Investor Presentation

A unique therapeutic approach based on the selective inhibition of NOX enzymes

Euronext: GKTX

February 2018
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Genkyotex: Global leader in NOX therapeutics

  - Cash position 14.6 M€ as of December 2017 - cash to Q1 2019

- Specialized in the development of oral small molecule NOX therapeutics discovered in-house
  - Strong composition of matter IP protection including U.S., Europe and Japan

- Two potential blockbusters in fibrosis and inflammatory pain:
  - GKT831: a Phase 2 compound with potent anti-fibrotic activity
    - Phase 2 in Primary Biliary Cholangitis (PBC, orphan disease) launched in H1 2017
    - Investigator-Initiated Phase 2 in type 1 diabetes and kidney disease launched in Australia, fully funded by the Juvenile Diabetes Research Foundation Australia (JDRF) and the Baker Institute
    - Potential to address additional fibrotic diseases like NASH, IPF and scleroderma
  - GKT771: a preclinical compound with potent and novel analgesic mechanism
    - Mechanism targets multiple pain processing and angiogenic pathways

- Discovery programs: CNS, Hearing Loss and Oncology

- Partnership with Serum Institute of India (SIIL) for the development of prophylactic vaccines

Multiple key clinical milestones expected in the next 12 months
Seasoned management team with international life sciences experience

Elias Papatheodorou
Chief Executive Officer

- More than 20 years of experience in biotechnology and multinational companies
- Ex- Philip Morris International, The Coca Cola Company, Novosom AG, Medigene AG and Covagen AG
- Covagen was acquired by Janssen Pharmaceuticals, a J&J Company.
- Strong track record in fundraising, business and corporate development and licensing transactions

Philippe Wiesel
Chief Medical Officer & EVP

- Lead clinical research programs at Serono’s EU and US offices, including the phase 3 program (ex-US) for Raptiva in psoriasis, leading to the first EMA approval of a biologic agent for psoriasis
- Conducted basic research in the laboratories of Professor Edgar Haber at Harvard Medical School, and of Professor Hans Brunner at the Division of Hypertension in Lausanne

Alexandre Grassin
VP Finance & Administration

- Diverse experiences in Finance with Novartis from 2007-2010 and Alexion from 2010 to 2012
- Financial Auditor with KPMG

14 employees (including 10 in R&D)
Discovery platform delivers clinical pipeline in indications with high medical need

**GKT831**
**NOX1/4**
- Multiple fibrotic disorders

**GKT771**
**NOX1**
- Inflammatory pain & angiogenesis

**R&D**
- New NOX inhibitors for CNS & hearing loss
- NOX inhibitors for oncology and combination therapies
- GTL003 developed in partnership with Serum Institute of India Ltd SIIL

**R&D**
- Planned CTA application in 2018

**2017**
- H2

**2018**
- H1
- Phase 2 – Primary Biliary Cholangitis (PBC)
- Interim results
- Final results

**2019**
- H2
- Phase 2 – Type 1 diabetes induced kidney disease
- Investigator initiated trial – Funded by the Juvenile Diabetes Research Foundation
- Final results

**2019**
- H1

Investigator initiated trial – Longitudinal study

Funded by the Juvenile Diabetes Research Foundation
NOX enzymes: a fundamental scientific discovery creates a new therapeutic class

1960’

PHOSPHORYLATION

Kinase inhibitors

Gleevec

1980’

UBIQUITINATION

Ligase and proteasome inhibitors

Velcade

2000’

OXIDATION

NOX inhibitors

NADPH oxidase

NOX enzymes control multiple stress responses pathways simultaneously:

✓ Excessive stress responses leads to multiple diseases
NOX inhibitors: pathway based medicine addressing validated disease targets

**NOX: NADPH Oxidase**

A family of 7 enzymes that amplify multiple signaling pathways

**Initial focus on fibrotic diseases by targeting NOX1 and NOX4**
NOX 1 & 4 are major drivers of fibrogenesis in multiple organs

INJURY
Steatosis
Cholestasis
Hep C/Hep B
Alcohol

Quiescent stellate cell

NOX/ROS

FIBROGENIC PATHWAYS

Hedgehog
PDGF
ET-1
TGFβ1
MMP-2
TLR4
MCP-1
PDGF
MCP-1

Proliferation
Contractility
Fibrogenesis
Matrix degradation
Chemotaxis
Retinoid loss
WBC chemoattraction

