

# 2018 Capital Markets Day

September 26, 2018  
Live in Utrecht and via Webcast



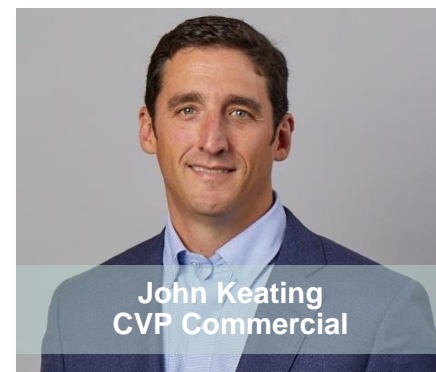
# Forward Looking Statement

This presentation contains forward looking statements. The words “believe”, “expect”, “anticipate”, “intend” and “plan” and similar expressions identify forward looking statements. All statements other than statements of historical facts included in this presentation, including, without limitation, those regarding our financial position, business strategy, plans and objectives of management for future operations (including development plans and objectives relating to our products), are forward looking statements. Such forward looking statements involve known and unknown risks, uncertainties and other factors which may cause our actual results, performance or achievements to be materially different from any future results, performance or achievements expressed or implied by such forward looking statements. Such forward looking statements are based on numerous assumptions regarding our present and future business strategies and the environment in which we will operate in the future. The important factors that could cause our actual results, performance or achievements to differ materially from those in the forward looking statements include, among others, risks associated with product discovery and development, uncertainties related to the outcome of clinical trials, slower than expected rates of patient recruitment, unforeseen safety issues resulting from the administration of our products in patients, uncertainties related to product manufacturing, the lack of market acceptance of our products, our inability to manage growth, the competitive environment in relation to our business area and markets, our inability to attract and retain suitably qualified personnel, the unenforceability or lack of protection of our patents and proprietary rights, our relationships with affiliated entities, changes and developments in technology which may render our products obsolete, and other factors. Further, certain forward looking statements are based upon assumptions of future events which may not prove to be accurate. The forward looking statements in this document speak only as at the date of this presentation.

# Agenda

14.00	Welcome	Introduction	Jan van de Winkel, President & CEO
14.10	Innovative Excellence: New Proprietary Technology	HexElect	Janine Schuurman, Corporate VP, Research & Innovation
14.20	Future Leapfrog Products: Pre-clinical Candidates	DuoHexaBody-CD37	Esther Breij, Director, Translational Research David Satijn, VP, New Antibody Products
14.30		DuoBody-CD40x4-1BB & DuoBody-PD-L1x4-1BB	
14.35	Q&A		
14.55	Reaching Our Inspirational Vision: Late Stage Clinical Pipeline	Tisotumab vedotin	Judith Klimovsky, Executive VP & CDO
15.05	Reaching Our Inspirational Vision: Early Stage Clinical Pipeline	Enapotamab vedotin (HuMax-AXL-ADC) GEN3013 (DuoBody-CD3xCD20) GEN1029 (HexaBody-DR5/DR5)	Tahi Ahmadi, Senior VP, Oncology and Translational Medicine
15.25	Building the Business: Translational Research	Building a world-class translational research unit	Kate Sasser, Corporate VP, Translational Research
15.30	Building the Business: Commercial Capabilities	Next steps	John Keating, Corporate VP, Commercial
15.35	Q&A		
15.55	Break		
16.25	The Next Generation of Therapeutics	Strategic alliances	Jan van de Winkel
16.45	Solid Financial Foundation	Strong foundation for building the business	David Eatwell, Executive VP & CFO
16.50	Closing	2018 Achievements & Beyond	Jan van de Winkel
16.55	Q&A		

# Today's Speakers



# Our Focus

## Core Purpose, Strategy & Vision



### Core Purpose

- To improve the lives of patients by creating & developing innovative antibody products



### Our Strategy


- Turn science into medicine
- Build a profitable & successful biotech
- Focus on Core Competence







### Vision

- By 2025, our own product has transformed cancer treatment and we have a pipeline of knock-your-socks off antibodies

# Innovation Powerhouse



Deep insight into antibody biology

-  Match in-house expertise with strategic partnerships
-  Proprietary technologies allow us to create truly differentiated products
  - For our own and partner pipelines to create revenue streams
  - Target variety of indications
-  Strong pipeline
  - 6 products in clinical dev.
  - Approx. 20 pre-clinical projects → multiple INDs in coming years
-  Continually innovating

# Innovation Powerhouse Scientific Excellence

>200 articles published in scientific journals since 2001<sup>1</sup>

**ARTICLES**

**nature medicine**

## Cooperative targeting of melanoma heterogeneity with an AXL antibody-drug conjugate and BRAF/MEK inhibitors

Julia Boshuizen<sup>1</sup>, Louise A Koopman<sup>2</sup>, Oscar Krijgsman<sup>1</sup>, Aida Shahrabadi<sup>1</sup>, Elke Gresnigt-van den Heuvel<sup>1</sup>, Maarten A Ligtenberg<sup>1</sup>, David W Vredevoogd<sup>1</sup>, Kristel Kemper<sup>1</sup>, Thomas Kuilman<sup>1</sup>, Ji-Ying Song<sup>2</sup>, Nora Pencheva<sup>2</sup>, Jens Thing Mortensen<sup>2</sup>, Marnix Geukes-Foppen<sup>1</sup>, Elisa A Rozeman<sup>1</sup>, Christian U Blank<sup>1</sup>, Maarten I Janmaat<sup>2</sup>, David Satijn<sup>2</sup>, Esther C W Breyer<sup>2</sup>, Daniel S Peepers<sup>1,5</sup> & Paul W H I Parren<sup>2,4,5</sup>

**Intratumor heterogeneity is a key factor contributing to therapeutic failure and, hence, cancer lethality. Heterogeneous tumors show partial therapy responses, and tyrosine kinase AXL. In melanoma, resistance to these inhibitors, rationalizing comprising a human AXL antibody-107-MMAE, as a single agent, of lung, pancreas and cervical cancer, and MAPK pathway inhibitors co-inhibitors potentiated the efficacy of combinatorial targeting of distinct validation of AXL-107-MMAE in**

**Intratumor heterogeneity is a common and challenging treatment, whether chemo-, targeted. Indeed, resistance in heterogeneous tumors is a common and challenging the benefit of single-agent clinical trials. In clinical responses, it has been proposed targeting at least two different pathways. Although our understanding of clones has increased considerably, advances, our knowledge about tumor used to develop rationalized combination. A disease exemplifying the clinical heterogeneity is melanoma. Selective show marked clinical activities and BRAF-mutant melanoma<sup>8-10</sup>. However, patients eventually relapse because of resistance<sup>11-14</sup>. The mechanisms of resistance are highly pleiotropic<sup>11-14</sup>. However, the diversity of these resistance, even in individual patients<sup>15,16</sup>. One of drug-resistant melanomas is the**

**nature medicine** VOLUME 24 NUMBER 2 FEBRUARY 2018

**Predicting response to immunotherapy in melanoma**  
Boosting hematopoietic stem cell recovery  
Vaccine-mediated prevention of tuberculosis

**JBC ARTICLE**  
Author's Choice

## Kinetic mechanism of controlled Fab-arm exchange for the formation of bispecific immunoglobulin G1 antibodies

Received for publication, October 5, 2017. Published in Press, November 17, 2017. DOI 10.1074/jbc.RA117.000303

Dennis R. Goulet<sup>1</sup>, Steven J. Orcutt<sup>1</sup>, Adam Zwolak<sup>1</sup>, Theo Rispens<sup>1</sup>, Aran F. Labrijn<sup>1</sup>, Rob N. de Jong<sup>1</sup>, William M. Atkins<sup>1</sup>, and Mark L. Chiu<sup>1,2</sup>

From the <sup>1</sup>Department of Medical Chemistry, University of Washington, Seattle, Washington 98195, <sup>2</sup>Biologics Discovery, Janssen Research & Development, LLC, Spring House, Pennsylvania 19477, the <sup>3</sup>Sanguin Research and Landsteiner Laboratory, Department of Immunopathology, Academic Medical Centre, University of Amsterdam, Meibergdreef 125, 1066 CX Amsterdam, The Netherlands, and <sup>4</sup>Genmab, Yalelaan 60, 3584 CM Utrecht, The Netherlands

Edited by Peter Cresswell

Bispecific antibodies (bsAbs) combine the antigen specificities of two distinct Abs and demonstrate therapeutic promise based on novel mechanisms of action. Among the many platforms for creating bsAbs, controlled Fab-arm exchange (cFAE) has proven useful based on minimal curation and the simplicity with which it can be implemented. Despite its widespread use in the pharmaceutical industry, the mechanism of cFAE has not been determined. Fluorescence correlation spectroscopy (FCS) and Förster resonance energy transfer (FRET) were employed to identify and characterize cFAE. Fluorescence correlation spectroscopy (FCS) and Förster resonance energy transfer (FRET) determine the affinity of parental (the heterodimer) interactions within the cFAE complex. FCS revealed a clear sequence of events with respect to experimental conditions, where the parental Ab into half-Ab controls the targeting two tumor antigens allows for inhibition of orthogonal signaling pathways and can increase specificity for cancer cells versus healthy tissue while also mitigating the incidence of cytotoxic side effects (Fig. 2). The observed kinetic mechanism of cFAE is consistent with a two-step process involving the formation of a bispecific intermediate (Fig. 3). The bispecific intermediate is formed by the association of a half-Ab with a full-Ab, followed by the dissociation of the full-Ab into two half-Abs, which then recombine to form the bispecific intermediate. This mechanism is consistent with the observed kinetic data and provides a rational basis for the design of bsAbs.

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This work was supported by National Institutes of Health (NIH) Grant GM007750 (to D.R.G.) and the University of Washington Medical Center. The authors declare no conflict of interest with the contents of this article. The availability of the authors and does not necessarily represent the views of the National Institutes of Health.

Author's Choice—Final version free via JBC

This article contains Figs. S1–S14, Schemes S1 and S2, and Methods.

<sup>1</sup> To whom correspondence should be addressed: Mark L. Chiu, Department of Medical Chemistry, University of Washington, Box 357350, Seattle, WA 98195. E-mail: chiu@u.washington.edu

<sup>2</sup> The abbreviations used are: bsAb, bispecific antibody; cFAE, controlled Fab-arm exchange; FCS, fluorescence correlation spectroscopy; pp120, phosphotyrosine; PBS, phosphate-buffered saline; FRET, Förster resonance energy transfer.

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**RESEARCH**

## Structures of C1-IgG1 provide insights into how danger pattern recognition activates complement

Deniz Ugarlar<sup>1</sup>, Stuart C. Howes<sup>2</sup>, Bart-Jan de Kruuk<sup>3</sup>, Roman I. Koning<sup>2,4</sup>, Rob N. de Jong<sup>5</sup>, Frank J. Beurskens<sup>6</sup>, Janine Schuurman<sup>7</sup>, Abraham J. Koster<sup>2,4</sup>, Thomas H. Sharp<sup>2,7</sup>, Paul W. H. I. Parren<sup>1,2,5</sup>, Piet Groen<sup>2,7</sup>

**Danger patterns on microbes or damaged host cells bind and activate C1, inducing innate immune responses and clearance through the complement cascade. How these patterns trigger complement activation remains elusive. Here, we present cryo-electron microscopy analyses of C1 bound to monoclonal antibodies in which we observed heteromeric structures of single and clustered C1-immunoglobulin G1 (IgG1) hexamer complexes. Distinct C1q binding sites are observed on the two Fc-CH2 domains of each IgG molecule. These are consistent with known interactions and also reveal additional interactions, which are supported by functional IgG1 mutant analysis.**

poorly understood (17). We used IgG1 monoclonal antibodies (mAbs) oligomerized through antigen-binding on liposomes or preformed antibody complexes in solution and applied tomography and single-particle cryo-electron microscopy (cryo-EM) to resolve the mechanisms of C1 binding and activation. Liposomes carrying di-2-nitrophenyl (DNP) haptens were incubated with an anti-DNP chimeric IgG1 mAb and C1 to allow extensive formation of surface-bound C1-IgG1 complexes (Fig. 1A). Tomograms showed marked structural variations in C1 binding to antibodies on these liposomes (Fig. 1A and Fig. S2, A and B). Alignment and classification of single-membrane-bound C1-IgG1 complexes (Fig. 1B) yielded a reconstruction at 25-Å resolution (Fig. S2, C and D). Focused alignment and classification on the Fc-C1 complex (excluding the membrane and Fab domains) (Fig. S2, B and E) revealed six densities corresponding to C1q domains binding an Fc-platform formed by six IgG1 molecules, a hexameric platform accounting for bound C1q, a hexameric and a trimeric C1q

**RESEARCH**

**STRUCTURAL IMMUNOLOGY**

**Structures of C1-IgG1 provide insights into how danger pattern recognition activates complement**

Deniz Ugarlar<sup>1</sup>, Stuart C. Howes<sup>2</sup>, Bart-Jan de Kruuk<sup>3</sup>, Roman I. Koning<sup>2,4</sup>, Rob N. de Jong<sup>5</sup>, Frank J. Beurskens<sup>6</sup>, Janine Schuurman<sup>7</sup>, Abraham J. Koster<sup>2,4</sup>, Thomas H. Sharp<sup>2,7</sup>, Paul W. H. I. Parren<sup>1,2,5</sup>, Piet Groen<sup>2,7</sup>

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**Science** 15 FEBRUARY 2018

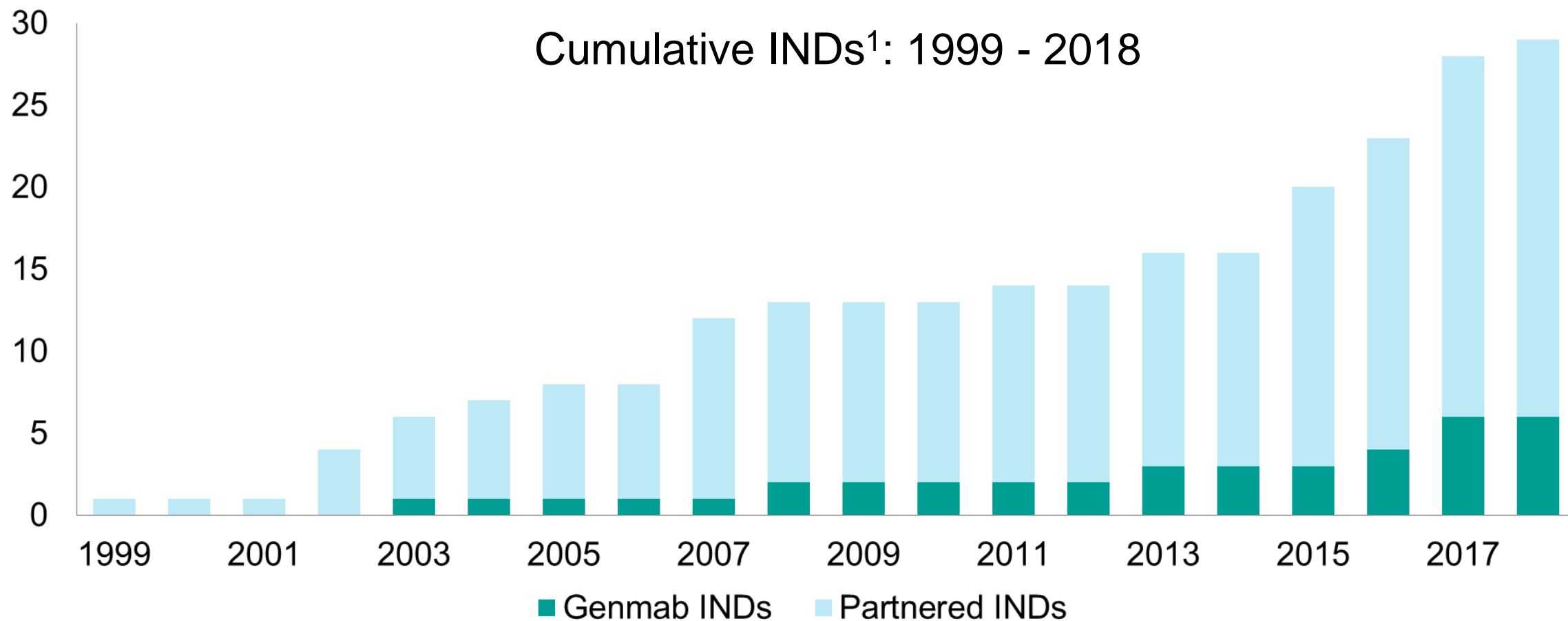
**GORDON RESEARCH CONFERENCES**  
Topics include venom evolution, green chemistry, meiosis, and more p. A58

Ugarlar et al., *Science* 359, 794–797 (2018)

<sup>1</sup>Based on PubMed and internal Genmab lists

# Innovation Powerhouse

## Strong IND Track Record



<sup>1</sup>Includes INDs filed by Genmab and partners; 6 of 27 INDs through December 2017 are ≥50% owned by Genmab

# Innovation in Action

## Two Marketed Products: Latest News

### DARZALEX<sup>®</sup> (daratumumab)



- Approved globally in various MM indications\*
- Submission of reg. apps. for split dosing in US & EU
- Submission of reg. application in China (pending acceptance)
- Blockbuster status: >USD 1 bn WW sales in 2017
- On track for 2018 sales guidance of USD 2.0 – 2.3 bn
- Ph III MAIA & CASSIOPEIA data anticipated in 2018
- Other key data: COLUMBA (Ph III SC D), GRIFFIN (Ph II D-RVd) and CANDOR (Ph III D+Kd) anticipated, 2019
- >80 clinical studies ongoing

### Arzerra<sup>®</sup> (ofatumumab)



- Marketed in various territories for certain CLL indications\*: in non-US markets, Novartis will transition from commercial to compassionate use

### ofatumumab

- Recruitment completed in Ph III SC RMS studies
- Ph III RMS data anticipated, 2019
- Potential regulatory filing soon after data, based on study completion & positive results
- Double digit royalty to Genmab, no costs

\*See local country prescribing information for precise indications

# Genmab R&D Center

## Connect, Inspire, Create



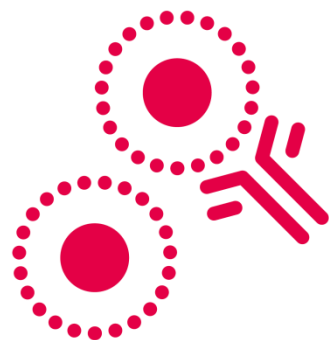
# Excellence in Innovation: Novel Proprietary Technology Platform

Janine Schuurman, Corporate Vice President, Research & Innovation



# Harnessing the Potency of the Immune System: Basic Immunological Principles to Technologies and Products

The power of our immune system inspires us



We are curious to understand basic immunological principles



We translate this to practical applications

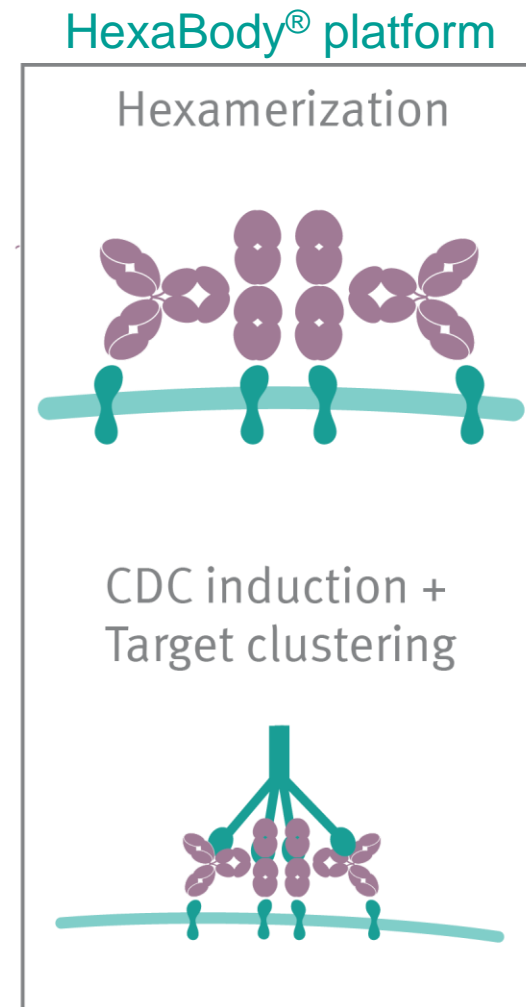
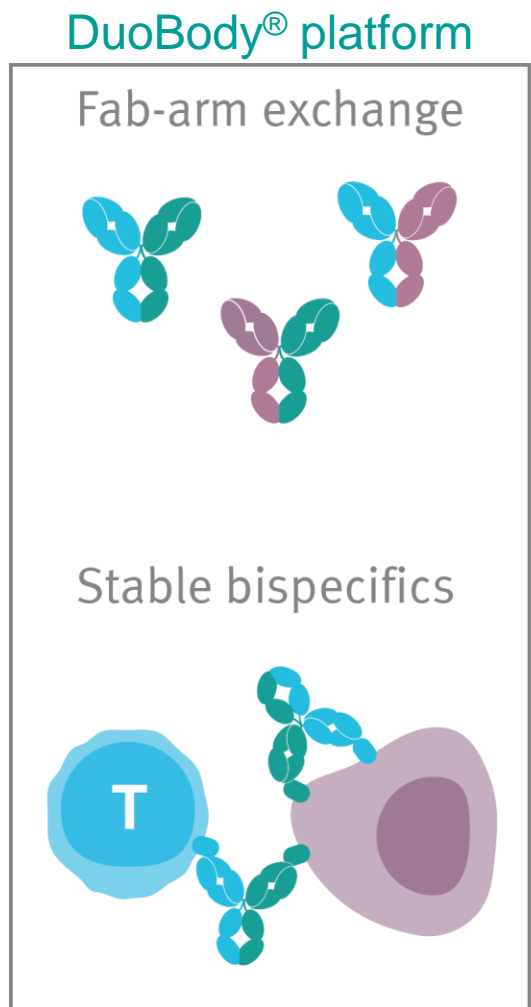


