# Corporate Presentation

January 2025

Sana Biotechnology®

# Cautionary Note Regarding Forward-Looking Statements

**This presentation contains forward-looking statements about Sana Biotechnology, Inc.** (the "Company," "we," "us," or "our") within the meaning of the federal securities laws. All statements other than statements of historical facts contained in this presentation, including, among others, statements regarding the Company's strategy, expectations, cash runway and future financial condition, future operations, and prospects, are forward-looking statements. In some cases, you can identify forward-looking statements by terminology such as "aim," "anticipate," "assume," "believe," "contemplate," "continue," "could," "design," "due," "estimate," "expect," "goal," "intend," "may," "objective," "plan," "positioned," "potential," "predict," "seek," "should," "target," "will," "would" and other similar expressions that are predictions of or indicate future events and future trends, or the negative of these terms or other comparable terminology. The Company has based these forward-looking statements largely on its current expectations, estimates, forecasts and projections about future events and financial trends that it believes may affect its financial condition, results of operations, business strategy and financial needs. In light of the significant uncertainties in these forward-looking statements, you should not rely upon forward-looking statements as predictions of future events. These statements are subject to risks and uncertainties that could cause the actual results to vary materially, including, among others, the risks inherent in drug development such as those associated with the initiation, cost, timing, progress and results of the Company's current and future research and development programs, preclinical studies, and clinical trials.

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 its Quarterly Report on Form 10-Q dated November 8, 2024. Except as required by law, the Company undertakes no obligation to update publicly any forward-looking statements for any reason.

# Sana Biotechnology

## Changing the Possible for Patients

**Recent data confirm Sana's hypoimmune platform (HIP) overcomes allogeneic rejection in people**

- Transplanted HIP-modified pancreatic islets overcome allogeneic and autoimmune rejection in type 1 diabetes
- We believe T1D result generalizable across many cell types and patient populations

**HIP technology provides foundation for multiple drugs across multiple large therapeutic areas**

- Type 1 diabetes – SC451
- B-cell mediated autoimmune diseases (lupus, vasculitis, others) – SC291
- Blood cancers – SC262

**Fusogen platform proof of concept for *in vivo* CAR T cells**

- Potential for potent CAR T cells with no conditioning chemotherapy and opportunity to transform the autoimmune landscape

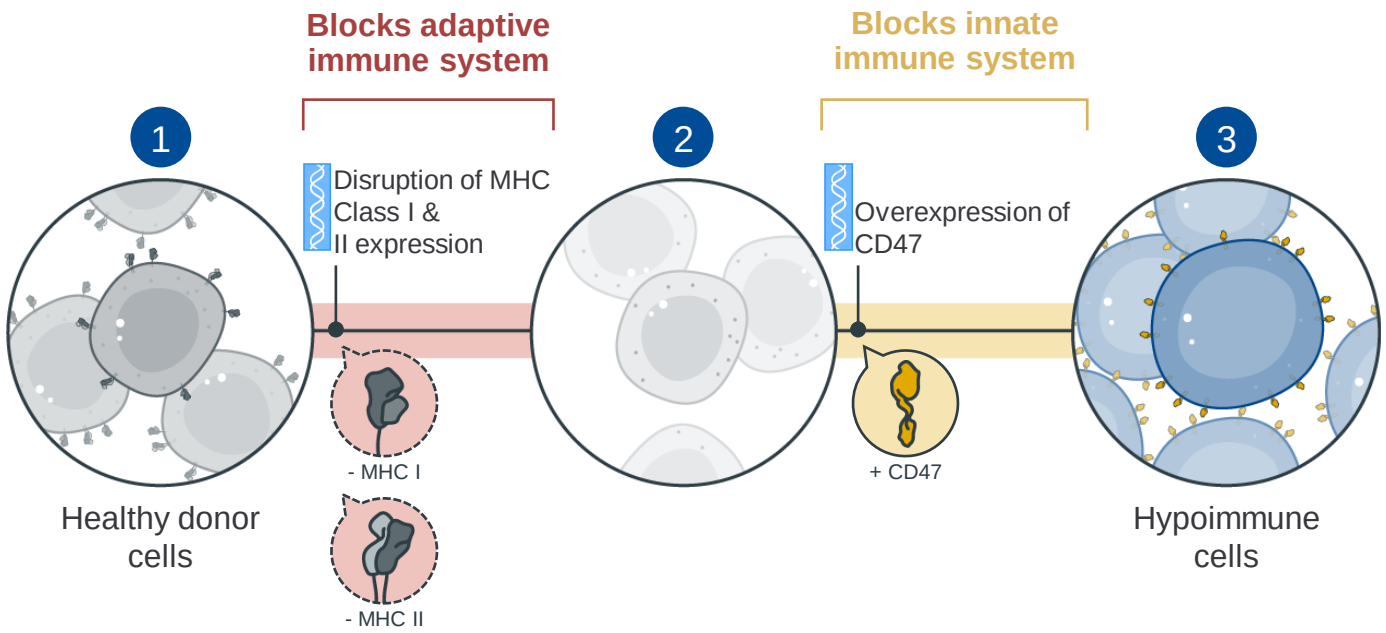
**Balance sheet allows potential for multiple data readouts**

# Overcoming allogeneic immune rejection has been key limitation in transplant and cellular medicine

## Allogeneic cell rejection

- ~75 years of transplants – immune rejection remains the largest problem
- Lifelong immunosuppression is current standard
- Genome modification efforts to date have generally been incomplete
- Autologous therapies have limited scalability and are only available for a small number of cell types
- Sana has published and/or presented positive data with the HIP platform showing the ability to overcome allogeneic rejection from many cell types and multiple species

## Sana's hypoimmune approach

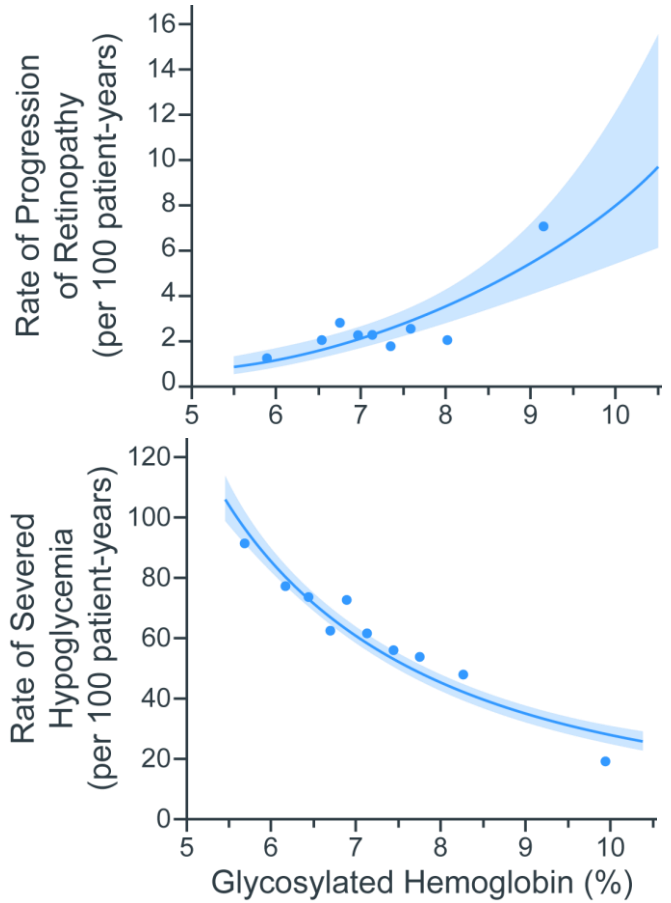



Current clinical platform with multiple ongoing approaches in research phase.

# Type 1 diabetes (T1D) remains a significant unmet need

- T1D is an autoimmune destruction of insulin-producing pancreatic beta cells and leads to lifelong insulin therapy requirement
- 8.4M people WW have T1D, and incidence is increasing. Prevalence is expected to double over next 15 years
- 80% of individuals with T1D are from high-income countries
- Insulin therapy has been transformative, but not curative
- T1D leads to more than a decade shorter life expectancy despite significant advances such as continuous glucose monitoring, insulin pumps, and novel forms of insulin
- Complications directly related to hyperglycemia include microvascular (retinopathy, nephropathy, neuropathy) and macrovascular (coronary artery disease, heart attacks, stroke, wound healing, and amputations) issues
- At the other extreme, severe hypoglycemia can be rapidly fatal

**Improvement in glycemic control reduces microvascular complications but increases the risk of hypoglycemia[1]**




[1]Diabetes Control and Complications Trial Research Group et. Al. N Engl J Med 1993; 329:977-986

# Advancing toward a cure for broad T1D population

## T1D is a disease of missing pancreatic beta cells


**Primary islet transplants provide long-term glucose control, but:**

- Supply is an issue
- Requires chronic Immunosuppression

**The Goal:**

**A single treatment with long-term normal blood glucose without immunosuppression or insulin therapy**


**Eliminate the need for immunosuppression**


**Stem-cell derived islets provide a scalable supply, but:**

- Still requires chronic immunosuppression

Abbreviations: T1D, type 1 diabetes.

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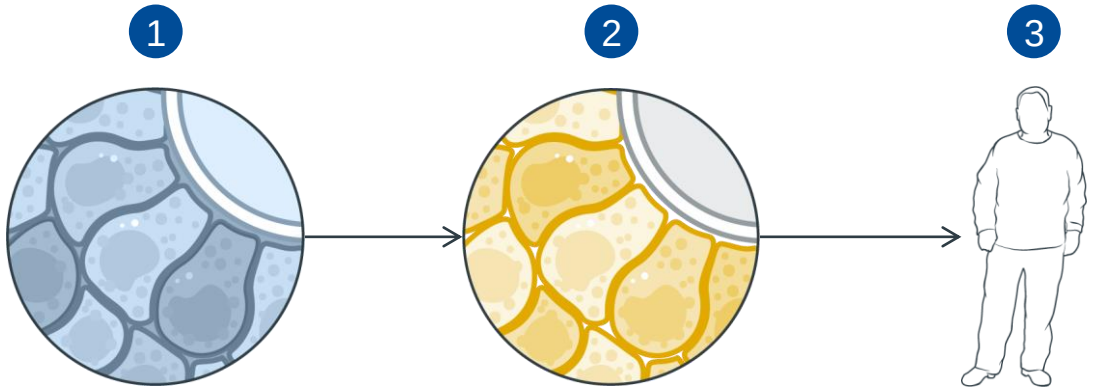
# Potential clinical validation of hypoimmune islet cells in T1DM patients

- Trial at Uppsala University Hospital
- Primary human HIP-modified islet cells transplanted in type 1 diabetes patients
- Intramuscular administration in forearm
- No immunosuppression
- Insights for SC451

**IST Design**




**Key Measured Outcomes**

Safety
Immune evasion
Cell survival
C-peptide

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# All primary and secondary endpoints met

## Primary and Secondary Endpoints: Data Summary

| Endpoints | D7 | D14 | D21 | D28 |
|---|---|---|---|---|
| **Safety (no AE/SAE related to drug)** | ✓ | ✓ | ✓ | ✓ |
| **Cell survival/function (C-peptide)** | ✓ | ✓ | ✓ | ✓ |
| **Graft visibility (MRI)** | ✓ | ✓ | Not performed (as per protocol) | ✓ |
| **Adaptive immune evasion** | ✓ | ✓ | ✓ | ✓ |
| **Innate immune evasion** | ✓ | ✓ | ✓ | ✓ |

