Nasdaq: CBIO

# HARNESSING THE CATALYTIC POWER OF PROTEASES

## Complement R&D Day

19 July 2021

CatalystBiosciences.com

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# 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; the potential markets for and advantages of the Company's complement product candidates, including CB 2782-PEG, CB 4332 and complement degraders; plans for the Company's collaboration with Biogen; and plans to 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 2022. 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, 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 May 6, 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.

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# Welcome

# Catalyst Biosciences:
## The Protease Medicines Company

**Nassim Usman, Ph.D. | President & CEO**

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# Complement R&D Day – July 2021

## Agenda

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

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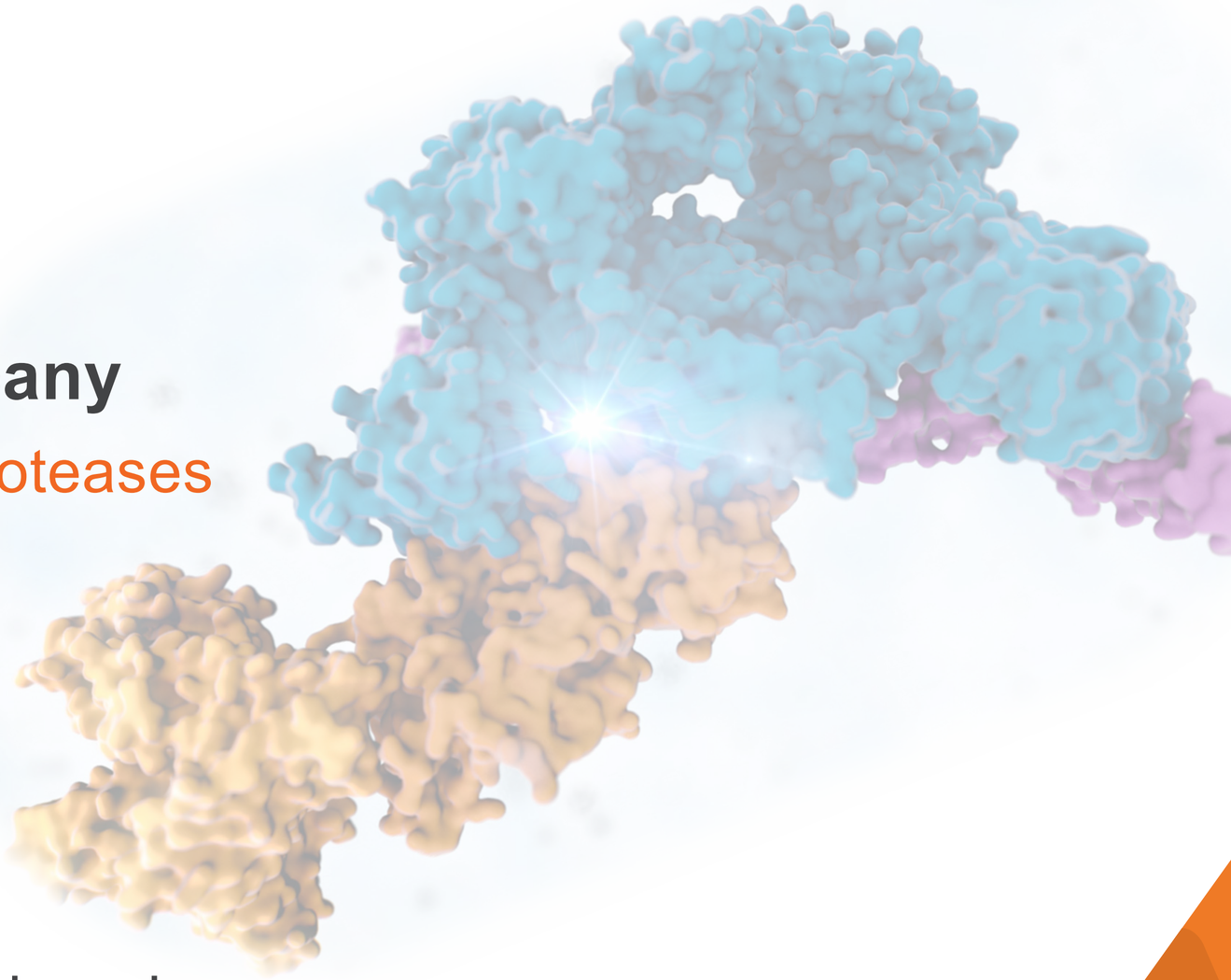
# The Protease Medicines Company

## Harnessing the catalytic power of proteases



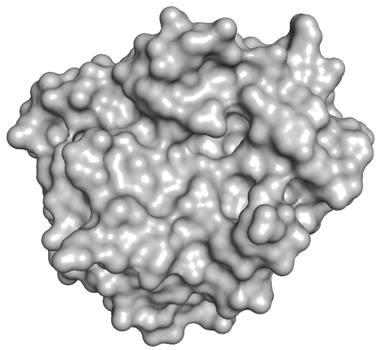
- Novel differentiated medicines
- Robust complement portfolio
- Clinical-stage assets
- Unique expertise in protease engineering

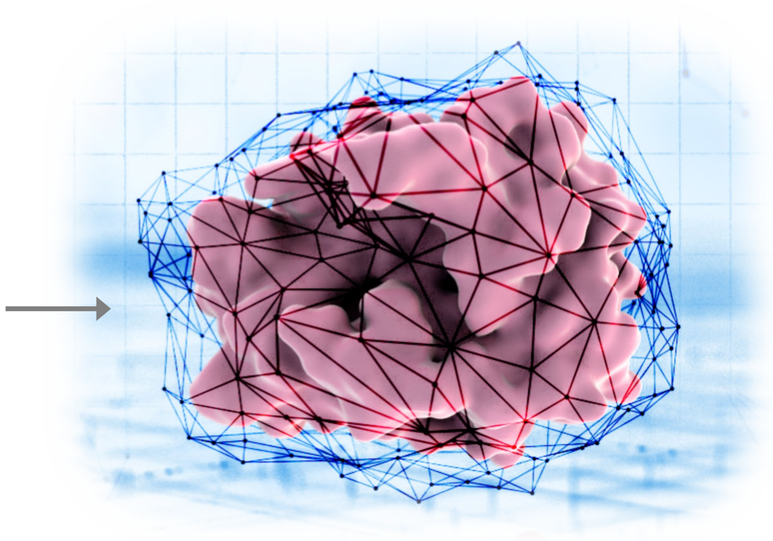
# Catalyst protease platform

## Unique expertise enables design of optimized & differentiated protease candidates

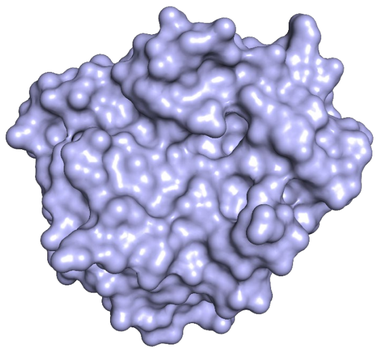
### Discovery Platform

Protease Scaffold





Protease Candidate



- 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

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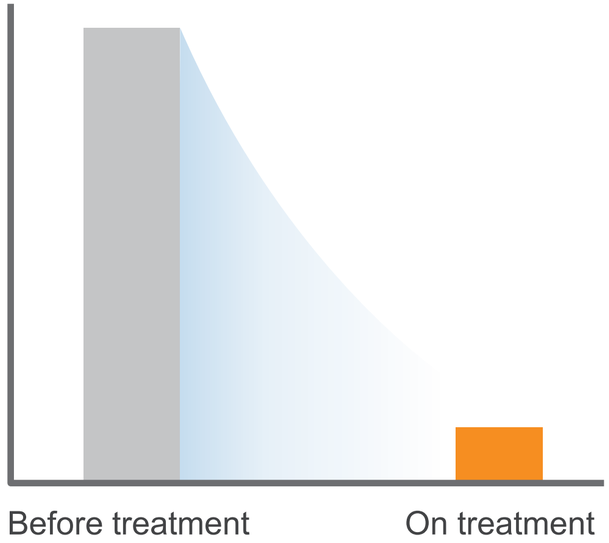
# Catalyst protease platform

## Validated across three programs

### Marzeptacog alfa (activated)

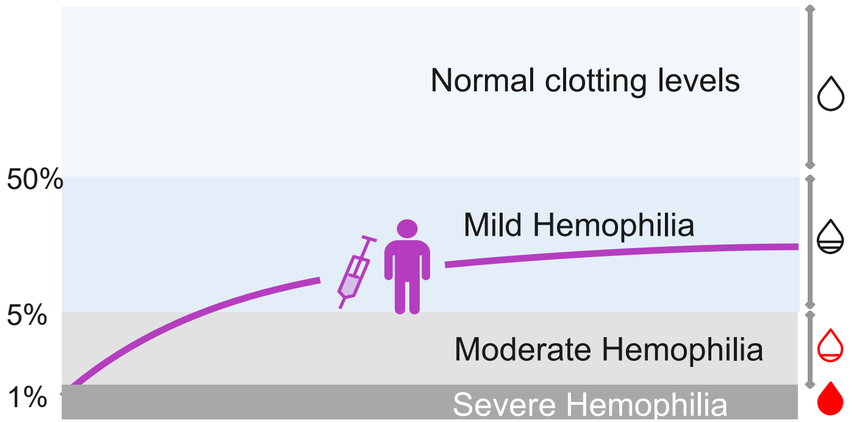
90% reduction
in annualized bleed rate




Engineered
rFVIIa protease

### Dalcinonacog alfa

Achieved sustained
& high target levels of FIX

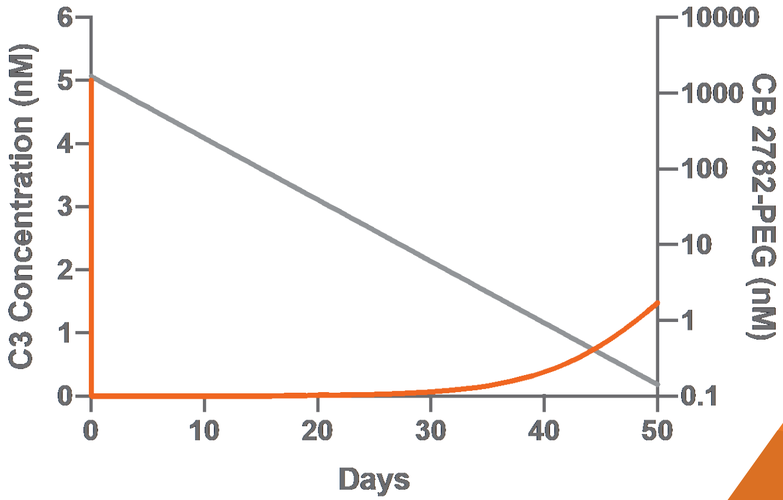



Engineered
rFIX protease

### CB 2782-PEG

Biogen

Best-in-class profile for dry AMD
Extended pharmacodynamics




Novel
C3 degrader

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# The Need for Complement Factor I Replacement

**Filomeen Haerynck, M.D., Ph.D. | KOL, University of Ghent**

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# Complement factor I deficiencies: *more than meets the eye*


**Prof. Dr. Filomeen Haerynck, MD, PhD**

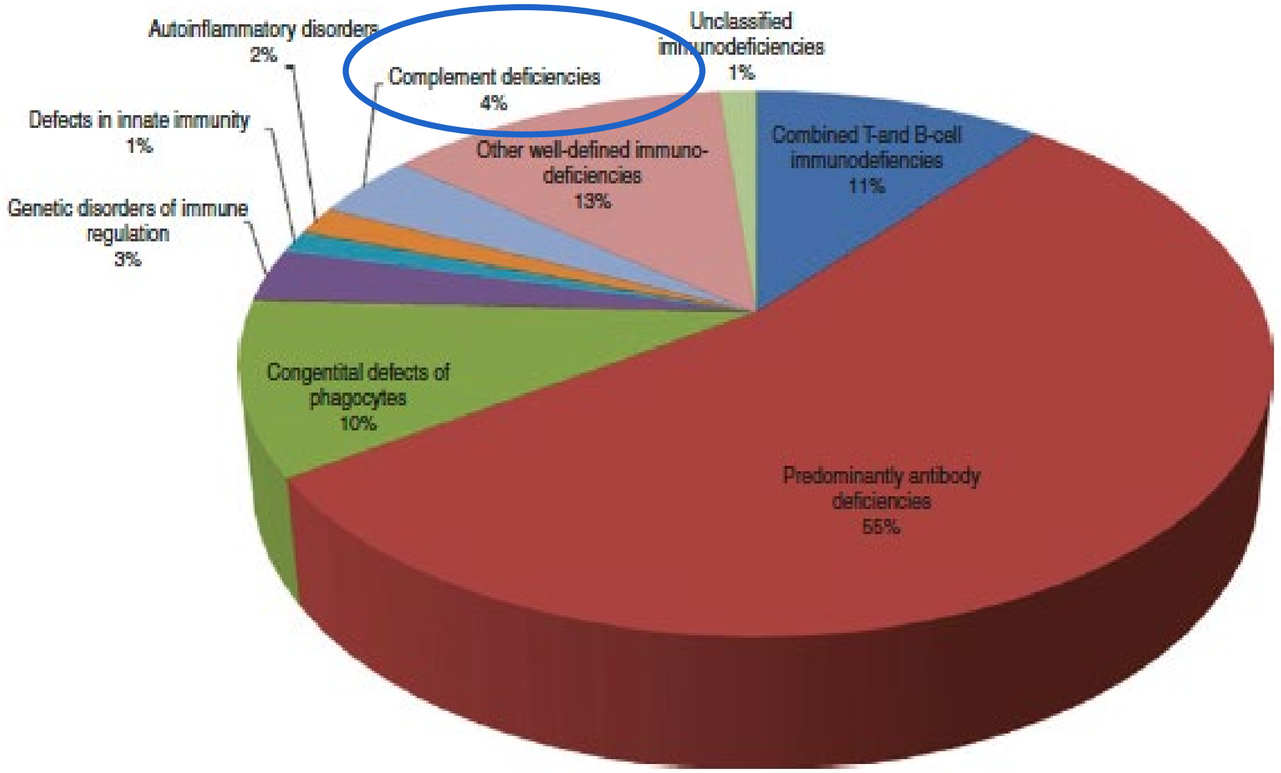
Centre for Primary Immune deficiency Ghent (CPIG)
PID research lab (PIRL)
Jeffrey Modell Foundation Diagnostic and Research centre
Ghent University Hospital
Belgium

