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 13, 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 8.01 Other Events.

On July 13, 2021, Catalyst Biosciences, Inc. (the "Company") gave a presentation on targeting complement in ARDS (Acute Respiratory Distress Syndrome), including the potential advantages of the Company's protease engineering platform, and data related to its discovery stage Complement Factor B degrader program (the "Complement Presentation") at the Hanson Wade ARDS Drug Development Summit, Digital Events. In addition, the Company posted an update to its corporate presentation (the "Presentation") on its website, ir.catalystbiosciences.com/presentations-events. A copy of the ARDS Complement Presentation is attached hereto as Exhibit 99.1.

The Complement Factor B degrader program is in discovery stage and targets diseases caused by deficient regulation of the complement system. Complement Factor B degraders are proteases engineered by the Company's proprietary protease engineering platform designed to specifically degrade complement Factor B, a component of the alternative pathway. The presentation discloses *in vitro* and *in vivo* results of studies with complement Factor B consistent with complement Factor B degraders reducing alternative complement pathway activation. *In vivo* studies in a mouse model of ARDS demonstrated that one of the Company's discovery stage complement Factor B degraders improved mouse respiratory parameters to a similar therapeutic level as the comparator LNP023 (iptacopan), a small molecule inhibitor of Factor B from Novartis currently in clinical trials.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits

Exhibit No.	Description
99.1	ARDS Complement 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 16, 2021

/s/ Clinton Musil
Clinton Musil
Chief Financial Officer

CATALYST BIOSCIENCES

ARDS summit, July 13th 2021

Complement System in ARDS

Natacha Le Moan

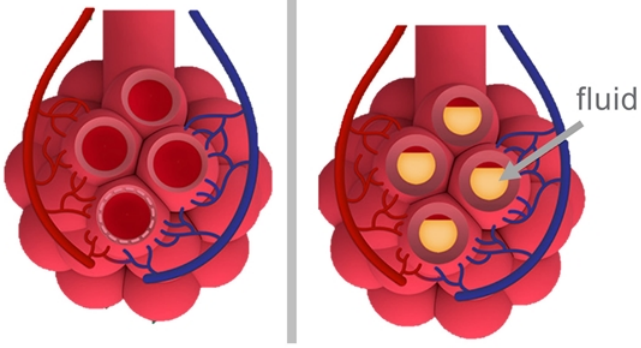
CatalystBiosciences.com

Target the complement cascade to maxim your ARDS therapeutic

- ARDS: unmet need after half a century of research
- Complement involvement in ARDS
- Challenges in developing complement therapeutics for ARDS
- Shifting the paradigm of preclinical models
- Translational research at Catalyst Biosciences: “CFB degrad

