Investor Overview

September 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; 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 LN or LN and SLE patients; the preclinical and clinical development of ADI-212, including the timing of regulatory filings, clinical startup activities, clinical updates and future data releases; the timing of initiation of enrollment of a Phase 1 trial for ADI-212 in mCRPC; expectations regarding the potential potency of ADI-212; timing of updates to the Company’s *in vivo* CAR T platform pipeline; ongoing preclinical programs and activities relating to autoimmune diseases, hematological malignancies and solid tumors; and expectations regarding Adicet’s uses of capital, expenses and financial results, including the expected cash runway. 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.

Industry and Market Information

Information regarding market share, market position and industry data pertaining to Adicet’s business contained in this presentation consists of estimates based on data and reports compiled by industry professional organizations and analysts and Adicet’s knowledge of their industry. Although Adicet believes the industry and market data to be reliable, this information could prove to be inaccurate. You should carefully consider the inherent risks and uncertainties associated with the market and other industry data contained in this presentation. Forward-looking information obtained from third-party sources is subject to the same qualifications and the additional uncertainties as the other forward-looking statements in this presentation.

Adicet Bio: Positioned to Deliver High Value Milestones

Clinical data Sept 2026

Prula-cel: Poised to Become the First Off-the-Shelf, Easy to Administer, One-Time, CAR-T Cell Therapy for Autoimmune Disease

- Bar for success
- Transformative therapeutic and market opportunity

Clinical data 1H/2027 & 2H/2027

ADI-212: IND Cleared; Advancing into Clinic for mCRPC

- Next-generation, gene-edited, armored cell therapy candidate designed to address the complexity of solid tumors
- Plan to initiate clinical enrollment 4Q/2026

Entering clinic 2H/2027

Advancing Diversified In Vivo CAR-T Portfolio Towards Clinical Development

- Programs targeting multiple myeloma, NHL and solid tumor indications
- First candidate entering clinical development 2H/2027

Prula-cel: prulacabtagene leucel
CAR= chimeric antigen receptor; IND= Investigational new drug; mCRPC= metastatic castrate-resistant prostate cancer; NHL= Non-Hodgkin lymphoma

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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	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 CRS / ICANS risk* • Efficacy comparable to autologous CAR T*
	SSc				
ADI-212 Target: PSMA (gene-edited w/ armor) [1]	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	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;[1] License agreement with CRISPR for gene-editing technology. CRISPR has opt-in right to participate in a 50/50 cost and profit split; *Cut-off date: August 31, 2025; Data cut included 7 patients (5 LN and 2 SLE) with a follow-up ranging from 2-9 months

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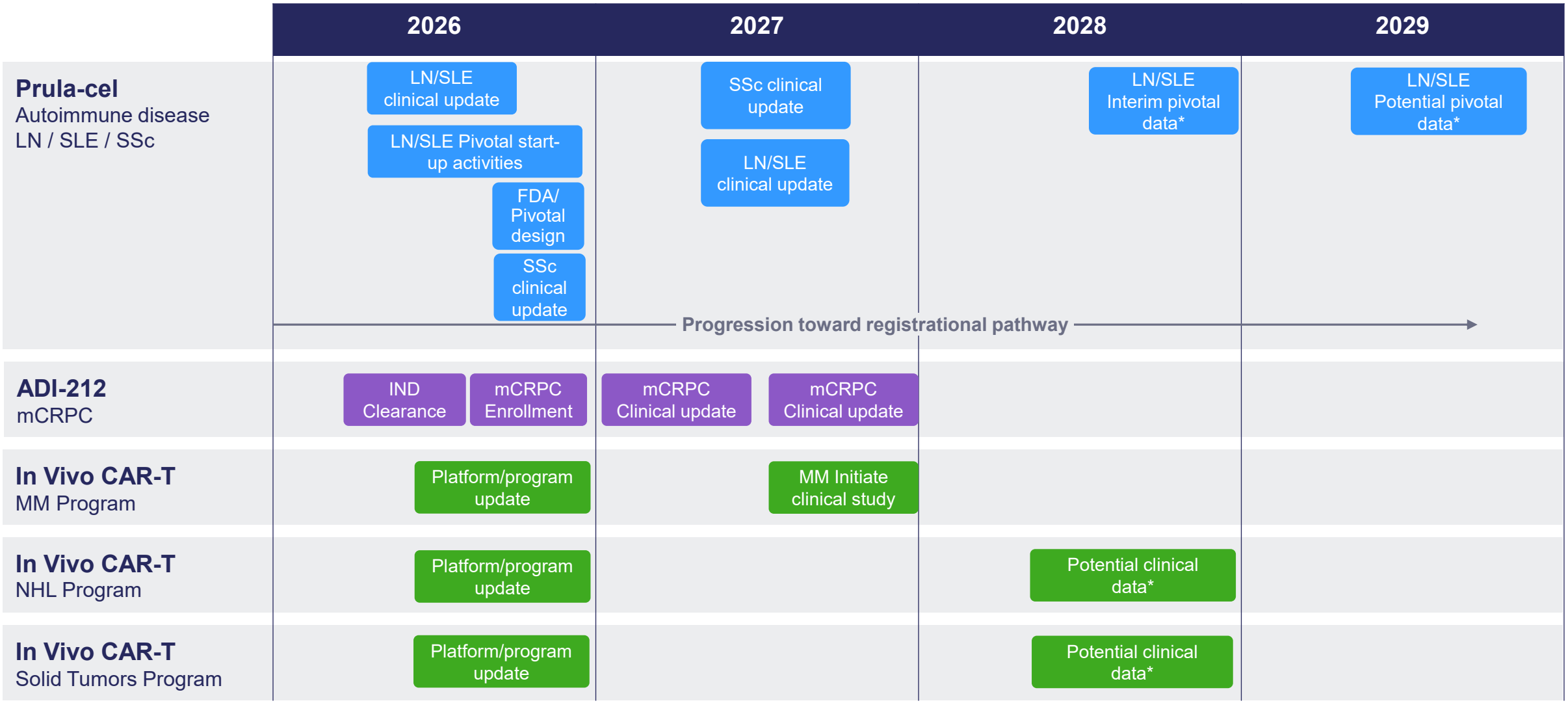
Anticipated Near-Term Value-Creating Milestones

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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Prula-cel

Bar for success to generate transformative market opportunity in patients with SLE with or without lupus nephritis

Autoimmune Indications

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Prula-cel: Potentially First Off-the-Shelf, Easy to Administer, One-Time Therapy With Efficacy Comparable to Autologous CAR-T & Favorable Safety Profile*

- Autologous CAR-T therapies have demonstrated compelling efficacy in patients with autoimmune diseases, however, logistical challenges and safety concerns likely to present substantial headwinds
- Efficacy data from alternative therapeutic classes, such as T-cell engaging antibodies and mRNA-LNP based in vivo CAR-T, have not to date demonstrated efficacy in-line with autologous CAR-T therapies
- Initial clinical data of prula-cel in lupus nephritis demonstrated clinical activity comparable to autologous CAR-T therapies with improvement in kidney function and SLEDAI other components
  - Patients discontinued immunosuppressants and tapered corticosteroids
- Well-tolerated safety profile with No IEC-HS, No ICANs, No Gr>2 CRS
- Clear evidence of immune reset following single treatment with Prula-cel

*Cut-off date: August 31, 2025; Data cut included 7 patients (5 LN and 2 SLE) with a follow-up ranging from 2-9 months

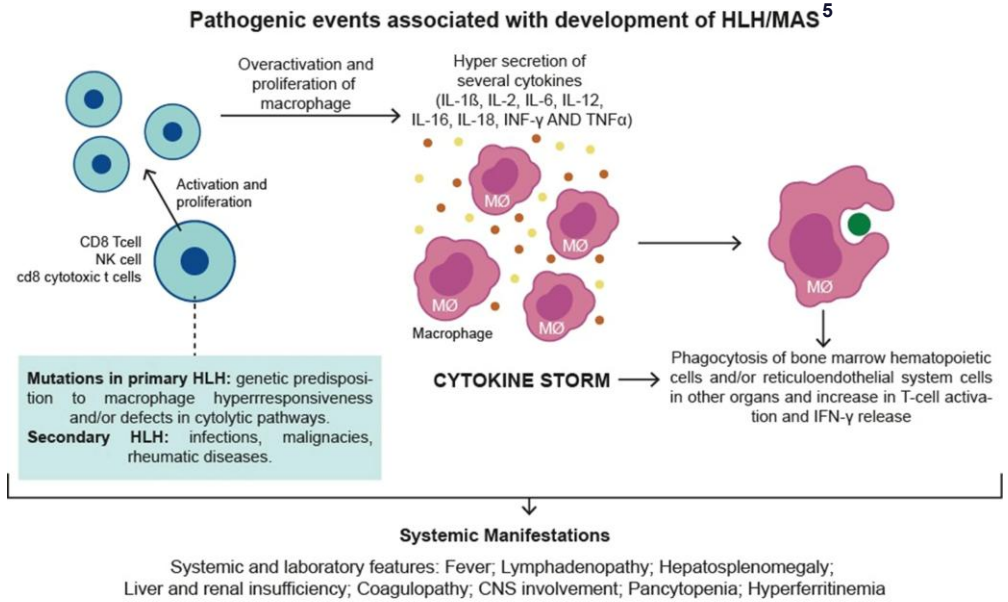
Prula-cel: Potentially First Off-the-Shelf, Easy to Administer, One-Time Therapy With Efficacy Comparable to Autologous CAR-T & Favorable Safety Profile*

