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): July 19, 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 July 19, 2021, 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 (embedded within the Inline XBRL document).

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: July 19, 2021

/s/ Clinton Musil
Clinton Musil
Chief Financial Officer

HARNESSING THE CATALYTIC POWER OF PROTEASES

Complement R&D Day

19 July 2021

CatalystBiosciences.com

Forward-looking statements

Certain information contained in this presentation and statements made orally during this presentation include forward-looking statemen involve substantial risks and uncertainties. All statements included in this presentation, other than statements of historical facts, are forw 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; the potential markets for and advantages of the Company's c product candidates, including CB 2782-PEG, CB 4332 and complement degraders; plans for the Company's collaboration with Biogen; a enroll the CB 4332 observational trial in mid-2021 and to conduct human clinical trials and report pK and biomarker data for CB 4332 in 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 tri and studies may be delayed as a result of COVID-19 and other factors, that trials may not have satisfactory outcomes, the risk that costs 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, competitio 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 May 6, 2021, and in other filings with the SEC. The forward statements in this presentation represent the Company's view as of the date of this presentation and the Company does not assume any to update any forward-looking statements, except as required by law.

CATALYST BIOSCIENCES

Welcome

Catalyst Biosciences:
The Protease Medicines Company

Nassim Usman, Ph.D. | President & CEO

CA
BI

Complement R&D Day – July 2021

Agenda

Time	Topic (Speaker)
12:00 - 12:05 pm	**Catalyst Biosciences: The Protease Medicines Company** **Nassim Usman, Ph.D.** | Catalyst President & CEO
12:05 - 12:25 pm	**The Need for Complement Factor I Replacement** **Filomeen Haerynck, M.D., Ph.D.** | KOL, Ghent University
12:25 - 12:45 pm	**Growing Complement Pathway Protease Platform** **Grant Blouse, Ph.D.** | Catalyst CSO
12:45 - 12:50 pm	**Milestones** **Clinton Musil** | Catalyst CFO
12:50 - 1:10 pm	**Q&A Session**

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The Protease Medicines Company

Harnessing the catalytic power of proteases

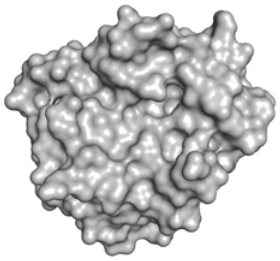
- Novel differentiated medicines
- Robust complement portfolio
- Clinical-stage assets
- Unique expertise in protease engineering

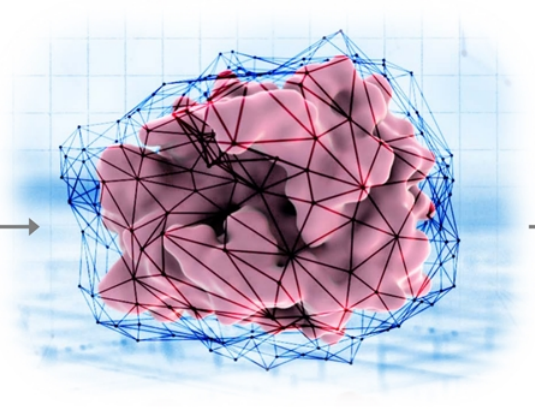
Catalyst protease platform

Unique expertise enables design of optimized & differentiated protease c

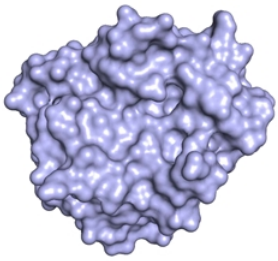
Discovery Platform

Protease Scaffold





Protease Candidate



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

Ou

+ Func natu the coag
+ Engi degr com
+ Mod biolo inac

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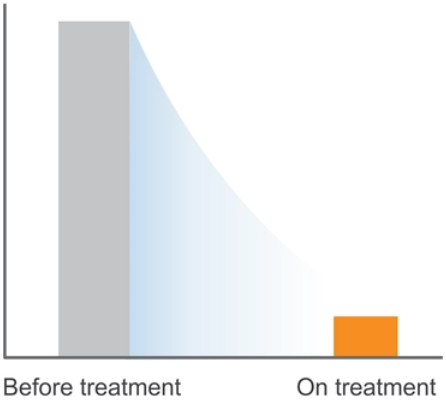
Catalyst protease platform

Validated across three programs

Marzeptacog alfa (activated)

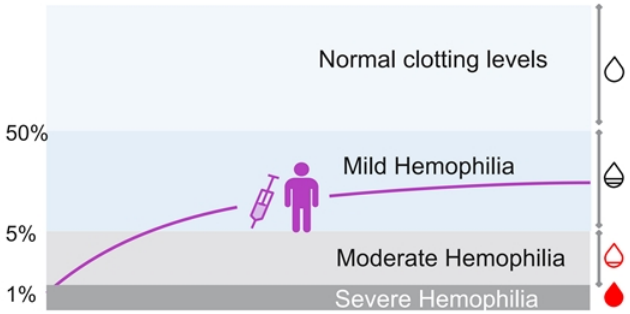
90% reduction
in annualized bleed rate



Engineered
rFVIIa protease

Dalcinonacog alfa

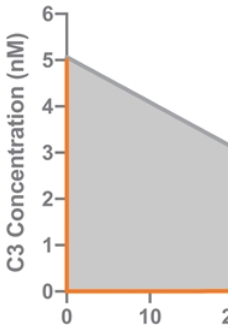
Achieved sustained
& high target levels of FIX



Engineered
rFIX protease

CB 2782-P

Best-in-class
Extended ph



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C3

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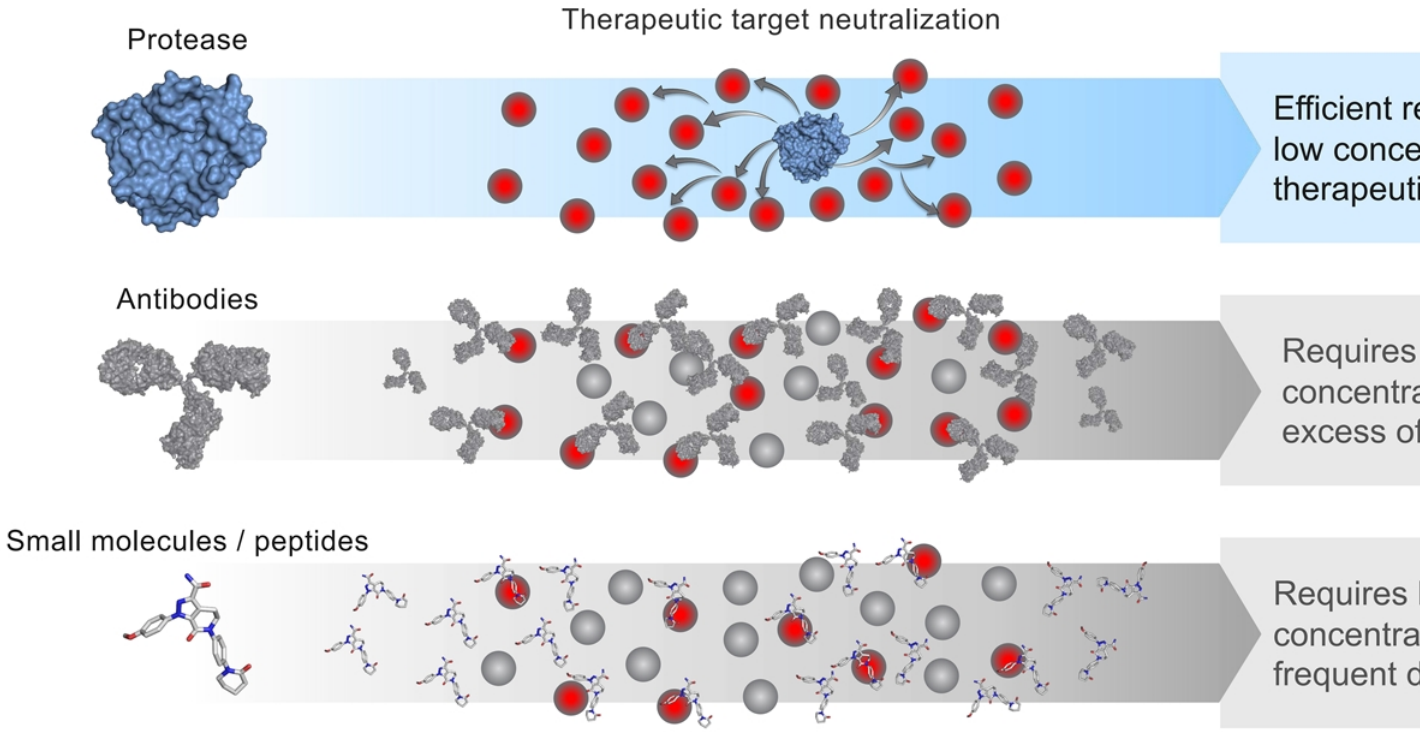
Growing Complement Pathway Protease Platform

Grant E. Blouse, Ph.D. | Chief Scientific Officer

CA
BI

Proteases are ideal for high abundancy targets & cascad

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



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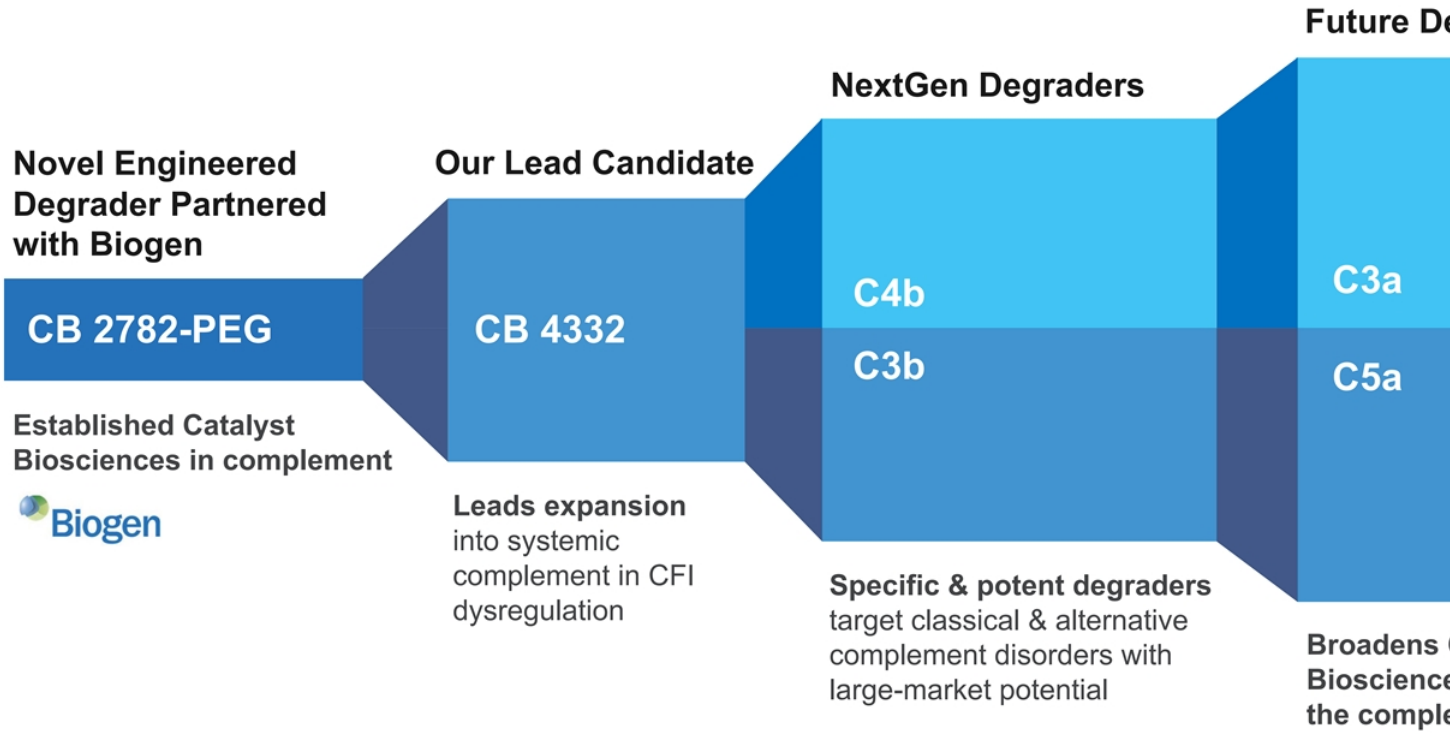
Multiple, high-value complement programs



