BLADE

THERAPEUTICS

Developing Cutting-Edge Treatments for
Debilitating Fibrotic and Neurodegenerative Diseases

Corporate Presentation

May 16, 2022

Disclaimer and Other Important Information

This Presentation (the “Presentation”) is for informational purposes only to assist interested parties in evaluating the business of Blade Therapeutics, Inc. (the “Target”). The Target and Biotech Acquisition Company ("BAC") have entered into a proposed initial business combination (the “Transaction” or “Business Combination”), pursuant to which the Target will become a wholly-owned subsidiary of BAC. In connection with the closing of the Business Combination, BAC will re-domesticate as a Delaware corporation and will change its name to “Blade Biotherapeutics, Inc.” The continuing combined entity is hereinafter referred to as the “Company” or the “Combined Entity”.

The information contained herein does not purport to be all-inclusive and none of the Target, nor any of its subsidiaries, stockholders, affiliates, representatives, control persons, partners, members, managers, directors, officers, employees, advisers, contract counterparties or agents make any representation or warranty, express or implied, as to the accuracy, completeness or reliability of the information contained in this Presentation. Recipients should consult with their own counsel and tax and financial advisors as to legal and related matters concerning the matters described herein, and, by accepting this Presentation, you confirm that you are not relying upon the information contained herein to make any investment decision. To the fullest extent permitted by law, in no circumstances will the Target or any of its subsidiaries, stockholders, affiliates, representatives, control persons, partners, members, managers, directors, officers, employees, advisers, contract counterparties or agents be responsible or liable for any direct, indirect or consequential loss or loss of profit arising from the use of this Presentation, its contents, its omissions, reliance on the information contained within it, or on opinions communicated in relation thereto or otherwise arising in connection therewith. In addition, this Presentation does not purport to be all-inclusive or to contain all of the information that may be required to make a full analysis of BAC, the Target, or the Business Combination. The general explanations included in this Presentation cannot address, and are not intended to address, your specific investment objectives, financial situations or financial needs.

In connection with the proposed Business Combination, BAC has filed with the Securities and Exchange Commission (the “SEC”), a registration statement on Form S-4, containing a preliminary proxy statement/prospectus of BAC. After the registration statement is declared effective, BAC will mail a definitive proxy statement/prospectus relating to the proposed Business Combination to its shareholders. This Presentation is not intended to constitute the basis of any voting or investment decision in respect of the Business Combination or the securities of BAC. BAC’s and the Target’s respective shareholders and other interested persons are advised to read, when available, the preliminary proxy statement/prospectus and the amendments thereto and the definitive proxy statement/prospectus and other documents filed in connection with the proposed Business Combination, as these materials will contain important information about BAC, the Target and the Business Combination. Shareholders will also be able to obtain copies of the preliminary proxy statement/prospectus, the definitive proxy statement/ prospectus and other documents filed with the SEC, without charge, once available, at the SEC’s website at www.sec.gov, or by directing a request to: Biotech Acquisition Company, 545 West 25th Street, 20th Floor, New York, NY 10001.

No Offer or Solicitation: This Presentation shall not constitute a “solicitation” as defined in Section 14 of the Exchange Act. This Presentation does not constitute (i) a solicitation of a proxy, consent or authorization with respect to any securities or in respect of the Business Combination or (ii) an offer to sell, a solicitation of an offer to buy, or a recommendation to purchase any security of BAC, the Target, or any of their respective affiliates nor shall there be any sale of securities, investment or other specific product in any jurisdiction in which such offer, solicitation or sale would be unlawful prior to registration or qualification under the securities laws of any such jurisdiction.

NEITHER THE SEC NOR ANY STATE SECURITIES COMMISSION HAS APPROVED OR DISAPPROVED OF THE SECURITIES OR DETERMINED IF THIS PRESENTATION IS TRUTHFUL OR COMPLETE.

Disclaimer and Other Important Information (cont'd)

Certain statements included in this Presentation are not historical facts but are forward-looking statements. Forward-looking statements generally are accompanied by words such as "believe," "may," "will," "estimate," "continue," "anticipate," "intend," "expect," "should," "would," "plan," "future," "outlook," and similar expressions, but the absence of these words does not mean that a statement is not forward-looking. These forward-looking statements include, but are not limited to, statements regarding estimates and forecasts of other performance metrics and projections of market opportunity. These statements are based on various assumptions, whether or not identified in this Presentation, and on the current expectations of the Target's management and are not predictions of actual performance. These forward-looking statements are provided for illustrative purposes only and are not intended to serve as, and must not be relied on by any person as, a guarantee, an assurance, a prediction or a definitive statement of fact or probability. Actual events and circumstances are difficult or impossible to predict and will differ from assumptions. Many actual events and circumstances are beyond the control of BAC and the Target. Some important factors that could cause actual results to differ materially from those in any forward-looking statements could include changes in domestic and foreign business, market, financial, political and legal conditions.

These forward-looking statements are subject to a number of risks and uncertainties, including the ability to protect and enhance the Target's respective corporate reputation and brand; the impact from future regulatory, judicial, and legislative changes in the Target's or the Company's industry; the timing, costs, conduct, and outcome of clinical trials and future preclinical studies and clinical trials, including the timing of the initiation and availability of data from such trials; the timing and likelihood of regulatory filings and approvals for product candidates; whether regulatory authorities determine that additional trials or data are necessary in order to obtain approval; the potential market size and the size of the patient populations for product candidates, if approved for commercial use, and the market opportunities for product candidates; the ability to locate and acquire complementary products or product candidates and integrate those into the Company's business; and, the uncertain effects of the COVID-19 pandemic; and those factors set forth in documents of BAC filed, or to be filed, with SEC. The foregoing list of risks is not exhaustive.

If any of these risks materialize or our assumptions prove incorrect, actual results could differ materially from the results implied by these forward-looking statements. There may be additional risks that are presently unknown or that the Target currently believes are immaterial that could also cause actual results to differ from those contained in the forward-looking statements. The data contained herein are derived from various internal and external sources. No representation is made as to the reasonableness of the assumptions made within or the accuracy or completeness of any projections or modeling or any other information contained herein. Any data on past performance or modeling contained herein are not an indication as to future performance.

Recipients of this Presentation should conduct their own independent evaluation and due diligence of the Target. The Target does not intend to update or otherwise revise this Presentation following its distribution and the Target makes no representation or warranty, express or implied, as to the accuracy or completeness of any of the information contained in this Presentation after the date of the Presentation.

Committed to Developing Cutting-Edge Treatments for Patients with Rare Progressive Diseases

Fibrosis
Idiopathic Pulmonary Fibrosis

5-year survival rate of 50%[1]

Median survival time of 3-5 years following diagnosis[2]

Neurodegeneration
Huntington's Disease & SCA3

Autosomal dominant, inherited progressive loss of neuronal function

Complications typically cause death 10-30 years after onset[3,4]

1. American Thoracic Society, General Information About Pulmonary Fibrosis.
2. Lederer, 2018, NEJM.
3. Bates, 2015 Nat Rev Dis Primers.
4. Klockgether, 2019, Nat Dis Primers.

Blade Therapeutics Investment Thesis

Orphan Diseases

Pursuing debilitating, progressive diseases in fibrosis and neurodegeneration

Differentiated Pipeline

Oral, small-molecule candidates with novel mechanisms of action and strong IP complemented by global commercial rights

Large Unmet Need

40-75%

of patients with IPF remain untreated with current therapies[1,2,3]

Significant Market

Strong projected growth in IPF market

~$4.0B → **~$8.8B**

(2021)[4] (2027 estimate)[5]

Phase 2 in IPF

Lead program targeting clinically-validated pathway for IPF as potential monotherapy or add-on to current therapies

$280M Proposed Valuation

compared to peer companies, prior M&A deals and external IPF market potential

BLADE THERAPEUTICS

1. Dempsey, 2021, Ann Am Thor Soc.
2. Lancaster, 2021, Respirol.
3. Maher, 2017, BMC Pulm Med.

4. Combined 2021 sales of OFEV (nintedanib) and ESBRIET (pirfenidone), as reported by Boehringer-Ingelheim and Roche, respectively, and converted to USD using "Yearly Average Currency Exchange Rates" on IRS.gov.
5. iHealthcareAnalyst, Inc. (press releasee Nov. 5, 2021).

Biotech Acquisition Company Overview

- Nasdaq-listed SPAC (Nasdaq: BIOT) completed $230 million IPO on January 28, 2021
- SPAC affiliated with SPRIM, a global healthcare consulting firm and clinical research organization
- SPRIM Global Investments is a leading life sciences venture capital firm with in-depth understanding of clinical-stage biotech companies

Competitive Differentiation

Deep industry and life science experience

Decades of diverse experience operating businesses and driving value creation across 17 countries

Management team has worked together for more than 20 years and has strong record of working with clinical-stage biotech companies

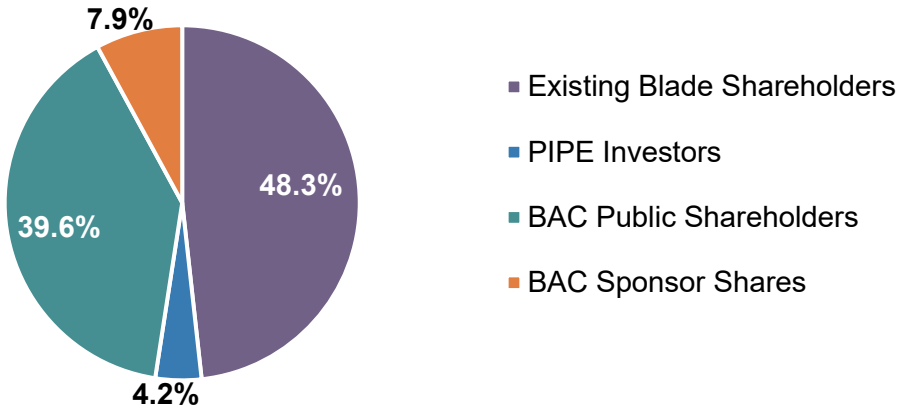
SPRIM

Transaction Overview

Post-Money Valuation

PF Transaction ($mm or mm, except share price)	
Combined Company Share Price	$10.00
PF Shares Outstanding[1][2]	58.0
Total Equity Value	**$580.3**
Less: Pro Forma Cash[3][4]	(230.3)
Plus: Debt[3]	2.8
Total Enterprise Value	**$352.8**

Pro Forma Ownership



Transaction Sources and Uses

Sources ($mm)	
Blade Shareholder Equity Rollover	$280.0
BAC Cash in Trust[4]	230.0
PIPE[5]	24.3
Total Sources	**$534.3**

Uses ($mm)	
Equity Issued to Blade Shareholders	$280.0
Cash to Balance Sheet[4]	$229.3
Estimated Transaction Expenses	$25.0
Total Uses	**$534.3**

(1) Assumes 28.0 million shares issued to Blade's existing shareholders (with no portion of the merger consideration rolled into assumed in-the-money options), approximately 2.4 million PIPE shares, 23.0 million BAC public shares, and 4.6 million founder shares. Assumes no redemptions by BAC's existing shareholders. Excludes the impact of 6.0 million BAC private placement warrants and 11.5 million BAC public warrants.

(2) Excludes 3.5 million Blade earn-out shares not yet issued (to be issued to Blade if the VWAP of BAC is greater or equal to $15.00 over 20 trading days within any 30 trading days within 5 years after close) and any awards to be issued under an expected new equity incentive plan. Founder shares exclude 1.15 million previously issued shares that will be placed in escrow (to be released to the sponsor if the VWAP of BAC is greater or equal to $15.00 over 20 trading days within any 30 trading days within 5 years after close). Assumes PIPE shares are issued at a price of $10.00.

(3) Blade estimated closing cash balance of $1mm and estimated closing debt balance of $2.8mm

(4) Assumes no redemptions by BAC's existing shareholders.

(5) Consists of existing Blade investors.

Developing Cutting-Edge Treatments for Debilitating Fibrotic and Neurodegenerative Diseases

Experts in Biology of Cell and Tissue Damage Responses

- Researching novel pathways foundational to cell- and tissue-damage responses
- Developing potential disease-modifying therapeutics

Differentiated Pipeline Led By Phase 2 Program in Fibrosis

- Non-competitive autotaxin inhibitor with direct anti-fibrotic activity and differentiating characteristics[1] – planned phase 2 study in lung fibrosis in 2Q-2022
- CNS-penetrant calpain inhibitor[1] for genetic orphan neurodegenerative conditions – planned phase 1 study in 2H-2022[2]

Deep Scientific & Industry Experience

- Experienced team with extensive expertise in fibrosis and neurodegeneration
- Strong track record of development and approvals of innovative medicines

1. Effects demonstrated in preclinical studies.
2. Pending regulatory review and feedback (e.g., FDA or ex-US regulatory authorities).

Leadership with Deep Scientific and Industry Experience

Wendye Robbins, M.D.
Chief Executive Officer

RAPT THERAPEUTICS · THIRD ROCK VENTURES · Stanford University

Jean-Frédéric Viret, Ph.D.
Chief Financial Officer

Coherus BIOSCIENCES · Tularik

Felix Karim, Ph.D.
EVP, Business Development

K A I PHARMACEUTICALS · AMGEN

Bassem Elmankabadi, M.D.
SVP, Clinical Development

FibroGen · AMGEN · UCLA Health

Daven Mody, PharmD
SVP, Regulatory Affairs

Theravance Biopharma · Johnson & Johnson · Impax

Prabha Ibrahim, Ph.D.
Chief Technical Officer

AMGEN · MERCK · Plexxikon

Michael Blash
SVP, Communications

MERCK · The Medicines Company

Developing Therapies to Target Key Pathways in Disease Progression

Target Pathway	Potential Treatment Effects	Diseases
Autotaxin (ATX)	Non-competitive, reversible inhibition supports potential for differentiated profile in fibrotic diseases and epithelial tumors	IPF, SSc-ILD NASH Oncology
Dimeric Calpains (CAPN)	Inhibition shown to enhance autophagy and reduce protein aggregates in preclinical models	Huntington's SCA3
	Inhibition blocks myofibroblast activation / differentiation, thereby inhibiting extracellular matrix production, in preclinical models	IPF, ILD NASH

Fibrotic diseases – idiopathic pulmonary fibrosis (IPF), systemic sclerosis – interstitial lung disease (SSc-ILD), non-alcoholic steatohepatitis (NASH)

Neurodegenerative diseases – Huntington's disease (HD), spinocerebellar ataxia type 3 (SCA3, aka Machado-Joseph disease or MJD)

Differentiated Pipeline of Novel Product Candidates

Compound & MoA	Indication	Est. Prevalence	PC	Ph1	Ph2	Ph3	Patent Expiry
Fibrosis							
Cudetaxestat (BLD-0409) Non-Competitive Autotaxin Inhibitor	Idiopathic Pulmonary Fibrosis (IPF)*	<200K (US) ~5M (WW) Orphan Disease					**2034 – 2036**
	Systemic Sclerosis – Interstitial Lung Disease (SSc-ILD)*	~47K (N. America) ~400-500K (WW) Orphan Disease					
	Liver Fibrosis	~16M (US) up to 505M (WW) *(NASH Prevalence)*					
Neurodegeneration							
BLD-2184 Calpain Inhibitor	Poly-Q (HD, SCA3)	Orphan Diseases					**2037 – 2040**

* Orphan drug designations granted by FDA for IPF and for SSc.

