



PTX-100 investor update and additional patient response

MELBOURNE Australia, 2 November 2022: Prescient Therapeutics (ASX: PTX), a clinical stage oncology company developing personalised therapies to treat cancer, will be holding an investor briefing on Wednesday 2nd November at 12pm (AEDT).

CEO and Managing Director Steven Yatomi-Clarke will provide a company update, outlining Prescient's cutting-edge pipeline and an update from the PTX-100 Phase 1b expansion cohort study.

Since Prescient's recent PTX-100 trial update on 25 October 2022, there has been an additional reported clinical response, with a patient with relapsed peripheral T cell lymphoma (PTCL) reporting a partial response. The increasing clinical responses observed in this study provides growing confidence in the potential for PTX-100 in this area of poorly met medical need.

To register for the investor briefing, visit this page:

<https://prescienttherapeutics.investorportal.com.au/investor-briefing/>

A copy of the investor presentation to be presented 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).

CellPryme-M: Prescient's novel, ready-for-the-clinic, CellPryme-M technology enhances adoptive cell therapy performance by shifting T and NK cells towards a central memory phenotype, improving persistence, and increasing the ability to find and penetrate tumours. CellPryme-M is a 24-hour, non-disruptive process during cell manufacturing. Cell therapies that could benefit from additional productivity in manufacturing or increased potency and durability in-vivo, would be good candidates for CellPryme-M.

CellPryme-A: CellPryme-A is an adjuvant therapy designed to be administered to patients alongside cellular immunotherapy to help them overcome a suppressive tumour microenvironment. CellPryme-A significantly decreases suppressive regulatory T cells; increases expansion of CAR-T cells in vivo; increases tumour penetration of CAR-T cells. CellPryme-A improves tumour killing and host survival of CAR-T cell therapies, and these benefits are even greater when used in conjunction with CellPryme-M pre-treated CAR-T cells.

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, where it has shown encouraging efficacy signals and safety.

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 is currently in a Phase 1b/2 trial in relapsed and refractory AML, where it has resulted in 4 complete remissions so far. PTX-200 previously generated encouraging Phase 2a data in HER2-negative breast cancer and Phase 1b in recurrent or persistent platinum resistant ovarian cancer.

Find out more at www.ptxtherapeutics.com or connect with us via Twitter [@PTX_AUS](https://twitter.com/PTX_AUS) and [LinkedIn](https://www.linkedin.com/company/ptxtherapeutics).

The Board of Prescient Therapeutics Limited has approved the release of this announcement.

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Supplemental COVID-19 Risk Factors

Please see our website: [Supplemental COVID-19 Risk Factors](#)



IN FRONT OF THE BIGGEST WAVE IN ONCOLOGY

November 2022

Disclaimer and safe harbor



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Corporate snapshot



ASX Ticker	PTX
Total Issued Capital	705 M shares
Listed Options	85.4 M
Unlisted Options	38.3 M
Share Price ¹	A\$0.17
Market Capitalisation¹	A\$120 M
Market Cap fully diluted¹	A\$140 M
Cash Position²	A\$21M
Top 20 Own	16%



1 - AS AT 24 OCT 2022

2 - UNAUDITED, POST OCT 2022 CAPITAL RAISING

Investment Highlights



World class pedigree.

We license from the best;
and work with the best



THE UNIVERSITY OF TEXAS



Many shots on goal for
substantial value creation



2 Cell Therapy platforms

Internal & external opportunities



2 Targeted Therapies

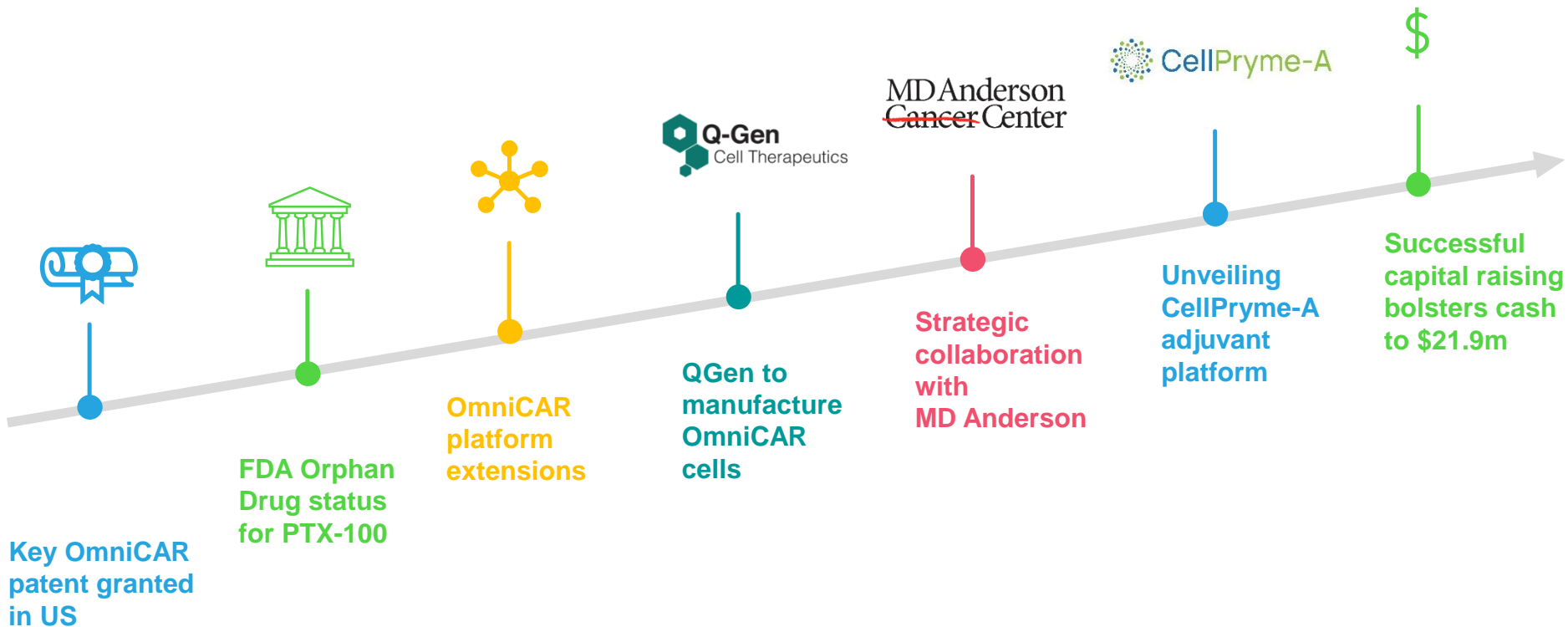
in clinical trials, showing activity



Upcoming newsflow

from multiple programs

Very productive Sept quarter



4 Innovative Personalised Oncology Assets

Cell therapy platforms



Next-gen CAR-T therapies

CD33/CLL-1
Acute Myeloid
Leukaemia

Her2/EGFRviii
Glioblastoma
Multiforme

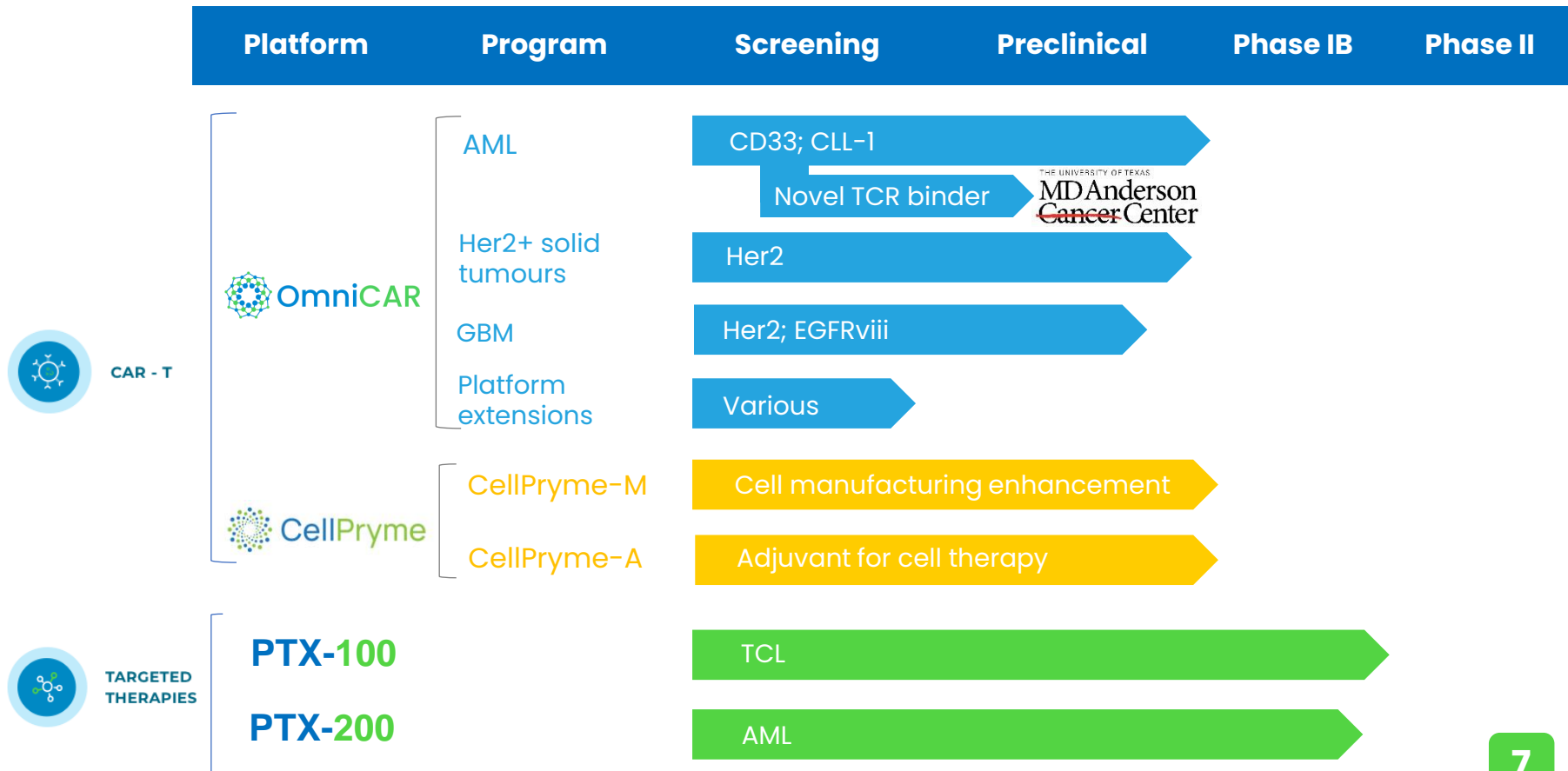
Her2
Breast, Ovarian &
Gastric cancers

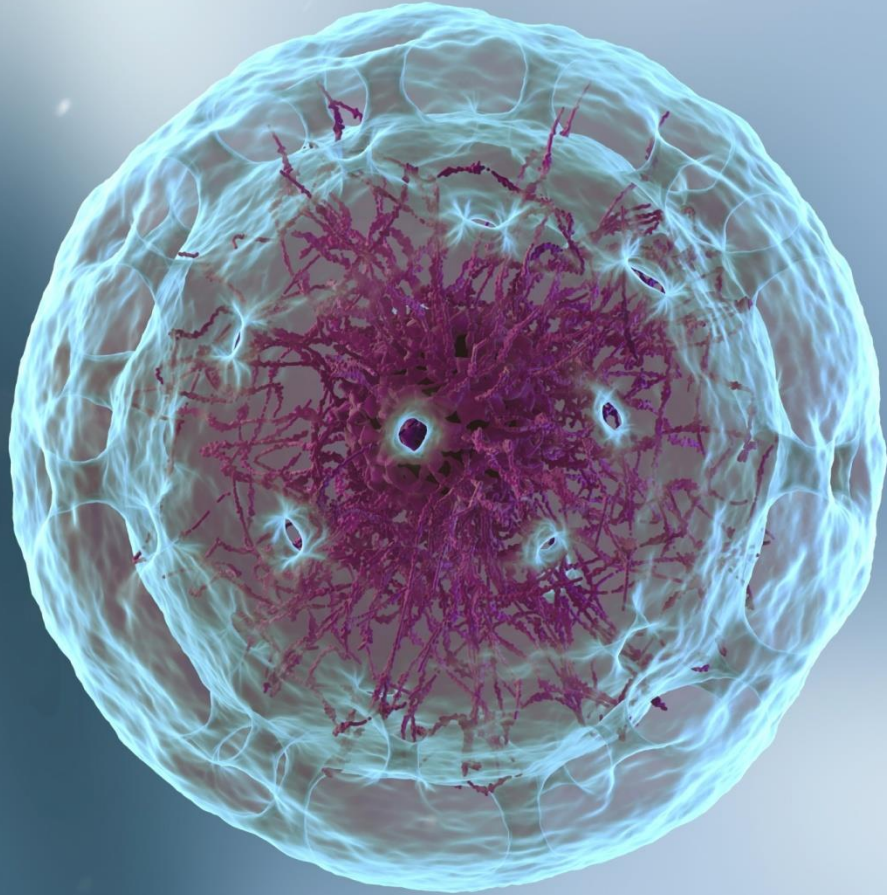
Targeted therapies

PTX-100
Ras / Rho
inhibitor

PTX-200
Novel AKT
inhibition

Innovative pipeline in personalised medicine





TARGETED THERAPIES

PTX-100

FIRST IN CLASS
RAS PATHWAY INHIBITOR

PTX-100 Phase 1B Summary

- Licensed from Yale University
- Targeting cancers predisposed to Ras & Rho mutations

Phase 1b Expansion cohort in T-cell lymphomas (TCL)

- Excellent safety profile
- Encouraging signal in TCL
 - Responses
 - Time on therapy
- Granted Orphan Drug Designation by US FDA for Peripheral TCL



