Programmed Cellular Immunotherapies

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

June 2021
Forward-Looking Statements

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

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

**Multiplexed Engineering**
Incorporate multiple mechanisms of action to eradicate cancer

**Treatment Paradigm**
Flexible out-patient treatment strategies to drive deep responses

**Off-the-Shelf**
Stable, cryopreserved for on-demand treatment and expanded patient reach

**Uniform Products**
Consistent identity, purity and potency of cell products

**Mass Production**
Reliable manufacturing process with high yield at low cost per dose
1. **Starting Cell Source.** Whether from the patient (autologous) or a healthy donor (allogeneic), the starting cell source is variable and can create batch-to-batch inconsistencies.

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

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

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

5. **Therapeutic Reach.** The overall patient experience, treatment setting, and cost-effectiveness, including the potential to reach patients earlier in care.
Master Cell Lines Enable Mass Production of Best-in-Class Cell Products

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

<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; Consistent</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><strong>Overall Paradigm</strong></td>
<td><strong>Process-centric</strong></td>
<td><strong>Product-centric</strong></td>
</tr>
</tbody>
</table>
Unique Biological Properties of Human iPSCs
Single-cell Isolation, Characterization & Selection for Creation of Master Engineered Cell Lines

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

- 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
iPSC Product Platform

Disruptive Approach Enabling Mass Production of Universal NK Cell and T-Cell Products

Clonal master iPSC lines are a renewable cell source that can be repeatedly used to mass produce homogeneous, cryopreserved cell product in a cost-effective manner.

‘On-Demand’ Delivery to Reach More Patients

Induced Pluripotent Stem Cells

Multiplexed Gene Engineering (one-time event)

Single-Cell Sorting & Clonal Selection

iPSC Expansion & Banking

Clonal Master Engineered iPSC Bank

Renewable Starting Cell Source

iT Cells

iNK Cells
iPSC Product Platform

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

- iPSCs
  - Day 0
  - > 10^6 iPSCs

- iCD34s
  - Day 10
  - > 1 million-fold expansion

- iNKs
  - Day 44
  - > 10^12 iNKs

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

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

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

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

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

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

**World Class Partnerships**: creating innovative iPSC-derived NK- and T-cell therapies with Janssen, Ono Pharmaceutical, University of Minnesota and Memorial Sloan Kettering
# 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>
<th>FT573</th>
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</thead>
<tbody>
<tr>
<td>Multi-faceted Innate Immunity</td>
<td></td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
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<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>
<td>✓</td>
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<tr>
<td>+ IL-15 Receptor Fusion</td>
<td>Enhance NK cell function</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
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<tr>
<td>+ CAR Insertion</td>
<td>Target tumor antigens</td>
<td>CD19</td>
<td>BCMA</td>
<td>MICA/B</td>
<td>B7H3</td>
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<tr>
<td>+ CD38 Knock-out</td>
<td>Enhance metabolic fitness</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
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<td>✓</td>
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<tr>
<td>Clinical Stage</td>
<td>P1</td>
<td>P1</td>
<td>P1</td>
<td>P1</td>
<td>SS</td>
<td>PC</td>
<td>PC</td>
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</tbody>
</table>

*P1 = Phase 1; SS = Phase 1 study start-up; PC = preclinical*
B-cell Malignancy Franchise
Novel High-Affinity, Non-Cleavable CD16a Fc Receptor
Optimizing Antibody-Dependent Cellular Cytotoxicity for Use with mAb Therapy

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

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

**Non-Cleavable**
TME causes shedding of CD16 and stifles ADCC

Issued patents covering composition of matter of mammalian cells incorporating hnCD16 receptor
FT516 & FT596: First-in-Class NK Cell Cancer Immunotherapies

Optimized Innate & Synthetic Biology

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

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

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

*Fc = fragment crystallizable; ADCC = antibody-dependent cellular cytotoxicity*
**FT516-101: B-Cell Lymphoma in Combination with Rituximab**

