Dr Geoff Nichol
Sangamo BioSciences, USA
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<td>Dr Geoffrey Nichol</td>
<td>Is an employee and US Section 16 Officer at Sangamo BioSciences; receives salary and holds shares and share options in Sangamo BioSciences</td>
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<tr>
<td>Date</td>
<td>October 2014</td>
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CCR5 knockout gene therapy trials

Geoff Nichol MB ChB FRACP
Executive Vice-President, R&D
Sangamo BioSciences

BHIVA Autumn Conference
2014
HIV – an infection and an immune system disease

- Primary infection of CD4 T cells
- Damage to mucosal barriers
- Chronic inflammatory state
- Loss of CD4 T cells by direct cytopathic and bystander mechanisms
- “Helpless” activation of CD8 T cells fails to clear infection

Reservoir established in memory CD4 cells

ART
Blocking the narrow door
A lesson from Nature – the CCR5Δ32 mutation

Individuals homozygous for the CCR5Δ32 allele are highly resistant to HIV-1 infection
“Berlin patient”

Long-Term Control of HIV by CCR5 Delta32/Delta32 Stem-Cell Transplantation

Gero Hütter, M.D., Daniel Nowak, M.D., Maximilian Mossner, B.S., Susanne Ganepola, M.D., Arne Müßig, M.D., Kristina Allers, Ph.D., Thomas Schneider, M.D., Ph.D., Jörg Hofmann, Ph.D., Claudia Kücherer, M.D., Olga Blau, M.D., Igor W. Blau, M.D., Wolf K. Hofmann, M.D., and Eckhard Thiel, M.D.

CCR5Δ32 donor

“Berlin patient”
SB-728-T – the product
ZFNs cause targeted gene disruption

CCR5 target

NHEJ error-prone repair

gene disruption
Zinc finger nucleases (ZFNs) “Designer restriction enzyme”

- Delivered with a non-integrating, replication-deficient, chimeric adenoviral 5/35 vector or mRNA electroporation
SB-728-T: Zinc finger nuclease driven CCR5 modified autologous CD4\(^+\) T-cells

- **SB-728 CCR5 ZFNs**
  - Expand, formulate and test
  - Median CCR5 modification ~25%
- **ZFN**
- **Enrich CD4+**
- **Apheresis**
- **SB-728-T**
- **Infusion**
  - Single Infusion of: 10-30 billion SB-728-T
  - CCR5 gene disruption
The infused product (SB-728-T) contains T-cells with a stem cell-like phenotype.
How we assay for CCR5 deletions

- ZFN mediated gene disruption generate a diverse array of short insertions and deletions to the targeted CCR5 locus.

```
INSERTIONS:
TTTGGGGCAACATGCTGGTCTCTGTATCCTGTATTTACATGGCATAAAGCTGAGGACATGACTGACATCTAGCTTGCTGCTG
TTTGGGGCAACATGCTGGTCTCTGTATCCTGTATTTACATGGCATAAAGCTGAGGACATGACTGACATCTAGCTTGCTGCTG
TTTGGGGCAACATGCTGGTCTCTGTATCCTGTATTTACATGGCATAAAGCTGAGGACATGACTGACATCTAGCTTGCT
TTTGGGGCAACATGCTGGTCTCTGTATCCTGTATTTACATGGCATAAAGCTGAGGACATGACTGACATCTAGCTTGCTGCTG
TTTGGGGCAACATGCTGGTCTCTGTATCCTGTATTTACATGGCATAAAGCTGAGGACATGACTGACATCTAGCTTGCTGCTG
TTTGGGGCAACATGCTGGTCTCTGTATCCTGTATTTACATGGCATAAAGCTGAGGACATGACTGACATCTAGCTTGCTGCTG
```

- Most frequently is a 5-bp insertion or “Pentamer Duplication” (CTGAT)
  - Approximately 16 to 39% (mean = 23%) of CCR5 allele disruptions

- In clonal studies bi-allelic disruption occurs in about 1/3 of disrupted cells – total CCR5 knockout
  - 2/3 if one allele already has the Δ32 mutation
## SB-728 – key exploratory clinical studies

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SB-728-T – pharmacokinetics and pharmacodynamics
**SB-728 – key exploratory clinical studies**

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Long-term CD4 T-cell reconstitution post SB-728-T

Infusion of CCR5-disrupted cells led to a sustained significant increase in CD4 T cell counts (mean of 103 cells/µL at 12 Months)
Long-term engraftment of CCR5 modified cells

![Graph showing the median and mean pentamer duplication per µL for 1X10^6, 2X10^6, and 3X10^6 Cells across different visits.](image)

- **1X10^6 Cells**: 102, 103, 104
- **2X10^6 Cells**: 201, 203, 302
- **3X10^6 Cells**: 303, 304, 305

