Sangamo THERAPEUTICS

J.P. Morgan 37th Annual Healthcare Conference

January 9, 2019

Forward-Looking Statements

This presentation contains forward-looking statements within the meaning of the "safe harbor" provisions of the Private Securities Litigation Reform Act of 1995, as amended. These forward-looking statements include, but are not limited to, the design of clinical trials and expected timing for enrollment and presentation of data; the anticipated clinical development milestones and other potential value drivers in the future; the expected benefits of our collaborations, the expanded capability of Sangamo's technologies; the research and development of novel gene-based therapies and the application of Sangamo's ZFP technology platform to specific human diseases; successful manufacturing of our product candidates; the potential of Sangamo's genome editing technology to safely treat genetic diseases; the potential for ZFNs to be effectively designed to treat diseases through genome editing; plans to conduct controlled withdrawal of weekly ERT infusions in MPS II subjects in the CHAMPIONS Study; anticipated next steps for the CHAMPIONS and Alta Studies, and Sangamo's expectation that it will present longer-term safety and efficacy results from the CHAMPIONS Study in February at the 2019 WORLDSymposium meeting. These statements are based upon our current expectations and speak only as of the date hereof. Our actual results may differ materially and adversely from those expressed in any forward-looking statements as a result of various factors and uncertainties. Factors that could cause actual results to differ include, but are not limited to, the dependence on the success of clinical trials of lead programs, the lengthy and uncertain regulatory approval process, uncertainties related to the timing of initiation, enrollment and completion of clinical trials, whether clinical trial results will validate and support the safety and efficacy of Sangamo's therapeutics, risks and uncertainties related to preliminary data, whether the preliminary data from the CHAMPIONS and Alta Studies and will be representative of final results, whether the final results from the CHAMPIONS and Alta Studies will validate and support the safety and efficacy of SB-913 and SB-525 respectively, and the reliance on partners and other third-parties to meet their obligations. Further, there can be no assurance that the necessary regulatory approvals will be obtained or that Sangamo and its partners will be able to develop commercially viable gene-based therapeutics. Actual results may differ from those projected in forward-looking statements due to risks and uncertainties that exist in Sangamo's operations. These risks and uncertainties are described more fully in Sangamo's Quarterly Reports on Form 10-Q for the quarter ended September 30, 2018 as filed with the Securities and Exchange Commission. Forward-looking statements contained in this presentation are made as of the date hereof, and Sangamo undertakes no obligation to update such information except as required under applicable law.

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We are committed to translating ground-breaking science into genomic medicines that transform patients' lives

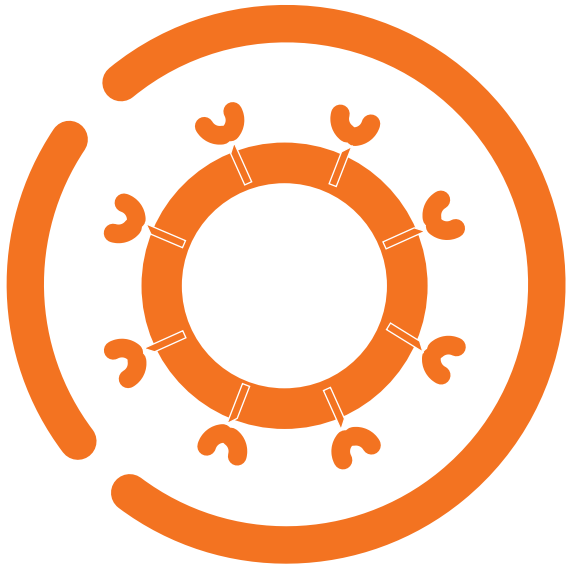
Why to invest in Sangamo in 2019

- Leading genomic medicine company active in gene editing, gene therapy, cell therapy and gene regulation
- Precise, efficient and specific gene editing technology (ZFNs) backed by a robust patent estate
- Broad portfolio of rare and large indications across inherited metabolic diseases, immunology, CNS, hematology and oncology
- Flow of clinical data readouts in 2019 and 2020 following enrollment progress of last twelve months
- Strong balance sheet and 4 validating biopharma partnerships (Kite, Pfizer, Bioverativ, Shire)

Proprietary genomic medicines pipeline focused on three therapeutic areas

Inherited Metabolic Diseases

Rare Populations



Immunology

Rare + Large Populations

Central Nervous System

Rare + Large Populations

Partnered therapeutic areas

Hematology

Oncology

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Clinical Development

- SB-913: MPS II
- SB-318: MPS I
- SB-525: Hemophilia A
- SB-FIX: Hemophilia B
- ST-400: Beta thalassemia

Active clinical trials in inherited metabolic diseases and hematology

Therapeutic Area	Research	Preclinical	Phase I/II Clinical	Phase III Registrational	Collaborator
Inherited Metabolic Diseases					
MPS I (SB-318)					
MPS II (SB-913)					
Hematology					
Hemophilia A (SB-525)					Pfizer
Hemophilia B (SB-FIX)					
Hemoglobinopathies (ST-400, BIVV-003)					Bioverativ A SANOFI COMPANY

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What is MPS II or Hunter syndrome?

About MPS II (Hunter syndrome)

- Inherited, X-linked metabolic disease caused by mutations in the gene encoding iduronate-2-sulfatase (IDS) enzyme
- Mutations in IDS gene result in loss of IDS metabolic enzyme activity
- Accumulation of toxic waste products called glycosaminoglycans (GAGs) in lysosomes leads to tissue and organ damage
- Enzyme replacement therapy (ERT) does not address all symptoms of the disease, e.g. neurocognitive decline

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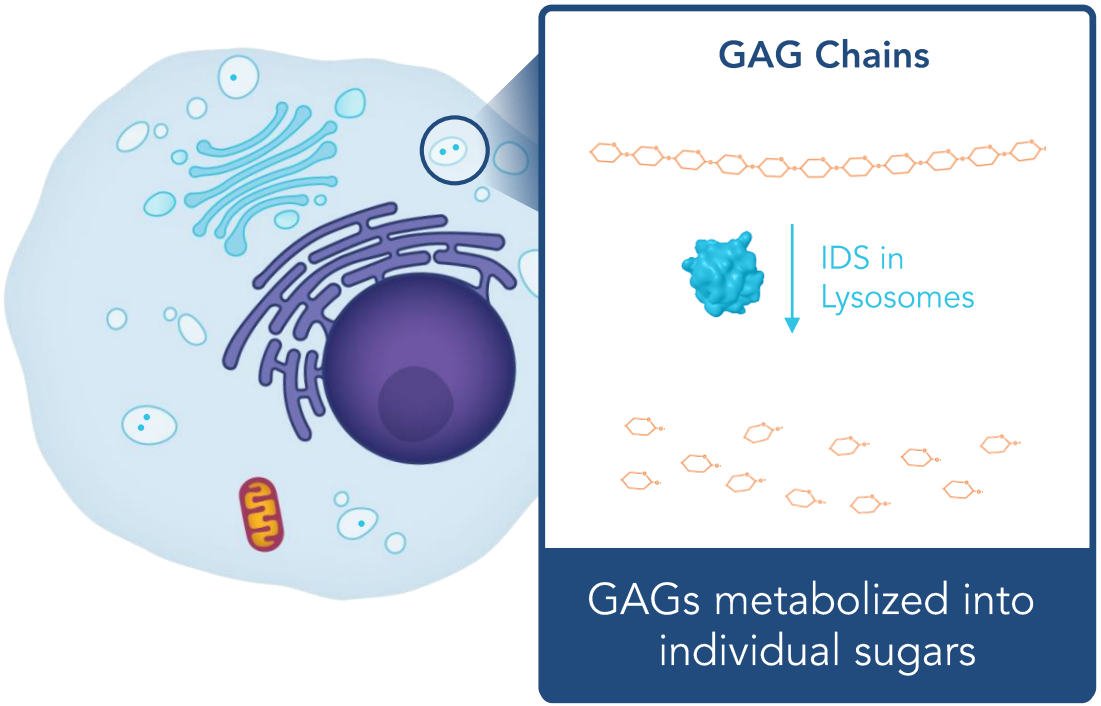
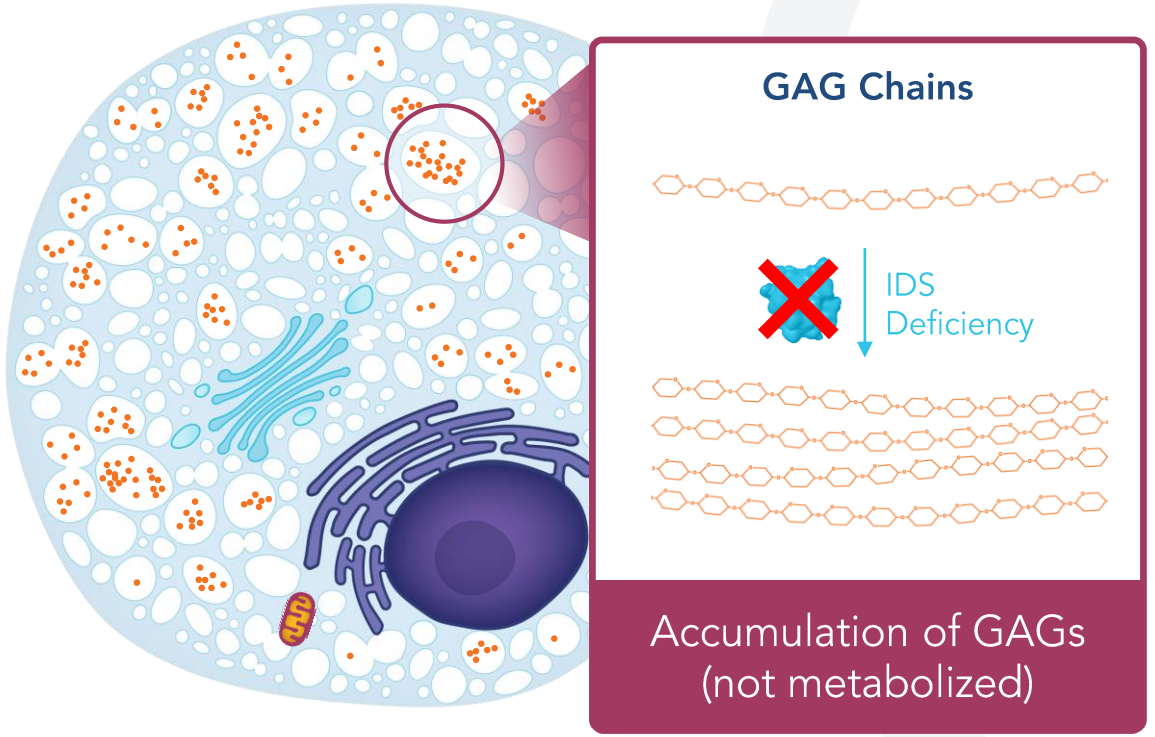
A lack of active IDS enzyme results in accumulation of GAGs in the lysosomes, leading to loss of cellular function and organ damage

