Cabaletta Bio®

Corporate Presentation

MAY 2022

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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 and clinical trials of DSG3-CAART and MuSK-CAART, 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 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.

Develop and launch the first curative targeted cellular therapies for patients with autoimmune diseases

Cabaletta Bio®

Cabaletta® overview

Developing highly specific CAAR T products to treat B cell-mediated autoimmune diseases

DesCAARTes™ trial in patients with mucosal pemphigus vulgaris (mPV) ongoing

- Favorable safety profile for DSG3-CAART demonstrated in cohorts A1 through A4 at doses up to 2.5 billion cells
  - No CRS, ICANS, dose-limiting toxicities or related SAEs observed in any patient in cohorts A1 to A4
- Dose-dependent increase in DSG3-CAART persistence observed in cohorts A1 to A4, as presented at 25th Annual ASGCT Conference
  - Cohort A4 persistence approached the lower end of the range seen with anti-CD19 CART with lymphodepletion in oncology[1,2,3]
- Cohort A5 dosing up to 7.5 billion cells ongoing with multiple additional cohorts possible to enhance in vivo DSG3-CAART exposure

MusCAARTes™ trial in patients with MuSK myasthenia gravis on track to initiate in 2022

Cell therapy pipeline[4] targeting diseases that affect over 80,000 U.S. patients

Cash runway through 3Q23 with $109.2M in cash and investments at the end of 1Q22

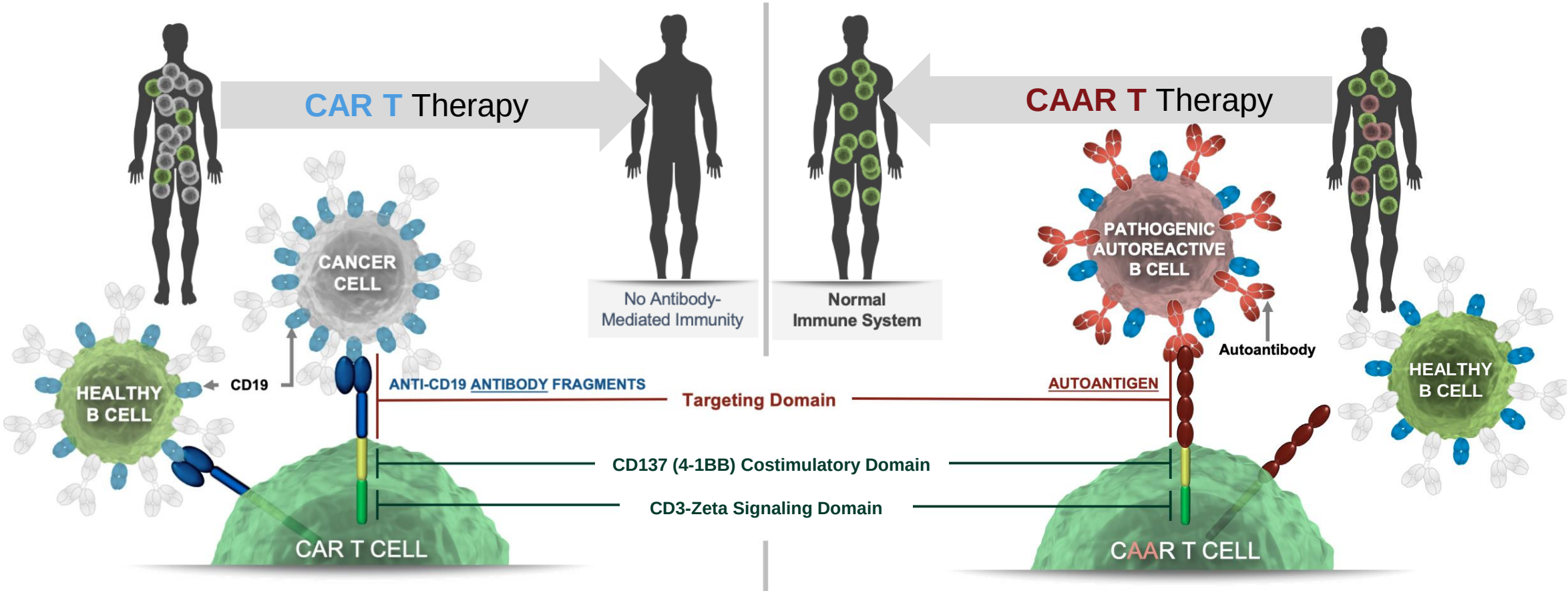
CRS – Cytokine release syndrome; ICANS – Immune effector cell-associated neurotoxicity syndrome; SAE – Serious adverse event

1. Mueller, Karen Thudium, et al. "Cellular kinetics of CTL019 in relapsed/refractory B-cell acute lymphoblastic leukemia and chronic lymphocytic leukemia." Blood, The Journal of the American Society of Hematology 130.21 (2017): 2317-2325.
2. Schuster SJ, Bishop MR, Tam CS, et al. Tisagenlecleucel in Adult Relapsed or Refractory Diffuse Large B-Cell Lymphoma. N Engl J Med. 2019;380(1):45-56. doi:10.1056/NEJMoa1804980

3. The range of persistence observed with anti-CD19 CART therapy in oncology has not been confirmed to be necessary or sufficient for clinical responses in patients with mPV.
4. Includes five disclosed product candidates and two undisclosed pipeline disease targets in our pipeline.

Our scientific platform leverages clinically validated CAR T technology

CAAR T product candidates are designed for selective and specific elimination of the pathogenic B cells



Chimeric **A**ntigen **R**eceptor **T** cell

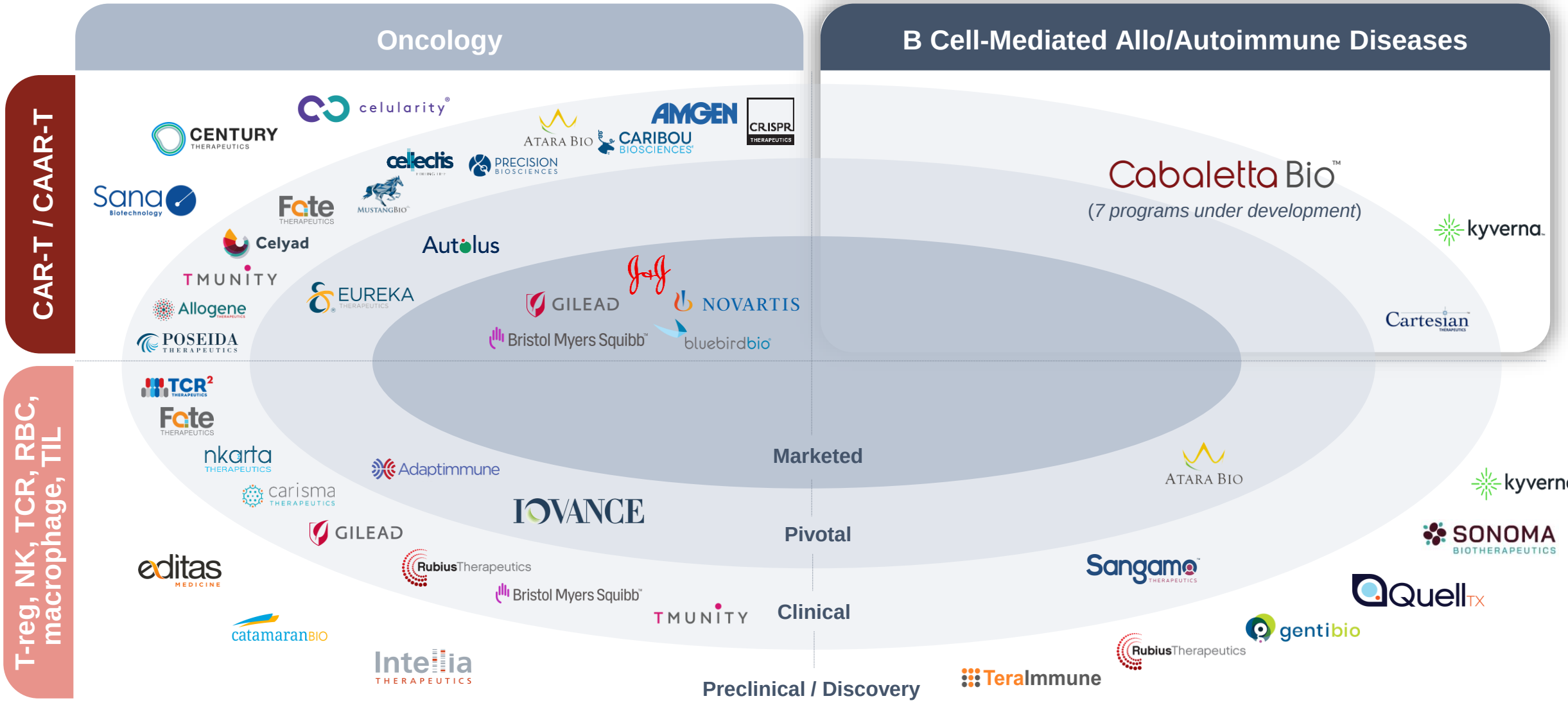
Kymriah

Chimeric **A**uto**A**ntibody **R**eceptor **T** cell

CABA CAAR T

Cabaletta: Advancing targeted cell therapy to autoimmunity

Foundational CAR T technology clinically validated in treating B cell-mediated cancers

