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## Silence Therapeutics - Summary

<table>
<thead>
<tr>
<th>Valuable Platform</th>
<th>Reproducible, proprietary gene silencing (RNAi) therapeutics platform, rapidly generating internal pipeline and out-licensing options. Platform validated through recently announced collaboration with Mallinckrodt</th>
</tr>
</thead>
<tbody>
<tr>
<td>Growing Clinical Pipeline</td>
<td>SLN124 (β-Thalassemia and MDS(^1)). Phase Ib trial to start in H2 2019. SLN360 (LPa) IND/CTA in 2020. SLN500 (C3) IND/CTA in 2021.</td>
</tr>
<tr>
<td>Strong Experienced Team</td>
<td>Pioneers in siRNA for over 18 years, growing clinical team, and experienced biopharma management team</td>
</tr>
<tr>
<td>Target Selection</td>
<td>Focused on targeting indications in rare diseases and large population targets, including new medicines for cardiovascular disease and complement-mediated diseases</td>
</tr>
<tr>
<td>Strong Financial Position</td>
<td>$46m of cash(^2) extends runway to key clinical milestones such as SLN360 and SLN124 Phase I trial readouts. Cash position recently strengthened by Mallinckrodt collaboration ($25m in upfronts(^3))</td>
</tr>
</tbody>
</table>

---

**HQ in London**

- HQ in London
- Approx. 45 employees across both sites

**R&D in Berlin**

- R&D in Berlin
- Approx. 45 employees across both sites

---

Notes:

1. MDS = Myelodysplastic syndrome
2. As of 31st Aug 2019 and £=$1.28
3. $20 upfront and $5m equity investment
2019 – Year-to-Date Accomplishments

R&D Highlights

> SLN124 Clinical Trial Application filed in March 2019 with first patient dosed expected in H2 2019

> SLN124 granted Orphan Drug Designation in January by the European Medicines Agency for the treatment of β-Thalassemia

> SLN360 entered IND-enabling studies in February 2019 and is on track for IND and/or CTA in H2 2020

Corporate Highlights

> Collaboration with Mallinckrodt Pharmaceuticals for up to 3 targets for complement-mediated diseases. Exclusive license for C3-targeting siRNA (SLN500). $20m upfront payment

> Management team strengthened with the addition of Dr. Rob Quinn as Chief Financial Officer, Dr. Giles Campion as Head of R&D and Chief Medical Officer, and Jorgen Wittendorff as Head of Manufacturing

> Board augmented with the appointment of Iain Ross as Chairman and James Ede-Golightly and Dr. Steven Romano as Non-Executive Directors

> Collaboration with Genomics England to identify novel target genes associated with human disease
Leadership

**CEO**

David Horn Solomon

- Experienced public company CEO, board member and biotech investor
- CEO of Zealand Pharma from 2008 to 2015, during which time the company went public on Nasdaq OMX and its lead product, Adlixin® was approved in the US
- Previously Faculty Columbia University and founder Carrot Capital Healthcare Ventures

**Head R&D, CMO**

Giles Campion

- Former Chief Medical Officer and SVP R&D at Prosensa (2009-2016), playing a major role in their Nasdaq IPO and subsequent sale to Biomarin for $680m
- Most recently CMO at Albumedix
- Spent 4 years in senior R&D roles at Novartis
- Medical degree and doctorate from Bristol University

**CFO**

Rob Quinn

- Chartered accountancy training at Deloitte before joining GSK
- Area Finance Director for Africa and Developing Countries at GSK
- Joined Silence in early 2017 as Head FP&A
- PhD in Biochemistry from the University of Manchester

© Silence Therapeutics 2019
Experienced Broader Management Team

Head of Manufacturing
Jorgen Wittendorff
- Joining in Oct 2019
- Over 25 years’ experience in the development of pharmaceutical products
- Most recently Senior Director, CMC Manufacturing and Supply at Ablynx, prior to acquisition by Sanofi

Head of HR
Linnea Elrington
- Experienced HR professional. Formerly Global Head of Organization Development at Glory Global Solutions
- Earlier served in senior HR roles at Deloitte
- Chartered member of the Institute of Personnel and Development

Head of BD
John Strafford
- Broad experience in biotech, management consulting and specialty pharma. Including Advanz Pharma, Navigant and Antisoma
- PhD in Biochemical Engineering from University College London

