



2nd **BOLOGNA** **WORKSHOP** on **IMMUNOTHERAPY** **FOR ACUTE MYELOID** **LEUKEMIA**

Bologna, Royal Hotel Carlton
September 29-30, 2026

AML-directed CAR-T cell Therapies: where we stand

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Director of the Munich Center for Cell & Gene Therapy, LMU Medizin

Disclosures

Research Support:

Amgen

Educational Grant:

Gilead/Kite, Roche, Janssen, BMS

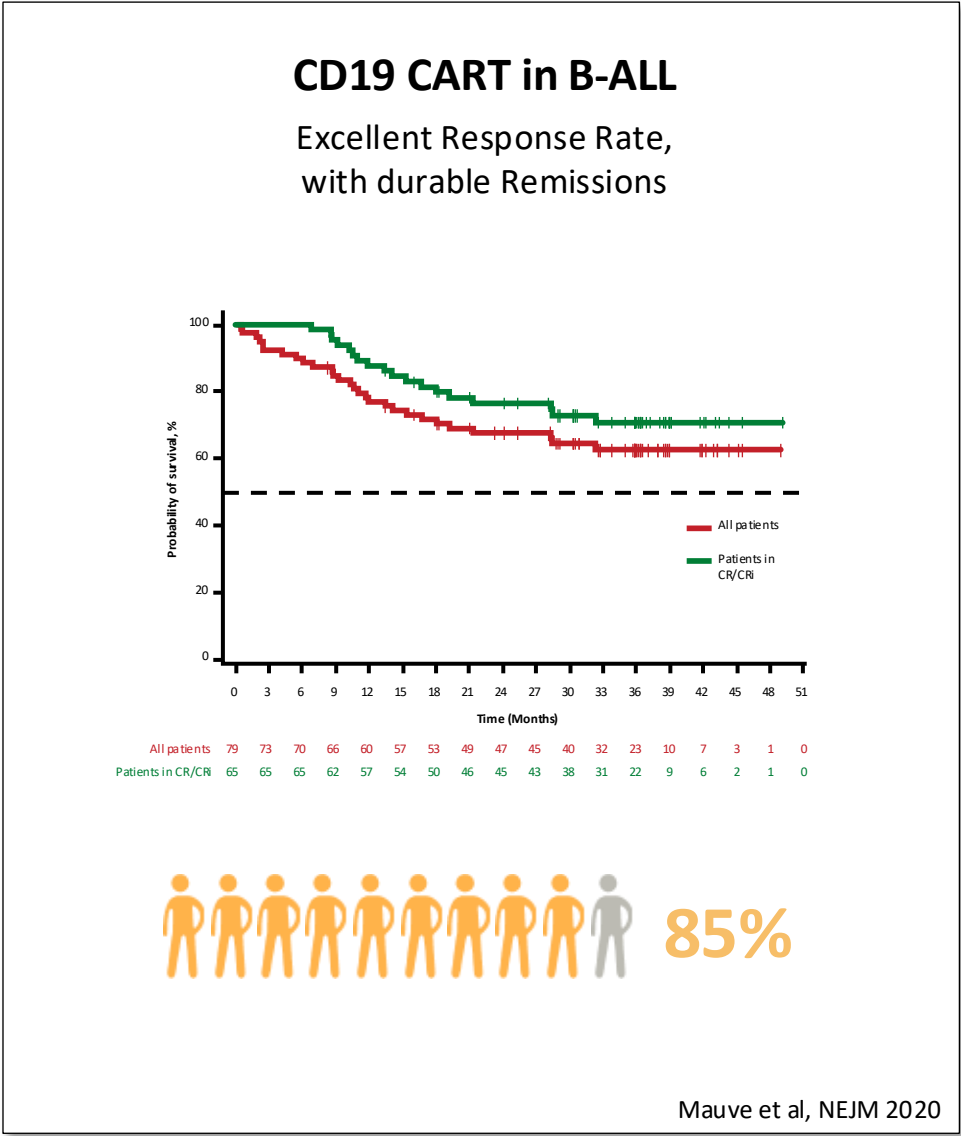
Consulting/ Scientific Advisory Board:

AbbVie, Amgen, Celgene/BMS, Culinan, Gilead/Kite, Janssen , Molecular Partners

Travel Support:

Gilead/Kite, Pierre Fabre, Roche

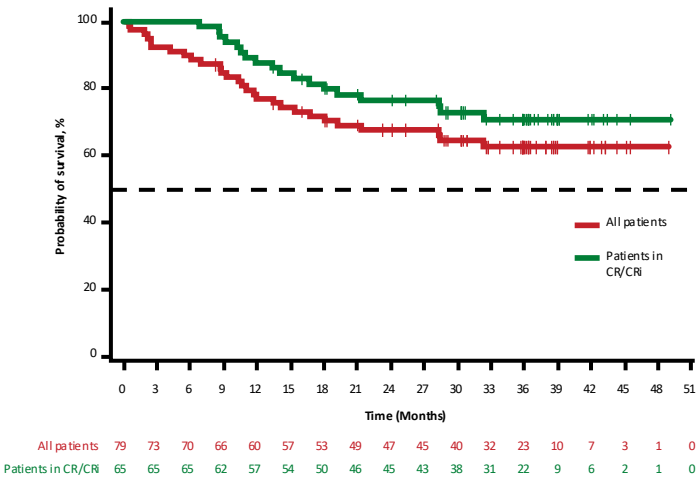
The Success of CD19 directed CART in ALL



The Success of CD19 directed CART in ALL did not translate to AML

CD19 CART in B-ALL

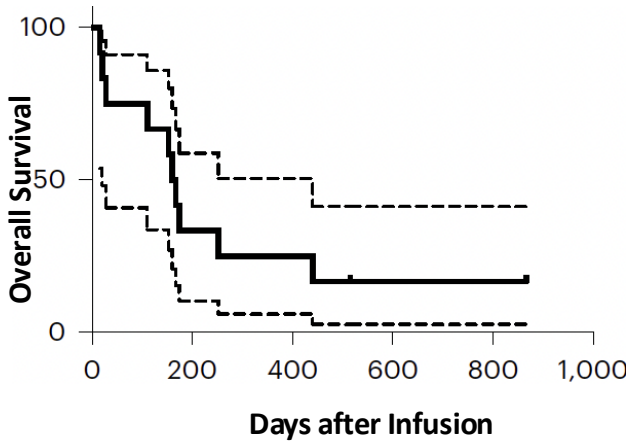
Excellent Response Rate,
with durable Remissions



Mauve et al, NEJM 2020

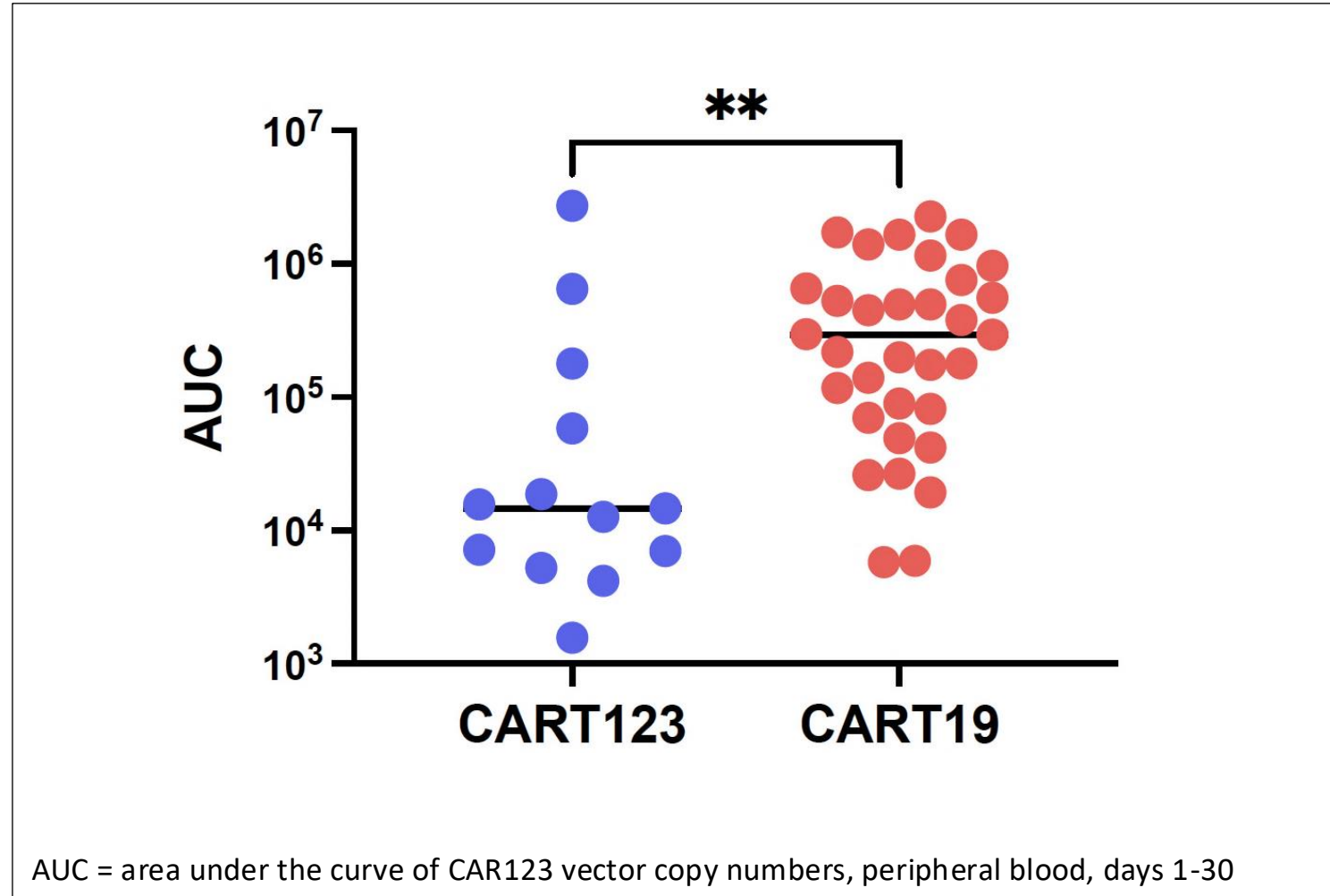
CD123 CART in AML

4 of 12 patients with CRs,
transient Remissions



Bhagwat et al, Saar Gill, Nature Medicine 2024

Reflected in poor CART T cell expansion in AML vs ALL



Results of Selected Early Clinical Trials directed against Myeloid Antigens

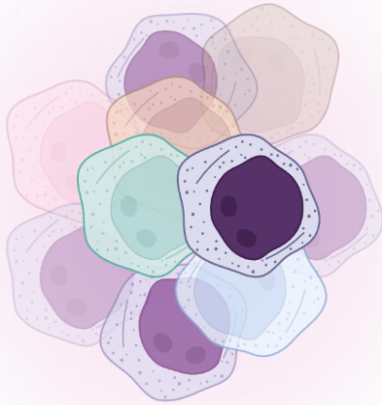
Proof-of-Concept shown with response in about 30% of the pts, but expected Hematotoxicity, hence often consolidated with allo SCT – thereby also eliminating all adoptively transferred CART Cells

	Tambaro et al. (2021)	Shah et al. (2023)	Naik et al. (2022)	Jin et al. (2022)	Bhagwat et al. (2024)
Target	CD33	CD33	CD123	CLL1	CD123
Co-stim	4-1BB	CD28	CD28	4-1BB	4-1BB
Screened		31	80		22
Enrolled	10	24	25		20
Infused	3	19	12	10	12
Response	0	2/5 at DL4 only	2/7 CR; 3/7 ORR	CR/CRI 7/10	3/12 CR
Toxicity Grade ≥3	CRS, hepatic Dysfunction, TLS G3 Sepsis G4	CRS G3/4 21% ICANS G3 SOS G3	No DLT Toxicity with dasa	CRS G3-5 60% Neutropenia G4 80% Infection G5 20%	CRS G3 8% CRS G5 2 17%

Mechanisms of CART Cell Resistance in AML

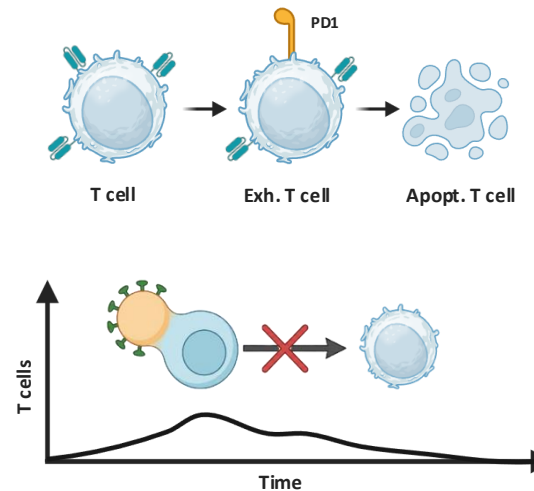
AML resistance

Myeloid Lineage Antigens with
on-target/off-leukemia toxicity
Antigen heterogeneity
Antigen sink
Interaction with MDSC / Monos
Genetics, Surfactome,
Secretome



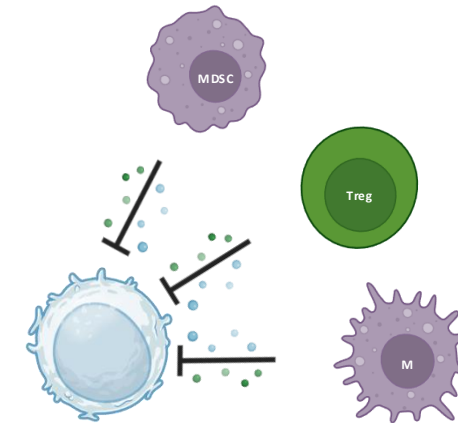
(CAR)-T Dysfunction

Impaired Cytotoxicity
Proliferative failure
T-cell exhaustion
Low persistence



Immunosuppression

Suppressive cytokines
Checkpoint ligands
Myeloid inhibition
Regulatory T cells



Strategies to improve Efficacy of CART in AML

AML Targeting:

- Adapter CART cells with on/off targeting modules
- Stem cell edited allo grafts followed by immunotherapy, e.g. CART
- TCR-tg T cells / endogenous T cells targeting intracellular antigens

Improve T Cell Fitness:

- CART + CCR
- CART pharmacy
- CART + switch receptors

Combine with Standard of Care or other Immunomodulators

- VEN/AZA
- Menin Inhibitors
- Innate Immunity

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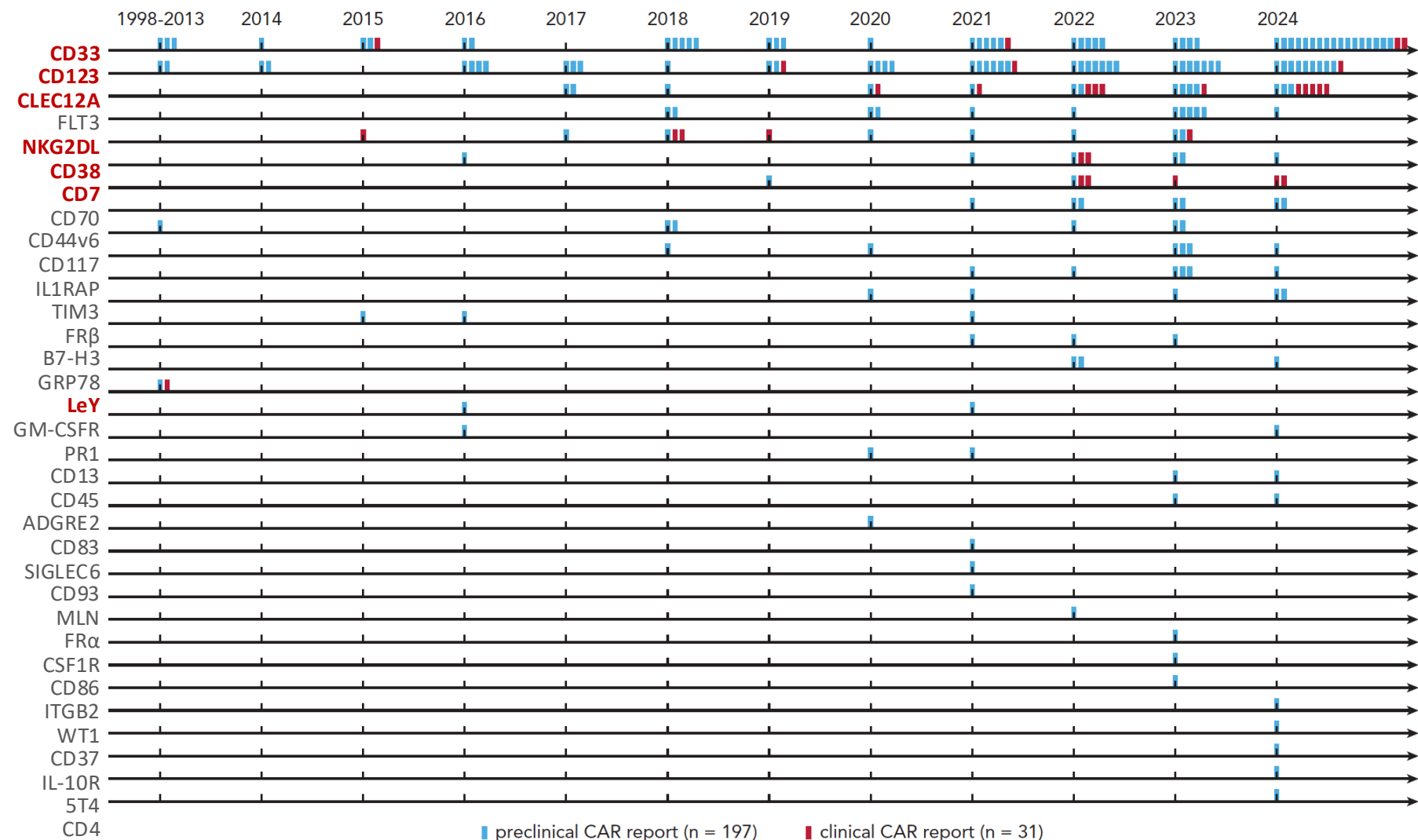
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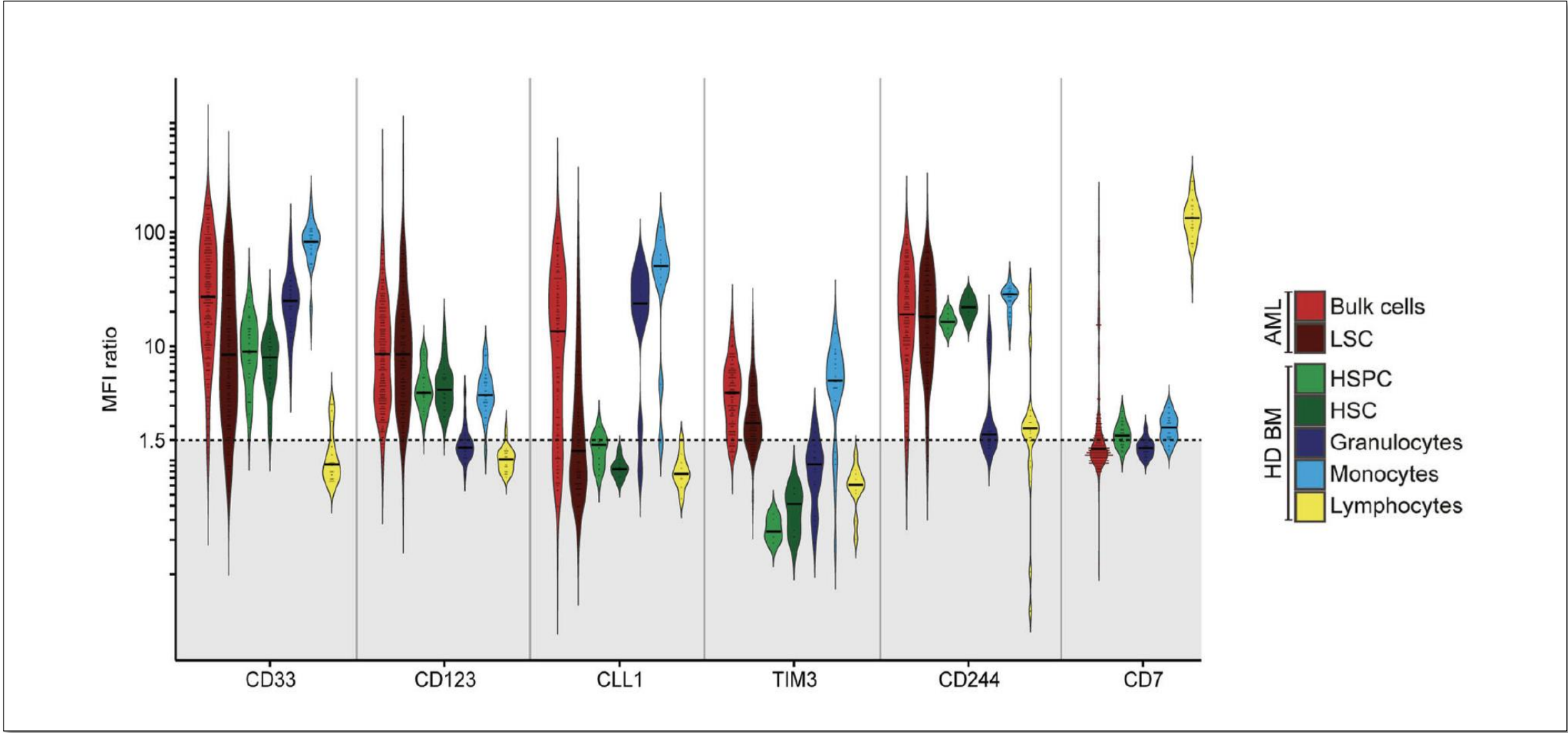
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Time Line of preclinical and clinical Reports on CART in AML



