Fate THERAPEUTICS

Better Cells For Better Therapies™

Programmed Cellular Immunotherapies

Transforming the Treatment of Cancer with Off-the-shelf, Multiplexed-engineered, iPSC-derived Cellular Immunotherapy

February 2022

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 safety and therapeutic potential of the Company's product candidates, the advancement of and plans and timelines related to the Company's ongoing and planned clinical studies and the clinical investigation of its product candidates, the timing for the Company's receipt of data from its clinical trials and preclinical studies, and the Company's clinical development and regulatory strategy. 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 studies of its product candidates, including interim results and results from earlier studies, may not be predictive of final results or results 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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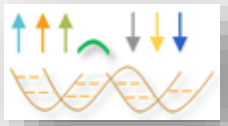
Fate Therapeutics

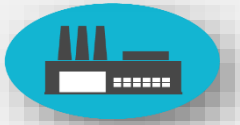
The Leading Developer of Off-the-shelf, iPSC-derived Cellular Immunotherapies



Disruptive Technology Platform: industry-leading iPSC product platform supported by 10+ years of internal R&D and dominant IP estate with 350+ issued patents

Leading Off-the-shelf NK & T-cell Product Pipeline: 9 clinical programs addressing unmet medical needs in lymphoma, multiple myeloma, AML and solid tumors

Demonstrated Clinical Benefit: treated 100+ patients with novel, multi-dose treatment paradigm showing compelling therapeutic benefit and differentiated safety profile



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

World Class Partnerships: creating novel iPSC-derived CAR NK and CAR T-cell therapies for hematologic malignancies and solid tumors with Ono and Janssen

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Changing the Game in Cell Therapy

Established Leadership Position in Off-the-shelf, Cell-based Cancer Immunotherapy

Multiplexed Engineering

Incorporate multiple mechanisms of action to eradicate cancer

Mass Production

Scalable manufacturing with high yield / low cost per dose

Uniform Products

Consistent identity, purity and potency of cell products

Off-the-Shelf

Stable, cryopreserved for on-demand treatment

Treatment Paradigm

Out-patient treatment strategies to maximize patient reach

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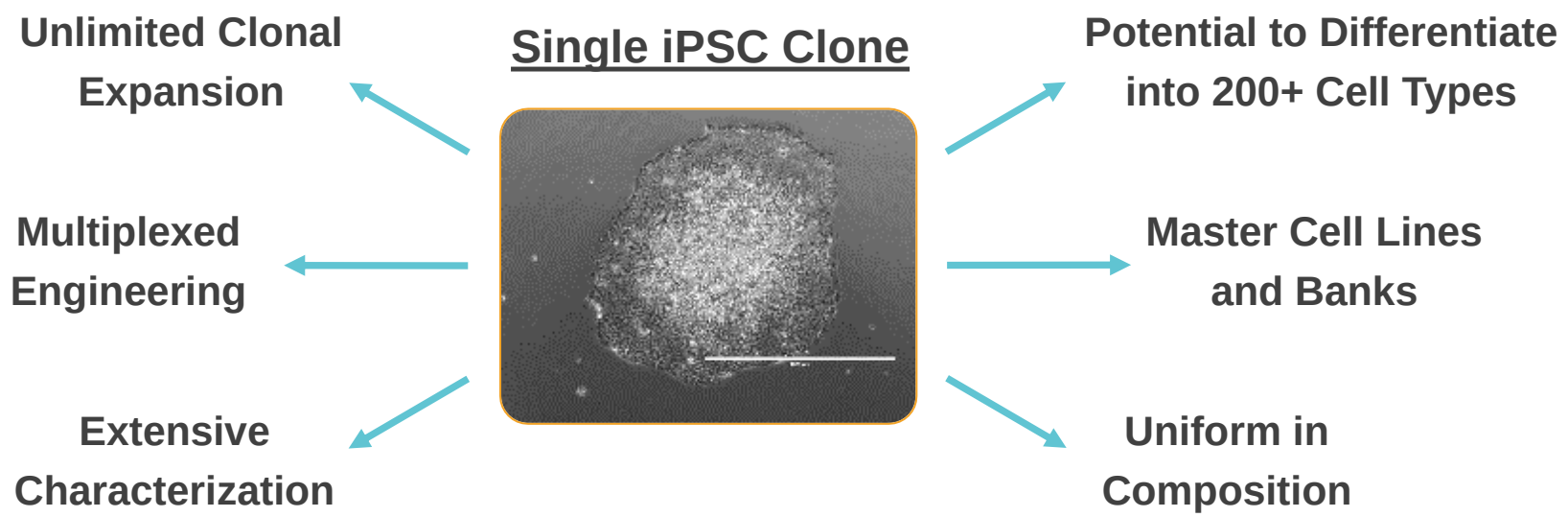
Creating Master Multiplexed-engineered iPSC Lines

Isolation, Characterization & Selection of Single Multiplexed-engineered iPSC

A Single Human Induced Pluripotent Stem Cell (iPSC)

A renewable source for mass production of cell products



Fate Therapeutics' iPSC product platform is supported by an IP portfolio with 350+ issued patents and 150+ pending patent applications

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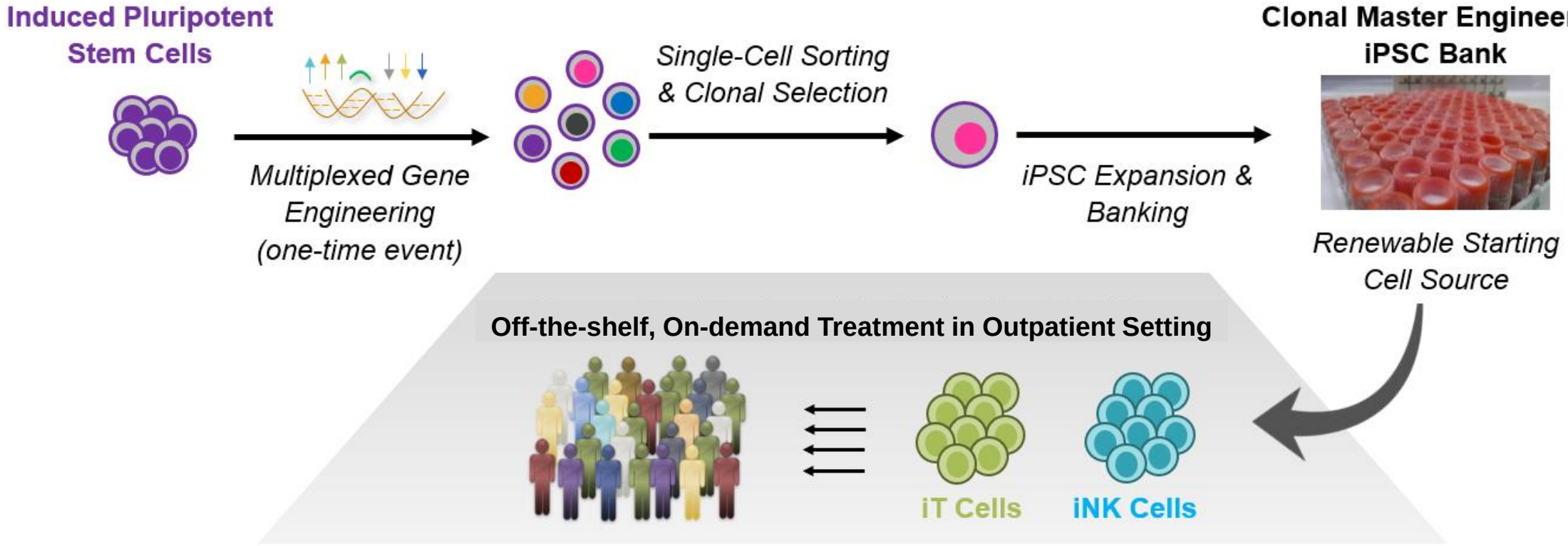
Mass Production of Off-the-shelf, Cell-based Cancer Immunotherapies

Use of Master Multiplexed-engineered iPSC Bank as Starting Material



- ✓ **Multiplexed engineering**
- ✓ **Homogeneous product**
- ✓ **Mass production**
- ✓ **Off-the-shelf**

Multiple tumor-fighting mechanisms

High quality; consistent purity and activity

High yield; low cost per dose

On-demand; expanded patient reach

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Transforming the Treatment of Cancer

Leveraging Unique Advantages of Off-the-shelf, iPSC-derived Cellular Immunotherapy

Earlier Treatment Settings	• Differentiated safety profile that enables early intervention • Off-the-shelf convenience that supports community reach
Outpatient Treatment	• Reliable administration without the need for hospitalization • Monoclonal antibody-like administration paradigm
Combination Therapies	• Leverage multiple, complementary mechanisms of action • Augment activity of standard anti-cancer therapies
More Frequent Administration	• Flexible dose & schedule to optimize clinical benefit • Multi-doses to drive deeper, durable anti-tumor response

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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**	**P1**	**SS**	**PC**

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

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

Projected 2022 Corporate Milestones

Hematologic Malignancies	• Launch registration study under RMAT for relapsed / refractory aggressive BCL • Initiate early-line aggressive BCL study for FT596 + R-CHOP • Generate clinical datasets with FT516 / FT596 (BCL), FT538 (MM, AML), FT576 (MM) and FT819 (BCM)
Solid Tumors	• Generate dose-escalation datasets with FT538 + mAb therapy to enhance ADCC • Initiate dose-escalation study of FT536 as novel pan-tumor targeting strategy • Complete IND-enabling studies of B7H3-targeted CAR programs
Innovation	• Nominate two novel multi-antigen targeted programs for solid tumors • Complete preclinical development of ADR functionality to enable conditioning-free cell therapy • Complete preclinical development of TSR functionality to enhance TME functional persistence
Partnerships	• Submit IND to FDA for first iPSC-derived CAR NK cell program under Janssen partnership • Complete IND-enabling studies for iPSC-derived CAR T-cell program under Ono partnership • Expand iPSC-derived product pipeline through additional collaborations
Corporate	• Complete tech transfer and initiate technical operations at commercial GMP facility • Continue expansion of dominant IP portfolio with 350+ issued patents • Maintain strong balance sheet

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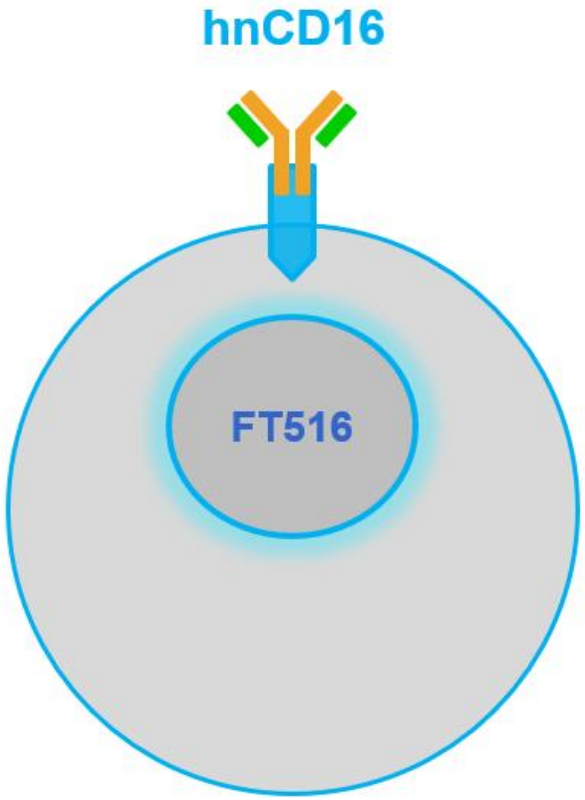
ADCC *= antibody-dependent cellular cytotoxicity;* ***ADR*** *= allo-defense receptor;* ***AML*** *= acute myeloid leukemia;* ***BCL*** *= B-cell lymphoma;* ***BCM*** *= B-cell malignancies;* ***MM*** *= multiple myeloma;* ***TME*** *= tumor microenvironment;* ***TSR*** *= tumor suppressive redirector*

B-cell Malignancy Franchise

Off-the-Shelf, iPSC-derived NK Cell Franchise for B-cell Malignancies

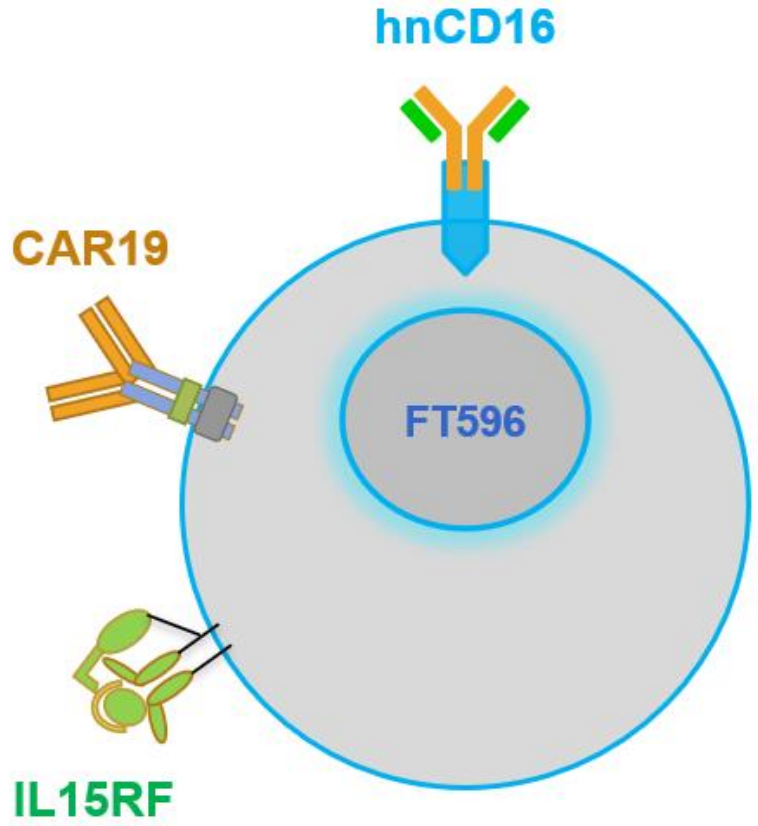
FT516 and FT596 Product Candidates



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 cell biology

IL-15RF: Interleukin-15 receptor fusion to promote survival, proliferation and anti-tumor activity



