Nasdaq: CBIO

CATALYST BIOSCIENCES

Corporate Overview

3 March 2021

CatalystBiosciences.com

CATALYST BIOSCIENCES

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 the product candidates of Catalyst Biosciences, Inc. (the “Company”) and the benefits of its protease engineering platform, potential markets for and advantages of MarzAA and DalcA; plans 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 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 delayed as a result of COVID-19 and other factors, that trials may not have satisfactory outcomes, that human trials will not replicate the results from earlier trials, the risk that costs required to develop or manufacture the Company’s products will be higher than anticipated, including as a result of delays in development and manufacturing resulting from COVID-19 and other factors, the risk that Biogen will terminate its agreement with the Company, competition and other risks described in the “Risk Factors” section of the Company’s Annual Report on Form 10-K filed with the Securities and Exchange Commission ("SEC") on February 20, 2020, Quarterly Report on Form 10-Q filed with the SEC on November 5, 2020, and in other filings with the SEC. The forward-looking statements in this presentation represent the Company's view as of the date of this presentation and the Company does not assume any obligation to update any forward-looking statements, except as required by law.

CATALYST BIOSCIENCES

The Protease Medicines Company

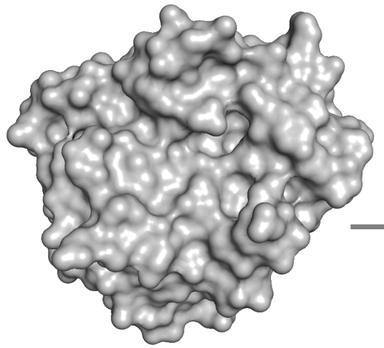
Harnessing the catalytic power of proteases

- Novel differentiated protease medicines
- Robust complement portfolio
- Clinical-stage hemophilia assets
- Late-stage asset in Phase 3

Catalyst's protease platform generates differentiated therapeutics

Unique expertise in protease biology enables design of optimized protease therapeutics

Protease scaffold



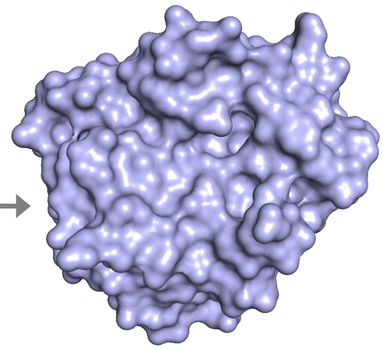
Discovery platform

- Structure guided design
- Molecular evolution
- Engineered regulation
- Pharmacokinetic improvement

Our proteases

+ Functionally enhance natural proteases in the complement & coagulation cascades
+ Engineer novel protein degraders in the complement cascade
+ Modulate or target biological activation or inactivation

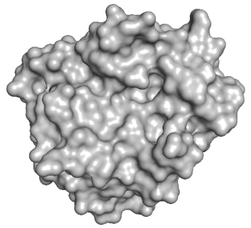
Therapeutic protease

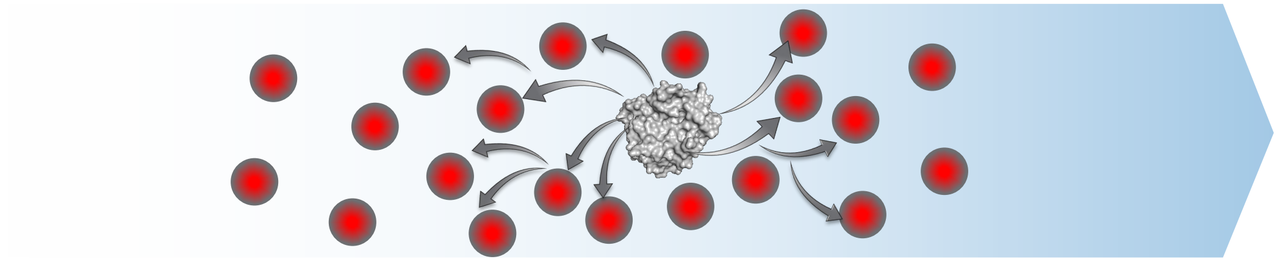


Proteases are ideal for high abundancy targets & cascades

A better way to regulate biological processes compared with antibodies & small molecules

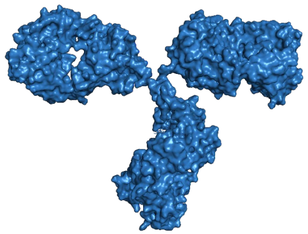
Proteases

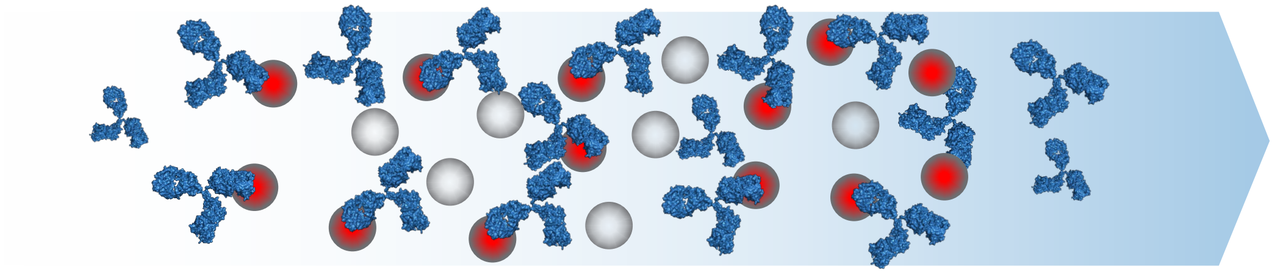




Efficient regulation at low concentrations of therapeutic protease

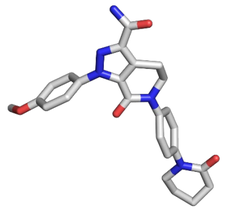
Antibodies

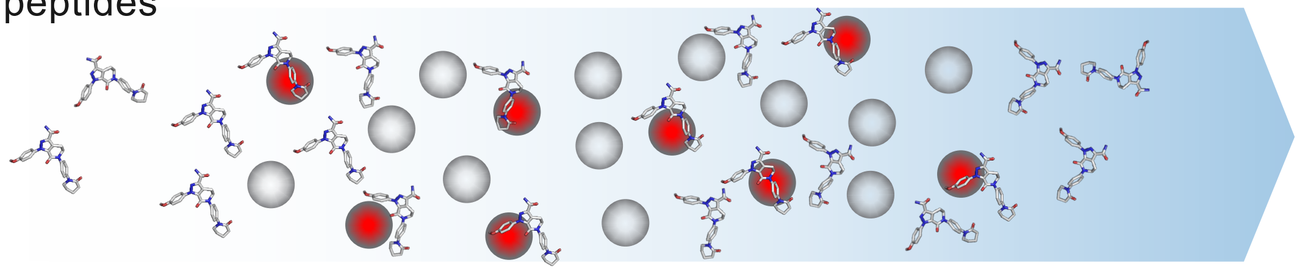




Requires high concentrations in excess of the target

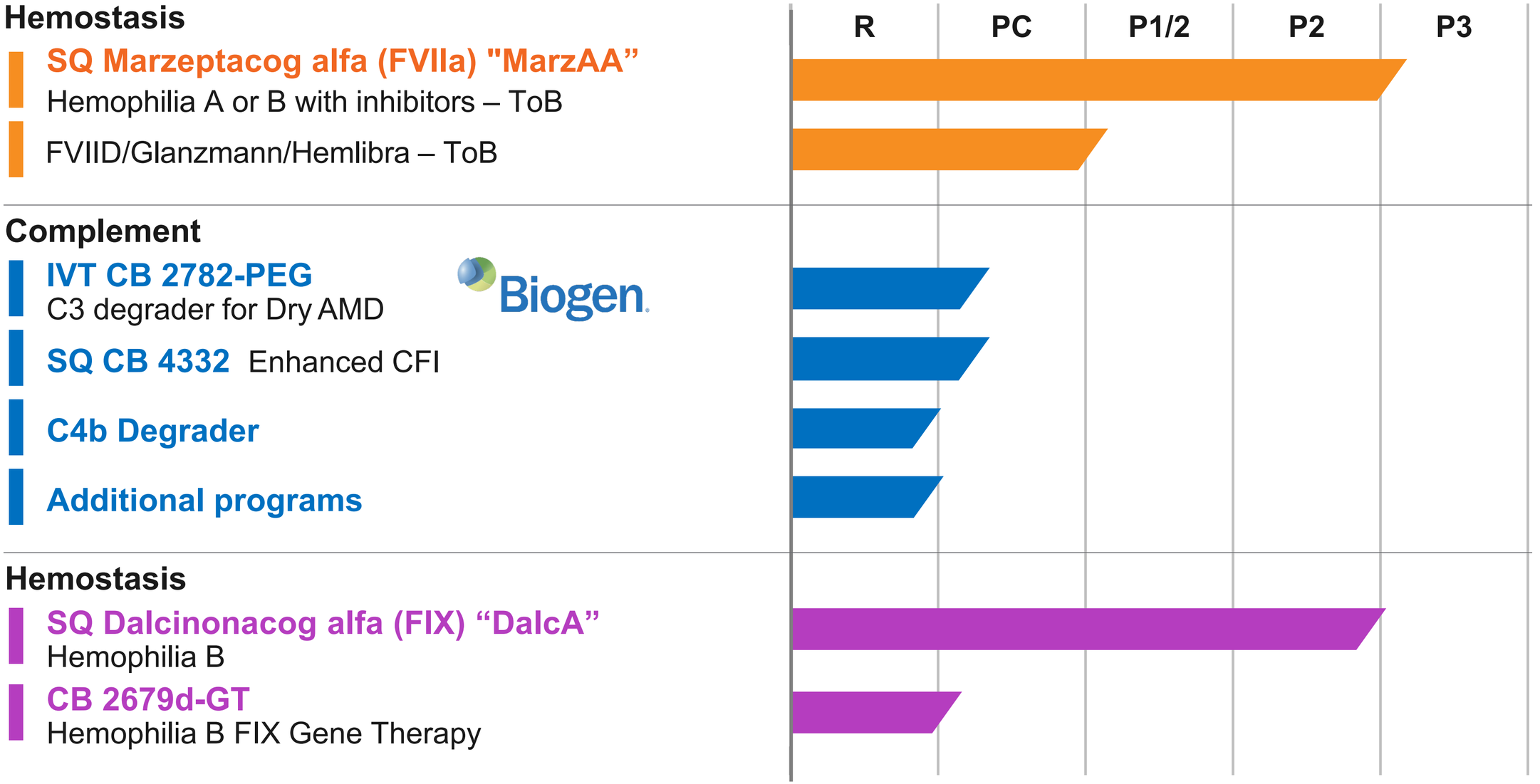
Small molecules / peptides





Requires high concentrations & frequent dosing

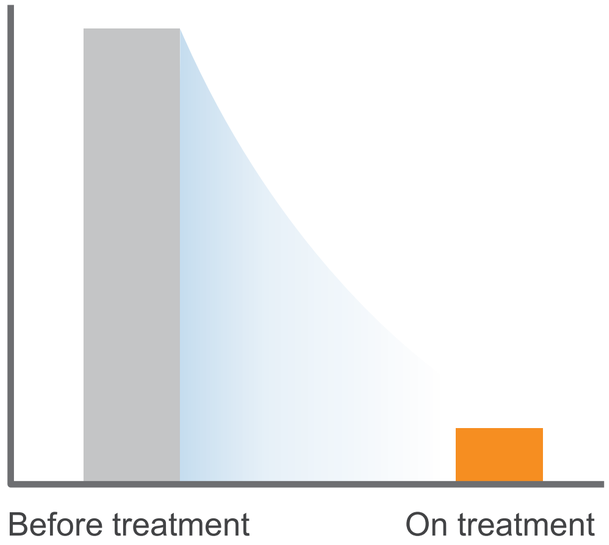
Pipeline



Clinical & partnering success of the CBIO protease platform

Marzeptacog alfa (activated)

