Prescient to Present at AVID 2022 Meeting

MELBOURNE Australia, 3 May 2022: Prescient Therapeutics Limited ("Prescient"; ASX: PTX), a clinical stage oncology company developing personalised therapies to treat cancer, is today presenting at the 9th Australasian Vaccines and Immunotherapeutics Development Meeting (AVID 2022), a scientific conference attended by researchers, clinicians and biopharmaceutical industry experts that focusses on immunotherapeutic drug development.

Prescient will present selected technical progress on its OmniCAR platform during the Cellular Immunotherapy session, which will be chaired by Professor Phil Darcy of the Peter MacCallum Cancer Centre. The presentation is attached.

- Ends -

To stay updated with the latest company news and announcements, please update your details on our investor centre.

About Prescient Therapeutics Limited (Prescient)

Prescient Therapeutics is a clinical stage oncology company developing personalised medicine approaches to cancer, including targeted and cellular therapies.

Cell Therapies

OmniCAR: is a universal immune receptor platform enabling controllable T-cell activity and multi- antigen targeting with a single cell product. OmniCAR's modular CAR system decouples antigen recognition from the T-cell signalling domain. It is the first universal immune receptor allowing post- translational covalent loading of binders to T-cells. OmniCAR is based on technology licensed from Penn; the SpyTag/SpyCatcher binding system licensed from Oxford University; and other assets.

The targeting ligand can be administered separately to CAR-T cells, creating on-demand T-cell activity post infusion and enables the CAR-T to be directed to an array of different tumour antigens. OmniCAR provides a method for single-vector, single cell product targeting of multiple antigens simultaneous or sequentially, whilst allowing continual re-arming to generate, regulate and diversify a sustained T-cell response over time.

Prescient is developing OmniCAR programs for next-generation CAR-T therapies for Acute Myeloid Leukemia (AML); Her2+ solid tumours, including breast, ovarian and gastric cancers; and glioblastoma multiforme (GBM).

Cell Therapy Enhancements: Prescient has several other initiatives underway to develop new cell therapy approaches.

Targeted Therapies

PTX-100 is a first in class compound with the ability to block an important cancer growth enzyme known as geranylgeranyl transferase-1 (GGT-1). It disrupts oncogenic Ras pathways by inhibiting the activation

of Rho, Rac and Ral circuits in cancer cells, leading to apoptosis (death) of cancer cells. PTX- 100 is believed to be the only GGT-1 inhibitor in the world in clinical development. PTX-100 demonstrated safety and early clinical activity in a previous Phase 1 study and recent PK/PD basket study of hematological and solid malignancies. PTX-100 is now in a Phase 1b expansion cohort study in T cell lymphomas.

PTX-200 is a novel PH domain inhibitor that inhibits an important tumour survival pathway known as Akt, which plays a key role in the development of many cancers, including breast and ovarian cancer, as well as leukemia. Unlike other drug candidates that target Akt inhibition, PTX-200 has a novel mechanism of action that specifically inhibits Akt without non-specific kinase inhibition effects. This highly promising compound has previously generated encouraging Phase 2a data in HER2-negative breast cancer and Phase 1b in recurrent or persistent platinum resistant ovarian cancer, with a Phase 1b/2 trial currently underway in relapsed and refractory AML.

The CEO and Managing Director of Prescient Therapeutics Limited has approved the release of this announcement.

Find out more at www.ptxtherapeutics.com or connect with us via Twitter @PTX_AUS and LinkedIn.

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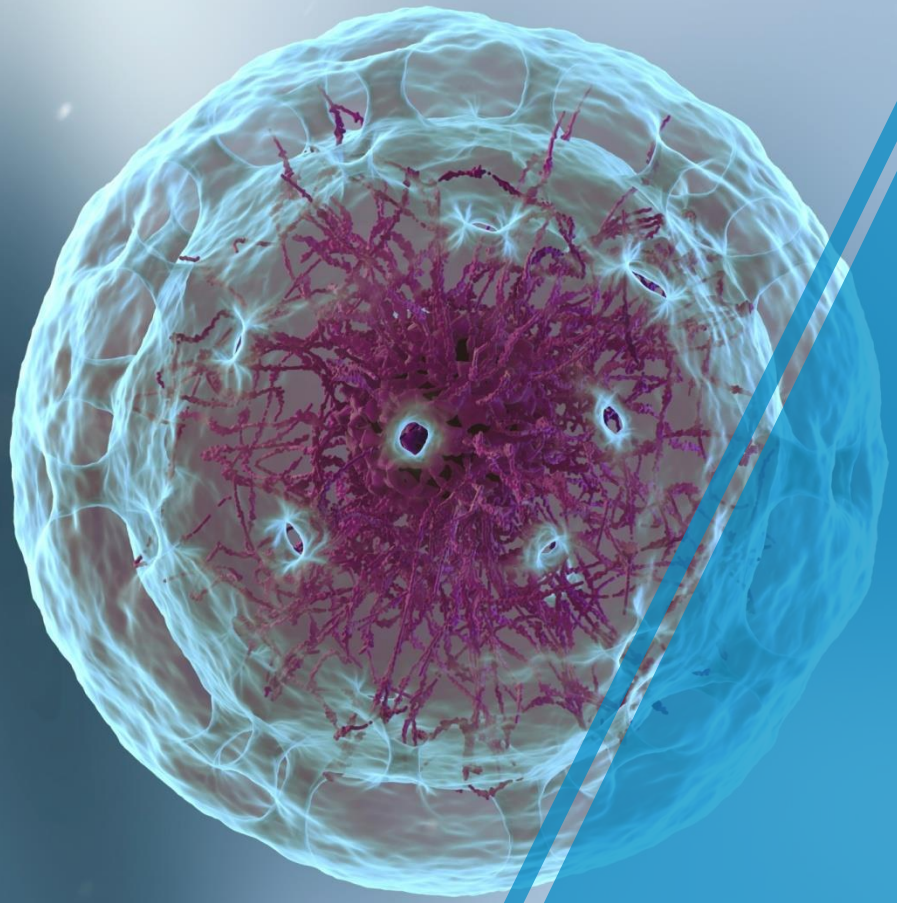
Supplemental COVID-19 Risk Factors

Please see our website : Supplemental COVID-19 Risk Factors



Prescient Therapeutics

OmniCAR –

A Covalent Universal Immune Receptor Platform for CAR-T Therapy

AVID2022

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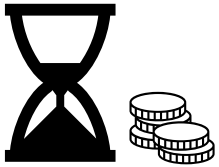
Key Challenges Confronting the field of CAR-T

