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): April 24, 2018

SANGAMO THERAPEUTICS, INC.
(Exact name of registrant as specified in its charter)

Delaware	**000-30171**	**68-0359556**
(State or other jurisdiction of incorporation)	**(Commission File Number)**	**(IRS Employer Identification No.)**

501 Canal Blvd., Richmond, California 94804
(Address of principal executive offices) (Zip Code)

(510) 970-6000
(Registrant's telephone number, including area code)

Not Applicable
(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 (see General Instruction A.2. below):

☐ 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))

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. ☐

In this report, “Sangamo,” “the Company,” “we,” “us” and “our” refer to Sangamo Therapeutics, Inc., a Delaware corporation, and its subsidiaries on a consolidated basis.

Item 2.02 Results of Operations and Financial Condition.

On April 24, 2018, Sangamo filed with the U.S. Securities and Exchange Commission (the “SEC”) a preliminary prospectus supplement (the “Preliminary Prospectus”) pursuant to Rule 424(b) under the Securities Act of 1933, as amended, relating to its Registration Statement on Form S-3 (File No. 333-224418) filed with the SEC on April 24, 2018, in connection with a proposed public offering. In the Preliminary Prospectus, Sangamo disclosed that, as of March 31, 2018, it had approximately $234.9 million of cash, cash equivalents and investments.

Forward-Looking Statements

Item 2.02 of this report contains forward-looking statements, including, without limitation, statements relating to Sangamo’s preliminary unaudited cash and marketable securities position as of March 31, 2018. These forward-looking statements are based upon Sangamo’s current expectations. Actual results could differ materially from these forward-looking statements as a result of certain factors, including, without limitation, risks related to preliminary financial results, including the risks that the preliminary financial results reported herein reflect information available to Sangamo only at this time and may differ from actual results, including in connection with Sangamo’s completion of financial closing procedures. You are cautioned not to place undue reliance on these forward-looking statements, which speak only as of the date of this report. Sangamo undertakes no duty to update such information except as required under applicable law.

The information contained in Item 2.02 of this report shall not be deemed “filed” for purposes of Section 18 of the Securities Exchange Act of 1934, as amended, or otherwise subject to the liabilities of that Section or Sections 11 and 12(a)(2) of the Securities Act of 1933, as amended. The information contained in Item 2.02 of this report shall not be incorporated by reference into any filing with the SEC made by Sangamo whether made before or after the date hereof, regardless of any general incorporation language in such filing.

Item 8.01 Other Events.

Sangamo is filing information for the purpose of supplementing and updating certain aspects of the description of its business from that described under the heading, “Item 1. Business” in Sangamo’s Annual Report on Form 10-K for the year ended December 31, 2017, filed with the SEC on March 1, 2018. The updated disclosure is filed herewith as Exhibit 99.1 and is incorporated herein by reference.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits

Exhibit Number	Description
99.1	Updated Company Disclosure

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.

DATE: April 24, 2018

SANGAMO THERAPEUTICS, INC.

By: /s/ HEATHER TURNER
Heather Turner
Senior Vice President and General Counsel

Throughout this Exhibit 99.1, unless the context indicates otherwise, the terms "Sangamo," "we," "us" and "our" refer to Sangamo Therapeutics, Inc., a Delaware corporation, and its subsidiaries on a consolidated basis. SANGAMO®, SANGAMO THERAPEUTICS®, Better Therapeutics By Design®, ZFP Therapeutic®, Engineering Genetic Cures®, and Pioneering Genetic Cures® are our registered trademarks in the United States. All other trademarks or trade names referred to in this Current Report on Form 8-K are the property of their respective owners.

COMPANY OVERVIEW

Our Business

We are a clinical stage biotechnology company focused on translating ground-breaking science into genomic therapies that transform patients' lives using our industry-leading platform technologies in genome editing, gene therapy, gene regulation and cell therapy.

We are a leader in the research and development of zinc finger proteins, or ZFPs, a naturally occurring class of proteins found in humans. We have used our knowledge and expertise to develop a proprietary technology platform in both genome editing and gene regulation. ZFPs can be engineered to make zinc finger nucleases, or ZFNs, proteins that can be used to specifically modify DNA sequences by adding or knocking out specific genes, or genome editing, and ZFP transcription factors or ZFP TFs, proteins that can be used to increase or decrease gene expression, or gene regulation. In the process of developing this platform, we have accrued significant scientific, manufacturing and regulatory capabilities and know-how that are generally applicable in the broader field of gene therapy and have capitalized this knowledge into a conventional gene therapy platform based on adeno-associated viral vector, or AAV, cDNA gene transfer.

