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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 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 but not limited to its Annual Report on Form 10-K dated March 24, 2021 and Quarterly Report on Form 10-Q dated November 8, 2021. Except as required by law, the Company undertakes no obligation to update publicly any forward-looking statements for any reason.
We believe the ability to modify the genome and use engineered cells as medicines will be one of the most (if not the most) important advances in healthcare over the next several decades.

Three aspirations drive us in our pursuit to deliver on the promise of cells as medicines:

- Repair and control the genes in any cell in the body
- Replace any cell in the body
- Broad access to our therapies

We continue to advance our technologies with multiple INDs planned as early as 2022.
Sana goal: fix cells in the body when possible or replace them when needed

<table>
<thead>
<tr>
<th><strong>in vivo Cell Engineering</strong></th>
<th><strong>ex vivo Cell Engineering</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Repair and control the genes of any cell in the body</td>
<td>Replace any cell in the body</td>
</tr>
<tr>
<td>• Deliver any payload (DNA, RNA, protein, organelle, integrating vs non-integrating)…</td>
<td>• Manufacture any cell at scale…</td>
</tr>
<tr>
<td>• To any cell (unlimited volume of distribution) in a…</td>
<td>• That engrafts (the right cell in the right environment)…</td>
</tr>
<tr>
<td>• Specific (e.g., just T cell),…</td>
<td>• Functions (understand exact phenotype desired)…</td>
</tr>
<tr>
<td>• And repeatable way (limit immunogenicity)</td>
<td>• And persists (overcome immune rejection and cellular signaling, such as apoptotic signaling)</td>
</tr>
</tbody>
</table>
## Assembling an experienced team across capabilities

<table>
<thead>
<tr>
<th>Delivery of Genetic Material</th>
<th>Modification of Genome</th>
<th>Immunology</th>
<th>Biology</th>
<th>Manufacturing</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Richard Mulligan, PhD</strong></td>
<td><strong>Ed Rebar, PhD</strong></td>
<td><strong>Sonja Schrepfer, MD, PhD</strong></td>
<td><strong>Sunil Agarwal, MD</strong></td>
<td><strong>Stacey Ma, PhD</strong></td>
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<tr>
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<td><strong>Christina Chaivorapol, PhD</strong></td>
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<td><strong>Craig Lichtenstein</strong></td>
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<table>
<thead>
<tr>
<th>PLATFORM</th>
<th>TECHNOLOGY</th>
<th>PROGRAMS (CELL TYPES)</th>
<th>THERAPEUTIC AREA</th>
<th>PRODUCT CANDIDATE</th>
<th>POTENTIAL INDICATIONS</th>
<th>POTENTIAL IND SUBMISSION</th>
</tr>
</thead>
<tbody>
<tr>
<td>in vivo cell</td>
<td>Fusogen</td>
<td>T cells</td>
<td>Oncology</td>
<td>SG295 (CD8/CD19)</td>
<td>NHL/ALL/CLL</td>
<td>As early as 2022</td>
</tr>
<tr>
<td>engineering</td>
<td></td>
<td></td>
<td></td>
<td>SG239 (CD8/BCMA)</td>
<td>Multiple myeloma</td>
<td>As early as 2024</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>SG242 (CD4/CD19)</td>
<td>NHL/ALL/CLL</td>
<td>As early as 2023</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>SG221 (CD4/BCMA)</td>
<td>Multiple myeloma</td>
<td>As early as 2024</td>
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<tr>
<td></td>
<td></td>
<td>Hepatocytes</td>
<td>Liver-related genetic disorders</td>
<td>SG328</td>
<td>OTC¹</td>
<td>As early as 2023</td>
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<tr>
<td></td>
<td></td>
<td>Hematopoietic stem cells</td>
<td>Hemoglobinopathies</td>
<td>SG418</td>
<td>Sickle cell disease</td>
<td>As early as 2023</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Beta-thalassemia</td>
<td>As early as 2023</td>
</tr>
<tr>
<td>ex vivo cell</td>
<td>Hypoimmune donor-derived</td>
<td>T cells</td>
<td>Oncology</td>
<td>SC291 (CD19)</td>
<td>NHL/ALL/CLL</td>
<td>As early as 2022</td>
</tr>
<tr>
<td>engineering</td>
<td></td>
<td></td>
<td></td>
<td>SC255 (BCMA)</td>
<td>Multiple myeloma</td>
<td>As early as 2023</td>
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<tr>
<td></td>
<td>Hypoimmune stem cell-derived</td>
<td>Beta cells</td>
<td>Diabetes</td>
<td>SC451</td>
<td>Type 1 diabetes</td>
<td>As early as 2023</td>
</tr>
<tr>
<td></td>
<td>Stem cell-derived</td>
<td>Glial progenitor cells</td>
<td>Central nervous system (CNS)</td>
<td>SC379</td>
<td>Huntington’s disease</td>
<td>As early as 2023</td>
</tr>
<tr>
<td></td>
<td>(to migrate to hypoimmune)</td>
<td>Cardiomyocytes</td>
<td>Cardiovascular</td>
<td>SC187</td>
<td>Pelizaeus-Merzbacher disease</td>
<td>As early as 2023</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Secondary progressive multiple sclerosis</td>
<td>As early as 2023</td>
</tr>
</tbody>
</table>

