



Investor Presentation

October 2026

Adicet Bio

Forward-Looking Statements

This presentation contains “forward-looking statements” of Adicet within the meaning of the Private Securities Litigation Reform Act of 1995 relating to the business and operations of Adicet. The words “anticipate,” “believe,” “continue,” “could,” “estimate,” “expect,” “intend,” “may,” “plan,” “potential,” “predict,” “project,” “should,” “target,” “would” and similar expressions are intended to identify forward-looking statements, although not all forward-looking statements contain these identifying words. These forward-looking statements include, but are not limited to, express or implied statements regarding: clinical development of Adicet’s product candidates, including future plans or expectations for prula-cel in autoimmune diseases and the potential safety, tolerability and efficacy for the treatment of autoimmune diseases and cancer as well as expectations to achieve a complete immune reset; expectations regarding future alignment with the FDA on regulatory path to approval and discussions to date; timing and success of the Phase 1 clinical trial of prula-cel in multiple autoimmune indications, including timing and expectations for enrollment and future data releases; expectations regarding the timing and initiation of a pivotal study for prula-cel in SLE patients with or without LN and potential expansion to include non-renal lupus; expectations regarding prula-cel’s potential to transform treatment for patients with lupus; expectations regarding the suitability of prula-cel for outpatient administration; expectations regarding the scalability of the Company’s off-the-shelf manufacturing platform; expectations regarding the pace of enrollment in the pivotal study for prula-cel in SLE patients with or without LN; expectations regarding prula-cel’s competitive positioning in lupus with and without nephritis; and estimates for the addressable patient population and commercial opportunity for prula-cel in LN and SLE. Any forward-looking statements in this presentation are based on management’s current expectations and beliefs of future events, and are subject to a number of 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, including without limitation, the effect of global economic conditions and public health emergencies on Adicet’s business and financial results, including with respect to disruptions to our preclinical and clinical studies, business operations, employee hiring and retention, and ability to raise additional capital; Adicet’s ability to execute on its strategy including obtaining the requisite regulatory approvals on the expected timeline, if at all; that positive results, including interim results, from a preclinical or clinical study may not necessarily be predictive of the results of future or ongoing studies; clinical studies may fail to demonstrate adequate safety and efficacy of Adicet’s product candidates, which would prevent, delay, or limit the scope of regulatory approval and commercialization; and regulatory approval processes of the U.S. Food and Drug Administration and comparable foreign regulatory authorities are lengthy, time-consuming, and inherently unpredictable; and Adicet’s ability to meet production and product release expectations. For a discussion of these and other risks and uncertainties, and other important factors, any of which could cause Adicet’s actual results to differ from those contained in the forward-looking statements, see the section titled “Risk Factors” in Adicet’s most recent annual report on Form 10-K, as well as discussions of potential risks, uncertainties, and other important factors in Adicet’s other filings with the U.S. Securities and Exchange Commission, including its quarterly report on Form 10-Q. All information in this presentation is as of the date of the release, and Adicet undertakes no duty to update this information unless required by law.

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Developing Broad Pipeline of Allogeneic $\gamma\delta 1$ CAR-T Cell and Best In Class Differentiated In Vivo CAR-T Therapies

► PRULA-CEL

Autoimmune Disease: LN/SLE/SSc

54%

12-month DORIS rate

50%

12-month CRR rate

- **Well tolerated** with no IEC-HS, no ICANS and no Grade >2 CRS; FDA-aligned outpatient administration
- **Single off-the-shelf dose resulted in meaningful clinical improvements**; all patients **discontinued immunosuppressants** and all but one patient **tapered background steroids***
- **Demonstrated immune reset**
- **Aligned with FDA** on single-arm LN pivotal Q4/2026; Potential to expand pivotal to include non-renal lupus
- **SSc data expected 1H/2026**

► ADI-212

Next-generation gene-edited & armored anti-PSMA $\gamma\delta 1$ CAR-T for mCRPC

- **Armored to enhance potency** and deliver multiple anti-tumor mechanisms
- IND cleared; **Plan to initiate enrollment 4Q/2026**

► IN VIVO CAR-T PIPELINE

Engineered to deliver highly efficient & selective T-cell transduction and deliver next-generation payloads

- **Cell specificity via novel homing and VSV-G fusagen technology**
- **Differentiated CAR design, and decreased susceptibility to immune mediated inactivation**

Developing Broad Pipeline of Allogeneic $\gamma\delta 1$ CAR-T Cell and Best In Class Differentiated In Vivo CAR-T Therapies

Off-The-Shelf $\gamma\delta 1$ CAR-T

Program	Indication	Research	IND-Enabling	Clinical	Potential Key Differentiation
Prula-cel Target: CD20	LN/SLE				- Off-the-shelf, outpatient dosing — no leukapheresis or personalized manufacturing - Quick, easy administration - Favorable safety profile; reduced IEC-HS, ICANS, CRS risk - Efficacy comparable to autologous CAR-T
	SSc				
ADI-212 Target: PSMA (gene-edited w/ armor)	mCRPC				- Validated target (PSMA) - Armoring enhances tumor-cell killing - Improved anti-tumor activity in the tumor microenvironment

In Vivo CAR-T – Cell-specific delivery via novel homing and VSV-G fusogen technology

Program	Indication	Research	IND-Enabling	Clinical	Potential Key Differentiation
Multiple Myeloma Target: BCMA Mono / Dual CAR	MM	PRE-CLINICAL			- Differentiated CAR design with effective BCMA targeting - Decreased susceptibility to immune-mediated inactivation and clearance
NHL Target: CD19 Dual CAR	NHL	PRE-CLINICAL			Differentiated dual-CAR design
Solid Tumors Target: Not disclosed	NA	PRE-CLINICAL			Multiple armoring technologies designed to enhance efficacy in solid tumors

AI= autoimmune; BCMA= B-cell maturation antigen; CRS= Cytokine release syndrome; ICANS= Immune effector cell-associated neurotoxicity syndrome; LN= Lupus nephritis; mCRPC= Metastatic castration-resistant prostate cancer; MM= multiple myeloma; NA=Not disclosed; NHL= Non-Hodgkin lymphoma; PSMA= Prostate specific membrane antigen; SLE= Systemic lupus erythematosus; SSc= Systemic sclerosis; Timing subject to site activation, patient enrollment, data readouts and regulatory feedback; Cut-off date: August 28, 2026; Data cut included 22 efficacy-evaluable patients

Anticipated Near-Term Value-Creating Milestones

Clinical, regulatory and platform catalysts expected across prula-cel, ADI-212 and in vivo CAR-T programs

	2026	2027	2028	2029
Prula-cel Autoimmune disease LN / SLE / SSc	LN/SLE clinical update LN/SLE Pivotal start-up activities Potential SLE expansion*	SSc clinical update* LN/SLE clinical update*	LN/SLE Interim pivotal data*	LN/SLE Potential pivotal data*
	Progression toward registrational pathway →			
ADI-212 mCRPC	IND Clearance mCRPC Enrollment	mCRPC Clinical update*	mCRPC Clinical update*	
In Vivo CAR-T MM Program	Platform/program update	MM Initiate clinical study*	MM Clinical Update*	
In Vivo CAR-T NHL Program	Platform/program update		Potential clinical data*	
In Vivo CAR-T Solid Tumors Program	Platform/program update		Potential clinical data*	

Adicet Bio Leadership Team



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Nick Harvey
Chief Financial Officer



Lloyd Klickstein, M.D., Ph.D
Interim Chief Medical Officer



Prula-cel

Autoimmune Indications



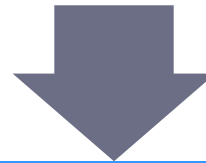
Major Unmet Needs in LN/SLE Remain

Limited disease control with existing therapies

- **Treatment-free remissions are rare**
- **Flares are common** despite chronic therapy, reflecting ongoing disease activity
- **Increased early mortality** due to organ damage, cardiovascular disease, infections, renal disease and other causes

Significant side effects associated with chronic therapies

- **Current chronic therapy with high dose corticosteroids and immunosuppressants associated with serious side effects** - infections, bone fractures and diabetes
- Disease and chronic therapies **negatively impact patients' QoL** - fatigue, emotional problems, rash, pain and ability to work



Need for one-time therapy with favorable safety profile that can deliver treatment-free remissions

Prula-cel: Potential to Transform the SLE and LN Patient Journey

Potential for Strong Adoption

- ✓ One time treatment approach
- ✓ Off-the-shelf
- ✓ Favorable safety profile
- ✓ Potential for outpatient therapy and accessibility in non-academic medical centers
- ✓ Potential for durable immunosuppressant-free clinical benefit
- ✓ Reduced steroid burden

Potentially shifting treatment paradigm to “treat once and monitor patient”

Prula-cel Delivered High Rates of Immunosuppression-Free CRRs and DORIS Remissions in Heavily Pre-Treated LN & SLE Patients

50%

12-Month CRR Rate

54%

12-Month DORIS Rate



Favorable Safety Profile

Generally well-tolerated — No IEC-HS,
No ICANS and No > Grade 2 CRS observed



Immunosuppressant-Free

All patients discontinued immunosuppressants



Steroid Taper Achieved

All but one patient tapered background steroids
to ≤5 mg prednisone equivalent

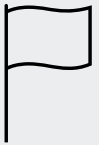


Demonstrated Immune Reset

Complete CD19+ B cell depletion followed by
naïve B cells recovery

Prula-cel Has Established Path to Potential Approval and Large Commercial Opportunity

Pivotal Readiness



Established Pivotal Trial Design

- Aligned with FDA on single arm pivotal study in LN
- Potential to expand pivotal to include non-renal lupus based on regulatory precedent

Compelling Commercial Opportunity



Potential One-Time, Off-the-Shelf, Outpatient Therapy

- Aligned with FDA to make prula-cel available for enrollment in outpatient setting
- Scalable Manufacturing



Strong Enrollment Execution

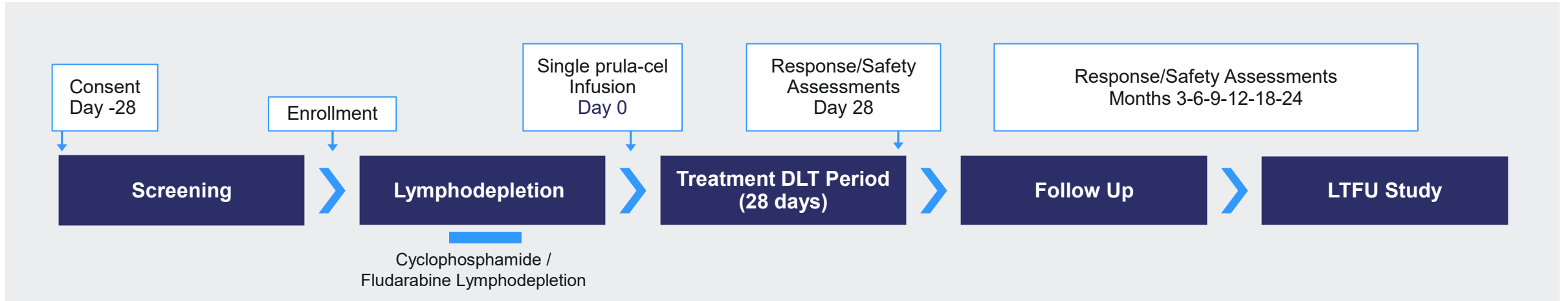
- Significant interest from investigators and patients to enroll



Significant Addressable Market

- 35,000 organ/life threatening LN¹ (US)
- 35,000 organ/life threatening non-renal SLE² (US)