Acute Respiratory Distress Syndrome (AR

A potentially fatal respiratory condition with seriou



+ **~200k cases**

+ **~40-50%** of h
within the firs

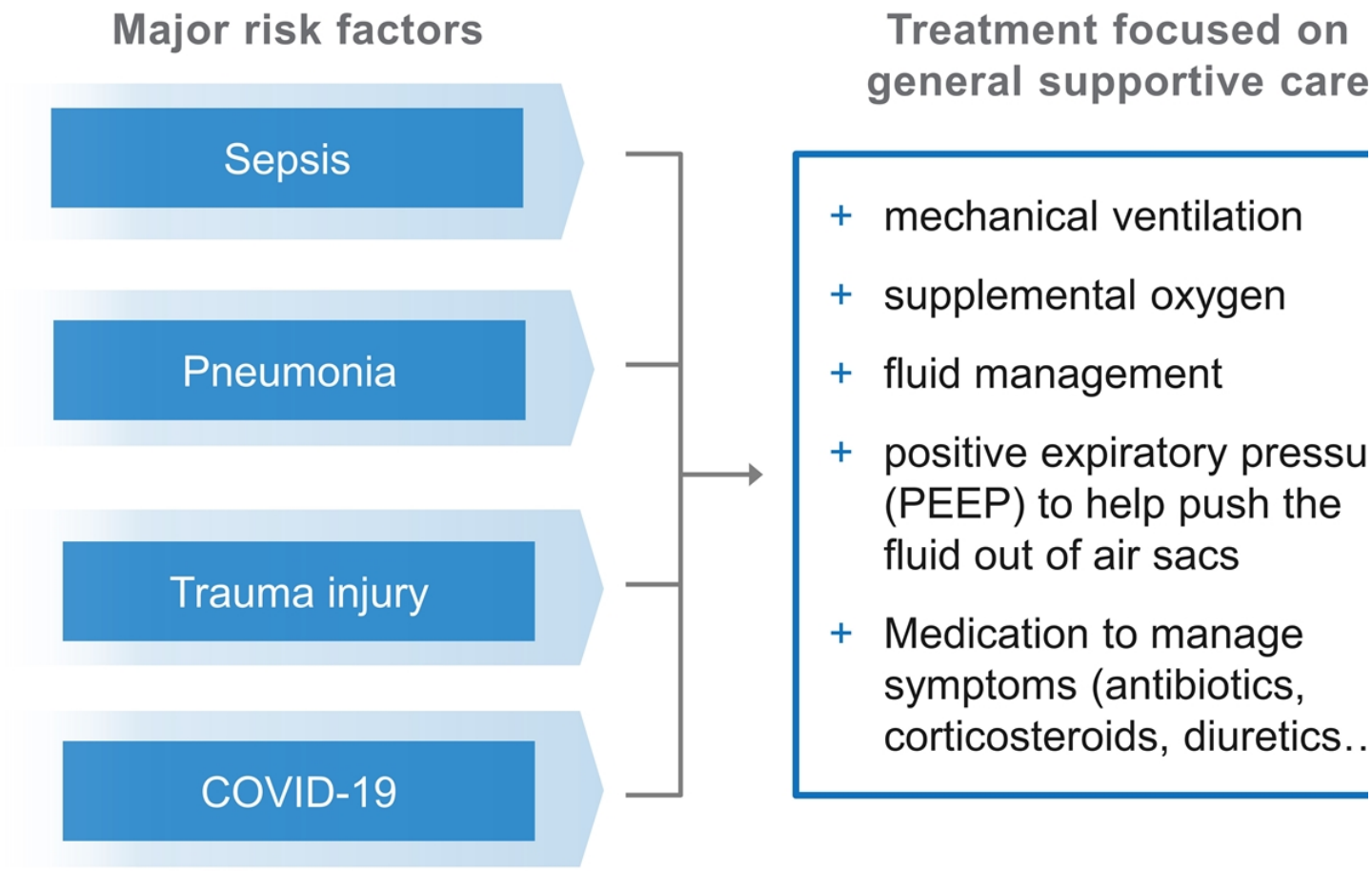
+ **~10%** of ARD
replacement

+ **~10%** of inter

+ Significant bu
estimated co

Acute Respiratory Distress Syndrome (AR

A potentially fatal respiratory condition with seriou



Pham et al.. American J

Target the complement cascade to maxim your ARDS therapeutic

ARDS: unmet need after half a century of research

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Translational research at Catalyst Biosciences: “CFB degrad

Complement system activation – a pathog

Complement inhibitors decrease lung inflammation

+ **Complement inhibition in COVID-19 patients with ARDS**: decrease systemic inflammation (AMY-101, IFX-1) recovery after severe ARDS cases (Eculizumab) and improved clinical inflammation indicators and recovery (Narsoplimab)[1].
+ **Serum/plasma**: upregulation of both the classical complement pathway (C1R, C1S, and C8A), the alternative pathway CFB, the complement modulators CFI and CFH, the MAC proteins such as sC5b-9, C5, C6, and the anaphylatoxins C5a and C3a[1].
+ **Lung tissue**: upregulation of complement proteins (MBL, MASP2, C4a, C4d, C3, and MAC C5b-9) seen in post-mortem lung tissues from COVID-19 patients[2, 3].

[1] L
[2] M
[3] T

Complement system activation in ARDS p

Conflicting observations due to heterogenous popu

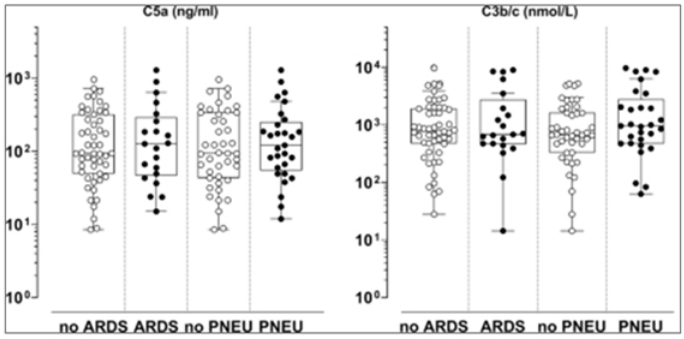
+ Lower C3 in patients with severe ARDS associated with death[1]

	Total	Mild	Moderate	Se
# Patients	201	31	61	1
C3	**5.62** ± 1.78	**12.27** ± 1.83	**7.13** ± 3.14	0.84
C4	**1.33** ± 0.58	**0.25** ± 0.13	**0.24** ± 0.11	**2.25**

+ Higher C3a in trauma patients predisposed to ARDS[2]

Time after admission	+ ARDS Median	+ ARDS Range	− ARDS Median	− ARDS Range	*P*
0 h	10·4	0–54·7	7·9	2·1–74·1	0·835
6 h	15·0	4·9–36·2	8·0	2·0–20·0	0·007*
12 h	10·8	3·4–23·2	6·3	2·8–14·3	0·047*
24 h	6·6	2·5–26·7	4·9	2·7–30·0	0·404
48 h	8·0	4·2–31·6	6·9	3·0–20·8	0·482
5 d	12·1	6·1–44·4	6·1	2·3–14·7	0·004*
7 d	19·5	5·1–44·4	6·9	4·8–17·6	0·003*
9 d	15·5	8·3–48·8	9·9	5·4–24·4	0·029*
11 d	17·1	7·7–39·2	7·1	3·8–26·7	0·008*
13 d	20·6	9·2–34·5	6·8	4·8–30·8	0·020*

+ No changes in C5a/C3b patients under invasive ventilation[3]



[1] *Song et al. BMC Pulmonary Medicine (2020)*
[2] *Zilow et al. Clin. exp. Immunol. (1990)*
[3] *De Beer et al. Intensive Care Medicine Experimental (202*

Target the complement cascade to maxim your ARDS therapeutic

ARDS: unmet need after half a century of research

Complement involvement in ARDS

Challenges in developing complement therapeutics for ARDS

Shifting the paradigm of preclinical models

Translational research at Catalyst Biosciences: “CFB degrad

ARDS treatments – a double edge sword

Therapeutic interventions to stabilize patients lead

Early therapeutic approaches driving complement dysregulation[1]

