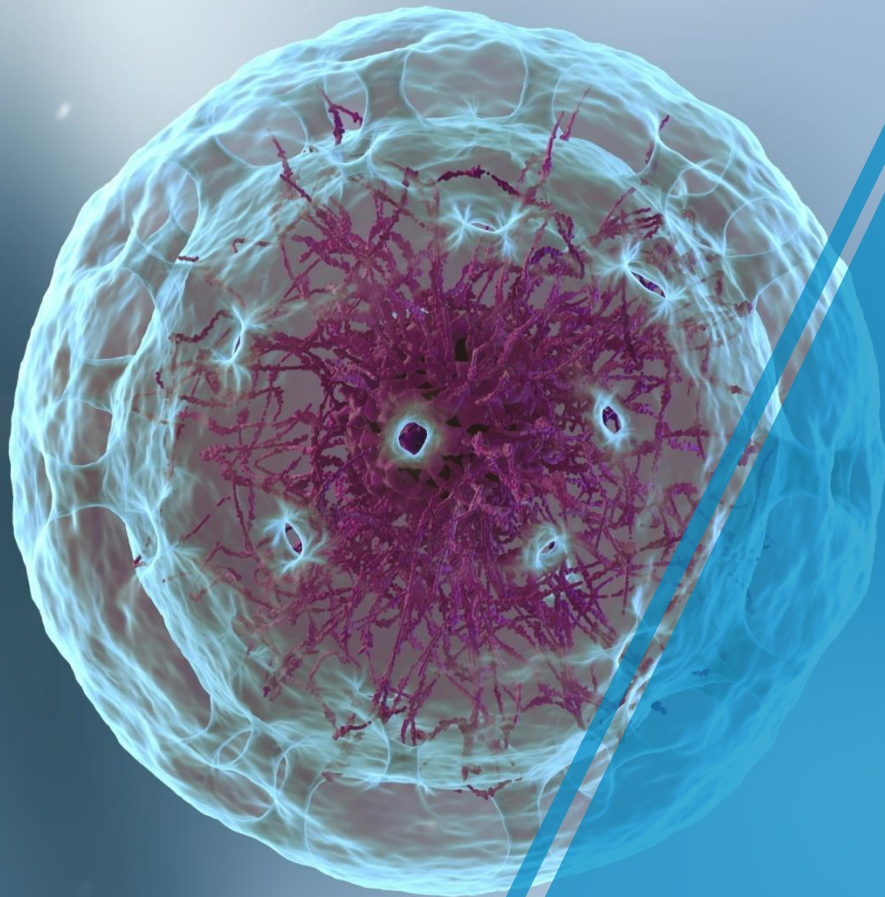




**Creating next-gen cell therapies
that are controllable, flexible & adaptable**

Prescient Therapeutics Limited (ASX: PTX)

Cell & Gene Meeting on the Mesa

October 2021

Disclaimer and Safe Harbor



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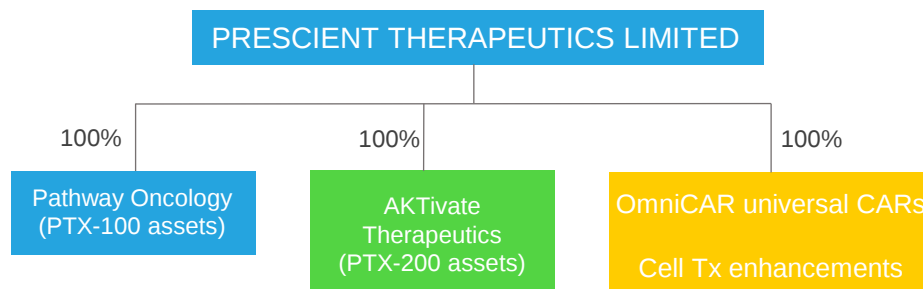
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OVERVIEW

- HQ in Melbourne, Australia, with activities in Australia and US
- Est 2014 with assets from Yale (PTX-100) and Moffitt/USF (PTX-200); and UPenn/Oxford (OmniCAR) in 2020
- Programs in US & Australia
- Listed on ASX, with wholly owned private subsidiaries



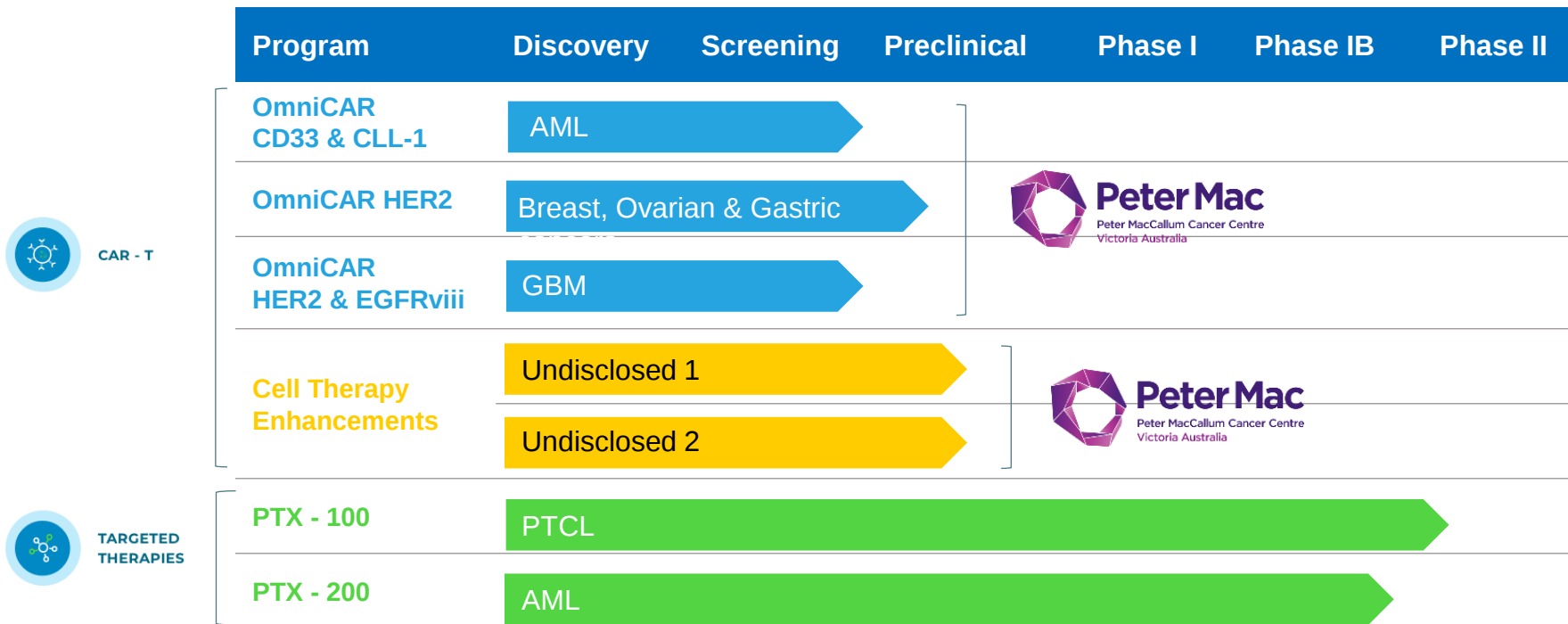
METRICS

ASX Ticker	PTX
Total Issued Capital	643 M shares
Listed Options	93.4 M
Unlisted Options	12.1 M
Share Price ¹	A\$0.27 (US\$0.20)
Market Capitalisation¹	A\$174 M (US\$127 M)
Market Cap fully diluted¹	A\$202 M (US\$148 M)
Cash Position²	A\$16 M (US\$12 M)
Top 20 Own	17%

1 - AS AT 17 SEPT 2021

2 - AT 30 JUNE 2021

Innovative Pipeline in Personalised Medicine

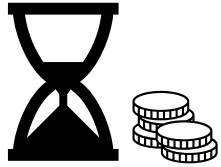




OmniCAR

Universal, Next Generation CAR-T

Key Challenges Confronting the field of CAR-T



Time and Cost
of delivering treatment



Targets

Finding targets that work;
Antigen heterogeneity - esp. in solid tumours



Safety

CAR-T can have serious
safety concerns



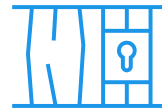
Exhaustion

Persistent stimulation of CAR-
Immune cells leads to exhaustion



No Control

Clinicians have no control
of cells post infusion



Escape

Antigen loss leads to relapse

OmniCAR Universal Immune Receptor Platform



- Pre-clinical **modularised** universal immune receptor (UIR) platform
- Potential best-in class UIR
- Based on multi-disciplinary technology licensed from **Penn**
- Only UIR system with post-translational covalent binding
- Unique, powerful and flexible
 - **Controllable activity**
 - **Flexible antigen targeting**



