UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
Washington, D.C. 20549

FORM 8-K

CURRENT REPORT
Pursuant to Section 13 or 15(d)
of the Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): February 7, 2024

SANA BIOTECHNOLOGY, INC.
(Exact name of registrant as specified in its charter)

Delaware	**001-39941**	**83-1381173**
(State or other jurisdiction of incorporation)	**(Commission File Number)**	**(IRS Employer Identification Number)**

188 East Blaine Street, Suite 400 **Seattle, Washington**	**98102**
(Address of principal executive offices)	**(Zip Code)**

Registrant's telephone number, including area code: (206) 701-7914

N/A
(Former name or former address, if changed since last report.)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	Trading Symbol(s)	Name of each exchange on which registered
Common Stock, $0.0001 par value per share	SANA	The Nasdaq Global Select Market

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§240.12b-2 of this chapter).

Emerging growth company ☒

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 2.02 Results of Operations and Financial Condition.

The information set forth in Item 8.01 under the caption "Preliminary Financial Information (Unaudited)" is incorporated into this Item 2.02 by reference.

Item 8.01 Other Events.

Preliminary Financial Information (Unaudited)

Sana Biotechnology, Inc. (the "Company," "we," "us," or "our") reports that it estimates its cash, cash equivalents and marketable securities as of January 2, 2024 were approximately $205.4 million. This estimate of the Company's cash, cash equivalents and marketable securities as of January 2, 2024 is preliminary, has not been audited, is based on information available to the Company as of the filing of this Current Report on Form 8-K (this "Report"), and is subject to change. Additional information and disclosure would be required for a more complete understanding of the Company's financial position as of January 2, 2024. The Company's independent registered public accounting firm has not audited, reviewed or performed any procedures with respect to this preliminary information and, accordingly, does not express an opinion or any other form of assurance about it.

Corporate Update

The Company is providing a corporate update, attached as Exhibit 99.1 to this Report and incorporated by reference herein.

Cautionary Note Regarding Forward-Looking Statements

This Report, including Exhibit 99.1 attached hereto, contains forward-looking statements, including statements about the Company's preliminary estimates of cash, cash equivalents and marketable securities as of January 2, 2024, the initial interim clinical data from the ARDENT trial and the Company's belief that it supports further dose escalation and expansion within the ARDENT trial and broader application of this technology in allogeneic cell therapies in other indications, our beliefs relating to our observations of data from our clinical trials and recently conducted studies as well as expectations regarding additional clinical data to be generated and reported by the Company. These forward-looking statements reflect the Company's views regarding current expectations and projections about future events and conditions and are based on currently available information. These forward-looking statements are not guarantees of future performance and are subject to risks, uncertainties and assumptions that are difficult to predict, including risks related to preliminary financial results, such as the risks that the preliminary financial information reflects information available to the Company only at this time and may differ from actual results, risks and uncertainties associated with discovering, developing and commercializing drugs that are safe and effective for use as human therapeutics and operating as a clinical stage company, including the potential for the Company's product candidates to cause serious adverse events, the timing, progress and results of the Company's clinical trials, the potential for any clinical trial results to differ from preclinical, interim, preliminary, topline or expected results, regulatory risks, and the Risk Factors identified in the Company's filings with the Securities and Exchange Commission (the "SEC"), including the Company's Annual Report on 10-K for the year ended December 31, 2022 and its Quarterly Report on Form 10-Q for the quarter ended September 30, 2023; therefore, the Company's actual results could differ materially from those expressed, implied or forecast in any such forward-looking statements. Expressions of future goals and expectations and similar expressions, including "may," "will," "should," "could," "aims," "seeks," "expects," "plans," "anticipates," "intends," "believes," "estimates," "predicts," "potential," "targets," and "continue," reflecting something other than historical fact are intended to identify forward-looking statements. Unless required by law, the Company undertakes no obligation to update publicly any forward-looking statements, whether as a result of new information, future events, or otherwise. However, readers should carefully review the reports and documents the Company files or furnishes from time to time with the SEC, particularly its Annual Reports on Form 10-K, Quarterly Reports on Form 10-Q, and Current Reports on Form 8-K.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits

See the Exhibit Index below, which is incorporated by reference herein.

