UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
Washington, D.C. 20549

FORM 8-K

CURRENT REPORT
Pursuant to Section 13 or 15(d) of
The Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): June 2, 2020

CABALETTA BIO, INC.
(Exact name of Registrant as Specified in its Charter)

Delaware	**001-39103**	**82-1685768**
(State or other jurisdiction of incorporation)	**(Commission File Number)**	**(I.R.S. Employer Identification No.)**
2929 Arch Street, Suite 600, Philadelphia, PA		**19104**
(Address of principal executive offices)		**(Zip Code)**

(267) 759-3100
(Registrant's telephone number, including area code)

Not Applicable
(Former name or former address, if changed since last report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

Title of Each Class	Trading Symbol(s)	Name of Each Exchange on Which Registered
Common Stock, par value $0.00001 per share	**CABA**	**The Nasdaq Global Select Market**

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§240.12b-2 of this chapter).

Emerging growth company ☒

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 5.07 Submission of Matters to a Vote of Security Holders.

Cabaletta Bio, Inc., a Delaware corporation (the "Company") held its Annual Meeting of Stockholders (the "Annual Meeting") on June 2, 2020. As of April 15, 2020, the record date for the Annual Meeting, there were 17,624,503 outstanding shares of the Company's common stock. The Company's stockholders voted on the following matters, which are described in detail in the Company's Definitive Proxy Statement filed with the U.S. Securities and Exchange Commission on April 23, 2020: (i) to elect one director, Brian Daniels, M.D., as a Class I director of the Company to serve for a three-year term expiring at the Company's 2023 annual meeting of stockholders and until his successor has been duly elected and qualified, subject to his earlier death, resignation or removal ("Proposal 1") and (ii) to ratify the appointment of Ernst & Young as the Company's independent registered public accounting firm for the fiscal year ending December 31, 2020 ("Proposal 2").

The Company's stockholders approved the Class I director nominee, Brian Daniels, M.D., recommended for election in Proposal 1 at the Annual Meeting. The votes cast at the Annual Meeting were as follows:

For	Withheld	Broker Non-Votes
9,666,638	1,147,329	823,061

The Company's stockholders ratified the appointment of Ernst & Young as the Company's independent registered public accounting firm for the fiscal year ending December 31, 2020, recommended for ratification in Proposal 2 at the Annual Meeting. The votes cast at the Annual Meeting were as follows:

For	Against	Abstain	Broker Non-Votes
11,529,472	107,556	0	0

No other matters were submitted to or voted on by the Company's stockholders at the Annual Meeting.

Item 7.01 Regulation FD Disclosure.

On June 3, 2020, the Company posted to the "Investors & Media" section of the Company's website at *www.cabalettabio.com* an updated corporate presentation providing a corporate overview and updated development plan (the "Corporate Presentation"). A copy of the Corporate Presentation is attached hereto as Exhibit 99.1 and is incorporated by reference into this Item 7.01 of this Current Report on Form 8-K.

The information contained in Item 7.01 of this Current Report on Form 8-K is being furnished and shall not be deemed to be "filed" for the purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the "Exchange Act"), or otherwise subject to the liabilities of that section and shall not be incorporated by reference in any filing under the Securities Act of 1933, as amended, or the Exchange Act, except as shall be expressly set forth by specific reference in such filing.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits

99.1 Cabaletta Bio, Inc. Corporate Presentation, dated June 3, 2020.

SIGNATURE

Pursuant to the requirements of the Securities Exchange Act of 1934, as amended, the registrant has duly caused this report to be signed on its behalf by the undersigned, hereunto duly authorized.

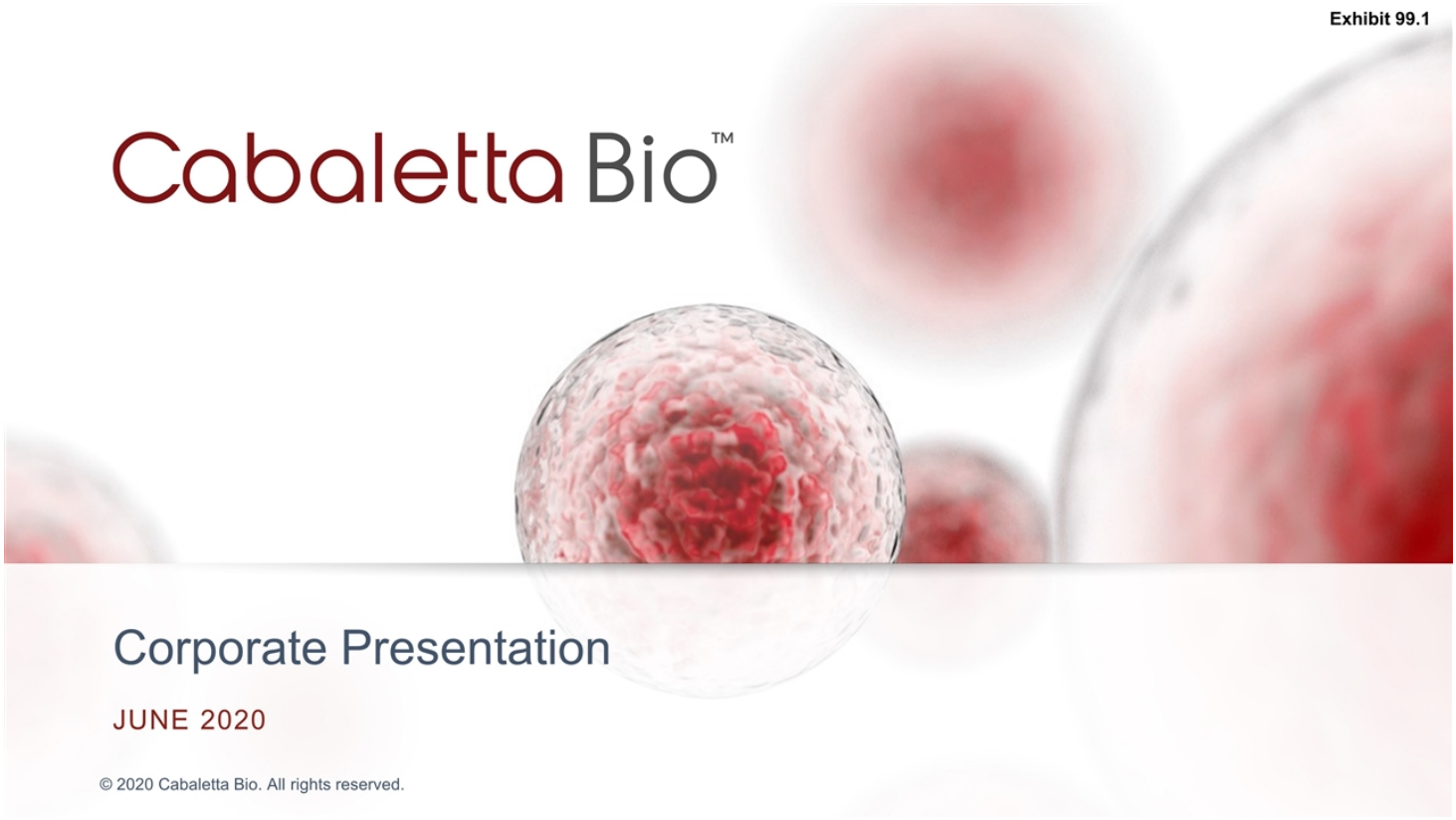
CABALETTA BIO, INC.