	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

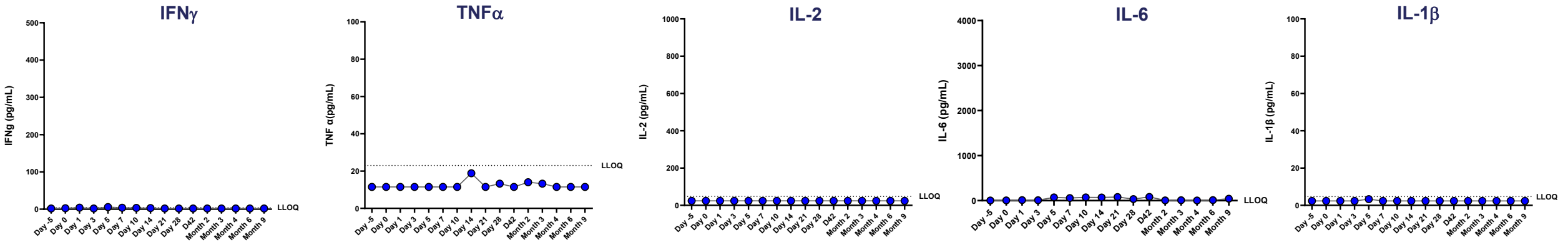
*Cut-off date: August 31, 2025; Data cut included 7 patients (5 LN and 2 SLE) with a follow-up ranging from 2-9 months

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 secretion of IFNγ, TNFα, IL-2, IL6, and IL18[5]
- Prula-cel does not lead to meaningful increase in these systemic markers in our autoimmune 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 31, 2025; seven patients (5 LN & 2 SLE) with a follow-up ranging from 2–9 months

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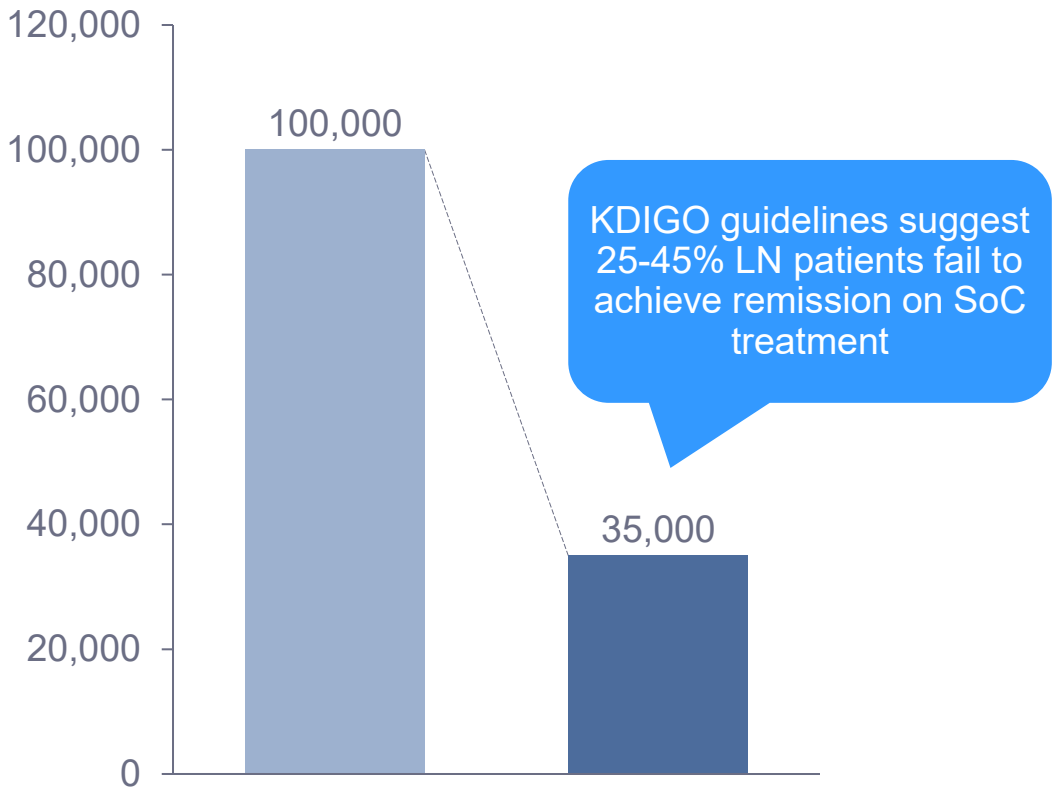
Significant Unmet Medical Need and Market Opportunity in LN

US Prevalence of LN (#)



- Lupus nephritis is a severe manifestation of SLE and occurs in approximately 40% of patients with SLE
- No curative therapies exist
- Roughly, 25-45% of LN patients fail to achieve remission after 24 months of SoC treatment
- **35,000 refractory LN patients in the US X CAR T Price** represents a **substantial market opportunity**
- Patients and payers may benefit from potential to lower associated healthcare costs – reduced progression to end-stage renal disease & requirements for dialysis and kidney transplant

Helmick CG et al. Arthritis & Rheumatism (2008)
Morales E et al. Nephron (2021)

Ding T. et al. Clin Exp Med (2025)
Kidney International Supplements (2012)
SoC= Standard of care

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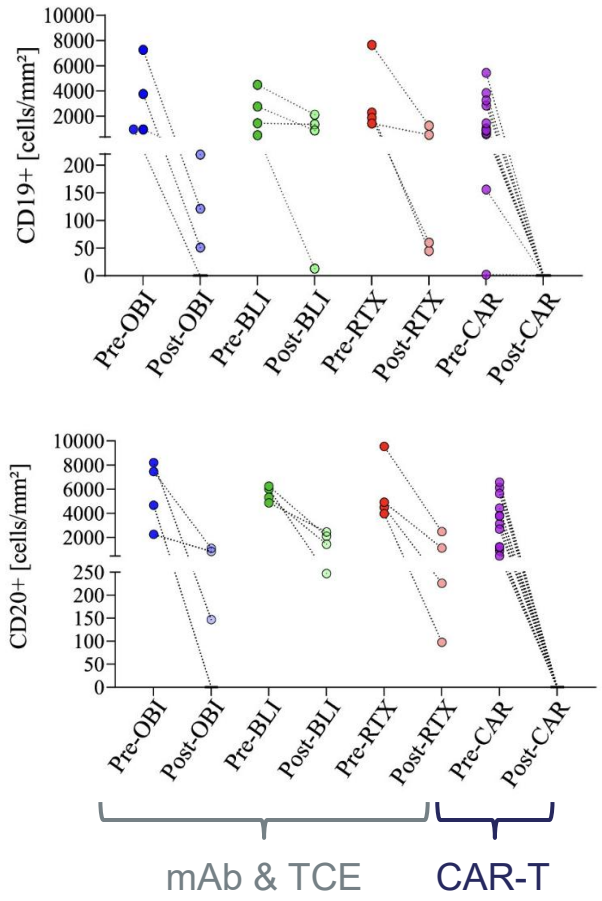
Incomplete B-Cell Depletion With Protein-Based Approaches (mAbs & TCEs) Associated With Less Robust Clinical Responses in Patients With Autoimmune Diseases



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

Tur C et al. Annals of the Rheumatic Diseases (2025)
RTX = rituximab, BLI = blinatumomab, OBI = obinutuzumab
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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Fierce Biotech July 2026

FIERCE Biotech

Roche drops TCE for lupus, but hasn't given up on autoimmune cell therapies

By James Waldron
Jul 23, 2026 4:51am

Roche may have scrapped work on a T-cell engager (TCE) for lupus, but the company's head of pharma told Fierce that the drugmaker still has a strategy for developing cell therapies for autoimmune diseases.

As part of a wider pipeline clearout disclosed during Roche's second-quarter earnings results released July 23, the Swiss pharma revealed that it had stopped work on RG6382, a T-cell-engaging bispecific antibody targeting CD19 and CD3. The company had been evaluating the therapy in an ongoing phase 1 open-label study of patients with systemic lupus erythematosus (SLE).

But a Roche spokesperson explained to Fierce this morning that after reviewing the data to date, "it has become clear that this molecule does not have the requisite characteristics to move forward in this indication."

Smaller Datasets of Autologous CAR-T Therapies

Obe-cel

- Six (6) LN patients dosed with obe-cel (50M cells)
- **3/6 (50%)** of patients achieved a CRR after treatment with obe-cel

