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): December 14, 2020

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 December 14, 2020, Catalyst Biosciences, Inc. (the "Company") gave a presentation on its complement programs and first subcutaneously-dosed systemic complement development candidate (the Complement Presentation") at the Company's Research & Development Call on Systemic Complement Regulator Programs. In addition, the Company posted an update to its corporate presentation (the "Corporate Presentation") on its website, ir.catalystbiosciences.com/presentations-events. A copy of the Complement Presentation is attached hereto as Exhibit 99.1 and a copy of the Corporate Presentation is attached hereto as Exhibit 99.2.

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	Complement Presentation slide deck.
99.2	Corporate 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: December 14, 2020

/s/ Clinton Musil
Clinton Musil
Chief Financial Officer

CATALYST BIOSCIENCES

Complement R&D Day

14 December 2020

CatalystBiosciences.com

Virtual complement R&D day agenda

Overview & KOL introduction

12:00 pm – 12:10 pm ET

Nassim Usman, Ph.D. – President and CEO

- Company vision
- Protease engineering platform overview
- Complement program strategy

12:10 pm – 12:25 pm ET

Ron Taylor, Ph.D. – Professor Emeritus, Biochemistry and Molecular Genetics, University of Virginia School of Medicine

- Complement pathways; the role of proteases
- Diseases associated with uncontrolled complement activation
- Current therapies and unmet needs

Catalyst'

12:25 pm

Grant Blous

- Protea
- CBIO
  - First
  - Pipe

12:45 pm

Clinton Mus

- Milest
- Q&A

Forward looking statements

Certain information contained in this presentation and statements made orally during this presentation include forward-looking statements that involve substantial risks and uncertainties. All statements included in this presentation, other than statement of historical facts, are forward-looking statements. Forward-looking statements include, without limitation, statements about Catalyst's product candidates and the benefits of its protease engineering platform, projected complement market opportunity, solution to fundamental shortcomings in current treatment options, plans to enroll the CB 4332 observational trial in the Company's complement program in mid 2021, and ongoing updates on CB 4322 and the C4b degrader. Actual results or events could differ materially from the plans, intentions, expectations and projections disclosed in the forward-looking statements. Various important factors could cause actual results or events to differ materially, including, but not limited to, the risk that trials and studies may be
factors, that tria
human trials wil
risk that costs r
Company's pro
as a result of de
resulting from C
will terminate C
described in the
Annual Report
Exchange Com
Quarterly Repo
5, 2020, and in
statements in th
views as of the
not assume any
statements, exc

Nassim Usman, Ph.D.

President and CEO

CATALYST BIOSCIENCES

Harnessing the catalytic power o

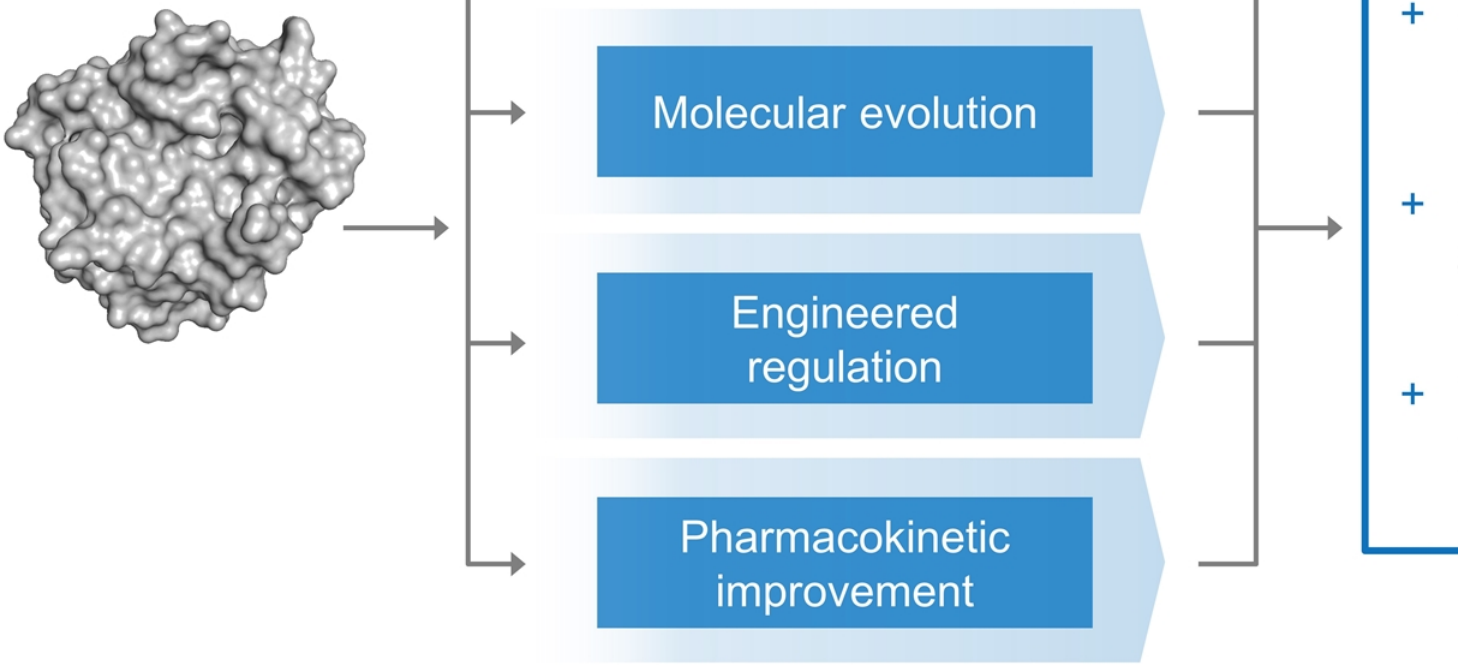
Nature's biological regulators

- Control key biological mechanisms
- Activate or inactivate biological pathways
- Can be tuned for high specificity and functionality
- Deficiencies often cause severe disease

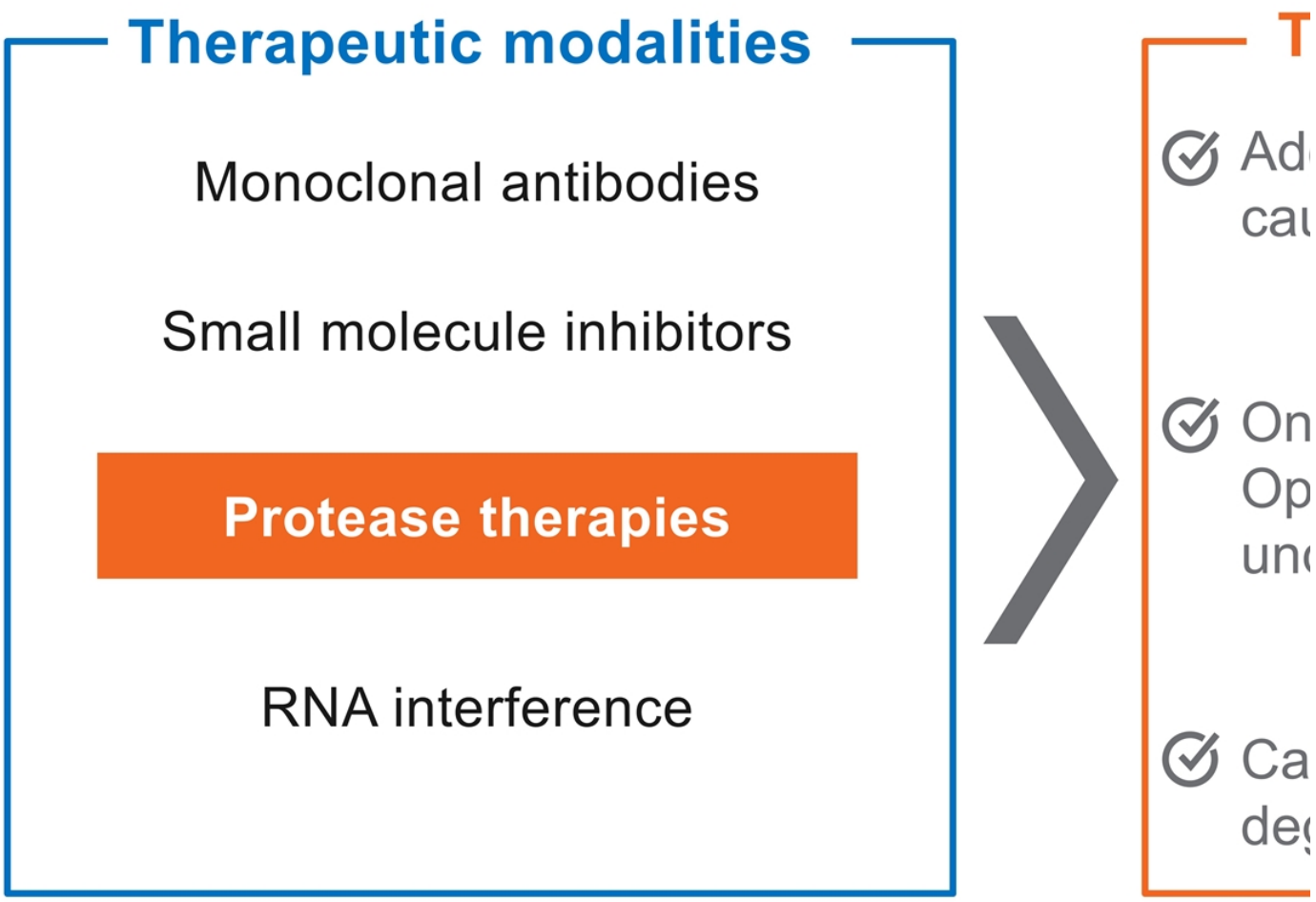
Catalyst's protease platform generates dif

Unique expertise in protease biology enables design of o

Discovery Platform



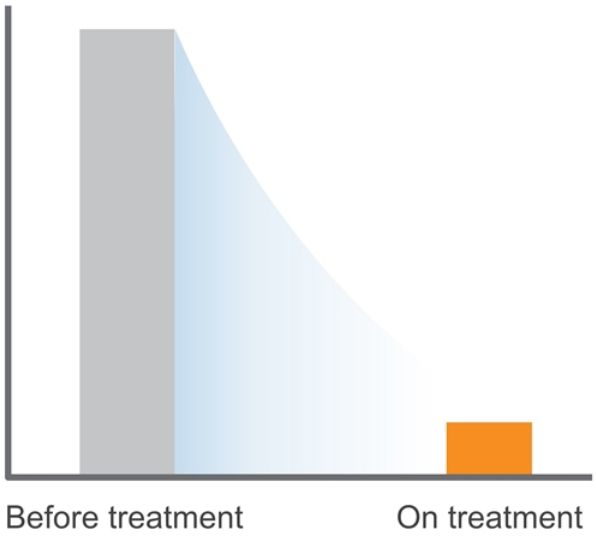
Protease therapeutics



Clinical & partnering success of the CBIO

Marzeptacog alfa (activated)

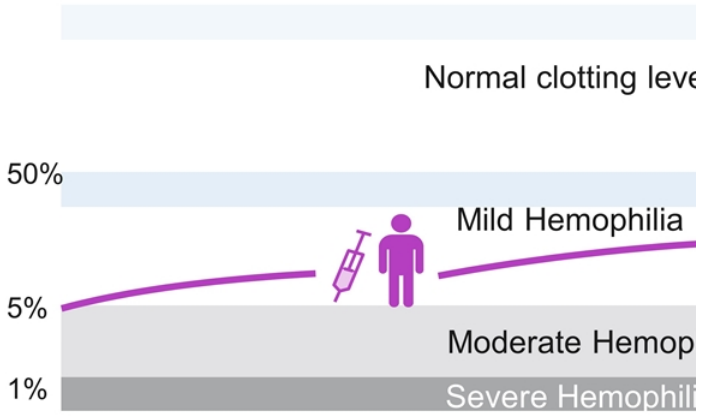
90% reduction
in annualized bleed rate



Engineered
rFVIIa protease

Dalcinonacog alfa

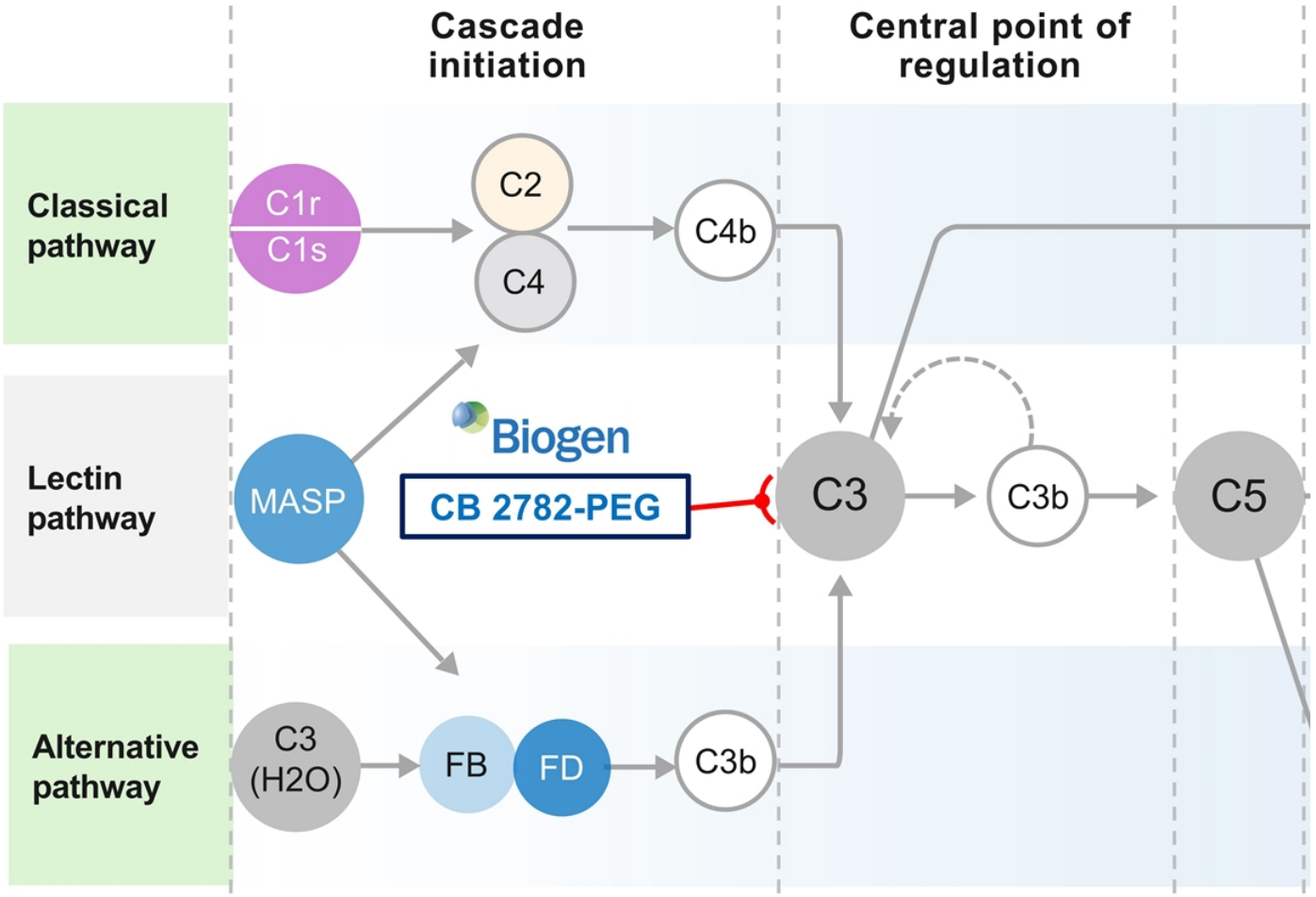
Achieved sustained
& high target levels of FIX



Engineered
rFIX protease

Catalyst is taking a targeted approach to c

80% of the complement system is regulated by pr

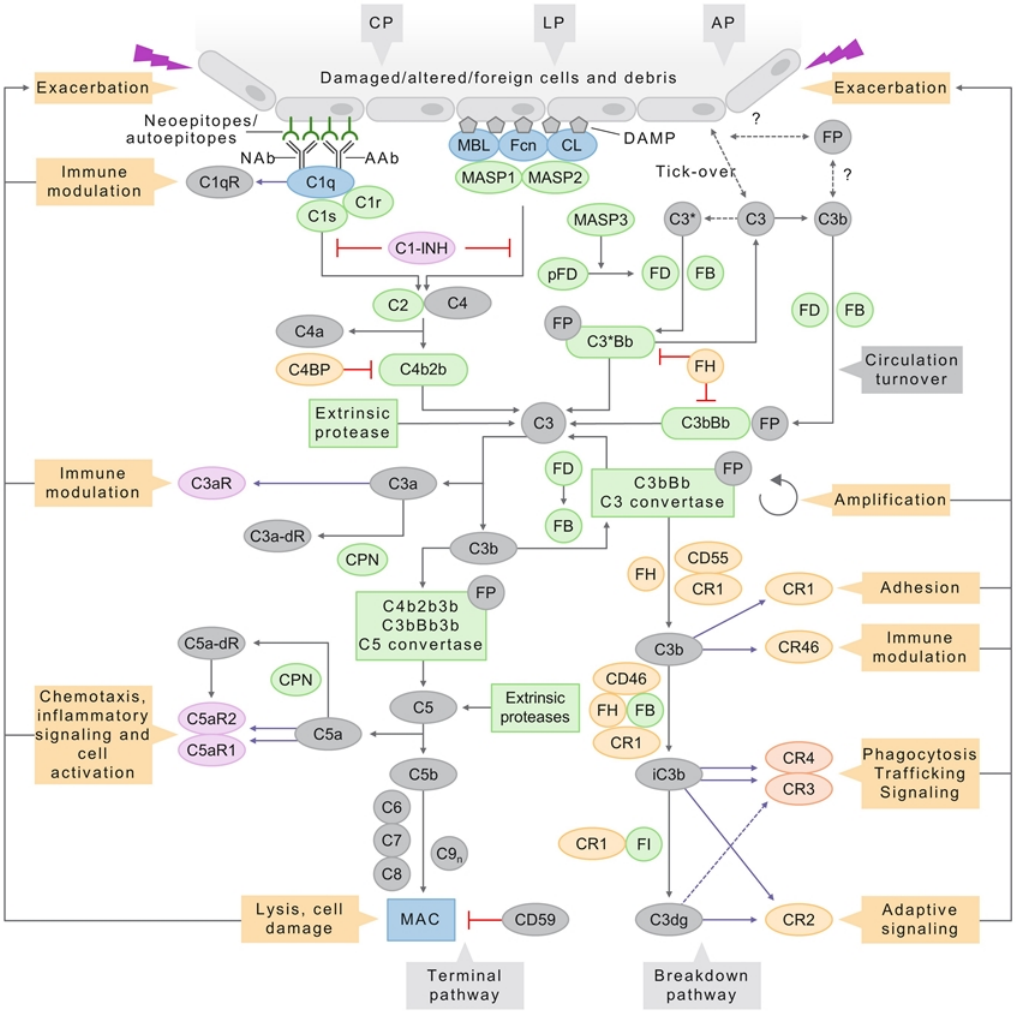


Grant E. Blouse, Ph.D.

