CORPORATE PRESENTATION

ABOLOGIX, Spin Off Oncology Company from the University of Geneva

February 2025

With the support of

Innosuisse VENTURE KICK FONGIT Kickfund

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INVESTMENT OPPORTUNITY SUMMARY

ABOLOGIX is a spin out from the University of Geneva. The lead asset is a monoclonal antibody, code-named H225, that blocks JAM-C (Junctional Adhesion Molecule C)

Primary indication is JAM-C(+) B-cell lymphomas that become resistant to standard treatments.
Scope to expand the treatment to other types of cancers that are JAM-C(+) including metastasis in solid tumors.
Product sales potential estimated at €200m in 2031 rising to 1.1b by 2043

THIS IS TRULY PERSONALIZED MEDICINE FOR ONCOLOGY PATIENTS THAT ARE DIAGNOSED AS JAM-C(+)

Patents issued in the USA, Canada and most European countries. Valid until at least 2038

With €1m raised to date, ABOLOGIX is seeking to raise seed capital of €2.5m to progress from early to late pre-clinical by mid 2027

JAM-C is a cell adhesion molecule. There has recently been lots of interest in targeting (blocking) cell-adhesion molecules in the treatment of cancer

FOUNDERS

Dr. Ignacio Faus, PhD, MBA

- 20+ years of experience in the pharmaceutical and biotechnology industries (BMS, Ferrer, Palau Pharma, The Sage Group, DRI Capital)

Prof. Beat Imhof, PhD

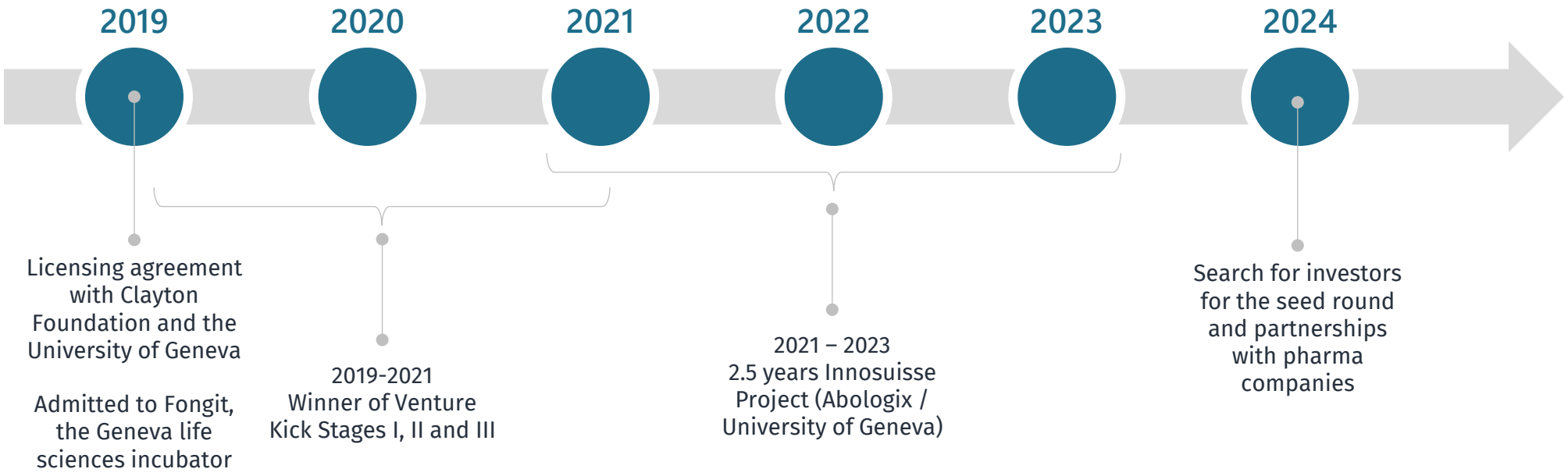
- Professor at the Medical Faculty, University of Geneva. 20+ years researching the role of cell adhesion molecules in cancer

Prof. Thomas Matthes, MD

- Professor at the Haematology Service of the University of Geneva

History of the company

- **Profs. Imhof and Matthes have worked on the biology of cell-adhesion molecule JAM-C for over 20+ years. Part of this academic work has been funded by Clayton Foundation (a non-profit US foundation located in Houston, Texas)**
- **This academic research led to the founding of Abologix in 2018, with the goal of designing novel therapeutical agents that block JAM-C (oncology indications)**



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THE PROBLEM. THE SOLUTION

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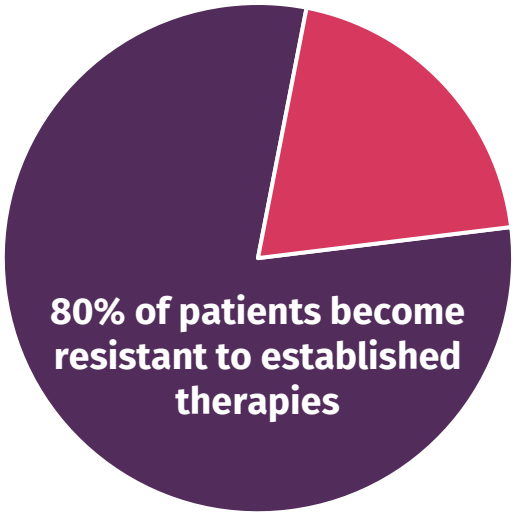
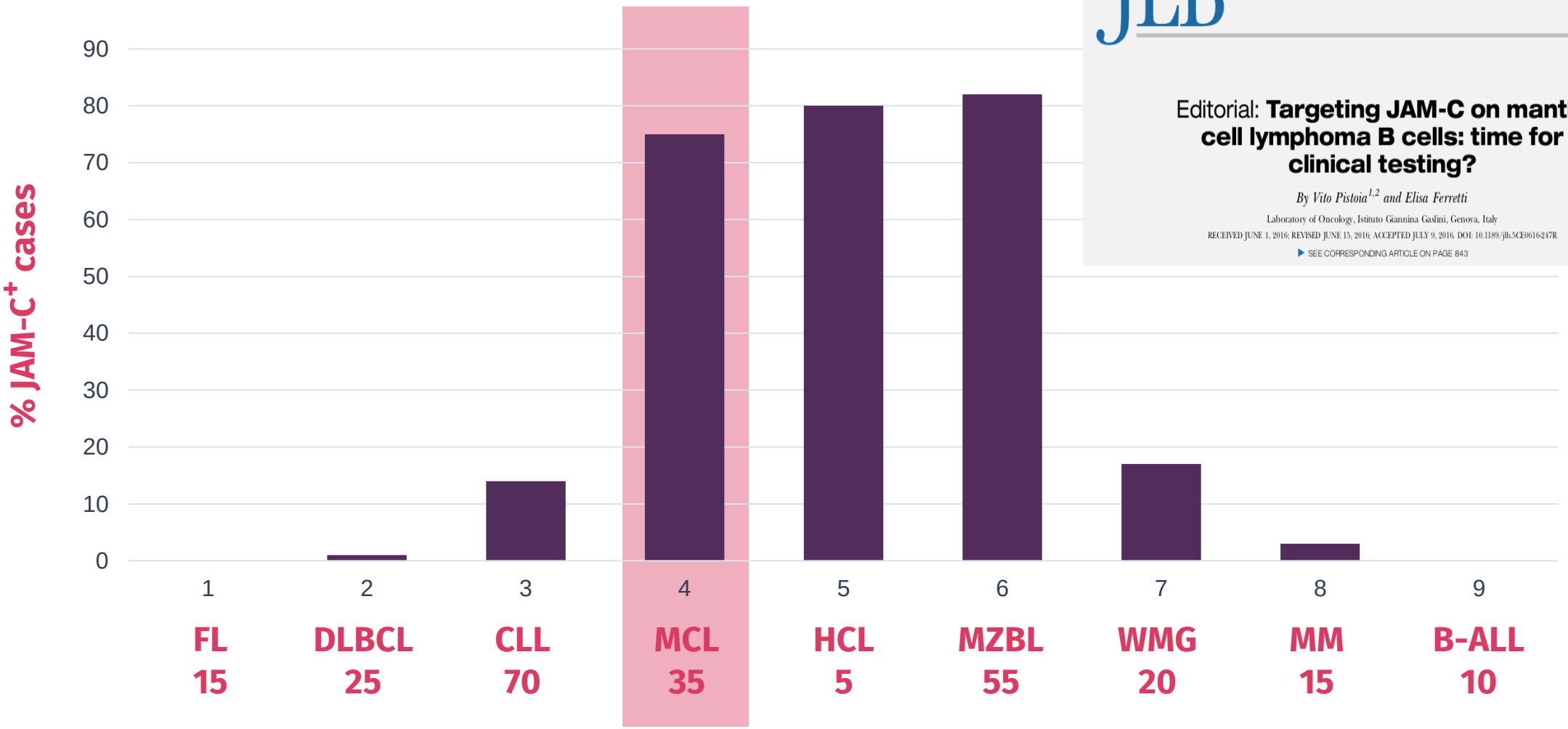
Non Hodgkin Lymhoma (NHL), a major subtype of blood cancer



- 7th most common cancer in the US
- Median age at diagnosis is 67 years
- First line therapy for patients is **rituximab** (Roche). Rituximab resistant patients have few options available and most succumb to their disease
- Market potential for NHL expected to reach USD 30 billion by 2030

JAM-C EXPRESSION IN MALIGNANT B-CELL CANCERS (250 CASES)



JLB

Editorial

Editorial: **Targeting JAM-C on mantle cell lymphoma B cells: time for clinical testing?**

By Vito Pistoia[1,2] and Elisa Ferretti

Laboratory of Oncology, Istituto Giannina Gaslini, Genova, Italy

RECEIVED JUNE 1, 2016; REVISED JUNE 15, 2016; ACCEPTED JULY 9, 2016. DOI: 10.1189/jlb.5CE0616-247R

SEE CORRESPONDING ARTICLE ON PAGE 843

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THIS NOVEL APPROACH FOR THE TREATMENT OF CANCER HAS RECEIVED ATTENTION IN THE SWISS BUSINESS PRESS

BIOTECH

AbologIx

Montant recherché: 2 millions de francs

La startup Abologix, spin-off de l'Université de Genève, développe deux produits, l'un pour le traitement du lymphome (cancer des globules blancs) et l'autre pour le traitement du cancer colorectal. Son premier développement, le H225, est un anticorps monoclonal. Sur des modèles d'animaux, le H225 s'avère efficace dans le traitement d'une forme très agressive du cancer du sang. La startup veut amener son produit en phase clinique. Plusieurs sociétés pharmaceutiques ont montré un intérêt pour ce projet de recherche.