Prula-cel Phase 1 Autoimmune Study Design



Part 1: Advancing study in multiple cohorts



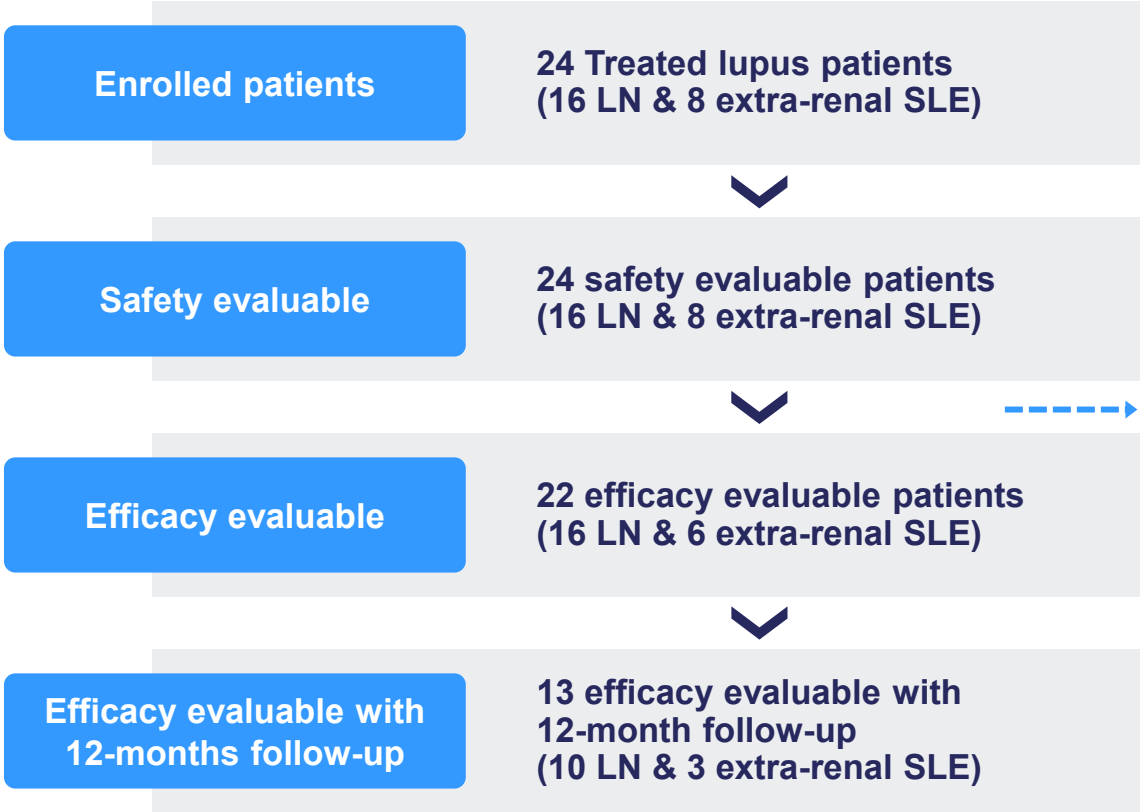
Part 2

Dose Expansion Cohorts

Prula-cel SLE/LN Patient Characteristics

Patient Characteristics

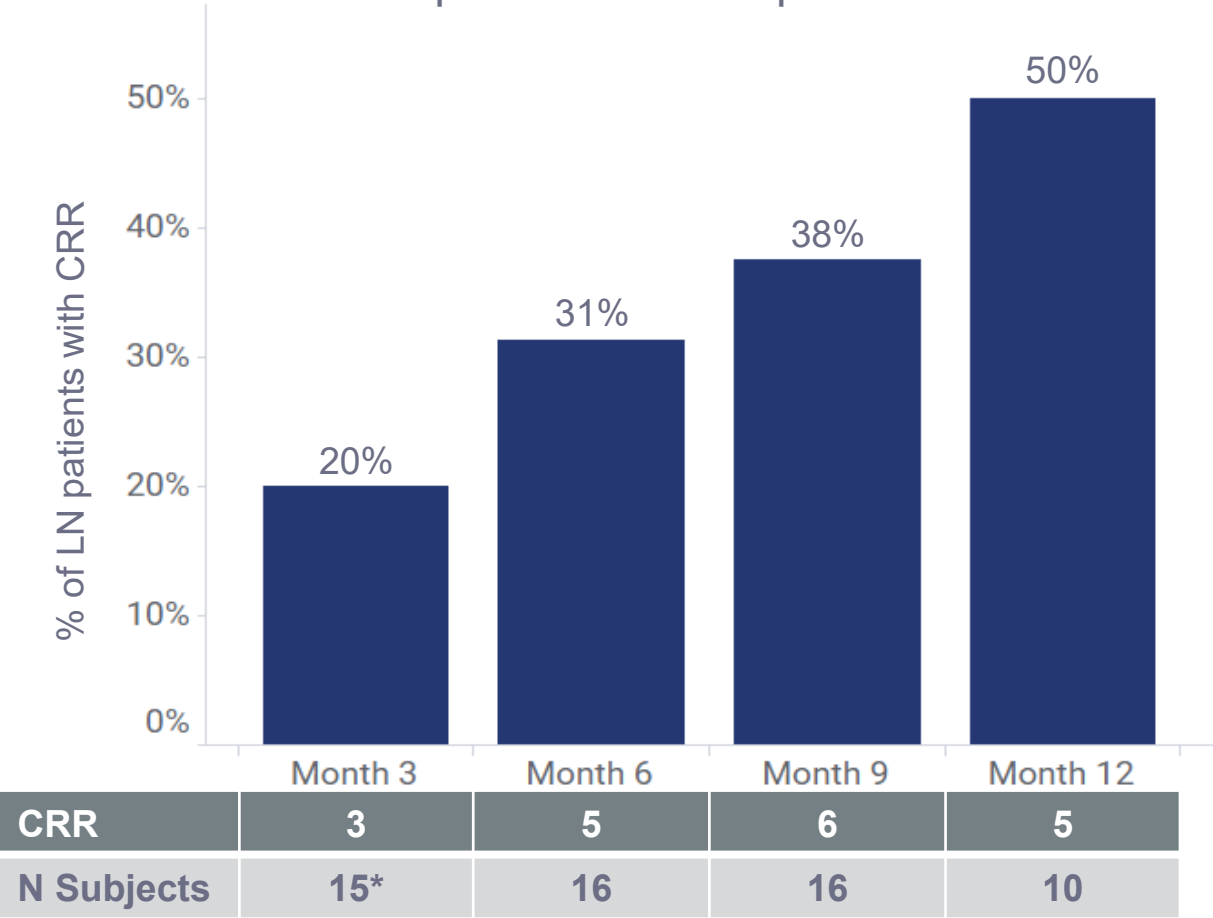
Demographics	
Age (mean yrs)	34.8
Female (%)	87.5%
Duration Since SLE Diagnosis (mean yrs)	7.2
Disease Activity	Mean
Baseline SLEDAI ¹	13
Baseline UPCR ²	2.8
Number of Prior Therapies	N (%)
2 or more	24 (100%)
3 or more	24 (100%)
4 or more	17 (71%)



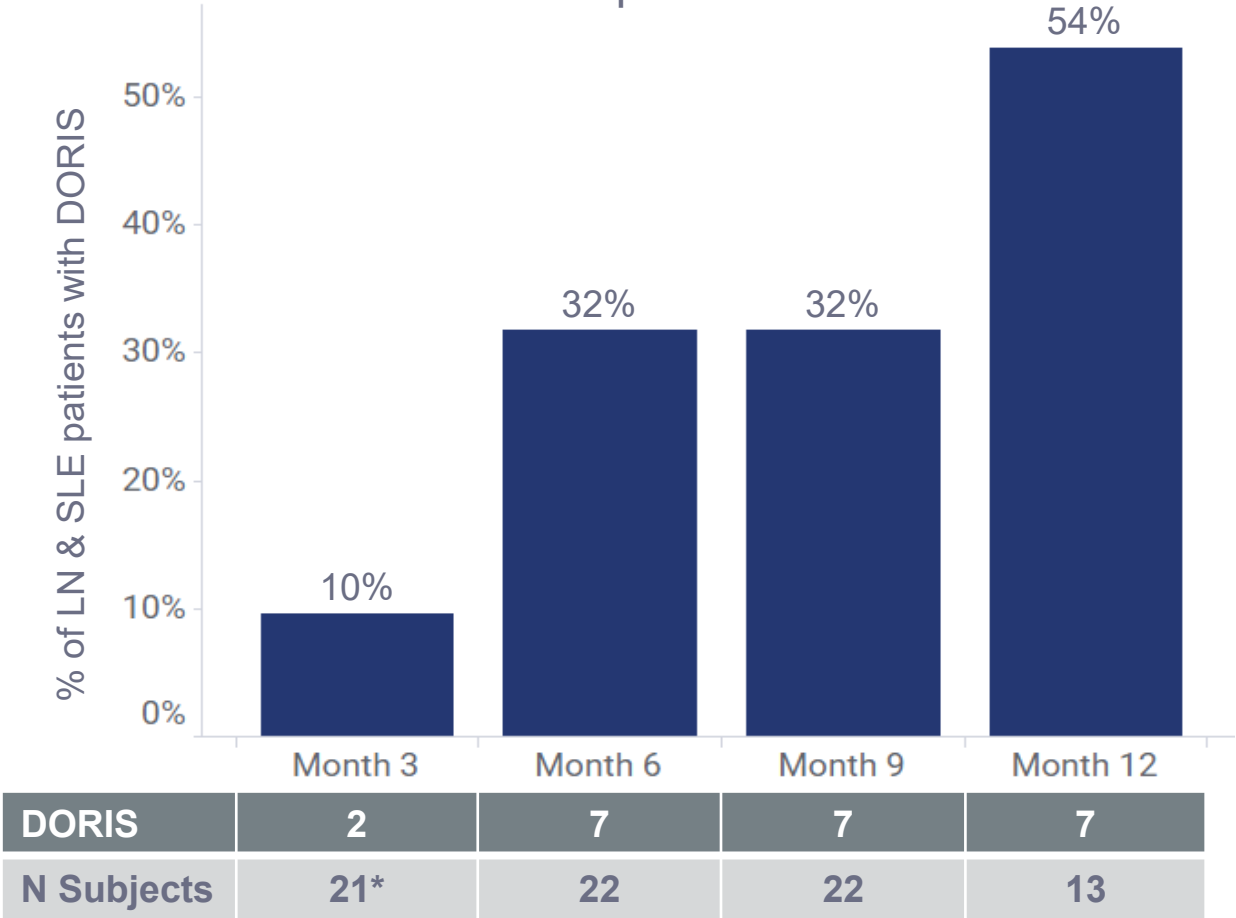
* One patient with Inclusion Body Myositis (excluded per protocol)
One patient with hypersensitivity reaction was not provided the prula-cel dose

High Rates of Both Complete Renal Response & DORIS Remissions Achieved By 12 Months Post Treatment With Prula-cel

Complete Renal Response in LN



DORIS Response in LN & SLE



All 12-mo CRRs and DORIS remissions ongoing (12-21 mo. follow up), except 1 pt with UPCr 0.65 g/g off immunosuppressants

CRR = UPCr ≤0.5 AND EITHER eGFR ≥60 mL/min/1.73m² OR no confirmed decrease from baseline in eGFR of >15% and no treatment or disease related eGFR-associated event; *One subject’s M3 UPCr was not evaluable; Cut-off date: August 28, 2026; Data cut included 22 efficacy-evaluable patients

DORIS Remission= Clinical SLEDAI (irrespective of serology) = 0 AND Physician global assessment score < 0.5; the subject may be on antimalarials, low-dose glucocorticoids (prednisolone ≤ 5 mg/day), and/or stable immunosuppressives including biologics; An additional 20% of the LN patients had at least a 50% decrease in proteinuria from their baseline levels at mo. 12, which is defined as partial renal response per protocol.

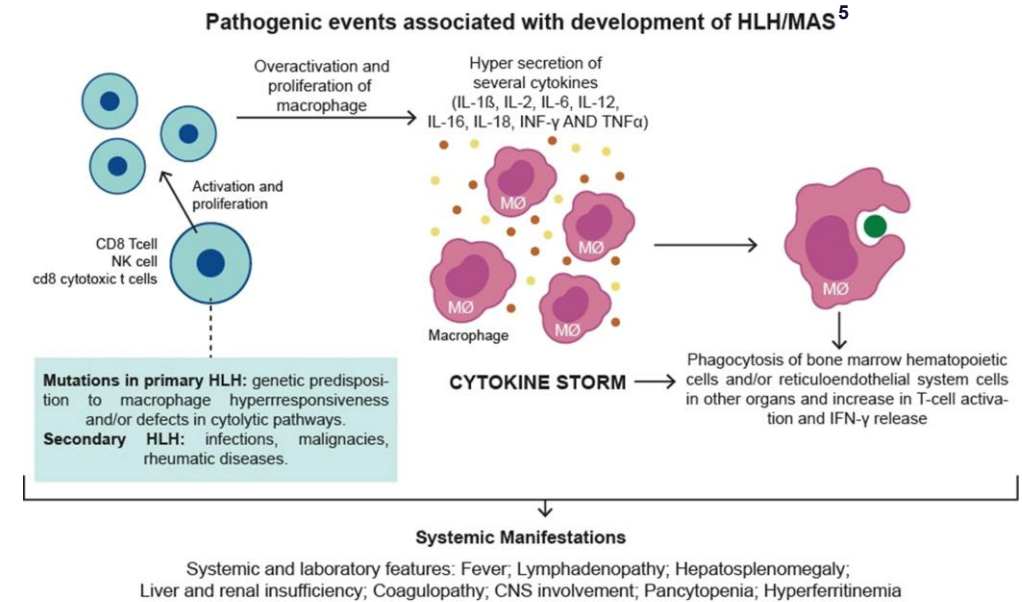
Generally Well-Tolerated Safety Profile: No DLT Observed, No IEC-HS, No ICANS, No Gr >2 CRS

Adverse events of interest	Prulacabtagene leucel (prula-cel) <i>LN/SLE, all dose levels</i>	Rapcabtagene autoleucel (rap-cel) ¹	Zolacabtagene autoleucel (zola-cel) ²	Obecabtagene autoleucel (obe-cel) ³
No. of Patients (n)	24	21	26	6
CRS (any / Gr 3+) (%)	25% / —	57% / --	77% / 4%	50% / --
ICANS (any Gr) (%)	--	5%	4%	--
Infections (any / Gr3+) (%)	13 (54%) / 2 (8.3%)	71% / 10%	15% any grade (Gr3+ rate not disclosed)	100% / 33%

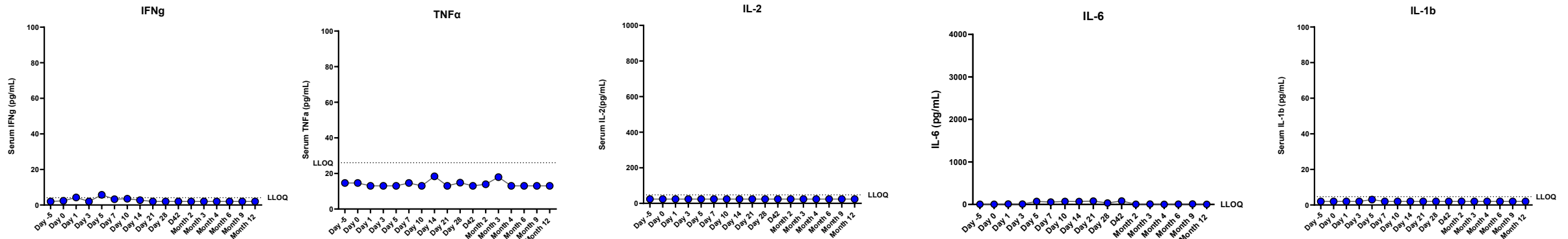
- Favorable safety profile with No IEC-HS, No ICANS and No Gr >2 CRS consistent across all autoimmune indications (48 patients dosed to date)
- Per alignment with FDA, prula-cel may be administered to SLE and LN patients in the outpatient setting
- $\gamma\delta$ CAR-T are generally well tolerated compared to $\alpha\beta$ CAR-T, likely due to different cytokine profile secreted upon activation as compared $\alpha\beta$ CAR-T

Prula-cel's Favorable Safety Profile in Autoimmune Compared to a Autologous $\alpha\beta$ CAR-T is Rooted in the Biology of $\gamma\delta 1$ T Cells

- $\gamma\delta 1$ T cells are defined by a clearly differentiated cytokine profile compared to $\alpha\beta$ T cells¹⁻⁴
- IEC-HS is primarily related to hyperproliferation of $\alpha\beta$ CAR-T and associated with secretion of IFN γ , TNF α , IL-2, IL6, and IL18⁵
- Prula-cel did not lead to meaningful increase in these systemic markers in SLE/ LN patients



Systemic Cytokine Values for SLE/LN Patients Receiving Prula-Cel



1. Nishimoto, K.P., *et al* (2022), Allogeneic CD20-targeted $\gamma\delta$ T cells exhibit innate and adaptive antitumor activities in preclinical B-cell lymphoma models. Clin Transl Immunol, 11: e1373.

