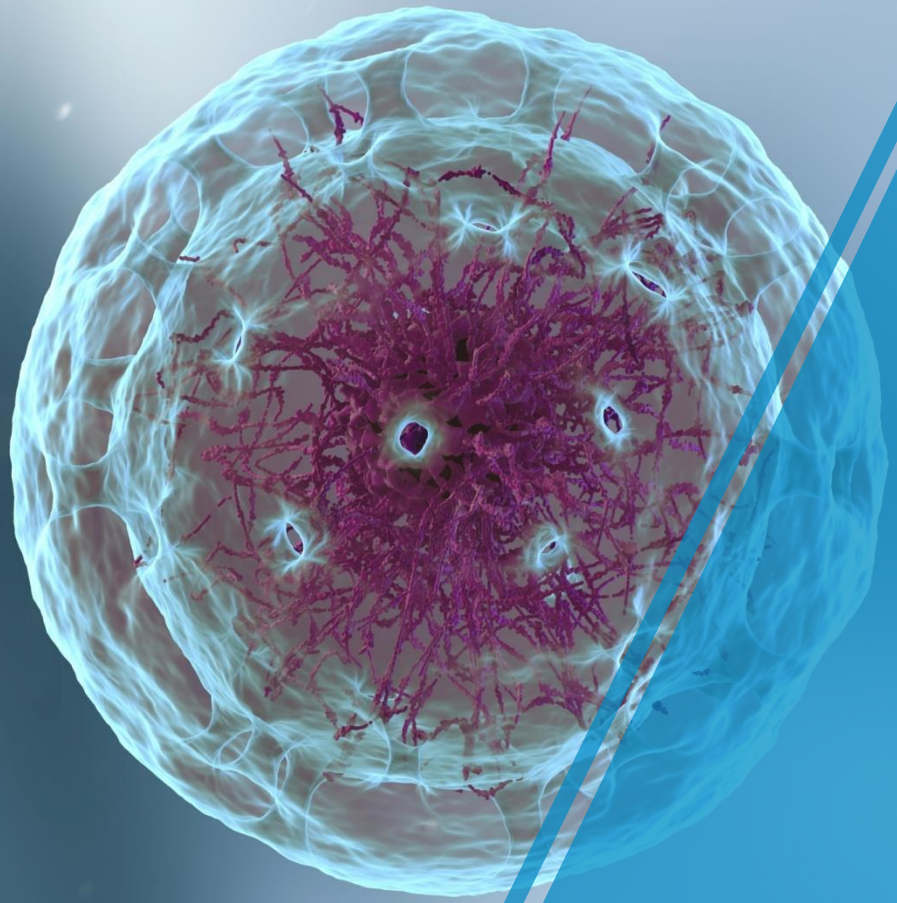

Prescient Therapeutics

OmniCAR

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

Certain statements made in this document are forward-looking statements within the meaning of the safe harbor provisions of the United States Private Securities Litigation Reform Act of 1995. These forward-looking statements are not historical facts but rather are based on the current expectations of Prescient Therapeutics Limited ("Prescient" or the "Company"), their estimates, assumptions, and projections about the industry in which Prescient operates. Material referred to in this document that use the words 'estimate', 'project', 'intend', 'expect', 'plan', 'believe', 'guidance', and similar expressions are intended to identify forward-looking statements and should be considered an at-risk statement. These forward-looking statements are not a guarantee of future performance and involve known and unknown risks and uncertainties, some of which are beyond the control of Prescient or which are difficult to predict, which could cause the actual results, performance, or achievements of Prescient to be materially different from those which may be expressed or implied by these statements. These statements are based on our management's current expectations and are subject to a number of uncertainties and risks that could change the results described in the forward-looking statements. Risks and uncertainties include, but are not limited to, general industry conditions and competition, general economic factors, the impact of pharmaceutical industry development and health care legislation in the United States and internationally, and challenges inherent in new product development. Investors should be aware that there are no assurances that results will not differ from those projected and Prescient cautions shareholders and prospective shareholders not to place undue reliance on these forward-looking statements, which reflect the view of Prescient only as of the date of this presentation. Prescient is not under a duty to update any forward-looking statement as a result of new information, future events or otherwise, except as required by law or by any appropriate regulatory authority.

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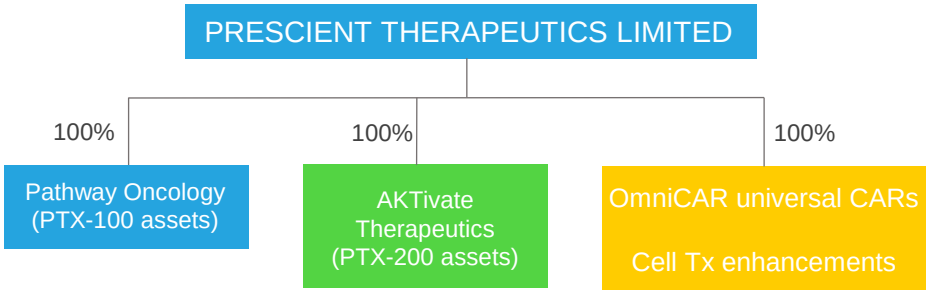
Corporate Snapshot

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 Capitalisation[1]	**A$174 M (US$127 M)**
Market Cap fully diluted[1]	**A$202 M (US$148 M)**
Cash Position[2]	**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

Prescient Therapeutics

CAR - T

TARGETED THERAPIES

Program	Discovery	Screening	Preclinical	Phase I	Phase IB	Phase II
OmniCAR CD33 & CLL-1	AML					
OmniCAR HER2	Breast, Ovarian & Gastric					
OmniCAR HER2 & EGFRviii	GBM					
Cell Therapy Enhancements	Undisclosed 1					
	Undisclosed 2					
PTX - 100	PTCL					
PTX - 200	AML					

Peter Mac
Peter MacCallum Cancer Centre
Victoria Australia

Peter Mac
Peter MacCallum Cancer Centre
Victoria Australia

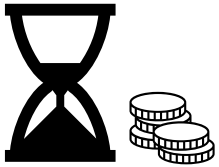
Prescient Therapeutics

OmniCAR

Universal, Next Generation CAR-T

Key Challenges Confronting the field of CAR-T

Prescient Therapeutics



Time and Cost

of delivering treatment

Targets

Finding targets that work;

