ER Congressi

New bispecifics and … trispecifics

4th meeting on
Innovative immunotherapies for Lymphoid Malignancies
Milano, Rosa Grand Hotel, January 22-23, 2026

Catherine Thieblemont
Paris, France

Supported by Lymphoma Australia

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Novartis						X	
Kyte/Gilead			x			X	
Roche			x			X	
Amgen						X	
Takeda						X	
Incyte						X	
Cellectis			x			X	
Bayer						X	
Abbvie						x	

Agenda

- **From monospecific to bispecific and trispecific antibodies : structures and mechanisms of action**
- **Trispecific antibodies**
- **Immune-interfacing multispecific bispecific antibodies**

>> Goal of the presentation

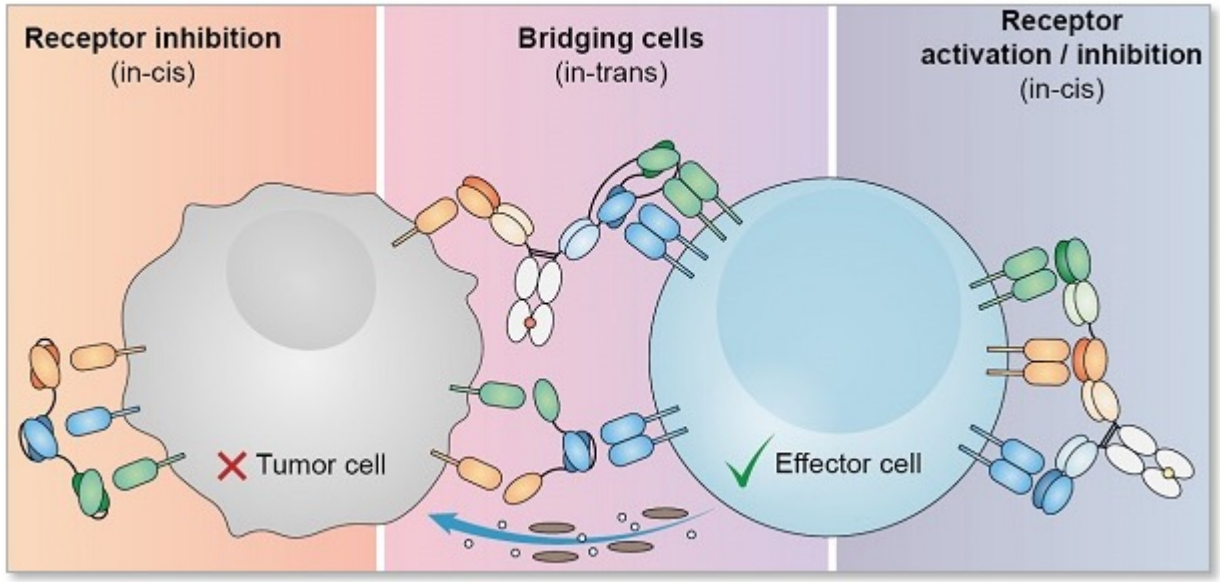
To provide an overview of the development of multispecific antibodies

These new antibodies are offering new therapeutic strategies, especially in hematologic malignancies where immune evasion is a key challenge

Evolution of antibody engineering: from IgG → bispecific → trispecific Abs

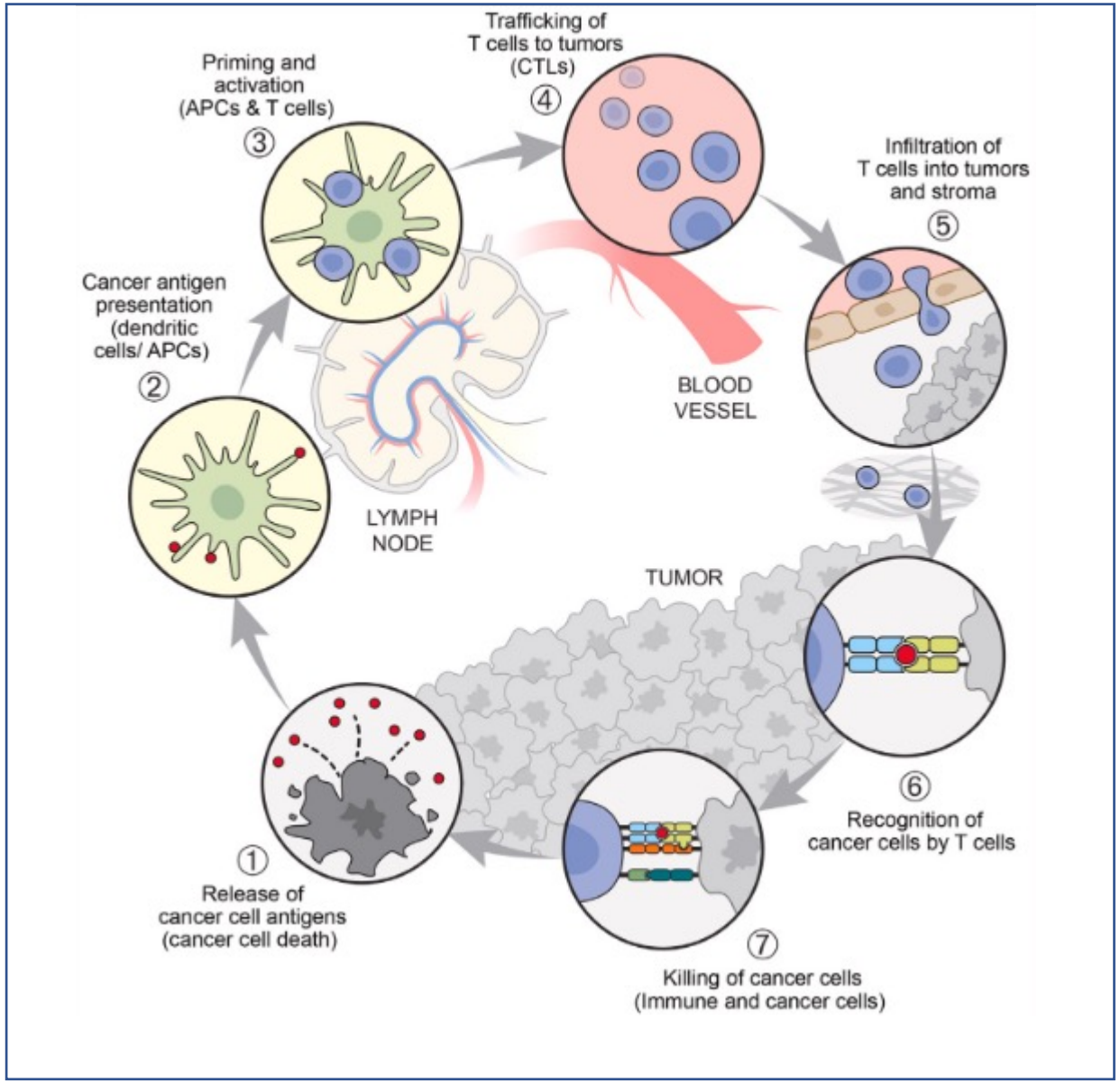
- **Monospecific**: One antigen-binding site (e.g., rituximab, targeting CD20)
- **Bispecific**: Two distinct antigen-binding sites (e.g., blinatumomab: CD3 x CD19)
- **Trispecific**: Three distinct binding specificities

Bridging the cells together tumor cells and effector cells



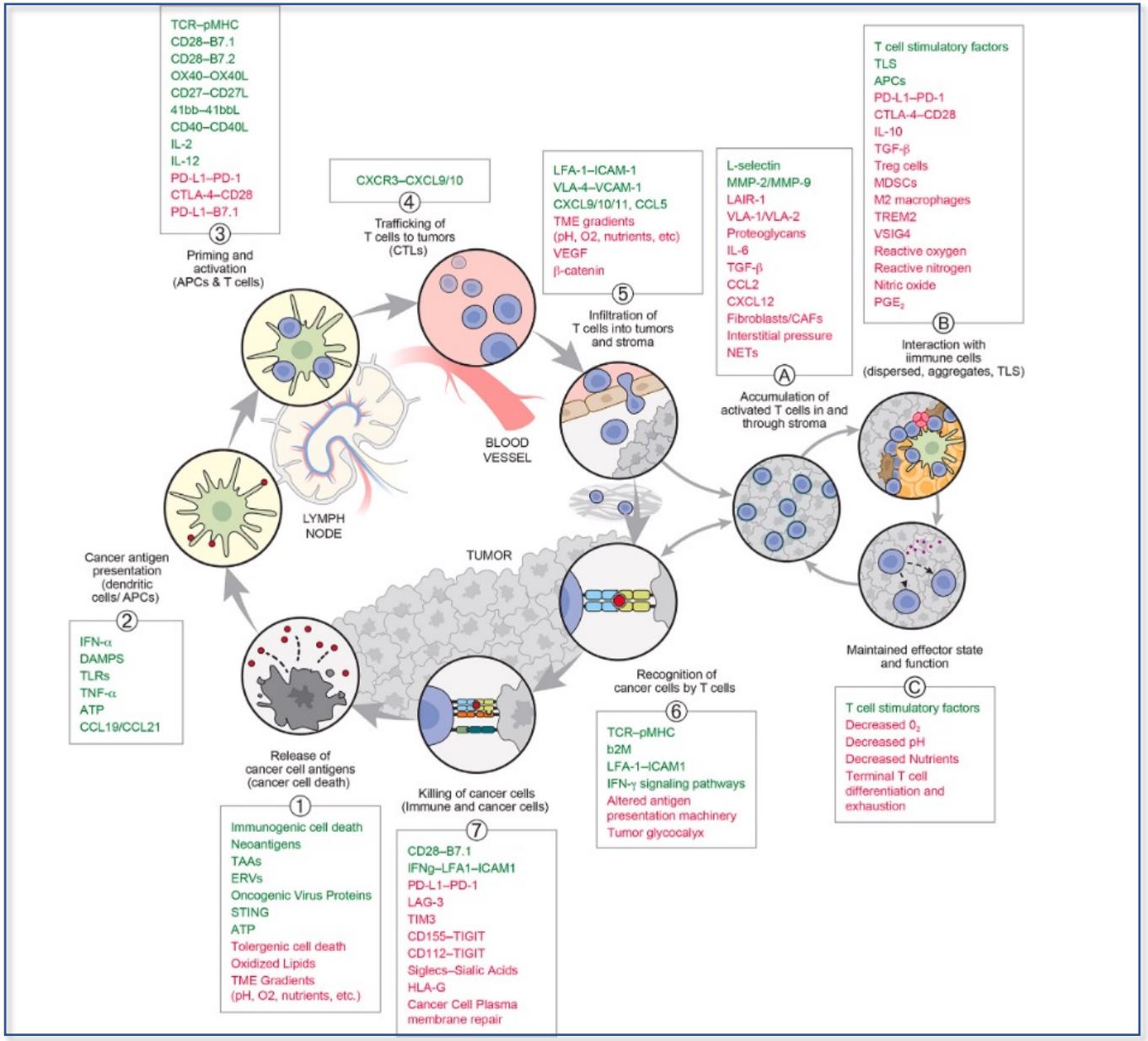
Tapia-Galisteo A et al. J Hemato oncol, 2023

The cancer-immunity cycle in 2013 : T-cells are mediators of cancer cell killing



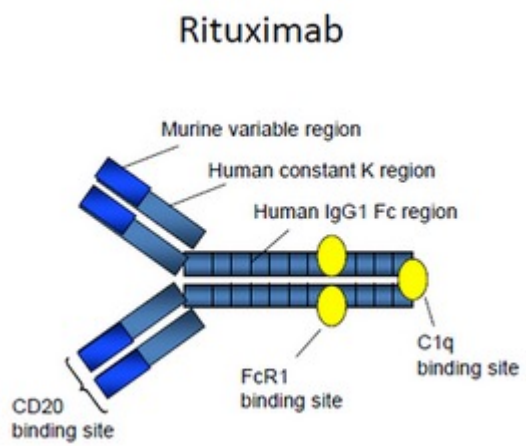
Ira Mellman I, Chen DS, Powles T, Turley FJ , Immunity, 2023;56:2188-2199

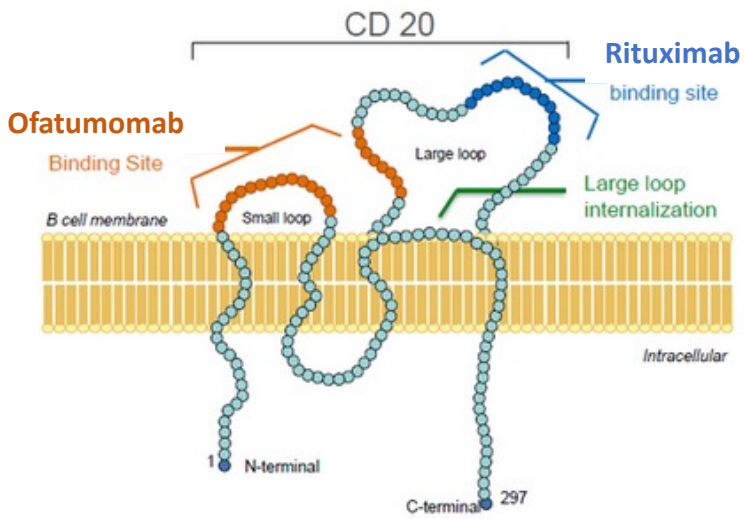
10 years later, in 2023, the cancer-immunity cycle : cell-cell communication and multiple stimulatory and inhibitory factors idendified



Ira Mellman I, Chen DS, Powles T, Turley FJ , Immunity, 2023;56:2188-2199

Monoclonal antibody binds to one epitope/antigen, e.g. CD20





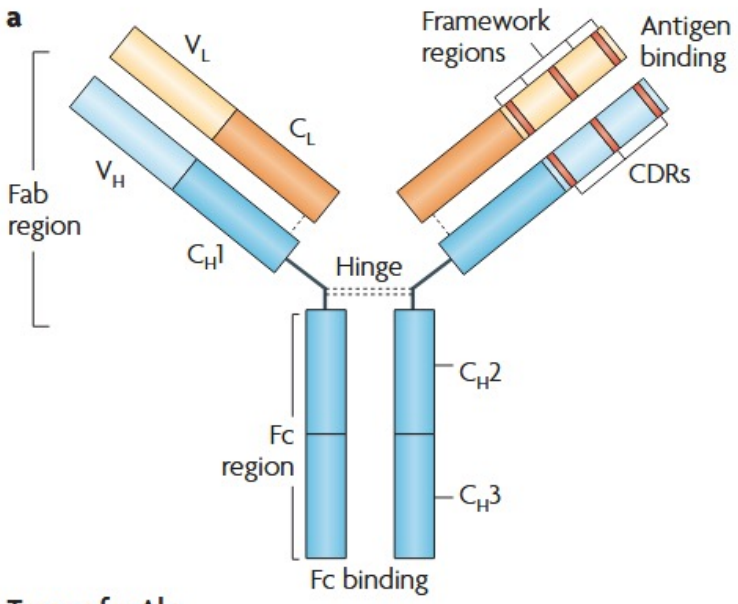
Response rate :
Rituximab monotherapy –first report
ORR : 31% in R/R DLBCL[1]

1. Maloney DG, Liles TM, Czerwinski DK, et al. Phase I clinical trial using escalating single-dose infusion of chimeric anti-CD20 monoclonal antibody (Idec-C2B8) in patients with recurrent B-cell lymphoma. Blood 1994;84(8):2457–2466

Target	Monoclonal antibodies developed in NHL
CD20	Rituximab Obitunuzumab Ofatumomab
CD19	Tafasitamab
CD79B	Polatuzumab vedotin (ADC)
CD30	Brentuximab vedotin (ADC)

