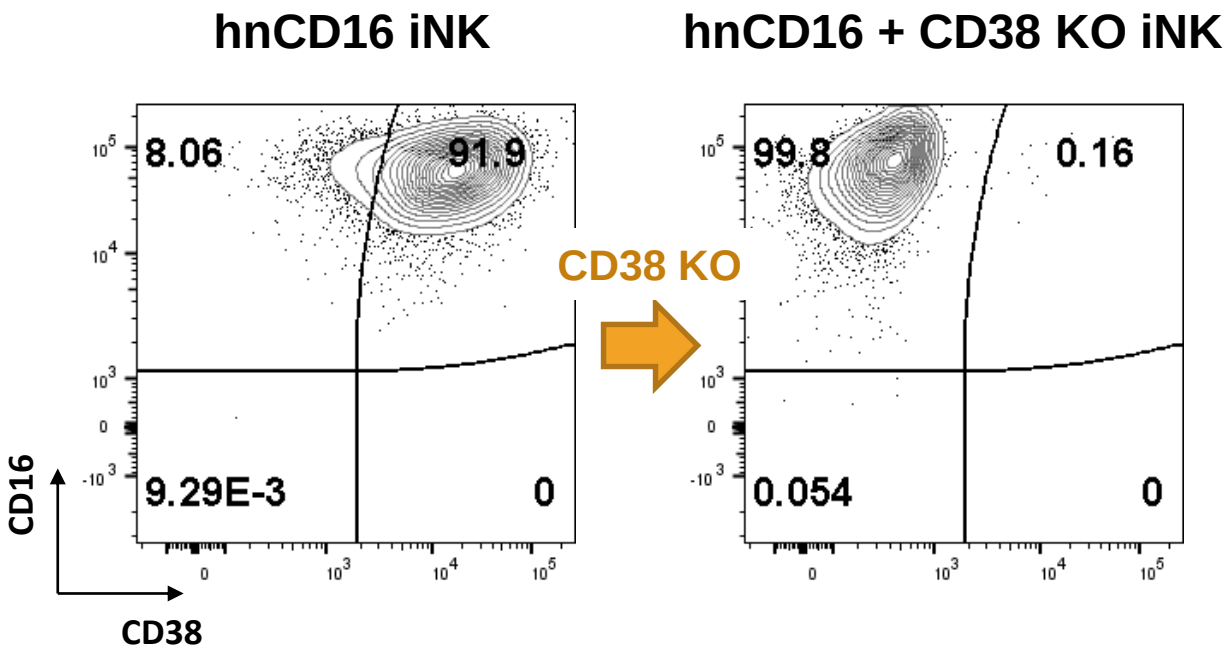
IL-15RF: Interleukin-15 receptor fusion, a potent cytokine complex that promotes survival, persistence and reduces the dependency for exogenous cytokine support

ASH Abstract #133: Off-the-Shelf NK Cell Immunotherapy with Targeted Disruption of CD38 to Prevent Fratricide and Enhance ADCC in Multiple Myeloma

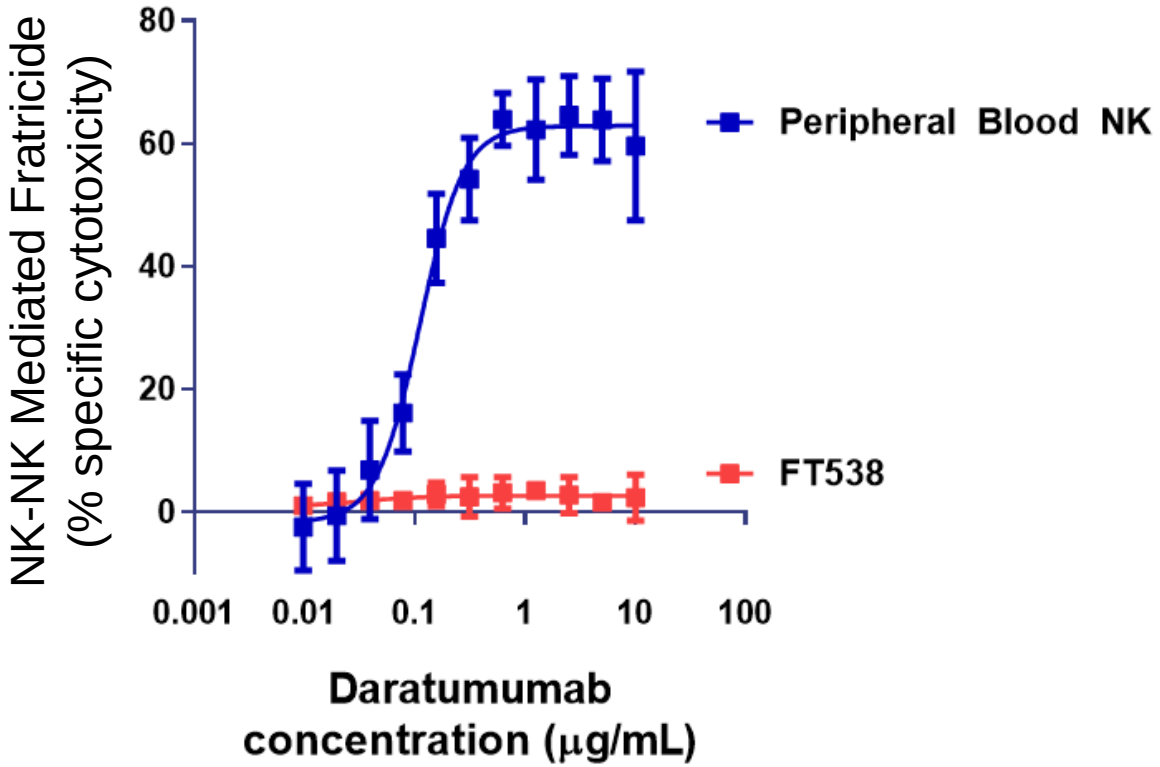
FT538 Off-the-Shelf hnCD16 CD38- NK Cell Product Candidate

