Nasdaq: CBIO

CATALYST BIOSCIENCES

Corporate Overview

28 September 2021

CatalystBiosciences.com

CATALYST BIOSCIENCES

Forward looking statements

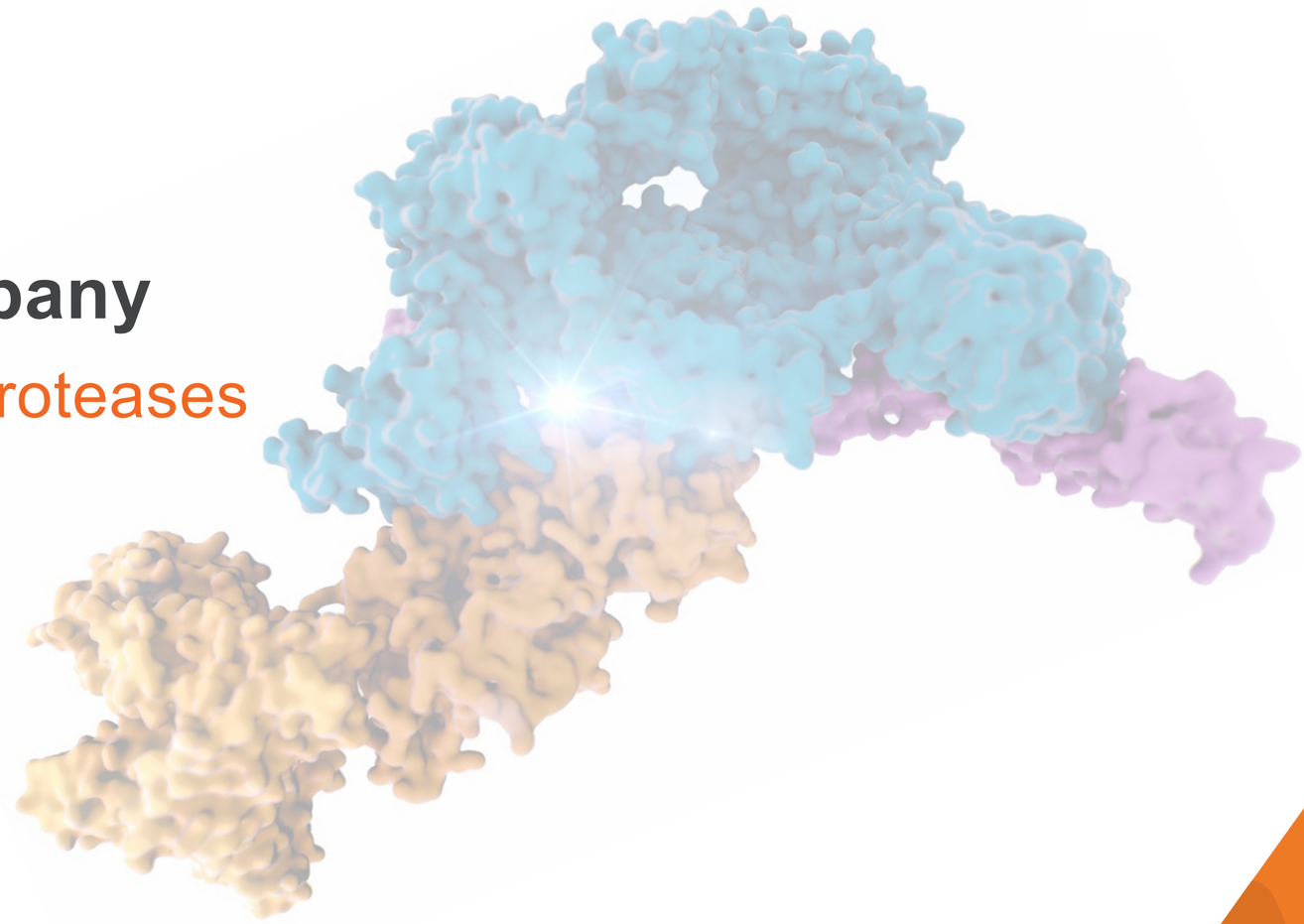
Certain information contained in this presentation and statements made orally during this presentation include forward-looking statements that involve substantial risks and uncertainties. All statements included in this presentation, other than statements of historical facts, are forward-looking statements. 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 commercial opportunities for and advantages of MarzAA and DalcA, including their potential to treat hemophilia subcutaneously; plans to enroll the Crimson 1 Phase 3 registration study and report on actions of the DSMB and treatment of 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; the potential markets for and advantages of the Company's complement product candidates, including CB 2782-PEG as a potential best-in-class C3 degrader for dry AMD, CB 4332 as a potential treatment for CFI deficiency, and complement degraders; plans for the Company's collaboration with Biogen; potential markets for the Company's 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 statements. Various important factors could cause actual results or events to differ materially, including, but not limited to, the risk that trials, studies or programs may be delayed or terminated 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, that the Company will need to raise additional capital, which may not be available on favorable terms, if at all, 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 March 4, 2021, on Form 10-Q filed with the SEC on August 5, 2021, 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



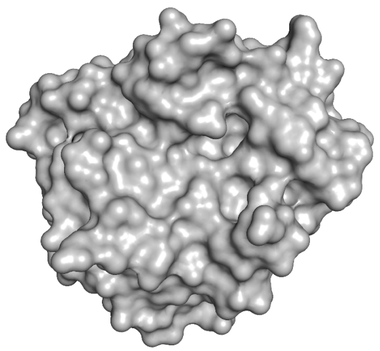
- Novel differentiated medicines
- Robust complement portfolio
- Clinical-stage assets
- Unique expertise in protease engineering

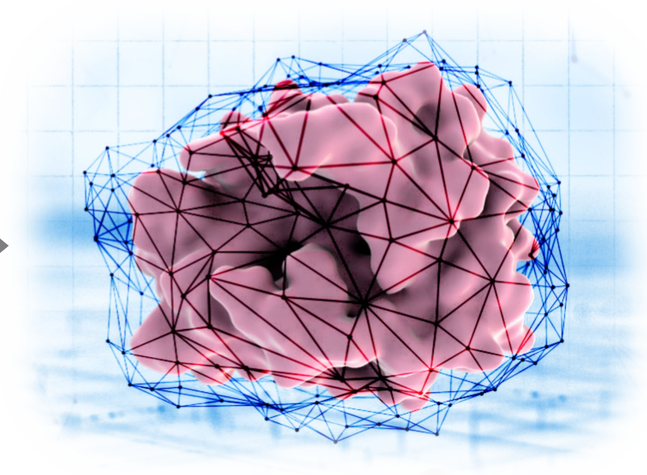
Catalyst protease platform

Unique expertise enables design of optimized & differentiated protease candidates

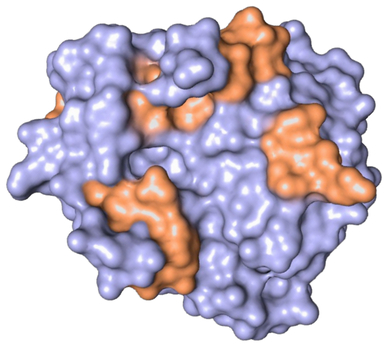
Discovery Platform

Protease Scaffold





Protease Candidate



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

Our Proteases

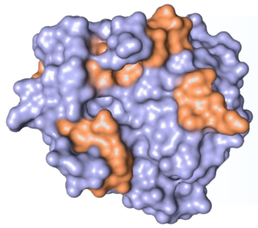
+ Functionally enhanced natural proteases in the complement & coagulation cascades
+ Engineered novel protein degraders in the complement cascade
+ Modulate or target biological activation or inactivation

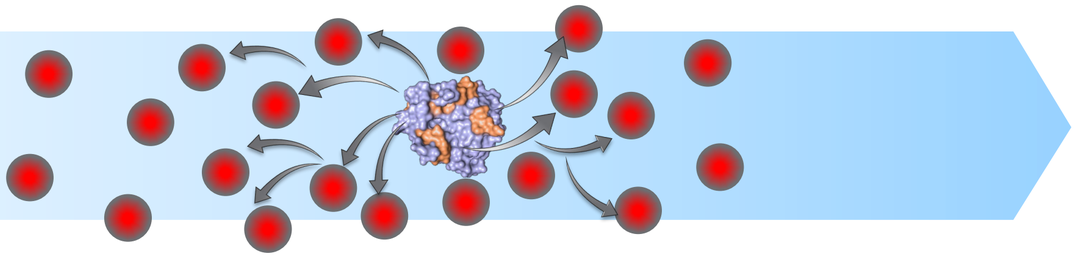
Proteases are ideal for high abundancy targets & cascades