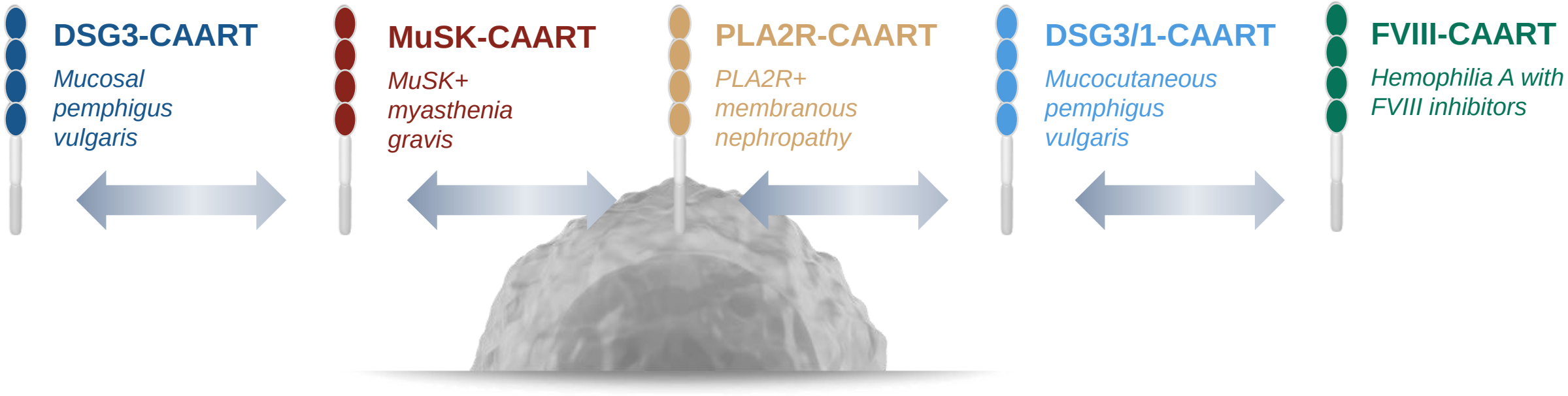


***Note**: Landscape for illustrative purposes only.*

Modular platform with “plug-and-play” architecture

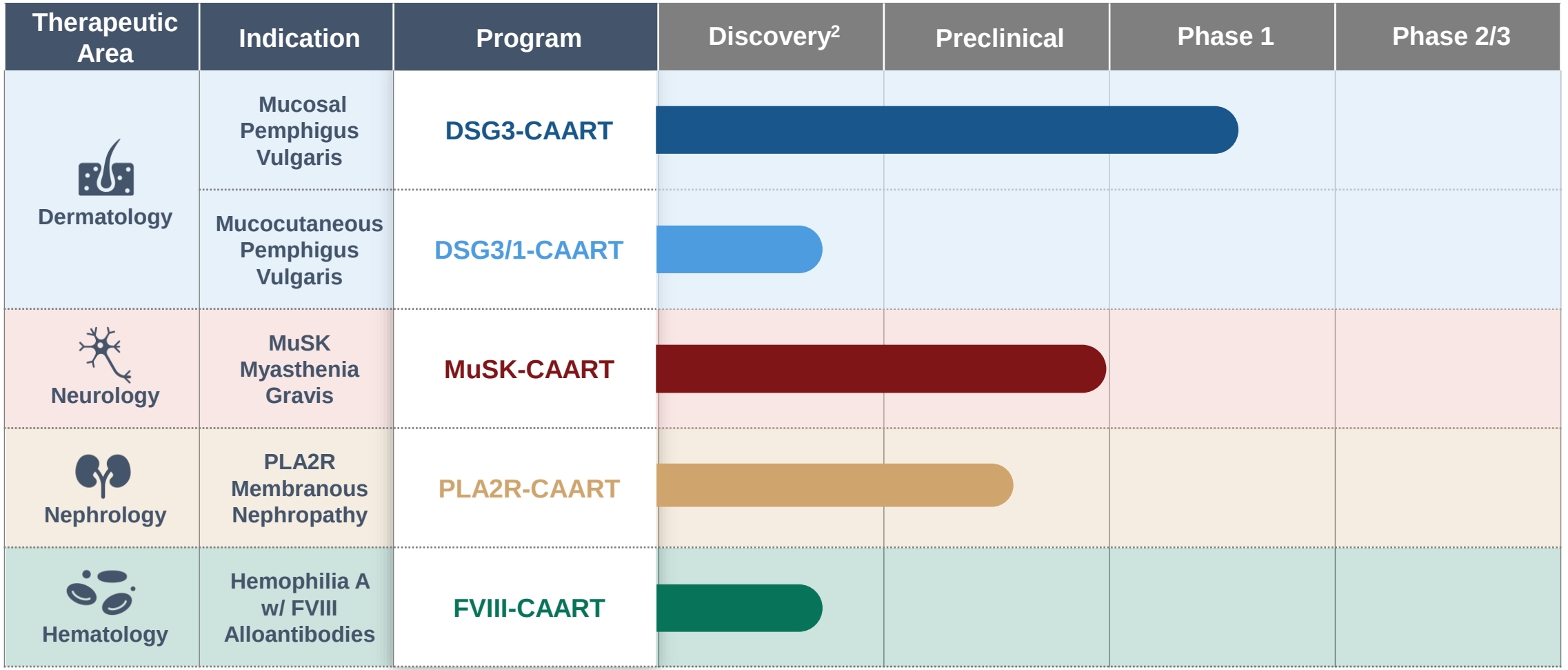
CABA™ platform designed to enable a portfolio of programs targeting B cell-mediated diseases

Swapping the extracellular domain, or autoantigen, creates **new product candidates**



Clinically validated engineered T cell platform is the foundational technology

Pipeline[1] includes multiple disease targets where cure is possible



Current pipeline includes 7 programs targeting diseases affecting >80,000 patients in the U.S.

1. Two additional undisclosed disease targets currently in discovery stage are not shown in our pipeline portfolio.
2. In our discovery stage, we perform epitope mapping and optimize CAAR construct and design.