Antigen heterogeneity - esp. in solid tumours

Safety

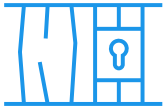
CAR-T can have serious safety concerns

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**

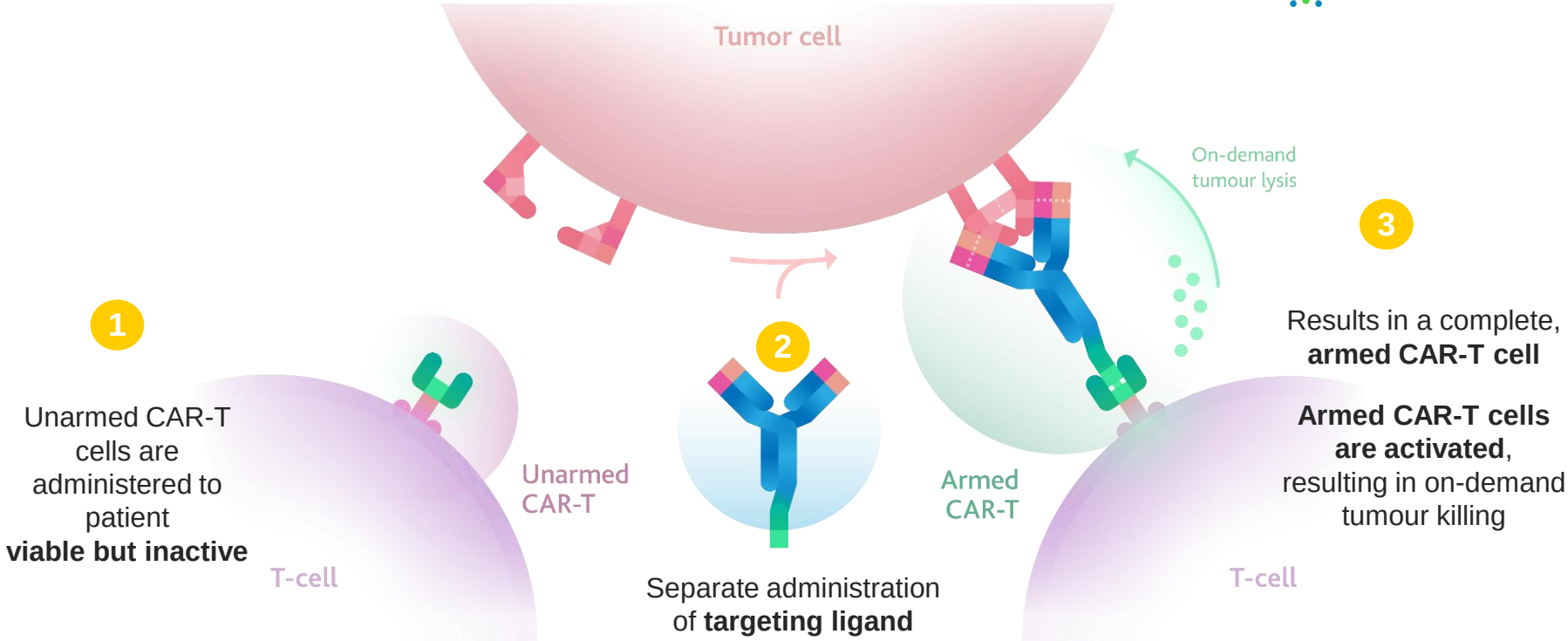
Co-inventors

Associate Professor
Daniel J. Powell, Jr

Professor
Andrew Tsourkas

Powell, DJ et al, JACS; 2020

How OmniCAR works



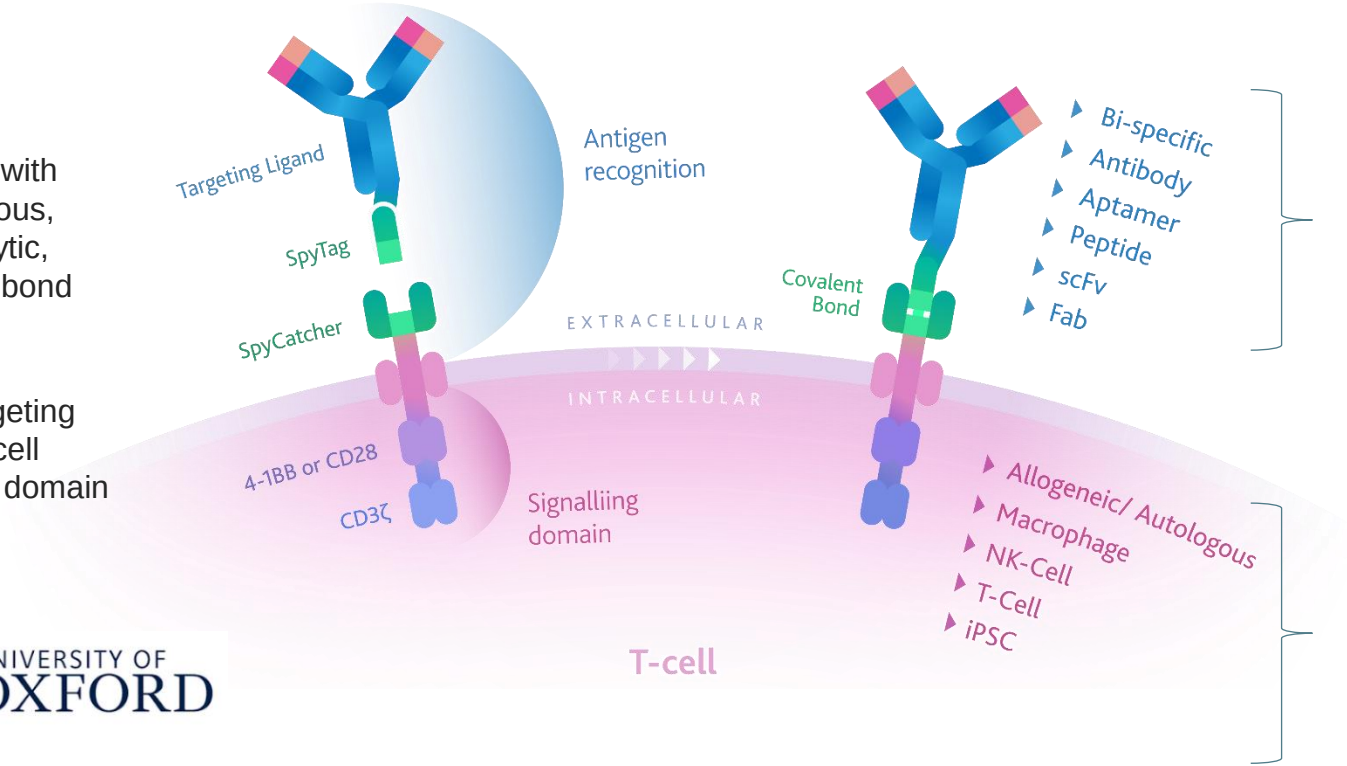
- CAR-T cell activity is **now controllable**
- Target specificity CAR-T cell can be **switched at will**, by administering a different targeting ligand

Concept to be tested in human under well controlled clinical trial(s) in compliance with regulatory requirements.

