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 γδ1 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/2027**

ADI-212

Next-generation gene-edited & armored anti-PSMA γδ1 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**

Cut-off date: August 28, 2026; Data cut included 22 efficacy-evaluable patients; CRR= Complete renal response; CRS= Cytokine release syndrome; DORIS= Definition of remission in systemic lupus; ICANS= Immune effector cell associated neurotoxicity syndrome; IEC-HS= Immune effector cell-associated hemophagocytic lymphohistiocytosis-like syndrome; LN= Lupus nephritis; SLE= Systemic lupus erythematosus*all but one patient tapered background steroids to ≤ 5mg prednisone equivalent; VSV-G= Vesicular Stomatitis Virus Glycoprotein G

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Developing Broad Pipeline of Allogeneic γδ1 CAR-T Cell and Best In Class Differentiated In Vivo CAR-T Therapies

Off-The-Shelf γδ1 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

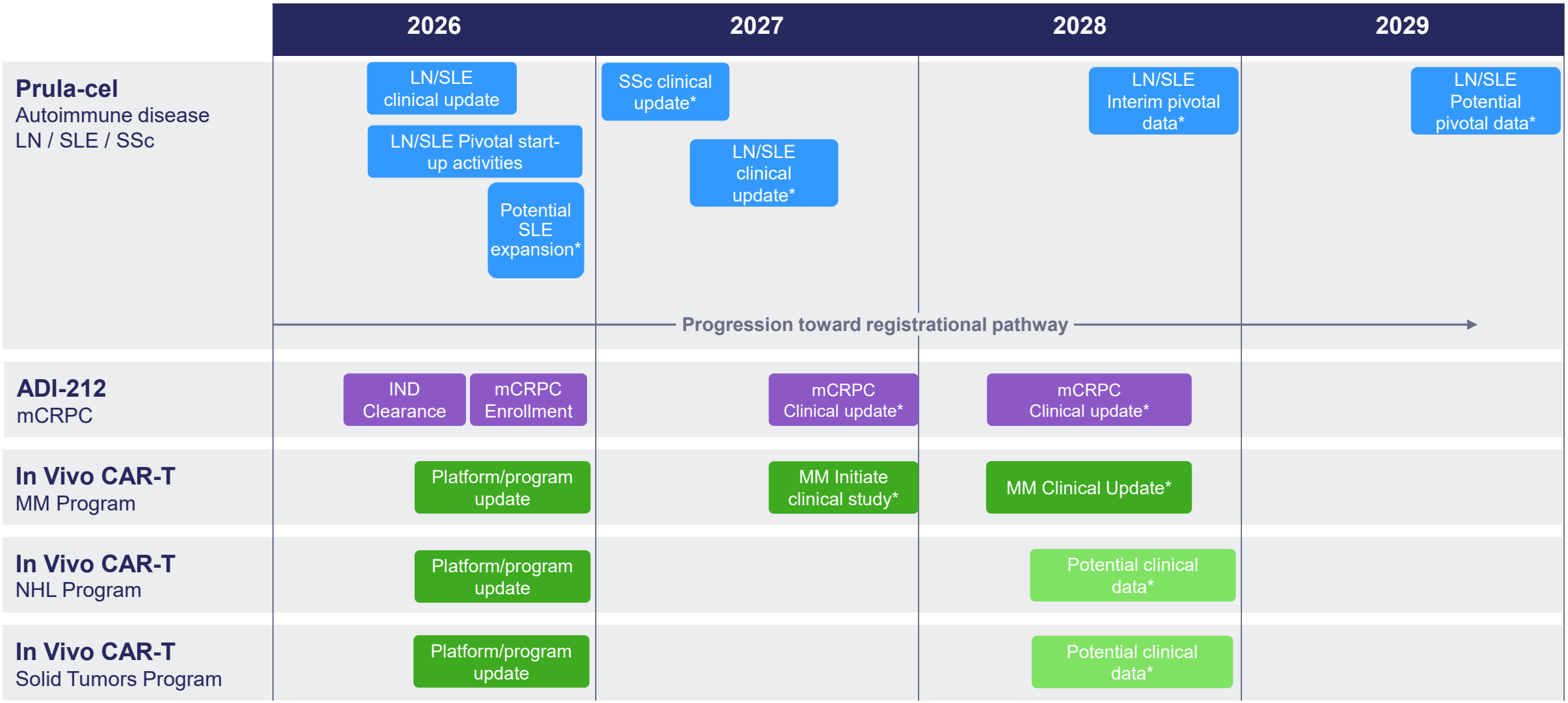
Program	Indication	Research	IND-Enabling	Clinical	Potential Key Differentiation
Multiple Myeloma Target: BCMA Mono / Dual CAR	MM	PRE-CLINICAL			• Differentiated CAR design with effective BCMA targeting • Decreased susceptibility to immune-mediated inactivation and clearance
NHL Target: CD19 Dual CAR	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

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Anticipated Near-Term Value-Creating Milestones

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



*Achievement and timing of forward-looking milestones subject to clinical progress, regulatory outcomes, financing ability and other business considerations

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Adicet Bio Leadership Team



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Prula-cel

Autoimmune Indications

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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

QoL= Quality of life

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”

Cut-off date: August 28, 2026; Data cut included 22 efficacy-evaluable patients

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

Cut-off date: August 28, 2026; Data cut included 22 efficacy-evaluable patients; CRR= Complete renal response; CRS= Cytokine release syndrome; DORIS= Definition of remission in systemic lupus; ICANS= Immune effector cell associated neurotoxicity syndrome; IEC-HS= Immune effector cell-associated hemophagocytic lymphohistiocytosis-like syndrome; LN= Lupus nephritis; SLE= Systemic lupus erythematosus

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

Strong Enrollment Execution

- Significant interest from investigators and patients to enroll

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

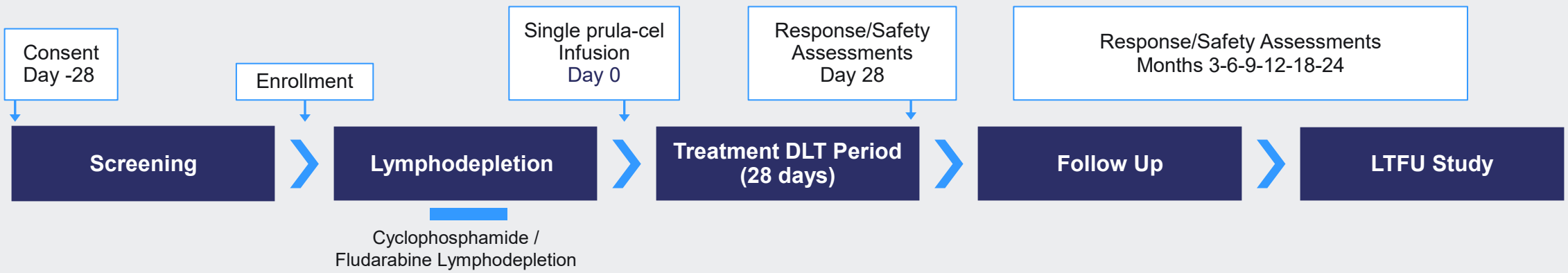
Significant Addressable Market

- 35,000 organ/life threatening LN[1] (US)
- 35,000 organ/life threatening non-renal SLE[2] (US)

[1]Fails to achieve remission after 24 months of standard of care treatment; [2]Patients who have organ/life threatening disease on current standard of care

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Prula-cel Phase 1 Autoimmune Study Design



Part 1: Advancing study in multiple cohorts

LN/SLE

SSc

3+3 design
1E8 starting dose*

Part 2

Dose Expansion Cohorts

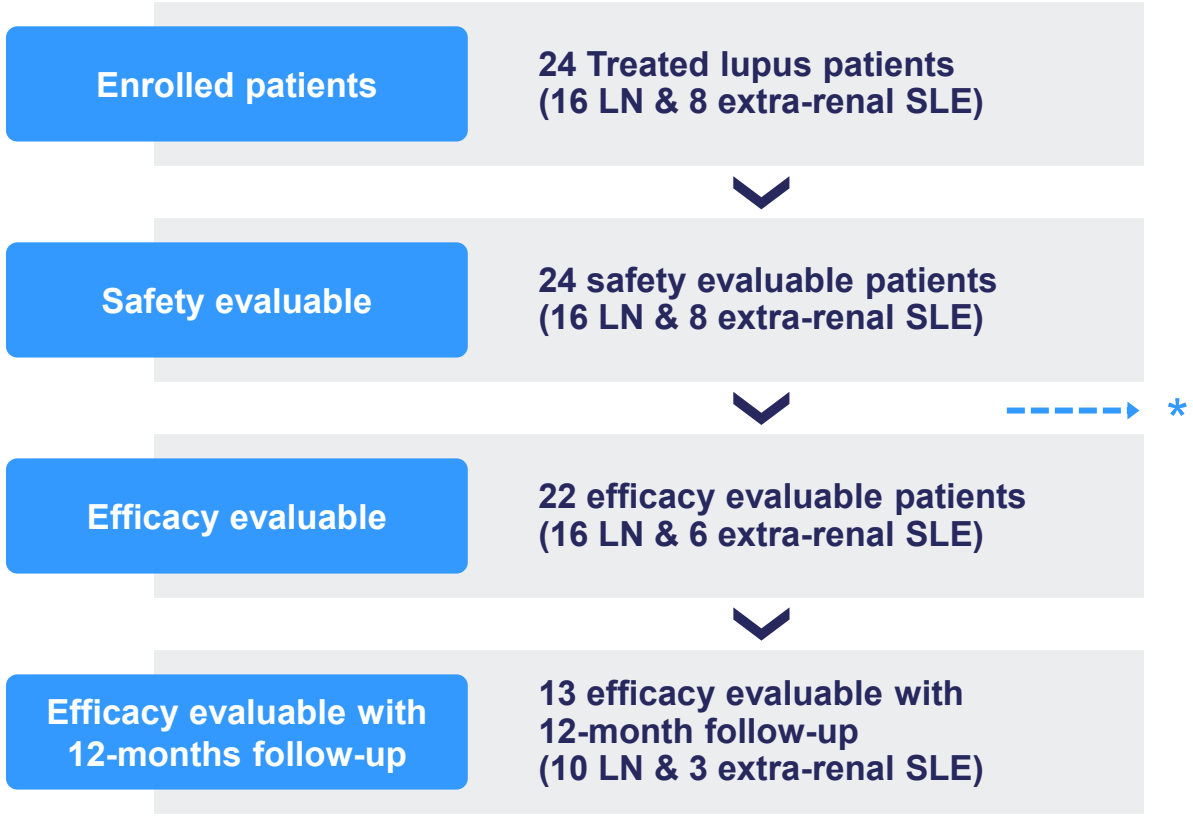
*The starting study dose was amended for all other cohorts to 3E8 with potential to escalate up to 1E9 (based on 3+3 design); DLT= Dose-limiting toxicity; LTFU= Long term follow up

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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