Uniquely Designed to Avoid Fratricide Induced by anti-CD38 mAbs and Enhance ADCC

hnCD19 + CD38 KO



Eliminates mAb-induced NK Cell Fratricide



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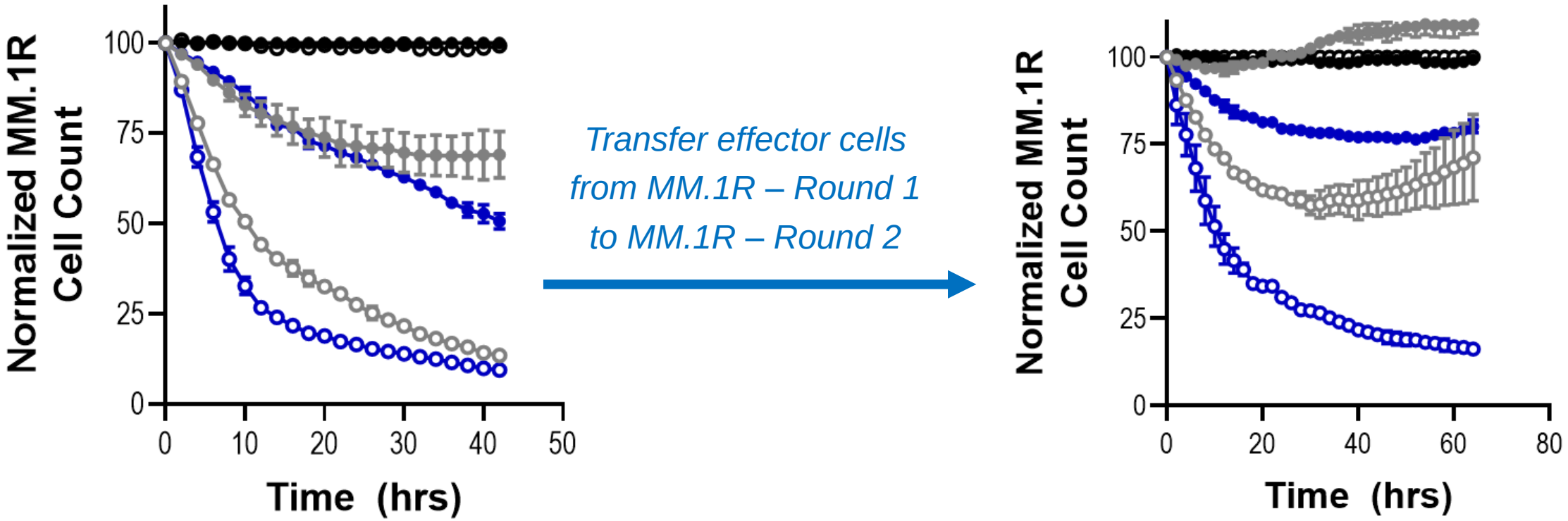
FT538 Off-the-Shelf hnCD16 CD38- NK Cell Product Candidate