Innovative technologies and differentiated antibody products

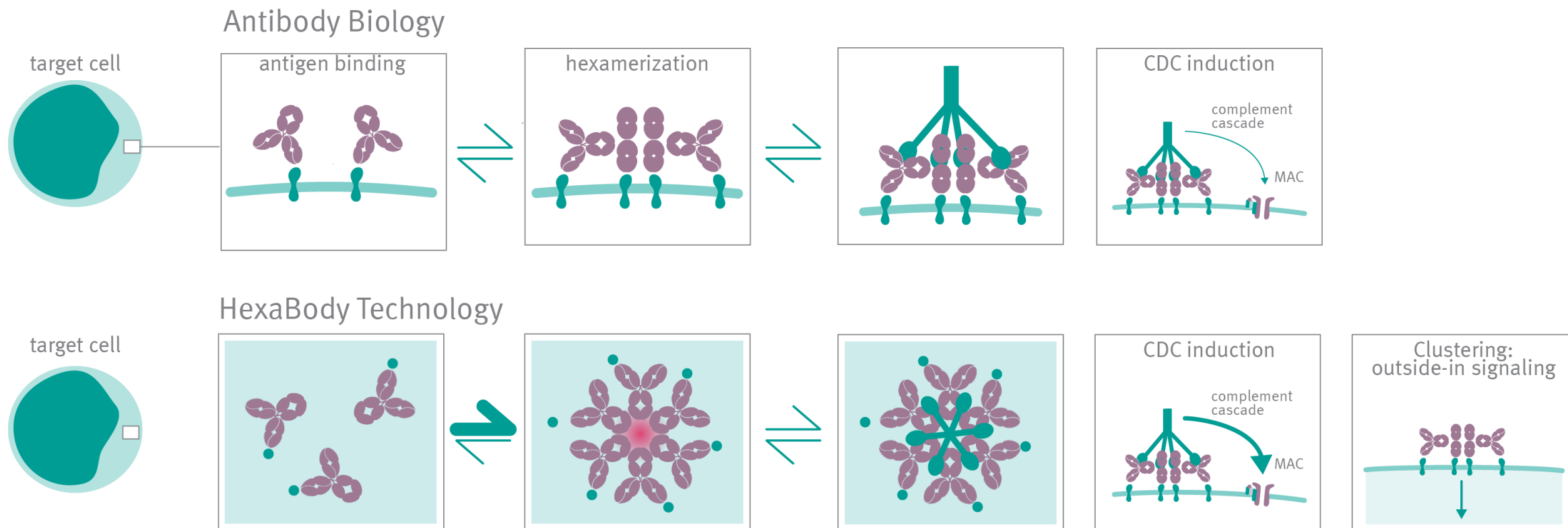


# Genmab Antibody Technology Innovation

## Creating Differentiated Antibody Products

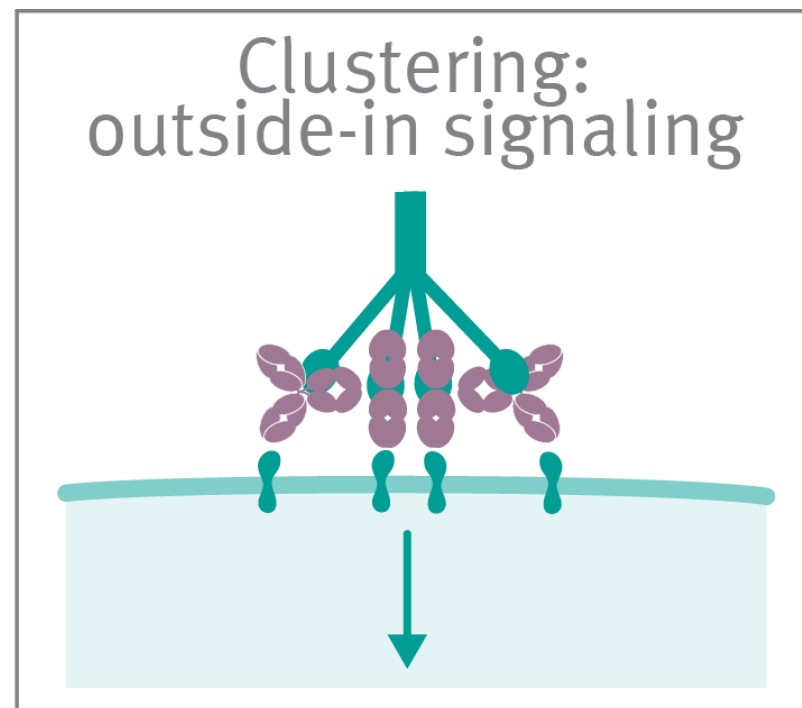
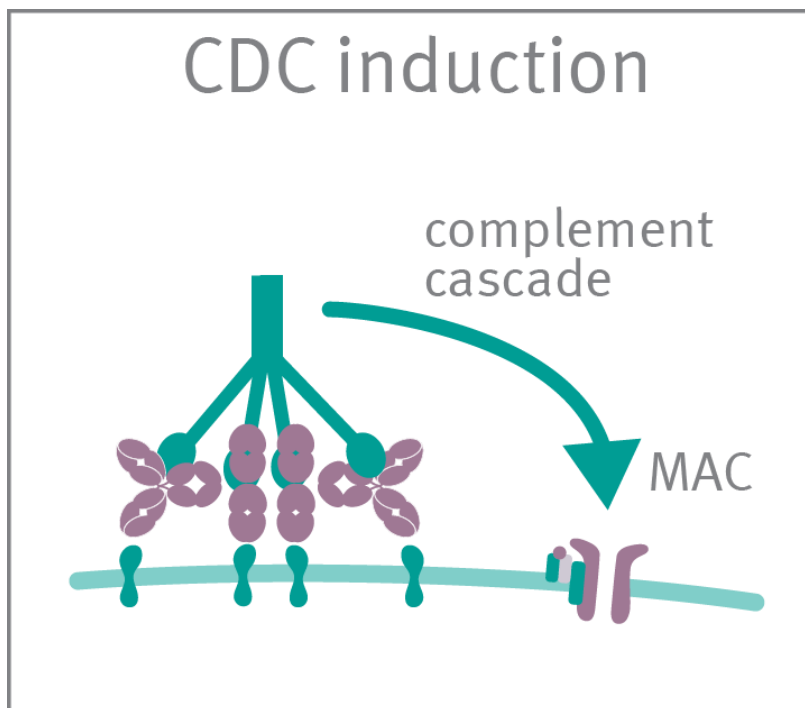


# Biology Of IgG Hexamerization: From Science To HexaBody<sup>®</sup> Platform



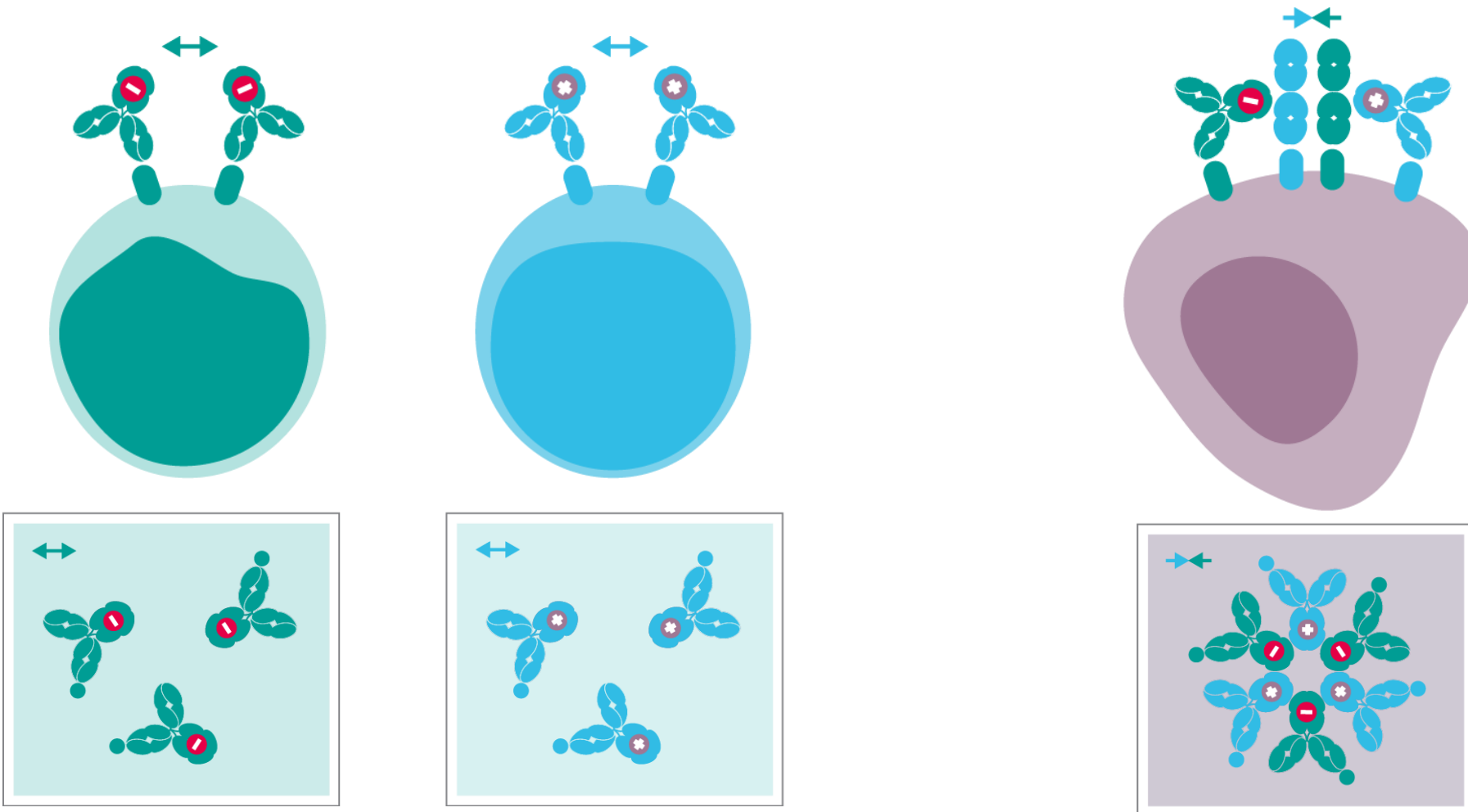
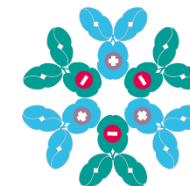
# HexaBody Platform

The concept of target-dependent hexamerization on the cell surface can be utilized to develop potentiated antibody therapeutics



# HexElect™ Platform:

Two Co-dependently Acting HexaBody Molecules: Introducing Selectivity, Maximizing Therapeutic Potency




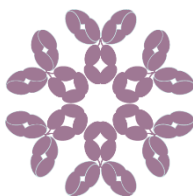
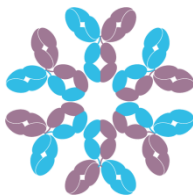
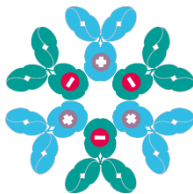
# HexElect Platform

## Proof of Principle: Selective Depletion of Cellular Subset



Two co-dependently acting HexaBody<sup>®</sup> molecules: to maximize therapeutic potency while minimizing potential toxicity

# Platform Technology Suite Boosting Our Product Pipeline

		Principle	Applications
<b>DuoBody</b>		Bispecific antibodies	Dual targeting: <ul style="list-style-type: none"> <li>- Recruitment (e.g. T cells)</li> <li>- Tumor heterogeneity</li> </ul>
<b>HexaBody</b>		Target-mediated enhanced hexamerization	Enhanced potency: <ul style="list-style-type: none"> <li>- CDC</li> <li>- Target clustering, outside-in signaling, apoptosis</li> </ul>
<b>DuoHexaBody™</b>		Bispecific antibodies with target-mediated enhanced hexamerization	Dual targeting + enhanced potency <ul style="list-style-type: none"> <li>- CDC</li> <li>- Target clustering, outside-in signaling, apoptosis</li> </ul>
<b>HexElect</b>		Two co-dependent antibodies with target-mediated enhanced hexamerization	Dual targeting + enhanced potency & selectivity: <ul style="list-style-type: none"> <li>- Co-dependent unlocking of potency</li> <li>- New target space, previously inaccessible</li> </ul>

# Future Leapfrog Products: Pre-clinical Candidate DuoHexaBody-CD37

Esther Breij, Director, Translational Research

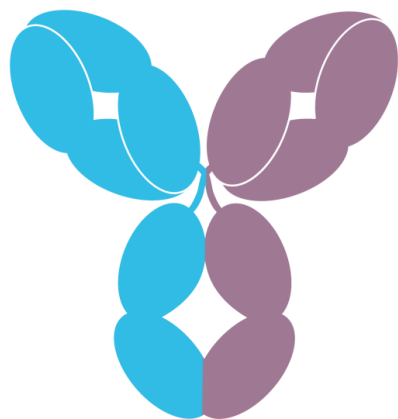


# DuoHexaBody-CD37

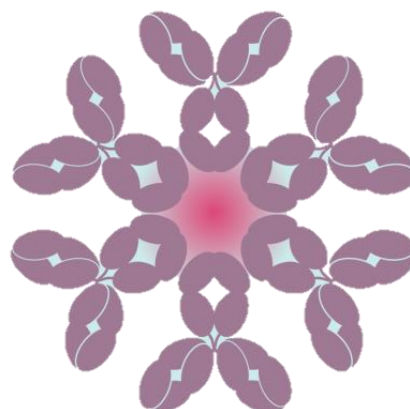
## Based on Two of Genmab's Proprietary Antibody Platforms

- Bispecific IgG1 with an E430G hexamerization-enhancing mutation in IgG Fc domain
- DuoHexaBody targets two non-overlapping epitopes on CD37

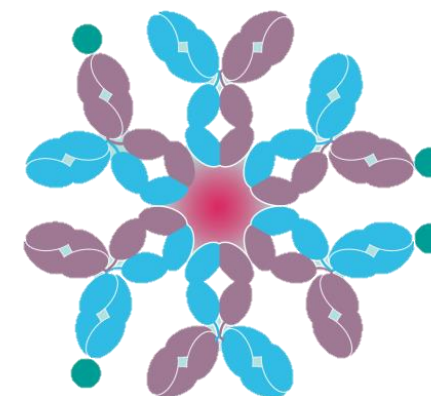
DuoBody



HexaBody



DuoHexaBody



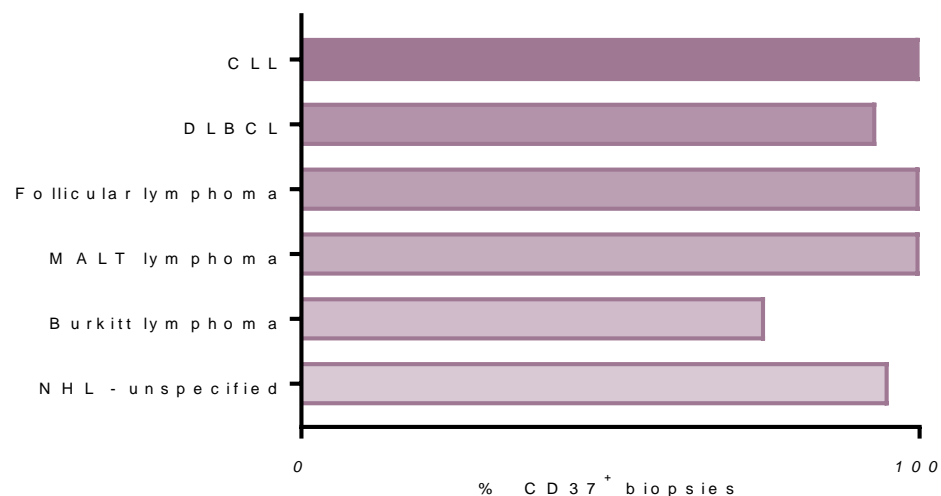
# DuoHexaBody-CD37

## CD37 is Novel Target for Hematological B cell Malignancies

### CD37

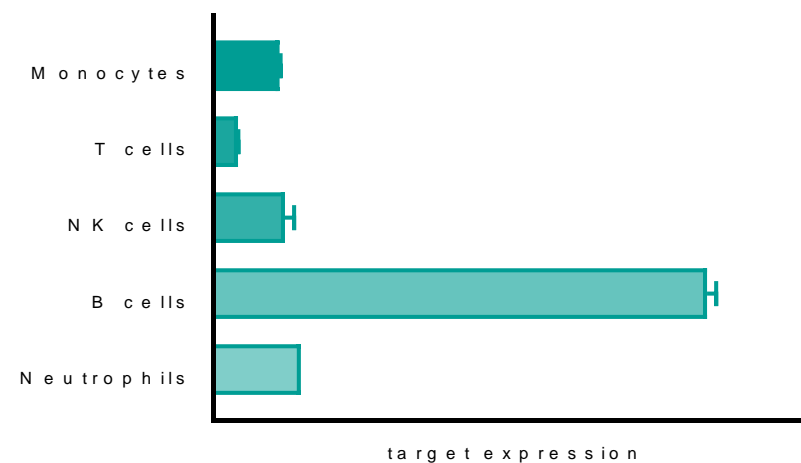
- abundantly expressed on the plasma membrane in B cell malignancies ranging from CLL to DLBCL
- membrane protein from the tetraspanin family
- normal tissue expression mostly restricted to B cells, low expression on other leukocyte subsets
- not expressed outside hematopoietic lineage

CD37 expression in B cell malignancies



adapted from Deckert et al, Blood 2013

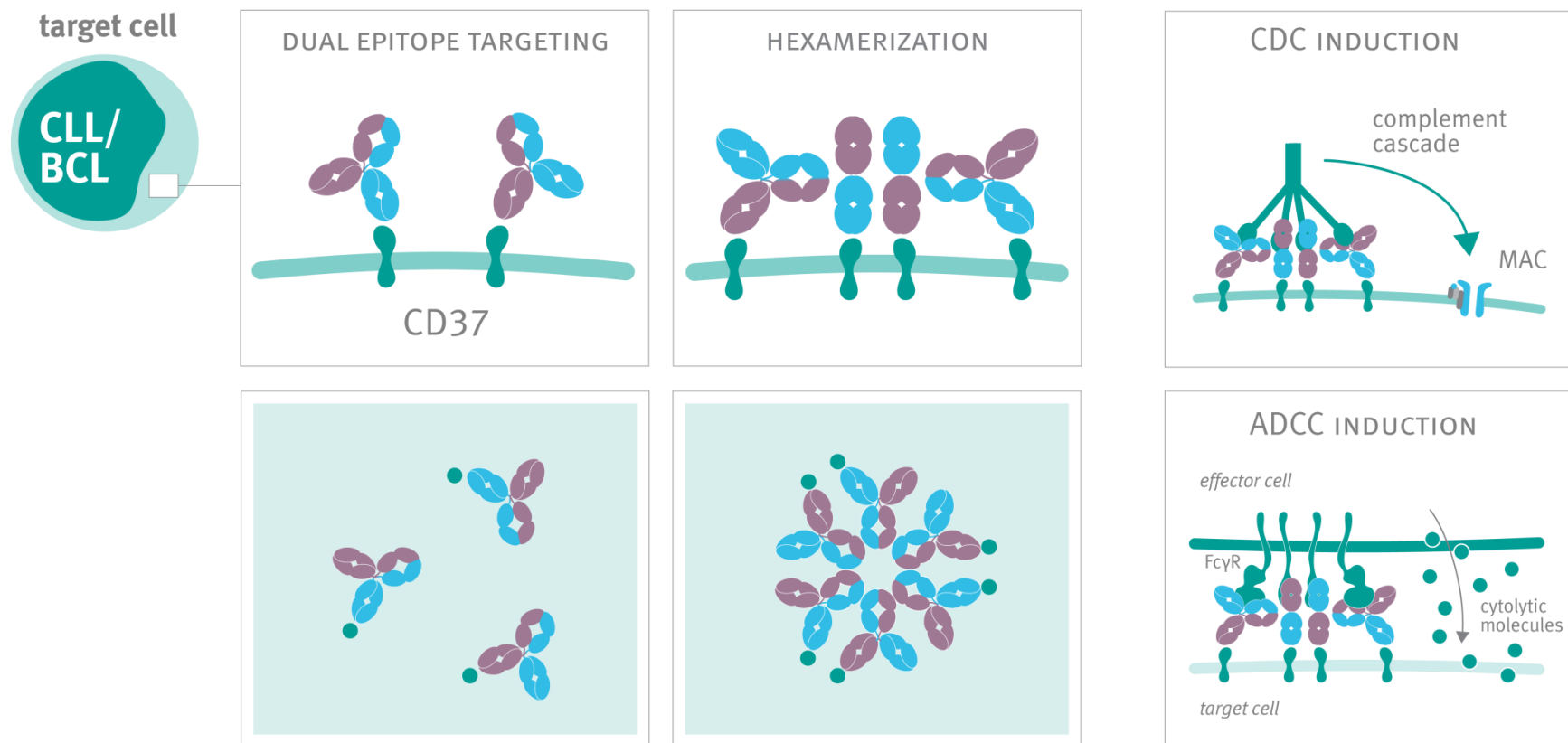
CD37 expression on normal leukocyte subsets



# DuoHexaBody-CD37

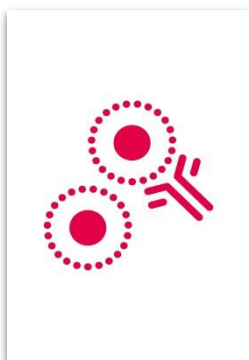
## Unique Mechanism of Action

- In pre-clinical settings DuoHexaBody-CD37 induces potent anti-tumor activity through superior complement-dependent cytotoxicity (CDC) and potent antibody-dependent cell-mediated cytotoxicity (ADCC)



# DuoHexaBody-CD37

## Promising Pre-clinical Data - CLL Patient Samples



Potent anti-tumor activity in untreated and refractory CLL  
[ex vivo]

# DuoHexaBody-CD37

## Promising Pre-Clinical Data – Therapeutic Activity *in vivo*

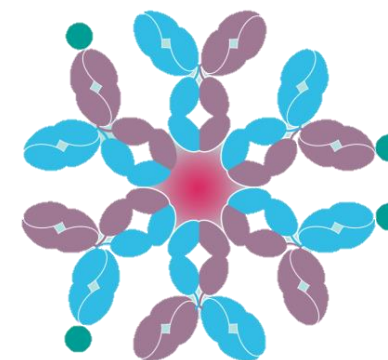



Therapeutic activity in non-Hodgkin lymphoma (NHL)  
xenograft model [mice]

# DuoHexaBody-CD37

## Overview

- DuoHexaBody molecule, based on Genmab's DuoBody & HexaBody platforms
- Targets two non-overlapping epitopes on CD37, a membrane protein widely expressed in B cell malignancies
- Pre-clinical Setting: superior cytotoxic activity through superior complement-dependent cytotoxicity (CDC) and efficient antibody-dependent cell-mediated cytotoxicity (ADCC)
- Highly potent killing of tumor cells from CLL patients *ex vivo*, including cells from treatment resistant patients
- Potent anti-tumor activity *in vivo* xenograft mouse model
- IND/CTA filing, H2 2019



A blue-tinted microscopic image of a DNA microarray. A pipette tip is positioned over one of the spots on the array. The spots are arranged in a grid and show various colors, likely representing different DNA sequences or probes.

