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

November 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 and announcement of data from its clinical trials and preclinical studies, the Company's clinical development and regulatory strategy, and the Company's expectations regarding progress and timelines, and potential payments under its collaborations, and the objectives, plans and goals of its collaborations. 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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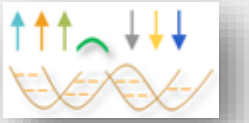
Fate Therapeutics

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



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

Deep Product Pipeline: robust pipeline of multiplexed-engineered NK and T-cell programs addressing unmet medical needs in hematologic malignancies and solid tumors

Demonstrated Clinical Benefit: treated 200+ patients with off-the-shelf, multi-dose treatment paradigm showing substantial therapeutic benefit

Scalable Manufacture: in-house GMP operations with demonstrated ability to mass produce 100s of cryopreserved doses of uniform cell product in single manufacturing campaign

World Class Partnerships: co-developing novel iPSC-derived CAR NK and CAR T-cell product candidates with Ono and Janssen for hematologic malignancies and solid tumors

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Changing the Game in Cell Therapy

Transforming the Cell Therapy Field with a Drug-like Cell Product Paradigm

Multiplexed Engineering

Multiple mechanisms of attack against cancer incorporated into cell product

Mass Production

Scalable GMP operations yielding 100s of doses in single campaign

Uniform Products

Batch-to-batch consistency of cell product features and functionality

Off-the-Shelf

Cryopreserved with long-term stability for storage and on-demand availability

Drug-like Treatment

Multi-dose schedules administered in the outpatient setting

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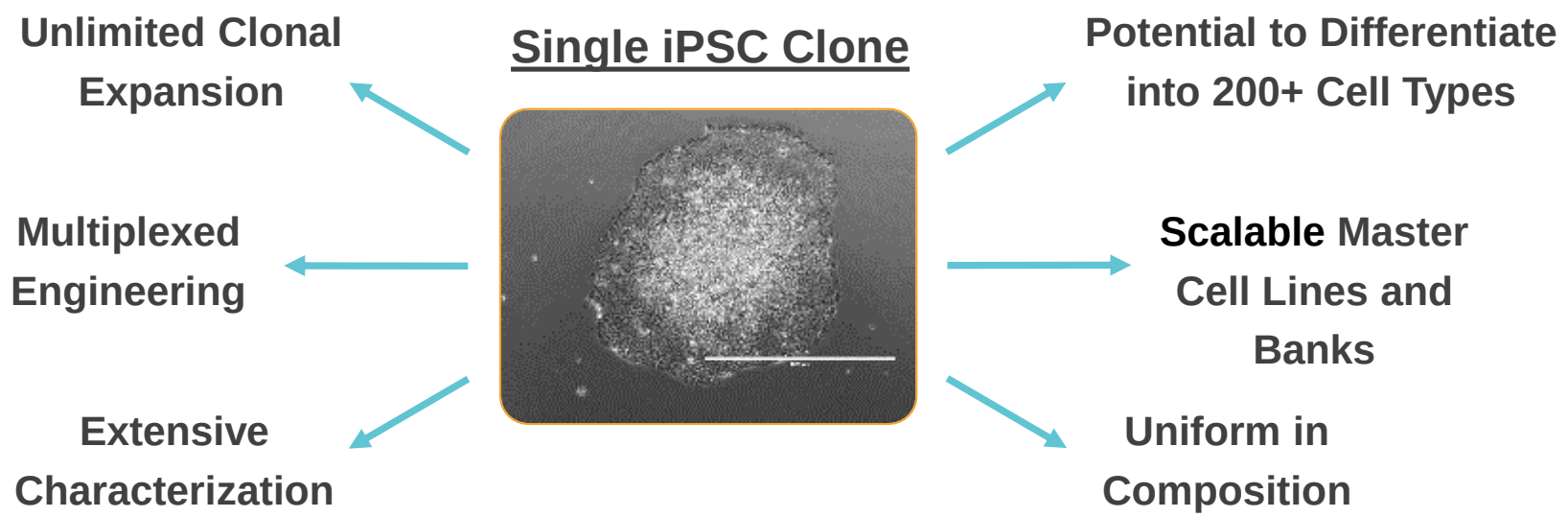
Disruptive iPSC Product Platform

Creating Multiplexed-engineered iPSC-Derived Cell Products

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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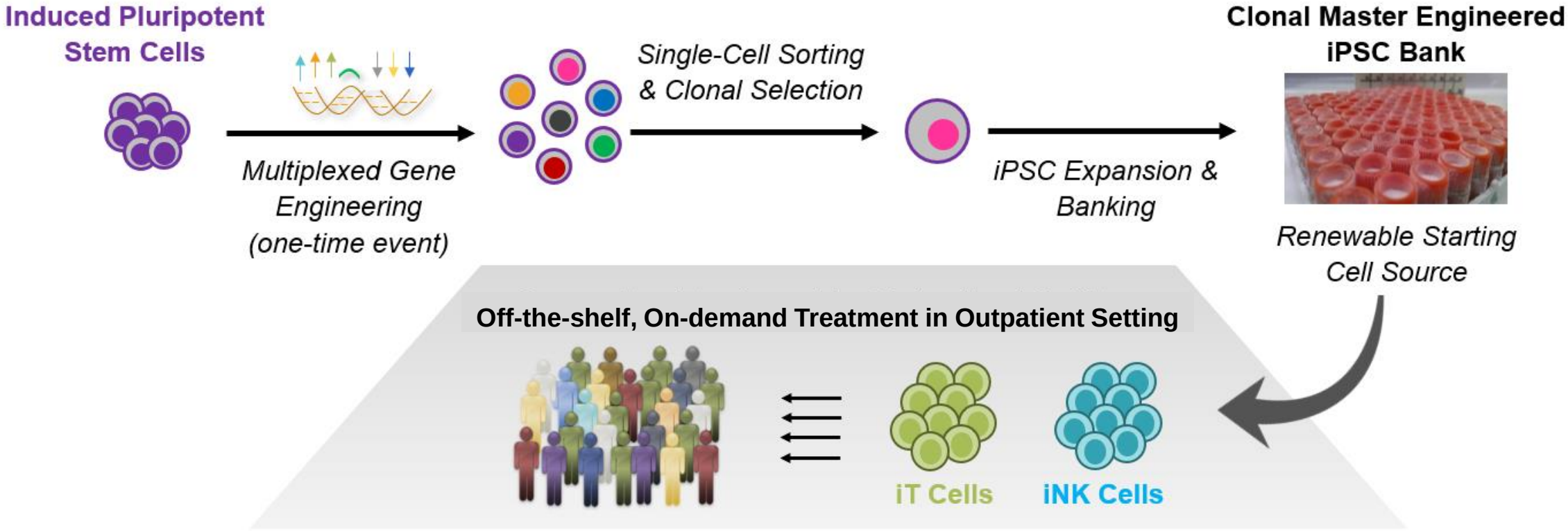
Disruptive iPSC Product Platform

Mass Production of Multiplexed-engineered Cell Products for Off-the-shelf Patient Treatment



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

Designed with multiple tumor-fighting mechanisms

High quality; consistent purity and activity

High yield; scalable for efficient manufacturing

On-demand; expanded patient reach

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Disruptive iPSC Product Platform

Novel Functional Armament Deployed in Attack Against Cancer

Direct Antigen Targeting

CARs directed to CD19, BCMA, MICA/B, B7H3

Combinations with mAbs / Engagers

hnCD16 and Synthetic CD3 receptors to synergize with mAbs and NK cell / T-cell engagers

Immunosuppressive Resistance

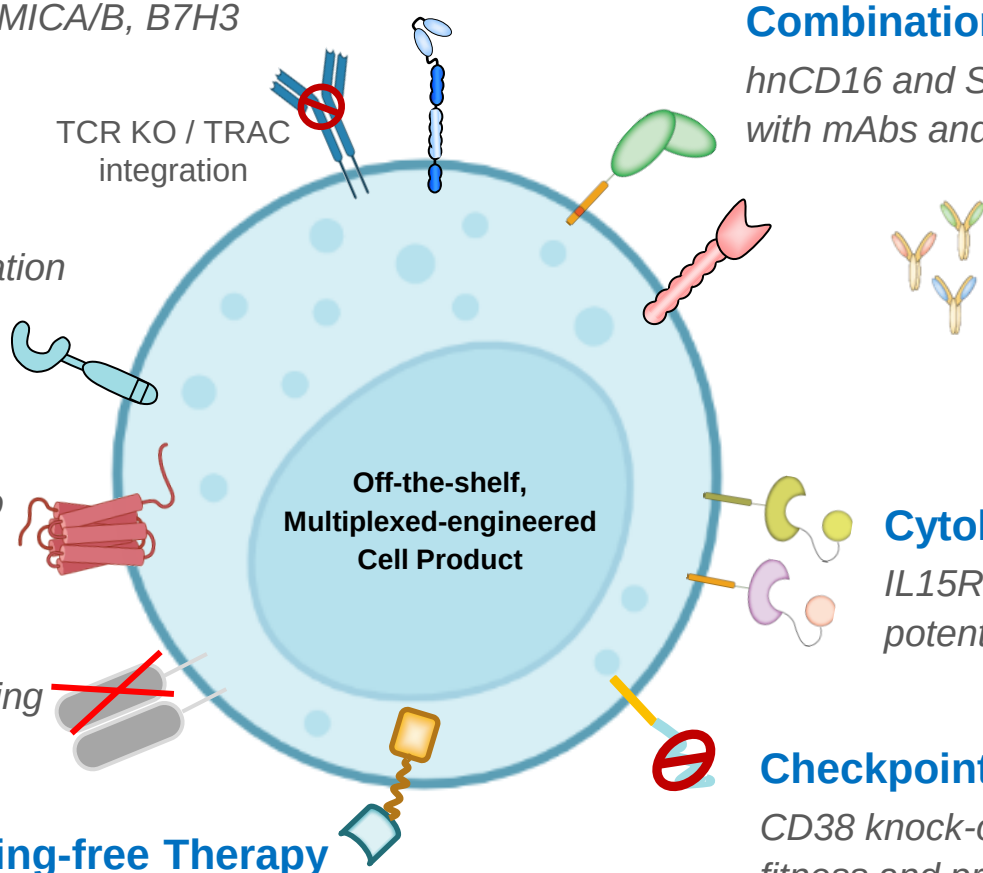
Synthetic TGFB redirector to promote activation in response to immuno-suppressive TME

Cell Homing

Synthetic CXCR2 receptor to promote cell trafficking

Stealth

Ligand/receptor engineering to prevent rejection



Cytokine Support

IL15RF and IL7RF for cell potentiation

Checkpoint KO

CD38 knock-out to promote metabolic fitness and prevent fratricide

Conditioning-free Therapy

Allo-defense receptor (ADR) to redirect host immune cell alloreactivity and promotes activation

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A Transformative Cell Product Approach for the Treatment of Cancer

Unique Advantages of Off-the-shelf, Multiplexed-engineered, iPSC-derived Cell Products

Flexible Administration	• On-demand treatment • Reliable and convenient without the need for hospitalization • Lower administrative burden
Combination Therapies	• Synergize with other anti-cancer agents • Activate endogenous immune system • Induce multiple complementary mechanisms of action
Earlier Intervention	• Off-the-shelf availability • Treatment paradigm enables add-on to early-line SOC regimens • Reach into the community setting with mass produced cell product