Date: June 3, 2020

By: /s/ Steven Nichtberger
Steven Nichtberger, M.D.
President and Chief Executive Officer

Exhibit 99.1

Cabaletta Bio™



Corporate Presentation

JUNE 2020

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 and does not constitute an offer to sell or a solicitation of an offer to buy securities, nor shall there be any sale of any securities in any state or jurisdiction in which such offer, solicitation or sale would be unlawful prior to registration or qualification under the securities laws of any such state or jurisdiction. 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 neither this Presentation, nor any sale of securities, shall 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" relating to our business, operations, and financial conditions, including, but not limited to, current beliefs, expectations and assumptions regarding the future of our business, future plans and strategies, our development plans, our preclinical and clinical results and other future conditions. 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. Factors which could cause actual results to differ materially from those in the forward-looking statements include, among others, the success, cost, and timing of our product candidate development activities and preclinical studies and clinical trials, our ability to obtain and maintain regulatory approval for our product candidates, our ability to commercialize our product candidates, future agreements with third parties in connection with the development or commercialization of our product candidates, the size and growth potential of the market for our product candidates, our ability to contract with third-party suppliers and manufacturers and our ability to develop internal manufacturing capabilities and facilities, the accuracy of our estimates regarding expenses, future revenue, capital requirements, and needs for additional financing, and our ability to obtain and maintain intellectual property protection for our product candidates. 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, uncertainties caused by adverse economic conditions, including, without limitation, 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. 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.

Cabaletta Bio™

Cabaletta overview

Engineering and developing CAAR T products to treat B cell-mediated autoimmune diseases

- Leveraging commercially approved CART19 technology though Penn partnership
- Pursuing targets where there is a biologic opportunity for cure with the CABA platform
- First mover advantage developing highly specific targeted cell therapy for autoimmune diseases

Lead clinical program, DSG3-CAART, in mucosal pemphigus vulgaris (mPV) patients

- DesCAARTes™ trial ready to launch with clinical acute tolerability (8 day) data from initial cohort by 1H21[1]
- Multiple clinical sites engaged across the U.S. supported by validated manufacturing partnership with Penn
- Fast Track and Orphan Drug Designations granted in 1H20

Preclinical pipeline led by MuSK-CAART for myasthenia gravis with IND filing anticipated in 2H21[1]

- IND-enabling studies ongoing with *in vivo* preclinical data presented at 2020 American Academy of Neurology
- Manufacturing validation on track with CMO partner to initiate in 2H20

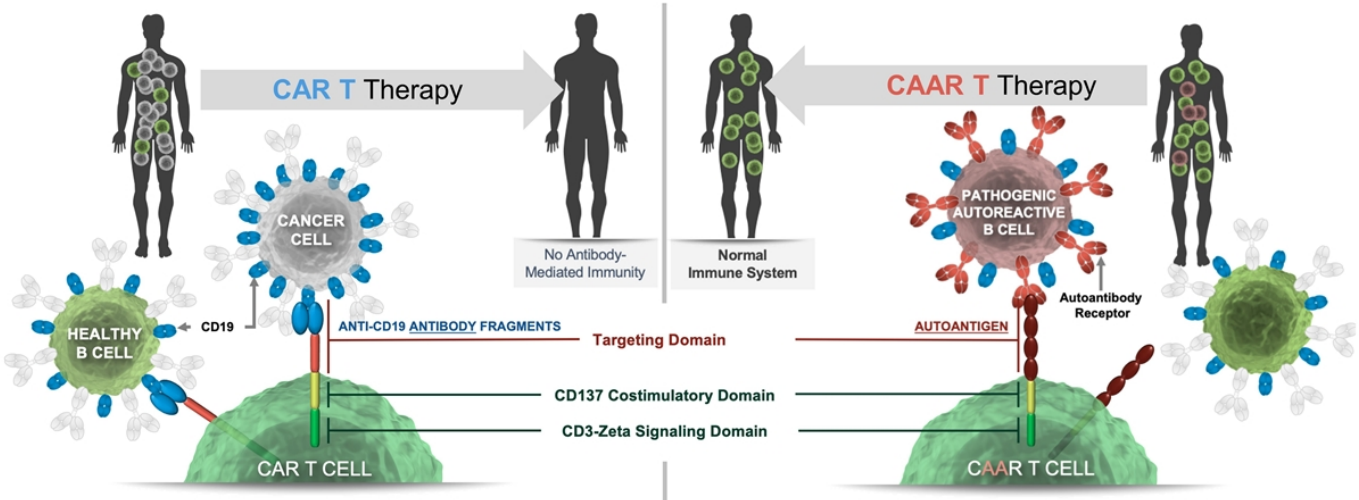
Issued U.S. patent on lead clinical program with emerging robust IP portfolio

- Issued CAAR T product patent covers all or any part of the relevant human antigens (DSG3 and DSG1)

Cash runway into at least 3Q22 with $131 million in cash at March 31, 2020

[1] Assumes lifting of current COVID-19-related restrictions and no future restrictions due to subsequent COVID-19 resurgence