Multiple Milestones

	Completed and Potential Milestones		Timing
✓	Cudetaxestat	Phase 1 CYP-DDI study completed	Nov-2021
✓	Cudetaxestat	Phase 1 DDI study with approved IPF therapies completed*	Jan-2022
✓	Cudetaxestat	End-of-phase 1 meeting with FDA	1Q-2022
✓	Cudetaxestat	American Thoracic Society presentations	May-2022
	Corporate	Complete merger with Biotech Acquisition Company (Nasdaq: BIOT)	2Q-2022
	Cudetaxestat	Initiate planned phase 2 study in IPF	2Q-2022
	BLD-2184	Initiate planned phase 1 study in healthy volunteers**	2H-2022
	Cudetaxestat	13-week blinded interim analysis for planned phase 2 study in IPF	Mid-2023
	BLD-2184	Data read out for planned phase 1 study in healthy volunteers**	1H-2023
	Cudetaxestat	Completion of planned phase 2 study in IPF	1Q-2024

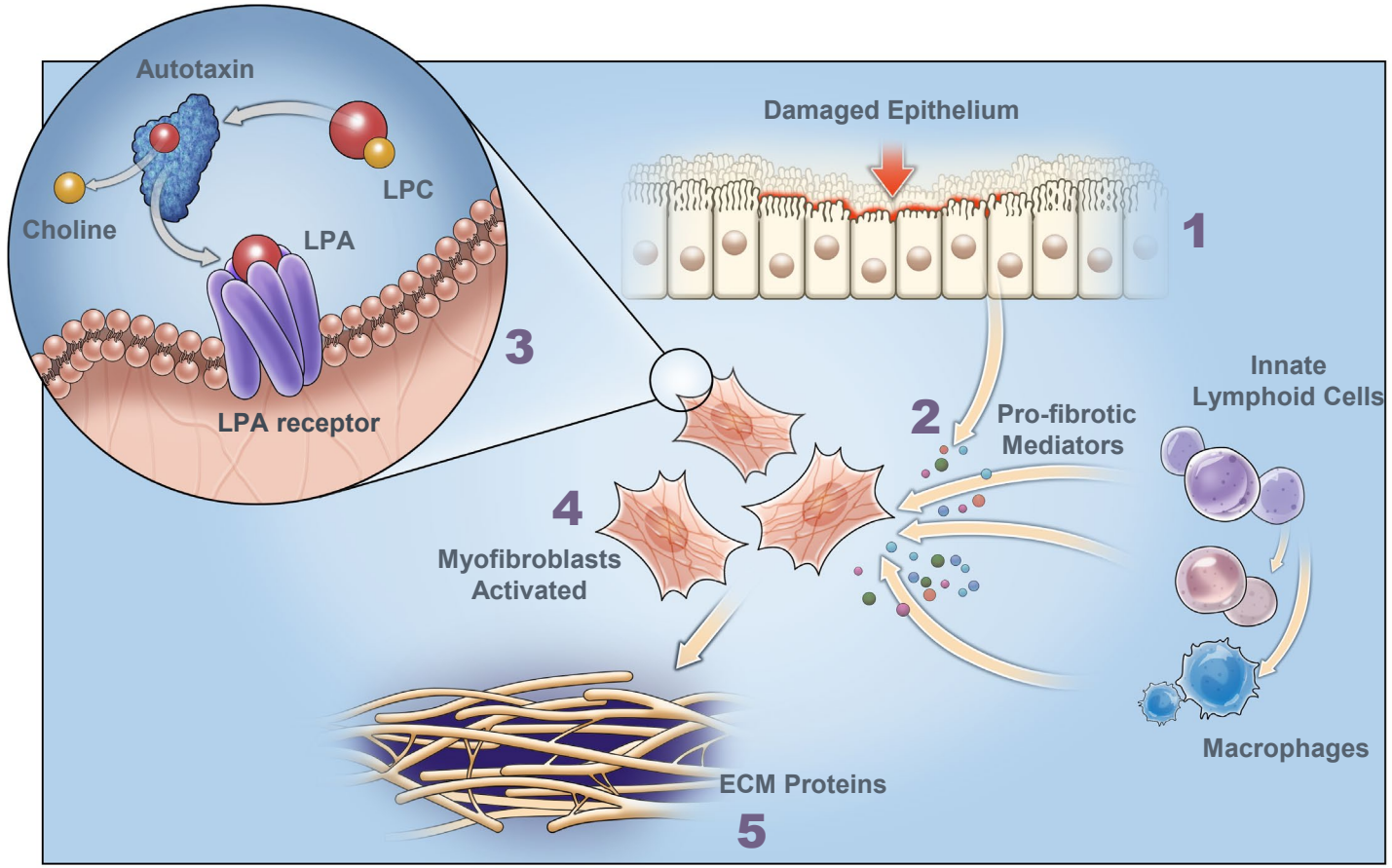
BLADE THERAPEUTICS

* Pirfenidone and nintedanib.
** Pending regulatory review and feedback (e.g., FDA or ex-US regulatory authorities).
✓ Indicates completed milestone.

Fibrosis – Cudetaxestat

Non-Competitive Autotaxin Inhibitor Targeting IPF

Autotaxin / Lysophosphatidic Acid (LPA) Drives Fibrosis



1 Dysregulated Damage Response
Fibrosis is triggered by dysregulated cell / tissue damage response following epithelial injury.

2 Release of Pro-fibrotic Mediators
Pro-fibrotic mediators, cytokines and the enzyme autotaxin are released. Increased autotaxin levels produce excessive lysophosphatidic acid (LPA).

3 Autotaxin Production of LPA
LPA binds to LPAR1 (receptor on myofibroblasts) and triggers signaling cascade resulting in migration, activation and release of additional mediators.

4 Myofibroblast Activation
Excessive LPA activates myofibroblasts.

5 Secretion of ECM Proteins
Activated myofibroblasts secrete ECM proteins (scarring) that disrupt normal organ architecture and function.

Cudetaxestat – Phase 2 Lead Program Targeting IPF

Progressing Toward Planned Phase 2 IPF Study

Cudetaxestat to be dosed as monotherapy or co-administered with pirfenidone or nintedanib

Extensive Phase 1 Clinical Program

Phase 1 data demonstrated PK/PD correlation and biomarker activity; well tolerated with no drug-related SAEs in 200+ healthy volunteers

Direct Anti-Fibrotic Activity

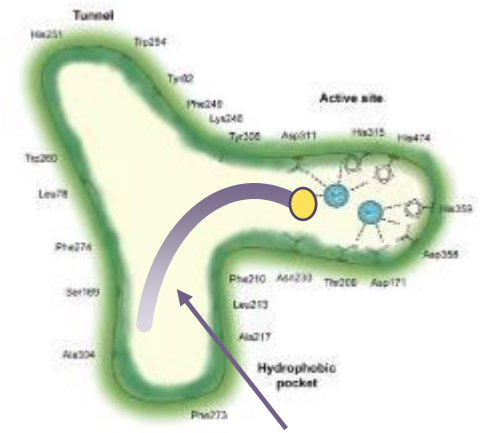
Robust *in vivo* anti-fibrotic activity in preclinical models of lung and liver fibrosis

Non-Competitive Autotaxin Inhibition

Differentiating characteristics support potential treatment profile in fibrosis

Non-Competitive Inhibition Supports Differentiated Profile in Fibrosis

ATX Tripartite Active Site

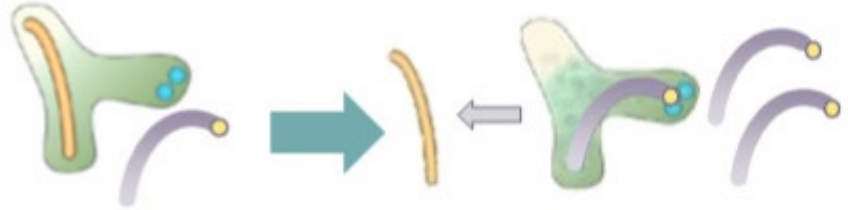


Substrate binding

Enzyme Products: LPA + Choline

Adapted from Salgado-Polo, 2019, Cancers.

High substrate (LPC) levels seen in diseased tissues and organs

Non-Competitive Inhibitor	**Competitive Inhibitor**
Does **not** compete with LPC for binding	Competes with LPC for binding
	
Potent inhibitor results in **no loss in potency**	Inhibition plateaus and results in **reduced potency**

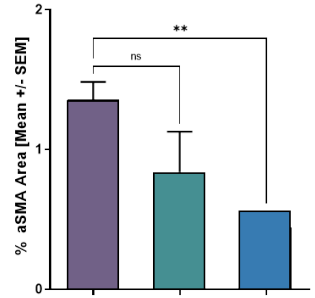
- Non-competitive inhibitor cudetaxestat shown to maintain potency in preclinical biochemical assay
  - Cudetaxestat expected to maintain potency in disease state
  - Potentially advantageous for efficacy and safety profile
- 50% inhibition at 250 μM concentration of LPC*
  - Cudetaxestat: 8 nM
  - GLPG-1690: ~400 nM (competitive inhibitor loses potency)

* Plasma concentrations of LPC in an animal model of liver fibrosis are between 200-250 μM.
Head-to-head clinical studies comparing cudetaxestat with currently approved or investigational therapies have not been conducted. Above data from preclinical biochemical study.

Cudetaxestat Displays Robust Activity (*in vivo*) on Lung Fibrosis Biomarkers and Pro-Fibrotic Genes

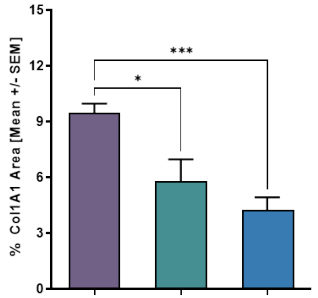
SMA gene expression

Marker of myofibroblast activation



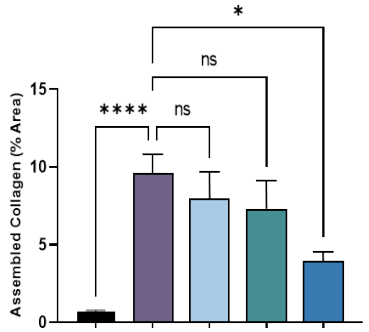
Collagen 1A1 gene expression

Primary component of fibrosis, produced by myofibroblasts



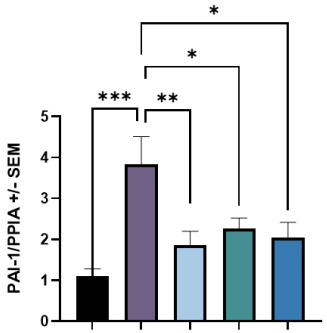
Assembled collagen

Represents advanced fibrosis



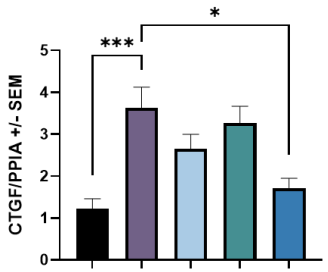
PAI-1 gene expression

Contributes to excessive ECM accumulation



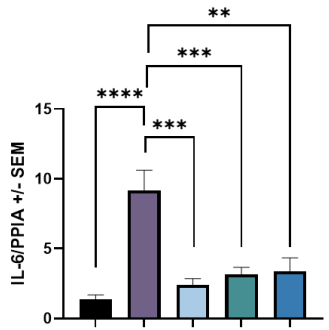
CTGF gene expression

Pro-fibrotic growth factor



IL-6 gene expression

Pro-fibrotic cytokine



Saline
Vehicle
Cude 3mg/kg
Cude 10mg/kg
Cude 30mg/kg

ANOVA One-Way

**p<0.05*

***p<0.01*

****p<0.001*

*****p<0.0001*

SMA (smooth muscle actin), collagen 1A1 and assembled collagen data from preclinical study conducted in mouse bleomycin lung fibrosis model dosed once daily.
PAI-1, IL-6 and CTGF (connective tissue growth factor) data from preclinical study conducted in mouse bleomycin lung fibrosis model dosed twice daily.

Completed Preclinical Studies Support Safety Profile of Cudetaxestat

	Completed Studies	Findings
Toxicology	Rat 4-week GLP tox	NOAEL 100 mpk
	Dog 4-week GLP tox	NOAEL 300 mpk
	Rat 26-week GLP tox	NOAEL 30 mpk (male) / 75 mpk (female)
	Dog 39-week GLP tox	NOAEL 300 mpk
Safety Pharmacology	Dog cardiovascular	No adverse findings; NOAEL 1000 mpk
	Rat respiratory	No adverse findings; NOAEL 750 mpk
	Rat Irwin	No adverse findings; NOAEL 750 mpk
	hERG	*in vitro* IC_{50} = 49.4 µM (corrected for plasma protein binding)
Gene Tox	Ames	Negative
	In vitro micronucleus	Negative
Drug-Drug Interactions	*In vitro* and *in vivo* profiling vs. approved IPF therapies (pirfenidone, nintedanib)	Unlikely DDI potential with approved IPF therapies

Consistent Tolerability Demonstrated in Extensive Phase 1 Clinical Program

	Study 1 SAD/MAD (N=80)	Study 2 Relative Bioavailability (N=34)	Study 3 CYP-DDI* (N=16)	Study 4 DDI with Approved IPF Therapies (N=83)
Objective	Evaluated safety, tolerability, PK and PD of single and multiple ascending doses of cudetaxestat to facilitate dose/dosing regimen selection for future studies	Investigated relative bioavailability of new tablet formulation of cudetaxestat to oral solution formulation	Evaluated effect of cudetaxestat on PK of a combination of probe substrates for CYP450 enzymes	Assessed effect of cudetaxestat on PK of two approved drugs for IPF (pirfenidone and nintedanib)
Summary Findings	• PK/PD correlation with sustained target engagement (LPA) • Data support clinical once-daily dose up to 750 mg or 500 mg BID (fed)	• Comparable PK and PD profile between the two formulations	• No alteration of plasma levels of substrates for CYP3A4, 2B6, 1A2, and 2C9; weak inhibition of CYP2D6 and induction of CYP2C19	• No significant DDI seen with cudetaxestat in combination with either pirfenidone or nintedanib

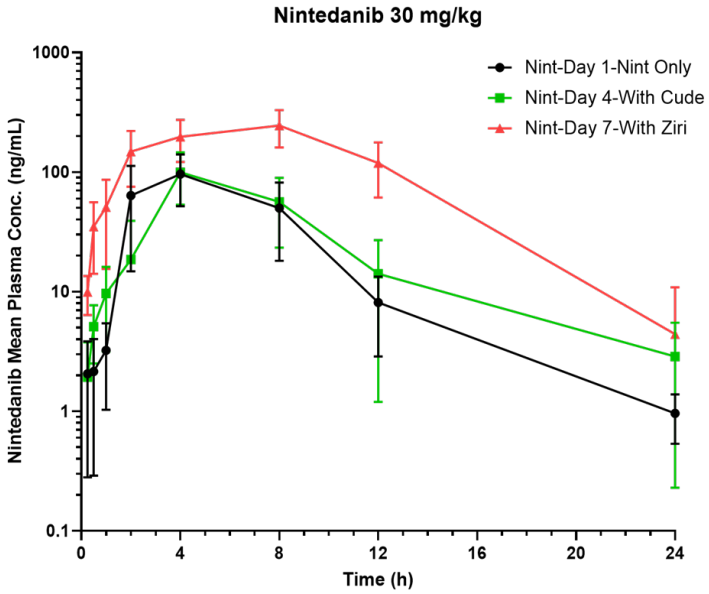
Cudetaxestat well tolerated with no reports of drug-related serious adverse events across all Phase 1 studies in healthy volunteers

All Phase 1 studies outlined above were conducted in healthy volunteers.
* “Drug-drug interaction” (DDI)

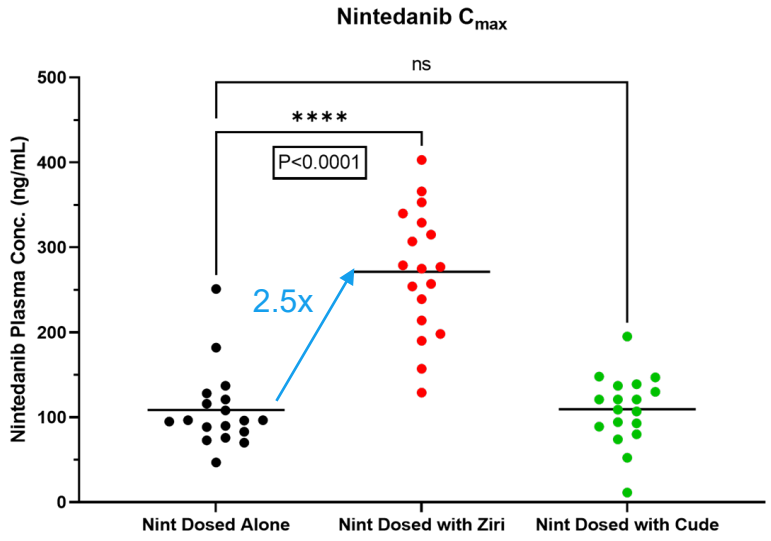
Nintedanib Exposure Comparison ± Cudetaxestat or GLPG-1690 (preclinical model)

- Nintedanib C_{max} and AUC
  - Increased when co-administered with GLPG-1690 by **2.5X and 4X** respectively (p<0.0001)
  - No statistically significant change when co-administered with cudetaxestat

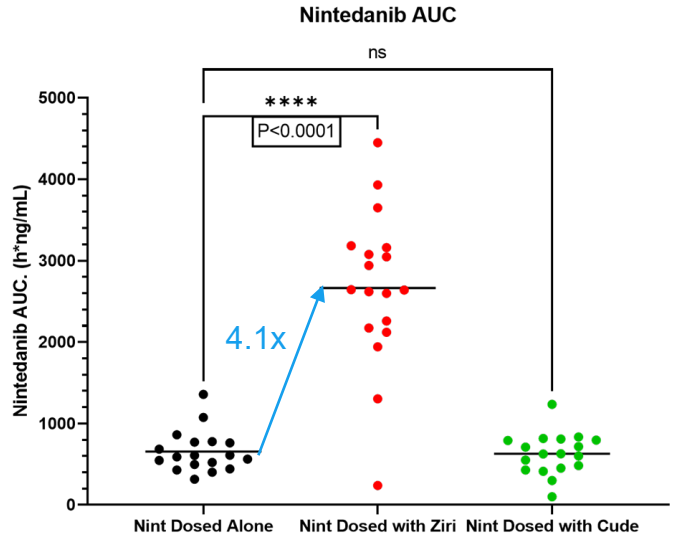
Nintedanib Plasma Conc. vs Time



Nintedanib Cmax ± Cude/GLPG



Nintedanib AUC ± Cude/GLPG



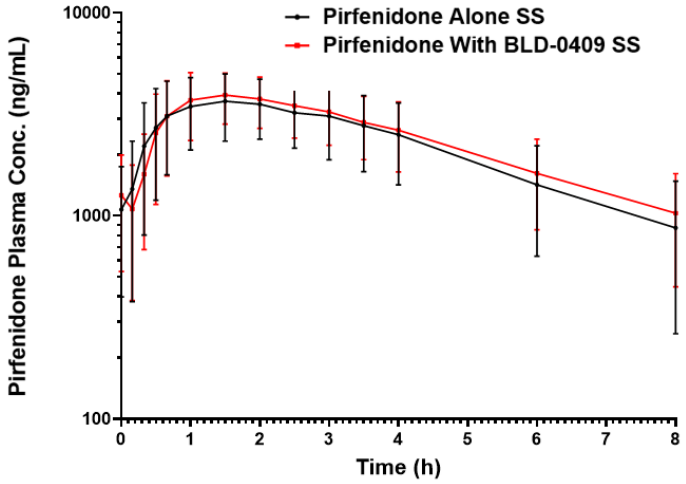
Preclinical study conducted in rat model.
W. Yu et al. "Preclinical Evaluation of Cudetaxestat (BLD-0409) for Potential Drug-Drug Interactions (DDI's)" poster presentation at American Thoracic Society 2022 International Conference, May 16, 2022.