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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*

Hematologic Malignancies

Hematologic Malignancy Product Pipeline

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

Program	Functionality & *Cell Type*	Target(s)	Indication(s)	Preclin	Phase 1
FT516	hnCD16 *iNK*	CD20	BCM + mAb		
		n/a	AML		
FT596	hnCD16 + IL15RF + CAR-CD19 *iNK*	CD19 ± CD20	BCM ± mAb		
FT819	CAR-CD19 *iT*	CD19	BCM		
FT538	hnCD16 + IL15RF + CD38-KO *iNK*	CD38	MM + mAb		
		n/a	AML		
FT576	hnCD16 + IL15RF + CD38-KO + CAR-BCMA *iNK*	BCMA ± CD38	MM ± mAb		
FT555/ Janssen	hnCD16 + IL15RF + CD38-KO + CAR-GPRC5D *iNK*	GPRC5D ± CD38	MM ± mAb		
Janssen	*iNK, iT*	Undisclosed	Undisclosed		

iPSC *= induced pluripotent stem cell* ***iNK*** *= iPSC-derived NK Cell* ***iT*** *= iPSC-derived T cell* ***mAb*** *= monoclonal antibody*
BCM *= B-cell malignancies* ***AML*** *= Acute Myeloid Leukemia* ***MM*** *= Multiple Myeloma*
hnCD16 *= high affinity, non-cleavable CD16 Fc receptor* ***IL15-RF*** *= IL15 receptor fusion* ***CD38-KO*** *= CD38 knock-out* ***CAR*** *= chimeric antigen receptor*

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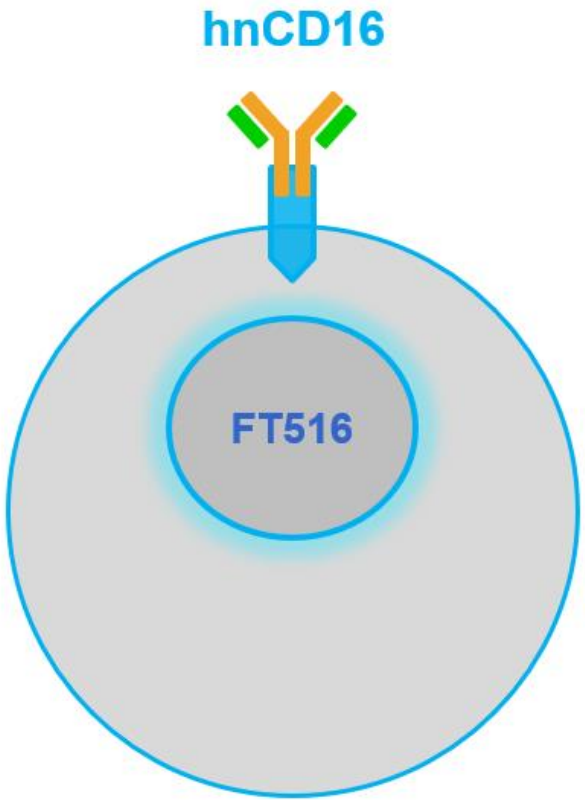
Non-Hodgkin Lymphoma

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

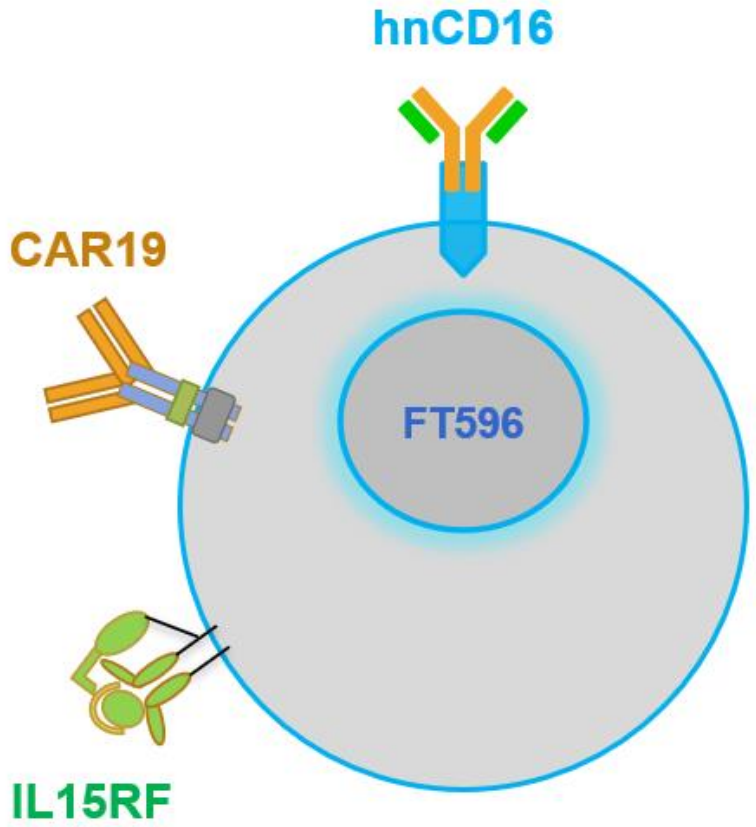
FT516 and FT596 Product Candidates



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



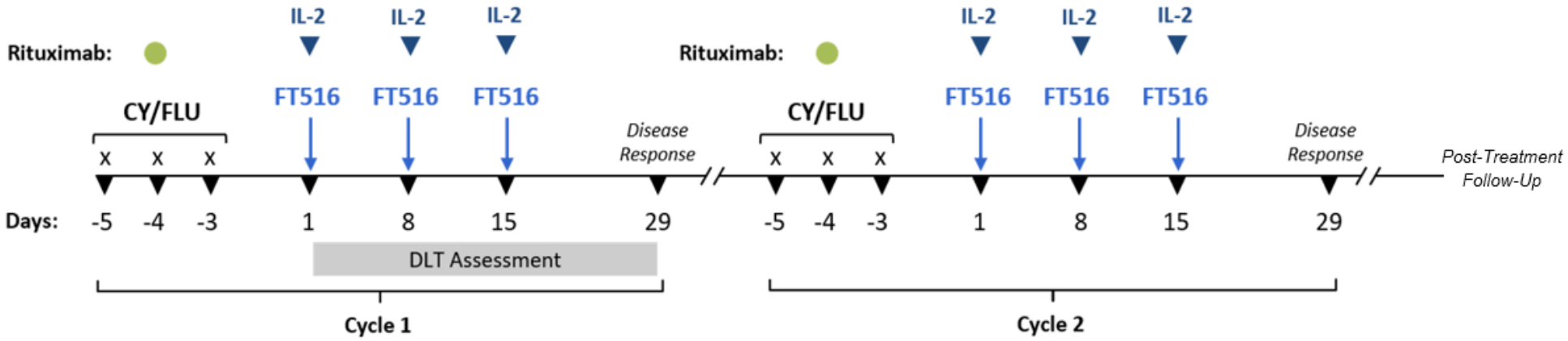
FDA RMAT Designation

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

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

Three-dose Treatment Schedule; Up to 2 Cycles

- 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 three-dose treatment schedule
  - Up to 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 once-weekly doses of FT516 with IL-2 cytokine support
  - No mandatory hospitalization required during the treatment period



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

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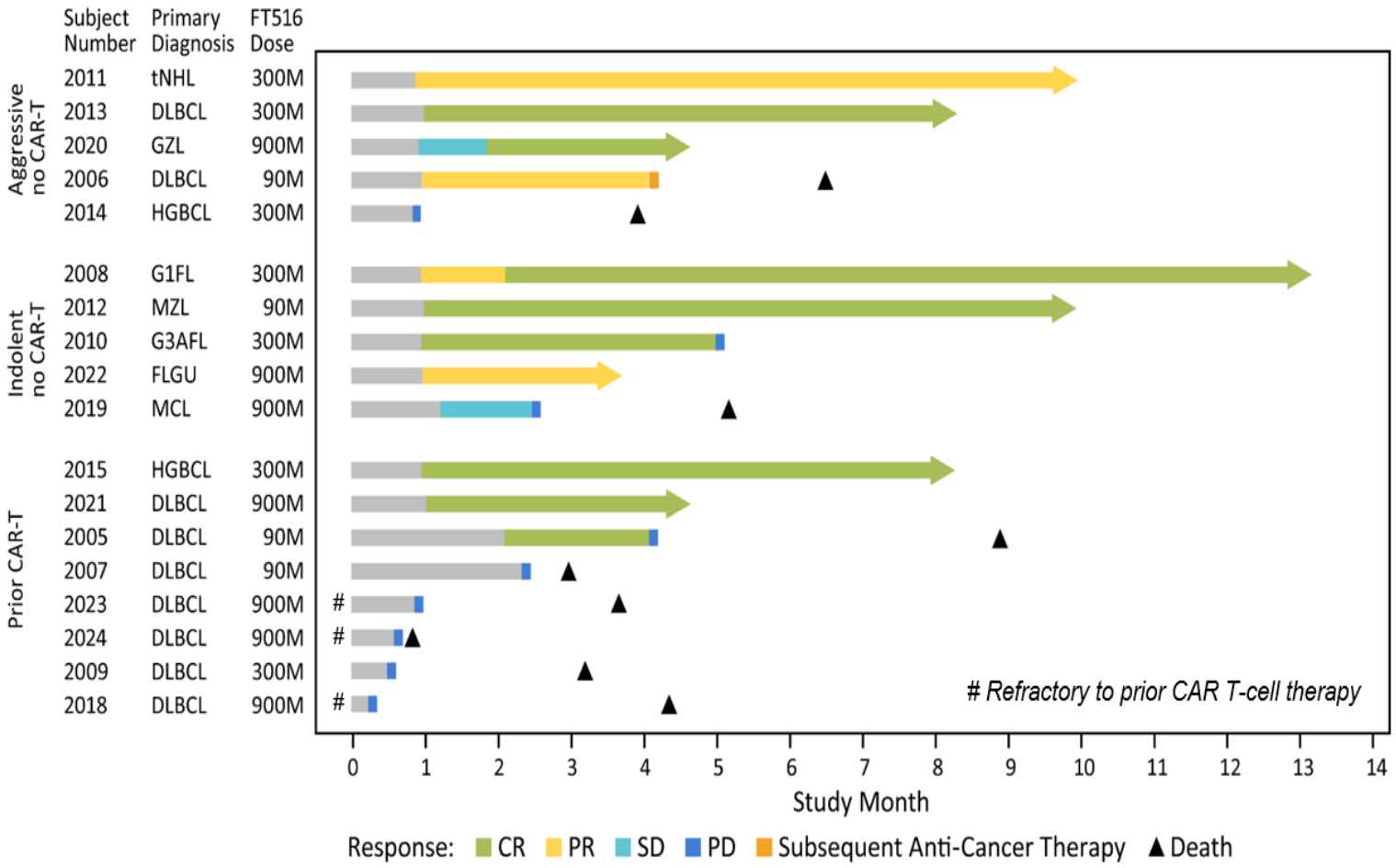
FT516-101: Phase 1 Study in R/R B-cell Lymphoma

