Cabaletta Bio®

Clinical & Translational Findings Following Resecabtagene Autoleucel Anti-CD19 CAR T Cell Therapy in Autoimmune Disease

Jenell Volkov, PhD
Senior Director, Translational Medicine

May 15th, 2025

Disclaimer

The following presentation, including any printed or electronic copy of these slides, the talks given by the presenters, the information communicated during any delivery of the presentation and any question and answer session and any document or material distributed at or in connection with the presentation (collectively, the "Presentation") has been prepared by Cabaletta Bio, Inc. ("we," "us," "our," "Cabaletta" or the "Company") and is made for informational purposes only. This Presentation does not purport to be a prospectus, to be complete or to contain all of the information you may desire. Statements contained herein are made as of the date of this Presentation unless stated otherwise, and this Presentation shall not under any circumstances create an implication that the information contained herein is correct as of any time after such date or that information will be updated or revised to reflect information that subsequently becomes available or changes occurring after the date hereof. This Presentation may contain "forward-looking statements" within the meaning of the Private Securities Litigation Reform Act of 1995 relating to our business, operations, and financial conditions, and include, but are not limited to, express or implied statements regarding our current beliefs, expectations and assumptions regarding: our business, future plans and strategies for our CAAR T technology; our ability to grow our autoimmune-focused pipeline; the ability to capitalize on and potential benefits resulting from our research and translational insights; including those related to any similarly-designed constructs or dosing regimens; the anticipated market opportunities for rese-cel in patients with autoimmune diseases; the Company's business plans and objectives; our expectations around the potential success and therapeutic and clinical benefits of rese-cel and our other product candidates, as well as our ability to successfully complete research and further development and commercialization of our drug candidates in current or future indications, including the timing and results of our clinical trials and our ability to conduct and complete clinical trials; expectation that clinical results will support rese-cel's safety and activity profile; our plan to leverage increasing clinical data and a unique development program for rese-cel; the clinical significance of the clinical data read-out at upcoming medical or scientific meetings; our belief that rese-cel has the potential to provide drug-free, durable meaningful clinical responses, through an immune reset, including the potential for achieving drug-free remission in patients with refractory myositis; the Company's advancement of separate Phase 1/2 clinical trials of rese-cel in patients with SLE, myositis, SSc and gMG and advancement of the RESET-PV and RESET-MS trials, including updates related to status, safety data, efficiency of clinical trial design and timing of data read-outs or otherwise; our ability to leverage our experience in autoimmune cell therapy; our ability to enroll the requisite number of patients, dose each dosing cohort in the intended manner and timing thereof, and advance the trial as planned in our Phase 1/2 clinical trials of rese-cel; the timing any planned regulatory filings for our development programs, including IND applications; the progress and results of our MusCAARTes™ Phase 1 trial, and impact around reported safety and clinical and translational data of cohorts from our MusCAARTes™ Phase 1 trial; Cabaletta's advancement of the whole blood manufacturing program to remove the burden of apheresis; statements regarding the timing of regulatory filings and interactions with regulatory authorities, including such authorities' review of safety information from our ongoing clinical trials and potential registrational pathway for rese-cel; our ability to successfully complete our preclinical and clinical studies for our product candidates, including our ability to enroll the requisite number of patients, dose each dosing cohort in the intended manner, and progress the trial; our ability to increase enrollment from our rapidly expanding clinical network in the RESET clinical trial program in the US and Europe; our ability to obtain and maintain regulatory approval of our product candidates, including our expectations regarding the intended incentives conferred by and ability to retain Fast Track Designations for our product candidates; our ability to accelerate our pipeline to approval and launch and to develop meaningful therapies for patients, including in collaboration with academic and industry partners and the ability to optimize such collaborations on our development programs; our ability to contract with third-party suppliers and manufacturers and retain such manufacturers, whether due to legislative action or otherwise; to implement an enhanced manufacturing process and further develop our internal manufacturing strategy, capabilities and facilities; our ability to execute our manufacturing strategy to enable expansion of clinical supply and efficiently scale commercial supply for rese-cel; our potential commercial opportunities, including value and addressable market, for our product candidates. Words such as, but not limited to, "look forward to," "believe," "expect," "anticipate," "estimate," "intend," "plan," "would," "should" and "could," and similar expressions or words, identify forward-looking statements.

Various risks, uncertainties and assumptions could cause actual results to differ materially from those anticipated or implied in our forward-looking statements. Such risks and uncertainties include, but are not limited to, risks related to the success, cost, and timing of our product candidate development activities and preclinical studies and clinical trials, risks related to our ability to demonstrate sufficient evidence of safety, efficacy and tolerability in our preclinical studies and clinical trials of rese-cel and MuSK-CAART, the risk that the results observed with the similarly-designed construct, including, but not limited to, due to dosing regimen, are not indicative of the results we seek to achieve with rese-cel, the risk that signs of biologic activity or persistence may not inform long-term results, the risk that persistence observed with effective CD19-CAR T oncology studies in combination with lymphodepletion is not indicative of, or applicable to, clinical responses in patients with mPV, risks related to clinical trial site activation or enrollment rates that are lower than expected, our ability to demonstrate sufficient evidence of safety, efficacy and tolerability in its preclinical studies and clinical trials of rese-cel; risks that modifications to trial design or approach may not have the intended benefits and that the trial design may need to be further modified; our ability to protect and maintain our intellectual property position, risks related to our relationships with third parties, uncertainties related to regulatory agencies' evaluation of regulatory filings and other information related to our product candidates, our ability to retain and recognize the intended incentives conferred by any Orphan Drug Designations and Fast Track Designations, risks related to regulatory filings and potential clearance, the risk that any one or more of our product candidates will not be successfully developed and commercialized, the risk that the results of preclinical studies or clinical studies will not be predictive of future results in connection with future studies, and risks related to volatile market and economic conditions and public health crises. New risks and uncertainties may emerge from time to time, and it is not possible to predict all risks and uncertainties. Except as required by applicable law, we do not plan to publicly update or revise any forward-looking statements contained herein, whether as a result of any new information, future events, changed circumstances or otherwise. Although we believe the expectations reflected in such forward-looking statements are reasonable, we can give no assurance that such expectations will prove to be correct. Accordingly, you are cautioned not to place undue reliance on these forward-looking statements. No representations or warranties (expressed or implied) are made about the accuracy of any such forward-looking statements. For a discussion of these and other risks and uncertainties, and other important factors, any of which could cause our actual results to differ materially from those contained in the forward-looking statements, see the section entitled "Risk Factors" in our most recent annual report on Form 10-K, as well as discussions of potential risks, uncertainties, and other important factors in our other and subsequent filings with the Securities and Exchange Commission. Certain information contained in this Presentation relates to or is based on studies, publications, surveys and other data obtained from third-party sources and the Company's own internal estimates and research. While the Company believes these third-party sources to be reliable as of the date of this Presentation, it has not independently verified, and makes no representation as to the adequacy, fairness, accuracy or completeness of, any information obtained from third-party sources. The Company is the owner of various trademarks, trade names and service marks. Certain other trademarks, trade names and service marks appearing in this Presentation are the property of third parties. Solely for convenience, the trademarks and trade names in this Presentation are referred to without the ® and TM symbols, but such references should not be construed as any indicator that their respective owners will not assert, to the fullest extent under applicable law, their rights thereto.

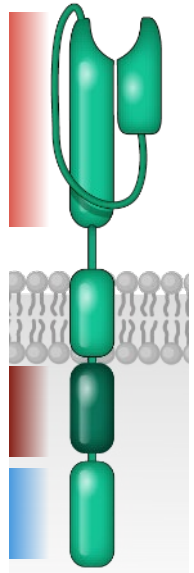
Rese-cel (CABA-201): CD19-CAR T designed for autoimmunity

Cabaletta's CD19 binder with similar in vitro & in vivo activity to FMC63[1,2] (binder used in academic report[3])

Fully human anti-CD19 binder

4-1BB costimulatory domain

CD3-ζ signaling domain



Rese-cel[4]

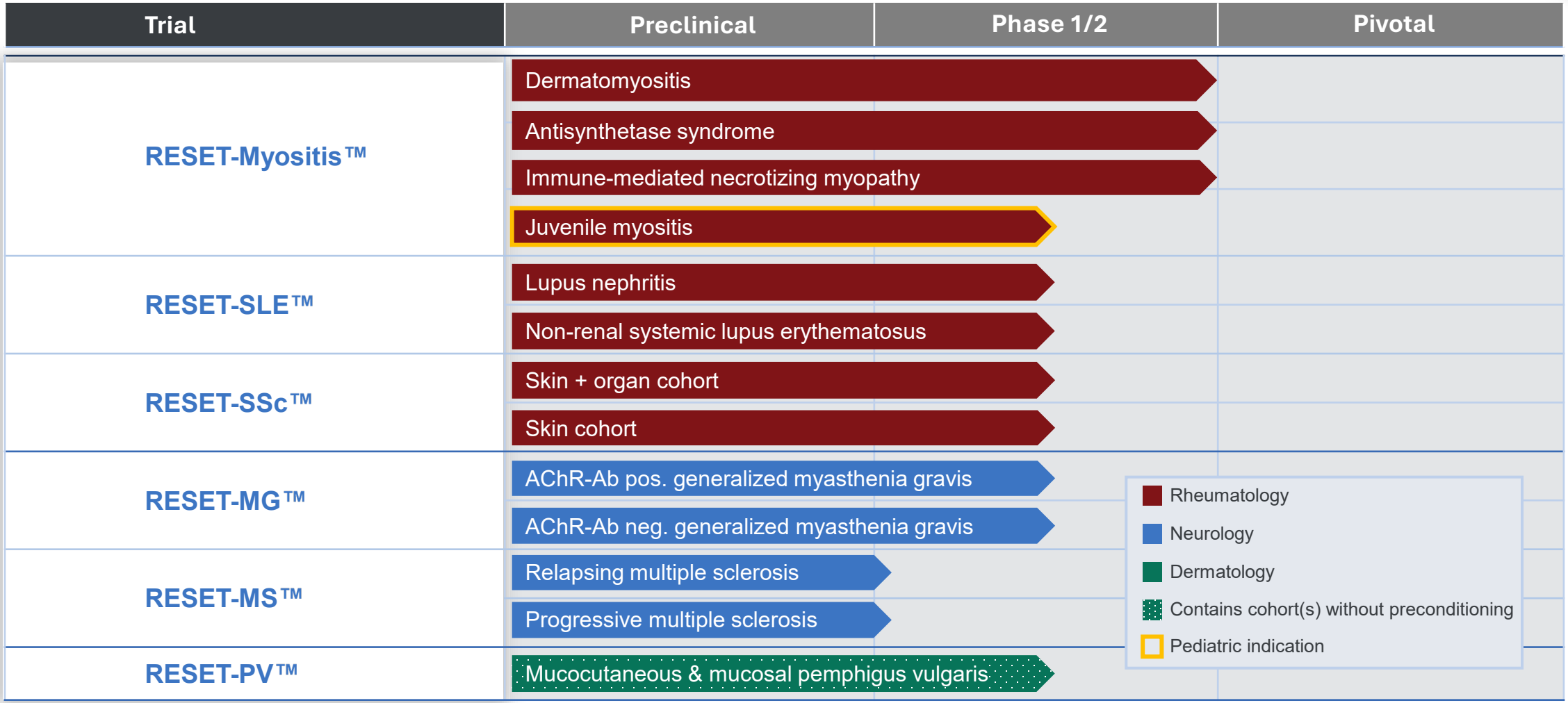
Rese-cel product design & clinical / translational data