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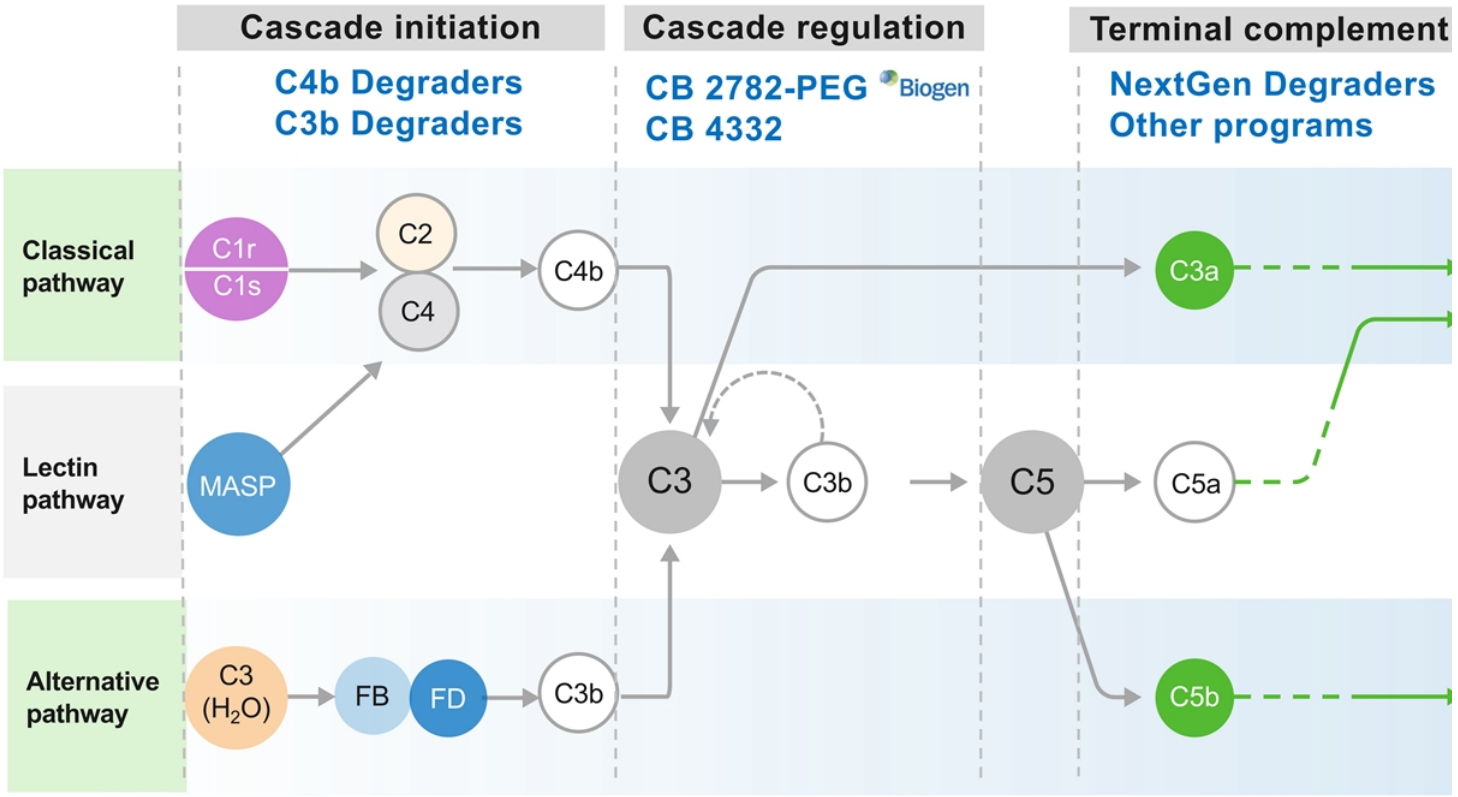
Unique targeted approach to complement regulation



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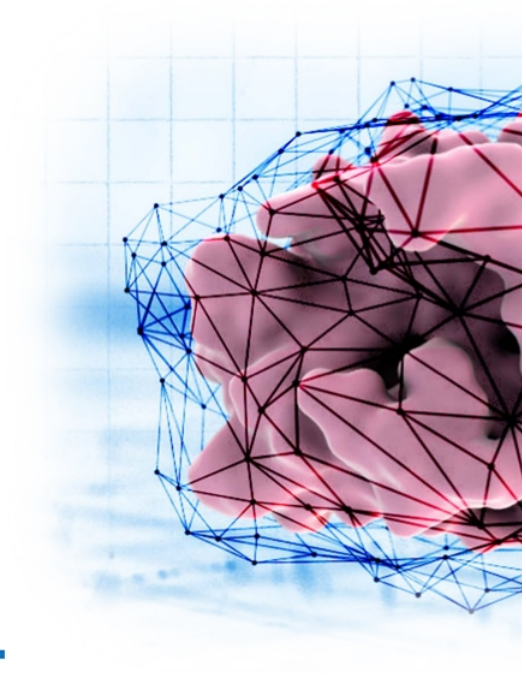

CB 2782-PEG

Novel engineered C3 degrader in complement

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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 co

+ $15M upfr milestones up to low
+ Catalyst: f clinical & activities
+ Biogen: IN activities, developm commerci

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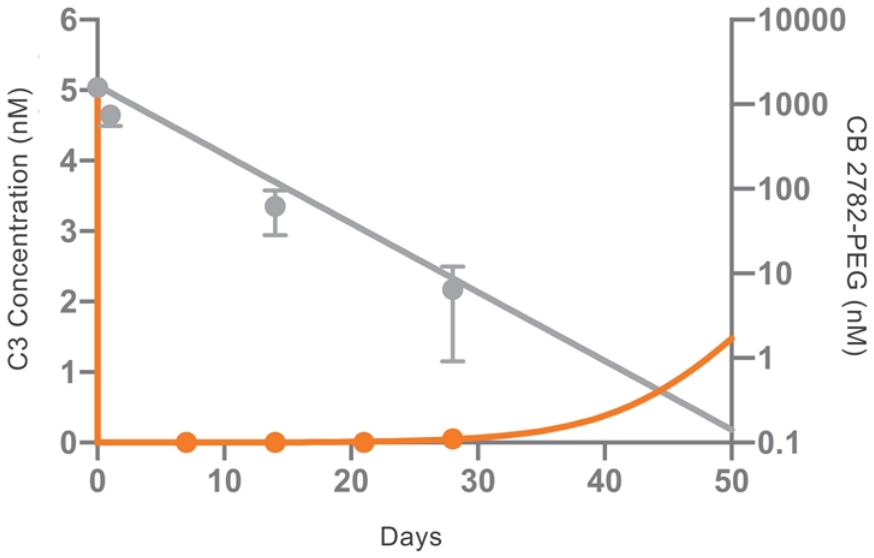
*Furfine *et al.* ARVO 2019

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



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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: Comparison to APL-2 & NGM621

Potential for a less frequent dosing regimen in dry AMD

	APL-2 (Apellis)	NGM621 (NGM Bio)	CB 2782-P
Category	PEGylated cyclic peptide	Antibody anti-C3	**Proteas**
Targets C3	Yes	Yes	Yes
Dose Frequency	Every 1-2 months	Every 1-2 months	**Every ~3 mo**
Half-life in Cyno VH	3.2 days	n/a	**4.1 days**
Dose level (risk of PEG overload)	15 mg (high)	15 mg (none)	**up to 1 mg**

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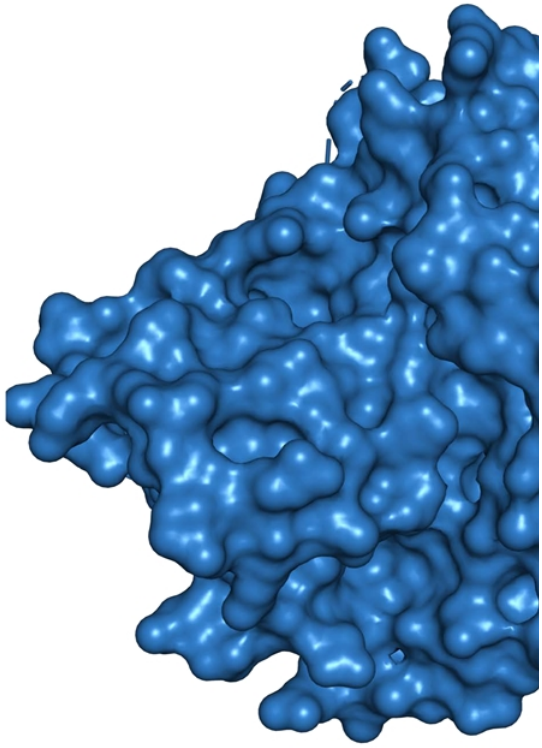
*Frequency estimated based on ocular PK-PD data in non-human

CB 4332: Enhanced Complement Factor I

Next clinical candidate



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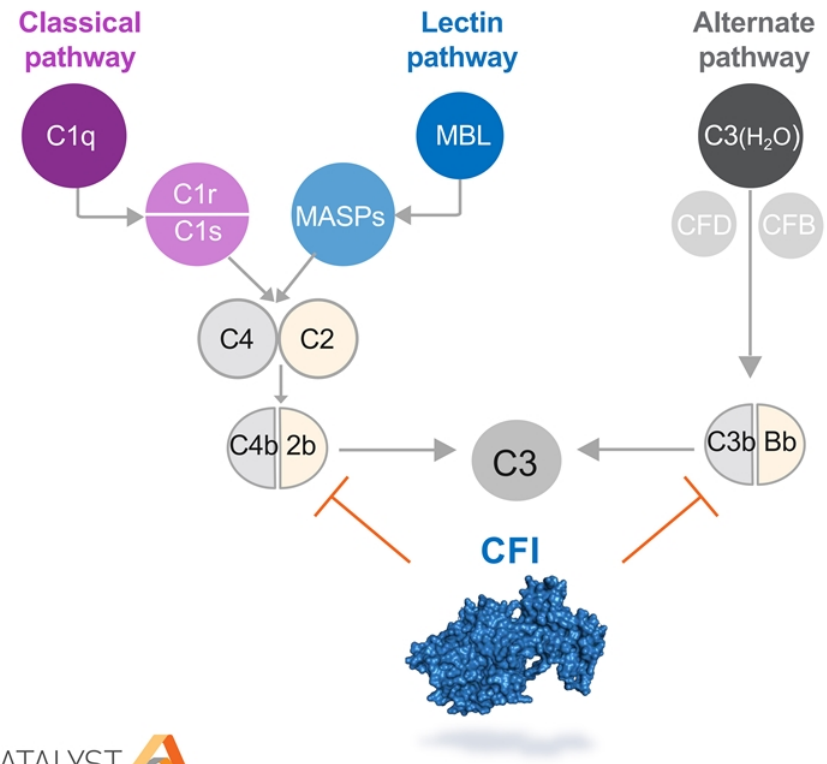
Complement Factor I

CFI is a key down-regulator of the complement cascade



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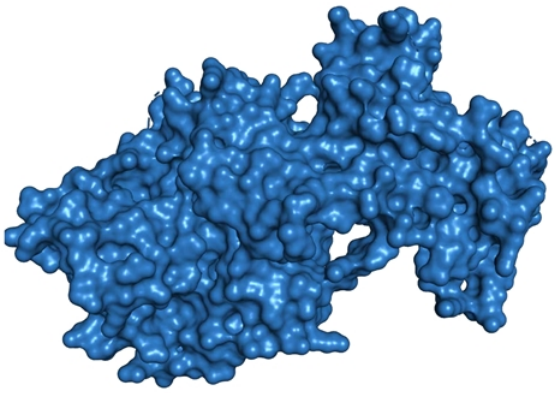
Applying the brakes to

- **CFI is a key regulator** activation targeting **both**
  - Classical & lectin path
  - Alternative pathway ir
- **CFI deficiency** triggers pathway activation
  - Secondary compleme
  - Significant C3 depleti
  - Susceptibility to infec autoimmune complex

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 thera** correct CFI dysreg
+ Targets population **treatment or who poorly to current**

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

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	✓

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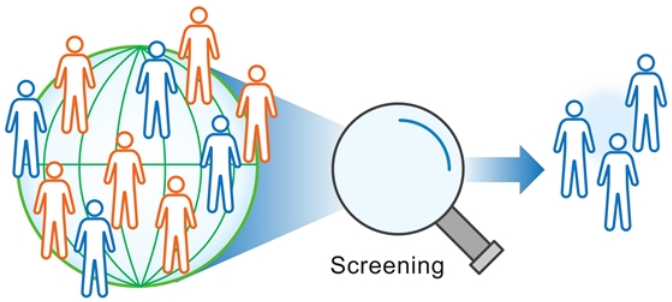
Screening & natural history of disease studies

ConFIrm & ConFIdence: preparing for Phase 1/2



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

The **C**on**FI**rm Study

The **C**on**FI**dence Study

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

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

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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 ascendir & 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, F Bb/FB ratio, iC3b, C3d, C3dg, AP50/AH50)
+ Establish a Recommended Dose Regimen witl the CFI normal range

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Diseases with CFI mutations have tremendous potential

US / EU5 market opportunity

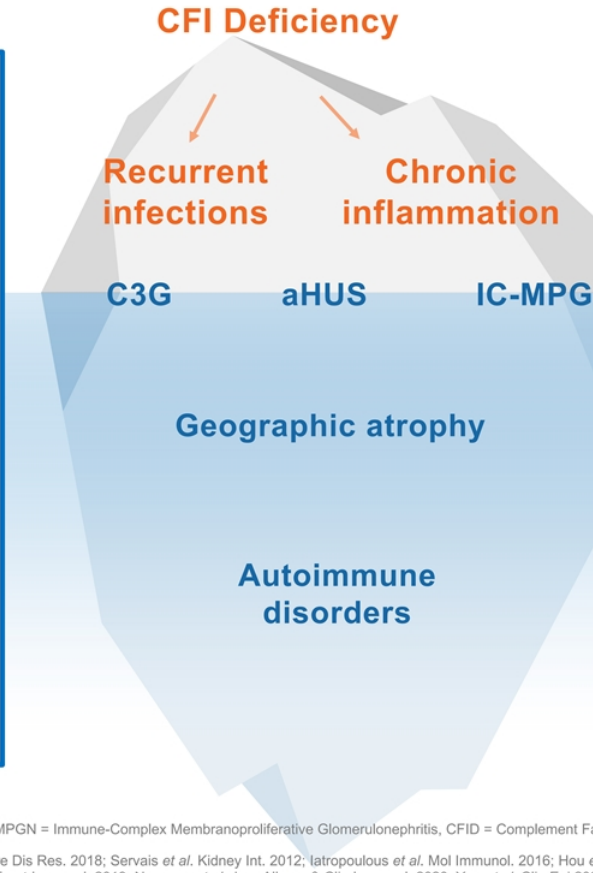
CFI Deficiency
First indication

$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



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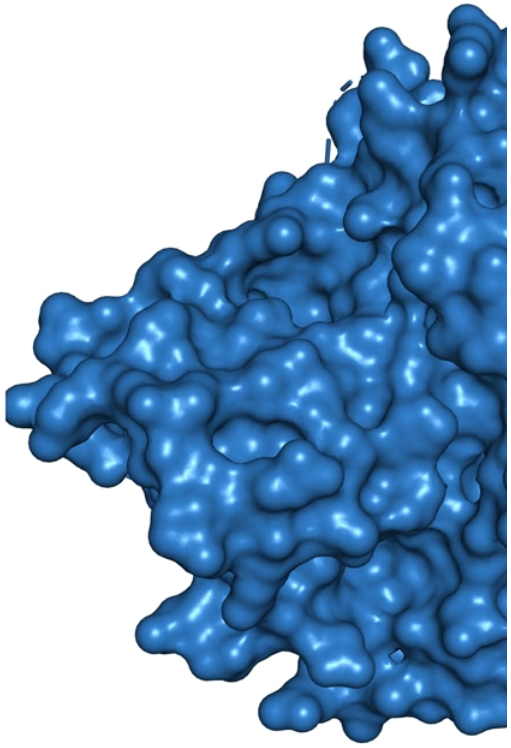
Note: aHUS = atypical Hemolytic Uremic Syndrome, C3G = Complement 3 Glomerulopathy, IC-MPGN = Immune-Complex Membranoproliferative Glomerulonephritis, CFID = Complement Fa