Positive Findings from Phase 1 Drug-Drug Interaction (DDI) Study of Cudetaxestat in Healthy Volunteers

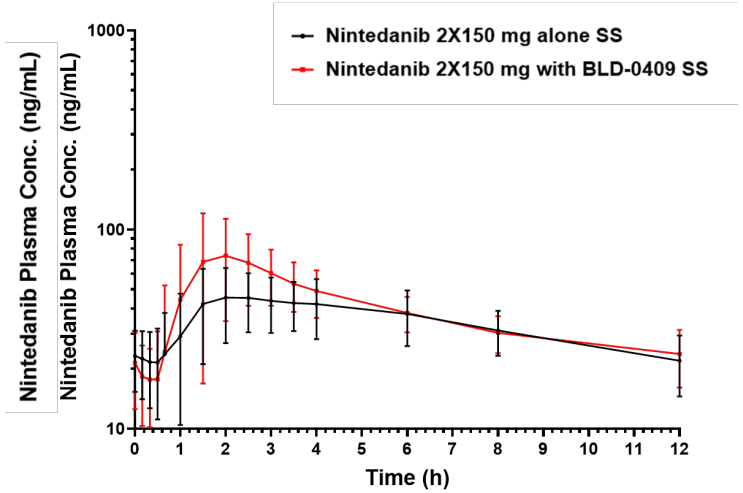
Key Findings

- No significant DDI when cudetaxestat co-administered with either pirfenidone or nintedanib
- Neither nintedanib nor pirfenidone significantly alter the exposure of cudetaxestat
- Most common TEAEs were mild GI upset (diarrhea and nausea), with no treatment-related SAEs

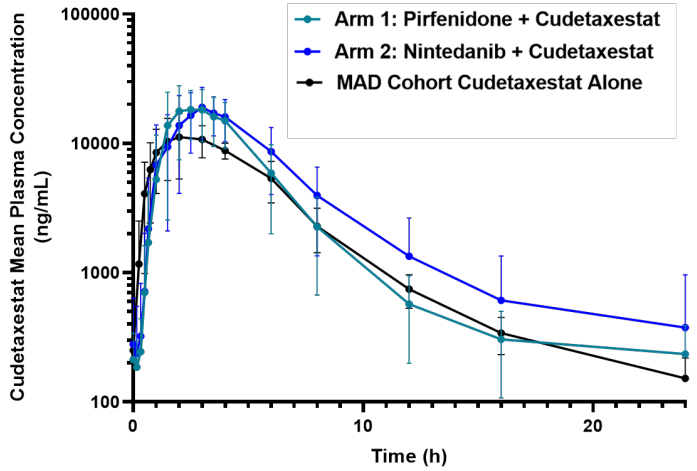
Pirfenidone Plasma Concentrations vs Time with & without Cudetaxestat (Mean ± SD)



Nintedanib Plasma Concentration vs Time with & without Cudetaxestat (Mean ± SD)



Cudetaxestat Plasma Concentration vs Time with Pirfenidone or Nintedanib (Mean ± SD)



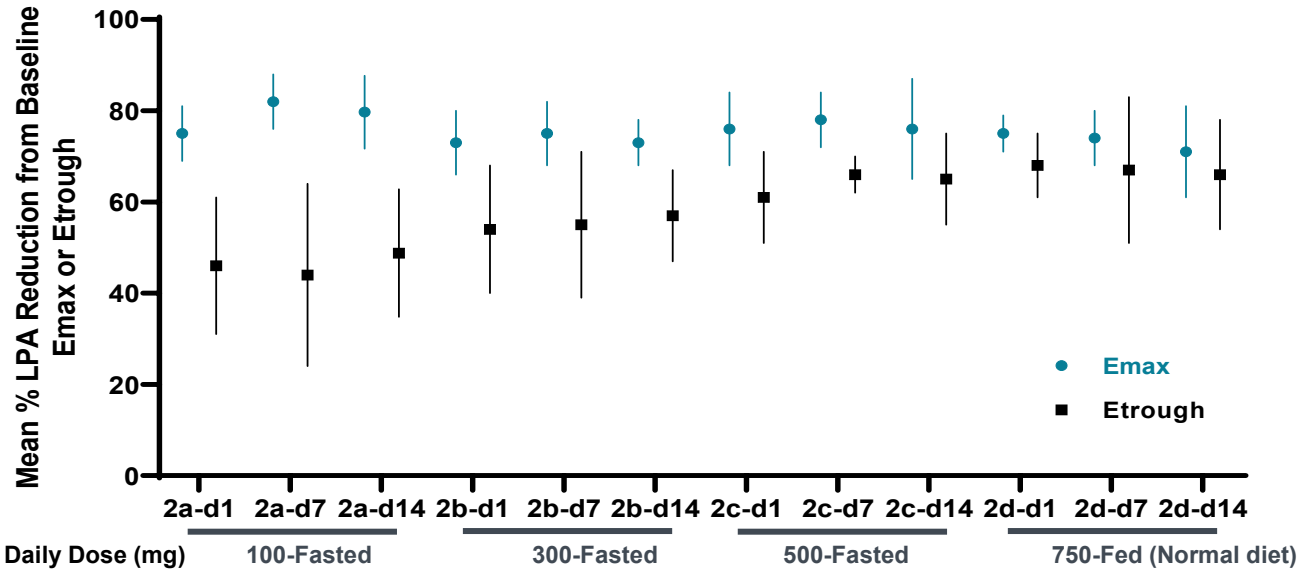
D. Mody et al. "Clinical Evaluation of Cudetaxestat for Safety, Tolerability, Pharmacokinetics, Pharmacodynamics, and Potential Drug-Drug Interactions" poster presentation at American Thoracic Society 2022 International Conference, May 16, 2022.

Cudetaxestat Phase 1 Study Demonstrated Tolerability, PK and Biomarker Activity

- 6 single ascending dose (SAD) cohorts completed; 100, 300, 500, 750, 1000 mg
- 4 multiple ascending dose (MAD) cohorts completed; 100, 300, 500, 750 mg (fed)
- Demonstrated PK/PD correlation in healthy volunteer MAD
- Data support clinical once-daily dose up to 750 mg or 500 mg BID (fed)
- Well-tolerated at target doses with no treatment-emergent serious adverse events (SAEs)

Peak-to-trough variation in LPA reduction

(Emax and Etrough)



D. Mody et al. "Clinical Evaluation of Cudetaxestat for Safety, Tolerability, Pharmacokinetics, Pharmacodynamics, and Potential Drug-Drug Interactions" poster presentation at American Thoracic Society 2022 International Conference, May 16, 2022.

RESPIRARE Phase 2 Study of Cudetaxestat in Patients with Idiopathic Pulmonary Fibrosis

Summary: Randomized, double-blinded, placebo-controlled study of investigational therapy cudetaxestat with or without approved therapy for idiopathic pulmonary fibrosis (IPF)

Number of Global Sites Planned: ~90

2Q-2022

SCREENING / ENROLLMENT

Patient Population

Patients with IPF* treated with or without approved IPF therapy (nintedanib or pirfenidone)

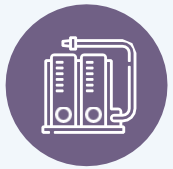
TREATMENT (N=~200)

- Cudetaxestat 250mg QD (n = ~50)
- Cudetaxestat 500mg QD (n = ~50)
- Cudetaxestat 500mg BID (n = ~50)
- Matching placebo (n = ~50)

26-week duration

Blinded Interim Analysis (13 weeks to assess biomarkers)

END OF TREATMENT



Primary Endpoint

Change in forced vital capacity lung volume measured in milliliters (mL) and assessed by clinic spirometry throughout the study duration

1Q-2024

FOLLOW-UP

Safety Assessments

4 weeks
+/- 3 days

BLADE THERAPEUTICS

* ATS, ERS, JRS, ALAT criteria

Cudetaxestat Phase 2 Study Summary Design in IPF

Primary Endpoints	• Change in Forced Vital Capacity (FVC) (L) from baseline at Week 26, assessed by central spirometry
Secondary Endpoints*	• Time to disease progression, defined as absolute percent predicted FVC (FVCpp) decline of ≥10% or death, whichever occurs first • Time to disease progression, defined as relative percent predicted FVC (FVCpp) decline of ≥5% or death, whichever occurs first • Change in absolute ppFVC from baseline to W26, assessed by central spirometry • Time to first acute IPF exacerbation during study (adjudicated) • Time to all-cause mortality during study (adjudicated) • Time to first respiratory hospitalization during study (adjudicated) • Change in Quantitative Lung Fibrosis (QLF) volume from baseline at Week 26 assessed by high-resolution computed tomography (HRCT)
Exploratory Endpoints*	• Percent change from baseline in LPA, PRO-C3 and PRO-C6
Blinded Interim Analysis	• Conduct at 13 weeks to assess biomarkers (LPA target engagement)
Safety*	• Treatment-emergent adverse events • Assessment of vital signs, clinical laboratory parameters, and electrocardiograms

* Select adjudicated endpoints and assessments noted.

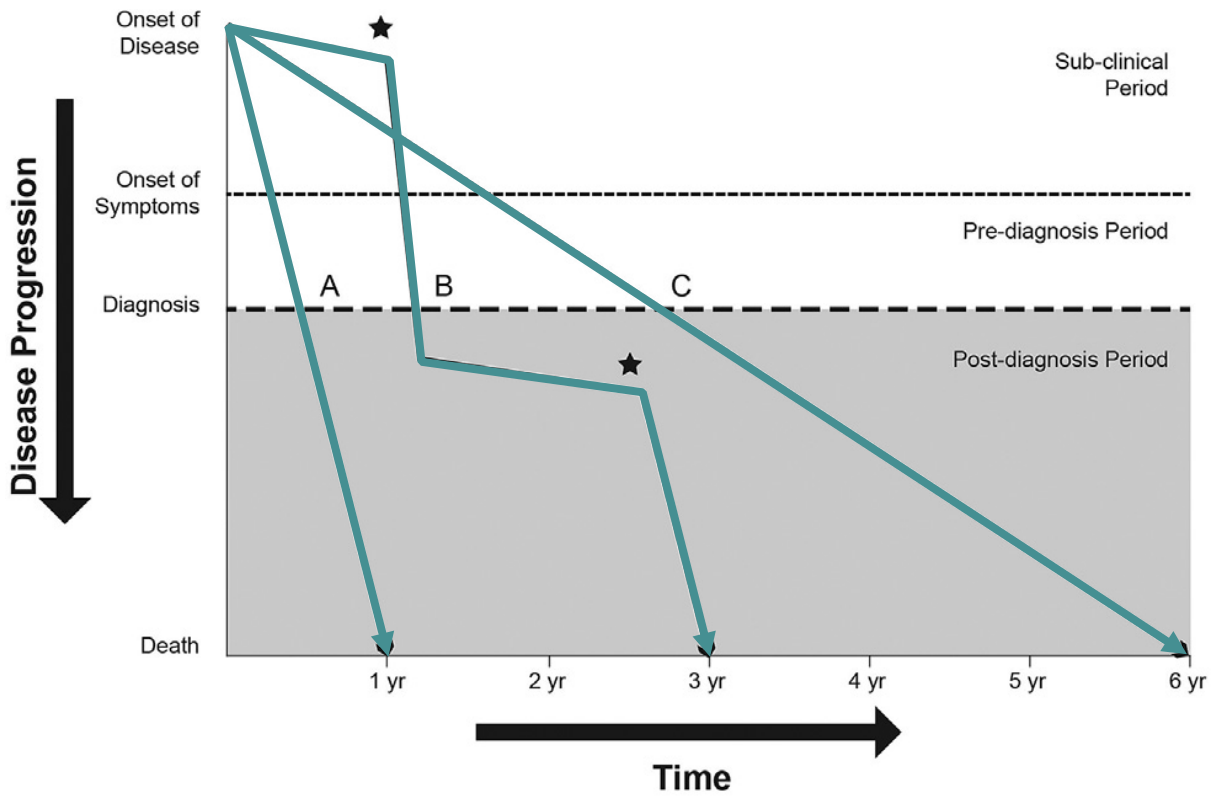
Cudetaxestat – Well-Defined Development Pathway in Lung Fibrosis

Completed and Potential Milestones	Timing
✓ **Phase 1 CYP-DDI study completed**	Nov-2021
✓ **Phase 1 DDI study with approved IPF therapies (pirfenidone, nintedanib) completed**	Jan-2022
✓ **End-of-Phase 1 meeting with FDA**	1Q-2022
○ **Initiate planned phase 2 clinical study in IPF**	2Q-2022
• 26-week study (N= ~200) to provide efficacy and safety data	
○ Blinded interim 13-week analysis (from LPI)	Mid-2023
○ Study completion	1Q-2024

Idiopathic Pulmonary Fibrosis Market Landscape and Opportunities

Lung Function Decline is the Hallmark of IPF

- IPF patients have a very poor prognosis
  - 5-year survival rate of 50%[1]
  - Median survival time of 3-5 years following diagnosis[2]
- Disease progression is highly variable
- Acute exacerbations (~5-10% of patients annually) are the leading cause of hospitalization and death[3]



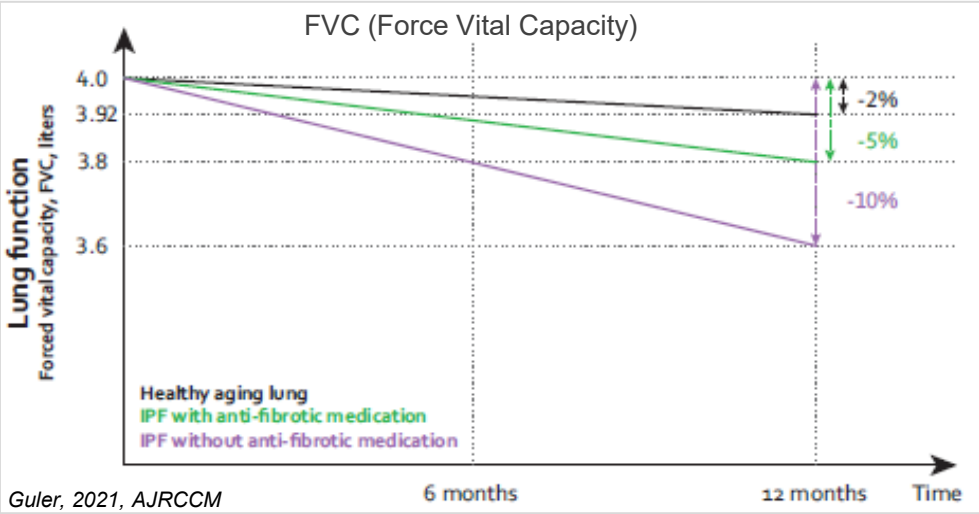
Adapted from Kim, 2015, Respir Med.

1. American Thoracic Society, General Information About Pulmonary Fibrosis.
2. Lederer, 2018, NEJM.
3. Kim, 2015, Respir Med.

Current Approved Anti-Fibrotic Therapies for IPF

Esbriet® (pirfenidone) and Ofev® (nintedanib)

- Only approved (standard of care) anti-fibrotic therapies
- Approved based on slowing the decline of FVC from baseline
  - Reduced FVC decline from baseline by 40 – 55% vs. pbo[1,2]
- Neither is curative nor showed a mortality benefit



Guler, 2021, AJRCCM

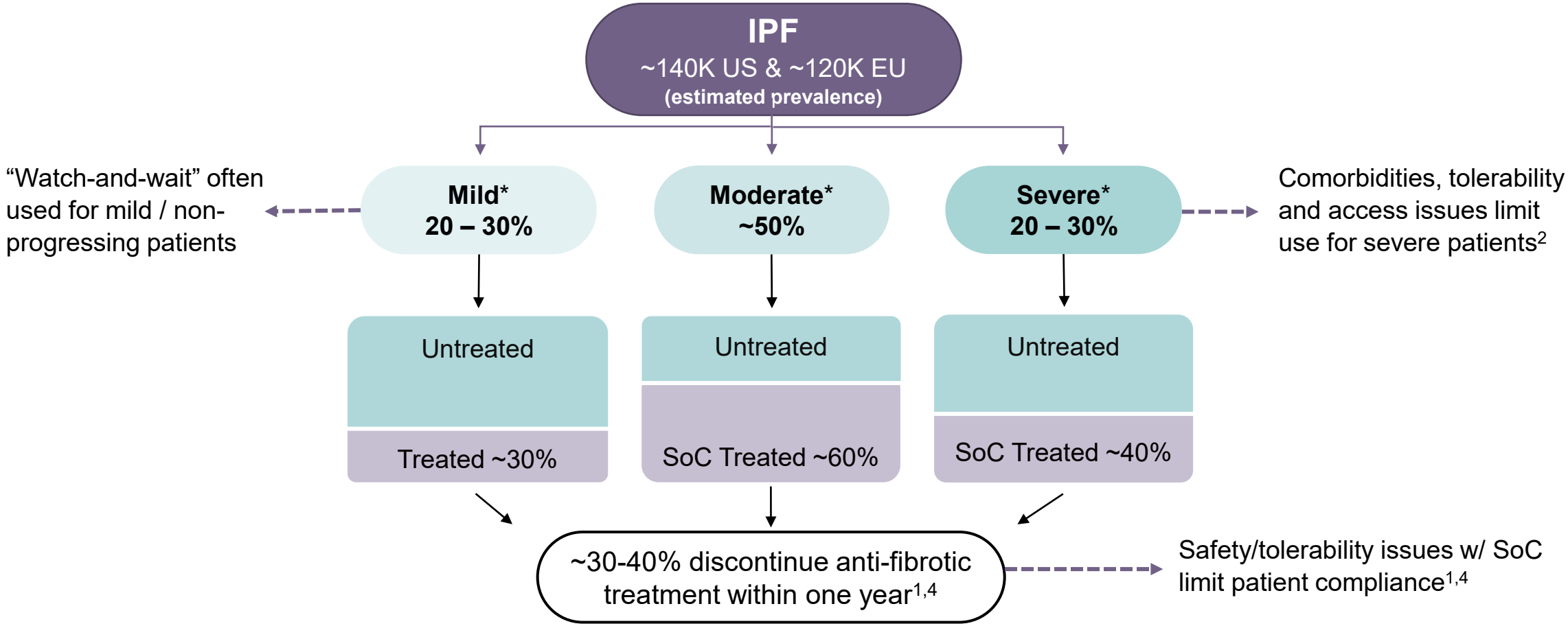
Both treatments have significant safety/tolerability issues[4]

Product	Safety / Tolerability[4]		
	TEAE	treated	placebo
Pirfenidone	– Nausea	36%	16%
	– Rash	30%	10%
	– Dyspepsia	19%	7%
	– Liver enzyme elevation	4%	1%
Nintedanib	– Diarrhea	76%	32%
	– Nausea	32%	14%
	– Vomiting	25%	10%
	– Liver enzyme elevation	13%	3%

1. Noble, 2016, Eur Resp J.
2. Richeldi, 2014, NEJM.
3. Guler, 2021, AJRCCM.
4. Esbriet® (pirfenidone) USPI and Ofev® (nintedanib) USPI.