Contact: ignaciofaus@abologix.com

Patrick Aebischer recommande cette startup

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THE MARKET. THE COMPETITION

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alacrita
expertise-based consulting

Clinical positioning assessment of H225 in non-Hodgkin lymphoma (NHL) with initial focus on mantle cell lymphoma (MCL)

A report for Abologix

EXECUTIVE SUMMARY

- H225 is a mechanistically novel antibody targeting JAM-C, which is highly expressed in NHL indications
- Preclinical data strongly support clinical development of H225 in a range of common NHL settings
- Despite the potential multiple applicability of H225 in many NHLs, MCL is considered the most appropriate initial indication

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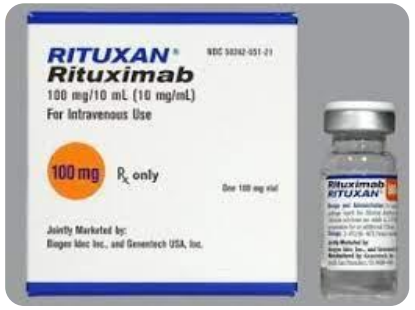
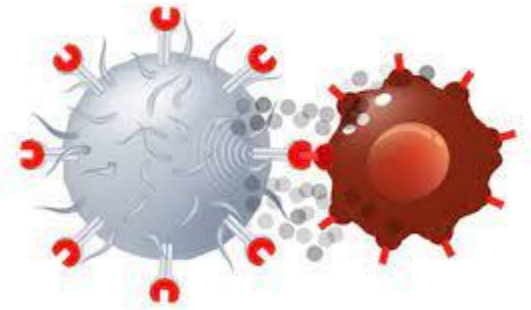
COMMERCIAL POSITIONING OF H225: LYMPHOMA PATIENTS WHO BECOME RESISTANT TO STANDARD TREATMENTS. MANTLE CELL LYMPHOMA (MCL) INDICATION AS PROOF-OF-CONCEPT FOR THE TREATMENT OF JAM-C(+) PATIENTS

First line therapy (rituximab) → **Second line therapy** → **Completely unresponsive to any therapies**



H225

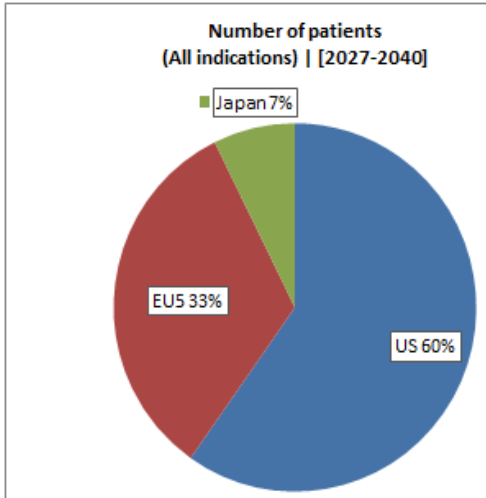
TECARTUS®
(brexucabtagene autoleucel) Suspension for IV infusion

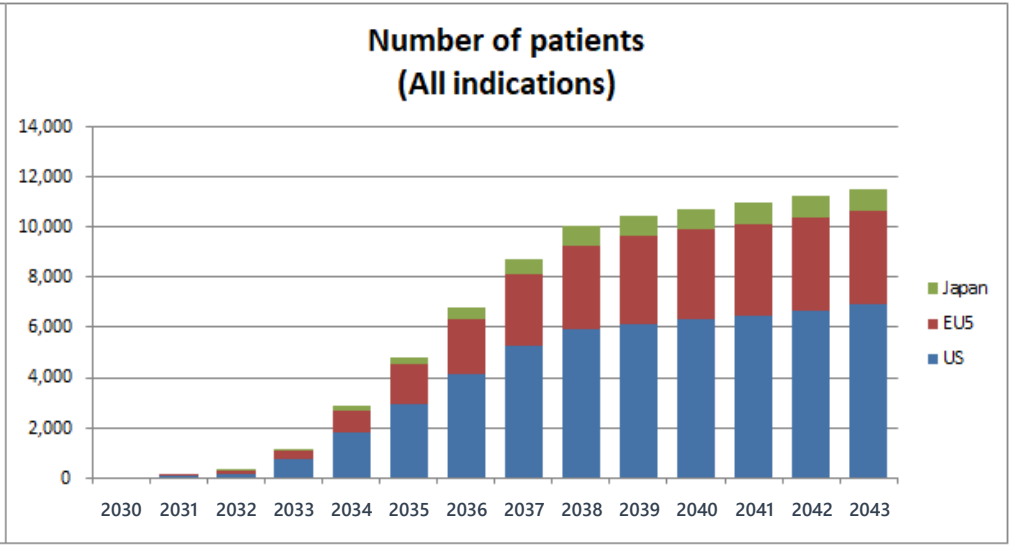


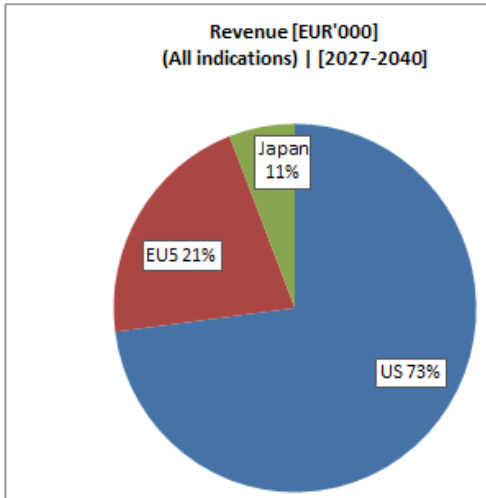
In the case of Mantle Cell Lymphoma we want to position H225 as second-line therapy for those patients that become resistant to rituximab and/or BTK inhibitors, before the last option that these patients have, CAR T cell therapy (TECARTUS)

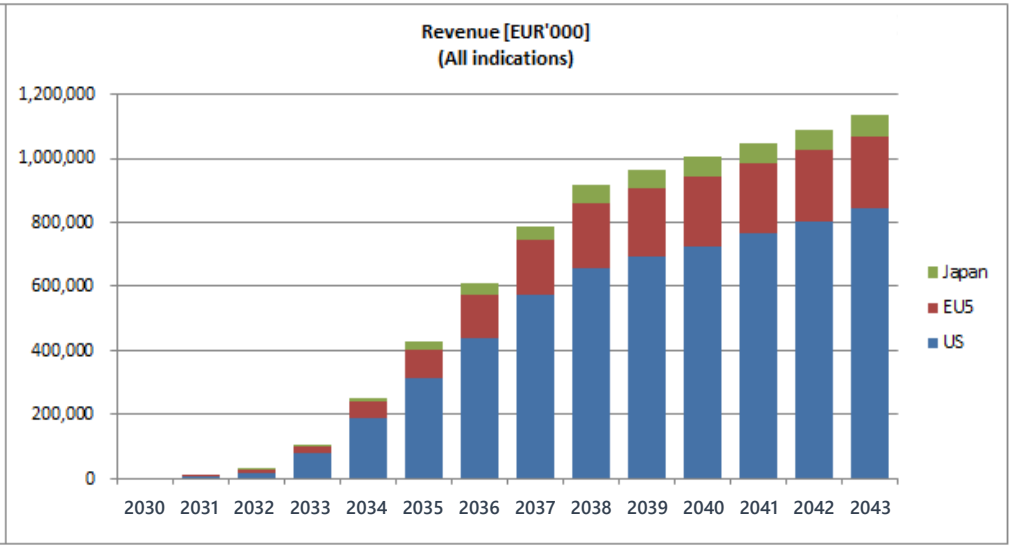
SALES PROJECTIONS IN 7 MAJOR MARKETS (USA, JAPAN, GERMANY, FRANCE, UK, ITALY AND SPAIN)

Market summary - [2027-2040]









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FURTHER POTENTIAL BY ADDRESSING METASTASIS IN SOLID TUMOURS

ELSEVIER

FEBS Letters

journal homepage: www.FEBSLetters.org

The Junctional Adhesion Molecule-B regulates JAM-C-dependent melanoma cell metastasis

Marie-Laure Arcangeli [a,b,c,*], Vincent Frontera [a,b,c], Florence Bardin [a,b,c], Jeanne Thomassin [b], Bruno Chetaille [b], Susanne Adams [d], Ralf H. Adams [d], Michel Aurrand-Lions [a,b,c,*]

The FASEB Journal • Research Communication

Endothelial cell junctional adhesion molecule C plays a key role in the development of tumors in a murine model of ovarian cancer

David A. Leinster,*,[1] Bartomeu Colom,*,[1] James R. Whiteford,* Darren P. Ennis,† Michelle Lockley,† Iain A. McNeish,† Michel Aurrand-Lions,‡ Triantafyllos Chavakis,§ Beat A. Imhof,‖ Frances R. Balkwill,† and Sussan Nourshargh*,[2]

A Novel Function of Junctional Adhesion Molecule-C in Mediating Melanoma Cell Metastasis

Harald F. Langer[1,3], Valeria V. Orlova[1,4], Changping Xie[5], Sunil Kaul[1], Darius Schneider[5], Anke S. Lonsdorf[2,6], Manuela Fahrleitner[3], Eun Young Choi[1], Vanessa Dutoit[5], Manuela Pellegrini[8], Sylvia Grossklaus[9,10], Peter P. Nawroth[5], Gustavo Baretton[11], Sentot Santoso[12], Sam T. Hwang[2,13], Bernd Arnold[7], and Triantafyllos Chavakis[1,5,9,10]

Cooperative Expression of Junctional Adhesion Molecule-C and -B Supports Growth and Invasion of Glioma

MIRNA TENAN,[1] MICHEL AURRAND-LIONS,[2] VALERIE WIDMER,[1] ALESSANDRO ALIMENTI,[3] KARIM BURKHARDT,[4] FRANÇOIS LAZEYRAS,[3] MARIE-CLAUDE BELKOUCH,[1] PHILIPPE HAMMEL,[2] PAUL R.WALKER,[1] MICHEL A. DUCHOSAL,[5] BEAT A. IMHOF,[2] AND PIERRE-YVES DIETRICH[1]*

Junctional Adhesion Molecule-C Promotes Metastatic Potential of HT1080 Human Fibrosarcoma*

Received for publication, September 13, 2006, and in revised form, November 29, 2006 Published, JBC Papers in Press, January 16, 2007, DOI 10.1074/jbc.M608836200

Chiaki Fuse, Yuuki Ishida, Tomoya Hikita, Tomohiro Asai, and Naoto Oku[1]

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Patents issued in the USA, Canada and most European countries. Valid until approx. 2038 (at the minimum)

Humanized monoclonal antibody will be the basis for an additional composition-of-matter patent application (with a new priority date)

