Programmed Cellular Immunotherapies
Leading Off-the-Shelf Development of Cell Therapy Products using Clonal Master iPSC Lines

November 2020
Forward-Looking Statements

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A Remarkable 2-Year Journey of Firsts

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

FT500

Best-in-Class Lymphoma
FT516
FT596

Best-in-Class Myeloma
FT538

Solid Tumors
FT576

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

July 2018

1st IND submission
87 employees
$78M in cash

Sept 2020

9 Cleared INDs
250+ employees
$500M+ in cash

FT500

ONO

Fate THERAPEUTICS
Off-the-Shelf, iPSC-derived NK Cell Cancer Immunotherapy Franchise

**Significant Clinical Progress**

- 7 INDs approved for iPSC-derived NK cells*
- 5 ongoing studies across hematologic and solid tumors
  - FT500: Dose expansion in solid tumors resistant to checkpoint inhibitor therapy
  - FT516: Dose escalation in AML and in combination with CD20-directed mAb for B-cell lymphoma
  - FT516: Dose escalation in combination with PDL1-directed mAb for solid tumors
  - FT596: Dose escalation ± CD20-directed mAb for non-Hodgkin lymphoma and for CLL
  - FT538: Dose escalation in AML and in combination with CD38-directed mAb for multiple myeloma
- 2 additional studies projected to begin enrollment by YE20
  - FT516: Dose escalation in recurrent ovarian cancer (UMN IIT)
  - FT596: Dose escalation for relapse prevention post-HSCT in B-cell lymphoma (UMN IIT)

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

Initial Clinical Observations

Early clinical observations support the transformative potential of iPSC Product Platform

- **Multi-dosing**
  - 35+ patients dosed with 150+ doses of iPSC-derived NK cells (FT500, FT516, FT596, FT538)
  - Demonstrated ability to administer up to 6 doses safely in an outpatient setting
  - No evidence of anti-product T- or B-cell mediated immunogenicity

- **Safety**
  - No DLTs, CRS, ICANS or GvHD at dose levels ≤ 300M cells / dose

- **Activity**
  - Evidence of anti-tumor activity at initial (low) doses
  - Observed across multiple assets / indications in patients with relapsed / refractory disease
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

<table>
<thead>
<tr>
<th>Cell Population Engineering</th>
<th>Single-cell iPSC Isolation, Characterization and Selection</th>
<th>Clonal Master Engineered iPSC Line</th>
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<td>1 edit</td>
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<td></td>
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<tr>
<td></td>
<td>✓ A myriad of additional safety and efficacy analyses</td>
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A Renewable Cell Source for Mass Production of Engineered Immune Cells
The Making of Bona Fide NK Cells from a Clonal Master Engineered iPSC Bank

Robust cGMP Process

- iPSCs
  - Day 0
  - > 1 million-fold expansion
  - 10^6 iPSCs

- iCD34s
  - Day 10
  - TRA181
  - SSEA4
  - CD34
  - CD43

- iNKs
  - Day 44
  - CD45
  - CD56

- > 10^12 iNKs

- Homogeneous cell product
- 100s-1,000s doses per campaign
- Low-cost per dose cGMP production
- Cryopreserved
- High post-thaw viability
## 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><strong>Cell Source</strong></td>
<td>Patient and Donor Cells</td>
<td>Renewable Master Cell Line</td>
</tr>
<tr>
<td><strong>Genetic Engineering</strong></td>
<td>Random &amp; Variable</td>
<td>Uniform &amp; Complete</td>
</tr>
<tr>
<td><strong>Characterization</strong></td>
<td>Imprecise</td>
<td>Well-defined</td>
</tr>
<tr>
<td><strong>Product Identity</strong></td>
<td>Heterogeneous</td>
<td>Homogeneous</td>
</tr>
<tr>
<td><strong>Manufacturing</strong></td>
<td>Limited Dose Availability</td>
<td>Off-the-Shelf Availability</td>
</tr>
<tr>
<td><strong>Cost-per-Dose</strong></td>
<td>High</td>
<td>Low</td>
</tr>
<tr>
<td><strong>Dosing</strong></td>
<td>Single Dose</td>
<td>Multiple Doses / Multiple Cycles</td>
</tr>
<tr>
<td><strong>Overall Paradigm</strong></td>
<td>Process-centric</td>
<td>Product-centric</td>
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Off-the-Shelf, iPSC-derived NK Cell Cancer Immunotherapy Franchise
Systematic Build of Industry-Leading iPSC-derived NK Cell Product Pipeline