SVP, Translational Research

Complement is a perfect fit to develop pro

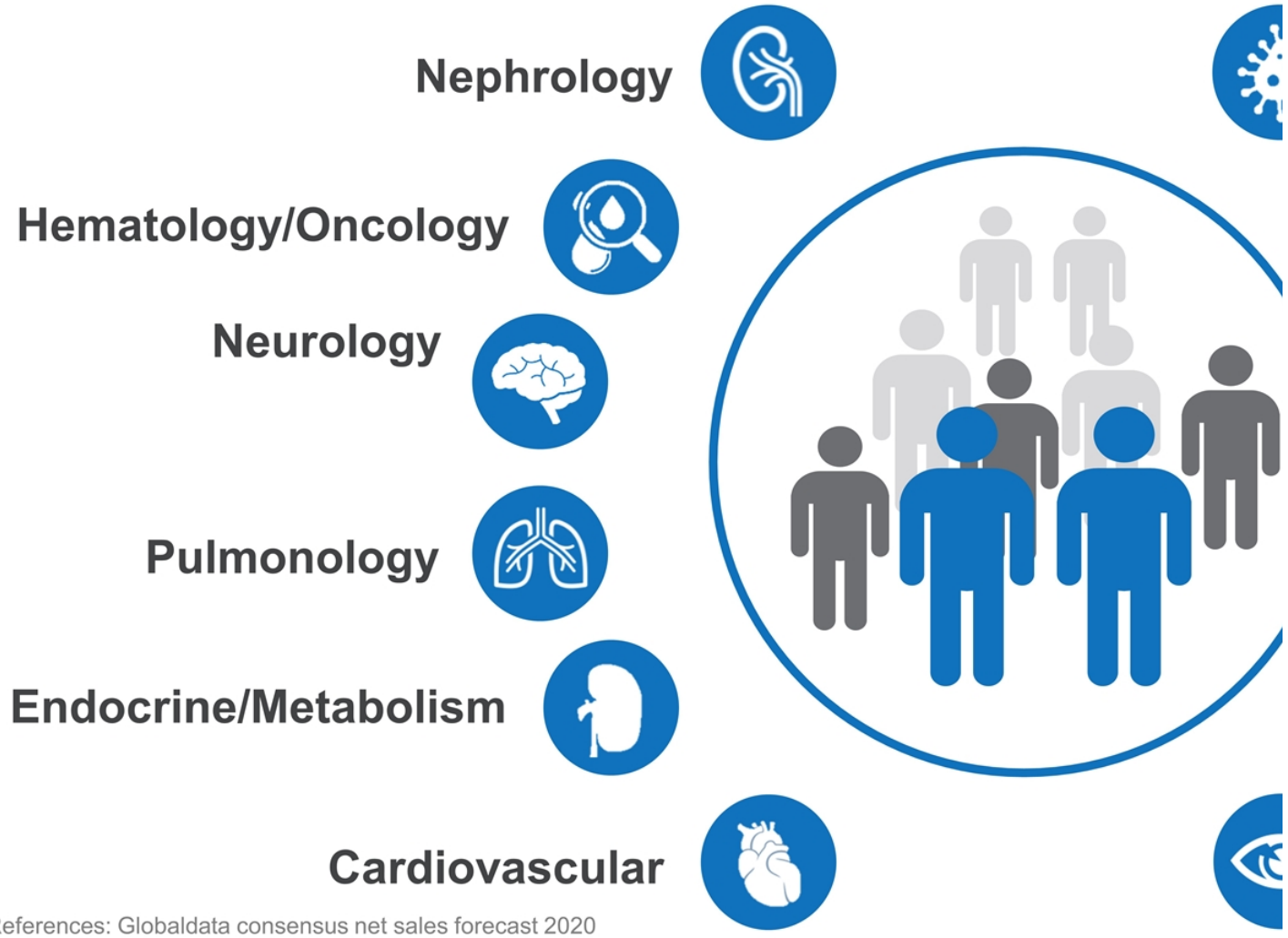
The complement pathway is driven by a protease ca



Reference: Figure adapted from Mastellos et al., Clinical promise of next-generation complement therapeutics. Nat

Complement plays a critical role in many

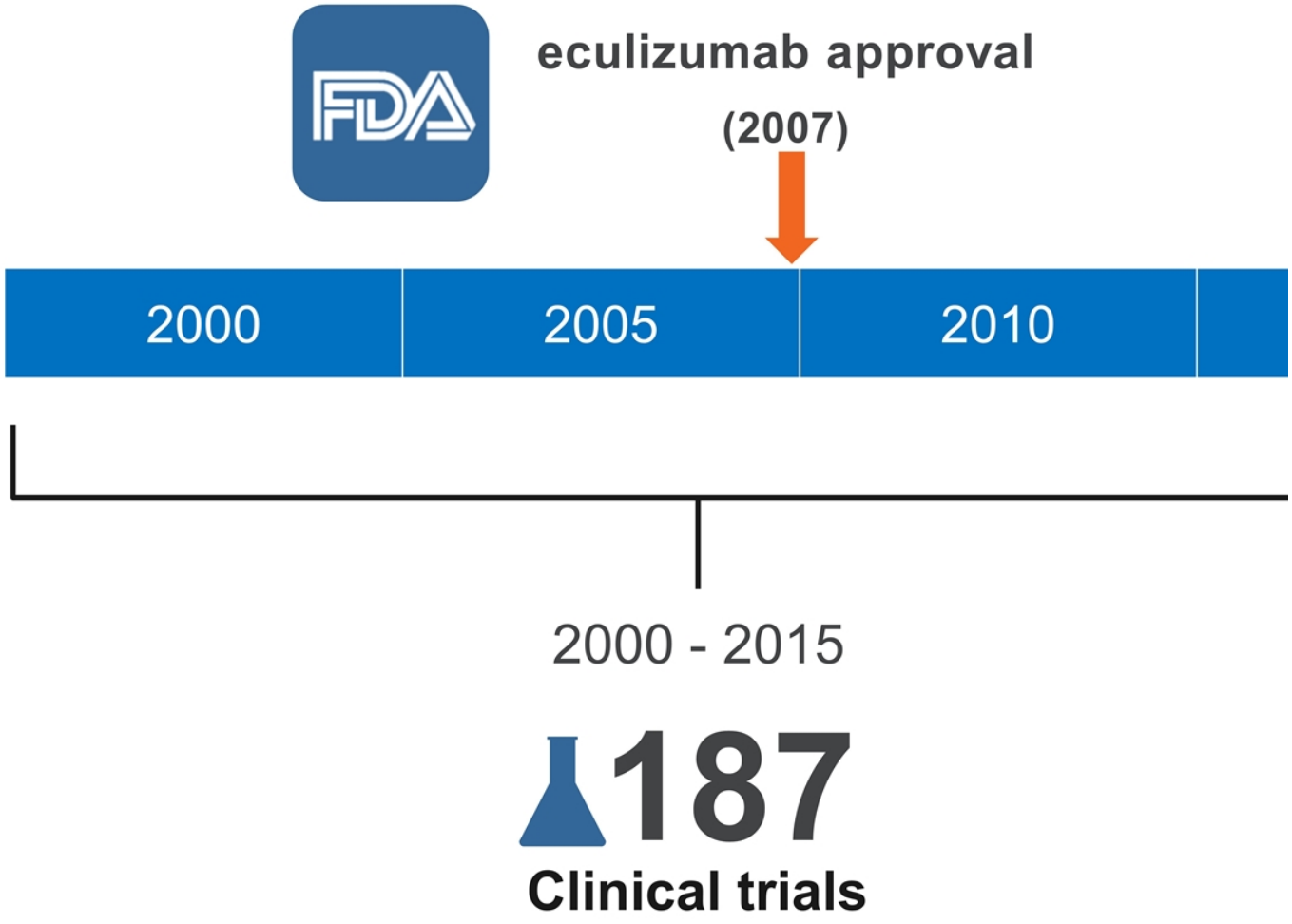
Late-stage complement therapies projected to achieve n



References: Globaldata consensus net sales forecast 2020

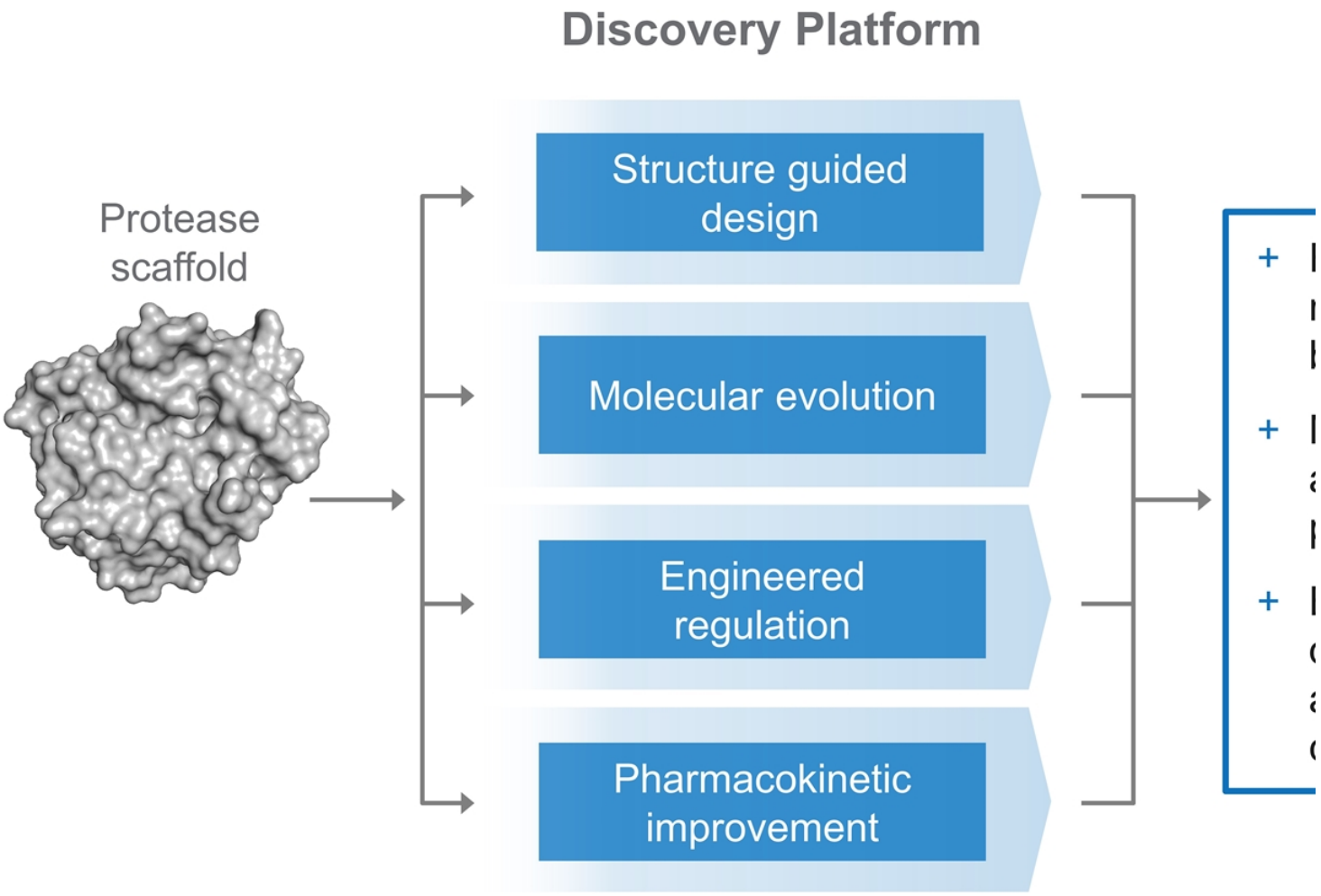
Investment in complement is driven by an

Scientific advancements facilitate increased interest in co



Catalyst's protease platform generates dif

Unique expertise in protease biology enables design of

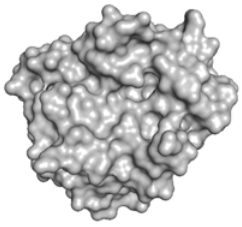


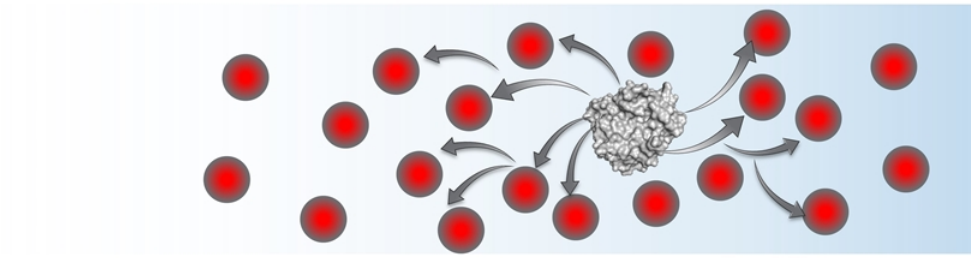
Proteases are ideal for high abundancy ta

