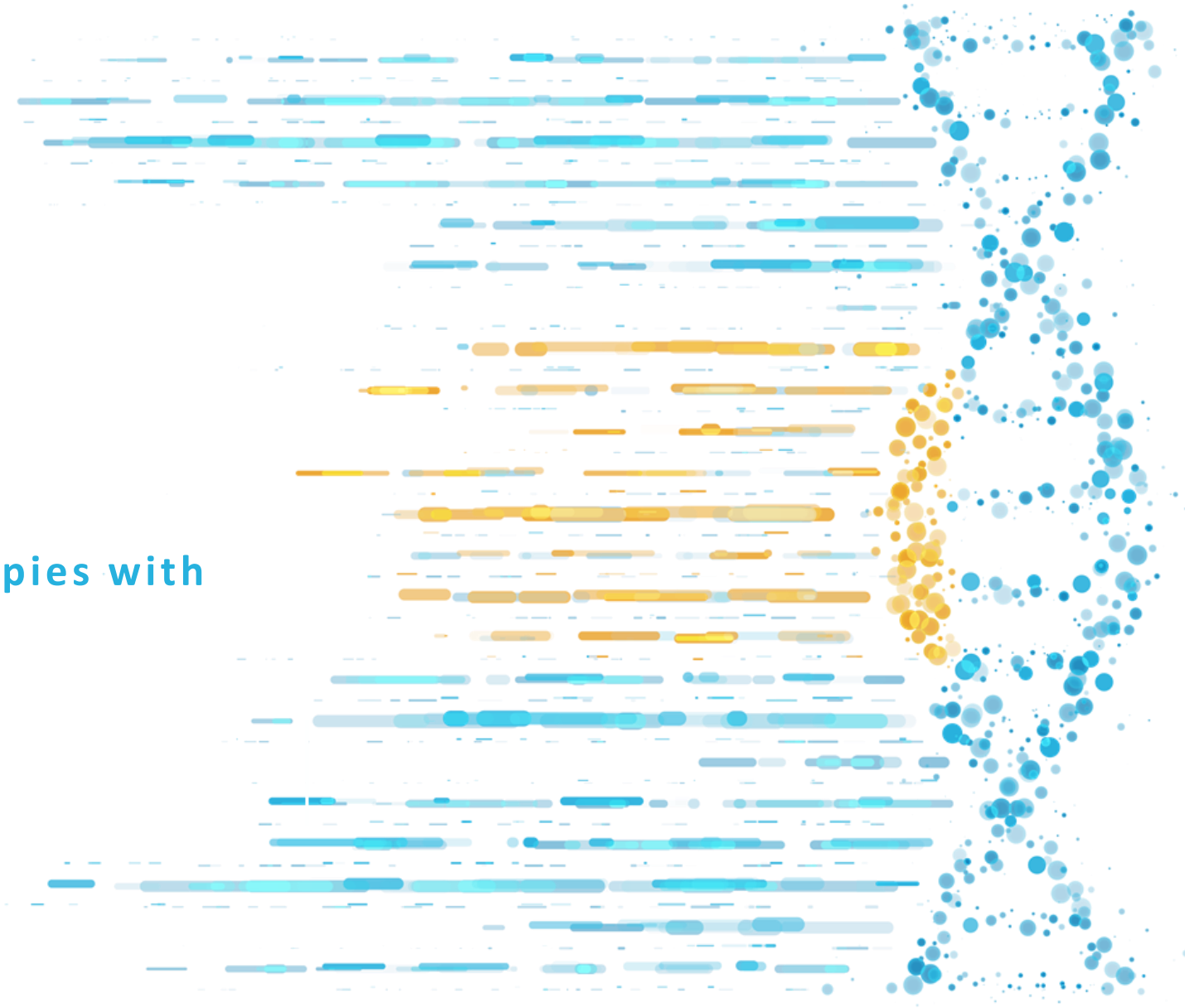
POSEIDA THERAPEUTICS

P-BCMA-ALLO1 Clinical Data Conference Call

A New Class of Cell & Gene Therapies with the Capacity to Cure



December 10, 2023

Forward-Looking Statements

This presentation and any accompanying oral commentary contain "forward-looking statements" about Poseida Therapeutics, Inc. within the meaning of the Private Securities Litigation Reform Act of 1995, as amended. Forward-looking statements are statements that are not historical facts and include, without limitation, statements related to future events; expected plans with respect to clinical trials, including timing of clinical data updates; the potential capabilities and benefits of our technology platforms and product candidates; our plans and strategy with respect to developing our technologies and product candidates; and future results of anticipated development efforts. Words such as "expect(s)," "feel(s)," "believe(s)," "will," "may," "might," "could," "anticipate(s)," “potentially” or negative of these terms or similar expressions are intended to identify forward-looking statements. These forward-looking statements are based on management's current expectations of future events only as of the date of this presentation and are subject to a number of important risks and uncertainties that could cause actual results to differ materially and adversely from those set forth in or implied by such forward-looking statements. These risks and uncertainties include, but are not limited to: the fact that our collaboration agreement with Roche may be terminated early; the fact that we will have limited control over the efforts and resources our collaborator devotes to advancing development programs under our collaboration agreement; risks associated with conducting clinical trials; whether any of our product candidates will be shown to be safe and effective; our ability to finance continued operations; our reliance on third parties for various aspects of our business; competition in our target markets; our ability to protect our intellectual property; our ability to retain key scientific or management personnel; and other risks and uncertainties described in our filings with the Securities and Exchange Commission, including under the heading “Risk Factors”. Except as required by law, we assume no obligation to update these forward-looking statements, or to update the reasons why actual results could differ materially from those anticipated in the forward-looking statements, even if new information becomes available in the future.

Introduction

Mark Gergen

Chairman and CEO

On a mission to advance a new class of cell & gene therapies

ALLOGENEIC CAR-T

The Future of Cell Therapy is Allo

Roche

IN VIVO GENE THERAPY

Moving Beyond Viral Vectors for Gene Therapy

OUR PEOPLE

Passionate and dedicated team working on treatments for patients with cancer and rare diseases

OUR PLATFORMS

Innovating with powerful and differentiated genetic engineering technologies

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The Future of Cell Therapy is Allogeneic

Kristin Yarema, Ph.D.

