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): September 7, 2021

CATALYST BIOSCIENCES, INC.
(Exact name of registrant as specified in its charter)

Delaware	**000-51173**	**56-2020050**
(State or other jurisdiction of incorporation)	**(Commission File Number)**	**(IRS Employer Identification No.)**

611 Gateway Blvd, Suite 710, South San Francisco, CA 94080
(Address of principal executive offices)

(650) 871-0761
(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	**CBIO**	**Nasdaq**

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 7.01 Regulation FD Disclosure.

On September 7, 2021, Catalyst Biosciences, Inc. (the "Company") posted an update to its corporate presentation (the "Presentation") on its website, ir.catalystbiosciences.com/presentations-events. A copy of the Presentation is attached hereto as Exhibit 99.1.

The information in this Item 7.01 of this Current Report on Form 8-K (including Exhibit 99.1) is being furnished and shall not be deemed "filed" for 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. The information in this Current Report shall not be deemed incorporated by reference in any filing under the Securities Act of 1933, as amended, or the Exchange Act, except as expressly set forth by specific reference in such a filing.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits

Exhibit No.	Description
99.1	Presentation slide deck.
104	Cover Page Interactive Data File (formatted as Inline XBRL).

SIGNATURES

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

CATALYST BIOSCIENCES, INC.

Date: September 7, 2021

/s/ Clinton Musil
Clinton Musil
Chief Financial Officer

CATALYST BIOSCIENCES

Corporate Overview

7 September 2021

CatalystBiosciences.com

Forward looking statements

Certain information contained in this presentation and statements made orally during this presentation include forward-looking statemen
substantial risks and uncertainties. All statements included in this presentation, other than statements of historical facts, are forward-
looking statements. Forward-looking statements include, without limitation, statements about the product candidates of Catalyst Biosciei
“Company”) and the benefits of its protease engineering platform, potential commercial opportunities for and advantages of MarzAA and
potential to treat hemophilia subcutaneously; plans to enroll the Crimson 1 Phase 3 registration study and report on actions of the DSME
bleed data for this study; plans to enroll the MAA Phase 1/2 study of MarzAA and report PK and treatment of bleed data for this study; th
and advantages of the Company's complement product candidates, including CB 2782-PEG as a potential best-in-class C3 degrader fo
a potential treatment for CFI deficiency, and complement degraders; plans for the Company's collaboration with Biogen; potential marke
CFI complement product candidates, and plans to enroll the CB 4332 observational trial and to conduct human clinical trials for CB 4332

Actual results or events could differ materially from the plans, intentions, expectations and projections disclosed in the forward-looking s
Various important factors could cause actual results or events to differ materially, including, but not limited to, the risk that trials, studies
delayed or terminated as a result of COVID-19 and other factors, that trials may not have satisfactory outcomes, that human trials will nc
from earlier trials, that the Company will need to raise additional capital, which may not be available on favorable terms, if at all, the risk
develop or manufacture the Company’s products will be higher than anticipated, including as a result of delays in development and man
from COVID-19 and other factors, the risk that Biogen will terminate its agreement with the Company, competition and other risks descri
Factors” section of the Company’s Annual Report on Form 10-K filed with the Securities and Exchange Commission ("SEC") on March 4
filed with the SEC on August 5, 2021, and in other filings with the SEC. The forward-looking statements in this presentation represent th
of the date of this presentation and the Company does not assume any obligation to update any forward-looking statements, except as r

CATALYST BIOSCIENCES

The Protease Medicines Company

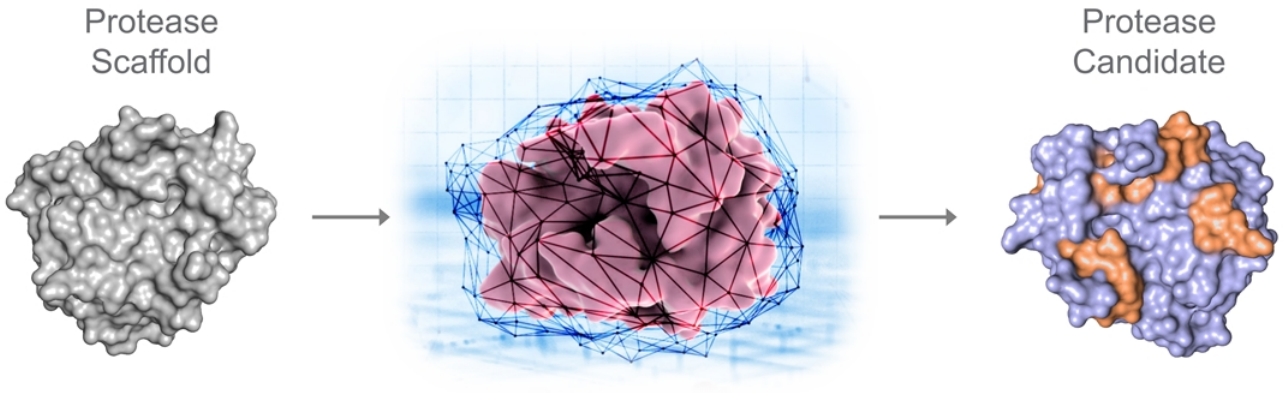
Harnessing the catalytic power of proteases

- Novel differentiated medicines
- Robust complement portfolio
- Clinical-stage assets
- Unique expertise in protease engineering

Catalyst protease platform

Unique expertise enables design of optimized & differentiated proteas

Discovery Platform



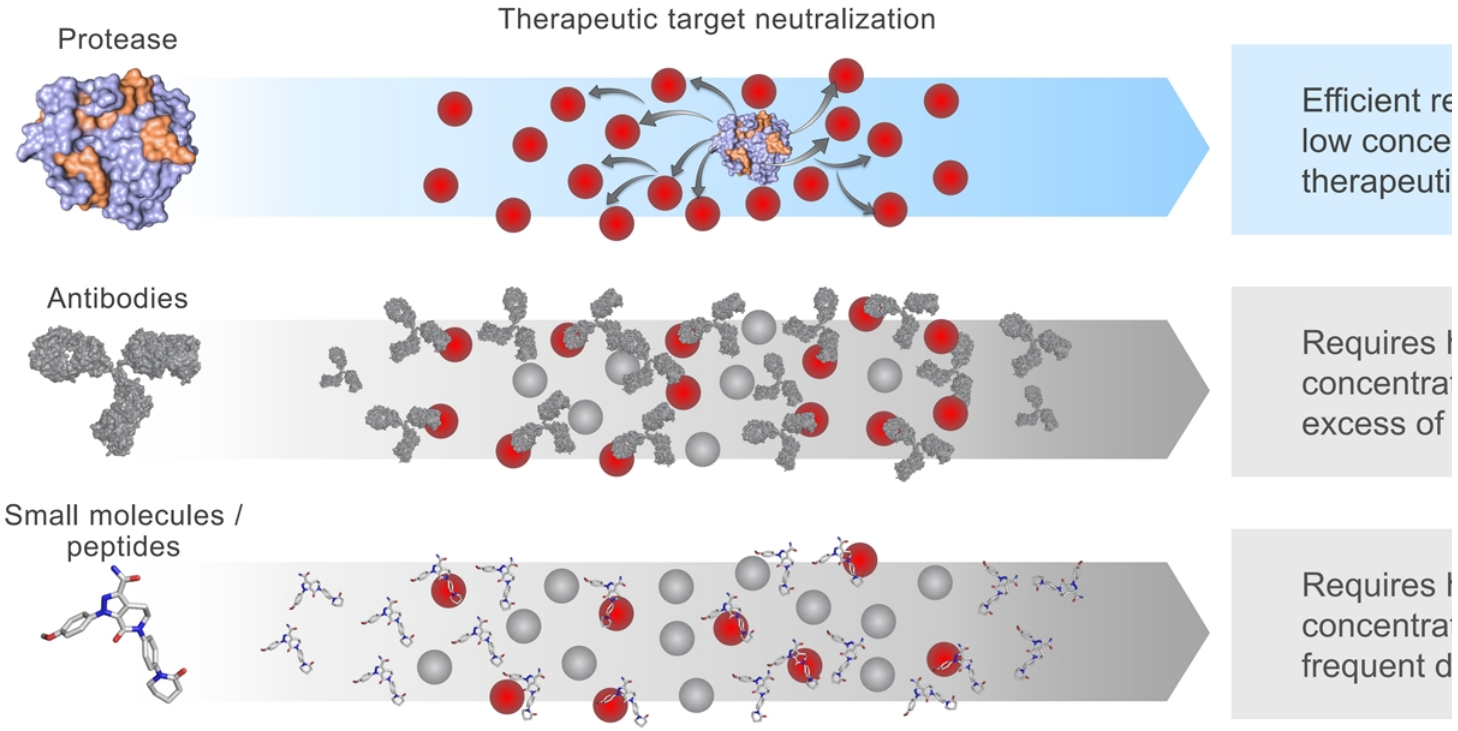
- Structure Guided Design
- Engineered Regulation
- Molecular Evolution
- Pharmacokinetic Improvement