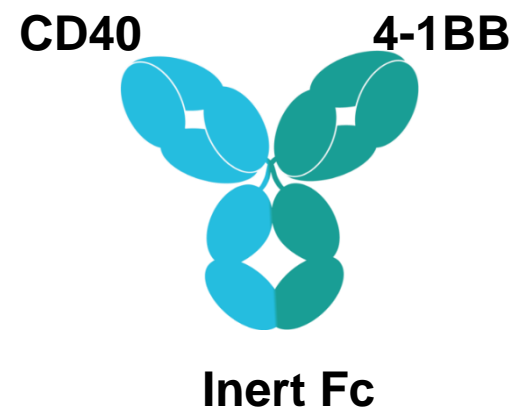
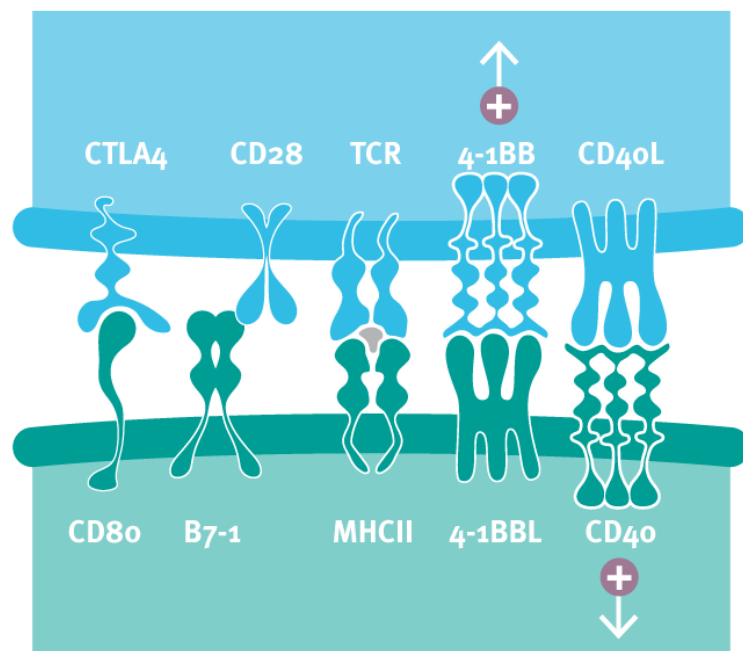
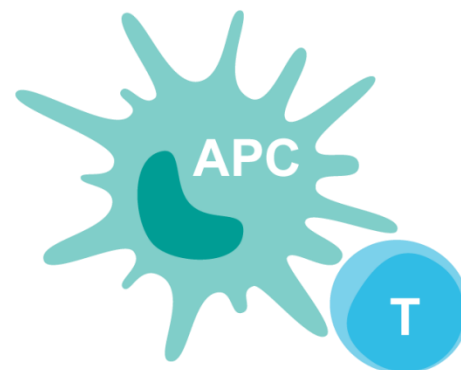
# **Future Leapfrog Products: Pre-clinical Candidates DuoBody-CD40x4-1BB & DuoBody-PD-L1x4-1BB**

David Satijn, Vice President, New Antibody Products



# DuoBody-CD40x4-1BB

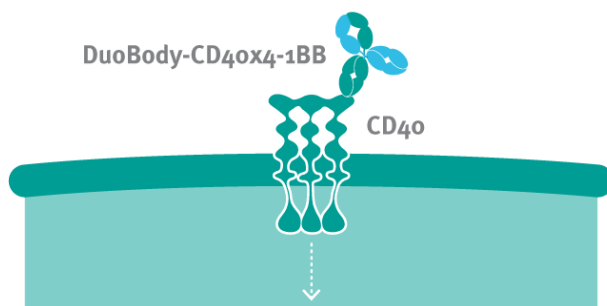
## Concept



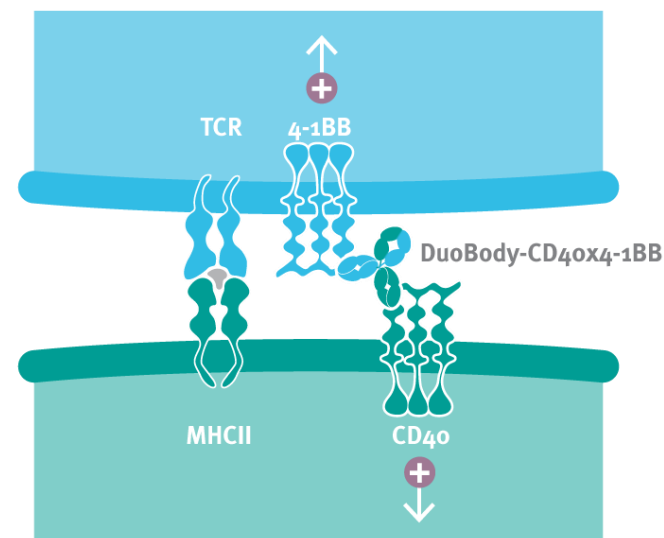
CD40 and 4-1BB are selected as targets to enhance both DC- and antigen-dependent T cell activation, using an inert DuoBody format.

# DuoBody-CD40x4-1BB Induces Conditional T-cell Proliferation

Without trans-presenting:



With trans-presenting:

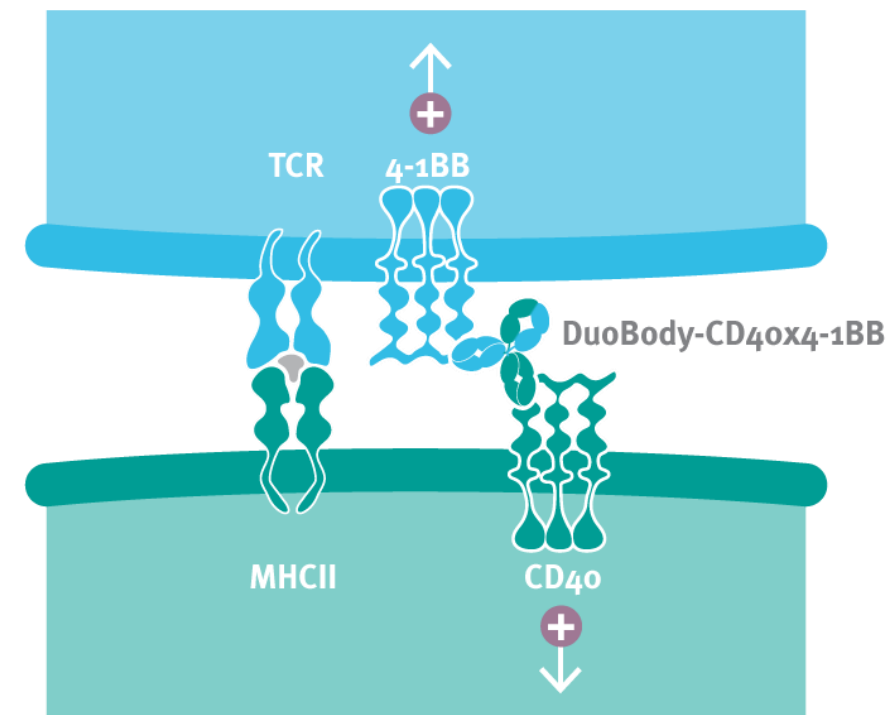


- Conditional, simultaneous targeting of CD40 and 4-1BB provides essential trans-activity for potent immune responses.
- DuoBody-CD40x4-1BB firmly enhances T-cell proliferation, whereas combination of IgG1-CD40 and IgG1-4-1BB is ineffective.

# DuoBody-CD40x4-1BB

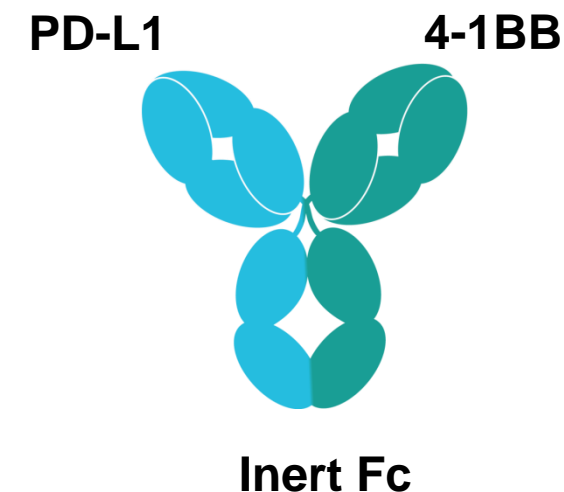
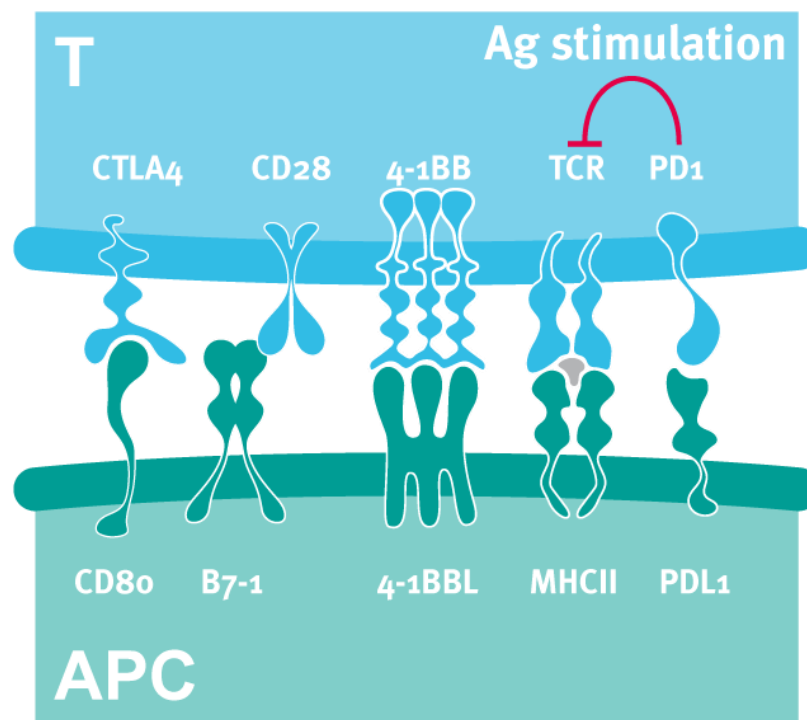
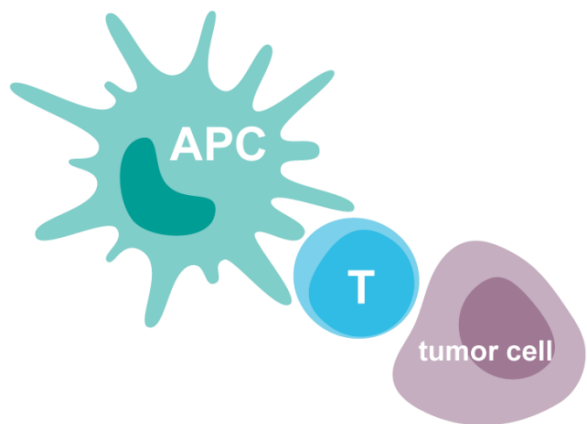
## Characteristics

- Bispecific antibody targeting CD40 and 4-1BB (CD137) using an inert Fc backbone.
- For treatment of solid cancers
- In pre-clinical settings simultaneously activates APC & enhances T cell activation
  - Enhances activation of APC's
  - Conditional activation and expansion of previously activated cytotoxic CD8<sup>+</sup> T cells
  - Clonal expansion of T cells
  - Cytokine production
- IND/CTA, H1 2019



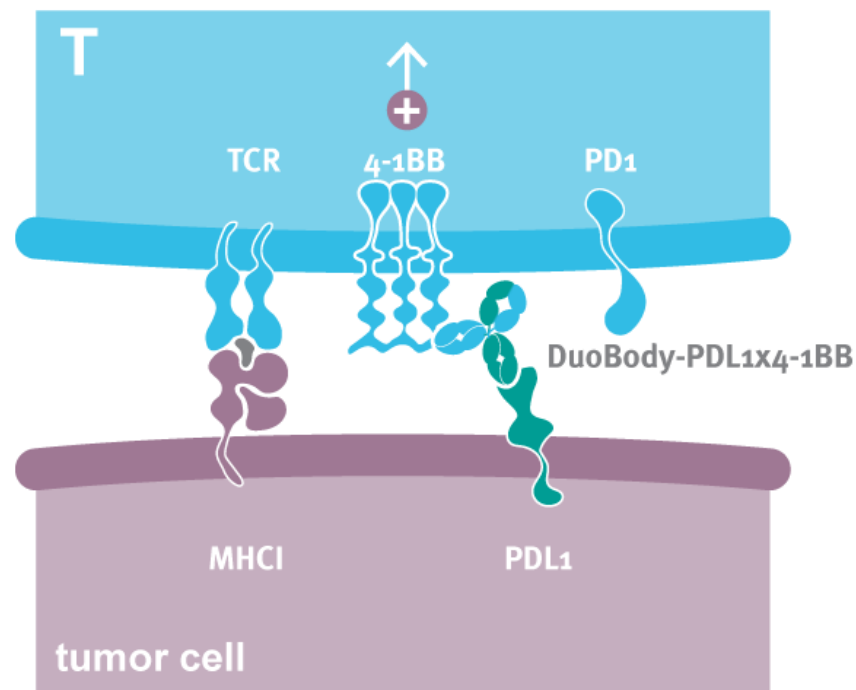
# DuoBody-PD-L1x4-1BB

## Concept



- PD-L1 and 4-1BB are selected as targets to block the inhibitory PD-1/PD-L1 axis and simultaneously activate essential co-stimulatory activity via 4-1BB using inert DuoBody antibody format.

# DuoBody-PD-L1x4-1BB Induces Conditional T-cell Proliferation

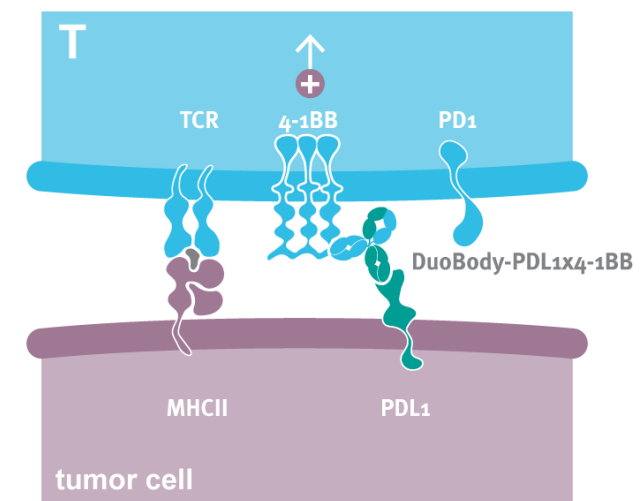
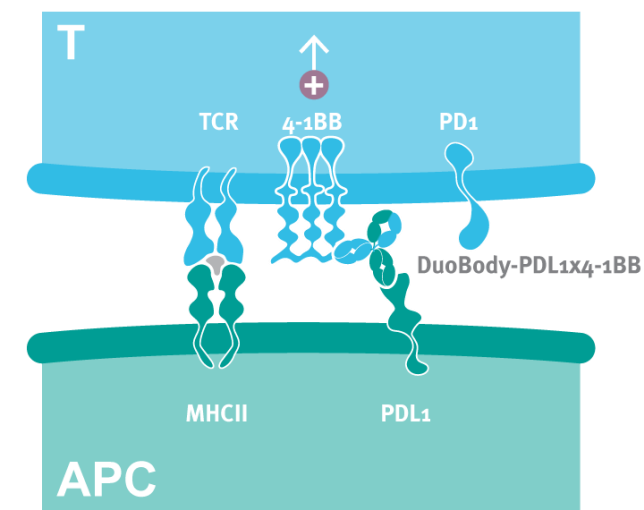


- Conditional transactivation of DuoBody-PD-L1x4-1BB leads to enhanced/synergistic proliferation of T-cells
- No enhanced or synergistic effects of combination of PDL1 and 4-1BB is observed

# DuoBody-PD-L1x4-1BB

## Characteristics

- Bispecific antibody targeting PD-L1 and 4-1BB using an inert Fc backbone.
- Treatment of solid cancers
- In pre-clinical settings promotes conditional T cell activation in a tumor-specific manner by simultaneous activation and release of key inhibitory brake.
  - Release of T cell inhibition through PD-1/ PD-L1 axis, also in the absence of 4-1BB.
  - Strong co-stimulation via the agonistic activity of 4-1BB.
  - T cell clonal expansion
- IND/CTA, H1 2019



# Q&A



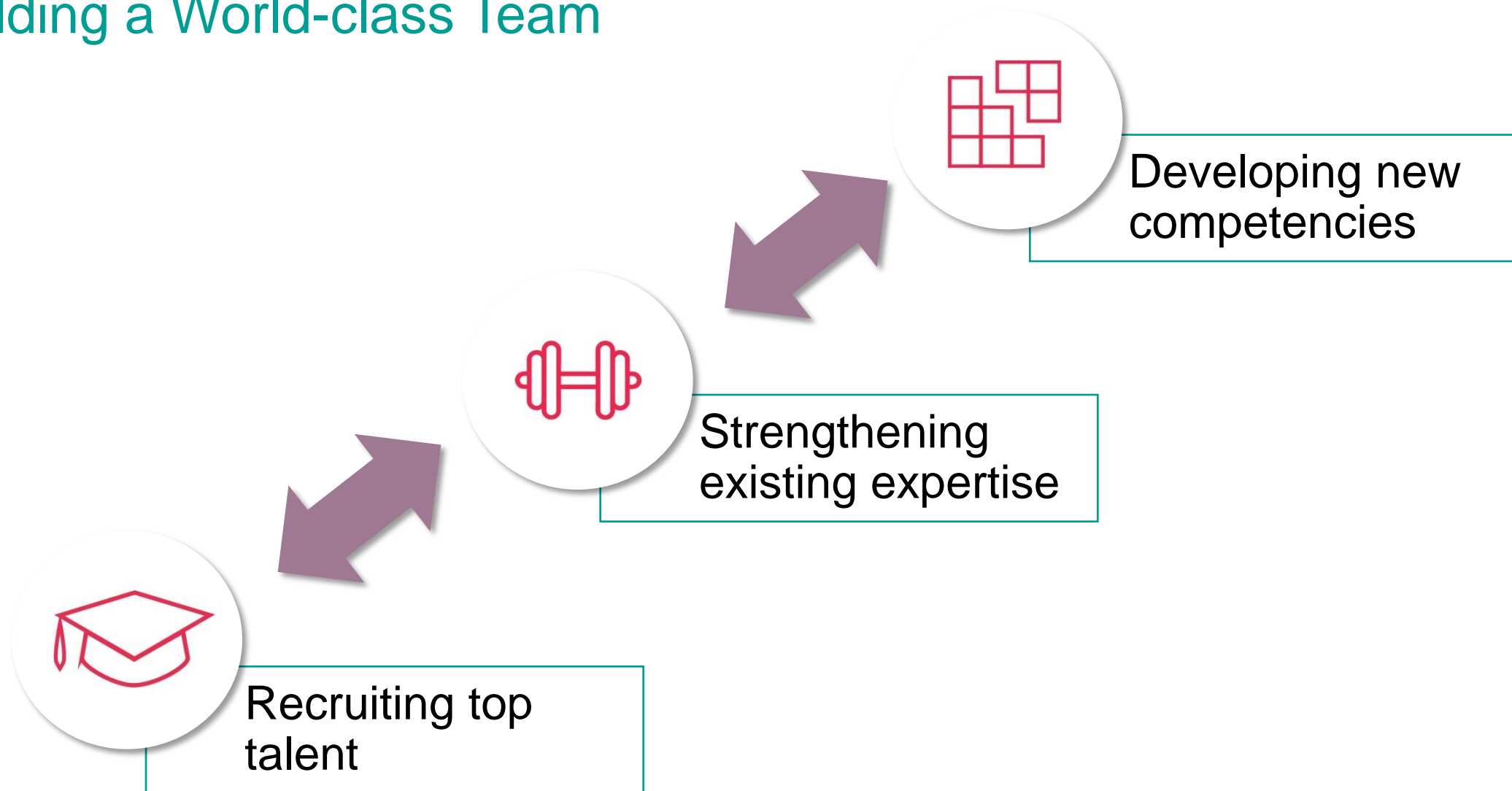
# Reaching Our Inspirational Vision: Late Stage Clinical Pipeline

Judith Klimovsky, Executive Vice President & Chief Development Officer



# Clinical Development

## Building a World-class Team



# Reaching Our Vision: Tisotumab Vedotin

## Background

Fully human antibody-drug conjugate (ADC)

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Targets Tissue Factor (TF)

Therapeutic potential in broad range of solid tumors

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Ongoing Studies

Ph I/II solid tumors: ovary, cervix, endometrium, bladder, prostate, esophagus, NSCLC, SCCHN

Ph II second line cervical cancer: potentially registrational

Ph II solid tumors: colorectal, NSCLC, pancreatic, SCCHN

Ph II ovarian cancer (planned)

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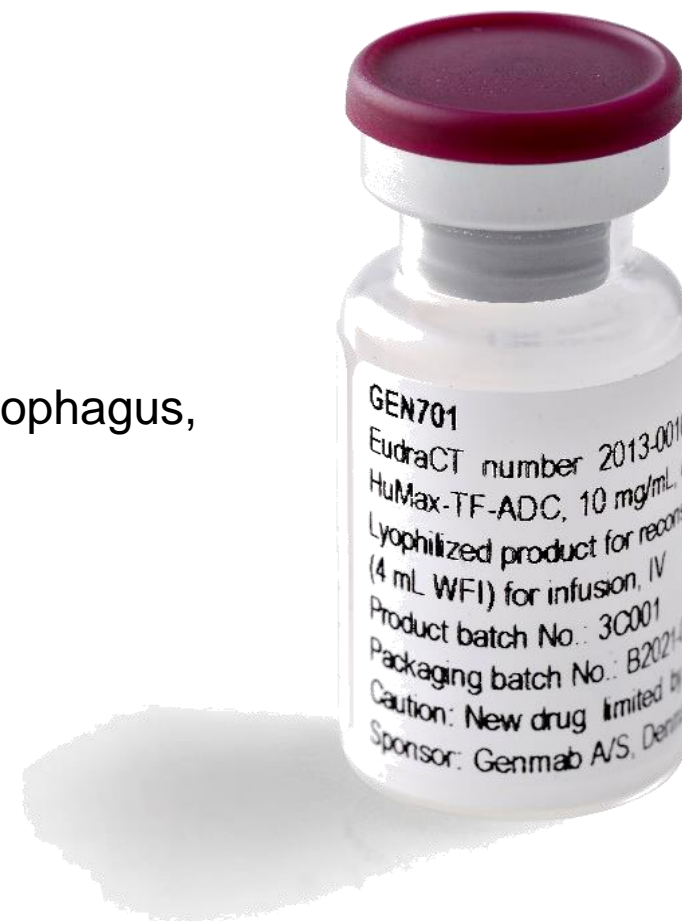
Planned Studies

Ph II first/second line combo. in cervical cancer

Ph I/II safety in Japan

---

50:50 Co-development with Seattle Genetics



# Reaching Our Vision: Tisotumab Vedotin

## Science Behind the Product

**Tisotumab vedotin** is antibody-drug conjugate targeting tissue factor with therapeutic potential in broad range of solid tumors