UZ GENT

UNIVERSITEIT GENT

Catalyst Bioscience meeting 19 July 2021

# Primary immune deficiencies > 400 different types

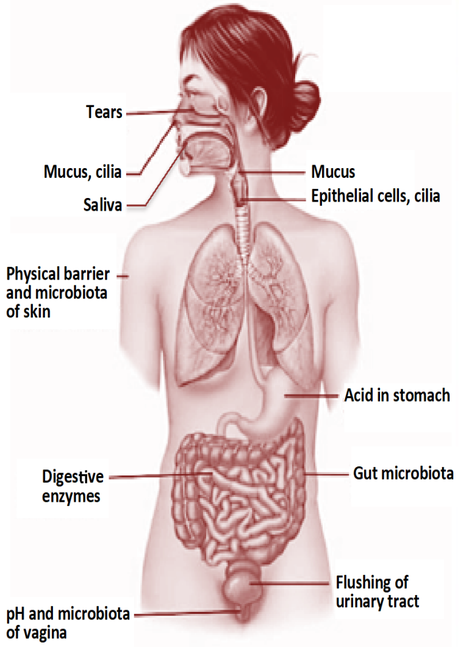


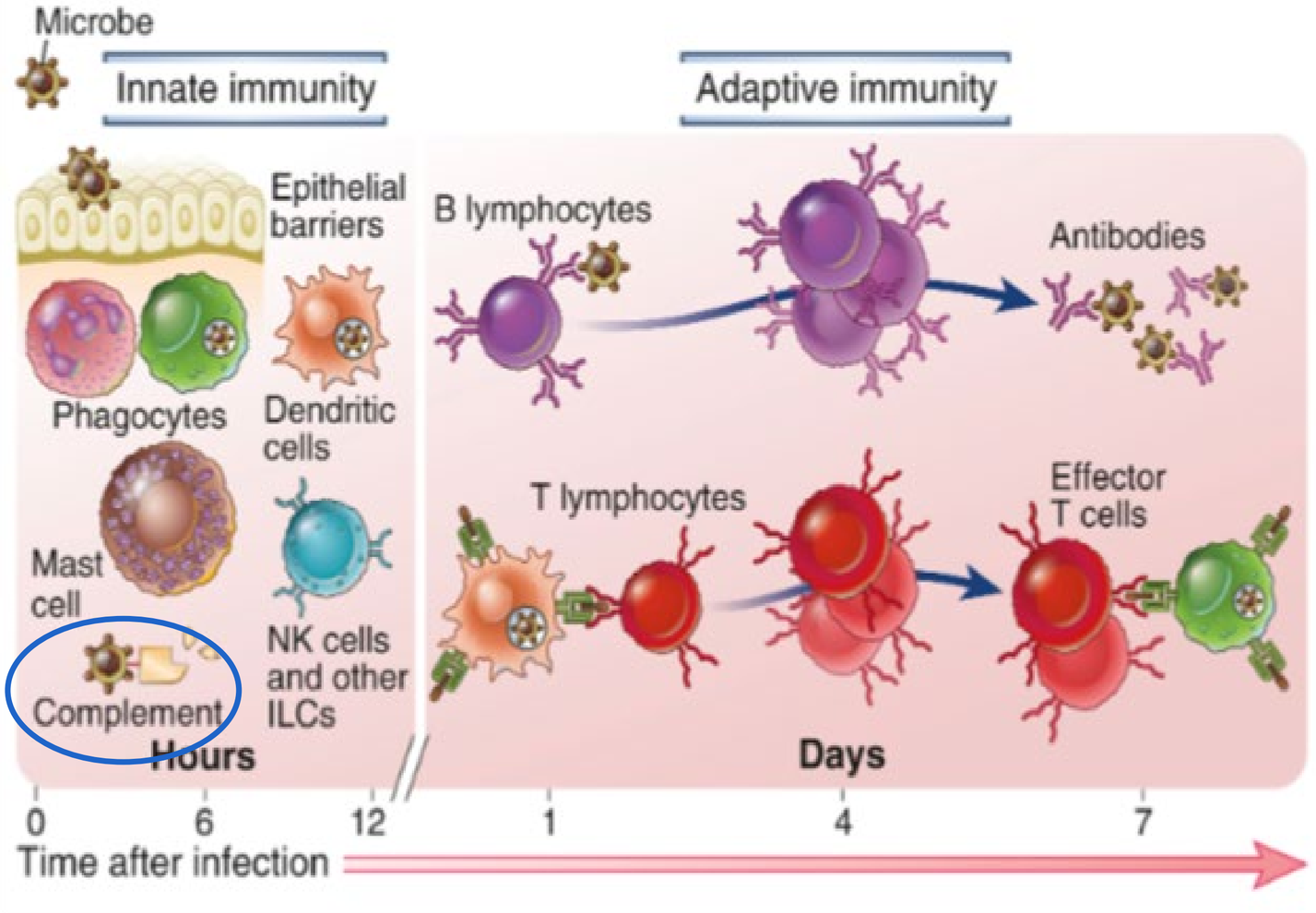

- Incidence of PID: < 1/2000
- Complement disorders: 3-5% of all PID patients (US/Europe)
  - 4900 patients
- Ghent University Hospital: 8-10% of PID cohort
  - 150 patients

*Immunol Res 2018; 66:367-380*
*JACI in press 2021 Thalhammer J*

# First line immune response

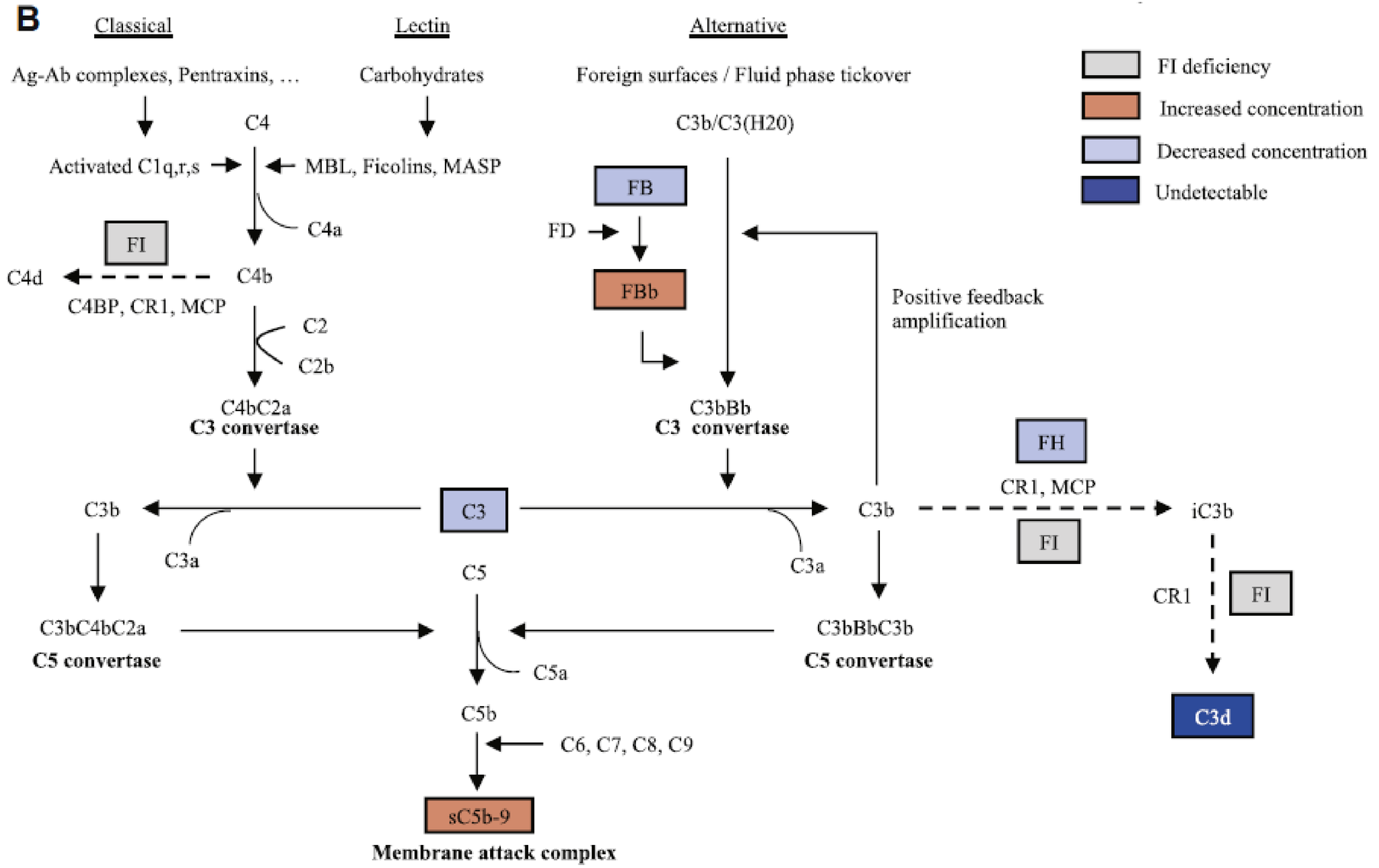
Anatomische barrière




*Abbas – Clinical immunology*

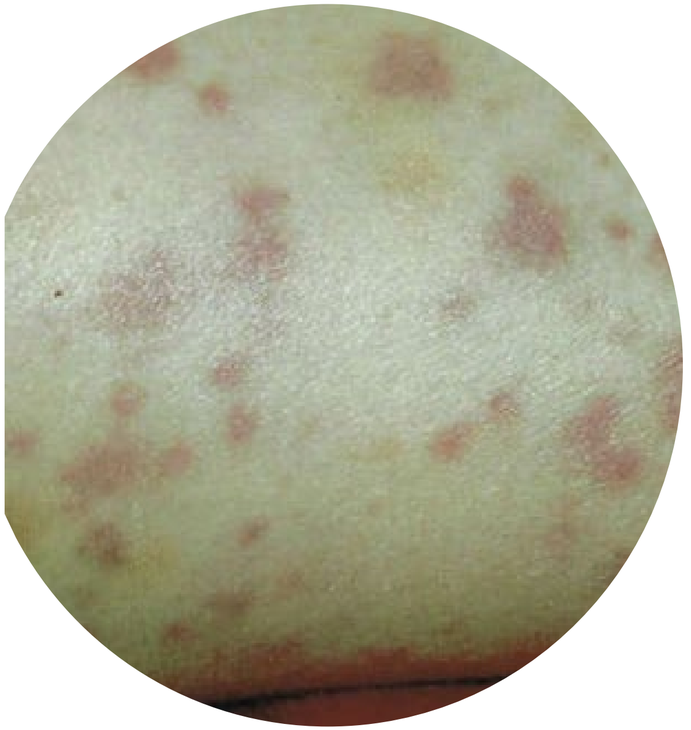
# Complement factor I: complement regulator




*Naesens L., Haerynck F. JACI 2021*

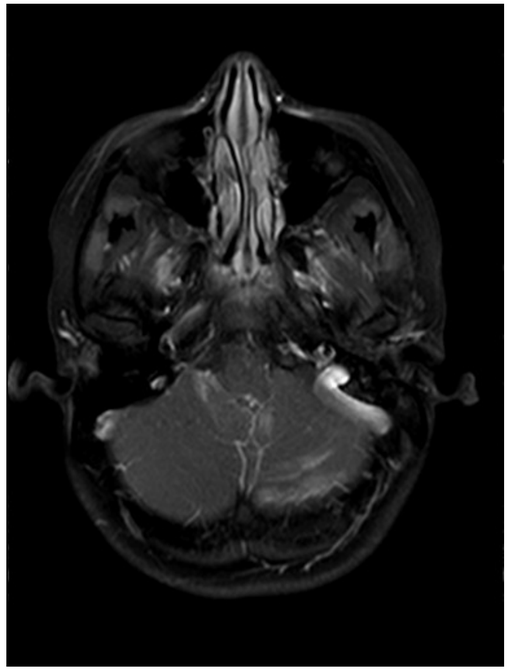
# Patient, 20 years

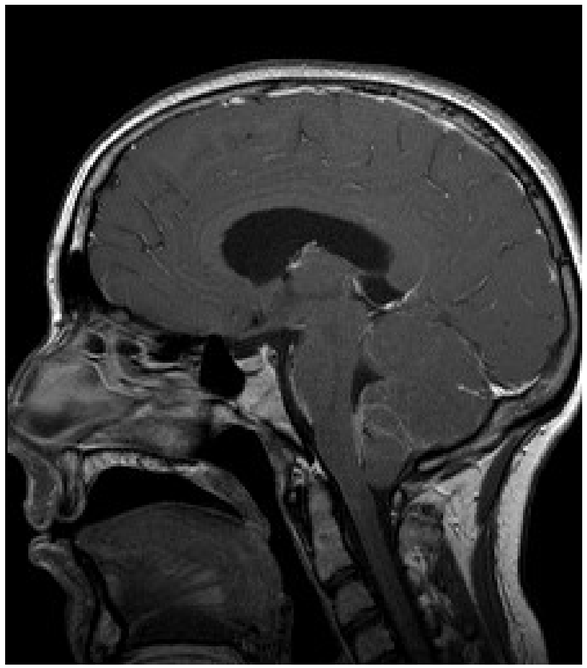
- 5 years: recurrent upper respiratory tract infections (otitis/mastoiditis)
  - monthly antibiotics
- 12 years: recurrent leucocytoclastic vasculitis
  - Rheumatological work-up: negative
  - C3 : 0,6 g/l ( 0,9-1,8 )  C4 : 0,1 ( 0,1 – 0,4 )
- Familial history: normal



# Patient, 15 years - old

- Recurrent episodes of acute headache, neck stiffness, N. Facialis paralysis, diplopia
- Lab:
  Mild neutrophilia, moderate elevated CRP, sedimentation rate
- Cerebrospinal fluid:
  neutrophils, increased protein, IgG, cultures negative
- MRI brain: Global abnormal signal intensity in cerebral cortex of cerebrum and cerebellum



