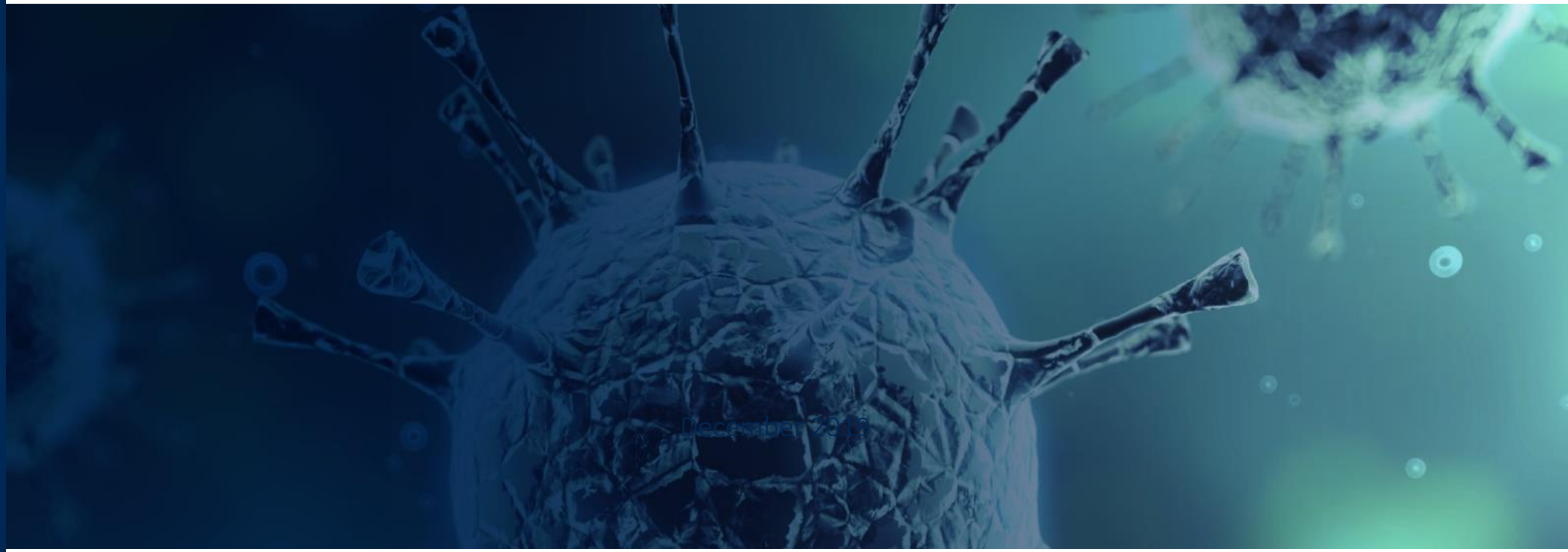
Autolus

Nasdaq: AUTL



Autolus AACR Data Update

June 2020

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Agenda

1. Welcome and Introduction: Dr. Christian Itin, Chairman and CEO

2. T Cell Lymphoma Program:
   - AUTO5 Data Review: Dr. Mathieu Ferrari, Associate Director of Binder Discovery

3. Autolus Solid Tumor Programs:
   - AUTO6NG Data Review: Dr. Muhammad Al-Hajj, SVP, Head of Translational Medicine
   - AUTO7 Data Review: Dr. Marco Della Peruta, Senior Scientist, Immunobiology

4. Summary & Next Steps: Dr. Christian Itin

5. Q&A: Dr. Christian Itin, Dr. Martin Pule (Founder & SVP, CSO), Dr. Vijay Reddy (SVP, CMO), Dr. Muhammad Al-Hajj, Dr. Mathieu Ferrari and Dr. Marco Della Peruta

Welcome and introduction

Dr. Christian Itin

Chairman and CEO

Broad expertise in translational research and binder discovery

Dr. Christian Itin
Chairman & CEO
Previously CEO of Micromet; led development of Blincyto®, the first FDA-approved redirected T cell therapy

Dr. Muhammad Al-Hajj
SVP, Head of Translational Medicine
Experienced drug hunter & cancer biologist. 15+ years Oncology R&D leadership, GlaxoSmithKline, Novartis, AstraZeneca

Dr. Martin Pule
Founder & SVP, CSO
Founder of Autolus; World leading expert in the development of CAR T cell therapies; Clinical senior lecturer & hon. consultant at UCL; Fulbright at Baylor

Dr. Marco Della Peruta
Senior Scientist, Immunobiology
Human Oncological Pathology at University of Verona, School of Medicine, Italy. Previously, lecturer in Haematology at UCL Cancer Institute, UCL, London, UK

Dr. Vijay Peddareddigari
SVP, CMO
Experienced oncologist and drug developer; MD Anderson, GSK and most recently J&J

Dr. Mathieu Ferrari
Associate Director of Binder Discovery
Experimental Medicine, Queen Mary University of London, UK. Previously, post-doctoral research fellow at Centre for Experimental Medicine and Rheumatology in Queen Mary University of London

Broad pipeline of next generation programs

Designed to address limitations of current T cell therapies

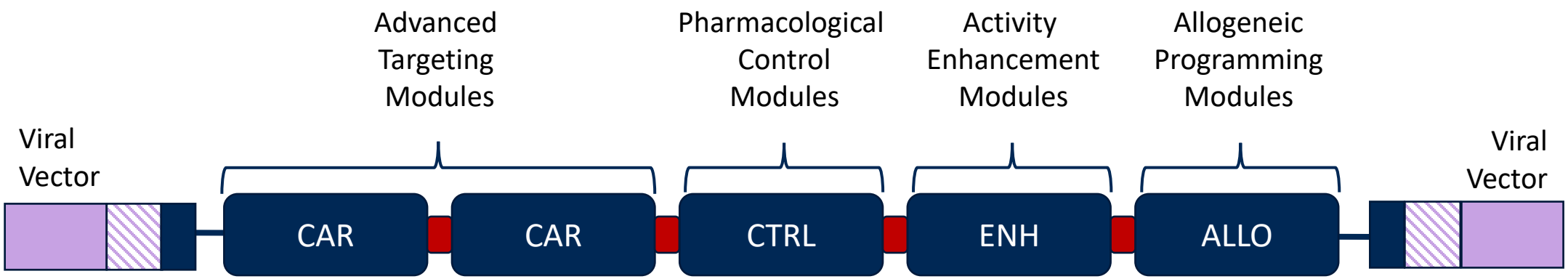
Product	Indication	Target	Pre-clinical	Phase 1
B Cell Malignancies				
AUTO1NG	ALL	CD19 & CD22		H2 2020
AUTO3NG	DLBCL	CD19 & CD22		Life cycle mgmt
T Cell Lymphoma				
AUTO5	TRBC2+ Peripheral TCL	TRBC2		H1 2021
GD2+ Tumors				
AUTO6NG	Neuroblastoma; Melanoma; Osteosarcoma; SCLC	GD2		H1 2021
Prostate Cancer				
AUTO7	Prostate Cancer	PSMA		H1 2021
Multiple Myeloma				
AUTO8	Multiple Myeloma	BCMA & CAR X		H2 2020

NG = Next Generation, SCLC = Small Cell Lung Cancer

Autolus

A broad toolkit building on our core strategy of modular innovation

Advanced T cell programming



Advanced Targeting Modules	Pharmacological Control Modules	Activity Enhancement Modules	Allogeneic Programming Modules
Fast off Rate CARs	**Safety Switches**	**dSHP2**	**GvHD**
Dual Targeting CARs	**Tunable CARs**	**dTGFβ Receptor**	**Immune Rejection**
Avidity Enhancing Spacers		**Chimeric Cytokine Receptors**	
Logic Gates		**Host Immune Cell Recruitment**	
AUTO1 AUTO1NG AUTO3 AUTO8	AUTO4 AUTO5 AUTO6 AUTO6NG AUTO7 AUTO8	AUTO3NG AUTO6NG AUTO7 AUTO8	1st clinical program start in 4Q 2020

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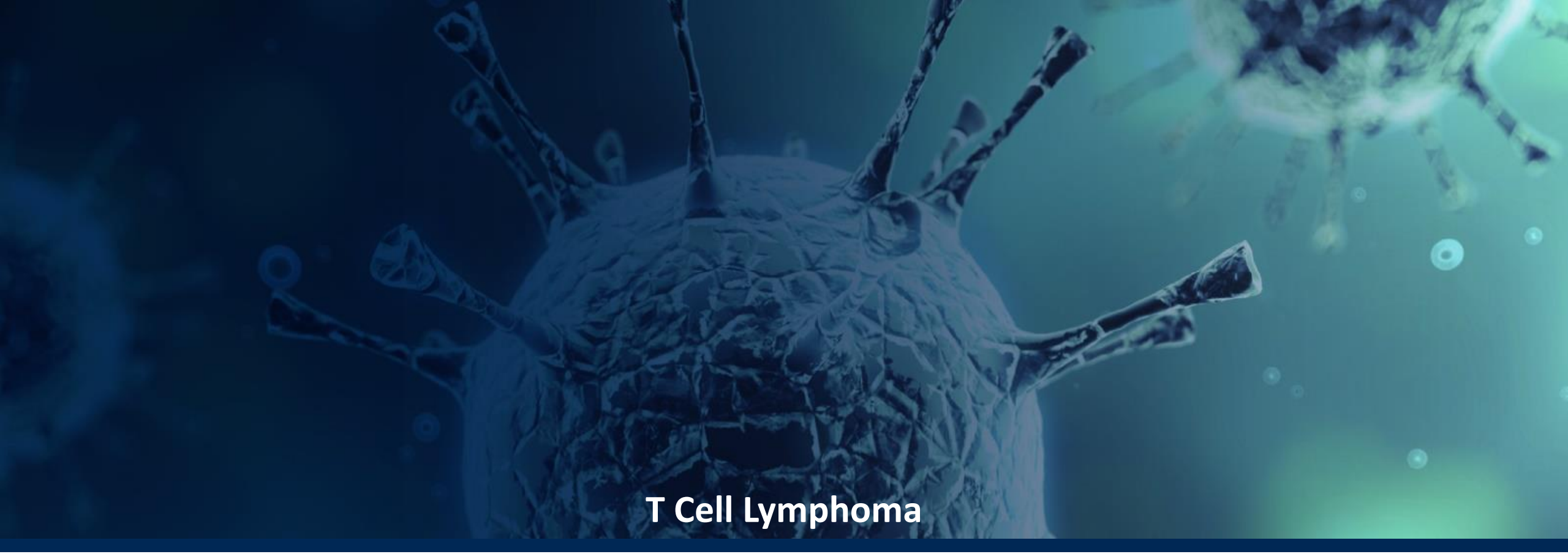


T Cell Lymphoma

AUTO5 – tailored for T Cell Lymphoma

T Cell Lymphoma

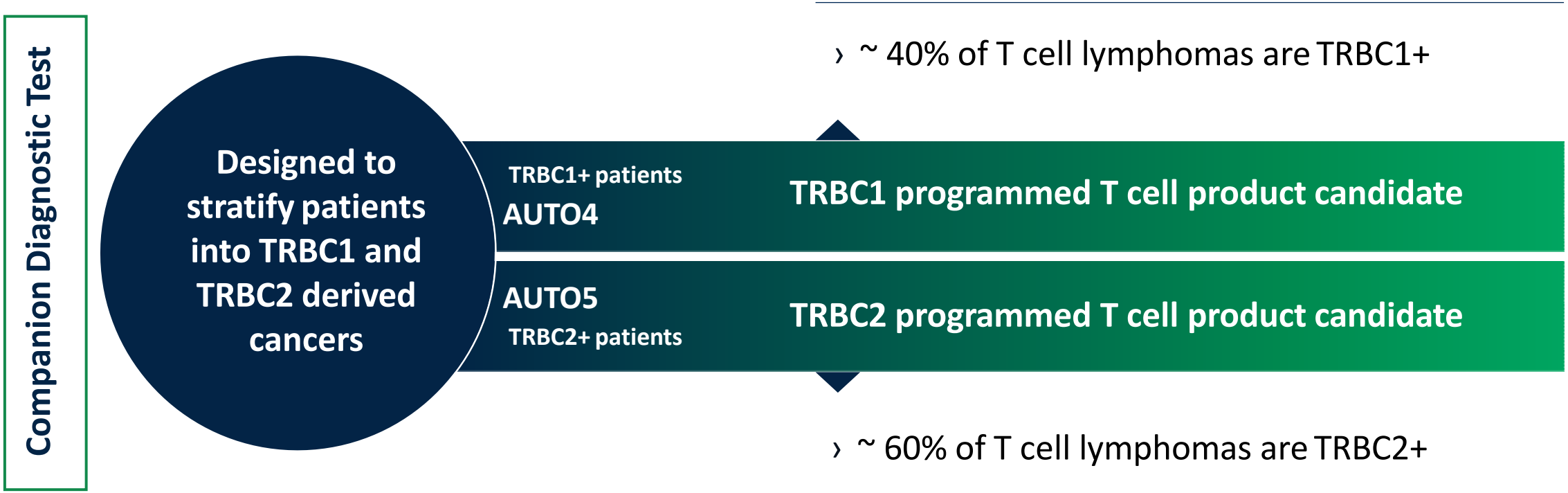
No standard of care after first relapse

There is currently no T cell therapy approved for T cell lymphoma

- T cell lymphoma is an aggressive disease with a very poor prognosis for patients
- Median 5 yrs OS: 32%
- Standard of care is variable and often based on high-dose chemotherapy and stem cell transplants
- A large portion of T cell lymphoma patients are refractory to or relapse following treatment with standard therapies
- T cell lymphomas have not benefited from advances in immunotherapeutic approaches