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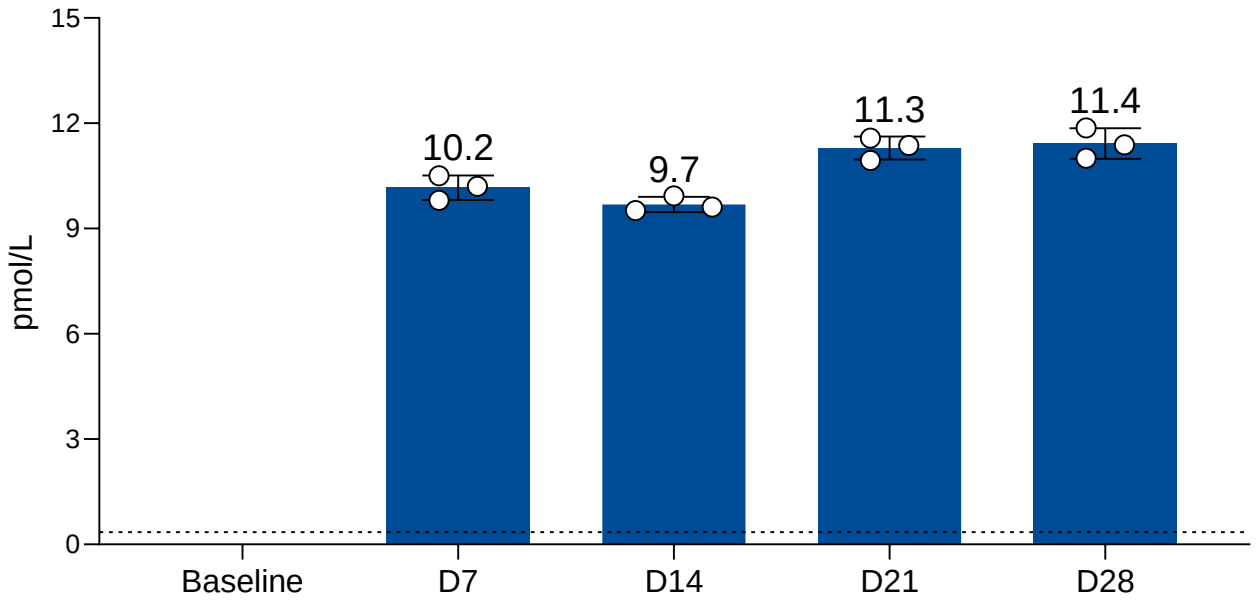
# Stable C-peptide demonstrates survival and function of cells after HIP islet cell transplantation

## Basal C-peptide in peripheral blood




**Summary:** No detectable C-peptide before transplantation; present and stable C-peptide observed after transplantation.

*Baseline: Below limit of detection (LOD).Sensitivity: 0.48 pmol/L. Dots represent technical triplicates. C-peptide analyzed in serum.*

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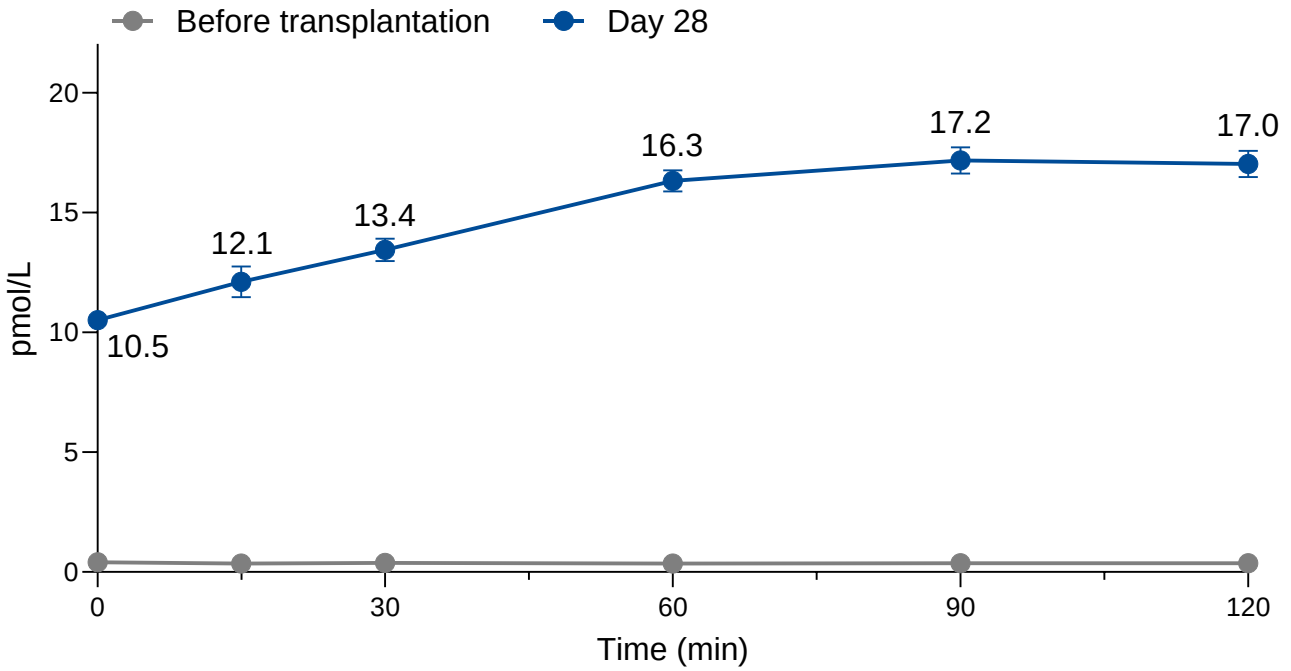
# Increased C-peptide levels with a mixed meal tolerance test (MMTT) highlight survival and function

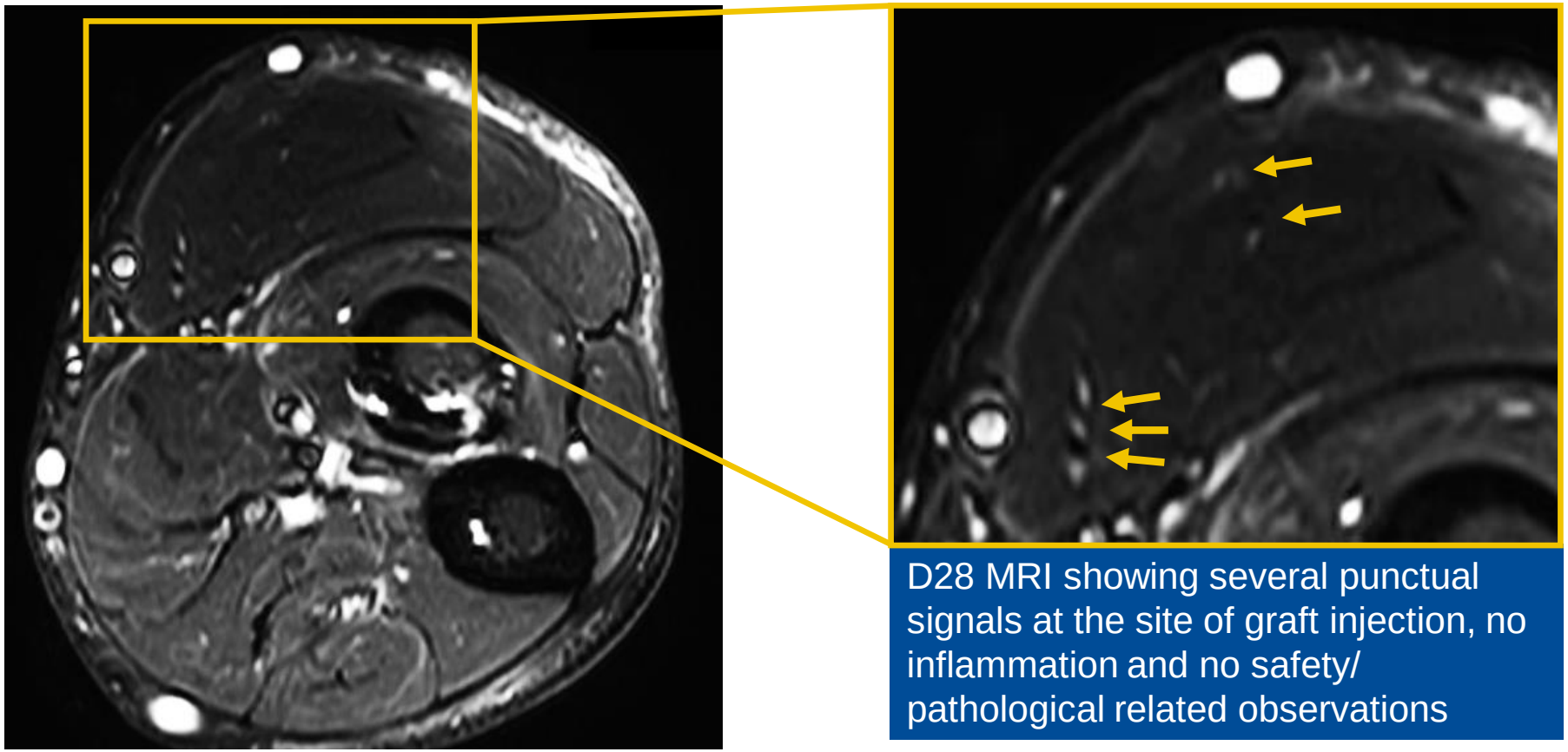



**Summary:** Before transplantation, C-peptide is below detection limit during MMTT. 28 days after UP421 transplantation, C-peptide is present and stimulated by MMTT.

Baseline: Below limit of detection (LOD).Sensitivity: 0.48 pmol/L.
Standard deviation represent technical triplicates. C-peptide analyzed in plasma samples.

# Day 28 MRI: Further evidence of graft survival

## MR T2-STIR-weighted trans images showing signal in musculus brachioradialis after injection of UP421



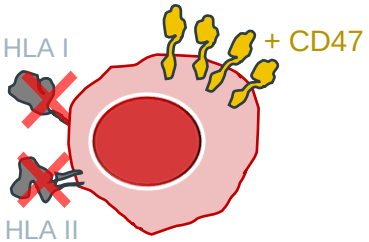
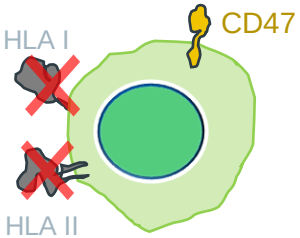
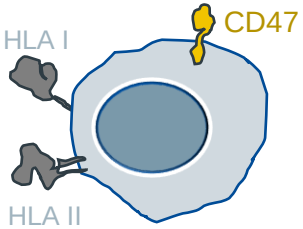
Arrows indicate the location of some examples of injected cells

D28 MRI showing several punctual signals at the site of graft injection, no inflammation and no safety/ pathological related observations

The MR T2-STIR-weighted sequence is sensitive to water and fluid and is a fat suppression technique to suppress the high signal from fat.
Abbreviations: STIR, short TI inversion recovery.

# The drug product's mixed cell population allows detailed immune analysis

## Donor islet cells contain wild type & double knockout cells as well as HIP cells

| Donor | |
|---|---|
| **HIP islet cells** |   |
| **dKO islet cells** |   |
| **WT islet cells** |   |

Abbreviations: dKO, double knock-out; HIP, hypoimmune; WT, wild type.

