Programmed Cellular Immunotherapies

Transforming the Treatment of Cancer with Off-the-shelf, Multiplexed-engineered, iPSC-derived Cellular Immunotherapy

November 2022
Forward-Looking Statements

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

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

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

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

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

**World Class Partnerships:** co-developing novel iPSC-derived CAR NK and CAR T-cell product candidates with Ono and Janssen for hematologic malignancies and solid tumors
Changing the Game in Cell Therapy

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

**Multiplexed Engineering**
Multiple mechanisms of attack against cancer incorporated into cell product

**Drug-like Treatment**
Multi-dose schedules administered in the outpatient setting

**Mass Production**
Scalable GMP operations yielding 100s of doses in single campaign

**Off-the-Shelf**
Cryopreserved with long-term stability for storage and on-demand availability

**Uniform Products**
Batch-to-batch consistency of cell product features and functionality
Disruptive iPSC Product Platform
Creating Multiplexed-engineered iPSC-Derived Cell Products

A Single Human Induced Pluripotent Stem Cell (iPSC)
A renewable source for mass production of cell products

- Unlimited Clonal Expansion
- Multiplexed Engineering
- Extensive Characterization
- Single iPSC Clone
- Potential to Differentiate into 200+ Cell Types
- Scalable Master Cell Lines and Banks
- Uniform in Composition

Fate Therapeutics’ iPSC product platform is supported by an IP portfolio with 350+ issued patents and 150+ pending patent applications
Disruptive iPSC Product Platform

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

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

Designed with multiple tumor-fighting mechanisms
High quality; consistent purity and activity
High yield; scalable for efficient manufacturing
On-demand; expanded patient reach
Disruptive iPSC Product Platform

Novel Functional Armament Deployed in Attack Against Cancer

Direct Antigen Targeting
CARs directed to CD19, BCMA, MICA/B, B7H3

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

Immunosuppressive Resistance
Synthetic TGFβ redirector to promote activation in response to immuno-suppressive TME

Cell Homing
Synthetic CXCR2 receptor to promote cell trafficking

Stealth
Ligand/receptor engineering to prevent rejection

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

Cytokine Support
IL15RF and IL7RF for cell potentiation

Checkpoint KO
CD38 knock-out to promote metabolic fitness and prevent fratricide
A Transformative Cell Product Approach for the Treatment of Cancer

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

- **Flexible Administration**
  - On-demand treatment
  - Reliable and convenient without the need for hospitalization
  - Lower administrative burden

- **Combination Therapies**
  - Synergize with other anti-cancer agents
  - Activate endogenous immune system
  - Induce multiple complementary mechanisms of action

- **Earlier Intervention**
  - Off-the-shelf availability
  - Treatment paradigm enables add-on to early-line SOC regimens
  - Reach into the community setting with mass produced cell product
## Off-the-Shelf, iPSC-derived, Cell-based Cancer Immunotherapy Franchise
### Projected 2022 Corporate Milestones

<table>
<thead>
<tr>
<th>Category</th>
<th>Milestones</th>
</tr>
</thead>
</table>
| **Hematologic Malignancies** | - Launch registration study under RMAT for relapsed / refractory aggressive BCL  
- Initiate early-line aggressive BCL study for FT596 + R-CHOP  
- Generate clinical datasets with FT516 / FT596 (BCL), FT538 (MM, AML), FT576 (MM) and FT819 (BCM) |
| **Solid Tumors** | - Generate dose-escalation datasets with FT538 + mAb therapy to enhance ADCC  
- Initiate dose-escalation study of FT536 as novel pan-tumor targeting strategy  
- Complete IND-enabling studies of B7H3-targeted CAR programs |
| **Innovation** | - Nominate two novel multi-antigen targeted programs for solid tumors  
- Complete preclinical development of ADR functionality to enable conditioning-free cell therapy  
- Complete preclinical development of TSR functionality to enhance TME functional persistence |
| **Partnerships** | - Submit IND to FDA for first iPSC-derived CAR NK cell program under Janssen partnership  
- Complete IND-enabling studies for iPSC-derived CAR T-cell program under Ono partnership  
- Expand iPSC-derived product pipeline through additional collaborations |
| **Corporate** | - Complete tech transfer and initiate technical operations at commercial GMP facility  
- Continue expansion of dominant IP portfolio with 350+ issued patents  
- Maintain strong balance sheet |

