Arming the immune system to fight cancer

Capital Markets Update

June 8th 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 – *Dr Svein Dueland*
- Financial update – *CFO, Erik Wiklund*
- Q&A
- Lunch

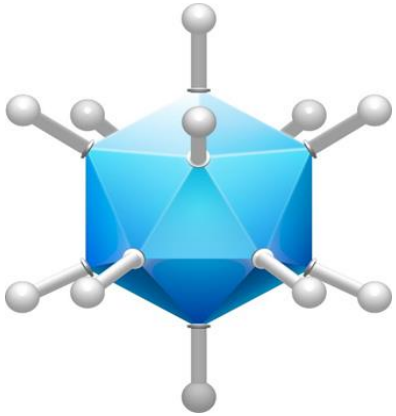
Targovax develops two novel cancer immunotherapy drugs – both with promising phase I/II data

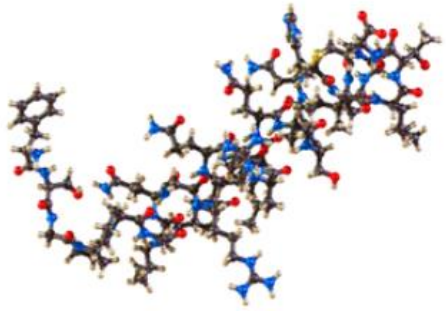
ONCOS-102
Oncolytic virus

- 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)

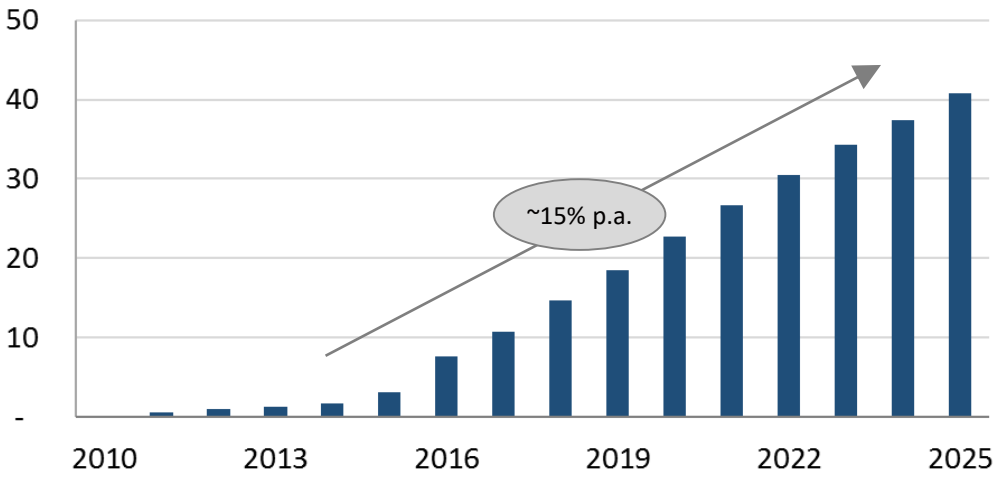




Immunotherapy is considered to have enormous potential, and the market is expected to reach 30-50b USD by 2025

Science, December 2013

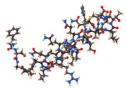
Estimated market size ($Bn)*



- 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 2009 2012 2013 2014

2007-2012
115 pts treated in ATAP

2012-2013
1st ph I trial

2013-2014
3 Orphan Designations

Oncos founded

ONCOS-102 IND

1989 1998 2002 2010 2011 2013

OUS and Norsk Hydro TG program (~230 pts)

1st TG01 trial

Hydro's pharma program discont.

Long term survival data
Targovax founded

Orphan Designation

TG01-01 trial initated

2015 2016 2017

2 collaborations
Mesothelioma trial initated

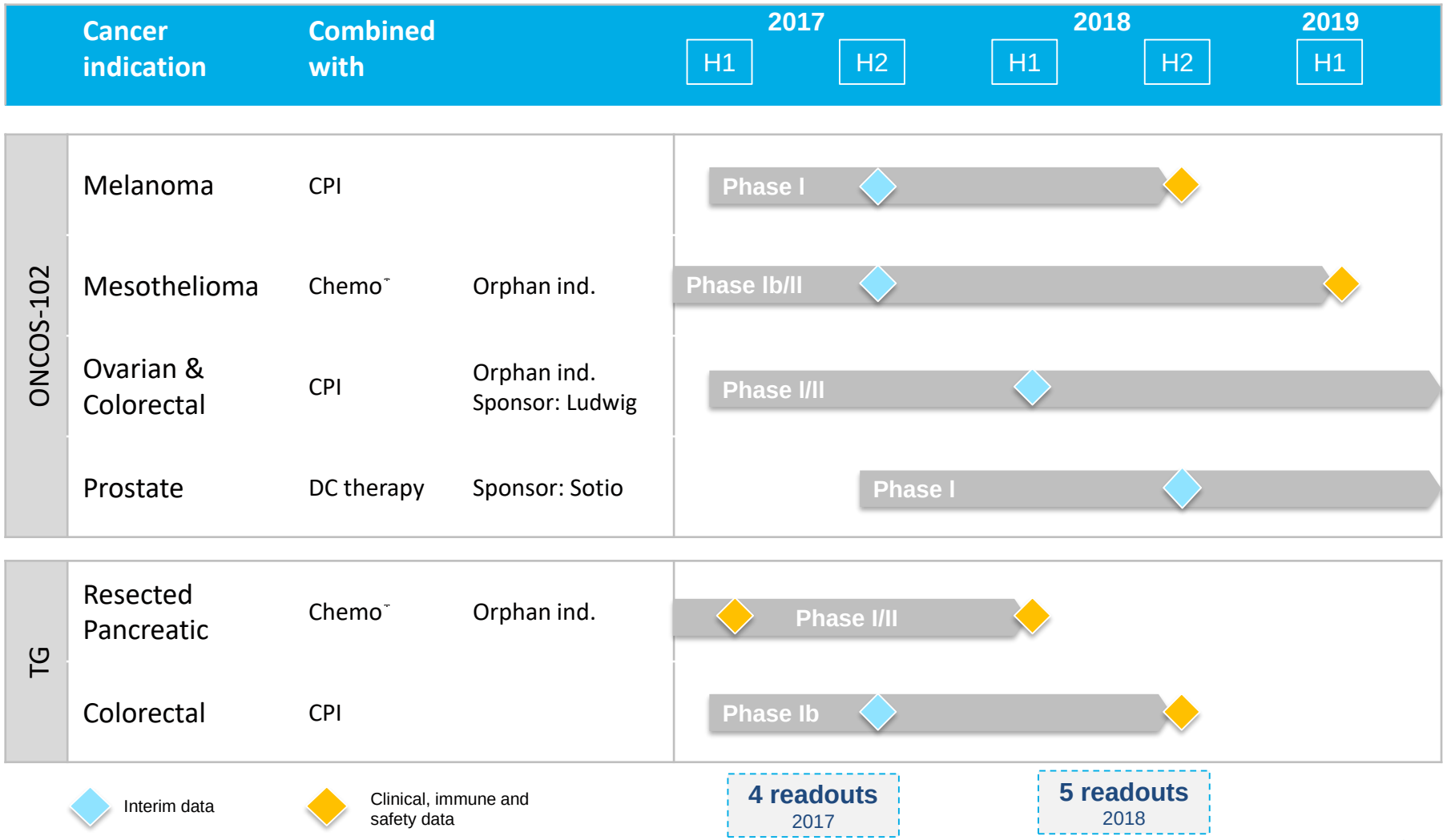
Melanoma trial initated

Oncos and Targovax merger

IPO

TG01 data
OSE main board
TG02 trial initiated

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



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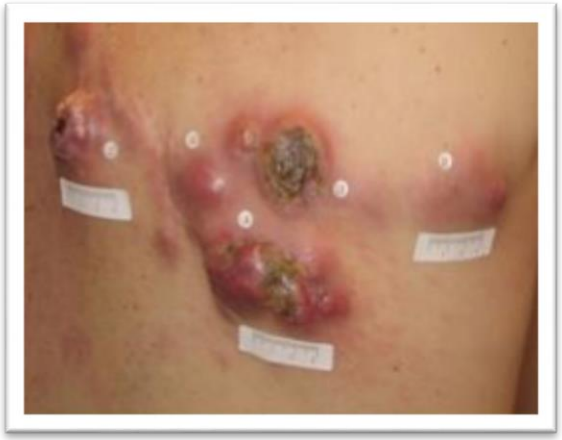
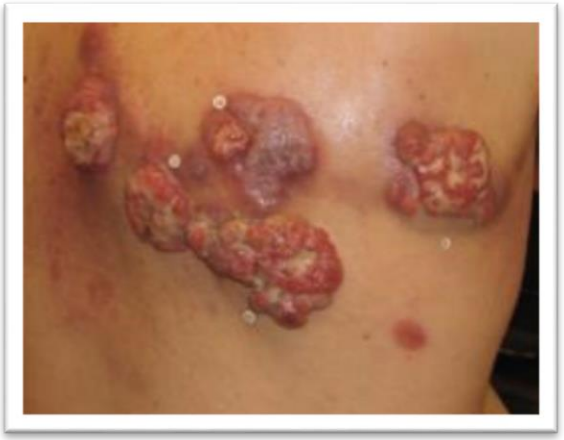
- **Introduction to immunotherapy**
- ONCOS-102 oncolytic virus platform
- TG RAS-peptide vaccine platform
- Targovax clinical program overview

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

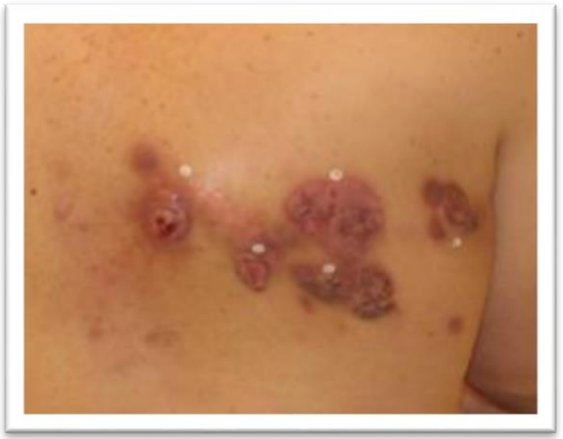
Case example – Patient in a Yervoy checkpoint inhibitor trial