**Immune analysis using patient's (recipient) immune cells after transplantation**

- T cells
- Donor-specific antibodies
- Natural killer cells
- Whole blood

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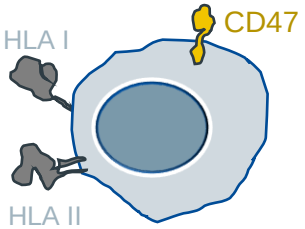
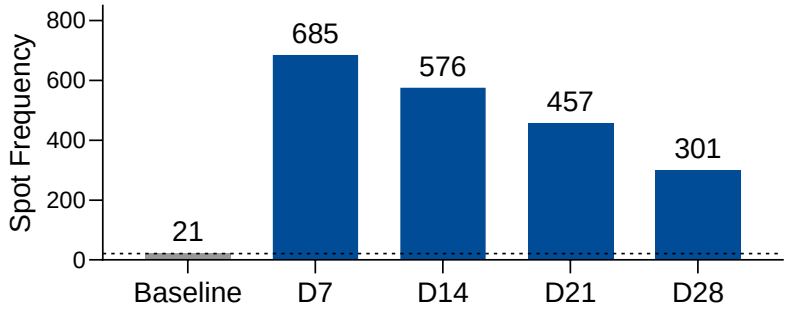
# Unmodified islet cells: Do **not** evade T cell or B cell immune response

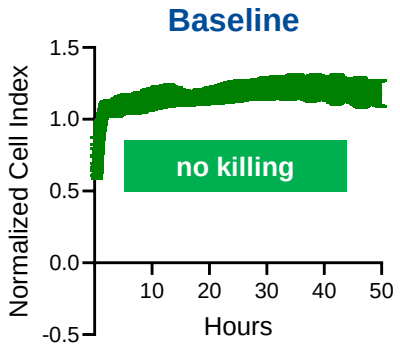
## WT islet cells

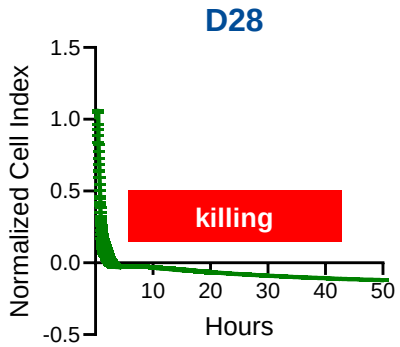



## WT islet cells

Patient's T cells are activated and kill WT islet cells with peak at 7 days after transplant

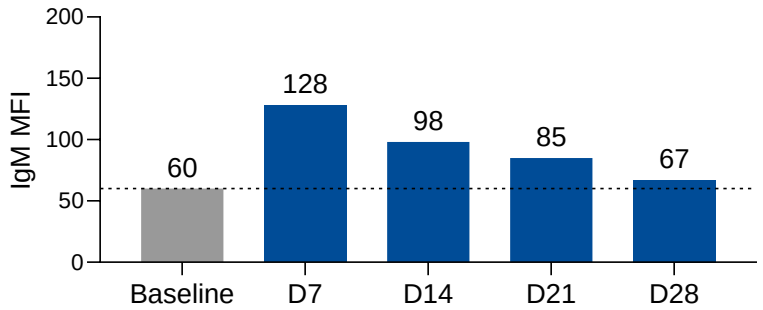


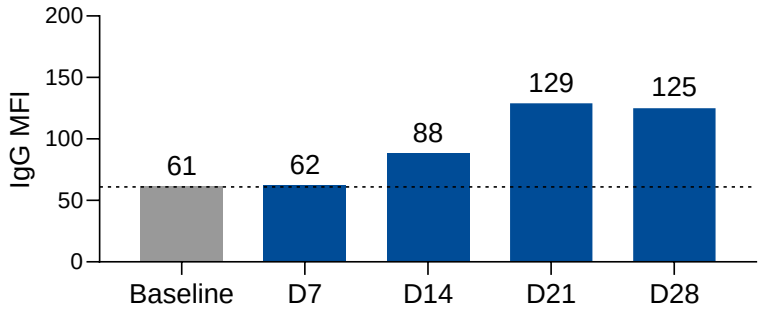




## WT islet cells

Patient's B cells produce donor-specific antibodies (switch from IgM to IgG at D14)

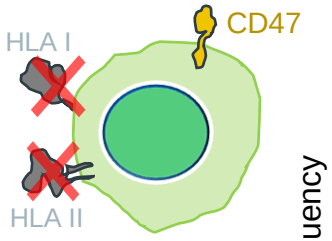



Abbreviations: D, day; WT, wild type.

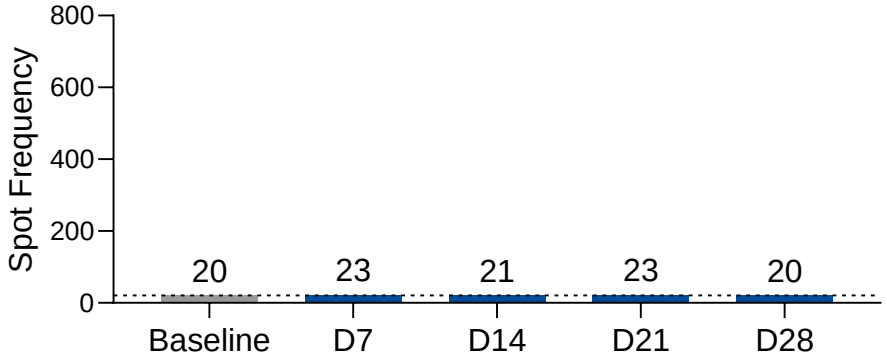
# dKO islet cells: Evade B and T cell responses but **are killed** by NK cells

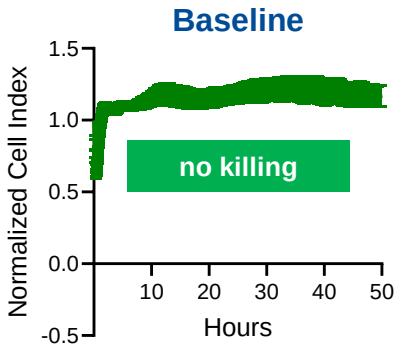
## dKO islet cells

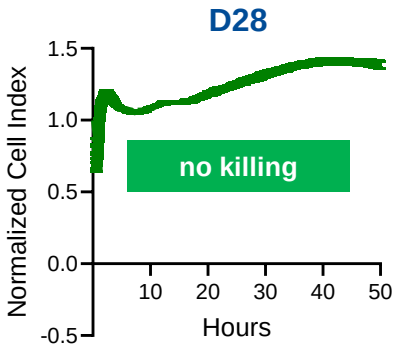



## dKO islet cells

No killing of HLA deficient cells by patient's T cells

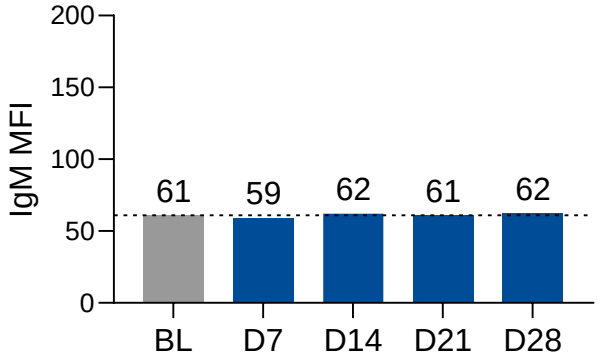


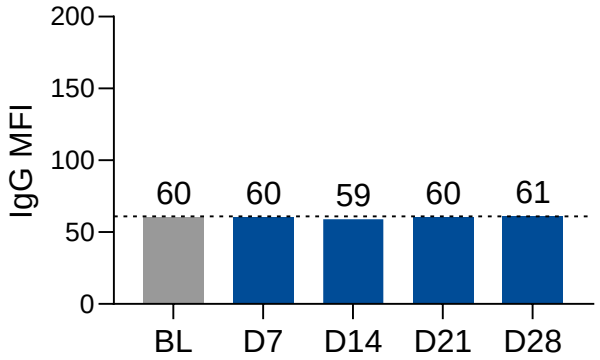




## dKO islet cells

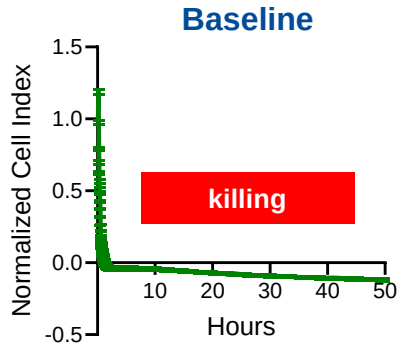
No binding of donor-specific antibodies to HLA deficient cells

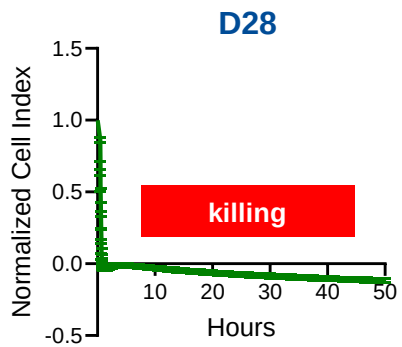



## dKO islet cells

Patient's NK cells are killing HLA I/II deficient cells (due to "missing-self")

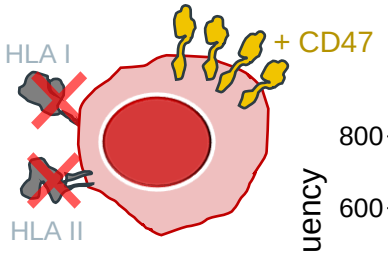



Abbreviations: BL, baseline.

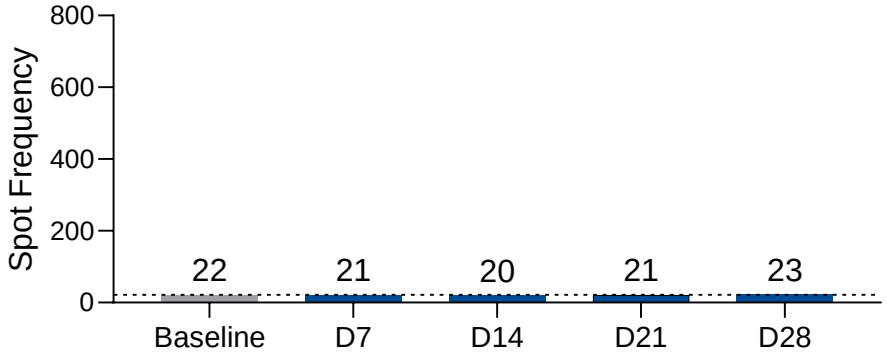
# HIP islet cells: Evade T cell, B cell, and NK cell immune responses

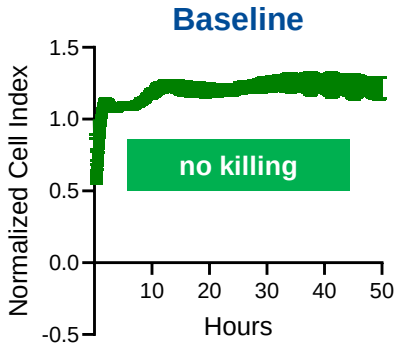
## HIP islet cells

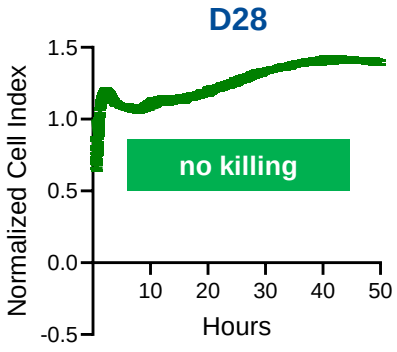



## HIP islet cells

No killing of HIP islet cells by patient's T cells

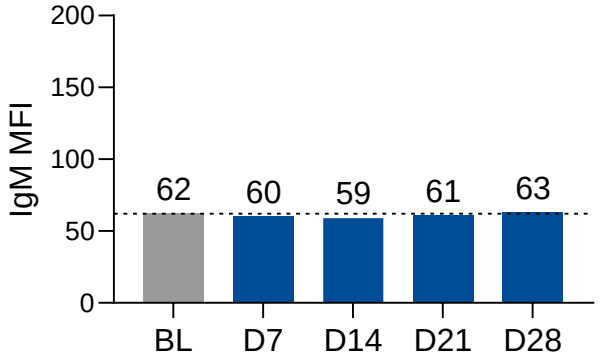


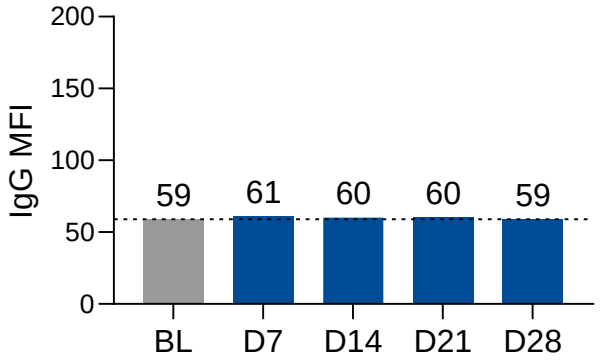




## HIP islet cells

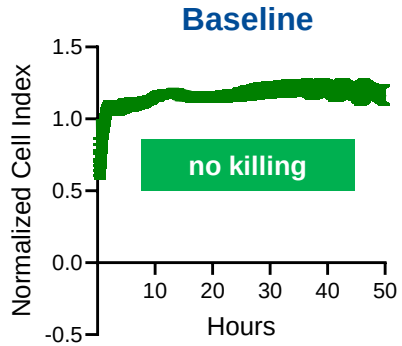
No binding of donor-specific antibodies to HIP islet cells