*ADCC = antibody-dependent cellular cytotoxicity; ADR = allo-defense receptor; AML = acute myeloid leukemia; BCL = B-cell lymphoma; BCM = B-cell malignancies; MM = multiple myeloma; TME = tumor microenvironment; TSR = tumor suppressive redirector*
Hematologic Malignancies
# Hematologic Malignancy Product Pipeline

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

<table>
<thead>
<tr>
<th>Program</th>
<th>Functionality &amp; Cell Type</th>
<th>Target(s)</th>
<th>Indication(s)</th>
<th>Preclin</th>
<th>Phase 1</th>
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<tr>
<td>FT516</td>
<td>hnCD16 iNK</td>
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<td>n/a</td>
<td>AML</td>
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<tr>
<td>FT576</td>
<td>hnCD16 + IL15RF + CD38-KO + CAR-BCMA iNK</td>
<td>BCMA ± CD38</td>
<td>MM ± mAb</td>
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<td>GPRC5D ± CD38</td>
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<td>iNK, iT</td>
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</table>

*iPSC = induced pluripotent stem cell  iNK = iPSC-derived NK Cell  iT = iPSC-derived T cell  mAb = monoclonal antibody  BCM = B-cell malignancies  AML = Acute Myeloid Leukemia  MM = Multiple Myeloma  hnCD16 = high affinity, non-cleavable CD16 Fc receptor  IL15-RF = IL15 receptor fusion  CD38-KO = CD38 knock-out  CAR = chimeric antigen receptor*
Non-Hodgkin Lymphoma
Off-the-Shelf, iPSC-derived NK Cell Franchise for B-cell Malignancies
*FT516 and FT596 Product Candidates*

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

**CAR19**: Chimeric antigen receptor that targets B-cell antigen CD19; optimized for NK cell biology

**IL-15RF**: Interleukin-15 receptor fusion to promote survival, proliferation and anti-tumor activity

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

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

Three-dose Treatment Schedule; Up to 2 Cycles

- First-in-human study assessing the safety and activity of FT516 combined with rituximab in patients with r/r B-cell lymphoma
  - Primary objective: identify DLT and determine maximum tolerated dose / maximum assessed dose
  - Additional objectives: safety, tolerability, preliminary activity, pharmacokinetics, and immunogenicity

- Novel three-dose treatment schedule
  - Up to two cycles of treatment, with each cycle consisting of 3 days of conditioning (Cy / Flu) and 1 dose of rituximab (375 mg/m²) followed by 3 once-weekly doses of FT516 with IL-2 cytokine support
  - No mandatory hospitalization required during the treatment period

Cyclophosphamide: 500 mg/m² IV x 3 days  Fludarabine: 30 mg/m² IV x 3 days  Rituximab: 1 dose at 375 mg/m² IV per cycle  IL-2: 6M units sc with each FT516 dose
FT516-101: Phase 1 Study in R/R B-cell Lymphoma

Interim Phase 1 Clinical Data

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

**Safety & Tolerability**
- No CRS, ICANS, GvHD
- No treatment discontinuations due to AEs

**Response Rates**
- 11 of 18 patients (61%) treated at ≥90M cells / dose achieved OR, including 8 of 10 patients (80%) naïve to treatment with prior CAR T-cell therapy

**Durability of Response**
- 11 patients (61%) remained in ongoing response at 3 months from initiation of treatment. As of data cutoff date:
  - Naïve CAR T. 6 of 10 (60%) patients continued in ongoing response at 9.1m MFU, including 4 patients >6m; longest FU = 13.2m
  - Prior CAR T. 2 of 8 (25%) patients continued in complete response at 6.5m MFU, including 1 patient >6m; longest FU = 8.3m

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

CAR = Chimeric antigen receptor; CR = Complete response; DLBCL = Diffuse large B-cell lymphoma; FLGU = Follicular lymphoma grade unknown; G1FL = Grade 1 follicular lymphoma; G3AFL = Grade 3A follicular lymphoma; GZL = Gray zone lymphoma; HGBCL = High-grade B-cell lymphoma; HGBCL - HGBCL; M = Million; MCL = Mantle cell lymphoma; FU = Follow-up; MZL = Marginal zone lymphoma (B-cell lymphoma, of small to intermediate size cells, most likely MZL); PD = Progressive disease; OR = Objective response; PR = Partial response; SD = Stable disease; R/R = Relapsed/refractory; tNHL = Transformed indolent lymphoma
FT596-101: Phase 1 Study in R/R B-cell Lymphoma

