Arming the immune system to fight cancer

Capital Markets Update

June 26th 2017
Important notice and disclaimer

This report contains certain forward-looking statements based on uncertainty, since they relate to events and depend on circumstances that will occur in future and which, by their nature, will have an impact on the results of operations and the financial condition of Targovax. Such forward-looking statements reflect the current views of Targovax and are based on the information currently available to the company. Targovax cannot give any assurance as to the correctness of such statements.

There are a number of factors that could cause actual results and developments to differ materially from those expressed or implied in these forward-looking statements. These factors include, among other things, risks or uncertainties associated with the success of future clinical trials; risks relating to personal injury or death in connection with clinical trials or following commercialization of the company’s products, and liability in connection therewith; risks relating to the company’s freedom to operate (competitors patents) in respect of the products it develops; risks of non-approval of patents not yet granted and the company’s ability to adequately protect its intellectual property and know-how; risks relating to obtaining regulatory approval and other regulatory risks relating to the development and future commercialization of the company’s products; risks that research and development will not yield new products that achieve commercial success; risks relating to the company’s ability to successfully commercialize and gain market acceptance for Targovax’s products; risks relating to the future development of the pricing environment and/or regulations for pharmaceutical products; risks relating to the company’s ability to secure additional financing in the future, which may not be available on favorable terms or at all; risks relating to currency fluctuations; risks associated with technological development, growth management, general economic and business conditions; risks relating to the company’s ability to retain key personnel; and risks relating to the impact of competition.
Capital Markets Update - Agenda

- Introduction – CEO, Øystein Soug
- Targovax’s technology and trials – CMO, Dr Magnus Jäderberg
- A physician’s view on pancreatic cancer – Prof Daniel Palmer
- Financial update – CFO, Erik Wiklund
- Q&A
Targovax develops two novel cancer immunotherapy drugs – both with promising phase I/II data

**ONCOS-102**
- Oncolytic virus
- Injected into the tumor
- Releases antigens
- Stimulates “killer” white blood cells (T-cells)

**TG01**
- Cancer vaccine
- Therapeutic vaccine
- Mimics antigens
- Stimulates “killer” white blood cells (T-cells)
Immunotherapy is considered to have enormous potential, and the market is expected to reach 30-50b USD by 2025

- 8 products currently on the market
- Market estimated to reach 40b USD in 2025
- Estimated that 2/3 of cancers will be treated with immunotherapy by 2025

* Citi Research, Barclays Capital, Leerink Swann, BMO Capital Markets
Targovax history

2007-2012
115 pts treated in ATAP

2007
OncoS founded

2009
Oncos founded

2012
1st ph I trial

2012
ONCOS-102 IND

2013
3 Orphan Designations

2013-2014

2013
3 Orphan Designations

2014
2 collaborations
Mesothelioma trial initiated

2015
2016
2017
Melanoma trial initiated

1989
1998
2002
2010
2011
2013

1st TG01 trial
Orphan Designation

Long term survival data
Targovax founded

OUS and Norsk Hydro
TG program (~230 pts)

Hydro’s pharma
program discont.

TG01-01 trial
initiated

Targovax founded

TG01 data
OSE main board
TG02 trial initiated

OncoS and
Targovax
merger

IPO
Two platforms and six clinical trials in total ensures a diversified program with frequent data readouts

<table>
<thead>
<tr>
<th>Cancer indication</th>
<th>Combined with</th>
<th>2017</th>
<th>2018</th>
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<td>Melanoma</td>
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<td>Prostate</td>
<td>DC therapy</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Resected Pancreatic</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Colorectal</td>
<td>CPI</td>
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<td></td>
<td></td>
</tr>
</tbody>
</table>

- Interim data
- Clinical, immune and safety data

4 readouts 2017
5 readouts 2018
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- Introduction to immunotherapy
- ONCOS-102 oncolytic virus platform
- TG RAS-peptide vaccine platform
- Targovax clinical program overview
IMMUNOTHERAPY

Immunotherapy is revolutionizing the way we treat cancer, in some cases curing previously thought incurable patients

Case example – Patient in a Yervoy checkpoint inhibitor trial

Prior to Yervoy*

4 weeks

8 weeks

20 weeks

8 months

1 year
The aim of cancer immunotherapy is to boost the natural cancer immunity cycle:

1. **Release of cancer antigens**
2. **Cancer antigen presentation**
3. **T-cell activation**
4. **T-cell trafficking**
5. **T-cell tumor infiltration**
6. **Cancer cell identification**
7. **Destruction of cancer cells**
Immunotherapies target different aspects of the cancer immunity cycle

**Checkpoint Inhibitors (CPIs)**
- CTLA-4 blockade: **Yervoy**
- PD-1 blockade:
  - **Keytruda**
  - **Opdivo**
- PD-L1 blockade:
  - **Tecentriq**
  - **Imfinzi**
  - **Bavencio**