President, Cell Therapy and Incoming CEO

Stem cell memory T cells (T_{SCM}) are an optimal cell type for CAR-T

LESS DIFFERENTIATED

MORE DIFFERENTIATED

T_{SCM} | T_{CM} | T_{EM} | T_{EFF}

Stem cell memory T cell

- Self-renewing
- Long lived
- Multipotent

Central memory T cell

Effector memory T cell

Effector T cell

- Non self-renewing
- Short lived
- Specialized - cytotoxic

STEMNESS MATTERS – Products with High % of T_{SCM} Cells Show:

- Strong correlation with best responses in the clinic
- More gradual tumor killing with less toxicity
- Better duration of response and potential for re-response – T_{SCM} engrafts and persists in bone marrow

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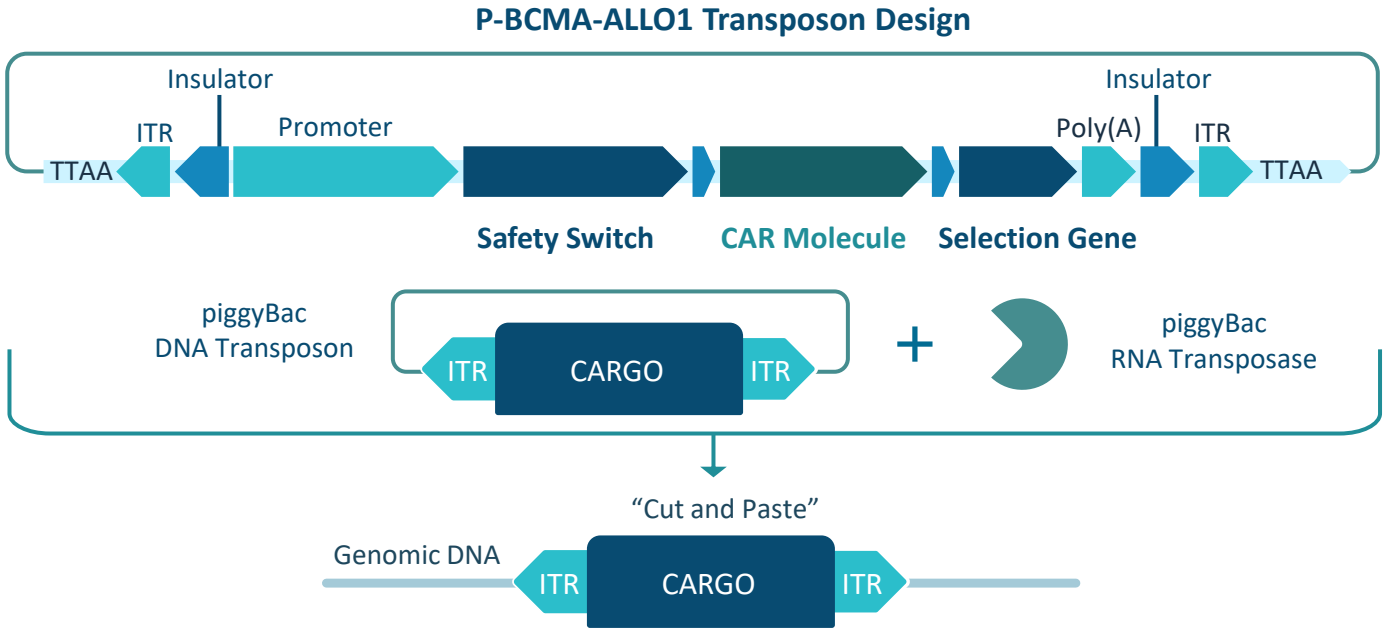
piggyBac® DNA Delivery System: proprietary non-viral approach to insert BCMA chimeric antigen receptor (CAR) into healthy donor T cells

Gene Insertion

piggyBac transposon system inserts transgene encoding BCMA-directed CAR

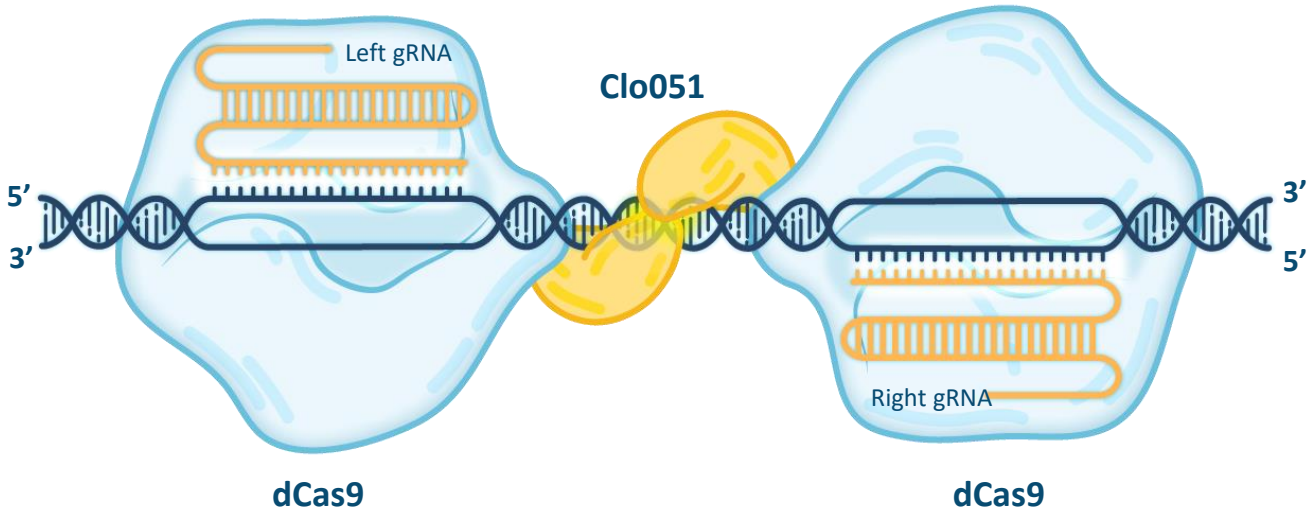


- Preferentially transposes naïve T cells resulting in T_{SCM} rich product
- Large cargo capacity delivers CAR, inducible safety switch, and selectable marker in single step

Cas-CLOVER™ Gene Editing System is used for gene editing of P-BCMA-ALLO1

Gene Editing

High fidelity gene editing system used to address graft vs. host and host vs. graft alloreactivity



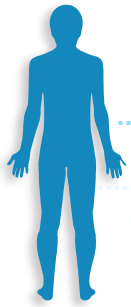
- High editing efficiency in resting T cells results in high % of T_{SCM}
- Low to no off target cutting
- Edits include TCR and B2M (MHC I) knockouts

Madison et al., Molecular Therapy – Nucleic Acids, 2022. (https://www.sciencedirect.com/science/article/pii/S216225312200155X)

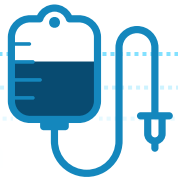
Poseida manufacturing platform for products including P-BCMA-ALLO1 is scalable and cost effective

High-yield Clinical Manufacturing

Allogeneic manufacturing process enhanced with Booster Molecule technology



HEALTHY DONOR



ONE LEUKOPAK

Manufacturing

T Cell Isolation

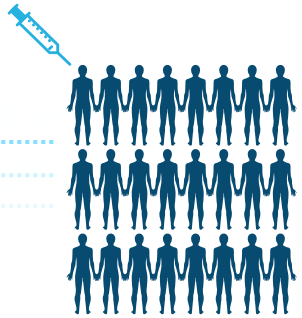
Non-viral Gene Editing

CAR-T Cell Selection and Expansion

Purification

Fill/finish

Storage in Inventory



~100 DOSES

- Production process preserves T_{SCM} phenotype
- Nearly all CAR-carrying cells
- “On demand” Delivery

