Programmed Cellular Immunotherapies

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

January 2021
Forward-Looking Statements

This presentation contains “forward-looking statements” within the meaning of the Private Securities Litigation Reform Act of 1995, including statements regarding the Company’s research and development activities and its progress, plans and timelines for its manufacture, preclinical development and clinical investigation of its product candidates, the timing for the Company’s receipt of data from its clinical trials and preclinical studies, the Company’s clinical development and regulatory strategy, and the therapeutic and market potential of the Company’s product candidates. These and any other forward-looking statements in this presentation are based on management’s current expectations of future events and are subject to a number of risks and uncertainties that could cause actual results to differ materially and adversely from those set forth in or implied by such forward-looking statements. These risks and uncertainties include, but are not limited to, the risk that results observed in prior studies of its product candidates will not be observed in ongoing or future studies involving these product candidates, the risk of a delay in the initiation of, or in the enrollment or evaluation of subjects in, any clinical studies, and the risk that the Company may cease or delay manufacture, or preclinical or clinical development, of any of its product candidates for a variety of reasons (including regulatory requirements, difficulties in manufacturing or supplying the Company’s product candidates, and any adverse events or other negative results that may be observed during preclinical or clinical development). These statements are also subject to other risks and uncertainties as further detailed in the Company’s most recently filed periodic report, and subsequent periodic reports filed by the Company, under the Securities Exchange Act of 1934, as amended, any of which could cause actual results to differ materially from those contained in or implied by the forward-looking statements in this presentation. The Company is providing the information in this presentation as of the date hereof and does not undertake any obligation to update any forward-looking statements contained in this presentation unless required by applicable law.
A Remarkable 2-Year Journey of Firsts

Building the Leading Off-the-Shelf NK Cell Cancer Immunotherapy Company

FT500

Best-in-Class Lymphoma

Best-in-Class Myeloma

Solid Tumors

July 2018

1st IND submission
87 employees
$78M in cash

Sept 2020

9 Cleared INDs
250+ employees
$930M+ in cash

• FT516 + mAbs
• FT538 + mAbs
• CAR MICA/B
• ONO Product 2
• Janssen Targets

FT516

FT596

FT538

FT576

On an as adjusted basis to include January 2021 common stock offering
Changing the Game in Cell Therapy

Necessary Hurdles to Overcome to Change the Game

1. **Multiplexed Engineering.** Embed multiple elements of synthetic biology to deliver multiple mechanisms of action, increase therapeutic efficacy and reduce toxicity.

2. **Uniform Product.** Minimize sources of variability (cell source, engineering, production, etc.) to consistently demonstrate identity, purity and potency of cell product.

3. **Mass Production.** Repeatedly operate a GMP manufacturing process that yields hundreds to thousands of doses in single batch to support multi-dose regimens, cost-effective treatment and widespread product availability.

4. **Off-the-shelf Availability.** Cryopreserve cell products in a fill / finish formulation that supports long-term stability, inventory build, and thaw-infuse administration to patients.

5. **Patient Accessibility.** Greatly simplify logistics to enable treatment of many patients on-demand, without delay, and with high convenience.
Changing the Game in Cell Therapy
Universal, Off-the-Shelf Cell Products Derived from Renewable Master Cell Lines

<table>
<thead>
<tr>
<th>Key Features</th>
<th>Cell Therapy 1.0 and 2.0</th>
<th>Cell Therapy 3.0</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cell Source</td>
<td>Patient and Donor Cells</td>
<td>Renewable Master Cell Line</td>
</tr>
<tr>
<td>Genetic Engineering</td>
<td>Random &amp; Variable</td>
<td>Uniform &amp; Complete</td>
</tr>
<tr>
<td>Characterization</td>
<td>Imprecise</td>
<td>Well-defined</td>
</tr>
<tr>
<td>Product Identity</td>
<td>Heterogeneous</td>
<td>Homogeneous</td>
</tr>
<tr>
<td>Manufacturing</td>
<td>Low Yield-to-Cell Dose Ratio</td>
<td>High Yield-to-Cell Dose Ratio</td>
</tr>
<tr>
<td>Packaging</td>
<td>Fresh / Short Shelf Life</td>
<td>Cryopreserved / Long Shelf Life</td>
</tr>
<tr>
<td>Dosing</td>
<td>Single Dose</td>
<td>Multiple Doses</td>
</tr>
<tr>
<td>Delivery</td>
<td>Complex Logistics</td>
<td>Off-the-Shelf</td>
</tr>
<tr>
<td>Overall Paradigm</td>
<td>Process-centric</td>
<td>Product-centric</td>
</tr>
</tbody>
</table>
Unique Advantages of Human iPSCs

Single-cell Isolation, Characterization & Selection

A Single Human Induced Pluripotent Stem Cell (iPSC)
A renewable source for making cell products

Unlimited Clonal Expansion
Multiplexed Engineering
Extensive Characterization

Single iPSC Clone

Potential to Differentiate into 200+ Cell Types
Master Cell Lines and Banks
Uniform in Composition

