Cabaletta Bio®

CAR T-cells to treat autoimmunity

NATIONAL ACADEMY OF MEDICINE OCT 2024

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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 and CARTA technologies and CABA™ platform; our ability to grow our autoimmune-focused pipeline; the ability to capitalize on and potential benefits resulting from the translational research partnership with Professor Georg Schett and the exclusive license agreement with IASO Bio; our expectations around the potential success and therapeutic benefits of CABA-201, including our belief that CABA-201 may enable an "immune system reset" and provide deep and durable responses for patients with Systemic Lupus Erythematosus ("SLE") and potentially for patients diagnosed with other autoimmune disease; our plans to initiate a Phase 1/2 clinical trial of CABA-201 in patients with SLE, including our anticipated progress, clinical trial design, ability to leverage our experience in autoimmune cell therapy and lupus product development; our planned initial clinical data read-out in the first half of 2024; our ability to enroll the requisite number of patients, dose each dosing cohort in the intended manner in our Phase 1/2 clinical trial of CABA-201; the timing any planned regulatory filings for our development programs; the progress and results of our DesCAARTes™ Phase 1 trial, including the significance and impact around reported safety and clinical and translational data of cohorts from our DesCAARTes™ trial, and our ability to advance dose escalation and initiate combination cohorts and to optimize our targeted cell therapy; our ability to implement a pre-treatment regimen and the potential ability to enhance in vivoDSG3-CAART exposure; the therapeutic potential and clinical benefits of our product candidates; the expectation that Cabaletta may improve outcomes for patients suffering from mucosal pemphigus vulgaris, myasthenia gravis, or other autoimmune diseases; our ability to escalate dosing as high as 10 to 15 billion cells in cohort A6m, initiate dosing in a combination cohort or otherwise; our ability to implement a pre-treatment regimen and the potential ability to enhance *in vivo* DSG3-CAART exposure; our ability to evaluate, and the potential significance of, the relationship between DSG3-CAART persistence and potential clinical responses in patients with mPV; our ability to safely retreat additional patients and whether we will continue to observe a lack of immune-mediated clearance of DSG3-CAART cells after retreatment and repeat dosing of patients; our ability to successfully complete our preclinical and clinical studies for our product candidates, including CABA-201, our ongoing Phase 1 DesCAARTes™ trial, and our ongoing Phase 1 MusCAARTes™ trial of MuSK-CAART, including our ability to enroll the requisite number of patients, dose each dosing cohort in the intended manner, and progress the trial; the ability of MuSK-CAART to target B cells that differentiate into antibody secreting cells, which produce autoantibodies against muscle-specific kinase; 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 Orphan Drug Designation and Fast Track Designation for our product candidates, as applicable; the further expansion and development of our modular CABA™ platform across a range of autoimmune diseases; our ability to contract with third-party suppliers and manufacturers, implement an enhanced manufacturing process and further develop our internal manufacturing strategy, capabilities and facilities; our potential commercial opportunities, including value and addressable market, for our product candidates; our expectations regarding our use of capital and other financial results; and our ability to fund operations into the first quarter of 2025. 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.

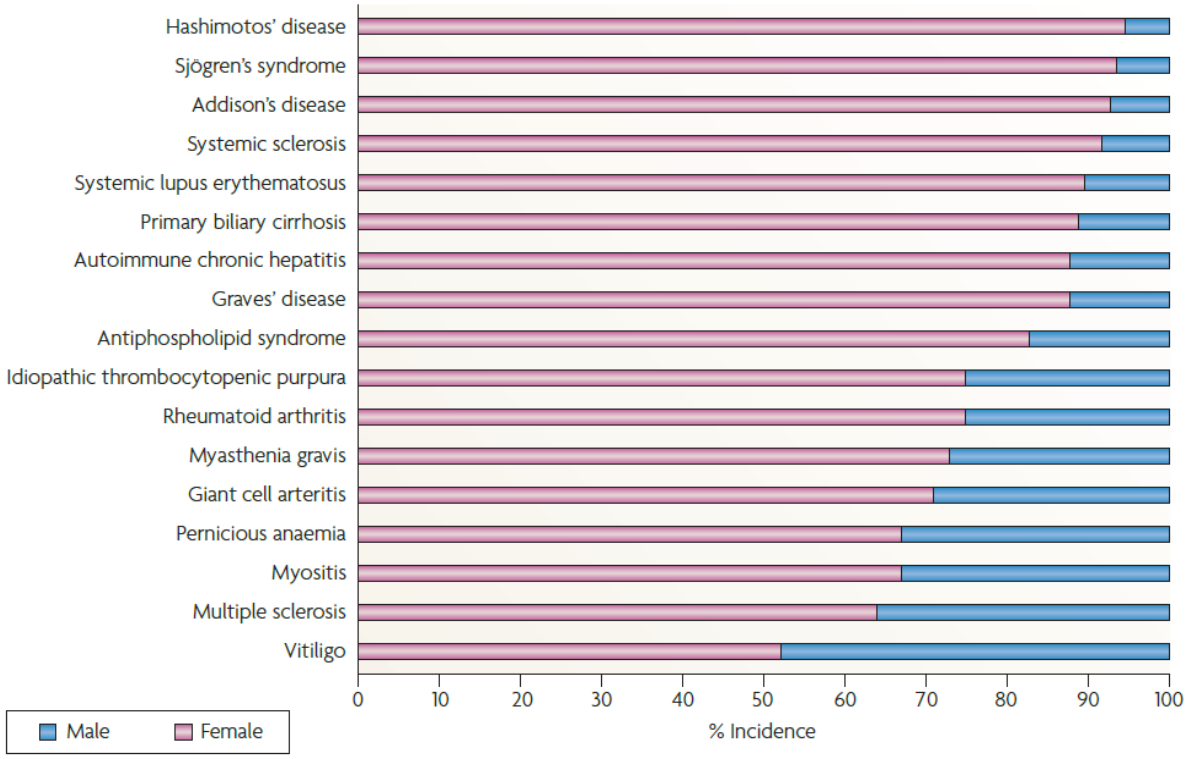
Various risks, uncertainties and assumptions could cause actual results to differ materially from those anticipated or implied in our forward-looking statements. Such risks and uncertainties include, but are not limited to, risks related to the success, cost, and timing of our product candidate development 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 CABA-201, DSG3-CAART and MuSK-CAART, the risk that the results observed with the similarly-designed construct employed in the recent *Nature Medicine* publication are not indicative of the results we seek to achieve with CABA-201, our plans to evaluate additional cohorts in the DesCAARTes™ trial, including a cohort implementing a pre-treatment regimen, the risk that signs of biologic activity or persistence may not inform long-term results, the risk that persistence observed with effective CART-19 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 protect and maintain our intellectual property position, risks related to our relationship 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 Designation and Fast Track Designations, 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, the impact of COVID-19 on the timing, progress, interpretability of data, and results of ongoing or planned clinical trials and risks relating to as a result of extraordinary events or circumstances such as the COVID-19 pandemic, and any business interruptions to our operations or to those of our clinical sites, manufacturers, suppliers, or other vendors resulting from the COVID-19 pandemic or similar public health crisis. 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.

Overview

- Autoimmune diseases background
  - Hypotheses as to why women have higher incidence and prevalence as compared to men
- Treatment options
  - B cell depletion – why it works and why it doesn't
- CD19 CAR T for Autoimmune disease
  - CAR-T background
  - Emerging data in SLE, myositis, and systemic sclerosis
- How to design **preclinical** and translational studies to ensure accurate representation of women
  - CABA-201 IND enabling studies

Autoimmune Disease – Global Impact

- An estimated 4.5% of the world's population lives with autoimmune disease[1]
  - Incidence and prevalence for autoimmune disease is substantially higher in women (compared to men)[1,5]
    - All autoimmune diseases – 4:1
    - Sjogren's - 19:1
    - SLE - 9:1
- Estimated economic burden of >$100 billion[2]
- Incidence is increasing[3,4]
  - Environmental factors
  - Improved surveillance and diagnoses
- Represents a global unmet medical need for which new therapies are needed