TRAUMA

Scene	ER	OR	ICU	Pathophysiology	Complement
Intubation/ventilation				ROS	Activation
Volume resuscitation				Hyperdilution	Depletion
Transfusion of blood products				Feed in of complement	Anaphylatoxins
Instrumentation/monitoring				Artificial surfaces	Activation
Tranexamic acid				Plasmin inhibition	Activation, C5a↓↑
		Surgical damage control		Additional tissue injury	Activation, depletion
		Osteosynthesis		Artificial surfaces	Activation
			Haemodialysis/ECMO	Artificial surfaces	Activation, depletion

Pote

Excessive activation

Activation

Baseline

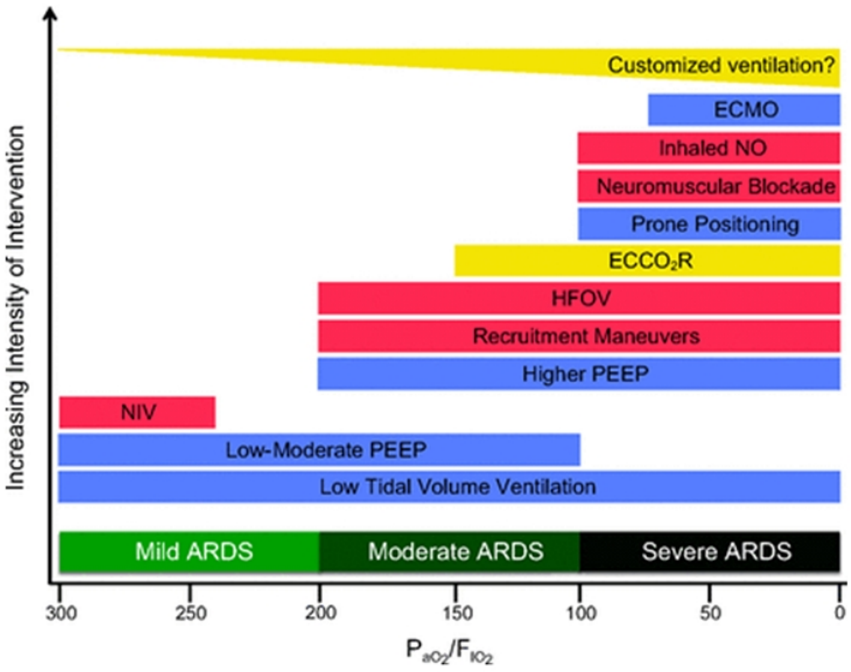
Depletion

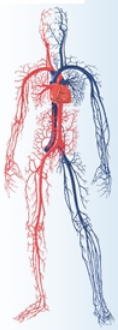
[1] *Huber-Lang et al. Br J Pharmacol. (2021)*
[2] *Karasu et al. Front Immunol (2019)*

Challenges in ARDS therapeutics targetin

Early recognition of severe ARDS is key for optimal

Potential interventions for management of ARDS





Improve clini

+ Identify “trea
+ Develop spe diagnose se
+ Dedicate spe teams/protoc severe ARDS
+ Identify failu treatment st managemen
+ Identify surro addition to m

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Develop animal models “as clinically relev

Not such a basic concept in ARDS

+ **Interspecies differences:**
  - Lung anatomy
  - Innate and adaptive immune responses
  - Histopathological hallmarks of lung damage
+ **Paradigm of ARDS animal models:**
  - Lack of co-interventions and organ support
  - Lack of pre-existing lung injury
  - Pretreatment or peri-injury therapeutic approaches
  - Lack of translatable endpoints

Translational challenges

Toward a paradigm shift in ARDS preclinic

Similarities between stroke and ARDS translational

Preclinical recommendations in Stroke (STAIR)

- Multiple models and species including large ones
- Comparison with SoC
- Aged animals with co-morbid conditions
- Delayed therapeutic treatment & late endpoints
- Randomized & blinded

Precl

+ M
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+ La
er
+ C
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+ Av
pr

Genetic depletion of complement compon

Not all pathologies will benefit equally from comple

Genotype	Mouse model	Conclusions [1, 2, 3, 4]	Ben
WT	SARS-CoV infection	Elevated C3 activation products	-
C3 $^{-/-}$	SARS-CoV infection	Partial reduction of respiratory dysfunction, immune cell infiltrations and cytokine response	+
CFB $^{-/-}$	SARS-CoV infection	Lower weight loss	+
C4 $^{-/-}$	SARS-CoV infection	Lower weight loss	+
MASP2 $^{-/-}$	S. pneumoniae infection	Increase mortality rate	-
C1q $^{-/-}$	S. pneumoniae infection	Increase mortality rate	-
C4 $^{-/-}$	S. pneumoniae infection	Increase mortality rate	-
CFB $^{-/-}$	S. pneumoniae infection	Increase mortality rate	-
C3 $^{-/-}$	S. pneumoniae infection	Increase mortality rate	-
C3 $^{-/-}$	LPS infusion	No effect	n/a
C5 $^{-/-}$	LPS infusion	No effect	n/a