Fate Therapeutics’ iPSC product platform is supported by an IP portfolio of 300+ issued patents and 150+ pending patent applications
Unique Advantages of Human iPSCs
Creating a Clonal Master Engineered iPSC Line

Cell Population Engineering

- 1 edit
- 2 edits
- 3 edits
- 4 edits

Correctly-edited
Incorrectly-edited

Single-cell iPSC Isolation, Characterization and Selection

- Determination of copy number
- Confirmation of genomic stability
- Confirmation of transgene integration site
- Confirmation of pluripotency and propensity to differentiate
- Confirmation of highly functioning cells
- Confirmation of uniform transgene expression and enhanced function
- A myriad of additional safety and efficacy analyses

Clonal Master Engineered iPSC Line

A Renewable Cell Source for Mass Production of Engineered Immune Cells
The Making of Bona Fide NK Cells from Clonal Master Engineered iPSC Bank

Robust cGMP Process

- iPSCs
  - Day 0
  - > 10^6 iPSCs

- iCD34s
  - Day 10
  - Homogeneous cell product
  - > 1 million-fold expansion

- iNKs
  - Day 44a
  - 100s-1,000s doses per campaign
  - Low-cost per dose cGMP production
  - Cryopreserved
  - High post-thaw viability
  - > 10^12 iNKs

a Rezner et al. ASH Annual Meeting 2020
Off-the-Shelf, iPSC-derived NK Cell Cancer Immunotherapy Franchise
Systematic Build of Industry-Leading iPSC-derived NK Cell Product Pipeline

<table>
<thead>
<tr>
<th>Clonal Master iPSC Line</th>
<th>Synthetic Biology</th>
<th>FT500</th>
<th>FT516</th>
<th>FT596</th>
<th>FT538</th>
<th>FT576</th>
<th>FT536</th>
</tr>
</thead>
<tbody>
<tr>
<td>Multi-faceted Innate Immunity</td>
<td>✔️</td>
<td>✔️</td>
<td>✔️</td>
<td>✔️</td>
<td>✔️</td>
<td>✔️</td>
<td>✔️</td>
</tr>
<tr>
<td>+ High-Affinity, Non-cleavable CD16</td>
<td>Augment mAb therapy</td>
<td>✔️</td>
<td>✔️</td>
<td>✔️</td>
<td>✔️</td>
<td>✔️</td>
<td>✔️</td>
</tr>
<tr>
<td>+ IL-15 Receptor Fusion</td>
<td>Enhance NK cell function</td>
<td>✔️</td>
<td>✔️</td>
<td>✔️</td>
<td>✔️</td>
<td>✔️</td>
<td>✔️</td>
</tr>
<tr>
<td>+ CAR Insertion</td>
<td>Target tumor antigens</td>
<td>CD19</td>
<td>BCMA</td>
<td>MICA/B</td>
<td>✔️</td>
<td>✔️</td>
<td>✔️</td>
</tr>
<tr>
<td>+ CD38 Knock-out</td>
<td>Enhance metabolic fitness</td>
<td>✔️</td>
<td>✔️</td>
<td>✔️</td>
<td>✔️</td>
<td>✔️</td>
<td>✔️</td>
</tr>
</tbody>
</table>

# of Synthetic Elements

|          | 0 | 1 | 3 | 3 | 4 | 4 |

Universal, Off-the-Shelf NK Cell Cancer Immunotherapy Pipeline
Clinical experience supports the transformative potential of iPSC Product Platform

- **Experience**
  - 35+ patients dosed with 150+ doses of iPSC-derived NK cells (FT500, FT516, FT596, FT538)
  - Treated diseases include lymphoma, AML and solid tumors

- **Safety**
  - Demonstrated ability to administer up to 6 doses safely in an outpatient setting
  - No CRS, ICANS or GvHD at dose levels ≤ 300M cells / dose
  - No evidence of anti-product T- or B-cell mediated immunogenicity

- **Activity**
  - Clear evidence of anti-tumor activity at initial low doses
  - Patient responses achieved in heavily pre-treated patients with relapsed / refractory disease
B-cell Malignancy Franchise
FT516: hnCD16 NK Cell Product Candidate

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

- CD16 is an activating receptor expressed on NK cells
  - Mediates antibody-dependent cellular cytotoxicity (ADCC), a potent anti-tumor mechanism by which NK cells recognize, bind and kill antibody-coated cancer cells
- CD16 occurs in two variants: high (158V) or low (158F) affinity for the Fc domain of IgG antibodies
  - Only ~15% of patients are homozygous for 158V
  - Numerous clinical studies with FDA-approved tumor-targeting antibodies have demonstrated that patients homozygous for 158V have improved clinical outcomes
- The endogenous NK cell compartment of a cancer patient is significantly impaired
  - Absolute NK cell numbers are low
  - CD16 expression levels are low and shedding inhibits ADCC
  - Tumor suppressive mechanisms contribute to NK cell exhaustion

How to bring the 158V CD16 NK cell experience to all patients?
FT516: hnCD16 NK Cell Product Candidate