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…**

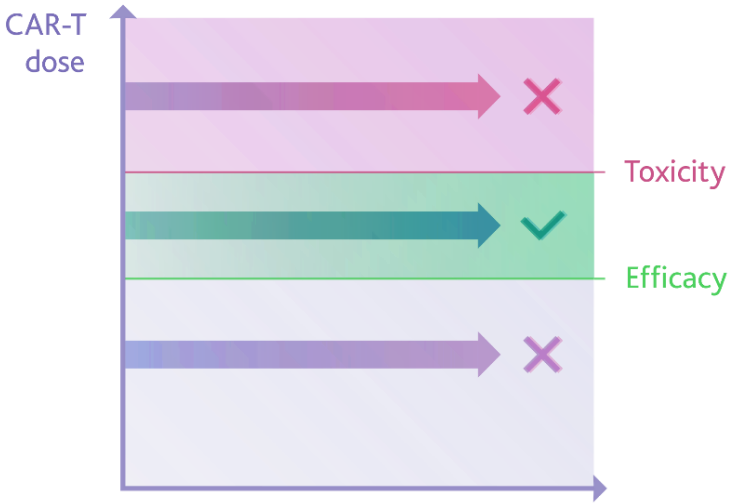
…with any immune cell

Adapted from Powell, DJ et al, JACS; 2020

Safety: Ability Control Dose & Activity

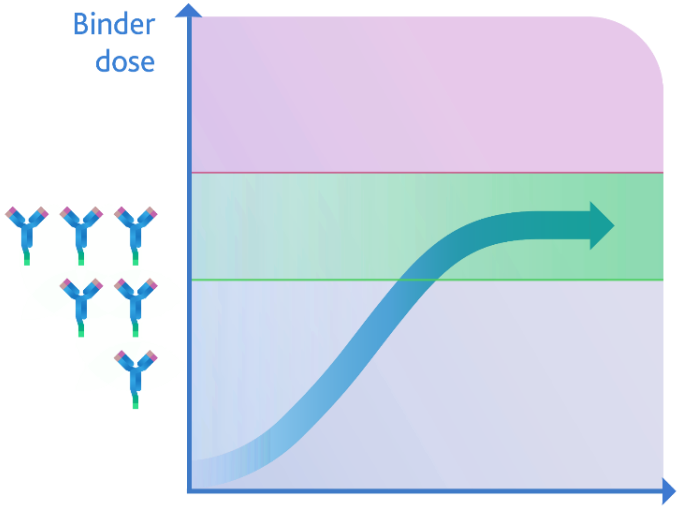
Conventional CAR-T

- 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

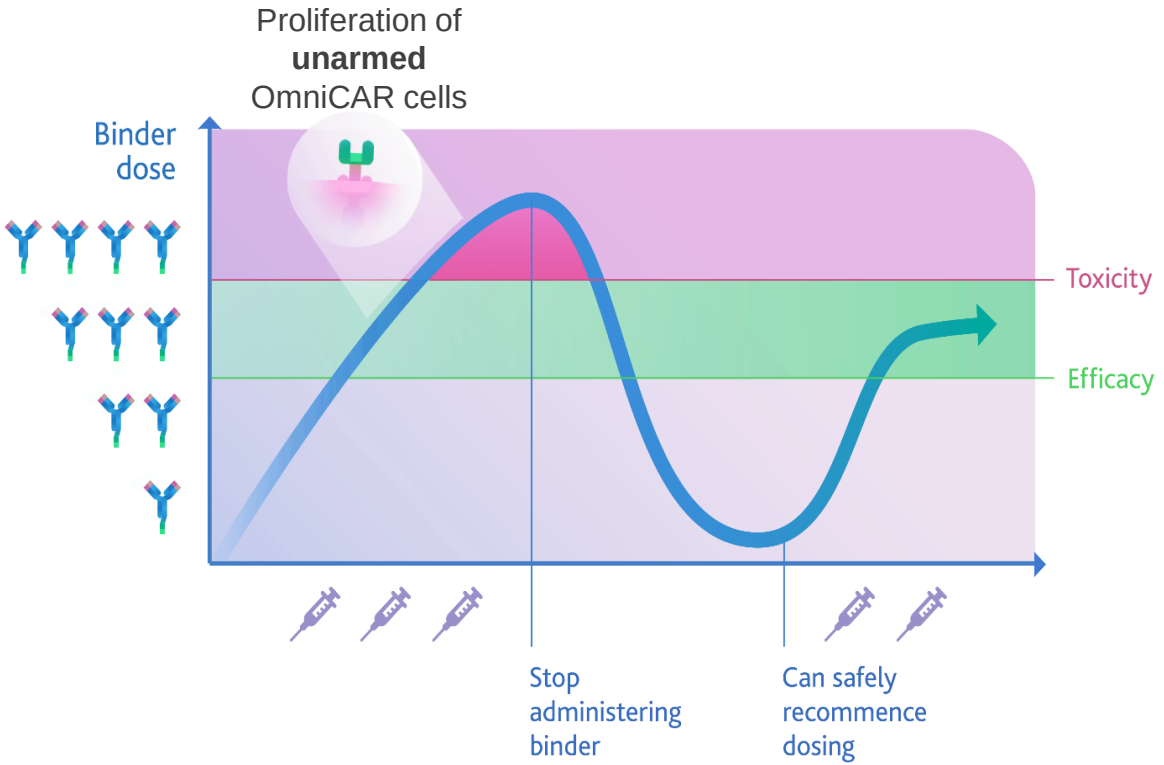
- Clinician control **post infusion**
- Controlling subsequent **dose** of binder controls CAR-T **activity**
- Titrate dose to **safe and efficacious** levels



Concept to be tested in human under well controlled clinical trial(s) in compliance with regulatory requirements.

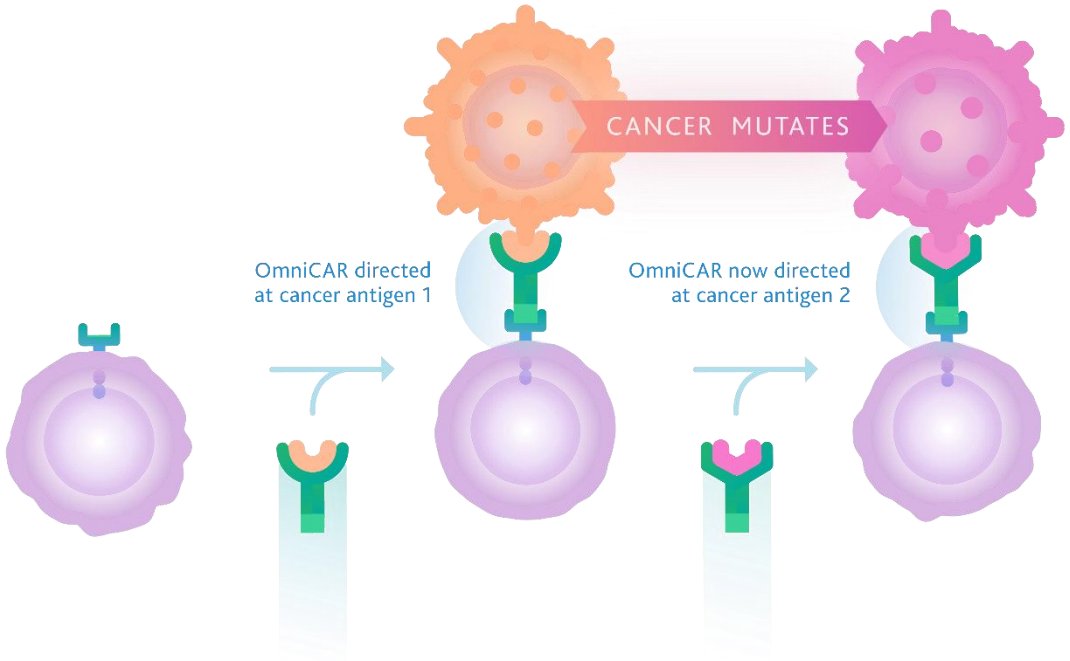
Safety: Built-in on/off switch

- 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**



Concept to be tested in human under well controlled clinical trial(s) in compliance with regulatory requirements.

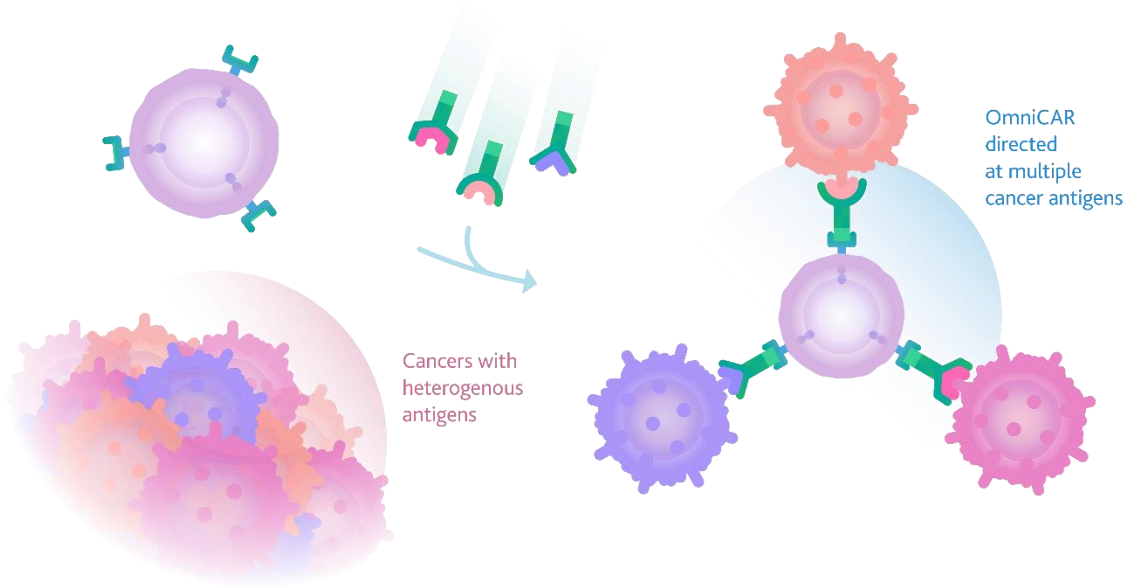
Target Multiple Antigens *Sequentially*



- 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

Concept to be tested in human under well controlled clinical trial(s) in compliance with regulatory requirements.

