

Corporate Presentation

March 2022



Cautionary Note Regarding Forward-Looking Statements

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For a detailed discussion of the risk factors that could affect the Company’s actual results, please refer to the risk factors identified in the Company’s SEC reports, including Annual Report on Form 10-K dated March 16, 2022. Except as required by law, the Company undertakes no obligation to update publicly any forward-looking statements for any reason.

Sana Biotechnology

Engineered Cells as Medicines

Ability to modify the genome and use engineered cells as medicines has the potential to be one of the most important advances in healthcare over the next several decades:

- Nearly every disease is caused by damage to or dysfunction of a cell
- Sana's ambition is to repair or replace any cell in the body

Platform progress, broad capabilities, and strong balance sheet enable us to execute on a broad vision:

- Goal: allo T and *in vivo* CAR T INDs this year with 2-3 INDs per year going forward
- Building expertise in key areas: delivery of genetic material, gene modification, immunology, biology, and manufacturing
- \$746.9M as of year-end 2021

Sana goal: Repair cells in the body when possible or replace them when needed

in vivo Cell Engineering

Repair and control the genes of any cell in the body

Deliver any payload...

(DNA, RNA, protein, organelle, integrating vs non-integrating)

To any cell...

(unlimited volume of distribution)

In a specific...

(e.g., just T cell)

And repeatable way

(limit immunogenicity)

ex vivo Cell Engineering

Replace any cell in the body

Manufacture any cell at scale...

That engrafts...

(the right cell in the right environment)

Functions...

(understand exact phenotype desired)

And persists

(overcome immune rejection and cellular signaling, such as apoptotic signaling)

Sana's platforms, technology, and programs

| PLATFORM | TECHNOLOGY | PROGRAMS (CELL TYPES) | THERAPEUTIC AREA | PRE-CLINICAL PRODUCT CANDIDATE | POTENTIAL INDICATIONS |
|--------------------------|---|--------------------------|---------------------------------|-----------------------------------|------------------------------|
| ex vivo cell engineering | Hypoimmune donor-derived | T cells | Oncology | SC291 [CD19] | NHL/ALL/CLL |
| | | | | SC276 [CD22 (+CD19)] | NHL/ALL/CLL |
| | | | | SC255 [BCMA] | Multiple myeloma |
| | Hypoimmune stem cell-derived | Beta cells | Diabetes | SC451 | Type 1 diabetes |
| | Stem cell-derived (to migrate to hypoimmune) | Glial progenitor cells | Central nervous system (CNS) | SC379 | Huntington's disease |
| | | | | | Pelizaeus-Merzbacher disease |
| in vivo cell engineering | Fusogen | T cells | Oncology | SG295 [CD8/CD19] | NHL/ALL/CLL |
| | | | | SG239 [CD8/BCMA] | Multiple myeloma |
| | | | | SG242 [CD4/CD19] | NHL/ALL/CLL |
| | | | | SG221 [CD4/BCMA] | Multiple myeloma |
| | | | | SG233 [CD8/CD22 (+CD19)] | NHL/ALL/CLL |
| | | Hepatocytes | Liver-related genetic disorders | SG328 | OTC ¹ |
| | | Hematopoietic stem cells | Hemoglobinopathies | SG418 | Sickle cell disease |
| | | | | | Beta-thalassemia |

¹Ornithine transcarbamylase deficiency

Hypoimmune technology: Protecting cells from immune rejection

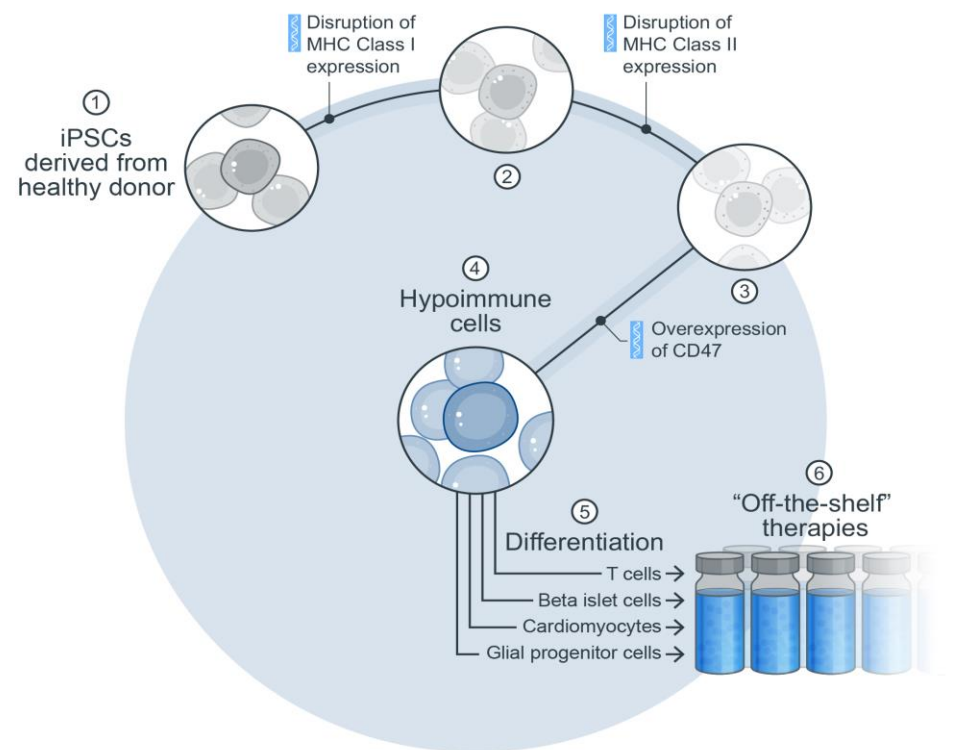
Sana approach: Leverage insights from nature to create hypoimmune cells (HIP)

“Allogeneic” fetus:

- Half of fetal proteins are from the father, not the mother.
- However, the fetus is not rejected by the mother.



How can we protect our engineered cells from getting attacked from the recipient's immune system?

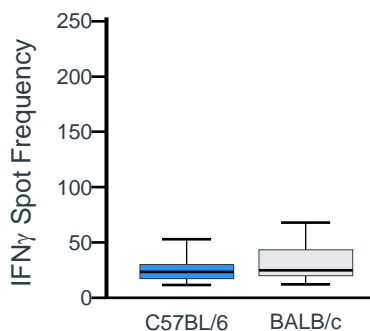


Hypoimmune cells evade rejection from the adaptive and innate immune system in mice

Evade the adaptive immune system

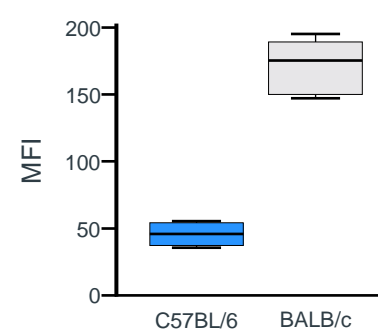
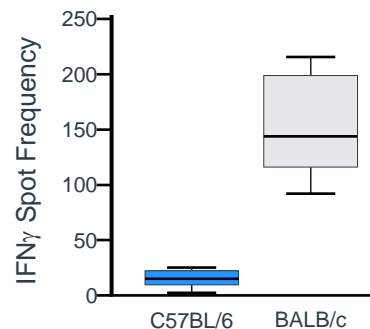
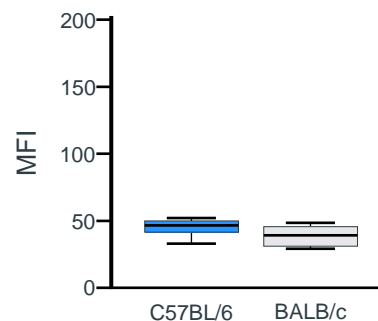
T cell Activation (ELISPOT)

No systemic T cell activation with HIP cell transplantation



IgM Binding (FACS)

No binding of donor-specific antibodies against HIP cells



Representative of results across 5 mice for the unmodified arm and across 6 mice for the hypoimmune arm.

