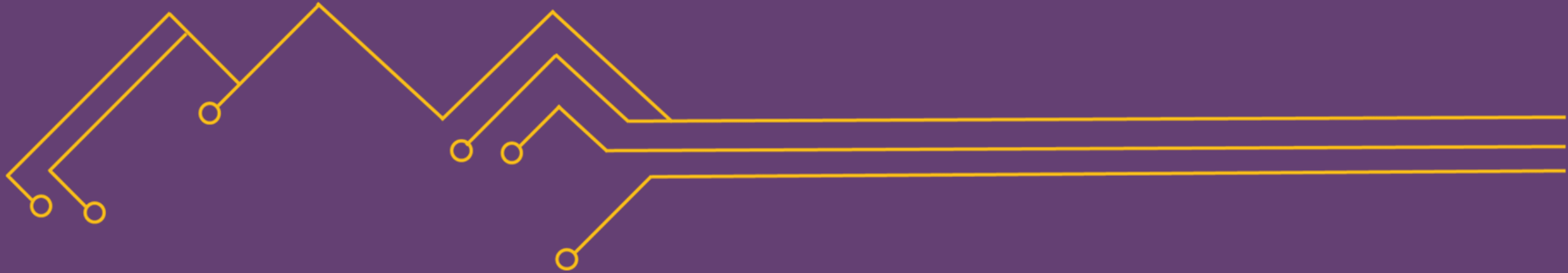




# CART

Leveraging biological complexity and logic gating to create novel CAR-T cell therapies



70% of cancers are **not treatable with targeted approaches.**

GoCART's **recognition dimer platform** unlocks safe targeting and changes this!

# Why CAR-T stops at blood cancers: no unique antigen in most tumors

Approved CAR-T therapy	Any indications outside advanced blood cancer?	Target	Type	Approval
Aucatzyl	No	CD19	Autologous	USA
Breyanzi	No	CD19	Autologous	USA & Europe
Kymriah	No	CD19	Autologous	USA & Europe
Tecartus	No	CD19	Autologous	USA & Europe
Yescarta	No	CD19	Autologous	USA & Europe
Abecma	No	BCMA	Autologous	USA & Europe
Carvykti	No	BCMA	Autologous	USA & Europe

- B-Cell cancers work because CD19/BCMA are uniquely overexpressed and expendable.
- Many tumors don't have such markers – healthy cells share the same antigens.
- Single-antigen CAR-T → off-tumor toxicity → UNUSABLE.
- Industry-wide bottleneck: Finding a single target that is **present on the tumor** cell and **absent from healthy** tissue is biologically impossible.

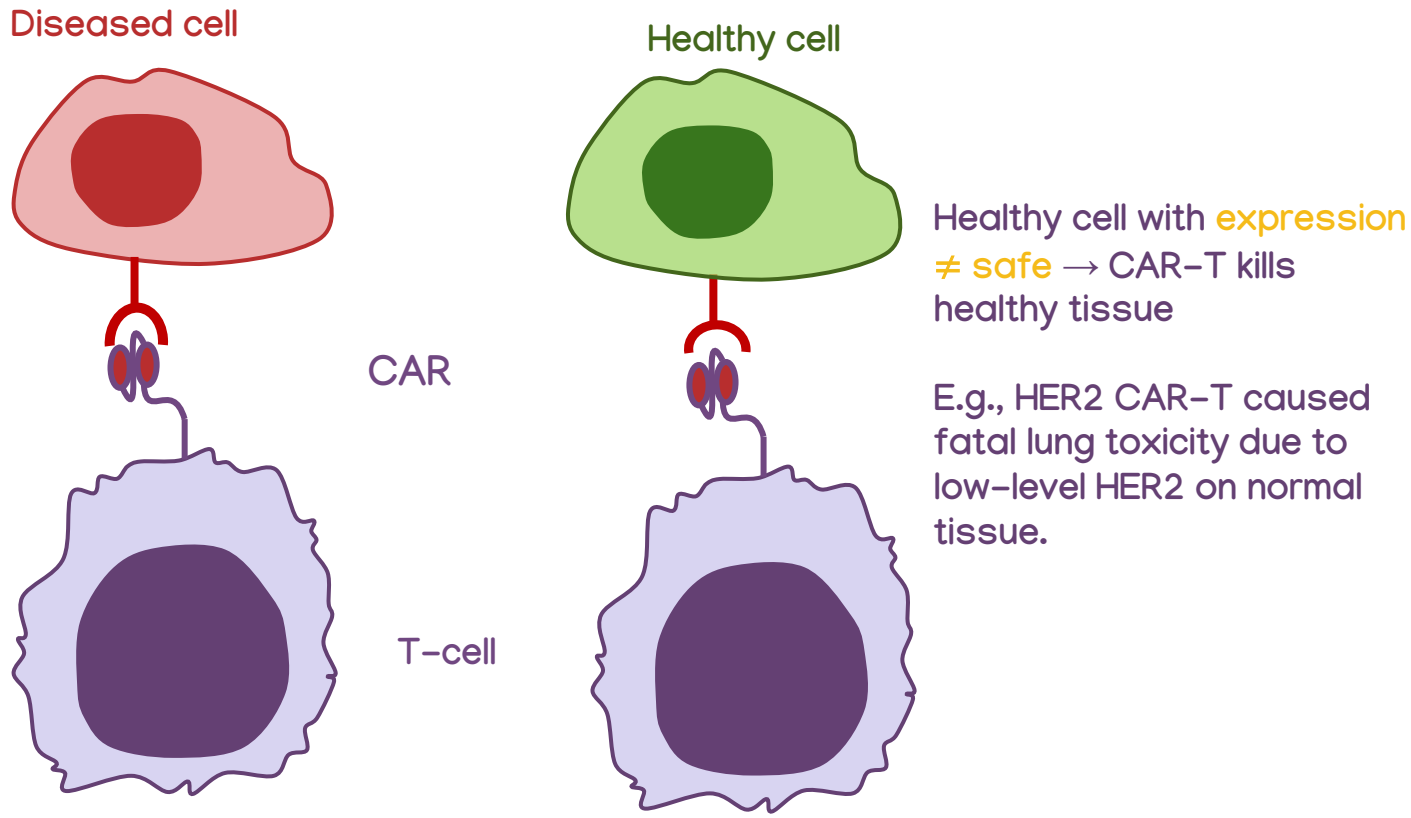
The field needs multi-antigen logic.