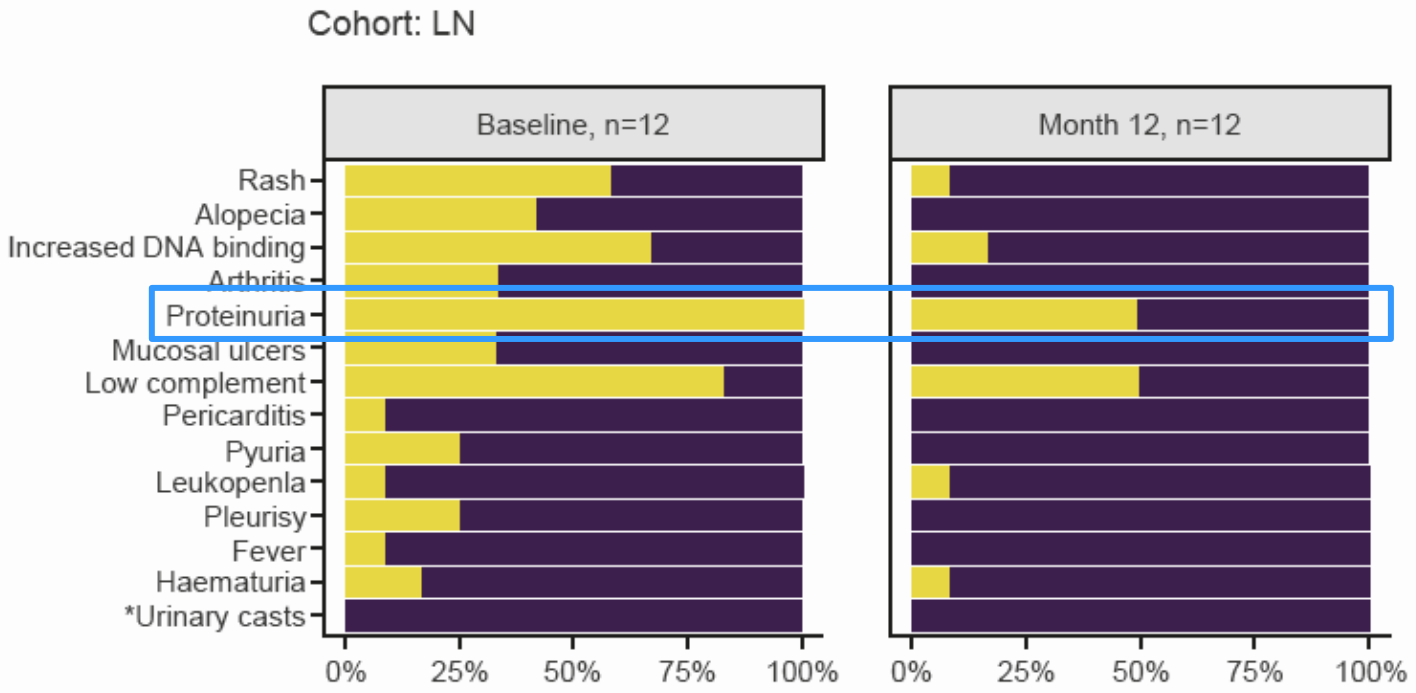
Rese-cel

- Four (4) LN patients dosed with rese-cel with 12-month follow-up
- **1/4 (25%)** of patients achieved a CRR after treatment with rese-cel

Overall, **4/10** (**40%**) CRR rate

Obe-cel and Rese-cel across smaller LN cohorts

Rap-cel (NVS): CRR Not Reported; Proteinuria Resolved in Half of LN Patients at 12-Months



- No data provided on CRR rate or eGFR
- 50% of patients resolved proteinuria domain of the SLEDAI-2K at 12-months
- Suggesting CRR rate is up to half
- **8 of the 21 patients** (38%) (n=12 LN & n=9 SLE) from the Phase I trial **received immunosuppressants during the course** of the study

Amoura Z. et al. EULAR2026

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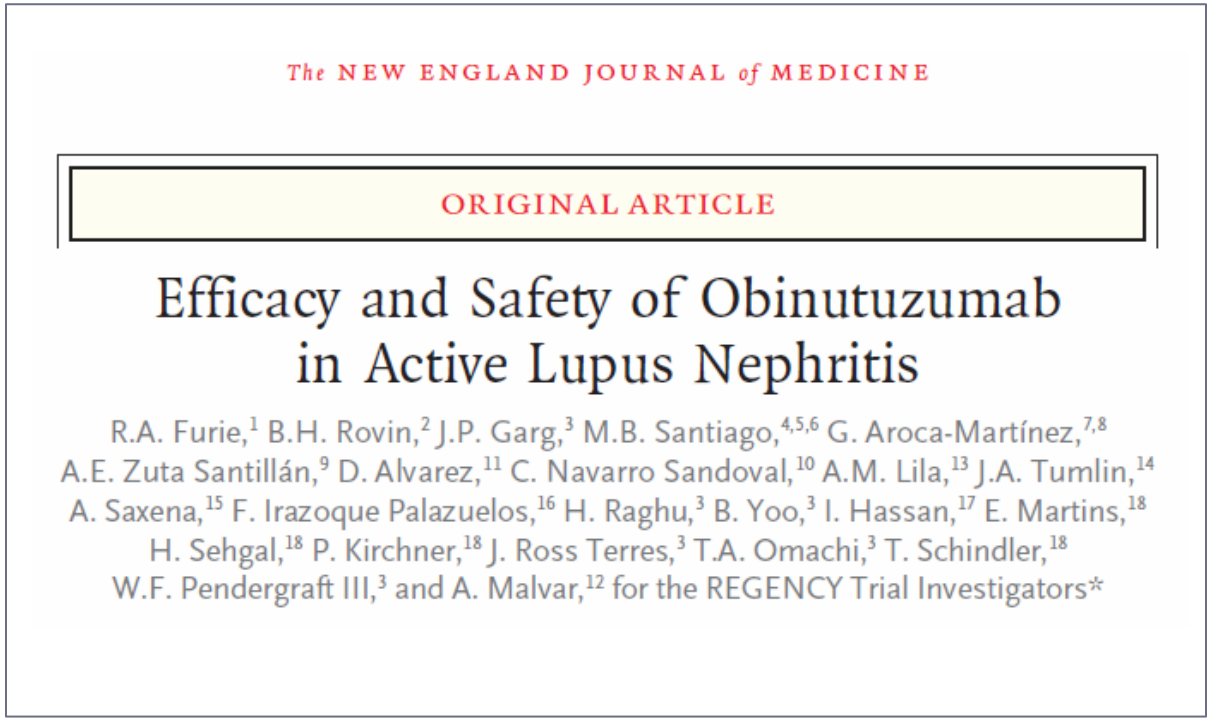
Obinutuzumab + Chronic Immunosuppression & Steroids CRR Rate of 43% at 18 Months

CRR rate of 43% for Obinutuzumab + standard therapy among previously diagnosed patients in Phase III LN study



The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Efficacy and Safety of Obinutuzumab in Active Lupus Nephritis

R.A. Furie,[1] B.H. Rovin,[2] J.P. Garg,[3] M.B. Santiago,[4,5,6] G. Aroca-Martínez,[7,8] A.E. Zuta Santillán,[9] D. Alvarez,[11] C. Navarro Sandoval,[10] A.M. Lila,[13] J.A. Tumlin,[14] A. Saxena,[15] F. Irazoque Palazuelos,[16] H. Raghu,[3] B. Yoo,[3] I. Hassan,[17] E. Martins,[18] H. Sehgal,[18] P. Kirchner,[18] J. Ross Terres,[3] T.A. Omachi,[3] T. Schindler,[18] W.F. Pendergraft III,[3] and A. Malvar,[12] for the REGENCY Trial Investigators*

Key considerations

- CRR - measured at week 76 (~18 months)
- Obi dosed in addition to standard therapy (daily dosing chronic immunosuppression and steroids)
- **72% infection rate**, including **17% infections as SAE**
- Median baseline **SLEDAI-2K score of 10**

Furie RA et al. NEJM (2025)

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Recent Data Suggest 40% CRR Rate As a Clinically Meaningful Benchmark For Cell Therapy In Heavily Treated Or Refractory Lupus Nephritis

Small auto CAR-T datasets

~40% CRR

Obinutuzumab + std therapy

43% CRR

(in previously diagnosed LN)

Rap-cel

CRR Not Reported; Proteinuria Resolved in Half of LN Patients at 12-Months

(~40% of pts received immunosuppressants on trial)