**T-cell therapy:** **CAR-T** (not yet approved)

**DC-therapy:** **Provenge**

**Oncolytic virus:** **Imlygic**

**Kite Pharma**

**Novartis**

**AMGEN**
Targovax is developing two novel proprietary immunotherapy platforms, with promising phase I/II data

**ONCOS-102**
Oncolytic virus
- Genetically tailored Adenovirus
- Selectively infects and lyses cancer cells
- Releases cancer antigens
- Triggers immune response

**TG01**
Peptide vaccine
- Cocktail of 7 synthetic peptides mimicking clinically relevant RAS mutations
- Generates RAS-specific T-cells
- T-cells kill cancer cells displaying mutated RAS antigens on their surface
TG01 and ONCOS-102 have distinct targeting mechanisms in the cancer immunity cycle

1. ONCOS-102
Oncolytic virus

2. TG01
Peptide vaccine

3. lymph node

4. blood vessel

5. tumor

6. tumor

7. blood vessel
By combining immunotherapies multiple aspects of the cancer immunity cycle can be modulated in parallel

<table>
<thead>
<tr>
<th>Immuno-oncology mechanisms</th>
<th>Wake up the immune system</th>
<th>Train cancer specific T-cells</th>
<th>T-cells attack the cancer</th>
<th>Disarm cancer defence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Car analogy</td>
<td>Ignite engine</td>
<td>Switch on GPS--targeting</td>
<td>Press the gas pedal</td>
<td>Release brakes</td>
</tr>
</tbody>
</table>

- **ONCOS-102** – Oncolytic virus
- **TG 01** – Peptide vaccine
- **Peptide viral vaccine T-Cell therapy (CAR)**
- **Check point inhibitors (CPIs)**
The goal is to turn cancer into a manageable chronic disease by combining immuno-oncology therapies.
Agenda

- Introduction to immunotherapy
- ONCOS-102 oncolytic virus platform
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ONCOS-102 works by making cancer antigens visible to the immune system, thus generating tumor specific T-cells

<table>
<thead>
<tr>
<th>Activate immune system:</th>
<th>Train T-cells:</th>
<th>Attack the cancer:</th>
</tr>
</thead>
<tbody>
<tr>
<td>o Virus injected directly into the tumor / peritoneum</td>
<td>o APCs present tumor specific antigens at lymph nodes</td>
<td>o Tumor specific T-cells circulate in the body</td>
</tr>
<tr>
<td>o Infected cells lyse and release cancer-specific antigens</td>
<td>o Production of tumor specific T-cells</td>
<td>o Identify lesions and kill the cancer cells</td>
</tr>
<tr>
<td>o Immune system picks up antigens</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

ONCOS-102 works by making cancer antigens visible to the immune system, thus generating tumor specific T-cells.
Most patients do not respond to checkpoint inhibitors (CPIs), due to lack of T-cells in the tumor microenvironment.

Response rate to checkpoint inhibitors (CPIs)

- **Melanoma**: ~40%
- **Renal Cell carcinoma**: ~70%
- **Triple Negative Breast**: ~70%-80%
- **Lung Carcinoma (NSCLC)**: ~80%
- **Head and Neck**: ~80%
- **Bladder**: ~84%

Complimentary immune priming medicines may make tumors respond better to checkpoint inhibitors.
ONCOS-102 phase I: Increased tumor infiltrating CD8+ T-cells in 11 of 12 cancer patients with a range of solid tumors

Only 1 patient showed no response
6 patients showed 2- to 5-fold increase
5 patients showed >8-fold increase

Ranki et al., Journal for Immunotherapy of Cancer 2016, 4(17)
In the initial Phase I ONCOS-102 trial tumor specific and systemic immune response was observed

**Evidence that immune system recognizes tumor threat**

Innate Immune System (biopsy)

- Induction of proinflammatory cytokines + fever (all patients)
- Infiltration of innate immune cells into tumors in 11 out of 12 patients

**Evidence that T-cells find the tumor and are cell killing**

Adaptive immune system (biopsy)

- Increase in T-cell infiltration into tumors (including CD8+ killer T-cells) in 11 out of 12 patients
- Observation in one non-injected distant metastasis

**Evidence of production of tumor antigen specific T-cells**

Anti-tumor immune response (blood)

- Systemic induction of tumor-specific CD8+ T-cells

---

**Correlation between post-treatment increase in innate immune cells and OS**

Scatterplot of ranks:

- Increase in CD68+ cells post-treatment
  - Scatterplot of ranks
  - Overall survival
  - $p=0.0004$, $R=0.86$

**Correlation between post-treatment increase in CD8+ T-cells and OS**

- Scatterplot of ranks
  - $p=0.0004$, $R=0.86$

**Ovarian patient:**

- NY-ESO-1, MAGE-A1, MAGE-A3, and Mesothelin specific CD8+ cells

**Mesothelioma patient:**

- MAGE-A3 specific CD8+ cells

**Associated with clinical benefit**

www.targovax.com
The encouraging Phase I results have triggered the initiation of a broad ONCOS-102 clinical program consisting of four new trials.