1. Binding to TF<sup>a</sup>

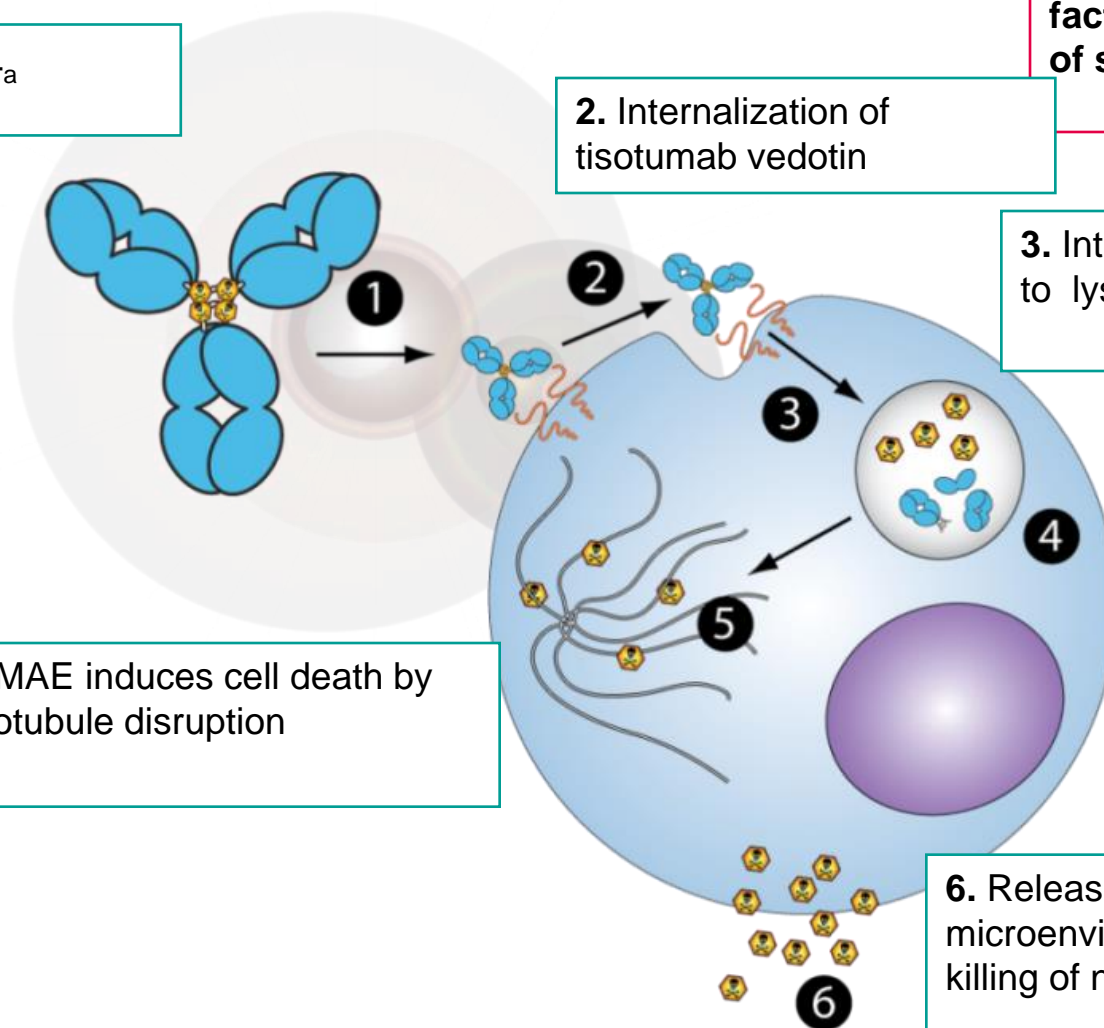
2. Internalization of tisotumab vedotin

3. Intracellular trafficking to lysosomes

4. Enzymatic degradation of tisotumab vedotin, intracellular release of MMAE

5. MMAE induces cell death by microtubule disruption

6. Release of MMAE in tumor microenvironment induces bystander killing of neighboring cancer cells



<sup>a</sup> Tissue factor is also known as TF, CD142, and thromboplastin

# Reaching Our Vision: Tisotumab Vedotin

## ESMO 2017: innovaTV 201, Cervical Cancer Cohort

### Baseline Patient Characteristics in Cervical Cancer Cohort

	Cervical (N=34)
Age (median, range), y	43 (21-73)
ECOG score, no (%)	
0	7 (21%)
1	26 (76%)
Missing	1 (3%)
Cancer type, no (%)	
Adenocarcinoma	15 (44%)
Adeno-squamous	3 (9%)
Squamous	15 (44%)
Missing/TBD	1 (3%)
Previous lines of systemic treatments, no (%)	
0 <sup>a</sup>	3 (9%)
1	13 (38%)
2	11 (32%)
3	4 (12%)
4	3 (9%)

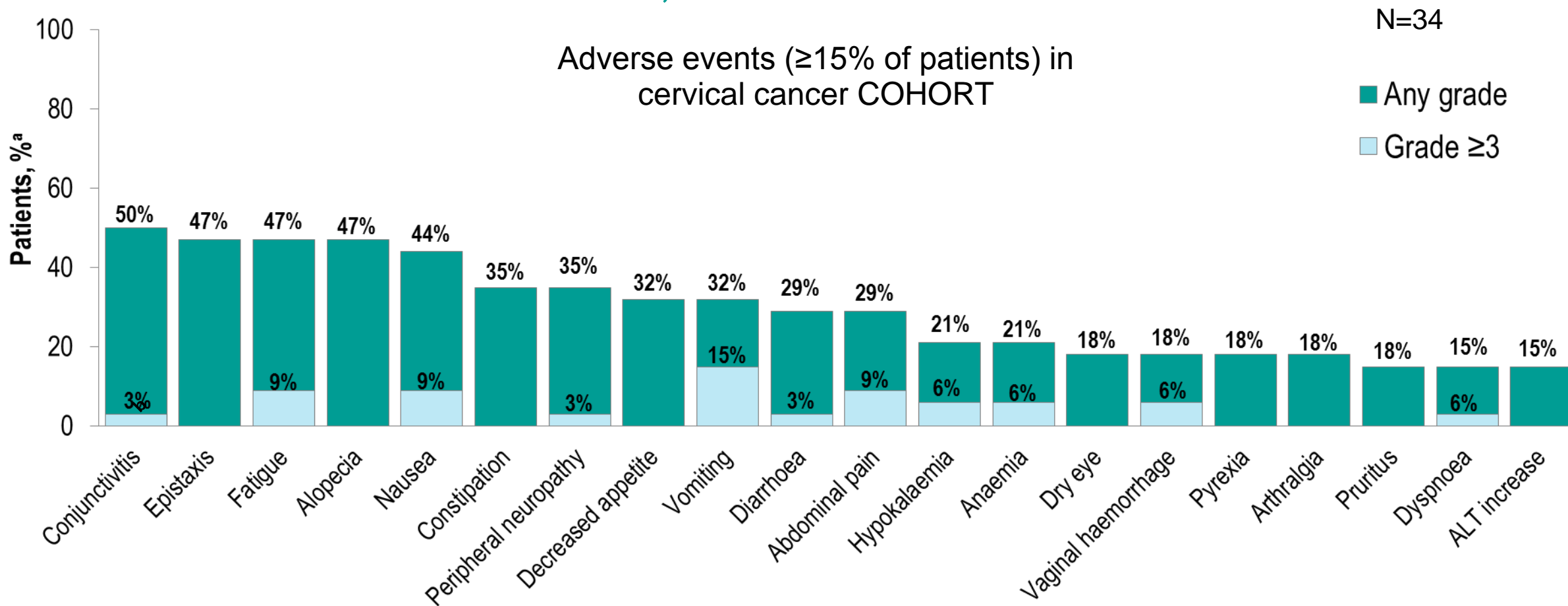
	Cervical (N=34)
Prior treatments, % <sup>b</sup>	
Platinum	91%
Taxane	91%
Bevacizumab <sup>c</sup>	71%
GOG 240 regimen <sup>d</sup>	68%
≥1 platinum doublet	17%
Prior radiotherapy <sup>e</sup>	74%

ECOG=Eastern Cooperative Oncology Group; TBD=to be determined.

<sup>a</sup>Patients progressed on therapy administered for treatment of locally advanced disease. <sup>b</sup>Missing data from 1 patient. <sup>c</sup>Including bevacizumab administered as combination therapy as either platinum/bevacizumab/paclitaxel or topotecan/bevacizumab/paclitaxel. <sup>d</sup>Combination therapy with cisplatin, paclitaxel, and bevacizumab. <sup>e</sup>External beam radiotherapy administered to the cervix or surrounding tissues. Data cutoff date July 24, 2017.

# Reaching Our Vision: Tisotumab Vedotin

## ESMO 2017: innovaTV 201, Cervical Cancer Cohort



ALT=alanine aminotransferase.

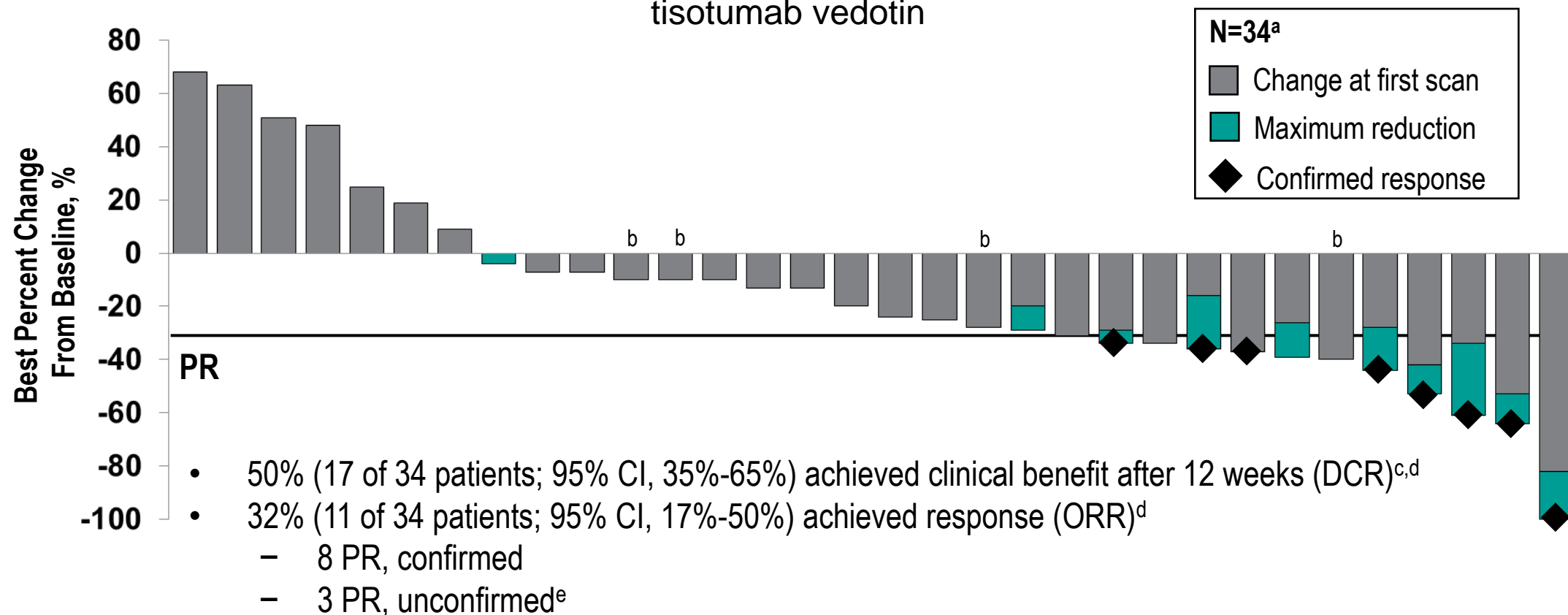
<sup>a</sup>Adverse events with events of any grade occurring in  $\geq 15\%$  of patients or of grade  $\geq 3$  in 2 or more patients. <sup>b</sup>Grade 2 conjunctivitis was reported in 32% of patients.

Data cutoff date July 24, 2017.

# Reaching Our Vision: Tisotumab Vedotin

## ESMO 2017: innovaTV 201, Cervical Cancer Cohort

32% of patients with recurrent/advanced cervical cancer achieved response with tisotumab vedotin

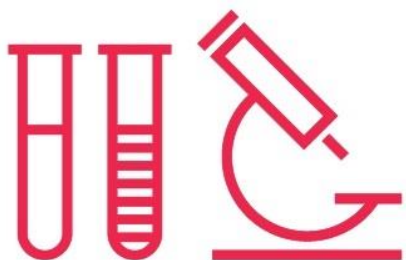


CI=confidence interval; CR=complete response; CT=computed tomography; DCR=disease control rate; ORR=overall response rate; PD=progressive disease; PR=partial response; RECIST=Response Evaluation Criteria in Solid Tumors; SD=stable disease.

<sup>a</sup>Two patients were withdrawn prior to CT scan, and so are not represented in the graph. <sup>b</sup>PD due to new lesion at same scan. <sup>c</sup>Clinical benefit was defined as the DCR rate, the proportion of patients who achieved a CR, PR, or SD after 12 weeks. <sup>d</sup>Response was as assessed by investigators using standard RECIST 1.1 criteria. <sup>e</sup>One of which is still ongoing. Data cutoff date July 24, 2017.

# Reaching Our Vision: Tisotumab Vedotin

## innovaTV 201, Expansion Cohorts

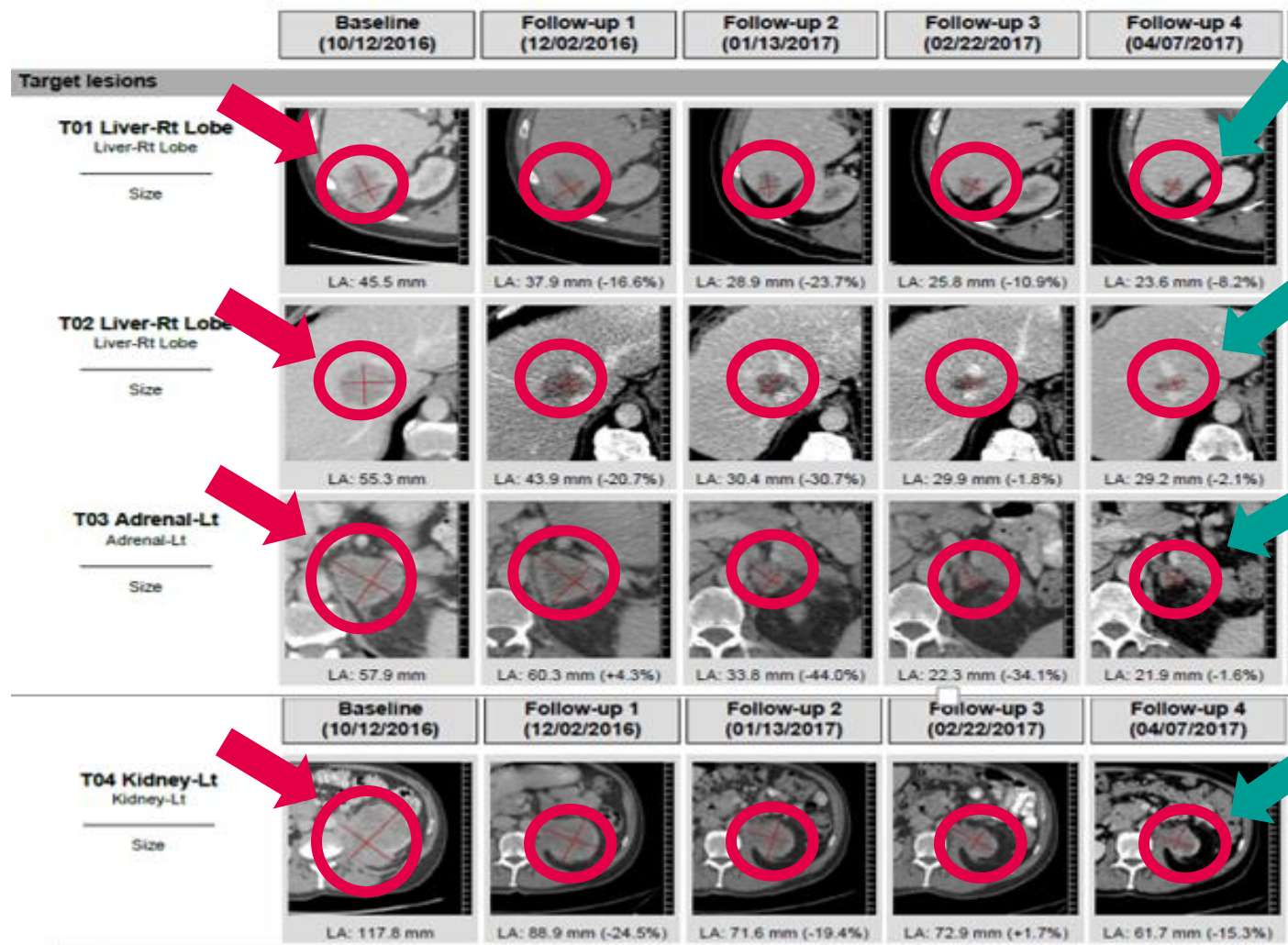


Active across several tumor types in Ph I/II expansion

- Bladder
- Cervical
- Endometrial
- Esophageal
- NSCLC
- Ovarian
- Prostate

# Reaching Our Vision: Tisotumab Vedotin

## innovaTV 201, Expansion Cohorts



### Prior treatments:

1. Cisplatin + Gemcitabine;
2. Vinflunine+ Pemetrexed;
3. Pembrolizumab

# Reaching Our Vision: Tisotumab Vedotin innovaTV 201, Expansion Cohorts



## Prior treatments:

1. 5-FU+Carboplatin;
2. Carboplatin + Paclitaxel;
3. Capecitabine

Esophageal Cancer

# Reaching Our Vision: Tisotumab Vedotin

## Clinical Development: Ongoing Ph II Studies

### Ph II, recurrent or metastatic cervical cancer (innovaTV 204)

- Potentially pivotal
- Up to 100 pts
- Single arm, monotherapy
- Primary endpoint: confirmed objective response rate
- Secondary endpoints: duration of response, PFS, OS

### Ph II, solid tumors (innovaTV 207)

- Colorectal neoplasms, NSCLC, pancreatic cancer, SCCHN
- Up to 200 pts
- Single arm, monotherapy
- Primary endpoint: ORR
- Secondary endpoints: Safety, disease control rate, duration of response, time to response, PFS, OS

### Ph II, ovarian cancer (innovaTV 208)

- Ovarian cancer, fallopian tube cancer, peritoneal cancer
- Up to 142 pts, incl 12 pt safety run-in
- Monotherapy:
- 2 schedules: every 3 weeks and dose dense
- Primary endpoints: Safety & ORR
- Not yet recruiting

# Reaching Our Inspirational Vision: Early Stage Clinical Pipeline

Tahi Ahmadi, Senior Vice President Oncology and Translational Medicine



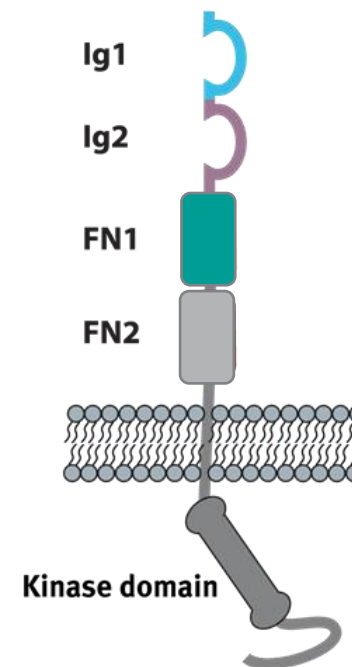
# Enapotamab Vedotin (HuMax<sup>®</sup>-AXL-ADC)



# Scientific Background

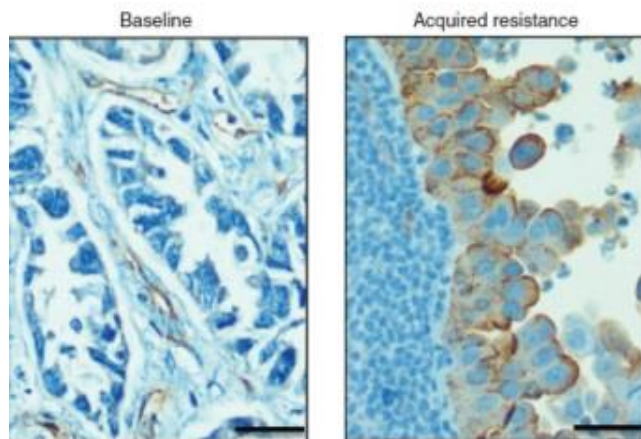
## AXL in Cancer Biology

- First identified in 1988 as a transforming gene in CML (*Liu et al., 1988*)
- AXL is aberrantly expressed in various cancers, including:
  - Sarcoma
  - Non-small cell lung cancer (NSCLC)
  - Ovarian cancer
  - Malignant melanoma
  - Glioblastoma
- AXL over-expression:
  - Associated with poor clinical prognosis in many cancer types
  - Prevalent in tumors with mesenchymal features
  - Associated with (acquired) resistance to therapy (TKI, chemo, immune checkpoint inhibitors)



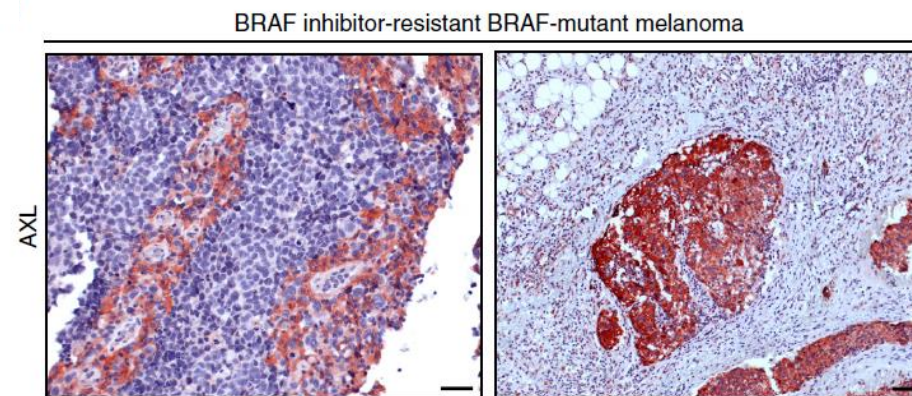
# AXL Expression is Associated with Resistance

Increased AXL expression in ~20-30% of NSCLC that progressed on Erlotinib/Gefitinib



Zhang et al., Nature Genetics 2012

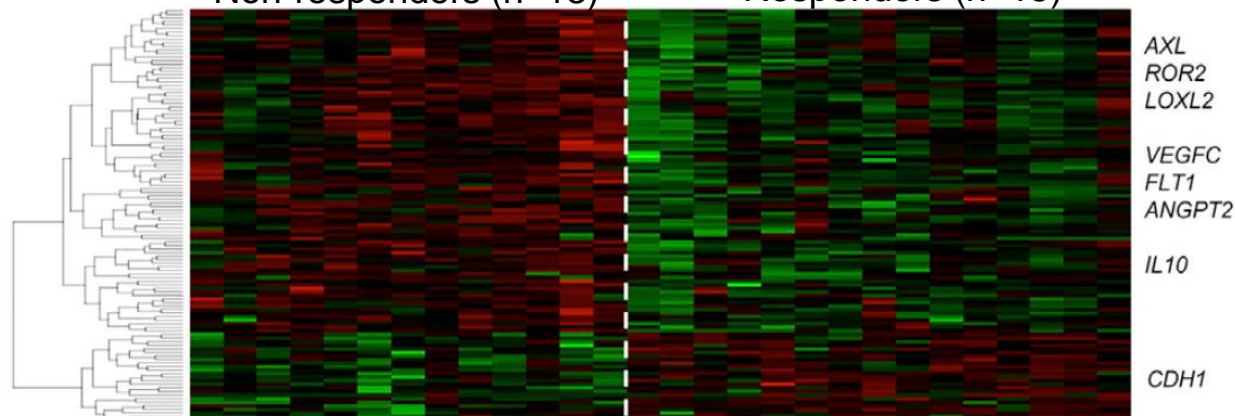
Increased AXL expression in 5/7 BRAF-mut and 2/3 NRAS-mut melanomas that progressed on BRAFi or MEKi, respectively



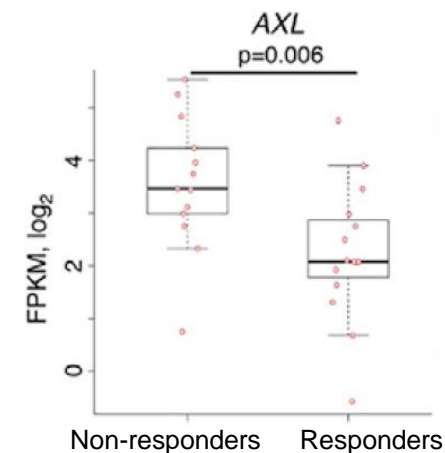
Boshuizen et al., Nature Medicine 2018

Increased AXL expression in melanoma tumors intrinsically resistant to PD-1 blockers