Our Pr

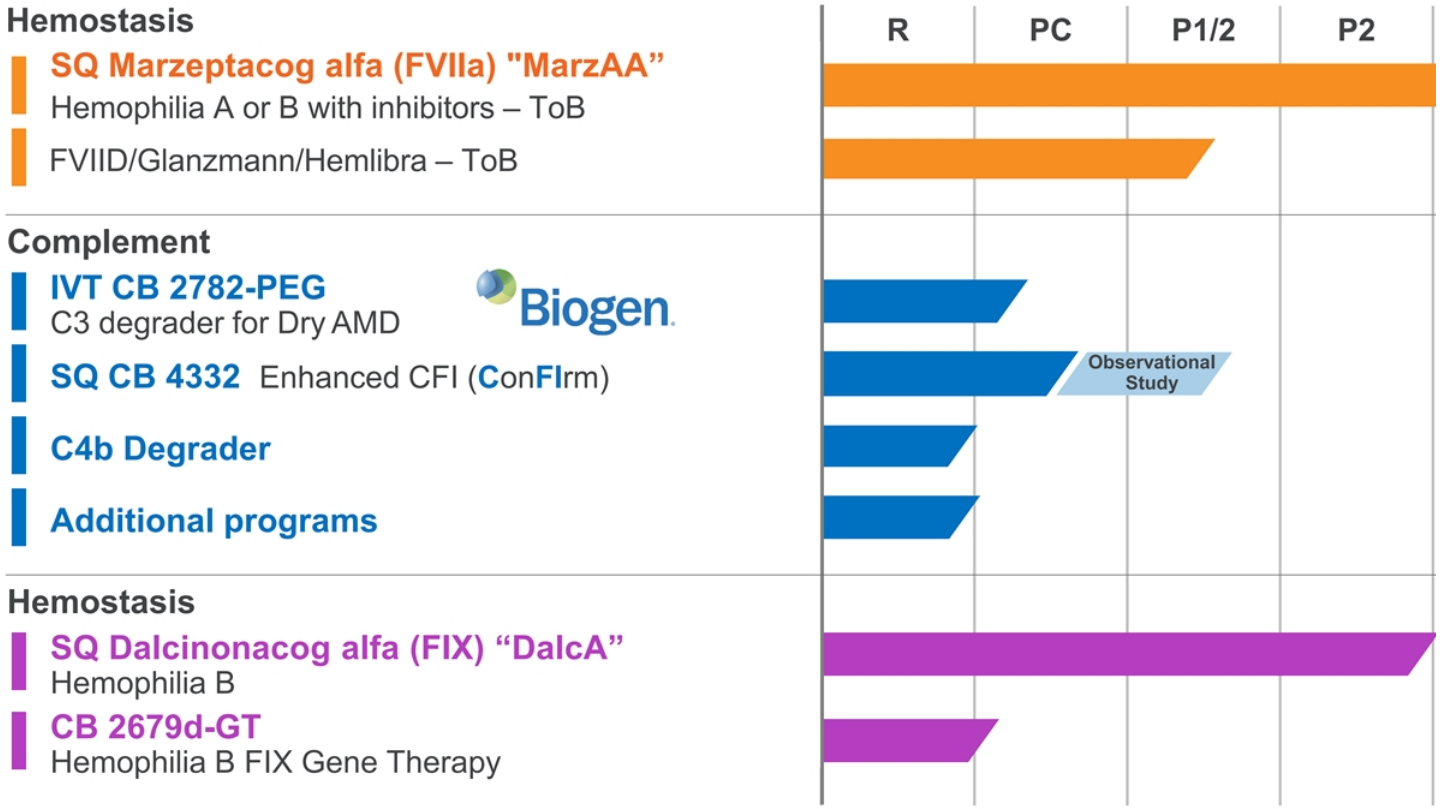
+ Fun
nat
the
coa
+ Eng
prot
the
cas
+ Moc
biol
or ir

Proteases are ideal for high abundancy targets & cascad

A better way to regulate biological processes compared with antibodies & s



Pipeline

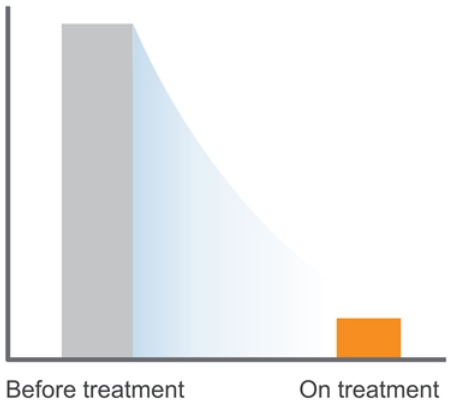


Catalyst protease platform

Validated across three programs

Marzeptacog alfa (activated)

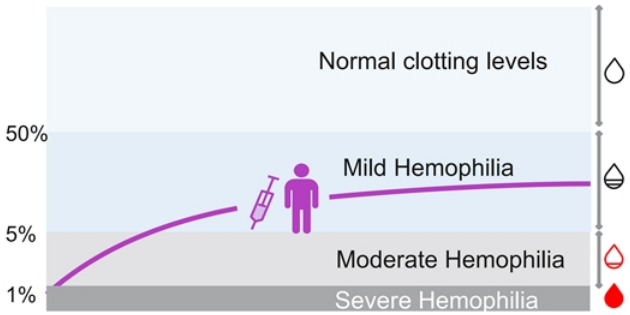
90% reduction in annualized bleed rate



Engineered rFVIIa protease

Dalcinonacog alfa

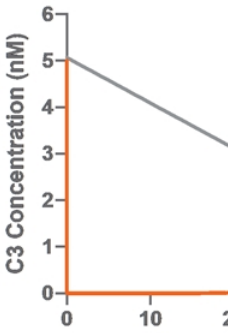
Achieved sustained & high target levels of FIX