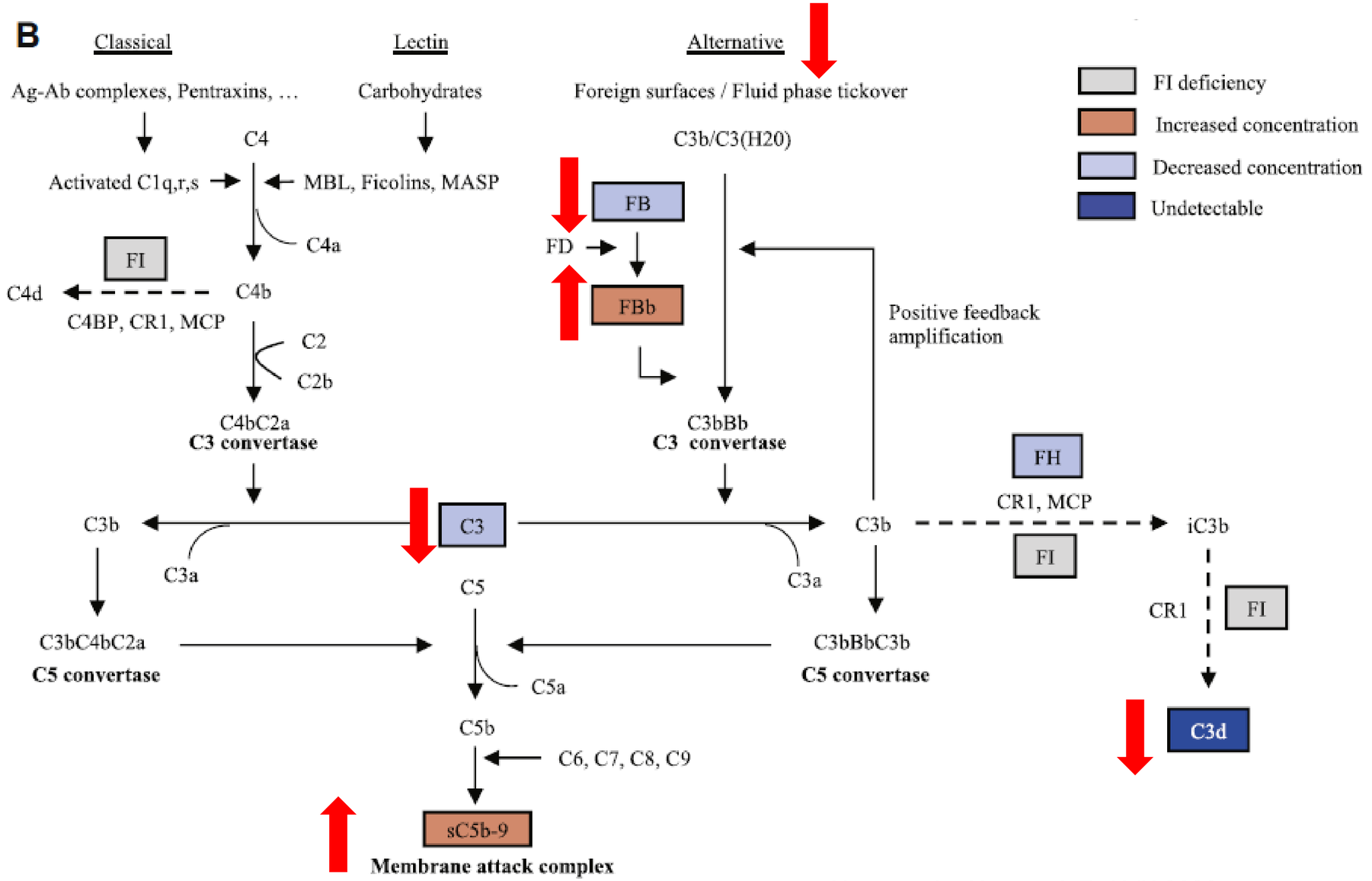


- **Recurrent aseptic meningo-encephalitis**
  - Treatment: steroids – plasmapheresis – immunosuppressive treatment (mofetil mycophenolate (MMF)
  - Persistent low complement C3 level

**→ COMPLEMENT INVESTIGATION**

# Complement investigation

**Serum Factor I concentration: normal**
**Serum Factor H concentration: normal**
**Factor H activation: normal**




*Naesens L., Haerynck F. JACI 2020*

# Complete factor I deficiency: different faces

*Naesens L. , Haerynck F. JACI 2021 Febr, 147*

| Characteristic | Patient A | Patient B | Patient C | Patient D | Patient E | Patient F | Patient G |
|---|---|---|---|---|---|---|---|
| Sex | Female | Female | Male | Male | Female | Female | Female |
| Age of onset | 3 mo | 7 y | 3 y | 12 y | 11 y | 13 y | 16 y |
| *CFI* mutation | Homozygous. c.257G>A p.(C86Y) | Compound heterozygous. c.355G>T p.(G119*) Exon deletion 2-13 | Homozygous. c.257G>A p.(C86Y) | Compound heterozygous. c.1367G>T p.(W456L) Exon deletion 2-13 | Homozygous. c.1015C>T p.(R339*) | Compound heterozygous. c.1367G>T p.(W456L) c.772G>A p.(A258T) | Compound heterozygous. c.1019T>C p.(I322L) c.1571A>C p.(D524V) |
| | Tortajada et al[8] | New | Tortajada et al[8] | Bienaime et al[5] | New | Bienaime et al[5] and Kavanagh et al[4] | Haerynck et al[6] Fremeaux-Bacchi et al[1] |
| Clinical features | | | | | | | |
| | Infectious | Infectious | Infectious | Infectious | Infectious/ Autoimmune | Autoimmune | Autoimmune |
| | *S pneumoniae* meningitis | *N meningitidis* meningitis | *S pneumoniae*, *S pyogenes* meningitis | *S pneumoniae* otitis and bacteremia | Vasculitis (cutaneous, cerebral), *S pyogenes* bacteremia, *N meningitidis* meningitis | Bickerstaff encephalitis, leukocytoclastic vasculitis | Aseptic meningoencephalitis, leukocytoclastic vasculitis |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| **Factor I conc (mg/dl)** | **<1,5** | **1,6** | **< 1,5** | **<1,5** | **3,3** | **<1,5** | **4,4** | (ref: 4-10) |
| **CH50 (U/mL)** | | **6** | **3** | **<13** | **18** | **<13** | 46 | 32 (ref: 23-63) |
| **AP50 (%)** | **0** | **0** | **0** | **0** | **0** | **0** | **0** | (ref:30-113) |
| **C3 (mg/dL)** | **19** | **23** | **42** | **25** | **22** | **42** | **57** | (ref 72-156) |
| **Factor B (mg/dL)** | **1** | **2** | **2** | **4** | **5** | **2** | **1** | (ref: 11-22) |
| **Factor Bb (mg/dL)** | **0,54** | **0,262** | **0,79** | **0,54** | **0,74** | **0,89** | **0,51** | (ref <0,15) |
| **C3d (mg/dL)** | <0,4 | <0,4 | <0,4 | <0,4 | <0,4 | <0,4 | <0,4 | |

# Complete factor I defect: different features

- Absent serum complement factor I = **Type I CFI deficiency**
- Normal serum complement factor I but dysfunction = **Type II CFI deficiency**
- Autosomal recessive disease: homozygous/compound heterozygous *CFI gene* mutations

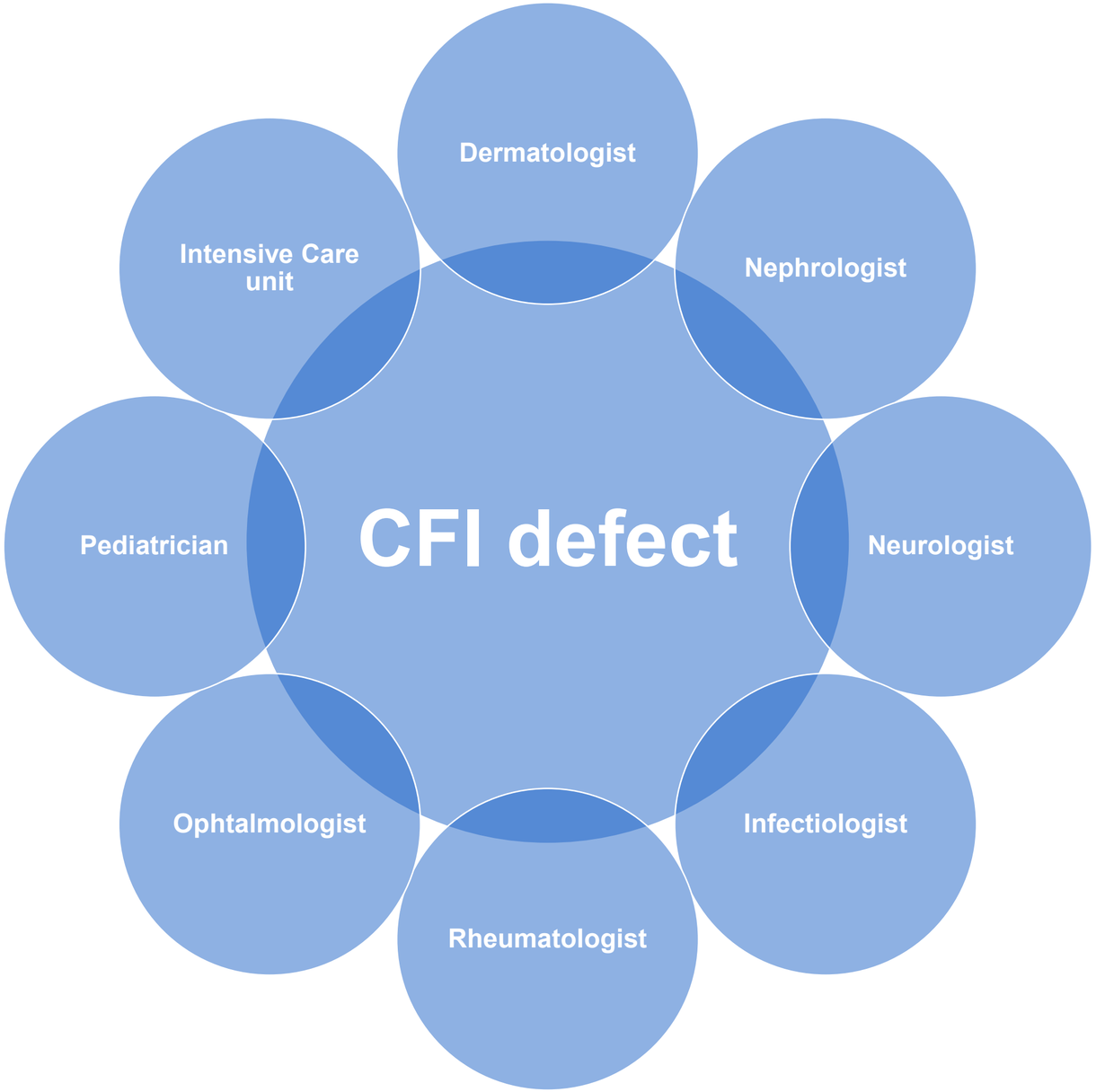
- **Infectious disease**:
  - Systemic invasive infections with encapsulated micro-organisms (*S. Pneumoniae, N. Meningitidis, H. Influenzae type B*)
  - septicemia, meningitis, osteomyelitis, septic arthritis, peritonitis, endocarditis)
- **Neurological disease**:
  - Recurrent haemorrhagic leukoencephalitis
  - Aseptic/neutrophilic central inflammation
- **Dermatological disease**:
  - recurrent leucocytoclastic skin vasculitis
- **Renal disease:**
  - Glomerulonephritis
- **Rheumatological disease:**
  - Juvenile idiopathic arthritis, Systemic lupus erythematosus

# Partial factor I deficiency: different features

- Heterozygous *complement factor I (CFI) gene* mutation

- Asymptomatic
- **Renal disease**: aHUS, C3 glomerulonephritis
- **Ophtalmological disease:** age-related macular degeneration (AMD)

# CFI defect: ‘more than meets the eye



# Current available treatment: only prophylaxis!

- Daily antibiotic prophylaxis
- Vaccinations
  - Conjugated 13-valent pneumococcal vaccine
  - Unconjugated pneumococcal vaccine (every 3-5y)
  - ActHib
  - 4 valent Meningococcal C
  - Meningococcal B
- Treatment of acute infections/neuroinflammation (immunosuppressiva)
- Screening:
  - Nephrology
  - Rheumatology

# Challenges for patients with CFI

- **Underdiagnosis of CFI defects**

  - Diagnostic delay
  - Unexplained deaths, unexplained chronic disease
  - Chronic neurological/renal complications → ↑ morbidity, health care cost

- **No curative treatment**

  - **Life-long** risk for invasive infections, death, acute neuroinflammation, renal disease
  - **Anxiety** patients/parents

**INCREASE AWARENESS**
**NEED OF SPECIFIC CURATIVE TREATMENT TO SAVE LIVES**

CPIG Center for Primary Immunodeficiencies Ghent

**UZ Gent / CPIG**
Pediatric Pulmonology and immunology
Pediatric Nephrology and Rheumatology
Pediatric and adult hematology
Pediatric Neurology
Clinical biology
Dermatology
Pediatric Intensive Care Unit

**Centre for Medical Genetics Ghent:**
Kathleen Claes
Marieke Debruyne
Elfride Debaere

**PID research lab (PIRL)**
Simon Tavernier
Leslie Naesens
Levi Hoste
Karlien Claes
Veronique Debacker
Lisa Roels


**ULB Erasme**
Patrick Stordeur

**Institut de Pathologie et de Génétique Gosselies**
K. Dahan

**Huderf**
Alina Ferster
Sophie Blumenthal

**UZ Brussel**
Jutte Vander Werff Ten Bosch

**Hospital for Sick Children Toronto Canada**
Christoph Licht

UZ GENT UNIVERSITEIT GENT jm Jeffrey Modell Foundation

# Growing Complement Pathway Protease Platform

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

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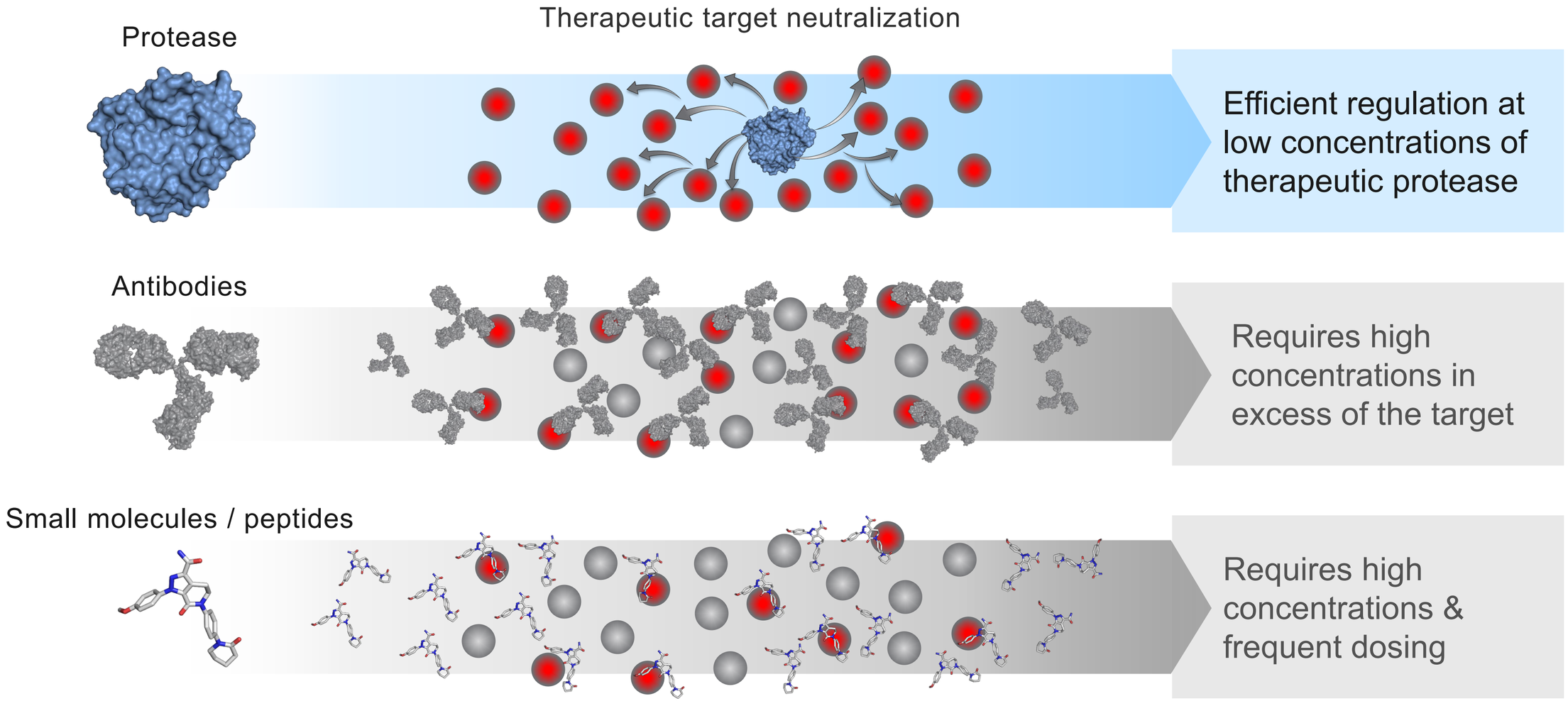
# Proteases are ideal for high abundancy targets & cascades