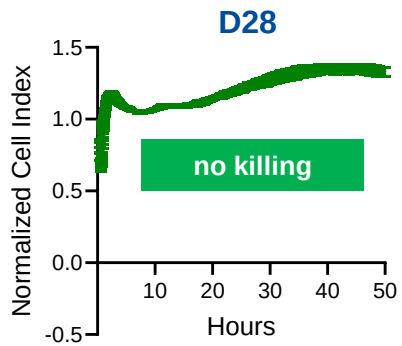



## HIP islet cells

No killing of HIP islet cells by patient's NK cells



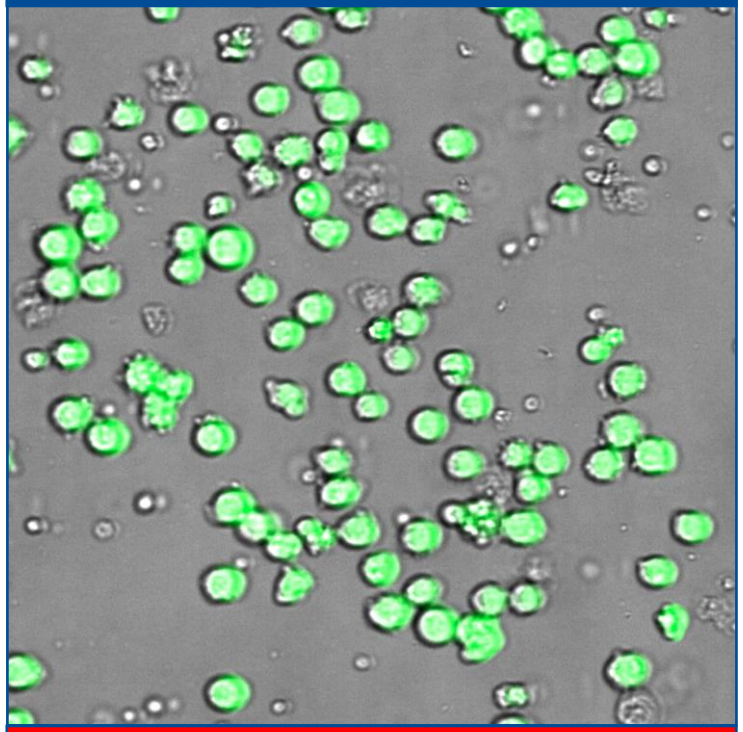
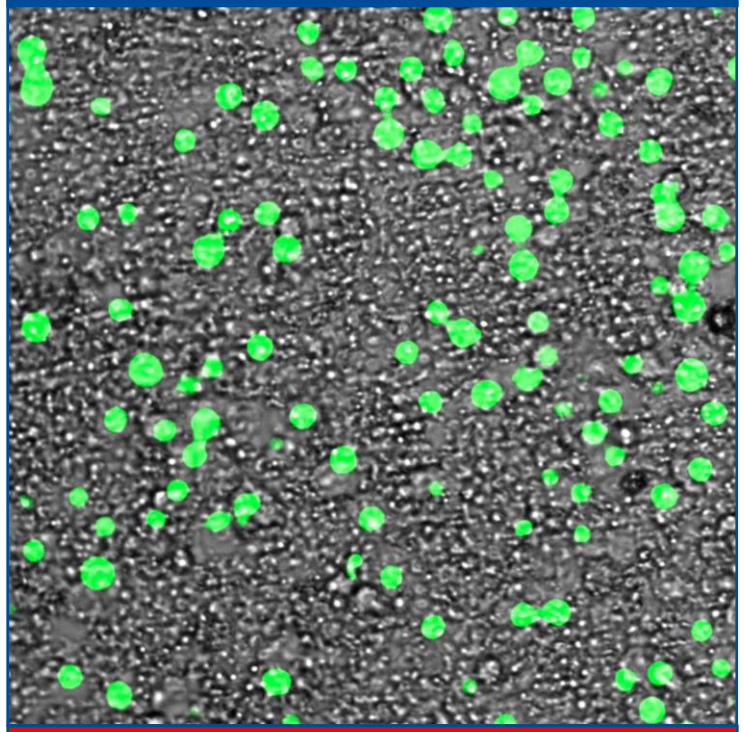
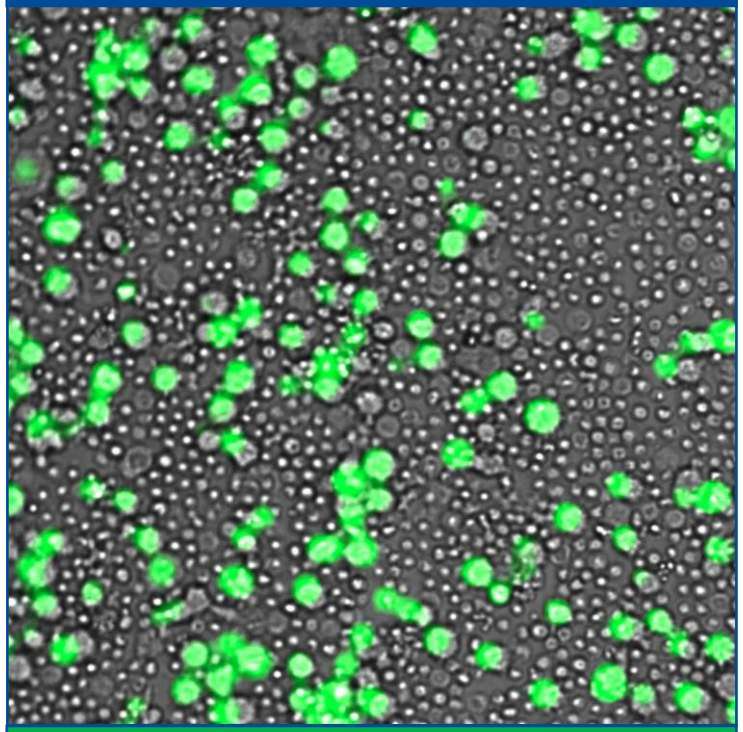
# HIP islet cells overcome patient's allogeneic and autoimmune barrier

*still image before movie*

**D7 sample:** PBMC (containing all immune cell populations) plus serum (containing antibodies and complement) killing assay

Donor islet cells with editing profile in column title

NK cells

| Target: WT islet cell | Target: HLAI/II dKO islet cell | Target: HIP islet cell |
|---|---|---|
|  |  |  |
| **Adaptive immune Killing** | **Innate immune Killing** | **No immune killing** |

Actual assay time = 4 hours.

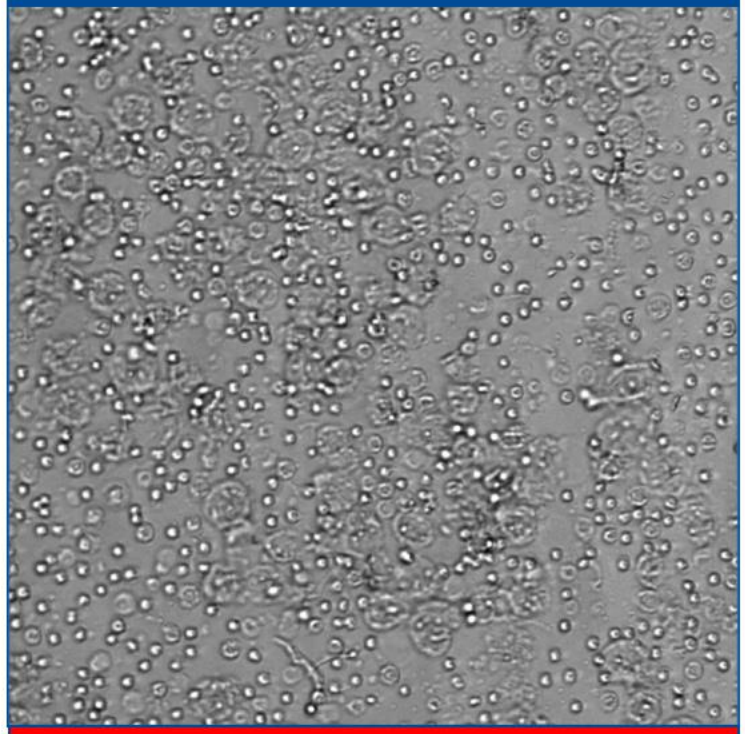
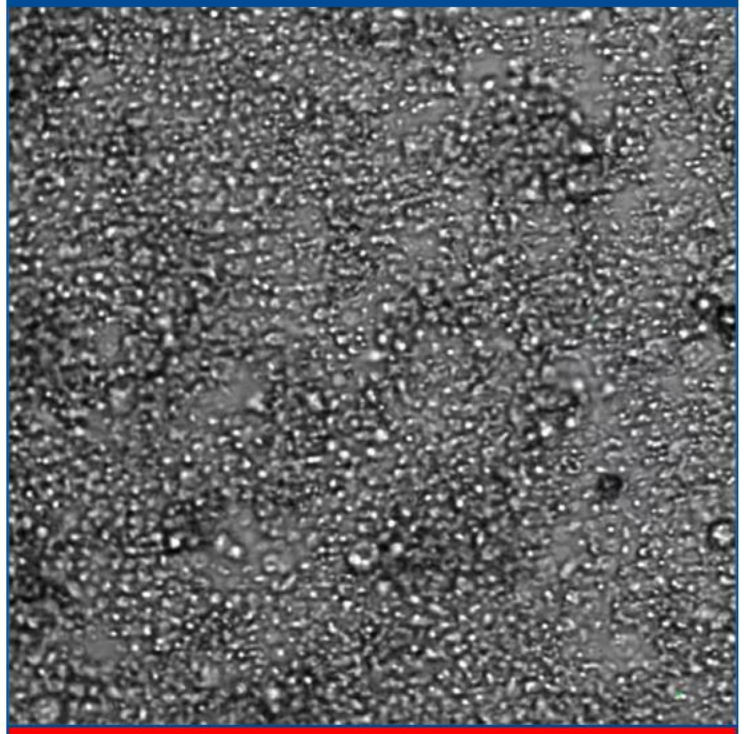
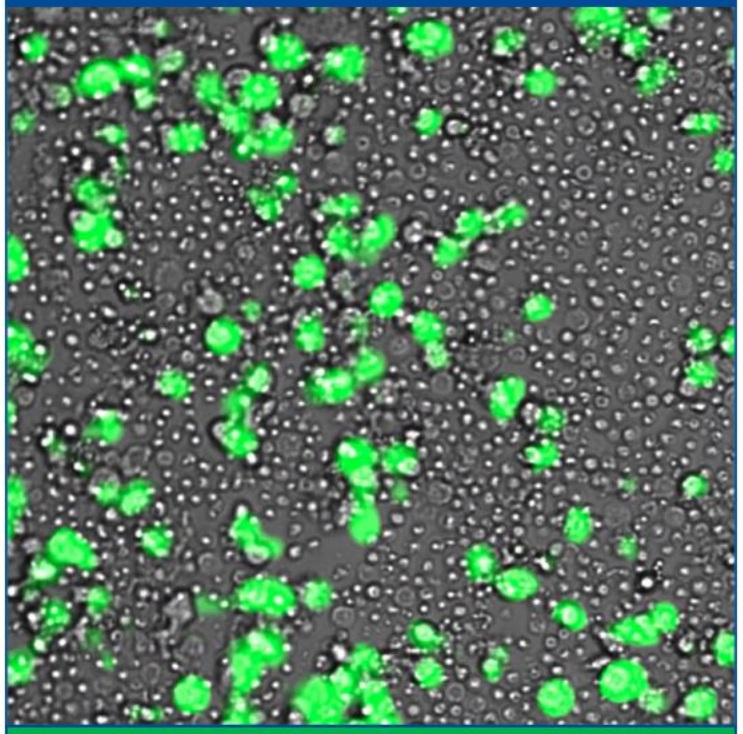
# HIP islet cells overcome patient's allogeneic and autoimmune barrier

*still image after movie*

**D7 sample:** PBMC (containing all immune cell populations) plus serum (containing antibodies and complement) killing assay

Donor islet cells with editing profile in column title

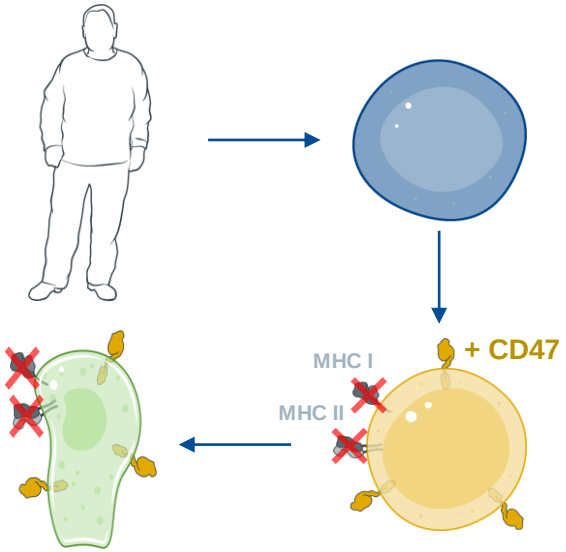
NK cells

| Target: WT islet cell | Target: HLAI/II dKO islet cell | Target: HIP islet cell |
|---|---|---|
|  |  |  |
| Adaptive immune Killing | Innate immune Killing | No immune killing |

Actual assay time = 4 hours.

