Jefferies 2015 Global Healthcare Conference

Engineering Genetic Cures™

Edward Lanphier
President and CEO
Sangamo BioSciences, Inc.

June 1, 2015
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. 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 in our strategy, sufficiency of our cash resources, product development and commercialization of our products, clinical trials, revenues from existing and new collaborations, our research and development and other expenses, our operational and legal risks and any other statements that are not historical fact. Sangamo BioSciences, Inc. Annual Report on Form 10-K, recent and forthcoming Quarterly Reports on Form 10-Q, recent Current Reports on Forms 8-K and 8-K/A, and other SEC filings discuss some of the important risk factors that may affect our business, results of operations and financial condition. We undertake no obligation to revise or update publicly any forward-looking statements for any reason.
Sangamo BioSciences

- Focused on the development of a new class of human therapeutics that function at the DNA level with the goal of “Engineering Genetic Cures”

- Driven by multiple robust technology platforms
  - Zinc finger proteins (ZFPs) can be designed to bind to any DNA sequence with singular specificity
  - Targeted genome editing and gene regulation
  - Broad AAV and mRNA delivery capabilities, manufacturing and IP

- Broad commercial applications in research reagents, transgenic animals, agriculture, manufacturing and human therapeutics

- Significant partnerships and strong balance sheet

- Dominant intellectual property position
Toolbox for **Engineering Genetic Cures™**

Zinc Finger Protein

- **Gene Regulation Domain**
  - ZFP Transcription Factor (ZFP TF)
    - Gene Repression
      - No Transcription (No mRNA → No Protein)
      - Repress
  - Gene Activation
    - Transcription & Translation (More mRNA → More Protein)
    - Activate

- **Gene Editing Domain**
  - ZFP Nuclease (ZFN)
    - Knockout
    - Correct/Add
Sangamo has successfully monetized its ZFP technology platform

<table>
<thead>
<tr>
<th>Partners</th>
<th>Applications</th>
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<tbody>
<tr>
<td>Sangamo BioSciences</td>
<td>• Proprietary ZFP Therapeutics® Programs</td>
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</table>
| Biogen | • Hemoglobinopathies  
• Sickle Cell Disease  
• β–thalassemia |
| Shire | • Hemophilia A & B  
• Huntington’s Disease  
• Two Remaining Targets |
| Dow AgroSciences | • Plant Agriculture  
• Research Tools  
• Transgenic Animals  
• Protein Manufacturing |
Current Sangamo ZFP Therapeutic Development Programs

**ZFP Therapeutics® Strategies**

1. **In Vivo Strategy (AAV, mRNA)**
   - **Systemic (Liver)**
     - IVPRP Replacement Strategies
       - Hemophilia—Factors 7, 8, 9, 10 – Shire
       - Lysosomal Storage Disorders
     - mRNA ZFN Knockout/Knockdown Strategies
       - RNAi / Antisense Targets (e.g. PCSK9)
   - **Direct Tissue**
     - Brain—Huntington’s disease – Shire
     - Heart—Congestive Heart Failure
     - Lung—Cystic Fibrosis
     - Monogenic Diseases—Eye, Muscle, Heart, Lung

2. **Ex Vivo Strategy (mRNA)**
   - **Non-Stem Cells**
     - T-cells – HIV
     - T-cells – Oncology
   - **Stem Cells**
     - CD34+
       - Hemoglobinopathies (SCD, β-thalassemia) - BIIB
       - HIV/AIDS INR
       - Rare Diseases (Several)
     - Universal Donor Cells
       - Several

Blue = Partnered programs
Green = Active proprietary Sangamo programs
# Sangamo ZFP Therapeutics Pipeline

**In Vivo** Protein Replacement Platform (IVPRP)

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Sangamo’s *In Vivo* Protein Replacement Platform

**Broad Applications in Hemophilia and LSDs**

**Albumin Gene:**
- Very strong promoter

**Donor:**
- Homology
- Gene of Interest
- Homology

- **Hemophilia A**
  - $6B per year
- **Hemophilia B**
  - $1B per year
- **Hunter**
  - $500M per year
- **Hurler**
  - $200M per year
- **Gaucher**
  - $1B per year
- **Others…**
  - $Billions… per year

**Requires < 1% for normal levels**
Sangamo’s *In Vivo* Protein Replacement Platform

**Hemophilia – Factor VIII & Factor IX**

**Albumin Gene:**
- Very strong promoter
- ZFN

**Donor:**
- Homology
- Gene of Interest
- Homology

Hemophilia A: $6B per year
Hemophilia B: $1B per year
Hunter: $500M per year
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Gaucher: $1B per year
Others…: $Billions… per year
In Vivo Protein Replacement Platform is leverageable to numerous Lysosomal Storage Disorders:

