CATALYST BIOSCIENCES

Corporate Overview

12 January 2022

CATALYST BIOSCIENCES

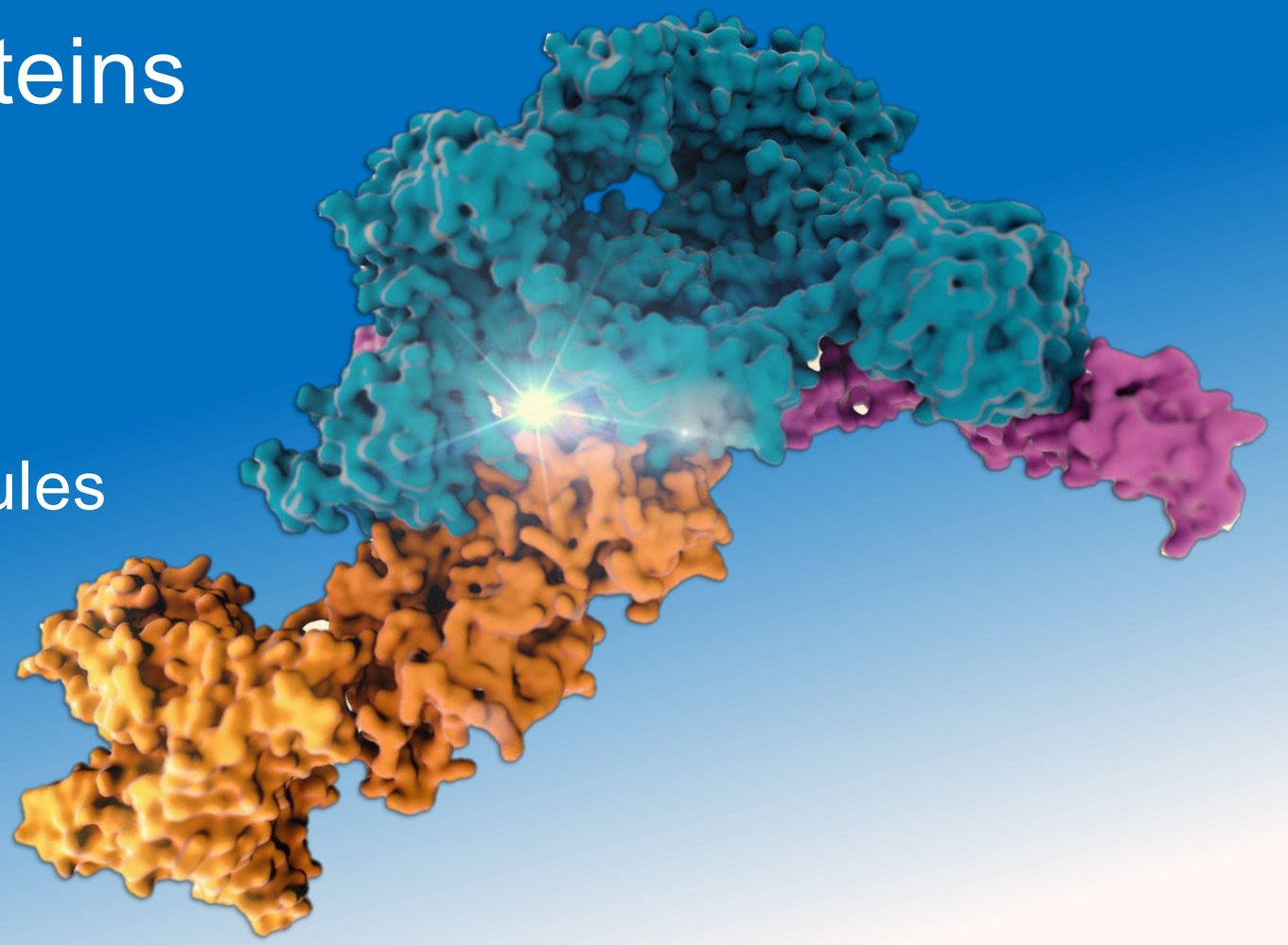
Forward looking statements

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. This press release contains forward-looking statements that involve substantial risks and uncertainties. Forward-looking statements include, without limitation, our plans for CB 4332 and the rest of our complement programs, our plans to continue to support Biogen in the development of CB 2782-PEG, the statement that complement has broad potential, can be combined with conventional therapies and will open opportunities in multiple disease settings, as well as statements about the benefits of our protease engineering platform.

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 clinical trials and preclinical studies may be delayed as a result of COVID-19, competitive products, and other factors, that Biogen could terminate our agreement for the development of CB 2782-PEG, that our complement degraders are not yet in human clinical trials and will require additional manufacturing validation and preclinical testing before entering human clinical trials, that we may need to raise additional capital, and other risks described in the "Risk Factors" section of our Annual Report on Form 10-K filed with the Securities and Exchange Commission (the "SEC") on March 4, 2021, the Quarterly Report on Form 10-Q filed with the SEC on November 12, 2021, and in other filings filed from time to time with the SEC. We do not assume any obligation to update any forward-looking statements, except as required by law.

Modulating Biological Systems with Nature's Regulatory Proteins

- Proteases are nature's key regulatory proteins
- Innovative engineered molecules to degrade or activate therapeutic targets
- Applicable across multiple disease areas



We harness the regulatory power of proteases

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Pipeline

Complement

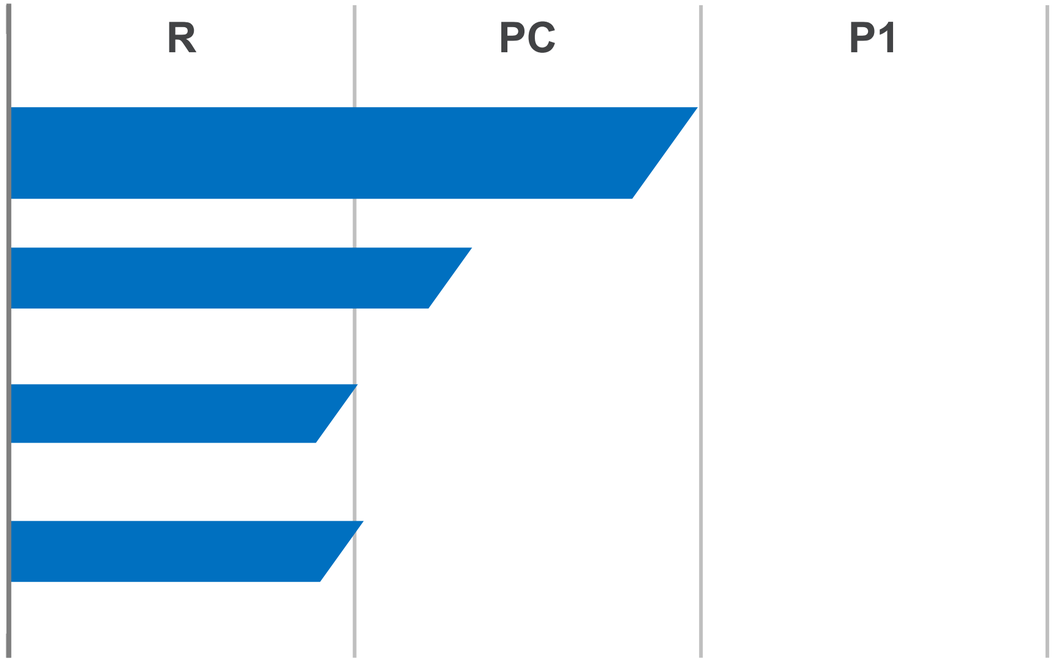
SQ CB 4332 Enhanced CFI

IVT CB 2782-PEG
C3 degrader for Dry AMD

Biogen

C3b/C4b degraders

C3a/C5a degraders



Partnering opportunities

Hemostasis

SQ Marzeptacog alfa (FVIIa) "MarzAA"
- Hemophilia A or B with inhibitors
- FVIID/Glanzmann/Hemlibra

SQ Dalcinonacog alfa (FIX) "DalcA"
- Hemophilia B

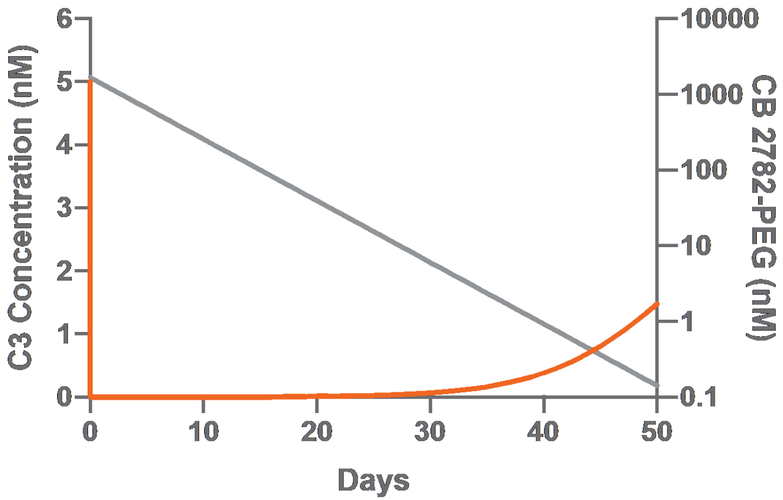
CB 2679d-GT
- Hemophilia B FIX Gene Therapy

Catalyst's protease platform in complement

Validated across three programs

CB 2782-PEG Biogen

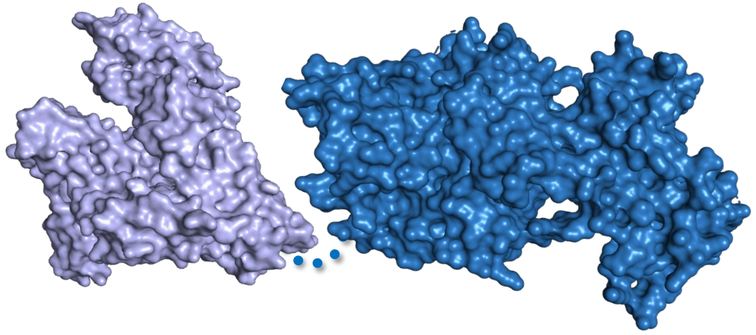
Best-in-class profile for dry AMD
Extended pharmacodynamics



Novel C3-degrader for dry AMD

CB 4332 PK extended CFI

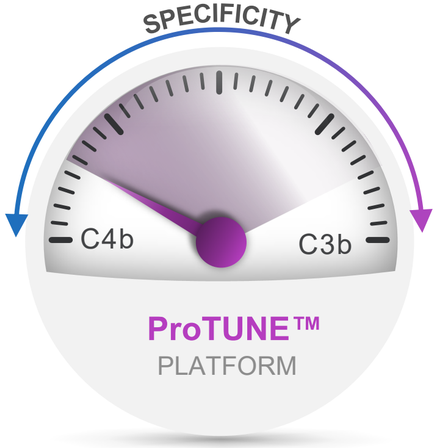
Restoring balance to complement where CFI activity is insufficient