- 4-1BB costimulatory domain with fully human binder
  - Binder with similar affinity & biologic activity to academic FMC63 binder while binding to the same epitopes[1,2]
- Same weight-based dose as in academic studies
  - Potential to provide immune reset based on initial clinical and translational data[5]
- Initial patients treated with rese-cel have shown compelling clinical responses with safety data that supports autoimmune development[6]

1. Peng, Binghao J, et al. "Preclinical specificity and activity of CABA-201, a fully human 4-1BB containing CD19 CAR T therapy for treatment-resistant autoimmune disease." Poster presented at: American Society Gene and Cell Therapy 26th Annual Meeting; 2023 May 19; Los Angeles, CA.
2. Dai, Zhenyu, et al. "Development and functional characterization of novel fully human anti-CD19 chimeric antigen receptors for T-cell therapy." Journal of Cellular Physiology 236.8 (2021): 5832-5847.
3. Müller, Fabian, et al. "CD19 CAR T-Cell Therapy in Autoimmune Disease—A Case Series with Follow-up." New England Journal of Medicine 390.8 (2024): 687-700.
4. Transmembrane domain in CABA-201 is CD8α vs. TNFRSF19 (Troy) utilized in the academic construct. The two transmembrane domains have not been shown to have a significant difference in function or IFN-γ production in preclinical studies. The CD8α transmembrane domain is employed in tisagenlecleucel.
5. Volkov, Jenell, et al. "Case study of CD19 CAR T therapy in a subject with immune-mediate necrotizing myopathy treated in the RESET-Myositis phase I/II trial." Molecular Therapy 32.11 (2024): 3821-3828.
6. Abstract 1733: Safety and Efficacy of CABA-201, a Fully Human, Autologous 4-1BB Anti-CD19 CAR T Cell Therapy in Patients with Immune-Mediated Necrotizing Myopathy and Systemic Lupus Erythematosus from the RESET-Myositis™ and RESET-SLE™ Clinical Trials. ACR 2024.

RESET™ clinical program for rese-cel, a CD19-directed CAR T

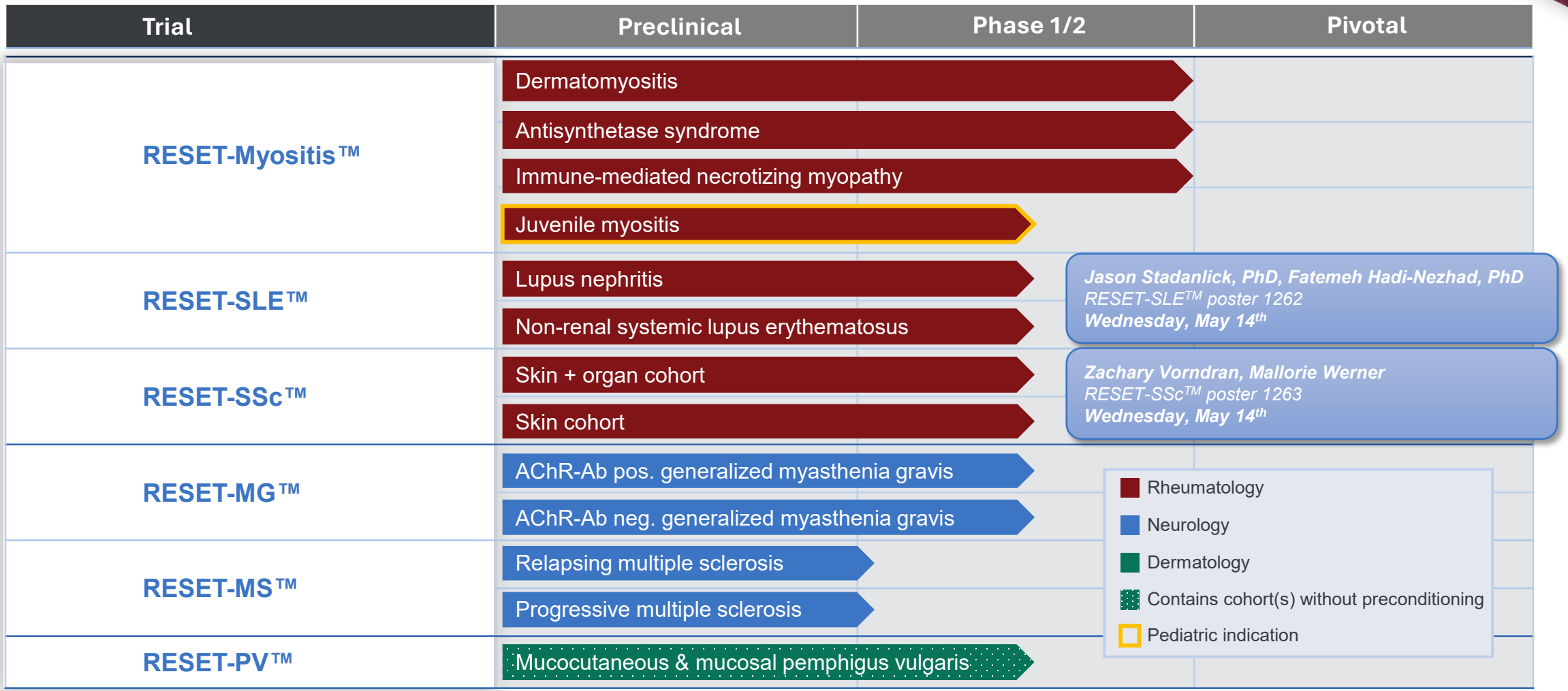
Multiple autoimmune diseases evaluated in disease-specific cohorts enrolling at over 60 sites in the US & Europe



AChR-Ab, acetylcholine receptor antibody; CABA, Cabaletta Approach to B cell Ablation; CAR, chimeric antigen receptor; RESET, REstoring SElf-Tolerance.
Cabaletta Bio: CABA-201. Available at: www.cabalettabio.com/pipeline/caba-201 (accessed October 2024).

RESET™ clinical program for rese-cel, a CD19-directed CAR T

Multiple autoimmune diseases evaluated in disease-specific cohorts enrolling at over 60 sites in the US & Europe



AChR-Ab, acetylcholine receptor antibody; CABA, Cabaletta Approach to B cell Ablation; CAR, chimeric antigen receptor; RESET, REstoring SElf-Tolerance.
Cabaletta Bio: CABA-201. Available at: www.cabalettabio.com/pipeline/caba-201 (accessed October 2024).

SLE and lupus nephritis: High unmet clinical need

Affects ~ 320,000 people in the US and >3 million globally; associated with multi-organ impacts and reduced quality of life[1–4]

Lupus is a chronic autoimmune disease affecting multiple organs, with potential for life threatening complications[1]

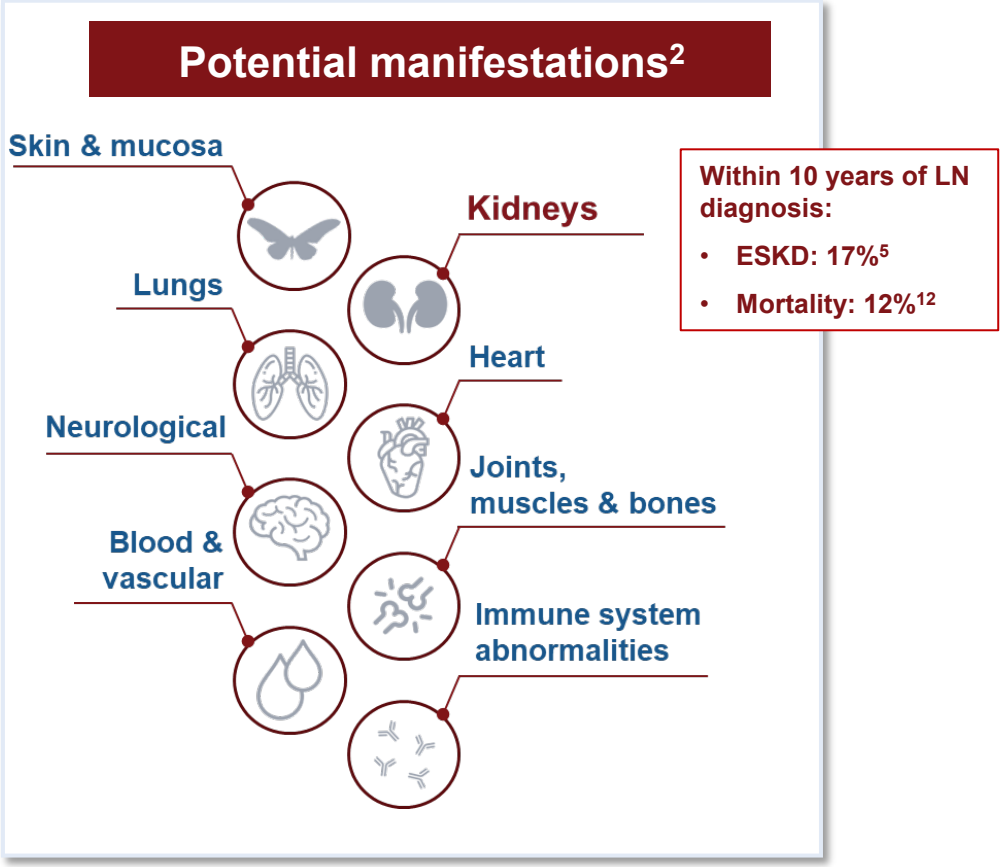
- ~40% of patients with SLE develop LN and face an increased risk of kidney failure and death[5]

Lupus negatively impacts quality of life, with fatigue as a common symptom

- Associated with higher mortality and diminished health-related quality of life compared with the general population[1,6]
- Disproportionately impacts women and people of color[5,7]

Current therapies include biologics, immunosuppressants and steroids

- Patients frequently require long-term immunosuppression[8]
- Durable, drug-free remission is rarely achieved[9]
- Current therapies carry significant burden for patients, including adverse effects and risk of relapse[10,11]



ESKD, end-stage kidney disease; LN, lupus nephritis; SLE, systemic lupus erythematosus.