- **Melanoma**
  - Phase I
  - 12 patients
  - Combination with PD-1 CPI in refractory patients
  - Proof-of-concept
  - Memorial Sloan Kettering

- **Mesothelioma**
  - Phase I/II - controlled
  - 30 patients
  - Combination with chemo
  - Randomized controlled trial
  - Ultra-orphan indication

- **Ovarian / colorectal**
  - Phase I/II - controlled
  - 78 patients
  - Collaboration with Ludwig & CRI
  - Combination with Medimmune’s durvalumab
  - Randomized controlled trial

- **Prostate**
  - Phase I
  - 10 patients
  - Partnered with Sotio
  - Combination with DC therapy

**Compassionate use program**
- Finland
- 115 patients
  - Testing within ATAP EU program
  - Individual clinical responses
  - Reassuring safety data

**Initial Phase I trial**
- Solid tumors
- 7 indications
  - 12 refractory patients
  - ONCOS-102 monotherapy
  - Correlation between immune activation and survival
Melanoma trial – will CPI refractory patients start responding after immune-priming with ONCOS-102?

**Setting**
- Advanced malignant melanoma patients not responding to CPIs
- Immune activate patients with ONCOS-102, then re-challenge with a CPI (Keytruda®)

**Site**
- 12 patients
- Memorial Sloan Kettering Cancer Centre

**Key endpoints**
- Safety
- Immune activation
- Clinical response data

**Sequence**
- ONCOS-102 – 3 weeks
- Keytruda – 5 months

**Proof-of-concept**
- Will CPI refractory melanoma patients start responding to Keytruda after challenge by ONCOS-102?
Agenda

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RAS is a key regulator of cell cycle that is mutated in 20-30% of all cancer patients, and >85% of pancreatic cancers.

One of the **most common mutations** in cancer.

RAS is one of the most well-defined neoantigens.

Results in cell division permanently switched on.

No existing therapies targeting RAS.

Occurs in >85% of pancreatic cancer patients.
The TG peptides prime the immune system to recognize and destroy RAS mutated cancer cells

**Activate immune system:**
- TG peptides injected into the skin with GM-CSF adjuvant
- APCs pick up the TG RAS antigens

**Train T-cells:**
- APCs migrate to lymph nodes and present RAS specific antigens
- Production of RAS specific T-cells

**Attack the cancer:**
- RAS specific T-cells identify mutated RAS antigens on cancer cell surface
- Killer T-cells destroy the cancer cells

Cocktail of 7 peptides covering all relevant RAS mutations in pancreas
These results are backed by encouraging 10 year survival data and immune response correlation from earlier trials.

Long-term data from earlier TG mono-therapy trials – resected pancreatic cancer

<table>
<thead>
<tr>
<th>Advanced pancreatic cancer</th>
<th>Evaluable patients</th>
<th>Median survival (from 1st vaccination)</th>
<th>1 year survival (from 1st vaccination)</th>
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<tbody>
<tr>
<td>TG01/GM-CSF (mono-therapy)</td>
<td></td>
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<tr>
<td>Detected immune response</td>
<td>14 / 25 (56%)</td>
<td>156 days</td>
<td>3 (21%)</td>
</tr>
<tr>
<td>Not detected Immune response</td>
<td>11 / 25 (44%)</td>
<td>109 days</td>
<td>1 (9%)</td>
</tr>
</tbody>
</table>

Historical control: 7.7% 10 year survival

4/20 (20%) patients > 10 years survival (resected patients)

Significantly better outcome for patients with immune response (non-resected)

(Clinical study report CTN RAS 98010 on file)

1 Wedén et al., 2011
2 Oettle H et al., JAMA 2007, vol 297, no 3
3 Oettle H et al., JAMA 2013, vol 310, no 14
We are currently working to replicate and expand on these encouraging clinical results

### Historical trials

- **Phase I**
  - Resected & non-resected
  - >100 patients
  - 10 year survival data
  - Correlation between immune response and survival
  - Excellent safety

### Completing trial

- **Phase I/II**
  - Resected pancreatic cancer
  - 32 patients
  - Encouraging survival data
  - Potent immune activation

### Planned / recruiting trials

- **Resected pancreas**
  - Phase II/III (tbd)
  - Randomized controlled trial
  - Aim to reach registration