Advised by ParkerHighlander PLLC

DEVELOPMENT PLAN.EXIT STRATEGY

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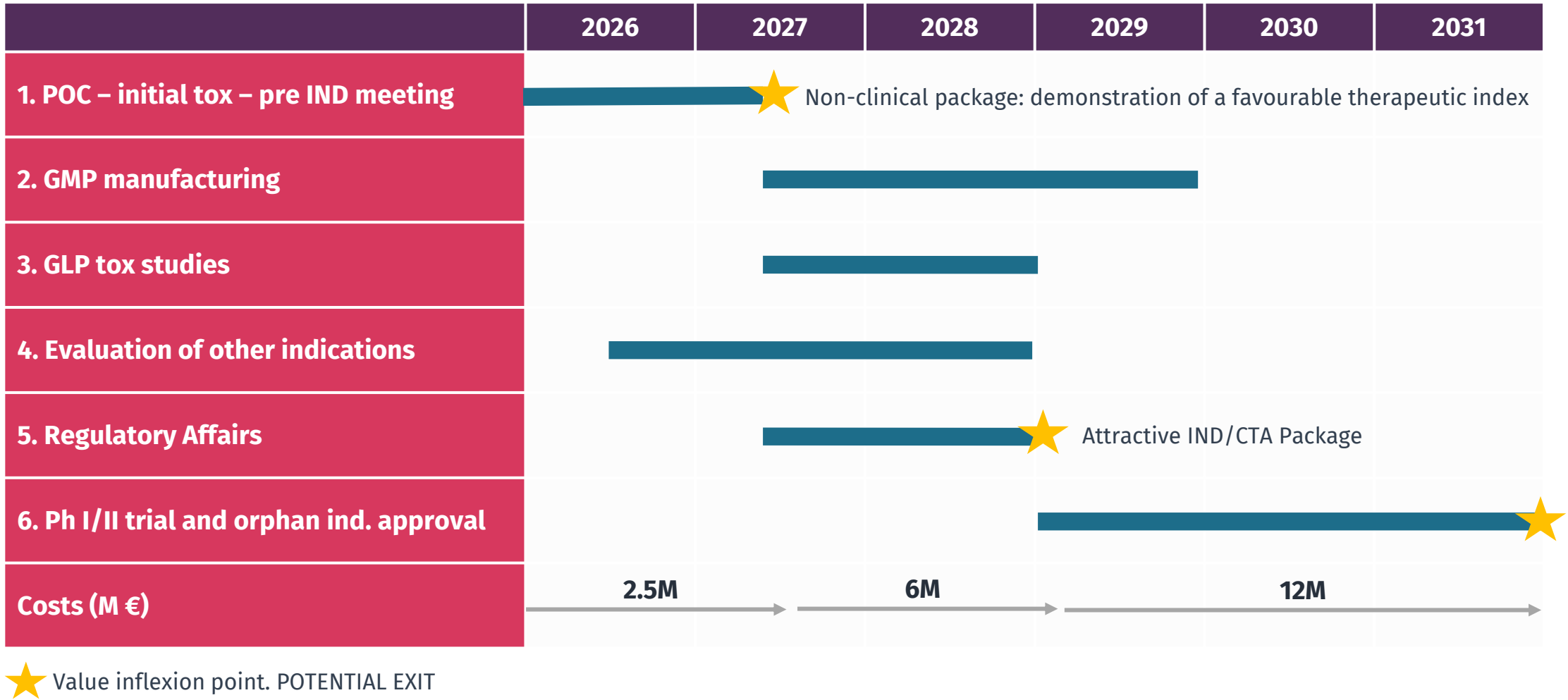
SUMMARY OF DEVELOPMENT PLAN FOR MANTLE CELL LYMPHOMA



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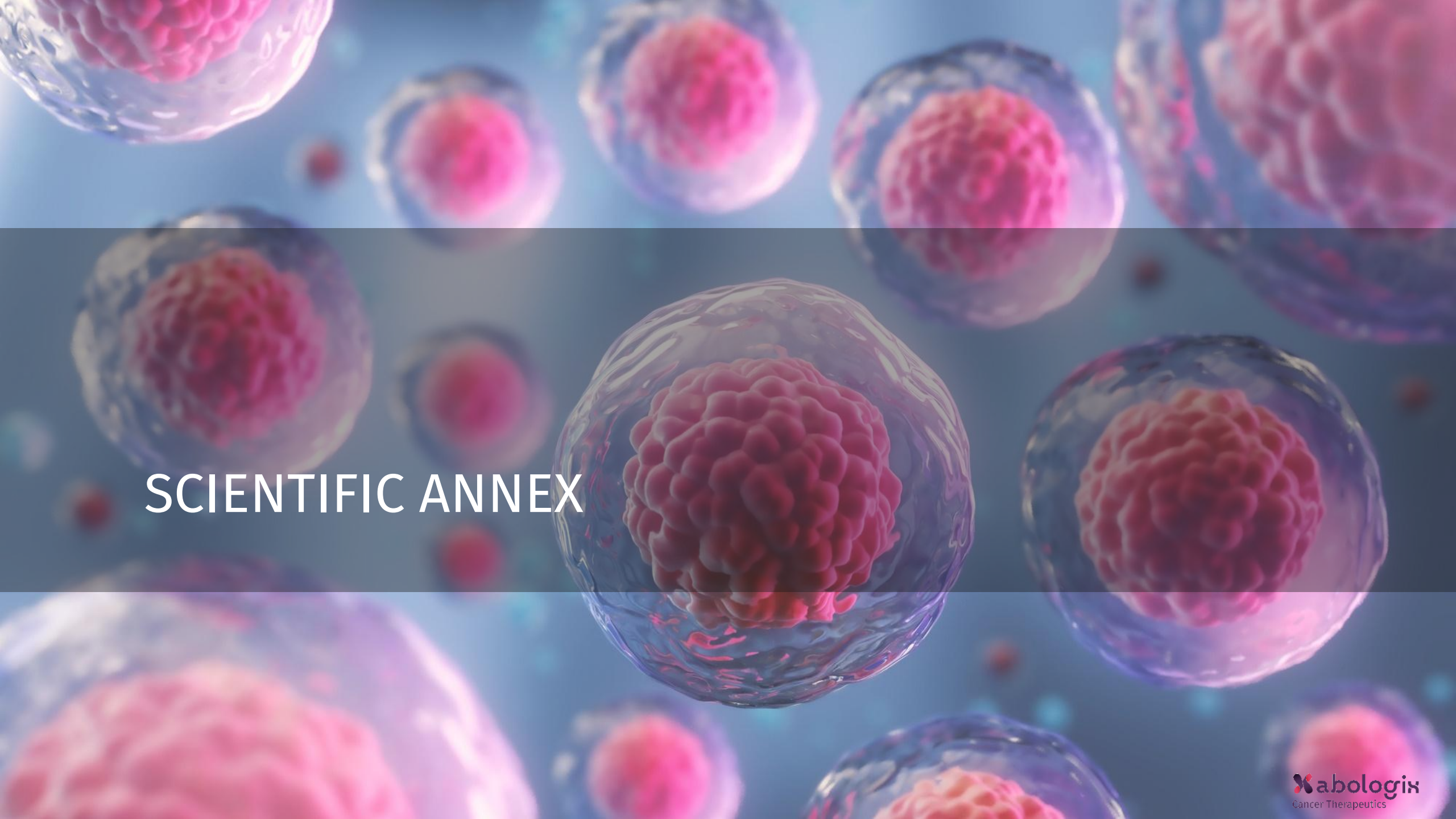
Targeted therapy for JAM-C (+) B-lymphoma patients that are resistant to first line therapy

- **Potential extension into the first line setting**
- **Initial PoC in Mantle Cell Lymphoma (MCL)**
- **Eventual use in JAM-C(+) solid tumours**

Potential revenues in the second-line setting (assuming 30 % of JAM-C(+) patients use it):
PEAK YEAR SALES OF 1.1 BILLION €