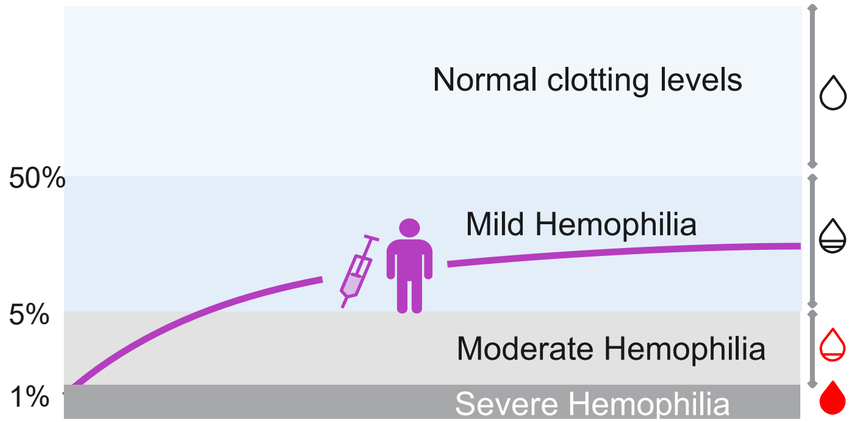
90% reduction
in annualized bleed rate



Engineered
rFVIIa protease

Dalcinonacog alfa

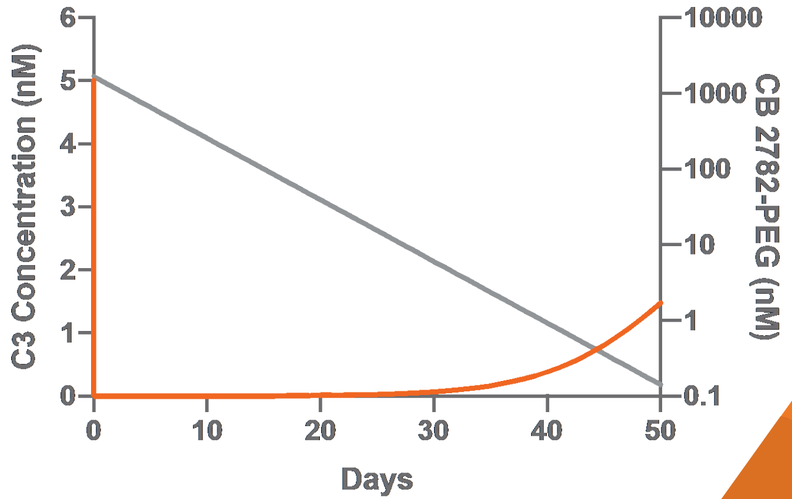
Achieved sustained
& high target levels of FIX



Engineered
rFIX protease

CB 2782-PEG Biogen

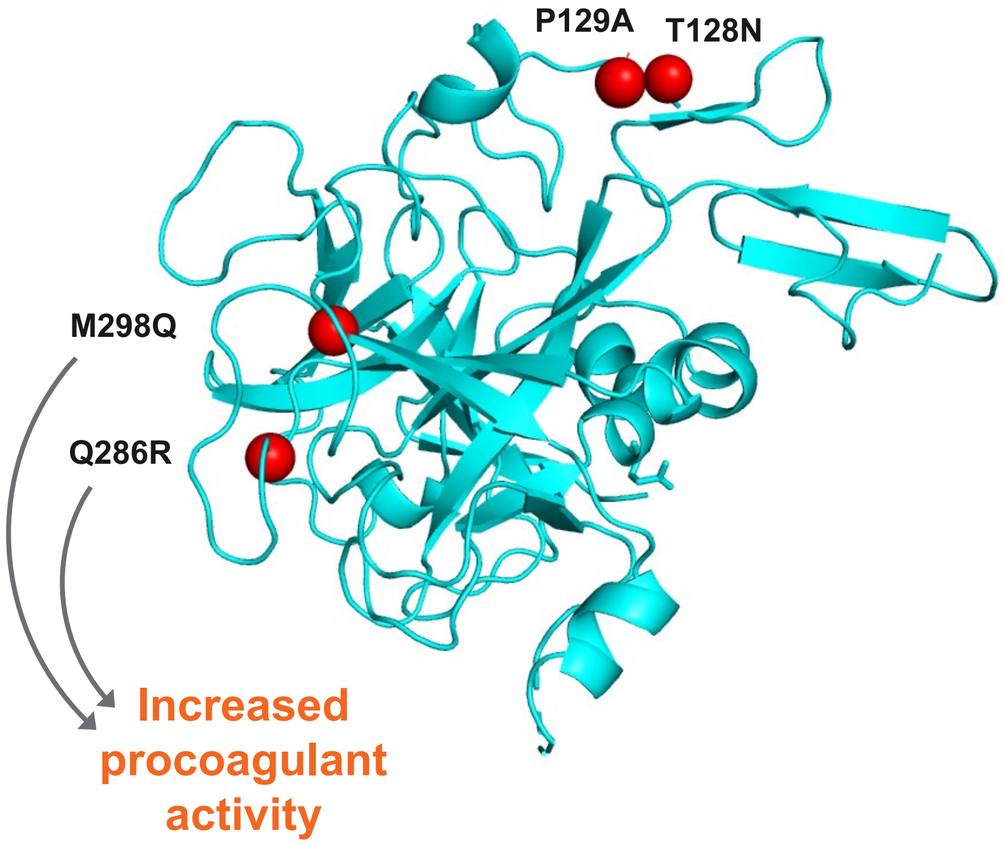
Best-in-class profile for dry AMD
Extended pharmacodynamics



Novel
C3 degrader

Marzeptacog alfa (activated) – MarzAA: SQ rFVIIa

Addresses a clear unmet need in hemophilia & other bleeding disorders



9-fold higher activity *vs* NovoSeven RT

+ Potency allows for SQ dosing that prolongs half-life
+ Simple, small volume SQ administration

Preclinical efficacy of SQ on-demand treatment

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

P2/3 prophylaxis efficacy & safety in HA or HB with inhibitors

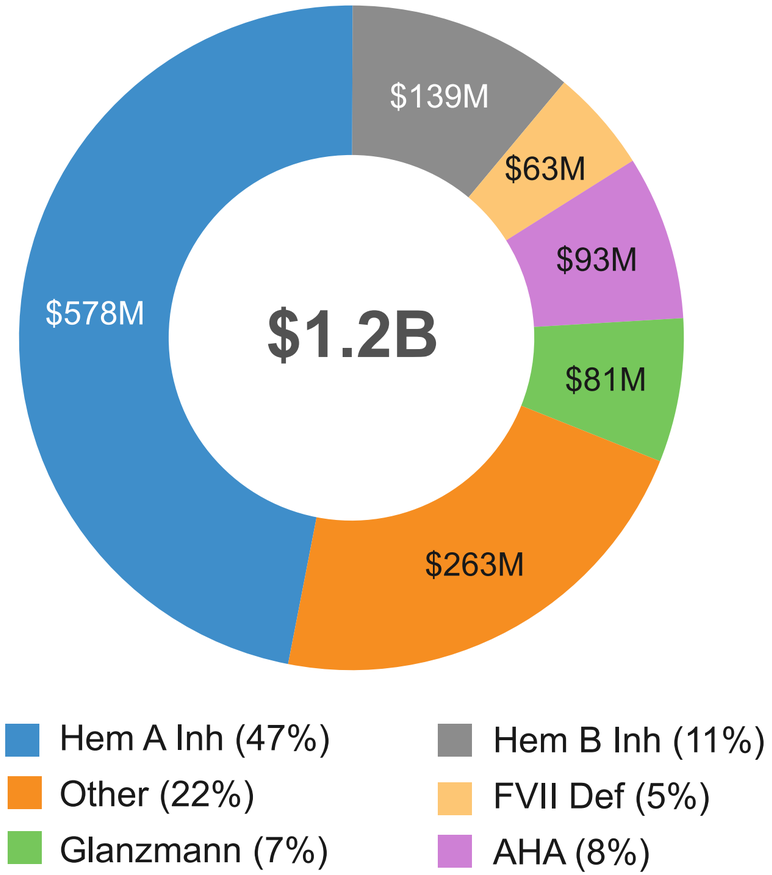
+ 46 patients treated including: single dose IV, up to 3 SQ doses/day, & daily SQ up to 97 days – no ADA

FDA Fast Track designation for treatment of episodic bleeding in Hem A or B with inhibitors

+ Granted on 2 December 2020

SQ MarzAA is a large commercial opportunity

Global NovoSeven sales breakdown by indication (2019)

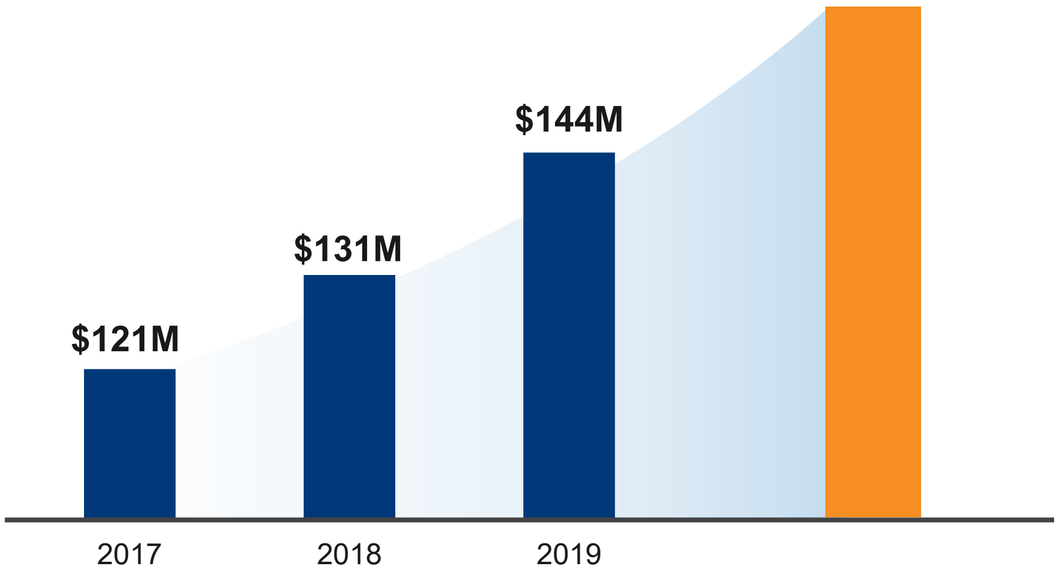


SQ MarzAA has a superior profile

- Faster & easier to administer *vs* N7 dosed every 2 hours IV until hemostasis
- MarzAA SQ half-life ~8x longer than N7
- Potential to control rebleeding
- Can be combined with Hemlibra *in vitro* without increased thrombogenicity
- Ideal for pediatrics and patients with venous access issues
- Prophylaxis efficacy demonstrated in P2

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

MarzAA could be the first prophylaxis for Glanzmann & FVIID



Growing number of Glanzmann Thrombasthenia and FVIID patients treated with NovoSeven