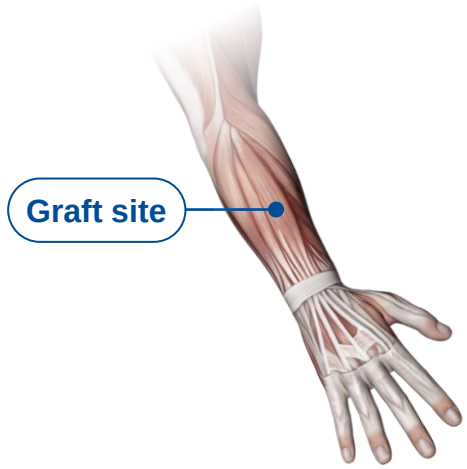
# SC451: A drug for the broad T1D population

## 1 Make hypoimmune islet cells from stem cells




## 2 Manufacture at scale

## 3 Deliver as a single therapy




**SC451 program – HIP stem cell-derived islet cell therapy delivered with no immunosuppression**

# Four major challenges to realizing the vision of SC451

1

**Overcoming immune rejection without immunosuppression**

*We believe this challenge has now been solved*

2

**Differentiating PSCs into islet cells at a purity, potency, and yield to enable clinical trial dosing**

*Many groups have done this successfully and so has Sana*

3

**Generating a gene-modified MCB from a GMP-compliant PSC line that is genetically stable and remains so after gene editing and differentiation into islet cells**

*We have done it in research*

4

**Manufacturing enough product to treat the patients that need it**

*We are working on the challenges of manufacturing at scale*

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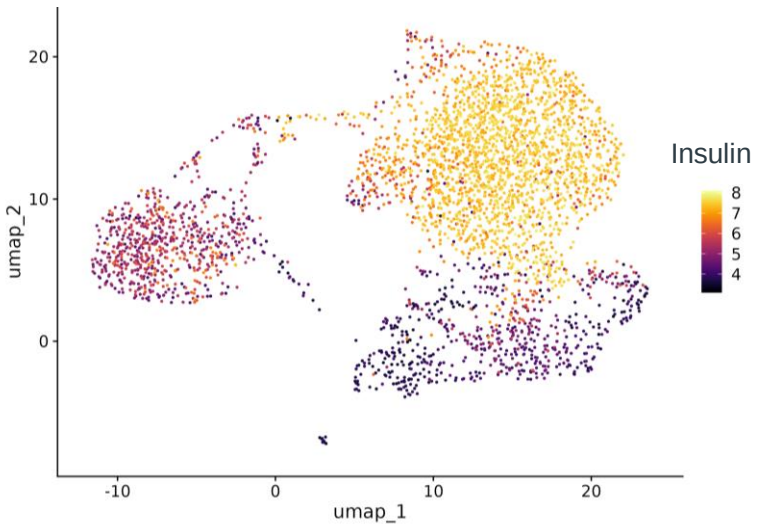
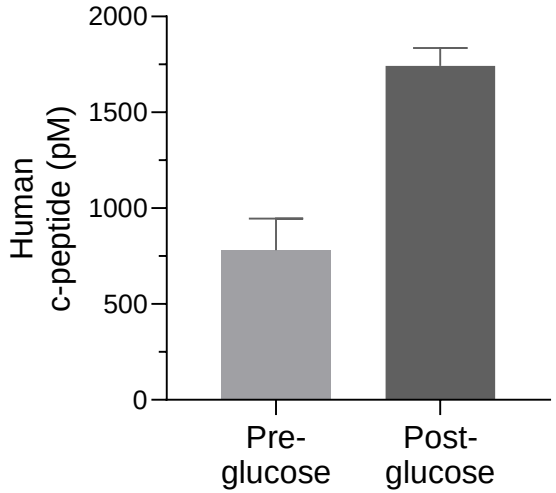
# HIP-modified PSC differentiated islet cells transplanted into muscle persist & control blood glucose in mice for >15 months

## PSCs differentiated into islets at high purity



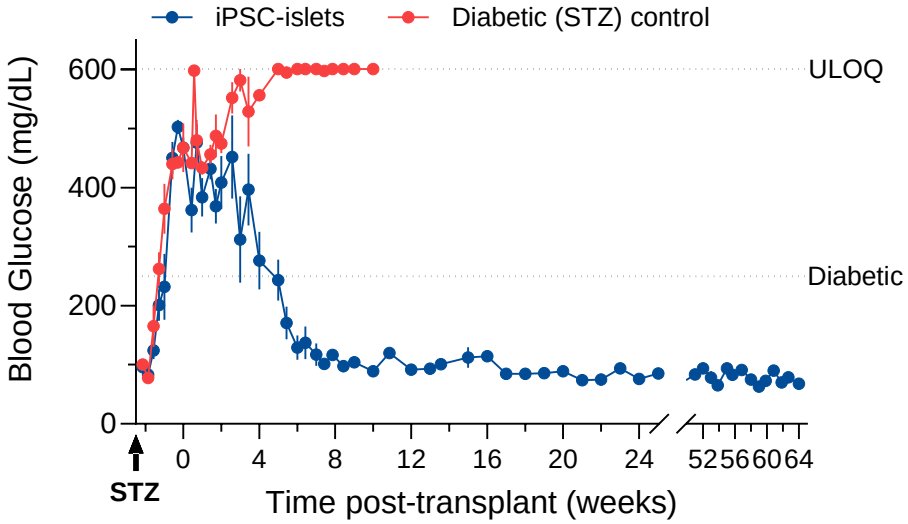

## HIP-modified PSC islets produce human c-peptide** *in vivo*

(performed at week 51)




## HIP-modified PSC islets persist and control blood glucose

(non-fasted blood glucose*)



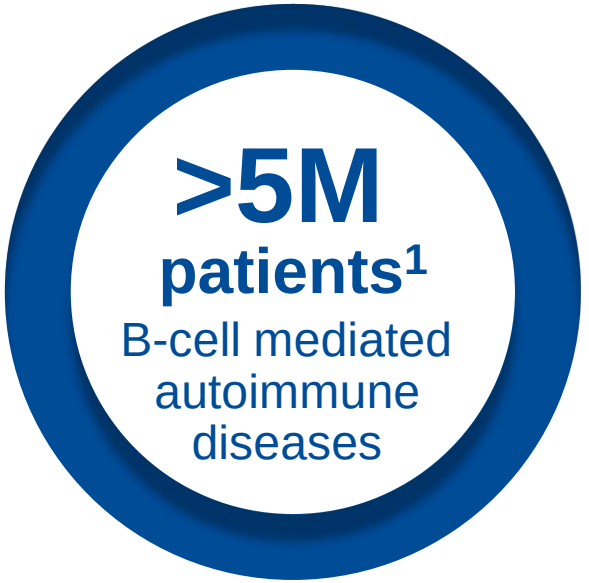

- Maintain normoglycemia (64+ weeks)
- Secrete c-peptide in response to glucose
- Retain strong expression of hCD47

Diabetic threshold at 250 mg/dL; data reported as mean ± S.E.M. **plasma human c-peptide after 5 hr fast (pre) and 30 min after I.P. 3 g/kg dextrose bolus (post); data is mean ± S.D.

# B-cells drive autoimmune disease in millions of patients

## >75 different types of autoimmune disorders with underlying B cell pathology and high unmet need

- SLE
- Vasculitis (granulomatosis with polyangiitis & microscopic polyangiitis)
- Neuromyelitis optical spectrum
- Pemphigus
- Relapsing and progressive MS
- Rheumatoid arthritis
- Lupus nephritis
- Sjögren's syndrome
- NMDAR encephalitis
- Thrombocytopenic purpura
- Amyloidosis
- Systemic sclerosis
- Autoimmune hemolytic anemia
- Chronic immune demyelinating polyradiculoneuropathy
- Immune-mediated necrotizing myopathy
- Membranous nephropathy




**Lupus Foundation of America** estimates over a million people have lupus in the US[2]

[1]Sana internal analysis; SciVida Autoimmune Factbook 2023, U.S; [2]www.lupus.org/resources/how-many-people-have-lupus-in-the-united-states.

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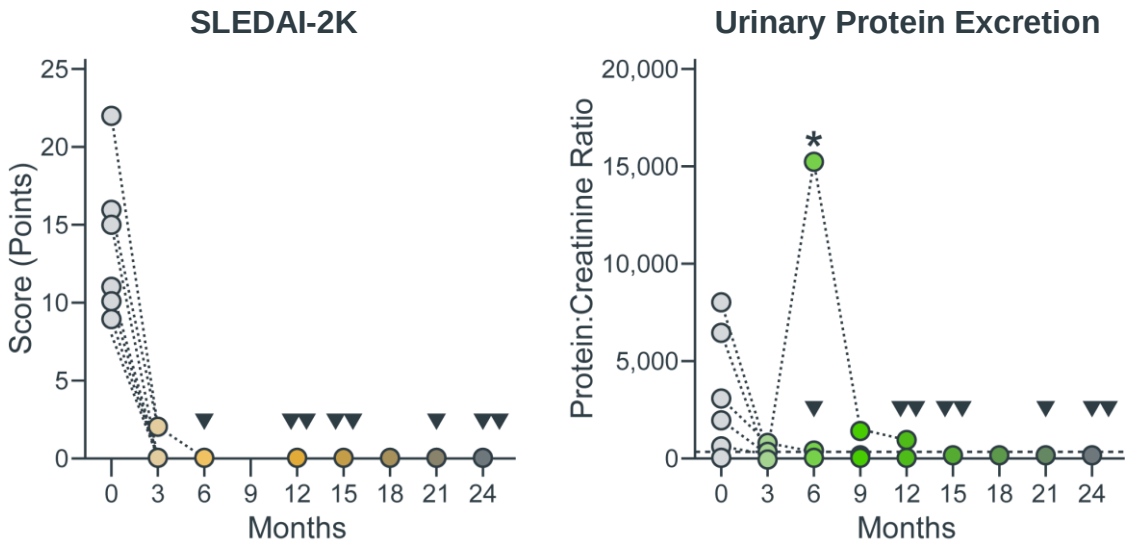
# Autologous CAR Ts have shown curative potential; allogeneic cells have inherent advantages

## CD19 Autologous CAR T treatment has delivered long term, drug-free remissions[1]




## Autologous CAR T Challenges

- Difficult to scale
- Prescription-to-infusion time over 4 weeks
- Patients must be taken off anti-inflammatory drugs for apheresis *and* treatment

## Allogeneic CAR T Promise

- Scalable
- Available to patients "off-the-shelf"
- No apheresis

**Allogeneic CAR Ts are the future**

[1]Muller F et al, NEJM 2024, Erlangen case series.