Our strategy is to maximize the value and therapeutic use of our technology platforms. In certain therapeutic areas we intend to capture the value of our proprietary genome editing and gene therapy products by forward integrating into manufacturing, development and commercial operations. In other therapeutic areas we intend to partner with biopharmaceutical companies to develop products.

We are focused on the development of human therapeutics for diverse diseases with well-characterized genetic causes. We have several proprietary clinical and preclinical product candidates in development and have strategically partnered certain programs with biopharmaceutical companies to obtain funding for our own programs and to expedite clinical and commercial development.

We have an ongoing Phase 1/2 clinical trial evaluating SB-525, a gene therapy for the treatment of hemophilia A, a bleeding disorder. We also have ongoing Phase 1/2 clinical trials evaluating three product candidates using our proprietary *in vivo* genome editing approach: SB-FIX for the treatment of hemophilia B, a bleeding disorder; SB-318, for the treatment of Mucopolysaccharidosis Type I, or MPS I; and SB-913 for the treatment of Mucopolysaccharidosis Type II, or MPS II. MPS I and MPS II are rare lysosomal storage disorders, or LSDs. We are also initiating a Phase 1/2 clinical trial evaluating ST-400, developed using our proprietary ZFN-mediated *ex vivo* cell therapy platform, for the treatment of beta-thalassemia, a blood disorder. In addition, we have proprietary preclinical and discovery stage programs in other LSDs, hematological disorders and monogenic diseases, including certain central nervous system, or CNS, disorders, cancer immunotherapy, immunology and infectious disease.

In February 2018, we entered into a global collaboration and license agreement with Kite Pharma, Inc., or Kite, a wholly owned subsidiary of Gilead Sciences, Inc., or Gilead, for the research, development and commercialization of potential engineered cell therapies for cancer. In this collaboration, we will work together with Kite on a research program under which we will design ZFNs and AAVs to disrupt and insert certain genes in T cells and natural killer, or NK, cells, including the insertion of genes that encode chimeric antigen receptors, or CARs, T-cell receptors, or TCRs and NK-cell receptors, or NKRs, directed to mutually agreed targets. Kite will be responsible for all clinical development and commercialization of any resulting products.

In December 2017, we entered into a new research collaboration and license agreement with Pfizer Inc., or Pfizer, for the development and commercialization of potential gene therapy products that use ZFP TFs to treat amyotrophic lateral sclerosis, or ALS, and frontotemporal lobar degeneration, or FTLD, linked to mutations of the *C9ORF72* gene. Under this agreement, we are working with Pfizer on a research program to identify, characterize and preclinically develop ZFP TFs that satisfy pre-agreed criteria. Pfizer is responsible for subsequent development, manufacturing and commercialization of licensed products.

In May 2017, we entered into a global collaboration and license agreement with Pfizer for the research, development and commercialization of SB-525, our gene therapy product candidate for hemophilia A, and closely related products. Under this agreement, we are responsible for conducting the Phase 1/2 clinical trial and certain manufacturing activities for SB-525, while Pfizer is responsible for subsequent worldwide development, manufacturing, marketing and commercialization of SB-525. We and Pfizer may also collaborate in the research and development of additional AAV-based gene therapy products for hemophilia A.

We have also established a collaborative partnership with Bioverativ, Inc., or Bioverativ, a wholly owned subsidiary of Sanofi, to research, develop and commercialize therapeutic gene-edited cell therapy products in hemoglobinopathies, including beta-thalassemia and sickle cell disease, or SCD. We expect to begin enrolling patients in a Phase 1/2 clinical study for beta-thalassemia in the first half of 2018. Bioverativ is responsible for subsequent development, manufacturing and commercialization of licensed products.

We have a substantial intellectual property position in the genome editing field including the design, selection, composition and use of engineered ZFPs to support our research and development activities. As of February 15, 2018, we either owned outright or have exclusively licensed the commercial rights to over 860 patents issued in the United States and foreign jurisdictions, and over 610 patent applications pending worldwide. We continue to license and file new patent applications that strengthen our core and accessory patent portfolio. We believe that our intellectual property position is a critical element in our ability to research, develop and commercialize products and services based on genome editing, gene therapy, gene regulation and cell therapy.

