Fate THERAPEUTICS

Better Cells For Better Therapies™

Programmed Cellular Immunotherapies

Leading the Development of Off-the-Shelf Cell-based Cancer Immunotherapies using Clonal Master Engineered iPSC Lines

June 2021

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.

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Changing the Game in Cell Therapy

iPSC-derived, Off-the-Shelf Cell Therapies to Eradicate Cancer

Multiplexed Engineering

Incorporate multiple mechanisms of action to eradicate cancer

Mass Production

Reliable manufacturing process with high yield at low cost per dose

Uniform Products

Consistent identity, purity and potency of cell products

Off-the-Shelf

Stable, cryopreserved for on-demand treatment and expanded patient reach

Treatment Paradigm

Flexible out-patient treatment strategies to drive deep responses

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Unique Challenges Confronting the Cell-based Cancer Immunotherapy Field

Key Considerations in Developing Best-in-Class Cell Products

1. ***Starting Cell Source***. Whether from the patient (autologous) or a healthy donor (allogeneic), the starting cell source is variable and can create batch-to-batch inconsistencies.

2. ***Cell Engineering***. A critical component of each and every manufacturing run performed at a cell population level, which is costly and creates batch-to-batch and cell-to-cell variability.

3. ***Cell Expansion***. A critical component of each and every manufacturing run required to achieve large numbers of cells, which can impact product viability and potency.

4. ***Product Profile***. The safety, tolerability, dose and efficacy of the product, including its potential to be effectively used with other standard-of-care therapies.

5. ***Therapeutic Reach***. The overall patient experience, treatment setting, and cost-effectiveness, including the potential to reach patients earlier in care.

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Master Cell Lines Enable Mass Production of Best-in-Class Cell Products

Transitioning the Field from a Process-centric to a Product-centric Therapeutic Paradigm

Key Features	Cell Therapy 1.0 and 2.0	Cell Therapy 3.0
Cell Source	Patient and Donor Cells	Renewable Master Cell Line
Genetic Engineering	Random & Variable	Uniform & Consistent
Characterization	Imprecise	Well-defined
Product Identity	Heterogeneous	Homogeneous
Manufacturing	Low Yield-to-Cell Dose Ratio	High Yield-to-Cell Dose Ratio
Packaging	Fresh / Short Shelf Life	Cryopreserved / Long Shelf Life
Dosing	Single Dose	Multiple Doses
Delivery	Complex Logistics	Off-the-Shelf
Overall Paradigm	**Process-centric**	**Product-centric**

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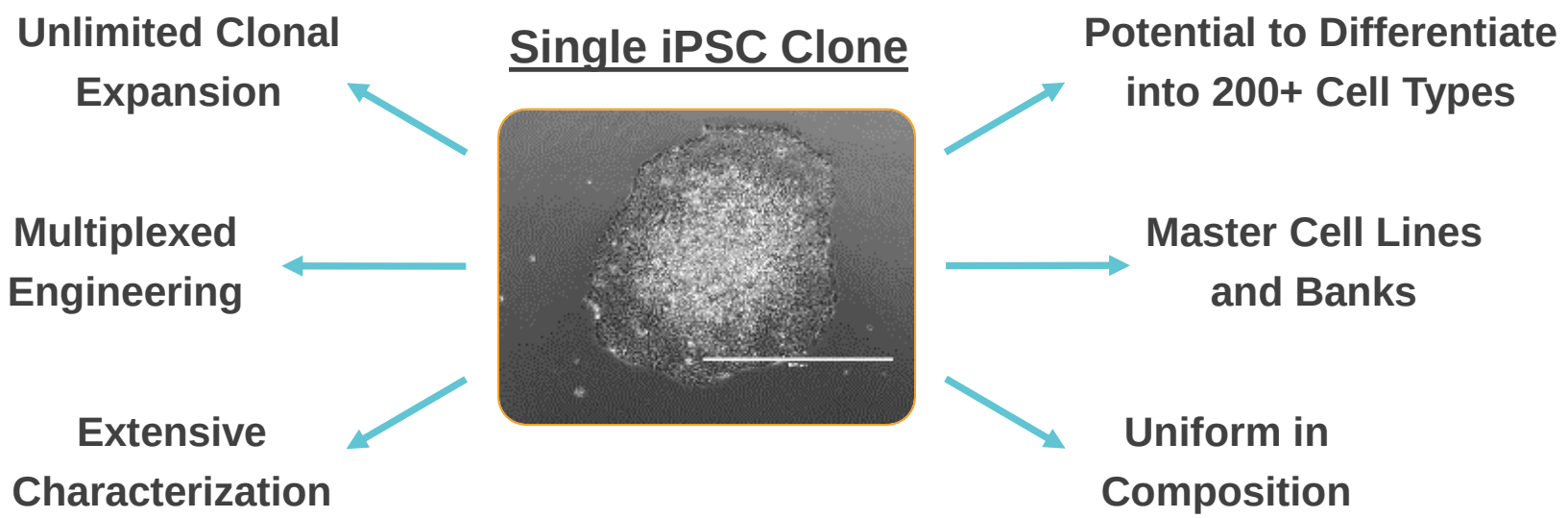
Unique Biological Properties of Human iPSCs

Single-cell Isolation, Characterization & Selection for Creation of Master Engineered Cell Lines

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 300+ 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

Induced Pluripotent Stem Cells

Multiplexed Gene Engineering (one-time event)

Single-Cell Sorting & Clonal Selection

iPSC Expansion & Banking

Clonal Master Engineered iPSC Bank

Renewable Starting Cell Source

'On-Demand' Delivery to Reach More Patients

iT Cells

iNK Cells

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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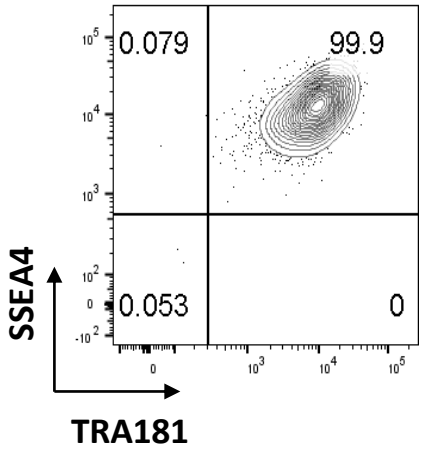
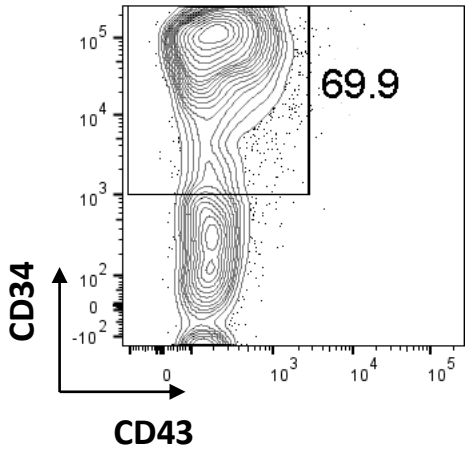
iPSC Product Platform

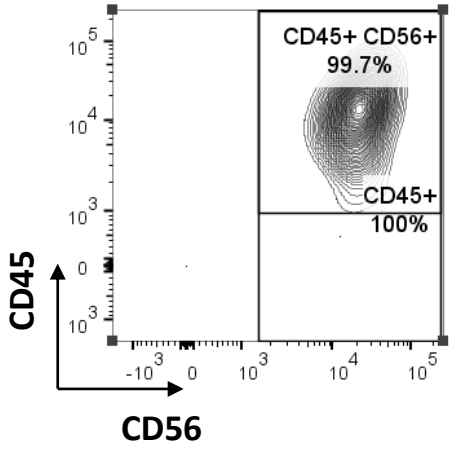
Robust In-house GMP Manufacture of Cryopreserved Off-the-Shelf Cell Products

iPSCs → **iCD34s** → **iNKs**

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







> 1 million-fold expansion

10^6 iPSCs

> 10^{12} iNKs

- *Homogeneous cell product*
- *100s-1,000s doses per campaign*
- *Low-cost per dose cGMP production*
- *Cryopreserved*
- *High post-thaw viability and potency*

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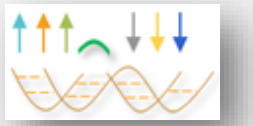
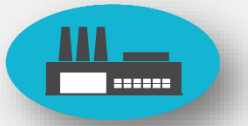
iPSC Product Platform

The Leading Developer of iPSC-derived Cell-based Cancer Immunotherapies



Disruptive Technology Platform: highly-edited master iPSC lines; worked closely with FDA to pioneer first-ever clinical investigation in U.S. of iPSC-derived cell therapy



Scalable Manufacture: demonstrated ability to manufacture 100s of cryopreserved doses of uniform product in single manufacturing campaign at low cost per dose

Leading Off-the-shelf NK- & T-cell Pipeline: multiple P1 programs addressing unmet medical needs in AML, Lymphoma / CLL, Multiple Myeloma and Solid Tumors

Demonstrated Clinical Benefit: treated 80+ late-stage patients with novel, multi-dose treatment paradigm showing differentiated safety profile and compelling therapeutic benefit

World Class Partnerships: creating innovative iPSC-derived NK- and T-cell therapies with Janssen, Ono Pharmaceutical, University of Minnesota and Memorial Sloan Kettering

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Off-the-Shelf, iPSC-derived NK Cell Cancer Immunotherapy Franchise

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

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

Clonal Master iPSC Line	Synthetic Biology	FT500	FT516	FT596	FT538	FT576	FT536	FT573
Multi-faceted Innate Immunity		✓	✓	✓	✓	✓	✓	✓
+ High-affinity, non-cleavable CD16	*Augment mAb therapy*		✓	✓	✓	✓	✓	✓
+ IL-15 Receptor Fusion	*Enhance NK cell function*			✓	✓	✓	✓	✓
+ CAR Insertion	*Target tumor antigens*			CD19		BCMA	MICA/B	B7H3
+ CD38 Knock-out	*Enhance metabolic fitness*				✓	✓	✓	✓
	# of Synthetic Elements	**0**	**1**	**3**	**3**	**4**	**4**	**4**
	Clinical Stage	**P1**	**P1**	**P1**	**P1**	**SS**	**PC**	**PC**

P1 = Phase 1; SS = Phase 1 study start-up; PC = preclinical

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B-cell Malignancy Franchise

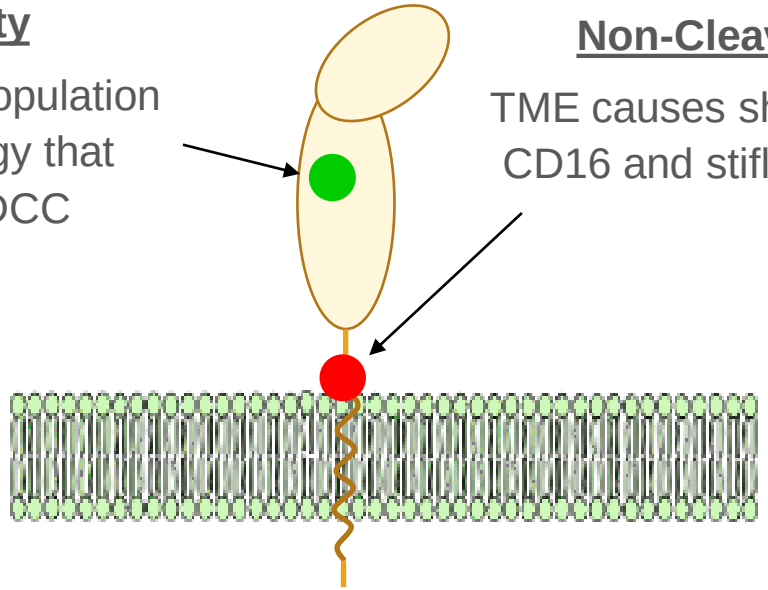
Novel High-Affinity, Non-Cleavable CD16a Fc Receptor

Optimizing Antibody-Dependent Cellular Cytotoxicity for Use with mAb Therapy

Novel High-affinity, Non-cleavable CD16 (hnCD16) Fc Receptor for Enhanced ADCC

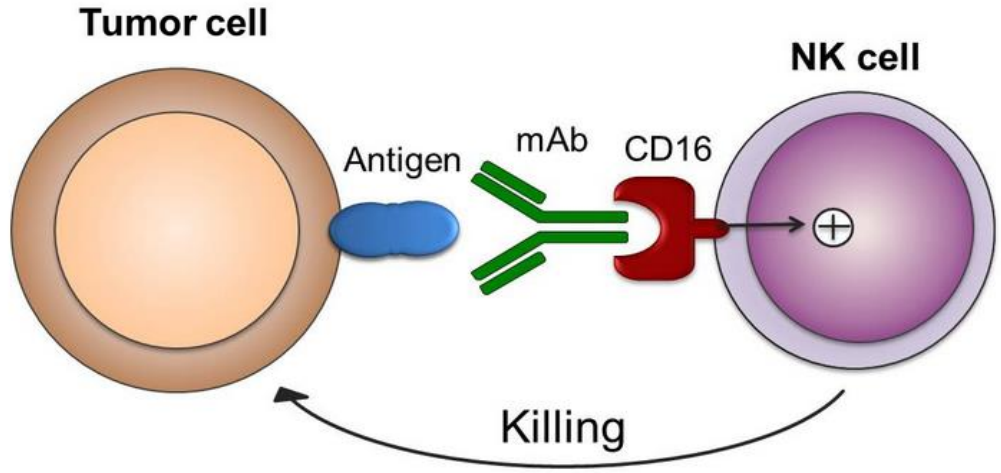
High Affinity

Only 15% of the population has CD16 biology that maximizes ADCC



Non-Cleavable

TME causes shedding of CD16 and stifles ADCC



Issued patents covering composition of matter of mammalian cells incorporating hnCD16 receptor

Rituxan Rituximab

GAZYVA obinutuzumab

DARZALEX (daratumumab)

Herceptin trastuzumab Precision • Power • Promise

ERBITUX Cetuximab

BAVENCIO avelumab Injection

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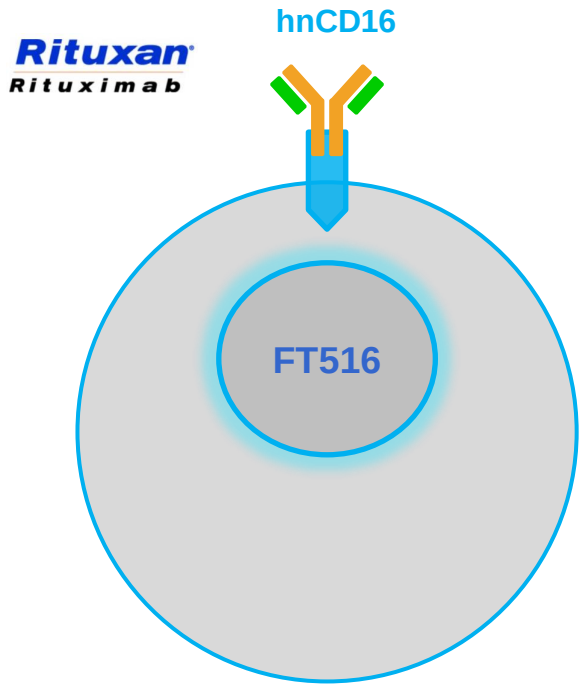
FT516 & FT596: First-in-Class NK Cell Cancer Immunotherapies

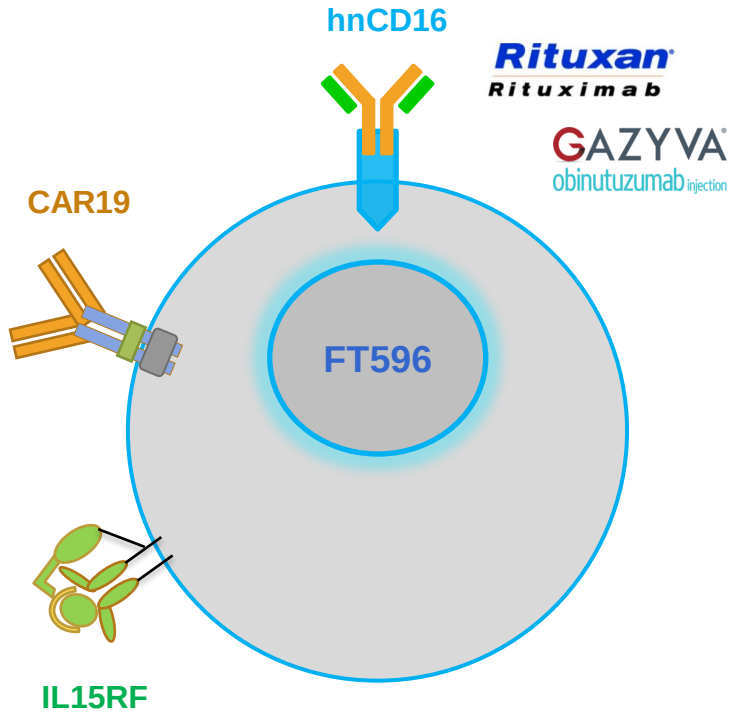
Optimized Innate & Synthetic Biology



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

CAR19: Chimeric antigen receptor that targets B-cell antigen CD19 (optimized for NK cells)