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

Concept to be tested in human under well controlled clinical trial(s) in compliance with regulatory requirements.

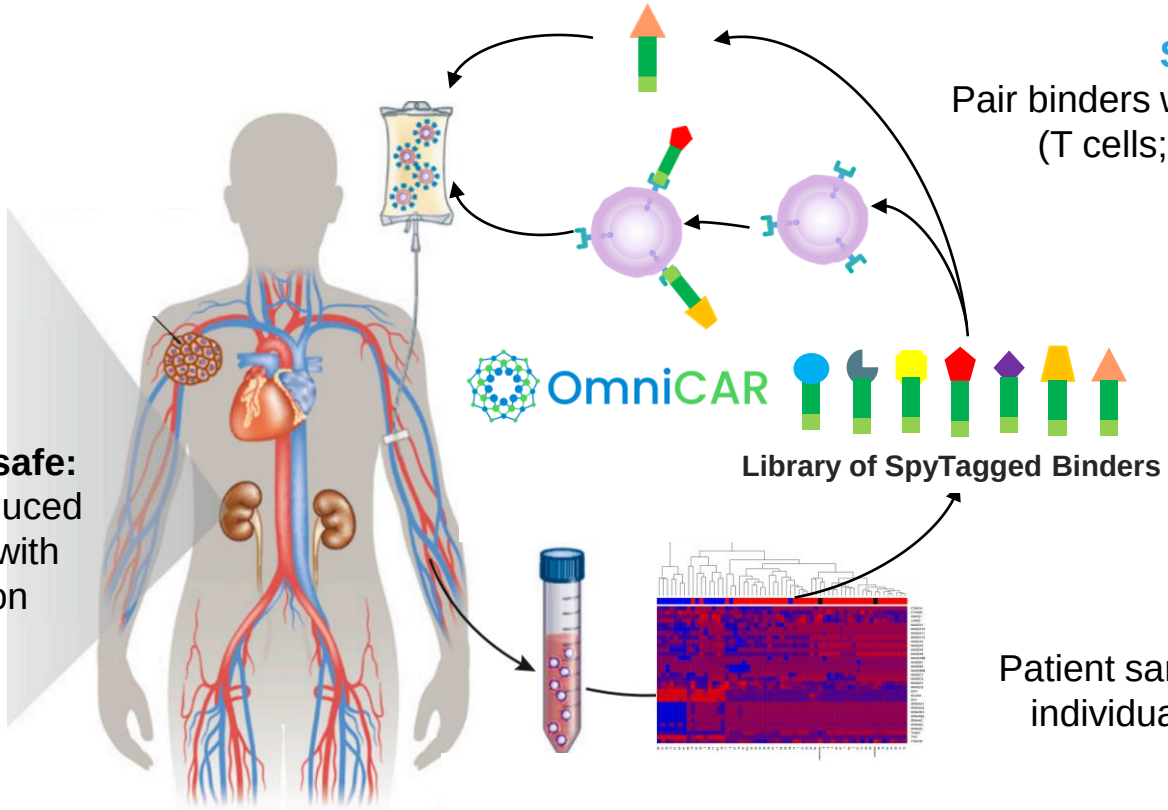
The future of ACT is efficient yet personalized:
OmniCAR cells + “plug & play” binder library

Prescient Therapeutics

Step 3:
Pair binders with OmniCAR cells
(T cells; NK; auto/allo)

Step 2:
Match patient’s antigens to corresponding binders

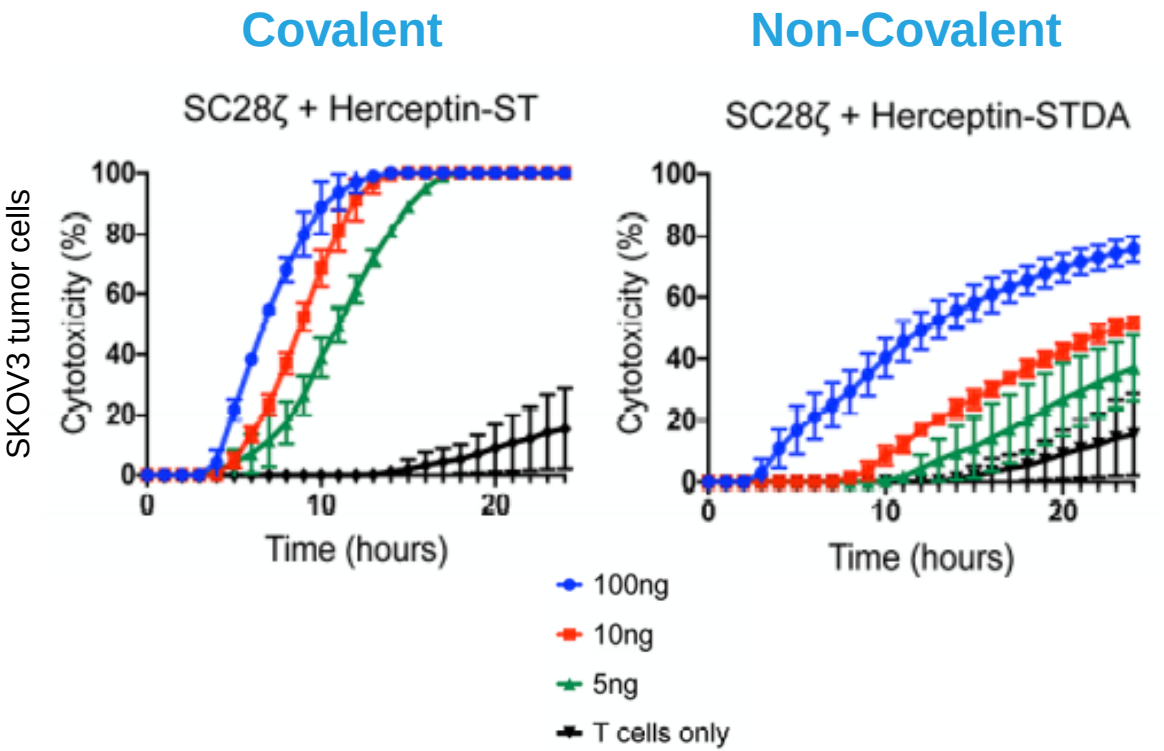
Step 4:
Tailored, optimised & safe:
OmniCAR- induced tumor killing with post-infusion control



Step 1:
Patient sample to determine individual antigen profile

Covalent Binding:

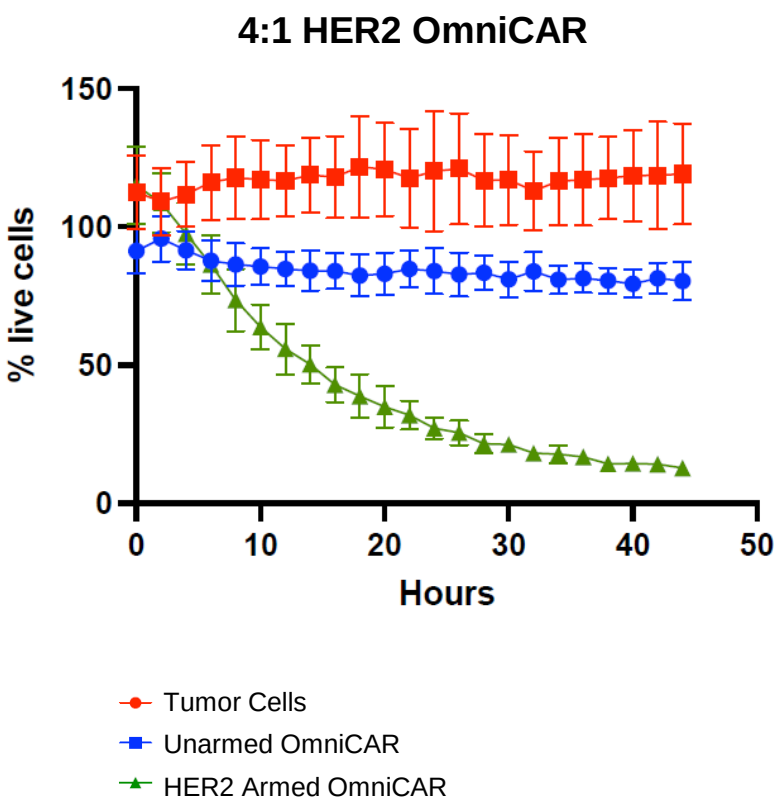
Superior tumor killing & other advantages

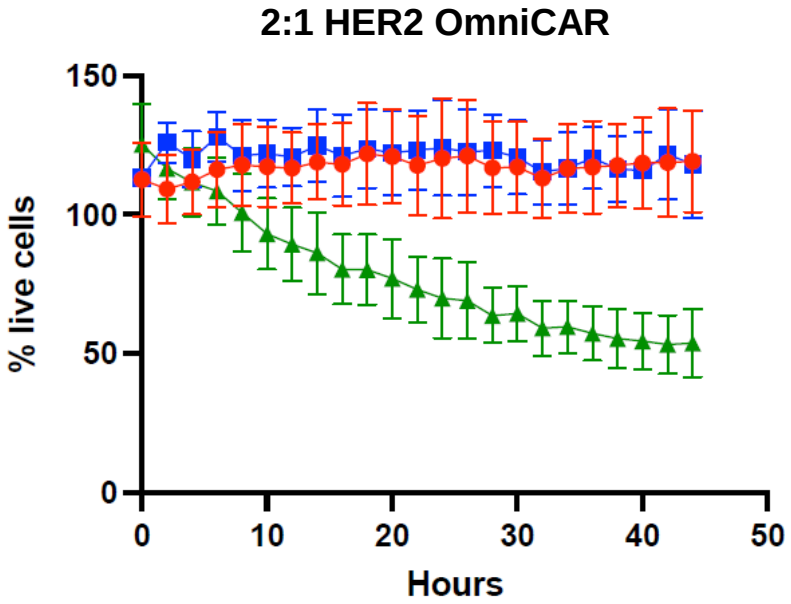


- **Covalent** binding improves SpyCatcher T-cell **loading and tumour cell lysis**
- Covalent binding has additional advantages in:
  - Efficacy
  - Predictability
  - Clinical utility
  - Regulatory considerations

Powell, DJ et al, JACS; 2020
STDA : A SpyTag variant containing an aspartic acid to alanine mutation (SpyTag-DA) abolishes covalent bond formation while allowing formation of a noncovalent complex

OmniCAR HER2: predictable cytotoxicity

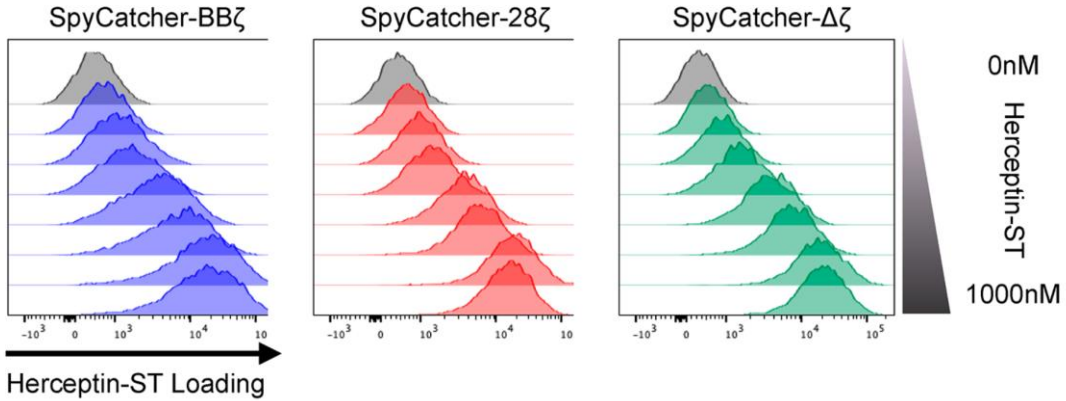




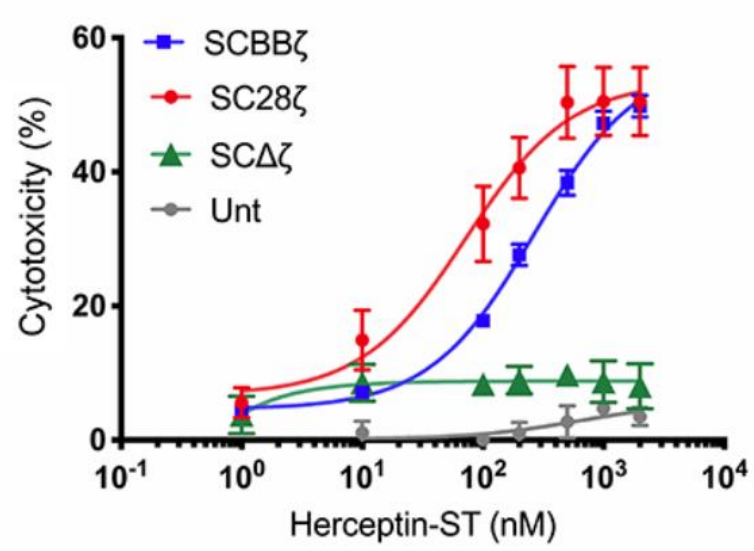
At 2:1, OmniCAR achieves cytotoxicity at a rate that aims to **balance efficacy** whilst **avoiding CRS and exhaustion**

Flexible Loading and Dose-Dependent Lysis

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



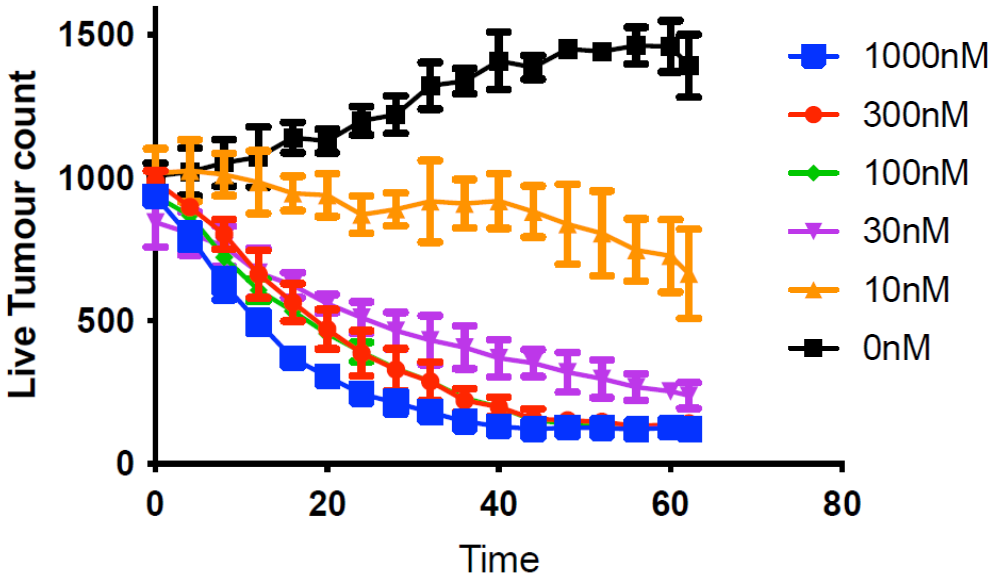
- Increasing targeting ligand concentration results in increased lytic capacity