Normal Cell



MPS II Cell



Toxic build up enlarges lysosomes, crowds critical organelles and engorges the cell

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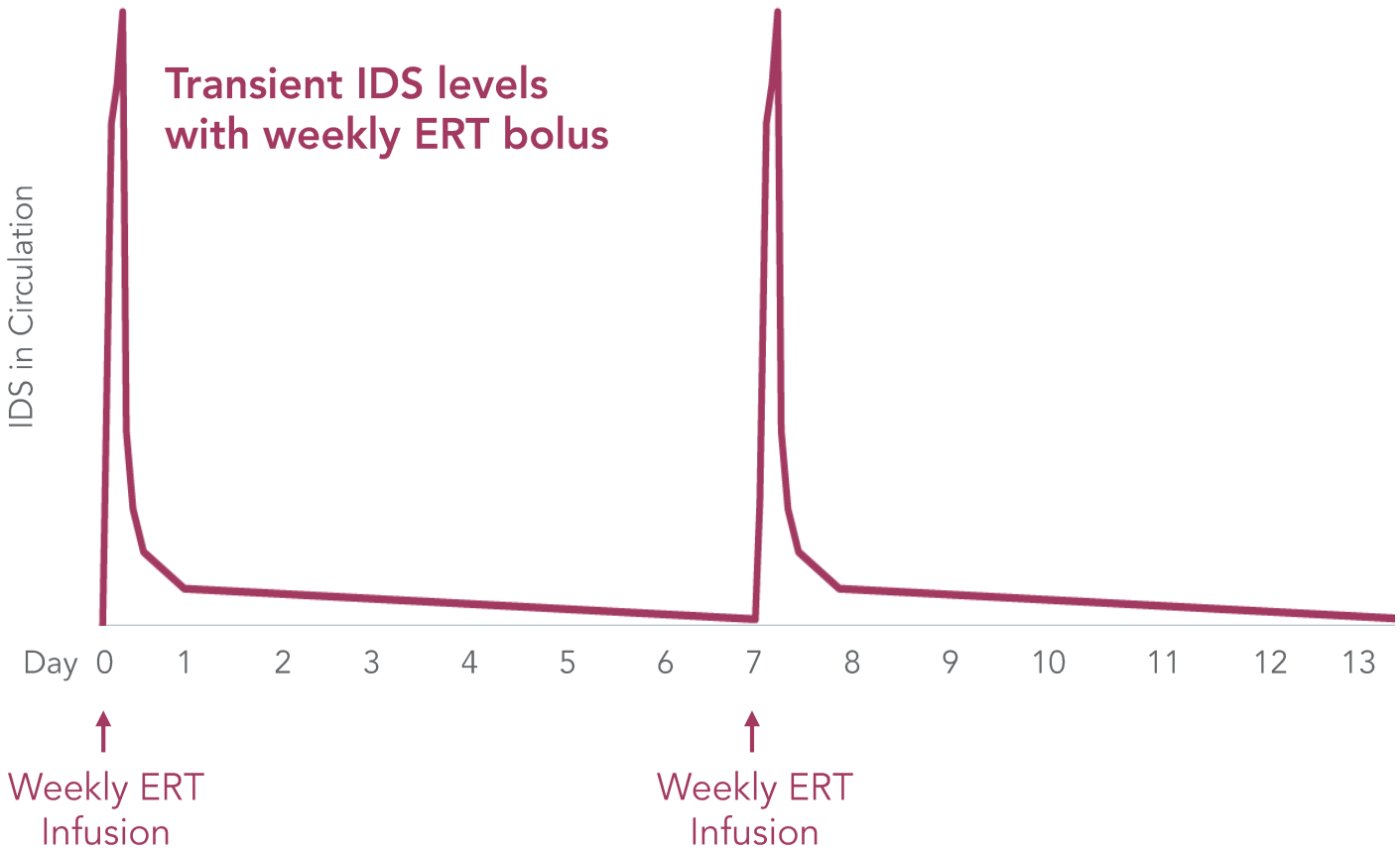
Weekly ERT infusions do not provide sustained exposure of IDS to the tissues, enzyme is rapidly cleared from circulation within hours

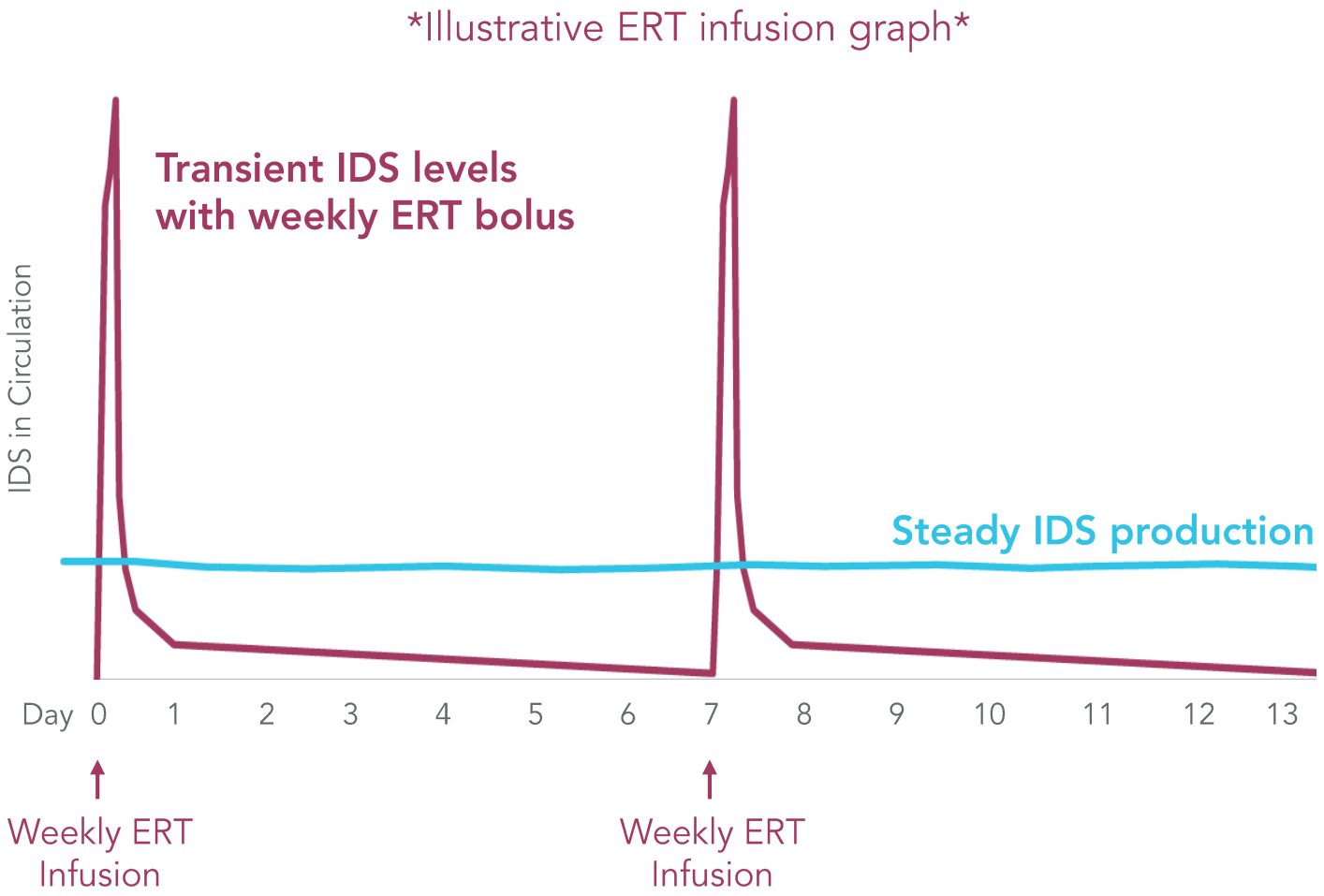
Illustrative ERT infusion graph



- ERT half-life is approximately 56 minutes[1]
- Large amount of enzyme is taken up by the liver due to high-capacity, low-specificity receptors on liver cell surface
- For significant period of time (i.e. 5-6 days out of the week), patients' enzyme levels are very low or absent
- PK modeling: steady IDS production allows for longer interaction of IDS with uptake receptor, leading to GAG reduction