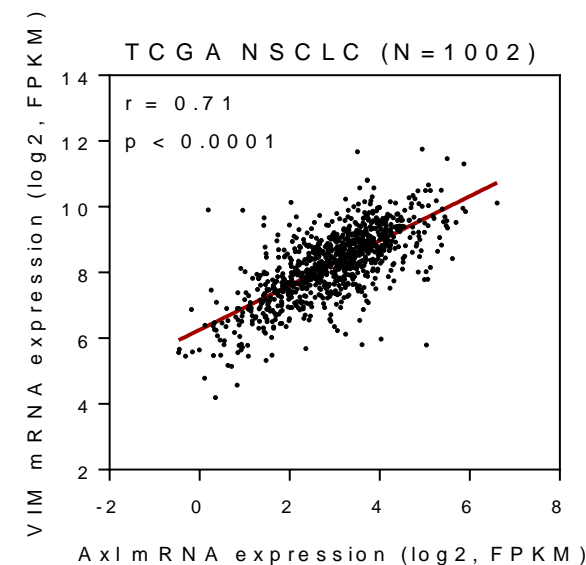
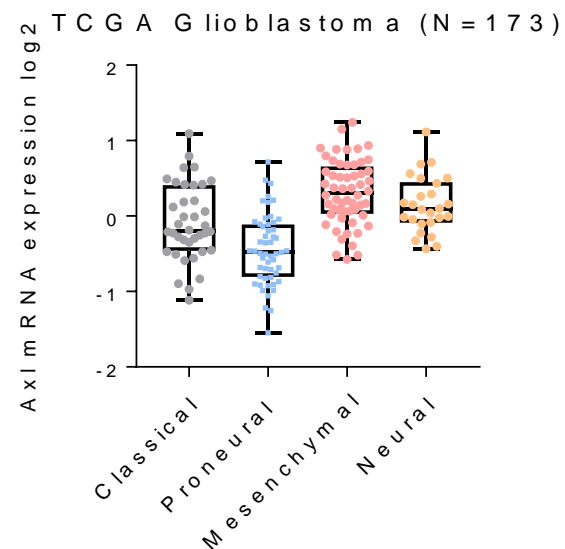
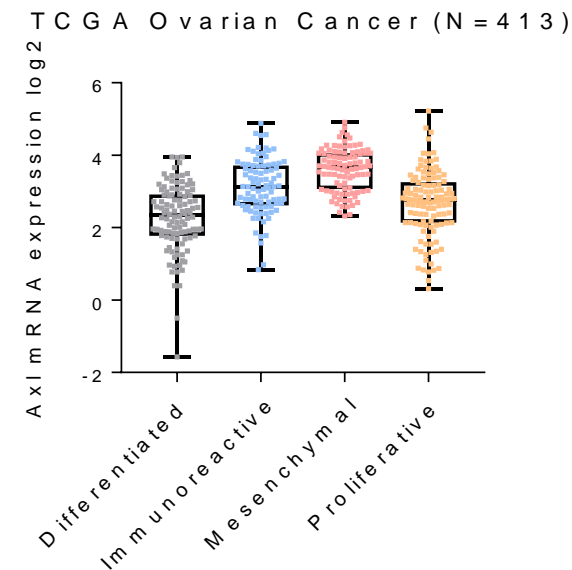
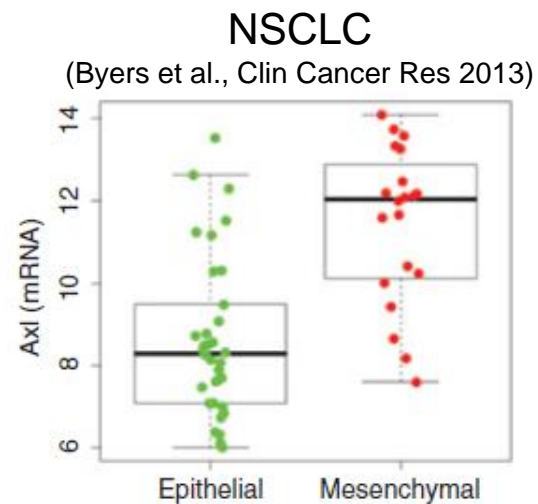
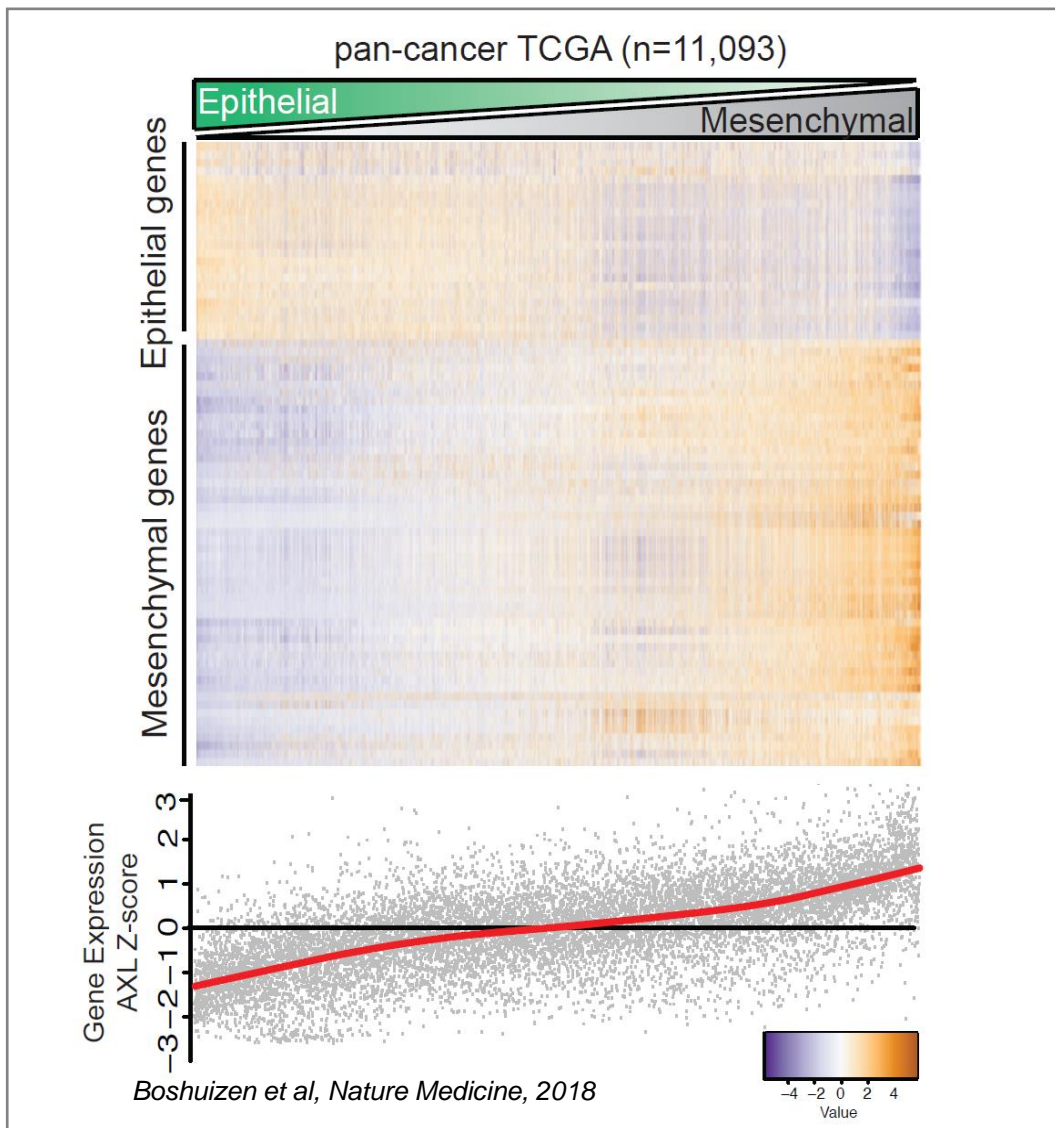
Non-responders (n=13)      Responders (n=15)



Hugo et al., Cell 2016

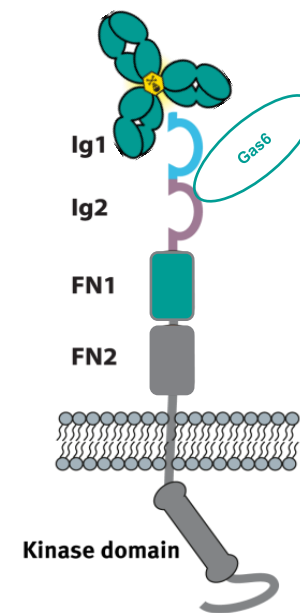


# AXL is Overexpressed in Advanced Tumors Enriched in Mesenchymal Features

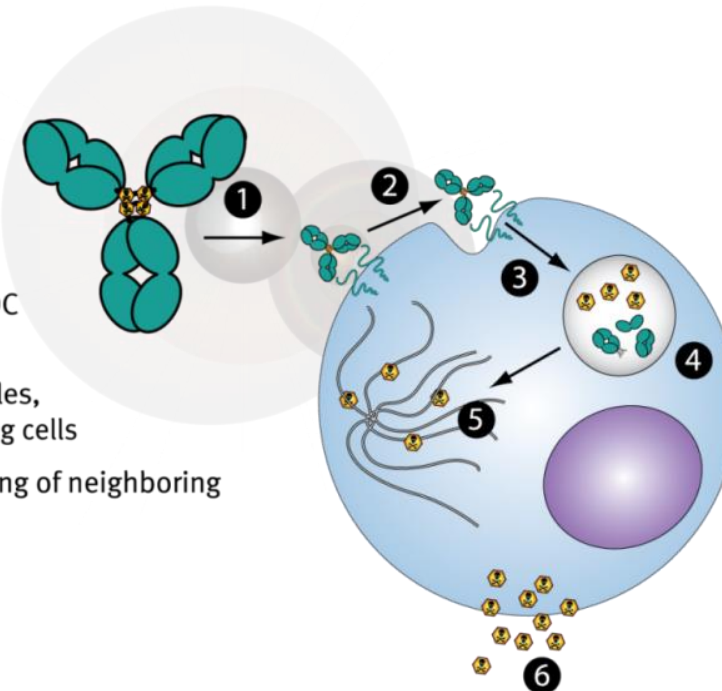


# Enapotamab Vedotin (HuMax-AXL-ADC)

- First-in-Class human anti-AXL mAb vc-linked to MMAE
- Binds Ig1-AXL, does not compete with Gas6
- Naked antibody internalizes efficiently
- AXL-dependent, MMAE-mediated cytotoxicity in AXL-expressing tumor cells
- May target heterogeneous tumors via bystander kill
- Potent anti-tumor activity in various solid tumor xenograft models

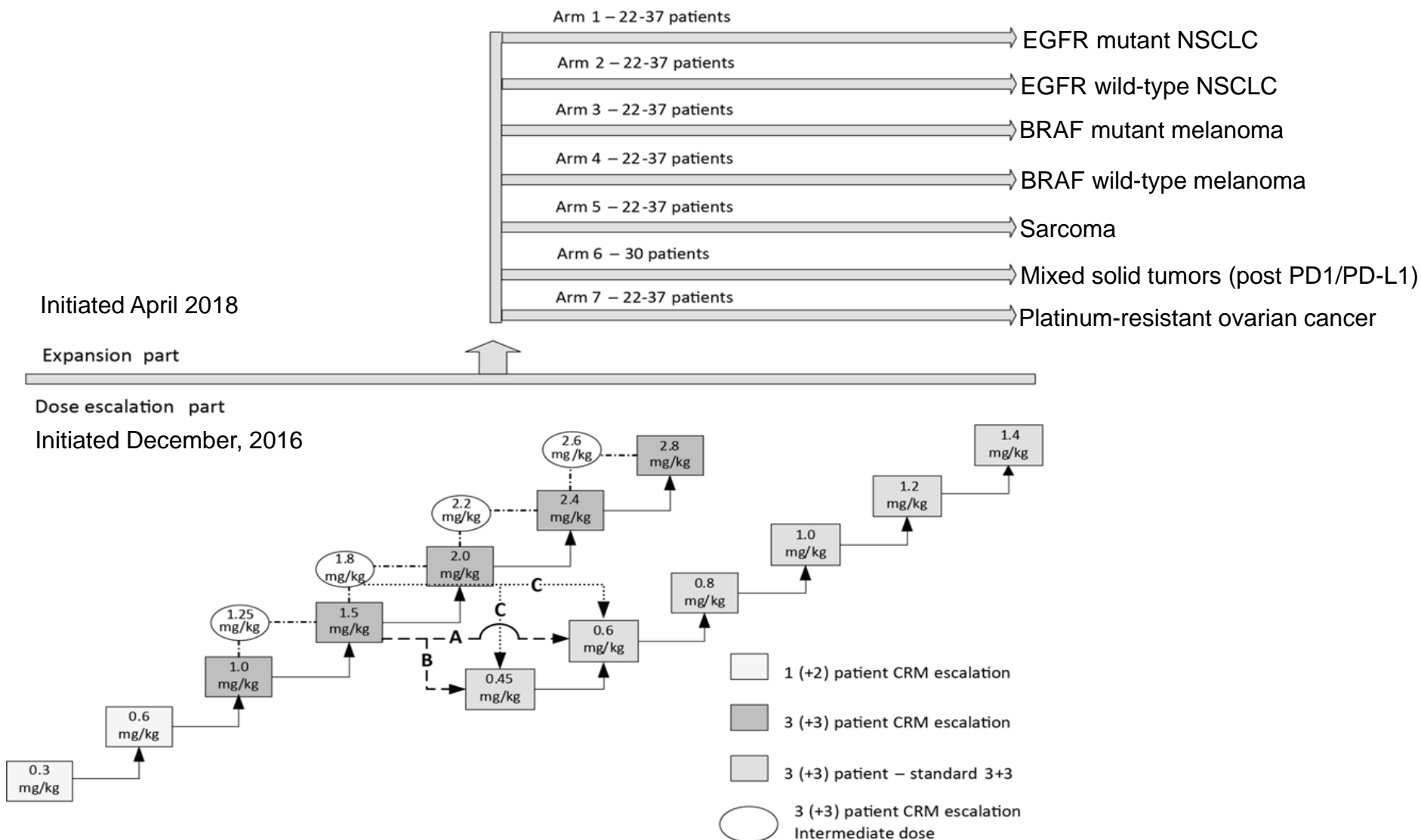


- 1 Binding to AXL-positive tumor cells
- 2 Internalization of HuMax-AXL-ADC
- 3 Trafficking to the lysosomes
- 4 Lysosomal degradation of HuMax-AXL-ADC and intracellular release of MMAE
- 5 MMAE-mediated disruption of microtubules, resulting in apoptotic cell death of dividing cells
- 6 Release of MMAE induces bystander killing of neighboring cancer cells



# Enapotamab Vedotin (HuMax-AXL-ADC)

## GCT1029-01 Phase I/II Trial Design



# Enapotamab Vedotin (HuMax-AXL-ADC)

## Cohort 2: Unmutated NSCLC



Case report: stage 4 NSCLC patient

# GEN1029 (HexaBody-DR5/DR5)

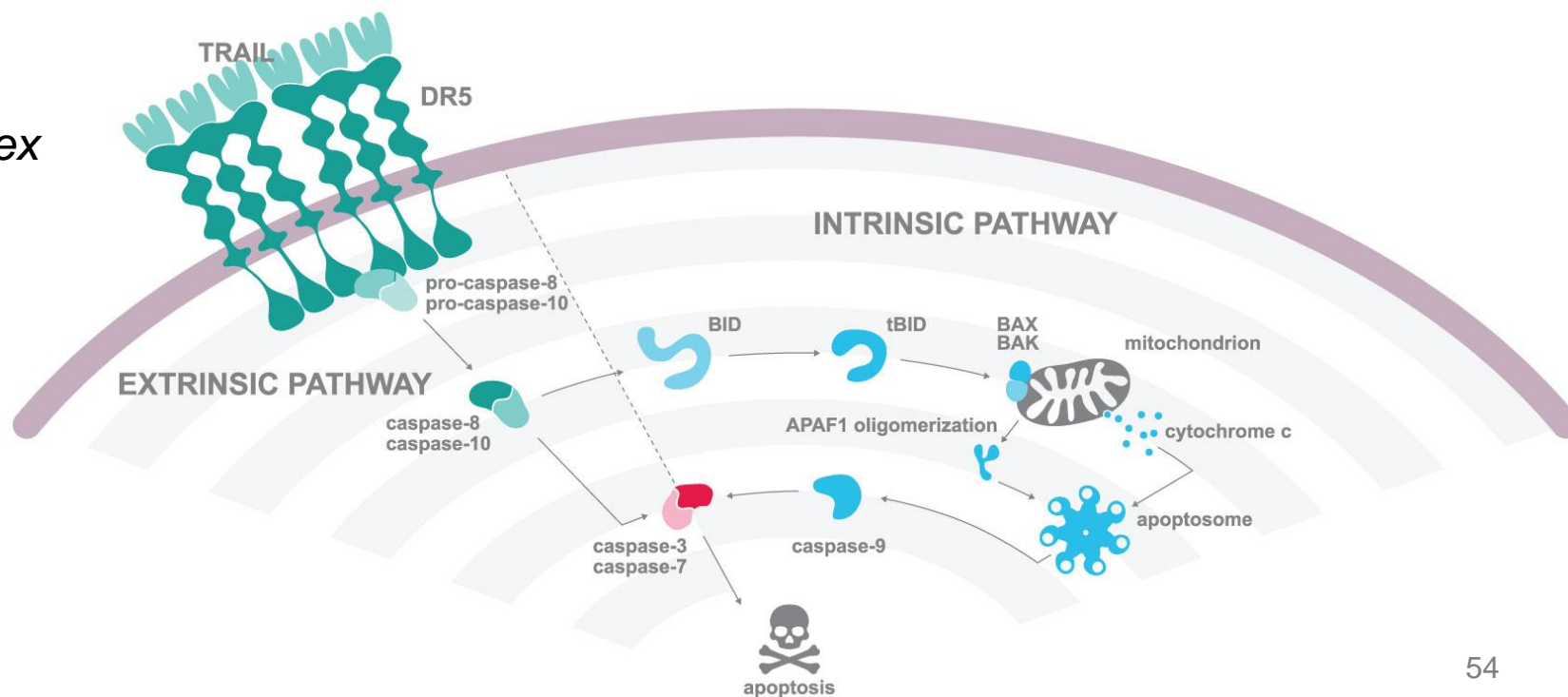
# DR5 as a Target for Anti-tumor Therapy

## DR5

- Death Receptor 5
- Tumor Necrosis Factor Receptor Superfamily member TNFRSF10b
- Receptor for TRAIL (TNF-related apoptosis-inducing ligand)

## DR5 activation upon TRAIL binding results in apoptotic signaling

- Binding of TRAIL trimers to DR5 on the cell surface
- DR5 hyperclustering
- Intracellular DISC formation  
*death-inducing signaling complex*
- Caspase activation
- Programmed cell death



# DR5 as a Target for Anti-tumor Therapy

## DR5 expression and function

- DR5 is ubiquitously expressed in normal & malignant tissues
- Role in attenuating autoimmune reactions
- Protective function against oncogenic transformation
  
- Malignant cells more sensitive to TRAIL-induced killing than normal healthy cells
- Increased DR5 expression reported for several tumor indications

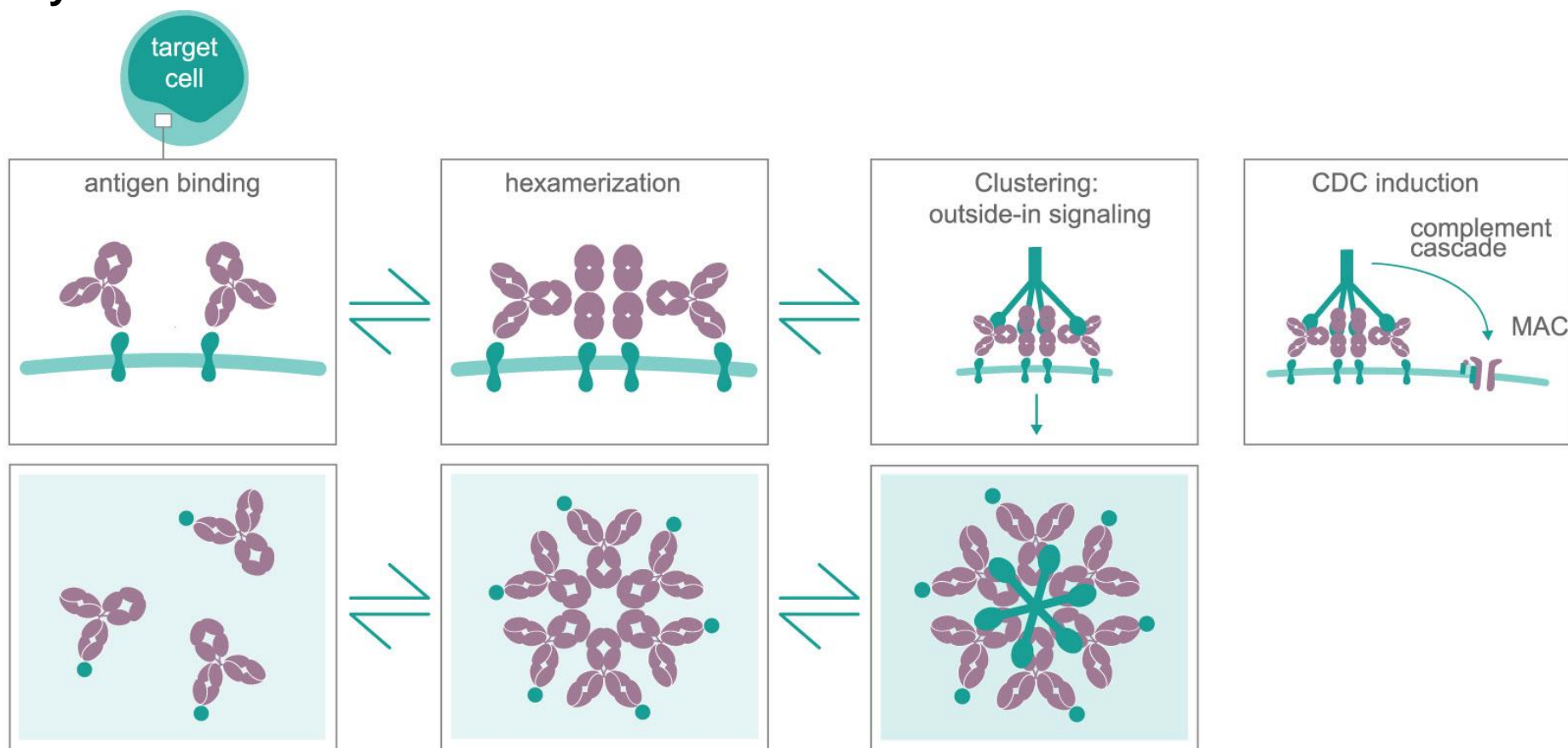
## Multiple DR5 agonists have been in clinical trials for treatment of cancer

- Clinical results with anti-DR5 monoclonal antibodies have been disappointing due to lack of efficacy
- DR5 agonist activity “conventional” IgG1 DR5-specific antibodies dependent on Fc $\gamma$ R-mediated crosslinking
- Lack of clinical activity has been attributed to insufficient Fc $\gamma$ R-mediated crosslinking in patients

# From Science to HexaBody Platform

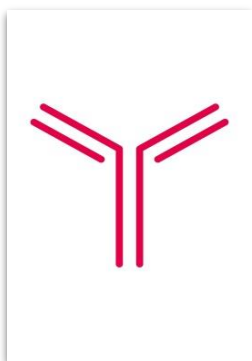
## Applications

- Enhanced C1q binding and complement activation
- **Improved antibody-mediated clustering of cell surface receptors, e.g. to induce death receptors agonist activity**



