SANGAMO THERAPEUTICS, INC
Form 424B5
April 26, 2018
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**Filed Pursuant to Rule 424(b)(5)**
**Registration No. 333-224418**

**CALCULATION OF REGISTRATION FEE**

| Title Of Each Class Of Securities To Be Registered | Amount To Be Registered | Proposed Maximum Offering Price Per Unit | Proposed Maximum Aggregate Offering Price | Amount Of Registration Fee |
|---|---|---|---|---|
| Common Stock, par value $0.01 per share | 14,156,500(1) | $16.25 | $230,043,125 | $28,640.37(2) |

(1) Includes 1,846,500 shares that may be purchased by the underwriters upon exercise of the underwriters over-allotment option.
(2) The filing fee is calculated and being paid pursuant to Rule 457(r) under the Securities Act of 1933, as amended, and relates to the Registration Statement on Form S-3 (File No. 333-224418) filed by the Registrant on April 24, 2018.

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**Prospectus Supplement**

**(To Prospectus dated April 24, 2018)**

**12,310,000 Shares**

**Common Stock**

We are offering 12,310,000 shares of our common stock.

Our common stock is listed on The Nasdaq Global Select Market under the symbol SGMO. On April 25, 2018, the last reported sale price of our common stock as reported on The Nasdaq Global Select Market was $17.30 per share.

| | Per Share | Total |
|---|---|---|
| Public offering price | $ 16.250 | $ 200,037,500 |
| Underwriting discounts and commissions(1) | $ 0.975 | $ 12,002,250 |
| Proceeds to Sangamo Therapeutics, before expenses | $ 15.275 | $ 188,035,250 |

(1) See the section entitled Underwriting for additional disclosure regarding underwriter compensation and estimated offering expenses.

We have granted the underwriters an option for a period of 30 days to purchase up to 1,846,500 additional shares of our common stock, solely to cover overallotments.

**Investing in our common stock involves a high degree of risk. See Risk Factors beginning on page S-13 of this prospectus supplement and in the Current Report on Form 8-K filed with the Securities and Exchange Commission on April 17, 2018, as well as our other filings that are incorporated by reference into this prospectus supplement and the accompanying prospectus.**

**Neither the Securities and Exchange Commission nor any state securities commission has approved or disapproved of these securities or determined if this prospectus supplement or the accompanying prospectus is truthful or complete. Any representation to the contrary is a criminal offense.**

Gilead Sciences, Inc. has indicated an interest in purchasing 3,076,923 of the shares of common stock offered hereby at the price offered to the public. Because this indication of interest is not a binding agreement or commitment to purchase, this entity may elect not to purchase any shares in this offering, or the underwriters may elect not to sell any shares in this offering to it.

The underwriters expect to deliver the shares to purchasers on or about April 30, 2018.

*Joint Book-Running Managers*

**BofA Merrill Lynch** **J.P. Morgan** **Cowen**

**April 25, 2018**

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### Prospectus Supplement



### Prospectus

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**We have not, and the underwriters have not, authorized anyone to provide you with information different than or inconsistent with the information contained in or incorporated by reference in this prospectus supplement, the accompanying prospectus and in any free writing prospectus that we have authorized for use in connection with this offering. We and the underwriters take no responsibility for, and can provide no assurance as to the reliability of, any other information that others may give you. We are not, and the underwriters are not, making an offer to sell these securities in any jurisdiction where the offer or sale is not permitted. You should assume that the information appearing in this prospectus supplement, the accompanying prospectus, the documents incorporated by reference in this prospectus supplement and the accompanying prospectus, and in any free writing prospectus that we have authorized for use in connection with this offering, is accurate only as of the date of those respective documents, regardless of the time of delivery of those respective documents. Our business, financial condition, results of operations and prospects may have changed since those dates. You should read this prospectus supplement, the accompanying prospectus, the documents incorporated by reference in this prospectus supplement and the accompanying prospectus, and any free writing prospectus that we have authorized for use in connection with this offering, in their entirety before making an investment decision. You should also read and consider the information in the documents to which we have referred you in the sections of this prospectus supplement entitled Where You Can Find More Information and Incorporation of Certain Information by Reference.**

## ABOUT THIS PROSPECTUS SUPPLEMENT

This document is in two parts. The first part is this prospectus supplement, which describes the terms of this offering of common stock and also adds to and updates information contained in the accompanying prospectus and the documents incorporated by reference into this prospectus supplement and the accompanying prospectus. The second part, the accompanying prospectus dated April 24, 2018, including the documents incorporated by reference therein, provides more general information. Generally, when we refer to this prospectus, we are referring to both parts of this document combined. To the extent there is a conflict between the information contained in this prospectus supplement, on the one hand, and the information contained in the accompanying prospectus or in any document incorporated by reference that was filed with the Securities and Exchange Commission, or SEC, before the date of this prospectus supplement, on the other hand, you should rely on the information in this prospectus supplement. If any statement in one of these documents is inconsistent with a statement in another document having a later date for example, a document incorporated by reference in the accompanying prospectus the statement in the document having the later date modifies or supersedes the earlier statement.