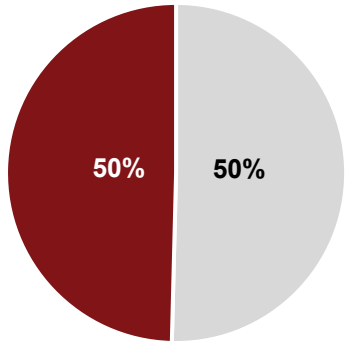
1. Zen M, et al. *Eur J Intern Med.* 2023;112:45–51. 2019;61:92–99. 2. Fu Q, et al. *Cell Mol Immunol.* 2021;18(8):2073–2074. 3. Helmick CG, et al. *Arthritis Rheum.* 2008;58(1):15–25. 4. Tian J, et al. *Ann Rheum Dis.* 2023;82(3):351–356. 5. Hoover PJ, et al. *Kidney Int.* 2016;90(3):487–92. 6. Refai RH, et al. *Sci Rep.* 2024;14(1):5234. 7. Lewis MJ, Jawad AS. *Rheumatology (Oxford).* 2017 Apr 1;56(suppl_1):i67-i77. 8. Kostopoulou M, et al. *Ann Rheum Dis.* 2024;83(11):1489–1501. 9. Nikfar M, et al. *Int J Clin Pract.* 2021;75(4):e13909. 10. Spies E, et al. *JMIR Form Res.* 2024;8:e52768. 11. Olesińska M, Saletra A. *Reumatologia.* 2018;56(1):45-54. 12. Hahn BH, et al. *Arthritis Care Res (Hoboken).* 2012 Jun;64(6):797-808.

Durable drug-free remission is rarely achieved in lupus[1]

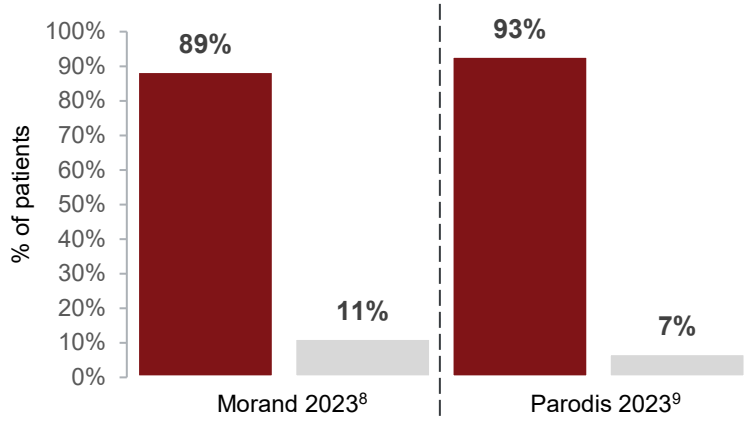
Lupus is associated with poor outcomes, requiring long-term and burdensome immunosuppressive therapy[2–6]

Systemic lupus erythematosus

SRI-4 response across all treatment arms in pooled data from biologic RCTs[7]

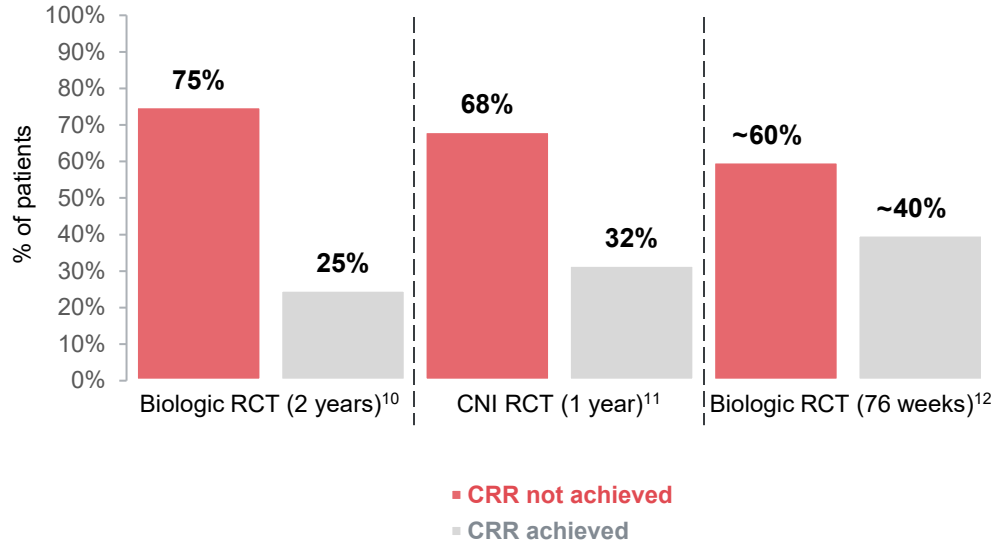


Patients achieving DORIS remission across all treatment arms in pooled data from biologic RCTs



Lupus nephritis

Patients achieving CRR across all treatment arms in advanced therapy RCTs



Response graphs are not representative of head-to-head trials and are provided for illustrative purposes only

CNI, calcineurin inhibitor; CRR, complete renal response; DORIS, definition of remission in SLE; RCT, randomized controlled trial; SLE, systemic lupus erythematosus; SRI, SLE Responder Index.

1. Nikfar M, et al. *Int J Clin Pract*. 2021;75(4):e13909. 2. Murimi-Worstell IB, et al. *BMJ Open*. 2020;10(5):e031850. 3. Gomez A, et al. *Front Med*. 2021;8:651249. 4. Refai RH, et al. *Sci Rep*. 2024;14(1):5234. 5. Spies E, et al. *JMIR Form Res*. 2024;8:e52768. 6. Olesińska M, Saletra A. *Reumatologia*. 2018;56(1):45–54. 7. Petri MA, et al. *Ann Rheum Dis*. 2022;81:323. Abstr No. POS0183. 8. Morand EF, et al. *Ann Rheum Dis*. 2023;82:33–34. Abstr No. OP0051. 9. Parodis I, et al. *Arthritis Rheumatol*. 2023;75 (Suppl 9). 10. Furie R, et al. *N Engl J Med*. 2020;383(12):1117–1128. 11. Rovin BH, et al. *Lancet*. 2021;397(10289):2070–2080. 12. Furie RA, et al. *N Engl J Med*. 2025;392(15):1471–1483.

B cells play a central role in the pathogenesis of lupus

B cells contribute to autoimmune diseases via four main mechanisms

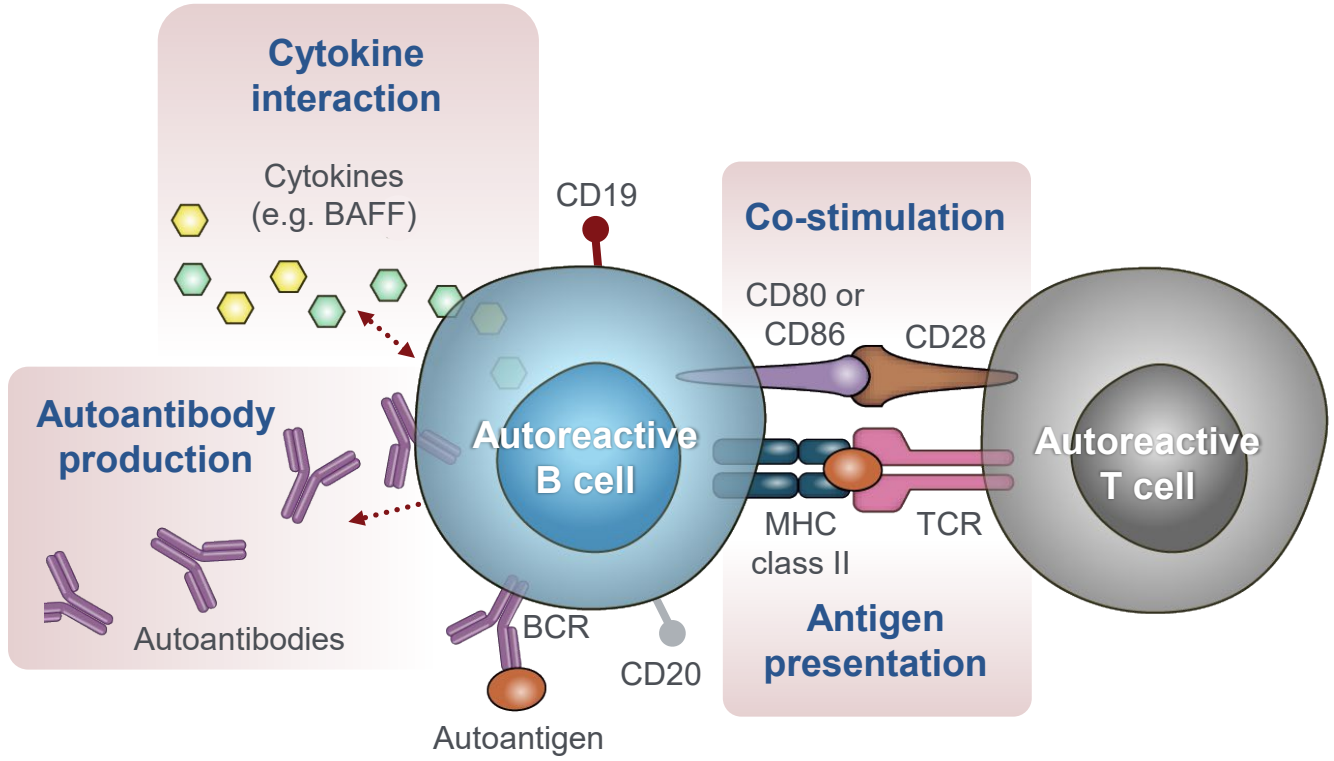


Image adapted from Rubin SJS, et al. 2019[2]

- B cells contribute to autoimmunity through a variety of mechanisms[1-3]
- B cell-directed therapies are important tools in the treatment of autoimmune diseases[3]

BAFF, B cell activating factor; BCR, B cell receptor; CD, cluster of differentiation; mAb, monoclonal antibody; MHC, major histocompatibility complex; RTX, rituximab; TCR, T cell receptor.