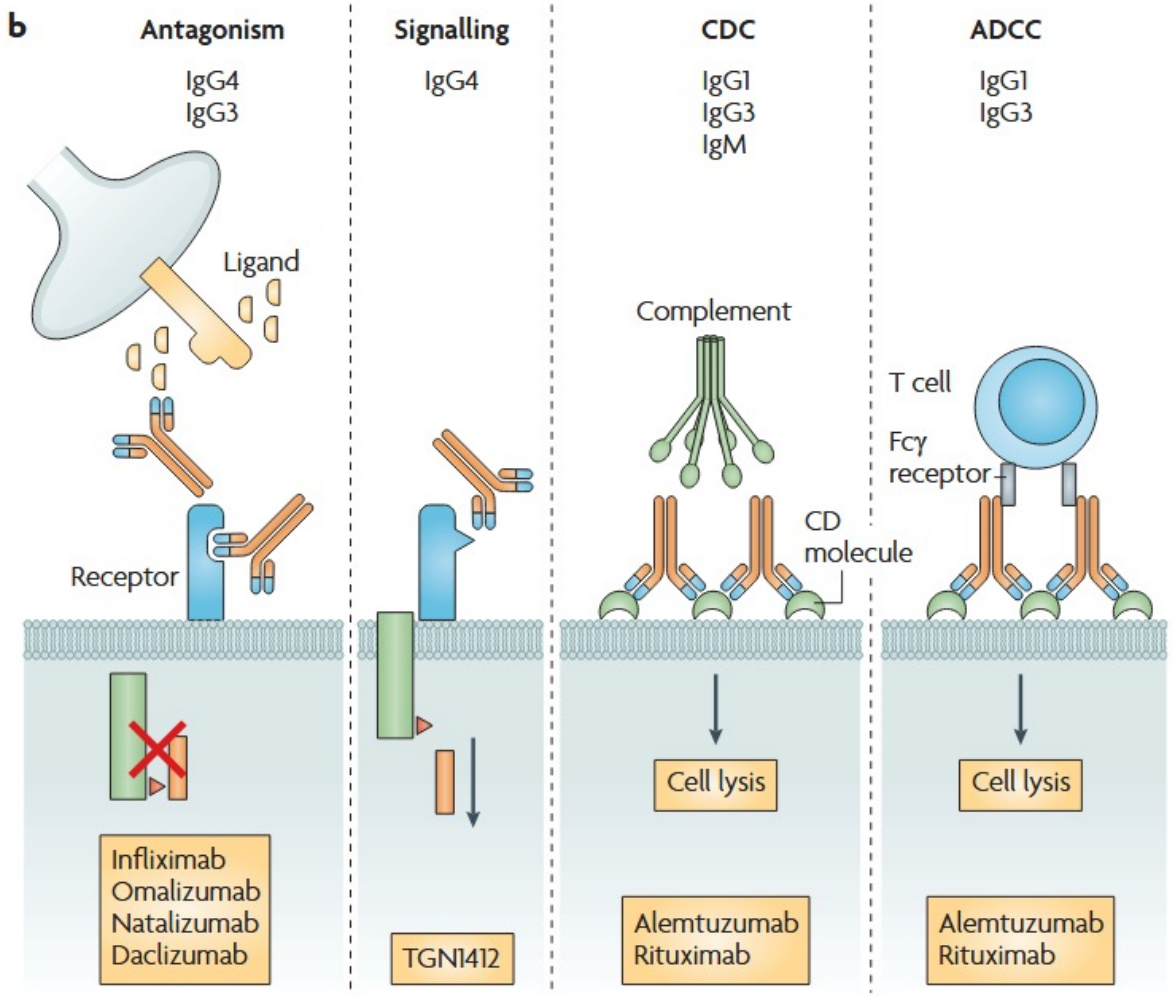
Mechanisms of actions => Lysis mediated by:	
ADCC Antibody-Dependent Cellular Cytotoxicity	Recruitement of immune effector cells such as **natural killer (NK) cells** and **macrophages;** release cytotoxic molecules (such as perforin and granzymes) to destroy the B cells
CDC Complement-Dependent Cytotoxicity	Activation of the **complement system:** This results in the formation of the **membrane attack complex (MAC)**, which leads to the lysis and death of the targeted B cells.
Apoptosis	Induction of **lysis** by binding to CD20

Monoclonal antibody binds to one epitope/antigen, e.g. CD20



Types of mAbs

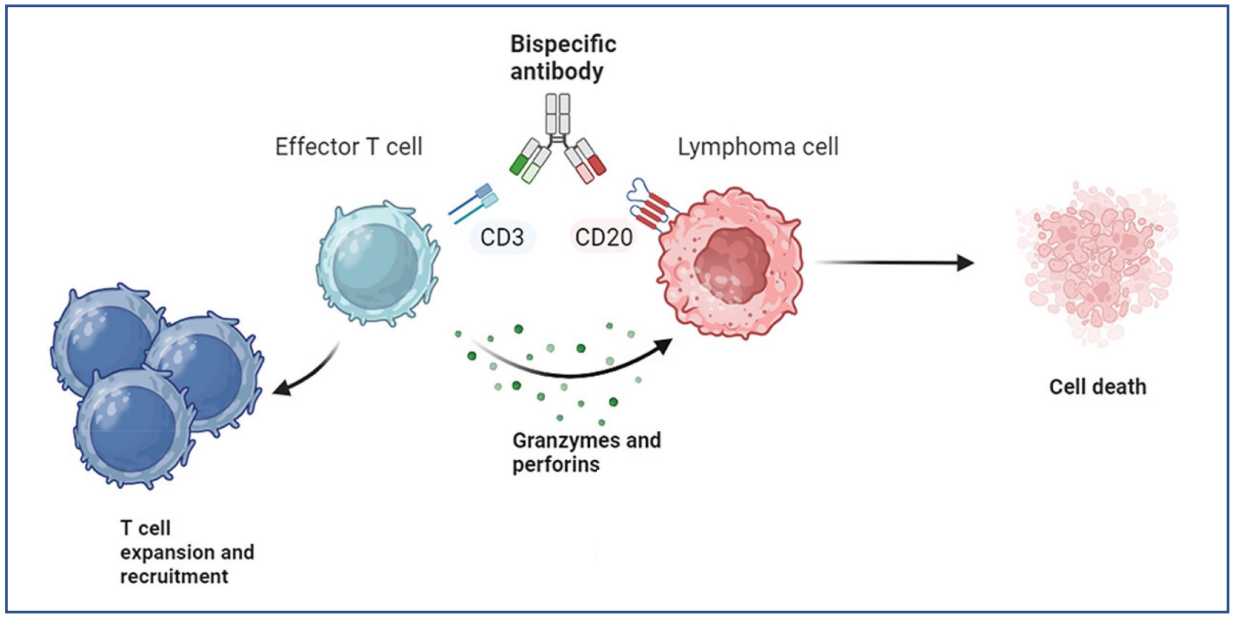
Murine	Entirely murine amino acids	'o' = mouse e.g. muromonab
Chimeric	Human constant (C) + murine variable (V) regions	'xi' = chimeric e.g. rituximab
Humanized	Murine complementarity determining regions (CDRs)	'zu' = humanized e.g. alemtuzumab
Human	Entirely human amino acids	'u' = human e.g. adalimumab

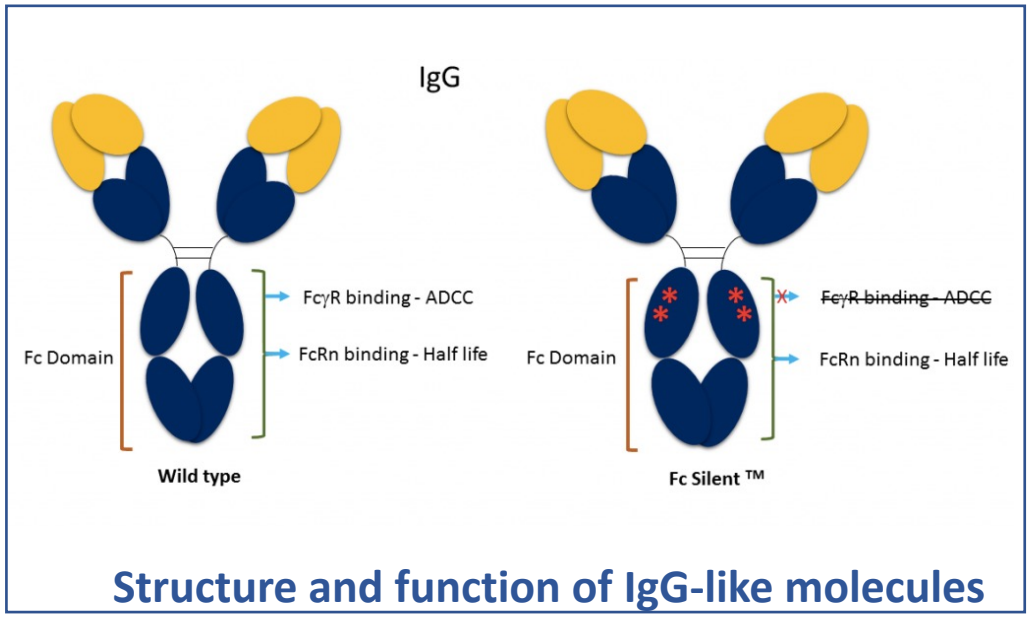


Hansel TT , Kropshofer H , Singer T, , Mitchell JA, George AJT Nature Rev Discovery 2010

Bispecific antibody binds to two epitopes/antigens

Bridging two types of cells together : tumor cells and effector cells





Structure and function of IgG-like molecules

- **BsAbs** binds **to CD3 on the T-cells** and **to a surface Ag on the tumour cells**, and form a **stable immunological synapse.**
- **This synapse is independent of FcγR and MHC and** initiates **T-cell activation, leading the release of cytotoxic granules and death of the target cell**.
- The activation of T-cells yields to **local T-cell proliferation** and **cytokine release**, **enhancing T-cell recruitment**

- **Different mutations** are introduced **in the Fc domain** of most IgG-like BsAb **to prevent Fc-mediated functions (ADCC, phagocytosis, CDC)**
- **FcγR and C1q** binding could result **in T-cell activation and cytotoxicity, fratricidal antibody and complement-dependent cellular cytotoxicity**

Cassanello et al. Oncoimmunology 2024, vol. 13, no. 1, 2321648

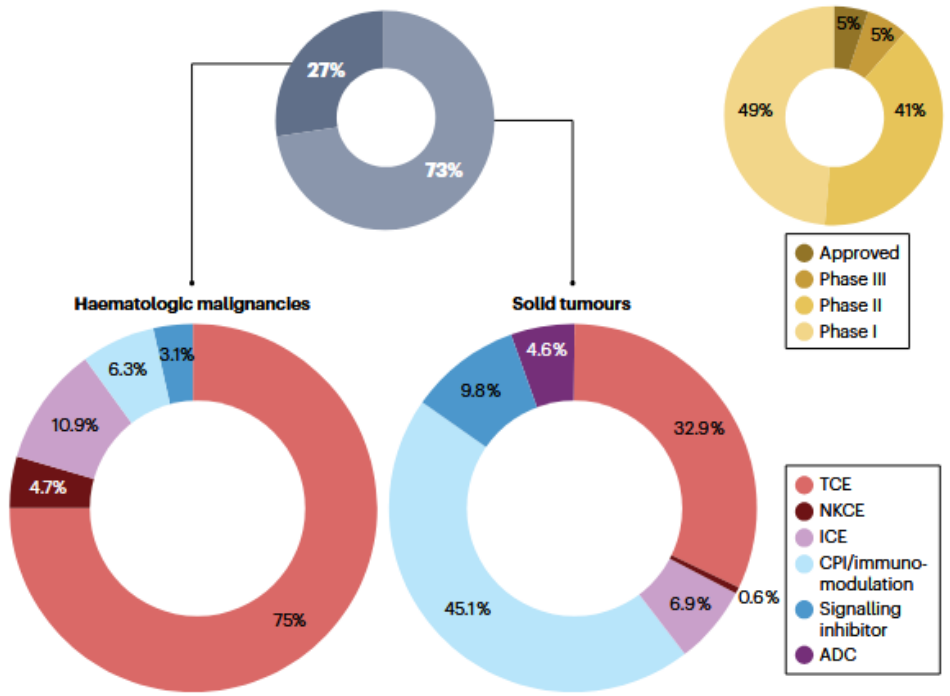
Since 2022 : approval of 10 more T-cell-recruiting bispecifics antibodies

10 approved T-cell engagers for cancer therapy

bsAb	International non-proprietary name	Targets	MoA	Format	Year of first approval/ region[a]	Indications	Company
	Catumaxomab	EpCAM×CD3ε	TDCC	Quadroma mouse/rat 1+1	2009 Withdrawn EU 2013	Ovarian ascites, intraperitoneal	Trion Pharma/ Fresenius
	Blinatumomab	CD19×CD3ε	TDCC	BiTE 1+1	2014 United States/EU, Japan	ALL	Amgen
	Amivantamab	EGFR×MET	Signalling inhibition, ADCC	Duobody 1+1	2021 United States/EU	NSCLC EGFR exon 20 insert mutation	J&J
	Tebentafusp	gp100-HLA-A*02×CD3ε	TDCC	scFv-TCR fusion 1+1	2022 United States/EU	Uveal melanoma	Immunocore
	Mosunetuzumab	CD20×CD3ε	TDCC	KiH 1+1 IgG	2022 United States/EU	Relapsed/refractory follicular NHL	Roche group
	Cadonilimab	PD1×CTLA4	Dual checkpoint inhibition	IgG-scFv tetrabody 2+2	2022 China	Hepatocellular carcinoma	Akeso Bio
	Teclistamab	BCMA×CD3ε	TDCC	Duobody 1+1	2022 United States/EU	Relapsed/refractory multiple myeloma	J&J
	Glofitamab	CD20×CD3ε	TDCC	CrossMAb 2+1	2023 United States/EU	Relapsed/refractory DLBCL	Roche group
	Epcoritamab	CD20×CD3ε	TDCC	Duobody 1+1	2023 United States/EU, Japan	Relapsed/refractory DLBCL	Genmab, Abbvie
	Talquetamab	GPRC5D×CD3ε	TDCC	Duobody 1+1	2023 United States/EU	Relapsed/refractory multiple myeloma	J&J
	Elranatamab	BCMA×CD3ε	TDCC	bsAb 1+1	2023 United States/EU	Relapsed/refractory multiple myeloma	Pfizer

ADCC, antibody-dependent cellular cytotoxicity; ALL, acute lymphocytic leukaemia; BCMA, B cell maturation antigen; BITE, bispecific T cell engager; bsAb, bispecific antibody; DLBCL, diffuse large B cell lymphoma; EGFR, epidermal growth factor receptor; EpCAM, epithelial cellular adhesion molecule; GPRC5D, G-protein-coupled receptor class C group 5 member D; MoA, mechanism of action; NSCLC, non-small cell lung cancer; NHL, non-Hodgkin lymphoma; scFv, single-chain variable fragment; TCR, T cell receptor; TDCC, T cell-dependent cellular cytotoxicity. [a]Region of approval limited to the United States, the European Union (EU), Japan and China; products may also be approved in other countries. Status as of end of 2023.