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

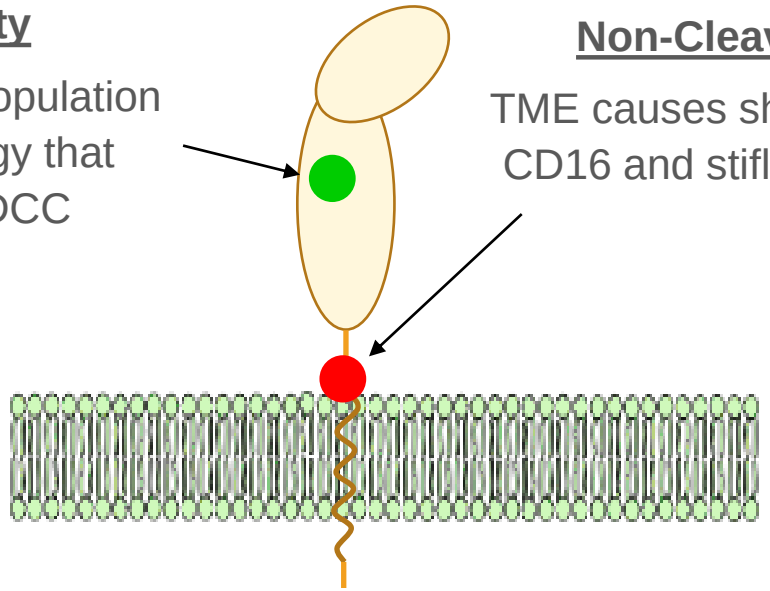
FT516: Novel High-Affinity, Non-Cleavable CD16a Fc Receptor

Optimizing Antibody-Dependent Cellular Cytotoxicity for Use with mAb Therapy

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

High Affinity

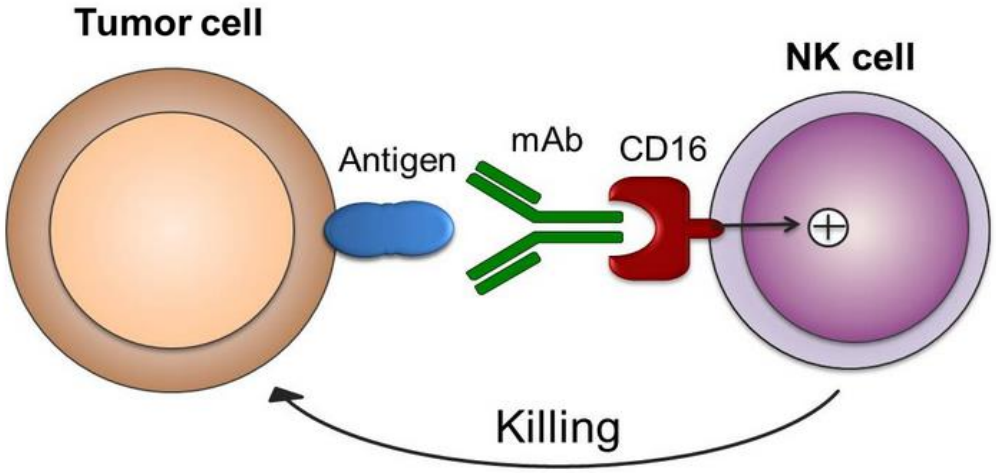
Only 15% of the population has CD16 biology that maximizes ADCC



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

RYBREVANT

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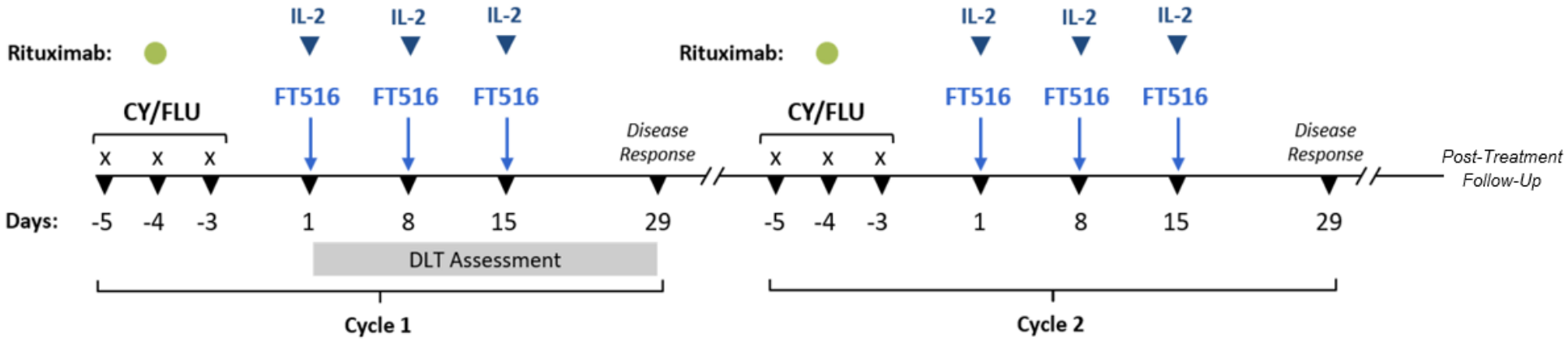
FT516-101: Phase 1 Study in R/R B-cell Lymphoma

- First-in-human study assessing the safety and activity of FT516 combined with rituximab in patients with r/r B-cell lymphoma
  - Primary objective: identify DLT and determine maximum tolerated dose/maximum assessed dose
  - Additional objectives: safety, tolerability, preliminary activity, pharmacokinetics, and immunogenicity
- Novel treatment paradigm
  - Two cycles of treatment, with each cycle consisting of 3 days of conditioning (CY / FLU) and 1 dose of rituximab (375 mg/m^2) followed by 3 weekly infusions of FT516 with IL-2 cytokine support
  - No mandatory hospitalization required during the treatment period



Cyclophosphamide: 500 mg/m² IV x 3 days Fludarabine: 30 mg/m² IV x 3 days Rituximab: 1 dose at 375 mg/m² IV per cycle IL-2: 6M units sc with each FT516 dose

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FT516-101: Interim Phase 1 Clinical Data in R/R B-cell Lymphoma

80% ORR in CAR T Naïve Population; 38% CR in Prior CAR T Population

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Interim Phase 1 Data at ≥90M Cells / Dose [1]

	CAR T Naïve		Prior CAR T
	Aggressive BCL	Indolent BCL	Aggressive BCL
n	5	5	8
Response [2]			
Objective Response (%)	4 (80%)	4 (80%)	3 (38%)
Complete Response (%)	2 (40%)	3 (60%)	3 (38%)
Durability of Response [3]			
Response Rate – 3 Months (%)	4 (80%)	4 (80%)	3 (38%)
Ongoing Responders[1] (%)	3 (60%)	3 (60%)	2 (25%)
Median Follow-up (months)	8.3 (4.6, 9.9)	9.9 (3.7, 13.2)	6.5 (4.6, 8.3)

[1] As of data cutoff date of October 18, 2021
[2] Cycle 2 Day 29 protocol-defined response assessment per Lugano 2014 criteria
[3] Measured from initiation of therapy

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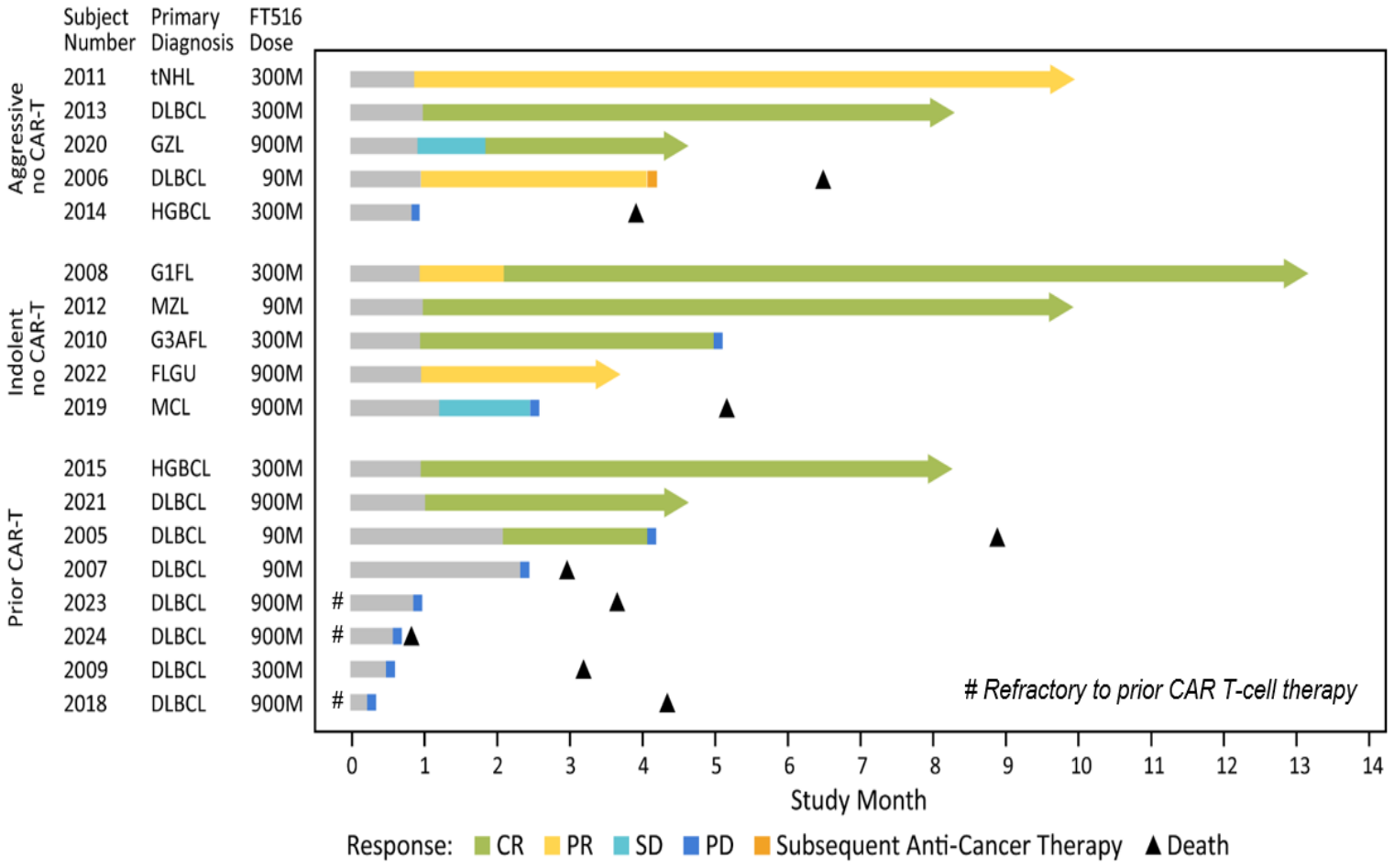
FT516-101: Interim Phase 1 Clinical Data in R/R B-cell Lymphoma

Median Duration of Response Not Reached at ≥90M Cells / Dose



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Safety & Tolerability

- No CRS, ICANS, GvHD
- No treatment discontinuations due to AEs

Response Rates

- 11 of 18 patients (61%) treated at ≥90M cells / dose achieved OR, including 8 of 10 patients (80%) naïve to treatment with prior CAR T-cell therapy

Durability of Response

- 11 patients (61%) remained in ongoing response at 3 months from initiation of treatment. As of data cutoff date:
- *Naïve CAR T*. 6 of 10 (60%) patients continued in ongoing response at 9.1m MFU, including 4 patients >6m; longest FU = 13.2m
- *Prior CAR T*. 2 of 8 (25%) patients continued in complete response at 6.5m MFU, including 1 patient >6m; longest FU = 8.3m

As of the data cutoff date (18 Oct 2021); Response assessment per Lugano 2014 criteria. Data subject to source document verification.

CAR = Chimeric antigen receptor; **CR** = Complete response; **DLBCL** = Diffuse large B-cell lymphoma; **FLGU** = Follicular lymphoma grade unknown; **G1FL** = Grade 1 follicular lymphoma; **G3AFL** = Grade 3A follicular lymphoma; **GZL** = Gray zone lymphoma; **HGBCL** = High-grade B-cell lymphoma; **M** = Million; **MCL** = Mantle cell lymphoma; **FU** = Follow-up; **MZL** = Marginal zone lymphoma (B-cell lymphoma, of small to intermediate size cells, most likely MZL); **PD** = Progressive disease; **OR** = Objective response; **PR** = Partial response; **SD** = Stable disease; **R/R** = Relapsed/refractory; **tNHL** = Transformed indolent lymphoma

FT516-101: Ongoing Dose Expansion

Dose Expansion at 900M Cells / Dose; Favorable Safety Profile Enables Exploration of Higher Cell Doses

R/R DLBCL post-aCD19 CAR T-cell therapy

- Signal observed in both FT516 and FT596 Phase 1 studies
- Potential fast-to-market pathway / pivotal study launch in 2H22
- Expansion in up to 30 patients; including community sites

3L+ R/R DLBCL and FL

- Potential favorable safety / competitive efficacy profile compared to auto CAR19 T-cell therapy
- Off-the-shelf, on-demand availability, broad patient accessibility
- Expansion in up to 30 patients; including community sites

Earlier-line rituximab-containing SOC regimen

- Cellular complement to R-Benda SOC regimen
- Assess activity without Cy / Flu conditioning
- Expansion in up to 30 patients; including community sites

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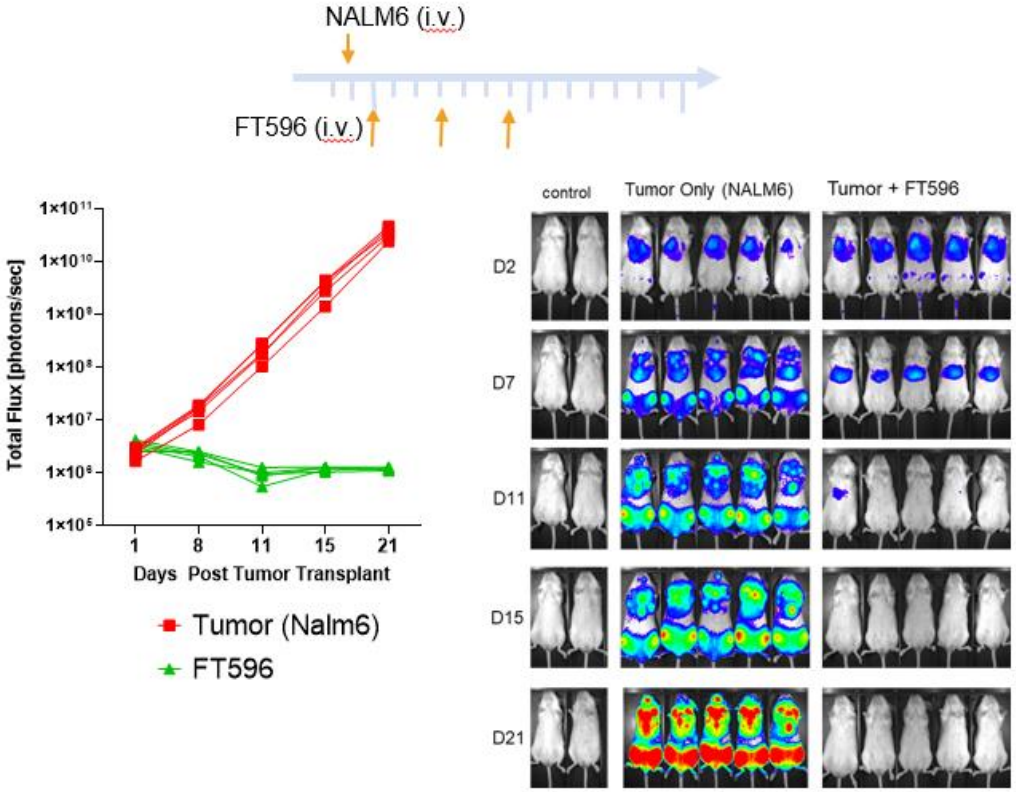
FDA **RMAT Designation**