Our Novel High-Affinity, Non-Cleavable CD16a Fc Receptor for Enhanced ADCC

Proprietary High-affinity, Non-cleavable CD16a (hnCD16) Fc Receptor for Enhanced ADCC

Resistance to Activation-induced Shedding as Compared to Healthy Donor NK Cells

Prolonged Survival In Vivo as Compared to Healthy Donor NK Cells

Zhu et al. Blood 2020
FT516-101: B-Cell Lymphoma in Combination with Rituximab

Phase 1 Study Design: Multiple Doses over Multiple Cycles

Cyclophosphamide: 500 mg/m² IV x 3 days
Fludarabine: 30 mg/m² IV x 3 days
IL-2: 6M units sc with each FT516 dose

Up to 6 doses of FT516

Regimen B – Rituximab Combination

- Relapsed / refractory B-cell lymphoma
- Dose Escalation: 30M, 90M, 300M, 900M cells per dose + mAb
- Dose Expansion: up to 15 subjects
## FT516-101: B-Cell Lymphoma in Combination with Rituximab

**Phase 1 Study: Patient Baseline Characteristics**

<table>
<thead>
<tr>
<th>FT516 Dose Cohort</th>
<th>Subject #</th>
<th>Age / Sex</th>
<th>Lymphoma Type</th>
<th>Prior Systemic Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>90M cells</td>
<td>2005</td>
<td>50 / M</td>
<td>DLBCL</td>
<td>1) R-CHOP</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2) Flu/Cy → Yesacarta</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>3) R-ICE</td>
</tr>
<tr>
<td></td>
<td>2006</td>
<td>65 / M</td>
<td>DLBCL</td>
<td>1) R-CEOP/MTX</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2) R-DHAX</td>
</tr>
<tr>
<td></td>
<td>2007</td>
<td>62 / M</td>
<td>DLBCL</td>
<td>1) R-CHOP</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>(Double-Hit)</td>
<td>2) R-EPOCH</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>3) Flu/Cy → R + Yesacarta</td>
</tr>
<tr>
<td>300M cells</td>
<td>2008*</td>
<td>68 / M</td>
<td>FL</td>
<td>1) R</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2) R-Bendamustine</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>3) R</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>4) R-CHOP</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Regimen</th>
<th>Best Response</th>
<th>DoR</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>PR</td>
<td>1 month</td>
</tr>
<tr>
<td></td>
<td>CR</td>
<td>8 months</td>
</tr>
<tr>
<td></td>
<td>PD</td>
<td>NA</td>
</tr>
<tr>
<td></td>
<td>PD</td>
<td>NA</td>
</tr>
<tr>
<td></td>
<td>CR</td>
<td>&lt;2 months</td>
</tr>
<tr>
<td></td>
<td>UNK</td>
<td>UNK</td>
</tr>
<tr>
<td></td>
<td>PD</td>
<td>PD</td>
</tr>
<tr>
<td></td>
<td>CR</td>
<td>2.5 months</td>
</tr>
<tr>
<td></td>
<td>CR</td>
<td>UNK</td>
</tr>
<tr>
<td></td>
<td>CR</td>
<td>UNK</td>
</tr>
<tr>
<td></td>
<td>PR</td>
<td>3 years</td>
</tr>
<tr>
<td></td>
<td>PR</td>
<td>13 months</td>
</tr>
<tr>
<td></td>
<td>CR</td>
<td>4 months</td>
</tr>
</tbody>
</table>

**DLBCL = Diffuse Large B-Cell Lymphoma; FL = Follicular Lymphoma; R = Rituximab; CHOP = cyclophosphamide, doxorubicin, vincristine, prednisone; ICE = ifosfamide, carboplatin, etoposide; CEOP = cyclophosphamide, etoposide, vincristine, prednisone; MTX = methotrexate; DHAX = dexamethasone, cytarabine, oxaliplatin; EPOCH = etoposide, prednisone, vincristine, cyclophosphamide, doxorubicin; Flu/Cy = fludarabine/cyclophosphamide conditioning; CR = Complete Response; PR = Partial Response; PD = Progressive Disease; NA = Not Applicable; UNK = Unknown; DoR = Duration of Response; * Information on prior systemic therapies per investigator communication (data not entered into database)

**Note:** As of November 16, 2020 database entry. Data subject to cleaning and source document verification.
**FT516-101: B-Cell Lymphoma in Combination with Rituximab**

*Phase 1 Safety, Tolerability and Protocol-Defined Response*

<table>
<thead>
<tr>
<th>FT516 Dose Cohort</th>
<th>Subject #</th>
<th>Prior Systemic Therapy</th>
<th>Relapsed / Refractory</th>
<th>Bridging Therapy</th>
<th>FT516 Doses</th>
<th>FT516-Related Safety</th>
<th>Immunogenicity¹</th>
<th>Post-C2 Response²</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>With Rituximab</td>
<td></td>
<td></td>
<td></td>
<td>DLT</td>
<td>Any Grade CRS</td>
<td>Any Grade ICANS</td>
</tr>
<tr>
<td>90M cells</td>
<td>2005</td>
<td>2</td>
<td>Refractory</td>
<td>No</td>
<td>6</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td></td>
<td>2006</td>
<td>2</td>
<td>Relapsed</td>
<td>No</td>
<td>6</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td></td>
<td>2007</td>
<td>3</td>
<td>Relapsed</td>
<td>No</td>
<td>6</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>300M cells</td>
<td>2008</td>
<td>4</td>
<td>Relapsed</td>
<td>No</td>
<td>6</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
</tbody>
</table>