Licensed from



Principal Investigator

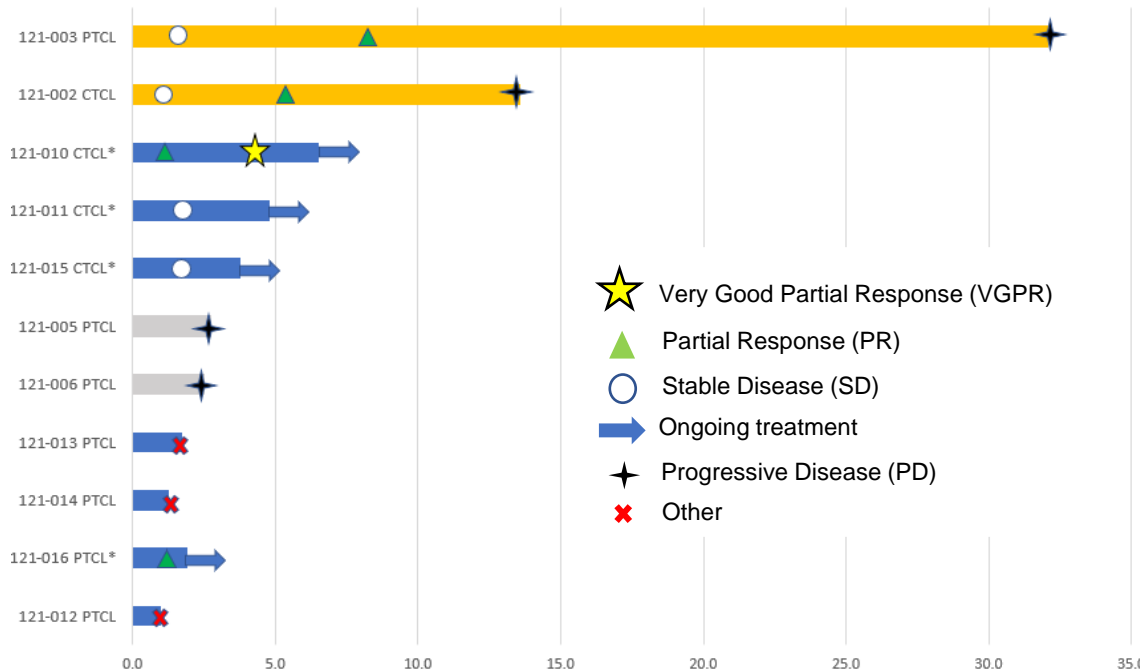


Professor H. Miles Prince, AM



Trial update: continued encouraging responses & time on treatment

CLINICAL REPOSSES & MONTHS ON TREATMENT



- 1 VGPR (ongoing)
- 3 PRs (1 ongoing)
- 2 SDs (both ongoing)

- 2 PD
- 3 withdrawn

PTX-100 dose

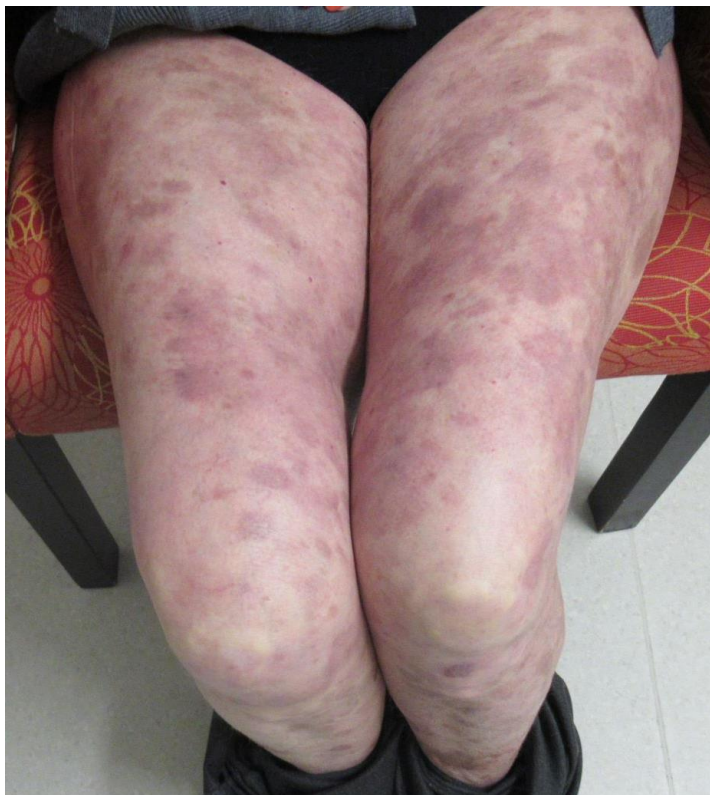
500 mg/m²

1000 mg/m²

2000 mg/m²

	121-012 PTCL	121-016 PTCL*	121-014 PTCL	121-013 PTCL	121-006 PTCL	121-005 PTCL	121-015 CTCL*	121-011 CTCL*	121-010 CTCL*	121-002 CTCL	121-003 PTCL
■ *still on study	1.0	1.9	1.3	1.8	2.5	2.8	3.8	4.8	6.5	13.6	32.1

Before



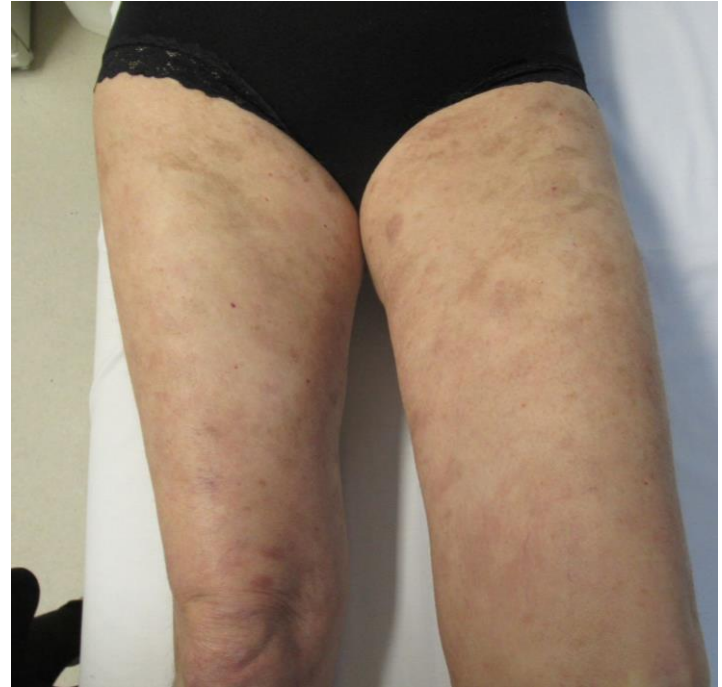
After



Before

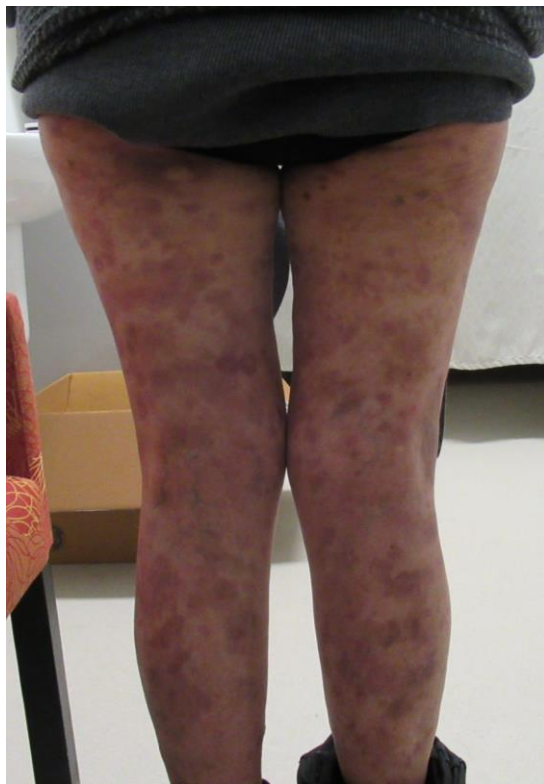


After



ASX: PTX

Before

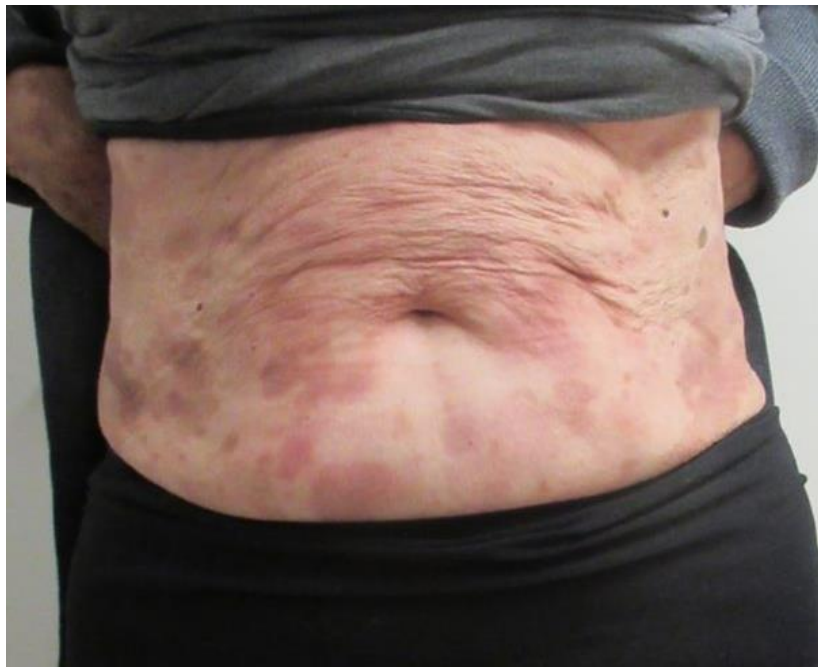


After



ASX: PTX

Before



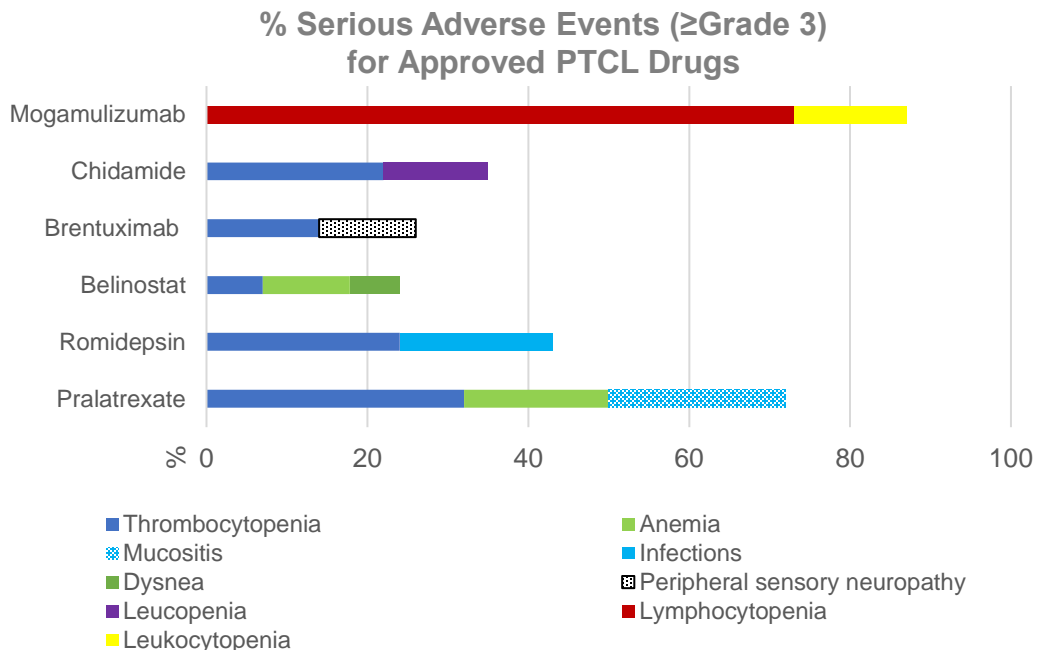
After



ASX: PTX

Favourable safety profile compared to peers

Approved PTCL drugs have troublesome safety profiles



PTX-100 HAS AN EXCELLENT SAFETY PROFILE

- No serious adverse events related to PTX-100
- Suits fragile patient population
- Good candidate for combination therapy

Now in Expansion Cohort for TCL

- 8 – 12 patients with r/r T cell lymphoma
- Expanding number for CTCL patients in light of responses
- Potential bridge to registration study
- Focussing on sweet spot in an area of considerable unmet need
- Shortest path to market

Case Study

- pralatrexate (Folotyn[®])
- Approved for PTCL
 - 5,600 cases/year in US
- US\$450,540 per patient, per year

FOLOTYN
(pralatrexate injection) 



PTX-200

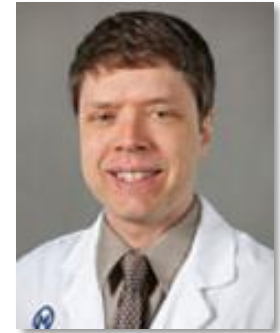
NOVEL AKT INHIBITION

Phase 1B trial underway: Acute Myeloid Leukemia



- Building upon encouraging Phase 1 results with PTX-200 (monotherapy)
- PI Professor Jeff Lancet at Moffitt, Key Opinion Leader in AML
- 24 patients with cytarabine held constant at 200-400 mg/m² as continuous infusion
 - 4 patients with CR/CRi so far
 - 1 patient with PR
- Currently treating expansion cohort at 45 mg/m²
- Granted Orphan Drug Designation by US FDA

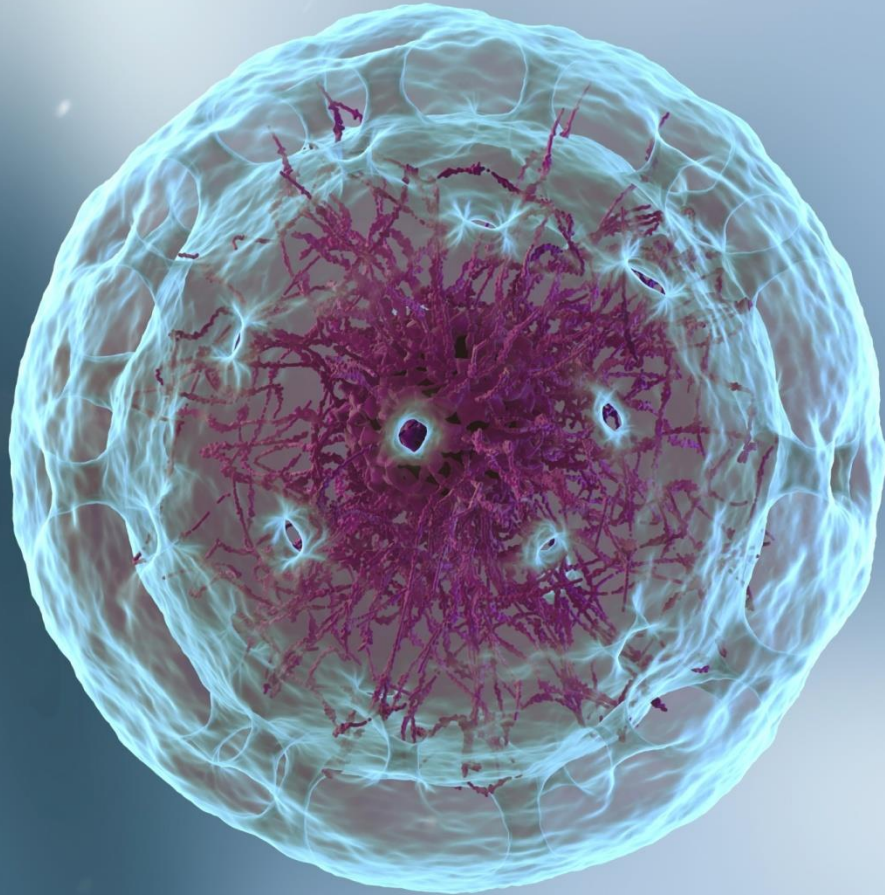
Principal Investigator



Jeffrey E Lancet, M.D.