Addressing T cell lymphomas

Three key elements - AUTO4, AUTO5 and a companion diagnostic test

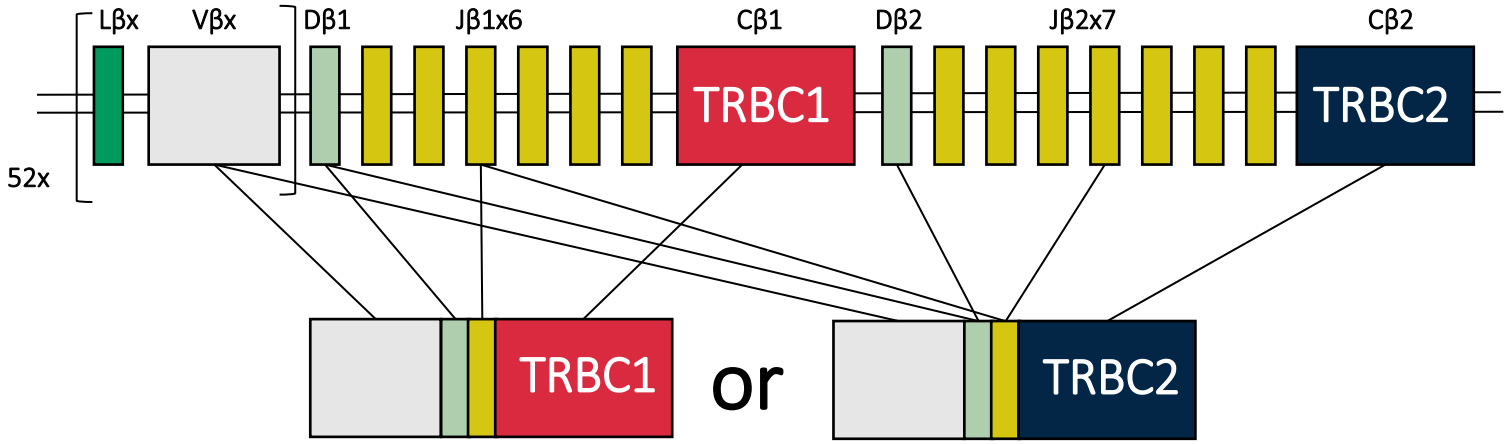


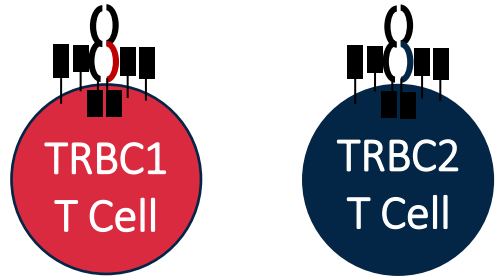
› ~ 40% of T cell lymphomas are TRBC1+

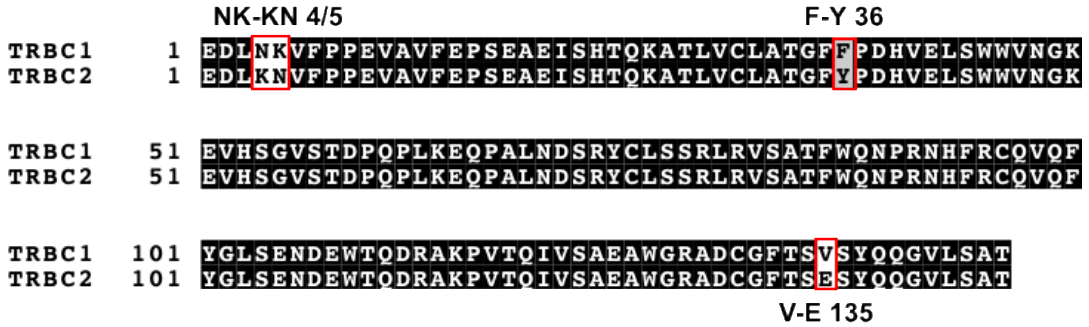
› ~ 60% of T cell lymphomas are TRBC2+

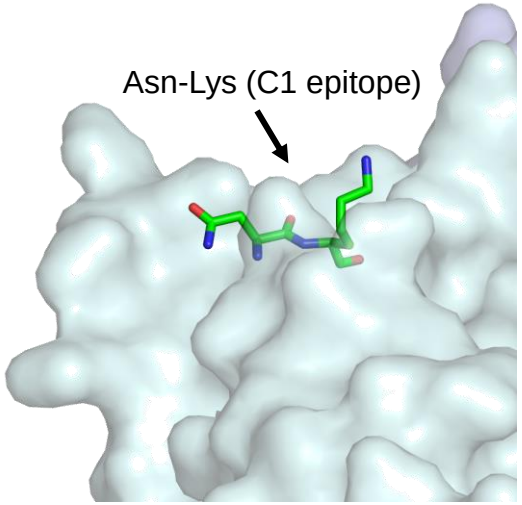
Challenges Targeting TCRβ

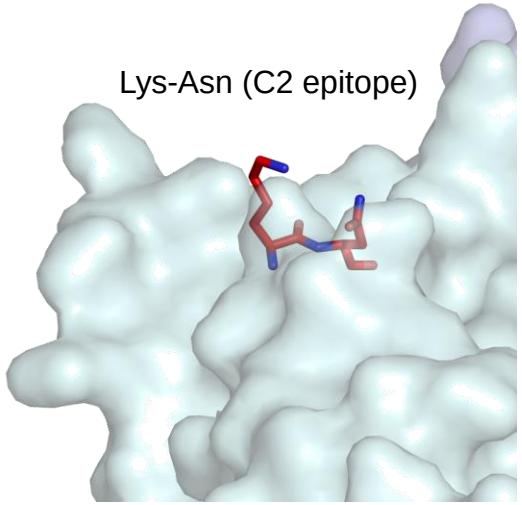
Differences between TRBC1 and TRBC2 are small











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Addressing T cell lymphomas

Three key elements - AUTO4, AUTO5 and a companion diagnostic test

Companion Diagnostic Test

Designed to stratify patients into TRBC1 and TRBC2 derived cancers

TRBC1+ patients
AUTO4

TRBC1 programmed T cell product candidate

AUTO5
TRBC2+ patients

TRBC2 programmed T cell product candidate

› ~ 40% of T cell lymphomas are TRBC1+

› ~ 60% of T cell lymphomas are TRBC2+

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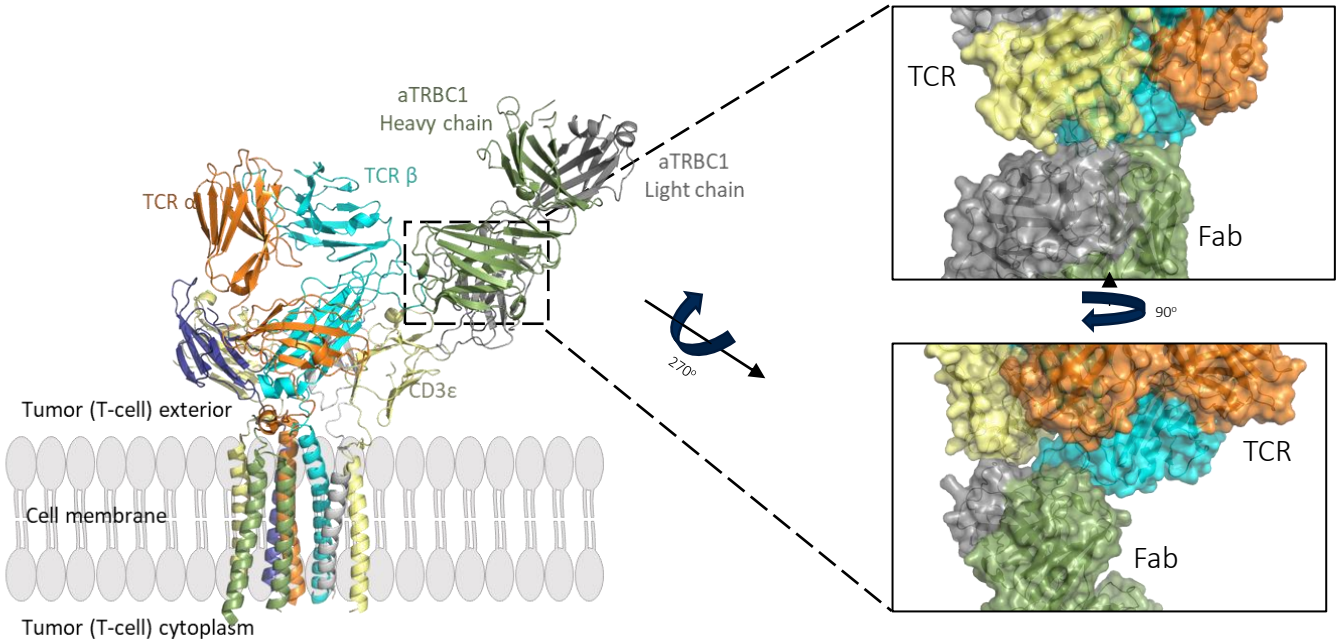
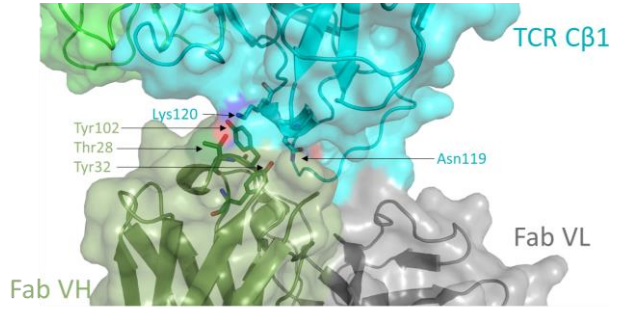
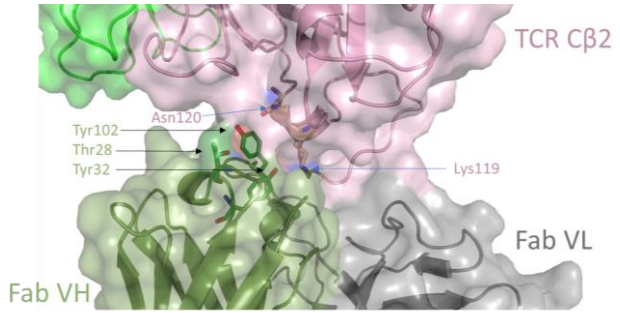
Analysis of αTRBC1-TCR contact interface (Crystalized complex 2.4Å)

αTRBC1 binds the TCR via specific contact with TCRβ1 chain in close proximity to CD3ε



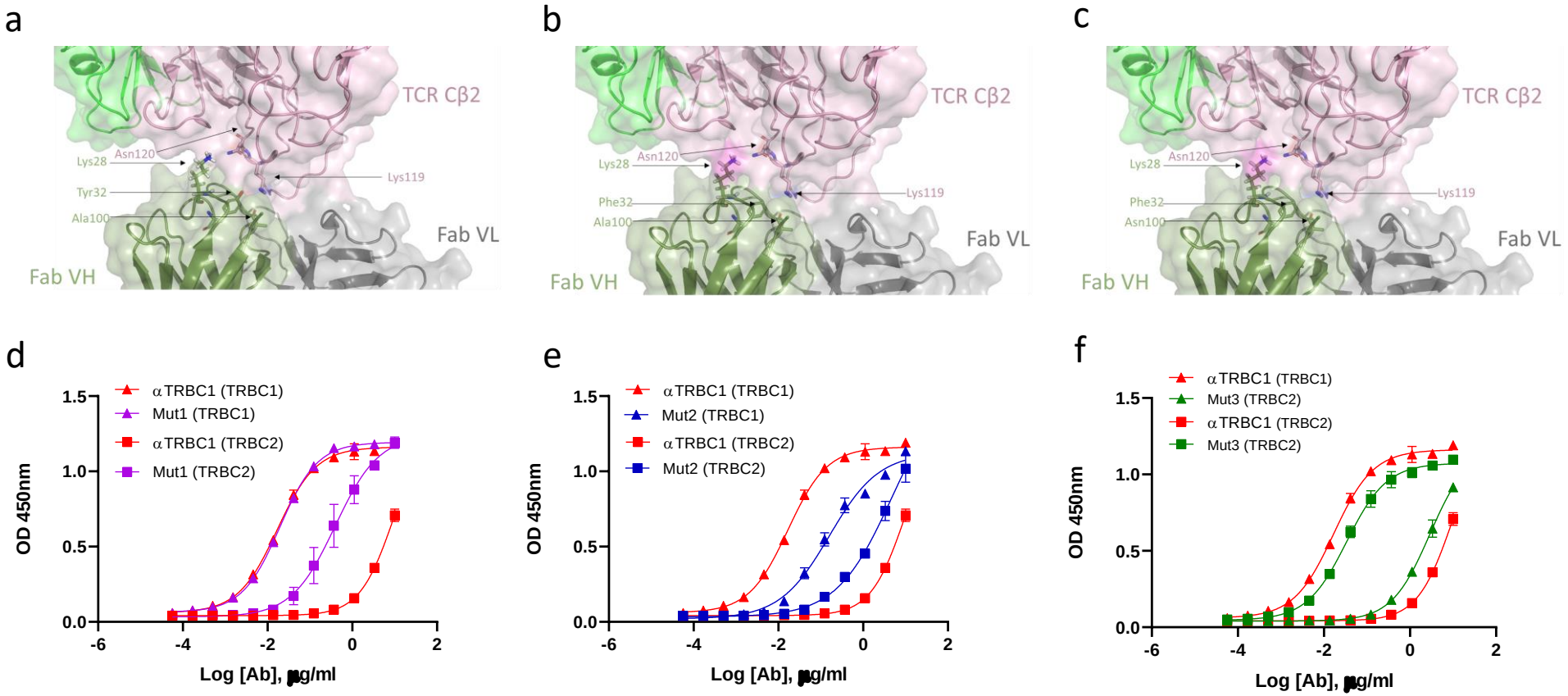




Interactions are driven via HCDR1 and HCDR3 with residues Asn119 and Lys120 on TCRβ1

Structure guided in-silico mutagenesis

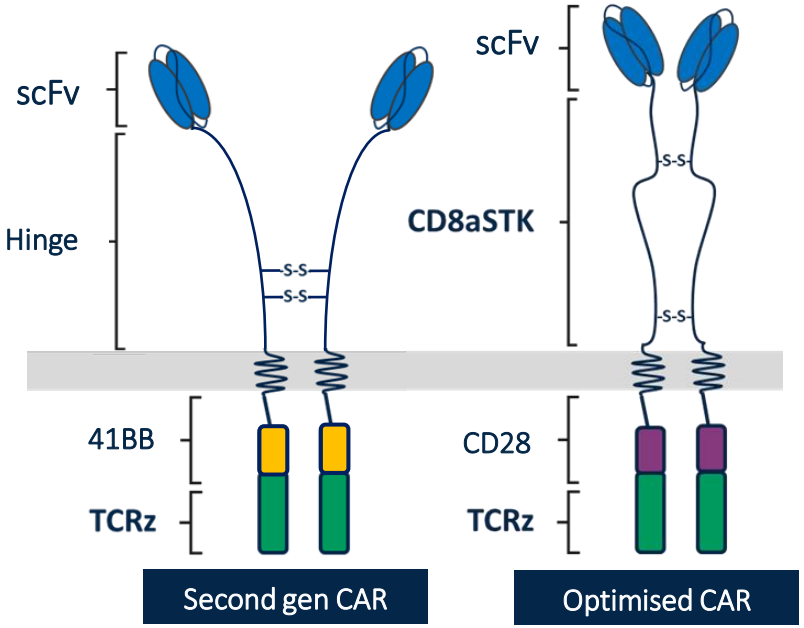
Molecular modelling identifies 3 key point mutations enhancing TRBC2 specificity

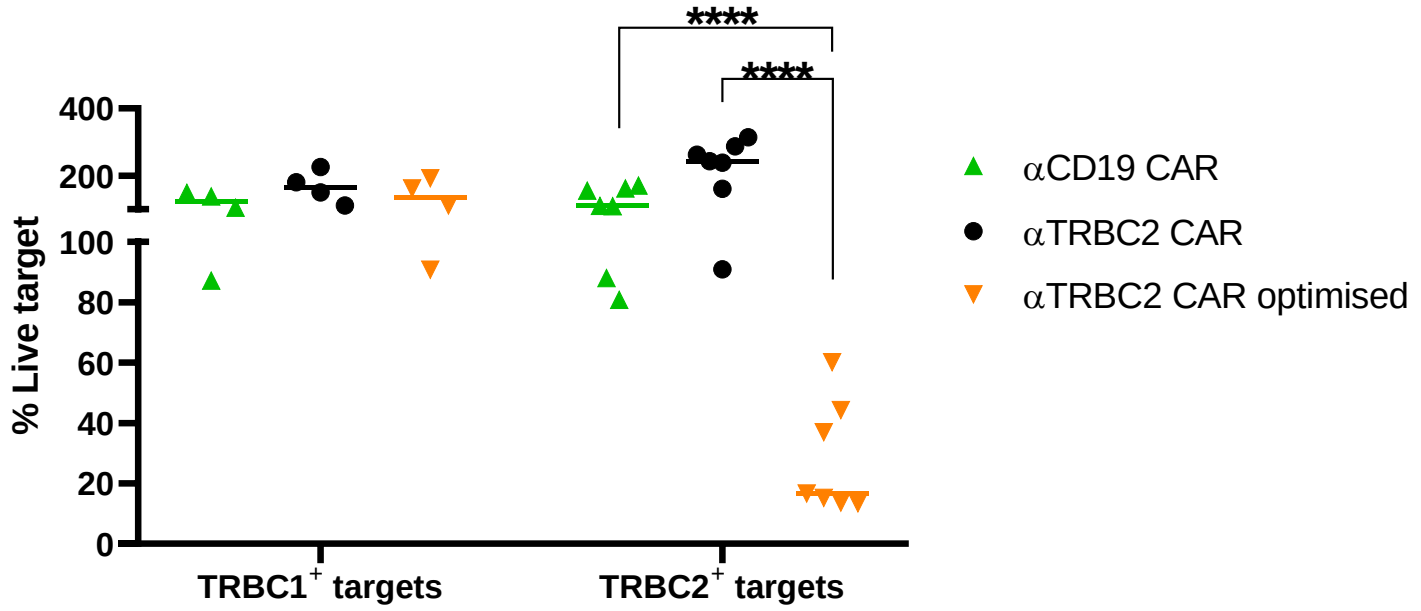


Mut3 (αTRBC2) shows an inversion of TCR specificity

CAR T cell *in-vitro* characterization

Optimized spacer/endodomain demonstrates specific cytotoxicity towards TRBC2 cells