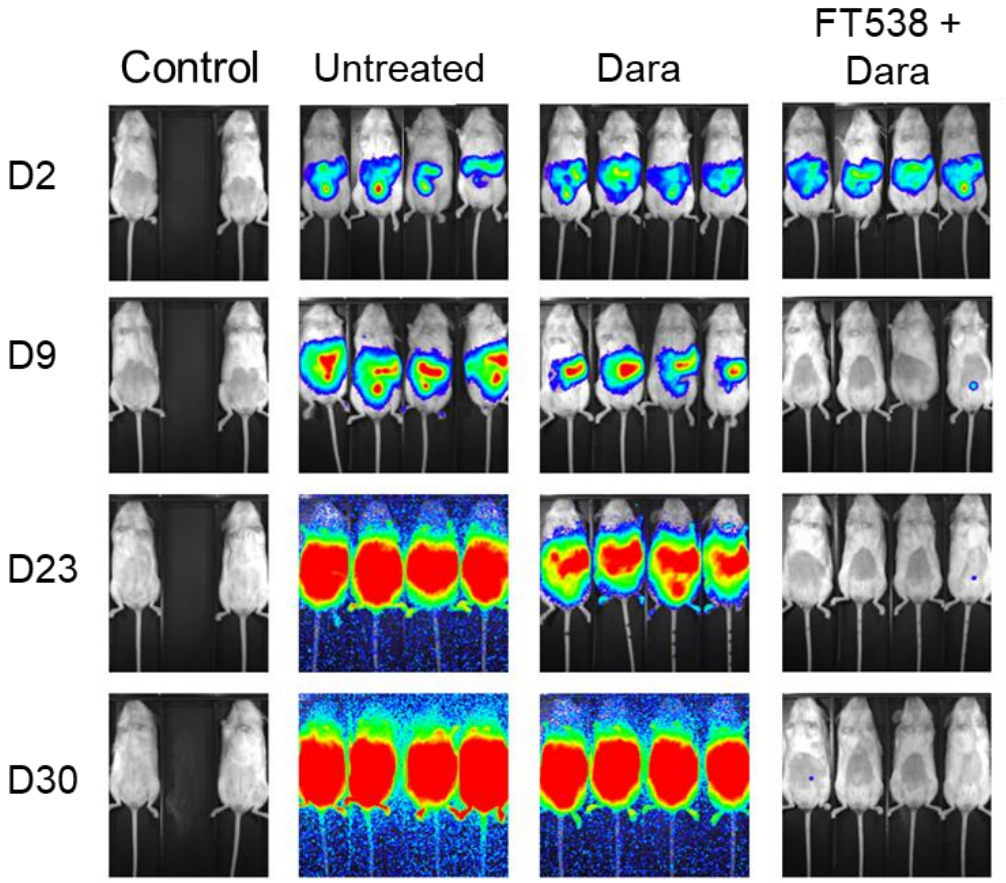
Enhanced Cytotoxicity vs. PB NK Cells in a Serial Stimulation Cytotoxicity Assay

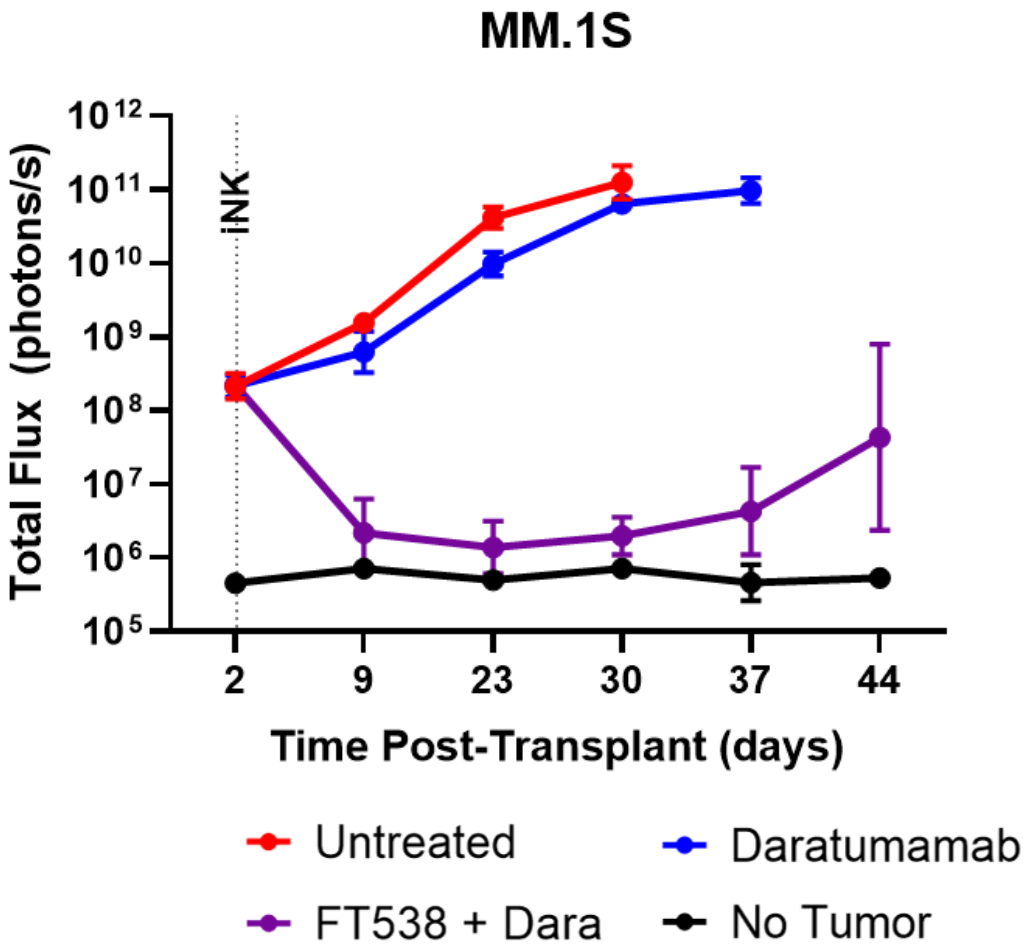
Overcome Endogenous NK Cell Deficiencies for Optimized ant-CD38 Activity in Myeloma