A better way to regulate biological processes compared w

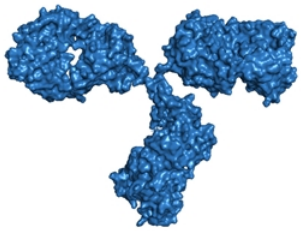
Therapeutic target neutralization

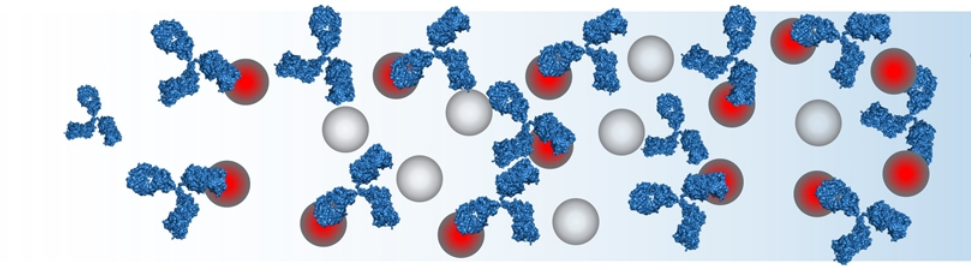
Protease



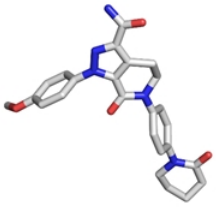


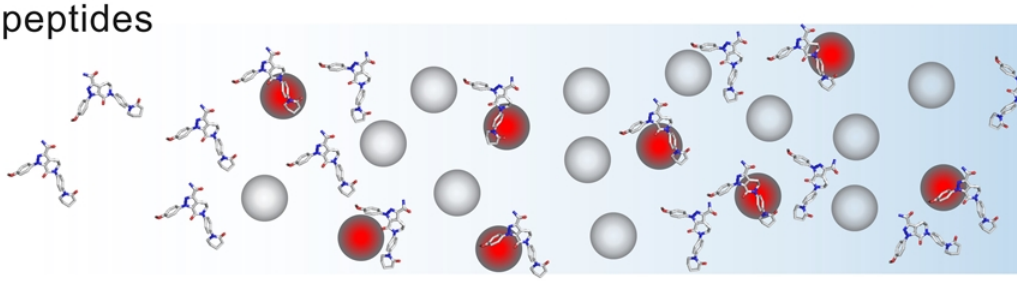
Antibodies





Small molecules / peptides

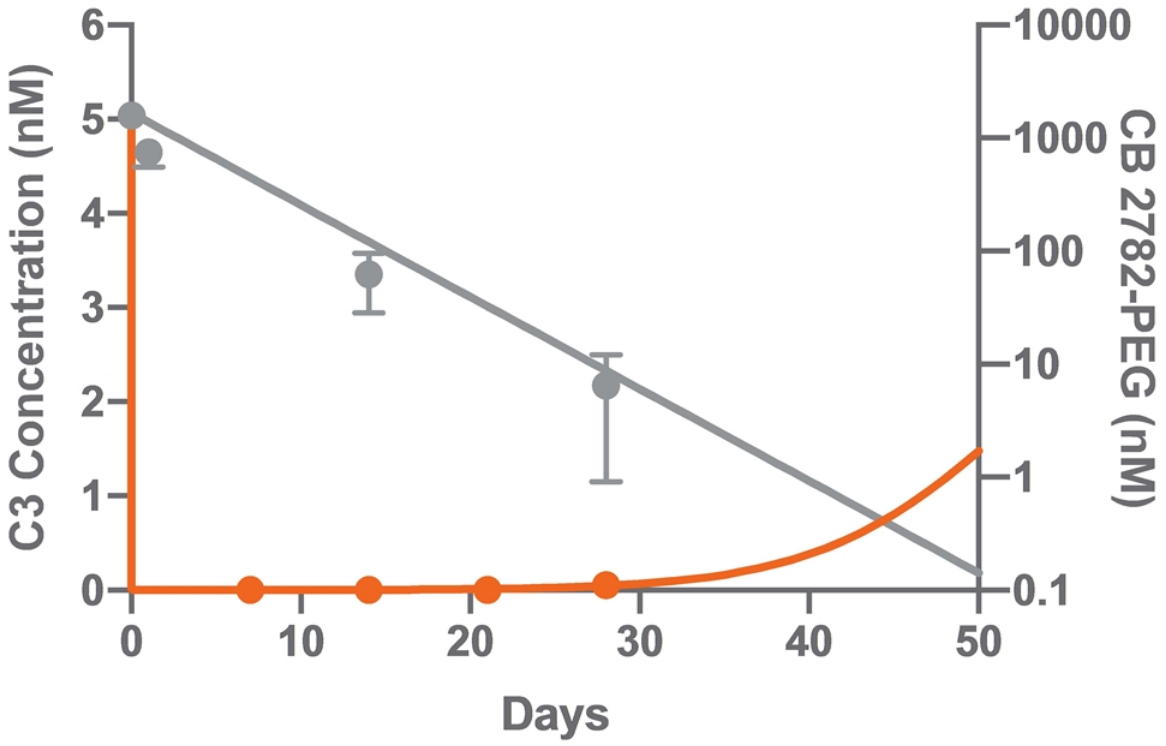




Protease advantage demonstrated *in vivo*

CB 2782-PEG Biogen Designed for a best-in-class anti-

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



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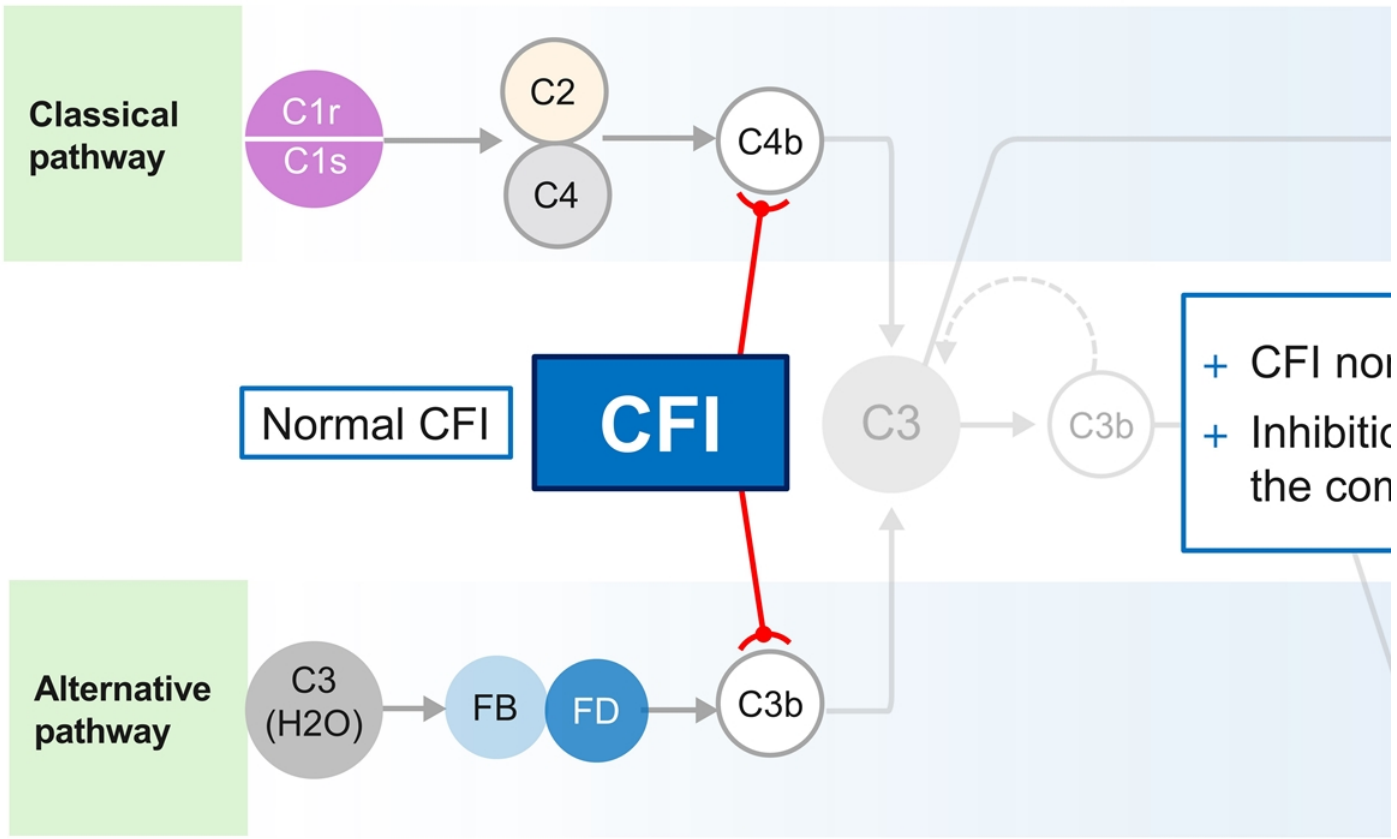
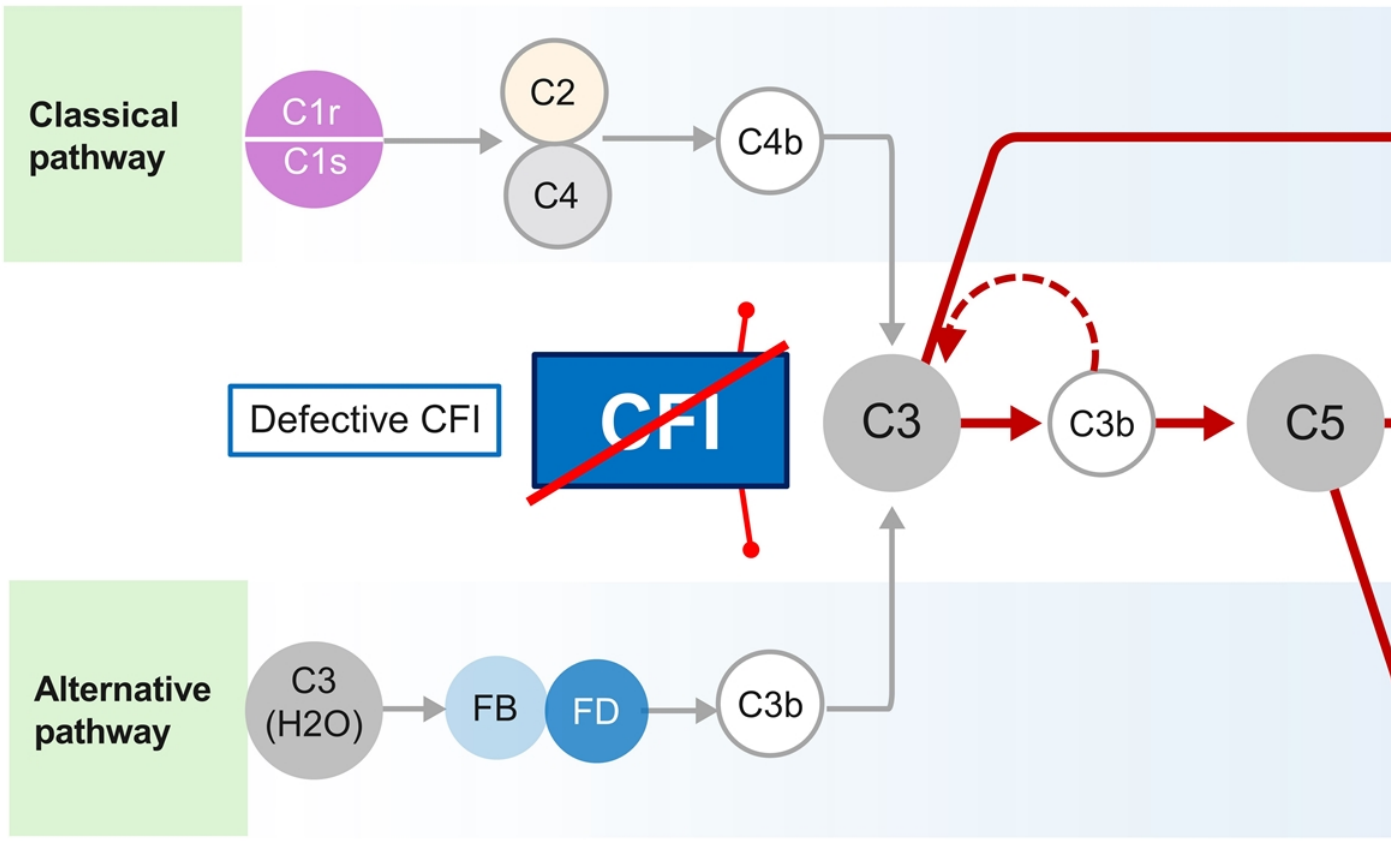
CB 4332: Enhanced Comp

CBIO's Next Development Candidat

Normal CFI: Key central regulator of comp

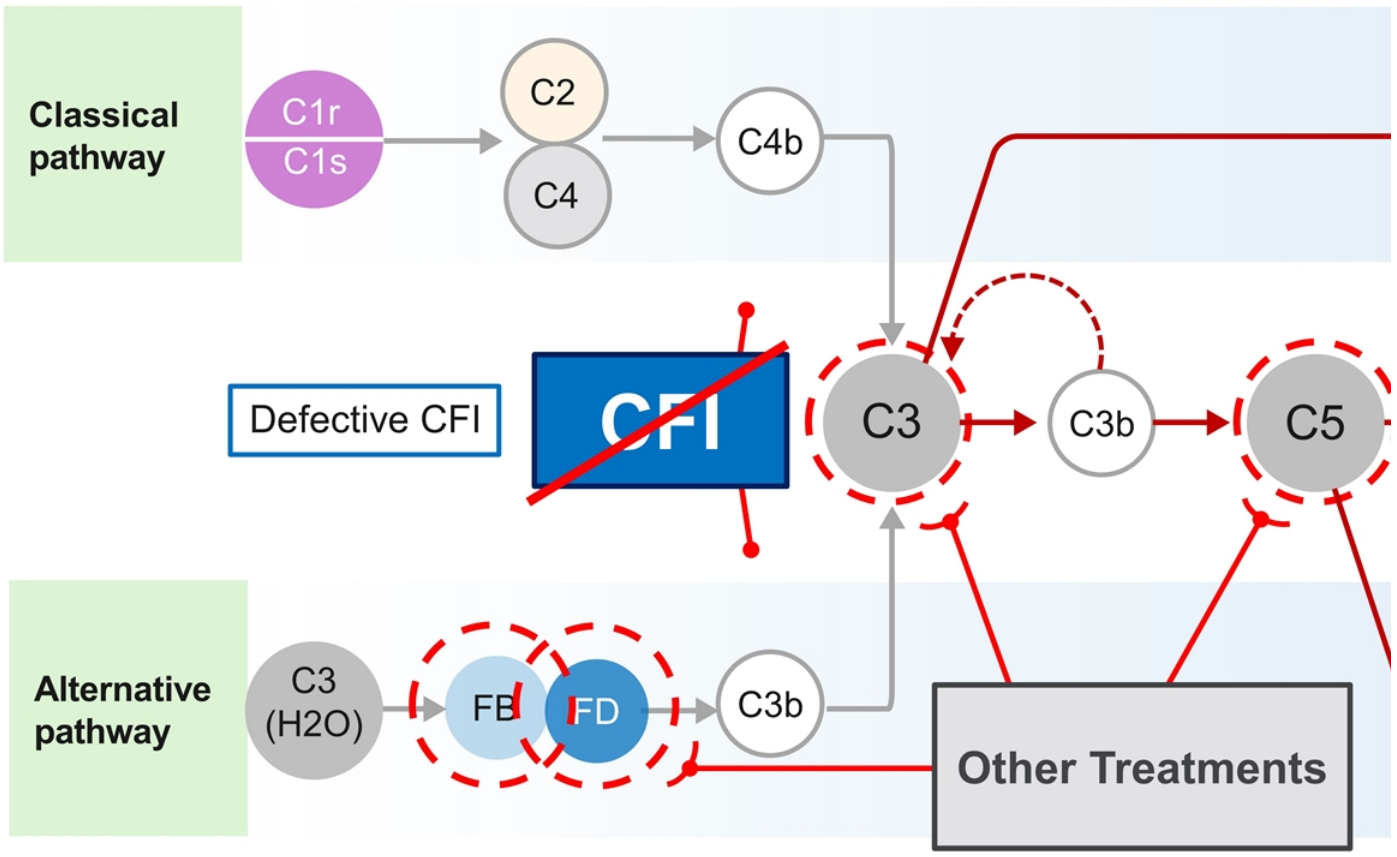


CFI dysregulation: Lack of proteolytic CFI



+ In patients with CFI mutations, C4b and C3b cannot be sufficiently
+ Dysregulation leads to overactivation of the complement pathway a

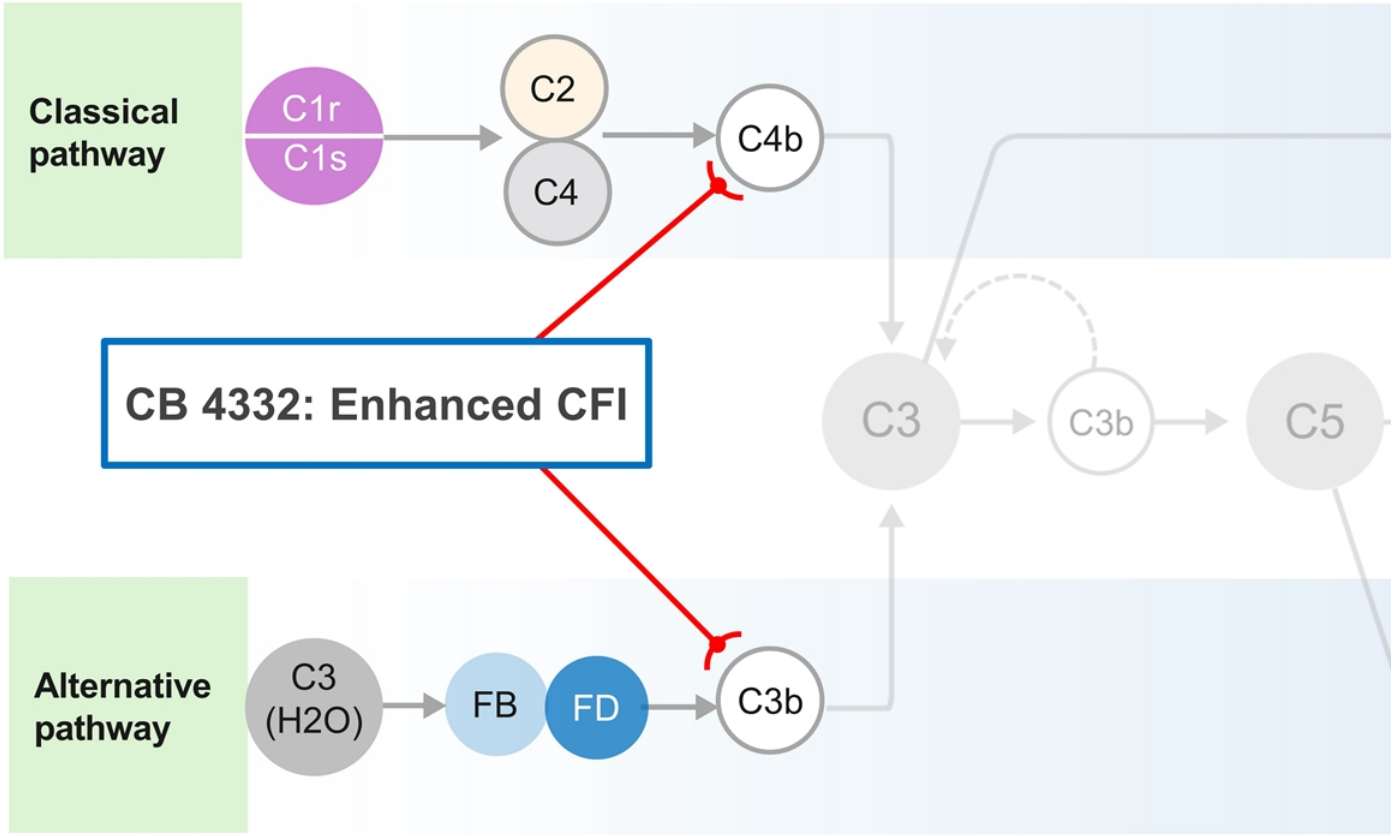
Other treatments do not directly address C



+ Current C5 blockade therapies do not address disease root cause
+ Small molecules and peptides are unable to fully block complemen

CB 4332 - Catalyst's enhanced CFI

Specifically addresses the problem by restoring CF



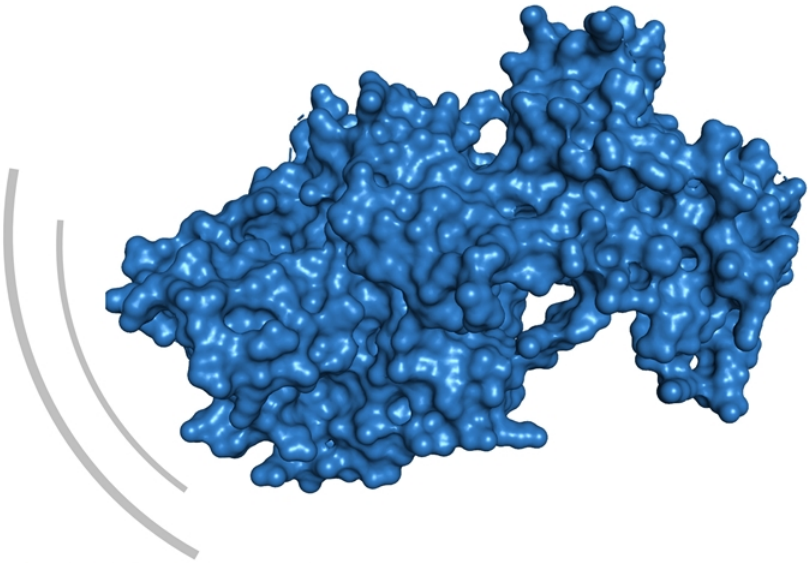
CB 4332 to address CFI deficiency at the r

CB 4332 designed to provide unique advantages

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

CB 4332: Enhanced Complement Factor I

CBIO's next SQ development candidate to restore C



Half-Life Extension Technology

+ **Engineered for an extended half-life**
  - Once weekly SQ therapy
+ **Full activity comparable to native CFI**
  - Classical and alternative pathway regulation
+ **Efficient high yield production process**

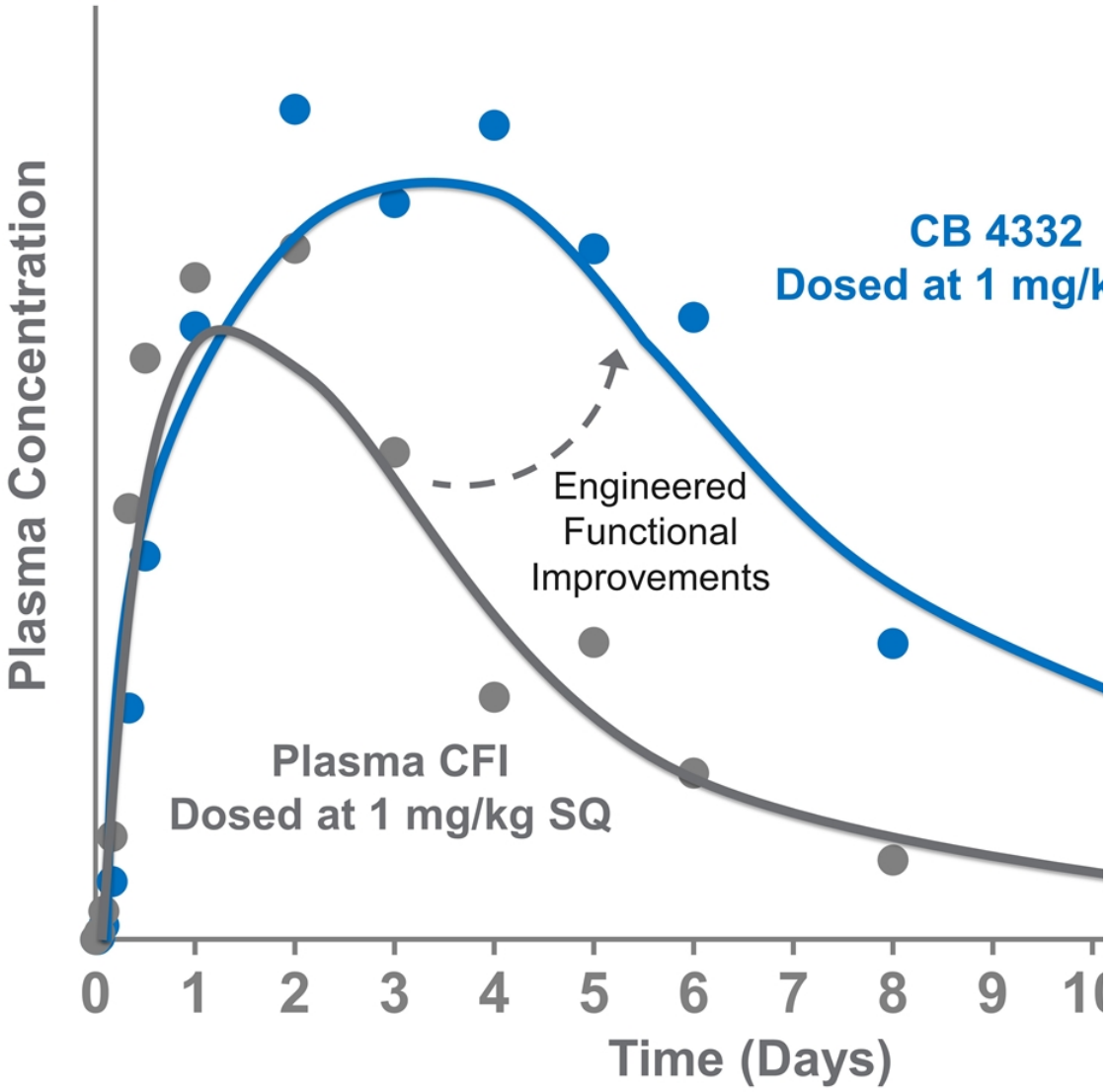
R

+ **Resto**
  in patie
+ **No sp**
  CFI dy
+ Target
  who r
  treatm
+ **Genet**