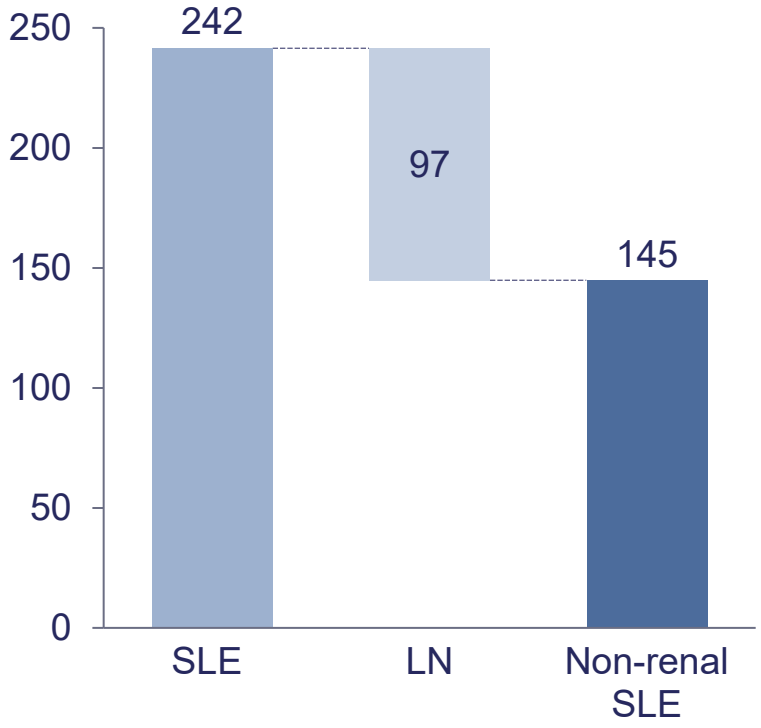
40% CRR clinically meaningful benchmark

Autolus Corporate Deck May 2026
Rese-cel EULAR 2026 poster

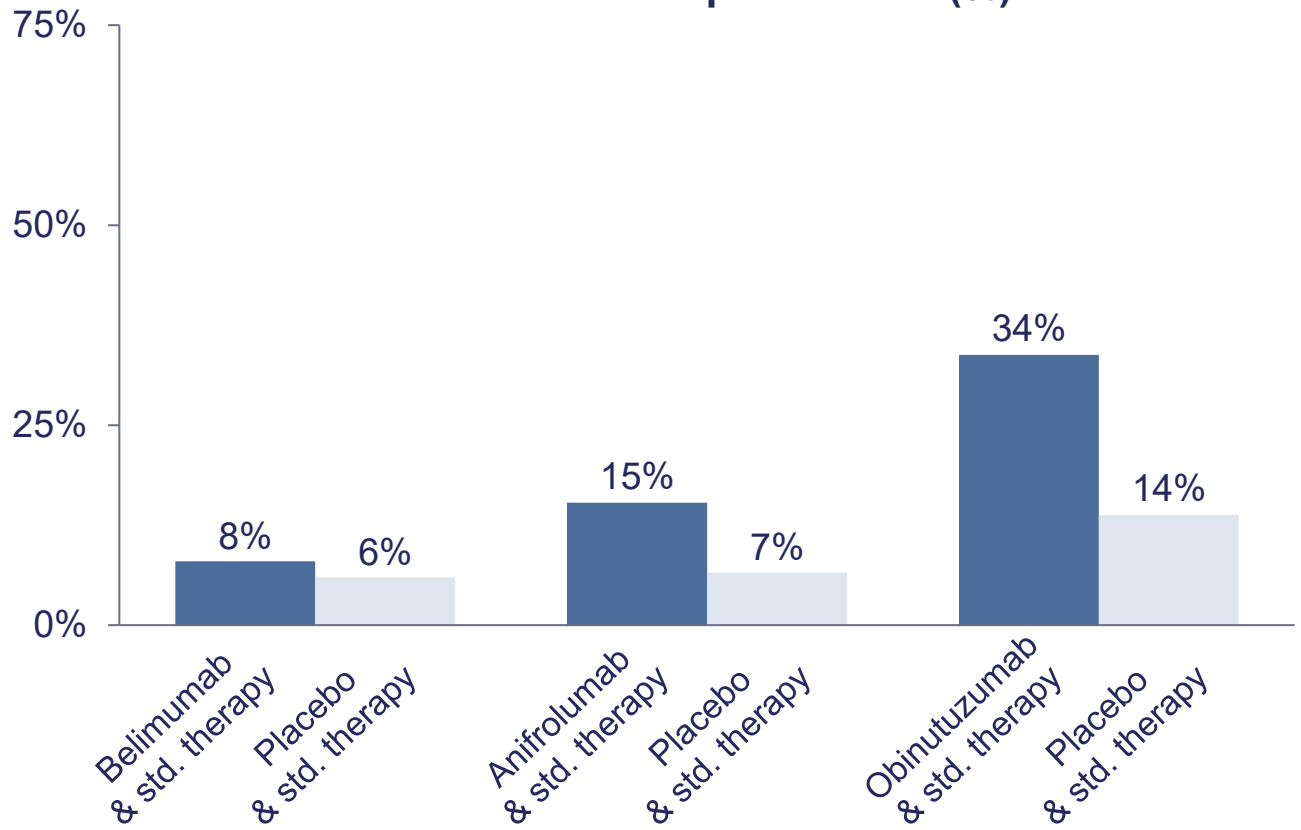
Amoura Z. et al. EULAR2026
Furie RA et al. NEJM (2025)

Recently Approved Therapies for SLE Are Delivered On Top of Standard Therapy & Fail to Achieve High DORIS Remission Rates

US Prevalence of SLE and non-renal SLE (K)



DORIS Remission Rates for Recent Approved Chronic Therapies in SLE (%)



None of existing therapies provide treatment free remissions

Helmick CG et al. Arthritis & Rheumatism (2007)

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

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ADI-212

Next-Generation, Gene-Edited, Armored Cell Therapy Candidate Designed to Address the Complexity of Solid Tumors

mCRPC

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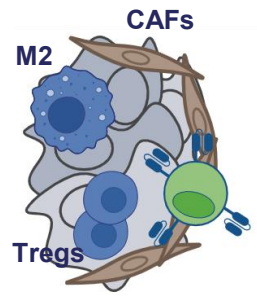
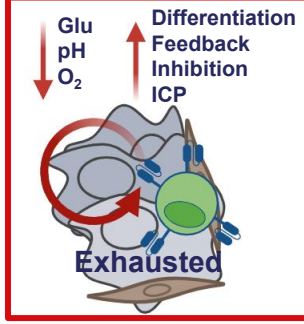
Current Challenges for Existing Cell Therapies in Solid Tumors



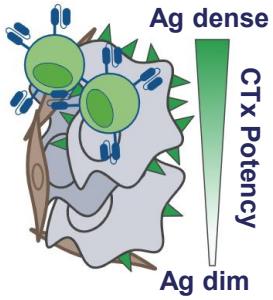
1 – Nonpermissive TME

- 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

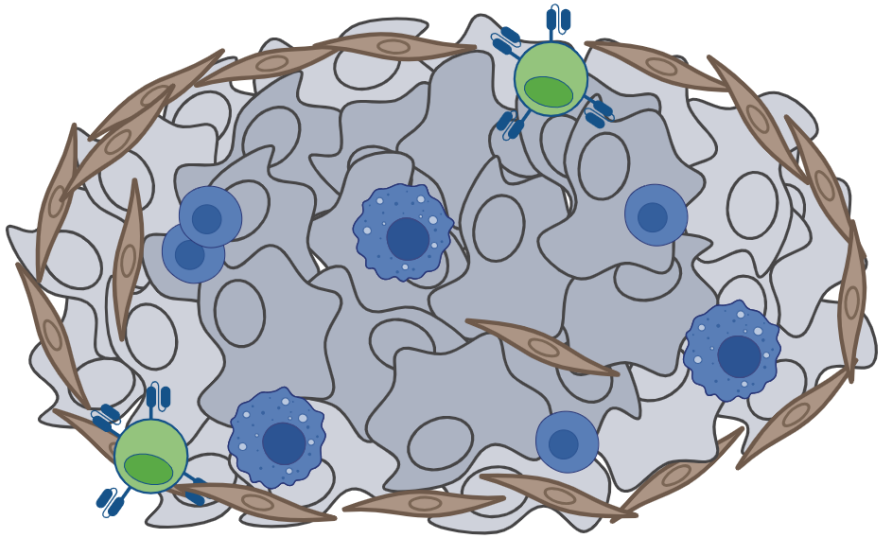
- Tumor cells express inhibitory receptors/factors
- Metabolic stress exhausts effector cells
- Feedback loops inhibit repetitive killing capacity



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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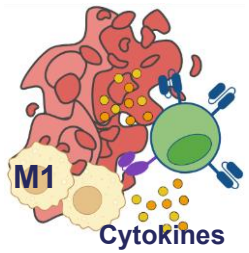
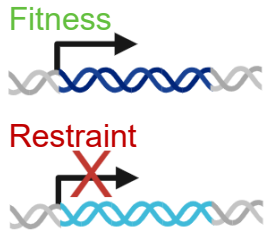
Three Strategic Pillars for Overcoming Solid Tumor Barriers