General Counsel
Barbara Ruskin
- GC and Chief Patent Officer
- Over 25 years’ experience in IP and corporate law, including as Partner at Ropes & Gray and GC roles at Bionor and MTEM
- PhD in Biochemistry from Harvard University
GalNAc-siRNA for Treatment of Disease

Liver-specific and long lasting siRNA activity after internalization of GalNAc conjugate
Reproducibly Silencing Disease-Associated Genes Using our Proprietary Platform Technology

**Platform delivery technology;**
GalNAc-siRNA able to mediate highly specific gene silencing in hepatocytes (liver) – “Specificity upon specificity”.

**Patient friendly:** Subcutaneous delivery and infrequent dosing (monthly or longer). Well tolerated\(^1\).

~7,000 proteins expressed in the liver. Silence can target any of them by adapting the siRNA sequence, using the same technology.

\(^1\) Well tolerated in animal models tested.
A Competitive Platform, With Continuous Fine-Tuning to Further Improve Performance

> **Modification pattern:** number of non-natural modifications reduced from c.50% to <15% through the discovery of novel modification patterns

> **End stabilization:** increased circulation half-life, increased activity and duration of action

> **Linker:** simplified and flexible synthesis, increased activity, and option to control circulation and intracellular half-life

> **GalNAc:** 2-3 fold increase in activity achieved through optimization of number and placement of GalNAc units

> **IP:** 12 siRNA chemistry patent applications filed 2017-2019

AMG 890 chart reproduced from Melquist et al “Targeting apolipoprotein(a) with a novel RNAi delivery platform as a prophylactic treatment to reduce risk of cardiovascular events in individuals with elevated lipoprotein(a)” AHA 2016 Scientific Sessions
## Development Pipeline

<table>
<thead>
<tr>
<th>Programme</th>
<th>Discovery</th>
<th>Pre-clinical</th>
<th>Clinical</th>
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<tbody>
<tr>
<td><strong>Haematology</strong></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Beta Thalassaemia</td>
<td></td>
<td></td>
<td>SLN124</td>
</tr>
<tr>
<td>Myelodysplastic syndrome</td>
<td></td>
<td></td>
<td>SLN124</td>
</tr>
<tr>
<td>Undisclosed indication</td>
<td></td>
<td></td>
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<td><strong>Cardiovascular diseases</strong></td>
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<tr>
<td>Cardiovascular disease</td>
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<td></td>
<td>SLN360</td>
</tr>
<tr>
<td><strong>Rare diseases</strong></td>
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<td></td>
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</tr>
<tr>
<td>Complement-mediated diseases</td>
<td></td>
<td></td>
<td>SLN500</td>
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<tr>
<td>Rare metabolic</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rare undisclosed</td>
<td></td>
<td></td>
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</table>

### Out-Licensed Technology to Quark Pharmaceuticals

<table>
<thead>
<tr>
<th>Programme</th>
<th>Discovery</th>
<th>Pre-clinical</th>
<th>Phase 1</th>
<th>Phase 2</th>
<th>Phase 3</th>
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</thead>
<tbody>
<tr>
<td><strong>Teprasiran – Delayed Graft Function (DGF)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Teprasiran – Acute Kidney Injury (AKI)</strong></td>
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<td></td>
<td></td>
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</tr>
</tbody>
</table>
Collaboration with Mallinckrodt Pharmaceuticals for Complement-Mediated Diseases

- Agreement provides Mallinckrodt with exclusive worldwide licence for Silence’s C3 complement asset (SLN500), with options to licence up to two additional complement-targeted assets.

- Silence will be responsible for development of each asset until the end of Phase I, after which Mallinckrodt will take over clinical development and responsibility for commercialisation.

- This deal provides further validation for Silence’s proprietary siRNA platform, strengthening our position as a leader in RNA interference therapeutics.

Summary of Terms

$20 million Upfront

Up to $10 million in research milestones for SLN500 and each optioned asset, in addition to funding for Phase 1 clinical development, including GMP manufacturing. 1st $2m milestone received.

Potential clinical and regulatory milestones of up to $100 million for SLN500, as well as commercial milestones of up to $563 million. Up to $703 million in clinical, regulatory and commercial milestones per additional asset, should Mallinckrodt exercise its option.