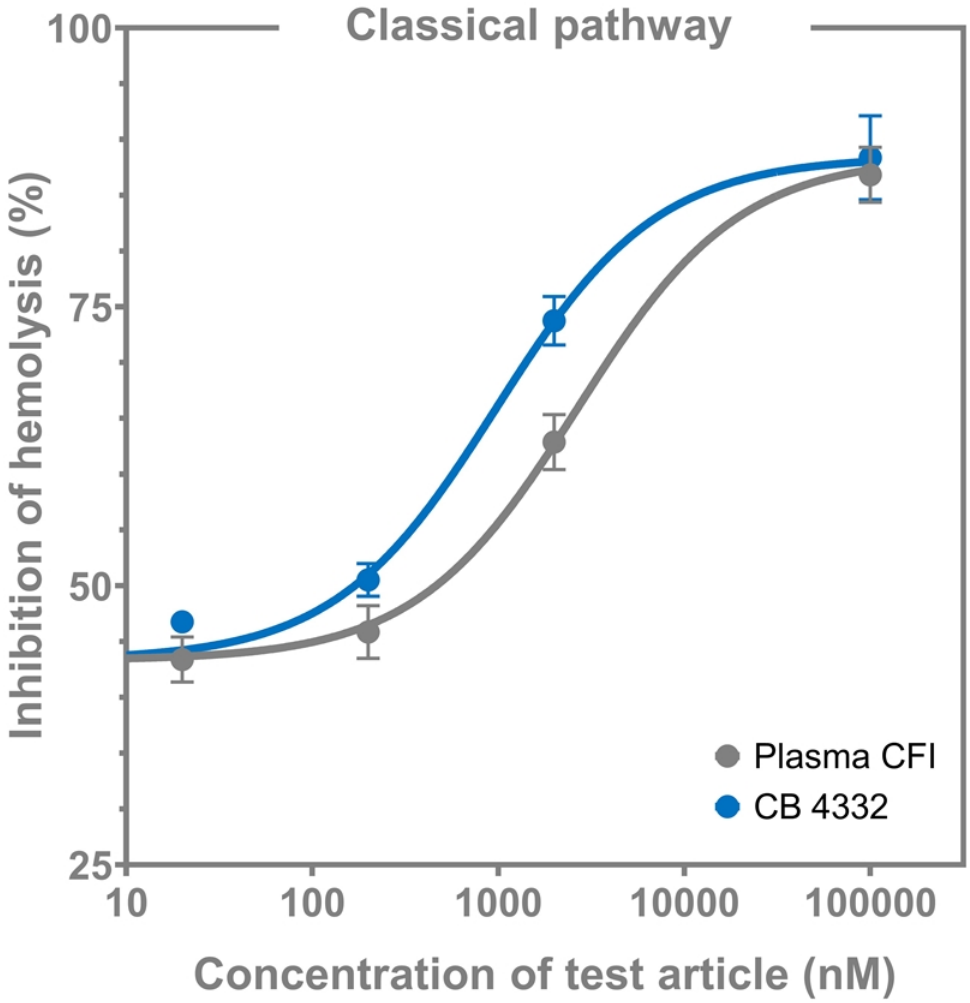
References: 1
Note: CFH =
Structural m

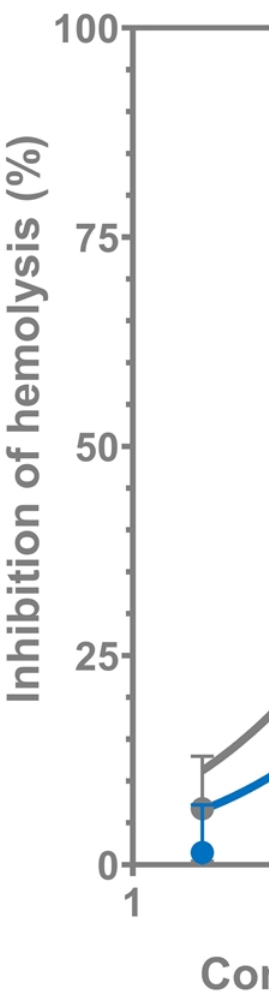
CB 4332 nonhuman primate pharmacokinetics

Systemic single-dose PK in nonhuman primates



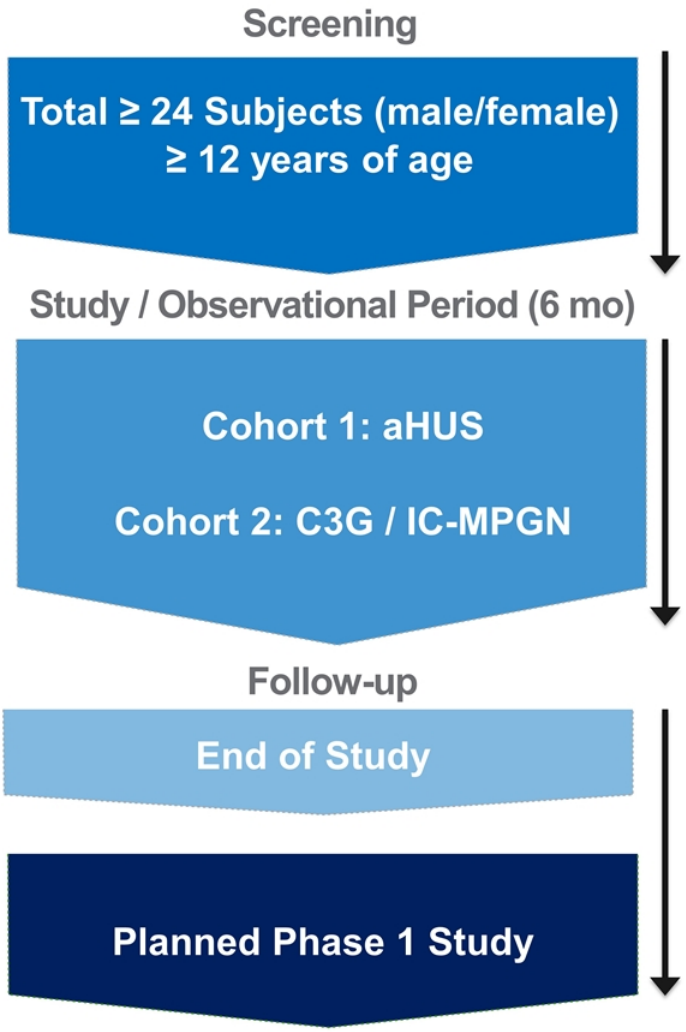
CB 4332 & plasma CFI perform similarly ir





CB 4332 - CFI dysregulation observational

Observational trial to identify CFI deficient patients for furth



Objectives

- **Primary O**

  Demonstrate
  manifestatior
  subjects requ
  Phase 1 stuc

- **Secondary**

  Monitor effica
  Monitor safet
  Record dosir
  Monitor QoL

Timeline

Observationa
Global phase
Intend to pur

Note: aHUS = atypical Hemolytic Uremic Syndrome, C3G = Complement 3 Gl
= Immune-Complex Membranoproliferative Glomerulonephritis

CB 4332 market opportunity

US / EU5 market opportunity

Additional opportunity in CFI Deficiency outside nephrology

$500M

Market opportunity in CFI deficient aHUS, C3G, IC-MPGN

Signif

0 Spe for

0 The

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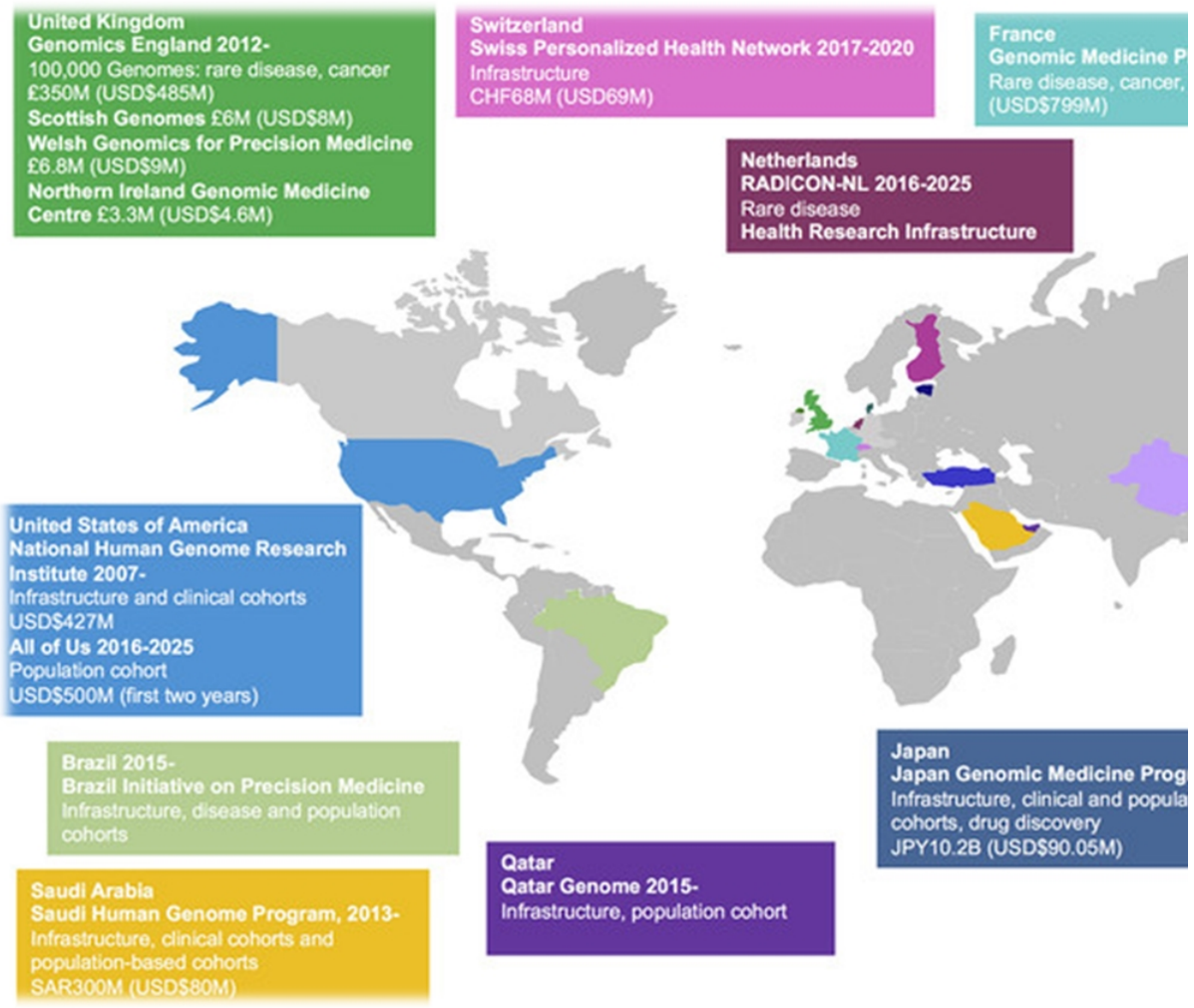
CFI muta

Note: aHUS = atypical Hemolytic Uremic Syndrome, C3G = Complement 3 Glomerulopathy, IC-MPGN = Immune-Complex M Factor I Deficiency

References: Bresin et al. JASN. 2005; Fremeaux-Bacchi et al. ASN. 2013; Rui-Ru et al. Jour Rare Dis Res. 2018; Servais et a al. Kidney Int. 2014; Alba-Domiguez et al. J rare Dis. 2012. El Sissy et al. Front. Immunol. 2019; Shields et al. Front Immunol. al. Clin Epi 2020; Smith et al. Nature Reviews. 2020; Noris et al. Clin J Am Soc Nephrol. 2010; CBIO KOL interviews

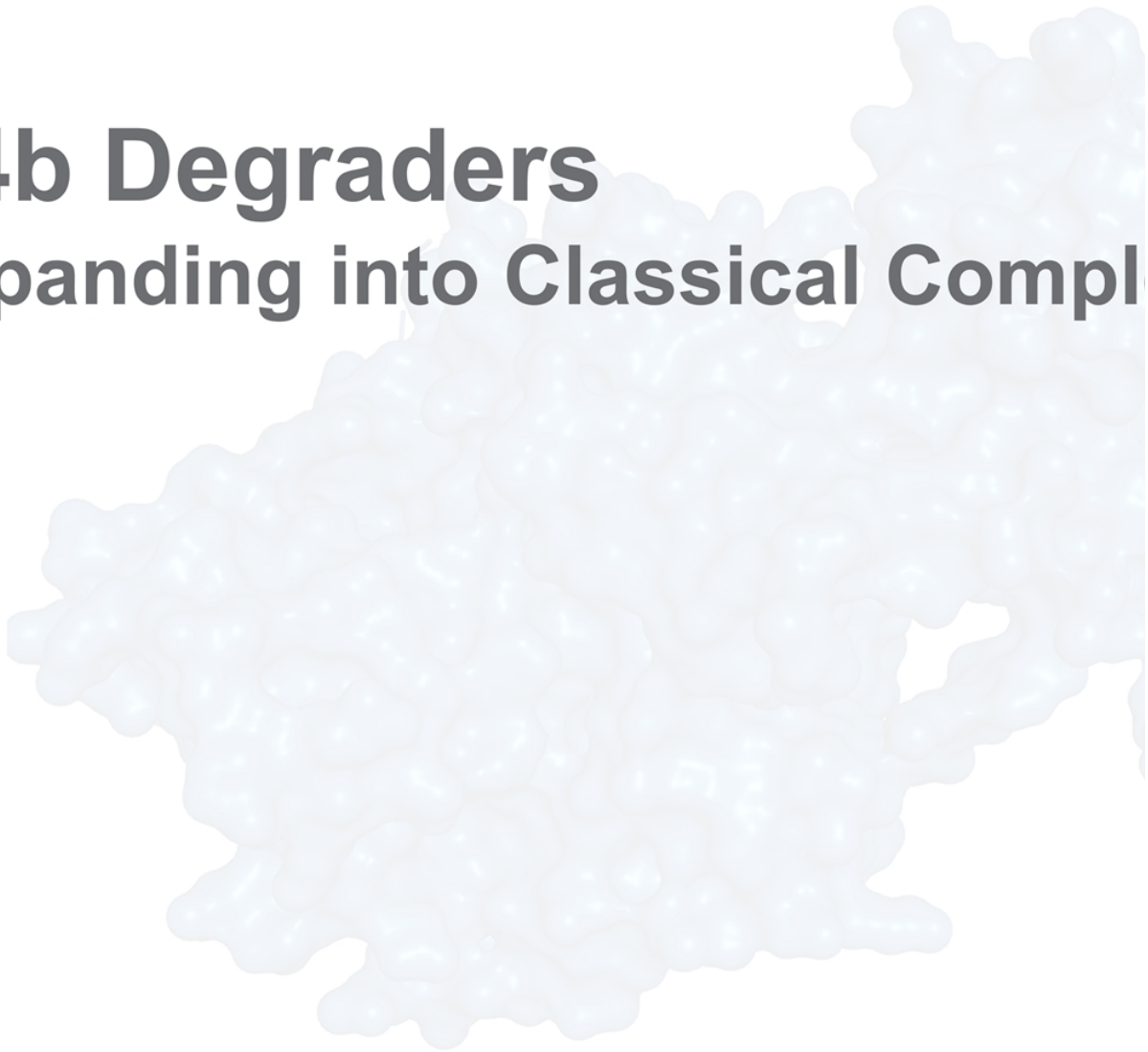
Nonfunctional CFI increasingly identified as g



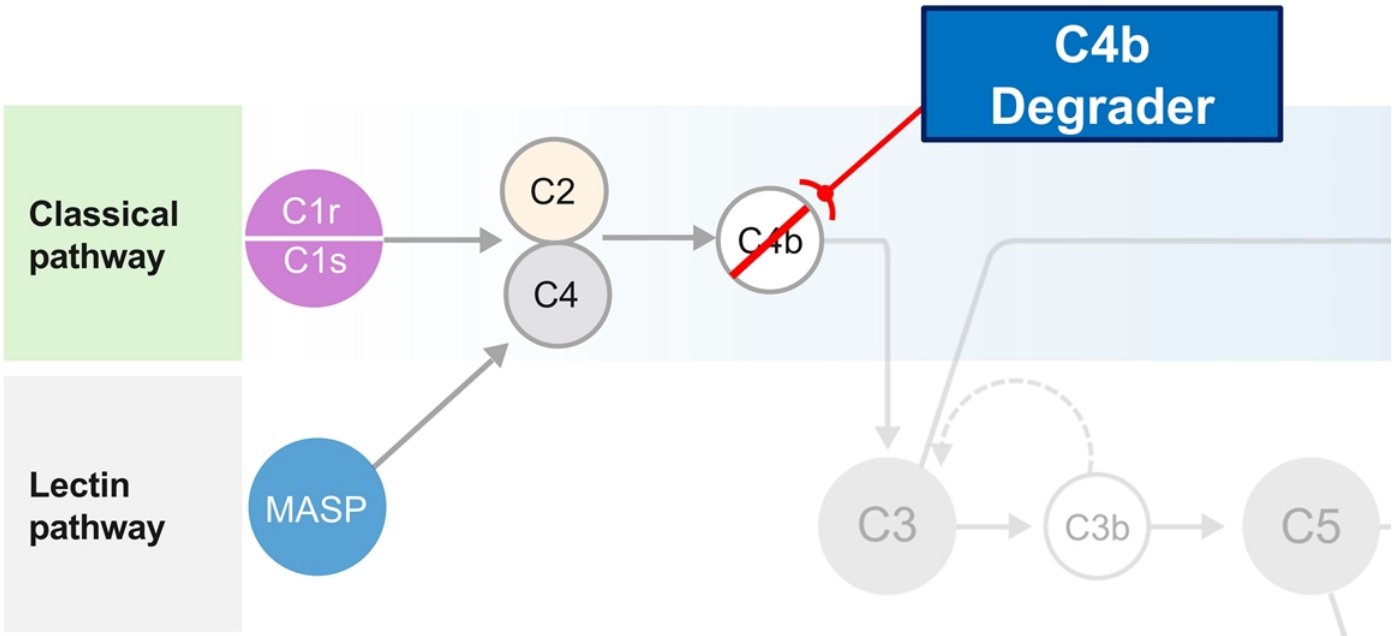
References: World Economic Forum. 2019

C4b Degraders

Expanding into Classical Compl



Catalyst C4b degrader complement therap



Selective & potent

+ Catalyst's protease platform allows for tuning specificity to individual targets
+ Leverages CB 4332 protease scaffold + efficient high yield production process
+ No competitors specifically targeting C4b