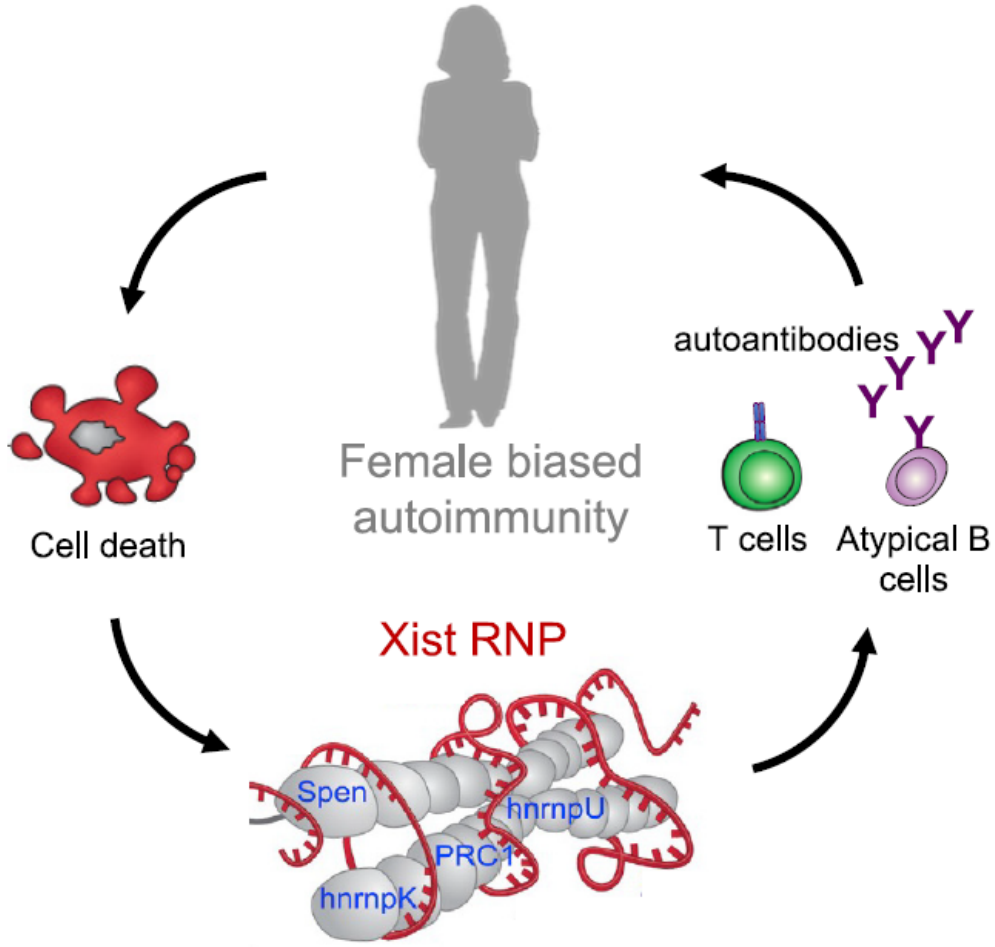


1. Hayter and Cook (2012) Updated assessment of the prevalence, spectrum and case definition of autoimmune disease. *Autoimmune Rev*
2. AARDA (2011) The Cost of Autoimmune Disease.
3. Dinse *et al* (2020) Increasing Prevlance of Antinuclear Antibodies in the United States. *Arthritis Rheumatol*
4. Rose (2016) Prediction and Prevention of Autoimmune Disease in the 21st Century. *Am J Epidemiol*
5. Libert et. al (2010), The X chromosome in immune functions: when a chromosome makes the difference. *Nature Reviews Immunology*

Why are women so prone to autoimmune disease?

Reactivation or breakthrough hypothesis as to why women are impacted disproportionally to autoimmune disease

- Many genes that control immune activity are present on the X-chromosome[1,2]
  - TLR7 (implicated in SLE)
  - CD40L (implicated in SLE)
- Breakthrough/loss of X-chromosome inactivation (XCI) can result in increased expression of key genes responsible for driving immune activity
  - long non-coding RNA (lncRNA) Xist is responsible for XCI
  - Xist is only found in XX and XXY individuals and is in, itself, **immunogenic** and associates with DNA and RNA auto-antigens in autoimmune disease patients
- Murine studies that express non-functional Xist in male mice show a similar pattern of autoimmunity as compared to female mice[1]



1. Dou et. al (2024), Xist ribonucleoproteins promote female sex-based autoimmunity. *Cell*
2. Libert et. al (2010), The X chromosome in immune functions: when a chromosome makes the difference. *Nature Reviews Immunology*

Current Treatment Modalities for Autoimmune Disease

Therapies listed in **bold** represent standard of care therapies commonly used to treat SLE

- Systemic therapies
  - Metabolic inhibitors: **mycophenolate mofetil** and methotrexate
  - Immune suppressants: **hydroxychloroquine**, and corticosteroids (**prednisone**), **voclosporin**
  - Cytotoxic therapies: cyclophosphamide

- Targeted therapies
  - B cell depletion: rituximab[1]
  - Cytokine blockers: **belimumab** (anti-BAFF) and **anifrolumab** (anti-IFNAR1)
  - T and B cell signaling blockade: BTK and JAK inhibitors

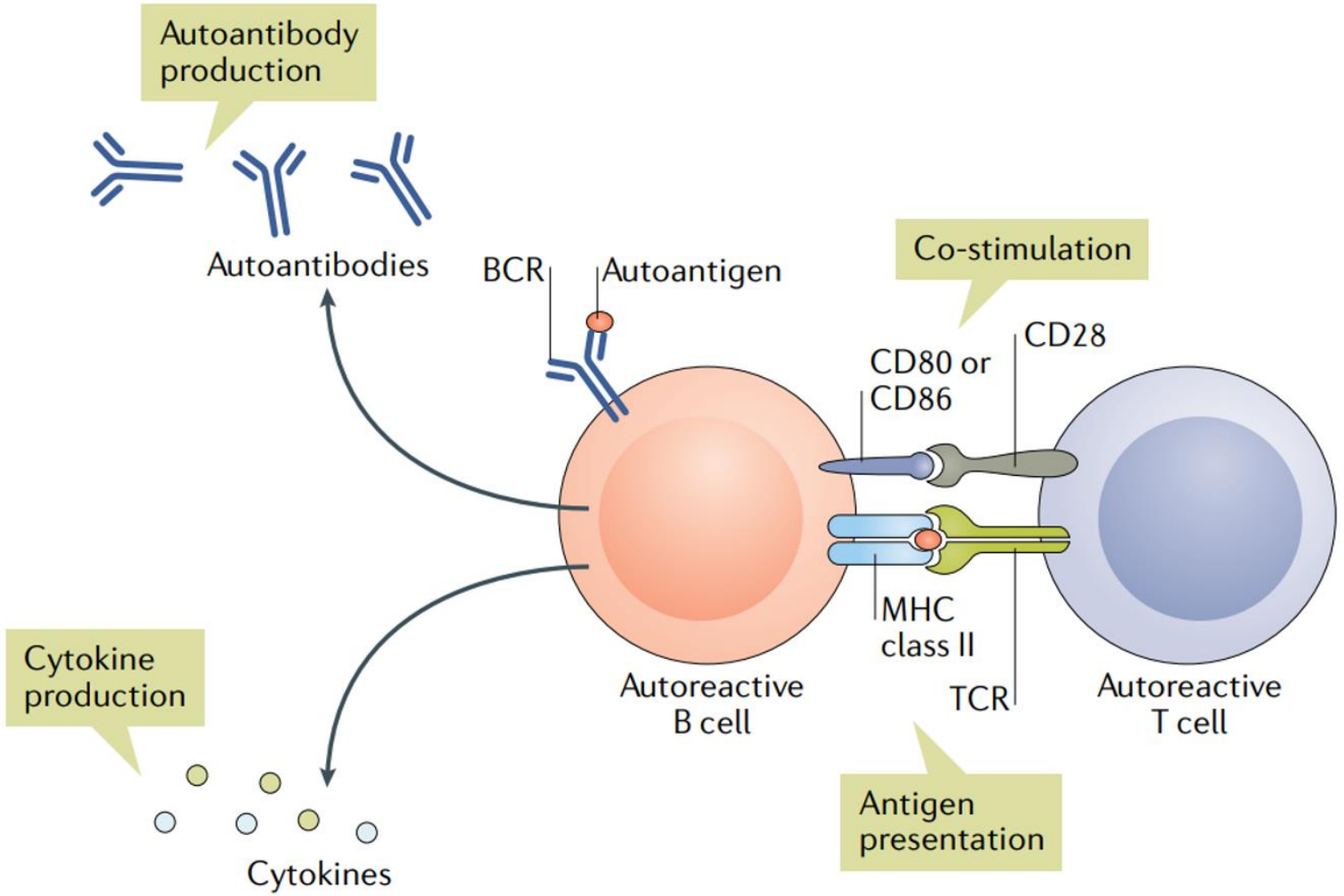
- These therapies remain largely non-curative, requiring chronic therapy

1. Kaegi *et al* (2019), Safety and Efficacy of Rituximab in Treating Immune-Mediated Disorders, *Front Immunol.*

B cell depletion is effective in diseases caused by both T and B cells

WHY?....

….because B cells play a central role in driving (autoreactive) T cell responses[1]



1. Rubin, Bloom and Robinson (2019) B Cell Checkpoints in Autoimmune Rheumatic Diseases; *Nat Rev Rheum*

Rituximab is not Commonly Curative

WHY?...