Our Product Development

Hemophilia A and B

Hemophilia is a rare bleeding disorder in which the blood does not clot normally. It is also a monogenic disease, or a disease that is caused by a genetic defect in a single gene. There are several types of hemophilia caused by mutations in genes that encode factors which help the blood clot and stop bleeding when blood vessels are injured. Individuals with hemophilia experience bleeding episodes after injuries and spontaneous bleeding episodes that often lead to joint disease such as arthritis. The most severe forms of hemophilia affect males. The standard treatment for individuals with hemophilia is replacement of the defective clotting factor with regular infusion of recombinant clotting factors or plasma concentrates. These therapies are expensive and sometimes stimulate the body to produce antibodies against the factors that inhibit the benefits of treatment. In these situations, other clotting factors such as Factor VII and X may be used to treat patients.

The most prevalent form of the disease, hemophilia A, is caused by a defect in the clotting Factor 8 gene. According to the National Hemophilia Foundation and the World Federation of Hemophilia, hemophilia A occurs in about one in every 5,000 male births in the United States, with approximately 16,000 males currently affected. Defects in clotting Factor 9 gene lead to hemophilia B. Hemophilia B occurs in about one in every 25,000 male births in the United States, with approximately 4,000 males currently affected.

SB-525 — Hemophilia A

We are developing SB-525, a gene therapy product candidate utilizing an AAV carrying a clotting Factor 8 gene construct that is driven by our proprietary synthetic liver specific promoter. In 2016, we presented preclinical data demonstrating production of supraphysiological levels of human Factor VIII clotting protein, or hFVIII, in mice and non-human primates, or NHPs. In these dose-ranging preclinical studies, mean hFVIII levels of 5 — 230% of normal were observed using AAV doses in the range of 6.00E+11 — 6.00E+12 vg/kg, the most potent dose response reported in NHPs for a human Factor 8 gene construct at the time.

In 2017, we initiated a Phase 1/2 clinical trial, the Alta Study, to evaluate the safety and efficacy of SB-525 in adults with severe hemophilia A. The Alta Study is an open-label, ascending-dose study designed to enroll up

to 20 adult subjects across six potential dose cohorts. In August 2017, we announced that the first subject was treated in our Alta Study. Currently, there are four patients dosed with SB-525. We expect to release preliminary data from the Alta Study in the third quarter of 2018.

SB-525 has been granted Orphan Drug and Fast Track designations by the U.S. Food and Drug Administration, or FDA, as well as Orphan Medicinal Product designation by the European Medicines Agency, or EMA.

SB-FIX — Hemophilia B

We are developing SB-FIX, an *in vivo* genome editing product candidate, to treat hemophilia B. Utilizing our ZFN genome editing technology, we are adding a new therapeutic copy of the Factor 9 gene precisely into the Albumin gene locus in liver cells, and using the strong endogenous Albumin promoter to drive expression of the newly inserted gene. We believe the potential of this approach to provide a permanent correction for a patient may be optimal for a pediatric population by reducing or eliminating the need for chronic infusions of replacement proteins or clotting factor products. We have published data demonstrating the potential utility of this approach for several different monogenic disease applications in addition to hemophilia B.

Preclinical studies of the Albumin genome editing approach have demonstrated that therapeutic levels of Factor IX clotting protein could be generated in a dose-dependent manner in NHPs. There were no significant alterations in circulating Albumin levels. Studies in mice also demonstrated stable Factor IX production for over one year. Preclinical studies in wildtype mice have demonstrated expression of therapeutic levels of human clotting Factor IX protein, or hFIX, from the liver and into the blood for the duration of the 60-week study. Additional preclinical studies in mouse models of hemophilia B demonstrated expression of therapeutic levels of hFIX from the liver and into the blood, which resulted in the correction of the clotting defect in hemophilia B mice treated with a single dose of SB-FIX. SB-FIX was also evaluated in preclinical NHP studies and demonstrated dose-dependent, therapeutic levels of hFIX expression, between 20-50% of normal, in wildtype cynomolgus monkeys, after a single administration of SB-FIX. Levels of hFIX were stable for up to three months in treated NHPs. Furthermore, there was a strong dose-response correlation between the level of gene modification at the Albumin locus and the levels of hFIX measured in the blood.