P-BCMA-ALLO1 Phase I study data presented at ASH 2023 includes product from 6 manufacturing lots and 6 different qualified donors

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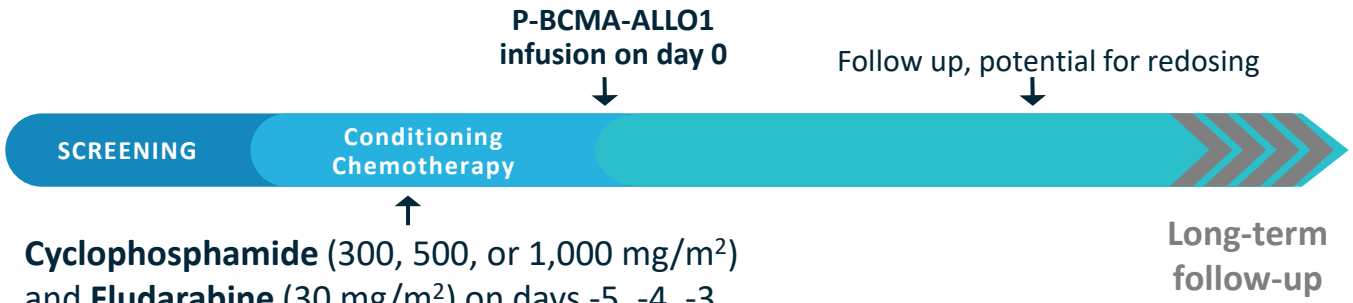
P-BCMA-ALLO1 Phase 1 Clinical Data Update

Rajesh Belani, M.D.

Vice President, Clinical Development

Study P-BCMA-ALLO1-001: open-label, multicenter, phase 1 study to assess the safety of P-BCMA-ALLO1 in patients with relapsed/refractory multiple myeloma



Key Inclusion Criteria:

- RRMM as defined by the IMWG
- Must have received PI, IMiDs & CD38 mAb or triple refractory
- ECOG 0 or 1

PRIMARY ENDPOINTS

- Assess safety and MTD based on DLT

SECONDARY OBJECTIVES

- Evaluate the anti-myeloma effect of P-BCMA-ALLO1
- Study effect of cell dose & LD regimen selection to guide dose selection for pivotal studies

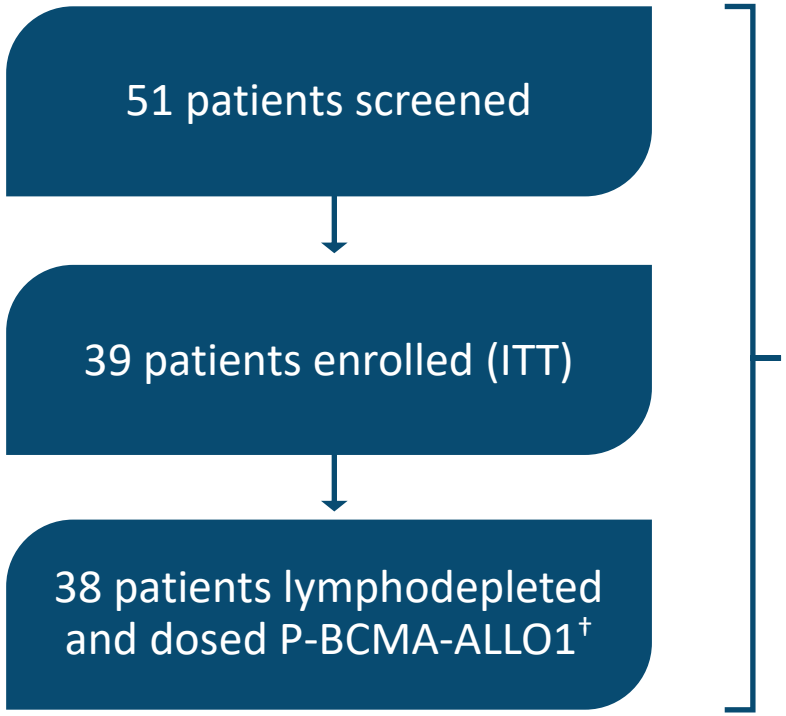
P-BCMA-ALLO1 doses and LD arms evaluated, and no. of patients infused at each cohort:

A. 0.25×10^6 cells/kg (n=1)
B. 0.75×10^6 (n=7)
C. 2×10^6 (n=9)
D. 6×10^6 (n=4)

Arm S (Cy 300); N = 21

E. 2×10^6 —— Arm P1 (Cy 500); N = 5
F. 2×10^6 —— Arm P2 (Cy 1,000); N = 6
G. 2×10^6 —— Arm C (Cy 300, CAR-T × 2); N=1
H. 2×10^6 —— Retreatment arm (Cy 300); N=1

Rapid and convenient CAR-T administration for entire intent-to-treat (ITT) population without need for apheresis



- 100% of ITT population underwent LD and received P-BCMA-ALLO1
  (1 patient had not begun LD by data cut)
- No patient required bridging therapy
- Median time from enrollment to:
  - Start of LD was 1 day*
  - P-BCMA-ALLO1 infusion was 7 days*

†Interim safety analysis on patients (n = 33) given an infusion of P-BCMA-ALLO1 (including cyclic arm patient) and with a minimum of 4 weeks follow-up. Data cutoff for safety and efficacy analysis was Oct. 23rd, 2023
*N=33, analysis excludes patient retreated with P-BCMA-ALLO1
ITT = intent-to-treat defined at enrollment; LD = lymphodepletion.

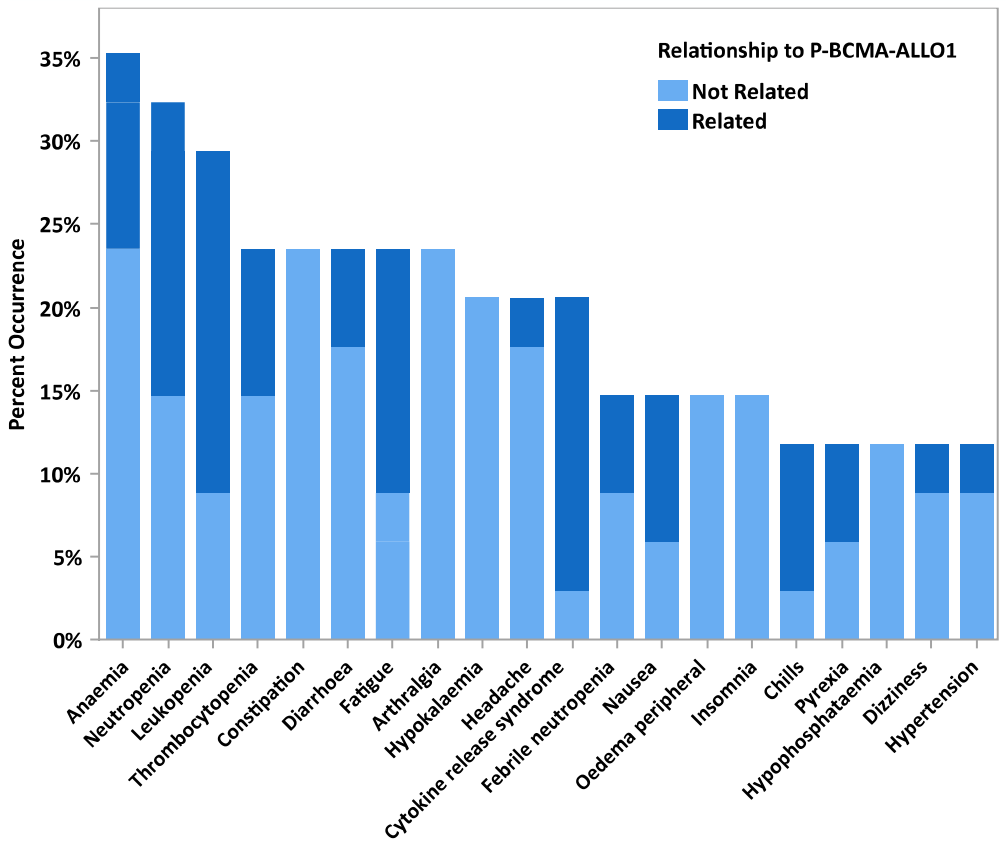
P-BCMA-ALLO1-001 study enrolled a heavily pretreated patient population

