



Off-the-shelf Cell-based Cancer Immunotherapy

Developing First-of-kind Cell Products using Clonal Master iPSC Lines

2019 ASH Dinner Discussion

December 6, 2019



Join Us for Dinner During the ASH Annual Meeting

Friday, December 6, 2019

7:00 - 9:00pm

Hyatt Regency Orlando

9801 International Drive
Orlando, FL 32819

RSVP by November 29

Michael Horowicz
michael.horowicz@sternir.com
212.362.1200

*Initial clinical data of FT500, first-ever
iPSC-derived cell therapy to undergo U.S.
clinical investigation, to be highlighted*

Special Guest Speakers

Jeffrey S. Miller, MD

*Deputy Director, Masonic Cancer Center
Director, Cancer Experimental Therapeutics
Initiative (CETI), University of Minnesota*

Michel Sadelain, MD, PhD

*Director, Center for Cell Engineering,
Memorial Sloan Kettering Cancer Center*

Eric Smith, MD, PhD

*Director of Clinical Translation, Cellular
Therapeutics Center, Memorial Sloan Kettering
Cancer Center*

ASH Oral Presentations

FT538: Preclinical Development of an Off-the-Shelf
Adoptive NK Cell Immunotherapy with Targeted
Disruption of CD38 to Prevent Anti-CD38 Antibody-
Mediated Fratricide and Enhance ADCC in Multiple
Myeloma When Combined with Daratumumab
Saturday, December 7, 2019, 9:30 AM, W415A

FT596: Translation of First-of-Kind Multi-Antigen
Targeted Off-the-Shelf CAR-NK Cell with Engineered
Persistence for the Treatment of B Cell Malignancies
Saturday, December 7, 2019, 4:00 PM, W415A

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.

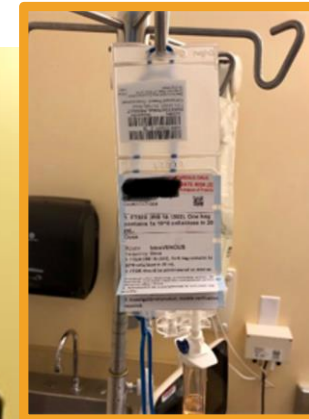


Introduction



2019 – A Break-through Year for the FATE iPSC Product Platform

Feb 2019 – First-ever Patient Treated with an iPSC-derived Cell Therapy in U.S.



FT500

From left: Sandip Patel, MD; Dan Kaufman, MD, PhD; Derek Ruff



2019 – A Break-through Year for the FATE iPSC Product Platform

Oct 2019 – First-ever Patients Treated with Cell Therapy derived from a Clonal Master Engineered iPSC Line

University of Minnesota opens first-ever U.S. clinical trial of engineered iPSC-derived cell therapy for blood cancers



MINNEAPOLIS, MN- October 21, 2019 - A new cancer clinical trial has opened at the M Health Fairview University of Minnesota Medical Center that leverages the groundbreaking research on stem cells and natural killer (NK) cells done at the Masonic Cancer Center and applies it to attack acute myeloid leukemia (AML) and B-cell lymphoma. The first-of-its-kind NK cell cancer immunotherapy, called FT516, is manufactured from a human induced pluripotent stem cell (iPSC) that has been genetically engineered to enhance its anti-tumor activity.

The first-in-human clinical trial of FT516, sponsored by Fate Therapeutics, will be run locally by Claudio Brunstein, MD, PhD, who is a professor of Medicine at the U of M Medical School, a member of the Masonic Cancer Center, and the medical director of the Adult Blood and Marrow Transplant and Cellular Therapy Program at M Health Fairview.



Claudio Brunstein, MD, PhD

"We potentially have an unlimited source of very similar, reproducible cancer fighters," said Brunstein. "This is opening a whole new door in cellular therapy. With increased modifications to these NK cells, we can elevate their ability to attack tumors. As we add more functionality to NK cells, we have the potential to bring together multiple anti-tumor mechanisms and more effectively target and kill cancer."



2019 – A Break-through Year for the FATE iPSC Product Platform

Sept 2019 – Secured FDA Clearance of IND Application for First-ever Cell Therapy Engineered with Three Anti-Tumor Modalities

Fate Therapeutics Announces the Opening of its cGMP Manufacturing Facility Dedicated to iPSC-derived Cell Therapies

State-of-the-Art Facility Designed to use Clonal Master iPSC Lines as Renewable Cell Source for Manufacture of Off-the-Shelf Product Pipeline

San Diego, CA – September 30, 2019 – Fate Therapeutics, Inc. (NASDAQ: FATE), a clinical-stage biopharmaceutical company dedicated to the development of programmed cellular immunotherapies for cancer and immune disorders, announced today that the Company has opened its current Good Manufacturing Process (cGMP) compliant manufacturing facility for the clinical production of its off-the-shelf natural killer (NK) cell and chimeric antigen receptor (CAR) T-cell product candidates. The



- *Completed GMP production of FT596 at FATE facility in November*
- *Single “small-batch” manufacturing campaign yielded ~320 cryopreserved, infusion-ready doses*
- *Estimated actual cost per dose: <\$2,500*



2019 – A Break-through Year for the FATE iPSC Product Platform

Aug 2019 – Issuance of Foundational U.S. Patent Covering iPSC-derived CAR T Cells

United States Patent
Themeli et al.

Patent No.: US 10,370,452 B2
Date of Patent: Aug. 6, 2019

EFFECTIVE GENERATION OF TUMOR-TARGETED T CELLS DERIVED FROM PLURIPOTENT STEM CELLS

Applicant: MEMORIAL SLOAN-KETTERING
CANCER CENTER, New York, NY
(US)

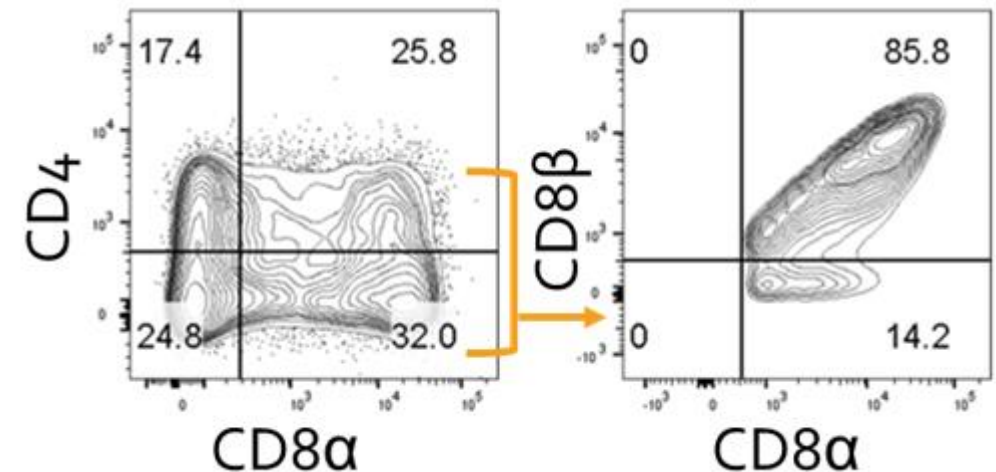
Inventors: Maria Themeli, New York, NY (US);
Michel Sadelain, New York, NY (US);
Christopher C. Kloss, New York, NY
(US)

Assignee: MEMORIAL SLOAN-KETTERING
CANCER CENTER, New York, NY
(US)

Claim 1. A population of T cells that are produced by in vitro differentiation of a pluripotent stem cell, wherein (i) the pluripotent stem cell expresses a chimeric antigen receptor (CAR), and (ii) the population of T cells comprises a T cell exhibiting a CD45RA⁺ CD27⁻ CD28⁻ CCR7⁻ CD62L⁻ phenotype.

- **Priority Date = April 3, 2013**
- **Publication Date = October 9, 2014**

iPSC-derived CAR T-cell Phenotype





2019 – A Break-through Year for the FATE iPSC Product Platform

Dec 2019 – No Morphologic Evidence of Leukemia, with Complete Neutrophil Recovery, Observed in First Patient Treated with FT516 Monotherapy for AML

41 year old male diagnosed with AML in January 2019

- Refractory to initial induction therapy and multiple additional lines of therapy

Enrolled in FT516 Study (Oct 2019)

- Early assessment following first three doses of FT516 with IL-2 cytokine support showed:
 - No morphologic evidence of leukemia, with evidence of hematopoietic recovery, in bone marrow
 - No circulating leukemic blasts in peripheral blood
 - Recovery of neutrophils (>1,000 per μL)
 - No observed CRS, neurotoxicity or GvHD
 - FT516 chimerism detected *in the bone marrow* at Day 18 by digital PCR



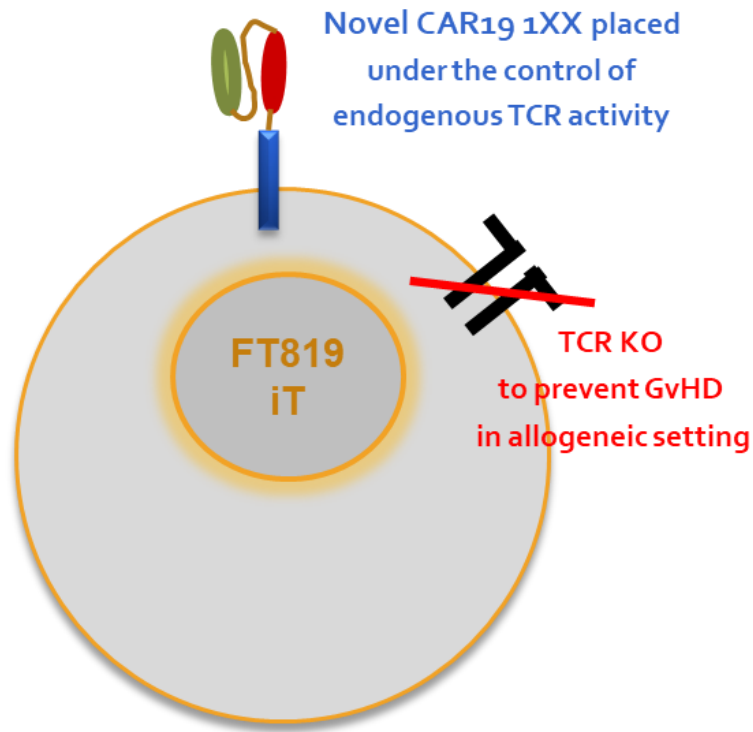
Off-the-Shelf T-cell Immunotherapy

FT819 Universal, Off-the-Shelf CAR19 T-Cell Product

Novel CAR19 Targeted to the TRAC Locus for Improved Safety and Efficacy



First-in-Class Off-the-Shelf Adoptive CAR19 T cell Product Uniformly Consisting of Novel Engineering Elements



→ **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

1H20 IND Submission Planned

Using Single-Cell iPSC System to Conduct Complex Engineering

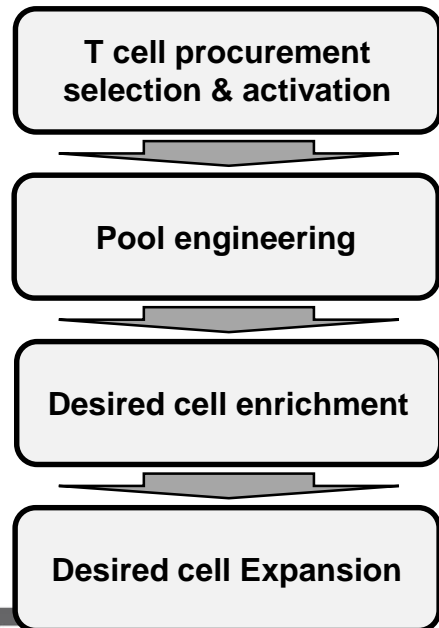
TRAC targeted, TCR less, 1XX CAR19 T cell Product Profile Comparison

LETTER

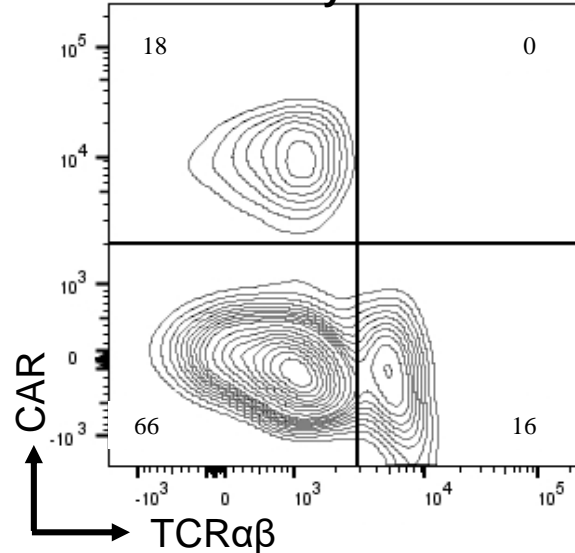
doi:10.1038/nature21405

Targeting a CAR to the *TRAC* locus with CRISPR/Cas9 enhances tumour rejection

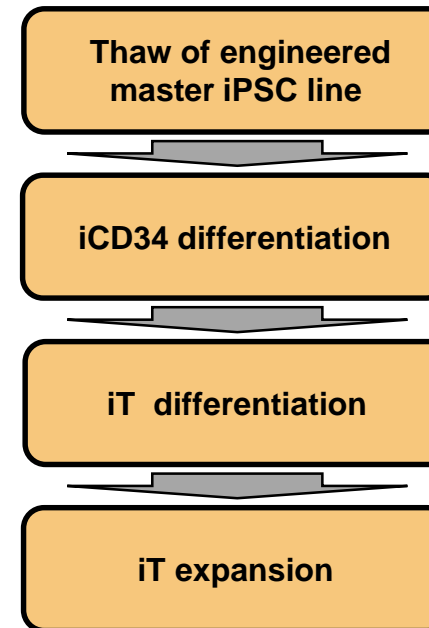
Justin Eyquem^{1*}, Jorge Mansilla-Soto^{1*}, Theodoros Giavridis¹, Sjoukje J. C. van der Stegen¹, Mohamad Hamieh¹, Kristen M. Cunanan², Ashlesha Odak¹, Mithat Gönen² & Michel Sadelain¹



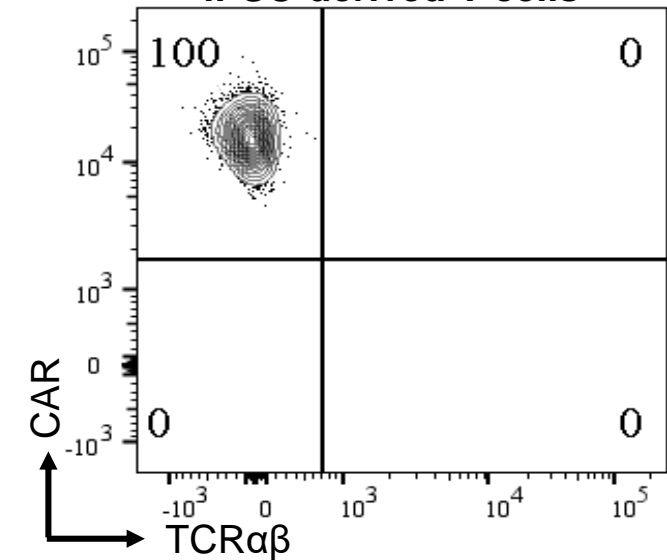
Pool Engineered Primary T Cells



VS.



Single-cell Engineered iPSC-derived T cells



FT819 Path to IND – iPSC Clone Generation

Multi-parameter iPSC engineering, clone screening and MCB selection



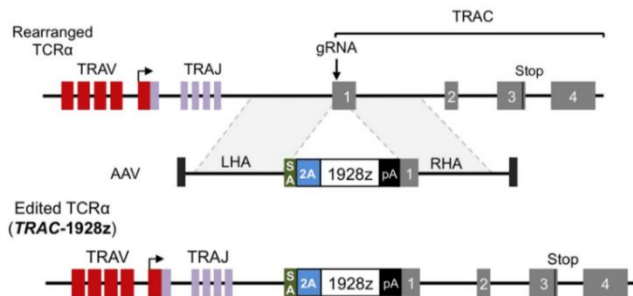
LETTER

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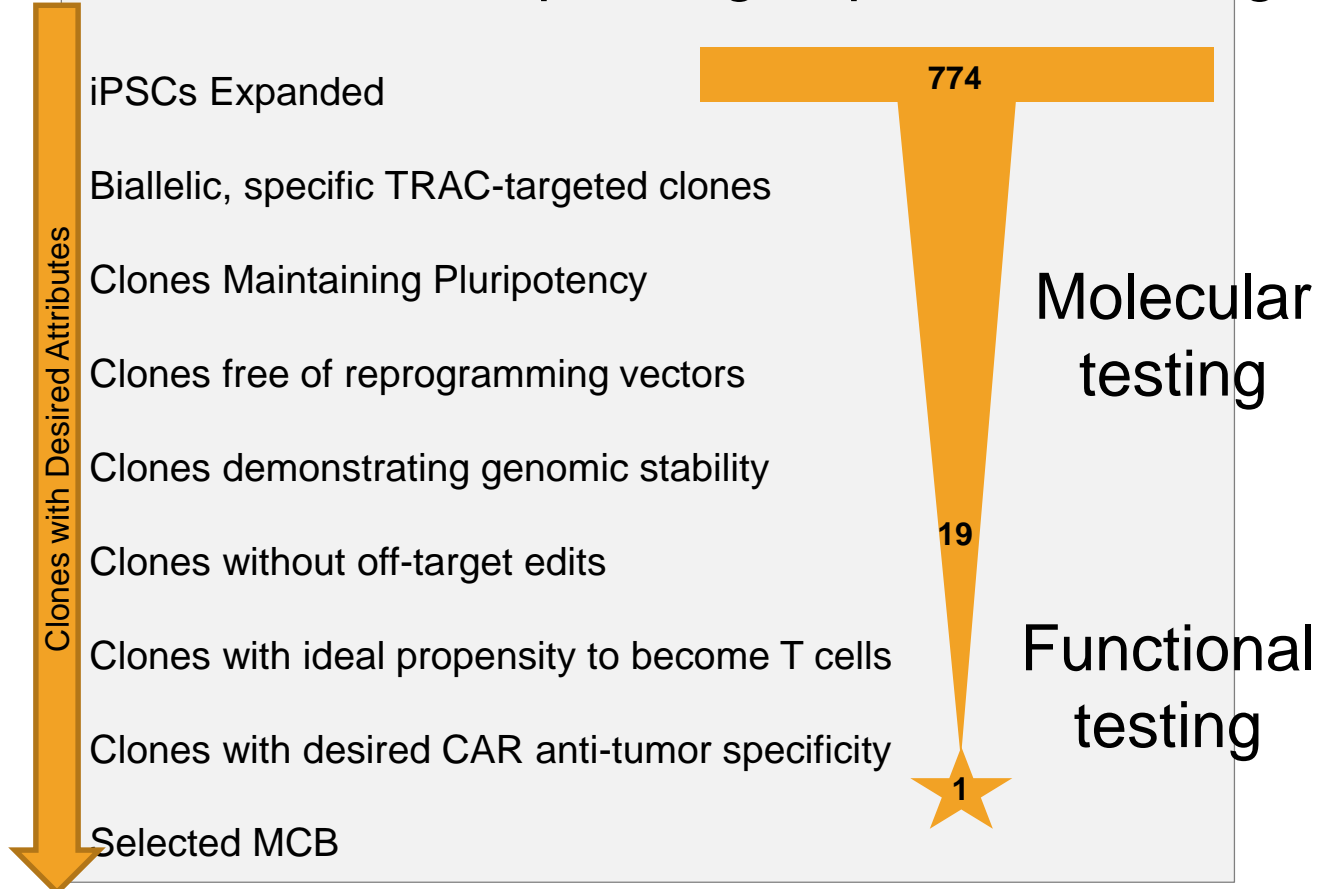
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TCCTAACCTGATCCTCTTGTCCACAGATATCCAGAACCTGACCCCTGCCGTGTACCAGCTGAGAGACTCTAAATCCAG...
AGGATTGGGACTAGGAGAACAGGGTGTCTATAGGCTCTGGGACTGGGACGGCACATGGTCGACTCTCTGAGATTAGGTC...
TRAC exon 1