C4b degraders target multiple high unmet

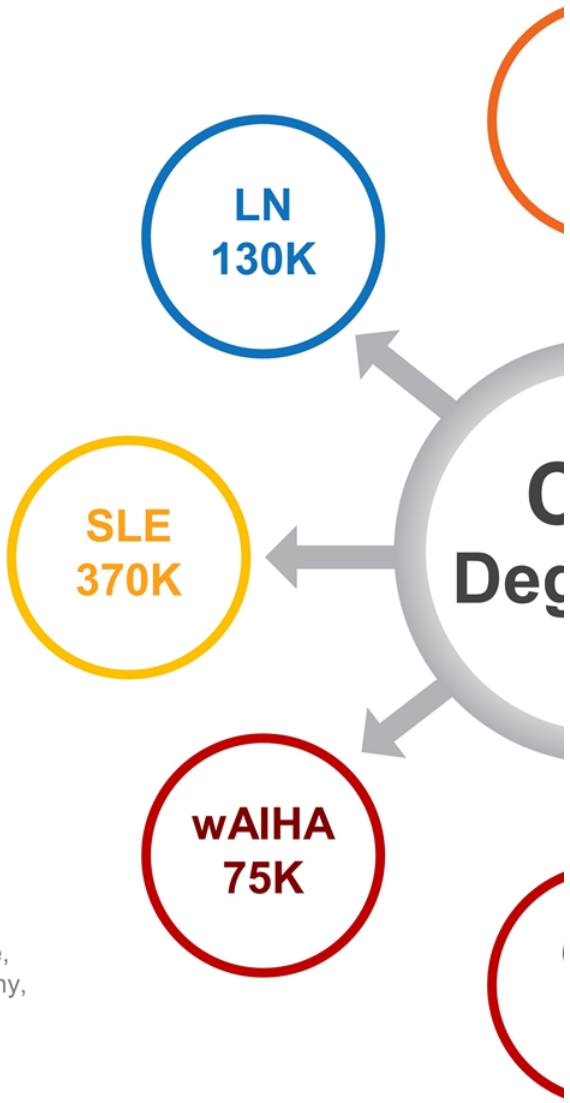
US & EU5 patient opportunity

Nephrology

Immunology

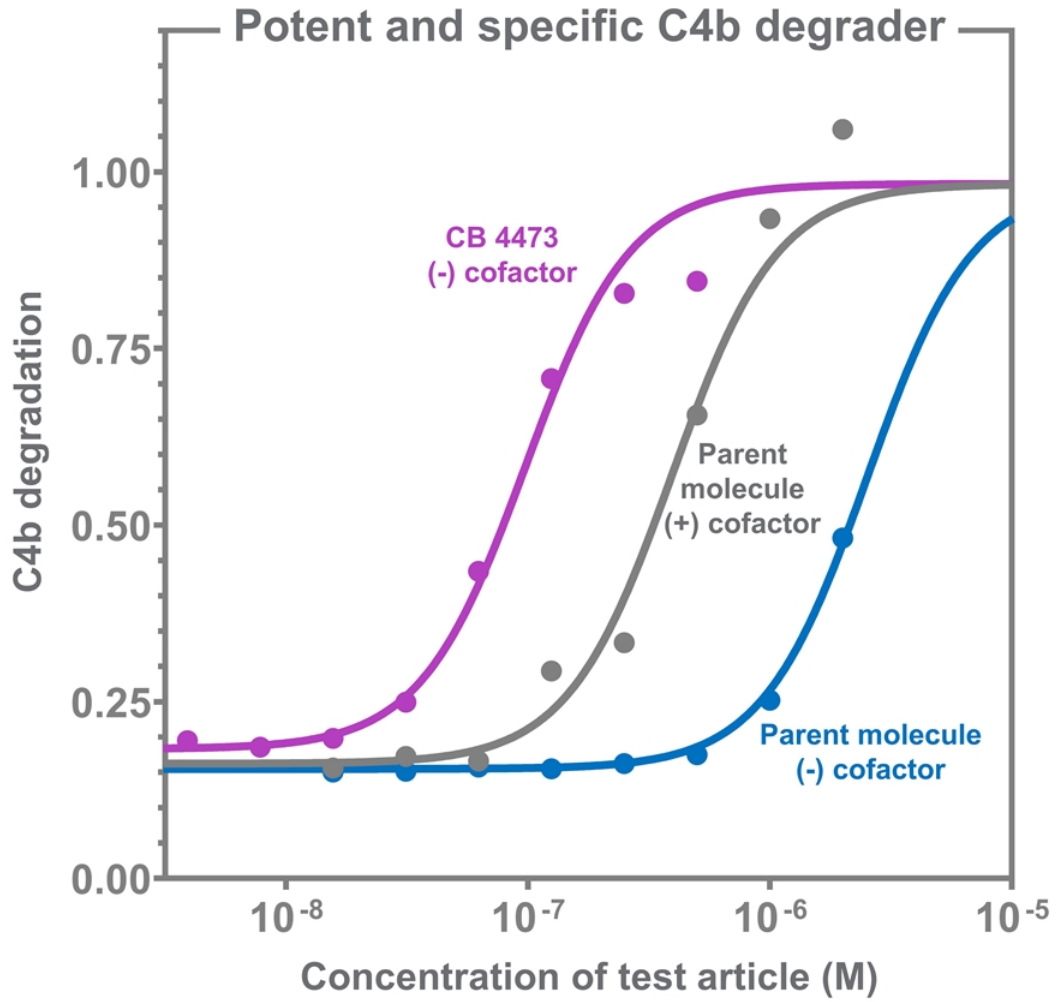
Hematology

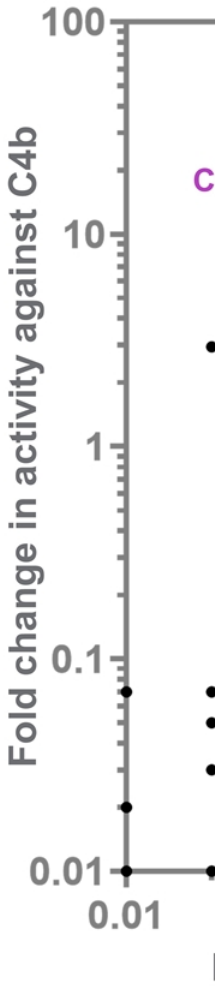
Neurology



Note: ALS = Amyotrophic lateral sclerosis, GBS = Guillain-Barré syndrome, gMG = Generalized Myasthenia Gravis, MMN = multifocal motor neuropathy, CAD = Cold agglutinin disease, wAIHA = warm Autoimmune hemolytic anemia, SLE = Systemic lupus erythematosus, LN = Lupus Nephritis, References: Data on file

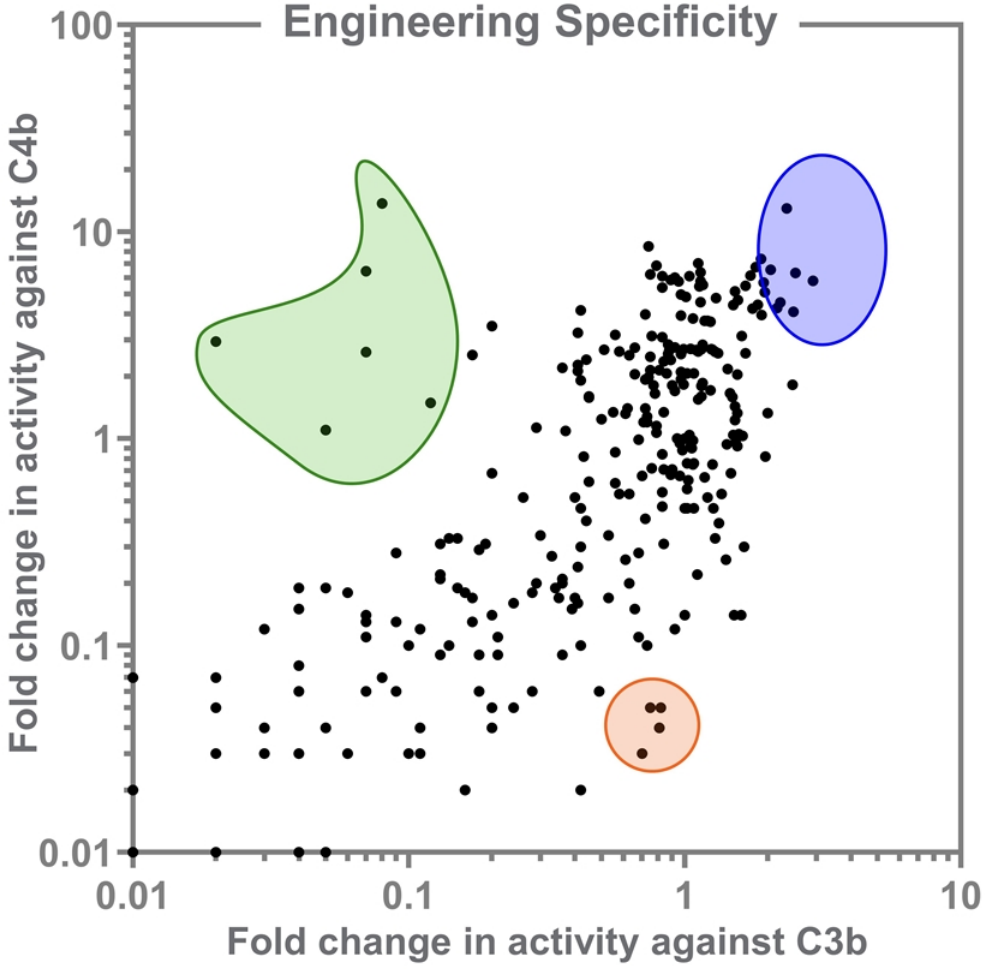
CB 4473 demonstrates engineered C4b pc





Tuning potency and selectivity to the thera

Catalyst is developing a portfolio of C3b and C4b d

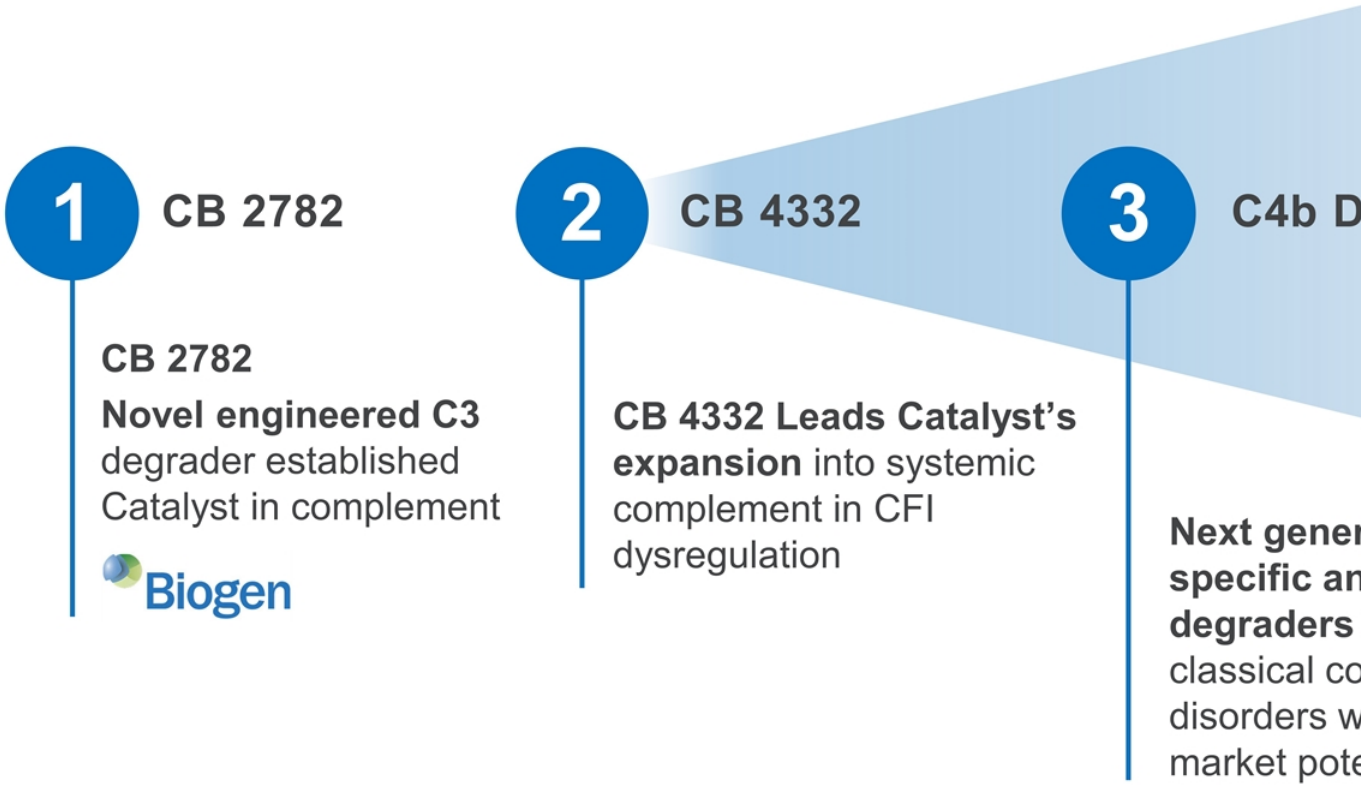


Classical pathv
>10x specificity

Alternative pat
>10X specificity

Central pathwa
Variants with inc

Catalyst's complement pipeline



Clinton Musil

CFO

Closing Remarks, Q&A

CBIO's complement program next steps

- **Observational trial for CB 4332**
- **Updates on C4b degrader and additional programs**
- **Progress CB 2782 in collaboration with Biogen**
- **CB 4332 in the clinic globally**

CATALYST BIOSCIENCES

Corporate Overview

14 December 2020

CatalystBiosciences.com

Forward looking statements

Certain information contained in this presentation and statements made orally during this presentation include forward-looking statements that involve 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 Catalyst's product candidates and the benefits of its protease engineering platform, potential markets for and advantages of MarzAA and DalcA; plans in Q4 2020 to enroll a pivotal Phase 3 registration study of MarzAA; the initiation of a Phase 1/2 trial in patients with FVII Deficiency, Glanzmann Thrombasthenia, and patients treated with Hemlibra; MarzAA as possibly the first prophylactic for FVII Deficiency and Glanzmann Thrombasthenia; the potential for MarzAA and DalcA to effectively and therapeutically treat hemophilia subcutaneously; projected complement market opportunity, solution to fundamental shortcomings in current treatment options, plans to enroll the CB 4332 observational trial in the Company's complement program in mid-2021, and ongoing updates related to CB 4322 and the C4b degrader.

Actual results c
expectations ar
statements. Va
events to differ
and studies ma
that trials may
replicate the re
develop or mar
anticipated, inc
manufacturing
Biogen will tern
risks described
Report on Form
Commission ("S
10-Q filed with
the SEC. The fo
the Company's
Company does
looking stateme

Catalyst Biosciences – Protease medicine

Protease engineering pl

Late-stage asset

SQ Marzeptacog alfa
(activated)
MarzAA (FVIIa)

Phase 3

Complement

CB 2782-PEG
IVT Anti-C3 Dry AMD

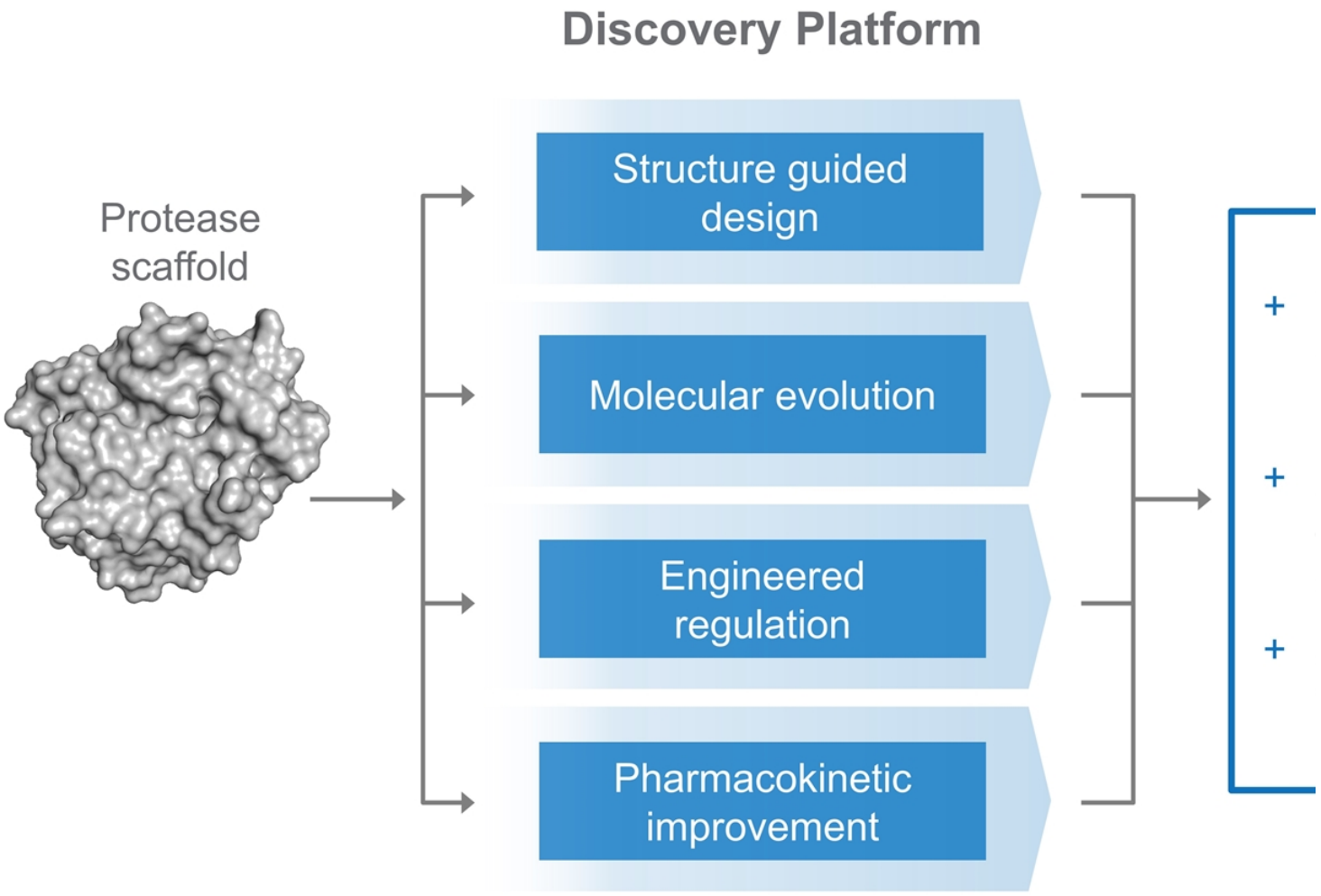
Biogen

CB 4332 SQ Enhanced CFI

SQ Systemic Complement
Degraders

Catalyst's protease platform generates dif

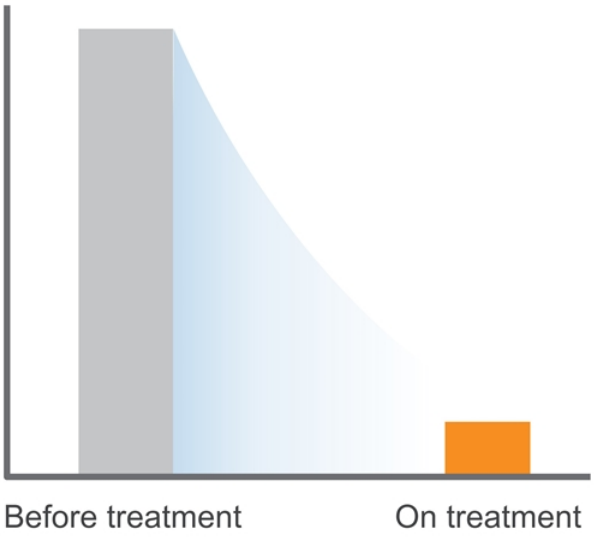
Unique expertise in protease biology enables design of op



Clinical & partnering success of the CBIO

Marzeptacog alfa (activated)