Tiered low double-digit to high-teen royalties on net sales.

Equity investment of $5 million, non-executive Director seat on Silence Board to be filled by Steven Romano, M.D., Mallinckrodt Chief Scientific Officer.
SLN124
for the treatment of
Iron Overload Disorders
Market Opportunity of SLN124

US and EU patients

β-Thalassemia

~40,000 TDT\(^1\)
~20,000 NTDT\(^2\)

MDS\(^3\)

>100,000

Benefits of SLN124

SLN124 aims to:
1. Reduce organ iron levels &
2. Enhance erythropoiesis

Reduced transfusion frequency &
Secondary iron overload burden

Standard of Care

Transfusions
Every 2-5 weeks

Chelators
Daily

Limitations:
- Infection risk
- Intolerance
- Patient burden
- Hospital-based

Advantages v Competition

Quality of Life: SoC: Transfusions + Chelators
Safety
Organ iron levels
Compliance
Less frequent dosing

Notes: 1 TDT = Transfusion Dependent Thalassemia 2 NTDT = Non Transfusion Dependent Thalassemia 3 MDS = Myelodysplastic Syndrome
SLN124 Mechanism of Action: Increasing Hepcidin by Silencing its Repressor TMPRSS6

TMPRSS6 (Transmembrane Protease Serine 6) is a negative regulator of the BMP/SMAD signaling pathway.

- Inhibition of TMPRSS6 in hepatocytes induces hepcidin expression.
- Hepcidin reduces absorption of dietary iron and the release of iron from cellular storage, thereby reducing circulatory iron levels.
- The liver is the predominant source of hepcidin.

Silencing TMPRSS6

1. Increases hepcidin levels
2. Reduces iron levels
3. Improves erythropoiesis
4. Reduces anemia & iron overload
SLN124 Lowers Iron Levels for at Least 6 Weeks after Single Administration in Mice

- Long-lasting functional mRNA KD in liver
- Reduction of serum iron levels for at least 6 weeks
- Well tolerated with long duration of action in mice
Therapeutic Activity of SLN124 - Disease Model of Hereditary Hemochromatosis (HFE⁻/⁻ mice)

Collaboration with
Prof. Dr. Martina Muckenthaler
Heidelberg University, Germany

Study design

<table>
<thead>
<tr>
<th>D1</th>
<th>wk 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>SC, n=6-7 HFE⁻/⁻ mice, 1 mg/kg or 3 mg/kg</td>
<td></td>
</tr>
</tbody>
</table>

- **TMPRSS6 mRNA (liver)**
  - Dose-dependent and robust silencing of TMPRSS6 mRNA in the liver
  - Kruskal-Wallis test with uncorrected Dunn's test against non-targeting control CTRL

- **Hepcidin (serum)**
  - Increase in serum hepcidin levels
  - Reduction of serum and kidney iron levels to physiological values

- **Iron (serum)**
  - Reduction of serum and kidney iron levels to physiological values
  - Kruskal-Wallis test with uncorrected Dunn's test against non-targeting control CTRL
SLN124 Reduces ROS and Improves RBC Parameters in a β-Thalassemia Disease Model

Collaboration with Prof. J. Vadolas & Dr. Grigoriadis Monash Medical Centre/Melbourne, Australia

Study design
d1
wk 2
wk 5
SC, n=6-8 Hbb<sup>th3</sup>/mice, 3mg/kg

Reactive oxygen species (ROS)

<table>
<thead>
<tr>
<th>Experiment</th>
<th>Median Fl</th>
</tr>
</thead>
<tbody>
<tr>
<td>WT mice</td>
<td>100</td>
</tr>
<tr>
<td>PBS</td>
<td>200</td>
</tr>
<tr>
<td>CTRL</td>
<td>300</td>
</tr>
<tr>
<td>TM PRSS6 siRNA</td>
<td>400</td>
</tr>
<tr>
<td>p = 0.0069</td>
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</table>

Reticulocyte proportion

<table>
<thead>
<tr>
<th>Experiment</th>
<th>Reticulocytes [%]</th>
</tr>
</thead>
<tbody>
<tr>
<td>WT mice</td>
<td>20</td>
</tr>
<tr>
<td>PBS</td>
<td>30</td>
</tr>
<tr>
<td>CTRL</td>
<td>40</td>
</tr>
<tr>
<td>TM PRSS6 siRNA</td>
<td>50</td>
</tr>
<tr>
<td>p = 0.0042</td>
<td></td>
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</tbody>
</table>