Yet only for B-cell tumors....

Sources: 1. FDA [AUCATZYL](#); 2. FDA [BREYANZI](#); 3. FDA [KYMRIAH](#); 4. FDA [TECARTUS](#); 5. FDA [YESCARTA](#); 6. FDA [ABECMA](#); 7. FDA [CARVYKTI](#)


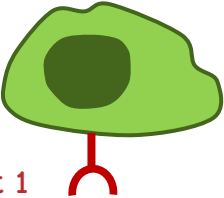

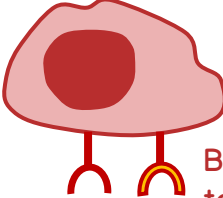


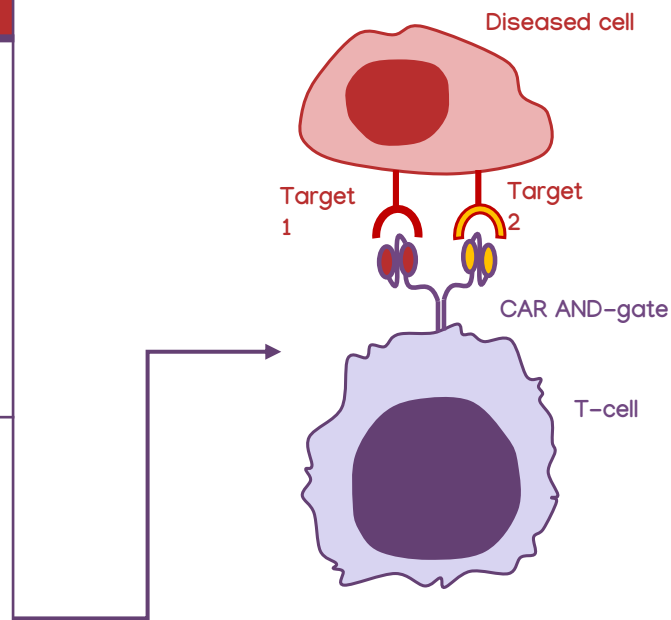
# Healthy tissues often express the same antigens as tumors triggering **deadly off-tumor toxicity**



- Most tumors do not present exclusive antigens – normal tissues express the same surface markers.
- Single-target CAR-T fires even at low antigen levels. This causes **off-tumor toxicity** leaving **70% of all tumors untreatable**.

# Two-antigen AND-gate enables safe & selective activation solving the specificity bottleneck

	No target 1	Target 1 present
No target 2	<p>Healthy cell, no CAR-T binding</p>  <p>No target</p>	<p>Healthy cell, no CAR-T binding</p>  <p>Target 1</p>
Target 2 present	<p>Healthy cell, no CAR-T binding</p>  <p>Target 2</p>	<p>Diseased cell, CAR-T binds</p>  <p>Both targets</p>



The field needs a synapse-level pattern recognizer – a CAR-T cell that verifies A+B on the same cell before killing.