[1]Elaprase®

Weekly ERT infusions do not provide sustained exposure of IDS to the tissues, enzyme is rapidly cleared from circulation within hours



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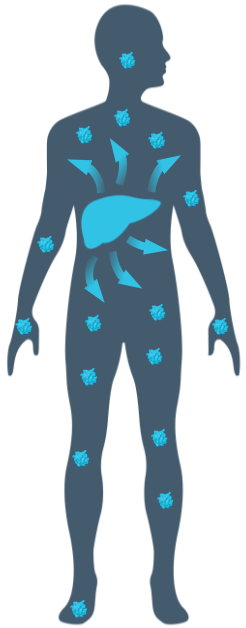
[1]Elaprase®

The goal of genome editing is to continuously produce IDS in order to stabilize or reduce GAG accumulation

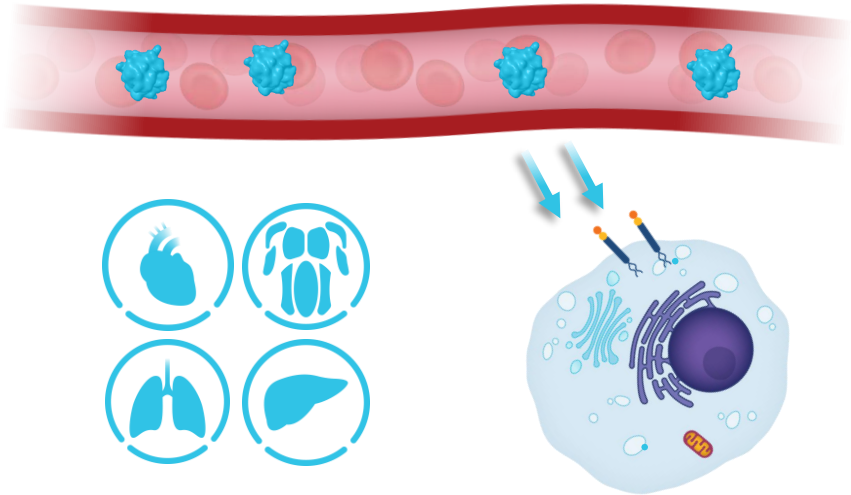
1

Edited liver cells steadily release IDS enzyme into the circulation



2

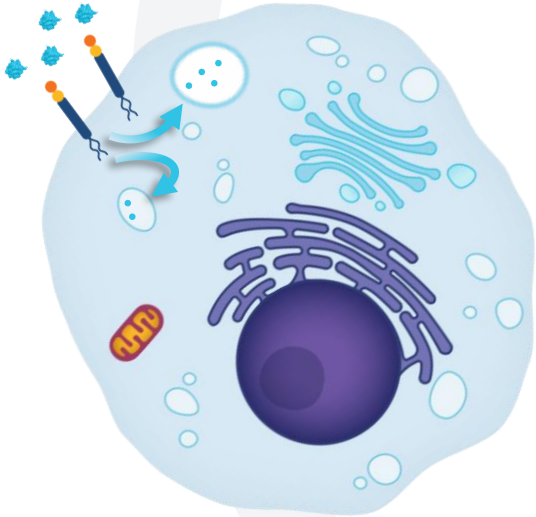
Stable enzyme levels in circulation increase IDS exposure in tissues throughout the body, facilitating receptor-mediated uptake of enzyme



Continuous IDS exposure may also facilitate uptake in tissues with limited vascularization

3

IDS enzyme is transported to lysosomes to metabolize GAGs



SB-913, *in vivo* genome editing for MPS II

Expanding high dose cohort, initiating adolescent dosing and ERT withdrawal

Phase I/II Open Label Study

9 Adults (18+ yrs), 2+ adolescents (12-17 yrs) and 2+ children (5-11 yrs) with MPS II

Dose Escalation Complete

Cohort 1	Cohort 2	Cohort 3
5e12 vg/kg	1e13 vg/kg	5e13 vg/kg

October SMC Review / Recommendations

- **Adult Cohort Expansion (5e13 vg/kg)**
- **Initiate Adolescent Dosing (12-17 years)**
- **Initiate ERT withdrawal**

Adult Cohort Expansion and Adolescent Screening Underway

5e13 vg/kg

Next steps

- Interim data update from cohorts 1-3 at WORLD*Symposium* 2019
- Complete ERT withdrawal
- Complete adult expansion cohort enrollment
- Adolescent cohort enrollment

FDA
- Orphan Drug
- Fast Track
- Rare Pediatric Disease

- Orphan Medicinal Product

IND open

CTA granted

Goals

Patient safety | Reduction in GAGs | IDS enzyme production | ERT withdrawal

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SB-318, *in vivo* genome editing for MPS I

High dose cohort enrollment complete

Phase I/II Open Label Study

Up to 9 adults (18+ yrs) with MPS I

Dose Escalation Complete

Cohort 1	Cohort 2	Cohort 3
5e12 vg/kg	1e13 vg/kg	5e13 vg/kg

October SMC Review / Recommendations

Advance Enrollment to the 5e13 vg/kg Dose Cohort

Next steps

- Preliminary data from first 3 patients at WORLD*Symposium* 2019
- SMC review and recommendations

- Orphan Drug
- Fast Track
- Rare Pediatric Disease

- Orphan Medicinal Product

IND open

CTA granted

Goals

Patient safety | Reduction in GAGs | IDS enzyme production | ERT withdrawal

SB-913 (MPS II) and SB-318 (MPS I):
A series of anticipated data events in 2019

First update: SB-913 and SB-318 interim data presentations

February 7th - WORLDSymposium 2019

- SB-913 (MPS II): Update on safety and biochemical changes at up to 24 weeks from 6 adult patients (low, mid and high-dose cohorts)
- SB-318 (MPS I): Preliminary safety and biochemical changes at up to 4 weeks from first 3 adult patients (mid and high-dose cohorts)

Potential additional updates throughout 2019 as programs progress and data accumulate

- Cohort expansion data
- Liver biopsy analyses
- ERT withdrawal
- Adolescent cohort updates
- Outcomes measurements

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SB-525, cDNA gene therapy for hemophilia A

Based on safety profile, SMC recommended advancing to a higher dose

Phase I/II Open Label Study

Up to 20 adult (18+ yrs) males with severe hemophilia A

Dose Escalation Ongoing

Cohort 1 **Cohort 2** **Cohort 3** **Cohort 4**

October SMC Review and Recommendations

Continue Escalation to an Additional Dose

Next steps

- Complete cohort 4 enrollment
- SMC review and recommendation

FDA

- Orphan Drug
- Fast Track

- Orphan Medicinal Product

IND open

Goals

Patient safety

FVIII activity

Reduction of bleeding events

Reduction of factor replacement use

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SB-FIX, *in vivo* genome editing for hemophilia B

First patient treated at high dose

Phase I/II Open Label Study

Up to 12 adult (18+ yrs) males and 3 adolescents (12-17 yrs) with severe hemophilia B

Dose Escalation Ongoing

Cohort 1	Cohort 2	Cohort 3
5e12 vg/kg	1e13 vg/kg	5e13 vg/kg

October SMC Review and Recommendations

Begin Adult Enrollment at the 5e13 vg/kg Dose Cohort

Next steps

- Complete 5e13 vg/kg cohort enrollment
- SMC review and recommendation

- Orphan Drug
- Fast Track

- Orphan Medicinal Product

IND open

CTA granted

Goals

Patient safety

FIX activity

Reduction of bleeding events

Reduction of factor replacement use

SB-525 (hemophilia A) and SB-FIX (hemophilia B):
Potential news flow in 2019

SB-525: hemophilia A

Study Update
- SMC cohort expansion recommendation

Data Update
- Following completion of dose escalation
- Safety and factor levels, factor replacement utilization, bleeding events

SB-FIX: hemophilia B

Study Update
- Patient enrollment and SMC recommendations

Data Update
- Following SMC review of first 2 patients in 5e13 vg/kg dose cohort
- Safety and factor levels

ST-400, gene-edited cell therapy for beta thalassemia

Phase I/II Open Label Study

6 adults (18+) with transfusion-dependent beta thalassemia

Patient Enrollment Ongoing

Potential Advantages

- Leverages naturally-occurring, protective mechanism to increase fetal-hemoglobin to reduce or potentially eliminate blood transfusions
- Highly efficient, precise gene editing; low risk of insertional mutagenesis
- Non-viral delivery of ZFNs

Next steps

- Complete enrollment of 6 patients

IND open

Goals

- Patient safety
- Successful engraftment
- Fetal hemoglobin (HbF) production
- Reduction / elimination of transfusions

