

Better Cells For Better Therapies™

Programmed Cellular Immunotherapies

Natural Killer Cell Franchise Update

November 10, 2018

Forward-Looking Statements

This presentation contains "forward-looking statements" within the meaning of the Private Securities Litigation Reform Act of 1995, including statements regarding the Company's advancement of and plans related to the Company's product candidates and clinical studies, the therapeutic potential of the Company's natural killer (NK) cell cancer immunotherapies, including FATE-NK100 and FT500, the Company's regulatory strategy and advancement of its clinical studies, and the Company's plans for its intended clinical investigation of FATE-NK100 and FT500. 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 of cessation or delay of ongoing or planned development and clinical activities for a variety of reasons (including requirements that may be imposed by regulatory authorities on the initiation or conduct of clinical trials or to support regulatory approval, or on the manufacture of its product candidates, any adverse events or other results that may be observed during development, or difficulties in manufacturing or supplying the Company's product candidates for clinical trials), the risk that results observed in prior preclinical studies of FATE-NK100 or FT500 may not be replicated in subsequent studies or ongoing or future clinical trials, and the risk that FATE-NK100 or FT500 may not produce therapeutic benefits or may cause other unanticipated adverse effects. 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.



Introduction Scott Wolchko, President & CEO

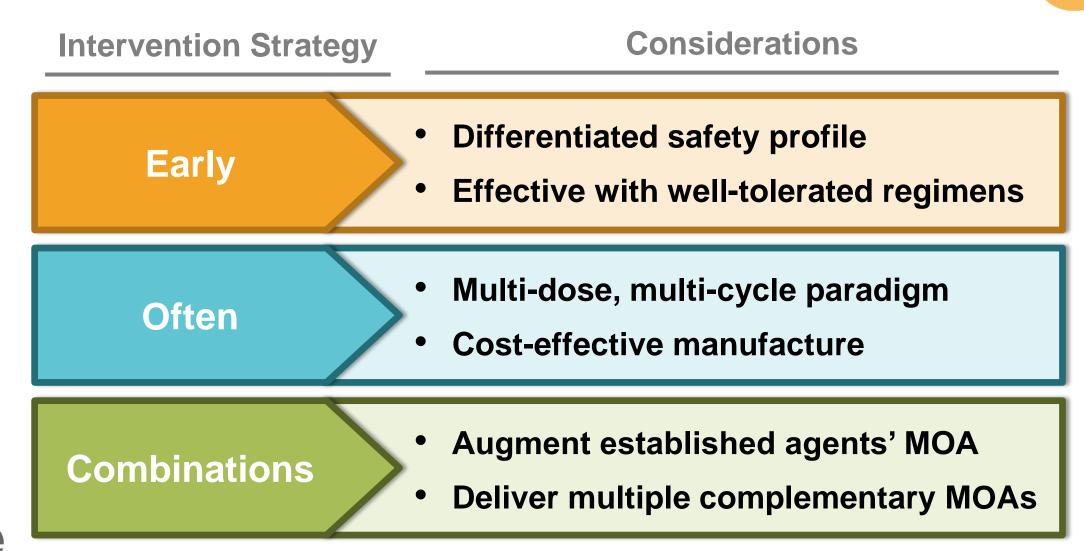


- Introduction Scott Wolchko, President & CEO
- Adaptive Memory NK Cells Jeffrey S. Miller, MD
- Translational Insights Dan Shoemaker, PhD, Chief Scientific Officer
- Clinical Observations Sarah A. Cooley, MD
- NK Cell Product Franchise Bob Valamehr, PhD, Chief Development Officer
- Concluding Remarks Scott Wolchko, President & CEO



Cell-based Cancer Immunotherapy

Therapeutic Vision for Long-Term Durable Responses



First-in-Class, Allogeneic Donor-derived NK Cell Cancer Immunotherapy

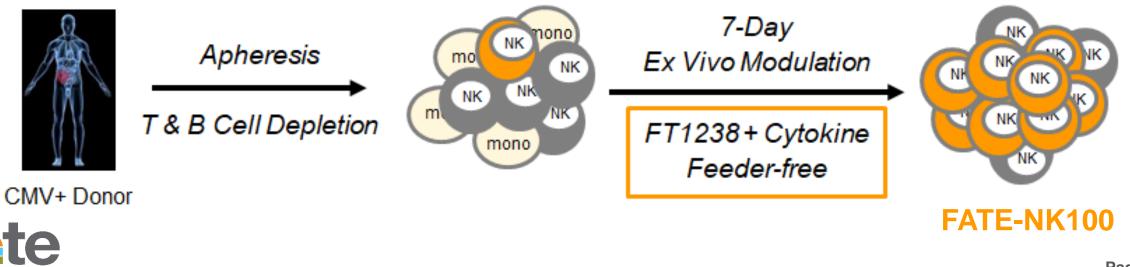


Adaptive Memory NK Cells

A Potent Subset of Activated NK Cells with Unique Anti-Tumor Properties

Heightened Effector Function Enhanced Persistence

Resistant to Immuno-regulatory Suppression



First-in-Human Phase 1 Clinical Trials



	VOYAGE	APOLLO	DIMENSION			
R/R AML		Recurrent Ovarian	Solid Tumors	EGFR+	HER2+	
NK100 Dose	Single	Single ¹	Single ¹ Single ¹ Single		Single + trastuzumab ¹	
Route of Admin	IV	IP			IV	
Dose Levels	3	3 3 3		4	4	
Range (x10 ⁷ /kg)	1, 3, 10	1, 3, 10	1, 3, 10	0.1, 1, 3, 10	0.1, 1, 3, 10	
Conditioning	Lympho-D	Lympho-C2x2	Lympho-C1x2	Lympho-C1x2	Lympho-C1x2	
Су	60 mg/kg x 2d	300 mg/m2 x 2d	300 mg/m2 x 1d	300 mg/m2 x 1d	300 mg/m2 x 1d	
Flu	25 mg/m2 x 5d	25 mg/m2 x 2d	25 mg/m2 x 2d	25 mg/m2 x 2d 25 mg/m2 x 2d 25		
IL-2 Regimen	6MU sc x 3/w x 2w	6MU ip x 3/w x 2w	6MU sc x 3/w x 2w	/w x 2w 6MU sc x 3/w x 2w 6MU sc x 3/w		



¹ A second treatment cycle is optional for subjects with SD or better at Day 28 or Day 56

Clinical Objectives & Observations in Ongoing Dose-Escalation Stages

Demonstrate Unique Properties & Functionality	NK100 has shown augmented biological properties and enhanced activity vs. original healthy donor's and the patient's NK cells
Establish Product Safety & Well-Tolerated Regimen	No NK100-related DLTs, one NK100-related SAE (Grade 3) and no reports of CRS / neurotoxicity ¹
Observe Evidence of Activity in Heavily Pre-Treated Patients	7 of 14 subjects in ongoing dose-escalation demonstrated evidence of clinical activity ²
Gain First Look at Potential for Multi-Dosing Paradigm	Delivered second treatment cycle of FATE-NK100 to 3 subjects, showing NK cell persistence and tolerance



¹ As of October 22, 2018 data cutoff. Database is not locked and final data are subject to change.

² For VOYAGE, includes mLFS; for APOLLO and DIMENSION, includes best overall response of stable disease or better.

Adaptive Memory NK Cells Jeffrey S. Miller, MD

Natural Killer Cells

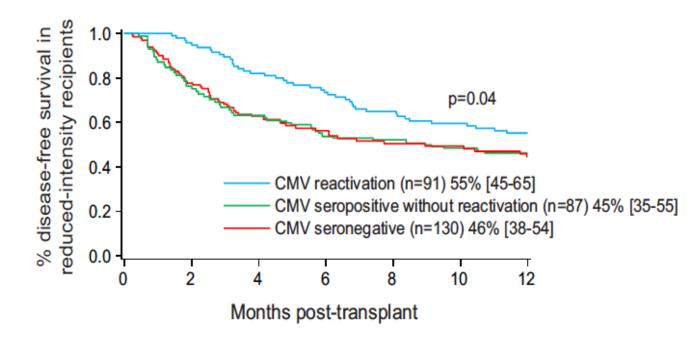
Unique Properties as compared to T Cells



Advantages of NK Cells

- Proven clinical safety (no GvHD or CRS)
- Demonstrated allogeneic feasibility
- Same potent effector machinery as T cells
- Multi-faceted antigen recognition
- Chemokine / cytokine production to prime adaptive immunity
- Potential to recruit and alter homing of T cells

Adaptive NK Cells Frequency Correlates with Improved Survival



Cichocki & Miller, Leukemia 30:456, 2016



Adaptive Memory NK Cells

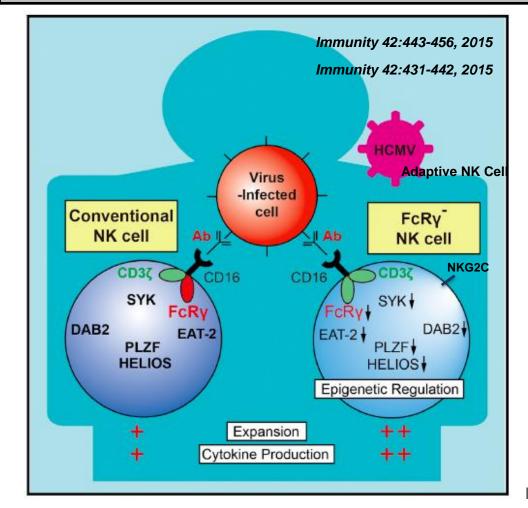
Introduction

Attributes Of Adaptive NK Cells

- Protect against relapse after HCT *Cichocki & Miller, Leukemia 30:456, 2016*
- Enhanced cytokine production
- Survive longer with properties of immune memory
- Mediate enhanced ADCC
- Decreased PD-1, TIGIT and NKG2A checkpoint expression
- Inherently resistant to immuno-regulatory suppression by MDSCs and T-regs Sarhan and Miller, CR (2016) & CIR (2018)