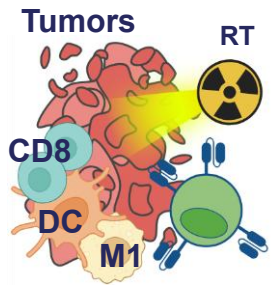
1 – Localized Cytokine Armoring

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



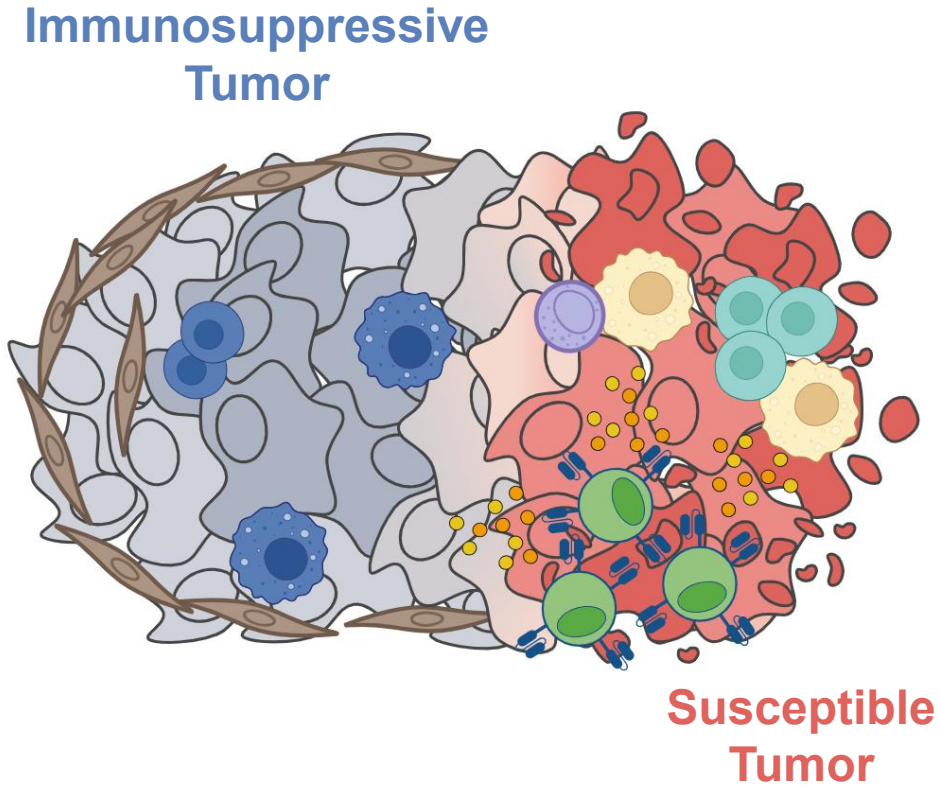
2 – Transcriptional Tuning

- 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

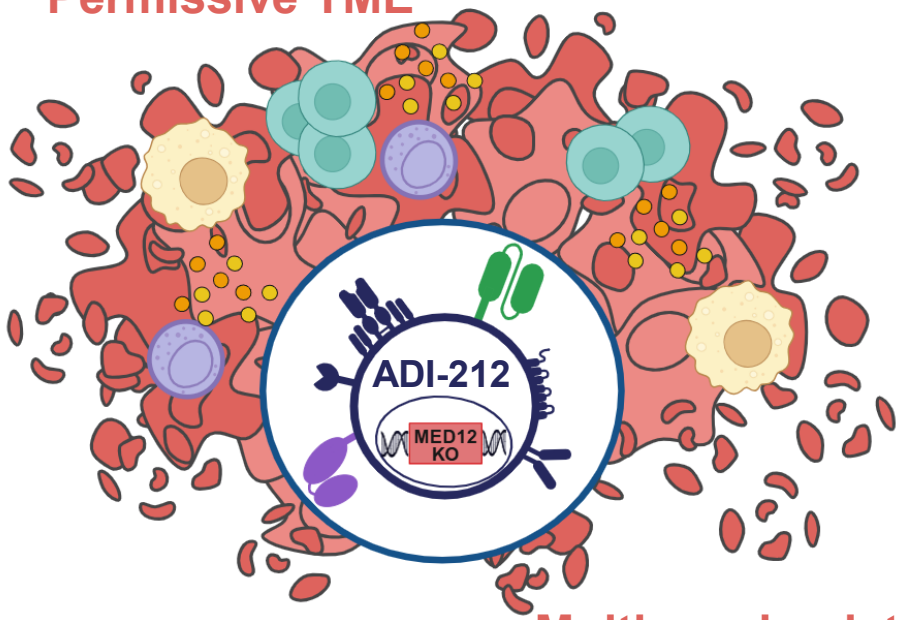
2 – Transcriptional Tuning

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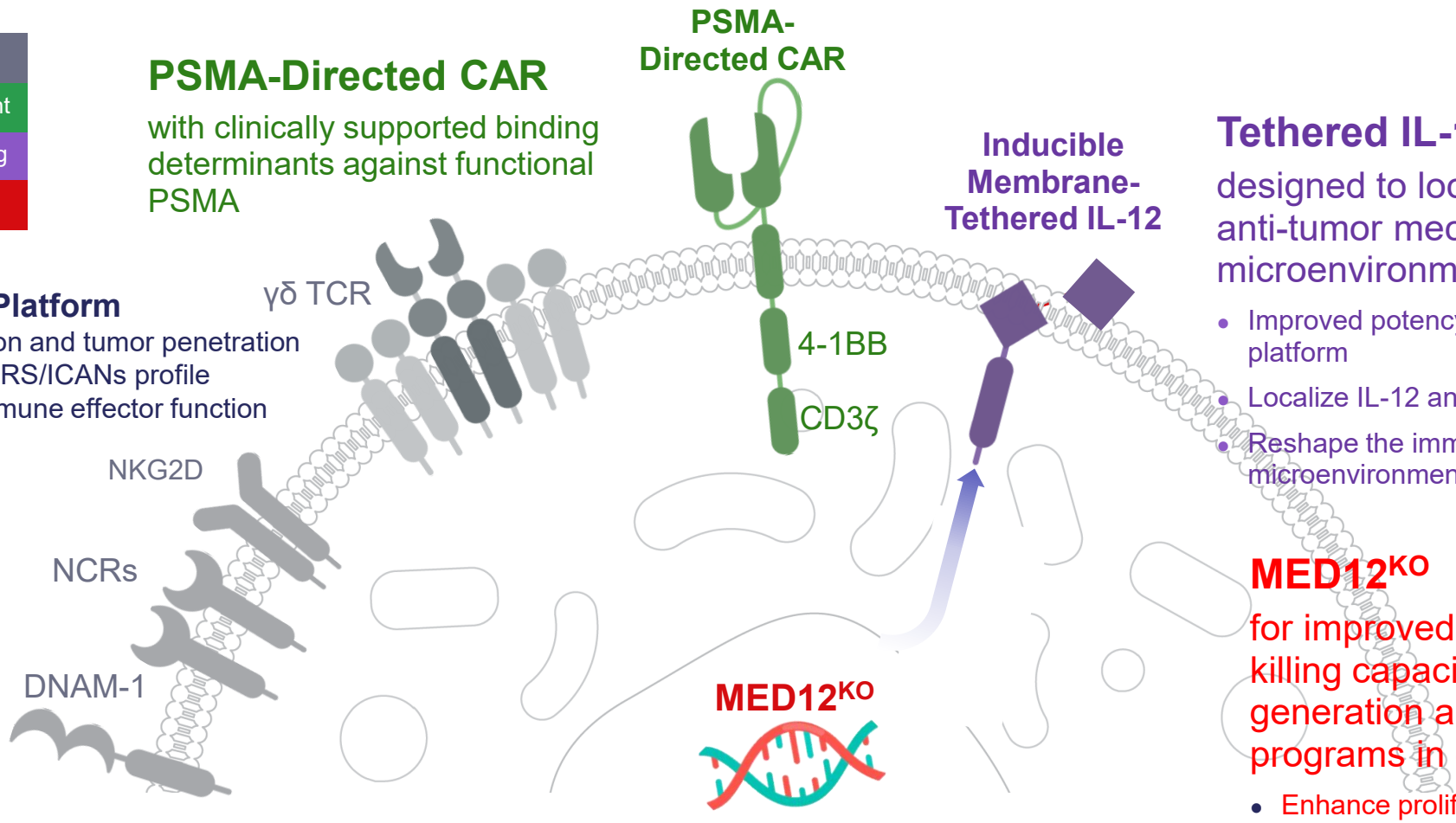
ADI-212 Designed To Address Multiple Barriers in a Single Off-the-Shelf Product

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

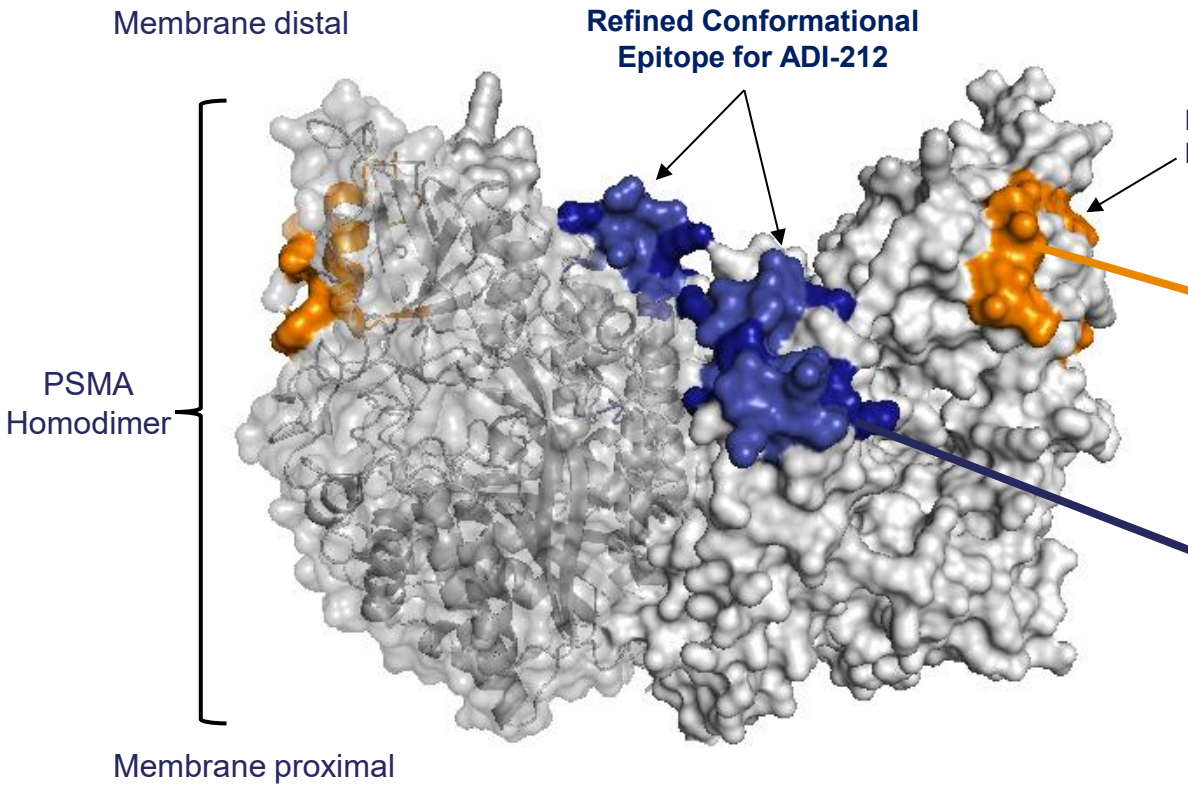
- Enhance proliferation and survival
- Improve potency & metabolic fitness advantages

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

Adicet Bio

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

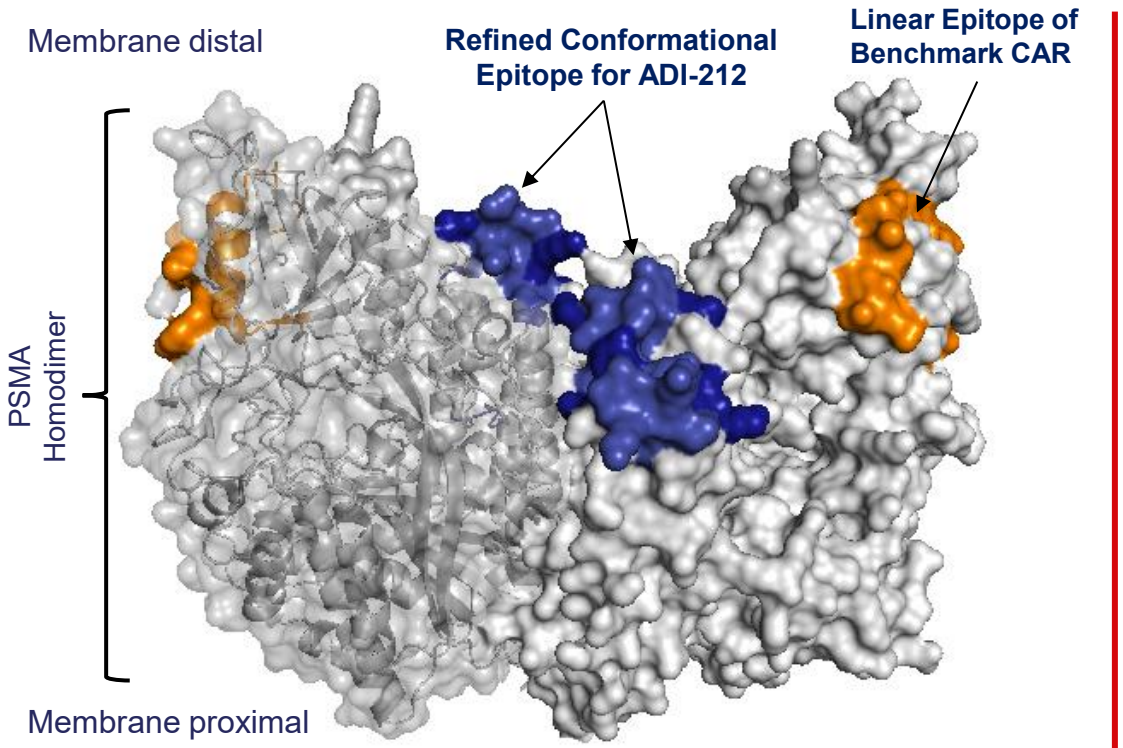
ADI-212 binder recognizes functional PSMA homodimer and demonstrated less tissue cross-reactivity when compared to benchmark binder for αβ PSMA-CAR T