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

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

MarzAA

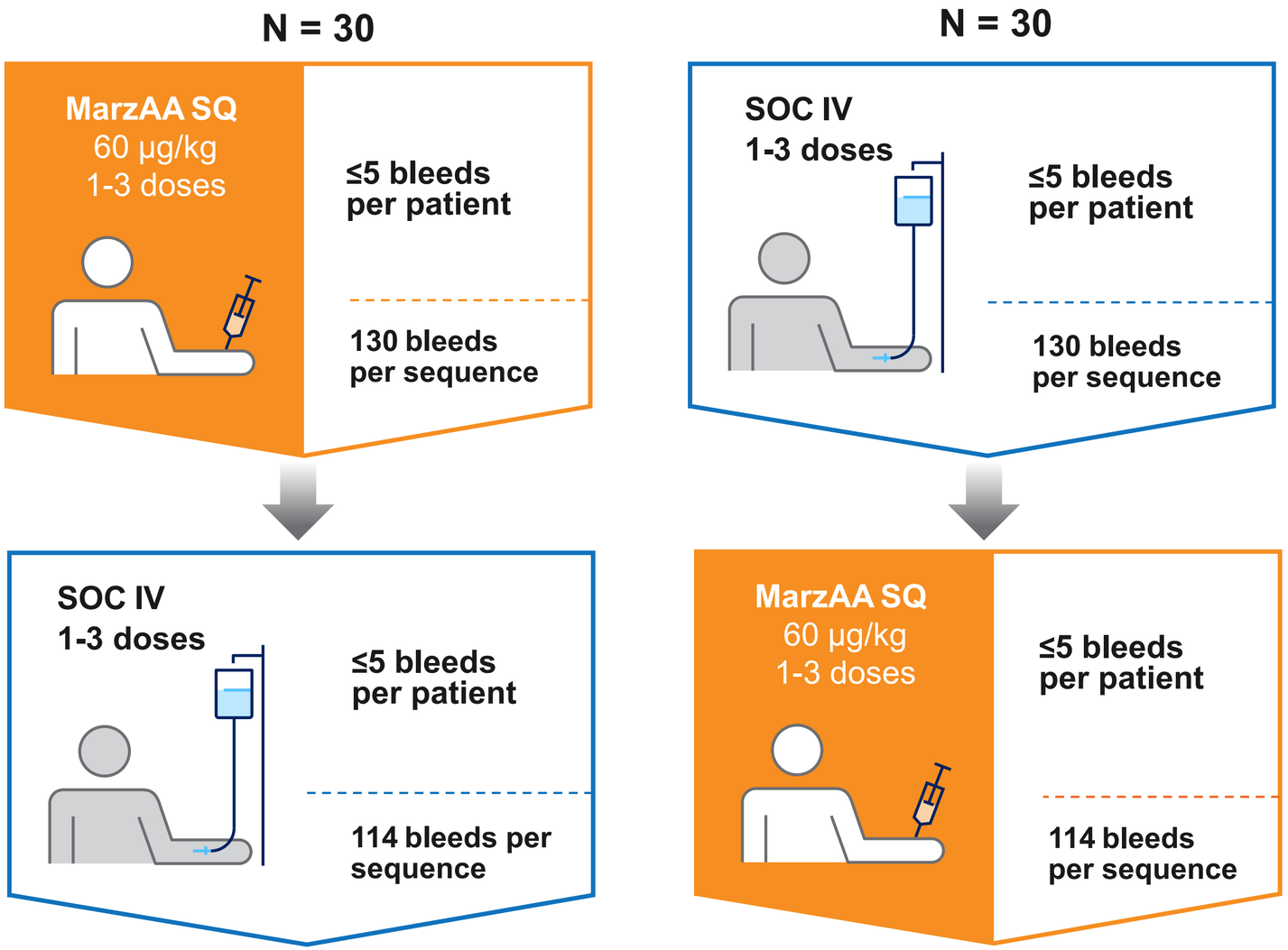
- MAA-102: PK MarzAA levels support SQ ToB
- Target levels are **rapidly achieved**
- Target levels can be maintained for 18 hours with a single SQ dose of 60 µg/kg

Clinical PK MarzAA levels support SQ ToB

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

Crimson 1 Phase 3 study: Treatment of episodic bleeding

Hemophilia A or B with inhibitors, ABR ≥ 8



- **Primary endpoint**

  Non-inferior hemostatic efficacy: standard 4-point scale at 24 h

- **Secondary endpoints**

  Time to bleed resolution; number of doses; rescue meds

- **Safety**

  Adverse events, anti-drug antibodies (ADA); thrombosis

Statistics

+ **SOC estimate 85%**
  Excellent/good treatment of bleeds
+ Non-inferiority margin of **12%**
+ **2.5%** significance, one-sided
+ **90%** power

MAA-202 Phase 1/2 study design

FVII deficiency, Glanzmann Thrombasthenia and HA on Hemlibra: N = 8 each

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

- **Phase 1**

  Primary endpoint:
  Pharmacokinetics

  Secondary endpoint:
  Pharmacodynamics

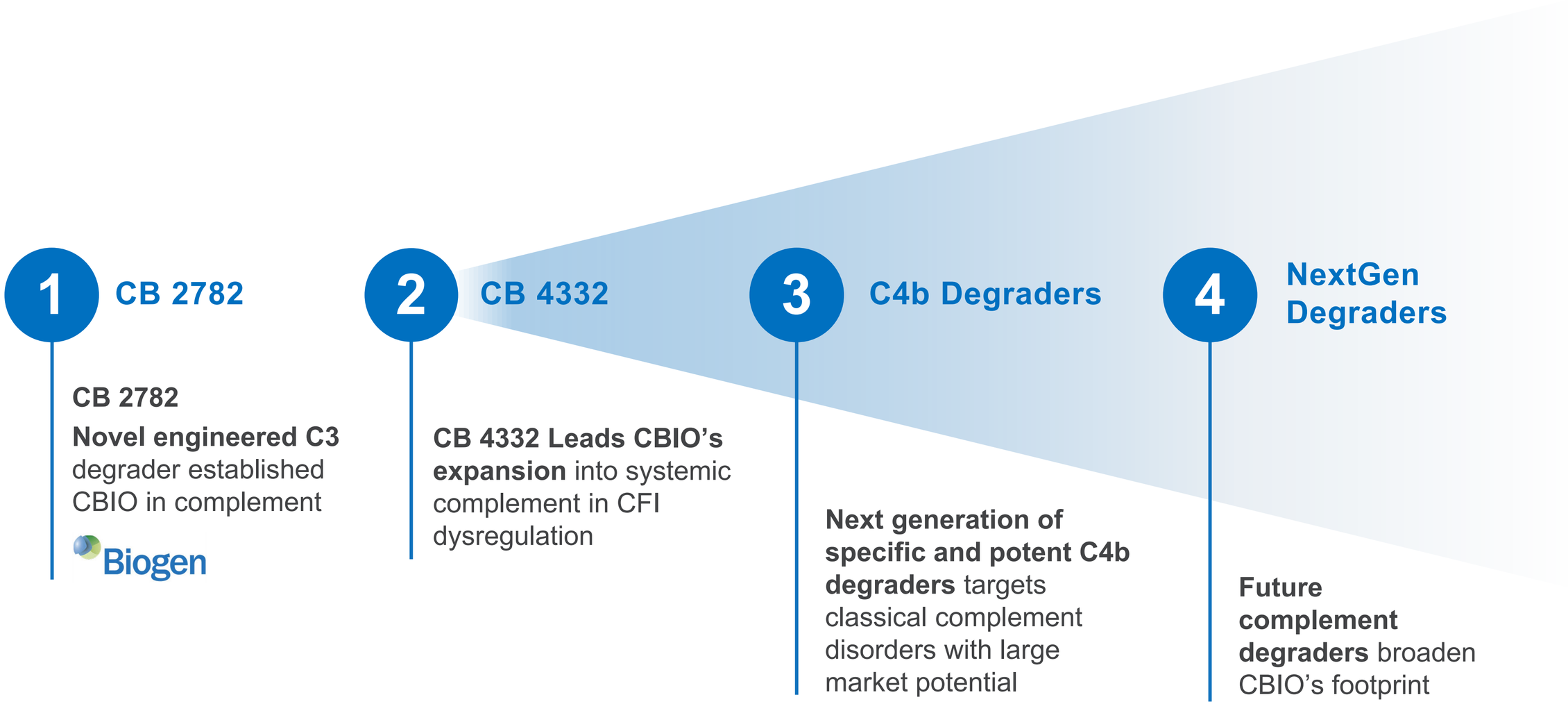
- **Phase 2 ToB**

  Primary endpoint:
  Hemostatic efficacy at 24 hours

  Secondary endpoints:
  Effective hemostasis at successive timepoints; doses needed; rescue meds