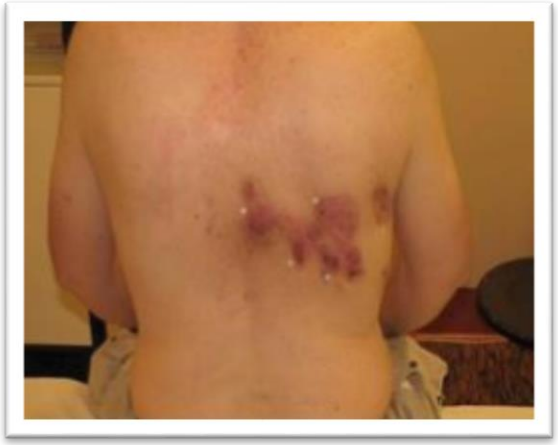
*Prior to Yervoy**



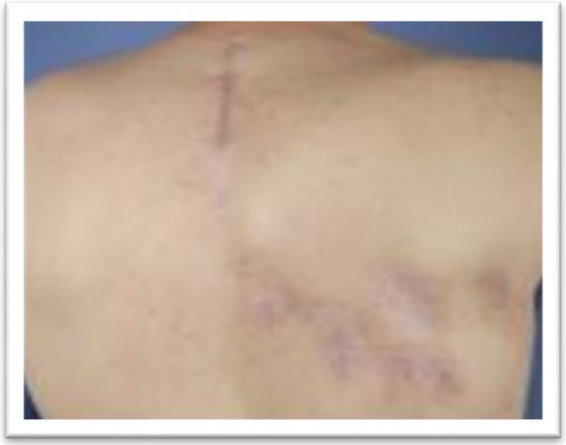
4 weeks



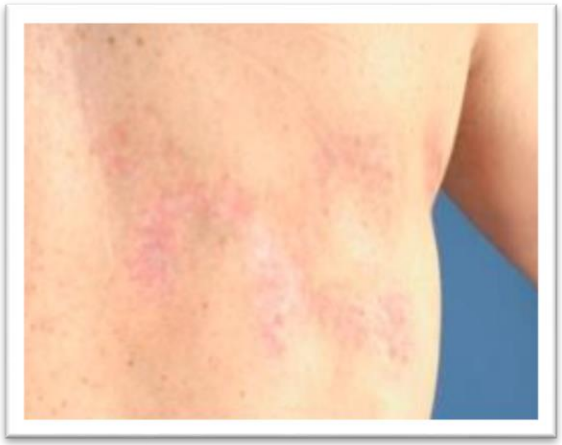
8 weeks



20 weeks

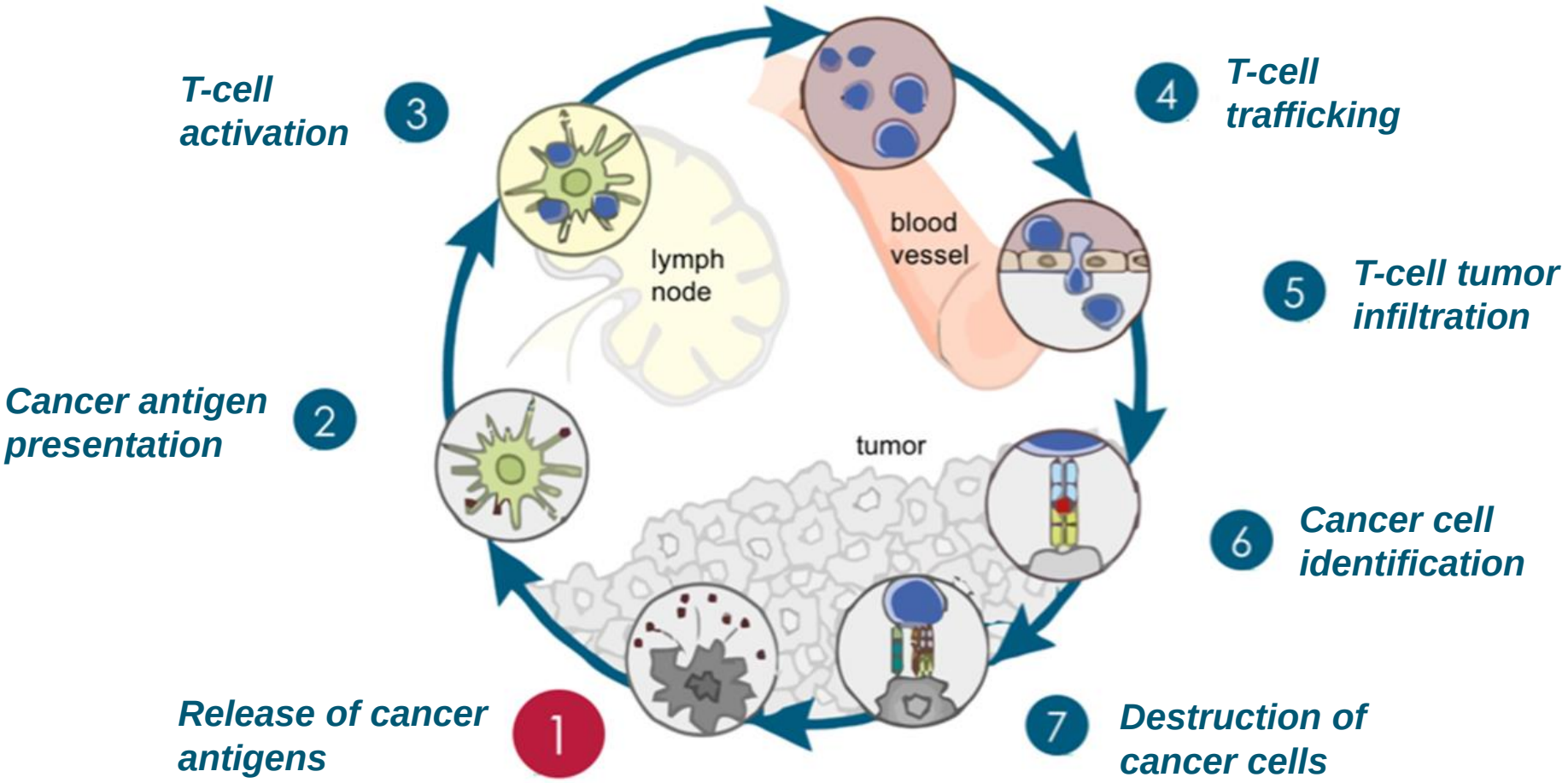


8 months

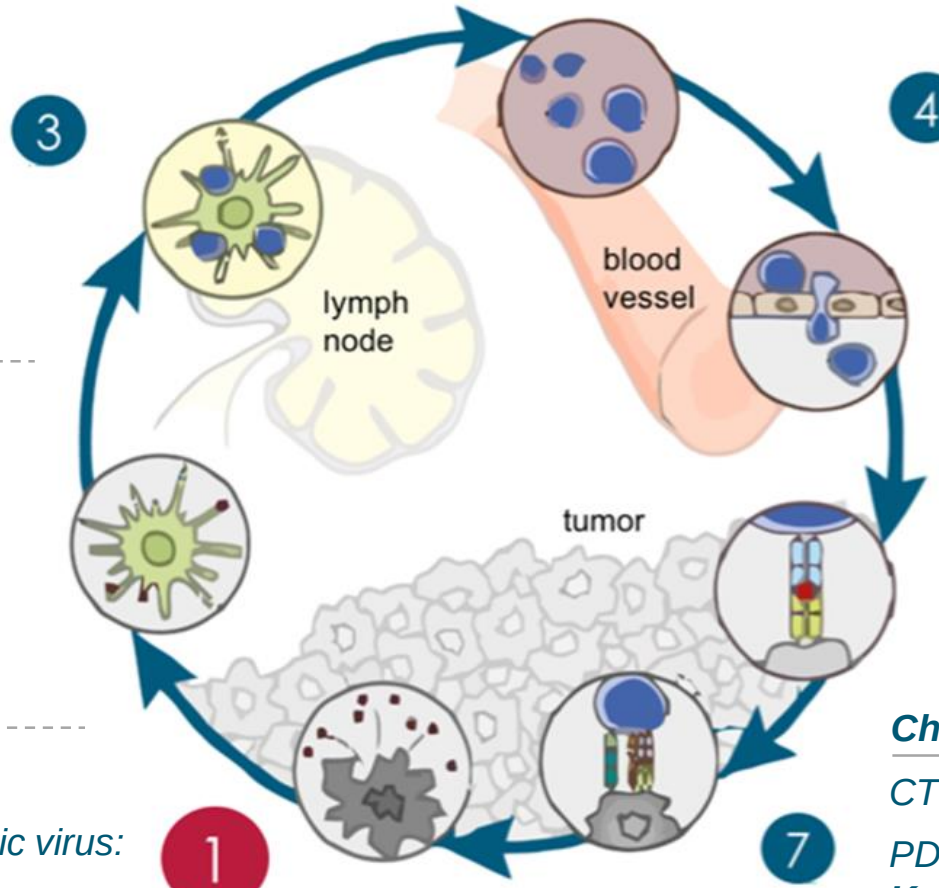


1 year

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



Immunotherapies target different aspects of the cancer immunity cycle



DC-therapy:
Provenge
2

Dendreon

Oncolytic virus:
Imlygic

AMGEN

Kite Pharma

NOVARTIS

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

Checkpoint Inhibitors *(CPIs)*

CTLA-4 blockade: ***Yervoy***

PD-1 blockade:
Keytruda
Opdivo

PD-L1 blockade:
Tecentriq
Imfinzi
Bavencio

MERCK

Bristol-Myers Squibb

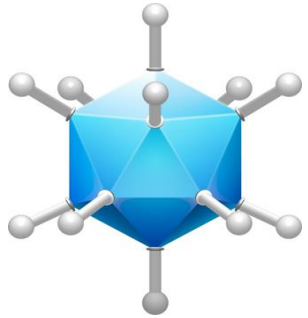
Roche

Pfizer

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

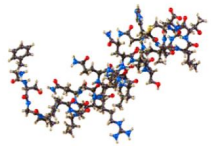
ONCOS-102
Oncolytic virus

- Genetically tailored Adenovirus
- Selectively infects and lyses cancer cells
- Releases cancer antigens
- Triggers immune response

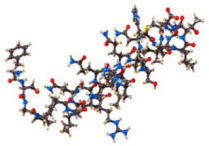


TG01
Peptide vaccine

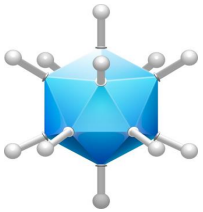
- Cocktail of 7 synthetic peptides mimicking clinically relevant RAS mutations
- Generates RAS-specific T-cells
- T-cells kill cancer cells displaying mutated RAS antigens on their surface



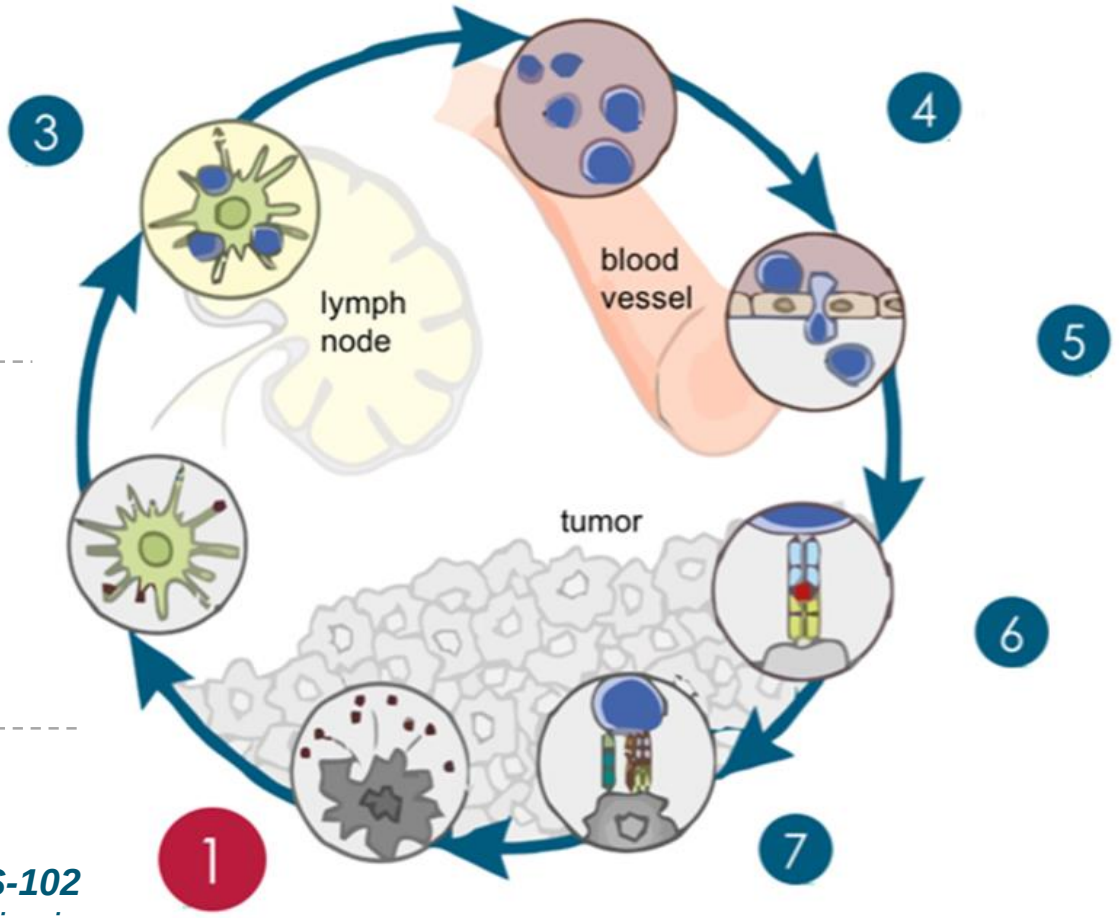
TG01 and ONCOS-102 have distinct targeting mechanisms in the cancer immunity cycle



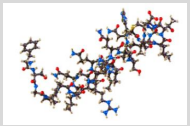
TG01
Peptide vaccine



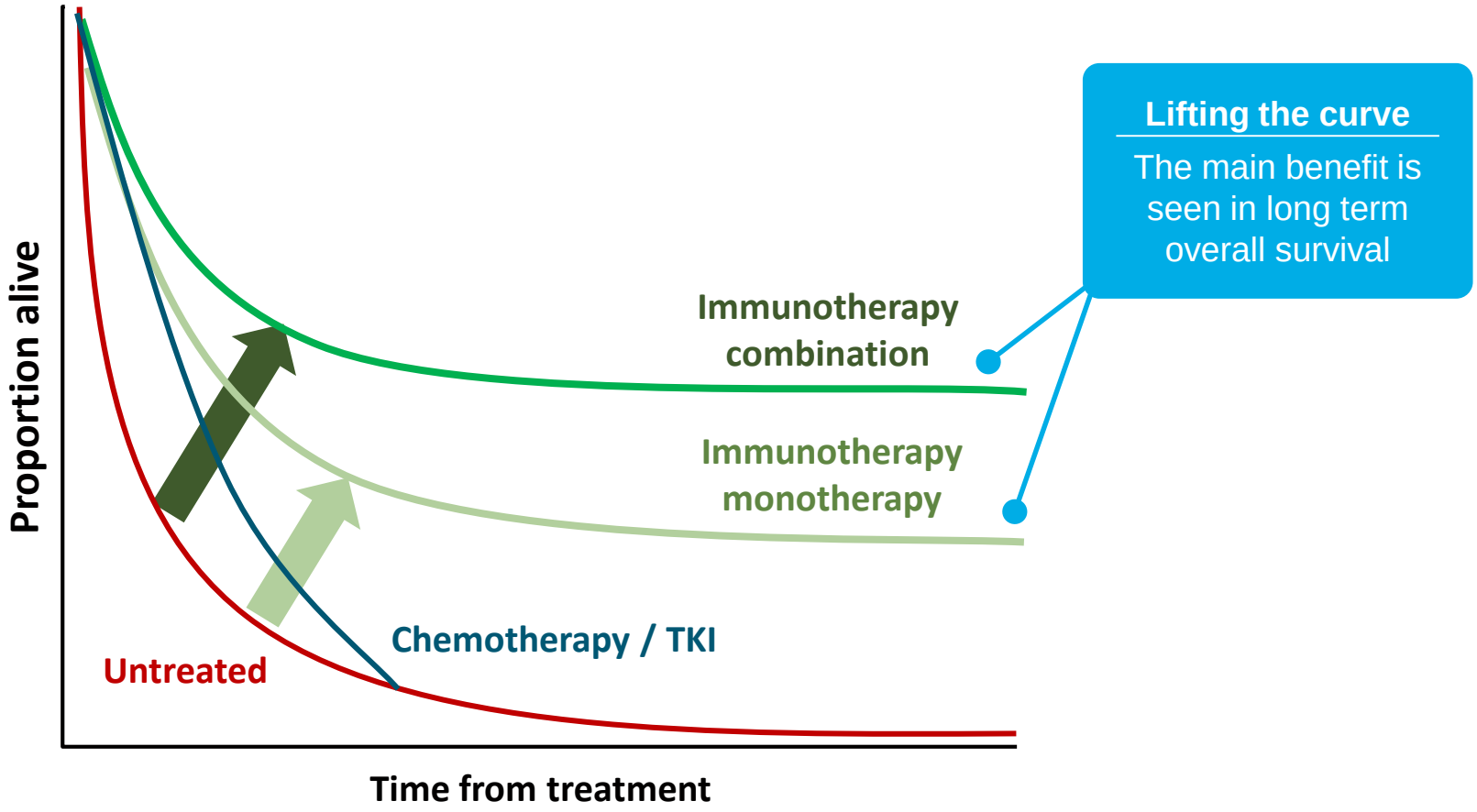
ONCOS-102
Oncolytic virus



By combining immunotherapies multiple aspects of the cancer immunity cycle can be modulated in parallel

Immuno-oncology mechanisms	Wake up the immune system	Train cancer specific T-cells	T-cells attack the cancer	Disarm cancer defence
 *Car analogy*	*Ignite engine*	*Switch on GPS– targeting*	*Press the gas pedal*	*Release brakes*
 **ONCOS-102** – Oncolytic virus	✓	✓	✓	-
 **TG 01** – Peptide vaccine	✓	✓	✓	-
Kite Pharma BAVARIAN NORDIC Peptide viral vaccine T-Cell therapy (CAR)	✓	✓	✓	-
MERCK AstraZeneca Genentech Bristol-Myers Squibb EMD Serono Pfizer 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**
- TG RAS-peptide vaccine platform
- Targovax clinical program overview

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

Activate immune system:

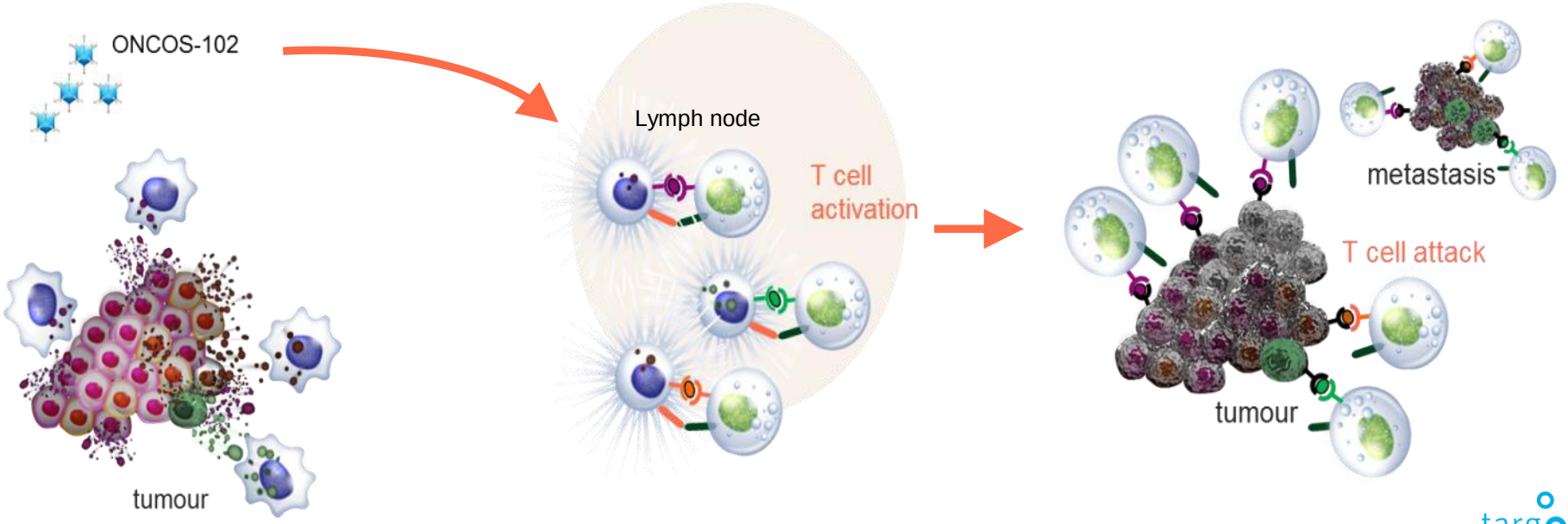
- Virus injected directly into the tumor / peritoneum
- Infected cells lyse and release cancer-specific antigens
- Immune system picks up antigens