Activated myofibroblast

Fibrosis

Pathways amplified by NOX1/4

NOX1 & NOX4 involved in multiple clinically validated fibrogenic pathways* and are the targets of our lead asset GKT831

*Sources:
Fibrosis, a severe disease reaching multiple organs

- **Eye**
  - Diabetic macular edema
  - Age-related macular degeneration
  - Glaucoma
  - Dry eye syndrome

- **Liver**
  - Primary biliary cholangitis (PBC) | Orphan disease
  - Non-alcoholic steatohepatitis (NASH)
  - Primary biliary sclerosis (PSC) | Orphan disease
  - Viral hepatitis
  - Alcoholic steatohepatitis

- **Gastrointestinal**
  - Crohn’s disease

- **Reproductive**
  - Infertility

- **Cancer**
  - Host derived tumor stroma
  - Myelofibrosis

- **Lung**
  - Idiopathic pulmonary fibrosis | Orphan disease
  - Cystic fibrosis | Orphan disease
  - Scleroderma | Orphan disease
  - Refractory asthma
  - COPD

- **Kidney**
  - Diabetic kidney disease
  - Focal segmental glomerulosclerosis | Orphan disease

- **Skin**
  - Scleroderma | Orphan disease
  - Keloids
  - Radiation & burn induced fibrosis

**Fibrosis: ~45% of all deaths in the developed world**

*Source* 1 The Journal of Clinical Investigation; Common and unique mechanisms regulate fibrosis in various fibroproliferative diseases; March 2007.
## GKT831: extensive preclinical and clinical programme

### Preclinical
- Established fibrotic models
  - PBC Models:
    - Bile duct ligation\(^1\)
    - MDR2 KO mice\(^2\)
  - NASH models:
    - STAM mice\(^3\)
    - Fast food diet\(^4\)
  - Toxic hepatitis model
    - CCL4-induced hepatitis and fibrosis\(^5\)

### Phase 1
- Single ascending dose
- Multiple ascending dose
- Food effect
- Drug interaction

### Phase 2 DKD (136 patients)
- 12-week treatment
- Indication: diabetic kidney disease

### Phase 2 PBC (102 patients)
- 24-week treatment
- Indication: PBC
- Launched in H1 2017 in Europe and North America

### Phase 2 DKD (IIT) (142 patients)
- 48-week treatment
- Indication: DKD
- Launched in H2 2017 in Australia

### Sources:
2. D. Brenner, UCD - preliminary results;
3. Stelic Institute, Tokyo - Keystone Fibrosis Symposia 2014;
4. N. Torok, UC Davis - Gastroenterology 2015;

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**Investor Presentation**
**Preclinical studies: over 40 publications in leading peer-reviewed journals**

Excess TGF-β mediates muscle weakness associated with bone metastases in mice

*Nat Med. 2015 Nov;21(11):1262-1271*

“GKT831 treatment prevented skeletal muscle oxidation and nitrosylation of RyR1, restored calstabin1 binding and improved EDL muscle–specific force. [...]”

NOX4-dependent fatty acid oxidation promotes NLRP3 inflammasome activation in macrophages

*Nat Med. 2016 Sep;22(9):1002-12*

“[...] our results demonstrate the potential of the NOX1 and NOX4 inhibitor GKT831, which is currently in phase 2 human clinical trials, as an NLRP3 inflammasome inhibitor [...]”

Reversal of Persistent Fibrosis in Aging by Targeting Nox4-Nrf2 Redox Imbalance

*Sci Transl Med. 2014 Apr 9;6(231):231ra47*

“GKT831 treatment led to a reversal of age-associated persistent fibrosis and reduced mortality. [...]”

Hepatocyte NADPH Oxidase 4 Regulates Stress Signaling, Fibrosis, and Insulin Sensitivity During Development of Steatohepatitis in Mice

*Gastroenterology. 2015 Aug;149(2):468-80*

“Inhibition of NOX4 by GKT831 improves inflammation and fibrosis in fast food diet-fed mice. [...]”