Current IPF Treatment Paradigm Results in Significant Undertreatment

Real-world studies indicate 40-75% of patients remain untreated with SoC (pirfenidone or nintedanib)[1,2,3]



Note: *Figure adapted from real-world data – see references cited below*

**No official standard exists for classifying disease severity in IPF. Physician-based judgments are highly subjective but do fall into frequently noted characteristics (e.g., Mild = ppFVC > 80%; Moderate* = ppFVC 55-80%; Severe = ppFVC <55%)

1. Dempsey, 2021, Ann Am Thor Soc.
2. Lancaster, 2021, Respirol.
3. Maher, 2017, BMC Pulm Med.
4. Wright, 2021, BMJ Open Respir Res.

Significant Addressable Market Opportunity in IPF

Current IPF Market[1,2,3,4]

~30-40% annual discontinuation[1,4]

Global IPF Market

2021[5]	2027 estimate[6]
~$4.0B	~$8.8B

Market growth driven by increased prevalence and incidence, premium-priced drugs, and approval of new treatments

Annual list pricing for SoC anti-fibrotic therapies (2021)

- Pirfenidone (patent expiry 2026): ~$130K/yr[7]
- Nintedanib (patent expiry 2029): ~$145K/yr[7]

1. Dempsey, 2021, Ann Am Thor Soc.
2. Lancaster, 2021, Respirol.
3. Maher, 2017, BMC Pulm Med.
4. Wright, 2021, BMJ Open Respir Res.
5. Combined 2021 sales of OFEV (nintedanib) and ESBRIET (pirfenidone), as reported by Boehringer-Ingelheim and Roche, respectively, and converted to USD using "Yearly Average Currency Exchange Rates" on IRS.gov.
6. iHealthcareAnalyst, Inc. (press release Nov. 5, 2021).
7. Drugs.com.

IPF – Key Competitors in Clinical Development

Company	Stage	Product	MOA	ROA, Dosing, Molecule	Differentiators	LOE
FibroGen	Ph 3	Pamrevlumab	Anti-CTGF mAb	IV, Q3W mAb	± IV monotherapy only	2035
Roche	Ph 3	PRM-151	Pentraxin-2 protein	IV, Q4W recomb protein	± IV add-on only, no monotherapy activity	2036
United Therapeutics	Ph 3	Treprostinil	Prostacyclin analog	Inhaled, QID small mol	± Inhaled add-on, reduces PAH	2028
Boehringer Ingelheim	Ph3	BI 1015550	phosphodiesterase 4B (PDE4B) inhibitor	PO, BID Small mol	+ Oral mono- or add-on therapy to SoC	Not disclosed
Galecto	Ph 2	GB-0139	Galectin-3 inhibitor	Inhaled, QD small mol	± Inhaled monotherapy only - DDI w/ SoC	2033
Bristol Myers Squibb	Ph 2	BMS-986278	LPA1 antagonist	PO, BID small mol	+ Oral mono- or add-on therapy to SoC	2034
HORIZON	Ph 2	HZN-825	LPA1 antagonist	PO, QD or BID small mol	± Oral monotherapy only	2034
PLIANT	Ph 2	PLN-74809	Integrin αvβ1 / αvβ6 antagonist	PO, QD small mol	+ Oral – add-on status under evaluation in current Ph2a study	2037
BLADE THERAPEUTICS	Ph 2	Cudetaxestat (BLD-0409)	ATX inhibitor (non-competitive/allosteric)	PO, QD or BID small mol	+ Oral mono- or add-on therapy to SoC	2034-2042

Includes all phase 3 programs and key phase 2 programs.

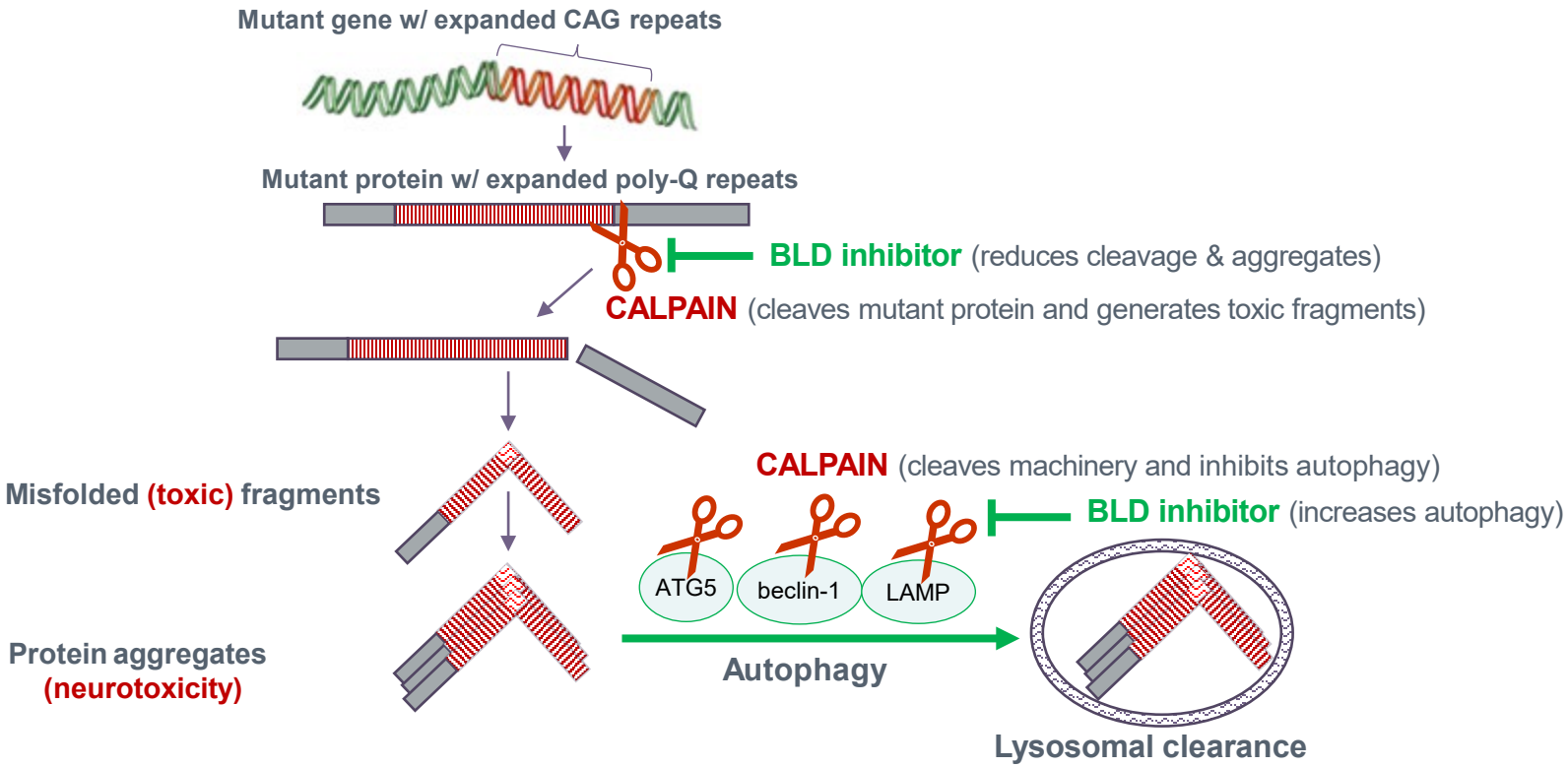
PAH = pulmonary arterial hypertension.
SoC = pirfenidone or nintedanib.

Neurodegeneration – BLD-2184

CNS-Penetrant Calpain Inhibitor for Poly-Q Neurodegenerative Conditions

Calpains Implicated in Progression and Autophagy of Neurodegenerative Diseases

Huntington's Disease (HD) HTT Gene and Spinocerebellar Ataxia Type 3 (SCA3)



Calpain inhibition shown in preclinical models to reduce cleavage of mutant proteins, aggregation and enhances clearance via autophagy

Weber, 2018, Neuropharmacol.
Gafni, 2002, J Neurosci.
Weber, 2019, BioMed Res Int.

Calpain Inhibitors Demonstrate Preclinical Evidence of Neuroprotective Effects

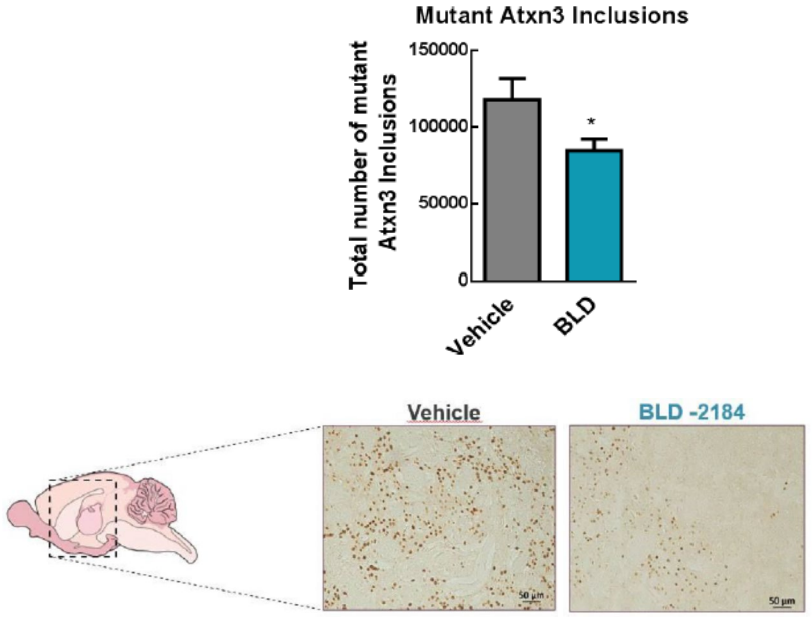
Novel Target for Disease Progression[1]	Calpains shown in preclinical studies to regulate formation of toxic proteins and autophagy (intracellular clearance), key components in incurable neurodegenerative Poly-Q diseases (HD, SCA3)
Preclinical Evidence of Neuroprotection[2]	Improvements in biomarkers, motor function and enhanced autophagy in SCA3 preclinical models (mouse, zebrafish models)
Development Candidate Selection	Ongoing preclinical and nonclinical activities in preparation for initiating planned phase 1 study (2H-2022)

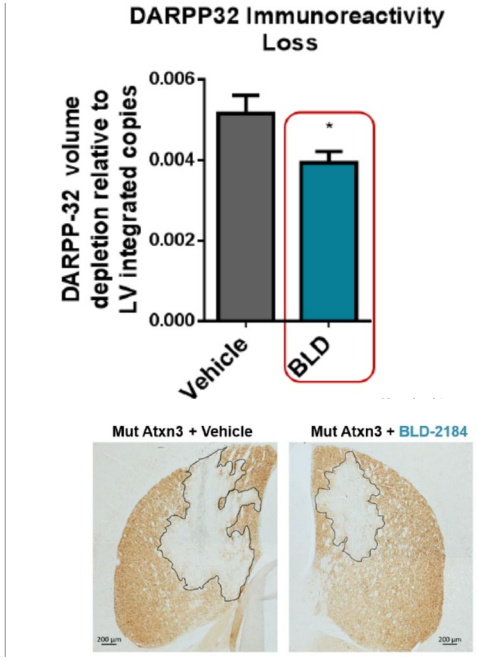
1. Weber, 2018, Neuropharmacol. Gafni, 2002, J Neurosci. Weber, 2019, BioMed Res Int.
2. Robinson, 2021, Cells.

BLD-2184 – Development Candidate for Neurodegenerative Diseases

Neuroprotective Effects in SCA3 Model

Fewer Ataxin-3 inclusions and decreased loss of dopaminergic neurons (mutant hATXN3 lentiviral mouse model)





Potent and active against cysteine proteases in preclinical models

Good oral bioavailability and CNS penetration with long half-life

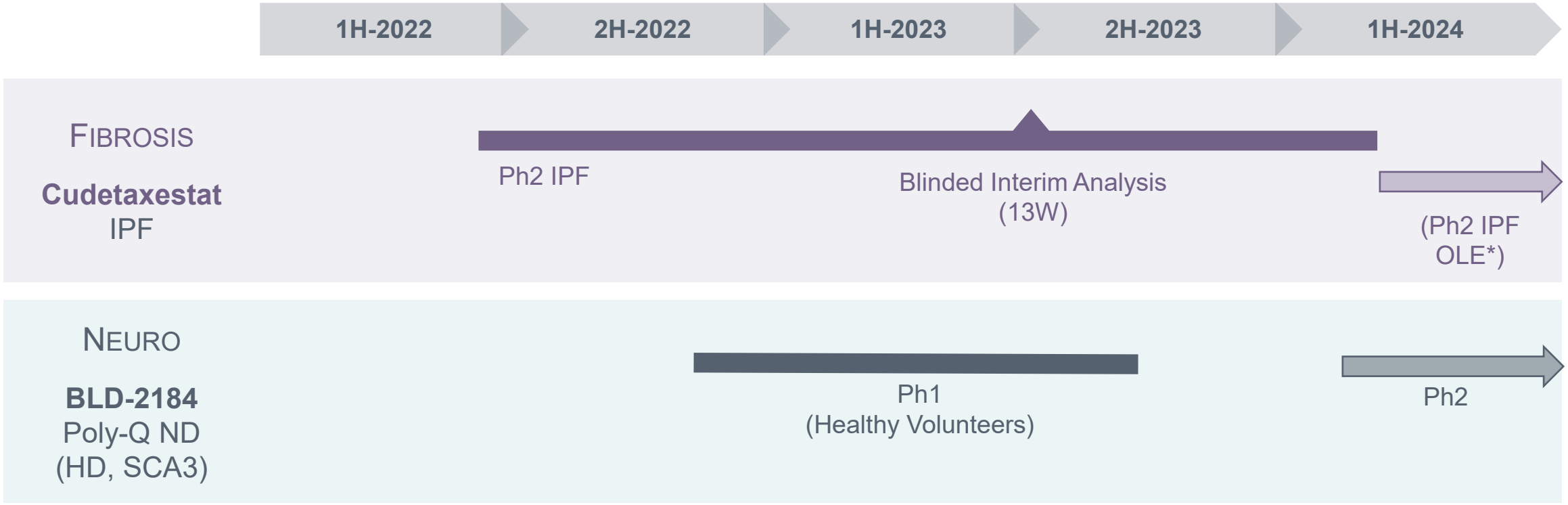
Preclinical evidence in mouse models

Attenuation of disease effects in SCA3 model

IND-enabling studies completed

Phase 1 planned to initiate 2H-2022

Planned Clinical Development in Fibrosis and Neurodegeneration



Note: clinical studies listed above are contingent upon regulatory review and feedback (e.g., FDA and ex-US regulatory authorities).
* Study currently tentative and in early planning.

Blade Therapeutics Investment Thesis

Orphan Diseases

Pursuing debilitating, progressive diseases in fibrosis and neurodegeneration

Differentiated Pipeline

Oral, small-molecule candidates with novel mechanisms of action and strong IP complemented by global commercial rights

Large Unmet Need

40-75%

of patients with IPF remain untreated with current therapies[1,2,3]

Significant Market

Strong projected growth in IPF market

~$4.0B → **~$8.8B**

(2021)[4] (2027 estimate)[5]

Phase 2 in IPF

Lead program targeting clinically-validated pathway for IPF as potential monotherapy or add-on to current therapies

$280M Proposed Valuation

compared to peer companies, prior M&A deals and external IPF market potential

1. Dempsey, 2021, Ann Am Thor Soc.
2. Lancaster, 2021, Respirol.
3. Maher, 2017, BMC Pulm Med.
4. Combined 2021 sales of OFEV (nintedanib) and ESBRIET (pirfenidone), as reported by Boehringer-Ingelheim and Roche, respectively, and converted to USD using "Yearly Average Currency Exchange Rates" on IRS.gov.
5. iHealthcareAnalyst, Inc. (press releasee Nov. 5, 2021).