- **…because Rituximab does not deplete all B cells within tissues[1]**
  - Difficulty in tissue penetration
  - Requirement for effector mechanisms to deploy cytotoxic effect
  - Therefore, requires repeat administration
  - This induces prolonged B cell aplasia
- Newer generations of anti-B cell depleting agents are emerging – may work better[2]

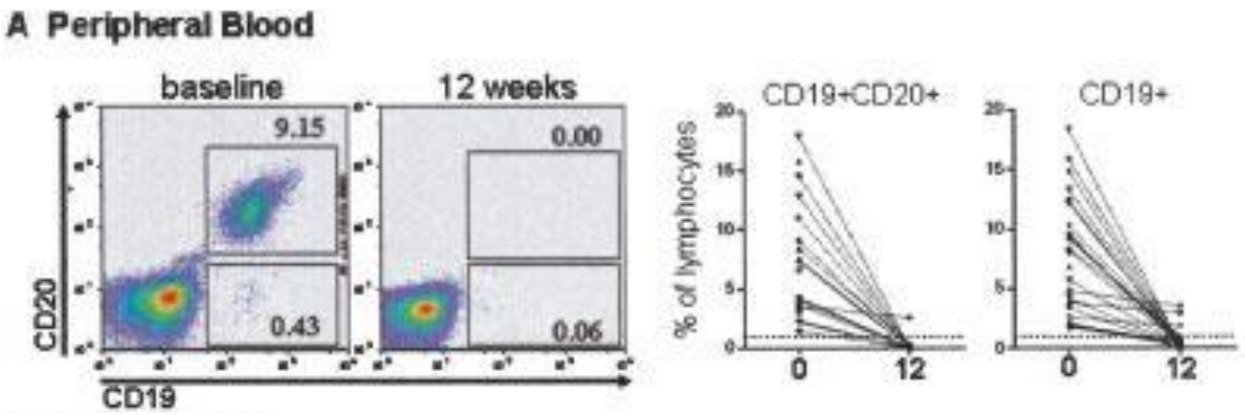
Organs	Diseases	RTX regimen	Other therapies	Delay between last RTX infusion and analysis	Number of patients	Residual B-cell populations	Reference no.
Bone marrow	ITP	4 weekly doses of 375 mg/m^2		3 mo	1	None	38
	RA	2 doses of 1000 mg (2 wk apart)		3 mo	6	Presence of residual CD19$^+$ cells (0.1% to 3.25% in the lymphoid gate), mainly precursors and immature B cells	39
	RA	2 doses of 1000 mg (2 wk apart)		3 mo	25	8 of 25 patients (32%) had residual CD19$^+$ cells (median 2.21%). No CD20$^+$ cells	40
	RA	2 doses of 1000 mg (2 wk apart)		3 mo	8	No significant reduction in CD19$^+$ cells numbers	41
	RA	2 doses of 1000 mg (2 wk apart)		1 to 3 mo	24	Presence of immature and/or transitional B cells (CD38^{++}CD24^{++}) and CD27$^+$IgD$^-$ B cells, while IgD$^+$ cells were completely depleted	42
Spleen	ITP	4 weekly doses of 375 mg/m^2		3 mo	1	None	38
	ITP	4 weekly doses of 375 mg/m^2	i.v. Ig (4 of 15)	3 to 6 mo	15	CD19$^+$ cells represented 0.06% to 1% of CD45$^+$ cells, including 75% plasma cells. Remaining CD19$^+$ cells were mostly CD27$^+$IgD$^-$ memory B cells. No residual germinal center	19
	AIHA	2 doses of 1000 mg (2 wk apart)	High-dose steroids	4 to 5 mo	4	CD19$^+$ cells represented 1.45% of CD45$^+$ cells, including 70% plasma cells. Remaining CD19$^+$ cells were mostly CD27$^+$IgD$^-$ memory B cells. No residual germinal center	43
	ITP	4 weekly doses of 375 mg/m^2 or 2 doses of 1000 mg (2 wk apart)	i.v. Ig (8 of 10) Steroids (1 of 10)	3 to 15 mo	10	CD19$^+$ cells represented 5.1% of splenocytes, but some patients had already reconstituted their B cells	44
	Prevention or treatment of antibody-mediated rejection	Single dose of 375 mg/m^2	Tacrolimus, mycophenolate mofetil, steroids, i.v. Ig, and rATG	2 to 45 d	7	Presence of few residual CD20$^+$CD79a$^+$ cells in some patients	45
Lymph nodes	Prevention of antibody-mediated rejection	Single dose of 500 mg	Tacrolimus, mycophenolate mofetil, steroids	1 mo	5	20% of lymphoid cells were CD19$^+$ (but not CD20$^+$), mainly CD27$^+$IgD$^-$ memory B cells	46
	Prevention of antibody-mediated rejection	Single dose of 375 mg/m^2	Tacrolimus, mycophenolate mofetil, steroids, i.v. Ig	1 mo	4	35% of lymphoid cells were CD19$^+$ (but not CD20$^+$), mainly CD27$^+$IgD$^-$ memory B cells	47
	RA	2 doses of 1000 mg (2 wk apart)		1 mo	14	10% of lymphoid cells were CD19$^+$, mainly CD27$^+$IgD$^-$ memory B cells	48
Salivary glands	Sjögren syndrome	2 doses of 1000 mg (2 wk apart) then 1000 mg every 6 mo		6 mo	19	No CD20$^+$ B cells. Ectopic germinal centers reduced from 53% to 5%	49
	Sjögren syndrome	2 weekly doses of 375 mg/m^2		4 mo	8	No residual B cells	50
Synovial tissue	RA	2 doses of 1000 mg (2 wk apart)		1 mo	17	Presence of residual CD22$^+$ B cells	51
	RA	2 doses of 1000 mg (2 wk apart)		2 to 6 mo	17	Presence of CD20$^+$ B cells in 2 of 17 patients (22%) and of CD79a$^+$ B cells in all patients	40
	RA	2 doses of 1000 mg (2 wk apart)		1 and 4 mo	16	Presence of residual CD22$^+$ B cells	52

1. Table excerpted from: Crickx et al (2020) Anti-CD20-mediated B-cell Depletion in autoimmune diseases
2. Furie RA, et al (2022) B-cell depletion with obinutuzumab for the treatment of proliferative lupus nephritis: a randomised, double-blind, placebo-controlled trial. Ann Rheum Dis

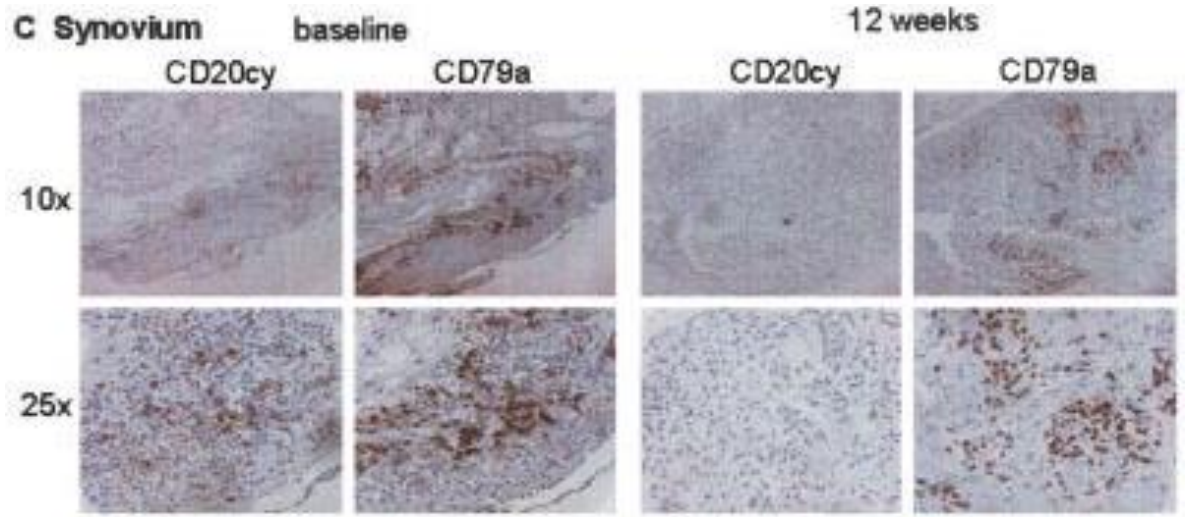
Rituximab has limited tissue penetrance

RA patients treated with rituximab have incomplete tissue B cell depletion

B cells mostly depleted in peripheral blood post-rituximab Tx



Limited synovial B cell depletion post-rituximab Tx



1. Onno Teng, YK et al. (2007), Immunohistochemical Analysis as a Means to Predict Responsiveness to Rituximab Treatment *Arthritis & Rheumatism*.