Engineered rFIX protease

CB 2782-F

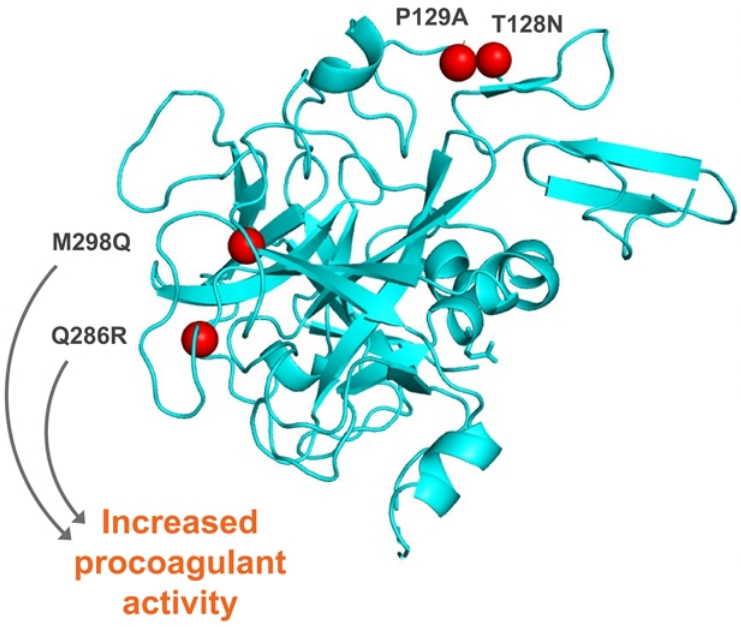
Best-in-class
Extended ph



Nove

Marzeptacog alfa (activated) – MarzAA: SQ rFVIIa

Designed to address a clear unmet need in hemophilia & other bleeding disc



9-fold higher activity *vs* NovoSeven

+ Potency allows for SQ dosing that prolong
+ NovoSeven RT is administered IV

Preclinical efficacy of SQ episodic T

+ HA mouse after tail cut; HA dog; HA rat

P2 proof of concept & preliminary sa with inhibitors – prophylactic ToB

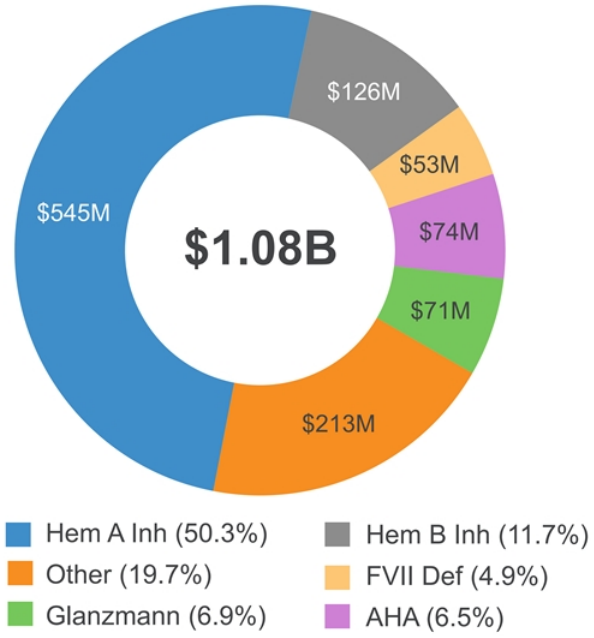
+ 46 patients treated including: single dose 3 SQ doses/day, & daily SQ up to 97 days

FDA Fast Track designations

+ HA/HB with inhibitors, episodic ToB
+ FVIID, episodic ToB

SQ MarzAA is a large commercial opportunity

Global NovoSeven sales breakdown by indication (2020)

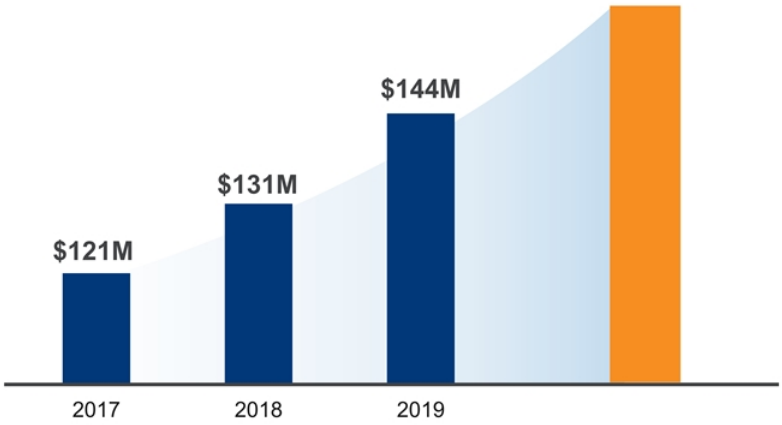


SQ MarzAA profile

+ SQ is patient-preferred & eliminate to fast & effective treatment
+ Ideal for pediatrics & patients with access issues
+ Long half-life without high Cmax fo control of bleeds
+ *In vitro* data support combination v Hemlibra® without increased throm
+ Prophylaxis opportunity demonstra

 Source: Adivo Associates market research; Catalyst Biosciences market research. Data on file.

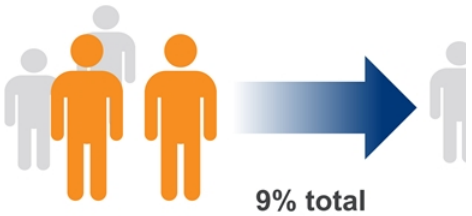
MarzAA could provide SQ prophylaxis for Glanzmann & F



Global NovoSeven on demand sales Glanzmann Thrombasthenia, FVIID

Unmet need in prophylaxis

Growing number of Glanzr Thrombasthenia and FVIID patients treated with Novo



9% total increase in patients over 3 years

Source: Catalyst Biosciences, Adivo Associates Market Research, Data on file. *Note: 2019 estimates Treated patients may be counted multiple patients may have multiple bleeding events per year needing factor treatment

Unmet need for a long-acting SQ episodic treatment of blee

NovoSeven

- Patients reported needing an average of **6 hours and 3 infusions** of NovoSeven to resolve bleeds
- Some bleeds take longer than 72 hours to resolve[1,2,3]

Current bypass agents require multiple infusions over the course of hours

MarzAA

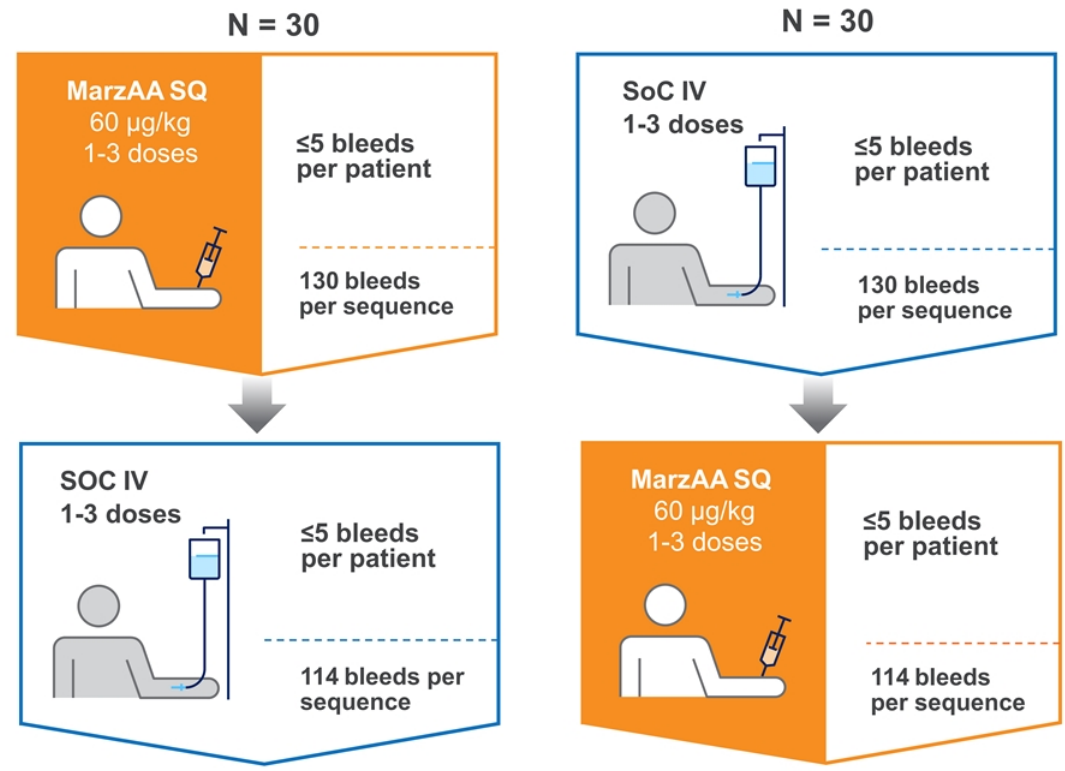
- MAA-102: PK MarzAA levels support SQ ToB
- Target therapeutic levels are **rapidly achieved**
- Target levels can be maintaine 18 hours with a single SQ dos 60 µg/kg