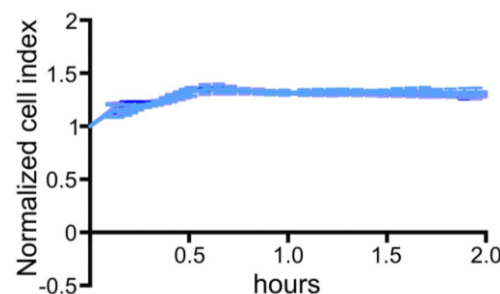
Evade the innate immune system

NK Cell Killing

No NK cell killing of HIP cells

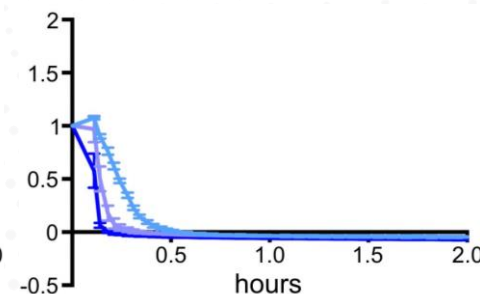
Wildtype Unmodified Cells

No killing of unmodified cells by NK cells



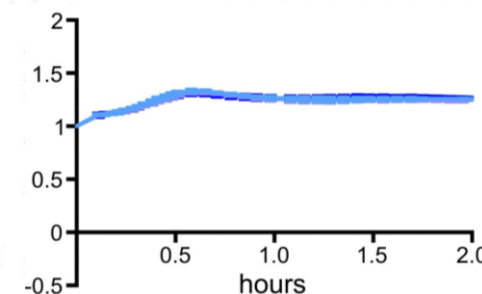
MHC Class I/II Disruption

Killing of partially edited iPSC; HLA I/II knockout by NK cells



MHC Class I/II Disruption & CD47 Overexpression

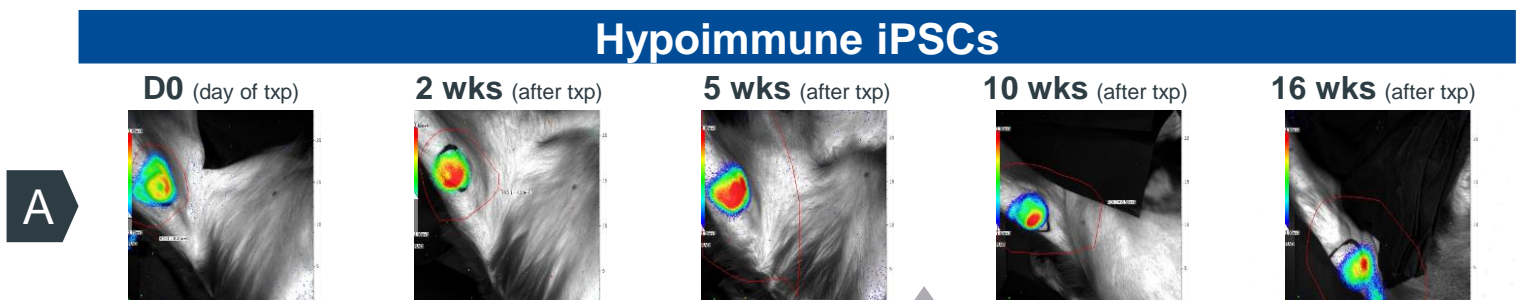
No killing of HIP cells by NK cells



— C57BL/6 mNK cells:miECs 0.5:1
— C57BL/6 mNK cells:miECs 0.8:1
— C57BL/6 mNK cells:miECs 1:1

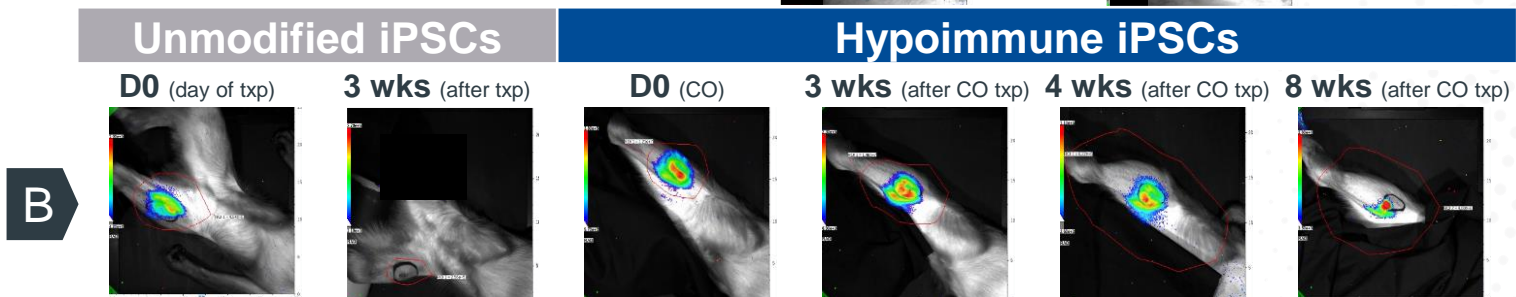
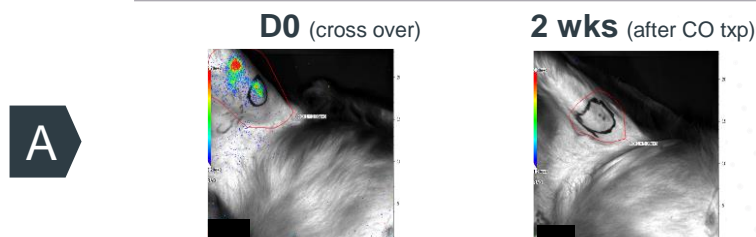
Deuse T, ..., Schrepfer S. *Nat Biotechnology*. 2019; 37:252-258

Hypoimmune cells survive *in vivo* in NHP while unmodified iPSCs get rejected



Representative of results across 4 NHPs per arm.

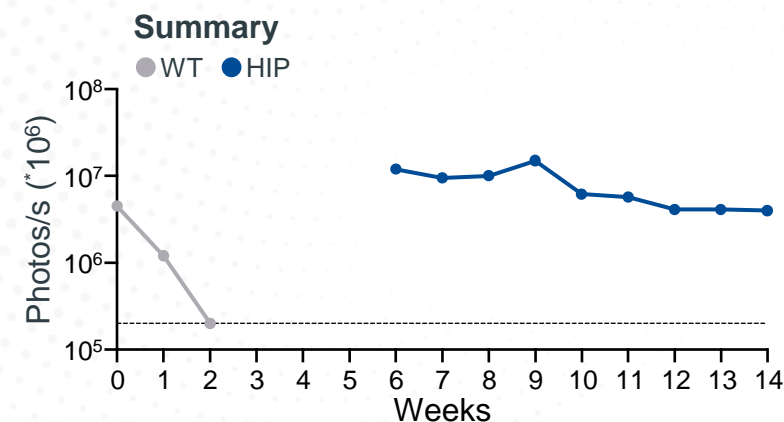
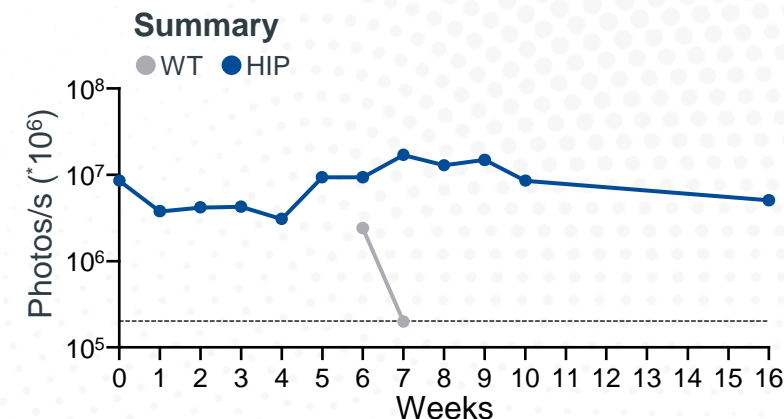
Unmodified iPSCs at 6 wks



Representative of results across 4 NHPs per arm.

- NHP unmodified iPSCs (wt) & NHP hypoimmune iPSCs (HIP) were transplanted into 8 allogeneic recipients

CO, cross over; Txp, transplant



Hypoimmune cells evade rejection from the adaptive and innate immune system in NHPs

Transplantation of NHP iPSCs into allogeneic NHPs: immune evasion is achieved even after prior sensitization

T cell Activation (ELISPOT)

No systemic T cell activation by hypoimmune cells

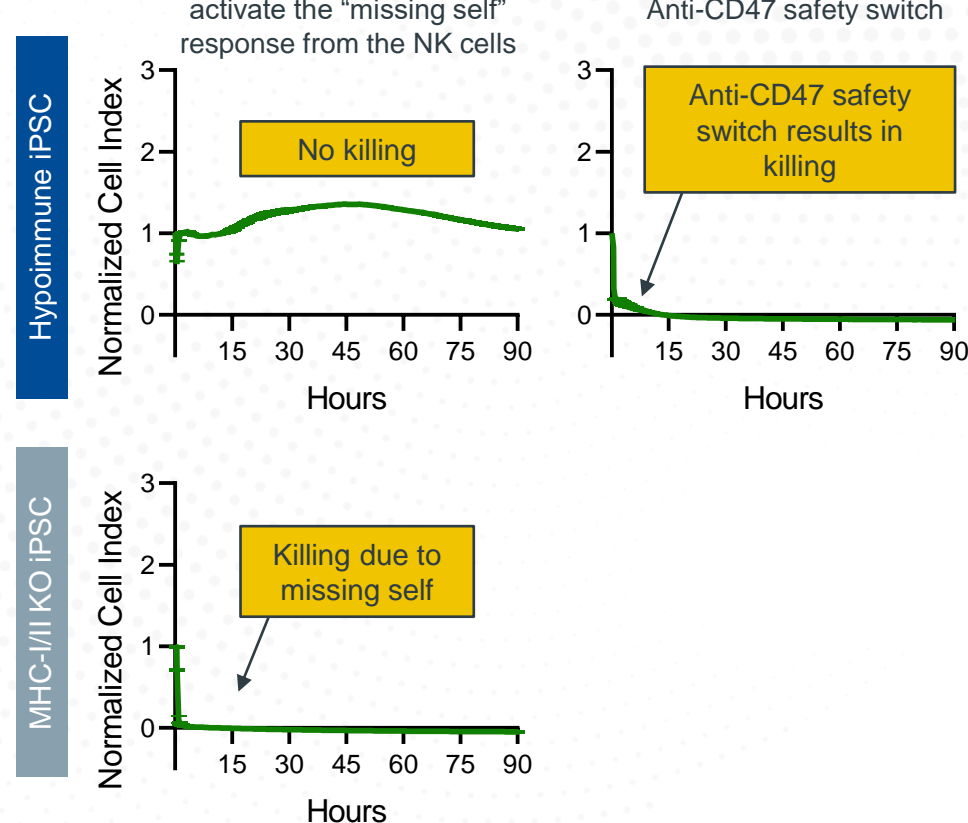
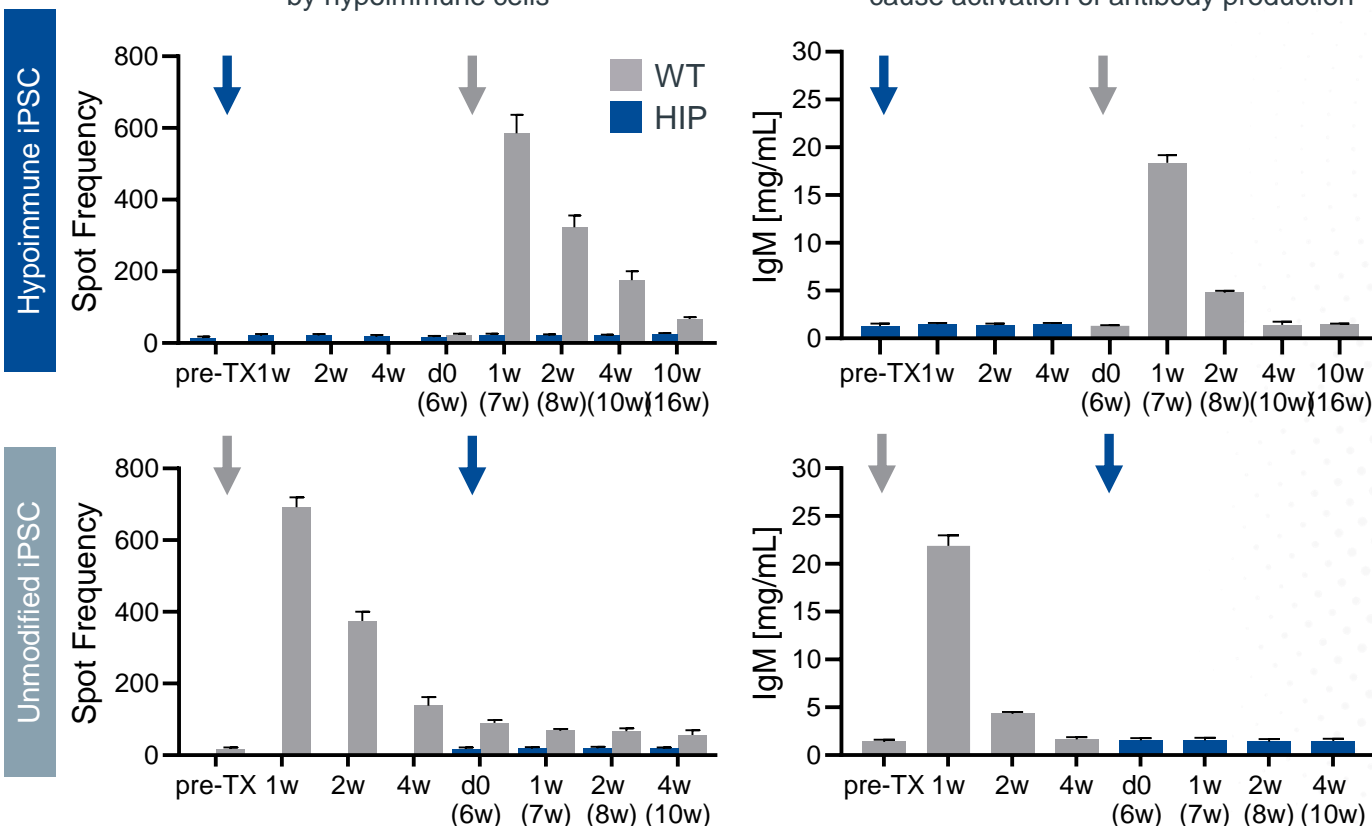
IgM Production (ELISA)