1. Regulated CAR expression for enhanced function
2. Elimination of TCR expression to avoid GvHD
3. Introduce next gen CAR1XX for optimized CAR signaling

Number of clones passing in-process testing



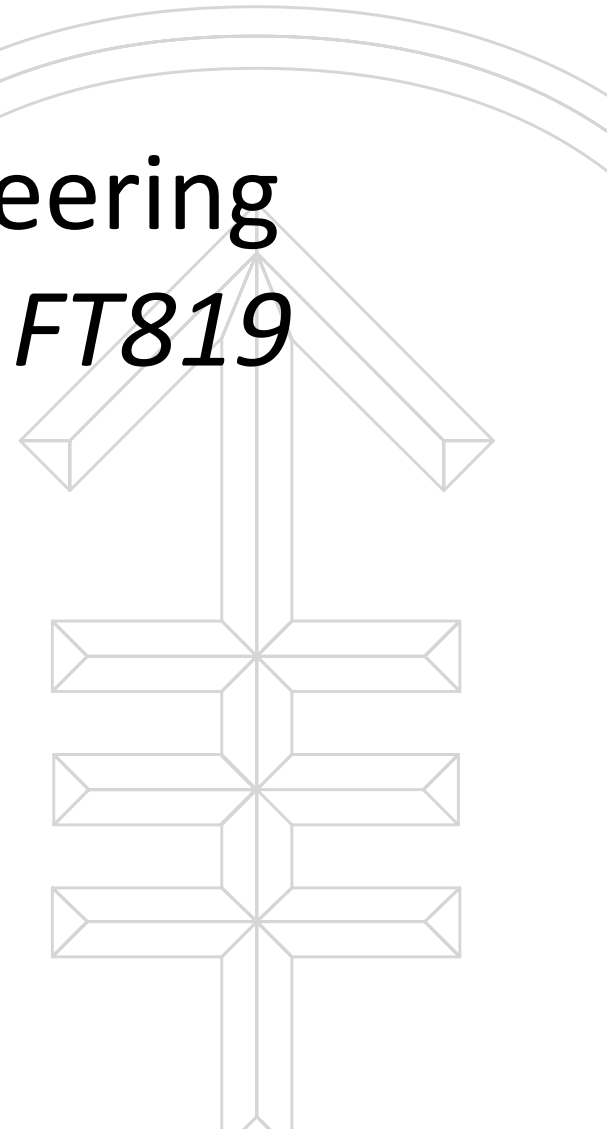


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New Directions in CAR T Cell Engineering

From current CD19 CAR therapy to *FT819*

Michel Sadelain, MD, PhD
Director, Center for Cell Engineering
Memorial Sloan Kettering Cancer Center
New York, NY



The rise of CAR T cell therapy (1)

Human T-lymphocyte cytotoxicity and proliferation directed by a single chimeric TCR ζ /CD28 receptor

John Maher, Renier J. Brentjens, Gertrude Gunset, Isabelle Rivière, and Michel Sadelain*
Nature Biotechnology, 2002

Eradication of systemic B-cell tumors by genetically targeted human T lymphocytes co-stimulated by CD80 and interleukin-15

RENIER J. BRENTJENS¹, JEAN-BAPTISTE LATOUCHE¹, ELMER SANTOS^{1,2}, FRANCESC MARTI⁵,
MICHAEL C. GONG¹, CLAY LYDDANE^{1,3}, PHILIP D. KING⁵, STEVEN LARSON²,
MARK WEISS¹, ISABELLE RIVIÈRE^{1,3,4} & MICHEL SADELAIN^{1,3,4}

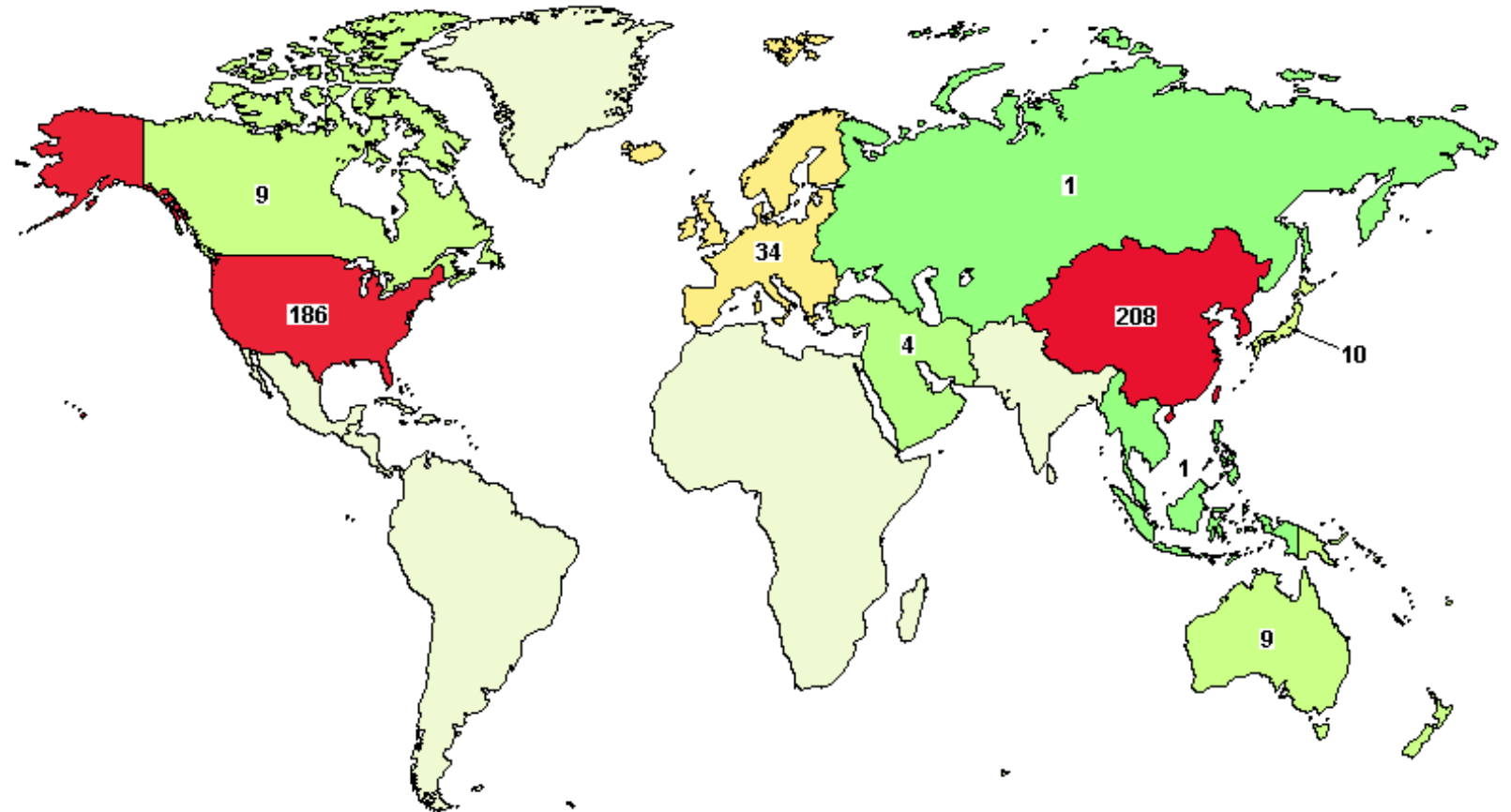
Nature Medicine, 2003



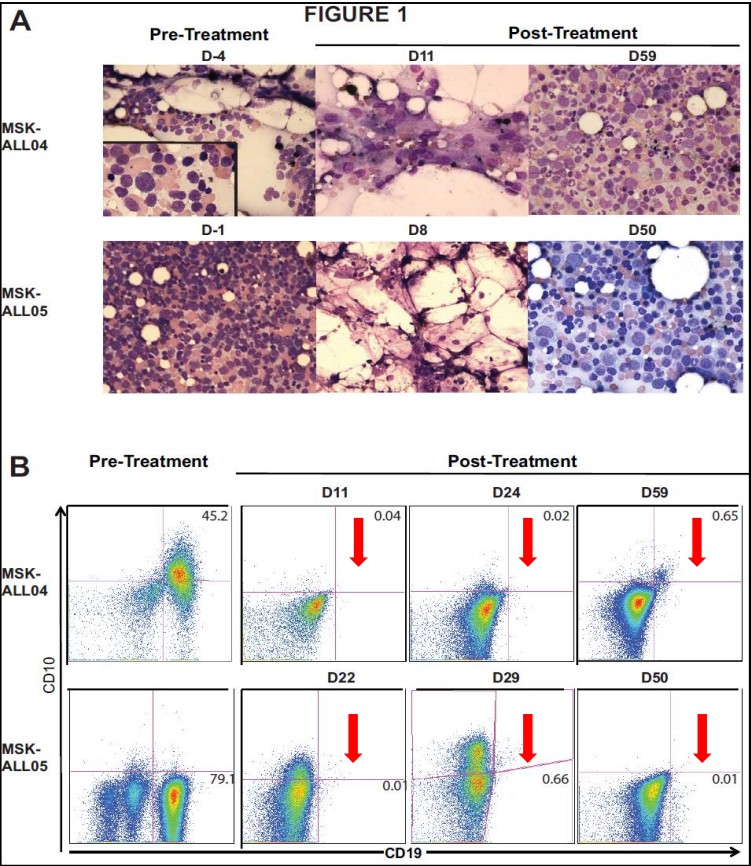
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The rise of CAR T cell therapy (2)

2019
430 studies registered
at clinicaltrials.gov



Rapid and complete eradication of refractory leukemia by 19-28z CAR T cells



Brentjens, Davila, Rivière *et al*,
Science Transl Med, March 2013



Breakthrough of the year
Science, December 2013

Table 1. Responses to CAR T-Cell Therapy.*

Disease	Response Rate percent	Comments	Reference
Leukemia			
B-cell acute lymphoblastic leukemia (in adults)	83–93	High initial remission rates; unresolved issue is whether CAR T-cell therapy is definitive therapy or should be followed by allogeneic hematopoietic stem-cell therapy	Park et al., ³¹ Davila et al., ³⁶ Turtle et al. ³⁷
B-cell acute lymphoblastic leukemia (in children)	68–90	Approximately 25% of patients reported to have a relapse with CD19-negative or CD19-low leukemia; CD22 CAR T cells may improve survival among some patients with CD19 relapses	Maude et al., ³⁴ Maude et al., ³⁸ Fry et al., ³⁹ Lee et al. ⁴⁰
Chronic lymphocytic leukemia	57–71	Relapse is rare in patients who have a complete response; ibrutinib appears to increase response rates	Porter et al., ⁴¹ Turtle et al. ⁴²
Lymphoma			
Diffuse large B-cell lymphoma	64–86	Approximately 40–50% of patients reported to have a durable complete response	Turtle et al., ⁴³ Kochenderfer et al., ⁴⁴ Schuster et al., ⁴⁵ Neelapu et al. ⁴⁶
Follicular lymphoma	71	At a median follow-up of 28.6 mo, the response was maintained in 89% of patients who had a response	Schuster et al. ⁴⁵
Transformed follicular lymphoma	70–83	A total of 3 of 3 patients with transformed follicular lymphoma had a complete response	Turtle et al., ⁴³ Schuster et al., ⁴⁵ Neelapu et al. ⁴⁶
Refractory multiple myeloma	25–100	B-cell maturation antigen CAR T cells; stringent complete response in approximately 25% of patients	Ali et al., ⁴⁷ Fan et al., ⁴⁸ Berdeja et al. ⁴⁹
Solid tumors			
Glioblastoma	ND	(q4) In case report from phase 2 study, complete response on magnetic resonance imaging after intravenous and cerebrospinal fluid administration of CAR T cells; complete response lasted 7.5 mo	Brown et al. ⁵⁰
Pancreatic ductal adenocarcinoma	17	In one patient with liver metastasis, CAR T-cell treatment produced a complete metabolic response in the liver but was ineffective against the primary pancreatic tumor	Beatty et al. ⁵¹

* ND denotes not determined.

June and Sadelain,
N Engl J Med, 2018



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Rapid and complete eradication of refractory leukemia by 19-28z CAR T cells

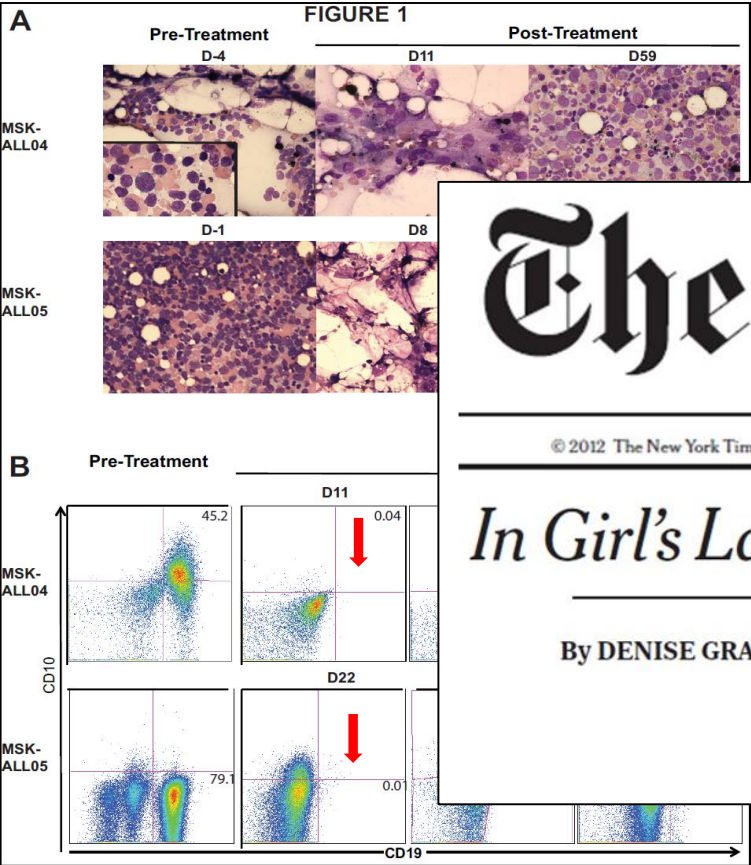


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		Approximately 25% of patients reported to have a relapse with CD19-negative or CD19-low leukemia; CD22 CAR T cells may improve survival among some patients with CD19 relapses	Maude et al., ³⁴ Maude et al., ³⁸ Fry et al., ³⁹ Lee et al., ⁴⁰
		Relapse is rare in patients who have a complete response; ibrutinib appears to increase response rates	Porter et al., ⁴¹ Turtle et al., ⁴²
		Approximately 40–50% of patients reported to have a durable complete response	Turtle et al., ⁴³ Kochenderfer et al., ⁴⁴ Schuster et al., ⁴⁵ Neelapu et al., ⁴⁶
		At a median follow-up of 28.6 mo, the response was maintained in 89% of patients who had a response	Schuster et al. ⁴⁵
		A total of 3 of 3 patients with transformed follicular lymphoma had a complete response	Turtle et al., ⁴³ Schuster et al., ⁴⁵ Neelapu et al., ⁴⁶
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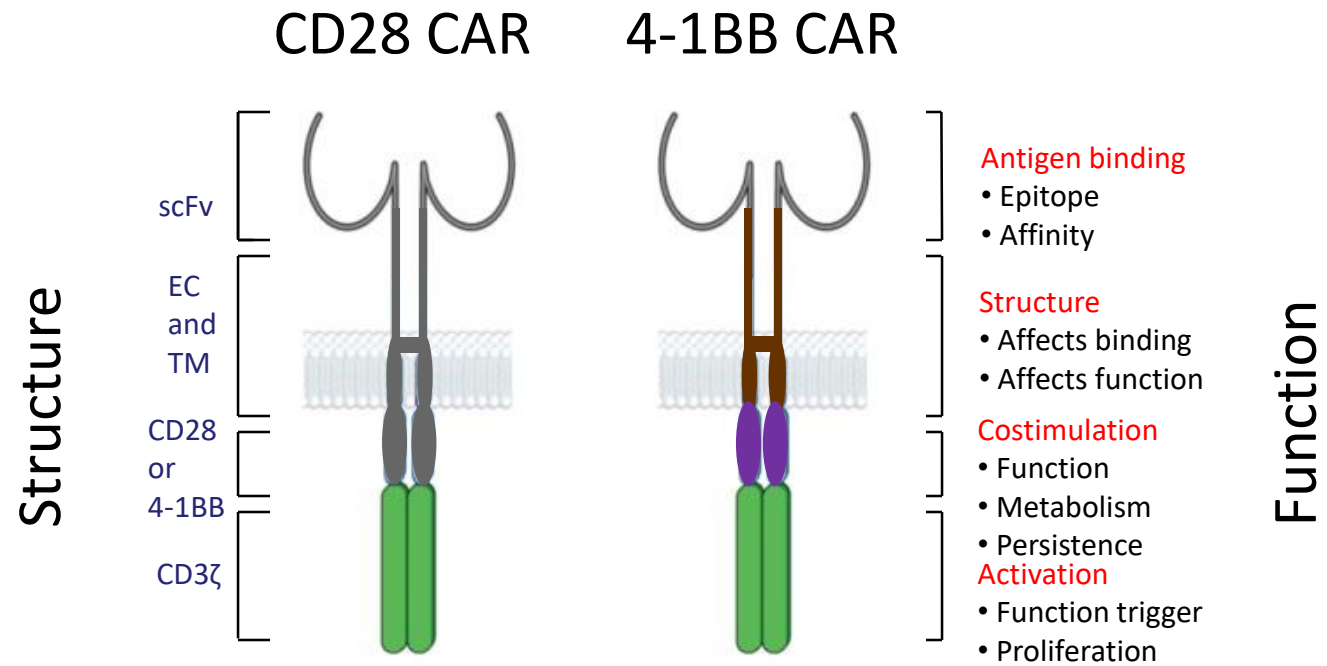
Brentjens, Davila, Rivière *et al*,
Science Transl Med, March 2013