IL-15RF: Interleukin-15 receptor fusion to promote survival, proliferation and trans-activation of NK cells and CD8 T cells



Fc = fragment crystallizable; ADCC = antibody-dependent cellular cytotoxicity

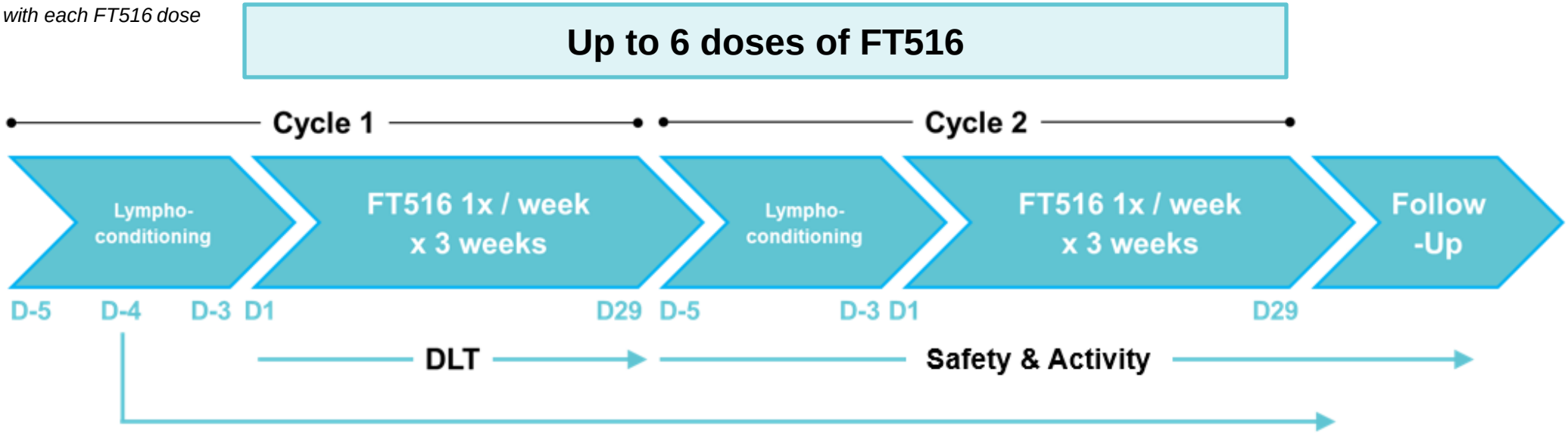
FT516-101: B-Cell Lymphoma in Combination with Rituximab

Phase 1 Study – Multiple Doses over Multiple Cycles in Out-patient Setting

Cyclophosphamide: 500 mg/m2 IV x 3 days

Fludarabine: 30 mg/m2 IV x 3 days

IL-2: 6M units sc with each FT516 dose



Rituximab: 1 dose at 375 mg/m^2 IV per cycle

<u>Regimen B</u> – 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

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FT516-101: B-Cell Lymphoma in Combination with Rituximab

Phase 1 Study – Interim Safety, Tolerability, and Response

Interim Data from FT516 Phase 1 Study in Relapsed / Refractory B-cell Lymphoma						
		Prior Systemic Therapy				
Subject #	Lymphoma Type	# Prior Regimens	# Prior CD20-Targeted	Prior CD19 CAR T	Most Recent Prior Response	FT516 Response[1]
Dose Cohort 2 – 90 million cells / dose						
2005	DLBCL	3	2	Y	Refractory	**CR**
2006	DLBCL	2	2	N	Relapsed	**PR**
2007	DLBCL (DH)	3	3	Y	Relapsed	PD
2012	iNHL	1	1	N	Relapsed	**CR**
Dose Cohort 3 – 300 million cells / dose						
2008	FL	6	6	N	Relapsed	**CR**
2009	DLBCL (DH/DE)	4	3	Y	Relapsed	PD
2010	FL	4	2	N	Relapsed	**CR**
2011	Transformed iNHL	4	2	N	Refractory	**PR**
2013	DLBCL	2	2	N	Refractory	**CR**
2014	HGBCL	1	1	N	Refractory	PD
2015	HGBCL (TH)	7	5	Y	Refractory	**CR**

As of March 11, 2021 database entry. Data subject to source document verification.

CR = Complete Response; **PR** = Partial Response; **PD** = Progressive Disease

CAR = Chimeric antigen receptor; **DH/DE** = Double-hit / double expressor; **DLBCL** = Diffuse large B-cell lymphoma; **FL** = Follicular lymphoma; **Gr** = Grade; **HGBCL** = High-grade B-cell lymphoma; **iNHL** = Indolent non-Hodgkin lymphoma; **TH** = Triple-hit; **Transformed iNHL** = Aggressive B-cell lymphoma transformed from iNHL

[1] Cycle 2 Day 29 protocol-defined response assessment per Lugano 2014 criteria

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FT516-101: B-Cell Lymphoma in Combination with Rituximab

Interim Clinical Observations

- Outpatient treatment paradigm of up to 6 doses was well-tolerated
  - No events *of any grade* of CRS, ICANS, or GvHD
  - No FT516-related SAEs or FT516-related Grade ≥3 AEs
  - No requirement for patient matching; no evidence of anti-product T- or B-cell mediated immunogenicity
- Objective response achieved in 8 of 11 patients (73%) treated with ≥ 90 million cells / dose
  - 6 of 11 (55%) patients achieved a CR
  - 2 of 4 patients (50%) previously treated with autologous CD19 CAR T-cell therapy achieved a CR
- Clear evidence that FT516 can drive responses in relapsed / refractory patients
  - Patients had received a median of 3 prior lines and a median of 2 prior lines containing CD20-targeted therapy
  - 8 of 11 patients had aggressive B-cell lymphoma
  - 5 of 11 patients were refractory to their most recent prior therapy

Dose Escalation Ongoing at 900M Cells per Dose

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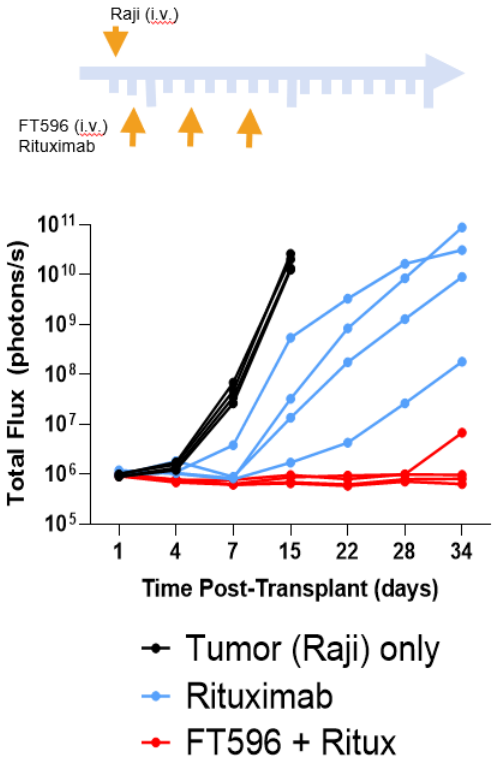
FT596: Multi-antigen Targeted CAR19 NK Cell Product Candidate

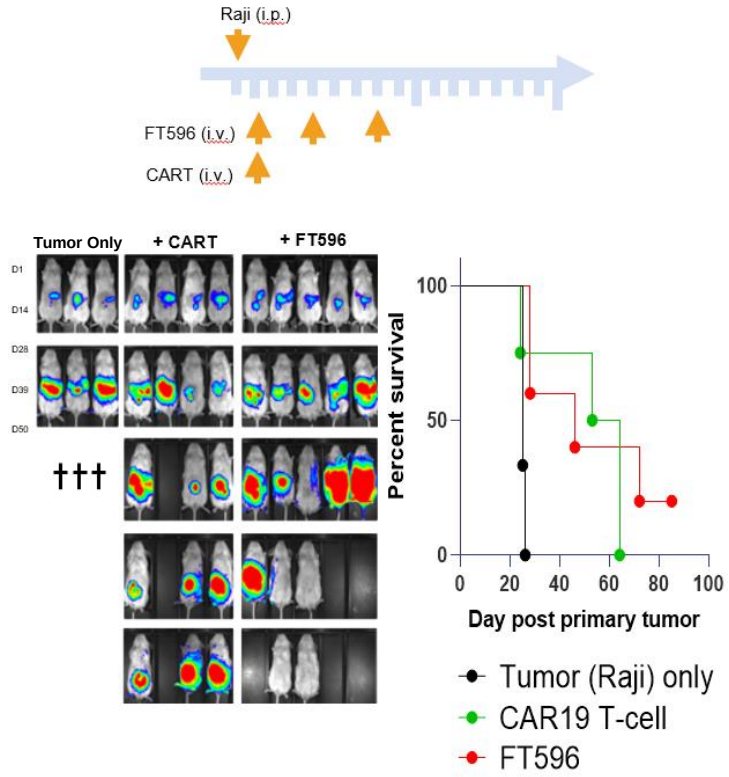
Dual-Antigen Targeting of CD19 and CD20 B-cell Antigens for Best-in-class Potential

hnCD16 Synergizes with CD20-targeted mAb



Lymphoma xenograft NSG immunodeficient mouse model

Multiple Doses of FT596 Compares Favorably to Single-dose Primary CAR19 T Cells

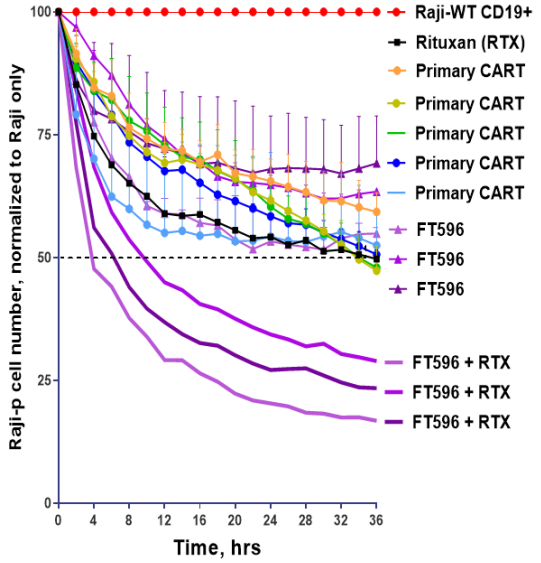


Lymphoma xenograft CD34 engrafted humanized NSG mouse model

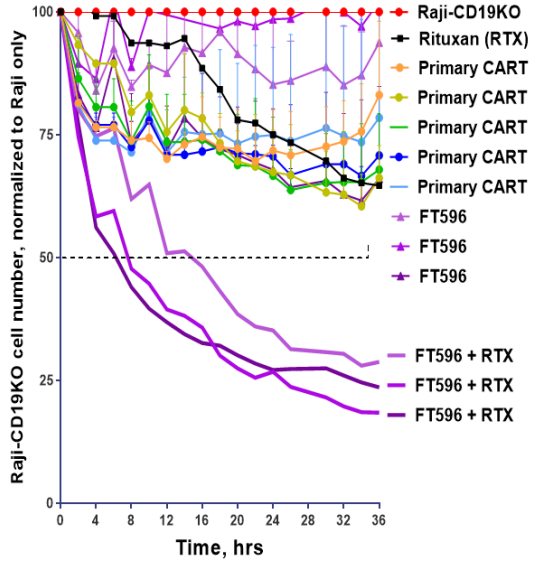
FT596 + CD20-targeted mAb Engage Multiple B-cell Antigens for Enhanced Anti-tumor Activity and Prevention of Antigen Escape

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

Prevention of Antigen Escape
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



Raji lymphoma line CD19- CD20+ CAR19 | rituximab

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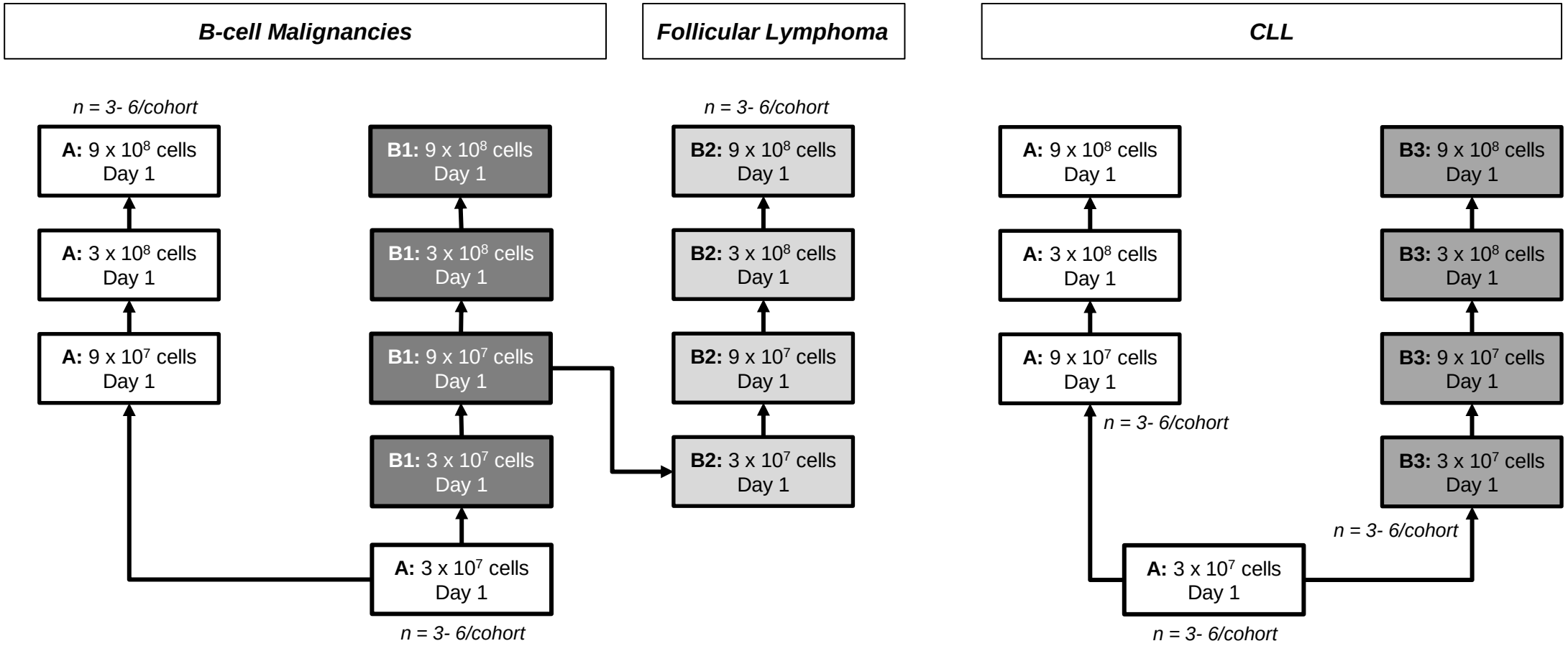
FT596-101: Phase 1 Dose Escalation Schema

Parallel Escalation of Single-dose Mono and mAb Combo in BCL and CLL



Regimen A: FT596 Mono

Regimen B1: FT596 + Rituximab

Regimen B2: FT596 + Obinutuzumab (BCL)

Regimen B3: FT596 + Obinutuzumab (CLL)

