Merger Announcement

CORPORATE PRESENTATION

OCTOBER 2023

Forward-Looking Statements

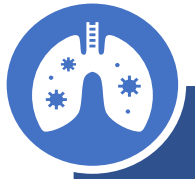
This presentation and various remarks we make during this presentation contain forward-looking statements of Aileron Therapeutics, Inc. ("Aileron", the "Company", "we", "our" or "us") within the meaning of the Private Securities Litigation Reform Act of 1995, including statements with respect to: future expectations, plans and prospects for the Company following the consummation of the merger transaction (the "Merger") between the Company and Lung Therapeutics, Inc. ("Lung Tx"); the expected closing of the concurrent private placement; the use of proceeds from the private placement and the sufficiency of the Company's cash resources; stockholder approval of the conversion of the non-voting preferred stock; the initial market capitalization of the Company following the Merger and the benefits of the Merger; and the milestones of the Company; the projected cash runway of the Company; the status and plans for clinical trials, including the timing of data; future product development; and the potential commercial opportunity of LTI-03 and LTI-01. We use words such as "anticipate," "believe," "estimate," "expect," "hope," "intend," "may," "plan," "predict," "project," "target," "potential," "would," "can," "could," "should," "continue," and other words and terms of similar meaning to help identify forward-looking statements, although not all forward-looking statements contain these identifying words. Actual results may differ materially from those indicated by such forward-looking statements as a result of various important factors, including risks and uncertainties related to the ability to recognize the anticipated benefits of the Merger, the outcome of any legal proceedings that may be instituted against the Company following the Merger and related transactions, the ability to obtain or maintain the listing of the common stock of the Company on The Nasdaq Stock Market following the Merger, costs related to the Merger, changes in applicable laws or regulations, the possibility that the Company may be adversely affected by other economic, business, and/or competitive factors, including risks inherent in pharmaceutical research and development, such as: adverse results in the Company's drug discovery, preclinical and clinical development activities, the risk that the results of preclinical studies and early clinical trials may not be replicated in later clinical trials, the Company's ability to enroll patients in its clinical trials, and the risk that any of its clinical trials may not commence, continue or be completed on time, or at all; decisions made by the U.S. FDA and other regulatory authorities, investigational review boards at clinical trial sites and publication review bodies with respect to our development candidates; our ability to obtain, maintain and enforce intellectual property rights for our platform and development candidates; our potential dependence on collaboration partners; competition; uncertainties as to the sufficiency of the Company's cash resources to fund its planned activities for the periods anticipated and the Company's ability to manage unplanned cash requirements; and general economic and market conditions; as well as the risks and uncertainties discussed in the "Risk Factors" section of the Company's Annual Report on Form 10-K for the year ended December 31, 2022, which is on file with the Securities and Exchange Commission, and in subsequent filings that the Company files with the Securities and Exchange Commission. These forward-looking statements should not be relied upon as representing the Company's view as of any date subsequent to the date of this presentation, and we expressly disclaim any obligation to update any forward-looking statements, whether as a result of new information, future events or otherwise, except as required by law.

This presentation contains estimates and other statistical data made by independent parties and by us relating to our clinical data, market size and other data about our industry. This data involves a number of assumptions and limitations, and you are cautioned not to give undue weight to such data and estimates. In addition, projections, assumptions and estimates of our future performance and the future performance of the markets in which we operate are necessarily subject to a high degree of uncertainty and risk.