Targeting the Myofibroblastic Cancer-Associated Fibroblast Phenotype Through Inhibition of NOX4

*J Natl Cancer Inst. 2018 Jan 1;110(1)*

“[...] pharmacological inhibition of NOX4 may have broad applicability for stromal targeting across cancer types. [...]”
Four Phase I studies: very good safety and pharmacodynamics (PD) profile

Safety and PK
- No dose limiting toxicity
- No safety signal
- Dose proportional PK up to 900mg/day
- GKT831 is rapidly absorbed after oral dosing (median tmax ~ 1h)
- Mean half-life of parent compound is 8-15 hours
- Minimal renal elimination (<2%)
- Multiple dosing does not affect PK parameters
- Very low probability of DDI* through CYP3A4
- Low variability in PK parameters when taken with meals

Pharmacodynamics
- GKT831 reduces ROS production induced by UVB\(^4\) \textit{in vitro}\(^1\)
- GKT831 is pharmacologically active in healthy subjects

Single and multiple doses of GKT831 were well-tolerated and pharmacologically active in healthy subjects

- Drug-drug interactions studies
- Sources:
  \(^1\)In vitro studies conducted at StratiCELL for Genkyotex, unpublished; \(^2\) Once-daily; \(^3\) Twice a day; \(^4\) Ultra-violet
Initial phase 2 results in diabetic kidney disease

Despite not achieving the primary endpoint, GKT831 significantly improved multiple predefined secondary efficacy endpoints in diabetic kidney disease. Most importantly, results support development in inflammatory and fibrotic indications.

- **Excellent safety profile up to 200mg BID for 12 weeks**
  - Well tolerated with fewer adverse events than placebo: moderate to severe AEs 57 vs 15 (p<0.001) n=68/arm

- **Primary endpoint: no significant difference on renal outcomes**
  - Possible reasons:
    - Duration of treatment: 12 weeks sufficient for drugs acting on intra-renal hemodynamics, but not to demonstrate direct anti-inflammatory or anti-fibrotic effects
    - Dose

- **Secondary endpoints: pharmacological activity demonstrated**
  - Statistically significant reduction in liver enzymes – GGT (p<0.05)
  - Strong trend for reduction in triglycerides (p=0.066)
  - Statistically significant reduction in inflammation - hsCRP (p<0.05)
  - Strong trend for reduction in additional inflammatory markers – serum amyloid protein A (p<0.08), IL-6 (p=0.2)

- **GKT831 significantly reduces the incidence of adverse events**

<table>
<thead>
<tr>
<th>Severity</th>
<th>Placebo</th>
<th>GKT831</th>
<th>Diff.</th>
</tr>
</thead>
<tbody>
<tr>
<td>All</td>
<td>119</td>
<td>69</td>
<td>-42%</td>
</tr>
<tr>
<td>Mild</td>
<td>62</td>
<td>54</td>
<td>-12%</td>
</tr>
<tr>
<td>Moderate</td>
<td>44</td>
<td>14</td>
<td>-68%</td>
</tr>
<tr>
<td>Severe</td>
<td>13</td>
<td>1</td>
<td>-93%</td>
</tr>
</tbody>
</table>

$p < 0.001$ (CMH analysis)
Liver fibrosis can be caused by a multitude of liver insults: fat accumulation, cholestasis and viruses

- Cholestasis (Primary biliary cholangitis, primary sclerosing cholangitis, progressive familial intra-hepatic cholestasis)
- Nonalcoholic fatty liver disease and non-alcoholic steatohepatitis (NAFLD and NASH)
  - in 2016 NASH has become the leading cause of liver transplant in the US\(^1\)
  - liver cirrhosis is the 6th cause of death in developed countries and the 9th in developing countries\(^2\)
- Viral hepatitis (HBV, HCV)

Liver fibrosis impacts 300 to 700 million people worldwide\(^2\)

Sources:
Primary Biliary Cholangitis (PBC): an orphan disease in the large liver fibrosis area

A quicker proof of concept (PoC) in smaller and shorter trial

- **Description**
  - Chronic autoimmune liver disease leading to the progressive destruction of the bile ducts
  - Bile, a fluid produced in the liver, plays a role in digesting food but is toxic when it accumulates in the bile ducts and liver cells

- **Prevalence**
  - Prevalence of between 2 - 40 cases per hundred thousand-population\(^1\)
  - Women make up about 90% of PBC cases
    - The disease most often develops during middle age and is usually diagnosed in people between the ages of about 30 to 60 years
    - There appears to be a genetic component to developing PBC

- **Current treatment**
  - Current medications only slow disease progression and manage symptoms