In 2016, we initiated a Phase 1/2, open-label, ascending dose clinical trial, the FIXtendz Study, to evaluate safety and efficacy of SB-FIX in adult males with severe hemophilia B. The FIXtendz Study is designed to enroll up to 12 subjects across three dose cohorts. In February 2018, the Medicines and Healthcare Products Regulatory Agency, or MHRA, of the United Kingdom granted Clinical Trial Authorisation, or CTA, for enrollment of subjects into the ongoing Phase 1/2 clinical trial evaluating SB-FIX for hemophilia B. The CTA permits evaluation of SB-FIX in both adults and adolescents. We expect to open clinical trial sites in the United Kingdom in the second half of 2018. Once preliminary safety has been demonstrated in the ongoing SB-FIX Phase 1/2 clinical trial in adults (18 years of older), we may begin enrolling adolescents (12 – 17 years of age) into the study.

SB-FIX has been granted Orphan Drug and Fast Track designations by the FDA.

Lysosomal Storage Disorders

LSDs are a heterogeneous group of rare inherited metabolic disorders including: MPS I, MPS II, Fabry disease, Gaucher disease and many others. These disorders are caused by defects in genes that encode proteins known as enzymes, which break down and eliminate unwanted substances in cells. These enzymes are found in structures called lysosomes which act as recycling sites in cells, breaking down unwanted material into simple products. A defect in a lysosomal enzyme leads to the accumulation of toxic levels of the substance that the enzyme would normally eliminate. These toxic levels may cause cell damage which can lead to serious health problems.

MPS I is caused by mutations in the gene encoding the alpha-L-iduronidase, or IDUA, enzyme, resulting in a deficiency of IDUA enzyme, which is required for the degradation of the glycosaminoglycans, or GAGs, dermatan sulfate and heparin sulfate. The inability to degrade GAGs leads to their accumulation within the lysosomes throughout the body. Individuals with this mutation experience multi-organ dysfunction and damage. Depending on the severity of the mutations and degree of residual enzyme activity, affected individuals may develop enlarged internal organs, joint stiffness, skeletal deformities, corneal clouding, hearing loss and cognition impairments. Three forms of MPS I, in order of increasing severity, include Scheie, Hurler-Scheie and Hurler syndromes. According to the National MPS Society, one in 500,000 births in the United States will result in Scheie syndrome, one in 115,000 births in Hurler/Scheie, and one in 100,000 births results in Hurler syndrome. There are approximately 1,000 MPS I patients in the United States.

MPS II is an X-linked disorder primarily affecting males and caused by mutations in the gene encoding the iduronate-2-sulfatase, or IDS, enzyme. This results in a deficiency of IDS enzyme, which is required for the degradation of GAGs. Similar to MPS I, the inability to degrade GAGs leads to their accumulation within the lysosomes throughout the body. Individuals with this mutation experience multi-organ dysfunction and damage. Children with MPS II appear normal at birth but begin showing symptoms of developmental delay by age 2 –3 years. Depending on the severity of the mutations and degree of residual enzyme activity, affected individuals may develop delayed development, enlarged internal organs, cardiovascular disorders, stunted growth and skeletal abnormalities and hearing loss. The disorder is progressive and symptoms range from mild (normal cognitive function) to severe (cognitively impaired). According to the National MPS Society, one in 100,000 male births in the United States will result in MPS II. There are approximately 500 MPS II patients in the United States.

Fabry disease is an X-linked disorder primarily affecting males and caused by a mutation in the gene encoding the alpha-galactosidase A, or alpha-Gal A, enzyme, resulting in a deficiency of alpha-Gal A enzyme, which is required for the degradation of the ganglioside globotriaosylceramide, a particular type of fatty substance. The inability to degrade this fatty substance leads to its accumulation within the lysosomes throughout the body. Individuals with this mutation experience multi-organ dysfunction and damage. Depending on the severity of the mutations and degree of residual enzyme activity, affected individuals may develop progressive kidney damage, heart attack, stroke, gastrointestinal complications, corneal opacity, tinnitus and hearing loss. Milder forms of the disorder present later in life and affect only the heart or kidneys. According to the National Institutes of Health U.S. National Library of Medicine, one in 40,000 to one in 60,000 male births in the United States will result in Fabry disease. There are approximately 2,200 males with Fabry disease in the United States. This mutation can also occur in females, however is less common and the frequency is unknown.

There are limited treatments currently available for MPS I, MPS II and Fabry disease. For individuals with MPS I, there are only two options: hematopoietic stem cell transplantation, or HSCT, for those with the most severe form of the disease (Hurler) and enzyme replacement therapy, or ERT, for patients with the attenuated forms of the disease (Hurler-Scheie, Scheie). However, the reported mortality rate after HSCT is approximately 15% and the survival rate with successful engraftment is 56%. Most patients with milder forms of the disease receive weekly ERT, usually in a doctor’s office. These IDUA enzyme infusions take on average four to six hours to administer. Weekly and bi-weekly ERT infusions are the only available options for MPS II and Fabry disease, respectively. Because of the availability of few treatment options that effectively and safely treat these diseases, there remains significant unmet medical need.