Co-inventors

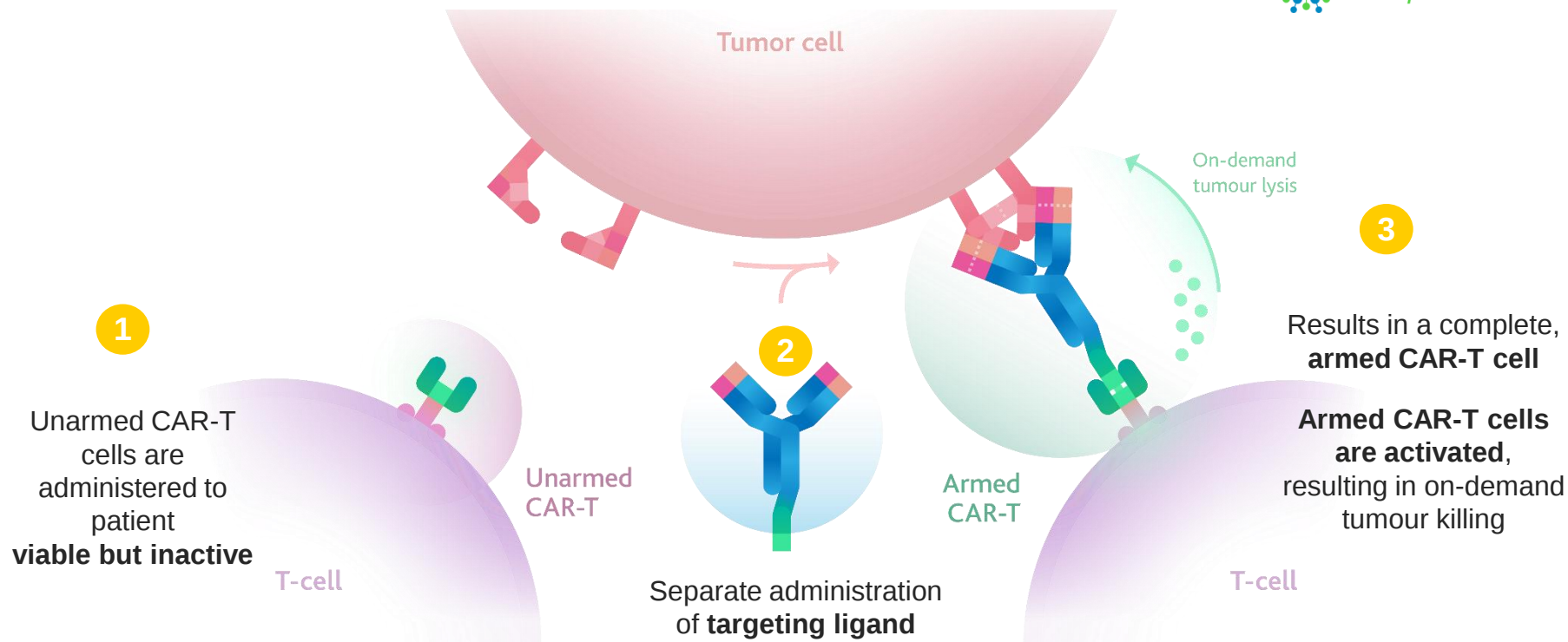


Associate Professor
Daniel J. Powell, Jr



Professor
Andrew Tsourkas

How OmniCAR works



CAR-T cell activity is **now controllable**

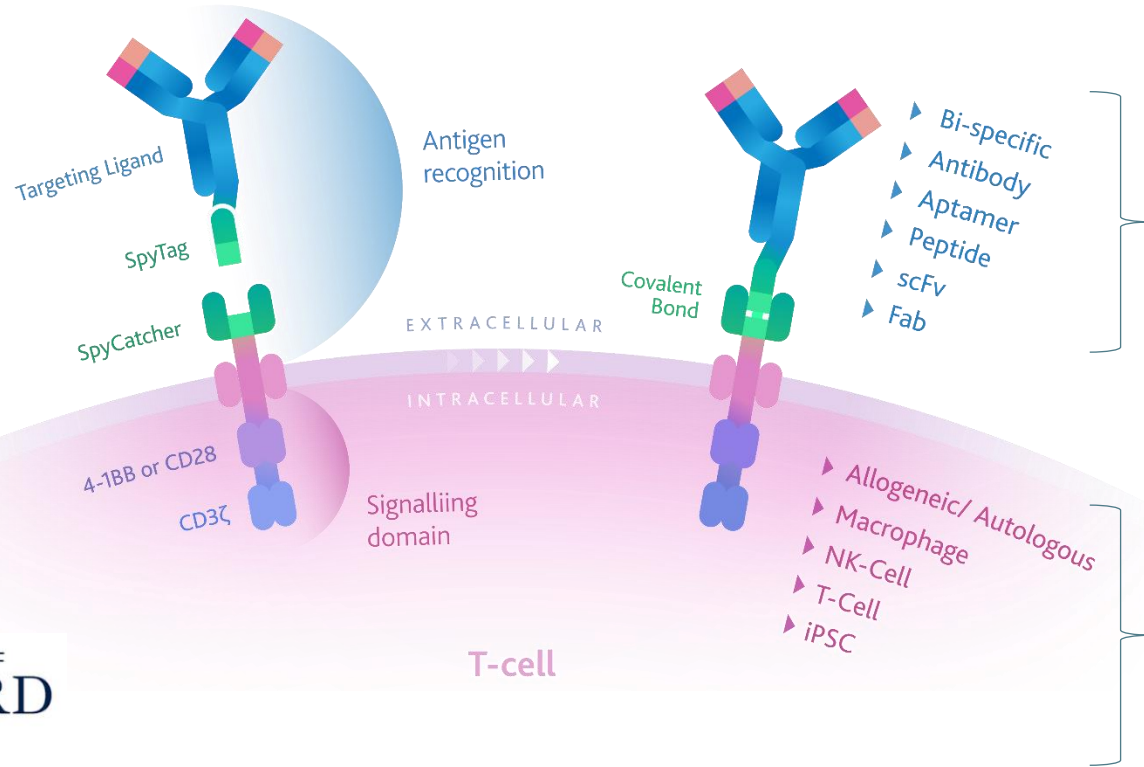


Target specificity CAR-T cell can be **switched at will**, by administering a different targeting ligand

An elegant and effective approach

Only UIR with spontaneous, autocatalytic, **covalent** bond formation

Binds targeting ligand to cell signalling domain



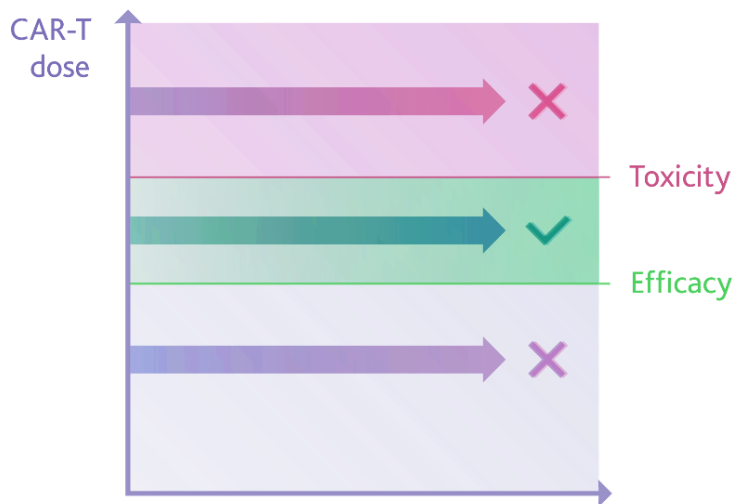
OmniCAR can use any type of **targeting ligand**...