2. Nishimoto KP *et al*. ADI- 270: an armored allogeneic gamma delta T cell therapy designed to target CD70- expressing solid and hematologic malignancies. Journal for ImmunoTherapy of Cancer 2025;13:e011704

3. Herrman, M. Aftab, B, *et al*. presented at: Prostate Cancer Foundation Scientific Retreat; September 2026.

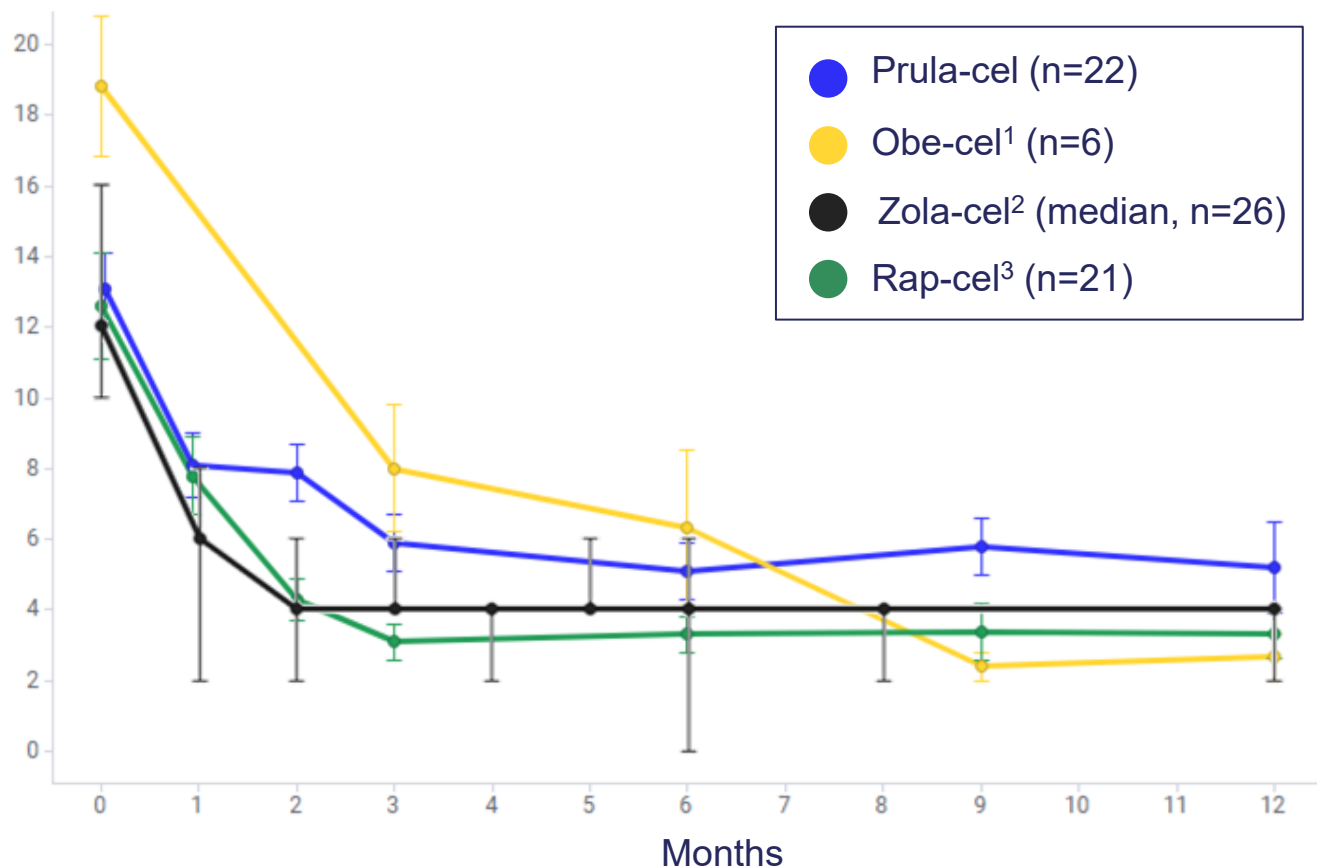
4. Perez XB. CAR Signaling Informs Mechanisms to Enhance Metabolism and Function in $\gamma\delta$ T Cells. Res Sq [Preprint]. 2026

5. Sztajnbnok, F., *et al*. Hemophagocytic lymphohistiocytosis and macrophage activation syndrome: two rare sides of the same devastating coin. Adv Rheumatol 64, 28 (2024).

Cut-off date: August 28, 2026; Data cut included 22 efficacy-evaluable and 24 safety-evaluable patients; No head-to-head studies have been conducted comparing autologous $\alpha\beta$ CAR-T to prula-cel.

SLEDAI Decline With Prula-cel In-Line With That Observed With Autologous $\alpha\beta$ CD19 CAR-T Therapies

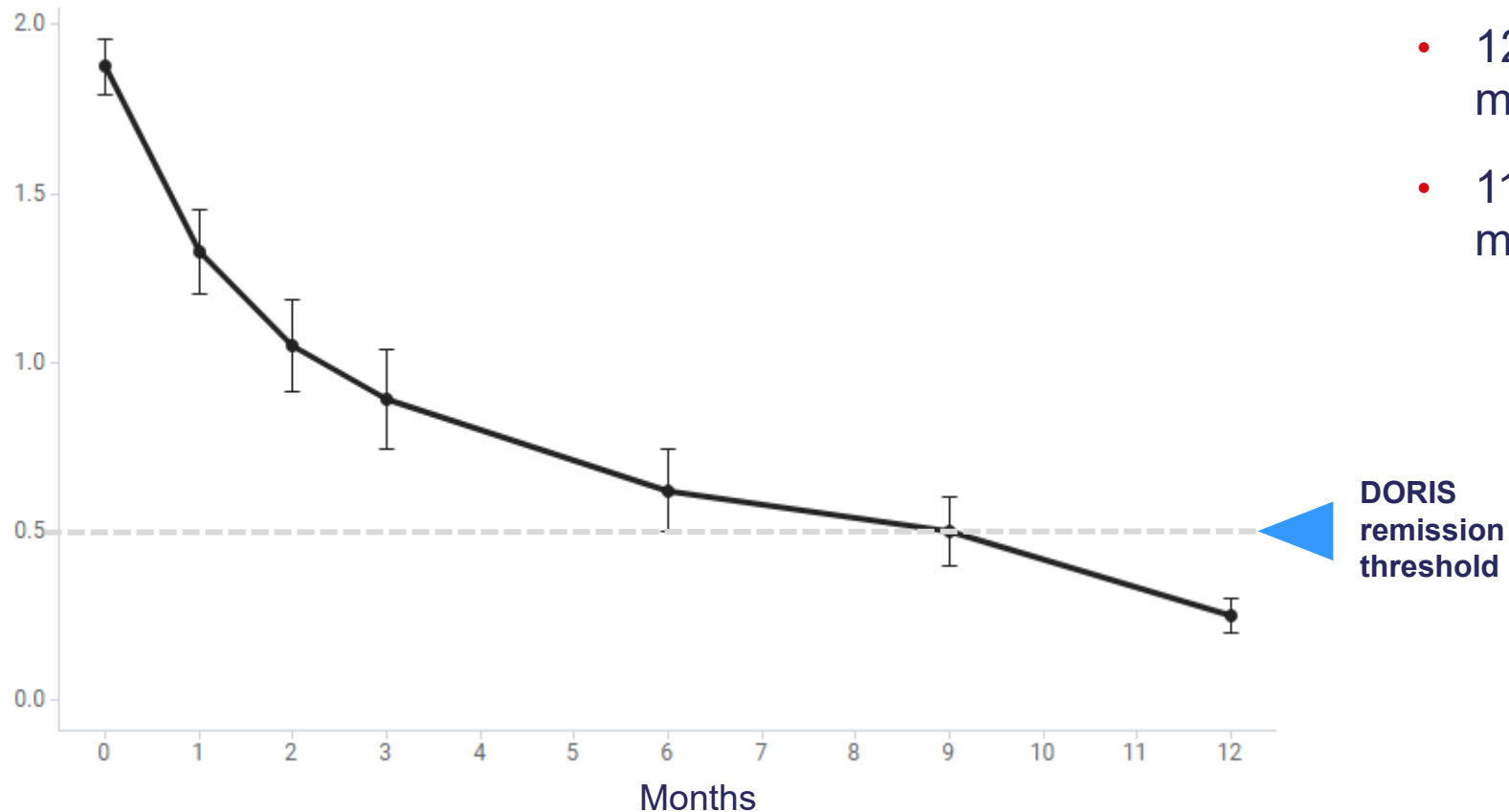
SLEDAI-2K over time (mean)



- Rapid and substantial decline in SLEDAI scores consistent with autologous CAR-T therapies
- Reductions in SLEDAI stable beyond 12 months

85% of Evaluable Patients with 12-Month Follow-Up Achieved PGA score of <0.5

Physician Global Assessment over time (mean)



- 12 / 22 patients achieved PGA <0.5 at month 6 and month 9
- 11 / 13 patients achieved PGA <0.5 at month 12

DORIS
remission
threshold

Patients Discontinued Immunosuppressants and Tapered Steroids to ≤ 5 mg Prednisone Equivalent

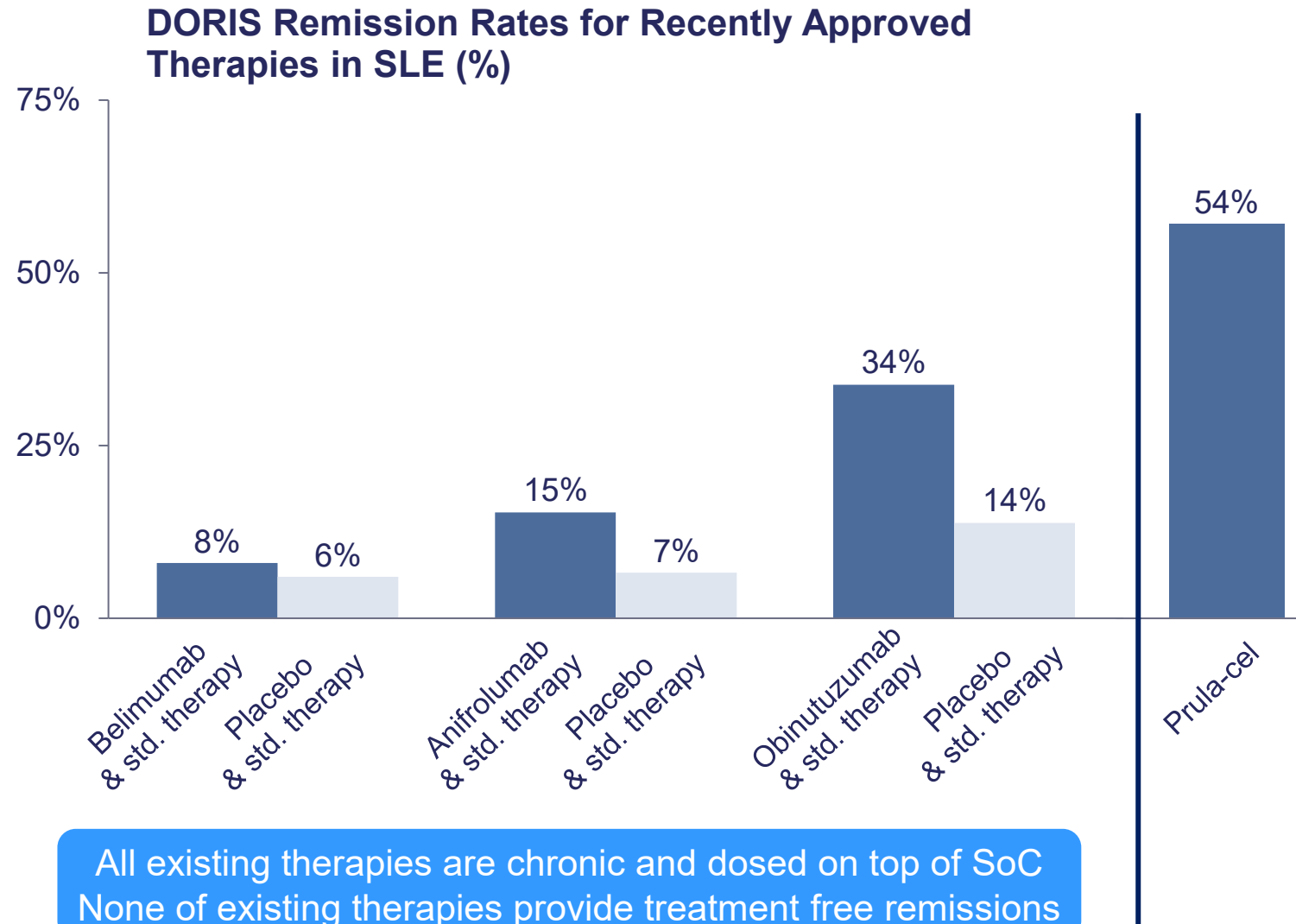
Immunosuppressants

All patients discontinued immunosuppressants

Steroids

Background steroids tapered to ≤ 5 mg prednisone in all patients but one

Recently Approved Therapies for SLE Are Delivered On Top of Standard Therapy & Have Not Shown High DORIS Remission Rates