SB-913 — MPS II

We are developing SB-913, an *in vivo* genome editing product candidate, to treat MPS II. Similar to SB-318, we are using our ZFN genome editing technology to add a new therapeutic copy of the IDS gene precisely into the *Albumin* gene locus in the genome of liver cells, using the strong endogenous Albumin promoter to drive expression of the newly inserted gene.

Preclinical mouse model data demonstrated robust levels of IDS enzyme expression in the liver, blood plasma and spleen of SB-913 treated mice, resulting in a 100-fold increase in IDS activity, with sustained elevated levels in the blood plasma over the course of the entire study. Additional preclinical mouse model data demonstrated stable production of therapeutic levels of IDS enzyme from the liver into the circulation and additional secondary tissues, including the spleen, lung, muscle, heart and brain, after a single intravenous administration of SB-913. This resulted in the significant reduction of GAG biomarkers across all the tissues. Behavioral data from Barnes maze tests, collected at the end of the four-month study demonstrated statistically significant preservation of cognitive learning and memory in mice treated with SB-913, compared to untreated mice.

In 2017, we initiated an open-label, dose-ascending Phase 1/2 clinical trial, the CHAMPIONS Study, to evaluate the safety and efficacy of SB-913 in adult male subjects with attenuated MPS II, designed to enroll up to nine subjects across three ascending dose cohorts. In November 2017, we announced that the first subject had been treated in the CHAMPIONS Study. In February 2018, we presented preliminary six-week safety data from the first subject enrolled in the CHAMPIONS Study. The data demonstrated that the subject tolerated the infusion well. Mild (Grade 1) adverse events related to the study drug were reported on the fourth day after dosing. These were dizziness, weakness and frequent urination, all of which resolved within one day without treatment. No other adverse events related to the study drug have been observed. Liver function tests have remained within normal limits for the patient since the infusion. Currently, there are four patients dosed with SB-913. We expect to present additional safety and preliminary efficacy data from the CHAMPIONS Study in the third quarter of 2018. We submitted a CTA in the first half of 2018 to initiate enrollment of adolescent and pediatric subjects in the United Kingdom into the Phase 1/2 clinical trial. We expect to open clinical trial sites in the United Kingdom in the second half of 2018.

SB-913 has been granted Orphan Drug, Rare Pediatric Disease and Fast Track designations by the FDA, as well as Orphan Medicinal Product designation by the EMA.

SB-318 — MPS I

We are developing SB-318, an *in vivo* genome editing product candidate, to treat MPS I. Using the same approach as our hemophilia B product candidate, SB-FIX, we are adding a new therapeutic copy of the IDUA gene precisely into the *Albumin* gene locus in the genome of liver cells, using the strong endogenous Albumin promoter to drive expression of the newly inserted gene. We believe the potential of this approach to provide a permanent correction for a patient may be optimal for a pediatric population by reducing or eliminating the need for chronic ERT infusions.

Preclinical mouse model data demonstrated robust levels of IDUA enzyme expression in the liver, blood plasma and spleen of SB-318 treated mice, resulting in a 10-fold increase in IDUA activity, with sustained elevated levels in the blood plasma over the course of the two-month study. Additional preclinical mouse model data demonstrated stable production of therapeutic levels of IDUA enzyme from the liver into the circulation and secondary tissues, including the spleen, lung, muscle, heart and brain, after a single intravenous administration of SB-318. This resulted in the significant reduction of GAG biomarkers in all of the tissues. Behavioral data from Barnes maze tests, collected at the end of the four-month study, demonstrated statistically significant preservation of cognitive learning and memory in mice treated with SB-318, compared to untreated mice.

In 2017, we initiated an open-label, dose-ascending Phase 1/2 clinical trial, the EMPOWERS Study, to evaluate SB-318 in adult subjects with attenuated MPS I. The EMPOWERS Study is designed to enroll up to nine subjects across three ascending dose cohorts. Based on the preliminary safety data from the CHAMPIONS Study discussed above, in April 2018, we amended the protocol for the EMPOWERS Study so that the patients would initiate treatment in the study at the mid-dose level. We expect to present preliminary safety and efficacy

data from the EMPOWERS Study in 2018. We submitted a CTA to initiate enrollment of adolescent and pediatric subjects in the United Kingdom into the Phase 1/2 clinical trial. We expect to open clinical trial sites in the United Kingdom in the second half of 2018.