Breakthrough of the year
Science, December 2013

June and Sadelain,
N Engl J Med, 2018



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FDA approved in 2017

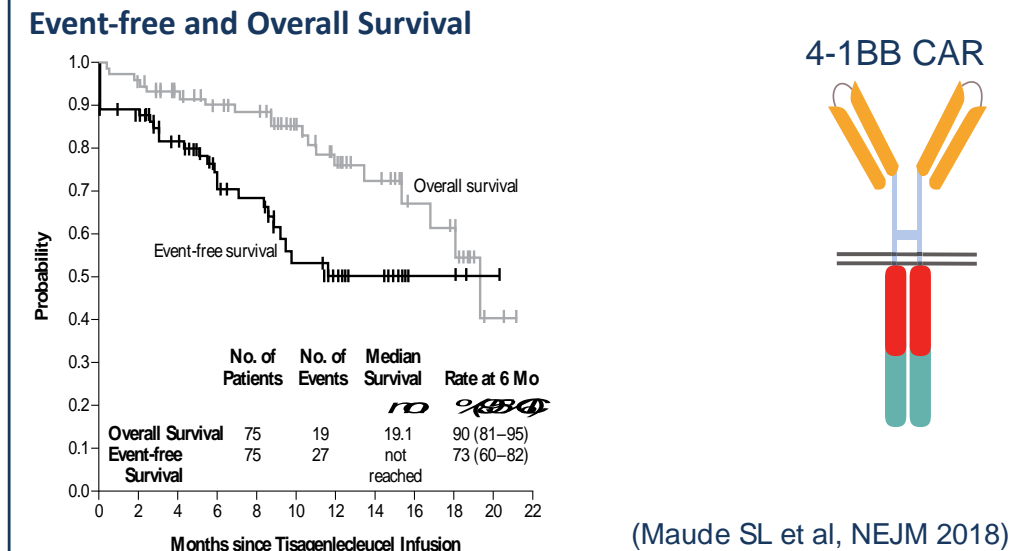
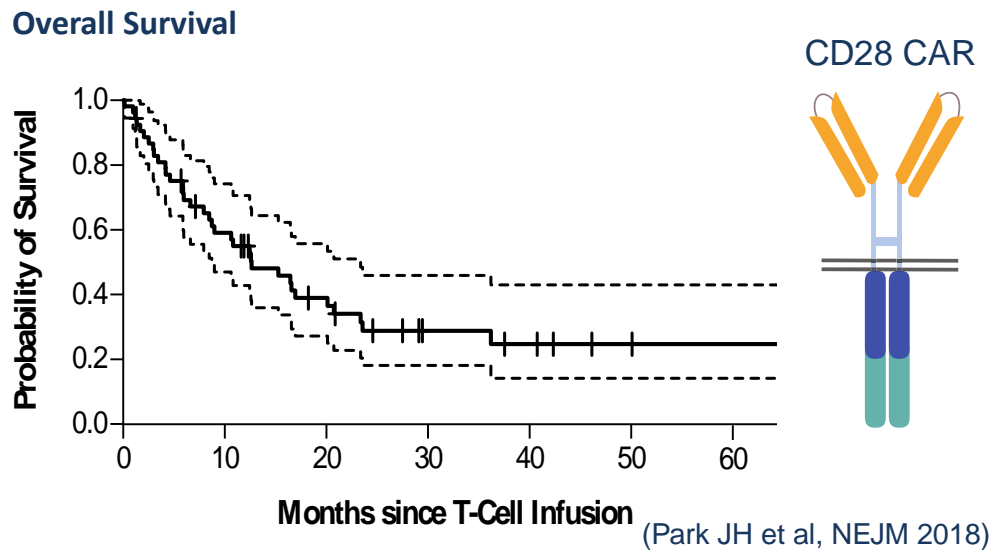
Axicabtagene Ciloleucel (Yescarta)	Tisagenlecleucel (Kymriah)
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Prototypic CD19 CARs

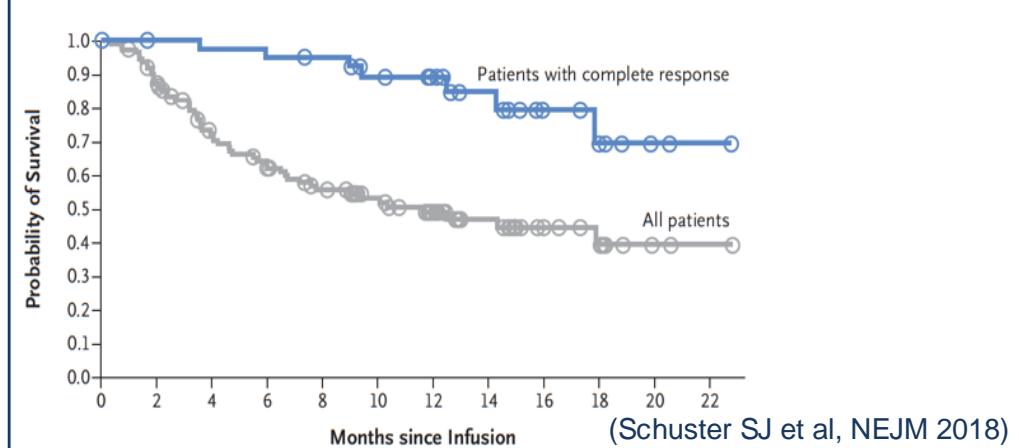
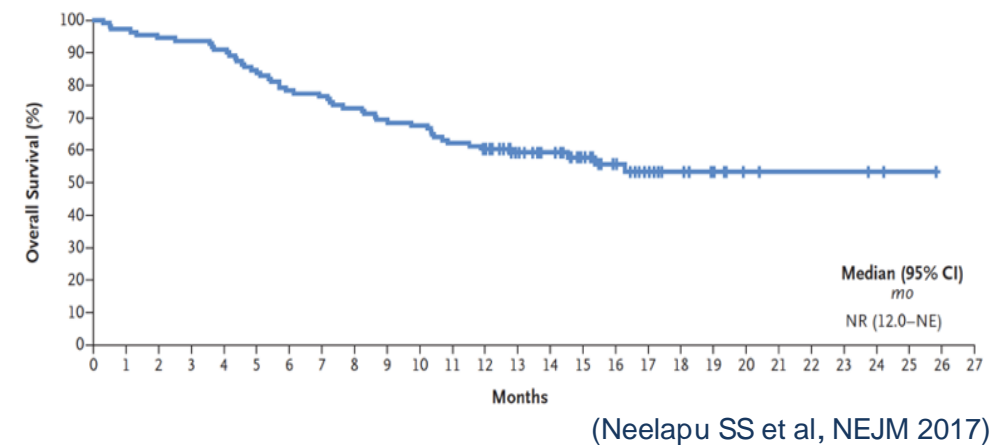


Clinical Success in B cell leukemia and lymphoma

ALL



Lymphoma



Increasing CAR therapy efficacy and availability

1. Regulated CAR expression
2. Titrated CAR signaling
3. “Off-the-shelf” TiPSC – derived CAR T cell production

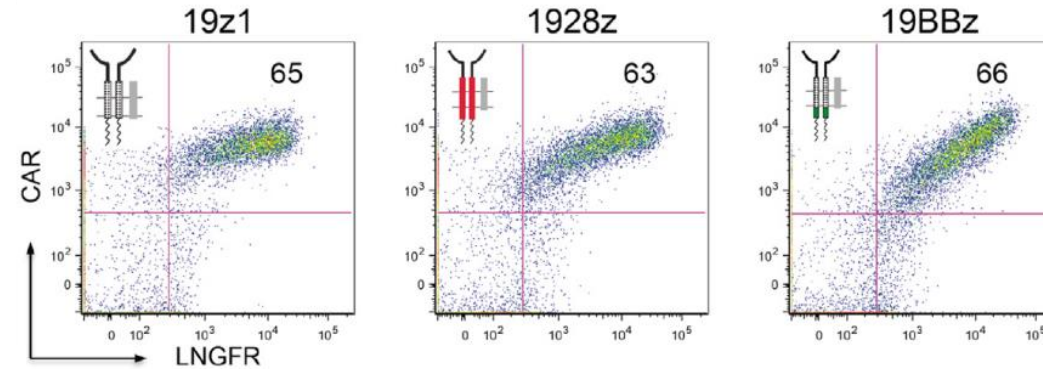


Viral vector integration – variegated expression

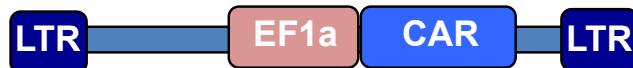
MLV-based vector



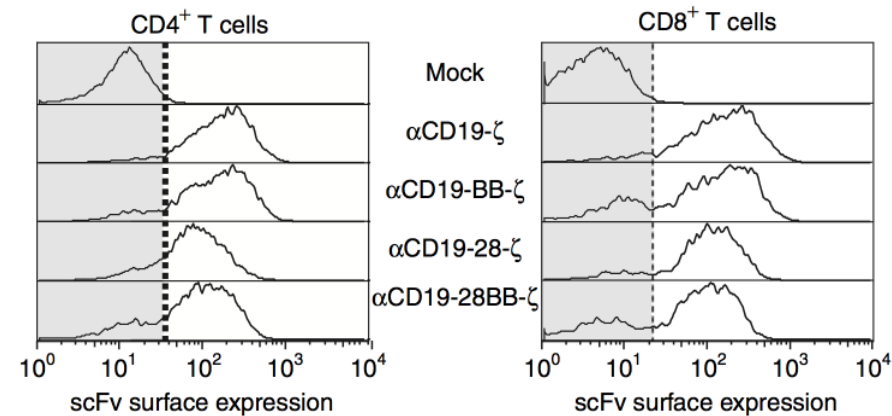
Zhao et al., Cancer Cell, 2015



HIV-based vector

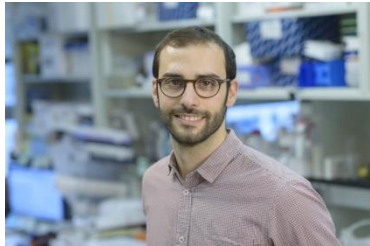
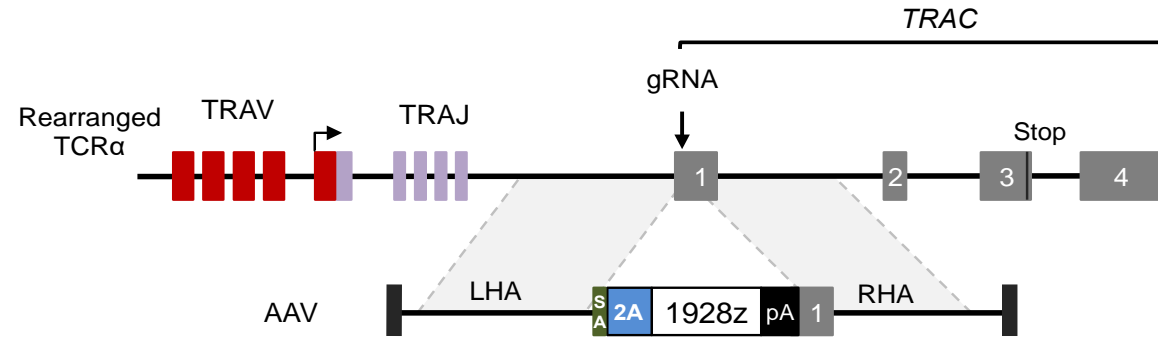
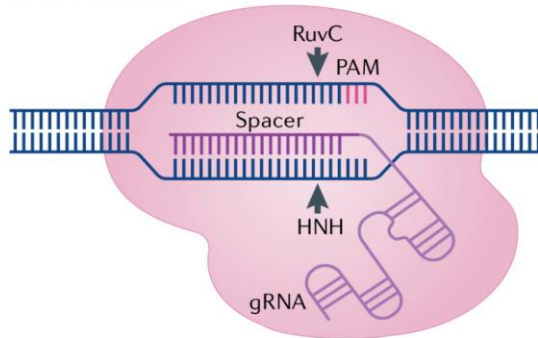


Milone et al., Mol. Therapy, 2009



CRISPR/Cas9-targeted integration into the *TRAC* locus

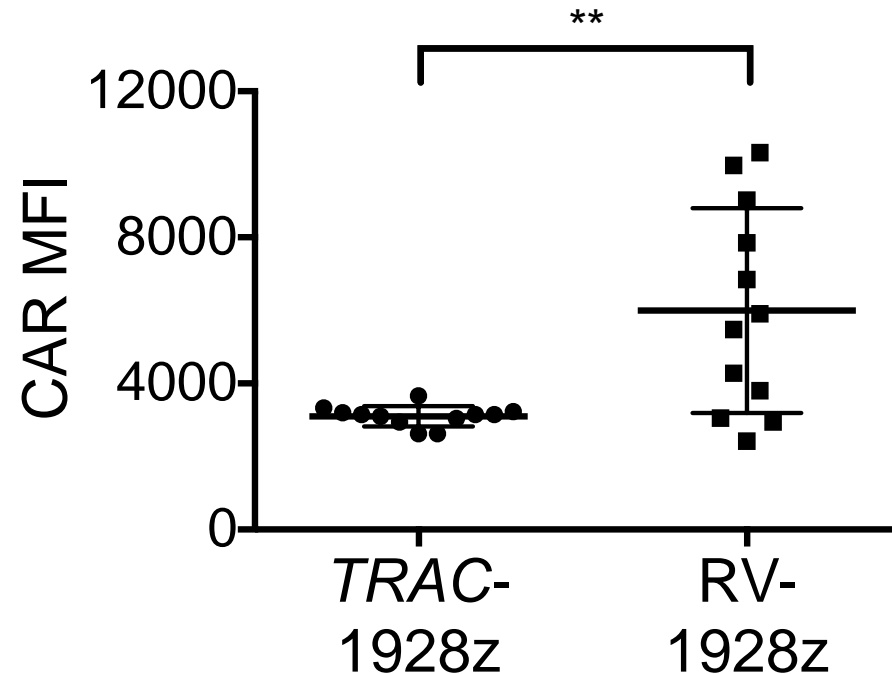
a Cas9 nuclease



Justin Eyquem, PhD



J. Mansilla-Soto, PhD



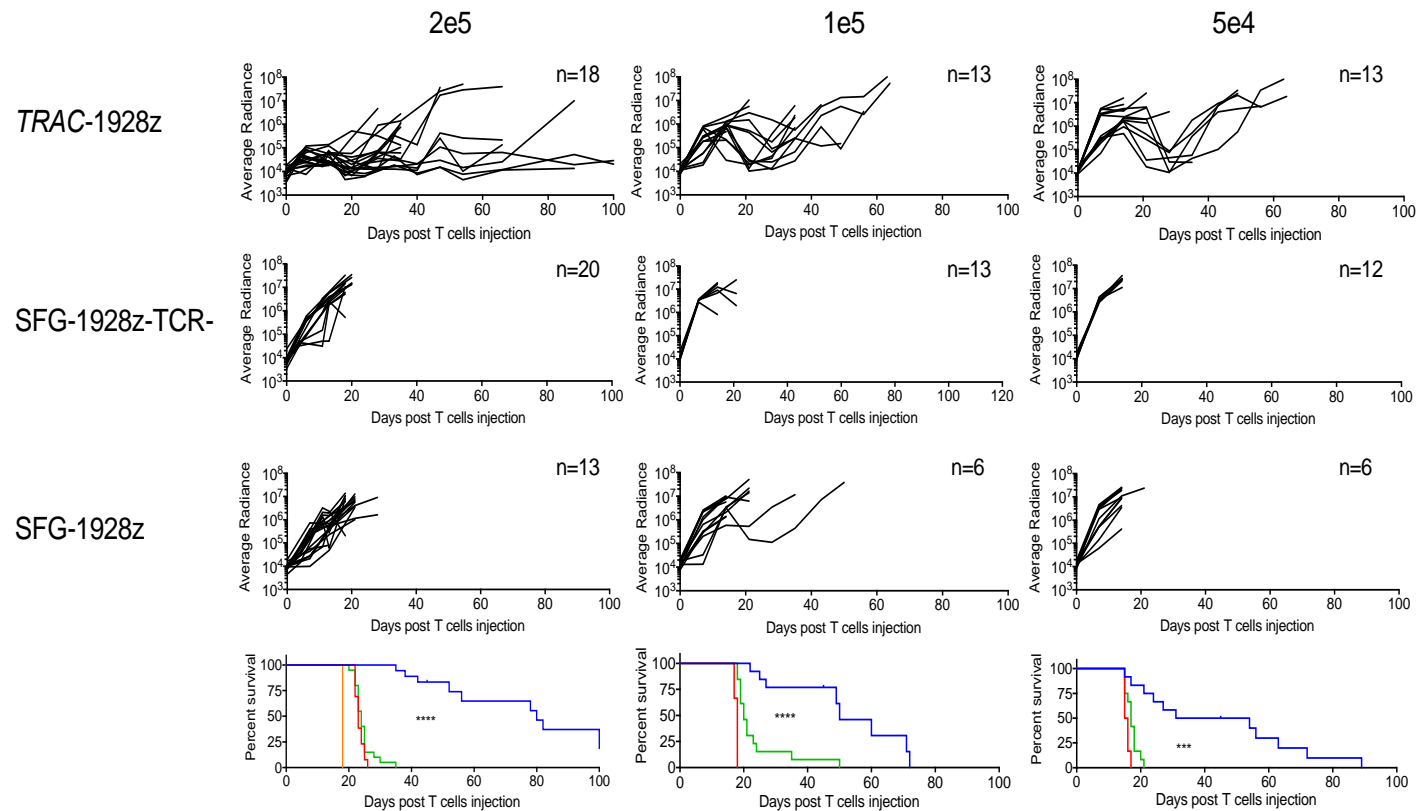
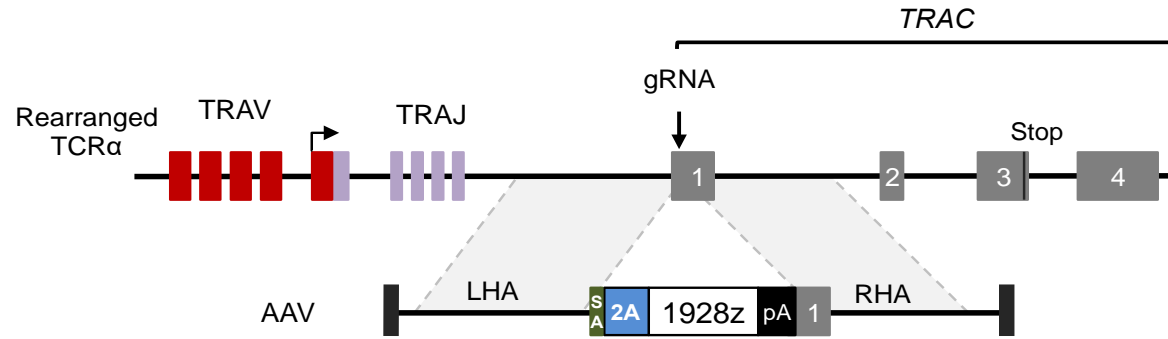
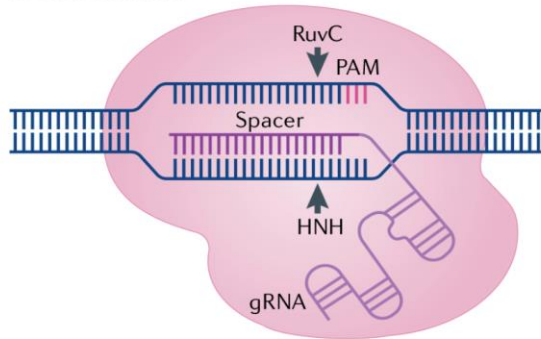
Eyquem, Mansilla-Soto
et al, *Nature*, 2017