Characteristic	Number of Patients (total n = 33[†])
Median age (min, max) years	68 (33, 85)
Female n (%) / Male n (%)	21 (64) / 12 (36)
Median time (min, max) since diagnosis, years	6.27 (1.48, 18.95)
Myeloma Diagnosis Subtype, n (%)	IgG, 20 (61), IgA, 9 (27), IgD, 1 (3) Kappa FLC, 21 (64), Lambda FLC, 12 (36) Other: Abnormal CBC, 1 (3)
High-Risk cytogenetics , n (%)	10 (30)
Median prior lines of therapy (min, max), n	7 (2, 17)
Prior anti-BCMA CAR-T and anti-BCMA therapy, n (%)	13 (39)

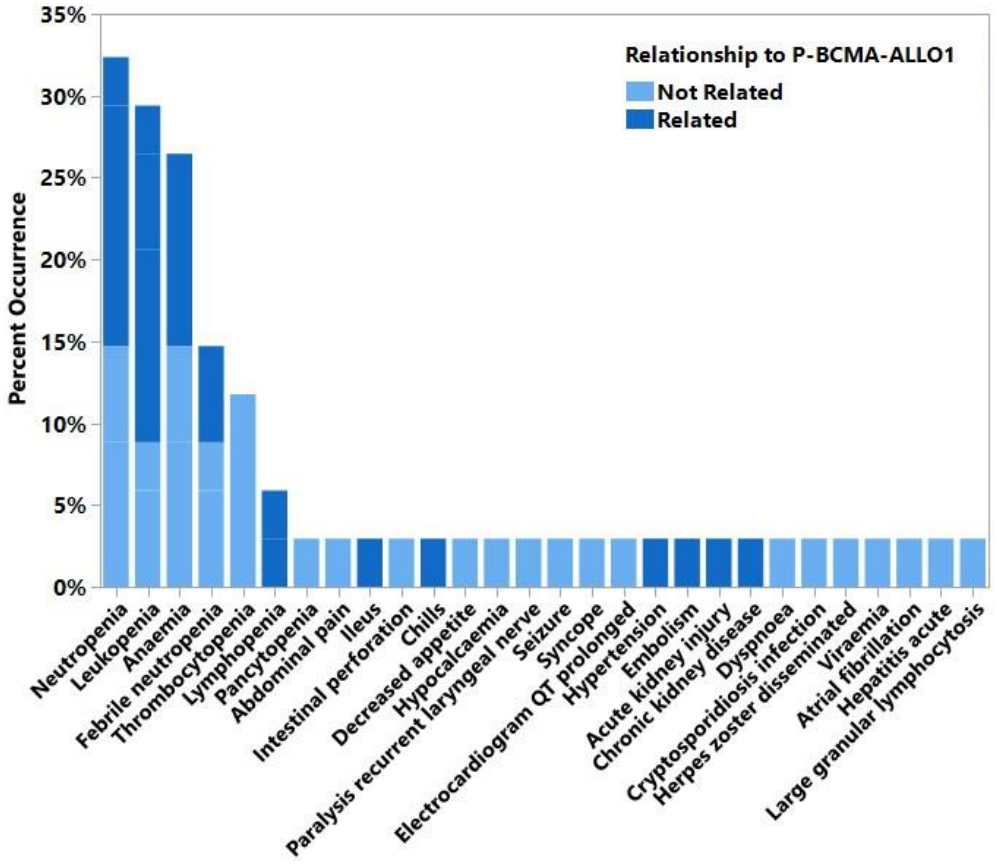
[†]Includes one patient non-evaluable for response assessment; re-treated patient is counted only once

P-BCMA-ALLO1 is well tolerated in RRMM patients

>10% Occurrence: All Toxicity Grades



All TEAEs ≥ 3



Safety Summary:

- Dose-levels through 6 × 10^6 cells/kg cleared with no DLTs
- No GvHD observed at any dose
- Grade ≥ 3 TEAEs were associated mainly with LD and myeloma
- Low CRS incidence (21%), Grade ≤ 2 in severity
- Neurotoxicity (Grade ≤ 2) observed in 2 patients (6%)
- Serious infections were uncommon even in the higher LD arms

P-BCMA-ALLO1 treated patient groups

Arm S (Single weight-based dose)
Cy 300 mg/m^2, flu 30 mg/m^2

- **Cohort -1** 0.25 x 10^6 — N=1
- **Cohort 1** 0.75 x 10^6 — N=7
- **Cohort 2** 2 x 10^6 — N=9[†]
- **Cohort 3** 6 x 10^6 — N=4

Arm C (P-BCMA-ALLO1 doses X 2)
Cy 300 mg/m^2, flu 30 mg/m^2

- **Cohort 2** **2 x 10^6** — N=1

Retreatment
Cy 300 mg/m^2, flu 30 mg/m^2

- **Cohort 2** **2 x 10^6** — N=1

Arm P1 (enhanced LD)
Cy 500 mg/m^2, flu 30 mg/m^2

- **Cohort 2** **2 x 10^6** — N=5[‡]