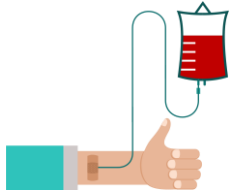


CR: COMPLETE REMISSION
CRI: COMPLETE RESPONSE WITH INCOMPLETE HEMATOLOGIC RECOVERY
PR: PARTIAL RESPONSE



CELL THERAPY PLATFORMS

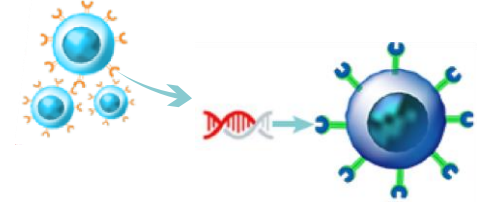
The CAR-T process



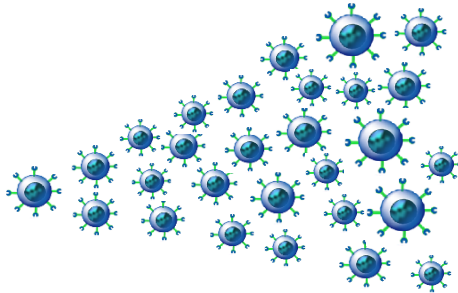
1 Blood is collected from the patient



2 T-Cells are isolated



3 T-Cells are genetically altered to have cancer-recognising receptors (CARs)

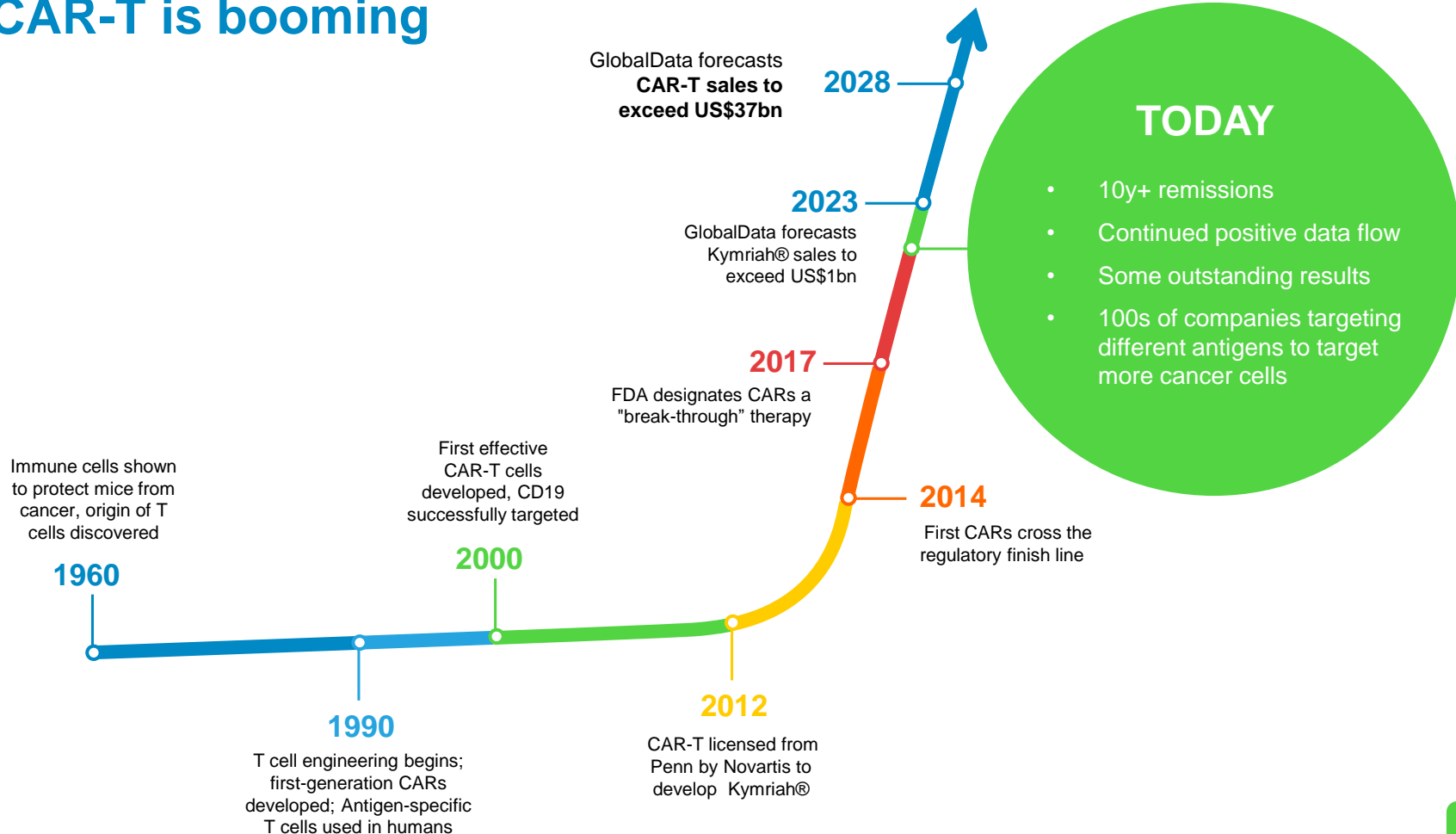


4 Millions of CAR-T cells are grown



5 CAR-T cells are administered to the patient

CAR-T is booming



Penn is a pioneer and world leader in CAR-T



- Novartis licensed CAR-T technology from Penn in 2012
- Kymriah® became the first CAR-T therapy approved by the FDA
- Used for certain blood cancers
- Cost of treatment in excess of \$500,000 per treatment
- GlobalData forecasts Kymriah® sales to exceed US\$1 billion in 2023



CAR-T's key challenges

Challenge



Safety / Control

No control post infusion



Targeting

Difficulties with targeting,
antigen heterogeneity



Escape

Difficulties with mutating antigens



Production efficiency

Cost prohibitive & slow



Exhaustion

Cells run out of steam



Trafficking

Cells cannot find their way



Tumor penetrance

Protective layer around tumor



Tumor microenvironment

Suppresses immune cells

Unsafe











Less effective

Not sustainable

Too expensive

Don't last

Platforms to overcome CAR-T's key challenges

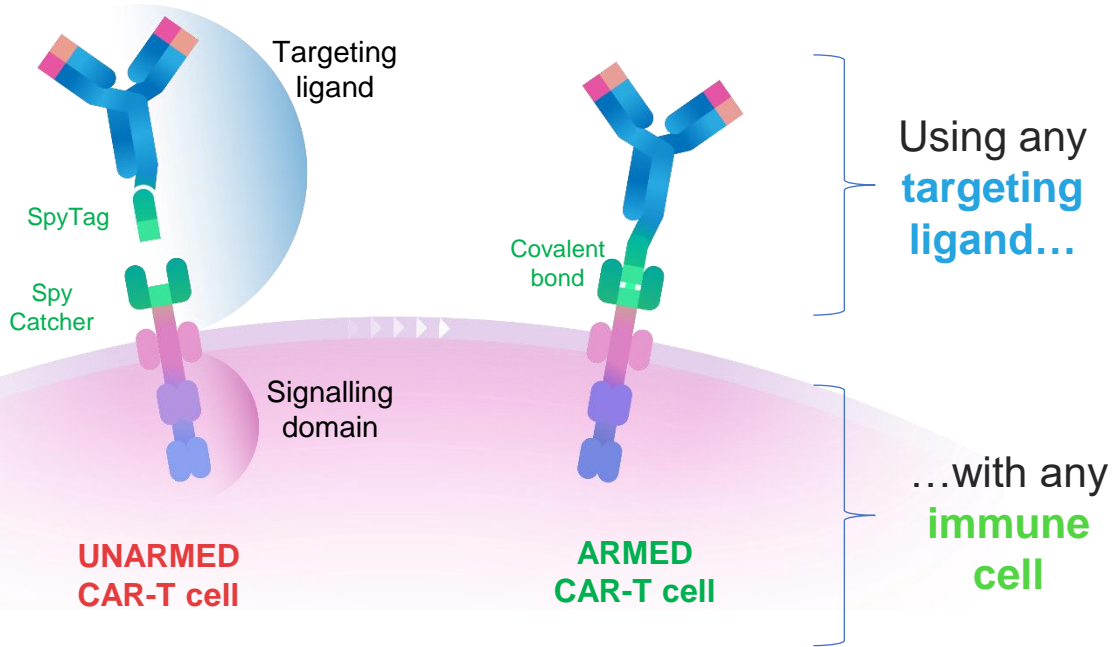
	Challenge	 OmniCAR	 CellPrime		
	Safety / Control	No control post infusion	✓	-	
	Targeting	Difficulties with targeting, antigen heterogeneity	✓	-	Safe
	Escape	Difficulties with mutating antigens	✓	-	Effective
	Production efficiency	Cost prohibitive & slow	✓	-	Sustainable
	Exhaustion	Cells run out of steam	✓	✓	Affordable
	Trafficking	Cells cannot find their way	✓	✓	
	Tumor penetrance	Protective layer around tumor	✓	✓✓	Enduring
	Tumor microenvironment	Suppresses immune cells	✓	✓✓	



OmniCAR

**Universal, Next Gen
CAR-T Platform**

OmniCAR: flexible, modular CAR platform



Associate Professor
Daniel J. Powell, Jr



Professor
Andrew Tsourkas



UNIVERSITY OF
OXFORD

T-cell



OmniCAR can do what conventional CAR-T cannot

Conventional CAR-T



- Soldier with only one map
- Single weapon
- Only trained to hit one target
- Incapable of redirection
- No communication or control in the field



Armed with **any** weapon
Including **several** at once



OmniCAR



Can be given **any** map;
Multiple deployments



Full **communication**
and **control** at all times,
even mid-mission



Can direct against
any target, including
simultaneous targets

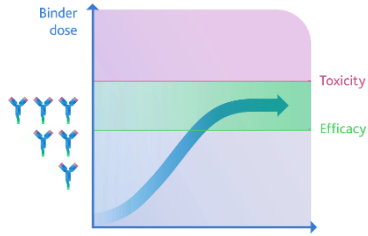


Send **images** back to
base in real time

OmniCAR: Control Features

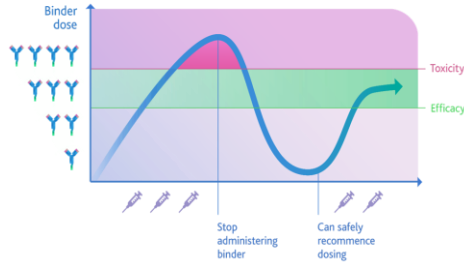
Modular and covalent architecture of OmniCAR enables true **post-infusion control** of CAR functionality

Dose Titration



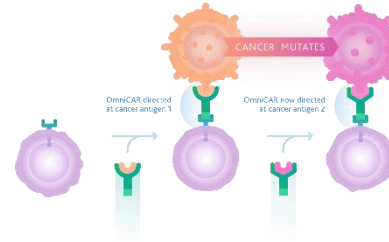
Control activity to **safe and efficacious** levels

On/off switch



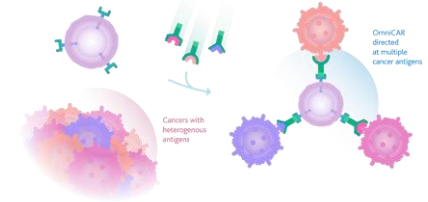
Turn therapy on/off/on without killing or re-administering cells = **safety & persistence**

Target Re-direction



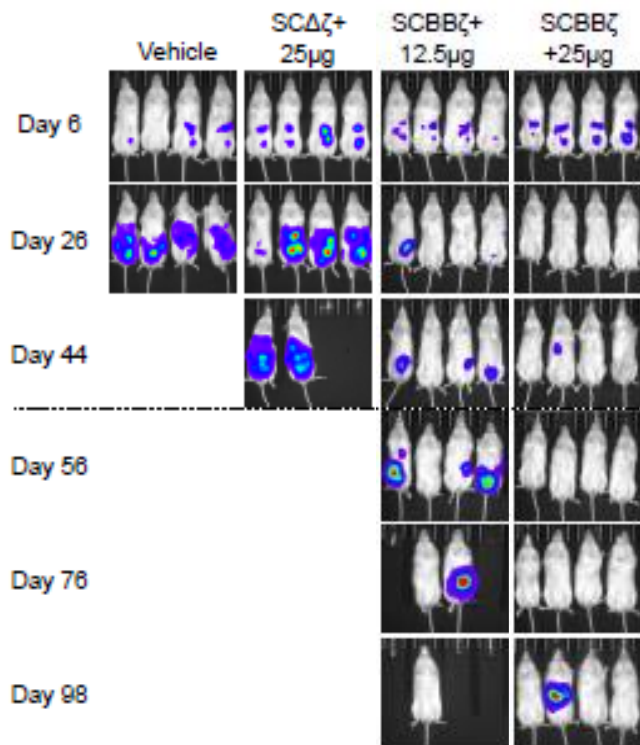
Re-direct cells from one cancer target to another in vivo