Prescient Therapeutics



Time and Cost

of delivering treatment

Targets

Finding targets that work;

Antigen heterogeneity - esp. in solid tumours

Safety

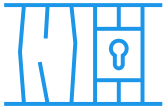
CAR-T can have serious safety concerns

Exhaustion

Persistent stimulation of CAR-Immune cells leads to exhaustion

No Control

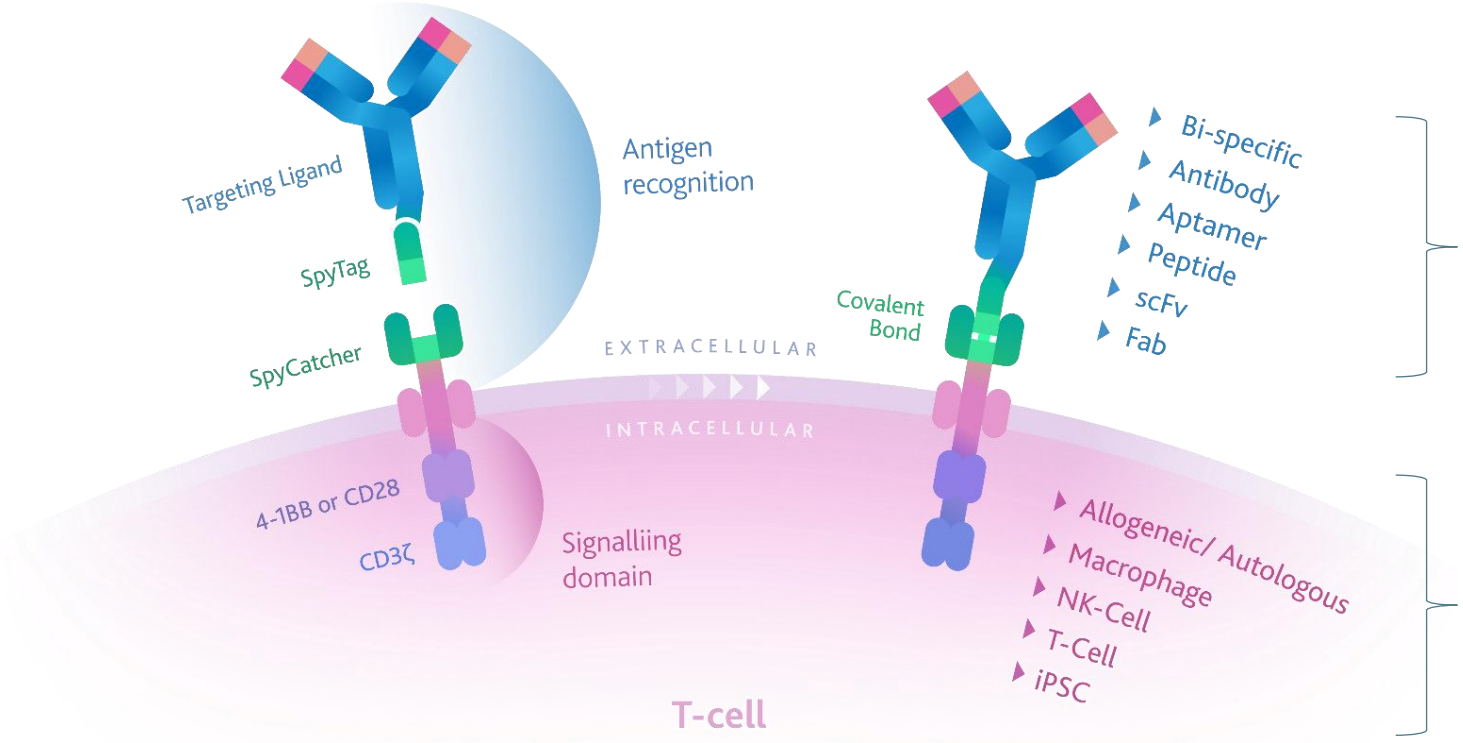
Clinicians have no control of cells post infusion



Escape

Antigen loss leads to relapse

The modularity of the OmniCAR platform



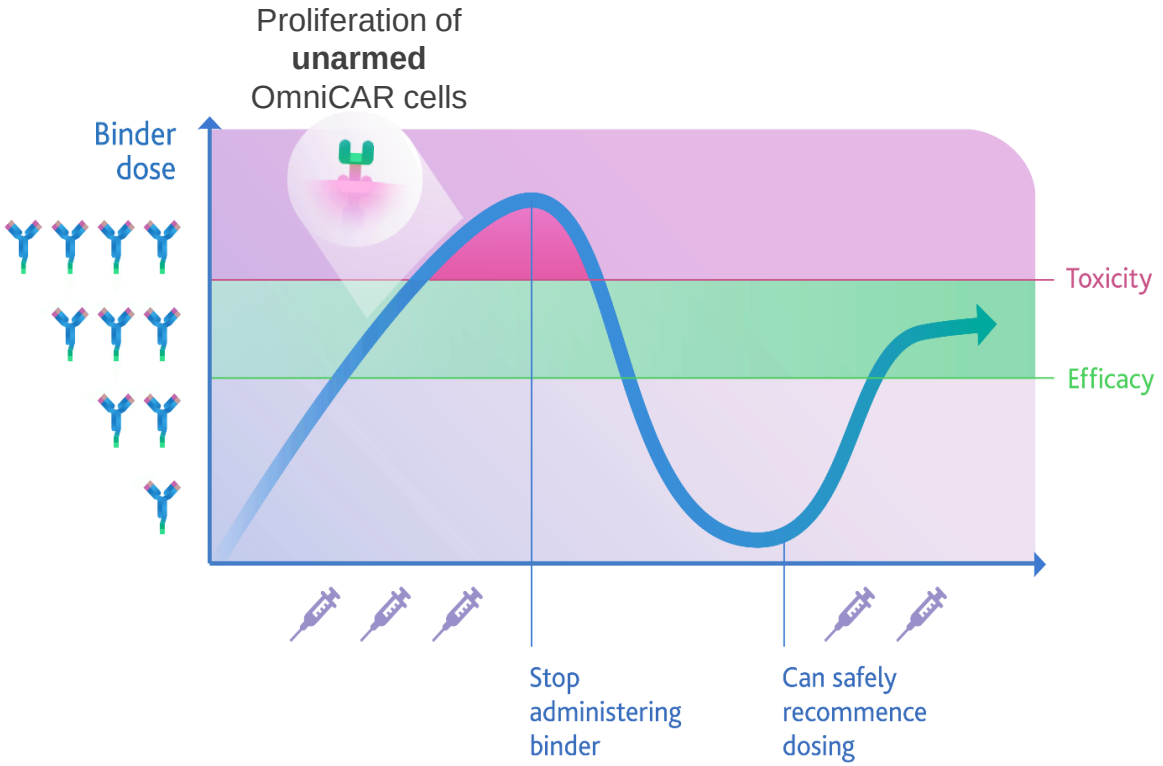
OmniCAR can use any type of **targeting ligand…**