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**Source:**
Boonstra K. et al. Epidemiology of primary sclerosing cholangitis and primary biliary cirrhosis: a systematic review. *J Hepatol*. 2012 May;56(5):1181-8
International liver fibrosis trial in primary biliary cholangitis

<table>
<thead>
<tr>
<th>Trial</th>
<th># patients</th>
<th>Design</th>
</tr>
</thead>
</table>
| Phase II | 102 PBC patients | • International trial conducted in North America and Europe  
• 24-week treatment with interim analysis on week 6 data  
• Placebo and 2 doses (400 mg once-daily and 400 mg twice daily) |

Primary endpoint

• A marker of liver injury (Change in serum Gamma Glutamyl Transferase - GGT)

Secondary endpoint

• Markers of liver fibrosis (ELF score, collagen fragments, transient elastography)  
• Markers of cholestasis (ALP, bilirubin)  
• Markers of liver injury (AST, ALT, CK-18)  
• Markers of inflammation (hsCRP, fibrinogen, IL-6)

A phase II study launched in H1 2017, with interim results expected during H1 2018 and final results expected by end of H2 2018

Sources:

1ALP: alkaline phosphatase; AST: aspartate aminotransferase; ALT: alanine aminotransferase; CK-18: cytokeratin-18; hsCRP: high sensitivity C-reactive protein; IL-6: interleukin-6
A unique positioning within the PBC/NASH competitive environment

Most products in development in liver fibrosis focus on metabolic or cholestatic pathways

<table>
<thead>
<tr>
<th>Metabolic / cholestatic</th>
<th>Inflammatory / fibrotic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Galmed</td>
<td>Tobira</td>
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<tr>
<td>Inventiva</td>
<td>(acquired by Allergan)</td>
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<tr>
<td>Allergan</td>
<td>Conatus Pharmaceuticals</td>
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<tr>
<td>Novartis</td>
<td>(licensed to Novartis)</td>
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<tr>
<td>Gilead</td>
<td>Gilead</td>
</tr>
<tr>
<td>(FXR agonists)</td>
<td>(ask-1 inhibitor)</td>
</tr>
<tr>
<td>Novo</td>
<td></td>
</tr>
<tr>
<td>Gilead</td>
<td></td>
</tr>
<tr>
<td>(ACC inhibitor)</td>
<td></td>
</tr>
</tbody>
</table>

GKT831’s mechanism of action includes direct anti-inflammatory / anti-fibrotic effects and can be combined with multiple metabolic or cholestatic mechanisms
Diabetic kidney disease (DKD) is a key diabetic complications and remains a major public health issue

**Diabetic kidney disease is the leading cause of end-stage renal disease**

- **Description**
  - Albuminuria is the initial disease manifestation, occurring ~10 years after diagnosis of type or type 2 diabetes
  - In DKD, progressive loss of renal function leads to dialysis, transplant, or death
  - Kidney fibrosis (glomerulosclerosis & intestinal fibrosis) drives disease progression

- **Prevalence**
  - DKD is the leading cause of end-stage renal disease
  - Affects 14% to 31% of people with type 1 diabetes after 20 years of diabetes
  - In the absence of DKD, survival of T1D patients is comparable to non-diabetics

- **Current treatment**
  - ACE inhibitors & angiotensin receptor blockers are the mainstay of treatment
  - Despite the proven efficacy, these therapies have a modest effect on the progressive loss of renal function
  - Once macro-albuminuria has developed, optimal standard of care does not prevent further decline in kidney function

Sources:
# Phase 2 trial in type 1 diabetes-induced kidney disease

<table>
<thead>
<tr>
<th>Trial</th>
<th># patients</th>
<th>Design</th>
</tr>
</thead>
</table>
| Phase II | 142 T1D DKD patients | - 48-week treatment in Australia. Trials conducted by Baker Heart and Diabetes Institute in Melbourne  
- GKT831 200mg BID against matching placebo |

### Primary endpoint

- Change in urinary albumin to creatinine ratio (UACR), adjusted for baseline

### Secondary endpoint

- Renal function: estimated glomerular filtration rate (eGFR), and cystatin C
- Renal injury: NGAL, KIM-1
- Inflammation: hsCRP, fibrinogen, IL-6
- Metabolomics and lipidomics profiles
- Exploratory epigenetics and transcriptomics studies

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**The DKD phase 2 trial launched in H2 2017**