FT596: hnCD16 + CAR19 + IL15-RF iPSC-derived NK Cell Product Candidate

Novel Dual-antigen Targeting Strategy to Overcome Tumor Heterogeneity and Antigen Escape

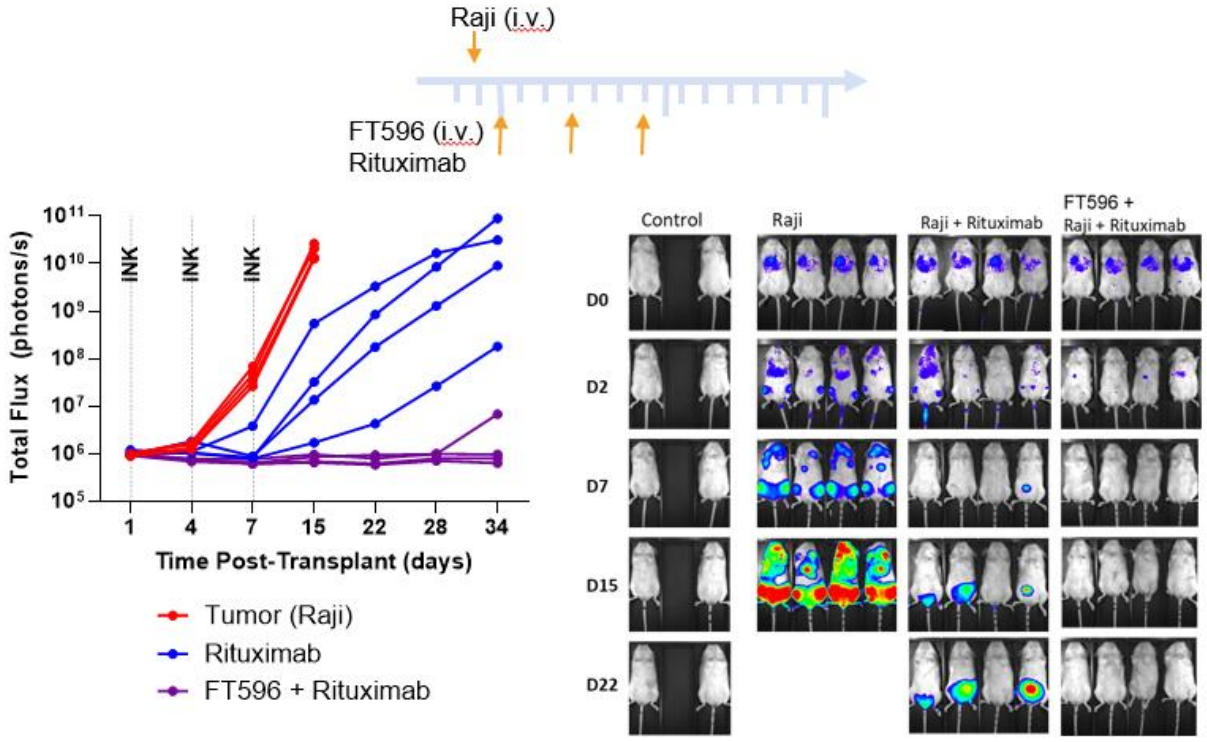
CAR-mediated Cytotoxicity

Leukemia xenograft NSG immunodeficient mouse model



hnCD16-mediated Cytotoxicity

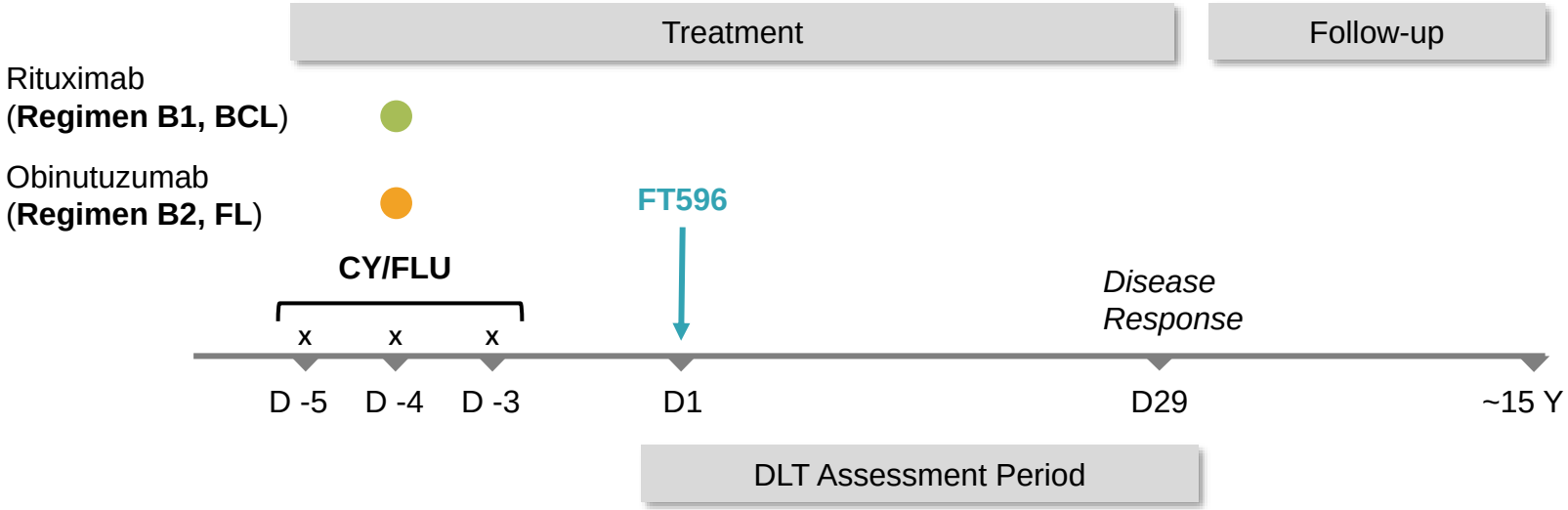
Lymphoma xenograft NSG immunodeficient mouse model



FT596-101: Phase 1 Study in R/R B-cell Lymphoma

Monotherapy and in Combination with CD20-targeted Monoclonal Antibody Therapy

- Single-dose treatment cycle
- Option for second single-dose treatment cycle
  - Initially required FDA consent
  - Requirement lifted based on acceptable safety and maintenance of clinical benefit
- 2-dose treatment schedule (FT596 administered on Days 1 and 15) initiated in November 2021



Monoclonal Antibody Therapy on Day -4: Rituximab 375 mg/m^2; Obinutuzumab 1000 mg/m^2; CY = Cyclophosphamide 500 mg/m^2; FLU = Fludarabine 30 mg/m^2

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FT596-101: Interim Phase 1 Clinical Data in R/R B-cell Lymphoma

75% ORR and 58% CR in Single-dose Combination Arm at ≥300M Cells

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Interim Phase 1 Data – Single Dose [1]				
1 Dose x 1 Cycle	**Monotherapy (n=13)**		**Combination (n=19)**	
Single-dose Level Cohorts (Cells)	OR	CR	OR	CR
30M	1/3 (33%)	0	0/3 (0%)	0
90M	3/4 (75%)	2	2/4 (50%)	2
300M [2]	4/5 (80%)	1	4/6 (67%)	3
900M [2]	0/1 (0%)	0	5/6 (83%)	4
aCD19 History (≥90M Cells)	n=10		n=16	
Naïve	7/9 (78%)	3	5/8 (63%)	4
Prior	0/1 (0%)	0	6/8 (75%)	5
Disease Histology (≥90M Cells)	n=10		n=16	
Aggressive	1/3 (33%)	0	6/11 (55%)	4
Mantle cell	0/1 (0%)	0	2/2 (100%)	2
Indolent	6/6 (100%)	3	3/3 (100%)	3

[1] As of data cutoff date of October 11, 2021, unless otherwise noted. Objective response and complete response are based on Cycle 1 Day 29 protocol-defined response assessment per Lugano 2014 criteria. Data subject to source document verification.

[2] Cycle 1 Day 29 protocol-defined response assessment completed subsequent to data cutoff date for one patient in the third single-dose cohort of 300 million cells in the Combination Arm and seven patients in the fourth single-dose cohort of 900 million cells (n=1 in Monotherapy Arm; n=6 in Combination Arm).

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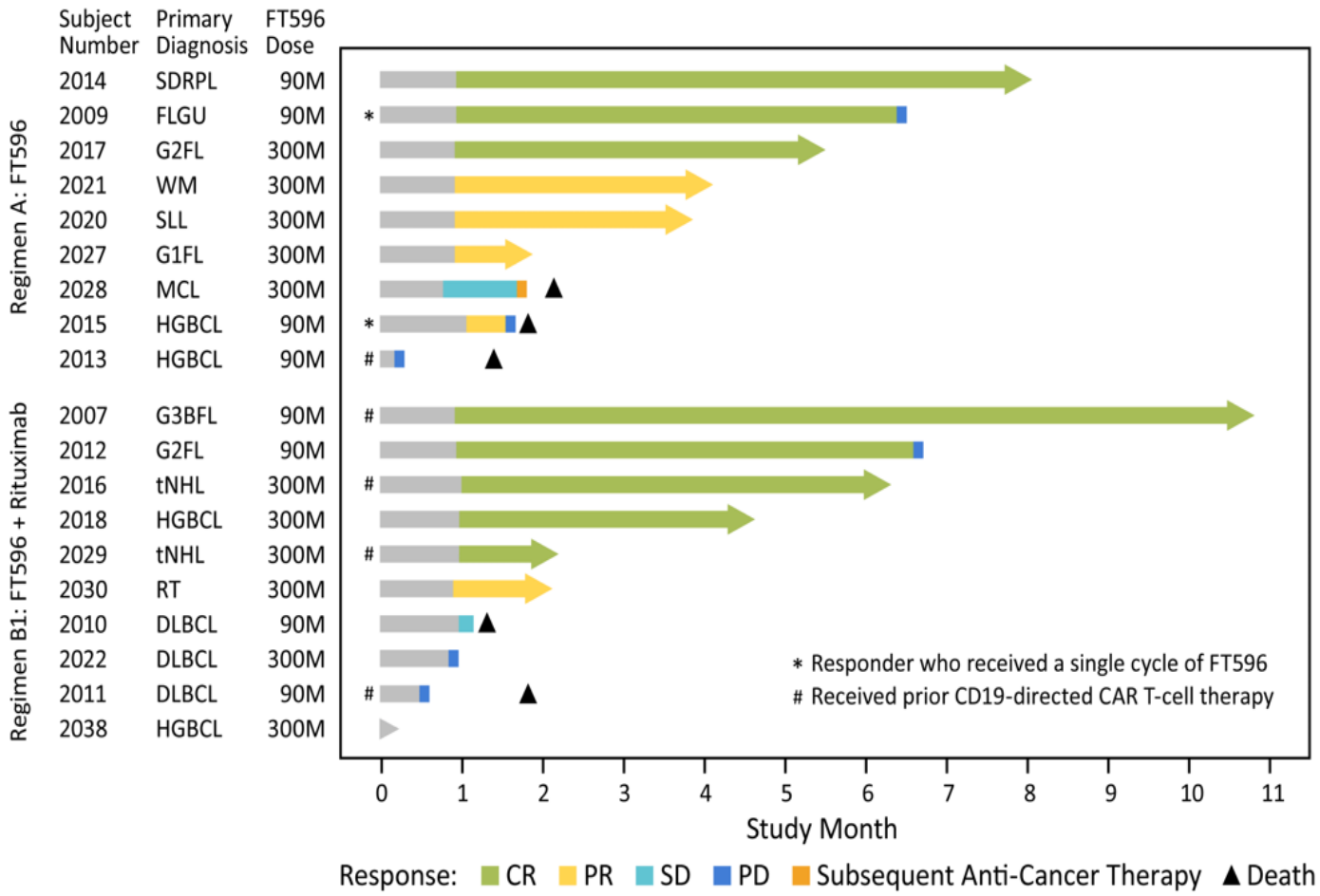
FT596-101: Interim Phase 1 Clinical Data in R/R B-cell Lymphoma

Median Duration of Response Not Reached at 90M and 300M Cell Dose



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Safety & Tolerability

- No DLTs, ICANS, or GvHD
- Observed CRS (n=3) was infrequent, low-grade, and of limited duration

Response Rates at 90M and 300M Cell Dose

- 13 of 19 patients (68%) achieve OR (n=7/9 in Monotherapy Arm; n=6/10 in Combination Arm), including 3 of 5 patients (60%) previously treated with auto CD19 CAR T-cell therapy

Durability of Response

- All patients treated with second FT596 single-dose cycle (n=11) reached six months in CR (n=4) or continued in ongoing response (n=7)
- *Combination Arm*. Of 6 responding patients, 5 patients continued in ongoing response at 4.6m MFU, including 2 patients in ongoing CR >6m; and 1 patient reached 6m in CR and subsequently had PD at 6.7m
- *Prior CAR T.* All 3 responding patients continued in ongoing response, including 2 patients in ongoing CR >6m

As of the data cutoff date (11 Oct 2021); Response assessment per Lugano 2014 criteria. Data subject to source document verification.