Powell, DJ et al, JACS; 2020

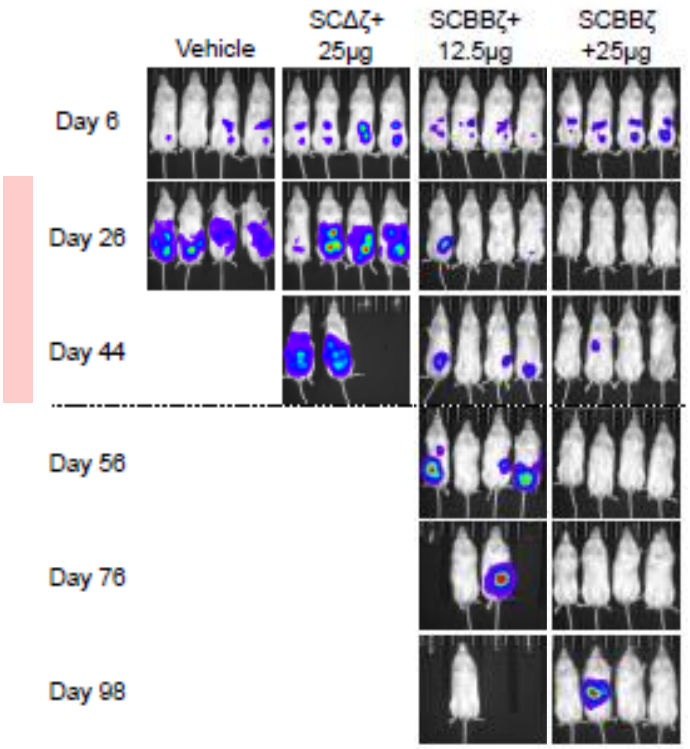
Dose response: High potency with less binder required

2:1 ST-EGFRviii binder vs U251 EGFRviii cells

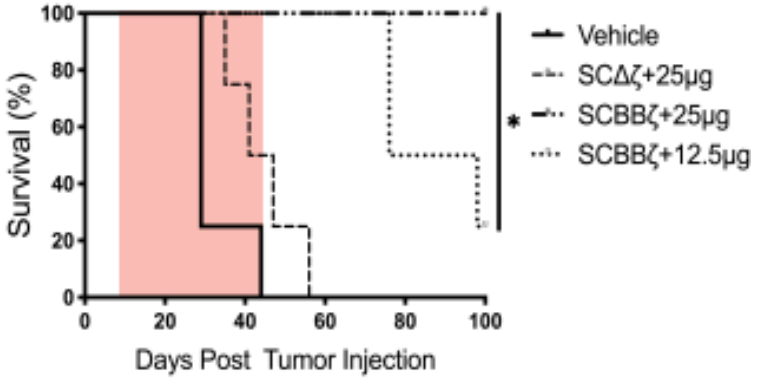


- **Dose-dependent CAR-T activity**
- V3 OmniCAR significantly more potent, and likely safer, than V1 system and competitor UIRs
- Potency with **60 fold less binder** (low nM range)
- Means **improved safety** and **lower cost of goods**

Control: Dose-dependent CAR-T activity



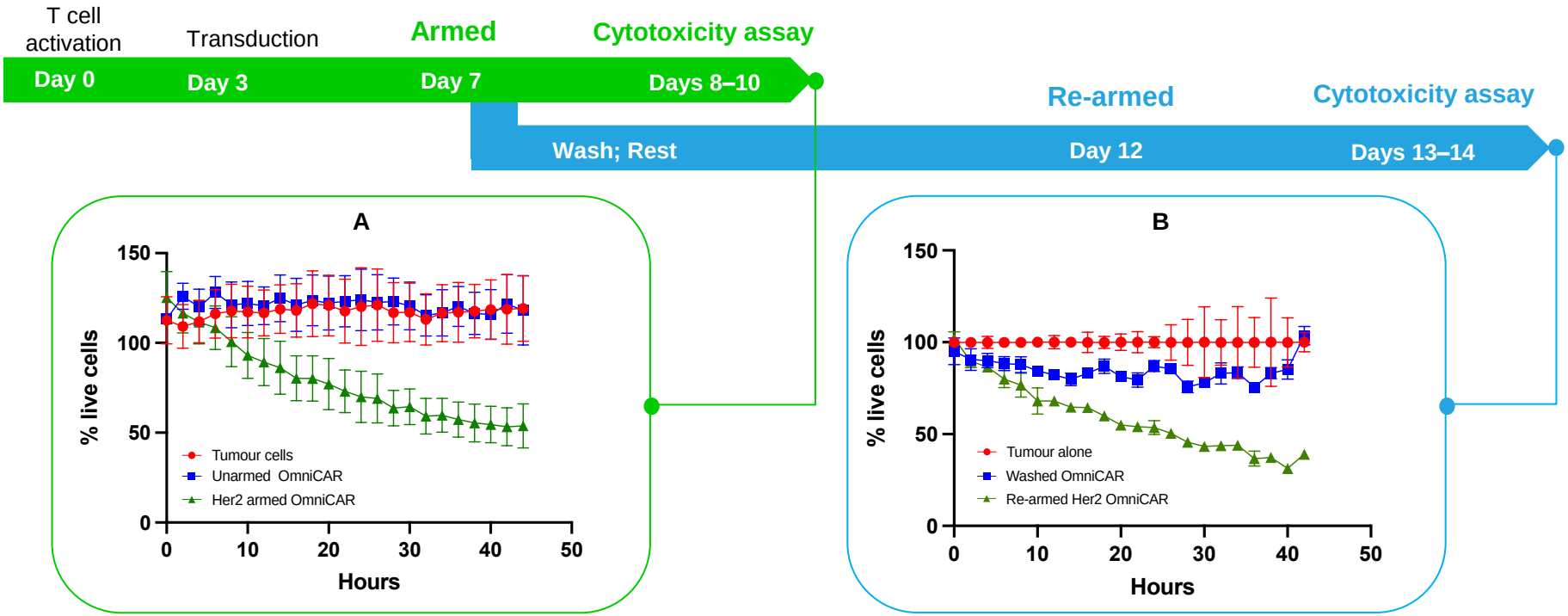
- Ovarian cancer model, using anti-HER2 OmniCAR
- Loading more binder results in **proportionate killing** of cancer…
- …and **proportionate survival**
- **Lasting effects** even when cease dosing of binder



Powell, DJ et al, JACS; 2020

= dosing window of binder

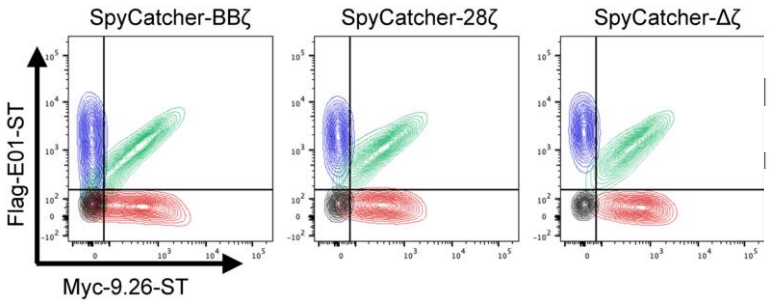
Re-Arming: OmniCAR Her2 can be Re-Armed



- 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

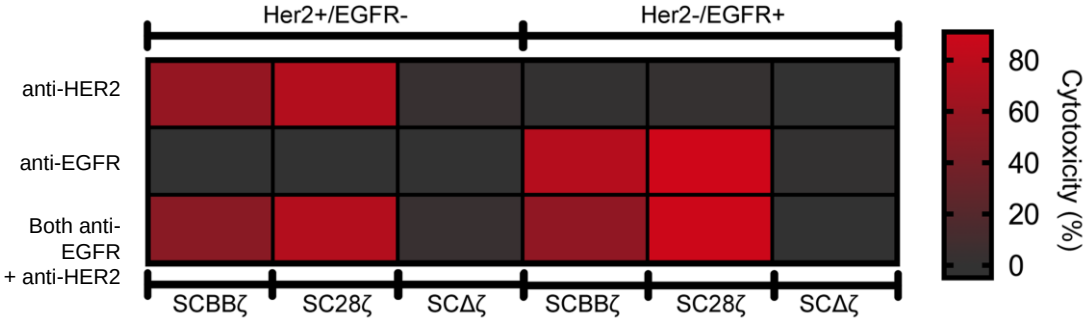
Equal arming



CAR-T equally armed with:

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

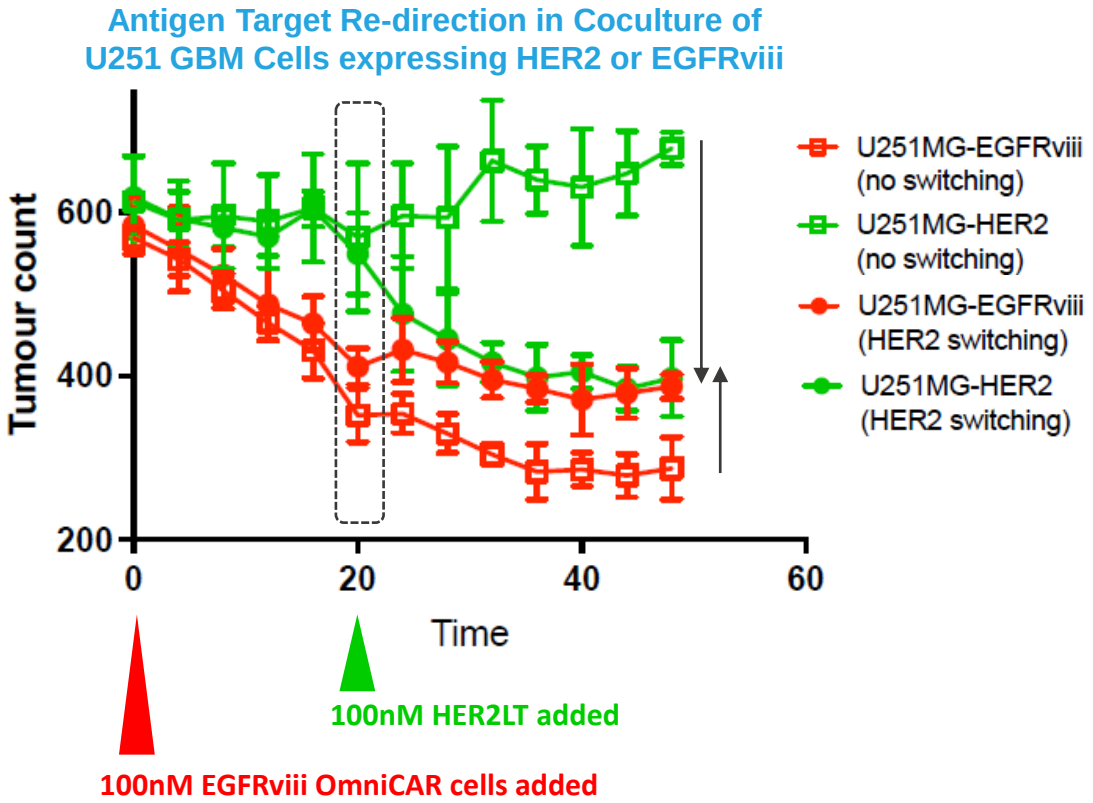
Specifically directed, at-will killing



- **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

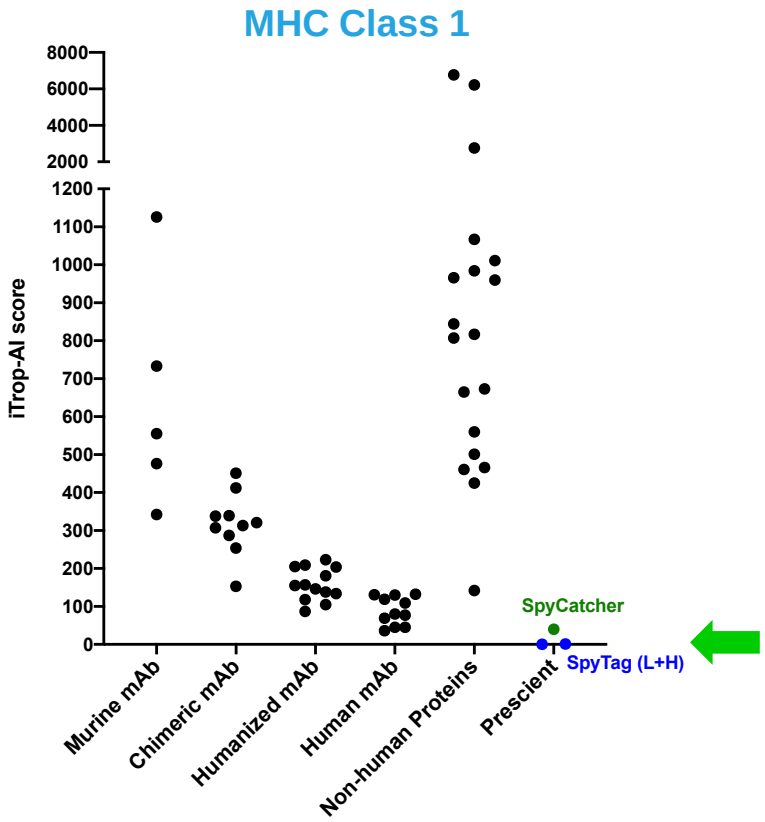
Powell, DJ et al, JACS; 2020

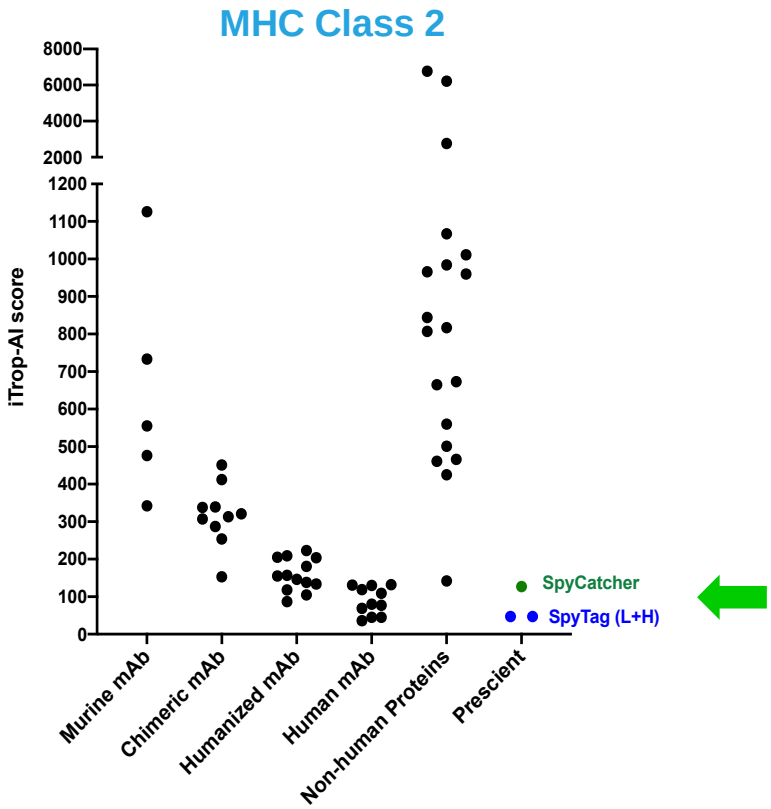
Redirection: Adding new ST-binder can re-direct cytotoxicity