FT596-101: Patient 2002 Case Study

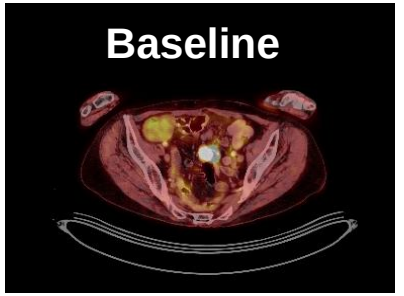
Dose Cohort 1 Monotherapy (Single dose of FT596 at 30M cells)

Patient History

- 76 y/o woman with r/r DLBCL
- Received 7 prior therapies
- Most recently refractory to experimental combo therapy comprised of expanded allogeneic NK cells, IL-2, and rituximab

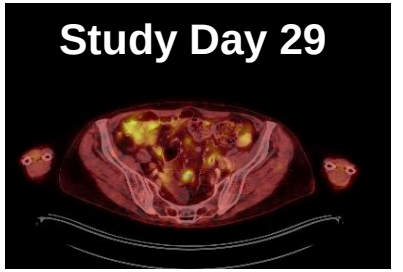
FT596 Safety & Activity

- Cycle 1: Partial response at Study Day 29 following first FT596 single-dose cycle
- Cycle 2: Deepening of response at Study Day 75 following second FT596 single-dose cycle
- DOR = 3.7 months, comparable to that of auto CD19 CAR-T cell therapy among patients who achieve PR as BOR
- No events *of any grade* of CRS, ICANS, or GvHD
- No FT596-related SAEs
- Grade ≥3 AEs considered probably related to Flu/Cy conditioning and possibly related to FT596 included decreases in neutrophil, white blood cell, and lymphocyte counts



SPD
1292 mm^2

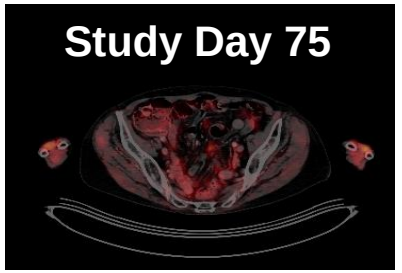
SUV
28



SPD
624 mm^2

SUV
6.6

Partial Response



SPD
420 mm^2

SUV
2.6

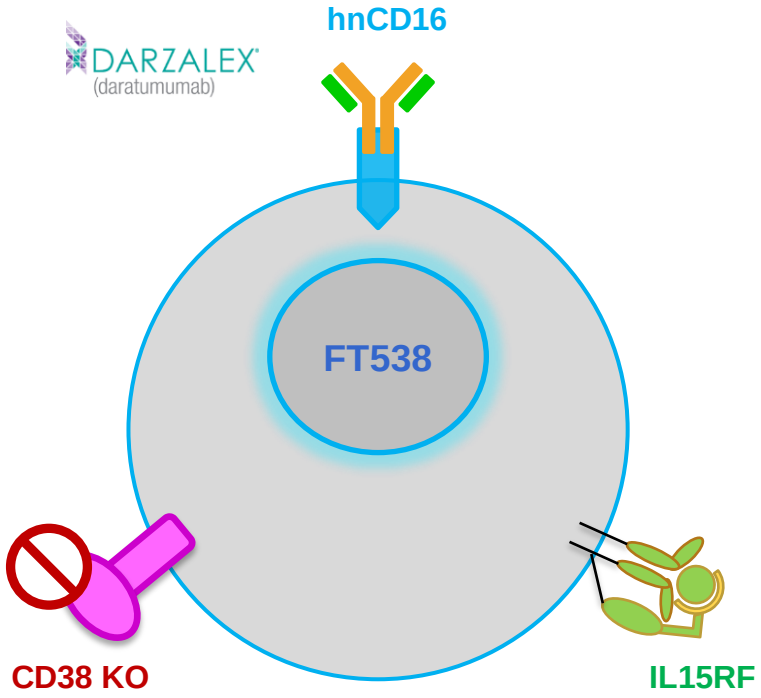
Partial Response

SPD: Sum of the Product of the Diameters; SUV: Standardized Uptake Value

Multiple Myeloma Franchise

FT538 & FT576: First-in-Class NK Cell Cancer Immunotherapies

Optimized Innate & Synthetic Biology

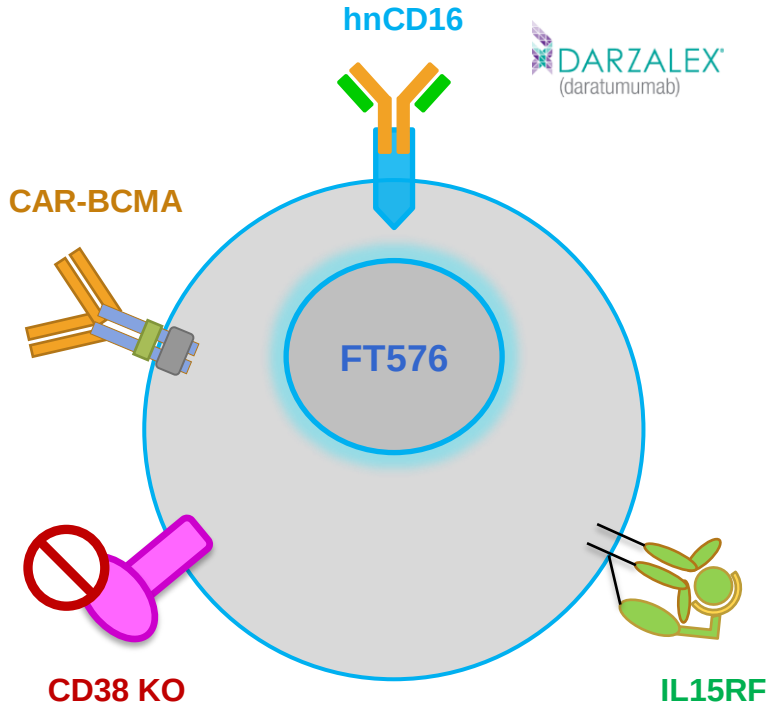


hnCD16: High-affinity 158V, non-cleavable CD16 Fc receptor to augment ADCC

IL-15RF: Interleukin-15 receptor fusion to promote survival, proliferation and trans-activation of NK cells and CD8 T cells

CD38 KO: resistance to anti-CD38 mAb-mediated fratricide; enhanced NK cell metabolic fitness and persistence

CAR-BCMA: Chimeric antigen receptor that targets B-cell Maturation Antigen (optimized for NK cells)



Fc = fragment crystallizable; ADCC - antibody-dependent cellular cytotoxicity; mAb – monoclonal antibody

Multiple Myeloma Disease Franchise

Planned Phase 1 Studies in Relapsed / Refractory MM

Program	FT538 (hnCD16 + IL15RF + CD38KO)	FT576 (hnCD16 + IL15RF + CD38KO + CAR-BCMA)
Treatment	FT538 +/- daratumumab or elotuzumab	FT576 +/- daratumumab
Setting	Relapsed / Refractory MM	Relapsed / Refractory MM
Dose / Schedule	3 once-weekly doses x 1 cycle; second cycle subject to FDA consent • DL1 = 100M • DL2 = 300M • DL3 = 1B • DL4 = 1.5B	Undisclosed
Status	IND allowed; study start-up ongoing	IND allowed; study start-up ongoing

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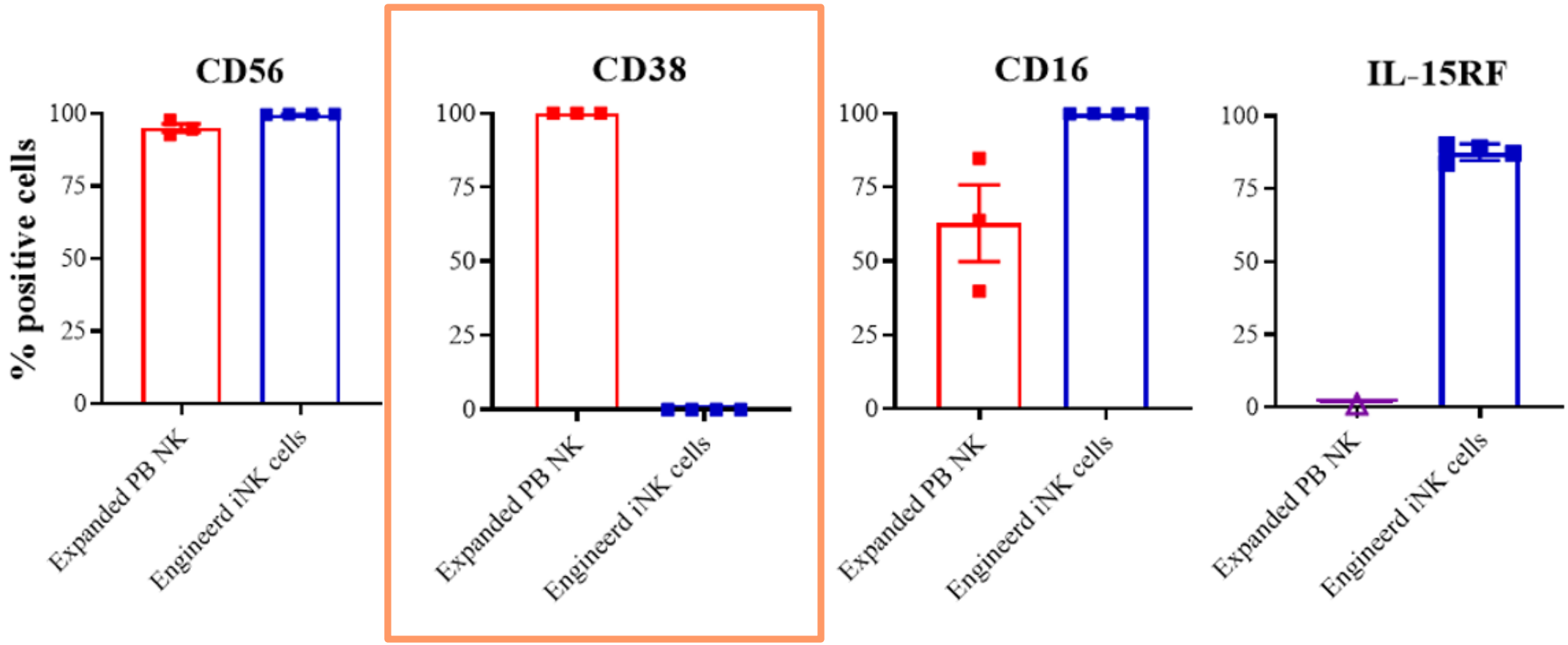
FT538: hnCD16 + IL-15RF + CD38KO NK Cell Product Candidate

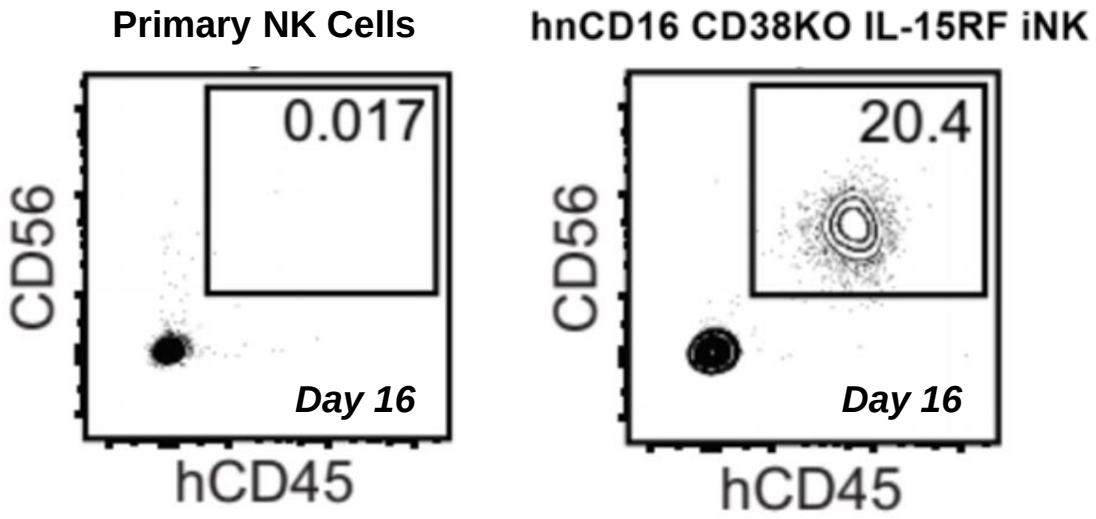
A Uniform, Well-Characterized Cell Product Optimized for Innate Immunity



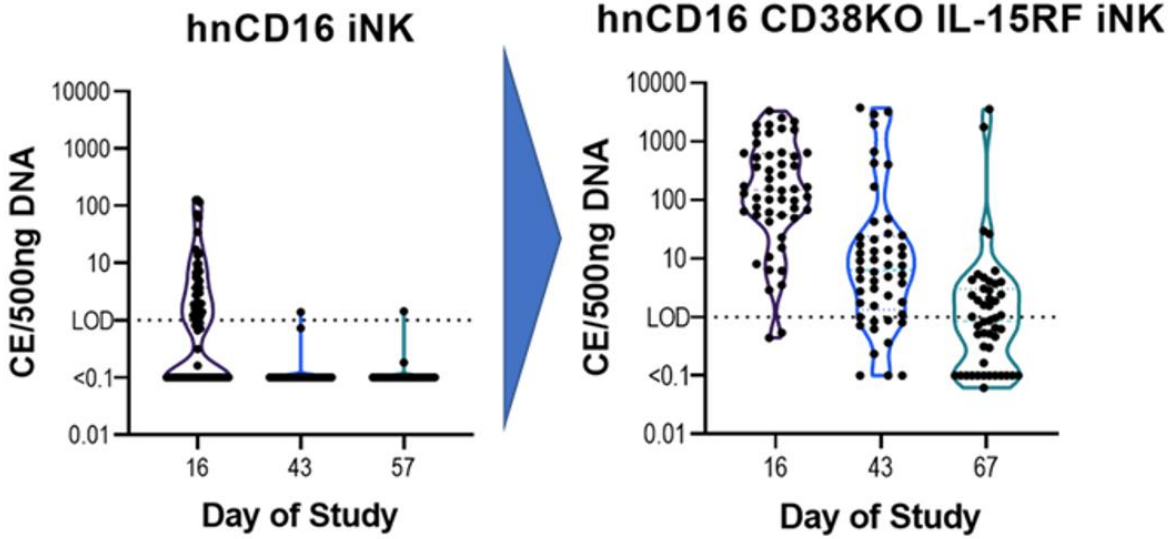
FT538: hnCD16 + IL-15RF + CD38KO NK Cell Product Candidate

Enhanced Persistence Without Cytokine Support

Primary NK vs. FT538 in NSG Mouse



FT516 vs. FT538 in NSG Mouse

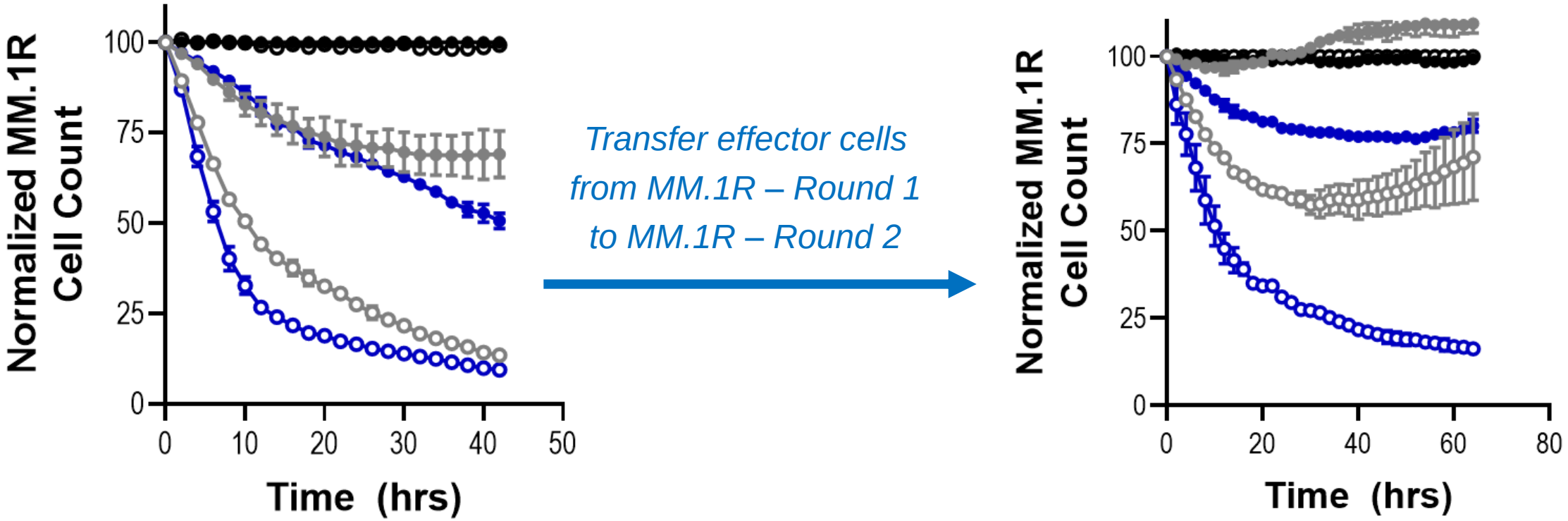


Cichocki et al. manuscript under review

FT538: hnCD16 + IL-15RF + CD38KO NK Cell Product Candidate

Enhanced Cytotoxicity vs. PB NK Cells in a Serial Re-stimulation Cytotoxicity Assay