...with any **immune cell**

Safety: Ability Control Dose & Activity

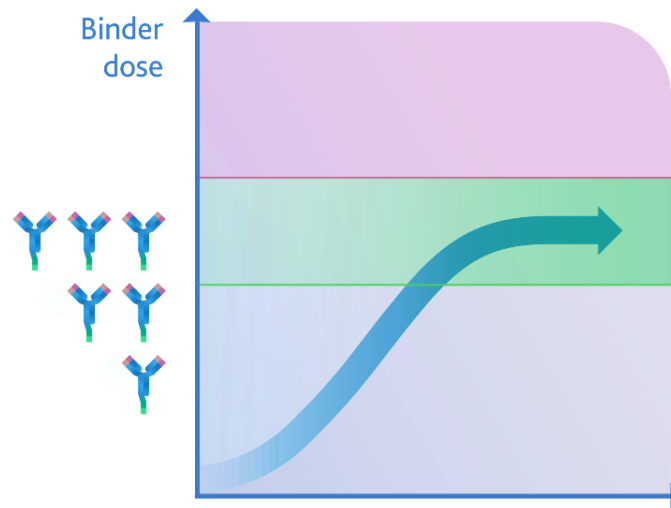
Conventional CAR-T

- Clinicians have **no control** over CAR-T activity once injected
- Estimate optimal dose **before infusion**
- Half-doses of CAR-T cells provide limited fidelity



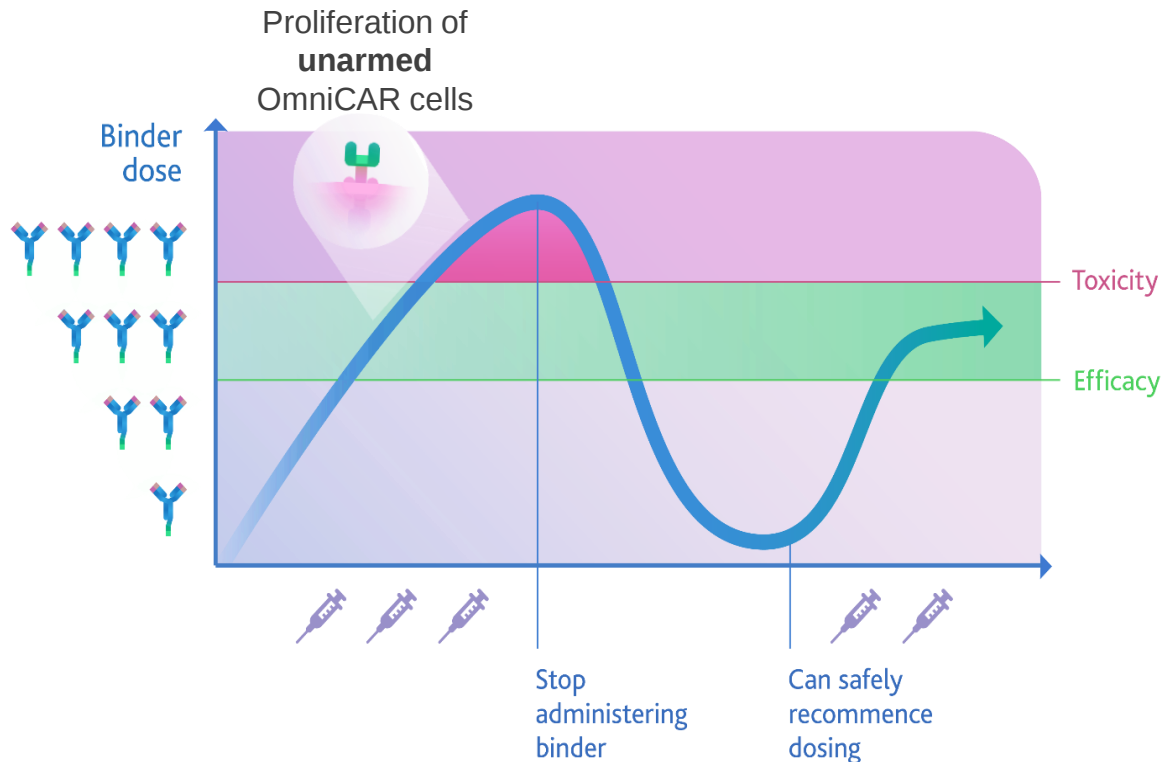
OmniCAR

- Clinician control **post infusion**
- Controlling subsequent **dose** of binder controls CAR-T **activity**
- Titrate dose to **safe and efficacious** levels

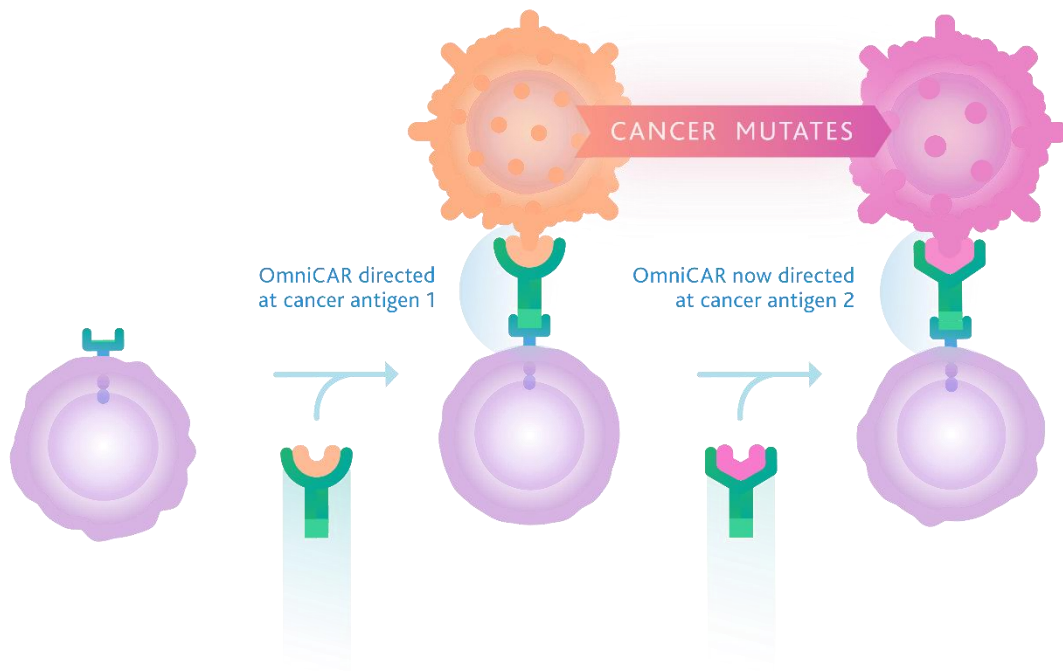


Safety: Built-in on/off switch

- Cell activity can be **switched off at-will**
- Cells remain **viable but inactive**
- OmniCAR can be **safely reactivated**
- No uncontrolled activity
- **Ongoing stimulation for greater efficacy & persistence**