¹Ornithine transcarbamylase deficiency
Sana’s fusosome technology makes use of a viral fusogen to enable the targeting of specific cells and the delivery of different therapeutic payloads.
Development of cell-specific *in vivo* delivery platform

Fusogen Technology

Enveloped viruses incorporate viral and cellular proteins (**fusogens**) expressed on the infected cell membrane upon release from infected cells.

Fusogens on surface of the resulting virus particles (**fusosomes**) mediate virus entry via direct fusion of virus and cell membranes.
*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
Blood cancers remain high unmet need; despite success, current CAR T solutions have limitations

Current ex \textit{vivo} approaches have limitations

Fusogen platform offers potential to overcome these limitations

T cell fusosome carrying CAR construct infused into patient; the patient is the bioreactor that creates CAR T
Using a T cell-targeted fusosome to make CAR T cells \textit{in vivo}

**Specificity**  
CD4/CD8 T cell transduction

**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

T cell inside a patient

Fusosome

Cancer cell

CAR T cell

Target cell killing

T cell amplification
Targeting of different T cell types by viral fusosomes

Sana has generated fusosomes that specifically target and transduce CD8, CD4 and CD3 T cells

- **CD8-targeted** fusosome in vitro primary T cells
- **CD4-targeted** fusosome in vitro primary T cells
- **CD3-targeted** fusosome in vitro primary PBMCs
IV administration of CD19 CAR delivered by fusogen can clear B cell tumors in humanized mice comparably to ex vivo CD19 CAR T

CD19 CAR delivered by fusogen: in vivo

<table>
<thead>
<tr>
<th>CD19 CAR: ex vivo</th>
<th>CD19 CAR T</th>
</tr>
</thead>
<tbody>
<tr>
<td><img src="D3" alt="Saline" /></td>
<td><img src="D3" alt="Saline" /></td>
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<tr>
<td><img src="D7" alt="Saline" /></td>
<td><img src="D7" alt="Saline" /></td>
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<td><img src="D15" alt="Saline" /></td>
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<td><img src="D19" alt="Saline" /></td>
<td><img src="D19" alt="Saline" /></td>
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<td><img src="D27" alt="Saline" /></td>
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</table>