Train T-cells:

- APCs present tumor specific antigens at lymph nodes
- Production of tumor specific T-cells

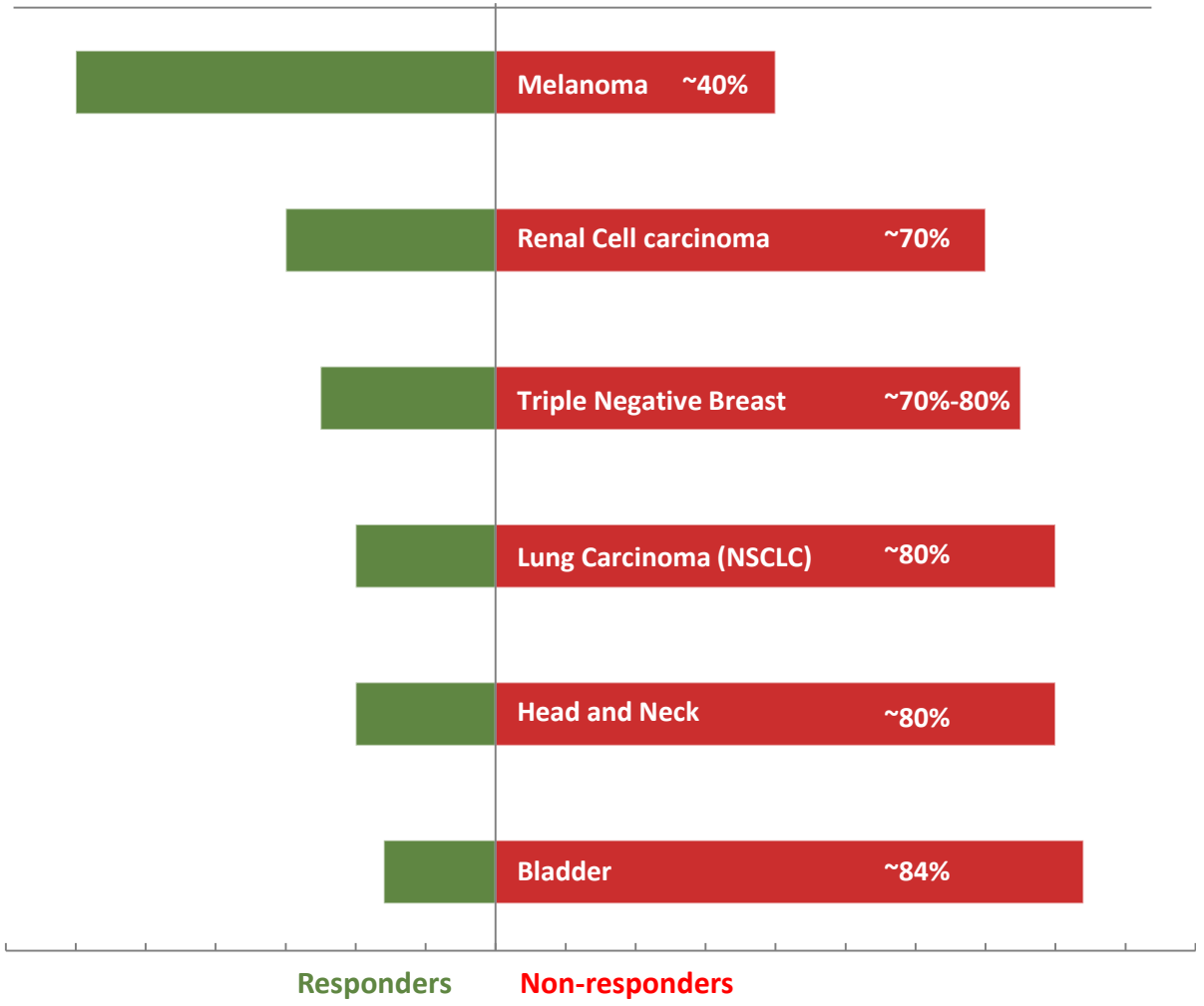
Attack the cancer:

- Tumor specific T-cells circulate in the body
- Identify lesions and kill the cancer cells



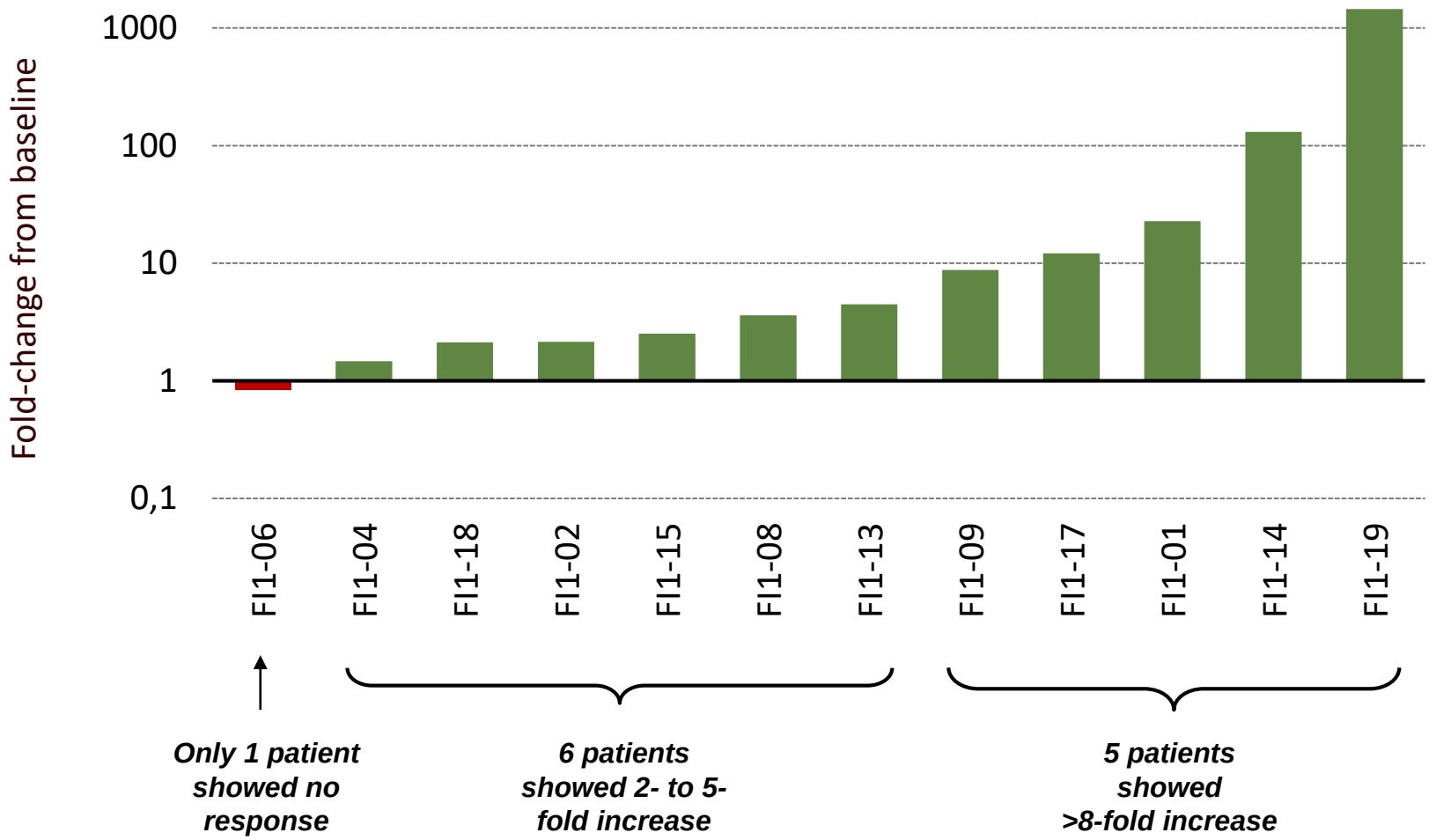
Most patients do not respond to check point inhibitors (CPIs), due to lack of T-cells in the tumor microenvironment

Response rate to checkpoint inhibitors (CPIs)



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



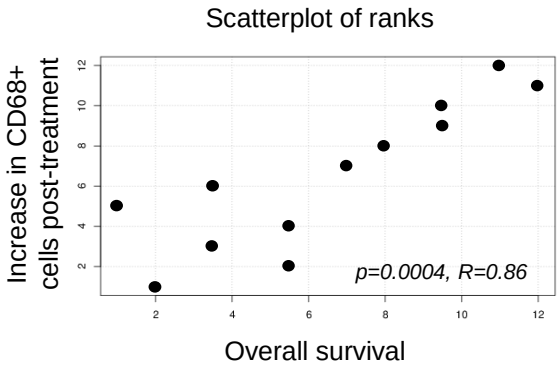
Ranki et al., Journal for Immunotherapy of Cancer 2016, 4(17)

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



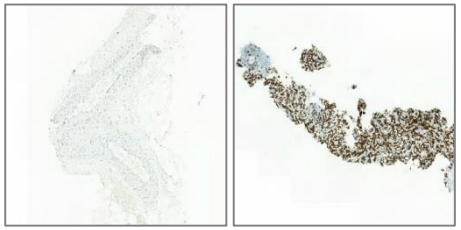
Correlation between post-treatment increase in innate immune cells and OS

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

OvCa. patient (FI1-19)



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

Evidence of production of tumor antigen specific T-cells

Anti-tumor immune response (blood)

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

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

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

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
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*

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Melanoma trial – will CPI refractory patients start responding after immune-priming with ONCOS-102?

Setting

- Advanced malignant melanoma patients not responsing 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?*

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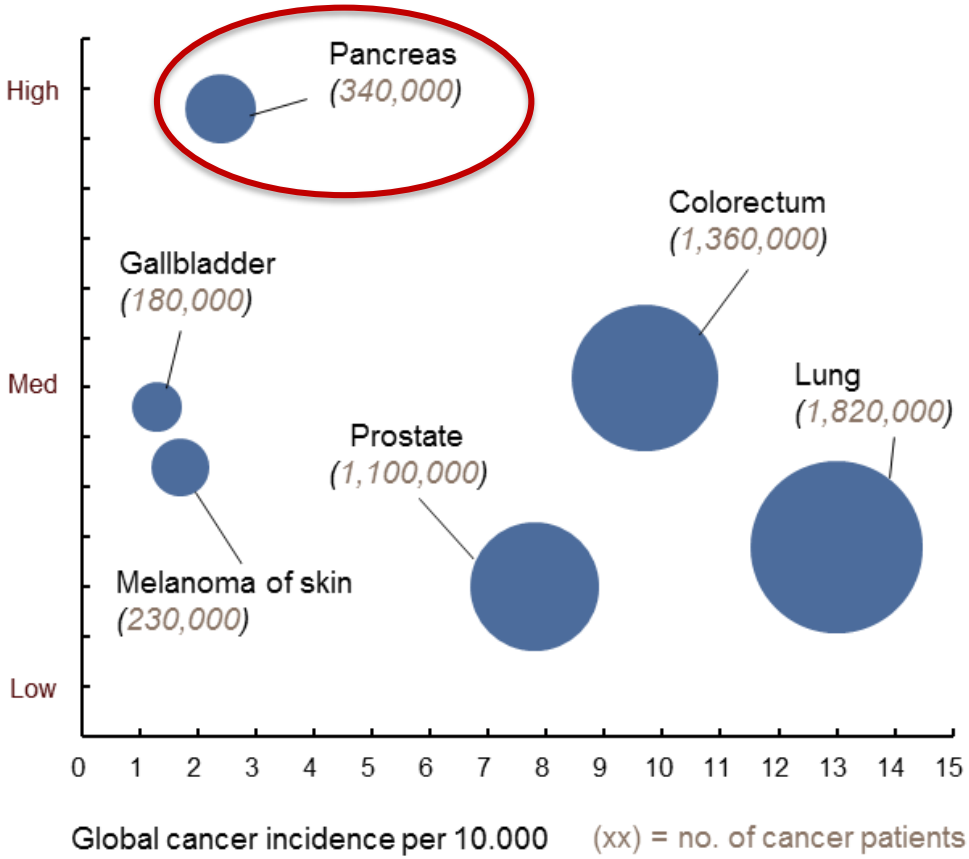
- **TG RAS-peptide vaccine platform**
- Targovax clinical program overview

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

Incidence of RAS mutations



- 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:

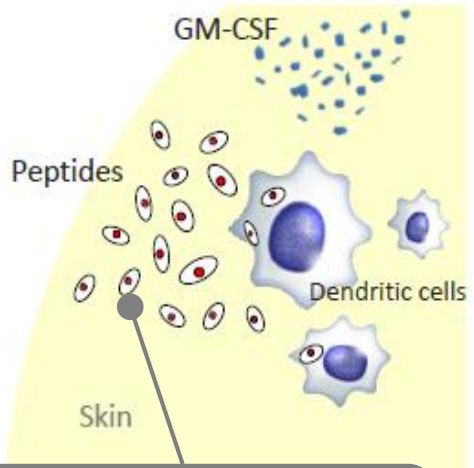
- TG peptides injected into the skin with GM-CSF adjuvant
- APCs pick up the TG RAS antigens

Train T-cells:

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

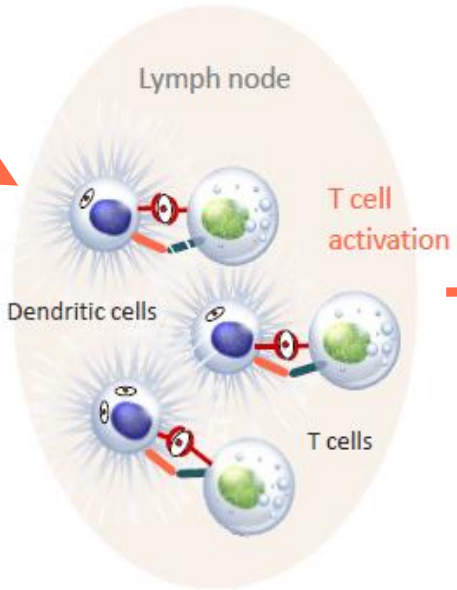
Attack the cancer:

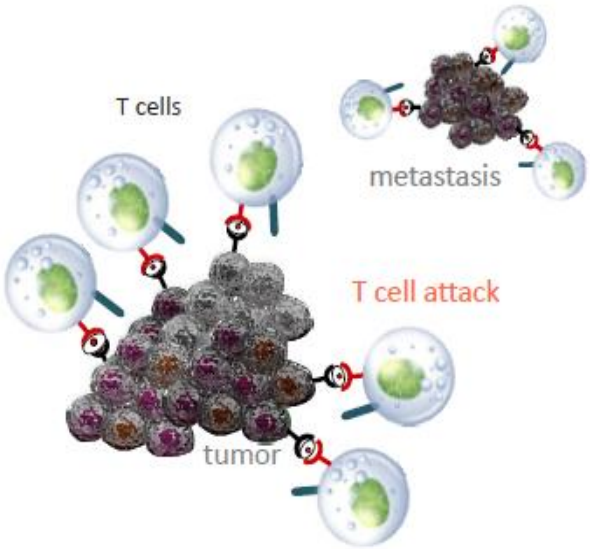
- RAS specific T-cells identify mutated RAS antigens on cancer cell surface
- Killer T-cells destroy the cancer cells



Cocktail of 7 peptides covering all relevant RAS mutations in pancreas

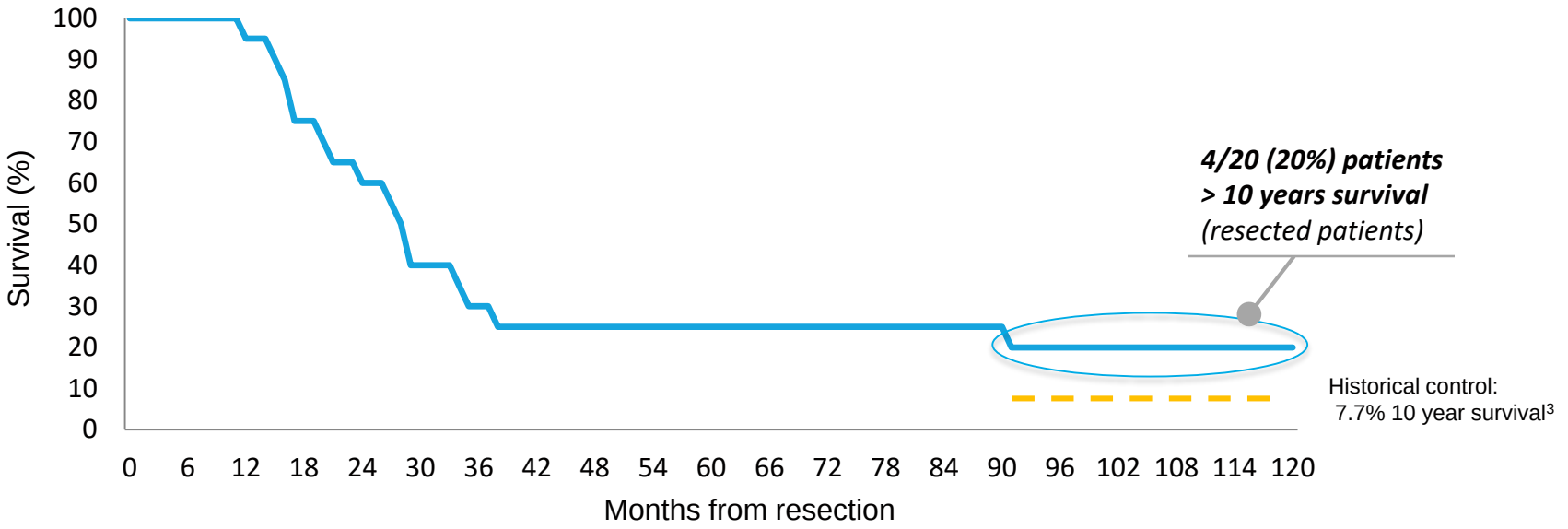
+ GM-CSF co-stimulation





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



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

(Clinical study report CTN RAS 98010 on file)

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

[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

Agenda

- Introduction to immunotherapy
- ONCOS-102 oncolytic virus platform
- TG RAS-peptide vaccine platform
- **Targovax clinical program overview**

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

	Cancer indication	Combined with		2017 H1	2017 H2	2018 H1	2018 H2	2019 H1
ONCOS-102	Melanoma	CPI		Phase I				
	Mesothelioma	Chemo~	Orphan ind.	Phase Ib/II				
	Ovarian & Colorectal	CPI	Orphan ind. Sponsor: Ludwig			Phase I/II		
	Prostate	DC therapy	Sponsor: Sotio				Phase I	
TG	Resected Pancreatic	Chemo~	Orphan ind.	Phase I/II				
	Colorectal	CPI				Phase Ib		

Interim data

Clinical, immune and safety data

4 readouts 2017

5 readouts 2018

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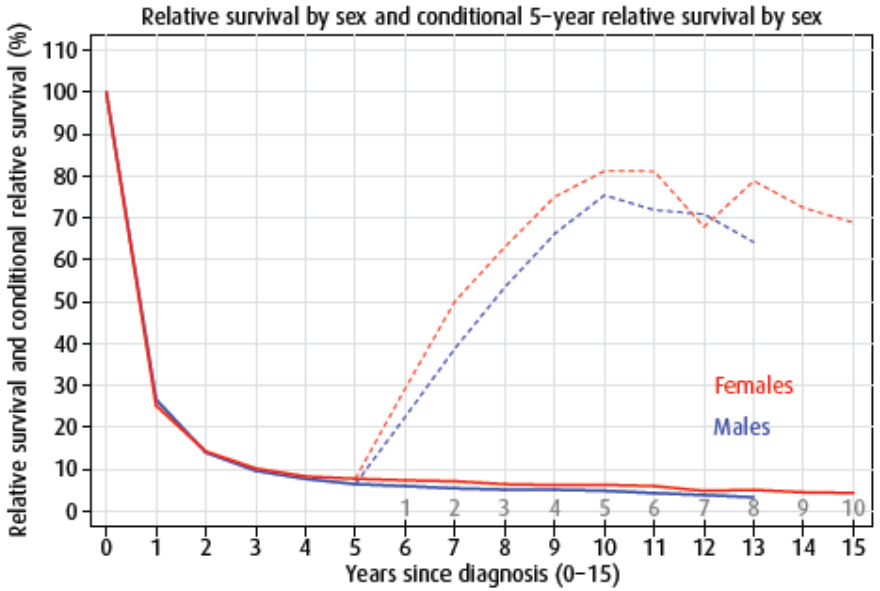
Targovax

Svein Dueland MD, Ph.D

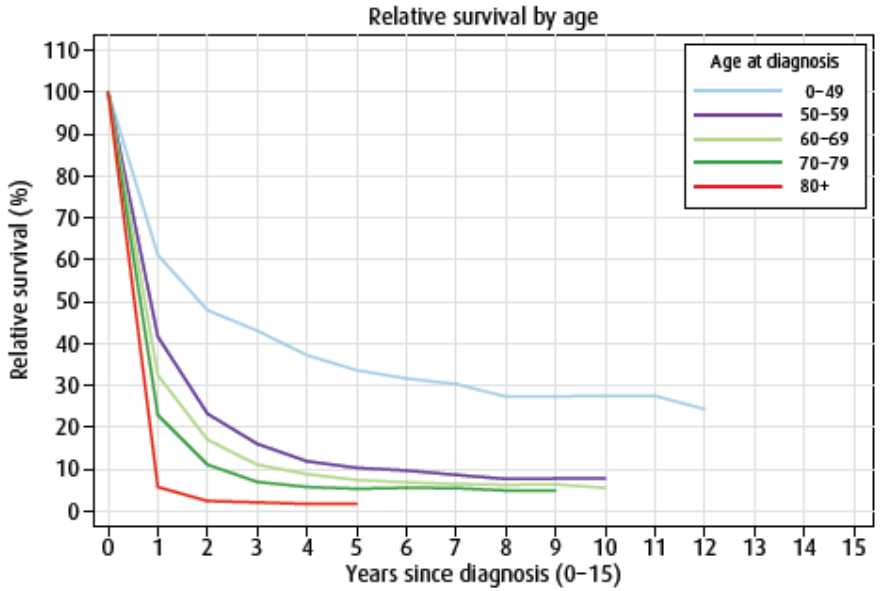
Oslo June 8th 2017

Cancer in Norway

Figure 10l: Pancreas (ICD-10 C25)



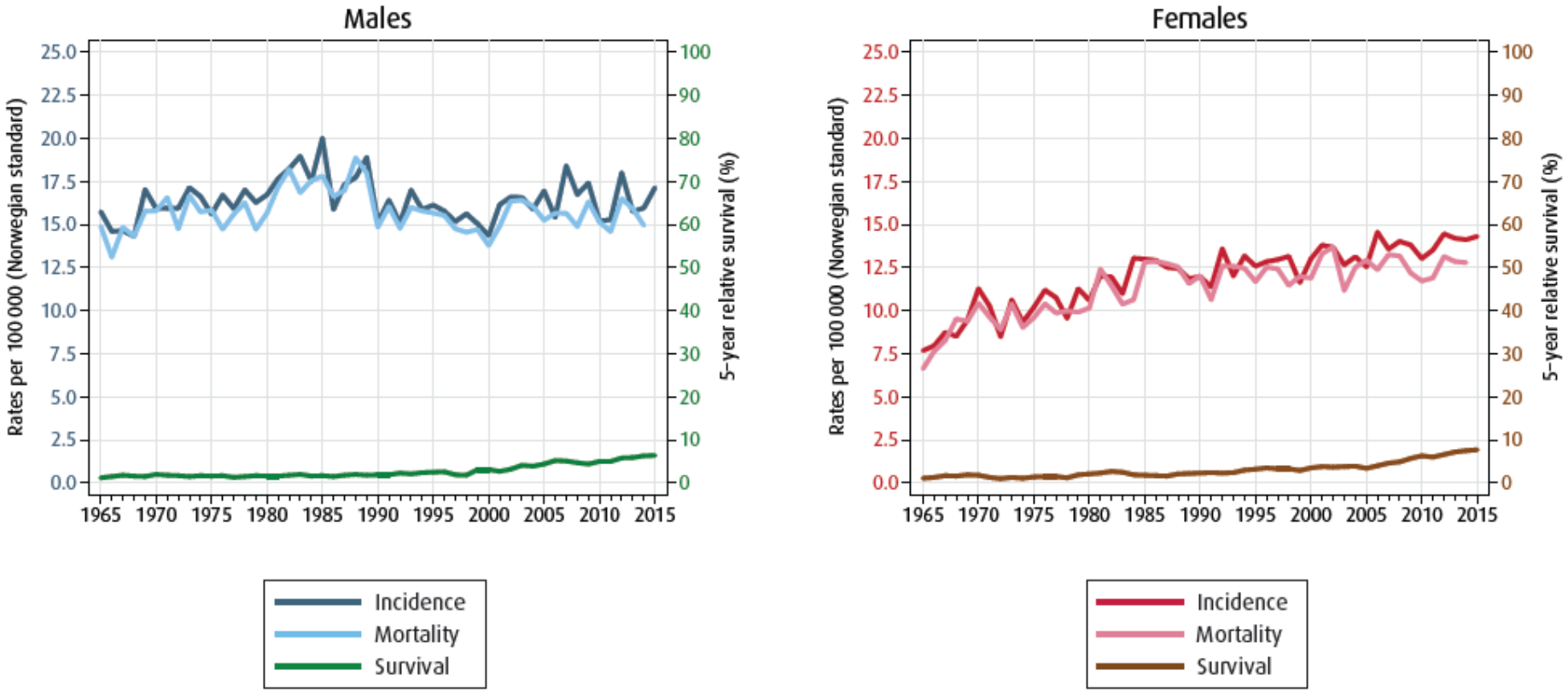
Dashed lines denote 5-year RS conditioned on surviving 1–10 years after diagnosis



Estimates are plotted if 20 or more patients are alive at start of the follow-up year

Cancer in Norway

Figure 11-I: Pancreas (ICD-10 C25)



Cancer in Norway

- New cases in 2015: 415 males and 410 females
- Deaths in 2014: 349 males and 364 females

- 5 year survival after diagnosed with pancreatic cancer is 6-8%
- Pancreatic cancer 4th most common cause of cancer related death both for males and females
- 10-20% of patients resectable

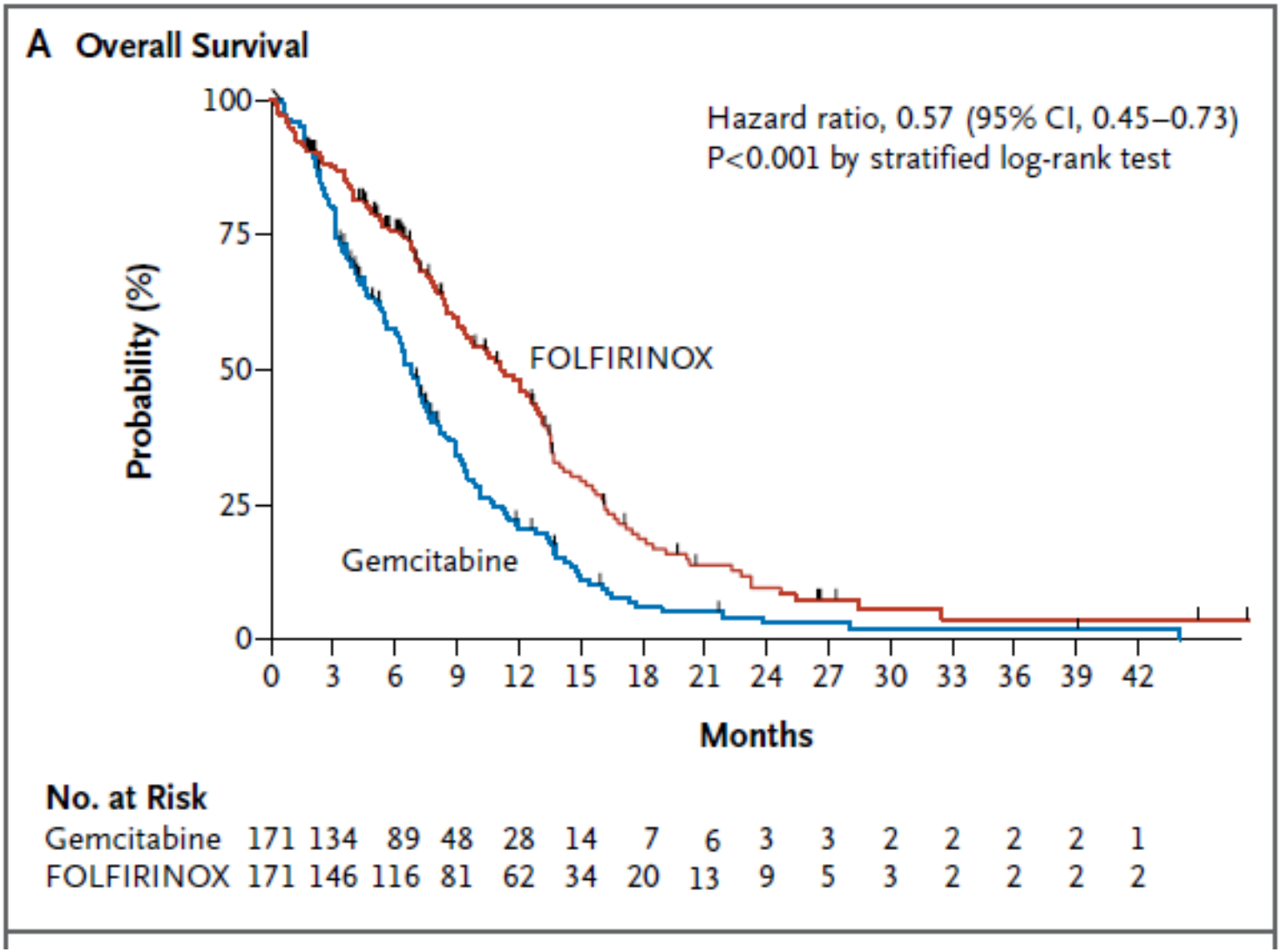
Metastatic disease

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

FOLFIRINOX versus Gemcitabine for Metastatic Pancreatic Cancer

Thierry Conroy, M.D., Françoise Desseigne, M.D., Marc Ychou, M.D., Ph.D., Olivier Bouché, M.D., Ph.D., Rosine Guimbaud, M.D., Ph.D., Yves Bécouarn, M.D., Antoine Adenis, M.D., Ph.D., Jean-Luc Raoul, M.D., Ph.D., Sophie Gourgou-Bourgade, M.Sc., Christelle de la Fouchardière, M.D., Jaafar Bennouna, M.D., Ph.D., Jean-Baptiste Bachet, M.D., Faiza Khemissa-Akouz, M.D., Denis Péré-Vergé, M.D., Catherine Delbaldo, M.D., Eric Assenat, M.D., Ph.D., Bruno Chauffert, M.D., Ph.D., Pierre Michel, M.D., Ph.D., Christine Montoto-Grillot, M.Chem., and Michel Ducreux, M.D., Ph.D., for the Groupe Tumeurs Digestives of Unicancer and the PRODIGE Intergroup*