Engineered CFI entering the clinic in 2022

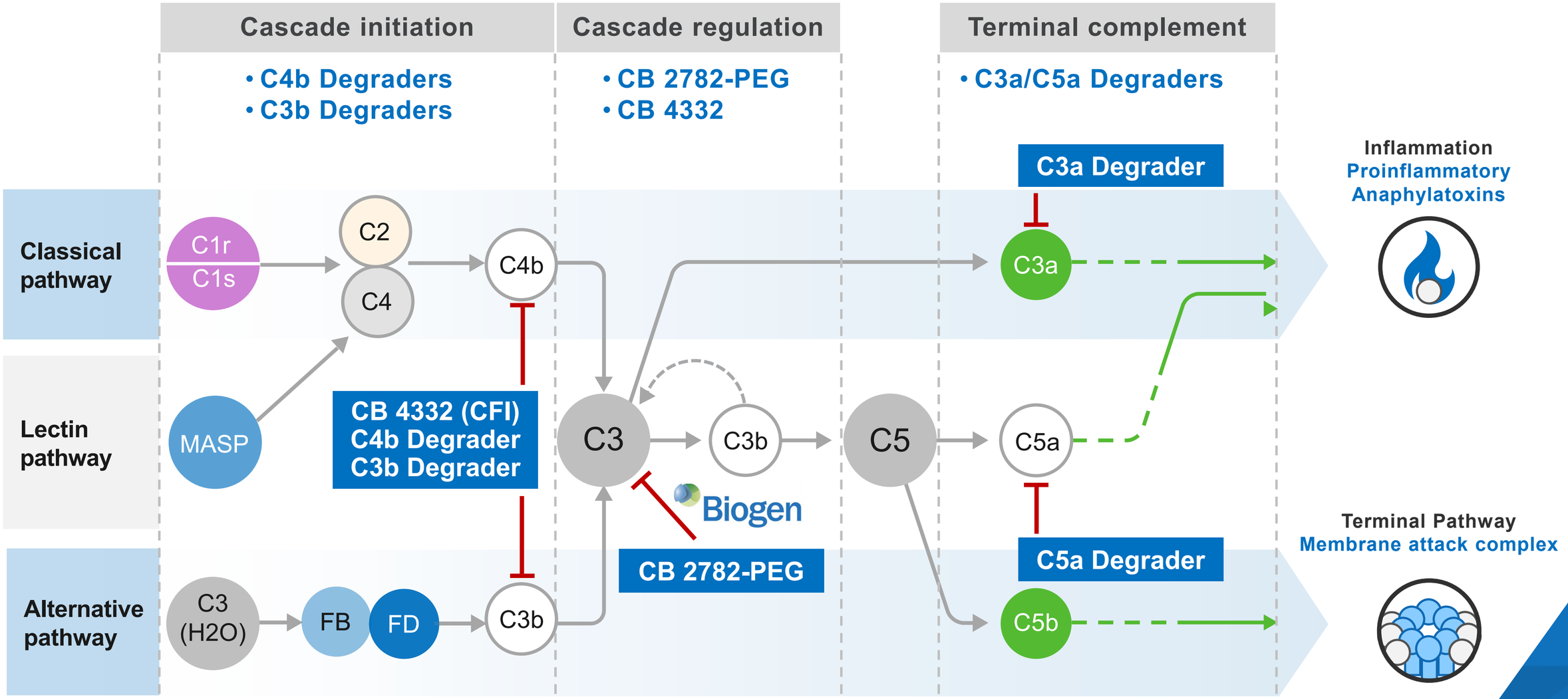
Engineered proteases

Protease platforms tailored to improve C3b & C4b regulation



C3b/C4b degrader platform delivering candidates in 2022

Unique targeted approach to complement regulation

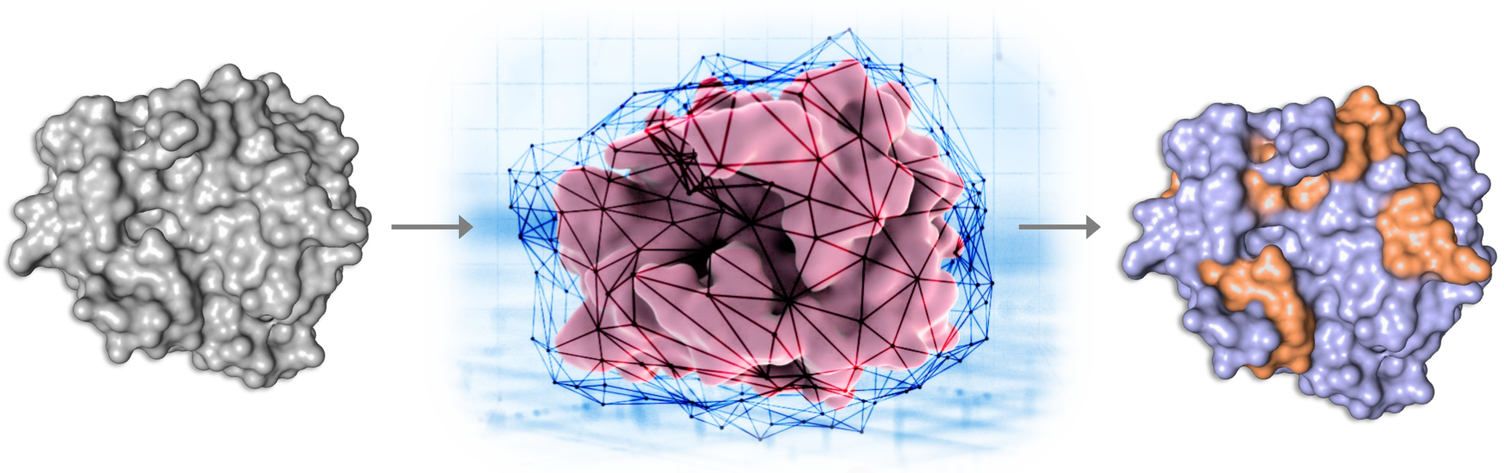


Catalyst protease and protein degrader discovery platform

Distinct expertise enables design of optimal therapeutic candidates

Protease Discovery Platform

Core protease → *Protease engineering* → **Drug candidate**



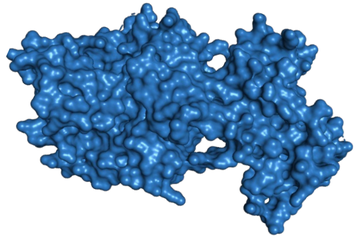
Therapeutic advantages of protein degraders

- Controlled target engagement
- Fast elimination of difficult targets
- Tunable for high potency & affinity
- Modulate instead of inactivating
- Can be combined with conventional therapies

Nature’s way to regulate complement

A platform based on the natural braking mechanisms of complement

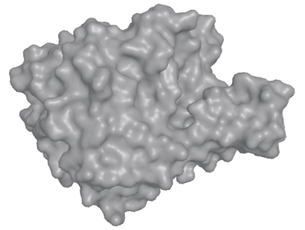
Complement Factor I



Nature’s Regulator of Complement at C3b/C4b

- Rebalances complement using the natural brakes (CFI)
- Multiple diseases driven by C3b/C4b deposition & immune activation
- Differentiated mechanisms to regulate at or around C3 & C5
- Safely regulate complement without broad immunosuppression
- Uses the natural regulatory protein to modulate the complement system

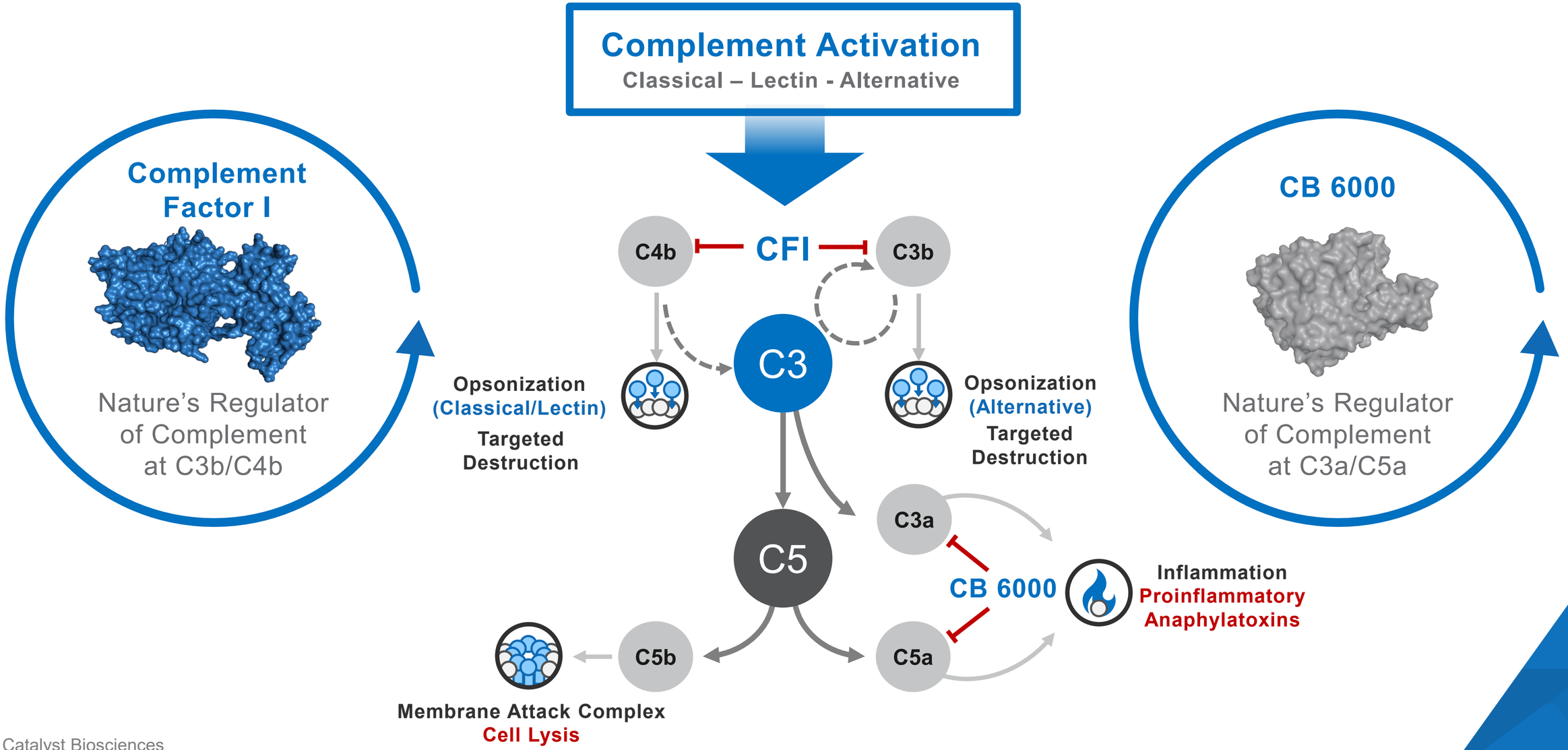
CB 6000



Nature’s Regulator of Complement at C3a/C5a

Nature's way to regulate complement

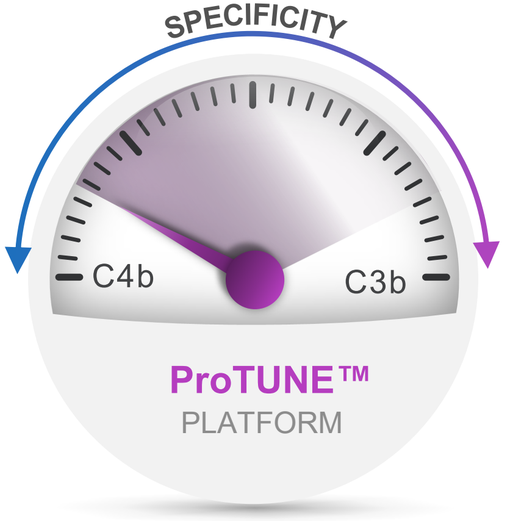
A platform based on the natural braking mechanisms of complement