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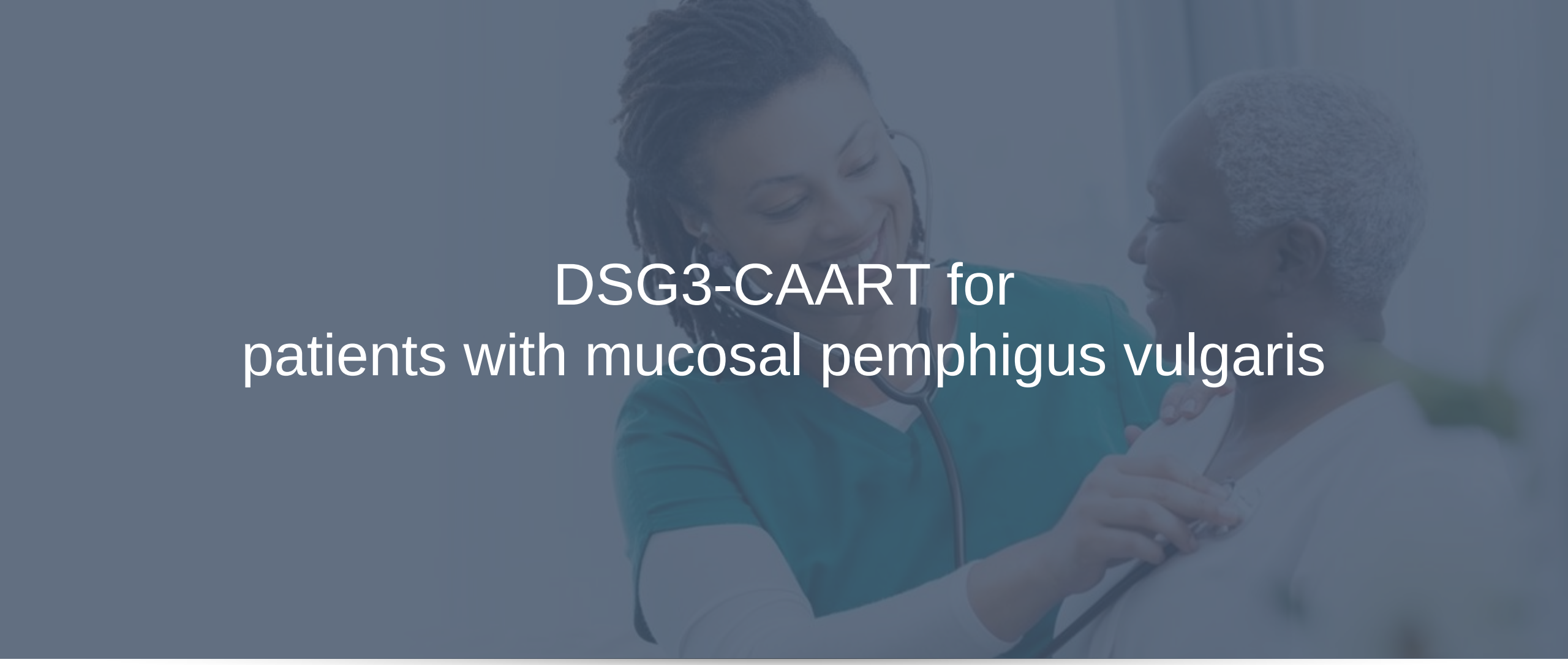

DSG3-CAART for patients with mucosal pemphigus vulgaris

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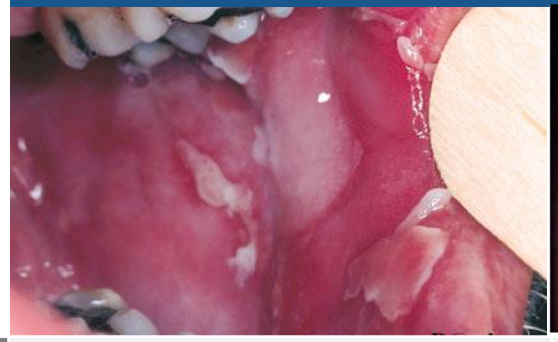
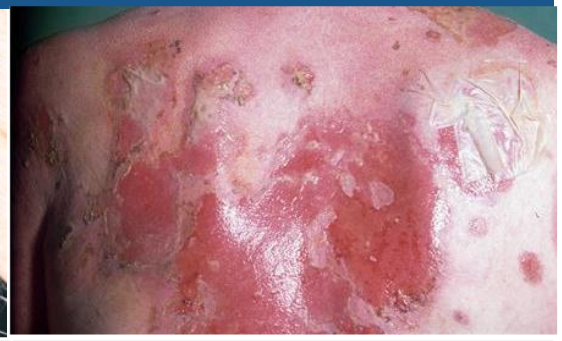
Overview of pemphigus vulgaris

Current treatments require broad immunosuppression associated with safety risks and transient efficacy

	Mucosal PV[1] 25% of U.S. pemphigus vulgaris	Mucocutaneous PV[2] 75% of U.S. pemphigus vulgaris
		
Associated Antibody	**Anti-DSG3**	**Anti-DSG3 + Anti-DSG1**
Clinical Signs	Painful blisters of the mucous membranes (mouth, nose, larynx, esophagus, eyes, genitalia, rectum)	Blisters on orifices and skin
US Disease Prevalence	**3,250 to 4,750**	**9,750 to 14,250**

Current Treatment Landscape

Broad immunosuppression[3,6]

- Modestly effective
- Poorly tolerated

Rituximab plus steroids (~3,500 mg/yr)[4]

- Response: ~40% of patients achieved a 16-week period with no lesions or medicines during 1 yr[4,5]
- 22% annual serious adverse event (SAE) rate[4]
  - 4-9%[3,4,5] annual risk of severe infection in PV
- Real world data indicate:
  - *Transient* remission ~ 70% CROT[6]:
    - ~30% relapse in 1 year[6]
    - >50% relapse within 2 years[6]
  - ~30% never achieve CROT[6]
  - ~1.9% lifetime risk of fatal infection[7]

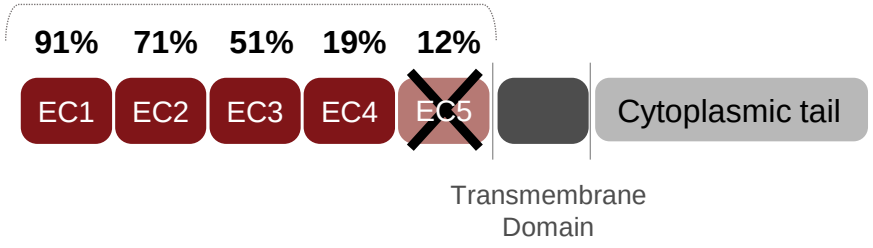
CROT = 8+ weeks without lesions while off systemic therapy

1. Image credit: D@nderm.
2. http://www.vgrd.org/archive/cases/2004/pv/DSCN4996%20copy.JPG
3. Joly, Pascal, et al. "First-line rituximab combined with short-term prednisone versus prednisone alone for the treatment of pemphigus (Ritux 3): a prospective, multicentre, parallel-group, open-label randomised trial." The Lancet 389.10083 (2017): 2031-2040.
4. Werth, Victoria P., et al. "Rituximab versus Mycophenolate Mofetil in Patients with Pemphigus Vulgaris." New England Journal of Medicine (2021).
5. Rituximab label, 08/2020 revision.
6. Kushner, Carolyn J., et al. "Factors Associated With Complete Remission After Rituximab Therapy for Pemphigus." JAMA dermatology (2019).
7. Tony, Hans-Peter, et al. "Safety and clinical outcomes of rituximab therapy in patients with different autoimmune diseases: experience from a national registry (GRAID)." Arthritis research & therapy 13.3 (2011): 1-14.