Bispecific Abs in clinical development

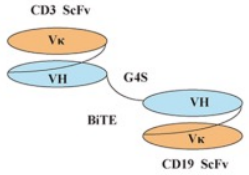


CPI, checkpoint inhibitor;
ICE, innate cell engager;
NKCE, natural killer cell engager

Klein C, Brinkmann U, Reichert JM, Kontermann RE. Nat Rev Drug discov 2024 Apr;23(4):301-319

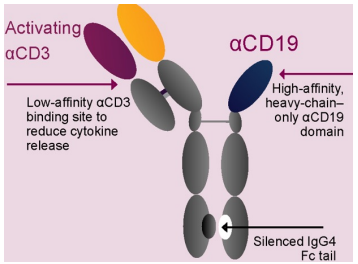
Bispecific antibodies in B-cell malignancies

CD3 x CD19



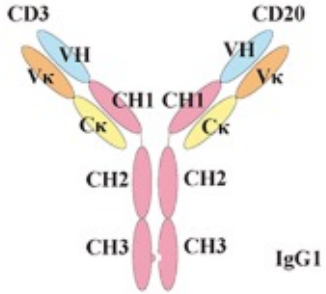
Blinatumomab

AZD0486

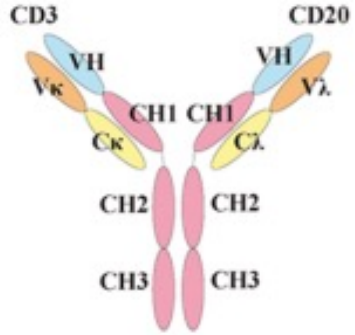


CD3 x CD20

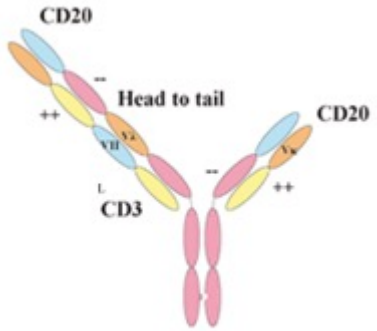
Ig G-based



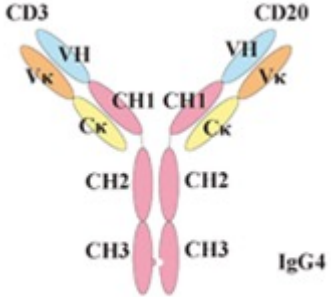
Mosunetuzumab



Epcoritamab

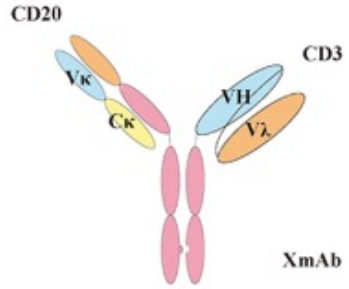


Glofitamab



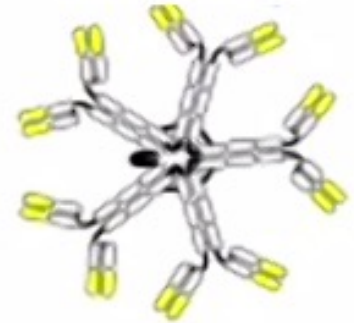
Odronextamab

Under development



Plamotamab

Ig M-based



Imvotamab

Adapted Ma et al. Frontiers in Immunology 2021

BsAbs single agents in R/R LBCL : space to improve

ORR : 40-67%

mPFS : 3.2 – 8.6 mo

in R/R LBCL

Clinical Trial	Phase	Drug(s)	Histology	Modifiers	N	ORR	CR	mPFS (months)	mDOR (months)	Grade 3–4 CRS	Grade 3–4 ICANS	Grade 3–4 NP	mFU (months)
NCT02500407	I/II	MOSUNETUZUMAB	DLBCL	Dose expansion cohort	88	40%	24%	3.2	7	2.3%	None	21.6%	10.1
NCT03625037	I/II	EPCORITAMAB	DLBCL	Dose expansion cohort	157	63%	39%	4.4	12	2.5%	0.6%	14.6%	10.7
NCT03075696	II	GLOFITAMAB	DLBCL	Dose expansion cohort	155	52%	39%	4.4	18.4	4%	3%	27%	12.6
NCT04657302	I	GLOFITAMAB	DLBCL	Dose expansion cohort	27	67%	52%	8.6	14.4	3.3%	3.3%	30%	15
NCT03888105	II	ODRONEXTAMAB	DLBCL	Dose expansion cohort	127	52%	31%	N/A	10.2	0.7%	None	N/A	26.2
NCT02924402	I	PLAMOTAMAB	DLBCL	Dose escalation cohort	19	47%	26%	N/A	N/A	None	None	16.7%	N/A
NCT04082936	I	IgM-2323	B-NHL	Dose escalation cohort	23	35%	22%	N/A	N/A	3.4%	N/A	N/A	N/A
NCT04594642	I	AZD0486	DLBCL	Dose escalation cohort	5	40%	20%	N/A	N/A	None	7%	15%	3.8
NCT04923048	I/II	GB261	B-NHL	Dose escalation cohort	22	73%	45%	N/A	NR	None	None	14.9%	4.1

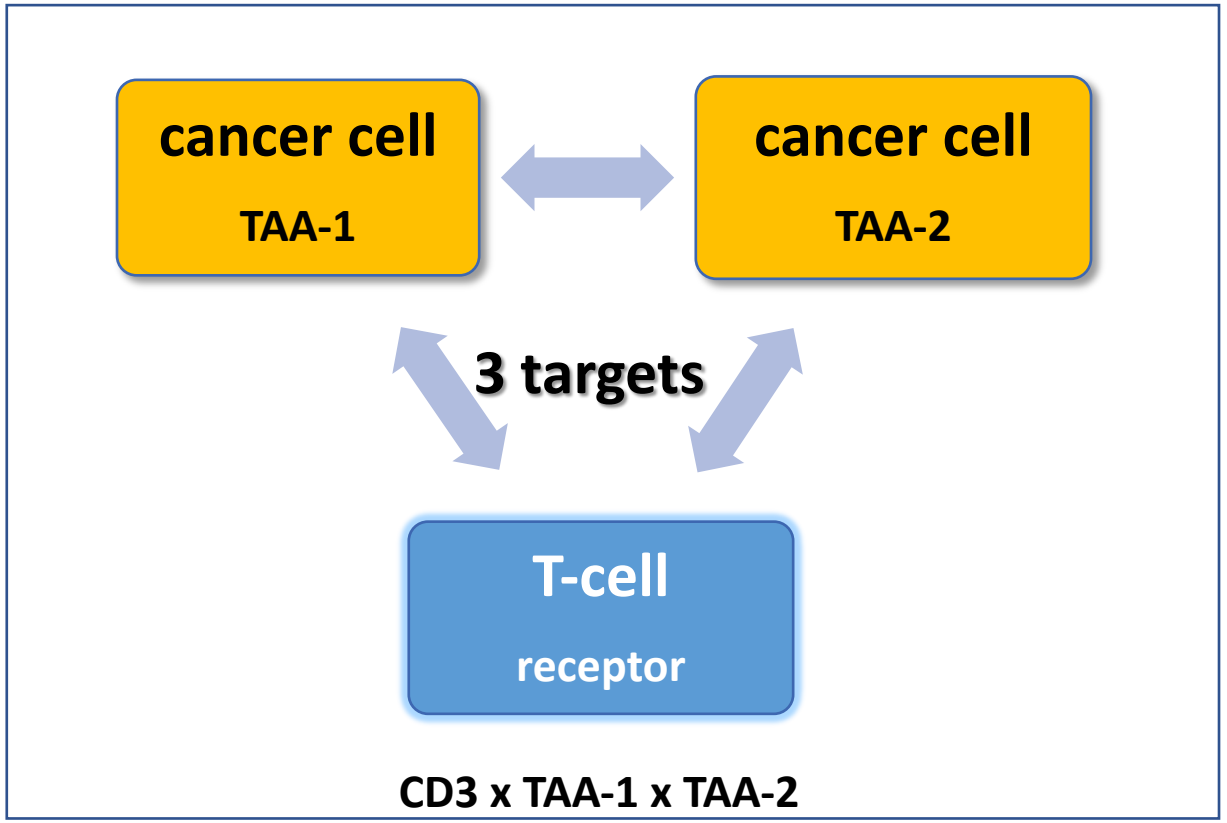
Adapted from : Cassanello et al. Oncoimmunology 2024, vol. 13, no. 1, 2321648

Agenda

- From monospecific to bispecific and trispecific antibodies : structures and mechanisms of action
- Trispecific antibodies
- Immune-interfacing multispecific bispecific antibodies

Diverse structures of the trispecific Abs

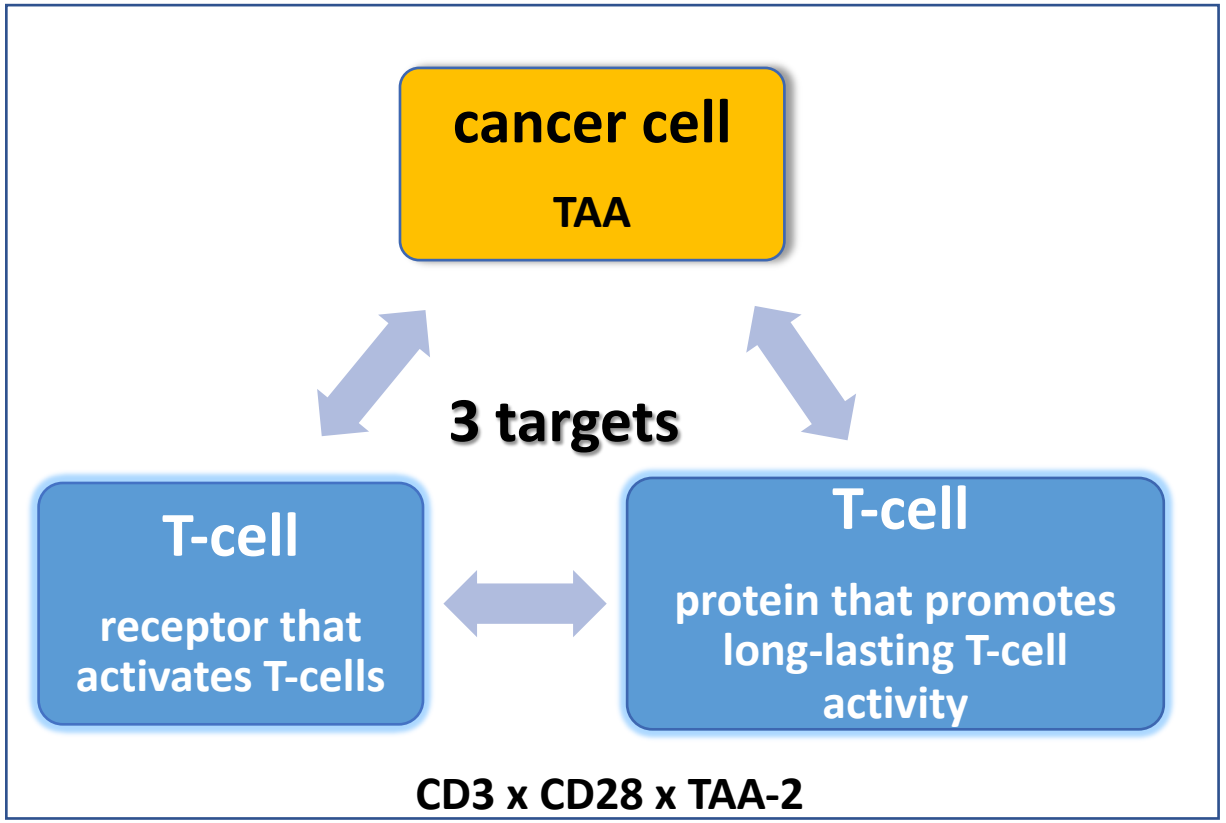
Dual targeting T-cell engagers



- **2 targets on the cancer cells**
- **CD3 drives T-cell activation** (without requiring antigen recognition by the TCR), which leads to the killing of the myeloma cell and the production and release of toxic cytokine molecules.

TAA = tumour-associated antigen

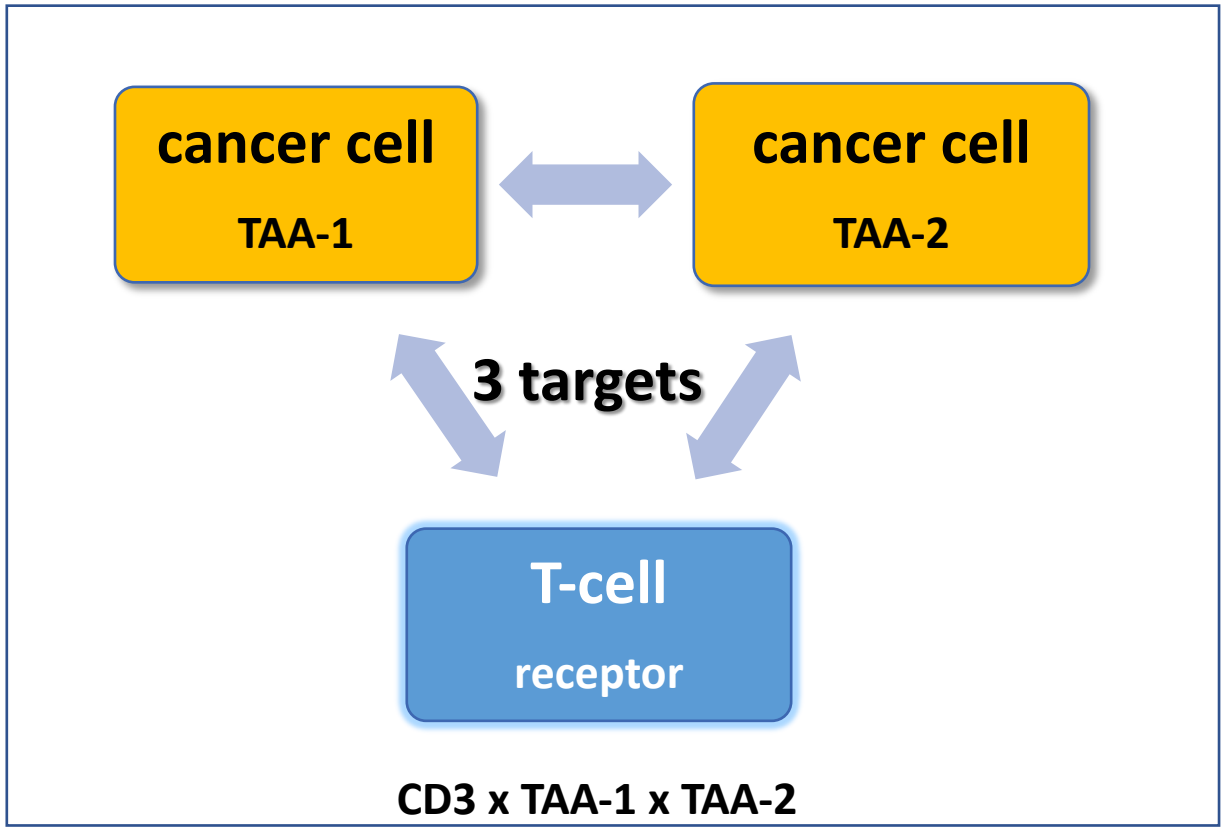
T-cell engagers with integrated co-stimulation



- **CD3 drives T-cell activation** (without requiring antigen recognition by the TCR), which leads to the killing of the myeloma cell and the production and release of toxic cytokine molecules.
- **CD28 = co-stimulatory receptor, which positively regulate T-cell activation =>** prolonged TCR activation

Diverse structures of the trispecific Abs

Dual targeting T-cell engager



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T-cell engager with integrated co-stimulation

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Dual targeting with trispecific antibodies

>>> to overcome Target Antigen loss or Target Antigen escape variants

TAA-1 x TAA-2 x CD3

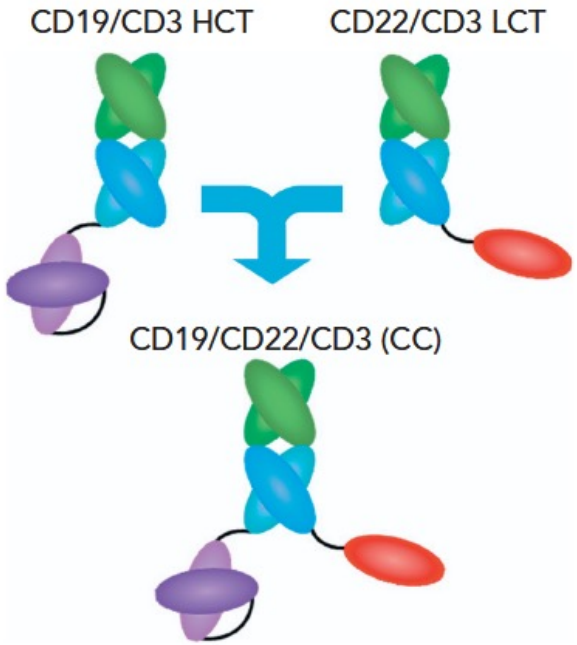
Drug	Targets	indication	Phase	NCT	compagny
A-2019	**CD19/CD20/CD3**	B-ALL	-	-	Wang et al
TsAb	**CD19/CD22/CD3**	B-ALL	-	-	Zhao et al
JNJ-80948543	**CD79b/CD20/CD3**	B-cell NHL, CLL	Phase I, First-in-human Study	NCT05424822	Janssen
1A46	**CD19/CD20/CD3**	B-cell NHL, ALL	Phase I, First-in-human Study	NCT05348889	GSK