Clinical MarzAA levels support S

Source: [1]NovoSeven PI Rev 7/2020; [2]Adivo Associates market research; [3]Catalyst Biosciences' market research; Data on file; Neuman *et al.* IS

Crimson 1 Phase 3 study: Treatment of episodic bleeding

Hemophilia A or B with inhibitors, ABR ≥ 8



Primary endpoi

+ Non-inferior hem
standard 4-point

Secondary end

+ Time to bleed re
number of doses

Safety

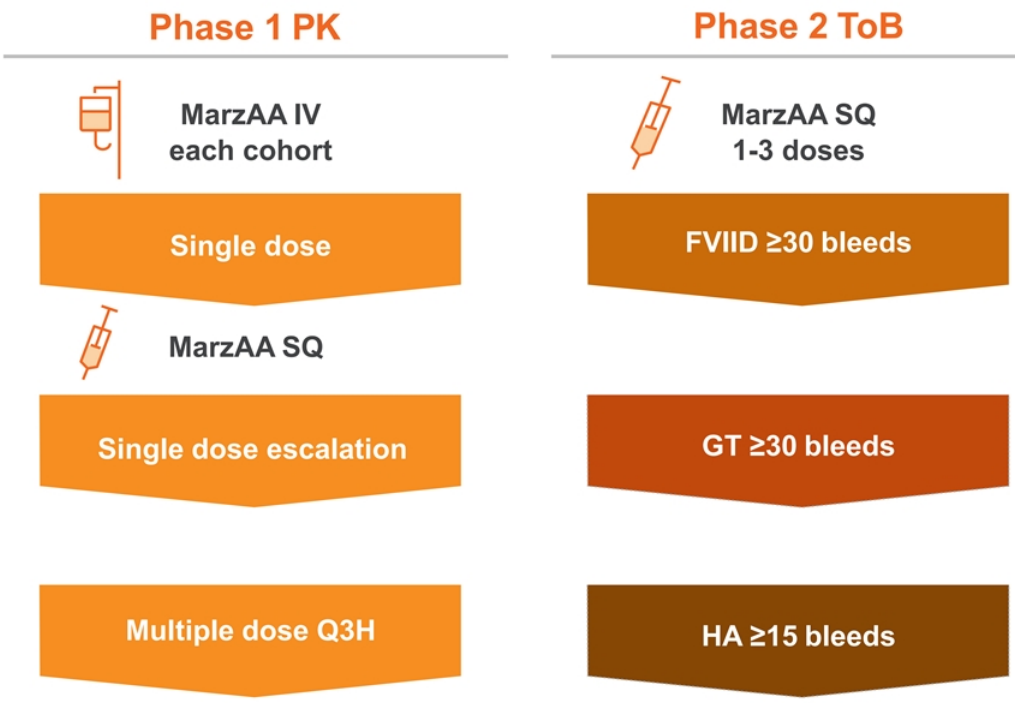
+ Adverse events,
antibodies (ADA

Statistics

+ **SoC estimate 85**
treatment of blee
+ Non-inferiority m
+ **2.5%** significanc
+ **90%** power

MAA-202 Phase 1/2 study design

FVII deficiency, Glanzmann Thrombasthenia and HA on Hemlibra: N =



Phase 1

+ **Primary endpoi**
  Pharmacokinetic
+ **Secondary endp**
  Pharmacodynam

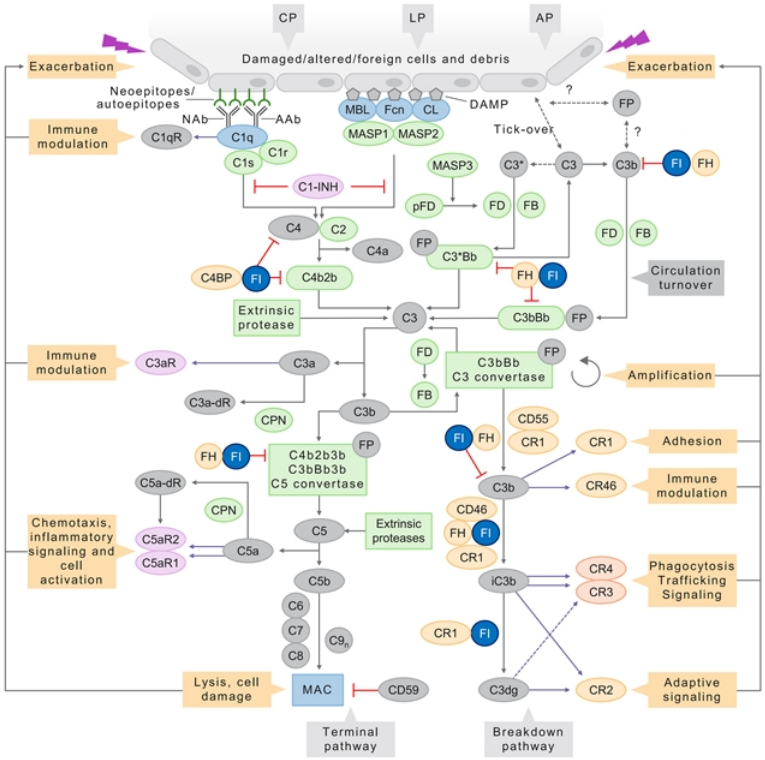
Phase 2 ToB

+ **Primary endpoi**
  Hemostatic effica
+ **Secondary endp**
  Effective hemost
  timepoints; dose
  rescue meds
+ **Safety:**
  Adverse events a