Interim Phase 1 Clinical Data



63rd ASH Annual Meeting and Exposition
DECEMBER 11-14, 2021 • ATLANTA, GA • IN-PERSON/VIRTUAL

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

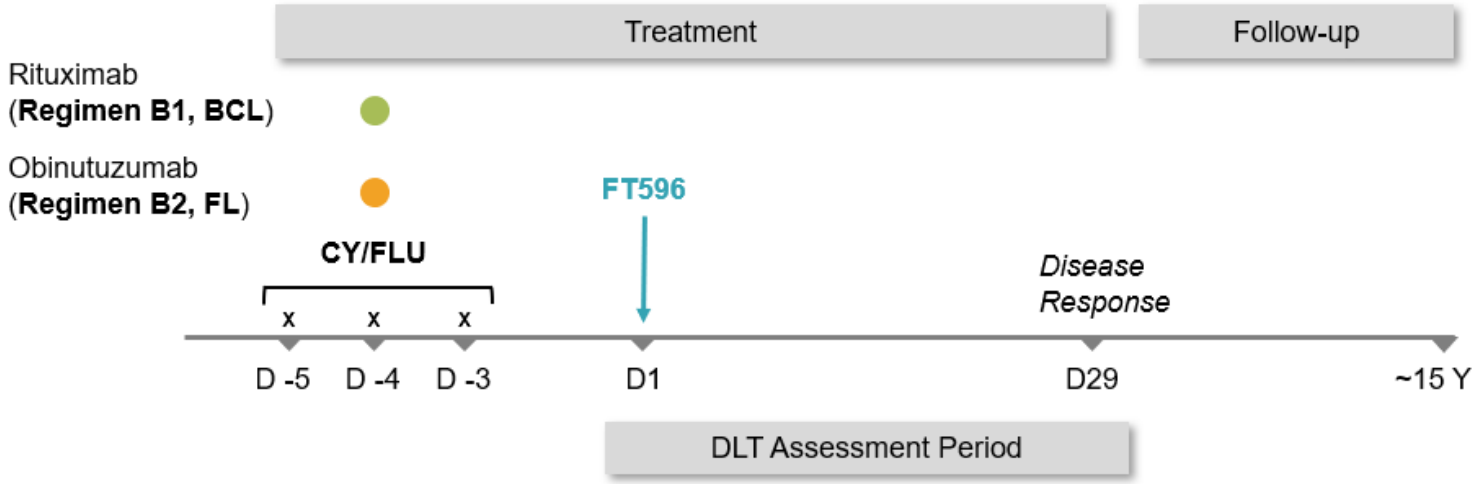
As of the data cutoff date (18 Oct 2021); Response assessment per Lugano 2014 criteria. Data subject to source document verification.

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

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

Single-dose Treatment Schedule; Up to 2 Cycles

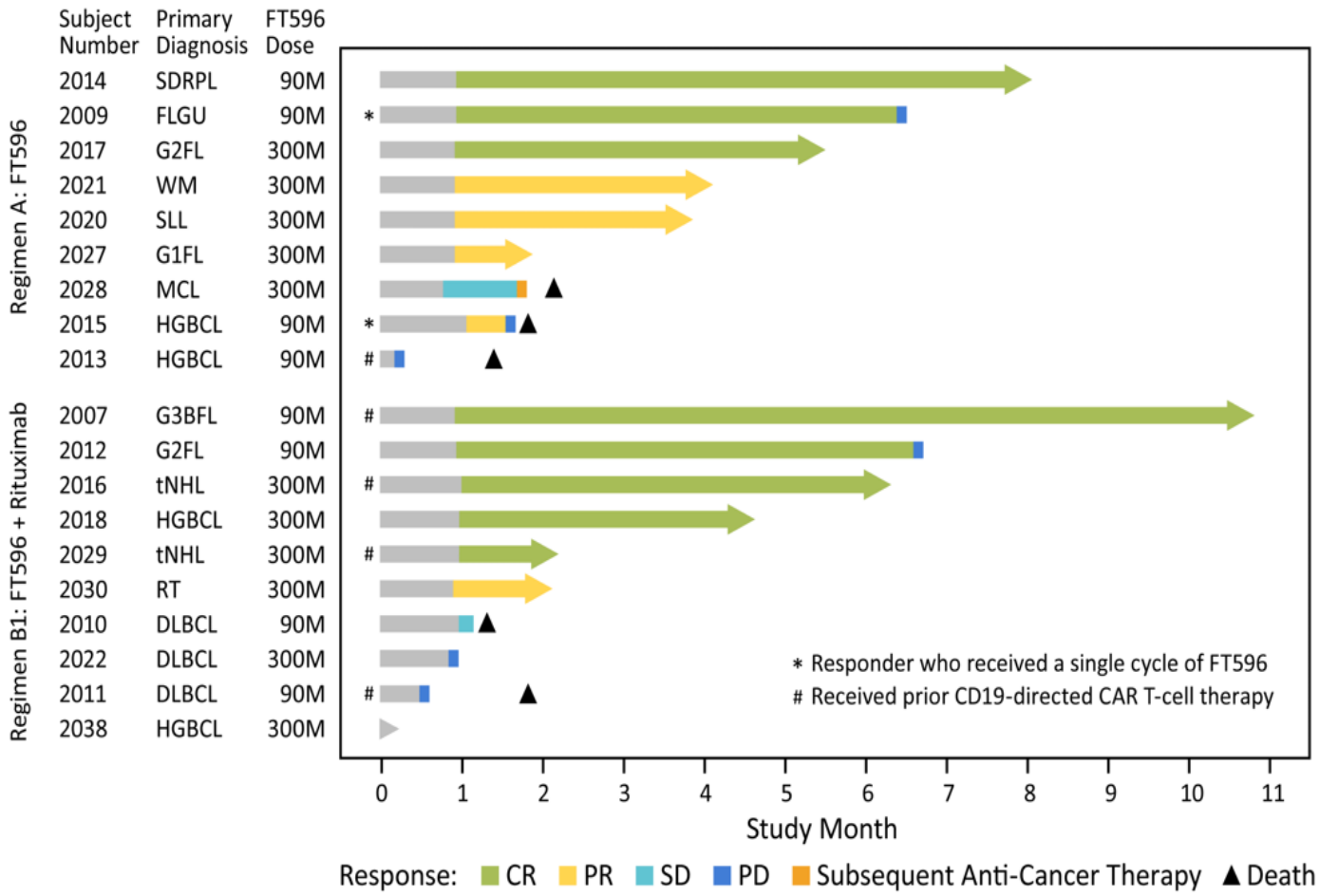
- First-in-human study assessing the safety and activity of FT596 as monotherapy and in combination with rituximab in patients with r/r B-cell lymphoma
  - Primary objective: identify DLT and determine maximum tolerated dose / dose schedule
  - Additional objectives: safety, tolerability, preliminary activity, pharmacokinetics, and immunogenicity
- Single-dose treatment schedule
  - Up to 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 a single dose of FT596 without cytokine support
  - No mandatory hospitalization required during the treatment period



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

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

Interim Phase 1 Clinical Data



63rd ASH Annual Meeting and Exposition
DECEMBER 11-14, 2021 • ATLANTA, GA • IN-PERSON/VIRTUAL

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

FT516 and FT596 NK Cell Programs for B-cell Malignancies

Ongoing Development Initiatives

FT516 Program

- Ongoing FDA interactions under RMAT Designation covering iPSC-derived product platform and late-stage clinical development pathways, including pivotal launch requirements and study design in patients that have progressed or failed CD19-targeted CAR T-cell therapy
- Ongoing P1 dose expansion at 900M cells per dose across multiple cohorts, including 3L+ aggressive lymphoma, 3L+ indolent lymphoma, and post CD19-targeted CAR T-cell therapy
- Ongoing P1 assessment of FT516 safety and activity following R-Benda administration (and without Cy / Flu chemotherapy conditioning)

FT596 Program

- Ongoing P1 dose expansion with single-dose treatment schedule at 900M cells in multiple disease-specific cohorts
- Ongoing P1 dose escalation with 2-dose treatment schedules at 900M cells / dose and at 1.8B cells / dose; 3-dose treatment schedule to be initiated subject to DLT clearance
- Opening clinical study assessing FT596 in 1L community setting *without Cy / Flu chemotherapy conditioning* as an add-on to R-CHOP SOC regimen, with first patient expected to be treated in 2H22

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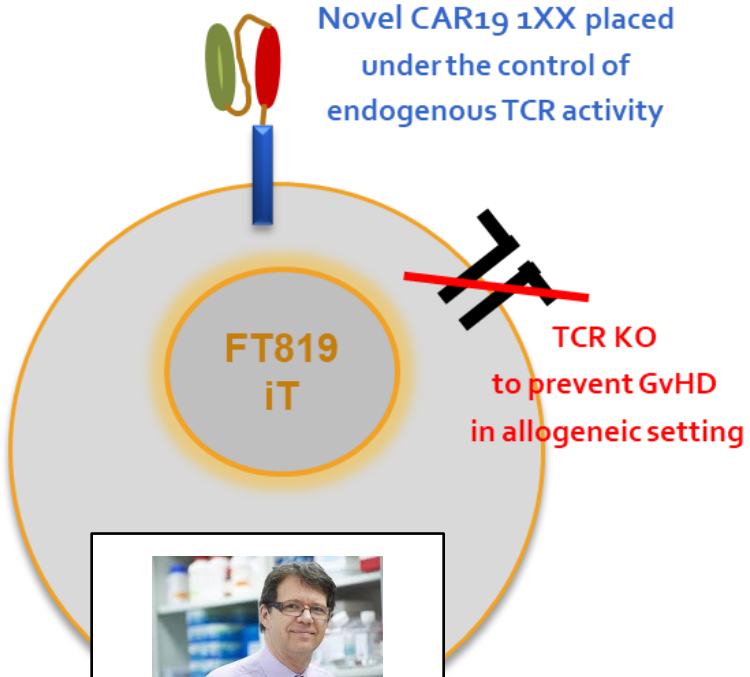
FT819: Off-the-Shelf CAR19 T-Cell Product Candidate