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

Therapeutic target neutralization

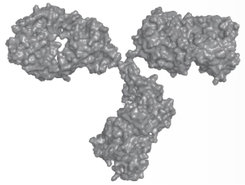
Protease

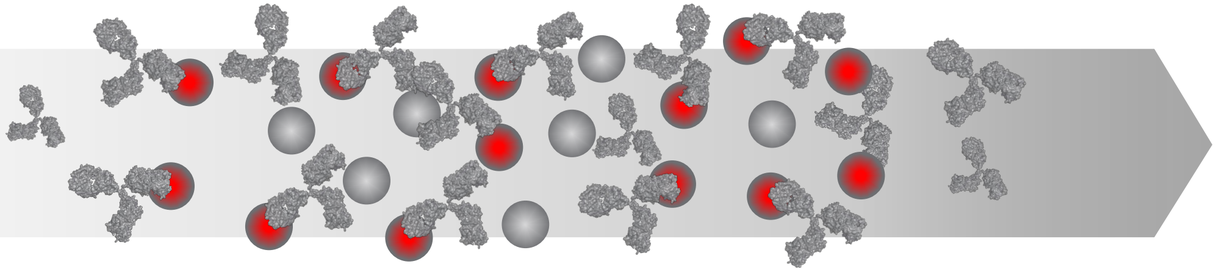




Efficient regulation at low concentrations of therapeutic protease

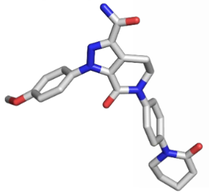
Antibodies

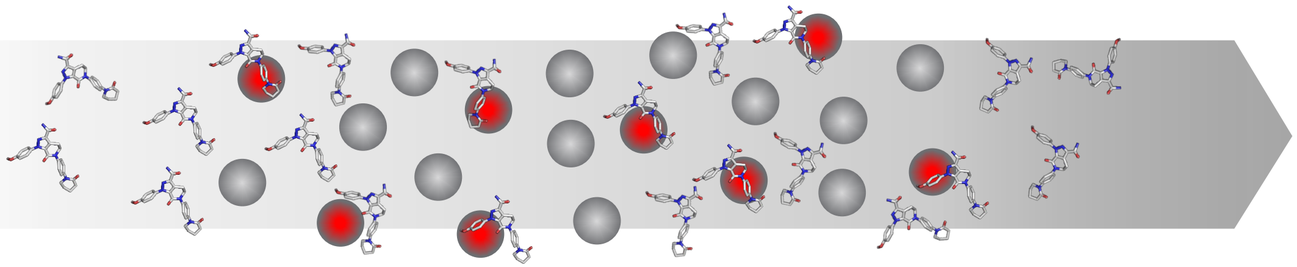




Requires high concentrations in excess of the target

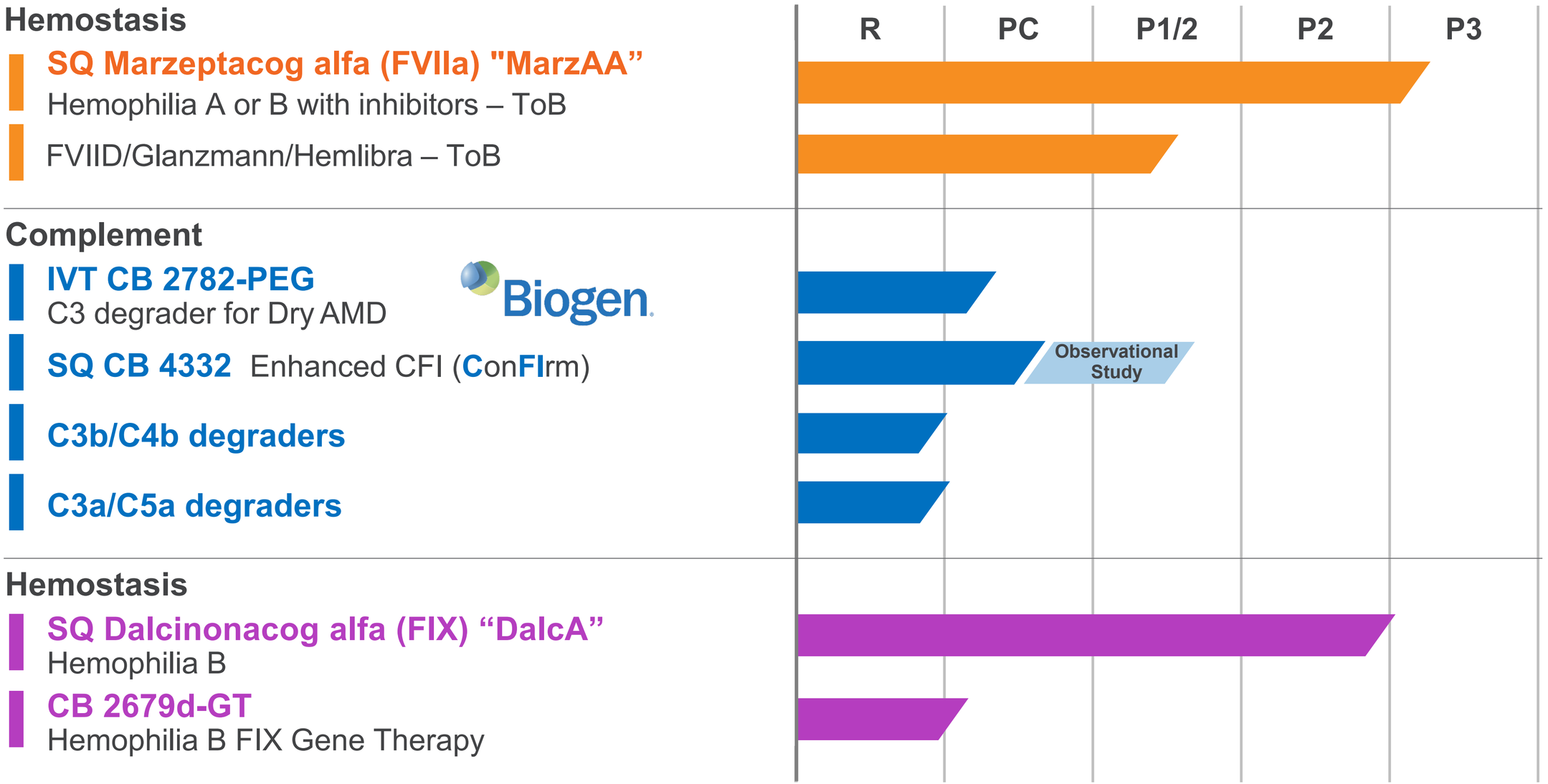
Small molecules / peptides





Requires high concentrations & frequent dosing

Pipeline

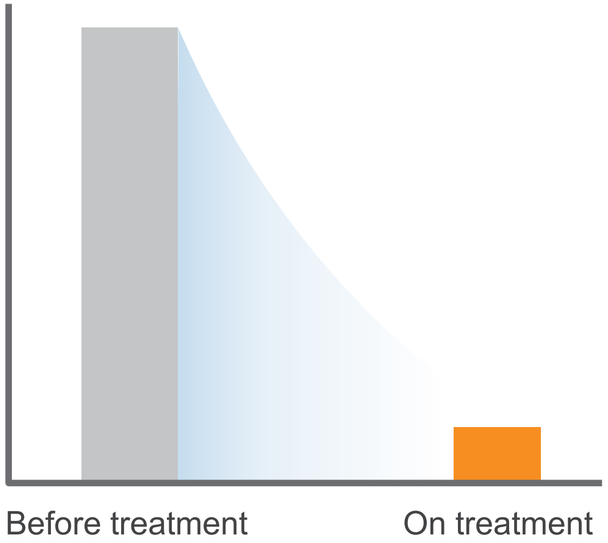


Catalyst protease platform

Validated across three programs

Marzeptacog alfa (activated)