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**Sources:**

1. NGAL: neutrophil gelatinase-associated lipocalin; KIM-1: kidney injury marker 1; hsCRP: high sensitivity C-reactive protein; IL-6: interleukin-6; T1D: type 1 diabetes
Solid patent protection in key countries

- **GKT831 (per se) and its derivatives in treating NADPH related disorders**

<table>
<thead>
<tr>
<th>Country</th>
<th>Application No.</th>
<th>Patent No.</th>
<th>Anticipated expiry</th>
<th>Type of protection</th>
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</table>

- **GKT831 (generically) and its derivatives in treating NADPH related disorders**

<table>
<thead>
<tr>
<th>Country</th>
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<th>Anticipated expiry</th>
<th>Type of protection</th>
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<td>20.03.2028</td>
<td>Pharmaceutical formulations/use</td>
</tr>
</tbody>
</table>

Solid IP portfolio with potential of term extensions in the US, Europe and Japan
A selective NOX1 inhibitor with broad therapeutic potential

- Potent, highly selective NOX1 inhibitor, expected to file CTA in 2018
- NOX1 plays key roles in angiogenesis, inflammation, and inflammatory pain
- GKT771 targets the NGF / TrkA / TRPV1 pain processing pathway: a clinically validated target for pain therapies
- GKT771 blocks angiogenesis through the VEGF\(^1\) pathway, a clinically validated anti-angiogenic target\(^1\)
- GKT771 shows potent activity in vitro and in vivo models of angiogenesis and inflammatory pain
- Combined mechanism of action (MoA) consistent with therapeutic potential in inflammatory pain and chronic inflammatory diseases
- Further therapeutic potential in eye diseases and itching
- Excellent ADME\(^2\) profile
- IP protection with NCE\(^3\)/use patent running until 2035

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1. VGEF: Vascular endothelial growth factor
2. ADME: Absorption, distribution, metabolism, and excretion
3. NCE: New chemical entity
Genkyotex on the stock market

- **Stock market information**
  - Market: Euronext Paris and Euronext Brussels
  - Number of shares: 77,850,006 (31.12.2017)

- **Cash & Cash equivalent (31.12.2017)**
  - M€ 14.6
  - Cash reach Q1.2019

- **Stock codes**
  - Name: GENKYOTEX
  - Mnemonic: GKTX
  - ISIN code: FR0011790542

- **Contacts Genkyotex**
  - Elias Papatheodorou – CEO
  - Alexandre Grassin – VP Finance and Administration

  Tel.: +41 (0) 22 880 10 25
  E-mail: info@Genkyotex.com
  Website: www.genkyotex.com

- **Shareholding structure** (as at February 28, 2017)

  (1) based on last major holding notifications
### Sustained news flow

<table>
<thead>
<tr>
<th>Year</th>
<th>Half</th>
<th>Events</th>
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<tbody>
<tr>
<td>2017</td>
<td>H1</td>
<td>Beginning of Phase 2 study (PBC)</td>
</tr>
<tr>
<td></td>
<td>H2</td>
<td>Interim Results Phase 2 study (PBC)</td>
</tr>
<tr>
<td>2018</td>
<td>H1</td>
<td>Final Results Phase 2 study (PBC)</td>
</tr>
<tr>
<td></td>
<td>H2</td>
<td>CTA Application</td>
</tr>
</tbody>
</table>

**GKT831**
- **NOX1/4**
- **Multiple fibrotic disorders**

**GKT771**
- **NOX1**
- **Inflammatory pain & angiogenesis**

**R&D**
- **Expected news flow from the R&D over the coming 2 years**

**Multiple potentially value-creating milestones expected in the next 12 months**
• Since its strategic combination with Genticel, Genkyotex owns Vaxiclase, (GTL003) a versatile platform well-suited for the development of various immunotherapies

• A partnership covering the use of Vaxiclase as an antigen per se has been established with Serum Institute of India Ltd (Serum Institute), the world’s largest producer of vaccine doses

• Objective of the collaboration:
  — to develop acellular multivalent combination vaccines against a variety of infectious diseases, including whooping cough

• Terms of the partnership:
  — covers territories outside the United States and Europe
  — up to $57 million in revenue for Genkyotex, before royalties on potential sales

• Current status:
  — last preclinical milestone was reached in November 2016, triggering a $1.2 million payment
  — In CTA\(^1\) enabling preclinical testing