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

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

### 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 – Interim Safety, Tolerability, and Response**

<table>
<thead>
<tr>
<th>Subject #</th>
<th>Lymphoma Type</th>
<th>Prior Systemic Therapy</th>
<th>FT516 Response&lt;sup&gt;1&lt;/sup&gt;</th>
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</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td># Prior Regimens</td>
<td># Prior CD20-Targeted</td>
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<tr>
<td><strong>Dose Cohort 2 – 90 million cells / dose</strong></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>2005</td>
<td>DLBCL</td>
<td>3</td>
<td>2</td>
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<tr>
<td>2006</td>
<td>DLBCL</td>
<td>2</td>
<td>2</td>
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<tr>
<td>2007</td>
<td>DLBCL (DH)</td>
<td>3</td>
<td>3</td>
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<tr>
<td>2012</td>
<td>iNHL</td>
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<td>1</td>
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<tr>
<td><strong>Dose Cohort 3 – 300 million cells / dose</strong></td>
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<td></td>
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<td>2008</td>
<td>FL</td>
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<tr>
<td>2009</td>
<td>DLBCL (DH/DE)</td>
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<td>3</td>
</tr>
<tr>
<td>2010</td>
<td>FL</td>
<td>4</td>
<td>2</td>
</tr>
<tr>
<td>2011</td>
<td>Transformed iNHL</td>
<td>4</td>
<td>2</td>
</tr>
<tr>
<td>2013</td>
<td>DLBCL</td>
<td>2</td>
<td>2</td>
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<tr>
<td>2014</td>
<td>HGBCL</td>
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<tr>
<td>2015</td>
<td>HGBCL (TH)</td>
<td>7</td>
<td>5</td>
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</table>

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

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

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

<sup>1</sup> Cycle 2 Day 29 protocol-defined response assessment per Lugano 2014 criteria
**FT516-101: B-Cell Lymphoma in Combination with Rituximab**

*Interim Clinical Observations*

- Outpatient treatment paradigm of up to 6 doses was well-tolerated
  - No events of any grade of CRS, ICANS, or GvHD
  - No FT516-related SAEs or FT516-related Grade ≥3 AEs
  - No requirement for patient matching; no evidence of anti-product T- or B-cell mediated immunogenicity

- Objective response achieved in 8 of 11 patients (73%) treated with ≥ 90 million cells / dose
  - 6 of 11 (55%) patients achieved a CR
  - 2 of 4 patients (50%) previously treated with autologous CD19 CAR T-cell therapy achieved a CR

- Clear evidence that FT516 can drive responses in relapsed / refractory patients
  - Patients had received a median of 3 prior lines and a median of 2 prior lines containing CD20-targeted therapy
  - 8 of 11 patients had aggressive B-cell lymphoma
  - 5 of 11 patients were refractory to their most recent prior therapy

*Dose Escalation Ongoing at 900M Cells per Dose*
**FT596: Multi-antigen Targeted CAR19 NK Cell Product Candidate**

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

- **hnCD16 Synergizes with CD20-targeted mAb**
- **Multiple Doses of FT596 Compares Favorably to Single-dose Primary CAR19 T Cells**
- **FT596 + CD20-targeted mAb Engage Multiple B-cell Antigens for Enhanced Anti-tumor Activity and Prevention of Antigen Escape**

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

- Prevention of Antigen Escape
  - In vitro high-capacity test using high effector:target ratio (3:1) to maximize response in absence of primary antigen availability

**Legend**:
- Tumor (Raji) only
- Rituximab
- FT596 + Rituximab

**Graphs**:
- Total Flux (photons/s)
- Percent Survival
- Raji lymphoma line CD19+ CD20+
  - CAR19 | rituximab
- Raji lymphoma line CD19+ CD20+
  - FT596 + RTX

**Mice Models**:
- Lymphoma xenograft NSG immunodeficient mouse model
- Lymphoma xenograft CD34 engrafted humanized NSG mouse model

**Key**:
- Tumor (Raji) only
- CAR19 T-cell
- FT596
**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>Regimen</th>
<th>Cells</th>
<th>Day</th>
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</thead>
<tbody>
<tr>
<td><strong>A</strong></td>
<td>(9 \times 10^8)</td>
<td>1</td>
</tr>
<tr>
<td><strong>A</strong></td>
<td>(3 \times 10^8)</td>
<td>1</td>
</tr>
<tr>
<td><strong>A</strong></td>
<td>(9 \times 10^7)</td>
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**B2:**