SCIENTIFIC ANNEX

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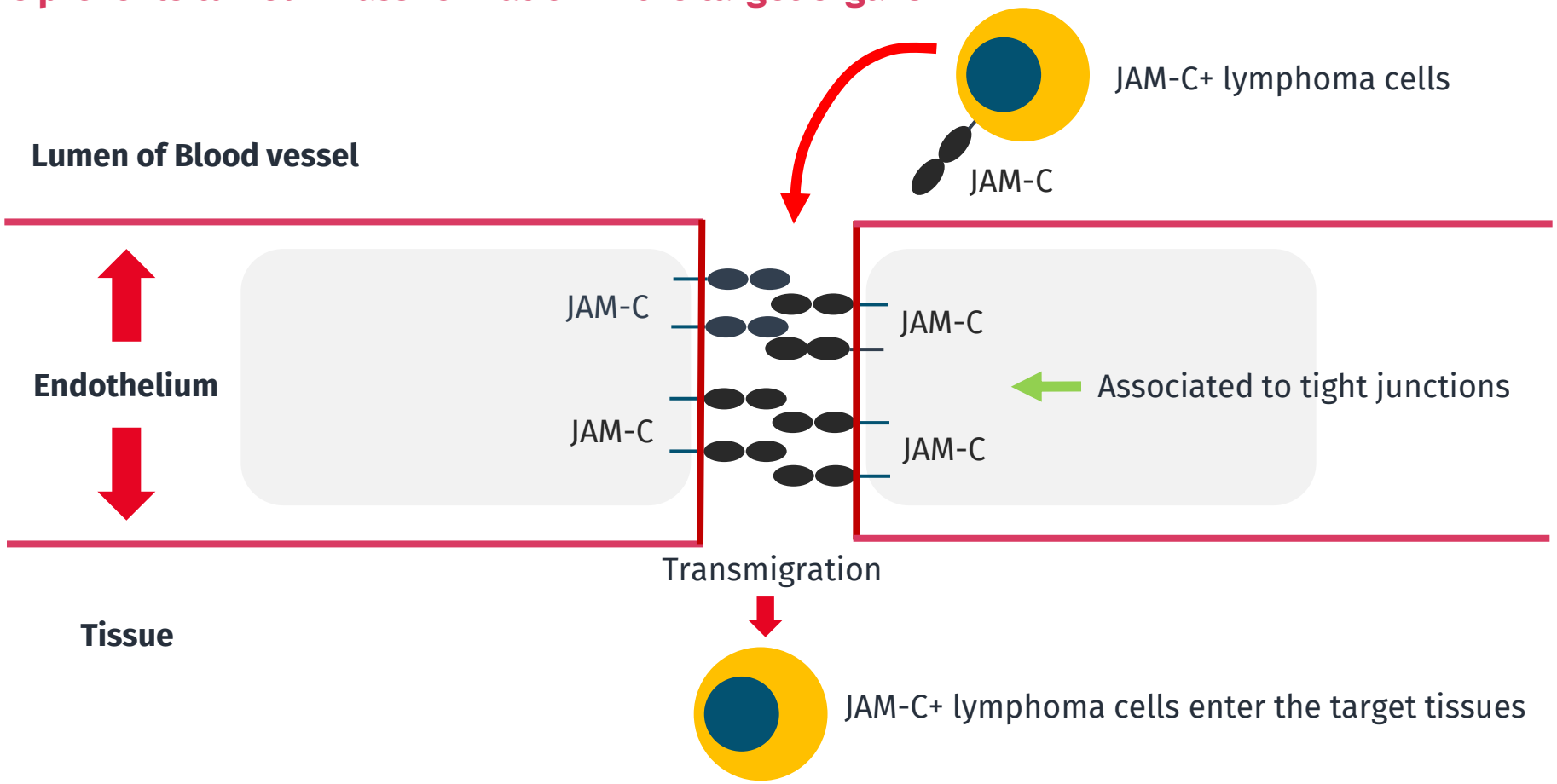
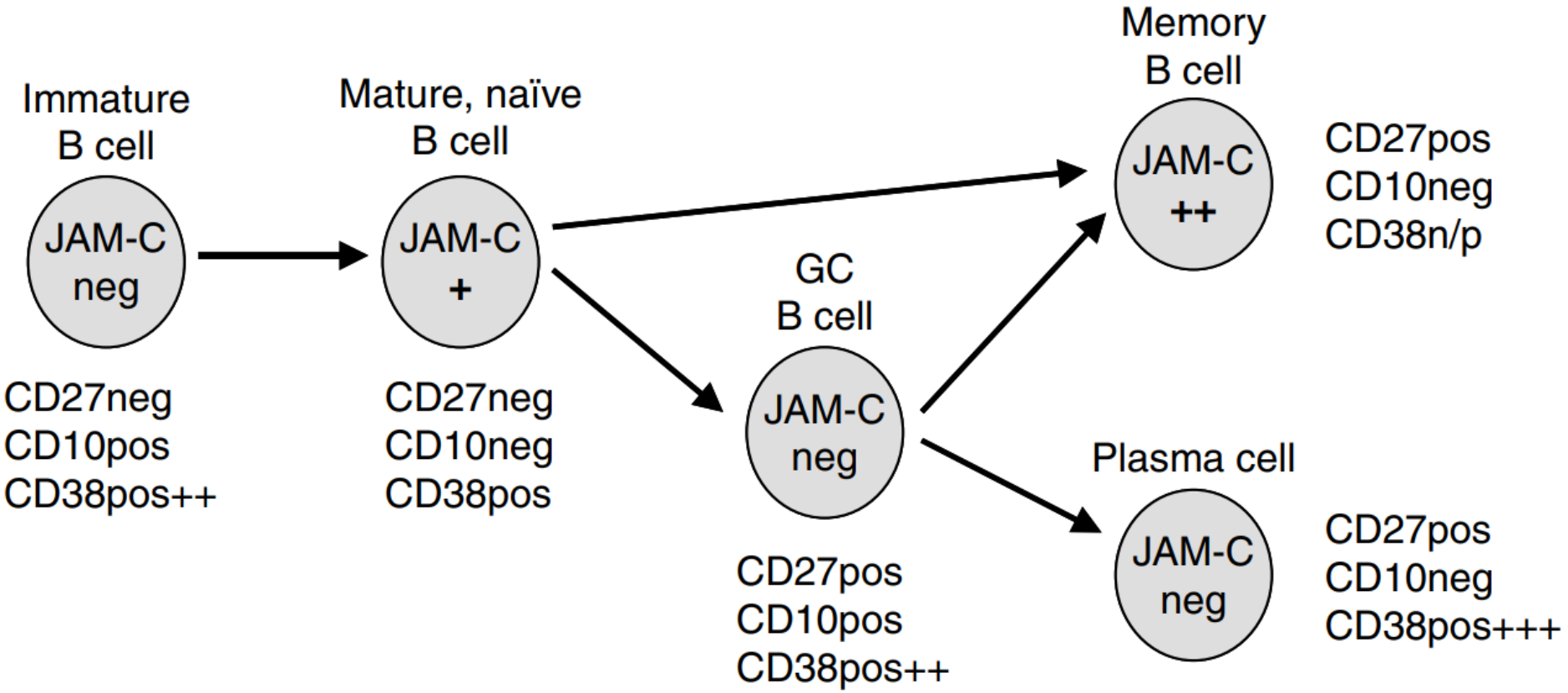
Blocking JAM-C prevents tumour mass formation in the target organs



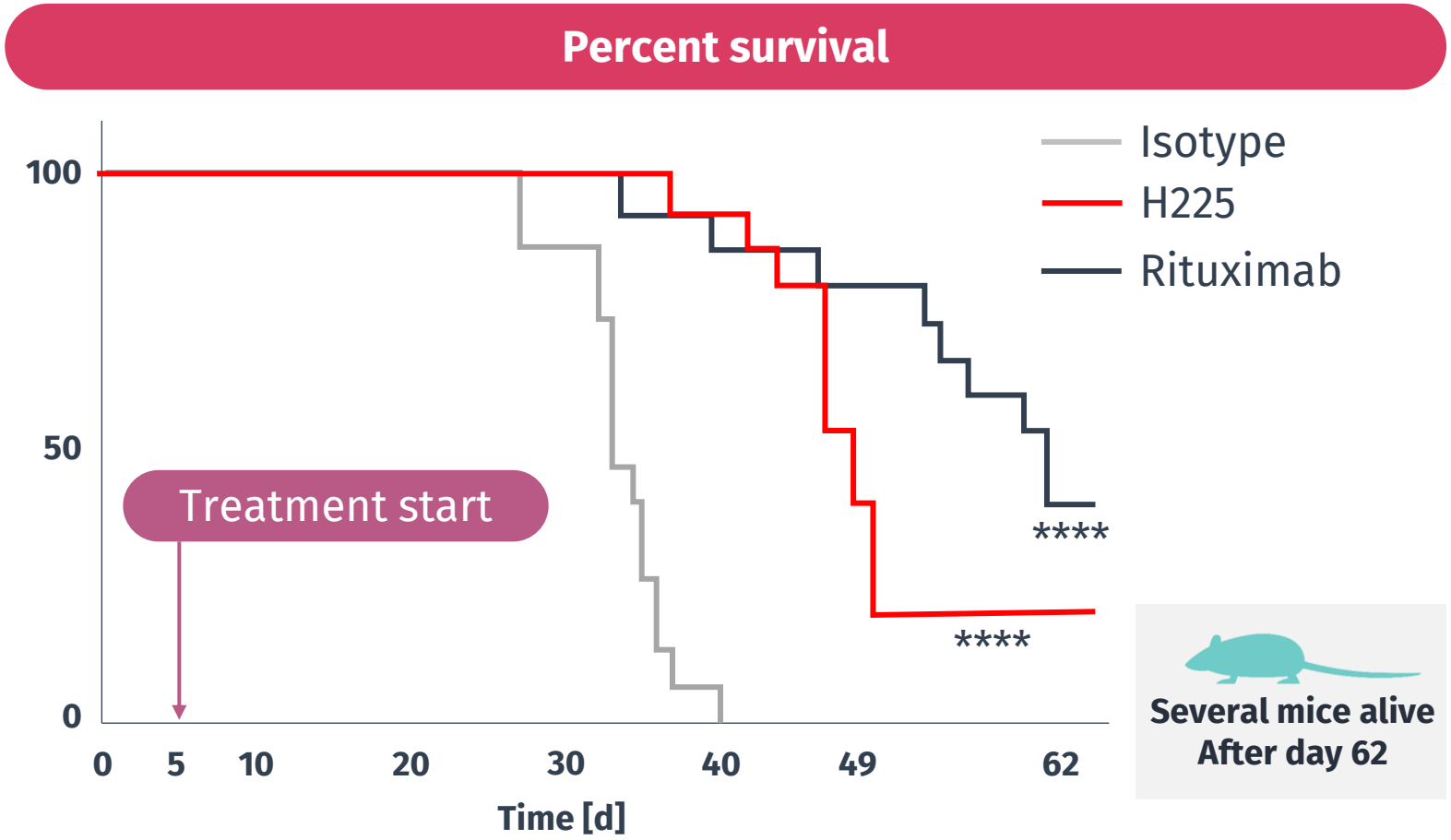
JAM-C(+) lymphoma cells express JAM-C on their surfaces and this allows their transmigration to the target organs (spleen, liver, lymph nodes). Blocking JAM-C blocks this transmigration event

CD20 IS EXPRESSED IN ALL B-CELL LINEAGES, WHILE JAM-C IS NOT EXPRESSED IN THE VERY LARGE AND IMPORTANT POPULATION OF ANTIBODY PRODUCING PLASMA CELLS (MATURE B CELLS)



The fact that JAM-C is not expressed on Germinal Center B-cells and plasma cells is advantageous as far as the therapeutic index is concerned (possibly limits the toxicology risks associated with a depression of humoral immunity)

H225 EXTENDS THE SURVIVAL OF MICE ENGRAFTED WITH HUMAN B-CELL LYMPHOMA



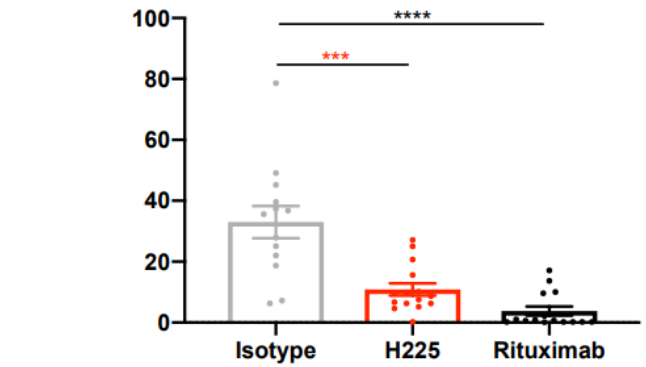
Several mice alive
After day 62

Chimeric H225 (recombinant rat / mouse) vs. rituximab (recombinant mouse / human)

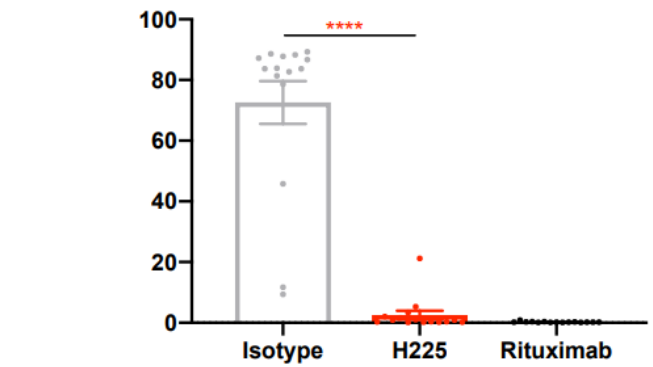
NOD / Scid mice engrafted with Mantle Cell Lymphoma (Jeko-1) cells