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



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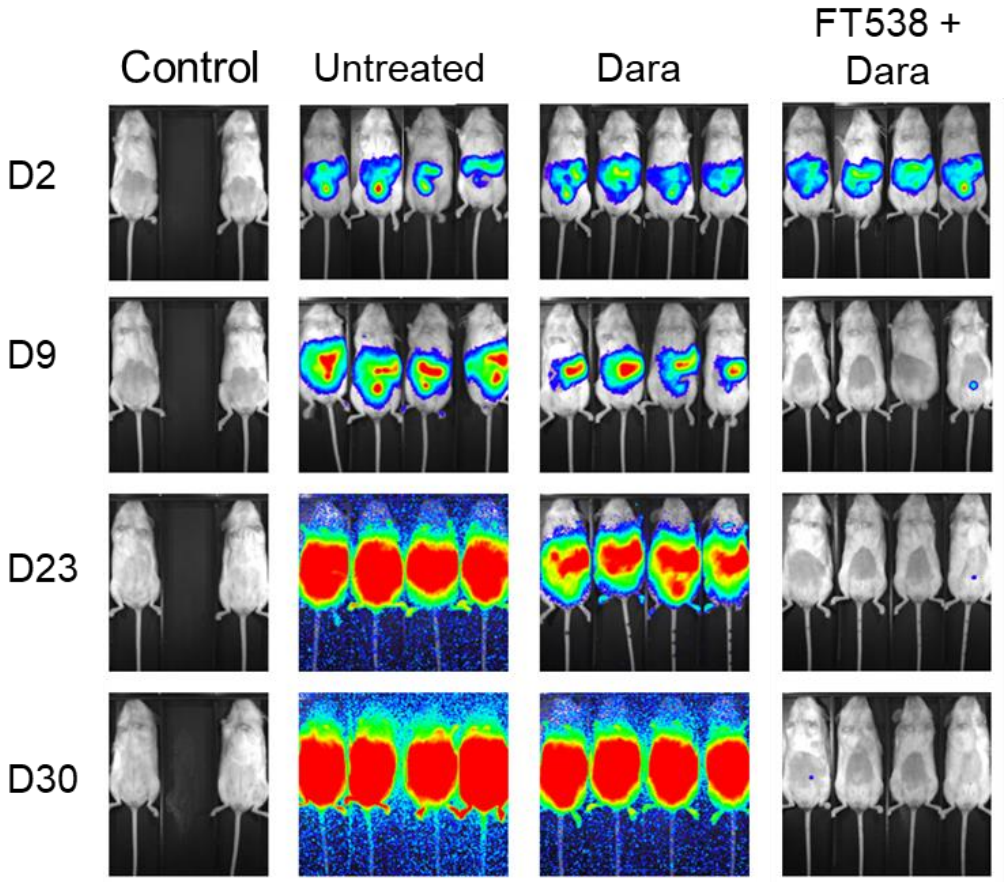
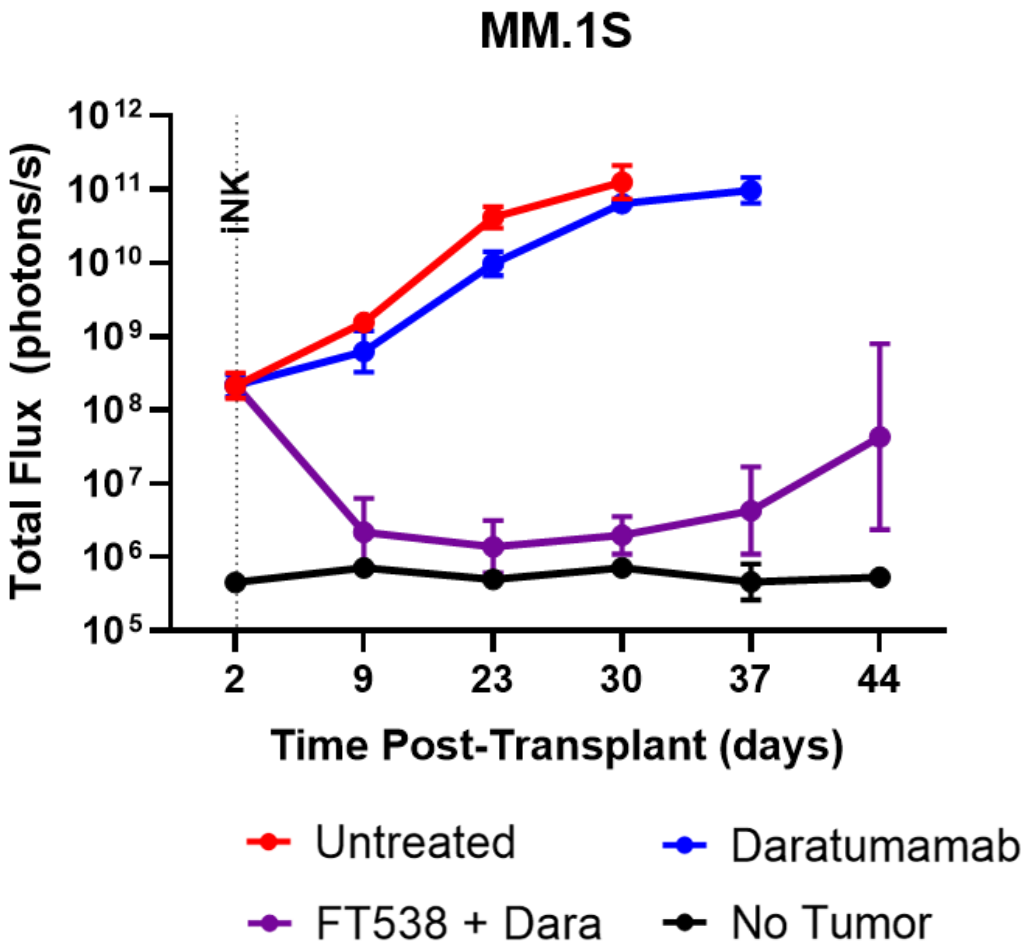
FT538: hnCD16 + IL-15RF + CD38KO NK Cell Product Candidate

Enhanced ADCC in Combination with anti-CD38 mAb In Vivo





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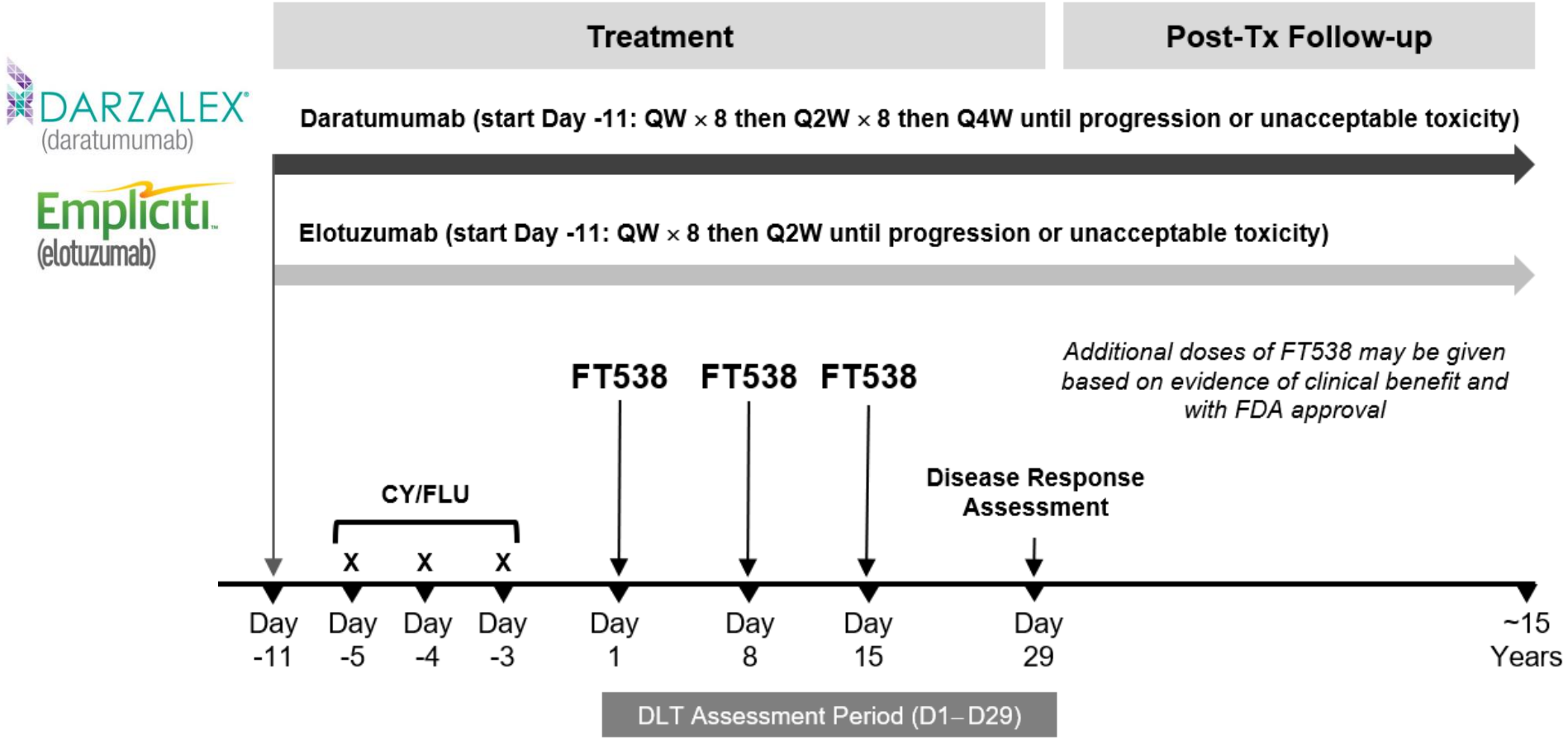
FT538-101: Relapsed / Refractory Multiple Myeloma

Multi-dose Combination with CD38-targeted and SLAMF7-targeted mAb



DL1 = 100M cells / dose

DL2 = 300M cells / dose

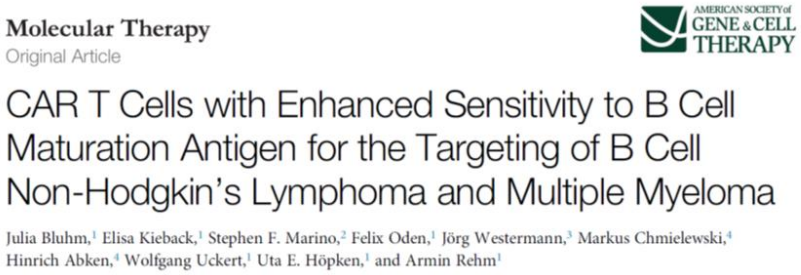
DL3 = 1.0B cells / dose

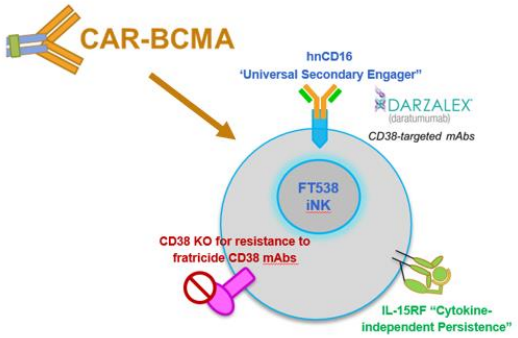
DL4 = 1.5B cells / dose

Fate THERAPEUTICS

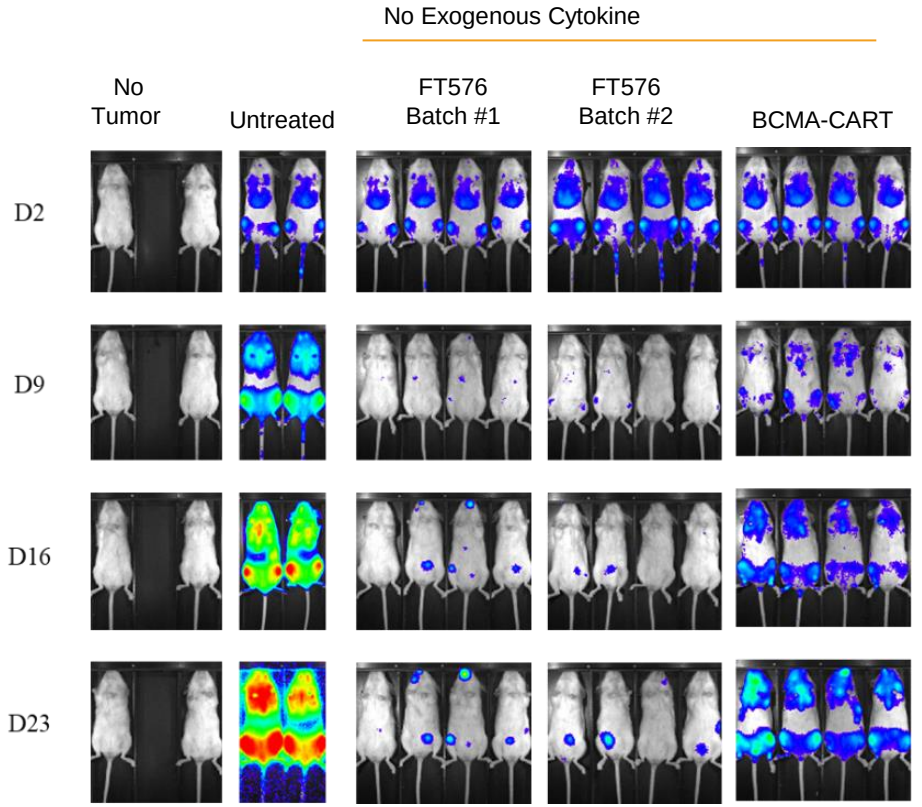
FT576: Multi-antigen Targeted CAR-BCMA NK Cell Product Candidate

BCMA Binding Domain with Differentiated Activation Threshold





- ✓ Novel BCMA binding domain triggers target cell lysis at low levels of BCMA expression (~100 BCMA molecules)
- ✓ FT576 monotherapy demonstrated deeper tumor regression and prolonged tumor control as compared to CAR T cells in *in vivo* preclinical studies
- ✓ The treatment of MM-bearing mice with FT576 + daratumumab exhibited greater anti-tumor activity as compared to each agent alone, demonstrating synergistic activity of BCMA-targeted CAR and CD38-targeted ADCC
- ✓ Potential novel therapeutic option for patients where BCMA is expression is low or where anti-BCMA immunotherapies have failed due to antigen escape