Anticipated Pivotal Design

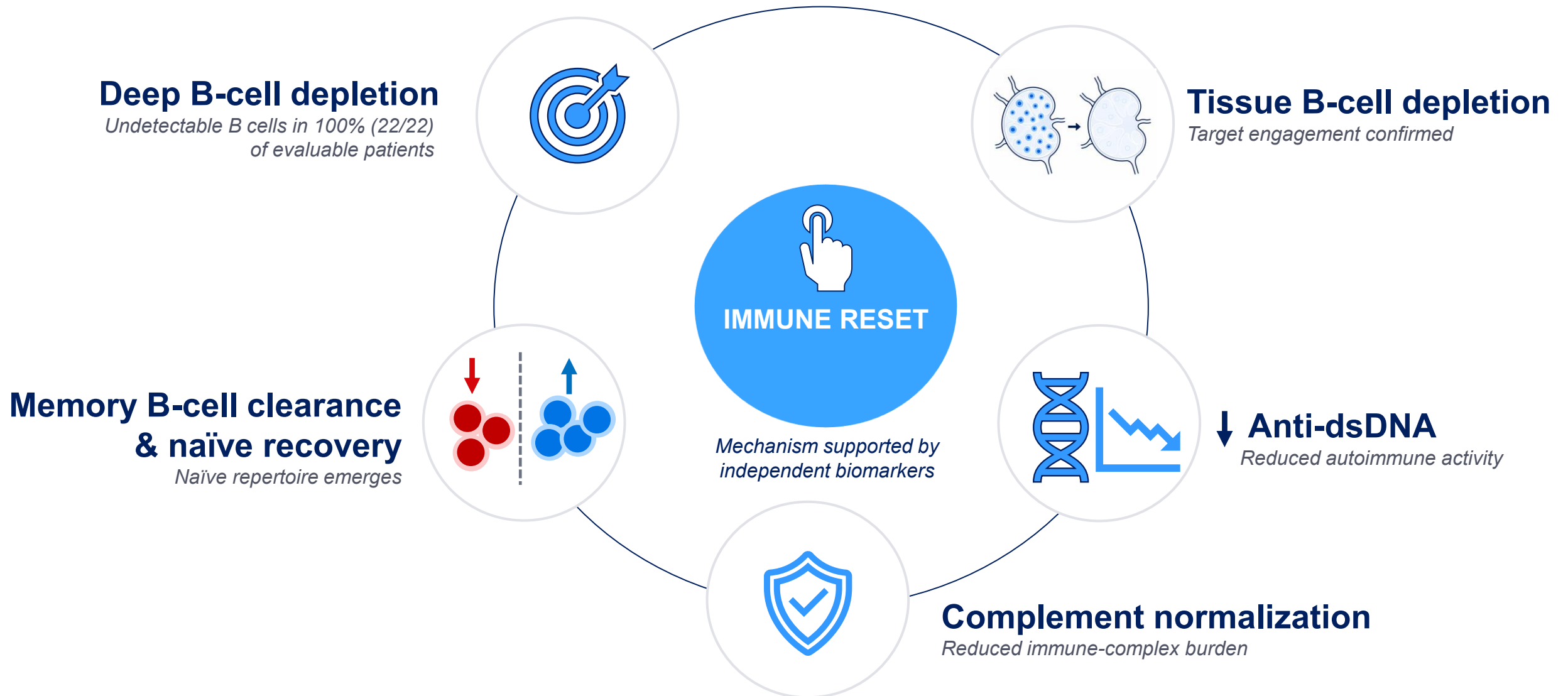
Single-arm study in LN per alignment with FDA

- Double-digit number of patients
- Key inclusion criteria
 - Participants must meet EULAR/ACR 2019 criteria for SLE
 - Biopsy-proven proliferative Class III or IV LN, with or without concomitant Class V involvement
 - Inadequate clinical response to at least two immunosuppressants
- Endpoint for LN Patients:
 - Complete renal response (LN) @ 12 months

Next steps

- Potential to expand study to include SLE
 - Consistent with regulatory precedent
 - Expected study size: Up to 90 patients
- Endpoint for SLE: DORIS remission
- Pivotal study start up activities in Q4; interim data anticipated 2028; pivotal readout in 2029

Multiple Independent Biomarkers Support Immune Reset



Deep B-cell Depletion with Naïve Recovery

KEY TAKEAWAY



Deep
B-cell depletion



Naïve dominant
Recovery ~1-3 months



Kinetics aligned with
leading CAR-T data



Immune reset

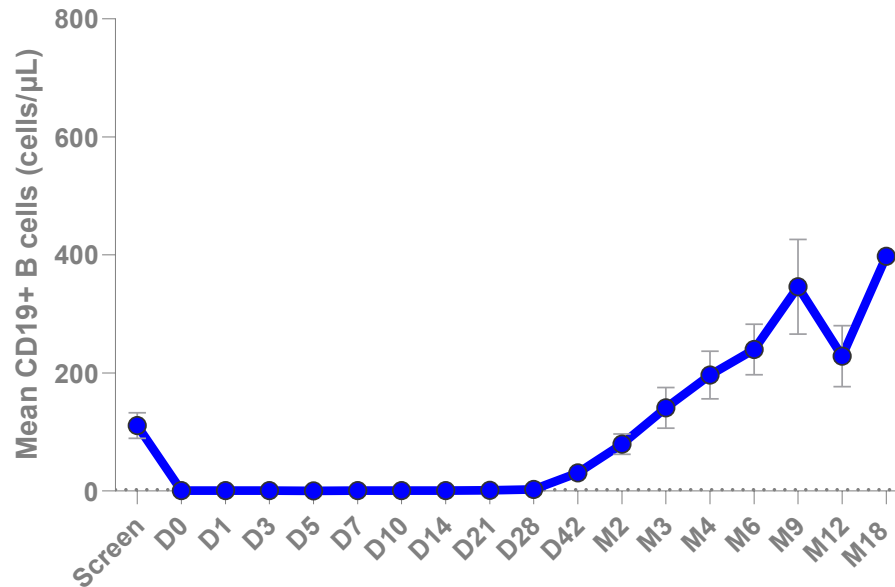


100%
(22/22)
evaluable patients

Achieved
Undetectable
CD19+ B cells

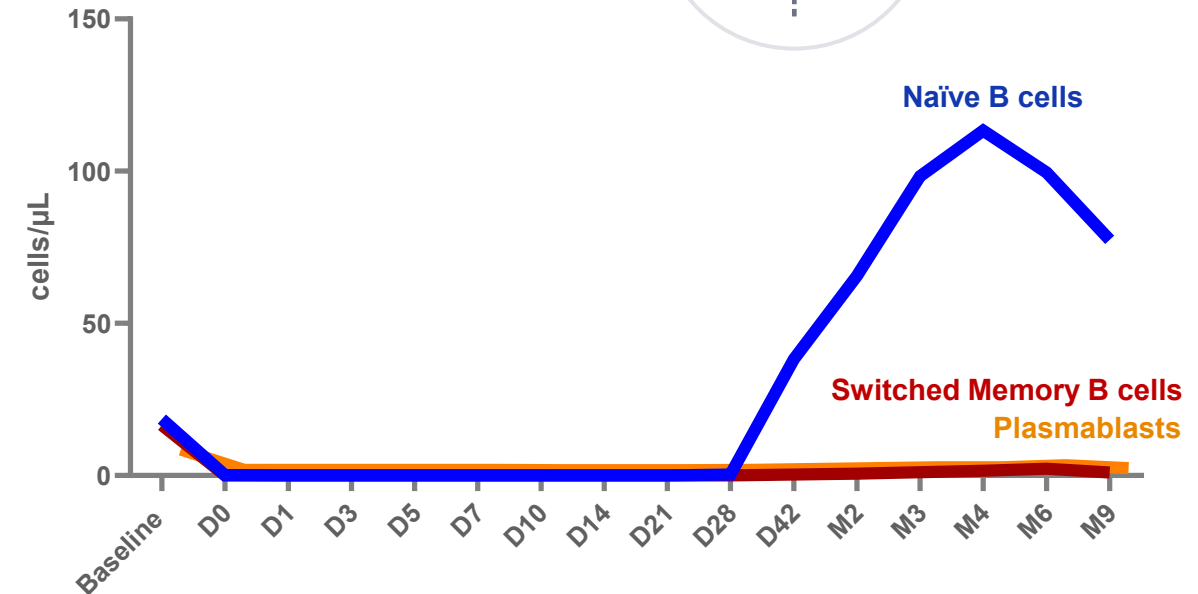
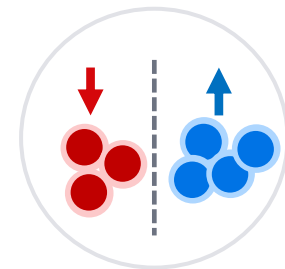
CD19⁺ B cell Depletion

Deep B cell depletion



Memory B-cell clearance & naïve recovery

Naïve repertoire emerges



CD19⁺ B-cell counts are presented as mean ± SEM. Naïve B-cell, switched memory B-cell, and plasmablast data are presented as median values at each study visit. B cells are considered undetectable when below the assay lower limit of detection (LLD; <2 cells/μL). D = day; M = month. CD19⁺ B cells = CD3⁺CD19⁺CD16/CD56⁻; naïve B cells = CD19⁺CD20⁺IgD⁺CD27⁻; switched memory B cells = CD19⁺CD20⁺IgD⁻CD27⁺; plasmablasts = CD19⁺CD20⁺IgD⁻CD27^{high}CD38^{high}CD138⁻.

Prula-cel Achieved Deep CD19+ B-cell Depletion in Tissues

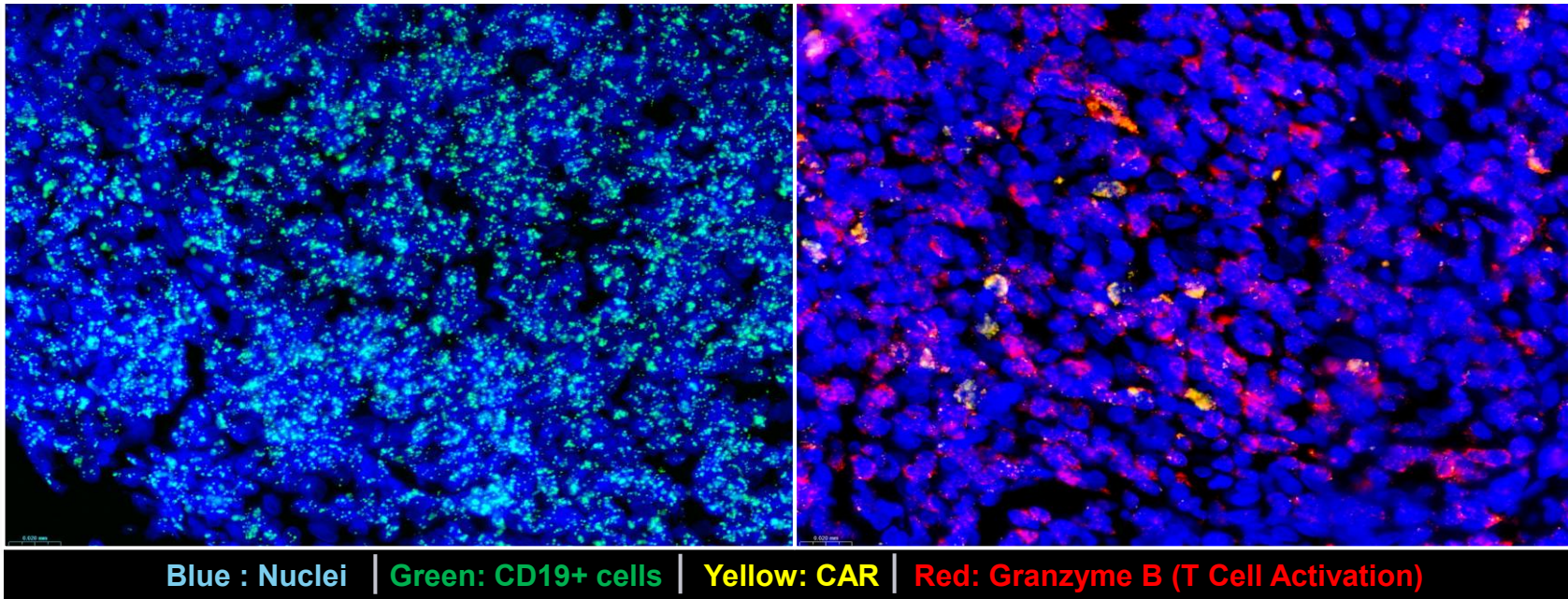


POC: *Prula-cel rapidly depleted CD19+ B cells within secondary lymphoid tissue*

MCL PATIENT LYMPH NODE BIOPSIES

Baseline

Day 10 Post Dose



Robust tissue infiltration by prula-cel and complete depletion of CD19+ B cells
observed at day 10 within secondary lymphoid tissue