Small Therapeutic Window for Myeloid Lineage Antigens



Adapter CAR T Cells – a “universal” Tool to target multiple TAA in AML

PROs

Multiple & sequential targeting

ON/OFF-switch

Intermittent resting phase
(counteracting T-cell exhaustion)

Works with a multitude of different
adapter molecules (AMs)

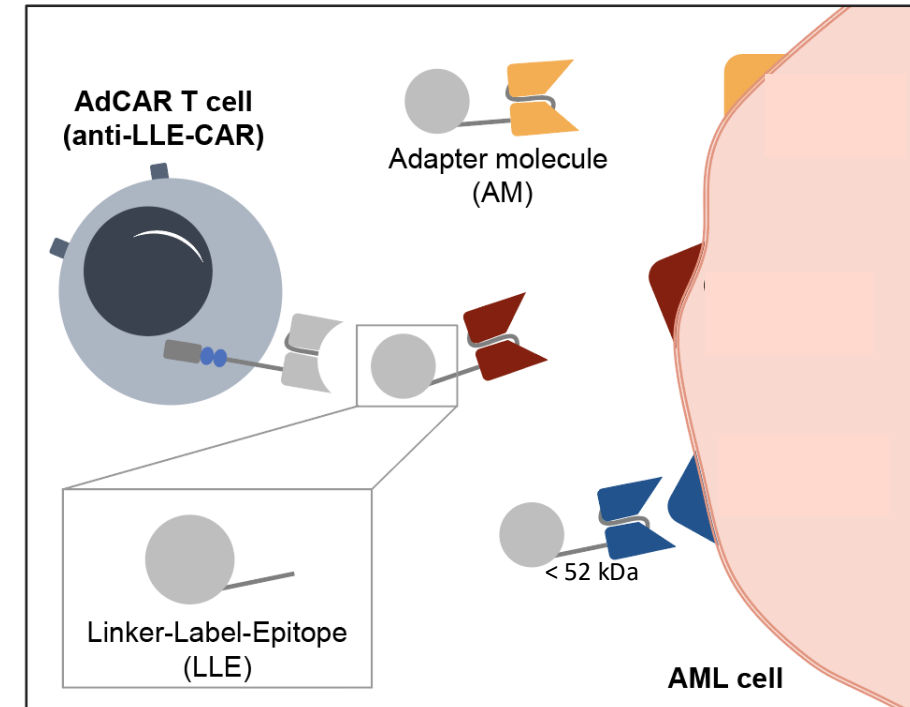
Universal platform across tumor entities

CONs

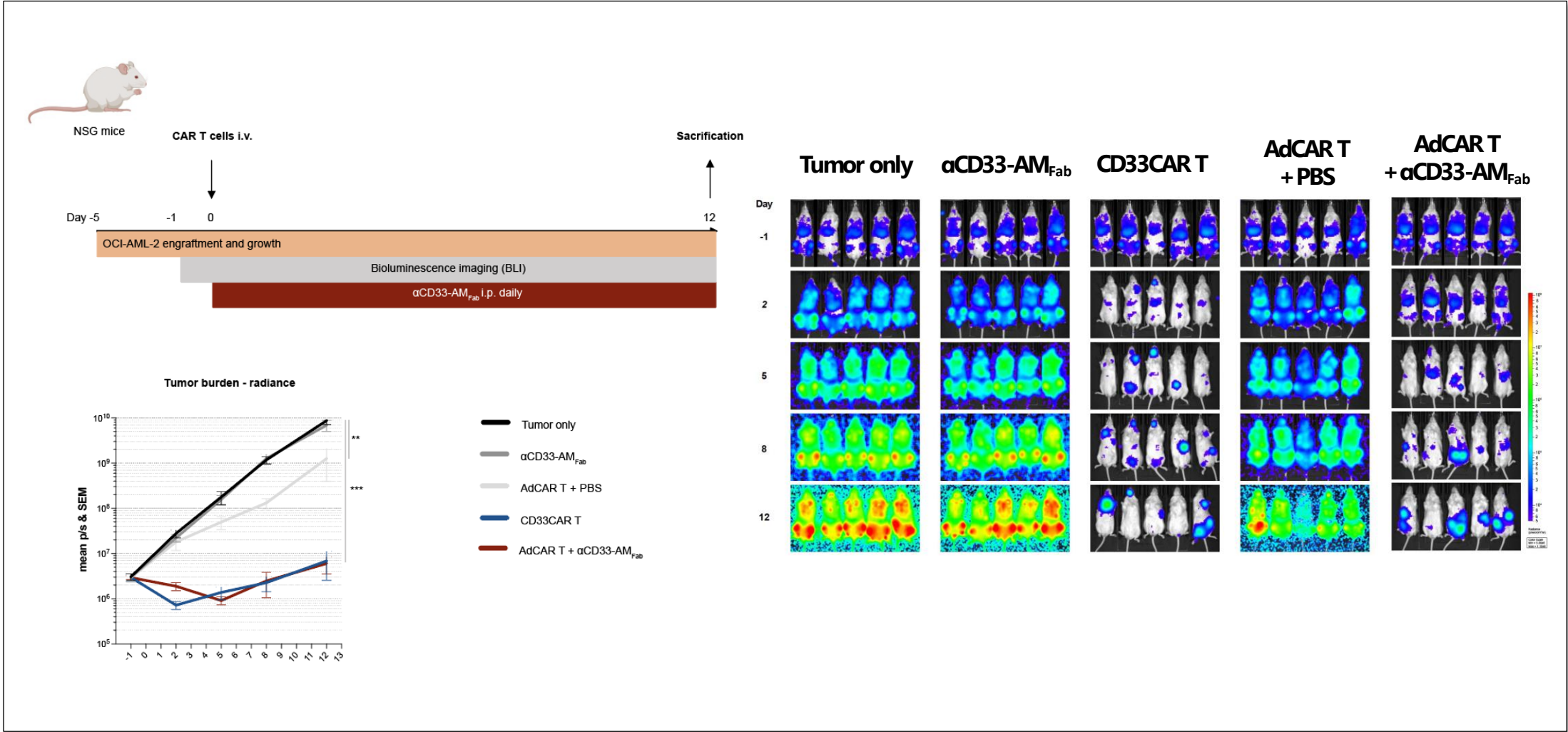
Potential antigen sink

Need for continuous infusion

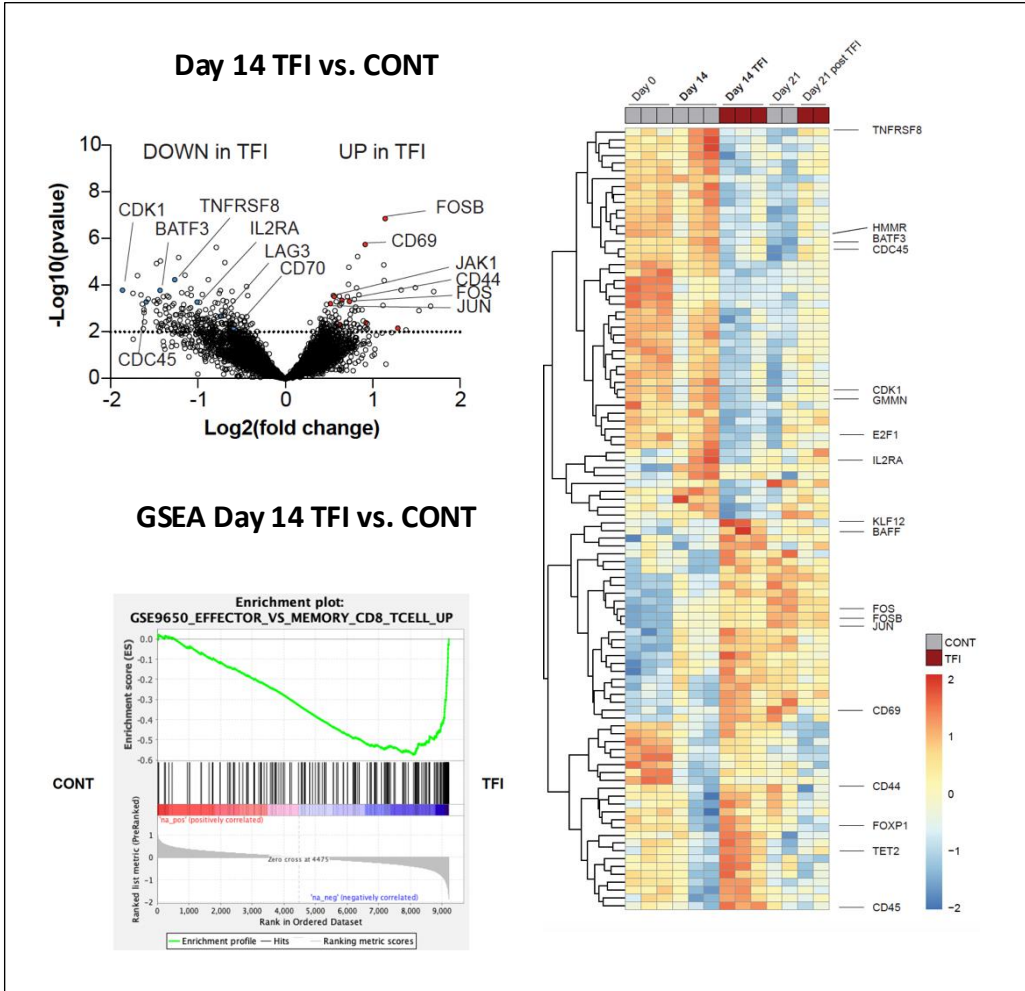
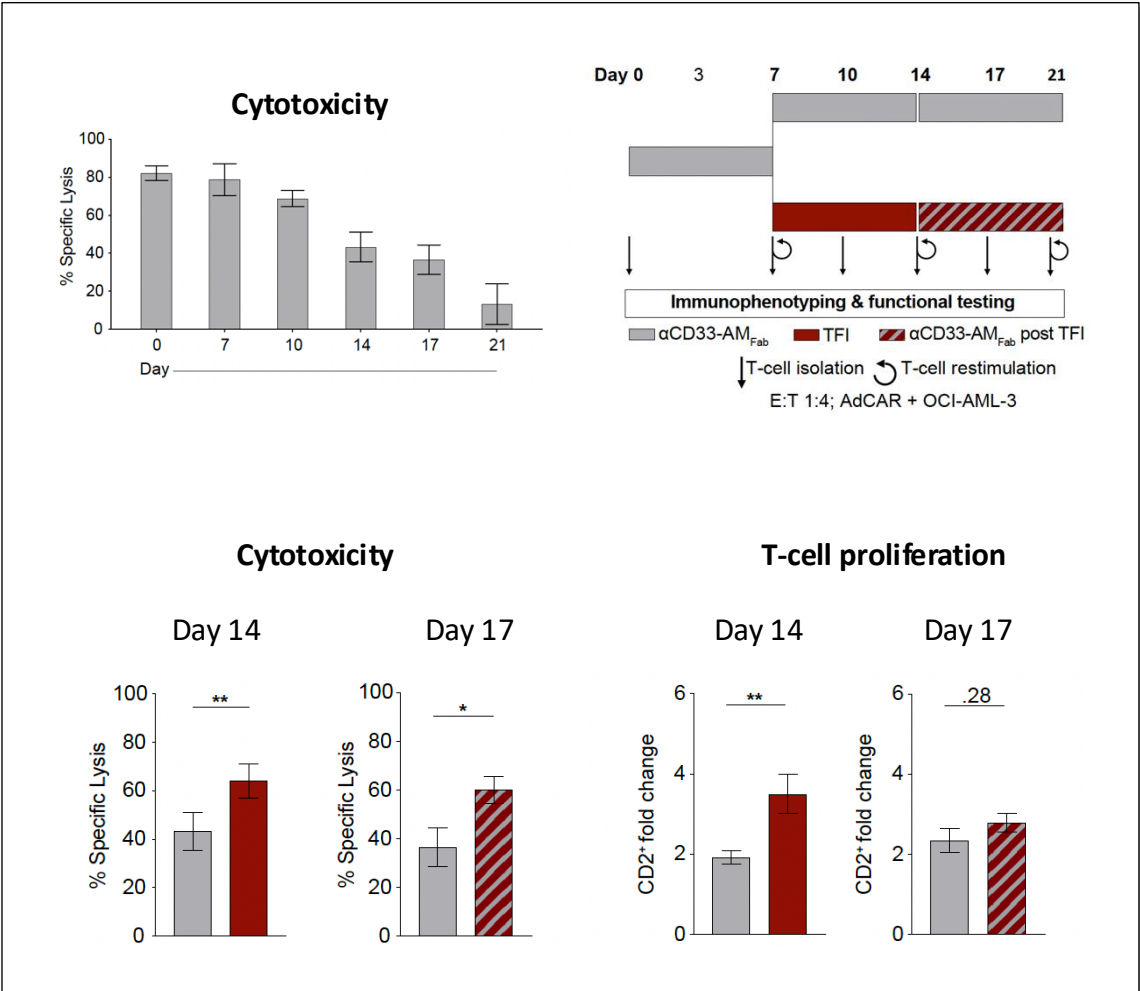
Efficacy compared conventional CART unknown



Mouse Model: Similar Activity of Adapter CD33 CART vs conventional CD33 CART

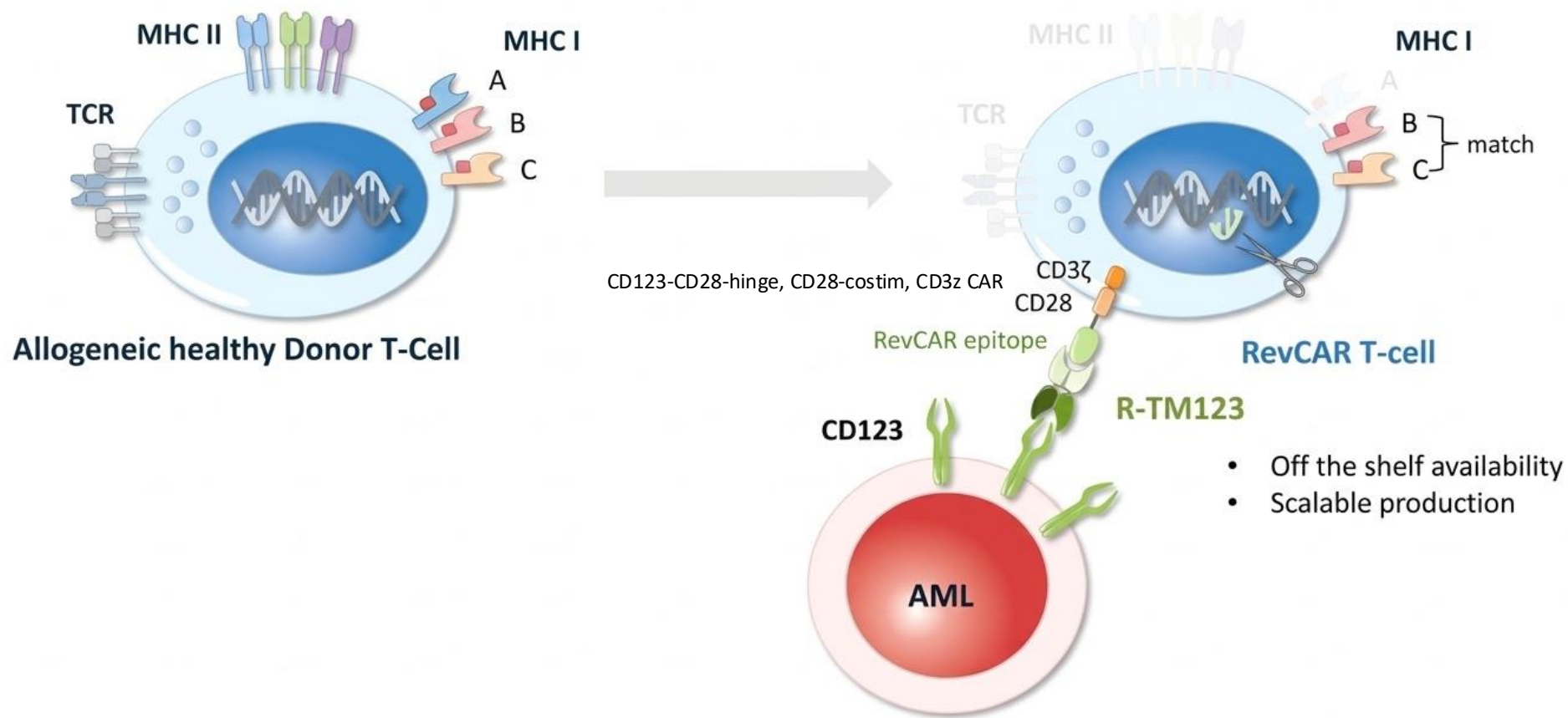


Treatment-free Intervals prolong AdCAR T-cell Function and lead to transcriptional Reprograming thereby counteracting T Cell Exhaustion



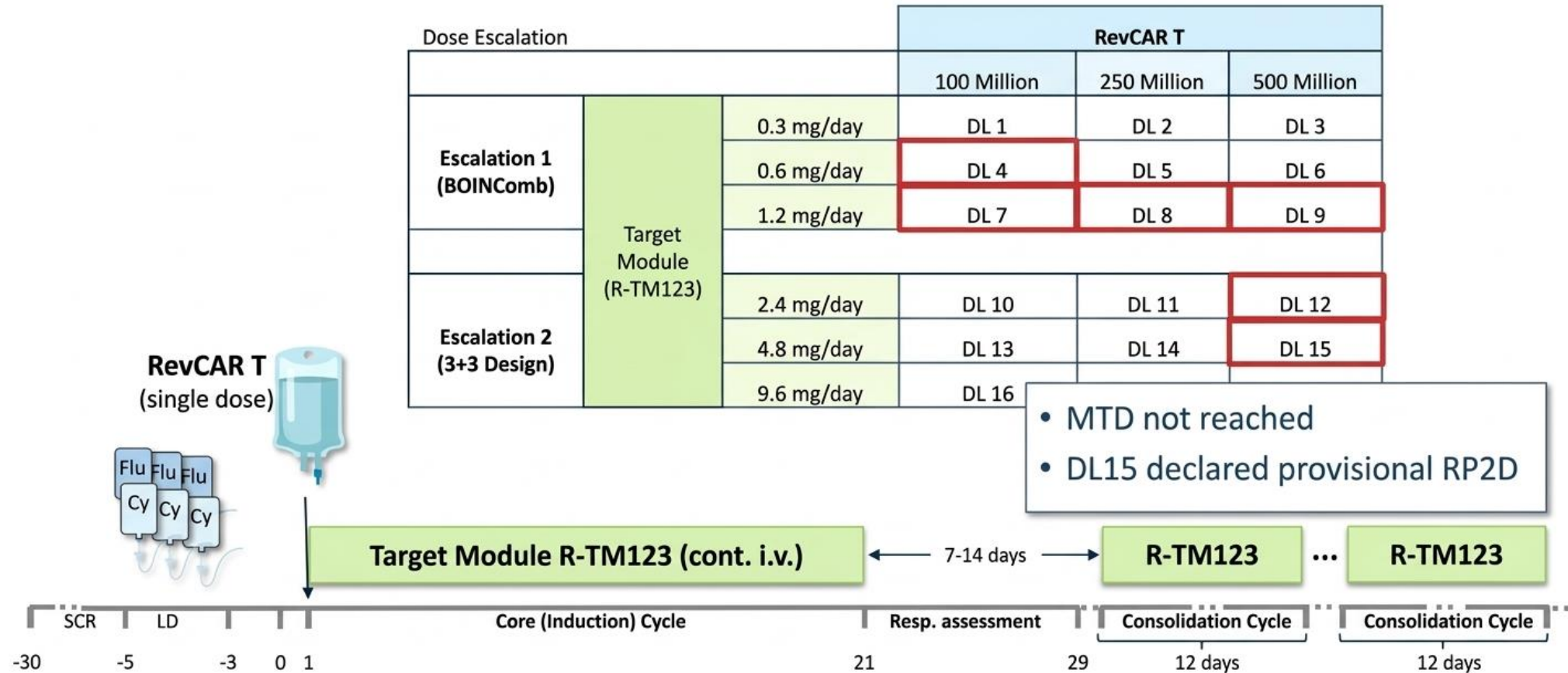
Phase I trial (AVC-201-01) using CD123-directed allogeneic CART in R/R AML

First-in-class switchable allogeneic CART therapy for CD123+ AML: Results from the phase Ia RevStAR-123 (AVD-201-01) Study, Allogeneic T cells with knockout of HLA-A, HLA-class II, TCR; HLA-B,C kept to prevent NK cell attack



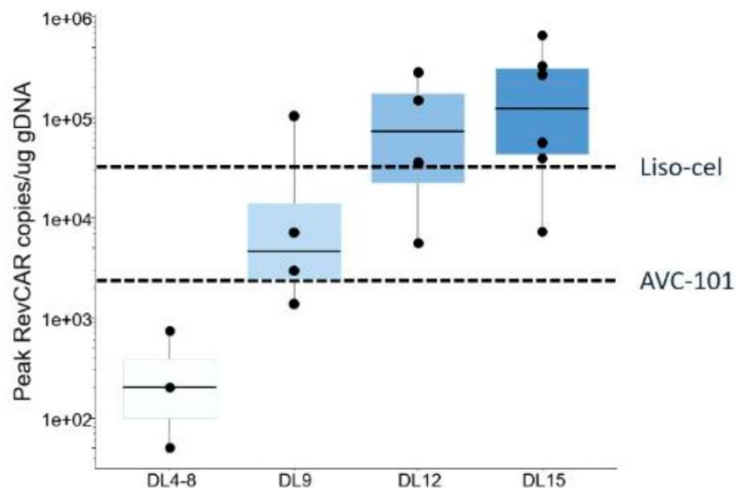
Study Design and Treatment Scheme for R/R CD123+ AML

Inclusion criteria: CD123 expression on > 20% of AML cells, HLA-type matched for one allele of HLA B and C

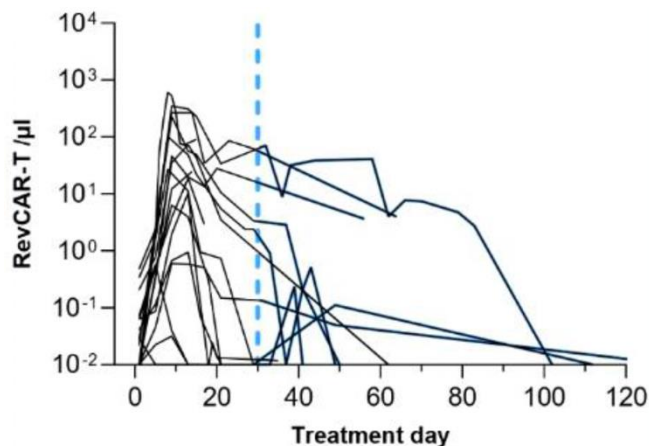


SCR – screening, LD – Lymphodepletion, Flu – Fludarabine (30 mg/m²/d), Cy – Cyclophosphamide (first 4 patients: 300 mg/m²/d, thereafter 600 mg/m²/d), Phase Ia was completed in April 2026 with 17 pts treated with the induction cycle and 7 completing at least one consolidation cycle.