1. Schett G, et al. *Ann Rheum Dis*. 2024 Jun 20:ard-2024-225727. 2. Rubin SJS, et al. *Nat Rev Rheumatol*. 2019;15(5):303–315. 3. Barnas JL, et al. *Curr Opin Immunol*. 2019;61:92–99.

RESET-SLE™ phase 1/2 trial: key inclusion & exclusion criteria

Designed to evaluate the safety and tolerability of rese-cel in lupus subjects with active, refractory disease

Key inclusion criteria[1]

Evidence of active disease despite prior or current treatment with standard of care

- Age ≥18 and ≤65 with an SLE diagnosis
- Positive ANA or anti-dsDNA at screening
- **SLE (non-renal)**: active, moderate to severe SLE, SLEDAI-2K ≥8; pure class V LN patients eligible for this cohort
- **LN**: active, biopsy-proven LN class III or IV (± class V)

Key exclusion criteria[1]

B cell depleting agent within prior 3-6 months; Previous CAR T therapy and/or HSCT

- Presence of kidney disease other than LN
- Current symptoms of severe, progressive, or uncontrolled pulmonary or cardiac disease

ANA, antinuclear antibody; anti-dsDNA, anti-double strand DNA antibodies; CAR, chimeric antigen receptor; LN, lupus nephritis; SLEDAI-2K, SLE disease activity index 2000; SLE, systemic lupus erythematosus.

1. ClinicalTrials.gov. Available at: www.clinicaltrials.gov/study/NCT06121297 (accessed May 2025).

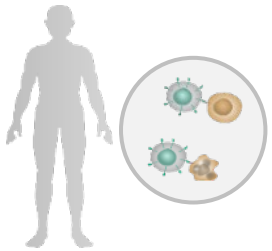
RESET™ clinical trials have consistent design principles

Individual trials in myositis, SLE, and SSc share common elements of preconditioning, dose, and study design

Screening

Day -5 to Day -3

Leukapheresis and rese-cel production



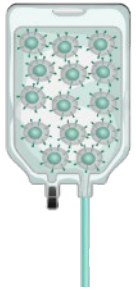
T cells isolated from patient's own PBMCs (autologous CAR T)

Preconditioning (PC)‡

FLU 25 mg/m² x 3 days
CY 1000 mg/m² x 1 day

Day 1

Single infusion of rese-cel



Weight-based dosing
1×10⁶ cells/kg

Day 29

Primary endpoint: Incidence and severity of AEs

Study follow-up through Year 3†

Additional Endpoints

- Adverse events & safety
- Clinical efficacy measuring:
  - Drug-free responses
  - Validated study-specific endpoints
- PK/PD analysis:
  - Rese-cel expansion
  - B cell depletion
  - B cell repopulation
- Biomarker analysis, including autoantibody levels

- Prophylactic tocilizumab is not currently used in the RESET™ protocols
- Patients discontinue all immunomodulatory medications before PC

†Follow up period encompasses 15 years in total, aligned to regulatory guidance for CAR T cell therapies. ‡ Preconditioning does not apply to RESET-PV™

AE, adverse event; CABA, Cabaletta Approach to B cell Ablation; FLU, fludarabine; CY, cyclophosphamide; PBMC, peripheral blood mononuclear cell; PD, pharmacodynamics; PK, pharmacokinetics; RESET, REstoring SElf-Tolerance; SLE, systemic lupus erythematosus; SSc, systemic sclerosis.

Cabaletta Bio: Data on file; 1. Peng BJ, et al. *Mol Ther Methods Clin Dev*. 2024;32(2):101267.

Baseline characteristics of first 7 patients in RESET-SLE™

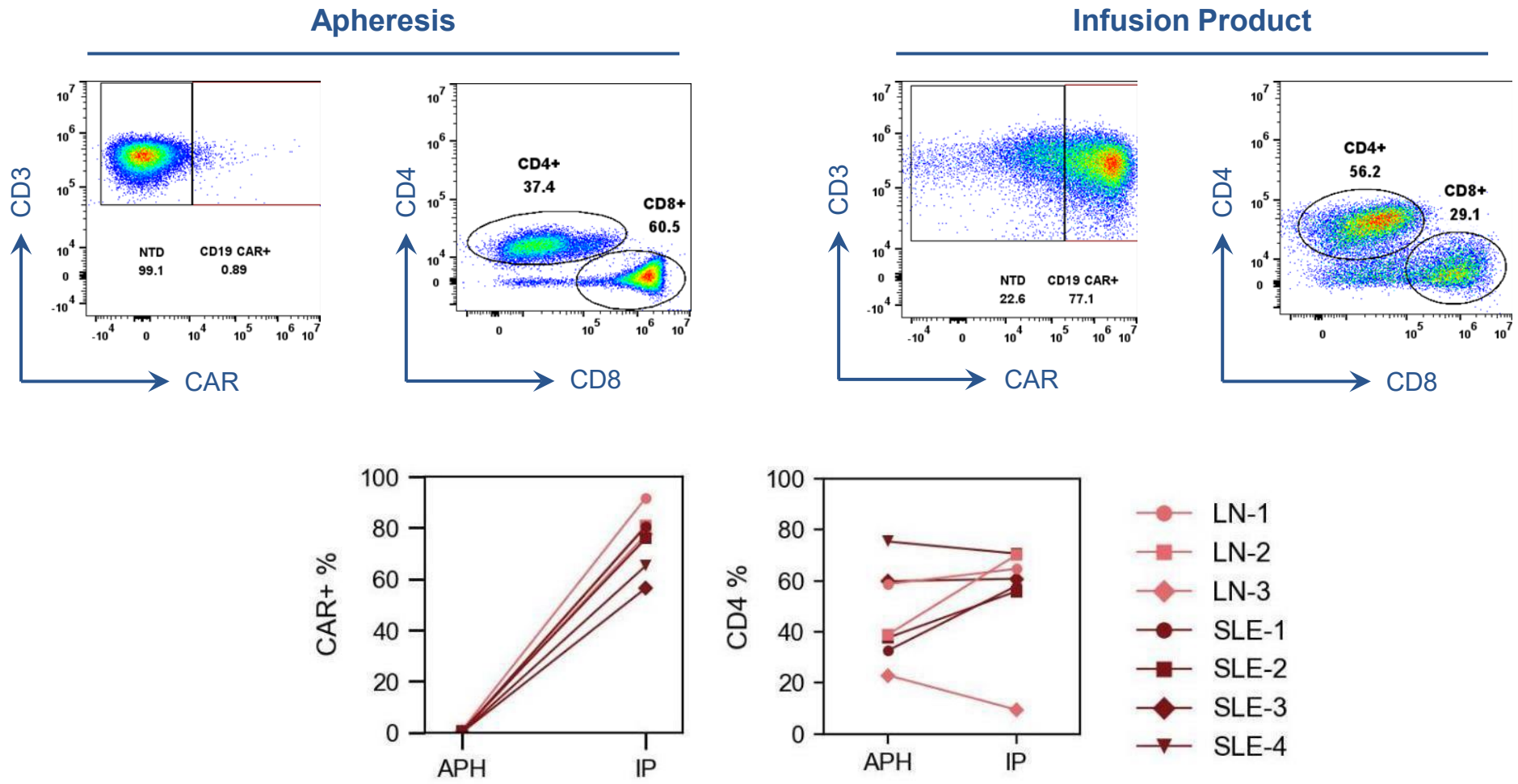
All patients in RESET-SLE™ had active, refractory disease and had failed B cell-targeting therapies

Cohort	Non-renal SLE				LN		
Patient / Cohort	**SLE-1†**	**SLE-2**	**SLE-3**	**SLE-4**	**LN-1**	**LN-2**	**LN-3**
Age, sex	26 M	36 F	44 F	37 F	24 F	35 F	26 F
Disease duration (y)	~6	~17	~9	~10	~2	~8	~16
Autoantibodies§	anti-dsDNA, anti-Sm	anti-dsDNA	anti-dsDNA	anti-dsDNA, anti-Sm	anti-dsDNA, anti-Sm	anti-dsDNA, anti-Sm	anti-Sm
Latest follow up	Week 44	Week 28	Week 20	Week 20	Week 32	Week 16	Week 4

†SLE-1 had class V LN; inclusion criteria for LN cohort requires class III/IV LN.
§All patients are antinuclear antibody positive.
dsDNA, double-stranded DNA; LN, lupus nephritis; RESET, REstoring SElf-Tolerance; SLE, systemic lupus erythematosus; Sm, Smith; y, years.
Cabaletta Bio: Data on file.

Rese-cel infusion product consists of mainly CD4+ CAR T cells

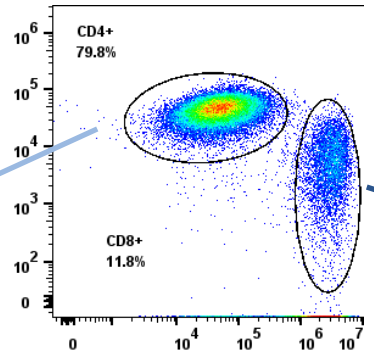
Percent transduced T cells is 50% or higher for all RESET-SLE™ subjects



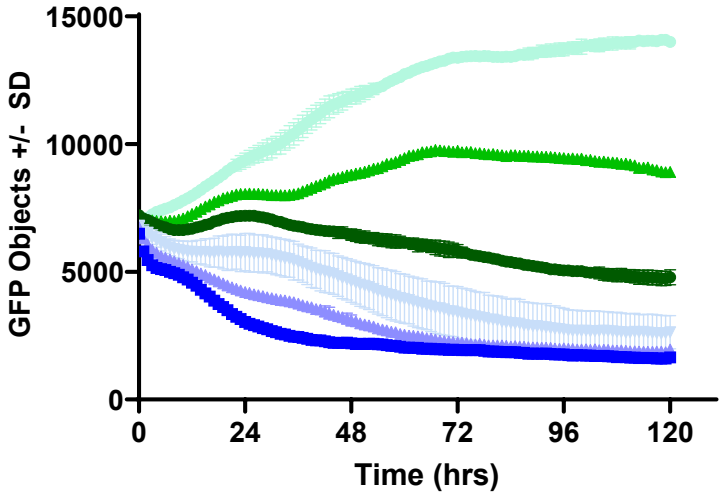
Apheresis and Infusion Product flow plots from SLE-2 shown. APH, apheresis; IP, infusion product.