CMV-Induced Adaptive NK Cells

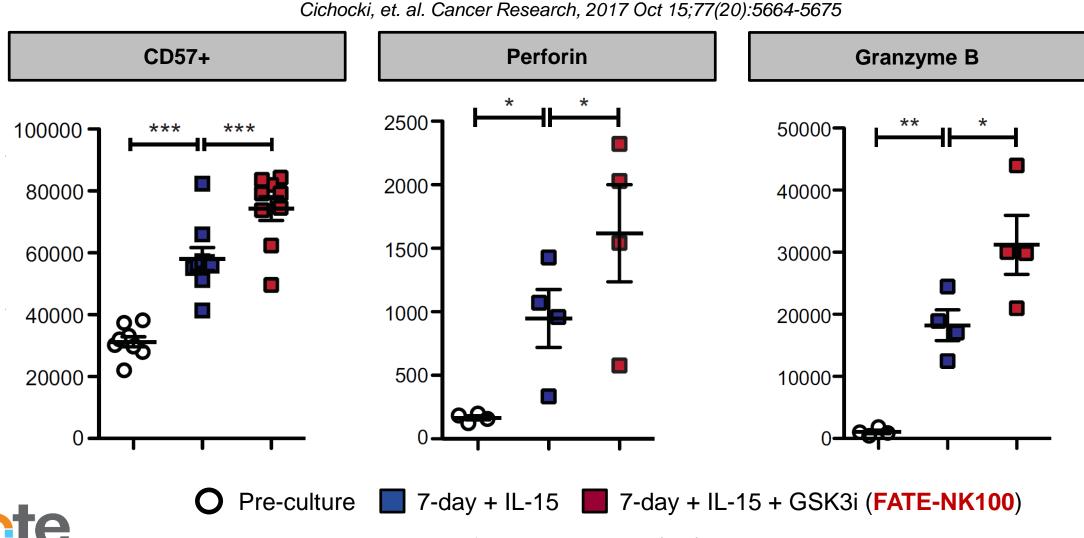




Augmented Biological Properties

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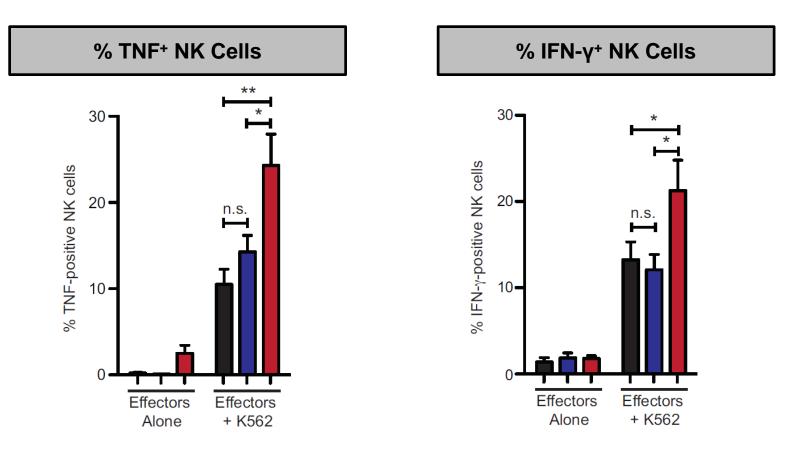


Median fluorescence intensity (MFI)

Enhanced Cytokine Production In Vitro



Cichocki, et. al. Cancer Research, 2017 Oct 15;77(20):5664-5675



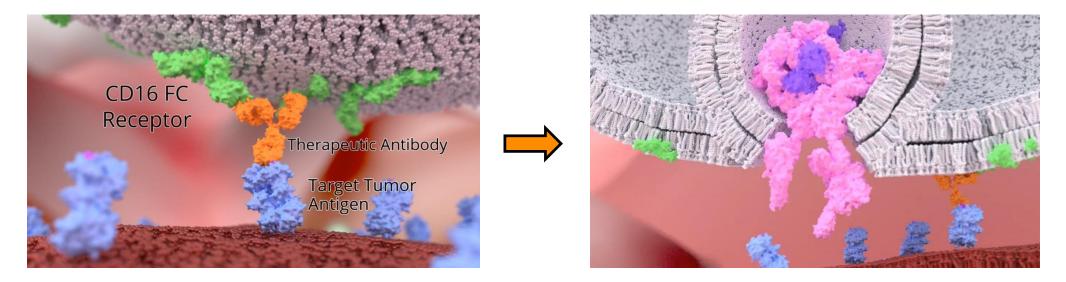
Overnight IL-15 Primed 7-day + IL-15 7-day + IL-15 + GSK3i (FATE-NK100)



Antibody Dependent Cellular Cytotoxicity (ADCC)

Mediated by NK Cells

 Antibody binds to tumor target via Fab and to Fc receptor on NK cells (CD16) via Fc, initiating release of perforins / granzyme resulting in tumor cell death

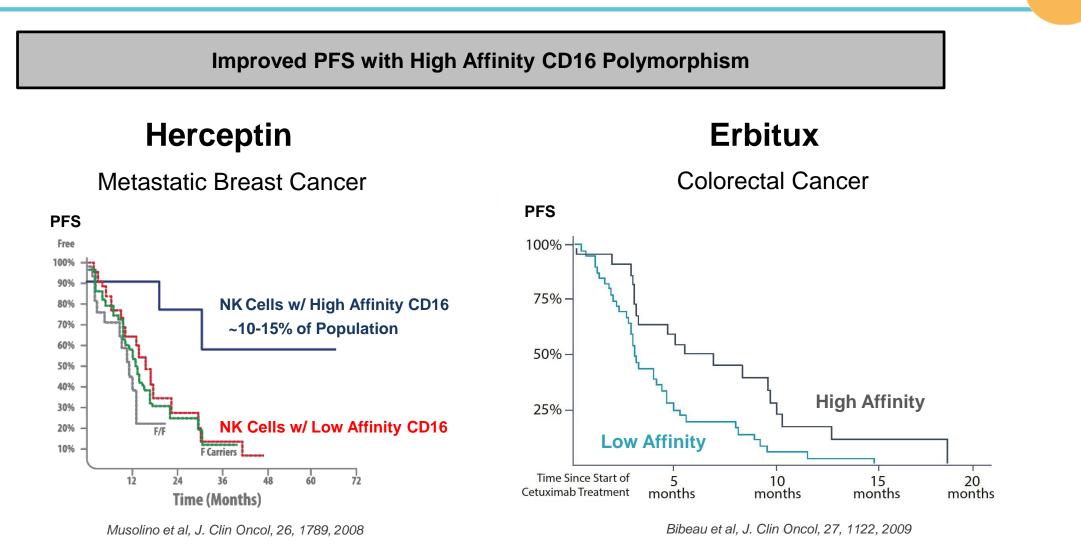


- ADCC contributes to anti-tumor activity of many FDA-approved antibodies
 - Herceptin, Erbitux, Rituximab, Darzalex, etc.