RevCART Peak Expansion & Persistence: shown are 4 different Dose Levels

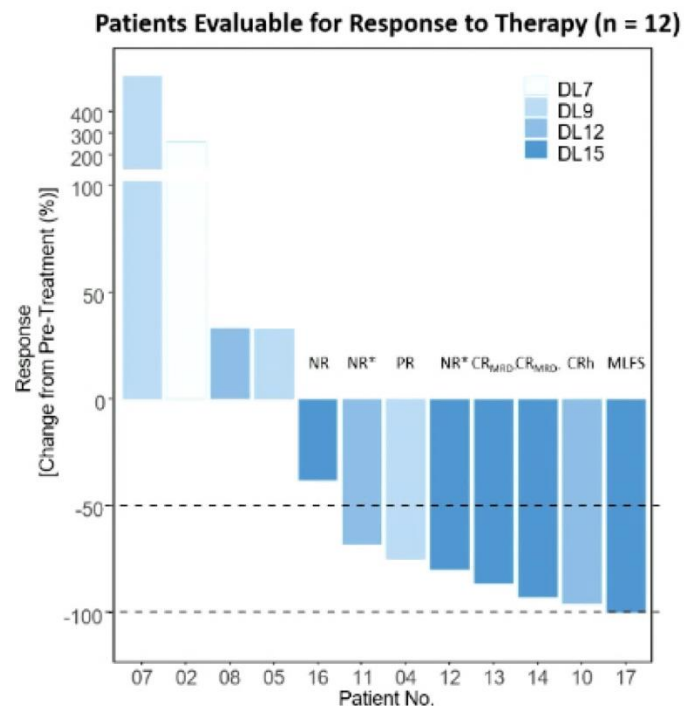


- Increase in peak expansion with increasing dose
- DL15 median peak expansion: 161,835 cps/ μ g gDNA (range 7,240 – 659,070)
- Exceeds AvenCell's autologous median CD123 CAR-T peak expansion
- DL12 and DL15 median expansion exceeds median expansion of Liso-Cel in r/r DLBCL

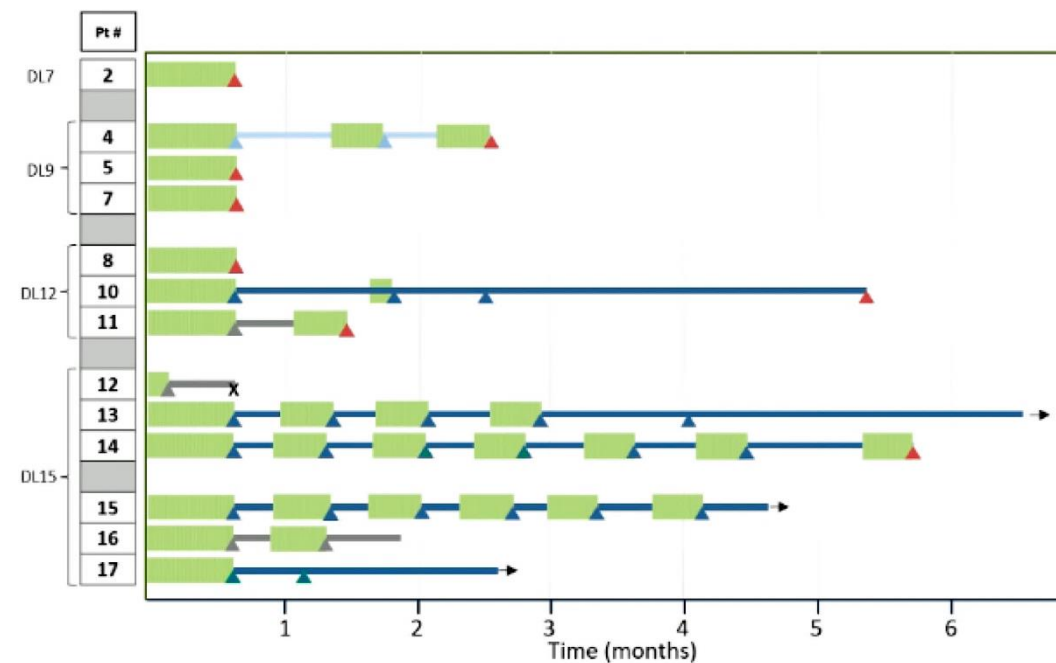


- Median persistence of **all patients** with at least one evaluable data point at any time on study is 43 days (range: 0 - 112; IQR: 18 – 91)
 - 8/17 pts (47%) had persistence of >30 days
- Median persistence of all patients at the **two highest Dose Levels** (DL12+DL15, excluding early drop-outs) was 42 days.
 - 5/8 pts (63%) had persistence of >30 days

Prolonged clinical Responses observed at the higher Dose Levels



- Overall, 4 responses in 12 evaluable patients
- At the **two highest dose levels** (DL12+15, n=8 evaluable patients):
 - 3/8 CR/CRh, including 2 CRMRD-
 - 2/3 MRD negative
 - 1/8 MLFS



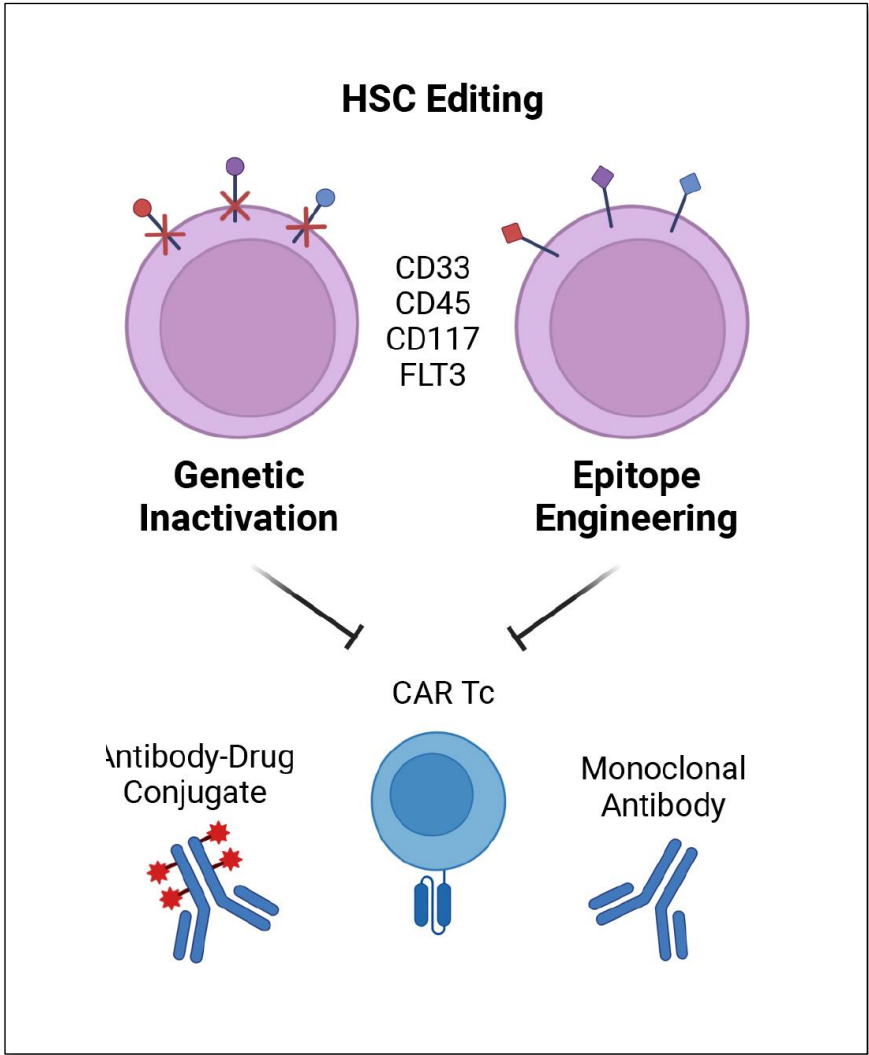
Response Type:

- ▲ CR / CRi / CRh / MLFS / MRD Conv.
- ▲ Partial Response
- ▲ Blast Reduction
- ▲ PD (Progressive Disease)
- X Patient Death
- Continuing Response/Tx

Target Module Infusion Status:

- R-TM123 infusion

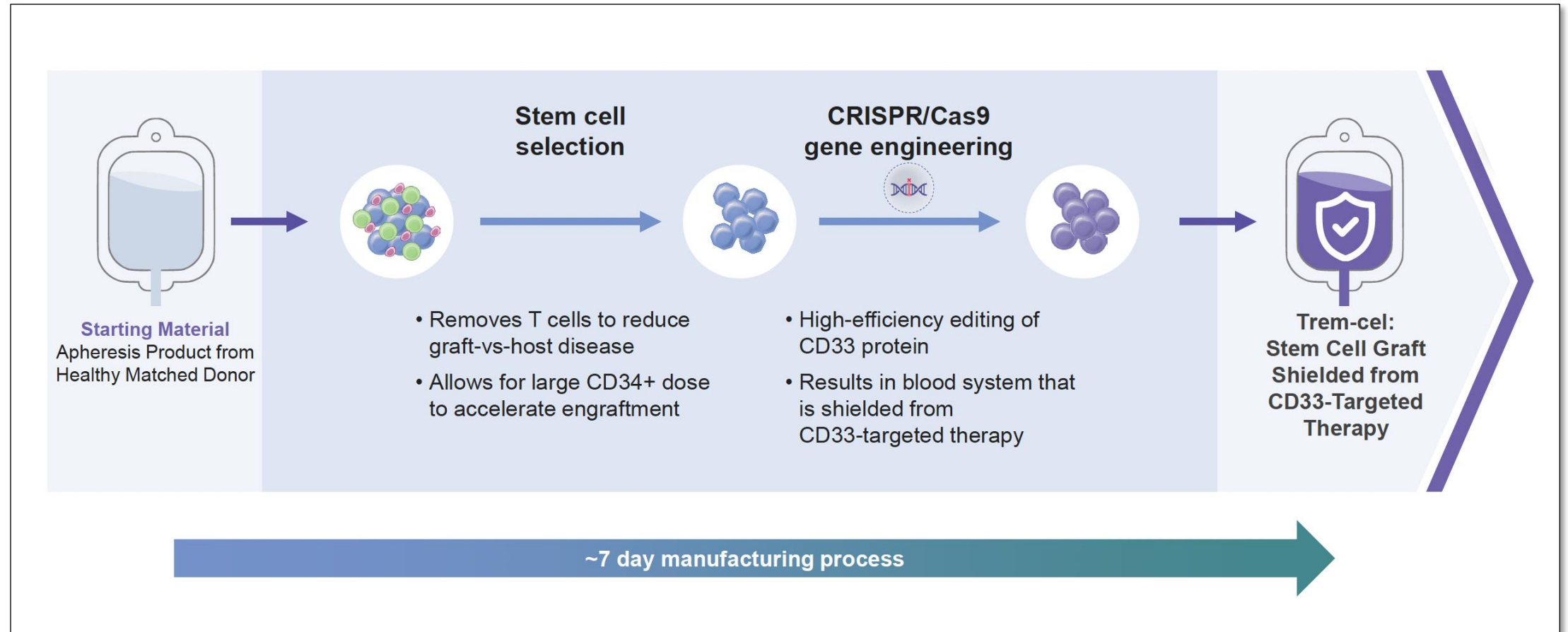
HSC Editing to make the HSC „invisible“ to the CART: Allo SCT followed by CART



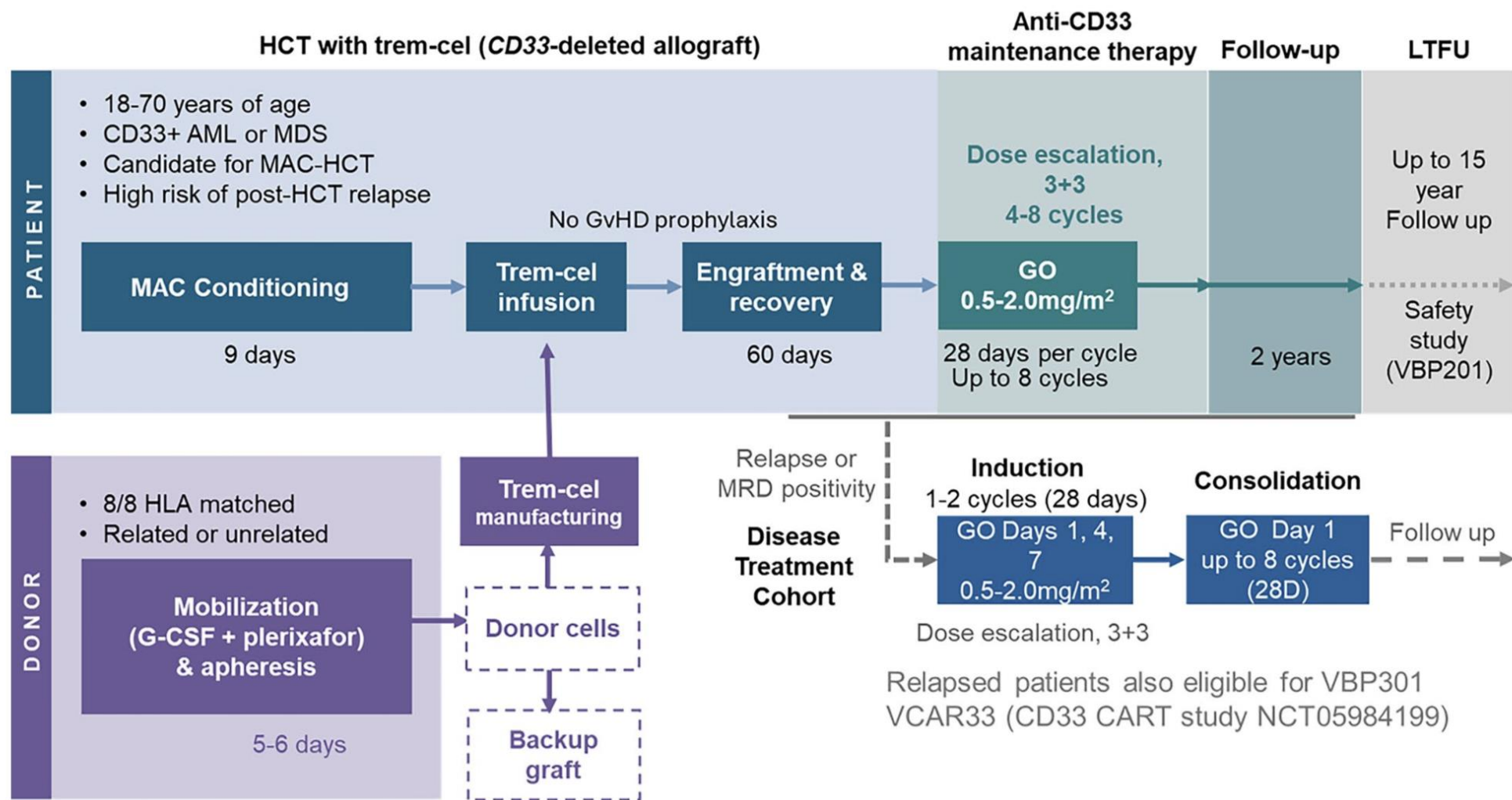
Concept	Edited SCT followed by targeted immunotx
Sequence	Target-KO or Epitope edited SCT => CAR T, CAR-NK, ADCs, TCE, moAb
Target Antigen	CD33, CD45, CD123, FLT-3, KIT
Hematopoietic Toxicity	None; Edited hematopoiesis invisible to CAR
CAR	2 nd /3 rd generation CART, Dual / Multiple CART
Cell Source	Stem cell donor
Potential	Increase safety by decreasing on-target-off-leukemia toxicity; multiple-targeting possible to overcome antigen escape
References	Kim et al, Cell 2018; Borot F. et al, PNAS 2019; Wellhausen et al, STM 2023; Humbert et al, Leukemia 2019, Chiesa et al, NEJM 2023; Casirati et al, Nature 2023; Marone et al, J Exp Med 2023; Reviews: Kim, Trends in Cancer 2023; Saniei et al, Cell Stem Cell 2023, Volta et al, J Exp Med 2023