Visits:
- Baseline
- Day 1
- Day 7
- Day 14
- Day 21
- Month 2
- Month 3
- Month 4
- Month 5
- Month 6
- Month 7
- Month 8
- Month 9
- Month 10
- Month 11
- Month 12
- LTFU M3
- LTFU M6
- LTFU M9
- LTFU M12
- LTFU M18
- LTFU M24

Pentamer Duplication per µL:
- Median
- Mean

10/9/2014
CCR5 modified T-memory stem cells expand and persist up to 12 months

TSCM

(CD45RO\text{low} \cdot RA\text{low} \cdot CCR7+CD27+CD95+)

- Median fold expansion of CCR5-modified cells relative to amount infused was 20.7 at Month 12
- In contrast, fold expansion in modified CM and EM were ~ 3 fold and < 1 fold
CCR5 gene modification level is maintained for three years in the TSCM fractions
SB-728-T traffics to the rectal mucosa

Percent CCR5 Disruption in Mucosal CD4

Time
Baseline 2-3 Weeks 6-12 Weeks 16-24 Weeks 36-48 Weeks

N=19  N=11  N=12  N=9  N=3

Outliers
75 %
25 %
Mean
Median
Std. Error
High levels of monocyte activation (DRhiCD86hiCD40hi) in HIV+ subjects at baseline

Classical Monocyte CD14++ CD16-

Inflammatory Monocyte CD14+ CD16+

\[ p = 0.0066 \]

\[ p < 0.0001 \]
Baseline levels of monocyte activation inversely correlate with levels of CCR5-modified cell engraftment

\[ r = -0.7505 \quad p = 0.0198 \]

Estimated CCR5-modified PBMC counts at day 21 relative to input

% HLA-DR\text{hi}CD86\text{hi} in CD14+ monocytes at BL

Estimated CCR5-modified PBMC counts at day 21 relative to input
Baseline levels of monocyte activation inversely correlate with levels of CD4 T-cell reconstitution.

**CD4 Persistence after 1 Year**

\[ \rho r = -0.8034 \quad p = 0.0091 \]

**CD4 Persistence after 3 Years**

\[ r = -0.8633 \quad p = 0.0027 \]
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Higher peak CD4 T-cell reconstitution and engraftment of SB-728-T is observed at a dose of 1 gm/m2 CTX.

**CD4 change from baseline**

**Pentamer duplication**
SB-728-T – effects on viral load during ART interruption
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First-in-human study of CCR5 KO published in NEJM
6 March 2014

Gene Editing of CCR5 in Autologous CD4 T Cells of Persons Infected with HIV

Pablo Tebas, M.D., David Stein, M.D., Winson W. Tang, M.D., Ian Frank, M.D., Shelley Q. Wang, M.D., Gary Lee, Ph.D., S. Kaye Spratt, Ph.D., Richard T. Surosky, Ph.D., Martin A. Giedlin, Ph.D., Geoff Nichol, M.D., Michael C. Holmes, Ph.D., Philip D. Gregory, Ph.D., Dale G. Ando, M.D., Michael Kalos, Ph.D., Ronald G. Collman, M.D., Gwendolyn Binder-Scholl, Ph.D., Gabriela Plesa, M.D., Ph.D., Wei-Ting Hwang, Ph.D., Bruce L. Levine, Ph.D., and Carl H. June, M.D.

- First genome edited therapy tested in man (ZFN modified CD4+ T cells)
- Infusions generally safe and well tolerated
- Marked increases in total CD4+ T cell levels
- Traffic to GALT (key battle ground of HIV infection)
- Modified cells show a selective survival advantage during ARD interruption
- One subject controlled viral load to below levels of detection prior to reinstating ARD
HIV viral load during treatment interruptions.

Tebas et al, 2014
Changes in VL correlate with levels of biallelic modification
6-week bi-allelic engraftment following Cytoxan - approaching threshold?

Estimated mean Biallelic Modification per µL
During treatment interruption

Log of Viral Load Drop from peak to end of Treatment Interruption

 rho = - 0.55, P= 0.017

*Delta 32 Subject
## SB-728 – key exploratory clinical studies

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Sustained functional control of viral load for more than one year

- Subject 04-502 (SB-728-902 Cohort 5)
  - Viral load controlled for more than 59 weeks (<500 VL copies/mL)
  - Subject remains off ART
  - Durable functional control achieved
# SB-728 – key exploratory clinical studies

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SB-728-1101: Viral load decreases from peak during TI

Four subjects with extended TI with VL <10,000 copies and CD4>500

Subjects

Log Viral load drop from peak to end of TI

Cohort (CTX)
- Cohort 1 (0.1 g/m²)
- Cohort 2 (0.5 g/m²)
- Cohort 3 (1.0 g/m²)
- Cohort 4 (2.0 g/m²)
- Cohort 5 (1.5 g/m²)