**AE** = adverse event; **DLT** = Dose limiting toxicity; **CRS** = Cytokine release syndrome; **ICANS** = Immune effector cell-associated neurotoxicity syndrome; **GvHD** = Graft-versus-Host Disease; **CR** = Complete response; **PR** = Partial Response; **PD** = Progressive Disease

¹ Host-vs-product alloreactivity measured Day 15 and Day 29 of Cycles 1 and 2 for T cells, and Day 29 of Cycles 1 and 2 for B cells.

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

Dose escalation ongoing at 300M cells / dose

Note: As of November 16, 2020 database entry. Data subject to cleaning and source document verification.
FT516-101: B-Cell Lymphoma in Combination with Rituximab

Initial Clinical Observations

- Up to 6 doses of FT516 were well-tolerated
  - No events of any grade of CRS, ICANS, or GvHD
  - No FT516-related Grade 3 or greater adverse events
  - No evidence of anti-product T- or B-cell mediated immunogenicity

- Objective response at post-C2 assessment observed in 3 of 4 patients treated with ≥ 90 million cells / dose
  - CR – 2005 (90M): relapsed following CAR19 T-cell therapy, and refractory to last prior rituximab regimen (RR)
  - PR – 2006 (90M): refractory to 1L RR, and 2L RR showed PR of minimal duration
  - CR – 2008 (300M): four prior RR with progressively shorter durations of response

- Clear evidence that FT516 can drive responses in relapsed / refractory patients
  - All patients previously treated with at least 2 prior rituximab-containing regimens
  - 3 of 4 patients have disease refractory to at least 1 prior rituximab-containing regimen
  - One patient had relapsed after achieving CR of <3 months duration on CAR19 T-cell therapy

- Clinical data strongly suggest that proprietary hnCD16 Fc receptor can effectively synergize with and enhance the MOA of tumor-targeted antibodies
FT596: Multi-antigen Targeted CAR19 NK Cell Product Candidate
Potential Best-in-Class Cell-based Cancer Immunotherapy for B-cell Malignancies

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

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

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

**IL-15RF**: Interleukin-15 receptor fusion, a potent cytokine complex that promotes survival, proliferation and trans-activation of NK cells and CD8 T cells
FT596-101: Phase 1 Dose Escalation Schema
Parallel Escalation of Single-dose Mono and mAb Combo in BCL and CLL

**B-cell Malignancies**

<table>
<thead>
<tr>
<th></th>
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</thead>
<tbody>
<tr>
<td>A: 9 x 10^6 cells Day 1</td>
<td>B1: 9 x 10^6 cells Day 1</td>
<td>B2: 9 x 10^6 cells Day 1</td>
<td>B3: 9 x 10^6 cells Day 1</td>
</tr>
<tr>
<td>A: 3 x 10^8 cells Day 1</td>
<td>B1: 3 x 10^8 cells Day 1</td>
<td>B2: 3 x 10^8 cells Day 1</td>
<td>B3: 3 x 10^8 cells Day 1</td>
</tr>
<tr>
<td>A: 9 x 10^7 cells Day 1</td>
<td>B1: 9 x 10^7 cells Day 1</td>
<td>B2: 9 x 10^7 cells Day 1</td>
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<td>B1: 3 x 10^7 cells Day 1</td>
<td>B2: 3 x 10^7 cells Day 1</td>
<td>B3: 3 x 10^7 cells Day 1</td>
</tr>
<tr>
<td>n = 3- 6/cohort</td>
<td>n = 3- 6/cohort</td>
<td>n = 3- 6/cohort</td>
<td>n = 3- 6/cohort</td>
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</table>

**Follicular Lymphoma**

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<tbody>
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<td>B1: 9 x 10^6 cells Day 1</td>
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<td>B3: 9 x 10^6 cells Day 1</td>
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<td>B3: 3 x 10^8 cells Day 1</td>
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<td>B1: 9 x 10^7 cells Day 1</td>
<td>B2: 9 x 10^7 cells Day 1</td>
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<td>B2: 3 x 10^7 cells Day 1</td>
<td>B3: 3 x 10^7 cells Day 1</td>
</tr>
<tr>
<td>n = 3- 6/cohort</td>
<td>n = 3- 6/cohort</td>
<td>n = 3- 6/cohort</td>
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</table>