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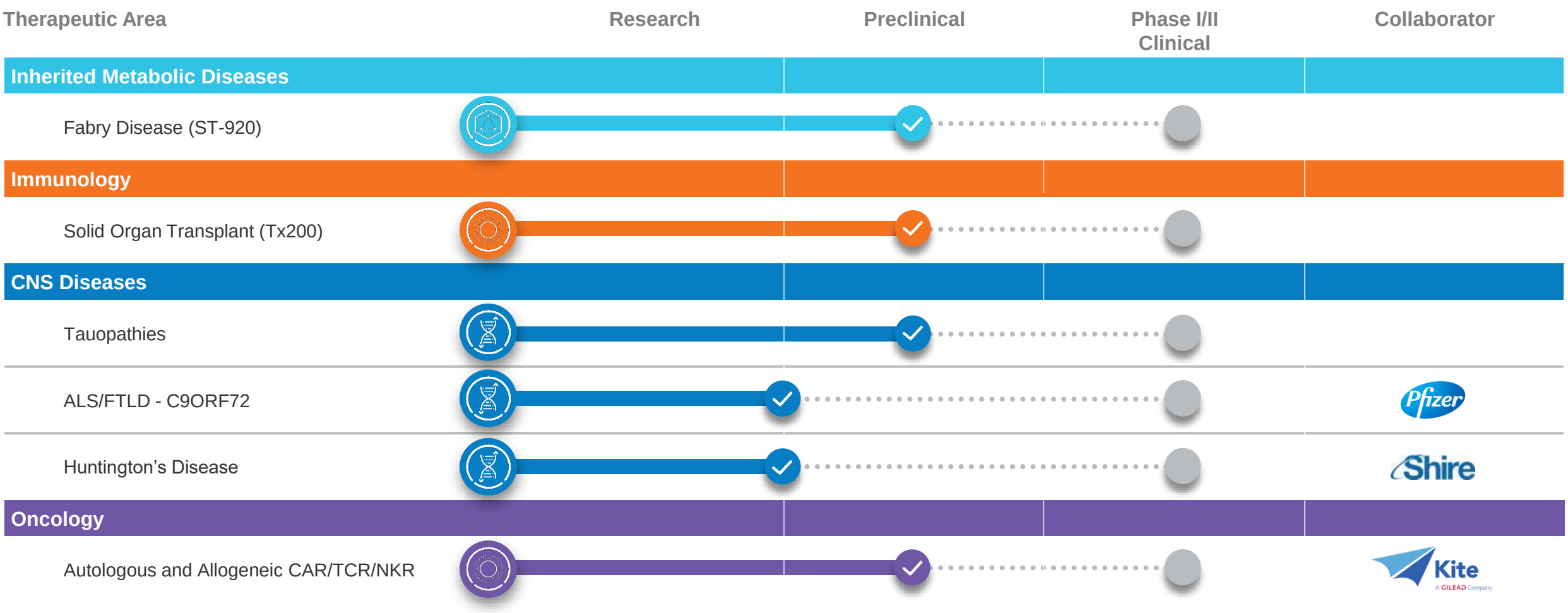
Preclinical Development

ST-920: Fabry disease

Tx200: Solid organ transplant

Gene-edited CAR-Tregs for immunology

Robust preclinical genomic medicine pipeline



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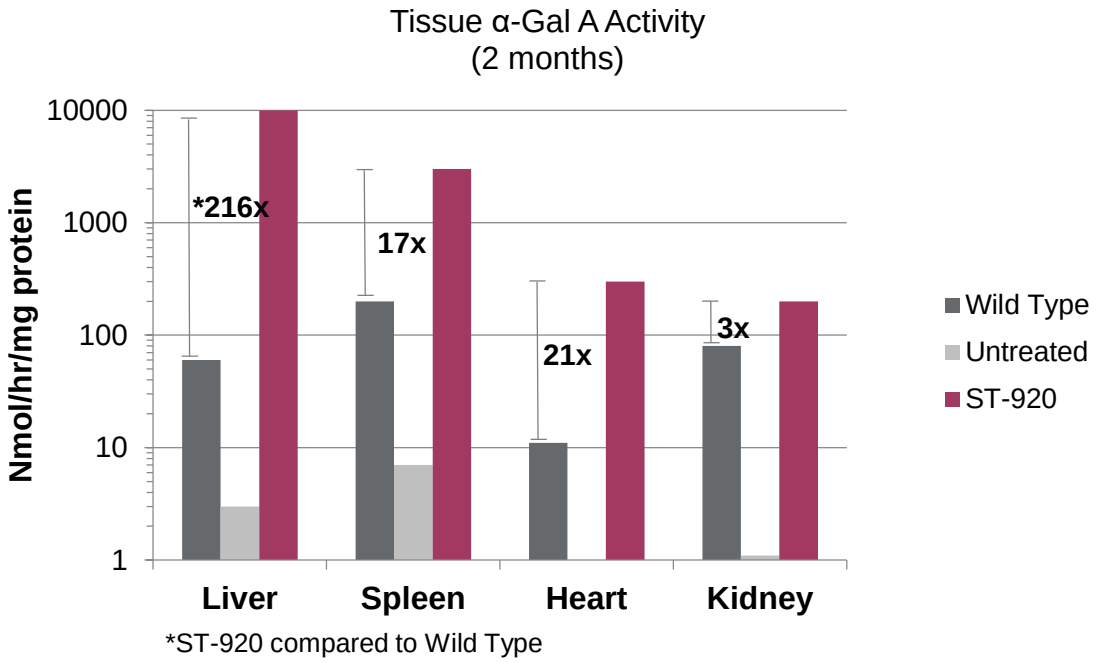
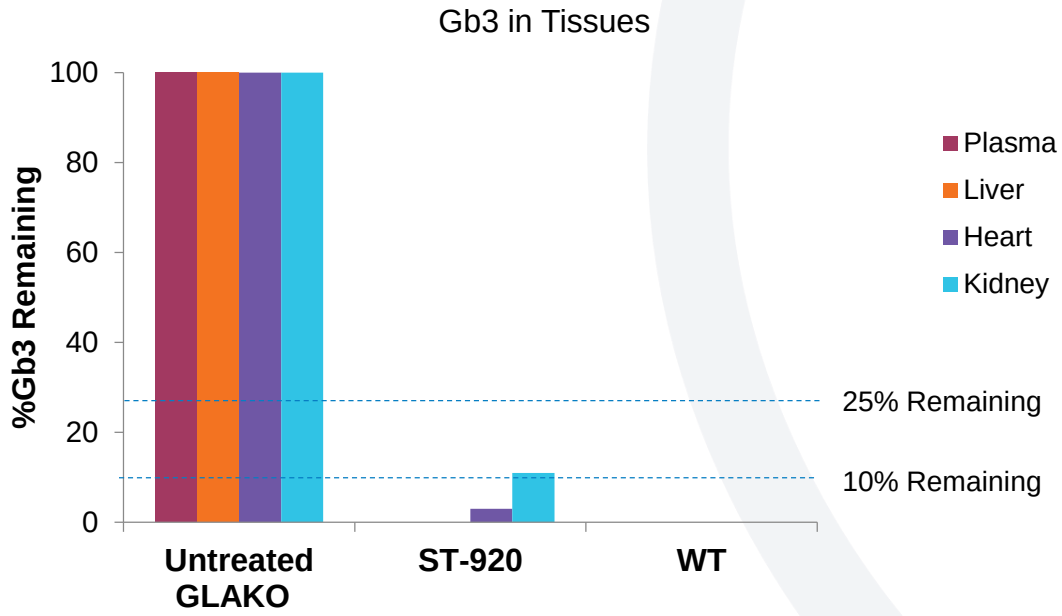
ST-920, gene therapy for Fabry disease

Sangamo's next inherited metabolic disease (IMD) asset

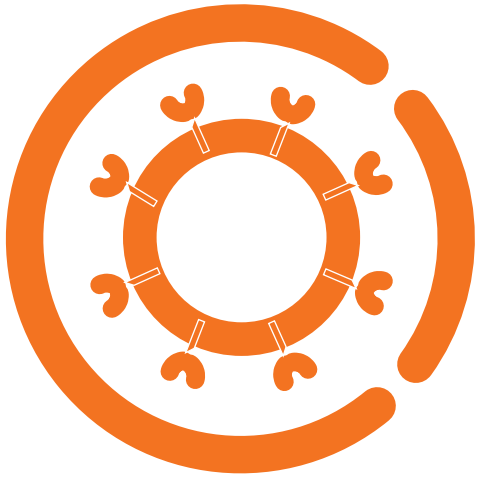
- ST-920 is a gene therapy designed to elevate α-Gal A enzyme
- 5,000 – 6,000 Fabry patients in US / EU; most diagnosed as adults
- Bi-weekly ERT infusions (standard of care) may not clear all substrate from secondary organs
- ST-920 clinical trial initiation expected in 2019





Sangamo's gene therapy resulted in strong expression of α-Gal A and Gb3 substrate reduction across tissue types in GLAKO murine model

CAR-Tregs have the potential to generate antigen and tissue specific cell therapy products for immunology