**Single-dose Treatment Schedule; Up to 2 Cycles**

- First-in-human study assessing the safety and activity of FT596 as monotherapy and in combination with rituximab in patients with r/r B-cell lymphoma
  - Primary objective: identify DLT and determine maximum tolerated dose / dose schedule
  - Additional objectives: safety, tolerability, preliminary activity, pharmacokinetics, and immunogenicity

- Single-dose treatment schedule
  - Up to two cycles of treatment, with each cycle consisting of 3 days of conditioning (Cy / Flu) and 1 dose of rituximab (375 mg/m²) followed by a single dose of FT596 without cytokine support
  - No mandatory hospitalization required during the treatment period

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**Monoclonal Antibody Therapy on Day -4:**
- Rituximab 375 mg/m²; Obinutuzumab 1000 mg/m²; CY = Cyclophosphamide 500 mg/m²; FLU = Fludarabine 30 mg/m²
**FT596-101: Phase 1 Study in R/R B-cell Lymphoma**

**Interim Phase 1 Clinical Data**

<table>
<thead>
<tr>
<th>Subject Number</th>
<th>Primary Diagnosis</th>
<th>FT596 Dose</th>
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<tbody>
<tr>
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<tr>
<td>2009</td>
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<td>2017</td>
<td>G2FL</td>
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<tr>
<td>2021</td>
<td>WM</td>
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</tr>
<tr>
<td>2020</td>
<td>SLL</td>
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<td>2027</td>
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<td>2028</td>
<td>MCL</td>
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<tr>
<td>2015</td>
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<td>90M</td>
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<tr>
<td>2013</td>
<td>HGBCL</td>
<td>90M</td>
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<tr>
<td>2007</td>
<td>G3BFL</td>
<td>90M</td>
</tr>
<tr>
<td>2012</td>
<td>G2FL</td>
<td>300M</td>
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<tr>
<td>2016</td>
<td>INHL</td>
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<tr>
<td>2018</td>
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<tr>
<td>2029</td>
<td>TNHL</td>
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<td>2030</td>
<td>RT</td>
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<tr>
<td>2010</td>
<td>DLBCL</td>
<td>90M</td>
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<tr>
<td>2022</td>
<td>DLBCL</td>
<td>300M</td>
</tr>
<tr>
<td>2011</td>
<td>DLBCL</td>
<td>90M</td>
</tr>
<tr>
<td>2038</td>
<td>HGBCL</td>
<td>300M</td>
</tr>
</tbody>
</table>

### Safety & Tolerability
- No DLTs, ICANS, or GvHD
- Observed CRS (n=3) was infrequent, low-grade, and of limited duration

### Response Rates at 90M and 300M Cell Dose
- 13 of 19 patients (68%) achieve OR (n=7/9 in Monotherapy Arm; n=6/10 in Combination Arm), including 3 of 5 patients (60%) previously treated with auto CD19 CAR T-cell therapy

### Durability of Response
- All patients treated with second FT596 single-dose cycle (n=11) reached six months in CR (n=4) or continued in ongoing response (n=7)
- **Combination Arm.** Of 6 responding patients, 5 patients continued in ongoing response at 4.6m MFU, including 2 patients in ongoing CR >6m; and 1 patient reached 6m in CR and subsequently had PD at 6.7m
- **Prior CAR T.** All 3 responding patients continued in ongoing response, including 2 patients in ongoing CR >6m
FT516 and FT596 NK Cell Programs for B-cell Malignancies

Ongoing Development Initiatives

**FT516 Program**

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

**FT596 Program**

- Ongoing P1 dose expansion with single-dose treatment schedule at 900M cells in multiple disease-specific cohorts
- Ongoing P1 dose escalation with 2-dose treatment schedules at 900M cells / dose and at 1.8B cells / dose; 3-dose treatment schedule to be initiated subject to DLT clearance
- Opening clinical study assessing FT596 in 1L community setting without Cy / Flu chemotherapy conditioning as an add-on to R-CHOP SOC regimen, with first patient expected to be treated in 2H22
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 CD3ζ 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
FT819: Enhanced Tumor Control vs. Primary CAR T Cells
Disseminated Xenograft Model of Lymphoblastic Leukemia