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</tr>
<tr>
<td><strong>B1</strong></td>
<td>(3 \times 10^8)</td>
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<tr>
<td><strong>B1</strong></td>
<td>(9 \times 10^7)</td>
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**B3:**

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<tr>
<td><strong>B1</strong></td>
<td>(3 \times 10^8)</td>
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<td><strong>B1</strong></td>
<td>(9 \times 10^7)</td>
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**Follicular Lymphoma**

<table>
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<td><strong>B2</strong></td>
<td>(9 \times 10^8)</td>
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</tr>
<tr>
<td><strong>B2</strong></td>
<td>(3 \times 10^8)</td>
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**CLL**

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<tbody>
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</tr>
<tr>
<td><strong>B3</strong></td>
<td>(9 \times 10^8)</td>
<td>1</td>
</tr>
<tr>
<td><strong>B3</strong></td>
<td>(3 \times 10^8)</td>
<td>1</td>
</tr>
</tbody>
</table>

- **Regimen A:** FT596 Mono
- **Regimen B1:** FT596 + Rituximab (BCL)
- **Regimen B2:** FT596 + Obinutuzumab (BCL)
- **Regimen B3:** FT596 + Obinutuzumab (CLL)
FT596-101: Patient 2002 Case Study

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

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

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

**Baseline**
- SPD: 1292 mm²
- SUV: 28

**Study Day 29**
- SPD: 624 mm²
- SUV: 6.6

**Study Day 75**
- SPD: 420 mm²
- SUV: 2.6

*SPD: Sum of the Product of the Diameters; SUV: Standardized Uptake Value*
Multiple Myeloma Franchise
FT538 & FT576: First-in-Class NK Cell Cancer Immunotherapies

Optimized Innate & Synthetic Biology

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

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

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

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

*Fc = fragment crystallizable; ADCC - antibody-dependent cellular cytotoxicity; mAb – monoclonal antibody*
Multiple Myeloma Disease Franchise

**Planned Phase 1 Studies in Relapsed / Refractory MM**

<table>
<thead>
<tr>
<th>Program</th>
<th>FT538 (hnCD16 + IL15RF + CD38KO)</th>
<th>FT576 (hnCD16 + IL15RF + CD38KO + CAR-BCMA)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Treatment</strong></td>
<td>FT538 +/- daratumumab or elotuzumab</td>
<td>FT576 +/- daratumumab</td>
</tr>
<tr>
<td><strong>Setting</strong></td>
<td>Relapsed / Refractory MM</td>
<td>Relapsed / Refractory MM</td>
</tr>
</tbody>
</table>
| **Dose / Schedule** | 3 once-weekly doses x 1 cycle; second cycle subject to FDA consent  
  • DL1 = 100M  
  • DL2 = 300M  
  • DL3 = 1B  
  • DL4 = 1.5B | Undisclosed |
| **Status**    | IND allowed; study start-up ongoing | IND allowed; study start-up ongoing          |
FT538: hnCD16 + IL-15RF + CD38KO NK Cell Product Candidate

A Uniform, Well-Characterized Cell Product Optimized for Innate Immunity
**FT538: hnCD16 + IL-15RF + CD38KO NK Cell Product Candidate**

*Enhanced Persistence Without Cytokine Support*

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**Primary NK vs. FT538 in NSG Mouse**

- Primary NK Cells
- hnCD16 CD38KO IL-15RF iNK

**FT516 vs. FT538 in NSG Mouse**

- hnCD16 iNK
- hnCD16 CD38KO IL-15RF iNK

*Day 16*

*Cichocki et al. manuscript under review*
**FT538**: hnCD16 + IL-15RF + CD38KO NK Cell Product Candidate

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

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

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**Graph Descriptions**

- **Left Graph**:
  - **Y-axis**: Normalized MM.1R Cell Count
  - **X-axis**: Time (hrs)
  - **Legend**:
    - **PB NK**
    - **FT538**
    - **PB NK + Dara**
    - **FT538 + Dara**

- **Right Graph**:
  - **Y-axis**: Normalized MM.1R Cell Count
  - **X-axis**: Time (hrs)
  - **Legend**: Same as left graph