Reduction in Anti-dsDNA Titers and Immune Complex Burden Supports an Emerging Immune Reset

Key Findings

91% (10/11)

of evaluable baseline anti-dsDNA-positive patients demonstrated **reductions in anti-dsDNA titers**

90% (9/10)

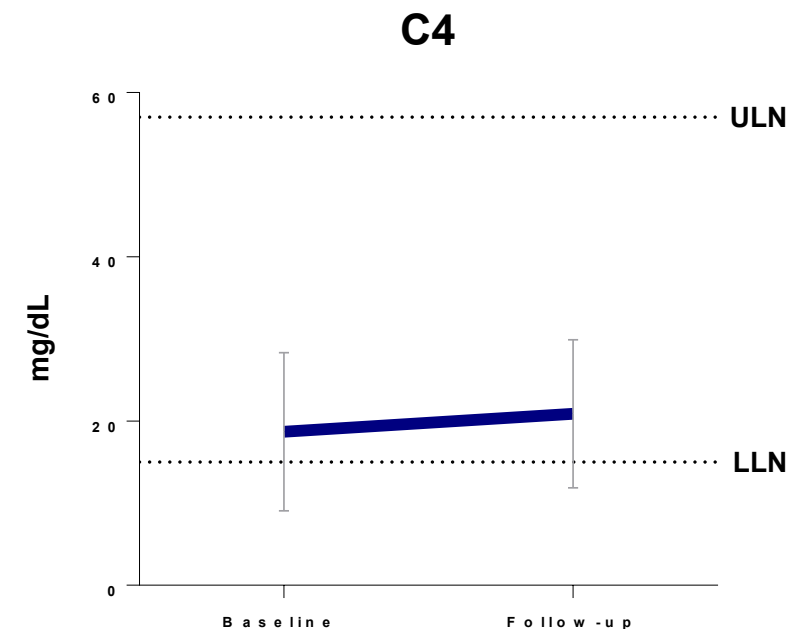
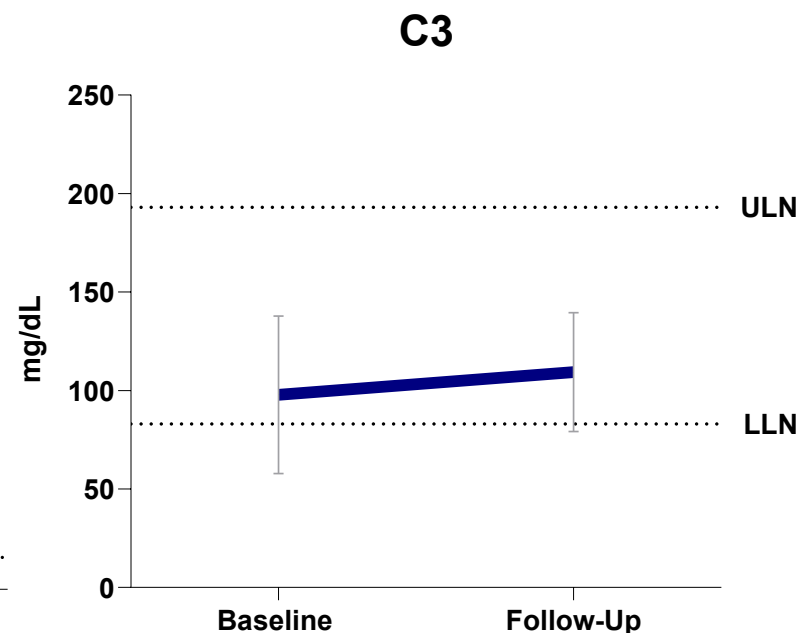
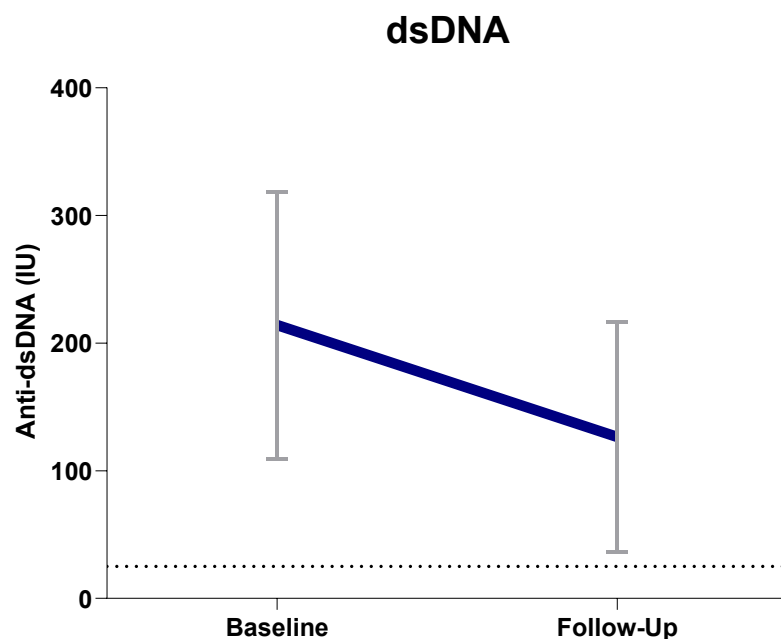
of patients with low baseline C3 and/or C4 demonstrated **complement recovery** (normalized or increased levels)

70% (7/10)

achieved **normalization** of at least one complement parameter



Findings are consistent with **reduced immune-complex burden** and support an emerging **immune reset mechanism**.

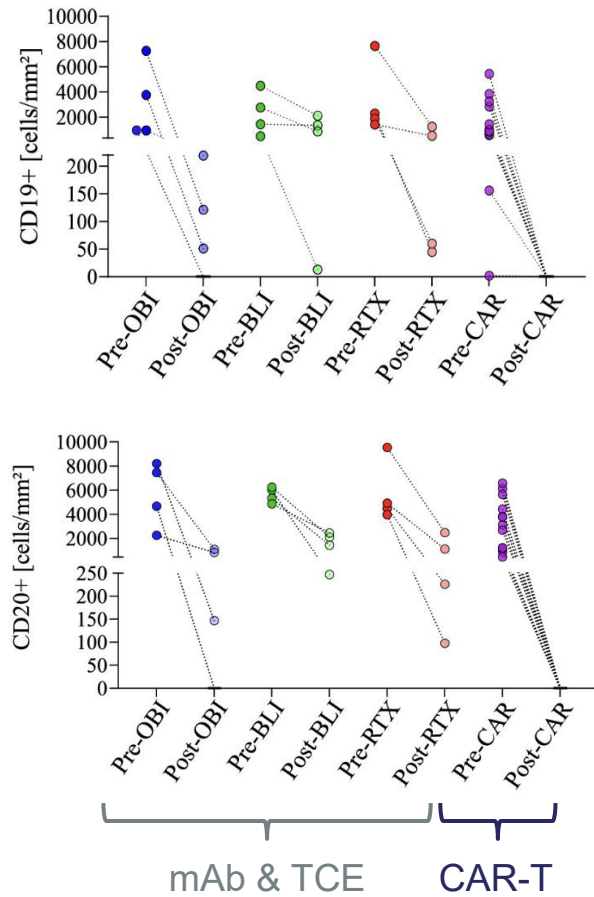


Of 22 efficacy evaluable patients, n=11 were baseline anti-dsDNA-positive and evaluable by ELISA. Data are presented as mean ± SD. For anti-dsDNA, the horizontal dotted line indicates the positivity cutoff. For C3 and C4, horizontal dotted lines indicate the LLN and ULN.

Prula-cel: Designed for Broad Adoption and Scalable Commercialization

	Autoimmune CAR-T	
	AUTOLOGOUS CAR-T <i>Current paradigm</i>	Prula-cel <i>Allogeneic / bank-ready $\gamma\delta 1$ CAR-T</i>
Treatment Workflow	 Physician withdraws immunosuppression, schedules leukapheresis weeks later; product manufactured per patient	 Readily available product — no leukapheresis or personalized manufacturing
Time to Treatment	 Several weeks vein-to-vein time for individual manufacturing and frequent need for bridging therapy	 Immediate dosing from cryopreserved inventory
Safety Profile	 Meaningful IEC-HS, ICANS and CRS risk requiring intensive monitoring	 No IEC-HS, No ICANS and No Gr>2 CRS
Site-of-Care Access	 Higher safety burden generally restricts use to specialized centers	 Well suited to community rheumatology and infusion settings
Manufacturing Cost & Scale	 Custom batch per patient drives high COGS & impedes scalability for prevalent autoimmune diseases	 Centralized batch production intended to lower cost & improve scalability

Incomplete B-Cell Depletion With Protein-based Approaches (mAbs & TCEs) Associated With Less Robust Clinical Responses in Patients With Autoimmune Diseases



		mAb & TCE				CAR-T							
		OBI 1	OBI 2	OBI 3	OBI 4	BLI 1	BLI 2	BLI 3	BLI 4	RTX 1	RTX 2	RTX 3	RTX 4
		SLE	IIM	SLE	SLE	RA	RA	RA	RA	RA	RA	IIM	IIM
	LN Clearance												
	Clinical response												
	Remission												
	Drug-Free State												
		CAR 1	CAR 2	CAR 3	CAR 4	CAR 5	CAR 6	CAR 7	CAR 8	CAR 9	CAR 10	CAR 11	CAR 12
		SLE	SSc	SLE	SSc	SLE	SLE	SSc	SSc	SSc	SSc	IIM	SSc
	LN Clearance												
	Clinical response												
	Remission		n.a.		n.a.				n.a.	n.a.	n.a.		n.a.
	Drug-Free State												

■ Achieved
 ■ Not achieved
 ■ Not available

Patients treated with mAbs and TCEs do not consistently achieve remissions and drug-free states in contrast to patients treated with CAR-T

Incomplete B-cell depletion in lymph nodes with mAbs and TCE approaches in contrast to complete depletion in all CAR-T treated patient samples

Prula-cel Potential Competitive Positioning in Lupus With and Without Nephritis

Autologous CAR-T

- Proven immunosuppressant free DORIS/CRR
- Safety Challenges: IEC-HS, ICANS, CRS
- Require leukapheresis & personalized manufacturing

Bi-Specifics

- Have not demonstrated efficacy comparable to autologous CAR-T
- Likely require semi-chronic dosing (immune dimming)
- Chronic immunosuppression may lead to high infection rate

Prula-cel Target Product Profile

- One-time, off-the-shelf therapy, available in outpatient setting
- Delivers immunosuppressant free remissions & steroids tapered to ≤ 5 mg prednisone equivalent comparable to autologous CAR-T
- Favorable safety profile: No IEC-HS, No ICANS, No CRS > Gr2
- Does not require leukapheresis or personalized manufacturing
- Delivers immune-reset with no permanent gene therapy

mRNA in vivo CAR-T

- Deliver transient expression CAR-T in patients
- Have not demonstrated complete B cell depletion & immune reset in patients
- Have not demonstrated efficacy comparable to autologous CAR-T

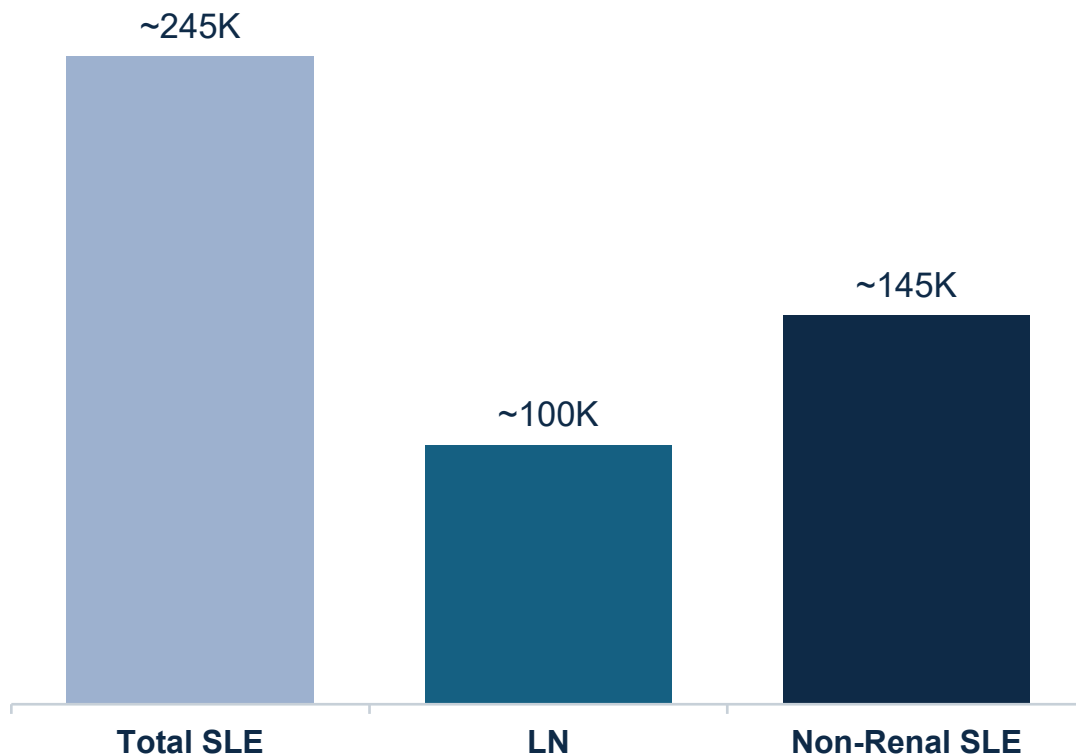
Lentivirus based in vivo CAR-T

- Deliver gene therapy to stay “forever” with unknown risk (e.g. insertional mutagenesis)
- May experience similar safety challenges to autologous CAR-T
- Have not demonstrated efficacy comparable to autologous CAR-T