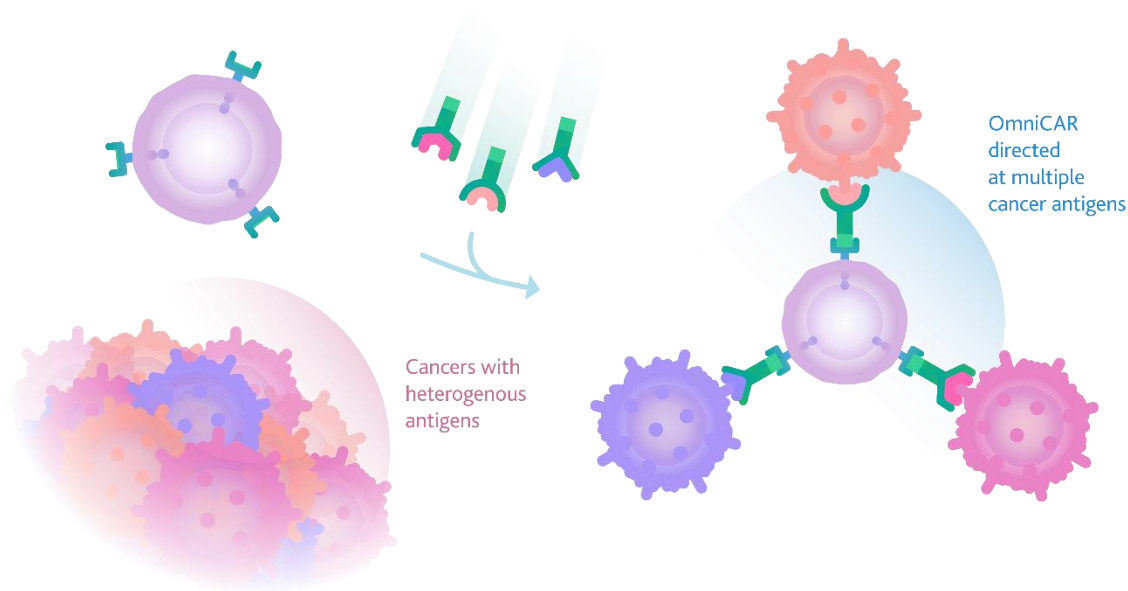


Target Multiple Antigens *Sequentially*



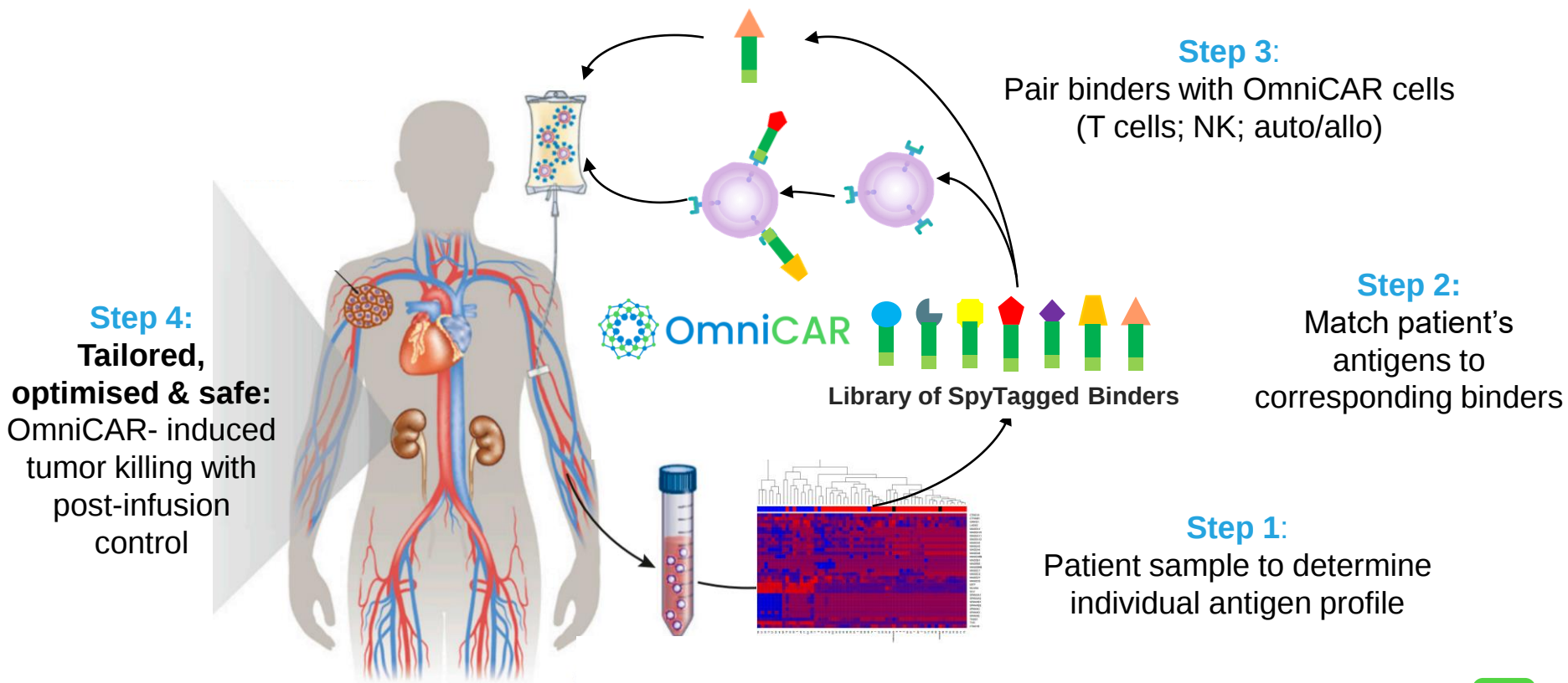
- Switching binder redirects the T-cell
- Uses single vector/cell product
- Addresses escape
- Useful for rapidly mutating cancers, esp those that cannot afford time for another CAR-T production run
 - E.g. AML

Target Multiple Antigens *Simultaneously*



- Multiple antigen targeting with single vector/cell product
- Could broaden anti-tumour immune response
- Prevents escape
- Tailor arming combinations and proportions
- Utility in many solid tumours

The future of ACT is efficient yet personalized: OmniCAR cells + “plug & play” binder library

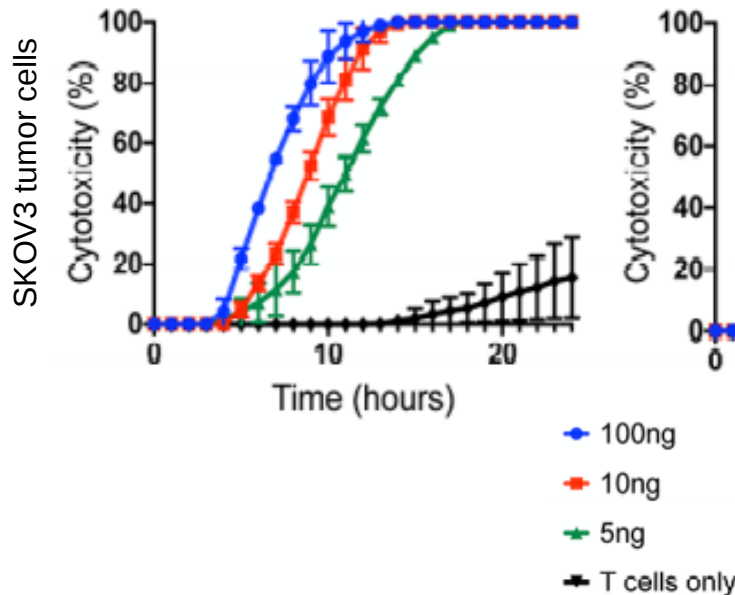


Covalent Binding:

Superior tumor killing & other advantages

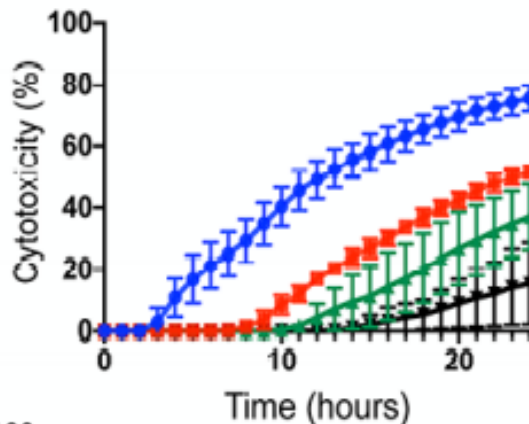
Covalent

SC28ζ + Herceptin-ST



Non-Covalent

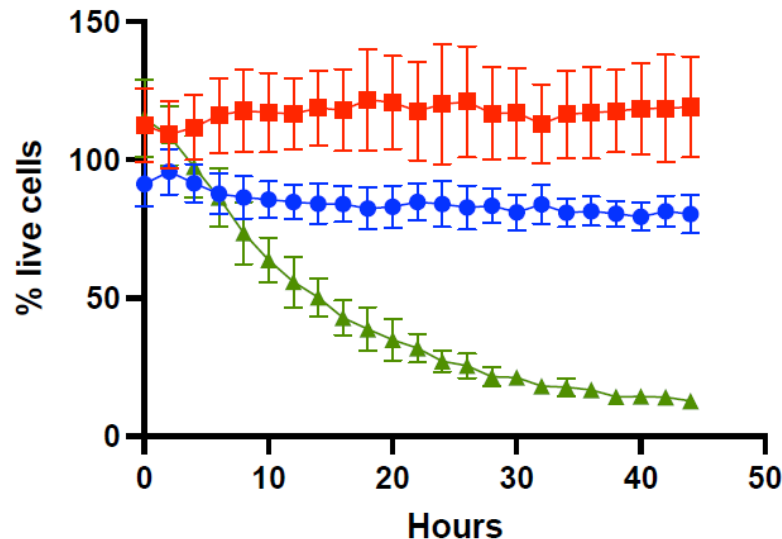
SC28ζ + Herceptin-STD A



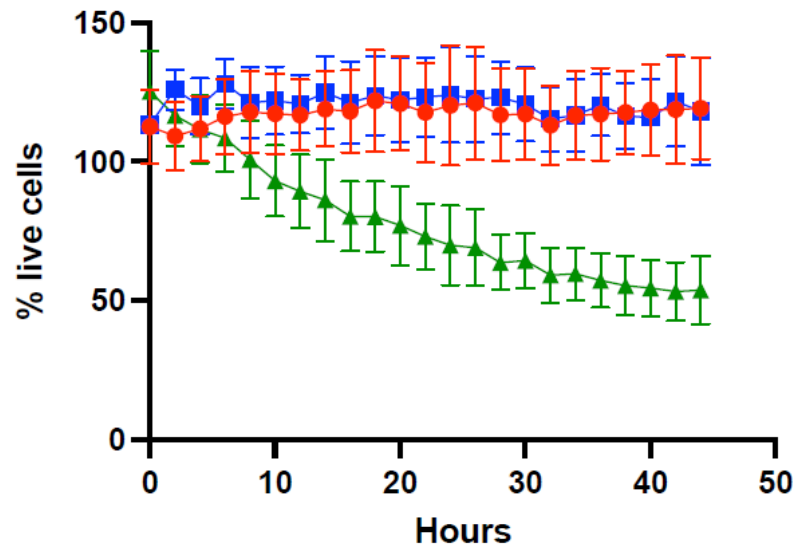
- **Covalent** binding improves SpyCatcher T-cell **loading and tumour cell lysis**
- Covalent binding has additional advantages in:
 - Efficacy
 - Predictability
 - Clinical utility
 - Regulatory considerations

OmniCAR HER2: predictable cytotoxicity

4:1 HER2 OmniCAR



2:1 HER2 OmniCAR

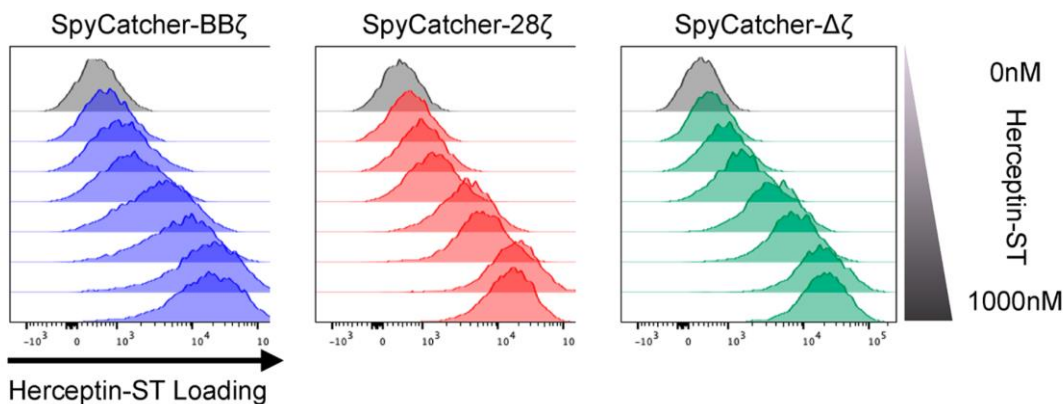


- Tumor Cells
- Unarmed OmniCAR
- ▲ HER2 Armed OmniCAR

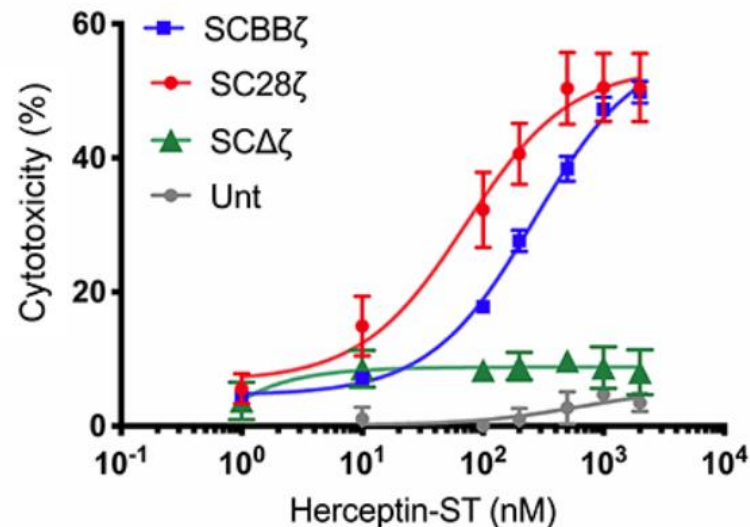
At 2:1, OmniCAR achieves cytotoxicity at a rate that aims to **balance efficacy** whilst **avoiding CRS and exhaustion**

Flexible Loading and Dose-Dependent Lysis

- OmniCAR T-cells capable of being armed with varying amounts of SpyTagged targeting ligand

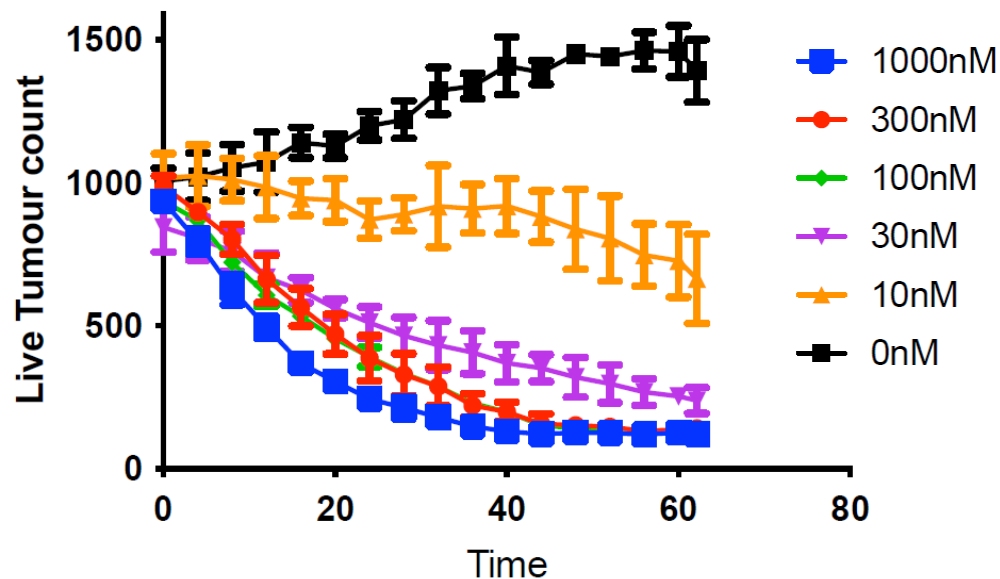


- Increasing targeting ligand concentration results in increased lytic capacity



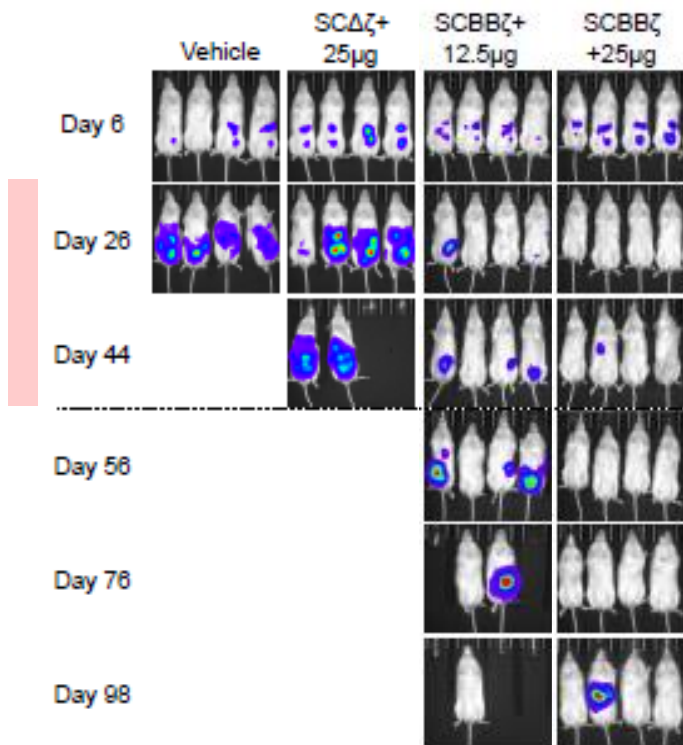
Dose response: High potency with less binder required