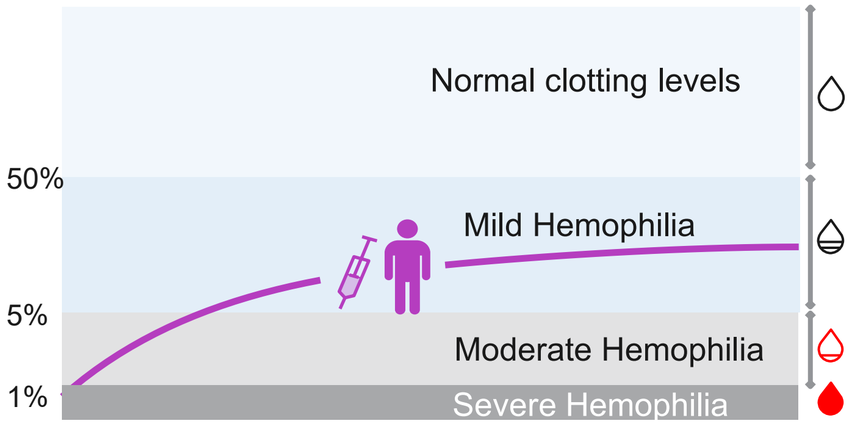
90% reduction in annualized bleed rate



Engineered rFVIIa protease

Dalcinonacog alfa

Achieved sustained & high target levels of FIX

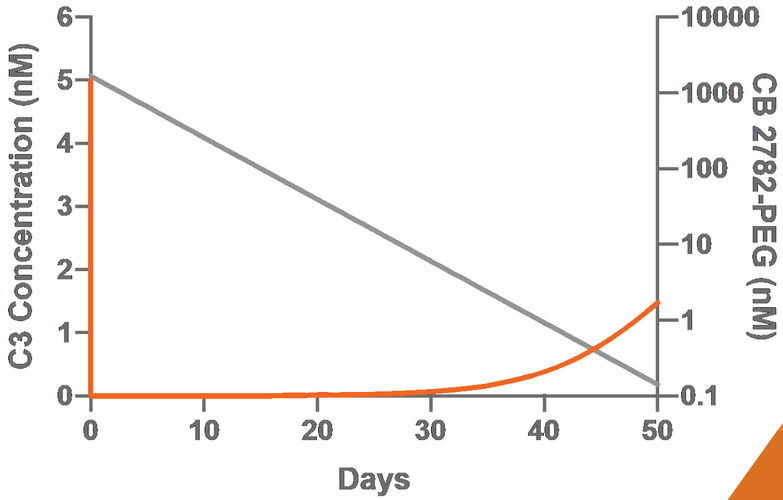


Engineered rFIX protease

CB 2782-PEG

Biogen

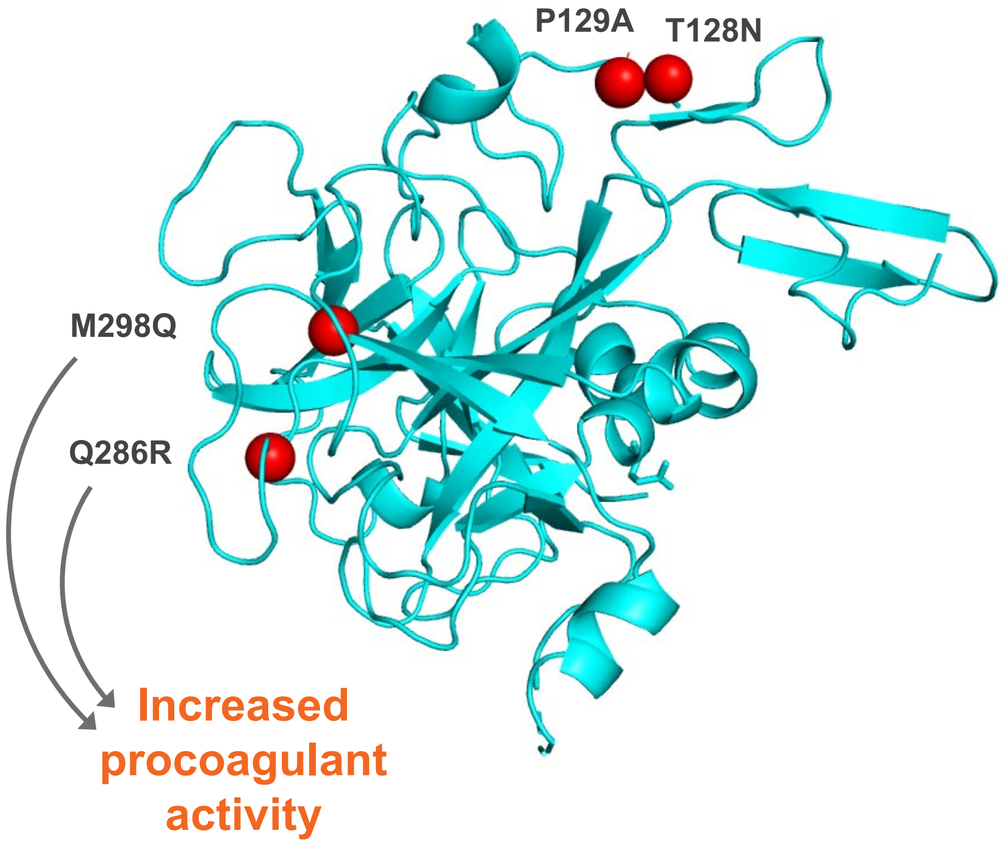
Best-in-class profile for dry AMD
Extended pharmacodynamics



Novel C3 degrader

Marzeptacog alfa (activated) – MarzAA: SQ rFVIIa

Designed to address 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
+ NovoSeven RT is administered IV

Preclinical efficacy of SQ episodic ToB

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

P2 proof of concept & preliminary safety in HA or HB with inhibitors – prophylactic ToB

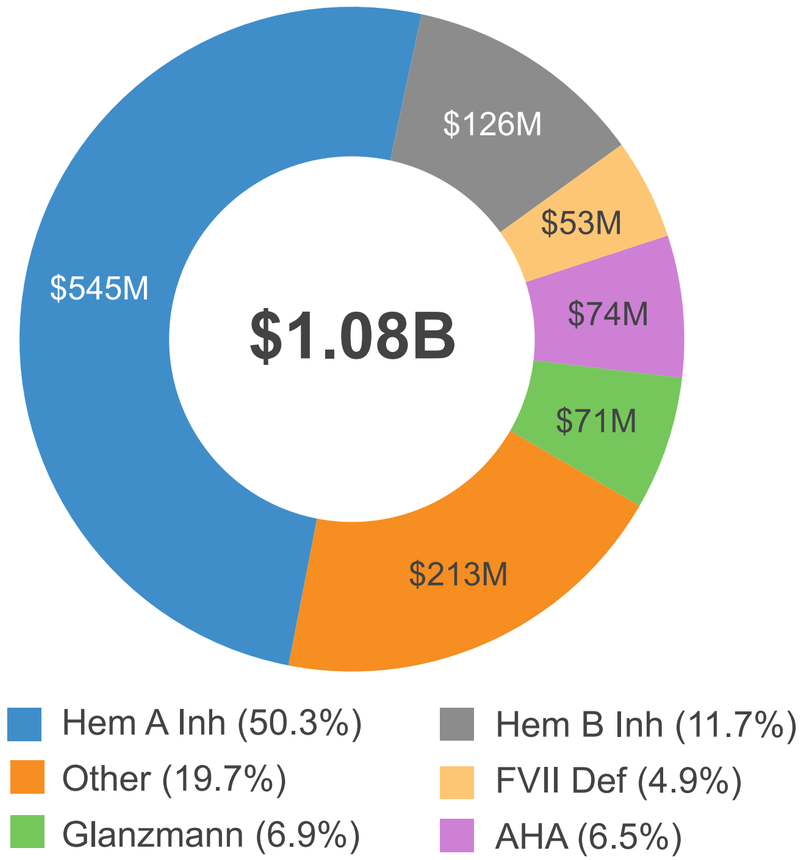
+ 46 patients treated including: single dose IV, up to 3 SQ doses/day, & daily SQ up to 97 days – no ADA

Multiple regulatory designations to date

+ FTD: HA/HB with inhibitors & FVIID
+ ODD: HA/HB with inhibitors & FVIID

SQ MarzAA is a large commercial opportunity

Global NovoSeven sales breakdown by indication (2020)

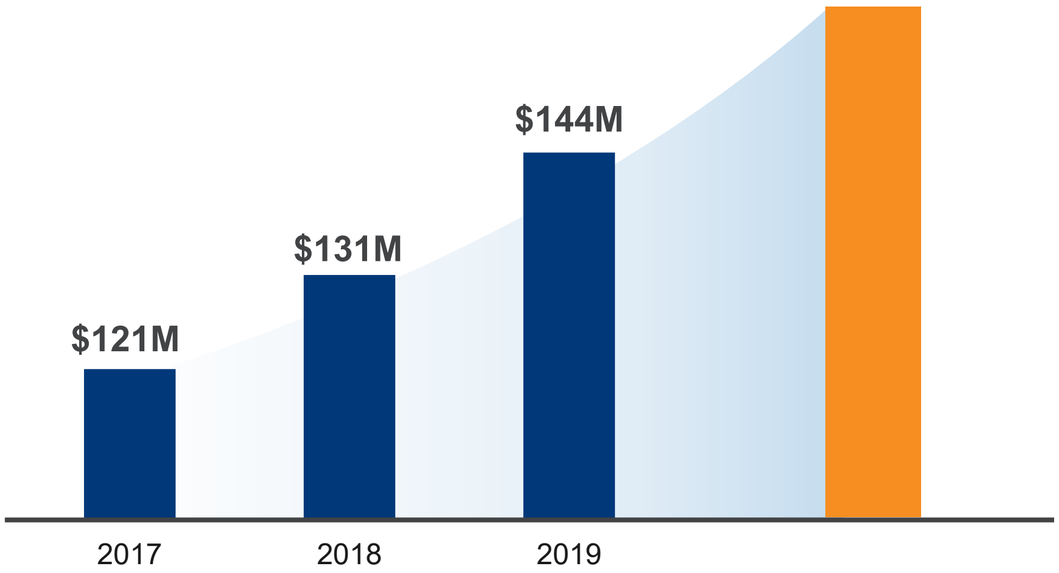


SQ MarzAA profile

- SQ is patient-preferred & eliminates IV barrier to fast & effective treatment
- Ideal for pediatrics & patients with venous access issues
- Long half-life without high Cmax for optimal control of bleeds
- *In vitro* data support combination with Hemlibra® without increased thrombogenicity
- Prophylaxis opportunity demonstrated in P2