Implantation of hypoimmune cells does not cause activation of antibody production

Killing by NK Cells

Hypoimmune cells do not activate the "missing self" response from the NK cells

Anti-CD47 safety switch



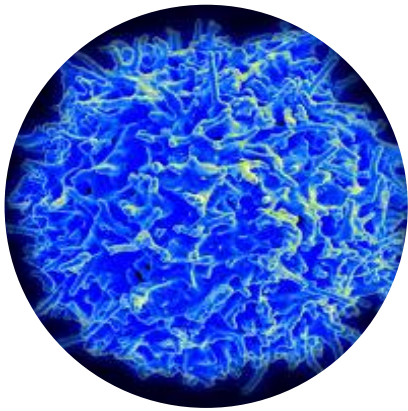
Representative of results across 4 NHPs per arm.

Sana is pursuing a broad *ex vivo* cell engineering strategy

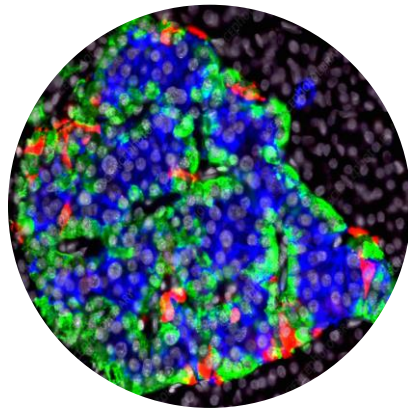
Transforming *ex vivo* cell engineering through development of hypimmune cell platform

Differentiate pluripotent stem cells with hypimmune edits

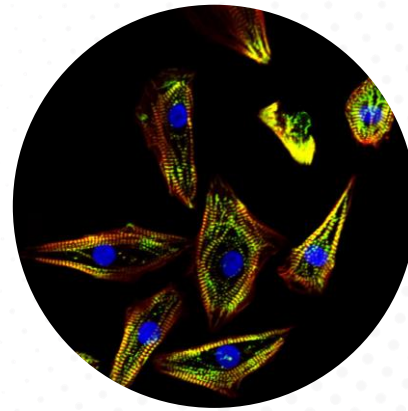
Programs that benefit from, but do not require hypimmune



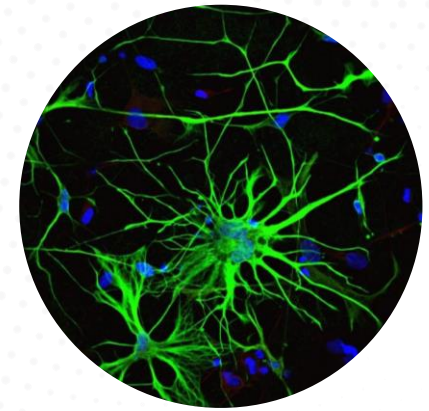
T cells



Pancreatic islets



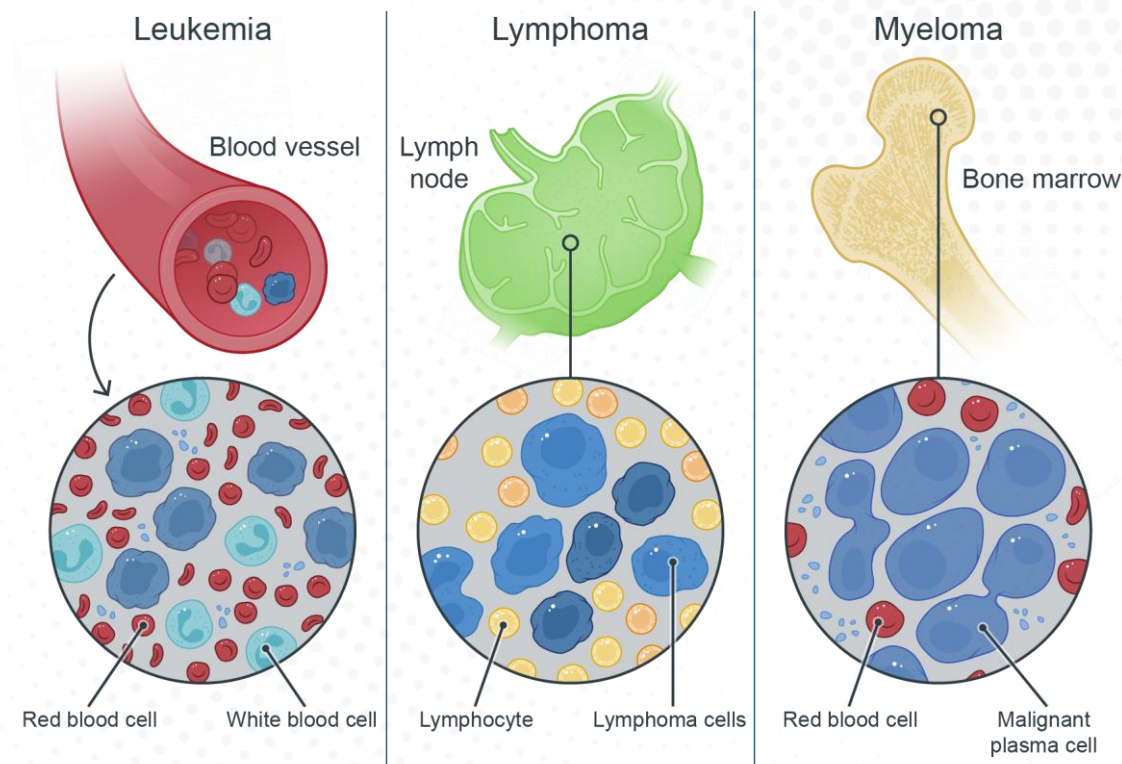
Cardiomyocytes



Glial progenitor cells

High unmet need remains for blood cancers

- Significant unmet need in B cell malignancies and multiple myeloma in the US and EU alone:
 - ~250,000 new cases annually¹
 - Est. 100,000 deaths annually¹
- <10,000 patients have been treated with CAR T therapy to date²
- Of those treated, many do not respond and many relapse
- Manufacturing and access challenges remain for many patients



¹World Health Organization, GLOBOCAN 2020

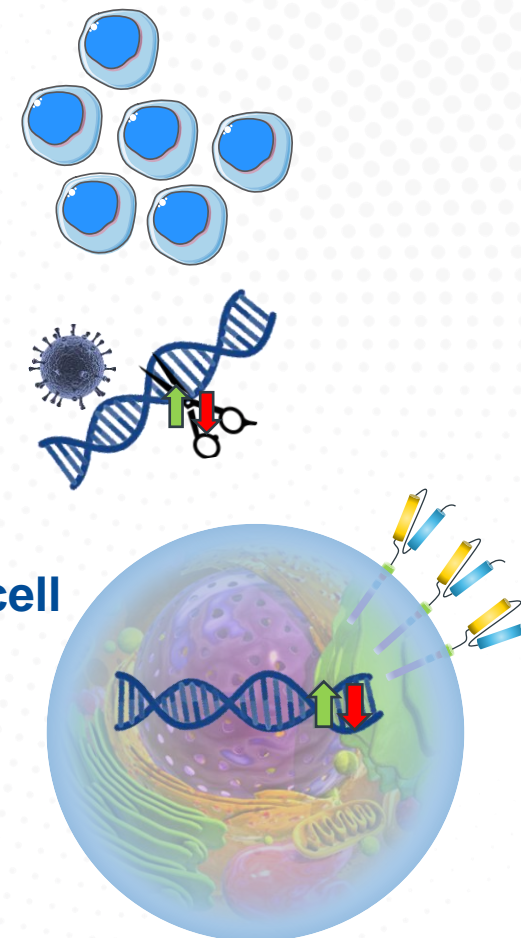
²Financial Reports, through Q3 2021; Evaluate Pharma, through Q3 2021