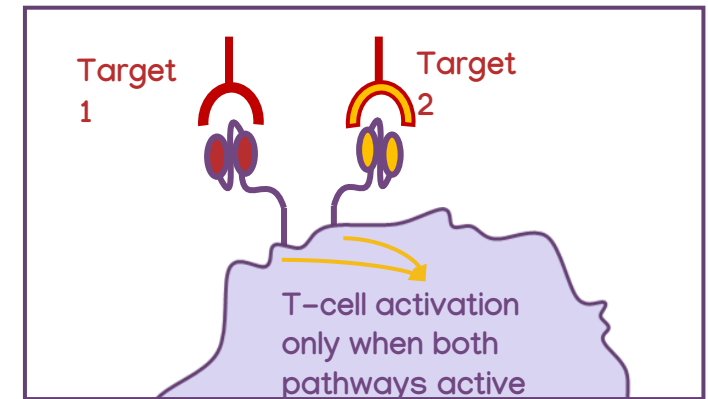
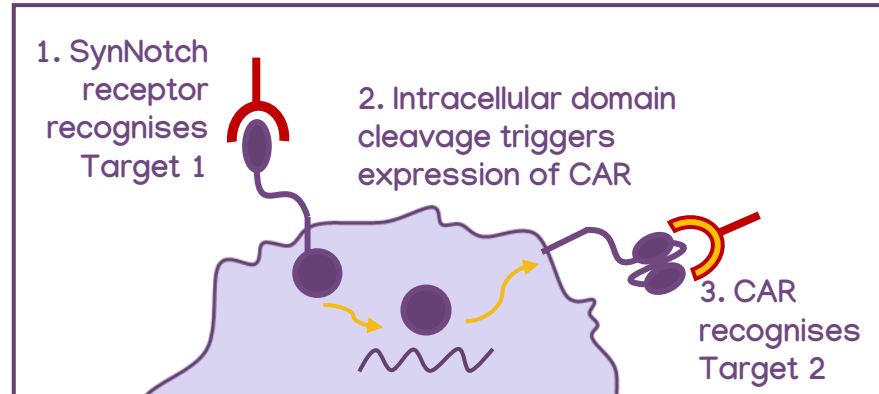
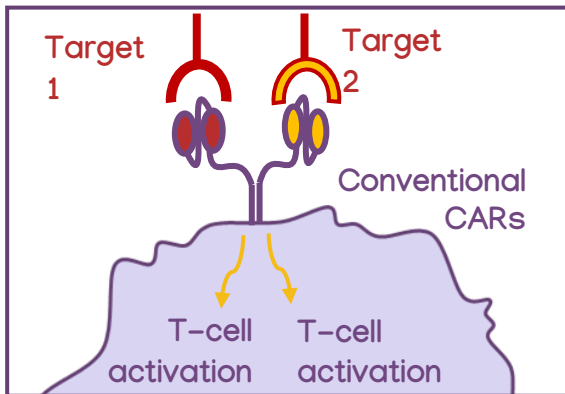
Sources: 1. Hamieh et al. Cancer Discov (2023); 2. Tousley et al. Nature (2023)

# Why current Boolean CAR designs still cannot deliver true specificity

Fails because activation happens when either antigen is present – **NOT same-cell coincidence sensing**.

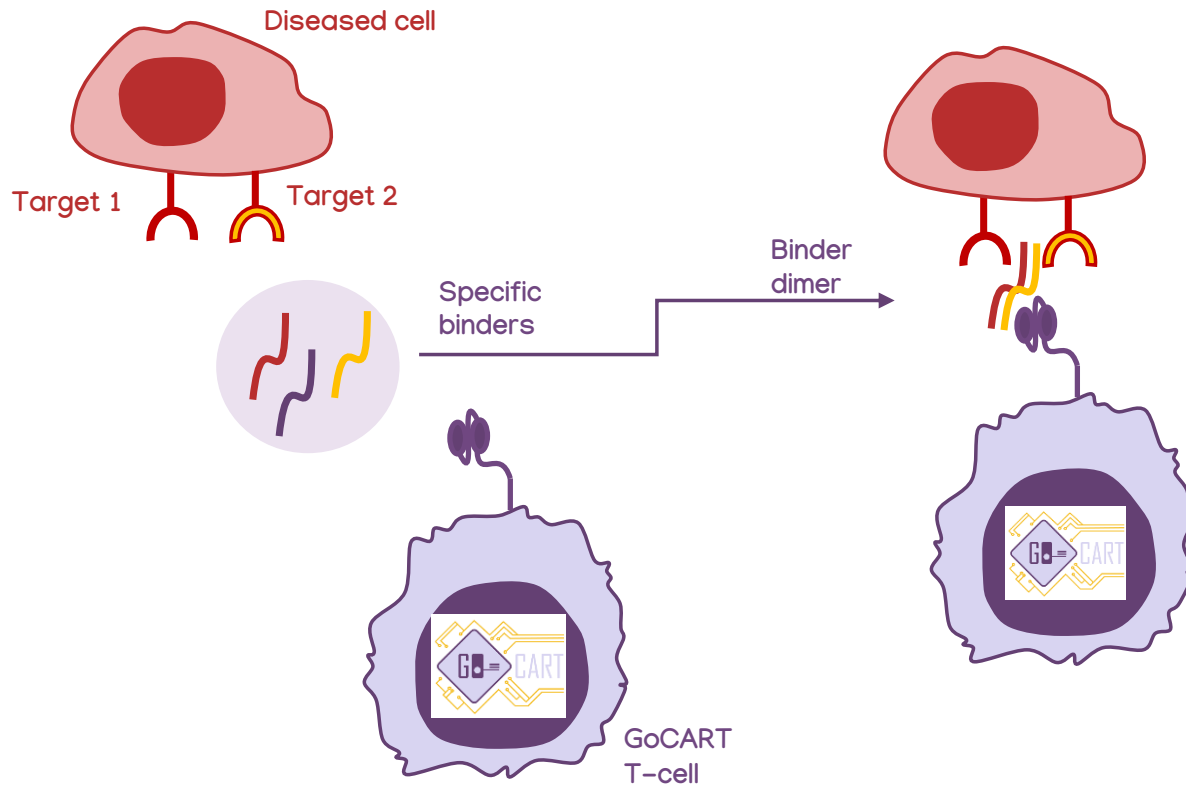
Sequential logic, **not spatial logic** – T1 and T2 can be on different cells. No same-cell discrimination → **unsafe** for groundbreaking new targets.