Our scientific platform leverages CAR T technology



Chimeric Antigen Receptor T cell

Kymriah

Chimeric AutoAntibody Receptor T cell

CABA CAAR T

CABA (Cabaletta Approach for Selective B cell Ablation) platform

Scientific, clinical and commercial assessment

to inform product candidate development

Epitope mapping

to determine regions targeted by autoantibodies

Optimize CAAR construct / design

with the goal of selectively ablating reactive B cells

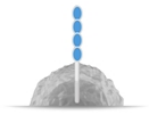
Preclinical *in vitro* and *in vivo* testing

to evaluate efficacy and safety

Vector & Clinical Cell manufacturing

MuSK-CAART

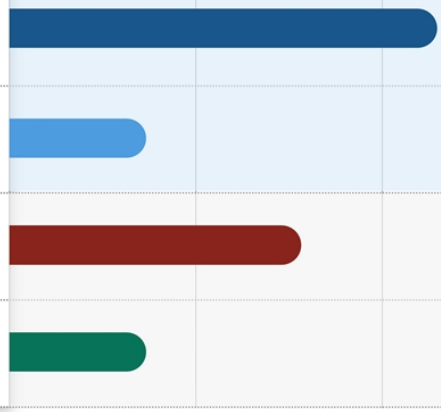
Clinical trials



DSG3-CAART

Pipeline addressing multiple disease targets

Indication	Program	Discovery[1]	Preclinical	Phase 1	Phase 2	Phase 3[2]
Mucosal Pemphigus Vulgaris	DSG3-CAART					
Mucocutaneous Pemphigus Vulgaris	DSG3/1-CAART					
MuSK Myasthenia Gravis	MuSK-CAART					
Hemophilia A w/ FVIII Alloantibodies	FVIII-CAART					

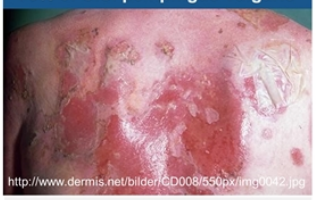


1 In our discovery stage, we perform epitope mapping and optimize CAAR construct and design.
2 May not be required if Phase 2 is a registrational clinical trial.

Cabaletta Bio™

Overview of Pemphigus Vulgaris

Working to meet an unmet need for patients

	Mucosal PV[1] 25% of U.S. pemphigus vulgaris	Mucocutaneous PV[1] 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
Disease Incidence	350 US / 600 EU	1,050 US / 1,800 EU
Disease Prevalence	**4,250 US / 6,250 EU**	**12,750 US / 18,750 EU**

Current Treatment Landscape

Broad immunosuppression

- Modestly effective
- Poorly tolerated

B cell depletion with rituximab[2,3,4]

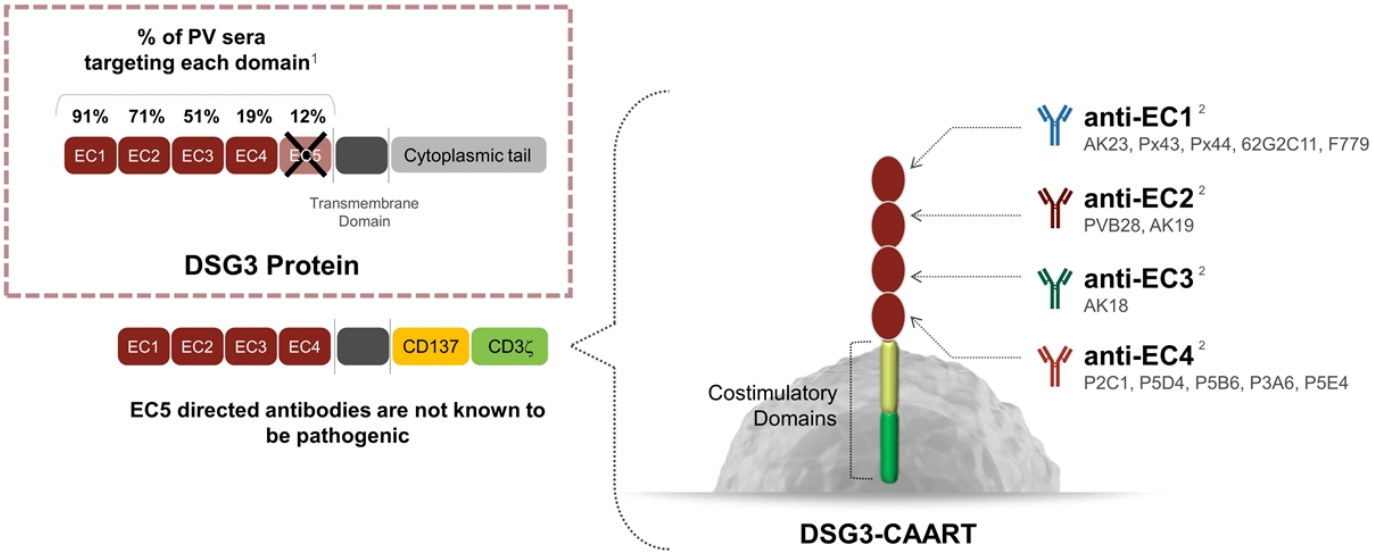
- Offers transient remission
- Majority of responders relapse

Chronic B cell depletion with rituximab

- 5.4% annual risk of severe infection
- Up to 1.9% lifetime risk of fatal infection

1. Image credit: D@nderm
2. 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.
3. Kushner, Carolyn J., et al. "Factors Associated With Complete Remission After Rituximab Therapy for Pemphigus." JAMA dermatology (2019).
4. Jancin, Bruce. "Rituximab bests mycophenolate in pemphigus vulgaris," Dermatology News, Nov 2019, Vol. 50:11, p. 2.; Press Release, Roche, 14 Oct 2019.

DSG3-CAART encompasses all known pathogenic epitopes



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.