<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>
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<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-associated antigen</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>CD19</td>
<td>BCMA</td>
</tr>
<tr>
<td>+ CD38 Knock-out</td>
<td>Resist CD38-mediated fratricide</td>
<td></td>
<td></td>
<td></td>
<td>✓</td>
<td>✓</td>
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</tr>
<tr>
<td><strong>Total # of Synthetic Elements</strong></td>
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<td>0</td>
<td>1</td>
<td>3</td>
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Off-the-Shelf, iPSC-derived NK Cell Cancer Immunotherapy Franchise

Integrating Additional Functional Components to Enhance Innate Immunity

1st Generation

FT500

2nd Generation

FT516

- High-affinity 158V, non-cleavable CD16 Fc receptor to augment ADCC
- Interleukin-15 receptor fusion to promote NK cell activity

3rd Generation

FT538

- CD38 knock-out to eliminate NK cell fratricide and improve metabolic signaling
**FT500: First iPSC-derived (non-engineered) NK Cell Product Candidate**

*Clinical Objectives*

**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: First-ever U.S. Clinical Study of iPSC-derived Cell Product**

*Phase 1 Dose Escalation in Advanced Solid Tumors*

- **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

Cy: 300 mg/m² IV x 2 days
Flu: 25 mg/m² IV x 2 days
Prior to Cycle 1 only
FT500: 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)
- In all cases, 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: 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 lines of FDA-approved ICI therapies

• 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: Phase 1 Dose Expansion Ongoing

Overcoming Resistance to Checkpoint Inhibitor Therapy in Advanced Solid Tumors

Patient who progressed on prior ICI

FT500 + ICI

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

<table>
<thead>
<tr>
<th>Dose Expansion Strategy</th>
<th>Rationale</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Tumor Enrichment</strong></td>
<td>• High % of tumor mutations leading to low / null MHC Class I expression</td>
</tr>
<tr>
<td>• NSCLC</td>
<td>• NSCLC: NK cell trafficking</td>
</tr>
<tr>
<td>• cHL</td>
<td>• cHL: POC in dose-escalation phase</td>
</tr>
<tr>
<td>• Accessible tumor biopsies</td>
<td></td>
</tr>
<tr>
<td><strong>Add IL-2 Support</strong></td>
<td>• IL-2 known to enhance NK cell function and persistence</td>
</tr>
</tbody>
</table>

FT500 Dosing: Up to six doses; three once-weekly doses at 300M cells / dose x 2 cycles
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 IgG1 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

- CD16 shedding in the tumor microenvironment can significantly limit NK cell anti-tumor activity

How to bring the 158V CD16 NK cell experience to all patients?
FT516: hnCD16 NK Cell Product Candidate
High-Affinity 158V, Non-Cleavable CD16 Fc Receptor for Enhanced ADCC

Pluripotent stem cell–derived NK cells with high-affinity noncleavable CD16a mediate improved antitumor activity

Engineered CD16a high-affinity antibody-binding receptor resists shedding upon activation

FT516

<table>
<thead>
<tr>
<th>Phase 1 Study</th>
<th>Regimen</th>
<th>Dosing (M cells)</th>
<th>Schedule</th>
<th>Status</th>
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<tbody>
<tr>
<td>AML</td>
<td>Monotherapy</td>
<td>90, 300, 900</td>
<td>3 once-weekly x 2</td>
<td>Dose escalation ongoing</td>
</tr>
<tr>
<td>B-cell Lymphoma</td>
<td>+ Rituximab</td>
<td>30, 90, 300, 900</td>
<td>3 once-weekly x 2</td>
<td>Dose escalation ongoing</td>
</tr>
<tr>
<td>Solid Tumors</td>
<td>+ Avelumab</td>
<td>90, 300, 900</td>
<td>3 once-weekly x 2</td>
<td>Dose escalation ongoing</td>
</tr>
<tr>
<td>Recurrent Ovarian Cancer (ITT)</td>
<td>Monotherapy</td>
<td>90, 300, 900</td>
<td>3 once-weekly x 1</td>
<td>Open to enrollment</td>
</tr>
</tbody>
</table>