B-LYMPHOMA HOMING IN THE TARGET ORGANS

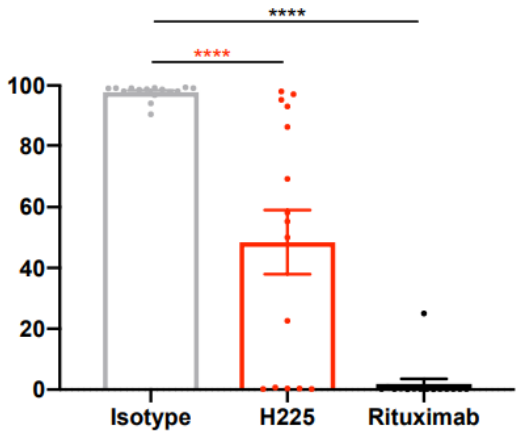
Blood



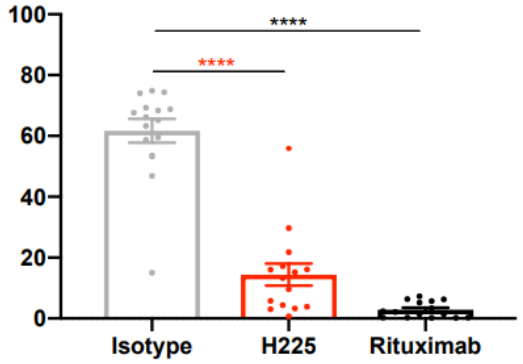
Spleen



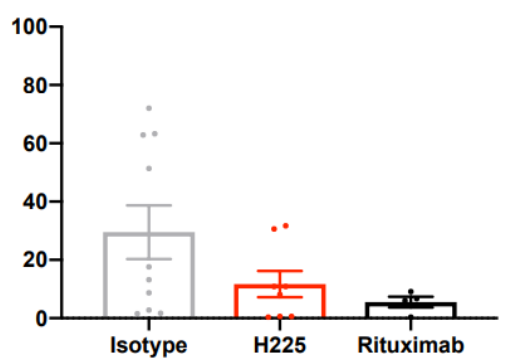
Bone marrow



Liver



Inguinal lymph nodes



Human hematopoietic cell infiltration in the peripheral blood and organs of NOD/Scid mice engrafted with Jeko-1 cells. Ratio (%) between human and mouse CD45+ cells. Results are expressed as mean +/- SEM. Statistical analysis with Mann-Whitney test: *** $p \leq 0.001$, **** $p \leq 0.0001$.

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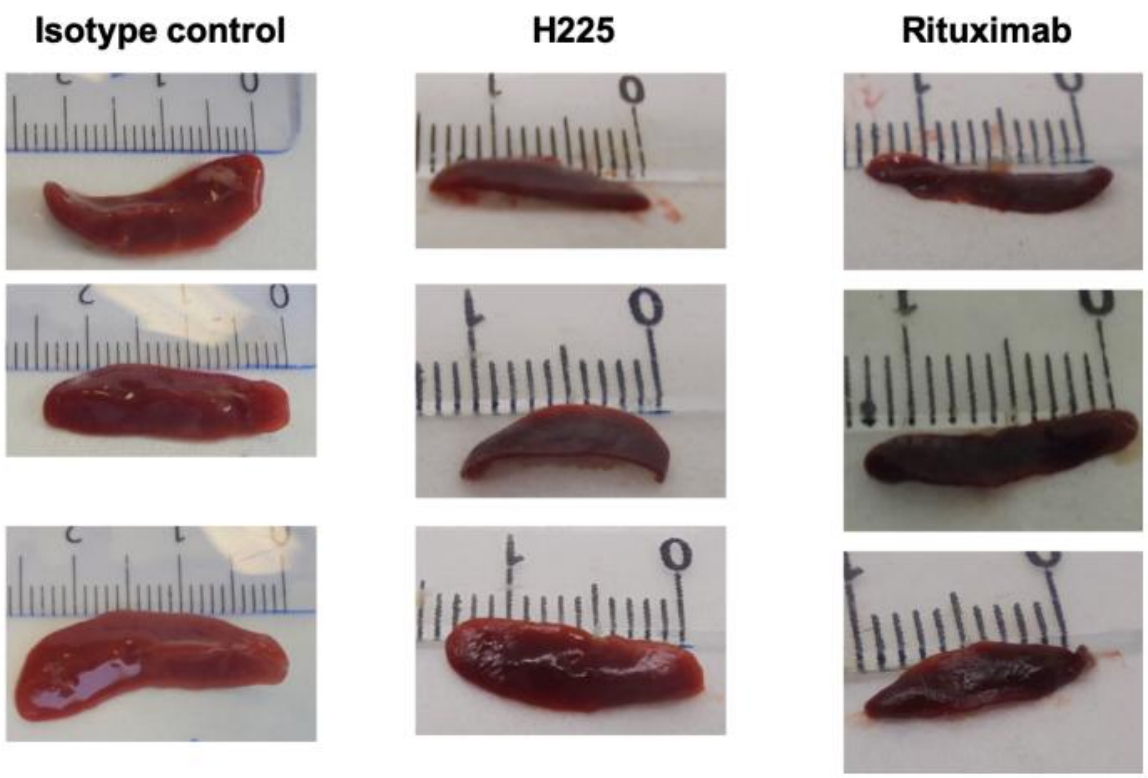
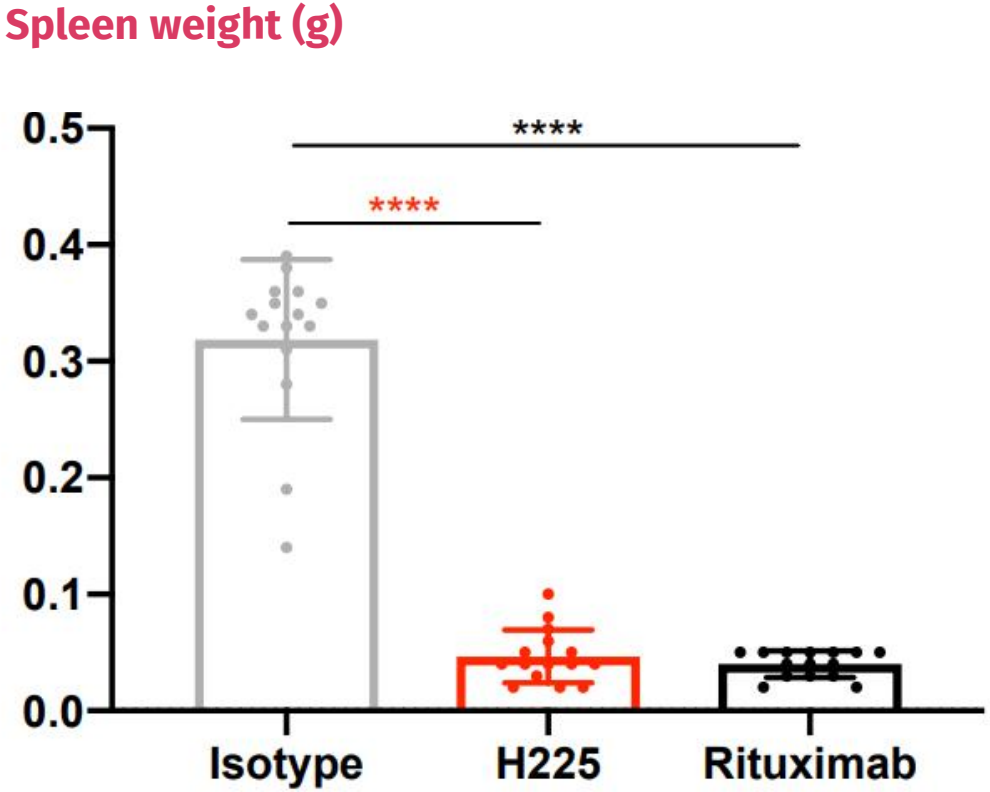
SPLENOMEGALY