DSG3-CAART preclinical data

No evidence of toxicity at clinically relevant doses with selective and specific target engagement

	INDICATOR	RESULTS
Tolerability	***In vitro* off-target toxicity**	No specific cytotoxicity at clinically relevant cell numbers vs FcγR-expressing cells No interactions confirmed with human membrane proteins
	***In vivo* off-target toxicity**	No off-target effects detected at clinically relevant doses
Target Engagement	**Anti-DSG3 autoantibody titer**	Serologic 'remission' - dose-dependent elimination of anti-DSG3 B cells and antibodies
	CAAR T cell engraftment	Dose-dependent increase in CAAR-positive cells observed via flow cytometry
	Tissue blistering	Histologic 'remission' - no blistering of oral mucosa
	Anti-DSG3 hybridoma outgrowth	Significantly delayed outgrowth despite soluble anti-DSG antibodies

DesCAARTes™: Phase 1 clinical trial in mucosal-dominant PV (mPV) patients

Open-label study to determine the maximum tolerated dose & fractionation of DSG3-CAART

Major Inclusion Criteria

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

Major Exclusion Criteria

- Rituximab in last 6 months
- Prednisone > 0.25mg/kg/day
- Other autoimmune disorder requiring immunosuppressive therapies
- Recent investigational treatment
- ALC < 1,000 at screening

SCREENING PERIOD → **TREATMENT PERIOD** (1-3 WEEKS) → **FOLLOWED** (2-4 WEEKS) → **Next Patient**

Part	Cohort	# Subjects
A – Dose Escalation Fractionated infusion at increasing dose levels	A1-A4	3 (+3) per cohort
B – Dose Consolidation Consolidating selected dose fractions into a single infusion	B1-B2	3 (+3) per cohort
C – Expansion[1] Expanded subject enrollment at final selected dose	C	~12
	Total	**~30 (+18)**

Study Endpoint & Objectives

Primary Endpoint: Adverse Events, including Dose Limiting Toxicity

Secondary Objectives: **DSG3 ELISA titer changes**, rate of/time to/duration of remission, manufacturing success rate, CAAR T expansion/persistence

1. FDA has requested, and the Company has agreed, that we will share data from cohort A to inform a discussion on the optimal design of cohort C. According to FDA guidance, the submission of cohort A data is not gating to planned enrollment in cohort B.

DesCAARTes™ clinical trial assessments and timeframes

Safety assessed acutely and at 3 months, with ability to measure CAAR T engagement by 6 months

Key Risks	Context and Mitigating Strategies	Options to Consider[2]
CRS / Neurotoxicity	• Product designed to kill <1% of B cells • Low initial dose with fractionation • No lymphodepletion	• Manage with standard protocols
Soluble antibody	• Fractionated dosing designed to mitigate against potential toxicity	• Pretreat w/ plasmapheresis or IVIG • Limit soluble anti-DSG3 antibody inclusion criteria
Insufficient efficacy / CAAR T engraftment	• Wide cell dose range planned	• Consider increased dose and/or preconditioning / lymphodepletion
Disease flare with medication taper	• Limit dose of corticosteroid use to enroll	• Local / oral steroids; plasmapheresis
Skin toxicity from cross-reactivity	• No preclinical signals	• Local / oral steroids; plasmapheresis
New autoimmune disorder or worsening	• Patients with active autoimmune disorders requiring immunosuppressants excluded	• Autoimmune therapies as needed

Declining anti-DSG3 titers[1]

Assuming selective B cell ablation in 2-4 weeks, serum IgG (half-life ~3 weeks) should fall within 6 months

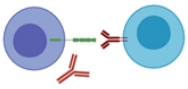
Key efficacy measures

- DSG3 antibody titer (ELISA)[1]
- Disease activity (clinical)
- Steroid / immunosuppressive use

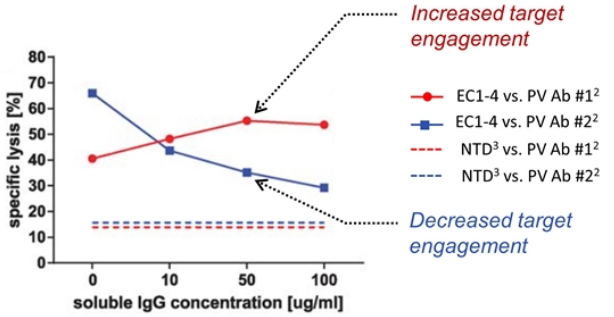
1. Spindler, Volker, et al. "Mechanisms causing loss of keratinocyte cohesion in pemphigus." Journal of Investigative Dermatology 138.1 (2018): 32-37.
2. To be potentially incorporated in future protocol amendments or trials after discussion with FDA.

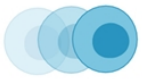
Soluble antibodies may alter the dynamics of DSG3-CAART proliferation

Preclinical data suggests soluble antibodies can partially activate or inhibit DSG3 CAAR T cells[1]

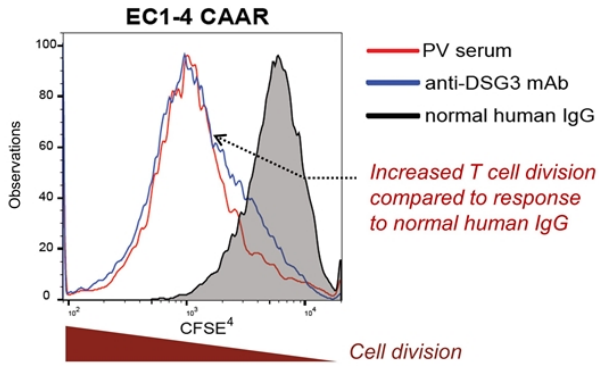


Inhibitory antibodies reduce but do not prevent DSG3-CAART target engagement





Soluble anti-DSG3 induces CAART cell proliferation



1. Ellebrecht, Christoph T., et al. "Reengineering chimeric antigen receptor T cells for targeted therapy of autoimmune disease." Science 353.6295 (2016): 179-184.
2. Antibodies derived from a hybridoma model.
3. Non-transduced T cell.
4. CFSE (carboxyfluorescein diacetate succinimidyl ester) is a dye used in flow cytometric monitoring that reduces in intensity as it is distributed in actively dividing cells. Non-dividing cells will retain more CFSE dye.