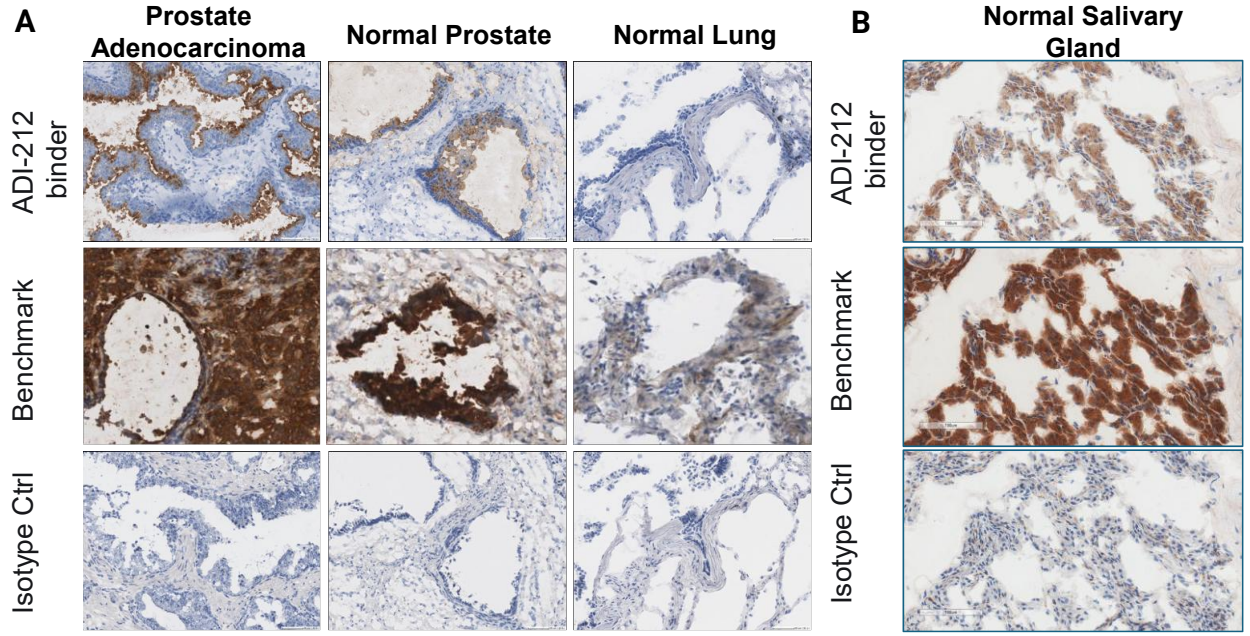
Ramadoss, Nitya S., Elizabeth Speltz, Tonya Capillo, et al. *Molecular Cancer Therapeutics* 22, no. 12_Supplement (2023): C117.

Adicet Bio

ADI-212 CAR is Refined for Improved Recognition of Functional PSMA Complexes and Demonstrated Reduced Off-Tissue Targeting

Adicet's binder showed specific staining and demonstrated lower incidence and intensity of off-tissue binding liabilities





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

Adicet Bio

Tethered Form of IL-12: Perpetuates a Multi-Pronged Anti-Tumor Attack

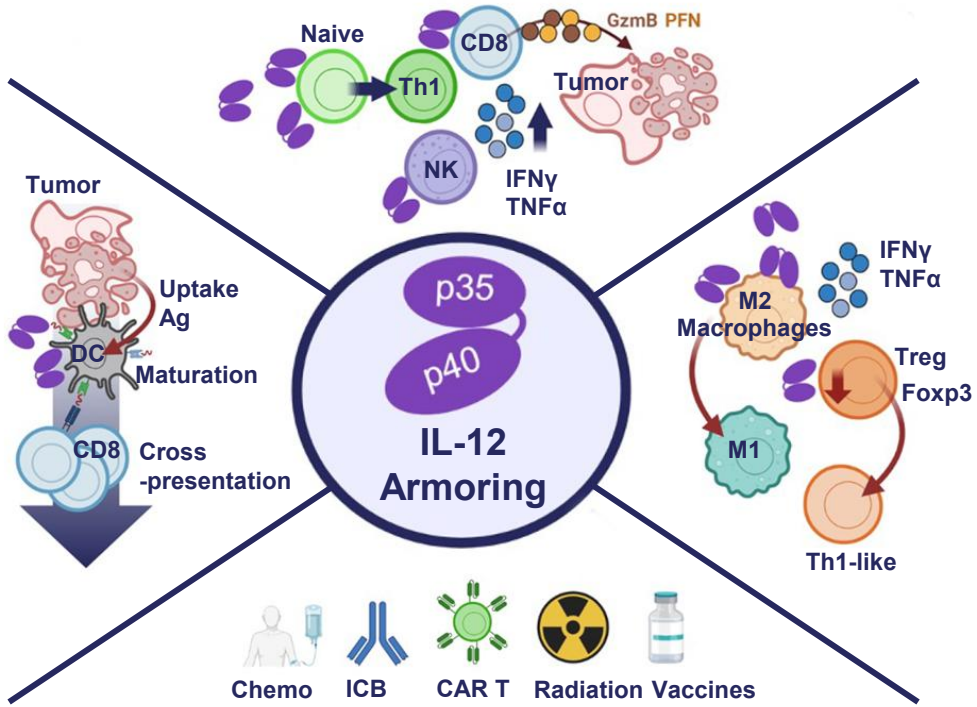
mbIL-12 Armoring Introduces Multiple Mechanisms for Efficacy Enhancement

Enhances Cytotoxic Cell Activation and Expansion

- Drives IFNγ and TNFα release from Effector T and NK cells
- Supports Th1 polarization, proliferation and persistence

Boosts Epitope Spreading

- Promotes DC maturation and tumor antigen uptake
- Broadens endogenous responses via cross-presentation



Reprograms Suppressive TME

- Repolarizes M2 macrophages toward an M1 phenotype
- Destabilizes FoxP3+ Tregs toward a Th1-like state

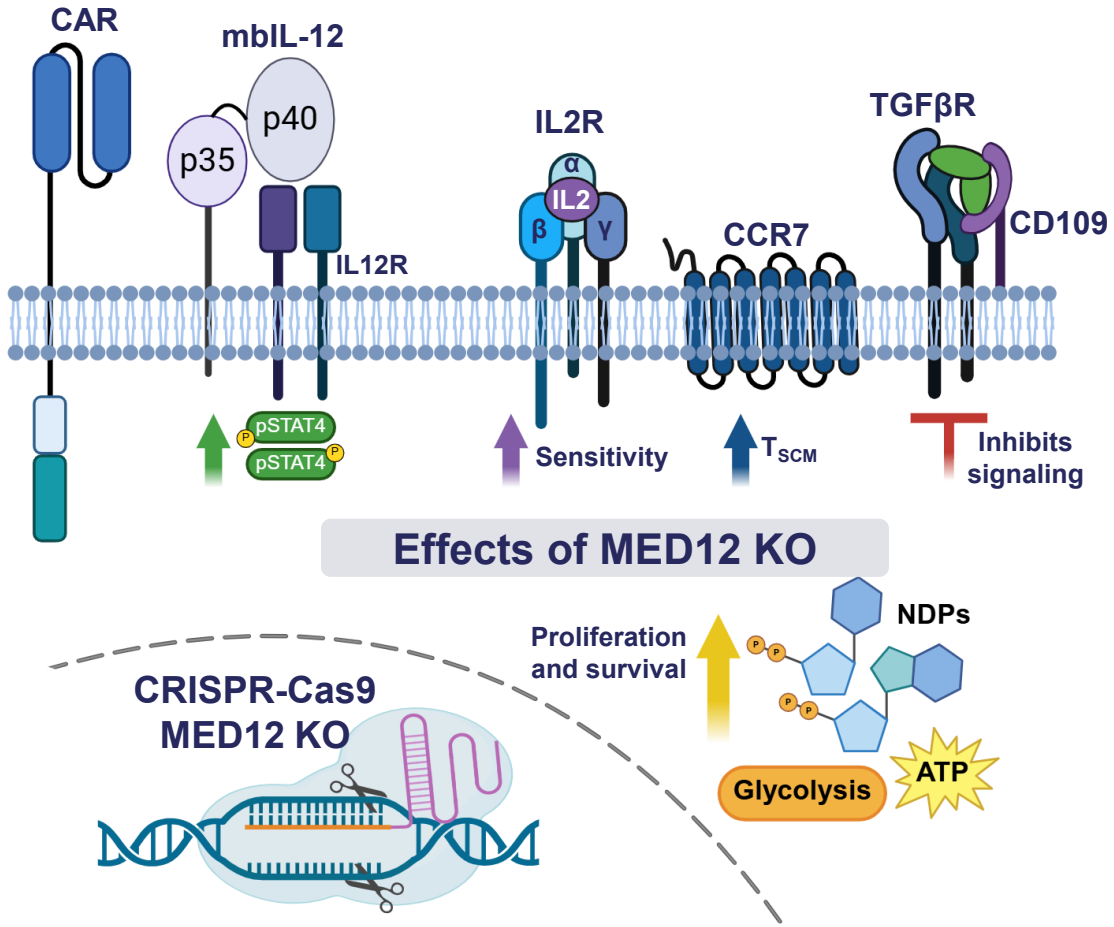
Potential Synergy with Therapies

- Complements CAR T, ICB, chemotherapy and radiation Tx
- May amplify vaccine-induced anti-tumor immunity