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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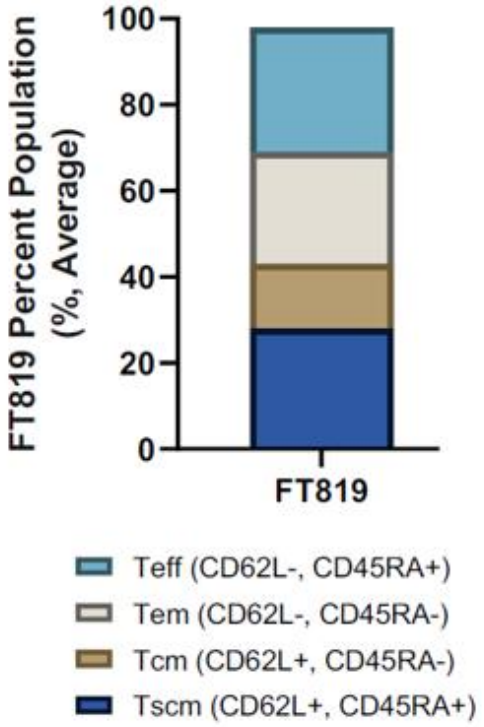
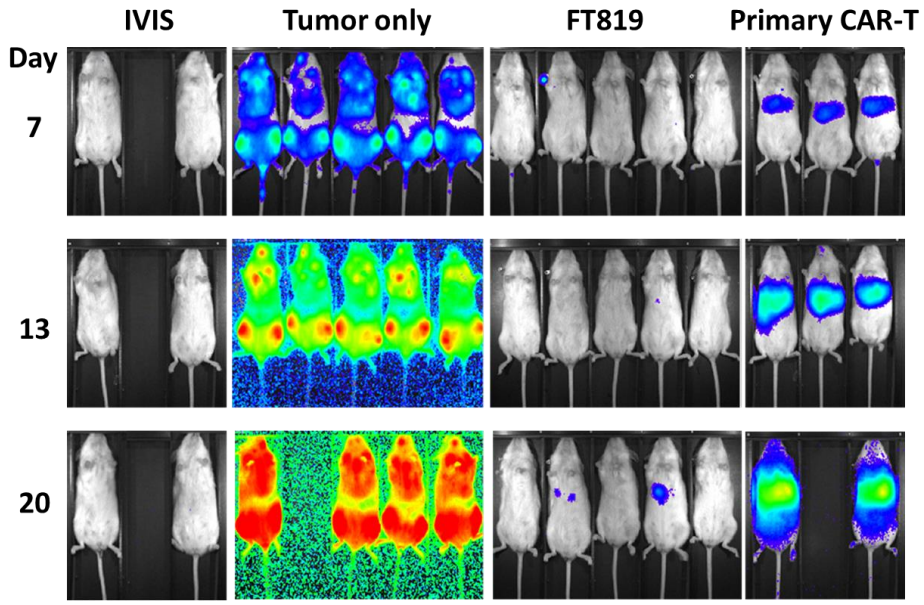
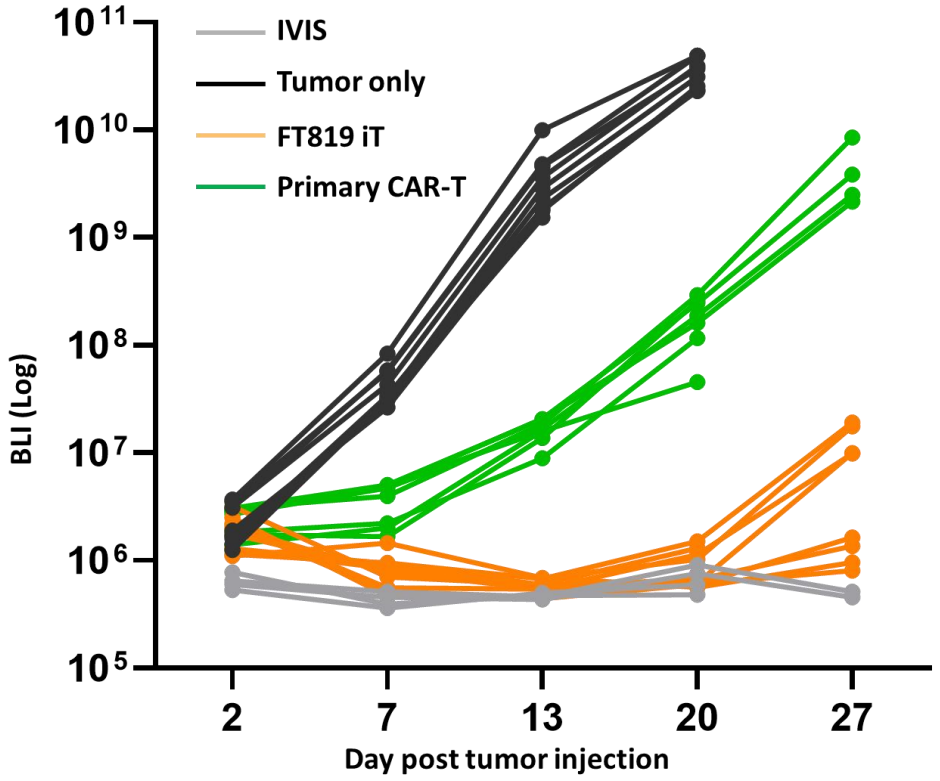
FT819: Enhanced Tumor Control vs. Primary CAR T Cells

Disseminated Xenograft Model of Lymphoblastic Leukemia

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







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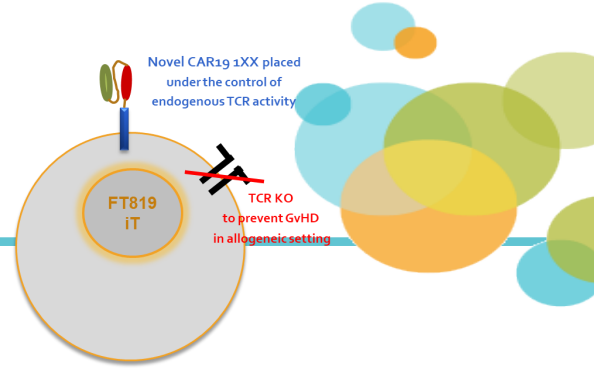
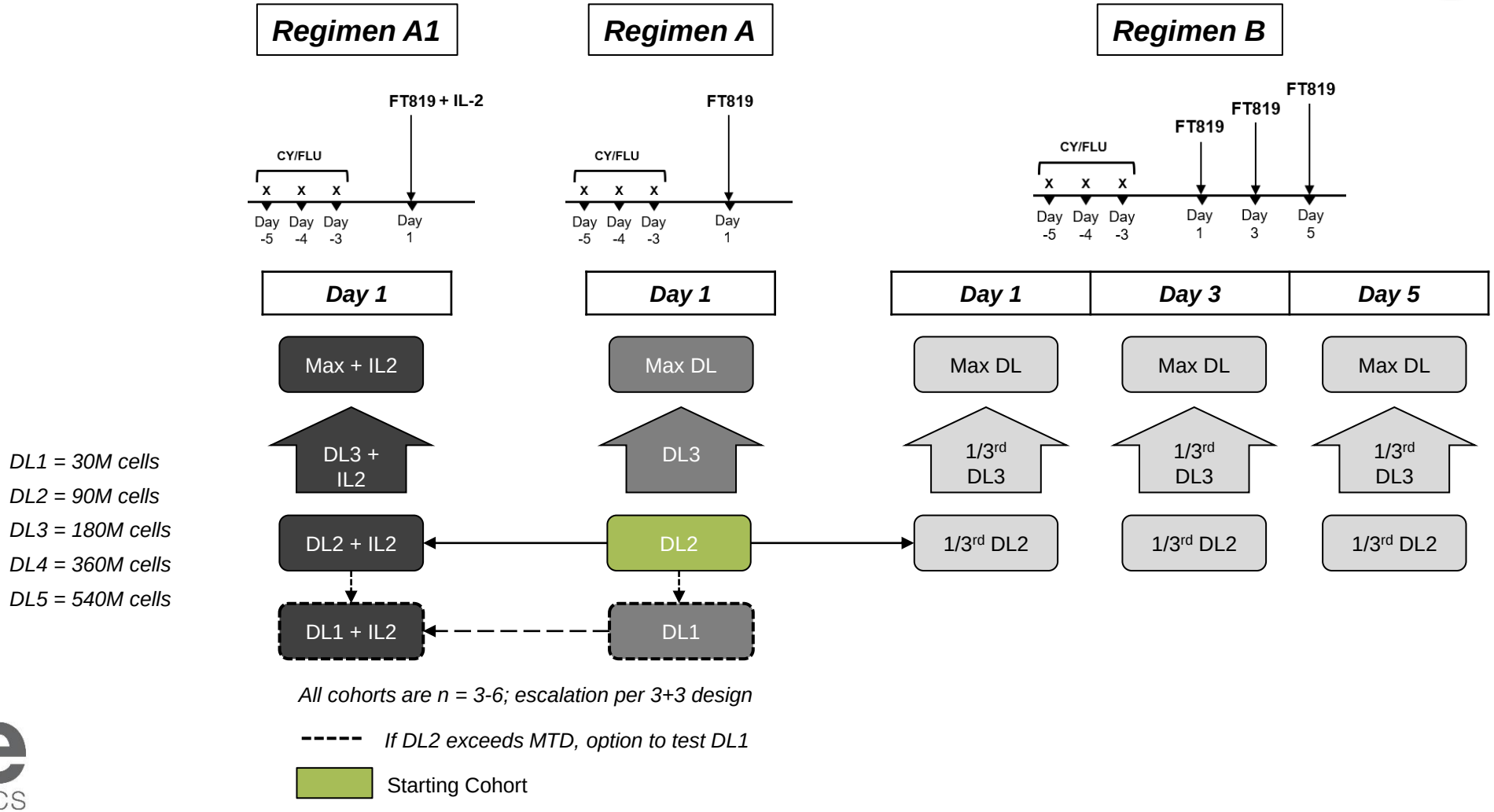
Memorial Sloan Kettering Cancer Center

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



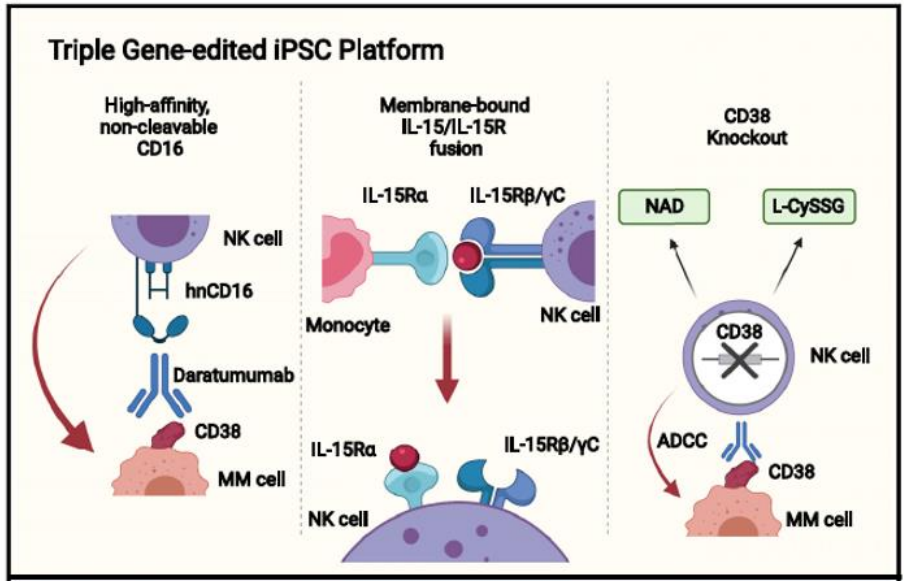
Multiple Myeloma Franchise

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

Cell Stem Cell

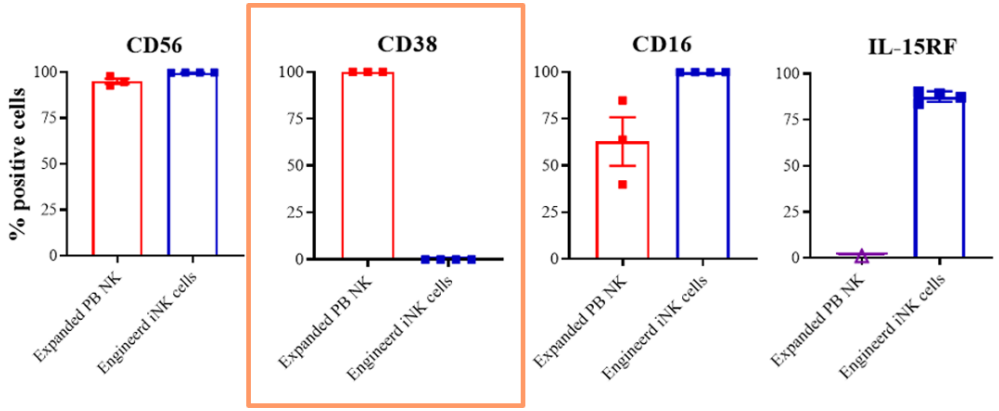
Woan et al., 2021, Cell Stem Cell *28*, 1–14
December 2, 2021 © 2021 Elsevier Inc.
https://doi.org/10.1016/j.stem.2021.08.013