ADCC: Clinical Proof-of-Concept

Enhanced ADCC Correlates with Improved Outcomes

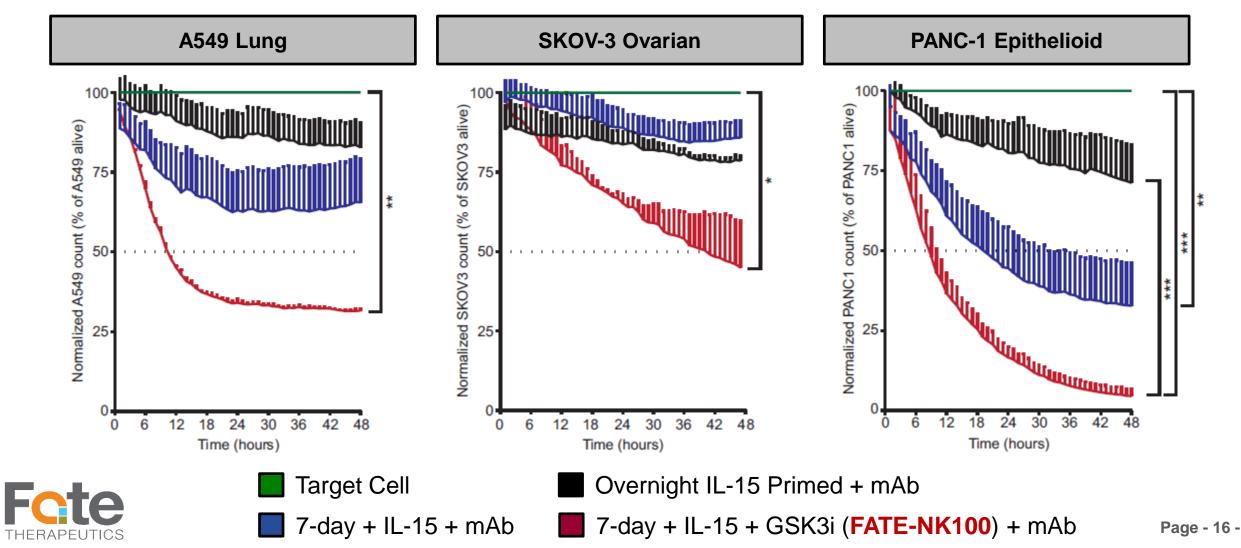




Enhanced ADCC In Vitro



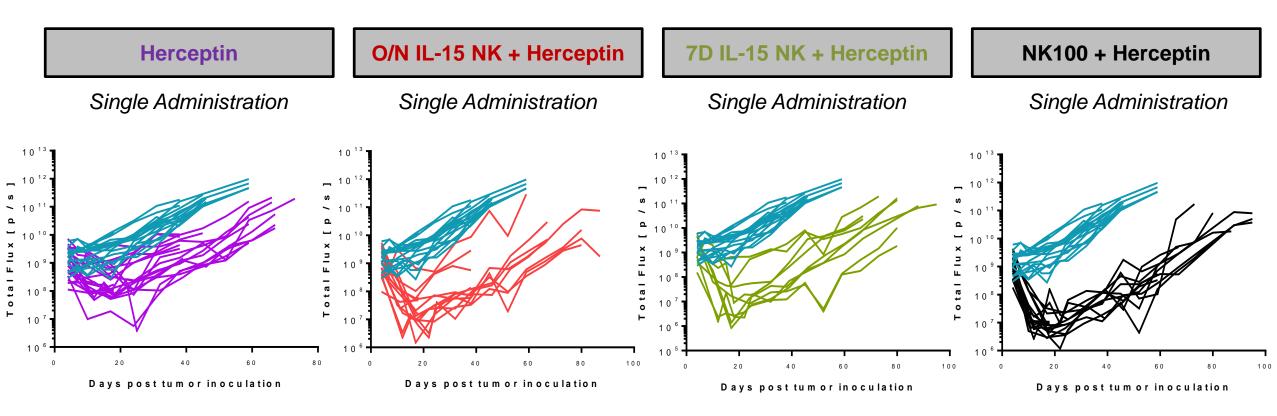
Cichocki, et. al. Cancer Research, 2017 Oct 15;77(20):5664-5675



Augmented and Consistent ADCC In Vivo with Herceptin

Masonic Cancer Center UNIVERSITY OF MINNESOTA

Cichocki, et. al. Cancer Research, 2017 Oct 15;77(20):5664-5675



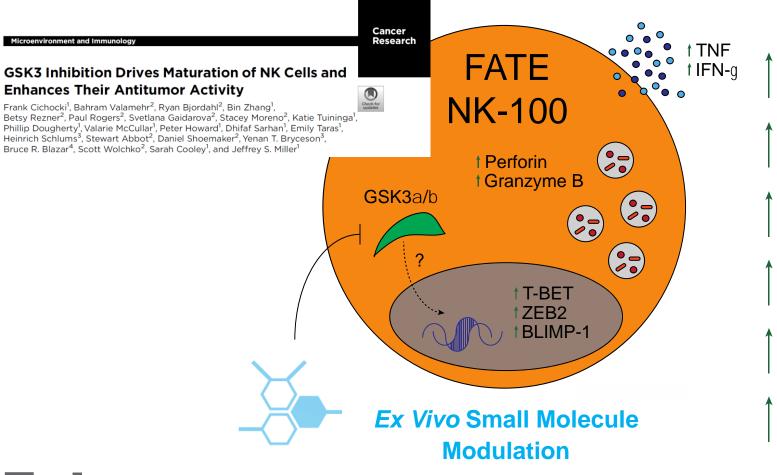
Control = SKOV-3 Ovarian Tumor Cells



Unique Biological Properties & Functionality



Published OnlineFirst August 8, 2017; DOI: 10.1158/0008-5472.CAN-17-0799



↑ Maturation during *ex vivo* expansion († CD57,† KIR,↓ NKG2A,↓ TIGIT)

Enrichment of CD57⁺NKG2C⁺ adaptive NK cells from CMV⁺ donors.

Tumor necrosis factor (TNF) and interferon (IFN)-g production.

Natural cytotoxicity against solid tumor targets

Antibody-dependent cellular cytotoxicity against solid tumor targets

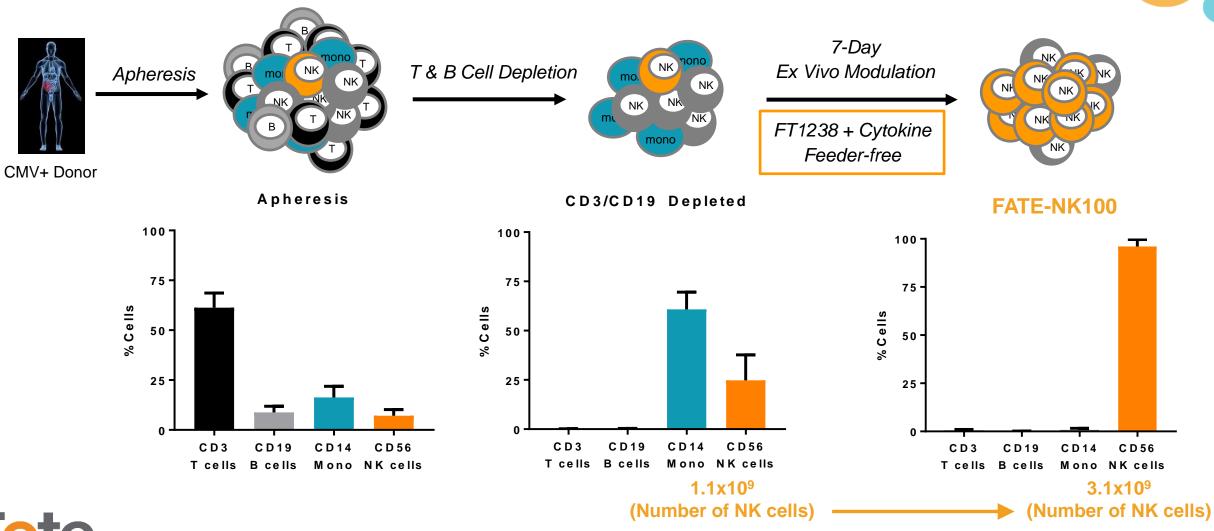
Tumor control in a xenogeneic model of ovarian cancer



Translational Insights *Dan Shoemaker, PhD, Chief Scientific Officer*

FATE-NK100: Clinical Manufacture

Robust Expansion & Production of a Pure Population of CD56+ NK Cells

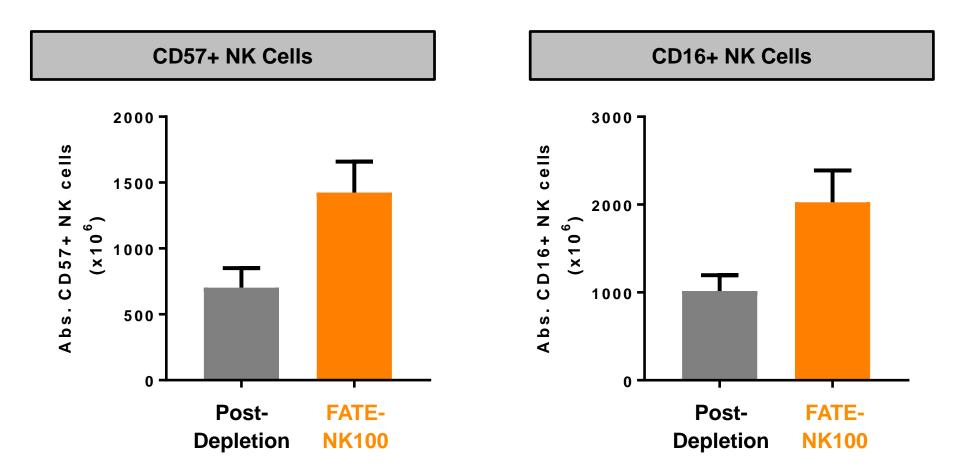


Fcte THERAPEUTICS

N = 15 (Voyage, Apollo, Dimension)

FATE-NK100: Clinical Manufacture

Comprised of Mature CD57+ NK Cells Expressing CD16 Fc Receptor

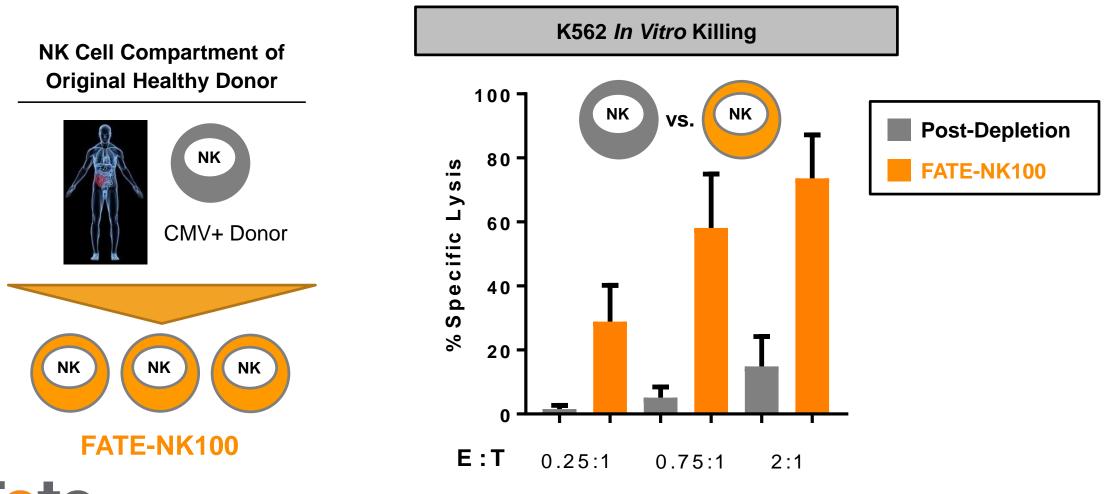






FATE-NK100: Clinical Manufacture

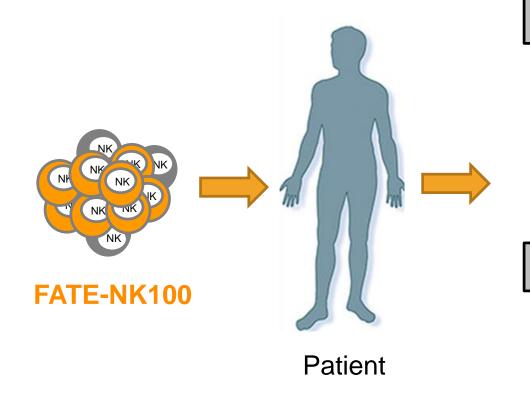
Enhanced Cytotoxicity as Compared to NK Cells from Original Healthy Donor