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

MarzAA could provide SQ prophylaxis for Glanzmann & FVIID



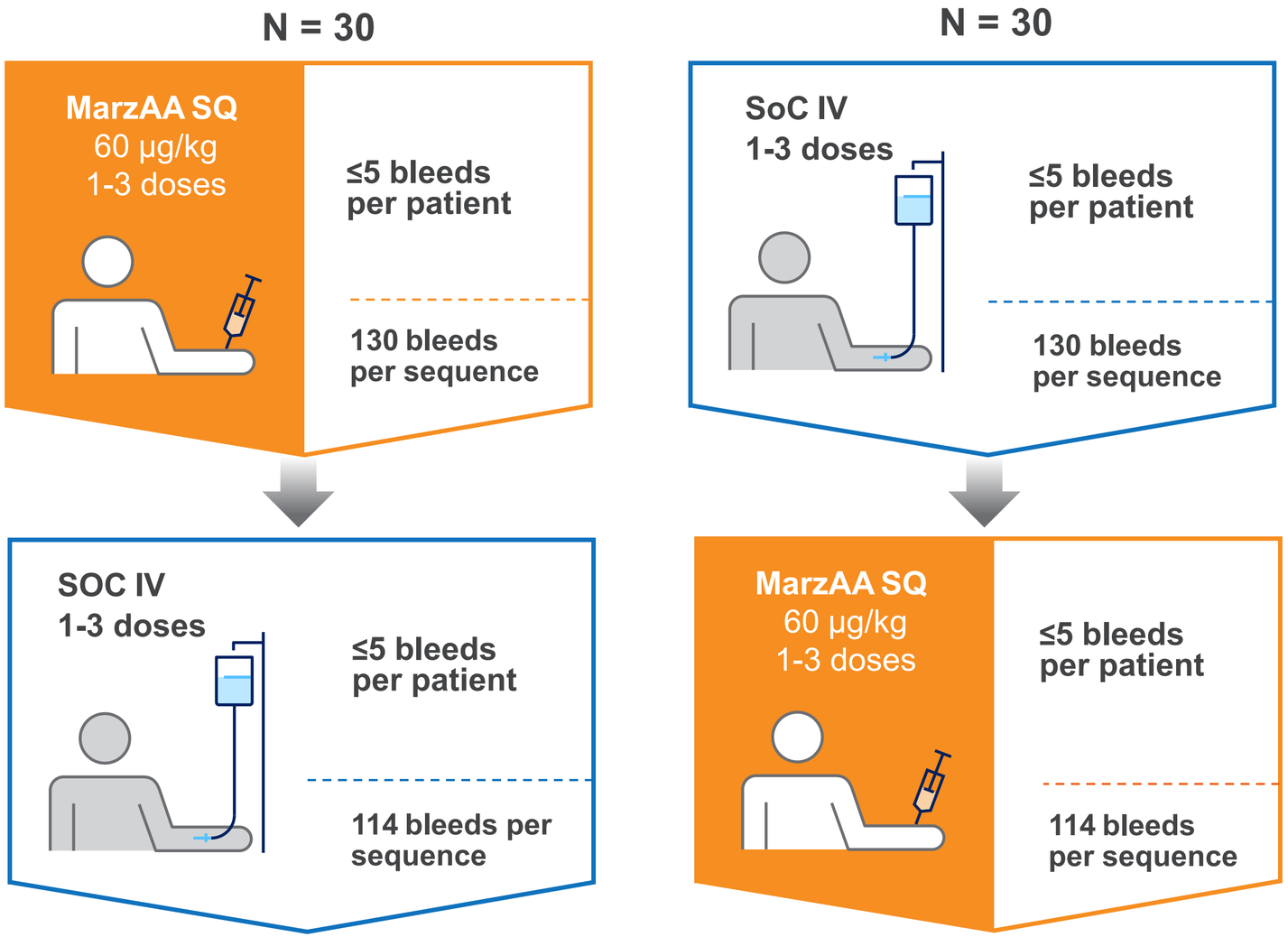
Growing number of Glanzmann Thrombasthenia and FVIID patients treated with NovoSeven

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 times as patients may have multiple bleeding events per year needing factor treatment

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

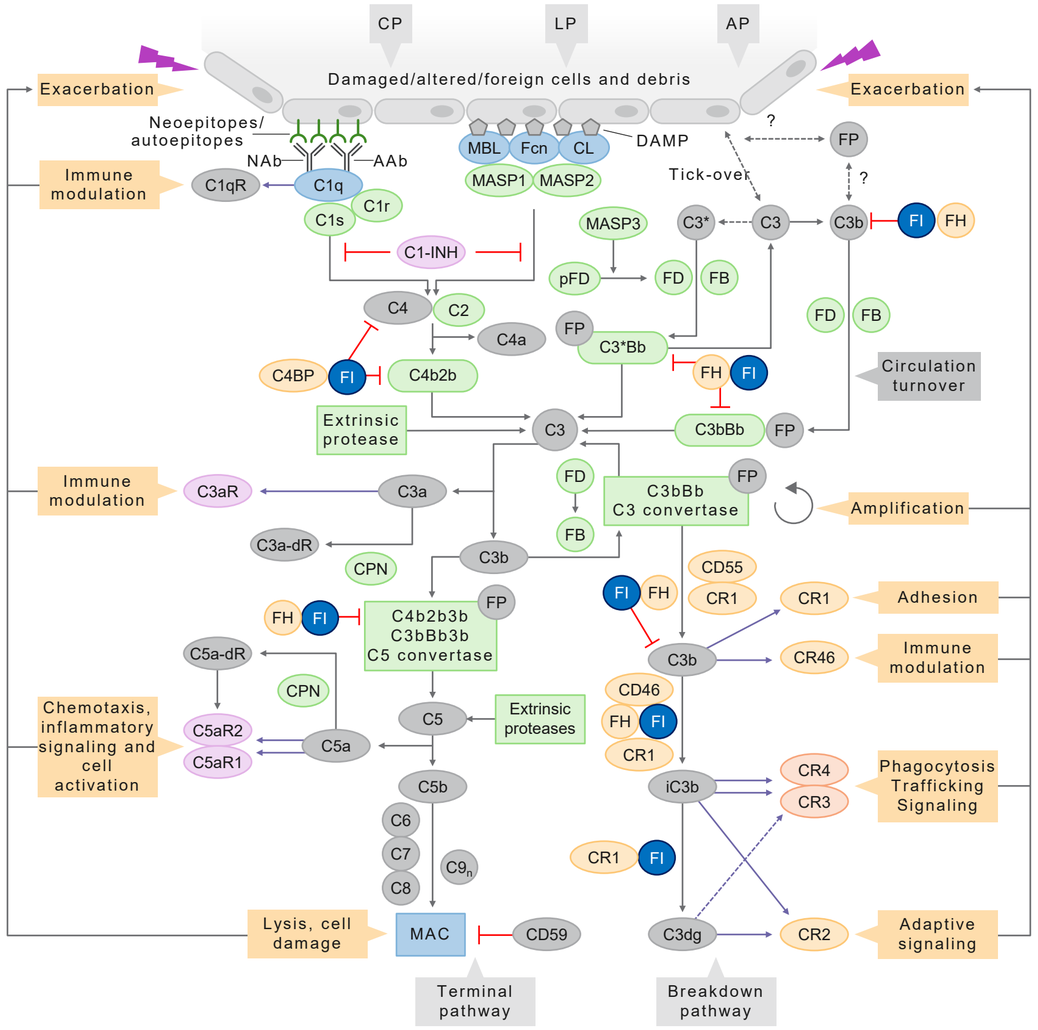
+ **Primary endpoint:** Hemostatic efficacy at 24 hours
+ **Secondary endpoints:** Effective hemostasis at successive timepoints; doses needed; rescue meds
+ **Safety:** Adverse events and ADA