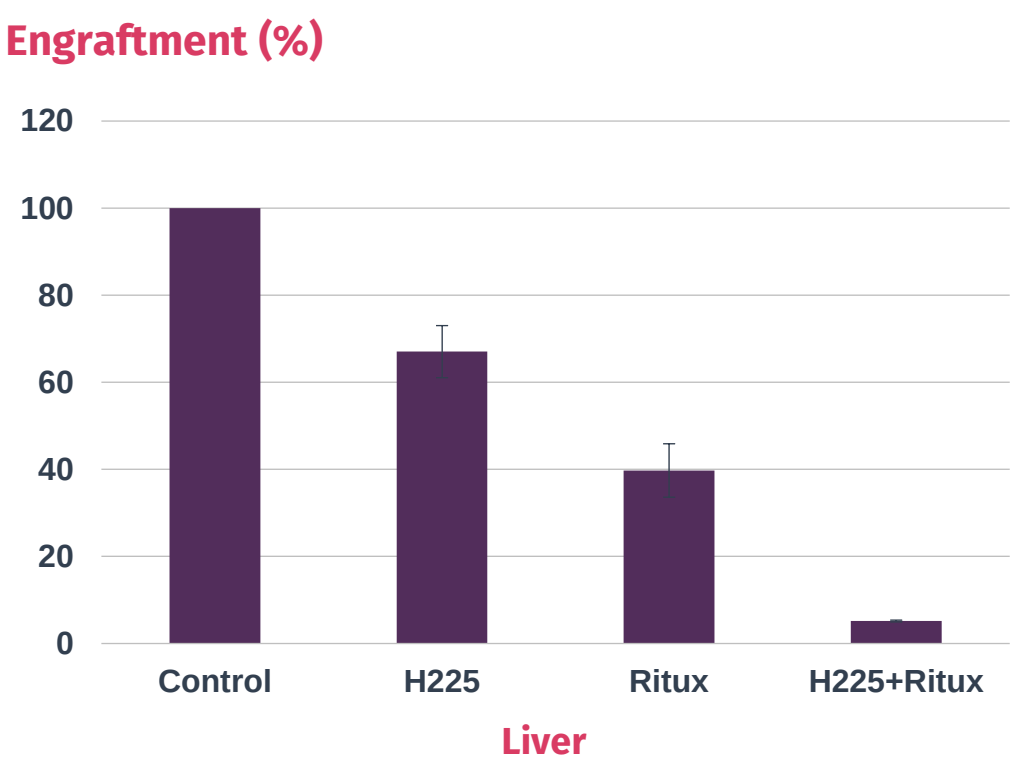




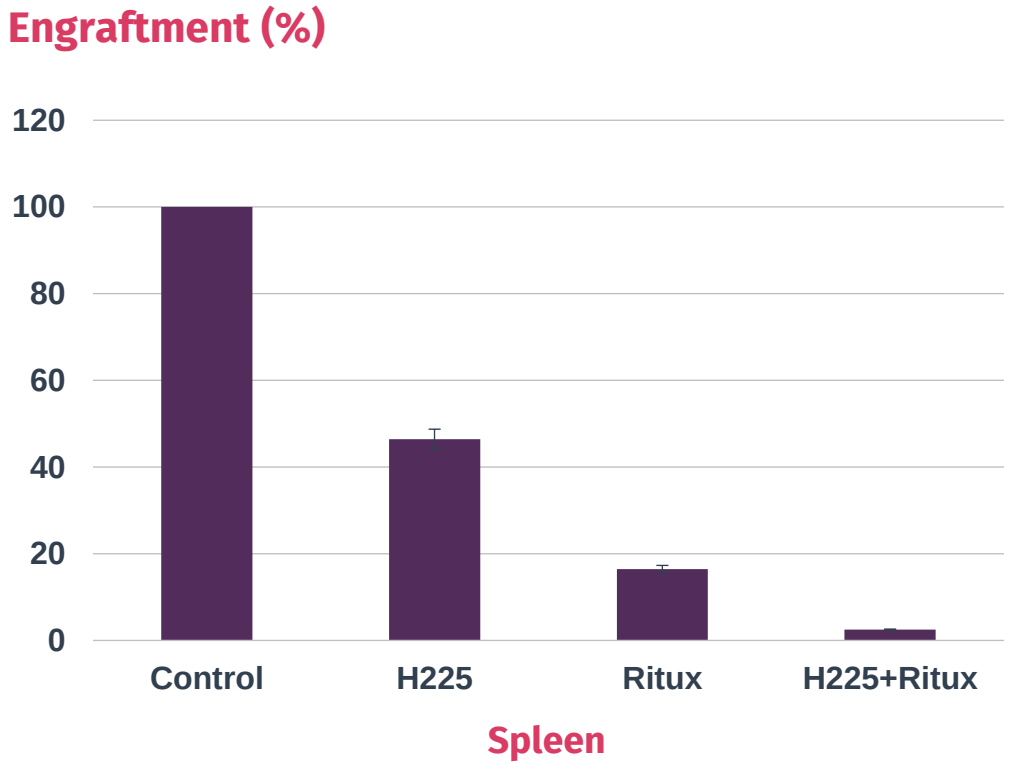
(Left pane) Selected examples of spleens, showing the splenomegaly in the Isotype control group. Ruler unit in cm. (Right pane) Analysis of the weight of spleens at sacrifice. Results are expressed as mean +/- SEM. Statistical analysis with Mann-Whitney test: *** $p \leq 0.001$, **** $p \leq 0.0001$.

THE COMBINATION OF H225 AND RITUXIMAB (STANDARD OF CARE) SHOWS SYNERGISTIC EFFECTS

Rituximab used at a suboptimal concentration



Liver

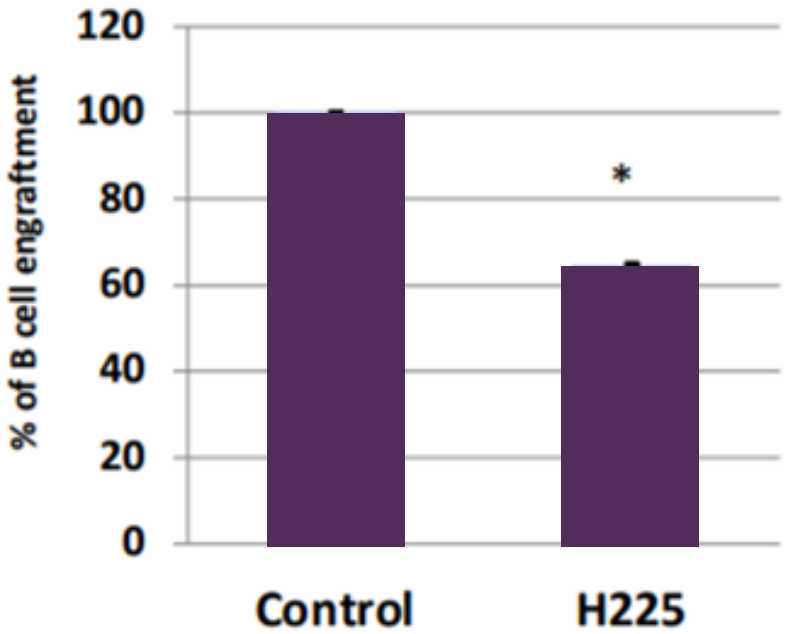


Spleen

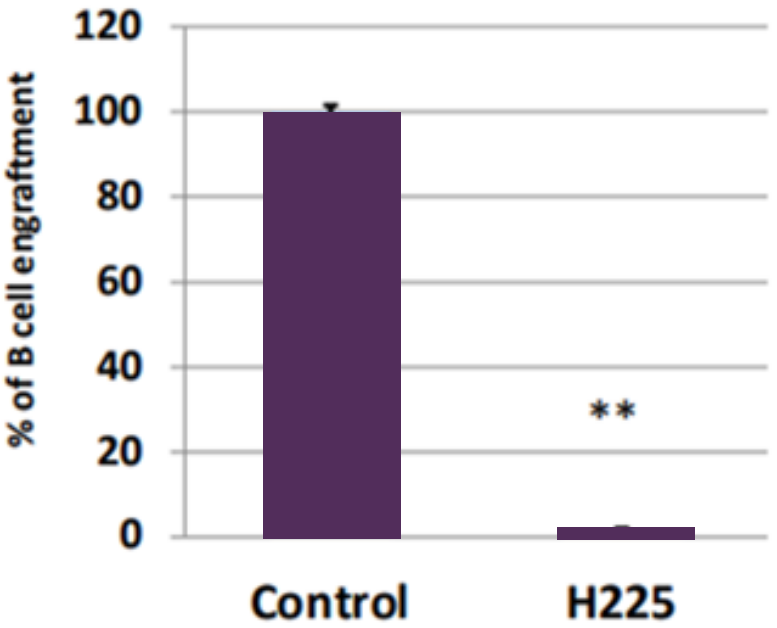
Rituximab 20 µg/mouse
H225: 50 µg/mouse

IN VIVO ENGRAFTMENT OF PRIMARY CELLS FROM A RITUXIMAB RESISTANT MANTLE CELL LYMPHOMA PATIENT (NSG MICE)

BM



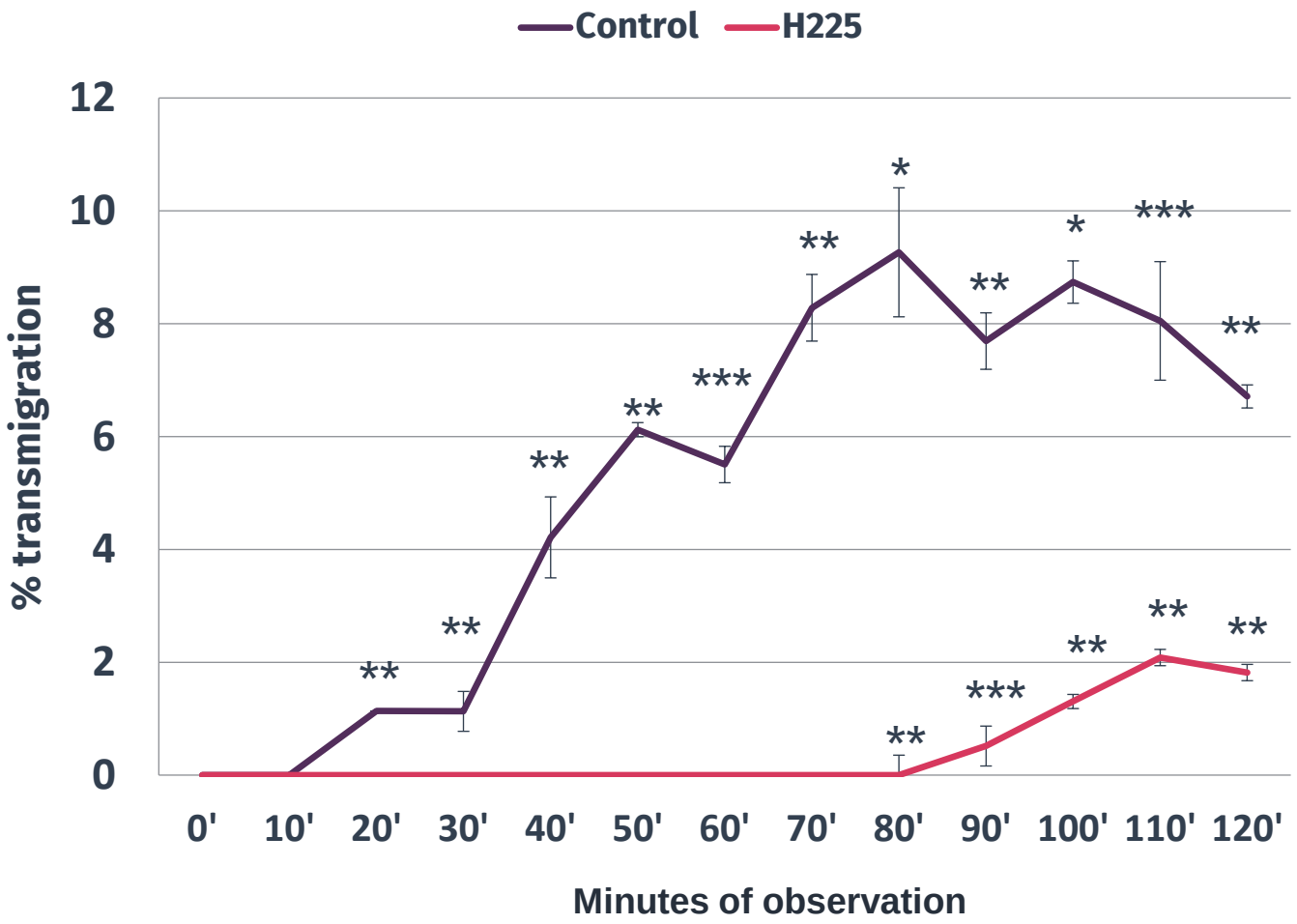
Spleen



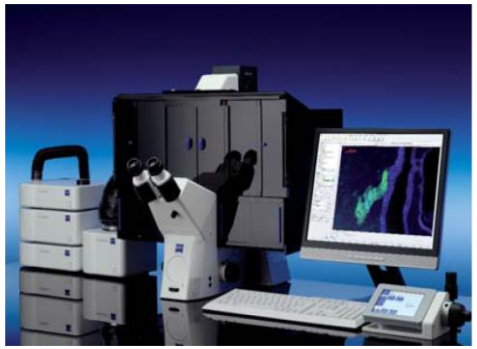
Primary cells from a rituximab resistant MCL patient were engrafted in NSG mice. Treatment with either H225 or a control antibody 2x per week. Animals sacrificed on Day 42