DSG3-CAART is designed to bind all known pathogenic autoantibodies

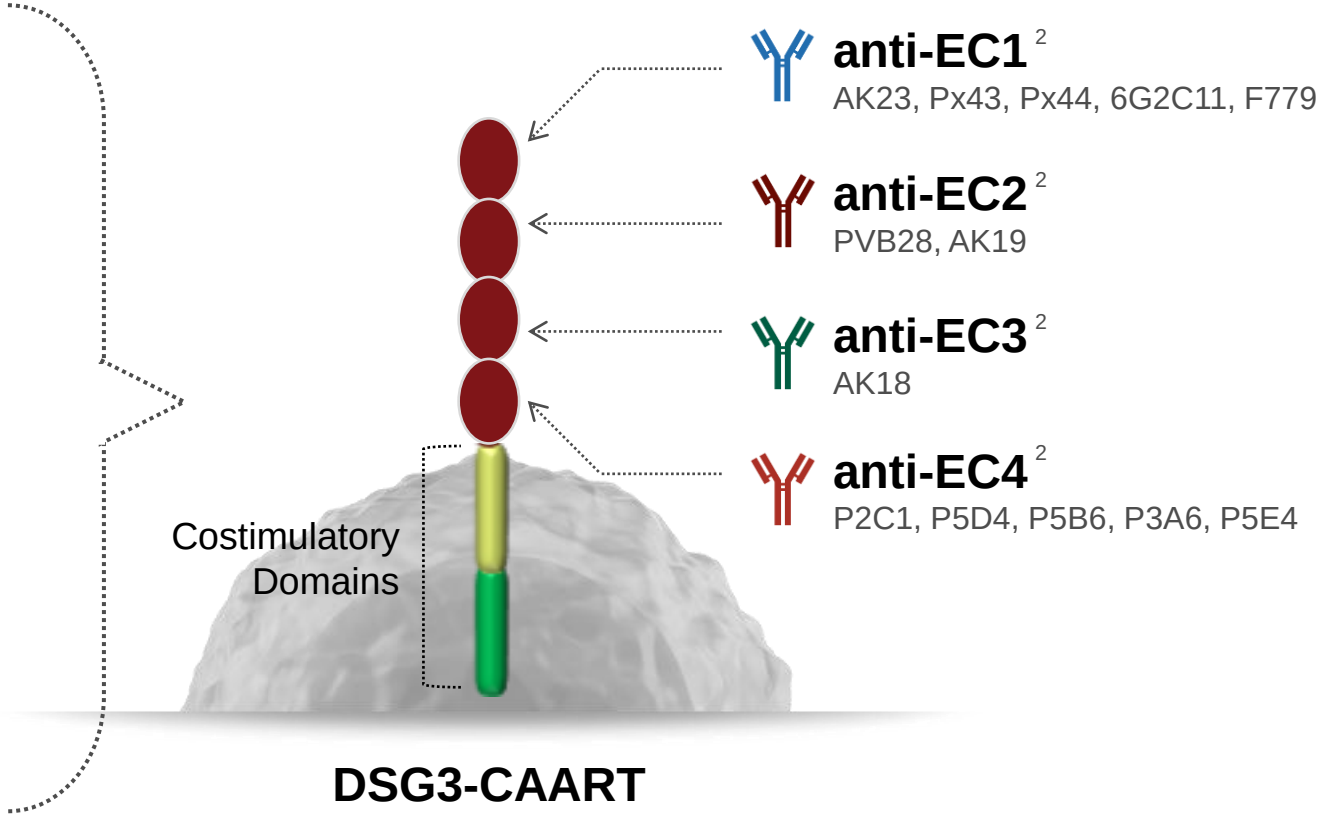
Inclusion of all disease-relevant epitopes enables a ‘one-size-fits-all’ approach for patients with mPV

% of PV sera targeting each domain[1]



DSG3 Protein

EC5 directed antibodies are not known to be pathogenic[3]



DSG3-CAART

1. Ohyama, Bungo, et al. "Epitope spreading is rarely found in pemphigus vulgaris by large-scale longitudinal study using desmoglein 2–based swapped molecules." Journal of investigative dermatology 132.4 (2012): 1158-1168.
2. Antibodies that target the specific extracellular domain are shown below each extracellular domain.
3. Amagai, Masayuki, et al. "Autoantibodies against the amino-terminal cadherin-like binding domain of pemphigus vulgaris antigen are pathogenic." The Journal of clinical investigation 90.3 (1992): 919-926.

DesCAARTes™ Phase 1 study of DSG3-CAART[1]

Orphan Drug Designation

Fast Track Designation

Trial in patients with mPV evaluating up to 750x dose range (20M up to 10-15B cells)

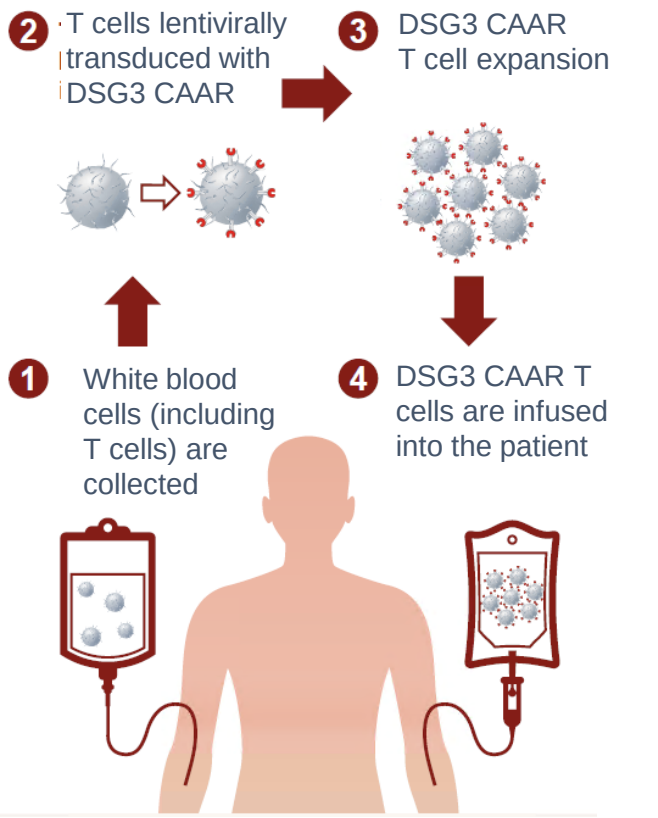
Screening → Apheresis → **DSG3-CAART INFUSION** → TREATMENT & FOLLOW-UP PERIOD → Next Patient

Major Inclusion Criteria

- Age: ≥18, confirmed diagnosis
- Inadequately managed by standard immunosuppressive therapies
- Confirmed diagnosis
- Active disease
- Anti-DSG3 antibody positive

Major Exclusion Criteria

- Recent rituximab
- Prednisone > 0.25 mg/kg/day
- Other autoimmune disorder requiring immunosuppressive therapies
- Recent investigational treatment
- ALC < 1,000 at screening



Adjunctive immunosuppressants are stopped; prednisone tapered to low dose prior to infusion

Part A Cohorts[2,3]	Subjects	Dose*
A1	3 (+3)	20M
A2	3 (+3)	100M
A3	3 (+3)	500M
A4	3 (+3)	2.5B
A5	3 (+3)	5.0B to 7.5B
A5e[4]	*3 (+3)*	*5.0B to 7.5B*
A6m[5]	*3 (+3)*	*10B to 15B*

Up to 750-fold dose escalation

Primary objective:
Determine the maximum tolerated dose of DSG3-CAART

Primary endpoint:
Adverse events, including dose-limiting toxicities (DLTs), related to DSG3-CAART within 3 months of infusion

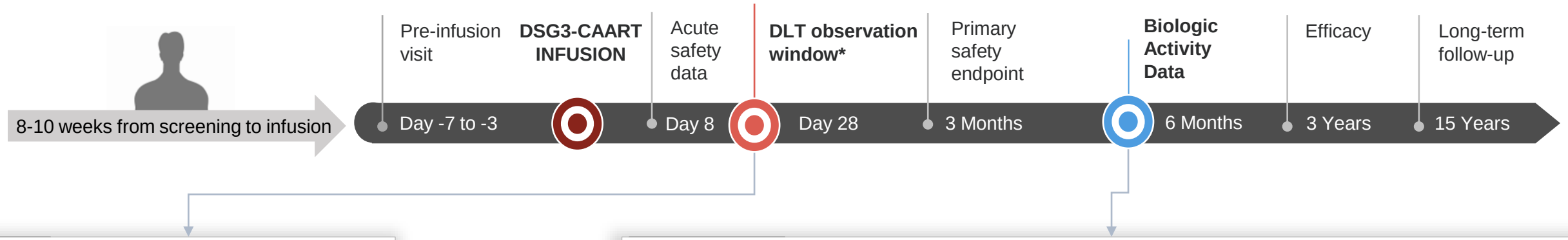
Secondary objectives:
- Manufacturing success rate
- DSG3-CAART persistence
- Anti-DSG3 antibody titer changes
- Concomitant medication changes
- Clinical disease activity score changes

1. Phase 1 Trial (NCT04422912).
2. FDA has requested, and the Company has agreed, that we will share data from part A to inform a discussion on the optimal design of part C. According to FDA advice, the submission of part A data is not gating to planned enrollment in part B.
3. Cohort progression following A5 (e.g. A5e or A6m) is to be prioritized according to emerging data and discussions with the FDA, as applicable.
4. Cohort A5e reflects an enhanced manufacturing process designed to amplify the already present cell subtypes in the product in order to potentially improve product potency and trafficking.
5. Cohort A6m reflects a multi-dose regimen where patients will receive a total of 10 to 15 billion cells.