True AND-gate but **low sensitivity & rigid design** – cannot optimize affinity per antigen. **Poor performance** at physiological antigen densities.



None of the existing dual-sensing designs can reliably detect *two antigens on the same cell at physiological densities* – the essential requirement for safe CAR-T therapy.

# GoCART Recognition Dimer



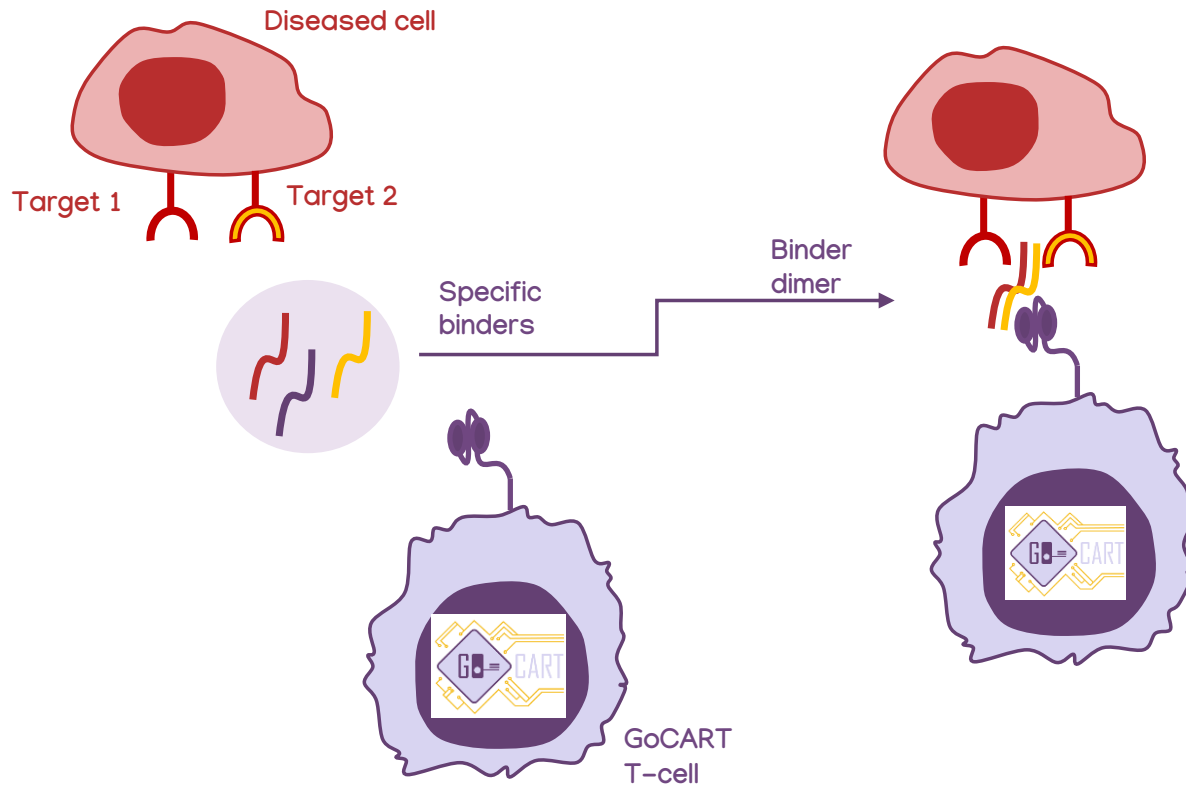
## *How does it work?*

Two strong binders latch onto A and B → proximity forces dimerization → signaling motif is exposed → CAR activates only when both antigens are on the same cell.