FT538 Off-the-Shelf hnCD16 CD38- NK Cell Product Candidate

Enhanced ADCC in Combination with anti-CD38 mAb In Vivo





In-license of Novel BCMA Binding Domain from Max Delbrück Center

Building on FT538 for Best-in-Class Multi-antigen Targeting Strategy in Multiple Myeloma

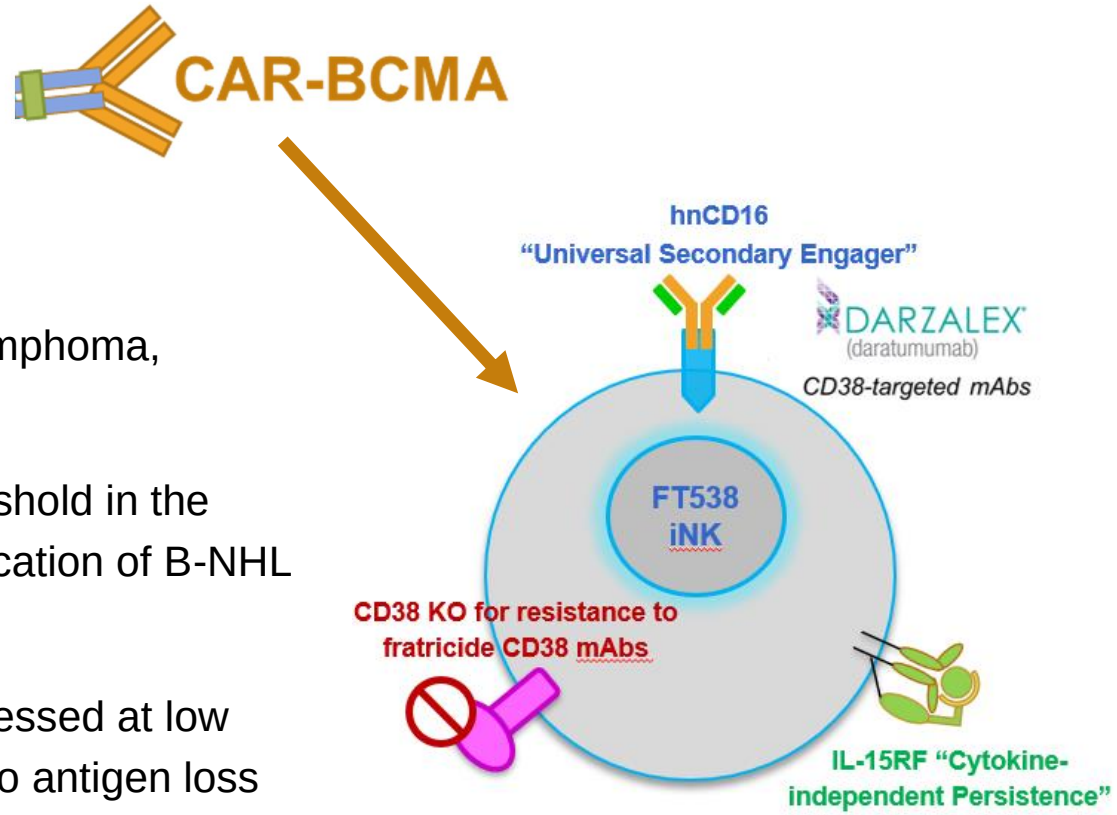
Molecular Therapy
Original Article

AMERICAN SOCIETY of GENE & CELL THERAPY

CAR T Cells with Enhanced Sensitivity to B Cell Maturation Antigen for the Targeting of B Cell Non-Hodgkin's Lymphoma and Multiple Myeloma