Generation of biallelic CRISPR/Cas9 edited Stem Cells for CD33-targeted Therapy

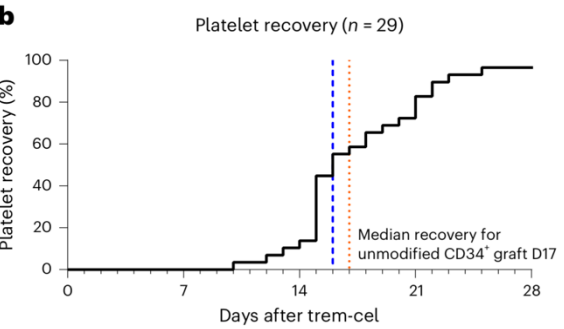
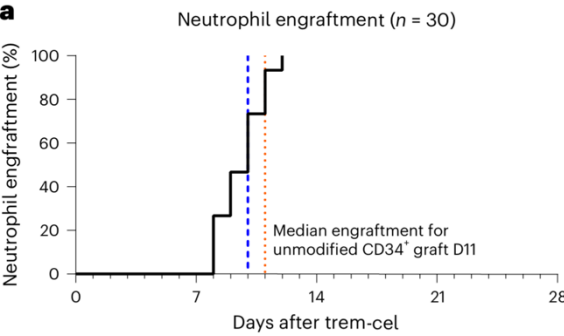
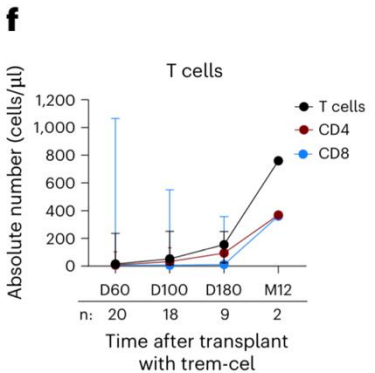
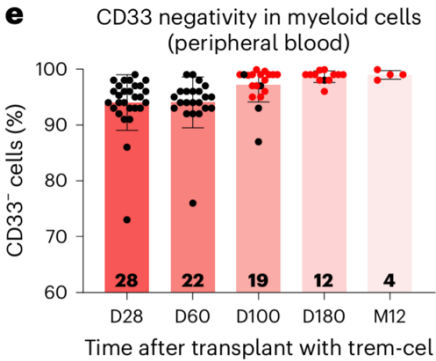
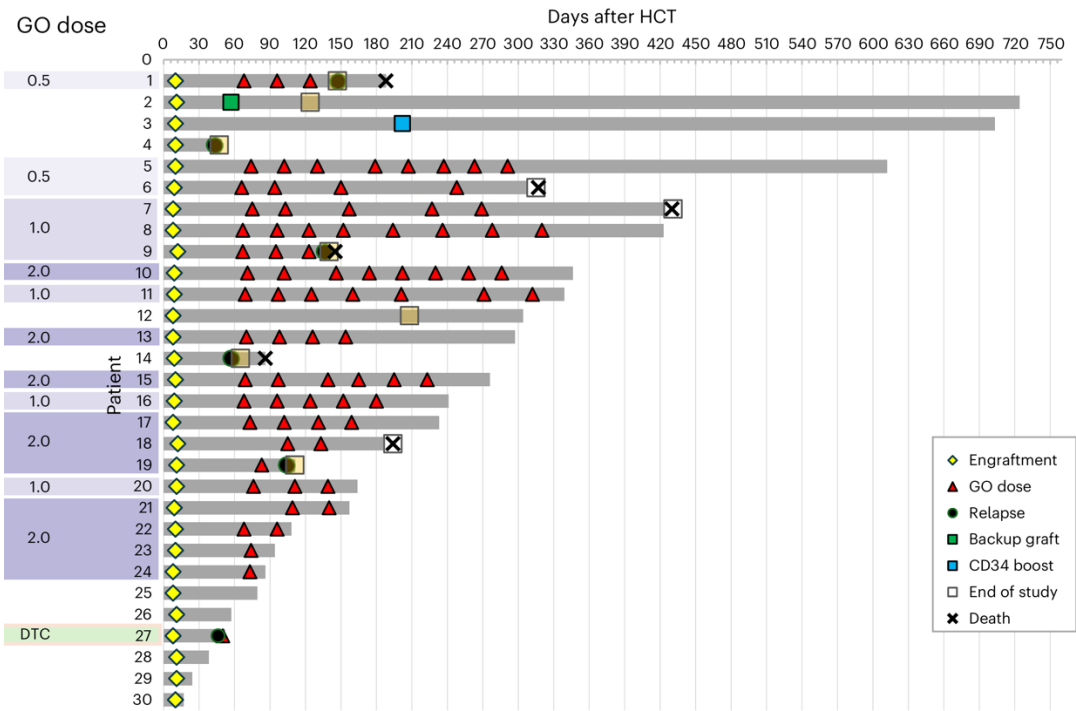
Phase I/II trial: G-CSF/Plerixafor-mobilized peripheral SCT; CRISPR/Cas9 Stem cell editing of CD33 Protein; Myeloablative Conditioning with Busulfan or TBI with ATG



VBP101 Study Design



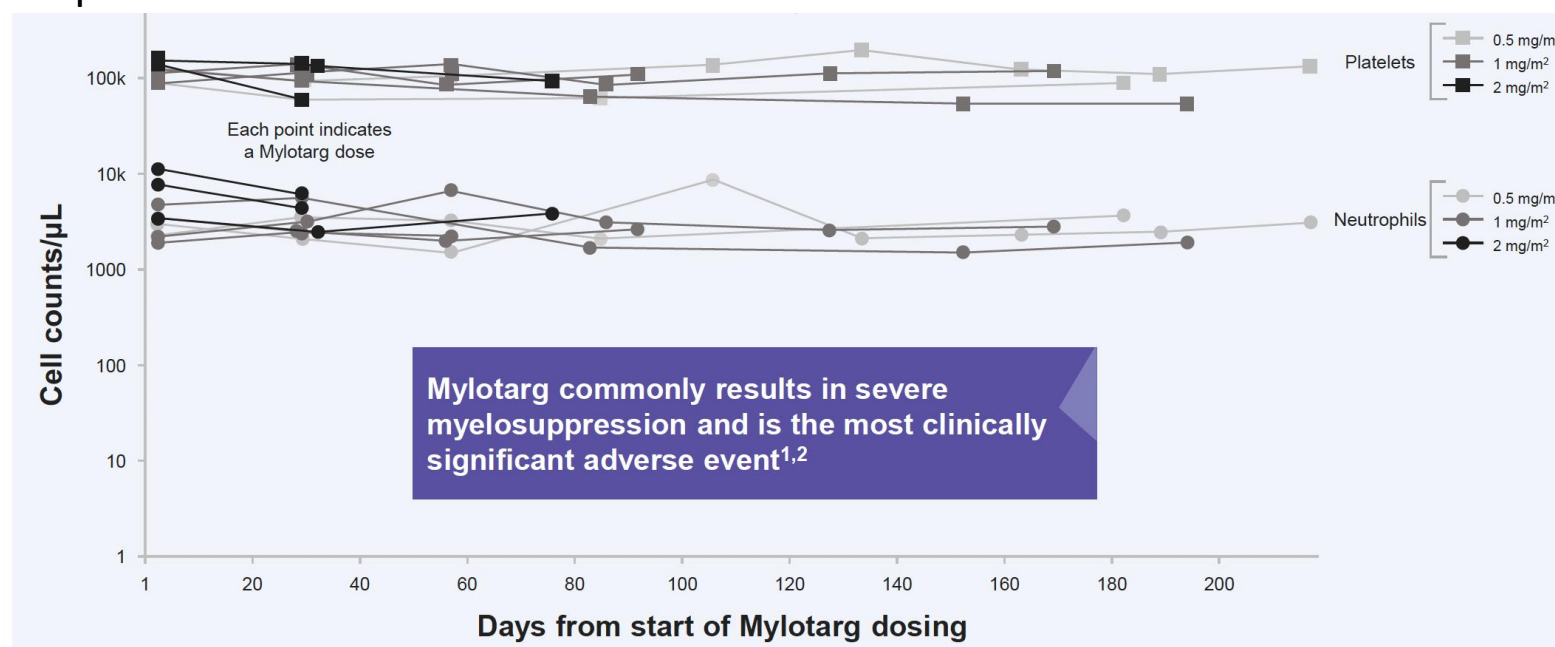
Engraftment of edited Stem Cells in all pts; Neutrophil Engraftment in all pts (primary endpoint) until Day 28, median Engraftment Day 10



No prolonged, high grade Neutropenia after GO observed

Initiation of a CD33 directed Antibody-drug-conjugate (ADC) at Day 60 post HCT;
Schedule: every 28 Days for 4-8 Cycles

19 pts treated

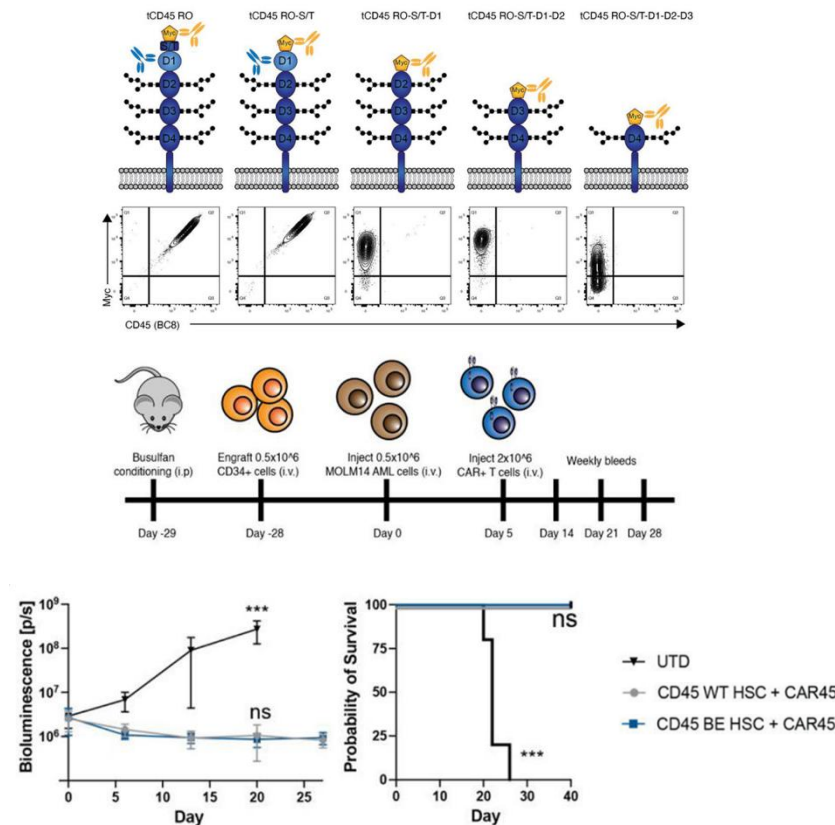


PROOF-of-CONCEPT as required by FDA (GO first, CART second)

Mono- and Multiple Epitope Editing of the SCT Graft followed by CART Transfusion

Epitope Editing through Integration of non-synonymous Mutation makes HSCP invisible to CART

CRISPR-based Editing of CD45 in HSPCs AND CD45 directed CART

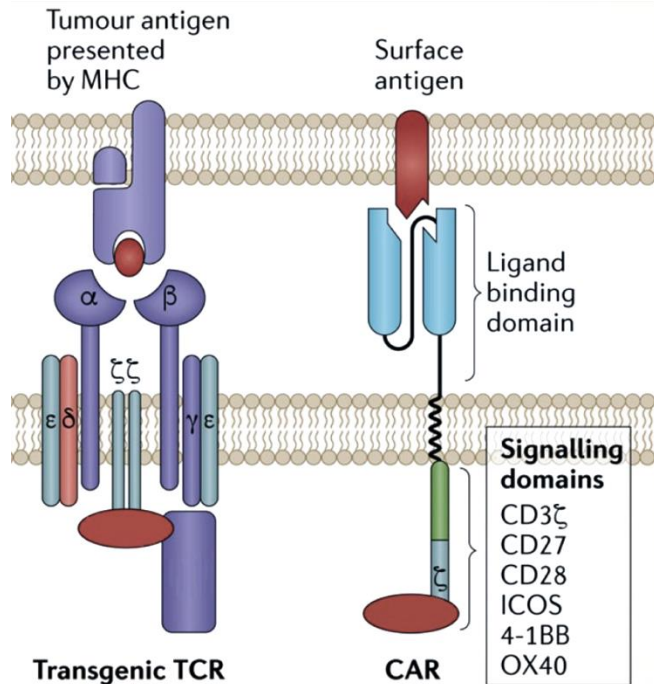


Wellhausen et al, STM 2023

Safety concerns

- Elimination of HSC if base editing **is not complete** and target antigen is still expressed at low levels (biallelic required)
- **Functional Impairment** and dysfunction of the edited HSC population, short or long term
- Generation of **immunogenic neopeptides** that could elicit a human or cellular immune response

Targeting intracellular Antigens presented by defined HLA Molecules



Garber K, Nature Biotechnology 2018

Identifier	Leukemia	Phase	Outcome measures	Status	Locations
NCT02550535	Autologous WT1 TCR-T cells ■ Myelodysplastic syndromes; ■ Acute myeloid leukaemia	Phase 1 Phase 2	■ Safety following gene-modified WT1 TCR T-cell therapy as measured by suspected unexpected serious adverse reactions (SUSARs); ■ Proportion of subjects achieving 1 or more IWG response criteria following gene-modified WT1 TCR T-cell therapy; ■ Safety and tolerability of gene-modified WT1 TCR therapy as measured by clinical laboratory parameters and adverse events. ■ Among 10 patients (6 AML, 3 MDS, and 1 TKI-resistant CML) enrolled in the study, All 6 AML patients survived, at last, follow-up (median 12 months) and median 3 months in the 3 patients with MDS. 3 deaths: 2 from disease progression and 1 from other causes.	Completed	■ AZ St. Jan Brugge-Oostende AV Brugge, Belgium ■ UZ Leuven Leuven, Belgium ■ Uniklinikum Dresden, Germany
UMIN0001159	Autologous WT1 siTCR-T cells ■ Acute myeloid leukemia; ■ Myelodysplastic syndromes	Unknown	■ No adverse events of normal tissue were seen. ■ 2 patients showed transient decreases in blast counts in bone marrow, which was associated with recovery of hematopoiesis.	Completed	■ Mie University Hospital, Japan ■ Ehime University Hospital, Japan ■ Fujita Health University Hospital, Japan ■ Nagoya University Hospital, Japan
NCT01621724	Autologous WT1 TCR-T cells ■ Acute myeloid leukemia; ■ Chronic myeloid leukemia	Phase 1 Phase 2	■ Identify organ toxicities and other side effects ■ Transduction efficiency and TCR expression on TCR-transduced cells ■ WT1-specific immune responses of TCR-transduced T cells	Completed	■ University Hospitals Bristol NHS Foundation Trust Bristol, UK ■ University College London Hospitals NHS Trust London, UK, NW1 2PG
NCT01640301	Allogeneic WT1 TCR-T cells ■ Recurrent adult acute myeloid leukemia; ■ Recurrent childhood acute myeloid leukemia; ■ Secondary acute myeloid leukemia	Phase 1 Phase 2	■ Antileukemic potential efficacy, in terms of duration of response (Arm I). ■ Efficacy, in terms of relapse rate (Arm I). ■ Incidence of chronic graft versus host disease (GVHD) (Arm I).	Active, not recruiting	■ Fred Hutchinson University of Washington Cancer Consortium Seattle, Washington, USA
NCT04284228	Allogeneic WT1/PRAME/Cyclin A1-antigen-specific CD8* T cells (NEXI-001 T-cell product) ■ Acute myeloid leukemia; ■ Myelodysplastic syndrome	Phase 1 Phase 2	■ Adverse events of special interest (AESIs) events of dose-limiting toxicities (DLTs) ■ AESI events of infusion-related reactions and cytokine release syndrome (CRS) ■ Survival, including median progressive-free survival (PFS), overall response rate (ORR), overall survival (OS).	Recruiting	■ City of Hope Comprehensive Cancer Center Duarte, California, USA ■ Advent Health Medical Group Blood & Marrow Transplant Orlando, Florida, USA ■ Karmanos Cancer Institute Detroit, Michigan, United States ■ University Hospital Dresden, Dresden, Germany ■ University Hospital Erlangen, Erlangen, Germany ■ University Hospital Frankfurt, Frankfurt, Germany
NCT03503968	Autologous PRAME TCR-T cells (MDG1011 cell product) ■ High-risk myeloid; lymphoid neoplasms (including relapse AML after allo-HSCT)	Phase 1 Phase 2	■ Adverse events and dose limiting toxicities (safety and tolerability). ■ Maximum tolerated dose (MTD) and/or recommended phase II dose (RP2D) of MDG101. ■ For feasibility: percent of all subjects who receive the planned target dose of MDG101.	Recruiting	■ University Hospital Erlangen, Erlangen, Germany ■ University Hospital Frankfurt, Frankfurt, Germany
NCT03326921	Allogeneic HA-1 TCR-T cells ■ Juvenile myelomonocytic leukemia ■ Recurrent acute biphenotypic leukemia ■ Recurrent acute undifferentiated leukemia	Phase 1	■ Feasibility of manufacturing minor H antigen (HA-1) T-cell receptor (TCR) CD8+ and CD4+ T cells. ■ Feasibility of administering minor H antigen (HA-1) T-cell receptor (TCR) CD8+ and CD4+ T cells. ■ Incidence of dose-limiting toxicities of HA-1 T-cell receptor (TCR) T cells.	Recruiting	■ Fred Hutchinson University of Washington Cancer Consortium Seattle, Washington, United States
NCT04464889	Autologous HA-1 H TCR-T cells ■ Acute myeloid leukemia ■ Myelodysplastic syndromes	Phase 1	■ Safety and tolerability of HA-1H TCR-transduced T cells: incidence and severity of adverse events. ■ Maximum tolerated dose (MTD) of HA-1H TCR-transduced T cells. ■ Recommended phase 2 doses (RP2D) of HA-1H TCR-transduced T cells.	Active, not recruiting	■ Leiden University Medical Centre Leiden, Zuid Holland, Netherlands

Kang et al, Front Oncol 2022

Targeting AML-specific Mutations

NPM1mutated-specific, TCR tg T cells

(van der Lee et al., *Journal of Clinical Investigation* 2019)
Clinical trial is currently recruiting.

JCI

The Journal of Clinical Investigation

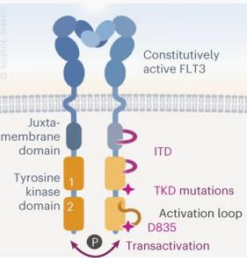
Mutated nucleophosmin 1 as immunotherapy target in acute myeloid leukemia

Dyantha I. van der Lee, ... , J.H. Frederik Falkenburg, Marieke Griffioen

J Clin Invest. 2019;129(2):774-785. <https://doi.org/10.1172/JCI97482>.

FLT3-TKD-specific, TCR tg T cells

(Giannakopoulou et al., *Nature Cancer* 2023)



nature cancer

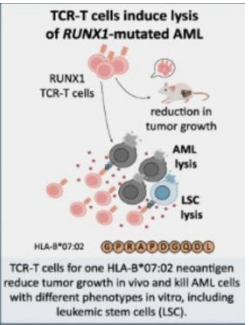
Article

<https://doi.org/10.1038/s43018-023-00642-8>

A T cell receptor targeting a recurrent driver mutation in FLT3 mediates elimination of primary human acute myeloid leukemia in vivo

RUNX1-frameshift mutations-specific T cells

(Struckman., *Leukemia* 2026)



Leukemia

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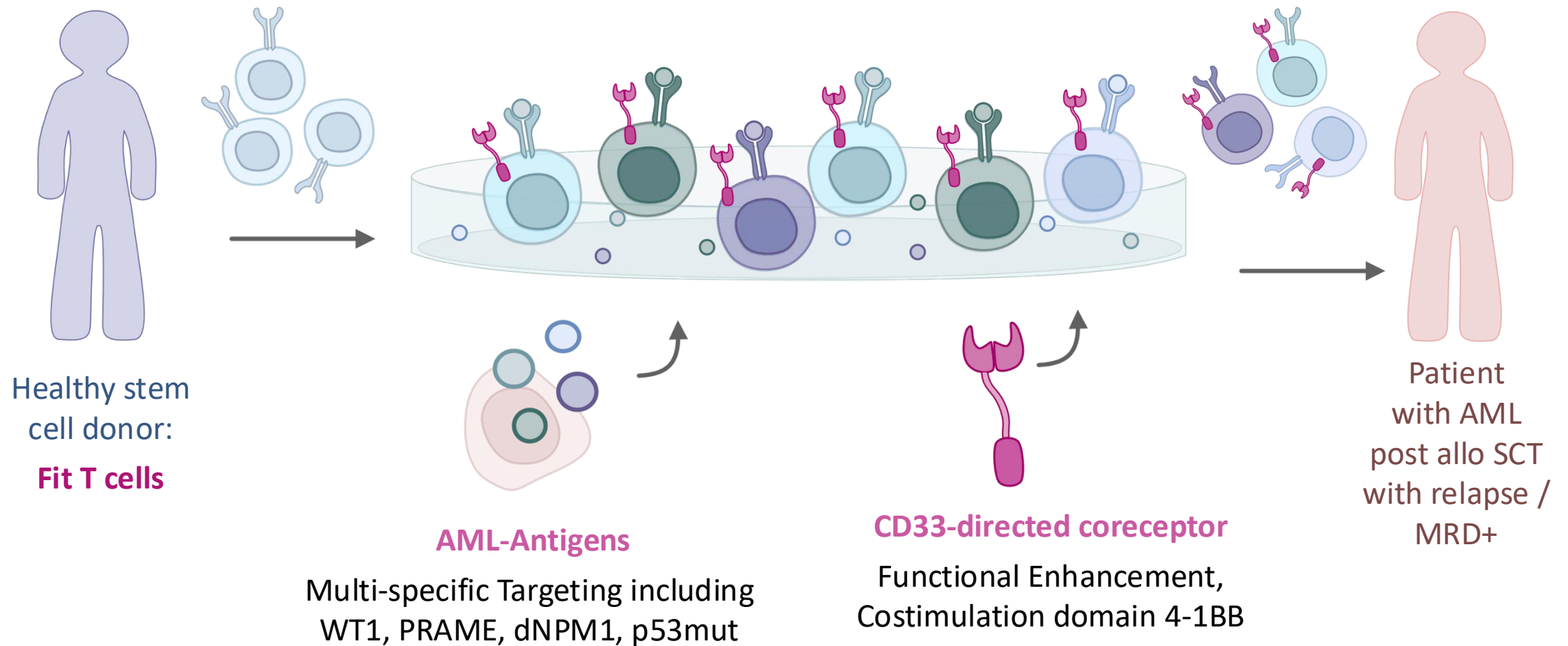
nature > leukemia > articles > article

Article | Open access | Published: 12 December 2025

A T-cell receptor targeting *RUNX1* frameshift mutations in acute myeloid leukemia