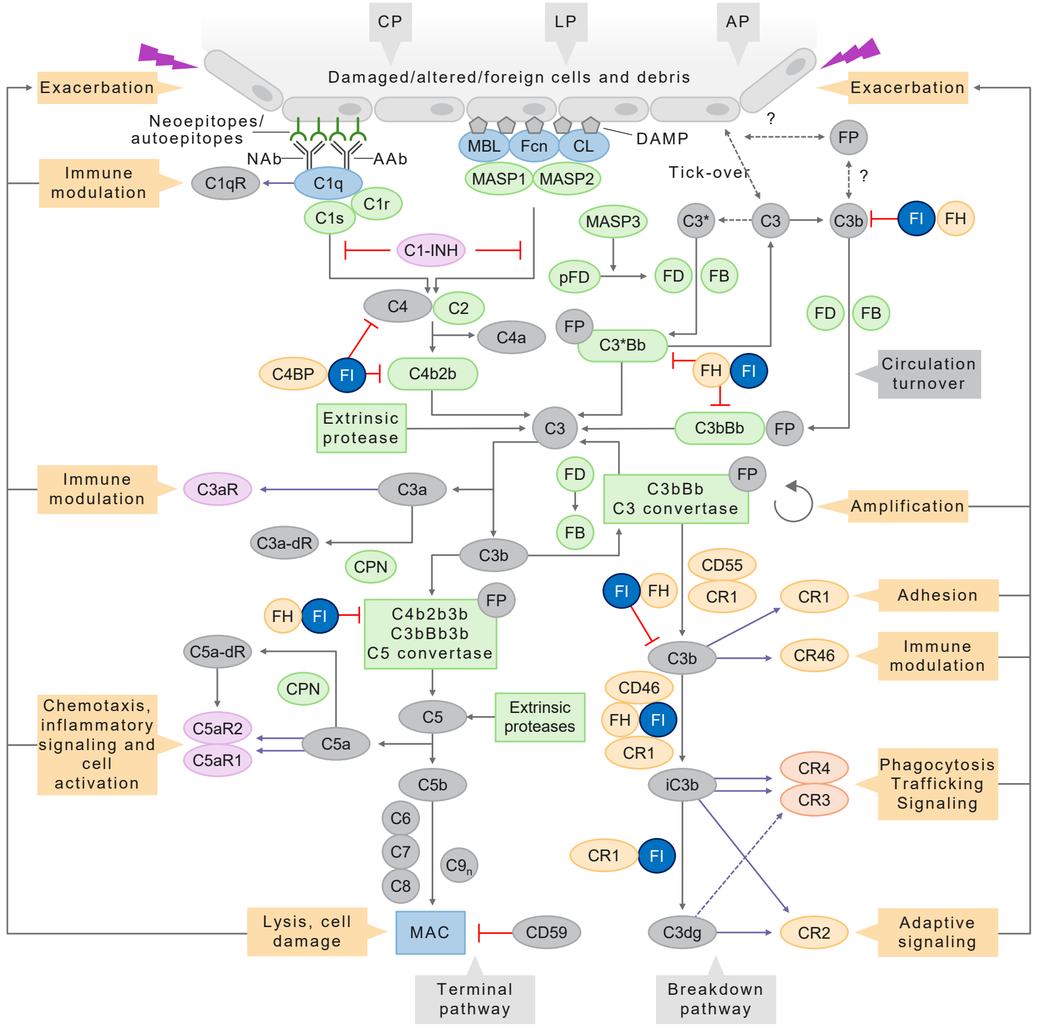
  Safety:
  Adverse events and ADA

CBIO’s complement pipeline



Complement is a perfect fit to develop protease therapeutics

The complement pathway is driven by a protease cascade



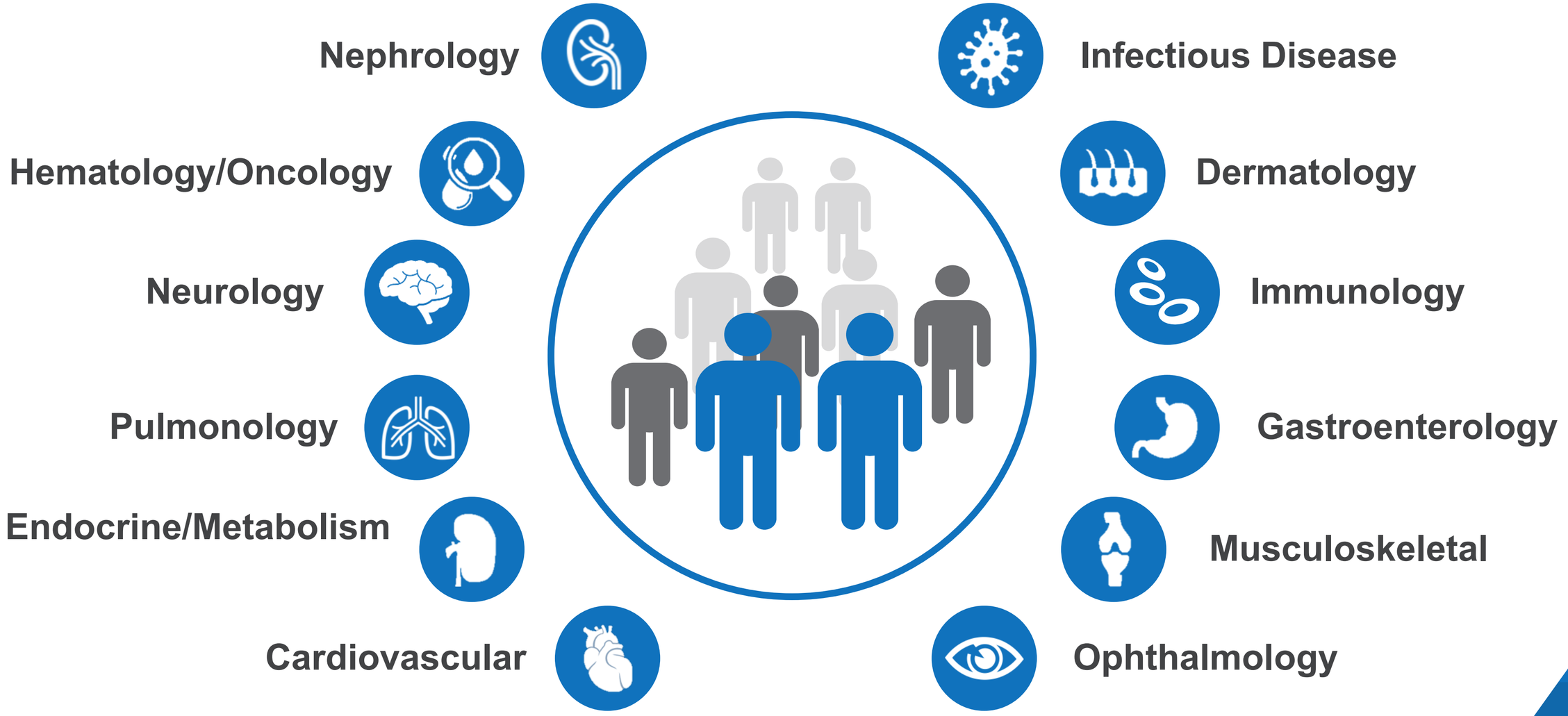
80%

of the complement cascade is regulated by proteases

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

Complement plays a critical role in many diseases

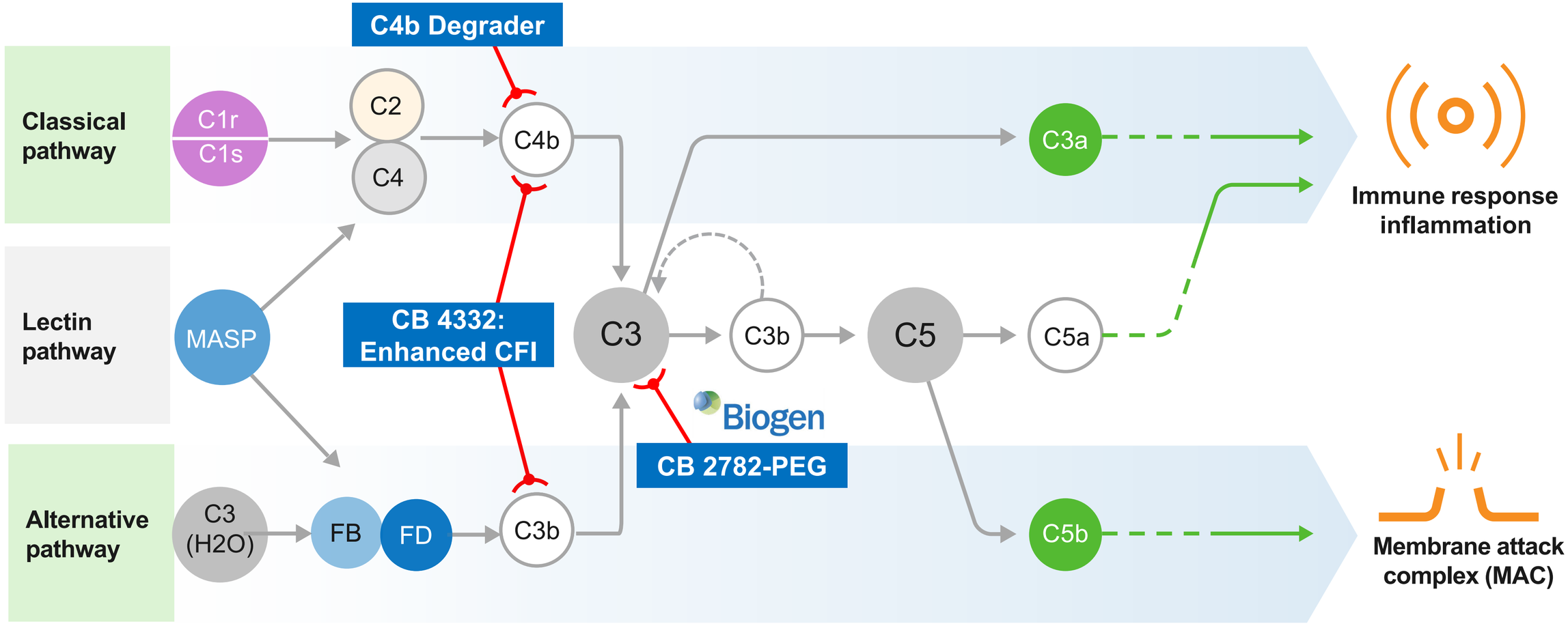
Late-stage complement therapies projected to achieve net sales over $12B by 2026



References: Globaldata consensus net sales forecast 2020

CBIO is taking a targeted approach to complement regulation

Engineered proteases address the root cause of the pathology

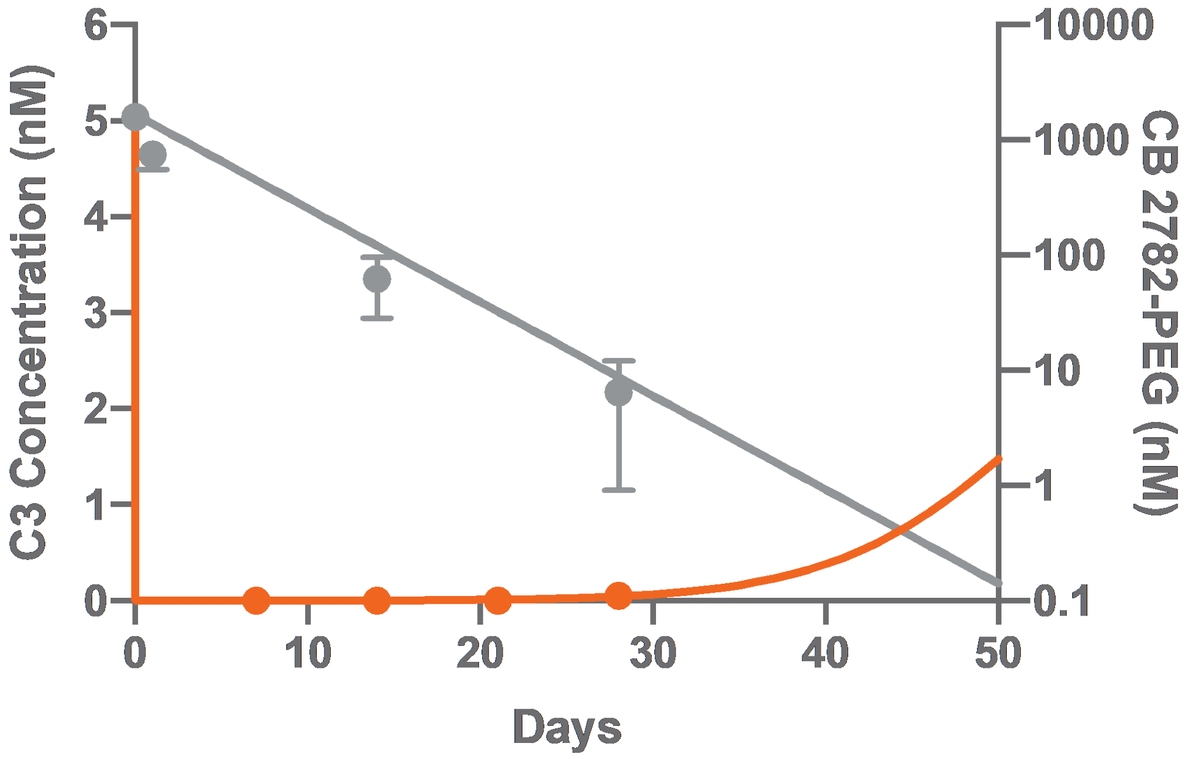


+ Current C5 blockade therapies do not address disease root cause leading to inadequate disease control
+ The catalytic power of proteases provides advantages over small molecules and antibodies

Protease advantage demonstrated *in vivo*

CB 2782-PEG – designed as a best-in-class C3 degrader in dry AMD

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

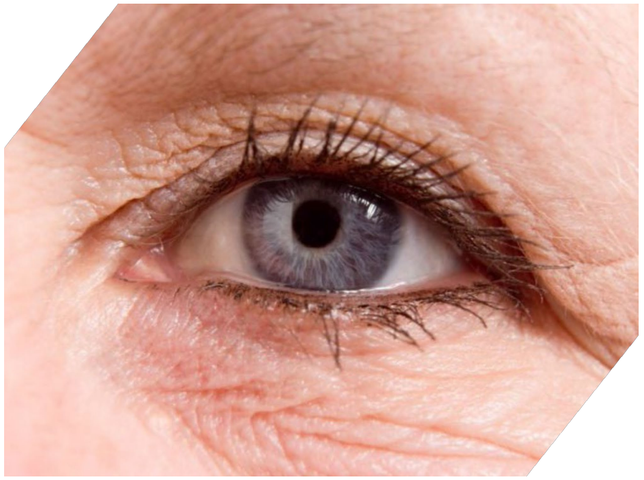


Catalytic advantage of proteases

- One therapeutic molecule neutralizes 1000s
- Fast & potent response
- Extended pharmacodynamic effect
- Can activate or degrade therapeutic targets
- Engineered novel protein degraders “sweep away” difficult to drug targets

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 therapy
+ Global market ~ >$5B
+ C3 is a clinically validated target (randomized P2) for the treatment of 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
+ Preclinical NHP PK & PD data* predict **best-in-class** human intravitreal **dosing 3 or 4 times a year**