Aileron Acquisition of Lung Therapeutics

Transaction Summary	• Aileron Therapeutics, Inc. (”Aileron”) Nasdaq: ALRN has acquired Lung Therapeutics, Inc. (“Lung Tx”) • Lung Tx has multiple clinical stage candidates targeting orphan pulmonary and fibrosis indications • Aileron announced a concurrent private placement of approximately $18 million which is expected to close on November 2, 2023 • Securities to be issued in the private placement consist of Series X non-voting convertible preferred stock and warrants to purchase Aileron common stock • Aileron intends to seek shareholder approval of the conversion of the Series X non-voting preferred stock into common stock • On a post-conversion basis (excluding any potential exercise of warrants issued in the private placement), shares of common stock outstanding is expected to be approximately 32.2 million, notwithstanding certain beneficial ownership limitations set at, and not to exceed 19.99%
Use of Proceeds	• As of September 30, 2023, on a pro forma basis to give effect to the receipt of gross proceeds of the private placement, the combined company cash and cash equivalents was approximately $29 million. • Proceeds from the private placement will primarily be used to complete the ongoing Phase 1b clinical study of LTI-03 and for general corporate purposes • Expected to support runway into fourth quarter of 2024
Key Management and Board	• Aileron to continue to be led by Aileron Chief Executive Officer, Manuel C. Aivado, M.D., Ph.D and Susan Drexler, M.B.A., C.P.A., Interim Chief Financial Officer, and by Brian Windsor, Ph.D, former CEO of Lung Tx, who has been appointed as President and Chief Operating Officer of Aileron • Board of Directors will be comprised of four continuing directors from Aileron: Chairman Josef H. Von Rickenbach, Manuel C. Aivado, M.D., Ph.D, Reinhard J. Ambros, Ph.D, and Nolan Sigal, M.D., Ph.D. and two directors from Lung Tx who have been appointed to the Aileron Board: William C. Fairey and Alan A. Musso

Clinical-stage Biotech with Pulmonary Pipeline



Therapies for Underserved Fibrosis and Pulmonary Conditions

LTI-03 ***Idiopathic Pulmonary Fibrosis***	***Phase 1b***	• Preclinical evidence supporting the ability to **protect healthy lung epithelial cells** and to **reduce pro-fibrotic signaling** • Demonstrated ability to increase sRAGE, a **prognostic biomarker** of IPF
LTI-01 ***Loculated Pleural Effusions***	***Phase 2b ready***	• **Potentially fatal disease** with no approved drugs • Completed Phase 1b and Phase 2 trials; similar mechanism as existing, off label therapeutic use
LTI-05 ***Cystic Fibrosis***	***PC***	• ENaC inhibitor intended for the **15-20% of CF pts.** who **do not respond to CFTR modulators** • **100% inhibition** and **localized** activity (safety profile) in preclinical studies

Members of Advisory Board and Consultants to Company are Leading KOLs in Targeted Indications

Idiopathic Pulmonary Fibrosis

Toby Maher, M.D., Ph.D.
Univ. of Southern California; National Lung and Heart Institute at Imperial College London

Andreas Gunther, M.D.
Justus-Liebig University, Giessen, Germany

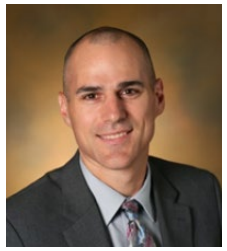
Fernando J. Martinez, M.D., M.S.
Weill Cornell Medical College