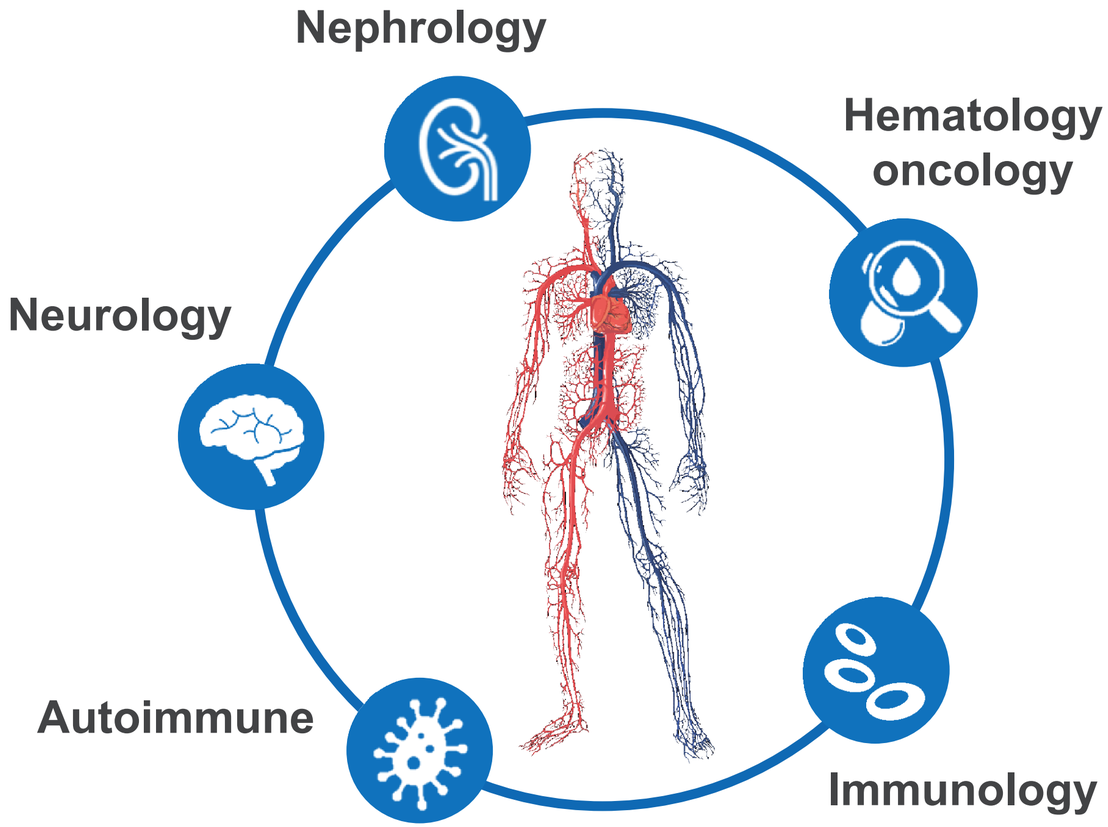


Our protease platforms are tailored to specific indications

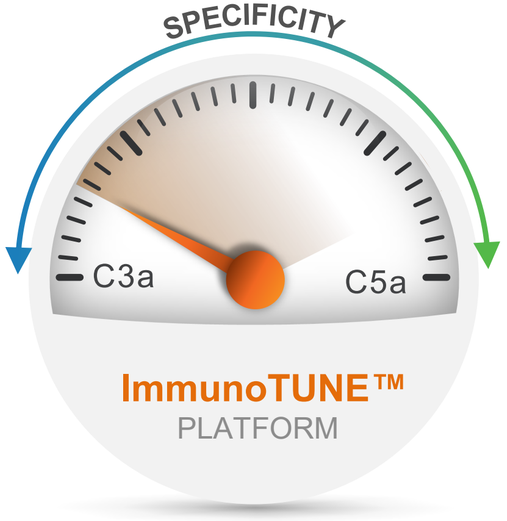
Tuning functionality to restore complement homeostasis & immunoregulation

ProTUNE™ C3b/C4b





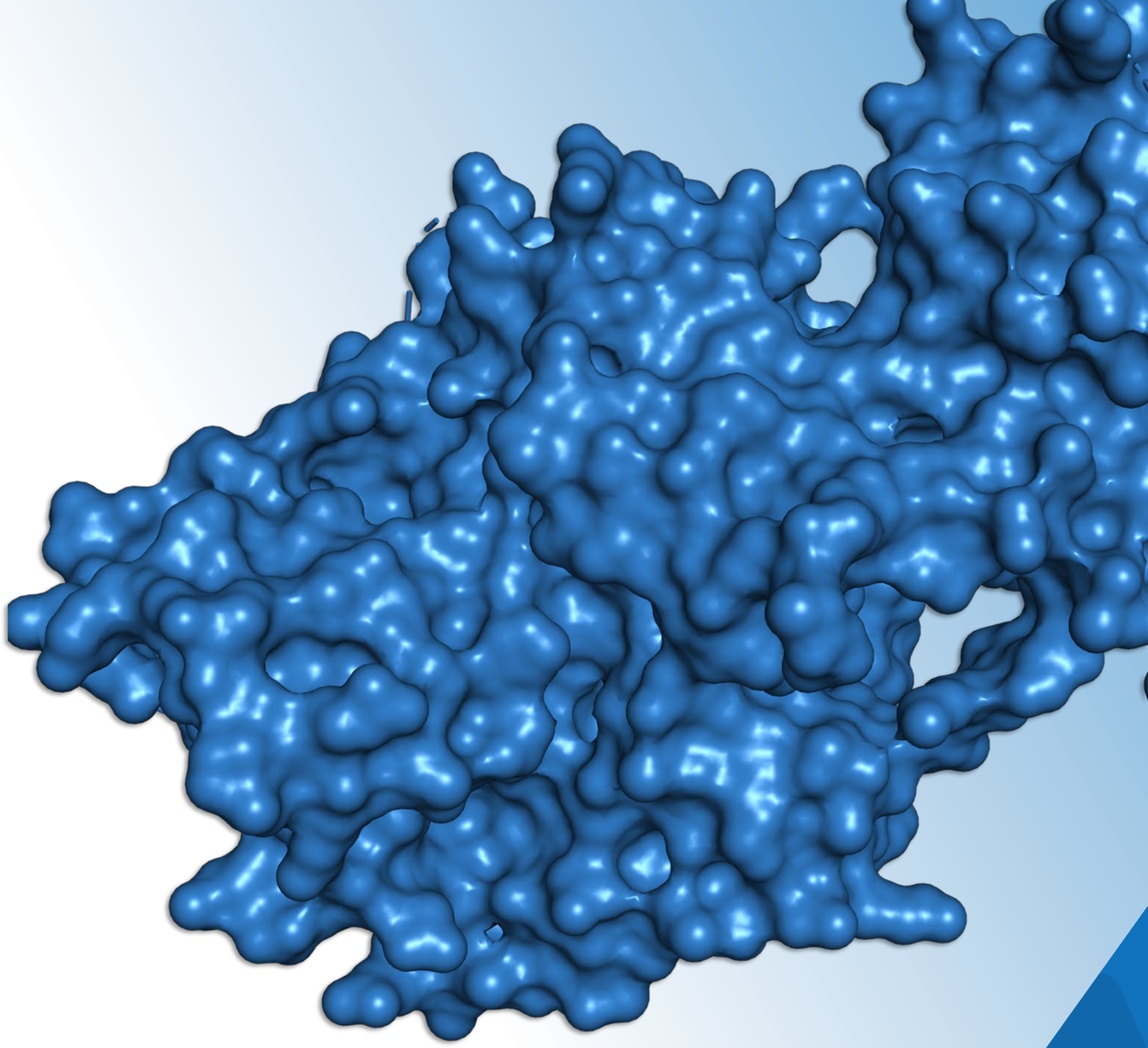
ImmunoTUNE™ C3a/C5a



Specific inhibition of complement components at different sites of the complement cascade allows a personalized approach to treating complement disorders

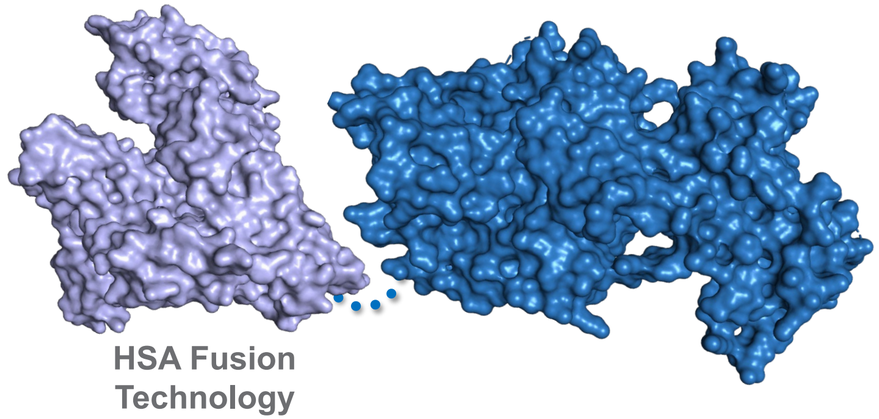
CB 4332

Half-Life Extended Complement Factor I to rebalance the complement system



CB 4332: Extended half-life Complement Factor I

Development candidate to restore regulation



+ **Engineered for an extended half-life**
  + Potential for once weekly SQ therapy
+ ***In vitro & ex vivo* activity comparable to native CFI**
  + Classical & alternative pathway regulation
+ **High yield production process**
+ **Safe GLP toxicology with a high dosing window**
+ **Entering the clinic in 2022**

Rationale & unmet need

+ **Rebalance the complement system** in patients with insufficient complement regulation
+ **No specific therapies exist** to correct complement dysregulation using natural proteases
+ Potentially targets a 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.

Systemic & ocular CFI to rebalance the complement system

CB 4332 has potential to address a breadth of mechanistically related diseases

CFI Replacement

Complete CFI Deficiency

Life-threatening recurrent infections & immune disorders

Partial CFI Deficiency

Kidney disorders

Ocular CFI

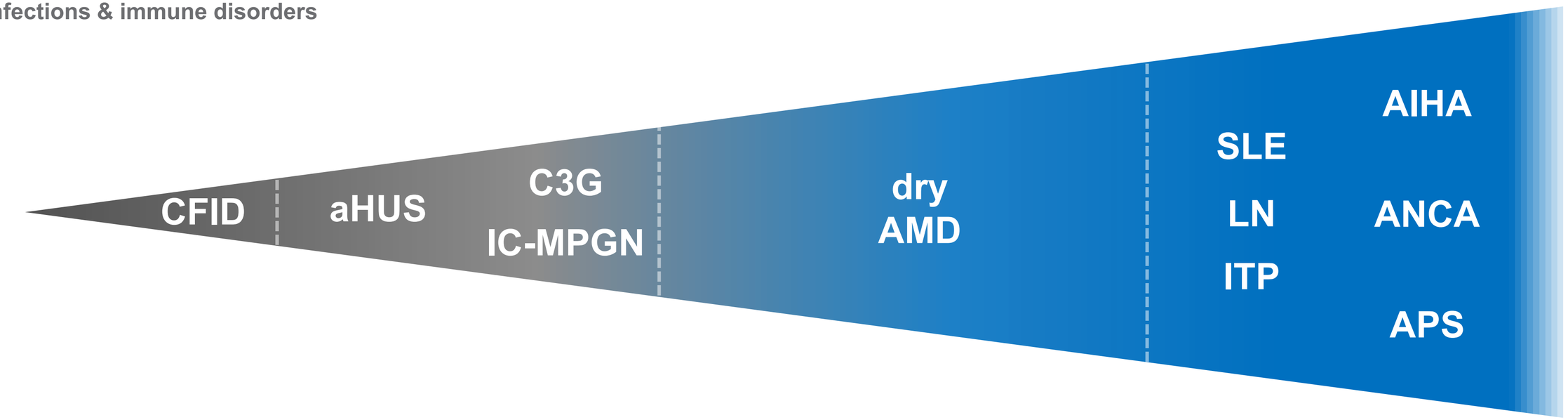
Re-Balancing Complement

Disorders of the eye

CFI Intervention

Re-Balancing Complement

Disorders of Complement

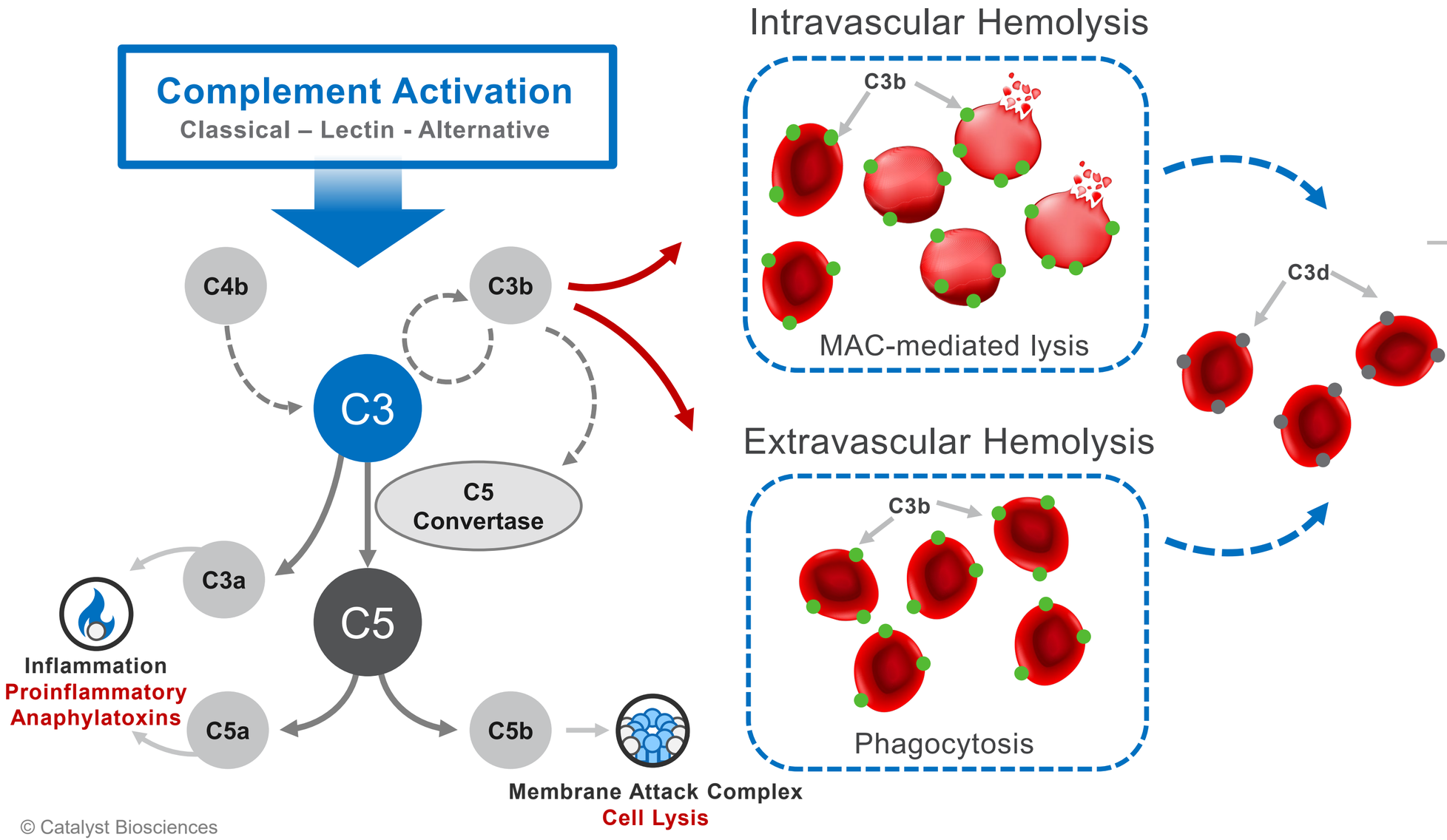


*Patient population estimate *does not include age-related macular degeneration US population with rare CFI variants*