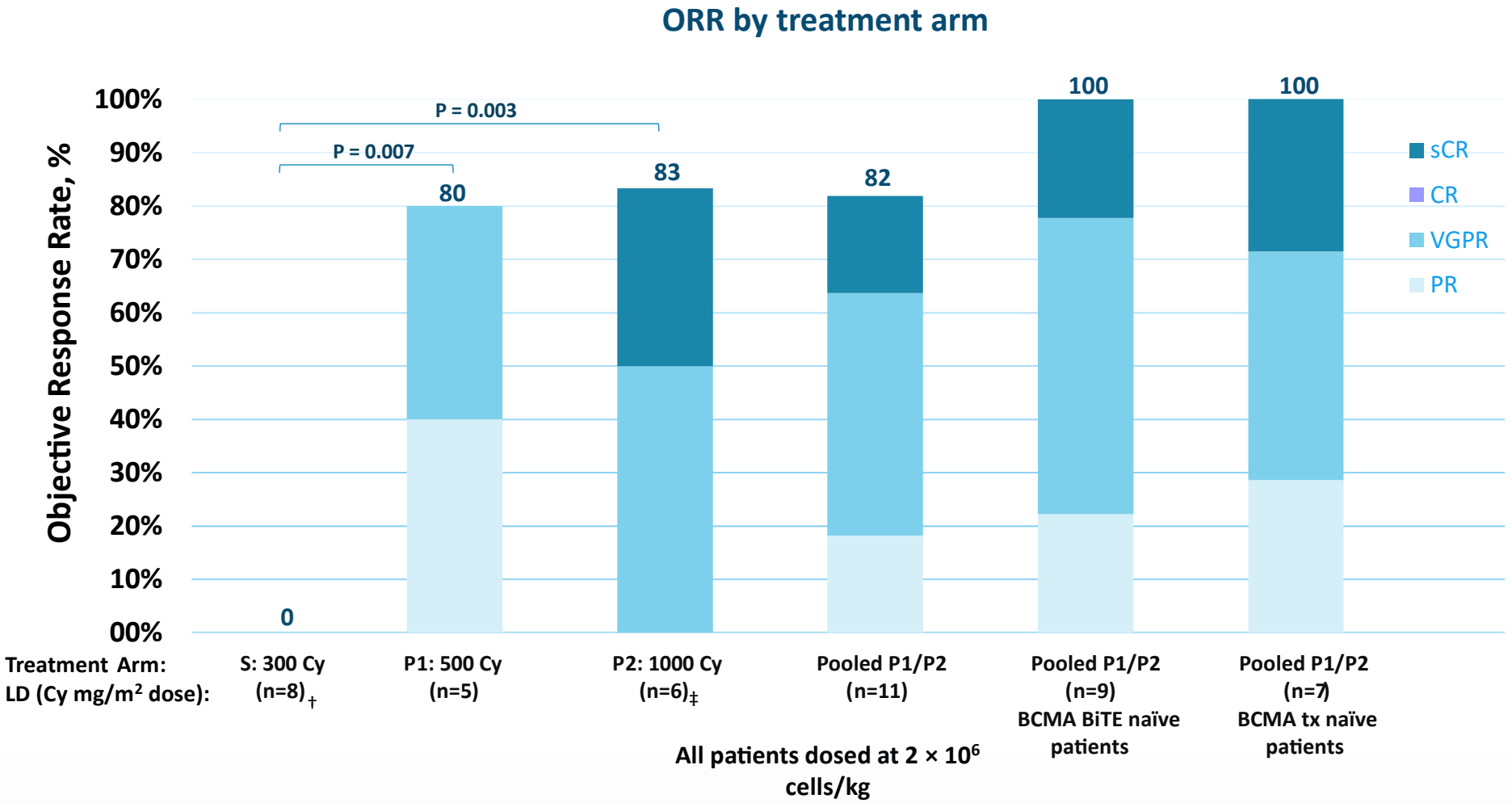
Arm P2 (enhanced LD)
Cy 1000 mg/m^2, flu 30 mg/m^2

- **Cohort 2** **2 x 10^6** — N=6[‡]

Cohort 2 at all three lymphodepletion regimens is focus of the efficacy analysis in the next slide

[†]one patient did not have measurable disease and was non-evaluable for response assessment.
[‡]one patient in P1 and one patient in P2 had received prior teclistamab.

Deep responses and a high response rate in BCMA naïve and prior BCMA therapy exposed RRMM patients receiving adequate lymphodepletion

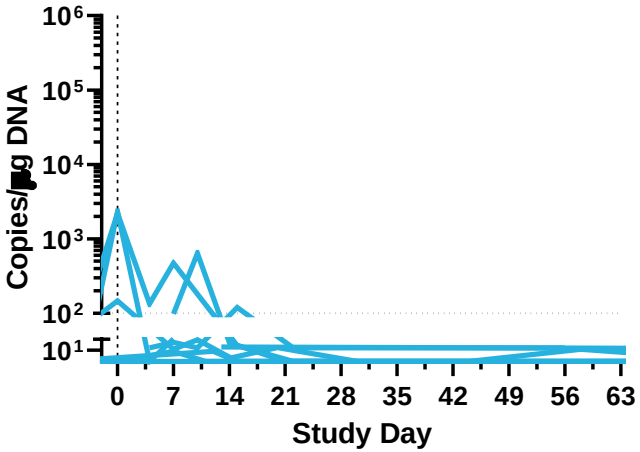


† Not included in total: one Arm S patient non-evaluable for response assessment and one Arm S patient that was retreated.
‡Two P2 patients who had received prior BCMA auto CAR-T (idecabtagene vicleucel and P-BCMA-101), both achieved VGPR.

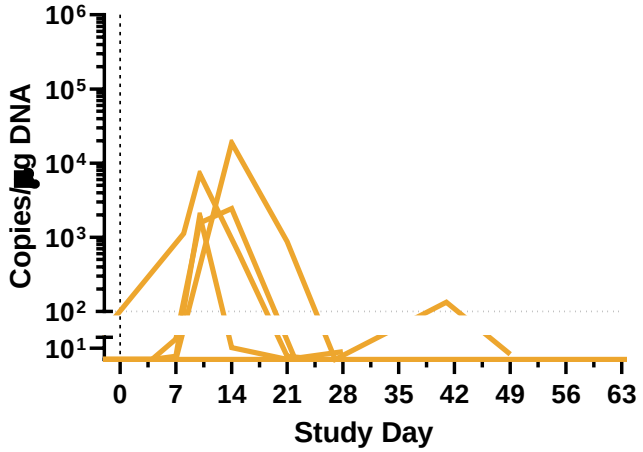
Higher cyclophosphamide LD doses markedly enhanced P-BCMA-ALLO1 expansion

A

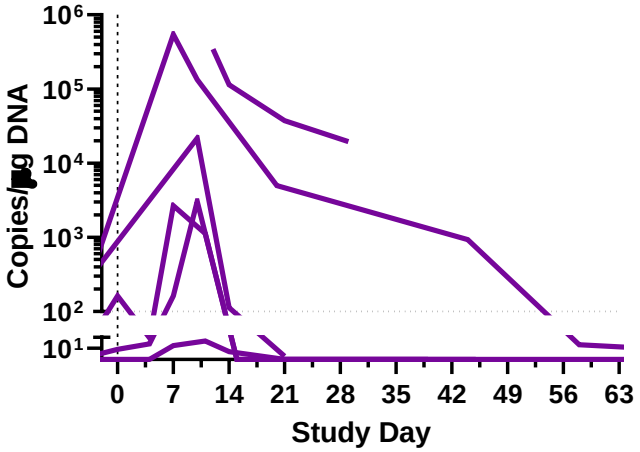
Arm S: 2×10^6 cells/kg
LD (Cy mg/m^2): 300