---

**Notes**

- **Transfer effector cells from MM.1R – Round 1 to MM.1R – Round 2**
FT538: hnCD16 + IL-15RF + CD38KO NK Cell Product Candidate

Enhanced ADCC in Combination with anti-CD38 mAb In Vivo
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

Treatment

Post-Tx Follow-up

Disease Response Assessment

Day -11 Day -5 Day -4 Day -3 Day 1 Day 8 Day 15 Day 29 ~15 Years

CY/FLU X X X X

FT538 FT538 FT538

DLT Assessment Period (D1–D29)

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

BCMA Binding Domain with Differentiated Activation Threshold

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

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

Jeffrey S. Miller, MD

Seminal 2005 Manuscript, >1,000 citations

• 300+ AML/MDS patients treated with allogeneic NK cells
  • Numerous clinical studies in relapsed / refractory AML have shown:
    – 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
  – 5-year survival rate ~28%
  – Significant opportunity for more effective, less toxic therapies
    o <50% of elderly patients respond to initial therapy
    o 20-40% of younger patients fail to respond to initial therapy
    o ~50% of patients who attain an initial CR eventually relapse

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a Fate Therapeutics, Internal Literature Review
AML Disease Franchise

Multiple Ongoing Phase 1 Studies in Relapsed / Refractory AML

<table>
<thead>
<tr>
<th>Program</th>
<th>FT516 (hnCD16)</th>
<th>FT538 (hnCD16 + IL15RF + CD38 KO)</th>
<th>FT538 (UMN IIT) (hnCD16 + IL15RF + CD38 KO)</th>
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</thead>
<tbody>
<tr>
<td><strong>Treatment</strong></td>
<td>FT516 Monotherapy</td>
<td>FT538 Monotherapy</td>
<td>FT538 + daratumumab</td>
</tr>
<tr>
<td></td>
<td>IL-2 cytokine support / dose</td>
<td>No IL-2 cytokine support</td>
<td>No IL-2 cytokine support</td>
</tr>
<tr>
<td><strong>Setting</strong></td>
<td>Relapsed / Refractory AML</td>
<td>Relapsed / Refractory AML</td>
<td>Relapsed / Refractory AML</td>
</tr>
<tr>
<td><strong>Dose / Schedule</strong></td>
<td>3 once-weekly doses x 2 cycles</td>
<td>3 once-weekly doses x 1 cycle; second cycle subject to FDA consent</td>
<td>3 once-weekly doses x 1 cycle;</td>
</tr>
<tr>
<td></td>
<td>• DL1 = 90M</td>
<td>• DL1 = 100M</td>
<td>• DL1 = 100M</td>
</tr>
<tr>
<td></td>
<td>• DL2 = 300M</td>
<td>• DL2 = 300M</td>
<td>• DL2 = 300M</td>
</tr>
<tr>
<td></td>
<td>• DL3 = 900M</td>
<td>• DL3 = 1B</td>
<td>• DL3 = 1B</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• DL4 = 1.5B</td>
<td>• DL4 = 1.5B</td>
</tr>
<tr>
<td><strong>Status</strong></td>
<td>DL3 enrolling</td>
<td>DL1 enrolling</td>
<td>IND allowed; study start-up ongoing</td>
</tr>
</tbody>
</table>
FT516-101: Monotherapy in Relapsed / Refractory AML

Patient Characteristics Reflect Extremely Poor Prognosis

<table>
<thead>
<tr>
<th>Subject #</th>
<th>Age</th>
<th># of Prior Lines</th>
<th>Risk Profile</th>
<th>Last Line of Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>1001</td>
<td>41</td>
<td>3</td>
<td>Intermediate</td>
<td>Yes</td>
</tr>
<tr>
<td>1003</td>
<td>64</td>
<td>3</td>
<td>Adverse</td>
<td>No</td>
</tr>
<tr>
<td>1005</td>
<td>58</td>
<td>4</td>
<td>Adverse</td>
<td>Yes</td>
</tr>
<tr>
<td>1006</td>
<td>68</td>
<td>1</td>
<td>Adverse</td>
<td>No</td>
</tr>
<tr>
<td>1007</td>
<td>85</td>
<td>1</td>
<td>Adverse</td>
<td>Yes</td>
</tr>
<tr>
<td>1008</td>
<td>33</td>
<td>6</td>
<td>Adverse</td>
<td>Yes</td>
</tr>
<tr>
<td>1011</td>
<td>60</td>
<td>3</td>
<td>Adverse</td>
<td>Yes</td>
</tr>
<tr>
<td>1012</td>
<td>56</td>
<td>5</td>
<td>Adverse</td>
<td>No</td>
</tr>
<tr>
<td>1015</td>
<td>59</td>
<td>3</td>
<td>Adverse</td>
<td>Yes</td>
</tr>
</tbody>
</table>