N = 10 (data not yet available for Dimension)

Characterization of Patient Samples



Blood / Serum

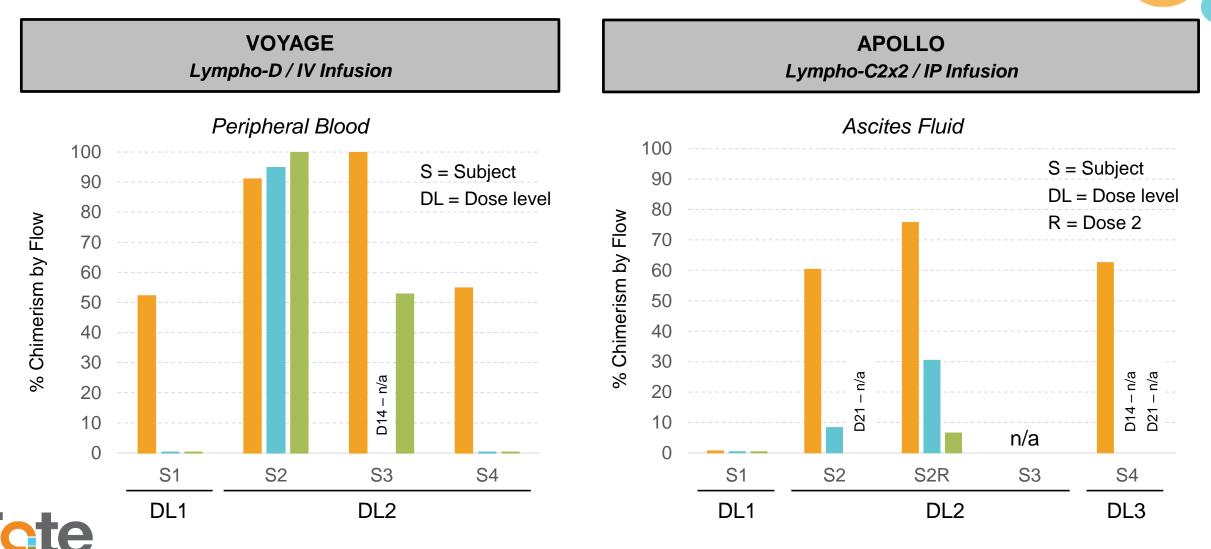
- Chimerism
- Function
- Immunophenotyping
- Cytokine production

Biopsy

- Chimerism
- Infiltration
- Immune activity / modulation

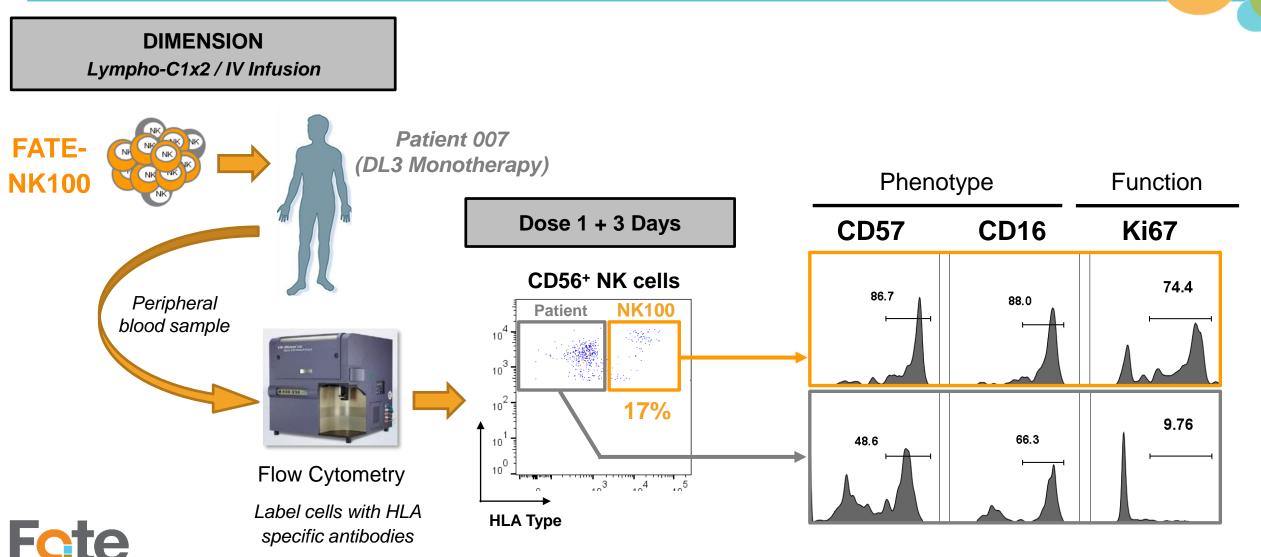


In Vivo Persistence of FATE-NK100 Day 7 Day 14 Day 21



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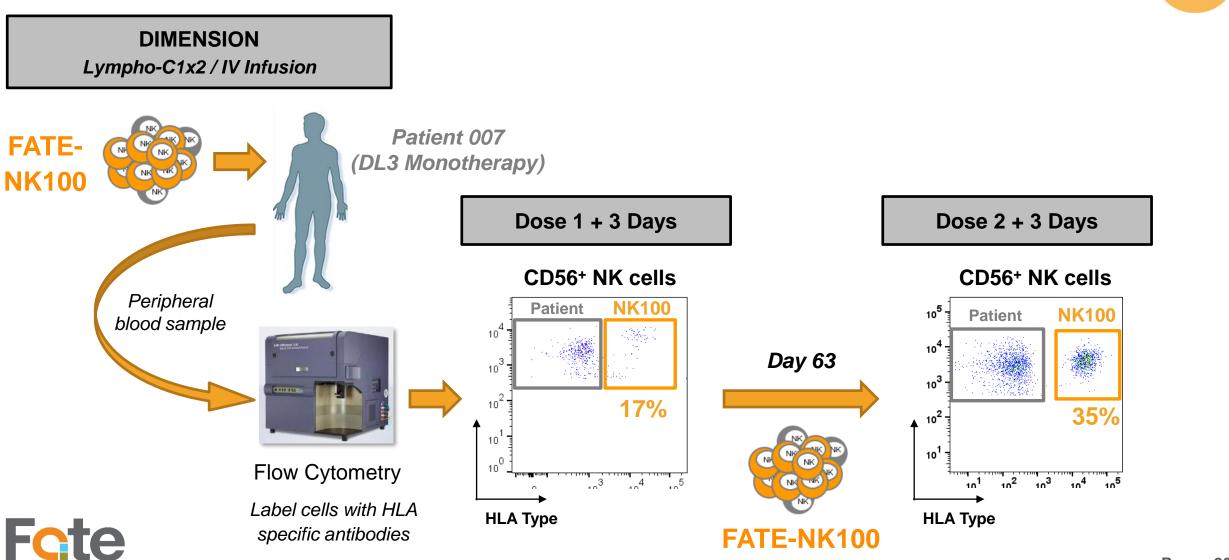
Enhanced Phenotype and Function In Vivo vs. Patient NK Cells



Day 7 In Vivo Persistence (Peripheral Blood) not detected in DIMENSION (N = 5)