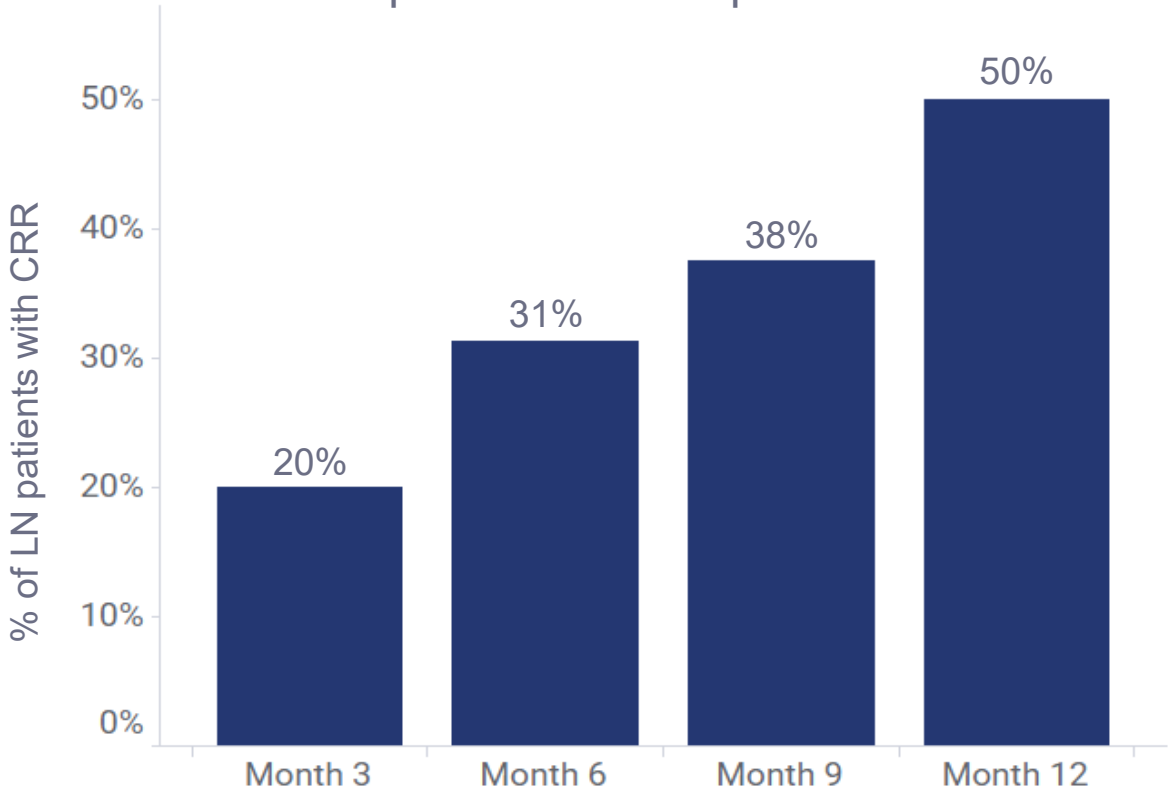
Cut-off date: August 28, 2026; Data cut included 22 efficacy-evaluable and 24 safety-evaluable patients; UPCR= Urine protein-to-creatinine ratio

1. For 22 efficacy evaluable subjects
2. For 16 LN subjects

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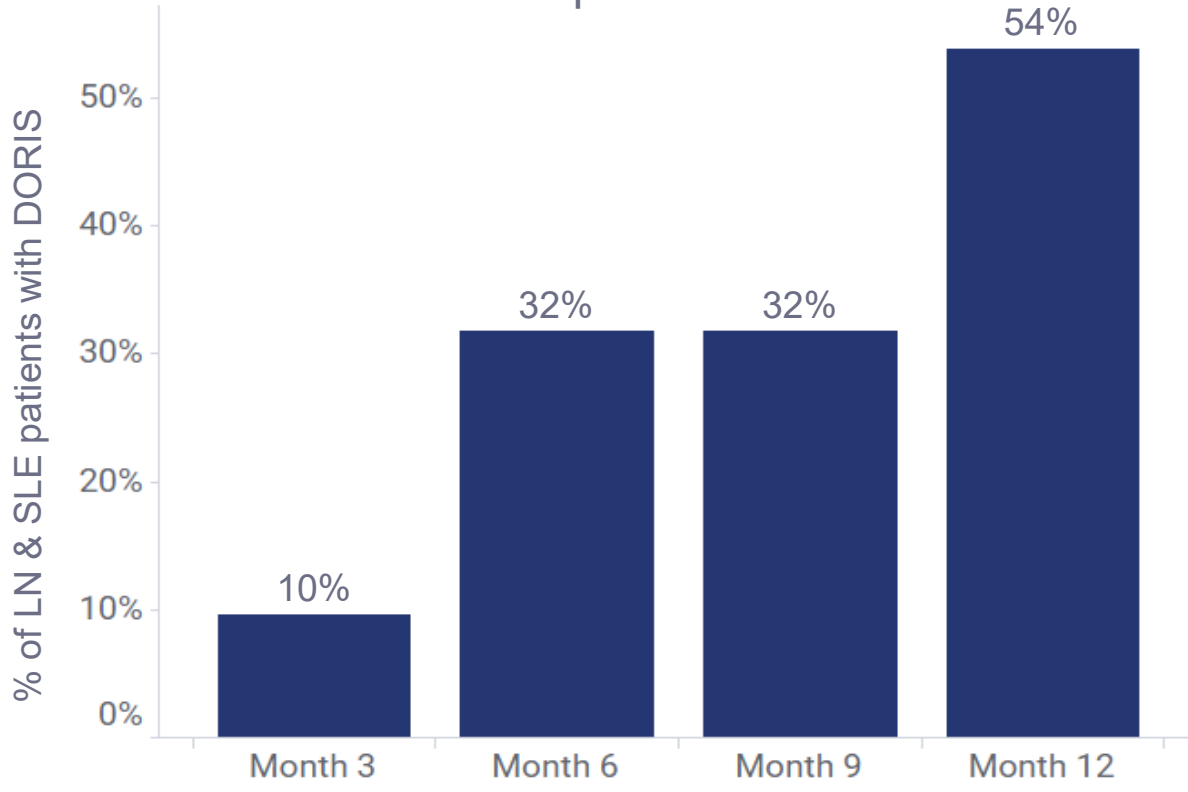
High Rates of Both Complete Renal Response & DORIS Remissions Achieved By 12 Months Post Treatment With Prula-cel

Complete Renal Response in LN



CRR	3	5	6	5
N Subjects	15*	16	16	10

DORIS Response in LN & SLE



DORIS	2	7	7	7
N Subjects	21*	22	22	13

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/1.73m^2 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.

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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)** *LN/SLE, all dose levels*	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
- γδ CAR-T are generally well tolerated compared to αβ CAR-T, likely due to different cytokine profile secreted upon activation as compared αβ CAR-T

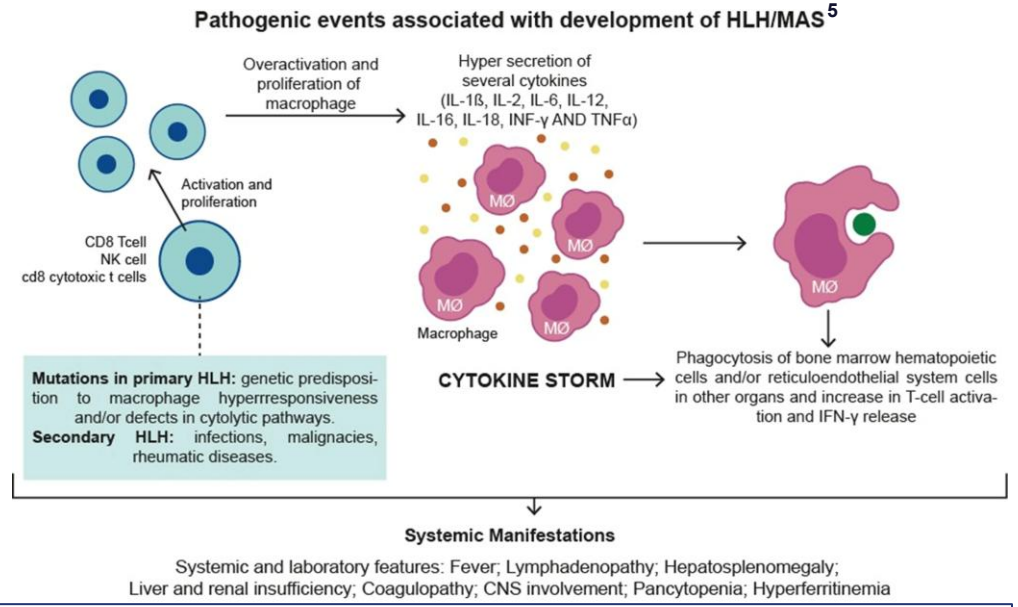
Cut-off date: August 28, 2026; Data cut included 22 efficacy-evaluable and 24 safety-evaluable patients
CRS and ICANS grading criteria: Lee DW. Biol Blood Marrow Transplant.2019 25:625-638; IEC-HS grading: Hines MR. Transplant Cell Ther.2023 29(7):438.e1-16

1. Amoura z et al. EULAR 2026
2. Schett G. et al. ACR 2025
3. Leandro M. et al. EULAR 2026

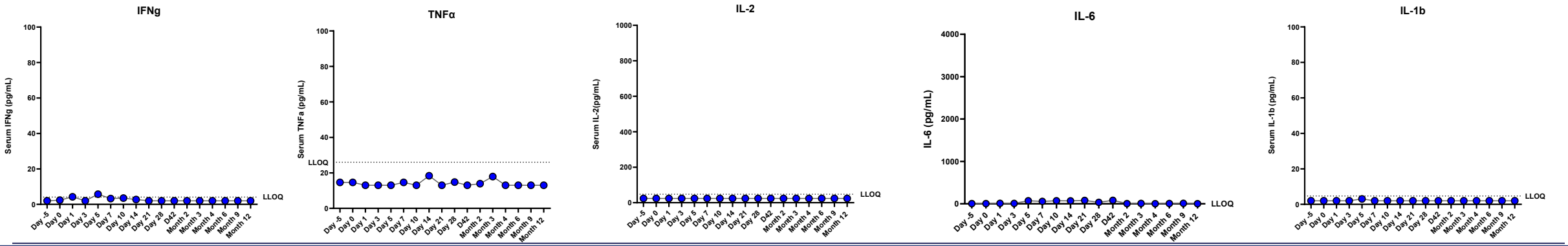
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Prula-cel's Favorable Safety Profile in Autoimmune Compared to a Autologous αβ CAR-T is Rooted in the Biology of γδ1 T Cells

- γδ1 T cells are defined by a clearly differentiated cytokine profile compared to αβ T cells[1-4]
- IEC-HS is primarily related to hyperproliferation of αβ CAR-T and associated with secretion of IFNγ, TNFα, IL-2, IL6, and IL18[5]
- 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