Conditioning Regimen: 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

ADCC activity in in vivo systemic tumor model (Raji-Luc tumor cells)
3 of 5 mice in hnCD16 iNK cell + anti-CD20 mAb group maintained complete remission at Day 100
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: Off-the-Shelf hnCD16 CD38- NK Cell Product Candidate**

*Enhancing Multiple Mechanisms of Innate Immunity*

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**Enhanced NK Cell Persistence & Metabolic Fitness**

**Enhanced NK Cell ADCC**

First patient dosed following IND Approval for AML as Monotherapy and in Combination with daratumumab for MM
Off-the-Shelf, iPSC-derived NK Cell Cancer Immunotherapy Franchise

Multi-antigen Targeting: CAR + Enhanced Innate Immunity
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: Phase 1 Dose Escalation Schema
Parallel Escalation of Single-dose Mono and mAb Combo in BCL and CLL

B-cell Malignancies

Regimen A: FT596 Mono
- A: 9 x 10^6 cells Day 1
- A: 3 x 10^7 cells Day 1
- A: 9 x 10^8 cells Day 1

Regimen B1: FT596 + Rituximab
- B1: 9 x 10^6 cells Day 1
- B1: 3 x 10^7 cells Day 1
- B1: 9 x 10^8 cells Day 1

Regimen B2: FT596 + Obinutuzumab
- B2: 9 x 10^6 cells Day 1
- B2: 3 x 10^7 cells Day 1
- B2: 9 x 10^8 cells Day 1

Regimen B3: FT596 + Obinutuzumab
- B3: 9 x 10^6 cells Day 1
- B3: 3 x 10^7 cells Day 1
- B3: 9 x 10^8 cells Day 1

Follicular Lymphoma

Regimen A: FT596 Mono
- A: 9 x 10^6 cells Day 1
- A: 3 x 10^7 cells Day 1
- A: 9 x 10^8 cells Day 1

Regimen B1: FT596 + Rituximab
- B1: 9 x 10^6 cells Day 1
- B1: 3 x 10^7 cells Day 1
- B1: 9 x 10^8 cells Day 1

Regimen B2: FT596 + Obinutuzumab
- B2: 9 x 10^6 cells Day 1
- B2: 3 x 10^7 cells Day 1
- B2: 9 x 10^8 cells Day 1

Regimen B3: FT596 + Obinutuzumab
- B3: 9 x 10^6 cells Day 1
- B3: 3 x 10^7 cells Day 1
- B3: 9 x 10^8 cells Day 1

CLL

Regimen A: FT596 Mono
- A: 9 x 10^6 cells Day 1
- A: 3 x 10^7 cells Day 1
- A: 9 x 10^8 cells Day 1

Regimen B1: FT596 + Rituximab
- B1: 9 x 10^6 cells Day 1
- B1: 3 x 10^7 cells Day 1
- B1: 9 x 10^8 cells Day 1

Regimen B2: FT596 + Obinutuzumab
- B2: 9 x 10^6 cells Day 1
- B2: 3 x 10^7 cells Day 1
- B2: 9 x 10^8 cells Day 1

Regimen B3: FT596 + Obinutuzumab
- B3: 9 x 10^6 cells Day 1
- B3: 3 x 10^7 cells Day 1
- B3: 9 x 10^8 cells Day 1

n = 3-6/cohort
FT596: First Clinical Observations

Phase 1 Monotherapy in Relapsed / Refractory DLBCL

**Patient 1 (single-dose of 30M FT596 cells as a monotherapy)**
- Treated with 4+ lines of therapy for relapsed / refractory diffuse large B-cell lymphoma (DLBCL)
- Most recently had disease progression following CD19-targeting CAR T-cell therapy (Yescarta)
- Day 29 protocol-defined response assessment = Progressive Disease (PD)