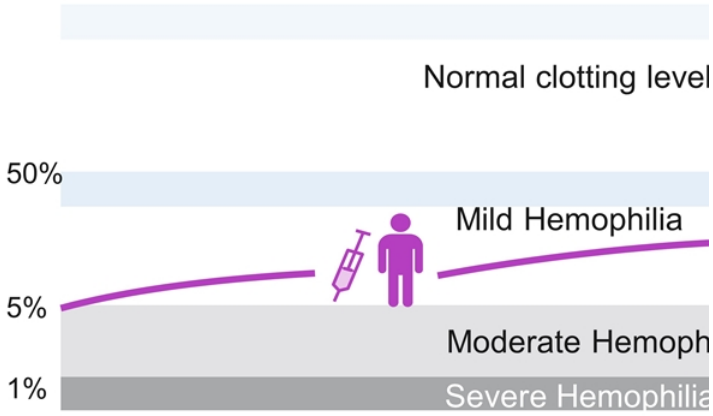
90% reduction
in annualized bleed rate



Engineered
rFVIIa protease

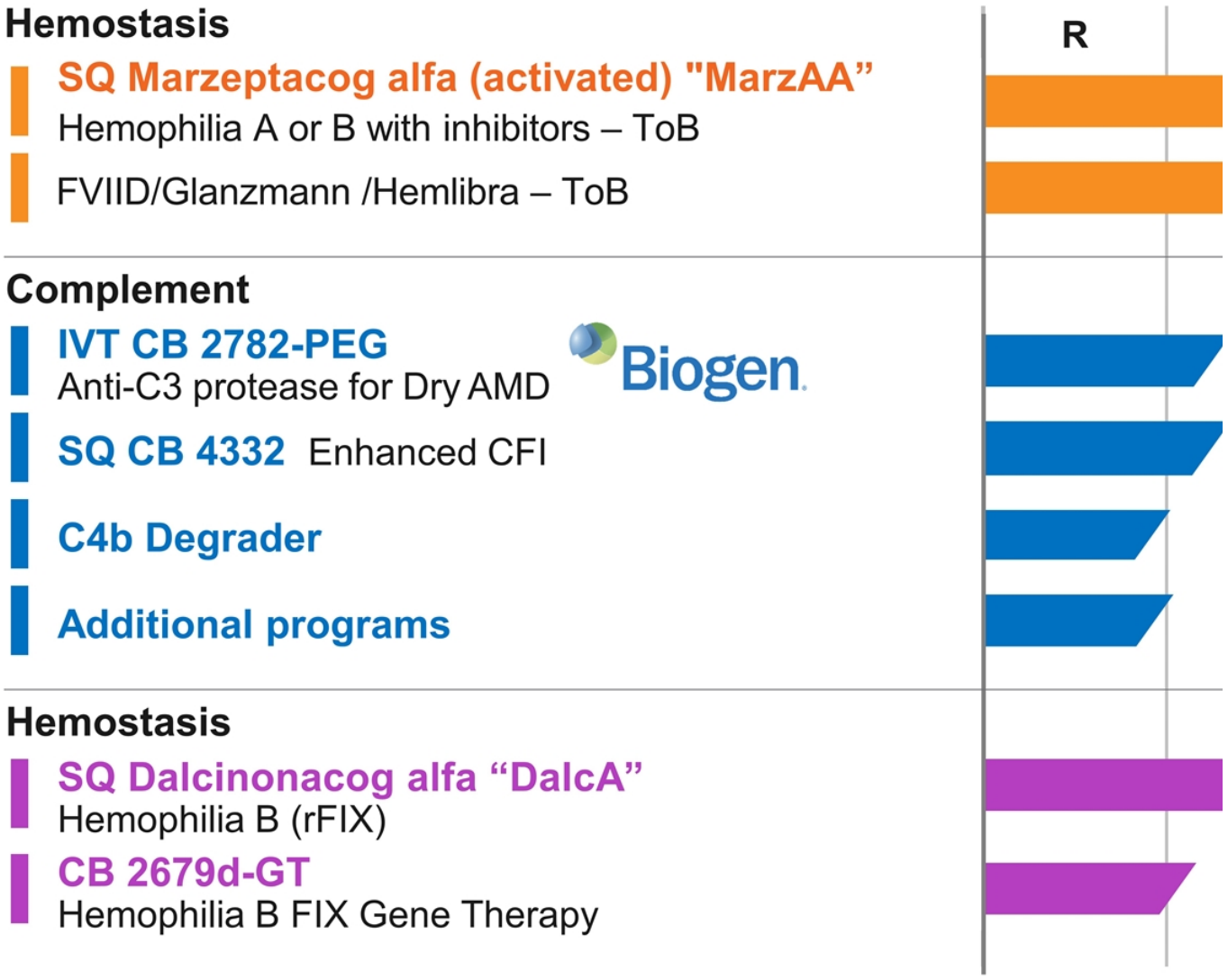
Dalcinonacog alfa

Achieved sustained
& high target levels of FIX



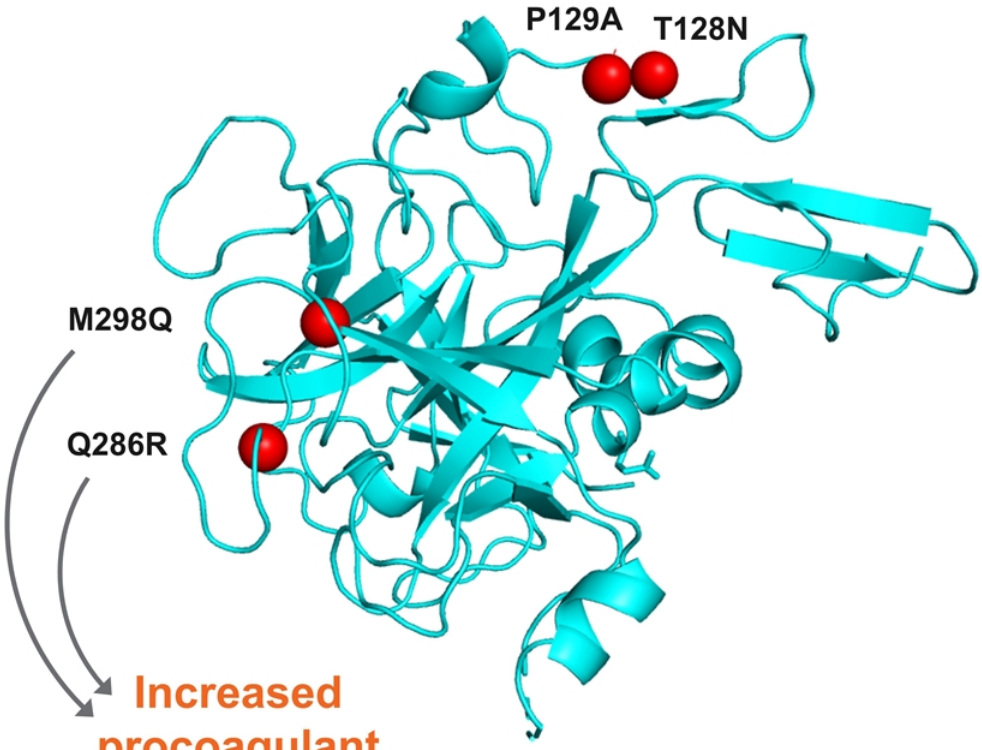
Engineered
rFIX protease

Pipeline



Marzeptacog alfa (activated) – MarzAA: SC

Addresses a clear unmet need in hemophilia & othe



9-fold higher a

+ Potency allows
+ Simple, small v

Preclinical effi

+ HA mouse afte

P2/3 prophylax HB with inhibi

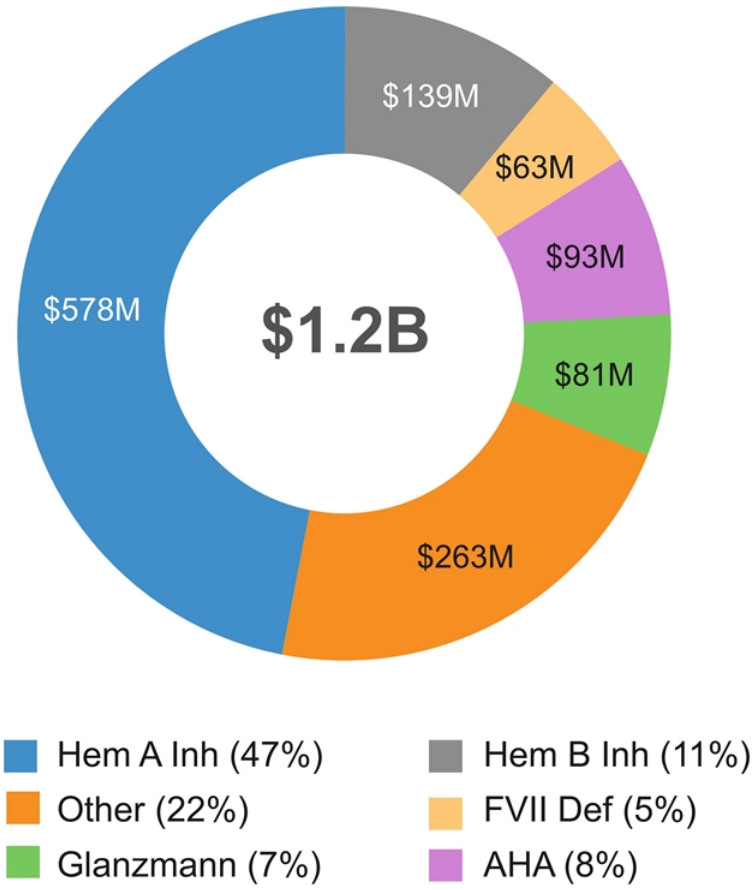
+ 46 patients trea
  3 SQ doses/da

FDA Fast Trac episodic bleed

+ Granted on 6 D

SQ MarzAA is a large commercial opportu

Global NovoSeven sales breakdown by indication (2019)

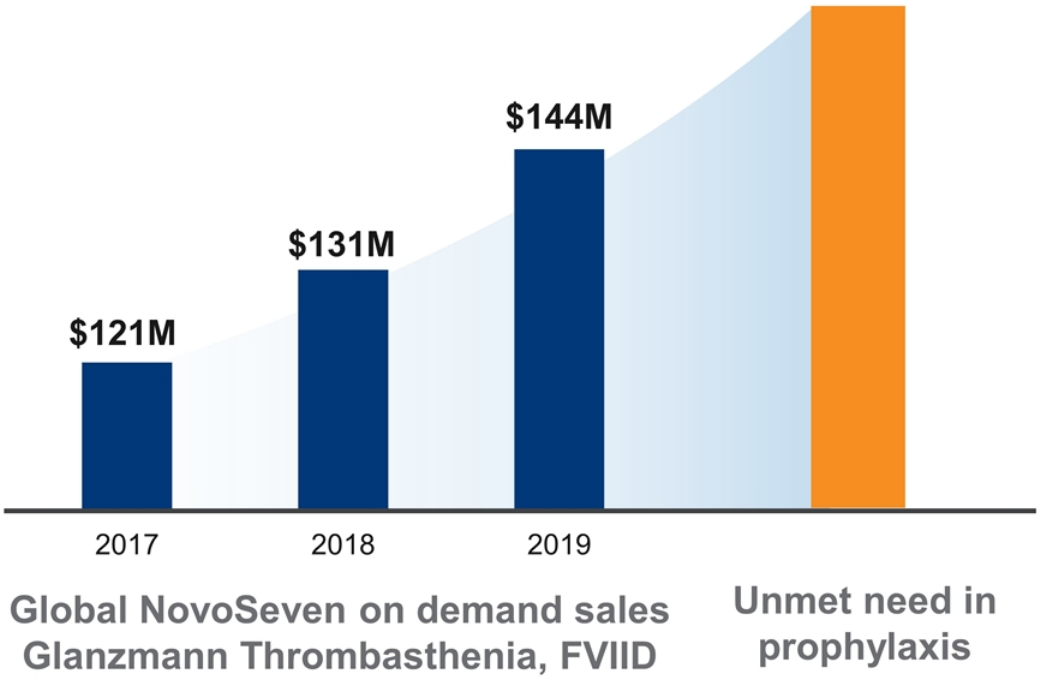


SQ MarzA

- Faster & every 2
- MarzAA
- 9-fold hi
- Potentia
- Stops b
- Can be *in vitro* v
- Ideal for access
- Prophyl

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

MarzAA could be the first prophylactic for

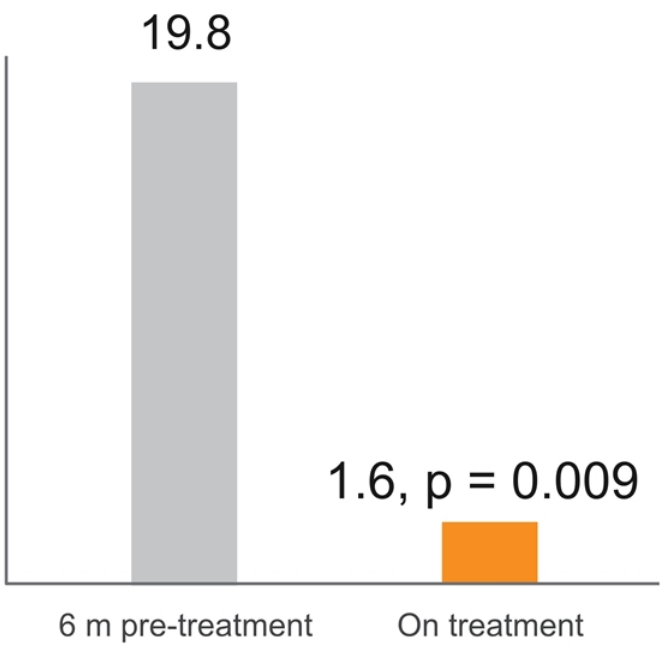


Source: Catalyst Biosciences, Adivo Associates Market Research, Data on file. *Note: Treated patients may be counted multipl
events per year needing factor treatment

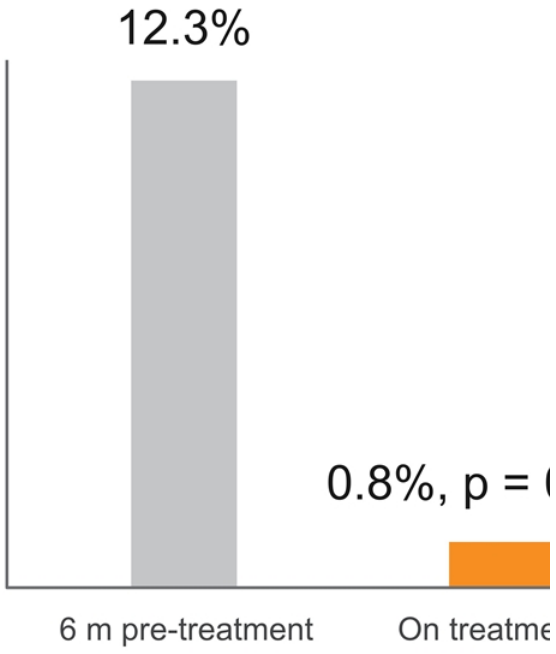
MarzAA is efficacious with daily prophylax

Phase 2: Daily SQ dosing for 44 – 97 days

Annualized bleed rate
n = 9



Proportion of days with bleeding
n = 9



Mahlangu *et al.* EAHAD 2020

Unmet need in treatment of a bleed

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

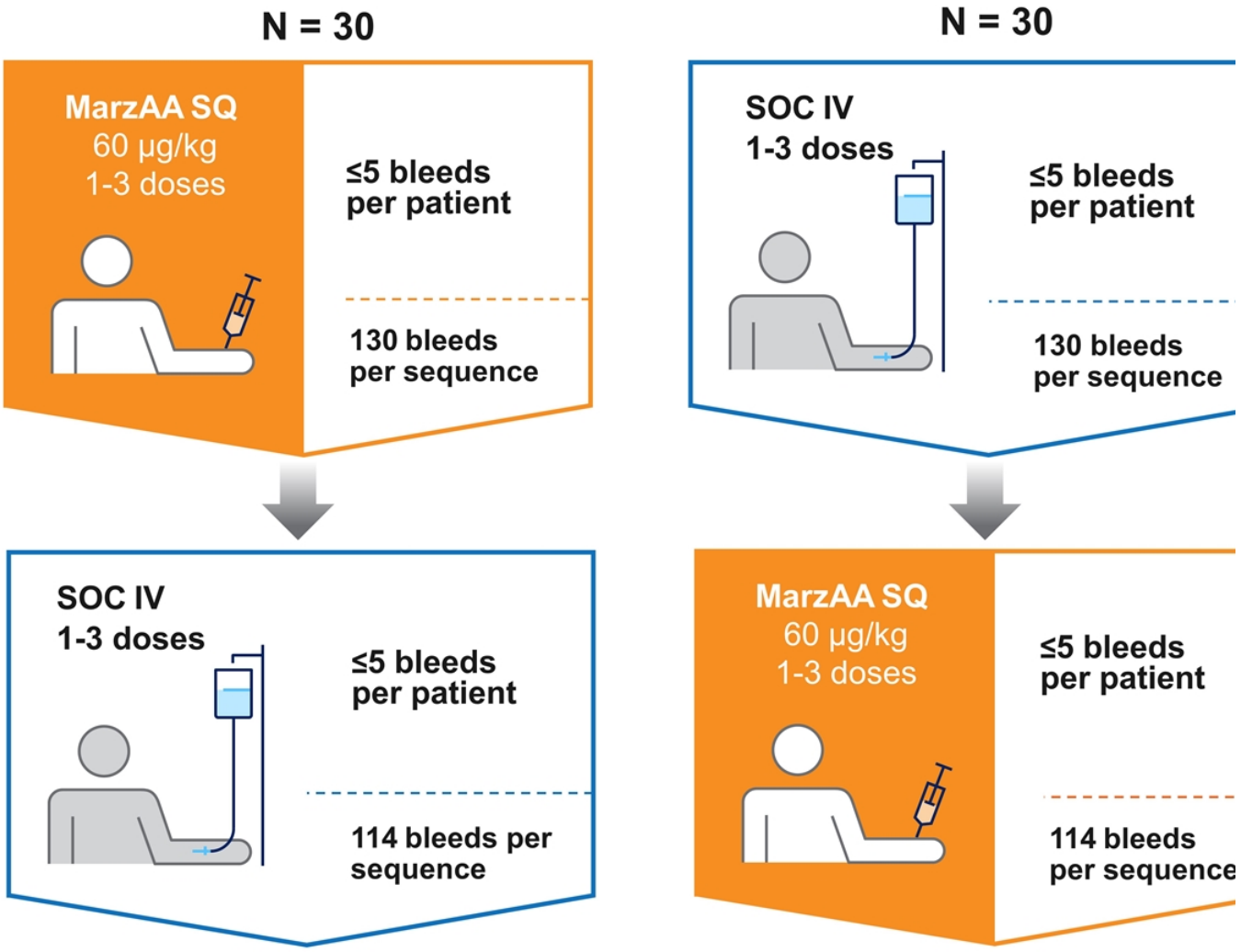
+ MA
  sup
+ Tar
+ Tar
  18
  60

Clini

Source: [1]NovoSeven PI Rev 7/2020; [2]Adivo Associates market research; [3]Catalyst Biosciences market research;