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 e 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 20 Nature Reviews. 2019; Noris *et al*. Clin J Am Soc Nephrol. 2010; CBIO KOL interviews



C3b & C4b Degraders

Expanding into classical complement disorders

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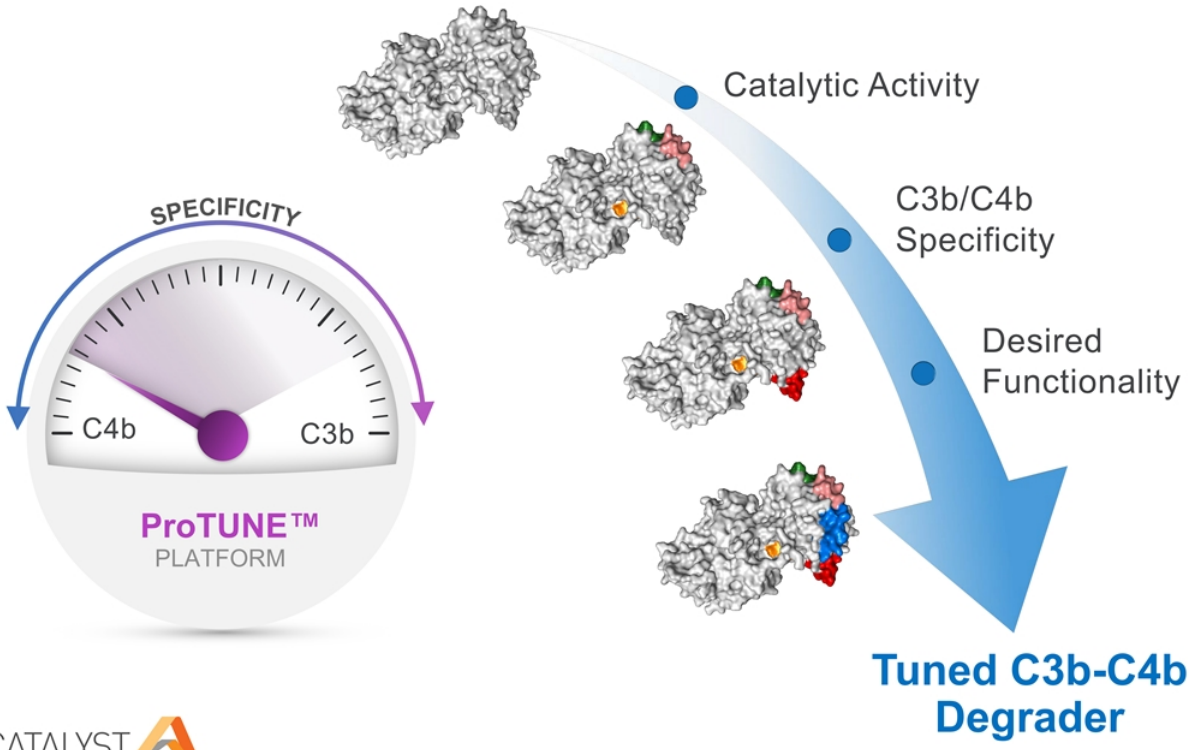
Dialing catalytic power & specificity into CFI

Using ProTUNE™ engineering platform to tune C3b & C4b degraders



Precision C

- Tunable
  control d
  complem
- Tunable
  toward C
  restore t
  to compl

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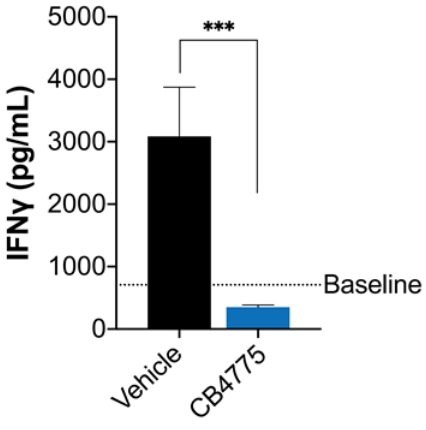
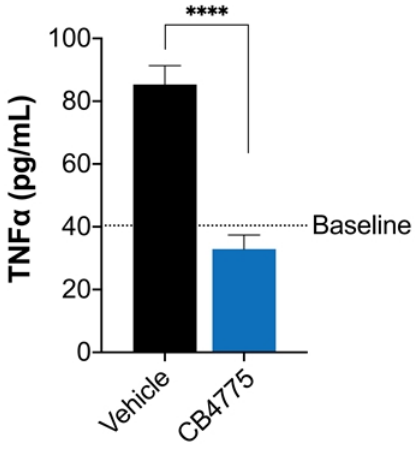
C3b-C4b degraders significantly reduce inflammation *in*

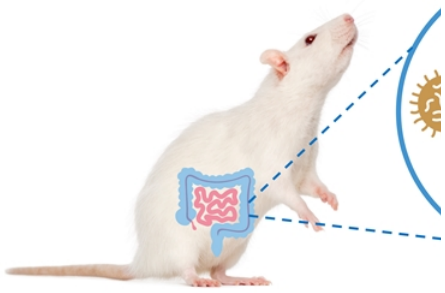
Significantly decrease in inflammatory markers involved in IgA nephr

Inflammatory markers in IgA nephropathy





Rat model of complement-media



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

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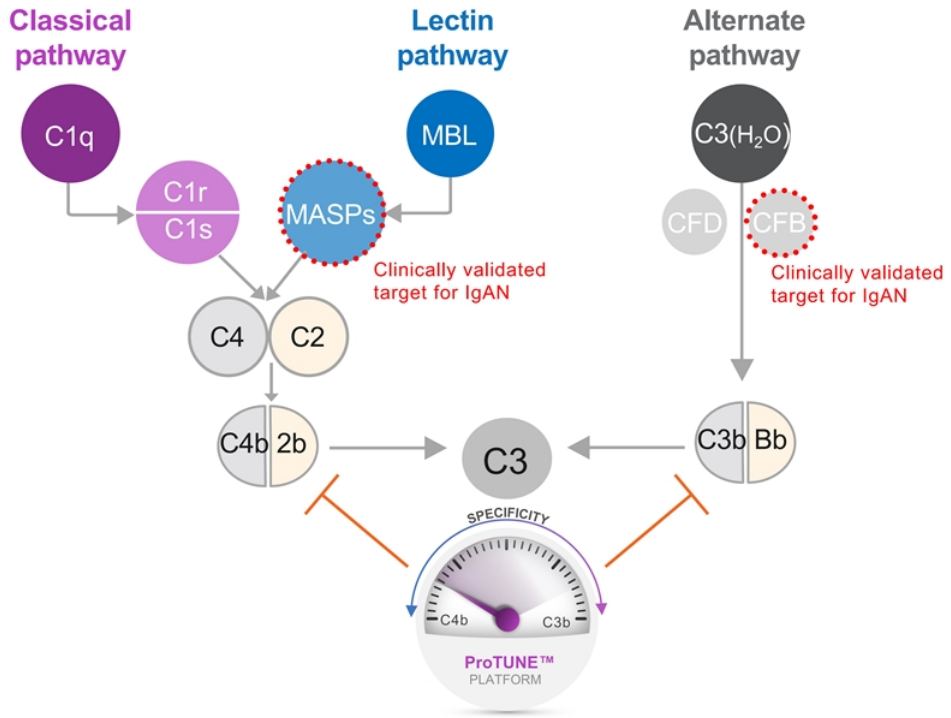
1. Yano, N. *et al.* Phenotypic Characterization of Cytokine Expression in Patients With IgA Nephropathy. *J Clin Immunol* **17**, 396–402 (1997). 2
al. Th1/Th2 predominance and proinflammatory cytokines determine the clinicopathological severity of IgA nephropathy. *Nephrol Dial Transpl* 1
(2001). Values 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 a alternate pathways

Rationale for IgA neph

+ Both lectin & alternate pa involved in IgA nephropa with severe clinical mani

Clinically validated tar

+ Inhibition of only MASP2 be insufficient to reduce nephropathy patients

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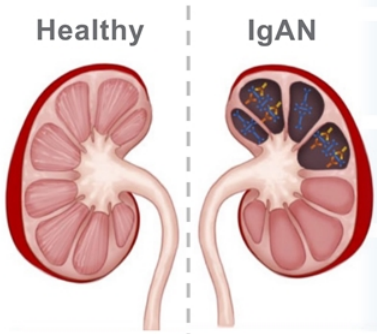
1. Medjeral-Thomas et al. Kidney International Reports (2018); 2. Bi et al. BMC Nephrology (2019); 3. Roos et al. J Am Soc Nephrol (2006)

C3b-C4b degraders for IgA nephropathy patients

Disease in which both lectin & alternative pathways drive pathogenes

High unmet need – current treatments only addressing symptoms



+ Most common form of glomerulopathies worldwid
+ Accumulation & deposition of IgA immune comple deterioration of renal function
+ **10%** patients with rapidly progressive glomerulon
+ **40%** of IgAN patients develop end stage renal dis 20 years & need dialysis/renal transplant in order
+ Significant burden on healthcare resources with a cost of **$49.2 billion** in 2020 in the US

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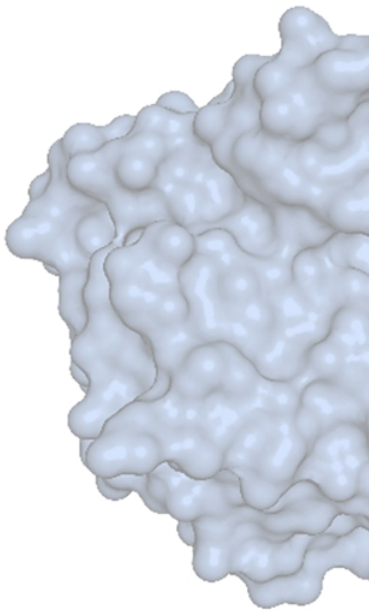
C3a & C5a Degraders

For inflammatory disorders

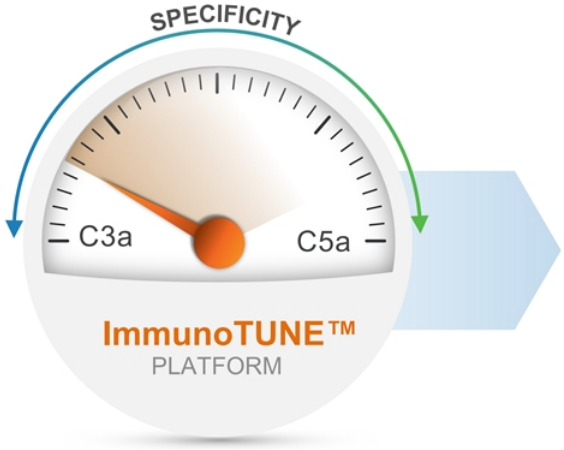


CATALYST BIOSCIENCES

Structural model based on PDB 2XRC

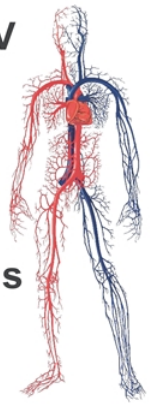
Dialing catalytic power & specificity to restore immunore

Using the ImmunoTUNE™ engineering platform to tune C3a & C5a de



Mast Cell & Neutrophil Disorders

ANCA-AAV
AK/VK
BP
IPF
Asthma
RA
Anaphylaxis
Cancer
CD
IBS



Mastocytosis

Precision CF

- Tunable po different lev of immunon
- Tunable sp toward C3a restore the to complem
- 1 molecule 1000s of ta

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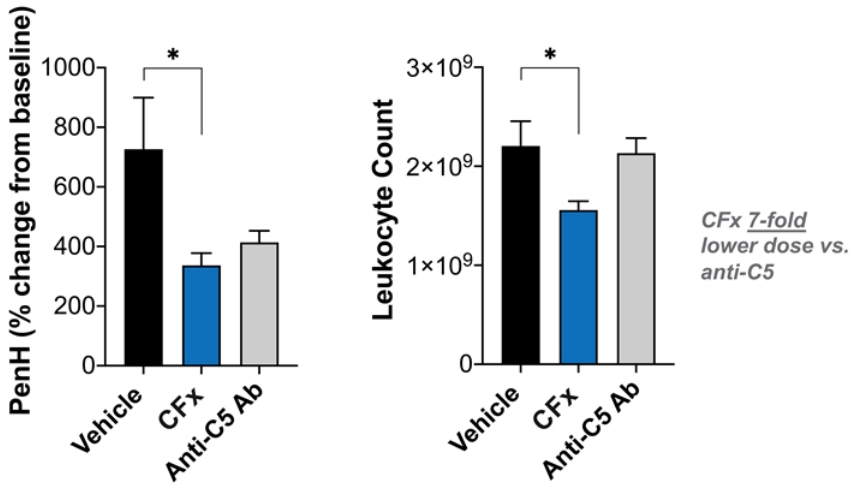
ANCA-AAV, anti-neutrophil cytoplasmic-antibody-associated vasculitis; IBS, inflammatory Bowel Syndrome; CD, Crohn's disease; RA, rheumatoid arthritis; BP, bullous pemphigoid; IPF, idiopathic pulmonary fibrosis; AK, Atopic keratoconjunctivitis; VK, vernal keratoconjunctivitis