## *Why is it sensitive?*

GoCART can use high affinity Binders because a **Binder dimer** is necessary to engage the cell. The AND-Gate works without making compromises on **affinity** or losing intracellular **signal enhancement**.

# GoCART Recognition Dimer



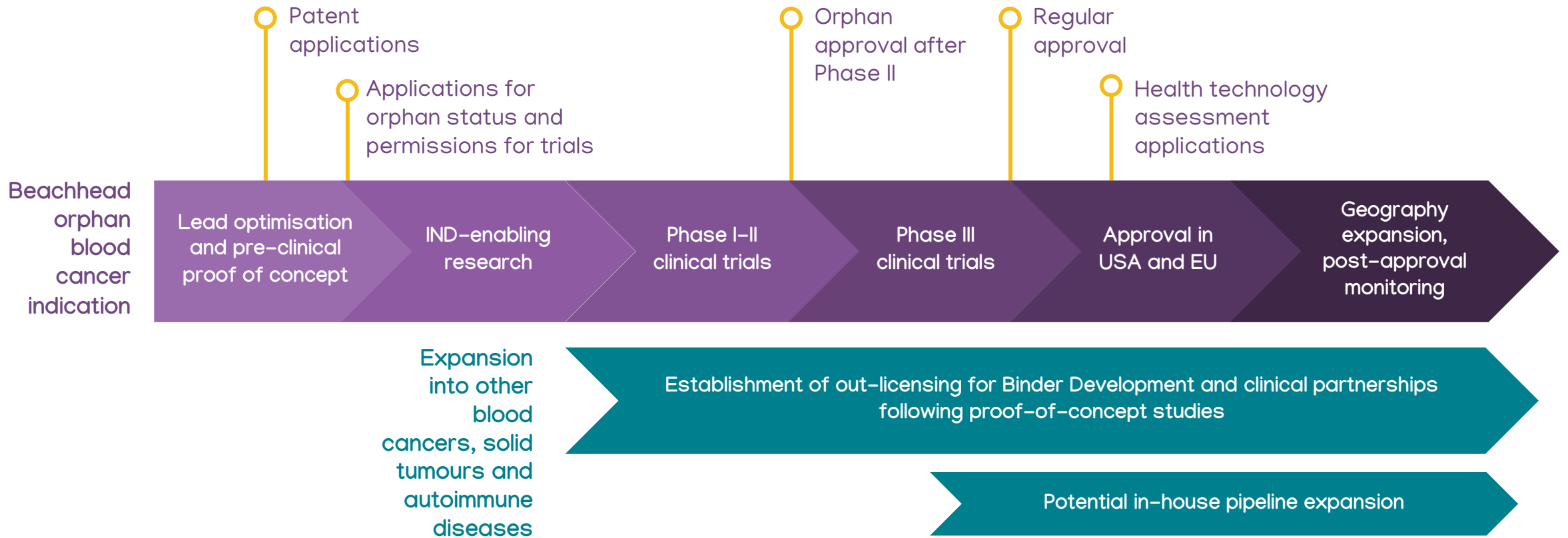
## *What makes it versatile?*

The binder modules can be swapped or tuned, allowing the same core design to be applied to many different antigen pairs.

## *Why is it better than existing AND-gates?*

Unlike SynNotch / Split CARs (no real AND-Gate) or LINK CARs (complex, bad sensitivity), GoCART directly triggers T-cell signaling with a **single, compact receptor** requiring **true A&B co-expression** and Binder engagement.