EXHIBIT INDEX

Exhibit Number	Description
99.1	Corporate Update
104	Cover Page Interactive Data File (embedded within the Inline XBRL document)

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

Sana Biotechnology, Inc.

Date: February 7, 2024

By: **/s/ Bernard J. Cassidy**

Bernard J. Cassidy

Executive Vice President and General Counsel

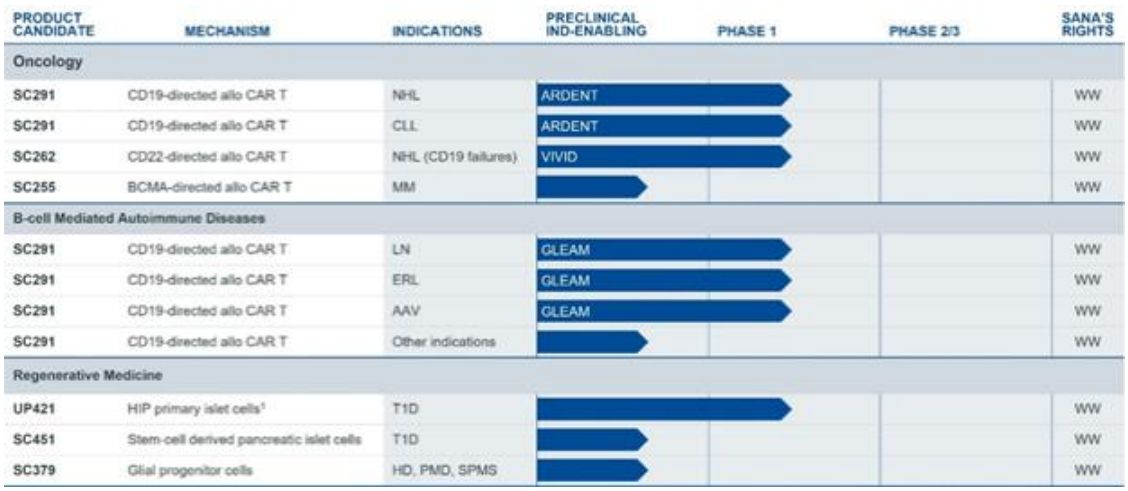
Overview

We were founded on the belief that engineered cells will be one of the most important transformations in medicine over the next several decades. The burden of diseases that can be addressed at their root cause through engineered cells is significant. We view engineered cells as having the potential to be as therapeutically disruptive as biologics to clinical practice. Our long-term aspirations are to be able to control or modify any gene in the body, to replace any cell that is damaged or missing, and to markedly improve access to cellular and gene- based medicines. We have brought together an experienced group of scientists, engineers, and company builders and combined them with the necessary technologies to move this vision forward. We are developing ex vivo and in vivo cell engineered cell therapies to revolutionize treatment across a broad array of therapeutic areas with unmet treatment needs, including in oncology, diabetes, autoimmune diseases, and central nervous system disorders, among others. Our platform progress, broad capabilities, and strong balance sheet enable us to execute on a broad vision.