Nadine E. Struckman, Georgia Koutsoumpli, Rob C. M. de Jong, Dyantha I. van der Lee, Dennis F. G. Remst, Sophie-Anne J. Smith, M. Willy Honders, Renate S. Hagedoorn, Arnoud H. de Ru, Masashi Matsuda, Fumihiko Ishikawa, Tamar Tak, Peter J. M. Valk, Miriam H. M. Heemskerk, Peter A. van Veelen, J. H. Frederik Falkenburg & Marieke Griffioen

AML-Peptide-stimulated healthy Donor T-Cells transduced with a CD33-4-1BB Coreceptor



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- Stem cell edited allo grafts followed by immunotherapy, e.g. CART
- TCR-tg T cells / endogenous T cells targeting intracellular antigens

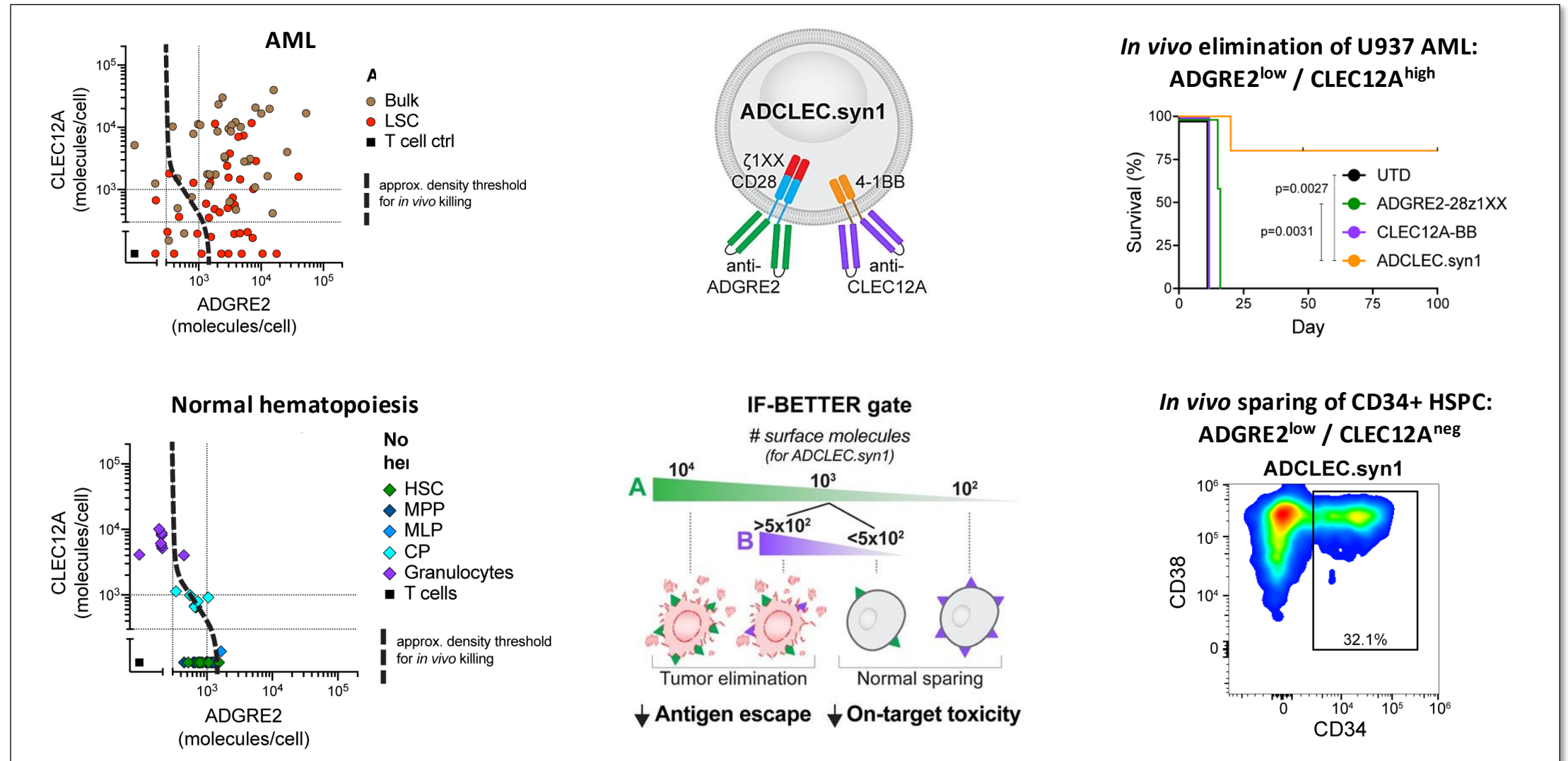
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- CART + switch receptors

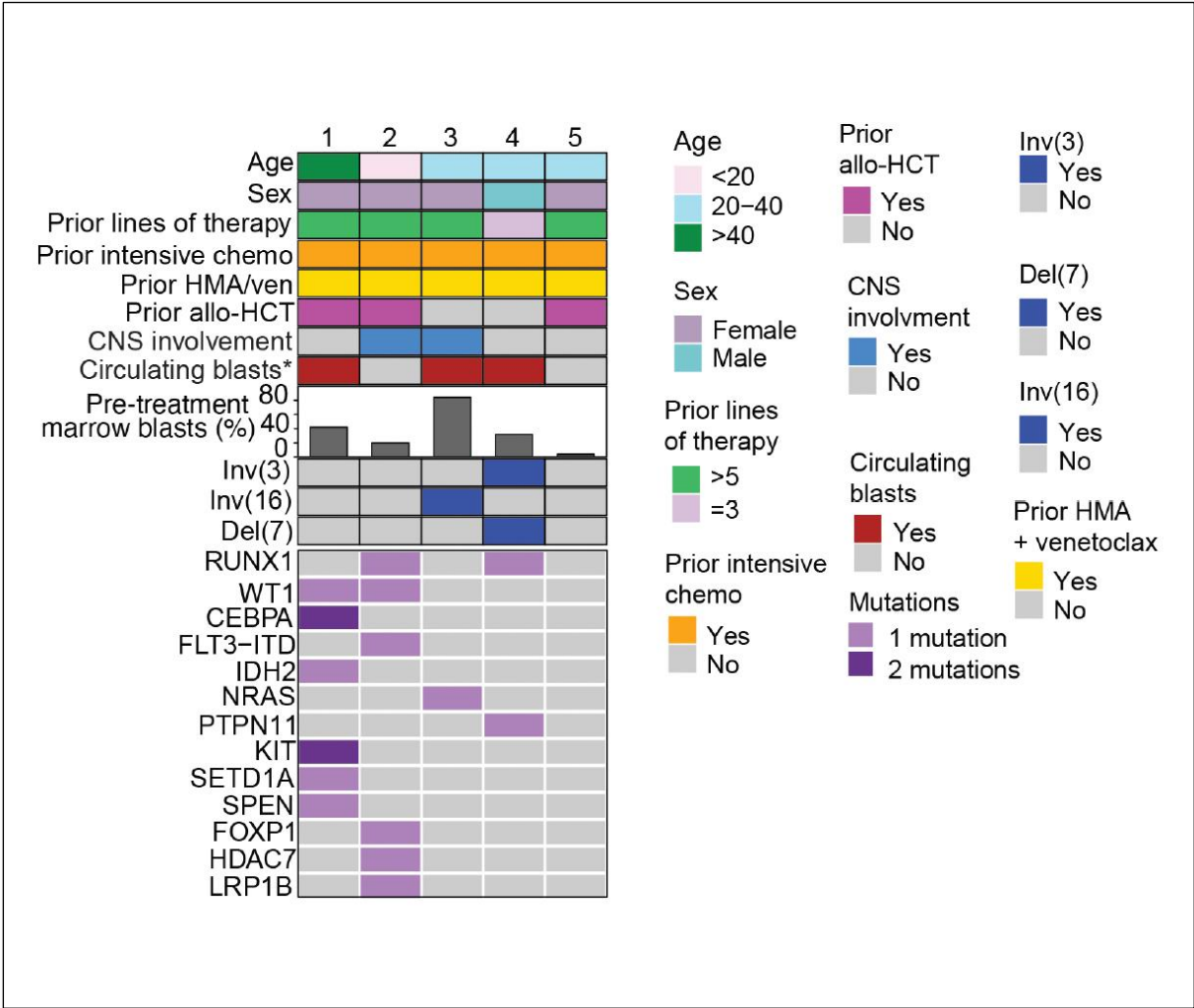
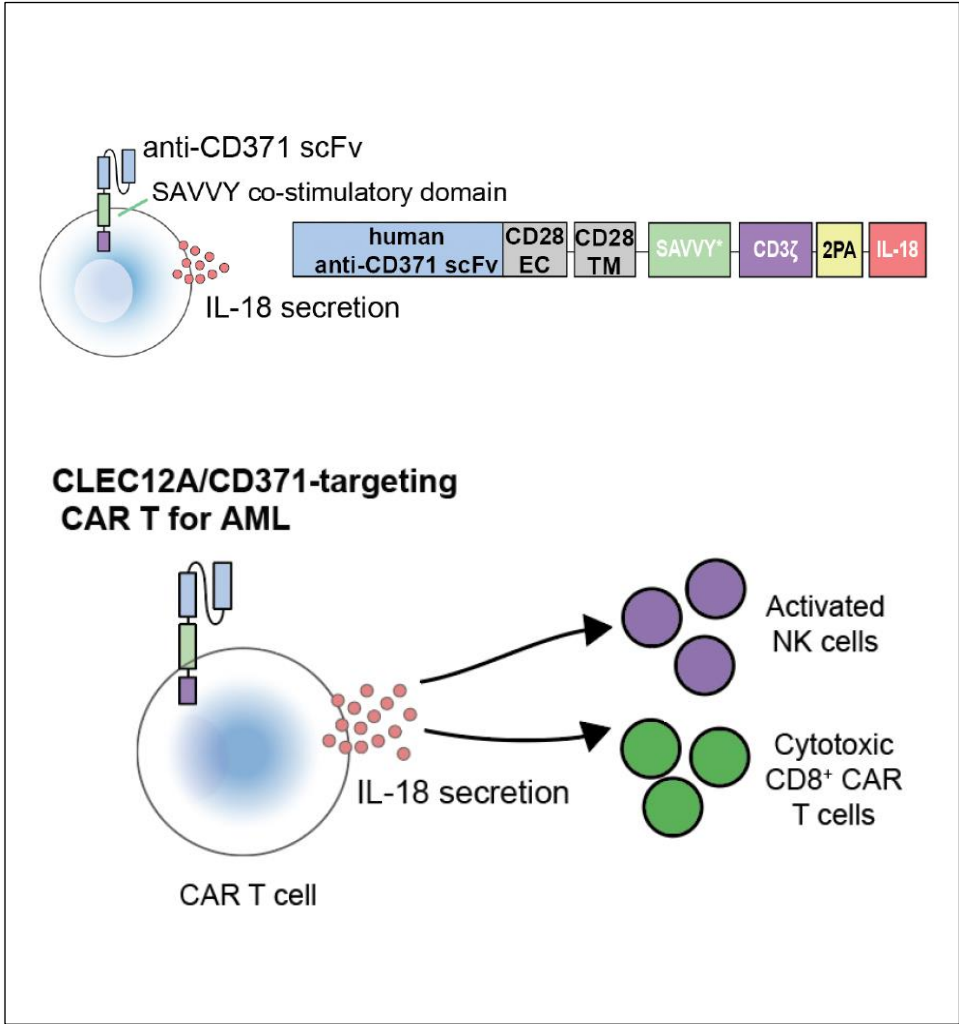
Combine with Standard of Care or other Immunomodulators

- VEN/AZA
- Menin Inhibitors
- Innate Immunity

Phase I at MSK: CAR: ADGRE2-CD28z3 + CCR: CLL1-41BB w/o zeta domain



Phase I Trial (CLEAR): CLL-1 CART Cells secreting IL-18



CLEAR: 5 Patients treated, 3/5 MRD negative by FLOW, promising CART Expansion

Pt Characteristics:

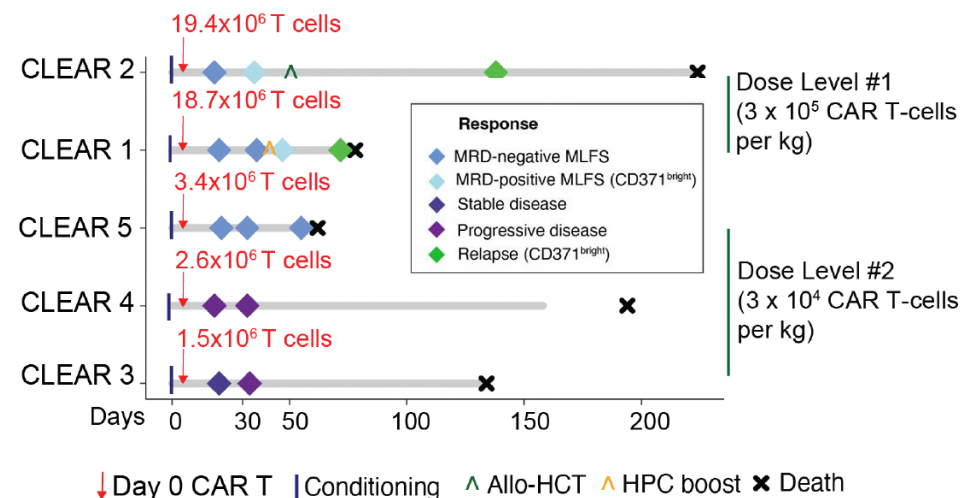
- Dose escalation
- 5 patients screened and infused
- median age 32 years (range, 16-45 years)
- Prior therapy: ven / cytarabine, 3 prior HSCT

Toxicities:

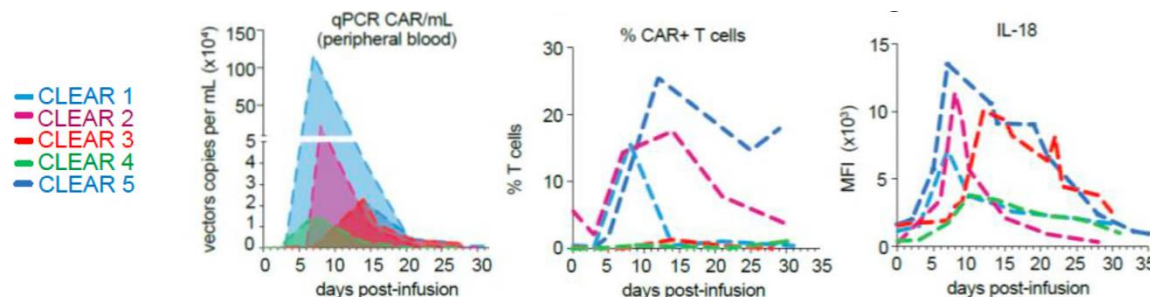
- 5/5 CRS, 2/5 G3-4
- 1/5 ICANS G3
- DLT x 2 with dose level 1, 1DLT at step down dose (3×10^{-4})

Responses:

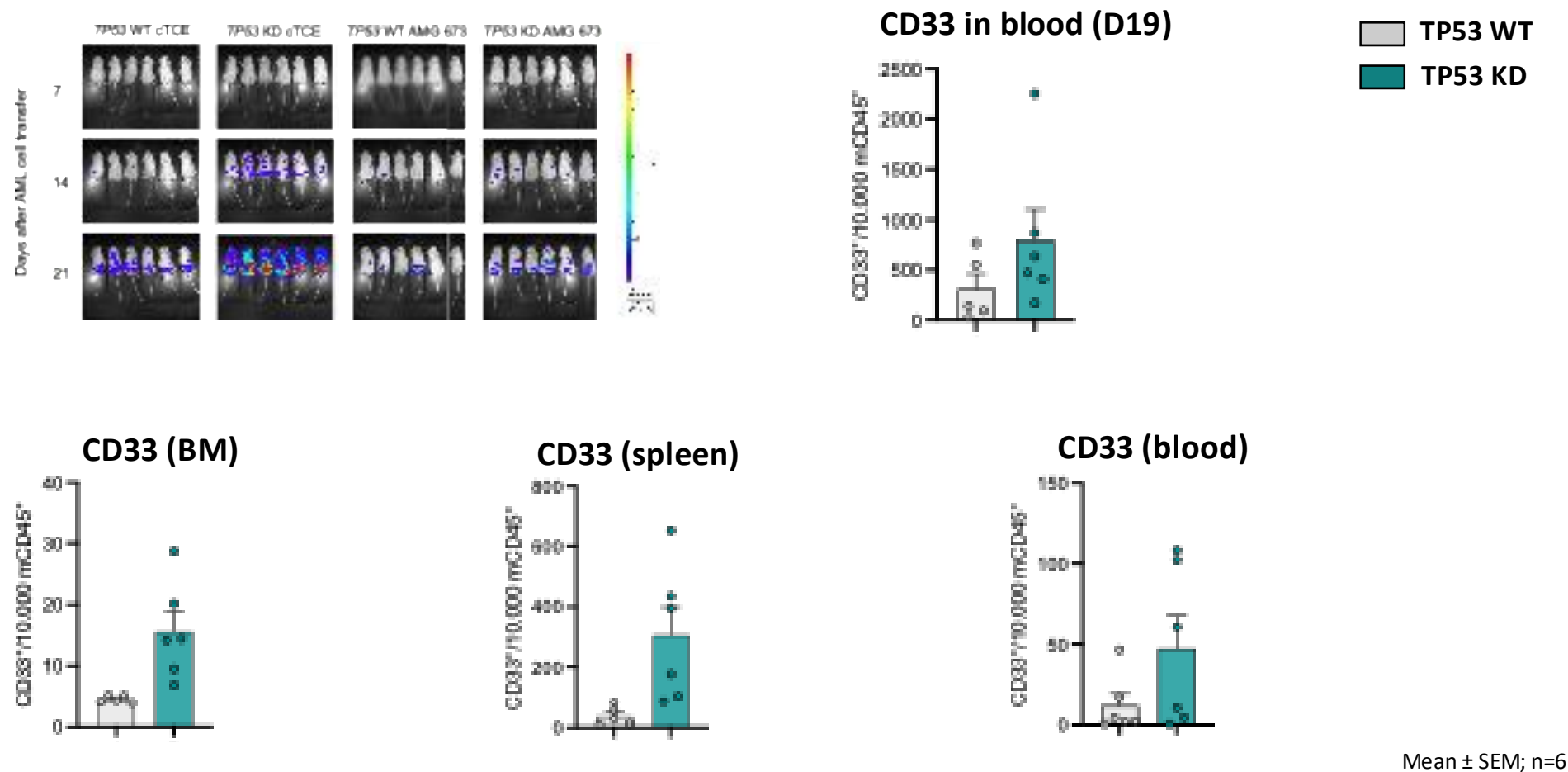
- 3/5 flow MRD neg MLFS (CAVE)
- 2/5 bridged to HSCT



Peak CAR T expansion co-incided with peak IL-18 levels (d7-14)