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



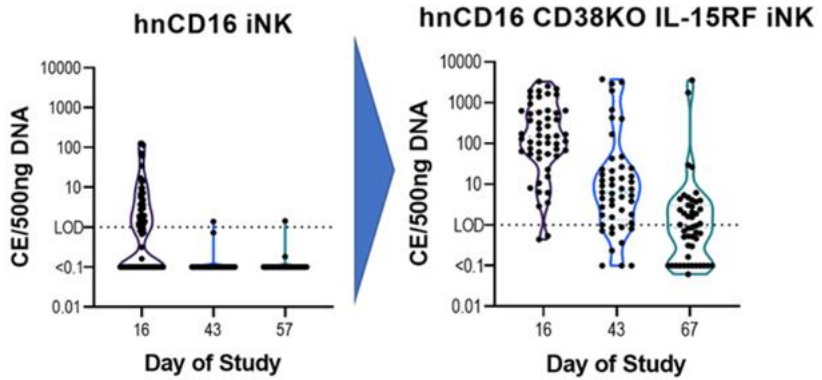
- ✓ *The deletion of the CD38 gene (CD38KO) enhances metabolic fitness, promotes persistence, and enables cytotoxic function in high oxidative stress environments (e.g., suppressive tumor microenvironment)*

Uniformly engineered with three functional elements designed to optimize innate immunity



Enhanced persistence without cytokine support

FT516 vs. FT538 in NSG Mouse



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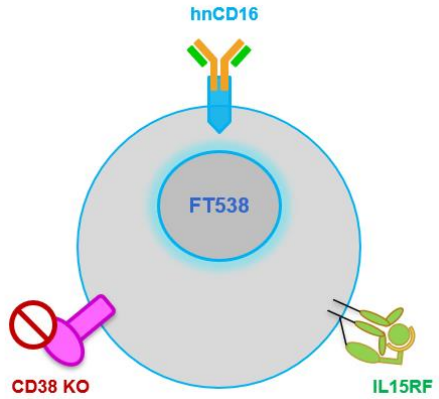
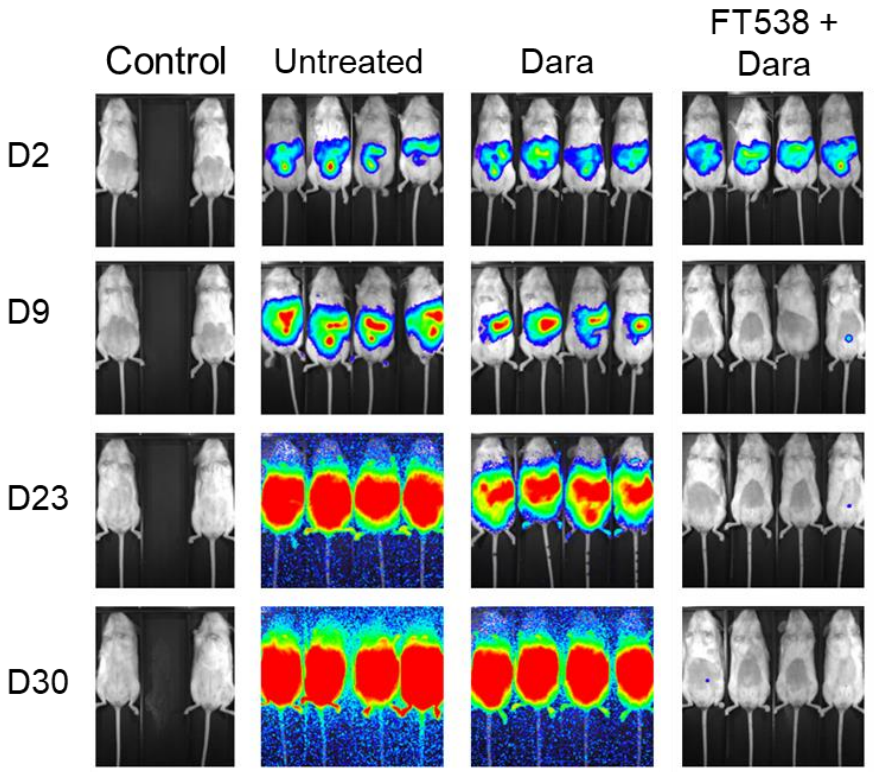
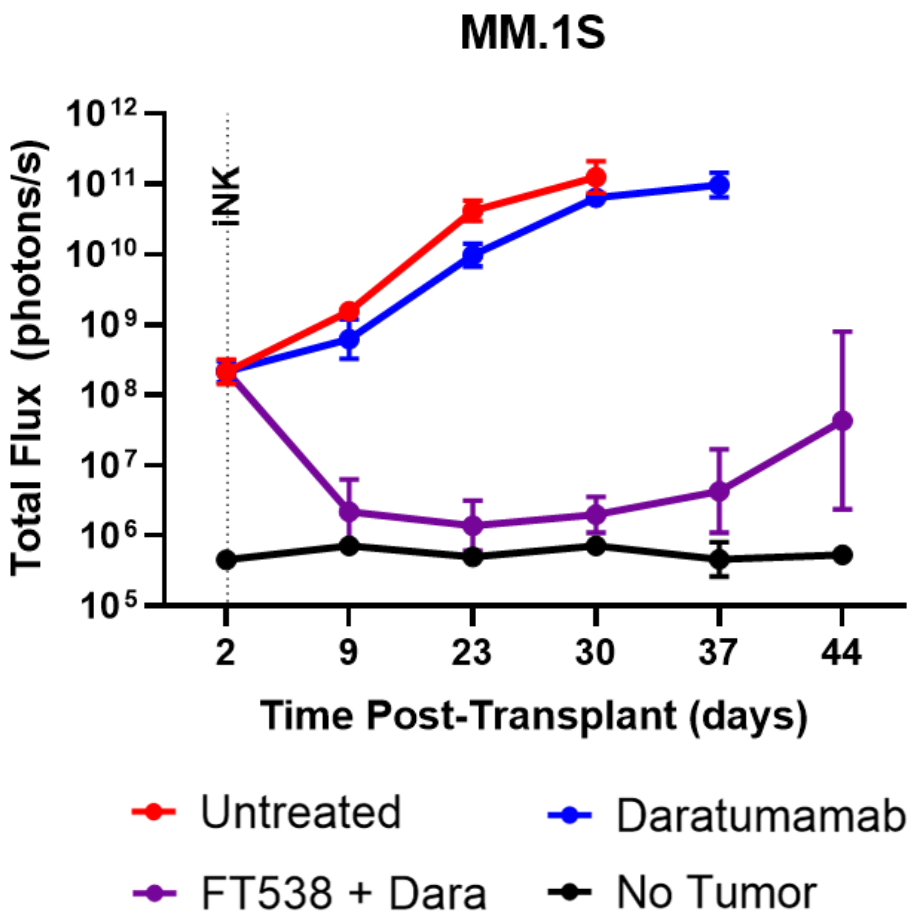
FT538: hnCD16 + IL-15RF + CD38KO NK Cell Product Candidate

Enhanced ADCC in Combination with anti-CD38 mAb In Vivo

Phase 1 Dose Escalation Ongoing in Combination with daratumumab







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

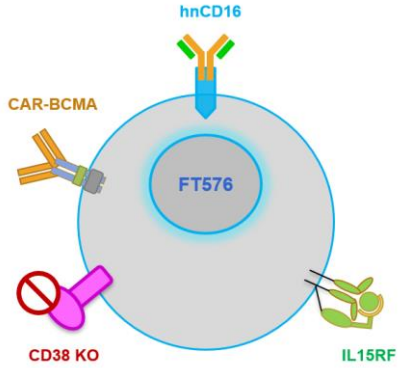
BCMA Binding Domain with Differentiated Activation Threshold