# Clinical & Commercial Strategy

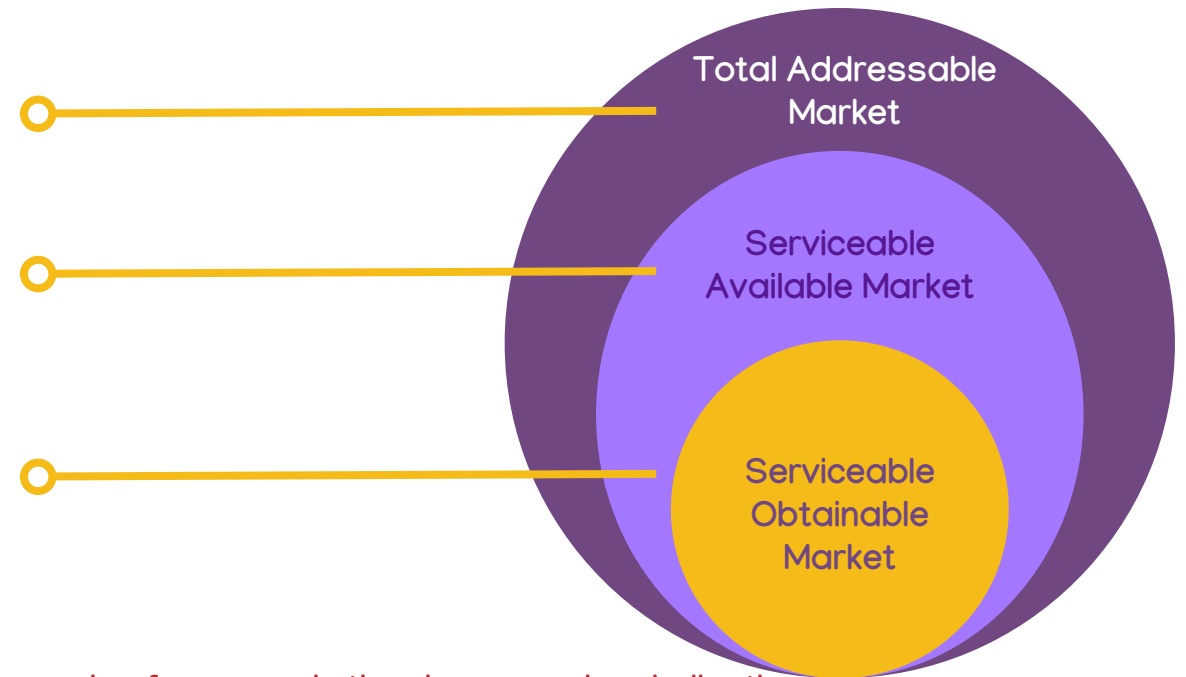


# CAR-T Market Size Is Estimated to Reach up to ~130bn USD in 2033, Creating a Large Opportunity for higher specificity

Total potential CAR-T patients in 2032  
(includes possible new indications):  
**2 million**

Short term SAM: Orphan blood cancers with  
dual-antigen co-expression  
**5,000 patients/year (US+EU)**

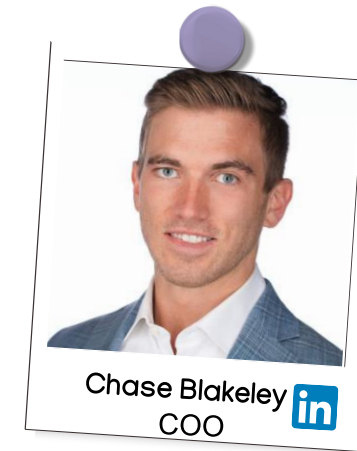
SOM (Market Year 1): One manufacturing site,  
20% market penetration with superior safety profile  
**1,000 patients per year**



Final ranges depend on the selected antigen pair and validated co-expression frequency in the chosen orphan indication.

We propose a B2B2C approach, providing CAR-T therapy access through licensing and strategic healthcare partnerships after clinical proof of concept in a beachhead indication

# A team combining CAR-T engineering, translational research, and operational execution



- Doctoral research in Translational Tumor Immunology at Max Delbrück Center (MDC Berlin) and InBiCA Cádiz
- Hands-on experience in CAR-T design, T-cell engineering and antigen recognition logic
- Medical training at Charité, with understanding of CAR-T toxicity, CRS and clinical challenges
- Leads Go-CART's scientific strategy, platform architecture, validation roadmap and KOL alignment
- Background in cancer biology & molecular biotechnology
- Led scientific outreach and partnerships as Head of iGEM NU-Kazakhstan
- Collaborated with Roche, AstraZeneca, Pfizer, and academic institutions
- Built public health & education initiatives (Breast Cancer Awareness Foundation)
- Responsible for strategic partnerships, scientific communication, and stakeholder engagement
- Experience managing complex operational workflows and international partnerships (Oracle, Tesla)
- Specializes in resource allocation, vendor management, CDMO coordination
- Oversees financing processes and operational execution for GoCART
- Leads the funding operation

# Advisory Board



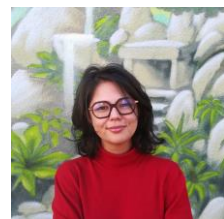
**Dr. Leonardo Chicaybam, Galapagos**

- Associate director – Translational science at Galapagos
- Accomplished scientist with more than 10 years of experience in immuno-oncology R&D and drug development
- Strong expertise in CAR-T cell therapy



**Prof. Dr. Curro Garcia-Cozar, University of Cádiz**

- Professor of Immunology, Instituto de Investigación Biomédica de Cádiz (INIBICA)
- 20+ years researching T-cell activation, immune tolerance, and tumor immunology
- Provides Laboratory Infrastructure and directly advises GoCART on experimental setup, immune-risk assessment, antigen biology, and clinical translation pathways