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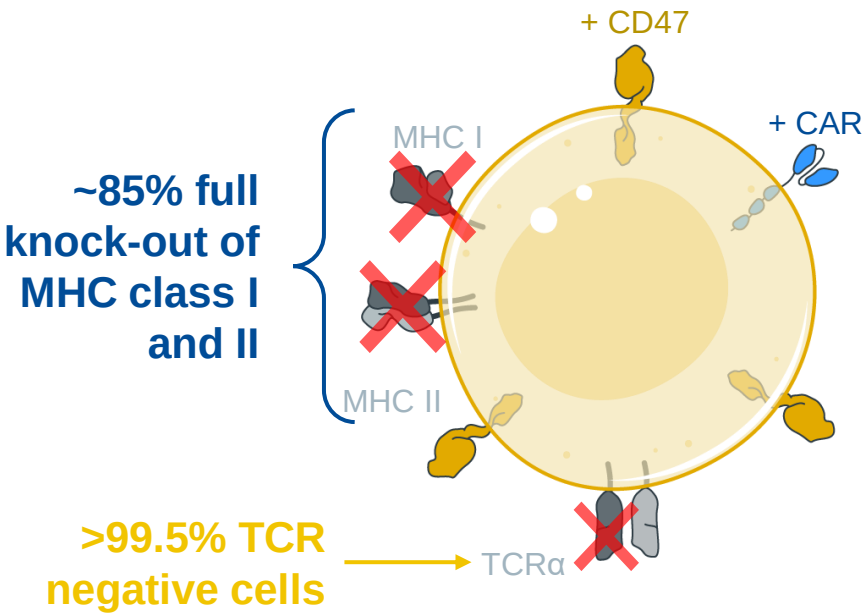
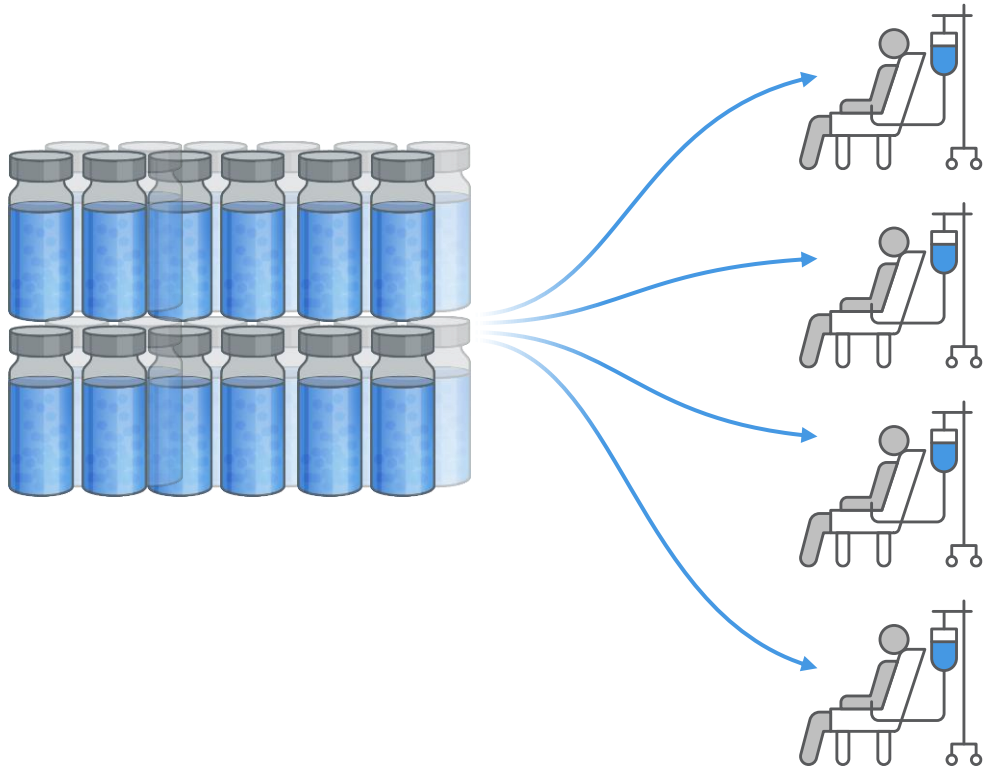
# Sana's T cell manufacturing process provides high yields of successfully edited cells

## SC291: Highly efficient editing of cells




## Product ready when the patient needs it

100s of autoimmune patient batches/manufacturing run[1]



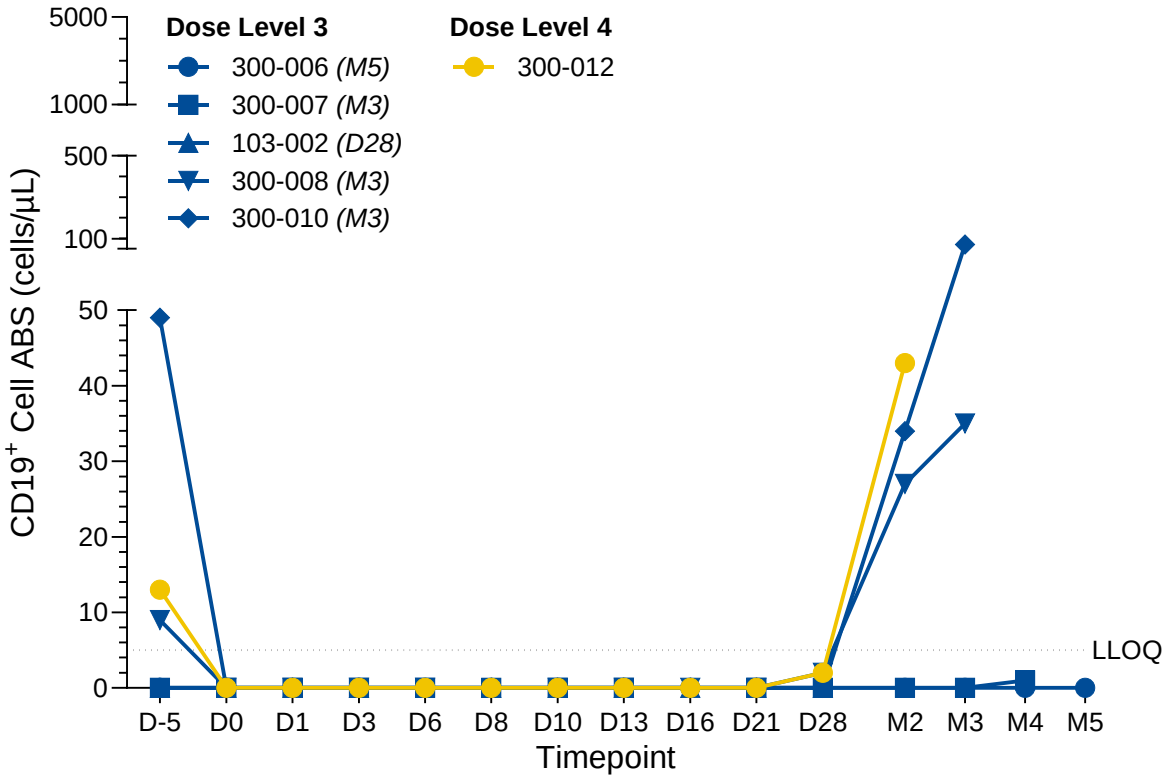
[1]100s of doses assumes an autoimmune dose consistent with public data on current autoimmune dose levels.

# SC291 can be safely administered and results in deep, dose-dependent B-cell depletion in oncology

## ARDENT Safety Data (N=16)

- No cases of Grade 2 or higher CRS
  - 3 cases of Grade 1 CRS
- No cases of ICANS
- 1 case of Grade 1 IEC-HS

### Deep B-cell depletion seen in NHL patients

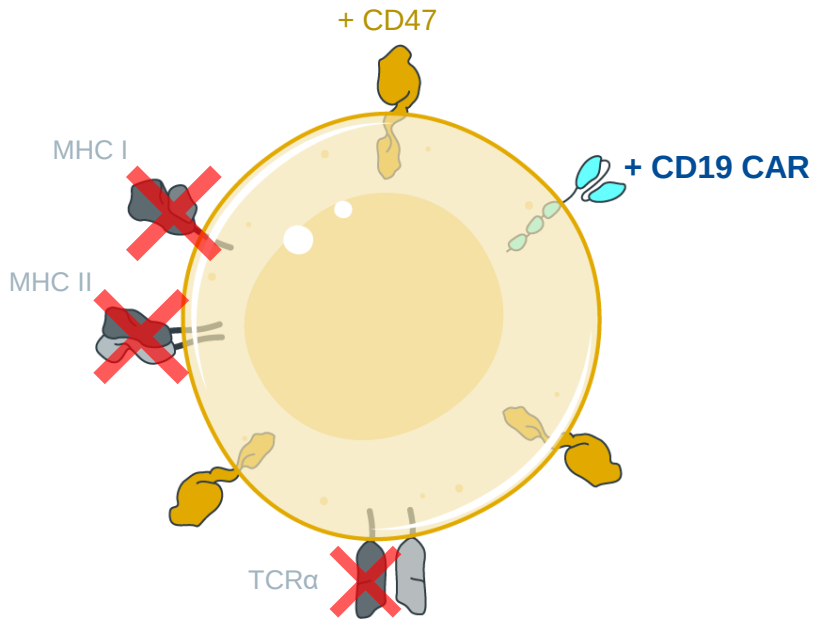



Data cutoff Nov 2024.
Abbreviations: CRS, cytokine release syndrome; ICANS, Immune effector cell-associated neurotoxicity syndrome; IEC-HS, immune effector cell associated HLH-like syndrome.

# SC291: GLEAM Phase 1 trial goals are to understand safety, dose, and early efficacy

- Key features of Phase 1 trial (GLEAM)
  - Patients with refractory systemic lupus erythematosus and ANCA-associated vasculitis
  - Dose escalation study
  - Potential to expand beyond these indications over time
- SC291 granted Fast Track designation in relapsed/refractory SLE
- Trial enrolled first patients in 2024