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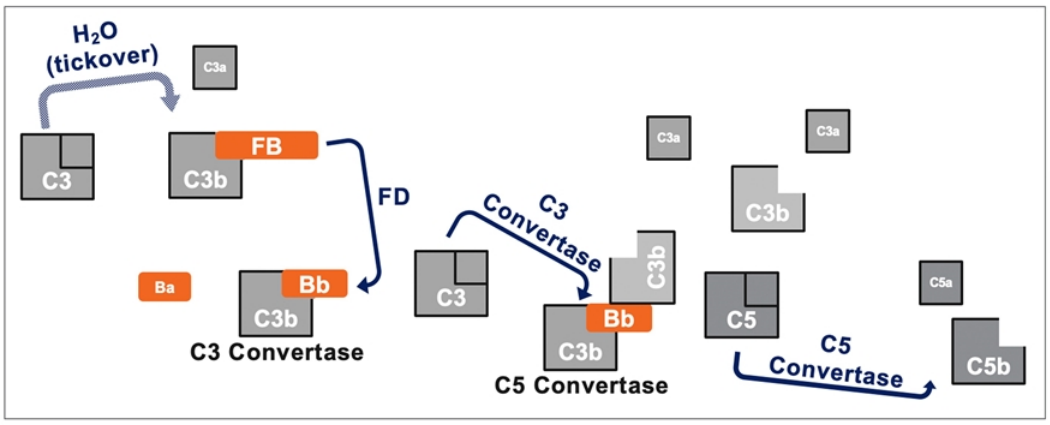
Gold standard assay to evaluate complem

Hemolysis assay principle

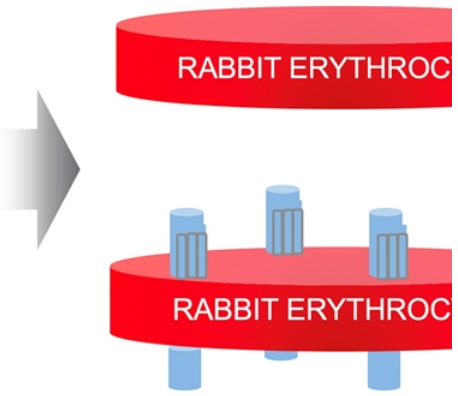
1 Serum pre-incubation

C3 and C5 convertase formation drives C5b accumulation



2 Add serum to R

Accumulated compon
C5b-C9 membrane
complex (MAC) for



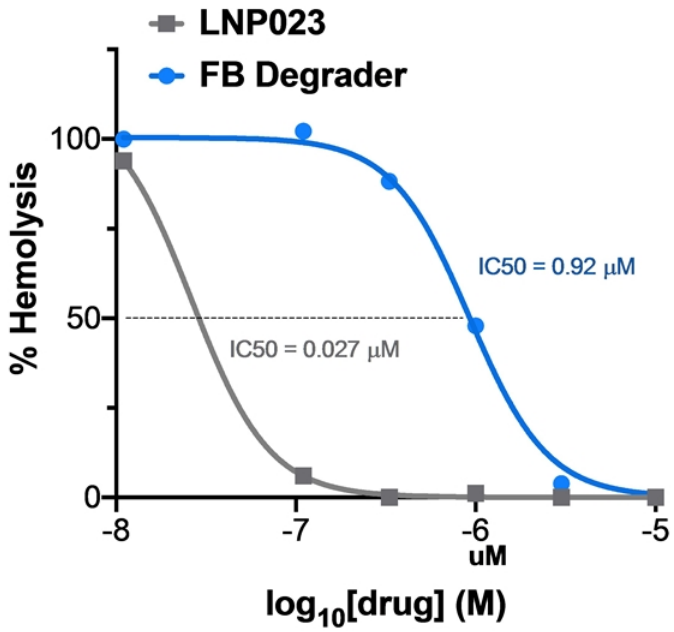
Rabbit red blood cells exp
complement cofactors an

Blom

Standard and modified alternate pathway

Comparison of CFB degrader vs LNP023 in two hem

Standard AP Hemolysis Assay (10 min)

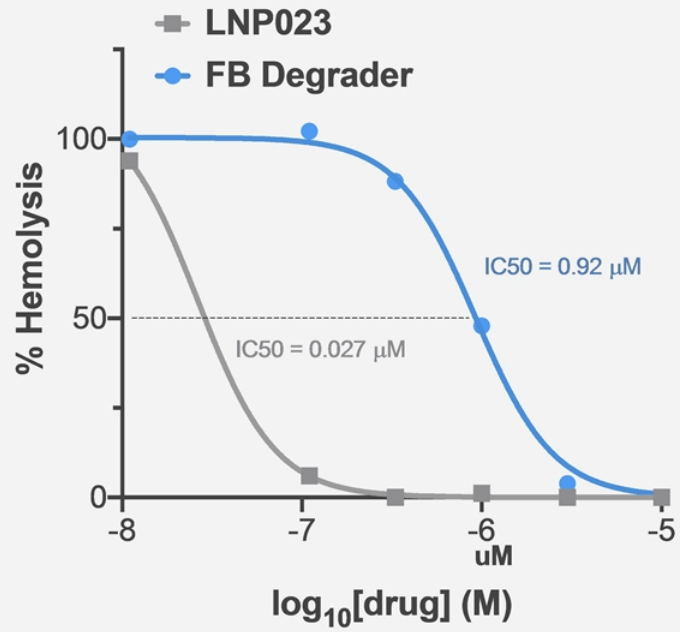


- **In an acute setting, LNP023 potency in hemolysis assay findings[1]**

Standard and modified alternate pathway

Comparison of CFB degrader vs LNP023 in two hem

Standard AP Hemolysis Assay (10 min)