Crimson 1 Phase 3 study: Treatment of ep

Hemophilia A or B with inhibitors, ABR ≥ 8



MAA-202 Phase 1/2 study design

FVII deficiency, Glanzmann Thrombasthenia and HA

Phase 1 PK

MarzAA IV
each cohort

Single dose

MarzAA SQ

Single dose escalation

Multiple dose Q3H

Phase 2 ToB

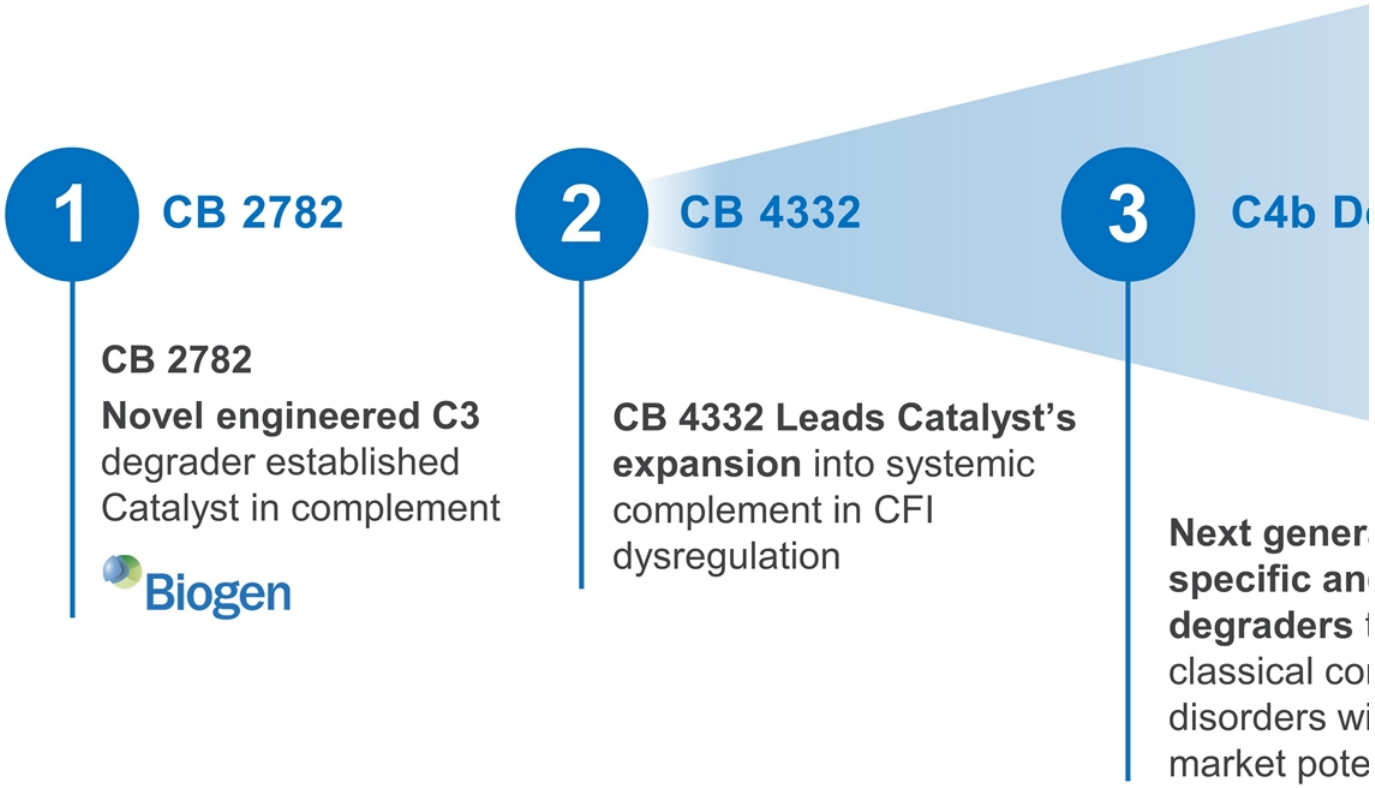
MarzAA SQ
1-3 doses

FVIID ≥30 bleeds

GT ≥30 bleeds

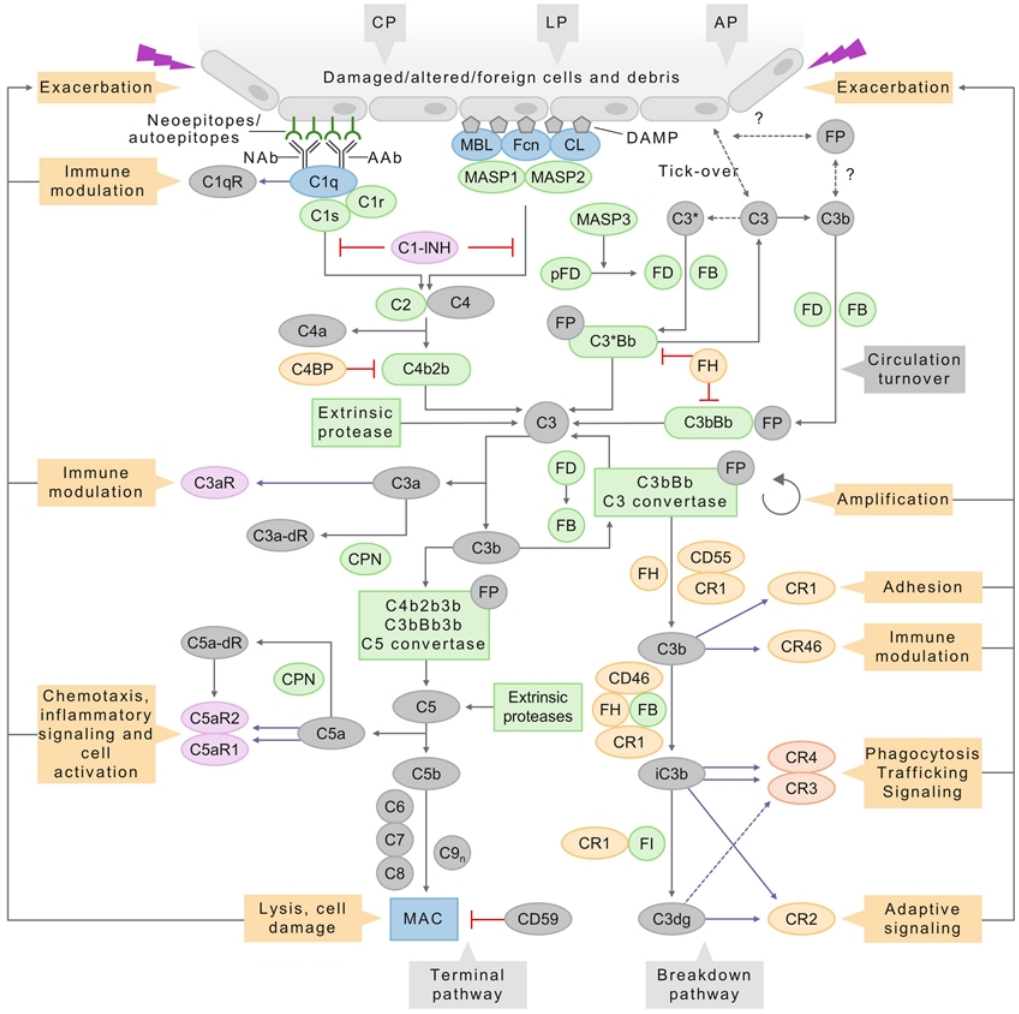
HA ≥15 bleeds

Catalyst's complement pipeline



Complement is a perfect fit to develop pro

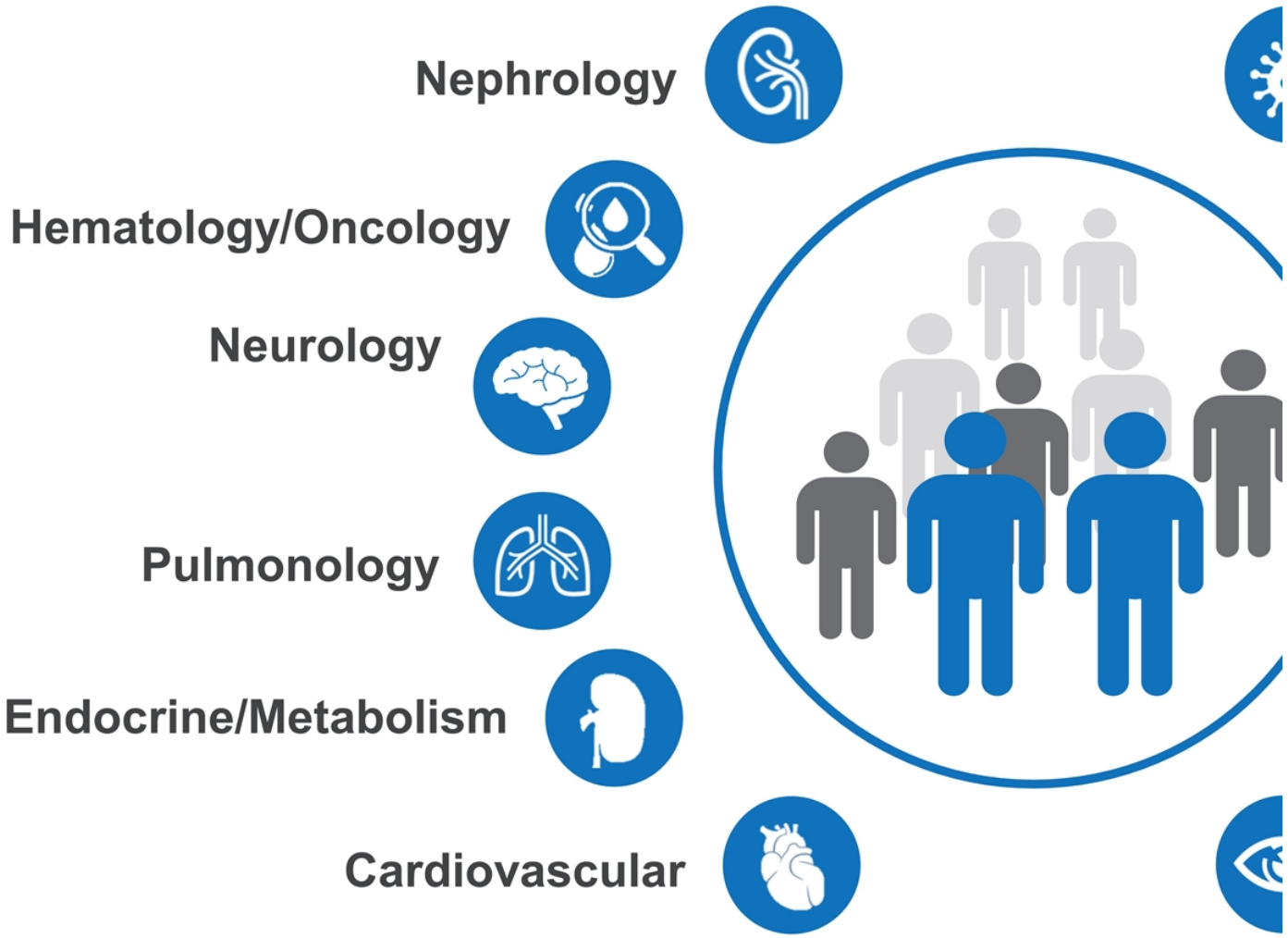
The complement pathway is driven by a protease ca



Reference: Figure adapted from Mastellos et al., Clinical promise of next-generation complement therapeutics. Nat

Complement plays a critical role in many

Late-stage complement therapies projected to achi



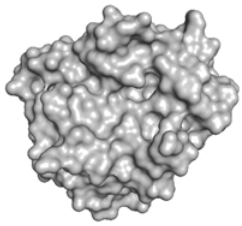
References: Globaldata consensus net sales forecast 2020

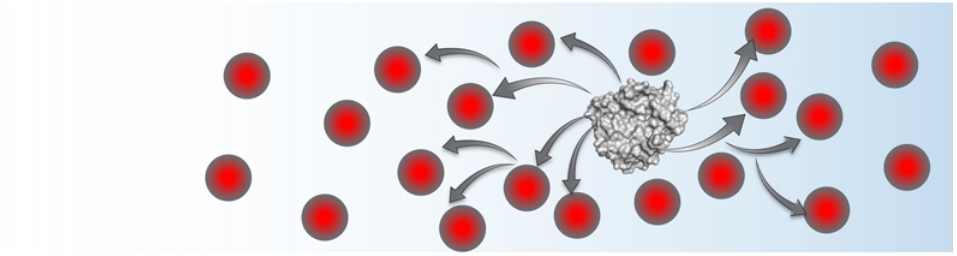
Proteases are ideal for high abundancy ta

A better way to regulate biological processes compare

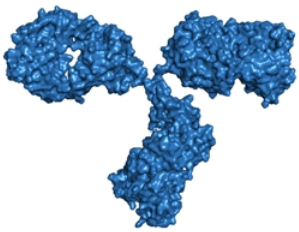
Therapeutic target neutralization

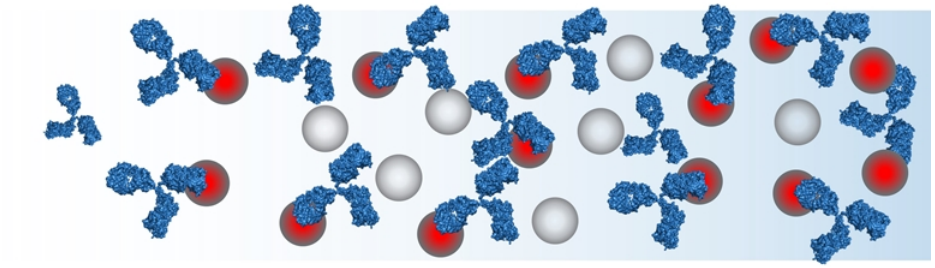
Protease



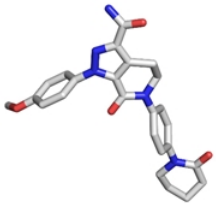


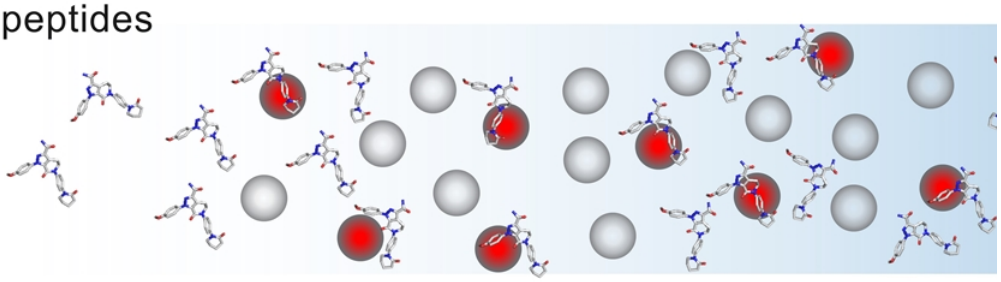
Antibodies





Small molecules / peptides

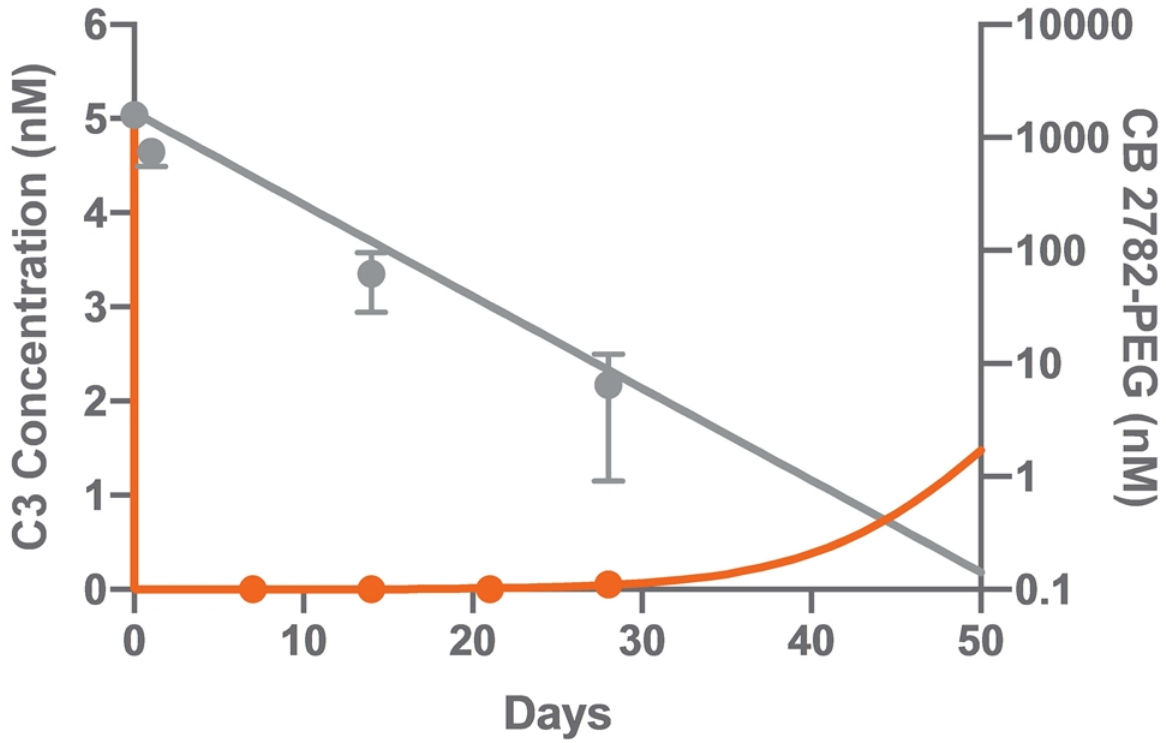




Protease advantage demonstrated *in vivo*

CB 2782-PEG Biogen Designed for a best-in-class

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



CB 2782-PEG long acting anti-C3 protease

Geographic atrophy in dry AMD can result in blindness

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

Best-in-class anti-C3 profile for dry AMD

+ Generated from Catalyst's proprietary **protease engineering platform**
+ Potent, selective and long acting anti-C3 protease that degrades C3 into ir
+ Preclinical NHP PK & PD data* predict **best-in-class** human intravitreal d

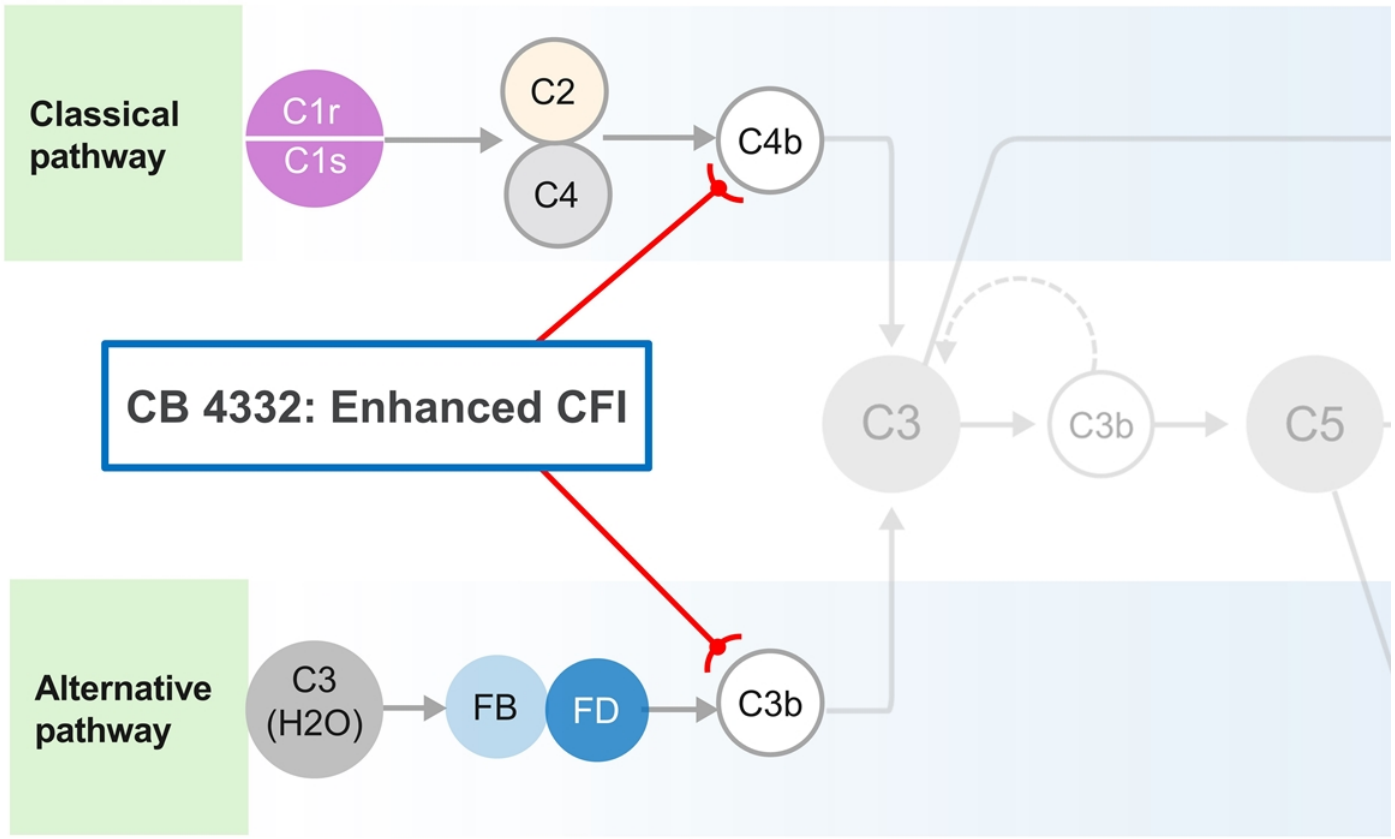
Biogen collaboration

+ $15M upfront, up to $340M in milestones and tiered royalties up to low dou
+ Catalyst to perform fully funded pre-clinical and manufacturing activities
+ Biogen responsible for IND-enabling activities, worldwide clinical developm

*Furfine *et al.* ARVO 2019

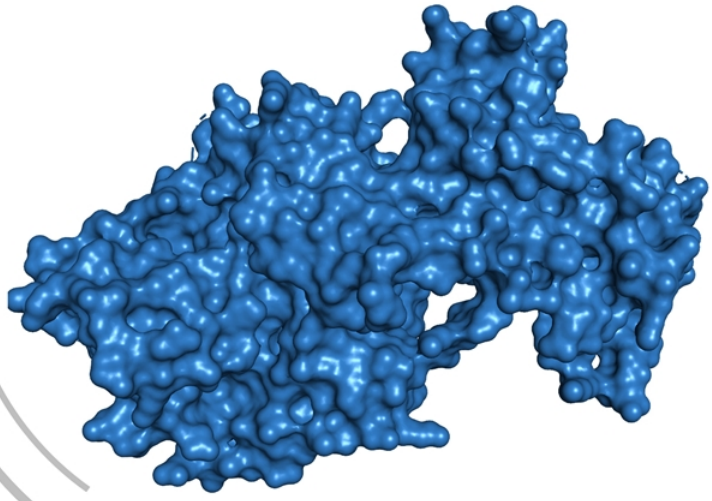
CB 4332 - Catalyst's enhanced CFI

Specifically addresses the problem by restoring CF



CB 4332: Enhanced Complement Factor I

CBIO's Next SQ Development Candidate to restore



Half-Life Extension Technology

Engineered for an extended half-life

+ Once weekly SQ therapy

Full activity comparable to native CFI

+ Classical and alternative pathway regulation

Efficient high yield production process

R

+ R
sy
dy

+ N
co

+ Ta
tr
to

+ G
p

References: [1]Bienaime et al. Kidney Int. 2010; [2]Ferreira et al. Nefrologia. 2016; Note: CFH = Complement factor H; Structura