* *20M, 100M, 500M, 2.5B, 5.0B to 7.5B and 10B to 15B refers to the number of DSG3-CAART cells infused for patients in dosing cohorts A1 to A (M – millions; B – billions).*

DesCAARTes™ clinical trial assessments & current timeframes

Safety assessed acutely, at 28 days & at 3 months, with data on biologic activity within 6 months



Day 28 safety data

Infusion of DSG3-CAART in context of circulating soluble DSG3 antibody

Other safety measures

- CRS / neurotoxicity
- Other adverse events

Primary safety endpoint at 3 months

Potential for disease flare during planned steroid taper +/- discontinuation of immuno-suppressive medications for mPV

Early evaluations of efficacy include[1]:

- Persistence of DSG3-CAART detected via qPCR
- DSG3 antibody titer changes (targeting sustained reduction)
- Change in mPV therapy and/or new systemic rescue therapy
- Disease activity changes based on clinically validated scales
  - e.g. PDAI, ODSS

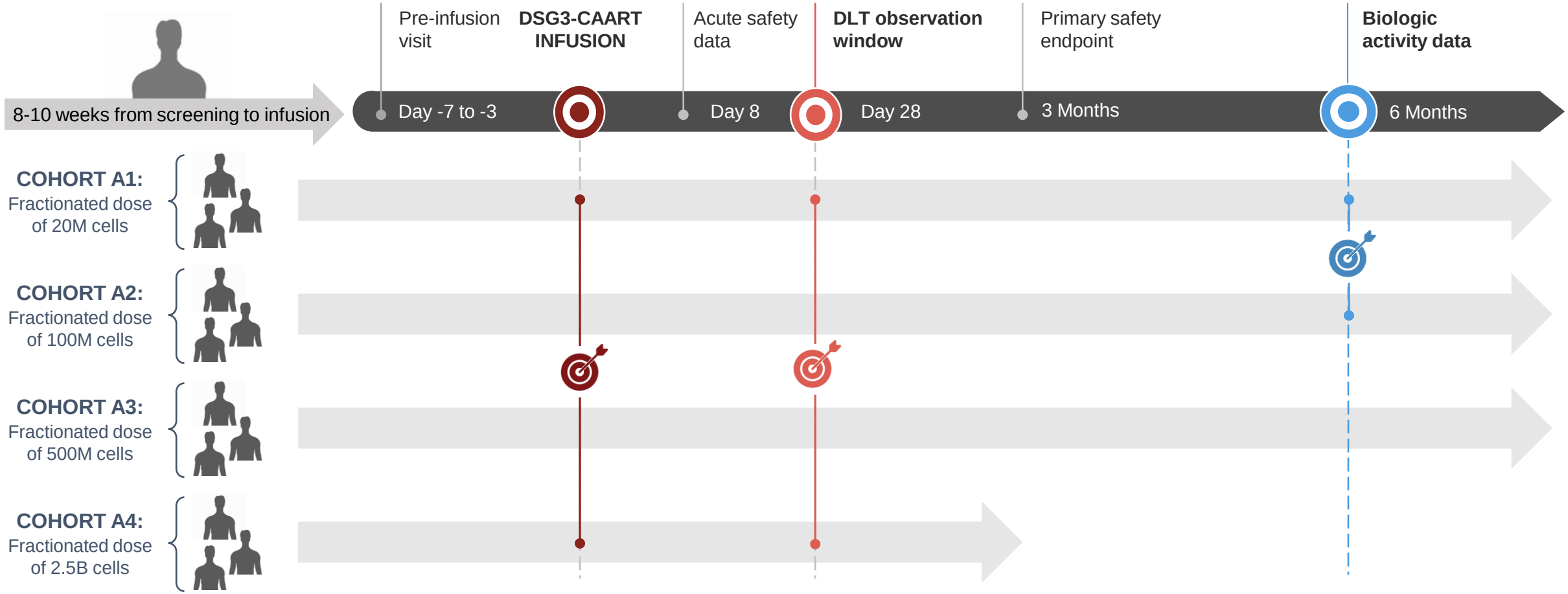
Data expected within 6 months of completion for each cohort

** Clearance of 28-day observation window without DLTs required to initiate next dosing cohort. DLTs include any grade 3 or 4 CRS or neurotoxicity, or any Grade 2 CRS or neurotoxicity that failed to improve to ≤ Grade 1 or baseline within 7 days*

1. This information represents data that we believe can be used to inform potential efficacy endpoints in future clinical development.
2. Spindler, Volker, et al. "Mechanisms causing loss of keratinocyte cohesion in pemphigus." Journal of Investigative Dermatology 138.1 (2018): 32-37.

No DLTs observed to date in first 4 cohorts of DesCAARTes™ trial

Favorable safety profile at all reported doses of DSG3-CAART, including the 2.5B cell dose cohort



Cohort A5 (two-split fractionated dose of 5.0-7.5B cells) progressing; additional clinical data for cohort A4 expected to be provided at a scientific meeting in mid-2022

** 20M, 100M, 500M, 2.5B, 5.0B to 7.5B and 10B to 15B refers to the number of DSG3-CAART cells infused for patients in dosing cohorts A1 to A6m (M – millions; B – billions).*

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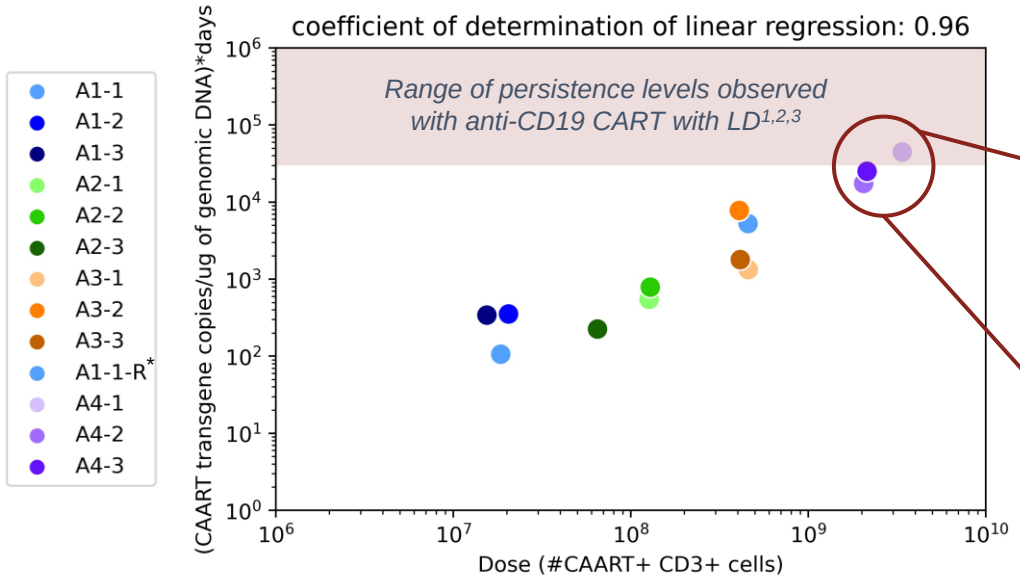
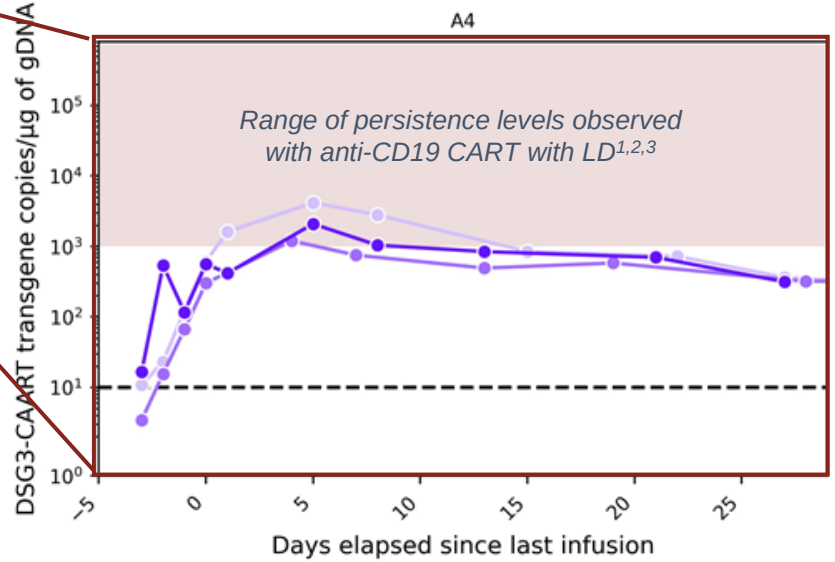
Dose-dependent persistence of DSG3-CAART in cohorts A1 to A4

DSG3-CAART persistence in cohort A4 approached levels seen in CART-19 with lymphodepletion in oncology