Molecular Therapy
Original Article

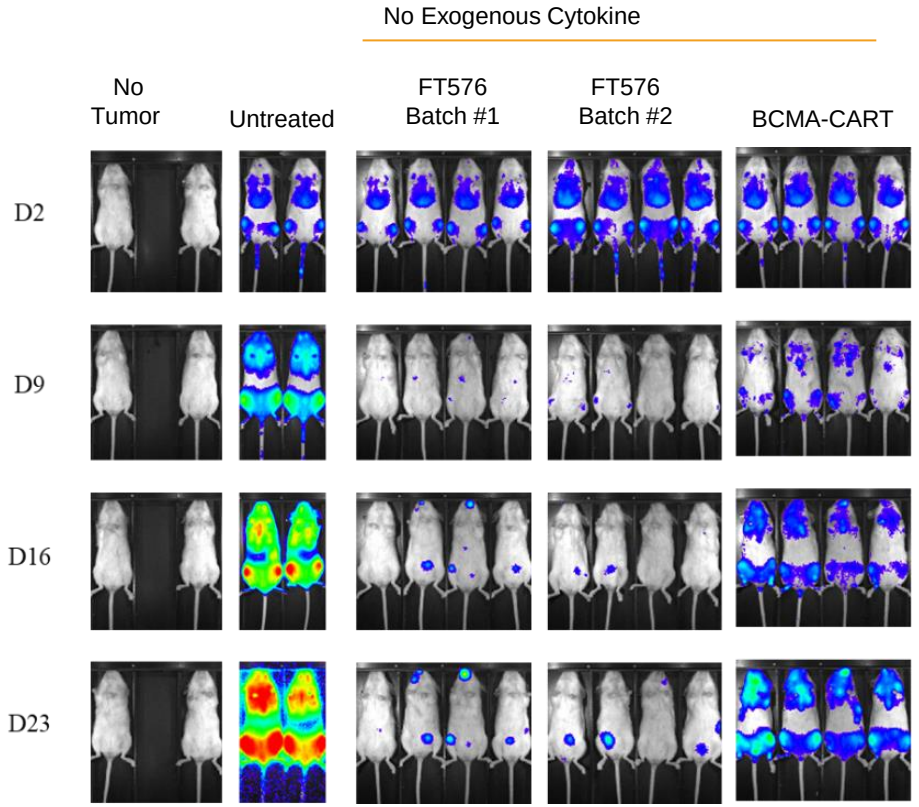
AMERICAN SOCIETY of GENE & CELL THERAPY

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

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



- ✓ 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

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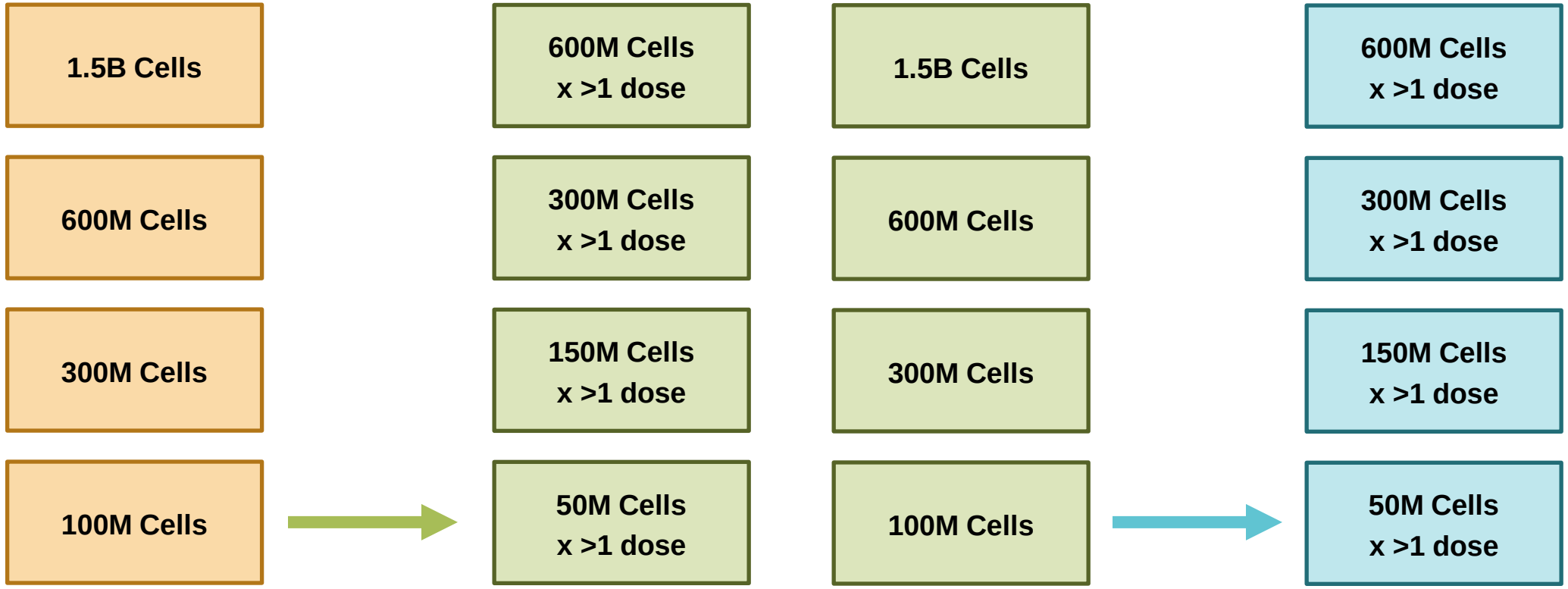
MDC MAX DELBRÜCK CENTER FOR MOLECULAR MEDICINE IN THE HELMHOLTZ ASSOCIATION

FT576-101: Phase 1 Dose Escalation Schema

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

Phase 1 Dose Escalation Ongoing in Single-dose Monotherapy and Single-dose Combination Cohorts



Additional treatment cycles permitted subject to FDA consent

AML Franchise

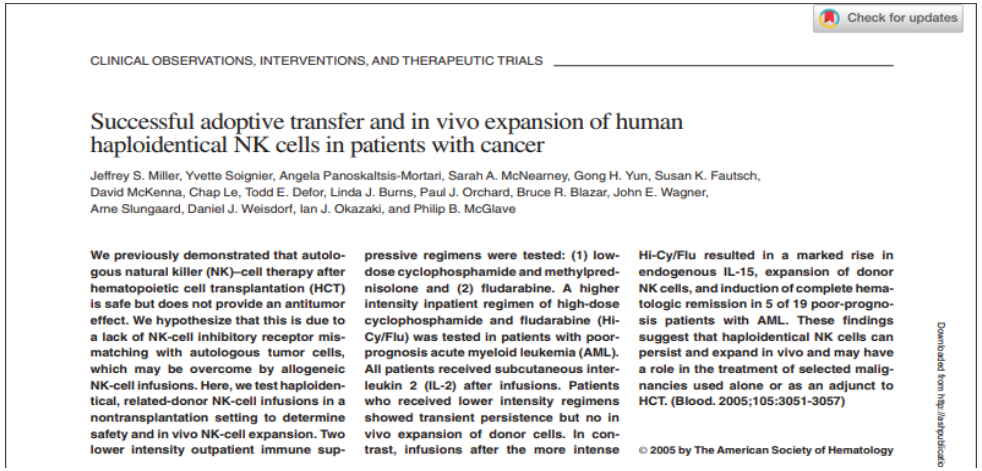
Off-the-Shelf, iPSC-derived NK Cell Franchise for AML

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]

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*

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 at 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

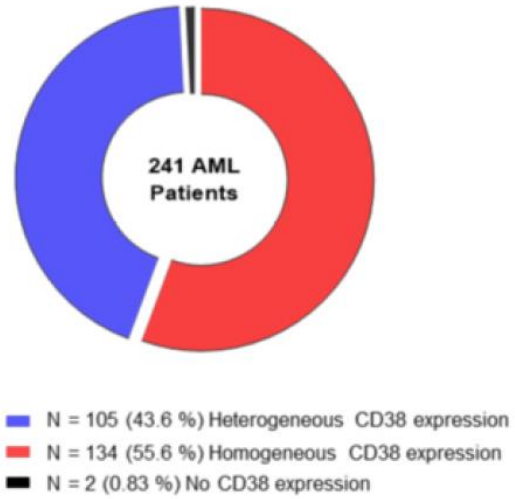
FT538 + daratumumab for Targeting of CD38 on Leukemic Blasts

EUROPEAN HEMATOLOGY ASSOCIATION

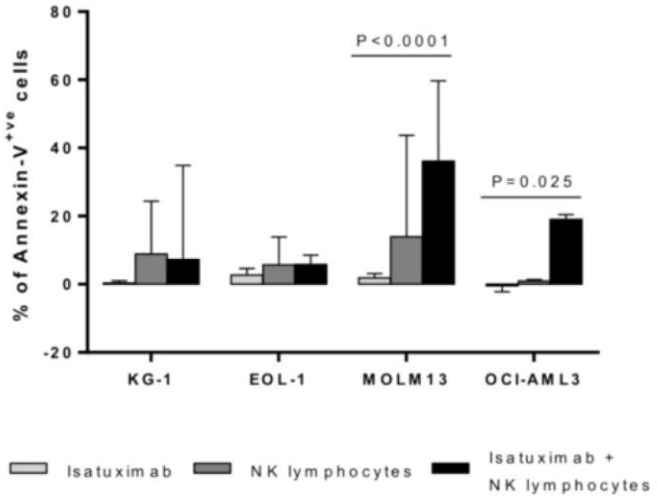
The mode of action of the anti-CD38 monoclonal antibody isatuximab in elderly acute myeloid leukemia

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.

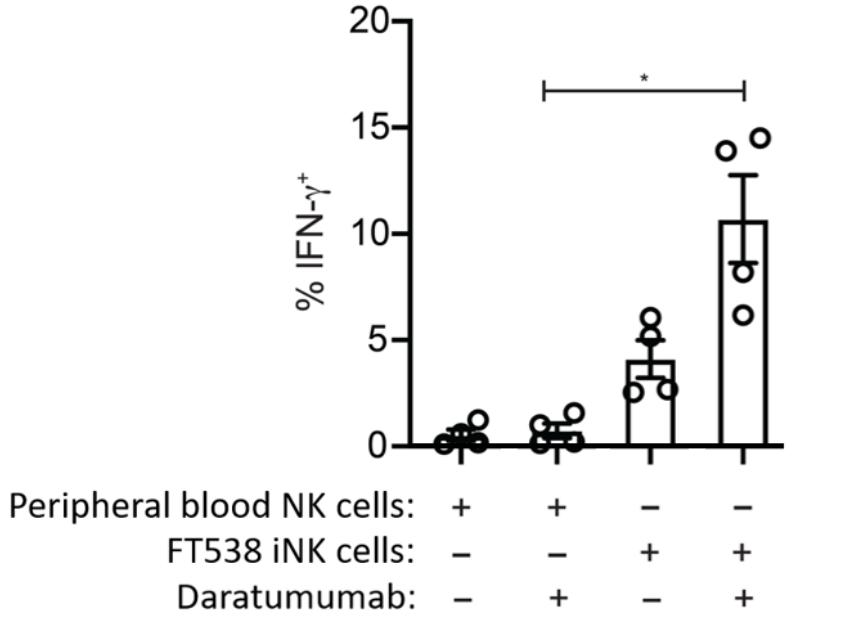
CIMA LAB diagnostics
Universidad de Navarra



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

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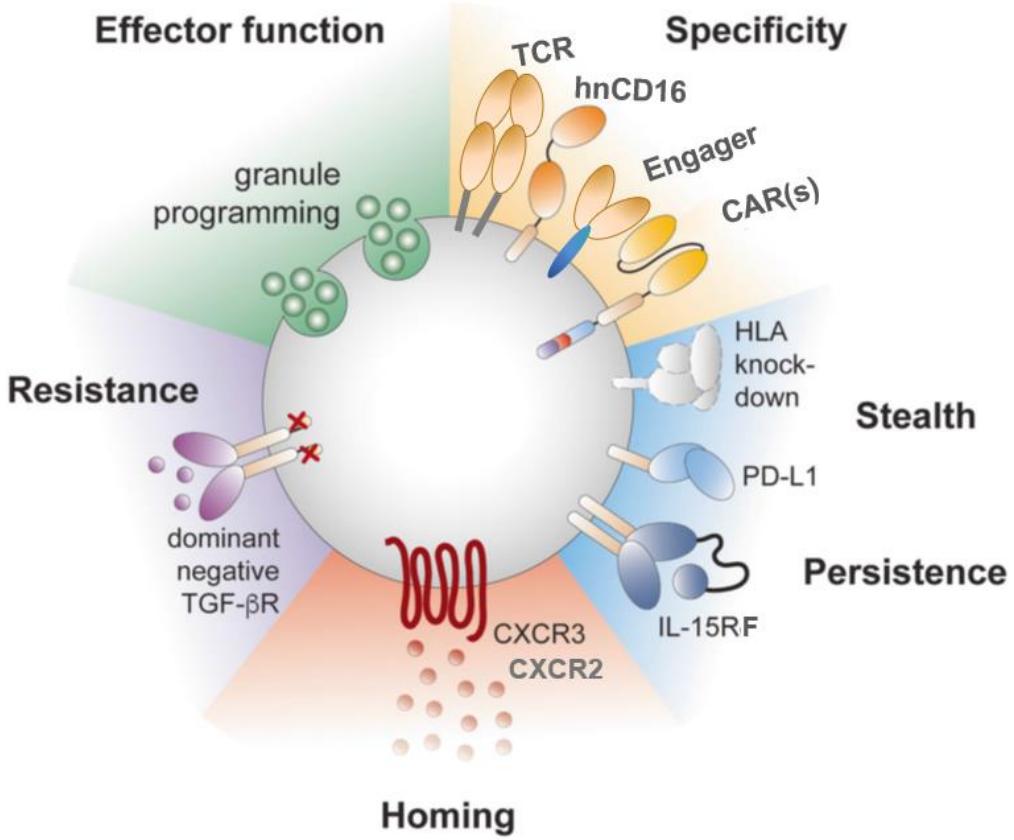
UMN IIT of FT538 + CD38-targeted daratumumab ongoing