Growing Complement Pathway Protease Platform

Complement is a perfect fit to develop protease therapeu

The complement pathway is driven by a protease cascade

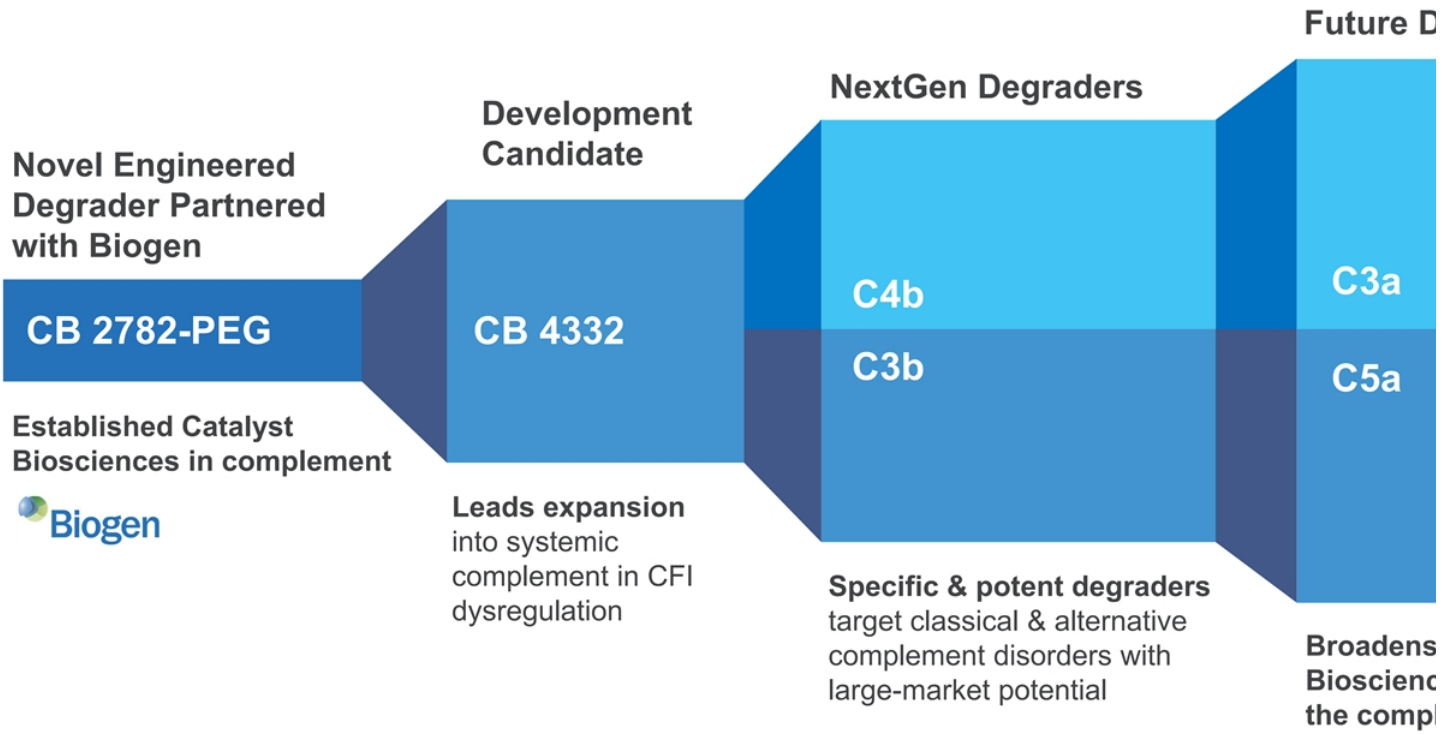


80

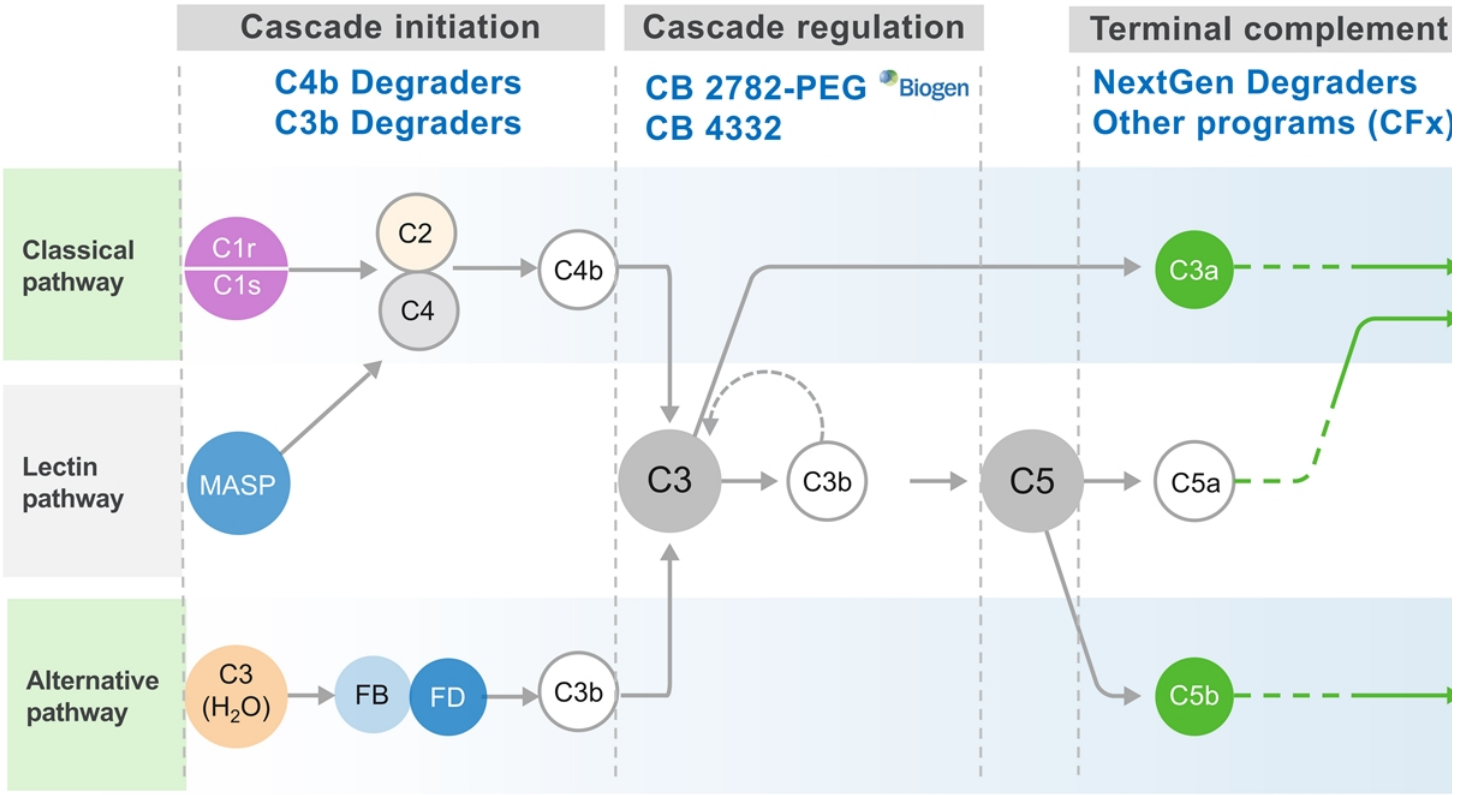
of the comple cascade is reg by proteas

 Source: Figure adapted from Mastellos *et al.*, Clinical promise of next-generation complement therapeutics. Nature Reviews. 2019

Multiple, high-value complement programs



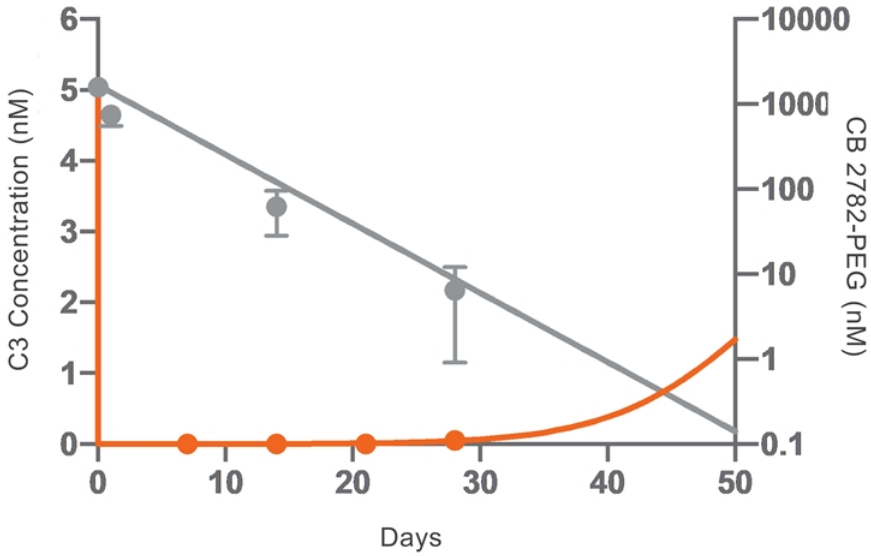
Unique targeted approach to complement regulation



CB 2782-PEG: Best-in-class C3 degrader for dry AMD

Protease advantage demonstrated *in vivo*

CB 2782-PEG degrades C3 levels in the eye for at least 28 days in a non-human primate model



Catalytic advantage

+ One therapeutic mole neutralizes 1000s
+ Fast & potent respons
+ Extended pharmacod
+ Can activate or degra therapeutic targets
+ Engineered novel pro degraders “sweep aw to drug targets

CB 2782-PEG: Long acting anti-C3 protease for dry AMD

Geographic atrophy is a high unmet need

+ Advanced stage of dry age-related macular degeneration (dAMD)
+ dAMD affects ~1M people in the US & >5M WW, no currently approved therapy
+ Global market ~ >$5B
+ C3 is a clinically validated target (randomized P2) for dAMD