Hematocrit

<table>
<thead>
<tr>
<th>Experiment</th>
<th>Haematocrit [%]</th>
</tr>
</thead>
<tbody>
<tr>
<td>WT mice</td>
<td>30</td>
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<tr>
<td>PBS</td>
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<tr>
<td>CTRL</td>
<td>50</td>
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<tr>
<td>TM PRSS6 siRNA</td>
<td>60</td>
</tr>
<tr>
<td>p = 0.0076</td>
<td></td>
</tr>
</tbody>
</table>

> Reduction of ROS to levels in healthy mice
> Normalization of reticulocyte proportion and improvement of hematocrit
> SLN124 significantly improves erythropoiesis in animal model for β-Thalassemia intermedia

ROS = reactive oxygen species; RBC = red blood cells

© Silence Therapeutics 2019
We are Planning to Initiate a Multicentre, Randomised, Placebo Controlled Phase Ib in 2019

This is a two part first-in-human, multicenter, randomized placebo controlled single ascending and multiple dose study to assess the preliminary safety, tolerability, PK and efficacy of SLN124 administered subcutaneously for the treatment of non-transfusion dependent thalassemia and low risk myelodysplastic syndrome.

Study design

> Part A:

- Single dose escalation study to evaluate the dose response to SLN124 with a view to identify the most appropriate dose to bring forward into the multiple dose portion of the study
- Primary aim to determine the safety and tolerability of SLN124 for the treatment of non-transfusion dependent β-thalassemia

> Part B:

- Bring forward the most efficacious and best tolerated dose to evaluate multiple administrations of SLN124 on hematinic parameters in patients with β-thalassemia and MDS
- Primary aim to determine the safety and tolerability of multiple doses of SLN124 for the treatment of non-transfusion dependent β-thalassemia and low risk myelodysplastic syndrome
SLN124 Summary

- **Preclinical package:** Robust data generated in several disease models.

- **Status:** Clinical Trial Application approved for SLN124 in April 2019 with MHRA. First patients expected to enter a Phase Ib study in H2 2019. Orphan Designation granted for β-Thalassemia.

- **Clinical plans:** Phase Ib planned in patients (β–Thalassemia and MDS). Network of KOLs established.

- **Dosing regimen:** Patient-friendly, with monthly or less frequent dosing expected and subcutaneous administration route.

- **Regulatory:** Positive feedback received at Scientific Advice meetings with both the UK (MHRA) and German (BfArM) national regulators.

- **SLN124 is well positioned for commercial success** against current standard of care and other medicines in development.
SLN360

for the treatment of

Cardiovascular Disease with High Lp(a)
Lp(a): Particle Heterogeneity

Tsimikas, JACC, 2017, 69:692
Normal Distribution Curve
Copenhagen General Population (n=6000)

Copenhagen General Population (n=6000)
Lp(a) Levels are About 90% Genetically Determined

Nordestgaard et al, EHJ, 2010, 31:2844
# Prevalence of Elevated Lp(a): US and Globally

<table>
<thead>
<tr>
<th>Prevalence</th>
<th>20%</th>
<th>10%</th>
<th>5%</th>
<th>1%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lp(a) Level</td>
<td>60 mg/dL</td>
<td>90 mg/dL</td>
<td>116 mg/dL</td>
<td>180 mg/dL</td>
</tr>
<tr>
<td>Number (USA)</td>
<td>64 million</td>
<td>30 million</td>
<td>16 million</td>
<td>3.2 million</td>
</tr>
<tr>
<td>Number (EU)</td>
<td>150 million</td>
<td>75 million</td>
<td>37.5 million</td>
<td>7.5 million</td>
</tr>
<tr>
<td>Number Globally</td>
<td><strong>1.4 billion</strong></td>
<td>700 million</td>
<td>350 million</td>
<td>70 million</td>
</tr>
</tbody>
</table>

Arterioscler Thromb Vasc Biol. 2016;36:2239-2245 and adapted from Tsimikas
Nonfatal MI and Coronary Death (9318 Cases)


MI = Myocardial infarction
Risk of MI as a Function of Lp(a) Levels in the General Population: Copenhagen City Heart Study