αTRBC2 CAR efficiently kills TRBC2⁺ PBMCs at endogenous levels of TCR

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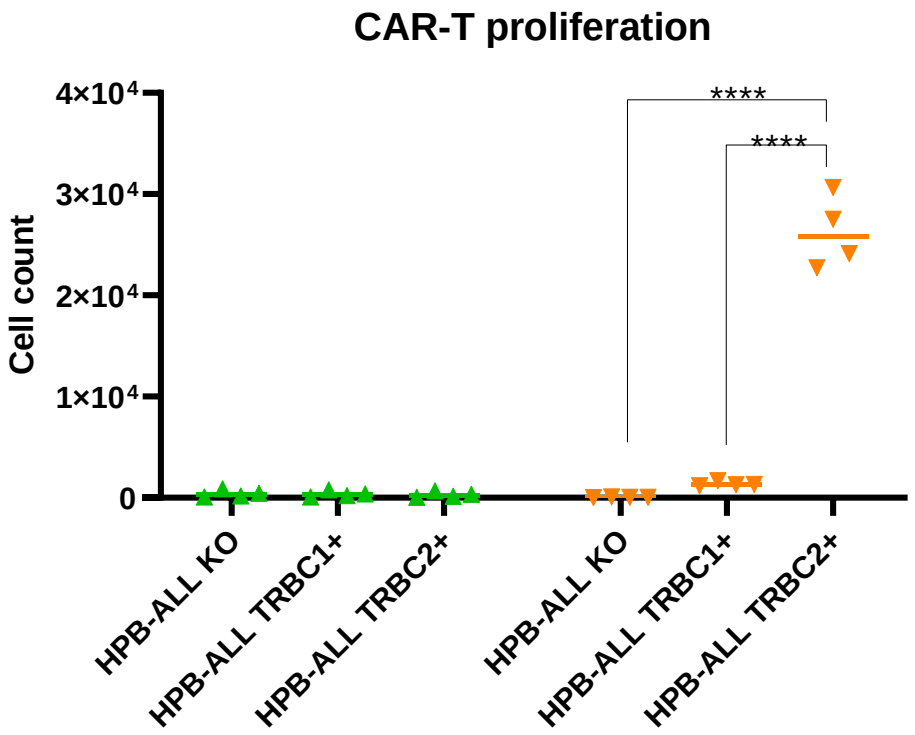
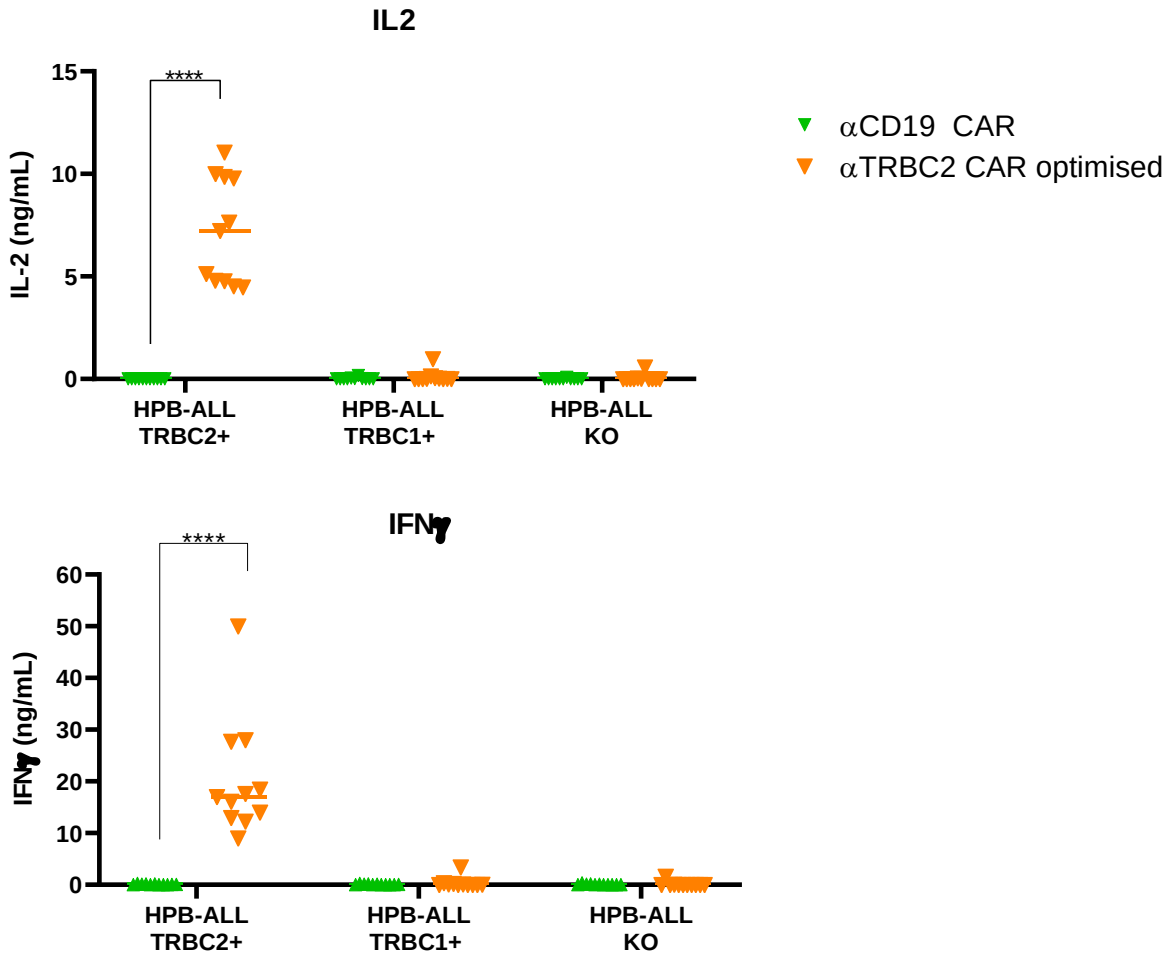
Normalized to NT PBMC population - 100% live target population is based on the non-transduced PBMC population
2-way Anova

CAR T cell *in-vitro* characterization

αTRBC2 CAR T cells show enhanced cytokine production and proliferative capacity





2-way Anova

Significant proliferation and cytokine release for αTRBC2 CAR against TRBC2⁺ target cells

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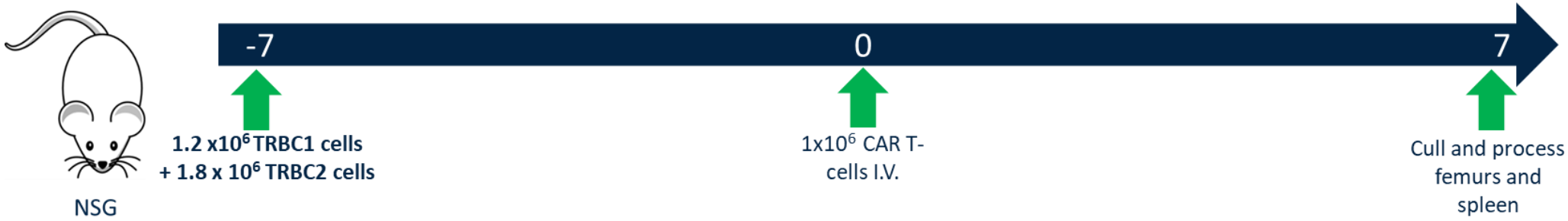
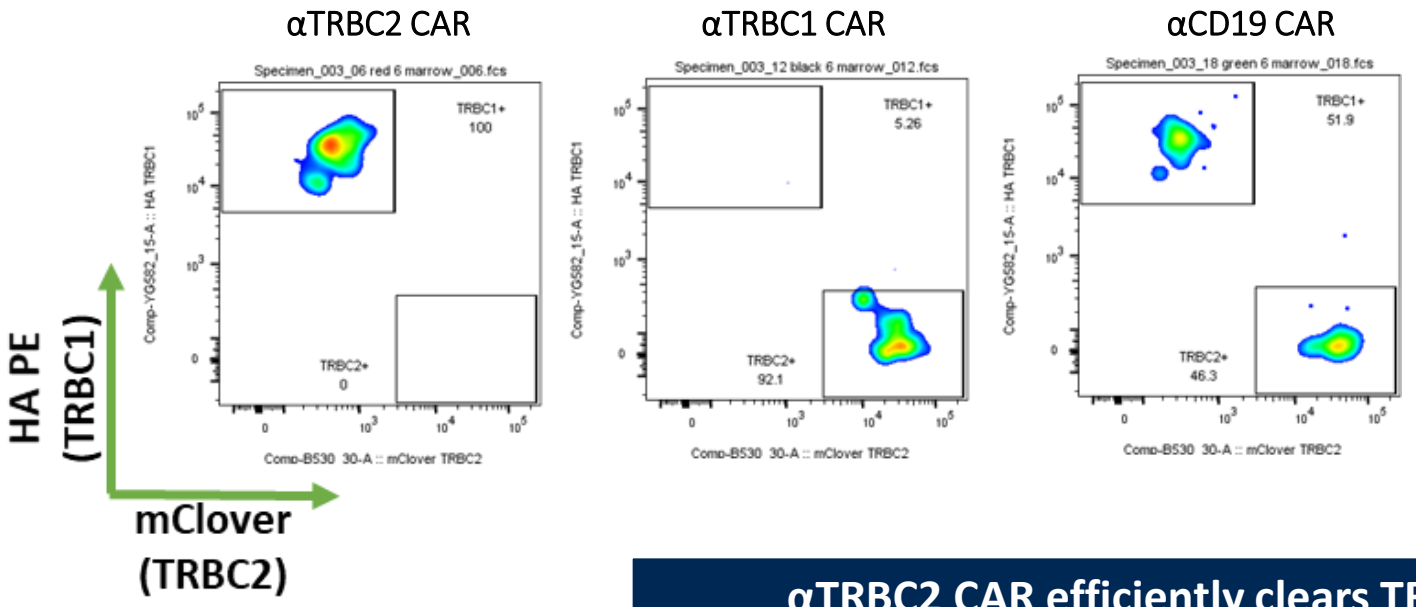
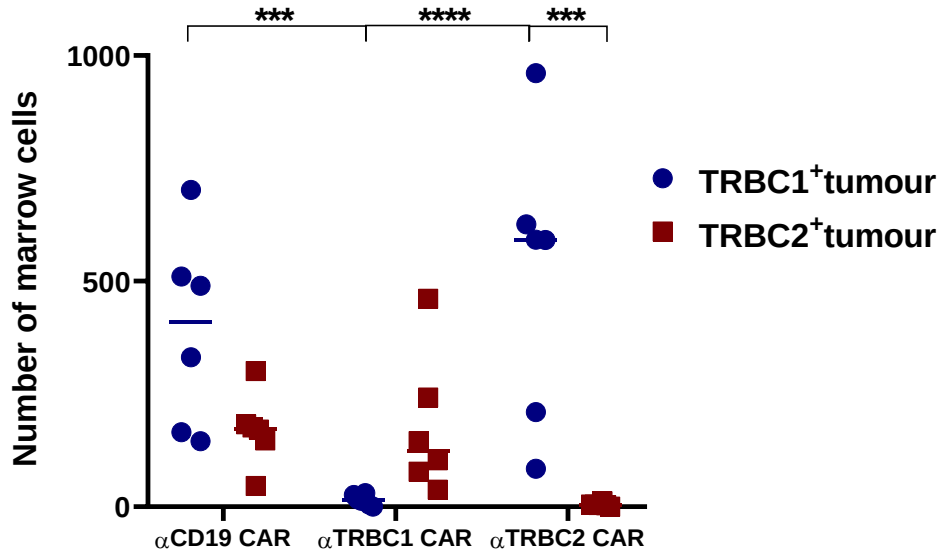
TRBC2 in-vivo CAR Activity

Selectivity of CAR targeting confirmed in mixed tumor NSG model







αTRBC2 CAR efficiently clears TRBC2⁺ targets sparing TRBC1⁺ cells in a mixed tumour model

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Summary

- Targeting TCR beta constant chain isoforms may provide a generic approach to tackle the multiple subtypes of T cell-lymphoma
- AUTO4 clinical study in progress
- Development of a selective αTRBC2 binder for CAR T therapy
- Preclinical studies package demonstrating utility of αTRBC2 CAR *in vitro* and *in vivo*
  - Efficient CAR T proliferation upon target cell interaction *in vitro*
  - Significant IFNγ and IL2 secretion in response to target cell encounter *in vitro*
  - *In vivo* clearance of TRBC2 tumor in single and mixed tumor NSG models

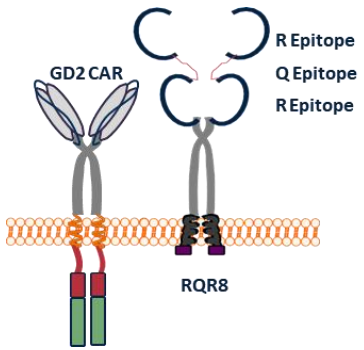
Solid Tumors

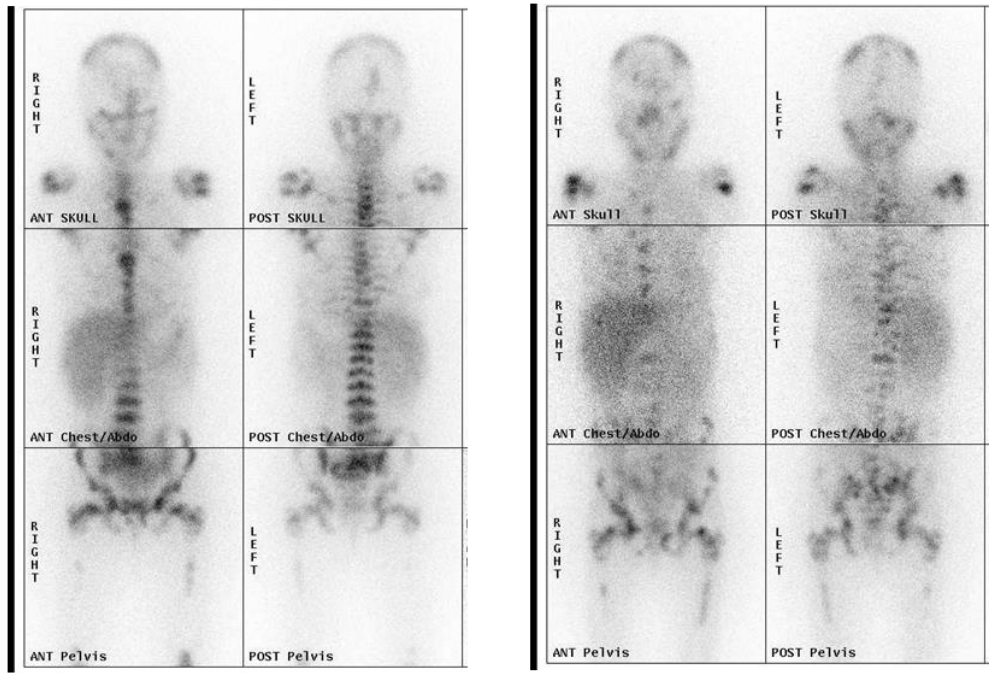
AUTO6NG – overcomes immune suppressive mechanisms in the tumor microenvironment (TME) and demonstrate preclinical anti-tumor activity in GD2-expressing solid tumors

AUTO6 designed to drive anti-tumor activity without neurotoxicity

AUTO6: GD2-targeted programmed T cell therapy in neuroblastoma

- Programmed CAR T clinical candidate
  - New binder design
    - Minimize on-target, off-tumor toxicity
    - Humanized to reduce immunogenicity
  - RQR8 safety switch
- Phase I trial in r/r Neuroblastoma conducted by CRUK in collaboration with UCL
- Autolus has the rights to clinical data and patents