Persistence ‘area under the curve’ throughout 29 days post infusion was dose dependent

Persistence in cohort A4 approached the lower end of the range observed with anti-CD19 CART with lymphodepletion in oncology[1,2,3]



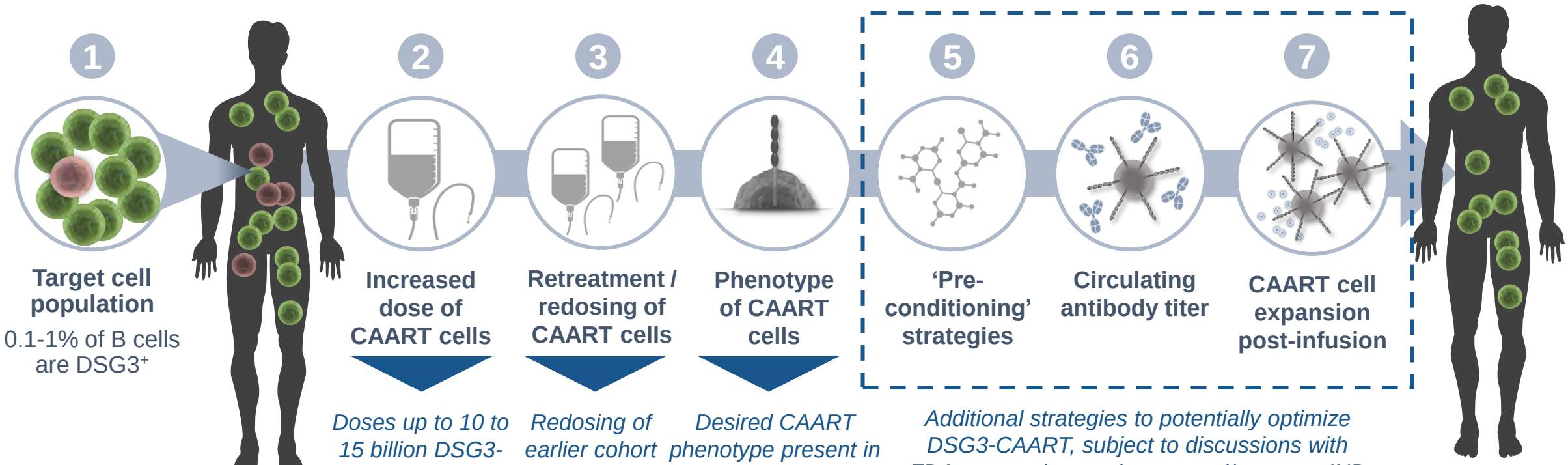
Ongoing & planned cohorts designed to further increase DSG3-CAART exposure

1. Mueller, Karen Thudium, et al. "Cellular kinetics of CTL019 in relapsed/refractory B-cell acute lymphoblastic leukemia and chronic lymphocytic leukemia." Blood, The Journal of the American Society of Hematology 130.21 (2017): 2317-2325.
2. Schuster SJ, Bishop MR, Tam CS, et al. Tisagenlecleucel in Adult Relapsed or Refractory Diffuse Large B-Cell Lymphoma. N Engl J Med. 2019;380(1):45-56. doi:10.1056/NEJMoa1804980
3. The range of persistence observed with anti-CD19 CART therapy in oncology has not been confirmed to be necessary or sufficient for clinical responses in patients with mPV.

* *A1-1-R refers to a subject in cohort A1 who was retreated at the cohort A3 dose (500 million cells).*

Optimizing DSG3-CAART product and patient profiles

Range of strategies to consider for targeted cell therapy approaches in patients with autoimmune diseases



Many options exist to optimize product and patient profiles, ongoing evaluation of strategies to enhance DSG3-CAART exposure

CAART cell

Pathogenic autoreactive B cell

Healthy B cell

MuSK-CAART for patients with MuSK myasthenia gravis

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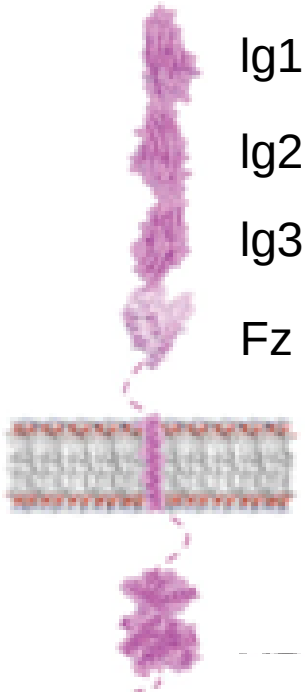
High unmet need in MuSK myasthenia gravis; a valuable CAAR target

All known extracellular domains can be included in the CAAR design

Similarities to pemphigus support clinical potential of CAAR T in MuSK MG

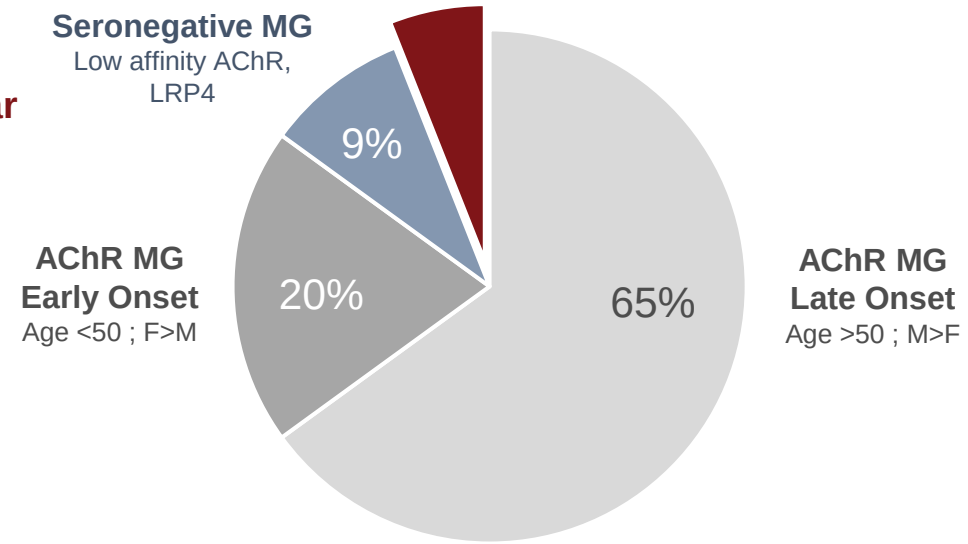
1. IgG4-dominant disease, similar to PV
2. Autoantibody titers drop after rituximab[1,2]
3. Pathogenic B cells are incompletely eliminated by rituximab and persist during relapse[3]