MM.1S-Luc cells

Miller et al. ASH Annual Meeting 2020

AML Franchise

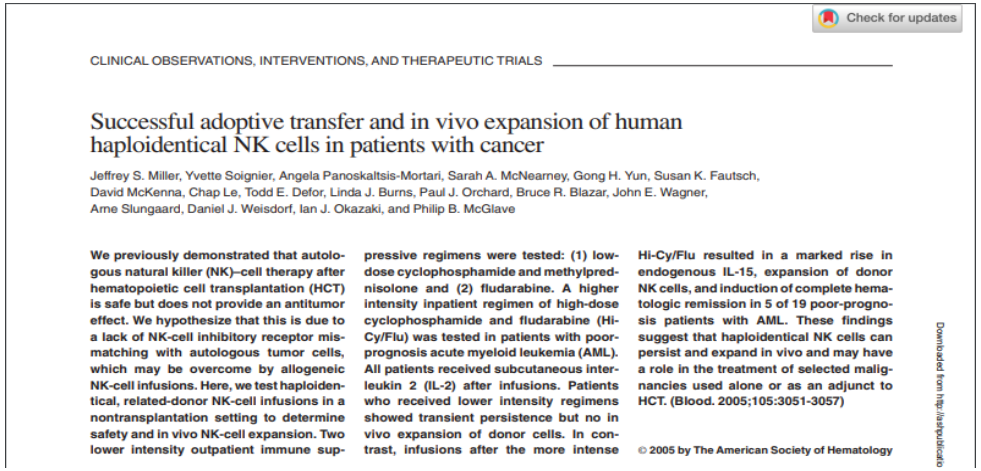
Rationale for NK Cell Therapy in AML

Clinical Precedent with Non-Engineered Allogeneic NK Cells

Jeffrey S. Miller, MD

UNIVERSITY OF MINNESOTA
Driven to Discover℠

Seminal 2005 Manuscript, >1,000 citations



Check for updates

CLINICAL OBSERVATIONS, INTERVENTIONS, AND THERAPEUTIC TRIALS

Successful adoptive transfer and in vivo expansion of human haploidentical NK cells in patients with cancer

Jeffrey S. Miller, Yvette Soignier, Angela Panoskaltsis-Mortari, Sarah A. McNearney, Gong H. Yun, Susan K. Fautsch, David McKenna, Chap Le, Todd E. Defor, Linda J. Burns, Paul J. Orchard, Bruce R. Blazar, John E. Wagner, Arne Slungaard, Daniel J. Weisdorf, Ian J. Okazaki, and Philip B. McGlave

We previously demonstrated that autologous natural killer (NK)–cell therapy after hematopoietic cell transplantation (HCT) is safe but does not provide an antitumor effect. We hypothesize that this is due to a lack of NK-cell inhibitory receptor mismatching with autologous tumor cells, which may be overcome by allogeneic NK-cell infusions. Here, we test haploidentical, related-donor NK-cell infusions in a nontransplantation setting to determine safety and in vivo NK-cell expansion. Two lower intensity outpatient immune suppressive regimens were tested: (1) low-dose cyclophosphamide and methylprednisolone and (2) fludarabine. A higher intensity inpatient regimen of high-dose cyclophosphamide and fludarabine (Hi-Cy/Flu) was tested in patients with poor-prognosis acute myeloid leukemia (AML). All patients received subcutaneous interleukin 2 (IL-2) after infusions. Patients who received lower intensity regimens showed transient persistence but no in vivo expansion of donor cells. In contrast, infusions after the more intense Hi-Cy/Flu resulted in a marked rise in endogenous IL-15, expansion of donor NK cells, and induction of complete hematologic remission in 5 of 19 poor-prognosis patients with AML. These findings suggest that haploidentical NK cells can persist and expand in vivo and may have a role in the treatment of selected malignancies used alone or as an adjunct to HCT. (Blood. 2005;105:3051-3057)

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- 300+ AML/MDS patients treated with allogeneic NK cells[a]
- Numerous clinical studies in relapsed / refractory AML have shown[a]:
  - CR rates = 20-35%
  - No GvHD
  - Minimal CRS / neurotoxicity
- Unmet need in AML remains high
  - ~21,000 newly diagnosed patients in the US alone every year[b]
  - 5-year survival rate ~28%[b]
  - Significant opportunity for more effective, less toxic therapies
    - <50% of elderly patients respond to initial therapy[c]
    - 20-40% of younger patients fail to respond to initial therapy[c]
    - ~50% of patients who attain an initial CR eventually relapse[d]

Fate THERAPEUTICS

[a] *Fate Therapeutics, Internal Literature Review*
[b] *National Cancer Institute Surveillance, Epidemiology, and End Results Program. Cancer Stat Facts: AML. 2015.*
[c] *Mangan J and Luger S. Salvage therapy for relapsed or refractory acute myeloid leukemia. Ther Adv Hematol. 2011; 2(2):73-82.*
[d] *Leopold LH, Willemeze R. The Treatment of Acute Myeloid Leukemia in First Relapse: A Comprehensive Review of the Literature. Leuk Lymphoma. 2002; 43(9); 1715-1727*

AML Disease Franchise

Multiple Ongoing Phase 1 Studies in Relapsed / Refractory AML

Program	FT516 (hnCD16)	FT538 (hnCD16 + IL15RF + CD38 KO)	FT538 (UMN IIT) (hnCD16 + IL15RF + CD38 KO)
Treatment	FT516 Monotherapy IL-2 cytokine support / dose	FT538 Monotherapy No IL-2 cytokine support	FT538 + daratumumab No IL-2 cytokine support
Setting	Relapsed / Refractory AML	Relapsed / Refractory AML	Relapsed / Refractory AML
Dose / Schedule	3 once-weekly doses x 2 cycles • DL1 = 90M • DL2 = 300M • DL3 = 900M	3 once-weekly doses x 1 cycle; second cycle subject to FDA consent • DL1 = 100M • DL2 = 300M • DL3 = 1B • DL4 = 1.5B	3 once-weekly doses x 1 cycle; • DL1 = 100M • DL2 = 300M • DL3 = 1B • DL4 = 1.5B
Status	DL3 enrolling	DL1 enrolling	IND allowed; study start-up ongoing

Fate THERAPEUTICS

FT516-101: Monotherapy in Relapsed / Refractory AML

Patient Characteristics Reflect Extremely Poor Prognosis

Subject #	Age	# of Prior Lines	Risk Profile		Last Line of Therapy	
			2017 ELN Risk Category	PIF	Regimen	Response
1001	41	3	Intermediate	Yes	Ven + Dec	Refractory
1003	64	3	Adverse	No	Ivosidenib	Refractory
1005	58	4	Adverse	Yes	7+3	Refractory
1006	68	1	Adverse	No	Ven + Dec	Relapse
1007	85	1	Adverse	Yes	Ven + Dec	Refractory
1008	33	6	Adverse	Yes	Gilteritinib	Refractory
1011	60	3	Adverse	Yes	CLAG-M	Refractory
1012	56	5	Adverse	No	Ven + Aza	Refractory
1015	59	3	Adverse	Yes	Ven + Dec	Refractory

All data based on database entry as of April 16, 2021. Data subject to source document verification.

8 of 9 with Adverse Risk 6 of 9 with Primary Induction Failure

8 of 9 with Refractory Disease

Aza = Azacitidine; **CLAG-M** = Cladribine, Ara-C, Filgrastim, Mitoxantrone; **Dec** = Decitabine; **PIF** = Primary Induction Failure; **Ven** = Venetoclax; **7+3** = Ara-C + Daunorubicin

FT516-101: Monotherapy in Relapsed / Refractory AML

Safety, Tolerability & Immunogenicity

Subject #	# Cells / Dose	# of Doses	FT516-Related Safety					SAEs	Immunogenicity	
			DLT	Any Grade CRS	Any Grade ICANS	Any Grade GvHD	Grade ≥ 3 AEs		T-cell	B-cell
1001	90M	6	No	No	No	No	None	None	No	No
1003	90M	6	No	No	No	No	FN (Gr3)	None	No	No
1005	90M	4	No	No	No	No	FN (Gr3)	None	No	No
1006	300M	6	No	No	No	No	None	None	No	No
1007	300M	6	No	No	No	No	None	None	No	No
1008	300M	3	No	No	No	No	None	None	No	No
1011	300M	3	No	No	No	No	None	None	No	No
1012	300M	6	No	No	No	No	None	None	No	No
1015	300M	3	No	No	No	No	FN (Gr3)	None	No	No

All data based on database entry as of April 16, 2021. Data subject to source document verification.

No events of any grade of CRS, ICANS, or GvHD No evidence of T- or B-cell mediated rejection

FT516 was well-tolerated; no discontinuations due to safety events

AE = Adverse Events; **CRS** = Cytokine Release Syndrome; **DLT** = Dose Limiting Toxicity; **FN** = Febrile Neutropenia; **GvHD** = Graft vs. Host Disease; **ICANS** = Immune Cell-Associated Neurotoxicity Syndrome; **SAE** = Serious Adverse Event

FT516-101: Monotherapy in Relapsed / Refractory AML

Best Overall Response

Subject #	# Cells / Dose	# of Doses	Baseline			Best Overall Response (BOR)		
			% Bone Marrow Blasts	Neutrophils (10^3/µl)	Platelets (10^3/µl)	% Bone Marrow Blasts	% Change in Bone Marrow Blasts	BOR (2017 ELN)
1001	90M	6	6%	0.8	12	0%	-100%	CRi
1003	90M	6	39%	0.1	22	31%	-21%	SD
1005	90M	4	40%	0.2	42	45%	+13%	SD
1006	300M	6	26%	0.4	30	0%	-100%	CRi
1007	300M	6	12%	1.9	59	0%	-100%	CRi
1008	300M	3	95%	0.2	24	98%	+3%	SD
1011	300M	3	91%	0.4	5	85%	-7%	PD
1012	300M	6	20%	0.2	42	0%	-100%	MLFS
1015	300M	3	44%	0.1	18	60%	+36%	PD

All data based on database entry as of April 16, 2021. Data subject to source document verification.

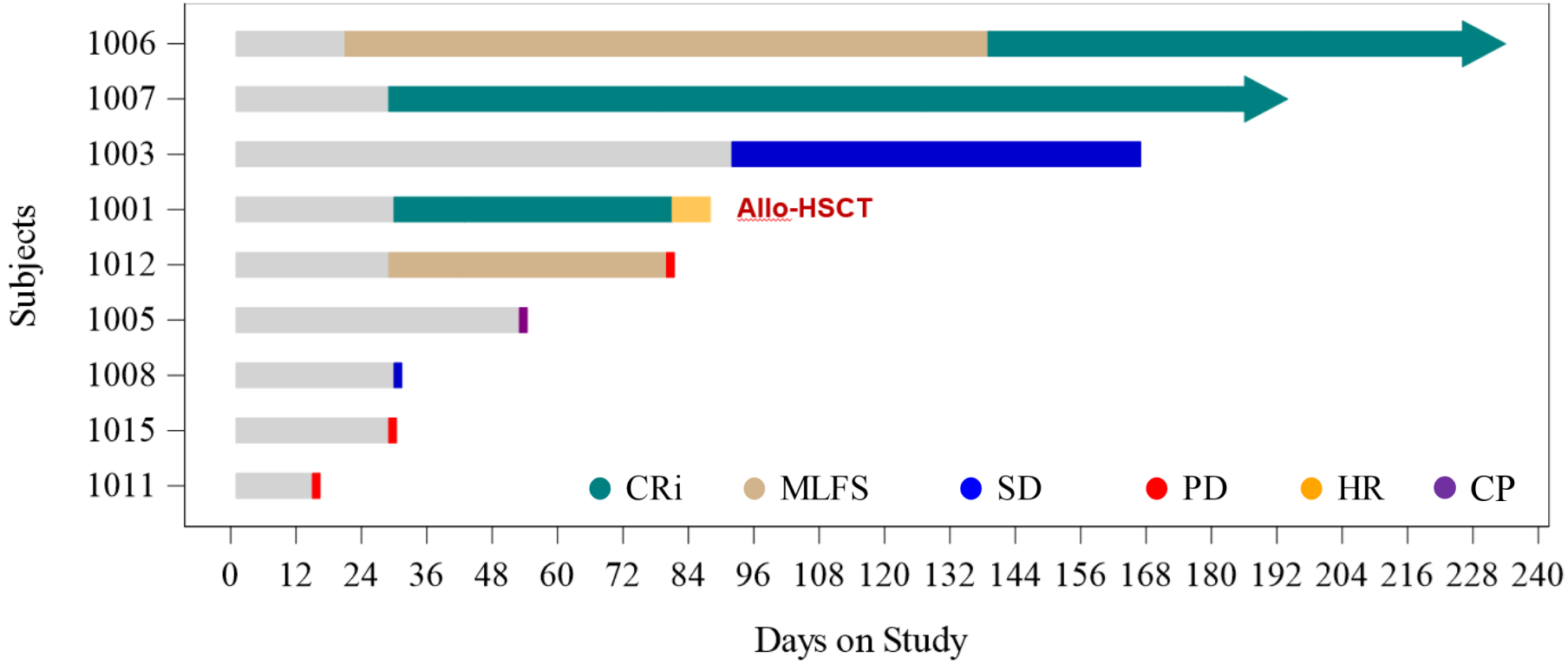
4 of 9 Patients Achieved Complete Leukemic Blast Clearance in Bone Marrow and Objective Response based on 2017 ELN Response Criteria

CRi = Complete Remission (CR) other than, with respect to hematologic recovery, CRi requires recovery of neutrophils to ≥1000/µL or platelets to ≥100,000/µL;
MLFS = Morphologic Leukemia Free State; **PD** = Progressive Disease; **SD** = Stable Disease

Fate THERAPEUTICS

FT516-101: Monotherapy in Relapsed / Refractory AML

Duration of Anti-leukemic Activity



Two Patients with CRi Remained in Remission with Ongoing DOR >6 months; No additional therapeutic intervention

Evidence of Evolving Response from MLFS to CRi

One Patient with CRi Proceeded to allo-HSCT

CRi = Complete Remission (CR) other than, with respect to hematologic recovery, CRi requires recovery of neutrophils to ≥1000/μL or platelets to ≥100,000/μL;
CP = Clinical Progression (evidence of progression not fulfilling 2017 ELN PD definition per investigator assessment); **HR** = Hematologic Relapse after CR/CRi;
MLFS = Morphologic Leukemia Free State; **SD** = Stable Disease; **PD** = Progressive Disease

FT538-101: Monotherapy in Relapsed / Refractory AML

Patient Characteristics

Subject #	Age	Prior Therapy	# of Prior Lines	Risk Profile		Last Line of Therapy	
				2017 ELN Risk Category	PIF	Regimen	Response
1001	72	Ven + Aza 7+3 Ven + Dec	3	Unknown	No	Ven + Dec	Refractory
1002	78	Ven + Aza Gilteritinib GTB-3550	3	Adverse	No	GTB-3550	Refractory
1003	79	7+3 Ven + Aza GTB-3550 Glasdegib + LDAC	4	Intermediate	No	Glasdegib + LDAC	Refractory

All data based on database entry as of April 16, 2021. Data subject to source document verification.

3 Heavily Pre-treated Patients with Refractory Disease

2 Patients were Refractory to TriKE (CD33-targeted Trispecific NK cell engager)

Aza = Azacitidine; **Dec** = Decitabine; **LDAC** = Low-dose Ara-C; **PIF** = Primary Induction Failure; **Ven** = Venetoclax; **7+3** = Ara-C + Daunorubicin; **GTB-3550** = investigational CD33-targeted Trispecific NK cell engager

FT538-101: Monotherapy in Relapsed / Refractory AML

100M Cells / Dose: Safety, Tolerability, Immunogenicity & Response

Subject #	% BM Blasts Baseline	FT538 Doses	FT538-Related Safety					Immunogenicity		Best Overall Response (2017 ELN)
			DLT	Any Grade CRS	Any Grade ICANS	Any Grade GvHD	Grade ≥3 AE	T-cell	B-cell	
1001	70%	2	NE	No	No	No	None	No	No	**SD**
1002	25%	6*	No	No	No	No	None	No	No	**SD**
1003	30%	6*#	No	No	No	No	None	No	No	**CRi**

All data based on database entry as of April 16, 2021. Data subject to source document verification.