- **Colorectal**
  - Phase I
  - 12 patients
  - Combination with CPI
  - >50% of patients RAS mutated
  - Currently recruiting patients

A randomized Phase II/III registration trial being designed.
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<td></td>
<td></td>
<td>Phase I</td>
</tr>
<tr>
<td>ONGS-102</td>
<td></td>
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| Resected Pancreatic | Chemo-        | Phase I/II |           |           |
| Colorectal         | CPI           |           |           | Phase Ib  |

Interim data
Clinical, immune and safety data

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- A physician’s view on pancreatic cancer – *Prof Daniel Palmer*

- Financial update – *CFO, Erik Wiklund*

- Q&A
Adjuvant therapy for resectable pancreatic cancer

London, June 2017

Daniel Palmer
Department of Molecular and Clinical Cancer Medicine
University of Liverpool and Clatterbridge Cancer Centre
Plan of the Talk

• Epidemiology
• The challenges of pancreatic cancer
• Palliative chemotherapy: current status
  – Gemcitabine
  – Folfirinox
  – Nab-paclitaxel
• Adjuvant therapies for resected pancreatic cancer
  – Clinical trials
  – Current state-of-the-art
• Where next?
  – Rationale for immunotherapy
• Targovax trial
Age-Standardised Ten-Year Net Survival Trends, Adults England and Wales, 1971-2011
Why so challenging?

- Advanced stage at time of diagnosis
- Even in operable cases
  - Challenging (and dangerous) surgery
  - High recurrence rates
- Relatively resistant to chemotherapy
  - Gemcitabine standard chemotherapy for 20 years
  - Gem vs weekly bolus 5-FU

Resectable: 10-20%
Locally advanced: 30-40%
Metastatic: 50-60%
Advanced pancreatic cancer is relatively resistant to chemotherapy

<table>
<thead>
<tr>
<th></th>
<th>5-FU</th>
<th>Gem</th>
</tr>
</thead>
<tbody>
<tr>
<td>Response</td>
<td>0%</td>
<td>5%</td>
</tr>
<tr>
<td>Median OS</td>
<td>4.4m</td>
<td>5.7m</td>
</tr>
<tr>
<td>P</td>
<td>0.025</td>
<td></td>
</tr>
<tr>
<td>1yr survival</td>
<td>2%</td>
<td>18%</td>
</tr>
<tr>
<td>CBR</td>
<td>4.8%</td>
<td>23.8%</td>
</tr>
<tr>
<td>P</td>
<td>0.022</td>
<td></td>
</tr>
</tbody>
</table>
Why so challenging?

Even in operable cases
• Challenging (and dangerous) surgery
• High recurrence rates

Long-term survival <10%

Is the problem due to:
• Local recurrence?
• Metastatic recurrence?
• Both?

Resectable 10-20%
Locally advanced 30-40%
Metastatic 50-60%
A Randomized Trial of Chemoradiotherapy and Chemotherapy after Resection of Pancreatic Cancer

John P. Neoptolemos, M.D., Deborah D. Stocken, M.Sc., Helmut Friess, M.D., Claudio Bassi, M.D., Janet A. Dunn, M.Sc., Helen Hickey, B.Sc., Hans Beger, M.D., Laureano Fernandez-Cruz, M.D., Christos Dervenis, M.D., François Lacaine, M.D., Massimo Falconi, M.D., Paolo Pederzoli, M.D., Akos Pap, M.D., David Spooner, M.D., David J. Kerr, M.D., and Markus W. Büchler, M.D., for the European Study Group for Pancreatic Cancer

NEJM 2004; 350:1200-10
A randomized trial of chemoradiotherapy and chemotherapy after resection of pancreatic cancer – ESPAC 1

![Graphs A and B showing survival rates with and without chemoradiotherapy and chemotherapy.]

- **Graph A**
  - Survival (%)
  - Months
  - No. at Risk:
    - No chemoradiotherapy: 144, 94, 57, 36, 22, 13
    - Chemoradiotherapy: 145, 94, 40, 29, 11, 5

- **Graph B**
  - Survival (%)
  - Months
  - No. at Risk:
    - No chemotherapy: 142, 89, 41, 18, 11, 7
    - Chemotherapy: 147, 99, 56, 38, 22, 11

*NEJM 2004; 350:1200-10*
median S(t)= 23.0 months (95%CI:21.1, 25.0)
median S(t)= 23.6 months (95%CI:21.4, 26.4)
χ^2_{LR}=0.74, p=0.39, HR_{GEM VS 5FU/FA}=0.94 (95%CI: 0.81, 1.08)

Neoptolemos et al JAMA 2010; 304: 1073-81
ESPAC - 4

722 patients pancreatic ductal adenocarcinoma ‘curative’ resection <12 wks

RANDOMISATION at Liverpool Cancer Trials Unit

GEMCITABINE
1000mg/m² - Days 1,8 and 15 for 6 cycles

GEMCITABINE
1000mg/m² - Days 1,8 and 15 for 6 cycles
CAPECITABINE
1660mg/m²/day – 21/28d i.e. 24 weeks