Growing Complement Pathway Protease Platform

CATALYST BIOSCIENCES

Complement is a perfect fit to develop protease therapeutics

The complement pathway is driven by a protease cascade

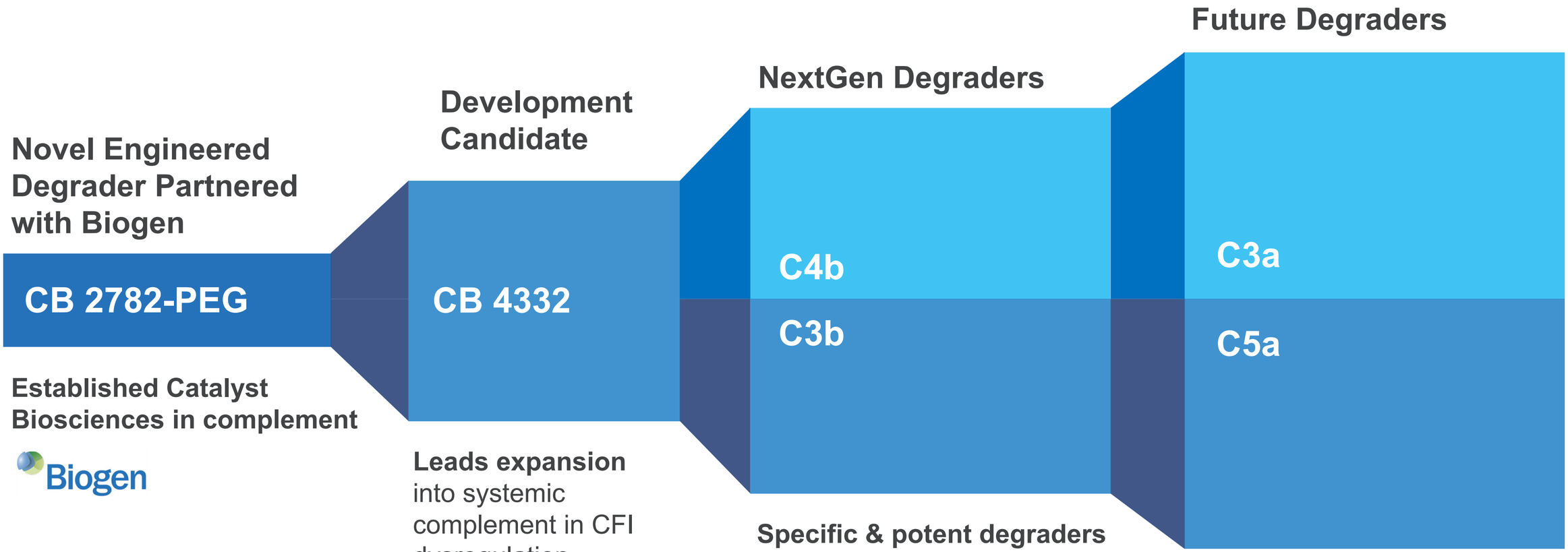


80%

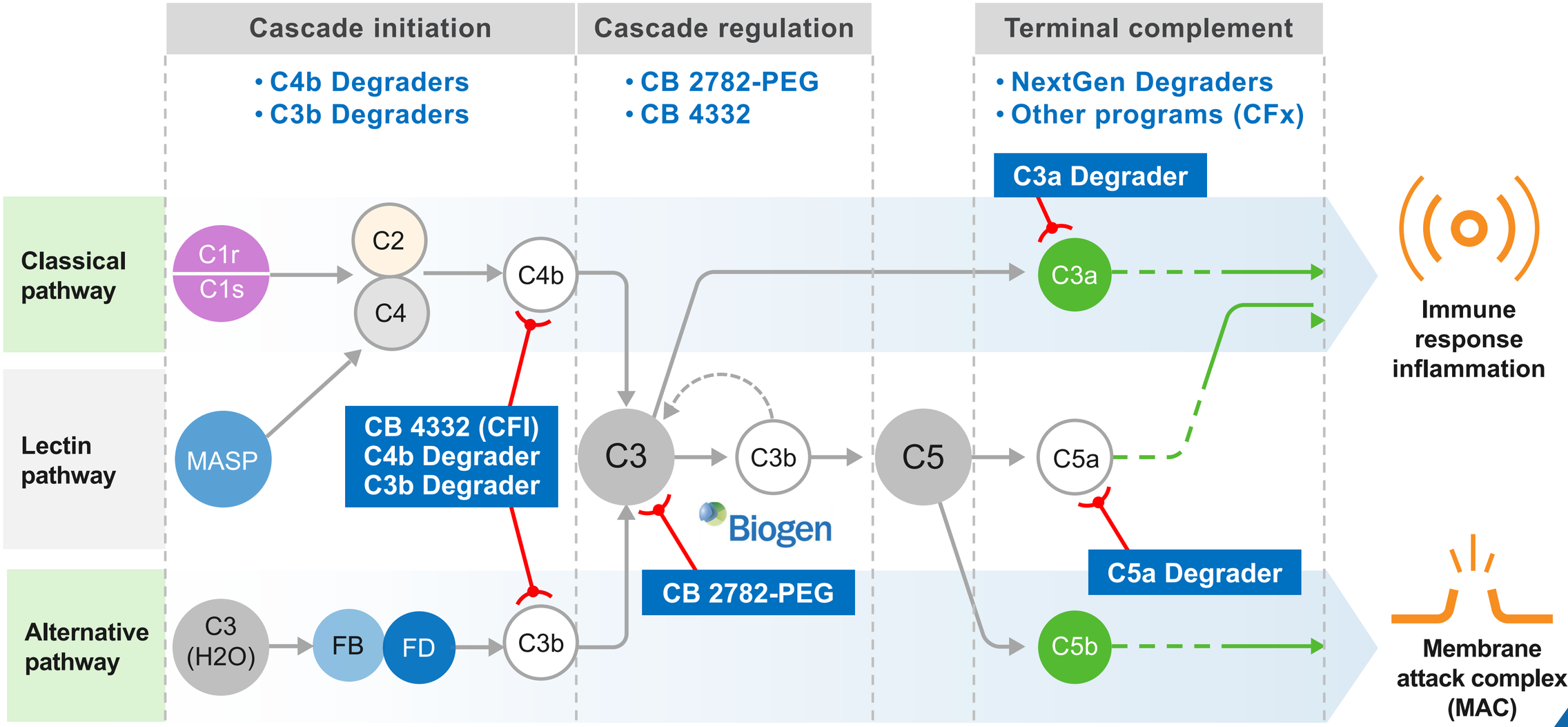
of the complement cascade is regulated by proteases

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

Multiple, high-value complement programs



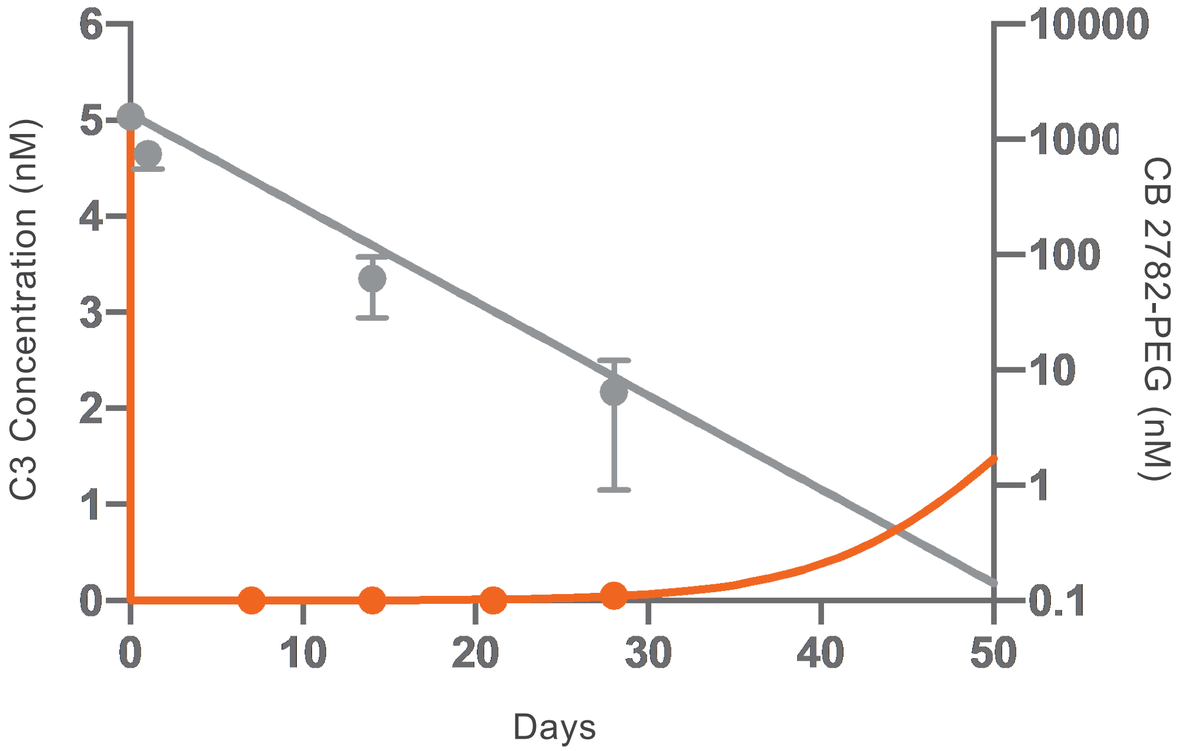
Unique targeted approach to complement regulation



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 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 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**