SB-318 MPS I has been granted Orphan Drug, Rare Pediatric Disease and Fast Track designations by the FDA, as well as Orphan Medicinal Product designation by the EMA.

ST-920 — Fabry Disease

We are developing ST-920 for Fabry disease. ST-920 is a gene therapy product candidate utilizing an AAV, carrying a galactosidase alpha, or GLA, gene construct, coding for the alpha-Gal A enzyme, driven by our proprietary synthetic liver specific promoter. We are currently conducting IND-enabling studies for ST-920 and expect to file an IND application with the FDA in 2018.

Hemoglobinopathies: Beta-thalassemia and Sickle Cell Disease

Mutations in the gene encoding beta-globin, the oxygen carrying protein of red blood cells, lead to hemoglobinopathies such as beta-thalassemia and sickle cell disease, or SCD. Both diseases manifest in the months after birth, when patients switch from producing functional fetal gamma-globin to a mutant form of adult beta-globin, which results in their condition. Naturally occurring increased levels of fetal hemoglobin have been shown to reduce the severity of both beta-thalassemia and SCD.

Beta-thalassemia is a rare disorder that results in greatly impaired production of healthy red blood cells despite bone marrow over activity, leading to life-threatening anemia, enlarged spleen, liver and heart, and bone abnormalities. We are focused on Beta-thalassemia major which is a severe form of thalassemia that requires regular, often monthly, blood transfusions and subsequent iron-chelation therapy to treat iron overload. The Centers for Disease Control and Prevention, or CDC, estimates that 1,000 people have beta-thalassemia major in the United States, and an unknown number carry the genetic trait and can pass it on to their children.

In SCD, the mutation causes the red blood cells to form an abnormal sickle or crescent shape. The cells are fragile and deliver less oxygen to the body's tissues. They can also get stuck more easily in small blood vessels and break into pieces that can interrupt healthy blood flow which further decrease the amount of oxygen flowing to body tissues. Almost all patients with SCD experience these painful vaso-occlusive crises, which can last from hours to days and may cause irreversible organ damage. Current standard of care is to manage and control symptoms, and to limit the number of crises. Treatments include administration of hydroxyurea, blood transfusions, iron-chelation therapy, pain medications and antibiotics. The CDC estimates that there are 90,000 to 100,000 Americans living with SCD, which occurs in approximately one out of every 365 African-American births and one out of every 16,300 Hispanic-American births.

ST-400 — Beta-thalassemia; BIVV-003 — SCD

We are developing ST-400 for the treatment of beta-thalassemia and our collaboration partner, Bioverativ, is developing BIVV-003 for the treatment of SCD. Both ST-400 and BIVV-003 are genome-edited cell therapies that use our ZFN genome editing technology to modify a patient's own, or autologous, hematopoietic stem/progenitor cells, or HSPCs, to produce functional red blood cells using fetal hemoglobin. Our genome editing technology can be used in HSPCs to precisely disrupt regulatory sequences that control the expression of key transcriptional regulators, such as the BCL11A erythroid enhancer sequence, to reverse the switch from expression of the mutant adult beta-globin back to the production of functional fetal gamma-globin.

The current standard of care for beta-thalassemia includes chronic blood transfusions, while the standard of care for SCD is a bone marrow transplant, or BMT, of HSPCs from a "matched" related donor, or an allogeneic

BMT. However, these therapies are limited due to the risk of iron overload with blood transfusions, requiring subsequent iron chelation therapy, and the scarcity of matched donors and the significant risk of Graft versus Host Disease, or GvHD, with BMTs after transplantation of the foreign cells. By performing genome editing in HSPCs that are isolated from and subsequently returned to the same patient (i.e., an autologous HSPC transplant), our approach has the potential to address these limitations. The goal of this approach is to develop a one-time long-lasting treatment for beta-thalassemia and SCD.

Preclinical data from clinical-scale in vitro studies have demonstrated that ST-400 and BIVV-003 can be manufactured by reproducible, high-level, ZFN-mediated modification in HSPCs mobilized in peripheral blood at clinical production scale (>108 cells), with an on-target modification efficiency of greater than 80%. Furthermore, erythroid differentiation of enhancer targeted cells showed modification of both BCL11A erythroid enhancer alleles in more than 50% of the erythroid colonies and resulted in a greater than four-fold increase in gamma globin mRNA and protein production, compared to controls. Specificity studies of ST-400 and BIVV-003 revealed no detectable off-target activity using state-of-the art, unbiased, highly sensitive oligo-capture assays. Preclinical data from *in vivo* studies in immune-deficient mice demonstrated robust long-term (19 weeks) engraftment and that targeted gene modification was maintained through multi-lineage differentiation in the bone marrow and peripheral blood.