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

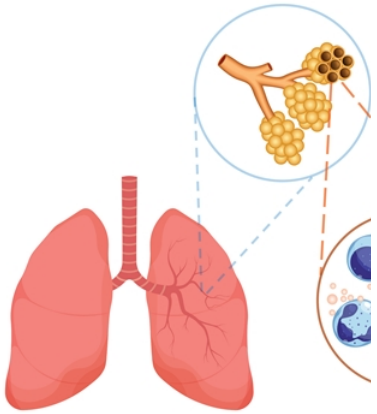
CFx improves respiratory function & reduces cell infiltrates

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 in
- CFx **compares well** on respiratory functions with anti-C5 antibody

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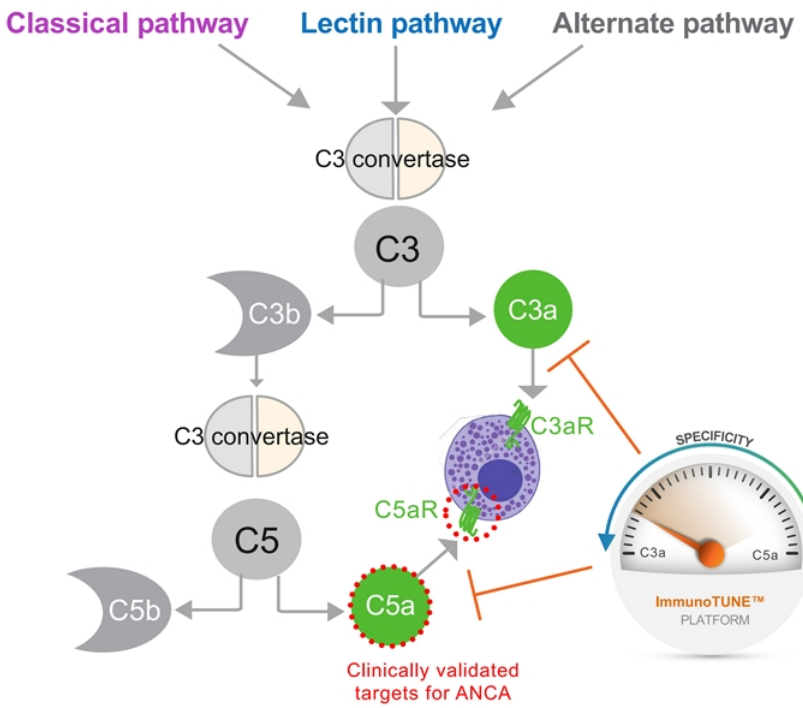
C3a-C5a degraders: Potential for ANCA-AAV patients

Dual targeting of both C3a & C5a with one protease medicine



Differentiation

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

Rationale for ANCA-AAV

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

Clinically validated targe

+ Inhibition of C5a or C5aR m insufficient to increase remi ANCA-AAV patients

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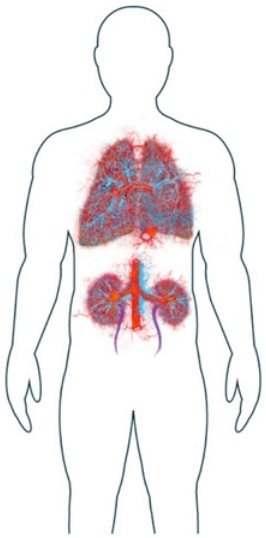
1. S. Moiseev et al. British Society for Immunology, Clinical and Experimental Immunology (2020); 2. Gou et al. Kidney International (2012).

C3a-C5a degraders: Potential for ANCA-AAV patients

Autoimmune disease where anaphylatoxins play a role in the pathoge

High unmet need – current treatments only addressing symptoms



+ Autoimmune disorder characterized by inflammati
  of small blood vessels
+ Clinical signs vary & affect several organs with fre
  involvement of upper respiratory track & kidneys
+ Severe pain due to neuropathy, pulmonary hemor
  of kidneys
+ **10-15%** of patients die in the 1st year of treatment
  conventional therapies (immunosuppressant & glu
+ The only treatments available are to manage the s

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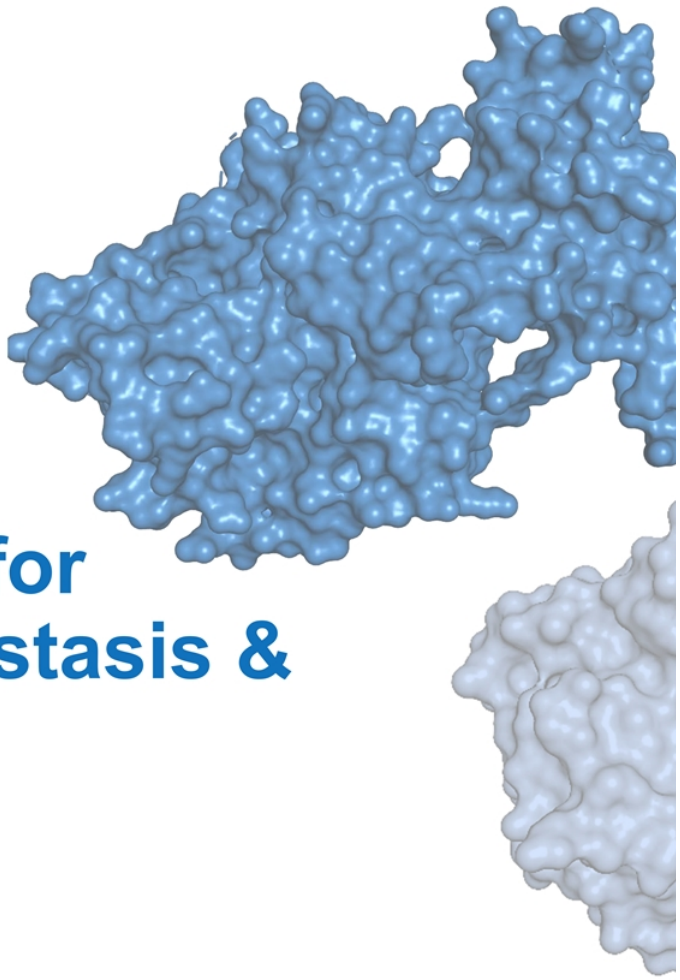
1. S. Moiseev et al. British Society for Immunology, Clinical and Experimental Immunology (2020); 2. Gou et al. Kidney International (2012).



Degraders

Protease platforms for complement homeostasis & immunomodulation

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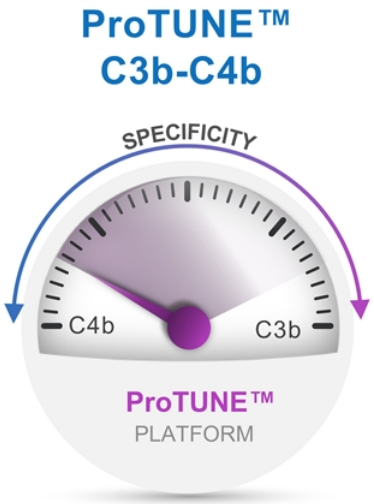
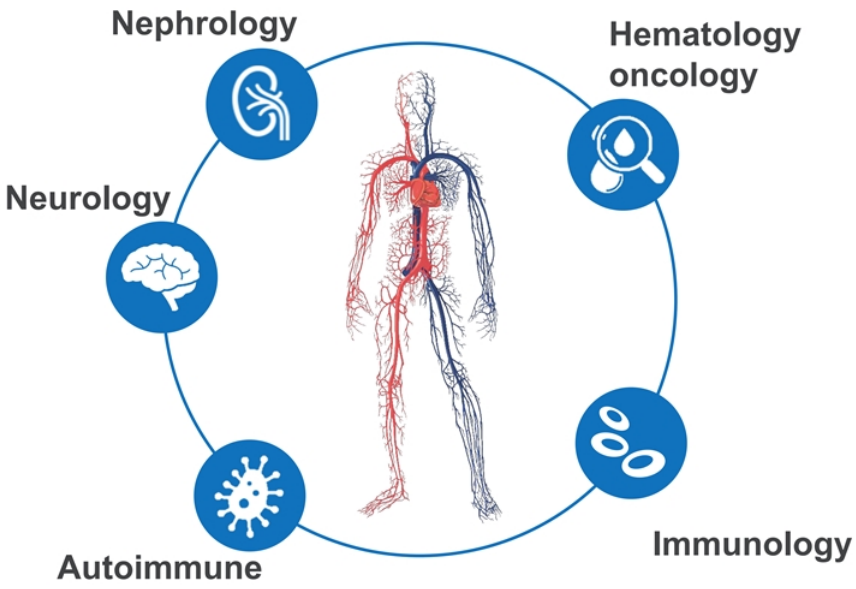
Our protease platforms are tailored to specific indications

Tuning functionality to restore complement homeostasis & immunore





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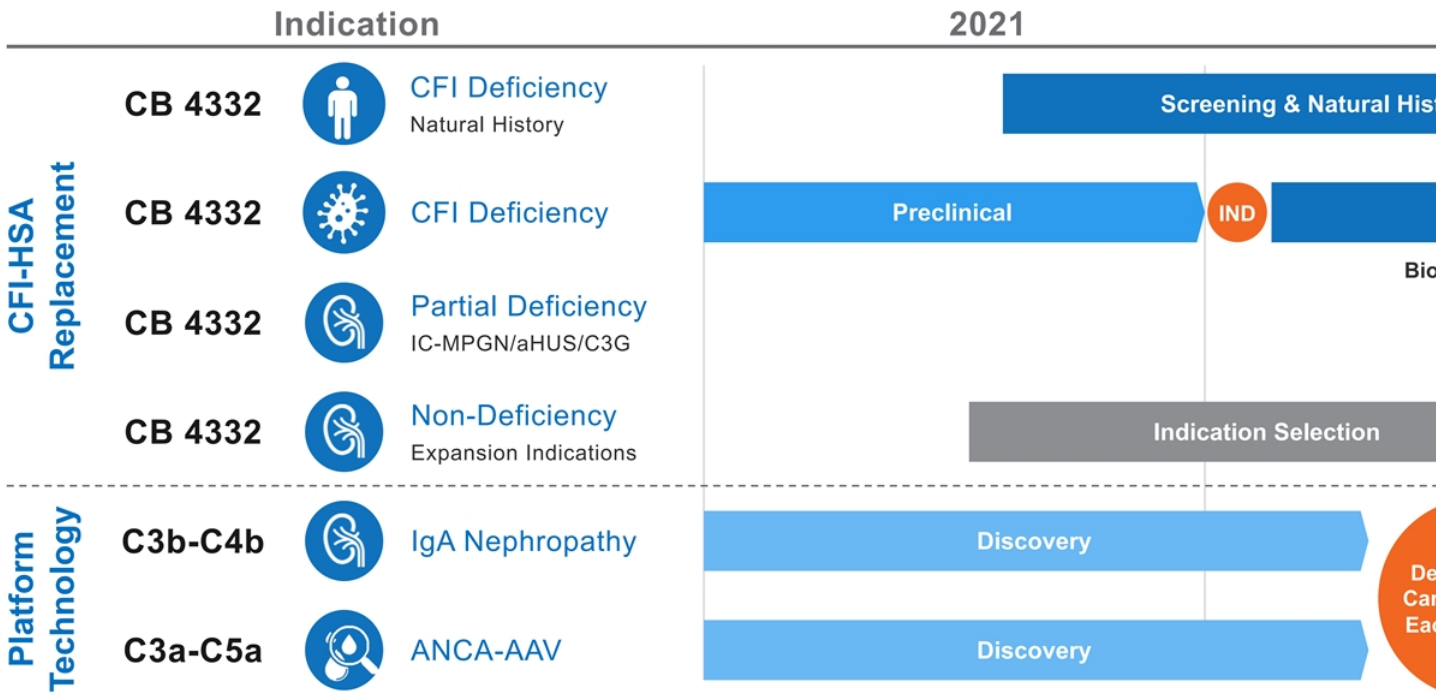
CB 4332 spearheads a deep pipeline in complement

Next development candidate in 2022



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Milestones

Clinton Musil | CFO

Closing Remarks, Q&A

CA
BI

Milestones: Catalyst Biosciences complement programs

Observational trial for CB 4332	Enrollment t start mid-202
Progress CB 2782-PEG in collaboration with Biogen	2021
CB 4332 in the clinic globally	Mid-2022
Development candidates in lead discovery programs	2022
Open-label PK & biomarker data for CB 4332	2022

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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 BIOSCIENCES

Corporate Overview

19 July 2021

CatalystBiosciences.com

Forward looking statements

Certain information contained in this presentation and statements made orally during this presentation include forward-looki involve substantial risks and uncertainties. All statements included in this presentation, other than statements of historical fa looking statements. Forward-looking statements include, without limitation, statements about the product candidates of Cata Inc. (the “Company”) and the benefits of its protease engineering platform, potential markets for and advantages of MarzAA to enroll a pivotal Phase 3 registration study of MarzAA; the dosing of a first patient in a Phase 1/2 trial in patients with FVII Glanzmann Thrombasthenia, and patients treated with Hemlibra; MarzAA as possibly the first prophylactic for FVII Deficien Thrombasthenia; the potential for MarzAA and DalcA to effectively and therapeutically treat hemophilia subcutaneously; the for and advantages of the Company's complement product candidates, including CB 2782-PEG, CB 4332 and complement the Company's collaboration with Biogen; and plans to enroll the CB 4332 observational trial in mid-2021 and to conduct hu and report pK and biomarker data for CB 4332 in 2022.