** FDA approved administration of Cycle 2*

 # Cycle 2 ongoing as of data cutoff

1 of 3 Patients Achieved Objective Response based on 2017 ELN Response Criteria

No events of any grade of CRS, ICANS, or GvHD and no SAEs No evidence of T- or B-cell mediated rejection

FT538 was well-tolerated; no discontinuations due to safety events

AE = Adverse Events; **CRS** = Cytokine Release Syndrome; **DLT** = Dose Limiting Toxicity; **GvHD** = Graft vs. Host Disease; **ICANS** = Immune Cell-Associated Neurotoxicity Syndrome; **NE** = Not Evaluable; **SAE** = Serious Adverse Event

CRi = Complete Remission (CR) other than, with respect to hematologic recovery, CRi requires recovery of neutrophils to ≥1000/μL or platelets to ≥100,000/μL; **SD** = Stable Disease

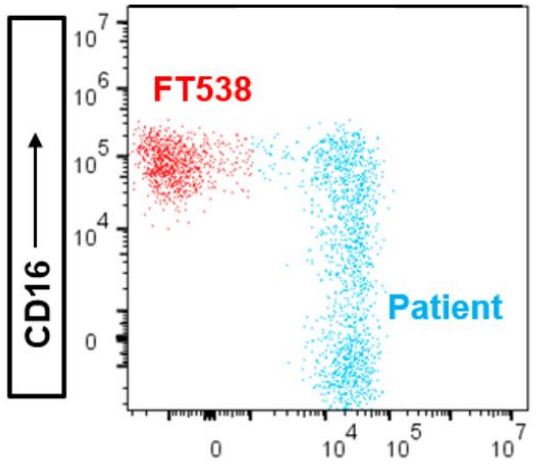
Fate THERAPEUTICS

FT538-101: Monotherapy in Relapsed / Refractory AML

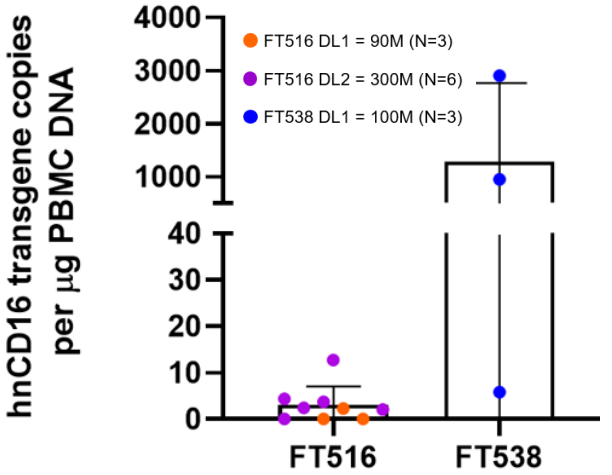
Subject 1003 (100M cells)

- **Patient Characteristics**
  - 79 y.o. male diagnosed with *de novo* AML in 2017
  - Multiple lines of prior therapy
    - Idarubicin + Ara-C (CR; DOR = 20 months)
    - Venetoclax + Azacitidine (PR; DOR = 1 month)
    - GTB-3550 (investigational CD33-IL15-CD16 NK cell engager) (**refractory**)
    - Glasdegib + Low dose Ara-C (**refractory**)
- **Baseline Disease Status**
  - 2017 ELN risk category = **Intermediate**
  - Bone Marrow: 30% blasts by morphology; 20-30% cellularity
  - Peripheral Blood: No blasts; ANC = 0.1 x 10^3/µL (neutropenic); Platelets = 35 x 10^3/µL (thrombocytopenic)
- **Clinical Course**
  - Received 3 doses of FT538
  - No events of any grade of CRS, ICANS, or GVHD
  - No FT538-related Grade ≥3 AEs
  - 2017 ELN response criteria (BOR) = **CRi**
    - Complete neutrophil recovery *exceeding* baseline (1.6x10^3/µL from 0.1x10^3/µL)
    - Second treatment cycle approved by FDA; follow-up ongoing

Day 7 CD16 Expression



Day 8 Peripheral Blood PK



FT516 / FT538: Monotherapy in Relapsed / Refractory AML

Initial Clinical Observations

- Phase 1 studies have enrolled an unfavorable patient population (n=12)
  - Median of 3 prior lines, with 11 patients refractory to their last prior therapy
  - 9 patients with adverse risk profile (with 1 patient unknown) based on 2017 ELN risk category
  - 11 patients had significant hematopoietic impairment at baseline, with both low neutrophil and platelet counts
- FT516 and FT538 as monotherapy exhibited favorable safety, and multi-dose treatment schedule was well-tolerated
  - No observed DLTs and no events of any grade of CRS, ICANS, or GVHD
  - Successfully administered in the outpatient setting
- 5 of 12 patients (42%) achieved an objective response with complete leukemic blast clearance in the bone marrow
  - FT516 (n=9): 3 CRi, 1 MLFS; FT538 (n=3): 1 CRi
  - Durable remissions >6 months achieved in 2 FT516 patients without any additional therapeutic intervention
- Additional engineered modalities of FT538 may confer further therapeutic advantages
  - CRi achieved in multiply-refractory patient, including to CD33-targeted NK cell engager, in first dose escalation cohort
  - FT538 detected in the peripheral blood at Day 8 post-infusion without administration of IL-2 cytokine support

Fate THERAPEUTICS

Relapsed / Refractory Acute Myeloid Leukemia

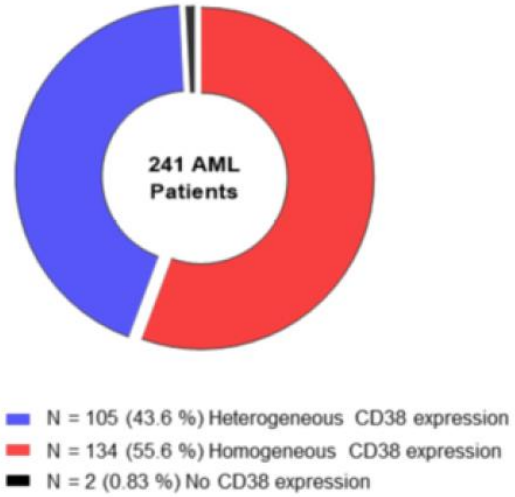
FT538 + daratumumab for Targeting of CD38 on Leukemic Blasts

EUROPEAN HEMATOLOGY ASSOCIATION

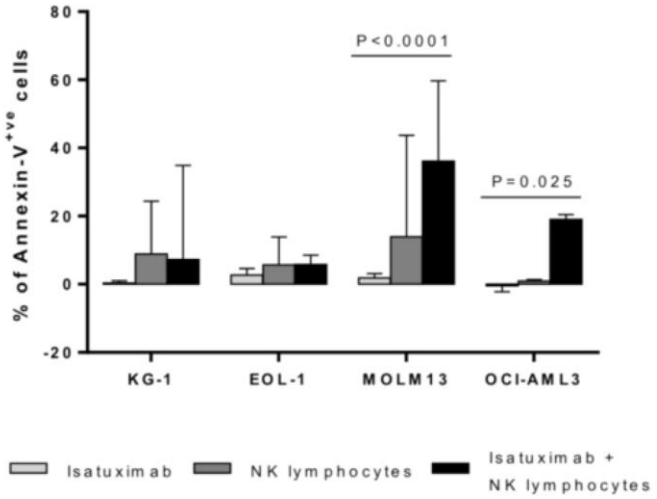
The mode of action of the anti-CD38 monoclonal antibody isatuximab in elderly acute myeloid leukemia

Aintzane Zabaleta 1*, Tomas Jelinek 1,2,3*, Catia Simoes 1, Laura Bianco 1, Daniel Alameda 1, Daniel Ajona 1,5,6, Cristina Perez 1, Diego Alignani 1, Sonia Garate 1, Maria-Jose Larrayoz 1, Maria-Jose Calasanz 1, Lucie Cerna 2, Michal Simicek 2, Roman Hajek 2, Felipe Prosper 1,7, David Martínez-Cuadrón 4, Juan Miguel Bergua 9, Susana Vives 10, Lorenzo Algarra 11, Mar Tormo 12, Pilar Martinez 13, Josefina Serrano 14, Pilar Herrera 15, Fernando Ramos 16, Olga Salamero 17, Esperanza Lavilla 18, Miguel Ángel Sanz 4, Pau Montesinos 4, Jesus F. San Miguel 1,8, Bruno Paiva 1,8
On behalf of the PETHEMA group.

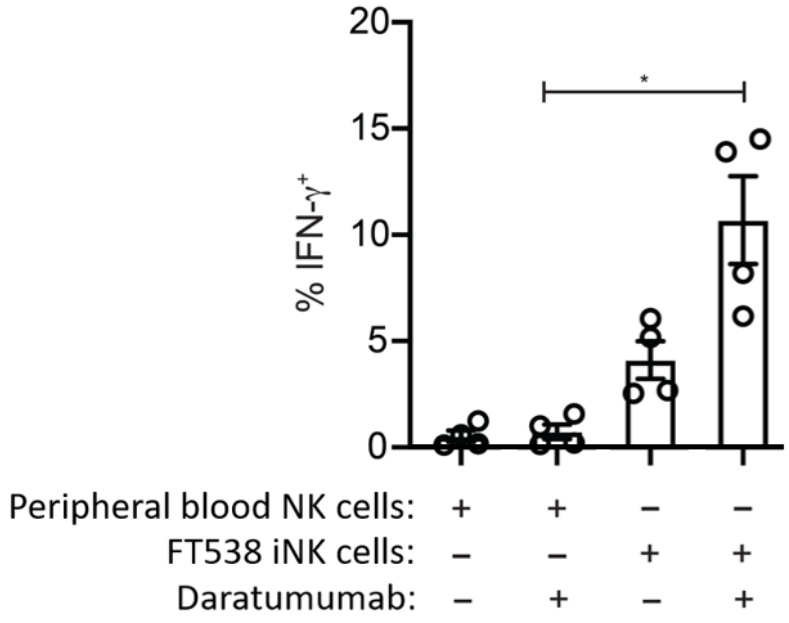
CIMA LAB diagnostics
Universidad de Navarra



CD38 expression from bone marrow samples was found in 239 of 241 newly-diagnosed AML patients



NK cells in combination with CD38-targeted mAb significantly enhances anti-leukemic activity against AML cell lines



FT538 uniquely elicits an anti-tumor response against patient-derived AML samples that is further enhanced when combined with daratumumab

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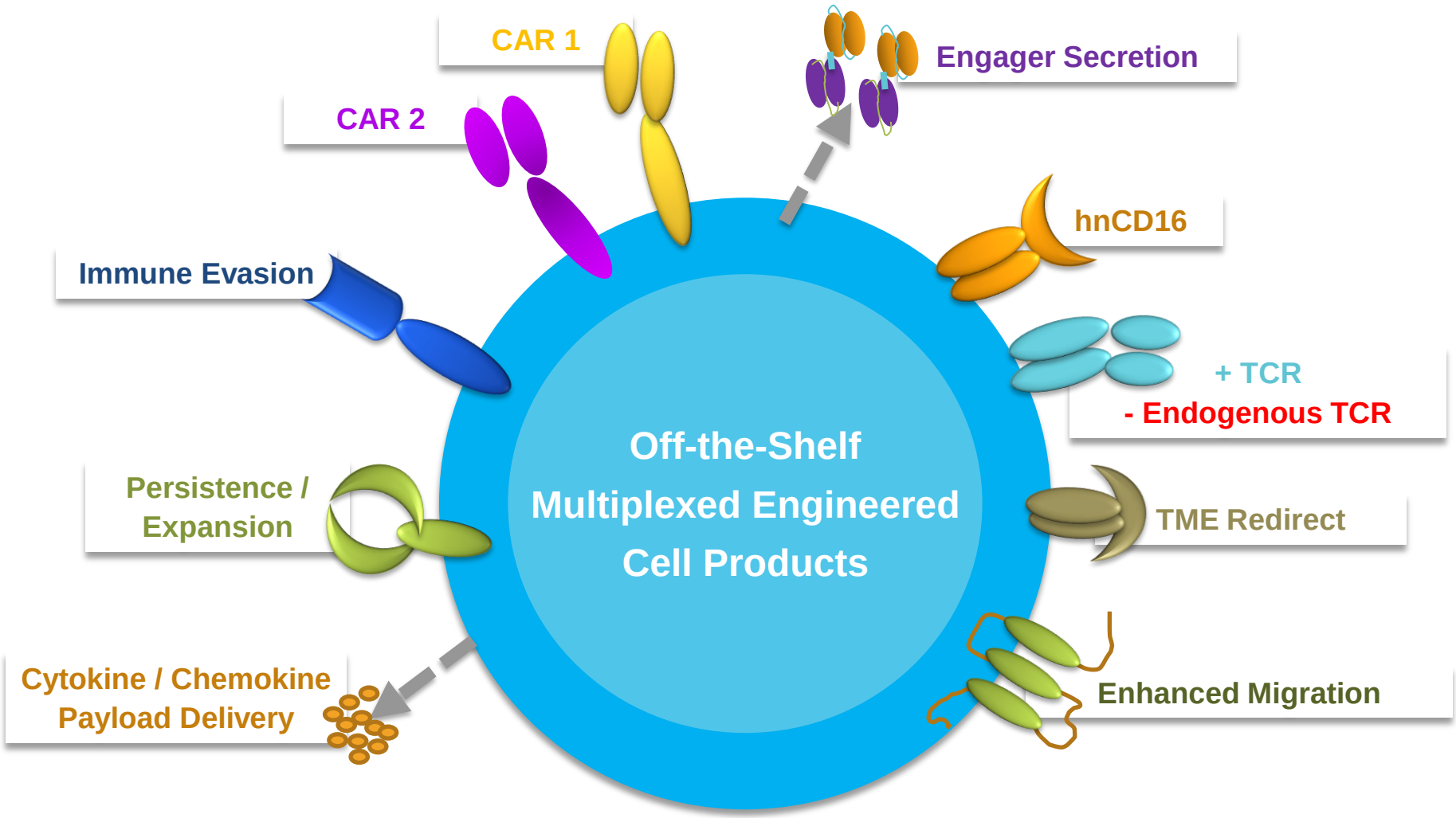
UMN IIT of FT538 + CD38-targeted daratumumab set to initiate in r/r AML

Solid Tumor Franchise

Developing Multi-functional Off-the-Shelf Cell Products for Solid Tumors

One Therapeutic Modality Incorporating Multiple Mechanisms in Fight Against Cancer



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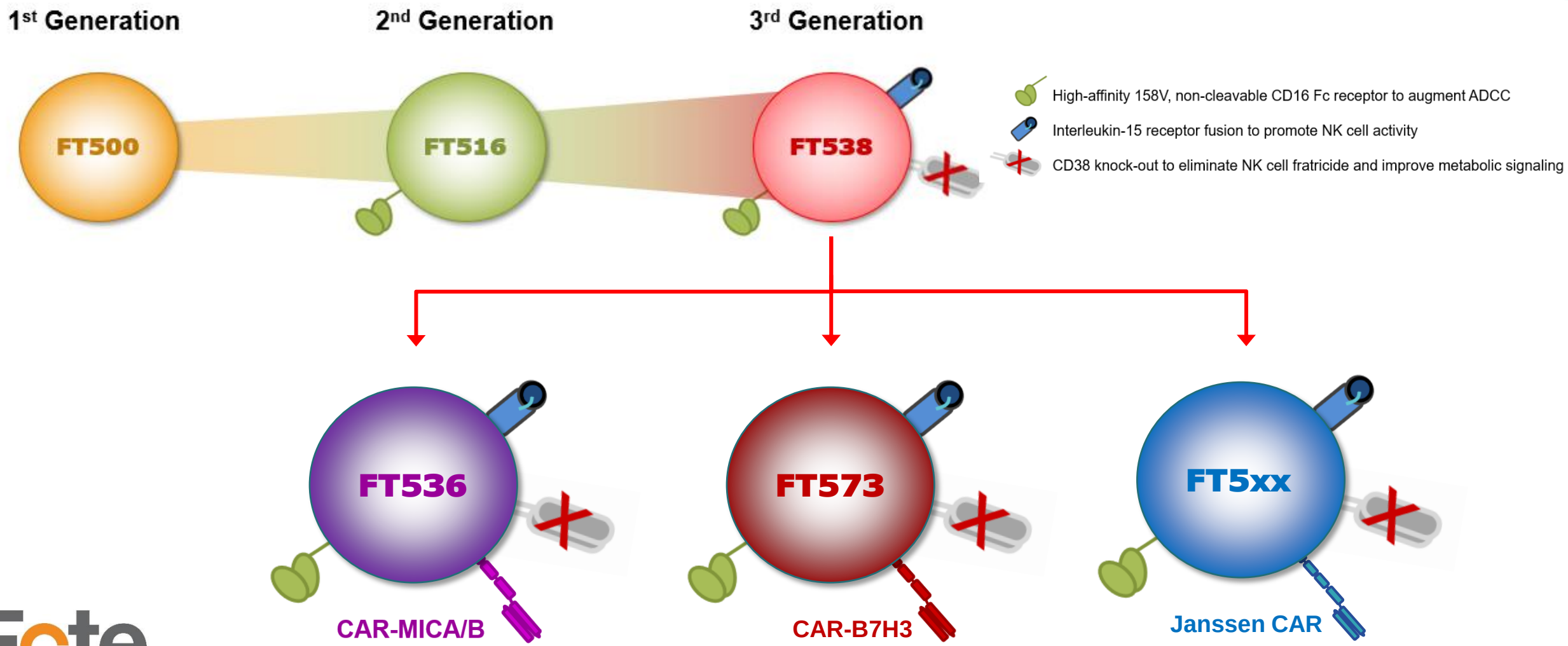
Off-the-Shelf, iPSC-derived NK Cell Cancer Immunotherapy Franchise