Best-in-class C3 degrader for dry AMD

+ Generated from Catalyst's proprietary **protease engineering platform**
+ Potent, selective & long acting, degrades C3 into inactive fragments
+ NHP PK & PD data* predict **best-in-class** human intravitreal **dosing 3 or 4 times a year**

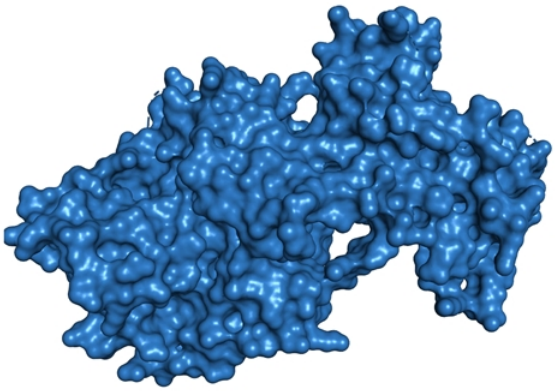
Biogen collaboratior

+ $15M upfront milestones & to low double
+ Catalyst: fully & manufactur
+ Biogen: IND-e WW clinical d commercializ

 *Furfine *et al*. ARVO 2019

CB 4332: SQ Enhanced Complement Factor I

Development candidate to restore regulation



+ **Engineered for an extended half-life**
  + Once weekly SQ therapy – no PEG
+ ***In vitro & ex vivo* activity comparable to native CFI**
  + Classical & alternative pathway regulation
+ **High yield production process**

Rationale & unme

+ **Rebalance the co**
  system in patients
  dysregulated CFI
+ **No specific therap**
  correct CFI dysregu
+ Targets population
  treatment or who
  poorly to current

[1]Bienaime *et al*. Kidney Int. 2010; [2]Ferreira *et al*. Nefrologia. 2016; Note: CFI = Complement factor I; Structural model based on PDB 2XRC.

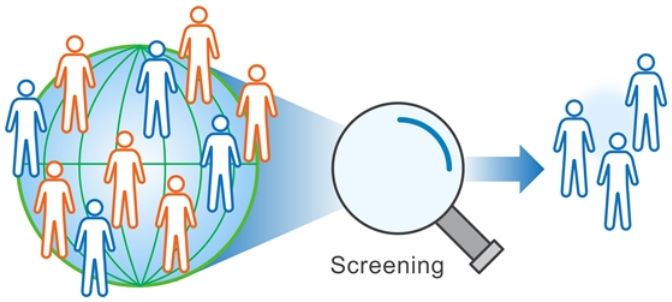
CB 4332: To address CFI deficiency at the root cause

Designed to provide unique advantages

Unmet needs in CFI deficiency	CB 4332 Designed to address
Blocks complement-initiated cell destruction in the circulation	✓
Directly addresses root cause of disease	✓
Addresses extravascular hemolysis	✓
Preserves normal immune functions, *e.g.* to fight off infections	✓
Convenient weekly SQ administration	✓

Screening & natural history of disease studies

ConFIrm & ConFIdence: preparing for Phase 1/2



Identifies Target Population / Feeds **ConFIdenc**
Study / Discovers Undiagnosed Disease

ConFIrm study

ConFIdence study

Prospective Clinical & Biomarkers Asse
of CFI-Deficiency Disease While on So

- **Identification of CFI-deficient patients & key investigators for CB 4332 trials**
- **Discover undiagnosed disease, create program awareness & inform on bion**

CB 4332: Phase 1/2 – First in human study

Study parts

Single Ascending Doses
(N = up to 12)

Multiple Ascending Doses
(N = up to 9)

Extended treatment to assess proof of concept
(N = up to 15)

Study design

+ Phase 1 open-label, single & multiple ascendin & extended duration proof of concept
+ Population: CFI-deficient patients

Proposed starting dose

+ 0.5 mg/Kg

Goals

+ Safety & tolerability
+ PK characterization
+ Assessment of complement biomarkers (C3, FI Bb/FB ratio, iC3b, C3d, C3dg, AP50/AH50)
+ Establish a Recommended Dose Regimen with the CFI normal range

Diseases with CFI mutations have tremendous potential

US / EU5 market opportunity

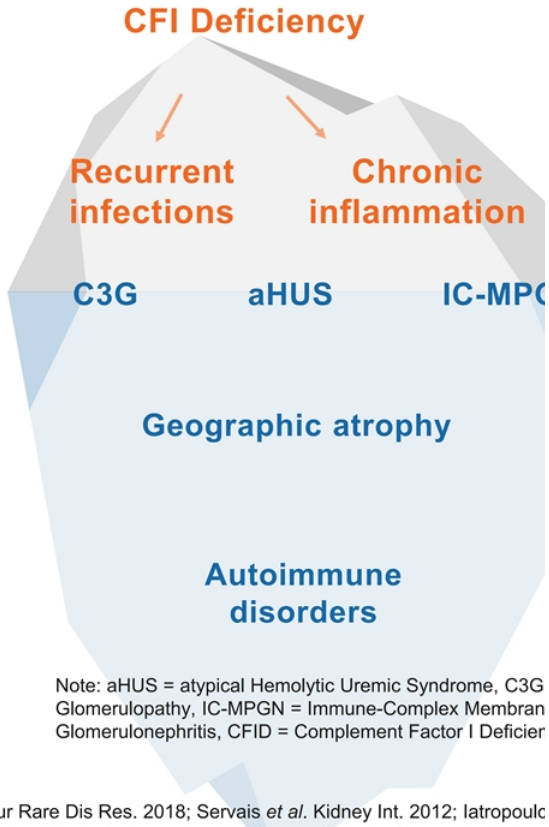
CFI Deficiency
First indication

$500M+
Market opportunity in CFI deficient populations

0 Specific systemic therapies in development for patients with dysregulated CFI

0 Therapies addressing the root cause of disease

0 Approved treatments for C3G, IC-MPGN, CFID

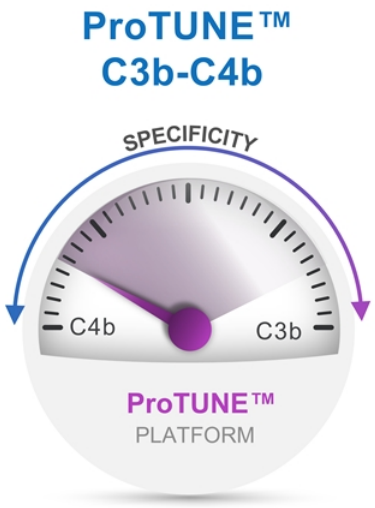


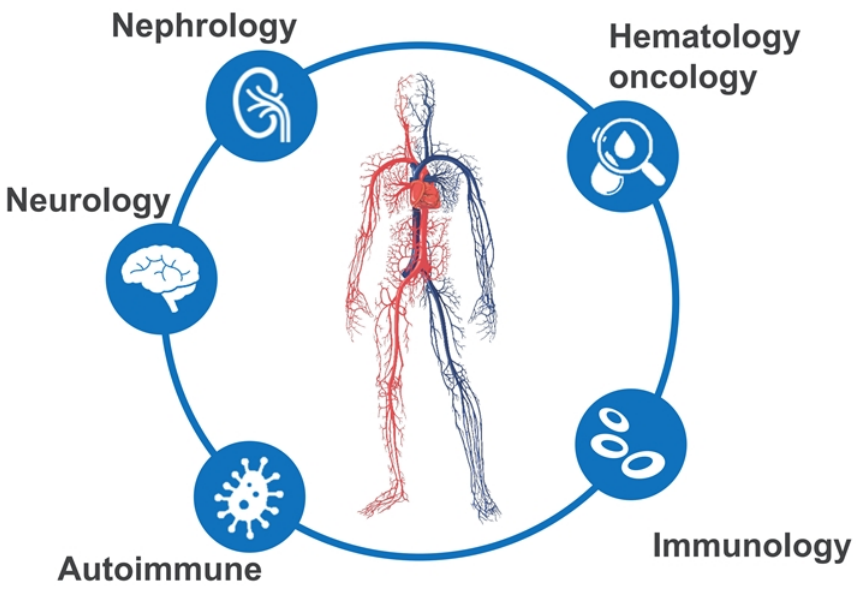
Note: aHUS = atypical Hemolytic Uremic Syndrome, C3G Glomerulopathy, IC-MPGN = Immune-Complex Membran Glomerulonephritis, CFID = Complement Factor I Deficier