~16-fold increase in **CFB**

+ increase incubation time

"Mo

% Hemolysis

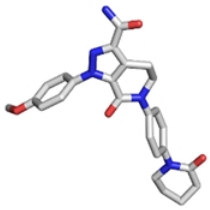
Drug : CFB ratio

- **LNP023 potency is markedly reduced from 10 to 180 m in target (Bb)**
- **FB degrader potency is independent of target concent**

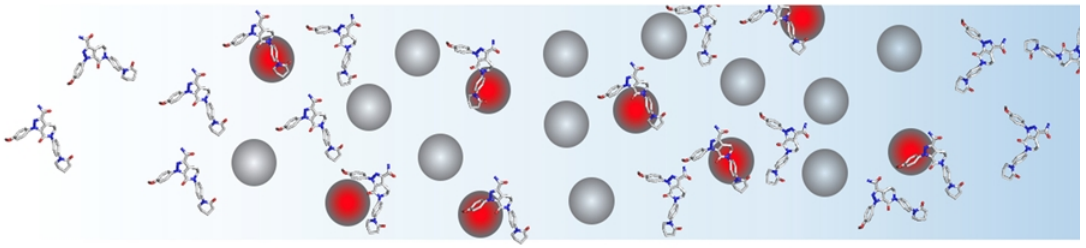
Proteases are ideal for high abundancy ta

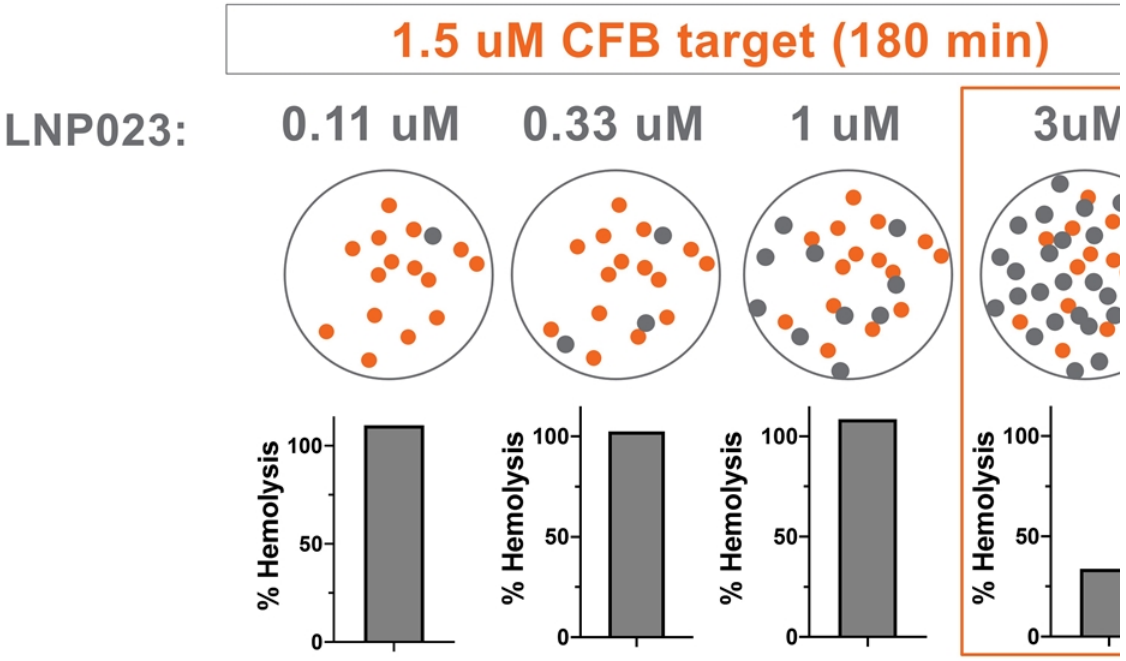
A better way to regulate biological processes comp

Small molecules



Therapeutic target neutralization

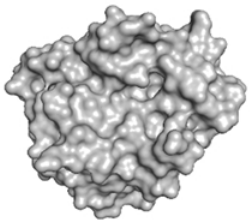




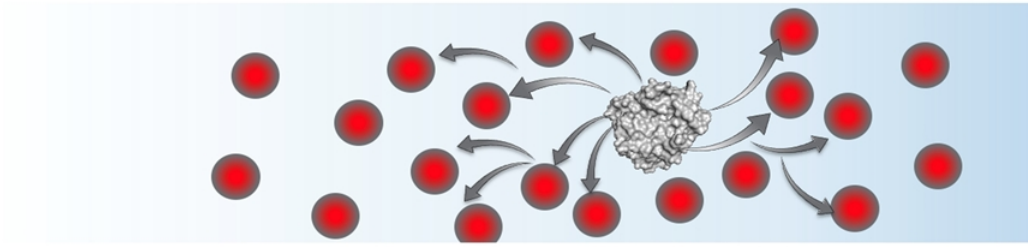
Proteases are ideal for high abundancy ta