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CRISPR/Cas9-targeted integration into the *TRAC* locus

a Cas9 nuclease



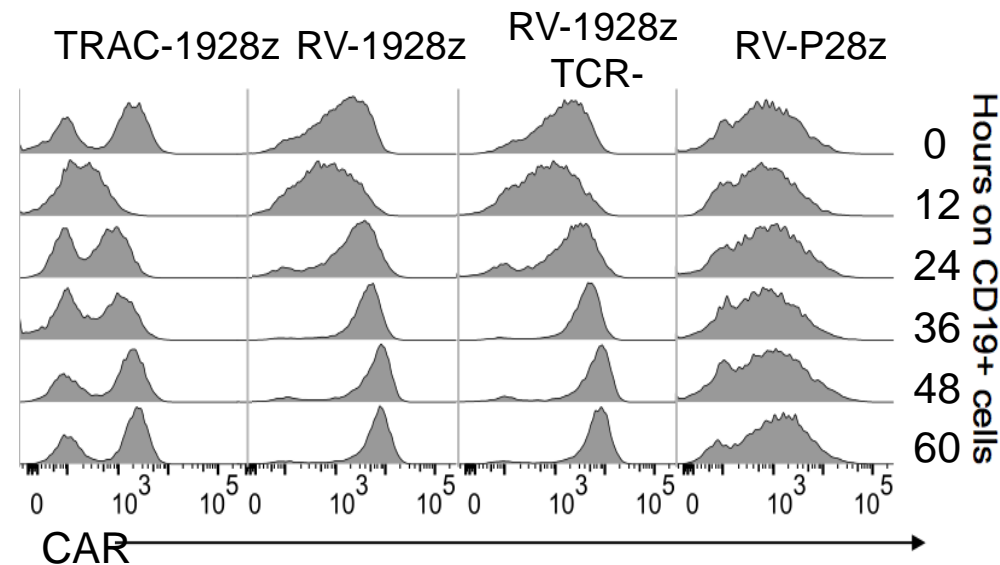
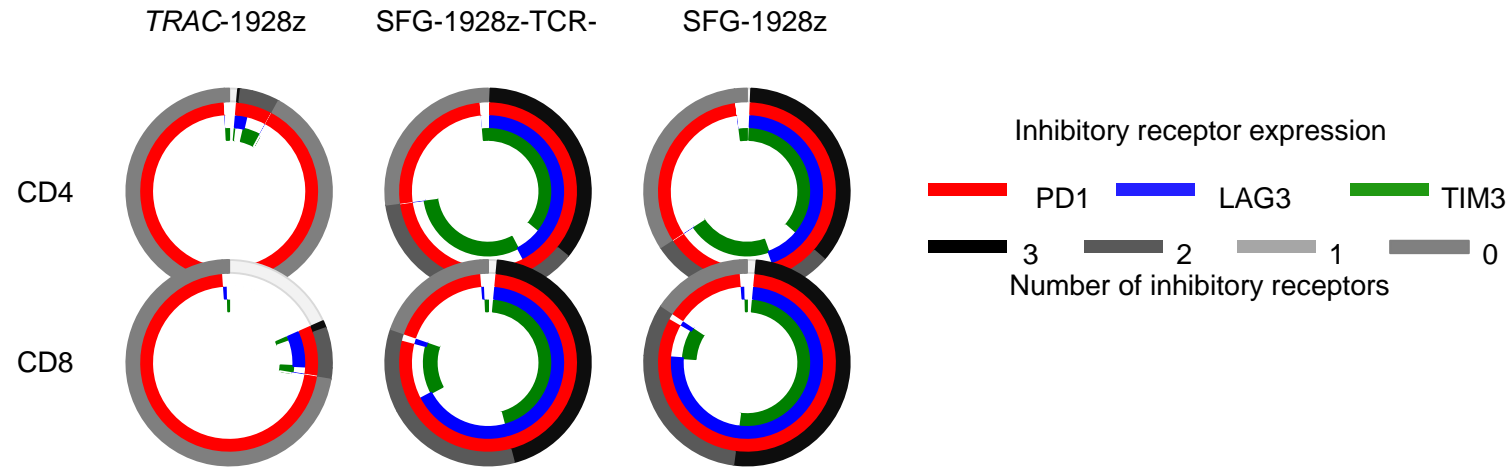
Eyquem, Mansilla-Soto
et al, *Nature*, 2017



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TRAC-CAR T cells are less exhausted in vivo

CAR surface expression is down-regulated upon exposure to antigen

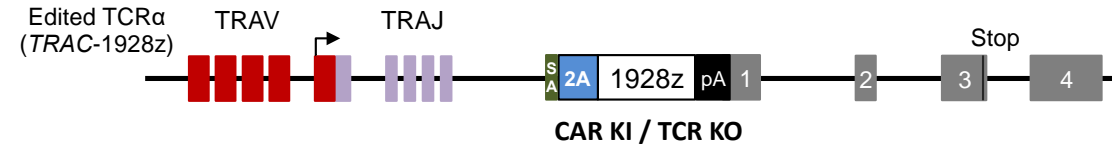


Eyquem, Mansilla-Soto
et al, *Nature*, 2017



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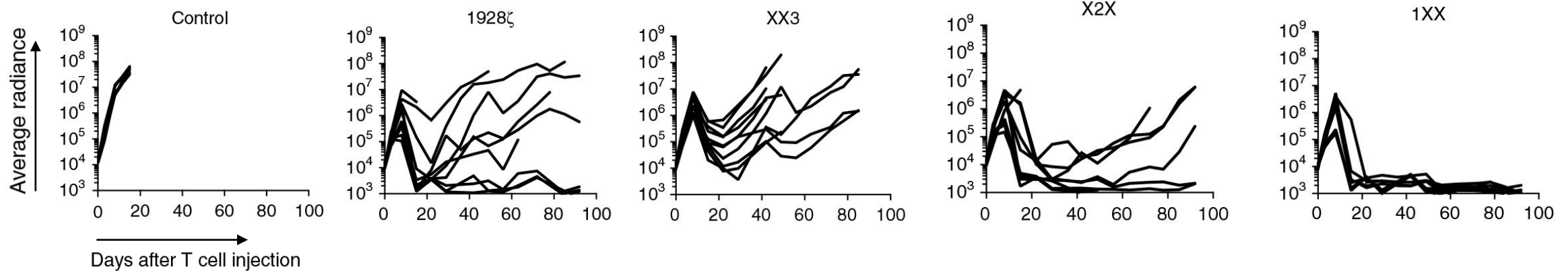
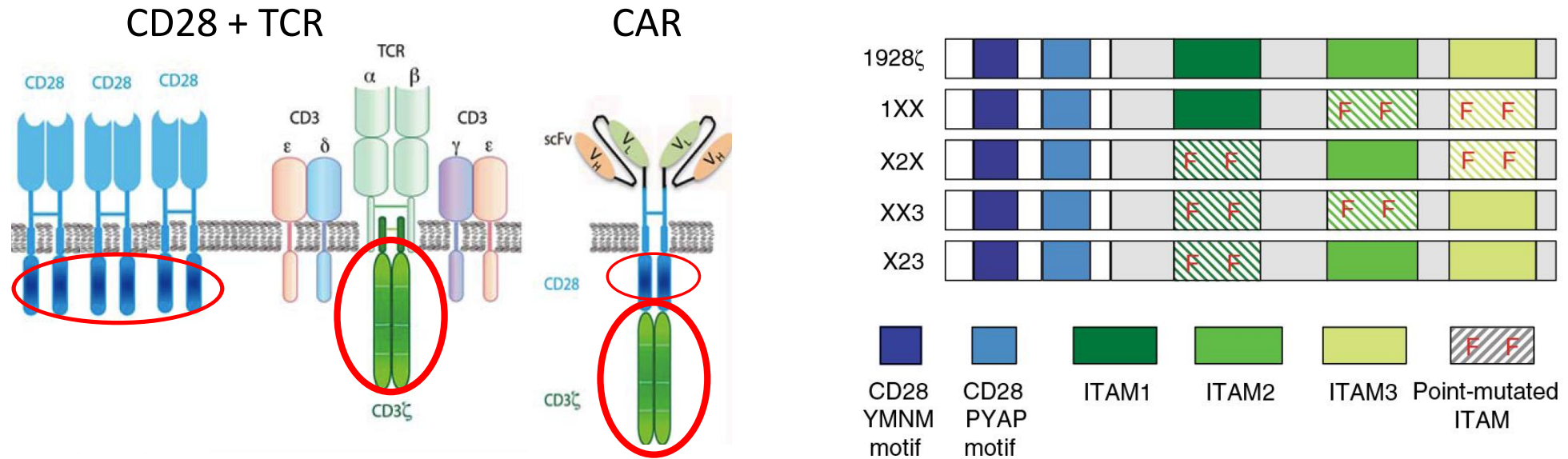
Summary *TRAC*-CAR



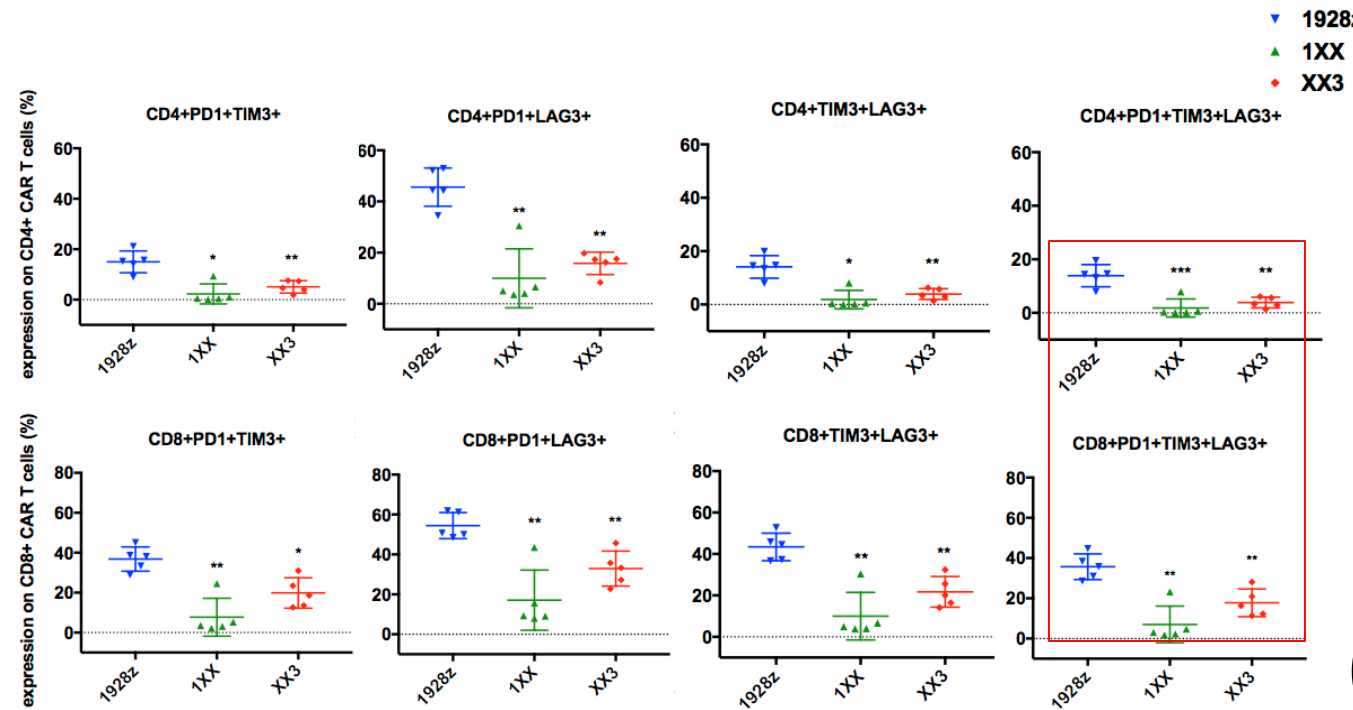
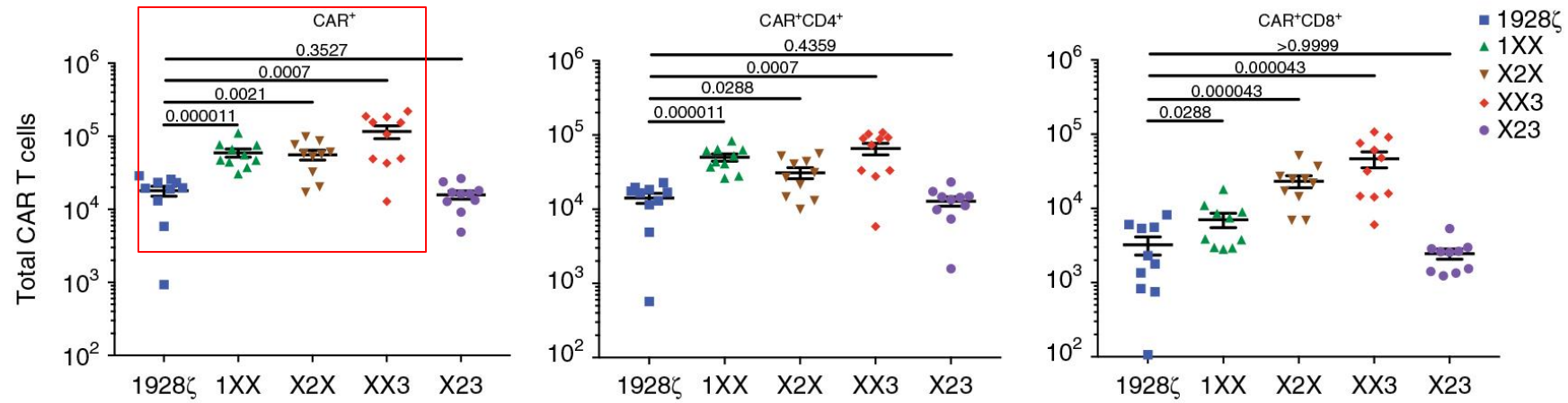
- *TRAC*-1928z T cells show enhanced *in vivo* anti-tumor activity relative to RV/LV CAR T cells
- The TCR alpha promoter provides highly homogeneous and optimal control of 1928z CAR expression
- *TRAC*-1928z sustains functional persistence by averting rapid T cell differentiation and exhaustion
- Established link between CAR expression level and therapeutic activity.
- Simultaneous CAR knock-in and TCR knock-out enables allogeneic application



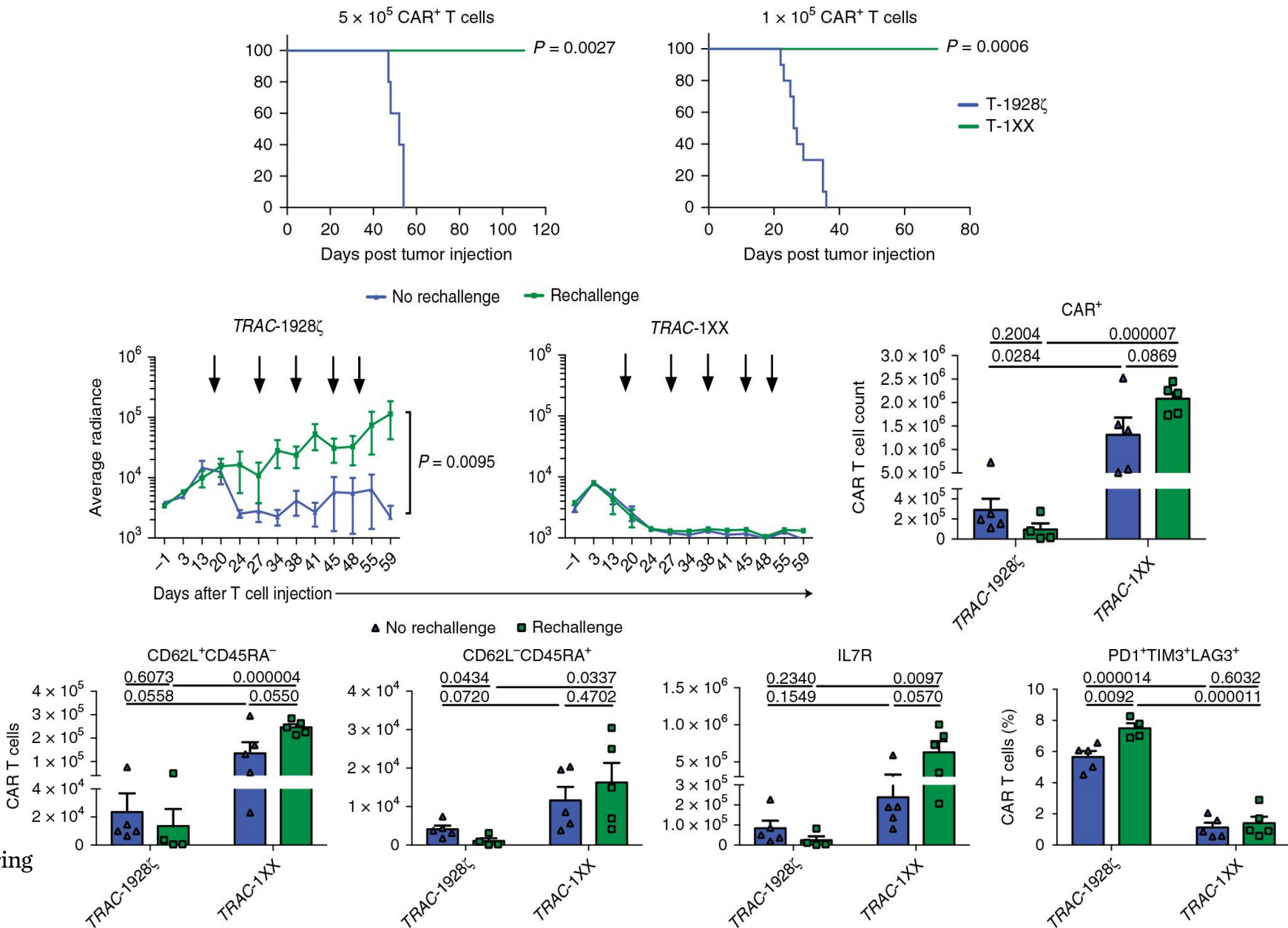
ITAM-based calibration of activation strength in CD28/CD3 ζ CARs