CB 4332 market opportunity

US / EU5 market opportunity

Additional opportunity in CFI Deficiency outside nephrology

$500M

Market opportunity in CFI deficient aHUS, C3G, IC-MPGN

Signif

0 Spe for

0 The

0 App

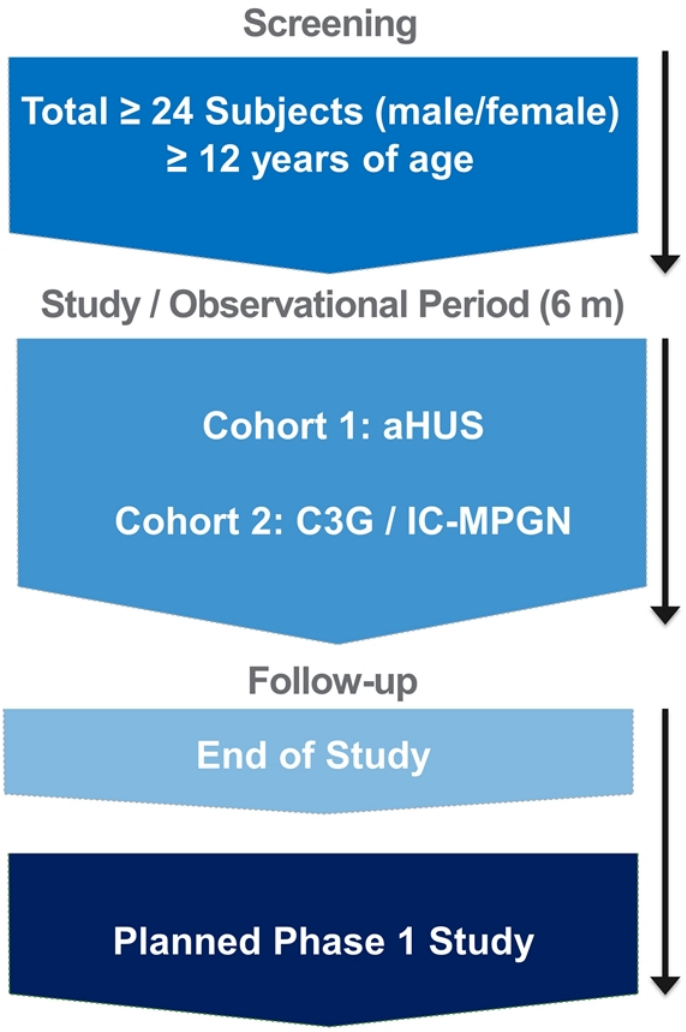
CFI muta

Note: aHUS = atypical Hemolytic Uremic Syndrome, C3G = Complement 3 Glomerulopathy, IC-MPGN = Immune-Complex M Factor I Deficiency

References: Bresin et al. JASN. 2005; Fremeaux-Bacchi et al. ASN. 2013; Rui-Ru et al. Jour Rare Dis Res. 2018; Servais et a al. Kidney Int. 2014; Alba-Domiguez et al. J rare Dis. 2012. El Sissy et al. Front. Immunol. 2019; Shields et al. Front Immunol. al. Clin Epi 2020; Smith et al. Nature Reviews. 2020; Noris et al. Clin J Am Soc Nephrol. 2010; CBIO KOL interviews

CB 4332 - CFI dysregulation observational

Observational trial to identify CFI deficient patients for furth



Objectives

- **Primary O**

  Demonstrate
  manifestatior
  patients requ
  Phase 1 stuc

- **Secondary**

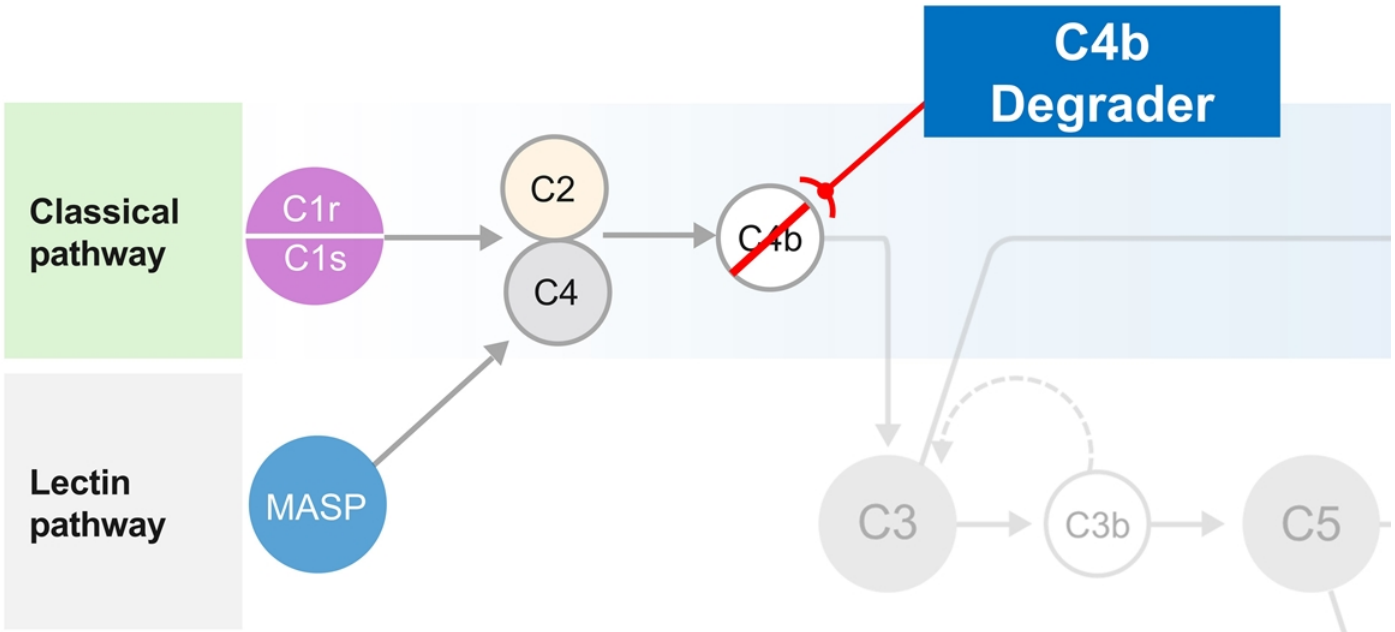
  Monitor effica
  Monitor safet
  Record dosir
  Monitor QoL

Timeline

Observationa
Global phase
Intend to pur

Note: aHUS = atypical Hemolytic Uremic Syndrome, C3G = Complement 3 Gl
= Immune-Complex Membranoproliferative Glomerulonephritis

Catalyst C4b degrader complement therap



Selective & potent

+ Catalyst's protease platform allows for tuning specificity to individual targets
+ Leverages CB 4332 protease scaffold + efficient high yield production process
+ No competitors specifically targeting C4b

C4b degraders target multiple high unmet

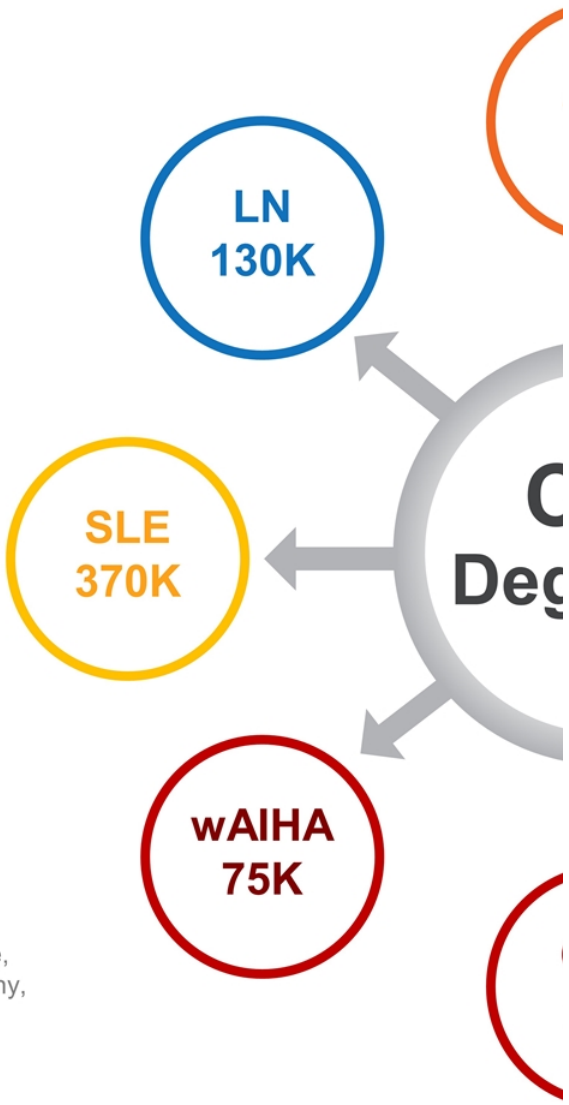
US & EU5 patient opportunity

Nephrology

Immunology

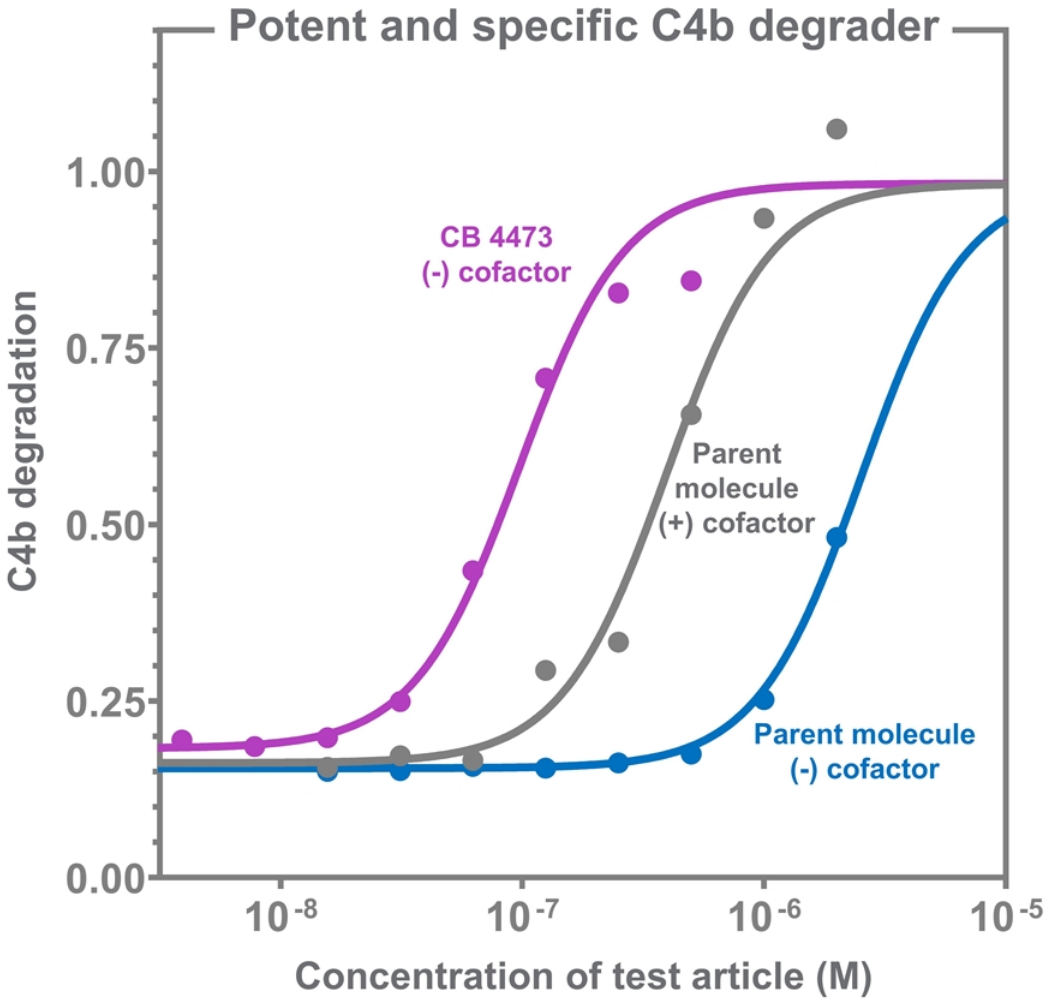
Hematology

Neurology

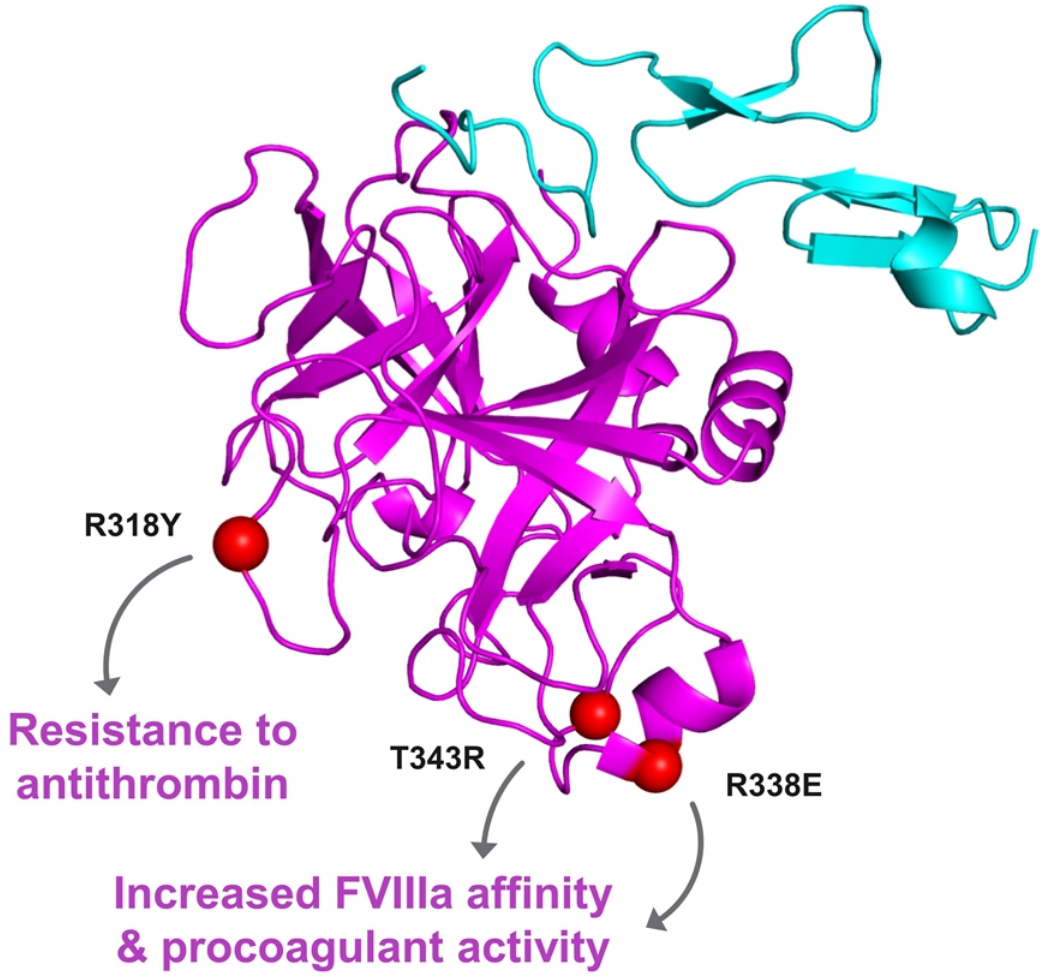


Note: ALS = Amyotrophic lateral sclerosis, GBS = Guillain-Barré syndrome, gMG = Generalized Myasthenia Gravis, MMN = multifocal motor neuropathy, CAD = Cold agglutinin disease, wAIHA = warm Autoimmune hemolytic anemia, SLE = Systemic lupus erythematosus, LN = Lupus Nephritis, References: Data on file

CB 4473 demonstrates engineered C4b pc

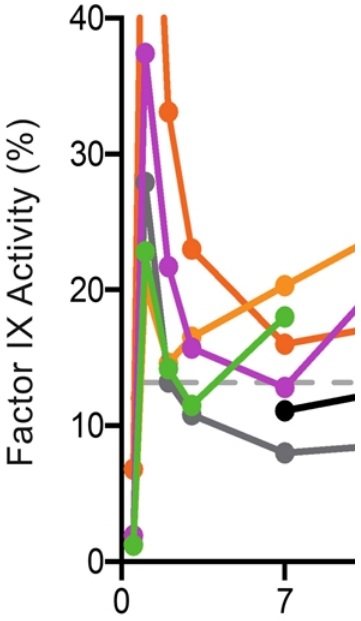


DalcA P2b demonstrated efficacy & safety



Differentiate

+ Small volum
+ Enhanced p
+ Excellent ex
+ Target level 100 IU/kg d



Catalyst's CB 2679d gene therapy for hem

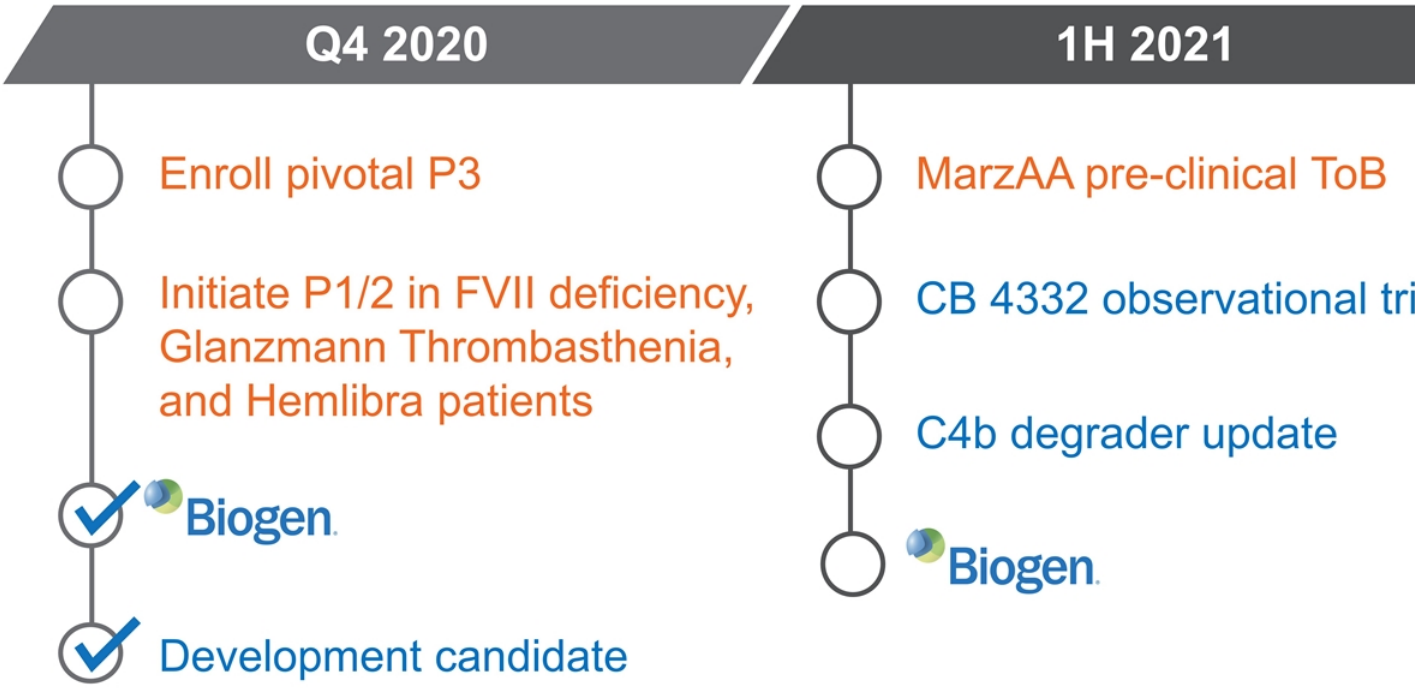
Engineered Capsid + **Novel Transgene** = **Lower AAV Dose**

FIX Transgene	AAV Capsid	Study Dose (vg/kg)	FIX Activity (U/mL)
CB 2679d-GT	**Novel Chimeric**	**$8.0x10^{10}$**	**20**
Padua	TAK-748*	$7.4x10^{11}$	20
Padua	TAK-748*	$7.4x10^{10}$	1

*Weiller *et al.* (2019) *Blood* Vol. 134, Supplement S1 P4633

Stanford University License & sponsored research agreement

Milestones



THANK YOU

Nasdaq: CBIO

CatalystBiosciences.com