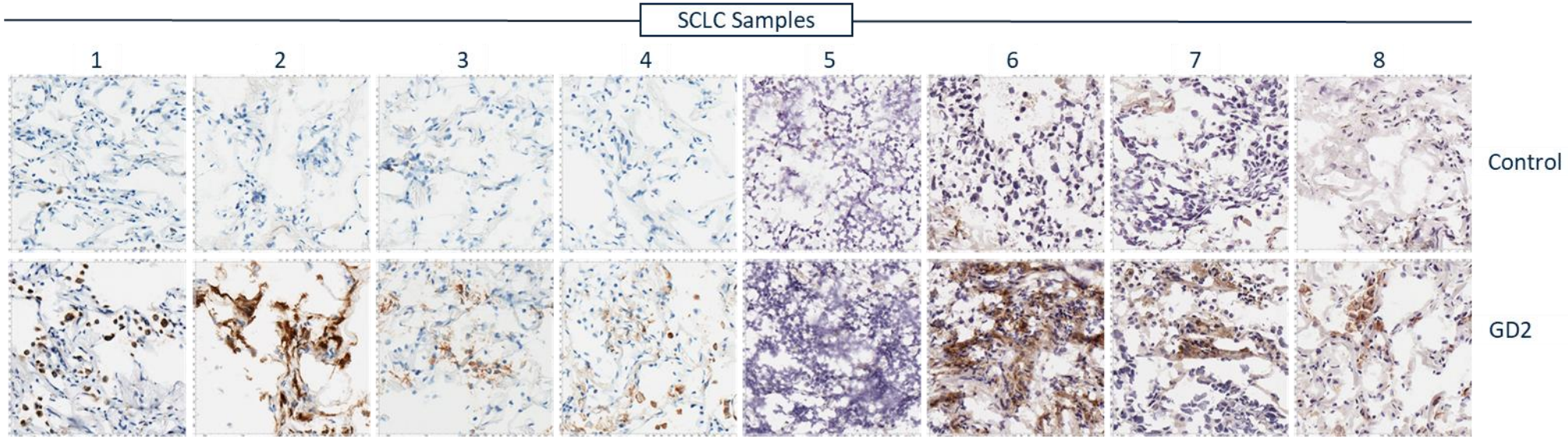


MIBG: iodine-123-meta-iodobenzylguanidine

Targeting GD2 in indications outside Neuroblastoma

Screening for GD2 expression in multiple solid tumors

- GD2 is highly expressed in Osteosarcoma, Small Cell Lung Cancer (SCLC), Melanoma, and absent in Breast Cancer (TMA IHC)
- Detailed expression in SCLC was analyzed further in a larger set (25 SCLC patients' biopsies)
  - 68% GD2 expressing (homogenous and heterogenous)
  - 32% No GD2



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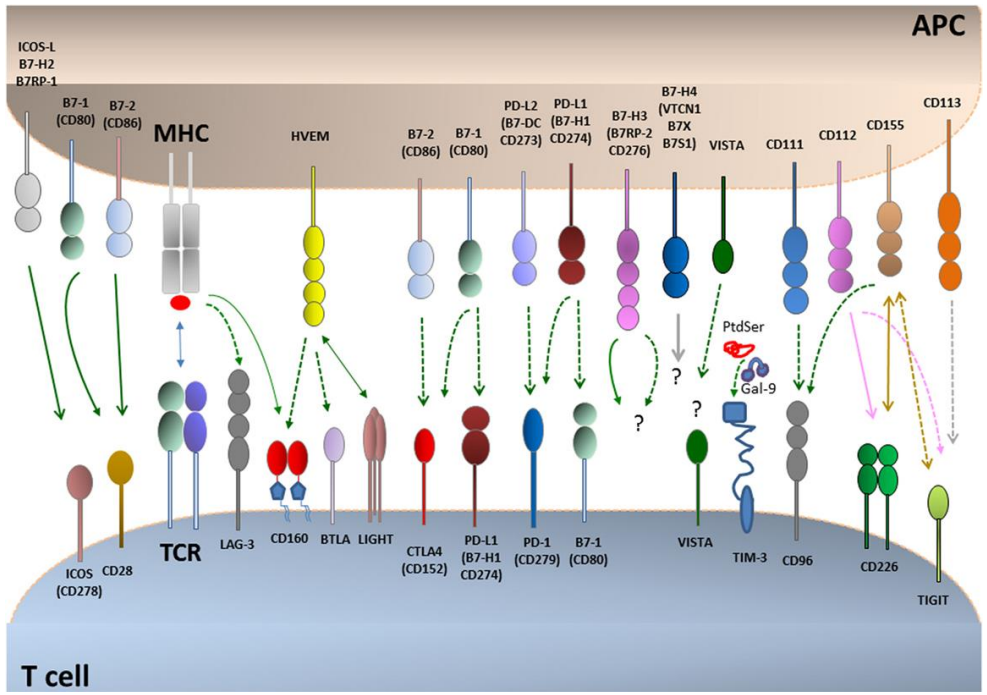
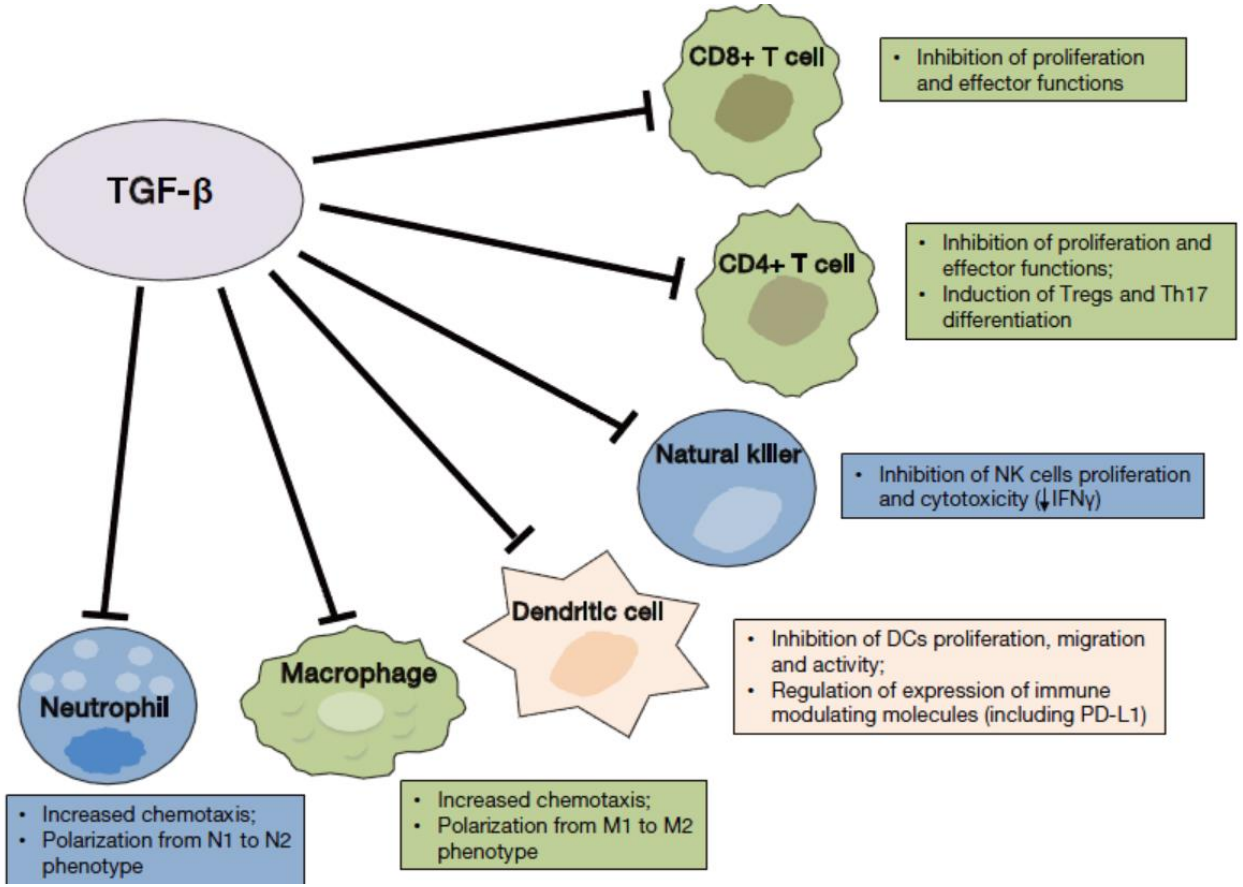
Immune suppressive pathways in SCLC

Action in T Cell Synapse



Nikolaos Patsoukis et al, Front. Immunol., 11 April 2017

Action on Immune Cell Migration



Mariacarmela Santarpia et al, TLCR., Vol 4, No 6 (December 2015)

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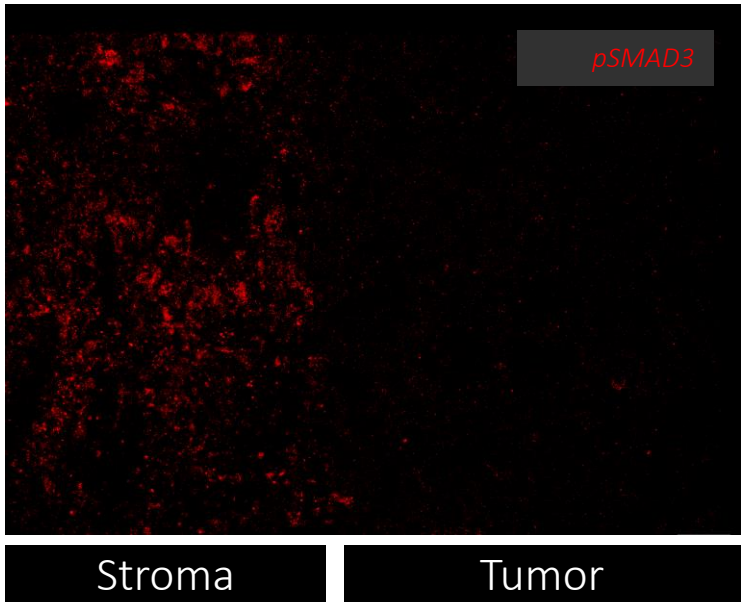
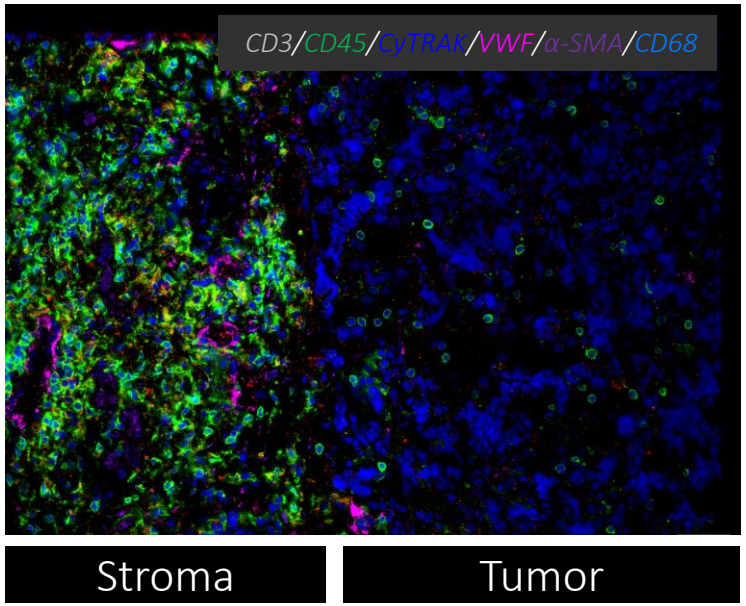
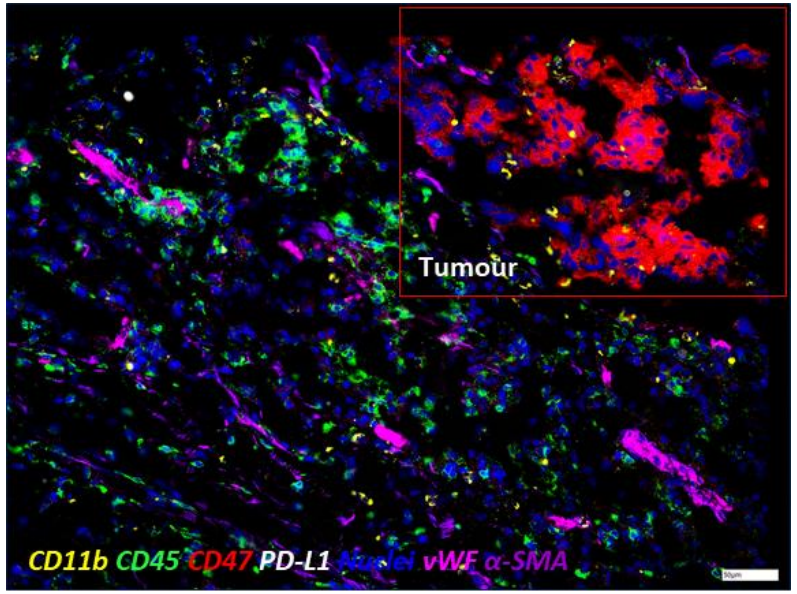
TGF-β status in a panel of SCLC biopsies

A strong pSMAD3 signal in these patients' biopsies





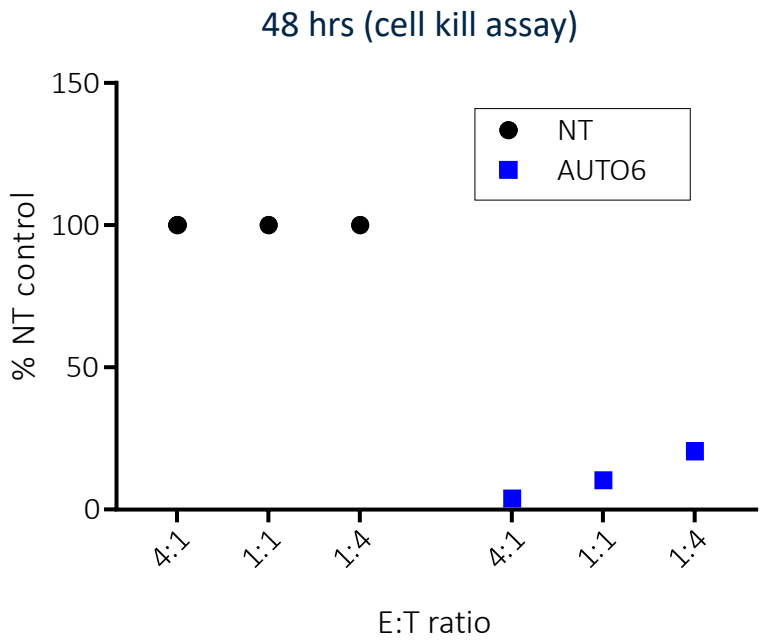


- Multiplex & multi-dimensional analysis by tissue cytometry
  - 28+ Markers Panel, analysed 10 SCLC biopsies
  - In depth characterization of the tumor cells and the components of the tumor microenvironment

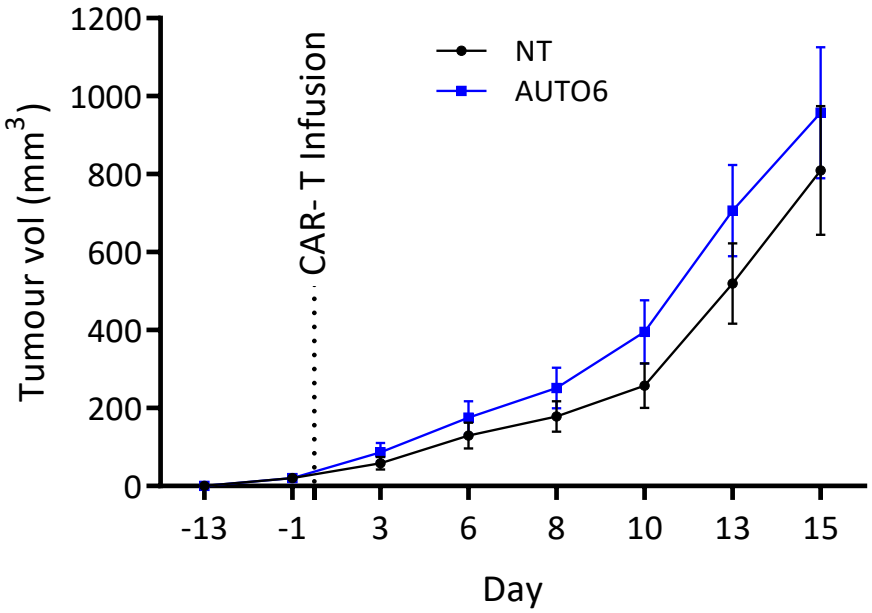
AUTO6 is active *in vitro* – but not *in vivo* in a SCLC model