CD19 CAR-T therapy for autoimmune disease

In the past several years, many papers have shown CD19 CAR-T efficacy in autoimmune disease

CORRESPONDENCE

CD19-Targeted CAR T Cells in Refractory Systemic Lupus Erythematosus

CD19-targeted CAR T cells in refractory antisynthetase syndrome

Anti-CD19 CAR T cells for refractory myasthenia gravis

Molecular Therapy
Original Article

Case study of CD19 CAR T therapy in a subject with immune-mediate necrotizing myopathy treated in the RESET-Myositis phase I/II trial

Safety and clinical activity of autologous RNA chimeric antigen receptor T-cell therapy in myasthenia gravis (MG-001): a prospective, multicentre, open-label, non-randomised phase 1b/2a study

Letter to the Editor (Case report)

Rescue therapy of antisynthetase syndrome with CD19-targeted CAR-T cells after failure of several B-cell depleting antibodies

nature medicine — ARTICLES
https://doi.org/10.1038/s41591-022-02017-5
Check for updates

Anti-CD19 CAR T cell therapy for refractory systemic lupus erythematosus

The NEW ENGLAND JOURNAL of MEDICINE
ESTABLISHED IN 1812 FEBRUARY 22, 2024 VOL. 390 NO. 8

CD19 CAR T-Cell Therapy in Autoimmune Disease — A Case Series with Follow-up

Fabian Müller, M.D., Jule Taubmann, M.D., Laura Bucci, M.D., Artur Wilhelm, Ph.D., Christina Bergmann, M.D., Simon Völkl, Ph.D., Michael Aigner, Ph.D., Tobias Rothe, Ph.D., Ioanna Minopoulou, M.D., Carlo Tur, M.D., Johannes Knitza, M.D., Soraya Kharboutli, M.D., Sascha Kretschmann, Ph.D., Ingrid Vasova, M.D., Silvia Spoerl, M.D., Hannah Reimann, Ph.D., Luis Munoz, M.D., Roman G. Gerlach, Ph.D., Simon Schäfer, Ph.D., Ricardo Grieshaber-Bouyer, M.D., Anne-Sophie Korganow, M.D., Dominique Farge-Bancel, M.D., Dimitrios Mougiakakos, M.D., Aline Bozec, Ph.D., Thomas Winkler, Ph.D., Gerhard Krönke, M.D., Andreas Mackensen, M.D., and Georg Schett, M.D.

CLINICAL SCIENCE

CD19-CAR T-cell therapy induces deep tissue depletion of B cells

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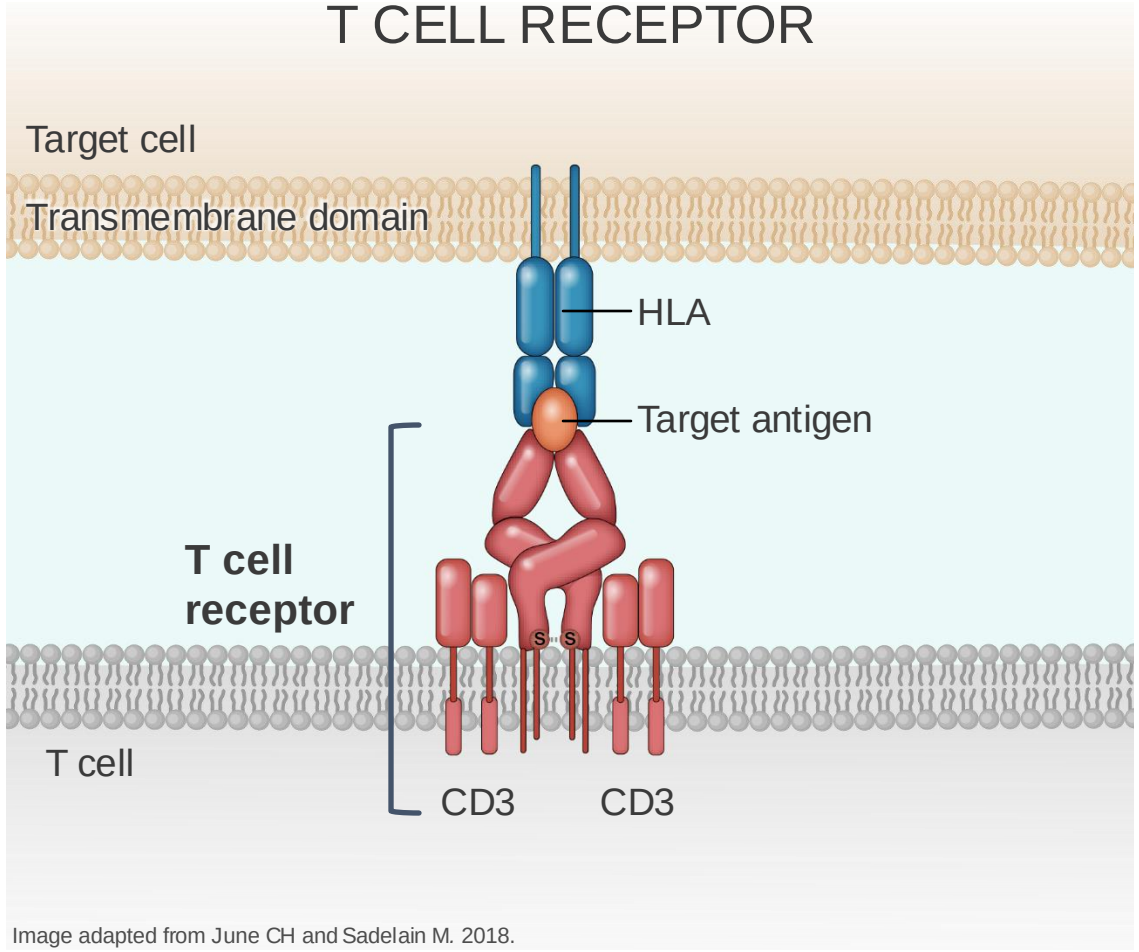
What Are Chimeric Antigen Receptor (CAR) T Cells?

Engineered T cells that combine the targeting ability of antibodies with the cell-killing machinery of T cells



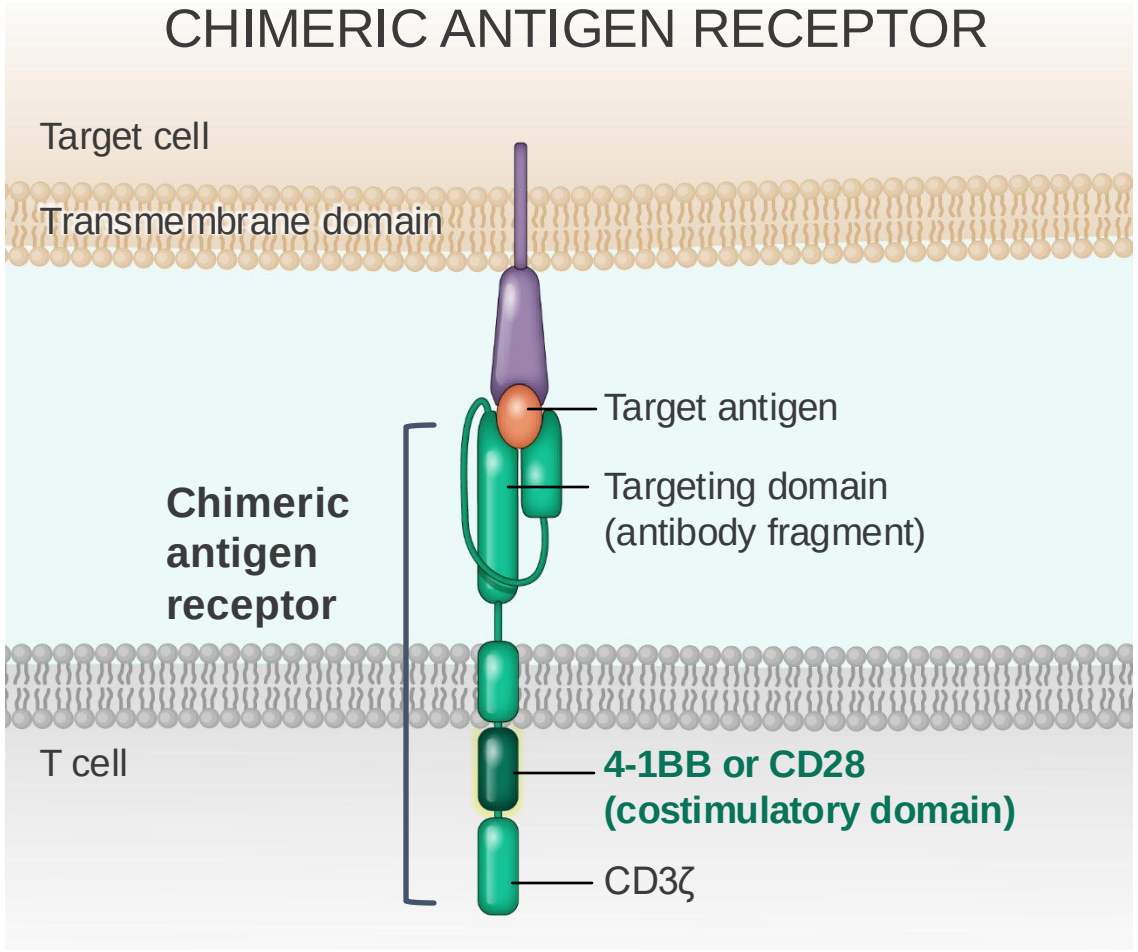


Image adapted from June CH and Sadelain M. 2018.

CD, cluster of differentiation; HLA, human leukocyte antigen.

June CH, Sadelain M. *N Engl J Med.* 2018;379;64-73.