All references in this prospectus supplement and the accompanying prospectus to Sangamo, the Company, we, us, or similar references refer to Sangamo Therapeutics, Inc., a Delaware corporation, and its subsidiaries on a consolidated basis, except where the context otherwise requires or as otherwise indicated.

This prospectus supplement, the accompanying prospectus, and the information incorporated herein and therein by reference include trademarks, trade names and service marks owned by us or other companies. 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 prospectus supplement, the accompanying prospectus and the information incorporated herein and therein by reference are the property of their respective owners.

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## PROSPECTUS SUPPLEMENT SUMMARY

*This summary highlights certain information about us, this offering and selected information contained elsewhere in or incorporated by reference into this prospectus supplement. This summary is not complete and does not contain all of the information that you should consider before deciding whether to invest in our common stock. For a more complete understanding of our company and this offering, you should read and consider carefully the more detailed information included or incorporated by reference in this prospectus supplement and the accompanying prospectus, including the factors described under the heading Risk Factors of this prospectus supplement, in our Current Report on Form 8-K filed with the SEC on April 17, 2018 and in our other filings that are incorporated by reference into this prospectus supplement and the accompanying prospectus, as well as the information included in any free writing prospectus that we have authorized for use in connection with this offering.*

### 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.

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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.

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## 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

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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.

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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.

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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

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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

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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

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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.

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**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.

**Certain Preliminary Financial Results**

As of March 31, 2018, we had approximately $234.9 million of cash, cash equivalents and investments. This amount is unaudited and preliminary, is subject to completion of financial closing procedures that could result in changes to the amount, and does not present all information necessary for an understanding of our financial condition as of March 31, 2018. This amount also does not reflect $150.0 million that we received in April 2018 under the terms of our collaboration agreement with Kite.

**Corporate Information**

We were incorporated in June 1995 in the state of Delaware and in January 2017, we changed our name from Sangamo BioSciences, Inc. to Sangamo Therapeutics, Inc. Our principal executive offices are located at 501 Canal Boulevard, Richmond, California 94804. Our telephone number is (510) 970-6000. Our website is *www.sangamo.com*. Information found on, or accessible through, our website is not a part of, and is not incorporated into, this prospectus supplement or the accompanying prospectus, and you should not consider it part of this prospectus supplement or the accompanying prospectus. Our website address is included in this document as an inactive textual reference only.

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**The Offering**

| | |
|---|---|
| Common stock offered by us | 12,310,000 shares |
| Common stock to be outstanding immediately after this offering | 97,908,534 shares |
| Option to purchase additional shares | The underwriters have an option to purchase up to 1,846,500 additional shares of our common stock, solely to cover overallotments. The underwriters may exercise this option at any time within 30 days from the date of this prospectus supplement. |
| Use of proceeds | We intend to use the net proceeds from this offering for working capital and other general corporate purposes, including support for our own and our partnered gene therapy, genome editing, cell therapy and gene regulation product candidates and research programs, our manufacturing facilities and other business development activities. See Use of Proceeds. |
| Nasdaq Global Select Market Symbol | SGMO |
| Risk factors | Investing in our common stock involves a high degree of risk. See Risk Factors. |

The number of shares of our common stock to be outstanding immediately after this offering as shown above is based on 85,598,534 shares of common stock outstanding as of December 31, 2017 and excludes:

- 8,287,456 shares of our common stock issuable upon the exercise of options outstanding as of December 31, 2017, having a weighted-average exercise price of $7.77 per share;
- 80,172 shares of our common stock issuable upon the vesting of restricted stock units outstanding as of December 31, 2017;
- 3,601,633 shares of our common stock reserved for future issuance under our Amended and Restated 2013 Stock Incentive Plan, or the 2013 Plan, as of December 31, 2017; and
- 835,674 additional shares of our common stock reserved for future issuance under our 2010 Employee Stock Purchase Plan, or the ESPP, as of December 31, 2017.

In addition, the number of shares of our common stock to be outstanding immediately after this offering as shown above excludes the up to $75.0 million of our common stock that remained available for sale at December 31, 2017 pursuant to an Amended and Restated At-the-Market Offering Program Sales Agreement, or the ATM Agreement, that we entered into with Cowen and Company, LLC, or Cowen, on May 26, 2017. Moreover, our compensation committee has reserved an additional 2,500,000 shares of our common stock for issuance under the ESPP, subject to the receipt of stockholder approval at our 2018 annual meeting of stockholders anticipated to be held on June 11, 2018, or the 2018 annual meeting. At the 2018 annual meeting, we will also be asking our stockholders to approve our 2018 Equity Incentive Plan, or the 2018 Plan, which is intended to be the successor of the 2013 Plan, and which will include a new reserve of 8,800,000 shares of our common stock (in addition to the shares of our common stock that are, or would become, available under our 2013 Plan). The number of shares of our common stock to be outstanding immediately after this offering as shown above does not reflect these additional shares.