Cell Product Characteristics

Engineered CAR-Tregs

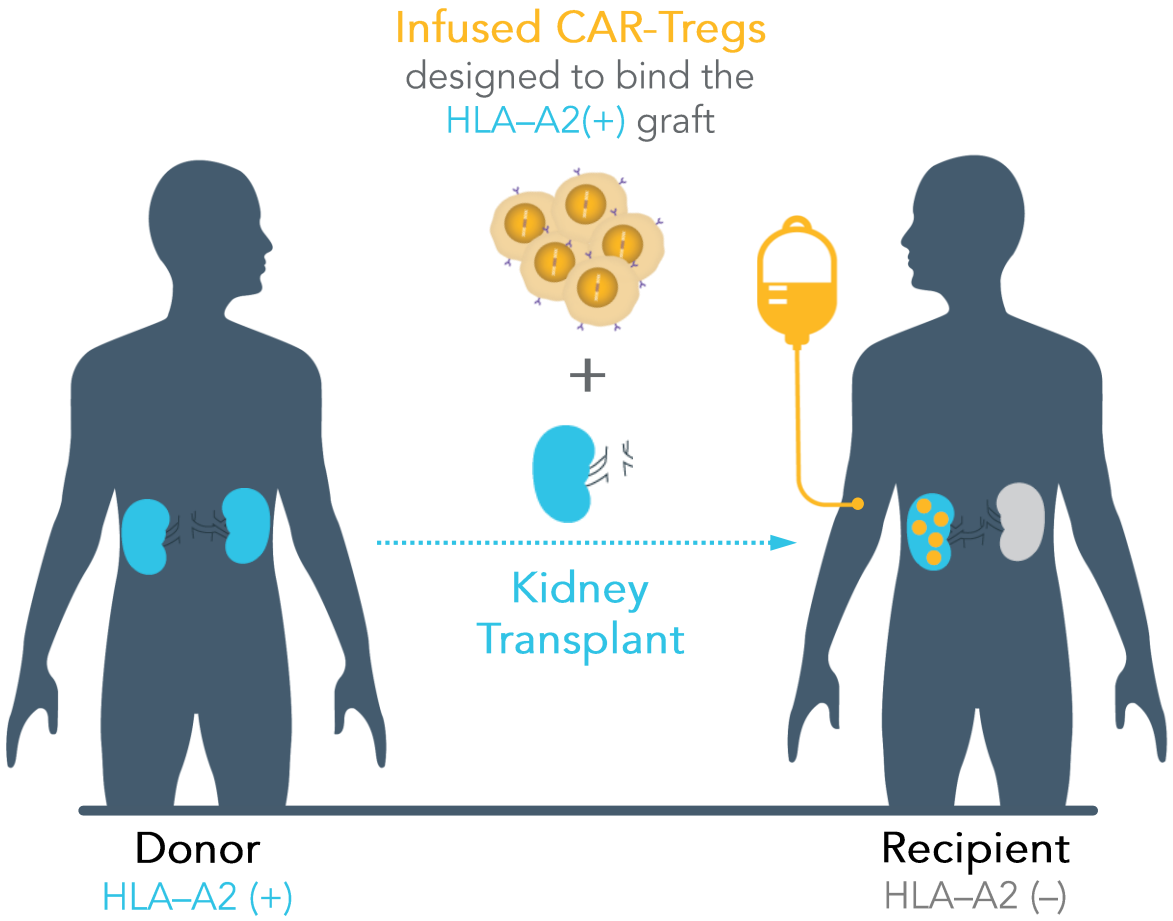
Antigen localized: tissue-specific activity

Antigen activated: better controlled cell product and dosing

Robust and scalable processes

Tx200: HLA-A2 CAR-Treg for solid organ transplant

Induction of site-specific immune tolerance



- HLA-A2 antigen on graft is recognized by CAR-Treg cells
- Activated CAR-Treg cells exert **site specific** suppressive function
- Goal: Achieve tolerance and long-term protection of graft

Clinical trial initiation expected in 2019

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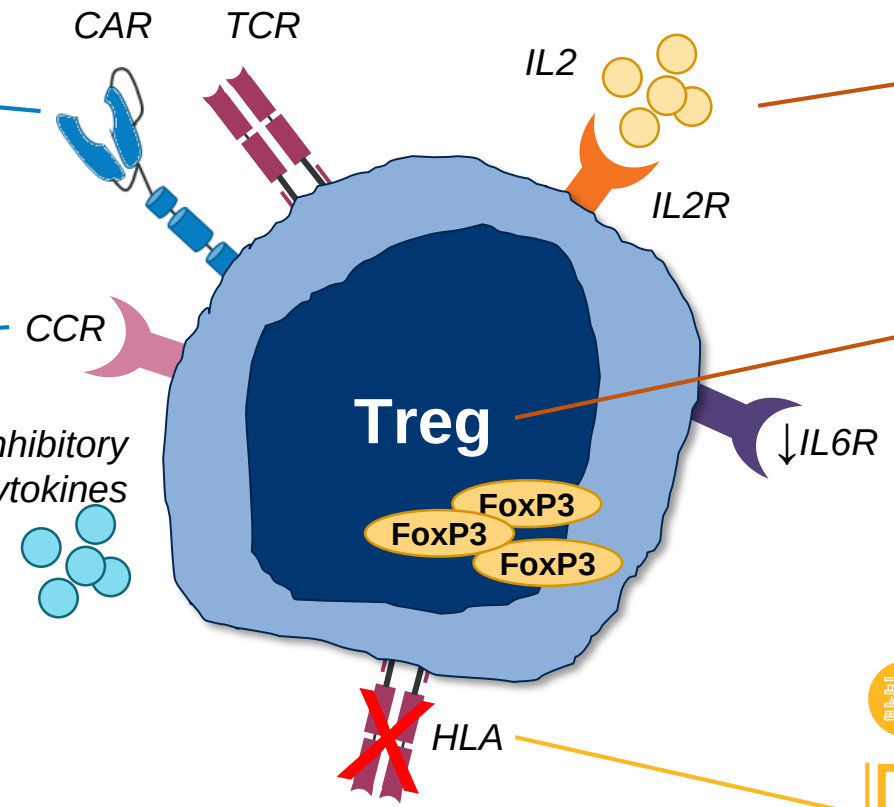
Sangamo plans to develop next generation CAR-Treg products with ZFN multiplex editing

Increase Efficacy + Potency

Target tissue-specific antigen(s) w/CAR

Promote localization to disease site(s) with chemokine receptors

Bolster immunosuppressive function



Increase Persistence + Stability

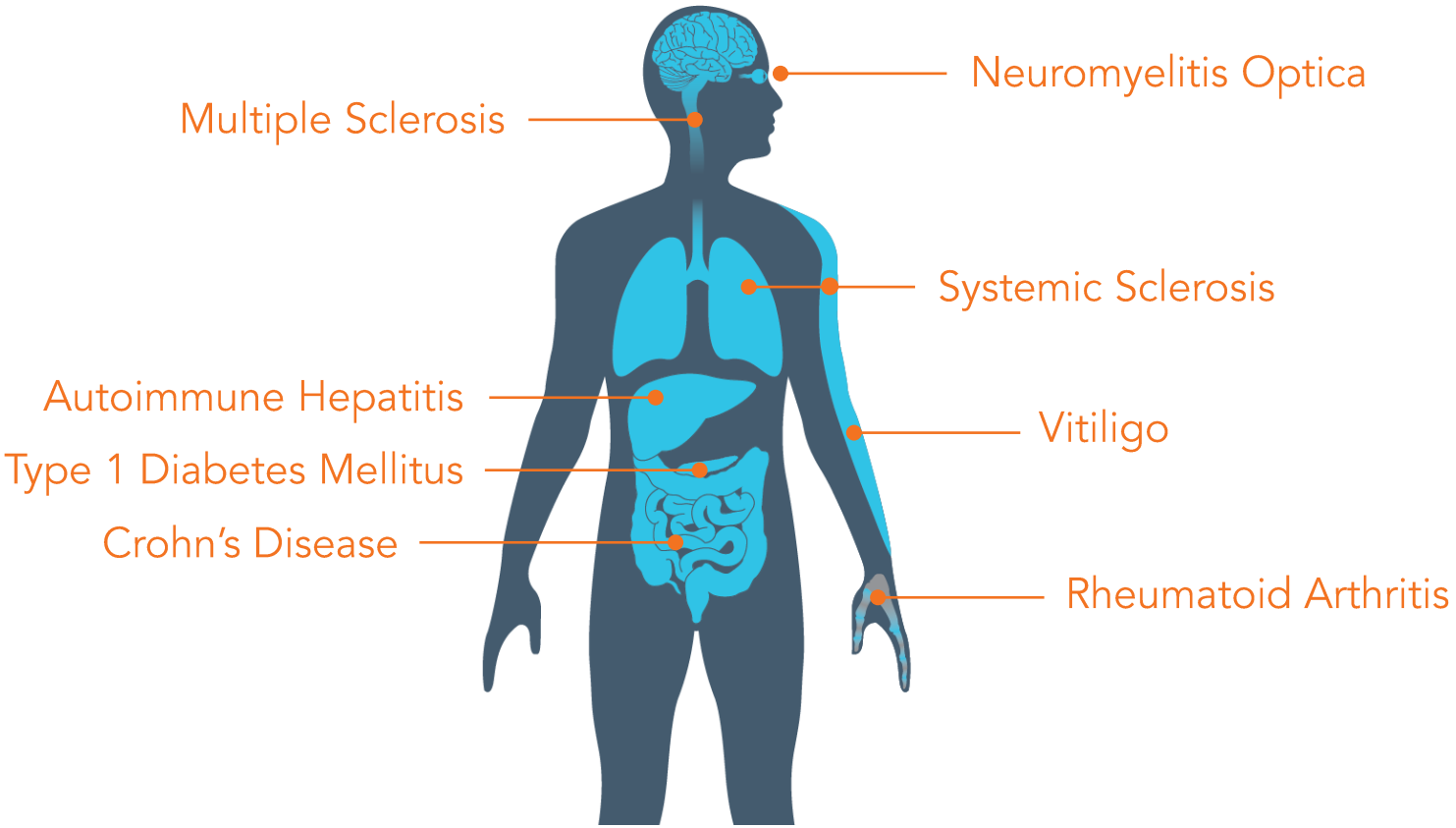
Increase Treg proliferation and durability