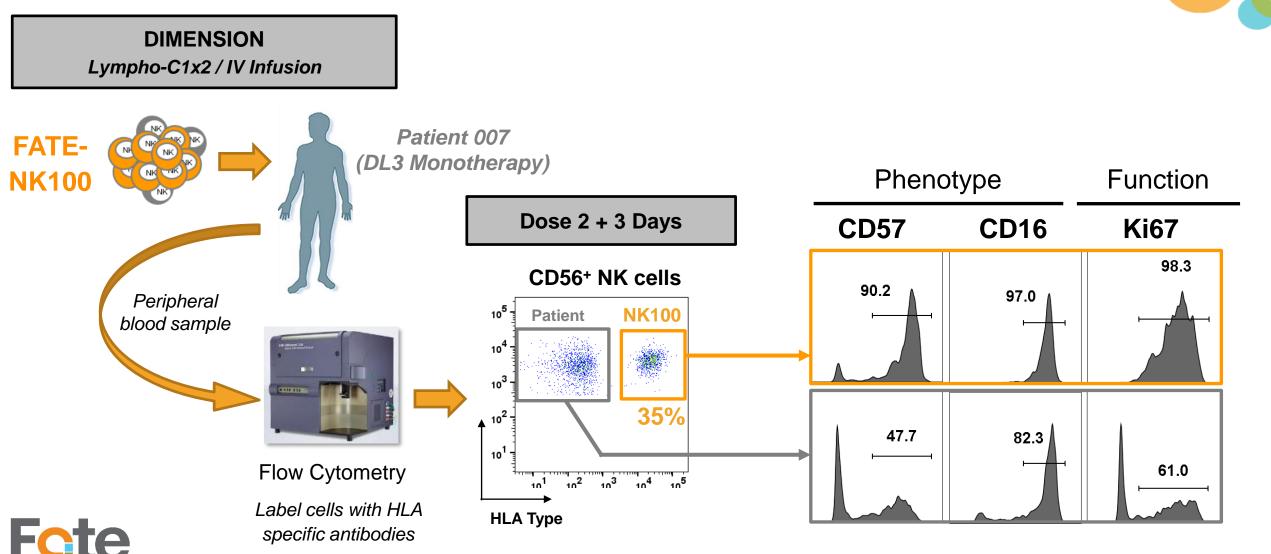
THERAPEUTICS

In Vivo Assessment of a Second Dose of FATE-NK100



THERAPEUTICS

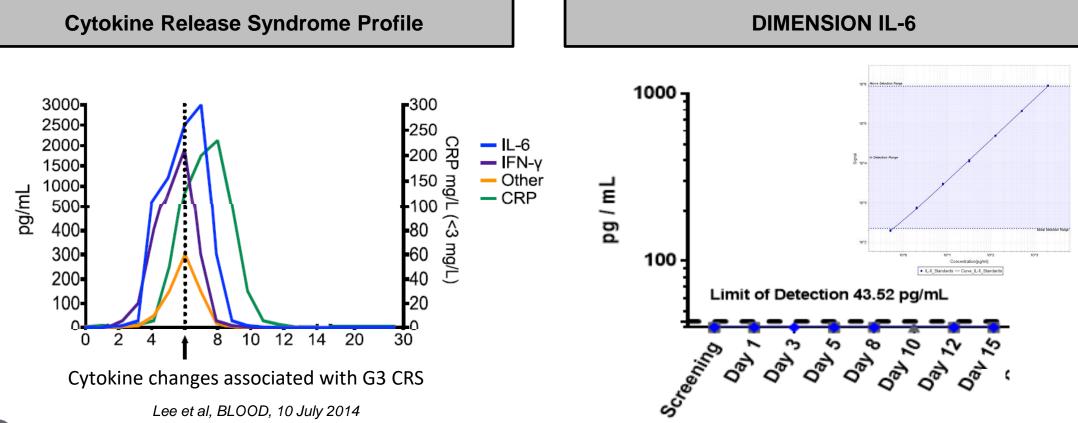
Dose 2 – Enhanced Phenotype and Function In Vivo vs. Patient NK Cells



Cytokine Analysis Suggests No Evidence of Cytokine Release Syndrome

DIMENSION

Lympho-C1x2 / IV Infusion





N = 4 (including 2 subjects receiving 2nd treatment cycle of FATE-NK100)

FATE-NK100: Translational Insights

Key Observations



- FATE-NK100 is highly purified NK cell product (>98% NK cells)
 - Has enhanced cytotoxicity in vitro vs. NK cell compartment of original healthy CMV⁺ donor
- Phenotype and function of FATE-NK100 is maintained *in vivo* post-infusion
 - Exhibits differentiated phenotype and enhanced proliferation vs. NK cell compartment of patient
- *In vivo* persistence of FATE-NK100 observed across all three studies
 - 6 of 7 subjects assessed in VOYAGE and APOLLO demonstrated persistence \geq 7 days
- Second treatment cycle of FATE-NK100 demonstrated persistence with retained function
 - Proof-of-concept for off-the-shelf, multi-dose NK cell treatment paradigm
- Cytokine screen suggests no evidence of CRS including after second treatment cycle



Clinical Observations Sarah A. Cooley, MD

Relapsed / Refractory AML



- 20,000 new AML cases / year in U.S.
 - Average age at diagnosis = 68
 - Overall 5-year survival = 27%
- Refractory disease following induction chemotherapy is common
 - CRs in 75% of patients <60 years old, but only 50% of patients >60 years old
- Relapse rates are high (60-70%) and treated with salvage chemotherapy
 - 10-15% achieve CR2 if early relapse (<12 months)
 - 40-60% of patients achieve CR2 if late relapse (>12 months)
 - IDH1 inhibitor ivosidenib approved with 25% CR (with DOR = 8 months), with 12% progressing to HCT
- Expect 10-15% CRs in r/r AML in first-in-human P1 study



Thol et al, Blood, 2015 ACS Cancer Facts and Figures 2018

VOYAGE Phase 1 Clinical Trial

Study Design: FATE-NK100 IV Monotherapy



VOYAGE: Relapsed / Refractory AML							
Pre-Conditioning	DL 3 (<i>n</i> =10)	>3-10 x 10 ⁷ / kg IV Monotherapy	Cytokine Support				
CY = 60 mg/kg x 2 day FLU = 25 mg/m2 x 5 da	(n=1)	>1-3 x 10 ⁷ / kg IV Monotherapy	IL-26M IU sc x 6 doses / 2 weeks				
Lympho-depleting	DL 1 (<i>n</i> =1)	1 x 10 ⁷ / kg IV Monotherapy					

Single treatment cycle only



VOYAGE Phase 1 Clinical Trial

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Study Design: FATE-NK100 IV Monotherapy

Safety Assessment

DLT is defined as <u>any</u> treatment related toxicity meeting one of the following criteria within 28 days of the FATE-NK100 infusion:

- Any non-hematologic, non-infectious grade 4 or 5 events
- Grade 3 adverse event of > 48 hours duration in the following organ systems: cardiac; pulmonary; hepatic; renal; or CNS

Efficacy Assessment

- CR is defined as leukemia clearance at <u>Day 42</u> (≤5% marrow blasts and no circulating peripheral blasts), recovery of neutrophils and platelets, and the absence of extramedullary disease.
- CRp is defined as leukemia clearance at <u>Day 42</u> (≤5% marrow blasts and no circulating peripheral blasts), recovery of neutrophils, but with incomplete recovery of platelets.



VOYAGE Phase 1 Clinical Trial



UNIVERSITY OF MINNESOTA

Dose-Escalation Clinical Data: FATE-NK100 IV Monotherapy

Dose Level ^a	Age / Sex	History / Most Recent Therapy & Outcome	% Blasts in Bone Marrow (Day 14)	NK100-related SAEs	Best Overall Response	Days on Study / Day 42 CR	
1	67 / M	AML complex cytogenetics PIF after 7+3, MEC, decitabine	48%	None	PD	23 / No	
2	62 / F	AML relapsed post-HiDAC Refractory to Clo/Cytarabine Refractory to ALT803-NK	Not detected	None	mLFS	32 / No	
2 ^{bc}	69 / M	AML complex cytogenetics, TP53 PIF after 7+3, ME	Not detected	None	mLFS	41 / No	
2	53 / M	MDS/AML complex cytogenetics PIF after 7+3, MEC	Not detected	None	mLFS d	Ongoing (Day 124) / No	
2	2 Two additional subjects to be enrolled; if no DLT, advance to DL3 (n=10)						



mLFS = morphologic leukemia-free state

^a Excludes first subject in DL1 (not evaluable).

^b FATE-NK100 dose failed to meet DL3, and subject was included in DL2.

^c Subject experienced DLT of acute renal failure <u>not</u> related to FATE-NK100.

^d mLFS with dysmegakaryopoiesis.

Data as reported by University of Minnesota. Final data are subject to change. Page - 34 -



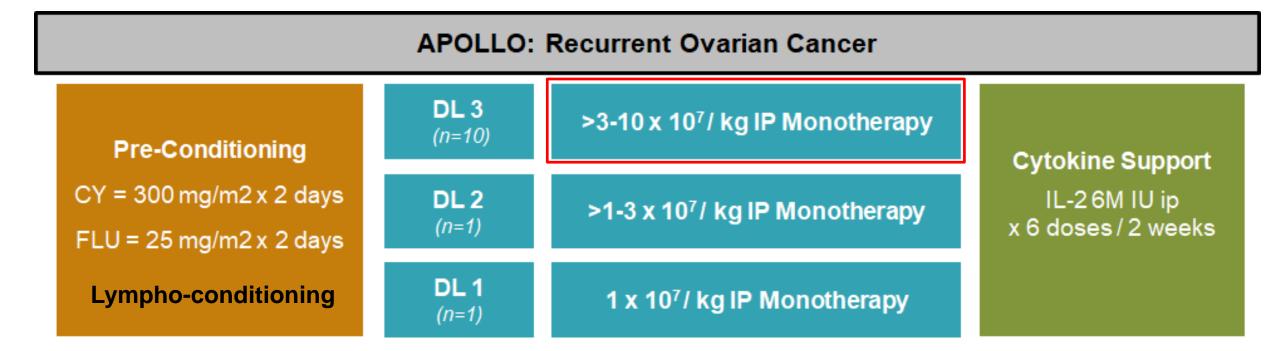
- 22,000 new cases / year and 14,270 deaths / year in the U.S.
 - Median age at diagnosis = 63
 - 61% diagnosed with advanced disease
 - Overall 5-year survival = 44% (27% with distant spread)
- Recurrence in most patients occurs within 12-18 months
 - If within 6 months, life expectancy is only 3-9 months
- Salvage therapy options have ORR 10-35% with PFS <8 months
 - Olaparib (PARP inhibitor) for platinum-resistant disease failing 3 lines (RR = 31%, PFS = 7 months)
 - Prior NK clinical experience: 4/14 (29%) PR with Time-to-Progression = 2 months