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Except as otherwise indicated, all information in this prospectus supplement assumes no exercise by the underwriters of their option to purchase additional shares.

Gilead Sciences, Inc. has indicated an interest in purchasing 3,076,923 of the shares of common stock offered hereby at the price offered to the public. Because this indication of interest is not a binding agreement or commitment to purchase, this entity may elect not to purchase any shares in this offering, or the underwriters may elect not to sell any shares in this offering to it.

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## RISK FACTORS

*Investing in our common stock involves a high degree of risk. Before deciding whether to invest in our common stock, you should consider carefully the risks described below and discussed under the section captioned Risk Factors contained in our Current Report on Form 8-K filed with the SEC on April 17, 2018 and in our other filings that are incorporated by reference in this prospectus supplement and the accompanying prospectus in its entirety, together with the other information in this prospectus supplement, the accompanying prospectus, and the documents incorporated by reference, and in any free writing prospectus that we have authorized for use in connection with this offering. If any of these risks actually occur, our business, financial condition, results of operations or prospects could be seriously harmed. This could cause the trading price of our common stock to decline, resulting in a loss of all or part of your investment.*

### Risks Relating to this Offering and our Common Stock

***Our stock price has been volatile and may continue to be volatile, which could result in substantial losses for investors in this offering.***

Our stock price has been volatile and may continue to be volatile, which could cause investors in this offering to incur substantial losses. An active public market for our common stock may not be sustained, and the market price of our common stock may continue to be highly volatile. The market price of our common stock has fluctuated significantly and may continue to be subject to substantial volatility in response to various factors, some of which are beyond our control, including but not limited to the following:

- announcements by us or collaborators providing updates on the progress or development status of product candidates;
- data from clinical trials;
- initiation or termination of clinical trials;
- changes in market valuations of similar companies;
- overall market and economic conditions, including the equity markets for emerging biotechnology companies;
- deviations in our results of operations from the guidance given by us;
- announcements by us or our competitors of new or enhanced products, technologies or services or significant contracts, acquisitions, strategic relationships, joint ventures or capital commitments;

announcement of changes in business and operations by our collaborators and partners, or changes in our existing collaboration agreements;

regulatory developments;

changes, by one or more of our security analysts, in recommendations, ratings or coverage of our stock;

additions or departures of key personnel;

future sales of our common stock or other securities by us, management or directors, liquidation of institutional funds that comprised large holdings of our stock; and

decreases in our cash balances.

Actual or potential sales of our common stock by our employees, including our executive officers, pursuant to pre-arranged stock trading plans could cause our stock price to fall or prevent it from increasing for numerous reasons, and actual or potential sales by such persons could be viewed negatively by other investors.

In accordance with the guidelines specified under Rule 10b5-1 of the Securities Exchange Act of 1934, as amended, or the Exchange Act, and our policies regarding stock transactions, a number of our employees,

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including executive officers and members of our board of directors, have adopted and may continue to adopt stock trading plans pursuant to which they have arranged to sell shares of our common stock from time to time in the future. Generally, sales under such plans by our executive officers and directors require public filings. Actual or potential sales of our common stock by such persons could cause the price of our common stock to fall or prevent it from increasing for numerous reasons.

***If you purchase shares of common stock in this offering, you will experience immediate and substantial dilution in your investment.***

Because the price per share of our common stock being offered is substantially higher than the net tangible book value per share of our common stock, you will suffer immediate and substantial dilution with respect to the net tangible book value of the common stock you purchase in this offering. Based on the public offering price of $16.25 per share and our net tangible book value as of December 31, 2017, if you purchase shares of common stock in this offering, you would suffer immediate and substantial dilution of $12.43 per share with respect to the net tangible book value of the common stock. See the section entitled Dilution for a more detailed discussion of the dilution you will incur if you purchase common stock in this offering.

***Future sales and issuances of our common stock or rights to purchase common stock, including pursuant to our equity incentive plans, could result in additional dilution of the percentage ownership of our stockholders, including investors in this offering, and could cause our stock price to fall.***

Additional capital will be needed in the future to continue our planned operations. To the extent we raise additional capital by issuing equity securities, including pursuant to the ATM Agreement, our stockholders, including investors in this offering, may experience substantial dilution. We may sell common stock, convertible securities or other equity securities in one or more transactions at prices and in a manner we determine from time to time. If we sell common stock, convertible securities or other equity securities in more than one transaction, our stockholders, including investors who purchase shares of common stock in this offering, will experience additional dilution, and any such issuances may result in downward pressure on the price of our common stock. We also cannot assure you that we will be able to sell shares or other securities in any other offering at a price per share that is equal to or greater than the price per share paid by investors in this offering, and investors purchasing shares or other securities in the future could have rights superior to existing stockholders.