# GEN1029 (HexaBody-DR5/DR5)

## Anti-tumor Activity Independent Of Fc $\gamma$ R-mediated Crosslinking



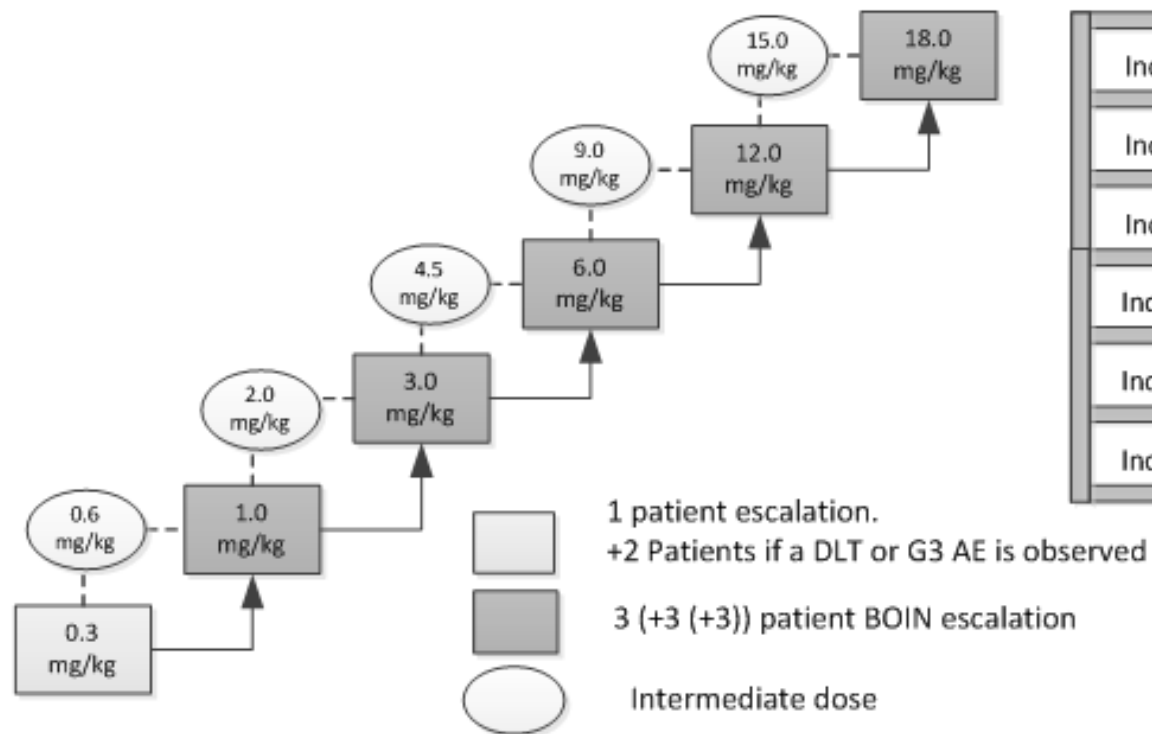
Anti-tumor activity of HexaBody-DR5/DR5 is independent of Fc $\gamma$ R-mediated effector function/crosslinking

- In contrast to conventional DR5-specific antibody conatumumab
- Fc $\gamma$ R independent tumor cell kill demonstrated in vitro and in vivo

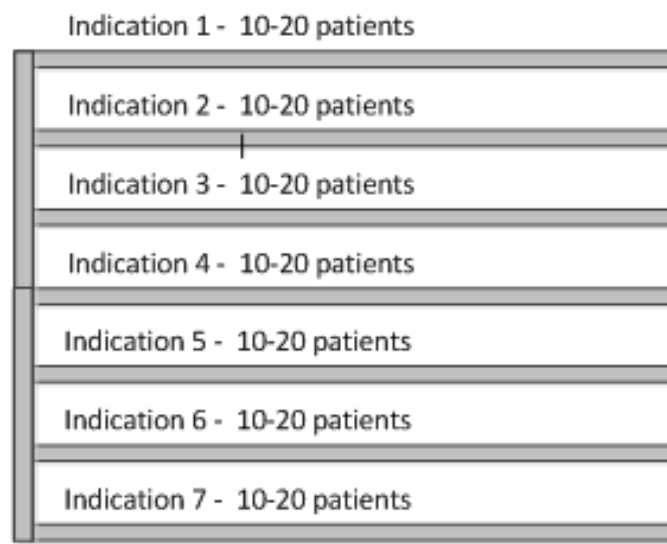
# GEN1029 (HexaBody-DR5/DR5) GCT1029-01 Phase I/II Trial Design

## Dose escalation phase

initiated May 2018



## Expansion phase



# GEN1029 (HexaBody-DR5/DR5)

## Dose Escalation



Case report: colorectal cancer patient

# GEN3013 (DuoBody-CD3xCD20)

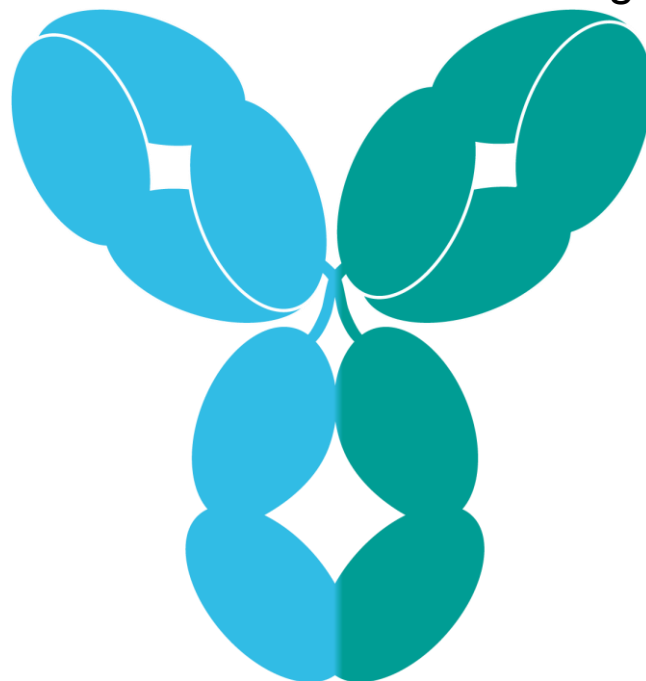
# GEN3013 (DuoBody-CD3xCD20)

## CD3 $\epsilon$ on T cells

- On all T cell subtypes
- Part of T cell receptor-CD3 complex
- Crosslinking induces T cell activation

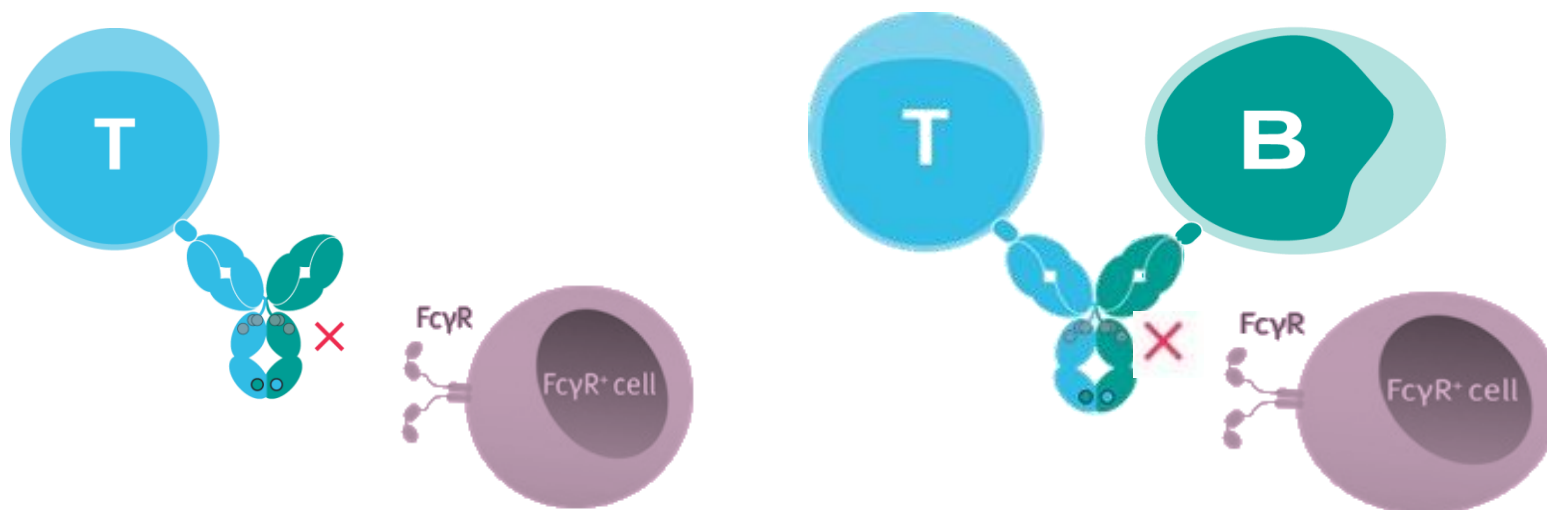
## CD20 on B cells

- On pre-B cells to plasmablasts
- Expressed on a wide variety of B cell malignancies
- Well-validated therapeutic target



## GEN3013 (DuoBody-CD3xCD20)

Has Inert Fc Region That Ensures Only Target-specific T cell Activation



### 3 point mutations are introduced that ensure:

- **no** Fcγ receptor binding (no ADCC or ADCP induction)
- **no** C1q binding (no CDC induction)
- **normal** FcRn binding (long plasma half-life like regular IgG1 antibodies)

# GEN3013 (DuoBody-CD3xCD20)

## Subcutaneous Administration Route

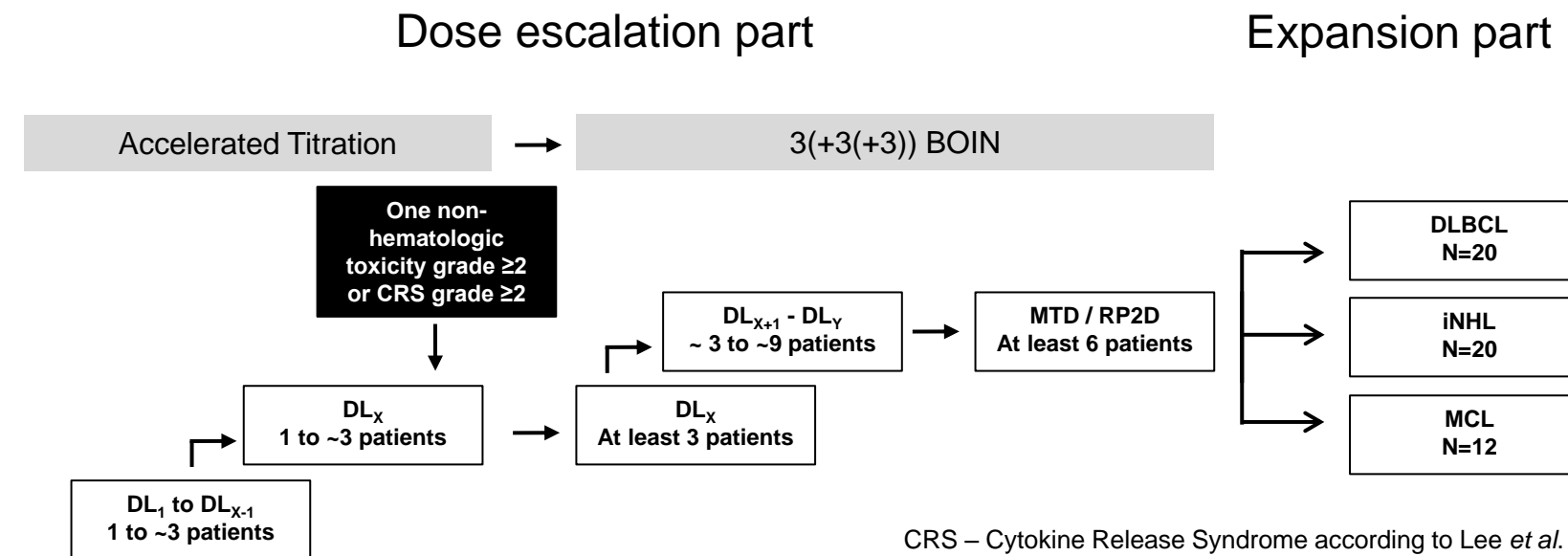


Pharmacokinetics, Cytokine Release and B cell depletion were compared between intravenous and subcutaneous administration routes

# GEN3013 (DuoBody-CD3xCD20)

## GCT3013-01 Phase I/II Trial

- Monotherapy Dose Escalation SC plus Expansion SC



Activated in June 2018

CRS – Cytokine Release Syndrome according to Lee *et al.*  
 DL – Dose Level  
 DLBCL – Diffuse large B cell lymphoma  
 iNHL – indolent Non-Hodgkin's lymphoma  
 MCL – Mantle cell lymphoma  
 MTD – Maximum tolerated dose  
 RP2D – Recommended phase 2 dose  
 BOIN – Bayesian Optimal Interval

# Building the Business: Translational Research

Kate Sasser, Corporate Vice President, Translational Research



# Genmab's Core Research Areas

Novel and cutting-edge ANTIBODY FORMATS and TECHNOLOGIES

Transformative antibody PRODUCTS that can be combined in modular fashion

Tools and data science capabilities for PERSONALIZATION of our antibody therapeutics

PEOPLE and PROCESSES to support Genmab's evolution



# Genmab is Building a Library of Antibody Therapeutics That can be Tailored to Patients



Genmab aims to invent, develop & commercialize differentiated antibody-based products; which can be utilized in a modular fashion, creating unique & tailored therapeutics to transform cancer treatment

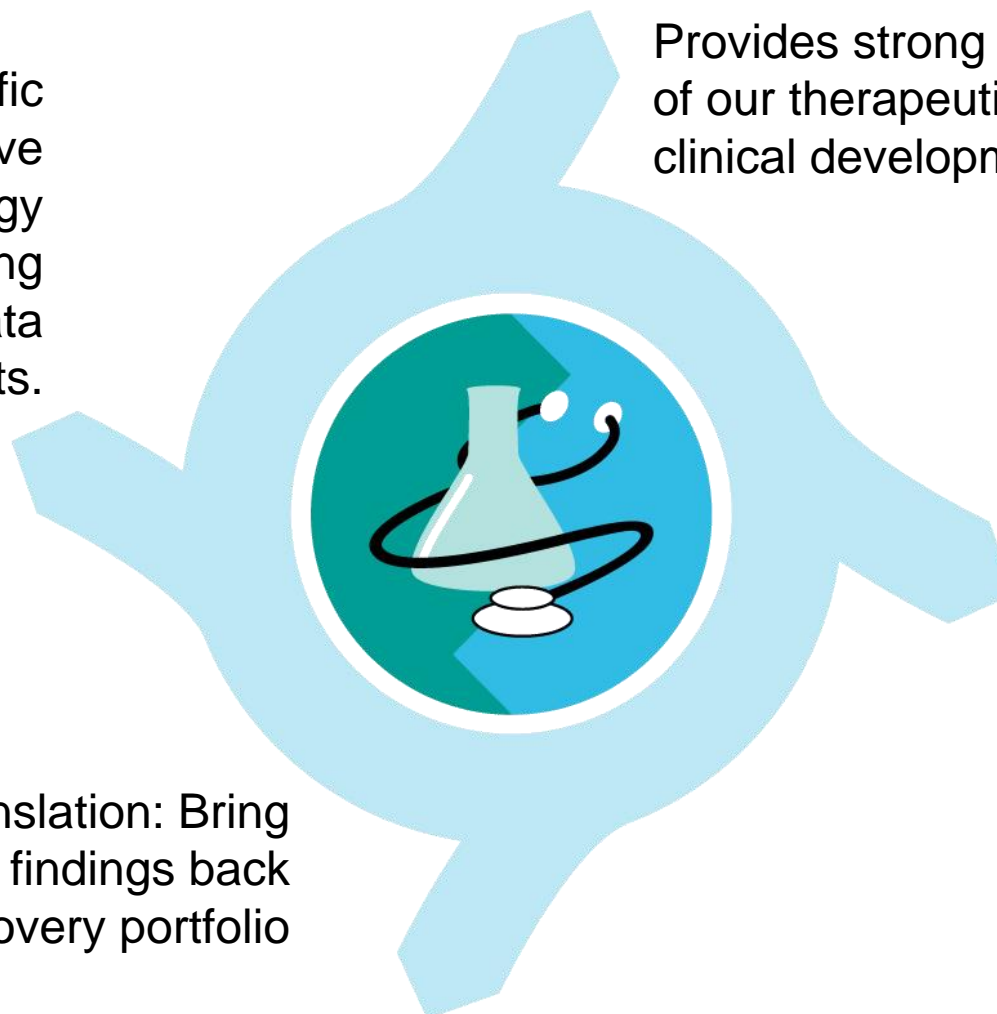
Tailoring of Genmab therapeutics requires strong  
**TRANSLATIONAL RESERACH**

# Mission for Genmab Translational Research

Develop Transformative Medicines by Finding the Right Medicine, at the Right Dose, for the Right Patient.

Generate deep scientific insights by utilizing innovative computational biology approaches and integrating translational & clinical data sets.

Provides strong biological understanding of our therapeutic products to help drive clinical development decision making.



Reverse Translation: Bring novel scientific findings back to our Discovery portfolio

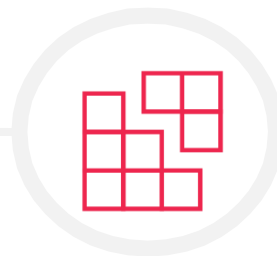
Understand the biology of our products, and our patients, to match the right drug with the right patients.

# Translational Research Will Focus on Patient Profiling in Order to Create Tailored Therapeutics in the Future



## Holistic patient characterization Clinical Translational Labs

- Establish strong translational laboratory capabilities to profile patient tumor and immune genotype/phenotype



## Match patients with appropriate therapy

- Establish diagnostic modalities to match Genmab antibody therapies with appropriate patient populations and drive personalized treatment recommendations



## Integrate Data to capture insights and identify novel targets and formats

- Expand Data Science capabilities to be able to probe our clinical and translational data to uncover novel targets and approaches.

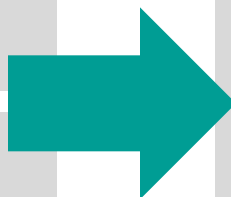
# Generating Insights from Data: Establishing Strong Data Sciences

Building robust DATA SCIENCES unit to obtain insights into diseases of interest, novel targets, our patients, and our products

## Access to data: Building our Genmab data sets

Establishing collaborations within academia, industry, biotech, and consortiums to bring in large data sets

Generate patient data from our own portfolio of products and clinical trials



## Data mining and insight generation

Processes data into actionable insight:

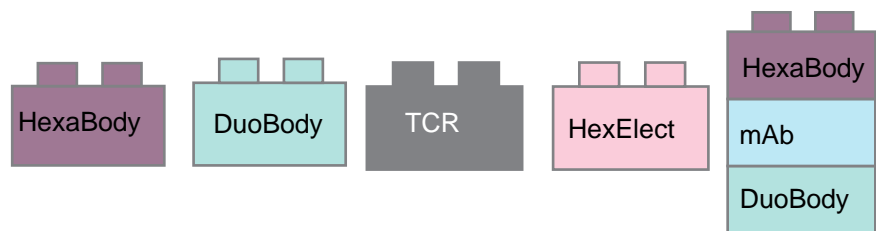
- Create and investigate hypotheses
- Identify novel targets and product profiles
- Develop in silico models for predicting mechanism of action, dose, and biomarkers for our therapeutics
- Speed clinical development decisions

## BENEFITS of INSIGHT

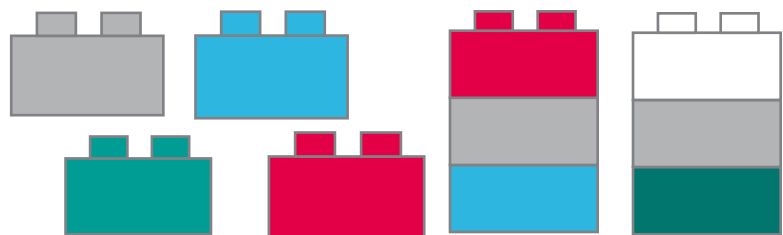
- Expand pipeline of early products
- Develop better models for predicting dose, toxicity, and patient biomarkers
- Make clinical dev decisions faster
- Understand patient populations and indications
- Develop patient diagnostic approaches

# Translational Research at Genmab Will Tie Together Core Research Areas of: PLATFORMS, PRODUCTS & PATIENTS

Maximizing & expanding our repertoire of antibody **PLATFORMS** and delivery methods

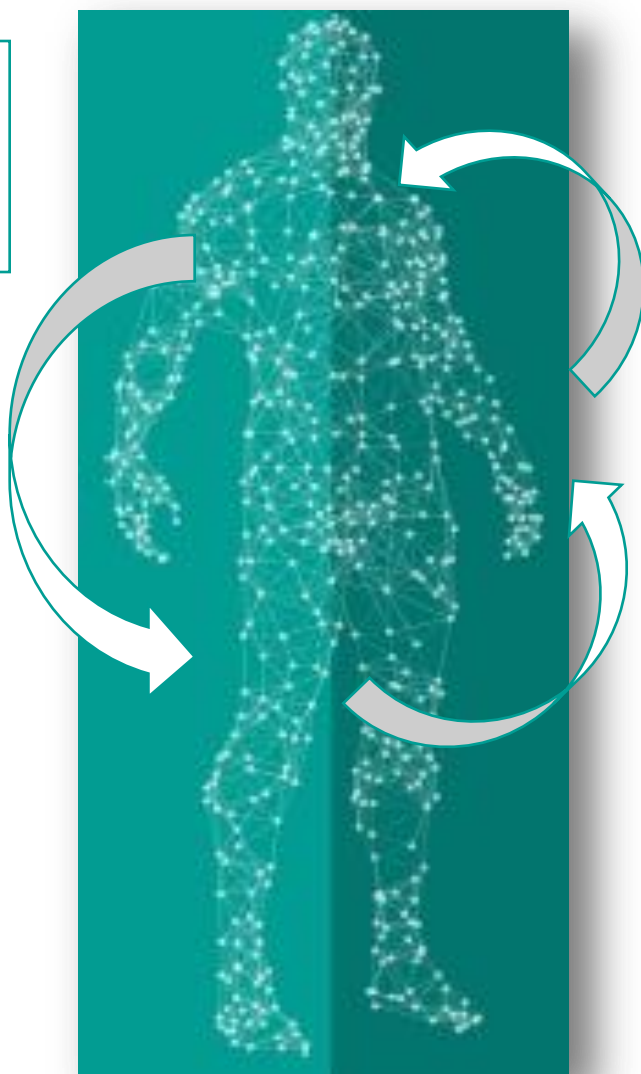
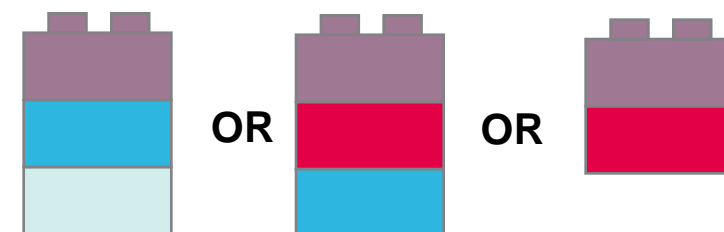


Identify novel targets and expand Genmab library of innovative **PRODUCTS**



In depth understanding of our products and our **PATIENTS**

In depth **TRANSLATIONAL RESEARCH** will link clinical and preclinical knowledge together, enabling bench to bedside development.