Sana's hypoimmune allo T is potentially best-in-class

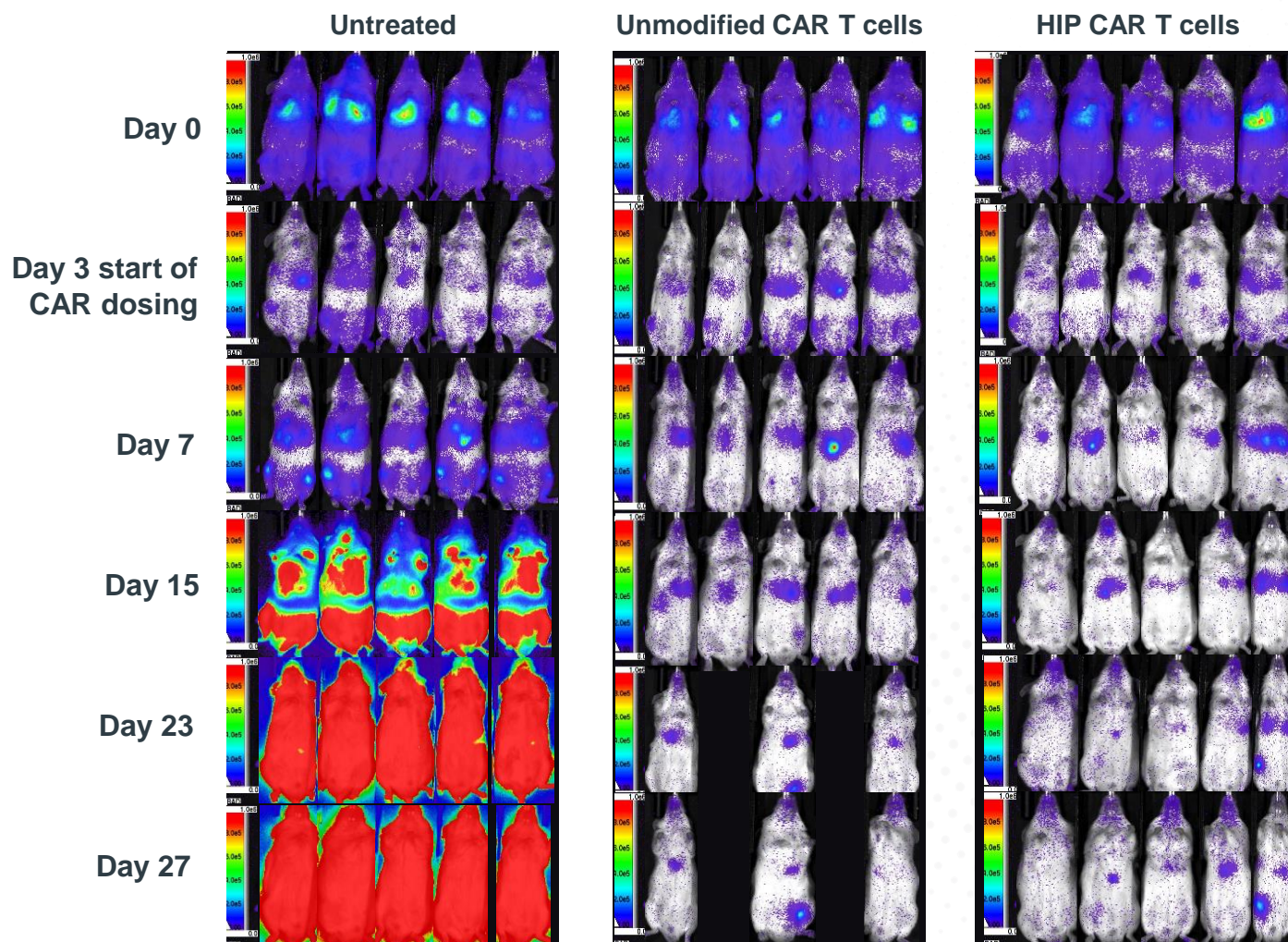
| Immune Challenges | Current Allo T | Sana Hypo Allo T |
|------------------------------|----------------|------------------|
| GvHD | ✓ | ✓ |
| HvGD: Adaptive immune system | ? | ✓ |
| HvGD: Innate immune system | ✗ | ✓ |

GvHD, graft versus host disease; HvGD, host versus graft disease.

- 1 Donor or iPSC T cells
- 2 Cell engineering
- 3 CD19 targeted HIP allogeneic T cell



CD19 HIP CAR T cells clear tumor *in vivo*



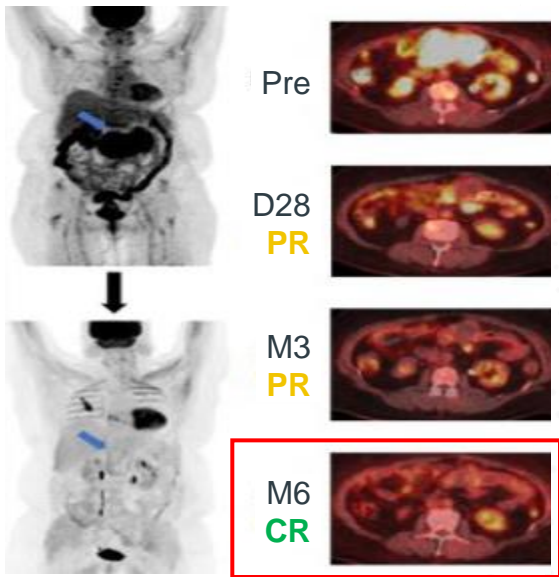
Clinical study shows CD22 CAR T efficacy in treating relapsed lymphoma after prior CD19 CAR T therapy

| Patient 1 | |
|-----------------------------|----------|
| Prior lines of therapy | 5 |
| Prior CAR T therapy | Yes |
| Product previously received | Yescarta |
| Antigen targeted | CD19 |

Blood 2021 Apr 29;137(17):2321-2325. doi: 10.1182/blood.2020009432.

LBCL

| | Total (N=24) |
|----------------------------------|----------------|
| Median follow up, months [range] | 8.6 [1.6-21.3] |
| Overall Response Rate*, n (%) | 19 (79%) |
| CR Rate | 14 (58%) |



Total (N=24)

Miklos et al, ASH 2021
Total is a combination of DL1 and DL2

| Minimal ICANS / CRS observed across dose levels | | | |
|---|------------------|-----------------|--------------|
| Parameter | DLBCL DL1 (N=15) | DLBCL DL2 (N=9) | Total (N=24) |
| Cytokine release syndrome*, n (%) | | | |
| None | 1 (7%) | 0 (0%) | 1 (4%) |
| Grade 1 | 6 (40%) | 1 (11%) | 7 (29%) |
| Grade 2 | 8 (53%) | 7 (78%) | 13 (54%) |
| Grade 3 | 0 (0%) | 1 (11%) | 1 (4%) |
| Neurologic events / ICANS*, n (%) | | | |
| Grade 1 | 1 (7%) | 1 (11%) | 2 (8%) |
| Grade 2 | 1 (7%) | 1 (11%) | 2 (8%) |

Miklos et al, ASH 2021

Best-in-class, broadly accessible allogeneic CAR T cells

- Expect to file our first allo T IND targeting CD19 as early as this year
- CD19/CD22 dual targeting offers potential of higher and more durable complete response rates
- Fully-human, clinically validated CD22 CAR with potential to address CD19 CAR T failures
- Fully-human, clinically validated BCMA CAR to potentially treat multiple myeloma
- Targets beyond CD19, CD22, and BCMA

Type 1 diabetes represents a large unmet need with a loss of ~15 years of life¹

Large unmet need remains

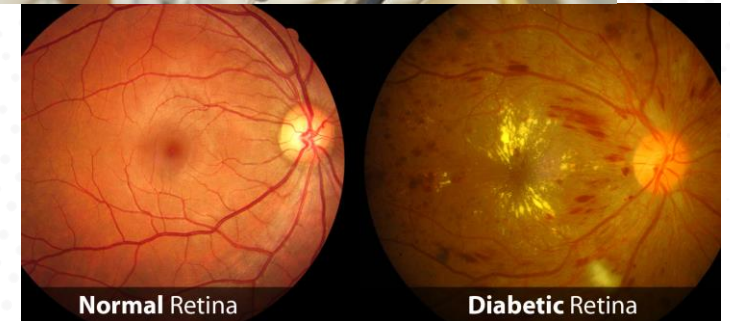
- Disease where destruction of insulin-producing beta cells in the pancreas results in inability to control blood glucose
- 1.6M patients with type 1 diabetes in the US and 2.4M in Europe²; 51k new patients/year combined³
- Long-term complications: end-organ damage, including heart attack, stroke, retinopathy, and nephropathy

→ **Our goal is to produce hypoimmune beta cells that evade the immune system and normalize glucose**

¹Rawshani *et al*, Lancet 2018

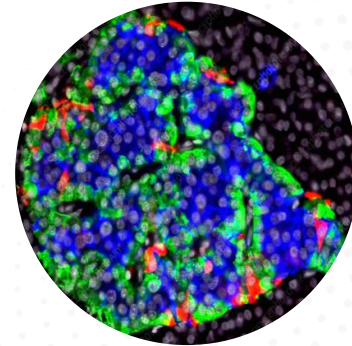
²Centers for Disease Control and Prevention, Diabetes Report, 2017-2018