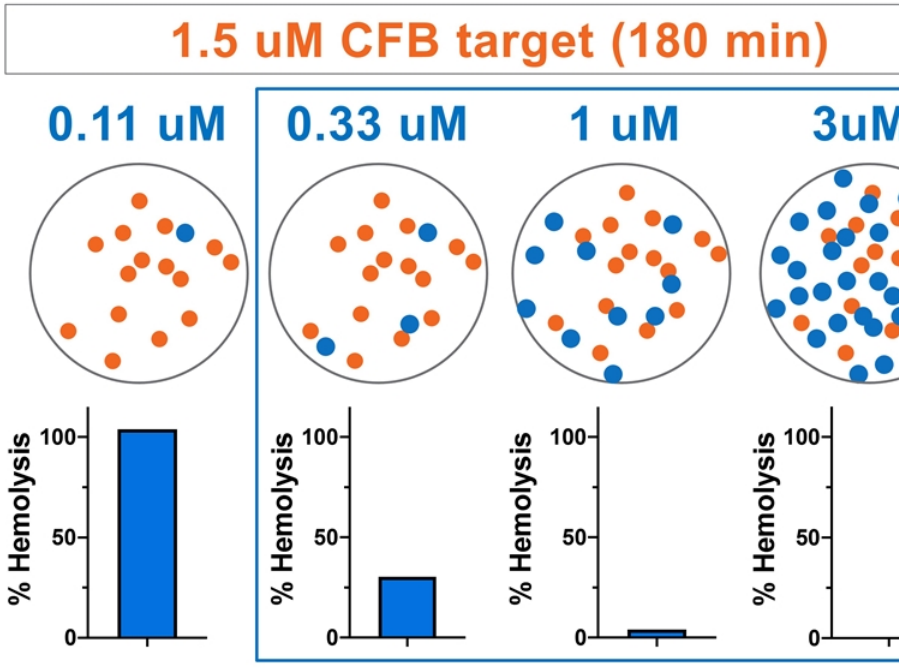
A better way to regulate biological processes comp

Protease therapeutics



Therapeutic target neutralization





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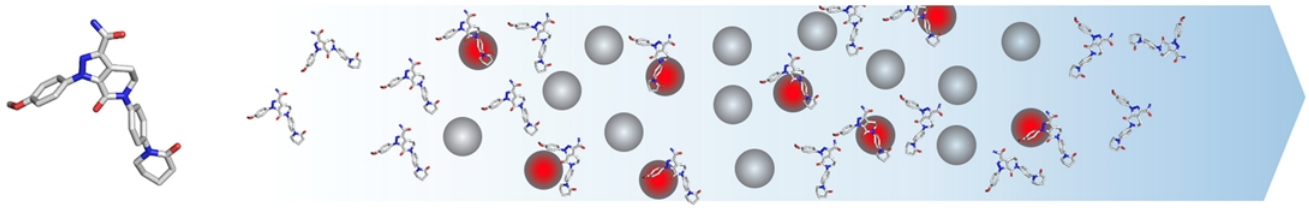
A better way to regulate biological processes comp

Small molecules

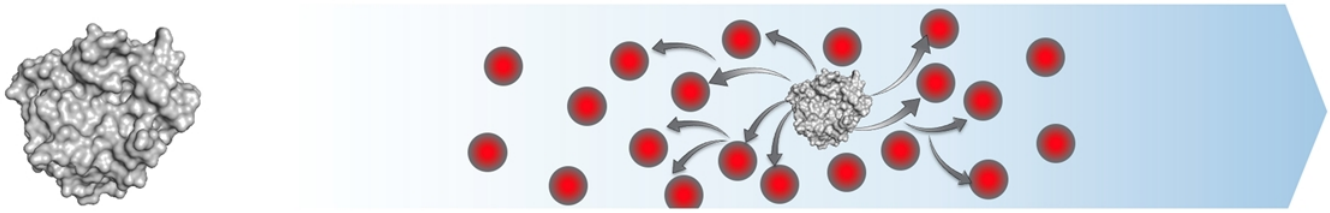
Therapeutic target neutralization



LNP

Protease therapeutics

Therapeutic target neutralization



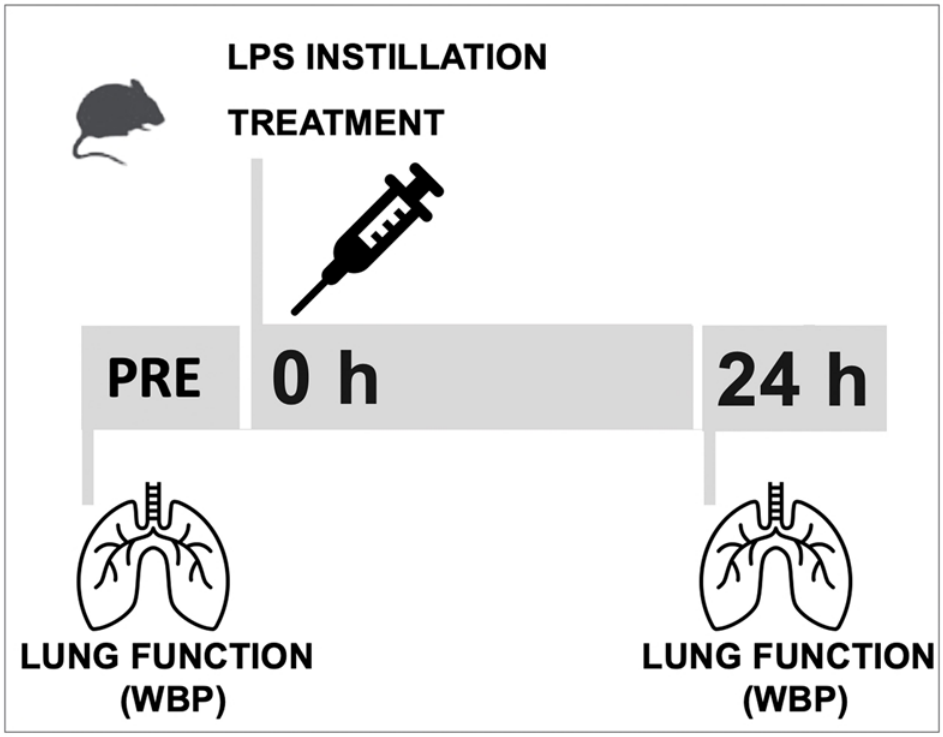
degr

+ CFB degraders offer potential advantages over small molecules to CFB over time to prevent complement activation in patients at risk