# Building the Business: Commercial Capabilities

John Keating, Corporate Vice President, Commercial



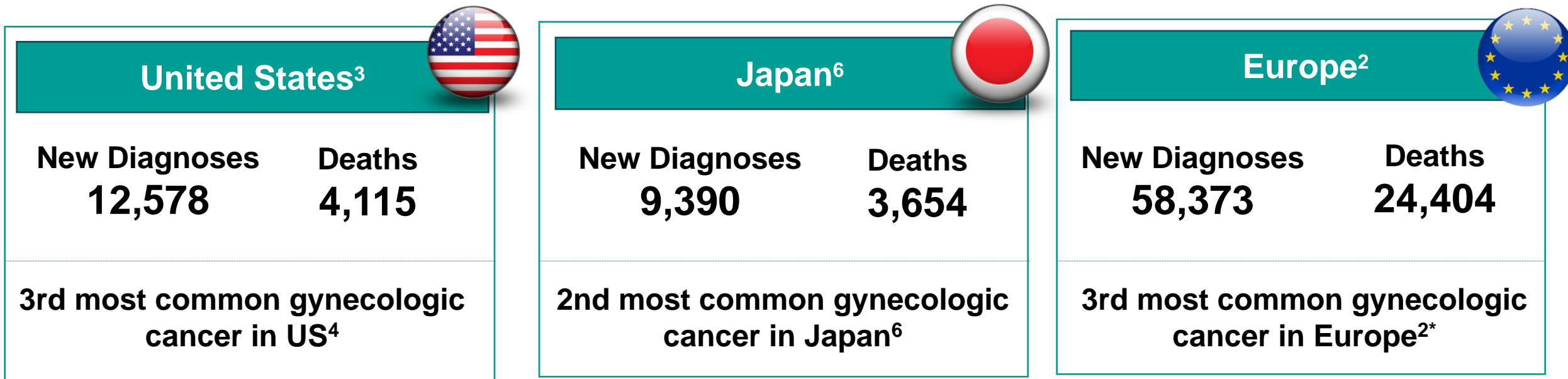
# Genmab: Building an Oncology Footprint



- Prepare now for successful *launch readiness by 2020*
- Build out **commercial leadership team** and *core capabilities* has started
- Organizational focus on *Europe, Japan and United States*

# Despite Increased Screening, Cervical Cancer Continues To Be a Health Burden in the US, Europe, and Japan

The overall incidence and mortality rate of cervical cancer has decreased due to regular screening with Pap tests, which effectively identifies cervical pre-cancer, however...<sup>1</sup>



In developed countries, incidence rates are low (<7.9 per 100,000 women) compared with **developing countries** in sub-Saharan Africa and Central and South America, where incidence is especially high (>30 per 100,000 women)<sup>5</sup>

\*Europe is defined as the 40 countries in the four United Nations-defined areas of Europe and the European Union (EU-27).

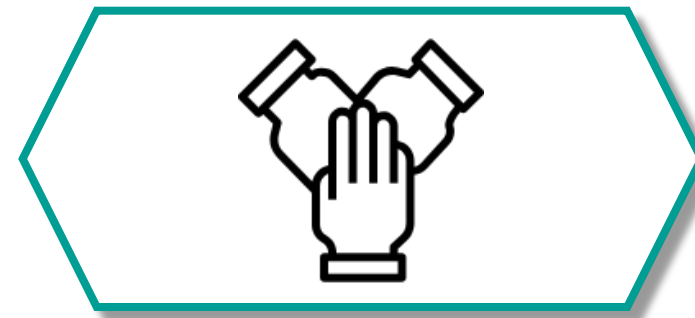
References: 1. American Cancer Society 2. EUCAN (2012) 3. Centers for Disease Control and Prevention. Cervical Cancer Statistics (2017) 4. UpToDate. 5. Ginsburg O et al. Lancet 2017 6. HPV Information Centre Japan (2017)

## US Commercial Readiness

 **Seattle Genetics**<sup>®</sup>

+

 **Genmab**



***Currently evaluating co-promotion options with Seattle Genetics.***

***Potential option to collaborate with Seattle Genetics to support the US launch of tisetumab vedotin and set up rest of portfolio for commercial success.***

# Japan Commercial Strategy: Build for a Unique Opportunity

*Independent entry presents high value with minimal upfront investment and stage-gated decisions*

## Low rate of cancer screening & HPV vaccinations

- In 2014, **32%** coverage rate for cervical cancer (vs. **83%** in US in 2010)
- In 2013, MHLW withdrew recommendation to use vaccines due to severe side effects
- Vaccination rate dropped from **70% to 0.7%**

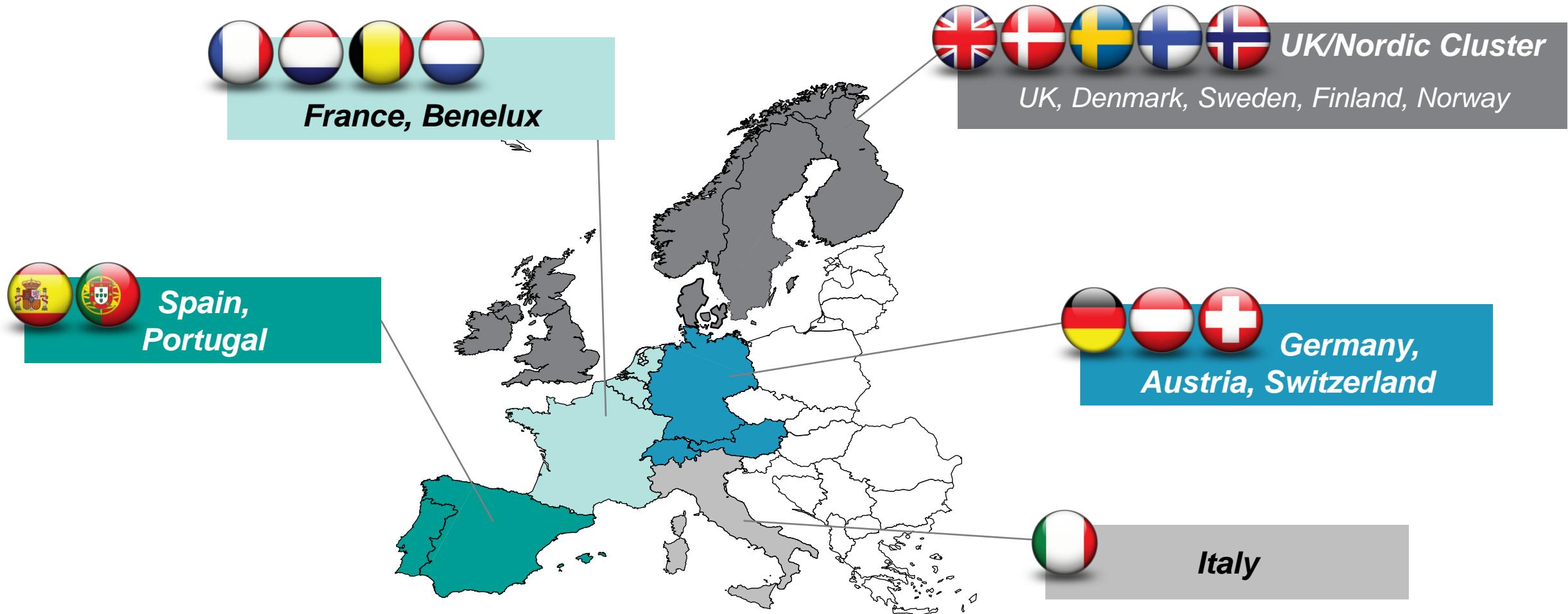
“Majority do not respond to treatment, and most **die within a year**”

## Commercial opportunity

- **No SoC for 2L treatment**
- **Modest commercial & medical affairs infrastructure** needed to support initial indication
- Clear path for **reimbursement and access**

“**High Unmet need for defined population** allows for modest investment to retain the most value”

# Potential EU Commercial Strategy: Build Footprint to Launch Tisotumab Vedotin Across Western Europe Ourselves ( 5 Local Clusters)



# Critical Steps for Operational Success in Genmab Lead Territories of Japan and Europe

- **Early investment in market access**, including building a seasoned value and reimbursement team today
- **Building local integrated teams** that have country-level authority to act independently and move quickly
- Engaging early with Japanese and European medical experts in cervical cancer and having the **medical affairs team in place**
- **Incorporating the voice of patient and key stakeholders** across Japan and Europe, understanding that there are important differences across countries
- A seasoned team in Japan and Europe with direct **sales and marketing experience**
- A **stepwise approach** that builds the team and footprint in a staged manner

# Near-Term Planning: Growth of People & Spend

**Today**

- *Global Commercial Operations Team*
- *Head of Commercial*
- *Head of Medical Affairs, Additional Staff*
- *Head of Market Access*
- *MSL's*

**Planning & Initial Growth**

**2019**

- *Building US, EU and Japan staff:*
  - *Sales & Marketing*
  - *Medical Affairs*
  - *Market Access*
  - *Systems*

**Hire Top Tier Leadership With Seasoned EU, Japanese and US Experience**

**2020**

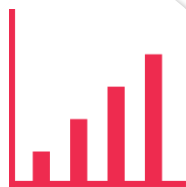
- *Additional Build:*
  - *Sales & Marketing*
  - *Medical Affairs*
  - *Supply Chain*
  - *G&A (EU)*
  - *Systems*

**Build Launch Readiness Through Key Deployment**

## Why We'll Be Successful



***Capital to support an oncology launch with appropriate resourcing***



***First launch in cervical cancer allows for initial focus on gynecologic cancers and provides foundation for future growth in new oncology indications***



***Europe oriented organization with existing partnerships in EU Oncology community***



***Early emphasis and investment on building the right team***

# Q&A



# Break



# The Next Generation of Therapeutics: Strategic Alliances

Jan van de Winkel, President & Chief Executive Officer



# Strategic Alliances

## Creating Next Generation Leapfrog Drugs

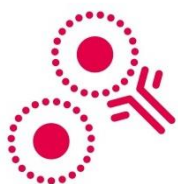


### Strategic alliances

- Immatics Biotechnologies
- BioNTech AG



Combines Genmab's proprietary technologies & antibody know-how with key outside capabilities




Strengthens Genmab's position in Immuno-Oncology



Further builds world-class Genmab pipeline

# Strategic Alliances

## Immatics



Strategic partnership: create & develop next gen. bispecific cancer immunotherapies



Genmab's tech. & antibody know-how + Immatics' XPRESIDENT® tech. & TCR technology



Exclusive access to multiple novel targets: joint research funded by Genmab



Strengthens our position in Immuno-Oncology



Genmab responsible for dev., manufacturing & WW commercialization

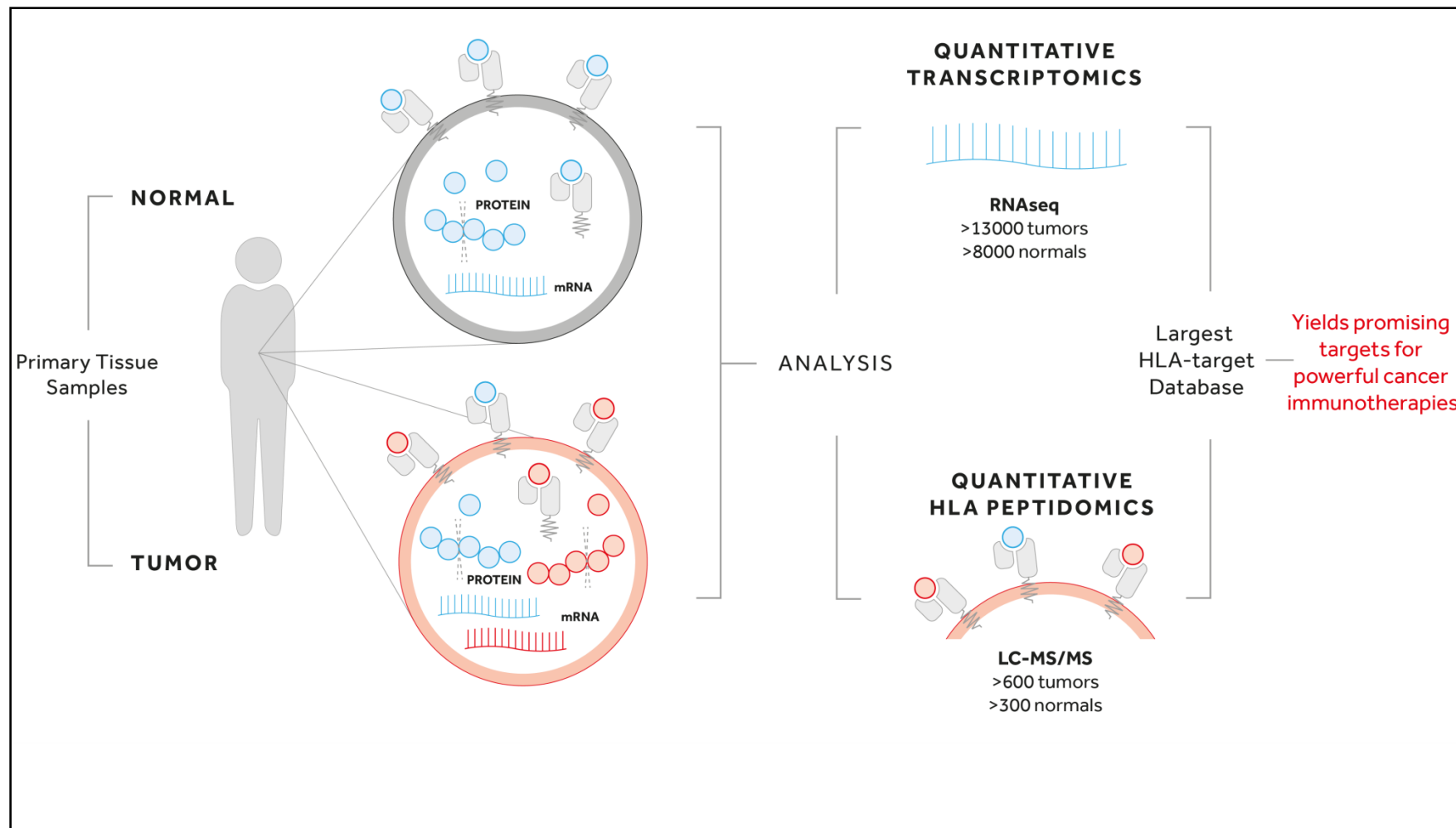


Immatics, option to limited co-promo. efforts in select EU countries



Upfront fee of USD 54M to Immatics, eligible for up to USD 550M in milestones per product & tiered royalties on sales

# Immatics XPRESIDENT® Platform



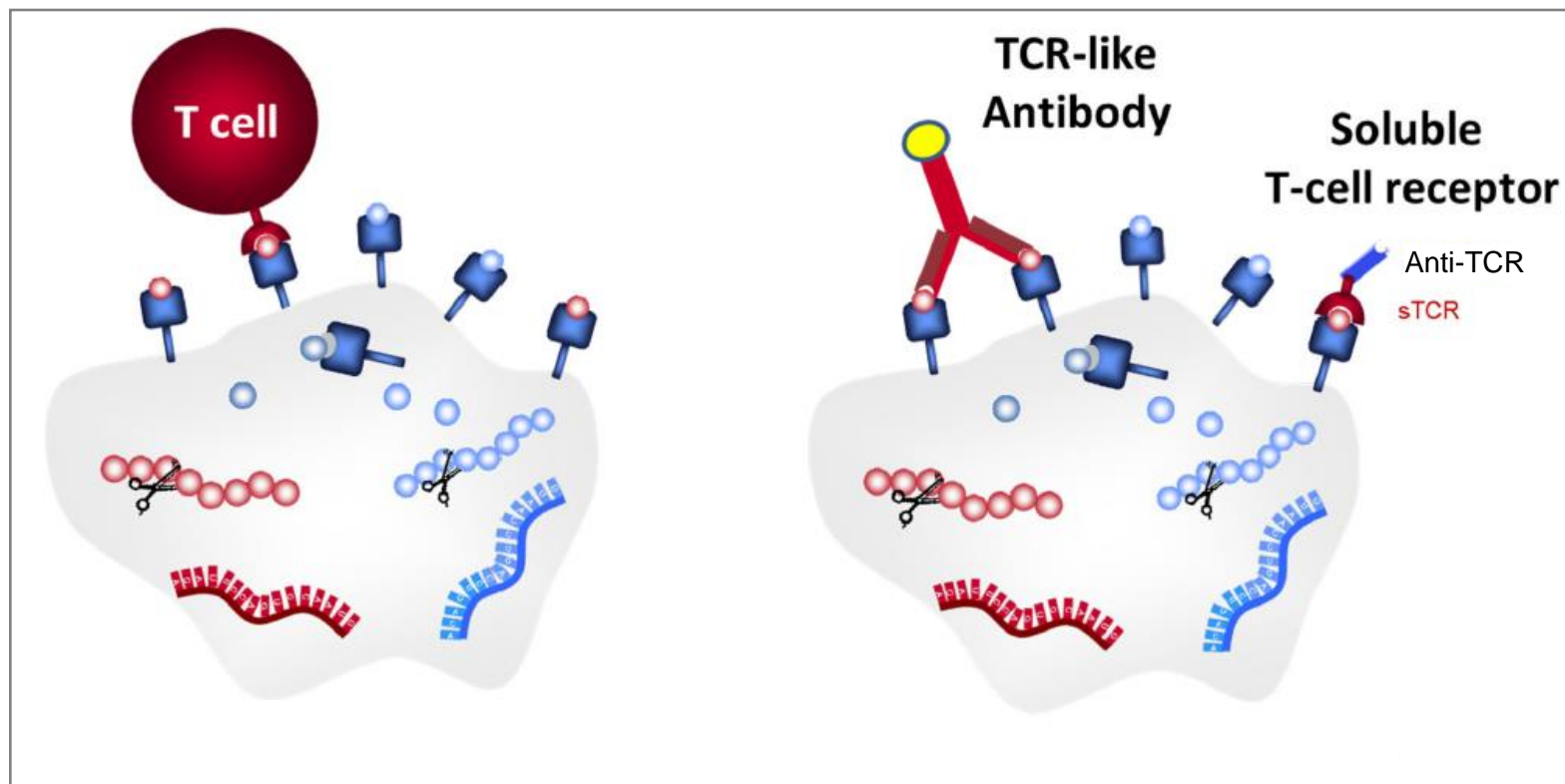
- Tumor biopsies as source
- Peptide identification
- Peptide selection
  - Quantitative transcriptomics
  - Quantitative peptidomics

# Immatics

## TUMAPs

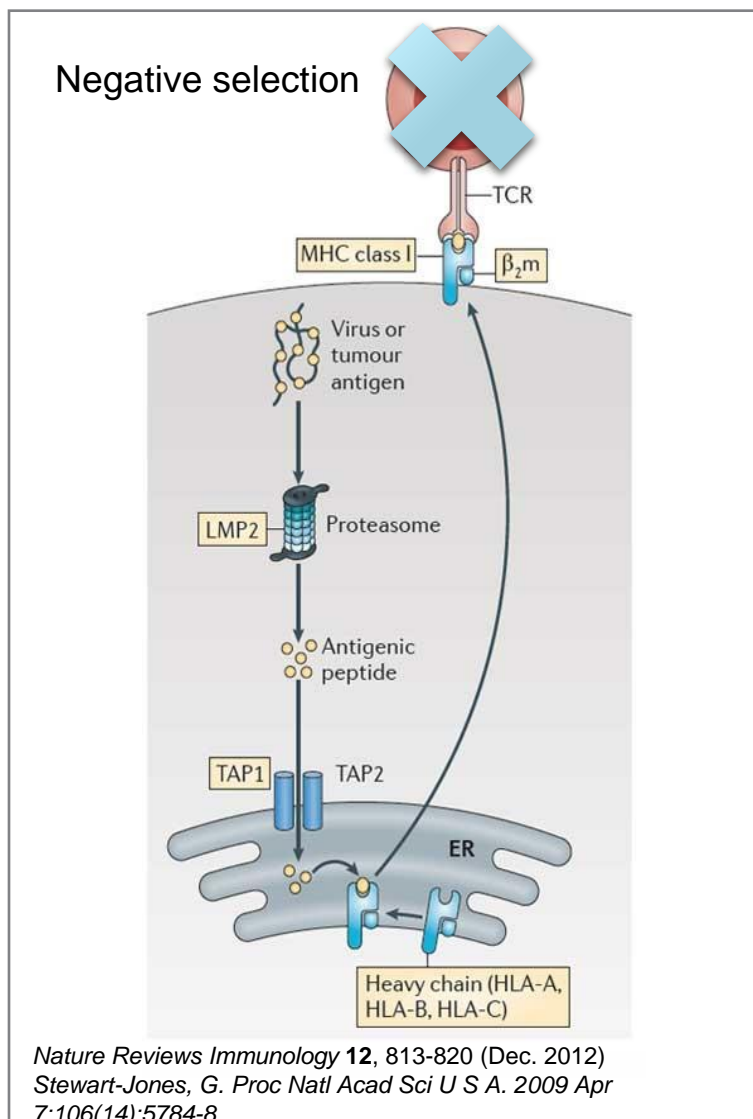
Targets for vaccines and adoptive cellular therapy

Intracellular targets for mAbs/sTCRs



# Immatics

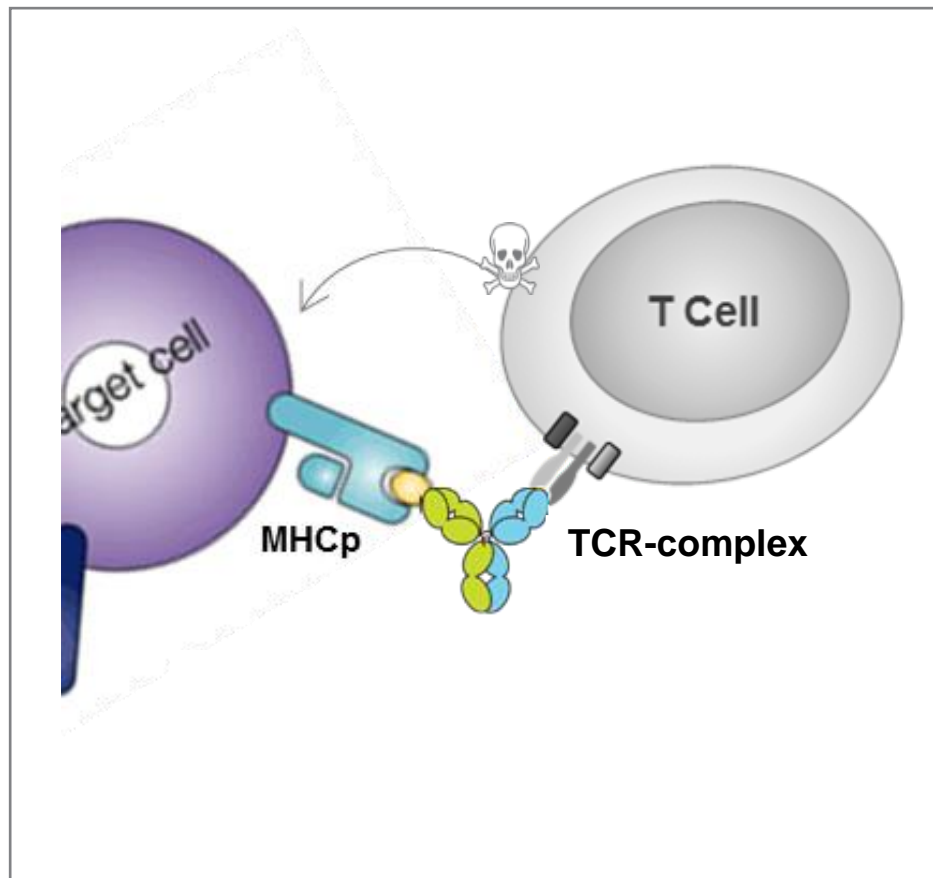
## Targeting Intracellular Antigens Presented on Surface HLA Molecules