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<thead>
<tr>
<th><img src="D7" alt="Saline" /></th>
<th><img src="D7" alt="SG295 &amp; Activated PBMC" /></th>
<th><img src="D7" alt="SG295 &amp; Non-Activated PBMC" /></th>
</tr>
</thead>
<tbody>
<tr>
<td><img src="D10" alt="Saline" /></td>
<td><img src="D10" alt="SG295 &amp; Activated PBMC" /></td>
<td><img src="D10" alt="SG295 &amp; Non-Activated PBMC" /></td>
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<tr>
<td><img src="D17" alt="Saline" /></td>
<td><img src="D17" alt="SG295 &amp; Activated PBMC" /></td>
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<tr>
<td><img src="D24" alt="Saline" /></td>
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<td><img src="D24" alt="SG295 &amp; Non-Activated PBMC" /></td>
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<tr>
<td><img src="D31" alt="Saline" /></td>
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<td><img src="D31" alt="SG295 &amp; Non-Activated PBMC" /></td>
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</table>
CD8 fusogen delivering a CD20 CAR causes B cell depletion in NHPs

- Dosing well tolerated in all animals treated with CD20 CAR T delivered by CD8 fusogen, no infusion-related toxicity or evidence of CAR-associated toxicity

- Substantial B cell depletion observed in 4/6 treated animals
Potential first-in-class fusogen T cell programs – potential to target large markets with a single IV administration

Next Steps
SG295 and SG239
- IND-enabling studies and scale GMP manufacturing
- Finalize development plan – expect initial indications in NHL for CD19 and multiple myeloma for BCMA

Future Development
- Build CD8 and CD4 fusogen programs
- CD19 indications beyond NHL
- Targets beyond CD19 and BCMA
Protecting cells from immune destruction is key to unlocking potential of *ex vivo* cell engineering

**Fetomaternal tolerance during pregnancy**

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

**Sana approach:** creating hypoimmune cells from human iPSCs

How can we protect our engineered cells from getting attacked from the recipient’s immune system?
Sana is pursuing a broad *ex vivo* cell engineering strategy

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

- Differentiate pluripotent stem cells with hypoimmune edits
- Programs that benefit from, but do not require hypoimmune

**Images:**
- T cells
- Pancreatic islets
- Cardiomyocytes
- Glial progenitor cells
Hypoimmune cells evade rejection from the adaptive and innate immune system in a mouse

**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

**Evade the innate immune system**

- **NK Cell Killing**
  - No NK cell killing with HIP

<table>
<thead>
<tr>
<th></th>
<th>Wildtype Unmodified Cells</th>
<th>MHC Class I/II Disruption</th>
<th>MHC Class I/II Disruption &amp; CD47 Overexpression</th>
</tr>
</thead>
<tbody>
<tr>
<td>No killing of unmodified cells by NK cells</td>
<td>Killing of partially edited iPSC; HLA I/II knockout by NK cells</td>
<td>No killing of HIP cells by NK cells</td>
<td></td>
</tr>
</tbody>
</table>

Deuse T, …, Schrepler S. Nat Biotechnology. 2019; 37:252-258
Hypoimmune cells in NHP: no systemic adaptive immune activation after transplantation of hypoimmune iPSCs into naïve and sensitized NHPs

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

<table>
<thead>
<tr>
<th>Unmodified iPSC</th>
<th>Hypoimmune iPSC</th>
</tr>
</thead>
</table>

- HIP cells did not activate the systemic adaptive immune system (T cells and B cells)
- HIP cells evaded immune responses in a crossover experiment in NHP with pre-existing immunity
- Data suggest the potential to treat autoimmune disorders such as type 1 diabetes
Hypoimmune cells do not elicit an innate immune response in allogeneic NHP recipients

**Transplantation of NHP iPSCs into allogeneic NHPs (n=4/group)**

<table>
<thead>
<tr>
<th>Killing by macrophages</th>
<th>Killing by NK cells</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypoimmune cells do not activate the “missing self” response from the macrophages</td>
<td>Anti-CD47 safety switch</td>
</tr>
<tr>
<td>Hypoimmune cells do not activate the “missing self” response from the NK cells</td>
<td>Anti-CD47 safety switch</td>
</tr>
</tbody>
</table>