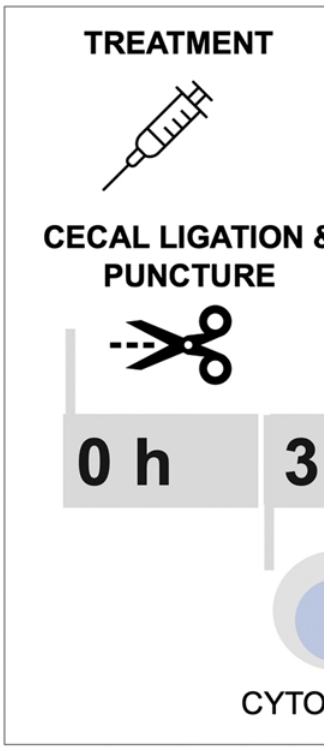
Iterative translational approach to screen for c

"High throughput" screening in acute models of co

Mouse LPS instillation



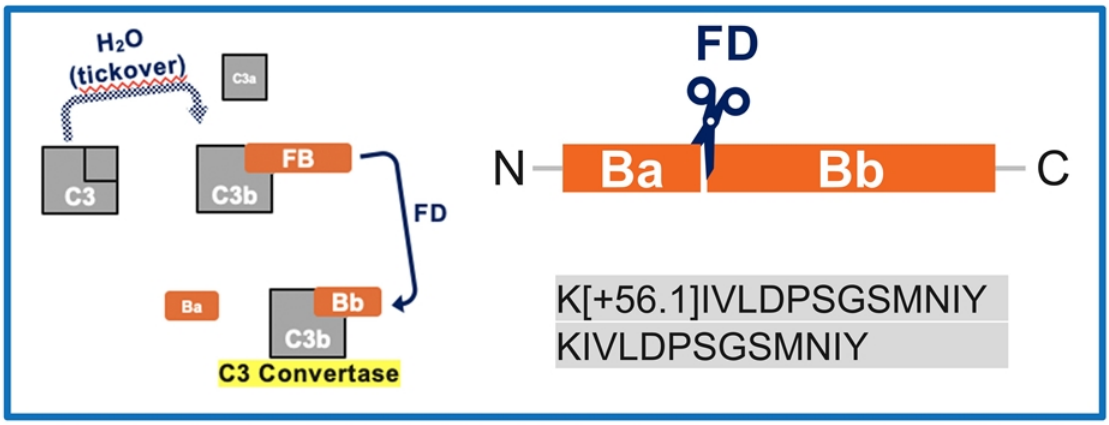
Rat microbial



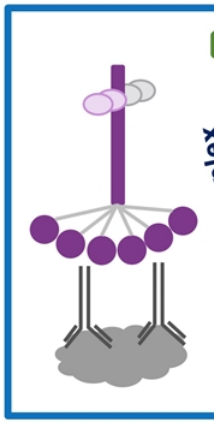
Mass spectrometry enables detection of c

Measure classical & alternate pathway activation wi

Alternate pathway (Bb fragment)



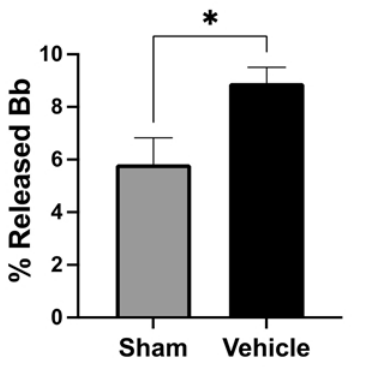
Classica

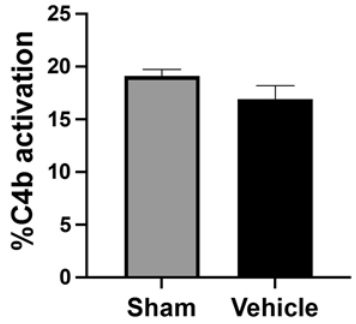


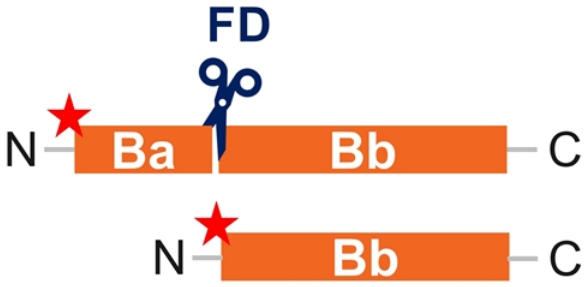
Mass spectrometry in LPS-induced ARDS

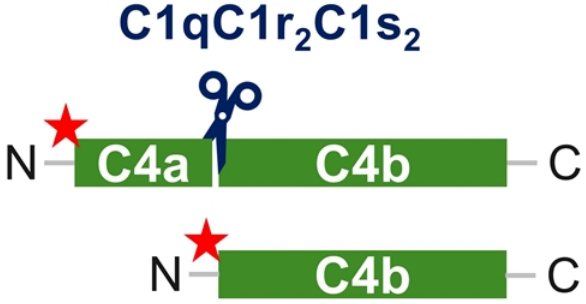
Differences in classical & alternate pathway activati