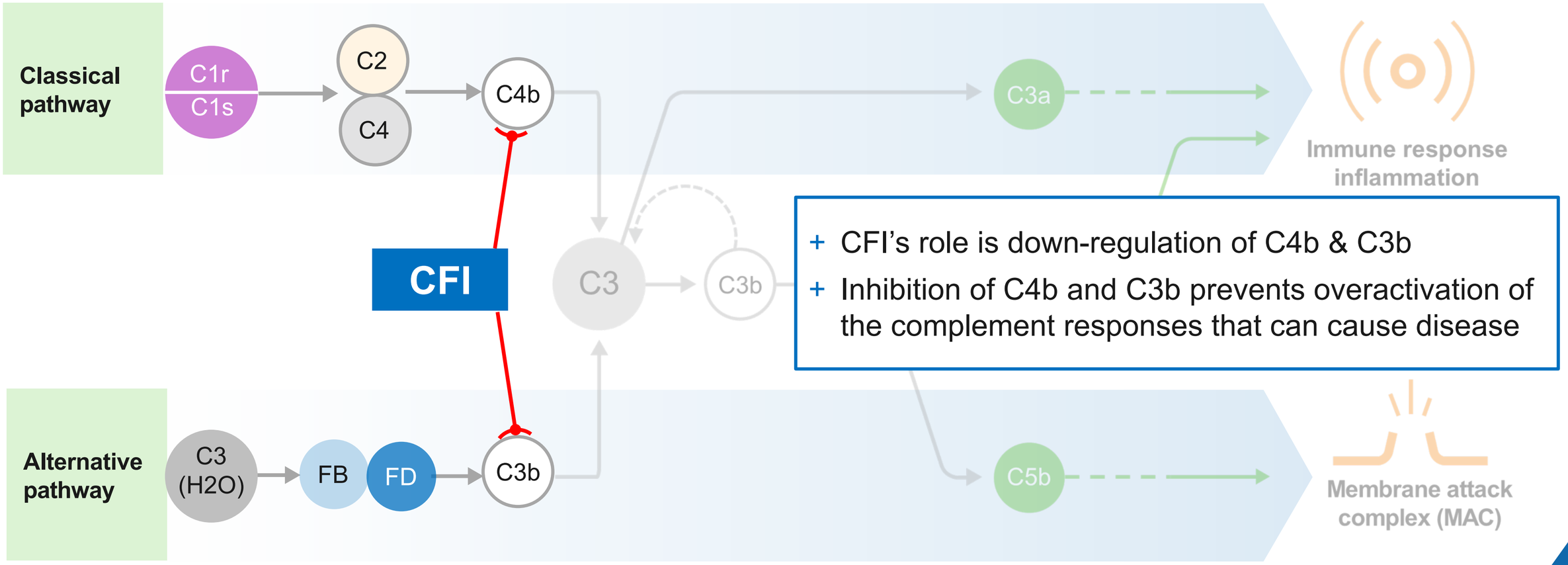
Biogen collaboration

+ $15M upfront, up to $340M in milestones and tiered royalties up to low double digits
+ Catalyst: fully funded pre-clinical and manufacturing activities
+ Biogen: IND-enabling activities, WW clinical development & commercialization

Biogen

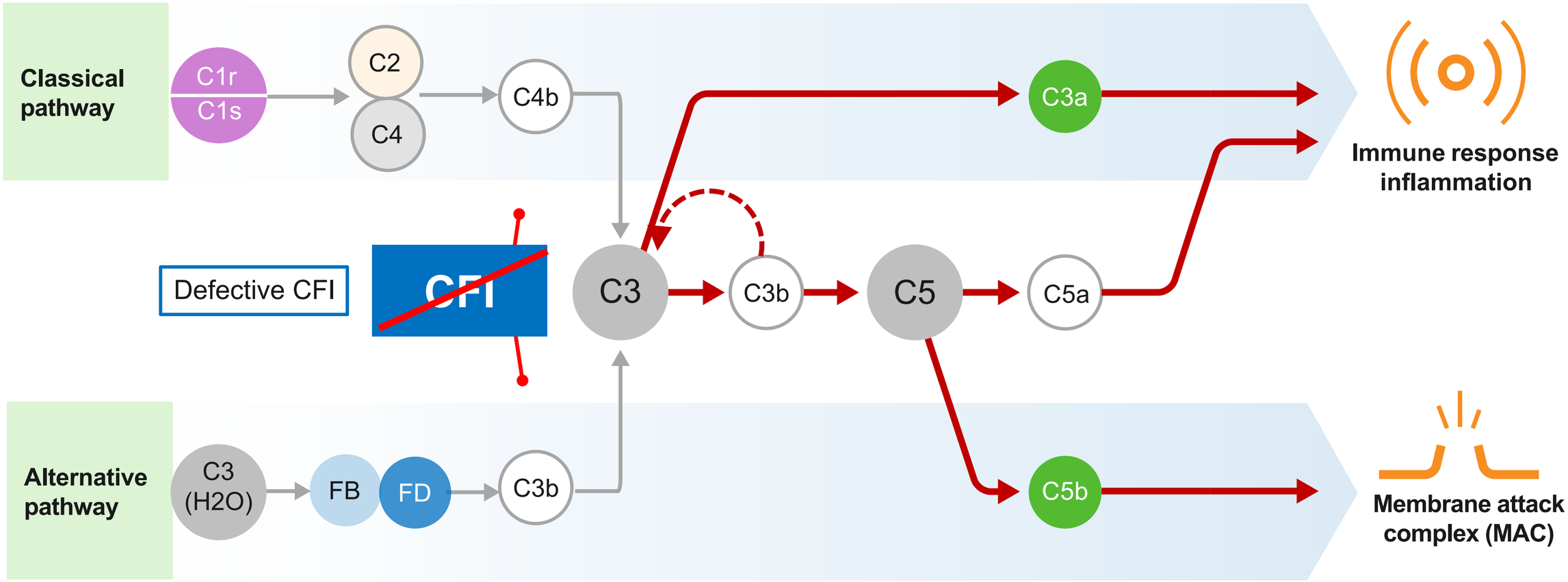
*Furfine *et al.* ARVO 2019

Normal CFI: Key central regulator of complement activation



+ CFI's role is down-regulation of C4b & C3b
+ Inhibition of C4b and C3b prevents overactivation of the complement responses that can cause disease

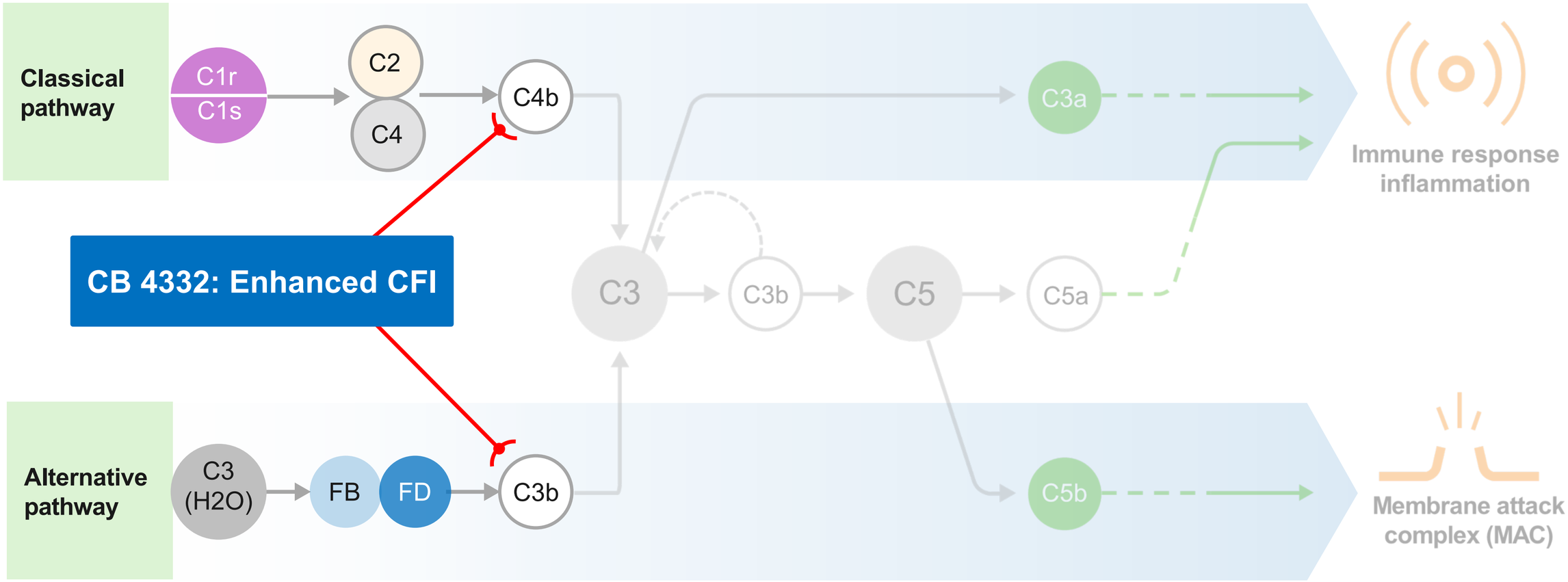
CFI dysregulation: Lack of proteolytic CFI activity causes disease



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

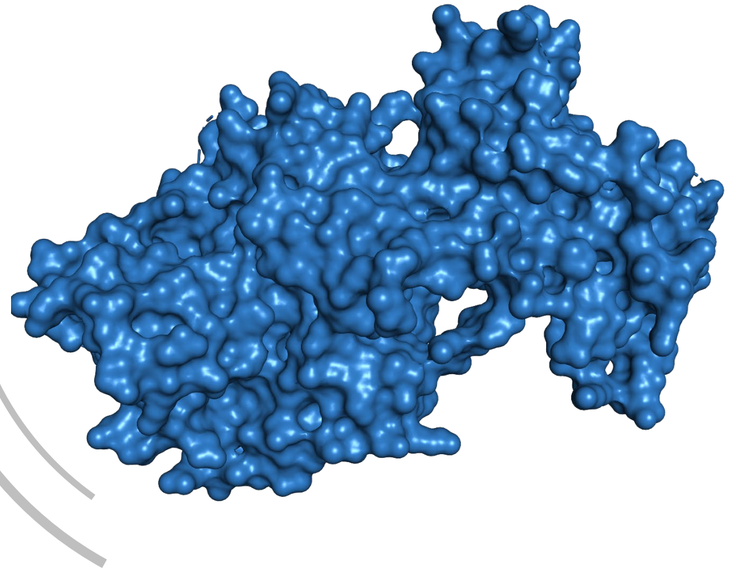
CB 4332 – CBIO's enhanced CFI

Specifically addresses the problem by restoring CFI regulation



CB 4332: Enhanced Complement Factor I

CBIO's next SQ development candidate to restore CFI regulation



+ **Engineered for an extended half-life**
  - Once weekly SQ therapy – no PEG
+ **Full activity comparable to native CFI**
  - Classical and alternative pathway regulation
+ **Efficient high yield production process**