3-MONTHLY FOLLOW UP FROM RANDOMISATION TO DEATH

Stratified log-rank test with 5% 2-sided α, for a 10% difference in 2 year survival, 90% power = 480 events = 722 patients, 361 in @ arm

<table>
<thead>
<tr>
<th>Target number of patients</th>
<th>722</th>
</tr>
</thead>
<tbody>
<tr>
<td>Start date</td>
<td>13/01/08</td>
</tr>
<tr>
<td>Number of sites opened</td>
<td>106</td>
</tr>
<tr>
<td>Planned close date</td>
<td>01/11/14</td>
</tr>
<tr>
<td>Target achieved</td>
<td>31/07/14</td>
</tr>
</tbody>
</table>
## Patient Demographics

<table>
<thead>
<tr>
<th></th>
<th>GEM n=366</th>
<th>GEMCAP n=364</th>
<th>TOTAL n=730</th>
</tr>
</thead>
<tbody>
<tr>
<td>*Age (years)</td>
<td>65 (37-80)</td>
<td>65 (39-81)</td>
<td>65 (37-81)</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>212 (58%)</td>
<td>202 (55%)</td>
<td>414 (57%)</td>
</tr>
<tr>
<td>Female</td>
<td>154 (42%)</td>
<td>162 (45%)</td>
<td>316 (43%)</td>
</tr>
<tr>
<td>Baseline PS</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>158 (43%)</td>
<td>150 (41%)</td>
<td>308 (42%)</td>
</tr>
<tr>
<td>1</td>
<td>199 (54%)</td>
<td>202 (56%)</td>
<td>401 (55%)</td>
</tr>
<tr>
<td>2</td>
<td>9 (3%)</td>
<td>12 (3%)</td>
<td>21 (3%)</td>
</tr>
<tr>
<td>Smoking</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Never</td>
<td>151 (41%)</td>
<td>146 (40%)</td>
<td>297 (41%)</td>
</tr>
<tr>
<td>Past</td>
<td>136 (37%)</td>
<td>148 (41%)</td>
<td>284 (39%)</td>
</tr>
<tr>
<td>Present</td>
<td>62 (17%)</td>
<td>61 (17%)</td>
<td>123 (17%)</td>
</tr>
<tr>
<td>Unknown</td>
<td>17 (5%)</td>
<td>9 (2%)</td>
<td>26 (3%)</td>
</tr>
<tr>
<td>*Surgery to Rand (days)</td>
<td>65 (23-111)</td>
<td>64 (21-111)</td>
<td>64 (21-111)</td>
</tr>
</tbody>
</table>

* Median (Range)
Survival by Treatment

HR = 0.82 (95% CI, 0.68-0.98)
\( \chi^2(1) = 4.61, p = 0.032 \)

Median \( S(t) = 25.5 \) months (95% CI: 22.7-27.9)
Median \( S(t) = 28.0 \) months (95% CI: 23.5-31.5)

No. at Risk
- Gem: 366, 302, 207, 109, 61, 27, 9
- GemCap: 364, 328, 219, 139, 83, 50, 19
ESPAC Trials Overall Survival

*Study time = time from surgery for ESPAC 1/3, study time = time from randomisation for ESPAC 4
Where next?

• Rationale for immunotherapy in cancer

• Targovax trial
Immunotherapy approaches

• T cell checkpoints
Checkpoint inhibition in advanced melanoma and lung cancer

- Significant survival benefit
- Some long-term survivors
- Non-specific immune stimulation
  - Risk of auto-immune toxicities
Advanced pancreatic cancer is resistant to checkpoint inhibitors

Myeloid derived suppressor cells

IDO
Arginase
TGF-β

Tumour antigen

Tumour cell

Down-regulate MHC
Down-regulate TAA
PD-L1 expression

Aberrant MHC class I or β2-microglobulin

CD4 T cell

IL-10

TGF-β

IL-6 and STAT-3
TGF-β

TGF-β
IL-10

Regulatory T cell

Immunosuppressive tumour micro-environment
Macrophages
MDSCs
T cell checkpoints
Multiple points of intervention for immunotherapy

Conventional chemotherapy
Radiotherapy

Engineered vaccines

Enhanced antigen presentation by dendritic cells

Activation/mobilisation of DC
Flt3L, TLR agonists, CD40 ligation

Blockade of immunological checkpoints

Inhibition of:
regulatory T-cells
MDSCs
M2-macrophages

CTLA-4 blockade
PD-1 blockade

Enhanced traffic and activity of tumour-specific T-cells
Arming the immune system to fight cancer

A Phase I/II trial of TG01/GM-CSF and gemcitabine as adjuvant therapy for treating patients with resected RAS-mutant adenocarcinoma of the pancreas
Earlier studies demonstrated that adjuvant vaccination with TG01/GM-CSF given as monotherapy to pancreatic cancer patients after tumor resection induce mutant RAS specific immune response in 100% of patients.