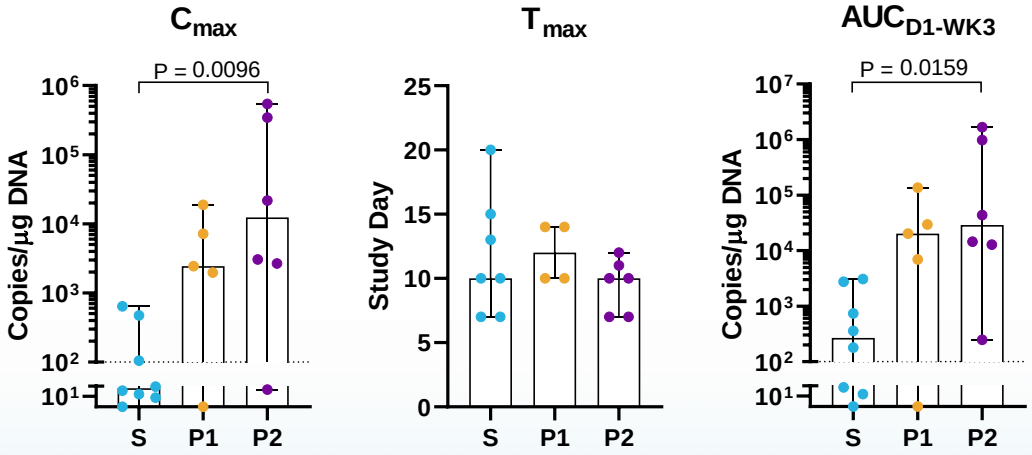
Arm P1: 2×10^6 cells/kg
LD (Cy mg/m^2): 500



Arm P2: 2×10^6 cells/kg
LD (Cy mg/m^2): 1000



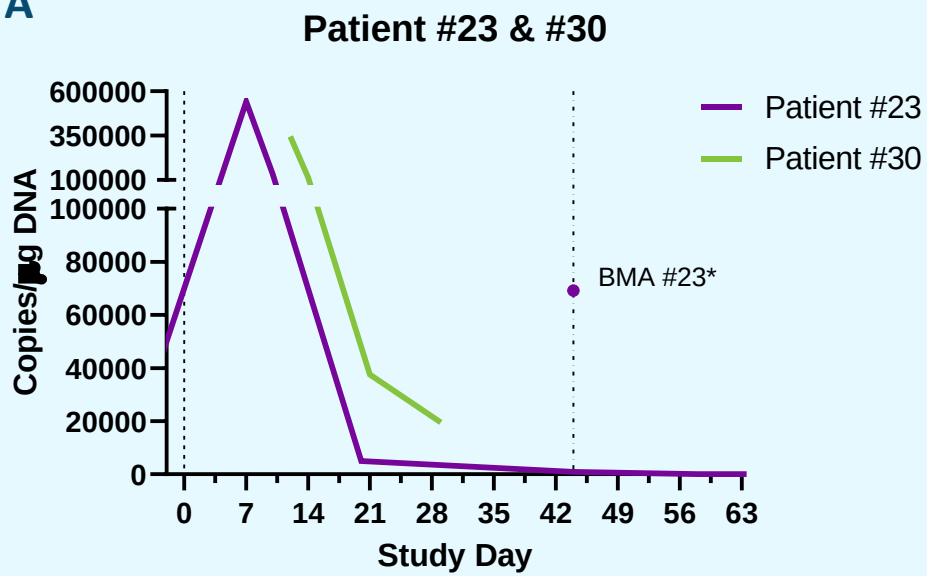
B



A) CK in Arm S, P1 and P2 at 2x10^6/kg cell dose and increasing Cy dose levels. Missing data points are due to insufficient DNA yield. **B)** C/T_{max} and AUC by cohort shown in A. Only n=7 (S) n=4 (P1) shown for T_{max}, since no signal >LOD for one subject per group. Kruskal-Wallis test with Dunn's multiple comparisons test; Median with range is shown. LOD of qPCR is 100 cp/µg.
CK = cellular kinetics; PBMC = peripheral blood mononuclear cells; cp/µg = transposon copies/µg of DNA.

P-BCMA-ALLO1 showed durable persistence in peripheral blood and bone marrow

A



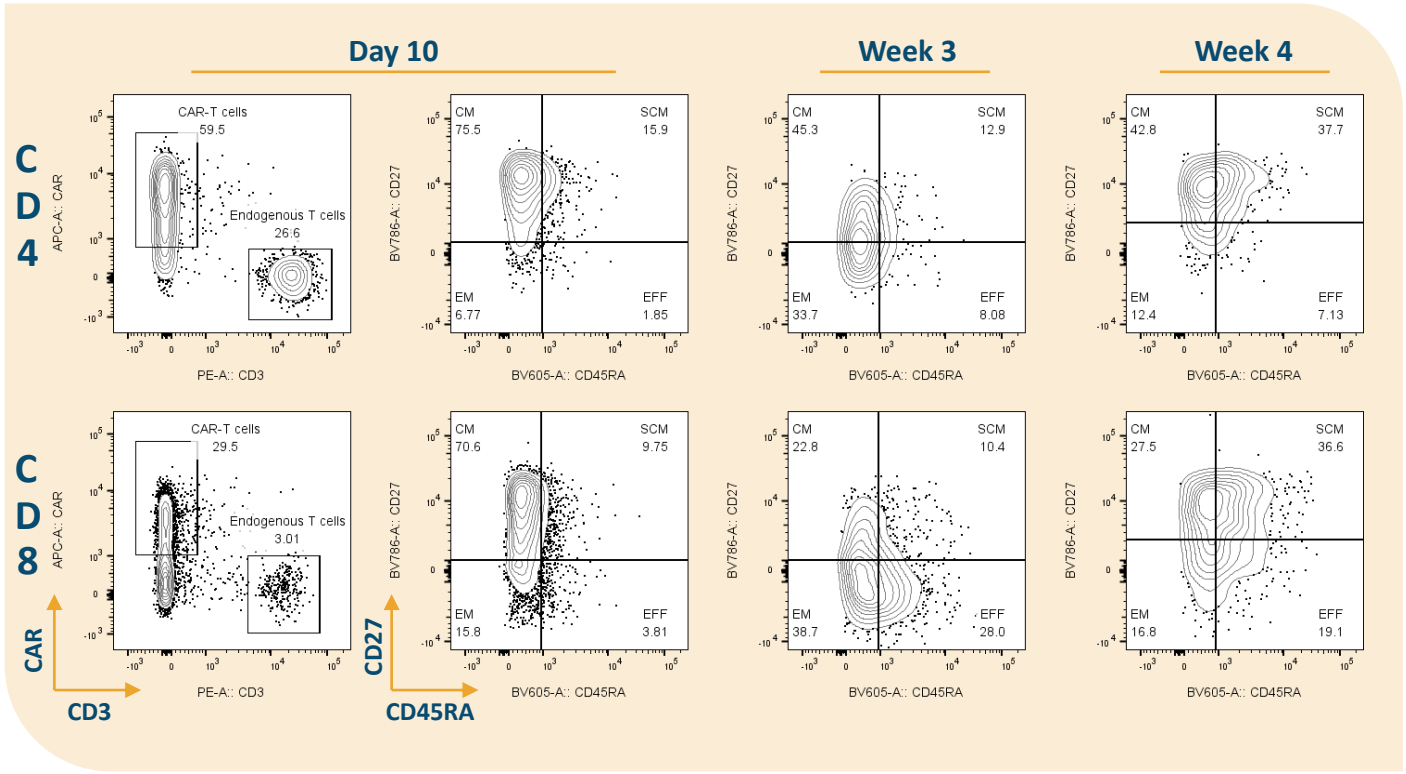
B

	Patient #30** (all PBMC)				Patient #23**	
	D10	WK2	WK3	WK4	WK6 (BMA)	WK6 (PBMC)
qPCR (cp/µg)	345,562	114,115	37,549	19,566	~69,000*	932
% CAR-T of all Lymphocytes	31.1	n/a	5.94	4.6	13.8	n/a