CAR = Chimeric antigen receptor; **CR** = Complete response; **DLBCL** = Diffuse large B-cell lymphoma; **FLGU** = Follicular Lymphoma Grade Unknown; **G2FL**= Grade 2 follicular lymphoma; **G3BFL** = Grade 3B follicular lymphoma; **HGBCL** = High-grade B-cell lymphoma; **M** = Million; **MCL** = Mantle cell lymphoma; **MFU** = Median follow up; **OR** = Objective response; **PD** = Progressive disease; **PR** = Partial response; **RT** = Richter transformation; **SD** = Stable disease; **SDRPL** = Splenic diffuse red pulp small B-cell lymphoma; **SLL** = Small lymphocytic lymphoma; **tNHL** = Transformed indolent lymphoma; **WM** = Waldenstrom macroglobulinemia

FT596-101: Ongoing Dose Escalation

2-dose x 2-cycle Escalation Cohorts in Combination with Rituximab

FT596 2-dose schedule
Day 1 & 15; up to 2 cycles

Potential to dose escalate

FT596 1-dose schedule
Up to 2 cycles

Cleared DLT at 900M cells

FT596 2-dose schedule
Day 1 & 15; up to 2 cycles

900M cells / dose

FT596 2-dose schedule
Day 1 & 15; up to 2 cycles

300M cells / dose

- ***Multiple disease-specific expansion cohorts***
- ***Investigation of FT596 + SOC immunochemotherapy regimens (e.g., FT596 + R-CHOP)***

Responding patients are also eligible for re-treatment following disease progression

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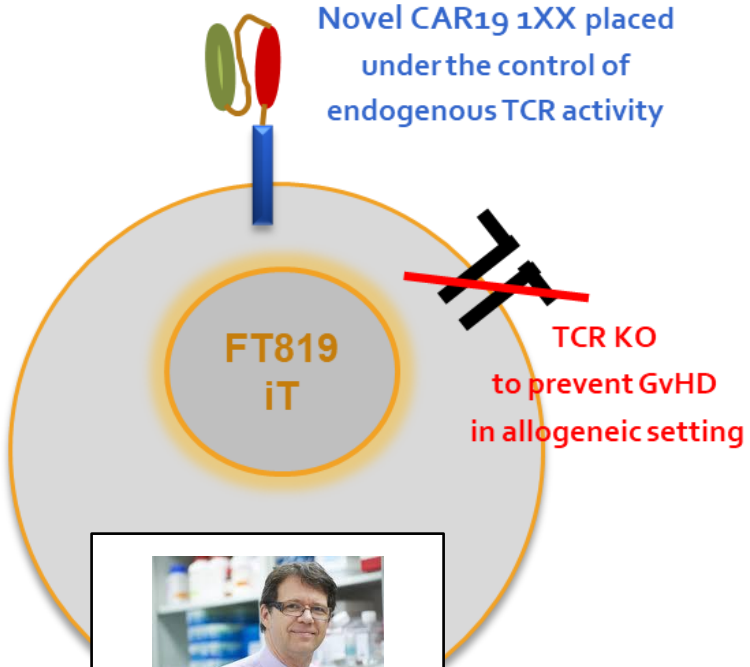
FT819: Off-the-Shelf CAR19 T-Cell Product Candidate

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

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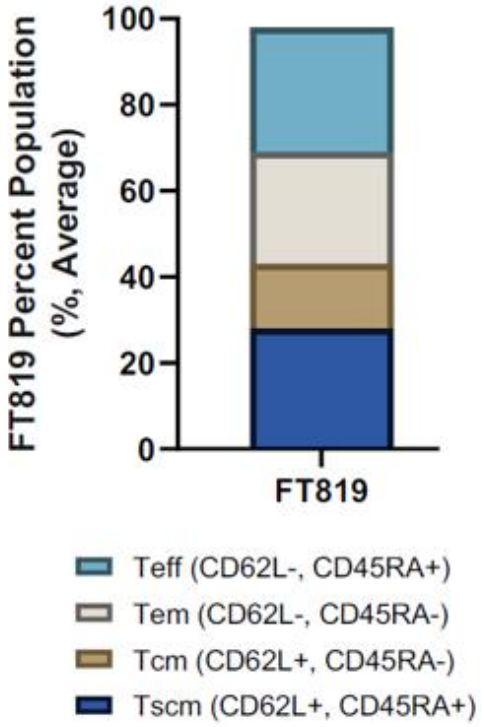
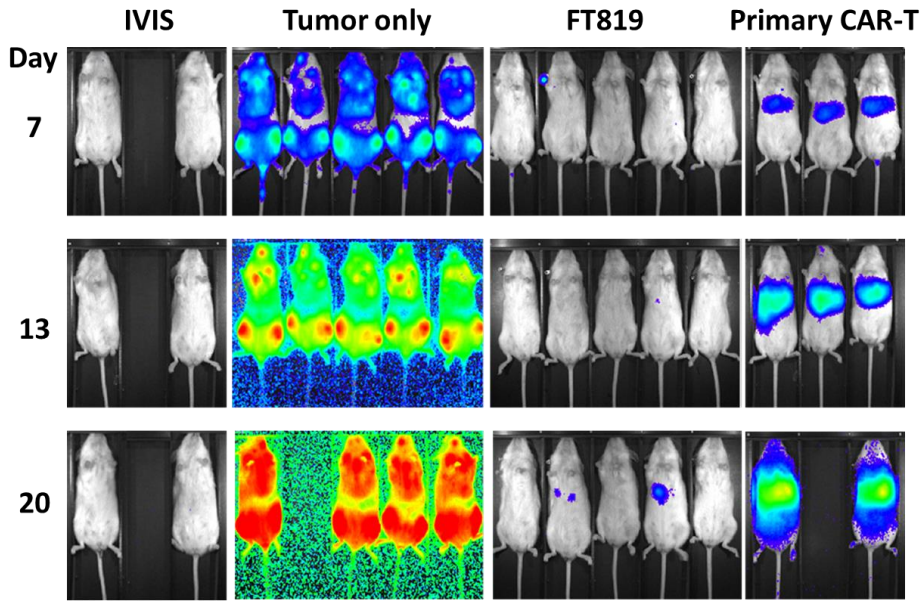
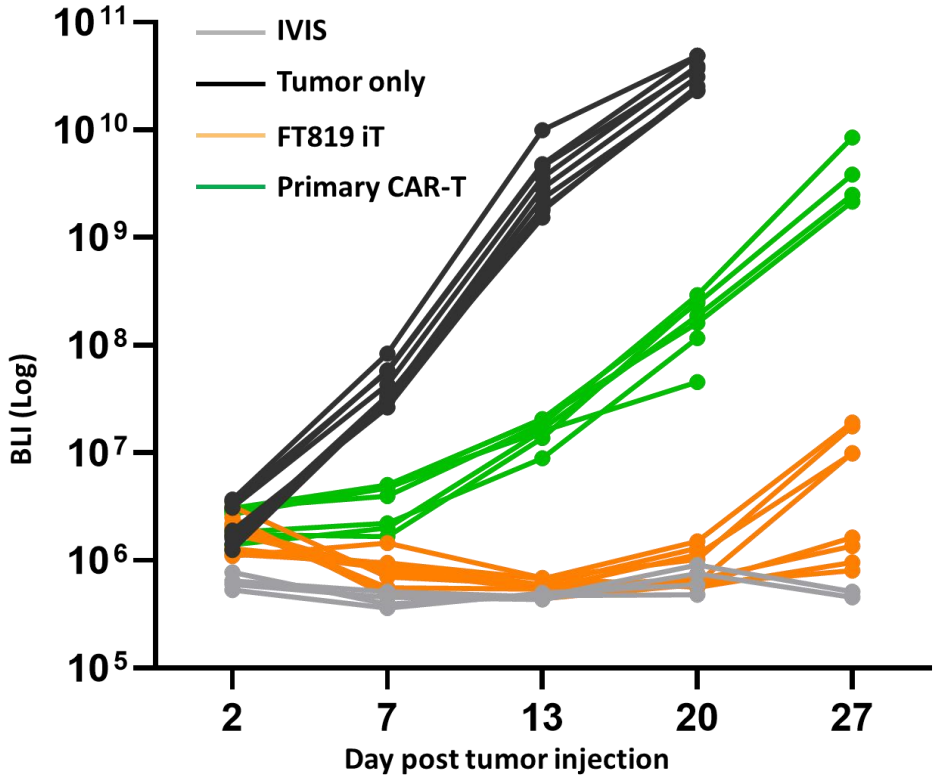
FT819: Enhanced Tumor Control vs. Primary CAR T Cells

Disseminated Xenograft Model of Lymphoblastic Leukemia

FT819 Consists of Memory Phenotype and Outcompetes Primary CAR T Cells In Vivo







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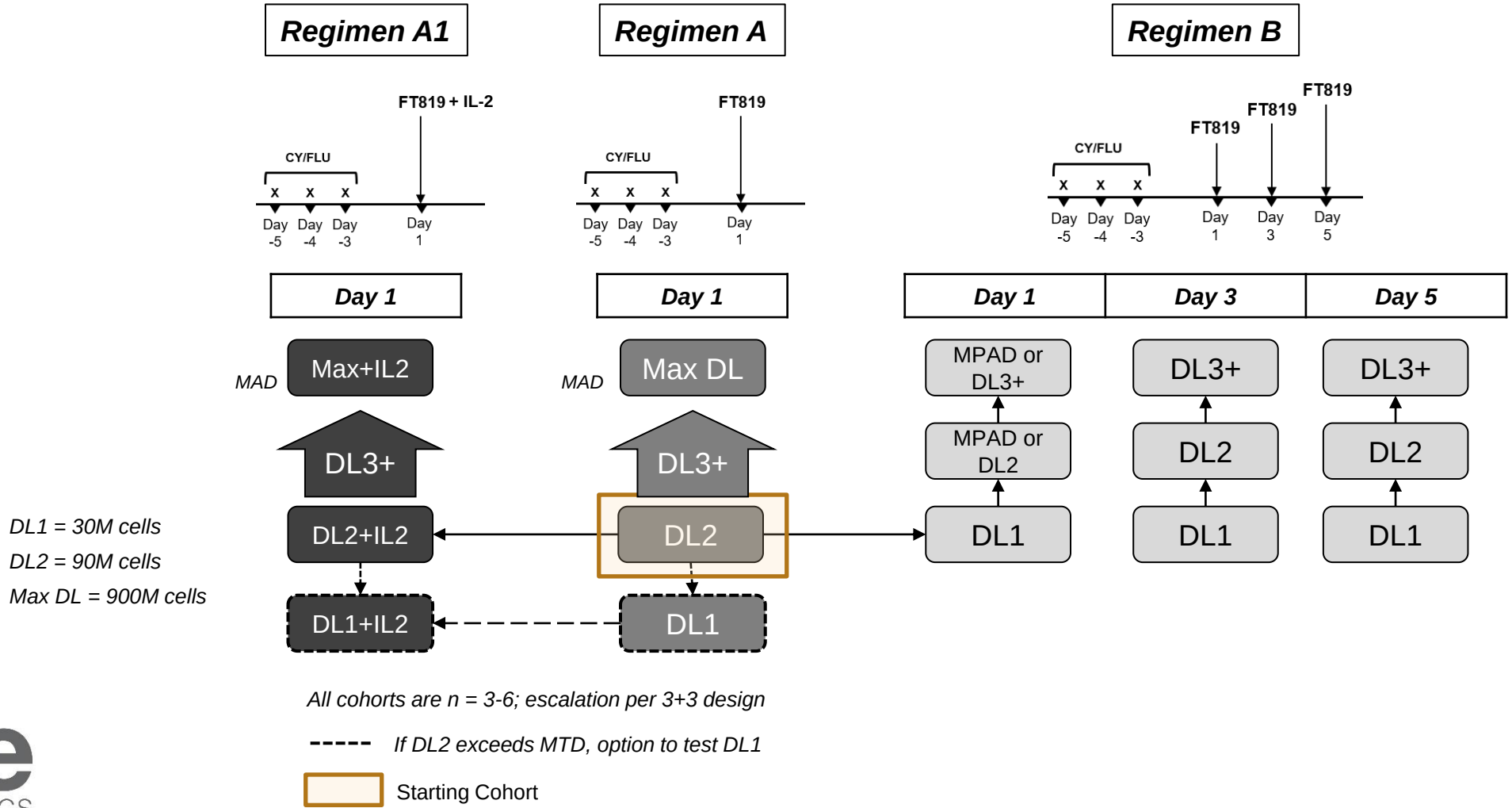
Memorial Sloan Kettering Cancer Center

FT819-101: Phase I Dose Escalation Schema

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

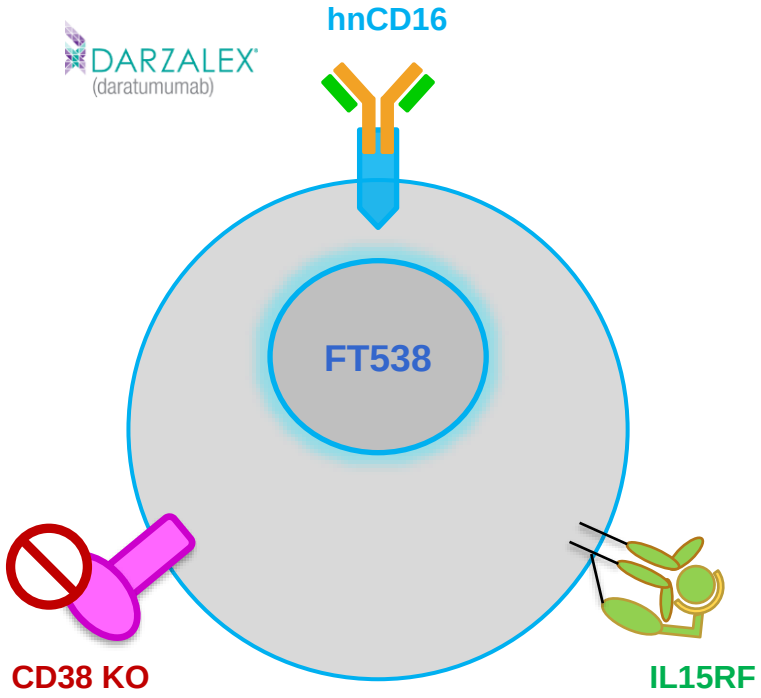
3 Indications x 3 Treatment Regimens; Enrolling DL2 (90M Cells) for BCL, CLL and pre-B ALL



Multiple Myeloma Franchise

Off-the-Shelf, iPSC-derived NK Cell Franchise for Multiple Myeloma

FT538 and FT576 Product Candidates

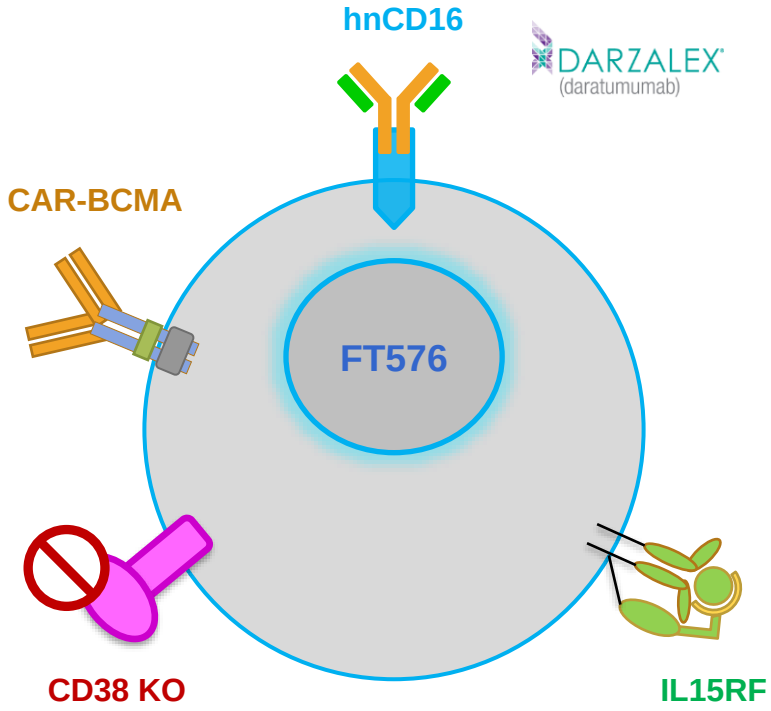


hnCD16: High-affinity 158V, non-cleavable CD16 Fc receptor to augment ADCC

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)