Julia Bluhm,[1] Elisa Kieback,[1] Stephen F. Marino,[2] Felix Oden,[1] Jörg Westermann,[3] Markus Chmielewski,[4] Hinrich Abken,[4] Wolfgang Uckert,[1] Uta E. Höpken,[1] and Armin Rehm[1]

- ✓ Validated CAR BCMA in diffuse large B cell lymphoma, follicular lymphoma, mantle cell lymphoma, and chronic lymphocytic leukemia
- ✓ BCMA CAR T cells triggered target cell lysis with an activation threshold in the range of 100 BCMA molecules, which allowed for an efficient eradication of B-NHL cells in vitro and in vivo
- ✓ Potential novel therapeutic option for patients where BCMA is expressed at low abundance or where anti-CD19 immunotherapies have failed due to antigen loss

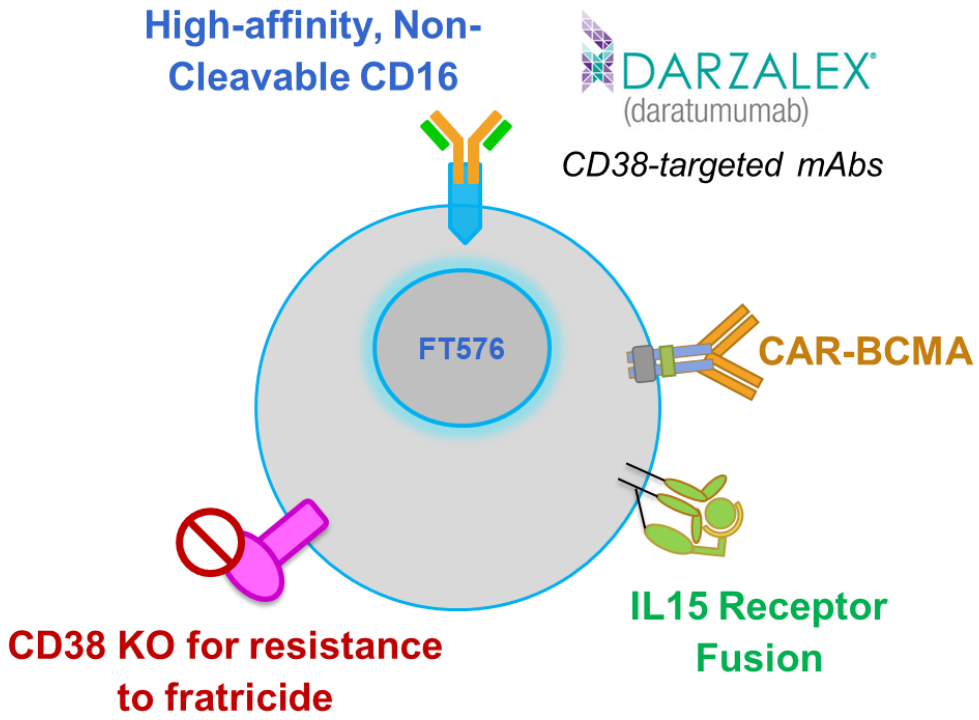


Fate THERAPEUTICS

FT576 Off-the-Shelf Multi-Targeted CAR-BCMA NK Cell Product Candidate

Potential Best-in-Class Cell-based Cancer Immunotherapy for Multiple Myeloma

Adoptive CAR NK cell Therapy Engineered with Four Anti-tumor Modalities



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

---

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

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

---

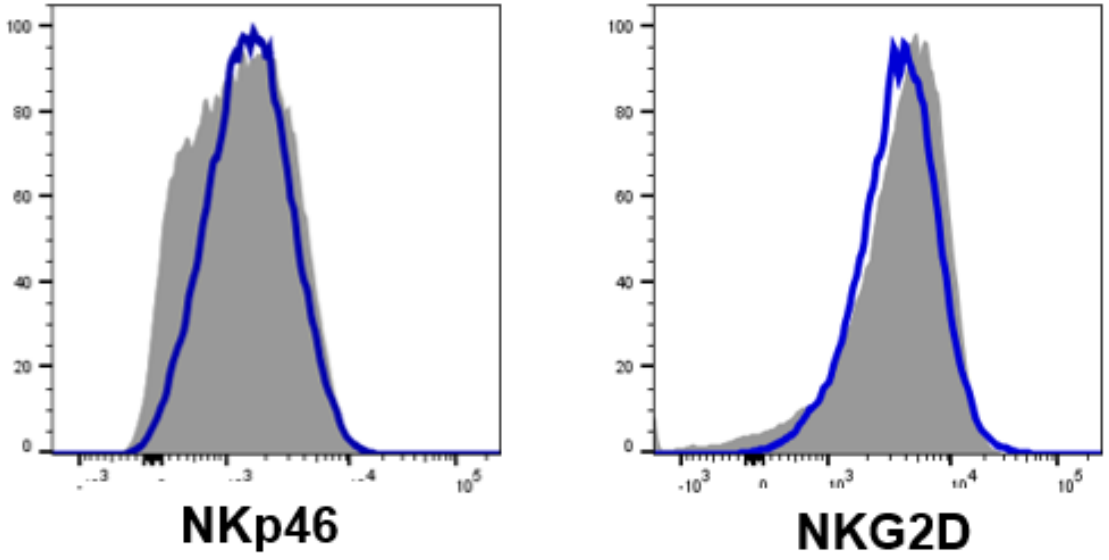
CD38 KO: Deletion of CD38 to eliminate anti-CD38 antibody mediated NK cell fratricide. Also shown to improve NK cell biology and potency through optimization of metabolic signaling