1928ζ ITAMs differentially regulate CAR T cell potency



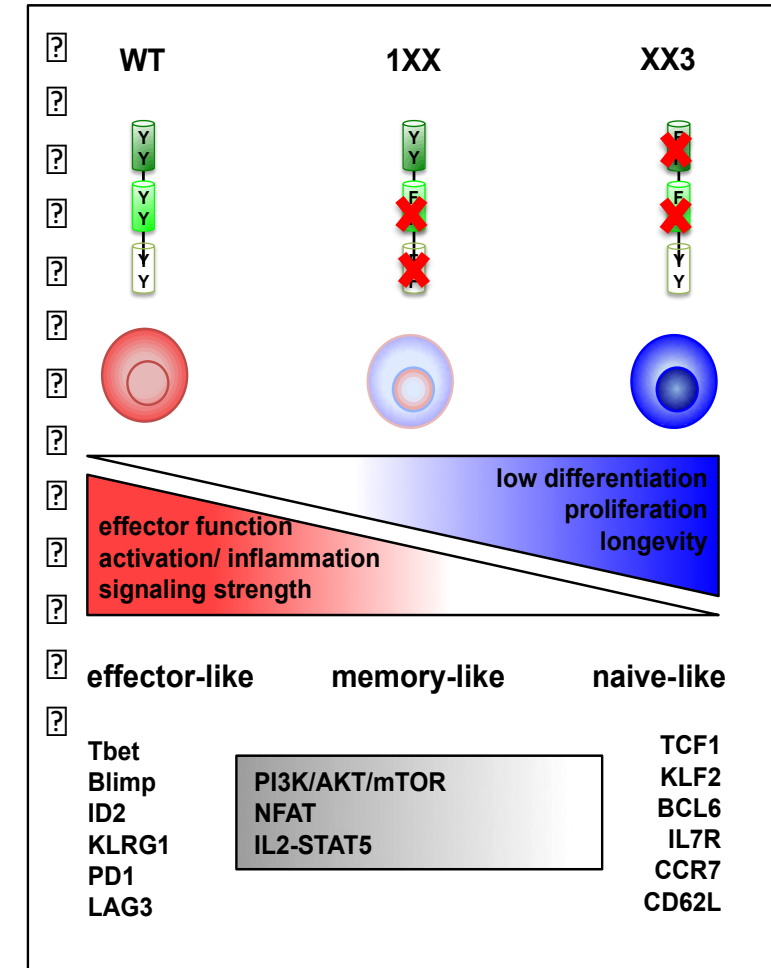
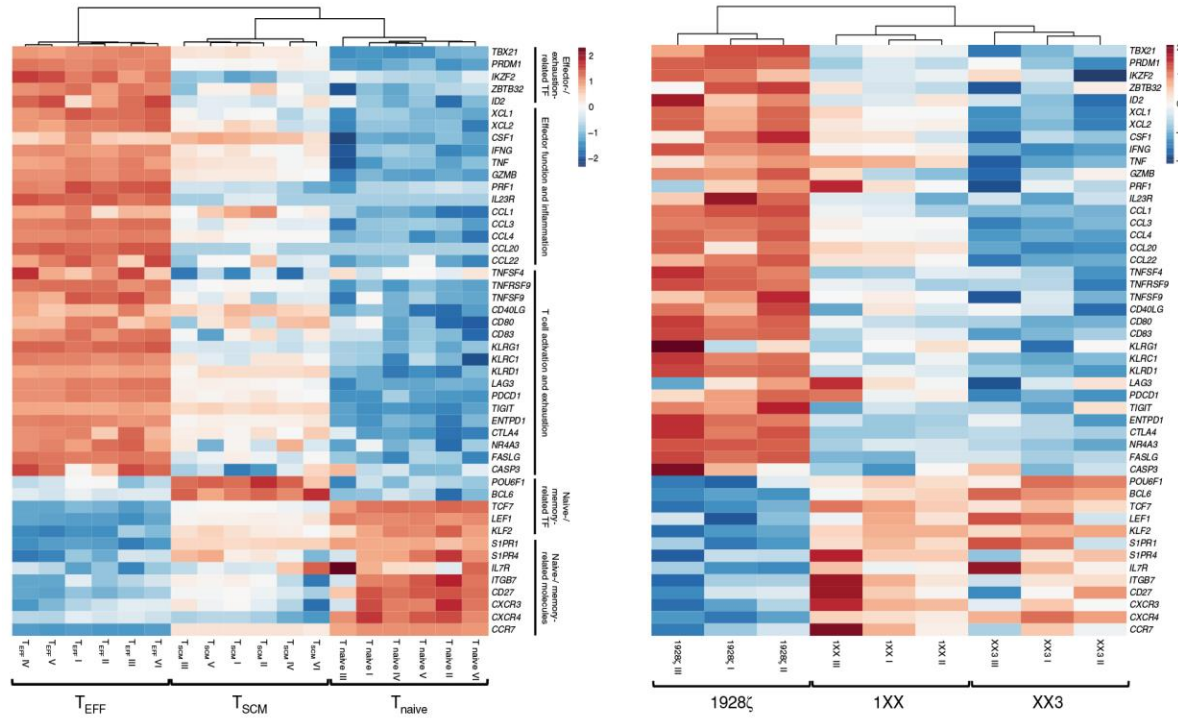
TRAC-1XX CAR T cells promote memory and protect against tumor re-challenge



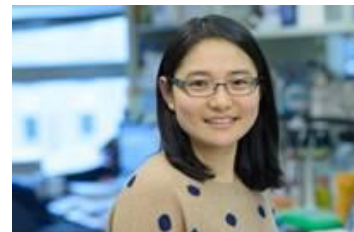
Feucht, Sun,
Nat Med 2019



CAR ITAM-calibration directs T cell fate

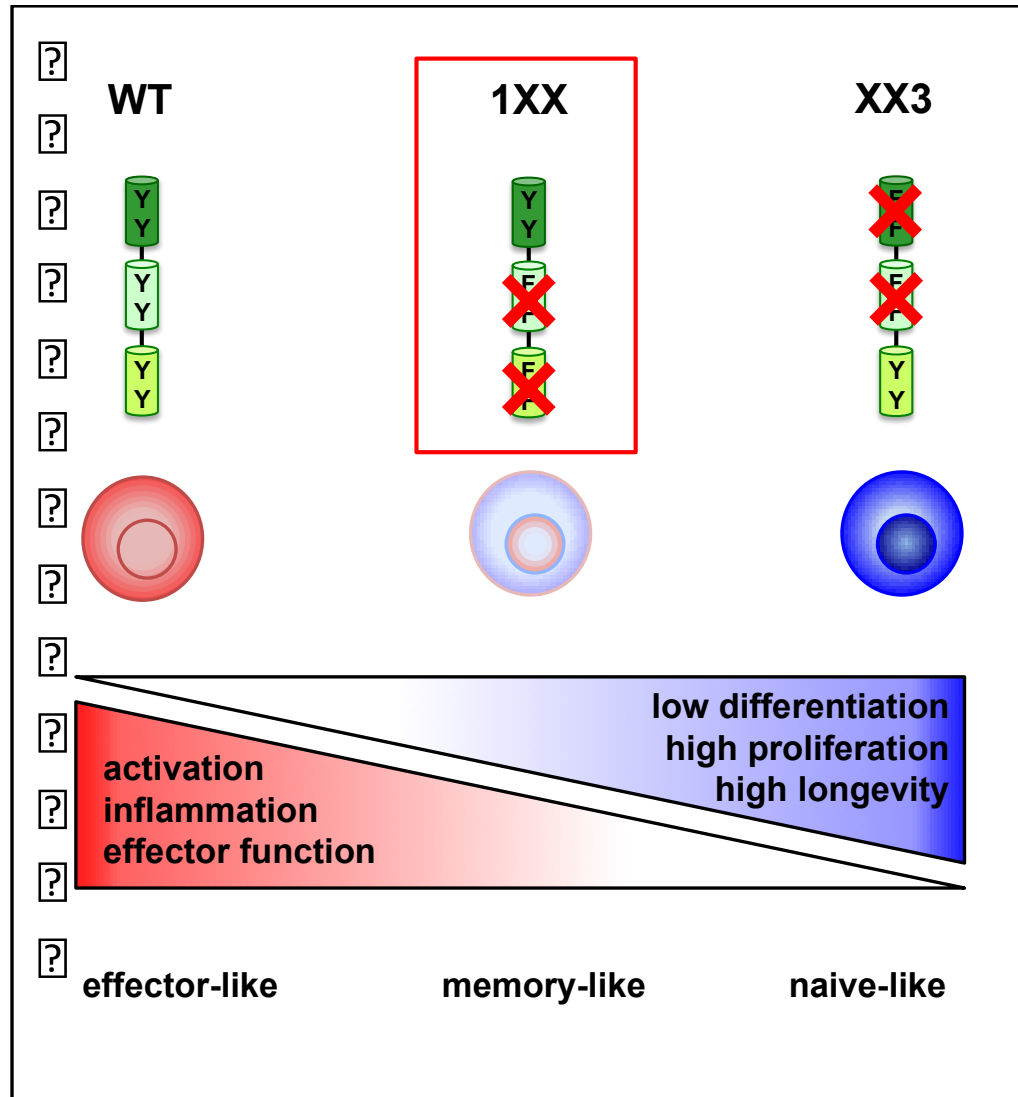


Judith Feucht, MD



Sun Jie, PhD

Summary 1XX

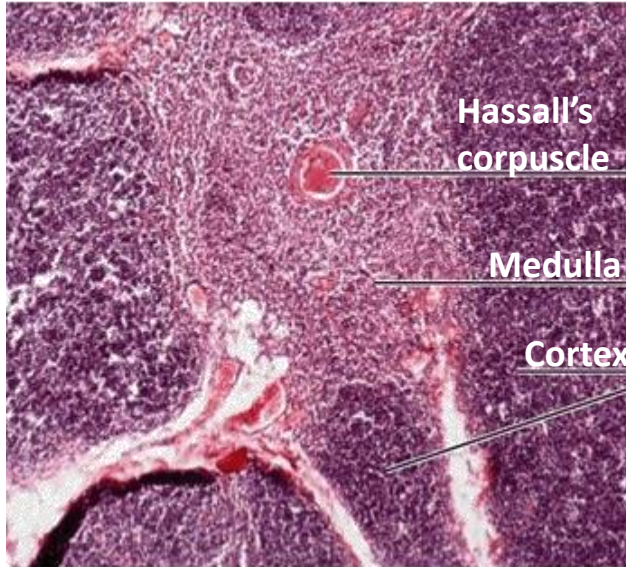


- CD3ζ ITAM domains within 1928ζ CARs direct qualitatively different CAR T cell functions
- ITAM number & position within 1928ζ CARs impact CAR function
- CD3ζ ITAM mutations direct T cells to different fates by balancing effector and memory programs
- 1XX leads to fast & efficient long-term tumor eradication by inducing strong effector function without shutting off memory programs
- Further reducing activation potential (XX3) results in high CAR persistence, but insufficient effector function

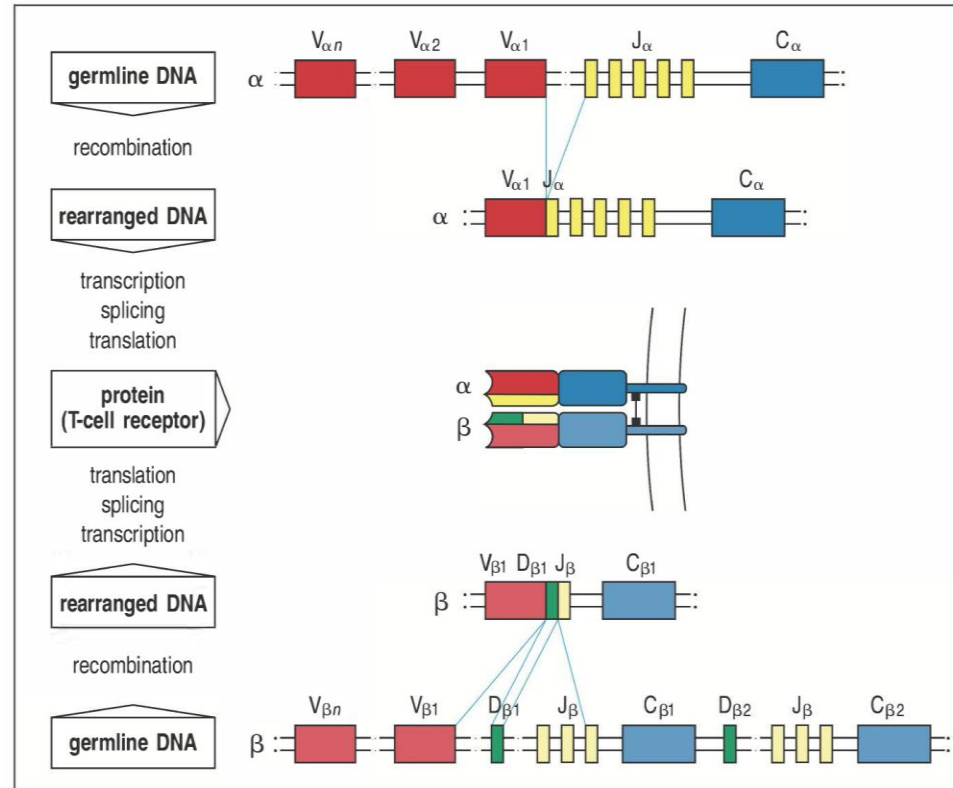


Where do T cells and their natural receptor come from

Thymic origin, VDJ recombination and clonal selection



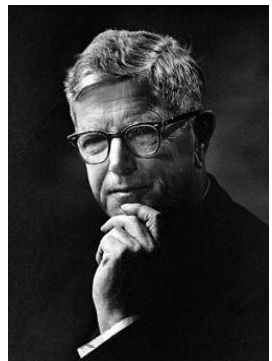
Jacques Miller



Immunobiology: The Immune System in Health and Disease. 5th edition. Janeway CA Jr, Travers P, Walport M, et al. New York: Garland Science; 2001.

The clonal selection theory

- Each lymphocyte bears a single receptor with a unique specificity.
- Interaction between a foreign molecule and a lymphocyte receptor capable of binding that molecule with a high affinity leads to lymphocyte activation and clonal expansion.
- Lymphocytes bearing receptors specific for ubiquitous self molecules are deleted at an early stage in lymphoid cell development and are therefore absent from the repertoire of mature lymphocytes.

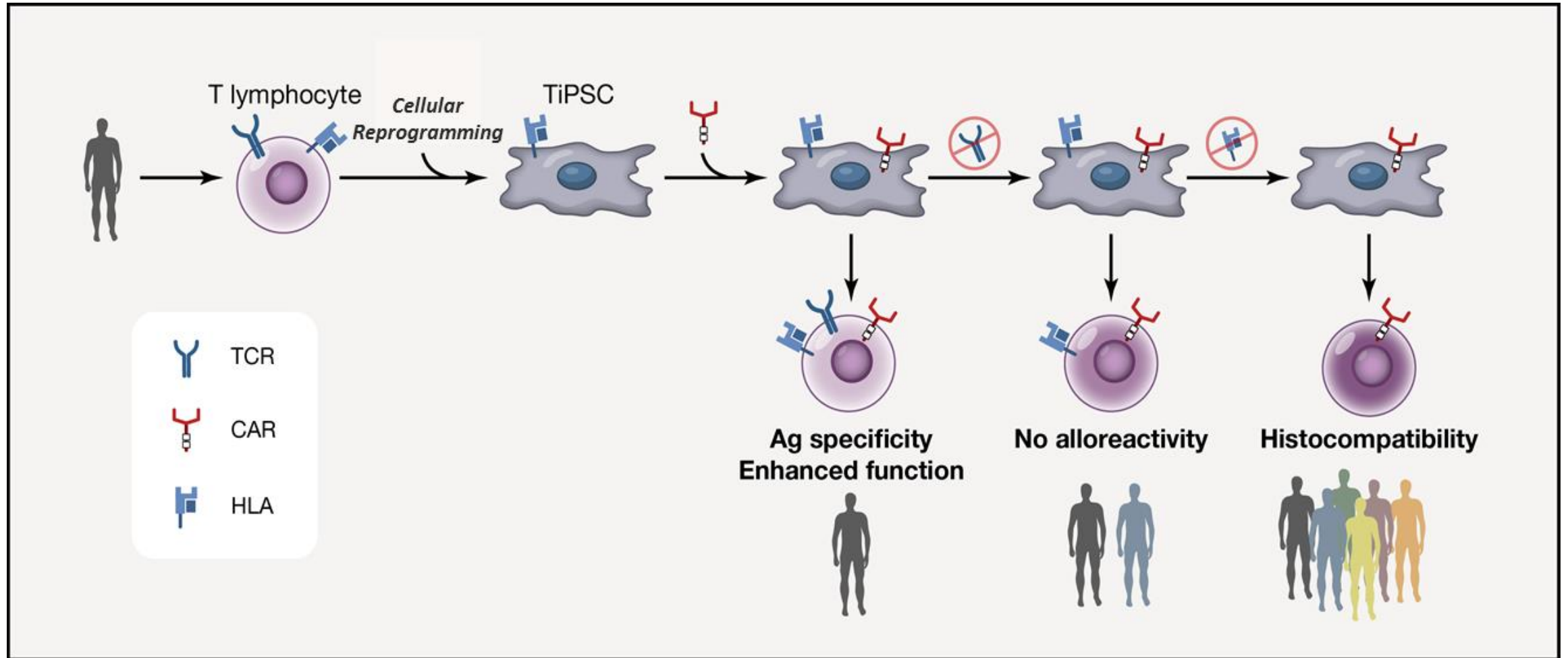


F. Macfarlane Burnet



Memorial Sloan Kettering
Cancer Center

“Off-the-shelf” CAR T cells produced from pluripotent stem cells



Adapted from: Themeli, Riviere & Sadelain, *Cell Stem Cells*, 2015



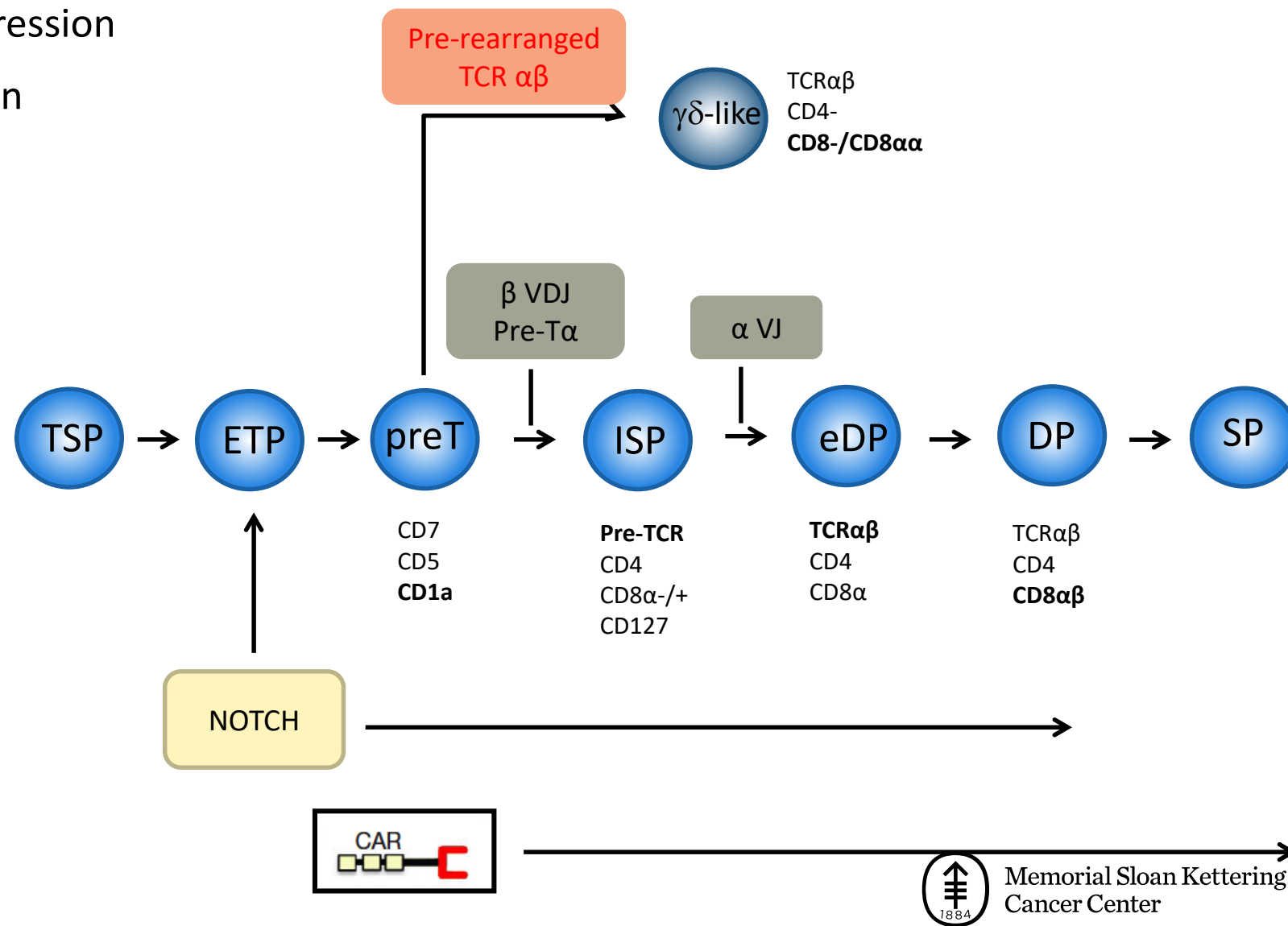
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TiPSC differentiation – reconciling Notch, pre-TCR, TCR and CAR