TAA Target-Associated-Antigen

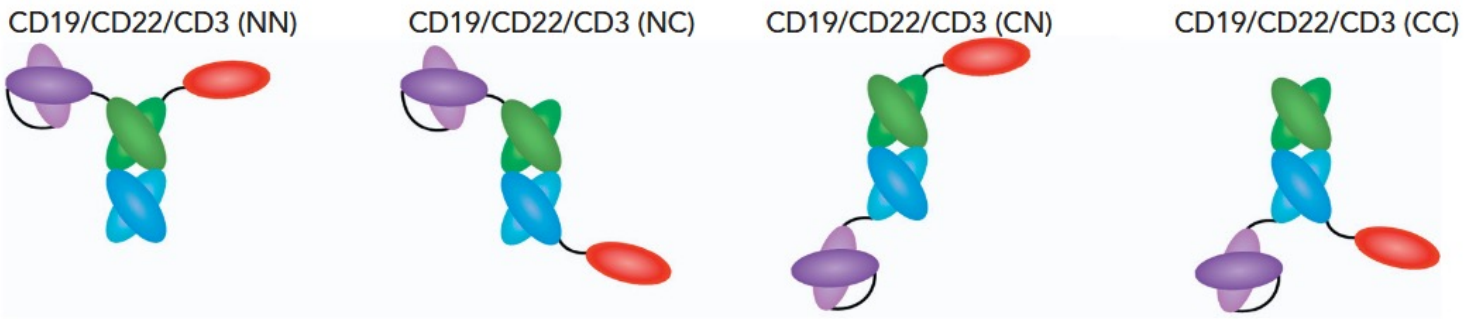
Sisi Wang, Lijun Peng, Wenqian Xu et al. Front Med 2022 Feb;16(1):139-149
Lijun Zhao, Shuhong Li, Xiaoyi Wei, et al.. Blood 2022; 140 : 1790-1802
Kuchnio A. Blood, 2022-11, Vol.140 (Supplement 1), p.3105-3106

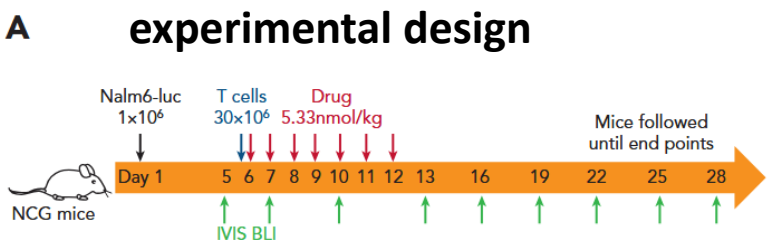
CD19/CD22/CD3 TsAb in ALL

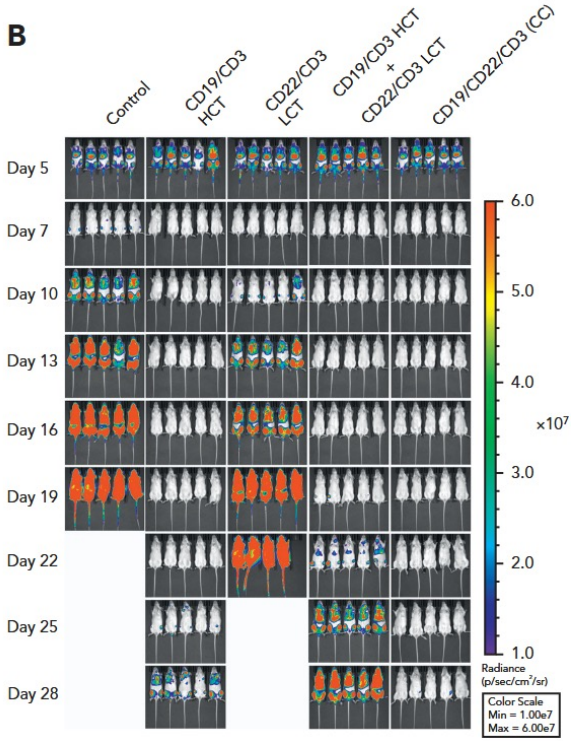
Schematic structure of CD19/CD22/CD3 by fusing CD19/CD3 and CD22/CD3



Schematic structures of different CD19/CD22/CD3 tsAbs







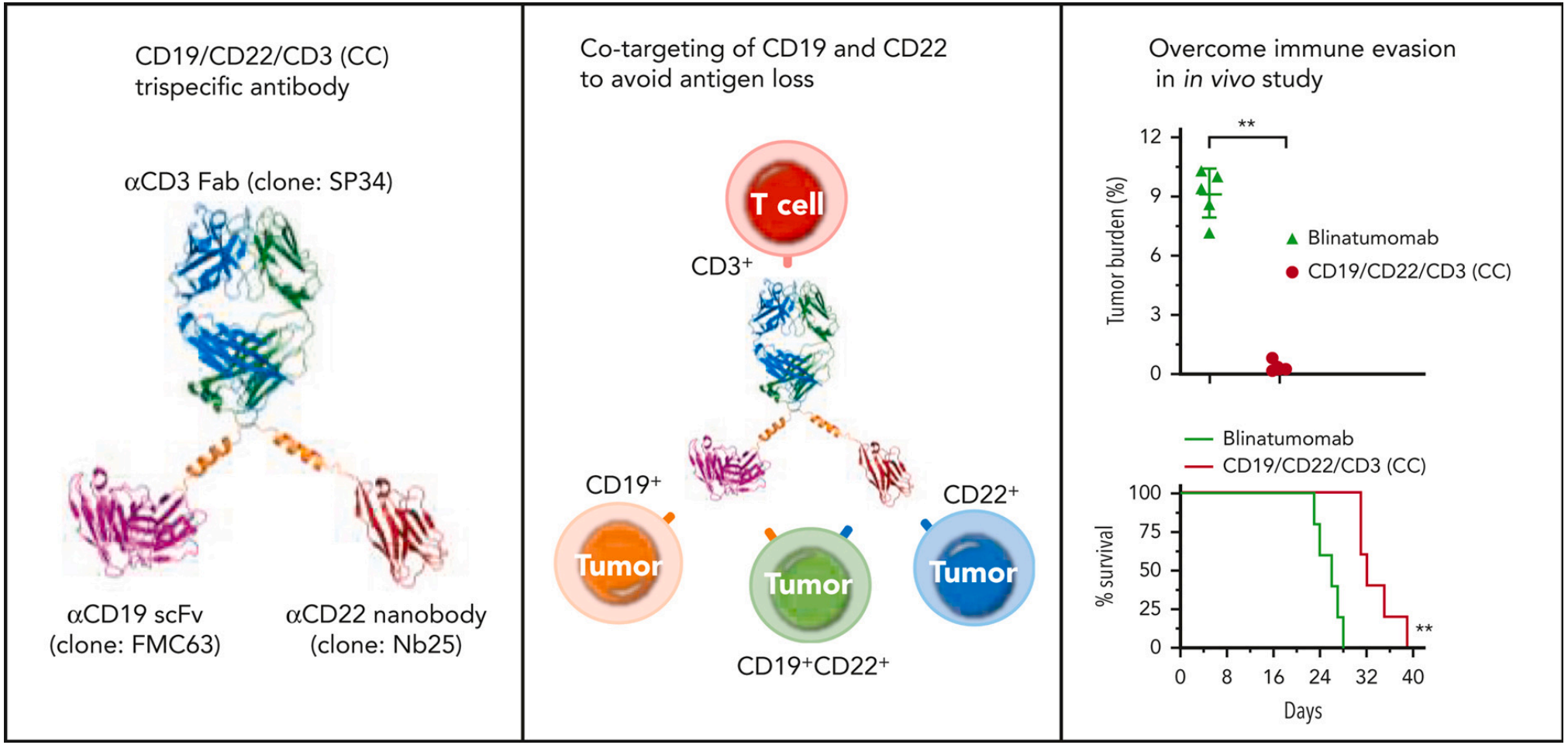
B-ALL patient samples for PDX model

Patient	Sex	Age, y	Diagnosis	Disease status	% Blasts in the blood
B-ALL Pt #1	F	29	B-ALL	De novo	75.50
B-ALL Pt #2	M	45	CML to Com-B-ALL	Relapse	35.11
B-ALL Pt #3	F	72	B-ALL	De novo	88.67
B-ALL Pt #4	M	14	Pre-B-ALL	De novo	94.85
B-ALL Pt #5	M	17	Pre-B-ALL	De novo	71.90
B-ALL Pt #6	F	67	B-ALL	De novo	89.83

Lijun Zhao et al. Blood 2022; 140 : 1790-1802

CD19/CD22/CD3 TsAb in ALL

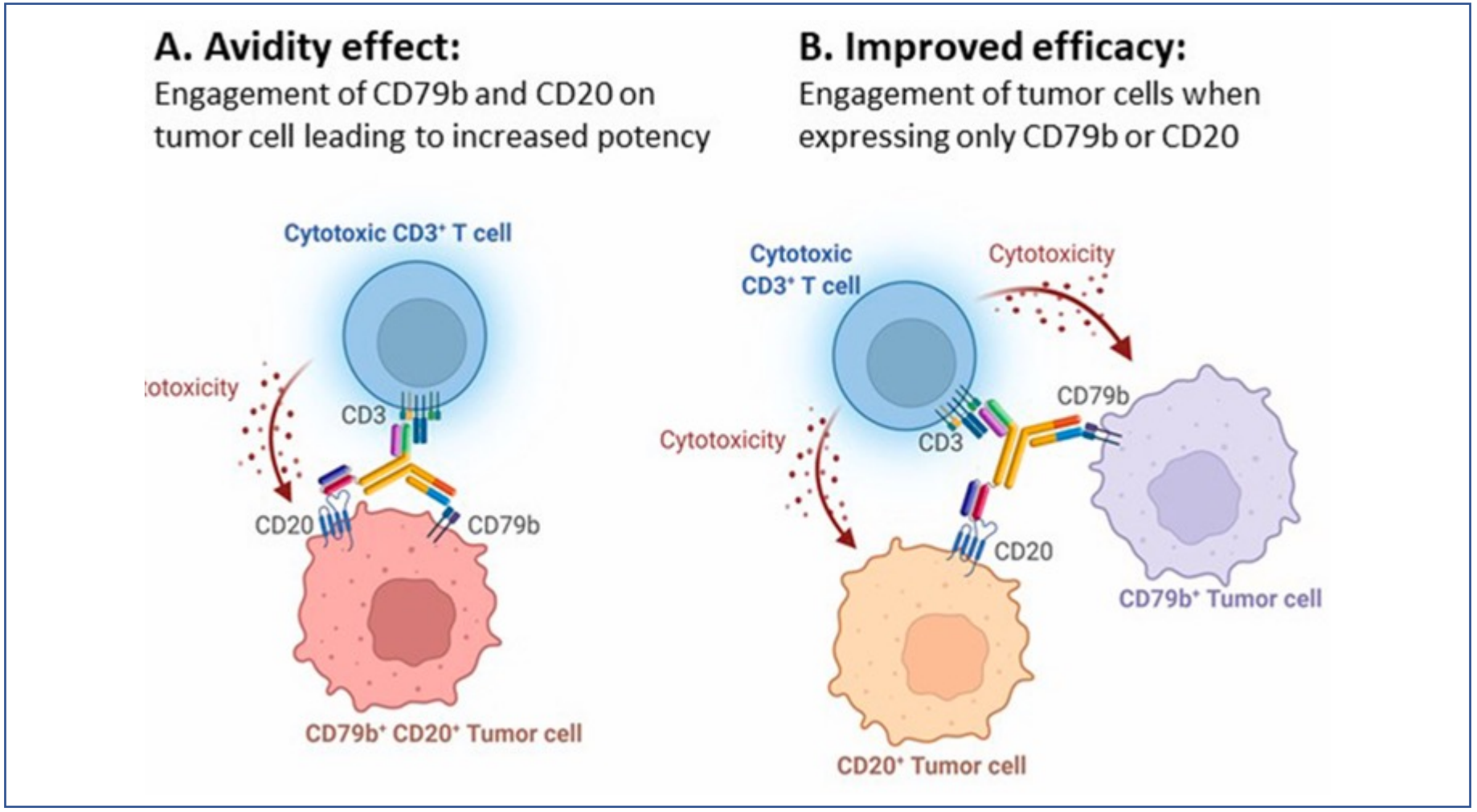
A novel CD19/CD22/CD3 trispecific antibody enhances therapeutic efficacy and overcomes immune escape against B-ALL



Lijun Zhao et al. Blood 2022; 140 : 1790-1802

CD79b/CD20/CD3 Trispecific in B-Cell Non-Hodgkin Lymphoma

CD79b/CD20/CD3



Kuchnio A. Blood, 2022-11, Vol.140 (Supplement 1), p.3105-3106

Phase I/II trial with CD79b/CD20/CD3 trispecific antibody in patients with NHL and CLL

: CD79b/CD20/CD3 TsAbs

Recruitment status	Recruiting
Disease entity	R/R NHL + CLL Including LBCL, FL, MW, MZL, MCL, CLL
Date of first enrollment	31/07/2024
Target sample size	180

Part A
Dose Escalation

JNJ-80948543 SC

↓

Putative RP2Ds

Part B
Cohort Expansion

Expansion Cohort(s)

JNJ-80948543 SC

RP2Ds as determined in Part A

Primary Endpoints
- Occurrence and severity of AEs
- DLT

Secondary Endpoints
- Serum concentration of JNJ-80948543
- Number of participants with anti-drugs Abs
- ORR
- CR
- GVPR (for WM)
- TTR
- DOR