³National Institutes of Health, Health Promot Perspect 2020



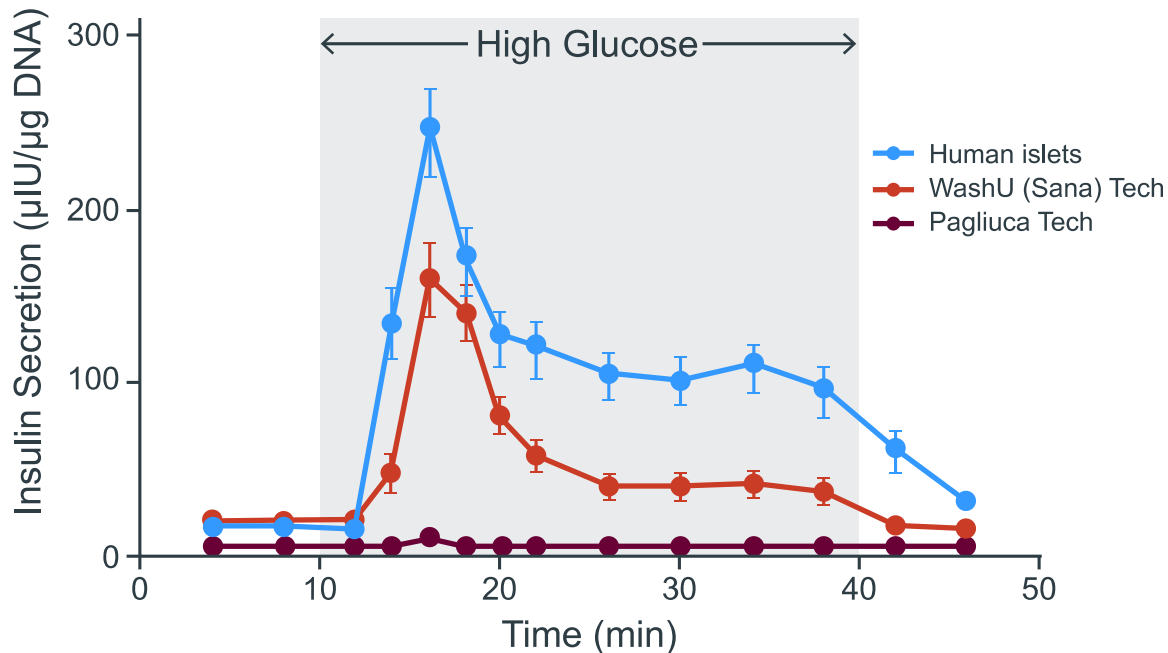
Progress toward turning beta cells into medicines

1. **Make** functional beta cells from iPSCs cells ✓
2. **Hide** beta cells from allogeneic rejection ✓
3. **Hide** beta cells from autoimmune reaction ✓
4. **Create** GMP supply chain

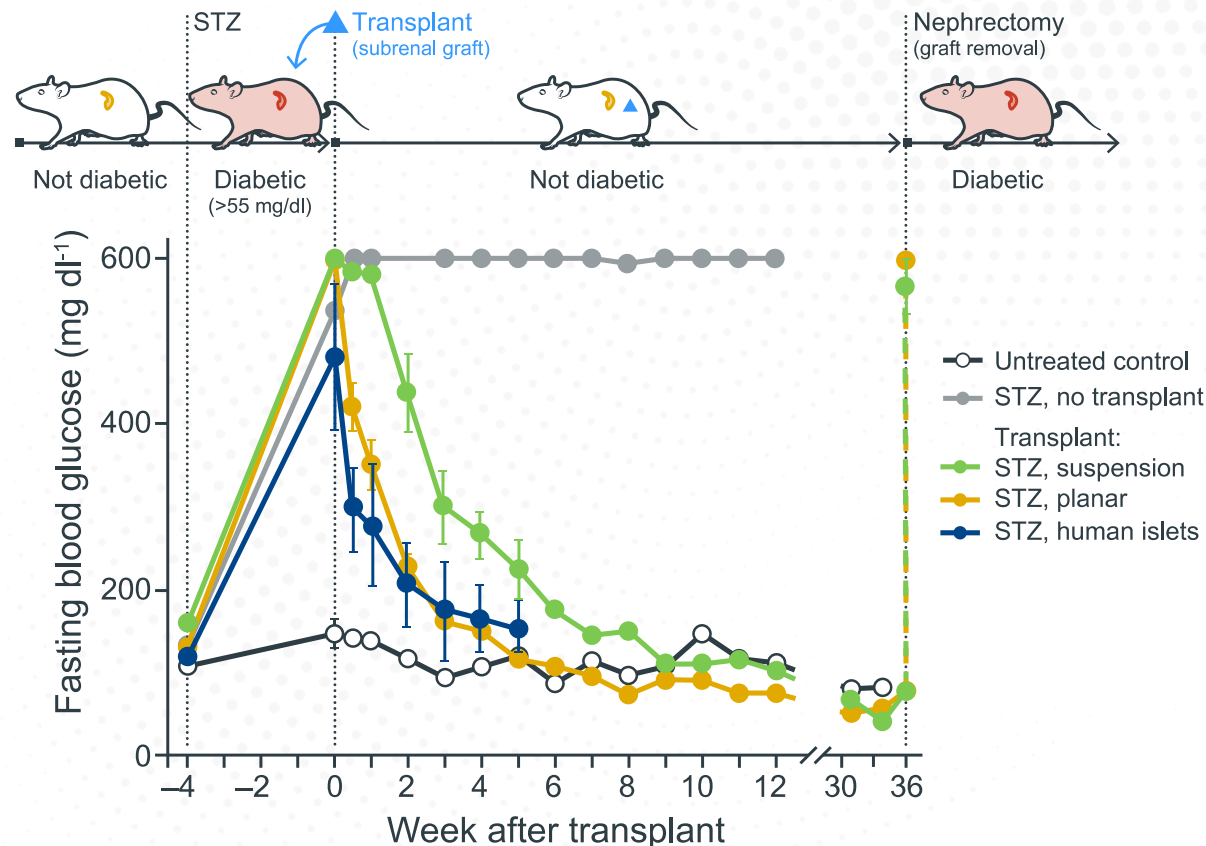


Stem cell-derived pancreatic islet cells lead to robust function

Superior insulin secretion and faster kinetics *in vitro*

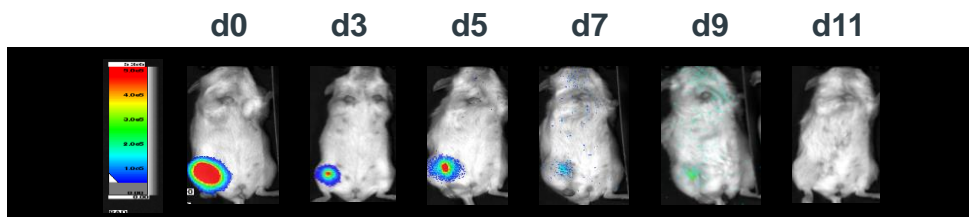


Robust rescue of type 1 diabetes mouse model

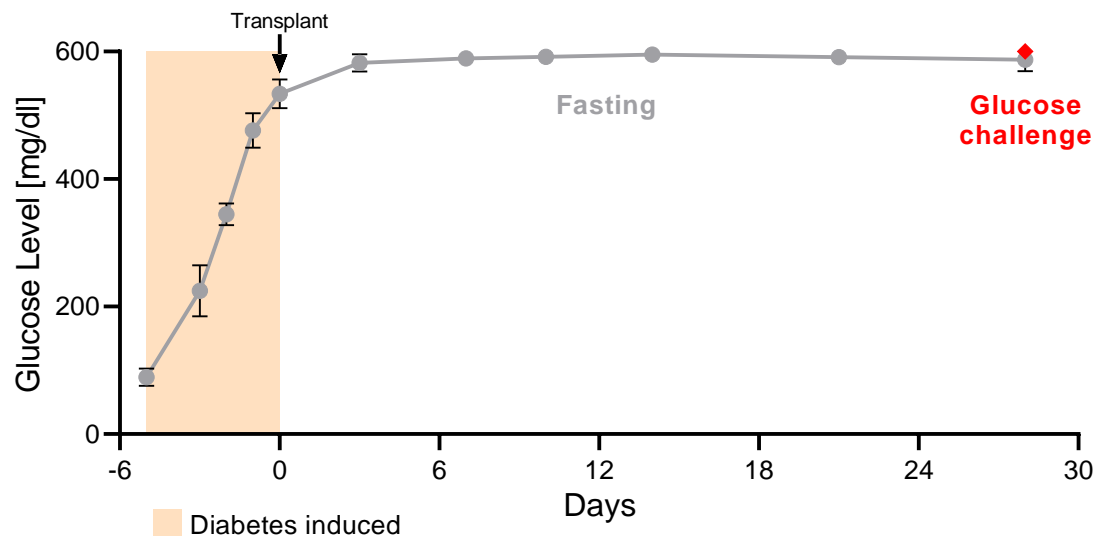


Hypoimmune pancreatic islet cells evade immune detection and regulate glucose levels