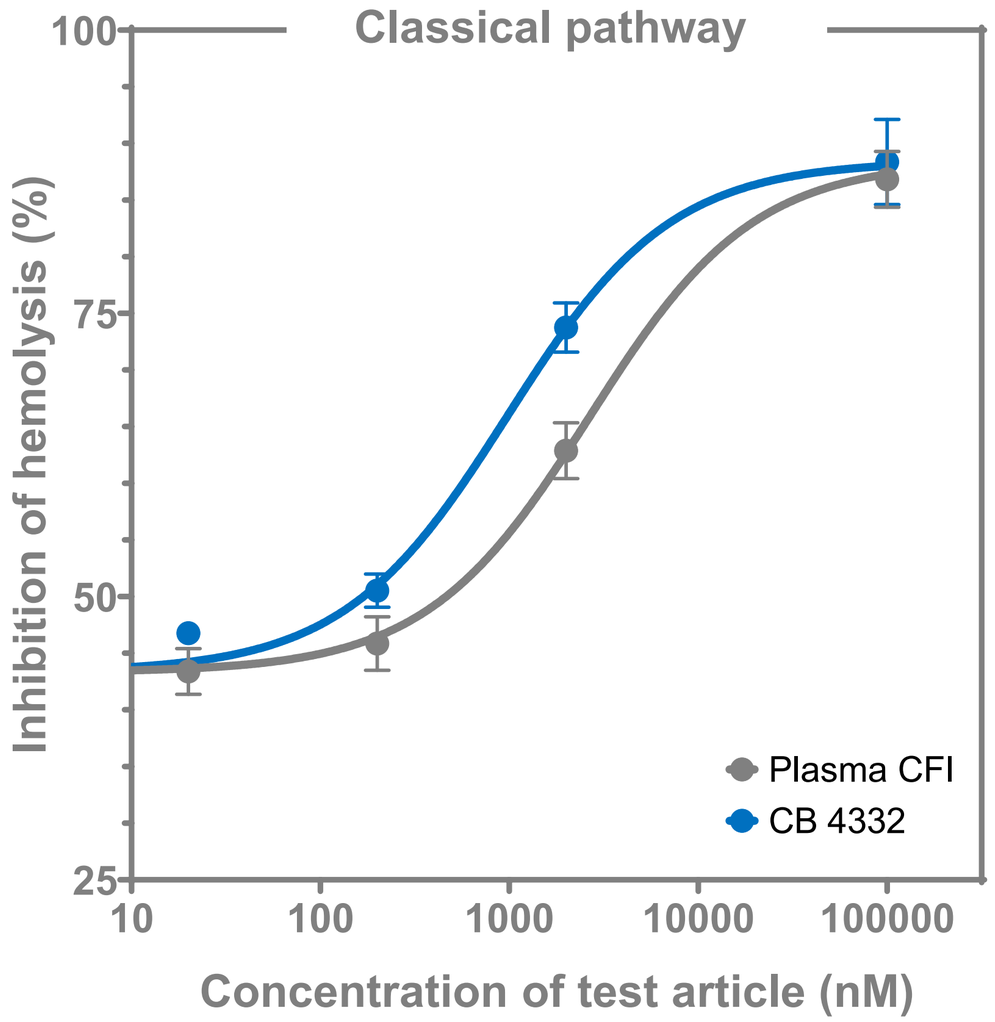
Rationale & unmet need

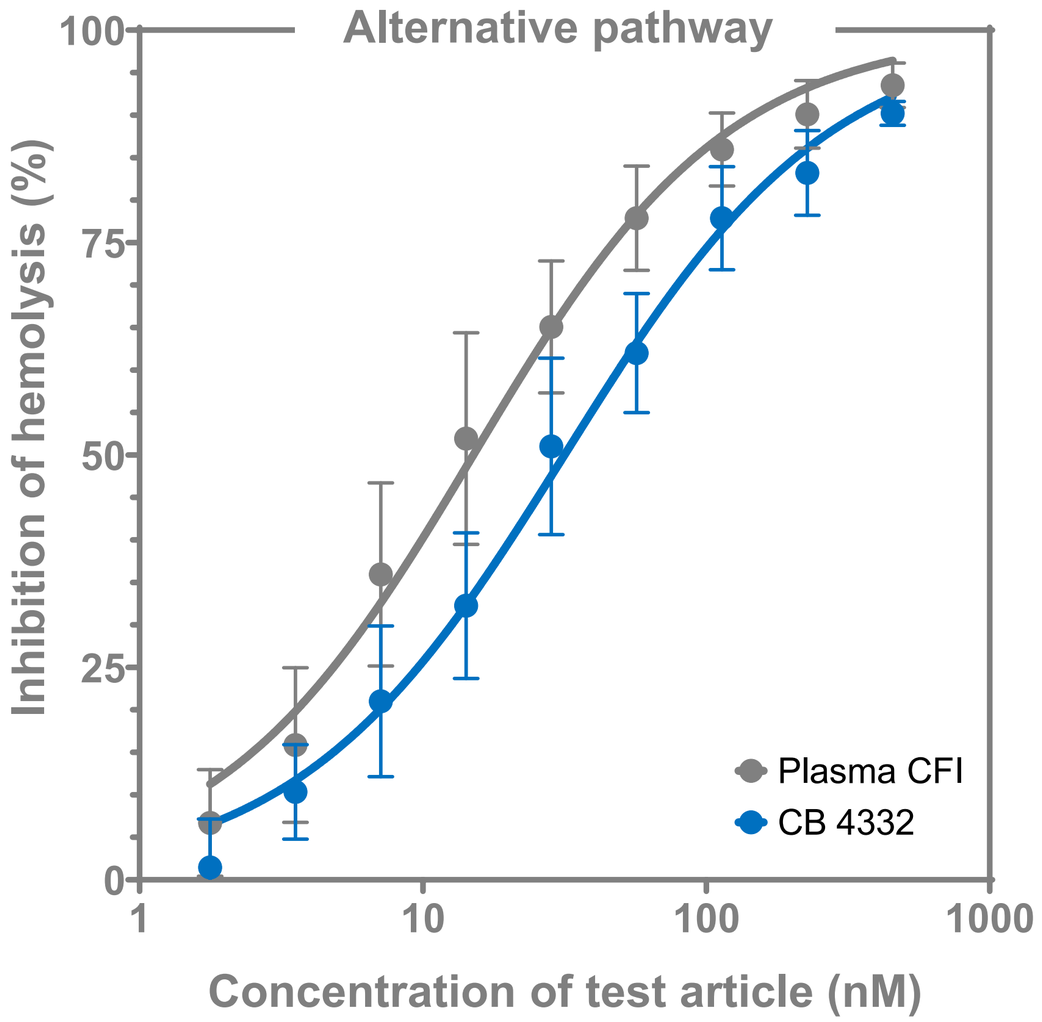
+ **Restores normal complement system** in patients with dysregulated CFI
+ **No specific therapies exist** to correct CFI dysregulation
+ Targets population with **no treatment or who respond poorly to current treatments**[1,2]
+ **Genetically defined patient population**

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

.

CB 4332 & plasma CFI perform similarly in human serum

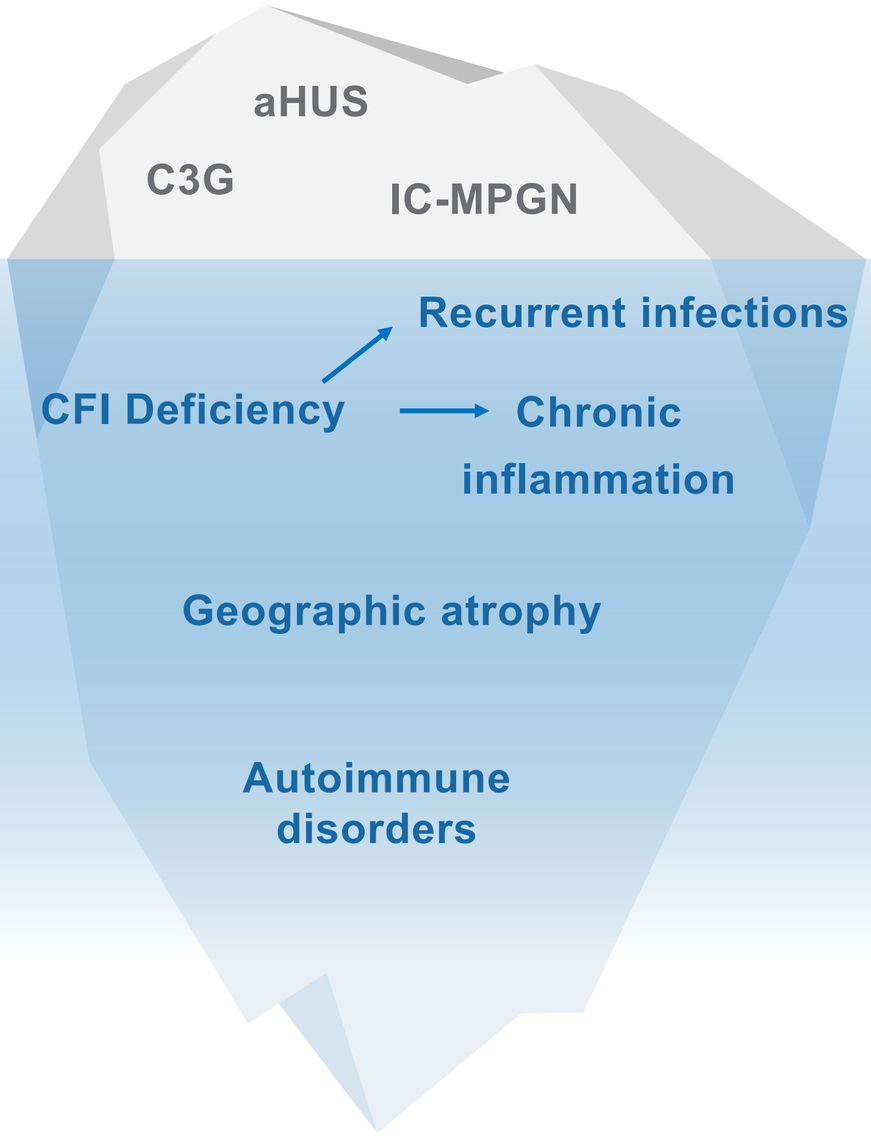




Diseases with CFI mutations have tremendous potential

Current development targets — **$500M**

Potential targets — **$10B+**



CB 4332 initial market opportunity

US / EU5 market opportunity

Additional opportunity in CFI Deficiency outside nephrology

$500M

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

Unmet needs

Significant opportunity for patients with CFI mutations

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

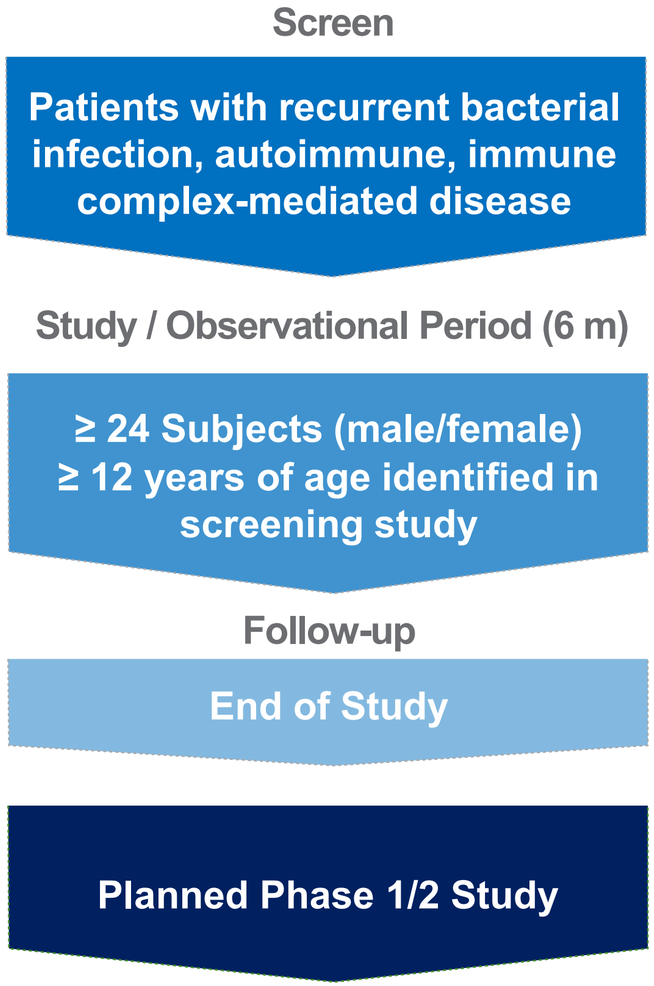
CFI mutations are significant drivers of disease