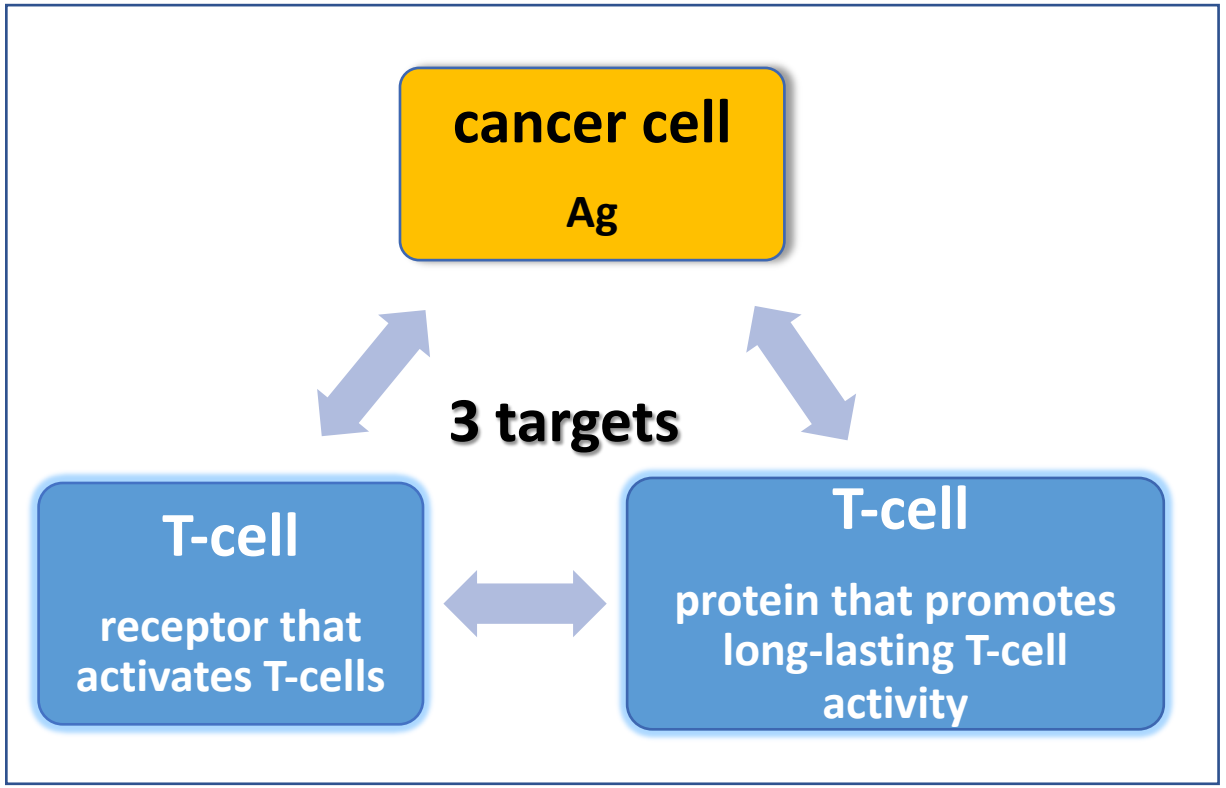
Clinicaltrial.gov : NCT06660563

Diverse structures of the trispecific Abs

Dual targetting T-cell engager

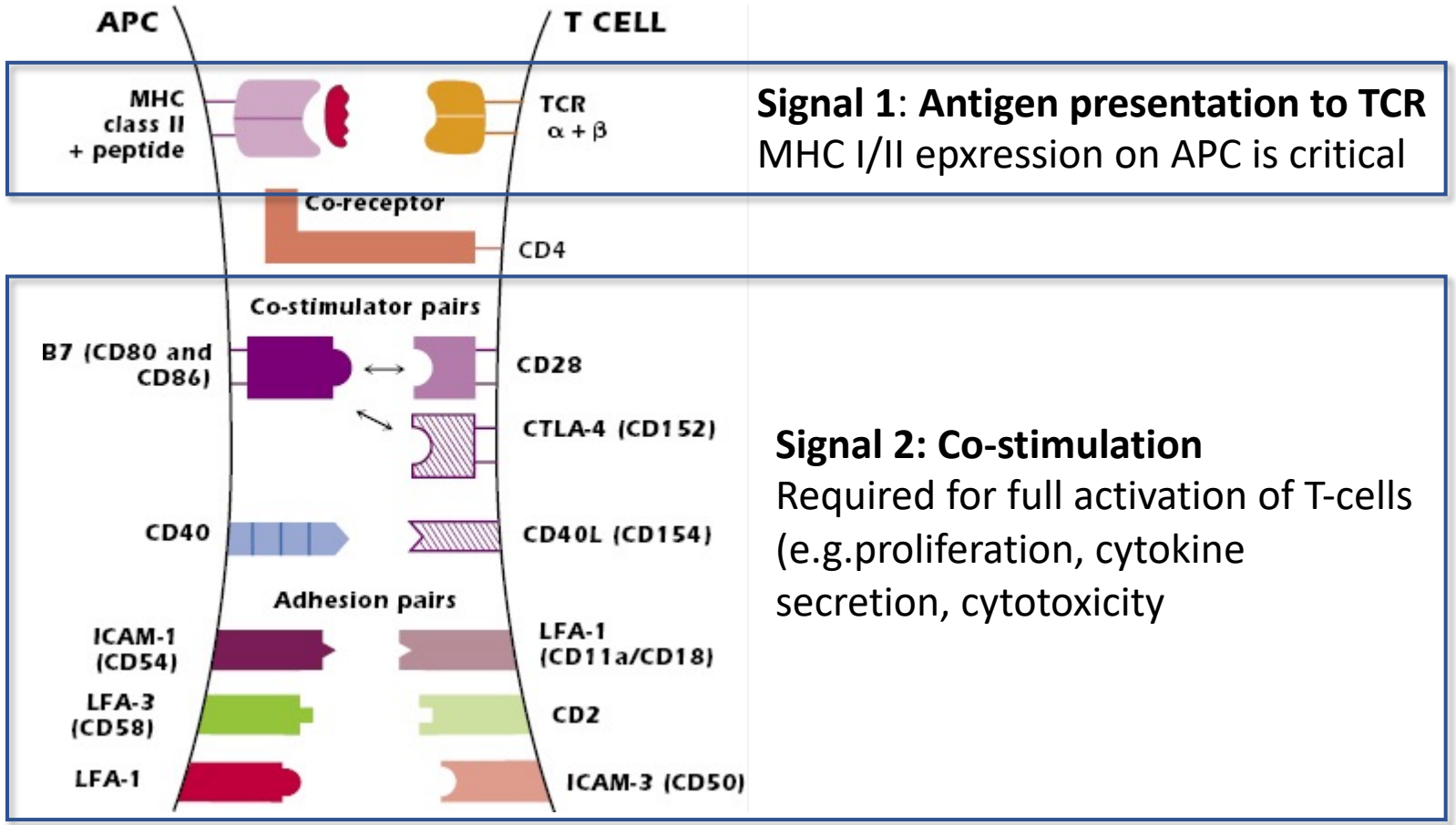
- 2 targets on the cancer cells
- **CD3 drives T-cell activation** (without requiring antigen recognition by the TCR), which leads to the killing of the myeloma cell and the production and release of toxic cytokine molecules.

T-cell engager with integrated co-stimulation



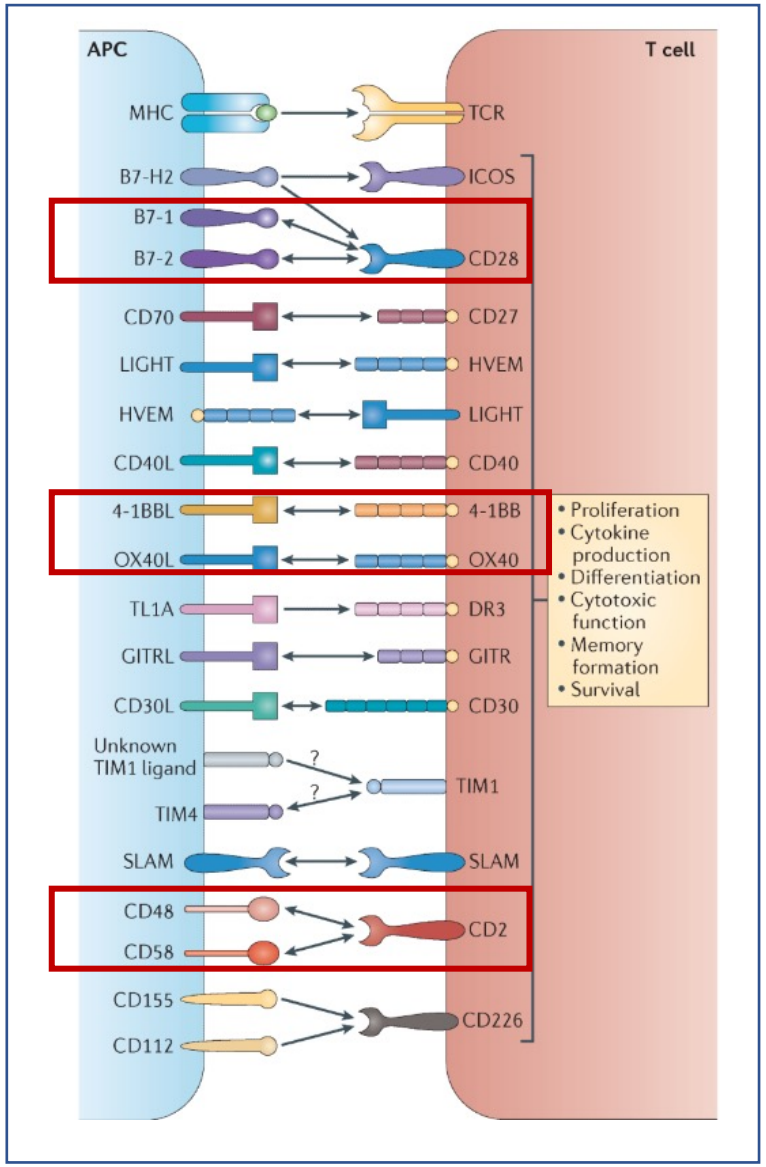
- **CD3 drives T-cell activation** (without requiring antigen recognition by the TCR), which leads to the killing of the myeloma cell and the production and release of toxic cytokine molecules.
- **CD28 = co-stimulatory receptor, which positively regulate T-cell activation =>** prolonged TCR activation

Physiological T-cell activation relies on Signal 1 (TCR-MHC) & Signal 2 (e.g. CD28-CD86)



Signal 1: **Antigen presentation to TCR**
MHC I/II epxression on APC is critical

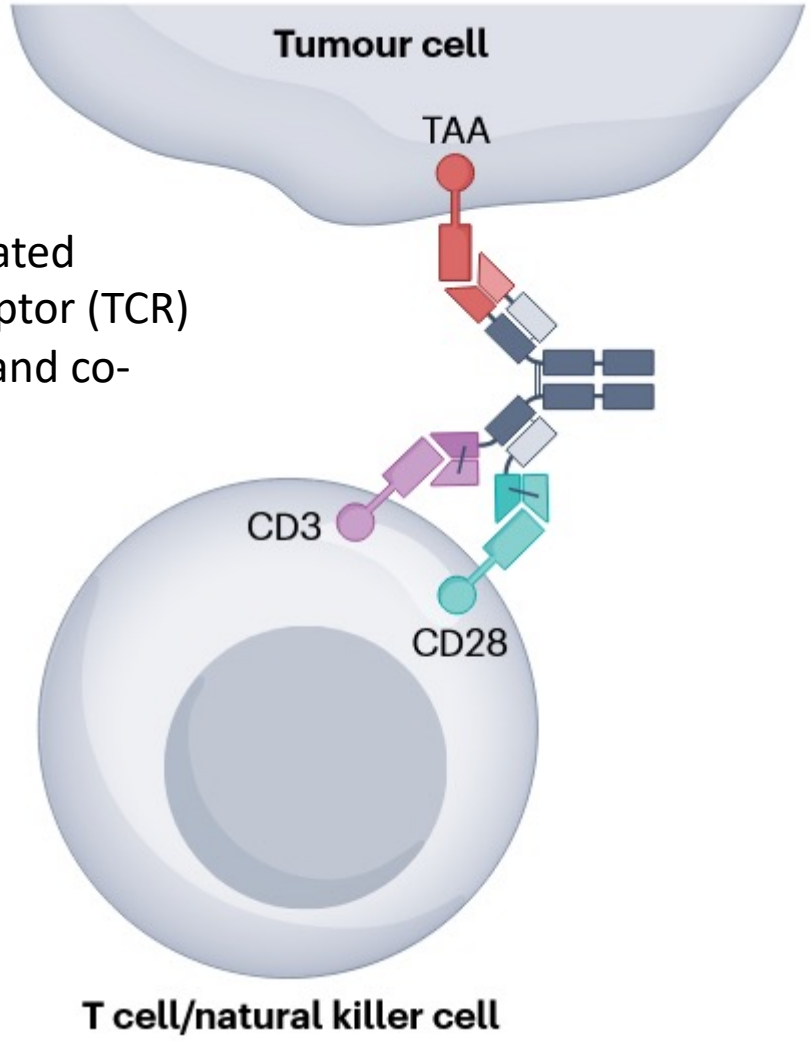
Signal 2: Co-stimulation
Required for full activation of T-cells (e.g.proliferation, cytokine secretion, cytotoxicity



Chen L, Flies D. Nat Rev Immunol, 2013

T-cell engager with integrated co-stimulation

Trispecific T-cell engager (TCE) with integrated costimulation for simultaneous T cell receptor (TCR) activation (signal 1) through CD3 binding and co-stimulation (signal 2) by binding to CD28.

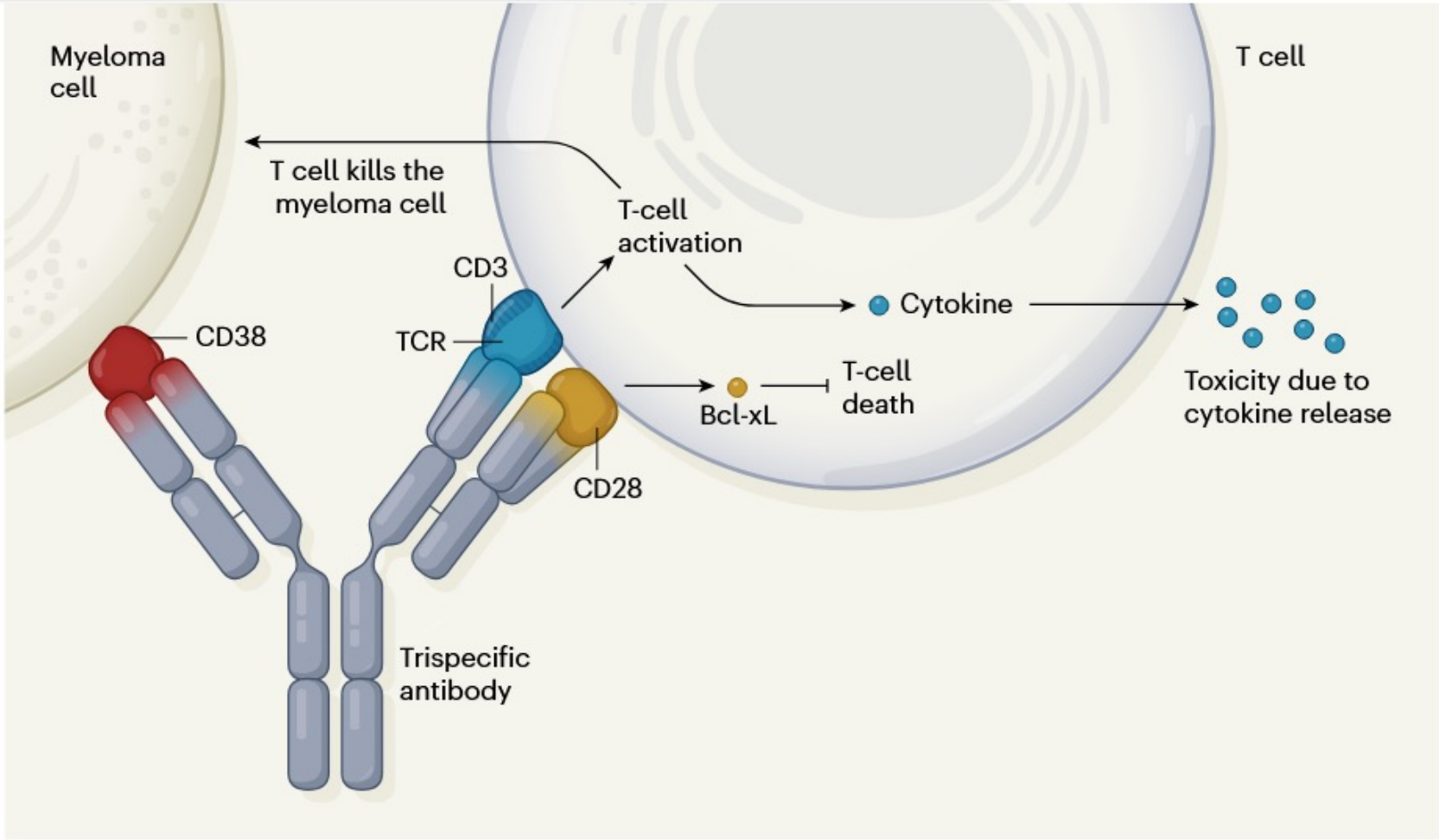


TAA = tumour-associated antigen

The first exemple : in myeloma

The trispecific antibody binds :

- **CD38** on a myeloma cell
- and the protein **CD28** and the protein complex **CD3** on a T-cell