This study evaluates safety, immunological response and Overall Survival of TG01-immunotherapy with adjuvant gemcitabine chemotherapy.
TG01-01 Study design

Modified cohort is ongoing (n=13). Last patient last visit May 2018
TG01-01 Study objectives

**Primary**
- To assess the safety of GM-CSF/TG01 vaccination and adjuvant chemotherapy
- To assess the immune response to GM-CSF/TG01 and the effect of adjuvant chemotherapy in patients receiving GM-CSF/TG01 after primary resection of pancreatic adenocarcinoma

**Secondary**
- To assess, at 2 years, the clinical efficacy of GM-CSF/TG01 in patients with resected pancreatic cancer

**Exploratory**
- To assess the relationship of KRAS status to recurrence
- To monitor CA19-9 levels
## Demographics

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Number of patients (N=19)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age</strong> (Y) median (min, max)</td>
<td>67 (49, 79)</td>
</tr>
<tr>
<td><strong>Gender, n (%)</strong></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>10 (53%)</td>
</tr>
<tr>
<td>Female</td>
<td>9 (47%)</td>
</tr>
<tr>
<td><strong>ECOG, n (%)</strong></td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>8 (42%)</td>
</tr>
<tr>
<td>1</td>
<td>11 (58%)</td>
</tr>
<tr>
<td><strong>CA19-9 (n=15) U/ml median (min, max)</strong></td>
<td>16 (8, 240)</td>
</tr>
<tr>
<td><strong>Hemoglobin (g/L) median (min, max)</strong></td>
<td>124.0 (104, 153)</td>
</tr>
<tr>
<td><strong>Disease staging at diagnosis</strong></td>
<td></td>
</tr>
<tr>
<td><strong>T stage</strong></td>
<td></td>
</tr>
<tr>
<td>T1</td>
<td>1 (5%)</td>
</tr>
<tr>
<td>T2</td>
<td>1 (5%)</td>
</tr>
<tr>
<td>T3</td>
<td>17 (90%)</td>
</tr>
<tr>
<td><strong>N stage</strong></td>
<td></td>
</tr>
<tr>
<td>N0</td>
<td>7 (37%)</td>
</tr>
<tr>
<td>N1</td>
<td>12 (63%)</td>
</tr>
<tr>
<td><strong>M stage</strong></td>
<td></td>
</tr>
<tr>
<td>M0</td>
<td>19 (100%)</td>
</tr>
<tr>
<td><strong>Resection surgical outcome, n (%)</strong></td>
<td></td>
</tr>
<tr>
<td>R0</td>
<td>6 (32%)</td>
</tr>
<tr>
<td>R1</td>
<td>13 (68%)</td>
</tr>
<tr>
<td><strong>KRAS mutation detected, n (%)</strong></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>16 (84%)</td>
</tr>
<tr>
<td>No</td>
<td>3 (16%)</td>
</tr>
<tr>
<td><strong>Time from surgery to first IMP adm (week) median (range)</strong></td>
<td>8 (7-12)</td>
</tr>
</tbody>
</table>
TG01-01 Efficacy (survival rate from resection)

<table>
<thead>
<tr>
<th>No. of patients</th>
<th>19</th>
</tr>
</thead>
<tbody>
<tr>
<td>Events</td>
<td>11 (57.9%)</td>
</tr>
<tr>
<td>Censored</td>
<td>8 (42.1%)</td>
</tr>
<tr>
<td>Median</td>
<td>143.7 weeks (34.1 months)</td>
</tr>
<tr>
<td>CI (95%)</td>
<td>(72.9, 174.1)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Time to overall survival (weeks)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overall</td>
</tr>
<tr>
<td>19</td>
</tr>
</tbody>
</table>

1 year* |
Assessed from resection (8 weeks before first IMP) |
17/19 (90%) |

2 years* |
13/19 (68%) |

Compares favorably with published historical two-year survival rates of resected cancer patients treated with gemcitabine alone of 30%-53%.
TG01-01 Immune response

Immune response by week 11 and entire study period (N=19)

<table>
<thead>
<tr>
<th>Study period</th>
<th>Immune responders</th>
<th>Immune responders DTH</th>
<th>Immune responders T-cells</th>
</tr>
</thead>
<tbody>
<tr>
<td>By end of initial treatment (week 11)</td>
<td>17/19 (89%)</td>
<td>16/19 (84%)</td>
<td>10/19* (53%)</td>
</tr>
<tr>
<td>Entire study period</td>
<td>18/19 (95%)</td>
<td>18/19 (95%)</td>
<td>14/19* (74%)</td>
</tr>
</tbody>
</table>