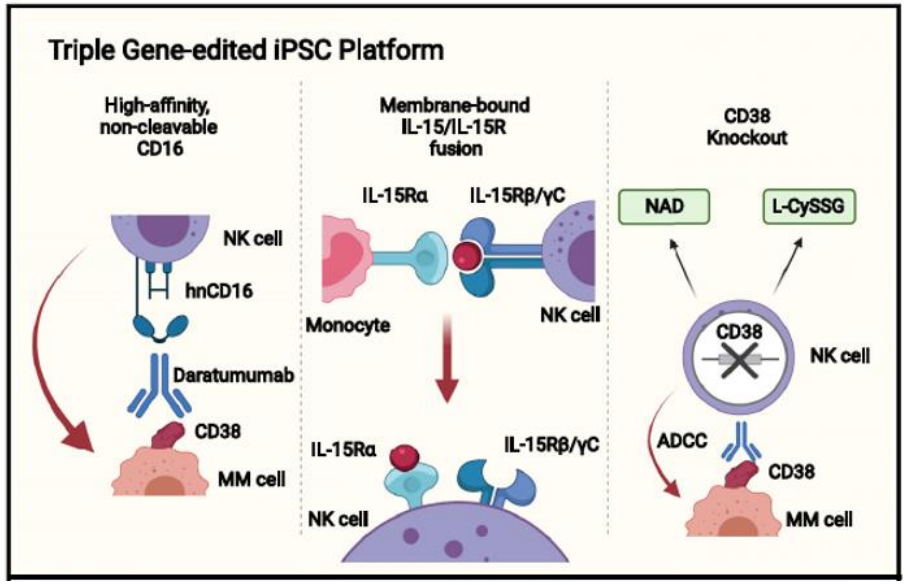
Fc = fragment crystallizable; ADCC - antibody-dependent cellular cytotoxicity; mAb – monoclonal antibody

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

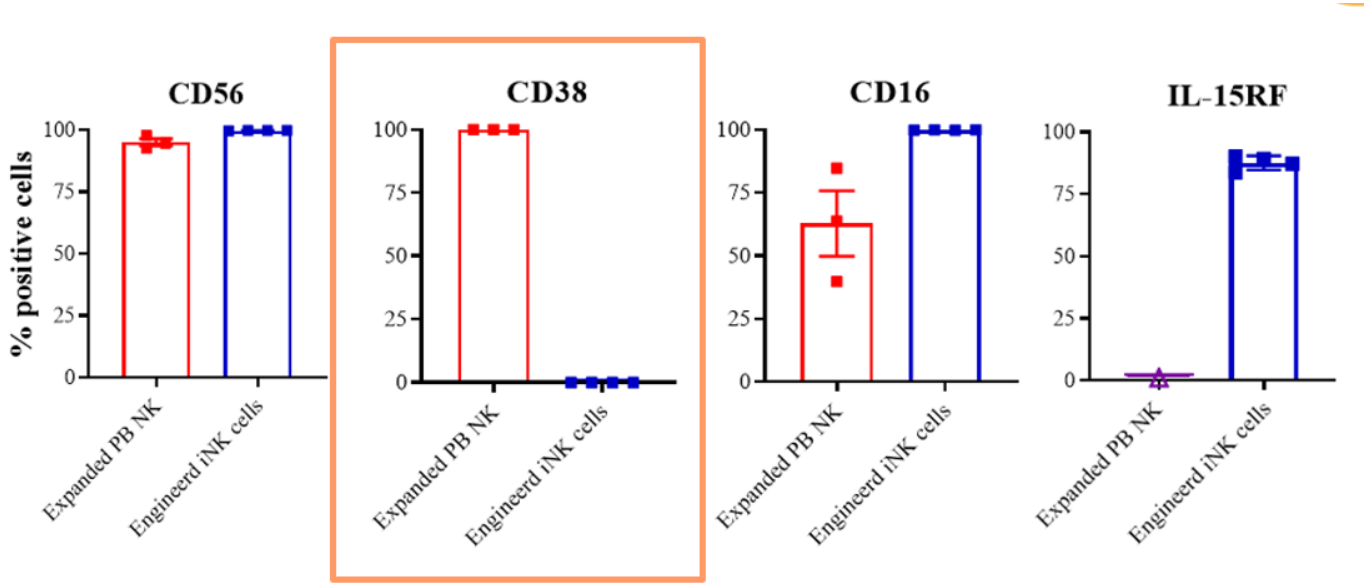
Cell Stem Cell

Woan et al., 2021, Cell Stem Cell *28*, 1–14
December 2, 2021 © 2021 Elsevier Inc.
https://doi.org/10.1016/j.stem.2021.08.013

Harnessing features of adaptive NK cells to generate iPSC-derived NK cells for enhanced immunotherapy



Uniformly engineered with three functional elements designed to optimize innate immunity



- ✓ *The deletion of the CD38 gene (CD38KO) enhances metabolic fitness, promotes persistence, and enables cytotoxic function in high oxidative stress environments (e.g., suppressive tumor microenvironment)*

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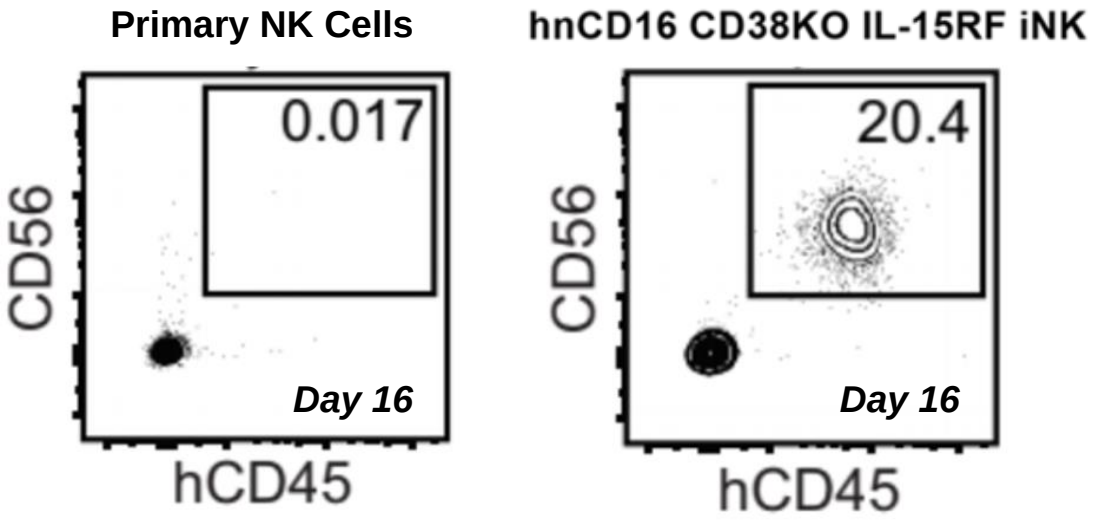
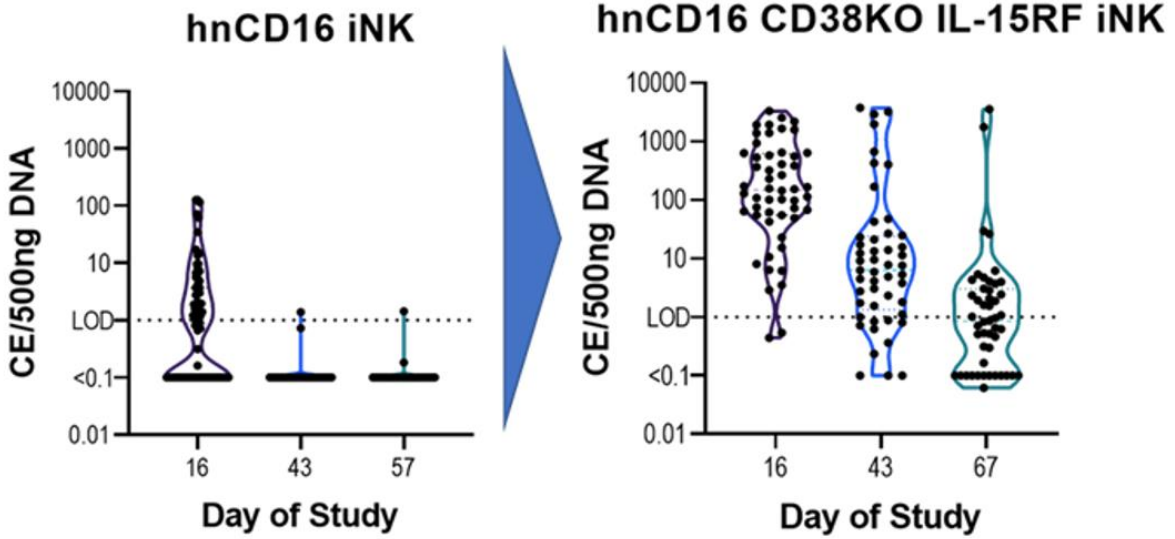
FT538: hnCD16 + IL-15RF + CD38KO NK Cell Product Candidate

Enhanced Persistence Without Cytokine Support

Primary NK vs. FT538 in NSG Mouse



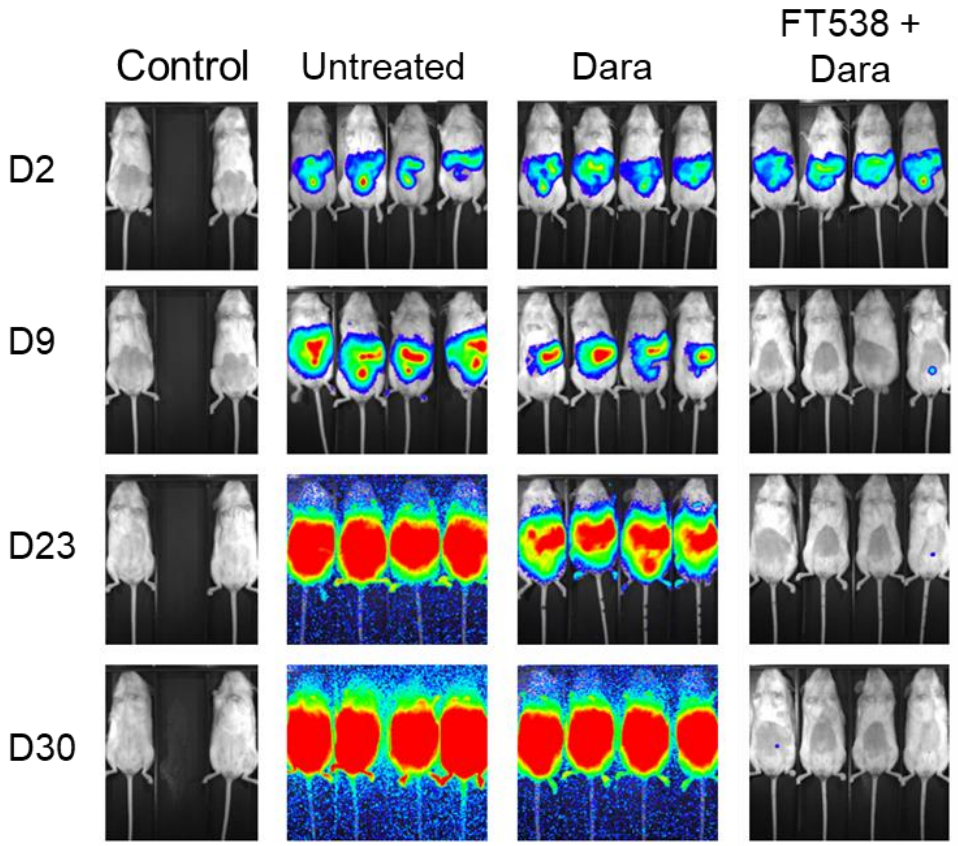
FT516 vs. FT538 in NSG Mouse

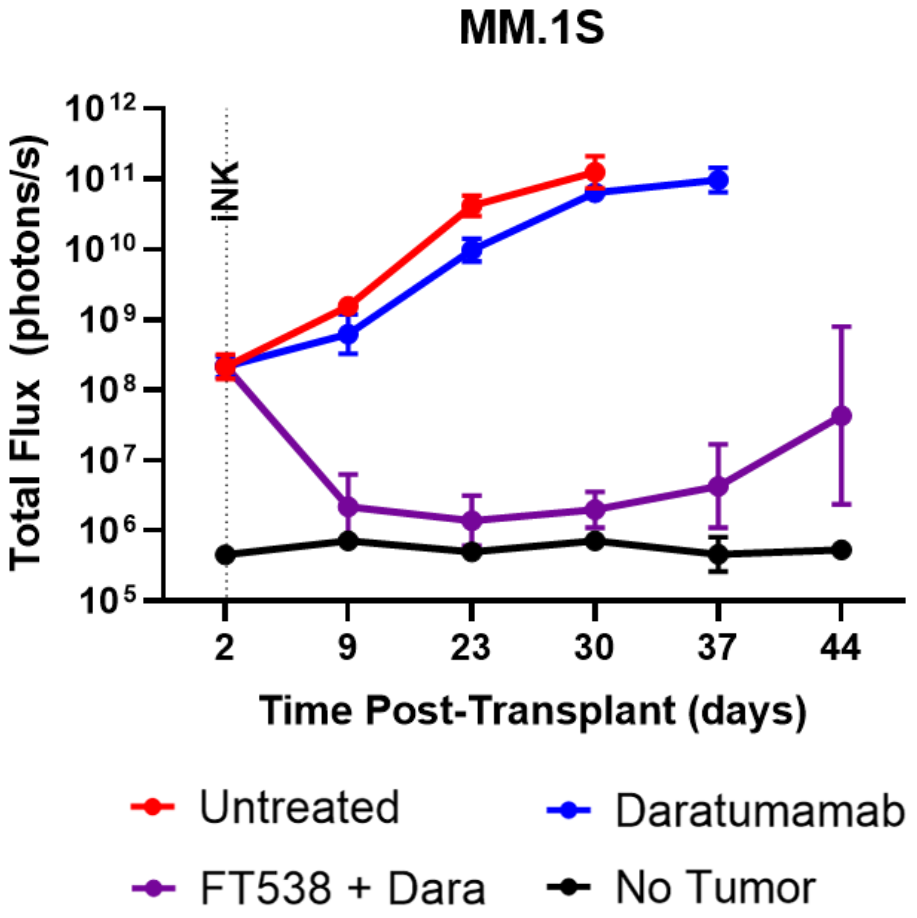


FT538: hnCD16 + IL-15RF + CD38KO NK Cell Product Candidate

Enhanced ADCC in Combination with anti-CD38 mAb In Vivo

First Patients Treated at 100M Cells / Dose in Combination with daratumumab





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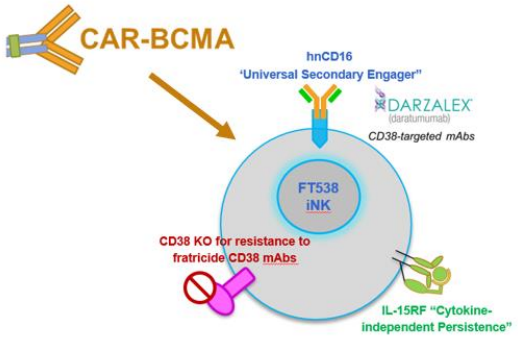
FT576: Multi-antigen Targeted CAR-BCMA NK Cell Product Candidate