Improve CAR-Treg stability and safety

Off-The-Shelf Approach

Genetic engineering to allow allogeneic application of CAR-Tregs

Market opportunity for CAR-Tregs holds significant potential in autoimmune diseases



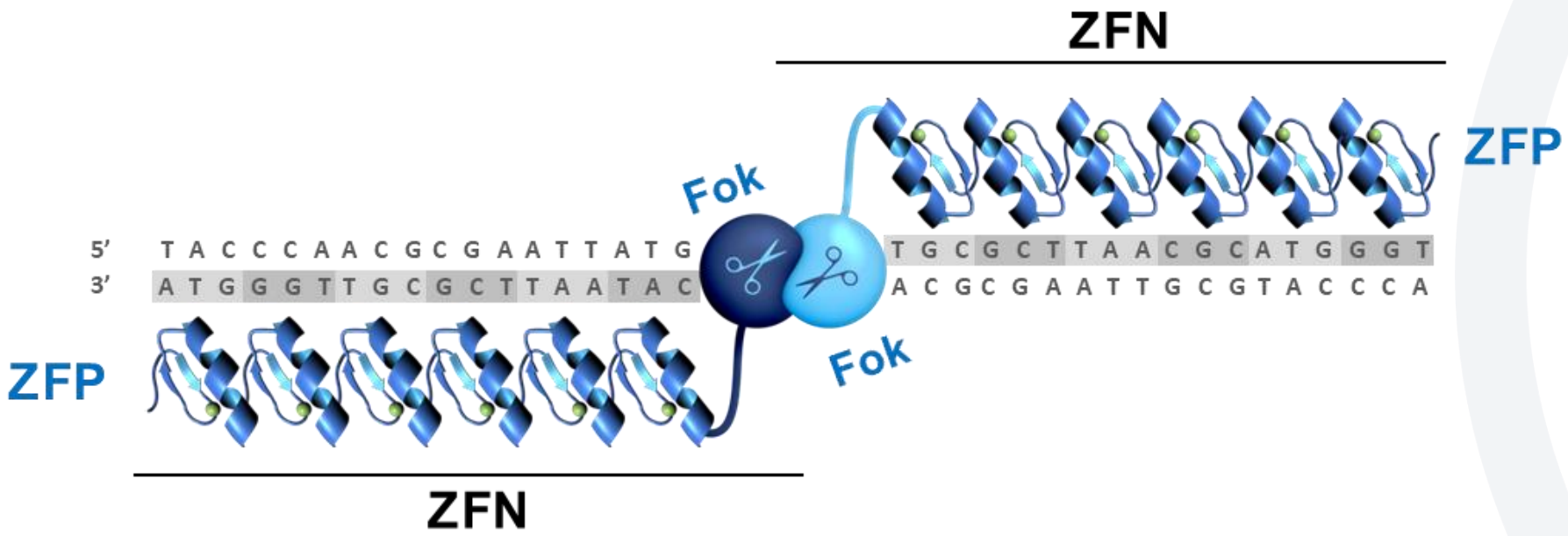
Several autoimmune diseases with large patient populations and high unmet need present significant market opportunities for CAR-Treg cell therapies