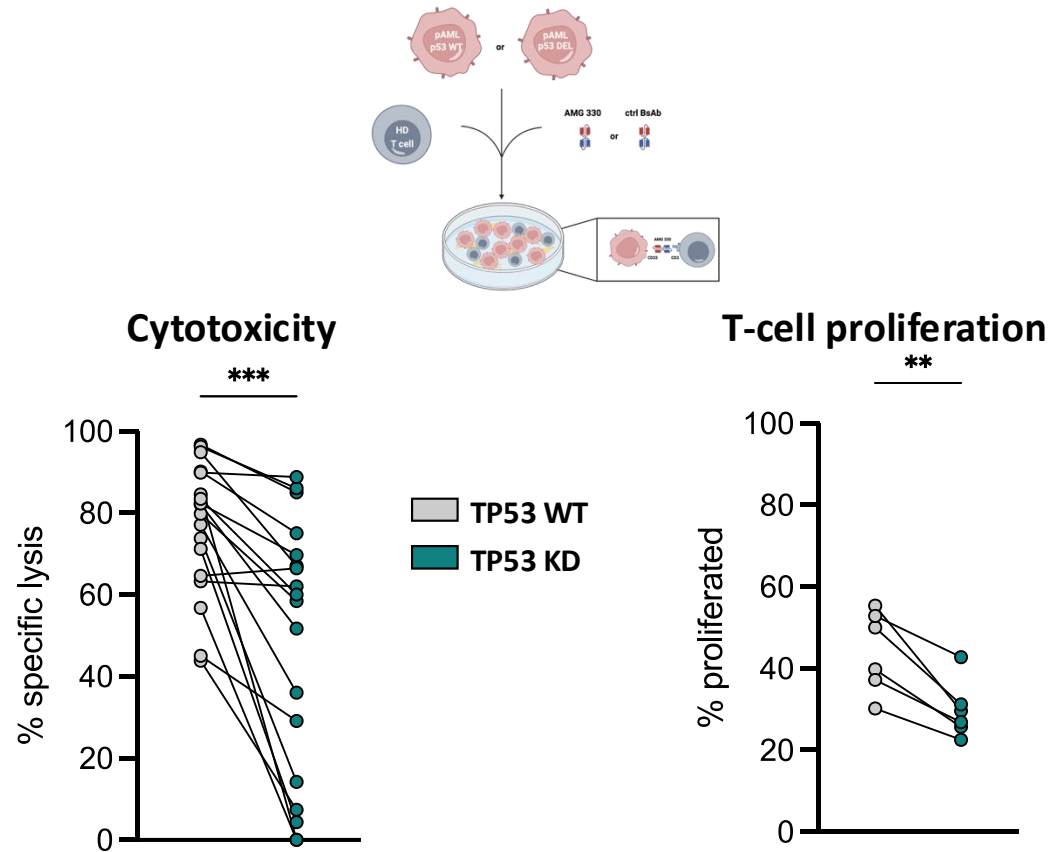


Diminished AMG 673-mediated Lysis of *TP53* KD in an *in vivo* NSG Mouse Model

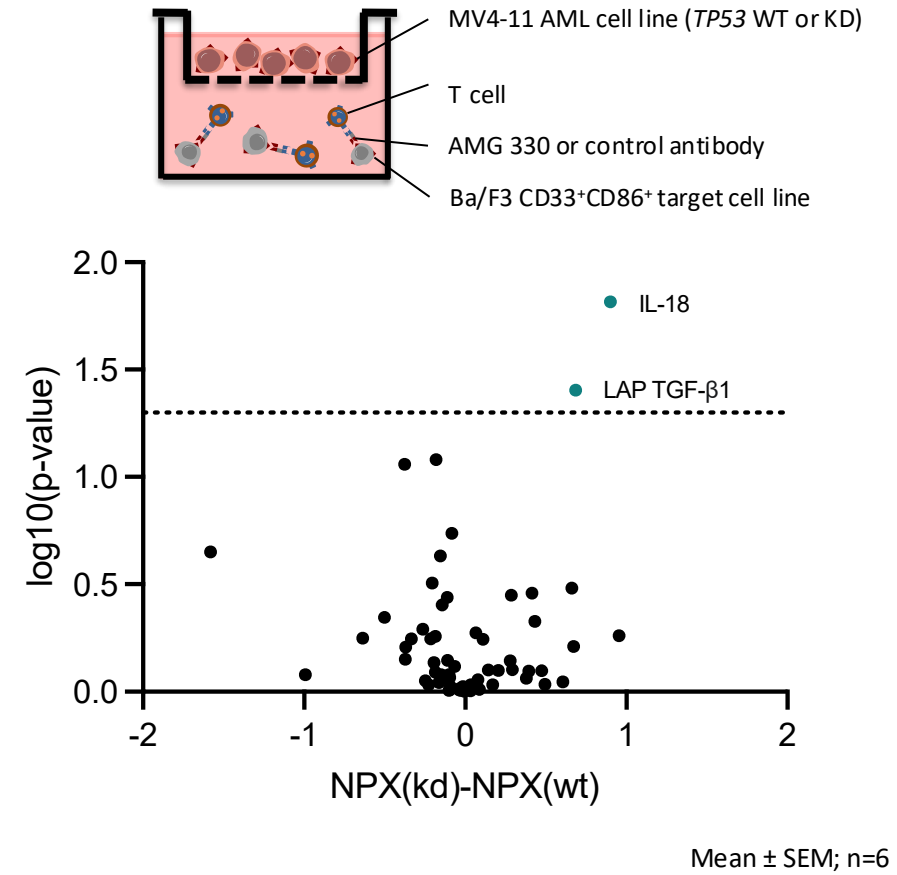


The Secretome of *TP53* KD AML Cells impairs T-cell Function

Comparison of a CD33 TCE against P53 wt vs p53 kd AML cells



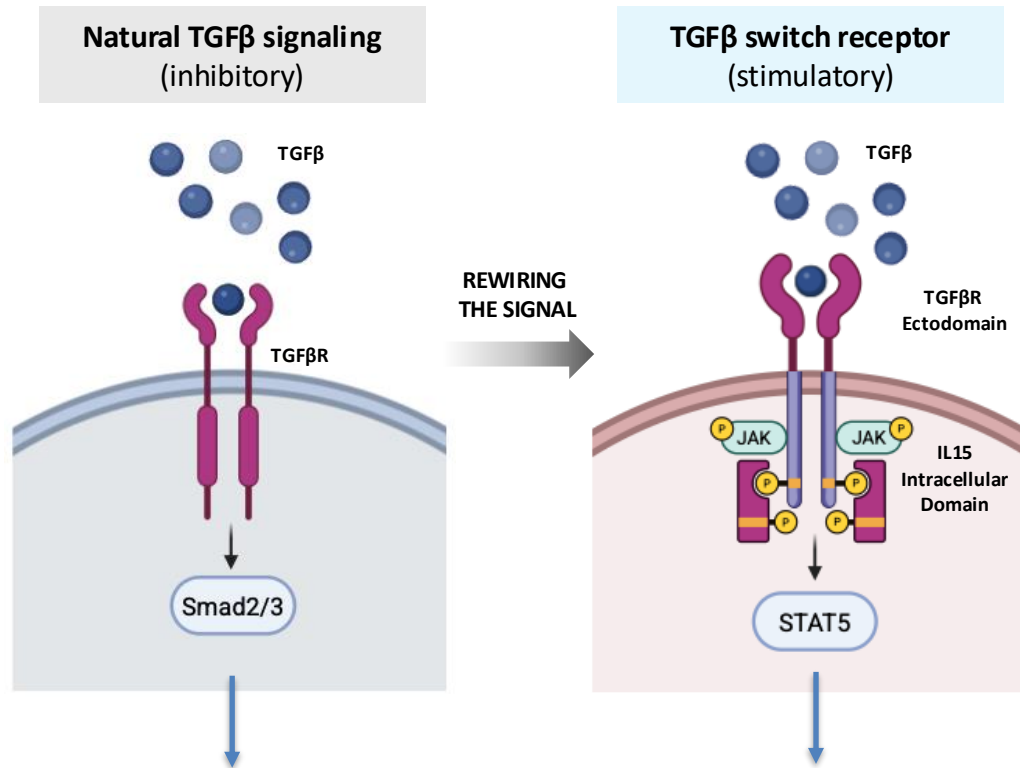
Secretome



Switch Receptors

Exploiting the immunosuppressive microenvironment as fuel to drive cell-based therapies against tumor

Switch Receptors: Rewiring inhibitory signal into stimulatory one



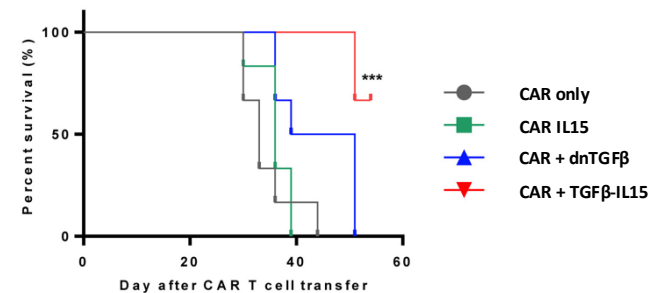
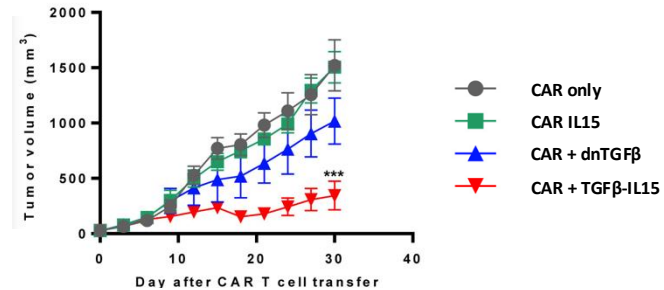
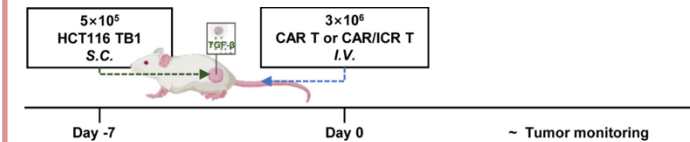
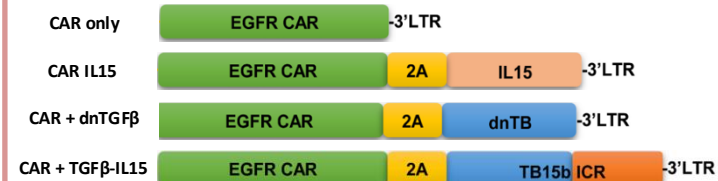
T- cell suppression

- ↓ Proliferation
- ↓ Cytokine production
- ↓ Cytotoxicity
- ↑ Exhaustion

T- cell activation

- ↑ Proliferation
- ↑ Cytokine production
- ↑ Cytotoxicity
- ↑ Persistence

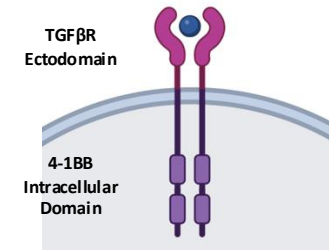
TGFβ-IL15 switch receptor



Adapted from: Zheng et al. Molecular Therapy, 2024; 33, 688-702

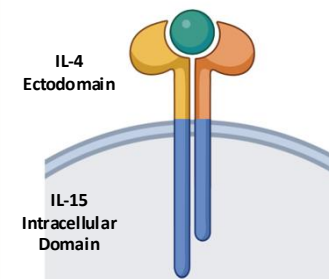
A broader switch receptor toolbox

TGFβ → 4-1BB



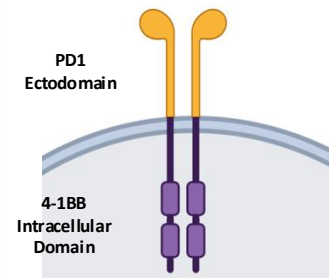
- Turns TGFβ signal into co-stimulatory signal
- Enhanced cytokine production, increased activation markers, improved anti-tumor activity

IL-4 → IL-15



- Converts IL-4 signal into STAT5 mediated IL-15 signal.
- Increased expansion, activation, cytotoxicity, cytokine production and reduced exhaustion.

PD-1 → 4-1BB



- Turns PD-1 inhibitory signal into co-stimulatory signal.
- Improved *in-vitro* cytotoxicity, persistence, proliferation, and tumor termination.

Strategies to improve Efficacy of CART in AML

AML Targeting:

- Adapter CART cells with on/off targeting modules
- Stem cell edited allo grafts followed by immunotherapy, e.g. CART
- TCR-tg T cells / endogenous T cells targeting intracellular antigens

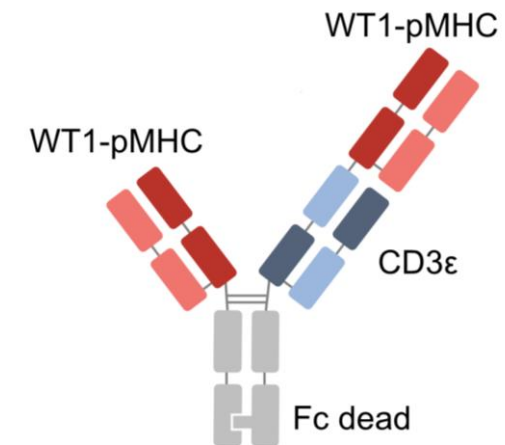
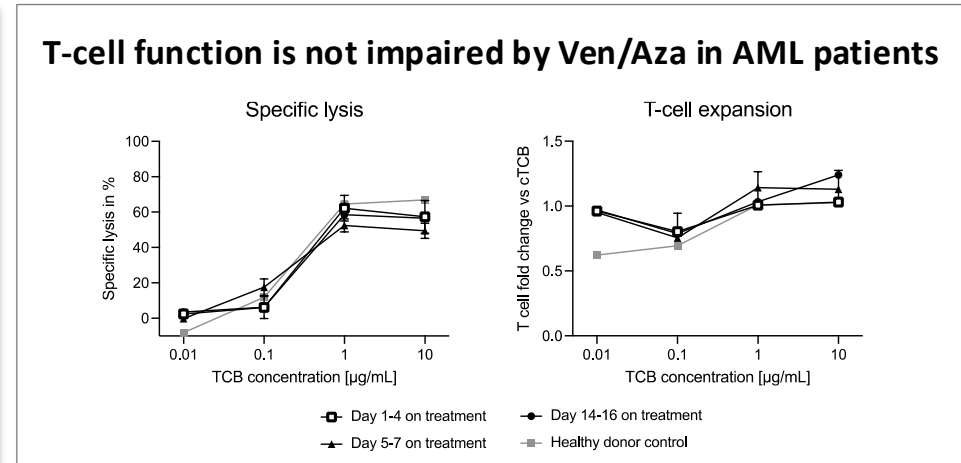
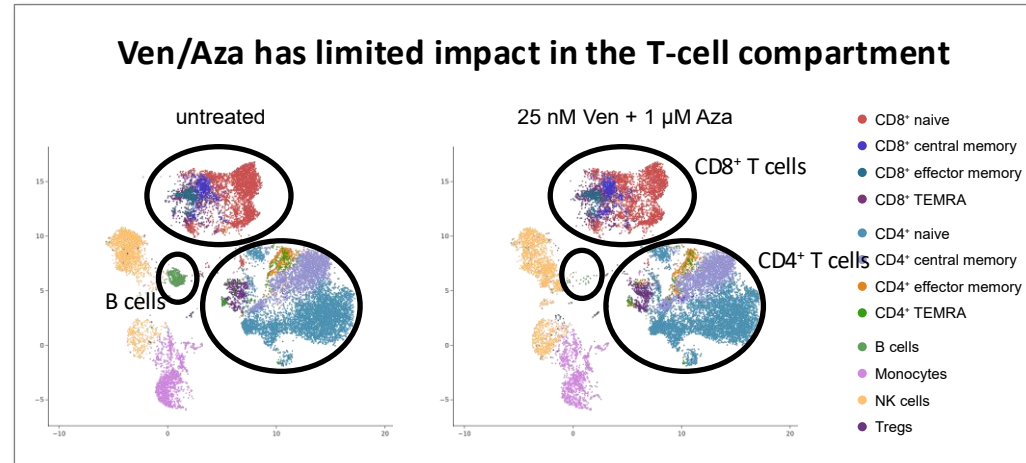
Improve T Cell Fitness:

- CART + CCR
- CART pharmacy
- CART + switch receptors

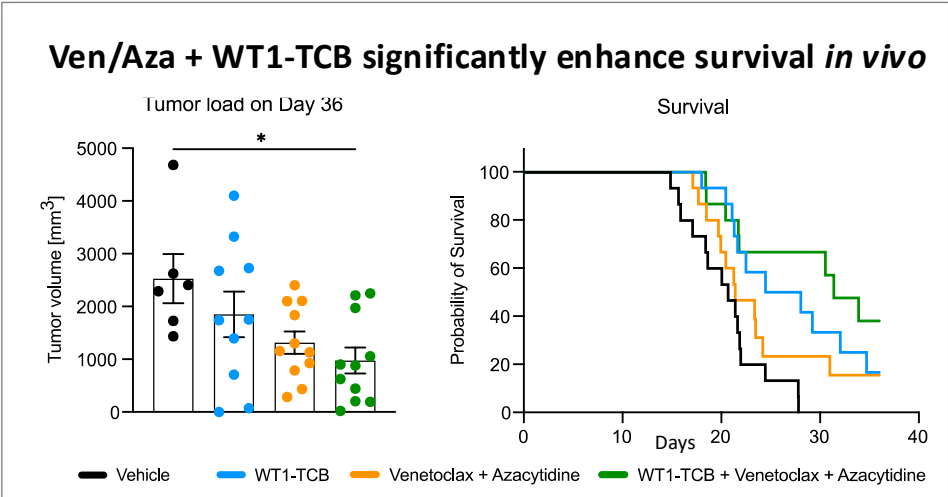
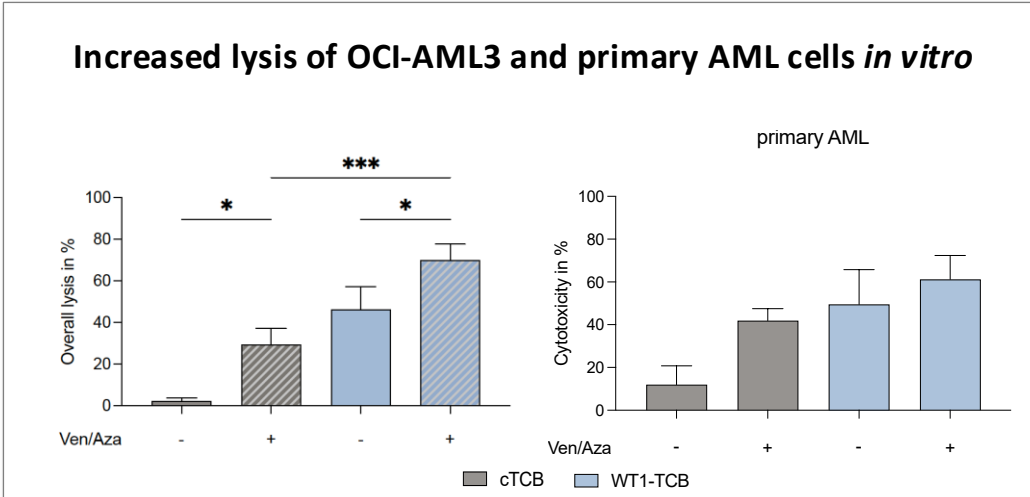
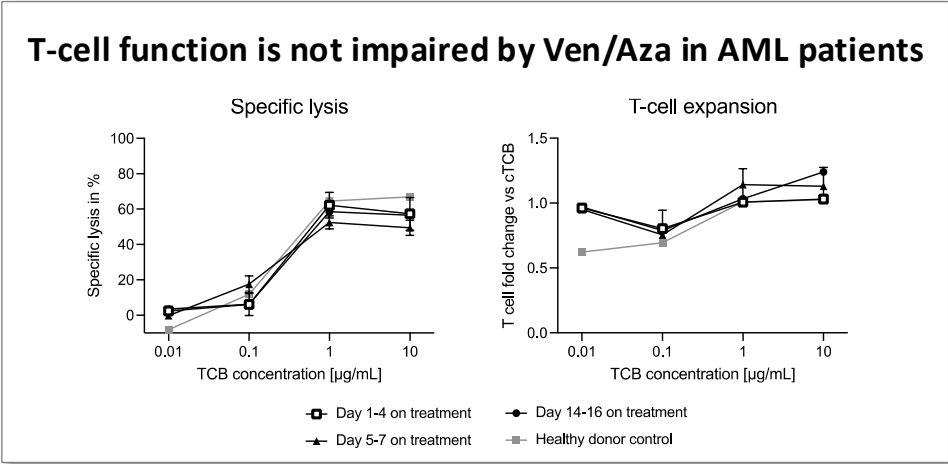
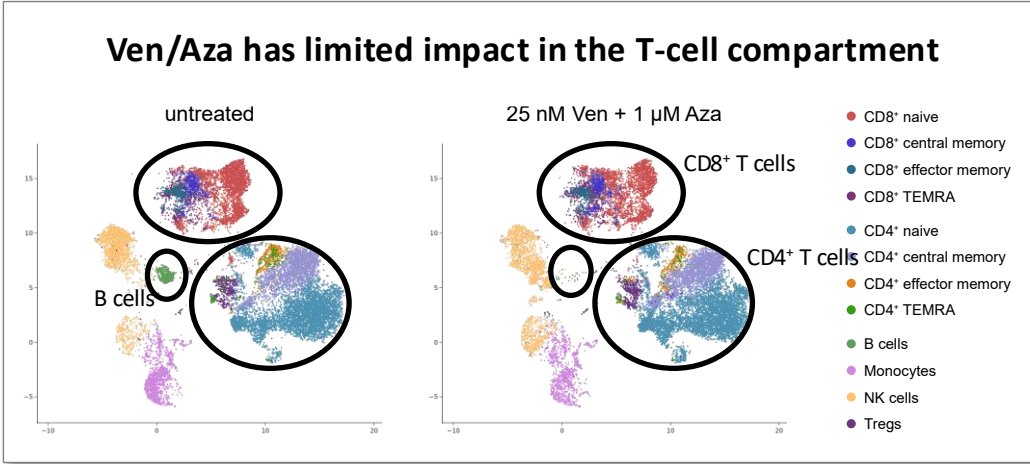
Combine with Standard of Care or other Immunomodulators