Actual results or events could differ materially from the plans, intentions, expectations and projections disclosed in the forwa statements. Various important factors could cause actual results or events to differ materially, including, but not limited to, th and studies may be delayed as a result of COVID-19 and other factors, that trials may not have satisfactory outcomes, that replicate the results from earlier trials, the risk that costs required to develop or manufacture the Company’s products will be anticipated, including as a result of delays in development and manufacturing resulting from COVID-19 and other factors, th Biogen will terminate its agreement with the Company, competition and other risks described in the “Risk Factors” section o Company’s Annual Report on Form 10-K filed with the Securities and Exchange Commission ("SEC") on March 4, 2021, on with the SEC on May 6, 2021, and in other filings with the SEC. The forward-looking statements in this presentation represe view as of the date of this presentation and the Company does not assume any obligation to update any forward-looking sta required by law.

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The Protease Medicines Company

Harnessing the catalytic power of proteases

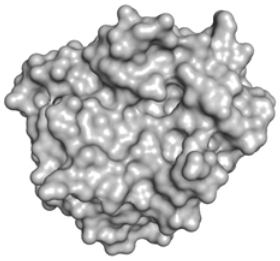
- Novel differentiated medicines
- Robust complement portfolio
- Clinical-stage assets
- Unique expertise in protease engineering

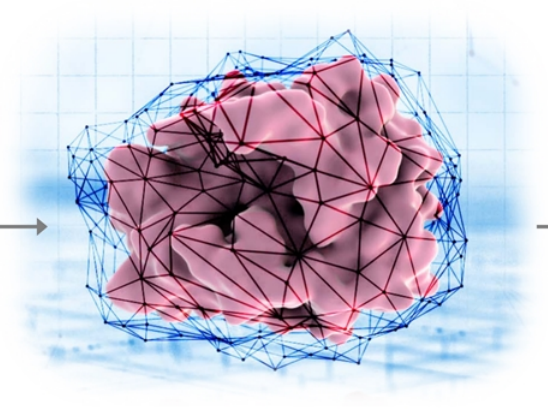
Catalyst protease platform

Unique expertise enables design of optimized & differentiated protease c

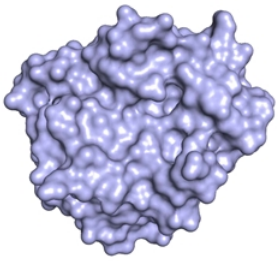
Discovery Platform

Protease Scaffold





Protease Candidate



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

Ou

+ Func
natu
the
coag
+ Engi
degr
com
+ Mod
biolo
inac

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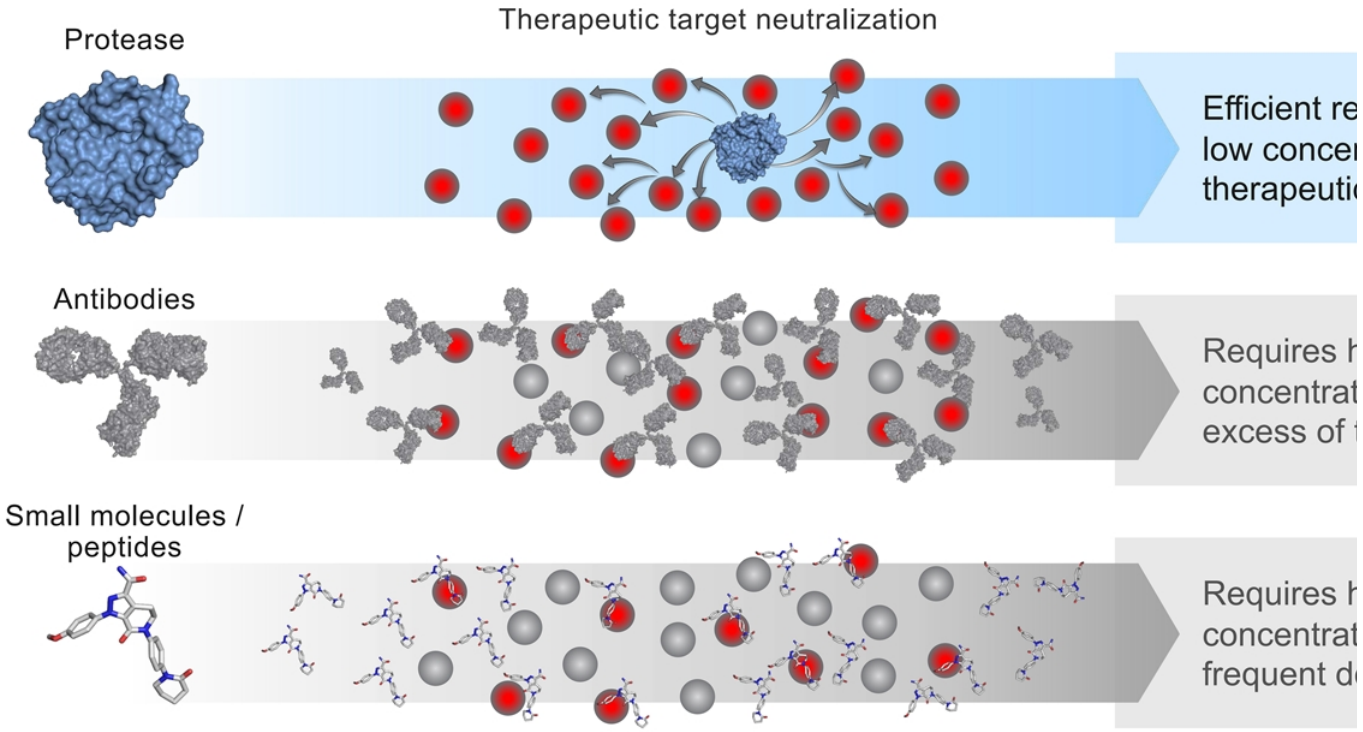
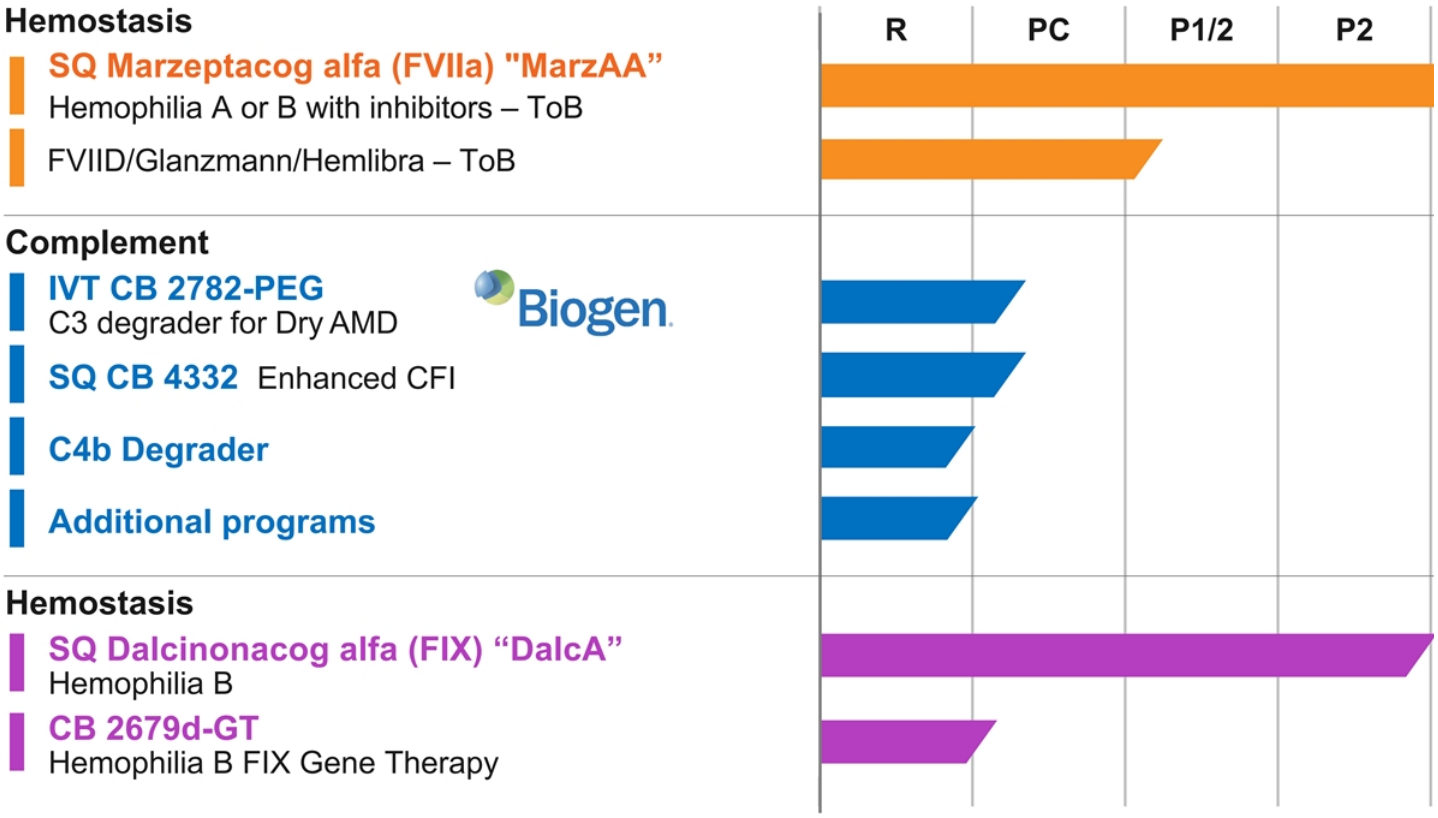
Proteases are ideal for high abundancy targets & cascad

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



Pipeline

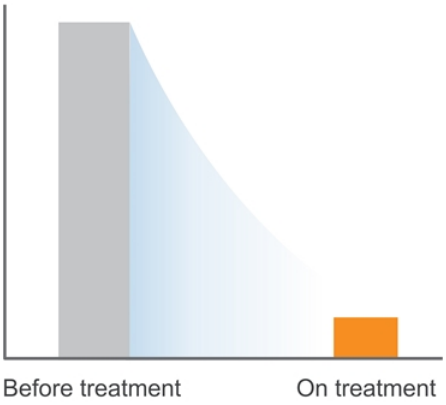


Catalyst protease platform

Validated across three programs

Marzeptacog alfa (activated)

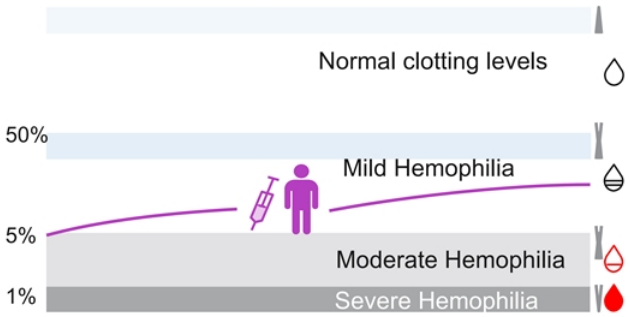
90% reduction
in annualized bleed rate



Engineered
rFVIIa protease

Dalcinonacog alfa

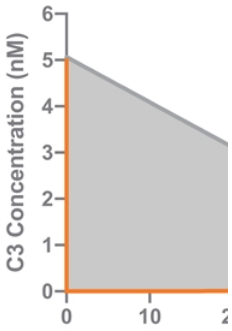
Achieved sustained
& high target levels of FIX



Engineered
rFIX protease

CB 2782-F

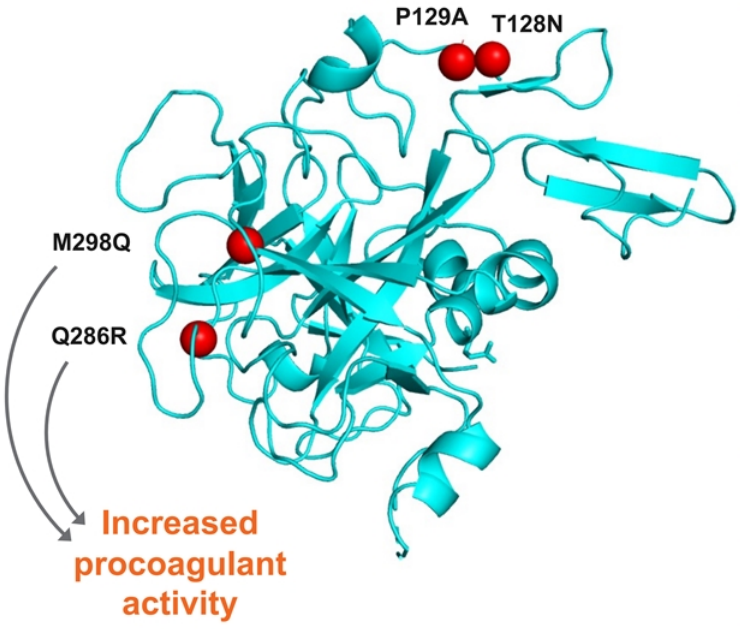
Best-in-class
Extended ph



No
C3

Marzeptacog alfa (activated) – MarzAA: SQ rFVIIa

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



Data* indicate a 9-fold higher activity

+ 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

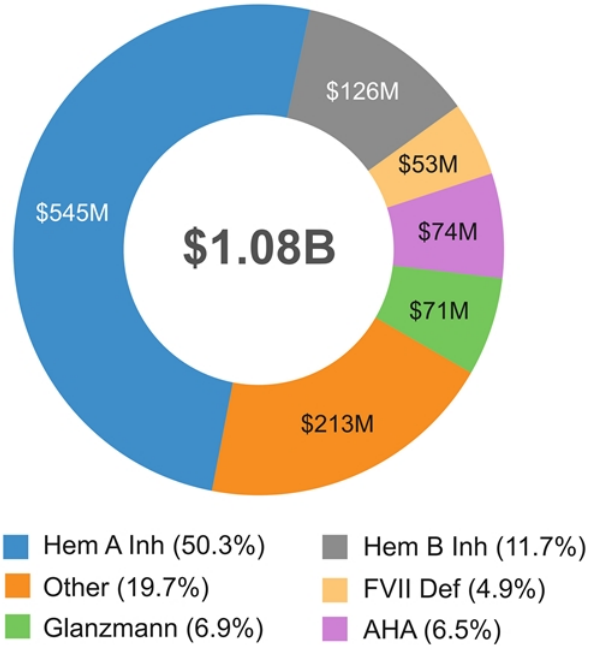
FDA Fast Track designations

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

*Pre-clinical and clinical trials

SQ MarzAA is a large commercial opportunity

Global NovoSeven sales breakdown by indication (2020)