Ganesh Raghu, M.D.
Univ. of Washington Medical Center

Loculated Pleural Effusions

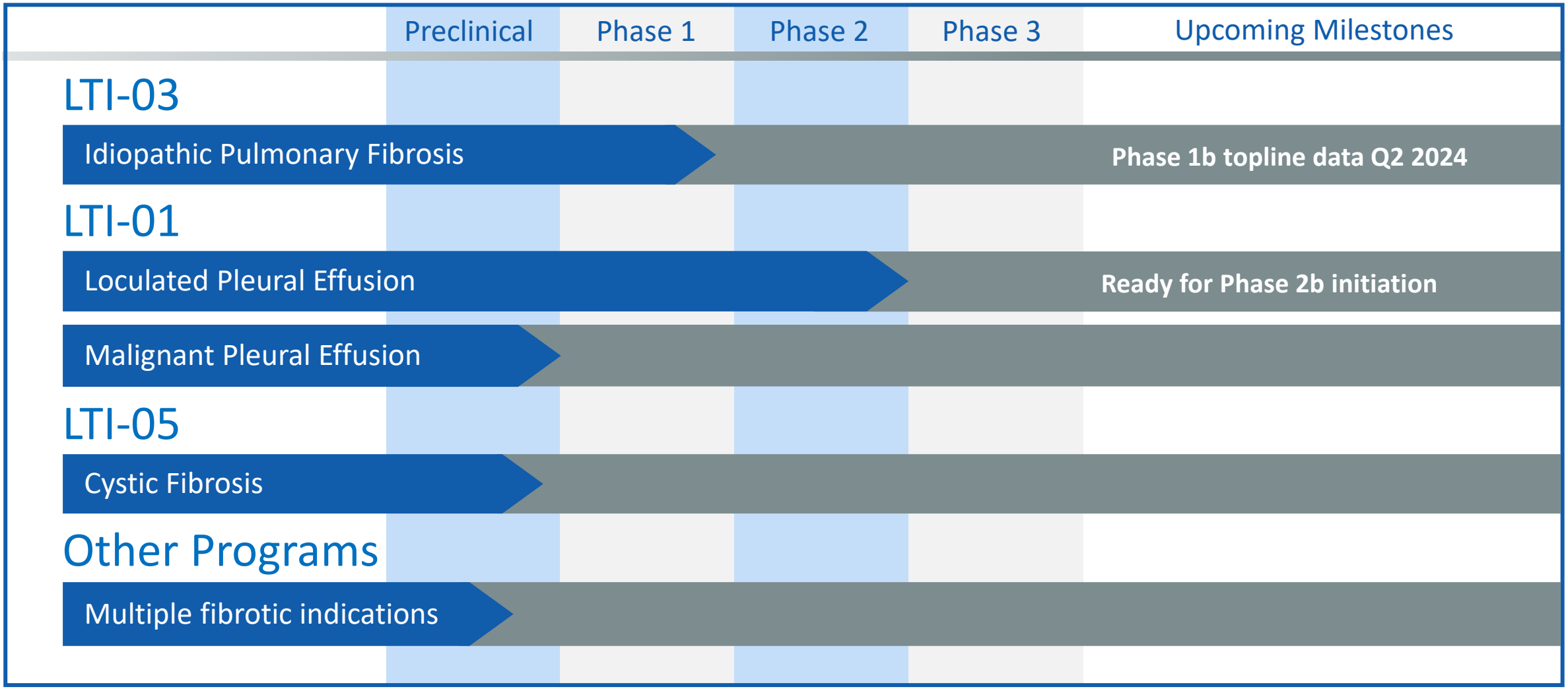
Najib M. Rahman, M.D.
Oxford Centre for Respiratory Medicine, UK