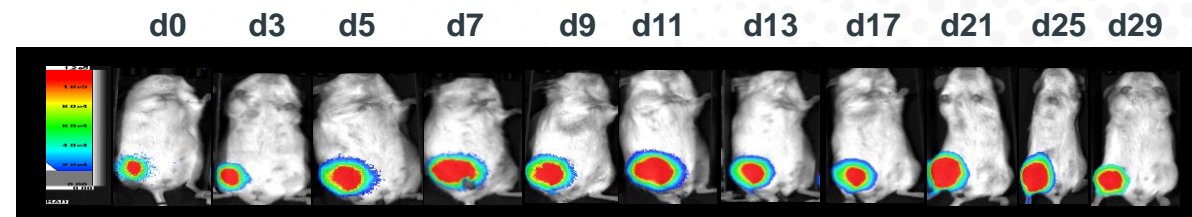
Allogeneic human unmodified islet cells



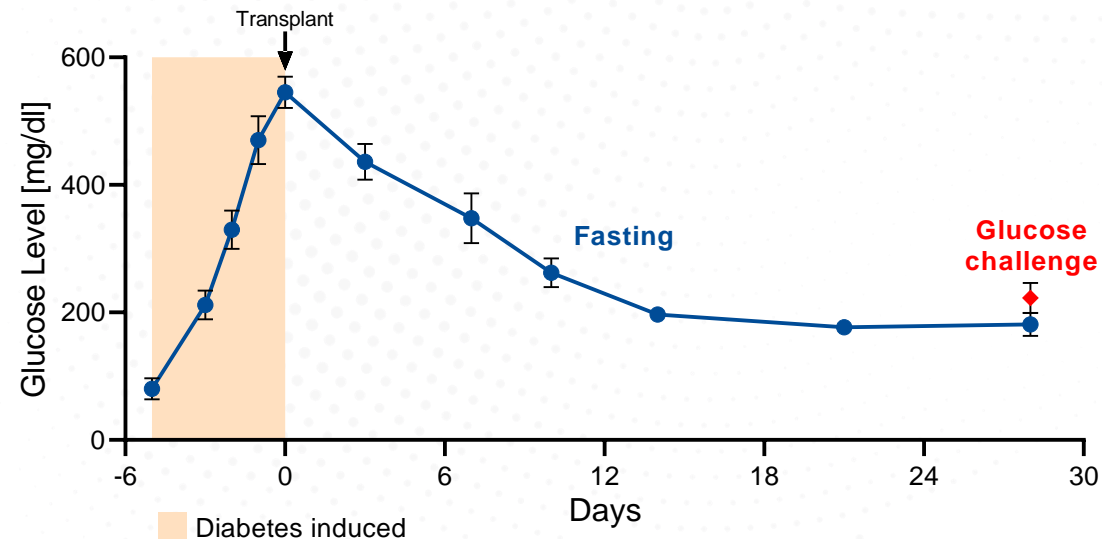
Glucose levels stay elevated



Allogeneic human hypoimmune islet cells

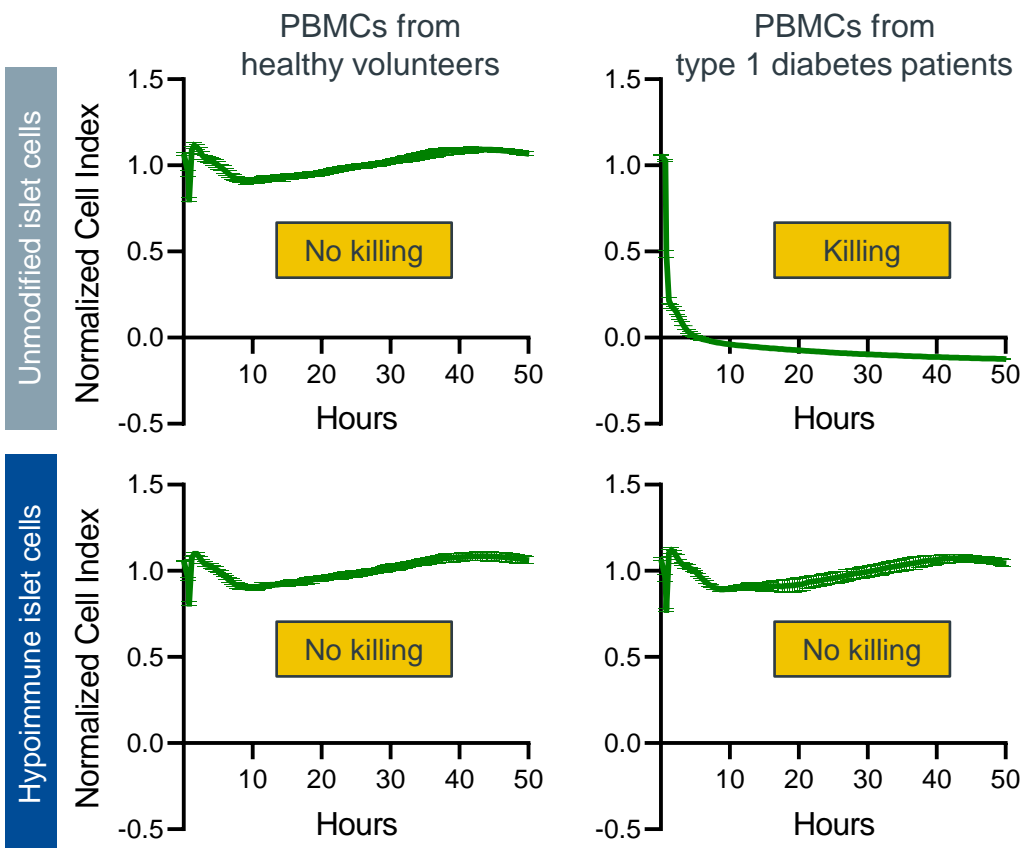


Glucose levels normalized



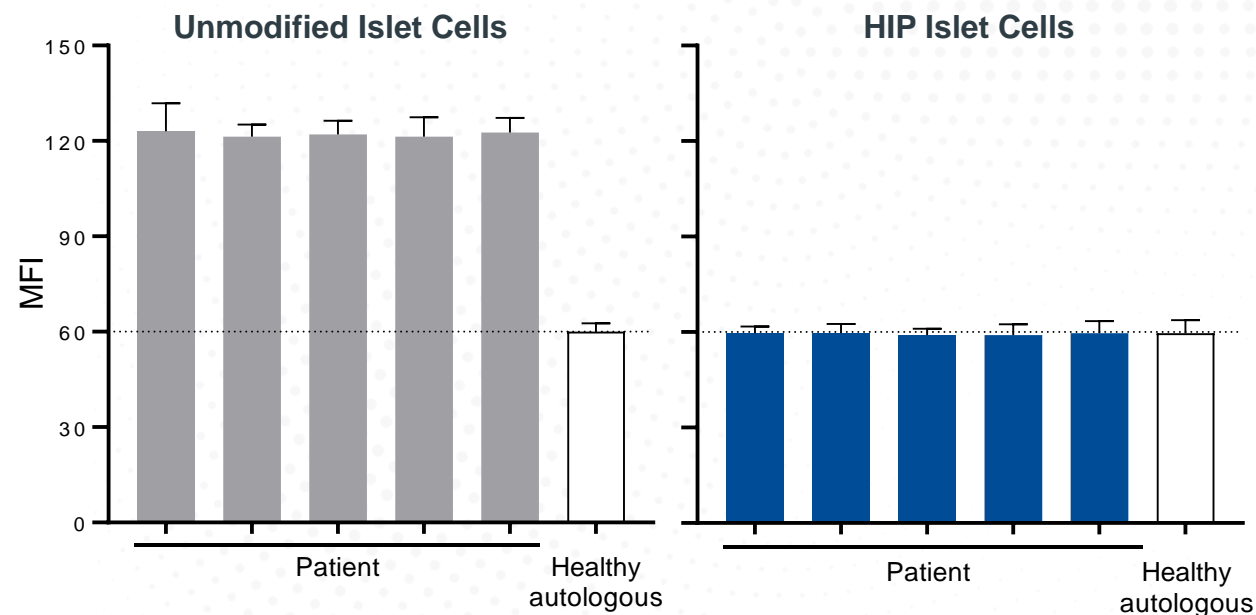
Hypoimmune pancreatic islet cells evade autoimmune killing in testing of blood from type 1 diabetes patients

T cells from PBMCs of type 1 diabetes patients kill unmodified islets, but not HIP islet cells



Antibodies from sera of type 1 diabetes patients bind to unmodified islets, but not HIP islet cells