Commercial Potential

~70K-Patient Organ-Threatening SLE Opportunity in the US

US Prevalence of SLE & Non-Renal SLE (thousands of patients)



Lupus Nephritis (LN)

~100K prevalent patients in the US

Roughly 25–45% of LN patients fail to achieve remission after 24 months of SoC treatment

→ **~35K addressable organ/life-threatening LN patients**



Non-Renal SLE

145K prevalent patients in the US

An estimated 25% of non-renal SLE patients have organ/life-threatening disease on current SoC (Adicet estimate; analyst reports)

→ **~35K addressable organ/life-threatening non-renal SLE patients**

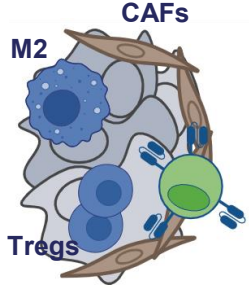
70K patients in the US alone have organ/life-threatening SLE, with or without nephritis

ADI-212

Prostate Cancer



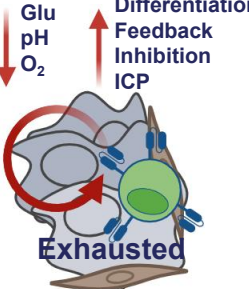
Current Challenges for Existing Cell Therapies in Solid Tumors



CAFs
M2
Tregs

1 – Nonpermissive TME

- Poor immune infiltrate or protected by suppressive cells
- Missing chemotactic and immunostimulating signals
- Cell therapy platform lacking tumor trafficking and/or expansion



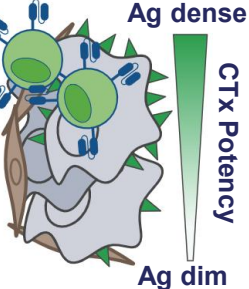
Glu
pH
O₂

Differentiation
Feedback
Inhibition
ICP

Exhausted

2 – Effector Cells Become Fatigued

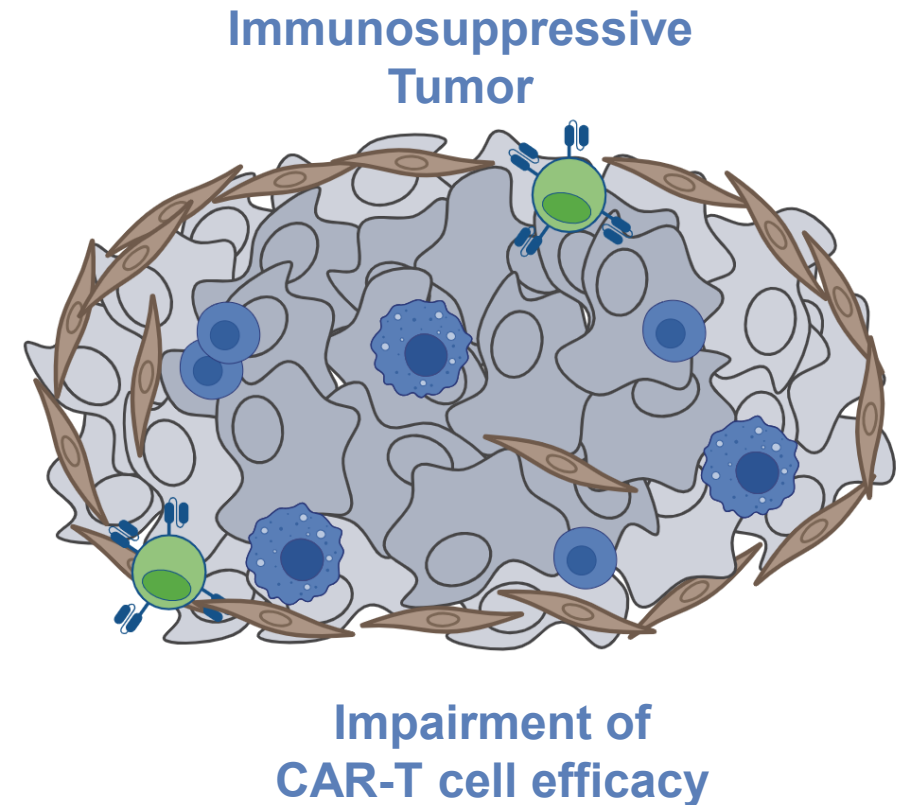
- Tumor cells express inhibitory receptors/factors
- Metabolic stress exhausts effector cells
- Feedback loops inhibit repetitive killing capacity



Ag dense
CTx Potency
Ag dim

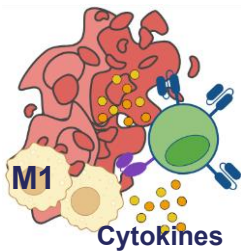
3 – Target Insufficiency

- Antigen (Ag) dimming
- Lack of diversity for Ag recognition
- Limited opportunity for endogenous effector participation



Three Strategic Pillars for Overcoming Solid Tumor Barriers

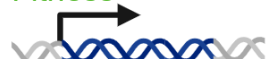
Tumors



1 – Localized Cytokine Armoring

- Transform to active and permissive immune TME
- Intrinsic and extrinsic immune activation
- Controlled¹ and localized² exposure

Fitness



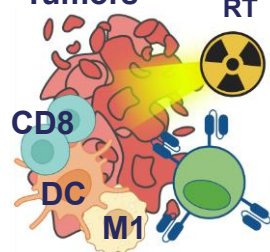
Restraint



2 – Transcriptional Tuning

- Tune transcriptional controls to enhance T cell fitness³ and relieve restraints⁴
- Reduced exhaustion to increase persistence

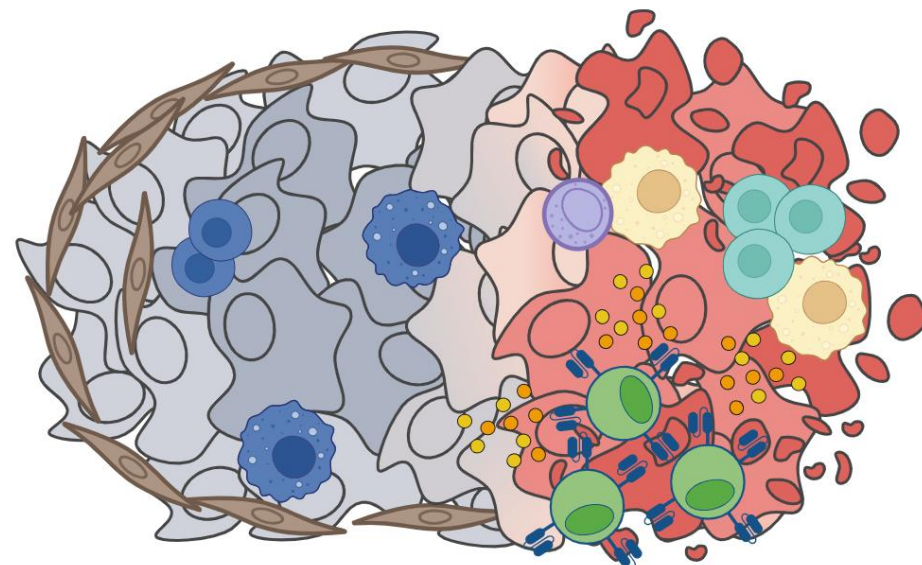
Tumors



3 – Target & Tumor Engagement

- Direct-antigen targeting
- Increased immune cell recruitment and antigen spreading
- Neoantigens from radioligand therapy⁵

Immunosuppressive Tumor



Susceptible Tumor

ADI-212 Integrates Localized Cytokine Armoring, Transcriptional Tuning, and Refined Tumor Targeting



1 – Localized Cytokine Armoring

- Membrane-tethered IL-12; CAR activation-induced
- Boosts CAR-T cell potency
- Modulates and repolarizes the TME
- Decreased cytokine release profile for safety



2 – Transcriptional Tuning

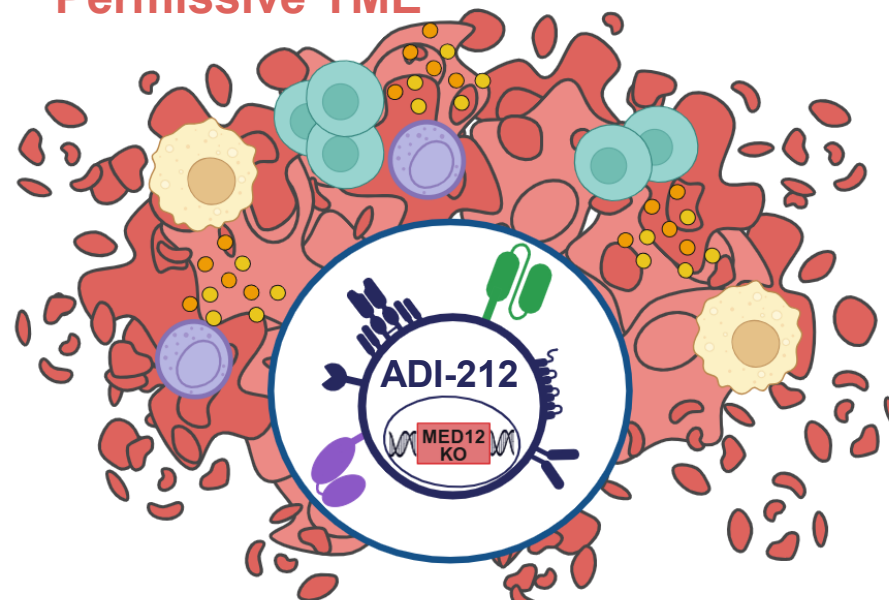
- MED12 mediates transcription
- CRISPR KO of MED12
- Greater T-cell stemness and reduced modulation
- Improved persistence of cytotoxicity



3 – Target & Tumor Engagement

- Refined epitope for recognizing on target tissues
- Promotes epitope spreading and endogenous effector participation
- Complimentary targeting via innate effector function

Conversion to Permissive TME



Multi-mechanistic Attack of Tumor

ADI-212 Designed To Address Multiple Barriers in a Single Off-the-Shelf Product

Adicet's $\gamma\delta$ T Platform

Target & Tumor Engagement

Localized Cytokine Armoring

Transcriptional Tuning

PSMA-Directed CAR

with clinically supported binding determinants against functional PSMA

PSMA-Directed CAR

Inducible Membrane-Tethered IL-12

Tethered IL-12 Cytokine

designed to localize and enhance anti-tumor mechanisms to the tumor microenvironment

- Improved potency & cytotoxicity of CAR-T platform
- Localize IL-12 anti-tumor activity
- Reshape the immunosuppressive microenvironment