Jason Akulian, M.D., M.P.H.
UNC School of Medicine

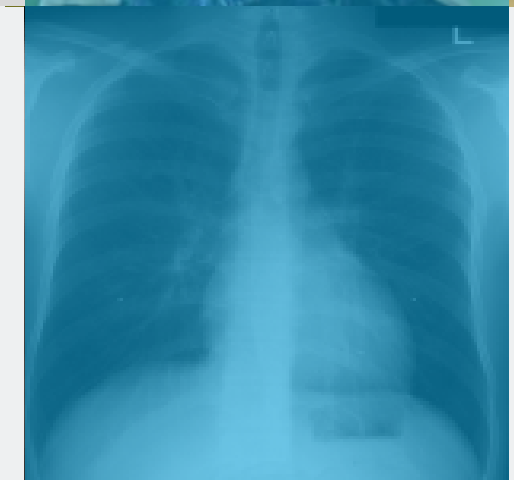


Fabien Maldonado, M.D.
Vanderbilt University Medical Center

Multiple Orphan Disease Programs with Upcoming Milestones



LTI-03: A Novel Treatment for Idiopathic Pulmonary Fibrosis

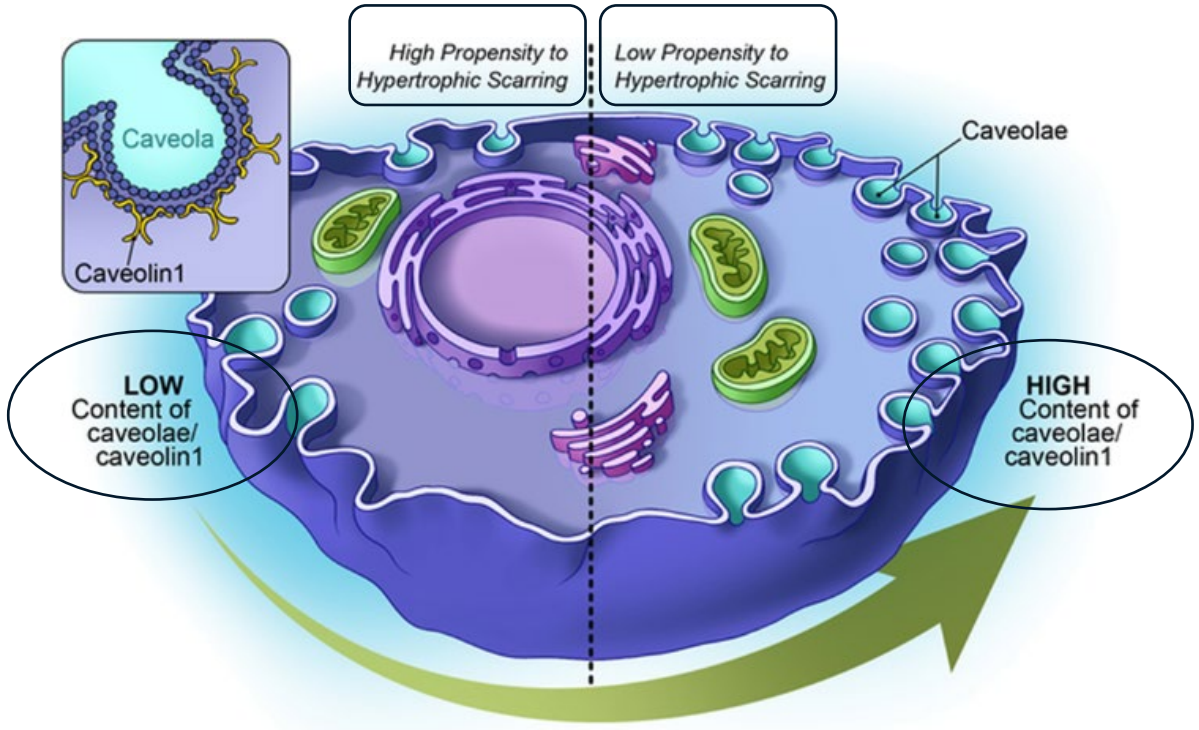


LTI-03 is a Peptide Region of Caveolin-1 Protein Indicated for Idiopathic Pulmonary Fibrosis

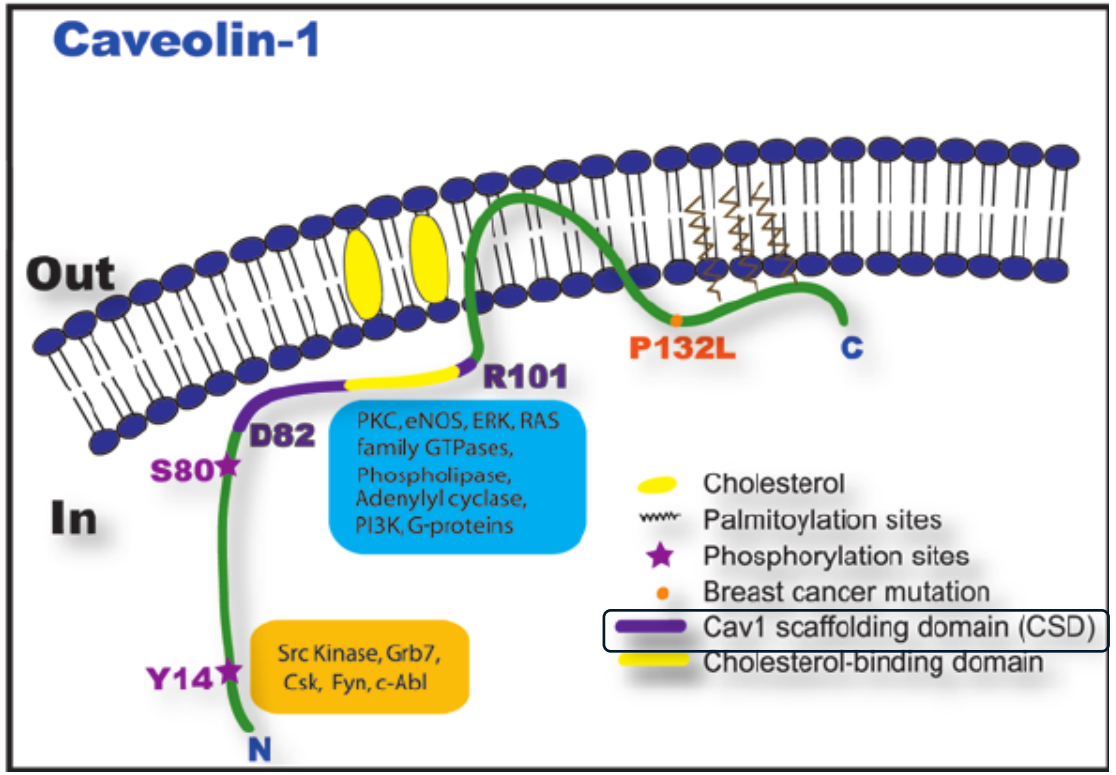
- Multiple preclinical studies support dual mechanism of Cav1 – ability to inhibit multiple pro-fibrotic pathways and protect lung epithelial cells
- We believe current SOC treatment options offer modest clinical benefit, have significant side effects and intolerance, and are not curative
  - ~100,000[1] IPF patients in the U.S. with expected median survival 2-5 years[2] from diagnosis
- Successfully completed Phase 1a randomized, double-blind placebo-controlled study in healthy normal volunteers
  - Currently in a Phase 1b randomized, double-blind placebo-controlled study
- sRAGE - prognostic biomarker of IPF disease preferentially increased in ex-vivo IPF tissue samples and Phase 1a treated patients
  - RAGE is primarily expressed by epithelial cells in lung tissue