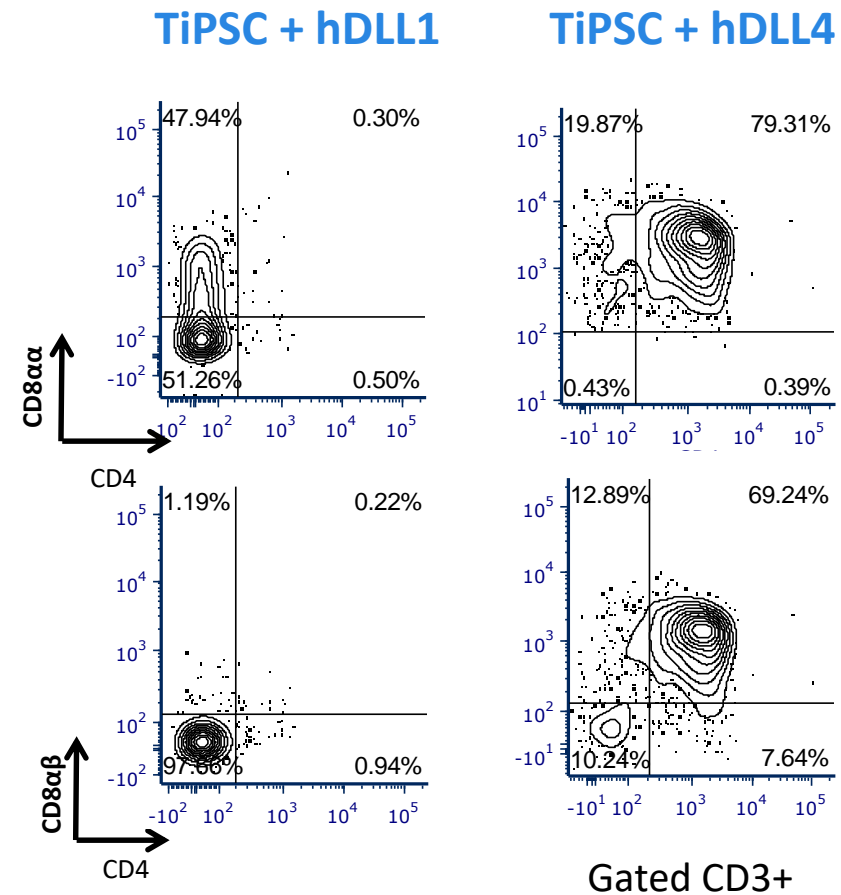
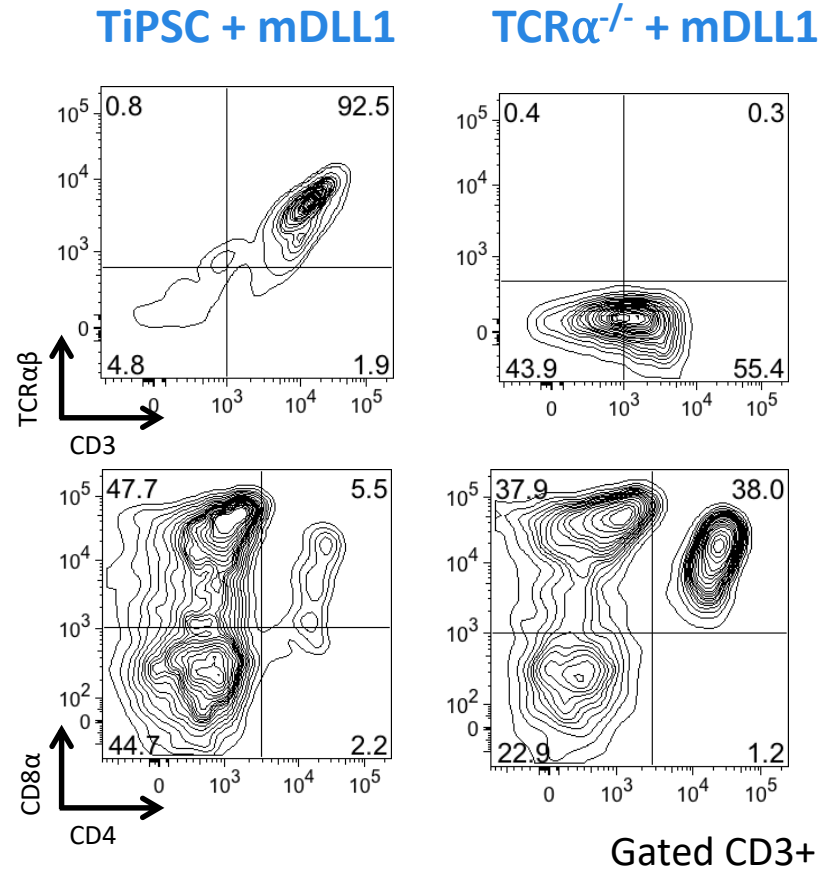
1. Early TCR $\alpha\beta$ expression
2. Notch stimulation
3. CAR expression



Sjoukje van der Stegen



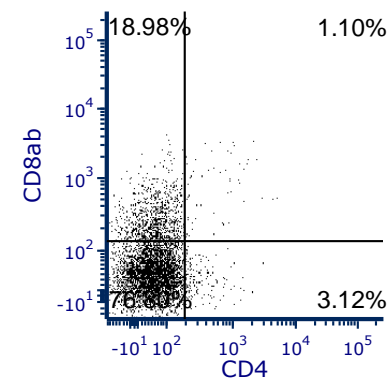
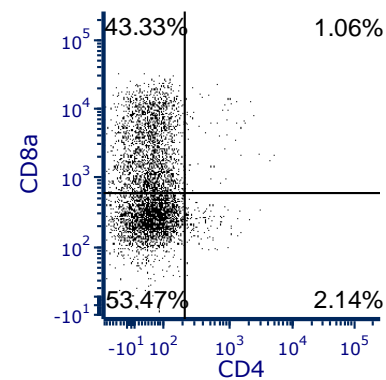
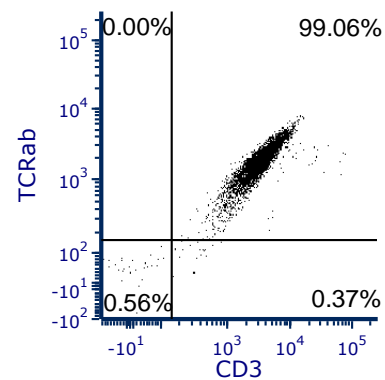
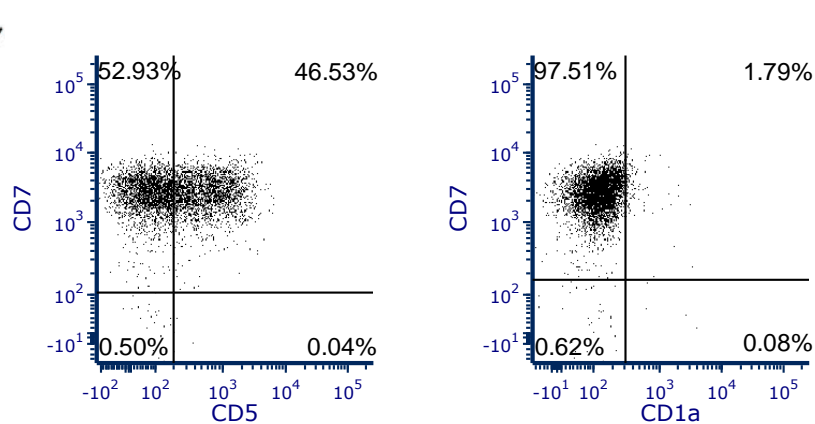
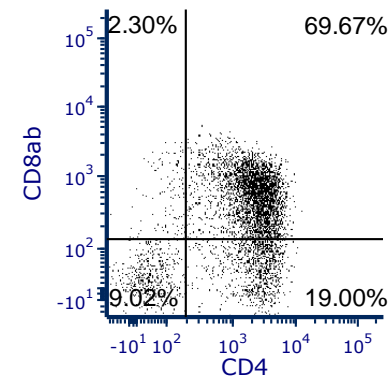
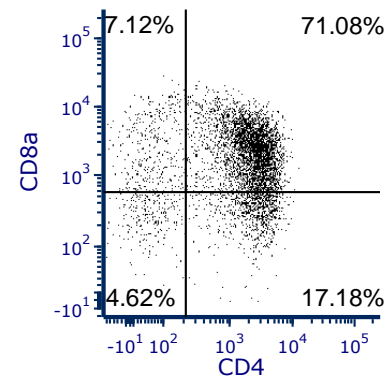
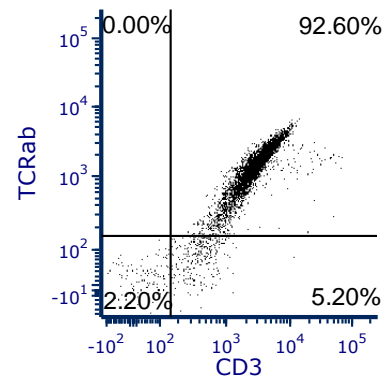
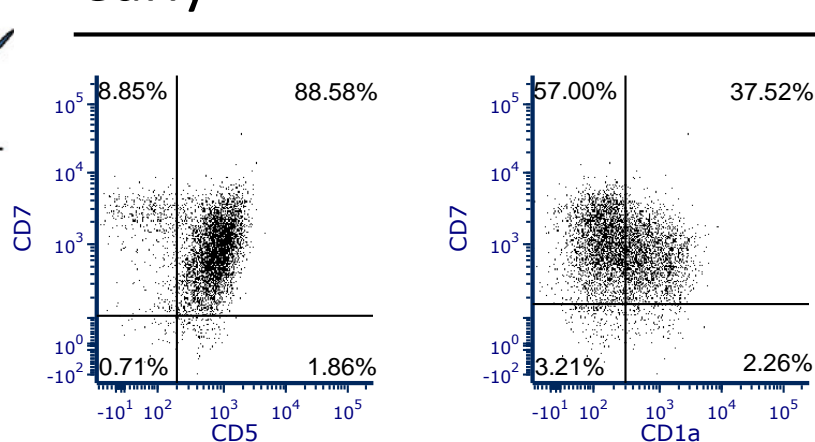
CD4⁺CD8 $\alpha\beta$ ⁺ DP T cell development – Central roles of TCR and Notch signaling



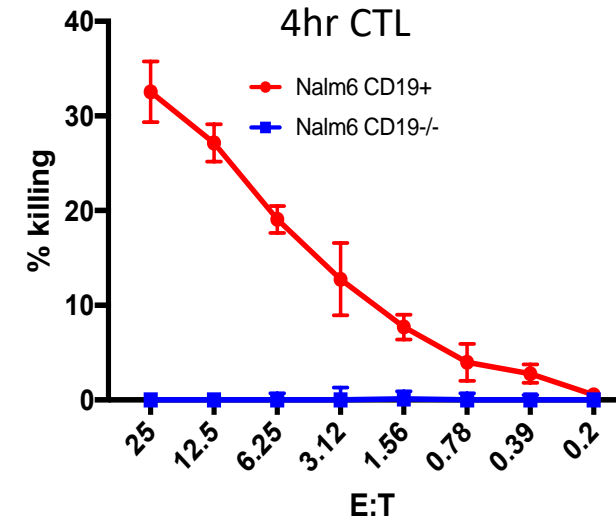
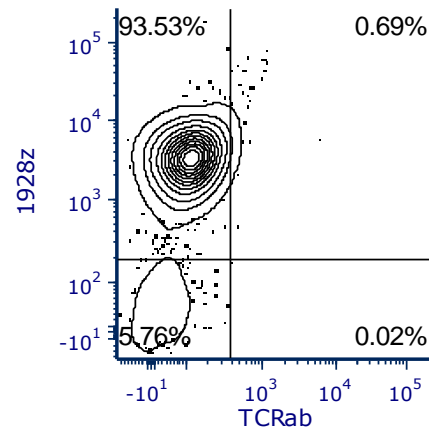
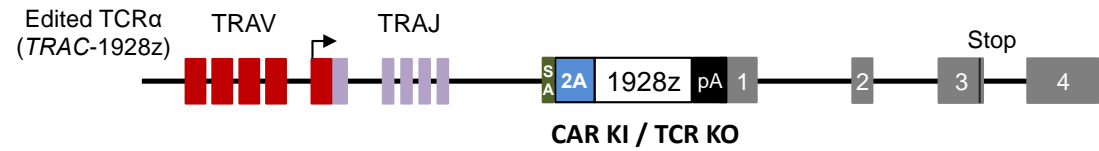
CAR expression influences DP T cell development

early

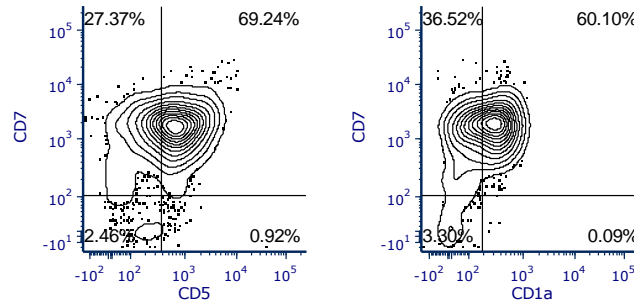
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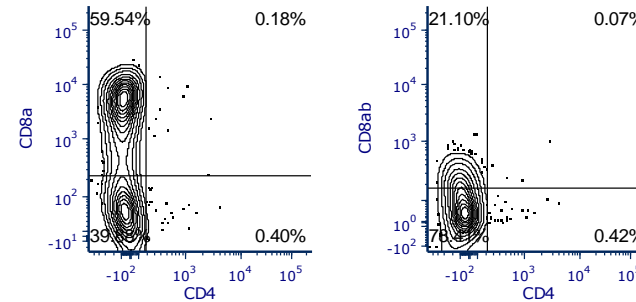
Solutions: *TRAC*-1928z TiPSC-derived T cells



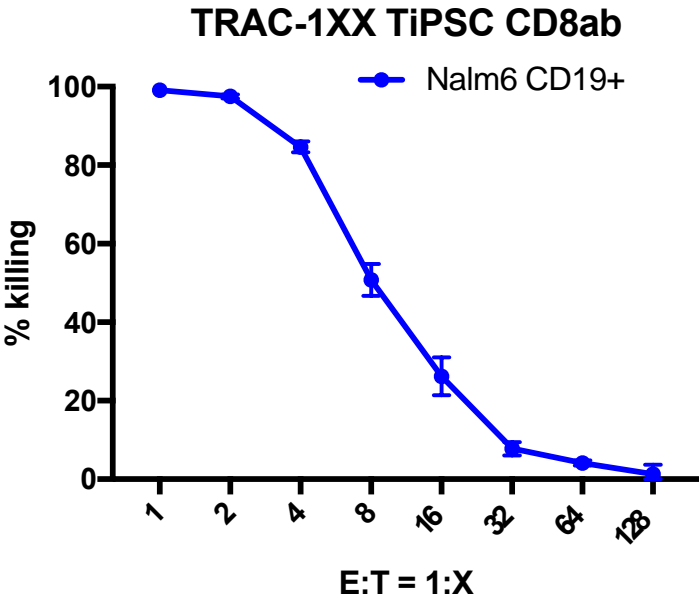
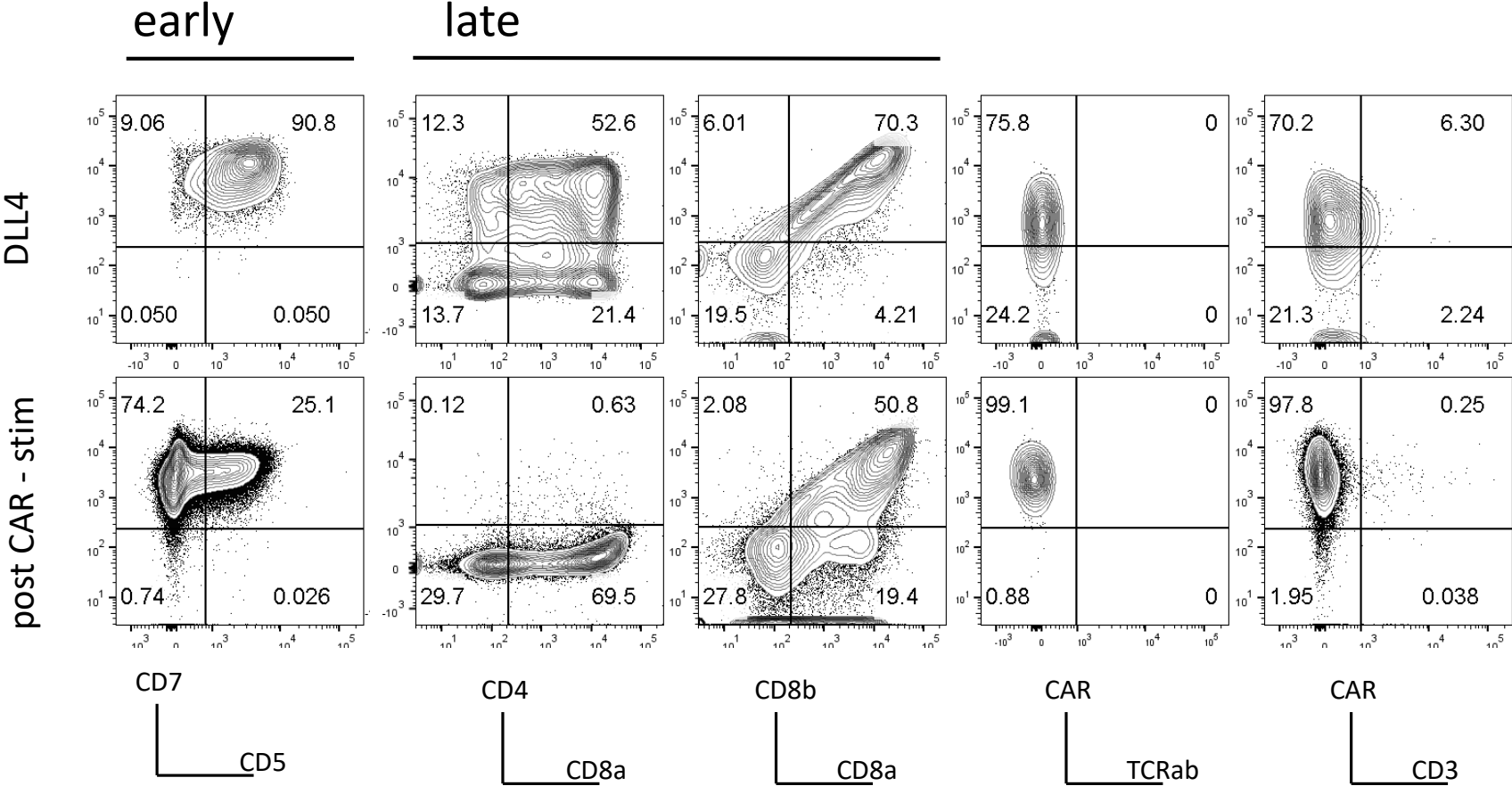
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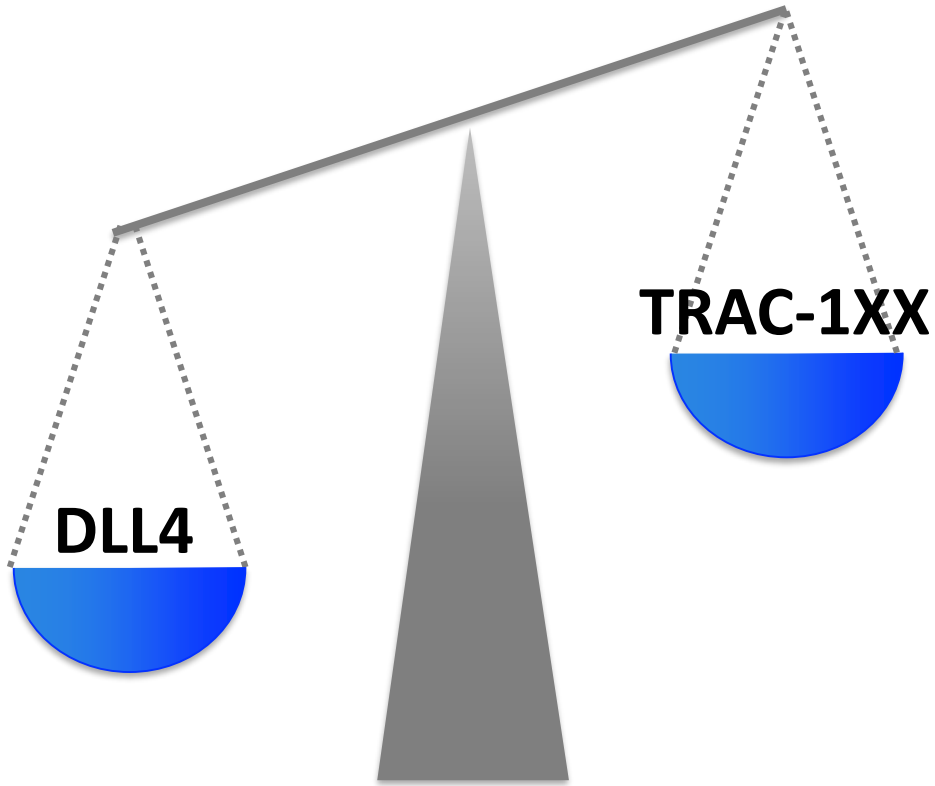
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Solutions: ITAM-control enhances $\alpha\beta$ lineage fate