- VEN/AZA
- Menin Inhibitors
- Innate Immunity

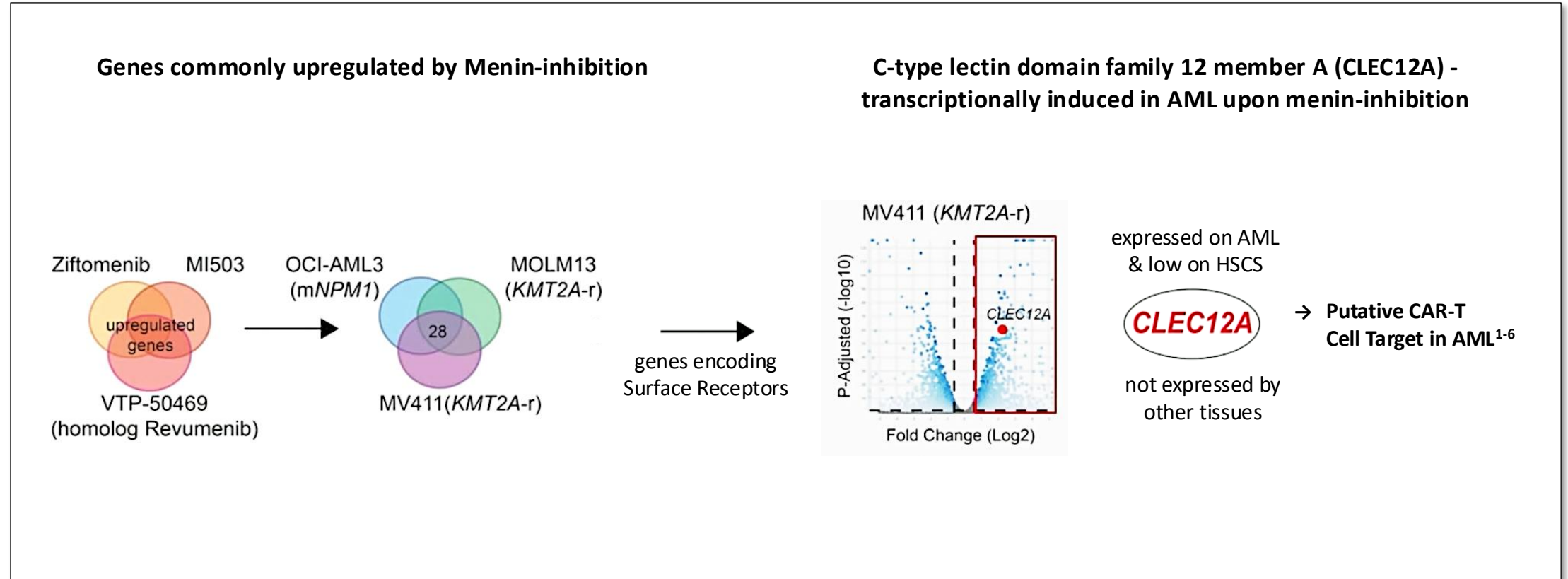
TCE + VEN/AZA: Immunmonitoring does not show any Impact on T-cell Function



TCE + VEN/AZA: Increased Cytotoxicity against AML Cell Lines + pAML Cells *in vitro* + *in vivo*

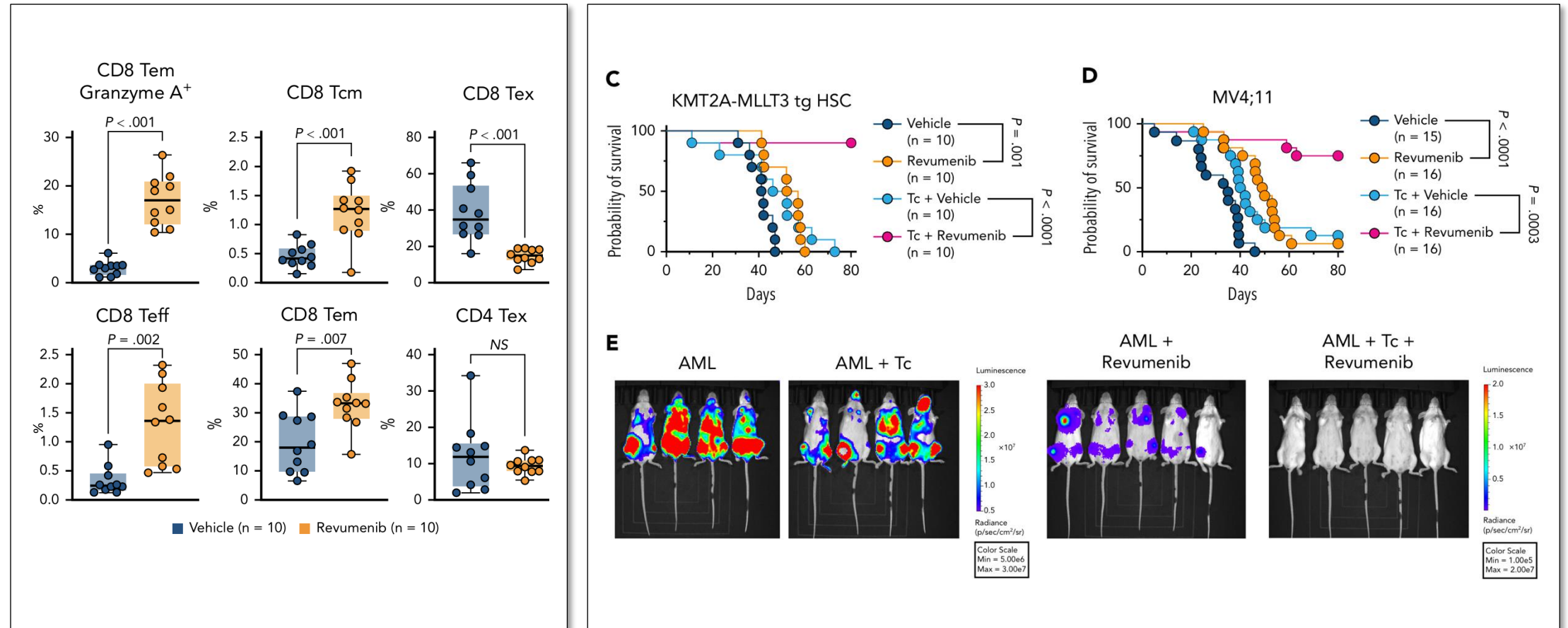


Increasing the Therapeutic Window: Menin-Inhibition leads to Differentiation of AML Cells and Induction of Immune Target Antigens like CLL1 (CLEC12a)



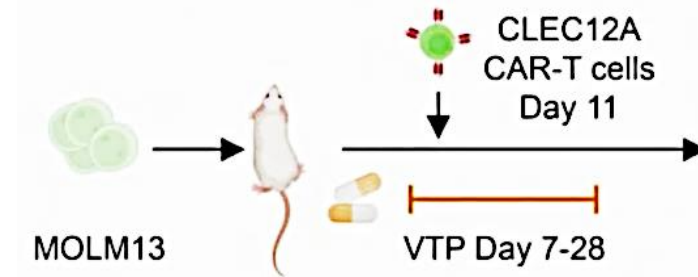
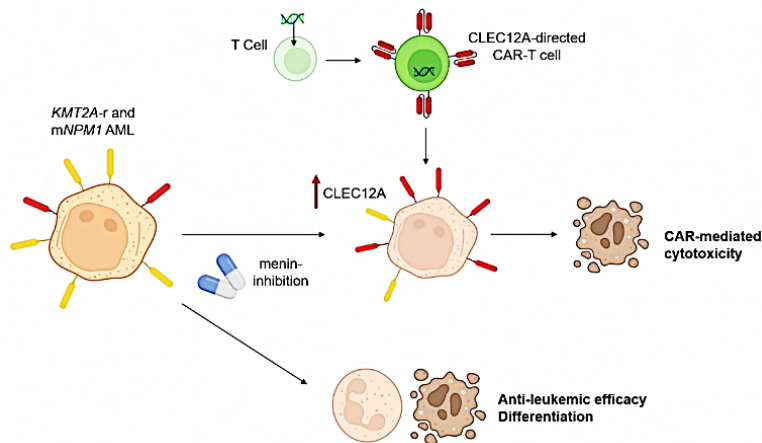
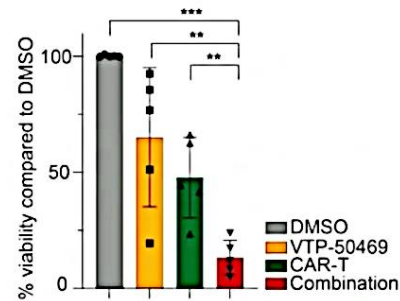
Menin-inhibition enhances Activation of T Cells *in vitro* and *in vivo*

Mode of Action: Menin inhibition increased Expression of multiple human endogenous Retroviruses, leading to Upregulation of interferon-stimulated gene Upregulation

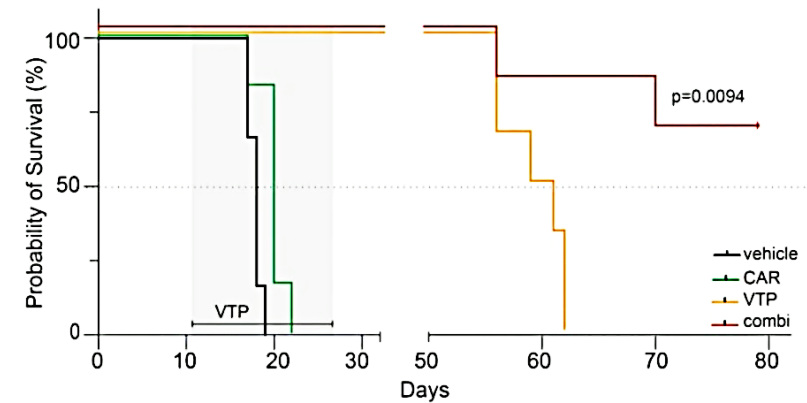


Revumenib (VTP-50469) led to a significantly enhanced anti-leukemic Activity of CLL1-directed CART *in vitro* and *in vivo*

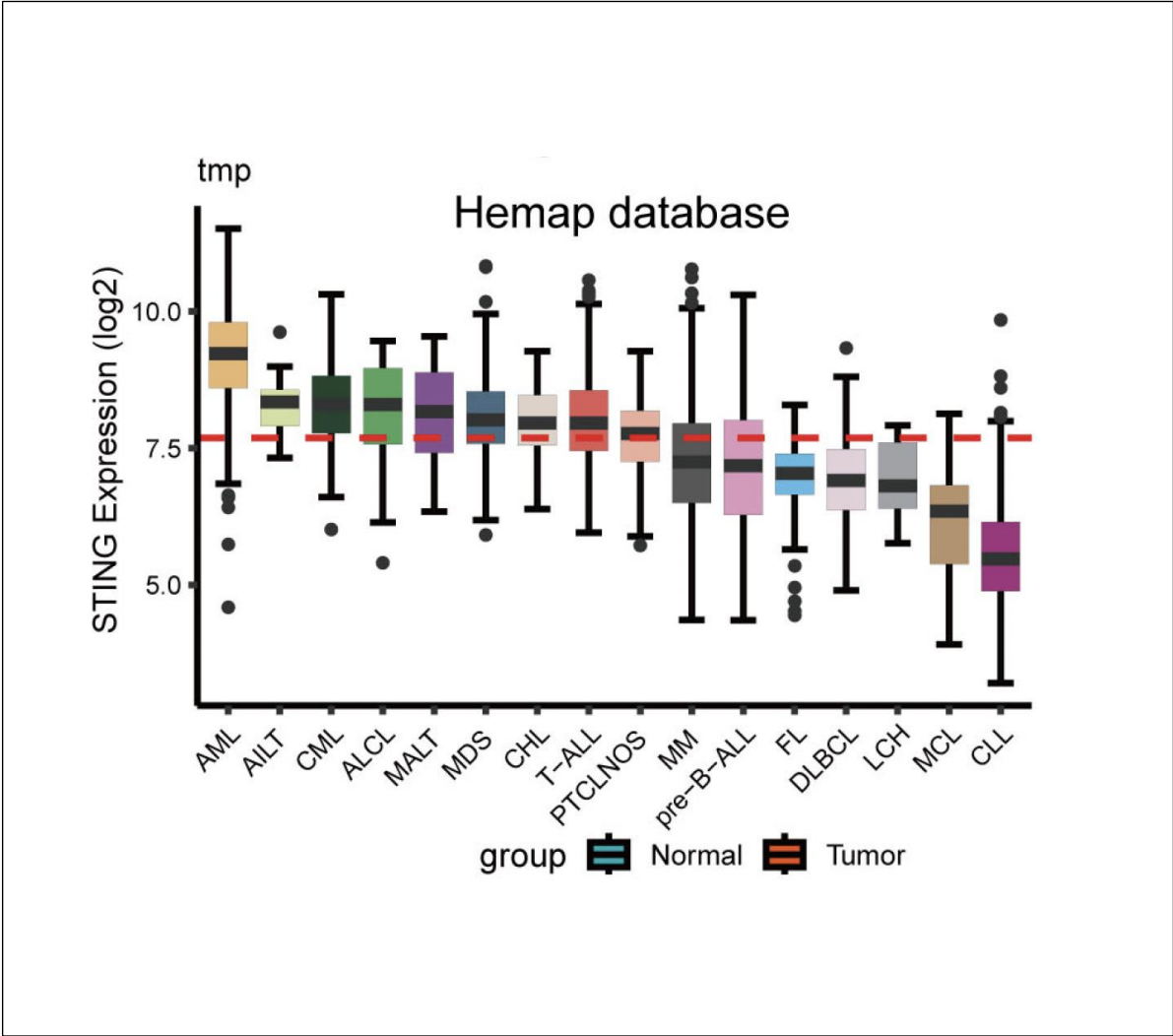
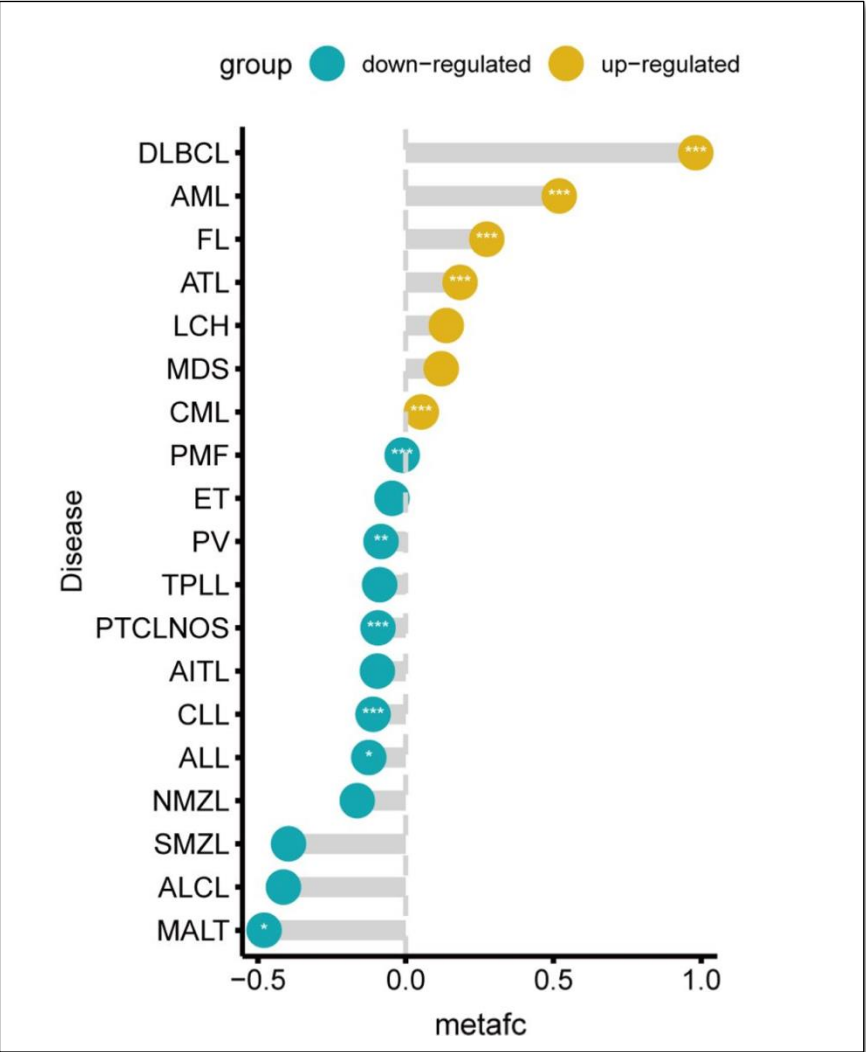
Superior anti-leukemic activity *in vitro* mNPM1 OCI-AML3