Multi-Antigen Targeting

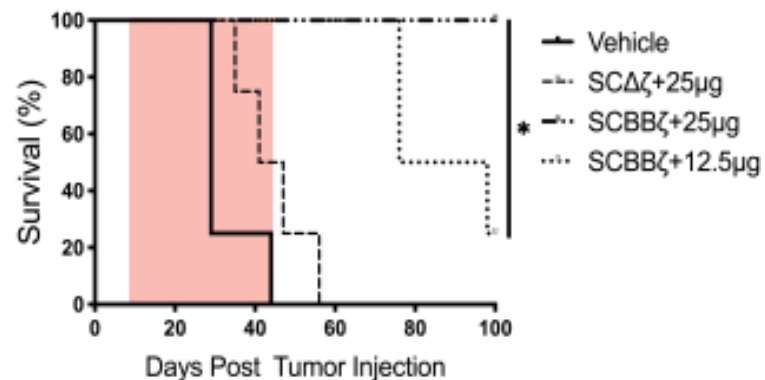


Target **multiple cancer antigens simultaneously** for thorough cancer killing

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



OmniCAR cells can be Re-Armed



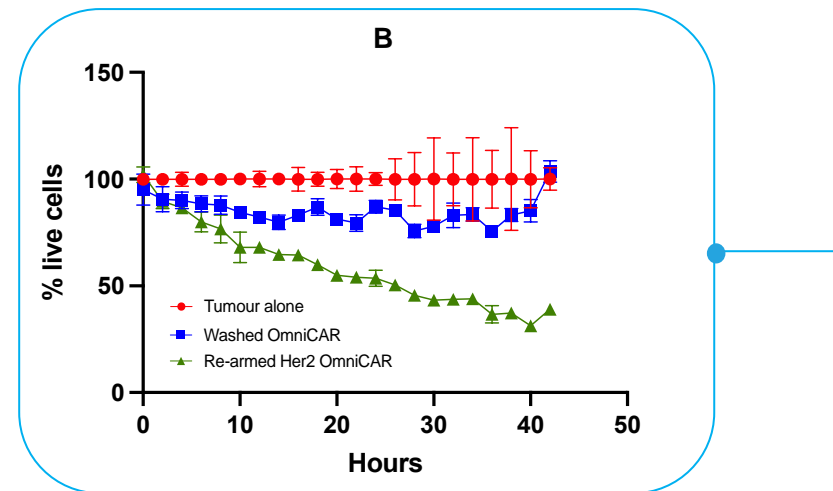
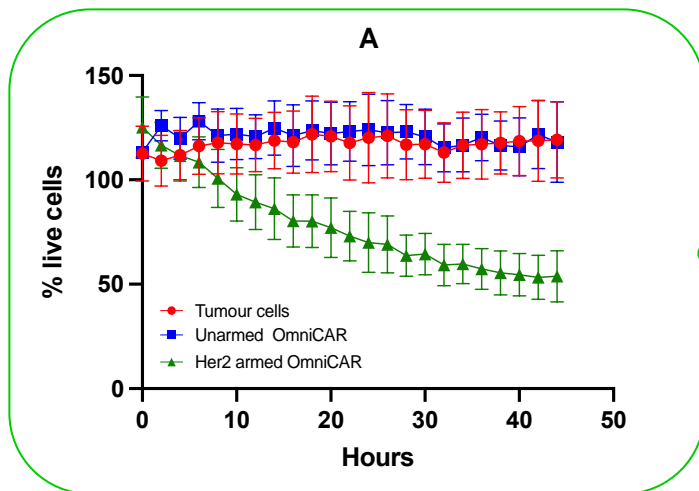
Wash; Rest

Re-armed

Cytotoxicity assay

Day 12

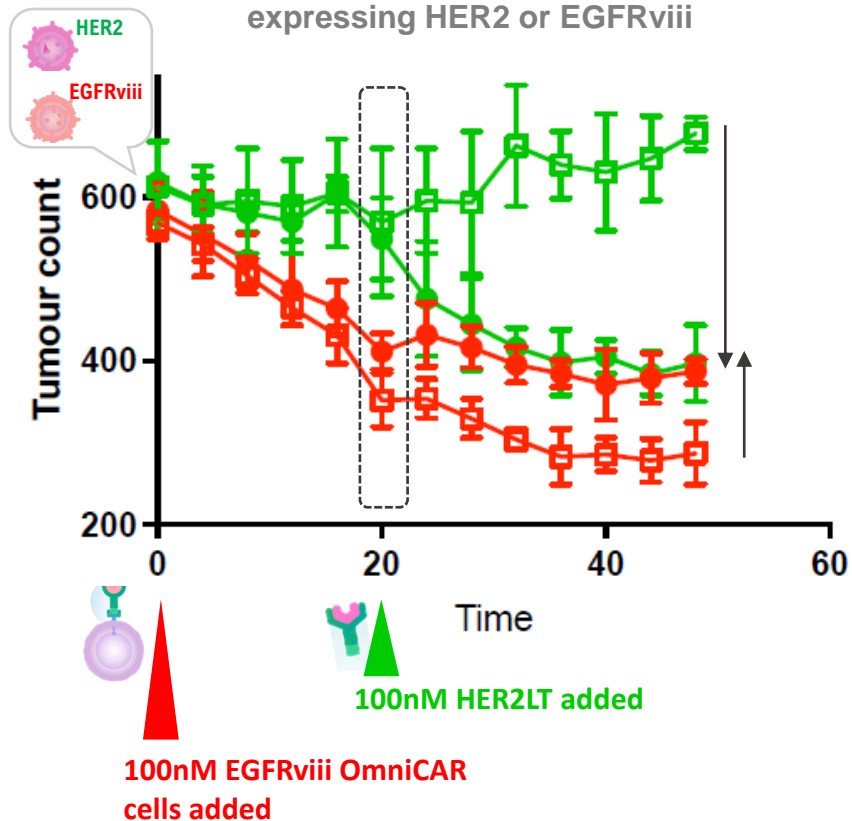
Days 13–14



- 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

OmniCAR cell can be Redirected

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



1

OmniCAR T cells **pre-armed** with EGFRviii binder

→ Rapid cytotoxicity to EGFRviii+ cells

2

Add Her2 binder

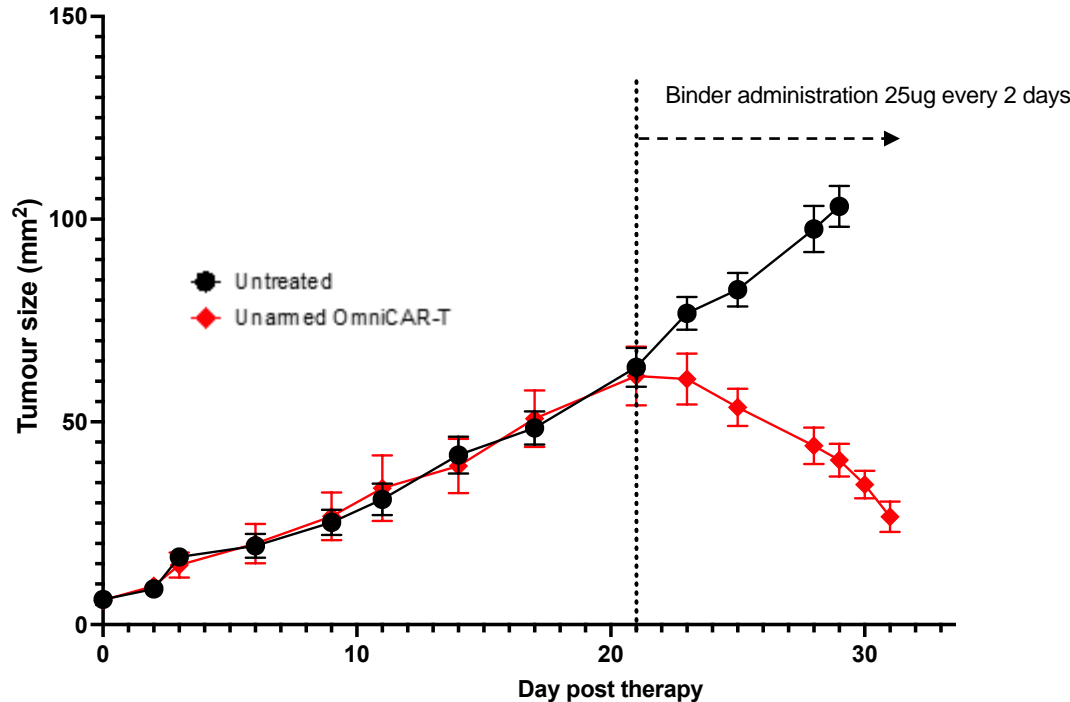
→ Rapid switching & cytotoxicity to Her2+ cells

No new cells required

OmniCAR cells viable & armable for weeks

Mice with OC25 tumours

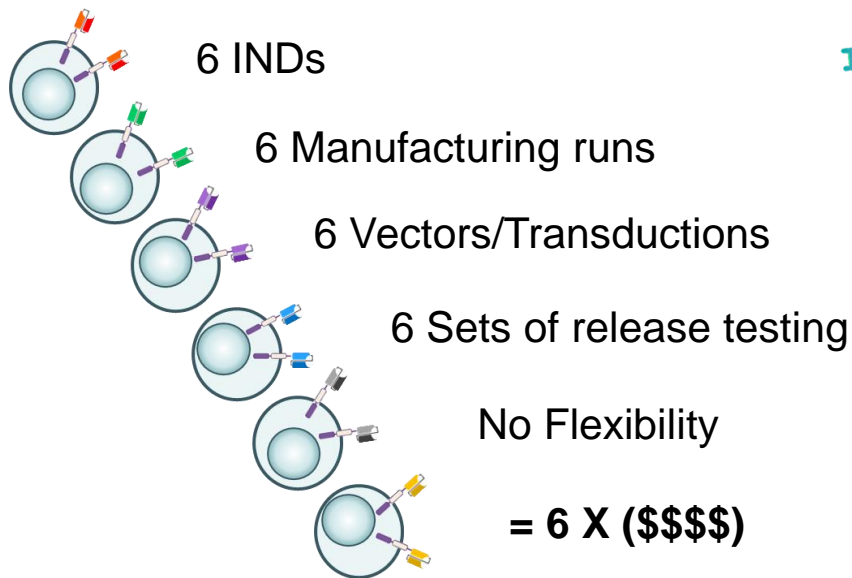
Binder administered from day 21



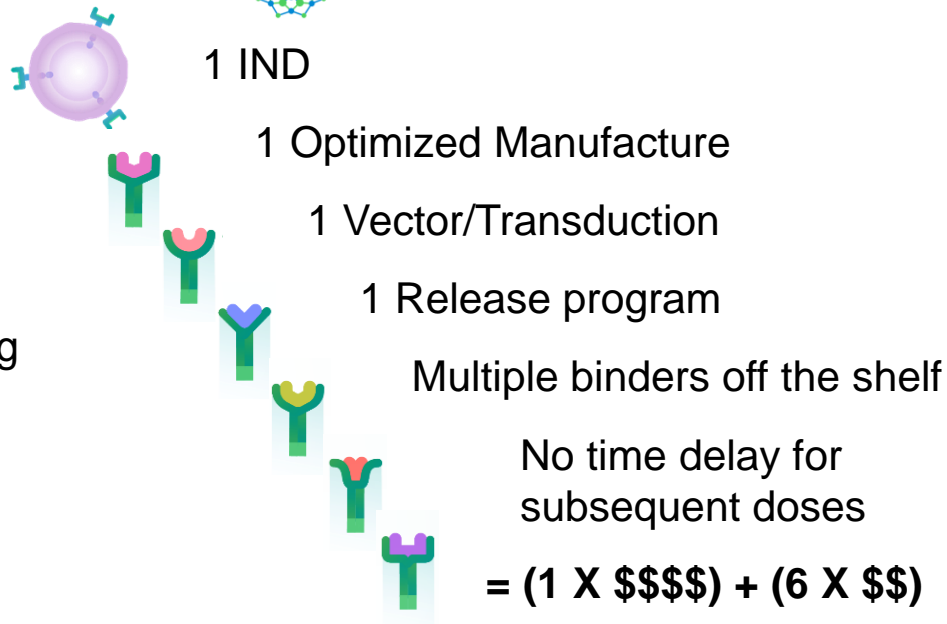
- Unarmed & armed OmniCAR-T cells are viable for weeks
- Can be armed at will
- Results in immediate cytotoxicity

Regulatory, manufacturing & COGS advantages

Conventional CAR-T



OmniCAR





OmniCAR

Next Gen CAR-T Programs

OmniCAR internal program summary

Targets	Indications	OmniCAR features	Comments
CD33 + CLL-1	Acute Myeloid Leukemia (AML)	<ul style="list-style-type: none"> • Titration for improved safety • Co-arming against CD33 & CLL-1 • Sequential targeting 	<ul style="list-style-type: none"> • Validated targets; expressed on 90%+ of AML blasts & LSCs • 1 of 5 programs worldwide; the only next-gen program
HER2	Ovarian; breast & gastric cancers	<ul style="list-style-type: none"> • Titration for improved safety • Persistent binder dosing for improved efficacy • TME and checkpoint enhancements 	<ul style="list-style-type: none"> • Most mature next-gen HER2 CAR-T program • Builds on Penn pre-clinical PoC
HER2 + EGFRviii	Glioblastoma multiforme (GBM)	<ul style="list-style-type: none"> • Titration for improved safety • Co-arming against HER2 & EGFRviii • Persistent binder dosing for improved efficacy 	<ul style="list-style-type: none"> • 1 of 3 multiple antigen programs in the world • Single antigen targeting is inadequate in GBM

OmniCAR progressing towards clinic

- Steady progress across all programs
- OmniCAR AML likely the first program in clinical trials
- Q-Gen Cell Therapeutics appointed as cell manufacturer
 - Clinical grade cells
 - Autologous T cells expressing SpyCatcher
 - Incorporating CellPryme-M for superior phenotype
- Prescient to articulate regulatory path and clinical development details shortly