ASH Abstract #3214: A Novel Multiplexed Engineered Off-the-Shelf NK Cell Immunotherapy for the Dual-Targeting of CD38 and BCMA for Multiple Myeloma

FT576 Off-the-Shelf Multi-Targeted CAR-BCMA NK Cell Product Candidate

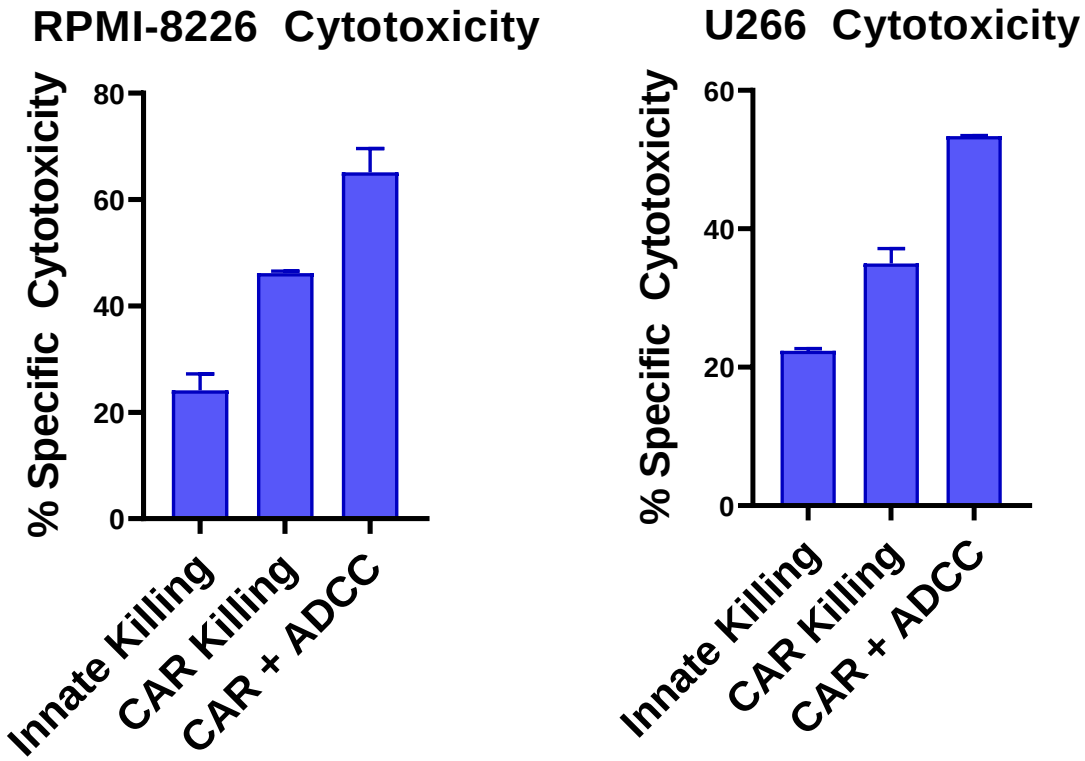
CAR-BCMA Modality Displays In Vitro Cytotoxicity