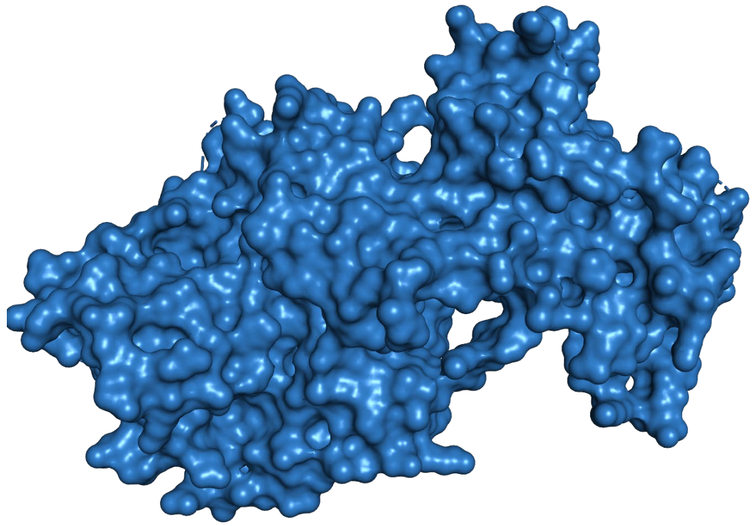
Biogen collaboration

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

*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 & unmet need

+ **Rebalance the 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]

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

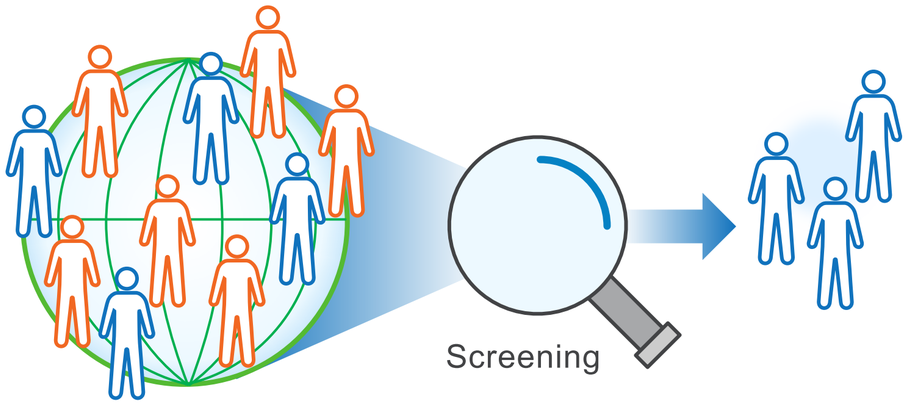
CB 4332: To address CFI deficiency at the root cause

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 **ConFIdence** Study / Discovers Undiagnosed Disease

ConFIrm study

ConFIdence study

Prospective Clinical & Biomarkers Assessment of CFI-Deficiency Disease While on SoC

CB 4332
Phase 1/2
Trials

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

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 ascending SQ doses & 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, FB, FBb, Bb/FB ratio, iC3b, C3d, C3dg, AP50/AH50)
+ Establish a Recommended Dose Regimen within the CFI normal range

Diseases with CFI mutations have tremendous potential

US / EU5 market opportunity

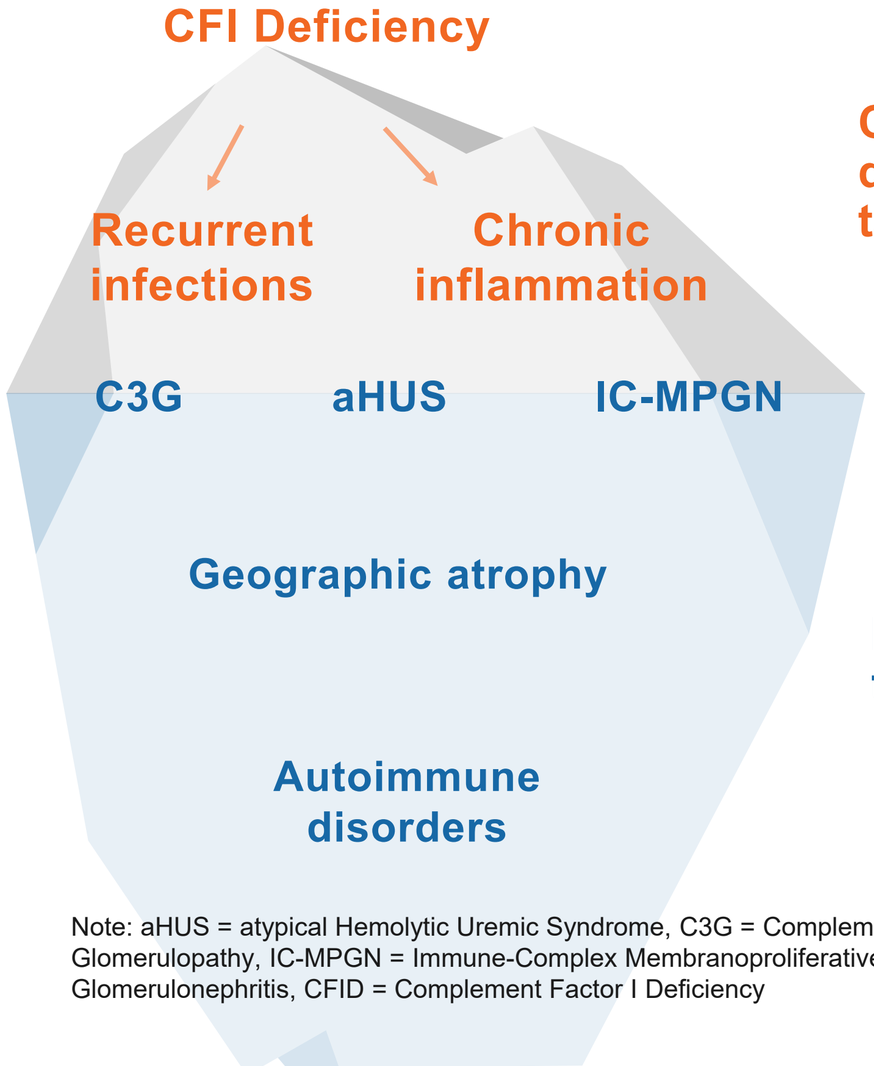
CFI Deficiency
First indication

$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



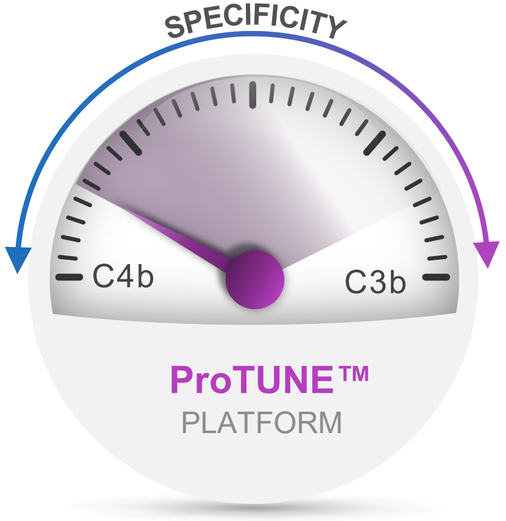
Note: aHUS = atypical Hemolytic Uremic Syndrome, C3G = Complement 3 Glomerulopathy, IC-MPGN = Immune-Complex Membranoproliferative Glomerulonephritis, CFID = Complement Factor I Deficiency

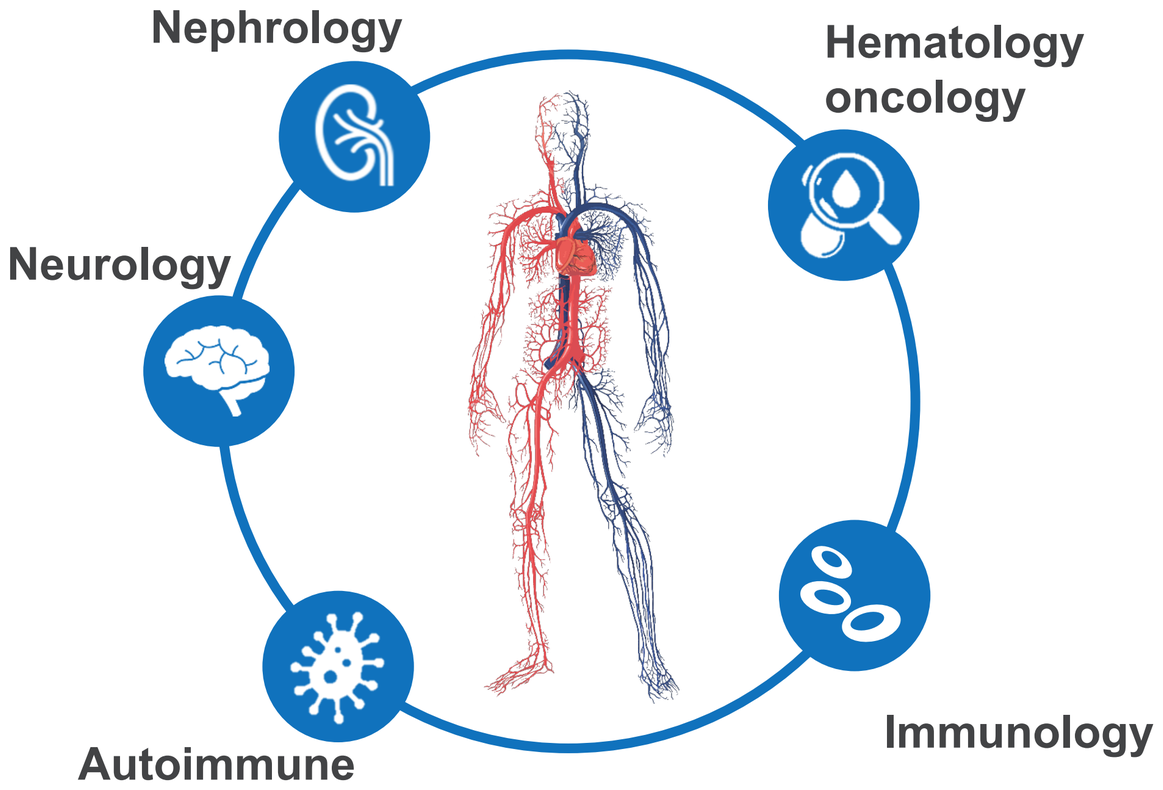
Bresin *et al*. JASN 2013; Fremeaux-Bacchi *et al*. ASN 2013; Rui-Ru *et al*. J Rare Dis Res 2018; Servais *et al*. Kidney Int 2012; Iatropoulous *et al*. Mol Immunol 2016; Hou *et al*. Kidney Int 2014; Alba-Domiguez *et al*. J Rare Dis Res 2012. El Sissy *et al*. Front Immunol 2019; Shields *et al*. Front Immunol 2019; Naesens *et al*. J 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