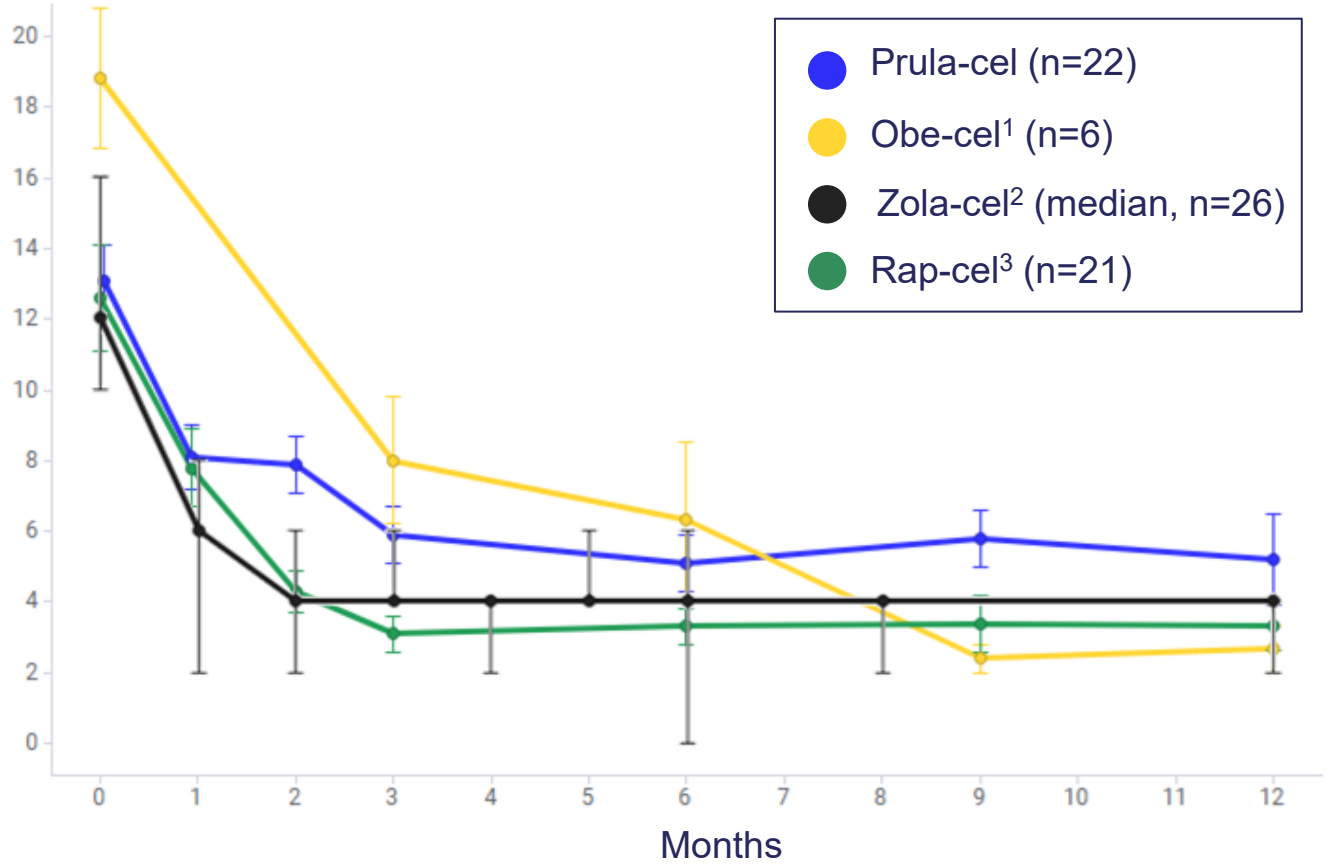
1. Nishimoto, K.P., *et al* (2022), Allogeneic CD20-targeted γδ T cells exhibit innate and adaptive antitumor activities in preclinical B-cell lymphoma models. Clin Transl Immunol, 11: e1373.
2. Nishimoto KP et al. ADI- 270: an armored allogeneic gamma delta T cell therapy designed to target CD70- expressing 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 γδ T Cells. Res Sq [Preprint]. 2026
5. Sztajnbok, 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 αβ CAR-T to prula-cel.

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SLEDAI Decline With Prula-cel In-Line With That Observed With Autologous αβ 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

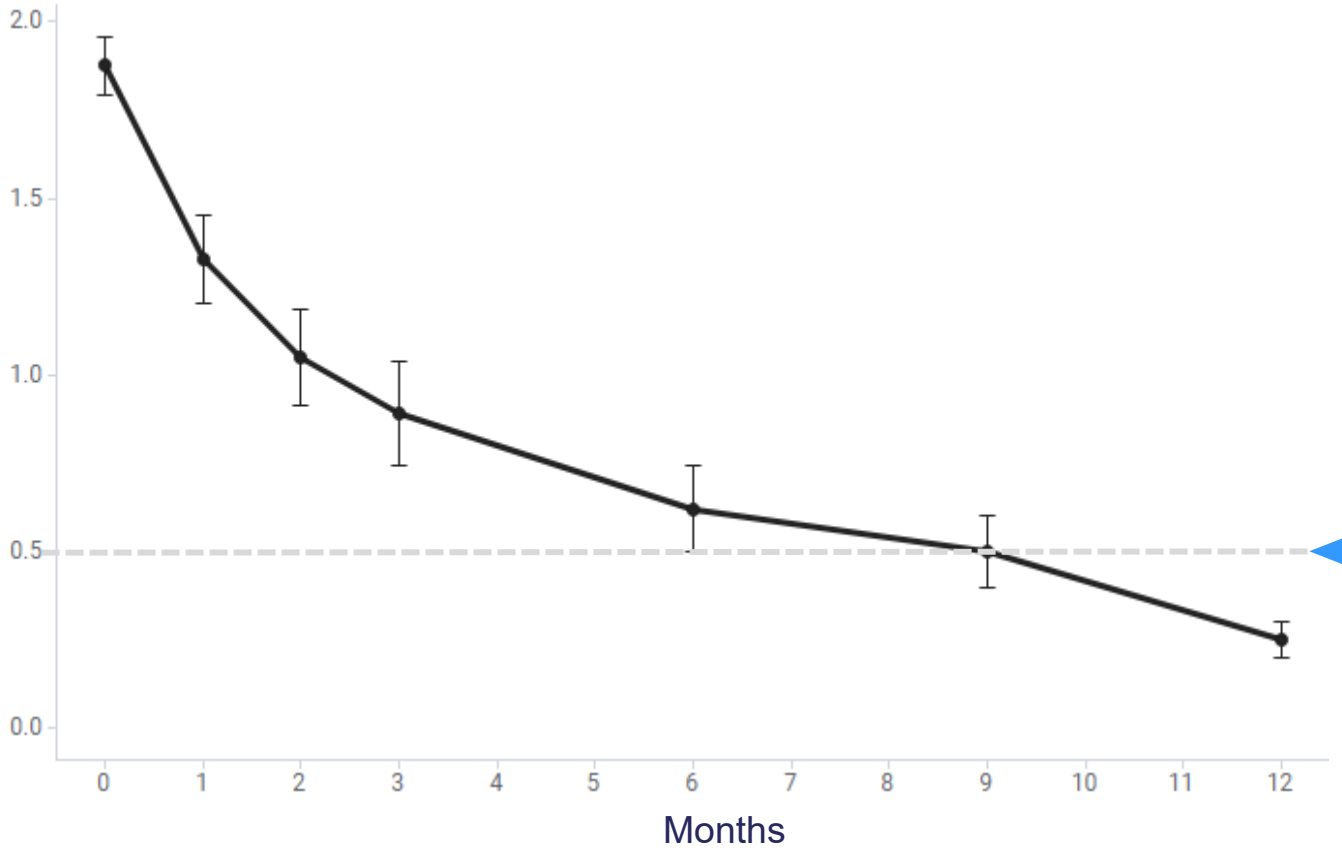
Cut-off date: August 28, 2026; Data cut included 22 efficacy-evaluable patients; Based on historical published data. No head-to-head studies have been conducted and cross trial comparisons may not be reliable due to difference in molecule composition, trial design and patient population and characteristics.

1. Leandro M. et al. EULAR 2026
2. Schett G. et al. ACR 2025
3. Amoura z et al. EULAR 2026

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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

Cut-off date: August 28, 2026; Data cut included 22 efficacy-evaluable patients

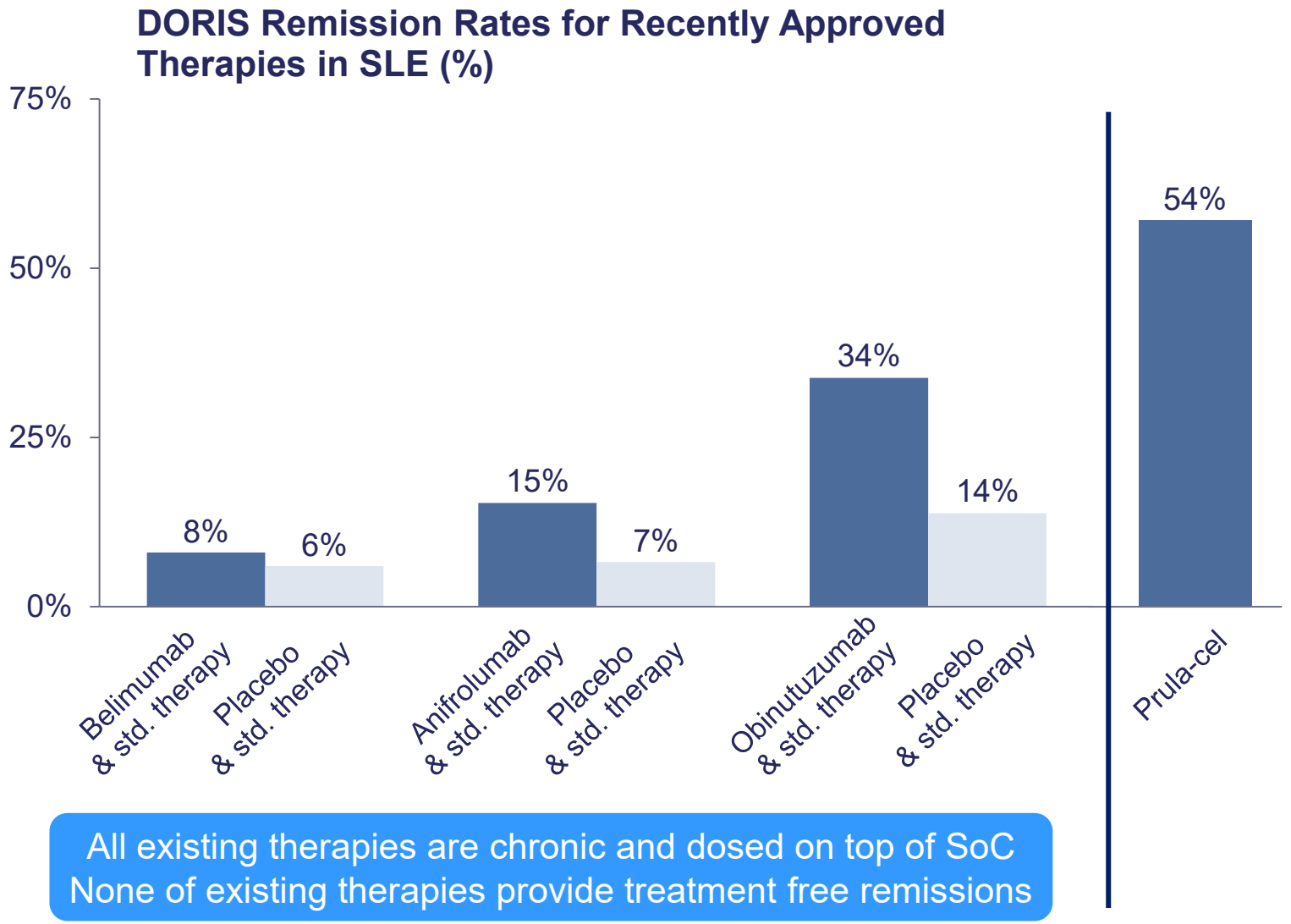
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Patients Discontinued Immunosuppressants and Tapered Steroids to ≤ 5 mg Prednisone Equivalent

Immunosuppressants	All patients discontinued immunosuppressants
Steroids	Background steroids tapered to ≤ 5mg prednisone in all patients but one

Cut-off date: August 28, 2026; Data cut included 22 efficacy-evaluable patients

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



All existing therapies are chronic and dosed on top of SoC
None of existing therapies provide treatment free remissions

Parodis I. et al. Lancet Rheumatol (2024)
Morand EF et al. Annals of the Rheumatic Diseases (2023)
Furie et al. EULAR 2026