MD Anderson Cancer Center



THE UNIVERSITY OF TEXAS
~~MD Anderson~~
Cancer Center
Making Cancer History®



MD Anderson's ECLIPSE platform has yielded novel TCR-like binders



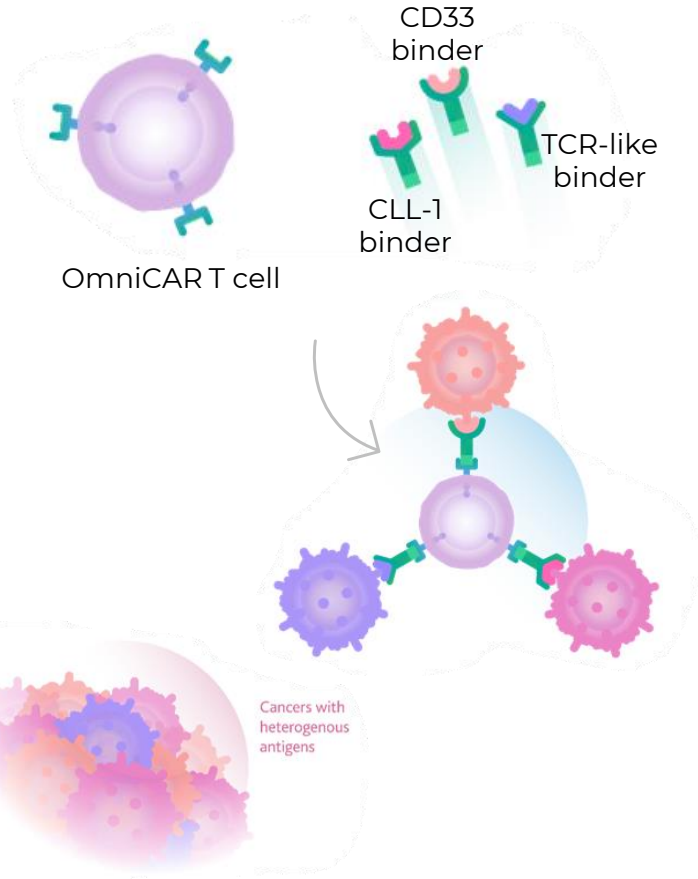
MD Anderson's novel TCR-like binder library

- Broad library of samples from leukemia patients (ECLIPSE platform)
- Uncovered unique TCR-like binders
- Targets blood cancer cells differently to CAR-T

Strategic Collaboration

- Strategic collaboration to add novel TCR binder to OmniCAR
- Create best-in-class adaptable CAR-Ts for blood cancers
- Shared costs and outcomes

Adding TCR-like binder to OmniCAR for AML



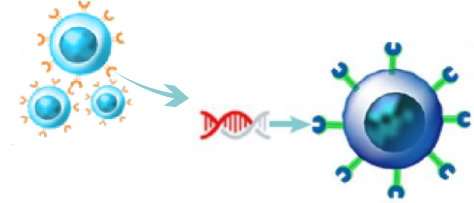
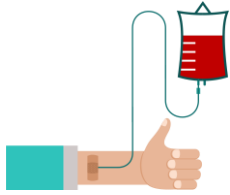
- Using “plug & play” features of OmniCAR to combine novel TCR-like binder with CD33 & CLL-1 for AML
- Create an unprecedented level of multivalency and control.
- **For the first time**, we will be able to test:
 - Multivalent T cells that **have both CAR and TCR** targeting ligands using the **single internal cytotoxic machinery**
 - Has the potential to **make OmniCAR-T cells >1000x more sensitive** to rare or low abundance antigens
 - The impact of periodic resting on TCR-directed killing



CellPryme

**CELL THERAPY
ENHANCEMENTS**

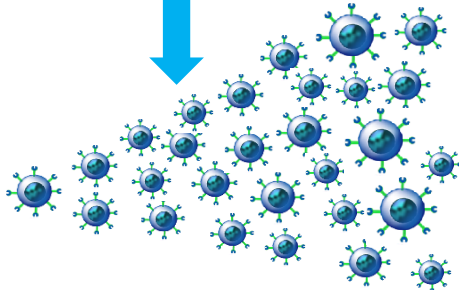
The CAR-T process



1 Blood is collected from the patient

2 T-Cells are isolated

3 T-Cells are genetically altered to have cancer-recognising receptors (CARs)



4 Millions of CAR-T cells are grown

5 CAR-T cells are administered to the patient



CellPryme-M

MANUFACTURING ENHANCEMENT

- Produces longer lasting, more “youthful” CAR-T cells
- Doubles helper T cells
- Doubles tumour control
- More chemokine receptors for locating tumours



CellPryme-A

ADJUVANT THERAPY

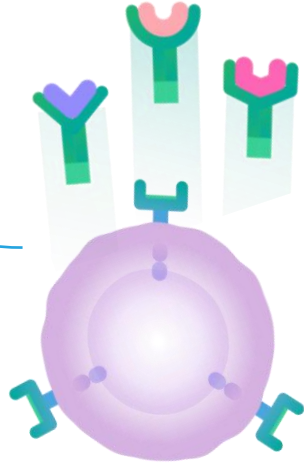
- Overcomes hostile TME
- Reduces Tregs
- Increases expansion of CAR-T cells *in vivo*
- Doubles penetration of CAR-T cells into tumours

CellPryme Complements OmniCAR



OmniCAR

- Multi-targeting
- Redirection
- Control & safety
- Any target; any cell



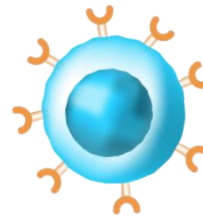
Next generation
Cell therapies



CellPryme-M

Process that produces
a better cell type

- Persistence
- Trafficking



Current generation cell
therapies



CellPryme-A



Adjuvant therapy

- Reduces Tregs
- Primes TME for cell therapy
- Boosts CAR-T cell expansion *in vivo*



CellPryme-M

**Cell manufacturing
enhancement**

CellPryme-M Executive Summary

PROCESS TO ENHANCE CELL THERAPIES



- Incorporate into standard manufacturing
- Current gen and next gen
- Complementary to OmniCAR

PRODUCES SUPERIOR CELLS



- More “youthful” T cells
- More effective tumour killing
- Longer lasting

READY FOR CLINICAL TESTING



CellPryme-M IP FULLY OWNED BY PTX



Developed by PTX
in collaboration with Peter Mac

CellPryme-M produces CAR-T cell types with ideal characteristics and attributes



Persistence

For longevity of effects and continued tumour control



Immune memory

Central memory T cells typically persist 10-20 years and as long as 75 years



Trafficking

CAR-T cells able to find their way to the tumour



Tumour penetrance

Cells that can penetrate solid tumours



Genomic stability

Cells with enhanced self-renewal due to greater genomic stability

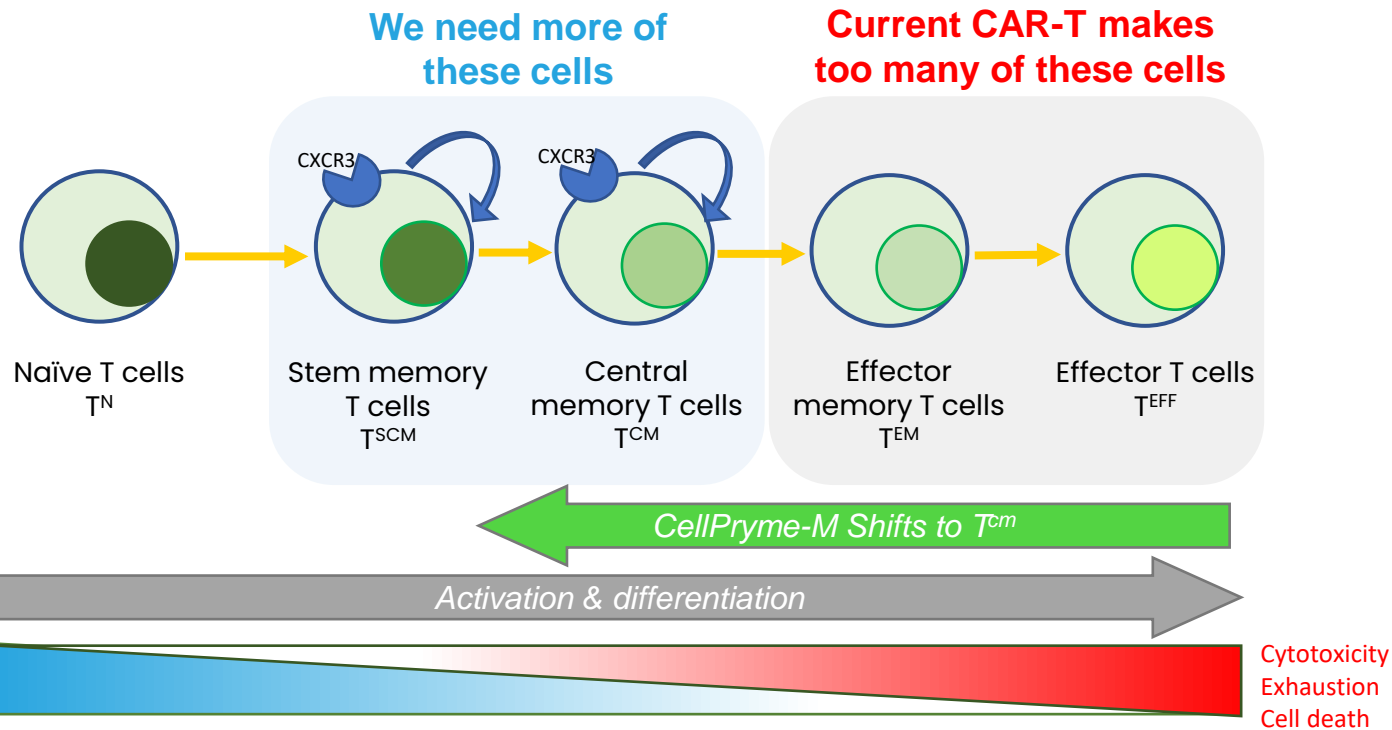


Anti-viral

Cells with potent anti-viral characteristics

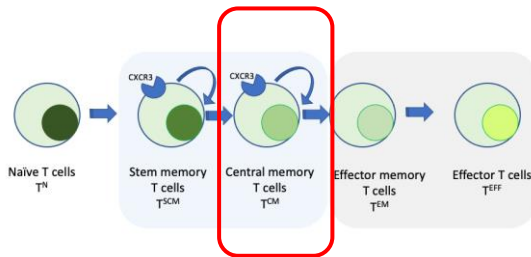
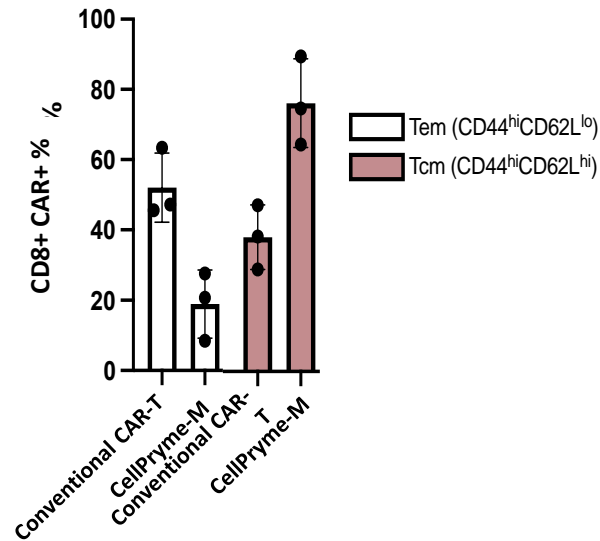
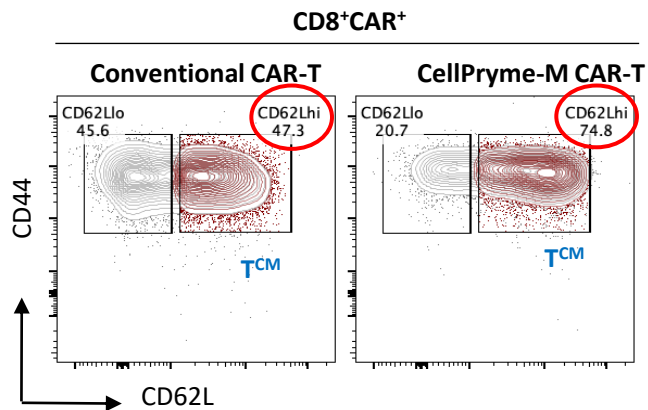
More memory cells required for clinical efficacy

- Clinical efficacy of CAR-T therapy remains dependent on the T cell phenotype
- It is possible to control this during the manufacturing step



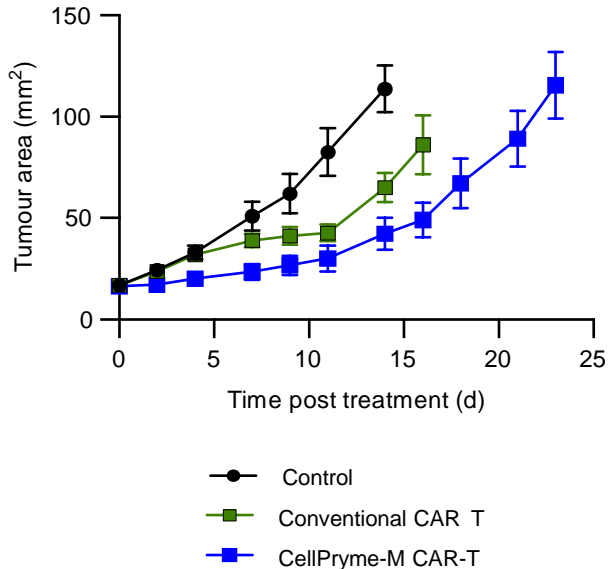
Greater Persistence: 50% more central memory cells than conventional CAR-T