Serum from healthy volunteers or type 1 diabetes patients

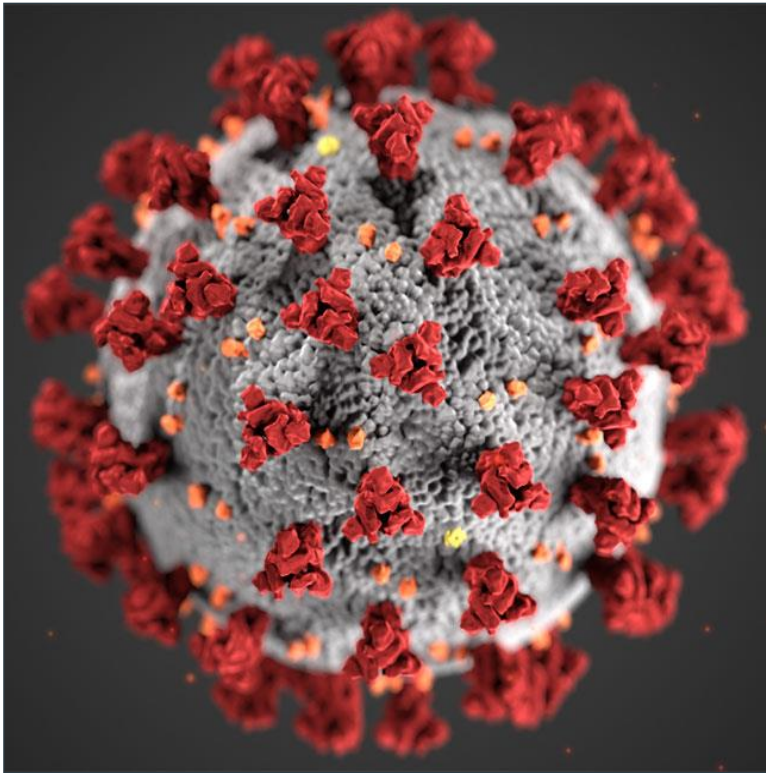


Robust GMP supply chain required to use iPSC-based therapies as medicines

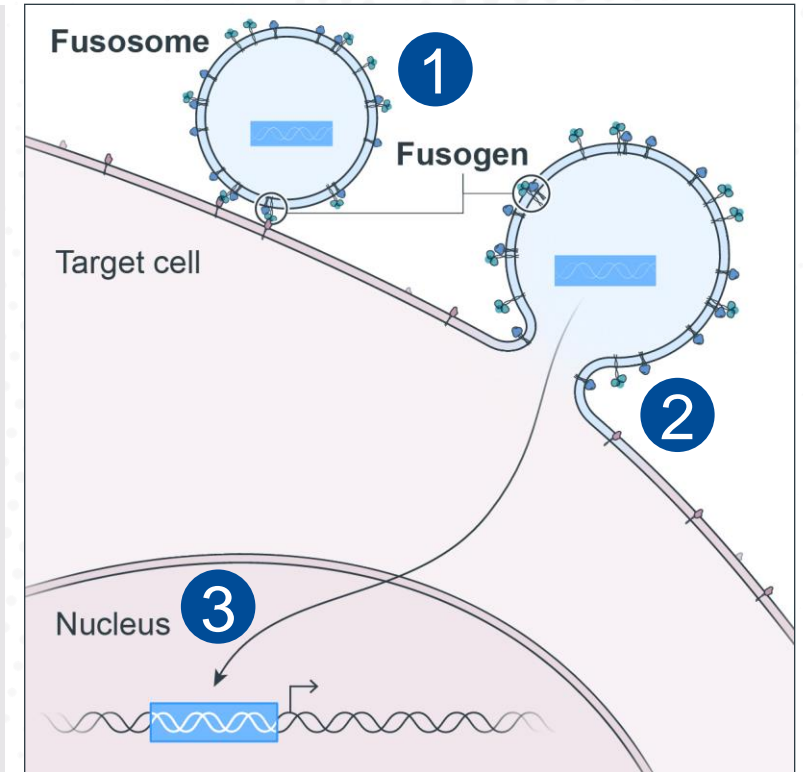
- | | | |
|---|--|--|
| 1 | GMP genomically stable cell lines | FCDI licenses and bespoke lines |
| 2 | GMP gene editing reagents | Beam license enables editing requirements for current programs |
| 3 | GMP gene-edited master cell bank | Creating internal master cell banks for GMP HIP-edited iPSCs |

Fusosome technology: Development of cell-specific *in vivo* delivery platform

Sana approach: Leverage insights from nature to deliver various payloads to specific cells

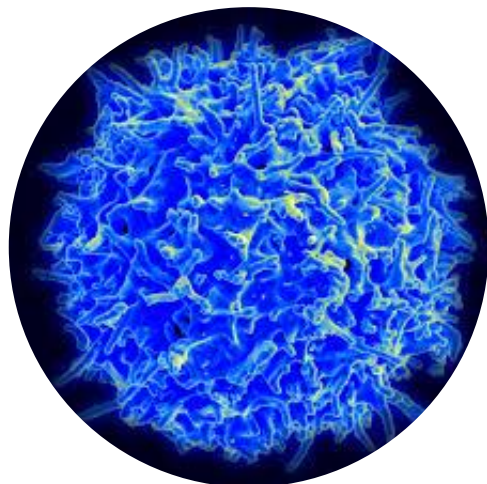


Source: CDC website

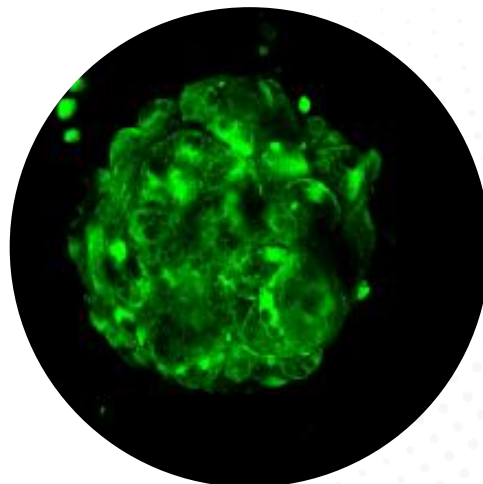


In vivo cell engineering: Creating targeted medicines across a diverse set of cell types

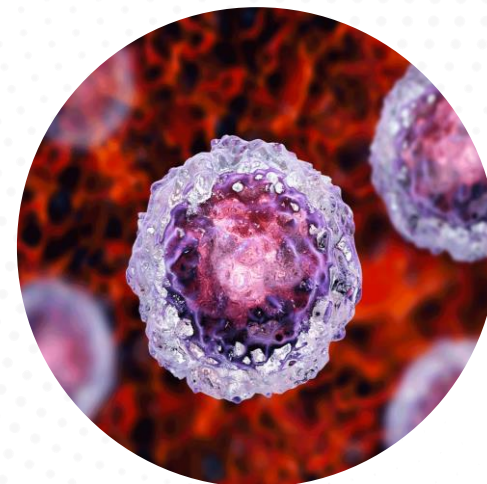
in vivo cell engineering strategy focused on developing therapies with transformative **fusogen platform delivery based on cell specificity and payload diversity**



T cells



Hepatocytes

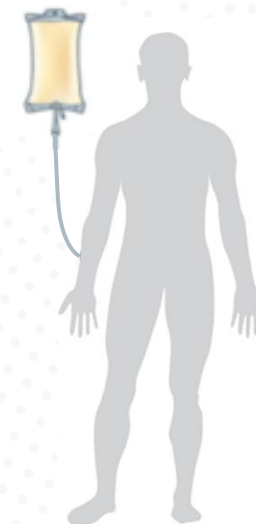
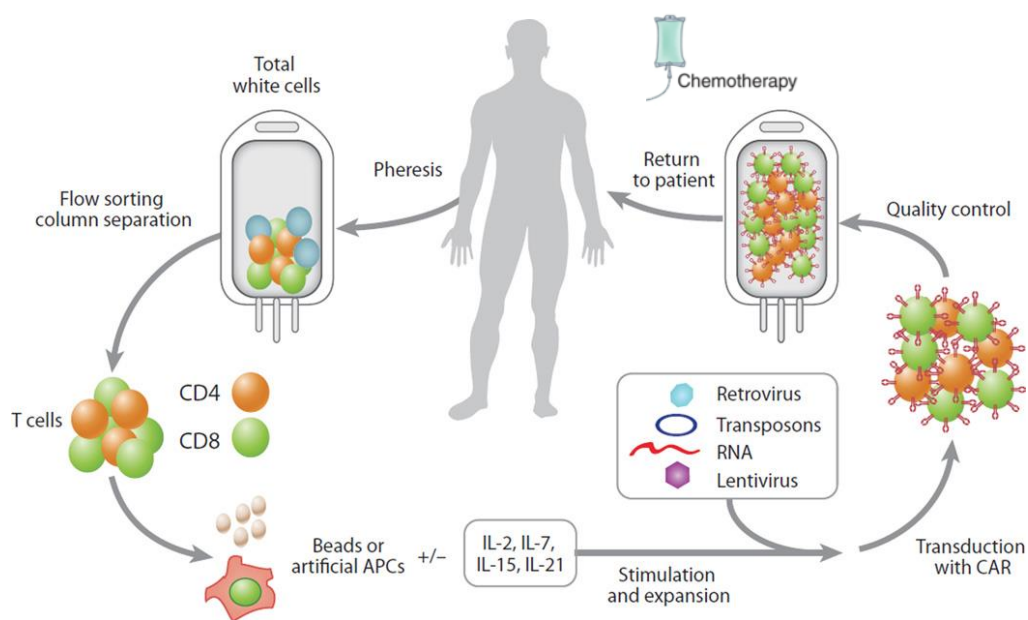


Hematopoietic
stem cells

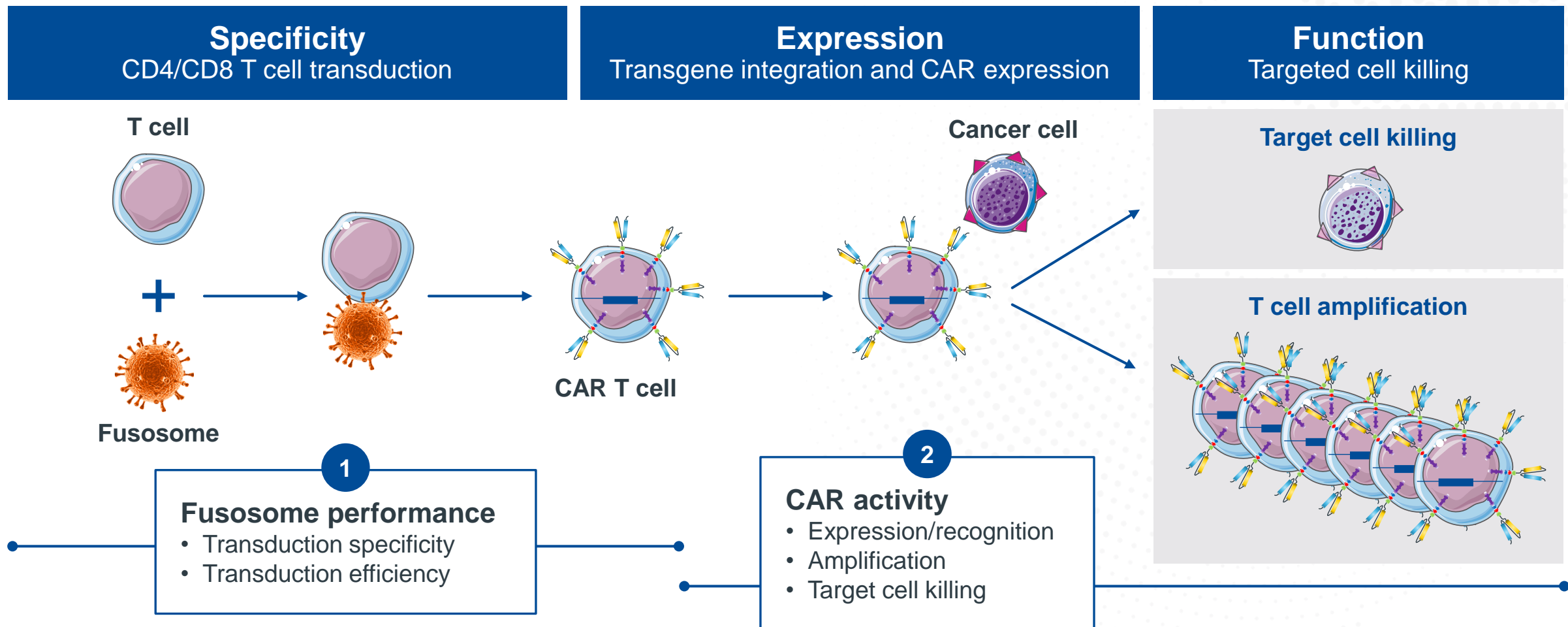
High unmet need remains for blood cancers