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

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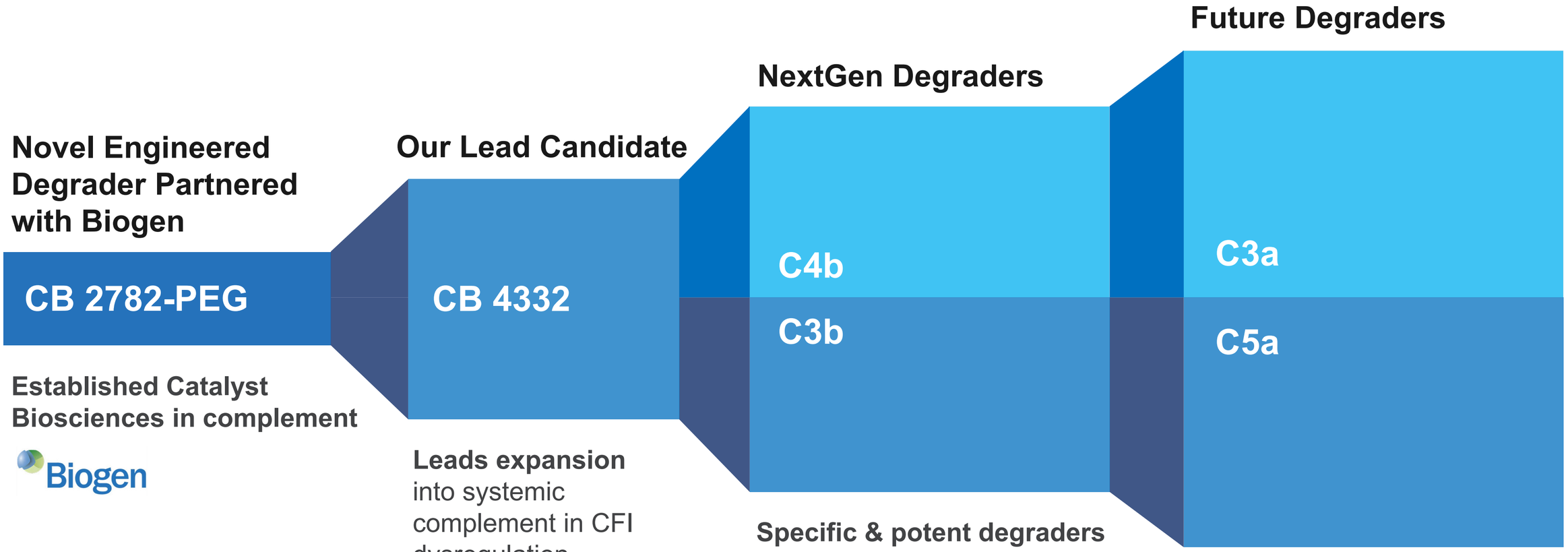
# Multiple, high-value complement programs

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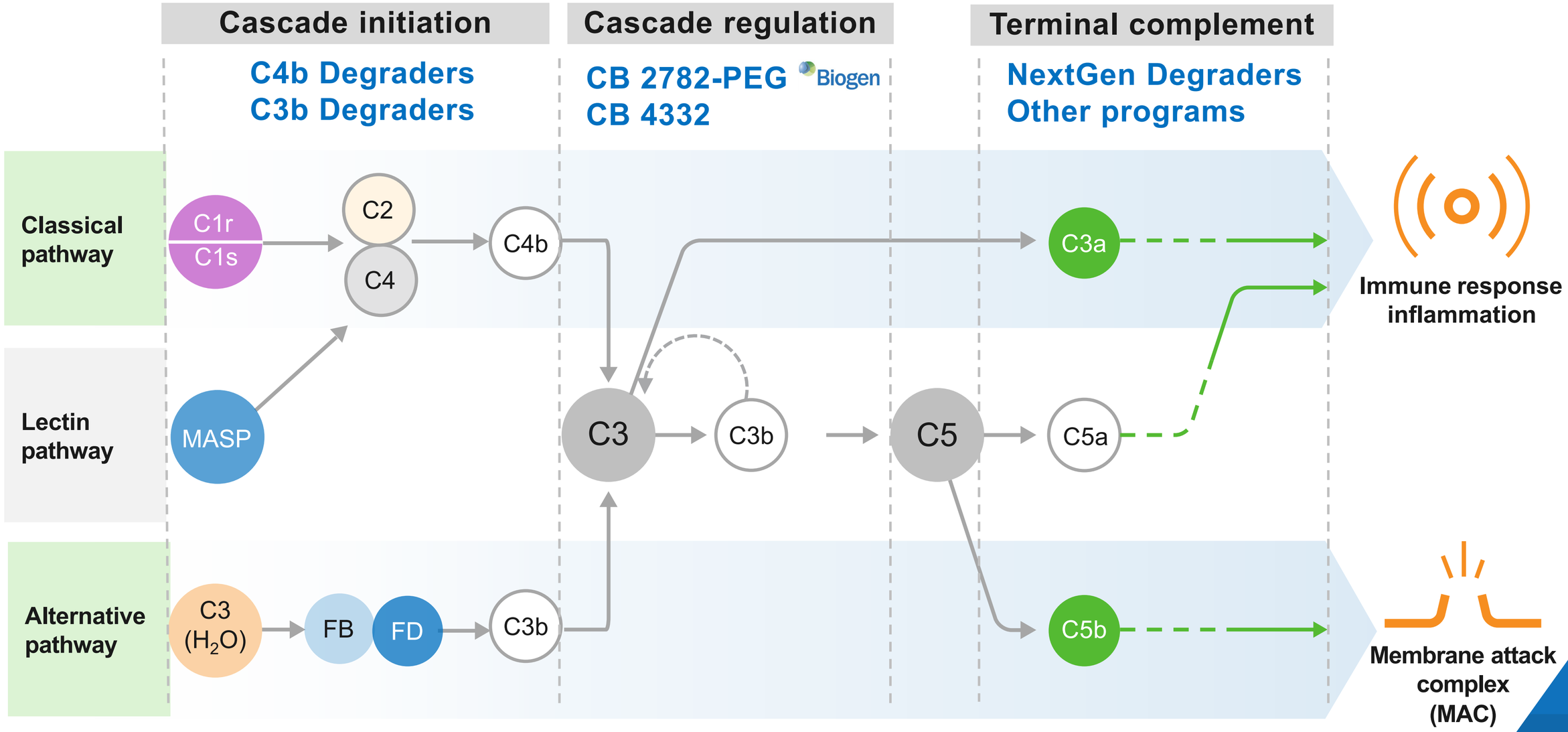
# Unique targeted approach to complement regulation

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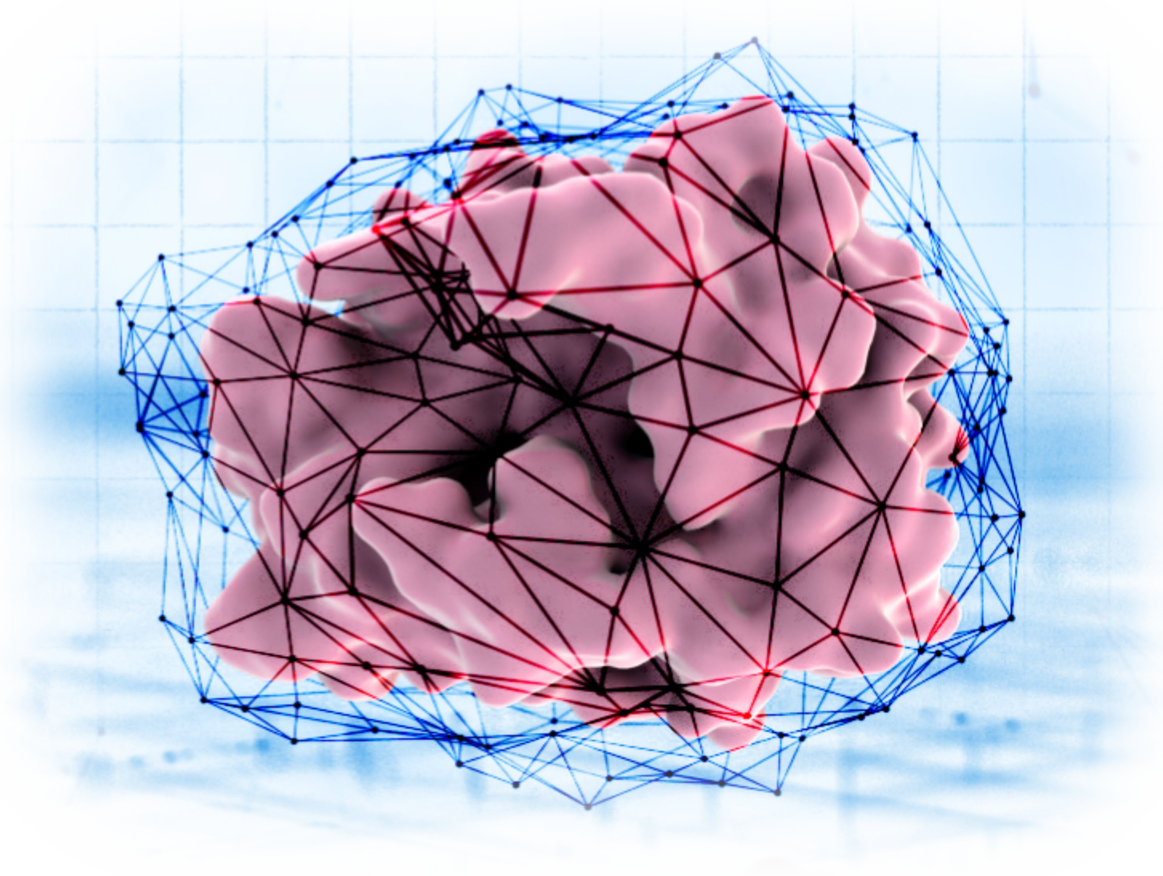
## CB 2782-PEG

# 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 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

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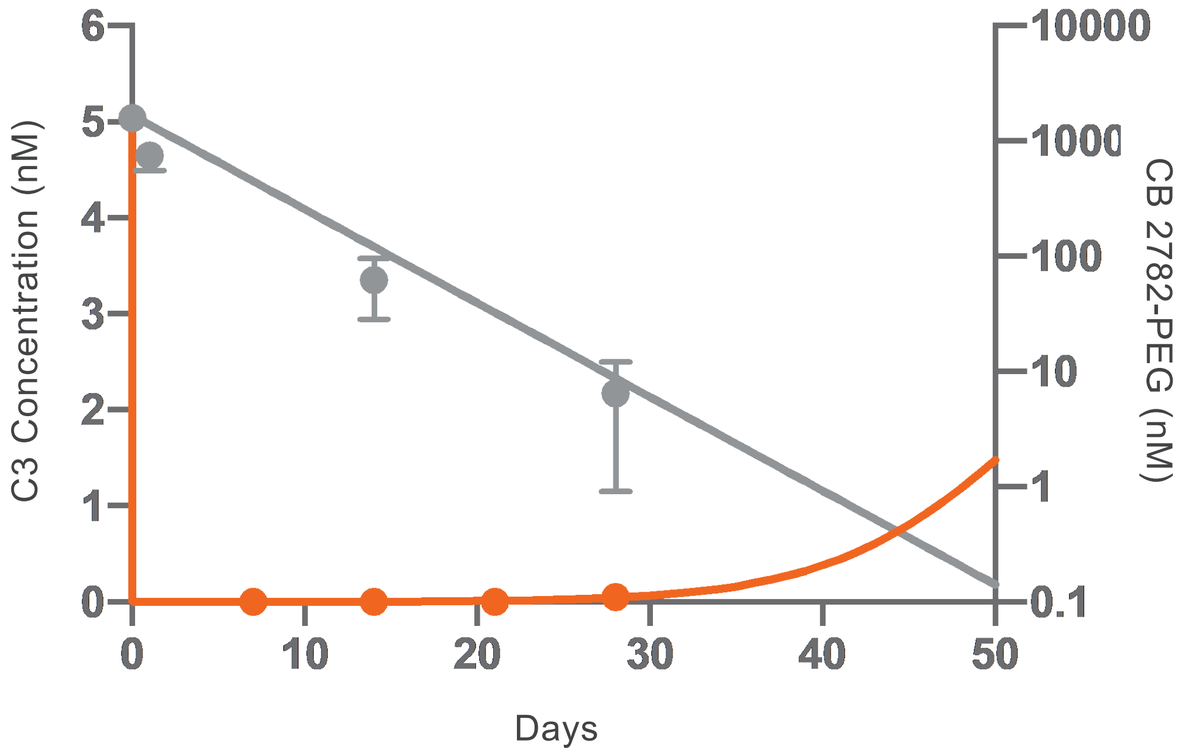
*Furfine *et al.* ARVO 2019

# 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




### 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

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# CB 2782-PEG: Comparison to APL-2 & NGM621