**Allogeneic HIP CAR T cell**



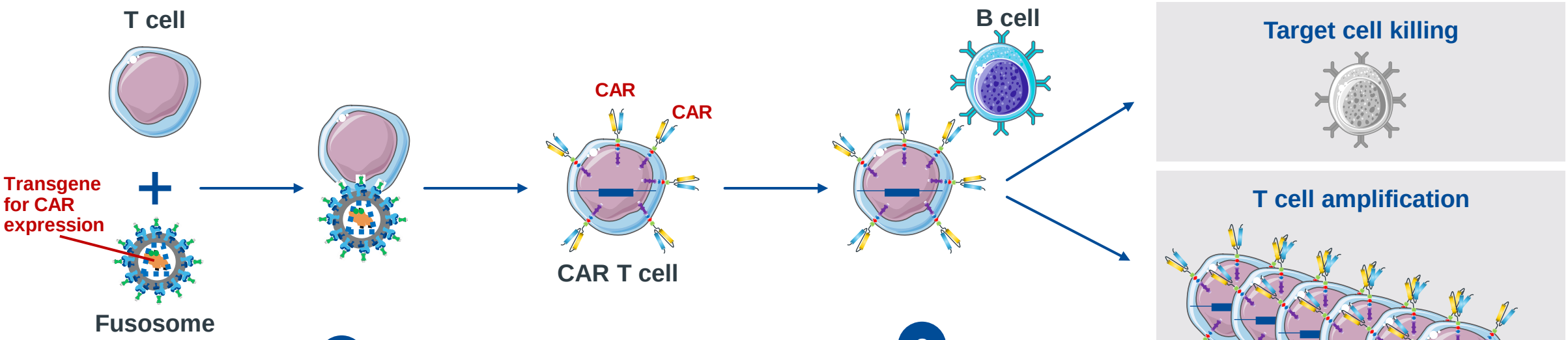

**An effective allogeneic CAR T offers potential to transform outcomes for patients**

# Sana is pursuing *in vivo* engineering of CAR T cells using a fusosome vector system

**Delivery**
Transduction of CD8 T cells

**Expression**
Transgene integration and CAR expression

**Function**
Targeted cell killing




**1**

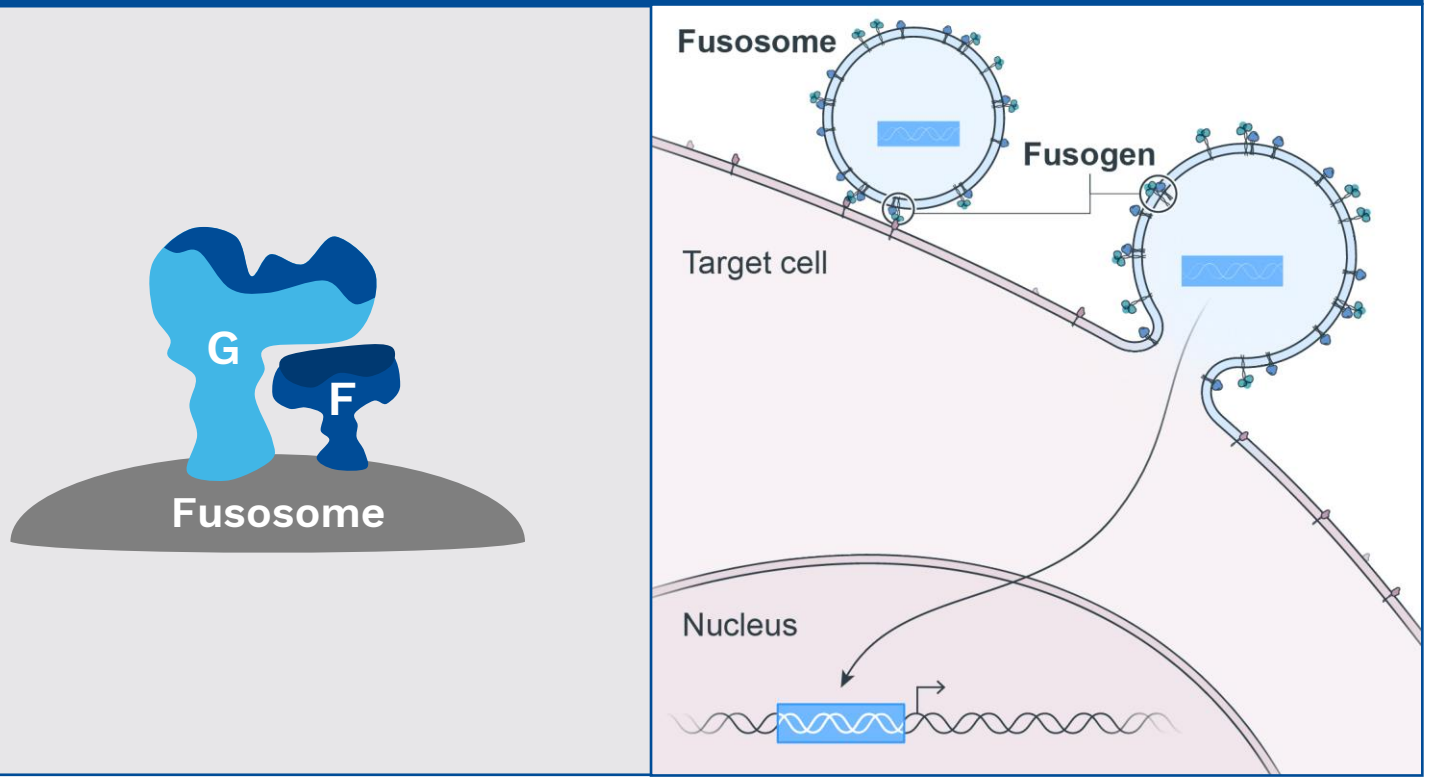
**Fusosome performance**
- Transduction specificity
- Transduction efficiency

**2**

**CAR activity**
- Expression/recognition
- Amplification
- Target cell killing

# Fusosome technology: Cell-specific *in vivo* delivery

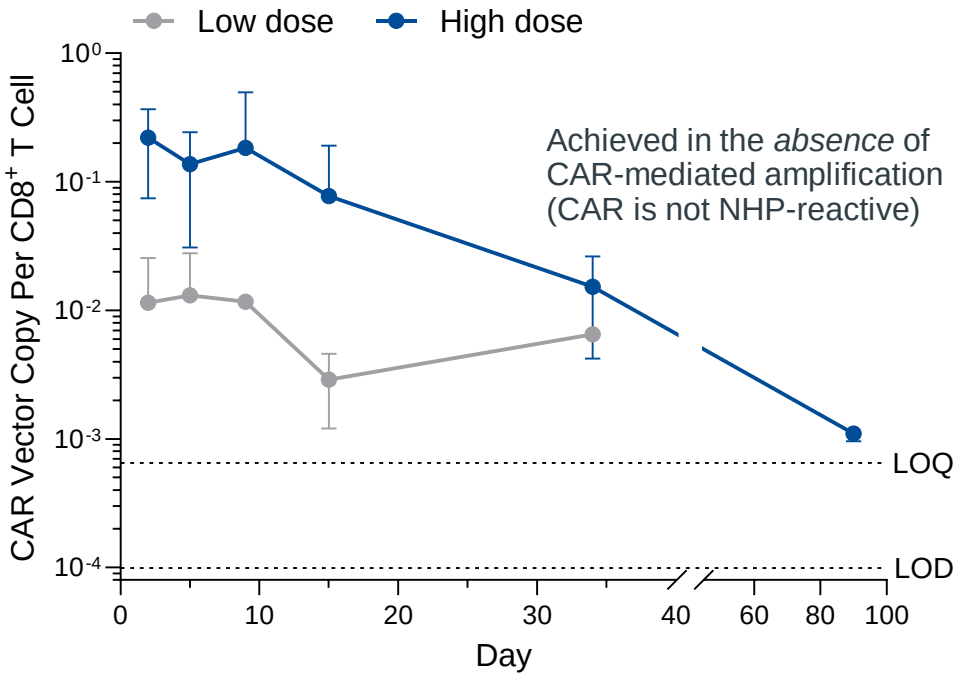
**Sana approach: Leverage insights from nature to deliver various payloads to specific cells without lymphodepletion**



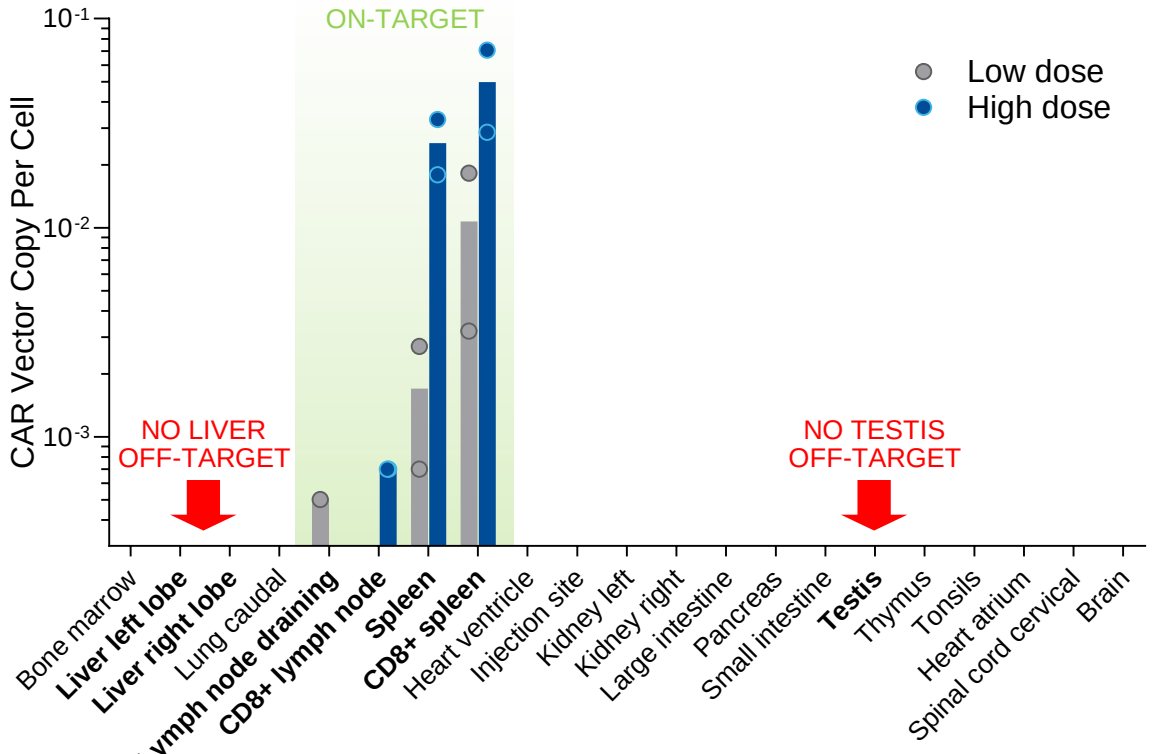
# Potent and cell-specific *in vivo* delivery demonstrated with SG299 in GLP tox study

4 NHPs at each dose received single SG299 injection

## Potent transduction of circulating CD8+ T cells




## No off-target transduction detected in hepatocytes or gonadal cells




Avg ± SEM plotted. Note that any values BLOQ are not plotted. 2 vehicle-control monkeys were BLOQ.
Abbreviations: BLOQ, below level of quantification; LOD, level of detection; LOQ, level of quantification.

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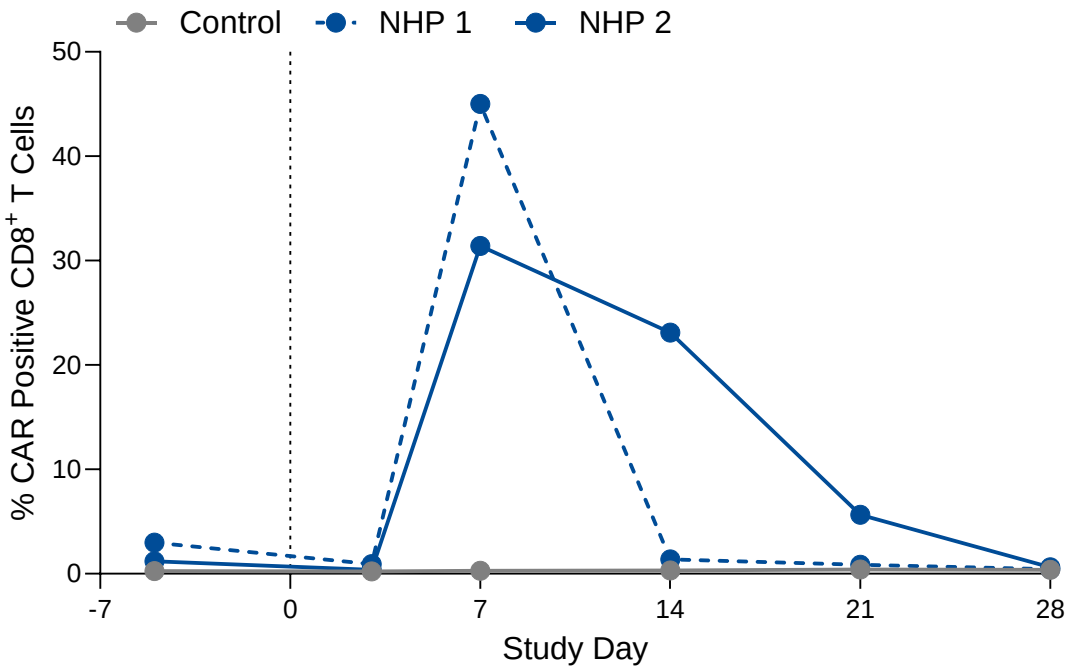
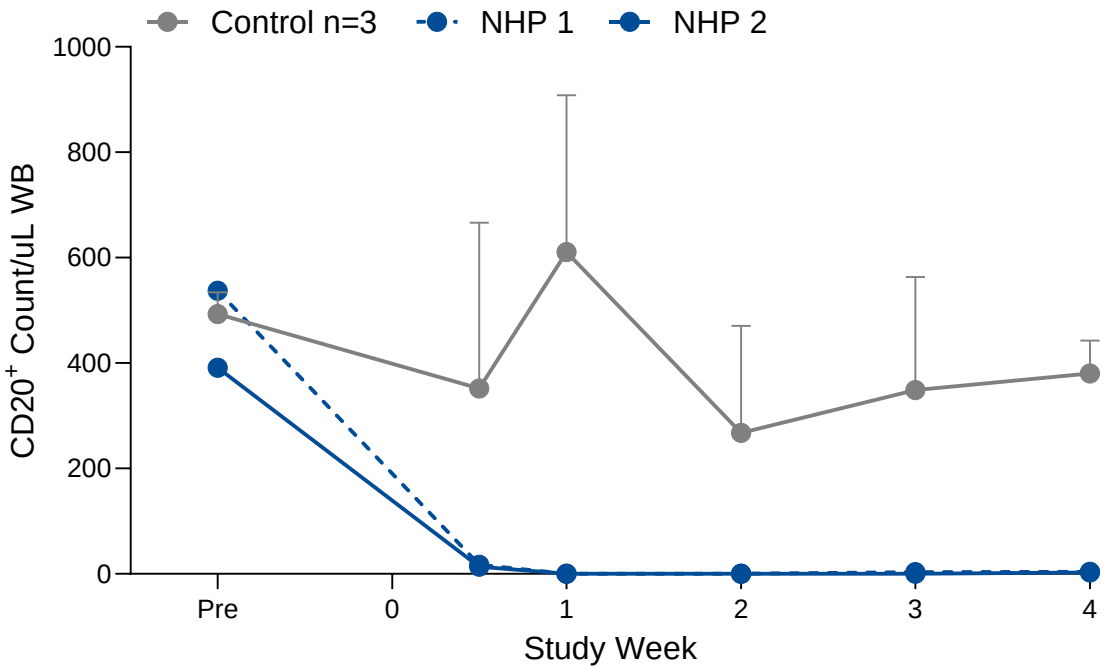
# Surrogate SG299 with additional component leads to T cell transduction, CAR expansion, and B-cell depletion