- Hemophilia A: $6B per year
- Hemophilia B: $1B per year
- Hunter: $500M per year
- Hurler: $200M per year
- Gaucher: $1B per year
- Others: $Billions per year
Lysosomal Storage Diseases
Hurler and Hunter Syndromes

Glycoaminoglycans (GAGs)

Iduronate 2-Sulfatase (IDS) in MPS II

α-L-Iduronidase (IDUA) in MPS I

Accumulation of GAGs like Dermatan and Heparan Sulfates in the lysosome of all tissues leads to dysfunction in several tissues in MPS I/II patients

Modified after Neufeld and Muenzer 2001
IVPRP for Hunter syndrome results in supraphysiological hIDS expression and activity in **Wildtype** mice

**IDS activity**

Supraphysiological levels of hIDS activity can be detected in liver (primary tissue), plasma and secondary tissues like spleen.
IVPRP for Hunter syndrome results in supraphysiological hIDS activity in MPS II mice

**IDS activity**

High levels of hIDUA activity can be detected in liver (primary tissue), plasma and secondary tissues (spleen, kidney and lung) of MPS II mice.
IVPRP for Hunter syndrome corrects GAG accumulation in MPS II mice

Significant reduction of urinary and tissue GAG levels in MPS II mice after day 21/56
IVPRP for Hurler syndrome corrects GAG accumulation in MPS I mice

Significant reduction of urinary and tissue GAG levels in MPS I mice after day 21/60
IVPRP – Proof of Concept for LSDs

- Increased hIDUA and hIDS *expression detected for the entire study duration*
  - 4 – 12 weeks
  - Previous hFIX levels stable for >60 weeks

- Data provide a *proof of concept* for use of ZFN-mediated IVPRP for expression of therapeutic proteins to treat LSDs
# Sangamo ZFP Therapeutics Pipeline

## Hemoglobinopathies

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Hemoglobinopathies – Genetic Disorders of Hemoglobin

- **ZFPs can be used to cure Hemoglobinopathies**
  - *Ex vivo* mRNA ZFN-modification of the BCL11A gene locus in CD34\(^+\) stem cells enables a *curative* autologous bone marrow transplant in *both* \(\beta\)-thalassemia and SCD

“Building upon emerging science related to fetal hemoglobin regulation, we intend to develop Sangamo’s novel gene editing technology to create a single approach that has the potential to *functionally cure* both sickle cell disease and beta-thalassemia.”

Douglas E. Williams, Ph.D., EVP R&D, Biogen Idec
ZFN-Mediated BCL11A Knock Out

BCL11A KO increases fetal globin

Bcl11a (enhancer)

Erythroid Lineage

All Others

RBC

HbF

BCL11A KO increases fetal globin
"We propose the GWAS-identified enhancer of BCL11A as a particularly promising therapeutic target for genome engineering in the β-hemoglobinopathies. Disruption of this enhancer would impair BCL11A expression in erythroid precursors with resultant HbF derepression while sparing BCL11A expression in nonerythroid lineages."
ZFN-Mediated BCL11A Enhancer Knock Out

Equivalent effect on fetal globin expression as BCL11A KO approach

HSC

Erythroid Lineage

RBC

HbF

All Others
Rationale for BCL11A Enhancer Knock Out Approach vs. BCL11A Knock Out

- **Efficacy** – BCL11A Enhancer KO demonstrates robust upregulation of HbF expression

- **Precision** – BCL11A Enhancer KO only knocks out Bcl11a expression in Erythroid Cell Lineages, without altering HSPCs biology, including path to lymphoid development for B cells, T cells and Natural Killer (NK) Cells.

- **Persistence** – BCL11A Enhancer KO results in better long term engraftment of modified cells

- **Consolidation** – both the beta-thalassemia and sickle cell disease programs will utilize the BCL11A Enhancer KO approach

*BCL11A Enhancer KO strategy is a better approach for both beta-thalassemia and SCD*
New BCL11A Enhancer Approach to Hemoglobinopathies

• Unanimous Sangamo / Biogen JSC decision to consolidate approaches

• BCL11A Enhancer approach has numerous advantages
  – Efficacy
  – Precision
  – Persistence
  – Consolidation

BCL11A Enhancer β-thal Phase 1 will begin in 2016
BCL11A Enhancer SCD IND remains on track for 2016
# Sangamo ZFP Therapeutics Pipeline

**SB-728-T for HIV/AIDS**

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SB-728-T: ZFN-mediated CCR5 Knockout in CD4+ T-cells