APOLLO Phase 1 Clinical Trial

Study Design: FATE-NK100 IP Monotherapy





Patients with stable disease or better at Day 28 may be considered for second treatment cycle on an individual basis





Study Design: FATE-NK100 IP Monotherapy

Safety Assessment

DLT is defined as any treatment emergent toxicity at least possibly related to FATE-NK100 or IL-2 meeting one of the following criteria within 28 days (14 days for ascites) of FATE-NK100 infusion:

- Grade 3 organ toxicity (cardiac, gastrointestinal, hepatic, pulmonary, renal/genitourinary, or neurologic) not pre-existing and lasting more than 72 hours.
- Any non-hematologic grade 4 toxicity
- Anemia or thrombocytopenia ≥ Grade 3, or neutropenia ≥ Grade 4, that persist at Day 28 despite use of growth factor support
- Any grade 3 or greater abdominal pain lasting more than three consecutive days and not controlled by standard analgesics
- Grade 3 or greater ascites within 14 days after FATE-NK100 administration in patients who had no ascites or Grade 1 ascites at enrollment and is not attributable to disease progression.

Efficacy Assessment



Assess the objective response rate (ORR) of this treatment at 28 days post-infusion.

APOLLO Phase 1 Clinical Trial



Masonic Cancer Center

Dose-Escalation Clinical Data: FATE-NK100 IP Monotherapy

Dose Level	Age / Sex	Prior Lines / Most Recent Therapy & Outcome	NK100-related SAEs	Best Overall Response	Days on Study	
1	64 / F	Prior Lines = 4 Topotecan – PD	None	PD	35	
2 *	71 / F	Prior Lines = 5 PARP inhibitor (12 cycles) – PD	Grade 3 (abdominal pain)	SD w/ decrease splenic mass	196	
2 ^a	68 / F	Prior Lines = 1 Carboplatin / Taxol (7 cycles) – PD	None	PD	33	
3	65 / F	Prior Lines = 6 Topotecan – PD	None	PD	35	
3	Dose Escalation and Dose Expansion (n=10)					

* Subject was treated with a second dose of FATE-NK100 on Day 50. PD was reported on Day 187.

^a FATE-NK100 dose failed to meet DL3, and subject was included in DL2.

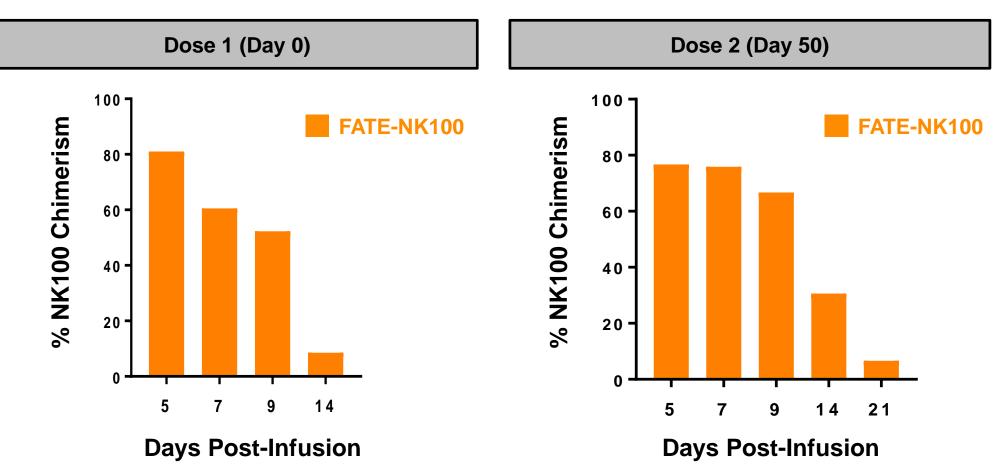
Data as reported by University of Minnesota. Final data are subject to change.



APOLLO Phase 1 Clinical Trial

Second Treatment Cycle Demonstrates Persistence

16186-UMN-002





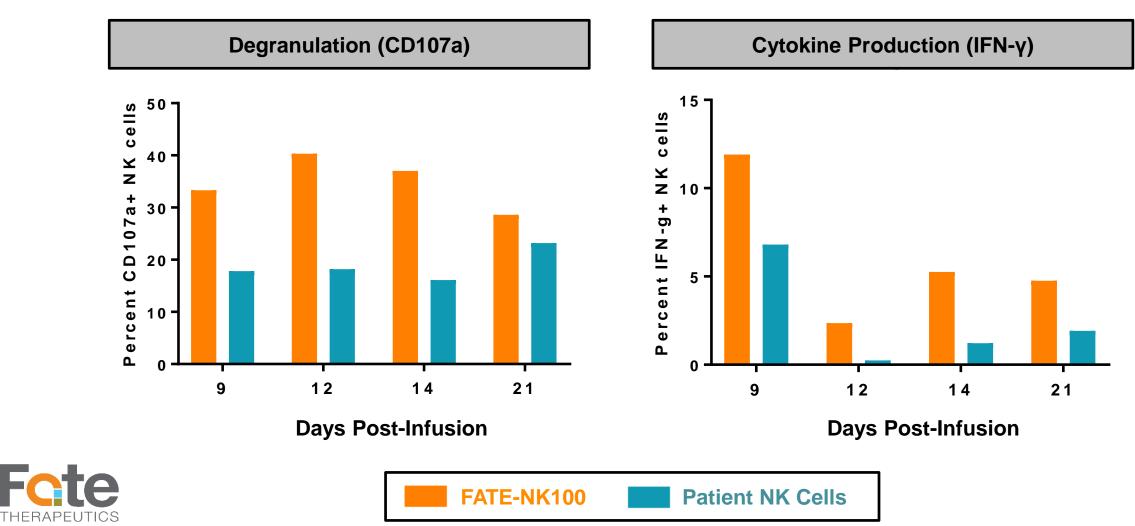
Ascites wash



APOLLO Phase 1 Clinical Trial

Dose 2 – Enhanced Functionality vs. Patient NK Cells

16186-UMN-002 – <u>Dose 2</u>



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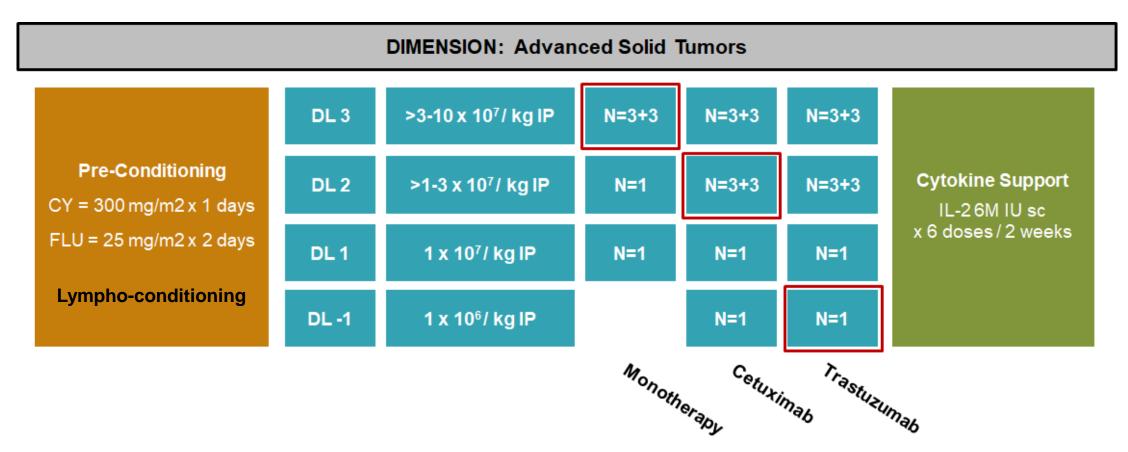
Advanced Solid Tumors

- DIMENSION is investigating FATE-NK100 as a monotherapy and in combination with trastuzumab or cetuximab in subjects with advanced solid tumors that have failed approved therapies
- Populations:
 - Monotherapy: any advanced solid tumor, "all-comer"
 - + cetuximab: any advanced EGFR1+ solid tumor
 - Colorectal cancer and head and neck squamous cell cancer must have failed cetuximab
 - + trastuzumab: any advanced HER2+ solid tumor
 - Gastric cancer must have failed trastuzumab
 - Breast cancer must have failed trastuzumab and either pertuzumab or ado-trastuzumab

First Combination of Donor-derived NK Cell Therapy with Tumor-Targeting Monoclonal Antibody Therapy for Solid Tumors



Study Design: Dose-Escalation FATE-NK100 IV Monotherapy + mAb Combination



Patients with stable disease or better at Day 28 may be considered for second

treatment cycle on an individual basis

mAb administration = Day -2 and Day 8

Study Design: Dose-Escalation FATE-NK100 IV Monotherapy + mAb Combination

Safety Assessment

DLT is defined as any treatment-related toxicity meeting one of the following criteria within 28 days of the FATE-NK100 infusion:

- Any non-hematologic AE Grade \geq 4.
- Any non-hematologic AE Grade 3 of >48 hours duration.
- Anemia or thrombocytopenia ≥ Grade 3, or neutropenia ≥ Grade 4, that persists at Day 29 despite use of growth factor support.