BCMA Binding Domain with Differentiated Activation Threshold

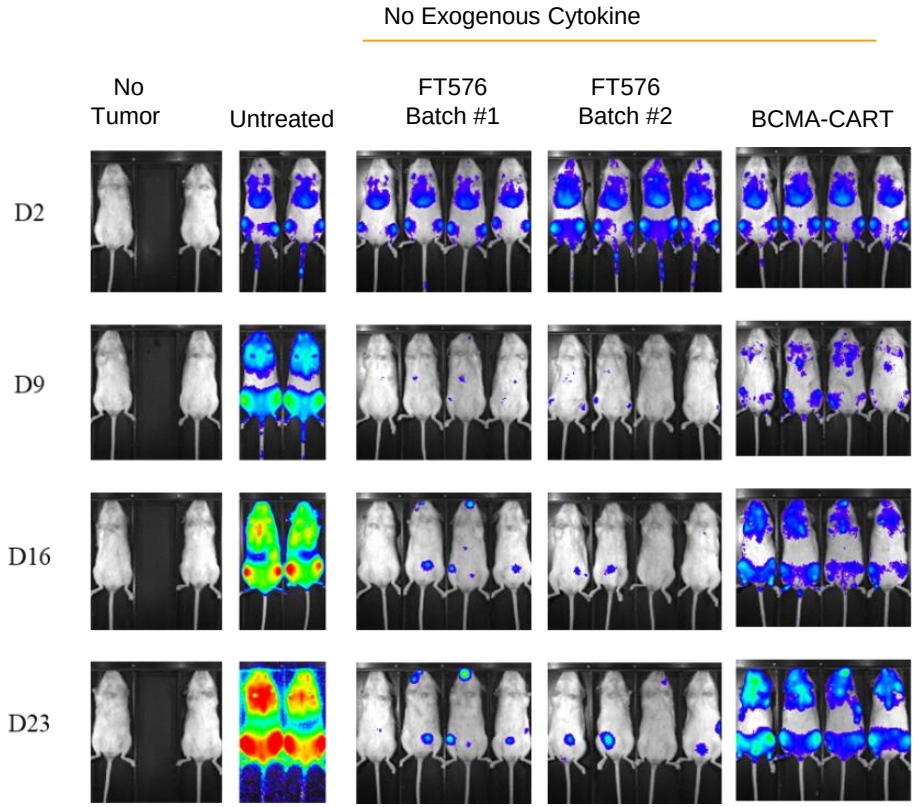
Molecular Therapy
Original Article

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]



- ✓ 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

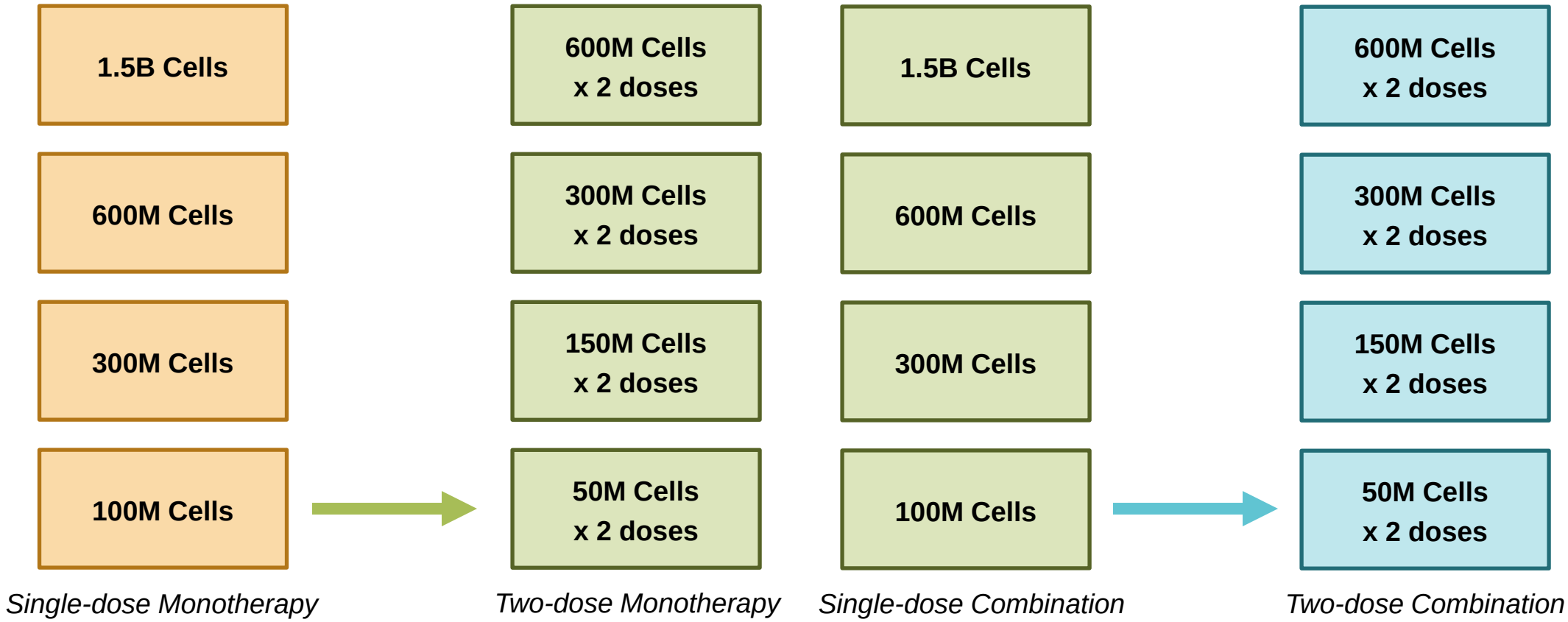


MM.1S-Luc cells

FT576: Phase 1 Dose Escalation Schema

Single- and Multi-dose Schedule as Monotherapy and in Combination with CD38-targeted mAb

Enrolling DL1 (100M Cells) Single-dose Monotherapy



Additional treatment cycles permitted subject to FDA consent

AML Franchise

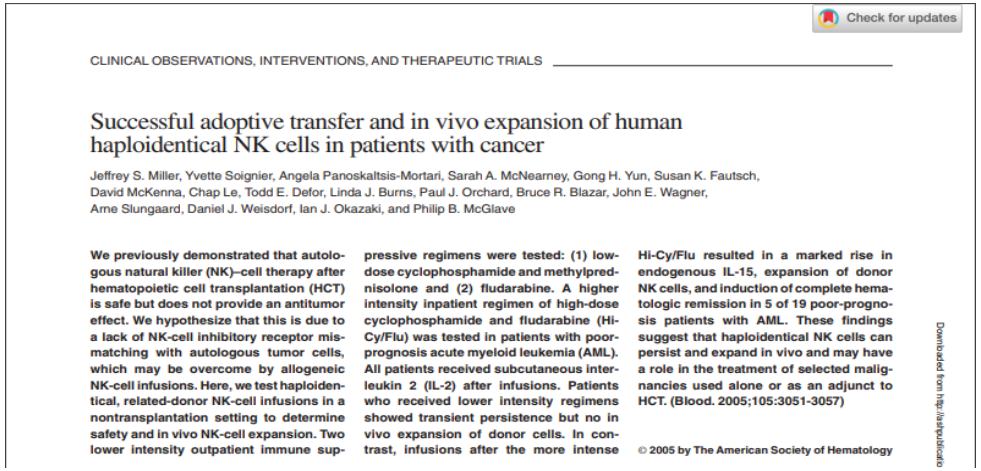
Off-the-Shelf, iPSC-derived NK Cell Franchise for AML

FT516 and FT538 Product Candidates

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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Downloaded from http://ashpublicatio

- 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]

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[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*

FT516 / FT538: Ongoing Phase 1 Studies as Monotherapy

Interim Phase 1 Data in Relapsed / Refractory AML

- Ongoing P1 studies of FT516 and FT538 as monotherapy have enrolled patients with poor prognosis (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

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Interim Phase 1 data includes 9 FT516 patients (3 at 90M cells / dose and 6 and 300M cells / dose) and 3 FT538 patients at 100M cells / dose. All data based on database entry as of April 16, 2021. Data subject to source document verification.

FT538: Ongoing Phase 1 Study in Combination with CD38-targeted mAb

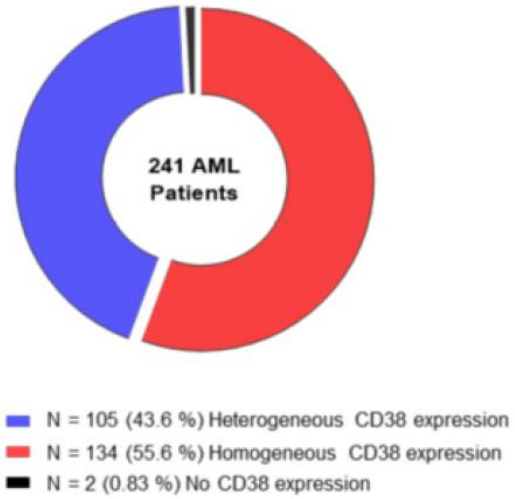
FT538 + daratumumab for Targeting of CD38 on Leukemic Blasts

EUROPEAN HEMATOLOGY ASSOCIATION

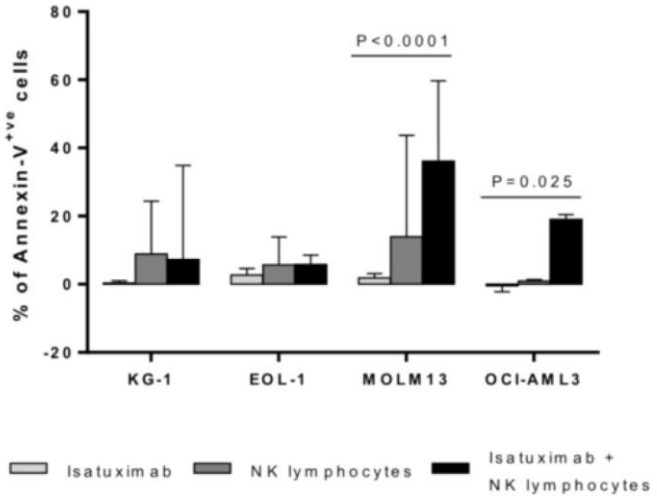
The mode of action of the anti-CD38 monoclonal antibody isatuximab in elderly acute myeloid leukemia

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.

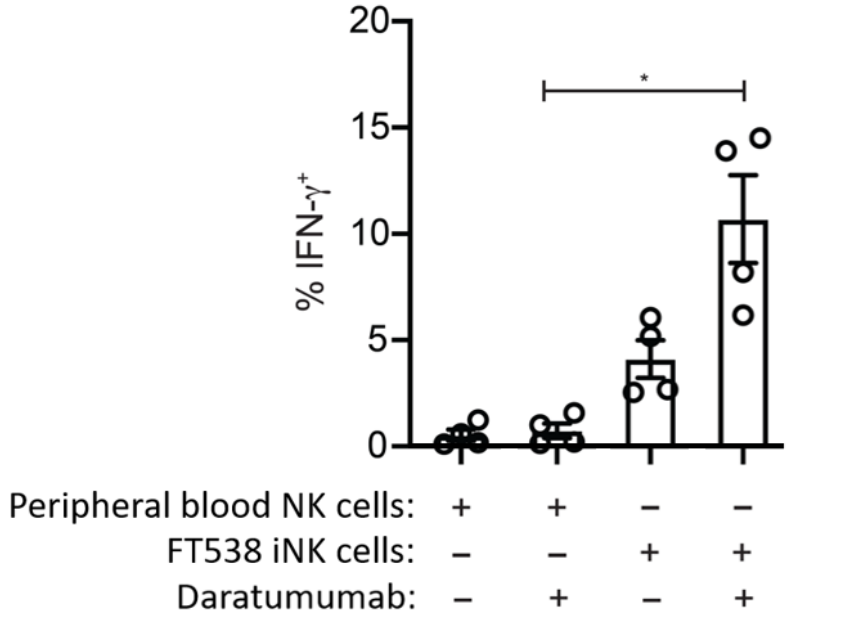
CIMA LAB diagnostics
Universidad de Navarra



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

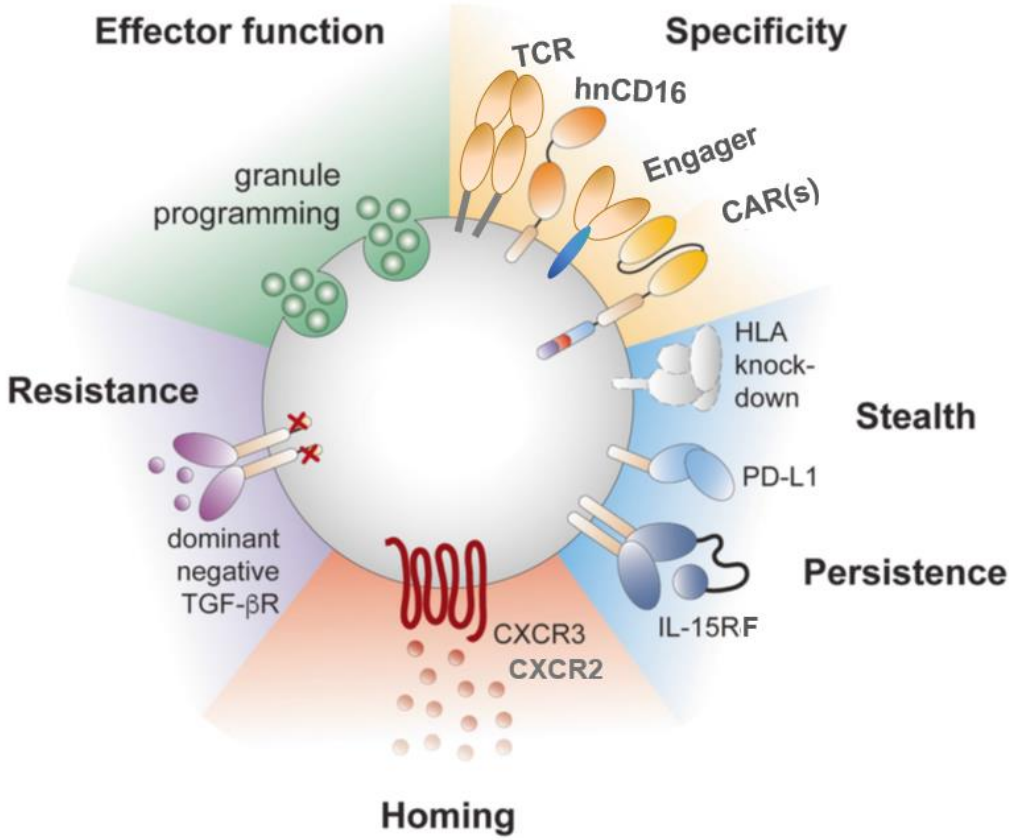
UMN IIT of FT538 + CD38-targeted daratumumab ongoing