- HIP cells do not activate the "missing self" response from macrophages and NK cells
- "Safety switch": CD47 blockade results in killing by innate cells, providing a possible "safety switch"
Hypopimmune cells survive and proliferate in allogeneic sensitized NHPs without immunosuppression

- Unmodified cells were rejected within 3 weeks
- Hypopimmune cells survive in a crossover experiment in NHPs with pre-existing immunity
- Data suggest the potential to treat autoimmune disorders such as type 1 diabetes
Hypimmune cells survive *in vivo* in NHP while unmodified iPSCs get rejected

- Hypoimmune cells survive in allogeneic NHPs
- Unmodified cells get rejected while hypoimmune cells continue to survive
Human hypoimmune cells differentiate into various cell types

Hypoimmune edits (HLA-I knockout, HLA-II knockout, CD47tg) do not affect differentiation capacity nor intrinsic cell function.

Sana’s hypoimmune allo T: potential best-in-class opportunity

IMMUNE CHALLENGES | CURRENT ALLO T | SANA’S HYPOIMMUNE ALLO T
--- | --- | ---
GvHD |  |  
HvGD: Adaptive Immune System |  |  
HvGD: Innate Immune System |  |  

We believe we are better positioned to overcome immune challenges versus existing allo T therapies

Note:
GvHD: Graft versus Host Disease
HvGD: Host versus Graft Disease

Donor or iPSC T cells
Cell engineering
CD19 targeted allogeneic T cell
CD19 HIP CAR T cells clear tumor *in vivo*
CD19 HIP CAR T cells do not activate adaptive or innate immune responses

- **T cells are not activated by HIP CAR T cells**
- **HIP CAR T cells do not incite an antibody response**
- **HIP CAR T cells evade innate cell “missing self” response**

**T Cell Activation (ELISPOT)**

**IgM Binding (FACS)**

**NK Cell Killing**

**Macrophage Killing**
Allogeneic CAR T cells: potential best-in-class CAR T platform for off-the-shelf therapies

Next Steps for SC291
- Develop GMP gene editing and manufacturing processes
- Finalize development plan – expect initial indication in NHL

Future Development
- SC291 for other B cell malignancies
- SC255 for multiple myeloma
- Targets beyond CD19 and BCMA
Type 1 diabetes represents a large unmet need with a loss of approximately 15 years of life

- Autoimmune disease where destruction of insulin-producing beta cells results in inability to control glucose
- 1.6 million patients with type 1 diabetes in the US and 2.4 million in Europe; 51k new patients/year combined
- Approximately 15-year shorter life expectancy*
- Long term complications: end-organ damage, including heart attack, stroke, peripheral vascular disease, retinopathy, nephropathy
- Our goal is to produce hypoimmune beta cells that evade the immune system and normalize glucose

*Rawshani et al, Lancet 2018
SC451: combining HIP edits with leading beta cells protocol offers transformative potential for type 1 diabetes patients

Superior insulin secretion and faster kinetics in vitro

Robust rescue of type 1 diabetes mouse model
Beta cells: potential to transform global diabetes pandemic with curative treatment

Next Steps for SC451

- GMP hypoimmune iPSC cell line
- Develop scalable GMP manufacturing process
- IND-enabling studies
Almost all diseases result from damage to or dysfunction in a cell

**Sana aspiration: engineered cells as medicines**

**The challenge:**

Address obstacles to using engineered cells as medicines

**Sana: engineered cells to treat a broad set of diseases**

Validate platforms and create important medicines
- Fusosome for CD19 CAR T *in vivo*
- Fusosome for BMCA CAR T *in vivo*
- Hypoimmune allo CD19 CAR T
- Hypoimmune allo BCMA CAR T

Unlock the potential of engineered cells as medicines in multiple diseases
- Hypoimmune cells for:
  - Type 1 diabetes
  - Heart disease
  - CNS disorders
- Fusosomes delivering payloads for other diseases
Thank You