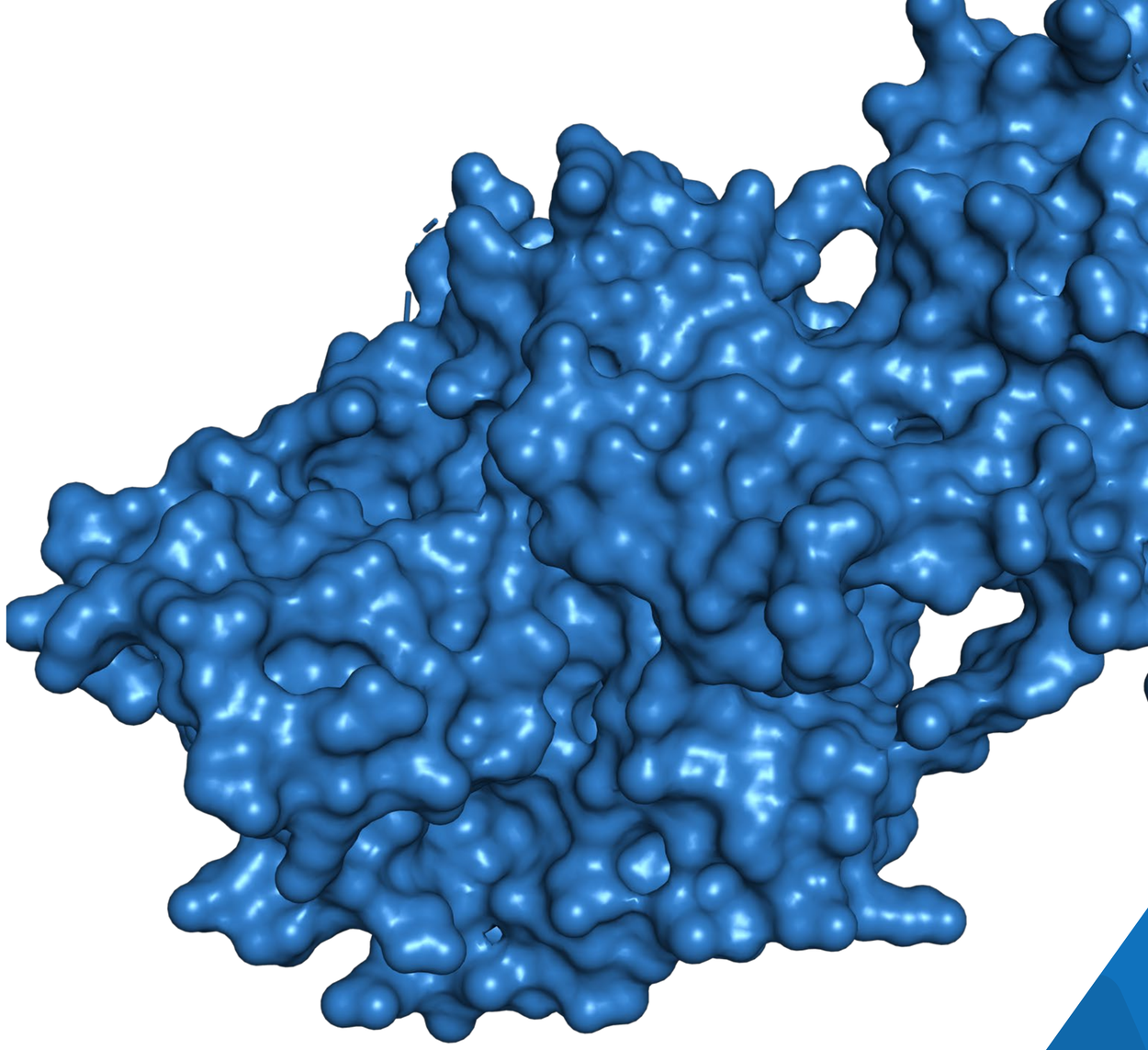
## Potential for a less frequent dosing regimen in dry AMD

| | APL-2 (Apellis) | NGM621 (NGM Bio) | CB 2782-PEG |
|---|---|---|---|
| **Category** | PEGylated cyclic peptide | Antibody anti-C3 | **Protease** |
| **Targets C3** | Yes | Yes | Yes |
| **Dose Frequency** | Every 1-2 months | Every 1-2 months | **Every ~3 months*** |
| **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 (low)** |

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

# CB 4332: Enhanced Complement Factor I

## Next clinical candidate



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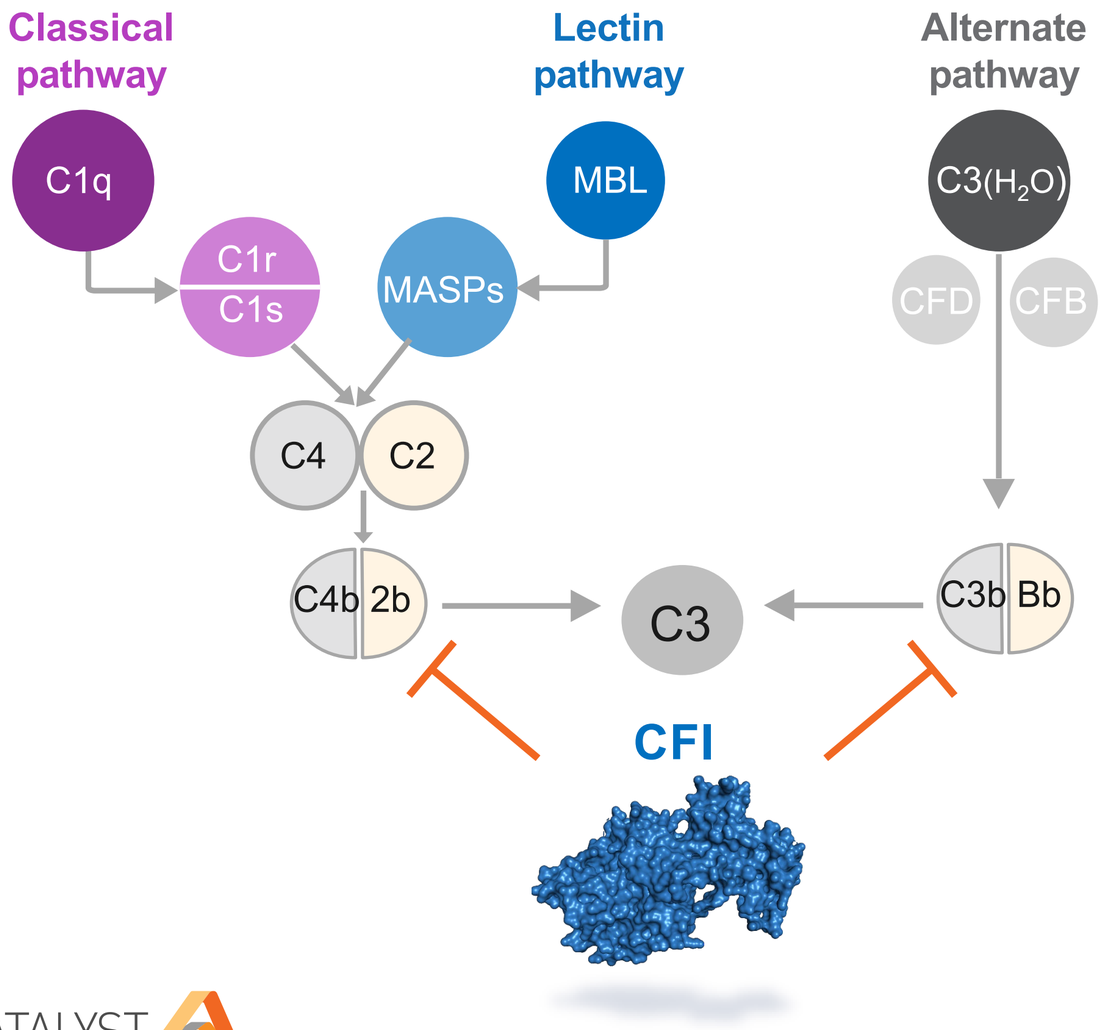
# Complement Factor I

## CFI is a key down-regulator of the complement cascade




### Applying the brakes to complement

- **CFI is a key regulator** of complement activation targeting **both C3b & C4b**
  - Classical & lectin pathway inhibitor
  - Alternative pathway inhibitor
- **CFI deficiency** triggers uncontrolled pathway activation
  - Secondary complement deficiency
  - Significant C3 depletion
  - Susceptibility to infections & increased autoimmune complex diseases

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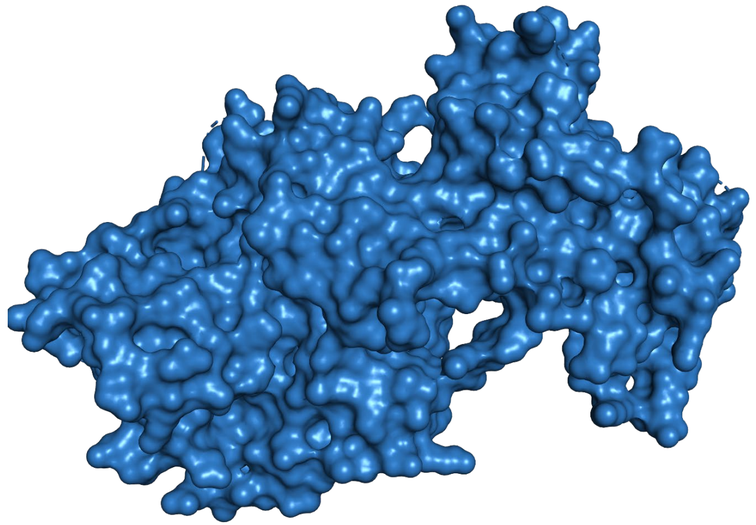
# CB 4332: SQ Enhanced Complement Factor I

## 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]

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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 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<br>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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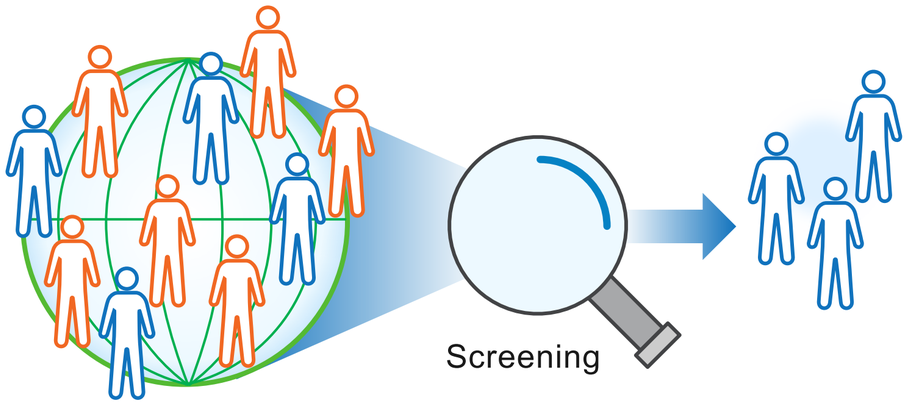
# Screening & natural history of disease studies

## ConFIrm & ConFIdence: preparing for Phase 1/2




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

The ConFIrm Study

The 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**

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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 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

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# Diseases with CFI mutations have tremendous potential