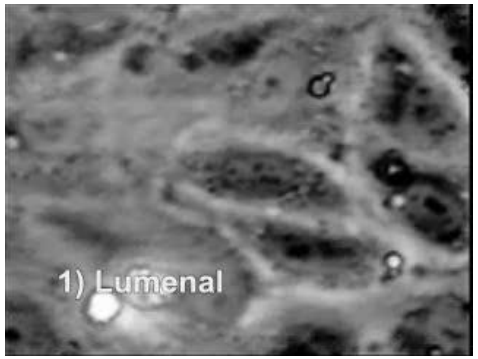
H225 reduces Jeko-1 cell Transendothelial cell migration in vitro

Blocking transmigration



MESENFLOW TECHNOLOGIES





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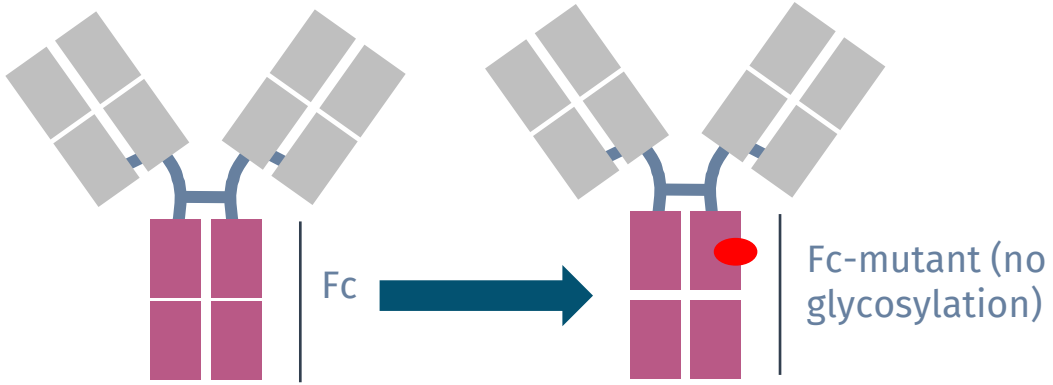
TESTING RECOMBINANT H225 ANTI JAM-C ANTIBODIES ON LYMPHOMA ENGRAFTMENT

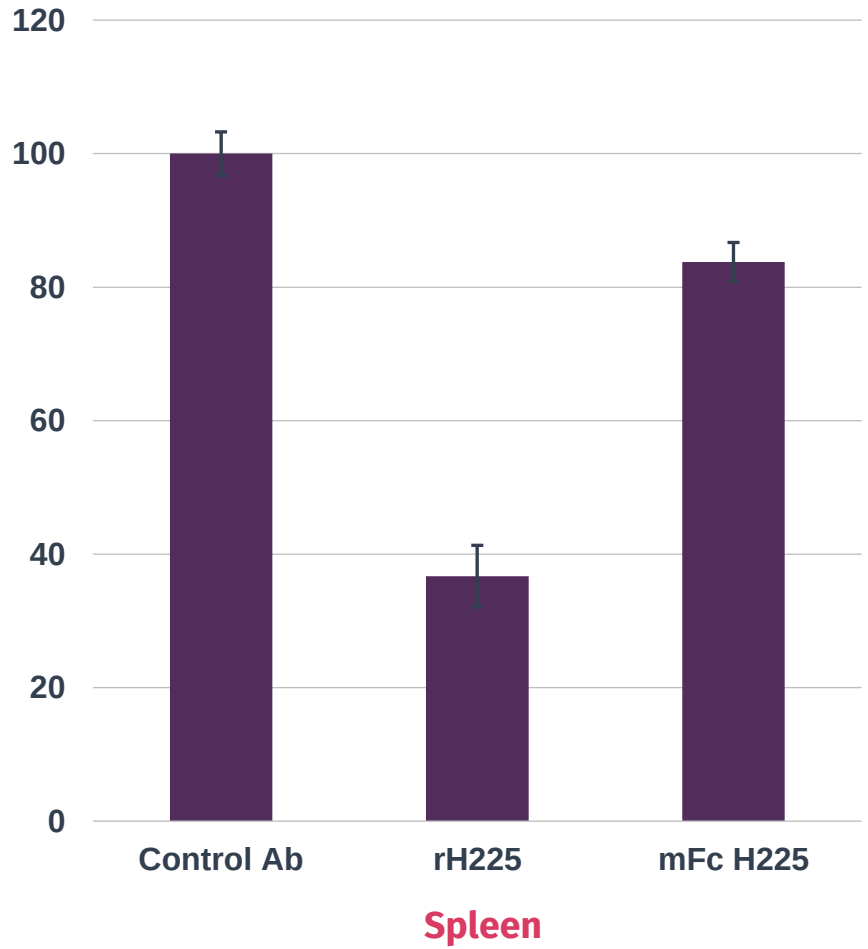
Antibody-dependent cell-mediated cytotoxicity (ADCC)



Rec225, mouse Fc, IgG2a

Rec225, mutated mouse Fc, IgG 2a

Mutated Fc has marginal effect ..... ADCC



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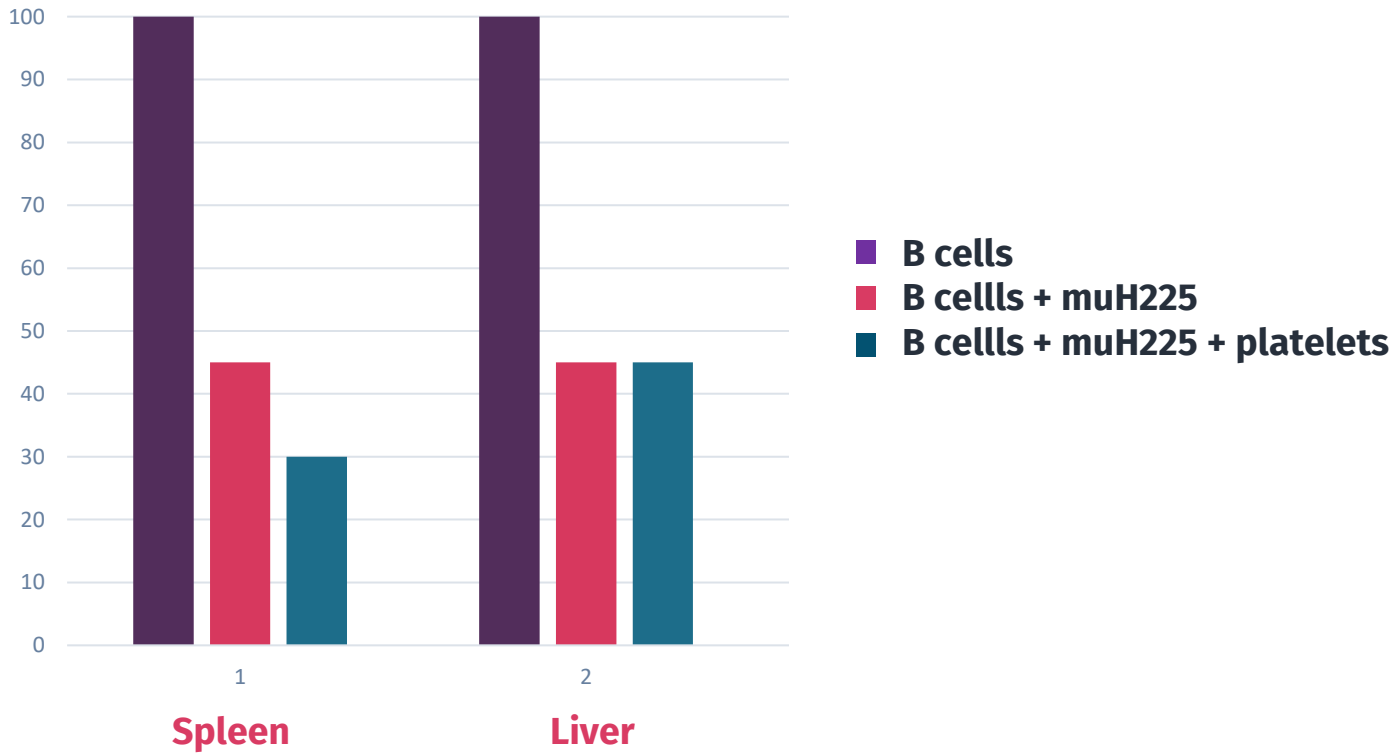
Injection of human platelets into mice does not affect blocking of B cell homing by anti JAM-C antibodies