1 United States National Library of Medicine website.
2 Nathan et al. Long-term Course and Prognosis of Idiopathic Pulmonary Fibrosis in the New Millennium. *Chest Journa*l Volume 140, ISSUE 1, P221-229, July 2011

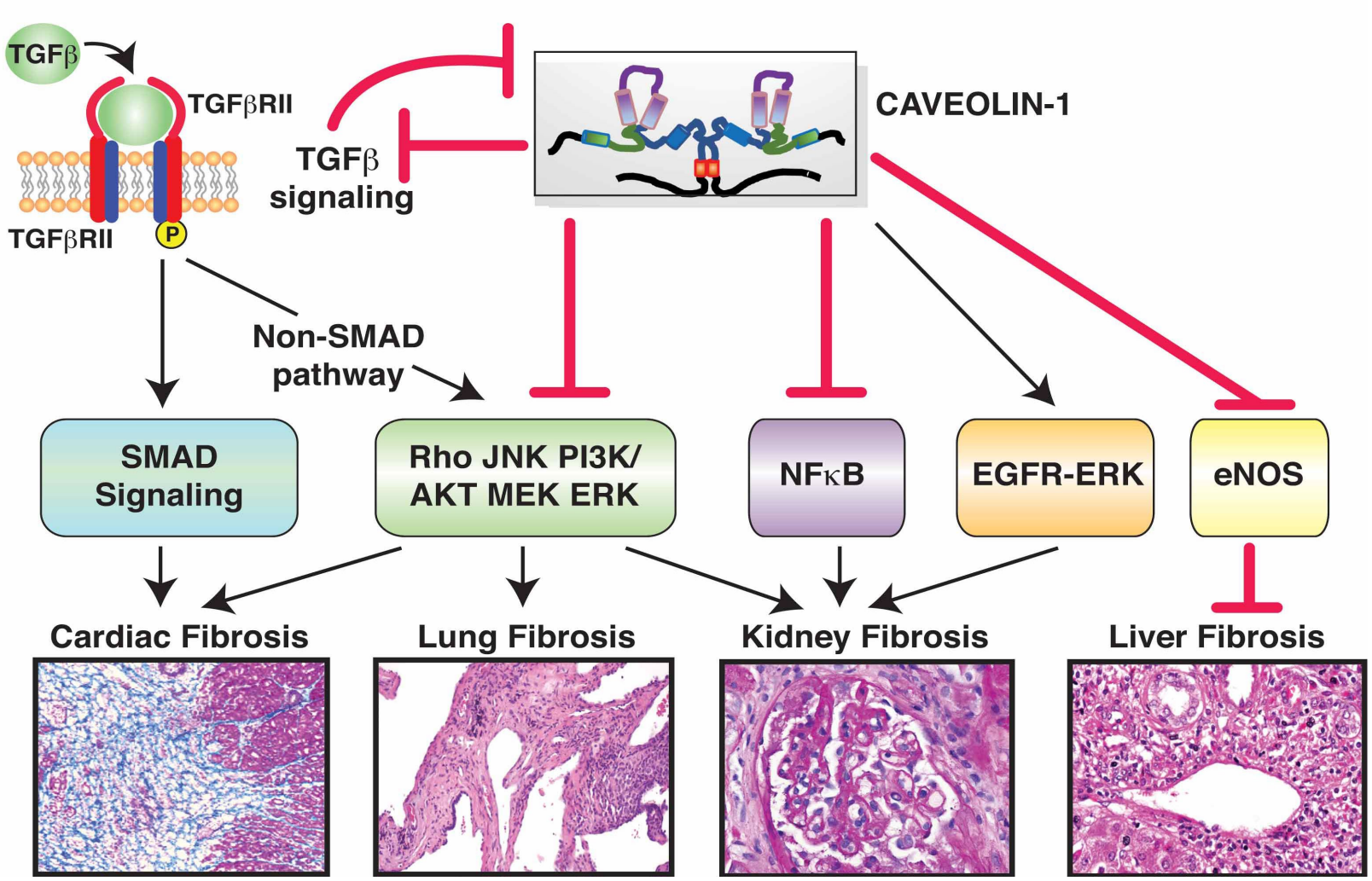
Caveolin-1: a Key Regulator in Fibrosis



Fibroblasts, epithelial cells, endothelial cells, myocytes, adipocytes, & immune cells.