Risks Related to the Business Combination

- BAC's shareholders will experience dilution due to the issuance of shares of common stock of BAC (after its re-domestication from the Cayman Islands to Delaware), and securities that are exchangeable for shares of common stock of BAC, to: (i) the Target's security holders as consideration in the merger and (ii) certain PIPE investors in the PIPE financing.
- The consummation of the Business Combination is subject to a number of conditions, including those set forth in the definitive Agreement and Plan of Merger (the "Merger Agreement"), and if those conditions are not satisfied or waived, the Merger Agreement may be terminated in accordance with its terms and the Business Combination may not be completed.
- If the Business Combination benefits do not meet the expectation of investors or securities analysts, the market price of BAC's securities, or following the consummation of the Business Combination, the securities of the combined company (the "Combined Entity"), may decline.
- Potential legal proceedings in connection with the Business Combination, the outcome of which may be uncertain, could delay or prevent the completion of the Business Combination.
- Following the consummation of the Business Combination, the Combined Entity will be an "emerging growth company" and it cannot be certain if the disclosure requirements applicable to emerging growth companies will make the Combined Entity's common stock less attractive to investors and may make it more difficult to compare performance with other public companies.
- The Combined Entity will incur significantly increased expenses and administrative burdens as a public company, which could have an adverse effect.
- The ability of BAC's shareholders to exercise redemption rights with respect to a large number of BAC's shares may not allow BAC to complete the Business Combination or for the Combined Entity to have the full cash available to execute its development and capital expenditure plans.
- There is no assurance that BAC's diligence will reveal all material risks that may present with regard to the Target.
- BAC may issue additional shares of common or preferred stock to complete the Business Combination or under an equity incentive plan after completion of the Business Combination, any one of which would dilute the interest of BAC's shareholders and likely present other risks.
- BAC's key personnel may negotiate employment or consulting agreements with the Combined Entity in connection with the Business Combination. These agreements may provide for them to receive compensation following the Business Combination and as a result, may cause them to have conflicts of interest in determining whether the Business Combination is advantageous.
- Because BAC's initial shareholders, executive officers and directors will lose their entire investment in BAC if the Business Combination or an alternative business combination is not completed, and because BAC's Sponsor, executive officers and directors will not be eligible to be reimbursed for their out-of-pocket expenses if the Business Combination is not completed, a conflict of interest may have arisen in determining whether the Target is appropriate for BAC's initial business combination.
- Some of the officers and directors of BAC, on the one hand, and the Target, on the other hand, may be argued to have conflicts of interest that may influence them to support or approve the Business Combination without regard to your interests.
- The value of the Sponsor's founder shares following completion of the Business Combination is likely to be substantially higher than the nominal price paid for them, even if the trading price of BAC's common stock at such time is substantially less than $10.00 per share.

Risks Related to the Business Combination (cont'd)

- BAC's shareholders and the Target's stockholders may not realize a benefit from the Business Combination commensurate with the ownership dilution they will experience in connection with the Business Combination.
- During the pendency of the Business Combination, BAC and the Target may not be able to enter into a business combination with another party because of restrictions in the Merger Agreement, which could adversely affect their respective businesses. Furthermore, certain provisions of the Merger Agreement may discourage third parties from submitting alternative takeover proposals, including proposals that may be superior to the arrangements contemplated by the Merger Agreement.
- If the conditions to the Merger are not met, including the approval by each party's respective shareholders, the Business Combination may not occur.
- Each of BAC and the Target may waive one or more of the conditions to the Business Combination, subject to certain limitations as set out in the Merger Agreement.
- U.S. federal income tax reform could adversely affect the Combined Entity and holders of the Combined Entity's securities.
- The Combined Entity will be affected by extensive laws, governmental regulations, administrative determinations, court decisions and similar constraints both domestically and abroad.
- Delaware law and the Combined Entity's proposed charter and bylaws may contain certain provisions, including anti-takeover provisions that limit the ability of stockholders to take certain actions and could delay or discourage takeover attempts that stockholders may consider favorable, as well as certain provisions limiting the ability of the Combined Entity's stockholders to choose the judicial forum for disputes with the Combined Entity or its directors, officers, or employees.
- The proposed charter will not limit the ability of the Sponsor or its affiliates to compete with the Combined Entity.
- The Combined Entity's business and operations could be negatively affected if it becomes subject to any securities litigation or stockholder activism, which could cause the Combined Entity to incur significant expense, hinder execution of business and growth strategy and impact its stock price.
- Upon effectiveness of the proposed domestication of BAC from the Cayman Islands to Delaware in connection with the Business Combination, the rights of holders of the Combined Entity's common stock arising under the Delaware General Corporate Law will differ from and may be less favorable to the rights of holders of BAC's shares arising under the Cayman Islands Companies Act.
- There is a risk that a U.S. Holder may recognize taxable gain with respect to its BAC shares at the effective time of the proposed domestication.
- BAC identified material weaknesses in its internal controls over financial reporting with respect to the accounting treatment of certain of its warrants. Failure to maintain effective internal controls over financial reporting could cause BAC to inaccurately report its financial results or fail to prevent fraud.

Risks Related to Combined Entity's Business

- The Target is very early in its development efforts, has completed few clinical trials, has no products approved for commercial sale, and has no historical product revenues, which makes it difficult to assess the Target's future prospects and financial results.
- The Target's ability to generate revenue and achieve profitability depends significantly on its ability to achieve its objectives relating to the discovery, development and commercialization of its product candidates.
- The Target has limited sales and distribution experience and needs to build a marketing and sales organization. We expect to invest significant financial and management resources to build these capabilities. To the extent any of the Target's product candidates for which it maintains commercial rights is approved for marketing, if we are unable to establish marketing and sales capabilities or enter into agreements with third parties to market and sell such product candidates, we may not be able to market and sell any product candidates effectively or generate product revenues.
- The marketing and sale of cudetaxestat or future approved products may be unsuccessful or less successful than anticipated. The Target is heavily dependent on the success of cudetaxestat, which has not been approved for the treatment of idiopathic pulmonary fibrosis or nonalcoholic steatohepatitis. If the Target is unable to advance cudetaxestat or our other product candidates through clinical development, obtain regulatory approval and ultimately commercialize our product candidates, or experience significant delays in doing so, the Target's business will be materially harmed.
- The Target is also dependent on the success of its other preclinical product candidates (BLD-2184 and other candidates). We cannot give any assurance that any product candidate will successfully complete clinical trials or receive regulatory approval, which is necessary before it can be commercialized.
- The clinical and commercial success of the Target's product candidates will depend on a number of factors, many of which are beyond the Target's control. The Target's future commercial success depends upon attaining significant market acceptance of its product candidates, if approved, among physicians, patients, third-party payors, and others in the health care community.
- Clinical development is a lengthy and expensive process with an uncertain outcome, and results of earlier studies and trials as well as data from any interim analysis of ongoing clinical trials may not be predictive of future trial results or approved label for clinical use. Clinical failure can occur at any stage of clinical development.
- Due to the Target's limited resources and access to capital in the past, the Target has decided to prioritize development of certain product candidates and may have forgone the opportunity to capitalize on product candidates or indications that may ultimately have been more profitable or for which there was a greater likelihood of success. If the Target is unable to raise substantial additional capital to finance its operations when needed, or on acceptable terms, the Target may be forced to delay, reduce or eliminate one or more of its research and drug development programs, future commercialization efforts, product development or other operations.
- The approach the Target is taking to discover and develop drugs is novel and may never lead to approved or marketable products.
- The Target may not be successful in its efforts to use and expand its novel, proprietary target discovery platform to build a pipeline of product candidates. The Target's product candidates may fail in development or suffer delays that adversely affect their commercial viability.
- The regulatory approval processes of the FDA and other comparable regulatory authorities are lengthy, time consuming and inherently unpredictable, which may affect the commercial viability of the Target's products in development. If the Target is unable ultimately to obtain regulatory approval for its product candidates, its business will be substantially harmed.
- In connection with the Target's global clinical trials, local regulatory authorities may have differing perspectives on clinical protocols and safety parameters, which impacts the manner in which the Target conducts these global clinical trials and could negatively impact the Target's chances for obtaining regulatory approvals or marketing authorization in different jurisdictions, or for obtaining the requested label or dosage for the Target's product candidates, if regulatory approvals or marketing authorizations are obtained. The results of the Target's clinical trials may not satisfy the requirements of different regulatory authorities.

Risks Related to Combined Entity's Business (cont'd)

- Even if the Target receives regulatory approval for any of its product candidates, the Target will be subject to ongoing obligations and continued regulatory review, which may result in significant additional expense. Additionally, the Target's product candidates, if approved, could be subject to labeling and other restrictions and market withdrawal and the Target may be subject to penalties if it fails to comply with regulatory requirements or experience unanticipated problems with its products.
- The Target's preclinical studies and its future clinical trials or those of any of its collaborators may fail to adequately demonstrate the safety and efficacy of any of its product candidates or reveal significant adverse events not seen in its preclinical studies or earlier clinical trials which would prevent or delay the development, regulatory approval, and commercialization of any of the Target's product candidates.
- The Target has limited experience as a company in conducting clinical trials.
- If the Target experiences delays or difficulties in the enrollment or maintenance of subjects in clinical trials, its regulatory submissions or the receipt of necessary marketing approvals could be delayed or prevented.
- Legislative and regulatory activity may exert downward pressure on potential pricing and reimbursement for any of the Target's product candidates, if approved, that could materially affect the opportunity to commercialize.
- The Target faces significant competition for its drug discovery and development efforts, and if the Target does not compete effectively, its commercial opportunities will be reduced or eliminated.
- The Target relies on adequate protection of its proprietary rights to compete effectively in its market. The Target's ability to compete may decline if it does not adequately protect its proprietary rights.
- The cost of maintaining the Target's patent protection is high and requires continuous review and compliance. The Target may not be able to effectively maintain its intellectual property position throughout our market.
- The Target may be involved in intellectual property disputes with third parties and competitors that could be costly and time consuming and negatively affect its competitive position.
- The Target relies on third parties for the conduct of most of its preclinical studies and clinical trials for its product candidates, and if its third-party contractors do not properly and successfully perform their obligations under the Target's agreements with them, the Target may not be able to obtain or may be delayed in receiving regulatory approvals for its product candidates.
- The Target current relies, and expects to continue to rely, on third parties to conduct many aspects of its product candidate manufacturing activities and the Target intends to rely on third parties for potential commercial product manufacturing. The Target's business could be harmed if those third parties fail to provide us with sufficient quantities of product or fail to do so at acceptable quality levels or prices.
- The Target's business, results of operations and future growth prospects could be materially and adversely affected by the COVID-19 pandemic.
- If the Target is unable to obtain, maintain and enforce patent protection for its technology and product candidates, or if the scope of patent protection obtained is not sufficiently broad, the Target's competitors could develop and commercialize technology and products similar or identical to those of the Target and the Target's ability to successfully develop and commercialize its technology and product candidates may be adversely affected.

BLADE THERAPEUTICS

Appendix

Strategic and Smart Capital to Advance Novel Therapies in Unmet Needs in Orphan Diseases

Backed by experienced life-science and strategic investors with deep experience investing in fibrosis therapeutic area

- Expected differentiated competitive support post de-SPAC

DEERFIELD® Advancing Healthcare®

Pfizer VENTURE INVESTMENTS

Bristol Myers Squibb

MPM Powering Breakthroughs in Life Sciences

OSAGE UNIVERSITY PARTNERS

1/ ONE VENTURES

Minimum cash condition of $75 million

- Allows sufficient runway through at least 1Q-2024

Multiple anticipated near- and long-term clinical milestones

- Phase 2 trial in IPF for cudetaxestat with anticipated blinded interim analysis in mid-2023
- Phase 1 trial* for BLD-2184 in healthy volunteers with anticipated data readout in 1H-2023

* Pending regulatory review and feedback (e.g., FDA or ex-US regulatory authorities).

Portfolio Underpinned by Strong IP Protections and Commercial Rights

	ATXi	CAPNi
Composition of Matter + Others (formulation, etc.)	2034 – 2042	2037 – 2040
	~15 CoM patents granted / allowed	Pipeline discovered in-house
Commercial Rights	Full global rights	Full global rights

Selected Fibrosis Comps

Public and IPO Comps

Company	Lead Program / Indication	Current Phase	Pre Money Val at IPO ($ mm)[1]	Recent IPO Date[1]	Price ($) 09/16/21	FD Equity Value ($ mm)	Enterprise Value ($ mm)
Morphic Holding, Inc.	MORF-057 / Ulcerative Colitis	Phase 1	$354	06/27/19	$66.79	$2,696	$2,265
Madrigal Pharmaceuticals, Inc.	MGL-3196 / NASH	Phase 3			80.15	1,330	1,008
FibroGen, Inc.	Pamrevlumab / IPF	Phase 3			11.72	1,086	722
Akero Therapeutics, Inc.	AKR-001 / NASH	Phase 2	341	06/20/19	23.89	877	648
Aligos Therapeutics, Inc.	ALG-010133 / CHB	Phase 1	430	10/16/20	16.38	722	544
Pliant Therapeutics, Inc.	PLN-74809 / IPF	Phase 2	423	06/03/20	19.00	700	456
Viking Therapeutics, Inc.	VK2809 / NASH	Phase 2			6.57	520	292
Intercept Pharmaceuticals, Inc.	Obeticholic Acid / PBC	Commercial			15.41	461	782
89bio, Inc.	BIO89-100 / NASH	Phase 2	123	11/11/19	20.06	412	242
Terns Pharmaceuticals, Inc.	TERN-101 / NASH	Phase 2	280	02/04/21	10.97	280	95
Vicore Pharma	VP01 / IPF	Phase 2			2.32	166	107
Mean			**$325**			**$841**	**$651**
Median			**$347**			**$700**	**$544**

Sources: Press Releases, Company Websites, PitchBook and Capital IQ as of September 16, 2021.
1. Data only includes recent IPOs since 2019.

Idiopathic Pulmonary Fibrosis Market Landscape and Opportunities

Systemic Sclerosis (SSc) Pathophysiology / Progression / Demographics

SSc is heterogeneous autoimmune disease often leading to chronic organ/tissue fibrosis

- Two primary disease segments: limited cutaneous (lcSSc) and diffuse cutaneous (dcSSc)
- Organ fibrosis leads to morbidity / mortality - dcSSc entails greater risk for organ involvement
- Onset is most frequently 25 – 55 years[1]
- 3-4x more common in women than men[1]

US prevalence rate of ~27.6 / 100K (~91K SSc patients in US[4])

- Majority of SSc patients exhibit some lung fibrosis – interstitial lung disease (SSc-ILD)[2]
- Progression of SSc-ILD is highly variable – typically slower than IPF[6]
- North America SSc-ILD prevalence ~47K[3]
- Global SSc-ILD prevalence ~400 – 500K[3,5]

1. National Scleroderma Foundation.
2. Denton, 2017, Lancet.
3. Bergamasco, 2019, Clin Epi.
4. Chifflot, 2008, Semin Arthritis Rheum.
5. Olson, 2018, Eur Resp Rev.
6. Goh, 2017, Arthritis Rheumatol.

SSc-ILD Approved Treatments

2028 Global SSc-ILD Market Opportunity

$2.5B [1]

Nintedanib (oral dose, BID) Pricing
~$145 K/yr[2]

Tocilizumab (IL-6R antagonist mAb – SC/Q1W) Pricing
~$59 K/yr[2]

No consensus approach to disease management[3]

- Immunosuppressive therapies are longstanding SoC

2 approved therapies

- Ofev® (nintedanib) and Actemra® (tocilizumab) approved based on slowing the decline in FVC
- KOL recommendations for use/adoption are emerging[4]

BLADE THERAPEUTICS

1. Coherent Market Insights (press release Nov. 30, 2021).
2. Drugs.com
3. Denton, 2017, Lancet.
4. Khanna, 2022, Arthritis Rheumatol.

SSc-ILD – Key Competitors in Clinical Development

Company	Stage	Product	MOA	ROA, Dosing, Molecule	Differentiators	LOE
HORIZON	Ph 2	HZN-825	LPA1 antagonist	PO, QD or BID small mol	+ Oral mono- or add-on therapy to SoC ± Excludes use of nintedanib or tocilizumab	2034
Prometheus Biosciences	Ph 2	PRA023	TL1A inhibitor	IV, Q2W mAb	+ Oral mono- or add-on therapy to SoC ± Excludes use of nintedanib or tocilizumab	Not disclosed
MERCK ACCELERON	Ph 1b/2	ACE-1334	TGF-beta 1, 3 ligand trap	SC, Q2W or Q4W recombinant protein	+ Oral mono- or add-on therapy to SoC + Add-on w/ nintedanib or tocilizumab	Not disclosed
BLADE THERAPEUTICS	Ph 2 Ready	Cudetaxestat (BLD-0409)	ATX inhibitor (non-competitive/ allosteric)	PO, QD or BID small mol	+ Oral mono- or add-on therapy to SoC + Add-on w/ nintedanib or tocilizumab	2034-2036

Includes all phase 3 programs and key phase 2 programs.

SoC = Mycophenolate mofetil.