FT819 Consists of Memory Phenotype and Outcompetes Primary CAR T Cells In Vivo
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
- DL1 + IL2
- DL3 + IL2
- DL2 + IL2
- DL1 + IL2

**Regimen A**

Day 1
- Max + IL2
- Max DL
- DL3
- DL2
- DL1

**Regimen B**

Day 1
- Max DL
- 1/3rd DL3
- 1/3rd DL2

Day 3
- Max DL
- 1/3rd DL3
- 1/3rd DL2

Day 5
- Max DL
- 1/3rd DL3
- 1/3rd DL2

DL1 = 30M cells
DL2 = 90M cells
DL3 = 180M cells
DL4 = 360M cells
DL5 = 540M cells

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

If DL2 exceeds MTD, option to test DL1

Starting Cohort
Multiple Myeloma Franchise
FT538: hnCD16 + IL-15RF + CD38KO NK Cell Product Candidate

Uniformly engineered with three functional elements designed to optimize innate immunity

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

Enhanced persistence without cytokine support
FT516 vs. FT538 in NSG Mouse
FT538: hnCD16 + IL-15RF + CD38KO NK Cell Product Candidate

Enhanced ADCC in Combination with anti-CD38 mAb In Vivo

Phase 1 Dose Escalation Ongoing in Combination with daratumumab
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

MM.1S-Luc cells
FT576-101: Phase 1 Dose Escalation Schema

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

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

- **Single-dose Monotherapy**
  - 100M Cells
  - 300M Cells
  - 600M Cells
  - 1.5B Cells

- **Two- and Three-dose Monotherapy**
  - 50M Cells x >1 dose
  - 150M Cells x >1 dose
  - 300M Cells x >1 dose
  - 600M Cells x >1 dose

- **Single-dose Combination**
  - 50M Cells
  - 150M Cells
  - 300M Cells
  - 600M Cells

- **Two- and Three-dose Combination**
  - 50M Cells x >1 dose
  - 150M Cells x >1 dose
  - 300M Cells x >1 dose
  - 600M Cells x >1 dose

Additional treatment cycles permitted subject to FDA consent
AML Franchise
Off-the-Shelf, iPSC-derived NK Cell Franchise for AML
FT516 and FT538 Product Candidates

Jeffrey S. Miller, MD

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
    - <50% of elderly patients respond to initial therapy
    - 20-40% of younger patients fail to respond to initial therapy
    - ~50% of patients who attain an initial CR eventually relapse

Fate Therapeutics, Internal Literature Review


FT516 / FT538: Ongoing Phase 1 Studies as Monotherapy

Interim Phase 1 Data in Relapsed / Refractory AML

- Ongoing P1 studies of FT516 and FT538 as monotherapy have enrolled patients with poor prognosis (n=12)
  - Median of 3 prior lines, with 11 patients refractory to their last prior therapy
  - 9 patients with adverse risk profile (with 1 patient unknown) based on 2017 ELN risk category
  - 11 patients had significant hematopoietic impairment at baseline, with both low neutrophil and platelet counts
- FT516 and FT538 as monotherapy exhibited favorable safety, and multi-dose treatment schedule was well-tolerated
  - No observed DLTs and no events of any grade of CRS, ICANS, or GVHD
  - Successfully administered in the outpatient setting
- 5 of 12 patients (42%) achieved an objective response with complete leukemic blast clearance in the bone marrow
  - FT516 (n=9): 3 CRi, 1 MLFS; FT538 (n=3): 1 CRi
  - Durable remissions >6 months achieved in 2 FT516 patients without any additional therapeutic intervention
- Additional engineered modalities of FT538 may confer further therapeutic advantages
  - CRi achieved in multiply-refractory patient, including to CD33-targeted NK cell engager, in first dose escalation cohort
  - FT538 detected in the peripheral blood at Day 8 post-infusion without administration of IL-2 cytokine support

Interim Phase 1 data includes 9 FT516 patients (3 at 90M cells / dose and 6 at 300M cells / dose) and 3 FT538 patients at 100M cells / dose. All data based on database entry as of April 16, 2021. Data subject to source document verification.
FT538: Ongoing Phase 1 Study in Combination with CD38-targeted mAb

FT538 + daratumumab for Targeting of CD38 on Leukemic Blasts

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 ongoing
Solid Tumor Franchise
Off-the-shelf, iPSC-derived Cell-based Cancer Immunotherapies