Preconditioning regimens in CAAR T cell therapy may not be required

Past successes in HIV and multiple myeloma without preconditioning plus differences in patient populations are relevant[1,2]

- Data suggest preconditioning may not be necessary to drive responses beyond leukemia / lymphoma
  - Recent MM patient data showed non-significant differences in ORR and rate of cytokine release syndrome in patients receiving CART-BCMA with and without lymphodepletion[1]
  - Studies from three clinical trials for patients with HIV showed CAR T cells were detected in 98% of samples tested for at least 11 years after infusion
- Potential activating effect of soluble antibody on engraftment and function is a key consideration in autoimmune patients
- Multiple infusions, higher dose, and cytokine support may offer more tolerable approach for autoimmune patients

Lymphodepletion Mechanism	Oncology	Autoimmunity
Reduction of T cell growth cytokine sinks[3]	+	+
Depletion of suppressor cells[4]	+	–

\+ *Lymphodepletion mechanism likely to apply*

– *Lymphodepletion mechanism unlikely to apply*

1. Cohen, Adam D., et al. "B cell maturation antigen–specific CAR T cells are clinically active in multiple myeloma." The Journal of Clinical Investigation 129.6 (2019).
2. Scholler, John, et al. "Decade-long safety and function of retroviral-modified chimeric antigen receptor T cells." Science translational medicine 4.132 (2012): 132ra53-132ra53.
3. Gattinoni et al. "Removal of homeostatic cytokine sinks by lymphodepletion enhances the efficacy of adoptively transferred tumor-specific CD8+ T cells." The Journal of Experimental Medicine 202(7):907-912
4. Wang, Shu and Plautz "Host lymphodepletion augments T cell adoptive immunotherapy through enhanced intratumoral proliferation of effector cells: Cancer Research 65(20): 9547-54.

Key clinical trial milestones and data readouts

Evaluate safety by dosing cohort

- Acute safety data to be evaluated at 8 days post-infusion
- Primary endpoint data at 3 months

Target engagement during dose escalation to be evaluated

- DSG3 antibody is necessary and sufficient to cause mPV (per consensus document[1])
  - DSG3 antibody half-life ~3 weeks
  - Potential evidence of reduction of anti-DSG3 levels may be observed within 6 months
  - Clinical responses including mucosal lesion response and absence of recurrence may take longer

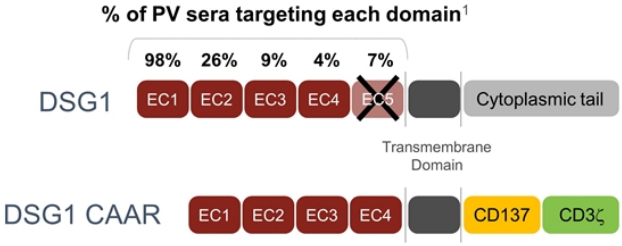
1. Spindler V, Eming R, Schmidt E, et al. Mechanisms Causing Loss of Keratinocyte Cohesion in Pemphigus. J Invest Dermatol 2018;138:32-7.

DSG3/1 CAART for mucocutaneous PV (mcPV)

Plan to potentially submit an IND after review of safety and target engagement data from DSG3-CAART

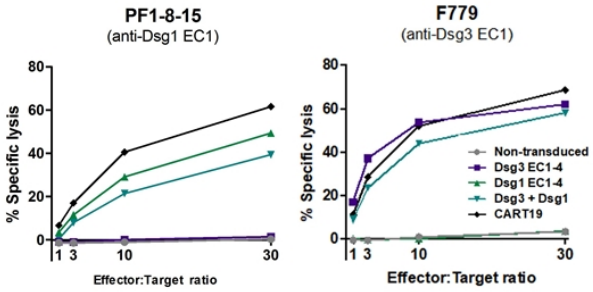
DSG3/1 CAARs designed for mcPV

- DSG3 and DSG1 autoantibodies
- Most severe and common form of PV (~75%)
- Mucosal blistering, plus skin erosion and blistering
- Managed with immune suppression, similar to mPV
  - High risk of relapse
  - Potential for hospitalizations and fatal infections



DSG1-CAART and DSG3-CAART both demonstrated specific cytotoxicity *in vitro*

- Together, DSG1-CAART and DSG3-CAART demonstrated cytotoxicity towards anti-DSG3 and anti-DSG1 B cells



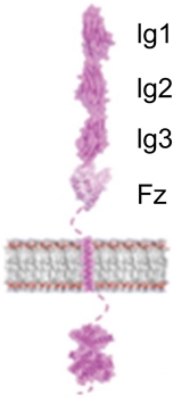
1. Ohyama, Bungo, et al. "Epitope spreading is rarely found in pemphigus vulgaris by large-scale longitudinal study using desmoglein 2–based swapped molecules." Journal of investigative dermatology 132.4 (2012): 1158-1168.

Cabaletta Bio™

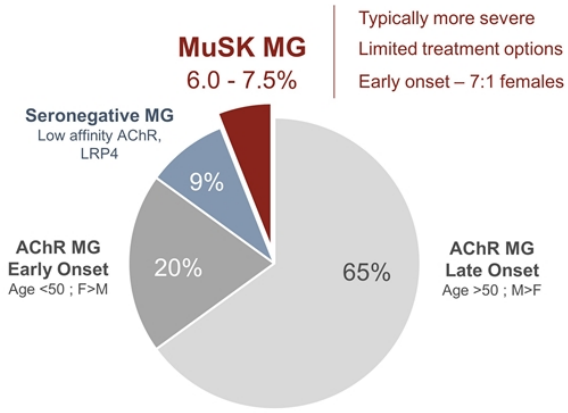
High unmet need in MuSK myasthenia gravis; a valuable CAAR target

All known extracellular domains can be included in the CAAR design

Similar to pemphigus, autoantibody titers drop after rituximab[1,2], supporting the clinical potential of CAART in MuSK myasthenia gravis