Frequently in disease, cells are damaged or missing entirely, and an effective therapy needs to replace the entire cell, an approach referred to as cell therapy or ex vivo cell engineering. A successful therapeutic requires an ability to manufacture cells at scale that engraft, function, and have the necessary persistence in the body. Of these requirements, long-term persistence related to overcoming immunologic rejection of another person's cells has been the most challenging, which has led many to focus on autologous, or a patient's own, cells as the therapeutic source. However, autologous therapies require a complex process of harvesting cells from the patients, manipulating them outside the body, and returning them to the patient. Products using this approach have had to manage significant challenges such as scalability, product variability, product quality, cost, patient accessibility, and limits on number of cell types that are amenable to this approach. Given these limitations, rather than using autologous cells to overcome immune rejection, we have invested in creating hypoimmune- modified cells that can "hide" from the patient's immune system. We are striving to make therapies that use pluripotent stem cells with our hypoimmune genetic modifications as the starting material, which we then differentiate into a specific cell type, such as a pancreatic islet cell, before treating the patient. Additionally, there are cell types for which effective differentiation protocols from a stem cell have not yet been developed, such as T cells. For these cell types, instead of starting from a pluripotent stem cell, we can use allogeneic, fully-differentiated cells sourced from a donor as the starting material to which we then apply our hypoimmune genetic modifications.

The process of repairing and controlling genes in the body, referred to as gene therapy or in vivo cell engineering, requires in vivo delivery of a therapeutic payload and modification of the genome. There are multiple methods available to modify the genome, but limited ability to deliver therapeutic payloads in vivo. Thus, delivery of a therapeutic payload is at the core of our strategic focus for our in vivo gene therapy program, with our ultimate goal being the delivery of any payload to any cell in a specific and repeatable way. Our initial effort is on cell-specific delivery and increasing the diversity and size of payloads. Using our fusogen technology, we have shown in preclinical studies that we can specifically target numerous cell surface receptors that, when combined with delivery vehicles to form fusosomes, allow cell-specific delivery across multiple different cell types.

We believe the time is right to develop engineered cell therapies across a broad range of therapeutic areas. Substantial progress in the understanding of genetics, gene editing, protein engineering, stem cell biology, immunology, process analytics, and computational biology have converged to create an opportunity to markedly increase the breadth and depth of the potential impact of cellular medicines. We are focused on creating transformative engineered cell therapies across a range of therapeutic areas. We are developing a broad pipeline of product candidates, which are summarized below:



[1] Investigator sponsored trial.

Abbreviations: ANCA-associated vasculitis (AAV); chronic lymphocytic leukemia (CLL); extrarenal systemic lupus erythematosus (ERL); Huntington's disease (HD); lupus nephritis (LN); multiple myeloma (MM); non-Hodgkin lymphoma (NHL); Pelizaeus-Merzbacher Disease (PMD); secondary progressive multiple sclerosis (SPMS); type 1 diabetes (T1D); worldwide (WW).

Clinical Updates

SC291 – ARDENT

In January 2024, we disclosed initial interim clinical data from our ongoing Phase 1 clinical trial evaluating our CD19-targeted directed allogeneic chimeric antigen receptor (CAR) T program, SC291, in patients with B-cell mediated autoimmune diseases, including non-Hodgkin's lymphoma (NHL) and chronic lymphoblastic leukemia (CLL), which we refer to as the ARDENT trial.

As of January 5, 2024, the cut-off date for our early interim analysis, six patients had been dosed with SC291 and four patients were evaluable (defined as patients dosed with SC291 who had at least one disease assessment), of whom three were dosed with 60M CAR T cells (Dose Level 1) and the other was dosed with 120M CAR T cells (Dose Level 2). With respect to the four evaluable patients at these two dose levels, we observed no dose limiting toxicities, no SC291-related serious adverse events, and no incidences of graft versus host disease. We also observed no cytokine release syndrome (CRS) or immune effector cell-associated neurotoxicity syndrome (ICANS) of any grade or any infections of Grade 3 or higher. Additionally, we observed at least a partial response in three of the patients, including ongoing complete responses in one patient from Dose Level 1 after three months and the patient from Dose Level 2 after two months.

The SC291 drug product contains CAR T cells that are fully edited hypoimmune cells, which we describe as HIP-edited CAR-T cells, along with partially edited cells, which we describe as non-HIP CAR T cells. *In vitro* testing showed evidence that blood and immune cells from each of the four evaluable patients had mounted an immune response to the non-HIP CAR T cells but not to the HIP-edited CAR T cells. Specifically, HIP-edited CAR T cells from the drug product were not rejected by the innate immune response mediated by the patient's natural killer (NK) cells, nor did the patients have T cell or antibody responses that recognized these cells. In contrast, we observed immune responses against the non-HIP CAR T cells in the drug product.