ZFP Technology

ZFN gene editing

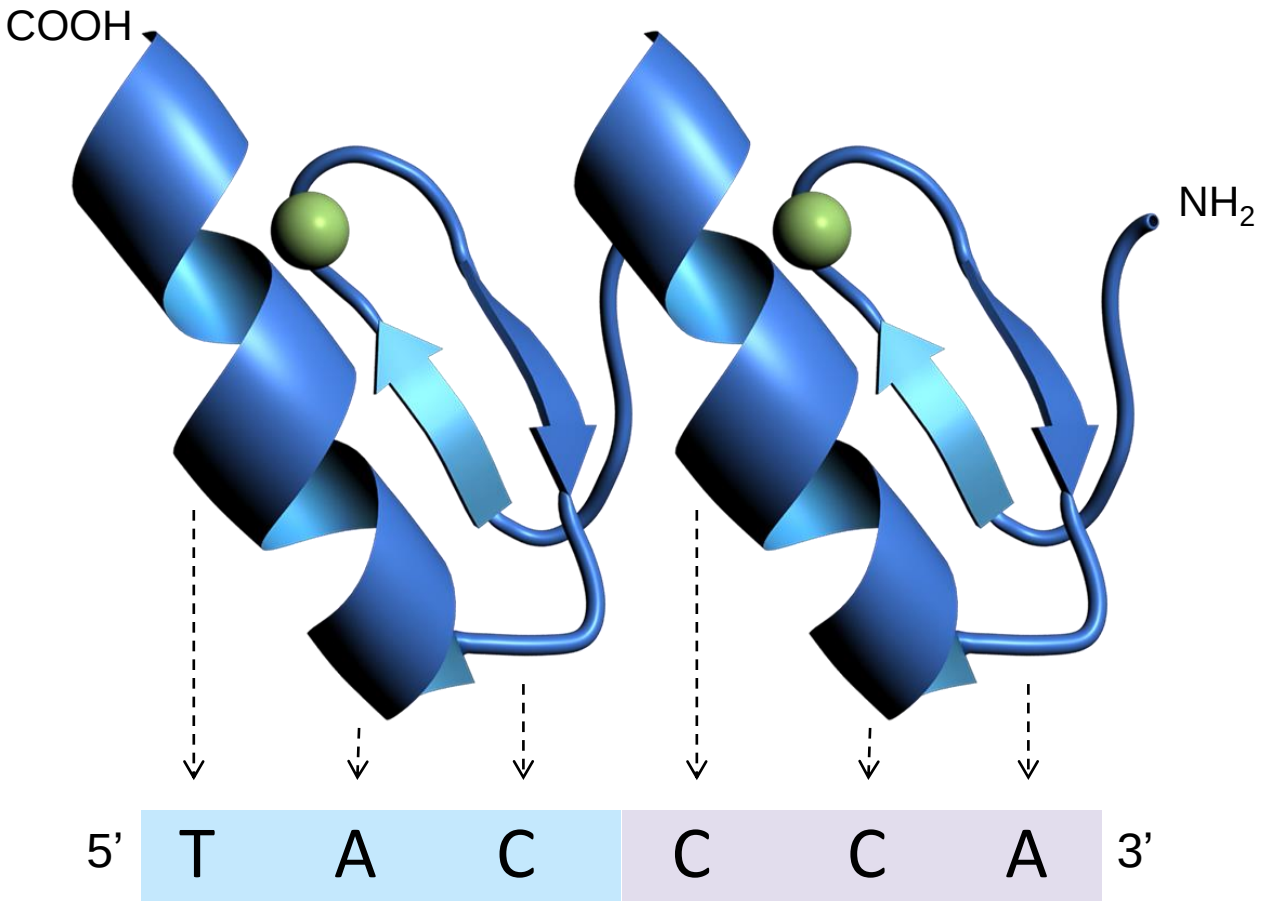
ZFP-TF gene regulation

ZFNs: The platform of choice for therapeutic gene editing

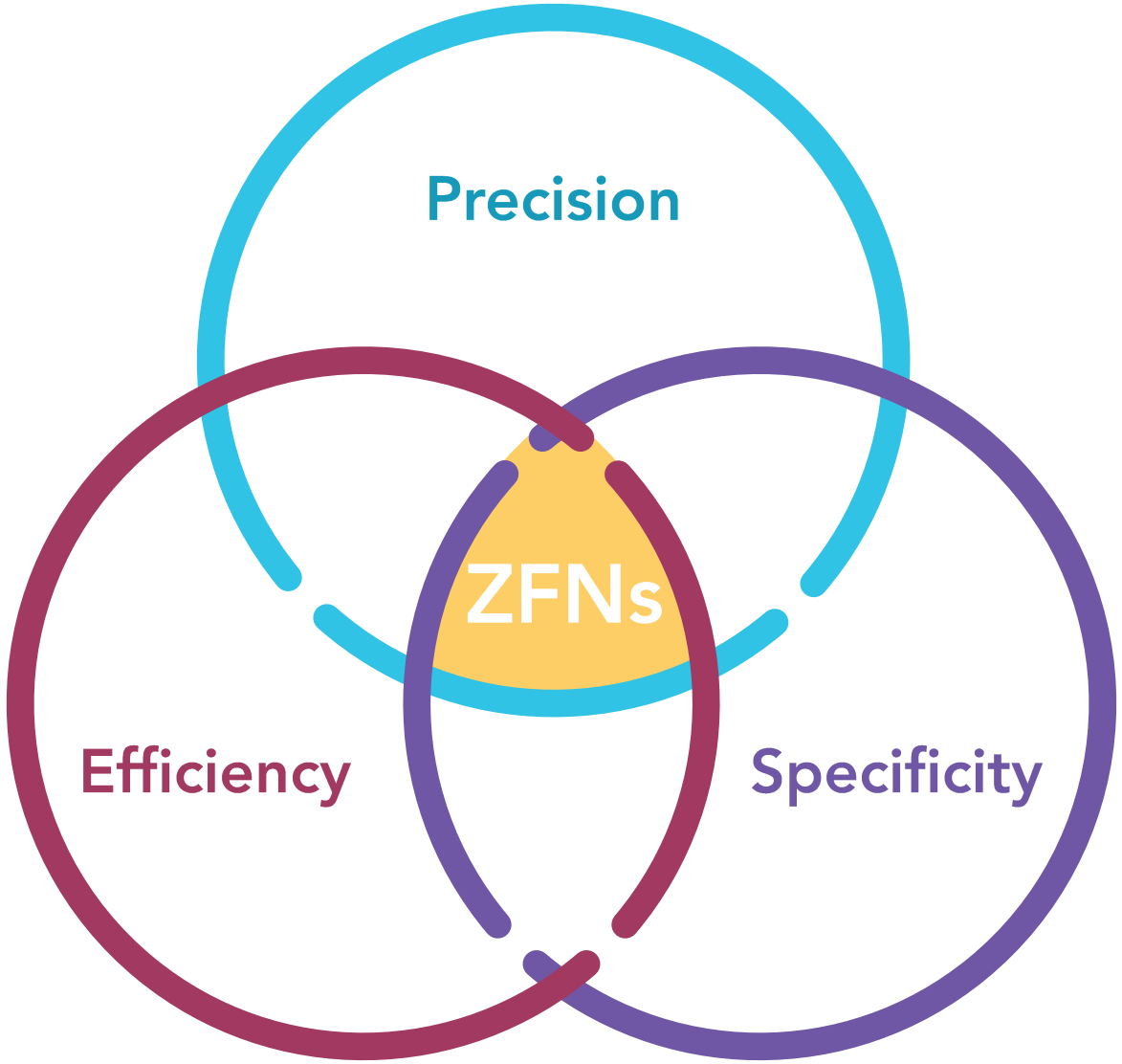


Sangamo's engineered zinc finger proteins

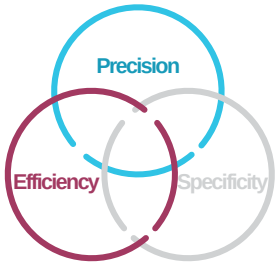
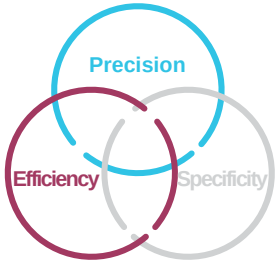
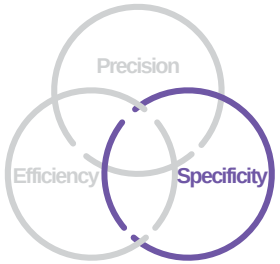
- A naturally occurring class of proteins which upregulate or downregulate gene expression
- Sangamo's ZFP library is derived from selected, designed and natural fingers



ZFNs: The platform of choice for therapeutic gene editing



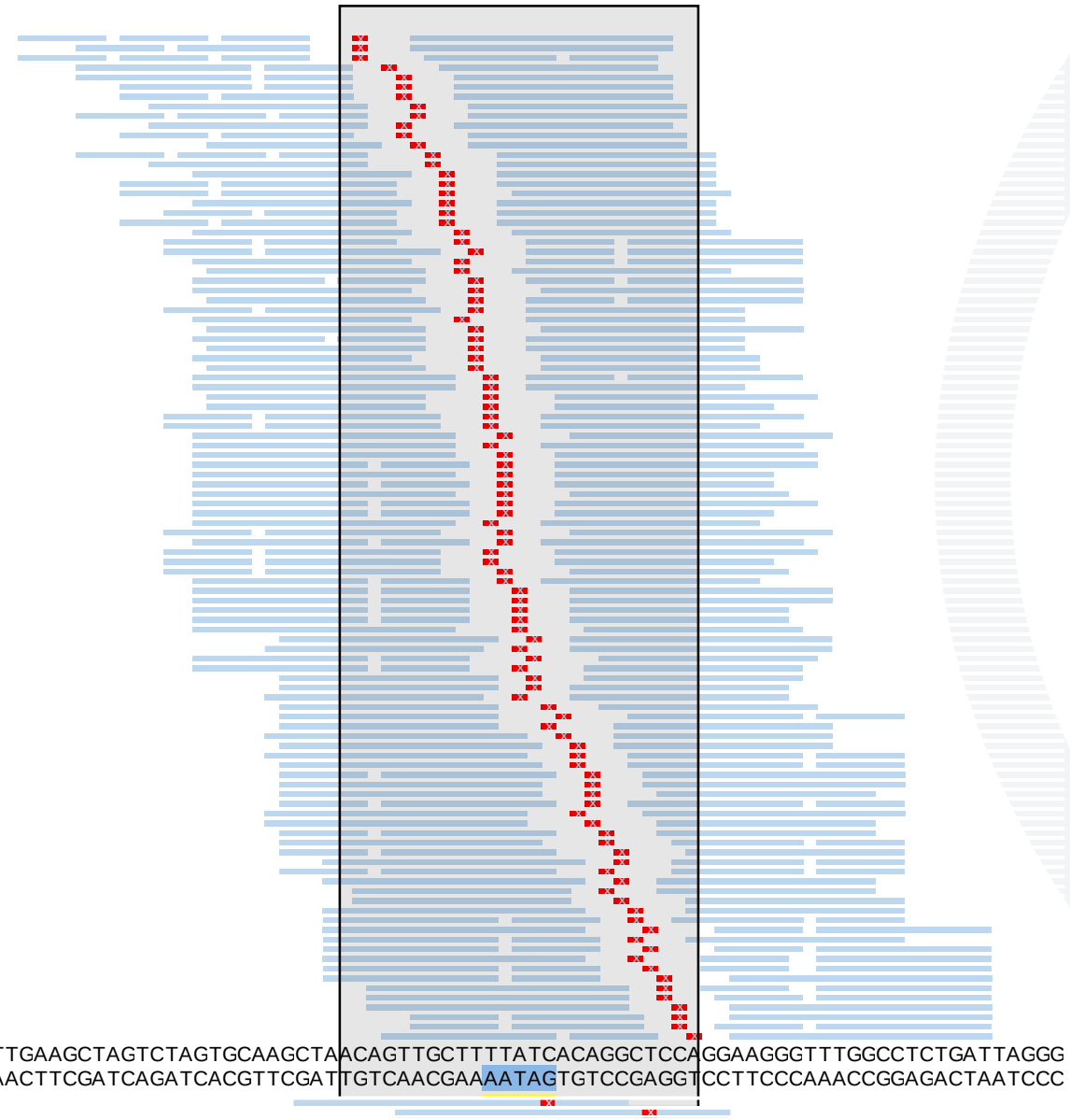
Recent innovations drive exceptional performance

	Innovation	Result
	**New linkers** for configuring DNA-binding modules	**300-fold** increase in design options for targeting any given sequence
	**New dimer architectures** yield higher modification activity	Increase DNA editing efficiency to as high as **99.5%**
	**Phosphate contact** tuning via replacement of key residues	Off-target cleavage undetectable (**>1000** fold reduction)

New architectures yield a dense array of ZFN designs for optimal on-target efficiency and maximum therapeutic effect

Example: Critical GATAA element in BCL11A

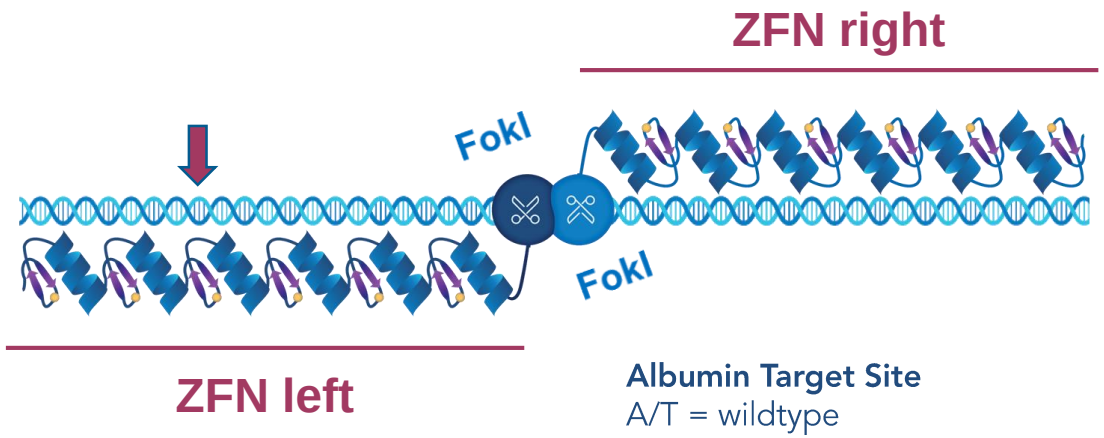
104 ZFN architectures available for targeting GATAA ± 10bp →