…with any **immune cell**

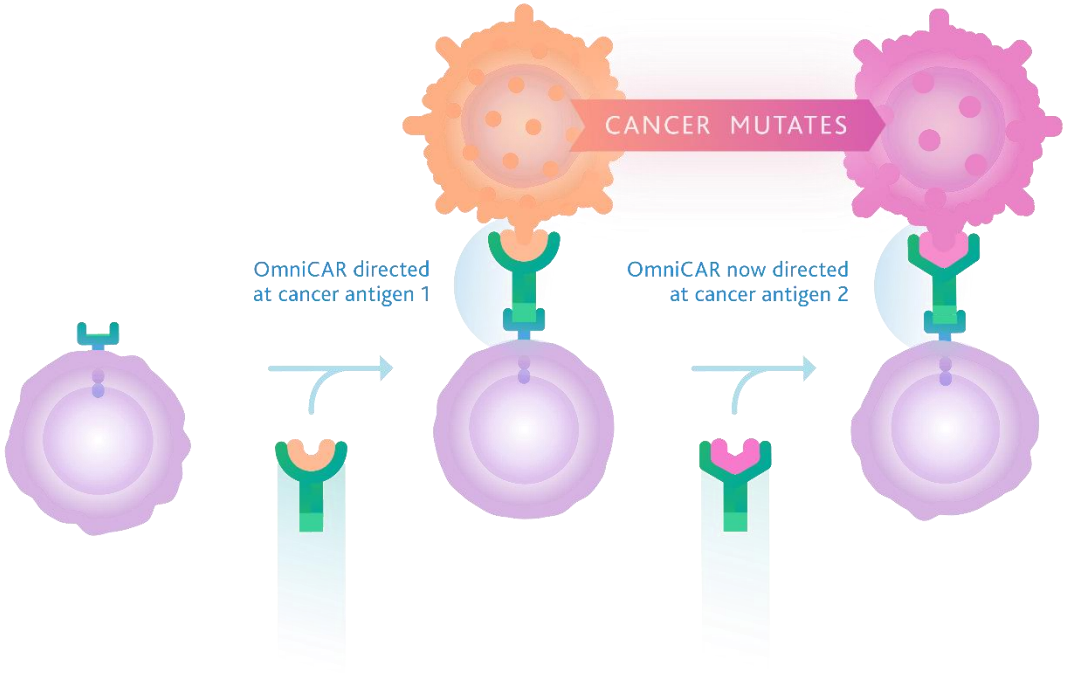
Adapted from Powell, DJ et al, JACS; 2020