CD4⁺ and CD8⁺ rese-cel populations exhibit cytolytic activity

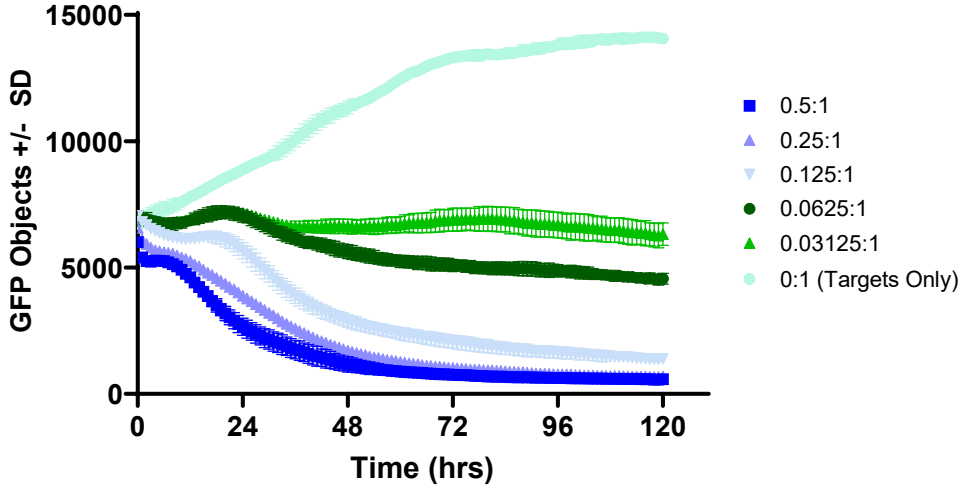
Similar cytolytic activity exhibited by both CD4⁺ and CD8⁺ rese-cel populations from an infusion product from a myositis patient



CD4⁺ rese-cel



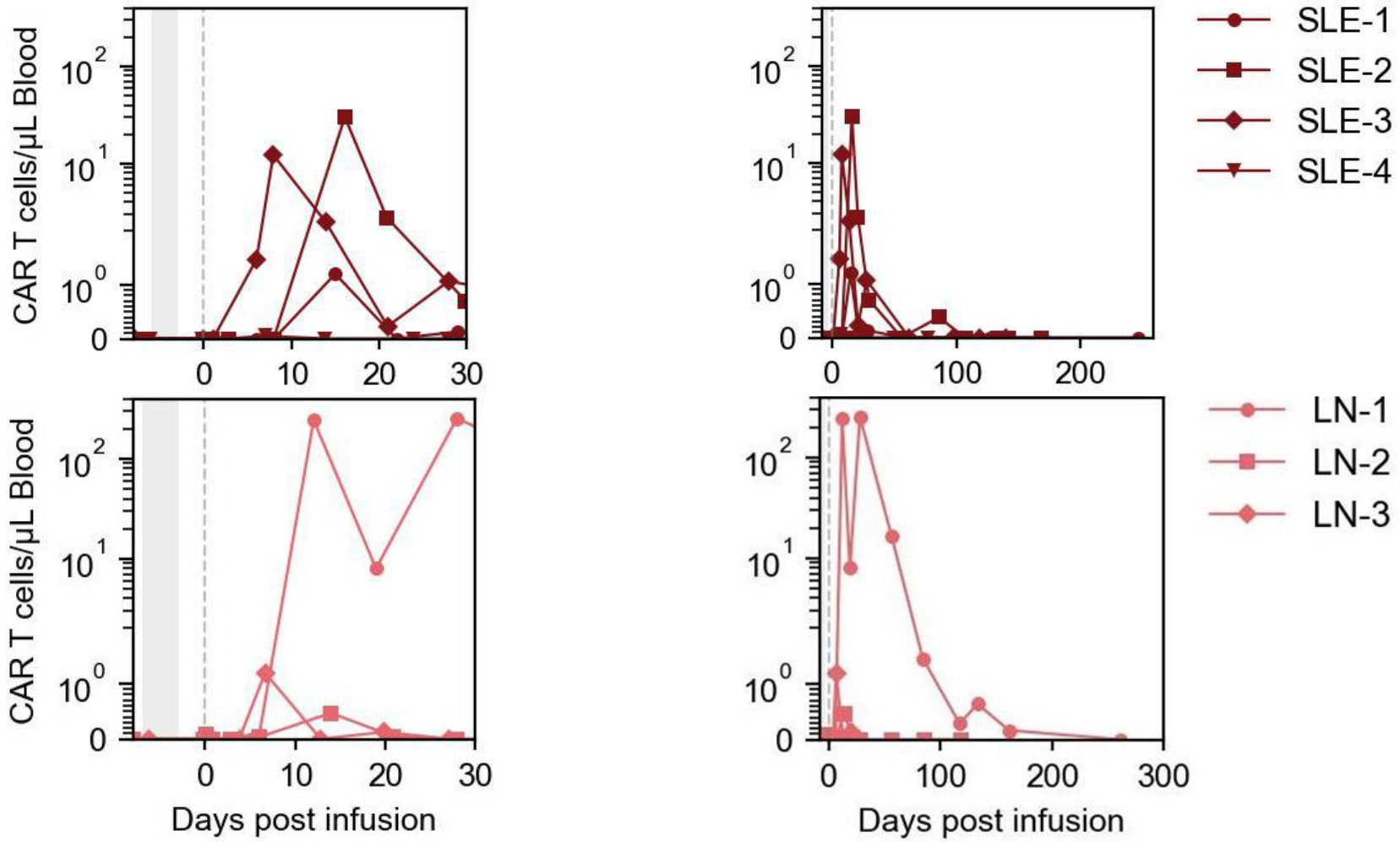
CD8⁺ rese-cel



Data shown from a subject in the immune-mediated necrotizing myopathy cohort of RESET-Myositis.

Rese-cel peak expansion is observed within 2 weeks post-infusion

Time to peak rese-cel concentration ranges from 7 to 16 days post-infusion*

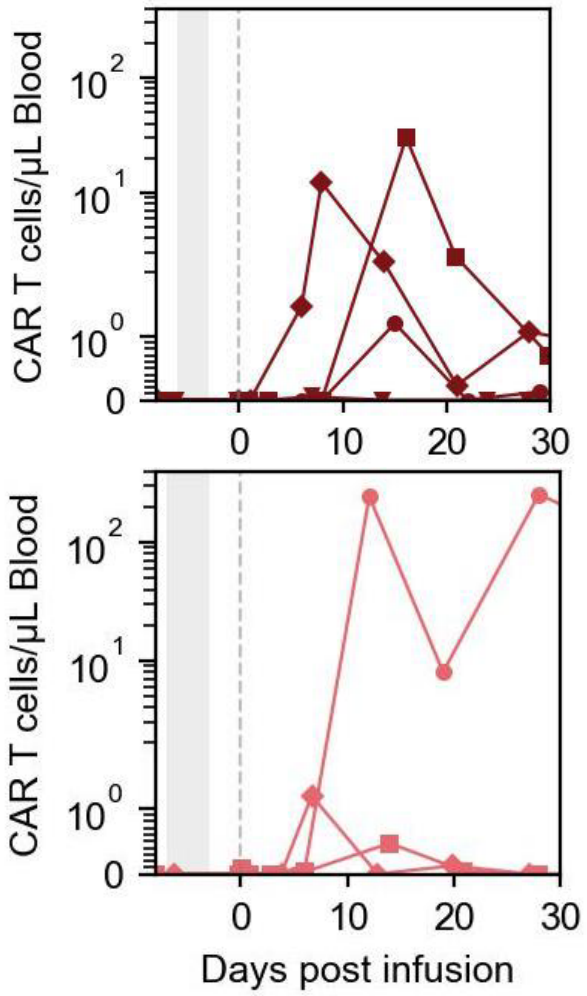


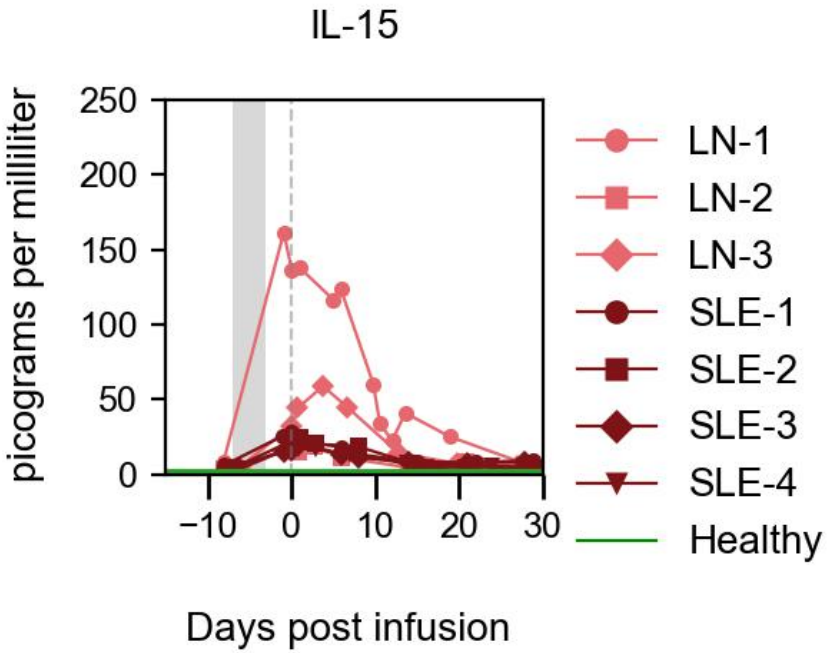
*Statement includes the first expansion peak for LN-1

CAR, chimeric antigen receptor; LN, lupus nephritis; rese-cel, resecabtagene autoleucel; SLE, systemic lupus erythematosus.