Cut-off date: August 28, 2026; Data cut included 22 efficacy-evaluable patients; SoC = Standard of Care; Based on historical published data. No head to head studies have been conducted and cross trial comparisons may not be reliable due to difference in molecule composition, trial design and patient population and characteristics

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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

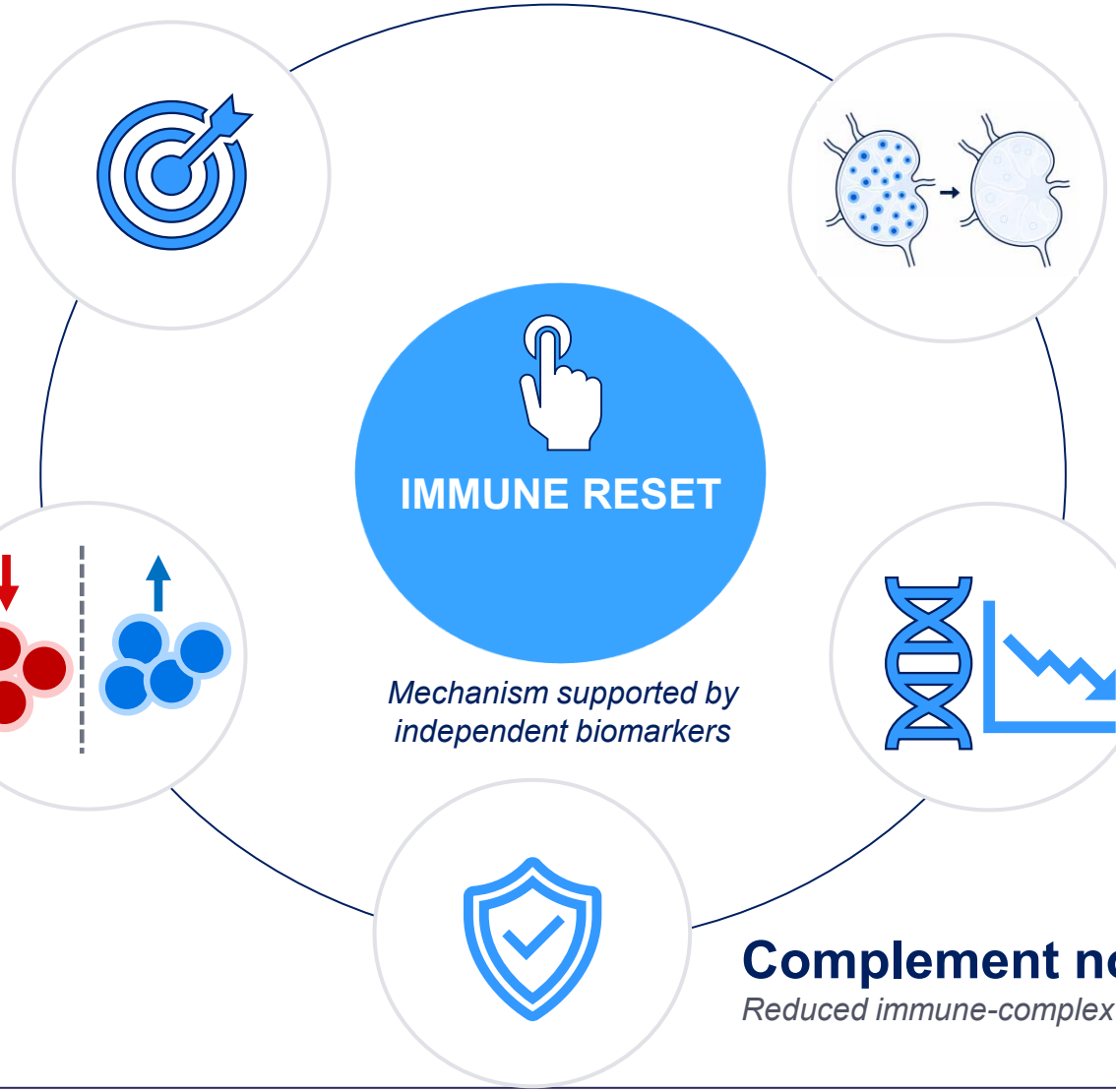
Next steps

- 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
Undetectable B cells in 100% (22/22) of evaluable patients

Tissue B-cell depletion
Target engagement confirmed



Mechanism supported by independent biomarkers

Memory B-cell clearance & naïve recovery
Naïve repertoire emerges

↓ Anti-dsDNA
Reduced autoimmune activity

Complement normalization
Reduced immune-complex burden

Cut-off date: August 28, 2026; Data cut included 22 efficacy-evaluable patients
Anti-dsDNA= Anti-double-stranded DNA

Tissue B-cell depletion demonstrated in other disease settings. Biopsies not available in the LN/SLE study

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Deep B-cell Depletion with Naïve Recovery

KEY TAKEAWAY

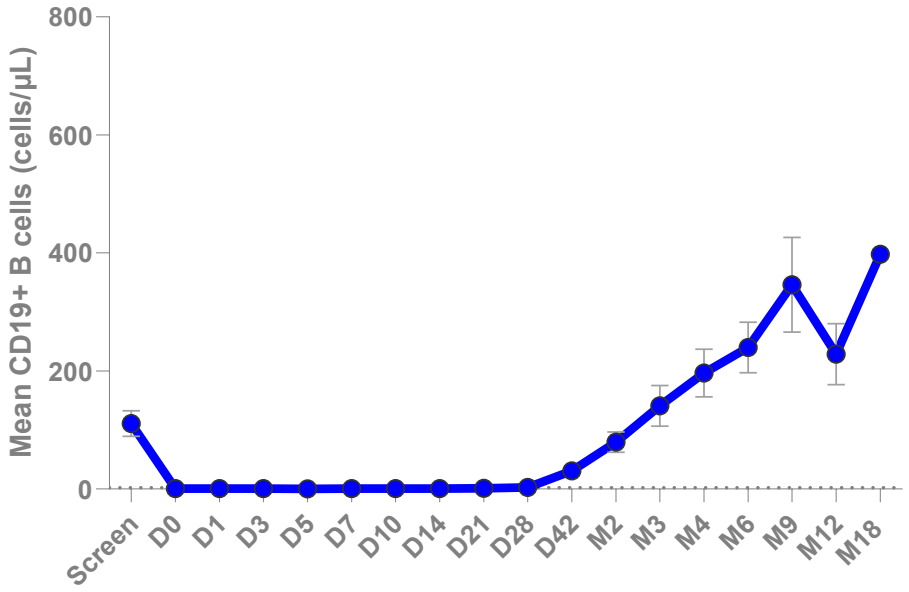
- **Deep B-cell depletion**
- **Naïve dominant Recovery ~1-3 months**
- **Kinetics aligned with leading CAR-T data**
- **Immune reset**

100%
(22/22)
evaluable patients

Achieved Undetectable CD19+ B cells

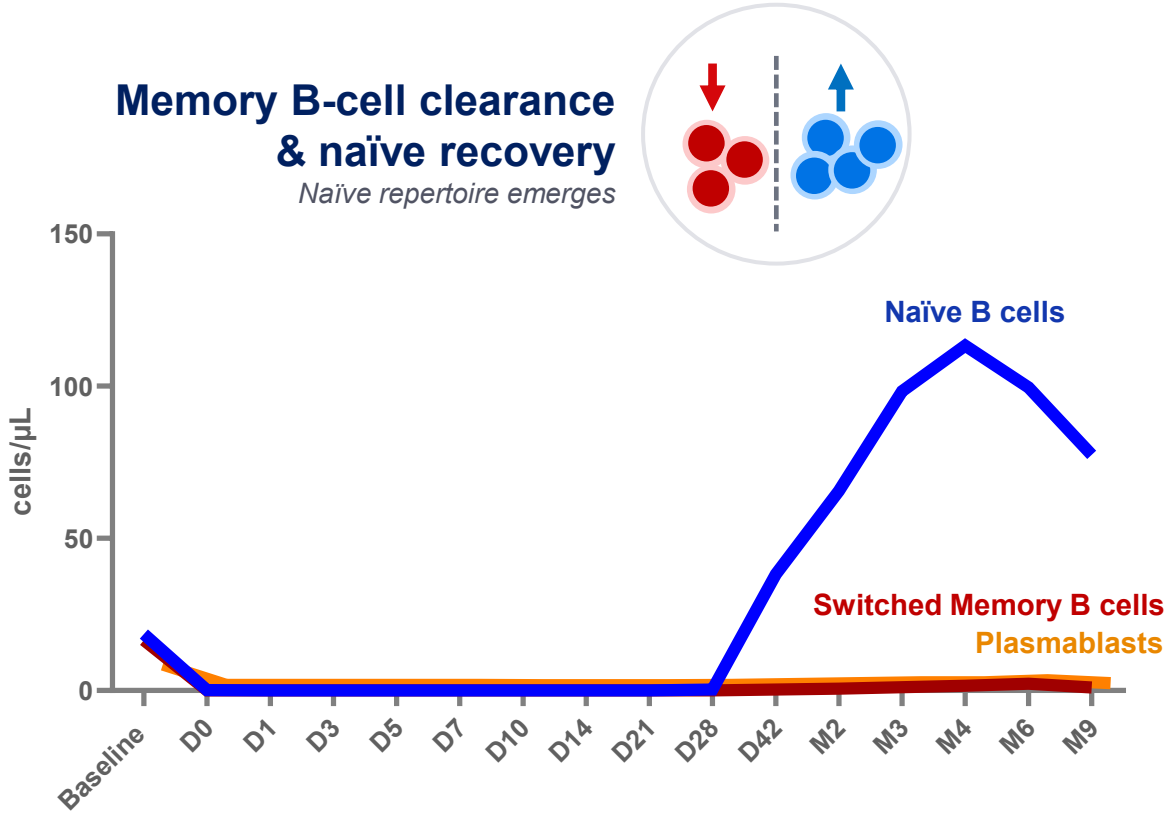
CD19⁺ B cell Depletion

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 = $CD19^{+}CD20^{+/-}IgD{-}CD27^{high}CD38^{high}CD138^{-}$.