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**

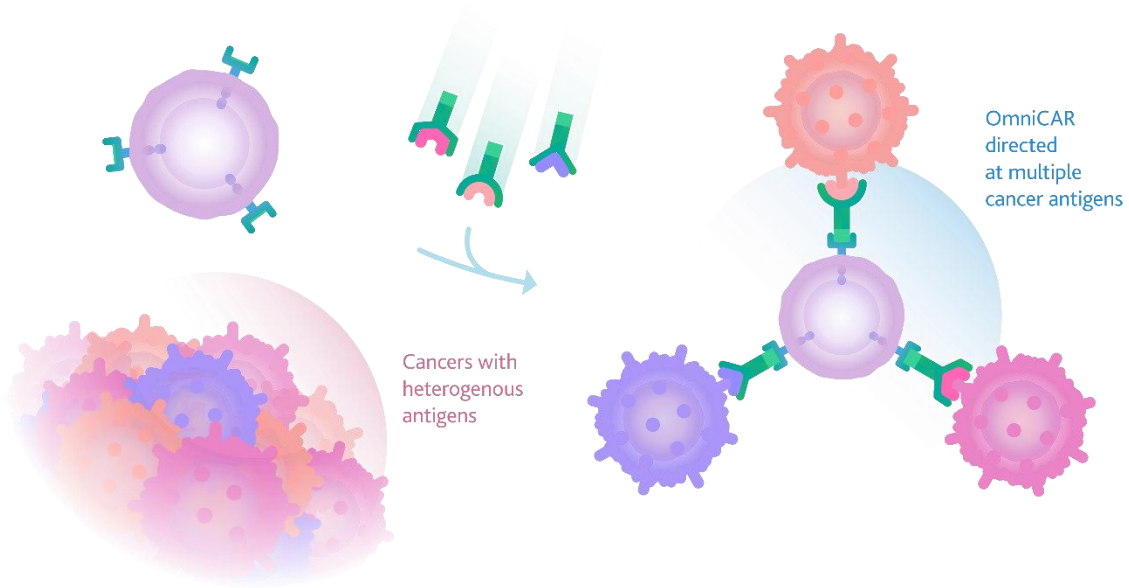


Target Multiple Antigens *Sequentially*



- 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

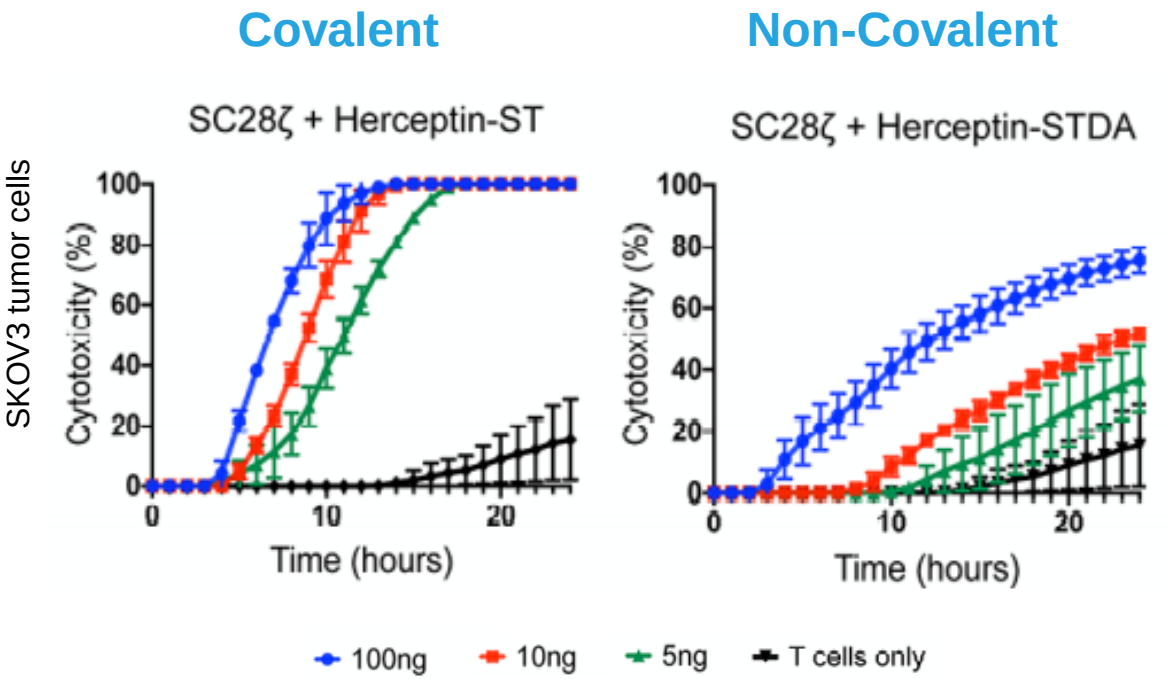
Target Multiple Antigens *Simultaneously*



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

Covalent Binding:

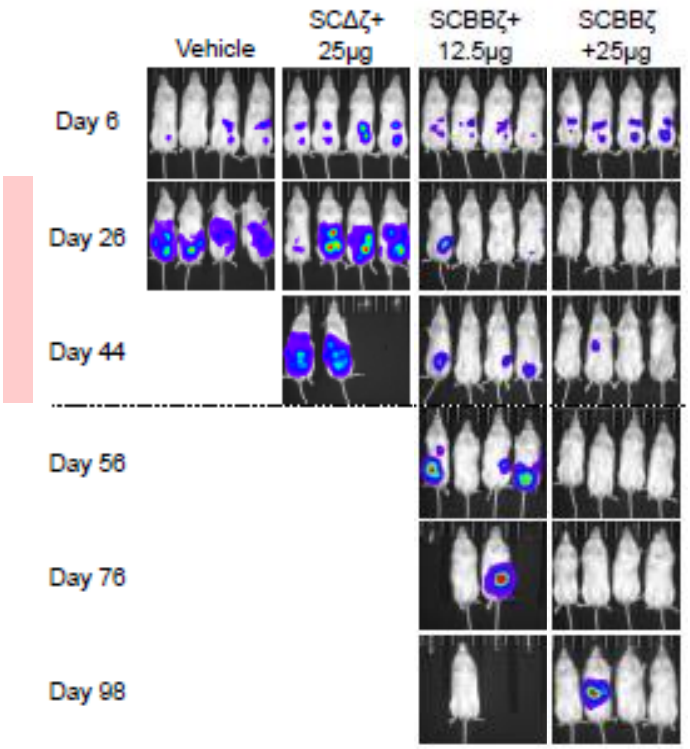
Superior tumor killing & other advantages



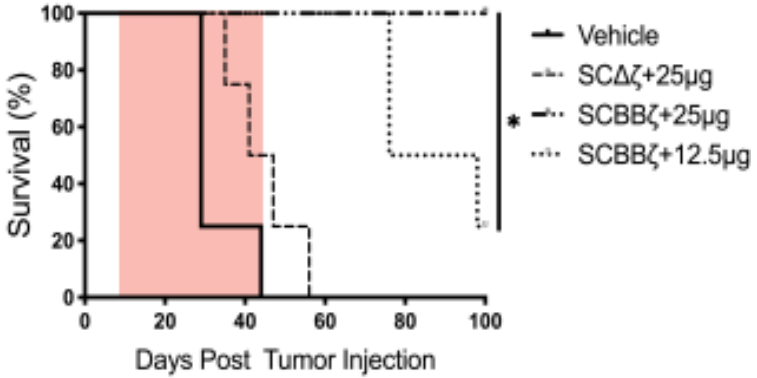
- **Covalent** binding improves SpyCatcher T-cell **loading and tumour cell lysis**
- Covalent binding has additional advantages in:
  - Efficacy
  - Predictability
  - Clinical utility
  - Regulatory considerations