Developing Synthetic Killer Cells for Solid Tumors

- Developing next-generation cancer immunotherapies must address numerous challenges that limit the effectiveness of today’s agents in treating solid tumors.
  - Depleted / dysfunctional immune cells
  - Immuno-suppressive microenvironment
  - Tumor heterogeneity and escape

- Cell-based cancer immunotherapies have the unique potential to bring rejuvenated immune cells to the fight against cancer.
  - Address deficiencies in patients’ endogenous immune system, mount multi-pronged attack, and synergize with complementary agents

- Fate Therapeutics has built a robust pipeline of off-the-shelf, multiplexed-engineered cell therapies for solid tumors.
  - Incorporate synthetic features specifically designed to exploit novel MOAs, synergize with approved agents, and overcome mechanisms of resistance

Modified after Saetersmoen et al. Seminars in Immunopathology 2019
## Solid Tumor Product Pipeline

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

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<tr>
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ST = solid tumors  
mAb = monoclonal antibody  

hnCD16 = high affinity, non-cleavable CD16 Fc receptor  
IL15-RF = IL15 receptor fusion  
CD38-KO = CD38 knock-out  
CAR = chimeric antigen receptor  
EGFR = Epidermal Growth Factor  
HER2 = Human Epidermal Growth Factor Receptor 2  
PD1 = Programmed Cell Death Protein 1  
MICA/B = MHC class I polypeptide-related sequence A/B  
B7H3 = B7 homolog 3 protein  
KLK2 = Kallikrein related Peptidase 2

### Orthogonal Mechanisms of Attack for Solid Tumors

Cooperation between Innate and Adaptive Immunity  |  Augmenting ADCC  
Overcoming Tumor Escape  |  Targeting Metabolic Profile of Cancer
FT538-102: Multi-arm, Dose-escalating Phase 1 Study

*Designed to Assess Cooperation with Adaptive Immune System and Enhanced ADCC*

- Multiple cycles each comprising Cy / Flu conditioning, 3 doses of FT538, and mAb therapy
  - Up to 2 cycles; potential to administer additional cycles with clinical benefit and upon relapse
  - FT538 dose ranging from 100M cells / dose to 1.5B cells / dose
  - Each mAb combination enrolls independently

<table>
<thead>
<tr>
<th></th>
<th>Pembrolizumab</th>
<th>Avelumab</th>
<th>Trastuzumab</th>
<th>Cetuximab</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Target</strong></td>
<td>PD1</td>
<td>PD-L1</td>
<td>HER2</td>
<td>EGFR</td>
</tr>
<tr>
<td><strong>Eligibility</strong></td>
<td>Tumors with documented PD-L1 expression</td>
<td>HER2+ tumors: ≥2+ by IHC; ≥4 signals/cell by ISH</td>
<td>EGFR+ tumors, incl. KRAS/NRAS and driver mutations</td>
<td></td>
</tr>
<tr>
<td><strong>Primary Cancers</strong></td>
<td>NSCLC, GE, HNSCC, TNBC, UC</td>
<td>Gastric, Breast</td>
<td>NSCLC, CRC, HNSCC</td>
<td></td>
</tr>
</tbody>
</table>

CRC = colorectal cancer; GE = gastroesophageal; HNSCC = head and neck squamous cell carcinoma; IHC = immunohistochemistry; ISH = in situ hybridization; NSCLC = non-small cell lung carcinoma; TNBC = triple-negative breast cancer; UC = urothelial carcinoma
FT536: Multi-antigen Targeted CAR-MICA/B NK Cell Product Candidate
Pan-tumor Targeting Strategy to Overcome Tumor Escape

- MICA/B are cell-surface proteins induced by cellular stress and transformation, and their expression has been reported for many cancer types.

- NKG2D, an activating receptor expressed on NK and T cells, targets the membrane-distal α1 and α2 domains of MICA/B, activating a potent cytotoxic response.

- Cancer cells frequently evade immune cell recognition by proteolytic shedding of the α1 and α2 domains of MICA/B, which can significantly reduce NKG2D function and cytolytic activity.

- Soluble MICA/B have been associated with poor clinical prognosis.

- Overcoming MICA/B shedding to effectively re-engage tumor cells is emerging as a novel pan-tumor targeting mechanism.