**CLL**

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<tbody>
<tr>
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<td>B1: 9 x 10^6 cells Day 1</td>
<td>B2: 9 x 10^6 cells Day 1</td>
<td>B3: 9 x 10^6 cells Day 1</td>
</tr>
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<td>B2: 3 x 10^7 cells Day 1</td>
<td>B3: 3 x 10^7 cells Day 1</td>
</tr>
<tr>
<td>n = 3- 6/cohort</td>
<td>n = 3- 6/cohort</td>
<td>n = 3- 6/cohort</td>
<td>n = 3- 6/cohort</td>
</tr>
</tbody>
</table>
76 y/o woman with DLBCL, GCB subtype

- Stage II at study entry (mesenteric mass)
- Medical history notable for COPD, coronary artery disease, fungal pneumonia
- Received 7 prior therapies
  - 5 prior therapies included rituximab
  - 2 prior therapies included Flu / Cy
- Refractory to last therapy
  - Flu / Cy followed by experimental combination therapy of donor-derived NK cell therapy (16x10^9 cells), IL-2, and rituximab
- Enrolled in FT596-101 study at first dose level
  - 110 days from last therapy
  - No bridging therapy administered

<table>
<thead>
<tr>
<th>Prior Regimen</th>
<th>Best Response</th>
<th>Approximate DOR</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Rituximab, cyclophosphamide, hydroxydaunomycin, oncovin, prednisone (R-CHOP)</td>
<td>CR</td>
<td>2 years</td>
</tr>
<tr>
<td>2 Rituximab, carboplatin, etoposide, ifosfamide (R-ICE)</td>
<td>CR</td>
<td>16 months</td>
</tr>
<tr>
<td>3 Carmustine, etoposide, cytarabine, melphalan (BEAM) followed by ASCT</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4 Rituximab and ADAM17 inhibitor (maintenance)</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>5 BET inhibitor</td>
<td>PD</td>
<td>-----</td>
</tr>
<tr>
<td>6 Gemcitabine and oxaliplatin (GemOx)</td>
<td>PR</td>
<td>&lt;1 month</td>
</tr>
<tr>
<td>7 Flu / Cy lympho-conditioning followed by rituximab and ACTR707</td>
<td>CR</td>
<td>4 months</td>
</tr>
</tbody>
</table>

DOR = Duration of Response  
CR = complete response  
ASCT = Autologous stem cell transplant  
PR = partial response  
BET = Bromodomain and extra-terminal motif  
SD = stable disease  
NA = Data Not Available  
PD = progressive disease
FT596-101: Patient 2002 Case Study – Clinical Course

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

- First FT596 Single-dose Treatment Cycle
  - Single-dose monotherapy at $3 \times 10^7$ cells

- Second FT596 Single-dose Treatment Cycle
  - Single-dose monotherapy at $3 \times 10^7$ cells
  - Administered following FDA consent based on review of Cycle 1 clinical data

---

**Cycle 1**

- **FT596**
- **Response Assessment**

**Cycle 2**

- **FT596**
- **Response Assessment**

---

**Dose Cohort 1 Monotherapy**

- **CY / FLU**
  - Cyclophosphamide: 500 mg/m² IV x 3 days; Fludarabine: 30 mg/m² IV x 3 days

---

**Safety and Efficacy Follow up**

**DLT Assessment Period**

**FDA Engagement for Retreatment**
**Adverse Events of Interest**

<table>
<thead>
<tr>
<th></th>
<th>Cycle 1</th>
<th>Cycle 2</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Any Grade</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cytokine Release Syndrome</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>ICANS (^1)</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Graft-versus-Host Disease</td>
<td>No</td>
<td>No</td>
</tr>
</tbody>
</table>

\(^1\) ICANS = Immune Effector Cell-Associated Neurotoxicity Syndrome

- No dose-limiting toxicities (DLTs)
- No FT596-related serious adverse events (SAEs)
- Safety profile of adverse events (AEs) of interest was similar between Cycle 1 and Cycle 2
- Grade $\geq$3 AEs considered probably related to Flu/Cy conditioning and possibly related to FT596 included decreases in neutrophil, white blood cell, and lymphocyte counts
- Grade $\geq$3 AEs not related to FT596 were consistent with lympho-conditioning chemotherapy, medical history and prior treatment regimens
- No evidence of B- or T-cell mediated anti-product immunogenicity

**Anti-Product Immunogenicity**

<table>
<thead>
<tr>
<th></th>
<th>Cycle 1</th>
<th>Cycle 2</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>B-Cell mediated</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Detectable anti-FT596 Class I HLA Antibodies</td>
<td>0 Measured D29</td>
<td>0 Measured D29</td>
</tr>
<tr>
<td><strong>T-Cell mediated</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>FT596-specific T-cell IFN(\gamma) Response by ELISpot</td>
<td>Not Detected Measured D18, D29</td>
<td>Not Detected Measured D18, D29</td>
</tr>
</tbody>
</table>

*Note: As of September 24, 2020 data cut*
**FT596-101: Patient 2002 Case Study – Activity & Pharmacokinetics**