Two Cas9 designs →

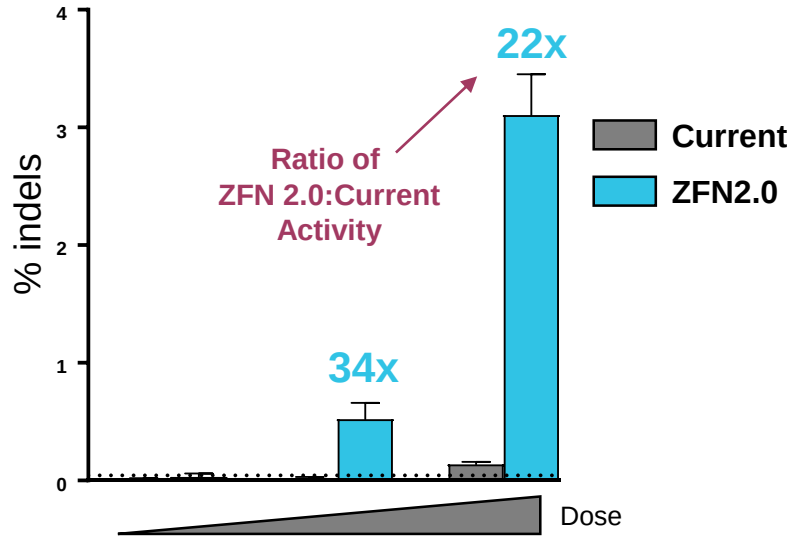
ZFN2.0 – Architectural enhancements create a next-generation ZFN for the albumin locus

Ability to cut both the wildtype and SNP target sequence



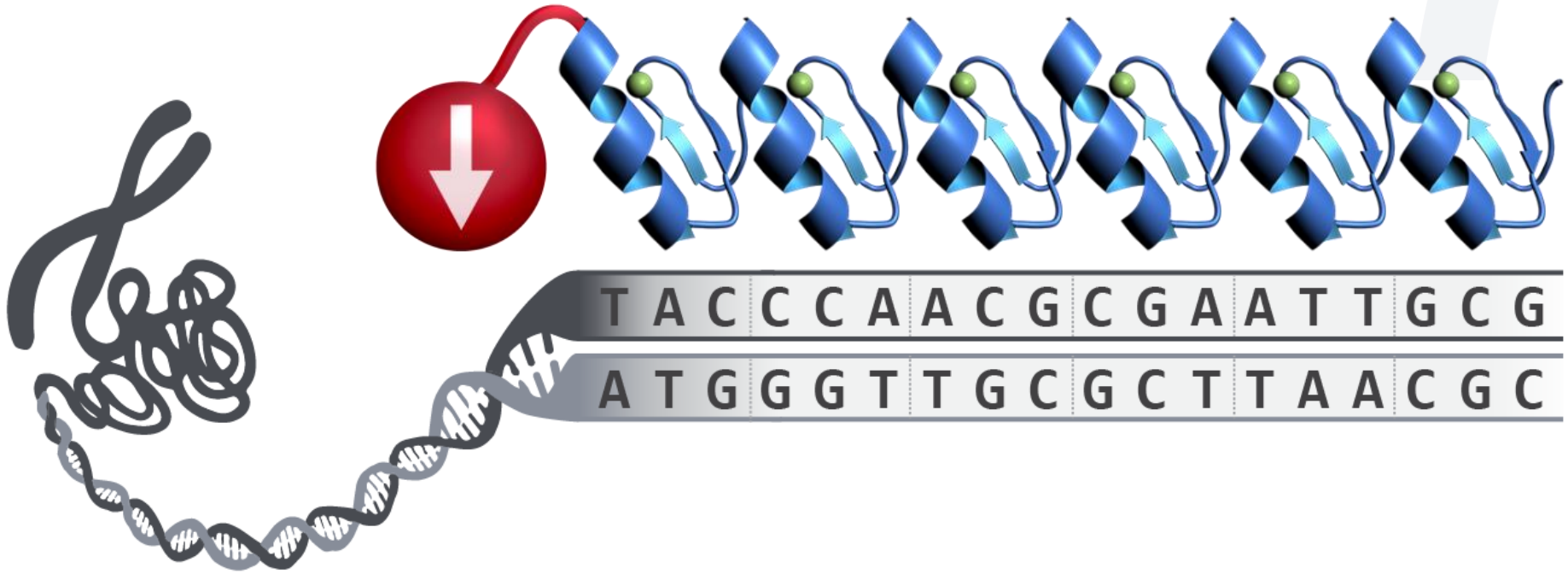
Albumin Target Site
A/T = wildtype
G/C = SNP

ZFN activity (% indels) increased 5-30x in primary human hepatocytes



‘Single-handed’ ZFP-Transcriptions Factors (ZFP-TFs) can be engineered to regulate any gene

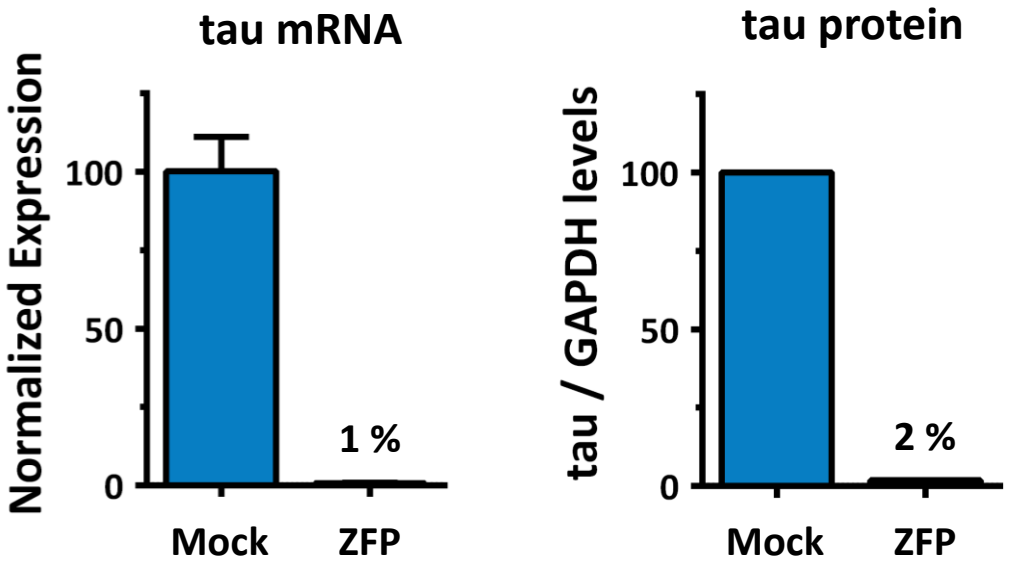
Sangamo is developing ZFP-TFs for CNS applications including tauopathies, Huntington’s disease[1], and C9ORF72-linked ALS and FTLD[2]



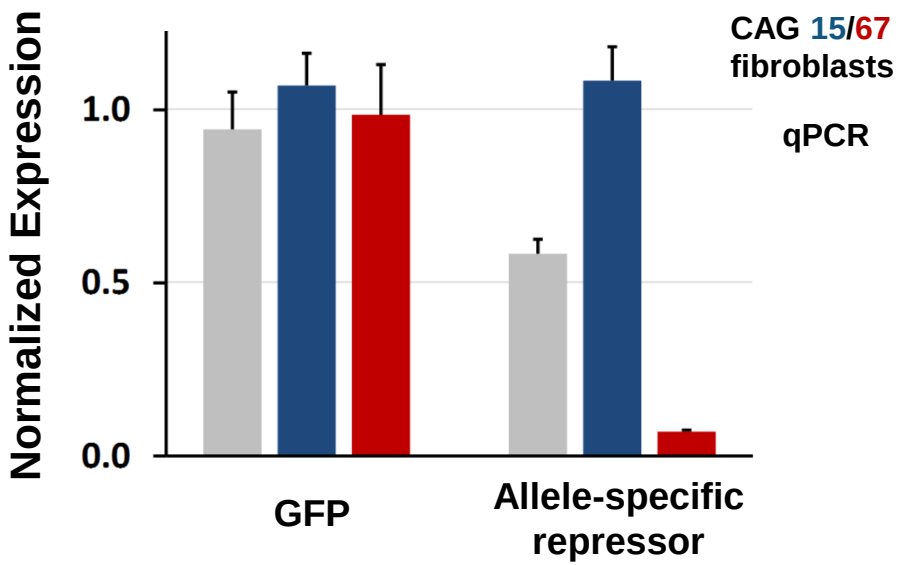
[1] In partnership with Shire
[2] In partnership with Pfizer

ZFP-TFs can be designed for pan-allelic or allele-specific gene repression

Pan-allelic repression:
Single-administration AAV ZFP-TF to lower all tau forms at the DNA level



Allele-specific repression
of mutant *HTT* gene in HD program[1]



[1] In partnership with Shire

In Closing

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- Flow of clinical data readouts in 2019 and 2020 following enrollment progress of last twelve months
- Strong balance sheet and 4 validating biopharma partnerships (Kite, Pfizer, Bioverativ, Shire)

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Thank you