- Preclinical data have shown that therapeutic antibodies targeting the membrane-proximal α3 domain inhibit MICA/B shedding, resulting in increased MICA/B cell-surface density and restoration of immune cell-mediated tumor immunity.
FT536: Multi-antigen Targeted CAR-MICA/B NK Cell Product Candidate

Durable Tumor Reduction of B16 MICA+ Metastatic Lung Lesions

1x10^4 cells
B16 MICA+ (iv)

1x10^7 cells
MICA/B CAR iNK

NSG Mice

+ 3 Days

+ 11 Days

Manual Metastases Count

Lung Tumor Burden

B16 MICA+ Tumor alone
CAR Negative iNK cells
MICA/B CAR+ iNK cells

Total Met Number
FT536-101: Multi-arm, Dose-escalating Phase 1 Study
Designed to Assess Cooperation with Adaptive Immune System and Enhanced ADCC

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

<table>
<thead>
<tr>
<th>Target</th>
<th>Monotherapy</th>
<th>Pembrolizumab, Avelumab</th>
<th>Trastuzumab</th>
<th>Cetuximab</th>
<th>Amivantamab</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eligibility</td>
<td>No biomarker-driven eligibility</td>
<td>Documented PD-L1 expression</td>
<td>Documented HER2 expression; NSCLC with HER2 mutation</td>
<td>EGFR+ tumors, incl. KRAS/NRAS and driver mutations</td>
<td>EGFR driver mutations, MET mutations</td>
</tr>
<tr>
<td>Primary Cancers</td>
<td>NSCLC, CRC, BC, Ovarian, Pancreatic</td>
<td>NSCLC, GE, HNSCC, TNBC, UC</td>
<td>Gastric, Breast</td>
<td>NSCLC, CRC, HNSCC</td>
<td>NSCLC</td>
</tr>
</tbody>
</table>

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

CRC = colorectal cancer; GE = gastroesophageal; HNSCC = head and neck squamous cell carcinoma; NA = Not applicable; NSCLC = non-small cell lung carcinoma; TNBC = triple-negative breast cancer; UC = urothelial carcinoma
B7H3-targeted, Multiplexed-engineered CAR Product Candidate

Pan-tumor Targeting Approach Aimed at the Metabolic Profile of Cancer

• B7H3 (CD276) belongs to the B7 superfamily of immune checkpoint molecules.

• B7H3 protein is aberrantly overexpressed in a wide variety of cancers
  – Limited expression in normal tissues
  – High levels found on immunologically “cold” tumors (e.g., prostate, HNSCC, GBM, soft tissue sarcomas)
  – Often associated with poor prognosis

• Shown to be a critical promoter of tumorigenesis and metastasis, and its expression is a metabolic hallmark of cancer.

• Multiple modalities targeting B7H3 have shown early clinical activity in patients with advanced solid tumors.
B7H3-targeted, Multiplexed-engineered CAR Product Candidate

Identification of Novel anti-camB7H3 scFv

CAR Design

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

![V_{H\text{H}} anti-camB7H3 scFv](image1)

![NK Cell CAR Construct](image2)

![T Cell CAR Construct](image3)

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

![Graph showing Total Flux](image4)
Collaborations & Financials
Oncology Innovation for Heme Malignancies & Solid Tumors

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

Strategic Collaboration

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

Significant Economics

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

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

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

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

Financial Terms
- $10m upfront
- 50-50 cost sharing to pre-IND milestone
- Up to $840 million in milestones, mid-single to low double-digit royalties
Financial Summary
As reported in Company’s Consolidated Financial Statements

<table>
<thead>
<tr>
<th>Financial Metric</th>
<th>Amount</th>
</tr>
</thead>
<tbody>
<tr>
<td>Revenue</td>
<td>$15.0M</td>
</tr>
<tr>
<td>Operating Expense¹</td>
<td>$101.4M</td>
</tr>
<tr>
<td>Cash &amp; Cash Equivalents</td>
<td>$519M</td>
</tr>
<tr>
<td>Total Shares Outstanding²</td>
<td>111.1M</td>
</tr>
</tbody>
</table>

2022 Cash, Cash Equivalents & Investments ≥$440M³

1 Includes $19.5m in stock-based compensation
2 Includes 14.0M shares of common stock from conversion of non-voting, preferred stock
3 Not including a $12.5M option exercise fee triggered by Ono’s opt-in decision in November for its first iPSC-derived CAR T cell candidate or other potential collaboration milestones in 4Q22