## US / EU5 market opportunity

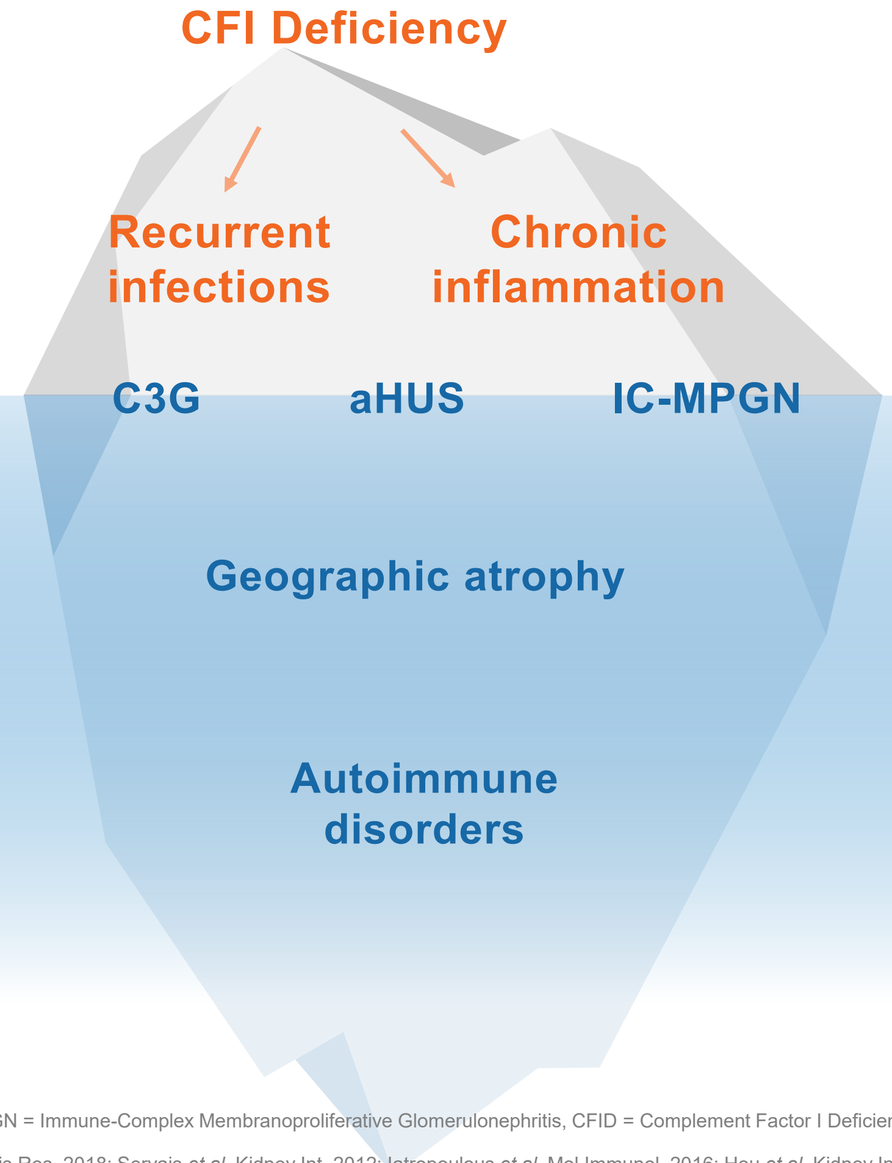

**CFI Deficiency**
First indication

**$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

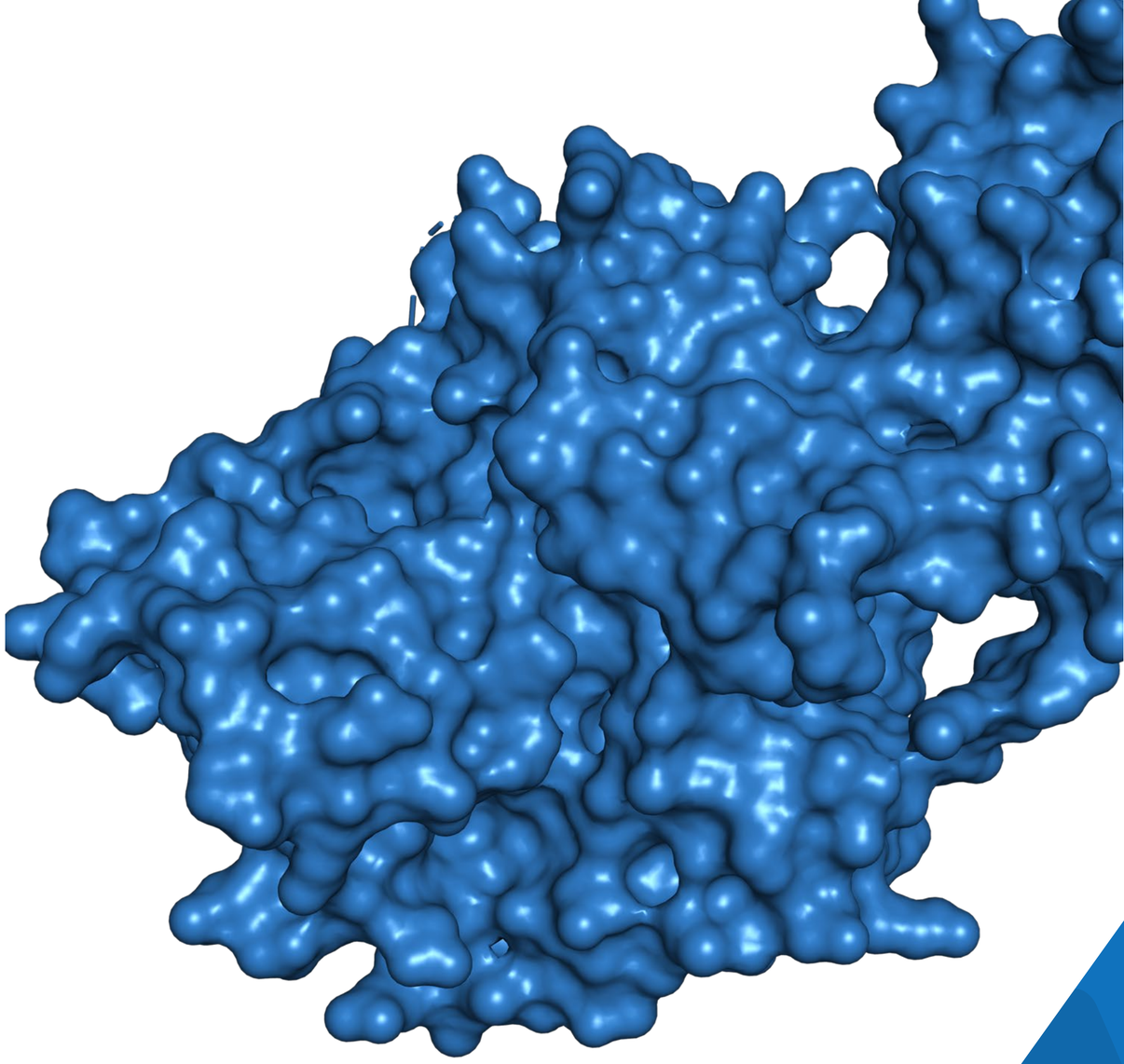



Note: aHUS = atypical Hemolytic Uremic Syndrome, C3G = Complement 3 Glomerulopathy, IC-MPGN = Immune-Complex Membranoproliferative Glomerulonephritis, CFID = Complement Factor I Deficiency

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

## C3b & C4b Degraders

# Expanding into classical complement disorders



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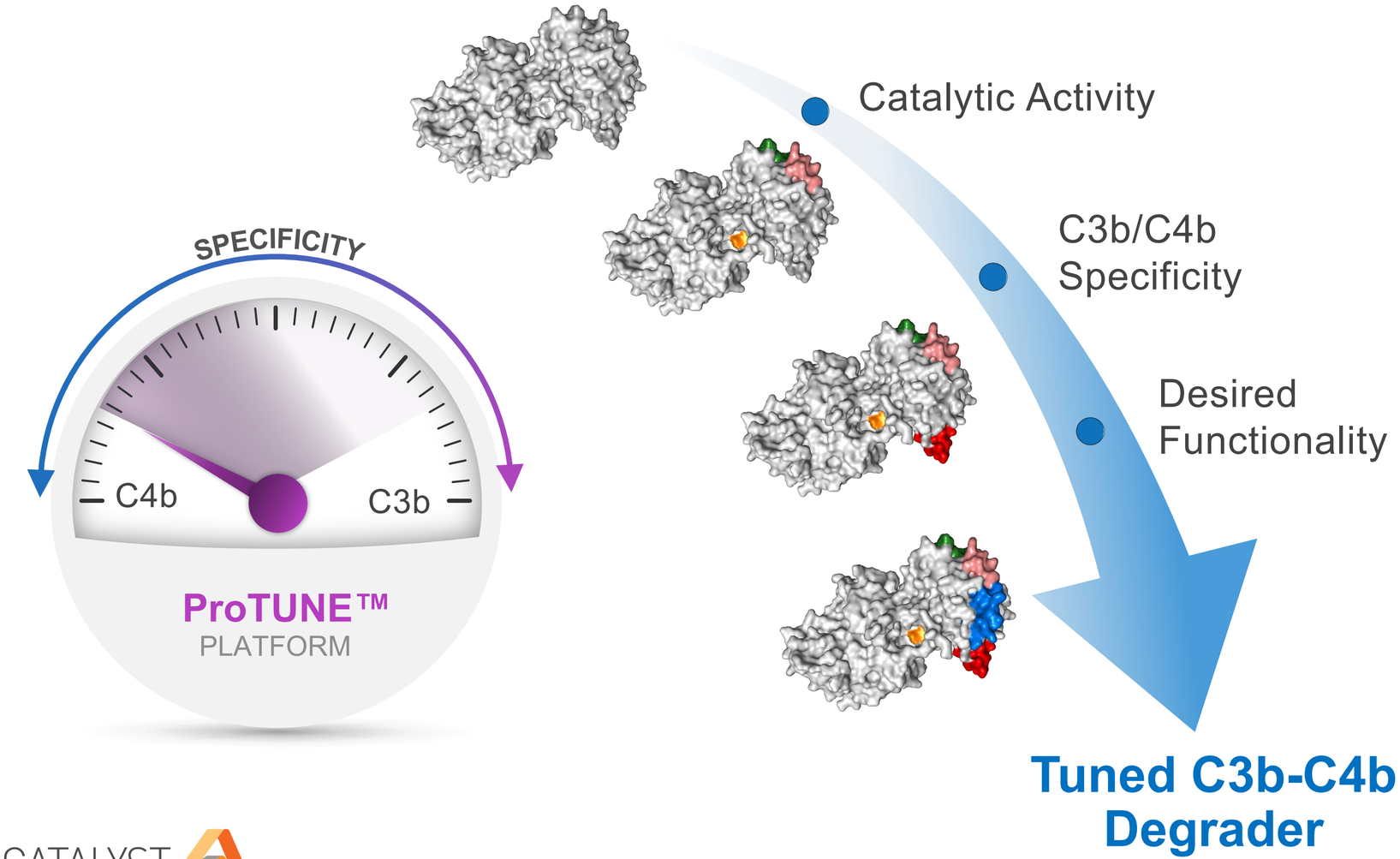
# Dialing catalytic power & specificity into CFI

## Using ProTUNE™ engineering platform to tune C3b & C4b degraders




### Precision CFI Therapeutics

- Tunable **potency** to control dysregulated complement
- Tunable **specificity** toward C3b & C4b to restore the **right** balance to complement

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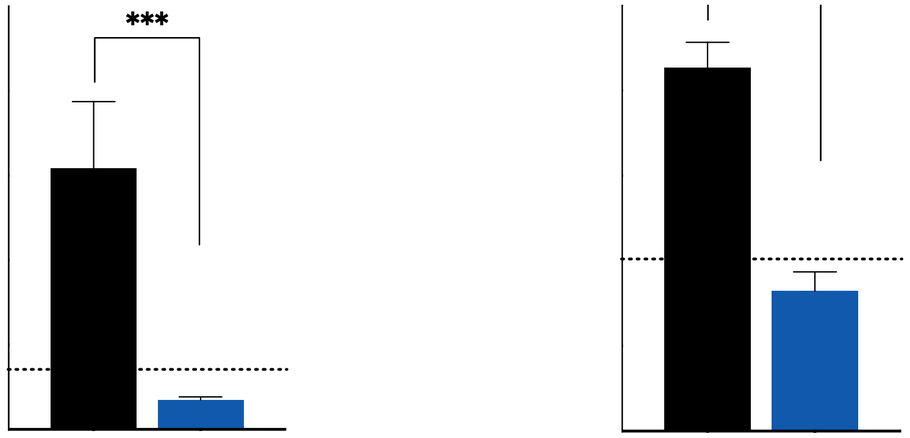
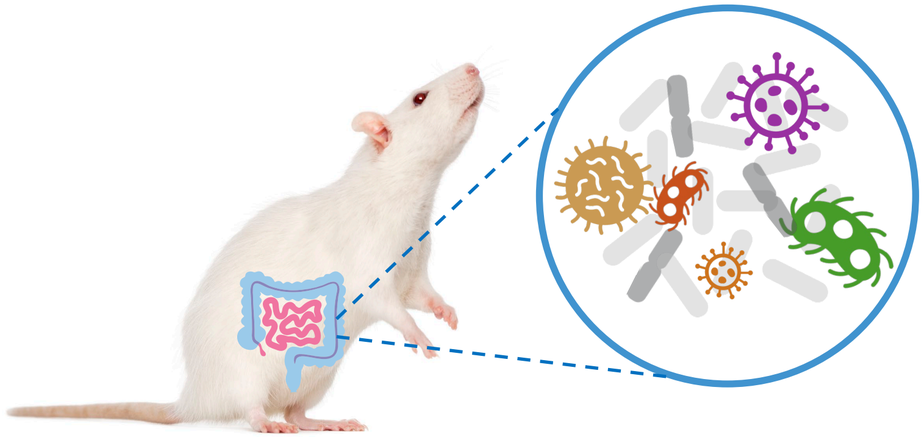
# C3b-C4b degraders significantly reduce inflammation *in vivo*

## Significantly decrease in inflammatory markers involved in IgA nephropathy

### Inflammatory markers in IgA nephropathy




### Rat model of complement-mediated inflammation

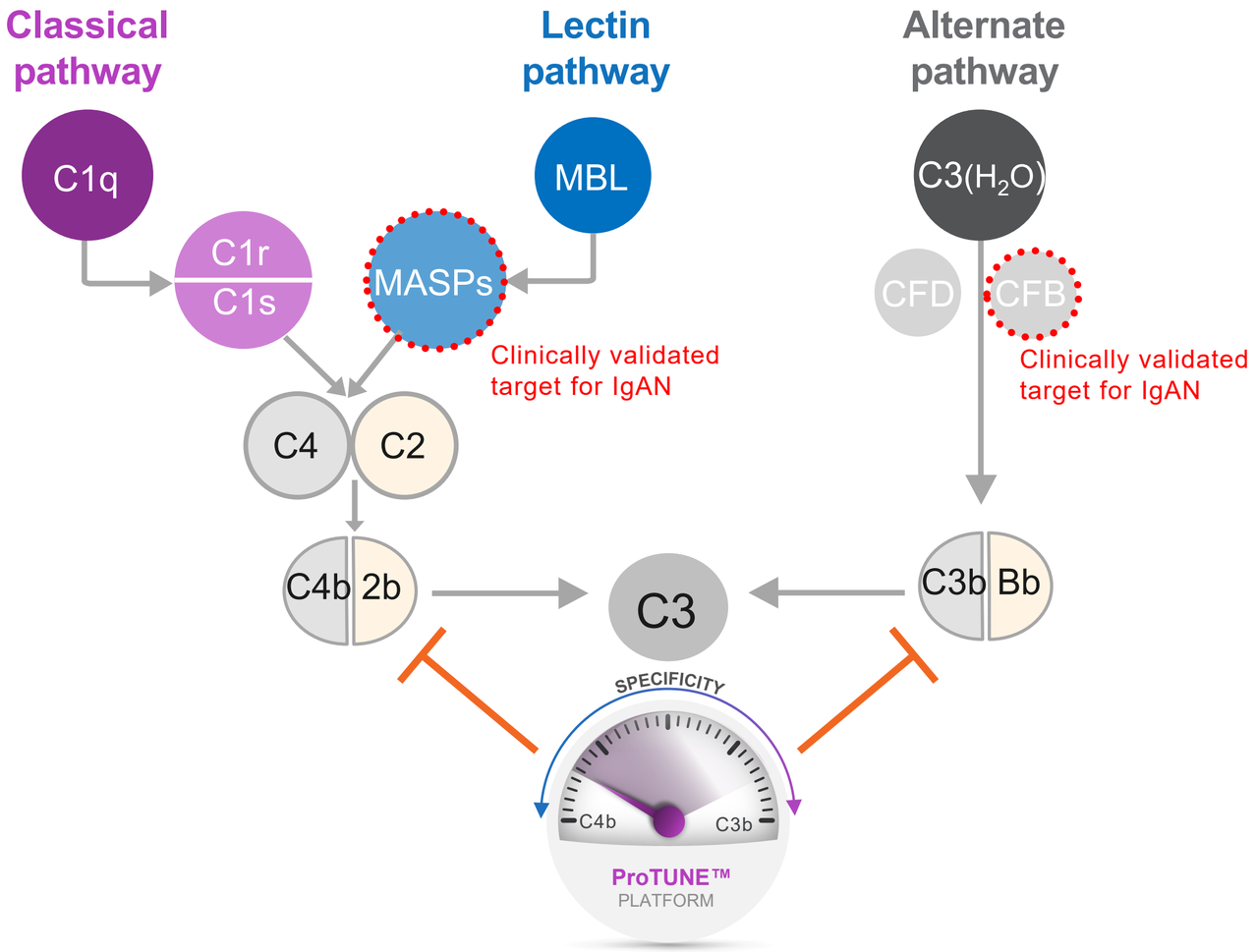


Reduction of **IFNγ & TNFα** involved in kidney damage & proteinuria in IgA nephropathy patients[1, 2]

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 and 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 & alternate pathways

## Rationale for IgA nephropathy

+ Both lectin & alternate 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

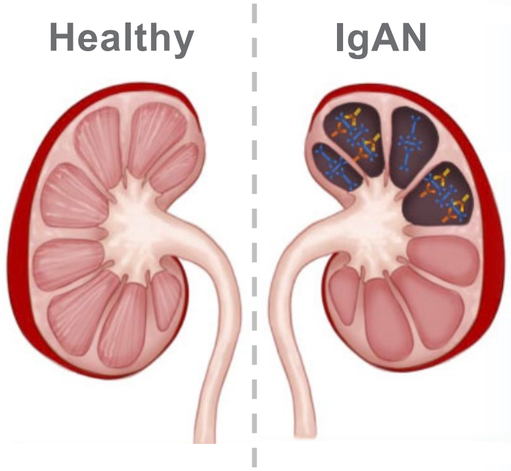
CATALYST BIOSCIENCES

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 pathogenesis

### High unmet need – current treatments only addressing symptoms




+ Most common form of glomerulopathies worldwide
+ Accumulation & deposition of IgA immune complexes leading to deterioration of renal function
+ **10%** patients with rapidly progressive glomerulonephritis
+ **40%** of IgAN patients develop end stage renal disease over 20 years & need dialysis/renal transplant in order to survive
+ Significant burden on healthcare resources with an estimated cost of **$49.2 billion** in 2020 in the US