*Three patients (week 11) and two patients (entire study period) without blood samples for analysis

Immune response after week 11 (n=11)

<table>
<thead>
<tr>
<th>Study time point</th>
<th>No. of pts with immune monitoring after week 11</th>
<th>Patients with positive immune response after week 11</th>
</tr>
</thead>
<tbody>
<tr>
<td>After week 11</td>
<td>11</td>
<td>9/11 (82%)</td>
</tr>
</tbody>
</table>
TG01-01 Overall survival and immune response

- Immune response by week 11
- No immune response by week 11
- Patients still alive
- Immune response later than week 11

Patient number (KRAS +/-)
TG01-01 Safety profile (N=19)

<table>
<thead>
<tr>
<th>Serious Adverse Events</th>
<th>Number of Events</th>
<th>Relationship to study treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anaphylactic reaction</td>
<td>2</td>
<td>Related to TG01 +/- GM-CSF</td>
</tr>
<tr>
<td>Hypersensitivity</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Dyspnea</td>
<td>1</td>
<td>Related to Gemcitabine and TG01/GM-CSF</td>
</tr>
<tr>
<td>Lung infection</td>
<td>1</td>
<td>Related to Gemcitabine</td>
</tr>
<tr>
<td>Pyrexia (fever)</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Anaemia</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Anaphylactic shock</td>
<td>1</td>
<td>Unrelated to study treatments</td>
</tr>
<tr>
<td>related to a concomitant medication (Emend)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hyperglycemia</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Urosepsis</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Pneumonia</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Viral upper respiratory tract infection</td>
<td>1</td>
<td></td>
</tr>
</tbody>
</table>
TG01-01 Conclusions

- TG01/GM-CSF generated early immune responses in 89% of patients with R0/R1 resected pancreatic cancer. This demonstrate that TG01 vaccination activate mutant RAS specific T cells.

- The regimen was generally well tolerated although some late, manageable allergic reactions were seen.

- Median OS of 33.1 months is encouraging in context of published data

- Immune activation at both DTH and PBMC level is associated with the positive clinical findings.
ESPAC Trials Overall Survival

No. at Risk

<table>
<thead>
<tr>
<th>Trial</th>
<th>CTx</th>
<th>No CTx</th>
<th>CRT</th>
<th>Gem</th>
<th>5FU</th>
<th>Gemcap</th>
</tr>
</thead>
<tbody>
<tr>
<td>E1</td>
<td>149</td>
<td>143</td>
<td>145</td>
<td>539</td>
<td>551</td>
<td>366</td>
</tr>
<tr>
<td>E1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>105</td>
<td>99</td>
<td>103</td>
<td>422</td>
<td>430</td>
<td>302</td>
</tr>
<tr>
<td>E1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>68</td>
<td>50</td>
<td>54</td>
<td>283</td>
<td>283</td>
<td>207</td>
</tr>
<tr>
<td>E1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>46</td>
<td>28</td>
<td>30</td>
<td>187</td>
<td>180</td>
<td>109</td>
</tr>
<tr>
<td>E1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>34</td>
<td>17</td>
<td>19</td>
<td>126</td>
<td>131</td>
<td>61</td>
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<tr>
<td>E1</td>
<td></td>
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<tr>
<td></td>
<td>19</td>
<td>10</td>
<td>10</td>
<td>93</td>
<td>81</td>
<td>27</td>
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<tr>
<td>E1</td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>16</td>
<td>8</td>
<td>8</td>
<td>64</td>
<td>56</td>
<td>9</td>
</tr>
</tbody>
</table>

*Study time = time from surgery for ESPAC 1/3, study time = time from randomisation for ESPAC 4
ESPAC Trials Overall Survival

*Study time = time from surgery for ESPAC 1/3, study time = time from randomisation for ESPAC 4
Capital Markets Update - Agenda

- Introduction – CEO, Øystein Soug
- Targovax’s technology and trials – CMO, Dr Magnus Jäderberg
- A physician’s view on pancreatic cancer – Prof Daniel Palmer
- Financial update – CFO, Erik Wiklund
- Q&A
# Financial summary – end of Q1 2017

## Operations

<table>
<thead>
<tr>
<th></th>
<th>NOK</th>
<th>USD</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cash</td>
<td>147m</td>
<td>17m</td>
<td>End of Q1 2017</td>
</tr>
<tr>
<td>Net cash flow</td>
<td>-24m</td>
<td>-3m</td>
<td>Total Q1</td>
</tr>
<tr>
<td>Annual run rate</td>
<td>104m</td>
<td>12m</td>
<td>Last four quarters</td>
</tr>
<tr>
<td>Annual opex</td>
<td>116m</td>
<td>13m</td>
<td>Last four quarters</td>
</tr>
</tbody>
</table>