AUTO6 not efficacious *in vivo* in the H446 lung cancer model

AUTO6 active *in vitro*



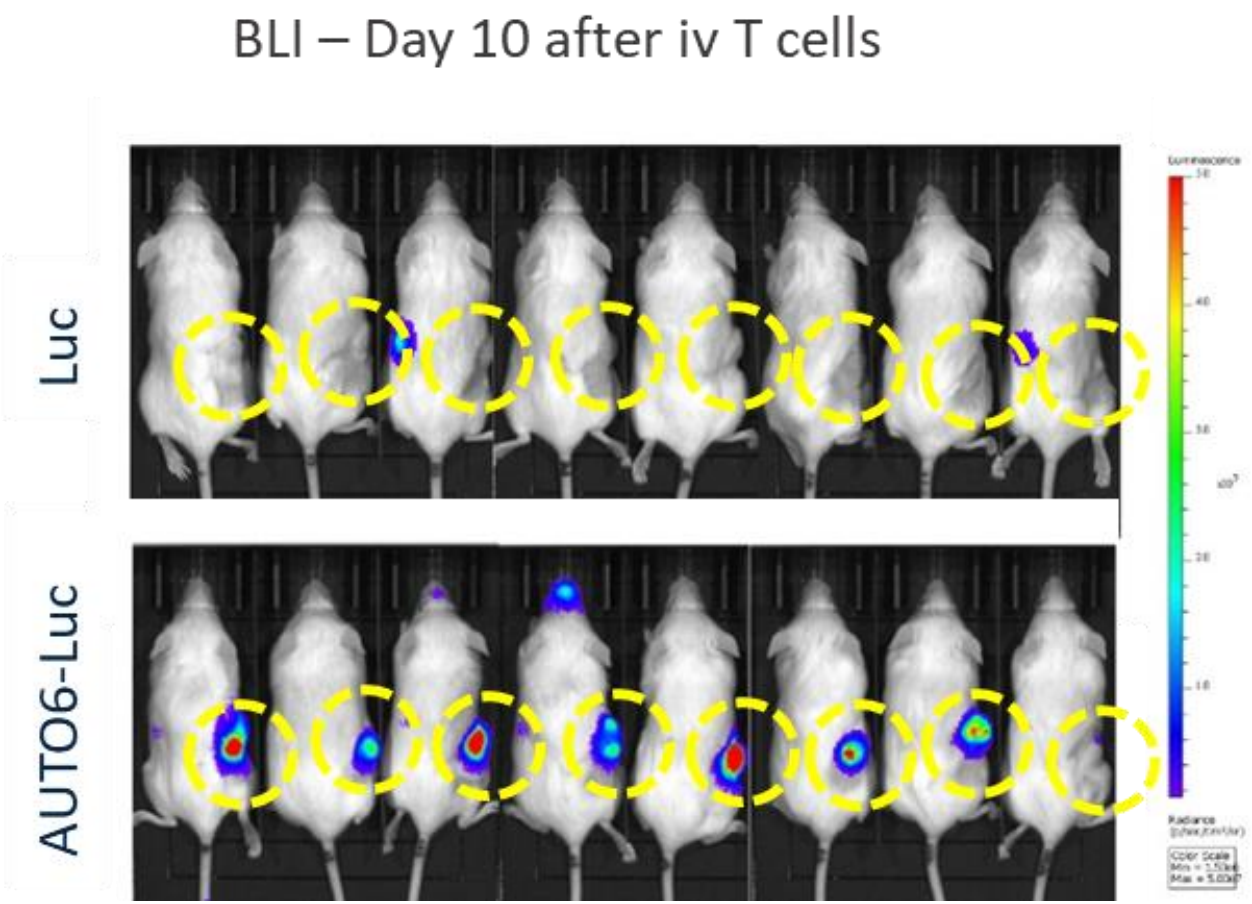
AUTO6 not active *in vivo* in the same model



H446, a SCLC cell line expressing GD2
Average tumor size at infusion 20 mm^3
Dose Administered: $7x10^6$ cells (IV)

AUTO6 CAR Ts actively traffic to tumor site

Trafficking is not culprit for lack of activity in the SCLC in vivo model



- AUTO6 cells co-localize with established subcutaneous H446 tumor
- However, CAR T cells do not appear to impact tumor cells
- Indicative of an inhibiting mechanism for the CAR Ts at the tumor site

Modular enhancements to drive AUTO6 efficacy

AUTO6NG designed to overcome tumor defenses, enhanced persistence and control

AUTO6

GD2 CAR

CAR design for optimal efficacy with minimal neurotoxicity

Safety Switch

To eliminate CAR Ts in the event of unexpected tox

dSHP2

To endow with checkpoint pathways resistance

Cytokine Signal

IL7R chimeric protein to improve persistence

dnTGFbRII* Receptor

To shield CAR Ts from immuno-suppressive signals

*dnTGFbRII, also referred to as dTGF βR

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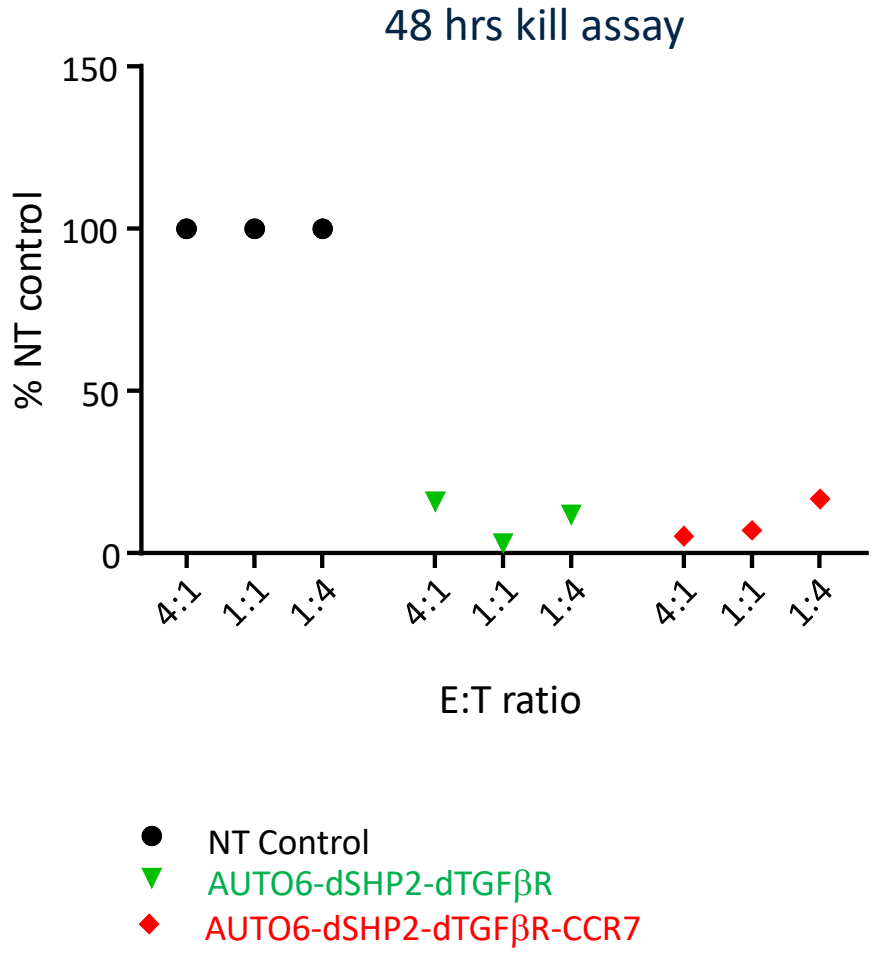
in vitro functional activity of AUTO6NG

Efficacy readily demonstrated in vitro in the H446 SCLC model

AUTO6NG without IL7R CCR*

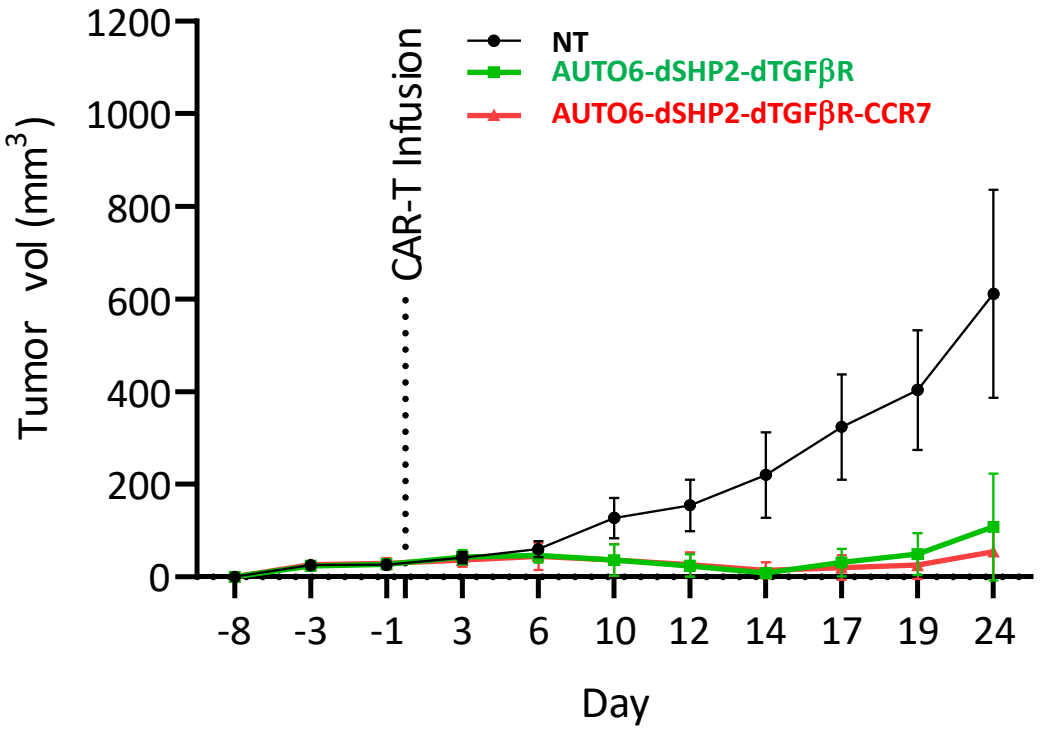
AUTO6NG



* IL7R CCR, also referred to as CCR7

in vivo functional activity of AUTO6NG in a SCLC

Addition cell programming modules drive efficacy in the H446 SCLC model



Average tumor size pre-infusion 30 mm^3

Dose Administered: $6x10^6$ CAR Ts IV

dnTGFbRII, also referred to as dTGF βR

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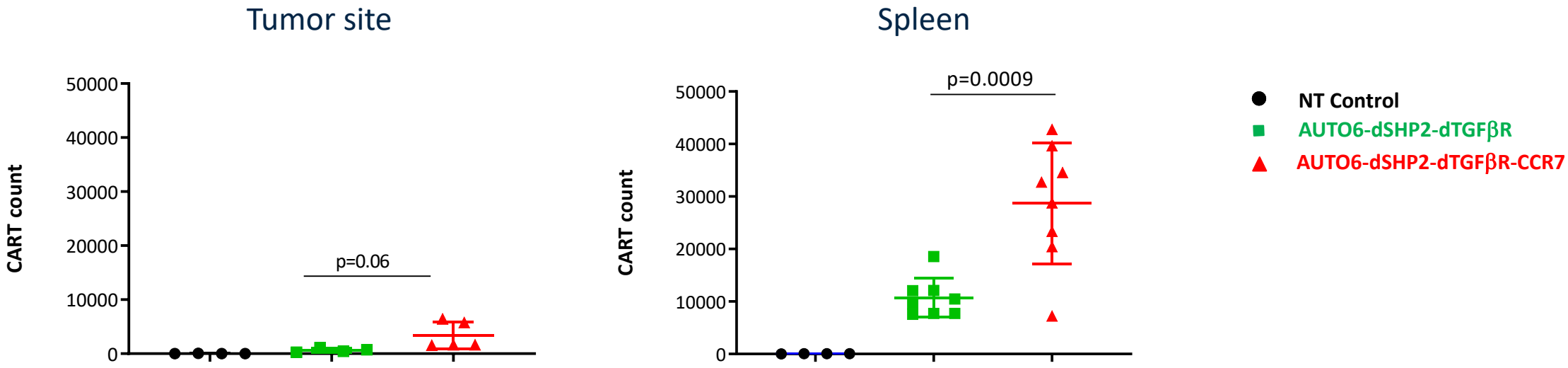
In vivo biomarkers analysis: IL7R CCR enhances CAR T expansion

CAR Ts readily detected by flow at time of euthanasia (day 34 post infusion)

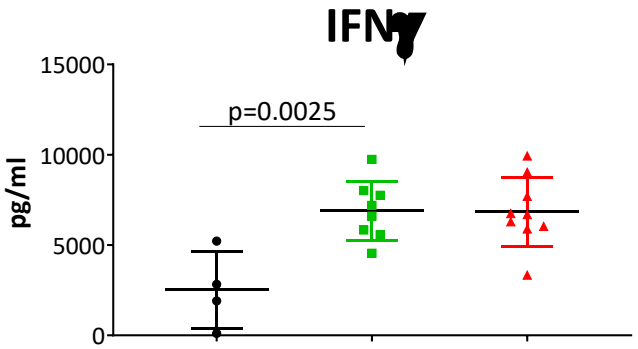


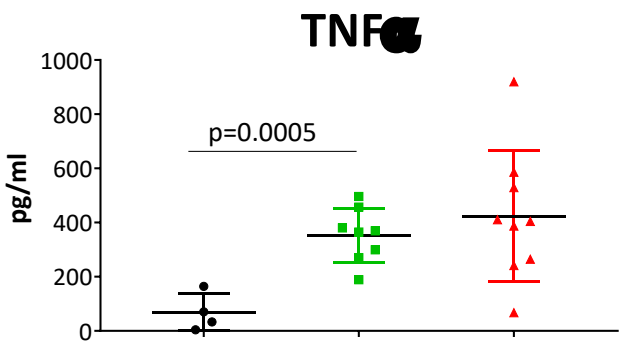
- CAR Ts persisted and detected by flow cytometry at tumor site and the spleen (Day 34)
- A higher number of CAR Ts persisted with the IL7R CCR* module

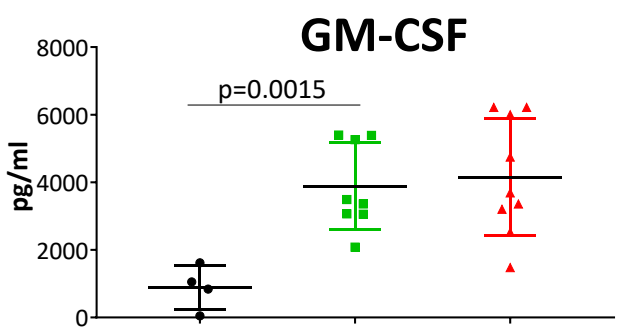
Autolus

* IL7R CCR, also referred to as CCR7

In vivo Biomarkers analysis: IL7R CCR does not drive cytokine secretion







● NT Control
■ AUTO6-dSHP2-dTGFβR
▲ AUTO6-dSHP2-dTGFβR-CCR7

Autolus

* IL7R CCR, also referred to as CCR7

Conclusion

AUTO6NG is highly active *in vitro* and *in vivo* in a SCLC tumor model

- AUTO6, a GD2 targeting CAR is clinical active in Neuroblastoma
- Our experimental data indicates that GD2 is an attractive SCLC CAR T target
- AUTO6 alone is not sufficient to drive in vivo efficacy in a SCLC mouse model and additional cell programming modules rendering the CAR T cells insensitive to TGF-b signalling and checkpoint inhibition (AUTO6NG) are required to drive efficacy
- The modules enabled AUTO6NG cells to persist and be detected by flow on day 34 post infusion, and this expansion enhancement was without increasing cytokines levels *in vivo*
- AUTO6NG will be clinically explored in GD2 expressing tumors