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

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**

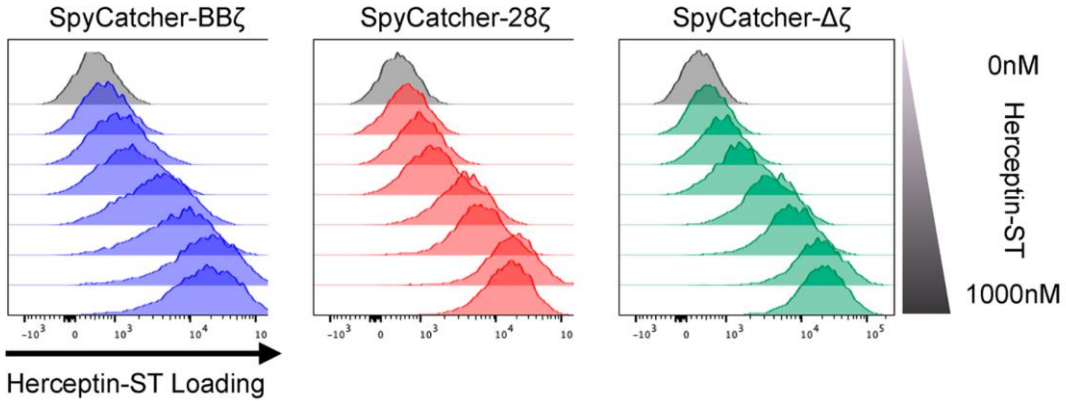


Powell, DJ et al, JACS; 2020

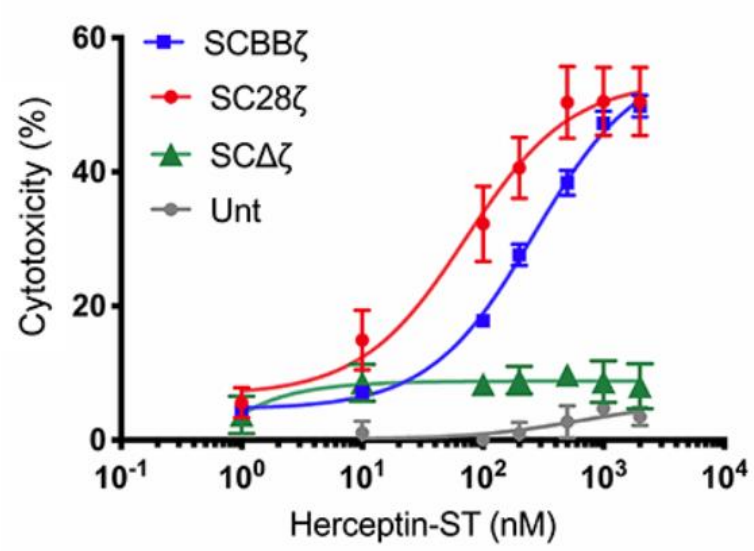
= dosing window of binder

Flexible Loading and Dose-Dependent Lysis

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



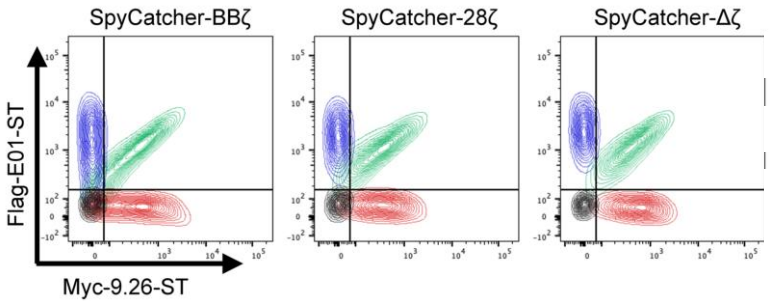
- Increasing targeting ligand concentration results in increased lytic capacity



Powell, DJ et al, JACS; 2020

Equal Arming & Equal Tumour Killing

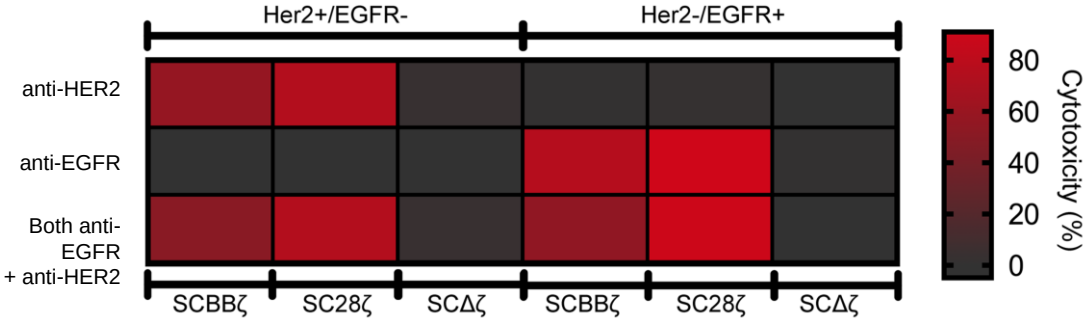
Equal arming



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

V3 of OmniCAR: Stronger, Faster, Better

Prescient Therapeutics

Approaching infinite affinity through engineering of peptide–protein interaction

Anthony H. Keeble[a,1], Paula Turkki[b,c,1], Samuel Stokes[a], Irsyad N. A. Khairil Anuar[a], Rolle Rahikainen[a], Vesa P. Hytönen[b,c,2], and Mark Howarth[a,2]

[a]Department of Biochemistry, University of Oxford, OX1 3QU Oxford, United Kingdom; [b]BioMediTech, Faculty of Medicine and Health Technology, Tampere University, 33520 Tampere, Finland; and [c]Fimlab Laboratories, 33520 Tampere, Finland

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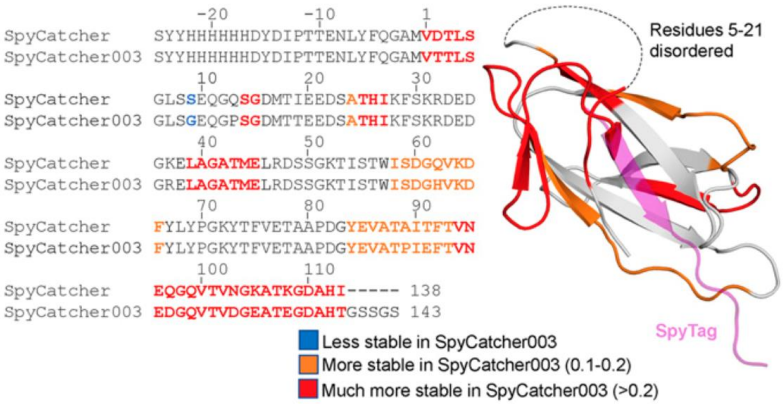
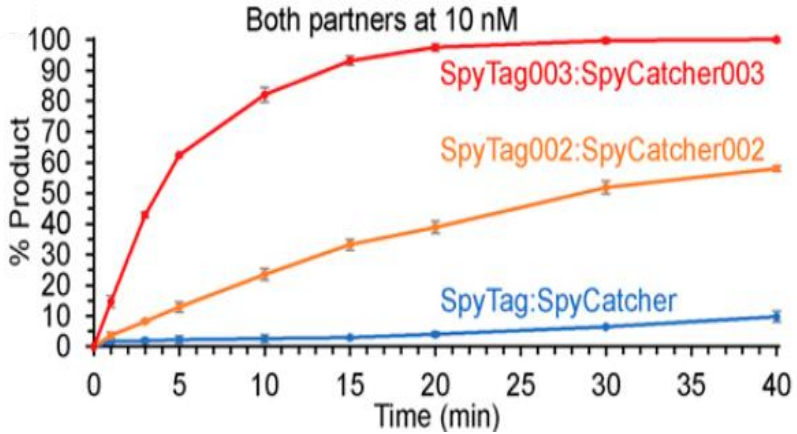
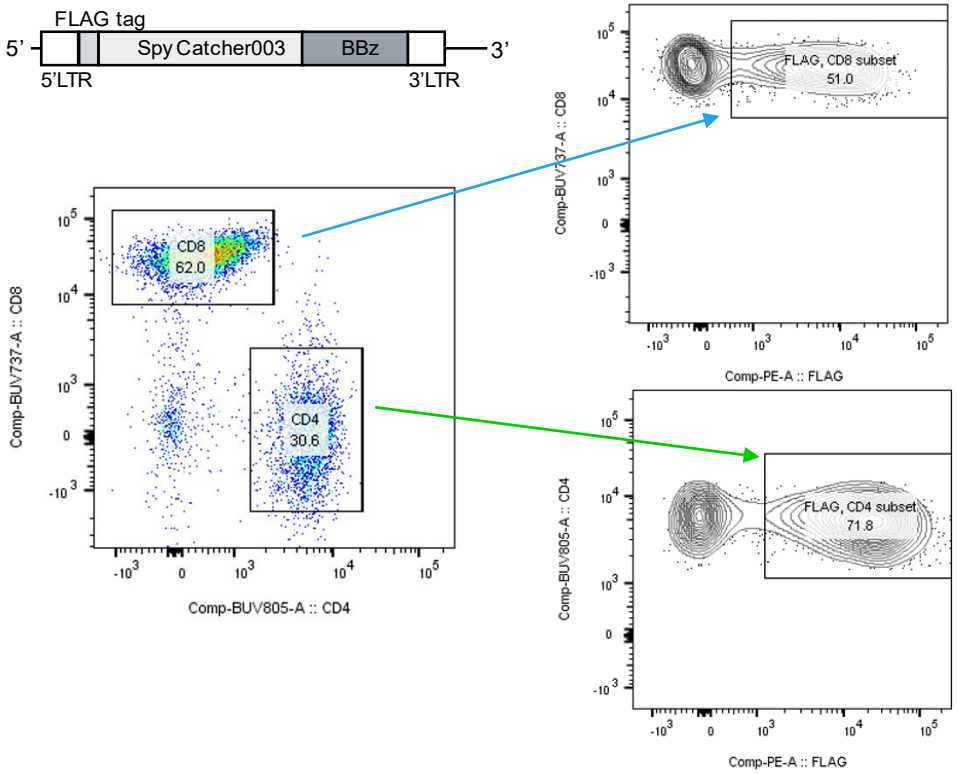