AMD: Age-Related Macular Degeneration, aHUS: atypical Hemolytic Uremic Syndrome, C3G: C3 Glomerulonephropathy, SLE: Systemic Lupus Erythematosus, LN: Lupus Nephritis, AIHA: Autoimmune Hemolytic Anemia, ANCA: ANCA-associated Vasculitis, ITP: Immune Thrombocytopenia, HAE: Hereditary Angioedema, APS: Antiphospholipid Antibody Syndrome

CB 4332 may target diseases of excessive C3b and C4b deposition

Deposition of C4b & C3b in AIHA lead to hemolysis & cellular destruction



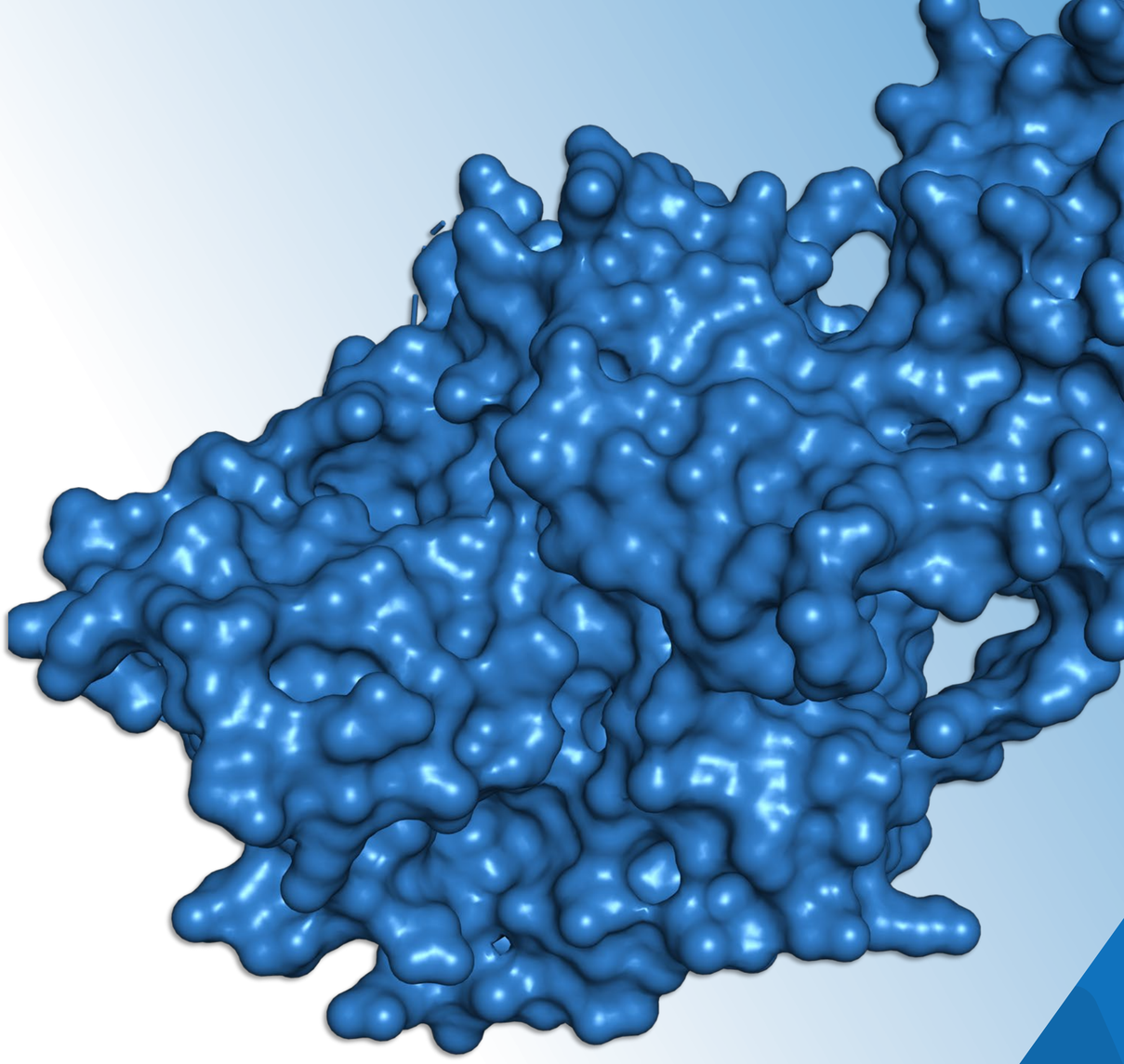
CB 4332 protects against C3b driven hemolysis

Degradation of C3b to C3d leads to cellular survival

CFI is the natural inactivator & degrader of C3b to C3d

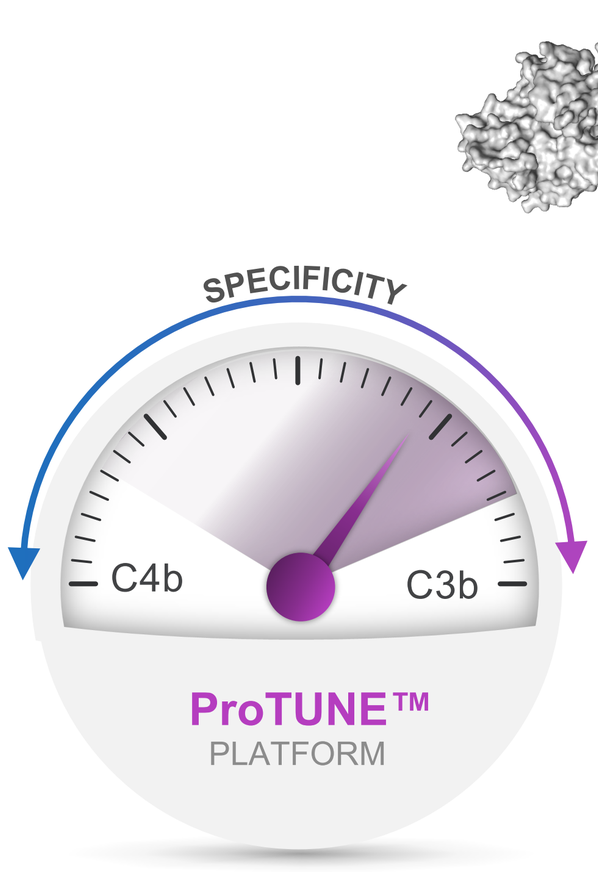
C3b & C4b Degraders

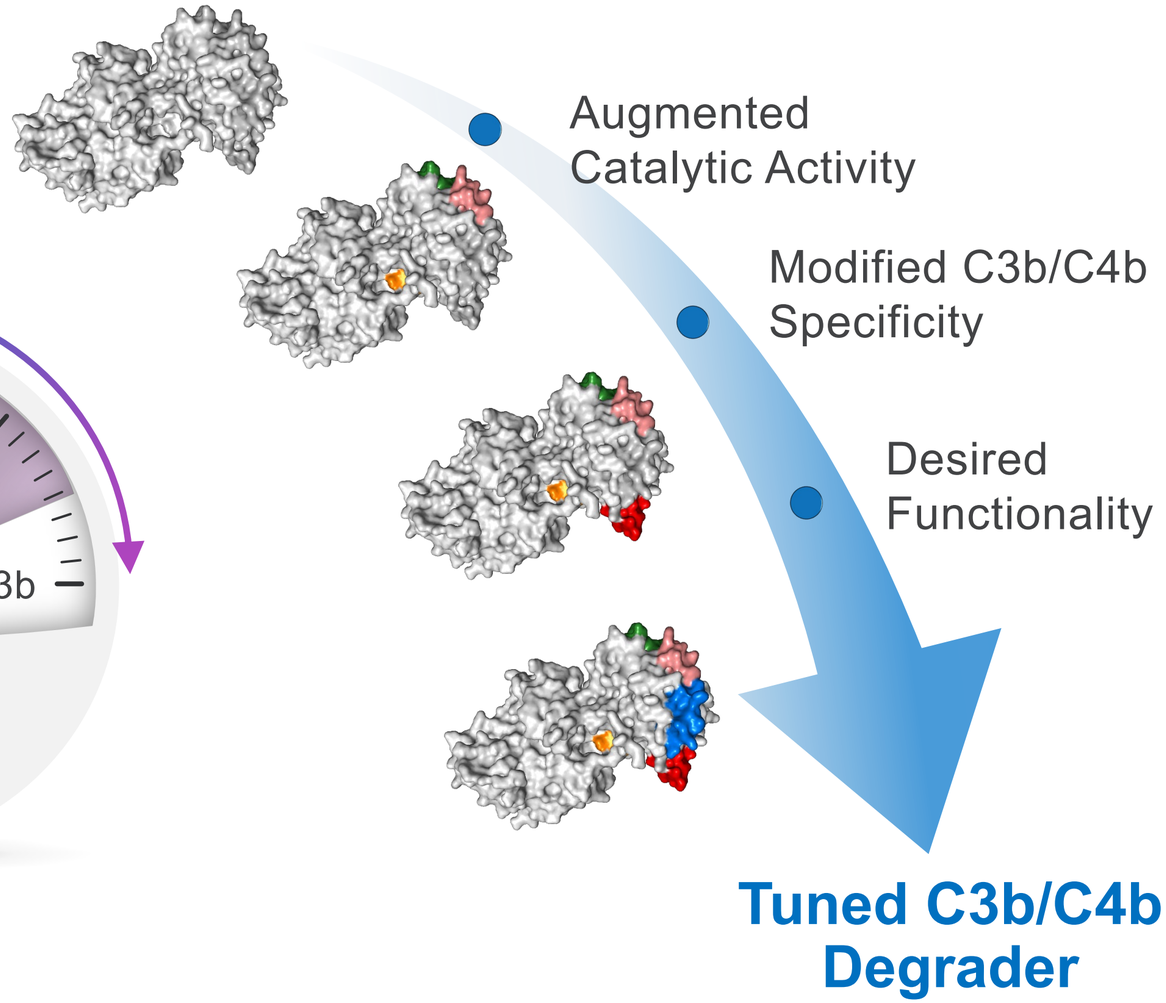
Broad applications in complement-mediated disorders