CellPryme-M increases central memory T cells 1.5-fold within 24hrs

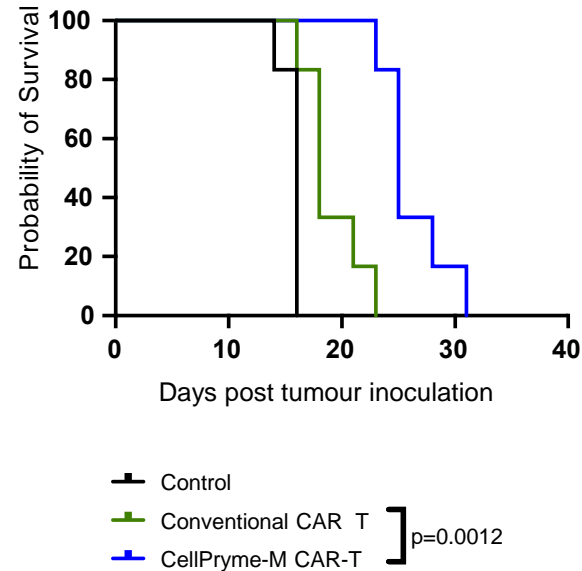


CellPryme-M doubles tumour control and survival

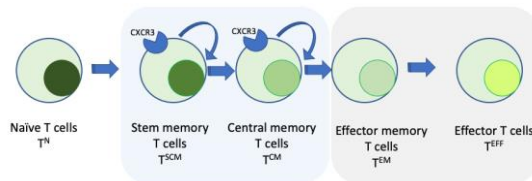
CellPryme-M nearly doubles CAR-T tumour control



CellPryme-M doubles survival



Greater Persistence/Less Exhaustion

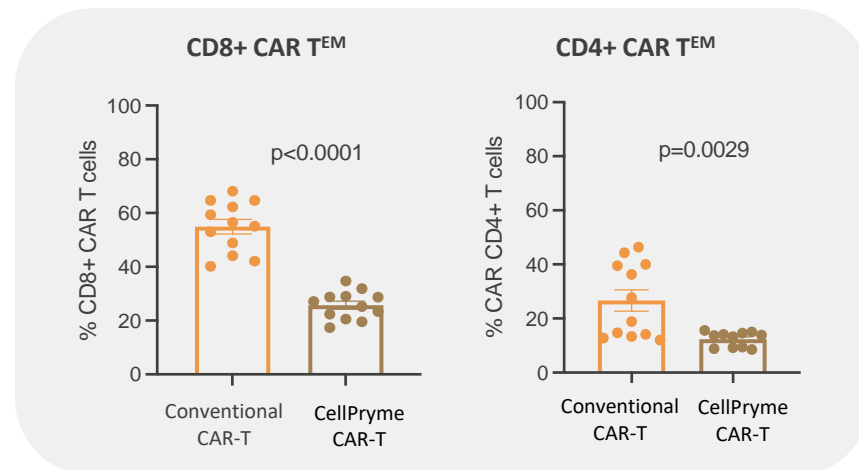
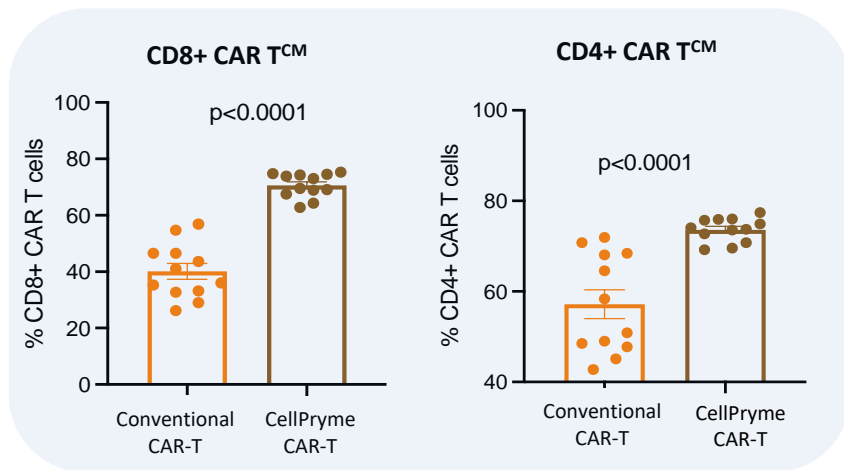


Sustained increase in T^{CM}

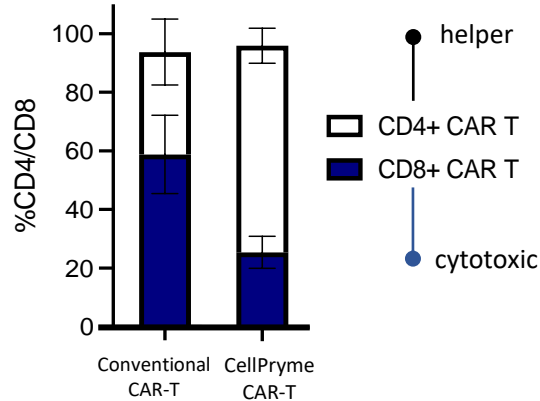
Sustained decrease in T^{EM}

for both cytotoxic CD8+ and helper CD4+

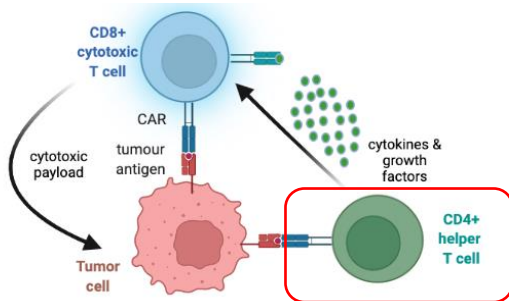
for both cytotoxic CD8+ and helper CD4+



Synergy: CellPrime-M doubles proportion of helper T cells



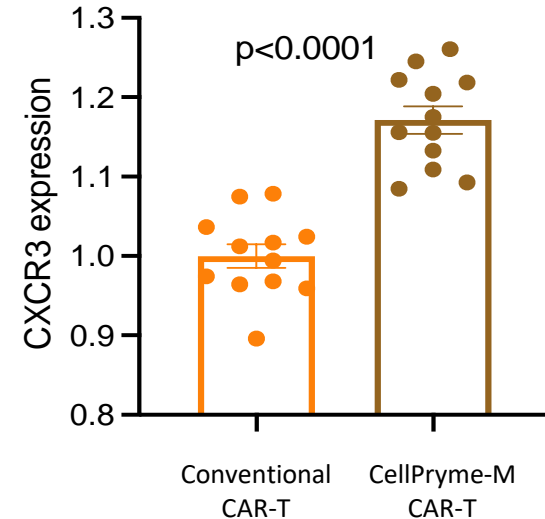
- Shift towards dominant helper CD4+ CAR T cells
- Helper T cells are known to prevent the exhaustion of cytotoxic CD8+ T cells
 - Some can also have tumour killing ability
- Helper & cytotoxic T cells work in synergy to increase CAR-T persistence

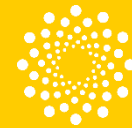


Trafficking: greater chemokine receptor expression

- Effector T cells can downregulate chemokine receptors (CXCR3), limiting the ability of conventional CAR-T cells to locate tumours
- CellPryme-M significantly increases CXCR3 expression on CAR-T cells
- Better trafficking to tumour site
- Better tumour penetrance

Chemokine receptor expression on CD8+ cytotoxic CAR-T cells





CellPryme-A

**Adjuvant for enhancing
cell therapies**

CellPryme-A Summary

ADJUVANT PLATFORM TO ENHANCE CELL THERAPIES

- Current gen and next gen
- Complementary to CellPryme-M & OmniCAR

BREAKS DOWN HOSTILE TME

- Two-thirds less intratumoral Tregs
- Increases CAR-T cell penetration into tumours

IMPROVES TUMOUR KILLING AND SURVIVAL

BOOSTS CAR-T EXPANSION *IN VIVO*

READY FOR CLINICAL TESTING

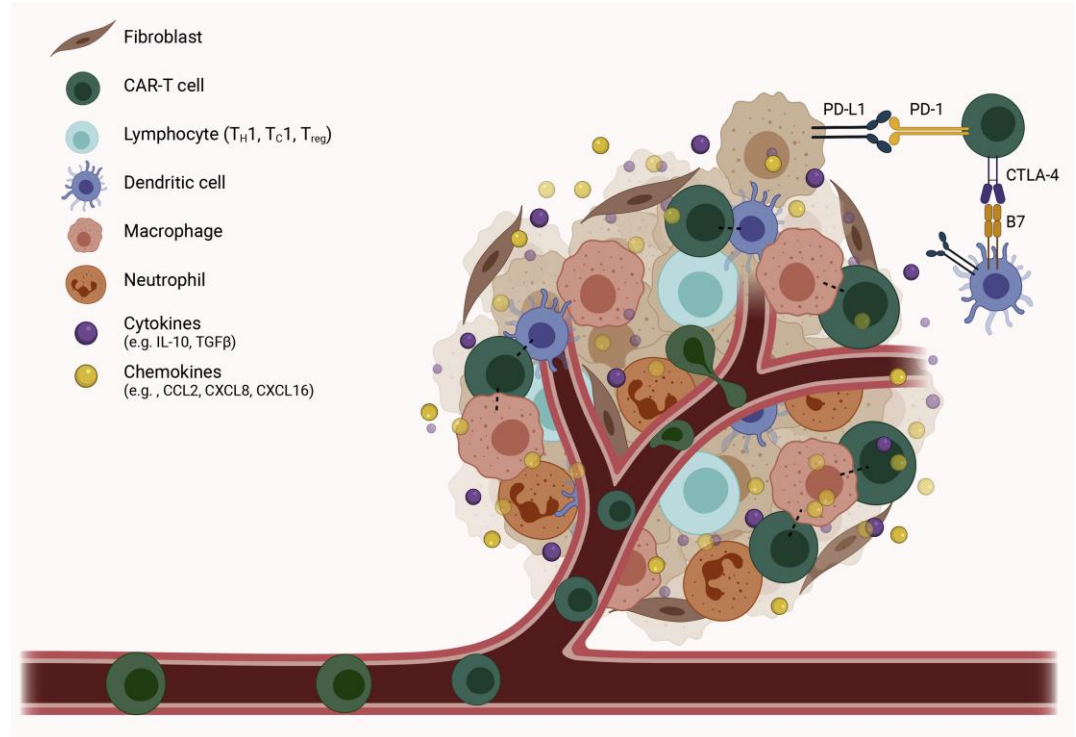
- GMP material ready

DEVELOPMENT OPPORTUNITIES

- PTX & 3rd party programs
- Use with any existing cell therapy for solid tumours

CellPryme-A addresses the hostile Tumour Microenvironment (TME)

- TME is the **complex ecosystem** surrounding solid tumours
- Protects and nurtures the cancer
- Acts as a **protective “force field”** that blunts the effectiveness of cancer therapies



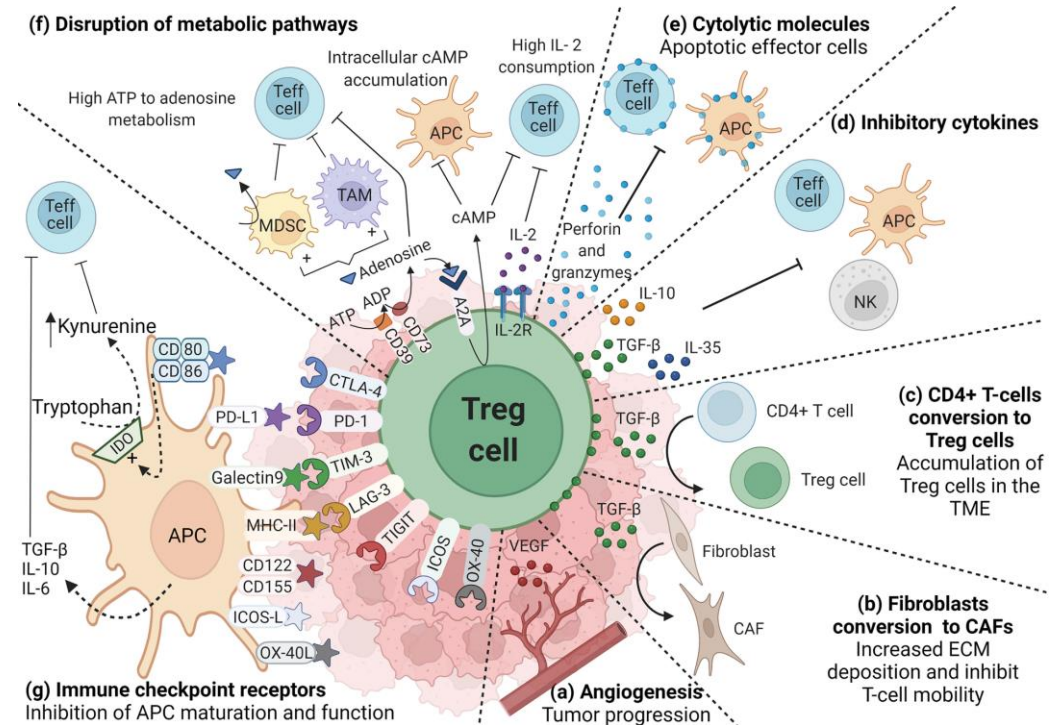
Treg cells are central players in the TME

Tregs are immunosuppressive cells in the TME

(i.e. suppresses the immune response)

- Causes T cell **exhaustion**
- Causes T cell **death**
- Produces cytokines that cause the tumour to go “**cold**”
- **Impairs NK** cell function

Reducing Tregs is key to successful CAR-T therapy



Summary of CellPryme-A effects



Boosts tumour killing by conventional CAR-T cells



Improved survival



Reduces problematic **Treg cells** by 66%



Dramatically increases

CAR-T cell expansion within

- 2x ↑ CAR-T cell expansion
 - 9x ↑ Cytotoxic T cells
 - 6x ↑ Helper T cells
- with CellPryme-M



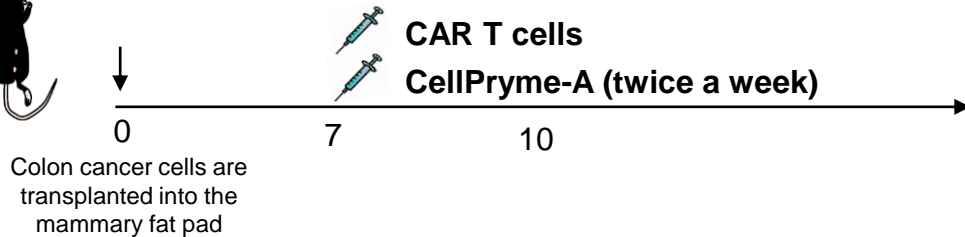
Increases ability of T cells to **penetrate solid tumours**