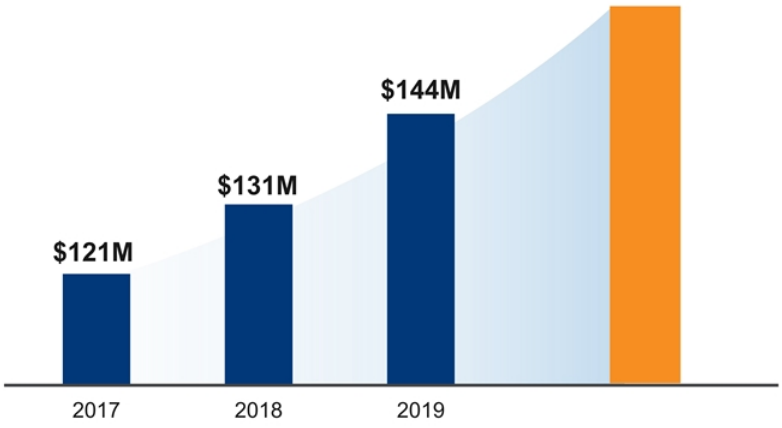


Source: Adivo Associates market research; Catalyst Biosciences market research. Data on file.

SQ MarzAA profile

+ SQ is patient-preferred & elimi barrier to fast & effective treatr
+ Ideal for pediatrics & patients v access issues
+ Long half-life without high Cma optimal control of bleeds
+ *In vitro* data support combinati Hemlibra without increased thrombogenicity
+ Prophylaxis opportunity demor P2

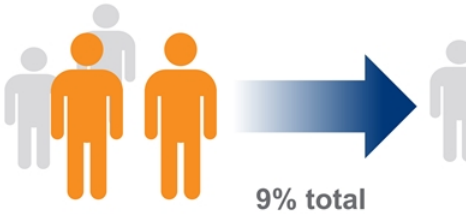
MarzAA could be the first prophylaxis for Glanzmann & F



Global NovoSeven on demand sales Glanzmann Thrombasthenia, FVIID

Unmet need in prophylaxis

Growing number of Glanz Thrombasthenia and F 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 times as patients may have bleeding events per year needing factor treatment

Unmet need for a long-acting SQ episodic treatment for 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

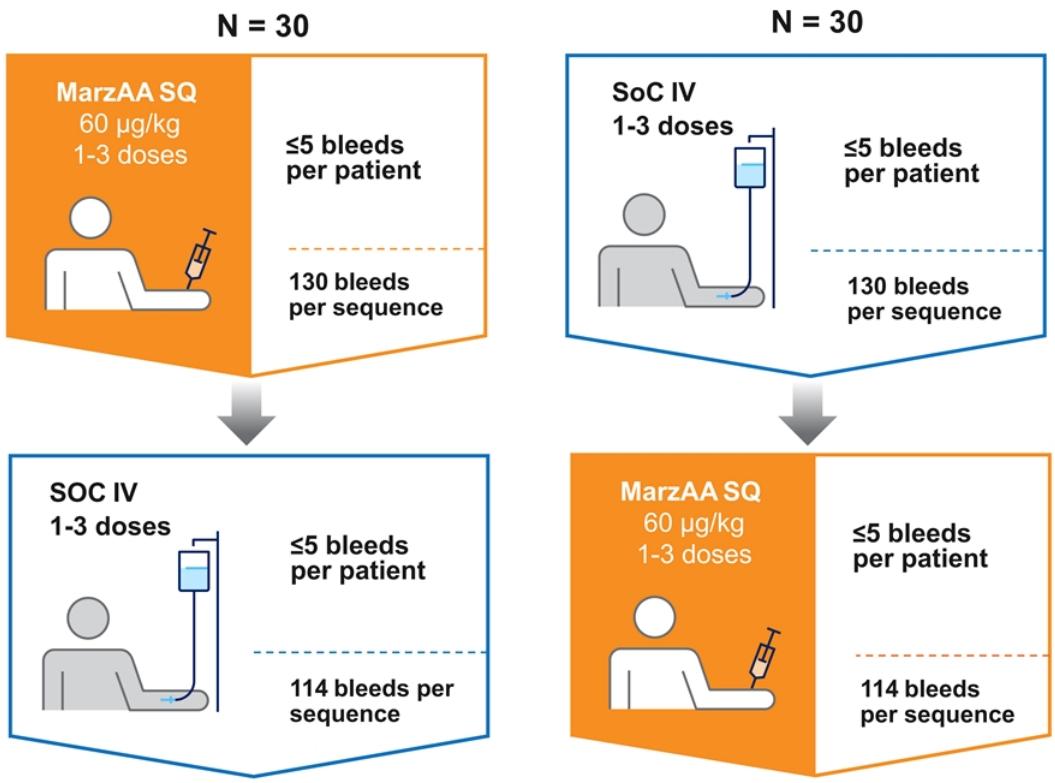
+ MAA-102: PK MarzAA lev support SQ ToB
+ Target therapeutic levels **rapidly achieved w/o a l**
+ Target levels can be main 18 hours with a single SC 60 µg/kg

Clinical ~~PK~~ MarzAA levels 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 endpoi

+ Non-inferior hem standard 4-point

Secondary end

+ Time to bleed re number of doses

Safety

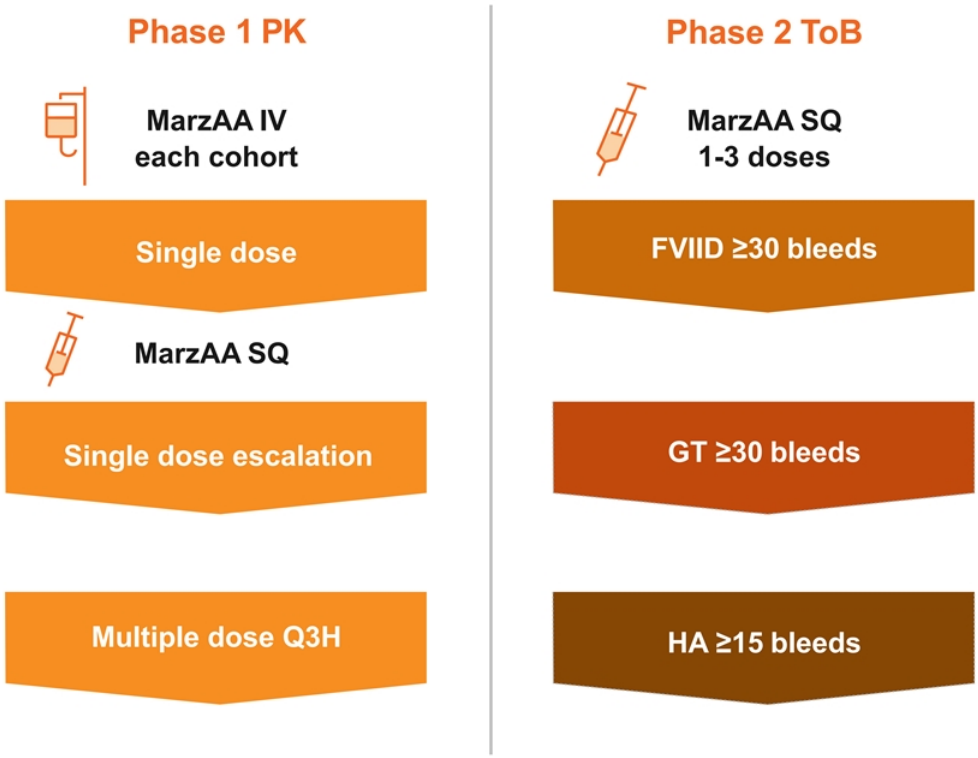
+ Adverse events, antibodies (ADA

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

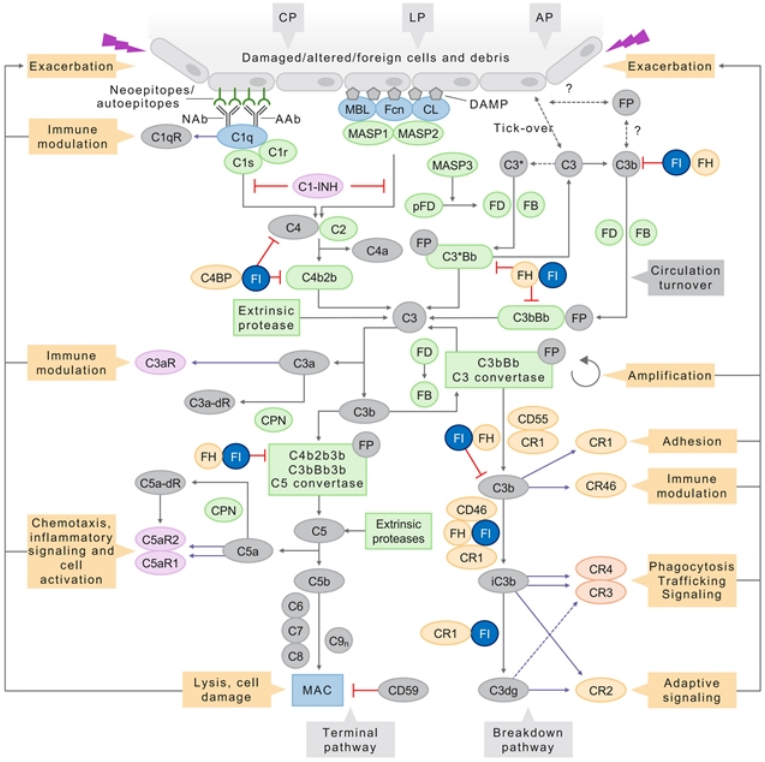
+ **Primary endpoi**
  Hemostatic effica
+ **Secondary endp**
  Effective hemost
  timepoints; dose
  rescue meds
+ **Safety:**
  Adverse events a