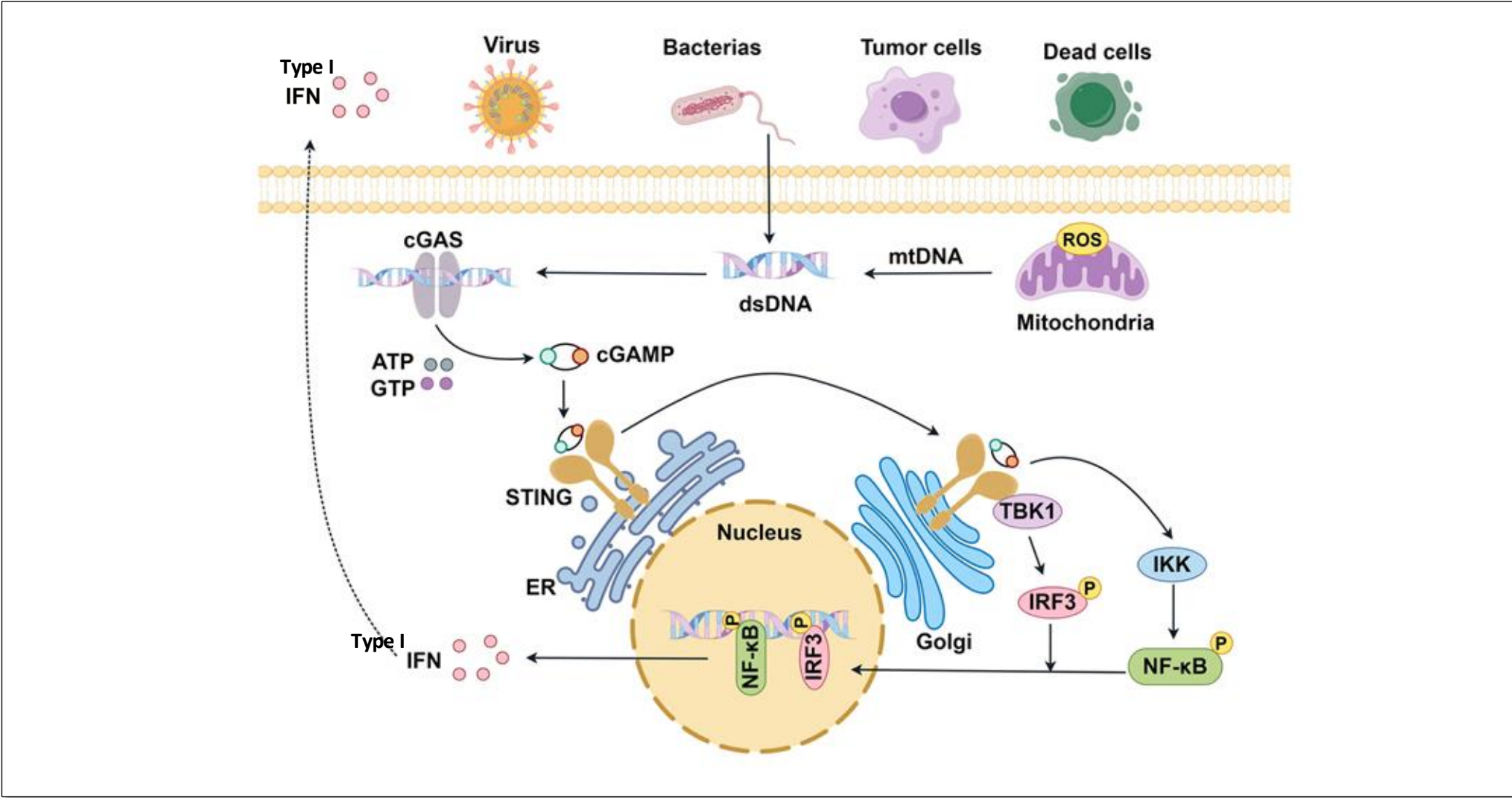
Survival



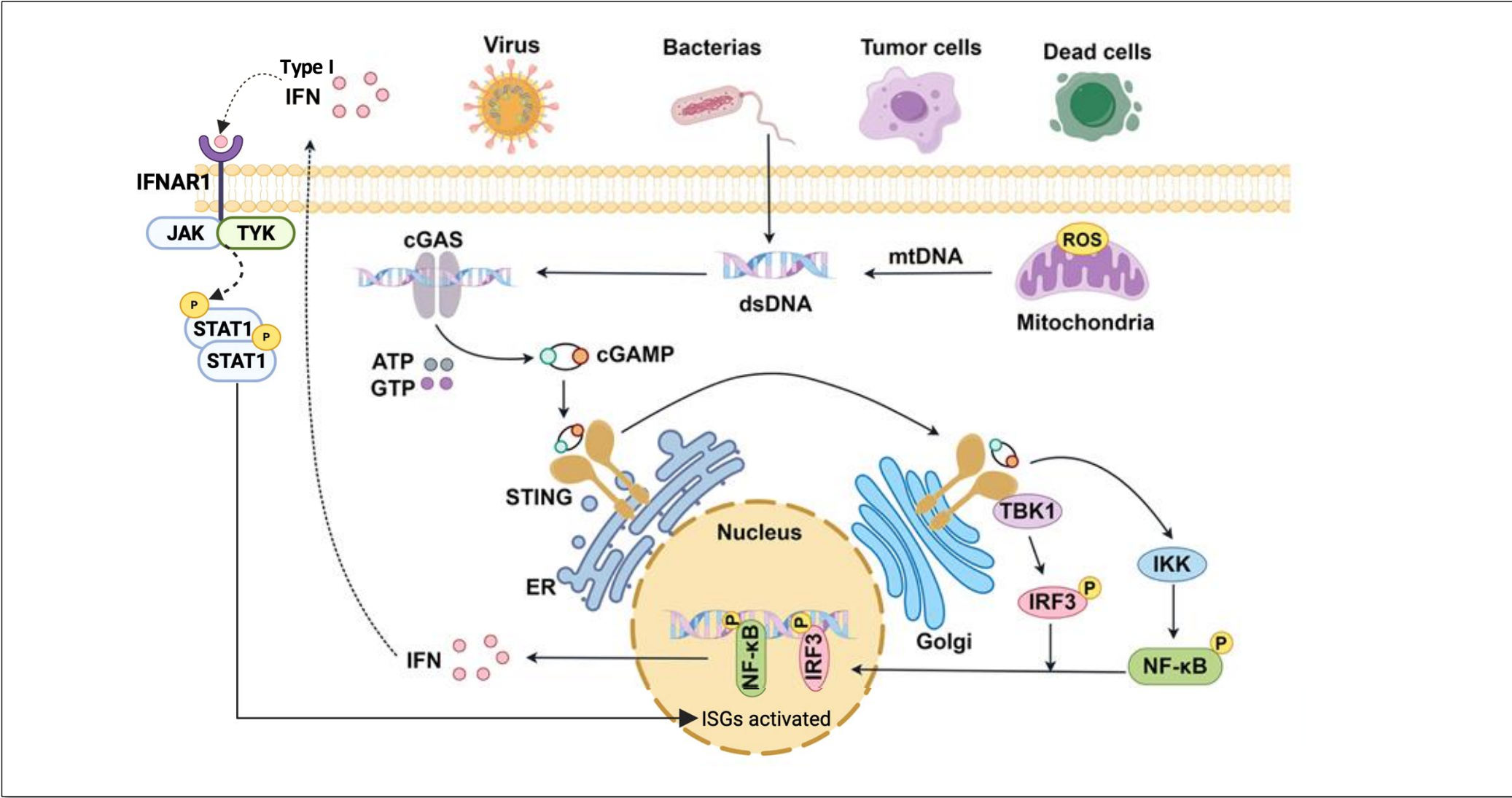
High Expression of STING in AML



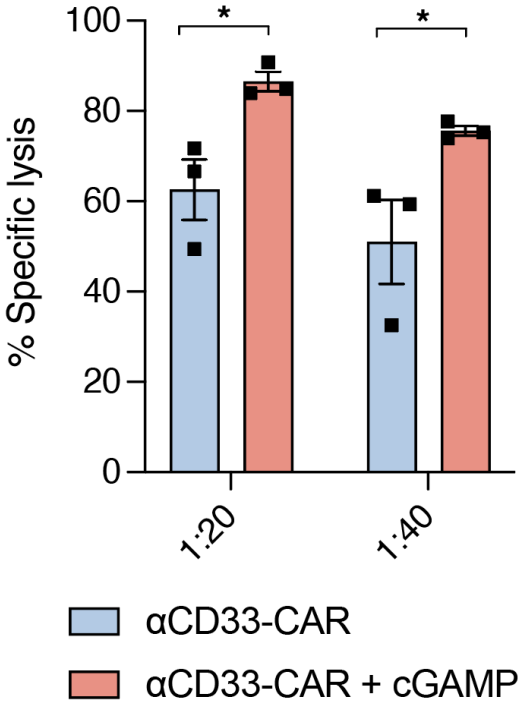
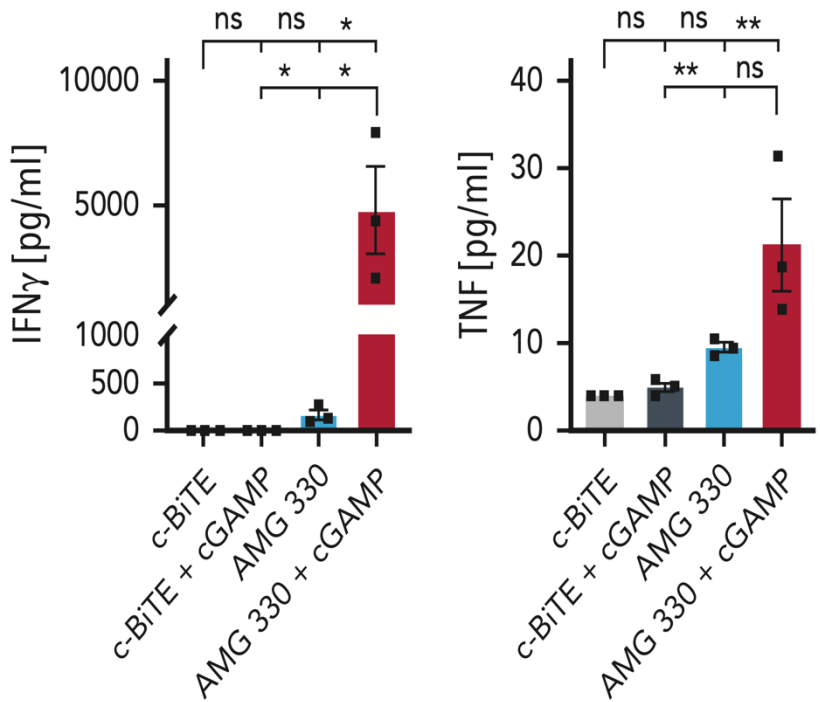
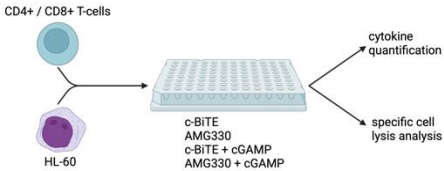
cGAS-STING Pathway



cGAS-STING Pathway

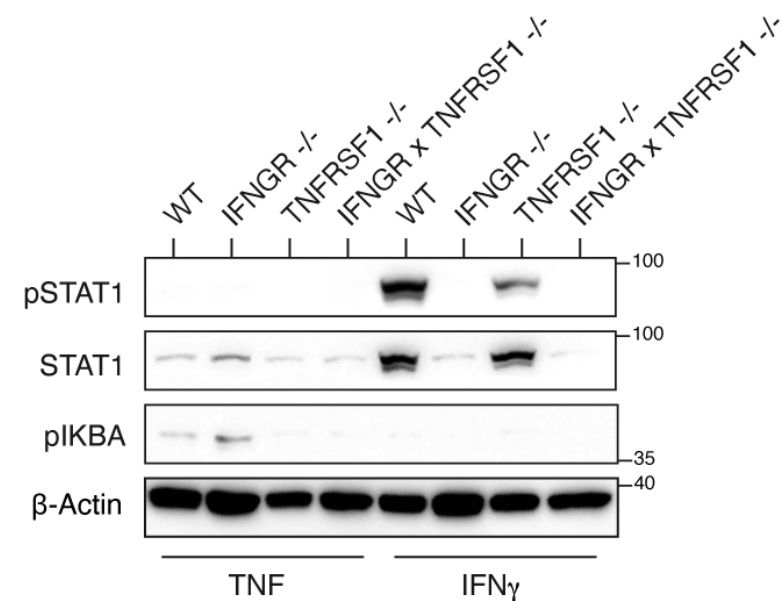


cGAMP boosts CD33-directed AML Killing by TCE or CART



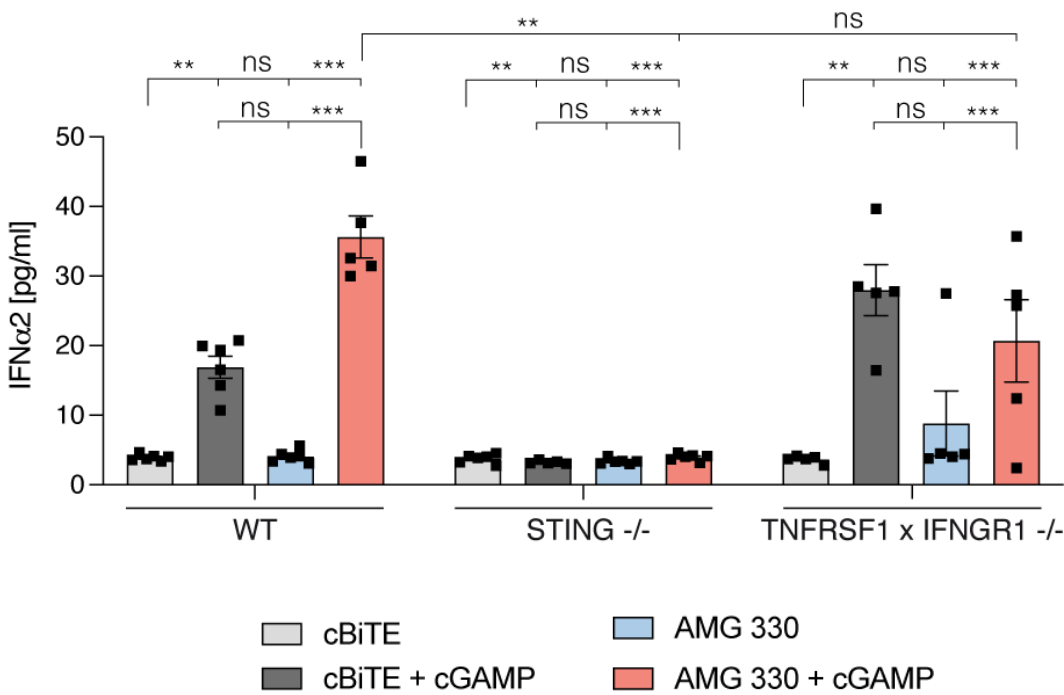
IFN- γ + TNF knockout or STING knockout AML Cells: lower IFN α 2a Secretion by AML Cells

CRISPR/Cas9 Knockout



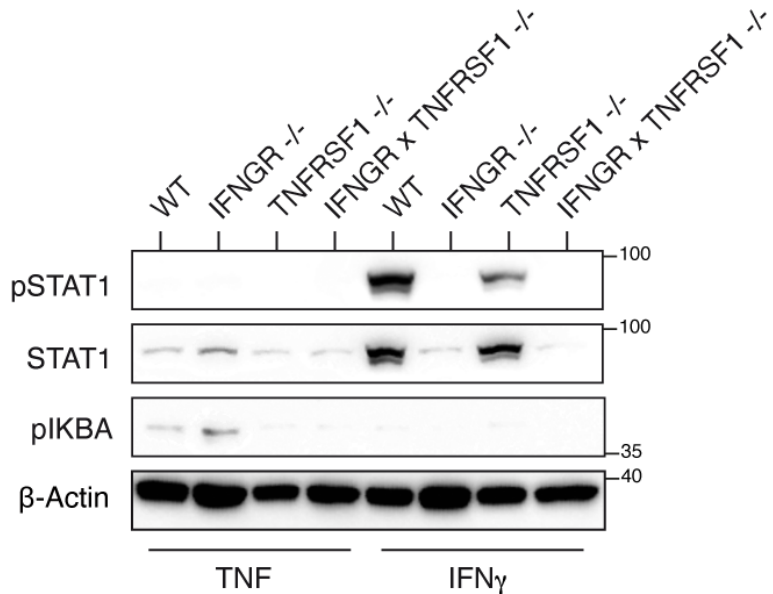
HL-60 cell lines of the indicated genotypes were treated with IFN- γ or TNF and phosphorylation of STAT1 (pSTAT1) or I κ B α (pIKBA) was assessed via immunoblotting.

IFN α 2a Secretion



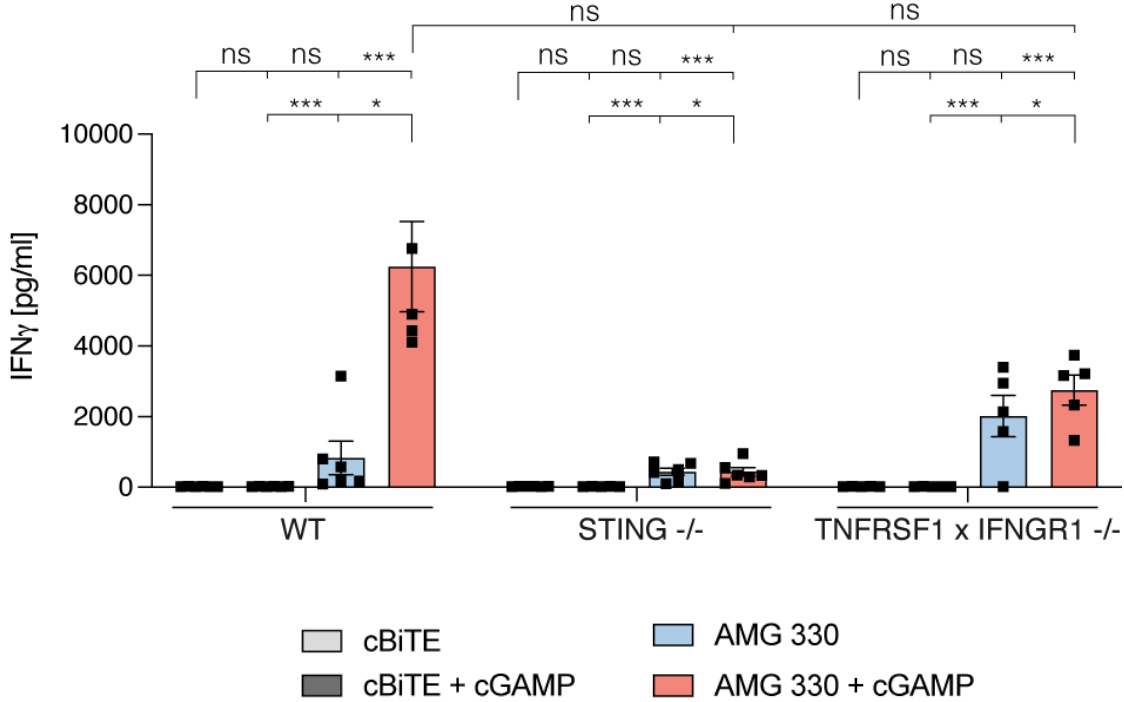
IFN- γ + TNF Knockout or STING Knockout AML Cells: lower IFN- γ Secretion by T Cells

CRISPR/Cas9 Knockout



HL-60 cell lines of the indicated genotypes were treated with IFN- γ or TNF and phosphorylation of STAT1 (pSTAT1) or I κ B α (pI κ B α) was assessed via immunoblotting.

IFN- γ Secretion



Take-home messages

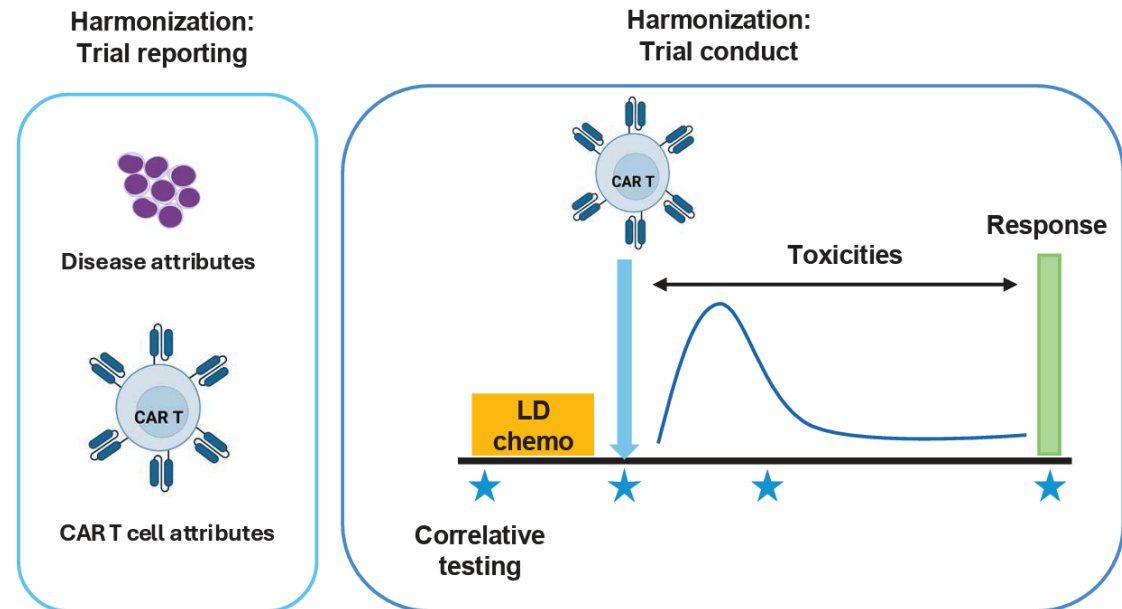
- T cells can cure AML — the GVL effect proves it. CAR-T has not yet reproduced it.
- Two separable problems: the target (shared myeloid antigens, narrow therapeutic window) and the T cell (dysfunction in the AML niche) induced by genetics & LME
- Target side: Adapter/TM guided Approaches, Stem cell editing to make the stem cell graft invisible to the CART given afterwards, targeting of intracellular, more leukemic specific target antigens
- T-cell side: enhance effector function and thereby overcome an hostile bone marrow environment by chimeric coreceptors, cytokine secreting CART, switch receptor CART
 - Combining CART with standard of care: HMA/VEN, Menin Inhibitors, STING agonists.
 - Patient Selection: early in treatment line (high risk first line), low tumor burden (MRD+)

IMPACT-AML: International multi-center Partnership in CAR T Cell Therapy for AML

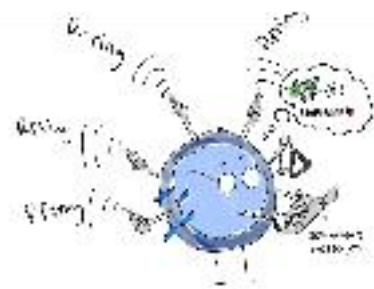
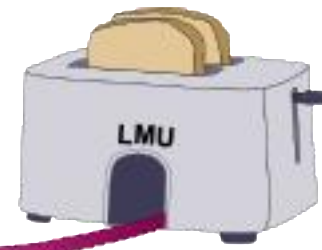
International Consensus Guidelines for the Conduct and Reporting of CAR T-Cell Clinical Trials in AML

Swati Naik, Richard Aplenc, Susanne HC Baumeister, Marco Becilli, Anand Shripad Bhagwat, Challice L Bonifant, Lihua E Budde, Christopher D Chien, Kevin J Curran, Anthony F Daniyan, Alexandra Dreyzin, Rebecca A Gardner, Sara Ghorashian, Stephen Gottschalk, LaQuisa C Hill, Mark Eric Kohler, Adam Joseph Lambie, Franco Locatelli, Maksim Mamonkin, Bilal Omer, Jae H Park, Concetta Quintarelli, Benedetta Rambaldi, Rebecca M Richards, David A Sallman, Tim Sauer, Nirali N Shah, Marion Subklewe, Corinne Summers, Sarah K Tasian, Naomi Taylor, Sarah Tettamanti, Michael R Verneris, M Paulina Velasquez, Saar I Gill

Outline of key recommendations for conduction and reporting of AML CAR T cell clinical trials



SUBKLEWE LAB
Translational Cancer Immunology



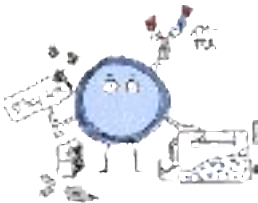
Veit



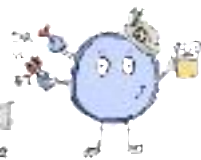
Marion



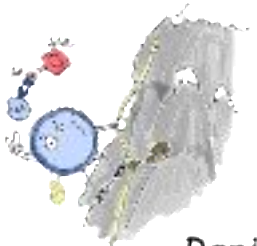
Sabine



Gerulf



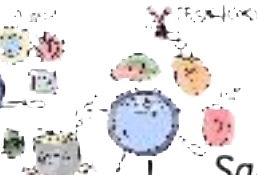
Lisa



Daniel



Pier



Saksham



Paulina



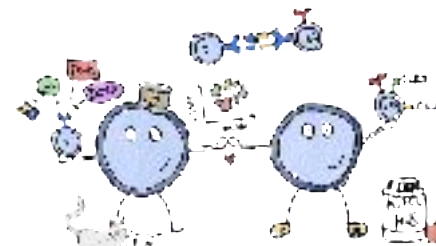
Anne-Sophie



Giulia

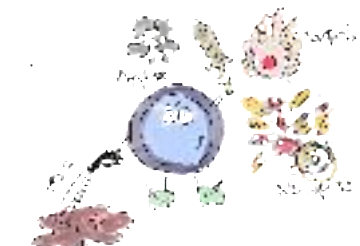


Rosella



Amelie

Lis



Cristina



LFL



Students



Savanna



Nora



Kieron



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Collaborators

Mirco Friedrich
Caitlin Zebley

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Karl-Peter Hopfner	Andreas Linder
Veit Hornung	



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