Folfirinox, median OS 11,1 moths
Gemcitabine, median OS 6,8 months

ESPAC-1 study

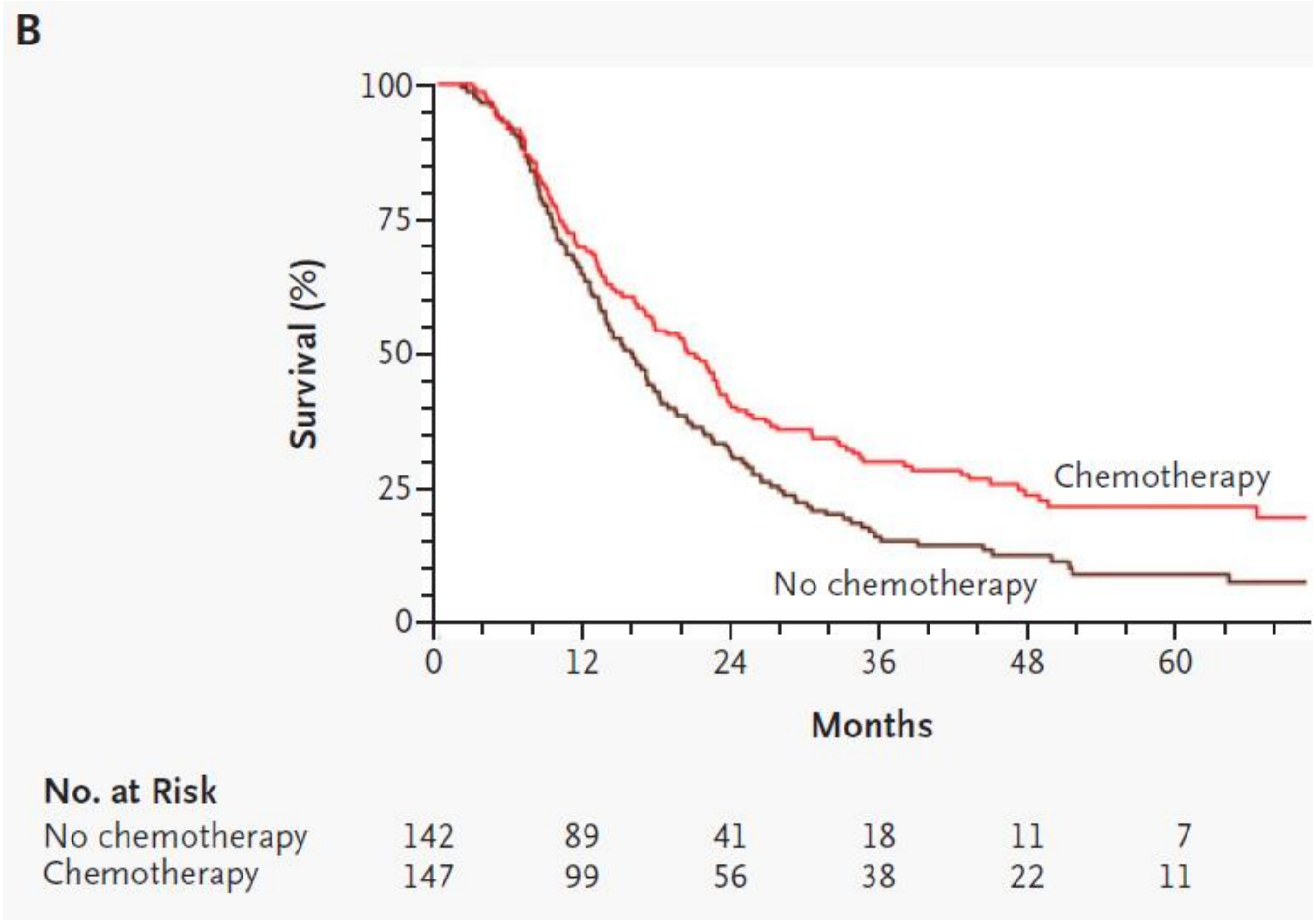
The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

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

ESPAC-1 study



Conko-001 study

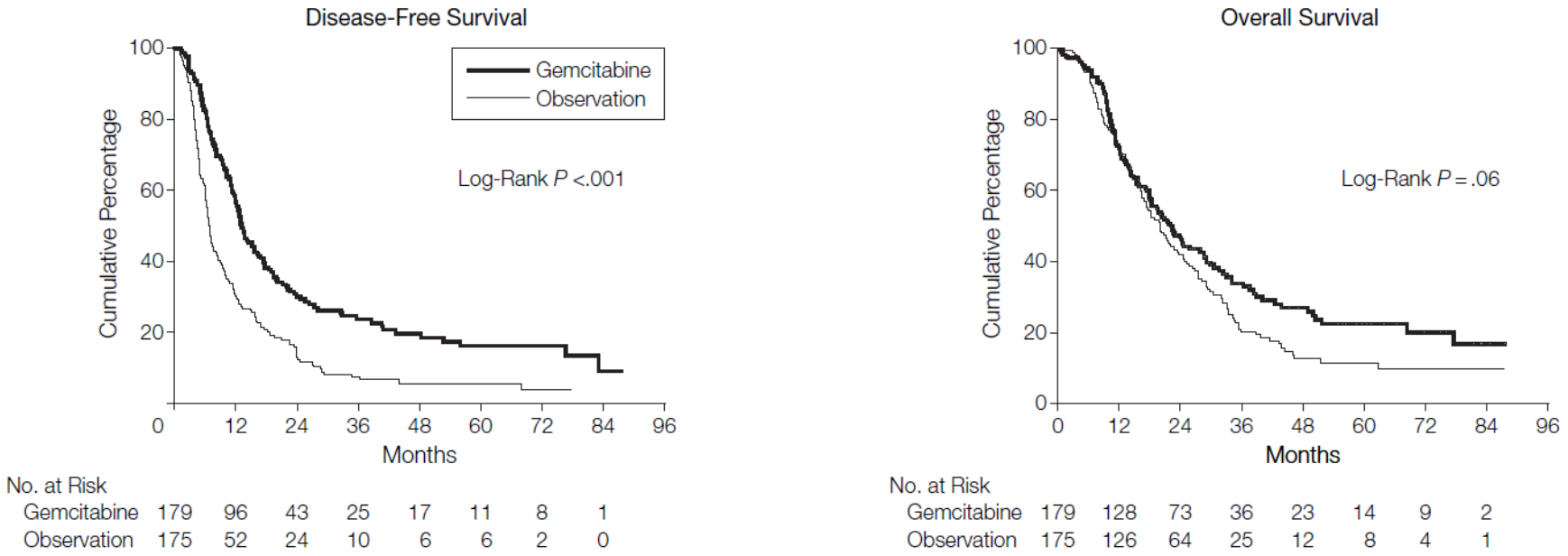
Adjuvant Chemotherapy With Gemcitabine vs Observation in Patients Undergoing Curative-Intent Resection of Pancreatic Cancer

A Randomized Controlled Trial

Oettle et al. JAMA 2007

Conko-001 study

Figure 2. Disease-Free and Overall Survival (Intent-to-Treat Analysis)



ESPAC-3

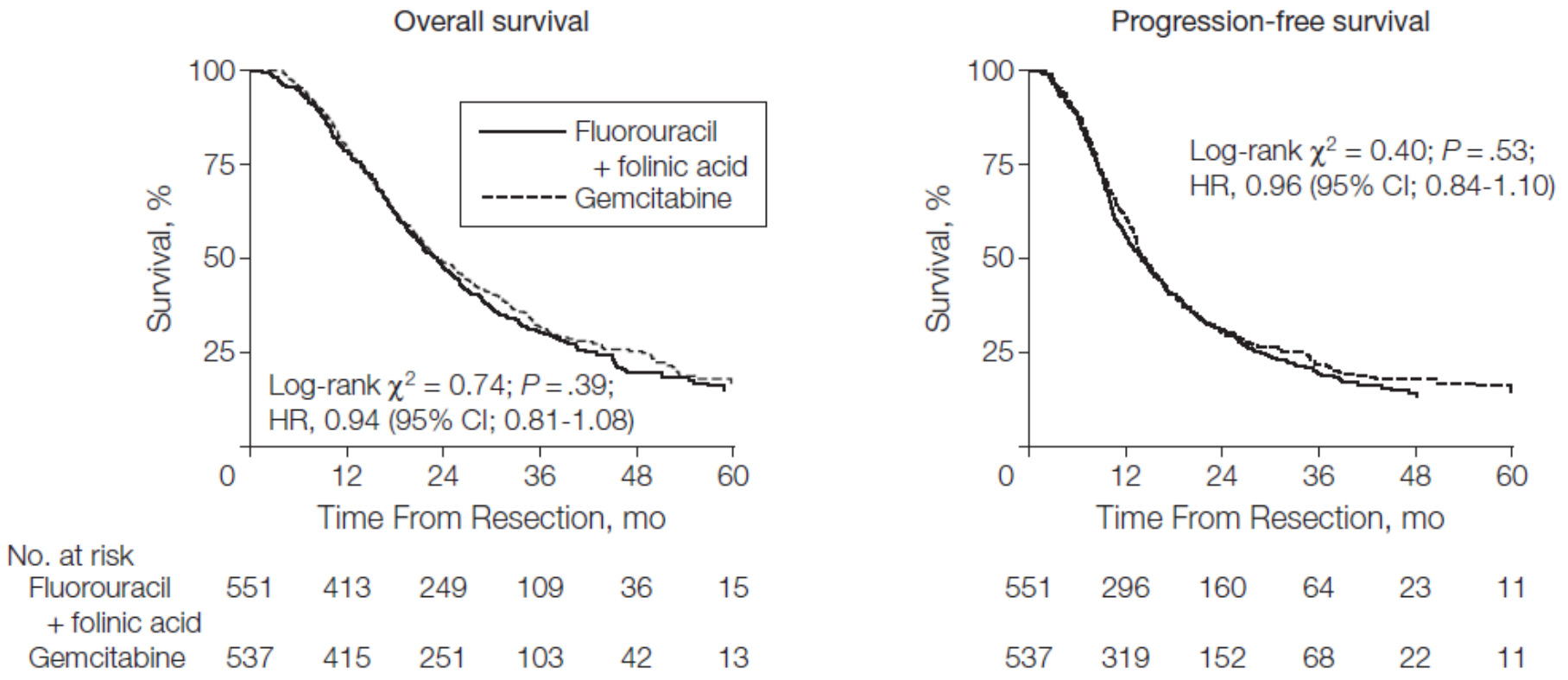
Adjuvant Chemotherapy With Fluorouracil Plus Folinic Acid vs Gemcitabine Following Pancreatic Cancer Resection

A Randomized Controlled Trial

Neoptolemos et al. JAMA 2010

ESPAC-3

Figure 2. Survival Results by Randomized Treatment



No. at risk						
Fluorouracil + folinic acid	551	413	249	109	36	15
Gemcitabine	537	415	251	103	42	13

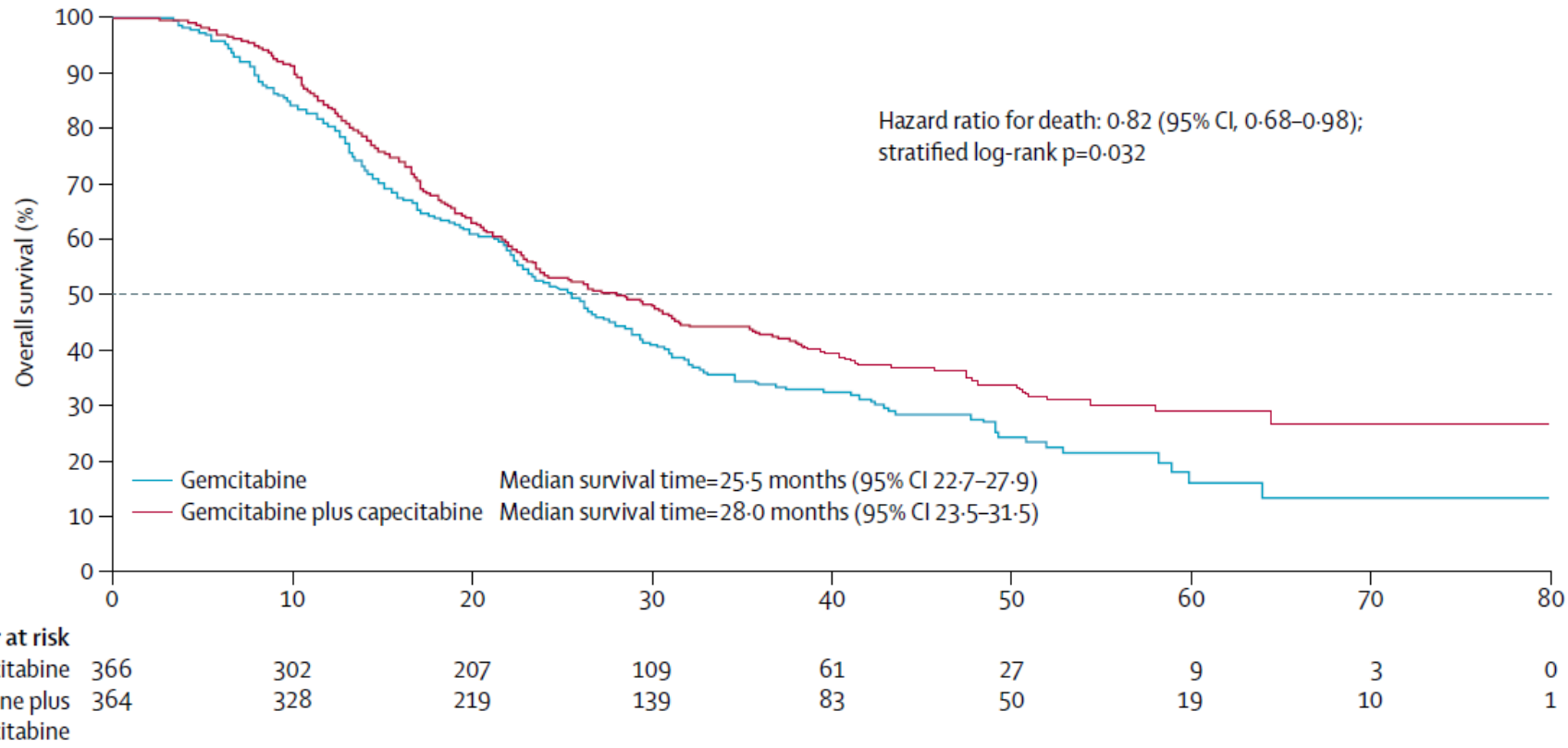
551	296	160	64	23	11
537	319	152	68	22	11