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

- Partial response at Study Day 29 following first FT596 single-dose cycle
- Deepening of response at Study Day 75 following second FT596 single-dose cycle
- DoR = 3.7 months, comparable to that of auto CD19 CAR-T cell therapy among patients who achieve PR as BOR
- FT596 demonstrated consistent, detectable PK in peripheral blood following each single-dose treatment cycle

---

**Baseline**

- SPD: 1292 mm²
- SUV: 28

**Study Day 29**

- SPD: 624 mm²
- SUV: 6.6

**Study Day 75**

- SPD: 420 mm²
- SUV: 2.6

---

**Partial Response**

- 67% reduction in tumor size

---

**Note:** As of September 24, 2020 data cut
Multiple Myeloma Franchise
FT538: hnCD16 + IL-15RF + CD38KO NK Cell Product Candidate
First-ever CRISPR-edited iPSC-derived Cell Therapy

Engineered with Three Components to Enhance Multiple Mechanisms of Innate Immunity

**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

**CD38KO**: Deletion of CD38 to eliminate anti-CD38 antibody mediated NK cell fratricide. Also shown to improve NK cell biology and potency through optimization of metabolic signaling

**IL-15RF**: Interleukin-15 receptor fusion, a potent cytokine complex that promotes survival, proliferation and trans-activation of NK cells and CD8 T cells
FT538: hnCD16 + IL-15RF + CD38KO NK Cell Product Candidate

Enhancing Multiple Mechanisms of Innate Immunity

Enhanced NK Cell Persistence & Metabolic Fitness

Enhanced NK Cell ADCC

Bjordahl et al. ASH Annual Meeting 2019
FT538-101: Relapsed / Refractory Multiple Myeloma
Multi-dose Combination with CD38-targeted and SLAMF7-targeted mAb

Daratumumab (start Day -11: QW x 8 then Q2W x 8 then Q4W until progression or unacceptable toxicity)

Elotuzumab (start Day -11: QW x 8 then Q2W until progression or unacceptable toxicity)

Additional doses of FT538 may be given based on evidence of clinical benefit and with FDA approval

DLT Assessment Period (D1–D29)

- DL1 = 100M cells / dose
- DL2 = 300M cells / dose
- DL3 = 1.0B cells / dose
- DL4 = 1.5B cells / dose
**FT576: Multi-antigen Targeted CAR-BCMA NK Cell Product Candidate**

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

Engineered with **Four** Anti-tumor Modalities for Multiple Myeloma

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

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

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

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

**IND Allowed in December 2020**
FT576: Multi-antigen Targeted CAR-BCMA NK Cell Product Candidate
BCMA Binding Domain with Differentiated Activation Threshold

✓ Validated CAR BCMA in diffuse large B cell lymphoma, follicular lymphoma, mantle cell lymphoma, and chronic lymphocytic leukemia

✓ BCMA CAR T cells triggered target cell lysis with an activation threshold in the range of 100 BCMA molecules, which allowed for an efficient eradication of B-NHL cells in vitro and in vivo

✓ Potential novel therapeutic option for patients where BCMA is expressed at low abundance or where anti-BCMA immunotherapies have failed due to antigen loss

Miller et al. ASH Annual Meeting 2020
Rationale for NK Cell Therapy in AML
Clinical Precedent with Non-Engineered Allogeneic NK Cell Therapy

• 300+ AML/MDS patients treated with allogeneic NK cells\textsuperscript{a}

• Numerous clinical studies in relapsed / refractory AML have shown\textsuperscript{a}:
  - CR rates = 25-35%
  - No GvHD
  - Minimal CRS / neurotoxicity

• Unmet need in AML remains high
  - ~21,000 newly diagnosed patients in the US alone every year\textsuperscript{b}
  - 5-year survival rate ~28\%\textsuperscript{b}
  - Significant opportunity for more effective, less toxic therapies
    - <50\% of elderly patients respond to initial therapy\textsuperscript{c}
    - 20-40\% of younger patients fail to respond to initial therapy\textsuperscript{c}
    - ~50\% of patients who attain an initial CR eventually relapse\textsuperscript{d}

\textsuperscript{a} Fate Therapeutics, Internal Literature Review
\textsuperscript{b} National Cancer Institute Surveillance, Epidemiology, and End Results Program. Cancer Stat Facts: AML. 2015.
\textsuperscript{d} Leopold LH, Willemez R. The Treatment of Acute Myeloid Leukemia in First Relapse: A Comprehensive Review of the Literature. Leuk Lymphoma. 2002; 43(9); 1715-1727
Off-the-Shelf, iPSC-derived NK Cell Cancer Immunotherapy Franchise