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

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

8 of 9 with Refractory Disease

Aza = Azacitidine; CLAG-M = Cladribine, Ara-C, Filgrastim, Mitoxantrone; Dec = Decitabine; PIF = Primary Induction Failure; Ven = Venetoclax; 7+3 = Ara-C + Daunorubicin
### FT516-101: Monotherapy in Relapsed / Refractory AML

**Safety, Tolerability & Immunogenicity**

<table>
<thead>
<tr>
<th>Subject #</th>
<th># Cells / Dose</th>
<th># of Doses</th>
<th>DLT</th>
<th>Any Grade CRS</th>
<th>Any Grade ICANS</th>
<th>Any Grade GvHD</th>
<th>Grade ≥ 3 AEs</th>
<th>SAEs</th>
<th>Immunogenicity</th>
</tr>
</thead>
<tbody>
<tr>
<td>1001</td>
<td>90M</td>
<td>6</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>None</td>
<td>None</td>
<td>No</td>
</tr>
<tr>
<td>1003</td>
<td>90M</td>
<td>6</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>FN (Gr3)</td>
<td>None</td>
<td>No</td>
</tr>
<tr>
<td>1005</td>
<td>90M</td>
<td>4</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>FN (Gr3)</td>
<td>None</td>
<td>No</td>
</tr>
<tr>
<td>1006</td>
<td>300M</td>
<td>6</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>None</td>
<td>None</td>
<td>No</td>
</tr>
<tr>
<td>1007</td>
<td>300M</td>
<td>6</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>None</td>
<td>None</td>
<td>No</td>
</tr>
<tr>
<td>1008</td>
<td>300M</td>
<td>3</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>None</td>
<td>None</td>
<td>No</td>
</tr>
<tr>
<td>1011</td>
<td>300M</td>
<td>3</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>None</td>
<td>None</td>
<td>No</td>
</tr>
<tr>
<td>1012</td>
<td>300M</td>
<td>6</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>None</td>
<td>None</td>
<td>No</td>
</tr>
<tr>
<td>1015</td>
<td>300M</td>
<td>3</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>FN (Gr3)</td>
<td>None</td>
<td>No</td>
</tr>
</tbody>
</table>

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

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

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

AE = Adverse Events; CRS = Cytokine Release Syndrome; DLT = Dose Limiting Toxicity; FN = Febrile Neutropenia; GvHD = Graft vs. Host Disease; ICANS = Immune Cell-Associated Neurotoxicity Syndrome; SAE = Serious Adverse Event
# FT516-101: Monotherapy in Relapsed / Refractory AML

## Best Overall Response

<table>
<thead>
<tr>
<th>Subject #</th>
<th># Cells / Dose</th>
<th># of Doses</th>
<th>Baseline</th>
<th>Best Overall Response (BOR)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>% Bone Marrow Blasts</td>
<td>Neutrophils (10^3/µl)</td>
<td>Platelets (10^3/µl)</td>
<td>% Bone Marrow Blasts</td>
</tr>
<tr>
<td>1001</td>
<td>90M</td>
<td>6</td>
<td>6%</td>
<td>0.8</td>
</tr>
<tr>
<td>1003</td>
<td>90M</td>
<td>6</td>
<td>39%</td>
<td>0.1</td>
</tr>
<tr>
<td>1005</td>
<td>90M</td>
<td>4</td>
<td>40%</td>
<td>0.2</td>
</tr>
<tr>
<td>1006</td>
<td>300M</td>
<td>6</td>
<td>26%</td>
<td>0.4</td>
</tr>
<tr>
<td>1007</td>
<td>300M</td>
<td>6</td>
<td>12%</td>
<td>1.9</td>
</tr>
<tr>
<td>1008</td>
<td>300M</td>
<td>3</td>
<td>95%</td>
<td>0.2</td>
</tr>
<tr>
<td>1011</td>
<td>300M</td>
<td>3</td>
<td>91%</td>
<td>0.4</td>
</tr>
<tr>
<td>1012</td>
<td>300M</td>
<td>6</td>
<td>20%</td>
<td>0.2</td>
</tr>
<tr>
<td>1015</td>
<td>300M</td>
<td>3</td>
<td>44%</td>
<td>0.1</td>
</tr>
</tbody>
</table>