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

References: 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; Iatropoulous *et al*. 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*. Front Immunol. 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 Soc Nephrol. 2010; CBIO KOL interviews

CB 4332 – CFI dysregulation observational study

Natural history of CFI deficient patients for subsequent CB 4332 treatment



- **Primary Objective**

  Demonstrate the phenotypic manifestation of CFI mutations in recurrent bacterial infection, autoimmune, immune complex-mediated disease as a prelude to a Phase 1/2 study

- **Secondary Objectives**

  Monitor efficacy / disease status over time during SOC

  Monitor safety and tolerability of SOC

  Record dosing and compliance with SOC

  Monitor QoL measures

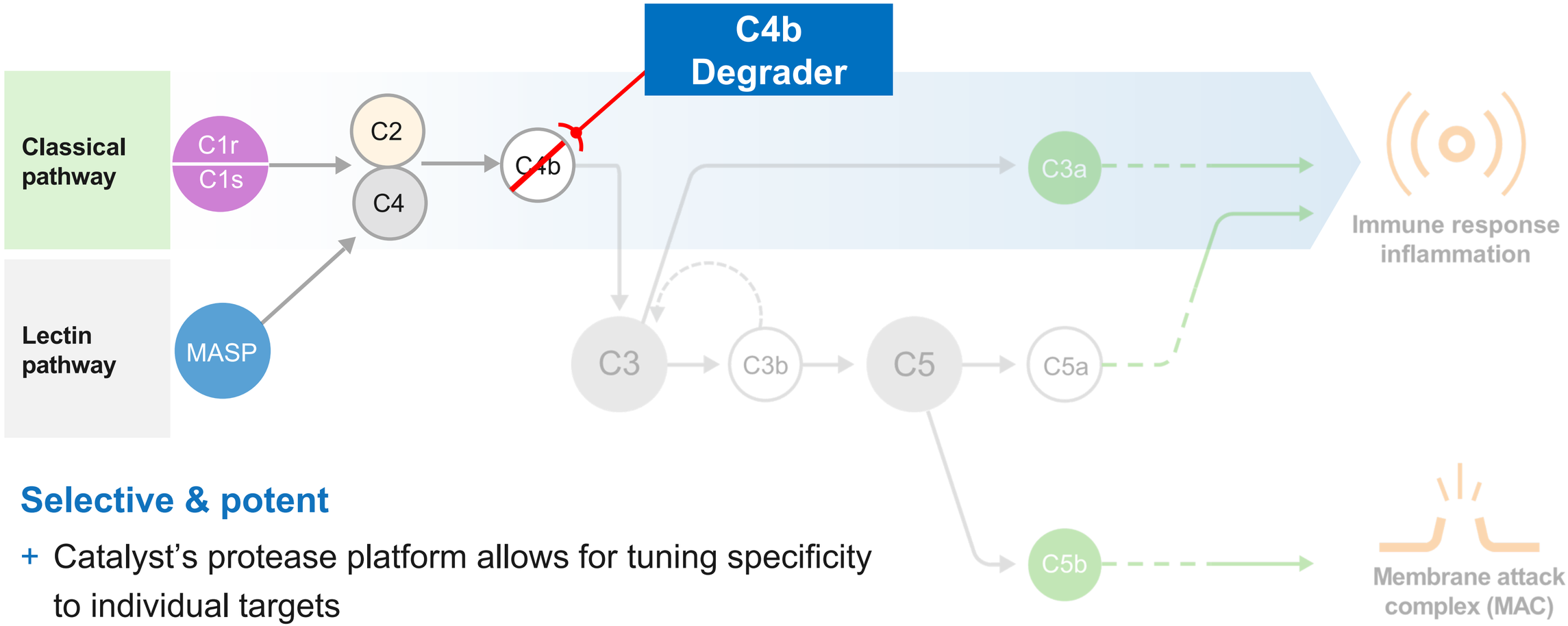
Timeline

Observational stage to start enrollment mid-2021

Global phase 1/2 in patients with CFI deficiency expected in 2022

Intend to pursue an accelerated regulatory path

CBIO C4b degrader complement therapy



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 or planning a weekly SQ injection
  - Approaches targeting C1q and C1s with antibodies require substantial & frequent IV dosing