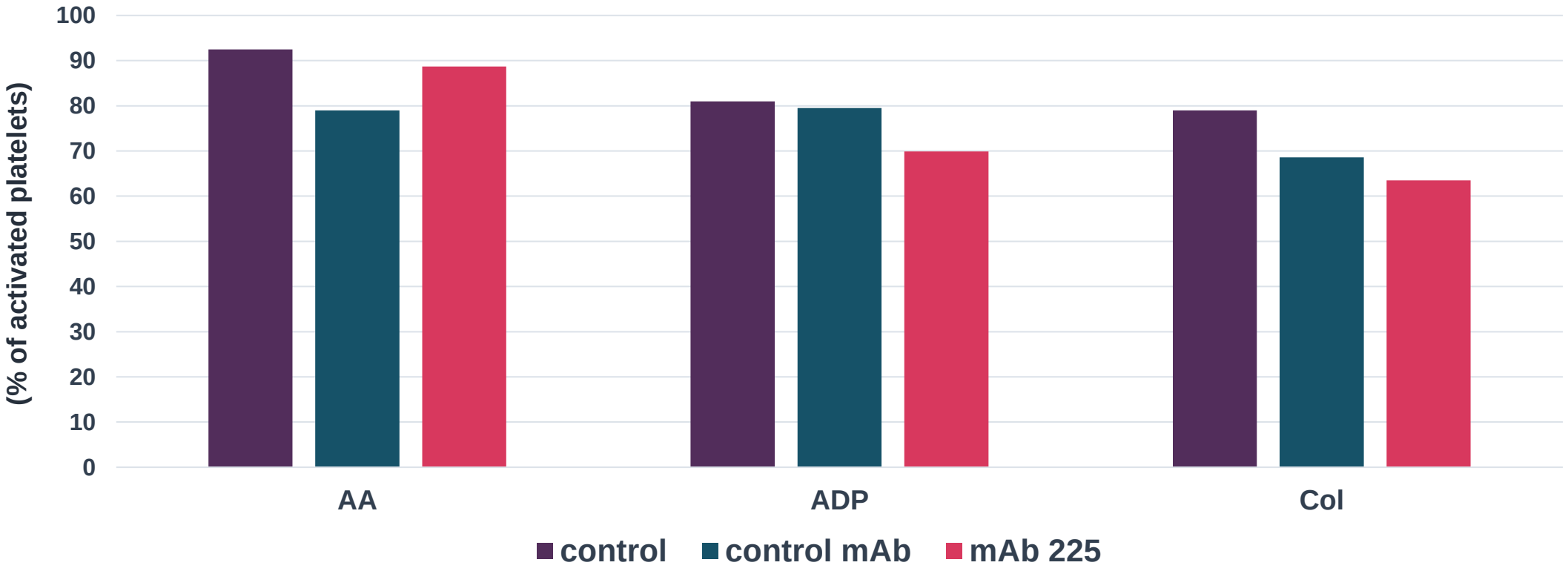
% compared to B cell controls



SAFETY: ANTI JAM-C ANTIBODIES HAVE NO EFFECT ON PLATELET AGGREGATION

Induction of Aggregation with Platelet Activators

AA: Arachidonic Acid
ADP: Adenosine 5'-Diphosphate
Col: Collagen



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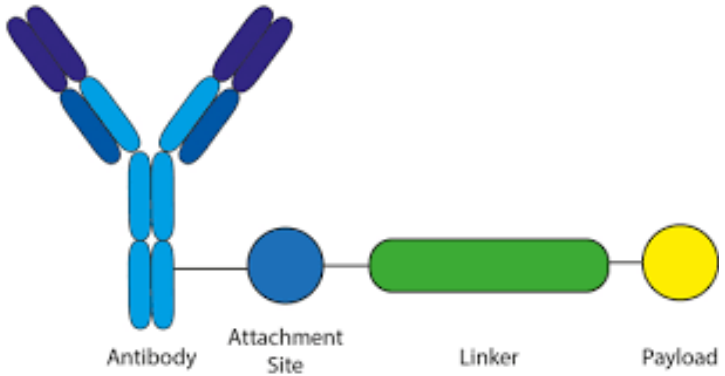
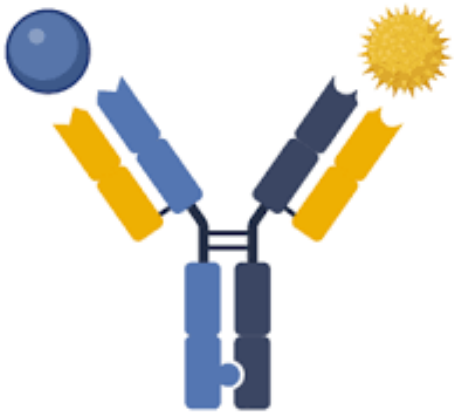
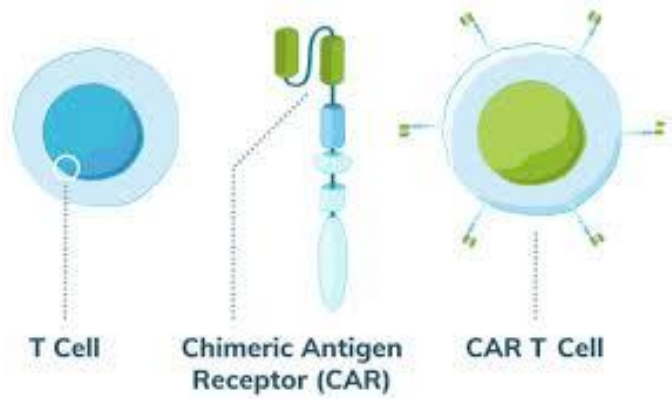
POSSIBLE WAYS TO MOVE FORWARD BEYOND A NAKED ANTIBODY



Drug antibody conjugates

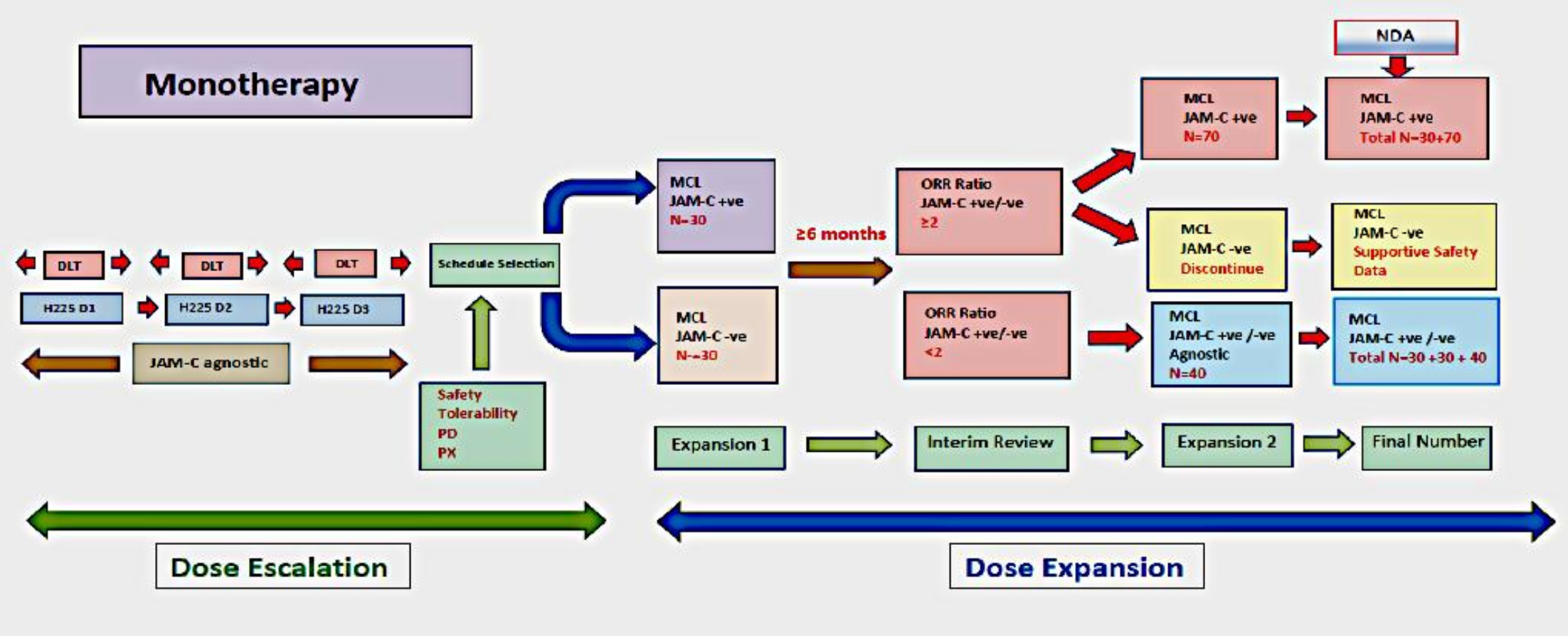


Bi-specific antibodies (anti-CD19 and anti-JAM-C for example)

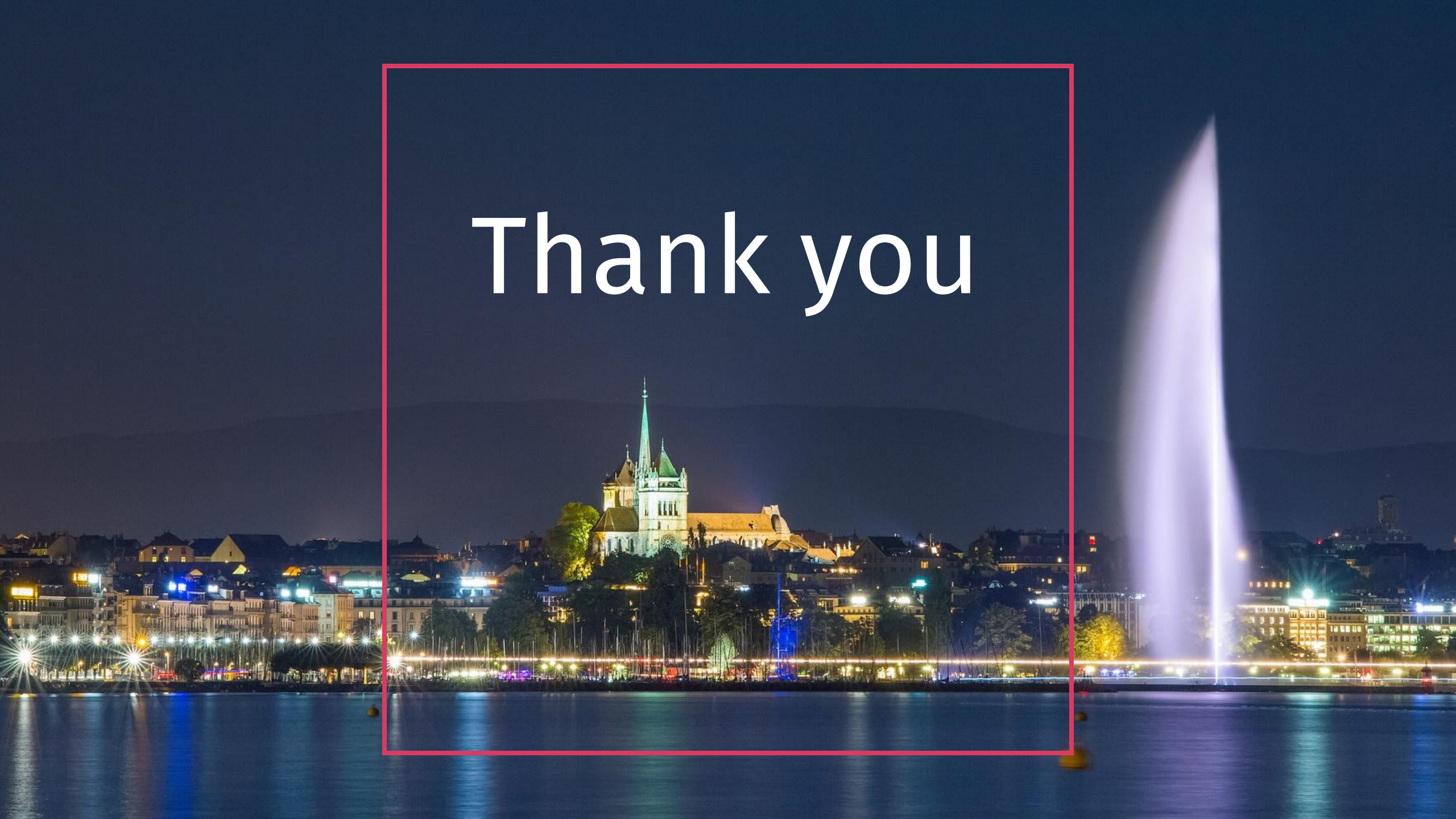


CAR-T cells

Clinical positioning assessment



The initial clinical development of H225 will be: (1) in the second line context, for patients with a short duration of initial response, with subsequent migration into the front-line setting and (2) single agent H225 (later to be expanded into H225+rituximab combination)


Thank you