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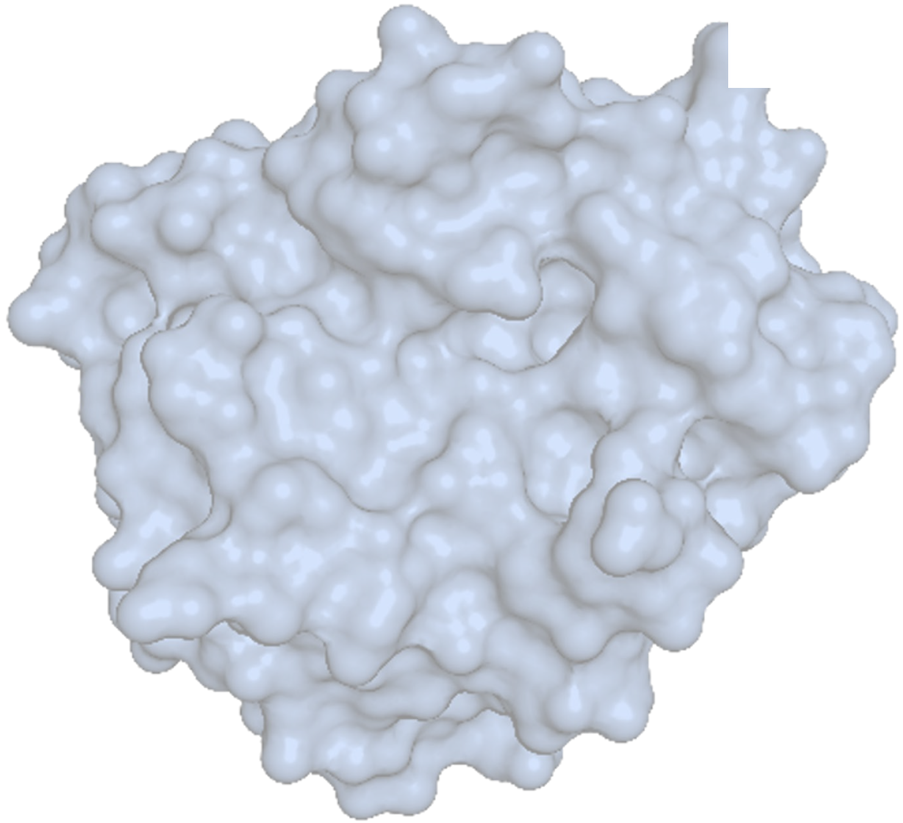
## C3a & C5a Degraders

# For inflammatory disorders



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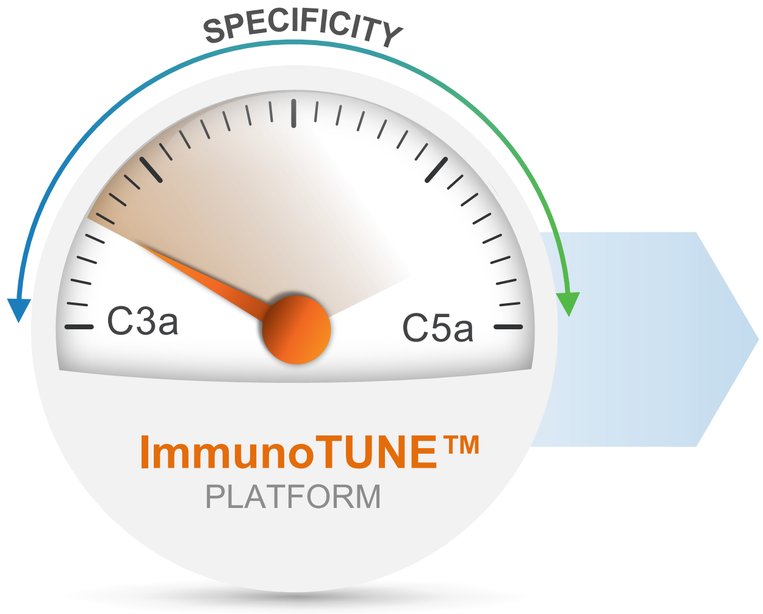
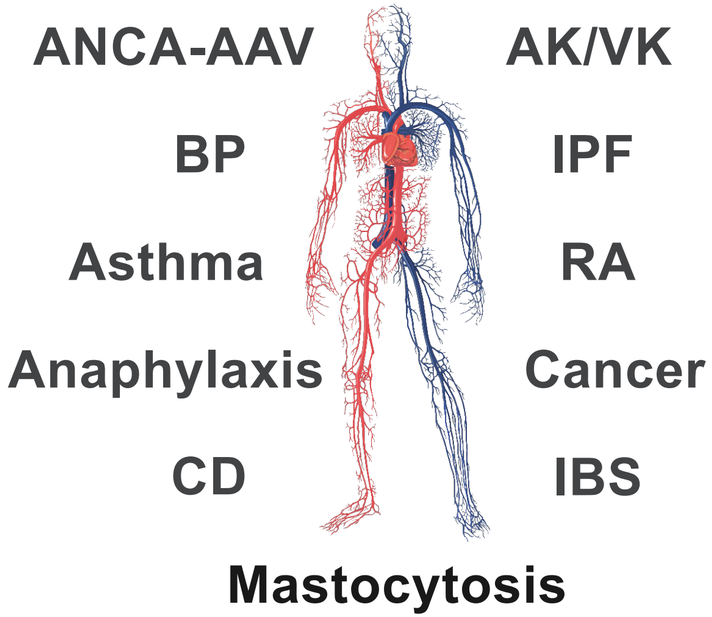
*Structural model based on PDB 2XRC*

# Dialing catalytic power & specificity to restore immunoregulation

## Using the ImmunoTUNE™ engineering platform to tune C3a & C5a degraders




### Mast Cell & Neutrophil Disorders




### Precision CFx Therapeutics

- Tunable **potency** for different level of immunomodulation
- Tunable **specificity** toward C3a & C5a to restore the **right** balance to complement
- 1 molecule can degrade 1000s of target molecules

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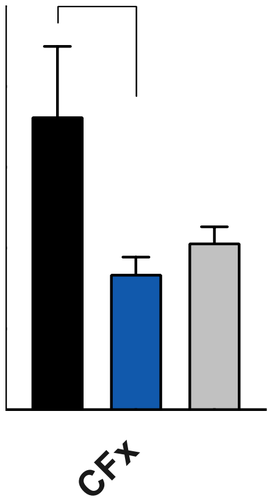
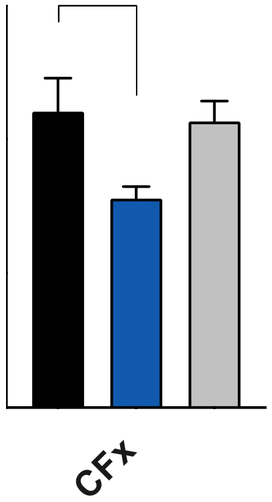
ANCA-AAV, anti-neutrophil cytoplasmic-antibody-associated vasculitis; IBS, inflammatory Bowel Syndrome; CD, Crohn's disease; RA, rheumatoid arthritis; BP, bullous pemphigoid; IPF, idiopathic pulmonary fibrosis; AK, Atopic keratoconjunctivitis; VK, vernal keratoconjunctivitis

# C3a-C5a degraders: Efficacy in an acute LPS-induced ARDS model

## CFx improves respiratory function & reduces cell infiltrates

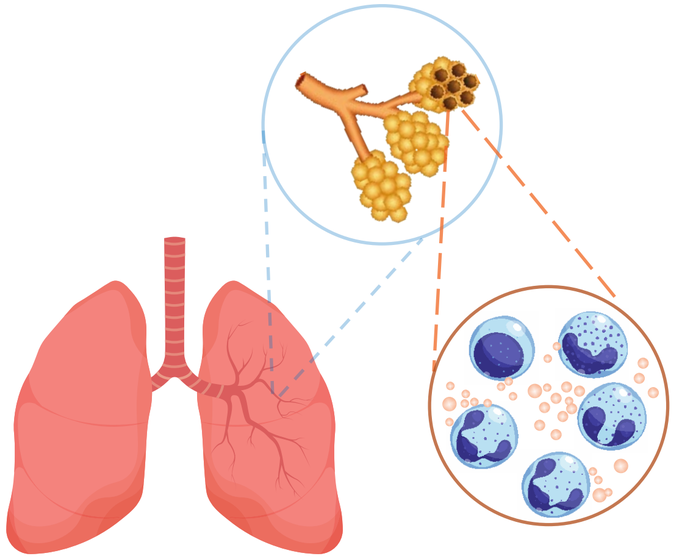
### Respiratory functions & cell infiltration at 24 h




*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

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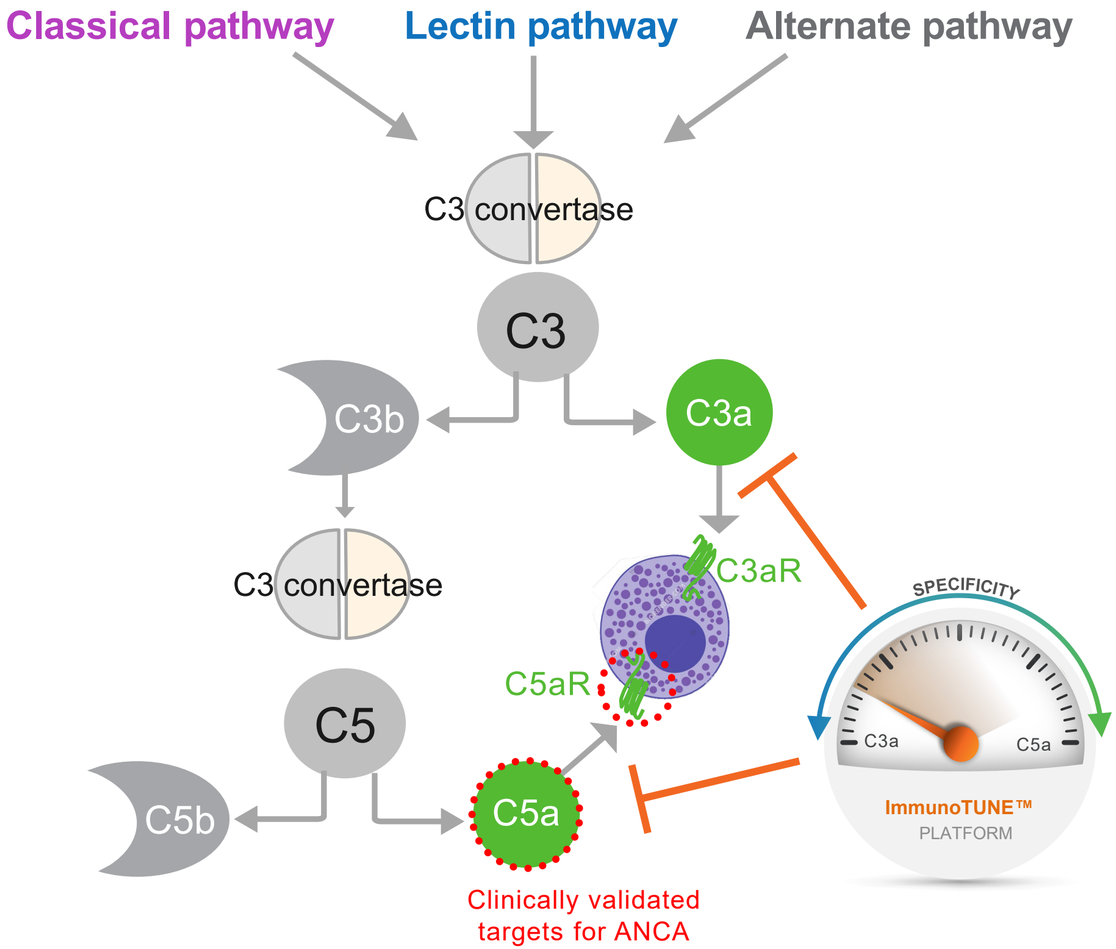
# C3a-C5a degraders: Potential for ANCA-AAV patients

## Dual targeting of both C3a & C5a with one protease medicine




## Differentiation

+ Degrade activation products 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