*Multiple Ongoing Phase 1 Studies in Relapsed / Refractory AML*

<table>
<thead>
<tr>
<th>Program</th>
<th>FT516 Monotherapy</th>
<th>FT538 Monotherapy</th>
<th>FT538 + anti-CD38 mAb</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Dose</strong></td>
<td>Three dose levels ranging from 90-900 million cells / dose</td>
<td>Four dose levels ranging from 100-1,500 million cells / dose</td>
<td>Four dose levels ranging from 100-1,500 million cells / dose</td>
</tr>
</tbody>
</table>
| **Schedule** | Lympho-conditioning ¹  
3 once-weekly doses + IL-2  
2 cycles | Lympho-conditioning ¹  
3 once-weekly doses | Lympho-conditioning ²  
3 once-weekly doses |
| **Assessment** | C1D30 Safety  
C2D30 Anti-tumor response | C1D30 Safety  
C1D30 Anti-tumor response | C1D30 Safety  
C1D30 Anti-tumor response |
| **Status** | Dose escalation ongoing | First patient treated | IND allowed (UMN IIT) |

¹ Cy 500 mg/m² x Flu 30 mg/m² x 3 days
² Cy 300 mg/m² x Flu 30 mg/m² x 2 days
Solid Tumor Franchise
Off-the-Shelf, iPSC-derived NK Cell Cancer Immunotherapy Franchise

The NK-Cell-Mediated Cancer Immunity Cycle

**Bridging Innate and Adaptive Immunity**

NK Cells

1. NK trafficking
2. NK mediated lysis

T Cells

3. T cell recruitment
4. T cell infiltration
5. T cell activation
Off-the-Shelf, iPSC-derived NK Cell Cancer Immunotherapy Franchise

*Multi-antigen Targeting: Enhanced Innate Immunity + CAR*

1\textsuperscript{st} Generation

- **FT500**

2\textsuperscript{nd} Generation

- **FT516**

3\textsuperscript{rd} Generation

- **FT538**

- **FT536**
  - CAR-MICA/B

- **FT5xx**
  - CAR-B7H3

- **FT5xx**
  - Janssen CAR

- High-affinity 158V, non-cleavable CD16 Fc receptor to augment ADCC
- Interleukin-15 receptor fusion to promote NK cell activity
- CD38 knock-out to eliminate NK cell fratricide and improve metabolic signaling
FT500-101: First-ever U.S. Clinical Study of iPSC-derived Cell Product
Phase 1 Dose Escalation in Advanced Solid Tumors

Cy: 300 mg/m² IV x 2 days
Flu: 25 mg/m² IV x 2 days
Prior to Cycle 1 only

- Regimen A: Monotherapy (n=9)
  - Salvage setting with patients having progressed or failed all FDA-approved therapies

- Regimen B: Combination with immune checkpoint inhibitor (ICI) therapy (n=6)
  - Tumor types where ICIs are approved
  - Salvage setting with patients having progressed or failed ICIs

- Two dose levels
  - 100M cells / dose and 300M cells / dose x up to 6 doses

Up to 6 doses over 45 days
FT500-101: First-ever U.S. Clinical Study of iPSC-derived Cell Product

Clinical Objectives

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

Assess Novel Paradigm

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

Key Clinical Read-outs

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

Key Molecular Read-outs

- Immune cell recovery
- Endogenous cytokine response (GvHD, CRS)
- Anti-product immunogenicity
FT500-101: Dose Escalation Clinical Results

Phase 1 Dose Escalation in Advanced Solid Tumors

Multi-dosing

• All 15 patients completed Cycle 1 (3 doses)
• 13 patients advanced to Cycle 2, with 11 of 13 patients completing Cycle 2 (3 additional doses)
• Among the 13 patients who initiated Cycle 2 treatment, dose discontinuation was due to disease progression
• 81 total doses of FT500 were administered to patients in the outpatient setting
• No B-cell or T-cell mediated anti-product responses observed despite post-conditioning immune recovery
FT500-101: Dose Escalation Clinical Results

Phase 1 Dose Escalation in Advanced Solid Tumors

Safety

• No dose-limiting toxicities, and no SAEs or Grade ≥ 3 AEs considered related to FT500, were observed

• No cases of cytokine release syndrome, immune effector cell-associated neurotoxicity syndrome, or graft-versus-host disease were observed

• No treatment-related discontinuations or deaths were observed

Efficacy

• Among 15 heavily pre-treated patients (10 who were refractory to prior therapy), 11 had a best overall response of SD

Patient Case Study - r/r cHL Resistant to anti-PD1 Therapy

• 29 y/o male with relapsed / refractory classical Hodgkin lymphoma (cHL)

• 14 prior therapies including multiple regimens containing FDA-approved ICI therapies; refractory to last prior regimen containing experimental anti-PD-1 therapy

• 84% reduction in size of a lymphonodal mass and a 58% reduction in size of all target lesions following three doses of FT500 plus anti-PD-1 therapy, however, new bone lesion was observed

IHC staining of the lymphonodal mass demonstrated post-treatment increases in the number of CD3+ and CD8+ cells and in the ratio of CD3+ and CD8+ cells to tumor cells, indicative of T-cell trafficking to the responding tumor bed.
FT500-101: Phase 1 Dose Expansion Ongoing
Targeting Solid Tumors Amendable to NK Cell Accessibility, Recognition, and Killing

Overcoming Resistance to Checkpoint Inhibitor Therapy in Advanced Solid Tumors

Patient who progressed on prior ICI

FT500 + IL2 + ICI

FT500 mediates both direct tumor lysis and T-cell recruitment / activation to re-sensitize ICI-resistant tumors