ESPAC-4

Comparison of adjuvant gemcitabine and capecitabine with gemcitabine monotherapy in patients with resected pancreatic cancer (ESPAC-4): a multicentre, open-label, randomised, phase 3 trial

John P Neoptolemos, Daniel H Palmer, Paula Ghaneh, Eftychia E Psarelli, Juan W Valle, Christopher M Halloran, Olusola Faluyi, Derek A O'Reilly, David Cunningham, Jonathan Wadsley, Suzanne Darby, Tim Meyer, Roopinder Gillmore, Alan Anthoney, Pehr Lind, Bengt Glimelius, Stephen Falk, Jakob R Izbicki, Gary William Middleton, Sebastian Cummins, Paul J Ross, Harpreet Wasan, Alec McDonald, Tom Crosby, Yuk Ting Ma, Kinnari Patel, David Sherriff, Rubin Soomal, David Borg, Sharmila Sothi, Pascal Hammel, Thilo Hackert, Richard Jackson, Markus W Büchler, for the European Study Group for Pancreatic Cancer

ESPAC-4 overall survival



ESPAC-4

- All 6 cycles of chemotherapy were given to **65%** of patients in the gemcitabine group and **54%** of patients in the gemcitabine+capecitabine group

		Median OS	5 yr OS
ESPAC-1	Control	16.1mo	8.0%
	5-FU	19.7mo	21.1%
ESPAC-3	Gemcitabine	23.6mo	17.5%
	5-FU	23.0mo	15.9%
Conko-001	Control	20.2mo	11.5%
	Gemcitabine	22.1mo	22.5%
ESPAC-4	Gemcitabine	25.5mo	16.3%
	Gemcitabine+Capecitabine	28.0mo	28.8%

Acta Oncologica, 2016; 55: 265–277

Taylor & Francis
Taylor & Francis Group

ORIGINAL ARTICLE

Impact of early disease progression and surgical complications on adjuvant chemotherapy completion rates and survival in patients undergoing the surgery first approach for resectable pancreatic ductal adenocarcinoma – A population-based cohort study

KNUT J. LABORI[1], MATTHEW H. KATZ[2], CHING W. TZENG[3], BJØRN A. BJØRNBETH[1], MILADA CVANCAROVA[4], BJØRN EDWIN[5,6], ELIN H. KURE[7], TOR J. EIDE[6,8], SVEIN DUELAND[9], TROND BUANES[1,6] & IVAR P. GLADHAUG[1,6]

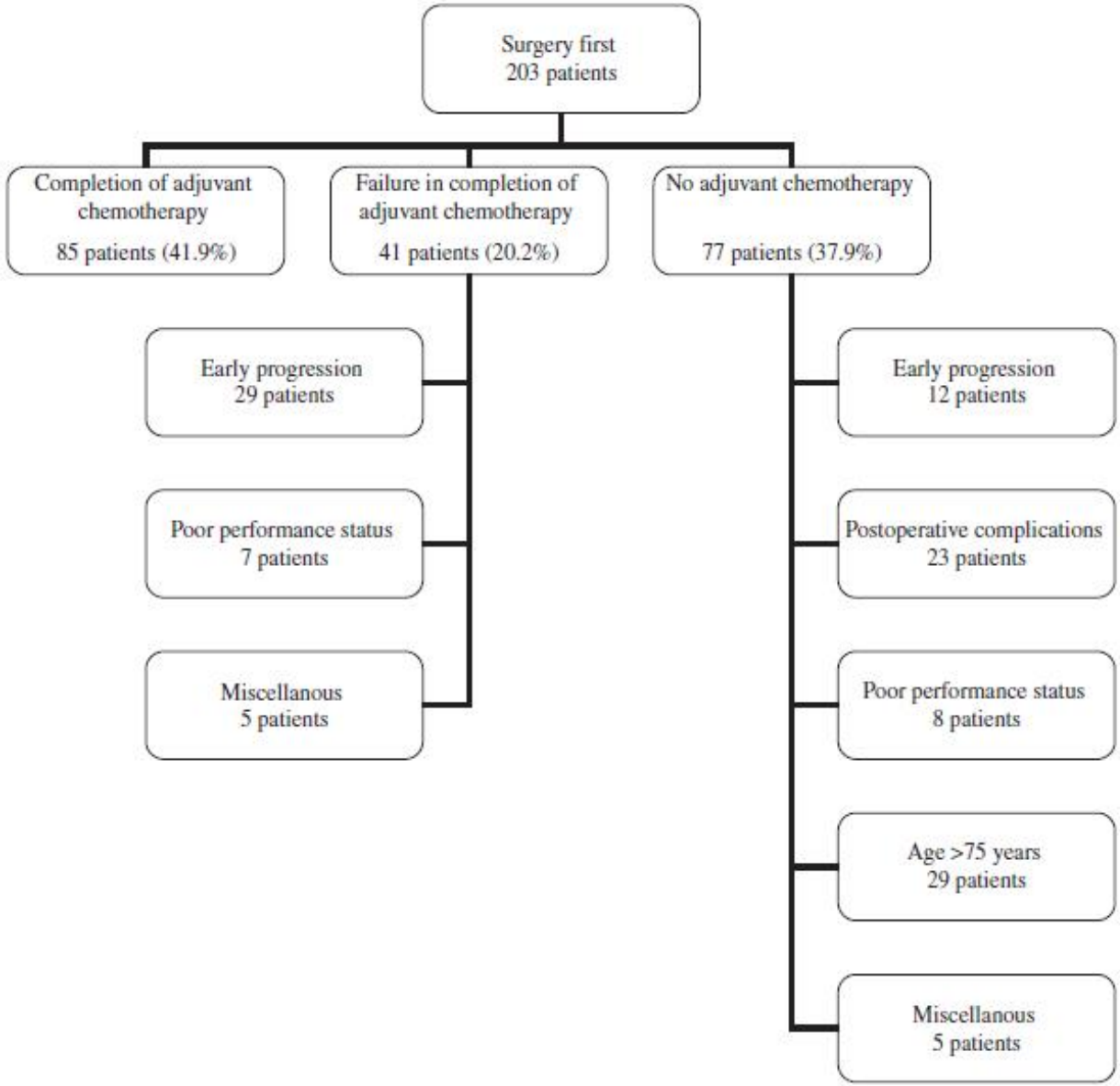
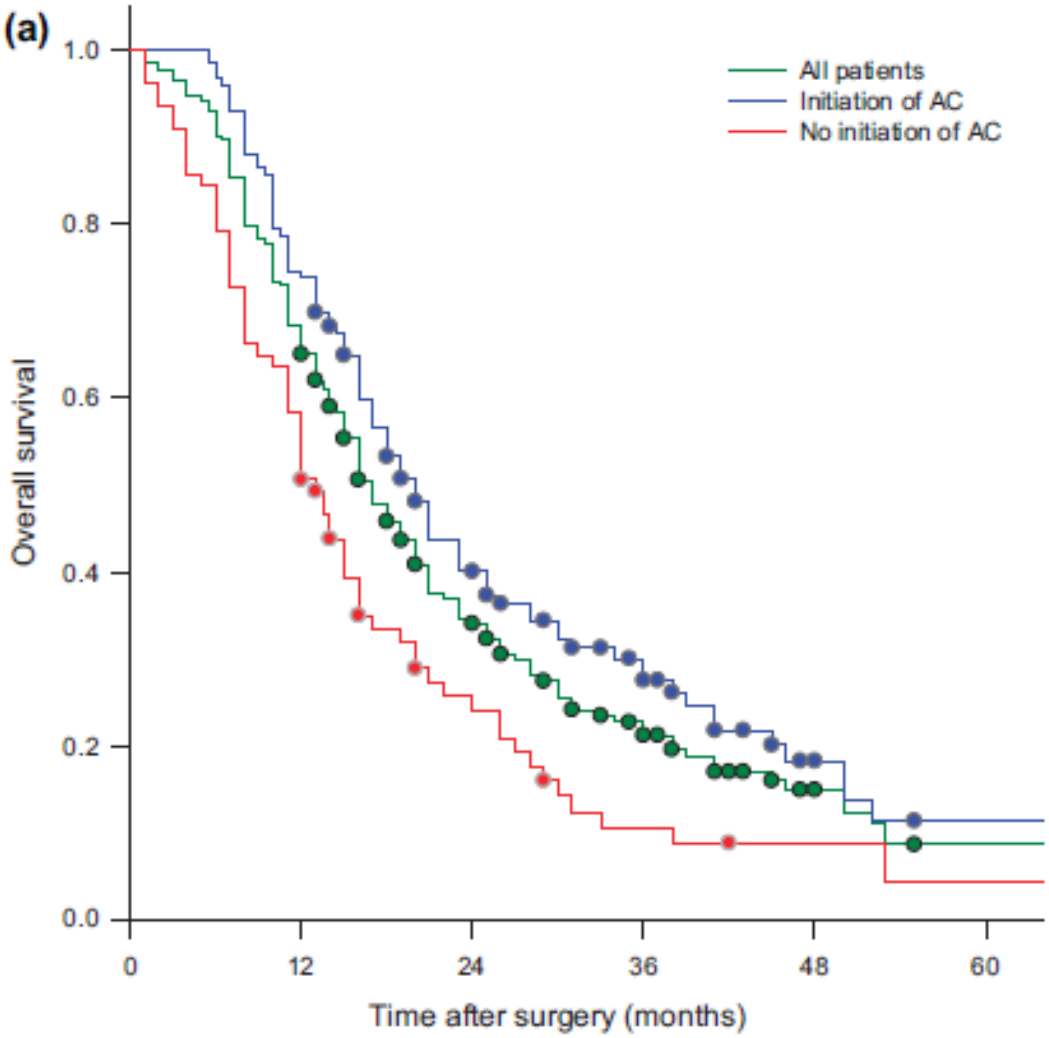
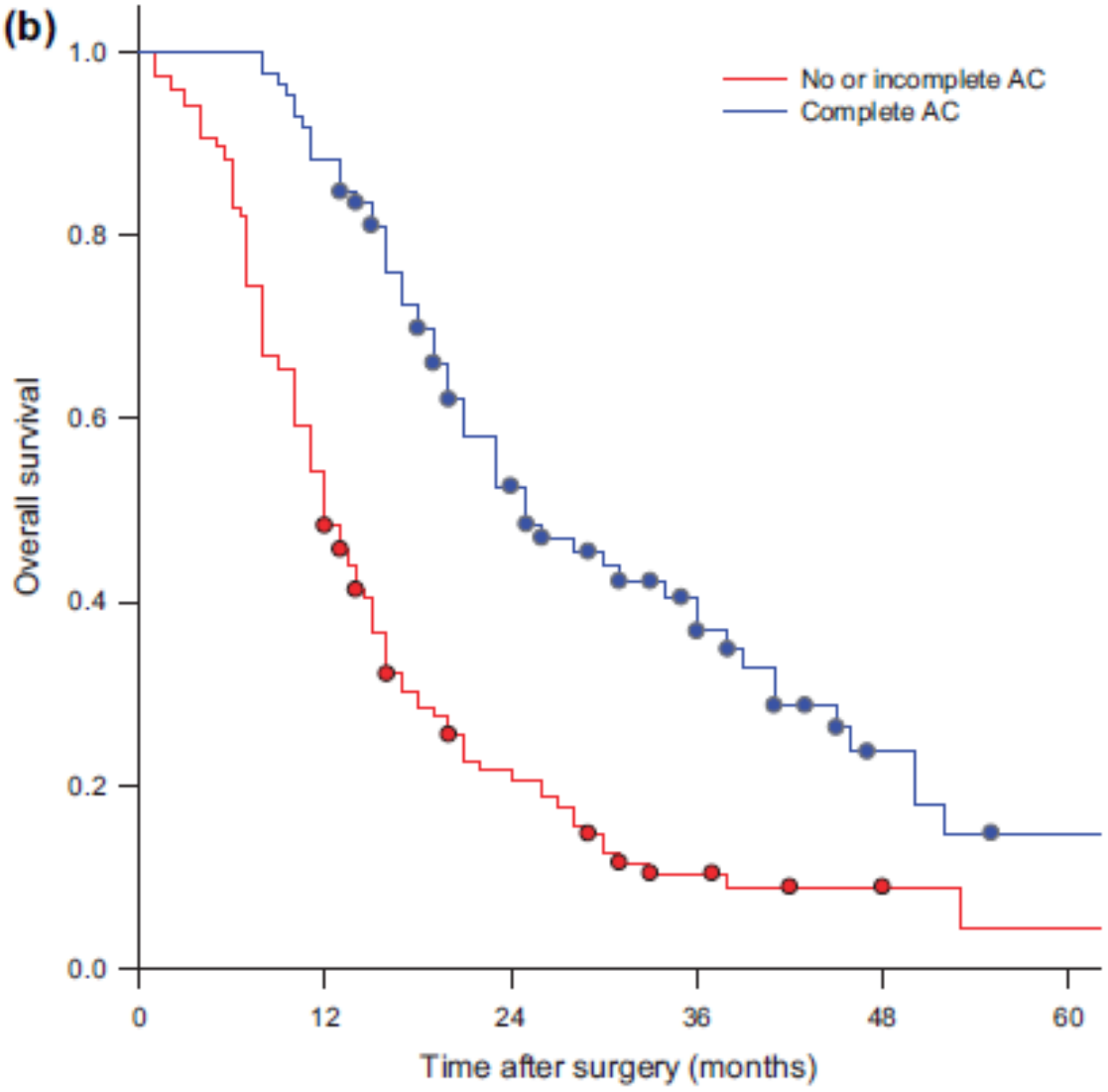


Figure 1. Reasons for not initiating or completing adjuvant chemotherapy.