**Dr. Elisabete Silva**

- Experience across diagnostics, oncology R&D and life-science business development
- Expertise in early-stage asset positioning and partnership structuring
- Supports GoCART on BD strategy, partner discovery, and market-entry pathways



**Dr. Jan Pille, UGA Biopharma, Max Delbrück Center Berlin**

- Former Group Leader, Helmholtz Innovation Lab “MDCell”
- Extensive background in CAR-T engineering and genetic modification workflows
- Expert in tech transfer & GMP-scale process development
- Supports GoCART on manufacturing readiness, process scalability and CDMO alignment



**Yusuf Demir - R&D Portfolio and Operations Professional**

- R&D Portfolio Manager at GARDP (London), overseeing global antibiotic development operations.
- Former strategic leader at Immunocore, managing high-impact research programs and multimillion-pound partnerships.
- Expertise in operations strategy, external collaborations, and scaling biotech programs.
- Board Member at Mentor Wise, supporting innovation and youth development in the UK.
- Holds advanced degrees in Cancer & Molecular Pathology, Innovation & Entrepreneurship, and Molecular Biology & Genetics.



# Our present business, Binder R&D partnerships

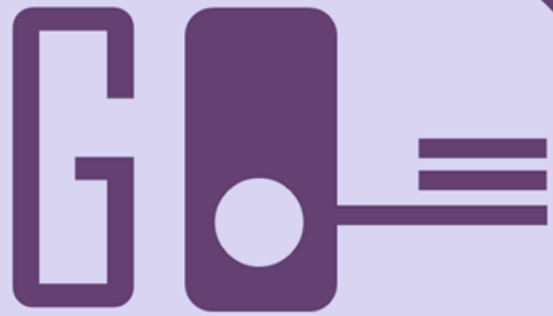


Partner	Role in GoCART R&D
WuXi Biologics	Wet-lab binder screening, binder optimization, recombinant expression
Oncera / ImmunoChem	AI-driven antigen modelling, epitope prediction, Antigen Binding Domain generation, tumor-avatar simulations

# What does \$150k buy?

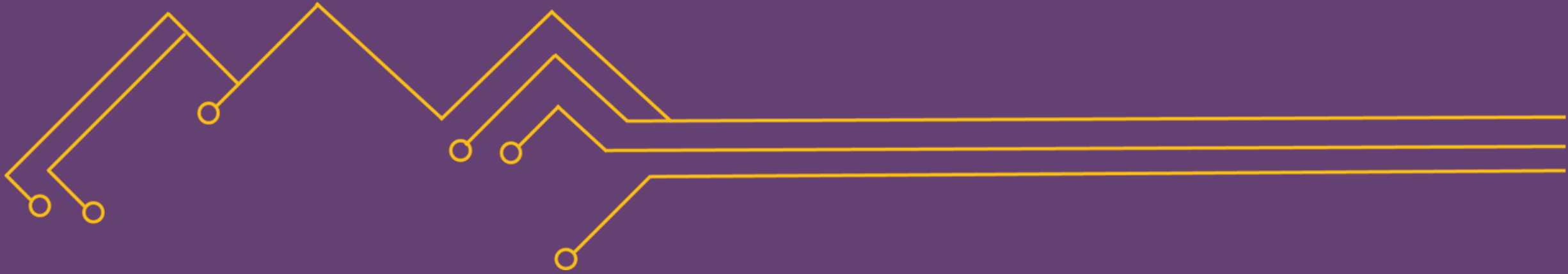
## Proposed GANTT Chart For the First Year

Year	1											
Work Package \ Month	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.
0. Co-Development of candidate sequences and an auto-predictor engine	█	█										
1. Establishment of the Transfection - FACS-FRET experimental setup	█	█										
2. candidate testing pipeline	█	█	█	█	█	█						
3. Generation of retroviral vector with CAR trunk					█	█	█					
4. Validation of System cytotoxicity in cell co-culture							█	█	█	█	█	
5. Application and preparation of in-vivo (NSG) tumor eradication											█	█



# GART

Contact:  
[henry.erdlei@charite.de](mailto:henry.erdlei@charite.de)