Dose Expansion Strategy | Rationale
--- | ---
**Tumor Enrichment** | • High % of tumor mutations leading to low / null MHC Class I expression
• NSCLC
• cHL
• NSCLC: NK cell trafficking
• cHL: POC in dose-escalation phase
• Accessible tumor biopsies

**Add IL-2 Support** | • IL-2 known to enhance NK cell function and persistence

FT500 Dosing: Up to six doses; three once-weekly doses at 300M cells / dose x 2 cycles
FT516-102: hnCD16 NK Cell Product Candidate for Advanced Solid Tumors

First Patient Treated in Combination with PD-L1-targeted mAb

Cyclophosphamide: 500 mg/m² IV x 3 days
Fludarabine: 30 mg/m² IV x 3 days
IL-2: 6M units sc with each FT516 dose

Up to 6 doses of FT516

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

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

Combination arms with PD1-, HER2-, EGFR-targeted mAbs are also allowed under IND
FT536: Multi-antigen Targeted CAR-MICA/B NK Cell Product Candidate
Pan-tumor Targeting Strategy for Solid Tumors

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

**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 CD8 T cells

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

**IND Submission Anticipated in 2021**
FT536: Multi-Targeted CAR-MICA/B NK Cell Product Candidate

Novel Pan-tumor Targeting Strategy for Solid Tumors

Science

Antibody-mediated inhibition of MICA and MICB shedding promotes NK cell-driven tumor immunity

- MICA/B are induced by cellular stress and transformation, and their expression has been reported for many cancer types
- NKG2D, an activating receptor expressed on NK and T cells, targets the membrane-distal α1 and α2 domains of MICA/B, activating a potent cytotoxic response
- Advanced cancer cells frequently evade immune cell recognition by proteolytic shedding of the α1 and α2 domains of MICA/B, which can significantly reduce NKG2D function and the cytolytic activity
- Therapeutic antibodies targeting the membrane-proximal α3 domain inhibited MICA/B shedding, resulting in a substantial increase in the cell surface density of MICA/B and restoration of immune cell-mediated tumor immunity
- We have developed a novel CAR targeting the conserved α3 domain of MICA/B (CAR-MICA/B)
- By uniquely targeting the α3 domain, FT536 prevents shedding and directly targets one of the most highly-expressed stress ligands on a broad range of tumors
iPSC-derived CAR T Cells
**FT819: Off-the-Shelf CAR19 T-Cell Product Candidate**

**Collaboration with Memorial Sloan Kettering Cancer Center**

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

**Novel CAR19 1XX placed under the control of endogenous TCR activity**

**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

**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

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

**IND Allowed by FDA for BCL, CLL and pre-B ALL**
FT819: Enhanced Tumor Control vs. Primary CAR T Cells

Disseminated Xenograft Model of Lymphoblastic Leukemia

Valamehr et al. Festival of Biologics Annual Meeting 2020
FT819-101: Phase I Dose Escalation Schema
Concurrent and Independent Dose Escalation in BCL, CLL and pre-B ALL

3 Indications x 3 Treatment Regimens

### Regimen A1

- **Day 1**: MAD (DL4+IL2), DL3+IL2, DL2+IL2, DL1+IL2

### Regimen A

- **Day 1**: MAD (FT819 + IL2), DL4, DL3, DL2

### Regimen B

- **Day 1**: MAD (CYP/FLU), MPAD or DL3, DL3, FT819
- **Day 3**: MPAD or DL2, DL2, FT819
- **Day 5**: FT819

**Dosing Regimen**
- **DL1**: $3 \times 10^7$ cells
- **DL2**: $9 \times 10^7$ cells
- **DL3**: $3 \times 10^8$ cells
- **DL4**: $9 \times 10^8$ cells

All cohorts are $n = 3$-$6$; escalation per 3+3 design

- If DL2 exceeds MTD, option to test DL1

**Starting Cohort**
Collaborations
Janssen Cancer Immunotherapy Collaboration (April 2020)

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

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

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

**Significant Economics**
- $100m upfront (+$50m equity put)
- Janssen pays for all collaboration costs
- $3+ billion in milestones, double-digit royalties
**ONO Cancer Immunotherapy Collaboration (September 2018)**

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

### 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
Financials
# Financial Summary

As reported in Company’s Consolidated Financial Statements

<table>
<thead>
<tr>
<th>Three Months Ended September 30, 2020</th>
</tr>
</thead>
<tbody>
<tr>
<td>Revenue</td>
</tr>
<tr>
<td>Operating Expense</td>
</tr>
<tr>
<td>Cash &amp; Cash Equivalents(^1)</td>
</tr>
<tr>
<td>Employees</td>
</tr>
<tr>
<td>Total Shares Outstanding(^2)</td>
</tr>
</tbody>
</table>

\(^1\) On an as adjusted basis to include January 2021 common stock offering

\(^2\) Includes 14.0M shares of common stock from conversion of non-voting preferred stock.