OS



All 17.0 months
Started 20.0 months
Completed 25.0
Not completed 12.0 months



Completed 25.0 months

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

TG01-01 Background

TG01

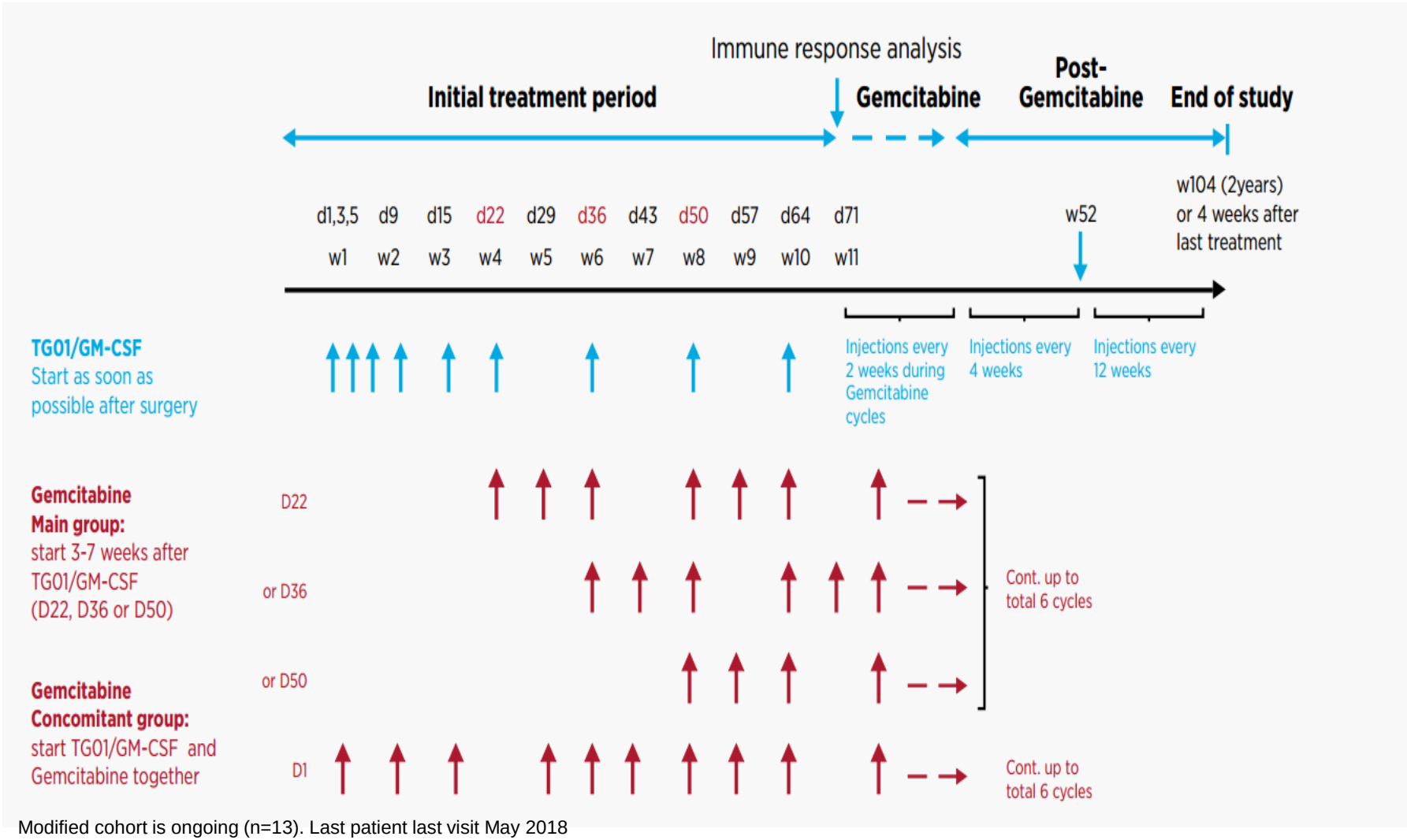
- injectable antigen-specific cancer immunotherapy
- targeted to treat patients with RAS mutation
  - found in more than 85% of pancreatic adenocarcinomas
- a mixture of 7 synthetic peptides
  - representing 7 of the most common 12 & 13 mutations in RAS assoc. with human cancer
- induces RAS mutant-specific T-cell responses which are enhanced by co-administration of GM-CSF

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

TG01-01 Endpoints

Primary

- Safety
  - Adverse events
  - Laboratory assessments
- Immune response
  - DTH responses
  - Proliferative T-cell responses

Secondary

- Disease free survival at 2 years
- Overall survival at 2 years

Exploratory

- Relationship between KRAS status in resected primary tumour and recurrence survival outcomes (including disease recurrence and overall survival)
- Monitor CA19-9 levels

TG01-01 Immunological assessment

- Two different antigen specific assays were used to asses the immunological response to TG01:
  - Delayed Type Hypersensitivity (DTH) skin test
    - A delayed hypersensitivity test (DTH) is an immune function test measuring the presence of activated T cell s that recognize TG01
    - The DTH-test is considered positive if the area of the skin reacts = immune system has responded to TG01
    - Measured by the patient (max 9 times)
  - T-cell proliferation assay*
    - In vitro assay showing proliferation response of TG01 specific T-cells after vaccination with TG01
    - Blood sampling and PBMC isolation on day 1, week 11, week 52 and end of study
    - T-cell responses are considered positive if the stimulation index (SI) is ≥2 indicating an increase in proliferation of TG01 specific T-cells after stimulation with peptide = the immune system has produced TG01 specific T-cells

* The analytical viability of the assay depends on: sample collection (time point), patient condition, isolation procedure, cell viability, proliferation assay, chemotherapy. Test may show false negative but not false positive

TG01-01 Patient disposition main cohort

	Number of patients (N=19)
Treated patients	19 (100%)
Patients completed study	1 (5%)
Patients prematurely discontinued	18 (95%)
Reason for withdrawal:	
Consent withdrawn	3 (16%)
Adverse event	4 (21%)
Death	2 (11%)*
Investigator decision	2 (11%)
Disease recurrence	7 (37%)

* Reason for death: pneumonia and disease progression, not treatment related

TG01-01 Baseline characteristics main cohort

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

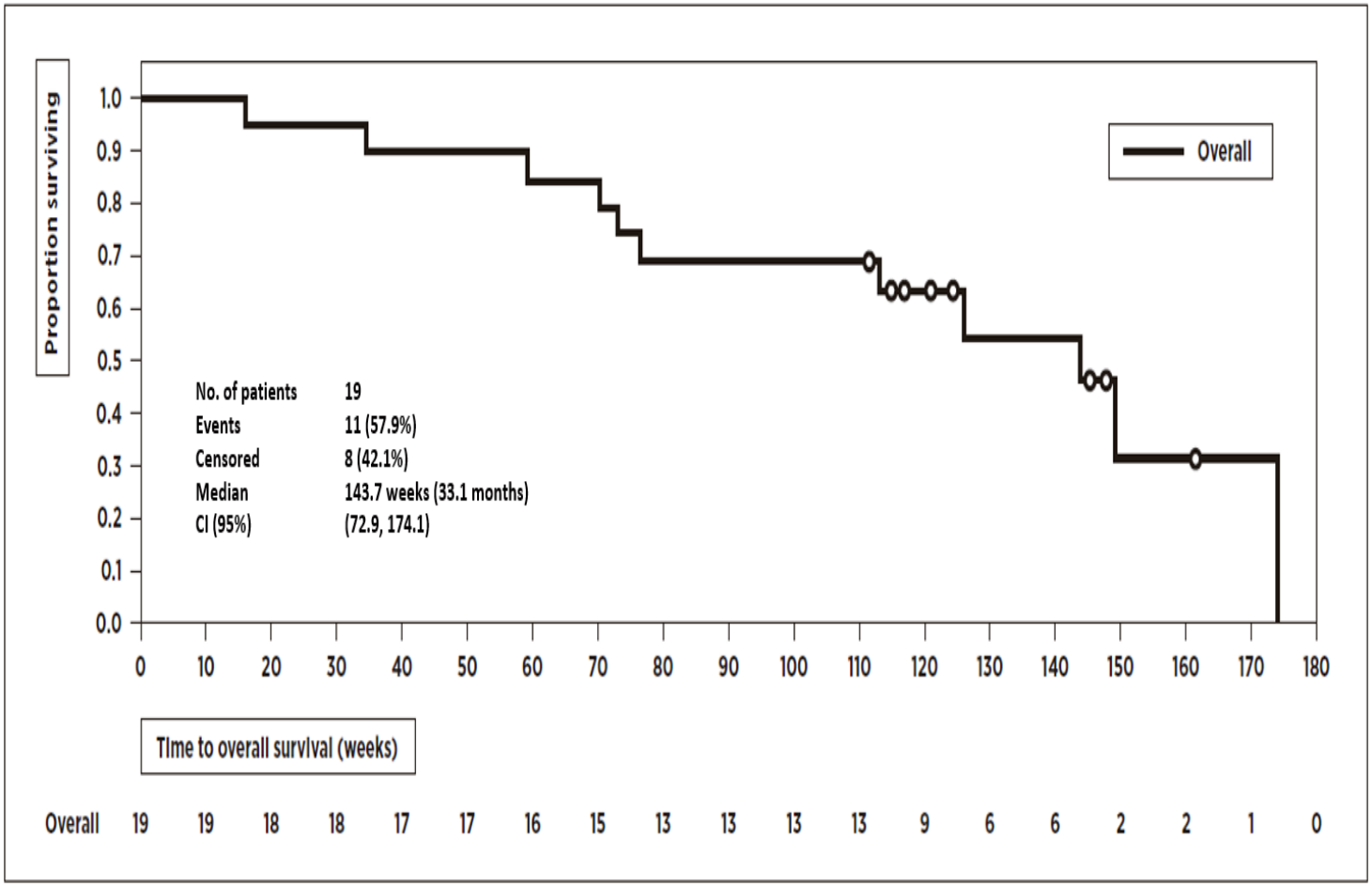
TG01-01 Efficacy (survival rate from resection)

	1 year*	2 years*
Assessed from resection (8 weeks before first IMP)	17/19 (90%)	13/19 (68%)

*This rate compares favorably with the available published historical two-year survival rates of resected cancer patients treated with gemcitabine alone of between 30% and 53%[1-5]

1 Neoptolemos JP et al.; JAMA; 304(10):1073-81 (2010)
2 Van Laethem J-L et al.; J. Clin. Onc.; 28(29):4450-56 (2010)
3 Oettle H, et al.; JAMA; 310(14):1473-81 (2013)
4 Sinn M, et al.;J Clin Oncol; 33(suppl; abstr 4007) (2015)
5 Uesaka K et al.;The Lancet; 388:248-257 (2016)

TG01-01 Overall survival (from resection)



In a recently published study (ESPAC-4) the OS in patients receiving gemcitabine alone was 27.6 months from resection (Neoptolemos JP et al.; The Lancet; 389:1011-1024 (2017))

Targovax patient

- Surgery 08.01.2014, N+ (1/8 lymph nodes), R1 resection
- 1. Gemcitabine treatment 18.03.2014
- CT thorax scan 20.06.2014 unspecific pulmonary lesions 5mm in size
- Last adjuvant gemcitabine treatment 19.08.2014
- CT thorax scan 06.07.2015 lesions increased in size to 15 mm, several lesions a few mm -2cm, pulmonary metastases diagnosed
- 1. Gemcitabine in metastatic setting 06.08.2015
- Death 08.06.2016

TG01-01 Immune response

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

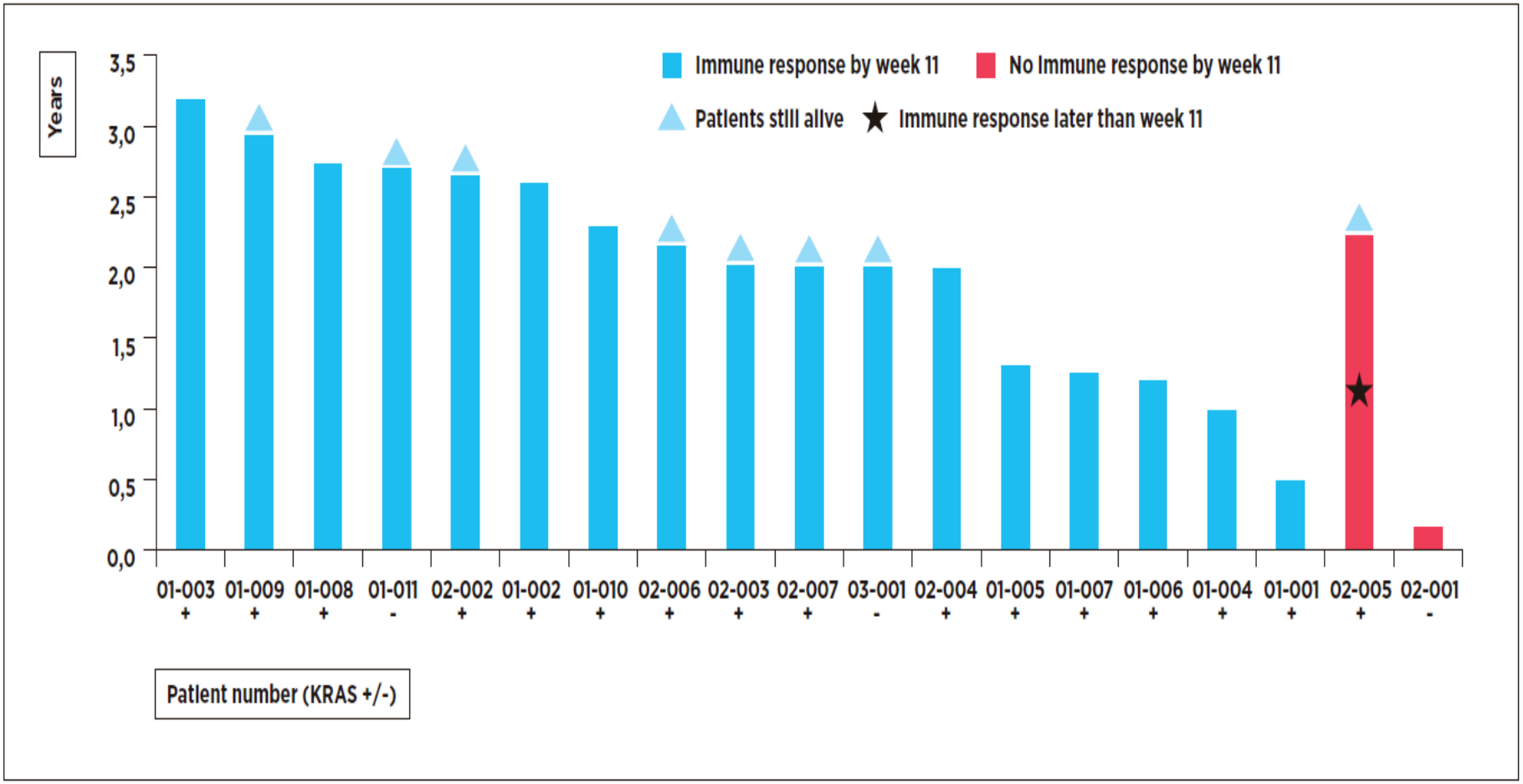
Study period	Immune responders	Immune responders DTH	Immune responders T-cells
By end of initial treatment (week 11)	17/19 (89%)	16/19 (84%)	10/19* (53%)
Entire study period	18/19 (95%)	18/19 (95%)	14/19* (74%)