**Acute effect on viral load (VL)**

**Hypothesis**
Maximizing biallelic CD4 / CD8 CCR5 KO provides functional control of HIV

**Reservoir Reduction**

**SB-728mR-1401 (Phase 2)**
- CTX Pre-treatment
- mRNA-based delivery allows repeat dosing

---

**Subject 04-502**
Days from Baseline
0 60 120 180 240 300 360 420 480 540 600 660 720
Viral load Copies/mL
Absolute CD4 Count
Pentamer Duplication/L
Viral Load
Set Point

---

**Log Change in HIV DNA per 10⁶ PBMC**
Linear Regression -3 years post infusion

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**Sangamo BioSciences**
Phase 2 HIV Program Update at ASGCT 2015

Updated Patient Data from SB-728-1101 Cohort 3*

Abstract 25: A Dose Escalation Study of Cyclophosphamide (CTX) to Enhance SB-728-T Engraftment

- Patient maintains functional control of viral load
- Remains on prolonged TI

- Approx. 1 log decrease in viral load following delayed onset of viremia
- Remains on prolonged TI
HIV Program Update at ASGCT 2015

Updated Analysis from SB-728-902 Cohorts 1-3
Adoptive Transfer of ZFN Mediated CCR5 Modified Engraftment of HIV Resistant T Memory Stem Cells and Decrease in Size of Latent Reservoir

**Key Takeaways**

- Cohorts 1-3 → 9 INRs received 1 infusion of SB-728-T
  - 10-30 billion autologous CCR5-modified CD4 cells
- Immune reconstitution in all 9 subjects at >3 years post-infusion
- T_{SCM} subset of CD4 T-cells showed highest levels of ZFN modification
- Expansion of T_{SCM} subset of CD4 T-cells correlates with decay of the HIV reservoir

Linear regression analysis between increased T_{SCM} and reservoir decay

\[ p = 0.042 \]
\[ R^2 = 0.526 \]
## Sangamo ZFP Therapeutics Pipeline

### Huntington’s Disease

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Huntington’s Disease: A fatal neurodegenerative monogenic disease

- Huntington’s disease is caused by a mutation in the *huntingtin* gene (multiple CAG repeats), causing production of a toxic protein.

HD occurs when the Huntingtin gene has >35 CAG repeats.

- How can ZFPs be used to treat Huntington’s disease?

ZFP TF selectively represses (↓) the mutant *huntingtin* gene → decreased production of mutant Huntingtin protein.
# Allele-specific repression in Huntington’s disease patient cell lines

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<th>Cell Line:</th>
<th>Patient #1</th>
<th>Patient #2</th>
<th>Patient #3</th>
<th>Patient #4</th>
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### Normalized Scaled Expression

- **Control**
  - **WT**: 1.0
  - **Mut**: 0.0

- **Patient Cell Line 2**
  - **WT**: 1.0
  - **Mut**: 0.5

- **Patient Cell Line 3**
  - **WT**: 1.0
  - **Mut**: 0.0

- **Patient Cell Line 4**
  - **WT**: 1.0
  - **Mut**: 0.27

- **Patient Cell Line 5**
  - **WT**: 1.0
  - **Mut**: 0.26
Huntington’s Disease Program
Data Summary

**ZFP TF treatment leads to a reversal of Huntington’s disease pathology** in patient-derived cells and mouse models of disease across the full range of disease mutations

- In HD patient-derived cell lines:
  - 90% repression of mutant Htt alleles with minimal repression of normal Htt alleles

- In mouse models:
  - Prevention of aggregation and clearance of Htt protein aggregates
  - Increased expression of markers associated with *medium spiny neurons* (cell type normally lost in HD patients), demonstrating ZFP-mediated protection of critical cell type
  - Statistically significant *reduction in and reversal of physical indications of the disease* e.g. motor defects / “clasping”
### Sangamo ZFP Therapeutic® Pipeline
8 New INDs and Multiple Clinical Read-outs by YE2016

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<td>Multiple</td>
<td>Lysosomal Storage Disorders</td>
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<tr>
<td>Multiple</td>
<td>Other Monogenic Diseases</td>
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2015 Financial Guidance

- **Cash and Investments**: at least $180 million at the end of 2015 – inclusive of research funding from Shire and Biogen, but exclusive of funds arising from additional new collaborations or partnerships, or other new sources.

- **Revenues**: in the range of $60 to $70 million - inclusive of research funding from Shire and Biogen.

- **Operating Expenses**: in the range of $100 to $110 million.
Summary

• Sangamo’s ZFP technology is a robust platform for genome editing and gene regulation

• Focused on the development of a new class of human therapeutics that function at the DNA level with the goal of “Engineering Genetic Cures”

• Major partnerships and sufficient cash to move all programs through value inflection points

• Our strategy creates significant near term value and mitigates risk via diversification and leverage
  – Business model – partnerships and proprietary programs
  – Diverse therapeutic product development strategies
  – Variety of addressable and well validated targets
  – Balance sheet strength