Reaction rates of v003 compared to previous versions

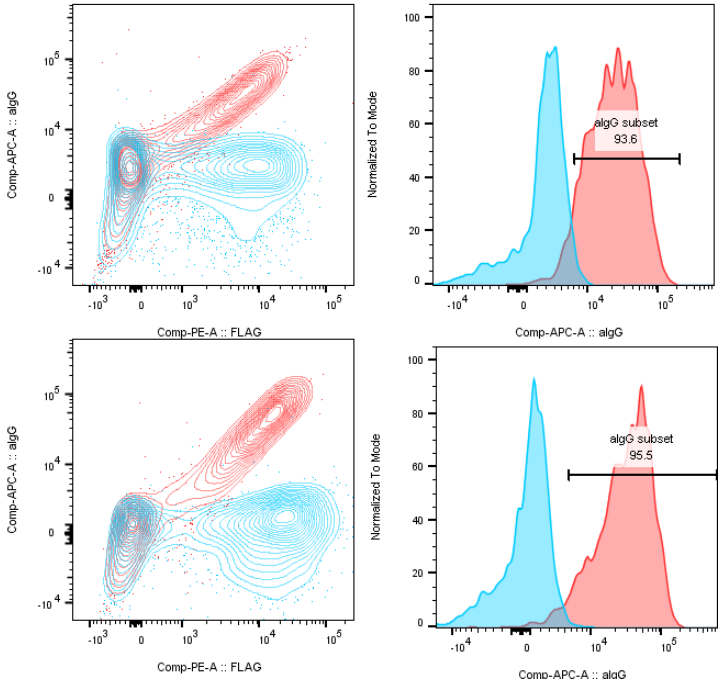
OmniCAR V3 construct

N-FLAG



Highly efficient arming

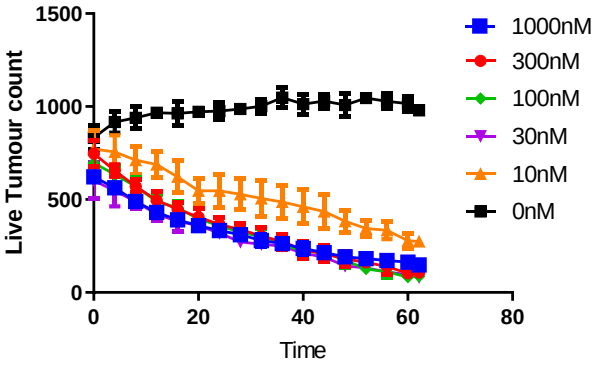
Almost 100% arming within 1 hr

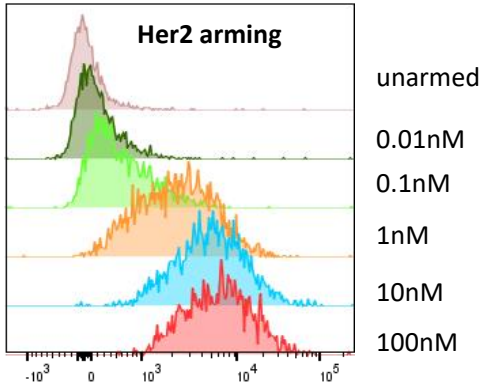


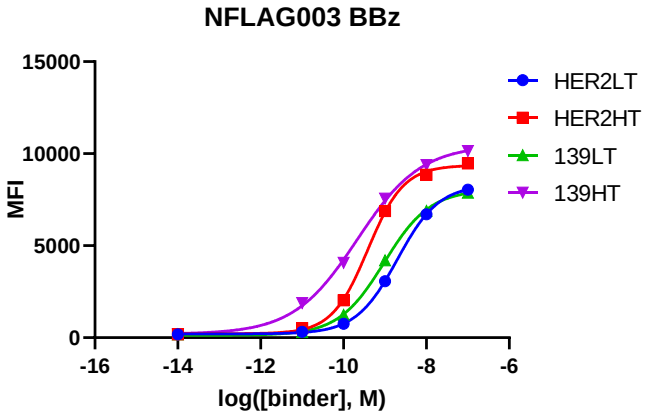
HER2 armed (1hr) + aIgG + aFLAG

Dose response: High potency with less binder required

2:1 Her2 armed OmniCAR vs U251-Her2+ cells