Fibrosis – Cudetaxestat

Non-Competitive Autotaxin Inhibitor Targeting IPF

Decisive Actions by Blade to Set Clear Path to Planned Phase 2 Study of Cudetaxestat

1Q-2021	Mid-2021	4Q-2021 – 1Q-2022
Assessed GLPG-1690 Study Implications	**Pursued Stepwise Approach**	**Completed Phase 1**
Statements from Gilead and Galapagos – IDMC recommendation: "… ziritaxestat's benefit-risk profile no longer supported continuing these studies."[1] – "… it's nothing that popped up that has linked to a mechanism of action that we could explain as of today"[2]	"Given the known safety/tolerability issues with SoC (pirfenidone/nintedanib)…"[3] – Evaluate potential DDI effect on various transporters (e.g., P-gp) and CYPs – Conduct dedicated DDI study to evaluate PK between cudetaxestat and approved IPF therapies[4] prior to dose ranging studies	Successfully completed Ph1 DDI studies on CYPs and DDI with approved IPF therapies – Cudetaxestat well tolerated with no reports of drug-related SAEs
Blade proactively sought pre-IND FDA feedback		FDA provided feedback necessary to proceed into phase 2 study of cudetaxestat dosed as monotherapy or co-administered with pirfenidone or nintedanib in patients with IPF

1. Gilead news release, 10-Feb-2021.
2. Galapagos earnings call transcript, 19-Feb-2021.
3. FDA pre-IND correspondence with Blade Therapeutics.
4. Approved IPF therapies = nintedanib and pirfenidone.

DDI Profiles of Cudetaxestat (BLD-0409) and GLPG-1690

- Nintedanib known to be a substrate for P-glycoprotein (P-gp)[1]
  - P-gp transporter functions as a biological barrier by excreting certain compounds out of cells (e.g., in gastrointestinal tract, liver, and kidney)
- Cudetaxestat is neither a substrate nor an inhibitor of P-gp at physiological concentrations
- GLPG-1690 is both a substrate and an inhibitor of P-gp

P-gp transporter *in vitro* assay using quinidine as substrate

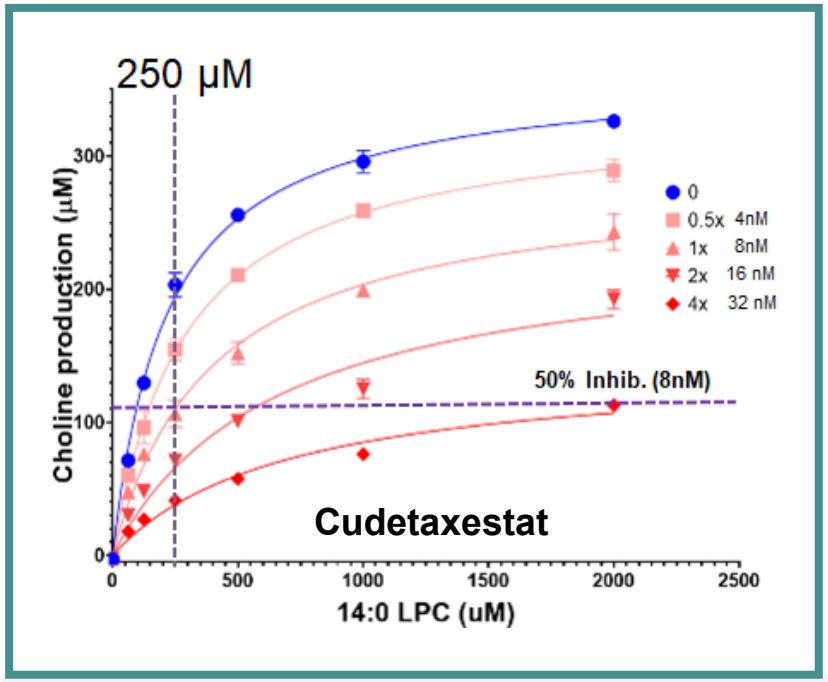
Compound	**P-gp Inhibition** (IC_{50} µM)	**P-gp Substrate** (Efflux Ratio @ 10µM)
Cudetaxestat	**Very Weak** (64.6 µM)	**Not a substrate** (1.7)
GLPG-1690	**Moderate** (7.8 µM)	**Yes** (60)
Nintedanib	Weak (>30 µM)[1]	**Yes** (16)
Pirfenidone	Weak (>100 µM)[2]	Not a substrate

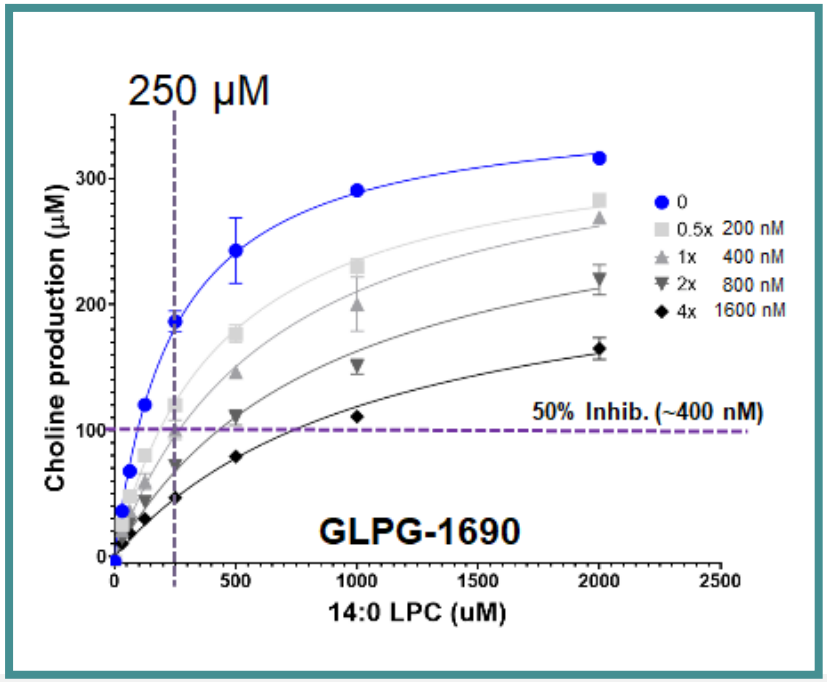
P-gp *in vitro* assay using nintedanib as substrate

Compound	**P-gp Inhibition** (IC_{50} µM)
Cudetaxestat	39.8 µM
GLPG-1690	3.84 µM

1. Wind, 2019, Clin. Pharm.
2. Esbriet® (pirfenidone) USPI.

Cudetaxestat – Potential 50x Potency Advantage vs. Ziritaxestat (GLPG-1690)



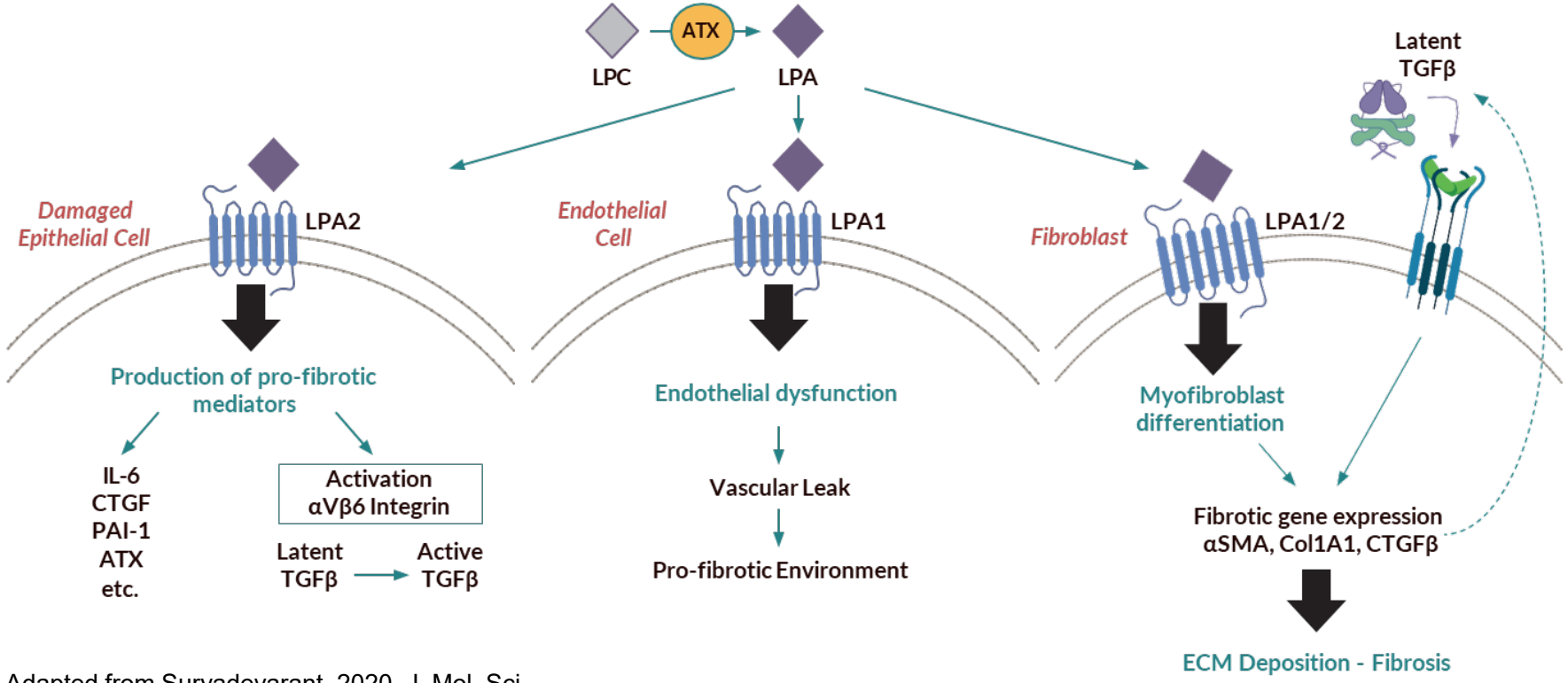


- Non-competitive inhibitor cudetaxestat shown to maintain potency in preclinical biochemical assay
  - Cudetaxestat expected to maintain potency in disease state
  - Potentially advantageous for efficacy and safety profile
- 50% inhibition at 250 uM concentration of LPC*
  - Cudetaxestat: 8 nM
  - GLPG-1690: ~400 nM (competitive inhibitor loses potency)

BLADE THERAPEUTICS

* Plasma concentrations of LPC in an animal model of liver fibrosis is between 200-250 µM.
Head-to-head clinical studies comparing cudetaxestat with currently approved or investigational therapies have not been conducted. Above data from preclinical biochemical study.

Role of ATX / LPA Signaling Pathway in Pulmonary Fibrosis



Adapted from Suryadevarant, 2020, J. Mol. Sci.

1 Autotaxin (ATX) generates ~80-90% of lysophosphatidic acid (LPA) in the body[1]

2 LPA signals through multiple LPA receptors on various cell-types to promote lung fibrosis[2]

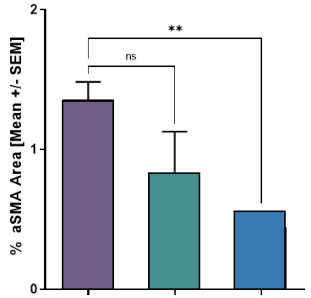
3 ATX - LPA signaling induces TGFβ production and these pathways synergize to drive the fibrotic process[2]

1. Blade data on file. 2. Suryadevera, 2020, Int J Mol Sci.

Cudetaxestat Displays Robust Activity (*in vivo*) on Lung Fibrosis

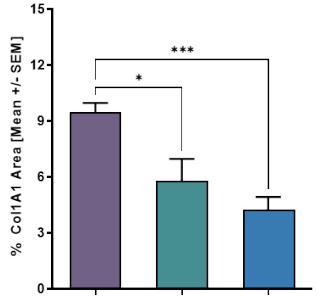
SMA gene expression

Marker of myofibroblast activation



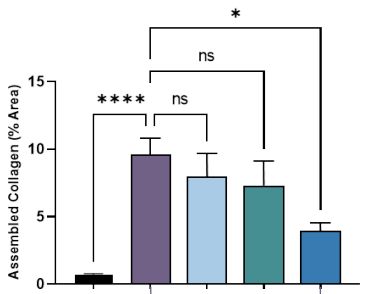
Collagen 1A1 gene expression

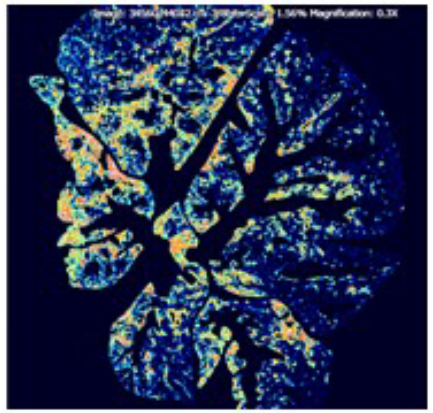
Primary component of fibrosis, produced by myofibroblasts



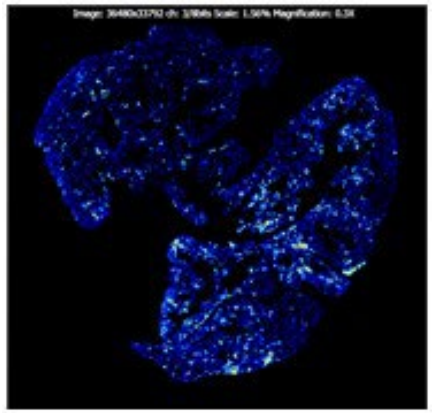
Assembled collagen

Represents advanced fibrosis





Bleomycin + Vehicle



Bleomycin + cudetaxestat 30 mg/kg

Saline
Vehicle
Cude 3mg/kg
Cude 10mg/kg
Cude 30mg/kg

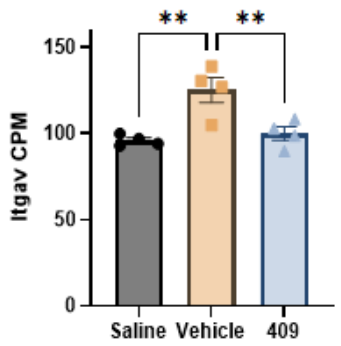
ANOVA One-Way
**p<0.05 | **p<0.01*
****p<0.001 | ****p<0.0001*

SMA (smooth muscle actin), collagen 1A1 and assembled collagen data from preclinical study conducted in mouse bleomycin lung fibrosis model dosed once daily.
PAI-1, IL-6 and CTGF (connective tissue growth factor) data from preclinical study conducted in mouse bleomycin lung fibrosis model dosed twice daily.