Rese-cel peak expansion is observed within 2 weeks post-infusion

Serum IL-15 levels relate inversely to white blood cell counts

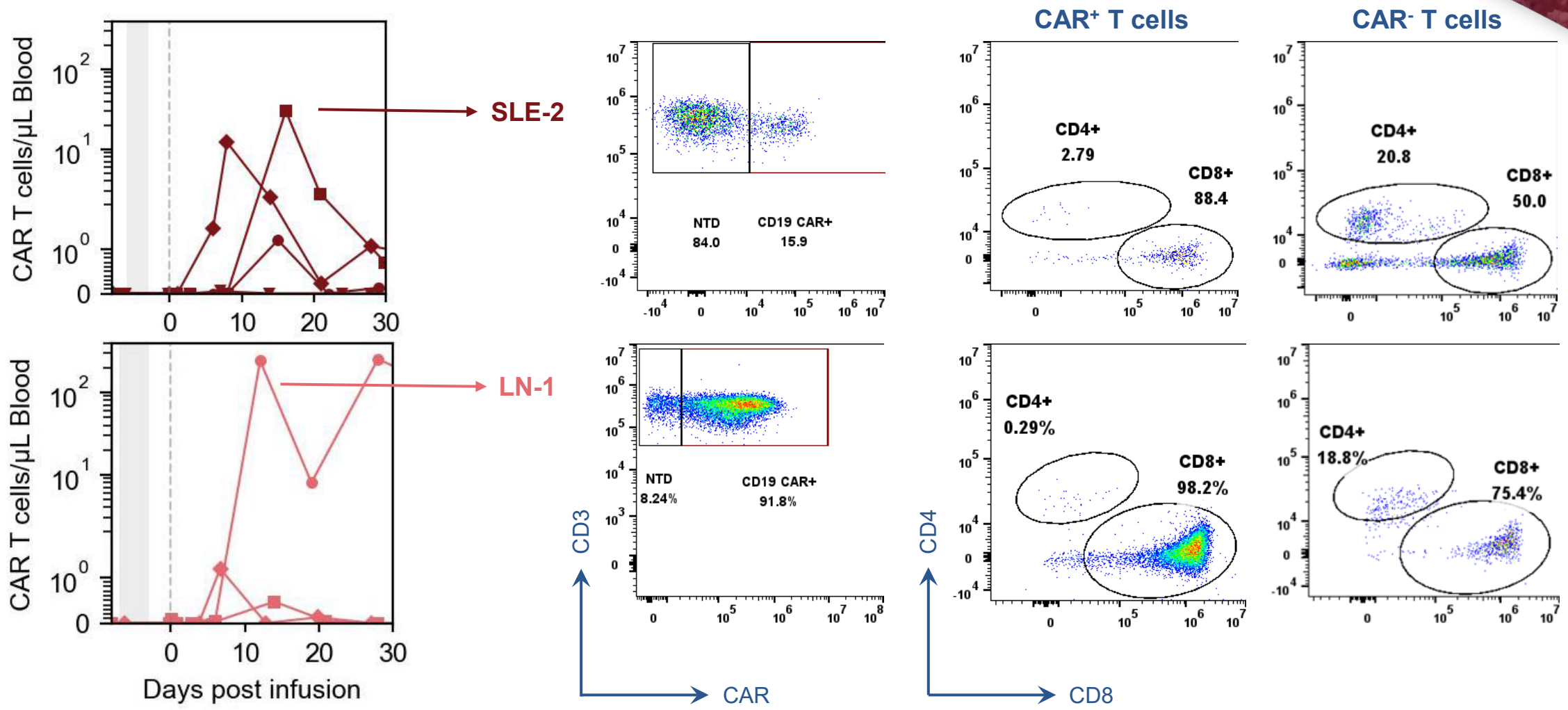




CAR, chimeric antigen receptor; LN, lupus nephritis; rese-cel, resecabtagene autoleucel; SLE, systemic lupus erythematosus; WBC, white blood cell.

Rese-cel peak expansion is observed within 2 weeks post-infusion

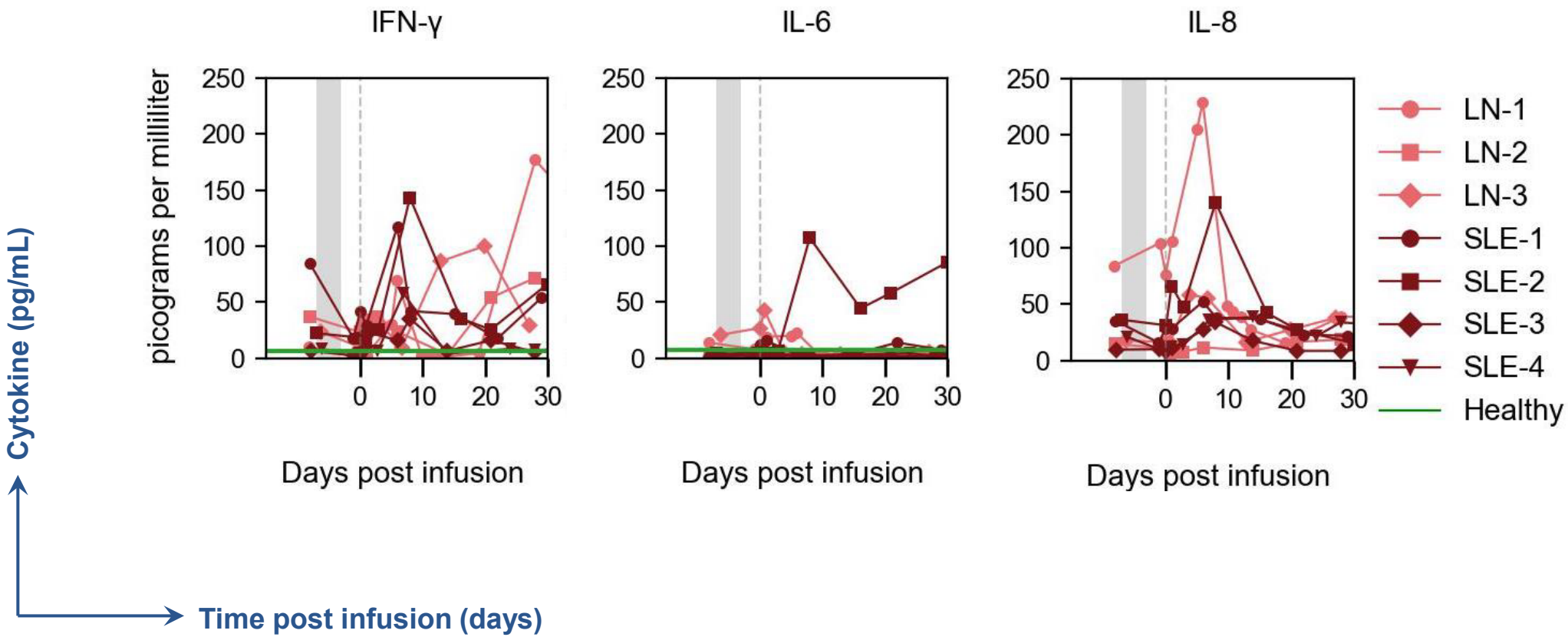
Rese-cel at peak expansion becomes CD8⁺ dominant in most patients



CAR, chimeric antigen receptor; LN, lupus nephritis; rese-cel, resecabtagene autoleucel; SLE, systemic lupus erythematosus.

Serum cytokine levels following rese-cel infusion

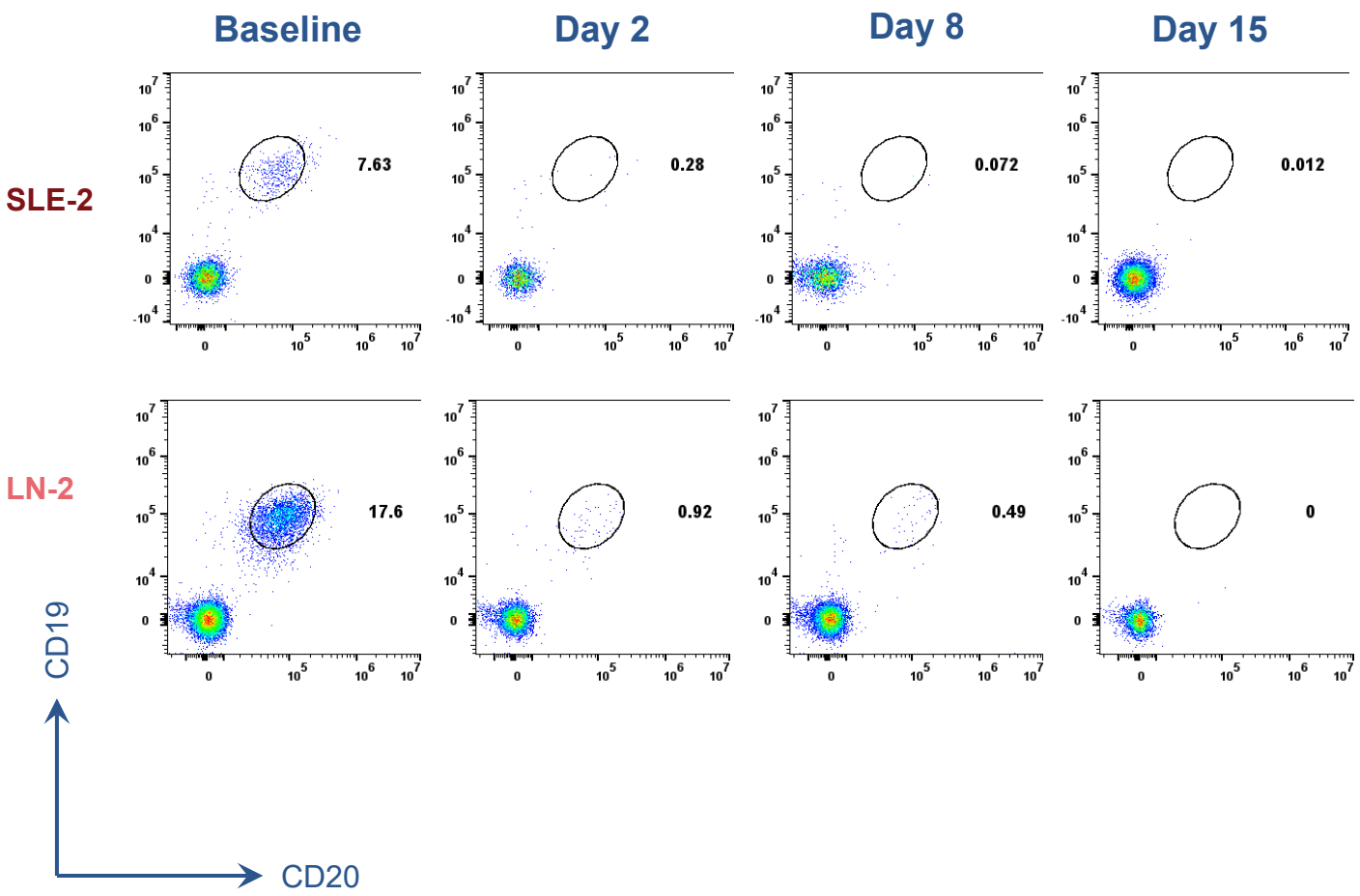
IFN-γ aligns with rese-cel activation; IL-6 and IL-8 correspond with safety events