<table>
<thead>
<tr>
<th>Percentile</th>
<th>mg/dL</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;95th</td>
<td>&gt;117</td>
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<tr>
<td>90th-95th</td>
<td>77-117</td>
</tr>
<tr>
<td>67th-89th</td>
<td>30-76</td>
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<tr>
<td>22nd-66th</td>
<td>5-29</td>
</tr>
<tr>
<td>&lt;22nd [Reference]</td>
<td>&lt;5</td>
</tr>
</tbody>
</table>

Multivariable Adjusted

\[ P < .001 \]

\[ HR (95\% CI) \]

Kamstrup et al. JAMA 2009; 301: 2331-9
SLN360 Targets Lp(a) - an Independent Risk Factor for Cardiovascular Disease

Lp(a) levels are genetically determined

Recognized as a major untreated risk factor in cardiovascular disease

Lp(a) levels are not significantly modifiable through diet or approved pharmacological therapies

Large population worldwide with up to 10% with >80mg/dL (2x increased MI risk)

Multiple mechanisms by which Lp(a) causes CVD

> Pro-atherogenic
> Pro-thrombotic
> Pro-inflammatory

Targeting Lp(a) with SLN360 has the potential to address major unmet needs in cardiovascular disease

Image obtained from the Journal of Lipid Research March 2016
Prolonged serum knockdown of Lp(a) in NHP

> Multiple dosing at 3mg/kg resulted in sustained reduction of Lp(a) serum levels (>90%) for at least over two months after first dose (max ~>95% KD)

> Similar outcome after single subcutaneous injection of SLN360 at 9mg/kg

> Over 85% KD at NADIR for single 3mg/kg injection with 50% KD still observed after 2 months post treatment
Rationale

> Lp(a) is a low-density lipoprotein produced predominantly by the liver and composed of Apo(a) and Apo B, both hepatocyte expressed genes.

> Genetically defined high Lp(a) serum levels are unaffected by diet and exercise and are an independent risk factor for CVD. There is no specific Lp(a) targeting therapy available at the moment.

> An LPA silencing siRNA would provide a specific, safe and durable approach for reducing Lp(a) levels in high risk patients.

Our Programme

> A potent lead sequence has been selected and tested in vivo in non-human primates (NHP).

> Proof of mechanism has been achieved in NHP: dose dependent reduction in both LPA (liver mRNA) and Lp(a) (serum protein) observed, with max 95% KD observed after multiple dosing.

> Our drug compares positively against published data by competitors, suggesting a superior performance.

> IND/CTA is planned for H2 2020
SLN500 for the treatment of Complement-Mediated Diseases
The complement system is part of the innate immune system and consists of 3 pathways:

- Classical pathway
- Lectin pathway
- Alternative pathway

It represents an activation cascade with various effector functions, such as MAC (membrane attack complex) formation for pathogen destruction and activation of immune cells.

First drug on the market targeting the complement pathway is Eculizumab (Soliris, C5 Ab).

There is unmet need because Eculizumab is not consistently effective for all patients (mutations, non responders) and indications.

Targeting the complement system offers a broad indication spectrum (such as Paroxysmal Nocturnal Hemoglobinuria, Myasthenia gravis, C3 Glomerulopathy, atypical Hemolytic Uremic Syndrome or Cold Agglutinin Disease).

Mastellos et al., 2017
Study design:
> n=4 healthy mice per group, serum sampling at day -3, 4, 10 and 14, liver tissue at day 14
> siRNAs: non-optimized GalNac conjugated chemistry

C3 knock down:
> >80% mRNA and protein knock down achieved using non-optimized siRNAs
→ Identification of 2 potent siRNA sequences for lead optimization and NHP studies (using advanced chemistry)
## Expected Newsflow & Company Milestones

<table>
<thead>
<tr>
<th></th>
<th>H1 2019</th>
<th>H2 2019</th>
<th>2020</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>SLN124</strong></td>
<td></td>
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<tr>
<td>File CTA</td>
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<tr>
<td>First In Human Dosing</td>
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<tr>
<td>First Interim Results</td>
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<tr>
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<td>IND-enabling studies</td>
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<td><strong>Quark Out-License in QPI-1002</strong></td>
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<tr>
<td>Potential release of DGF Phase 3 results</td>
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<td>✓ ✓</td>
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