Solid Tumor Franchise

Off-the-shelf, iPSC-derived Cell-based Cancer Immunotherapies

Developing Synthetic Killer Cells for Solid Tumors

- Developing next-generation cancer immunotherapies must address numerous challenges that limit the effectiveness of today's agents in treating solid tumors.
  - Depleted / dysfunctional immune cells
  - Immuno-suppressive microenvironment
  - Tumor heterogeneity and escape
- Cell-based cancer immunotherapies have the unique potential to bring rejuvenated immune cells to the fight against cancer.
  - Address deficiencies in patients' endogenous immune system, mount multi-pronged attack, and synergize with complementary agents
- Fate Therapeutics has built a robust pipeline of off-the-shelf, multiplexed-engineered cell therapies for solid tumors.
  - Incorporate synthetic features specifically designed to exploit novel MOAs, synergize with approved agents, and overcome mechanisms of resistance



Modified after Saetersmoen et al. Seminars in Immunopathology 2019

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Emerging Product Pipeline for Solid Tumors

Multiplexed-engineered, iPSC-derived NK and T-cell Product Candidates

Clonal Master iPSC Line	Synthetic Biology	FT500	FT516	FT538	FT536	FT573	JNJ	Ono
Cell Type		NK	NK	NK	NK	NK	NK / T	T
# of Synthetic Elements		0	1	3	4	4	≥4	≥4
+ High-affinity, non-cleavable CD16	*Augment mAb therapy*		✓	✓	✓	✓		
+ IL-15 Receptor Fusion	*Enhance NK cell function*			✓	✓	✓		
+ CAR Insertion	*Target tumors*				MICA/B	B7H3	✓	✓
+ CD38 Knock-out	*Enhance metabolic fitness*			✓	✓	✓		
P1 = Phase 1; SS = Phase 1 study start-up; PC = Preclinical	**Clinical Stage**	P1	P1	P1	SS	PC	PC	PC

Pilot Programs | Multiplexed-engineered Candidates

Orthogonal Mechanisms of Attack for Solid Tumors

Cooperation between Innate and Adaptive Immunity | Augmenting ADCC

Overcoming Tumor Escape | Targeting Metabolic Profile of Cancer

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FT538-102: Multi-arm, Dose-escalating Phase 1 Study

Designed to Assess Cooperation with Adaptive Immune System and Enhanced ADCC

- Multiple cycles each comprising Cy / Flu conditioning, 3 doses of FT538, and mAb therapy
  - 2 cycles; potential to administer additional cycles with clinical benefit and upon relapse
  - FT538 dose ranging from 100M cells / dose to 1.5B cells / dose
  - Each mAb combination enrolls independently

	Pembrolizumab	Avelumab	Trastuzumab	Cetuximab
Target	PD1	PD-L1	HER2	EGFR
Eligibility	Tumors with documented PD-L1 expression		HER2+ tumors: ≥2+ by IHC; ≥4 signals/cell by ISH	EGFR+ tumors, incl. KRAS/NRAS and driver mutations
Primary Cancers	NSCLC, GE, HNSCC, TNBC, UC		Gastric, Breast	NSCLC, CRC, HNSCC

CRC = colorectal cancer; GE = gastroesophageal; HNSCC = head and neck squamous cell carcinoma; IHC = immunohistochemistry; ISH = in situ hybridization; NSCLC = non-small cell lung carcinoma; TNBC = triple-negative breast cancer; UC = urothelial carcinoma

First Patients Enrolled in Dose-escalating Phase 1 Study

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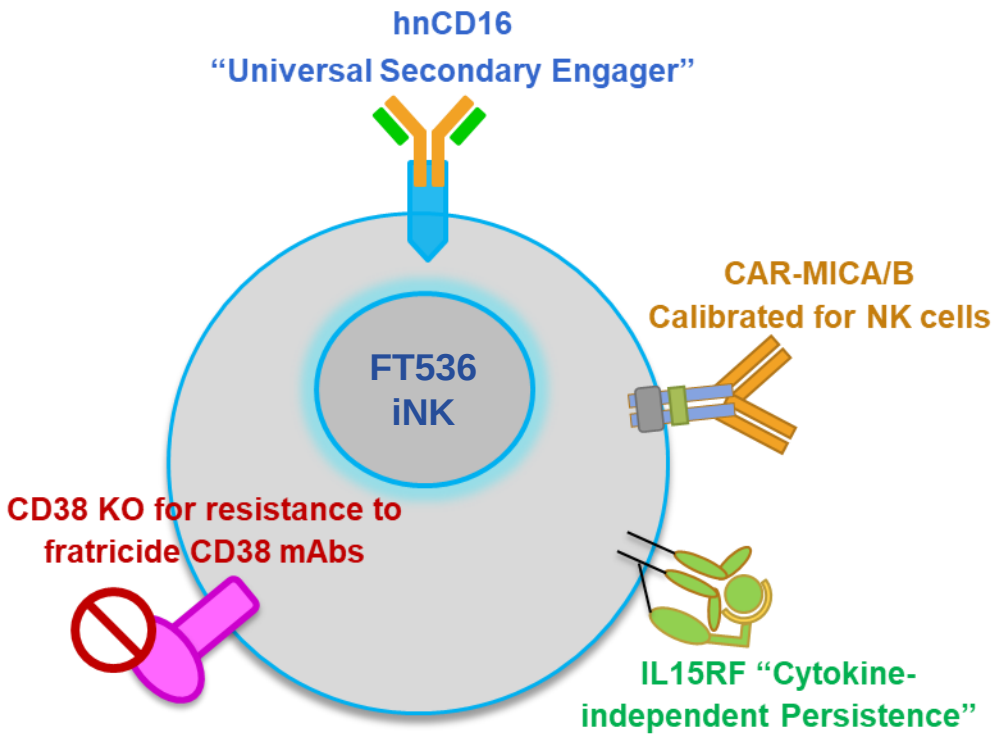
FT536: Multi-antigen Targeted CAR-MICA/B NK Cell Product Candidate

Pan-tumor Targeting Strategy to Overcome Tumor Escape

Engineered with Four Anti-tumor Modalities for Solid Tumors



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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CAR-MICA/B: 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, the conserved α3 domain of MICA/B

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

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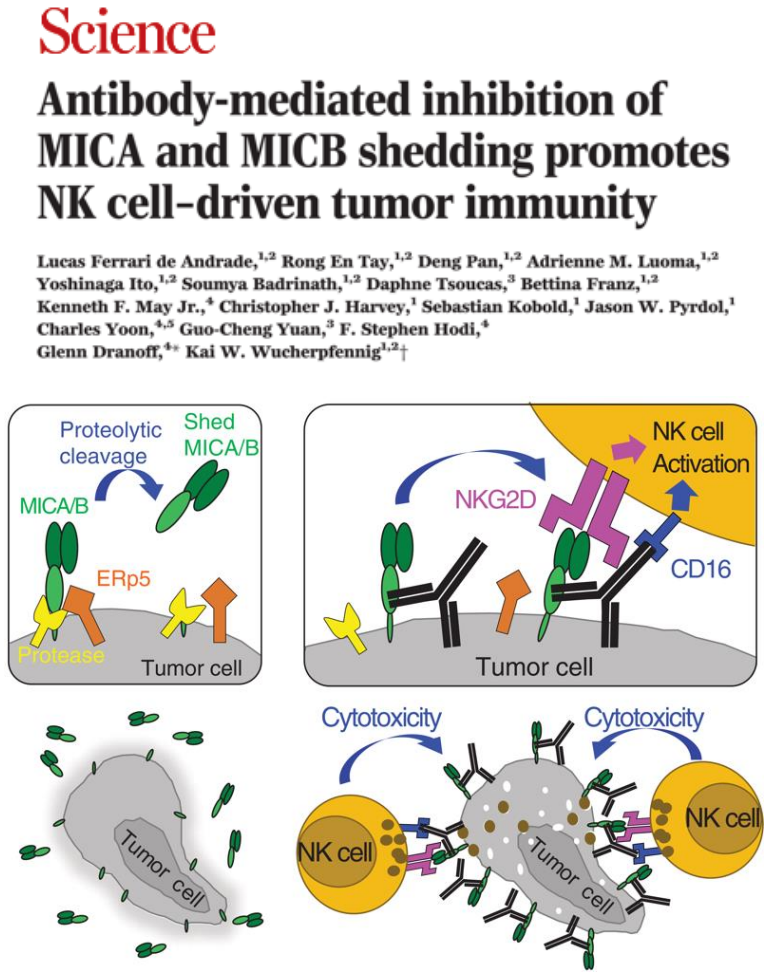
CD38 KO: Deletion of CD38 to eliminate anti-CD38 antibody mediated NK cell fratricide. Also shown to improve metabolic fitness and induce transcriptional program that drives NK cell activation and effector function

IND Allowed and Phase 1 Start-up Ongoing to Assess Monotherapy and Combination with Checkpoint Inhibitor Therapy and with Tumor-targeting mAbs

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

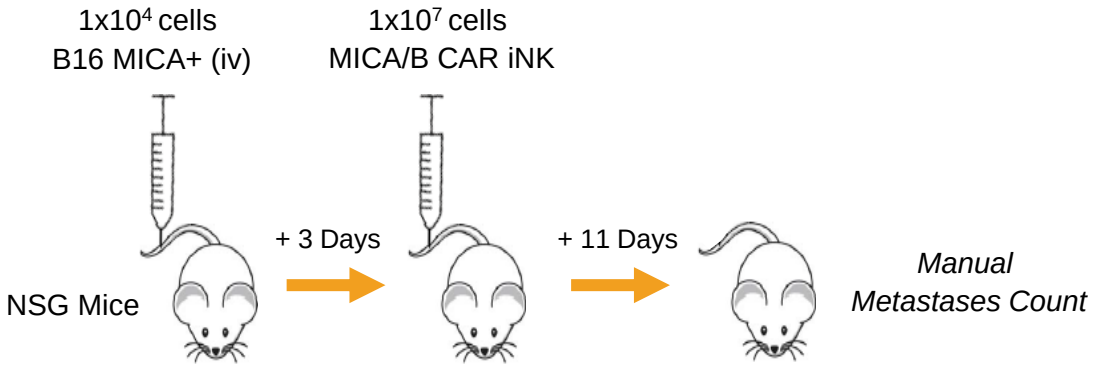
Pan-tumor Targeting Strategy to Overcome Tumor Escape

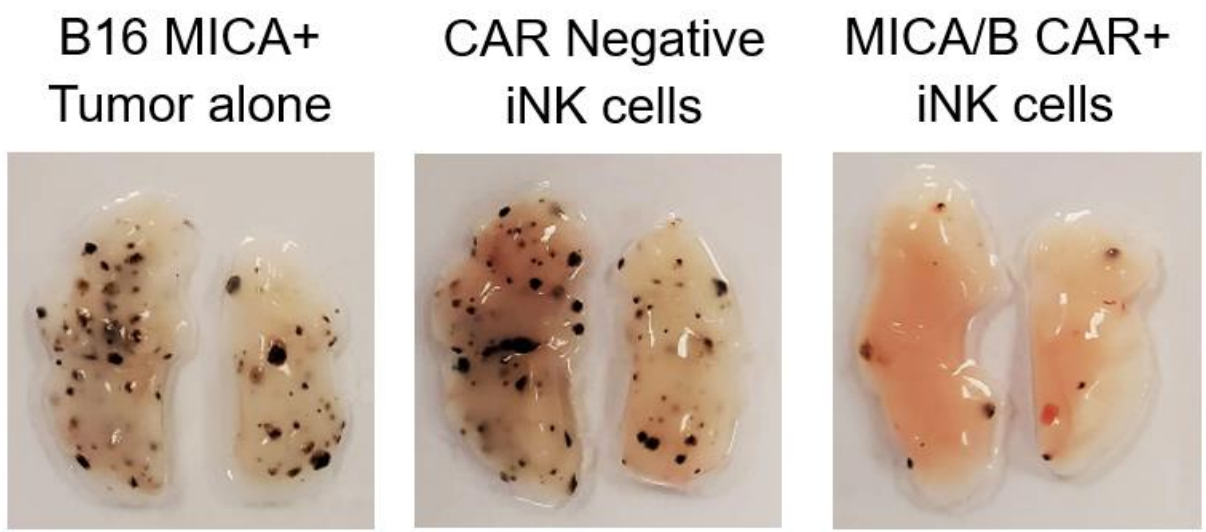
- MICA/B are cell-surface proteins induced by cellular stress and transformation, and their expression has been reported for many cancer types.
- NKG2D, an activating receptor expressed on NK and T cells, targets the membrane-distal α1 and α2 domains of MICA/B, activating a potent cytotoxic response.
- 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 cytolytic activity.
- Soluble MICA/B have been associated with poor clinical prognosis in cancer patients.
- Overcoming MICA/B shedding to effectively re-engage tumor cells is emerging as a novel pan-tumor targeting mechanism.
- Preclinical data have shown that therapeutic antibodies targeting the membrane-proximal α3 domain inhibit MICA/B shedding, and result in a substantial increase in the cell-surface density of MICA/B and restoration of immune cell-mediated tumor immunity.