MuSK has similar modular structure and size as DSG3

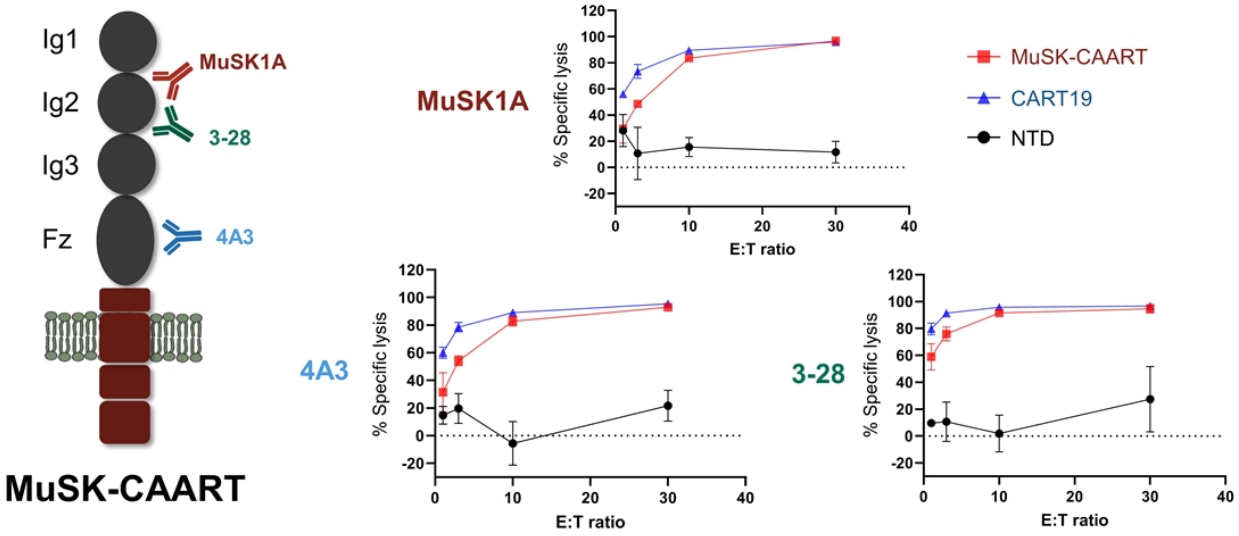


Prevalence: ~65,000 patients in the US

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.

MuSK-CAART *in vitro* selective & specific target engagement[1]

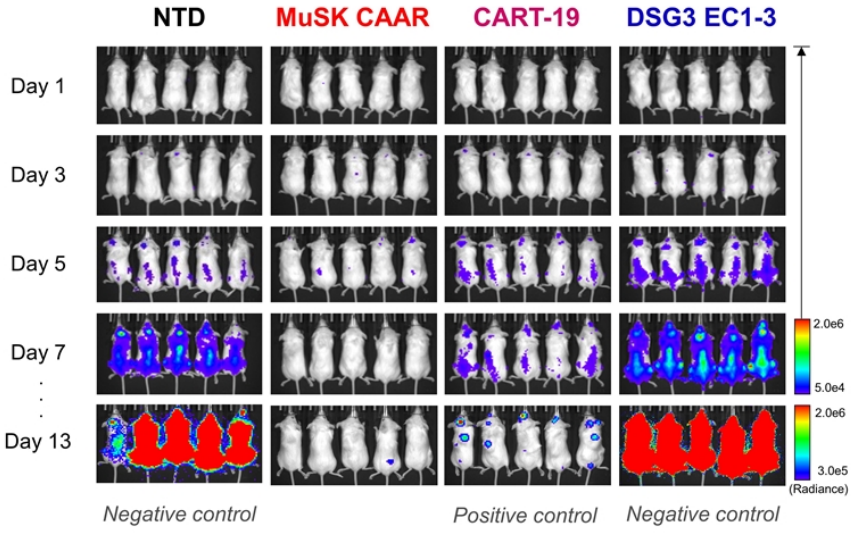
MuSK-CAART showed similar potency against target cells that bind to different epitopes

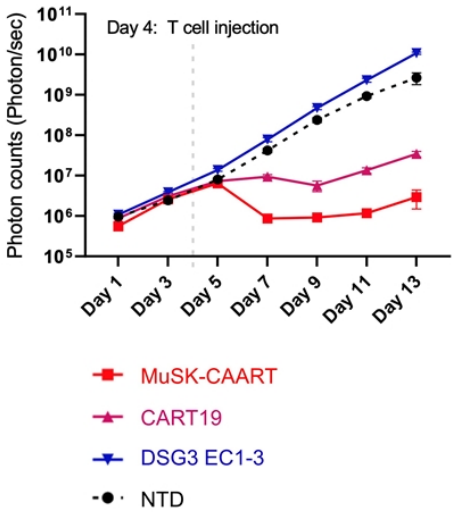


1. https://cslide-us.ctimeetingtech.com/aan2020/attendee/eposter/poster/2402?q=payne

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

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





1. https://cslide-us.ctimeetingtech.com/aan2020/attendee/eposter/poster/2402?q=payne
2. Target cells represent a B cell tumor line (CD19 positive) that has been modified to express the anti-MuSK antibody.

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Hemophilia A and neutralizing alloantibodies

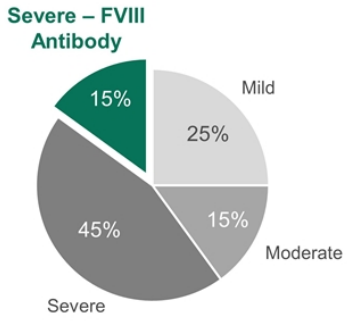
B cell-mediated inhibition of Factor VIII reduces efficacy of replacement therapy

- FVIII neutralizing alloantibodies can block protein or gene replacement therapy
  - Increased incidence in severe disease where native FVIII is not present
- Inhibitors persist in 70% of patients despite tolerance induction therapy[3]
- Managing Hemophilia A patients with inhibitors can cost up to $1M per year

	HEMOPHILIA A POPULATION (US ONLY)		
	Mild	Moderate	Severe
FVIII levels	5-40%	1-5%	<1%
% of cases	25%[1]	15%[1]	60% (~12,000 patients)[1,2]
Consequence	Bleeding after trauma or surgery	Hemarthrosis, synovitis muscle contracture cerebral stroke, death	Debilitating hemarthrosis muscle necrosis strokes, death
Risk of FVIII neutralizing alloantibodies	Minimal	Moderate (~10%)[3]	High (~25%)[3] ~3,000 US patients