Our IND for ST-400 was cleared by the FDA in September 2017, and we have designed an open-label, single arm Phase 1/2 clinical trial to evaluate the safety and efficacy of ST-400 in up to six adult subjects with beta-thalassemia. In March 2018, we initiated the first clinical site for this beta-thalassemia study and we expect to begin enrolling patients in the first half of 2018.

Bioverativ is our partner for ST-400 and is responsible for the clinical development of BIVV-003 for SCD.

CNS-Tauopathies

We are using our ZFP-TF gene regulation platform to develop potential gene therapies for tauopathy disorders, including Alzheimer's disease and other neurodegenerative diseases. We believe a reduction in tau protein levels can help reduce intracellular tau protein aggregation and the formation of neurofibrillary tangles in neurons, potentially ameliorating or reversing disease progression. We believe this approach may have a significant advantage compared to monoclonal antibody-based approaches to Alzheimer's disease and other tauopathy disorders because it is designed to selectively down-regulate the *tau* gene in neurons with the goal of reducing all forms of the tau protein globally across the CNS. In contrast, monoclonal antibody-based approaches are limited in that they can only bind to certain forms of tau proteins.

Preclinical studies in wildtype mice demonstrated that a single administration of *tau*-targeting ZFP-TFs resulted in up to 70% reduction of tau mRNA and protein expression across the entire CNS, as well as sustained and well-tolerated ZFP-TF expression with minimal impact on inflammatory markers. Additional preclinical studies in amyloid mouse models of Alzheimer's disease demonstrated up to 80% reduction of tau protein levels in the brain and cerebrospinal fluid, as well as significantly reduced neuritic dystrophy after a single administration of ZFP-TFs in mice with established disease pathology.

We are currently conducting preclinical studies in NHPs to evaluate our ZFP-TFs in larger mammalian species. We intend to seek a partner with disease area expertise for the clinical development and commercialization of this program.

C9ORF72–linked ALS/FTLD

In December 2017, we entered into a research collaboration and license agreement with Pfizer to develop and commercialize gene therapy products that use our ZFP TFs to treat ALS and FTLD linked to mutations of the

C9ORF72 gene. ALS and FTLD are part of a spectrum of neurodegenerative disorders caused by mutations in the C9ORF72 gene that involve hundreds of additional repetitions of a six base pair sequence of DNA. This ultimately leads to the deterioration of motor neurons, in the case of ALS, or neurons in the frontal and temporal lobes, in the case of FTLD. Currently, there are no cures to halt or reverse the progression of ALS or FTLD. The C9ORF72 mutation is linked to approximately one-third of cases of familial ALS. We and Pfizer plan to investigate allele-specific ZFP-TFs with the potential to differentiate the mutant C9ORF72 allele from the wildtype allele and to specifically down-regulate expression of the mutant form of the gene.

Huntington's Disease

Huntington's disease is an inherited, progressive neurologic disease for which there is no treatment or cure. The disease is caused by a particular type of mutation in a single gene, the HTT gene. Most patients inherit one normal and one defective or mutant copy of the HTT gene, which causes Huntington's disease. The mutation is characterized by expansion of a repeated stretch of DNA sequence within the gene called a "CAG repeat." A normal copy of the HTT gene usually has 10 to 29 of these CAG repeats but a defective copy has many more — generally greater than 39 repeats. While the protein produced by the normal copy of the gene appears to be essential for development (mice lacking the gene do not survive to birth), the product of the mutated gene is damaging to cells. Symptoms, which include deterioration of muscle control, cognition and memory, usually develop between 35 and 44 years of age. It is known that the greater the number of CAG repeats, the earlier the onset. Huntington's disease is usually fatal within 15 to 20 years after the onset of symptoms. The disease has a high prevalence for an inherited disorder. According to the Huntington's Disease Society of America, approximately 30,000 people in the United States have Huntington's disease. In addition, it is estimated that approximately 200,000 people in the United States are at risk of developing the disease.