2:1 ST-EGFRviii binder vs U251 EGFRviii cells

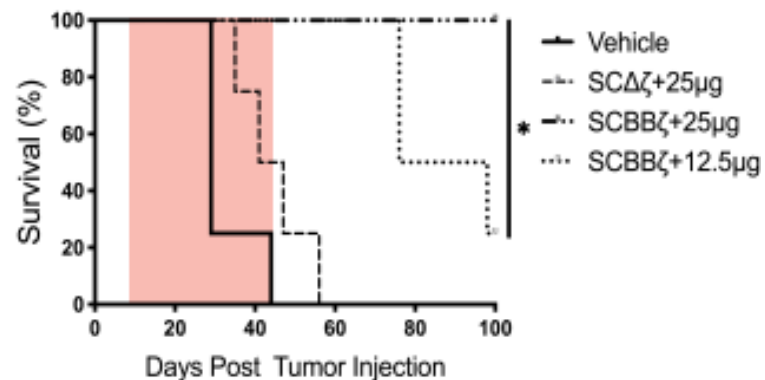


- Dose-dependent CAR-T activity
- V3 OmniCAR significantly more potent, and likely safer, than V1 system and competitor UIRs
- Potency with **60 fold less binder** (low nM range)
- Means **improved safety** and **lower cost of goods**

Control: Dose-dependent CAR-T activity



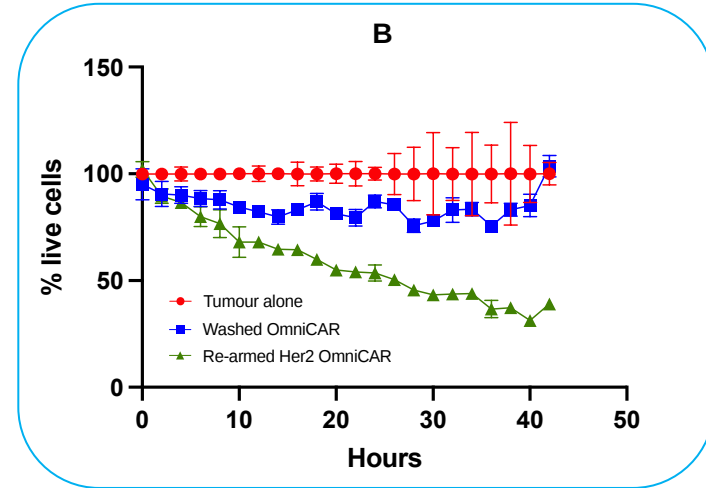
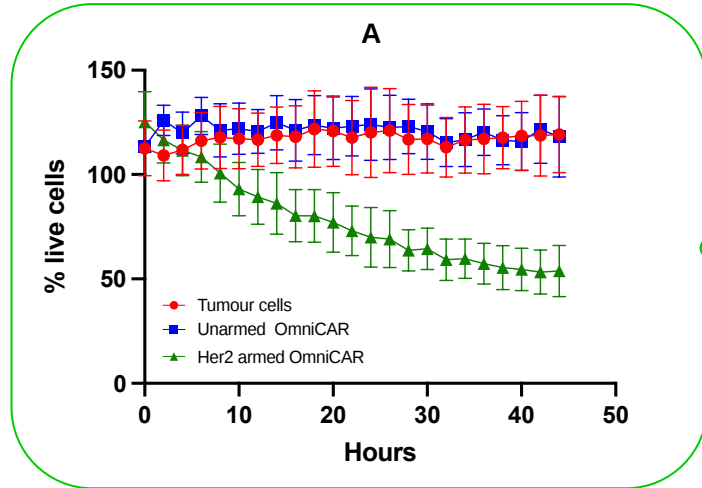
- Ovarian cancer model, using anti-HER2 OmniCAR
- Loading more binder results in **proportionate killing** of cancer...
- ...and **proportionate survival**
- **Lasting effects** even when cease dosing of binder



Re-Arming: OmniCAR Her2 can be Re-Armed



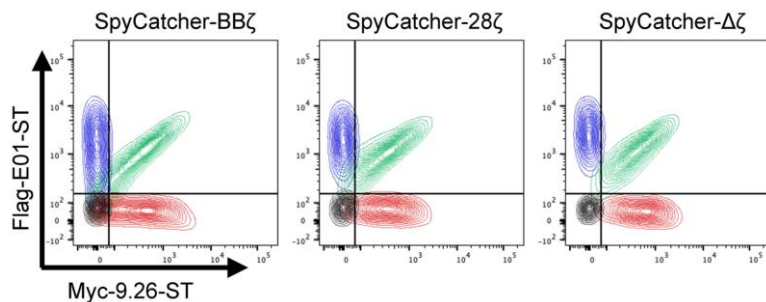
Wash; Rest



- OmniCAR T cells can be re-armed
- Re-arming results in **same levels and kinetics of cytotoxicity** as pre-armed
- Another example of **flexible** yet **predictable** activity

Equal Arming & Equal Tumour Killing

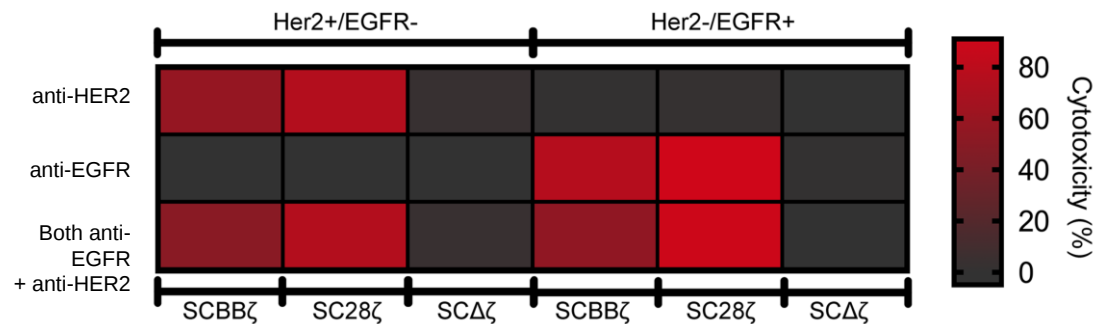
Equal arming



CAR-T equally armed with:

- Both anti-EGFR + anti-HER2
- anti-EGFR
- anti-HER2
- control

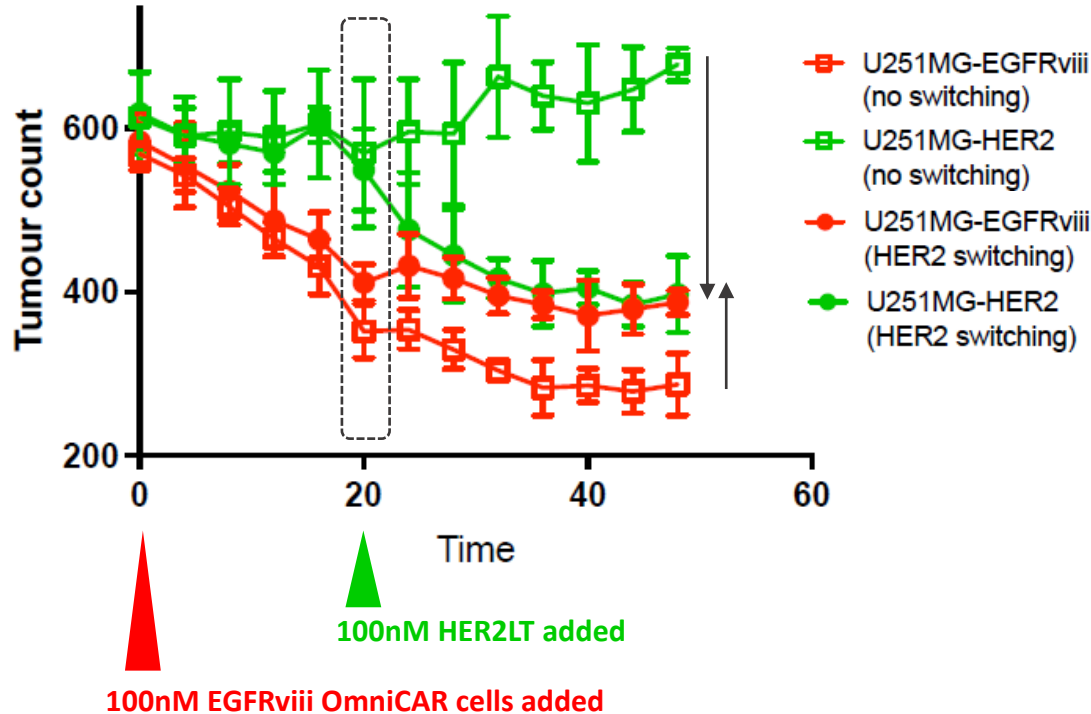
Specifically directed, at-will killing



- Only kills cells that the CAR-T is armed against
- OmniCAR CAR-T cells have similar specific tumour killing capacity, whether **dual**-armed or **single**-armed