TME, Tumor Microenvironment; M1, M1 Macrophages; M2, M2 Macrophages; DC, Dendritic cells; Treg, Regulatory T cells; NK, Natural Killer; Th1, Type 1 helper T cells; IFNγ, Interferon-gamma; TNFα, Tumor necrosis factor-alpha; GzmB, Granzyme B; PFN, Perforin; ICB, Immune checkpoint blockade

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

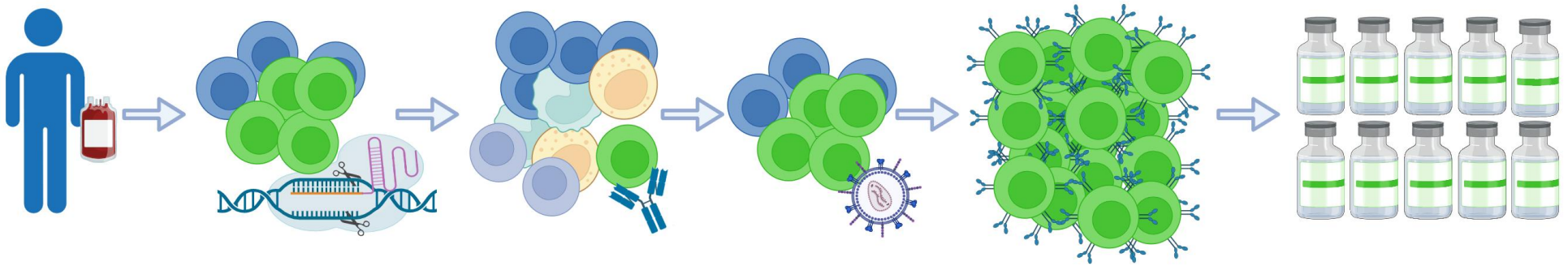
Synergy

MED12 KO

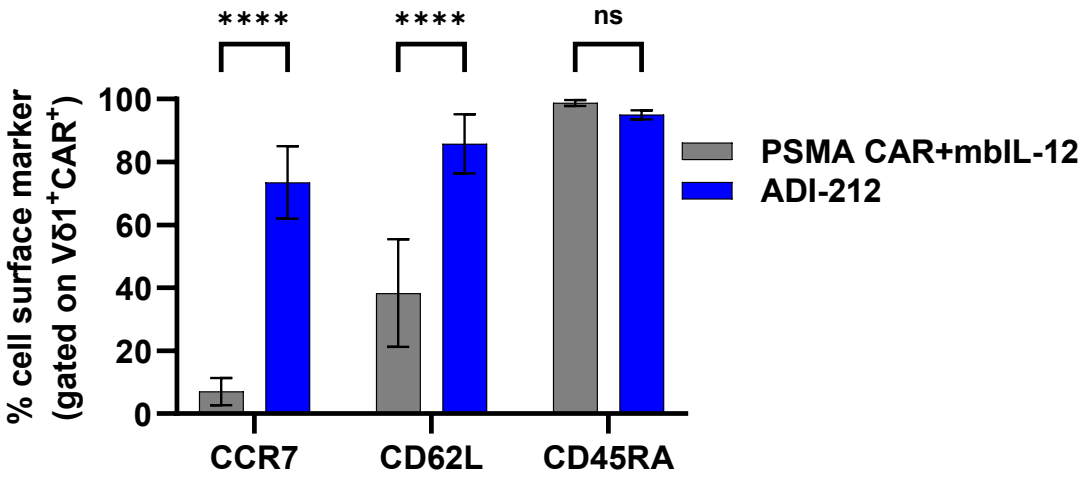
TME, Tumor Microenvironment, CAR, Chimeric antigen receptor; CRISPR, Clustered Regularly Interspaced Short Palindromic Repeats; KO, Knockout
ATP, Adenosine triphosphate, NDP, Nucleotide diphosphate, Tscm, Stem cell memory T cells, TGFβR, Transforming growth factor-beta receptor

Adicet Bio

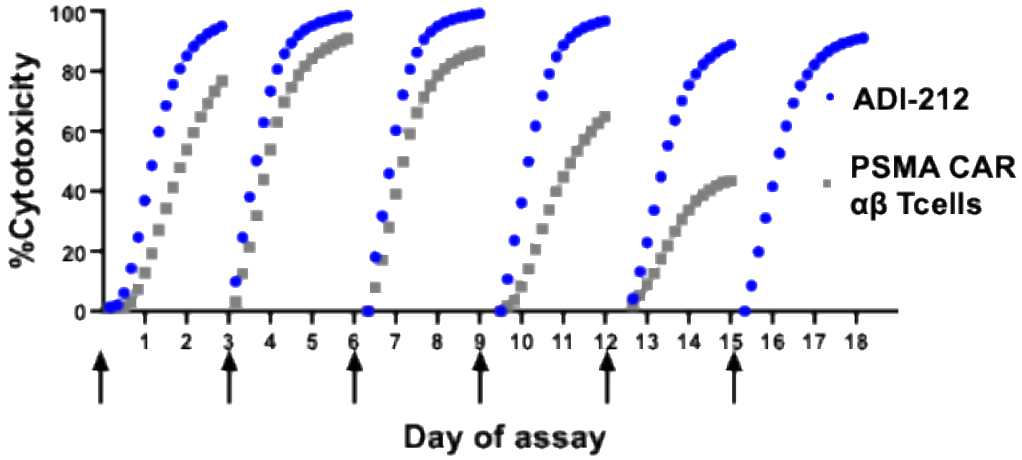
ADI-212 is Enriched for Vδ1 Memory T cells and Has the Capacity for Sustained Expansion and Tumor Killing



ADI-212 phenotype is largely comprised of Naïve and Stem-like T cells



ADI-212 sustains higher anti-tumor activity across multiple tumor rechallenges compared to clinically relevant CAR-T benchmarks

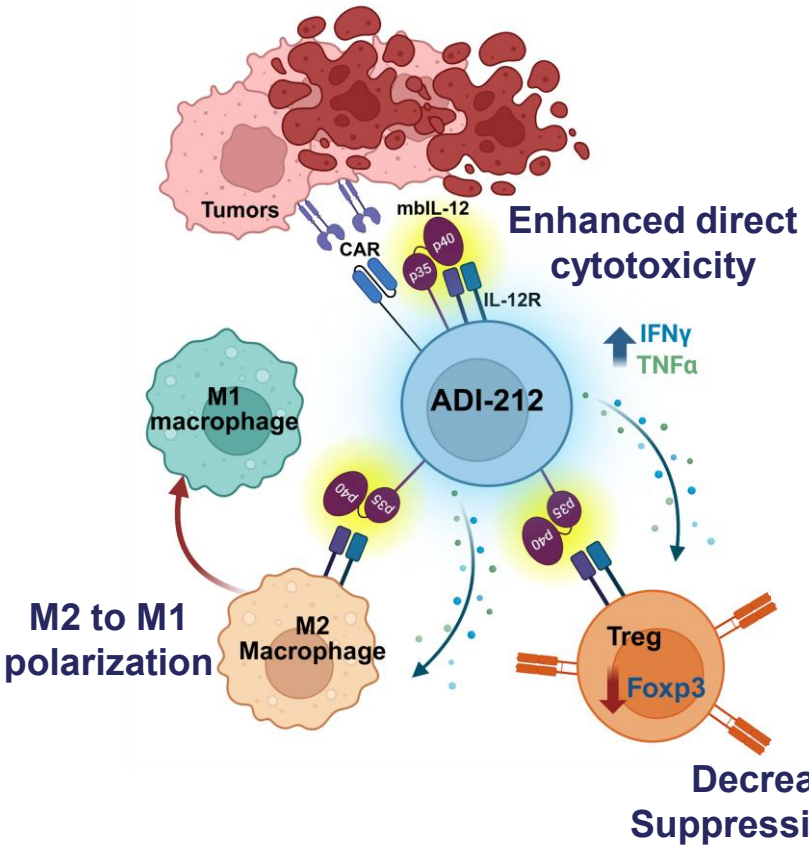


KO, Knockout; **PBMC**, Peripheral blood mononuclear cells

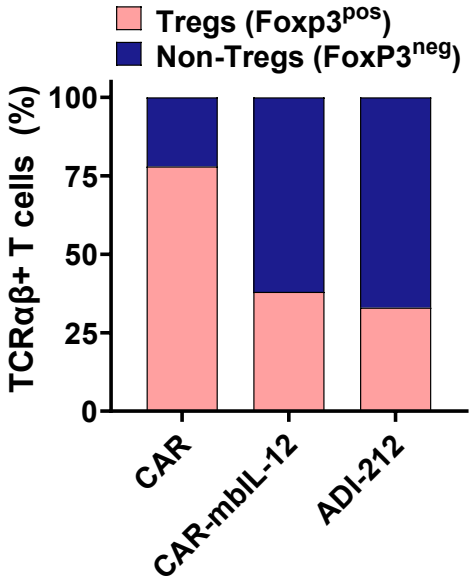
Ramadoss, Nitya S., Elizabeth Speltz, Tonya Capillo, et al. *Molecular Cancer Therapeutics* 22, no. 12_Supplement (2023): C117.