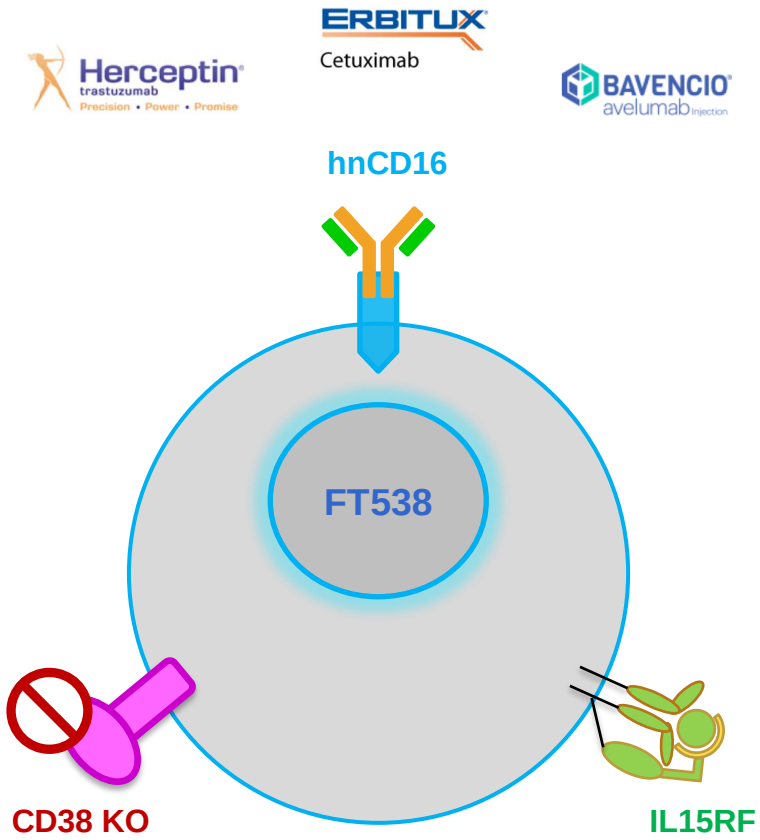
Evolution Toward Multiplexed-Engineered Cell-based Cancer Immunotherapies



Fate THERAPEUTICS

FT538-102: First-Ever CRISPR-edited iPSC-derived Cell Therapy

Incorporates Three Functional Components to Enhance Innate Immunity



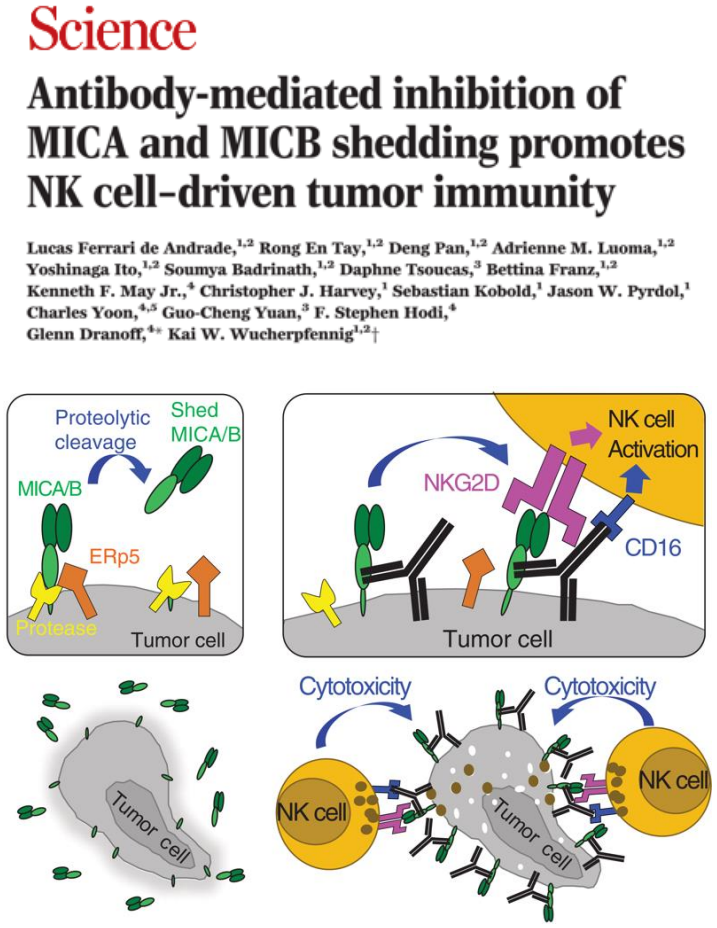
- Innate immunity is severely compromised in patients with cancer
  - Depleted / dysfunctional NK cell compartment
  - Inferior ADCC capacity due to naturally-occurring, low-affinity CD16
  - Diminished ADCC activity through down-regulation or shedding of CD16
  - Exhaustion of NK cells within immunosuppressive tumor micro-environment
- FT538 is engineered to synergize with antibodies and effectively kill solid tumors
  - ✓ High-affinity, non-cleavable CD16 Fc receptor to synergize with mAbs and enhance ADCC
  - ✓ IL-15 receptor fusion to promote survival, proliferation and trans-activation of NK and T cells
  - ✓ CD38 knock-out to improve potency and metabolic fitness of NK cells
- FT538 provides proof-of-concept for multiplexed-engineered, iPSC-derived NK cells and serves as foundation for building CAR-targeted product candidates (FT536, FT573)

Phase 1 Dose Escalation in Combination with EGFR-targeted cetuximab, HER2-targeted trastuzumab, and PDL1-targeted avelumab to initiate in 2H21

Fate THERAPEUTICS

FT536: Multi-targeted CAR-MICA/B NK Cell Product Candidate

Novel Pan-tumor Targeting Strategy for Solid Tumors



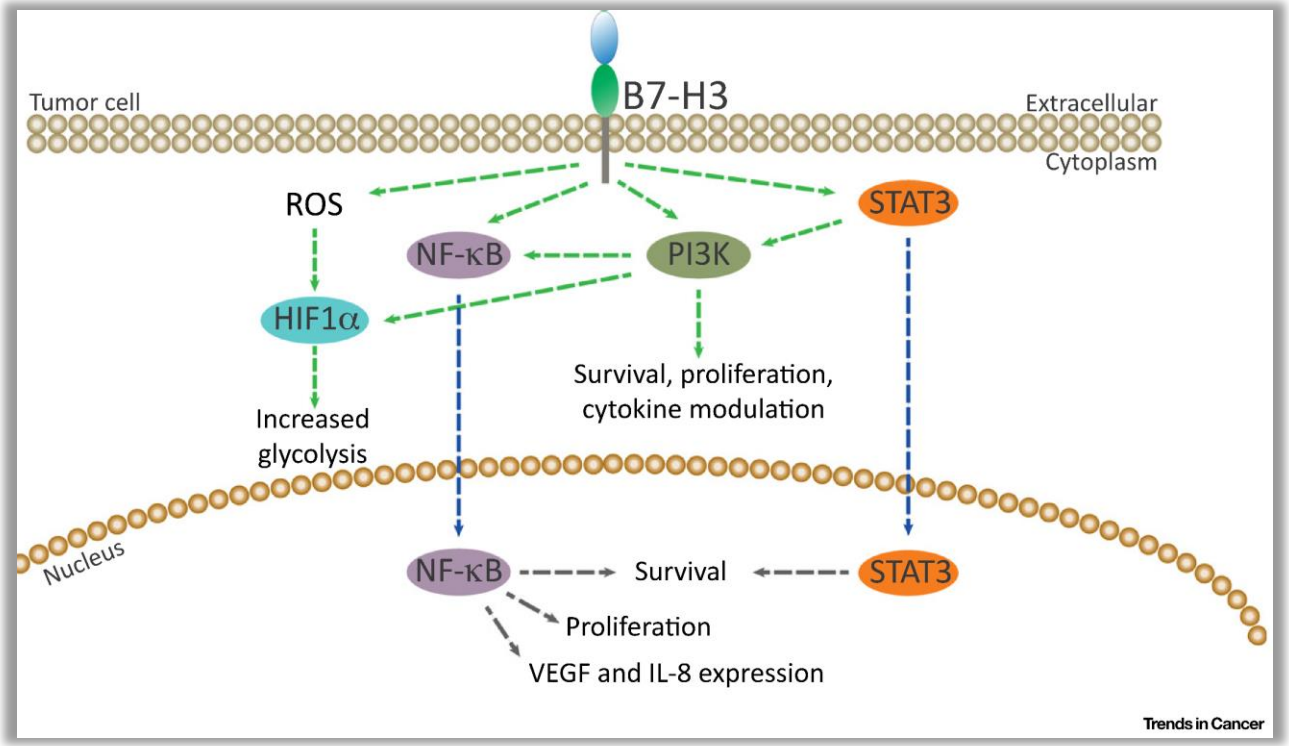
- ✓ MICA/B are induced by cellular stress and transformation, and their expression has been reported for many cancer types
- ✓ NKG2D, an activating receptor expressed on NK and T cells, targets the membrane-distal α1 and α2 domains of MICA/B, activating a potent cytotoxic response
- ✓ Advanced cancer cells frequently evade immune cell recognition by proteolytic shedding of the α1 and α2 domains of MICA/B, which can significantly reduce NKG2D function and the cytolytic activity
- ✓ Therapeutic antibodies targeting the membrane-proximal α3 domain inhibited MICA/B shedding, resulting in a substantial increase in the cell surface density of MICA/B and restoration of immune cell-mediated tumor immunity
- ✓ We have developed a novel CAR targeting the conserved α3 domain of MICA/B (CAR-MICA/B)
- ✓ By uniquely targeting the α3 domain, FT536 prevents shedding and directly targets one of the most highly-expressed stress ligands on a broad range of tumors

Fate THERAPEUTICS

Dana-Farber Cancer Institute

FT573: Multi-targeted CAR-B7H3 NK Cell Product Candidate

Novel Pan-tumor Targeting Strategy for Oncogenic Cells and Prevention of Metastasis



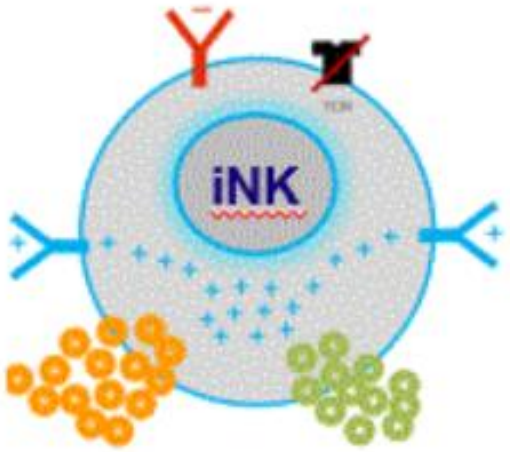
https://doi.org/10.1016/j.trecan.2018.03.010

- ✓ B7H3 (CD276) belongs to the B7 superfamily of immune checkpoint molecules and is overexpressed in a wide variety of cancers, often associated with poor prognosis
- ✓ B7H3 induces the Warburg effect and plays a key role in promoting metastasis and cancer stem cell-like properties
- ✓ B7H3 also promotes resistance to cancer drugs and angiogenesis
- ✓ B7H3-specific monoclonal antibodies and antibody-drug conjugates have shown anti-tumor activity against B7H3+ tumor cells in xenograft mouse models
- ✓ We are developing a novel CAR to target a defined region of B7H3 as pan-tumor targeting approach

Fate THERAPEUTICS

FT500-101: First-ever U.S. Clinical Study of iPSC-derived Cell Product

Phase 1 Dose Expansion Ongoing in Advanced Solid Tumors



Program	FT500 Dose Expansion (non-engineered iPSC-derived NK cell)
Rationale	Assess direct tumor lysis and T-cell recruitment / activation to re-sensitize ICI-resistant tumors with FT500
Treatment	FT500 + ICI + IL2
Setting	Relapsed / Refractory NSCLC and cHL who failed prior ICI
Dose / Schedule	Up to 6 doses (300M cells / dose) over 45 days following 1x Cy/Flu conditioning
Status	Dose expansion ongoing

Phase 1 Dose-Escalation Results (n=15)

- No dose-limiting toxicities; no FT516-related SAEs or FT516-related Grade ≥ 3 AEs
- No events of any grade of CRS, ICANs or GVHD
- 81 total doses of FT500 were administered in the outpatient setting; no discontinuations other than disease progression
- Among 15 heavily pre-treated patients, 10 were refractory to prior therapy and 11 had a best overall response of SD

ICI = Immune Checkpoint Inhibitors (e.g. pembrolizumab, nivolumab, atezolizumab)
CRS = cytokine release syndrome; ICANs = immune effector cell-associated neurotoxicity syndrome; GVHD = graft-versus-host disease

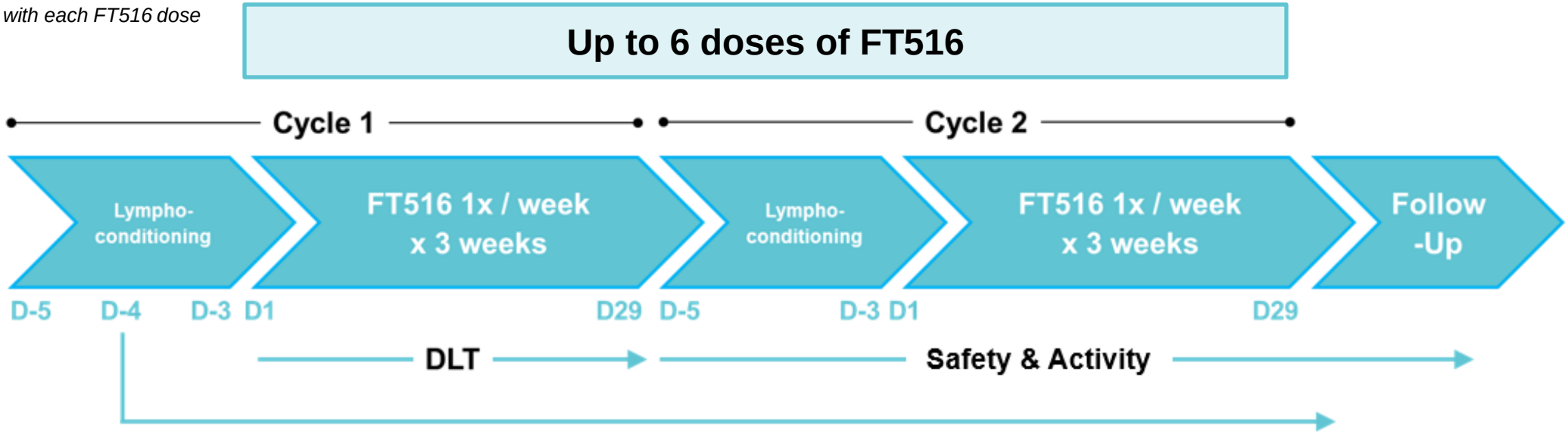
FT516-102: Combination with PDL1-targeted mAb for Advanced Solid Tumors

Phase 1 Dose Escalation Ongoing

Cyclophosphamide: 500 mg/m2 IV x 3 days

Fludarabine: 30 mg/m2 IV x 3 days

IL-2: 6M units sc with each FT516 dose



Avelumab: 800 mg every 2 weeks IV until disease progression or unacceptable toxicity