EC50 in the very low nM range

	HER2LT	HER2HT	139LT	139HT
NFlag003 EC50 (nM)	2.001	0.3878	0.9588	0.2147

Potency of OmniCAR comparable to conventional CAR

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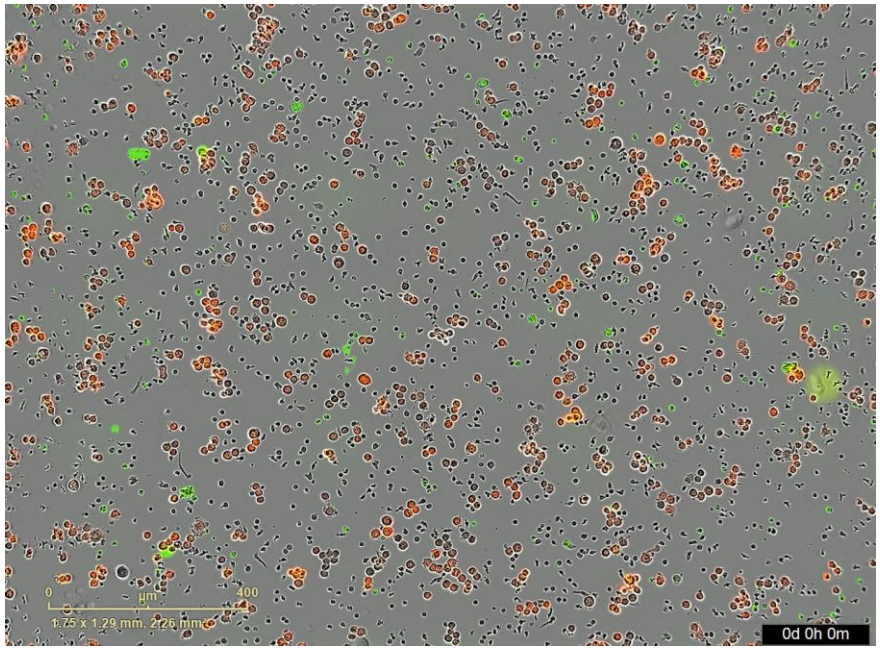
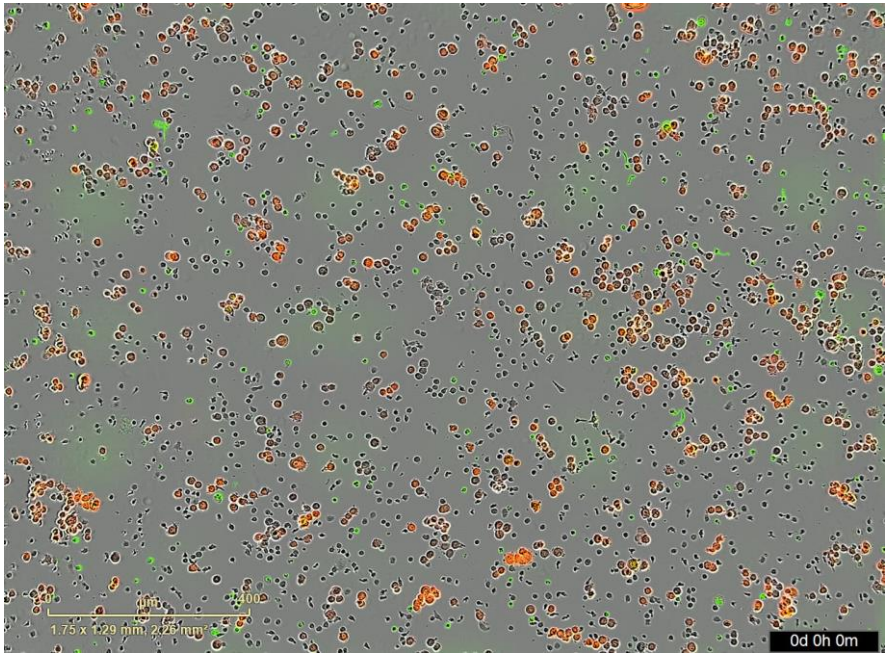
Conventional CAR T (Her2)

MCF7 (2:1 ratio) **Caspase 3/7**

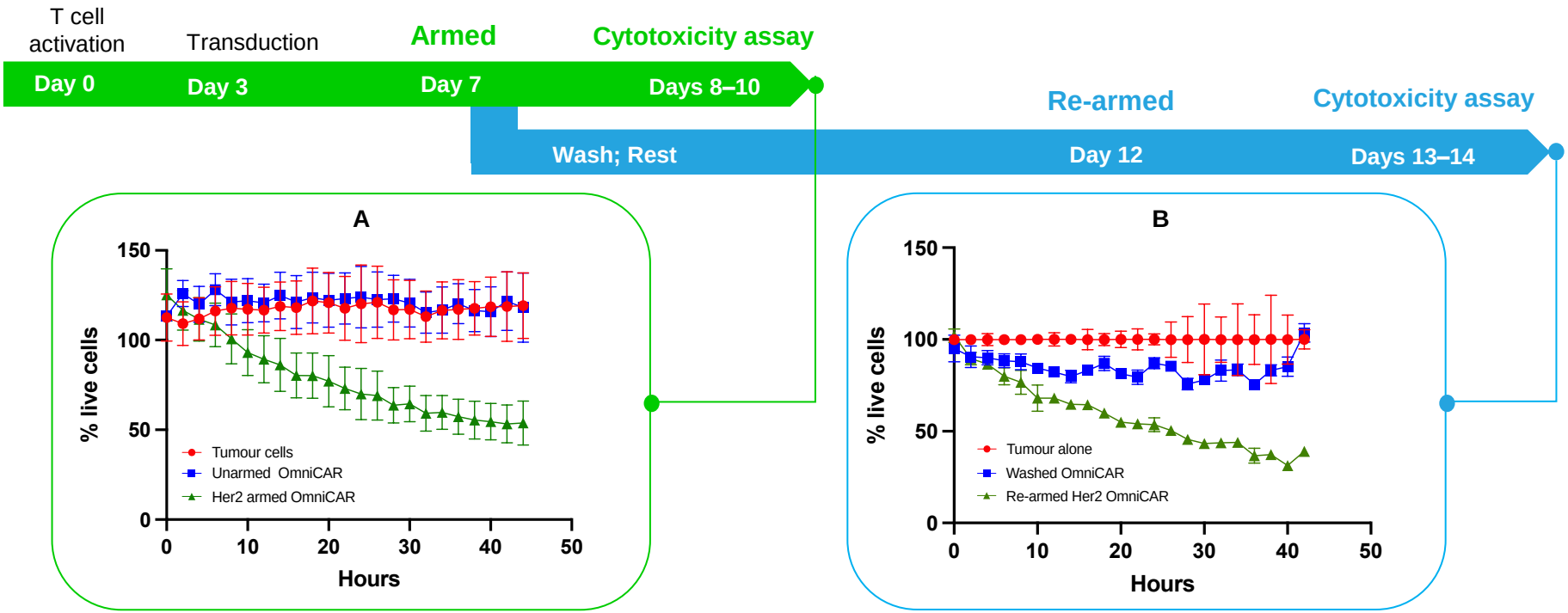


OmniCAR T (Her2)