Summary T-iPSC

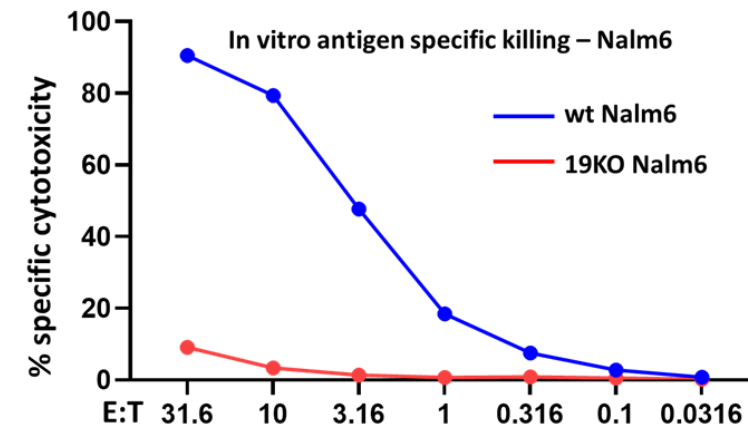
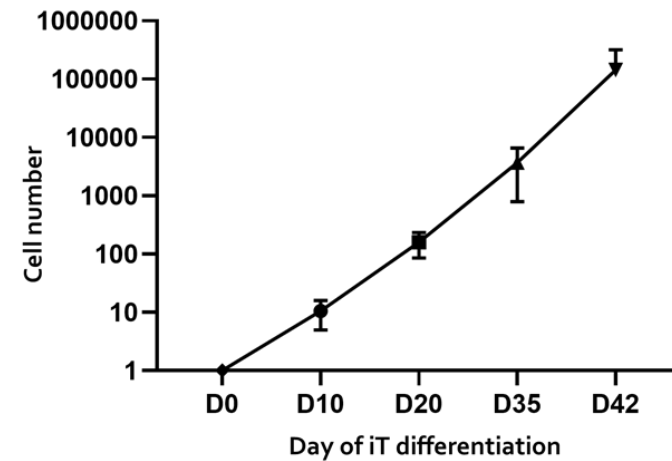
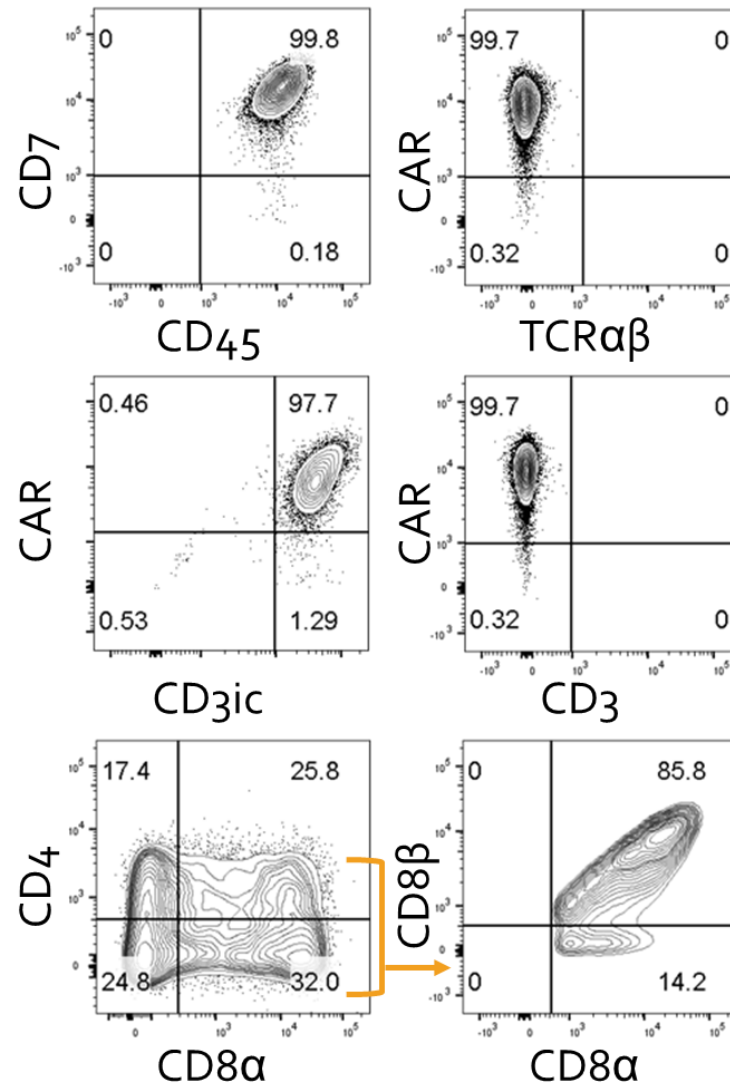
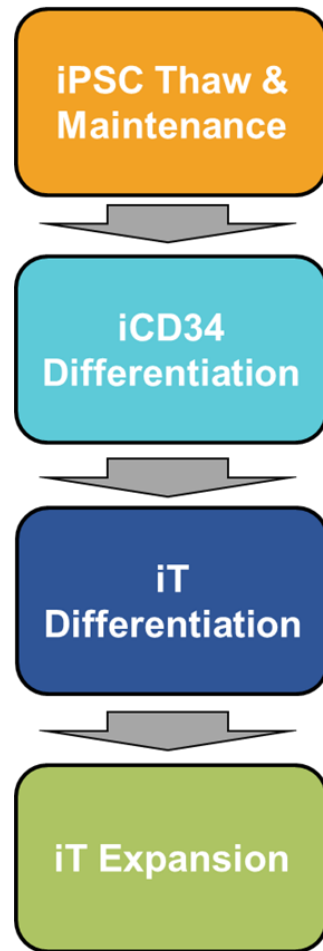


- Early TCR $\alpha\beta$ expression skews T cell differentiation
- hDLL4 can induce CD4/CD8 DP T cells
- CD8 SP T cells arise from DP population
- CAR expression interferes with DP T cell development
- Delayed CAR expression can facilitate DP > SP conversion
- *TRAC*-encoded CAR expression with controlled ITAM strength enhances $\alpha\beta$ T cell development



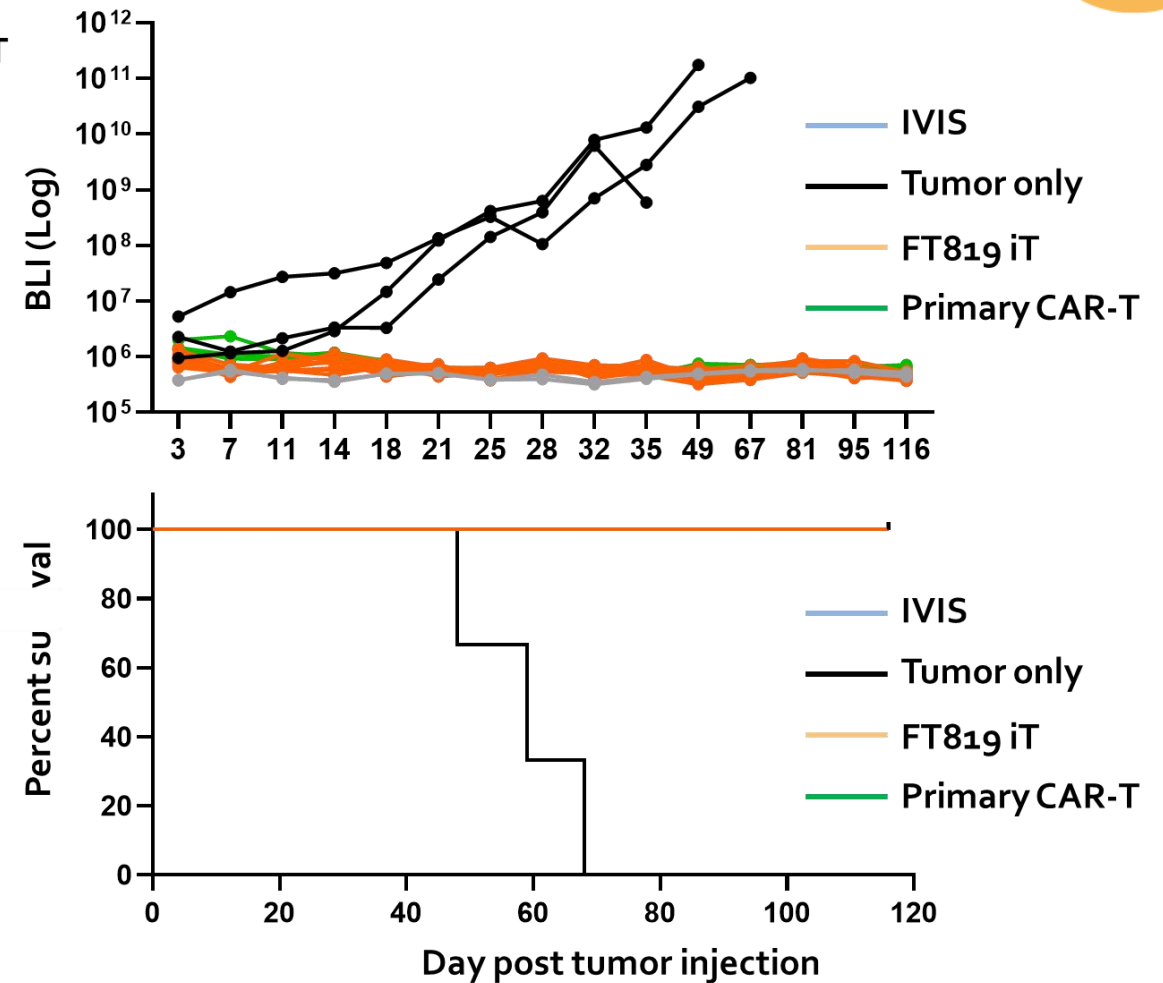
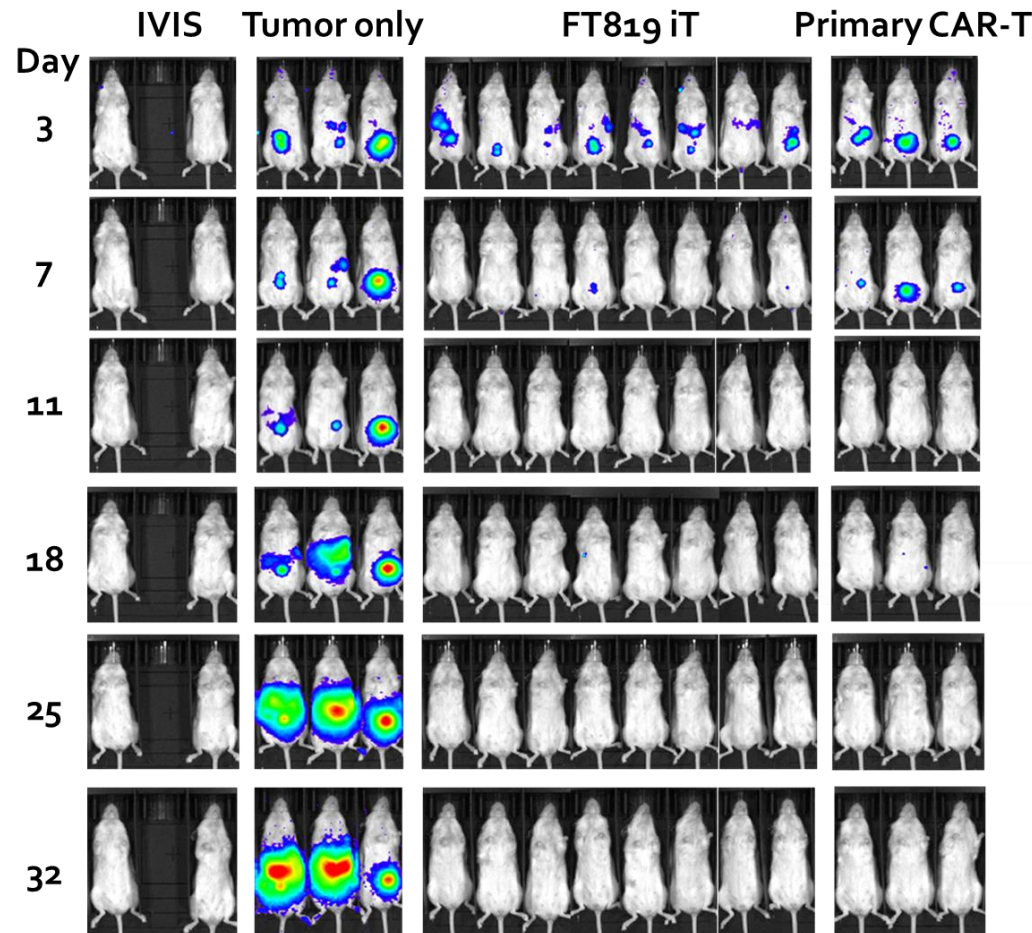
FT819 Phenotype, Potency, Specificity and Proliferation Capacity

Derived from a TRAC-targeted CD19 CAR, TCR-null Master iPSC line



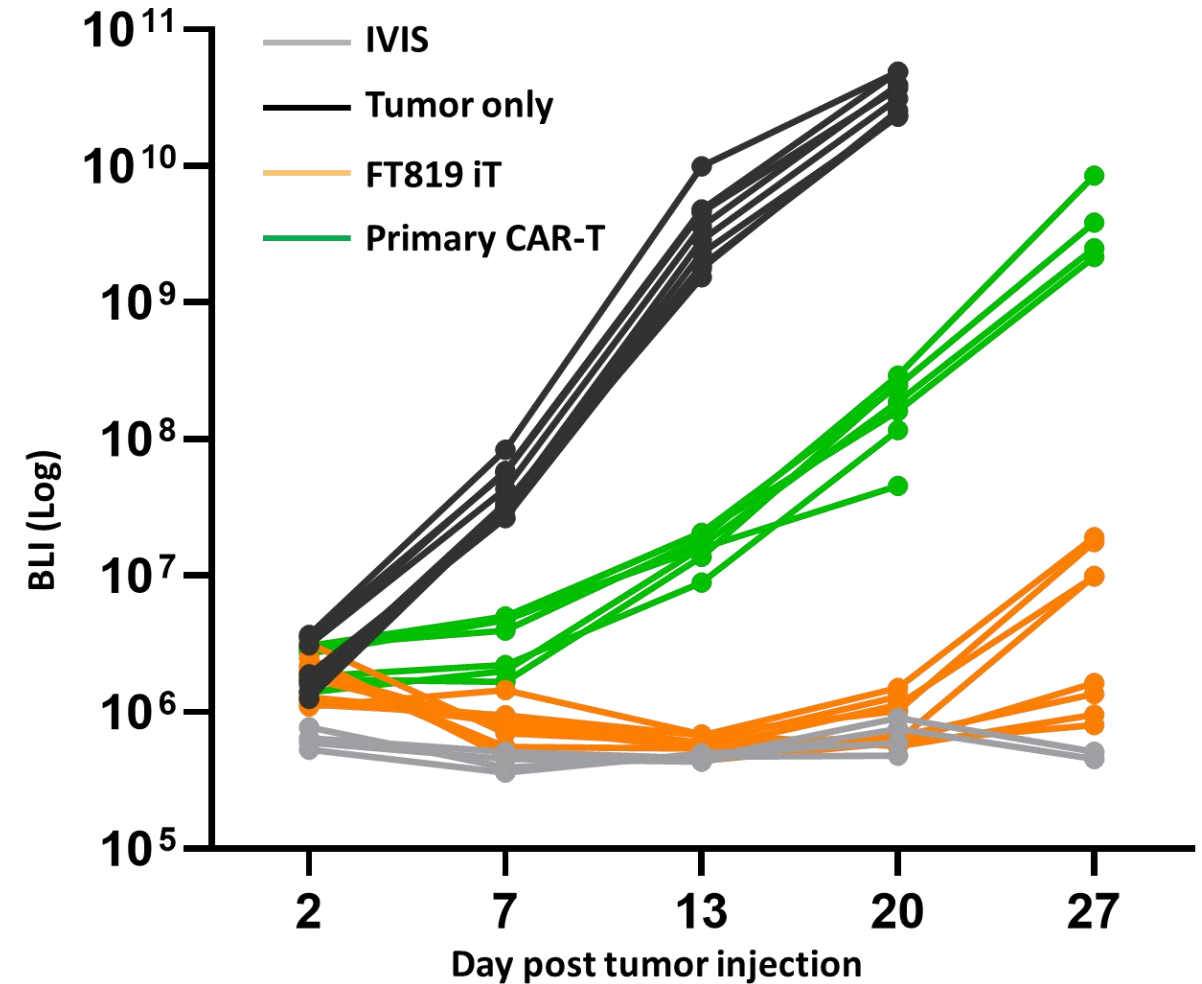
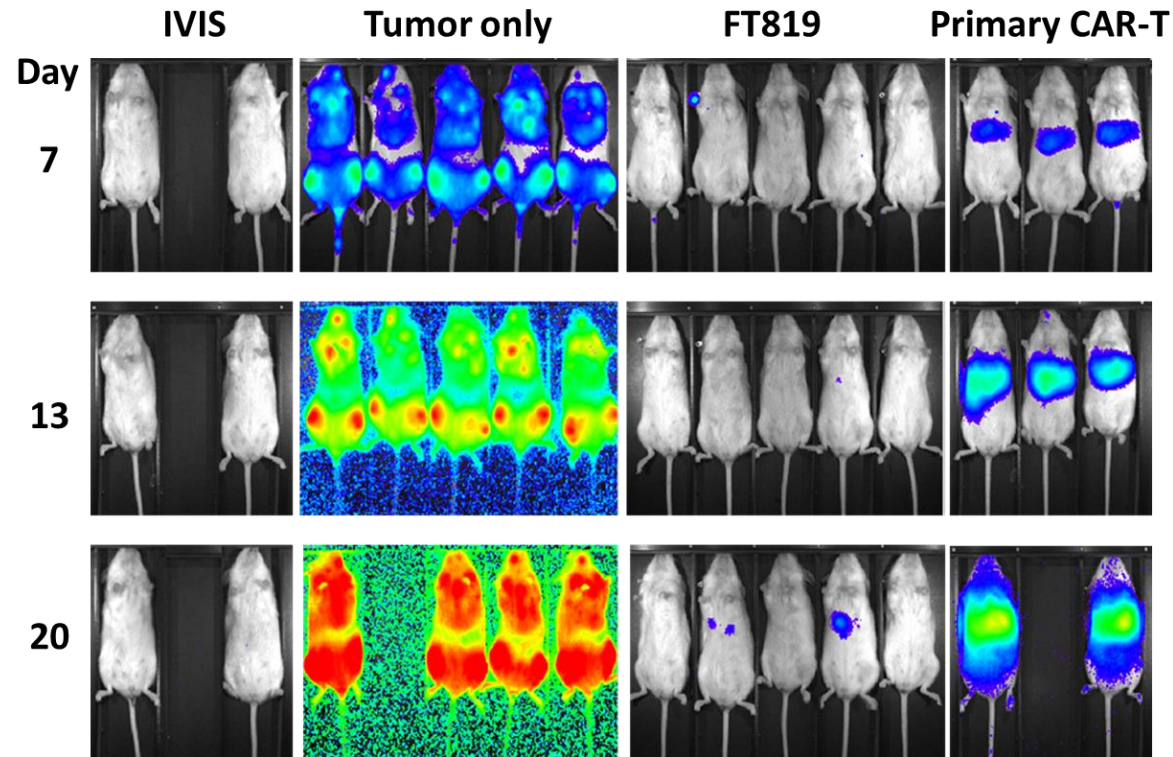
FT819 Controls Tumor Growth and Promotes Long-term Survival