Cudetaxestat Displays Robust Activity (*in vivo*) on Key Fibrotic Genes

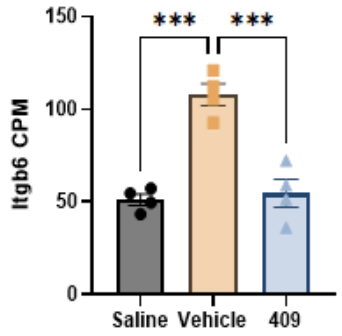
αV Integrin gene expression

Pro-fibrotic signaling



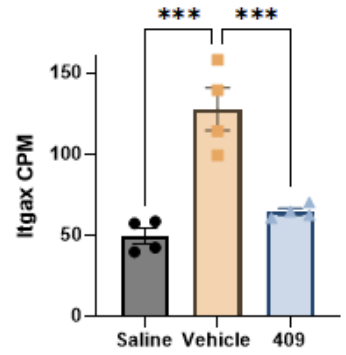
β6 Integrin gene expression

Pro-fibrotic signaling



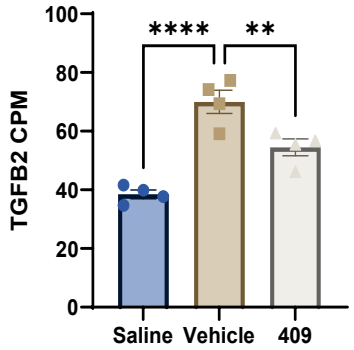
αX Integrin gene expression

Pro-fibrotic signaling



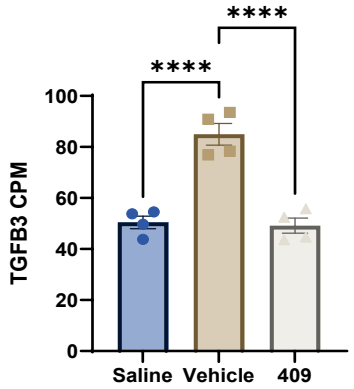
TGFβ2 gene expression

Pro-fibrotic growth factor



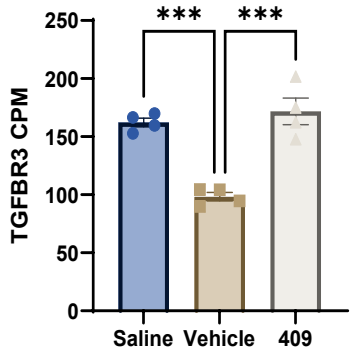
TGFβ3 gene expression

Pro-fibrotic growth factor



TGFβR3 gene expression

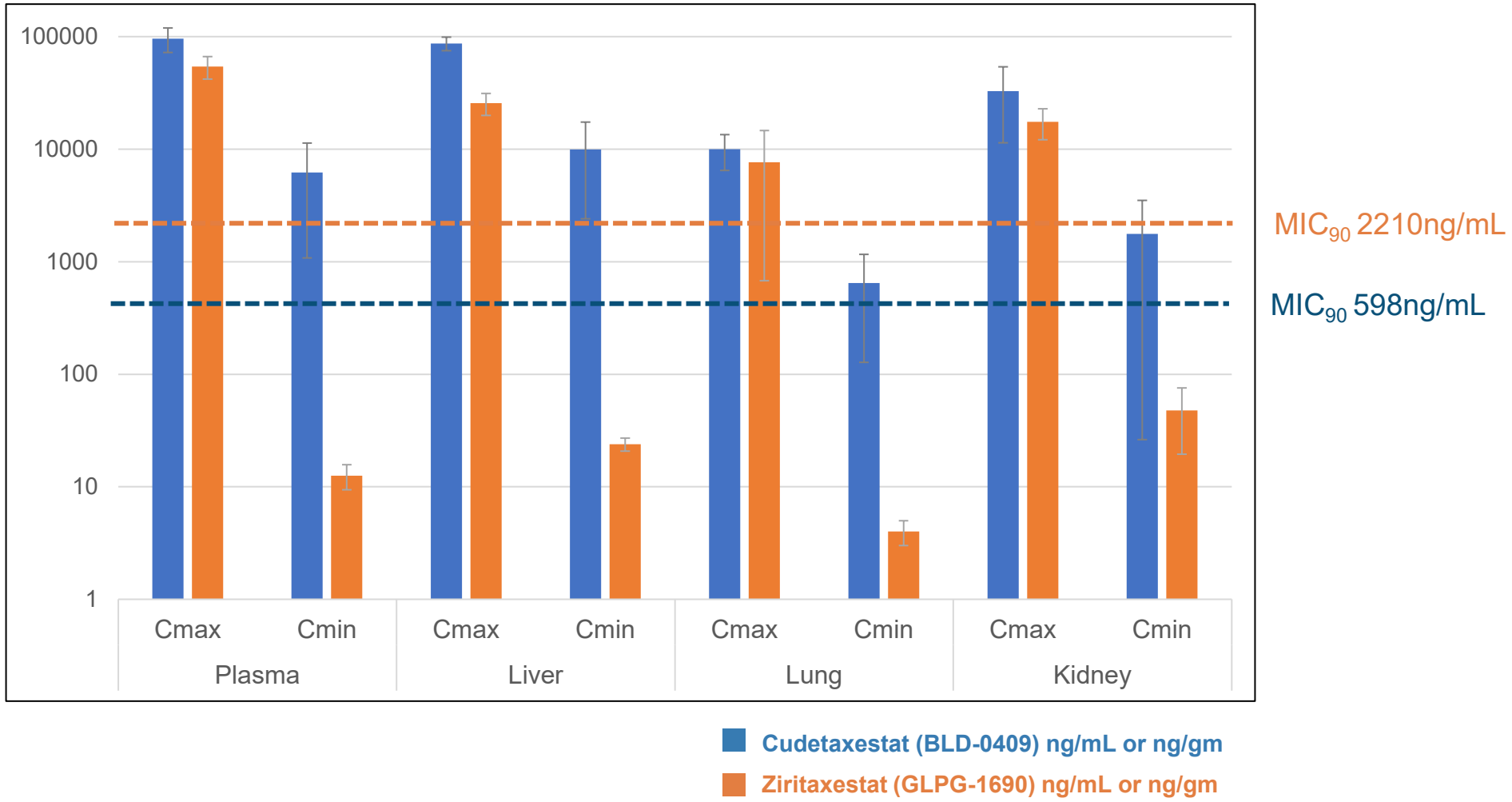
Anti-fibrotic growth factor receptor



ANOVA One-Way
**p<0.05 | **p<0.01*
****p<0.001 | ****p<0.0001*

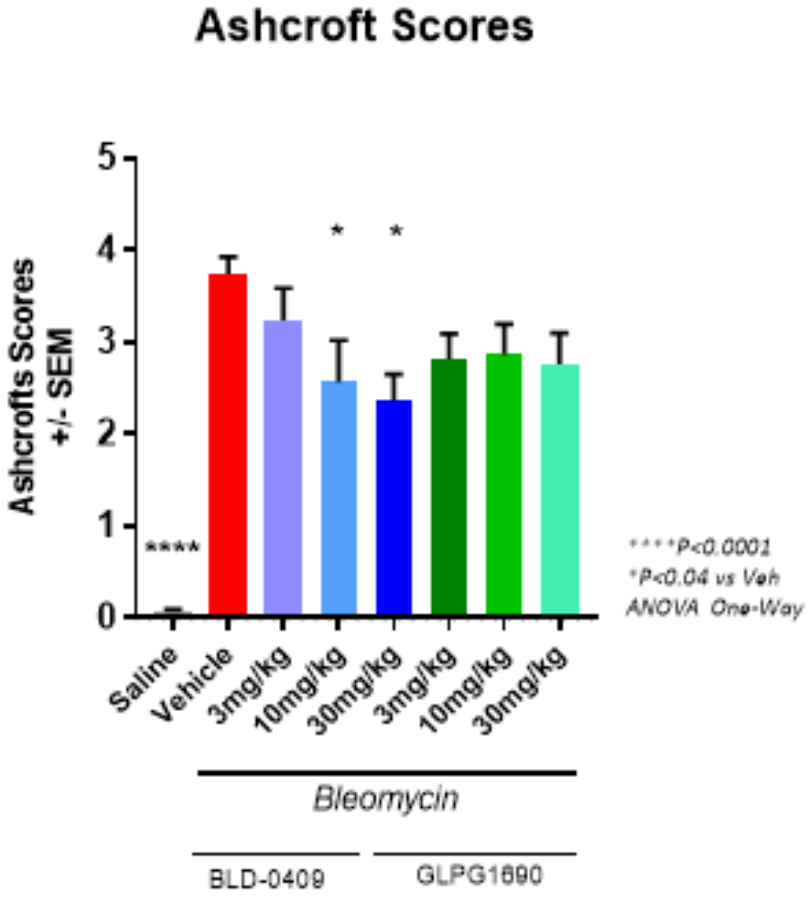
TGFβ = Transforming Growth Factor beta.

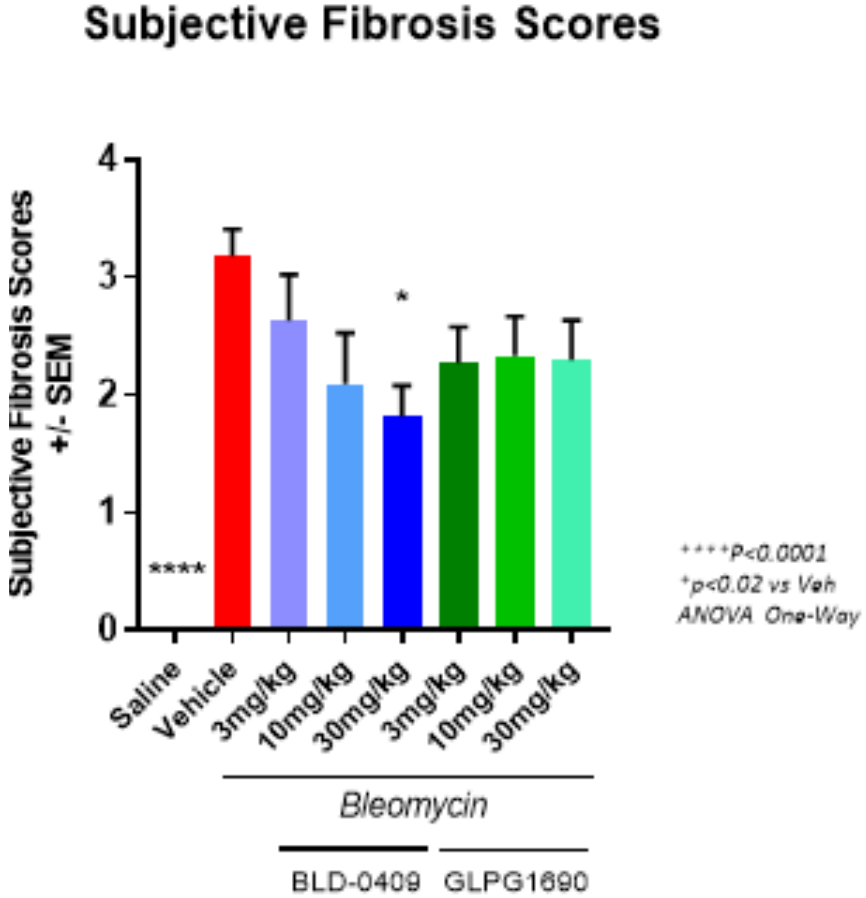
Non-Competitive Inhibition with Cudetaxestat Achieved More Consistent Tissue Exposure (*in vivo*) in Preclinical Study



Head-to-head clinical studies comparing cudetaxestat with currently approved or investigational therapies have not been conducted. Above data from preclinical mouse PK study.

Cudetaxestat (BLD-0409) Demonstrated Robust Activity (*in vivo*) in Preclinical Study





Endpoints:

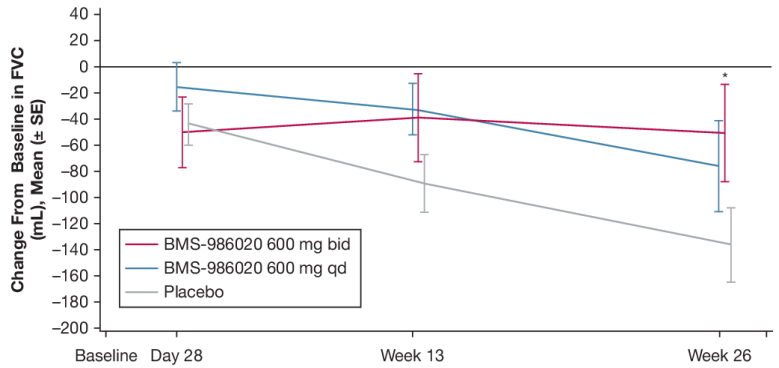
- LPA 18:2 in plasma, BALF, liver and kidney
- Histopathology for fibrosis
- Markers of fibrosis (aSMA, Col1A1)
- Biomarkers downstream of LPA receptors (exploratory)

Head-to-head clinical studies comparing cudetaxestat with currently approved or investigational therapies have not been conducted. Above data from preclinical study conducted in mouse bleomycin lung fibrosis model dosed once daily.

Supporting Peer Clinical Evidence on Lung Function (FVC)

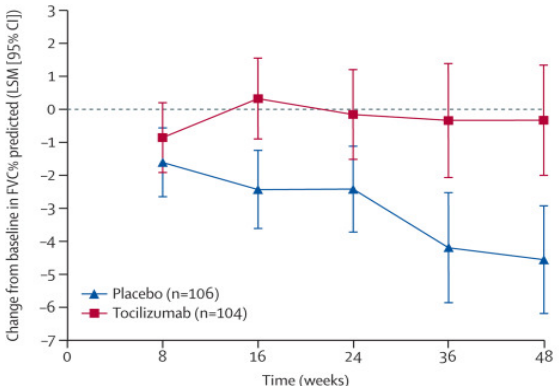
BMS-986020 (LPA1 antagonist) Ph2[1]

Significant FVC benefit at wk 26 in BID group in IPF



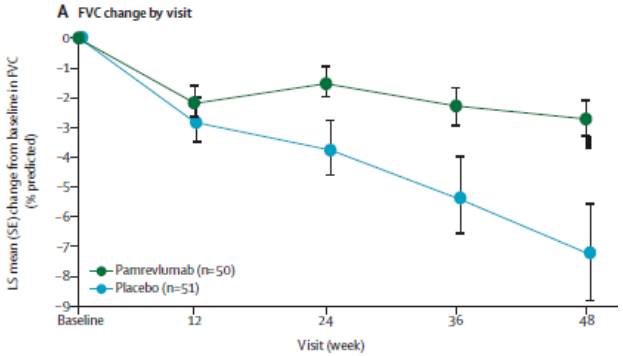
Tocilizumab (IL-6R antagonist) Ph3[2]

Significant FVC benefit at wk 48 in SSc-ILD



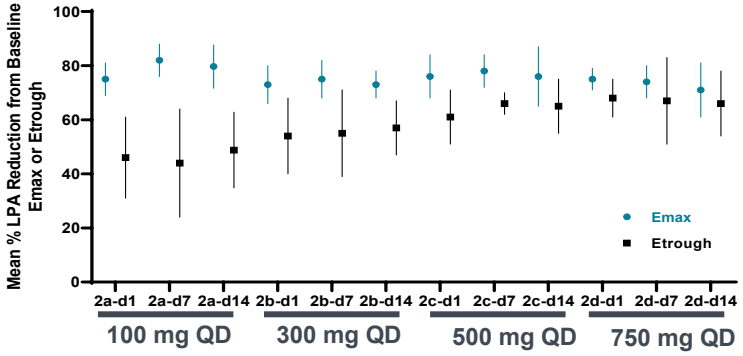
Pamrevlumab (anti-CTGF mAb) Ph2[3]

Significant FVC benefit at wk 48 in IPF

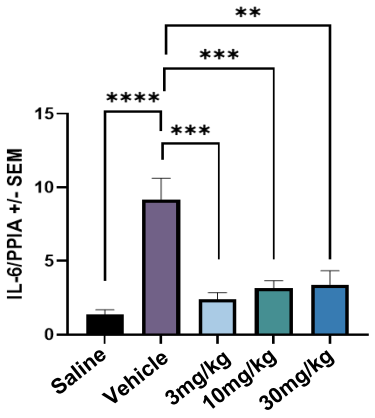


Cudetaxestat - Ph1 MAD in Healthy Volunteers LPA Reduction

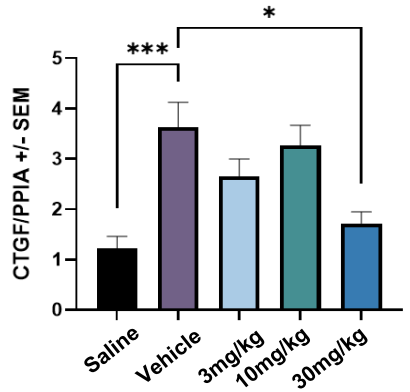
(Peak-to-trough variation)



Cudetaxestat - Mouse Bleo Lung model IL-6 gene expression



Cudetaxestat - Mouse Bleo Lung model CTGF gene expression



1. Palmer, 2018, Chest.
2. Khanna, 2020, Lancet.
3. Richeldi, 2020, Lancet.

Cudetaxestat Phase 2 Basket Study in SSc-ILD Preliminary Design*

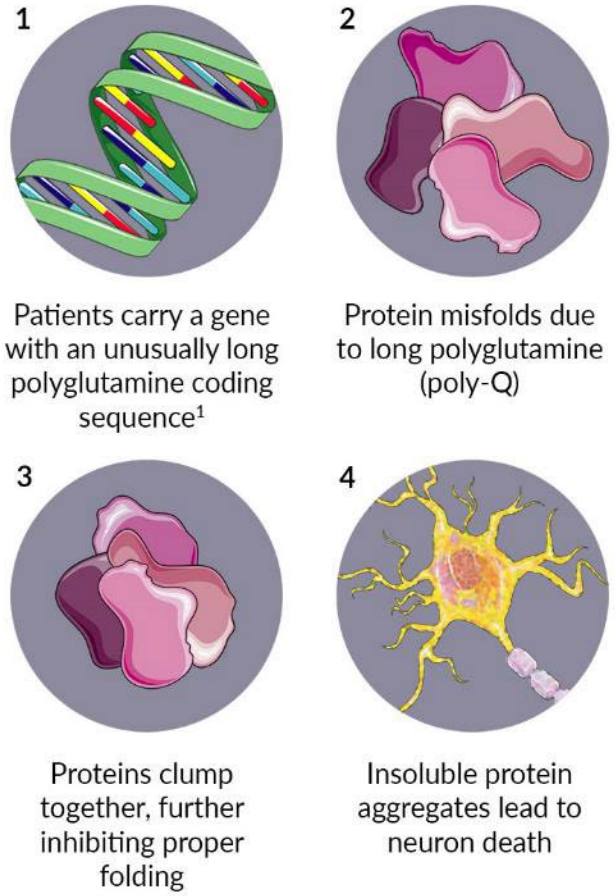
Design	• Multicenter, double-blind, randomized, placebo-controlled clinical study • Patients with interstitial lung disease secondary to systemic sclerosis (ACR/EULAR criteria) • 48-week treatment duration
Primary Endpoint	• Change from baseline in FVC (L)
Secondary Endpoints**	• Composite endpoints • Composite Response Index in Systemic Sclerosis (CRISS) score • Health Assessment Questionnaire Disability Index (HAQ-DI) • Absolute FVCpp • Change in Modified Rodnan Skin Score (mRSS)* • All cause mortality
Safety**	• Treatment-emergent adverse events • Assessment of vital signs, clinical laboratory parameters, and electrocardiograms

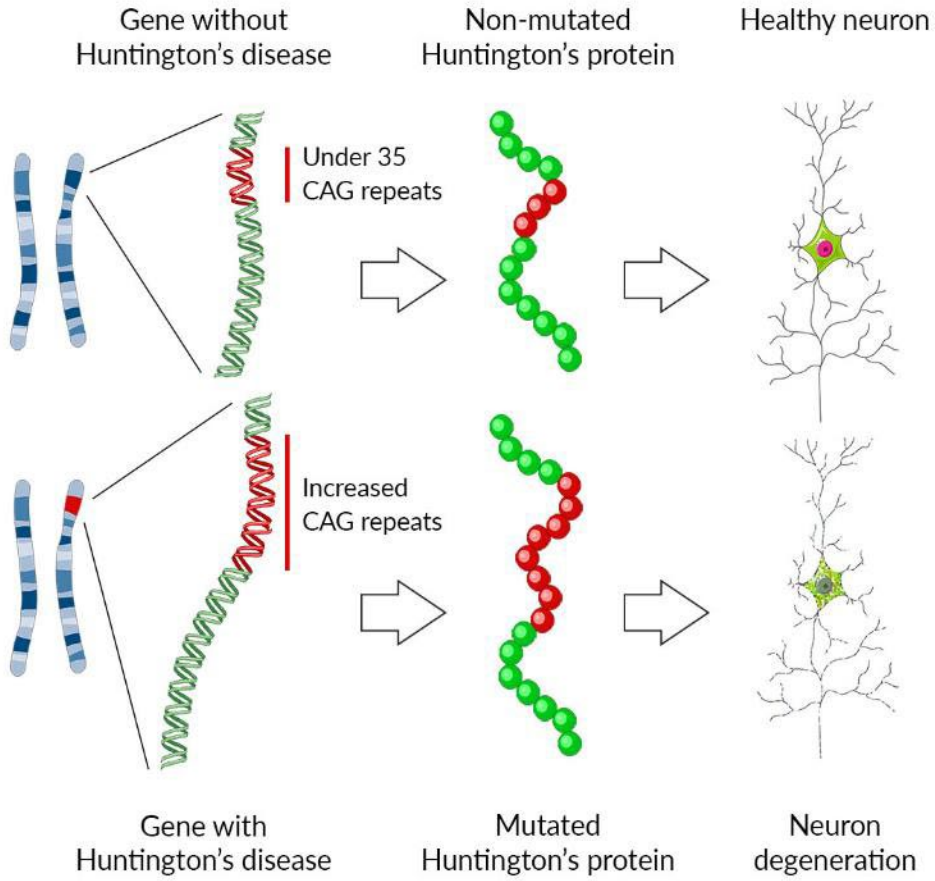
* Pending FDA review and feedback.
** Select adjudicated endpoints and assessments noted.