Improved catalytic power & specificity for CFI variants

ProTUNE™ platform has been used to generate specific C3b/C4b degraders



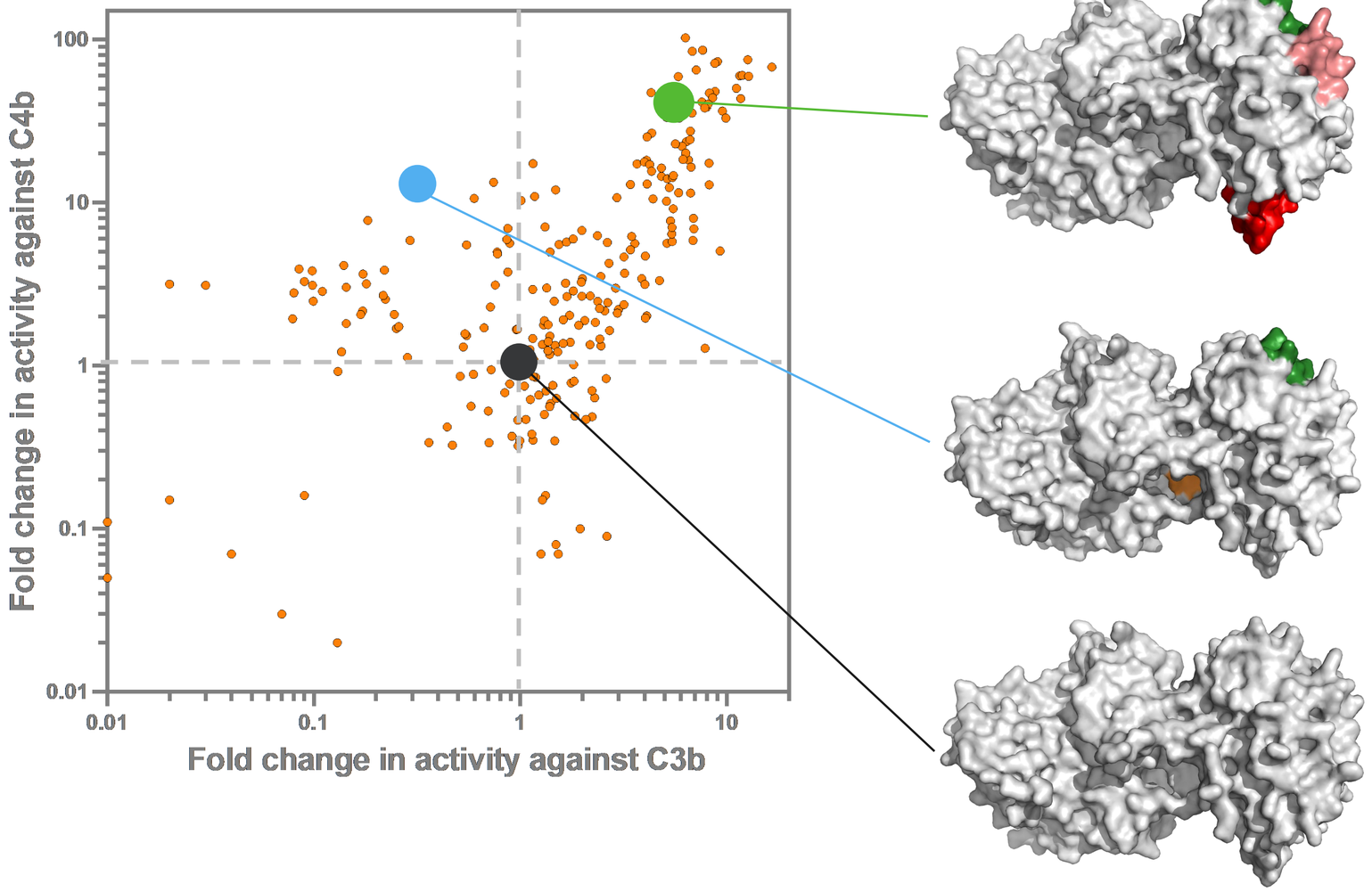


Precision CFI Therapeutics

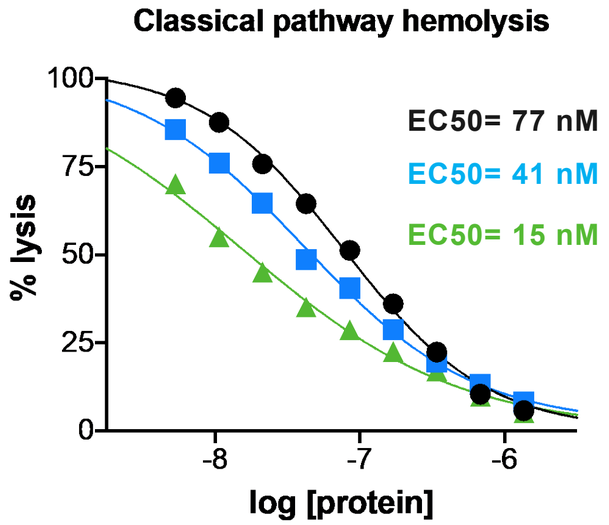
- **Tunable potency** to control dysregulated complement
- **Tunable specificity** toward C3b & C4b to restore balance to the complement cascade
- **Preserves innate immune response** by sparing cascade leading to MAC formation

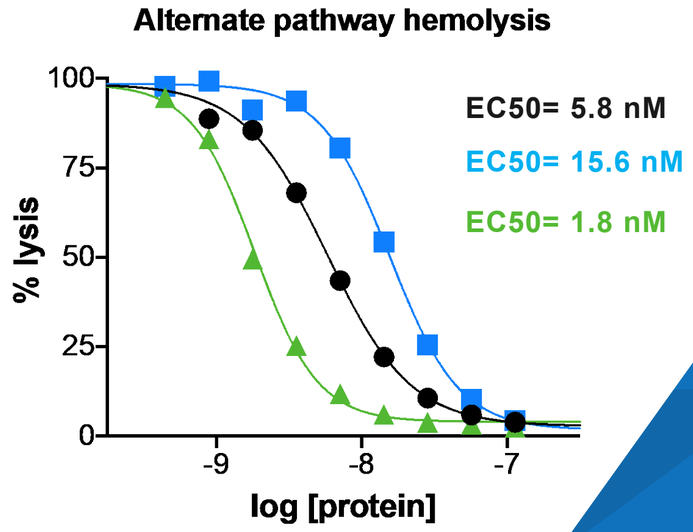
Using ProTUNE™ Platform to tune C3b & C4b cleaving capabilities

Rational Design



Reduction of Hemolysis

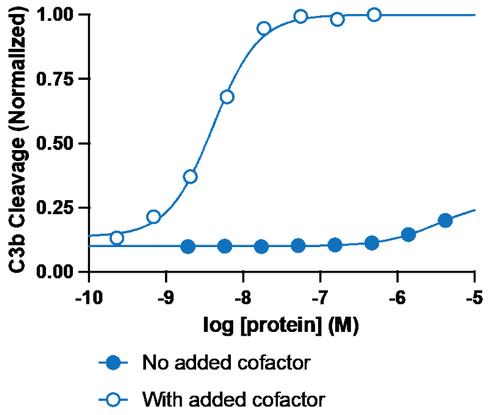




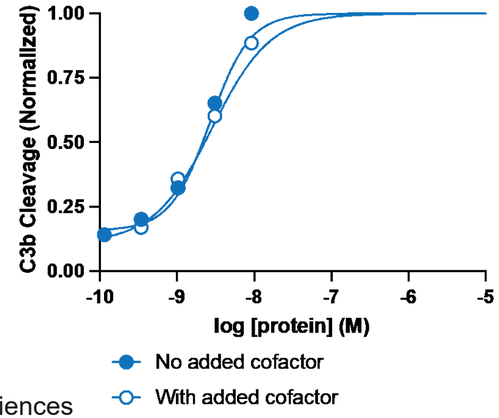
Cofactor independent CFI may target patient subpopulations

ProTUNE™ platform has generated degraders that work without cofactors

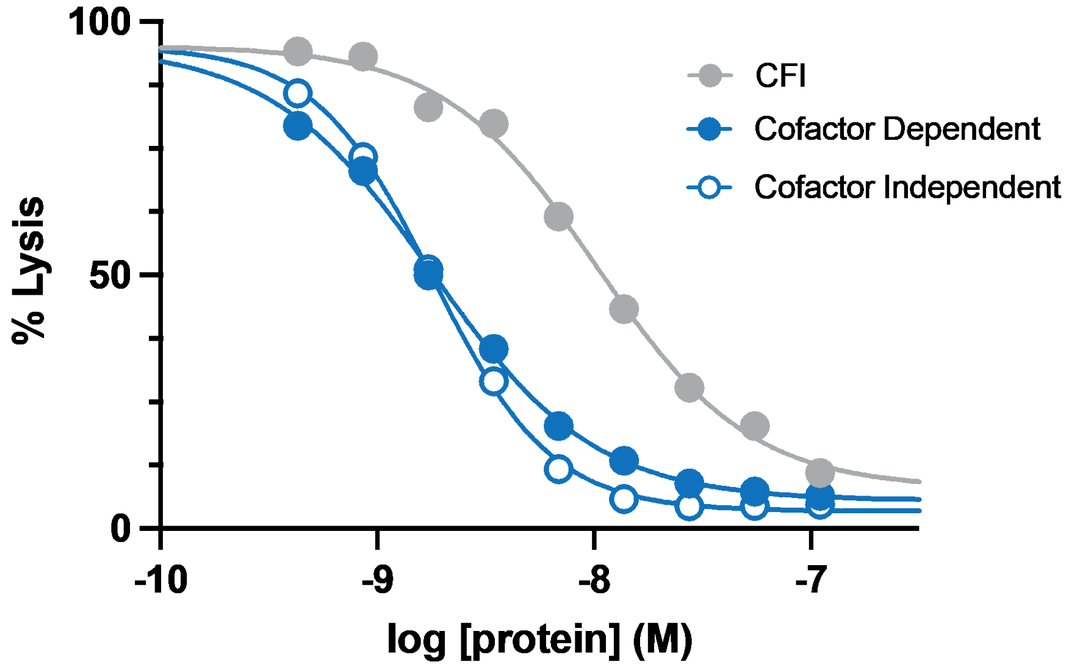
Cofactor Dependent



Cofactor Independent



Alternative Pathway Hemolysis



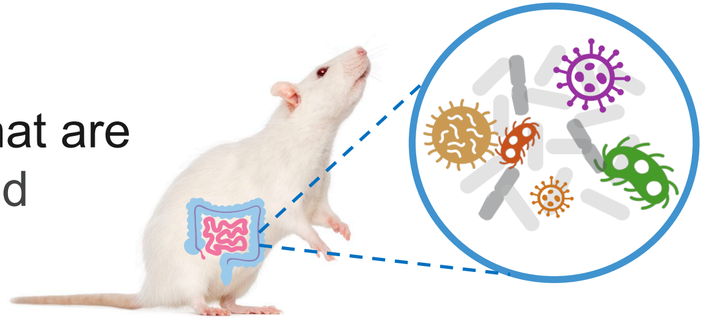
Cofactor independent molecules broaden the scope of addressable patients

Independently regulates C3b/C4b levels when cofactors are insufficient

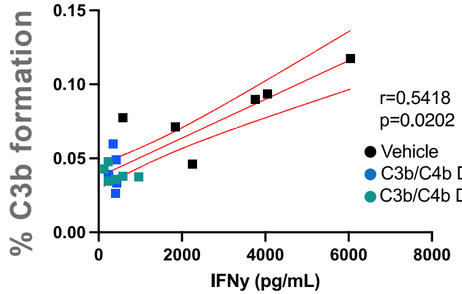
C3b & C4b degraders significantly reduce inflammation *in vivo*

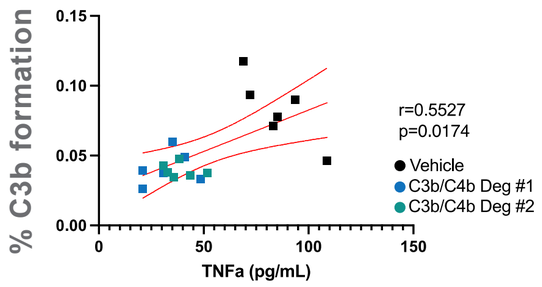
Rat sepsis model of complement activation

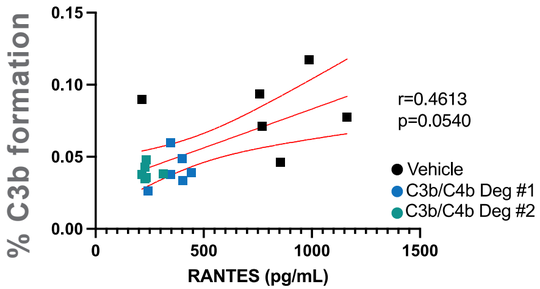
Reduction of **IFNγ, TNFα,** and **RANTES** that are chemokines involved in kidney damage and proteinuria in IgA nephropathy patients



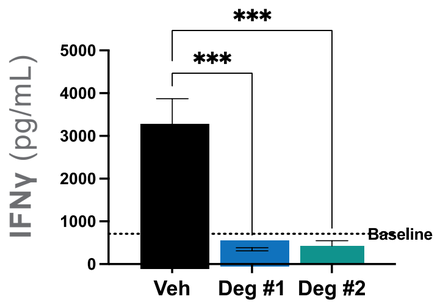
Concomitant reduction of inflammatory markers and complement C3 cleavage