Cut-off date: August 28, 2026; Data cut included 22 efficacy-evaluable patients

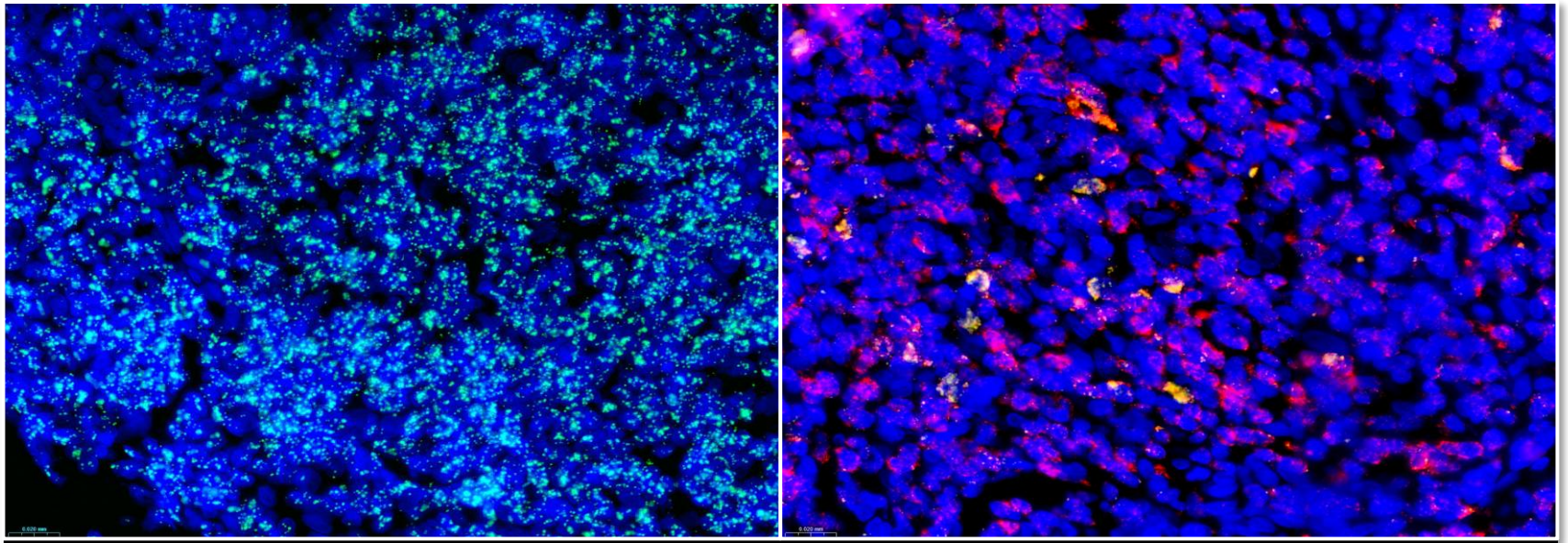
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Prula-cel Achieved Deep CD19+ B-cell Depletion in Tissues

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

MCL PATIENT LYMPH NODE BIOPSIES

Baseline | **Day 10 Post Dose**



Blue : Nuclei | Green: CD19+ cells | Yellow: CAR | Red: Granzyme B (T Cell Activation)

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

MCL = Mantle cell lymphoma; 73y M, 2 prior lines (including SCT), 1E9 Dose Level CR

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

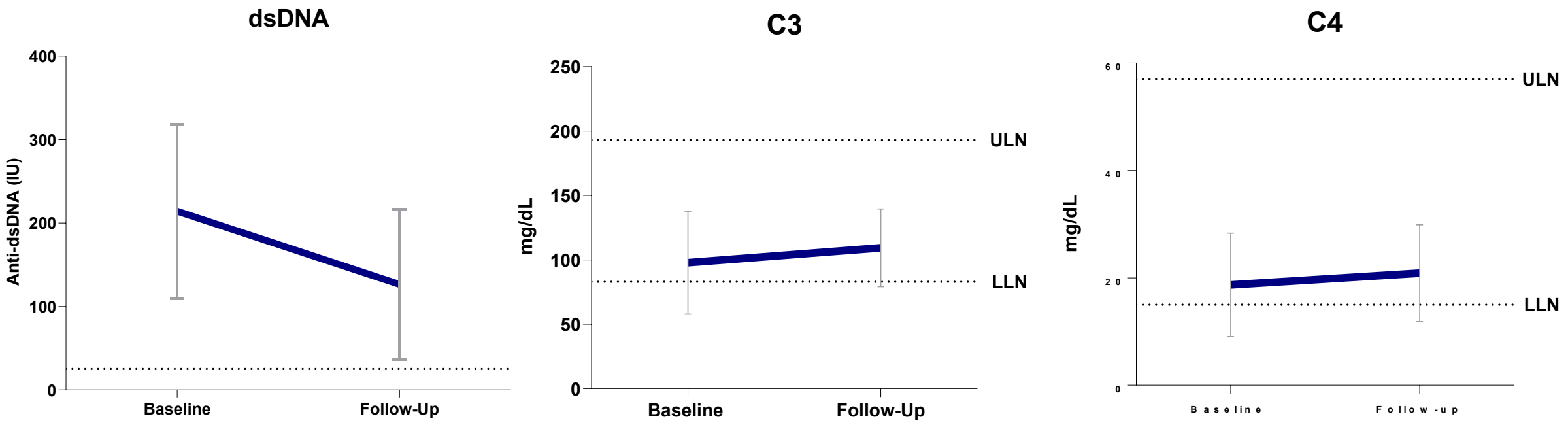
Key Findings

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.

Cut-off date: August 28, 2026

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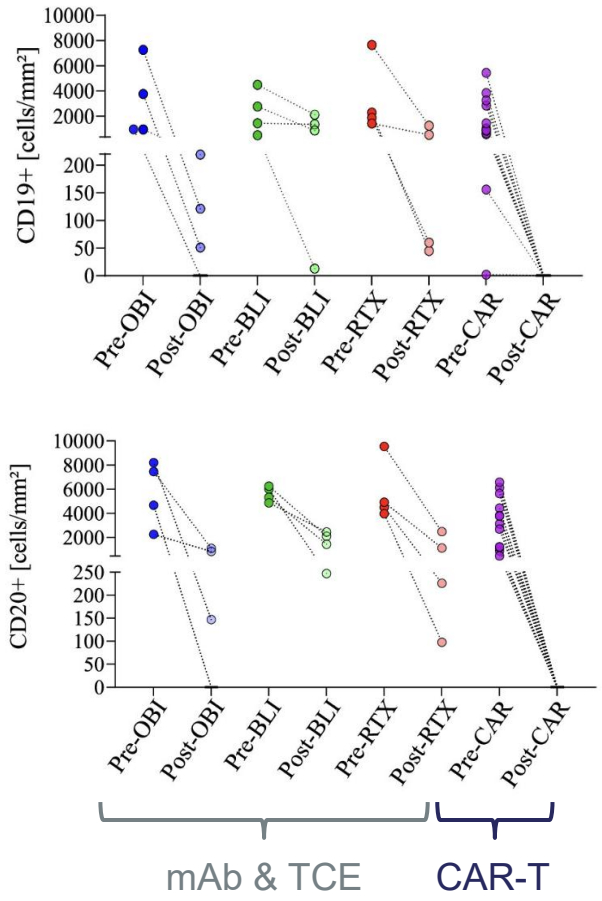
Prula-cel: Designed for Broad Adoption and Scalable Commercialization

	Autoimmune CAR-T	
	AUTOLOGOUS CAR-T *Current paradigm*	**Prula-cel** *Allogeneic / bank-ready γδ1 CAR-T*
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

Prula-cel product profile based on cut-off date: August 28, 2026; Data cut included 22 efficacy-evaluable patients

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



mAb & TCE	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-T	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

RTX = rituximab, BLI = blinatumomab, OBI = obinutuzumab; Tur C et al. Annals of the Rheumatic Diseases (2025); Based on historical published data. No head to head studies have been conducted and cross trial comparisons may not be reliable due to difference in molecule composition, trial design and patient population and characteristics

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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

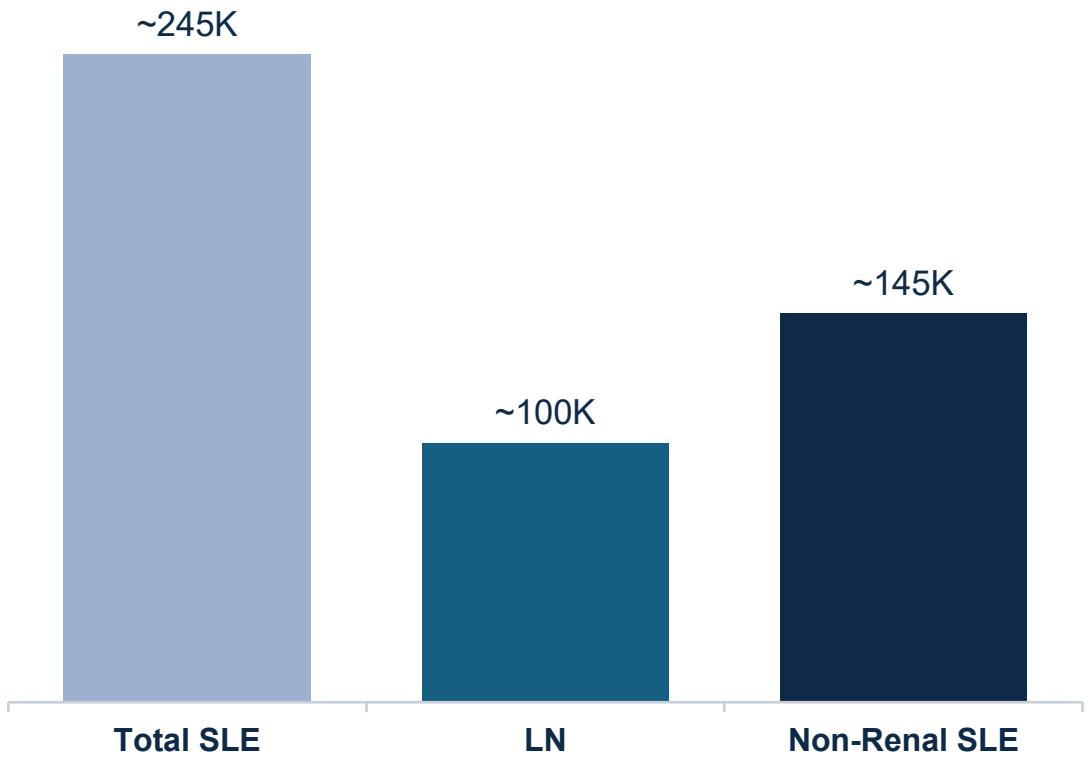
- Deliver gene therapy to stay “forever” with unknown risk (e.g. insertional mutagenesis)
- May experience similar safety challenges to autologous CAR-T
- Have not demonstrated efficacy comparable to autologous CAR-T

Chen YH et al. NEJM (2026)
Wang Q et al. NEJM (2025)

Prula-cel target product profile based on cut-off date: August 28, 2026; Data cut included 22 efficacy-evaluable patients

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

Helmick CG et al. Arthritis & Rheumatism (2007) | Morales E et al. Nephron (2021)

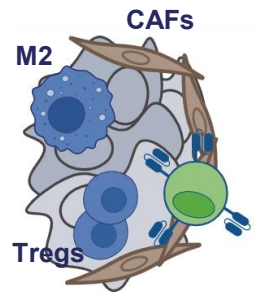
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ADI-212

Prostate Cancer

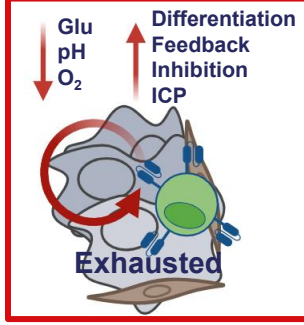
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Current Challenges for Existing Cell Therapies in Solid Tumors



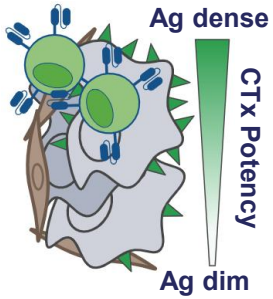
1 – Nonpermissive TME

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



2 – Effector Cells Become Fatigued

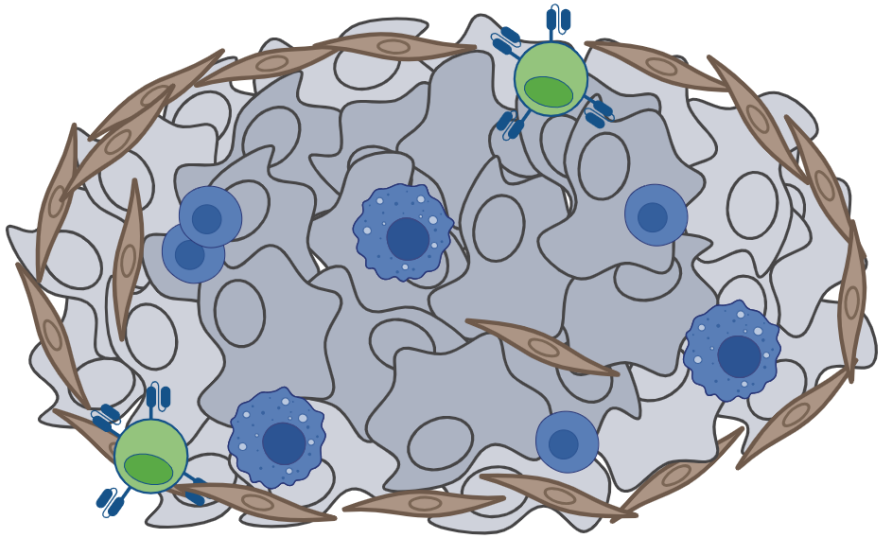
- Tumor cells express inhibitory receptors/factors
- Metabolic stress exhausts effector cells
- Feedback loops inhibit repetitive killing capacity