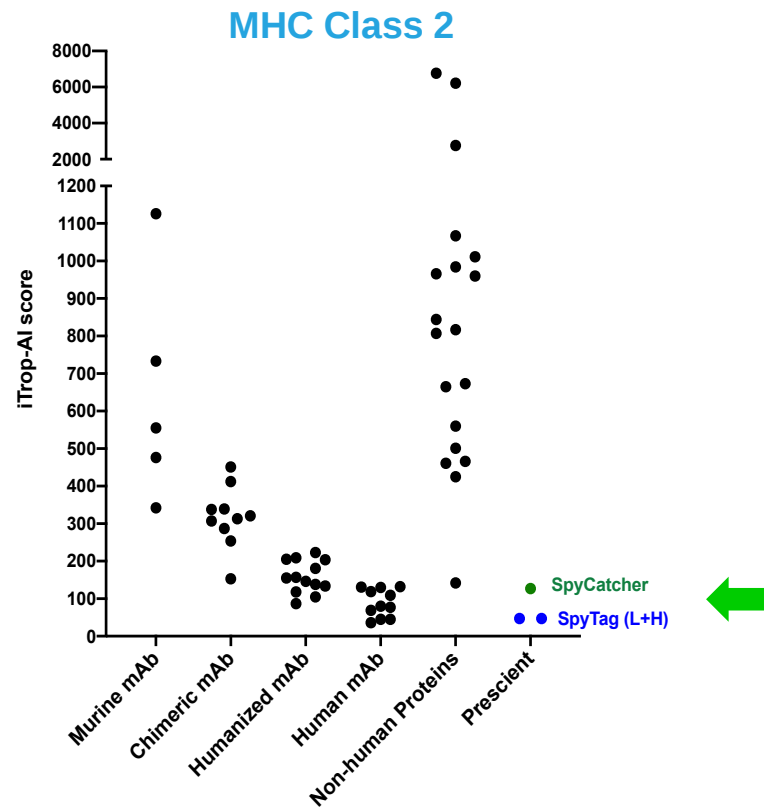
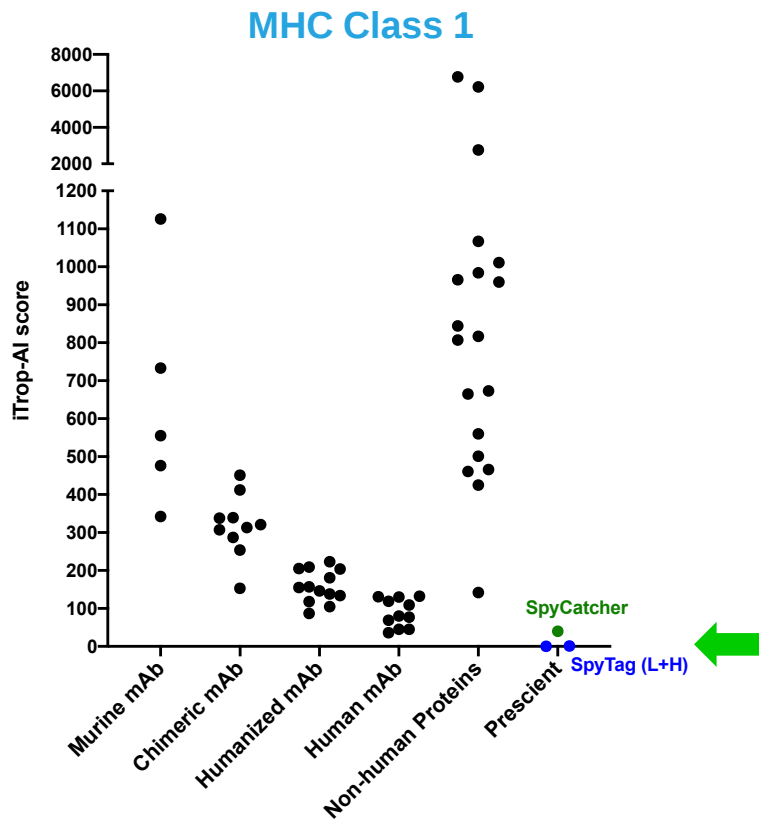
Redirection: Adding new ST-binder can re-direct cytotoxicity

Antigen Target Re-direction in Coculture of
U251 GBM Cells expressing HER2 or EGFRviii



- Rapid cytotoxicity to EGFRviii
- **Rapid switching and cytotoxicity** against HER2+ tumours upon administration of new binder
- OmniCAR cells can be **re-directed to different antigens** upon administration of a different SpyTagged binder **without new cells**

In-silico immunogenicity on par with Human mAbs



OmniCAR Internal Program Summary

Targets	Indications	OmniCAR features	Comments
CD33 + CLL-1	Acute Myeloid Leukemia (AML)	• Titration for improved safety • Co-arming against CD33 & CLL-1 • Sequential targeting	• Validated targets; expressed on 90%+ of AML blasts & LSCs • 1 of 3 programs worldwide; the only next-gen program
HER2	Ovarian; breast & gastric cancers	• Titration for improved safety • Persistent binder dosing for improved efficacy • TME and checkpoint enhancements	• Most mature next-gen HER2 CAR-T program • Builds on Penn pre-clinical PoC
HER2 + EGFRviii	Glioblastoma multiforme (GBM)	• Titration for improved safety • Co-arming against HER2 & EGFRviii • Persistent binder dosing for improved efficacy	• 1 of 3 multiple antigen programs in the world • Single antigen targeting is inadequate in GBM

AML

OmniCAR CD33/CLL-1

For CAR-T to succeed in AML, it must overcome:



Safety

AML patients are especially ill with many unable to tolerate vigorous therapies like CAR-T



Rapid Mutations

AML can mutate mid-therapy, quickly rendering single CAR-Ts ineffective



Rapid Disease Progression

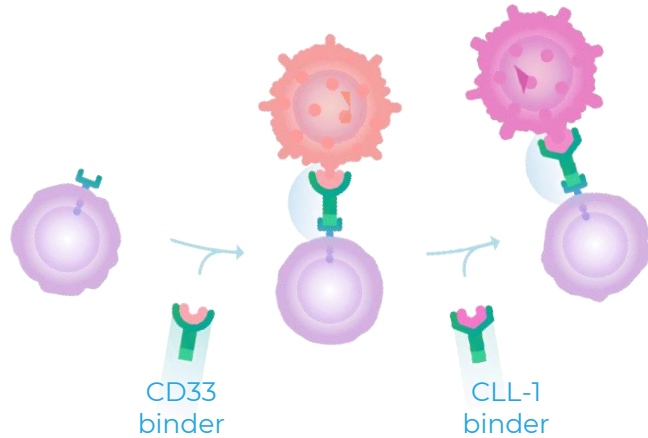
Even if multiple current generation CAR-T therapies were available, resistant patients are likely to progress before subsequent therapies are manufactured for them