BAVENCIO® avelumab Injection

Avelumab Arm

- Advanced solid tumors for which anti-PD-L1 mAb is approved
- Dose Escalation: 90M, 300M, 900M cells per dose + avelumab
- Dose Expansion: up to 30 patients in two 15-patient expansion cohorts

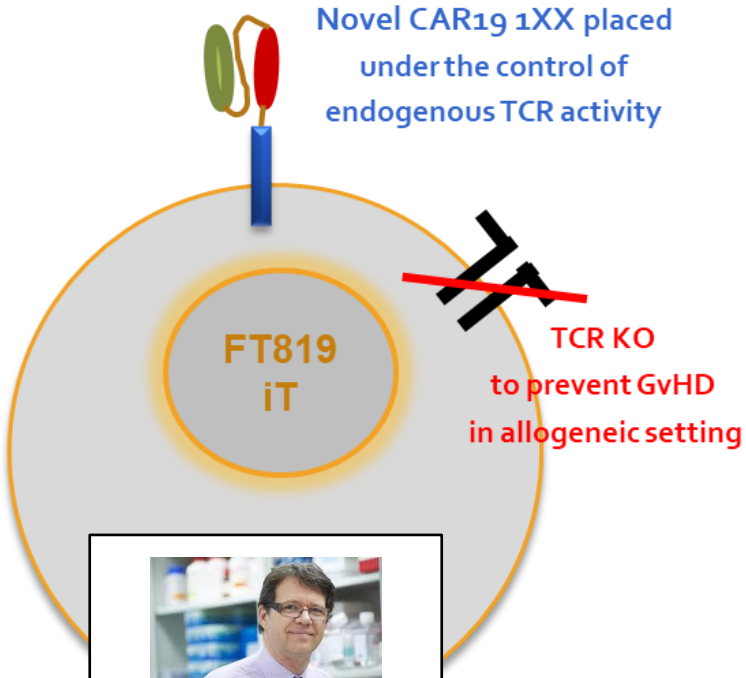
Fate THERAPEUTICS

iPSC-derived CAR T Cells

FT819: Off-the-Shelf CAR19 T-Cell Product Candidate

Collaboration with Memorial Sloan Kettering Cancer Center

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



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

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

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

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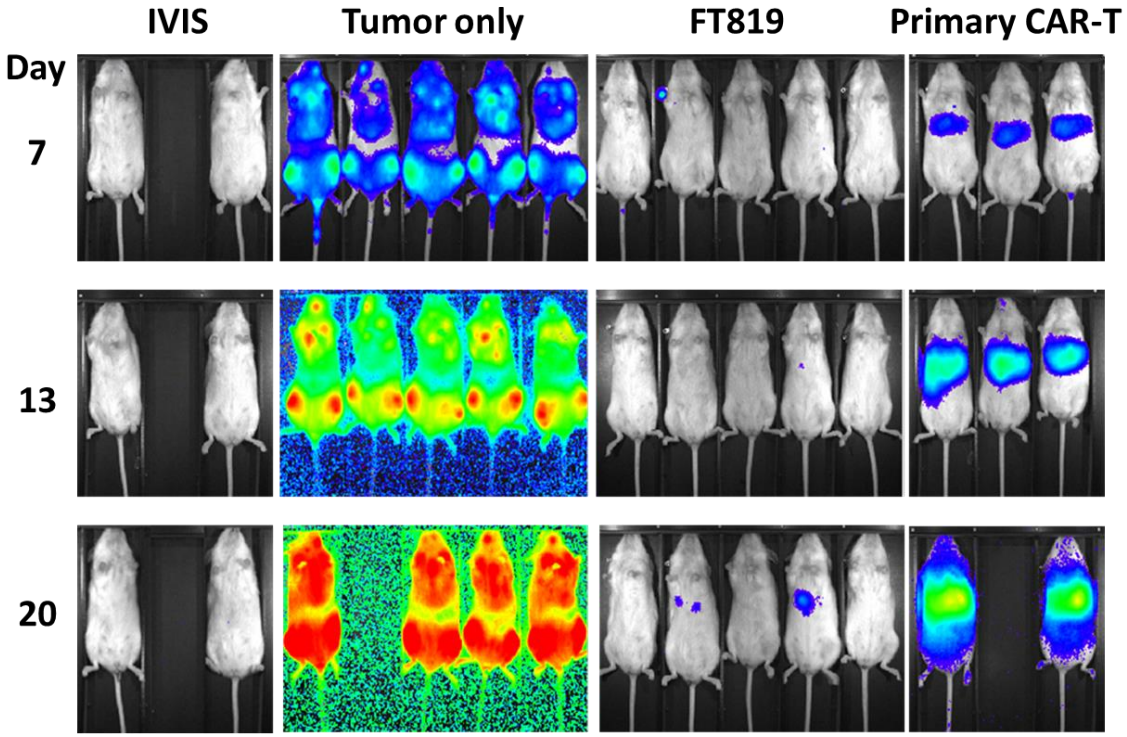
TCR null: Bi-allelic disruption of TRAC at the clonal level for complete removal of TCR expression and the elimination for the possibility of GvHD in allogeneic setting

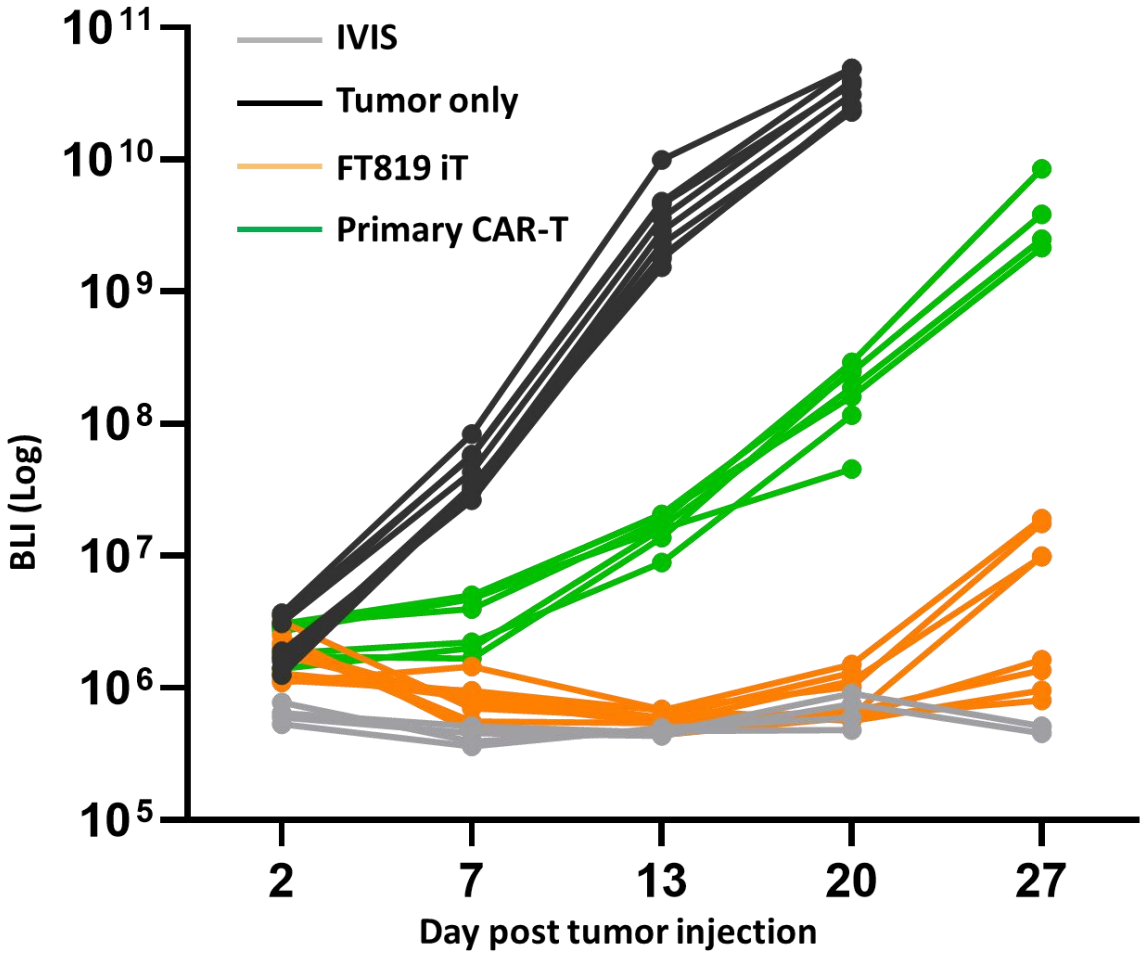
IND Allowed by FDA for BCL, CLL and pre-B ALL

Fate THERAPEUTICS

FT819: Enhanced Tumor Control vs. Primary CAR T Cells

Disseminated Xenograft Model of Lymphoblastic Leukemia





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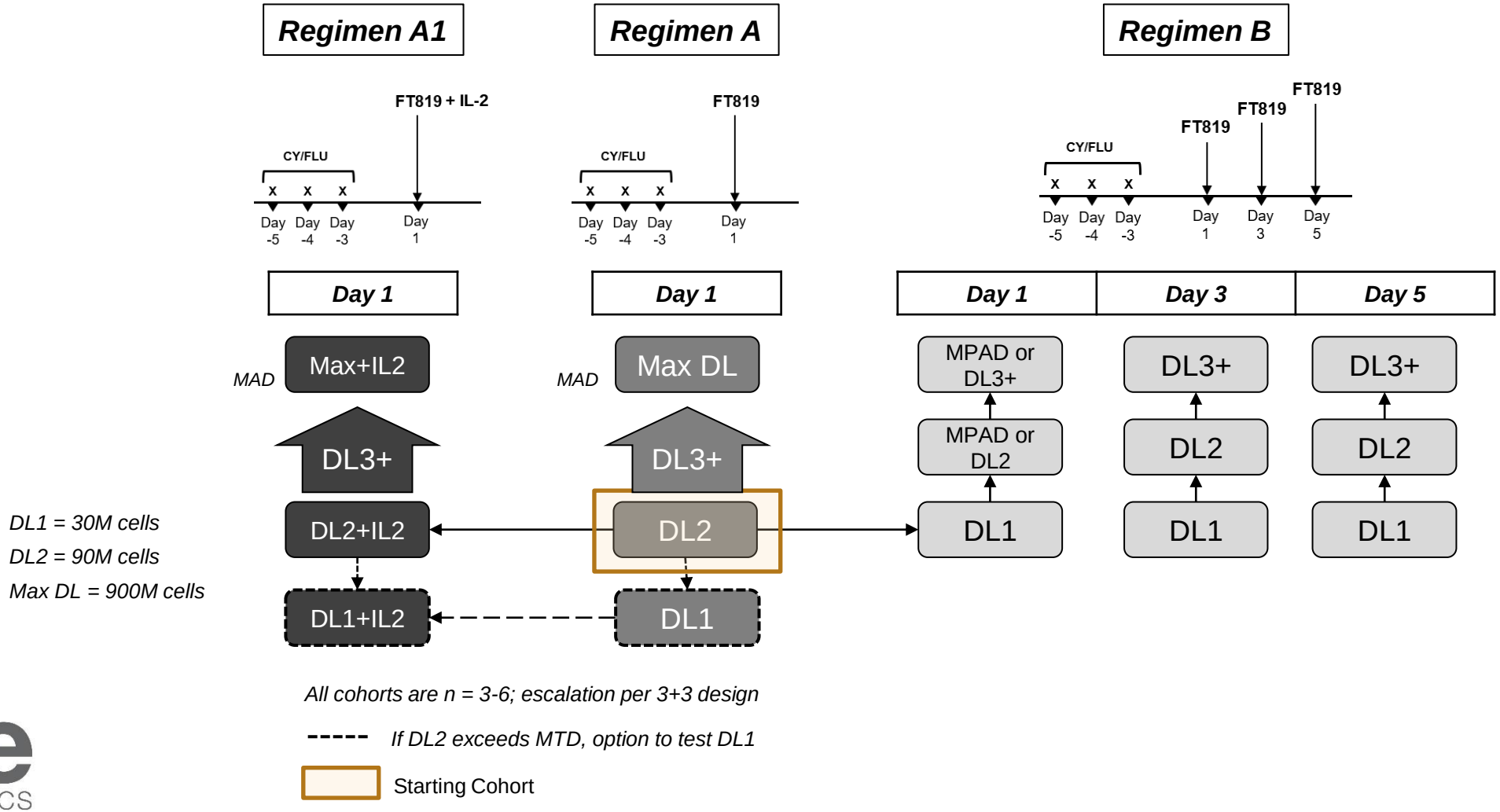
Memorial Sloan Kettering Cancer Center

Valamehr et al. Festival of Biologics Annual Meeting 2020

FT819-101: Phase I Dose Escalation Schema

Concurrent and Independent Dose Escalation in BCL, CLL and pre-B ALL

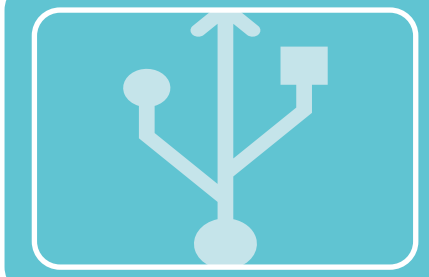
3 Indications x 3 Treatment Regimens



Collaborations

Janssen Cancer Immunotherapy Collaboration (April 2020)

Off-the-shelf, iPSC-derived CAR NK Cell and CAR T-Cell Collaboration

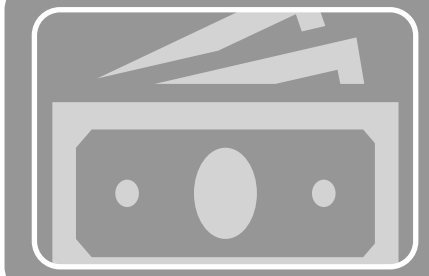


Oncology Innovation

- Proprietary antigen domains contributed by Janssen
- Up to 4 targets including hematologic malignancies and solid tumors
- Substantial investment in next-generation cellular features / functionality

Strategic Collaboration

- FATE leads preclinical development to IND submission
- Janssen option to global clinical development and commercialization
- FATE retains option to 50-50 US commercialization



Significant Economics

- $100m upfront (+$50m equity put)
- Janssen pays for all collaboration costs
- $3+ billion in milestones, double-digit royalties

ONO Cancer Immunotherapy Collaboration (September 2018)

Off-the-shelf, iPSC-derived CAR T-Cell Collaboration

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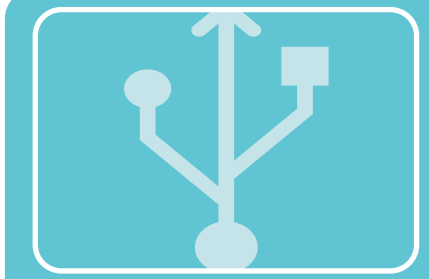
ONO PHARMACEUTICAL CO.,LTD.

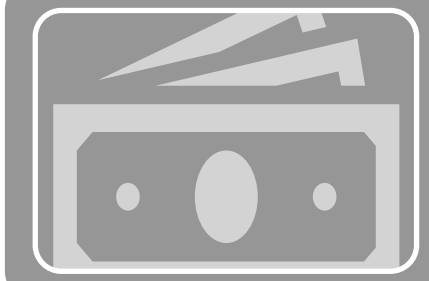


Oncology Innovation

- Proprietary antigen domain contributed by Ono
- Targeting solid tumors
- Potential to include additional antigen binding domains

Strategic Collaboration

- FATE leads preclinical development to pre-IND milestone
- Ono option to global development and commercialization
- FATE retains option to 50-50 worldwide rights ex Asia



Financial Terms

- $10m upfront
- 50-50 cost sharing to pre-IND milestone
- Up to $895 million in milestones, mid-single to low double-digit royalties

Fate THERAPEUTICS

Financials

Financial Summary

As reported in Company's Consolidated Financial Statements

Three Months Ended March 31, 2021	
Revenue	$11.1M
Operating Expense[1]	$57.3M
Cash & Cash Equivalents	$888M
Employees	300+
Total Shares Outstanding[2]	107.9M

[1] *Includes $13m in stock-based compensation*

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

Fate THERAPEUTICS

Better Cells For Better Therapies™