US Hemophilia A Population

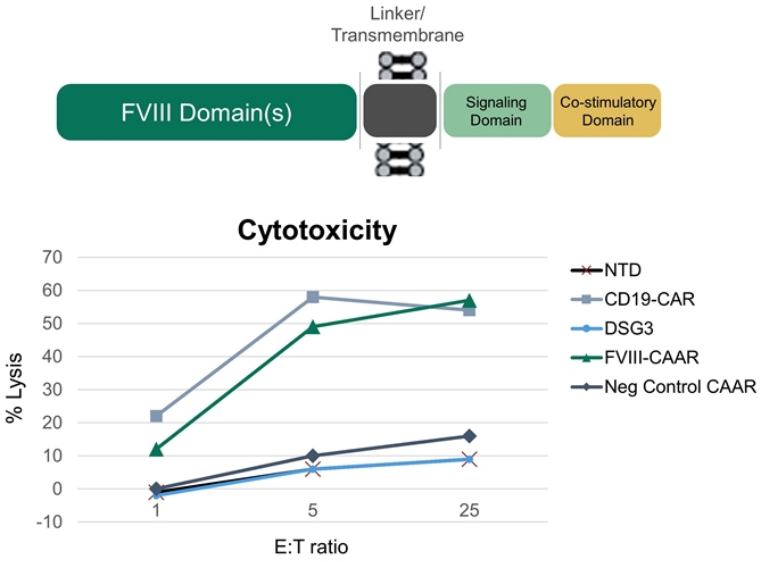
Total US: 20,000 patients



1. https://www.hemophilia.org/Bleeding-Disorders/Types-of-Bleeding-Disorders/Hemophilia-A.
2. Peyvandi, Flora, et al. "A randomized trial of factor VIII and neutralizing antibodies in hemophilia A." New England Journal of Medicine 374.21 (2016): 2054-2064.
3. Arruda, Valder R., and Ben J. Samelson-Jones. "Gene therapy for immune tolerance induction in hemophilia with inhibitors." Journal of Thrombosis and Haemostasis 14.6 (2016): 1121-1134.

FVIII-CAART *in vitro* proof of concept data

Selective and specific target engagement



Cabaletta is engineering optimized constructs

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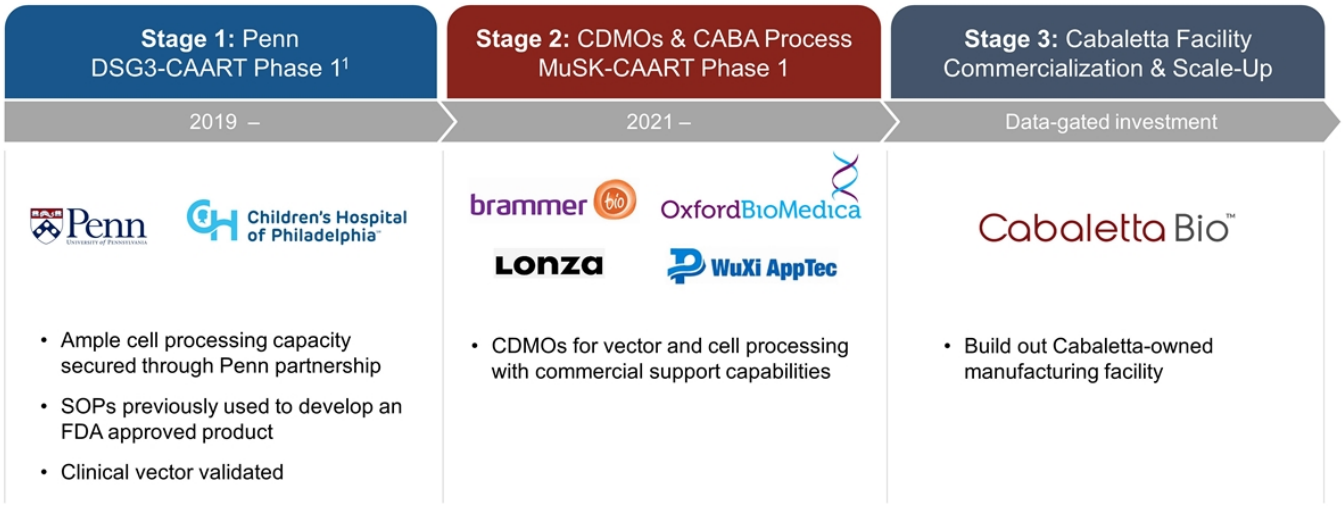
Manufacturing strategy

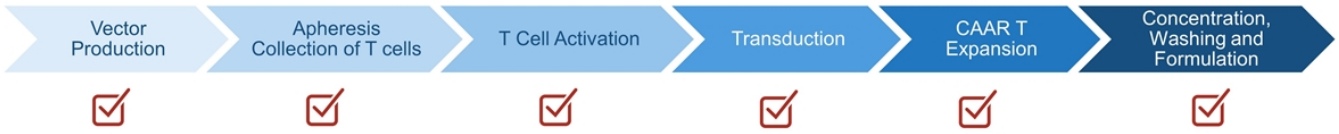
Three-stage approach allows for efficient allocation of capital while leveraging experienced partners



1. Penn-sponsored CD19 CAR IND CMC data has been cross-referenced in DSG3-CAART IND to provide additional data reflecting alignment.

Parallel steps in manufacturing process[1] for CAR T vs CAAR T

Vector production and cell processing are key risks mitigated by strategy, partnership, process and people



Conserving the clinically validated CD19 CAR cell manufacturing process minimizes confounding risks

- Penn process, not Novartis process; avoids Kymriah release challenges[2]

Cross referenced the Penn IND to minimize operational risk

- Development work has demonstrated efficient manufacture of DSG3-CAART cells from PV patients[3]

Multiple runs contractually secured each month at Penn currently on track

- Subject to future COVID-19 impact

DSG3 vector supply secured for next 2-3 years

1. Penn-sponsored CD19 CAR IND CMC data has been cross-referenced in DSG3-CAART IND to provide additional data reflecting alignment.
2. Manufacturing challenges were due to release specifications: https://www.biopharmadive.com/news/novartis-hits-car-t-manufacturing-snag-as-kymriah-sales-disappoint/528202/.
3. T cells isolated from patients with a range of treatment regimens of low to high intensity were tested; the highest intensity regimen and patients with ALC<1000 cells/uL expanded less well and will be excluded from the trial design.