Efficacy Assessment

Assess the objective-response rate (ORR) defined as the proportion of patients who achieve partial response (PR) or complete response (CR) for target lesions per RECIST 1.1 at any time on study.



Dose-Escalation Clinical Data: FATE-NK100 IV Monotherapy

Dose Level	Age / Sex	Diagnosis	Prior Lines / Most Recent Therapy & Outcome	NK100-related SAEs	Best Overall Response / % Change - Target Lesion	Days on Study
1	59 / M	Head & Neck	Prior Lines = 4 Experimental (virus) – PD	None	PD / 1% (non-target PD)	43
2	36 / F	Sinonasal	Prior Lines = 4 Tazemetostat – PD	None	SD / 0%	60
3*	66 / F	Uterine	Prior Lines = 2 Docetaxel (Taxotere) / Carboplatin – PD	None	SD / 0%	149#
3*	54 / F	Colorectal	Prior Lines = 3 5-FU / Bevacizumab / Irinotecan – PD	None	SD / +18% (non-target PR)	94 #
3	30 / M	Cardio-Esophageal Junction Adenocarcinoma	Prior Lines = 3 Idasanutlin – PD	None	PD/+24%	34
3	58 / F	Renal	Prior Lines = 2 Carboplatin / Etoposide – Mixed Response	Pending		
3	52/M	Melanoma	Prior Lines = 3 Pembrolizumab – n/a		Pending	



* Subject was treated with a second dose of FATE-NK100

Ongoing

As of October 22, 2018 data cutoff. Database is not locked and final data are subject to change.

Case Study I: Clinical Observations

- 66 y/o female with platinum-resistant uterine cancer
- Received two prior lines of therapy
 - Paclitaxel / Carboplatin Not evaluable
 - Docetaxel (Taxotere) / Carboplatin PD
- Treated with FATE-NK100 at DL3 (monotherapy); received a second dose on Day 63
 - No FATE-NK100 related SAEs or AEs
 - No evidence of CRS
- Best RECIST response: SD
 - Days on study = 149 (ongoing)





Dose-Escalation Clinical Data: FATE-NK100 IV Combination with mAb

Dose Level / mAb	Age / Sex	Diagnosis	Prior Lines / Most Recent Therapy & Outcome	NK100-related SAEs	Best Overall Response / % Change – Target Lesion	Days on Study
-1 cetuximab	53/F	Colorectal	Prior Lines = 4 Trifluridine / Tipiracil – PD	None	PD / 38%	34
1 cetuximab	72/M	Urothelial	Prior Lines = 5 Carboplatin / Gemcitabine – PD	None	PD / 82%	38
2 cetuximab	72/F	Endometrial	Prior Lines = 2 Carboplatin / Paclitaxel – PD	Pending		
2 cetuximab	61 / M	NSCLC	Prior Lines = 6 Docetaxel / Ramucircumab – PD	Pending		
-1 trastuzumab	75/F	Recurrent Gastric	Prior Lines = 5 Trastuzumab Emtansine – PD		Pending	

As of October 22, 2018 data cutoff. Database is not locked and final data are subject to change.



FATE-NK100

Summary of Key Safety & Clinical Data Across Dose-Escalation

	FATE-NK100 Related		Symptoms of CRS /	
	DLTs	SAEs	Neurotoxicity	Clinical Activity ¹²
VOYAGE	None	None	None	3 of 4
APOLLO	None	Grade 3 (1)	None	1 of 4
DIMENSION				
Monotherapy	None	None	None	3 of 5
Cetuximab	None	None	None	0 of 1
Trastuzumab	Pending			

¹ For VOYAGE, includes mLFS; for APOLLO and DIMENSION, includes best overall response of stable disease or better.



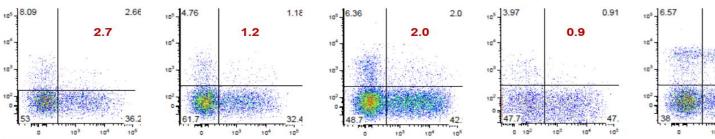
² Excludes DL-1 for DIMENSION combination with mAb.

As of October 22, 2018 data cutoff. Database is not locked and final data are subject to change.

NK Cell Product Franchise Bob Valamehr, PhD, Chief Development Officer

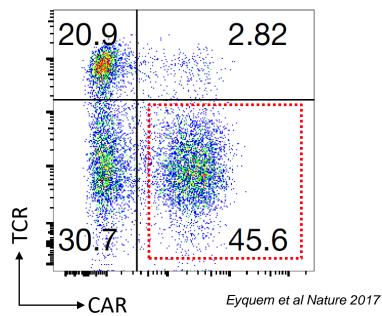
Heterogeneity of Healthy Related Donor Cells **Heterogeneity of Engineering Blood Composition** 104 NK cells NKT cells NK cells NK cells NKT cells NKT cells VK cells NKT cells NK cells NKT cells 1.17 20.9 0.65 2.64True T cells True T cells rue T cell rue T 81.3 NonNK&T cells 77.8 NonNK&T cells 75.4 NonNK&T cells NonNK&T cells NonNK&T cells 79 12.7 11.4 10.7 0 102 0 102

CD57 / NKG2C NK Cell Compartment



NKG2C

CAR Targeted into TRAC Locus using CRISPR

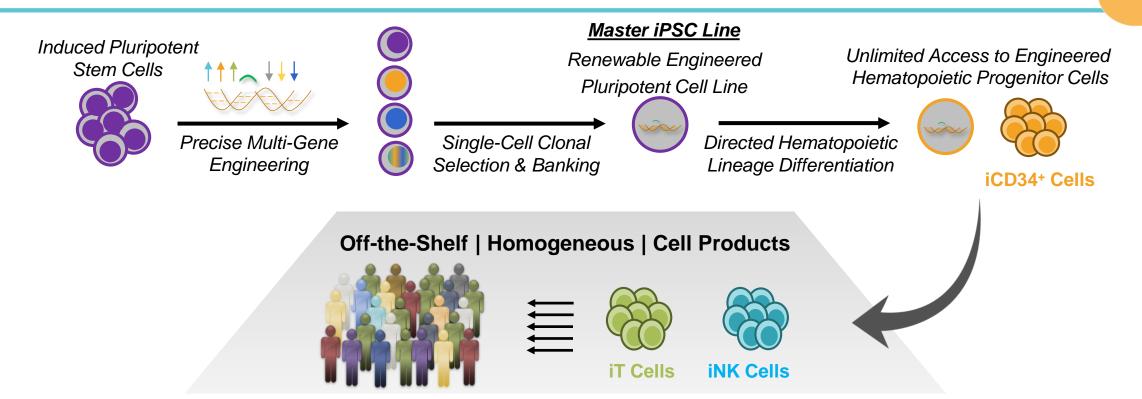


25.5

25.5

Our iPSC Product Platform

Off-the-Shelf Cell Products Derived From Clonal Master iPSC Lines



Does not require patient-sourced cells Off-the-shelf production of cells Multi-dosed enabling Consistent, homogeneous and reliable product forms Unprecedented scalability Cost-effective

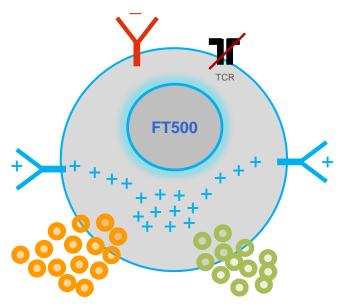


Addresses Critical Limitations of Patient-Sourced Cellular Therapies

FT500 Universal, Off-the-Shelf NK Cell Product

Combination with Checkpoint Blockade Therapy

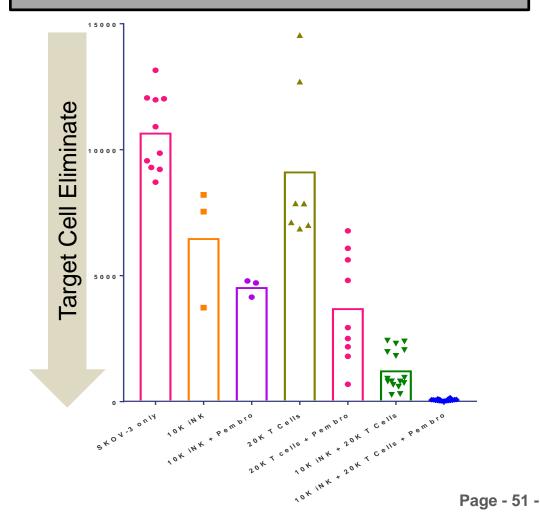
Multi-faceted Functionality to Augment CPB Therapy