C4b degraders target multiple high unmet need diseases

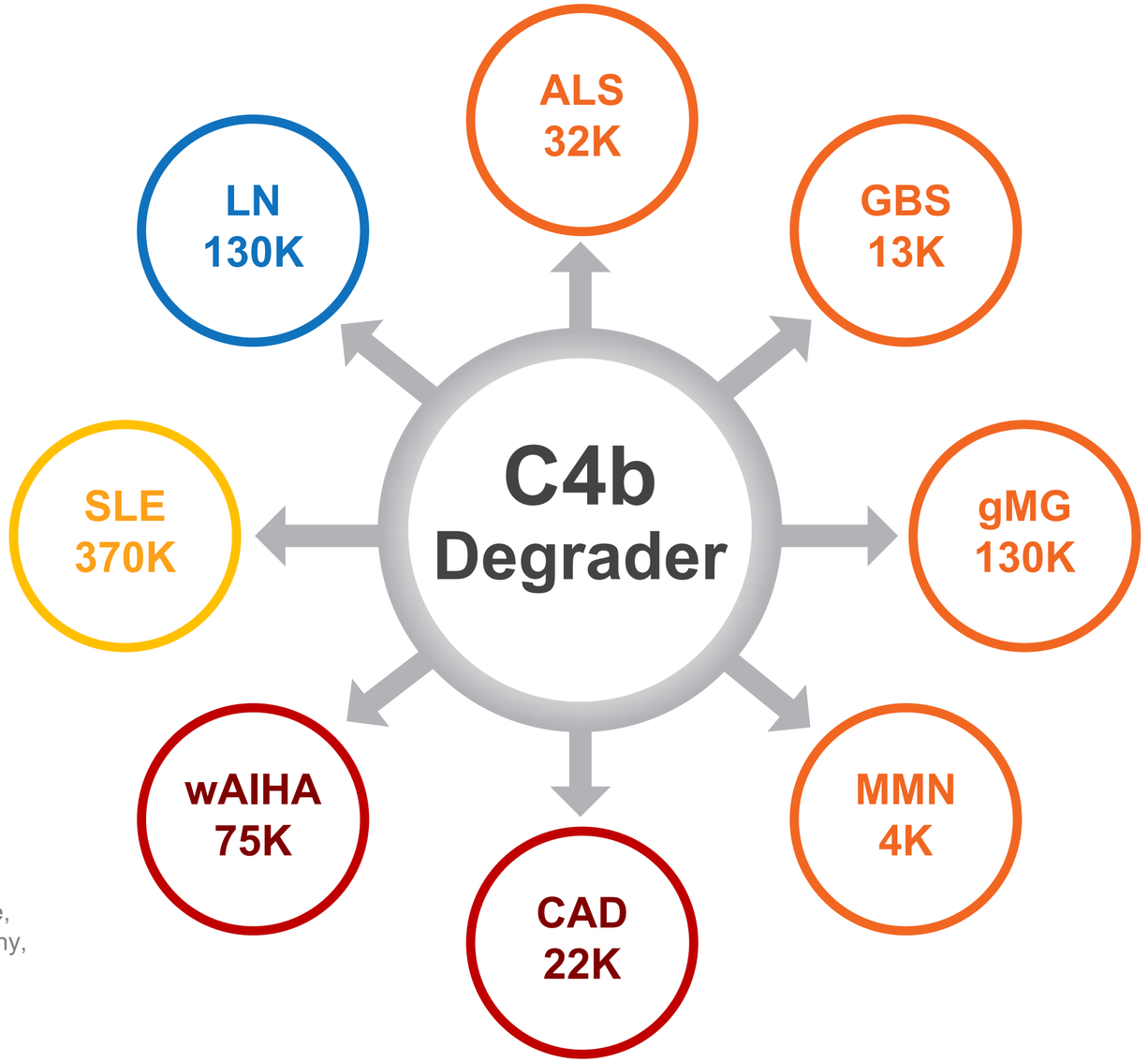
US & EU5 patient opportunity

Nephrology

Immunology

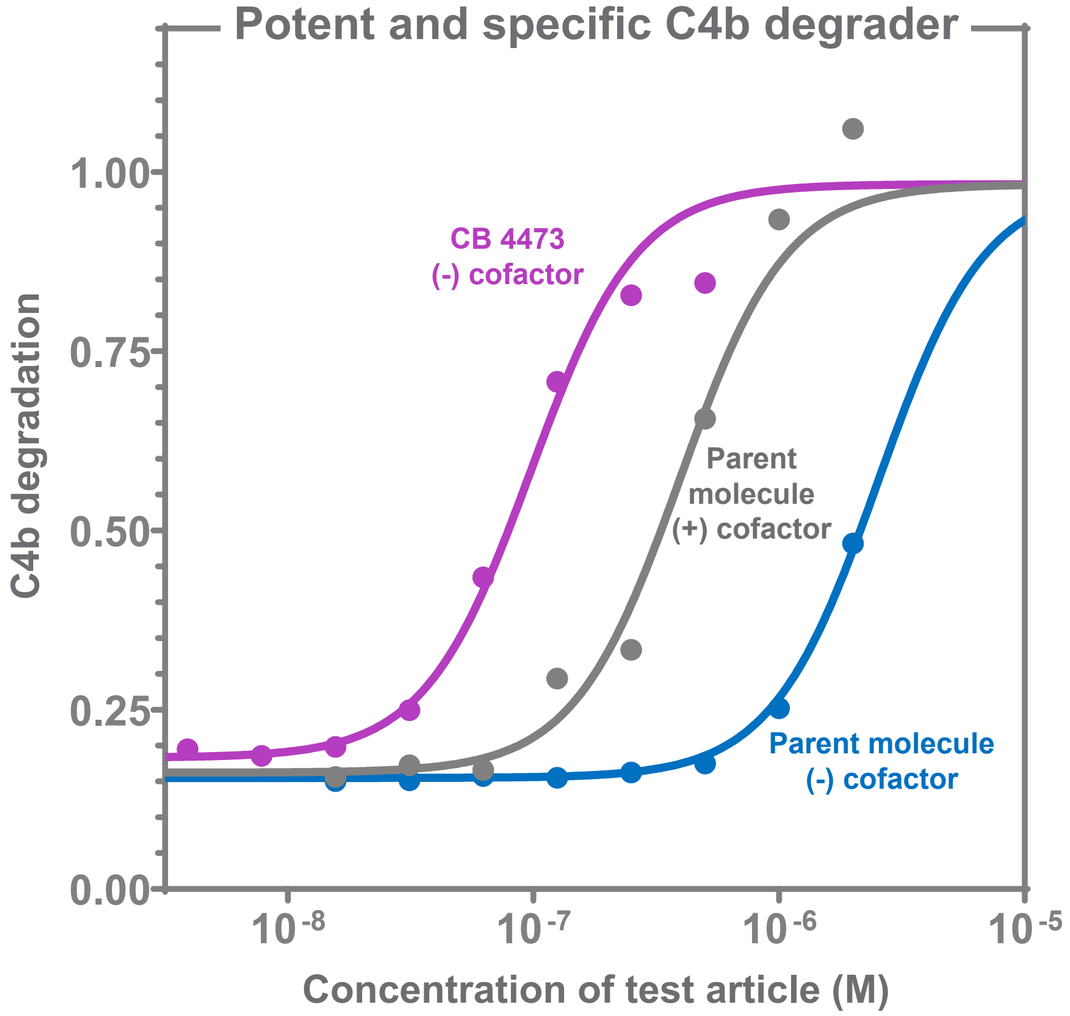
Hematology

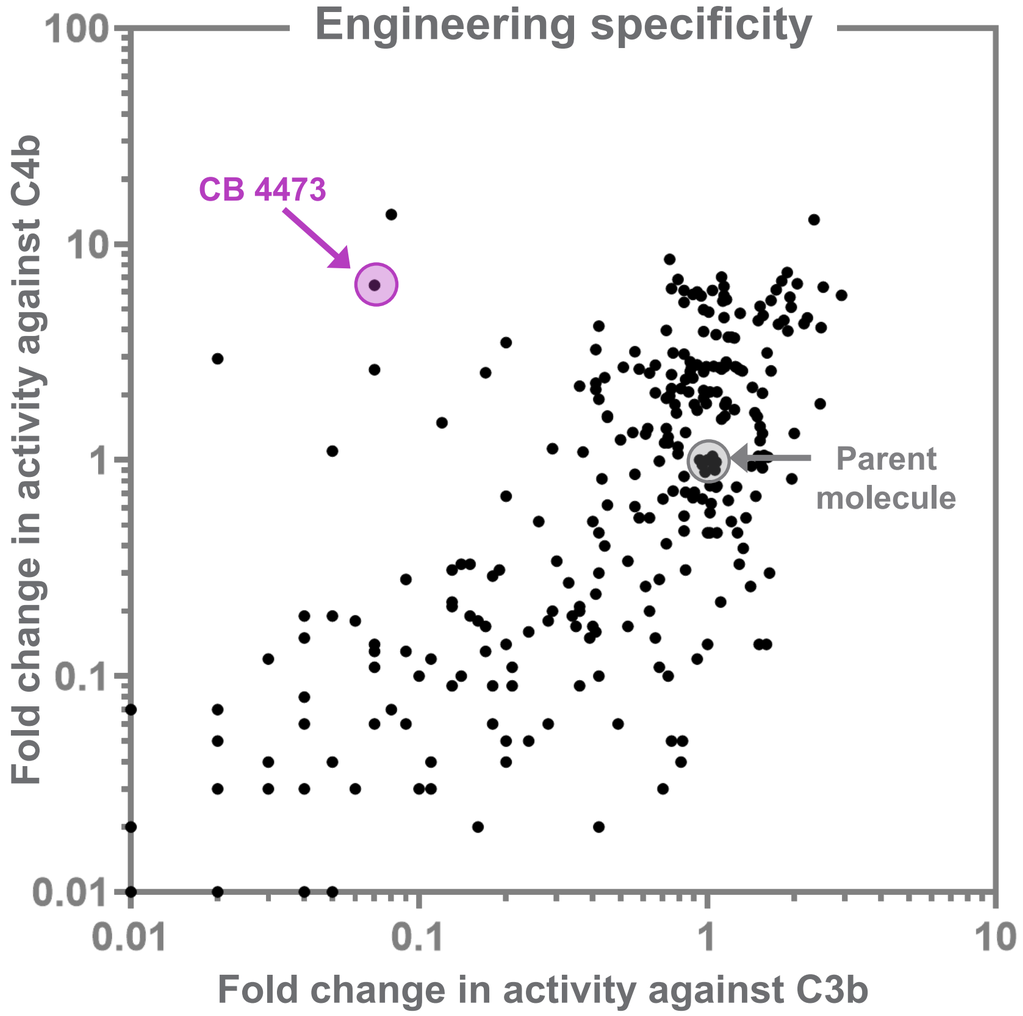
Neurology



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 potency & specificity





Milestones

2021

- CB 4332 observational trial
- MAA 202 PK data
- MAA 304 first DSMB
- C4b degrader updates
- CB 2782-PEG Biogen

Mid-year 2022

- MAA 304 final DSMB
- MAA 304 ToB data
- MAA 202 ToB data
- CB 4332 P1/2
- C4b degrader updates
- CB 2782-PEG Biogen

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

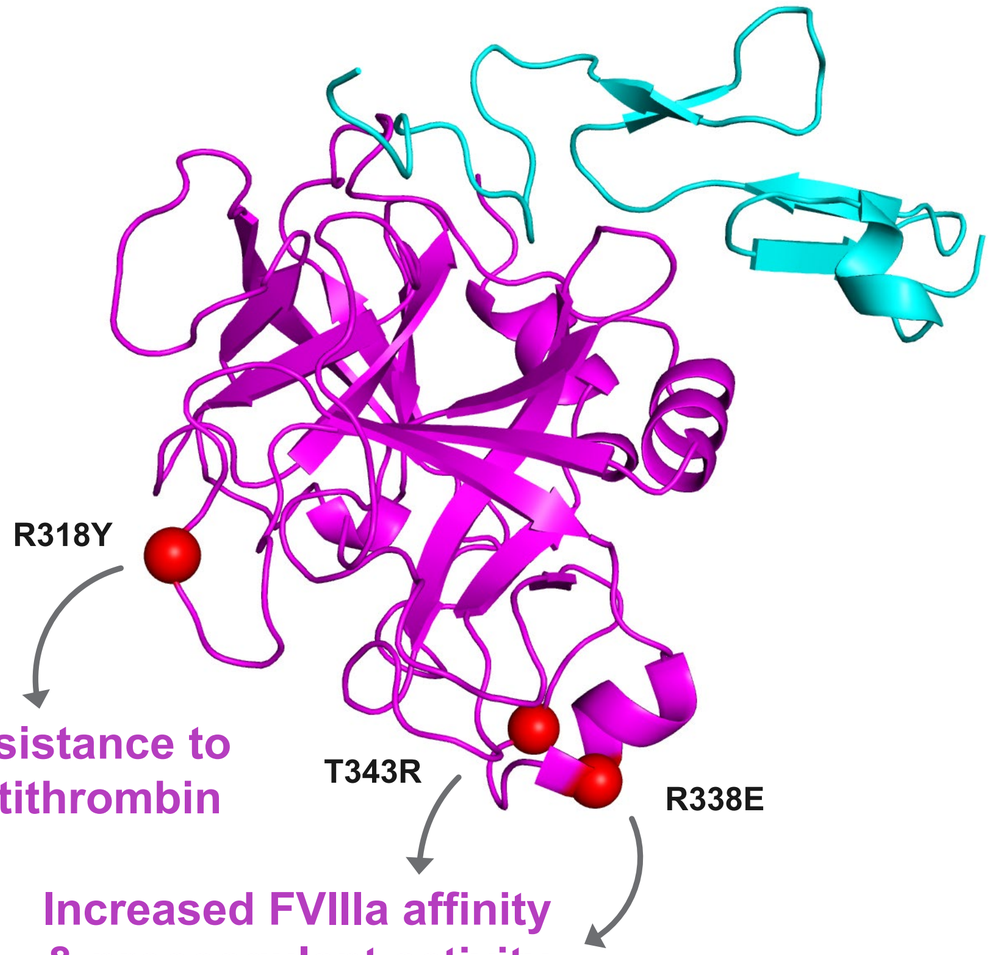
THANK YOU

Nasdaq: CBIO

CatalystBiosciences.com

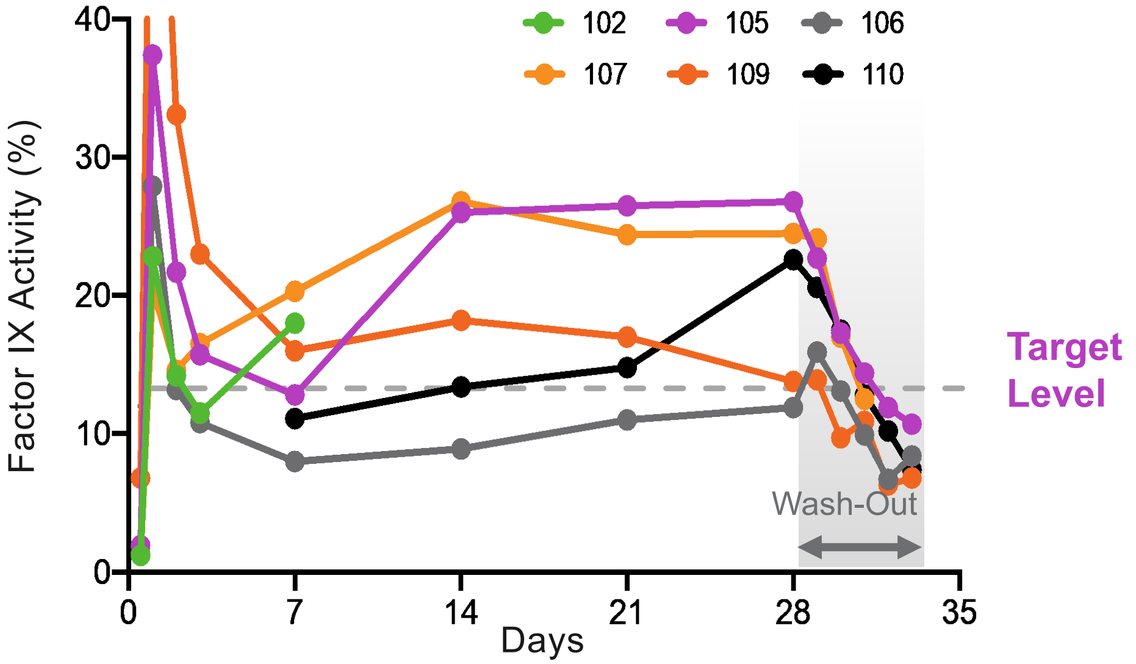
CATALYST BIOSCIENCES

DalcA P2b demonstrated efficacy & safety

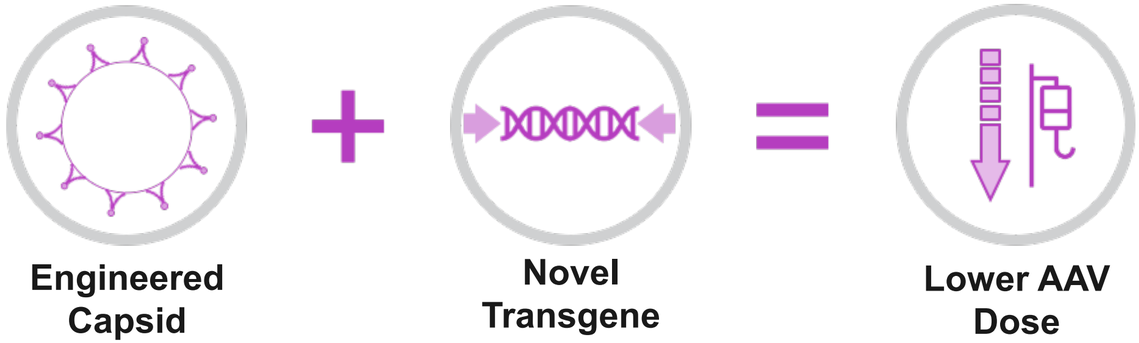


Differentiated from marketed IV FIXs

+ Small volume SQ administration
+ Enhanced pharmacokinetics with prolonged half-life
+ Excellent extravascular distribution
+ **Target levels >12% achieved with daily SQ 100 IU/kg dosing for 28 days**



Catalyst's CB 2679d gene therapy for hemophilia B



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

CB 2679d-GT has a superior profile *vs* Padua in preclinical studies

+ Stable high activity levels with 1/10th vector dose in mouse model
+ 4 to 5-fold reduction in bleeding time when compared to the Padua
+ Potential for improved efficacy & safety at 1-2 log reduced dose

Achieved high initial FIX levels in NHP

+ Presented at World Federation of Hemophilia Virtual Summit 2020
+ Additional vector optimization & dose ranging studies ongoing

Wholly-owned & issued patents covering gene therapy