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Personalized Manufacturing of CAR T Cells

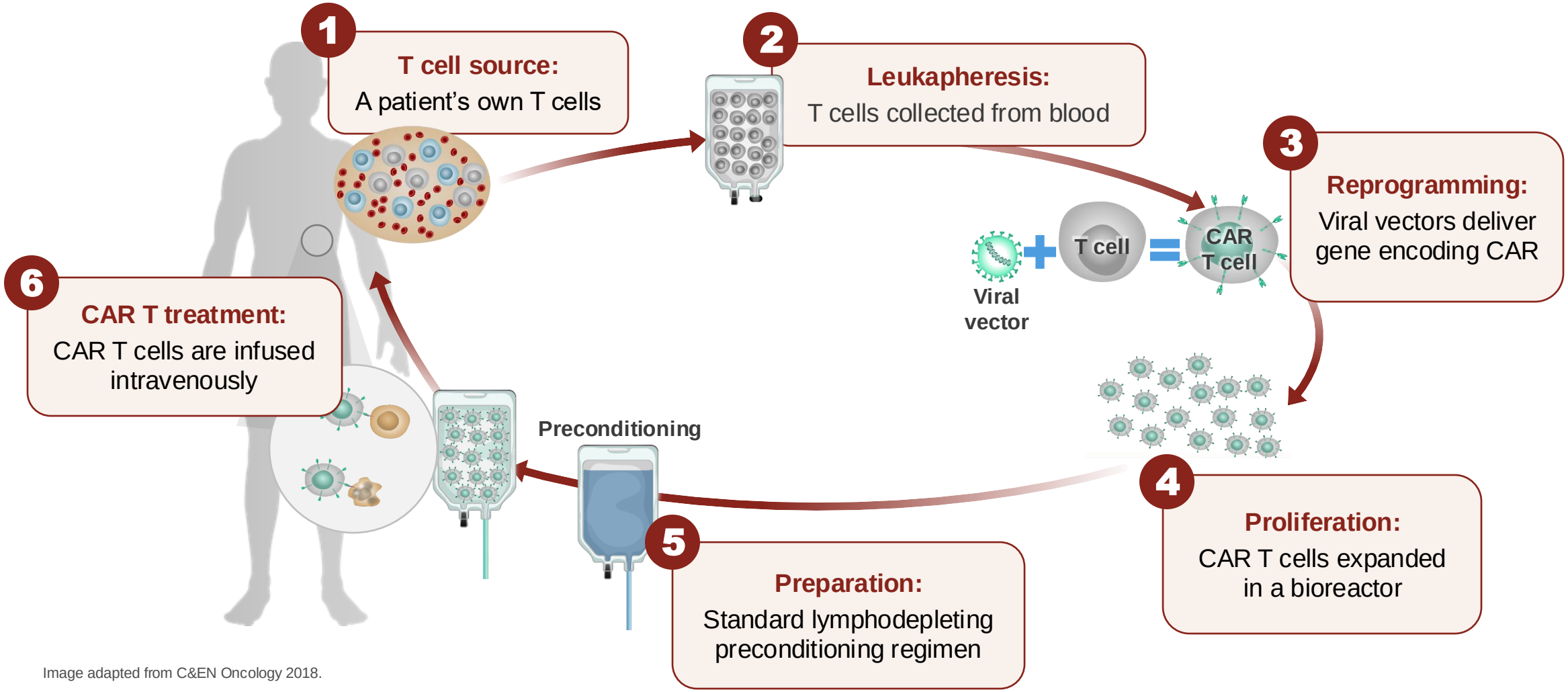


Image adapted from C&EN Oncology 2018.

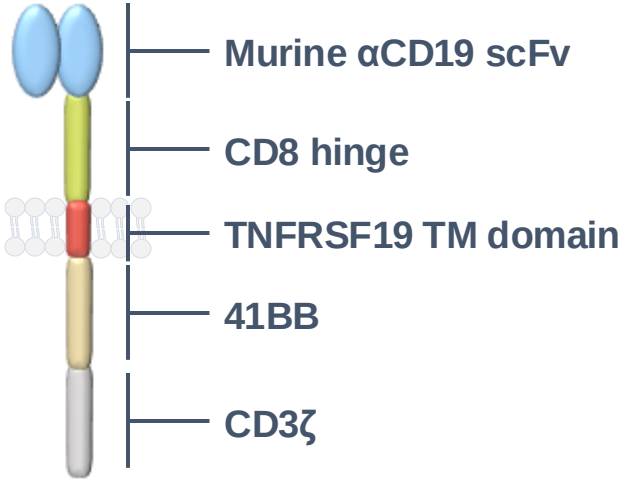
C&EN Oncology. Accessed June 10, 2024. https://cen.acs.org/pharmaceuticals/oncology/Controlling-CAR-T-scientists-plan/96/i19.

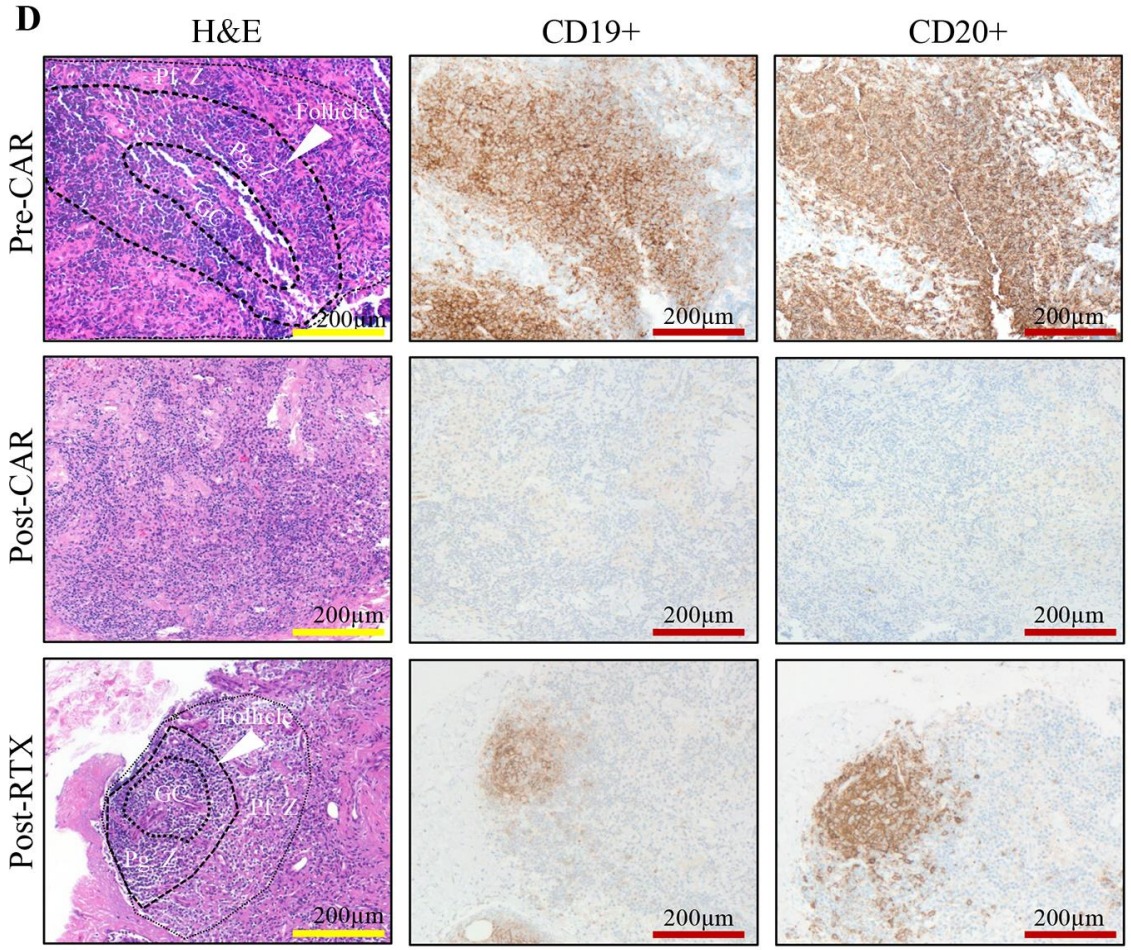
CD19 CAR-T cells are capable of deep B-cell depletion

CD19 CAR-T cells have far better penetrance into lymph nodes as compared to rituximab

nature medicine ARTICLES
Anti-CD19 CAR T cell therapy for refractory systemic lupus erythematosus

FAU FRIEDRICH-ALEXANDER UNIVERSITÄT ERLANGEN-NÜRNBERG





1. Maschan et. al (2021), Multiple site place-of-care manufactured anti-CD19 CAR-T cells induce high remission rates in B-cell malignancy patients, *Nature Communications*
2. Tur et. al (2024), CD19-CAR T-cell therapy induces deep tissue depletion of B cells, *Annals of the Rheumatic Diseases*