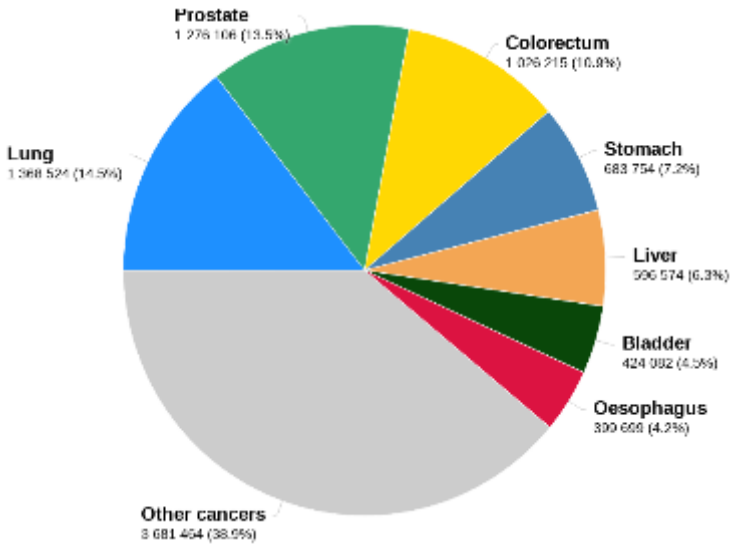
AUTO7 – Anti-PSMA humanized CAR T cell with improved persistence and resistance to tumor microenvironment for metastatic castration resistant prostate cancer (mCRPC)

Metastatic Castration-Resistant Prostate Cancer (mCRPC)

mCRPC is a large commercial market but has few effective treatment options

- Prostate cancer is the second most common male cancer in the world[1]
  - affects ~1.3 million and kills >360,000 people each year
  - addressable patient population for 2nd line mCRPC is projected at c.80,000 patients*
- High unmet medical need[2]
  - patients diagnosed with localized disease usually treated with surgery or radiotherapy; however, many develop more advanced recurrent disease
  - patients with advanced prostate cancer are primarily treated with hormone ablation therapy, yet tumors ultimately progress to become castration resistant[3,4,5]
  - once metastasized the 5-year survival rate falls to less than 30%
  - existing therapies are not curative, while cell based therapies have the potential to be curative

Estimated number of new cases worldwide



1. Data source: Globocan 2018; 2. Litwin MS et al. JAMA 2017; 3. Karzai, et al. South Med J. 2015; 4. Mohler ML, et al. Expert Opin Ther Pat 2012;
5. Papalia MA & Davis SR. Treat Endocrinol 2003
* Autolus assumptions

Biological hurdles to address in mCRPC therapy

Therapeutic approach needs to address a complex tumor microenvironment (TME)

- Opportunity for immunotherapy exists but multiple parameters need to be addressed due to the complex TME influencing the response to immunotherapy:
  - Late stage prostate cancers appear to be immunologically cold tumors with minimal T cell infiltrates[1]
  - mCRPC have limited response to single-agent checkpoint inhibition; despite promising results in early trials, anti-CLTA4 Ipilimumab showed a marginal response in mCRPC patents[2,3]
  - PD-1/PDL1 targeting antibodies have been equally inadequate[4,5]
  - Tackling overproduction of TGFβ could overcome the immunosuppressive effects of this cytokine in the TME[6]
  - Acidification of the TME play an important role in the efficacy of therapeutics that target tumors[7]
- An effective immunological approach will have to combine multiple modalities. Systemic combination using protein or small molecule drugs risks inducing severe and cumulative adverse events

Autolus

1. Spranger S et al. Oncoimmunology 2016; 2. Beer TM et al. J Clin Oncol 2017; 3. Kwon ED et al. Lancet Oncol 2014; 4. Kwon et al. Lancet Oncol 2014; 5. Antonarakis ES et al J Clin Oncol 2020; 6. Siegel PM et al. Nat. Rev. Cancer. 2003; 7. Pilon-Thomas S et al. Cancer Res 2016

AUTO7 strategy is a CAR T approach with modular cellular programming

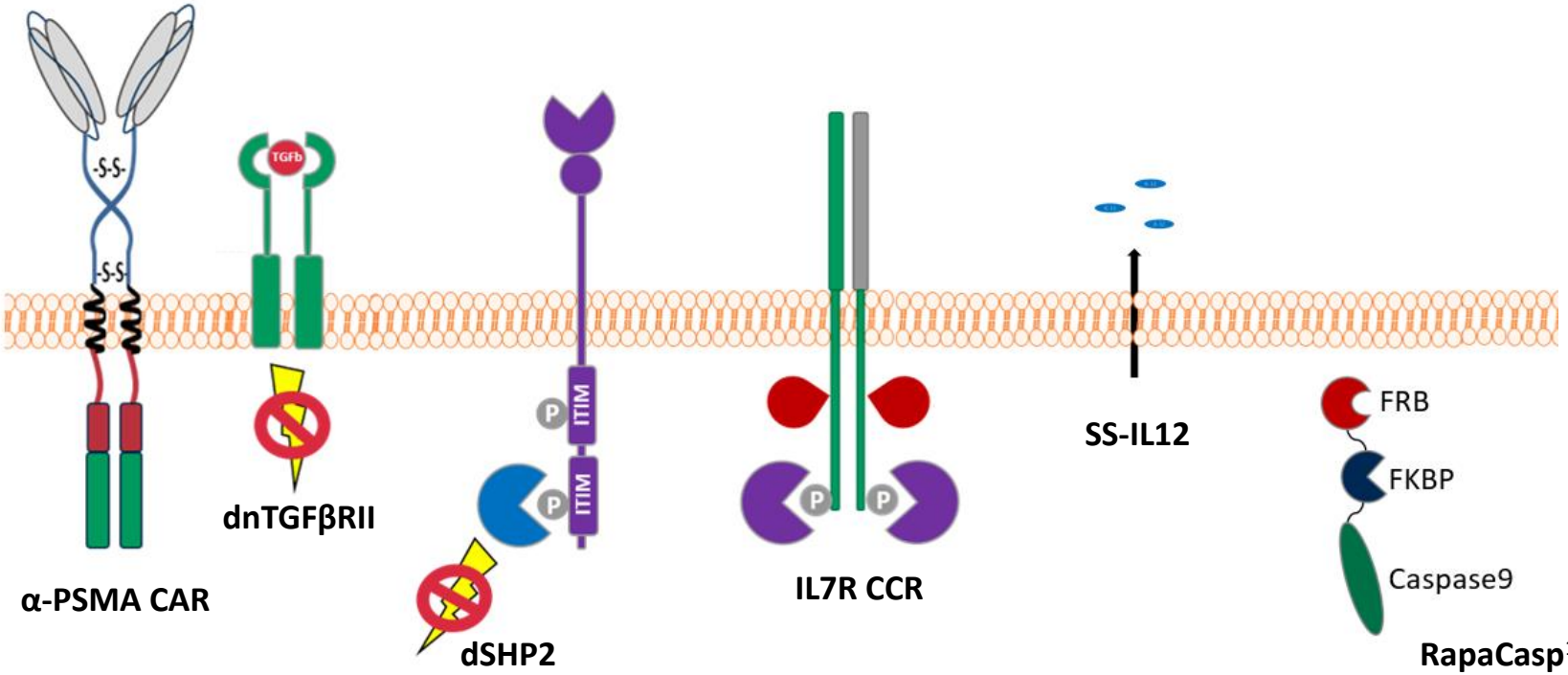
Our goal is to generate resilient CAR T cells withstanding hostile tumor environment

Feature	Biology	Protein Module	Systemic Impact
Specific Targeting	Novel anti-PSMA binder	PSMA-CAR	No
Resilience to Checkpoint Blockade	Block inhibitory receptor signals	dSHP2	No
Avoid TME-driven inhibition	Block TGFβ signal	dnTGFβRII	No
Support CAR T cell Survival	Promotes Jak/Stat signal	IL7R CCR	No
Induce Specific Immune Response	Activate immune response at the tumor site	Low level secreted IL-12	No – limited to immediate tumor environment

IL7R CCR, also referred to as CCR7
dnTGFbRII, also referred to as dTGF βR

AUTO7 modules designed to tackle complex solid tumour environment

Technology toolbox utilized to overcome hostile TME



PSMA CAR	dnTGFβRII	dSHP2	Cytokine signal	Immune Adjuvant	Safety Switch
Effective targeting of PSMA expressing tumor cells	Truncated TGFβRII receptor to shield inhibitory effect of TGFβ	Truncated SHP2 to block checkpoint inhibition	Cytokine signal to overcome minimal co-stimulation	Secretion of IL-12 cytokine to promote host immune activation	To terminate therapy in the event of unexpected toxicities

Modules delivered using gamma-retroviral vector

Autolus

1. Stavrou M et sl. Mol Ther 2018

An ultra sensitive AUTO7 CAR directed to the PSMA ligand on mCRPC

Module	Function
α-PSMA CAR	Specific lysis of mCRPC
RapaCasp	Suicide gene
dnTGFβRII	Shield TGFβ suppression
dSHP2	block checkpoint inhibition
IL7_CCR	Promote CAR T cell persistence
SS-IL12	Host Immune Cell Recruitment

- PSMA is one of the most commonly expressed genes found in prostate cancers
- PSMA pattern of expression can vary with the degree of differentiation, and heterogeneity of expression can vary within the same tumor[1]

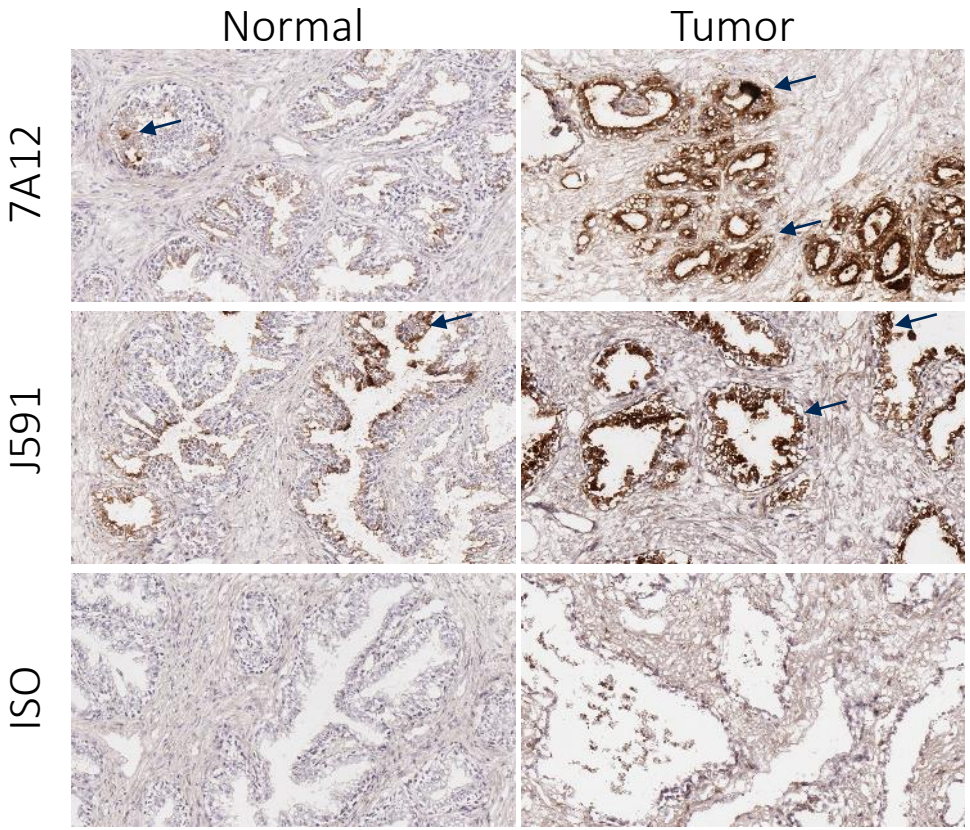
Autolus

1. Silver DA et al. Clin Cancer Res 1997

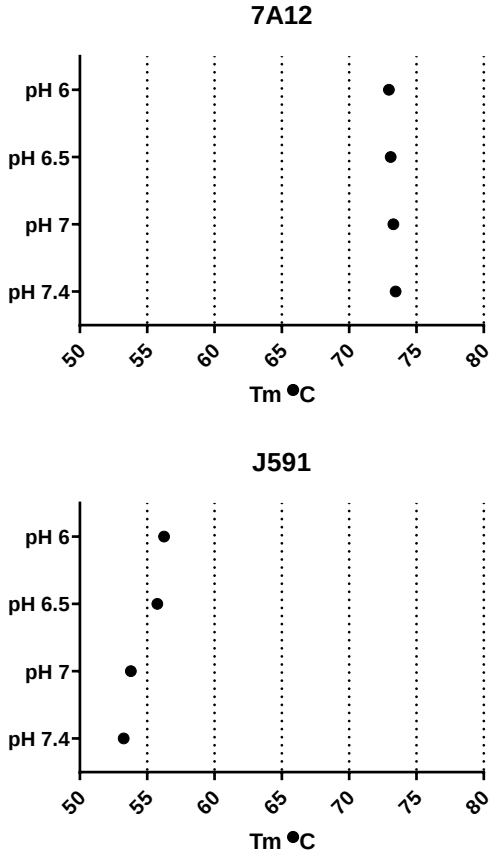
An ultra sensitive AUTO7 CAR directed to the PSMA ligand on mCRPC

In house binder campaign to develop a resilient anti-PSMA binder that is stable at low pH

Staining on frozen prostate tissues



Effect of pH on stability

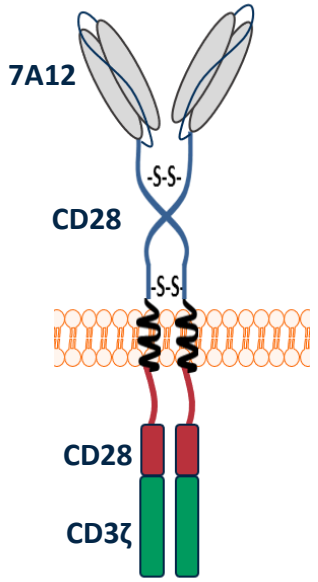


Autolus

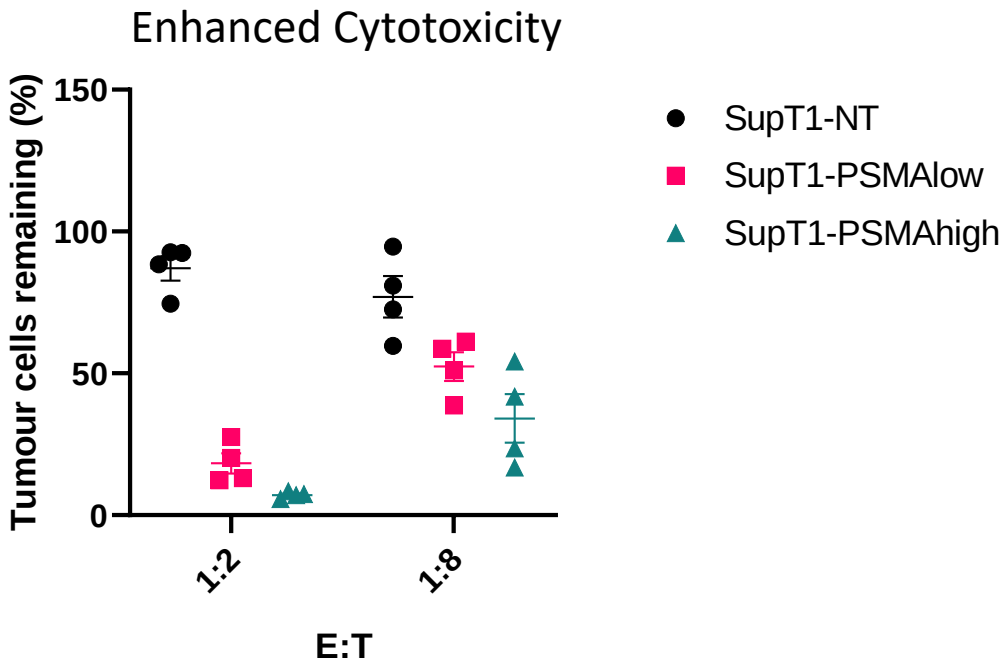
An ultra sensitive AUTO7 CAR directed to the PSMA ligand on mCRPC