3 – Target Insufficiency

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

Immunosuppressive Tumor

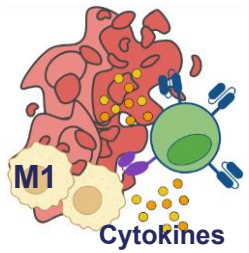


Impairment of CAR-T cell efficacy

TME, Tumor Microenvironment; CAF, Cancer associated fibroblasts; M2, M2 Macrophage; Ag, Antigen

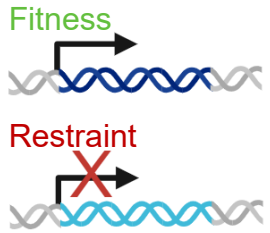
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Three Strategic Pillars for Overcoming Solid Tumor Barriers



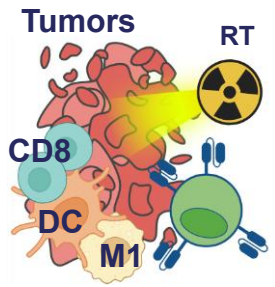
1 – Localized Cytokine Armoring

- Transform to active and permissive immune TME
- Intrinsic and extrinsic immune activation
- Controlled[1] and localized[2] exposure



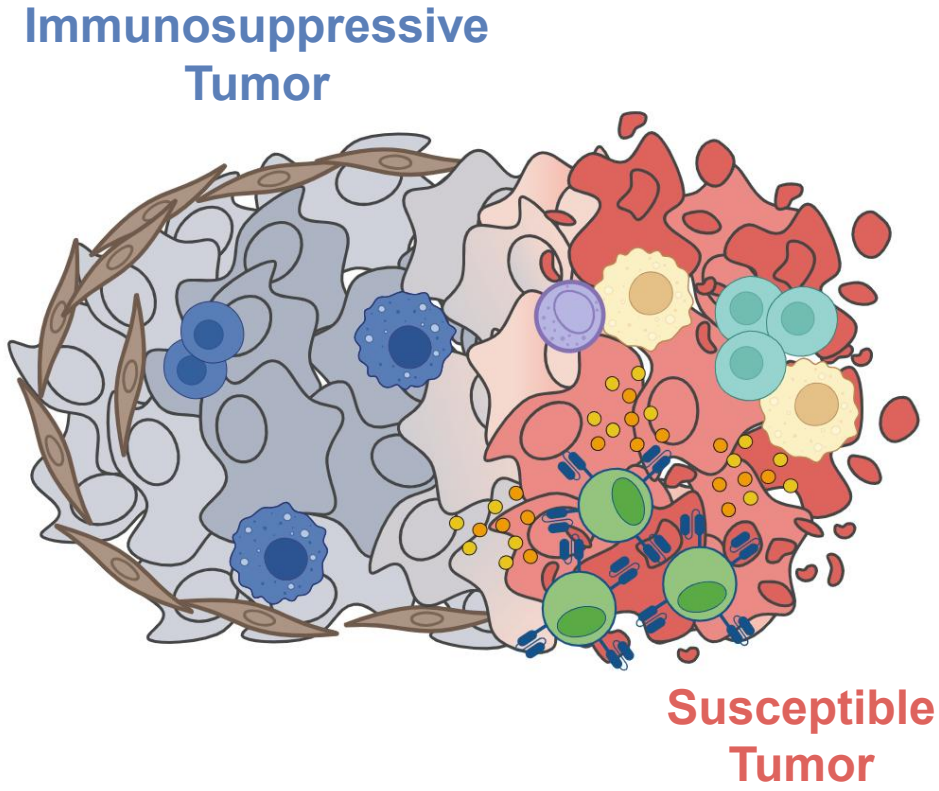
2 – Transcriptional Tuning

- Tune transcriptional controls to enhance T cell fitness[3] and relieve restraints[4]
- Reduced exhaustion to increase persistence



3 – Target & Tumor Engagement

- Direct-antigen targeting
- Increased immune cell recruitment and antigen spreading
- Neoantigens from radioligand therapy[5]



TME, Tumor Microenvironment; CTx, Existing Cell Therapies; TF, Transcription factor; M1, M1 Macrophages; DC, Dendritic cells; RT, Radiation therapy

[1] Chen et al. Nature 2025. Rewiring endogenous genes in CAR T cells for tumour-restricted payload delivery. Nature 2025 [2] Lopez et al. Mol Ther Adv 2026. IL-12-engineered human PSMA-CAR T cells for the treatment of advanced prostate cancer
[3] Chan et al. Nature 2024. FOXO1 enhances CAR T cell stemness, metabolic fitness and efficacy [4] Chen et al. JCI Insight 2025. Enhancing the potency of CAR-T cells against solid tumors through transcription factor engineering [5] Liu et al. bioRxiv 2026. Radioligand therapy in combination with CAR T cells overcomes the heterogeneous immunosuppressive prostate tumor microenvironment

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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

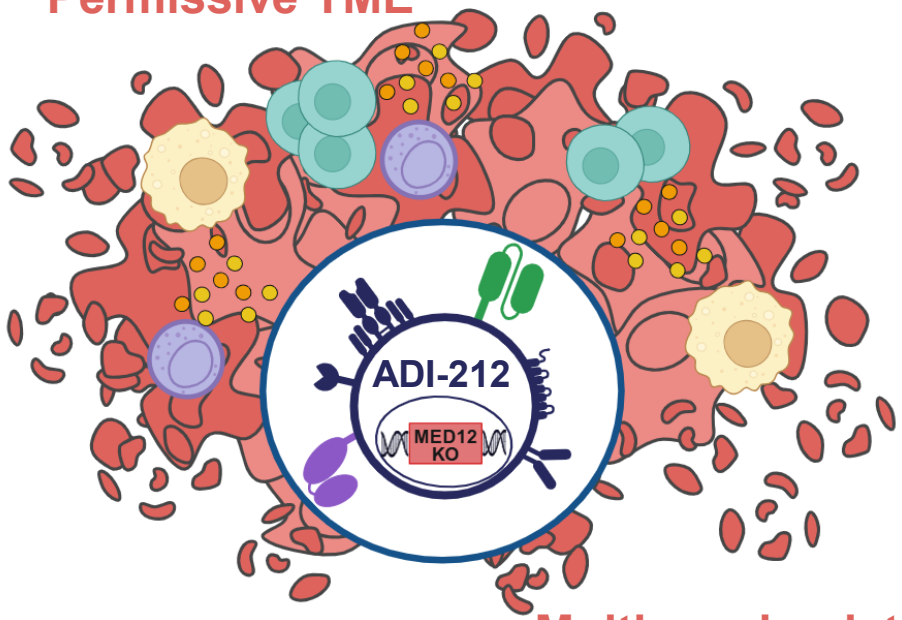
- MED12 mediates transcription
- CRISPR KO of MED12
- Greater T-cell stemness and reduced modulation
- Improved persistence of cytotoxicity



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

TME, Tumor Microenvironment; CRISPR, Clustered Regularly Interspaced Short Palindromic Repeats; KO, Knockout; PSMA, Prostate-Specific Membrane Antigen

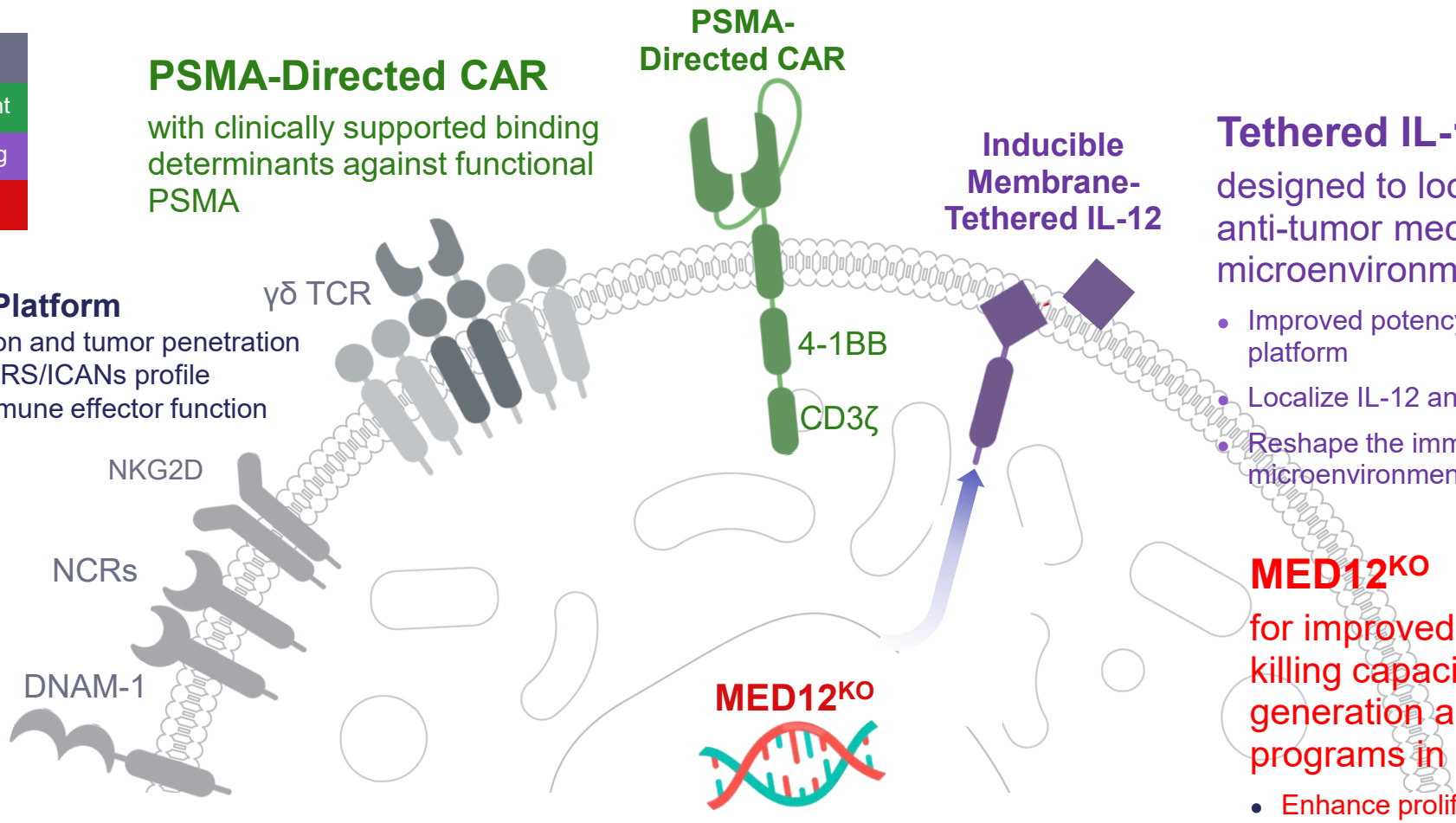
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ADI-212 Designed To Address Multiple Barriers in a Single Off-the-Shelf Product