Bresin *et al*. JASN. 2013; Fremeaux-Bacchi *et al*. ASN. 2013; Rui-Ru *et al*. Jour Rare Dis Res. 2018; Servais *et al*. Kidney Int. 2012; Iatropoulc Mol Immunol. 2016; Hou *et al*. Kidney Int. 2014; Alba-Domiguez *et al*. J rare Dis. 2012. El Sissy *et al*. Front. Immunol. 2019; Shields *et al*. Fron 2019; Naesens *et al*. Jour Allergy & Clin Immunol. 2020; Yan *et al*. Clin Epi 2020; Smith *et al*. Nature Reviews. 2019; Noris *et al*. Clin J Am So 2010; CBIO KOL interviews

Our protease platforms are tailored to specific indications

Tuning functionality to restore complement homeostasis & immunore

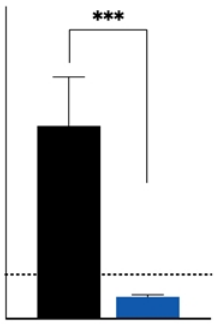


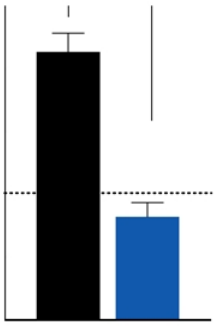


C3b-C4b degraders significantly reduce inflammation *in*

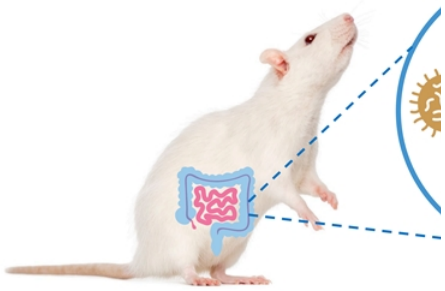
Significantly decrease in inflammatory markers involved in IgA nephr

Inflammatory markers in IgA nephropathy





Rat model of complement-media

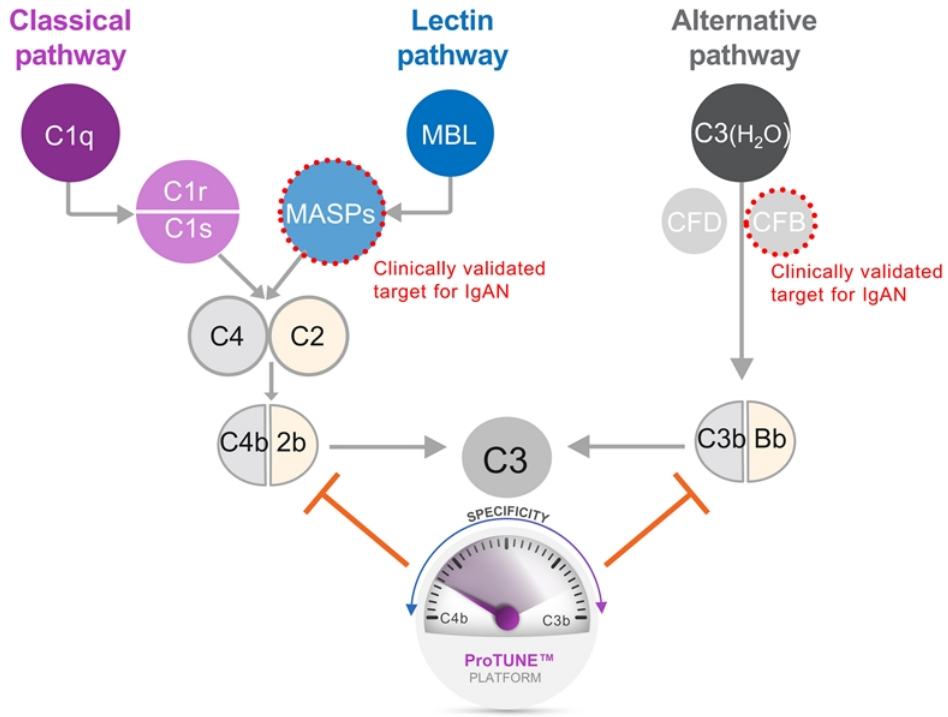


Reduction of **IFNγ** & **TNFα** involved in kidney damage & proteinuria in IgA nephropat

1. Yano, N. *et al.* Phenotypic Characterization of Cytokine Expression in Patients With IgA Nephropathy. *J Clin Immunol* **17**, 396–402 (1997). 2
Th1/Th2 predominance and proinflammatory cytokines determine the clinicopathological severity of IgA nephropathy. *Nephrol Dial Transpl* **16**, 2
Values are mean +/- SEM, ***p<0.001 using One Way or Two-way ANOVA.

C3b-C4b degraders for IgA nephropathy patients

Dual targeting of alternate & lectin pathways



Differentiation

+ Dual targeting mode of a alternative pathways

Rationale for IgA neph

+ Both lectin **&** alternative involved in IgA nephropa with severe clinical mani

Clinically validated tar

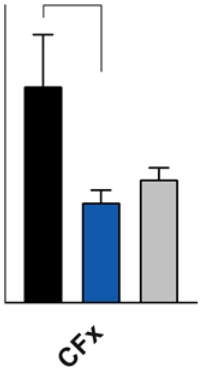
+ Inhibition of only MASP2 be insufficient to reduce nephropathy patients

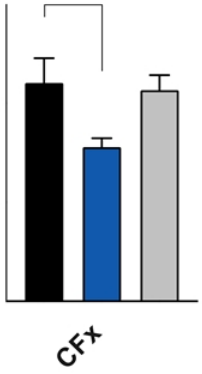
1. Medjeral-Thomas *et al*. Kidney International Reports (2018); 2. Bi *et al*. BMC Nephrology (2019); 3. Roos *et al*. J Am Soc Nephrol (2006)

C3a-C5a degraders: Efficacy in an acute LPS-induced ARD

CFx improves respiratory function & reduces cell infiltrates

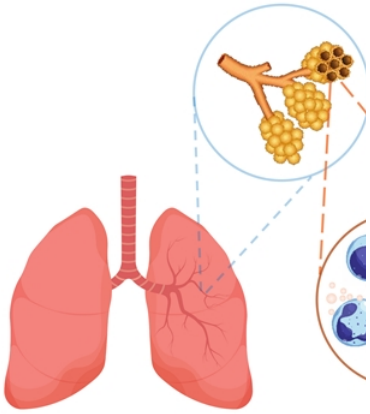
Respiratory functions & cell infiltration at 24 h