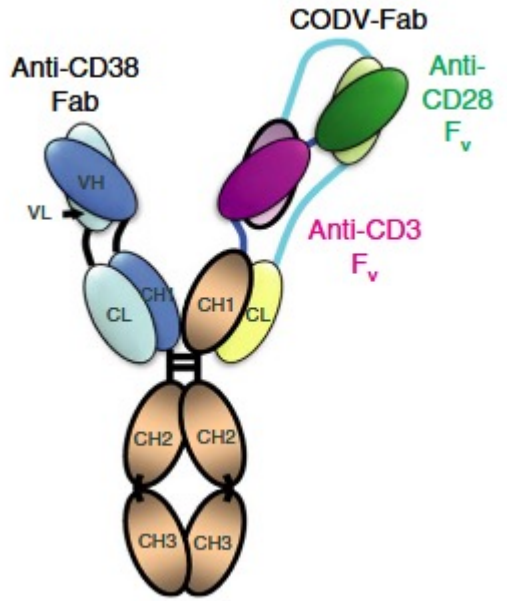
Alfred L. Garfall & Carl H. June. Nature 2019 « ***Three is a charm for an antibody to fight cancer*** »

Trispecific Antibodies in Development

Drug	Targets	indication	Phase	NCT	compagny
SAR442257	**CD38 /CD28xCD3**	Multiple myeloma, AML	Phase I, First-in-human Study	NCT04401020	
PIT565	**CD19/CD3xCD2**	B-cell NHL, B-ALL	Phase I, First-in-human Study	NCT05397496	

Trispecific antibodies enhance the therapeutic efficacy of tumor-directed T cells through T cell receptor co-stimulation

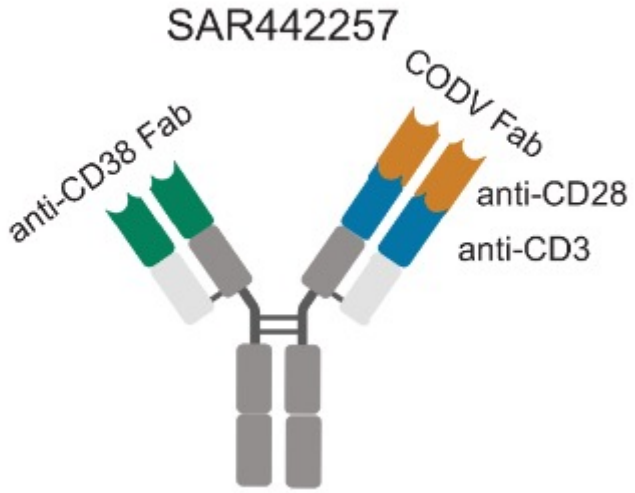
Research and Development



- **The trispecific antibody** interacts with **CD38, CD3 and CD28 to enhance both T cell activation and tumor targeting**
- **The engagement of both CD3 and CD28 affords efficient T cell stimulation, whereas the anti-CD38 domain directs T cells to myeloma cells.**

Wu Lan et al. Nature Cancer, 2019 ; 1: 86–98

CD38/CD28xCD3 Trispecific T-Cell Engager (TCE) in multiple myeloma

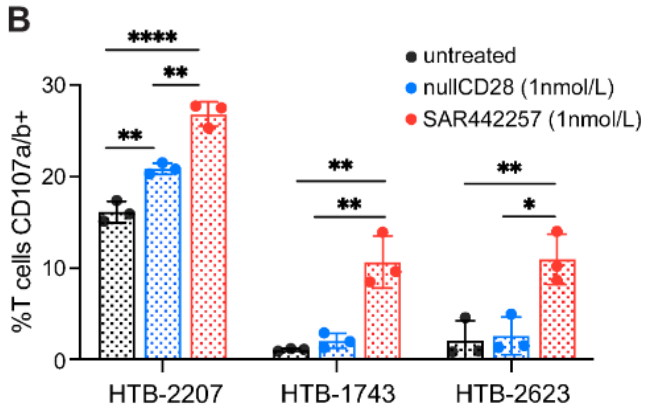


Effect of SAR442257 on multiple myeloma cell viability in 3 populations of patients :

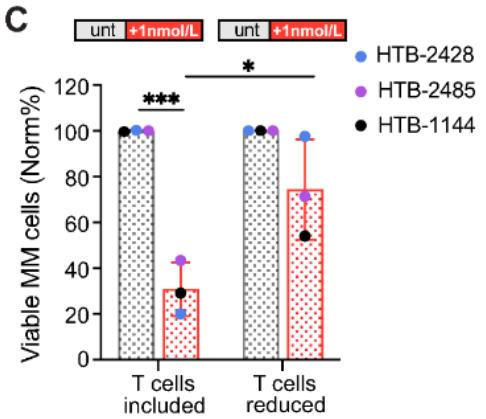
- **newly diagnosed multiple myeloma**
- **Dara-exposed**
- **Dara-exposed + post-BCMA therapy patients**

34 samples

Highly effective in patients relapsing after BCMA therapy



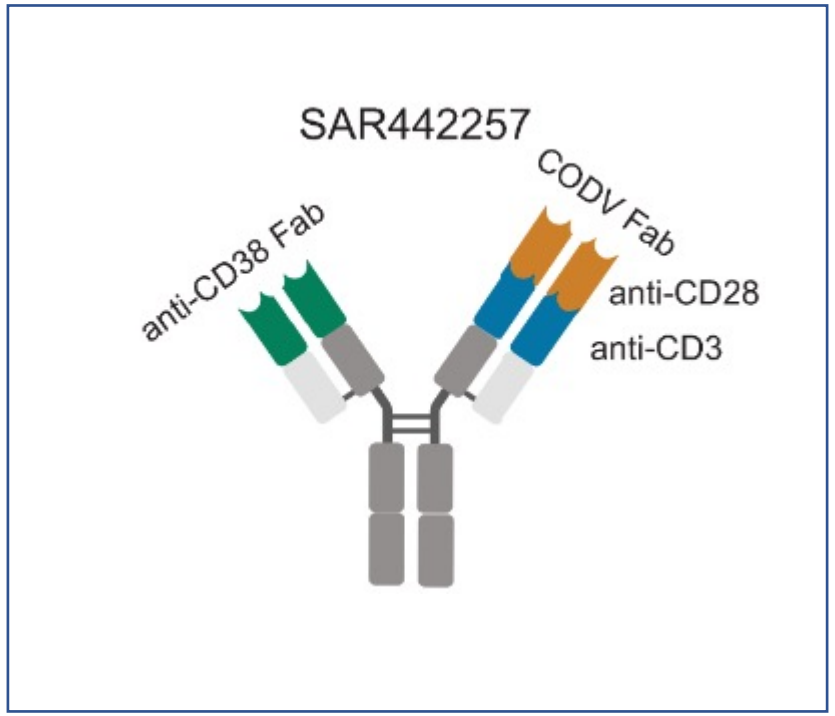
higher degranulation of CD3+ T cells in the presence of 1 nmol/L SAR442257



Efficacy of SAR442257 in My-DST

Keller et al. Cancer Res Commun; 4(3) March 2024

CD38/CD28xCD3 Trispecific T-Cell Engager (TCE) As a Potentially Active Agent for the Treatment of Older Patients with AML

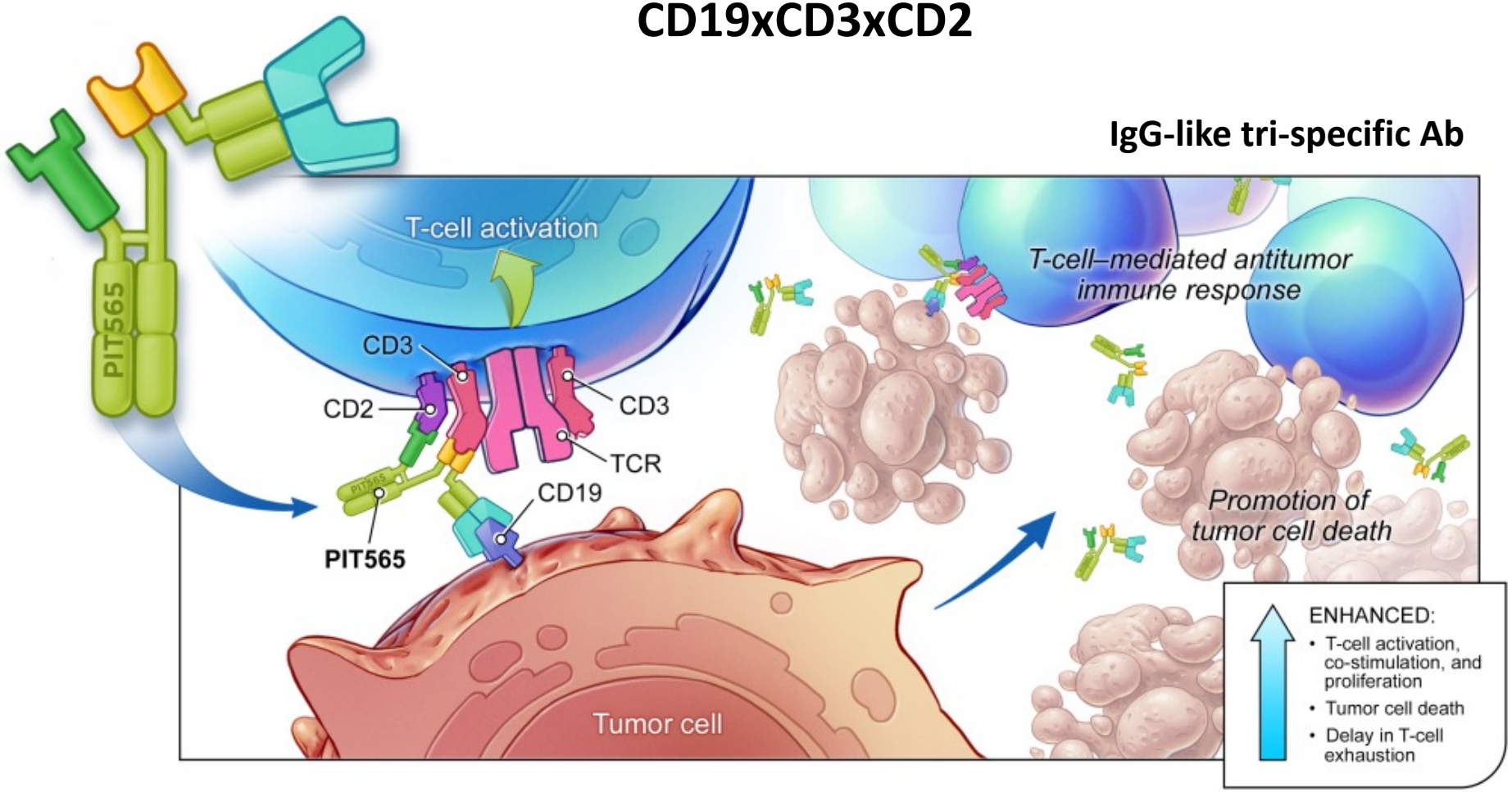


- **CD38/CD28xCD3 TCE exerted its anti-tumor efficacy regardless of CD38 density**
- **AML patients expressing both high and low/heterogenous levels of CD38 could benefit from T-cell based immunotherapeutic strategies targeting CD38.**

Schjesvold Fredrik, et al. Blood 144 (2024) 1992–1994 – ASH 2024

A phase 1 study of PIT565, CD19xCD3xCD2, Trispecific Ab, First-in-class in patients with R/R B-cell NHL (R/R HGBCL, PMBL, FL3B, ALL)

CD19xCD3xCD2



Irit Avivi Mazza et al. ASH 2023, P3099

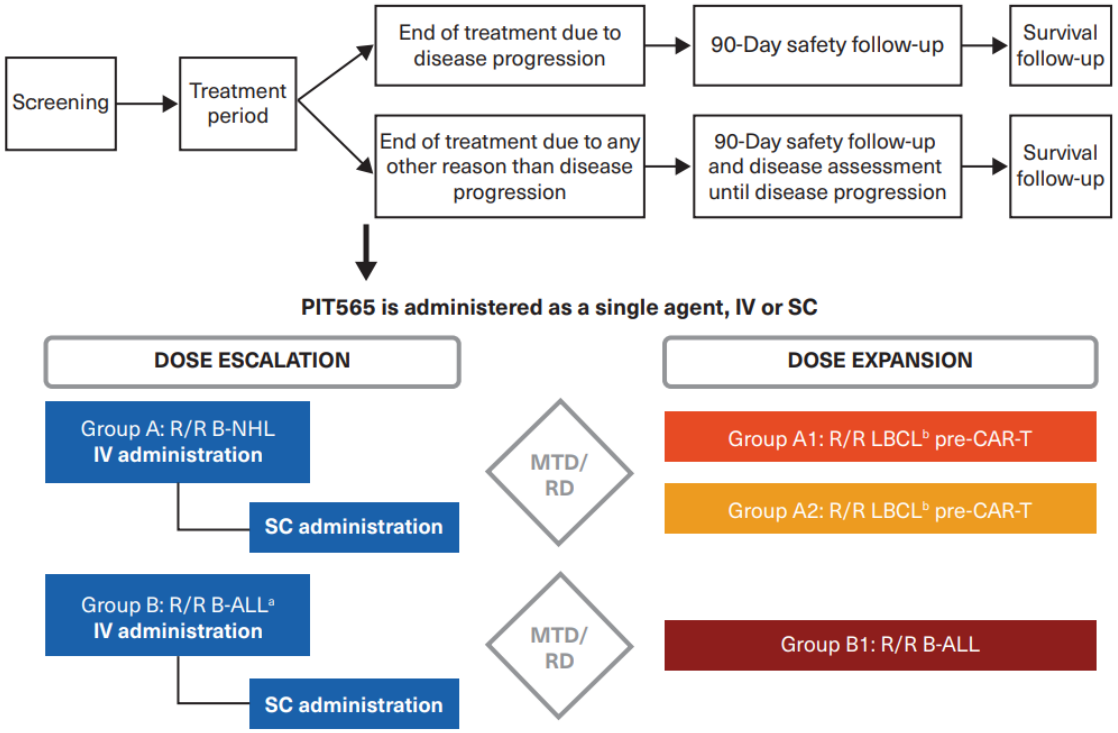
PIT565, trispecific Ab : CD19/CD3/CD2

n=140 patients

Key Inclusion Criteria
• Eligible patients (age: ≥18 years) with R/R B-NHL and R/R CD19-positive B-ALL who had relapsed/failed to respond to ≥2 lines of prior therapy, including an αCD20 monoclonal antibody–containing chemotherapy combination regimen
• ECOG ≤2
• R/R B-NHL patients must have at least 1 bi-dimensionally measurable nodal lesion or 1 bi-dimensionally measurable extranodal lesion as measured on positron emission tomography-computed tomography scan
• For R/R B-ALL patients, conformed morphologic of BM (≥5% blasts)