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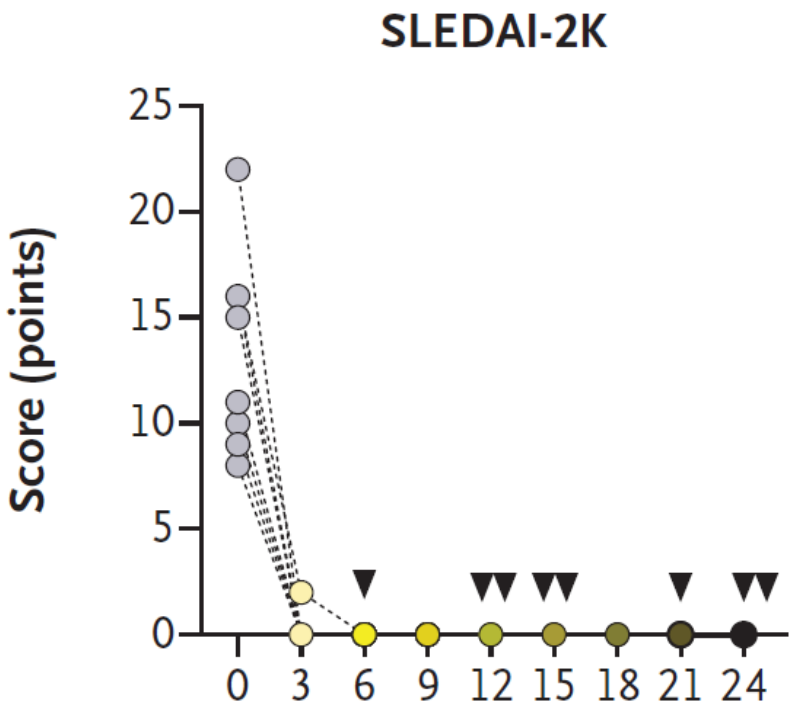
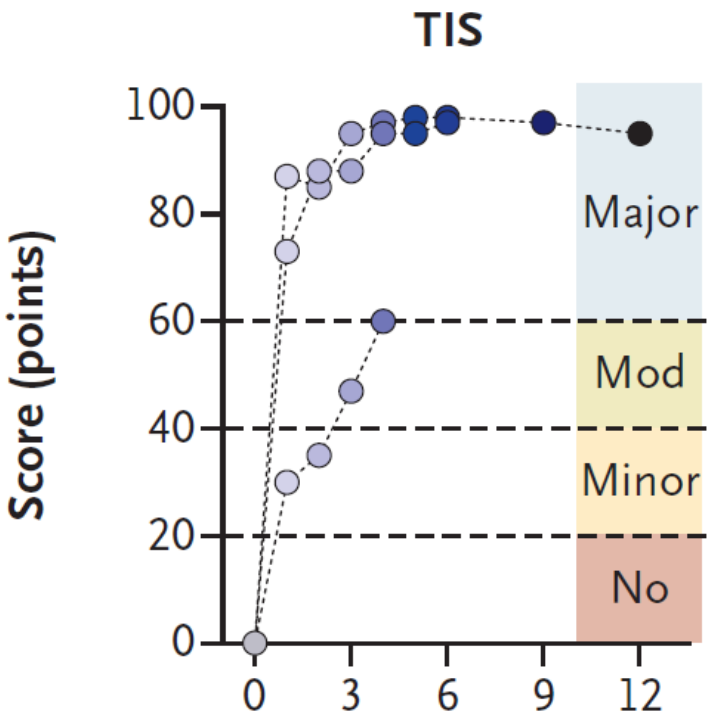
Responses observed by 3 months across multiple autoimmune diseases

Long-term remissions observed across multiple different autoimmune diseases

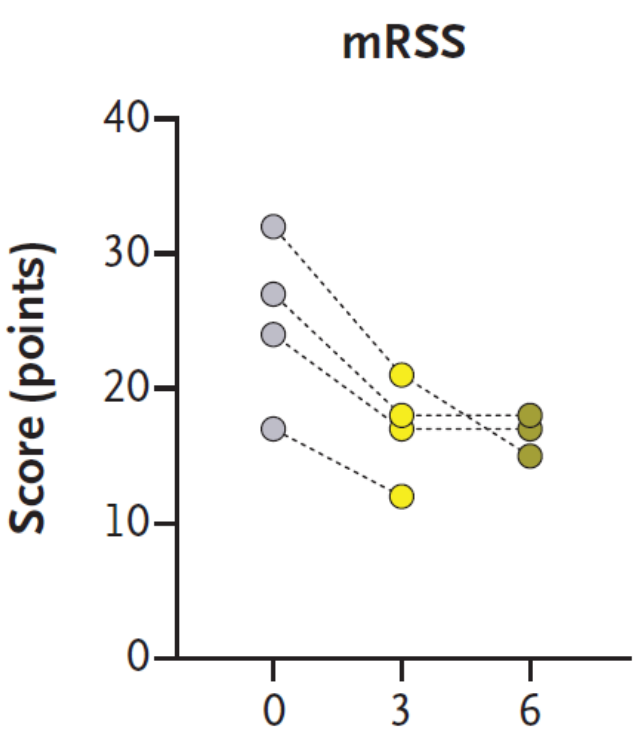
SLE



Myositis



SSc



Months

1. Müller, et. al (2024) CD19 CAR T-cell Therapy in Autoimmune Disease – A Case Series with Follow-up

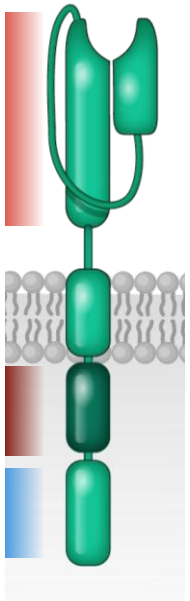
CABA-201: CD19-CAR T specifically designed for autoimmunity

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
Similar binding affinity & biologic activity to FMC63, with binding to the same epitopes[1,2]

4-1BB costimulatory domain
Same co-stim. domain as used in academic studies

CD3-zeta signaling domain



CABA-201[5]

Clinical data reported by IASO using licensed CD19 binder in oncology[4]

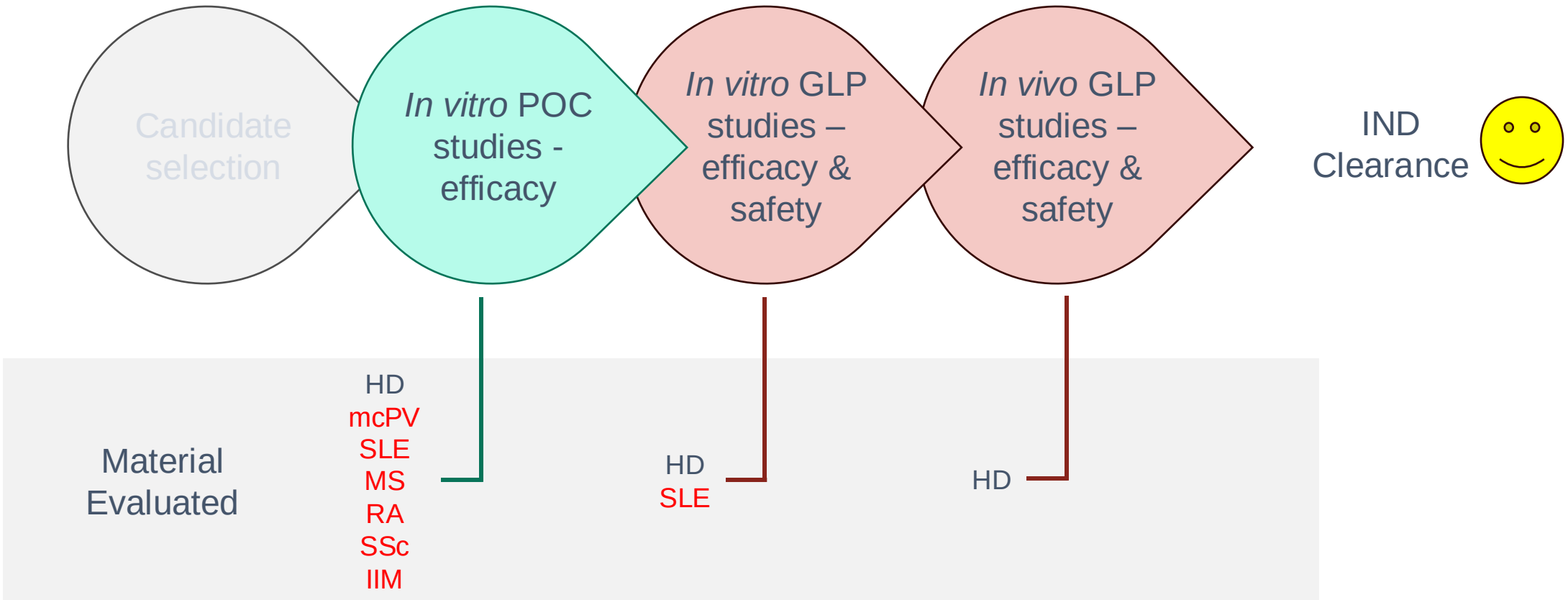
- **Fully human binder**
  Evaluated as dual-CAR combined with CD22 binder with standard Flu/Cy preconditioning
- **Data reported in ~20 patients to date**
  B cell leukemia and lymphoma in IIT in China
- **Safety data supports autoimmune development**

IIT – Investigator-initiated trial; Flu/Cy – Fludarabine/Cyclophosphamide

1. Peng, et al. Molecular Therapy Methods and Clinical Development 2024
2. Dai, Zhenyu, et al. Journal of Cellular Physiology 2021
3. Müller, Fabian, et al. New England Journal of Medicine 2024
4. Evaluated as part of CT120, a dual-CD19xCD22 CAR T product candidate under development by Nanjing IASO Biotherapeutics, Co., Ltd. (IASO Bio).
5. 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.