Adicet Bio

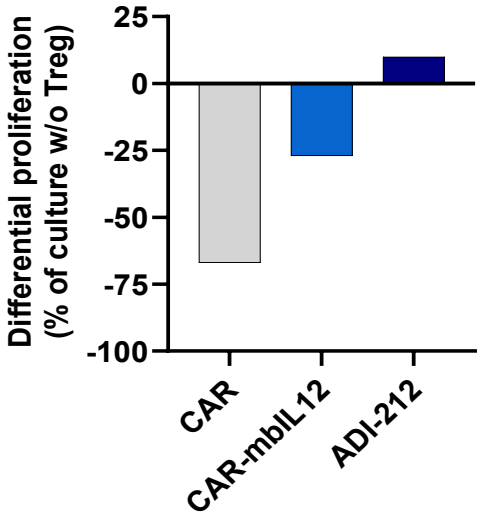
ADI-212 Capacity to Reshape Tumor Microenvironments and Maintain Potent Anti-Tumor Killing in the Presence of Immunosuppressive Cells



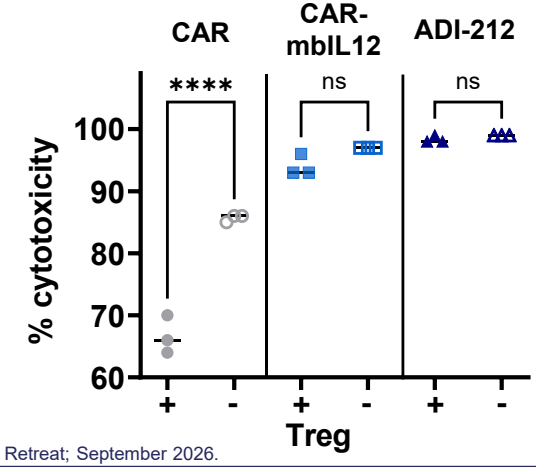
1 mbIL-12 on ADI-212 modulates Treg Population in Cocultures



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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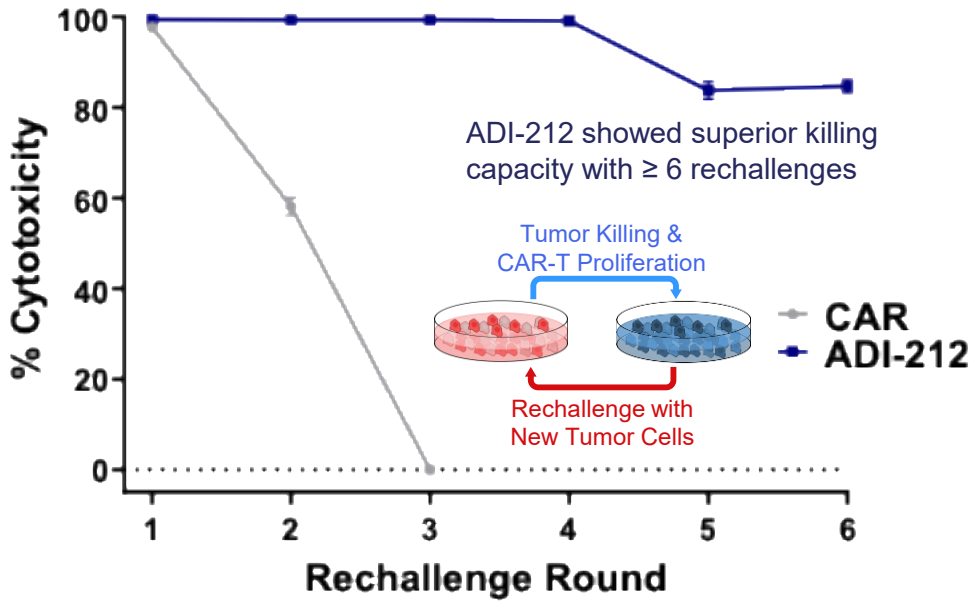
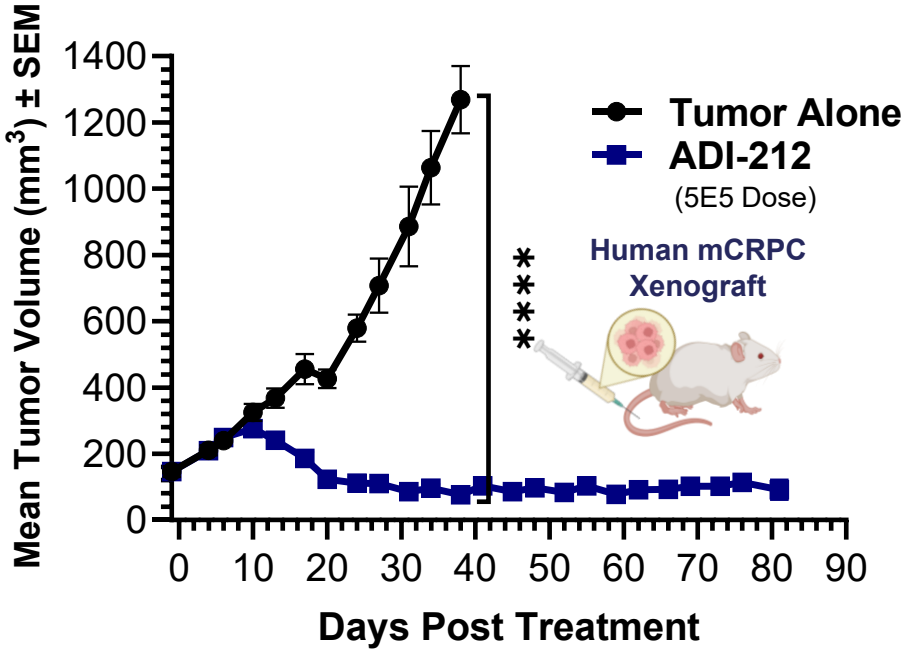
ADI-212 Demonstrated Highly Potent Preclinical Activity in Solid Tumors

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

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

Clinical data from first program expected 2027
Clinical data from second and third program potentially in 2028

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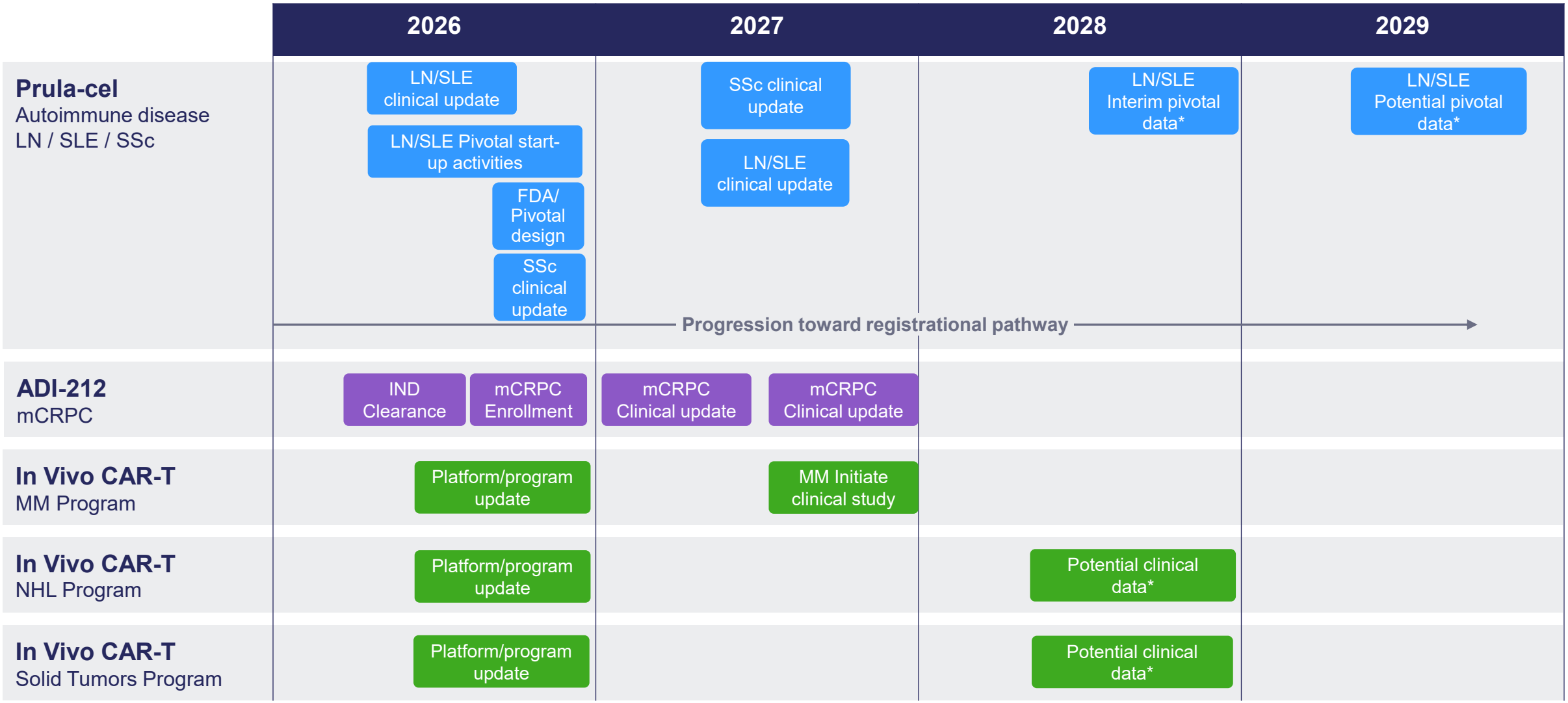
Expected Near-Term Value-Creating Milestones

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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Investor Overview

September 2026

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