* Subject Continuing TI

TI= Treatment interruption
Red box: Δ32 Heterozygote
# Viral Load: Copies/mL
Meaningful reductions in VL seen during TI in Cytoxan-treated subjects

Subject 04-019 (SB-728-1101)
- CTX dose – 1.0 gm/m^2
- >2 log reduction in Viral load (VL)
- Sustained control for 39 weeks
- Subject remains off ART

Subject 01-060 (SB-728-1101)
- CTX dose – 1.5 gm/m^2
- >2 log reduction in VL
- Subject remains off ART
SB-728-T – effects on the HIV reservoir
HIV reservoir

- Laid down at time of initial infection
- HIV DNA integrated within CD4 memory cells
- Reservoir size driven by time from infection to start of ART
- Highly stable on chronic ART
- Maintenance is a dynamic process
  - Activation cycling of CD4 reservoir cells creates a target for immunotherapies

Barouch and Deeks, Science 345, 169 (2014)
## SB-728 – key exploratory clinical studies

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Step 1. “Digitize” sample into 20,000 droplets. Effectively reducing level of background gDNA

Step 2. Run PCR to endpoint. Quantification is no longer dependent upon PCR kinetics

Step 3. Fluorescent analysis drop by drop (yes or no). Copy number is calculated by Poisson distribution
Reduction of PBMC HIV DNA (ddPCR) observed in SB-728-T treated subjects (Median 0.9 log decrease at Month 36)
Raltegravir/maraviroc +/- IL-7 - increased CD4 counts **BUT** increased HIV pro-viral DNA

**Figure 3: Median change from baseline in CD4 counts**

**Figure 2: Median change from baseline in HIV DNA in the PBMCs**

Katlama et al CROI 2013
CD8 T-cells responsive to HIV GAG post-infusion correlate with the decay of CD4 T-cells harboring integrated HIV DNA

\[ \text{Spearman } r = 0.8857 \quad p = 0.033 \]

\[ \text{Spearman } r = 0.8286 \quad p = 0.058 \]

Reservoir Reduction
Ratio [Integrated HIV DNA (copies/10e6 cells)] BL/M36
Gene therapy for HIV
SB-728-T - Next steps

- IND for mRNA electroporation of CD4 cells is open – SB-728mR-T
  - Allows potential for retreatment
- Key proof-of-concept Phase II study commencing:
  - Optimal subject population
    - Short time from initial infection to ART
    - Favorable macrophage inflammatory profile
  - Optimal Cytoxan dose (1 g/m²)
  - 9 subjects in 2 cohorts will receive multiple doses of SB-728-mR-T
    - Cohort 1: SB-728-mR-T infusions of 2 equal doses 14 days apart
    - Cohort 2: SB-728-mR-T infusions of 3 equal doses 14 days apart
  - Objective: define proportion of subjects with functional control outcome
- Reservoir assay work continues
Using ZFNs to protect CD34+ HSCs
SB-728 CD34+ HSCs in HIV
ZFN-treated HSC mice control R5-tropic HIV-1

blood

HIV-1 RNA

Weeks Post Infection

untreated
ZFN

HIV-1_{\text{BaL}}

IND in 2014 in collaboration with City of Hope and California Institute of Regenerative Medicine
Summary and conclusions

- Ex vivo CCR5 knockout using ZFNs - a very appealing strategy for treatment of HIV
- T cell program has shown
  - Sustained increase in total CD4 count and CCR-modified CD4 cell engraftment with tissue trafficking
    - Influenced by host factors related to inflammation
    - Optimized by conditioning with Cytoxan 1 g/m²
  - Control of VL to undetectable or <1000 copies in a CCR5 Δ32 heterozygote for more than 1 year
  - Two subjects with a 2-log decrease in viral load with Cytoxan conditioning, sustained in one case for >39 weeks
  - Downward trends in viral reservoir in PBMCs over three years
    - Related to CD8 activation/numbers
- Optimized Phase II program commenced for SB-728-mR-T
- IND open for HSC program in 2014
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- Gary Lee, PhD
- Winson Tang, MD
- Shelley Wang, MD
- Marty Giedlin, PhD
- Shirley Clift
- Baolu Chen, PhD
- Michael Holmes, PhD
- Philip Gregory, DPhil

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**University of California, Los Angeles**
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- Gary Blick, MD

**Orlando Immunology Center**
- Edwin DeJesus, MD

**Ricky K Hsu, MD, PC**
- Ricky Hsu, MD

**Southwest CARE Center**
- Trevor Hawkins, MD

**Central West Clinical Research, Inc.**
- David Parks, MD

**Clinical Research Puerto Rico**
- Javier O. Morales-Ramírez, M.D

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- Pablo Tebas, MD
- Bruce Levine, PhD