Humanized α-PSMA CAR in AUTO7 is effective at killing high and low density PSMA positive cells

PSMA CAR



Target cell line	PSMA expression levels by ABC
SupT1-NT	0
SupT1-PSMAlow	578
SupT1-PSMAhigh	16500



Autolus

dnTGFβRII module desensitizes AUTO7 T cells to TGFβ

Module	Function
α-PSMA CAR	Specific lysis of mCRPC
RapaCasp	Suicide gene
dnTGFβRII	Shield TGFβ suppression
dSHP2	block checkpoint inhibition
IL7_CCR	Promote CAR T cell persistence
SS-IL12	Host Immune Cell Recruitment

- TGFβ is an anti-inflammatory cytokine and is expressed at high levels in the TME[1]
- T cells are key targets for TGFβ mediated immune suppression; tumor-derived TGFβ compromises T-cell mediated antitumor immunity[2]
- TGFβ signalling promotes the epithelial-to-mesenchymal transition associated with high-grade mCRPC malignancy[3] and acts as an important mediator of metastasis to specific organ sites

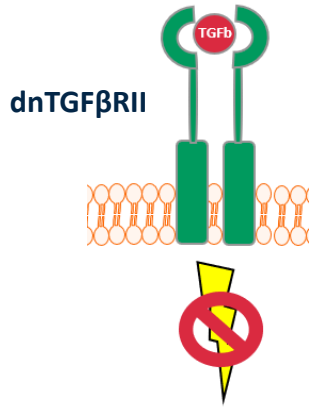
Autolus

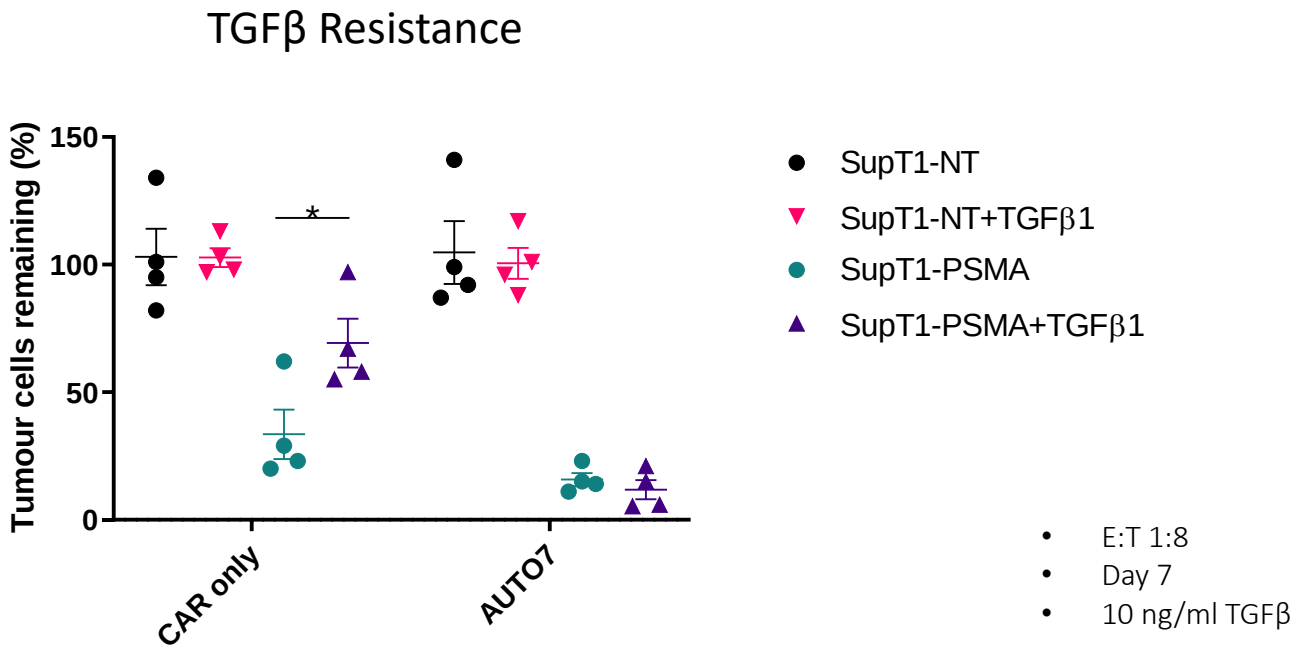
1. Siegel PM et al. Nat. Rev. Cancer. 2003; 2. Gorelik L et al. Nature Med 2001; 3. Montanari M et al. Oncotarget. 2017

dnTGFβRII module desensitizes AUTO7 T cells to TGFβ

- dnTGFβRII maintains AUTO7 T cells cytolytic function in presence of an inhibitory cytokine TGFβ

dnTGFβRII





- E:T 1:8
- Day 7
- 10 ng/ml TGFβ

Autolus

dSHP2 module shields AUTO7 T cells from checkpoint inhibition

Module	Function
α-PSMA CAR	Specific lysis of mCRPC
RapaCasp	Suicide gene
dnTGFβRII	Shield TGFβ suppression
dSHP2	block checkpoint inhibition
IL7_CCR	Promote CAR T cell persistence
SS-IL12	Host Immune Cell Recruitment

- SHP2 mediates signaling from many inhibitory receptors such as PD-1[1]
- mCRPC are known to overexpress the PD-1 ligand, PD-L1[2]
- Antibody blockade of the PD-1/PD-L1 pathway in mCRPC has resulted in partial clinical responses[3]

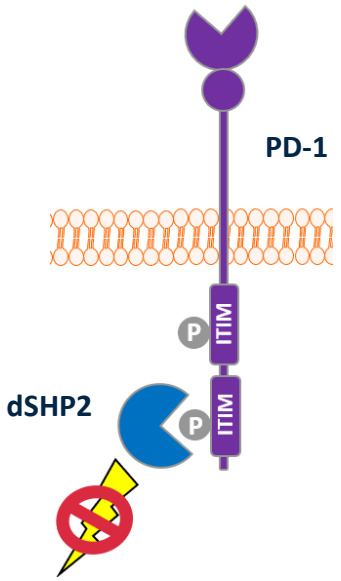
Autolus

1. Salmond RJ et al. Trens Immunol 2006; 2. Gevensleben H et al. Clin Cancer Res 2016; 3. Antonarakis ES et al J Clin Oncol 2020

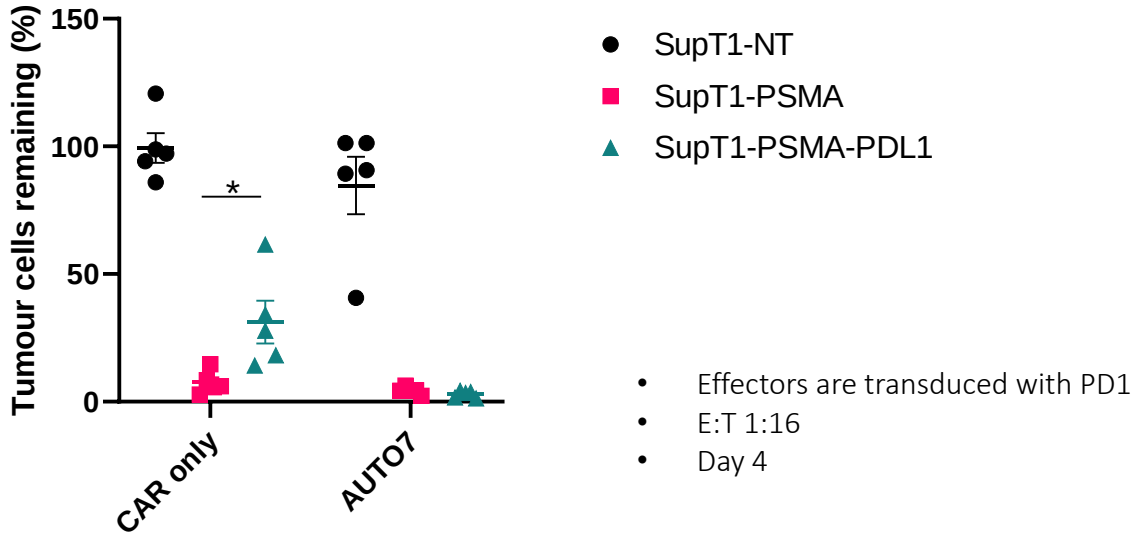
dSHP2 module shields AUTO7 T cells from checkpoint inhibition

dSHP2 module maintains AUTO7 cytolytic function in the presence of PD1/PDL1 signalling

dSHP2



Checkpoint Resistance



- Effectors are transduced with PD1
- E:T 1:16
- Day 4

Autolus

IL7R CCR module induces AUTO7 T cell persistence

Module	Function
α-PSMA CAR	Specific lysis of mCRPC
RapaCasp	Suicide gene
dnTGFβRII	Shield TGFβ suppression
dSHP2	block checkpoint inhibition
IL7R CCR	Promote CAR T cell persistence
SS-IL12	Host Immune Cell Recruitment

- CAR T cell persistence correlates with clinical efficacy[1,2]
- CAR T programs to solid tumors report poor persistence; T cells require cytokine signaling to proliferate
- IL7R CCR delivers a constitutive IL7 signal to the CAR T cells and prolongs CAR T cell persistence

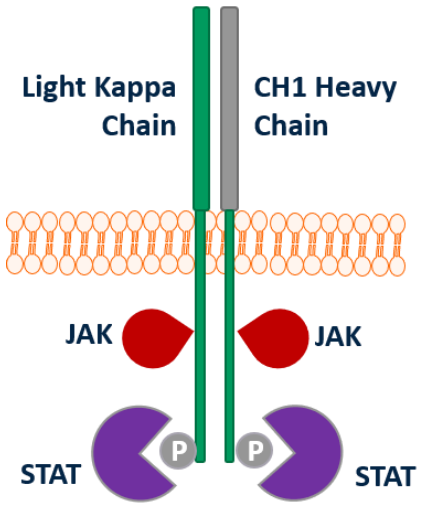
Autolus

1. Mueller KT et al. BLOOD 2017; 2. Mueller KT et al. Clin Cancer Res 2018

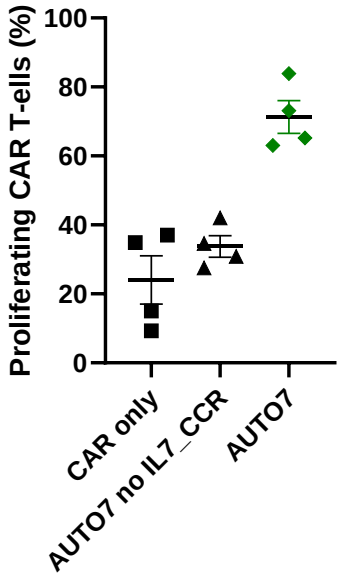
IL7R CCR module induces AUTO7 T cell persistence

IL7R CCR extends AUTO7 activity through multiple rounds of re-stimulation

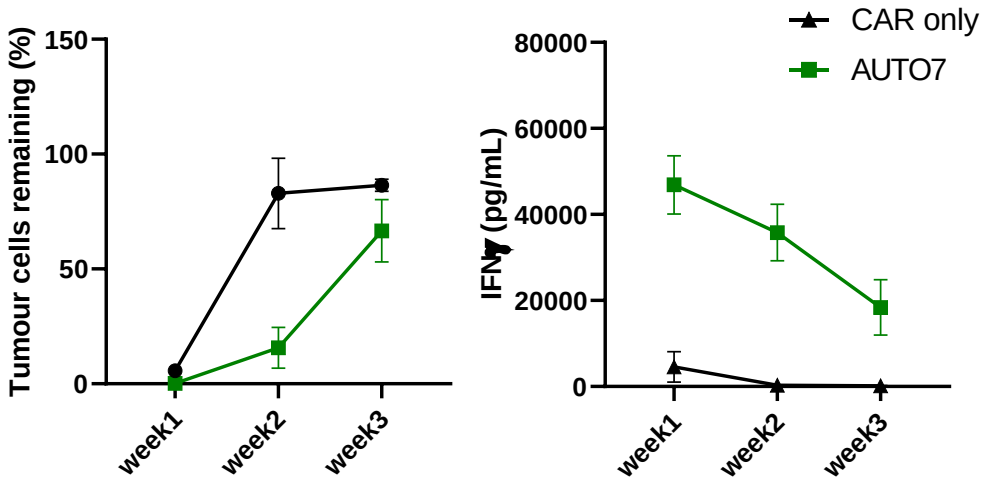
Cytokine signal



CCR driven proliferation



Enhanced serial killing



- E:T 1:2, CART-cells weekly rechallenged with fresh SupT1-PSMA target cells

Autolus

SS-IL12 module maintains potent adjuvant activity with no toxicity

Module	Function
α-PSMA CAR	Specific lysis of mCRPC
RapaCasp	Suicide gene
dnTGFβRII	Shield TGFβ suppression
dSHP2	block checkpoint inhibition
IL7_CCR	Promote CAR T cell persistence
SS-IL12	Host Immune Cell Recruitment

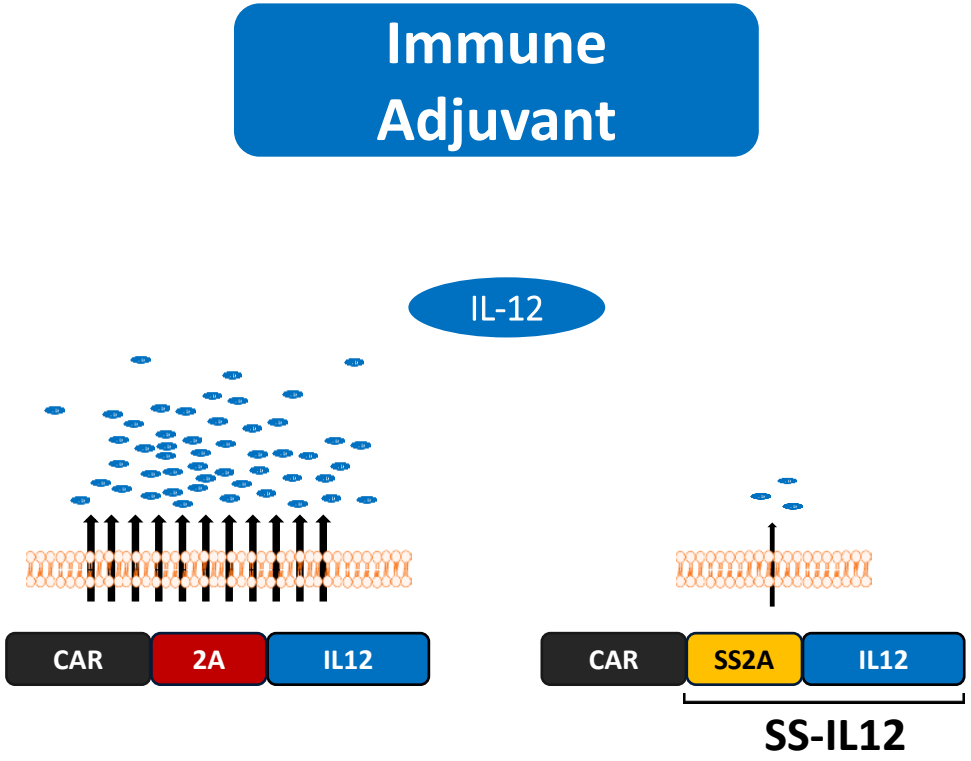
- IL-12 is a potent adjuvant that promotes a powerful anti-tumor response when combined with checkpoint inhibitors and adoptive T cell transfer[1]. However, IL-12 has been associated with systemic toxicity[2]
- Engineered version of the IL-12 (SS-IL12) has been designed to constrain expression while maintaining its adjuvant activity