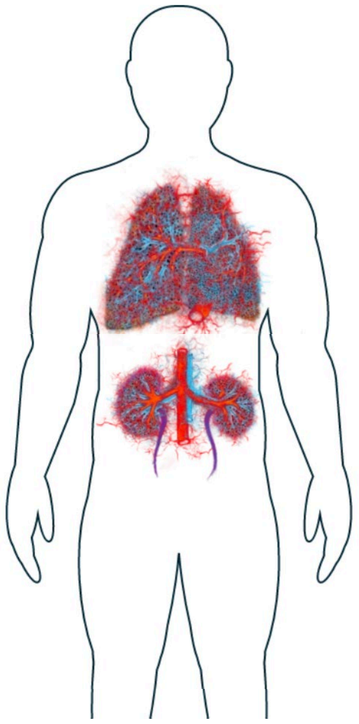
CATALYST BIOSCIENCES

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 pathogenesis

### High unmet need – current treatments only addressing symptoms



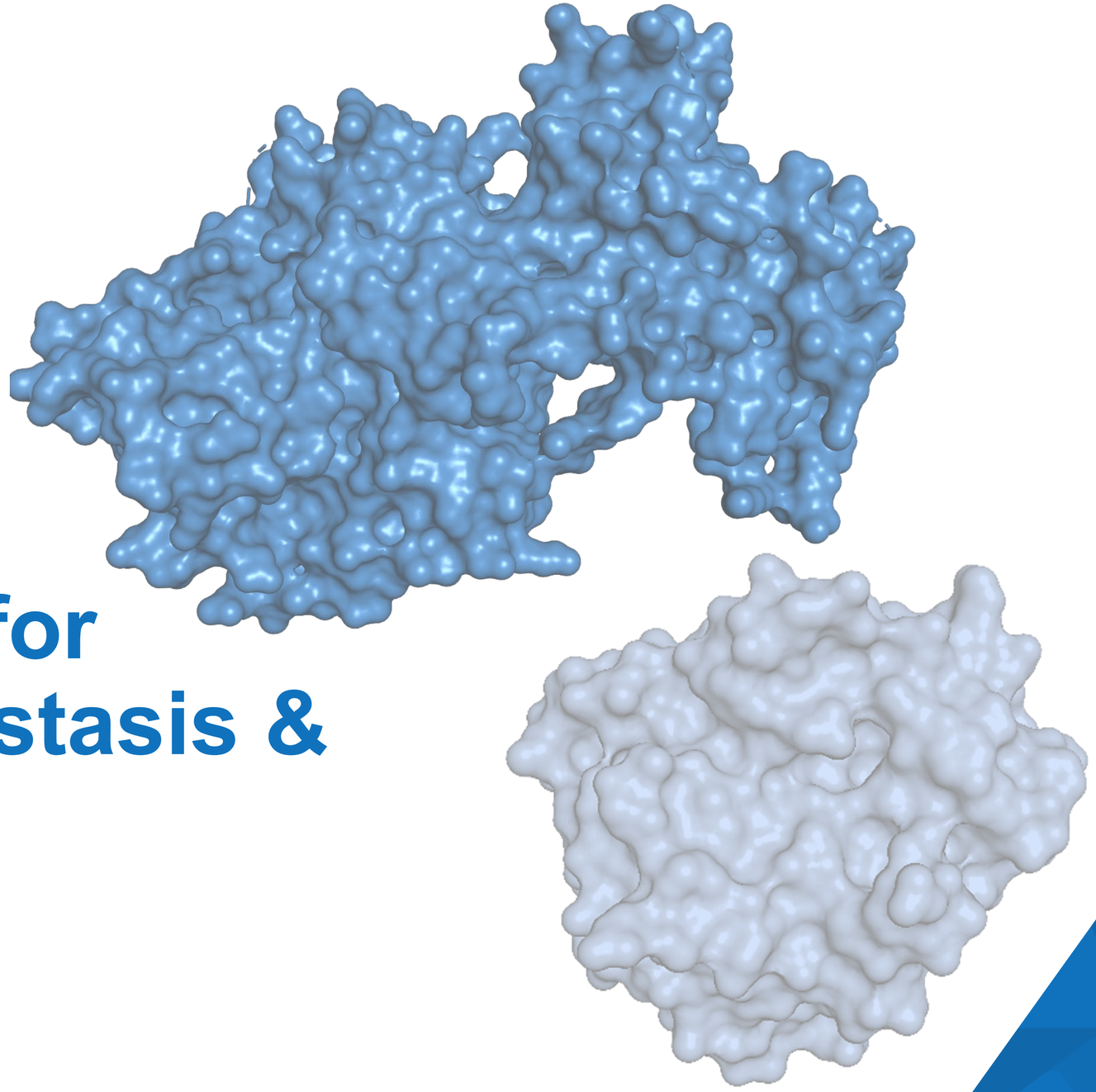
+ Autoimmune disorder characterized by inflammation & destruction of small blood vessels
+ Clinical signs vary & affect several organs with frequent involvement of upper respiratory track & kidneys
+ Severe pain due to neuropathy, pulmonary hemorrhages, failure of kidneys
+ **10-15%** of patients die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

CATALYST BIOSCIENCES

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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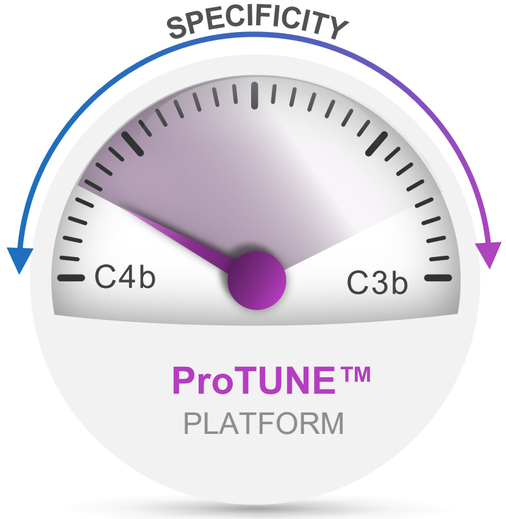
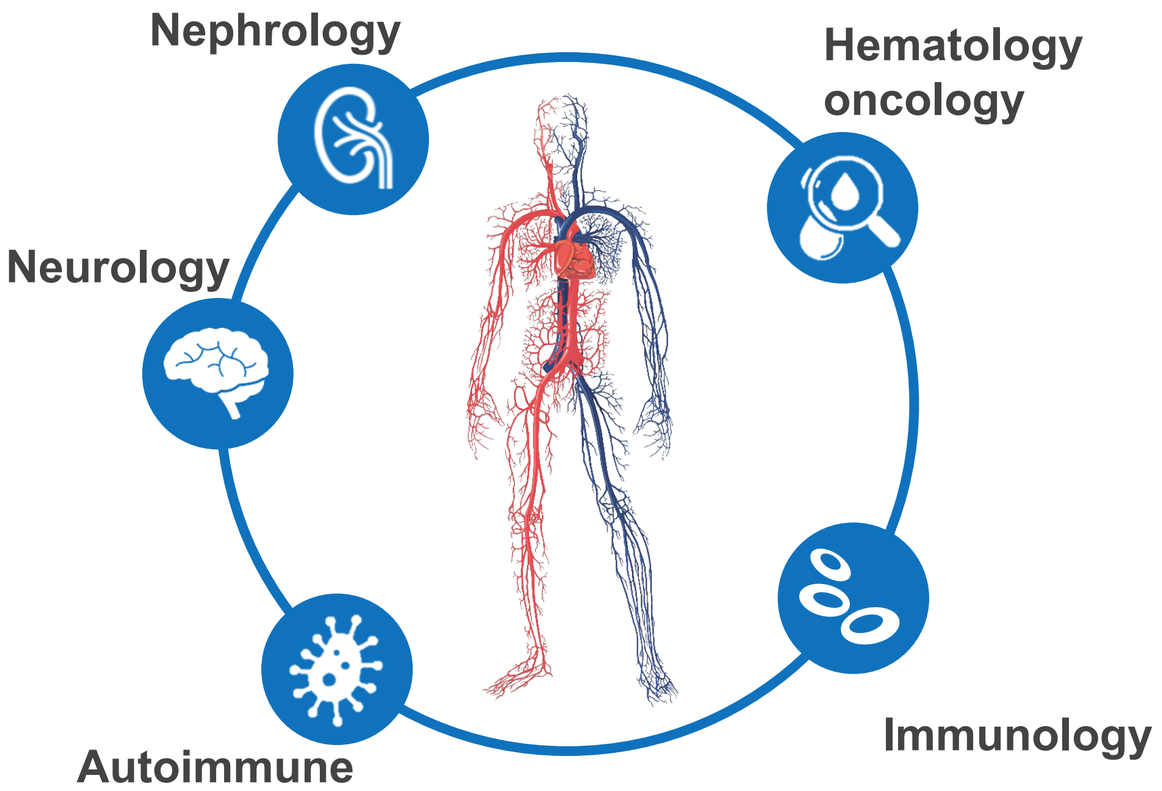
# Our protease platforms are tailored to specific indications

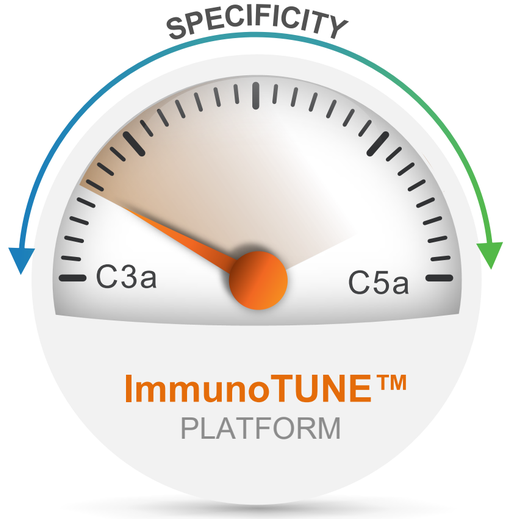
## Tuning functionality to restore complement homeostasis & immunoregulation

**ProTUNE™**
**C3b-C4b**




**ImmunoTUNE™**
**C3a-C5a**

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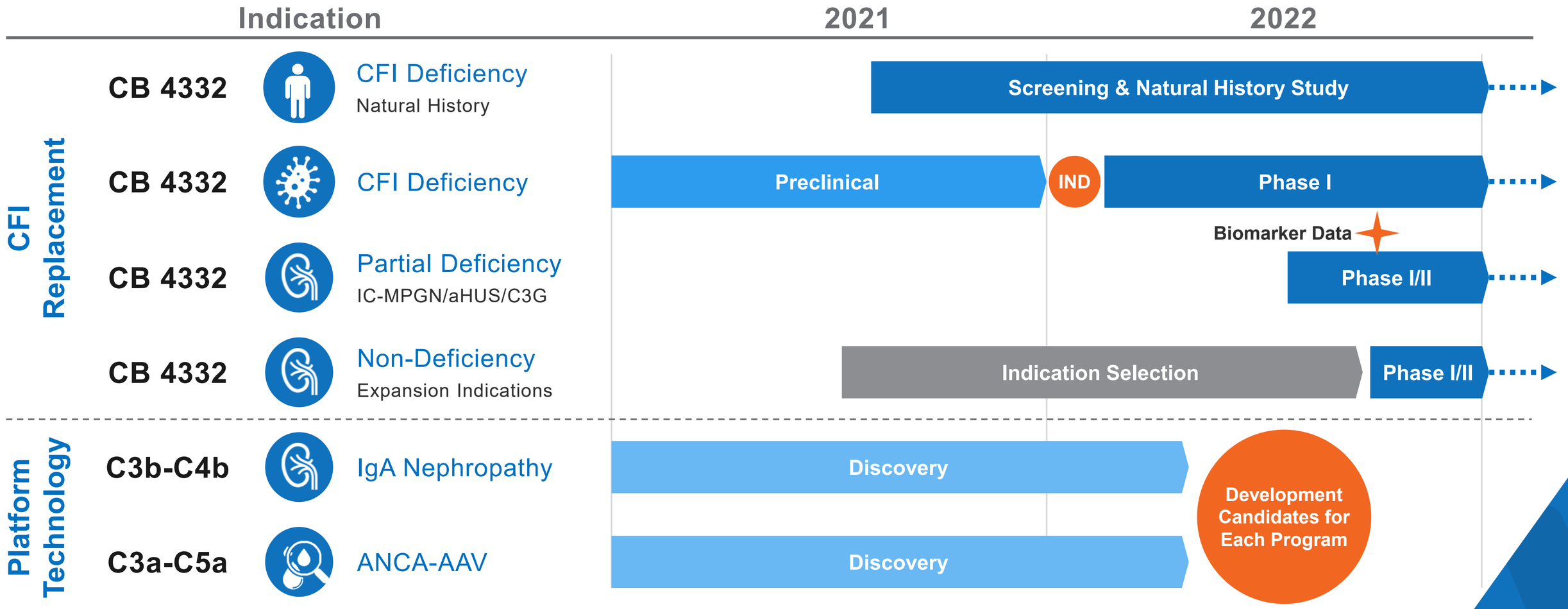
# CB 4332 spearheads a deep pipeline in complement

## Next development candidate in 2022

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# Milestones

**Clinton Musil | CFO**

**Closing Remarks, Q&A**

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# Milestones: Catalyst Biosciences complement programs

| | |
|---|---|
| **Observational trial for CB 4332** | Enrollment to start mid-2021 |
| **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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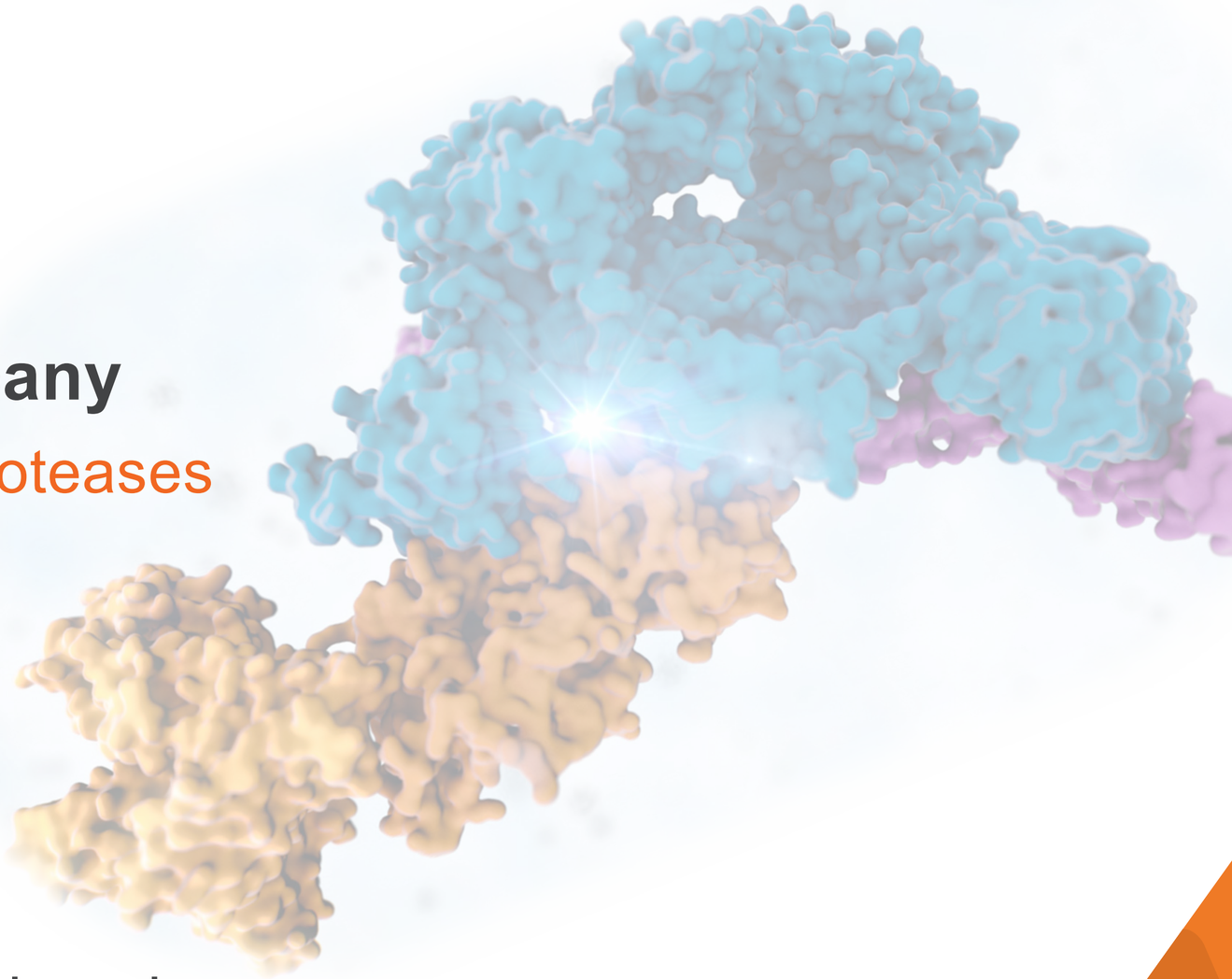

## The Protease Medicines Company

Harnessing the catalytic power of proteases

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