## The share

<table>
<thead>
<tr>
<th></th>
<th>OSE: TRVX</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Market Cap</td>
<td>NOK ~1bn</td>
<td>USD ~120m</td>
</tr>
<tr>
<td>Daily turnover</td>
<td>NOK 10m</td>
<td>USD 1m</td>
</tr>
<tr>
<td>Debt</td>
<td>NOK 43m</td>
<td>USD 5m</td>
</tr>
<tr>
<td>No. of shares</td>
<td>42.2m</td>
<td>46.0m fully diluted per April 18</td>
</tr>
<tr>
<td>Analysts</td>
<td>DNB, ABG Sundal Collier, Arctic, Redeye, Norske Aksjeanalyser</td>
<td></td>
</tr>
</tbody>
</table>
TRVX was upgraded to the main list on OSE in March, and has showed a positive trend in share turnover in 2017

*Development in daily average share turnover (NOK million / day)*

- **NOK ~1b market cap**
- **NOK 10m NOK avg. daily turnover in last 3 months**
- **NOK 850m total turnover in Q1**
- **560k shares avg. daily volume in Q1**
- **>3,700 owners**
- **42.2m shares* (46.0 fully diluted)**

* Up until 8th June
* Before Private Placement 8th June
Strong shareholder base as per May 2017

<table>
<thead>
<tr>
<th>Shareholder</th>
<th>Estimated ownership</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Shares m</td>
<td>Relative</td>
</tr>
<tr>
<td>HealthCap</td>
<td>Sweden</td>
<td>11,2</td>
</tr>
<tr>
<td>RadForsk</td>
<td>Norway</td>
<td>4,1</td>
</tr>
<tr>
<td>Nordea</td>
<td>Norway</td>
<td>3,0</td>
</tr>
<tr>
<td>Nordnet Livsforsikring</td>
<td>Norway</td>
<td>1,5</td>
</tr>
<tr>
<td>KLP</td>
<td>Norway</td>
<td>1,3</td>
</tr>
<tr>
<td>Statoil</td>
<td>Norway</td>
<td>0,9</td>
</tr>
<tr>
<td>Danske Bank (nom.)</td>
<td>Norway</td>
<td>0,8</td>
</tr>
<tr>
<td>Timmuno AS</td>
<td>Norway</td>
<td>0,7</td>
</tr>
<tr>
<td>Prieta AS</td>
<td>Norway</td>
<td>0,7</td>
</tr>
<tr>
<td>Nordnet Bank AB (nom.)</td>
<td>Sweden</td>
<td>0,7</td>
</tr>
<tr>
<td>Thorendahl Invest AS</td>
<td>Norway</td>
<td>0,3</td>
</tr>
<tr>
<td>Sundt AS</td>
<td>Norway</td>
<td>0,3</td>
</tr>
<tr>
<td>Netfonds Livsforsikring AS</td>
<td>Norway</td>
<td>0,3</td>
</tr>
<tr>
<td>Avanza Bank AB (nom.)</td>
<td>Sweden</td>
<td>0,3</td>
</tr>
<tr>
<td>The Bank of NY Mellon (nom.)</td>
<td>Belgium</td>
<td>0,2</td>
</tr>
<tr>
<td>Tobech Invest AS</td>
<td>Norway</td>
<td>0,2</td>
</tr>
<tr>
<td>Istvan Molnar</td>
<td>Norway</td>
<td>0,2</td>
</tr>
<tr>
<td>Danske Bank (nom.)</td>
<td>Norway</td>
<td>0,2</td>
</tr>
<tr>
<td>NHO - P665AK</td>
<td>Norway</td>
<td>0,2</td>
</tr>
<tr>
<td>Kristian Falnes AS</td>
<td>Norway</td>
<td>0,2</td>
</tr>
<tr>
<td><strong>Top 20</strong></td>
<td></td>
<td><strong>27,0</strong></td>
</tr>
<tr>
<td><strong>Other shareholders (3772)</strong></td>
<td></td>
<td><strong>15,2</strong></td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td></td>
<td><strong>42,2</strong></td>
</tr>
</tbody>
</table>

New shareholders in Private Placement:
- Nyenburgh
- Trium
- Millenium Capital Partners
- Interogo
- AP3
- Aramea AM

42.2m ordinary shares
- Management ownership: 2.1%
- 3,792 shareholders

46.0m shares fully diluted*
- Average strike price on options ~NOK 21
- Total dilutive effect of options is 7.9%

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1 Includes outstanding options (3,634,263) and Restricted Stock Units (169,128) to Board members
Multiple near term value inflection points
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