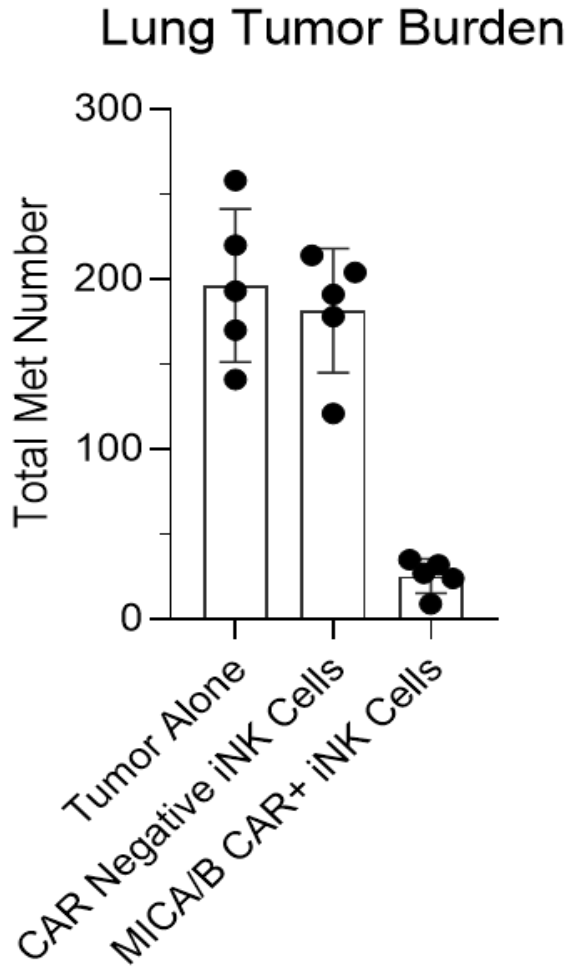


FT536: Multi-antigen Targeted CAR-MICA/B NK Cell Product Candidate

Durable Tumor Reduction of B16 MICA+ Metastatic Lung Lesions



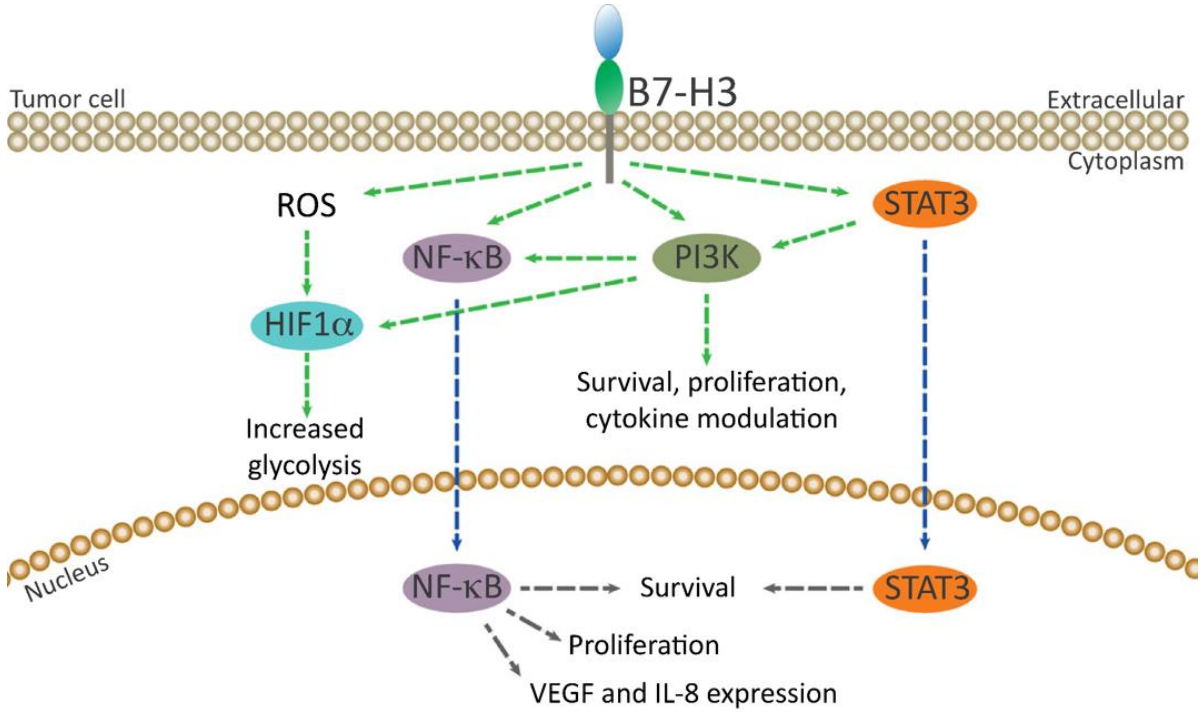




FT573: Multi-antigen Targeted CAR-B7H3 NK Cell Product Candidate

Pan-tumor Targeting Approach Aimed at the Metabolic Profile of Cancer

- B7H3 (CD276) belongs to the B7 superfamily of immune checkpoint molecules.
- B7H3 protein is aberrantly overexpressed in a wide variety of cancers, with limited expression at low level in normal tissues, and is often associated with poor prognosis.
- Recent studies have shown that B7H3 is a critical promoter of tumorigenesis and metastasis, and its expression is a metabolic hallmark of cancer.
- Multiple modalities targeting B7H3 have shown early clinical activity in patients with advanced solid tumors.



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

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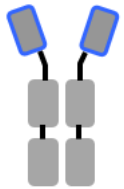
FT573: Multi-antigen Targeted CAR-B7H3 NK Cell Product Candidate

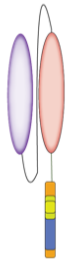
Identification of Novel anti-camB7H3 scFv

CAR Design

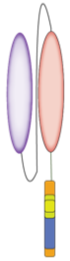
- Based on camelid antibodies
- Maintain high target affinity and specificity associated with conventional antibodies
- Demonstrate good physiochemical stability, reduced immunogenicity, and preferred agility associated with their reduced size
- Generated single-domain targeting sequence (V_HH)
- Created CAR motifs for each of NK cells and T cells



V_HH anti-cam B7H3 scFv

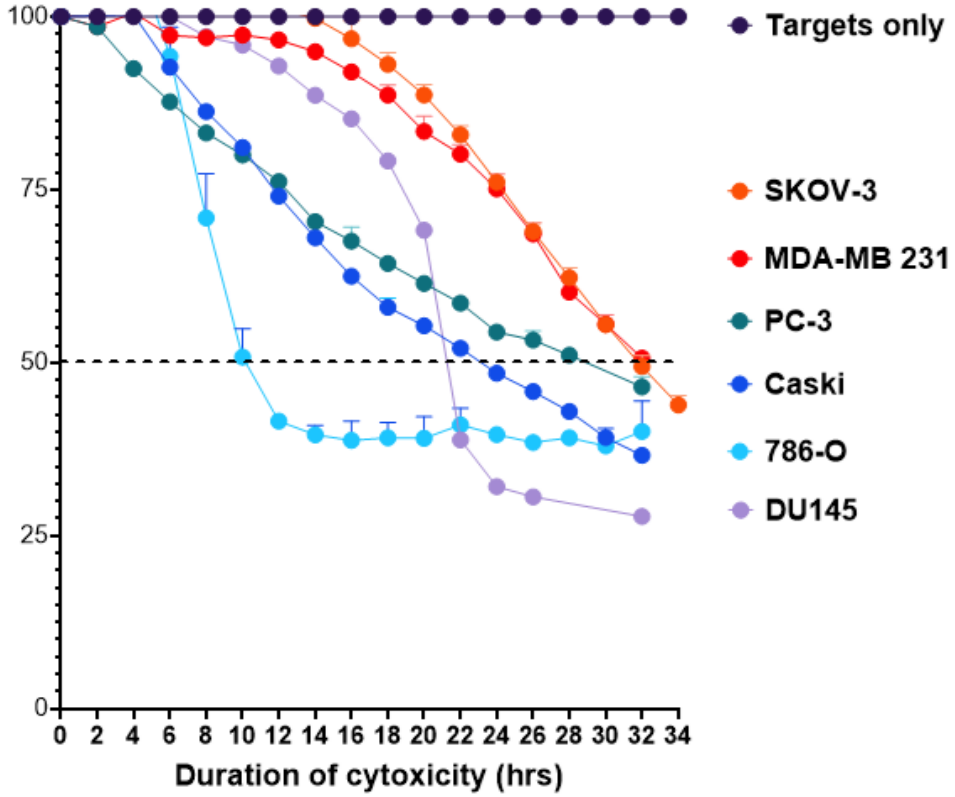


NK Cell CAR Construct



T Cell CAR Construct

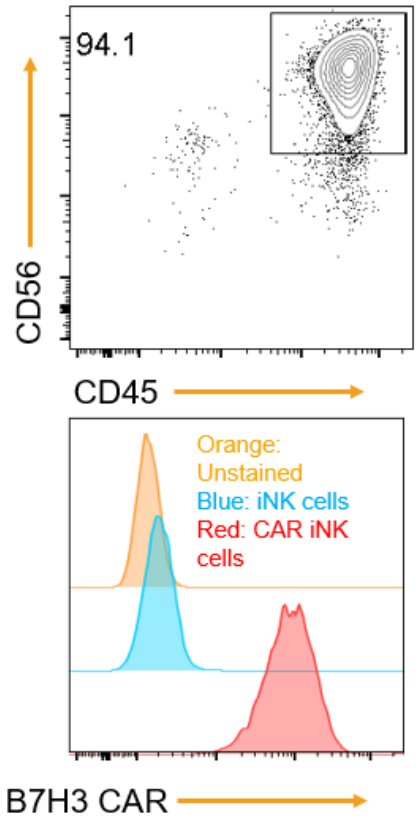
Robust Recognition and Activity Against Multiple Solid Tumor Lines



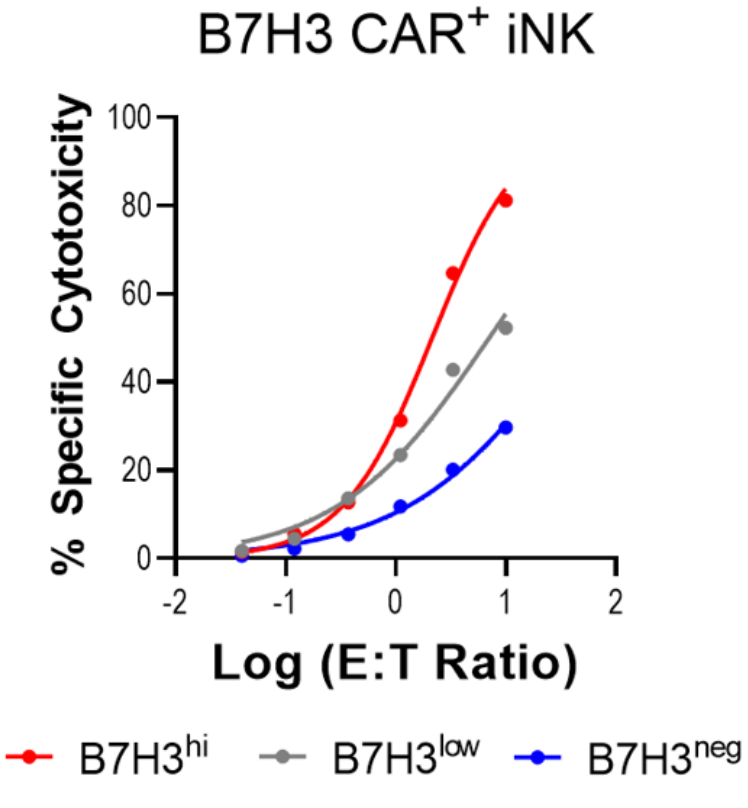
FT573: Multi-antigen Targeted CAR-B7H3 NK Cell Product Candidate

Pan-tumor Targeting Approach Aimed at the Metabolic Profile of Cancer

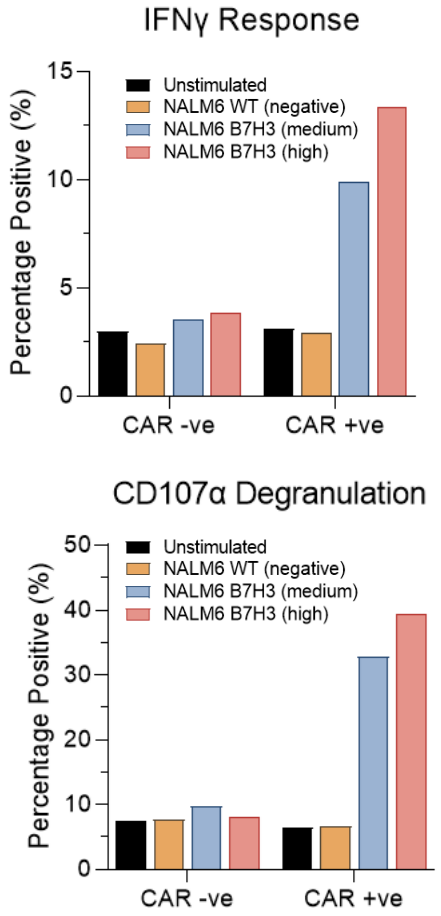
Uniform Expression of B7H3 CAR



Antigen-specific Cytotoxicity



Cytokine Release & Degranulation



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Collaborations & Financials

Janssen Cancer Immunotherapy Collaboration (April 2020)

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

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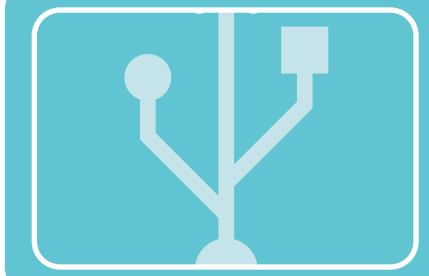
janssen PHARMACEUTICAL COMPANIES OF Johnson & Johnson

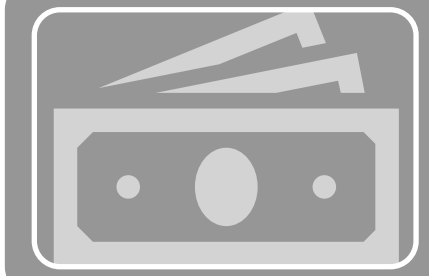


Oncology Innovation

- 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

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ONO Cancer Immunotherapy Collaboration (September 2018)

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

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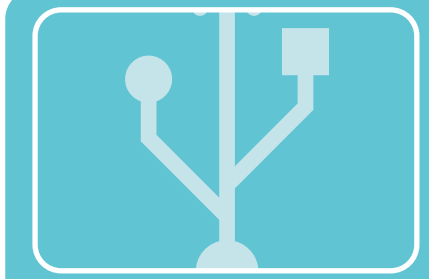
ONO PHARMACEUTICAL CO.,LTD.

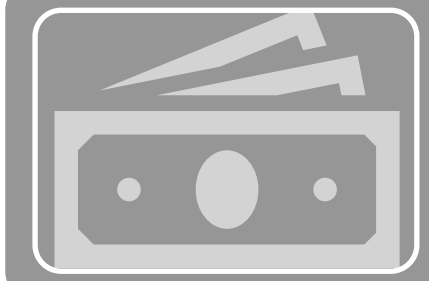


Oncology Innovation

- 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

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Financial Summary

As reported in Company's Consolidated Financial Statements

Three Months Ended September 30, 2021	
Revenue	$14.2M
Operating Expense[1]	$68.9M
Cash & Cash Equivalents	$804M
Employees	~400
Total Shares Outstanding[2]	109.4M

[1] *Includes $13.5m in stock-based compensation*

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

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Better Cells For Better Therapies™