- 4x ↑ Cytotoxic T cells
- 3x ↑ Helper T cells



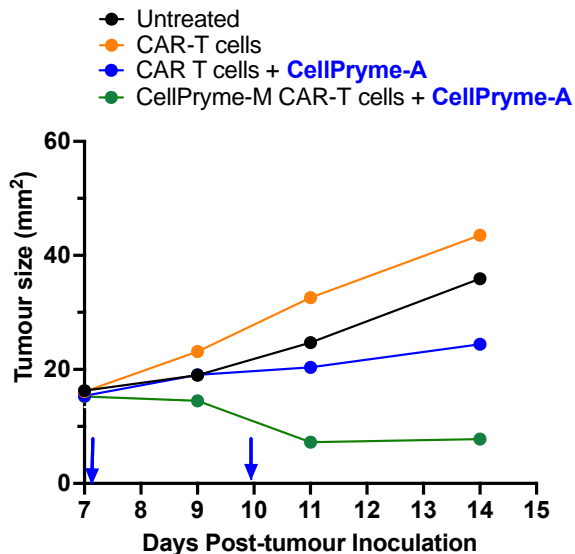
Synergises with CellPryme-M for **even greater benefits**

CellPryme-A significantly boosts CAR-T efficacy

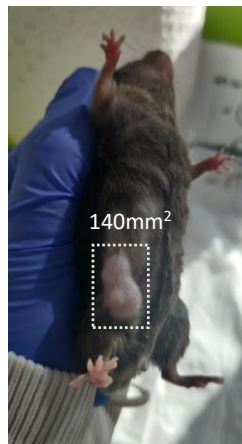


Experimental end point

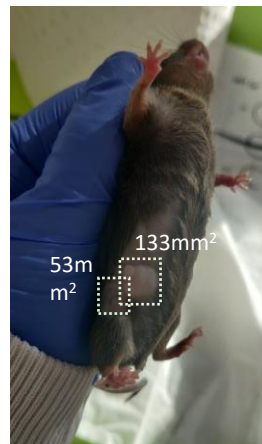
Tumour size > 150 mm² or ≤ 28 days unless other humane endpoints are reached



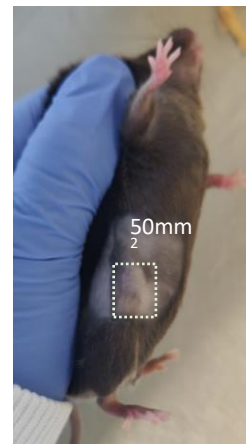
Untreated



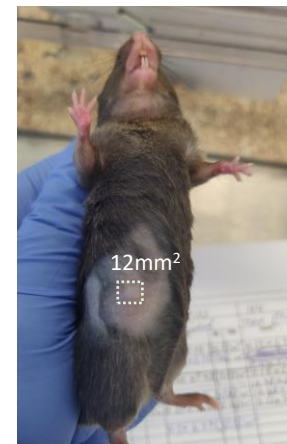
CAR-T cells alone



CellPryme-A + CAR-T cells



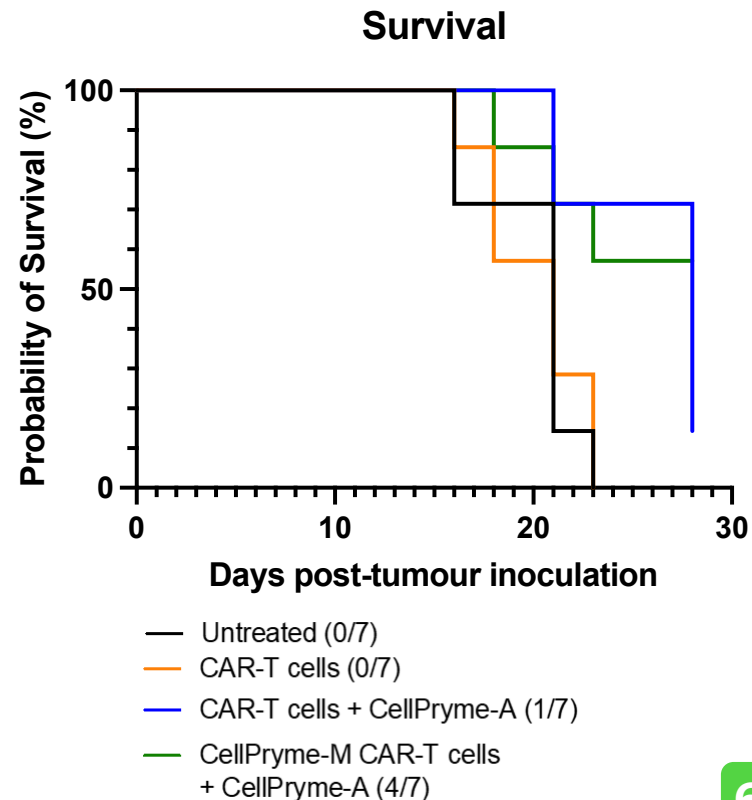
CellPryme-A + CellPryme-M CAR-T cells



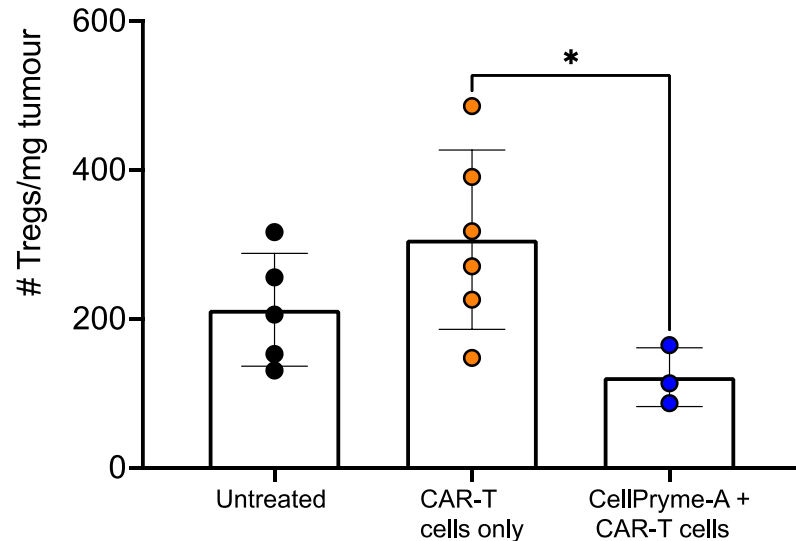
CellPryme-A improves survival

- Highly aggressive and resistant cancer model
- CAR-T cells did not improve survival in this model
- CellPryme-A improved the survival of animals given CAR-T cells by over 20% (5 days)
- The combination of CellPryme-A and CellPryme-M treated CAR-T cells **extended survival beyond the study period** in half of the animals under experimentation

Note: Animal ethics approval up to 150mm² or up to 28 days on study unless other humane endpoints are reached



CellPryme-A significantly decreases problematic Tregs in tumours

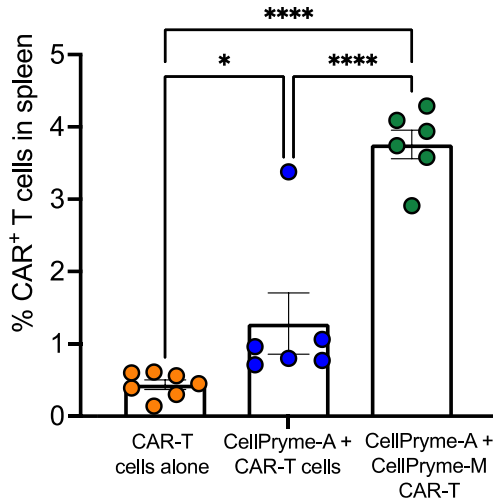


CellPryme-A significantly reduced
intra-tumoural Tregs by two-thirds

Conventional Her2 CAR-T cell therapy followed by CellPryme-A adjuvant therapy

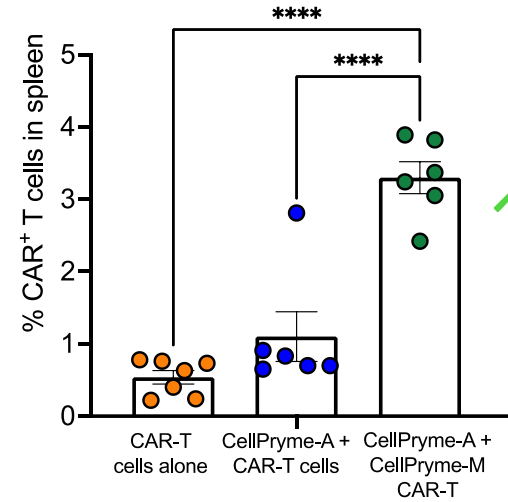
* $p < 0.05$; Kruskal-Wallis one-way ANOVA

CellPryme-A synergises with CellPryme-M to dramatically expand CAR-T cells *in vivo*



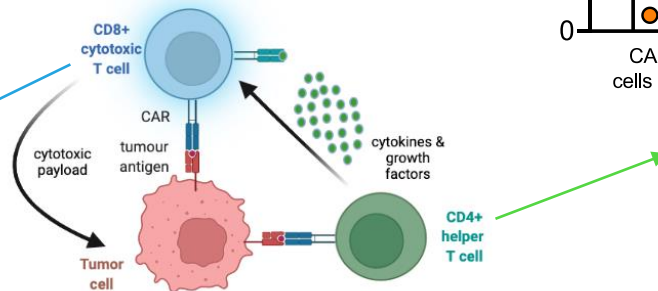
↑9x

CD8+ Cytotoxic T cells

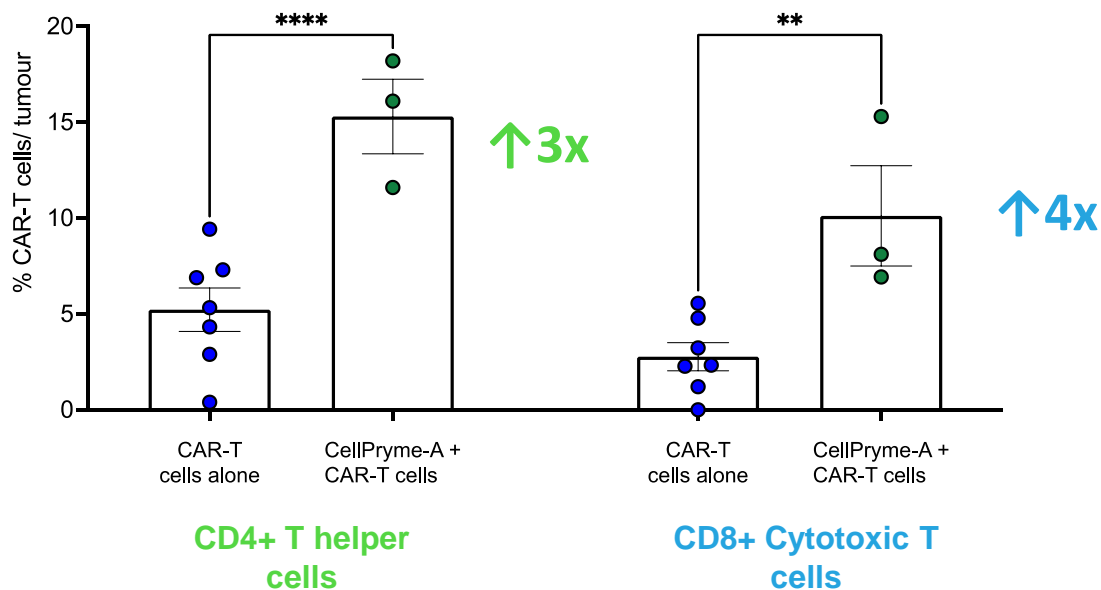


↑6x

CD4+ T helper cells



CellPryme-A significantly increases CAR-T cell penetration into tumours



p<0.01, **p<0.0001, Mann-Whitney test

Tumours collected from parallel cohort of animals at Day 21

CellPryme-A ready for the clinic



CLINIC-READY THERAPY

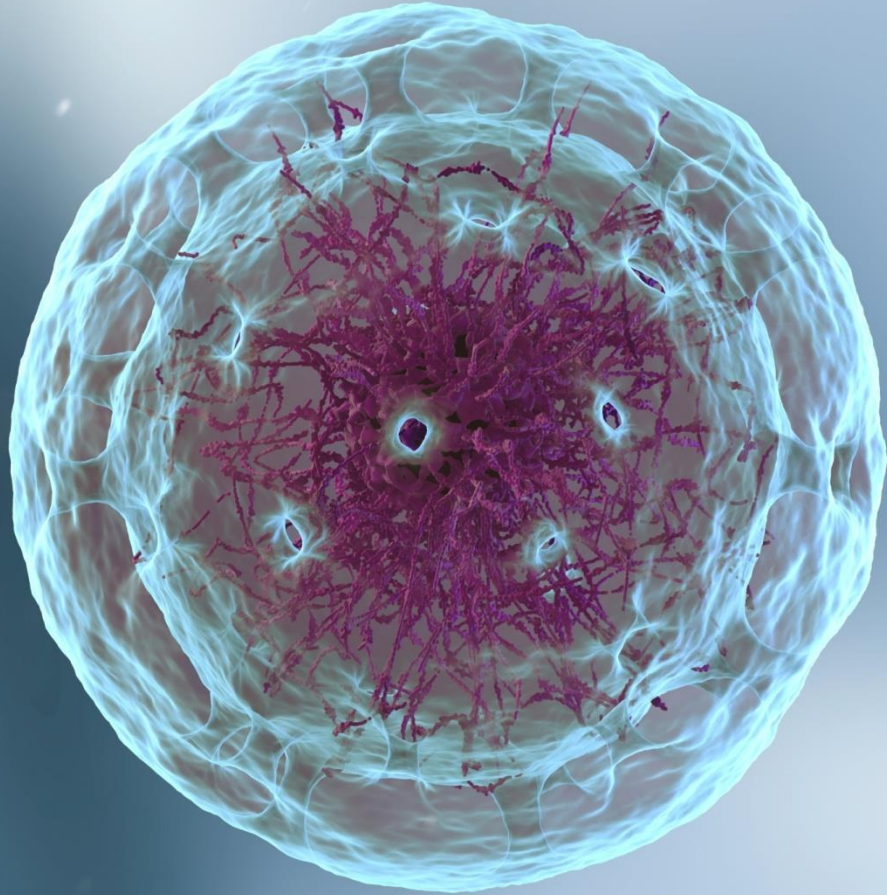
- Ready for clinical testing as adjuvant/neoadjuvant therapy
- Straightforward to incorporate adjuvant into other CAR-T programs
- Robust regulatory package
- Clinical grade material available