Current *ex vivo* approaches have limitations

Fusogen platform offers potential to overcome these limitations



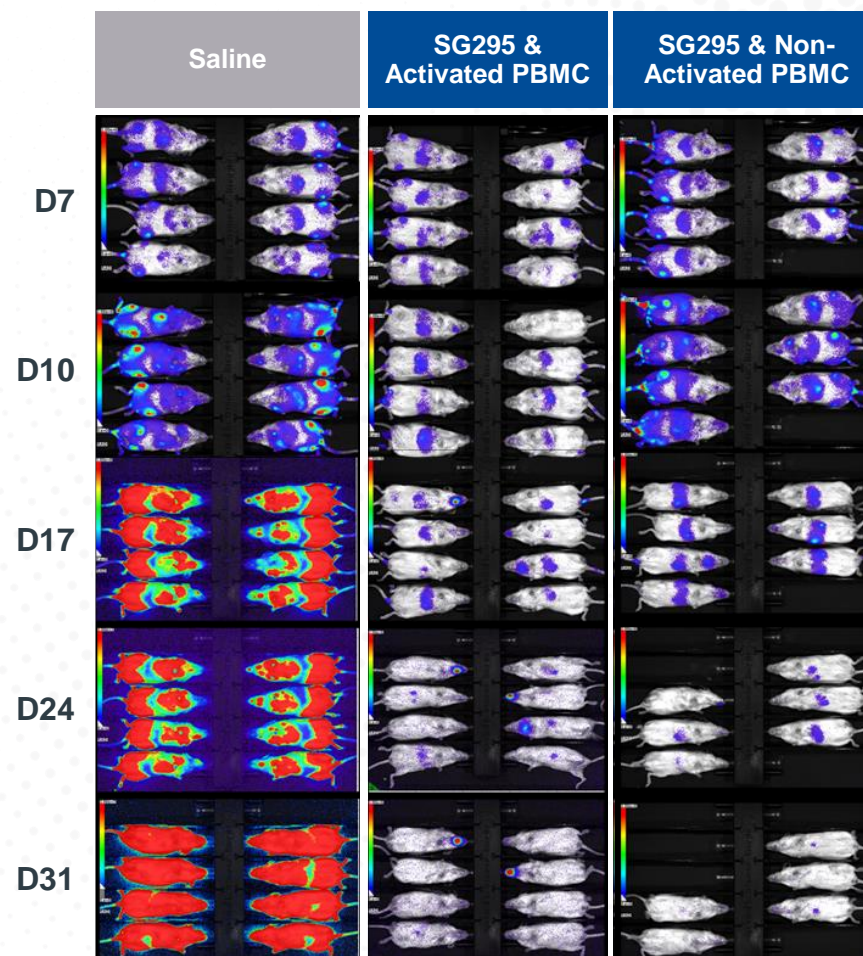
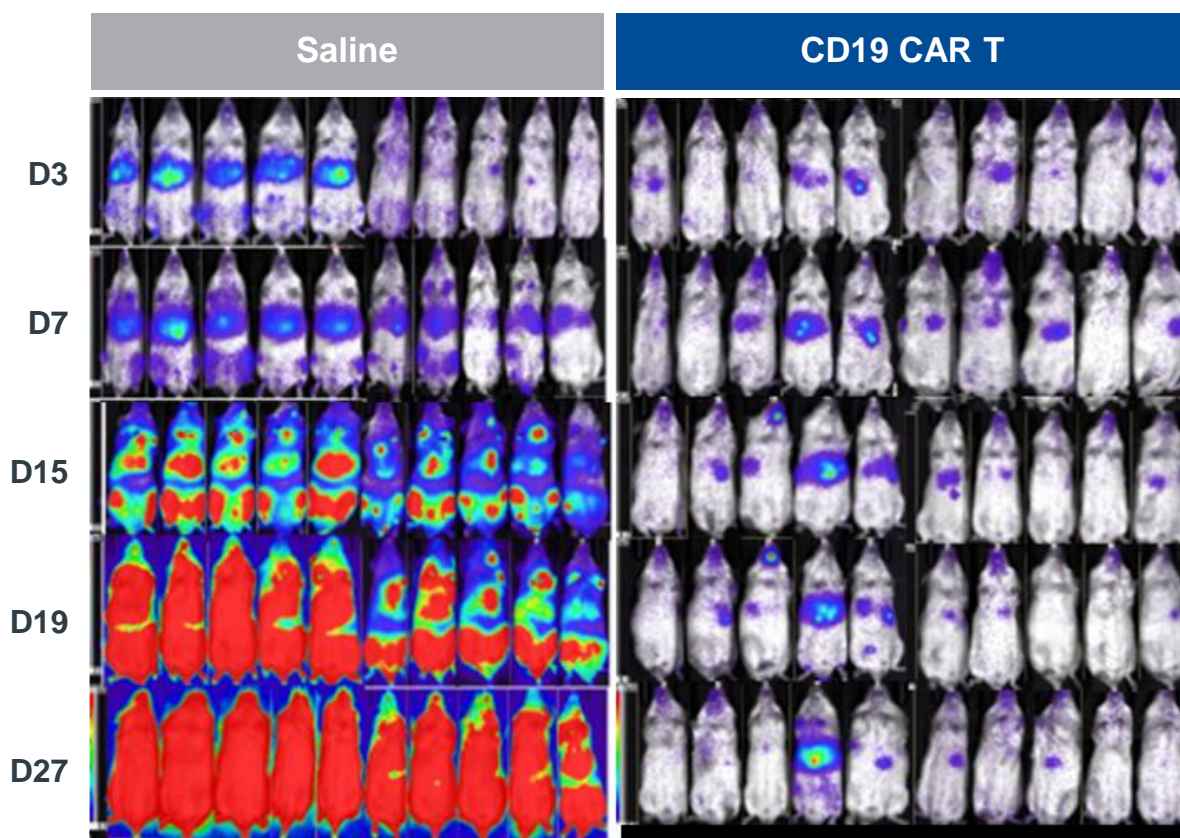
T cell fusosome carrying CAR construct infused into patient



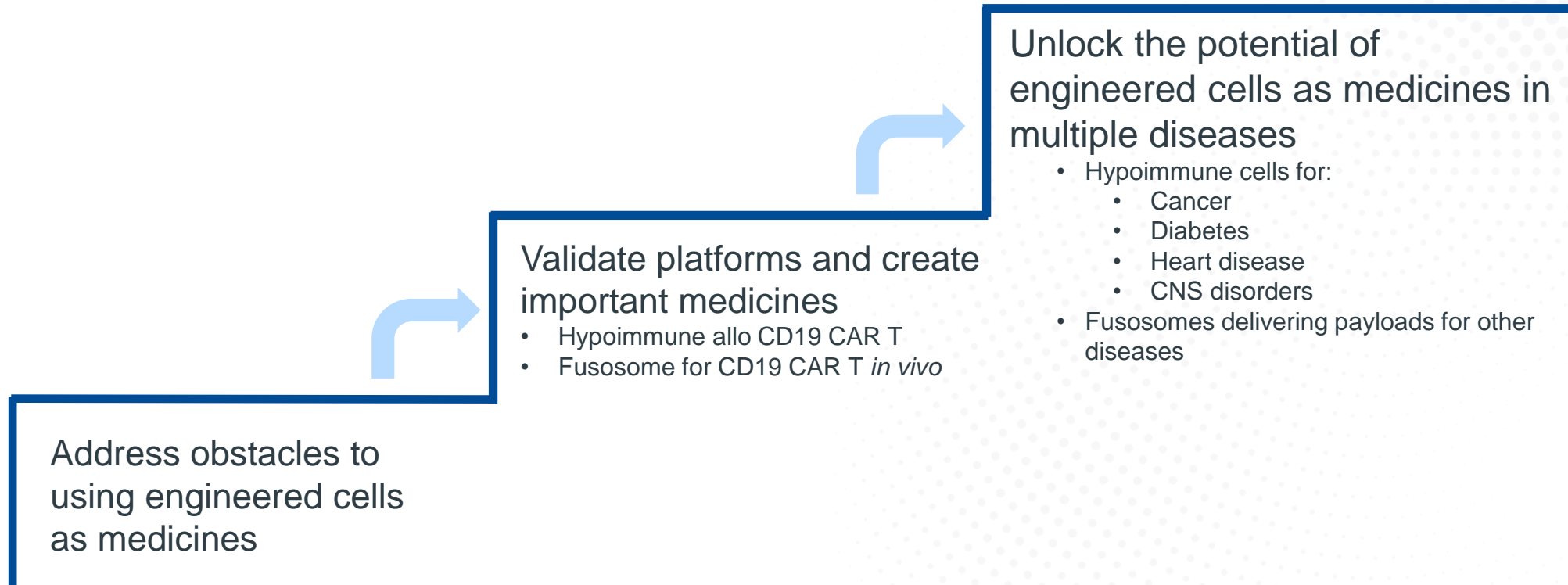
IV administration of CD19 CAR delivered by fusosome can clear B cell tumors in humanized mice comparably to *ex vivo* CD19 CAR T

CD19 CAR delivered by fusosome: *in vivo*

CD19 CAR T: *ex vivo*



Sana aspiration: Engineered cells as medicines



Thank You

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