Preclinical Development of CABA-201

Schematic overview

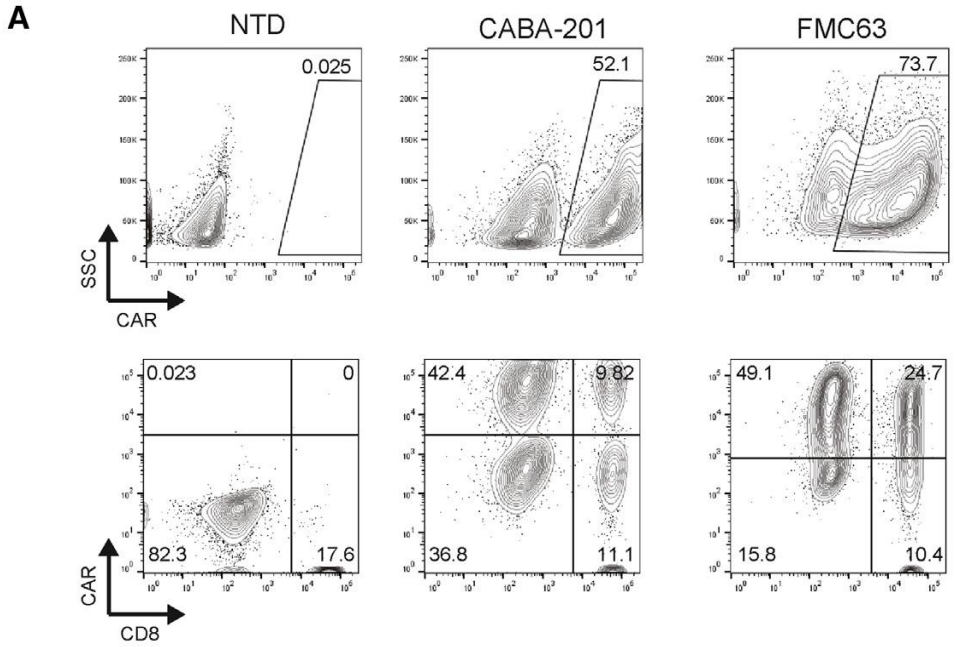


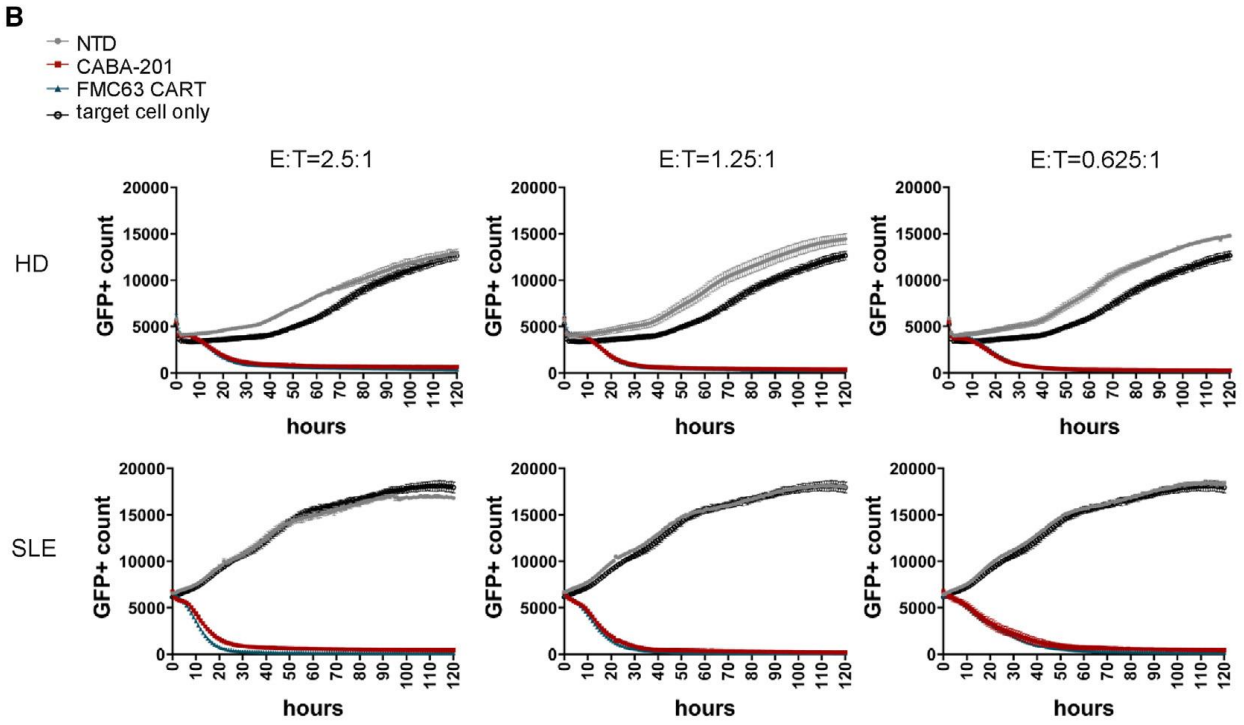
HD – Healthy donor, mcPV – Mucocutaneous pemphigus vulgaris, SLE – Systemic lupus erythematosus; IIM – myositis; SSc – Systemic sclerosis; MS – Multiple sclerosis, RA – Rheumatoid arthritis
RED color denotes exclusive use of female autoimmune disease donors

SLE or HD derived CABA-201 demonstrate on-target cytolysis

In vitro GLP studies – efficacy & safety

CABA-201 expresses in SLE or HD derived T-cells and can lyse CD19^{+} NALM6 cells



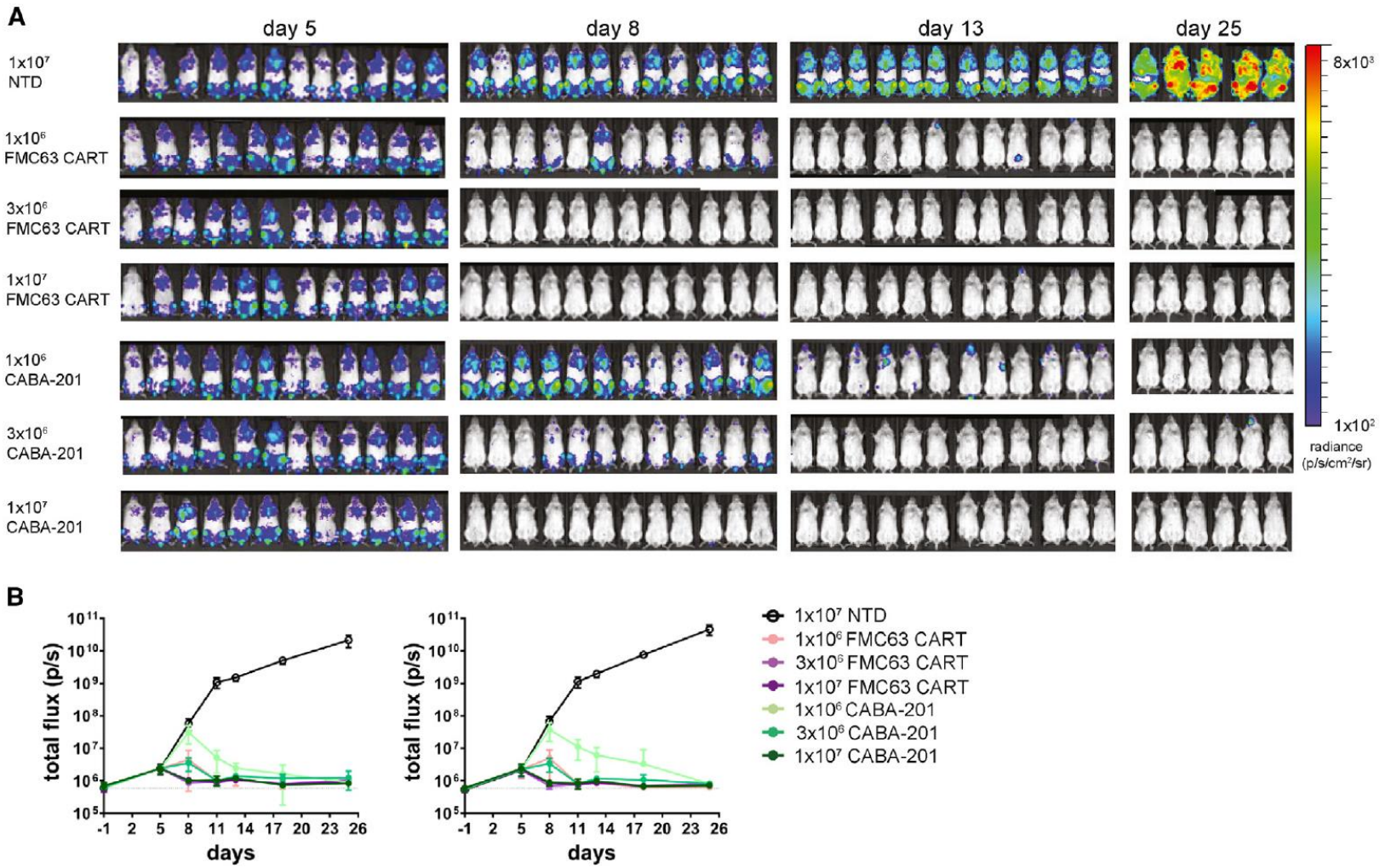


1. SLE – Systemic lupus erythematosus; HD – Healthy donor
2. Peng et al, Molecular Therapy Methods and Clinical Development 2024