Belgium, France, Israel, Italy, Japan, Singapore, Spain, United States

Study Design

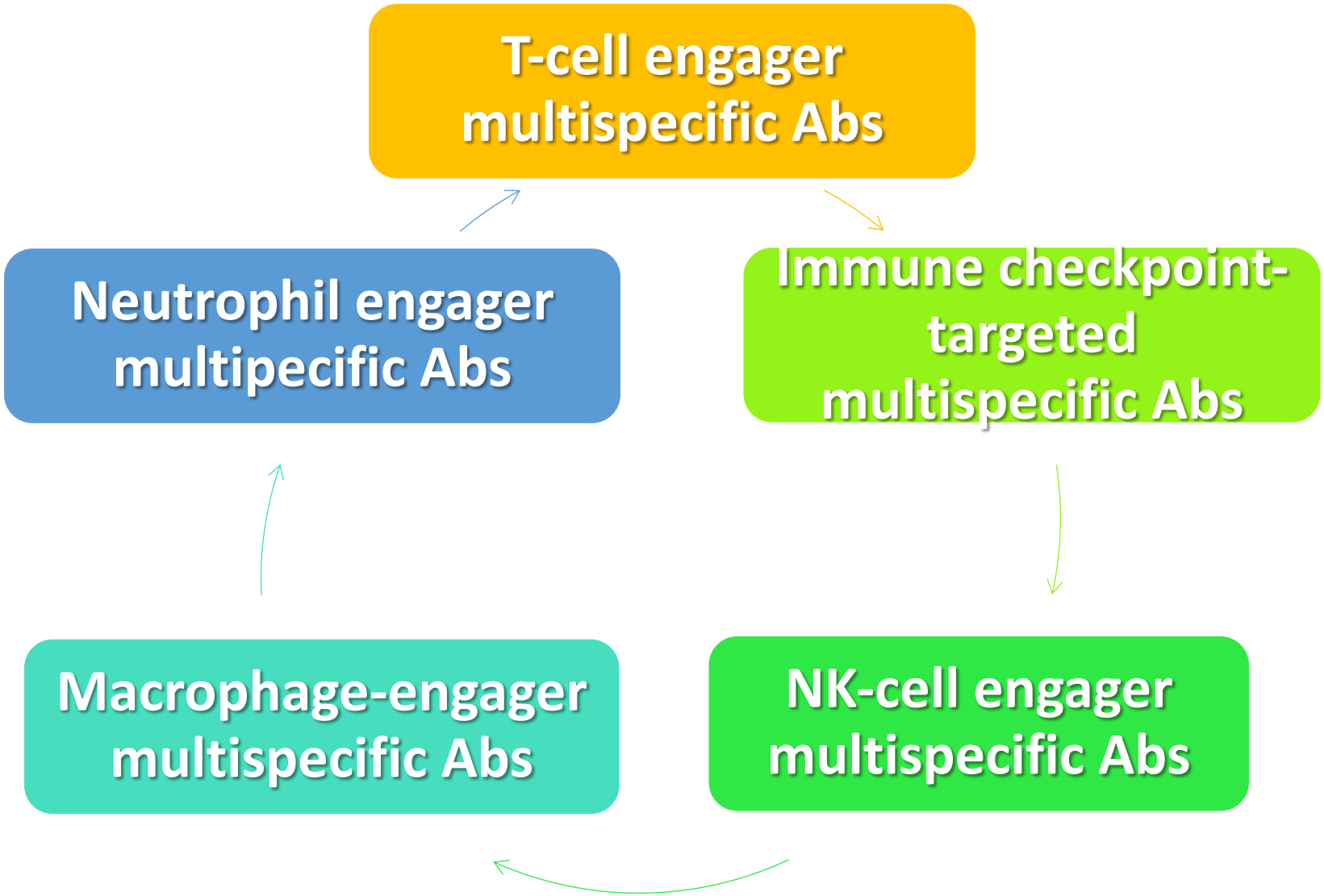


Irit Avivi Mazza et al. ASH 2023, P3099

Agenda

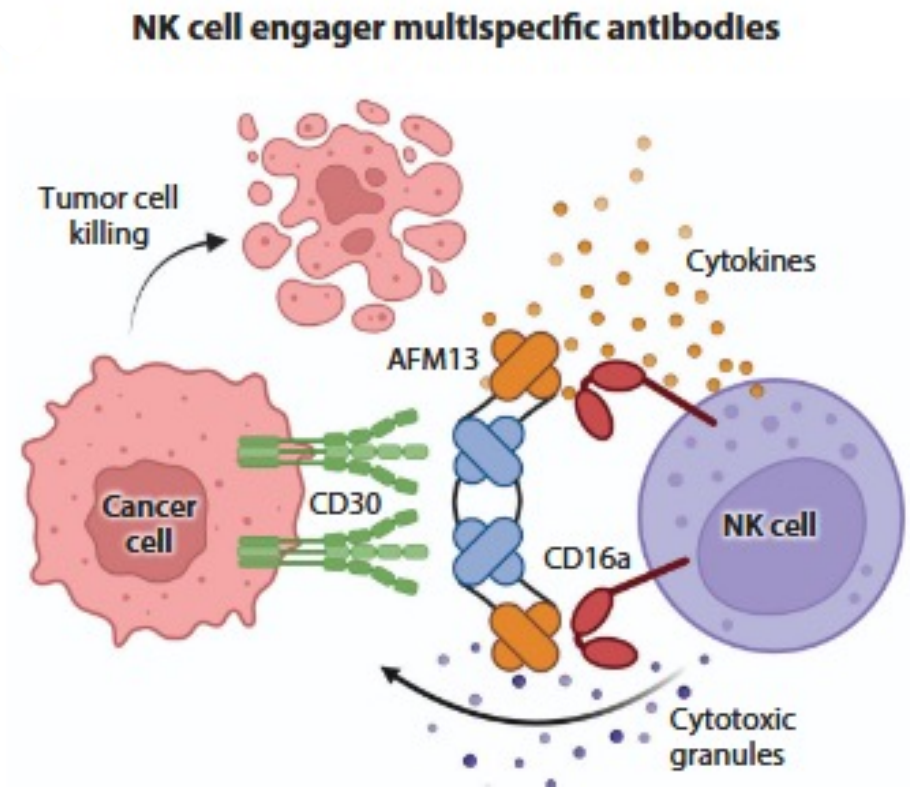
- From monospecific to bispecific and trispecific antibodies : structures and mechanisms of action
- Trispecific antibodies
- Immune-interfacing multispecific bispecific antibodies

Immune-interfacing multispecific antibodies



Immune-interfacing multispecific antibodies

NK-cell engager multispecific Abs



An NK cell–recruiting bispecific Ab, AFM13, which binds CD16a on NK cells to facilitate their destruction of CD30-expressing malignant cells.

Tapia-Galisteo A et al. J Hemato oncol, 2023

Redirecting NK cells to kill tumors

NK-cell engager multispecific Abs

>> To engage NK cells as cytolytic effector.

>> To reduce the Cytokine Release Syndrome (CRS)

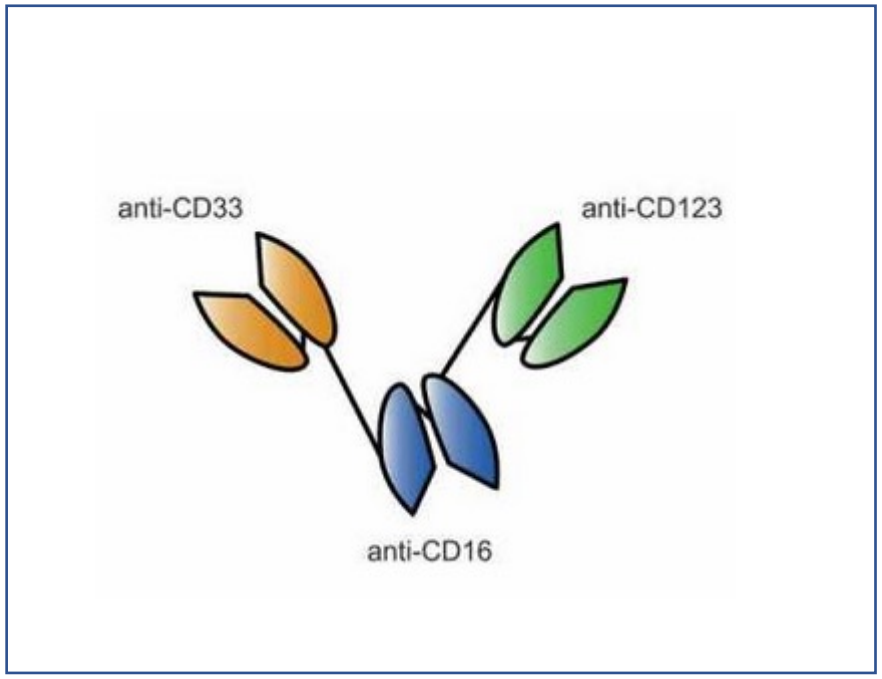
Multispecific NK cell engagers in clinical trials (all stages), as of July 2023

Drug name	Format	Specificity	Indication	Clinical trial	Phase	Sponsor
AFM13	TandAb	CD30/CD16a	HL, NHL	NCT04074746	1/2	
AFM28	IgG-like ROCK	CD123/CD16a	AML	NCT05817058	1	
LAVA-051	Gammabody	CD1d/NKT TCR	CLL, MM, AML	NCT04887259	1/2	
IPH6101/SAR443579	ANKET	CD123/CD16/NKp46	AML, MDS	NCT05086315	1/2	
IPH6401/SAR445514	ANKET	BCMA/CD16/NKp46	R/R MM	NCT05839626	1/2	
CC-92328/DF3001	TriNKET	BCMA/CD16/NKG2D	R/R MM	NCT04975399	1	
CC-96191/DF2001	TriNKET	CD33/CD16/KNG2D	R/R AML	NCT04789655	1	

Tapia-Galisteo et al. Journal of Hematology & Oncology 2023; 16:83

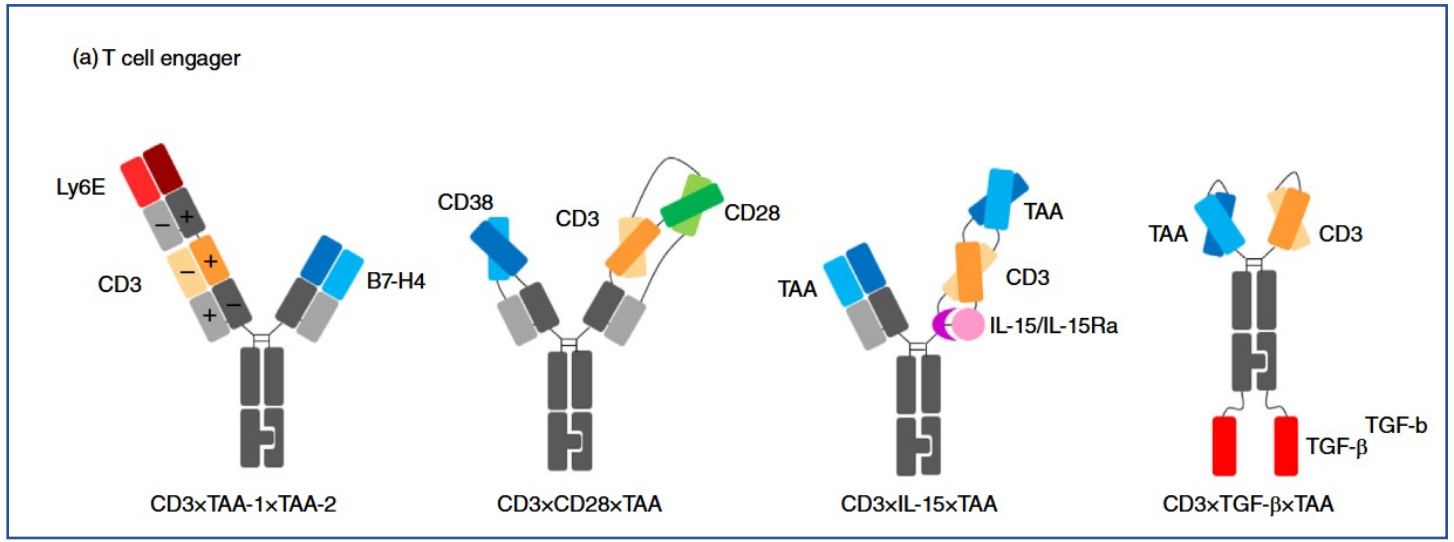
Dual-targeting triplebody 33-16-123 (SPM-2) to engage NK cells as cytolytic effector

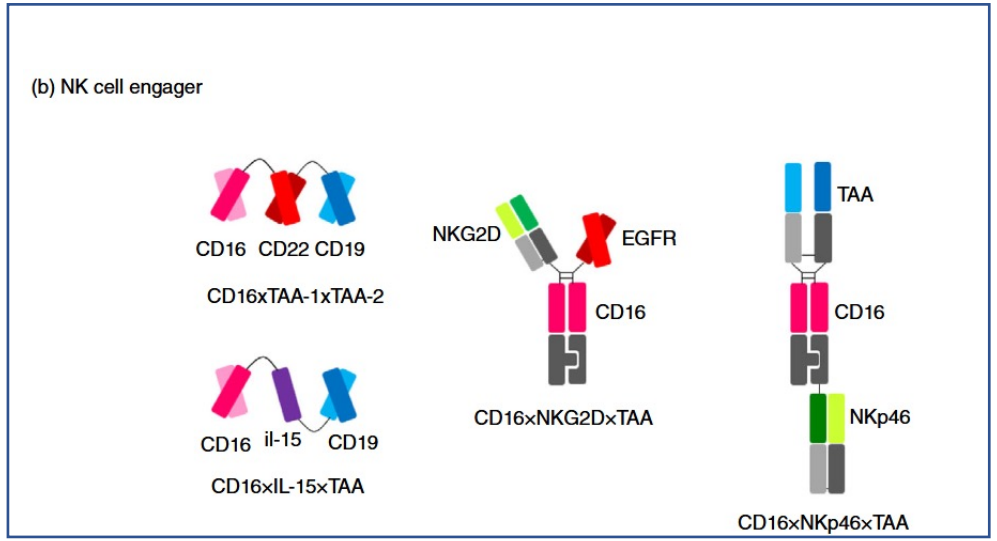
CD33 × CD16a × CD123 TsAb to engage NK cells as cytolytic effector

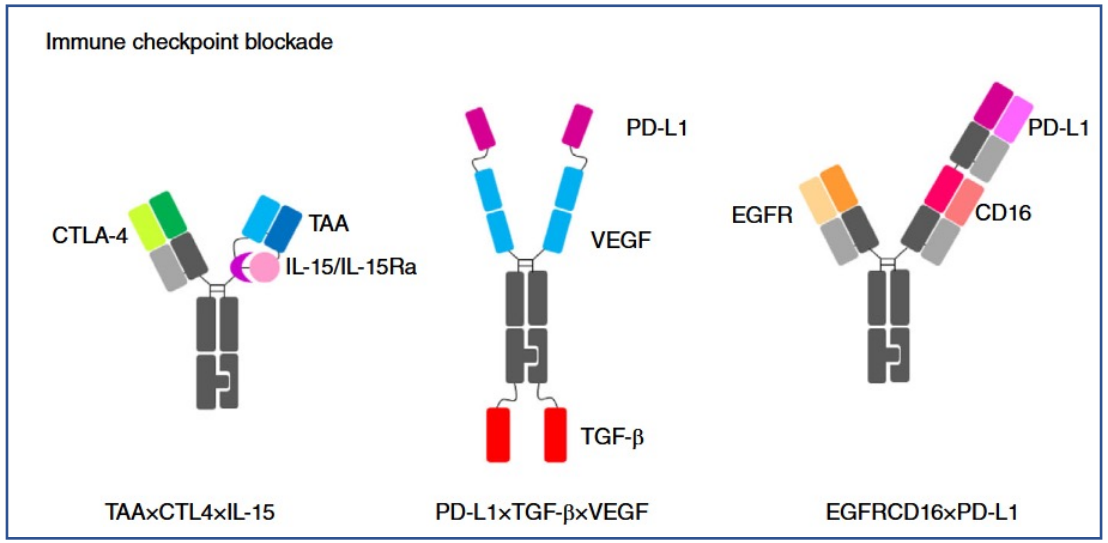


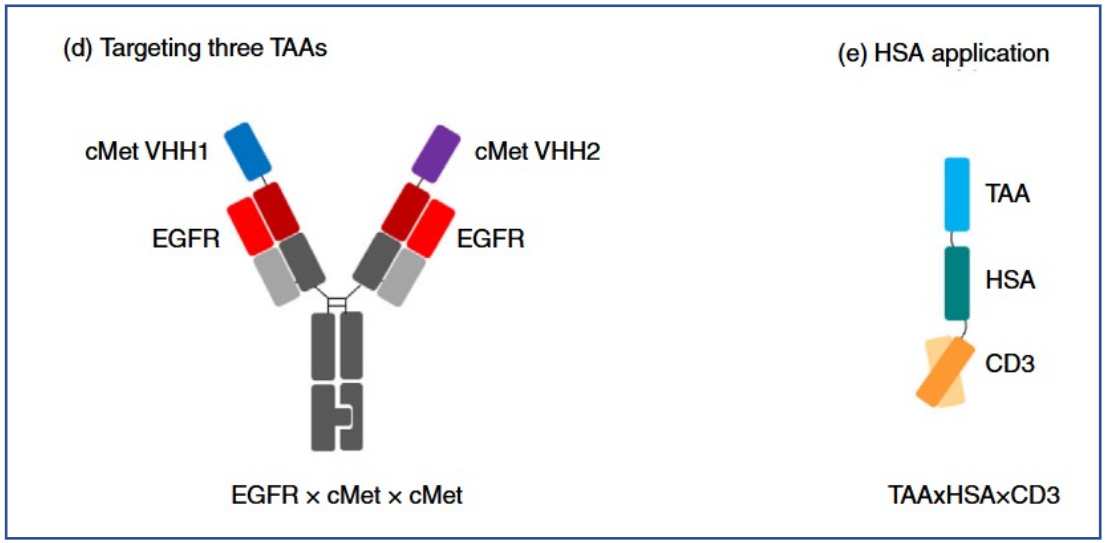
Braciak TA, Oncoimmunology 2018; 7(9):e1472195