MuSK has similar modular structure and size as DSG3

MuSK MG
6.0 - 7.5%

Typically more severe
Limited treatment options
Early onset – 7:1 females

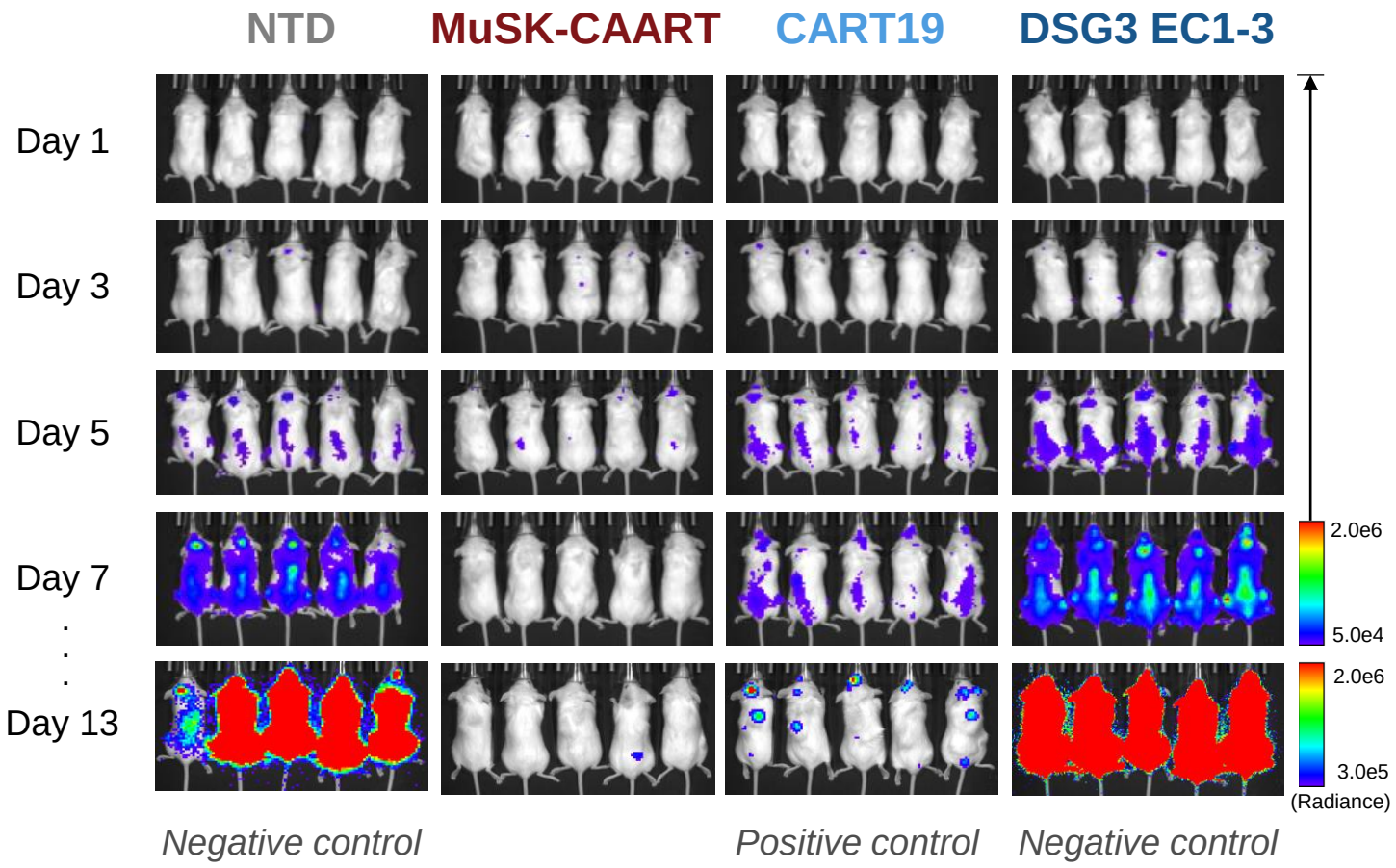


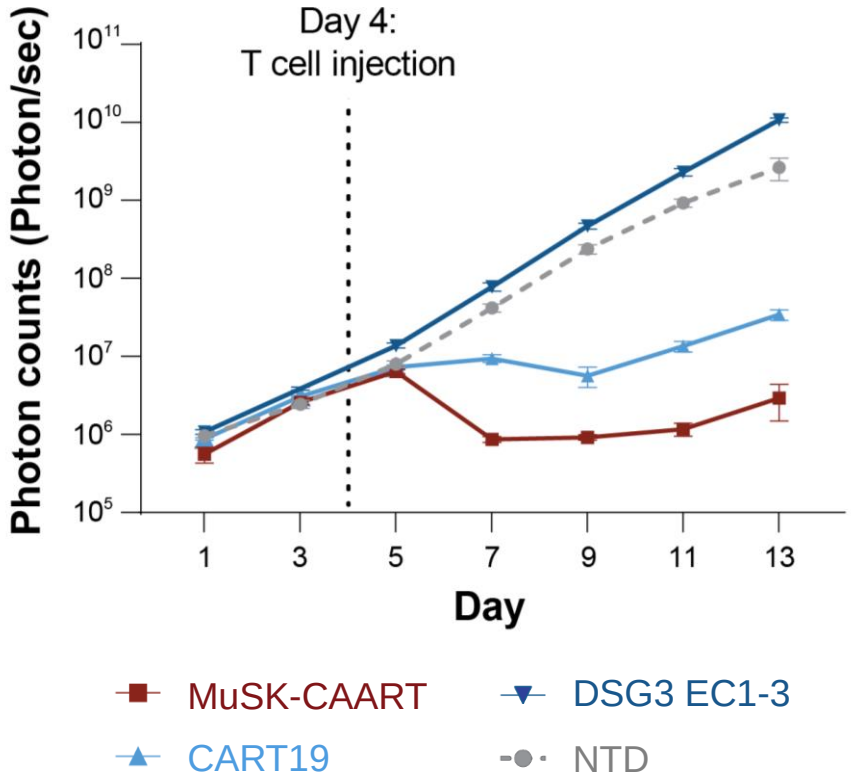
Total US MG Prevalence: 50,000 to 80,000 patients

1. Hain, Berit, et al. "Successful treatment of MuSK antibody–positive myasthenia gravis with rituximab." Muscle & Nerve: Official Journal of the American Association of Electrodiagnostic Medicine 33.4 (2006): 575-580.
2. Illa, Isabel, et al. "Sustained response to Rituximab in anti-AChR and anti-MuSK positive Myasthenia Gravis patients." Journal of neuroimmunology 201 (2008): 90-94.
3. Jiang, Ruoyi, et al. "Single-cell repertoire tracing identifies rituximab-resistant B cells during myasthenia gravis relapses." JCI insight 5.14 (2020).

MuSK-CAART demonstrated specific *in vivo* target engagement[1]

MuSK-CAART eliminated anti-MuSK target cells[2] in an animal model where CART19 cells were a positive control





MuSK-CAART IND open and planning to initiate first-in-human MuSK-CAART trial in 2022

1. https://cabalettabio.com/technology/posters-publications; recently presented at AAI Immunology 2022, MGFA International and ASGCT 2022 conferences in May 2022.
2. Target cells represent a B cell tumor line (CD19 positive) that has been modified to express the anti-MuSK antibody.

MusCAARTes™ study of MuSK-CAART

Fast Track Designation

Strategy for upcoming trial evaluating MuSK-CAART informed by progression of DesCAARTes™ study

Open-label study to determine the maximum tolerated dose & to evaluate safety of MuSK-CAART

Major Inclusion Criteria

- Age: ≥18
- MG severity Class I-IVa
- MGC ≥ 4
- Anti-MuSK antibody positive
- Negative anti-AChR antibody test

Major Exclusion Criteria

- Prednisone > 0.25-0.5 mg/kg/day
- Other autoimmune disorder requiring immunosuppressive therapies

SCREENING → MANUFACTURING → TREATMENT (SINGLE INFUSION) → MONITORING (2-4 WEEKS) → Next Patient

2 *Single dose rather than fractionation*

Part	Cohort	# Subjects
A – Dose Escalation Increasing dose levels with 2 (+4) design *(A1 – 100M*; A2 – 500M*; A3 – 2.5B*)*	A1-A3	2 (+4) per cohort
Highest planned selected dose[1] *(A4 – 5 to 7.5B*)*	A4	6
B – Expansion Expanded subject enrollment at final selected dose	B	~12

1 *Higher starting dose*

3 *versus 3 (+3) design in DesCAARTes™ trial*

Study Endpoint & Objectives

Primary Endpoint: Adverse Events, including DLT

Secondary & Tertiary Objectives: CAAR T expansion/persistence, effect on serum anti-MuSK antibody titer, use of concomitant systemic medications, effect on clinical symptoms, manufacturing success rate

** 100M, 500M, 2.5B and 5.0B to 7.5B refers to the number of MuSK-CAART cells infused for patients in dosing cohorts A1 to A4 (M – millions; B – billions).*
1. A total of 6 subjects will need to have received the final selected dose in Part A of the study.