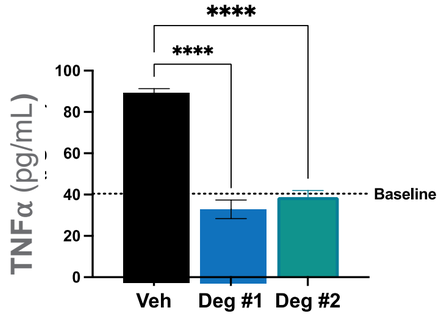


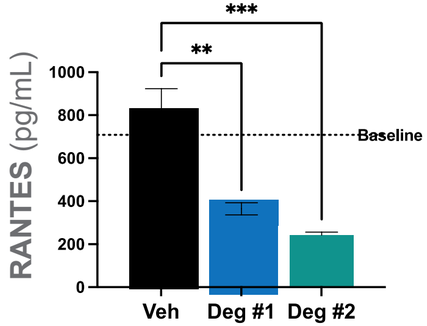




Inflammatory markers in IgA nephropathy





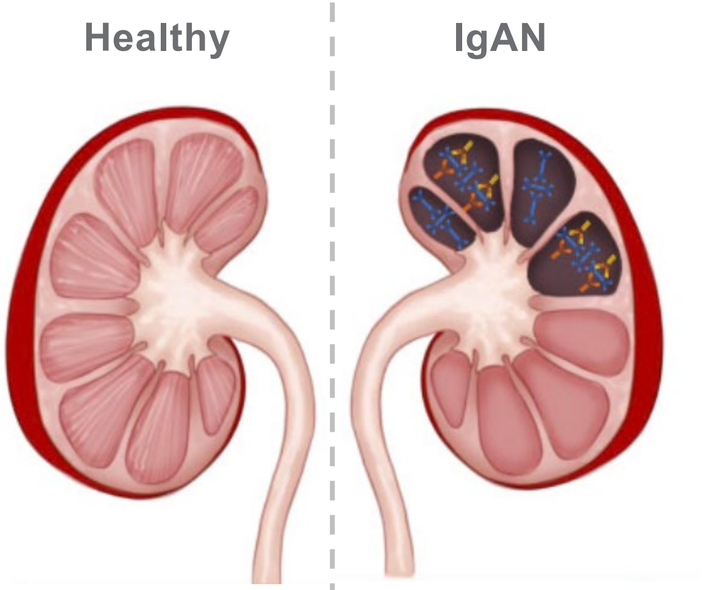


Example: C3b/C4b degraders for IgA nephropathy patients

Disease in which both lectin & alternative pathways drive pathogenesis

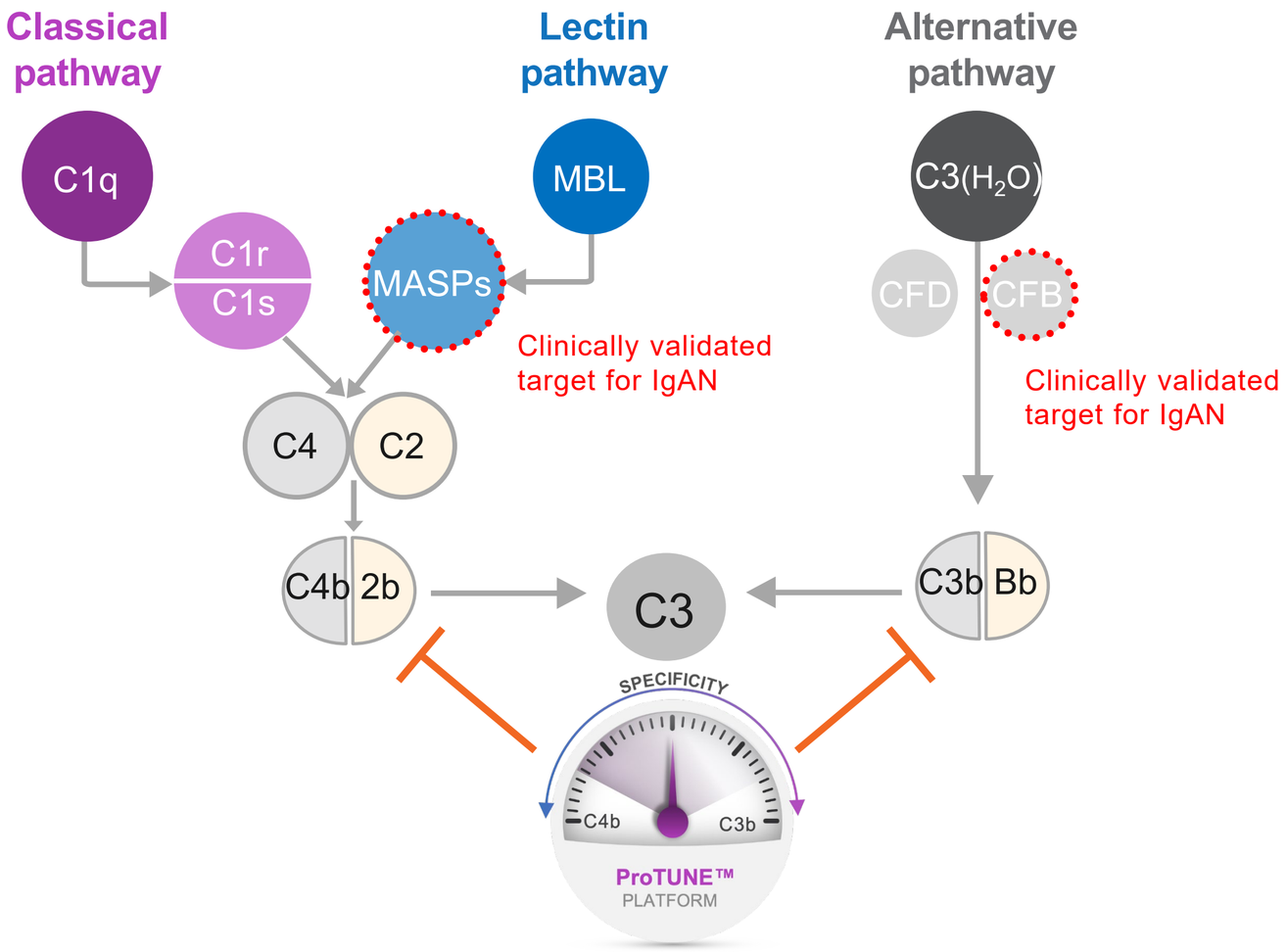
High unmet need – current treatments only address symptoms

- Most common form of glomerulopathy with accumulation & deposition of IgA immune complexes deteriorating 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
- **C3b/C4b degraders** will modulate the alternative & lectin pathways to address complement dysregulation with low off-target effects
- Significant burden on healthcare resources with an estimated cost of **$49.2 billion** in 2020 in the US



Example: C3b/C4b degraders for IgA nephropathy

Dual targeting of alternative & 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]

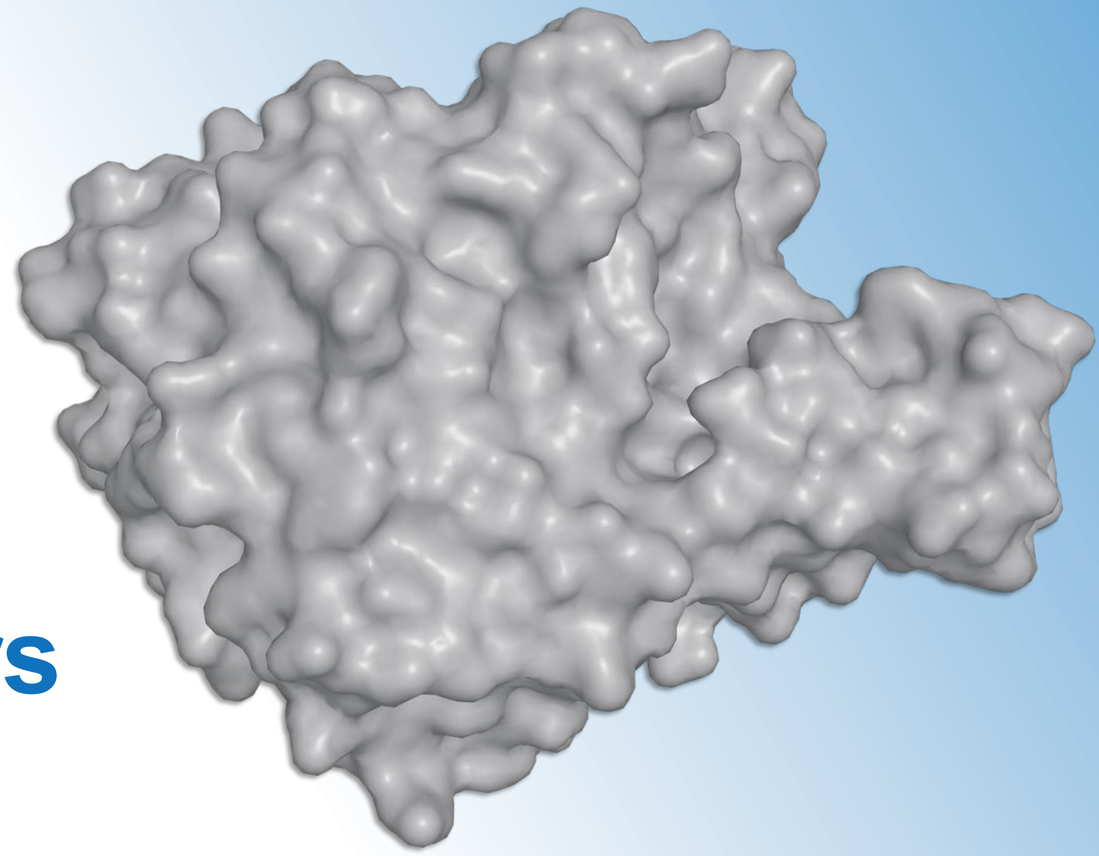
Clinically validated targets

+ 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)