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

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

**CRi** = Complete Remission (CR) other than, with respect to hematologic recovery, CRi requires recovery of neutrophils to ≥1000/µL or platelets to ≥100,000/µL; **MLFS** = Morphologic Leukemia Free State; **PD** = Progressive Disease; **SD** = Stable Disease
FT516-101: Monotherapy in Relapsed / Refractory AML

Duration of Anti-leukemic Activity

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

**Evidence of Evolving Response from MLFS to CRi**

**One Patient with CRi Proceeded to allo-HSCT**

CRi = Complete Remission (CR) other than, with respect to hematologic recovery, CRi requires recovery of neutrophils to ≥1000/µL or platelets to ≥100,000/µL; CP = Clinical Progression (evidence of progression not fulfilling 2017 ELN PD definition per investigator assessment); HR = Hematologic Relapse after CR/CRi; MLFS = Morphologic Leukemia Free State; SD = Stable Disease; PD = Progressive Disease
# FT538-101: Monotherapy in Relapsed / Refractory AML

## Patient Characteristics

<table>
<thead>
<tr>
<th>Subject #</th>
<th>Age</th>
<th>Prior Therapy</th>
<th># of Prior Lines</th>
<th>Risk Profile</th>
<th>Last Line of Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>1001</td>
<td>72</td>
<td>Ven + Aza 7+3</td>
<td>3</td>
<td>Unknown</td>
<td>Ven + Dec</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ven + Dec</td>
<td></td>
<td>No</td>
<td>Refractory</td>
</tr>
<tr>
<td>1002</td>
<td>78</td>
<td>Ven + Aza Gilteritinib GTB-3550</td>
<td>3</td>
<td>Adverse</td>
<td>GTB-3550</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>No</td>
<td>Refractory</td>
</tr>
<tr>
<td>1003</td>
<td>79</td>
<td>7+3 Ven + Aza GTB-3550 Glasdegib + LDAC</td>
<td>4</td>
<td>Intermediate</td>
<td>Glasdegib + LDAC</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>No</td>
<td>Refractory</td>
</tr>
</tbody>
</table>

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

### 3 Heavily Pre-treated Patients with Refractory Disease

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

Aza = Azacitidine; Dec = Decitabine; LDAC = Low-dose Ara-C; PIF = Primary Induction Failure; Ven = Venetoclax; 7+3 = Ara-C + Daunorubicin; GTB-3550 = investigational CD33-targeted Trispecific NK cell engager
### FT538-101: Monotherapy in Relapsed / Refractory AML

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

<table>
<thead>
<tr>
<th>Subject #</th>
<th>% BM Blasts Baseline</th>
<th>FT538 Doses</th>
<th>FT538-Related Safety</th>
<th>Immunogenicity</th>
<th>Best Overall Response (2017 ELN)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>DLT</td>
<td>Any Grade CRS</td>
<td>Any Grade ICANS</td>
</tr>
<tr>
<td>1001</td>
<td>70%</td>
<td>2</td>
<td>NE</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>1002</td>
<td>25%</td>
<td>6*</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>1003</td>
<td>30%</td>
<td>6*#</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
</tbody>
</table>

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

* FDA approved administration of Cycle 2

# Cycle 2 ongoing as of data cutoff

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

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

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

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

**CRI** = Complete Remission (CR) other than, with respect to hematologic recovery, CRi requires recovery of neutrophils to ≥1000/µL or platelets to ≥100,000/µL; **SD** = Stable Disease
FT538-101: Monotherapy in Relapsed / Refractory AML

Subject 1003 (100M cells)