HD derived CABA-201 demonstrate cytolytic activity *in vivo*

In vivo GLP studies – efficacy & safety

CABA-201 exhibits comparable cytolytic activity to FMC63 CAR-T cells in NSG/NALM6 model

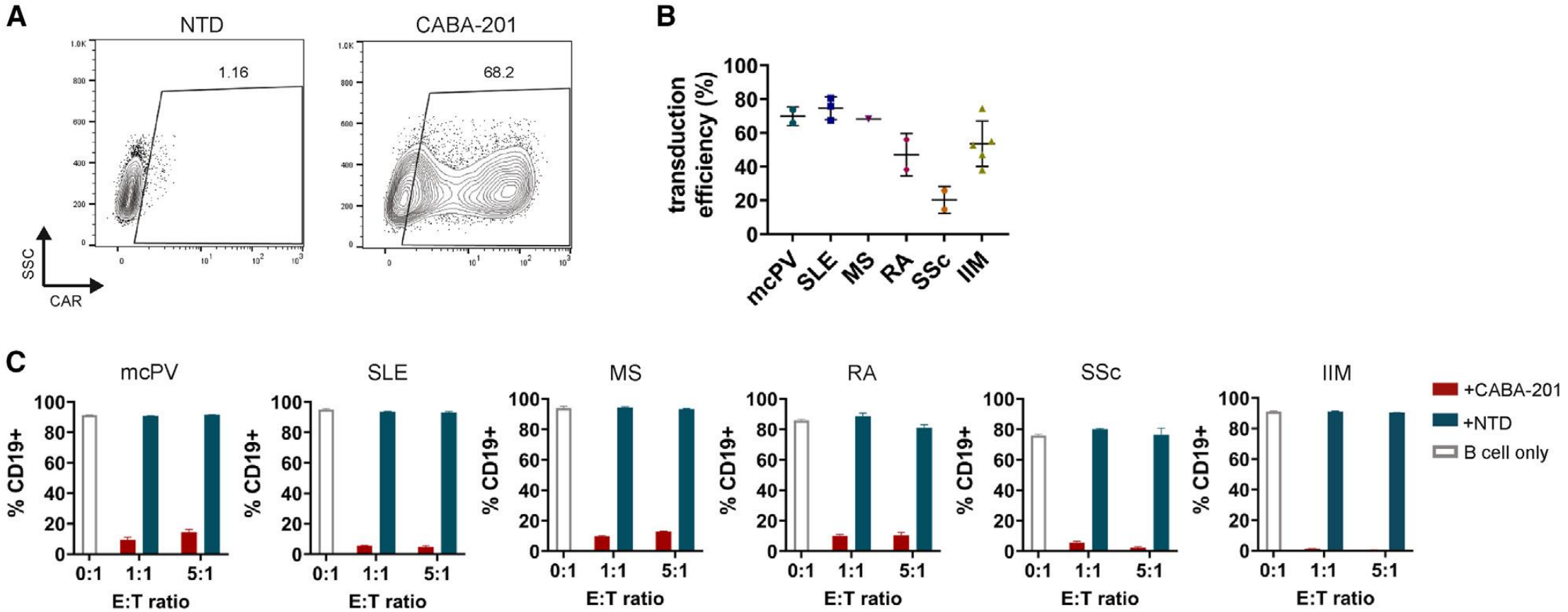


1. HD – Healthy donor
2. Peng et al, Molecular Therapy Methods and Clinical Development 2024

In vitro POC studies - efficacy

Patient or HD derived CABA-201 demonstrate on-target cytolysis

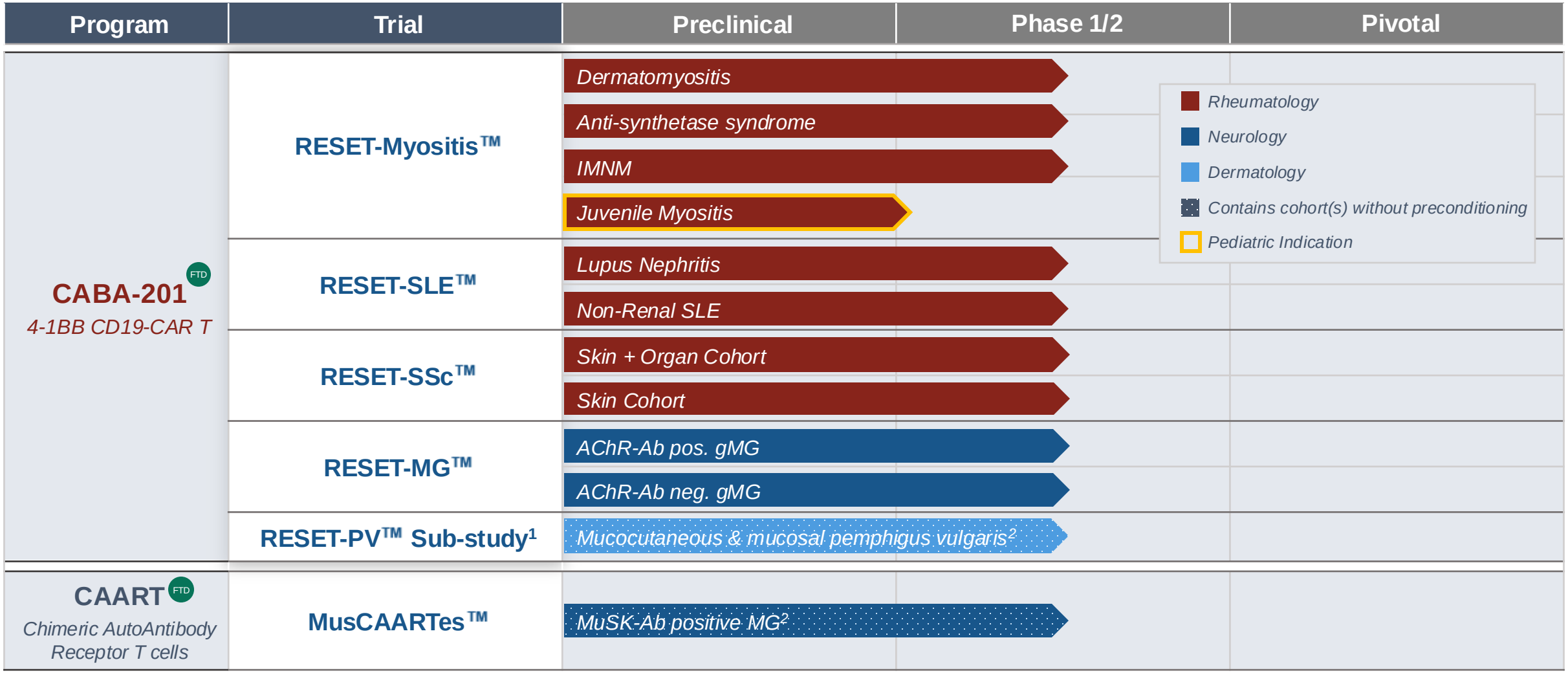
CABA-201 expresses in autoimmune patient derived T-cells and can lyse patient matched CD19^{+} B-cells



1. mcPV – Mucocutaneous pemphigus vulgaris, SLE – Systemic lupus erythematosus; IIM – myositis; SSc – Systemic sclerosis; MS – Multiple sclerosis, RA – Rheumatoid arthritis
2. Peng et al, Molecular Therapy Methods and Clinical Development 2024

Pipeline targeting autoimmune diseases with high unmet need

Innovative and scalable clinical strategy with potential for accelerated development path



RESET™ – **RE**storing **SE**lf-**T**olerance; IMNM – Immune-mediated necrotizing myopathy; SLE – Systemic lupus erythematosus; Ab – Antibody; AChR – Acetylcholine receptor; gMG – Generalized myasthenia gravis

1. Sub-study incorporated into DesCAARTes™ study. 2. Currently being evaluated in a Phase 1 trial.

● FDA Fast Track Designation received in dermatomyositis, SLE and lupus nephritis, systemic sclerosis, mucosal pemphigus vulgaris, and MuSK-Ab positive MG.

Cabaletta Bio®

Summary

CD19 CAR T-cells for autoimmune disease

- Autoimmune incidence is increasing & women are disproportionately impacted
  - Incomplete inactivation of X-chromosome is a likely hypothesis
- B cells are a major driver of autoimmune disease
  - Antibody secreting function
  - As an antigen presenting cells: B-cells secrete pro-inflammatory cytokines
- CD19 CAR T-cells have been observed to eliminate all B cells in autoimmune disease patients
  - Superior penetrance as compared to standard biologics approaches
  - Durable complete responses observed up to three years so far
  - Favorable safety profile observed for 41BBz CAR T-cells
- Preclinical IND enabling studies for CABA-201 were designed to take gender into account
  - Use of female T-cells for CABA-201 generation and *in vitro* experiments
  - Use of female NSG mice for *in vivo* experiments

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