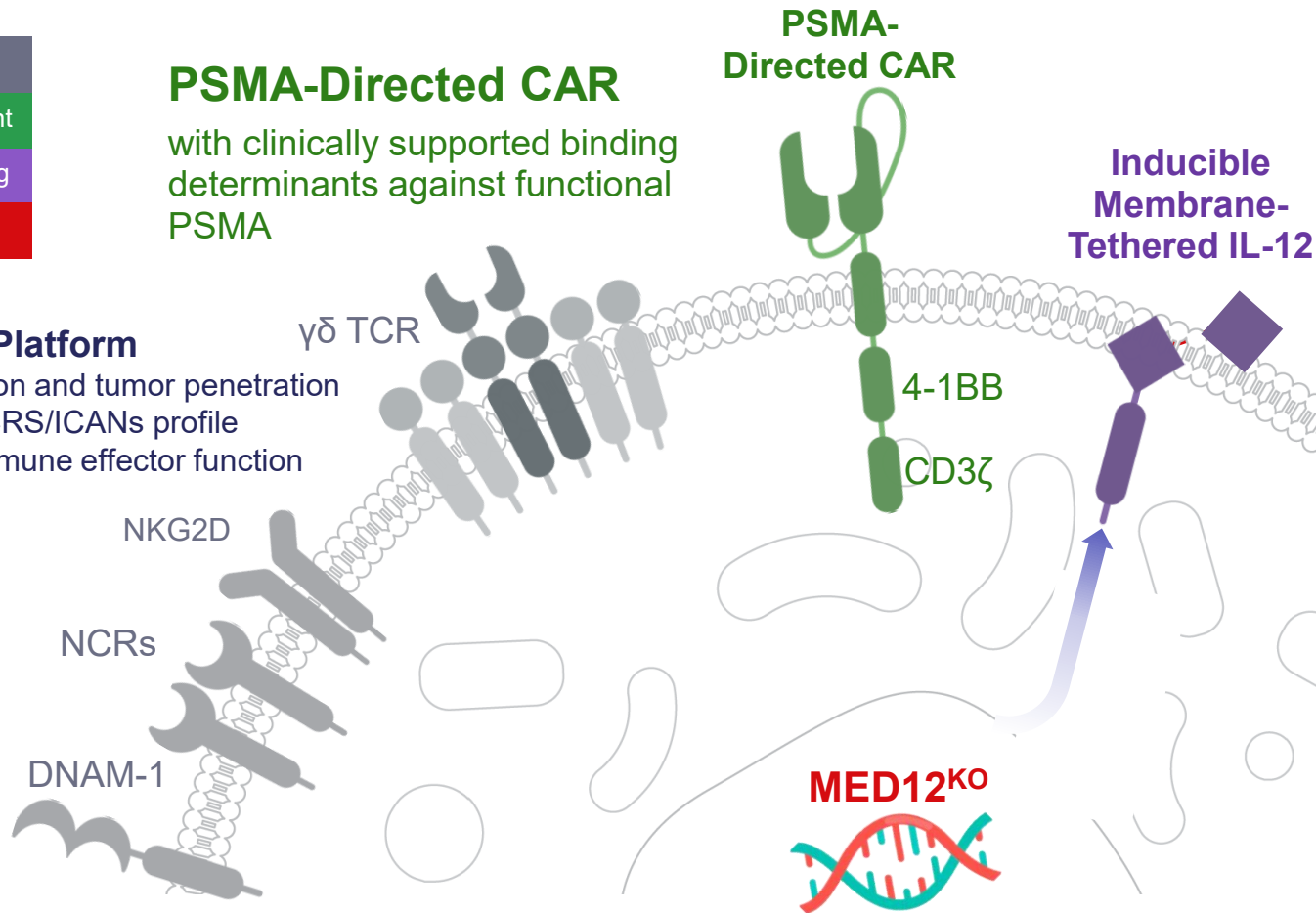
MED12^{KO}

for improved potency and tumor-cell killing capacity compared to previous generation alpha-beta and $\gamma\delta$ CAR-T programs in oncology

- Enhance proliferation and survival
- Improve potency & metabolic fitness advantages

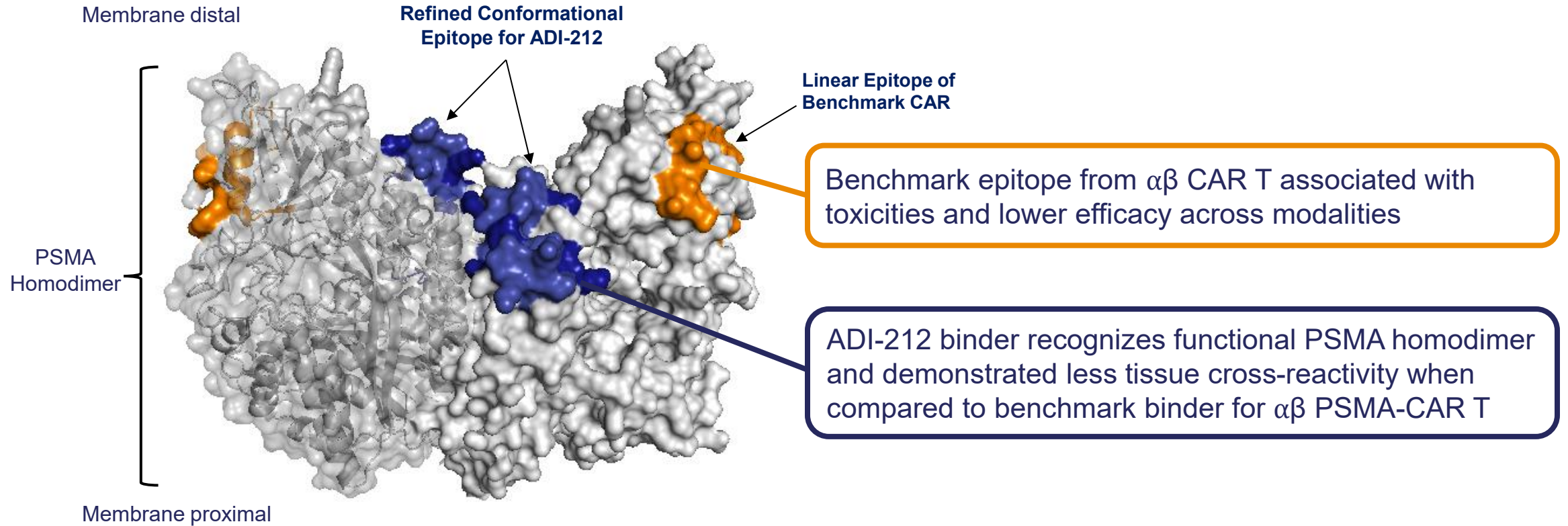
Adicet's $\gamma\delta$ 1 CAR-T Platform

- Demonstrated expansion and tumor penetration
- Favorable safety and CRS/ICANS profile
- Innate and adaptive immune effector function

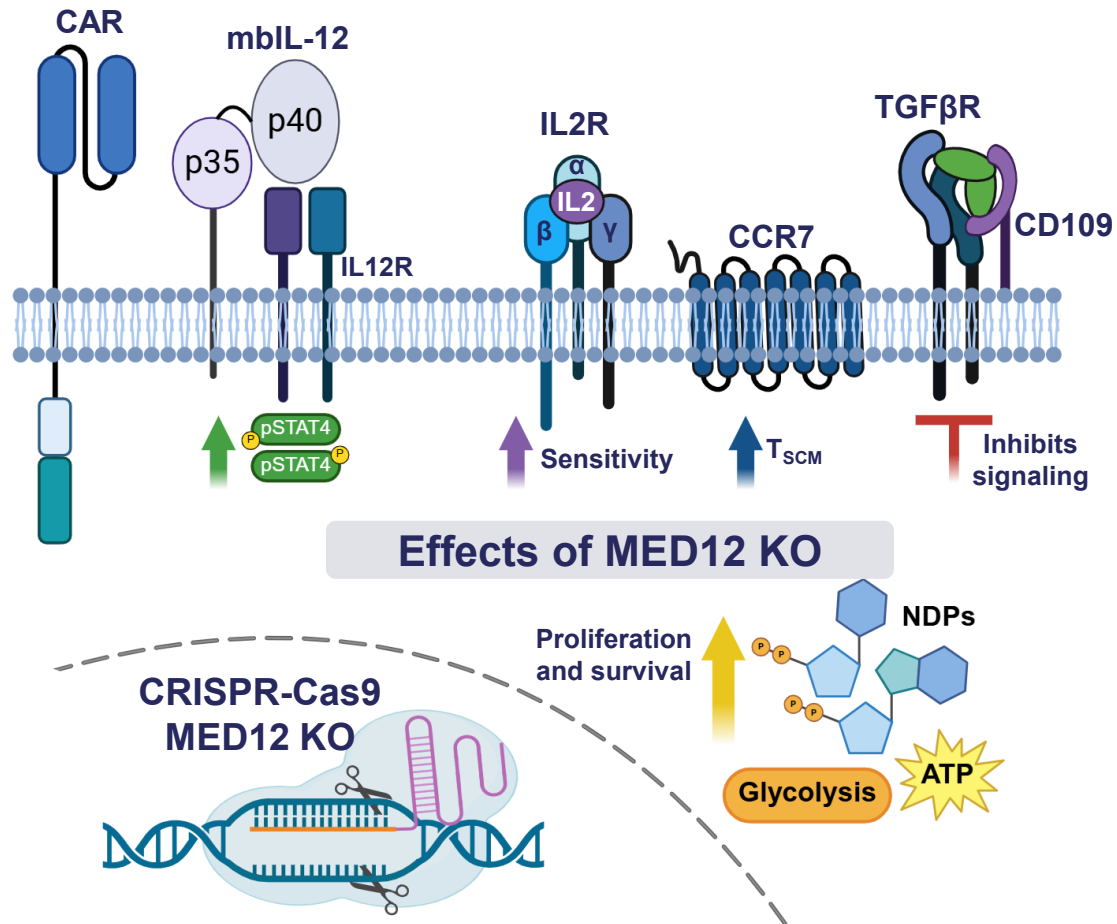


PSMA CAR Design is Tailored for PSMA Target Recognition

Adicet's proprietary CAR targeting has similar conformational requirements to active and approved therapeutics with less off tissue targeting



ADI-212: Genetic Tuning via MED12 KO Designed to Enhance Potency and Introduce Synergy Across Armoring Strategies

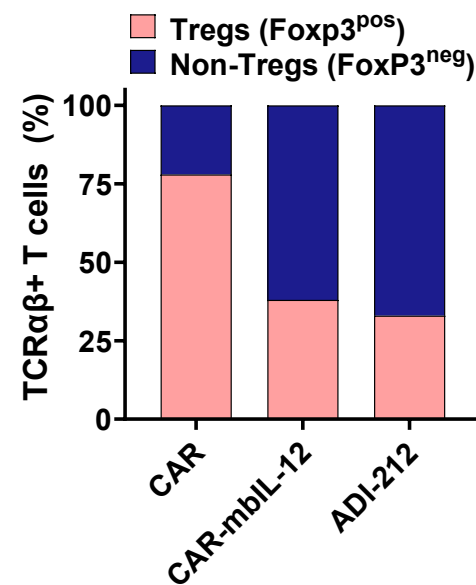
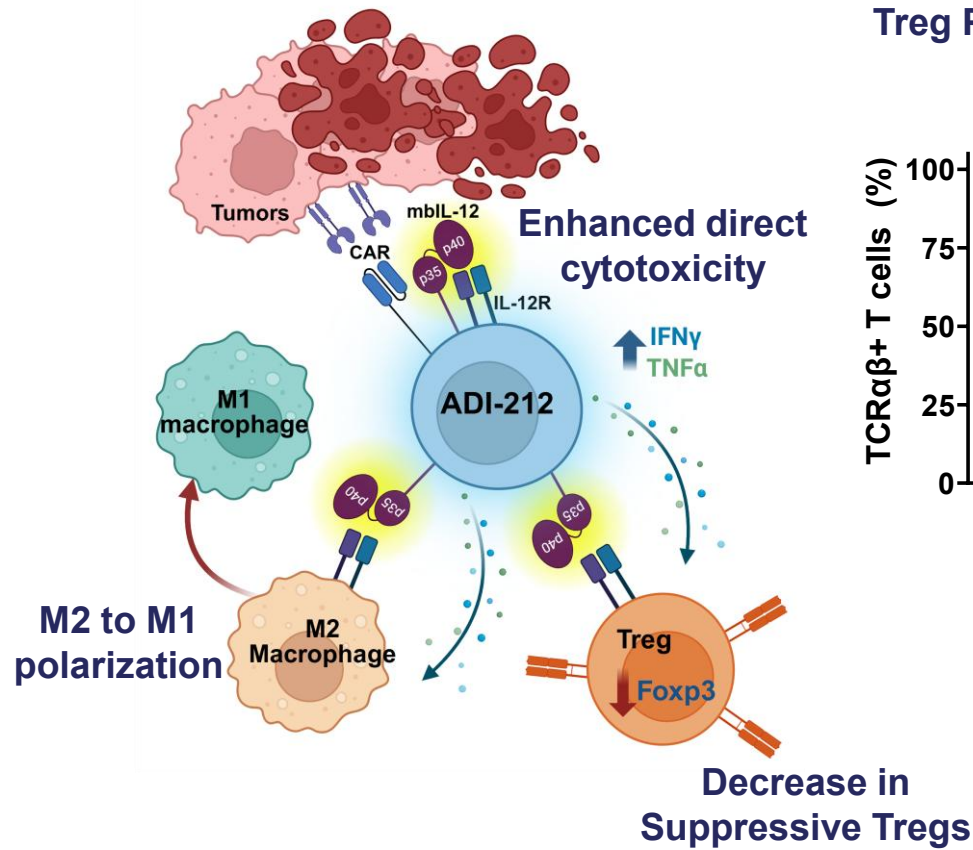


mbIL-12 armoring and MED12 KO provide a unique synergy for our CAR Vδ1 T cell platform

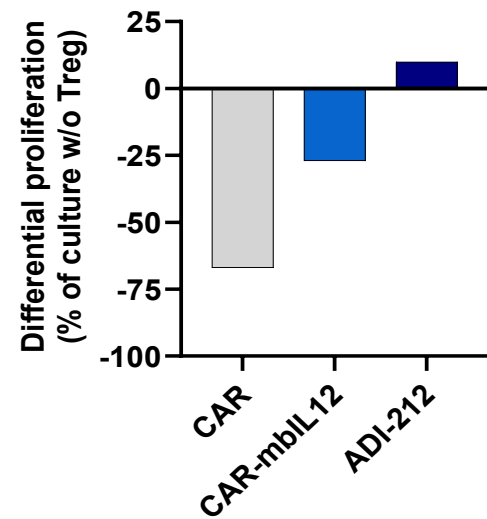
- ✓ Localize IL-12 signaling to Site of Action
 - ✓ Reshape the immunosuppressive solid TME
 - ✓ Increase tumor killing potential for CAR-T while decreasing suppressive barriers
 - ✓ Maintain proliferation and survival
 - ✓ Maximize potency and manufacturability
 - ✓ Metabolic fitness advantages
- mbIL-12
- Synergy
- MED12 KO