Our protease platforms are tailored to specific indications

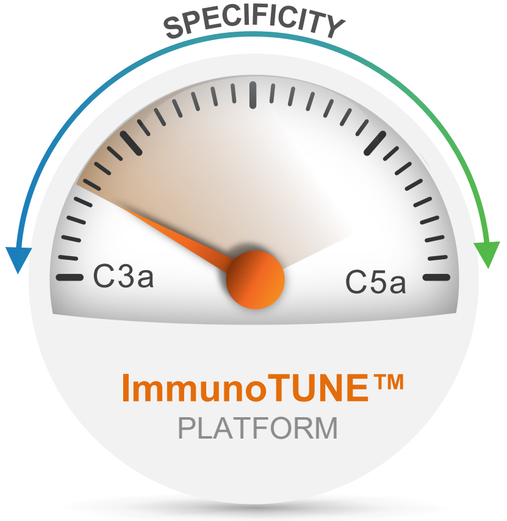
Tuning functionality to restore complement homeostasis & immunoregulation

ProTUNE™
C3b-C4b





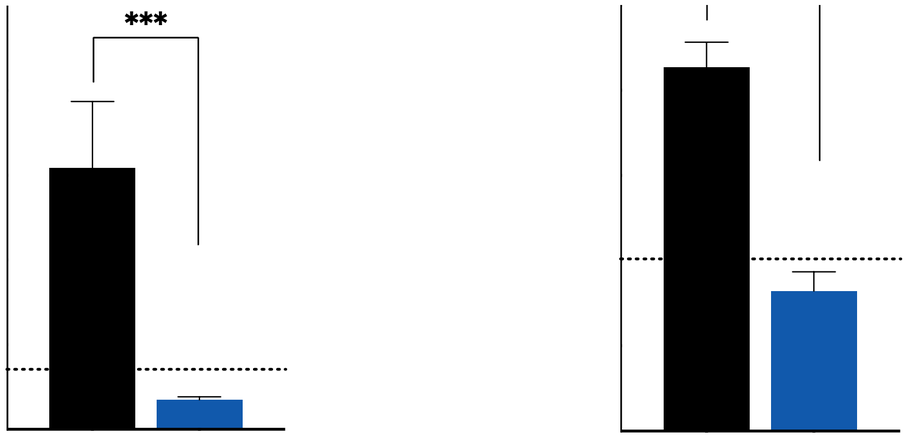
ImmunoTUNE™
C3a-C5a



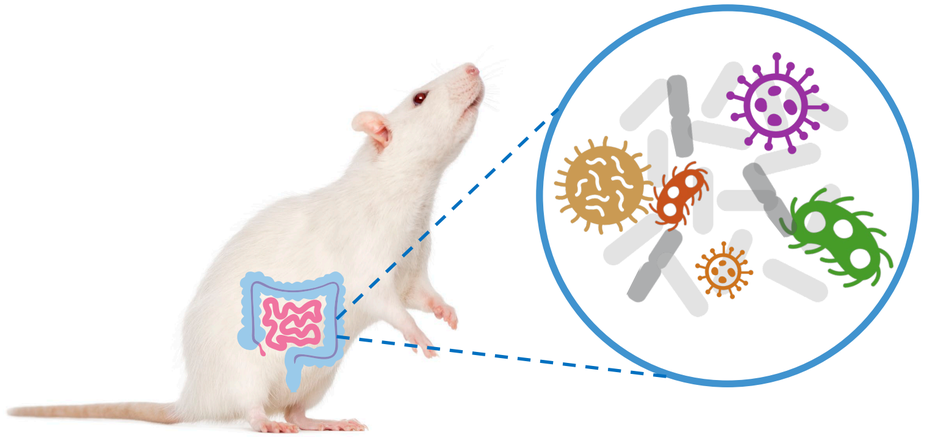
C3b/C4b degraders significantly reduce inflammation *in vivo*

Significantly decrease in inflammatory markers involved in IgA nephropathy

Inflammatory markers in IgA nephropathy



Rat model of complement-mediated inflammation

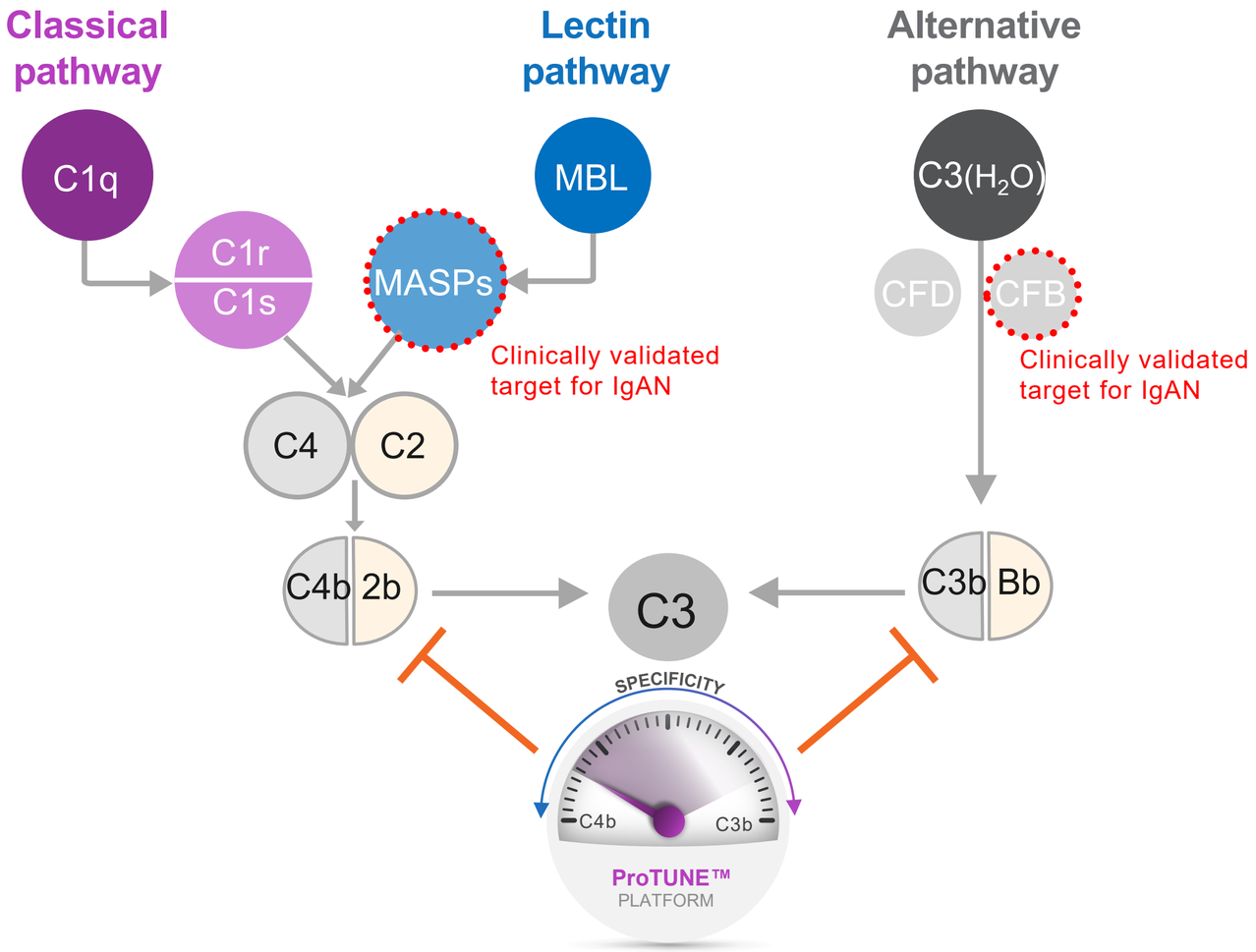


Reduction of **IFNγ & TNFα** involved in kidney damage & proteinuria in IgA nephropathy patients[1, 2]