***We will have broad discretion in the use of the net proceeds from this offering and may not use them effectively.***

We will have broad discretion in the use of the net proceeds from this offering and could spend the proceeds in ways that do not improve our results of operations or enhance the value of our common stock. Our failure to apply these funds effectively could have a material adverse effect on our business, impair or delay our ability to develop our product candidates, and cause the price of our common stock to decline.

***Our stock price is also influenced by public perception of gene therapy and government regulation of potential products.***

Reports of serious adverse events in a retroviral gene transfer trial for infants with X-linked severe combined immunodeficiency (X-linked SCID) in France and subsequent FDA actions putting related trials on hold in the United States had a significant negative impact on the public perception and stock price of certain companies involved in gene therapy. Stock prices of these companies declined whether or not the specific company was involved with retroviral gene transfer for the treatment of infants with X-linked SCID, or whether the specific company s clinical trials were placed on hold in connection with these events. Other potential adverse events in the field of gene therapy

may occur in the future that could result in greater governmental regulation of our potential products and potential regulatory delays relating to the testing or approval of our potential products. These external events may have a negative impact on public perception of our business, which could cause our stock price to decline.

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***If securities or industry analysts do not publish research or publish inaccurate or unfavorable research about our business, our stock price and trading volume could decline.***

The trading market for our common stock will depend in part on the research and reports that securities or industry analysts publish about us or our business. In the event securities or industry analysts who cover us downgrade our stock or publish inaccurate or unfavorable research about our business, our stock price would likely decline. If one or more of these analysts cease coverage of our company or fail to publish reports on us regularly, demand for our stock could decrease, which might cause our stock price and trading volume to decline.

***We do not intend to pay dividends on our common stock so any returns will be limited to the value of our stock.***

We have never declared or paid any cash dividends on our common stock. We currently anticipate that we will retain future earnings for the development, operation and expansion of our business and do not anticipate declaring or paying any cash dividends for the foreseeable future. Any return to stockholders, including investors in this offering, will therefore be limited to the appreciation of their stock.

***Anti-takeover provisions in our certificate of incorporation and Delaware law could make an acquisition of our company more difficult and could prevent attempts by our stockholders, including investors in this offering, to remove or replace current management.***

Anti-takeover provisions of Delaware law and in our certificate of incorporation and our bylaws may discourage, delay or prevent a change in control of our company, even if a change in control would be beneficial to our stockholders. In addition, these provisions may frustrate or prevent any attempts by our stockholders, including investors in this offering, to replace or remove our current management by making it more difficult for stockholders to replace members of our board of directors. In particular, under our certificate of incorporation our board of directors may issue up to 5,000,000 shares of preferred stock with rights and privileges that might be senior to our common stock, without the consent of the holders of the common stock. Moreover, without any further vote or action on the part of the stockholders, the board of directors would have the authority to determine the price, rights, preferences, privileges, and restrictions of the preferred stock. This preferred stock, if it is ever issued, may have preference over, and harm the rights of, the holders of common stock. Although the issuance of this preferred stock would provide us with flexibility in connection with possible acquisitions and other corporate purposes, this issuance may make it more difficult for a third party to acquire a majority of our outstanding voting stock.

Similarly, our authorized but unissued common stock is available for future issuance without stockholder approval. Our certificate of incorporation further provides that stockholders may not take action by written consent.

In addition, our amended and restated bylaws, as amended:

- establish advance notice requirements for nominations for election to the board of directors or proposing matters that can be acted upon at stockholders meetings; and
- prohibit stockholders from calling a special meeting of stockholders.

We are also subject to Section 203 of the Delaware General Corporation Law, which provides, subject to certain exceptions, that if a person acquires 15% of our voting stock, the person is an interested stockholder and may not engage in business combinations with us for a period of three years from the time the person acquired 15% or more or

our voting stock. The application of Section 203 may, in some circumstances, deter or prevent a change in control of our company even when such change may be beneficial to our stockholders.

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***Our amended and restated bylaws, as amended, provide that the Court of Chancery of the State of Delaware will be the exclusive forum for the adjudication of certain disputes, which could limit the ability of investors in this offering to obtain a favorable judicial forum for disputes with us or our directors, officers, or employees.***

Our amended and restated bylaws, as amended, provide that the Court of Chancery of the State of Delaware is the sole and exclusive forum for:

any derivative action or proceeding brought on our behalf;

any action asserting a claim of breach of a fiduciary duty owed by any director, officer or other employee of Sangamo to us or our stockholders;