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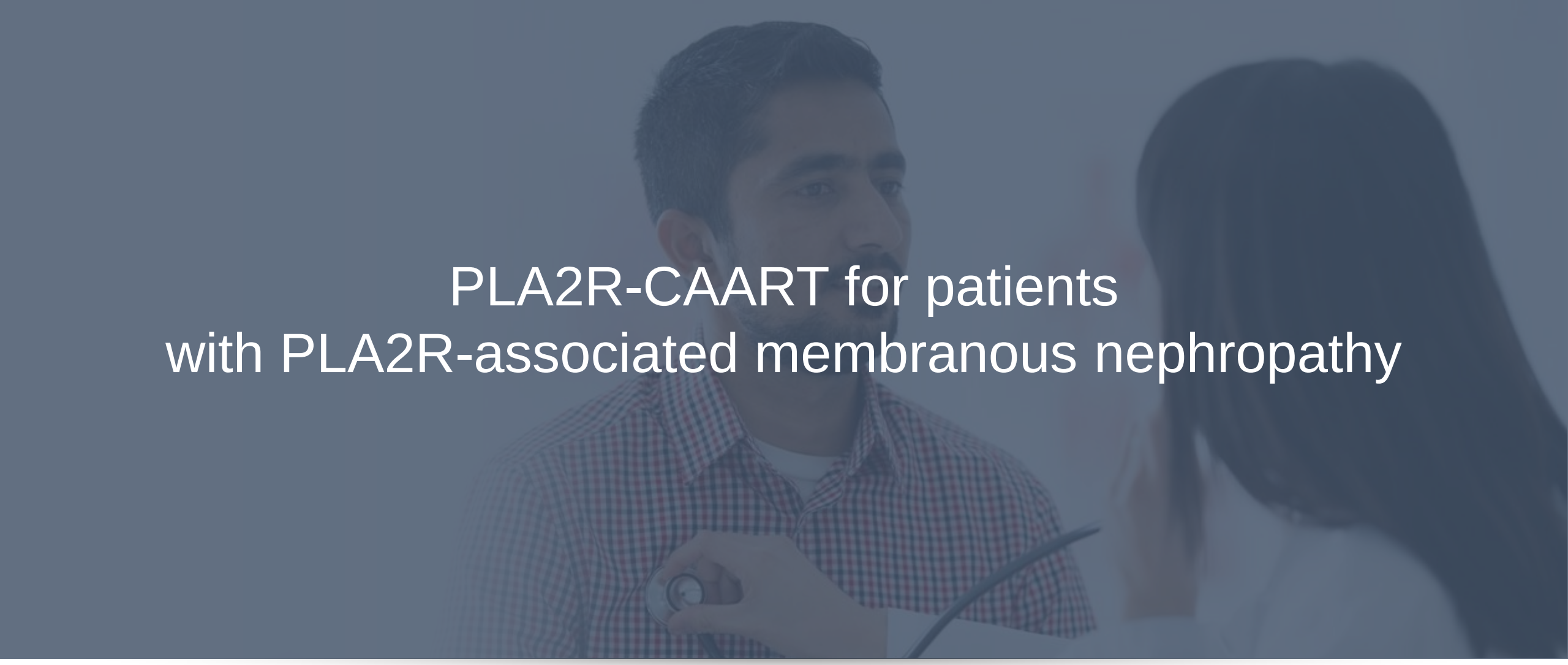

PLA2R-CAART for patients with PLA2R-associated membranous nephropathy

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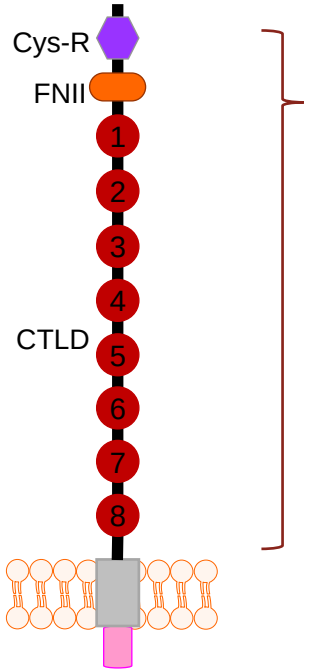
Preclinical stage CAART program in membranous nephropathy

Primary MN is an immune-mediated kidney disease with anti-PLA2R antibodies in ~75% of patients

Accumulating evidence of a correlation between PLA2R antibodies and disease activity, remission and relapse in primary MN

1. PLA2R autoantibody levels routinely used as diagnostic and prognostic markers
2. Autoantibody titer shown to rise rapidly before clinical manifestations
3. Co-localization of antigen & PLA2R antibodies at site of damage in kidney



PLA2R+ MN is attractive for CAAR development

- Epitopes are well-defined
- IgG4-dominant disease, similar to PV and MuSK MG
- Eligible US population
  - prevalence of ~4,000-8,000;
  - incidence of ~700-1,400 / yr

PLA2R-CAART showed *in vitro* antigen-specific cytotoxicity[1]

- PLA2R CAARs demonstrated activity in the presence of physiologic levels of anti-PLA2R autoantibodies
- Candidate CAAR contains targets that bind >95% of anti-PLA2R autoantibodies
- Demonstrated no off-target binding interactions in membrane protein array

1. As presented at the ASN Kidney Week 2021.

Manufacturing

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Manufacturing strategy

Three-stage approach allows for efficient allocation of capital while leveraging experienced partners

Stage 1: Penn[1]	**Stage 2:** CDMOs & CABA Process	**Stage 3:** Cabaletta Facility Commercialization & Scale-Up
2019 –	2021 –	Data-gated, staged investment
• Cell processing capacity secured through Penn partnership • SOPs previously used to develop an FDA approved product • Clinical vector validated	• CDMOs for vector and cell processing with commercial support capabilities	• Leasing followed by engineering and build out of Cabaletta-owned manufacturing facility

1. Penn-sponsored CD19 CAR IND CMC data has been cross-referenced in DSG3-CAART IND to provide additional data reflecting alignment; cross reference not required for MuSK-CAART IND.

Corporate Summary

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Cabaletta Bio leadership

LEADERSHIP TEAM

Steven Nichtberger, M.D.
President, CEO & Chairman

MERCK
tengion
Penn

Samik Basu, M.D.
Chief Scientific Officer

Adaptimmune
MERCK

Gwendolyn Binder, Ph.D.
President, Science & Technology

Adaptimmune
Penn

David J. Chang, M.D., M.P.H.
Chief Medical Officer

AstraZeneca
Penn

Arun Das, M.D.
Chief Business Officer

Goldman Sachs
Children's Hospital of Philadelphia

Michael Gerard
General Counsel

Spark Therapeutics
SANDOZ A Novartis Division

Heather Harte-Hall
Chief Compliance Officer

Adaptimmune
Pfizer

Anup Marda
Chief Financial Officer

Bristol-Myers Squibb

Martha O'Connor
Chief HR Officer

Bristol-Myers Squibb
accentureconsulting

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Mark Simon, M.B.A.

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Michael C. Milone, M.D., Ph.D.
Co-Founder and Co-Chair

Jay Siegel, M.D.

Drew Weissman, M.D., Ph.D.

Multiple potential data catalysts with possible pipeline read-through

DesCAARTes™ trial ongoing: Currently progressing cohort A5 (5.0-7.5B cells)

- Data presented at ASGCT 2022 demonstrate dose-dependent increase in persistence
  - Cohort A4 persistence approached the lower end of the range seen in anti-CD19 CART with LD in oncology[1,2,3]
  - Demonstrated a favorable safety profile in cohorts A1 to A4
- Additional data from the DesCAARTes trial anticipated at scientific meetings in mid-2022
  - Clinical and translational data for cohort A4[4]
  - 28-day safety data for cohort A5[4]
- Multiple additional planned cohorts designed to enhance DSG3-CAART exposure

MuSK-CAART: Plan to initiate first-in-human trial in 2022; received FDA Fast Track Designation

Expanding network of academic & industry partners

WuXi AppTec
ARTISANBIO
Penn
University of Pennsylvania
Yale
Stanford Medicine
OxfordBioMedica
Children's Hospital of Philadelphia
UC DAVIS
University of California
IOWA

1. Mueller, Karen Thudium, et al. "Cellular kinetics of CTL019 in relapsed/refractory B-cell acute lymphoblastic leukemia and chronic lymphocytic leukemia." Blood, The Journal of the American Society of Hematology 130.21 (2017): 2317-2325.
2. Schuster SJ, Bishop MR, Tam CS, et al. Tisagenlecleucel in Adult Relapsed or Refractory Diffuse Large B-Cell Lymphoma. N Engl J Med. 2019;380(1):45-56. doi:10.1056/NEJMoa1804980
3. The range of persistence observed with anti-CD19 CART therapy in oncology has not been confirmed to be necessary or sufficient for clinical responses in patients with mPV.
4. Assumes no dose-limiting toxicities are observed during each cohort, uninterrupted enrollment and no delays due to COVID-19 resurgence occur in the trial.

* *20M, 100M, 500M, 2.5B, 5.0B to 7.5B and 10B to 15B refers to the number of DSG3-CAART cells infused for patients in dosing cohorts A1 to A6m (M – millions; B – billions).*

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Corporate Presentation

MAY 2022