- Intracellular proteins are being degraded by proteasome and presented on cell surface by MHC class I (HLA) to T cells
- T Cell receptors (TCR) can target cellular proteins through recognition of HLA-presented peptides
- Self-reactive T cells are being deleted through negative selection
- Cancer antigens are often regarded as 'self' → Immune tolerance
- TCR-mimicking antibodies (TCRm) represent a novel class of antibodies capable of selectively targeting tumors via MHC/peptide complexes

# Immatics

## Proof-of-Principle



# Strategic Alliances

## BioNTech



Strategic partnership:  
research, develop &  
commercialize  
bispecific antibody  
products



Genmab's DuoBody tech + BioNTech's proprietary antibodies



Antibodies against key immunomodulatory targets



For any products jointly selected for dev., costs & ownership shared 50:50



Supports strategy of creating broad pipeline of next-generation differentiated therapeutics

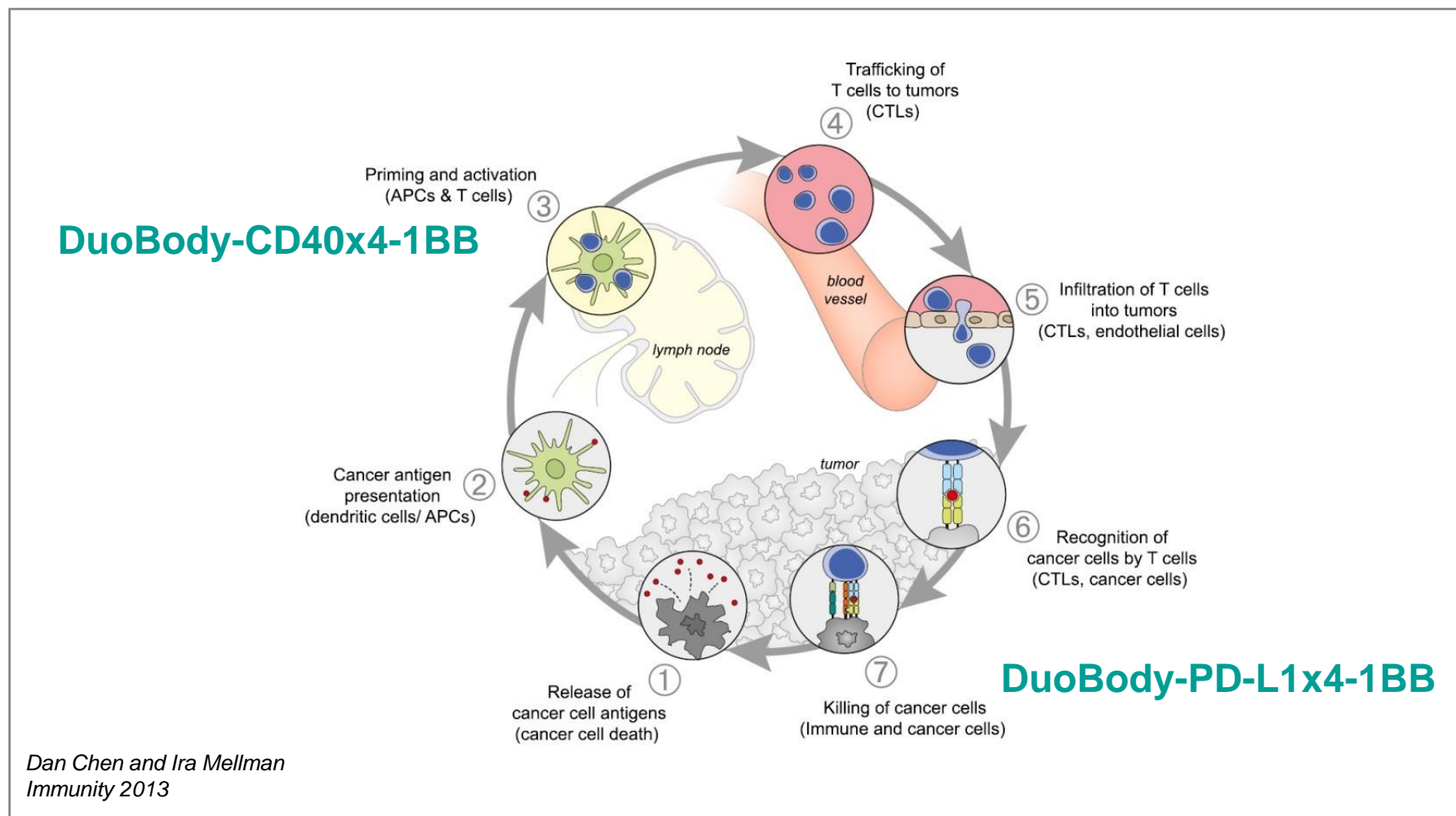


IND candidates in development



Upfront fee of USD 10M to BioNTech, additional potential USD 5M if certain assets selected for development

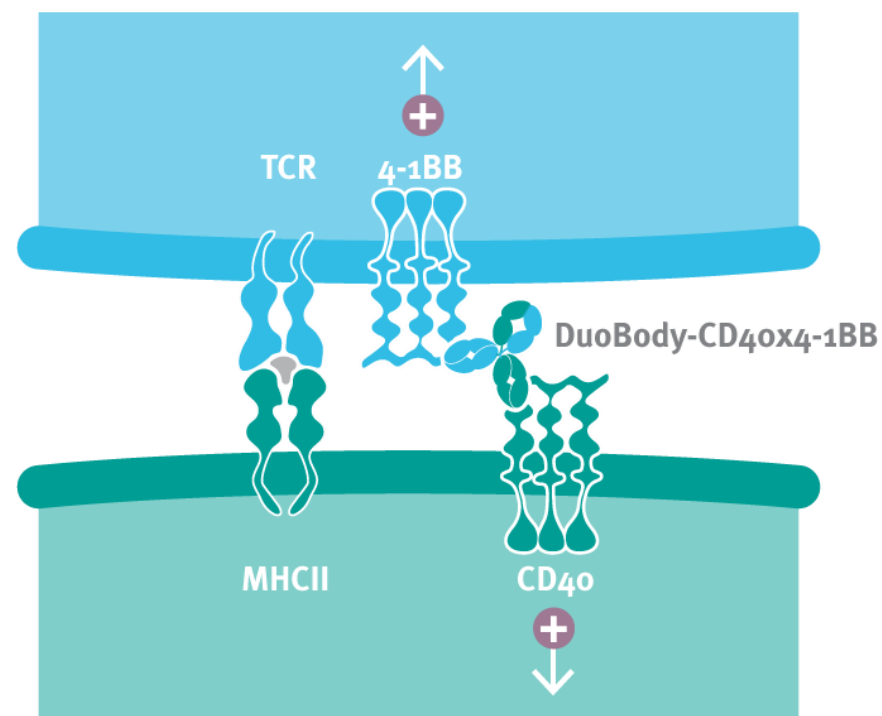
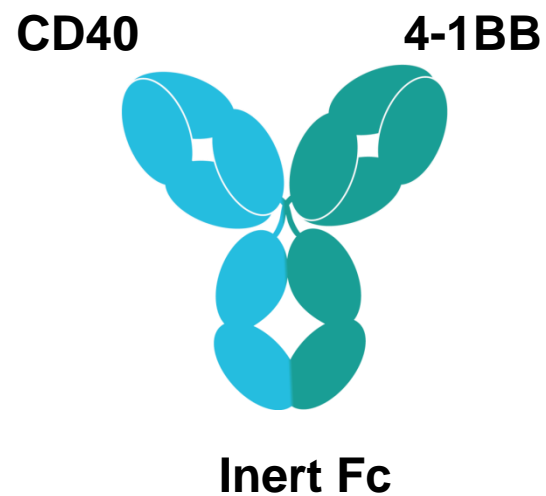
# Genmab-BioNTech Concepts in Cancer Immunity Cycle



- Series of stepwise events must be initiated & allowed for anticancer immune response to proceed & expand iteratively: depicted in the Cancer-Immunity Cycle
- DuoBody-CD40x4-1BB related to step 3 of cancer immunity cycle, leading to priming & activation of APCs and T cells
- DuoBody-PD-L1x4-1BB related to step 7 & direct killing of cancer cells

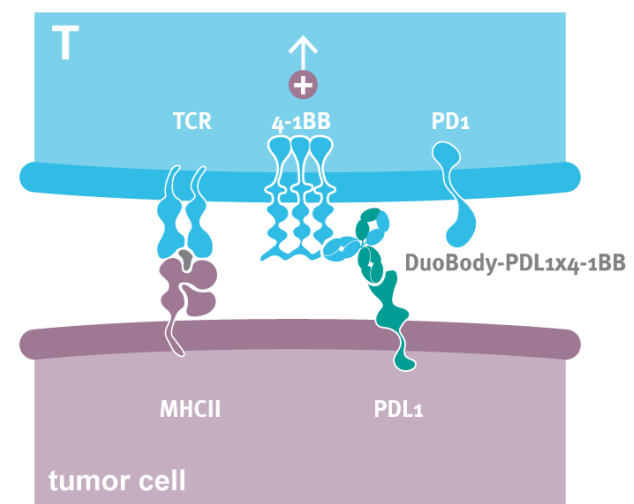
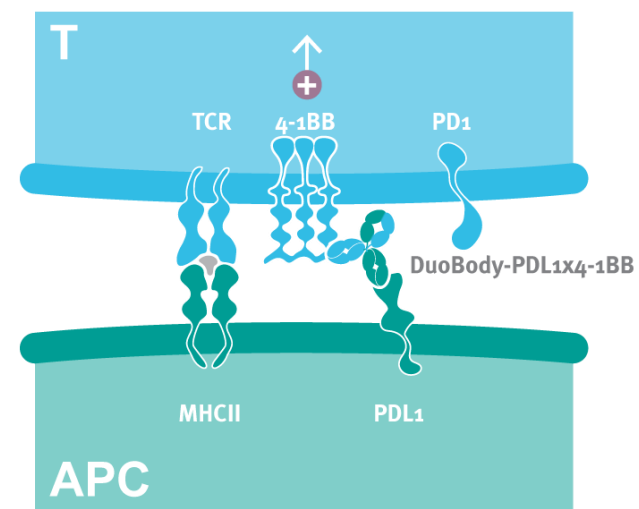
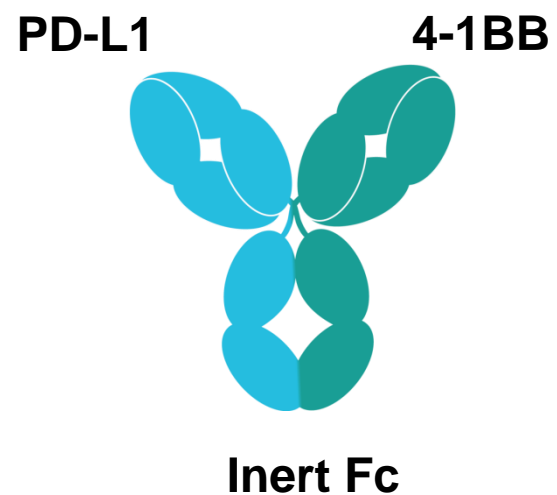
# BioNTech

## DuoBody-CD40x4-1BB



# BioNTech

## DuoBody-PD-L1x4-1BB



# Solid Financial Foundation

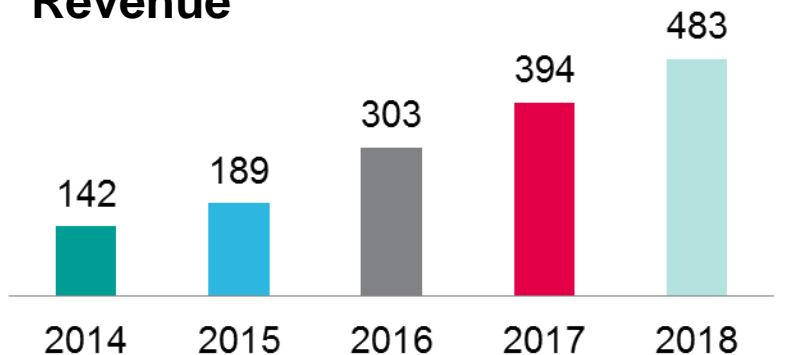
David Eatwell, Executive Vice President & Chief Financial Officer



# Strong Financial Foundation

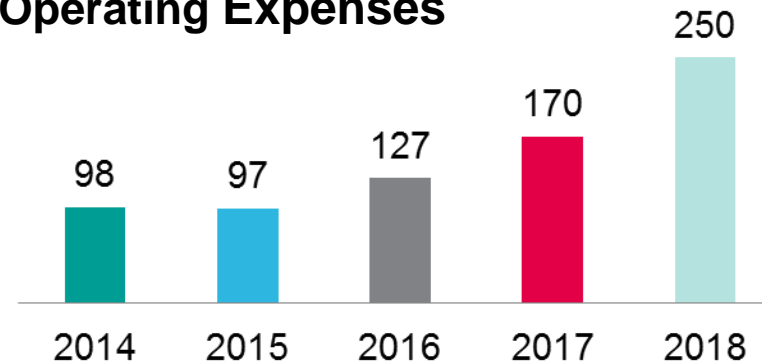
## Expanding Top Line

### Revenue



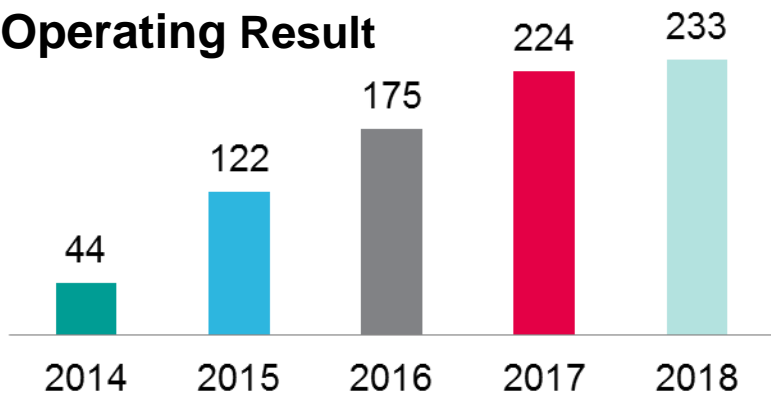
## Increasing Investment

### Operating Expenses



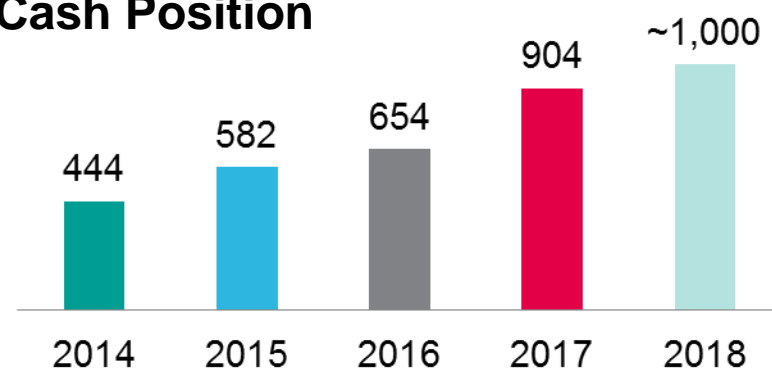
## Sustainable Profits

### Operating Result



## Well Capitalized

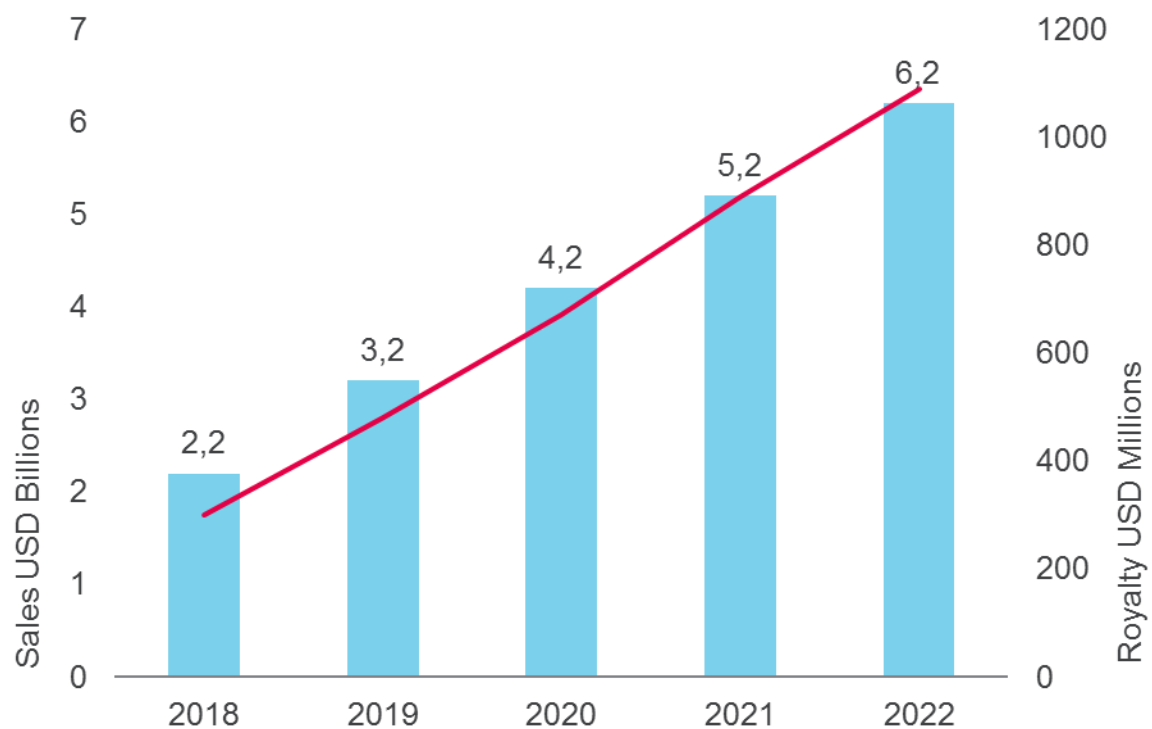
### Cash Position



# DARZALEX

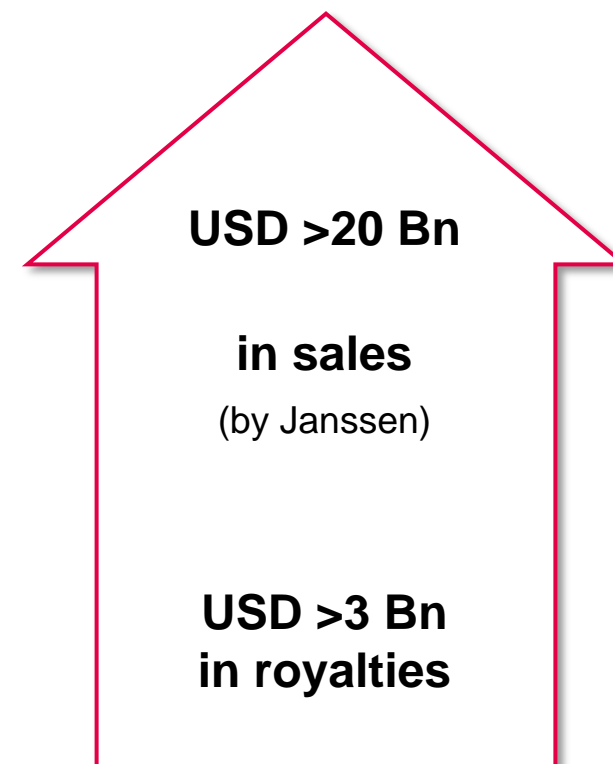
## Significant Earnings Potential

Analysts Projecting \$1bn+ Annual Growth



Tiered royalty 12-20%

Bars = Sales  
Line = Royalty



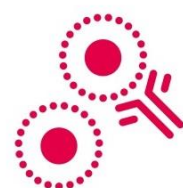
From 2018-2022

# Disciplined Investment



## Portfolio

- 2017: 2 products
- 2018: 4 products
- 2019: 7+ products



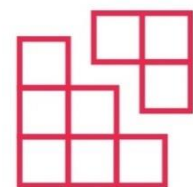
## Next Gen. Technologies

- DuoBody
- HexaBody
- HexElect



## Strategic Alliances

- BioNTech
- Immatics



## Capabilities

- Expanding: R&D and support
- Adding: Translational Research and Commercial

# In Summary

## Positioned for Success

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Significant earnings potential



Ability to invest in pipeline & technologies



In control of our destiny



Selectively invest in value creating products



Option to in-license / acquire or out-license



Retain ownership & achieve 2025 vision

# 2018 Achievements & Beyond

Jan van de Winkel, President & Chief Executive Officer



# 2018 Achievements

## DARZALEX

✓ FDA approval in U.S. in 1L MM, based on ALCYONE data

✓ EC approval in EU in 1L MM, based on ALCYONE data

✓ New Ph III D+RVd trial in 1L MM initiated

✓ Upward sales trajectory

## Ofatumumab

✓ Recruitment completed in Ph III SC RMS studies

## Tisotumab Vedotin

✓ Ph II study in multiple solid tumors started

✓ Ph II study in 1L/2L Cervical cancer planned

✓ Ph II study in Ovarian cancer planned

✓ First pts treated in Ph II Cervical cancer study

✓ Total 55 Cervical pts treated in Ph I/II study

✓ Promising data in multiple solid tumors

## Other Pipeline

✓ Enapotamab vedotin (HuMax-AXL-ADC) expansion

✓ Pts dosed: GEN1029 & GEN3013

✓ Agreement with Immatics

✓ Pre-clin. milestone in DuoBody collab. w/ Novo Nordisk

✓ First pts dosed in Ph I JNJ-64407564 study

✓ JNJ-61186372 data in NSCLC

# Key Events on the Horizon

## Anticipated Near-term Newsflow

### DARZALEX

Phase III MAIA MM efficacy analysis in frontline

Phase III CASSIOPEIA MM efficacy analysis in frontline

Key 2019 readouts: COLUMBA, GRIFFIN, CANDOR

### Ofatumumab

Ph III RMS Data, 2019

Potential regulatory filing soon after data, based on study completion & positive results

### Tisotumab vedotin

New Ph II study starts: ovarian & 1L/2L cervical

Promising solid tumor data

### Other pipeline

Accelerate new programs towards the clinic

Enter new strategic collaborations

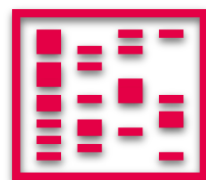
# 2019: Anticipation of a Very Exciting Year



2019 promises to be  
an exciting year

- Aiming for 3 - 5 new INDs
- Read-out of ofatumumab SC data in RMS, followed by possible filing
- Read-out of daratumumab SC data, followed by possible filing
- Data from enapotamab vedotin (HuMax-AXL-ADC) expansion cohorts
- Data from HexaBody-DR5/DR5 trial
- Data from DuoBody-CD3xCD20 trial
- Progress expansive clinical development tisotumab vedotin
- Potential Ph III teprotumumab read out
- Move from 6 to 10 Genmab-owned products evaluated in clinic

# Positioned to Become a Leader in Cancer Immunotherapy



## Substantial existing pipeline

- DARZALEX blockbuster & growing
- Expansive rapid development tisotumab vedotin
- Promising truly differentiated products in early clin. development

## Developing new proprietary next generation Ab technologies

- DuoBody & HexaBody technologies validated
- Novel HexElect antibody platform

## Strategic alliances

- Pipeline expanded with additional next-generation bispecific products
- Supportive of future advancement

## Building world-class team

- Expanding on existing broad expertise
- Building new capabilities

## Well capitalized for strong growth

- Expenses more than paid for by growing revenues marketed products
- Able to invest in new products & next generation technologies

# Q&A