- **Patient Characteristics**
  - 79 y.o. male diagnosed with *de novo* AML in 2017
  - Multiple lines of prior therapy
    - Idarubicin + Ara-C (CR; DOR = 20 months)
    - Venetoclax + Azacitidine (PR; DOR = 1 month)
    - GTB-3550 (investigational CD33-IL15-CD16 NK cell engager) (**refractory**)
    - Glasdegib + Low dose Ara-C (**refractory**)

- **Baseline Disease Status**
  - 2017 ELN risk category = **Intermediate**
  - Bone Marrow: 30% blasts by morphology; 20-30% cellularity
  - Peripheral Blood: No blasts; ANC = 0.1 x 10^3/µL (neutropenic); Platelets = 35 x 10^3/µL (thrombocytopenic)

- **Clinical Course**
  - Received 3 doses of FT538
  - No events of any grade of CRS, ICANS, or GVHD
  - No FT538-related Grade ≥3 AEs
  - 2017 ELN response criteria (BOR) = **CRi**
    - Complete neutrophil recovery *exceeding* baseline (1.6x10^3/µL from 0.1x10^3/µL)
    - Second treatment cycle approved by FDA; follow-up ongoing
FT516 / FT538: Monotherapy in Relapsed / Refractory AML

Initial Clinical Observations

- Phase 1 studies have enrolled an unfavorable patient population (n=12)
  - Median of 3 prior lines, with 11 patients refractory to their last prior therapy
  - 9 patients with adverse risk profile (with 1 patient unknown) based on 2017 ELN risk category
  - 11 patients had significant hematopoietic impairment at baseline, with both low neutrophil and platelet counts

- FT516 and FT538 as monotherapy exhibited favorable safety, and multi-dose treatment schedule was well-tolerated
  - No observed DLTs and no events of any grade of CRS, ICANS, or GVHD
  - Successfully administered in the outpatient setting

- 5 of 12 patients (42%) achieved an objective response with complete leukemic blast clearance in the bone marrow
  - FT516 (n=9): 3 CRi, 1 MLFS; FT538 (n=3): 1 CRi
  - Durable remissions >6 months achieved in 2 FT516 patients without any additional therapeutic intervention

- Additional engineered modalities of FT538 may confer further therapeutic advantages
  - CRi achieved in multiply-refractory patient, including to CD33-targeted NK cell engager, in first dose escalation cohort
  - FT538 detected in the peripheral blood at Day 8 post-infusion without administration of IL-2 cytokine support
Relapsed / Refractory Acute Myeloid Leukemia

FT538 + daratumumab for Targeting of CD38 on Leukemic Blasts

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

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

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

UMN IIT of FT538 + CD38-targeted daratumumab set to initiate in r/r AML
Solid Tumor Franchise
Developing Multi-functional Off-the-Shelf Cell Products for Solid Tumors

One Therapeutic Modality Incorporating Multiple Mechanisms in Fight Against Cancer

Off-the-Shelf Multiplexed Engineered Cell Products

- Cytokine / Chemokine Payload Delivery
- Immune Evasion
- Persistence / Expansion
- Enhanced Migration
- TME Redirect
- + TCR
- - Endogenous TCR
- CAR 1
- CAR 2

Engager Secretion
hnCD16
Off-the-Shelf, iPSC-derived NK Cell Cancer Immunotherapy Franchise

Evolution Toward Multiplexed-Engineered Cell-based Cancer Immunotherapies

1st Generation

2nd Generation

3rd Generation

FT500

FT516

FT538

FT536

FT573

FT5xx

CAR-MICA/B

CAR-B7H3

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

Incorporates Three Functional Components to Enhance Innate Immunity

- Innate immunity is severely compromised in patients with cancer
  - Depleted / dysfunctional NK cell compartment
  - Inferior ADCC capacity due to naturally-occurring, low-affinity CD16
  - Diminished ADCC activity through down-regulation or shedding of CD16
  - Exhaustion of NK cells within immunosuppressive tumor micro-environment

- FT538 is engineered to synergize with antibodies and effectively kill solid tumors
  - High-affinity, non-cleavable CD16 Fc receptor to synergize with mAbs and enhance ADCC
  - IL-15 receptor fusion to promote survival, proliferation and trans-activation of NK and T cells
  - CD38 knock-out to improve potency and metabolic fitness of NK cells