LPS instillation







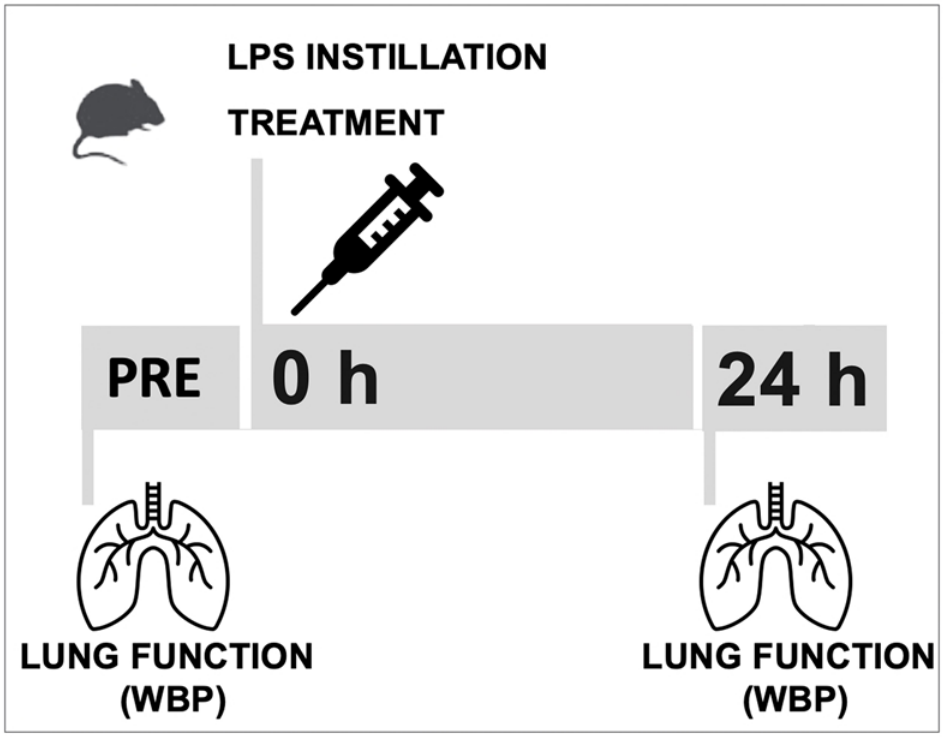


Alternate pathway activation

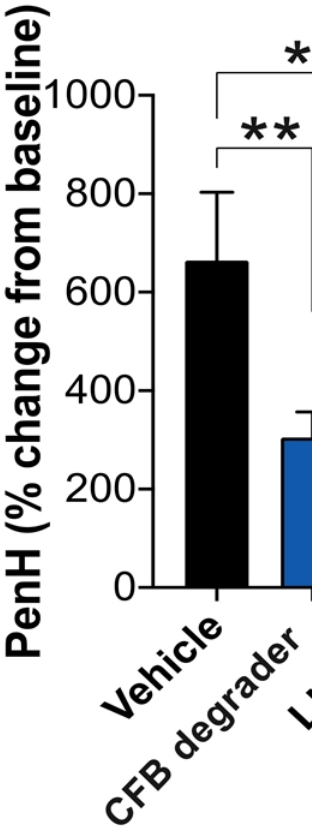
CFB degraders compare well vs LNP023

CFB degraders reduce inflammation in acute roden

Study paradigm:



Improven



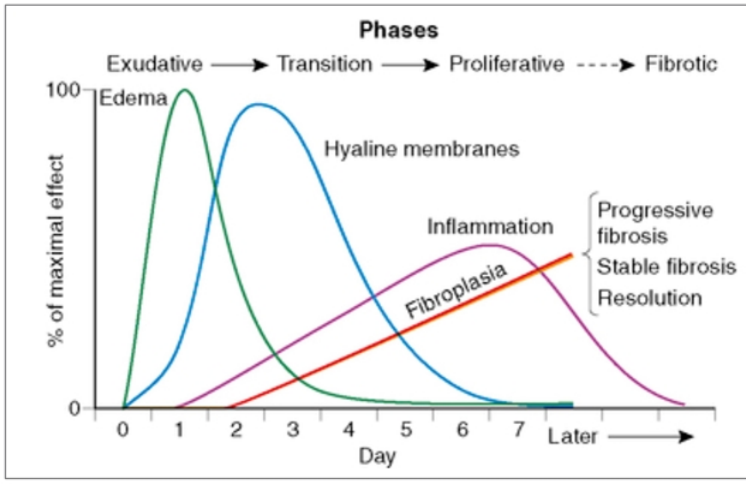
#CFB degrader 325

Discussion points

- **Which complement targets should be selected in chronic phase of ARDS?**

- **In absence of clear subpopulation within ARDS p may be suitable to examine the efficacy of compl**

- **Which complement measurements would be mea**



Modified from Katzenstein A: Acute lung injury patterns: diffuse alveolar damage and bronc obliterans–organizing pneumonia. In: Katzenstein A, Askin F, eds. Katzenstein and Askin's Surgical Pathology of Non-Neoplastic Lung Disease, 3rd ed. Philadelphia: Saunders; 1997.