OmniCAR is uniquely placed to address these challenges for CAR-T in AML

CD33 & CLL-1 are excellent AML targets for CAR-T

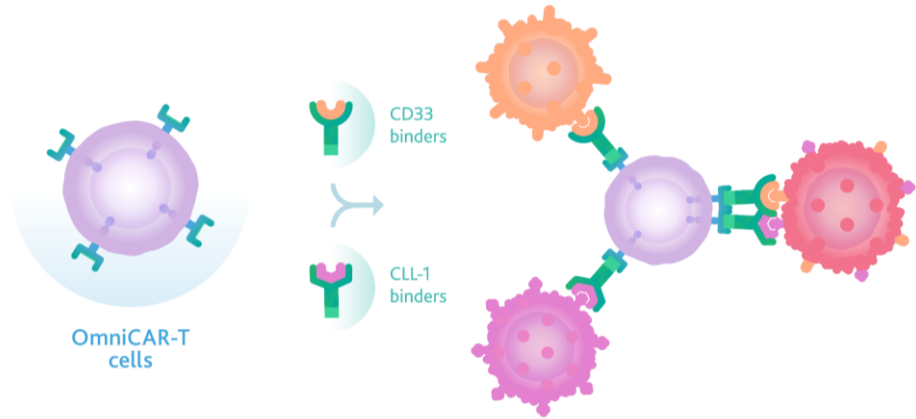
- CD33
 - Validated target in AML with approved anti-body drug conjugate (gemtuzumab ozogamicin, or Mylotarg)
 - CD33 is constantly expressed on both normal and malignant myeloid cells
 - CD33 expressed on >90% adult and childhood AML blasts and on leukemia stem cells, which have the ability to indefinitely replicate to produce cancerous leukemic cells, leading to relapse
- CLL-1
 - Expressed on 92% of AML cells
 - Absent from normal hemopoietic stem cells
 - Importantly, CLL1 is expressed on leukemic stem cells, which produce subsequent cancer cells leading to relapse

Sequentially



- Address antigen escape by redirecting T-cells without new dose of T-cells
- May be a more tolerable approach for sick AML patients

Simultaneously



- Co-Arming against CD33 & CLL1 on a single T-cell product
- Target several cancer cell populations at once:

CD33+

CLL1+

CD33+ CLL1+
- Could broaden anti-tumour immune response.

Solid Tumors

OmniCAR HER2

Key challenges for CAR-T in solid tumours



Targets

Limited targets that are cancer-specific
Leads to on-target, off-tumour effects



Safety

Ability to titrate doses safely and switch off in the event of adverse events
Especially important for on-target, off-tumour activity



Trafficking

Inability of T-cells to reach tumour sites and penetrate physical barriers



TME

Overcoming an immunosuppressive Tumour Microenvironment once they get there

OmniCAR's features enable it to address these challenges for CAR-T in solid tumours

Huge market opportunities for HER2+ cancers

	New cases/year worldwide ¹	Proportion that are HER2+ ^{2,3,4}	New HER2+ cases/year
Ovarian Cancer	300,000	29%	87,000
Breast Cancer	1,700,000	20%	340,000
Gastric Cancer	952,000	22%	209,440

- OmniCAR T cells armed against HER2
- Builds upon the encouraging work already undertaken by UPenn with HER2
- Makes OmniCAR HER2 the most advanced next-generation HER2 CAR-T program
- Prescient will take a “basket study” approach to HER2+ cancers
- Even when failing HER2 therapies, tumours can still express HER2, making these patients potential candidates for anti-HER2 CAR-T therapy

1. World Cancer Research Fund
2. Shang AQ, et al. Relationship between HER2 and JAK/STAT-SOCS3 signaling pathway and clinicopathological features and prognosis of ovarian cancer. *Cancer biology & therapy*. 2017:1–9
3. Luo, H et al, The prognostic value of HER2 in ovarian cancer: A meta-analysis of observational studies. *PLoS ONE* 13(1) 2018
4. Bang YJ, et al. Trastuzumab in combination with chemotherapy versus chemotherapy alone for treatment of HER2-positive advanced gastric or gastro-oesophageal junction cancer (ToGA): a phase 3, open-label, randomised controlled trial. *Lancet* 2010;376:687-97.

GBM

OmniCAR HER2/EGFRviii

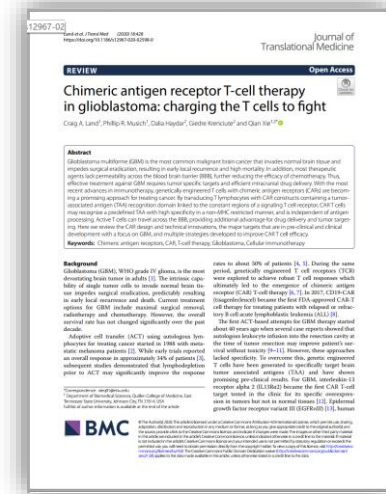
CAR-T challenges in GBM: single antigen targeting

- Composition of GBM, and its ability to rapidly mutate, limits the effectiveness of CAR-Ts only targeting a single antigen
- Targeting a single antigen targeting can result in relapse

“A major limitation of a single-antigen targeting in GBM is the inherent heterogeneity and plasticity of the tumor cells, allowing some cells to escape CAR-T cell killing due to the loss of the targeted antigen...”

“...single antigen-targeting CAR-T cells fail to completely eradicate brain tumors resulting in antigen negative relapses”

- By contrast, CAR-Ts targeting multiple antigens have demonstrated **anti tumor responses** and **more importantly prevented antigen escape *in vivo***



Two targets are better than one in GBM

- Single antigen targeting has been inadequate in GBM
- By contrast, **combination** of HER2 and other antigen targeting shows early promise in overcoming relapse
- Prescient will also explore other targets for GBM



HER2

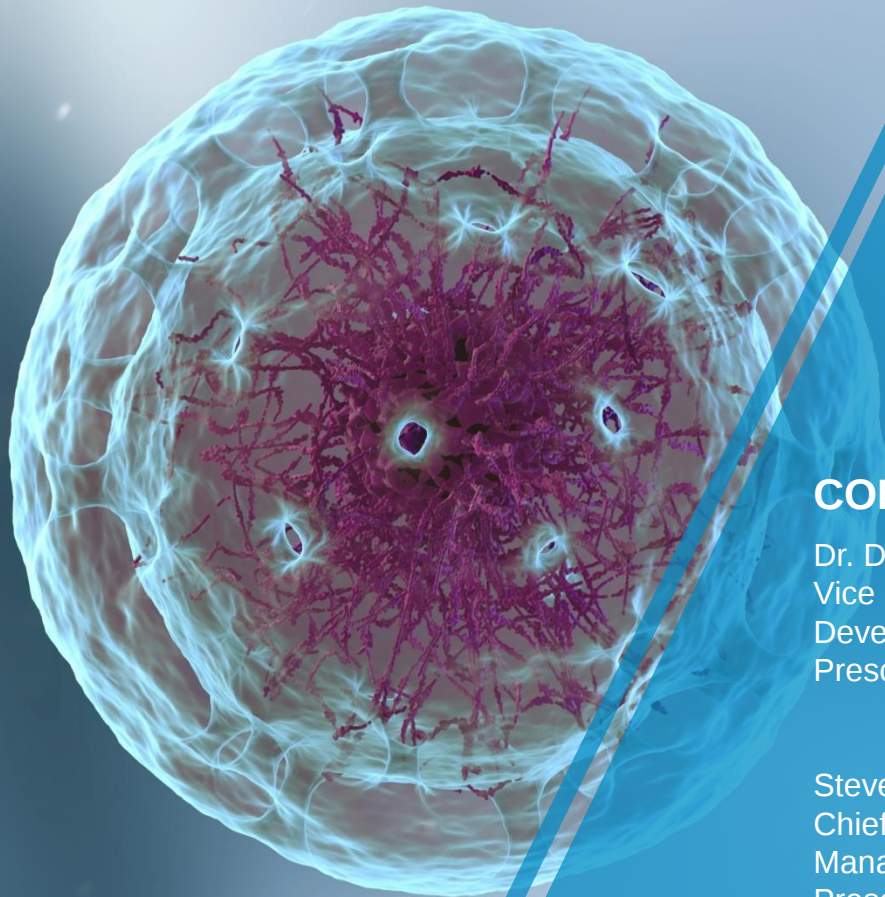
- HER2 occurs in 80% of GBM
- Linked with poor survival



EGFRviii

- EGFRviii occurs in 45% of GBM
- Importantly, EGFRviii is only present on GBM and **is not found on healthy tissues**

- OmniCAR addresses problems encountered by current generation CAR-T
- 3 in-house OmniCAR programs, all highly differentiated and representing large opportunities
 - CD33/CLL-1 for AML
 - HER2+ solid tumours
 - HER2/EGFRviii for GBM
- Prescient is open to licensing and collaboration. OmniCAR can enhance the safety, flexibility and efficacy of third-party CAR programs
 - Agnostic on targets; indication; cell type



Prescient
Therapeutics

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