# Appendix

# All seven CAR-T therapies currently approved by FDA are anti-CD19 or BCMA

Name	Indication(s)	Any indications outside advanced blood cancer?	Target	Type	Status
Aucatzyl (obecabtagene autoleucel) <sup>1</sup>	Adult r/r B-cell precursor ALL	No	CD19	Autologous	FDA approved
Breyanzi (lisocabtagene maraleucel) <sup>2</sup>	<ul style="list-style-type: none"> <li>• Adult r/r LBCL</li> <li>• Adult r/r CLL, SLL, FL (under accelerated approval)</li> <li>• Adult r/r MCL</li> </ul>	No	CD19	Autologous	FDA & EMA approved
Kymriah (tisagenlecleucel) <sup>3</sup>	<ul style="list-style-type: none"> <li>• R/r B-cell precursor ALL, age ≤25 years</li> <li>• Adult r/r LBCL</li> <li>• Adult r/r FL (under accelerated approval)</li> </ul>	No	CD19	Autologous	FDA & EMA approved
Tecartus (brexucabtagene autoleucel) <sup>4</sup>	<ul style="list-style-type: none"> <li>• Adult r/r MCL (under accelerated approval)</li> <li>• Adult r/r B-cell precursor ALL</li> </ul>	No	CD19	Autologous	FDA & EMA approved
Yescarta (axicabtagene ciloleucel) <sup>5</sup>	<ul style="list-style-type: none"> <li>• Adult r/r LBCL</li> <li>• Adult r/r FL (under accelerated approval)</li> </ul>	No	CD19	Autologous	FDA & EMA approved
Abecma (idecabtagene vicleucel) <sup>6</sup>	Adult r/r multiple myeloma	No	BCMA	Autologous	FDA & EMA approved
Carvykti (ciltacabtagene autoleucel) <sup>7</sup>	Adult r/r multiple myeloma	No	BCMA	Autologous	FDA & EMA approved

Clinical trial pipeline includes other targets (e.g. Claudin18.2, CD30, GPRC5D) and other types of therapy (e.g. allogeneic, double-targeting), but the number of trials is relatively small, and the overall diversity of novel CAR-Ts in development is low

Abbreviations: ALL: acute lymphoblastic leukaemia; CLL: chronic lymphocytic leukaemia; FDA: Food and Drug Administration; EMA: European Medicines Agency; FL: follicular lymphoma; ML: mantle cell lymphoma; R/r: relapsed or refractory; SLL: small lymphocytic lymphoma. Sources: 1. FDA [AUCATZYL](#); 2. FDA [BREYANZI](#); 3. FDA [KYMRIAH](#); 4. FDA [TECARTUS](#); 5. FDA [YESCARTA](#); 6. FDA [ABECMA](#); 7. FDA [CARVYKTI](#)



# Strategic & Technical FAQ

- **How does adding a soluble binder layer affect the development timeline?** - The complexity is offset by existing infrastructure. The lab pipeline already handles vector design, CAR expression, and functional testing for similar systems (Zip-CARs<sup>1</sup>). Only the FACS-FRET Assay is a new experimental element. This allows us to move at established speeds while building a more sophisticated architecture.
- **What is the manufacturing strategy for clinical scale?** - We are an asset-light organization. We use established production partners like for Lentiviral vector production. We outsource design of Antigen Binding Domains and testing to specialized Antibody partners and interested commercial and institutional partners for a commission.<sup>2</sup> This removes early capital expenditure on manufacturing and at the same time grows the value of our platform through an increased number of therapeutic options with every new combination.



# Strategic & Technical FAQ

- **Why lead with Multiple Myeloma (MM) in a ‘crowded’ market?** - Incumbents rely heavily on BCMA, which is subject to downregulation and immune-escape and has yet failed to deliver satisfactory results regarding sustained complete remissions. Our architecture targets CD56<sup>1</sup> and CD38<sup>2</sup>. These targets are harder for tumors to downregulate. MM provides a pragmatic beachhead with established models, but the platform is easily adaptable to our second Beachhead Indication AML (CD56<sup>3</sup> + CD33<sup>4</sup>) where no approved CAR-T exists.
- **How does GoCART's toxicity management differ from current toxicity management solutions without “AND”-Gate?** - Current solutions choose lower binder affinities to reduce healthy tissue activation to “bearable” levels. This compromises potency and increases the risk of relapse. Our extracellular logic gate allows for high-affinity binders. We protect healthy tissue through physical thermodynamic thresholds. Toxicity can be tuned via binder dosage without weakening the cytotoxic punch. Namely, this Solution is unfeasible for ‘undruggable’ Antigens like CD56, leaving many indications without a realistic targeted Immunotherapy option.



# Strategic & Technical FAQ

- **Is \$150k sufficient to reach a critical milestone?** - The \$150k is a lean Seed Round designed to reach a specific inflection point: in-vitro proof of concept. This includes finalizing the in-silico pipeline, validating the binder-binder interface, and filing initial patents. This data package de-risks the project to trigger a \$5M Series A for animal studies and IND-enabling work.
- **What is the primary competitive moat?** - Most competitors are racing to optimize binders for the same few targets. We are building an "Operating System" that makes undruggable antigens usable and supply it to third party ABD-Developers. We provide the logic gated cell platform that allows for high-affinity targeting of previously 'undruggable' antigens while protecting essential tissues. In a gold rush analogy: We do not dig the next hole, we provide pickaxes.