## Surrogate SG299 transduces T cells & expresses a CAR that recognizes NHP B cells

### CD8⁺ CAR⁺ cells expand in circulation



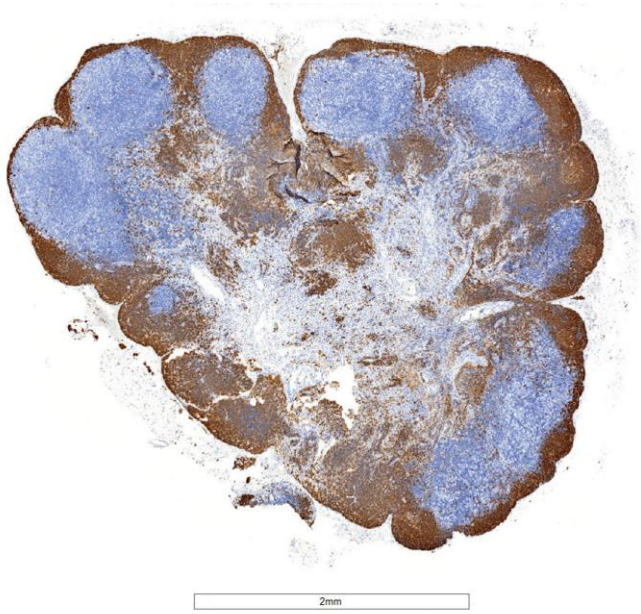

### Deep B-cell depletion achieved in peripheral blood



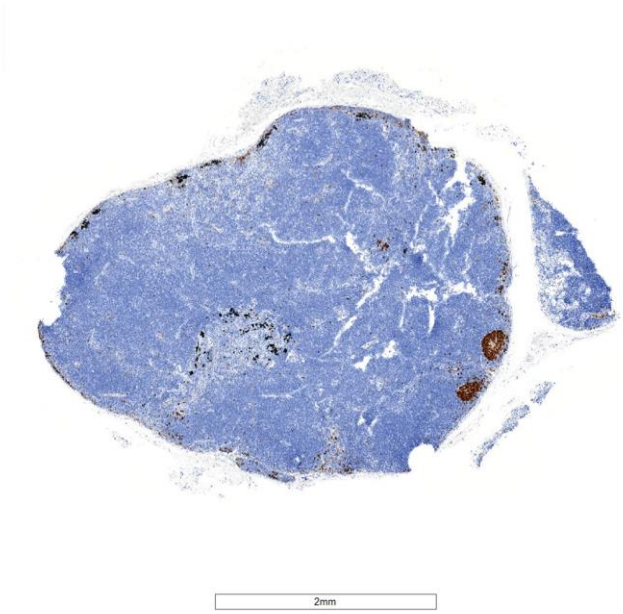
# Study shows B cell clearance in lymph nodes without lymphodepletion

**Anti CD20 staining of day 28 lymph node biopsy**

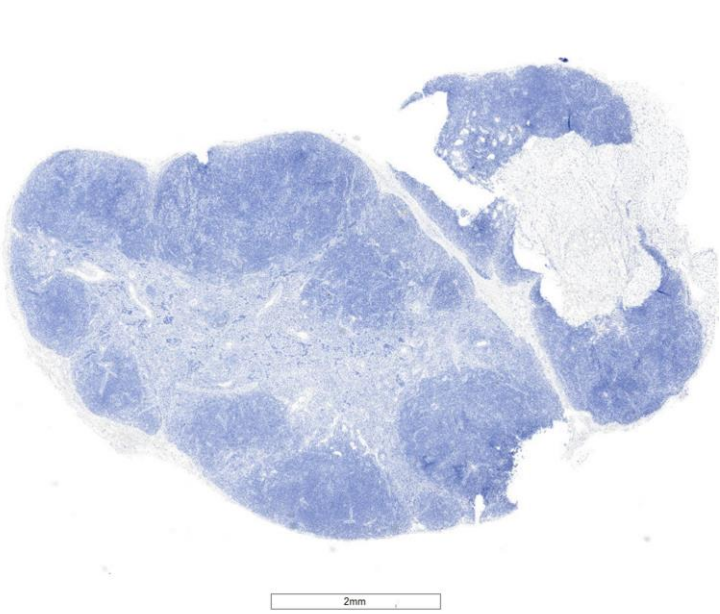
**Control**




**NHP 1**




**NHP 2**




Brown indicates CD20 IHC staining; black reflects tattoo ink.

# SC262: Targets growing population of patients with inadequate response to CD19 therapy

## CD19 CAR T relapsed patients represent large and growing unmet need[1]

Estimated ~12,000 B cell malignancy patients treated with CD19 CAR T in 2027[2]

- Potential of ~7,500 CAR T failures annually in 2027[2]
- Median survival of ~5 months post-CD19 CAR T therapy failure[3]

Estimated ~35-40% of CAR T patients with durable complete responses[4]

= 1,000 people

## CD22 CAR T is a promising approach to treat CD19 therapy failure[5]

- Autologous CD22 CAR T results in >50% CR rate in CD19 CAR failure DLBCL patients
  - High rates of non relapse mortality reported in long term follow up of autologous CD22 CAR T-treated patients
- High rate of CRs also seen in CD19 failure ALL patients[6]

[1]US, EU5, and Japan.[2]Clarivate DRG NHL Market Forecast Nov 2021; 2027 Forecast is 2L+ LBCL patients; internal analysis of secondary EPI data.
[3]Di Blasi et al. *Blood*.2022; DESCAR-T registry.[4]DiBlasi et al. *Blood.* 2022: [5]2024 ASH Kramer et al.;[5]2022 ASH Miklos/Stanford; [6]2018 *Nature Med* Fry, et al.

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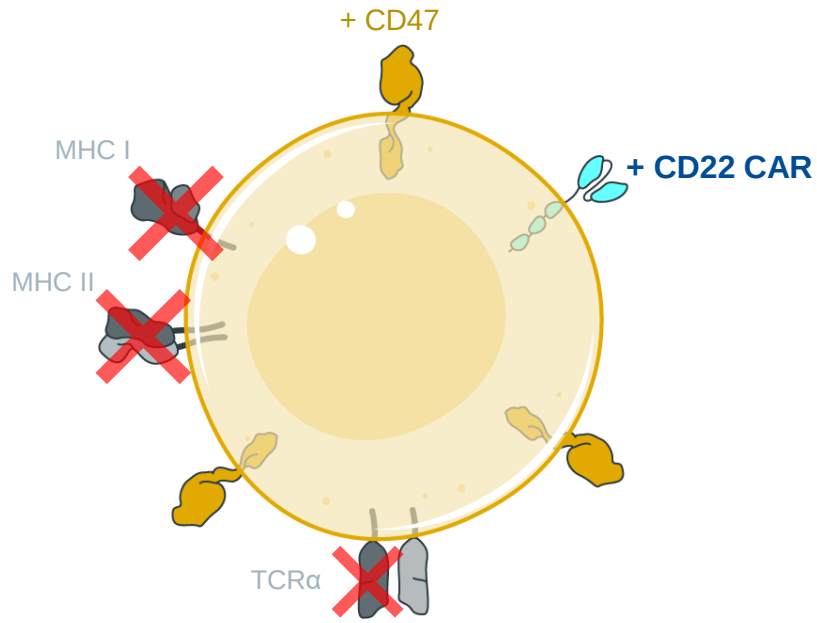
# SC262: VIVID Phase 1 trial goals are to understand safety, dose, and early efficacy

- Key features of Phase 1 trial (VIVID)
  - CD19 CAR T exposed patients with relapsed and/or refractory NHL
  - Starting dose of 90 million CAR T cells
- Expect to generate and share data
  - Safety and tolerability
  - Early response rates

**Allogeneic HIP CAR T cell**




**An effective allogeneic CAR T offers potential to transform outcomes for patients**

# We anticipate meaningful clinical data in multiple diseases in 2025 and beyond

- **HIP platform** shows ability to overcome allogeneic and autoimmune rejection **in people** across multiple cell types
- **Type 1 diabetes** – all components for a curative therapy are now in place:
  - Patients have remained off insulin for over a decade after islet transplant (but with immunosuppression to date)
  - Stem cell-derived islets offer a more scalable solution (but with immunosuppression to date)
  - Have now shown that we can eliminate immunosuppression with HIP modifications
- **SC451**, a HIP-modified stem cell-derived pancreatic islet therapy, contains all components and is advancing toward the clinic
- **SC291** leads to deep B-cell depletion and has significant potential in B-cell mediated autoimmune diseases. Ongoing GLEAM study
- **Fusogen platform** offers the potential to treat B-cell mediated autoimmune diseases and B-cell cancers with NO lymphodepletion
- **SC262**, a HIP-modified CD22 CAR T, has meaningful potential in treating CD19 CAR T relapsed patients. Ongoing VIVID study

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# Sana pipeline positioned to deliver meaningful clinical data

| PRODUCT CANDIDATE | MECHANISM | INDICATIONS | PRECLINICAL IND-ENABLING | PHASE 1 | PHASE 2/3 | SANA'S RIGHTS |
|---|---|---|---|---|---|---|
| **Type 1 Diabetes** | | | | | | |
| **UP421** | HIP primary islet cells[1] | T1D | | | | WW |
| **SC451** | Stem-cell derived pancreatic islet cells | T1D | | | | WW |
| **B-cell Mediated Autoimmune Diseases** | | | | | | |
| **SC291** | CD19-directed allo CAR T | SLE | | **GLEAM** | | WW |
| **SC291** | CD19-directed allo CAR T | AAV | | **GLEAM** | | WW |
| **SC291** | CD19-directed allo CAR T | Other indications | | | | WW |
| **SG299** | *In vivo* CD19-directed CAR T | Autoimmune disease | | | | WW |
| **Oncology** | | | | | | |
| **SC262** | CD22-directed allo CAR T | NHL (CD19 failures) | | **VIVID** | | WW |
| **SG299** | *In vivo* CD19-directed CAR T | Hematological malignancies | | | | WW |

[1]Investigator sponsored trial. Abbreviations: AAV, ANCA-associated vasculitis; SLE, systemic lupus erythematosus; NHL, non-Hodgkin lymphoma; SLE, systemic lupus erythematosus; T1D, type 1 diabetes; WW, worldwide.

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# Thank You

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