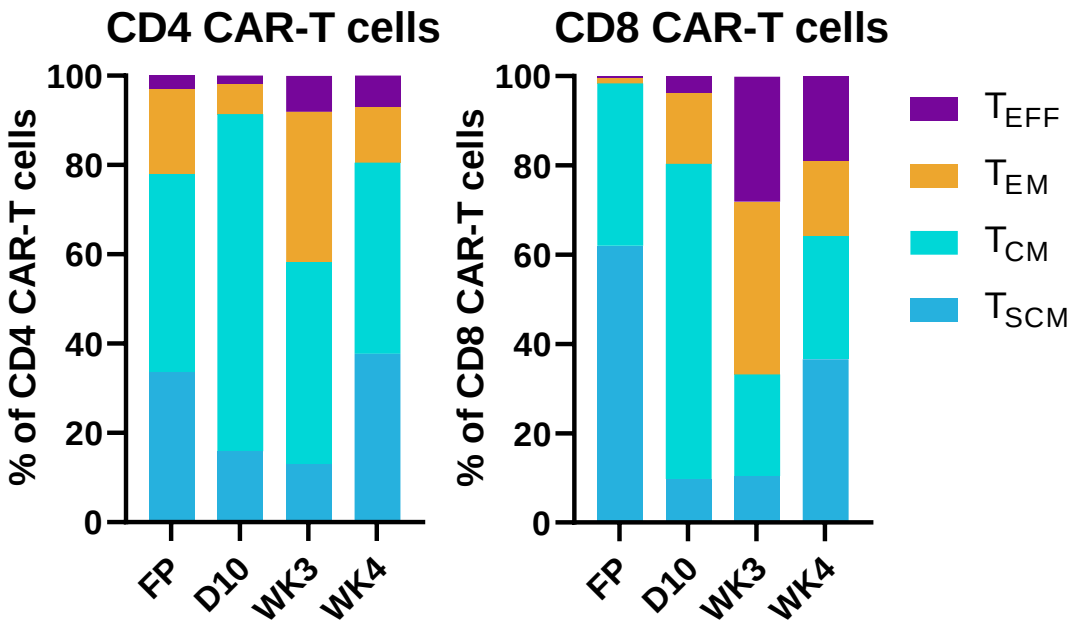
A) Cellular kinetics in patient #23 and #30 in peripheral blood (PBMC) and bone marrow aspirate (BMA) for subject #23. **B)** qPCR signal compared to frequency of CAR-T cells identified by Flow. Missing data points due to insufficient DNA yield. BM = bone marrow. BMA = bone marrow aspirate. BMMC = bone marrow mononuclear cells.
*qPCR was performed on DNA derived from BMA. The standard curve efficiency was 86%, which is below our acceptance criteria (≥90%); consequently, the result was expressed as an approximation. 'n/a': not assayed due to insufficient PBMC counts for FACS and qPCR assays.
**MM response achieved: sCR (MRD-) for patient #23 and VGPR (patient #30)

Peripheral blood samples of patient #30 demonstrate P-BCMA-ALLO1 T_{SCM} cells differentiation to T_{EFF} phenotype