- FT538 provides proof-of-concept for multiplexed-engineered, iPSC-derived NK cells and serves as foundation for building CAR-targeted product candidates (FT536, FT573)

Phase 1 Dose Escalation in Combination with EGFR-targeted cetuximab, HER2-targeted trastuzumab, and PDL1-targeted avelumab to initiate in 2H21
FT536: Multi-targeted CAR-MICA/B NK Cell Product Candidate

**Novel Pan-tumor Targeting Strategy for Solid Tumors**

- MICA/B are induced by cellular stress and transformation, and their expression has been reported for many cancer types
- NKG2D, an activating receptor expressed on NK and T cells, targets the membrane-distal α1 and α2 domains of MICA/B, activating a potent cytotoxic response
- Advanced cancer cells frequently evade immune cell recognition by proteolytic shedding of the α1 and α2 domains of MICA/B, which can significantly reduce NKG2D function and the cytolytic activity
- Therapeutic antibodies targeting the membrane-proximal α3 domain inhibited MICA/B shedding, resulting in a substantial increase in the cell surface density of MICA/B and restoration of immune cell-mediated tumor immunity
- We have developed a novel CAR targeting the conserved α3 domain of MICA/B (CAR-MICA/B)
- By uniquely targeting the α3 domain, FT536 prevents shedding and directly targets one of the most highly-expressed stress ligands on a broad range of tumors
**FT573: Multi-targeted CAR-B7H3 NK Cell Product Candidate**

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

- B7H3 (CD276) belongs to the B7 superfamily of immune checkpoint molecules and is overexpressed in a wide variety of cancers, often associated with poor prognosis.

- B7H3 induces the Warburg effect and plays a key role in promoting metastasis and cancer stem cell-like properties.

- B7H3 also promotes resistance to cancer drugs and angiogenesis.

- B7H3-specific monoclonal antibodies and antibody-drug conjugates have shown anti-tumor activity against B7H3+ tumor cells in xenograft mouse models.

- We are developing a novel CAR to target a defined region of B7H3 as pan-tumor targeting approach.

https://doi.org/10.1016/j.trecan.2018.03.010
**FT500-101: First-ever U.S. Clinical Study of iPSC-derived Cell Product**

*Phase 1 Dose Expansion Ongoing in Advanced Solid Tumors*

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

**Phase 1 Dose-Escalation Results (n=15)**

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

ICI = Immune Checkpoint Inhibitors (e.g. pembrolizumab, nivolumab, atezolizumab)
CRS = cytokine release syndrome; ICANs = immune effector cell-associated neurotoxicity syndrome; GVHD = graft-versus-host disease
FT516-102: Combination with PDL1-targeted mAb for Advanced Solid Tumors

Phase 1 Dose Escalation Ongoing

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

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
iPSC-derived CAR T Cells
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

**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
**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
  - **DL3+**
  - **DL2+IL2**
  - **DL1+IL2**

**Regimen A**

- **Day 1**
  - MAD
  - **Max IL2**
  - **Max DL**

**Regimen B**

- **Day 1**
  - MAD
  - **DL2**

- **Day 5**
  - **DL3+**

- **Day 3**
  - **DL2**

- **Day 4**
  - **DL1**

**DL1** = 30M cells

**DL2** = 90M cells

**Max DL** = 900M 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

<p>| | |</p>
<table>
<thead>
<tr>
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</thead>
<tbody>
<tr>
<td>Three Months Ended March 31, 2021</td>
<td></td>
</tr>
<tr>
<td><strong>Revenue</strong></td>
<td>$11.1M</td>
</tr>
<tr>
<td><strong>Operating Expense(^1)</strong></td>
<td>$57.3M</td>
</tr>
<tr>
<td><strong>Cash &amp; Cash Equivalents</strong></td>
<td>$888M</td>
</tr>
<tr>
<td><strong>Employees</strong></td>
<td>300+</td>
</tr>
<tr>
<td><strong>Total Shares Outstanding(^2)</strong></td>
<td>107.9M</td>
</tr>
</tbody>
</table>

\(^1\) Includes $13m in stock-based compensation

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