1. Yano, N. *et al.* Phenotypic Characterization of Cytokine Expression in Patients With IgA Nephropathy. *J Clin Immunol* **17**, 396–402 (1997). 2. Lim, C. S. *et al.* Th1/Th2 predominance & proinflammatory cytokines determine the clinicopathological severity of IgA nephropathy. *Nephrol Dial Transpl* **16**, 269–275 (2001).
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 action: lectin & alternative pathways

Rationale for IgA nephropathy

+ Both lectin & alternative pathways are involved in IgA nephropathy & correlate with severe clinical manifestation[1, 2, 3]

Clinically validated targets

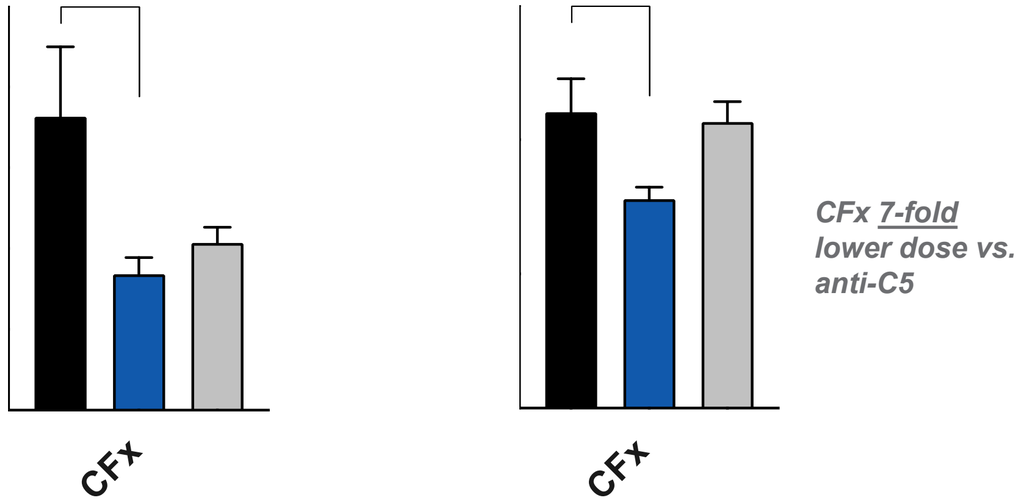
+ Inhibition of only MASP2 or Factor B may be insufficient to reduce proteinuria in IgA nephropathy patients

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: Acute LPS-induced ARDS model efficacy

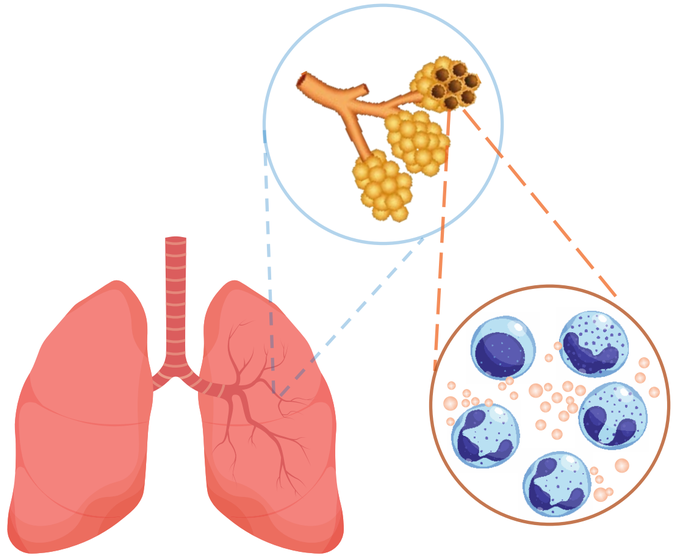
CFx improves respiratory function & reduces cell infiltrates

Respiratory functions & cell infiltration at 24 h



CFx 7-fold lower dose vs. anti-C5

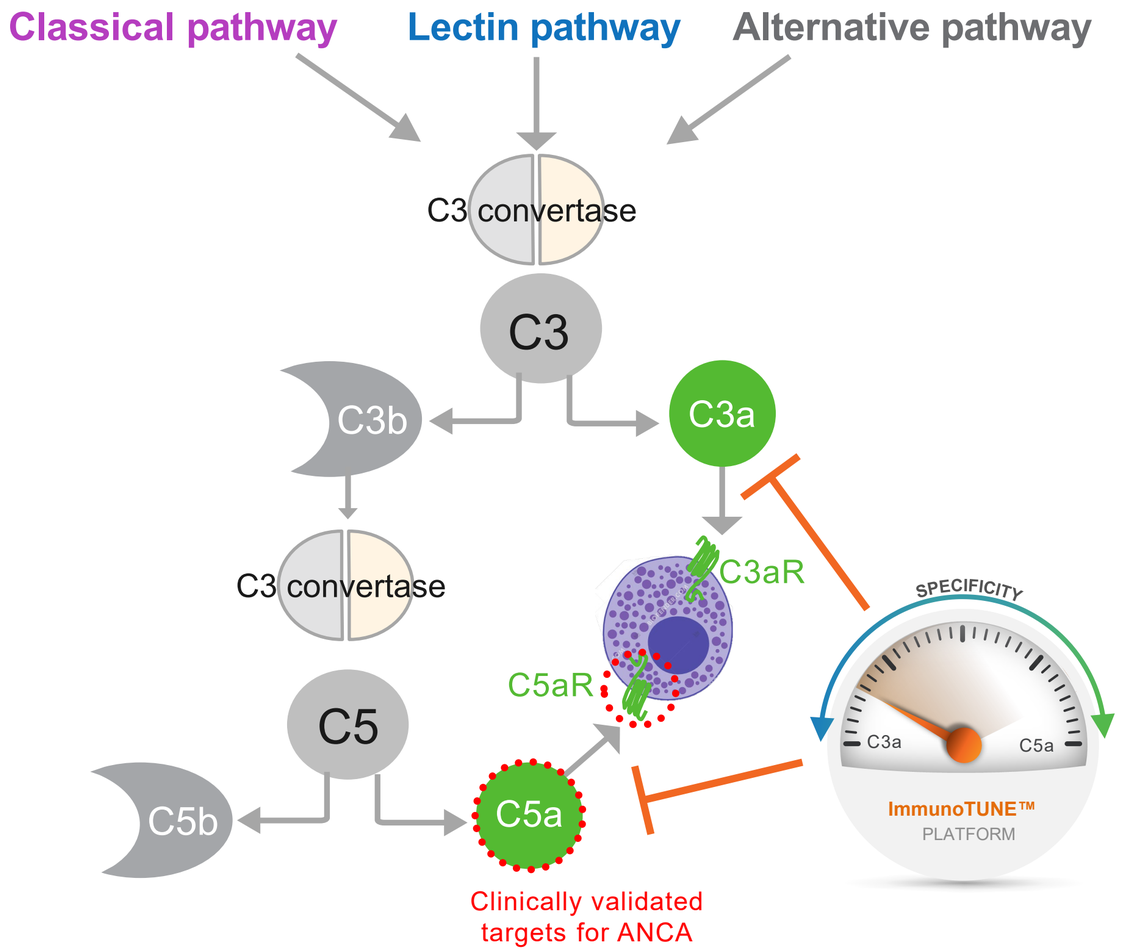
Mouse LPS model of lung inflammation



- 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 treatment

Dual targeting of both C3a & C5a with one protease medicine



Differentiation

+ Degrade activation products of C3 (C3a) & C5 (C5a) that are inflammatory mediators
+ May provide beneficial function *via* **C5L2** pathway

Rationale for ANCA-AAV

+ Both C3a **&** C5a are higher in active AAV patients[1, 2]

Clinically validated targets

+ Inhibition of C5a or C5aR may be insufficient to increase remission rates in ANCA-AAV patients

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

Milestones

2021

- MAA-304 FPI
- MAA-202 FPI
- FTD HA/B ToB & FTD FVIID
- ODD FVIID
- CB 4332 observational trial
- Degrader platform updates
- CB 2782-PEG Biogen

2022

- **Q1:** MAA-304 DSMB
- MAA-202 PK data
- MAA-304 updates
- MAA-202 ToB data
- **Q1:** CB 4332 IND
- CB 4332 P 1/2
- Degrader platform DC
- CB 2782-PEG Biogen

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

THANK YOU

Nasdaq: CBIO

CatalystBiosciences.com