Continuous optimization of target combinations, antibody forms, and sequences modification



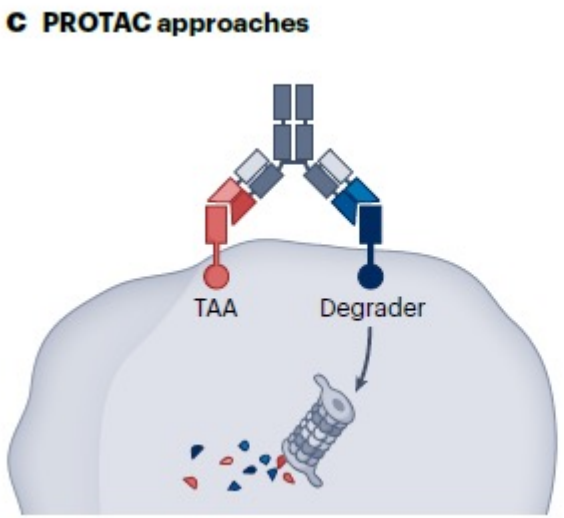




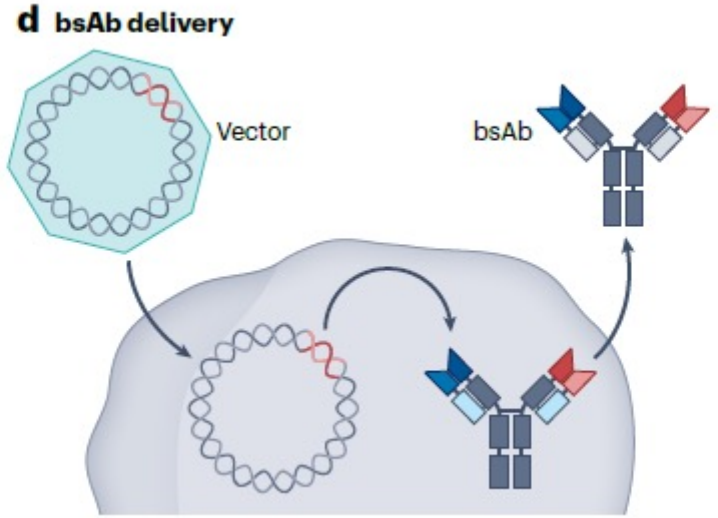


Yao Y, Hu Y, Wang F. Immunology. 2023;169:389–399

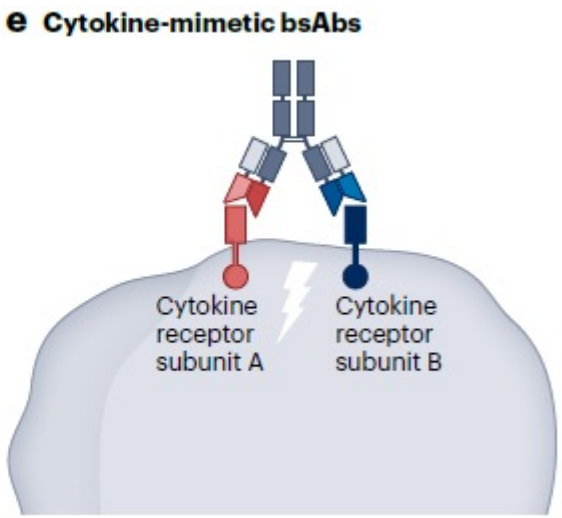
Emerging concepts



Proteolysis target chimera (PROTAC)
Bind a TAA and a degrader molecule



Deliber drugs

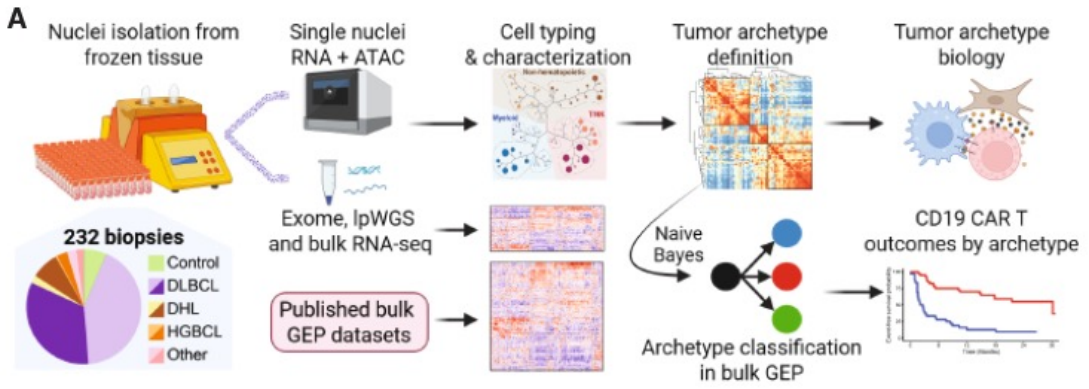


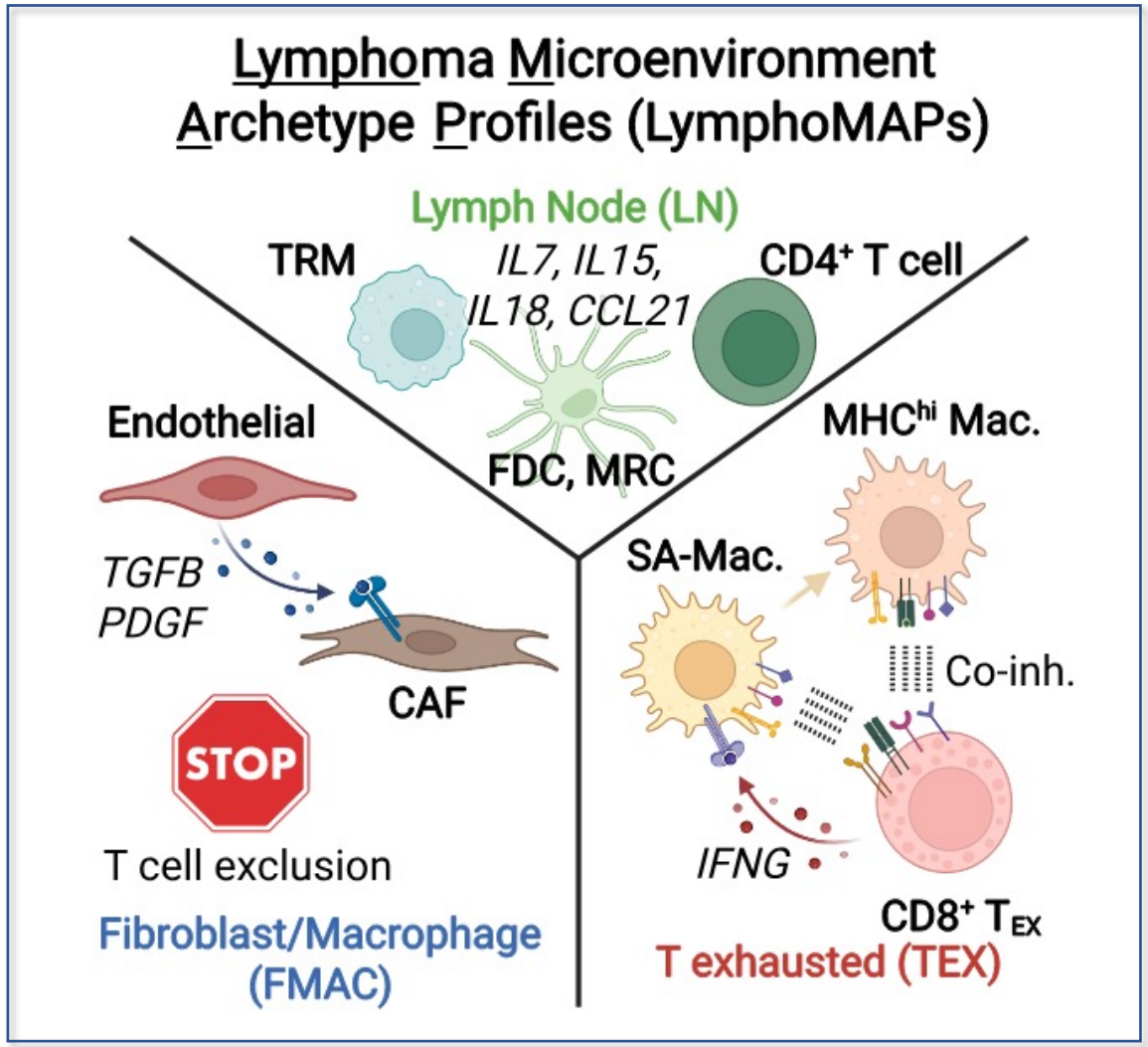
Trigger cytokine receptor pathways, mimicking cytokine action

Klein C et al [ ]) Nat Rev Drug Discovery 2024

LymphoMAPs : characterization of 3 lymphoma associated ImmunoProfiles

- **Single nucleus multiome profiling** of **non-malignant cell subsets** from **217 LBCLs**
- Discovery of **Lymphoma Microenvironment archetype profiles** (LymMAPs) : **Cell-cell communication pathways**
- Association with **outcome of patients treated with anti-CD19 CAR T-cells**

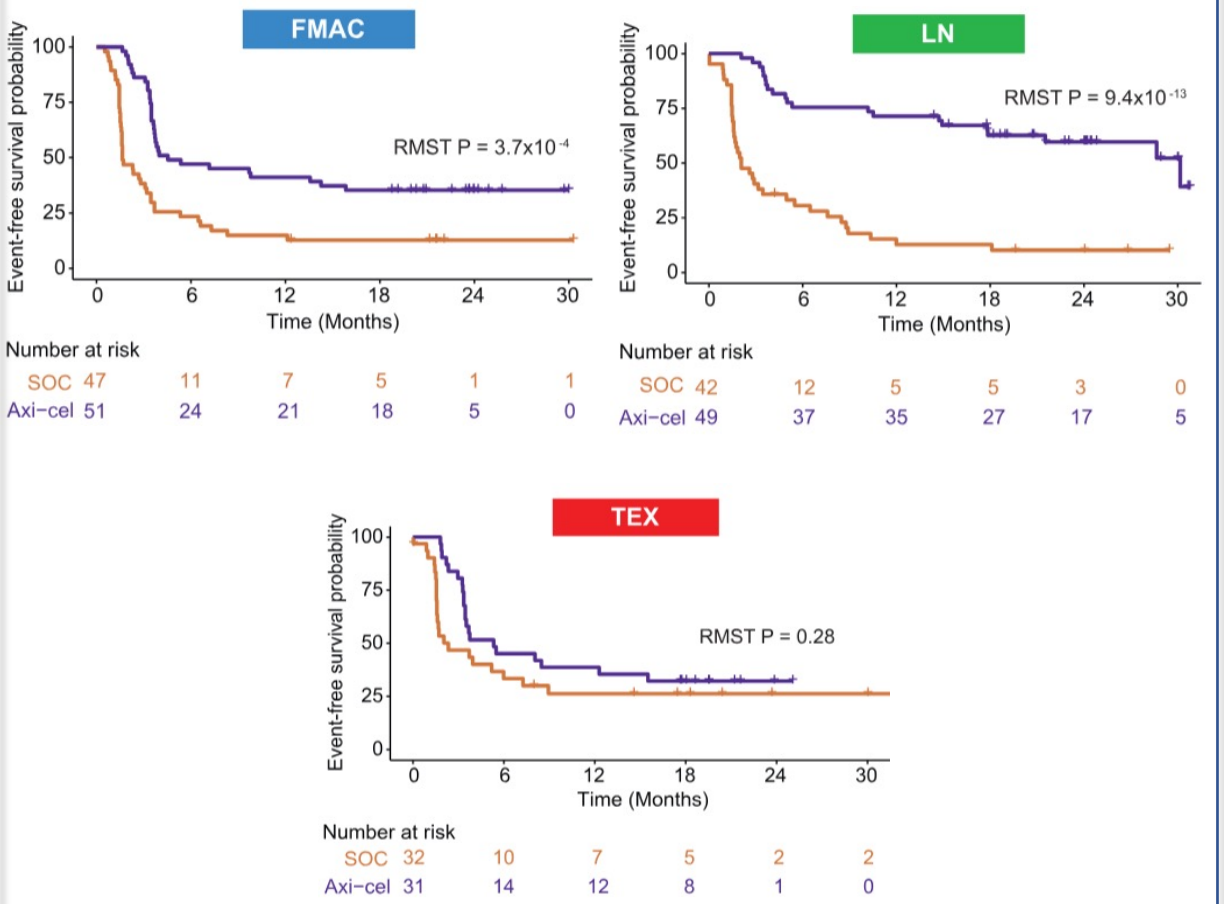




Li et al Plenary session ICML 2025; Cancer Cell 2025, 43, 1347–1364

Association between archetype profiles and response to first-line therapy and CART19

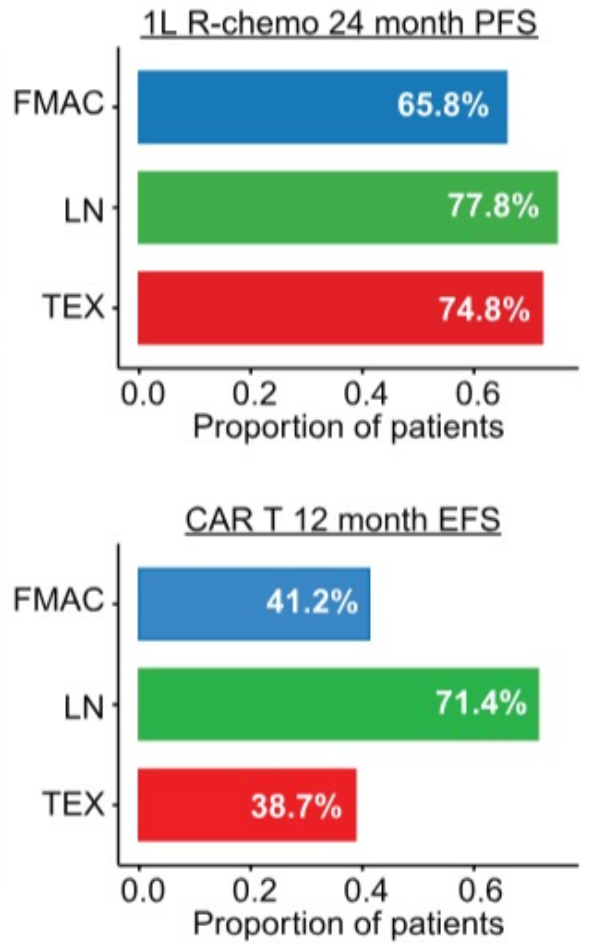
ZUMA7 :TEX > LN > FMAC



FMAC
- Enriched for DZsig+ mol. subtype
- Enriched for high-risk features
- More frequent in later LOT
- Inferior outcomes to 1L R-chemo

LN
- Enriched for GCB mol. subtype
- Low SUVmax
- More frequent in earlier LOT
- Best outcomes post CAR T

TEX
- Enriched for ABC mol. subtype
- No assoc. with high-risk features
- Consistent across LOT
- No sig. benefit from CAR T



Li et al Plenary session ICML 2025; Cancer Cell 2025, 43, 1347–1364

Conclusion

- **Technologies evolving allowing modulation of Abs constructs**
- **Bispecifics and Trispecific antibodies represent a flexible platform, offering a way to deliver precise combinations of immunomodulatory signals (for example, a co-stimulatory signal and a checkpoint blocker) specifically in the tumour microenvironment**
- **Several are in early clinical trials with encouraging preliminary data**

Thank you for you attention !

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- **Patients and their families**

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