Growing Complement Pathway Protease Platform

CA
BI

Complement is a perfect fit to develop protease therapeu

The complement pathway is driven by a protease cascade

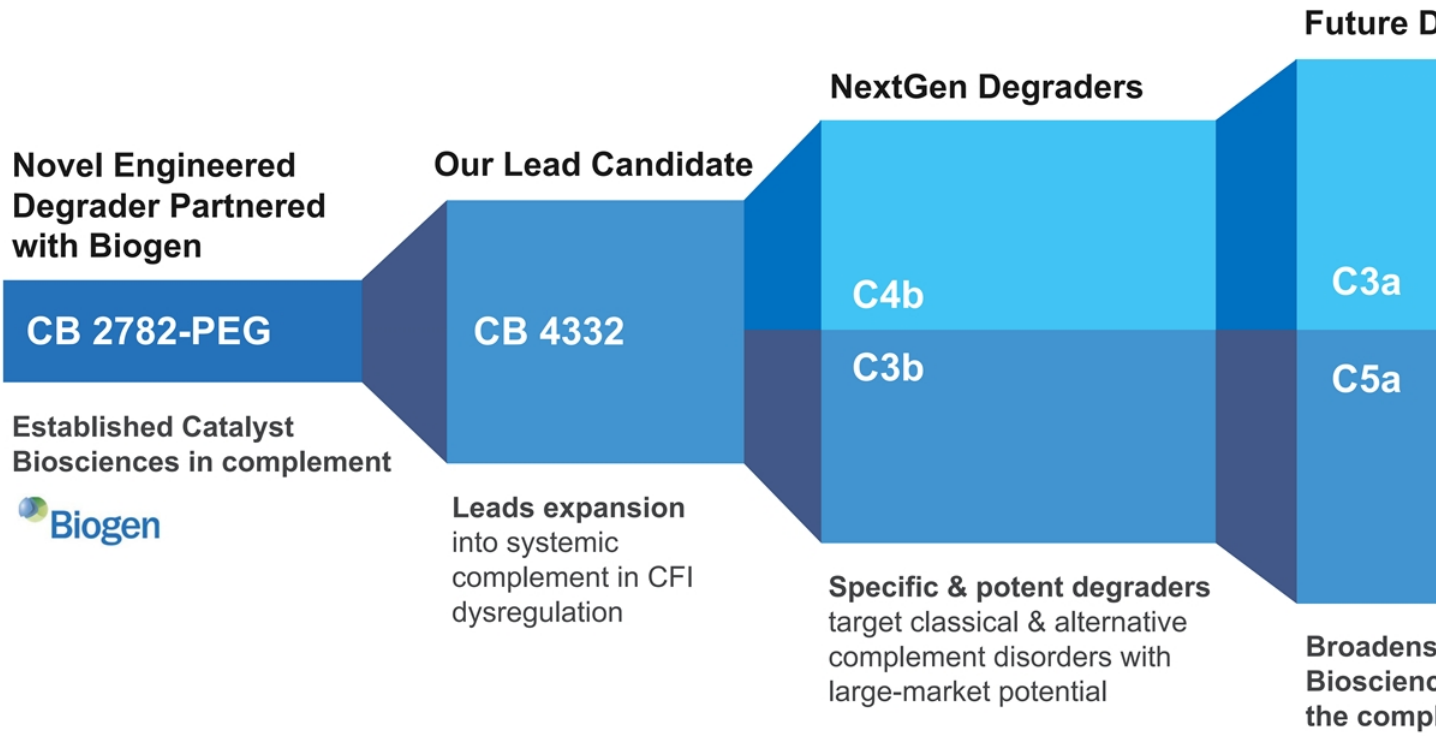


80

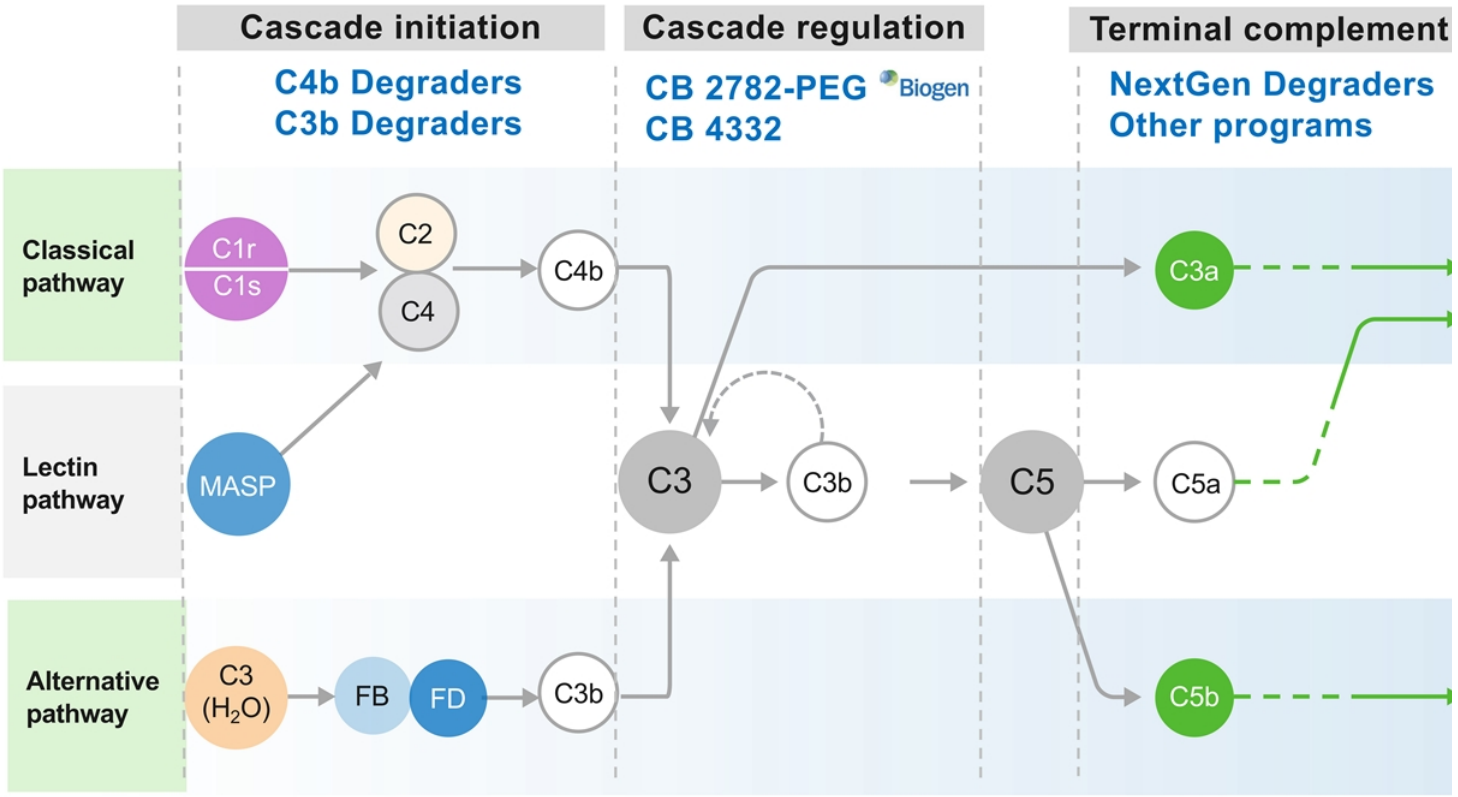
of the comple
cascade is reg
by proteas

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

Multiple, high-value complement programs



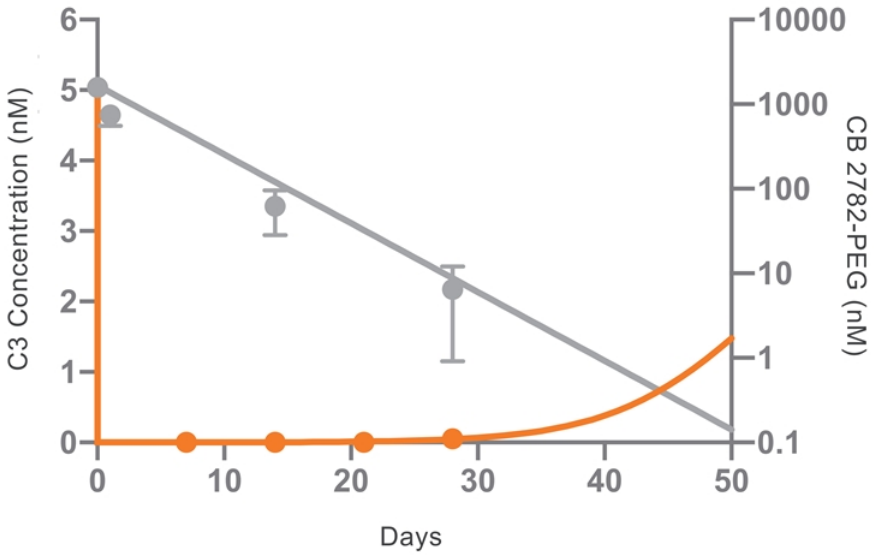
Unique targeted approach to complement regulation



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**

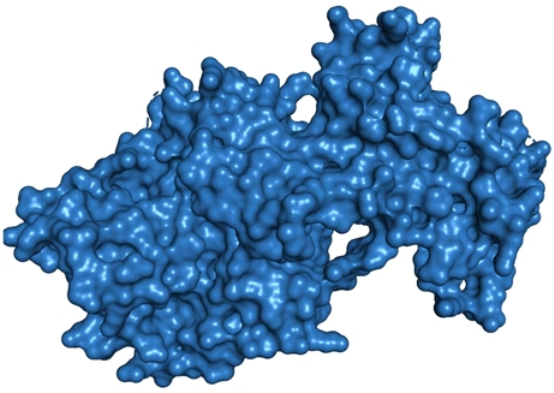
Biogen collaboratior

+ $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 & un

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

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.

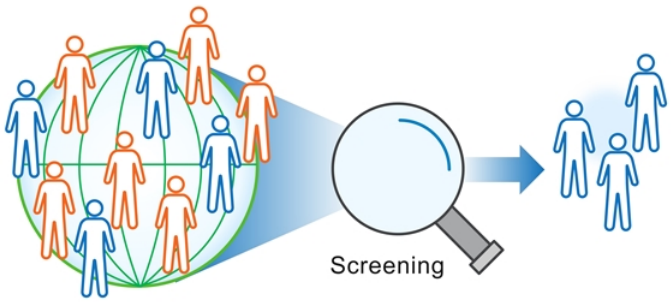
CB 4332: To address CFI deficiency at the root cause

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

The **C**on**FI**rm Study

The **C**on**FI**dence Study

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

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

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 ascendir & 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, F Bb/FB ratio, iC3b, C3d, C3dg, AP50/AH50)
+ Establish a Recommended Dose Regimen witl the CFI normal range

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Diseases with CFI mutations have tremendous potential

US / EU5 market opportunity

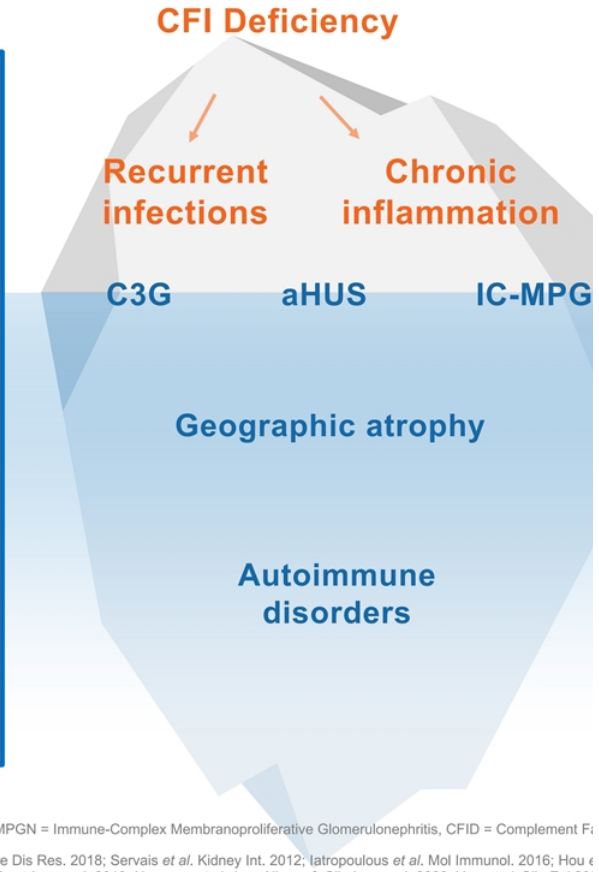
CFI Deficiency
First indication

$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



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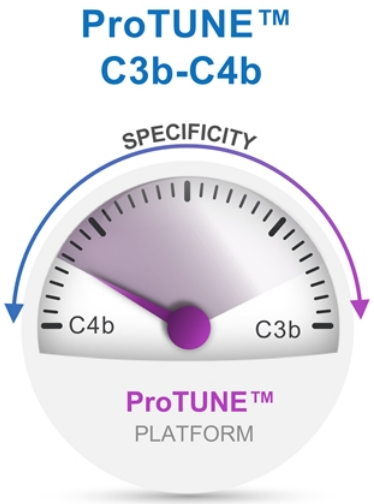
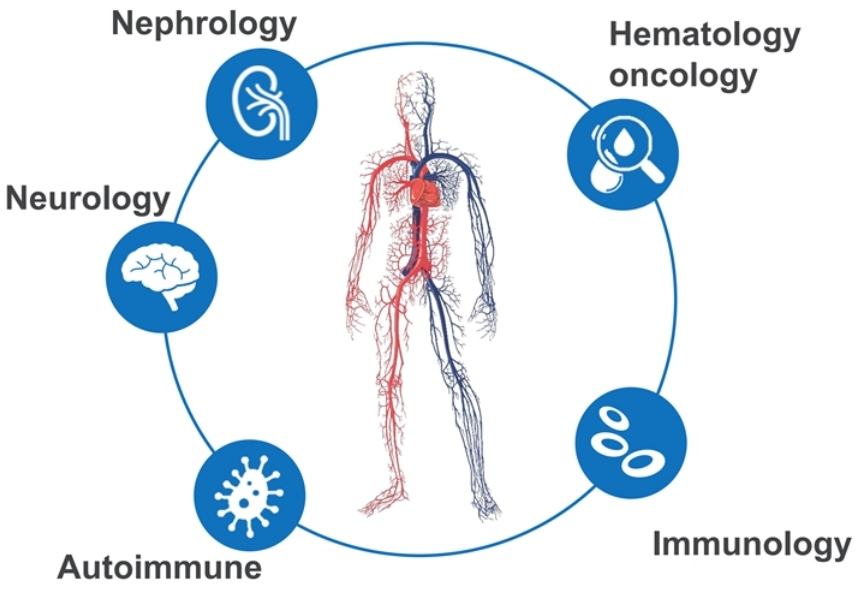
Note: aHUS = atypical Hemolytic Uremic Syndrome, C3G = Complement 3 Glomerulopathy, IC-MPGN = Immune-Complex Membranoproliferative Glomerulonephritis, CFID = Complement Fa

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 *e* 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 20 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 & immunore





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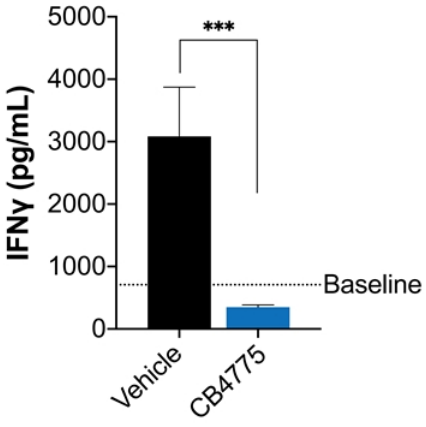
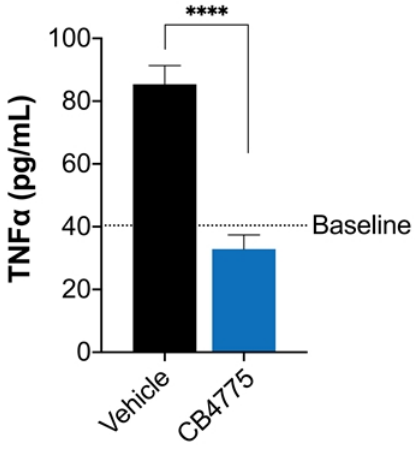
C3b-C4b degraders significantly reduce inflammation *in*

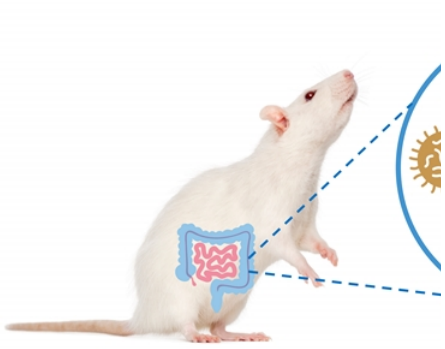
Significantly decrease in inflammatory markers involved in IgA nephr