**Patient 2 (single-dose of 30M FT596 cells as a monotherapy)**
- Treated with 7+ lines of therapy for relapsed / refractory diffuse large B-cell lymphoma (DLBCL)
- Most recently refractory to experimental combo therapy comprised of expanded allogeneic NK cells, IL-2, and rituximab
- Day 29 protocol-defined response assessment = Partial Response (PR)
  - 73% reduction standardized uptake value (SUV) and a 52% reduction in tumor size by PET-CT
  - Peak FT596 cell expansion detected at Day 8 (~1800 transgene copies / µg DNA)
  - Potential to re-treat with FDA consent
- Administered a second cycle of lympho-conditioning followed by single dose of 30M cells

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No events of cytokine release syndrome, neurotoxicity, or graft-versus-host disease were reported by investigators in either patient
**FT576: Multi-Targeted CAR-BCMA NK Cell Product Candidate**

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

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

- **High-affinity, Non-Cleavable CD16**
  - *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**
  - *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**
  - *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**
  - *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.

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*IND filing anticipated by YE 2020*
FT576: Multi-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-CD19 immunotherapies have failed due to antigen loss
FT536: Multi-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 filing anticipated in 2021
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
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

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: 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
  - DL4
  - DL3
  - DL2
  - DL1

**Regimen B**

- Day 1
  - MAD
  - MPAD or DL3
  - DL3

- Day 3
  - MPAD or DL2
  - DL2

- Day 5
  - DL1

*DL1 = 3 x 10^7 cells
DL2 = 9 x 10^7 cells
DL3 = 3 x 10^8 cells
DL4 = 9 x 10^8 cells*

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

If DL2 exceeds MTD, option to test DL1

Starting Cohort
Janssen Cancer Immunotherapy Collaboration

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

Upfront payment of $50M in cash + $50M purchase of FATE c/s at 47% premium

- FATE to incorporate Janssen proprietary antigen binding domains into iPSC-derived CAR NK- and CAR T-cells
  - Up to 4 antigen targets, including targets expressed on hematologic malignancies and solid tumors
- FATE to preclinically develop product candidates to IND submission
  - Janssen to pay for all collaboration costs
- Janssen to conduct global clinical development and commercialization
  - FATE retains a right to opt-in to 50-50 commercialization arrangement in U.S.
  - FATE primarily responsible for clinical and commercial manufacture
- FATE eligible to receive up to $3.0BN in milestones ($1.8BN in dev / reg; $1.2BN in commercial) plus double-digit royalties on commercial sales
# iPSC Product Platform

## Industry-Leading Off-the-Shelf Cell-based Cancer Immunotherapy Pipeline

<table>
<thead>
<tr>
<th>Engineered Mechanisms</th>
<th>AML</th>
<th>NHL / CLL</th>
<th>MM</th>
<th>Solid Tumors</th>
</tr>
</thead>
<tbody>
<tr>
<td>FT500 Innate</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>FT516 + hnCD16</td>
<td></td>
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</tr>
<tr>
<td>FT538 + hnCD16 + IL15RF + CD38KO</td>
<td></td>
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<tr>
<td>FT596 + hnCD16 + IL15RF + CAR19</td>
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</tr>
<tr>
<td>FT576 + hnCD16 + IL15RF + CD38KO + CAR-BCMA</td>
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</tr>
<tr>
<td>FT536 + hnCD16 + IL15RF + CD38KO + CAR-MICA/B</td>
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<tr>
<td>FT819 Adaptive + CAR19</td>
<td></td>
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<tr>
<td>Janssen Innate and Adaptive CAR ¹</td>
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<td></td>
</tr>
<tr>
<td>Ono Adaptive CAR ²</td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>

¹ Includes CAR NK and T-cells directed to up to 4 antigen targets.
² Includes CAR T-cells directed to up to 2 antigen targets.
## Financial Summary

**As of September 30, 2020**

<table>
<thead>
<tr>
<th>Three Months Ended September 30, 2020</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Revenue</td>
<td>$7.6M</td>
</tr>
<tr>
<td>Operating Expense, Adjusted ¹</td>
<td>$31.2M</td>
</tr>
<tr>
<td>Cash &amp; Cash Equivalents</td>
<td>$502M</td>
</tr>
<tr>
<td>Employees</td>
<td>250+</td>
</tr>
<tr>
<td>Total Shares Outstanding ²</td>
<td>100.9M</td>
</tr>
</tbody>
</table>

¹ Excludes non-cash stock-based compensation expense of $7.8M.

² Includes 14.0M shares of common stock from conversion of non-voting preferred stock.