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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Solid Tumor Product Pipeline

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

Program	Functionality & *Cell Type*	Target(s)	Indication(s)	Preclin	Phase 1
FT538	hnCD16 + IL15RF + CD38-KO *iNK*	EGFR, HER2, PD1/PD-L1	ST + mAb		
FT536	hnCD16 + IL15RF + CD38-KO + CAR-MICA/B *iNK*	MICA/B	ST ± mAb		
FT873	hnCD16 + IL7RF + CAR-B7H3 *iT*	B7H3	ST ± mAb		
Janssen	*iNK, iT*	KLK2; 1 undisclosed target	ST		
FT825/ Ono	*iNK, iT*	HER2; 1 undisclosed target	ST		

***iPSC** = induced pluripotent stem cell **iNK** = iPSC-derived NK Cell **iT** = iPSC-derived T cell **ST** = solid tumors **mAb** = monoclonal antibody*
***hnCD16** = high affinity, non-cleavable CD16 Fc receptor **IL15-RF** = IL15 receptor fusion **CD38-KO** = CD38 knock-out **CAR** = chimeric antigen receptor*
***EGFR** = Epidermal Growth Factor **HER2** = Human Epidermal Growth Factor Receptor 2 **PD1** = Programmed Cell Death Protein 1 **MICA/B** = MHC class I polypeptide-related sequence A/B **B7H3** = B7 homolog 3 protein **KLK2** = Kallikrein related Peptidase 2*

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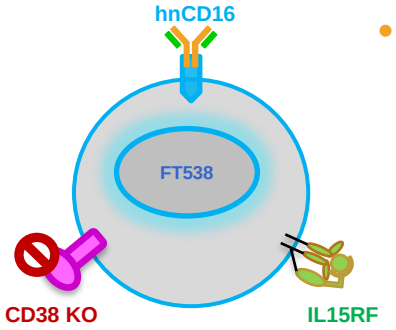
FT538-102: Multi-arm, Dose-escalating Phase 1 Study

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
  - Up to 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

Phase 1 Enrollment Ongoing in Combination with mAb Therapy

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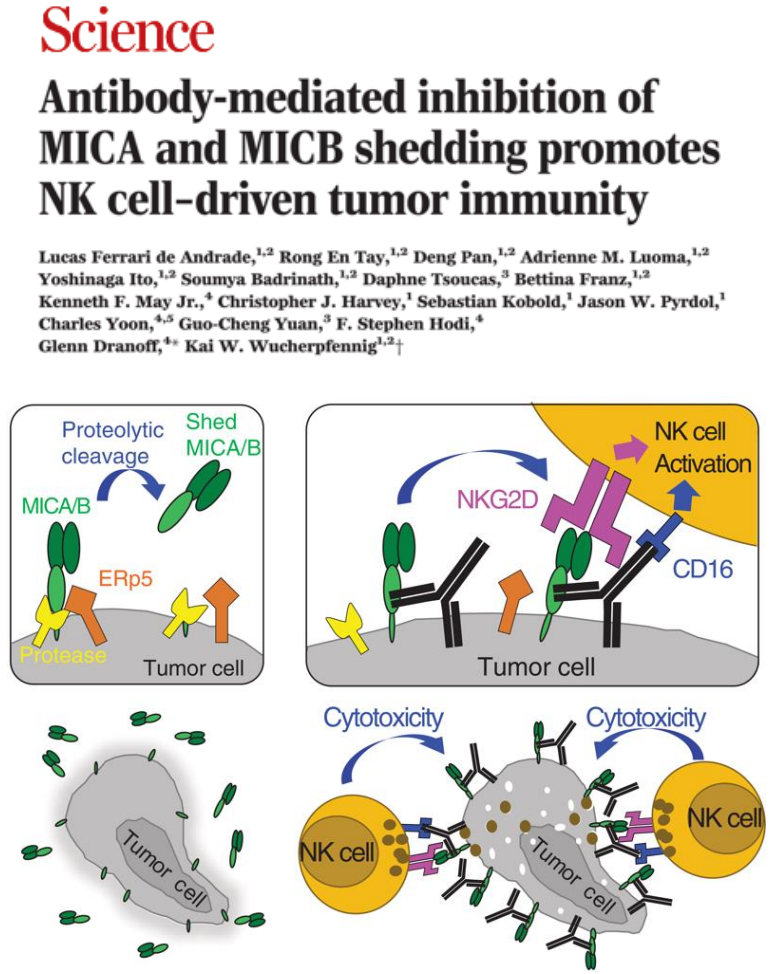
FT536: Multi-antigen Targeted CAR-MICA/B NK Cell Product Candidate

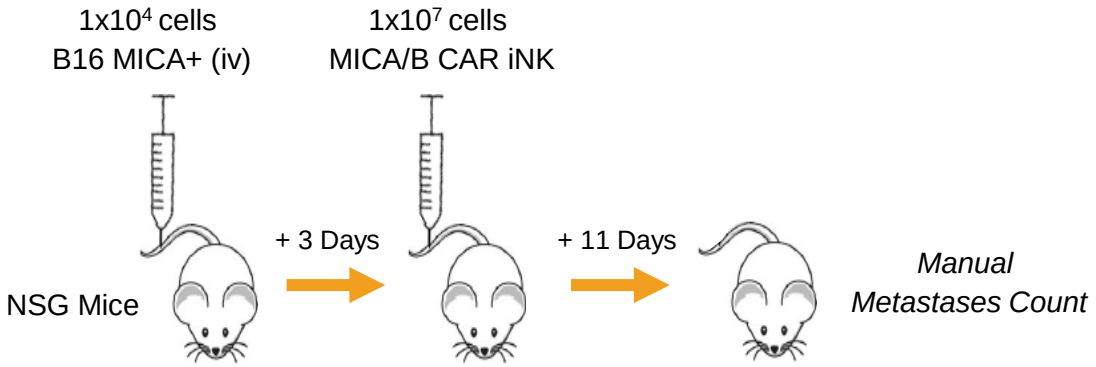
Pan-tumor Targeting Strategy to Overcome Tumor Escape

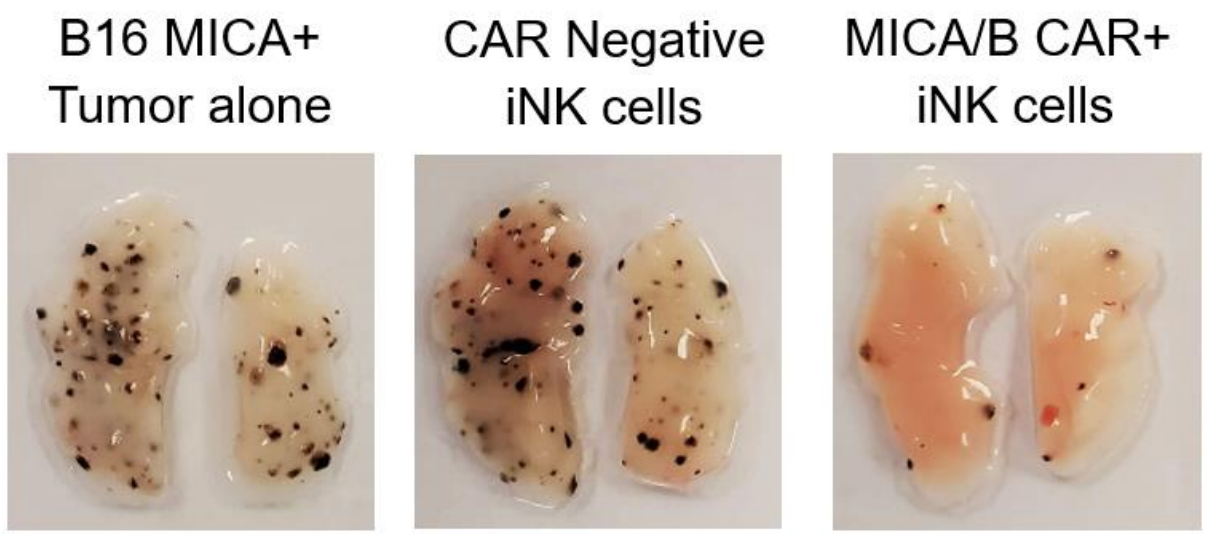
- MICA/B are cell-surface proteins induced by cellular stress and transformation, and their expression has been reported for many cancer types.
- NKG2D, an activating receptor expressed on NK and T cells, targets the membrane-distal α1 and α2 domains of MICA/B, activating a potent cytotoxic response.
- 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.
- 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, resulting in increased MICA/B cell-surface density and restoration of immune cell-mediated tumor immunity

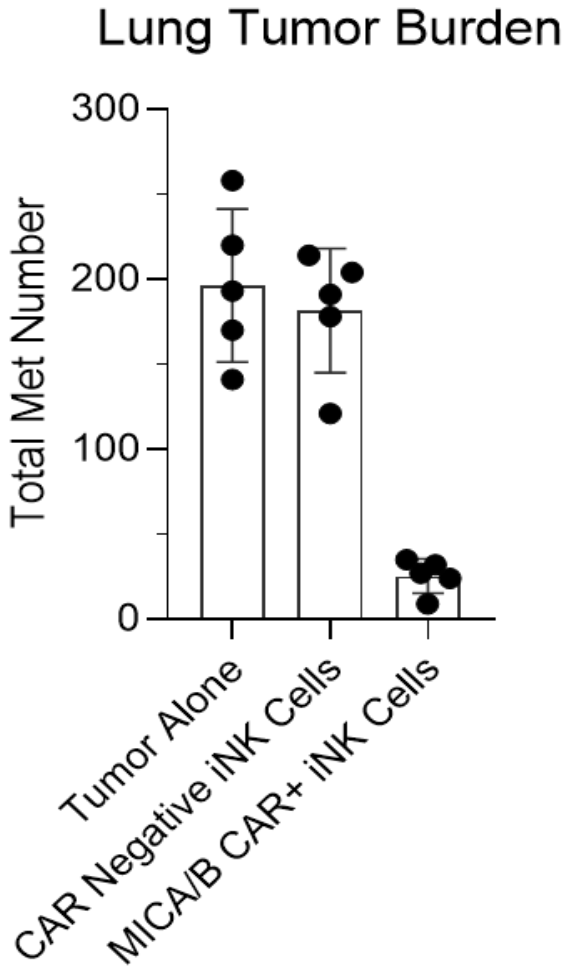


FT536: Multi-antigen Targeted CAR-MICA/B NK Cell Product Candidate

Durable Tumor Reduction of B16 MICA+ Metastatic Lung Lesions

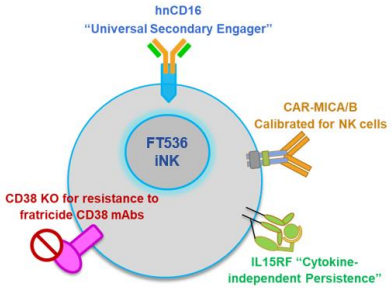






FT536-101: Multi-arm, Dose-escalating Phase 1 Study

Designed to Assess Cooperation with Adaptive Immune System and Enhanced ADCC



- Multiple cycles each comprising Cy / Flu conditioning, 3 doses of FT536, ± mAb
  - Up to 2 cycles; potential to administer additional cycles with clinical benefit and upon relapse
  - FT536 dose ranging from 100M cells / dose to 3B cells / dose
  - Each mAb combination enrolls independently