Activated NK Cell Phenotype



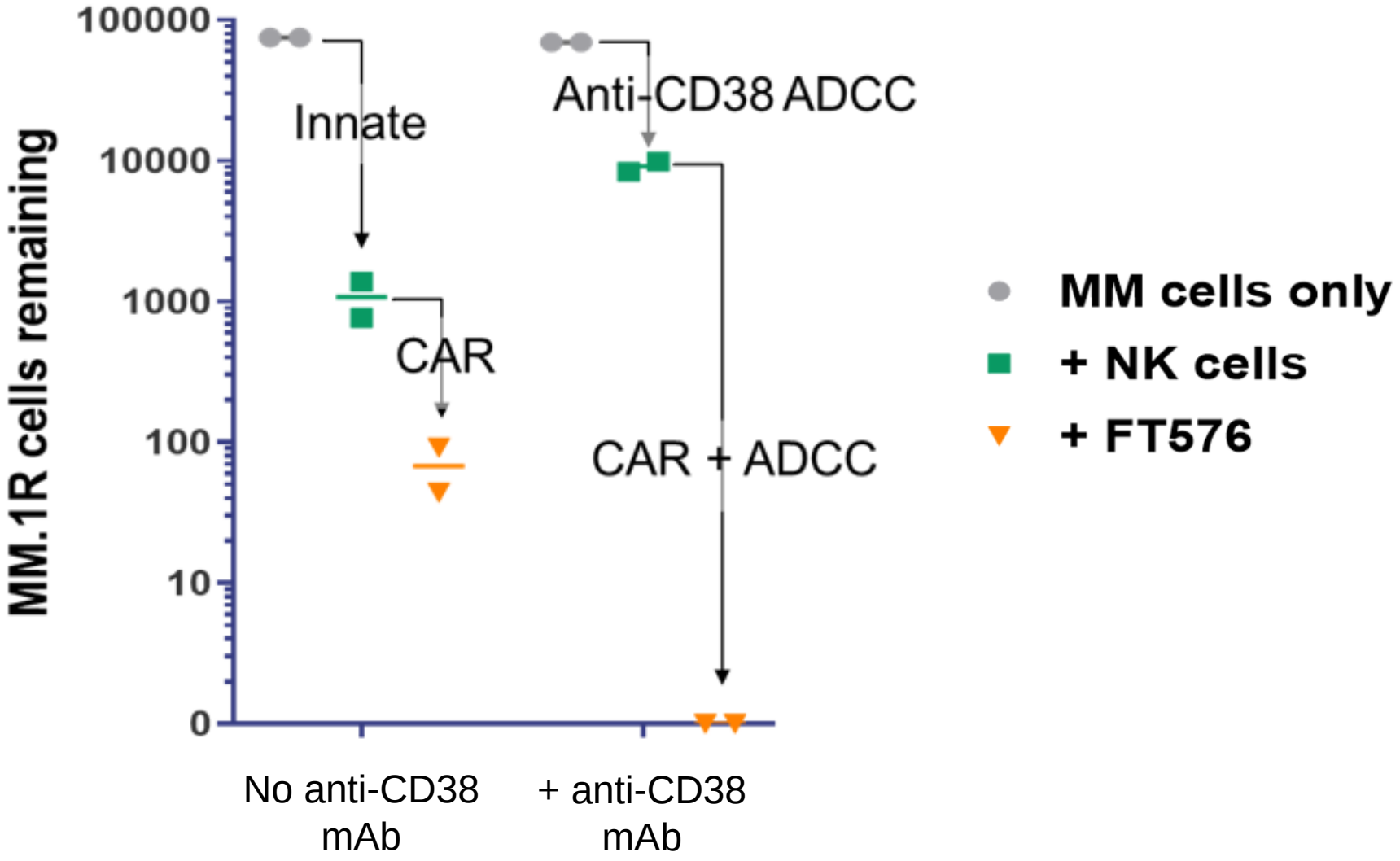
Peripheral Blood NK Cells FT576

Compounded Effect of FT576



FT576 Off-the-Shelf Multi-Targeted CAR-BCMA NK Cell Product Candidate

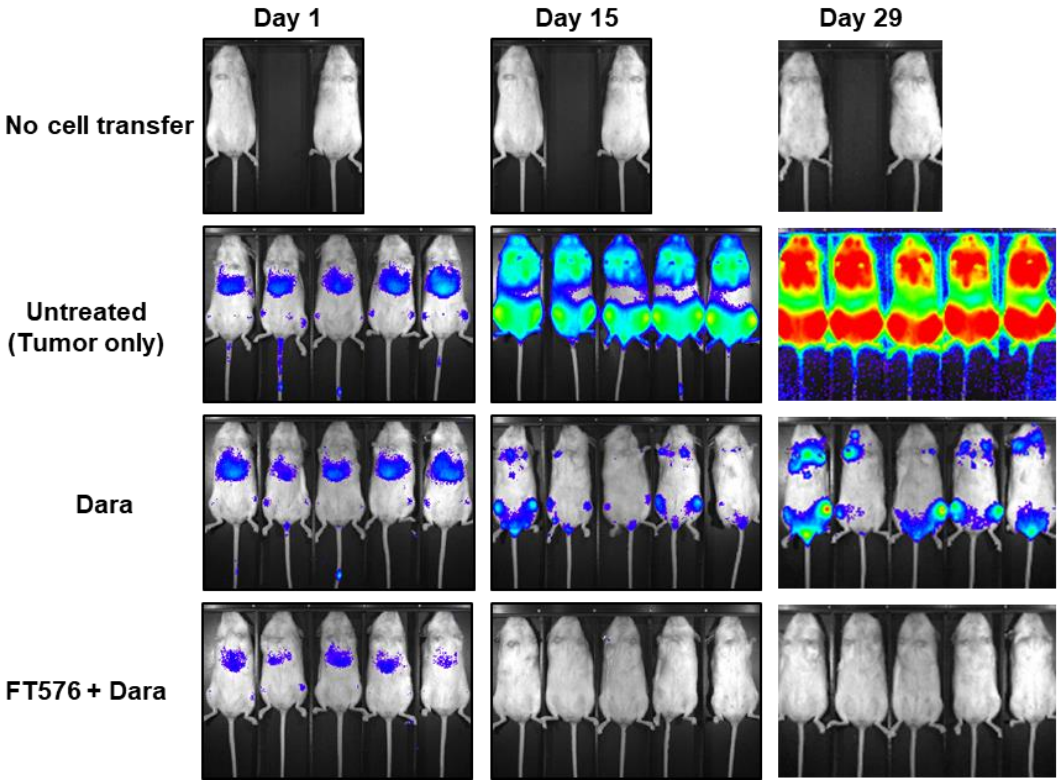
Displays a Unique and Potent Multi-antigen Targeting Capacity

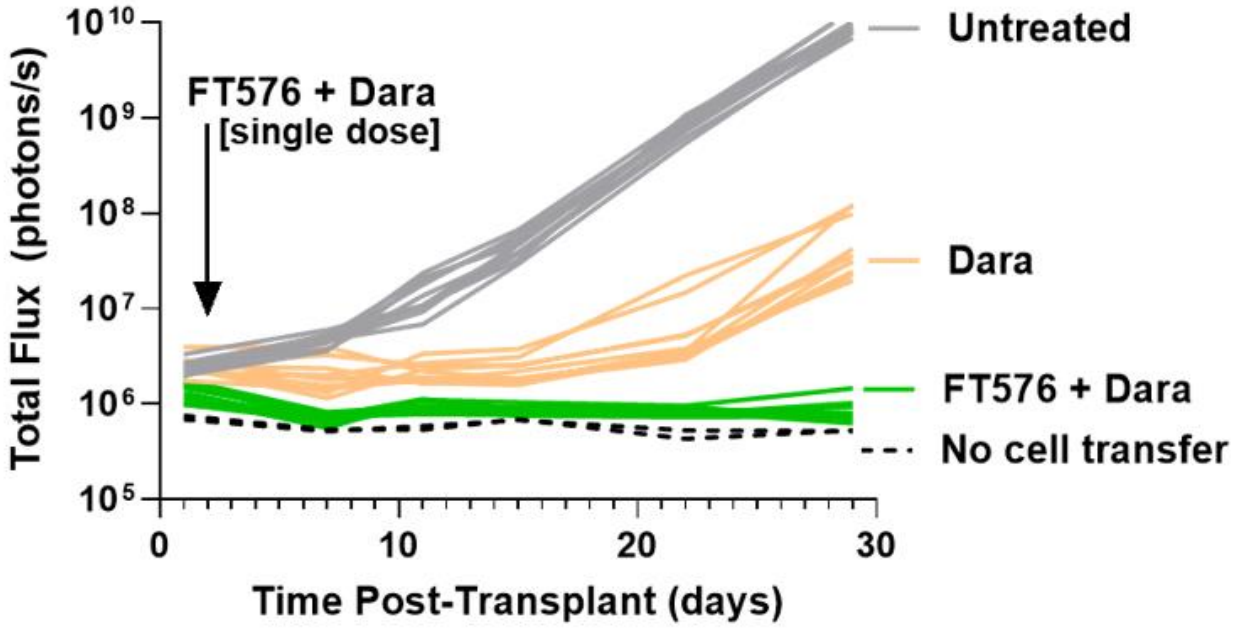


FT576 Off-the-Shelf Multi-Targeted CAR-BCMA NK Cell Product Candidate

Synergizes with Daratumumab to Effectively Eliminate Tumor Burden

Disseminated Xenograft Model of Multiple Myeloma





iPSC Product Innovation

iPSC Product Platform

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

Single iPSC Clone

(Engineered) Single Pluripotent Stem Cell

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

Expansion & Banking

Unlimited Supply of Clonal Master iPSC 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

"to reach more patients in need"

Fate THERAPEUTICS

Better Cells For Better Therapies™