Adicet's γδ T Platform
Target & Tumor Engagement
Localized Cytokine Armoring
Transcriptional Tuning

PSMA-Directed CAR

with clinically supported binding determinants against functional PSMA



Adicet's γδ1 CAR-T Platform

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

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}

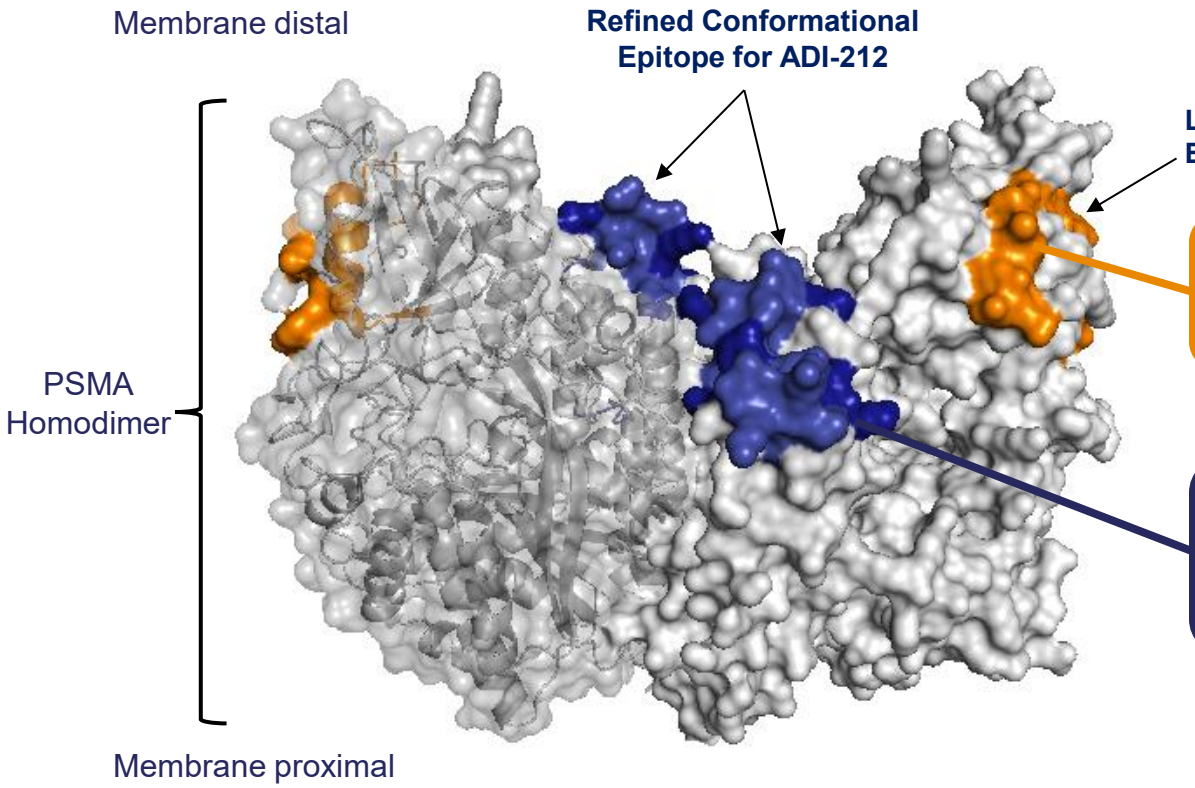
for improved potency and tumor-cell killing capacity compared to previous generation alpha-beta and γδ CAR-T programs in oncology

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

CAR, Chimeric antigen receptor; KO, Knockout; PSMA, Prostate-Specific Membrane Antigen; NCR, Natural cytotoxicity receptors; CRS, Cytokine Release Syndrome, ICANS, Immune Effector Cell-Associated Neurotoxicity Syndrome

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



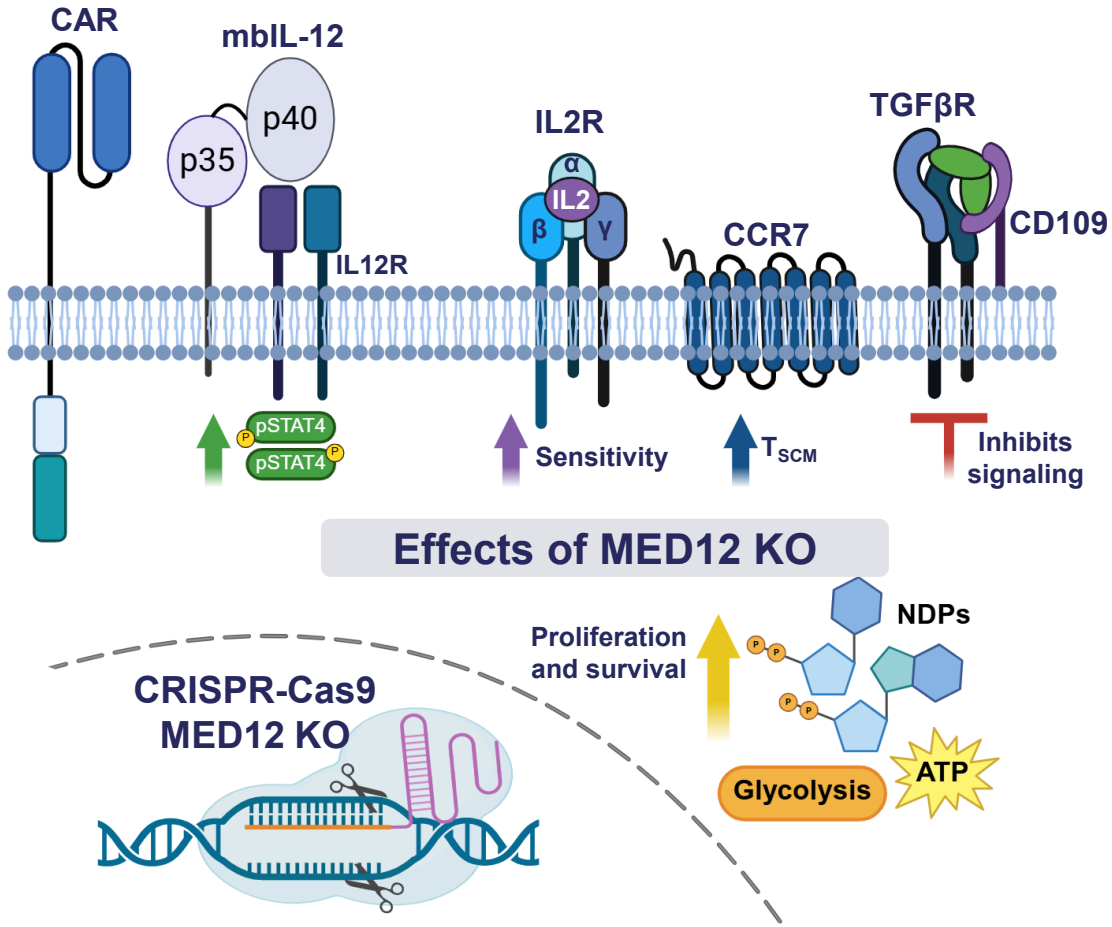
Benchmark epitope from αβ CAR T associated with toxicities and lower efficacy across modalities

ADI-212 binder recognizes functional PSMA homodimer and demonstrated less tissue cross-reactivity when compared to benchmark binder for αβ PSMA-CAR T

Ramadoss, Nitya S., Elizabeth Speltz, Tonya Capillo, et al. *Molecular Cancer Therapeutics* 22, no. 12_Supplement (2023): C117.

Adicet Bio

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

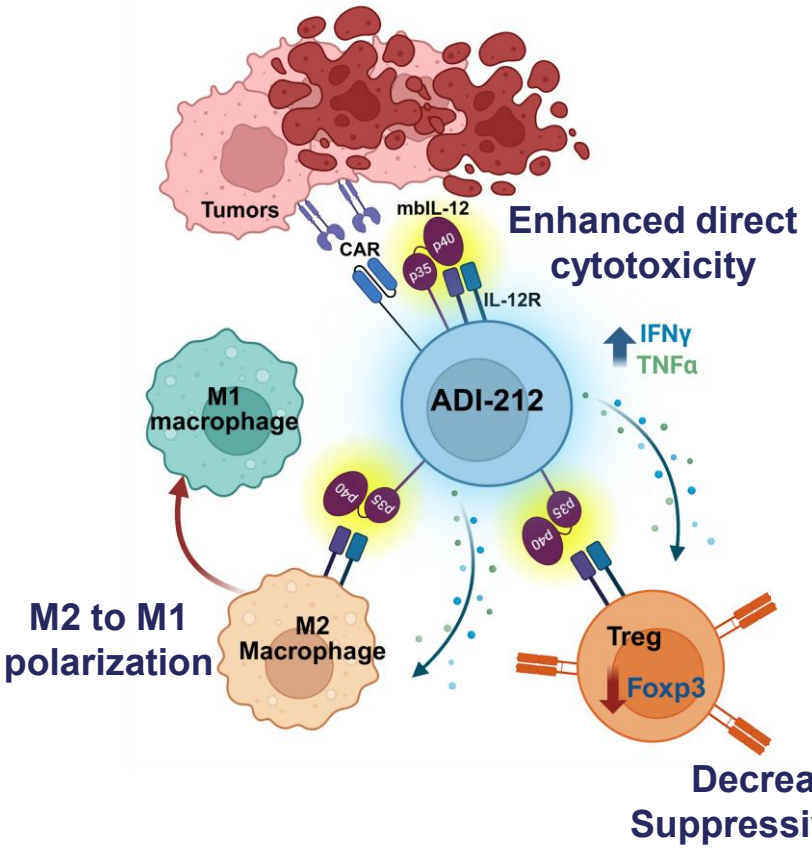
Synergy

MED12 KO

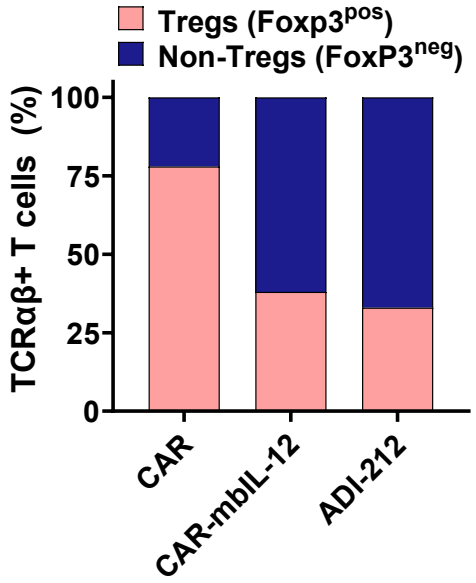
TME, Tumor Microenvironment, CAR, Chimeric antigen receptor; CRISPR, Clustered Regularly Interspaced Short Palindromic Repeats; KO, Knockout
ATP, Adenosine triphosphate, NDP, Nucleotide diphosphate, Tscm, Stem cell memory T cells, TGFβR, Transforming growth factor-beta receptor