	Monotherapy	Pembrolizumab, Avelumab	Trastuzumab	Cetuximab	Amivantamab
Target	NA	PD-(L)1	HER2	EGFR	EGFR-MET
Eligibility	No biomarker-driven eligibility	Documented PD-L1 expression	Documented HER2 expression; NSCLC with HER2 mutation	EGFR+ tumors, incl. KRAS/NRAS and driver mutations	EGFR driver mutations, MET mutations
Primary Cancers	NSCLC, CRC, BC, Ovarian, Pancreatic	NSCLC, GE, HNSCC, TNBC, UC	Gastric, Breast	NSCLC, CRC, HNSCC	NSCLC

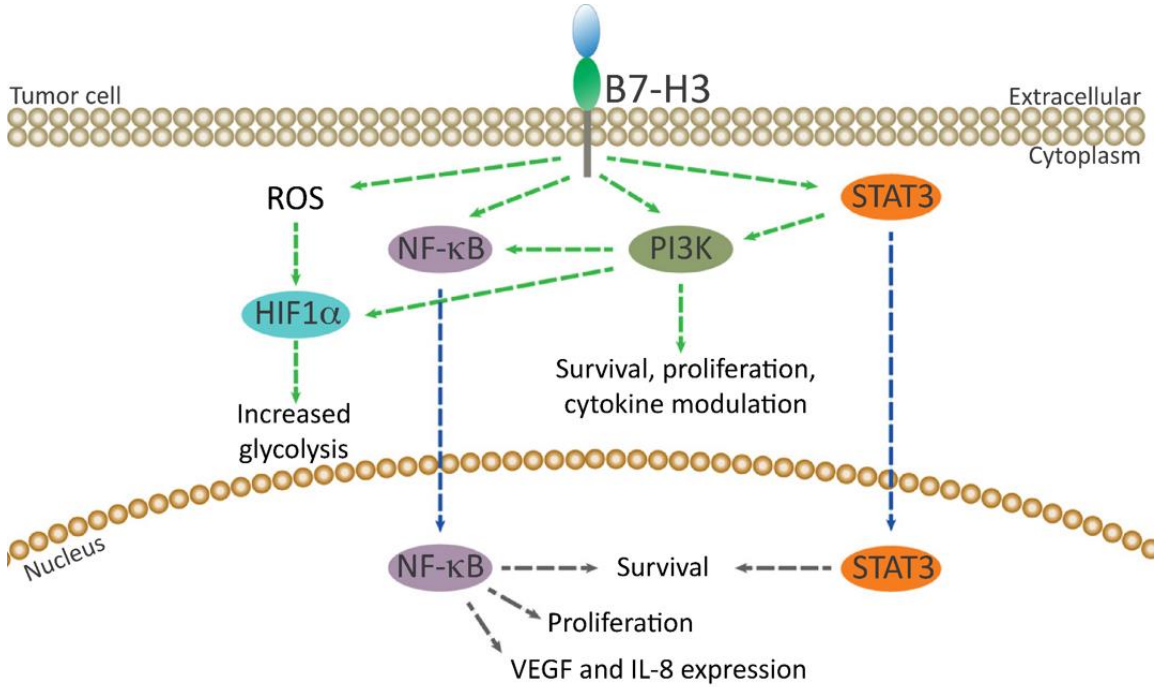
CRC = colorectal cancer; GE = gastroesophageal; HNSCC = head and neck squamous cell carcinoma; NA = Not applicable; NSCLC = non-small cell lung carcinoma; TNBC = triple-negative breast cancer; UC = urothelial carcinoma

Phase 1 Dose Escalation Ongoing; DLT Clearance of DL1 as Monotherapy Initiates Combination with mAb Therapy

B7H3-targeted, Multiplexed-engineered CAR 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
  - Limited expression in normal tissues
  - High levels found on immunologically “cold” tumors (e.g., prostate, HNSCC, GBM, soft tissue sarcomas)
  - Often associated with poor prognosis
- Shown to be 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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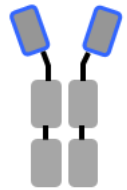
B7H3-targeted, Multiplexed-engineered CAR Product Candidate

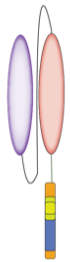
Identification of Novel anti-camB7H3 scFv

CAR Design

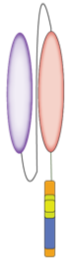
- Based on camelid antibodies
- Maintain high target affinity and specificity associated with conventional antibodies
- Demonstrate good physiochemical stability, reduced immunogenicity, and preferred agility associated with their reduced size
- Generated single-domain targeting sequence (V_HH)
- Created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



V_HH anti-cam B7H3 scFv

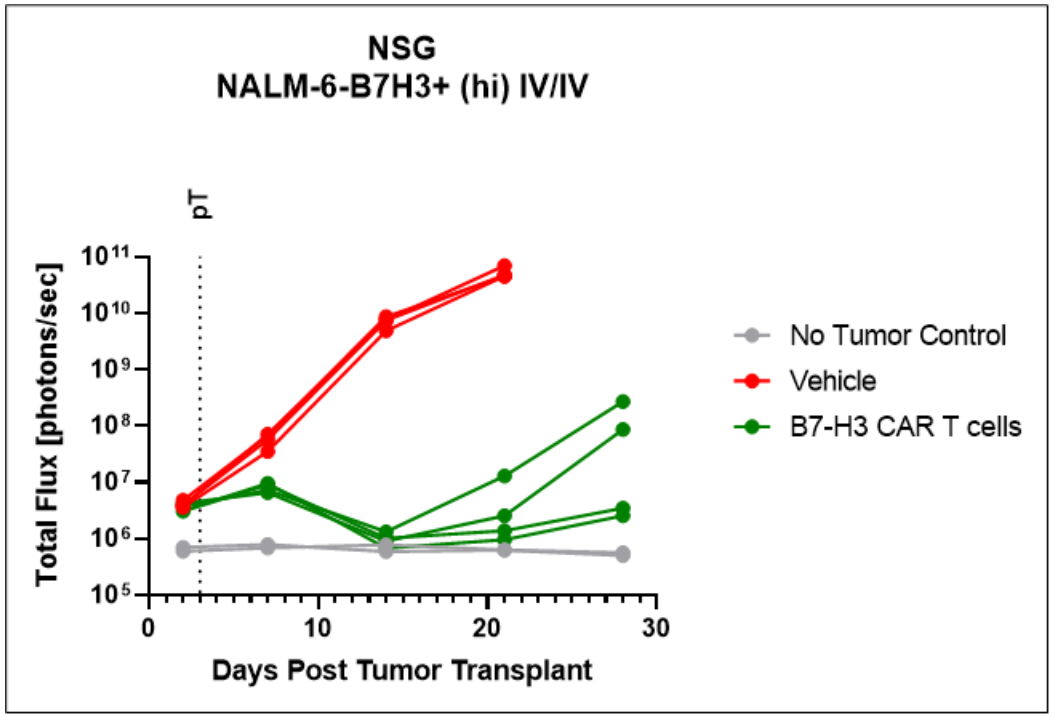


NK Cell CAR Construct



T Cell CAR Construct

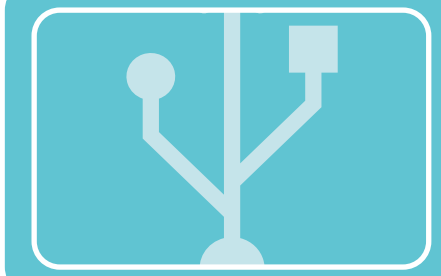
camB7-H3 CAR-T cells Show Durable Control and Prevent Disease Progression *in vivo*



Collaborations & Financials

Janssen Cancer Immunotherapy Collaboration

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

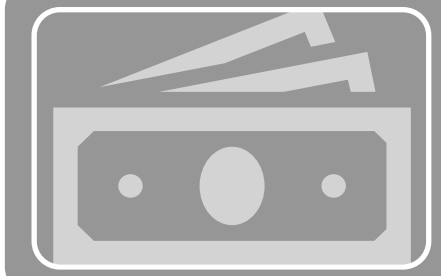


Oncology Innovation for Heme Malignancies & Solid Tumors

- Proprietary antigen binding domains contributed by Janssen
- Four targets selected; 2 for heme malignancies and 2 for solid tumors
- Substantial investment in next-generation cellular features / functionality

Strategic Collaboration

- FATE leads preclinical development to IND submission
- Janssen maintains option to worldwide development and commercialization
- FATE retains right to opt-in to 50-50 arrangement in US



Significant Economics

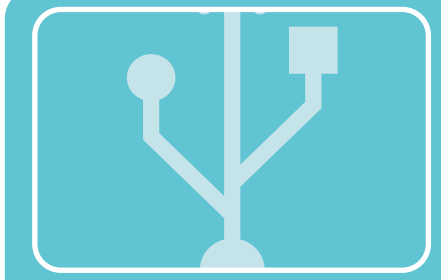
- $100m upfront (+$50m equity put)
- Janssen pays for all collaboration costs
- $3+ billion in milestones, double-digit royalties

ONO Cancer Immunotherapy Collaboration

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

Fate THERAPEUTICS

ONO PHARMACEUTICAL CO.,LTD.

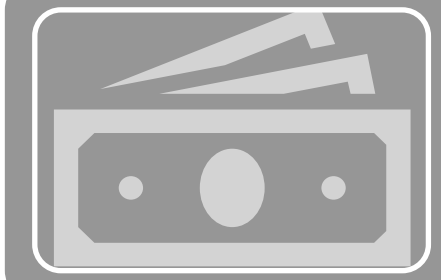


Oncology Innovation for Solid Tumors

- Proprietary antigen binding domains contributed by Ono
- Multiplexed-engineered, CAR-targeted product candidates
- Incorporating multiple MOAs to address solid tumor microenvironment

Strategic Collaboration

- FATE leads preclinical development to pre-IND milestone
- Ono maintains option to worldwide development and commercialization
- FATE retains right to opt-in to 50-50 arrangement in US and Europe



Financial Terms

- $10m upfront
- 50-50 cost sharing to pre-IND milestone
- Up to $840 million in milestones, mid-single to low double-digit royalties

Fate THERAPEUTICS

Financial Summary

As reported in Company's Consolidated Financial Statements

Three Months Ended September 30, 2022 *As reported in Company's Consolidated Financial Statements*	
Revenue	$15.0M
Operating Expense[1]	$101.4M
Cash & Cash Equivalents	$519M
Total Shares Outstanding[2]	111.1M

2022 Cash, Cash Equivalents & Investments ≥$440M[3]

[1] *Includes $19.5m in stock-based compensation*

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

[3] *Not including a $12.5M option exercise fee triggered by Ono's opt-in decision in November for its first iPSC-derived CAR T cell candidate or other potential collaboration milestones in 4Q22*

Fate THERAPEUTICS

Better Cells For Better Therapies™