Inflammatory markers in IgA nephropathy





Rat model of complement-media



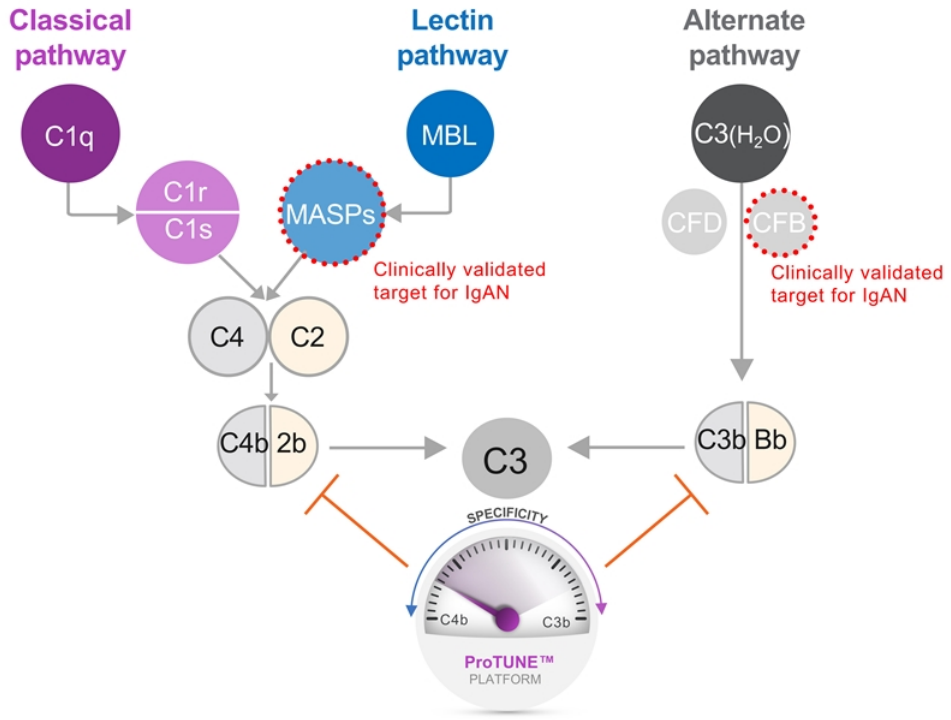
Reduction of **IFNγ** & **TNFα** involved in kidney damage & proteinuria in IgA nephropat

CATALYST BIOSCIENCES

1. Yano, N. *et al.* Phenotypic Characterization of Cytokine Expression in Patients With IgA Nephropathy. *J Clin Immunol* **17**, 396–402 (1997). 2
al. Th1/Th2 predominance and proinflammatory cytokines determine the clinicopathological severity of IgA nephropathy. *Nephrol Dial Transpl* 1
(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 a alternate pathways

Rationale for IgA neph

+ Both lectin & alternate pa involved in IgA nephropa with severe clinical mani

Clinically validated tar

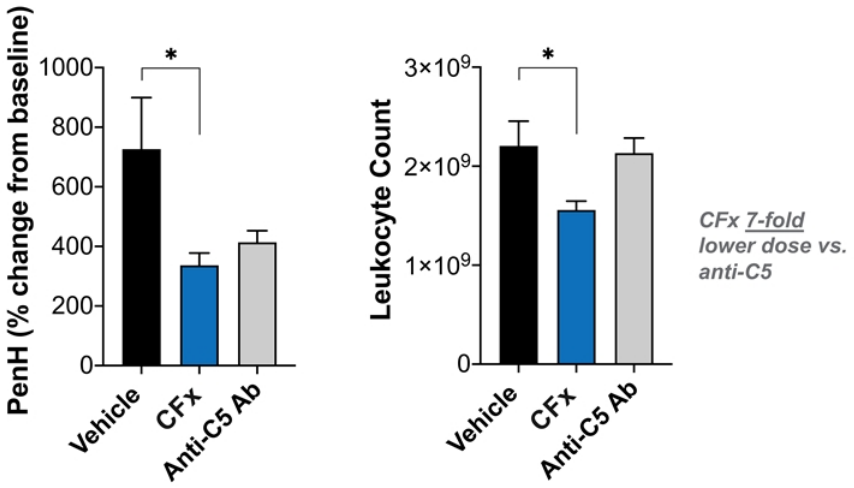
+ Inhibition of only MASP2 be insufficient to reduce nephropathy patients

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 AR

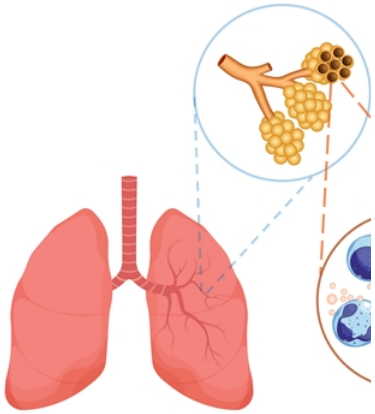
CFx improves respiratory function & reduces cell infiltrates

Respiratory functions & cell infiltration at 24 h



CFx 7-fold lower dose vs. anti-C5

Mouse LPS model of lung

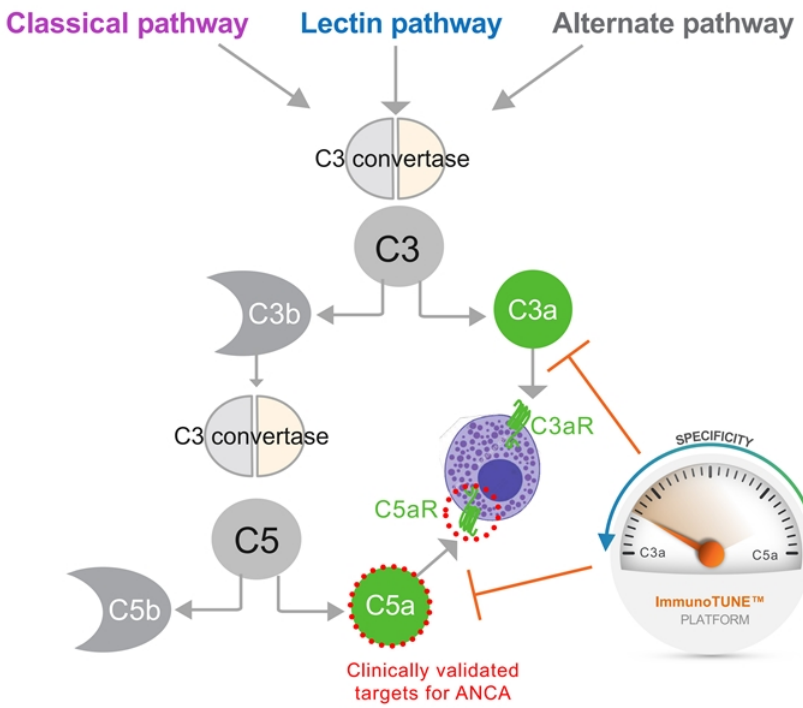


- CFx **outperforms** anti-C5 antibody in reducing inflammatory cell in
- CFx **compares well** on respiratory functions with anti-C5 antibody

CATALYST BIOSCIENCES

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 func **C5L2** pathway

Rationale for ANCA-AAV

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

Clinically validated targe

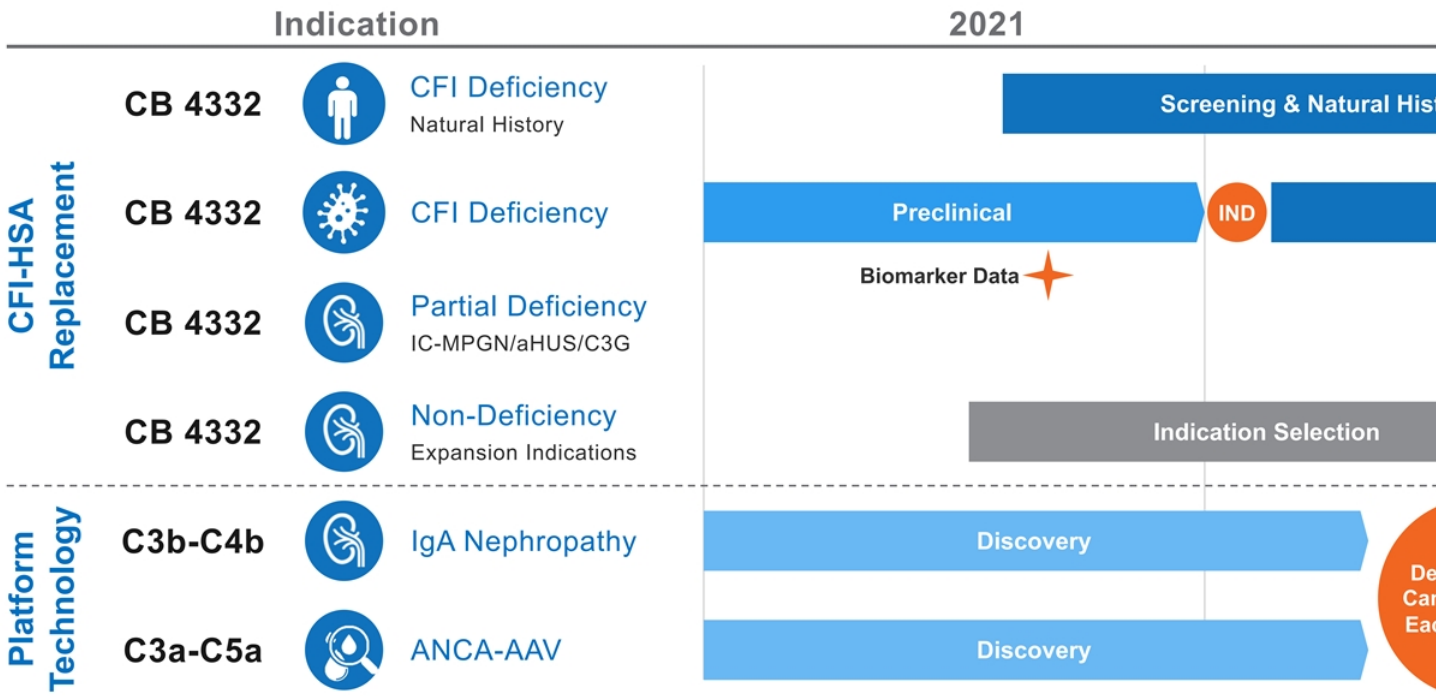
+ Inhibition of C5a or C5aR m insufficient to increase remi ANCA-AAV patients

CATALYST BIOSCIENCES

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

Next development candidate in 2022



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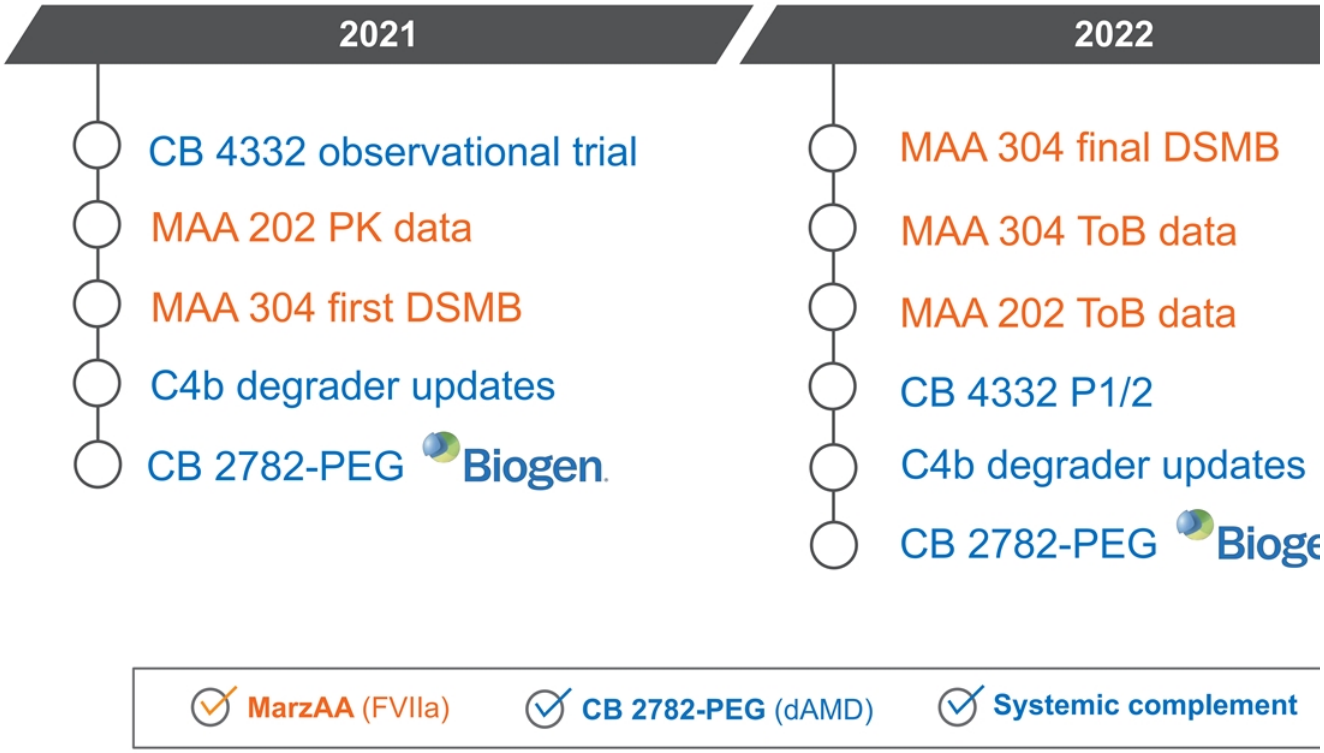
Milestones



THANK YOU

Nasdaq: CBIO

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