- 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 immunogenicty 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:

Prescient Therapeutics

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

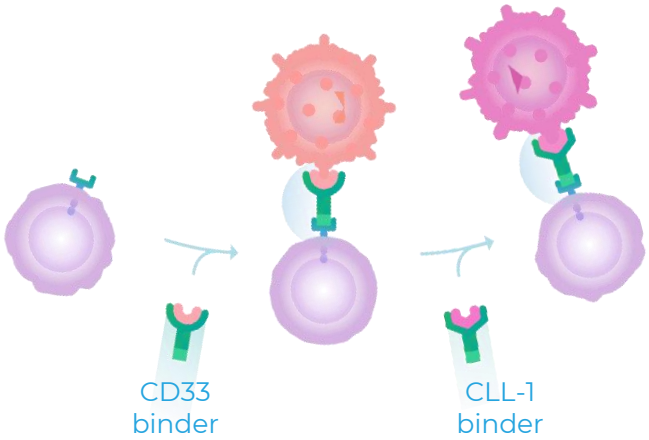
- CD33
  - Validated target in AML with approved anti-body drug conjugate (gemtuzumab ozogamicin, or Mylotarg)
  - CD33 is constantly expressed on both normal and malignant myeloid cells
  - CD33 expressed on >90% adult and childhood AML blasts and on leukemia stem cells, which have the ability to indefinitely 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

Borot, F,et al; Gene-edited stem cells enable CD33-directed immune therapy for myeloid malignancies; *PNAS*; Jun 2019, 116 (24)
Ma, H., Padmanabhan, I.S., Parmar, S. et al. Targeting CLL-1 for acute myeloid leukemia therapy. *J Hematol Oncol* 12, 41 (2019).

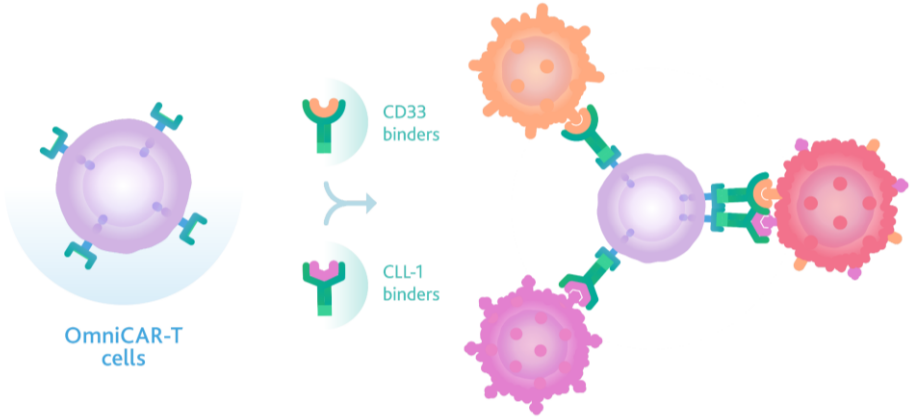
Targeting Multiple Antigens *Sequentially* OR *Simultaneously*

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

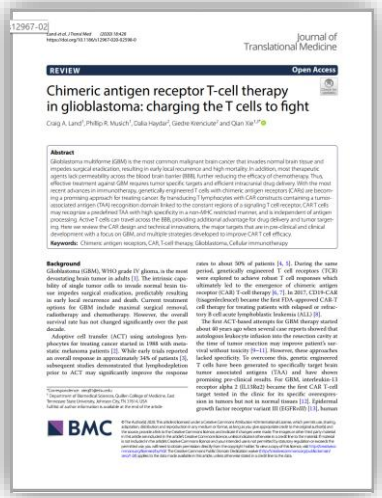
CAR-T challenges in GBM: single antigen targeting

- 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*


12967-02

Journal of Translational Medicine

REVIEW Open Access

Chimeric antigen receptor T-cell therapy in glioblastoma: charging the T cells to fight

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Abstract

Keywords: Chimeric antigen receptors, CAR, T-cell therapy, Glioblastoma, Cellular immunotherapy

Background

BMC

Land, CA, et al; Chimeric antigen receptor T-cell therapy in glioblastoma; *J Transl Med;* 2020

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

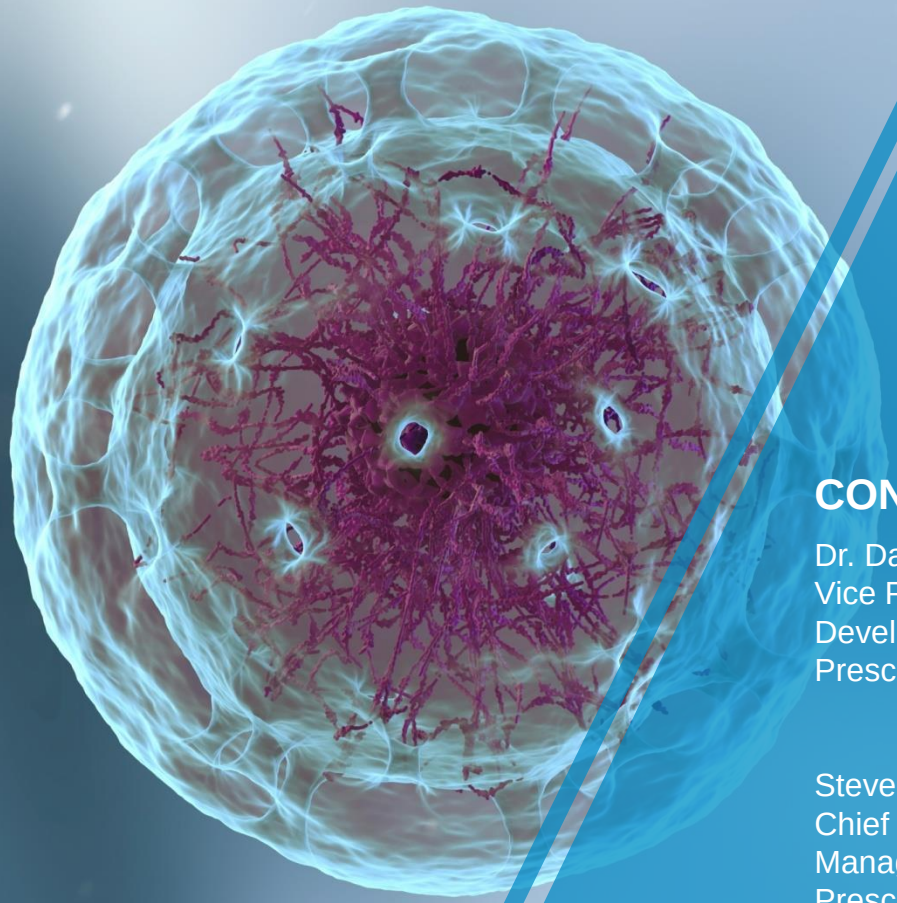
- HER2 occurs in 80% of GBM
- Linked with poor survival

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

Zh*ang*, L et al; HER2-targeted recombinant protein immuno-caspase-6 effectively induces apoptosis in HER2-overexpressing GBM cells in vitro and in vivo; *Oncology Reports*; Sept 2016
Land, CA, et al; Chimeric antigen receptor T-cell therapy in glioblastoma; *J Transl Med* 2020
Hegde M, et al. Tandem CAR T cells targeting HER2 and IL13Ralpha2 mitigate tumor antigen escape. *J ClinInvest*. 2016

Summary

- 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



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