Intraperitoneal Xenograft Model of Lymphoblastic Leukemia

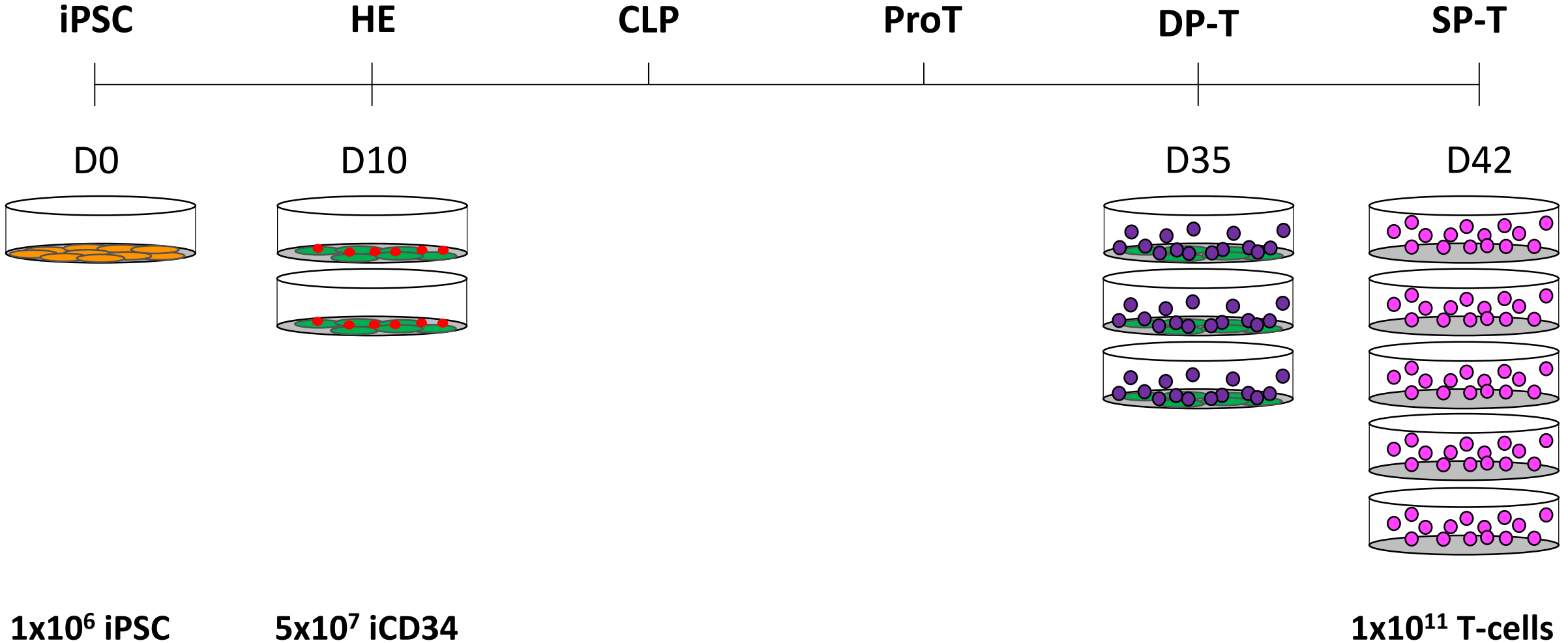


FT819 Displays Enhanced Control of Tumor Growth

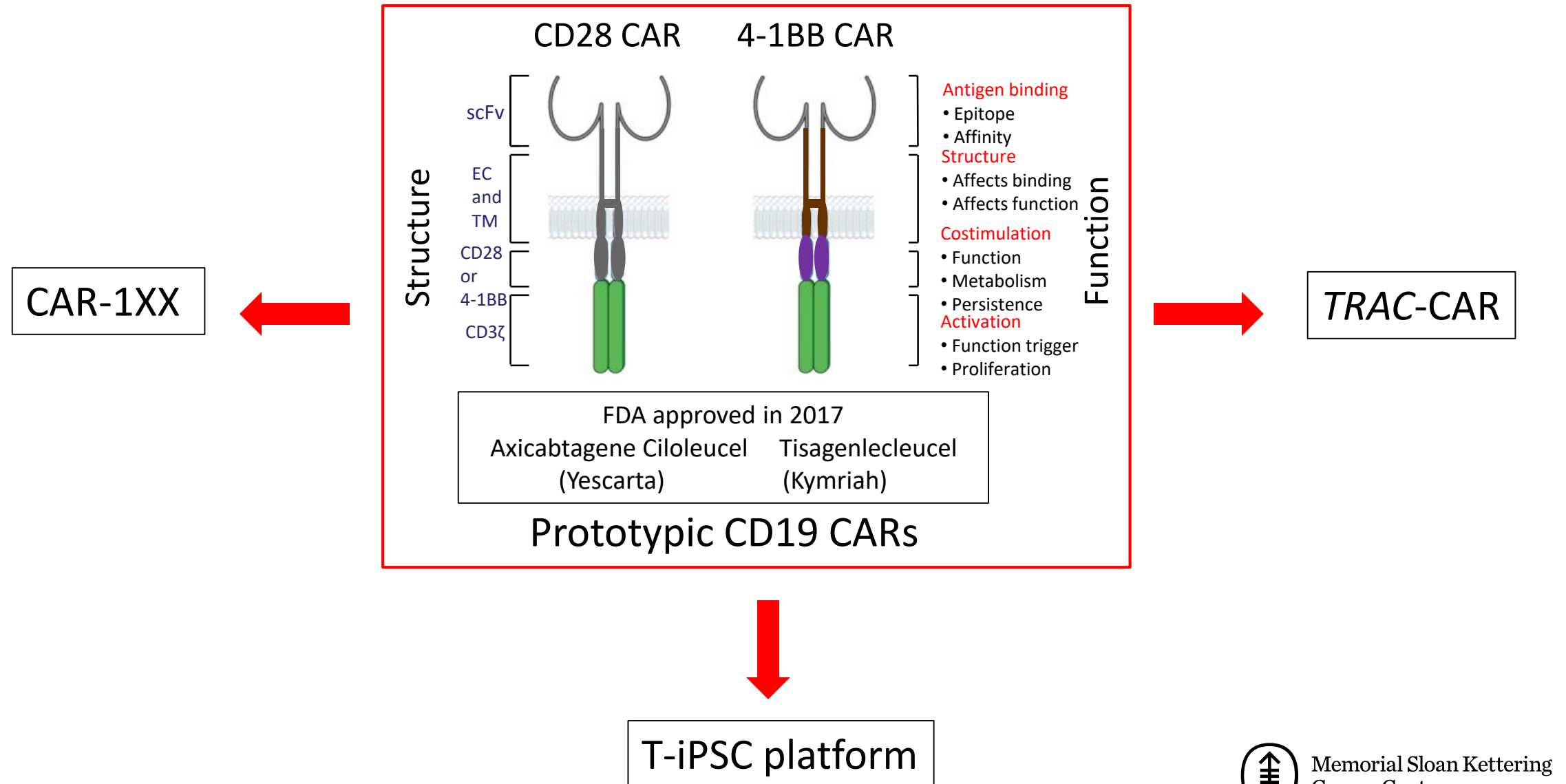
Disseminated Xenograft Model of Lymphoblastic Leukemia



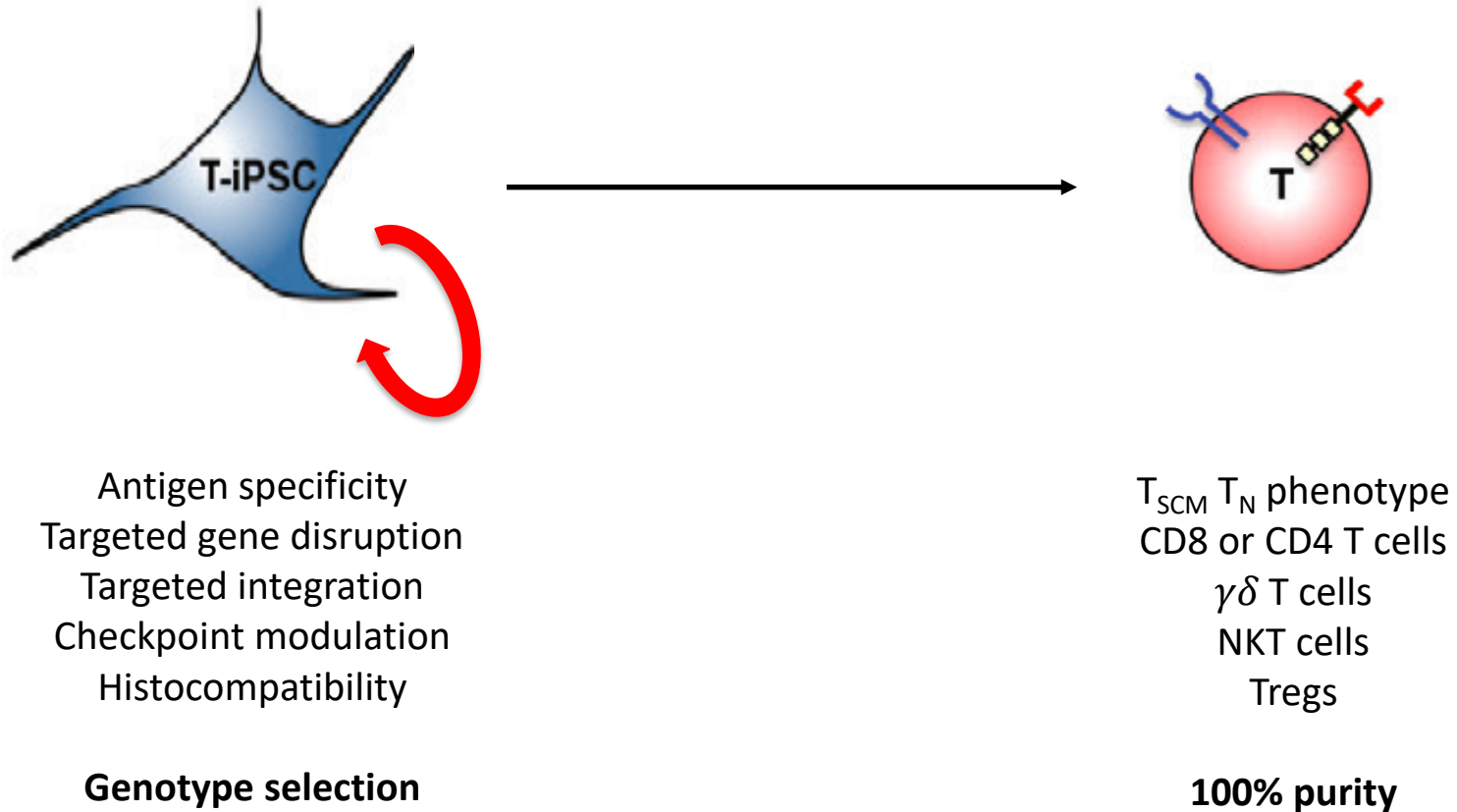
In vitro CAR T cell development



From current CD19 CAR therapy to *FT819*



TiPSC for T cell immunotherapy

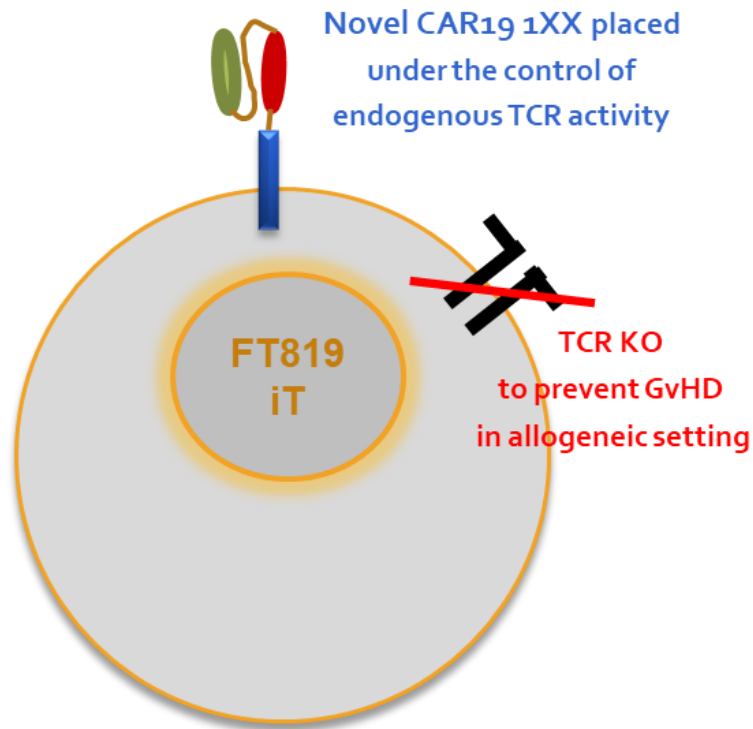


FT819 Universal, Off-the-Shelf CAR19 T-Cell Product

Novel CAR19 Targeted to the TRAC Locus for Improved Safety and Efficacy



First-in-Class Off-the-Shelf Adoptive CAR19 T cell Product Uniformly Consisting of Novel Engineering Elements



→ **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

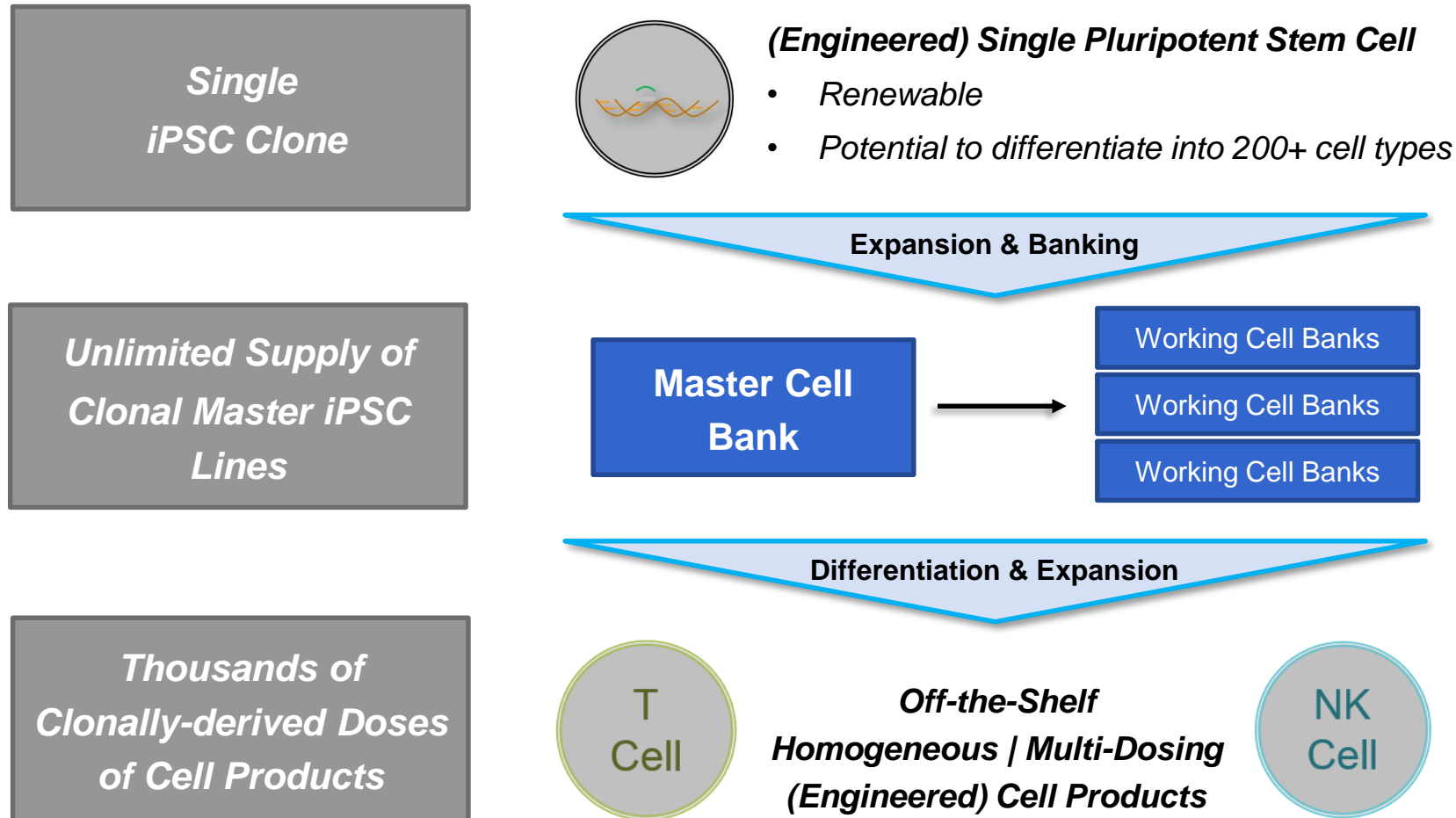
→ **TCR null:** Bi-allelic disruption of TRAC at the clonal level for complete removal of TCR expression and the elimination for the possibility of GvHD in allogeneic setting



iPSC Product Innovation

iPSC Product Platform

Clonal Master iPSC Lines for Off-the-Shelf Cell Products



“to reach more patients in need”