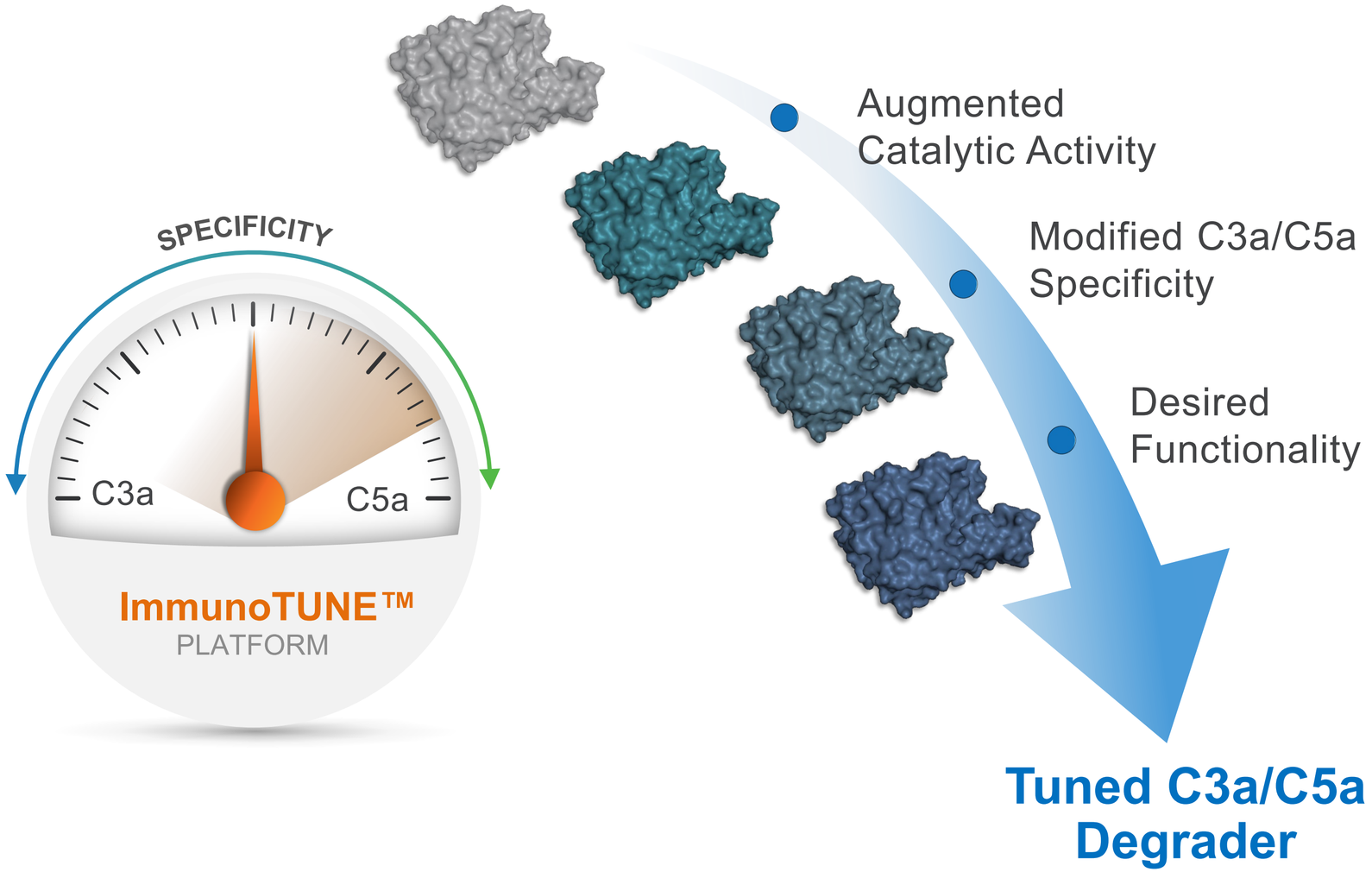
C3a & C5a Degraders

For inflammatory disorders



Dialing catalytic power & specificity to restore immunoregulation

Using the ImmunoTUNE™ engineering platform to tune C3a/C5a degraders



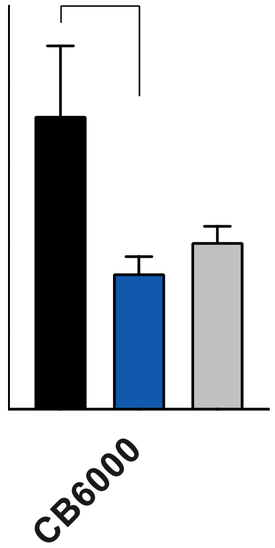
Precision Therapeutics

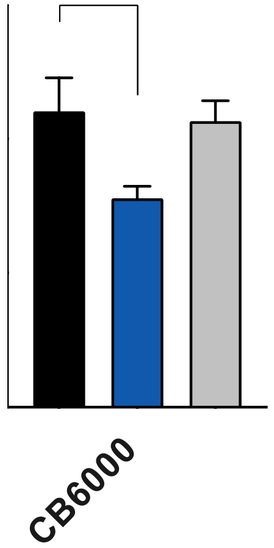
- **Tunable potency** to control dysregulated complement
- **Tunable specificity** toward C3a & C5a to restore balance to the complement cascade
- **Preserves host immune response**

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

Improves respiratory function & reduces cell infiltrates in an acute setting

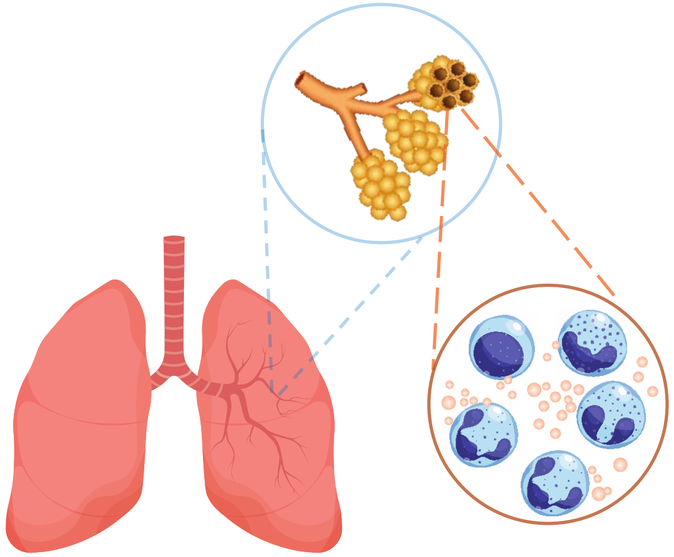
Respiratory functions & cell infiltration at 24 h





CFx <u>7-fold</u> lower dose vs. anti-C5

Mouse LPS model of lung inflammation

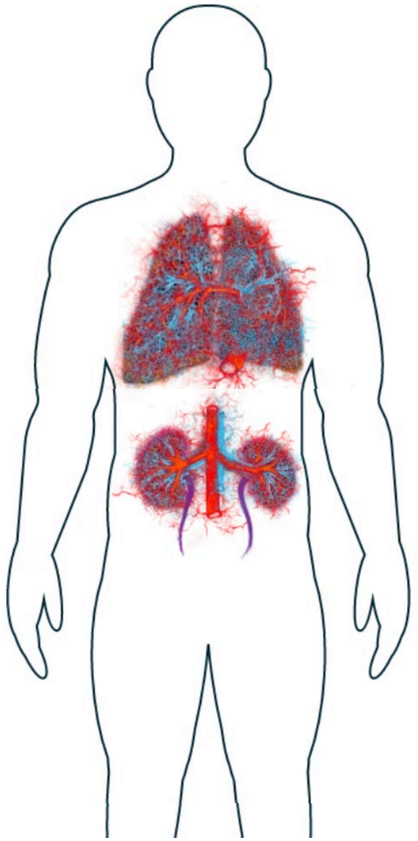


- CB 6000 **outperforms** anti-C5 antibody[1] in reducing inflammatory cell infiltration
- CB 6000 **compares well** on respiratory functions with anti-C5 antibody

[1]Mouse surrogate of Solaris (BB5.1)

Example: C3a/C5a degraders: Potential for ANCA Vasculitis patients

Autoimmune disease where anaphylatoxins play a role in the pathogenesis



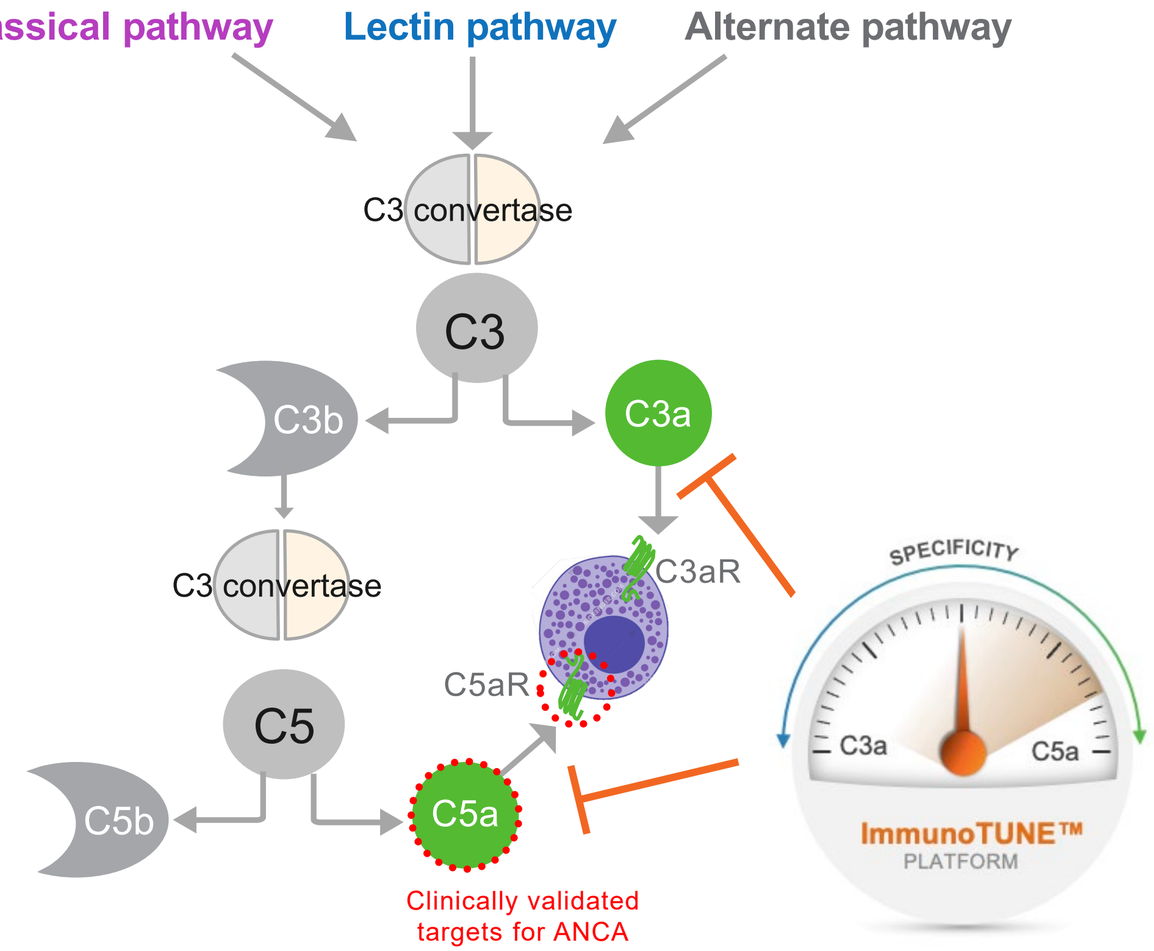
High unmet need – current treatments only addressing symptoms

- 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 within the 1st year of treatment with conventional therapies (immunosuppressant & steroids)
- The only treatments available are to manage the symptoms

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

Example: C3a/C5a degraders: Potential for ANCA Vasculitis patients

Dual targeting of both C3a & C5a with one protease medicine



Rationale for ANCA Vasculitis

+ Both **C3a & C5a** levels are elevated in active ANCA associated vasculiltis patients [1, 2]

Differentiation

+ Degrade activation products of C3 (C3a) & C5 (C5a) that are inflammatory mediators
+ Unlike Avacopan, may provide beneficial function via blocking the **C5L2 pathway, an alternative receptor for C5a**

Clinically validated targets

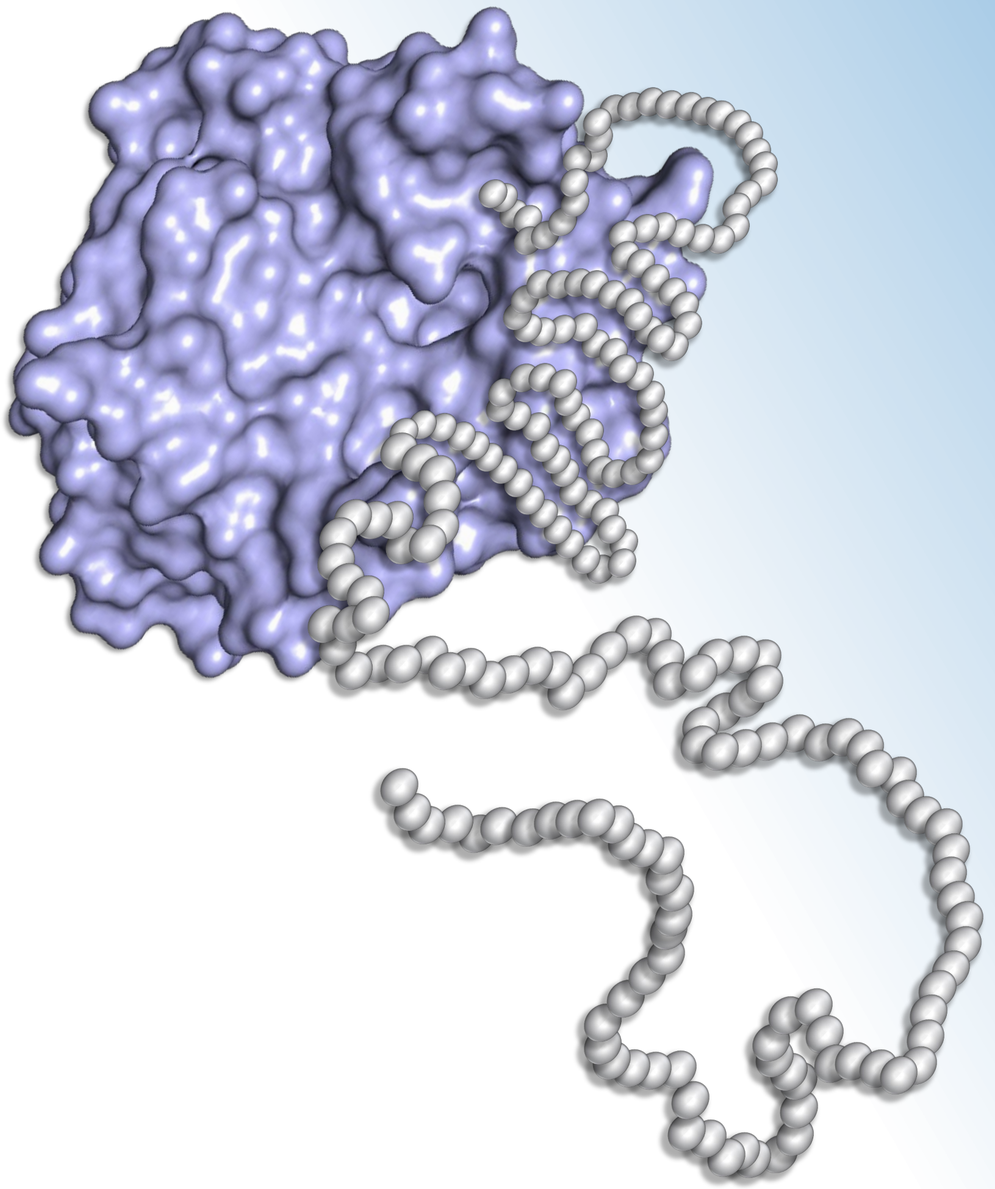
+ Inhibition of **C5a or C5aR alone may be insufficient** to increase remission rates in ANCA associated vasculitis patients