Importantly, this evidence suggests that the patients had an intact immune system capable of recognizing allogeneic cells and that the HIP CAR T cells were able to evade these responses. These results were consistent across all four evaluable patients and provide early support for the idea that the immune evasion profile of our HIP gene edits in multiple pre-clinical models may translate into human subjects. We believe this observation supports further dose escalation and dose expansion in the ARDENT trial and broader application of our HIP technology in allogeneic cell therapies in other indications.

We are continuing to enroll and dose patients in the ARDENT trial and expect to share additional data in 2024.

SC291 – GLEAM

In November 2023, the FDA cleared our Investigational New Drug application (IND) to evaluate SC291 in patients with lupus nephritis, extrarenal lupus, and antineutrophil cytoplasmic antibody-associated vasculitis, which we refer to as our GLEAM trial.

In the ongoing ARDENT trial, we have observed the pharmacodynamic effect of peripheral blood B cell depletion, which refers to diminishing B cell counts in the peripheral blood, associated with SC291 treatment in patients. We believe this may increase the possibility that SC291 treatment may confer similar benefit to patients with B cell-mediated autoimmune disorders as autologous CD19 CAR T cell therapies.

We expect to share data from the GLEAM trial in 2024.

SC262 – VIVID

In January 2024, the FDA cleared our IND to evaluate SC262, our hypoimmune-modified CD22 CAR T program, in patients with relapsed or refractory B-cell malignancies who have received prior CD19 CAR T therapy, which we refer to as our VIVID trial. We expect to share data from the VIVID trial in 2024.

Pancreatic Islet Cell Program

In January 2024, we presented data from a study transplanting allogeneic HIP-modified pancreatic islet cells into a fully immunocompetent, diabetic non-human primate (NHP). Subsequent to diabetes being induced in the NHP with streptozotocin, daily insulin injections were performed to re-establish glucose control. After 78 days, the NHP underwent transplantation of HIP primary islets by intramuscular injection, resulting in insulin independence without the use of any immunosuppression. As early as one week after the transplantation, the NHP's serum c-peptide level had normalized, and it remained stable throughout the follow-up period of six months. The NHP showed tightly controlled blood glucose levels for six months, was completely insulin-independent, and was continuously healthy throughout this period with no use of any immunosuppression. Up to six months following HIP primary islet transplantation, peripheral blood mononuclear cells and serum were obtained from the NHP for immune analyses. HIP primary islets showed no T cell recognition, no graft-specific antibodies, and were protected from NK cell and macrophage killing. To demonstrate that the NHP's insulin-independence was fully dependent on the HIP primary islets and that there was no regeneration of the animal's endogenous islet cell population, we triggered the destruction of the HIP primary islets using a CD47-targeting antibody. This resulted in a loss of glycemic control and return to exogenous insulin dependence. We believe these data demonstrate potential evidence for immune evasion of HIP primary islets, graft-mediated insulin-independence of the diabetic NHP, and a potential safety strategy.

In November 2023, the Swedish Medical Products Agency authorized a clinical trial application for an investigator-sponsored, first-in-human study (IST) evaluating UP421, an allogeneic, primary islet cell therapy engineered with our HIP technology, in patients with type 1 diabetes mellitus. Subjects in this study will receive no immunosuppression. We expect data from the IST to be shared in 2024. We believe that immunology insights gained from the IST, particularly with respect to whether HIP modifications lead to long-term survival and evasion of either allogeneic or autoimmune killing of the transplanted cells, may provide direct insights applicable to our preclinical-stage pluripotent stem cell-derived hypoimmune pancreatic islet product candidate, SC451.