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

Immune response after week 11 (n=11)

Study time point	No. of pts with immune monitoring after week 11	Patients with positive immune response after week 11
After week 11	11	9/11 (82%)

TG01-01 Overall survival and immune response



TG01-01 Safety profile (N=19)

Serious Adverse Events Preferred term	Number of Events	Relationship to study treatment
Anaphylactic reaction	2	Related to TG01 +/- GM-CSF
Hypersensitivity	1	
Dyspnea	1	Related to Gemcitabine and TG01/GM-CSF
Lung infection	1	Related to Gemcitabine
Pyrexia (fever)	2	
Anaemia	1	
Anaphylactic shock related to a concomitant medication (Emend)	1	Unrelated to study treatments
Hyperglycemia	1	
Urosepsis	1	
Pneumonia	1	
Viral upper respiratory tract infection	1	

TG01-01 Safety profile (N=19)

Grade 3/4 treatment emergent Adverse Events

SOC	Grade 3		Grade 4	
Adverse event	**Patients**	**Events**	**Patients**	**Events**
Any adverse event	13	32	5	6
Blood and lymphatic system disorders				
Neutropenia*	6	6	1	1
Anaemia	1	1		
Gastrointestinal disorders				
Abdominal pain	2	2		
Diarrhoea	1	1		
Abdominal pain upper	1	1		
General disorders and administration site conditions				
Fatigue	1	1		
Immune system disorders				
Anaphylactic reaction			2	2
Anaphylactic shock	1	1		
Infections an dinfestations				
Urosepsis	1	1		
Investigations				
Neutrophil count decreased*	4	7	1	1
Hemoglobin decreased	1	1		
Platelets count decreased	1	1		
Metabolism and nutrition disorders				
Hyperglycaemia	1	1	2	2
Diabetes mellitus	1	1		
Hypokalaemia	1	1		
Psychiatric disorders				
Depression	1	1		
Respiratory, thoracic and mediastinal disorders				
Pulmonary embolism	1	1		
Vascular disorders				
Hypertension	3	4		

* All reported neutropenia and neutrophil count decreased were related to chemotherapy.

Toxicity

	Gemcitabine (n=366)			Gemcitabine plus capecitabine (n=359)			p value grade 1–2	p value grade 3–4
	Grade 1–2	Grade 3–4	Grade 5	Grade 1–2	Grade 3–4	Grade 5		
Anaemia	213 (58%)	14 (4%)	0	201 (56%)	8 (2%)	0	0·549	0·279
Diarrhoea	151 (41%)	6 (2%)	0	161 (45%)	19 (5%)	0	0·331	0·008
Fatigue	241 (66%)	19 (5%)	0	230 (64%)	20 (6%)	0	0·641	0·870
Fever	74 (20%)	6 (2%)	0	62 (17%)	6 (2%)	0	0·342	1·000
Infection and infestations, other	56 (15%)	24 (7%)	0	37 (10%)	9 (3%)	1 (<1%)	0·046	0·012
Lymphocyte count decreased	100 (27%)	11 (3%)	0	78 (22%)	9 (3%)	0	0·085	0·821
Neutropenia	147 (40%)	89 (24%)	0	175 (49%)	137 (38%)	0	0·021	0·0001
Hand-foot syndrome	8 (2%)	0	0	111 (31%)	26 (7%)	0	<0·0001	<0·0001
Platelets	87 (24%)	7 (2%)	0	104 (29%)	8 (2%)	0	0·129	0·800
Thromboembolic events	7 (2%)	9 (2%)	0	16 (4%)	8 (2%)	0	0·058	1·000
White blood cell count decreased	136 (37%)	28 (8%)	0	141 (39%)	37 (10%)	0	0·593	0·242
Acute kidney injury	4 (1%)	2 (1%)	0	1 (<1%)	0	0	0·373	0·499
Multi-organ failure	0	0	1 (<1%)	0	0	0	NA	NA
Cardiac disorders	4 (1%)	1 (<1%)	1 (<1%)	3 (1%)	0	0	1·000	1·000
Benign, malignant, and unspecified neoplasms (including cysts and polyps), other	1 (<1%)	0	3 (1%)	0	1 (<1%)	0	1·000	0·495

Fisher's exact test was used to show statistically significant differences between the two groups. NA=not applicable.

Table 5: **Grade 1–5 adverse events with gemcitabine alone and gemcitabine plus capecitabine**

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.
- OS was encouraging in view of published reports with a median OS of 33.1 months.
- We believe that the immune activation at both DTH and PBMC level is associated with the positive clinical findings.

Capital Markets Update - Agenda

- Introduction – *CEO, Øystein Soug*
- Targovax's technology and trials – *CMO, Dr Magnus Jäderberg*
- A physician's view on pancreatic cancer – *Dr Svein Dueland*
- **Financial update – *CFO, Erik Wiklund***
- Q&A
- Lunch

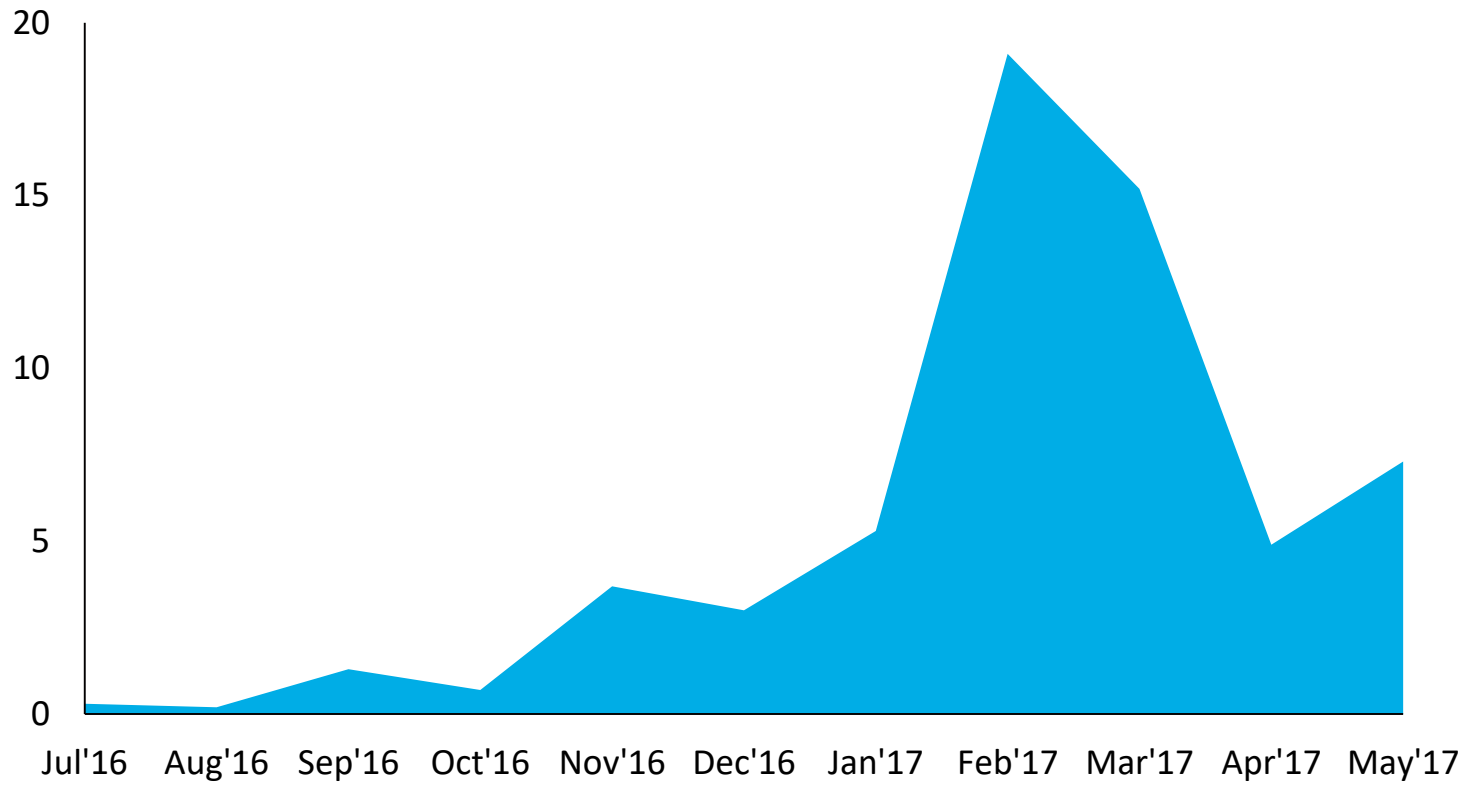
Financial summary – end of Q1 2017

Operations			
Cash	**NOK 147m**	USD 17m	*End of Q1 2017*
Net cash flow	**NOK -24m**	USD -3m	*Total Q1*
Annual run rate	**NOK 104m**	USD 12m	*Last four quarters*
Annual opex	**NOK 116m**	USD 13m	*Last four quarters*

The share	OSE: TRVX		
Market Cap	**NOK ~1bn**	USD ~120m	*At share price NOK ~24*
Daily turnover	**NOK 14m**	USD 1.6m	*Last three months avg.*
Debt	**NOK 43m**	USD 5m	*EUR 6m conditional*
No. of shares	**42.2m**		*46.0m fully diluted per April 18*
Analysts	DNB, ABG Sundal Collier, Arctic, Redeye, Norske Aksjeanalyser		

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 ~1.2b market cap***
- ***NOK 9m 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)*

Strong shareholder base as per May 2017

Shareholder		Estimated ownership	
		Shares m	*Relative*
HealthCap	Sweden	11,2	26,4 %
RadForsk	Norway	4,1	9,7 %
Nordea	Norway	3,0	7,0 %
Nordnet Livsforsikring	Norway	1,5	3,5 %
KLP	Norway	1,3	3,1 %
Statoil	Norway	0,9	2,2 %
Danske Bank (nom.)	Norway	0,8	1,9 %
Timmuno AS	Norway	0,7	1,7 %
Prieta AS	Norway	0,7	1,7 %
Nordnet Bank AB (nom.)	Sweden	0,7	1,6 %
Thorendahl Invest AS	Norway	0,3	0,7 %
Sundt AS	Norway	0,3	0,7 %
Netfonds Livsforsikring AS	Norway	0,3	0,6 %
Avanza Bank AB (nom.)	Sweden	0,3	0,6 %
The Bank of NY Mellon (nom.	Belgium	0,2	0,5 %
Tobech Invest AS	Norway	0,2	0,5 %
Istvan Molnar	Norway	0,2	0,4 %
Danske Bank (nom.)	Norway	0,2	0,4 %
NHO - P665AK	Norway	0,2	0,4 %
Kristian Falnes AS	Norway	0,2	0,4 %
Top 20		**27,0**	**64,1 %**
Other shareholders (3772)		*15,2*	*35,9 %*
Total		**42,2**	**100,0 %**

42.2m ordinary shares

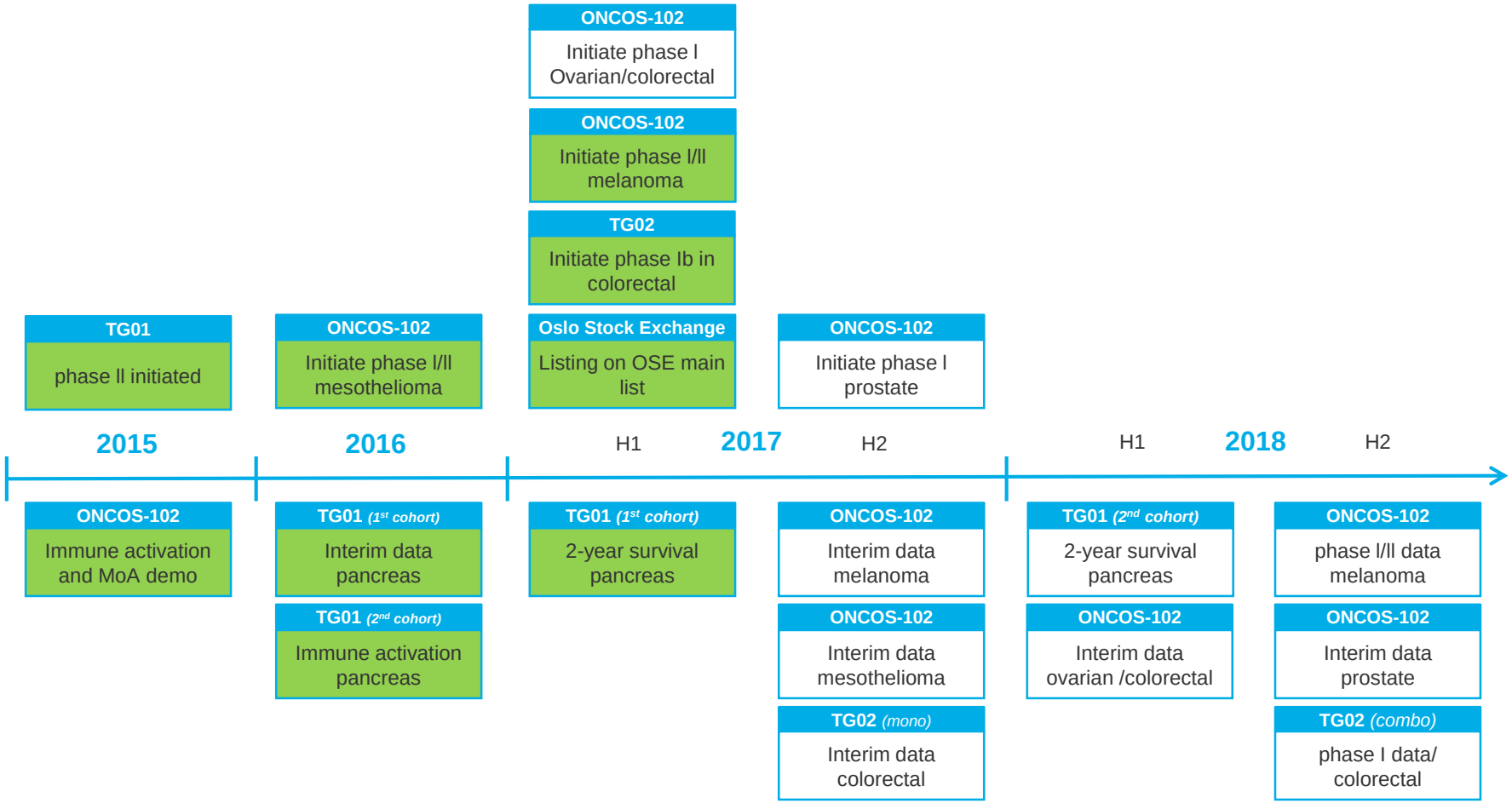
- Management ownership: 2.1%
- 3,792 shareholders

46.0m[1] shares fully diluted

- Average strike price on options ~NOK 21
- Total dilutive effect of options is 7.9%

[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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