CFx 7-fold lower dose vs. anti-C5

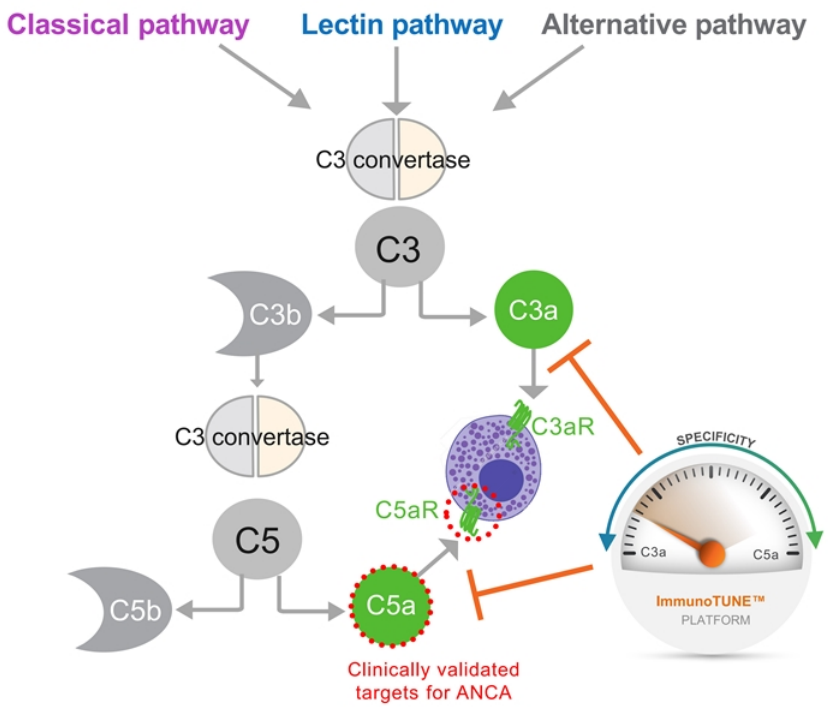
Mouse LPS model of lung



- CFx **outperforms** anti-C5 antibody in reducing inflammatory cell infiltration
- CFx **compares well** on respiratory functions with anti-C5 antibody

C3a-C5a degraders: Potential for ANCA-AAV patients

Dual targeting of both C3a & C5a with one protease medicine



Differentiation

+ Degrade activation products C5 (C5a) that are inflammat
+ May provide beneficial functi **C5L2** pathway

Rationale for ANCA-AAV

+ Both C3a **&** C5a are higher i patients[1, 2]

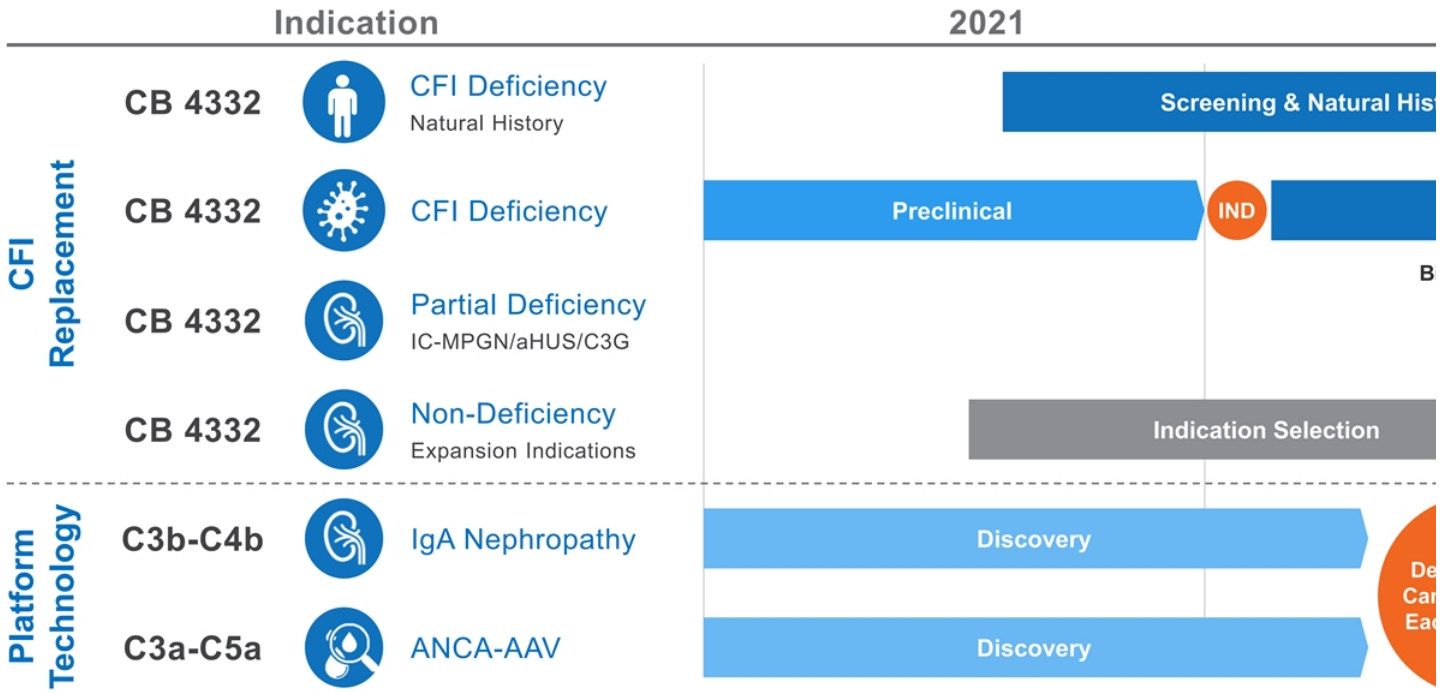
Clinically validated target

+ Inhibition of C5a or C5aR ma insufficient to increase remis ANCA-AAV patients

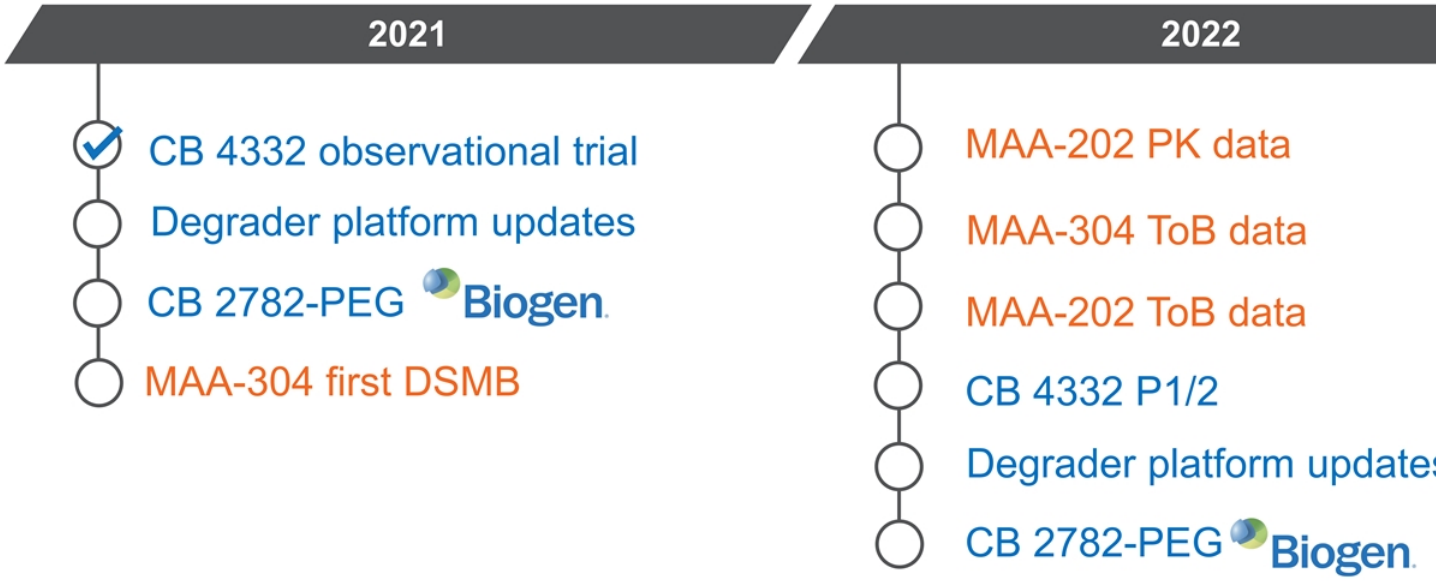
1. S. Moiseev *et al*. British Society for Immunology, Clinical and Experimental Immunology (2020); 2. Gou *et al*. Kidney International (2012).

CB 4332 spearheads a deep pipeline in complement

IND & next development candidate in 2022



Milestones



MarzAA (FVIIa) **CB 2782-PEG** (dAMD) **Systemic complement**

THANK YOU

Nasdaq: CBIO

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