Adicet Bio

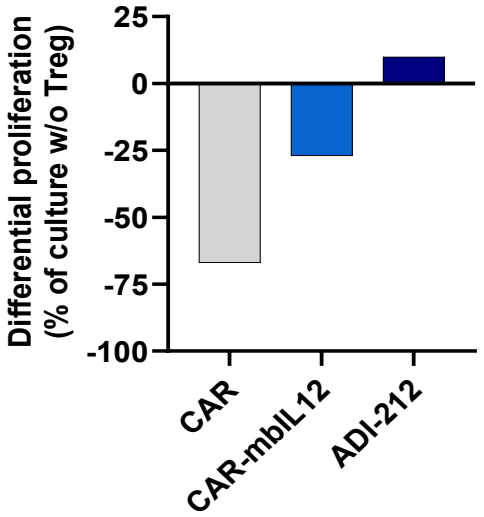
ADI-212 Capacity to Reshape Tumor Microenvironments and Maintain Potent Anti-Tumor Killing in the Presence of Immunosuppressive Cells



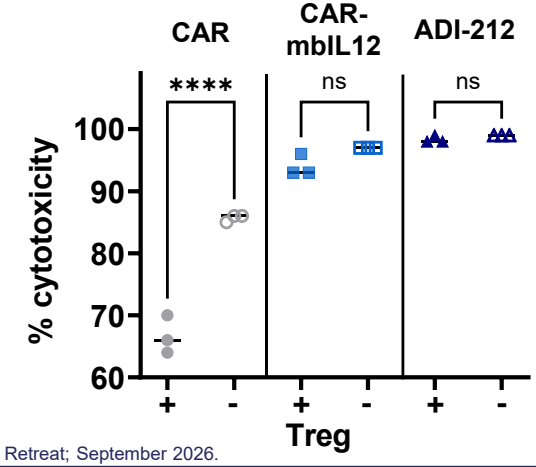
1 mbIL-12 on ADI-212 modulates Treg Population in Cocultures



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; IFNγ, Interferon-gamma; TNFα, Tumor necrosis factor-alpha;

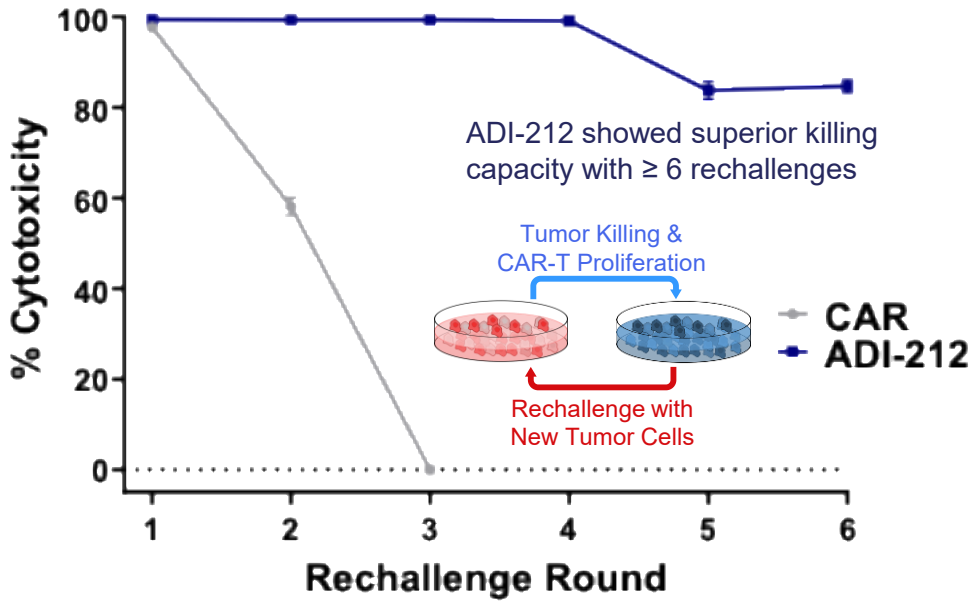
Herrman, M. Aftab, B, et al. presented at: Prostate Cancer Foundation Scientific Retreat; September 2026.

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.

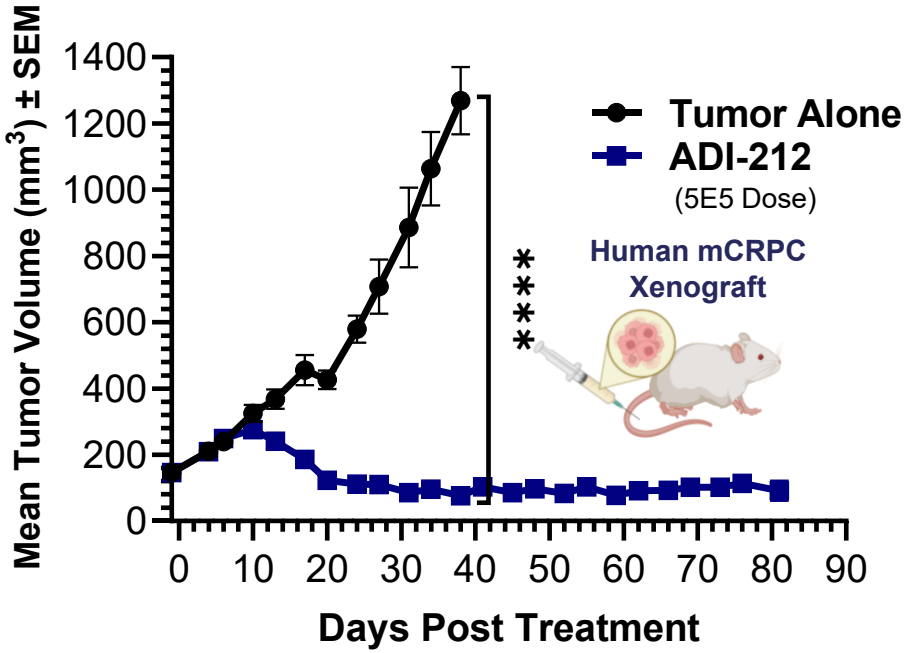
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ADI-212 Demonstrated Highly Potent Preclinical Activity in Solid Tumors

Durable In Vitro Cytotoxicity Demonstrated Against PSMA+ Prostate Cancer Cells



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



mCRPC, Metastatic Castration Resistant Prostate Cancer; PSMA, Prostate-Specific Membrane Antigen
Herrman, M. Aftab, B, et al. presented at: Prostate Cancer Foundation Scientific Retreat; September 2026
3:1 E:T for rechallenge assessments

Adicet Bio

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



Inclusion/ Exclusion Criteria

- Adenocarcinoma of the prostate
- Metastatic castrate-resistant prostate cancer
- Target lesions positive for PSMA
- Prior Pluvicto is allowed
- Prior CAR-T is not allowed
- ECOG 0 or 1

End Points

Safety, RP2D, PCWG3 evaluation (bone scan, CT/MRI scan, PSA levels)

Study Design

- 3+3 Dose escalation
- Backfills to doses deemed safe
- Potential for redosing

CT= Computed Tomography; DLT= Dose limiting toxicity; ECOG= Eastern Cooperative Oncology Group Performance Status scale; LTFU= Long-term follow up; MRI= Magnetic resonance imaging; PCWG3= Prostate Cancer Clinical Trials Working Group 3; PSA= Prostate-specific antigen; RP2D= Recommended Phase 2 dose

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In Vivo CAR-T Portfolio

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

Hematologic malignancies and solid tumors

Adicet Bio

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

Adicet Bio

Developing Broad Pipeline of Allogeneic γδ1 CAR-T Cell and Best In Class Differentiated In Vivo CAR-T Therapies

Off-The-Shelf γδ1 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

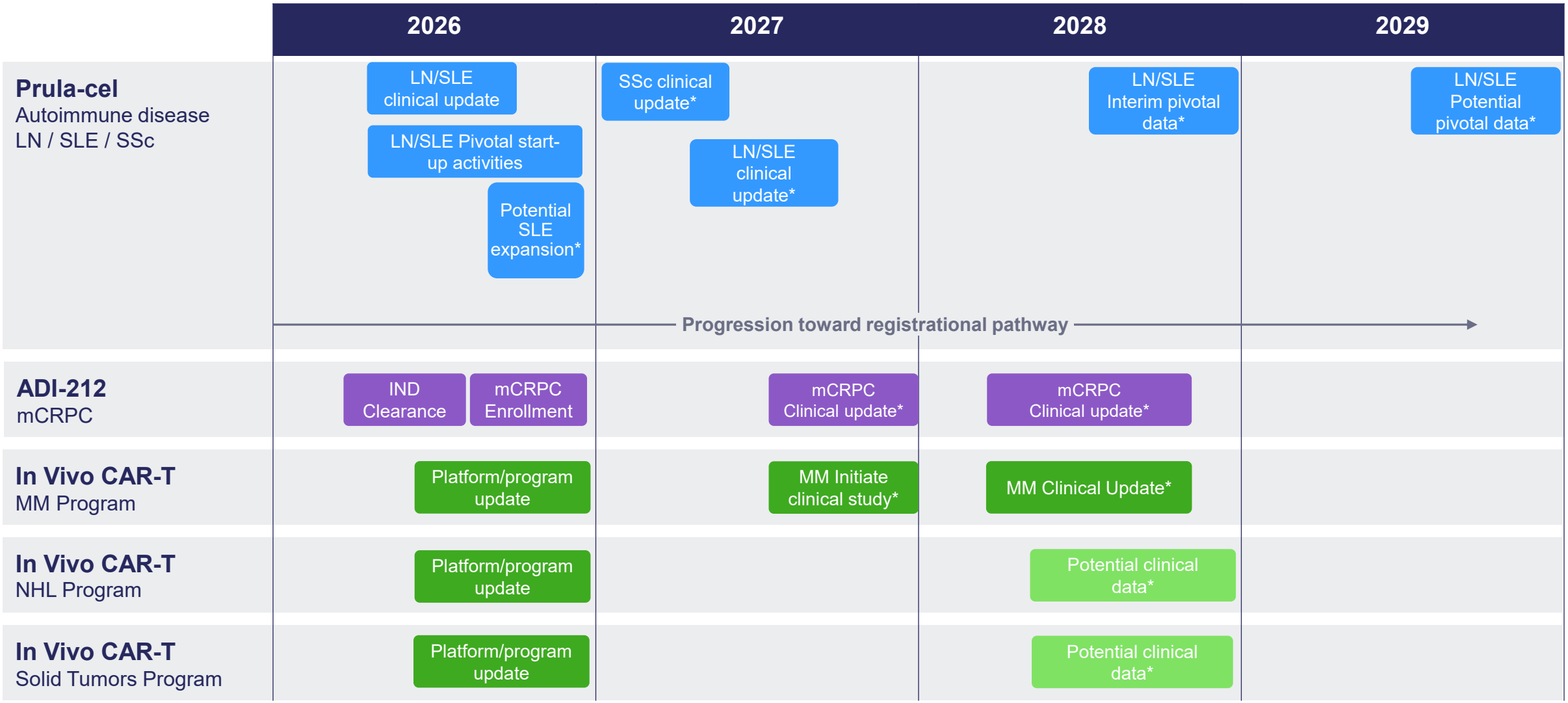
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Multiple Myeloma Target: BCMA Mono / Dual CAR	MM	PRE-CLINICAL			• Differentiated CAR design with effective BCMA targeting • Decreased susceptibility to immune-mediated inactivation and clearance
NHL Target: CD19 Dual CAR	NHL	PRE-CLINICAL			• Differentiated 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

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Anticipated Near-Term Value-Creating Milestones

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



*Achievement and timing of forward-looking milestones subject to clinical progress, regulatory outcomes, financing ability and other business considerations

Adicet Bio

Investor Presentation

October 2026

Adicet Bio