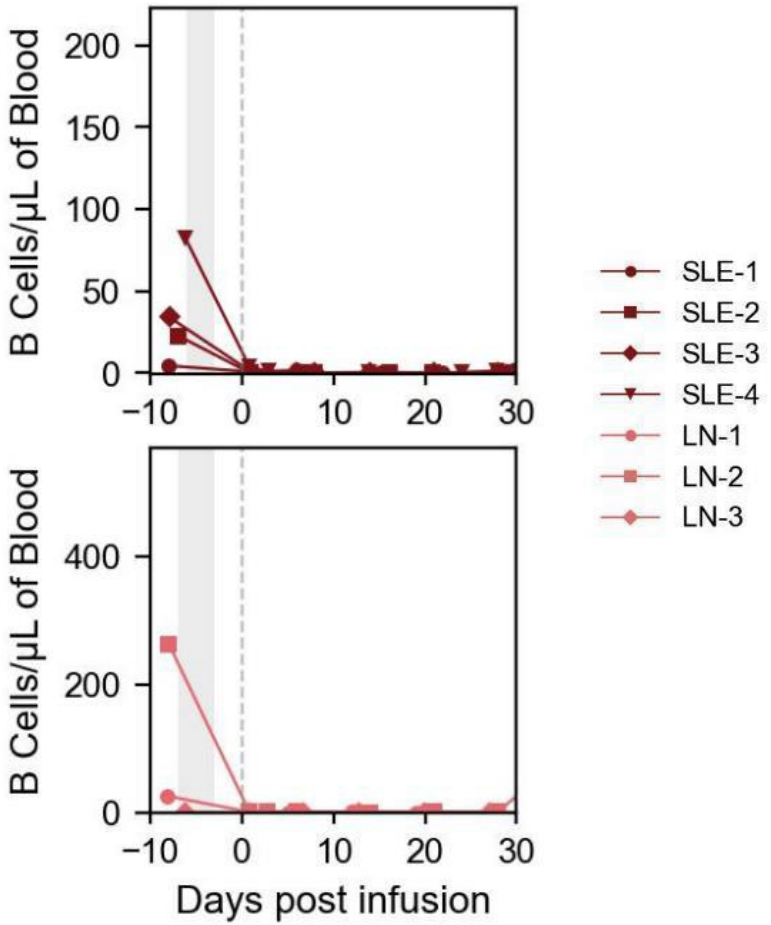


Serum cytokine levels quantified using mesoscale discovery (MSD) platform. C_{max}, maximum concentration; LN, lupus nephritis; rese-cel, resecabtagene autoleucel; SAE, serious adverse event; SLE, systemic lupus erythematosus.

Peripheral B cells are rapidly reduced after rese-cel infusion

Time to minimum B cell counts is approximately 2 weeks in lupus patients

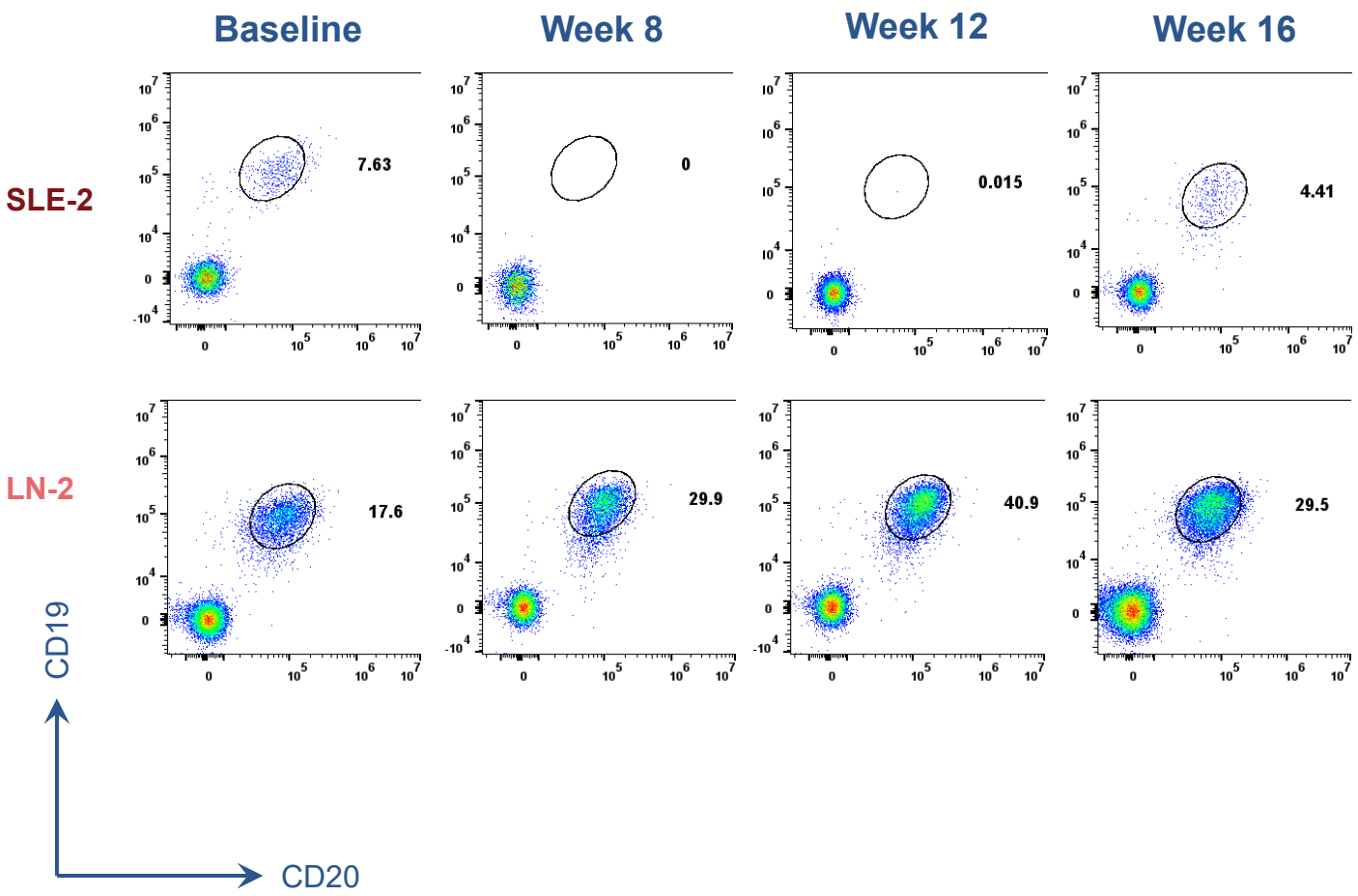


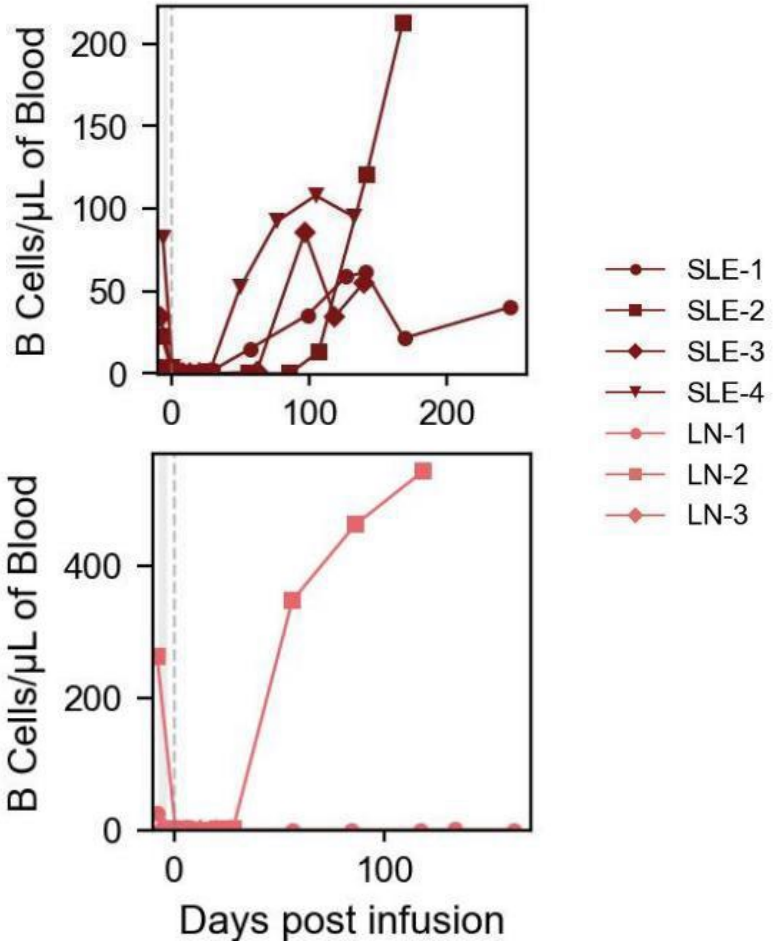


LLOD of B-cell flow assay is 0.03%. LN, lupus nephritis; rese-cel, resecabtagene autoleucel; SLE, systemic lupus erythematosus.

Peripheral B cell depletion is transient after rese-cel infusion

B cell repopulation occurs as early as 2 months post-infusion in lupus patients

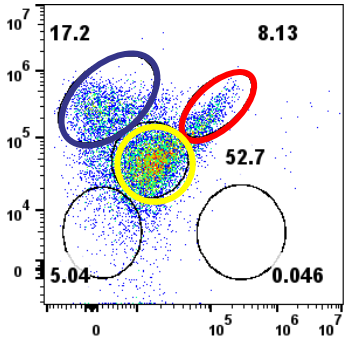




LLOD of B-cell flow assay is 0.03%. LN, lupus nephritis; rese-cel, resecabtagene autoleucel; SLE, systemic lupus erythematosus.