CAR-T cells



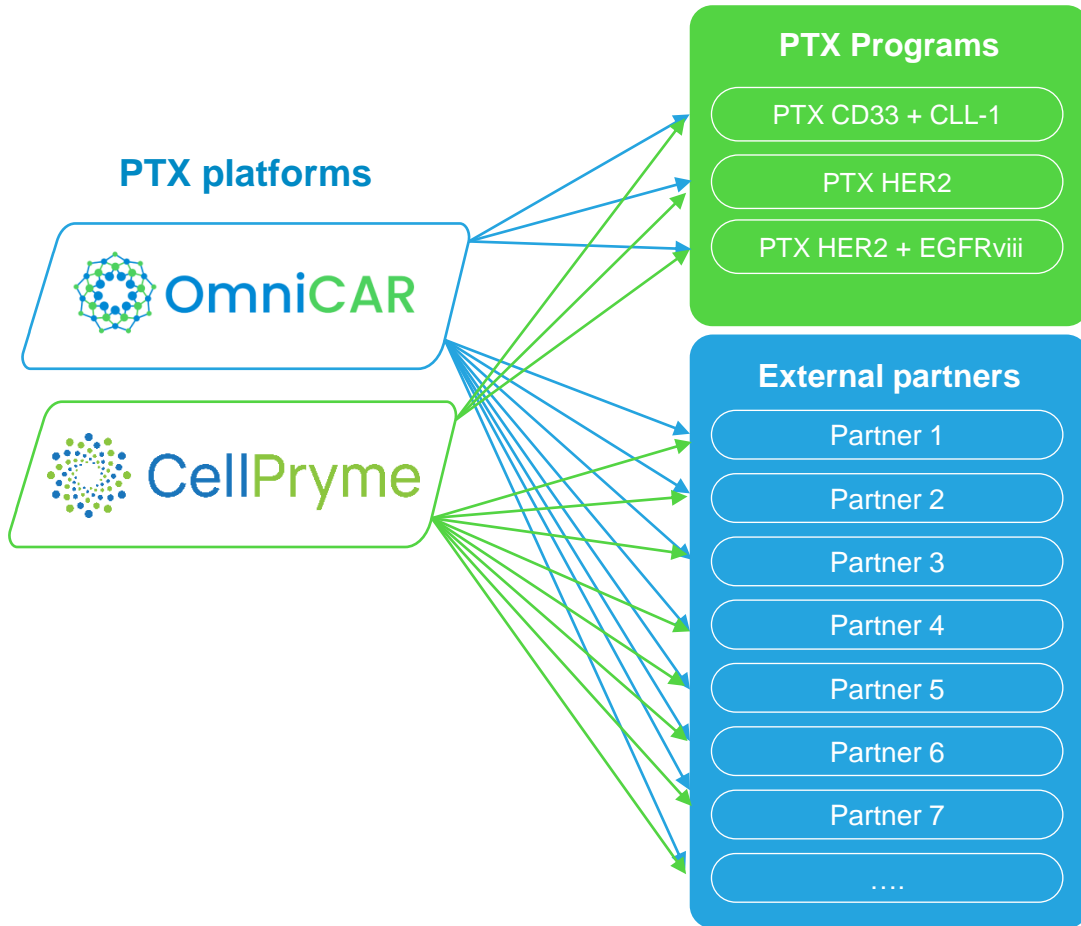
 CellPryme-A





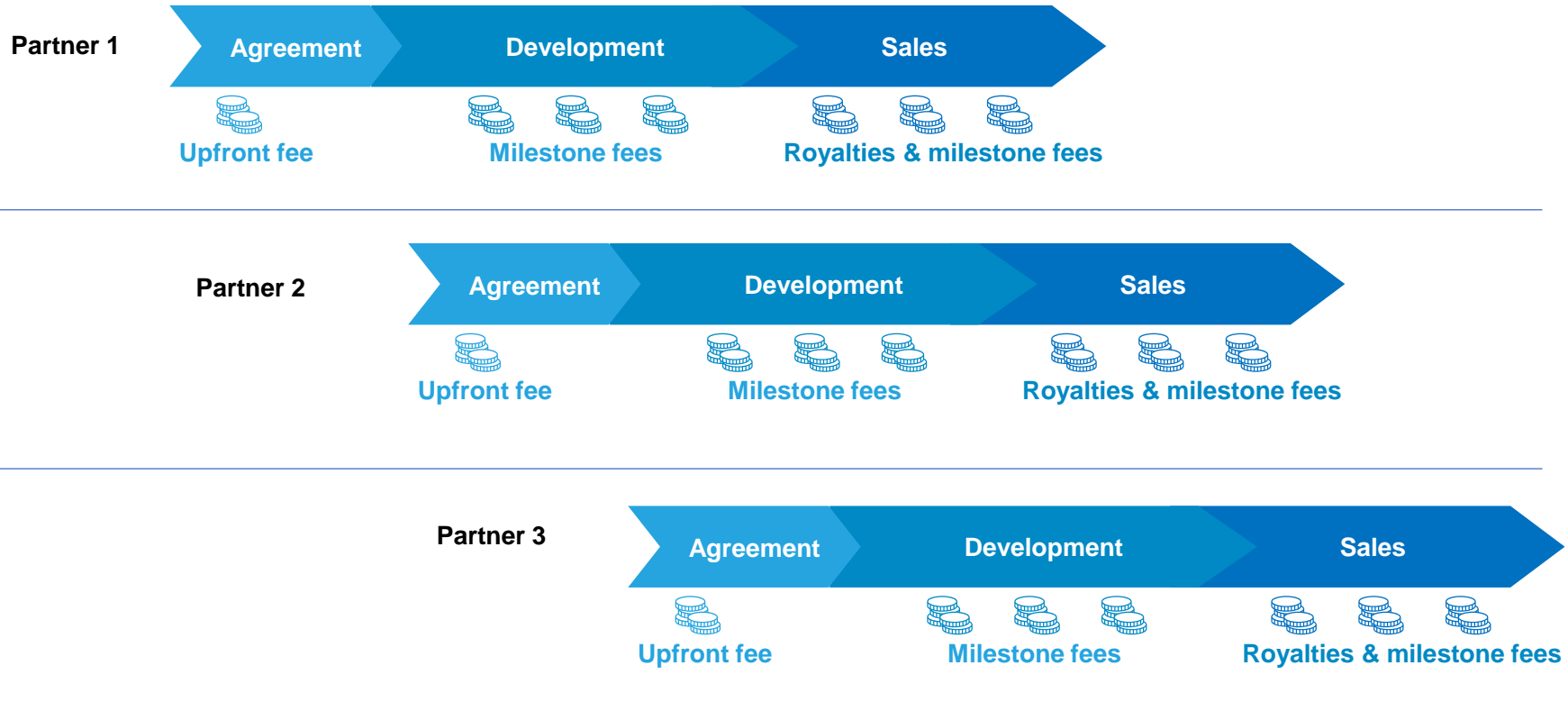
BUSINESS MODEL & SCOPE

Prescient's CAR-T platform business model



- Huge market
- “Shovels to CAR-T goldrush”
- Diversified risk
- Highly scalable
- Earlier revenue potential

Commercial models - Partners



...Partner x

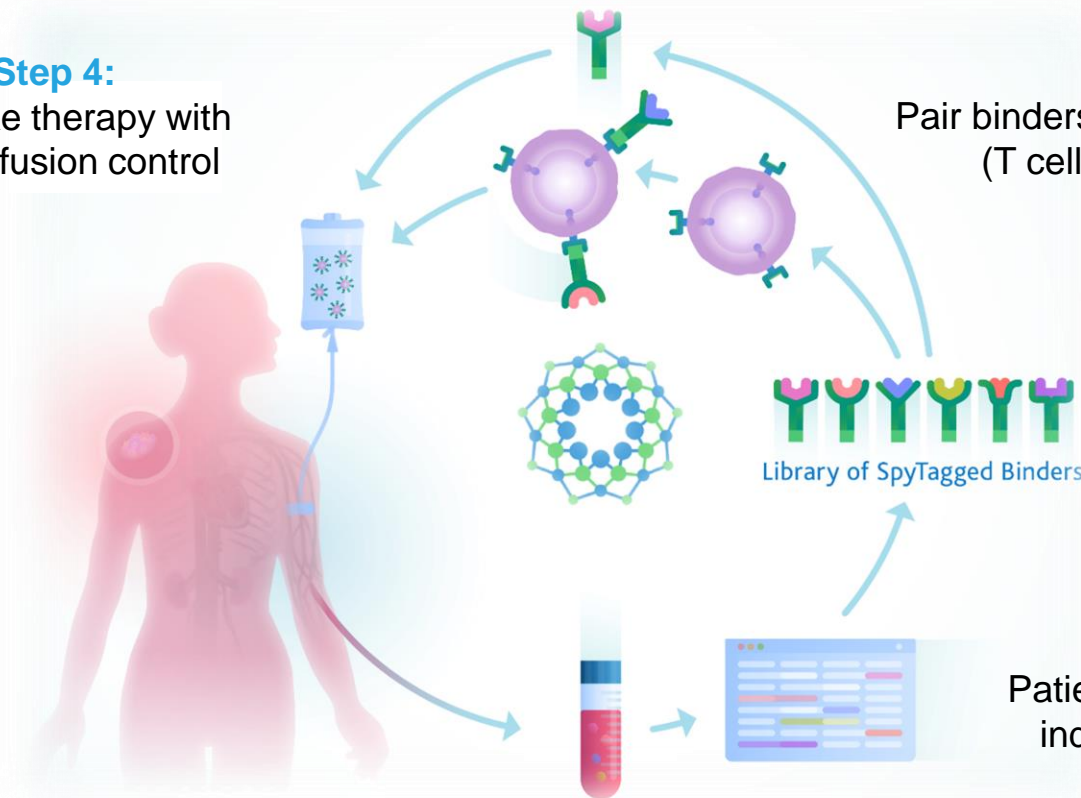
The End Game: Personalized “Plug & Play” Cell Therapy Ecosystem

Step 4:
Bespoke therapy with
post-infusion control

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

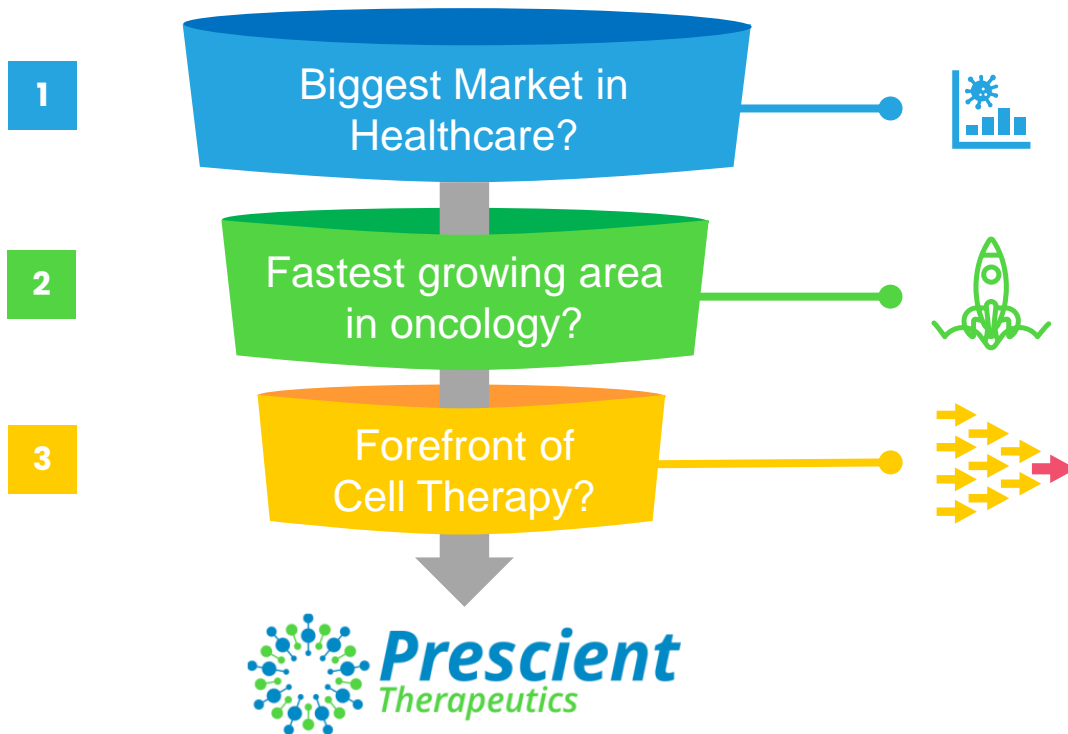
Step 2:
Match patient's antigens
to corresponding binders

Step 1:
Patient sample to determine
individual antigen profile



Summary

Top-down analysis is sensible for investors



Oncology*

- 2021: US\$ 280bn
- 2029: US\$ 536bn (8.2% CAGR)

Cell Therapies (CAR-T)

- >US\$37bn by 2028[^]

Prescient Therapeutics

- Next gen platforms
- Scalable
- Controllable
- Any target; any cell
- “Shovels to goldrush” position
- Top pedigree

Investment Thesis Summary

4 blue chip oncology assets



2 next gen platforms



PTX-100 & PTX-200
in clinic



Top pedigree



OmniCAR PTX-100



CellPryme PTX-200

Superior positioning & model



Internal products
+ external partnering



Shovels to goldrush



Highly scalable



Huge & growing market



\$280bn industry



Growing demand



Cell therapy is the future





Thank you!

ASX code: PTX

www.ptxtherapeutics.com