A

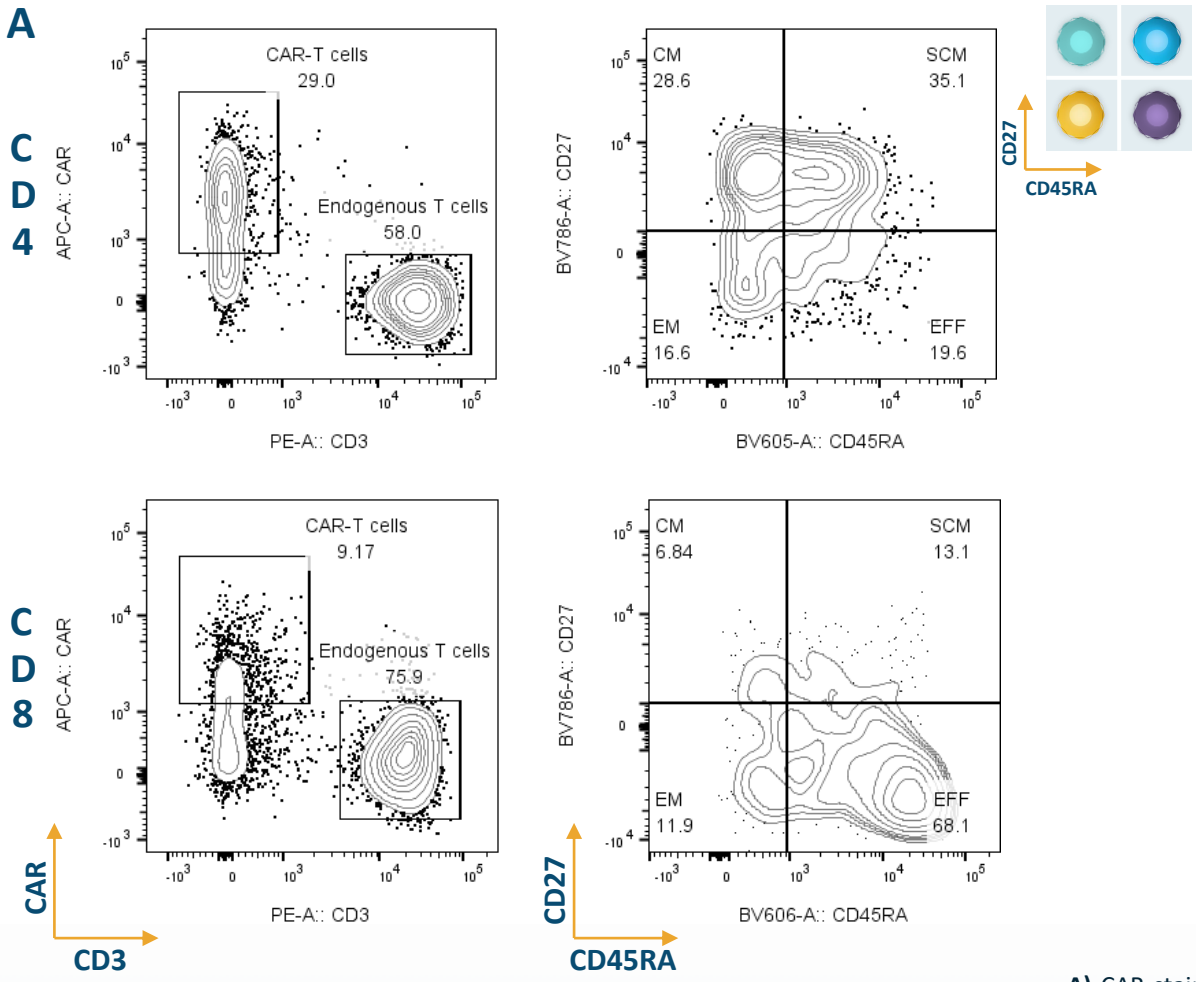


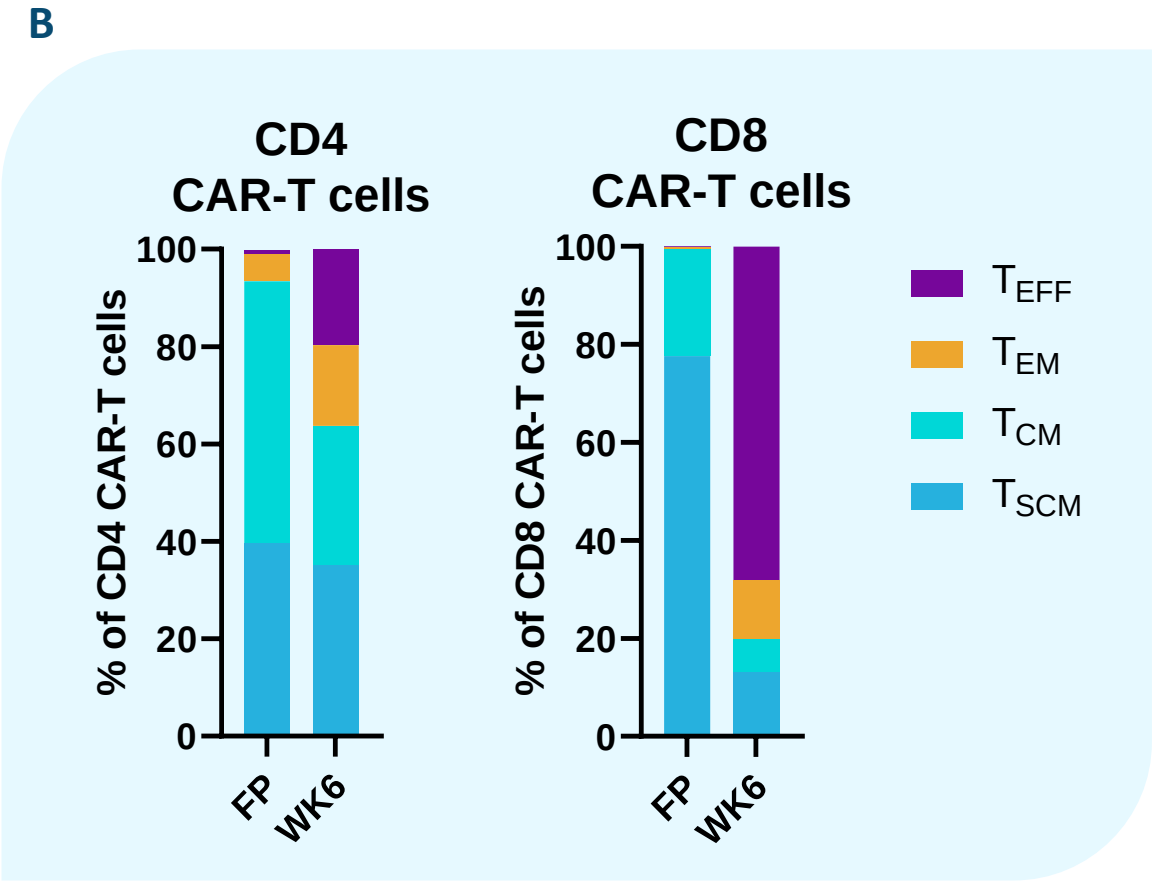
B



A) Phenotype of CAR-T cells (% CD3-BCMA+ of live, single cells, CD45+CD2+, CD4+ or CD8+; CAR-T identified with BCMA protein) in peripheral blood by Flow over time (Day 10, Week 3 and Week 4) in patient #30 based on CD45RA and CD27 expression. Representative CAR staining shown for D10; endogenous T cells are defined as BCMA-CD3+**.** Top row CD4 CAR-T cells, bottom row CD8 CAR-T cells. **B)** Final drug product (FP) phenotype for patient #30 in comparison to CAR-T phenotype by % T_{SCM}, T_{CM}, T_{EM} and T_{EFF} of CD4 and CD8 CAR-T cells (as shown in quadrant gates) in PBMC samples at indicated timepoints.

T_{SCM}-rich P-BCMA-ALLO1 cells traffic to bone marrow and differentiate to T_{EFF} as observed on day 44 marrow aspirate in patient #23





A) CAR staining and CAR-T phenotype of bone marrow mononuclear cells (BMMC) for patient #23 at Week 6 (Day 44) post-infusion. Top row CD4+ CAR-T cells, bottom row CD8+ CAR-T cells. **B)** Final drug product (FP) phenotype for patient #23 in comparison to CAR-T phenotype by % T_{SCM}, T_{CM}, T_{EM} and T_{EFF} of CD4 and CD8 CAR-T cells (as shown in quadrant gates) in bone marrow specimen at Week 6. Gating strategy of BCMA-CAR-T cells and CAR-T sub-populations (described in previous slide)

Closing Remarks

Kristin Yarema, Ph.D.

President, Cell Therapy and Incoming CEO

P-BCMA-ALLO1 is a promising “off-the-shelf” T_{SCM}-rich allogeneic CAR-T therapy based upon preliminary phase 1 results

- **Rapid, accessible treatment to meet urgent patient needs**
  - 100% treatment of the ITT population with in-spec product and no bridging therapy
  - Median “brain-to-vein” time (enrollment to infusion) of 7 days, including lymphodepletion
- **Favorable emerging safety profile**
  - No GvHD or DLT and low rates of CRS, neurotoxicity all Gr ≤ 2
- **Deep clinical responses in heavily pretreated patients receiving adequate lymphodepletion**
  - 82% overall ORR in pooled P1/P2 cohorts
  - 100% ORR in all P1/P2 patients who had received any prior anti-myeloma treatment, including auto BCMA CAR T, other than teclistamab
  - sCR rate 40% (2/5 pts) in P2 cohort responders
  - Some P1, P2 patients achieving MRD- status, including patients those with high-risk cytogenetics, extra medullary disease
- **Prodrug-like P-BCMA-ALLO1 cells demonstrate expansion, trafficking to site of malignancy, differentiation, and persistence**
- **Further clinical development of P-BCMA-ALLO1 is ongoing**

Thank you

We appreciate all contributors including our patients, team, investigators and Roche