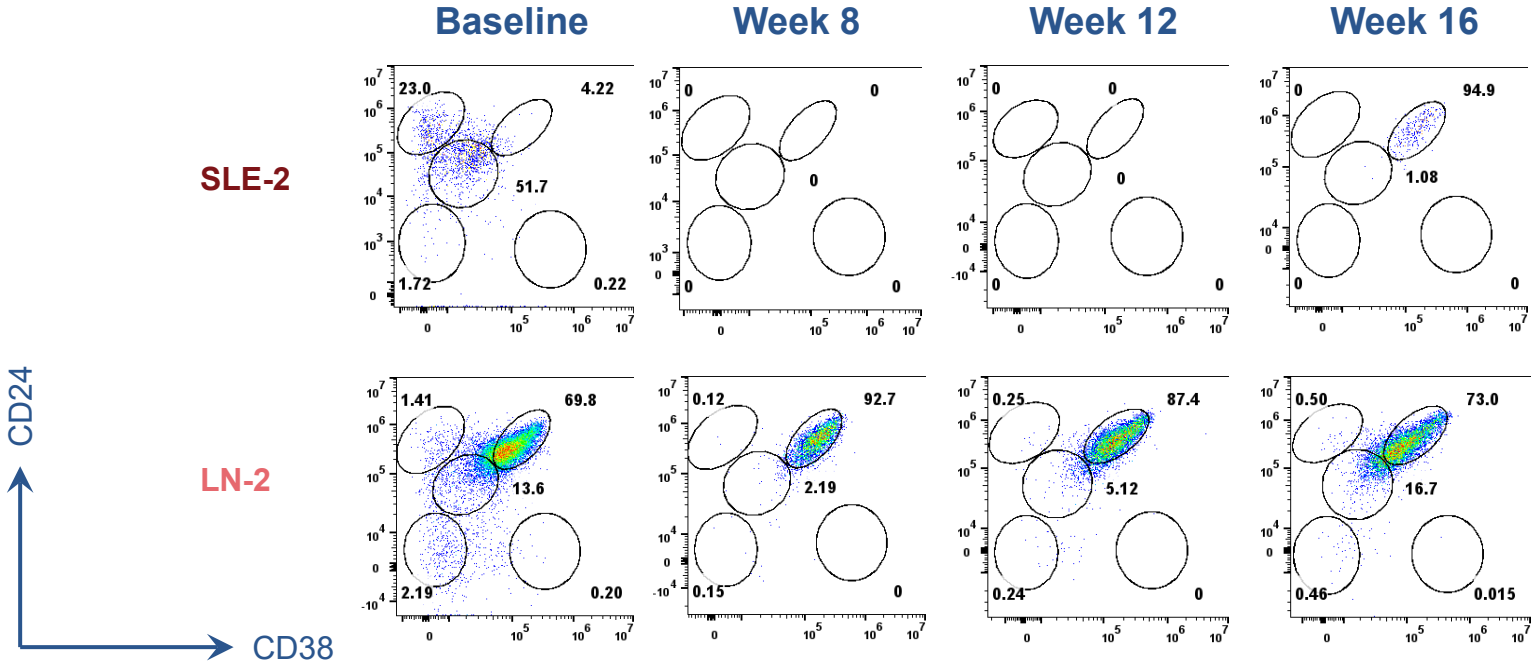
Re-emergent B cells are transitional naïve

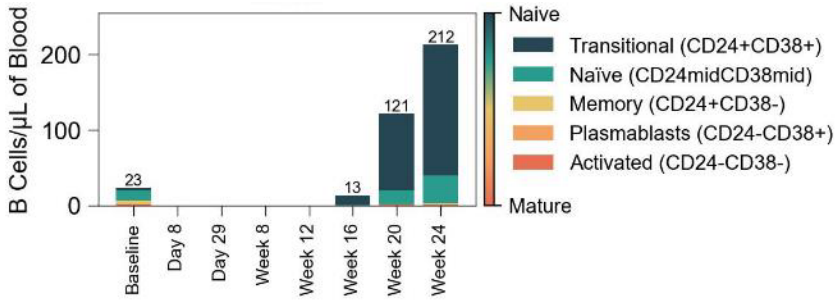
Transitional naïve B cells (CD24^{hi}CD38^{hi}) imply recent bone marrow emigration

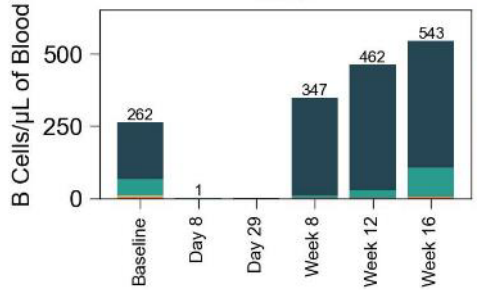


Transitional naïve
Naïve
Memory

Jason Stadanlick, PhD, Fatemeh Hadi-Nezhad, PhD
RESET-SLE™ poster – Abstract 1262
Wednesday, May 14th





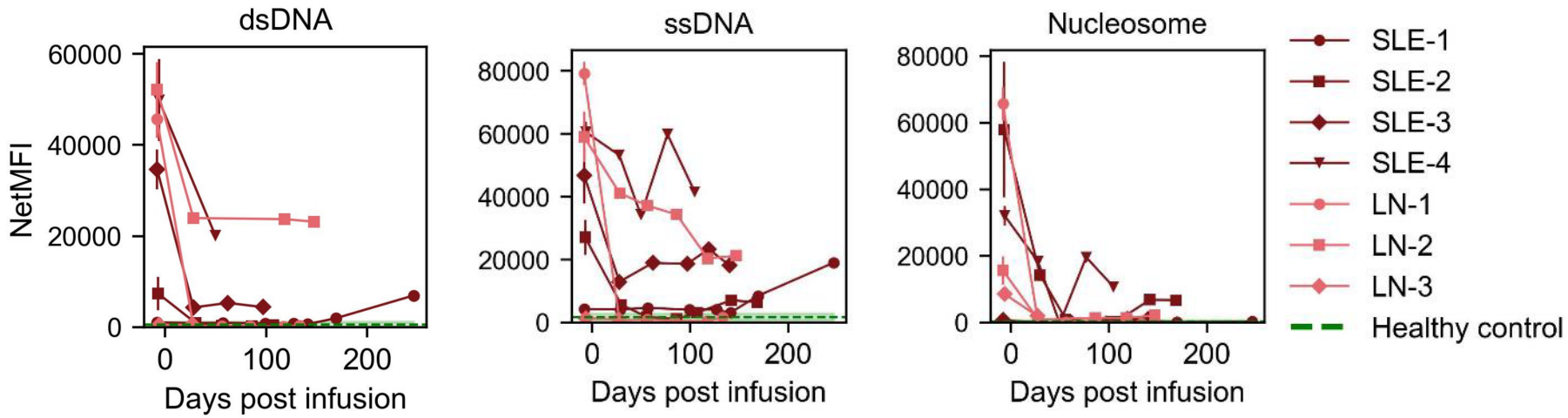


LN, lupus nephritis; SLE, systemic lupus erythematosus.

Serum autoantibody levels decrease after rese-cel infusion

High-sensitivity Luminex assay reflects decreases in lupus related antibodies post-infusion

- **SLE Autoantibody Luminex assay (RUO)**
  - 19 unique beads covalently-linked to nucleic acids or full-length protein antigen enabling detection of serum antibodies
  - More sensitive than some standard clinical tests

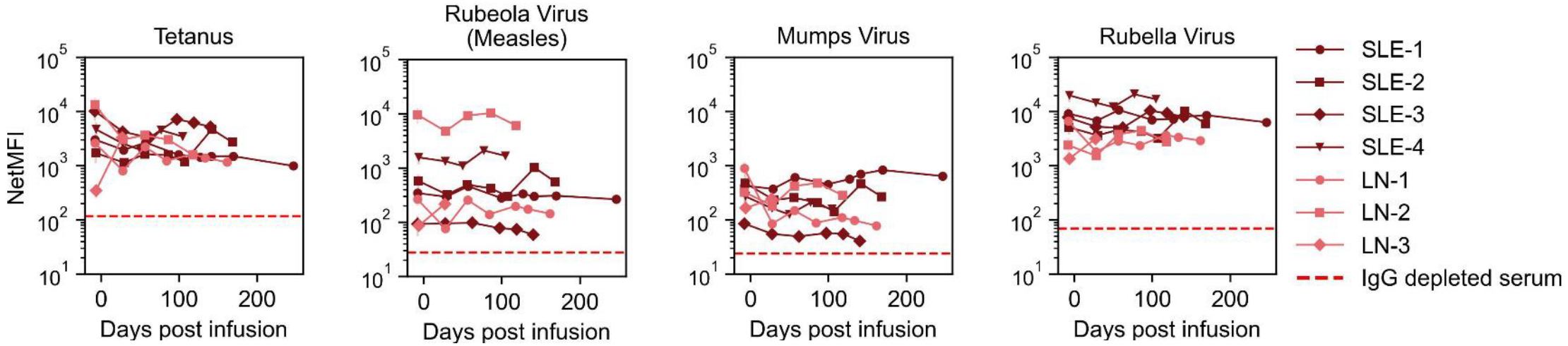


Healthy donor line represents a pool of sera from 3 donors run as an assay control. dsDNA, double stranded DNA; LN, lupus nephritis; SLE, systemic lupus erythematosus; ssDNA, single stranded DNA.

Serum vaccine antibody levels remain stable after rese-cel infusion

Antibodies to Tetanus, Measles, Mumps, and Rubella detected by Luminex assay

- **Vaccine Luminex assay (RUO)**
  - 14 unique beads covalently-linked to full-length or subunits of protein antigens enabling detection of serum antibodies



Antibody titer data generated using custom, high-sensitivity Luminex bead-based panel. LN, lupus nephritis; SLE, systemic lupus erythematosus.

Summary of early translational data from RESET-SLE™

- Infusion products are predominantly CD4+ in all but 1 patient
  - CD4+ T cells lyse target B cells similarly to CD8+ T cells
- Rese-cel peak expansion is observed by approximately 2 weeks after rese-cel infusion
  - In most patients, CAR T cell contraction was observed within 30 days post-infusion
  - CAR T cells are mainly CD8+ dominant at peak expansion
  - Serum IFN-γ induction was observed before or simultaneously with peak expansion
- B cells are rapidly reduced in blood following rese-cel infusion
  - B cells begin to reconstitute by 8 weeks post-infusion in most patients
  - B cells exhibit a transitional naïve B cell phenotype upon re-emergence
- Rese-cel provided compelling efficacy in highly active and refractory SLE and LN patients with a favorable safety profile to date
  - Clinical safety & efficacy data from the RESET-SLE™ trial will be shared at the EULAR Congress in June 2025

As of data cutoff March 31st, 2025. CAR, chimeric antigen receptor; IFN-γ, interferon-gamma; LN, lupus nephritis; rese-cel, resecabtagene autoleucel; SLE, systemic lupus erythematosus

Acknowledgements

Patients and caregivers involved in the RESET™ clinical program

Site investigators and staff involved with these patients from the RESET™ clinical program

- Children's Hospital of Philadelphia
- University of California, Davis
- University of North Carolina
- University of Rochester Medical Center
- Massachusetts General Hospital

Cabaletta Bio team

- Biostatistics
- Clinical Development
- Clinical Operations
- Computational Biology
- Manufacturing
- Medical Affairs
- Translational Medicine
- Quality Assurance