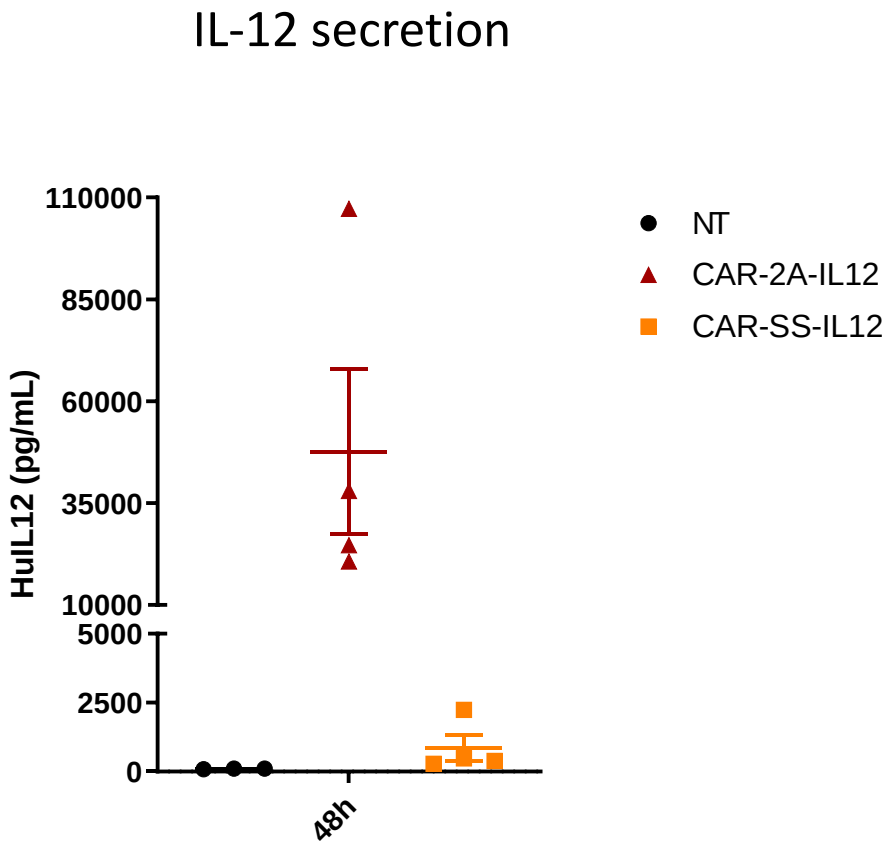
Autolus

1. Quetglas JI et al. Cancer Immunol Res 2015; 2. Tugues S et al. Cell Death Differ 2015

SS-IL12 module maintains potent adjuvant activity with no toxicity

SS-IL12 module prevents toxicity by drastically reducing IL-12 secretion

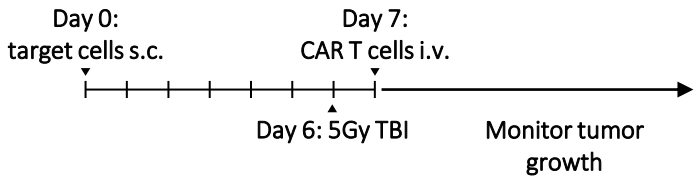


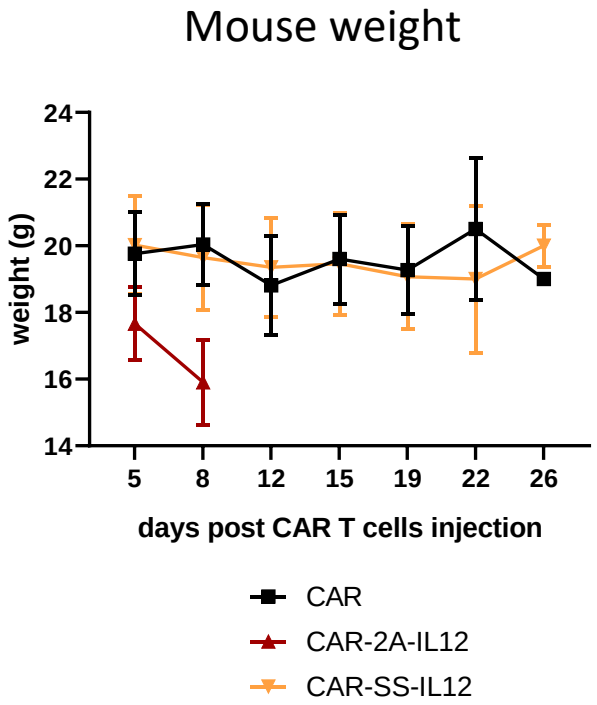


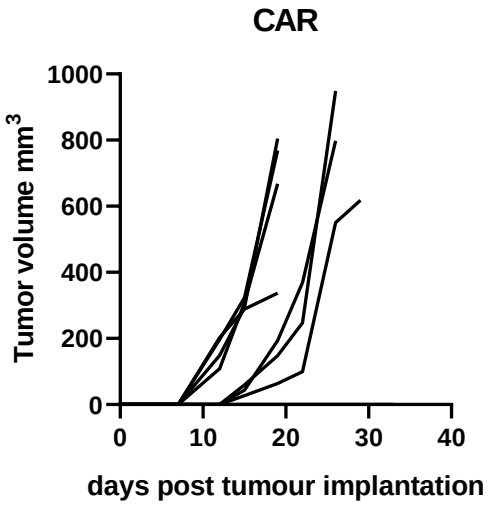
SS-IL12 module maintains potent adjuvant activity with no toxicity

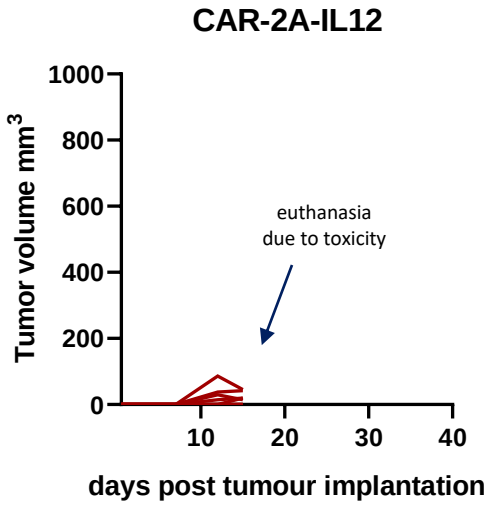
SS-IL12 module prevents toxicity and maintains potent anti-tumor activity in a murine model

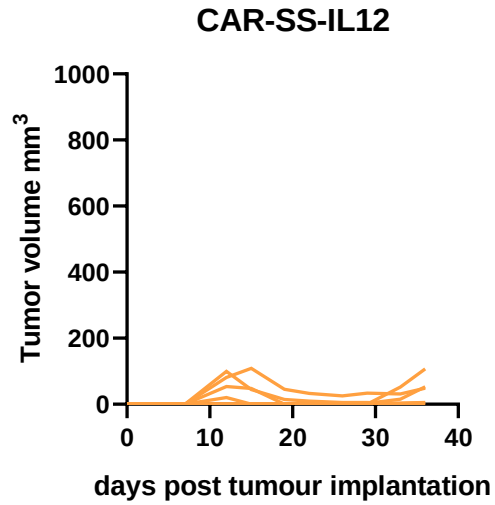
- Mouse B16.F10 cells in C57BL/6 mice







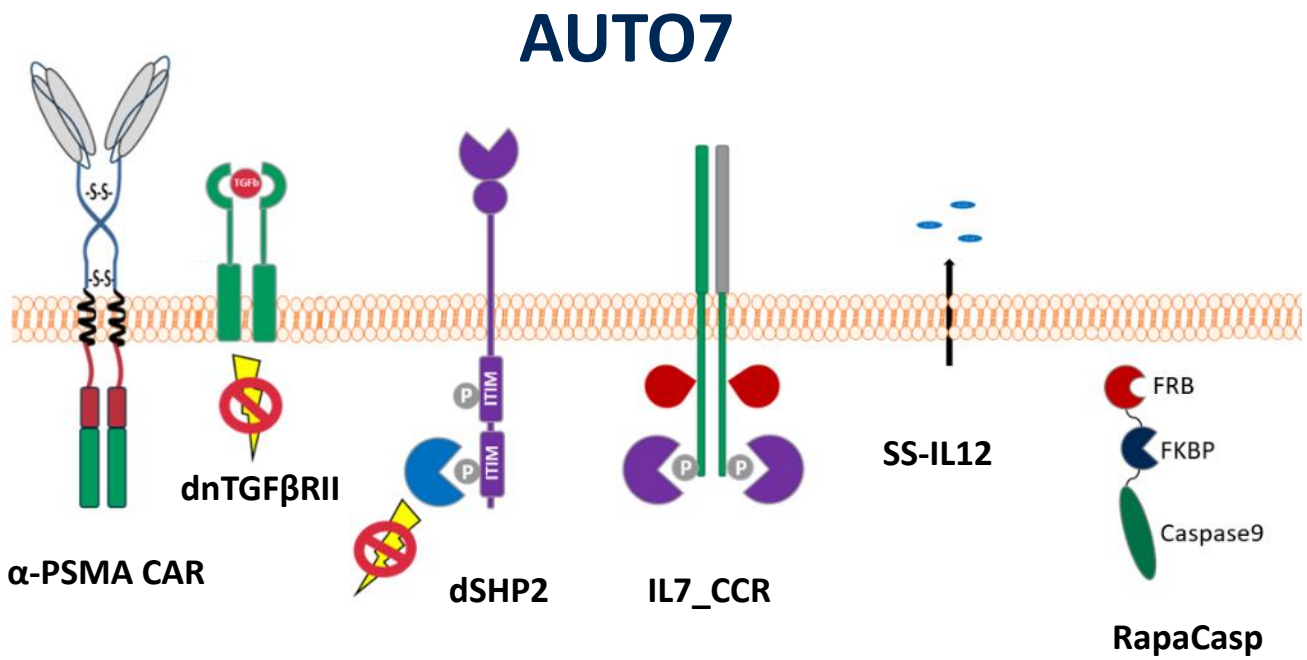




Autolus

AUTO7 demonstrates potent activity against PCa in a xenograft model

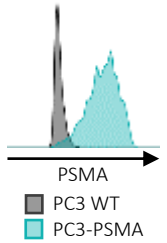
Module	Function
α-PSMA CAR	Specific lysis of mCRPC
RapaCasp	Suicide gene
dnTGFβRII	Shield TGFβ suppression
dSHP2	block checkpoint inhibition
IL7R CCR	Promote CAR T cell persistence
SS-IL12	Host Immune Cell Recruitment

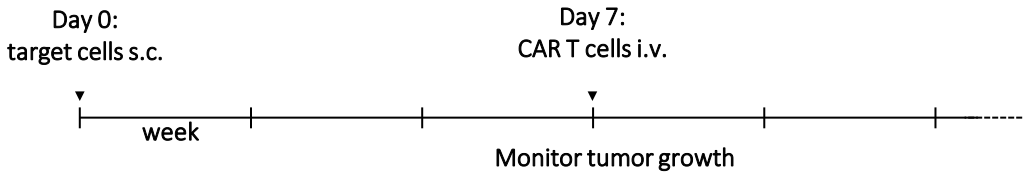


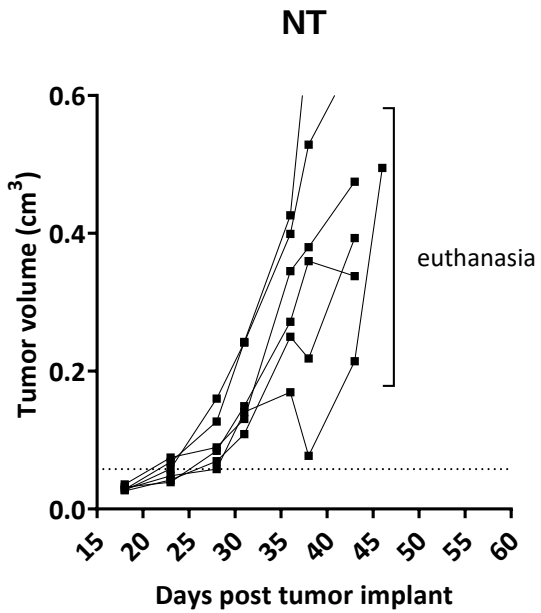
AUTO7 demonstrates potent activity against PCa in a xenograft model

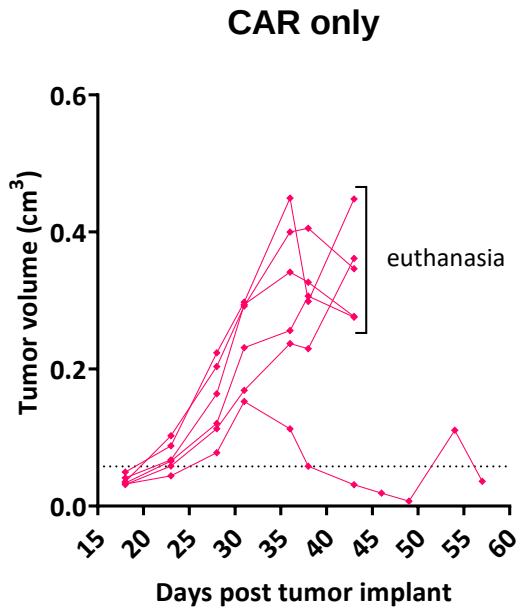
AUTO7 CAR T-cells completely eradicated PSMA positive tumor with no signs of toxicity

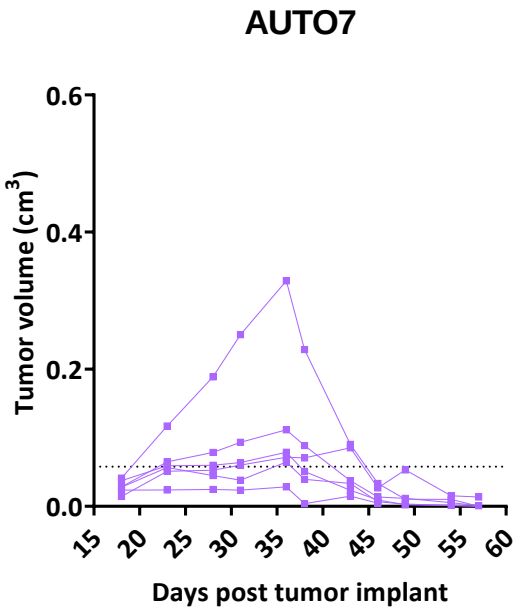
- Human PC3-PSMA prostate cancer model in NSG mouse











Autolus

AUTO7 overcomes immunotherapeutic challenges for mCRPC

Modular CAR T with interchangeable components designed to overcome challenges in mCRPC

PSMA CAR	AUTO7 CAR T cells are highly potent in cytotoxicity assays against high and low PSMA expressing tumor cells
dSHP2	AUTO7 CAR T cells are desensitized to PD-1/PD-L1 mediated immunosuppression
dnTGFβRII	Truncated TGFβRII receptor shields AUTO7 CAR T cells from TGFβ inhibitory effect
Cytokine signal	IL7R CCR module confers AUTO7 CAR T cells cytokine independent proliferation and improved ability to serially kill targets
Immune Adjuvant	SS-IL12 module in AUTO7 CAR T cells maintains potent adjuvant activity with no toxicity

Summary and Next Steps

Dr. Christian Itin

Chairman and CEO

Multiple clinical data points expected through 2020

Product	Indication	Target	Event
B Cell Malignancies			
AUTO1	Adult ALL	CD19	• Ph1 long-term follow up Q2 & Q4 2020 • Ongoing recruitment and dose last patient H1 2021
AUTO1NG	Pediatric ALL	CD19 & 22	• Start Ph1 H2 2020
AUTO3	DLBCL	CD19 & 22	• Decision on Ph2 Q3 2020 • Full Ph1 data H2 2020
AUTO3NG	DLBCL	CD19 & 22	• Ready to start Ph1 H2 2020, life cycle mgmt
Multiple Myeloma			
AUTO8	Multiple Myeloma	BCMA & CAR X	• Start Ph1 study H2 2020
T Cell Lymphoma			
AUTO4	TRBC1+ Peripheral TCL	TRBC1	• Ph1 interim data H1 2021
GD2+ Tumors			
AUTO6NG	Neuroblastoma; Melanoma; Osteosarcoma; SCLC	GD2	• Start Ph1 H1 2021
Allogeneic Approach			
Undisclosed	Undisclosed	Undisclosed	• Start Ph1 Q4 2020

Q&A

Dr. Christian Itin (Chairman and CEO)

Dr. Martin Pule (Founder & SVP, CSO)

Dr. Vijay Reddy (SVP, CMO)

Dr. Muhammad Al-Hajj (SVP, Head of Translational Research)

Dr. Mathieu Ferrari and Dr Marco Della Peruta

Thank you