Neurodegeneration – BLD-2184

CNS-Penetrant Calpain Inhibitor for Poly-Q Neurodegenerative Conditions

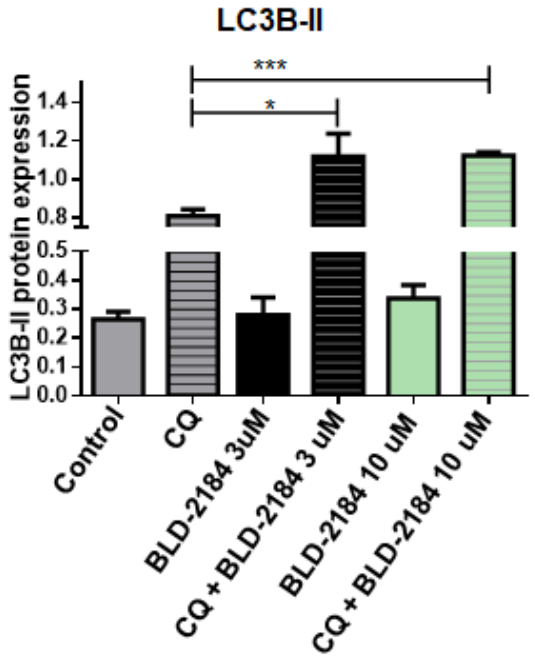
Misfolded Proteins Trigger Progressive Neurodegenerative Diseases
(e.g., Huntington's disease, Spinocerebellar ataxia type 3 (SCA3/MJD)





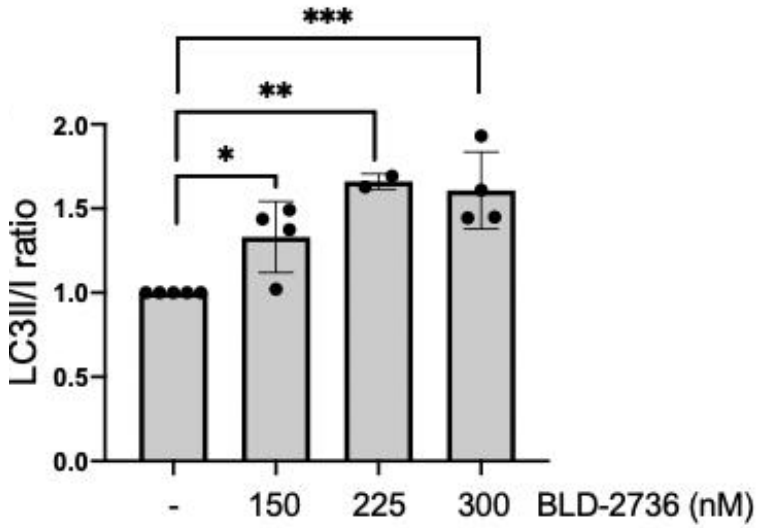
Calpain Inhibitors Enhance Autophagy in Preclinical Studies (*in vitro, in vivo*)

BLD-2184 increases autophagic flux in neuro2A cells (*in vitro*)



Increased autophagic flux as measured by LC3B-II (in presence of chloroquine, which blocks lysosomal degradation)

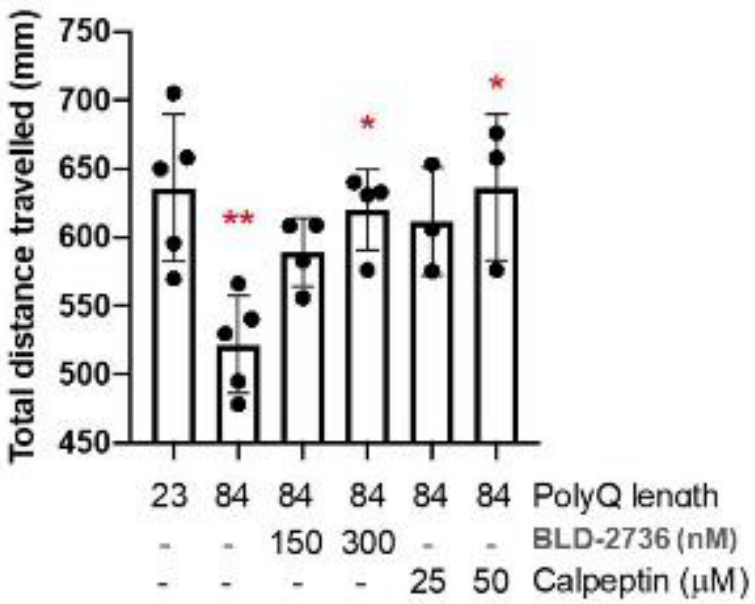
BLD-2736 increases autophagic flux in zebrafish larvae (*in vivo*)

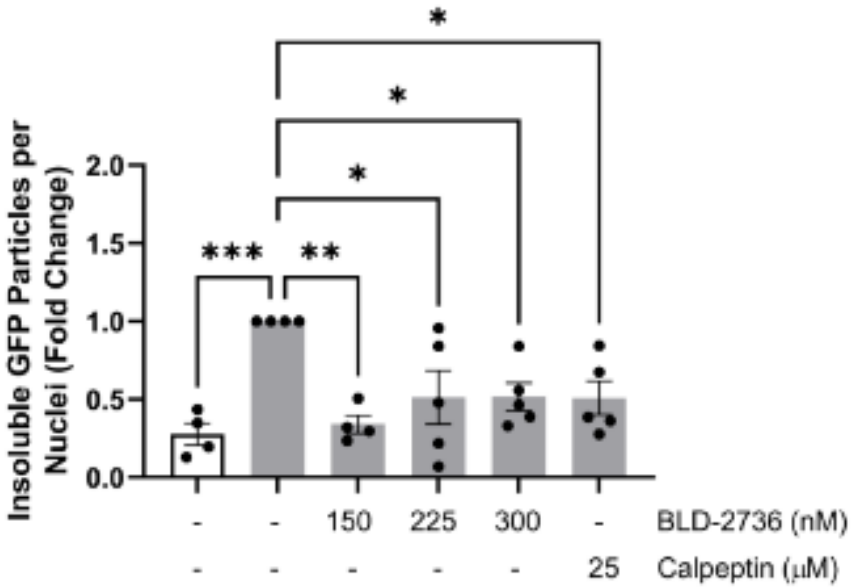


Increased autophagic flux (measured as ratio of LC3-II to LC3-I)

Neuroprotective Effects of Calpain Inhibitor Demonstrated in Preclinical Model

Improved motor function (swimming) and decreased aggregates
(BLD-2736 in mutant hATXN3 zebrafish model)





Robinson et al. *Cells* 2021, 10, 2592.

BLD-2184 Planned Phase 1 Study Design*

Study Title: Phase 1A, Randomized, Double-Blind, Placebo-Controlled, Single & Multiple Ascending Dose Study to Evaluate the Safety, Tolerability, and Pharmacokinetics of BLD-2184 in Healthy Volunteers

2H-2022 | Screening / Enrollment | Drug Administration (N=72) | End of Study | 1H-2023

Eligibility confirmation

Healthy volunteers (HVs)

Part 1 – SAD (N=40)
Single Ascending Dose

- 5 cohorts
- Assess PK parameters

Part 2 – MAD (N=32)
Multiple Ascending Doses

- 4 cohorts
- Assess maximal tolerable dose

* Pending regulatory review and feedback (e.g., FDA or ex-US regulatory authorities).

Neurodegeneration Market Landscape and Opportunities

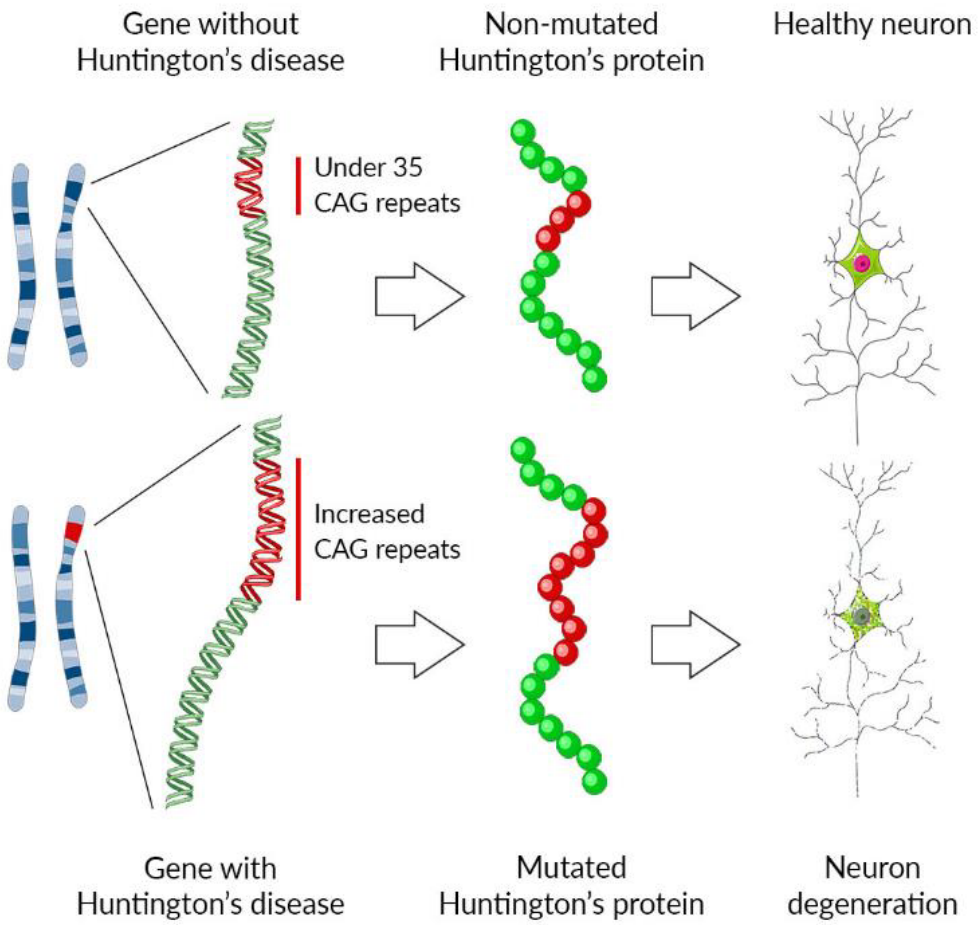
Significant Needs in Orphan Neurodegenerative (Poly-Q) Diseases

	Huntington's Disease (HD)	Spinocerebellar Ataxia 3 (SCA3) (aka, Machado-Joseph Disease or MJD)
Prevalence	~5.7 per 100K (US, CA, EU, Australia) ~20–50K (US)	~2.7 per 100K (global) ~2K (US), ~5K (EU), ~1.7K (JP), ~5K (BR), ~20–100K (PRC)
Disease	Autosomal dominant (poly-Q) progressive neurodegeneration	Autosomal dominant (poly-Q) progressive neurodegeneration
Diagnosis	Movement disorder (rapid, jerky), psychiatric and dementia	Cerebellar ataxia, muscle weakness and loss of muscle mass

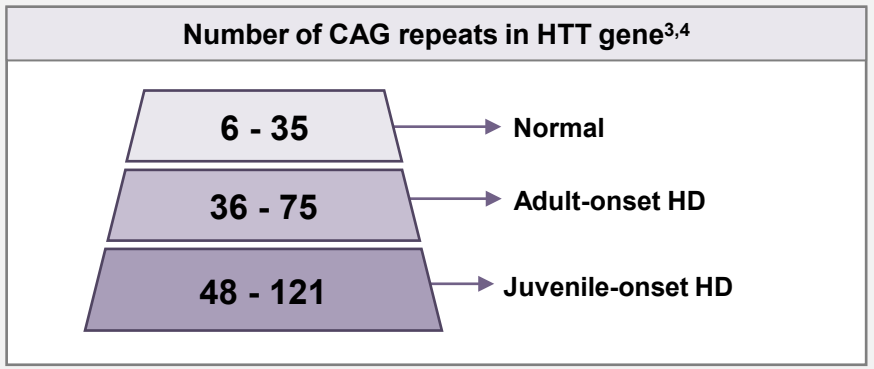
Significant unmet need for disease-modifying therapies that are safe and well-tolerated

Physio-pedia.com – Huntington Disease.
Huntington Study Group.
Physio-pedia.com. Machado-Joseph Disease (Spinocerebellar Ataxia Type 3).
Ruano, 2014, Neuroepidemiol.
Nobrega, 2018, Adv Exp Med Biol.
NIH-NINDS.

HD – Misfolded Proteins Trigger Progressive Neurodegenerative Diseases



- Current SoC is symptomatic treatment and supportive care
  - Chorea treated with antipsychotics and dopamine-depleting agents
  - List price of deutetrabenazine $55 – $155K/yr ($4.6K - $14K depending on dose)[1,2]
- HD complications typically cause death 10-30 years after onset



1. Drugs.com.
2. Austedo® (deutetrabenazine) USPI.
3. Podvin, 2019, J Neurol.
4. Singh, 2021, Curr Gene Ther.

SCA3 (aka Machado-Joseph Disease or MJD)

SCA3 Subtype[1]	Time of Onset	Rate of Progression
Type 1	Early onset 5-30 (mean 24 yrs)	Fast
Type 2	Average onset (mean ~36 yrs)	Intermediate
Type 3	Late onset (mean ~50 yrs)	Slow

- Clinically heterogeneous
  - Subtypes classified based on phenotype and progressive disease
- Mean age of onset: ~40 years[1]
  - Most patients require a wheelchair 10-15 years after disease onset[2]
- No approved therapy to treat underlying disease
  - Current SoC is symptomatic treatment and supportive care

1. Nobrega, 2018, Adv Exp Med Biol.
2. NIH-NCBI (https://www.ncbi.nlm.nih.gov/books/NBK1196/).

HD – Key Competitors in Clinical Development

Company	Stage	Product	MOA	ROA, Dosing, Molecule	Differentiators	LOE
prilenia	Ph 3	Pridopidine	Sigma type 1 receptor agonist	PO, BID small mol	• mRNA splicing modulator	2020
ANNEXON biosciences	Ph 2	ANX-005	Complement C1q mAb antagonist	IV, Q2W mAb	• Targets aberrant complement-mediated (C1q)	2037
AOP ORPHAN FOCUS ON RARE DISEASES	Ph 2	Selisistat	SIRT1 Inhibitor	PO, QD small mol	• To enhance clearance of mutant Htt protein	Unclear
NOVARTIS	Ph 2	Branaplam	SMN2 modulator	PO, QW small mol	• Stabilizes SMN2 mRNA splicing modulator	2035
Roche	Ph 2	Tominersen	Antisense targeting HTT gene	Intrathecally Q8W or Q16W antisense	• No clinical benefit for most HD patients • May also impact normal HTT gene product	2032
uniQure	Ph 2	AMT-130	HTT gene silencing (miRNA)	3 intra-brain injections one-time gene therapy AAV-miRNA	• Adeno-assoc. virus (AAV) delivery of miRNA	2037 - 2038
VACCINEX	Ph 2	pepinemab (VX15/2503)	SEMA4D antagonist	IV, Q4W mAb	• Didn't meet pre-specified co-primary endpoints • >beneficial changes in more advanced disease	2030 - 2038
BLADE THERAPEUTICS	Ph 1-ready	BLD-2184	Calpain inhibitor	PO, QD or BID small mol	• Reduce mutant protein fragments • Enhance autophagy - clearance • Reduce mutant protein aggregates	2037 – 2040

Includes all phase 3 programs and key phase 2 programs.

Limited Competitive Pipelines in SCA3

Company	Stage	Product	MOA	ROA, Dosing, Molecule	Differentiators	LOE
biohaven pharmaceuticals	Ph 3	Troriluzole	Tripeptide prodrug conjugate of riluzole	PO, QD small mol	• Increase glutamate uptake from the synapse • Reduce ataxia in all SCA diseases	2036
SEELOS THERAPEUTICS	Ph 2	Trehalose	Low mol weight disaccharide	IV, Q24W small mol	• Reduce mutant protein aggregates • Enhance autophagy	2033
NOVARTIS	Ph 2	Rimtuzalcap	SK channel activator (calcium-activated K+ channels)	PO, BID small mol	• Reduce tremors and ataxia in all SCA diseases	Not Disclosed
BLADE THERAPEUTICS	Ph 1-ready	BLD-2184	Calpain inhibitor	PO, QD or BID small mol	• Reduce mutant protein fragments • Enhance autophagy - clearance • Reduce mutant protein aggregates	2037 – 2040

Includes global phase 3 programs and key phase 2 programs.