Research in animal models of the disease has shown that lowering the levels of the mutant HTT protein can prevent, or even reverse, disease progression. However, to date most "HTT-lowering" methods decrease levels of both the normal and mutant forms of HTT, raising potential safety concerns given the importance of normal HTT protein. In collaboration with Shire, we are developing ZFP TFs that can selectively repress the expression of the mutant disease-causing form of HTT while leaving expression levels of the normal gene unchanged. Preclinical studies in animal models of the disease are ongoing and Shire is responsible for all clinical development activities including filing the IND application.

We previously announced a data security incident involving the compromise of a senior executive's company email account. While we are continuing to analyze the effects of the incident, along with the appropriate remediation of our information technology systems, at this time we do not believe that our ongoing clinical trials or any data from these trials have been compromised, particularly because we restrict the receipt of data from our clinical trials to a very limited number of employees.

New ZFN Architectures

In 2017, our scientists reported on platform advancements that substantially enhanced the precision, efficiency and specificity of ZFNs for therapeutic genome editing. We believe these advances enable rapid development of ZFNs to target chosen genomic sites with high levels of targeted modification and with no detectable off-target activity. These advances include the development of new linkers that enable base skipping between adjacent zinc finger modules as well as a reconfiguring of the ZFN architecture to allow optional placement of the Fok1 nuclease domain at either the carboxy terminal or the amino terminal end. The enhancements also include the identification of key amino acid substitutions that can be used to tune biochemical properties and remove non-specific binding contacts between the ZFN and the DNA backbone.

Advancements in T Cell Editing Capabilities

In 2018, we presented preclinical data demonstrating our ability to accomplish highly efficient multiplex genome editing of T cells. Efficient multiplex editing, the ability to make multiple genetic changes in a single step, enables simultaneous disruption of certain genes to prevent the body from rejecting the treatment and integration of new genes to equip the modified T cells with targeted antitumor functions. In the presented data, we described a T cell with four edits achieved in a single step. The four simultaneous edits included triple knockout of TCR (93% efficiency), b2 microglobulin, or B2M, (96% efficiency), CISH, a checkpoint gene (93% efficiency), and targeted insertion of green fluorescent protein, or GFP, (91% efficiency), resulting in 76% of the modified T cells with all four edits.

Our T cell engineering capabilities have advanced rapidly in the last two years with improvements in our ZFN design capabilities. These novel architectural enhancements have resulted in a 300-fold increase in potential design options for a given genetic sequence, yielding higher on-target modification activity in preclinical testing, with *ex vivo* editing efficiencies now reaching as high as 99.5%, and off-target cleavage consistently below the level of detection. We believe these improvements potentially allow for the use of substantially reduced doses of mRNA and AAV, enabling a highly efficient gene editing process that maintains T cell phenotypes, functions and proliferative capacity during *ex vivo* cell expansion.

Forward-Looking Statements

This Exhibit 99.1 contains forward-looking statements regarding our current expectations. These forward looking statements include, without limitation, references to: the potential value and therapeutic use of our technology programs; our intent to partner with other companies to advance the development and commercialization of our programs; anticipated activities under our collaborations and the scope thereof; the anticipated initiation, enrollment, scope and rate of progress of, and data availability and other milestones related to, our preclinical studies and clinical trials and those of our collaborators, as well as the anticipated timing thereof; anticipated regulatory submissions and the timing thereof; our belief as to the criticality of our intellectual property position; our beliefs as to the advancements in our T cell editing capabilities; and other statements that are not historical fact. These statements are not guarantees of future performance and are subject to certain risks, uncertainties and assumptions that are difficult to predict. Factors that could cause actual results to differ include, but are not limited to, our dependence on the success of the clinical trials of our lead programs; uncertainties related to the initiation, enrollment and completion of clinical trials and the timing of data availability; whether clinical trial results will validate and support the safety and efficacy of our product candidates; our ability to maintain our collaborative relationships and our reliance on collaborators and other third parties to meet their obligations; our ability to establish additional collaborations or strategic partnerships; our ability to obtain or protect intellectual property rights related to our product candidates; technological challenges we may encounter; the lengthy and uncertain regulatory approval process; our ability to develop commercially viable products; technological developments by our competitors; and other risks and uncertainties affecting Sangamo and its development programs. Actual results may differ from those projected in forward-looking statements due to risks and uncertainties that exist in our operations and business environments. These risks and uncertainties are described more fully under the heading “Risk Factors” contained in Exhibit 99.1 to our Current Report on Form 8-K, filed with the Securities and Exchange Commission on April 17, 2018. Forward-looking statements contained in this Exhibit 99.1 are made as of the date hereof, and Sangamo undertakes no duty to update such information except as required under applicable law.