ADI-212 Capacity to Reshape Tumor Microenvironments and Maintain Potent Anti-Tumor Killing in the Presence of Immunosuppressive Cells

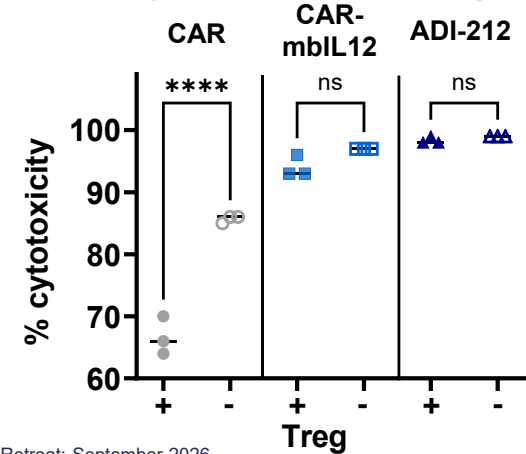
1 mbIL-12 on ADI-212 modulates Treg Population in Cocultures



2 ADI-212 Expansion is Resilient to Suppressive Tregs



3 ADI-212 is Resilient to Inhibition by Suppressive Tregs



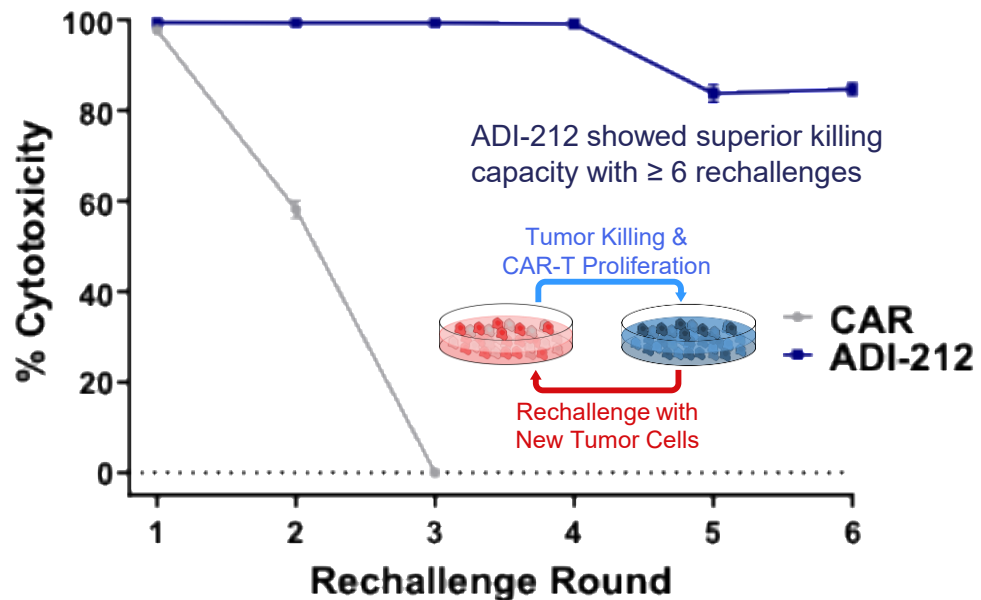
TME, Tumor Microenvironment; M1, M1 Macrophages; M2, M2 Macrophages; Treg, Regulatory T cells; IFN γ , Interferon-gamma; TNF α , Tumor necrosis factor-alpha;

Herrman, M. Aftab, B, et al. presented at: Prostate Cancer Foundation Scientific Retreat; September 2026.

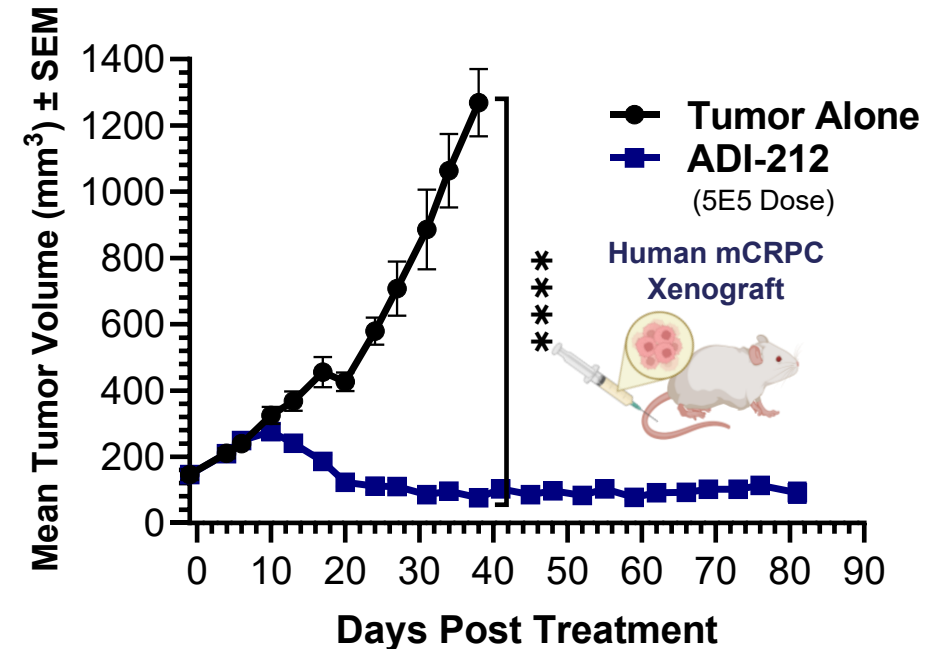
Watkins SK, et al. IL-12 rapidly alters the functional profile of tumor-associated and tumor-infiltrating macrophages in vitro and in vivo. *J Immunol.* 2007.
 Yu X, et al. Overexpression of IL-12 reverses the phenotype and function of M2 macrophages to M1 macrophages. *Int J Clin Exp Pathol.* 2016.
 Tian Y, et al. CCR5 and IL-12 co-expression in CAR T cells improves antitumor efficacy by reprogramming tumor microenvironment in solid tumors. *Cancer Immunol Immunother.* 2025.

ADI-212 Demonstrated Highly Potent Preclinical Activity in Solid Tumors

Durable In Vitro Cytotoxicity Demonstrated Against PSMA+ Prostate Cancer Cells

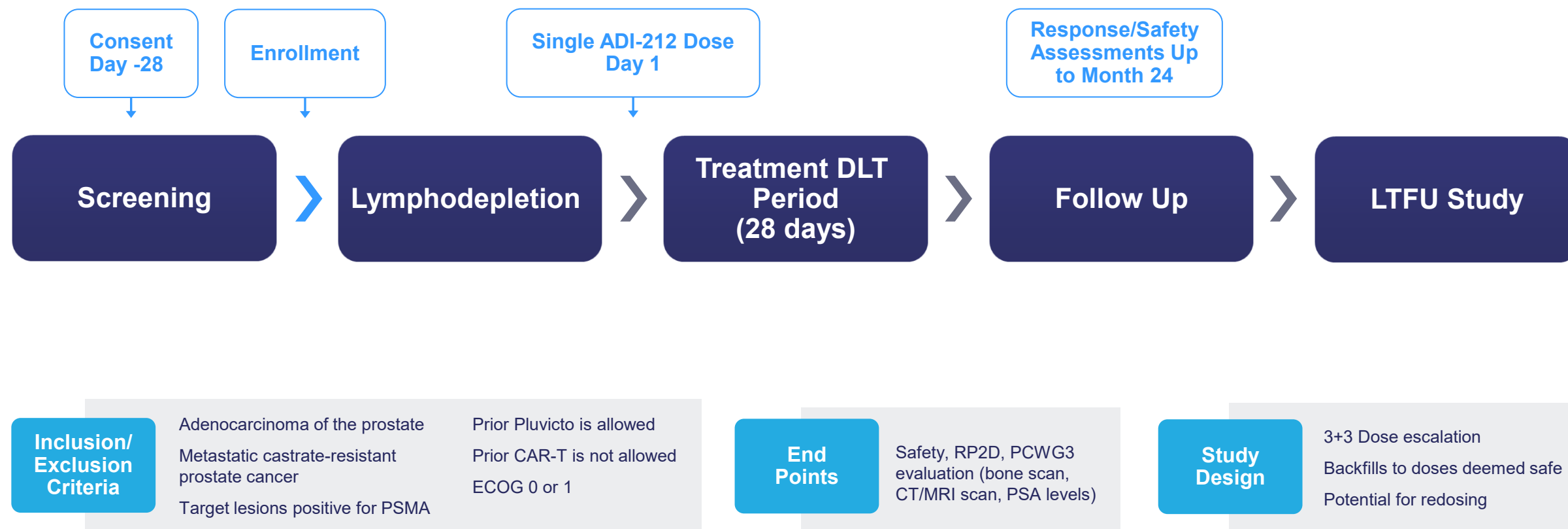


Highly Potent as Single Dose in a mCRPC Tumor In Vivo Model



ADI-212 First-in-Human Dose Escalation Study

IND Cleared in US and Aiming to Extend Globally to Additional Regions
First-Patient Dosing Anticipated in 4Q26 with Clinical Data Expected 1H27 & 2H27



In Vivo CAR-T Portfolio

Differentiated in vivo CAR-T approach and pipeline designed to address significant unmet needs

Hematologic malignancies and solid tumors



Extending Adicet's Off-the-Shelf Development Capabilities to a Next Generation In Vivo CAR-T Design

Goal:

Differentiated in vivo CAR-T programs engineered to achieve highly efficient, selective T-cell transduction and deliver next-generation payloads (advanced CARs, armoring, biology-modifying constructs) with superior potency, efficacy, and durability.

In Vivo CAR T – Cell-specific delivery via novel homing and VSV-G fusogen technology

Program	Indication	Research	IND-Enabling	Clinical	Key Differentiation
Multiple Myeloma Target: BCMA Mono / Dual CAR	MM	PRE-CLINICAL			• Differentiated CAR design with effective BCMA targeting • Decreased susceptibility to immune-mediated inactivation and clearance
NHL Target: CD19 Dual CAR	NHL	PRE-CLINICAL			• Differentiated dual-CAR design
Solid Tumors Target: Not disclosed	NA	PRE-CLINICAL			• Multiple armoring technologies enhance efficacy in solid tumors

Plan to initiate first program in 2H/2027; data potentially in 2028
Clinical data from second and third program potentially in 2028

Developing Broad Pipeline of Allogeneic $\gamma\delta 1$ CAR-T Cell and Best In Class Differentiated In Vivo CAR-T Therapies

Off-The-Shelf $\gamma\delta 1$ CAR-T

Program	Indication	Research	IND-Enabling	Clinical	Potential Key Differentiation
Prula-cel Target: CD20	LN/SLE				- Off-the-shelf, outpatient dosing — no leukapheresis or personalized manufacturing - Quick, easy administration - Favorable safety profile; reduced IEC-HS, ICANS, CRS risk - Efficacy comparable to autologous CAR-T
	SSc				
ADI-212 Target: PSMA (gene-edited w/ armor)	mCRPC				- Validated target (PSMA) - Armoring enhances tumor-cell killing - Improved anti-tumor activity in the tumor microenvironment

In Vivo CAR-T – Cell-specific delivery via novel homing and VSV-G fusogen technology

Program	Indication	Research	IND-Enabling	Clinical	Potential Key Differentiation
Multiple Myeloma Target: BCMA Mono / Dual CAR	MM	PRE-CLINICAL			- Differentiated CAR design with effective BCMA targeting - Decreased susceptibility to immune-mediated inactivation and clearance
NHL Target: CD19 Dual CAR	NHL	PRE-CLINICAL			Differentiated dual-CAR design
Solid Tumors Target: Not disclosed	NA	PRE-CLINICAL			Multiple armoring technologies designed to enhance efficacy in solid tumors

AI= autoimmune; BCMA= B-cell maturation antigen; CRS= Cytokine release syndrome; ICANS= Immune effector cell-associated neurotoxicity syndrome; LN= Lupus nephritis; mCRPC= Metastatic castration-resistant prostate cancer; MM= multiple myeloma; NA=Not disclosed; NHL= Non-Hodgkin lymphoma; PSMA= Prostate specific membrane antigen; SLE= Systemic lupus erythematosus; SSc= Systemic sclerosis; Timing subject to site activation, patient enrollment, data readouts and regulatory feedback; Cut-off date: August 28, 2026; Data cut included 22 efficacy-evaluable patients

Anticipated Near-Term Value-Creating Milestones

Clinical, regulatory and platform catalysts expected across prula-cel, ADI-212 and in vivo CAR-T programs

	2026	2027	2028	2029
Prula-cel Autoimmune disease LN / SLE / SSc	LN/SLE clinical update LN/SLE Pivotal start-up activities Potential SLE expansion*	SSc clinical update* LN/SLE clinical update*	LN/SLE Interim pivotal data*	LN/SLE Potential pivotal data*
	Progression toward registrational pathway →			
ADI-212 mCRPC	IND Clearance mCRPC Enrollment	mCRPC Clinical update*	mCRPC Clinical update*	
In Vivo CAR-T MM Program	Platform/program update	MM Initiate clinical study*	MM Clinical Update*	
In Vivo CAR-T NHL Program	Platform/program update		Potential clinical data*	
In Vivo CAR-T Solid Tumors Program	Platform/program update		Potential clinical data*	



Investor Presentation

October 2026

Adicet Bio