MCF7 (2:1 ratio) **Caspase 3/7**



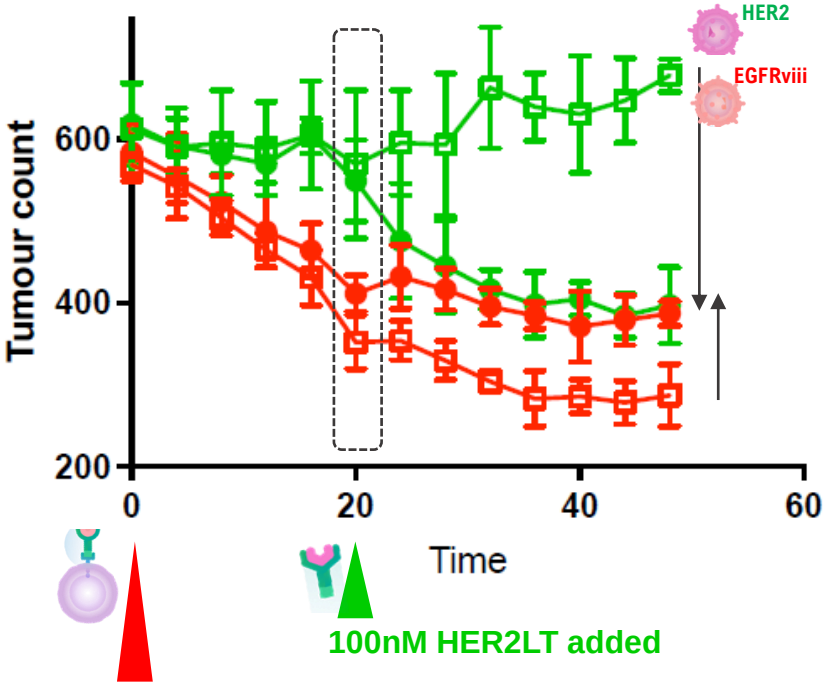
Re-Arming: OmniCAR Her2 can be Re-Armed



- OmniCAR T cells can be re-armed
- Re-arming results in **same levels and kinetics of cytotoxicity** as pre-armed

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

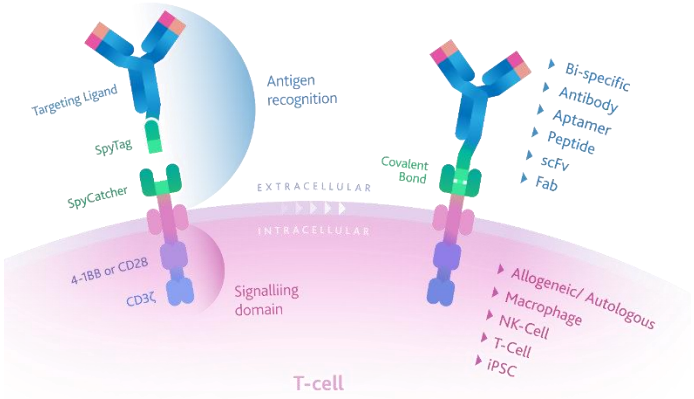
Antigen Target Re-direction in Coculture of U251 GBM Cells expressing HER2 or EGFRviii



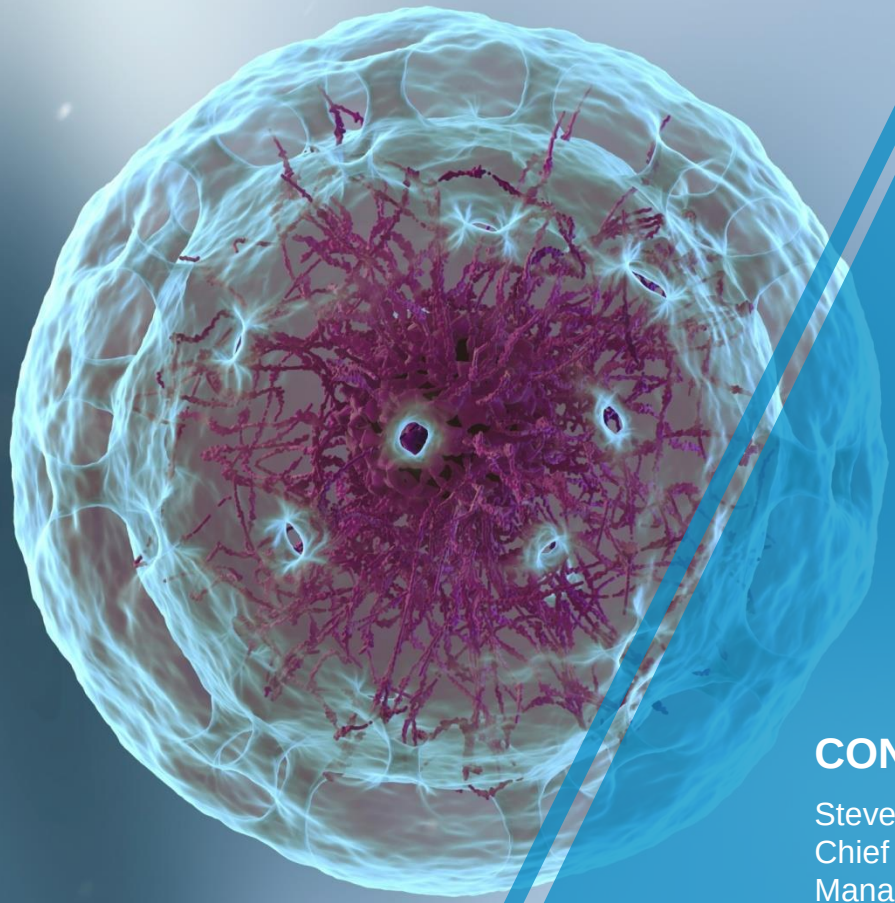
- Rapid cytotoxicity to EGFRviii
- **Rapid switching and cytotoxicity** against HER2+ tumours upon administration of new binder
- OmniCAR cells can be **re-directed to different antigens** upon administration of a different SpyTagged binder **without new cells**

Summary and Conclusions

- OmniCAR is a universal, modular CAR system
- Agnostic on antigen; targeting ligand; immune cell
- Modularity does not appear to compromise cytotoxicity
- Covalent modularity enables:
  - Control of cytotoxicity
  - Predictable loading
  - Multi-valence
  - Target re-redirection



Special thanks to Phil Darcy and his team at Peter Mac



Prescient Therapeutics

Thank you.

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