IND Submitted

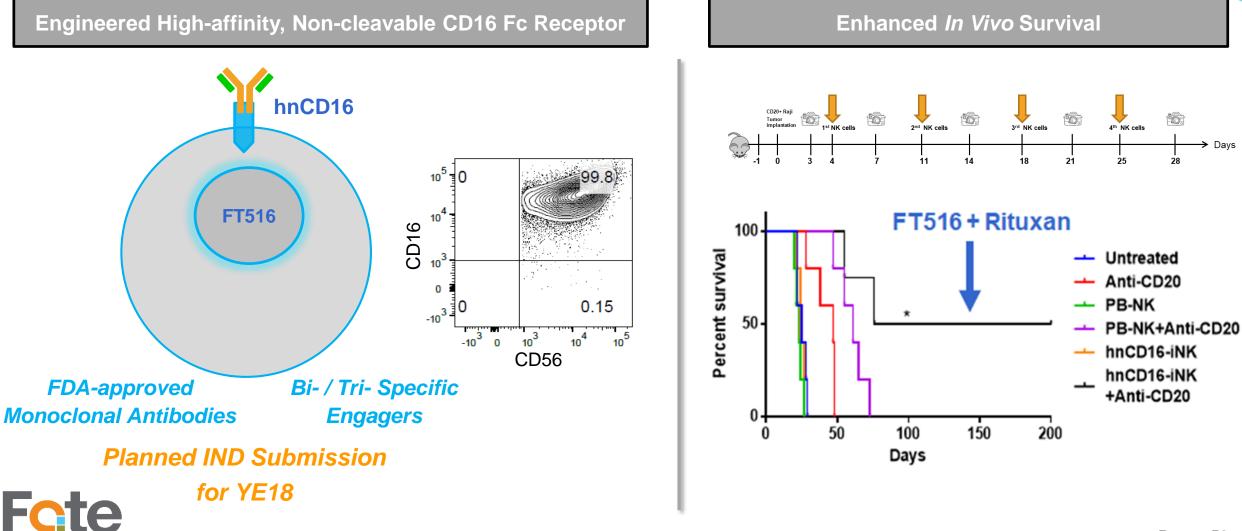
- Activating Receptors to Seek and Eliminate Stressed Cells Independent of a Single Unique Antigen
- Inhibitory Receptors "Check" NK
 Cell Activation, Prevent
 Cytotoxicity Towards Healthy Cells
- ✓ Unlike T cells, NK cells do not elicit GvHD
- Secretion of proinflammatory cytokines and chemokines to recruit and activate the adaptive immune system

3-D Ovarian Cancer Tumor Model



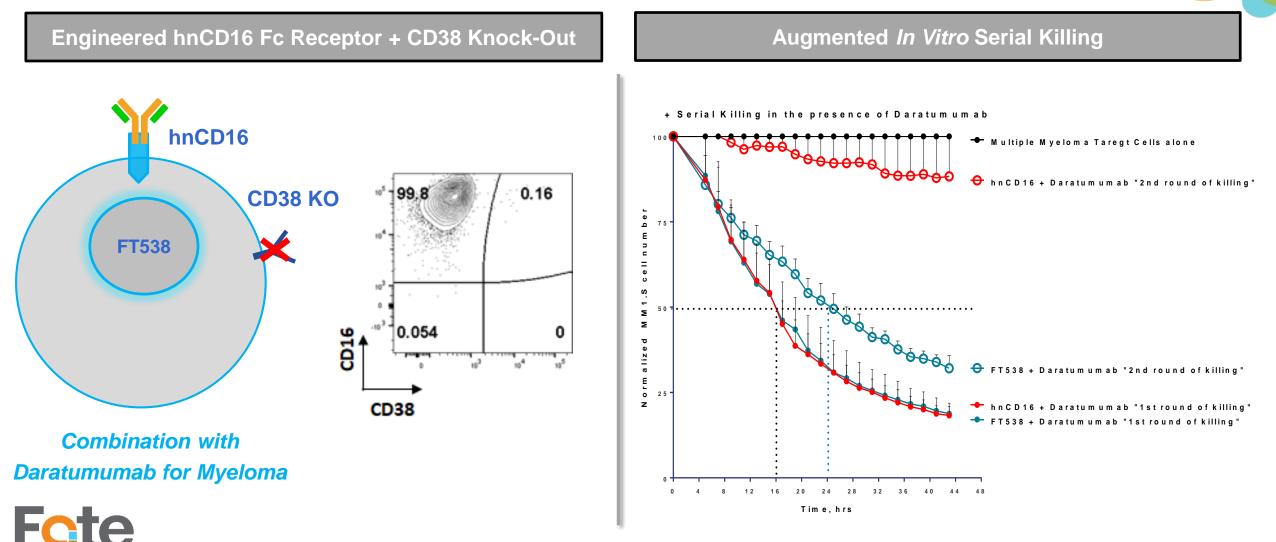
FT516 Universal, Off-the-Shelf hnCD16 NK Cell Product

Enhanced ADCC for Combination with Monoclonal Antibody Therapy



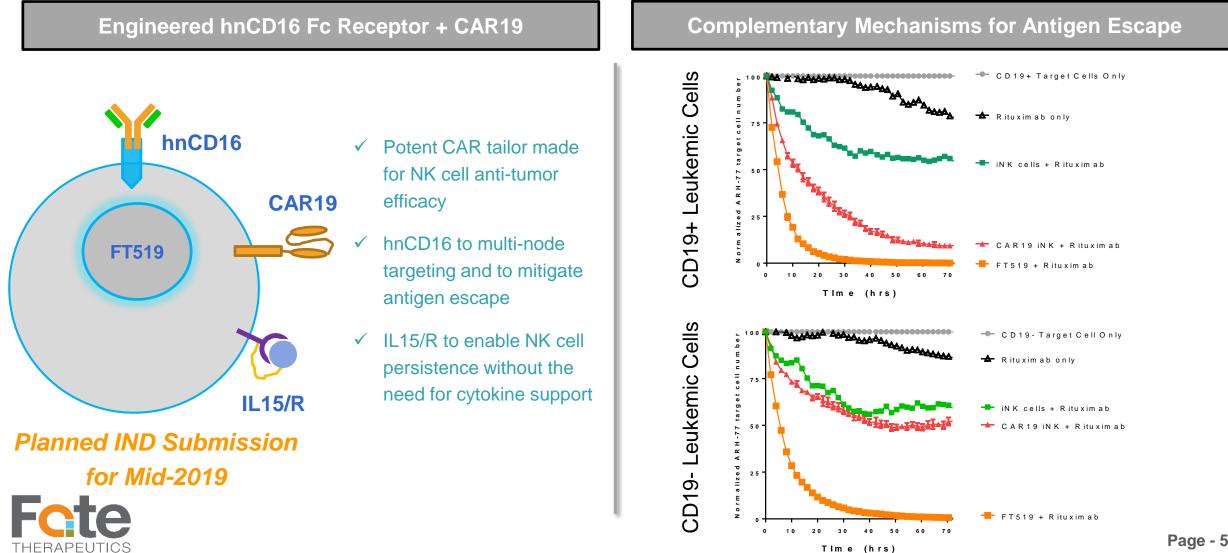
FT538 Universal, Off-the-Shelf hnCD16 / CD38- NK Cell Product

Enhanced ADCC with Elimination of Fratricide for Daratumumab Combo



FT519 Universal, Off-the-Shelf hnCD16 / CAR19 NK Cell Product

Dual-Targeting for Antigen Escape



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Coming Soon...ASH Annual Meeting

FATE Investor Event: Friday, November 30



American Society of Hematology

Helping hematologists conquer blood diseases worldwide



University of Minnesota Driven to Discover∺





Dan Kaufman, MD PhD



Memorial Sloan Kettering Cancer Center

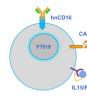
Michel Sadelain, MD PhD



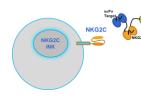
FT500 iPSC-Derived NK Cells and **Anti-PD1 Antibody Synergize** to Enhance T-Cell Cytokine and Cytolytic Responses Against Multiple Tumors



FT538 CD38 Deficient, CD16 Engineered NK Cells Exhibit
 ^a Enhanced Antibody Dependent Cellular Cytotoxicity without
 NK Cell Fratricide to Augment Anti-Myeloma Immunity in
 Combination with Daratumumab

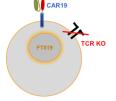


FT519 Off-the-Shelf Natural Killer Cells with Multi-Functional
Engineering Using a Novel Anti-CD19 Chimeric Antigen
Receptor Combined with Stabilized CD16 and IL15 Expression
to Enhance Directed Anti-Tumor Activity



iPSC-Derived NK Cells Genetically Modified to
 Express NKG2C/DAP12 Mediate Potent Function
 When Targeted through an NKG2C/IL15/CD33 Tri Specific Killer Engager





FT819 Pluripotent Cell-Derived Off-the-Shelf TCR-Less CAR-Targeted Cytotoxic T Cell
 Therapeutic for the Allogeneic Treatment of B Cell Malignancies

Concluding Remarks Scott Wolchko, President & CEO

NK Cell Franchise

FATE-NK100 to FT500 Series

- Therapeutic strategy: early, often, combinations
 - Supported by current safety and clinical activity observations with FATE-NK100
- Significant learnings from FATE-NK100 clinical trials are accruing to franchise
 - > Gaining valuable insights: conditioning, immune response, multi-dosing, tumor types
 - > Developing key relationships with top medical investigators and clinical sites
- Off-the-shelf, iPSC-derived NK cell paradigm is disruptive
 - Plan to advance FATE-NK100 based on compelling clinical responses observed in dose-expansion
 - > FT500 and FT516 have the potential to step into the FATE-NK100 clinical footprint
- Completed adventitious agents testing of iPSC Master Cell Bank for FT500 as requested by FDA
 - > No adventitious agents were detected in *in vivo* and *in vitro* testing
 - Response to FDA has been submitted



Feite Therapeutics

Better Cells For Better Therapies™