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

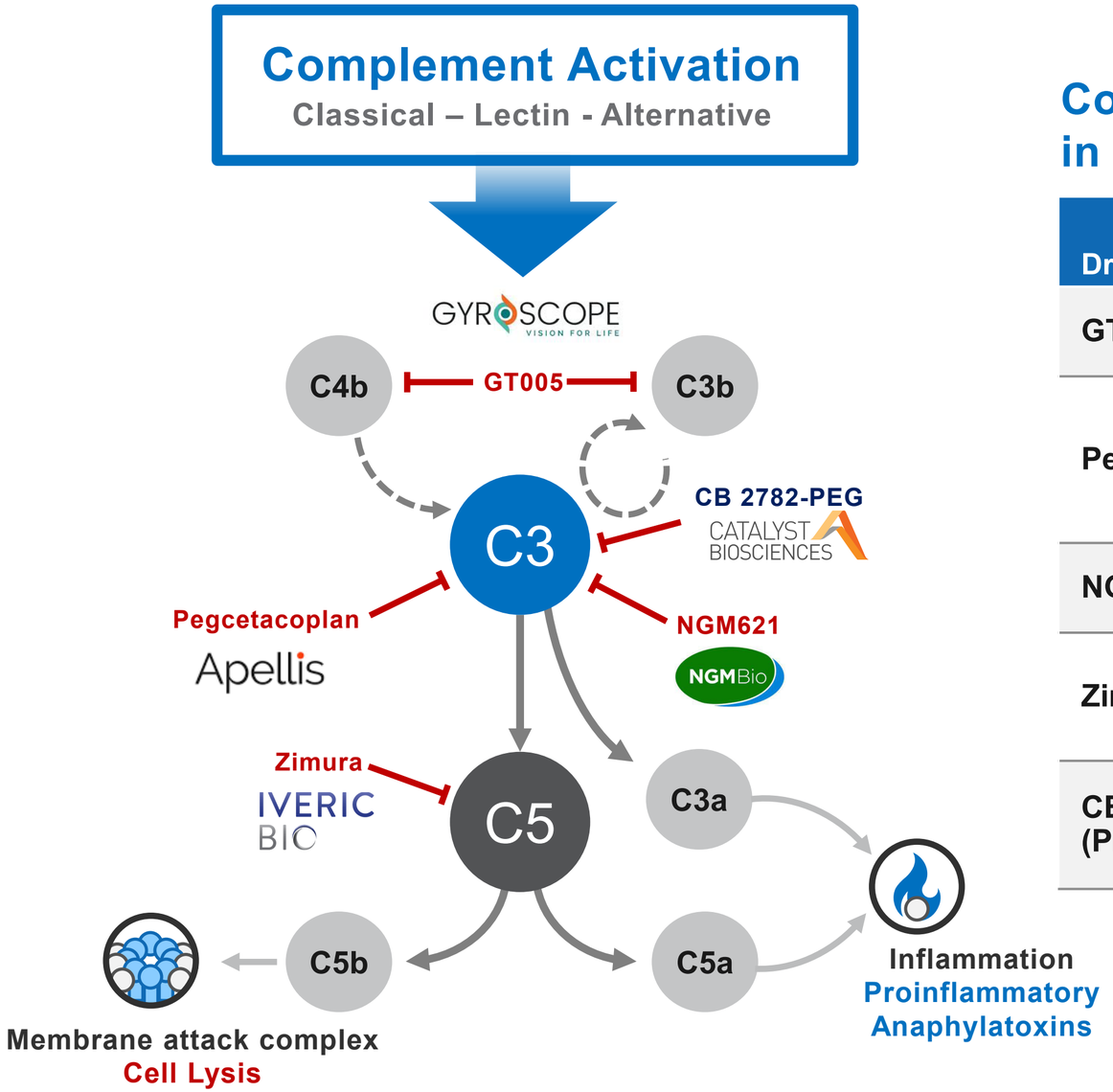
CB 2782-PEG

Novel engineered C3 degrader

Partnered with Biogen



Complement inhibition is a validated approach in dry AMD



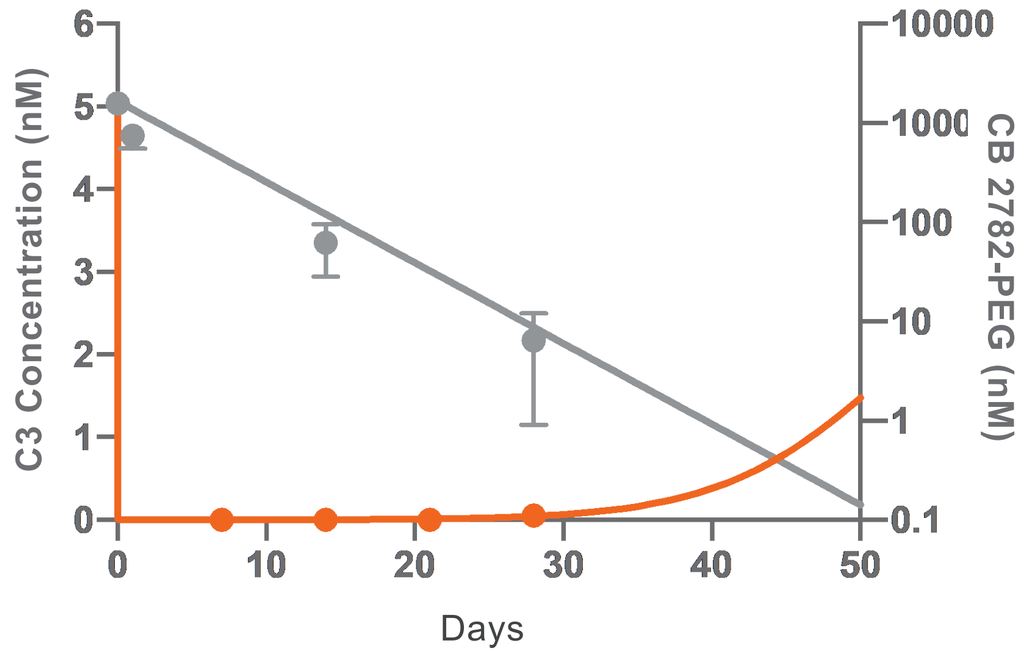
Complement inhibition shows promising reduction in GA growth

Drug	Category	Route	Dose level	Frequency	Inhibition GA growth
GT005	CFI gene therapy	Subretinal	$2E^{11}$ vg	Once	Unknown
Pegcetacoplan	PEGylated amino acid cyclic peptide	Intravitreal	15 mg	1-2 months	20-30%
NGM621	Antibody anti-C3	Intravitreal	15 mg	1-2 months	Unknown
Zimura	pegylated RNA aptamer	Intravitreal	2 mg	1-2 months	20-30%
CB 2782-PEG (Preclinical)	PEGylated anti-C3 protease	Intravitreal	Low undisclosed	2-3 months Model of NHP data	Unknown

CB 2782-PEG: Best-in-class C3 degrader for dry AMD

The 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

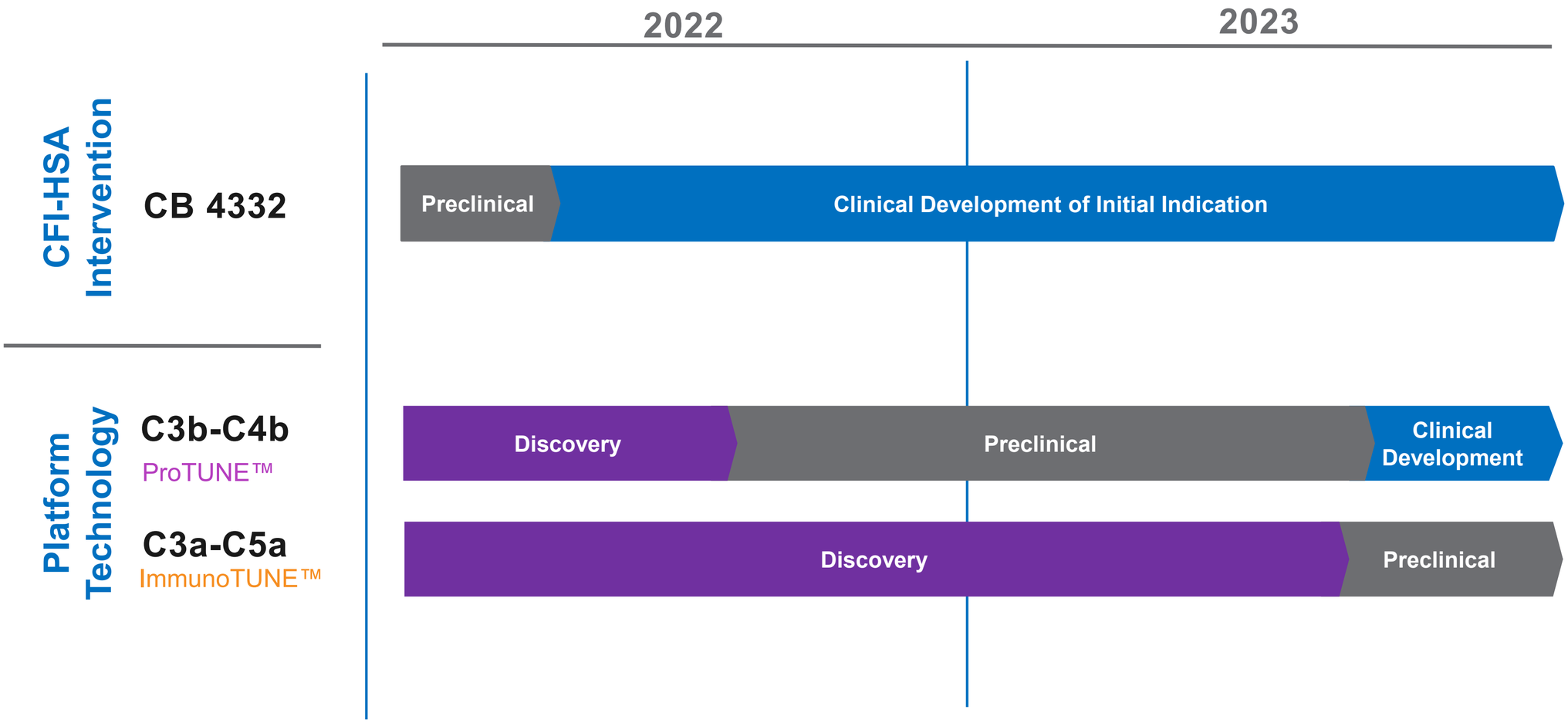
Catalyst Biosciences protease platform has broad potential

Building on nature’s way of regulating key processes in health & disease

- Catalyst develops enhanced **natural core proteases & protease degraders targeting complement regulation**
- Catalyst has designed optimized, next-generation **complement degrader candidates**
- Complement dysregulation serves as **driver** for many diseases with **unmet needs**
- Catalyst has **protease programs** designed to take advantage of nature’s natural complement regulators that **restore complement homeostasis** & potentially treat **other complement-mediated disorders**
- Application of Catalyst's protease & protein degrader technology could unlock treatments in **immunology, nephrology, hematology, ocular diseases, and beyond**

Overview of complement portfolio

Multiple value generating events in 2022 & 2023



THANK YOU

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