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Leadership team

LEADERSHIP TEAM

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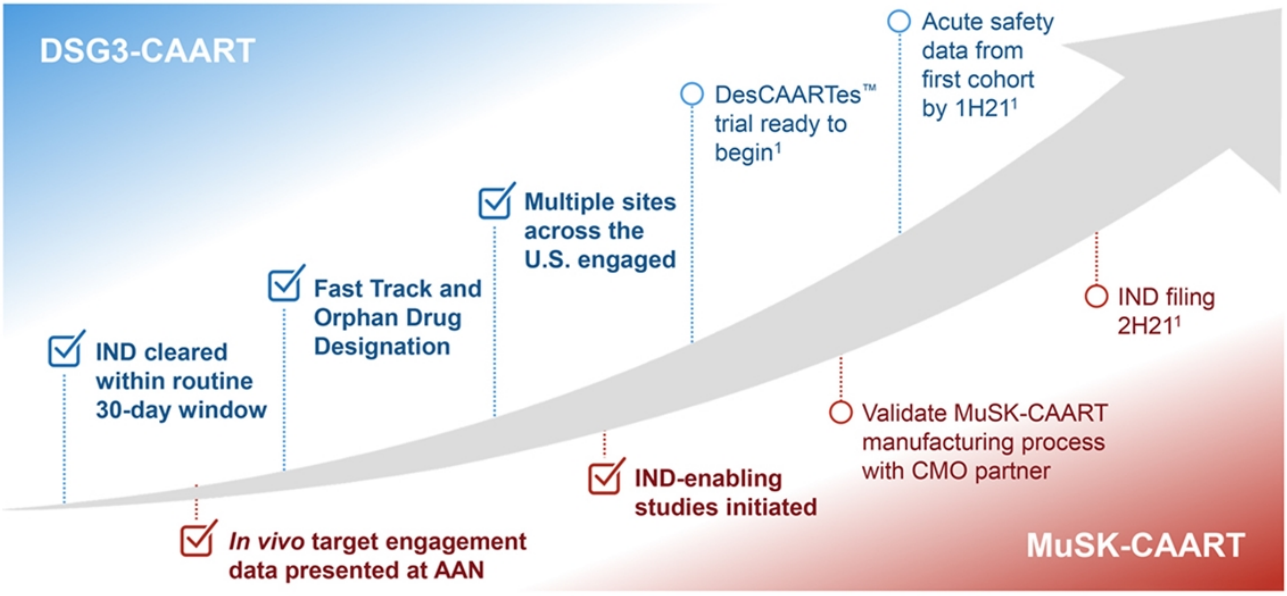
janssen FDA

Iain McInnes

University of Glasgow

Recent highlights and anticipated upcoming milestones



1 Assumes lifting of current COVID-19-related restrictions and no future restrictions due to subsequent COVID-19 resurgence.

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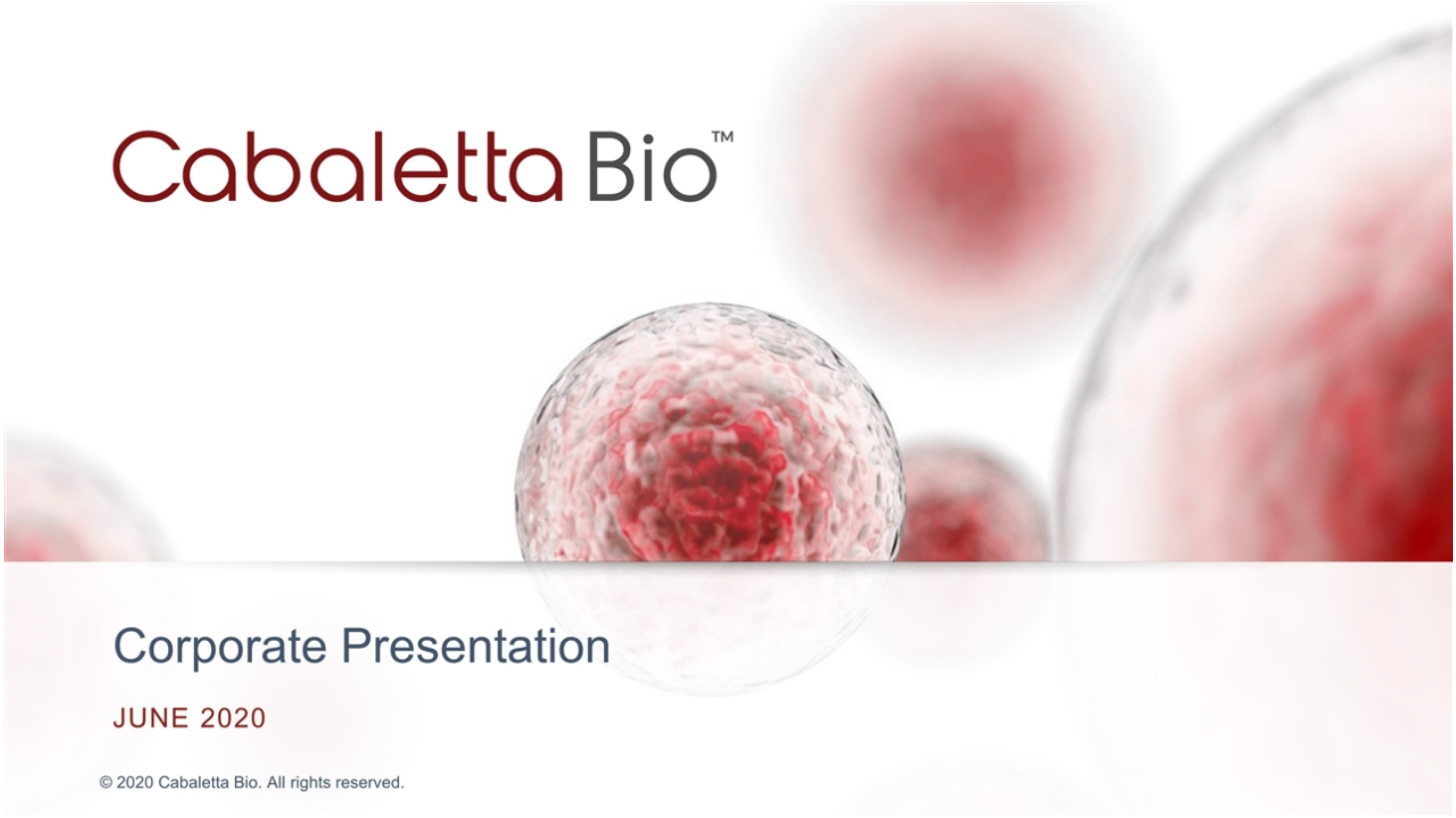

Corporate Presentation

JUNE 2020