Caveolin-1 Modulates Multiple Fibrosis-Related Pathways

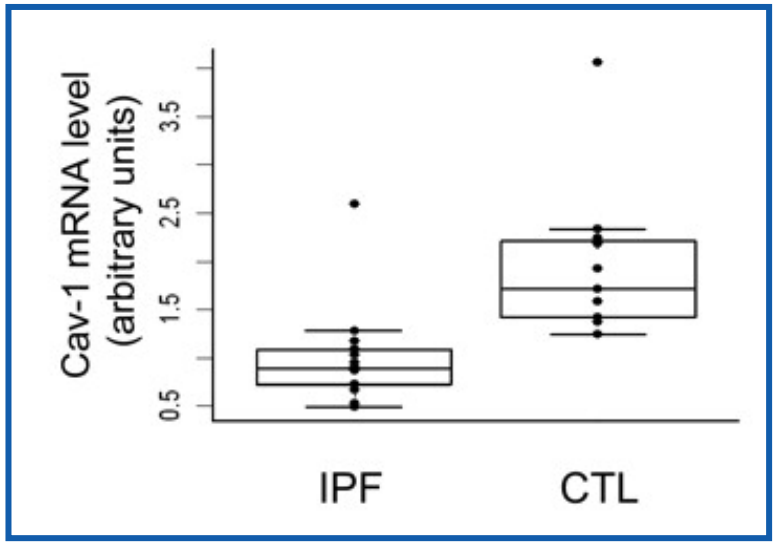


Adapted from Gvaramia et al, Matrix Biology, 2013

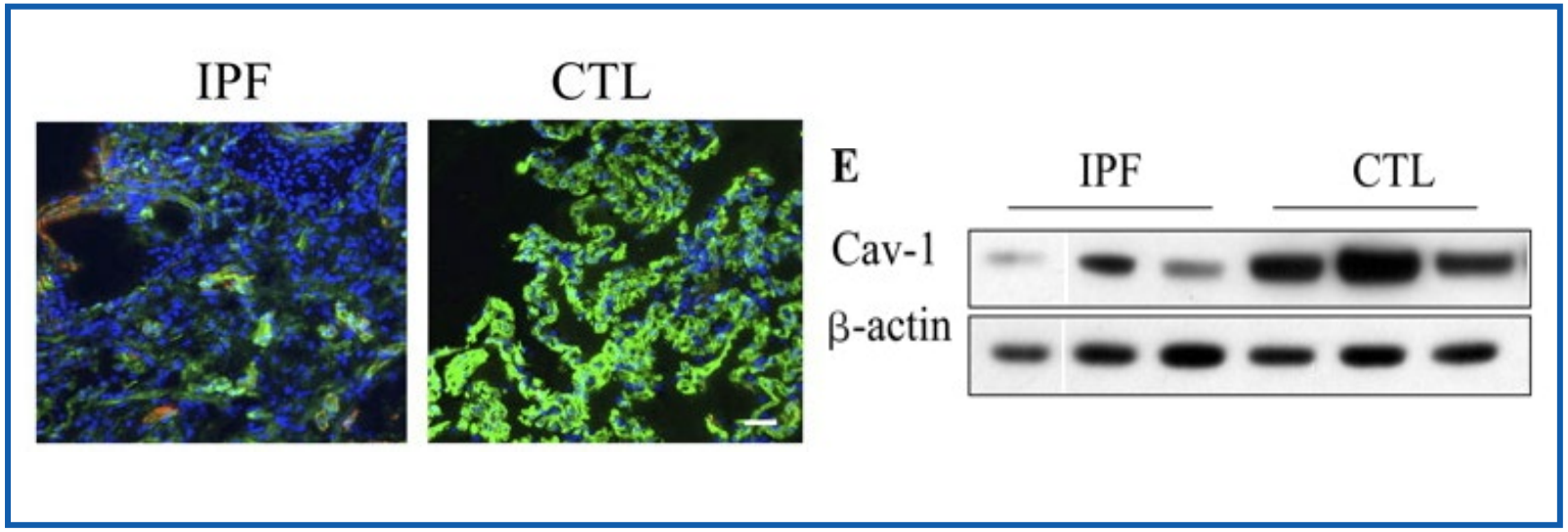
Caveolin-1 is Downregulated in IPF

Caveolin-1: a critical regulator of lung fibrosis in idiopathic pulmonary fibrosis

Xiao Mei Wang,[1] Yingze Zhang,[1] Hong Pyo Kim,[1] Zhihong Zhou,[1]
Carol A. Feghali-Bostwick,[1] Fang Liu,[1] Emeka Ifedigbo,[1] Xiaohui Xu,[2]
Tim D. Oury,[3] Naftali Kaminski,[1] and Augustine M.K. Choi[1]



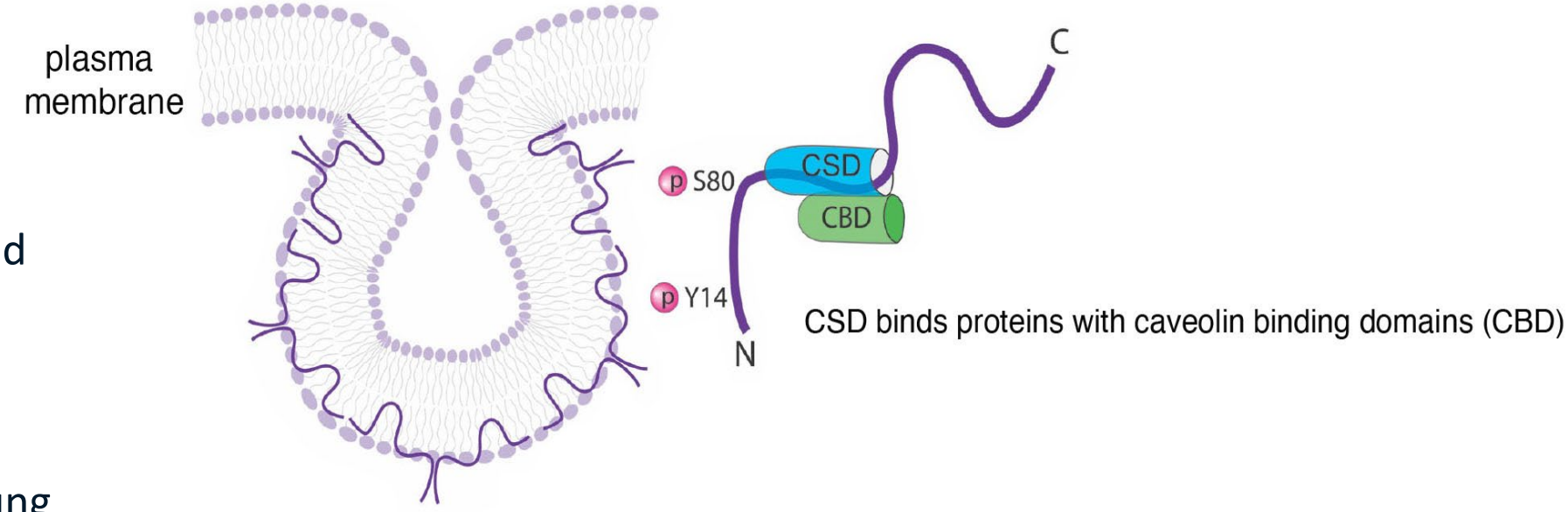
mRNA levels



Protein expression

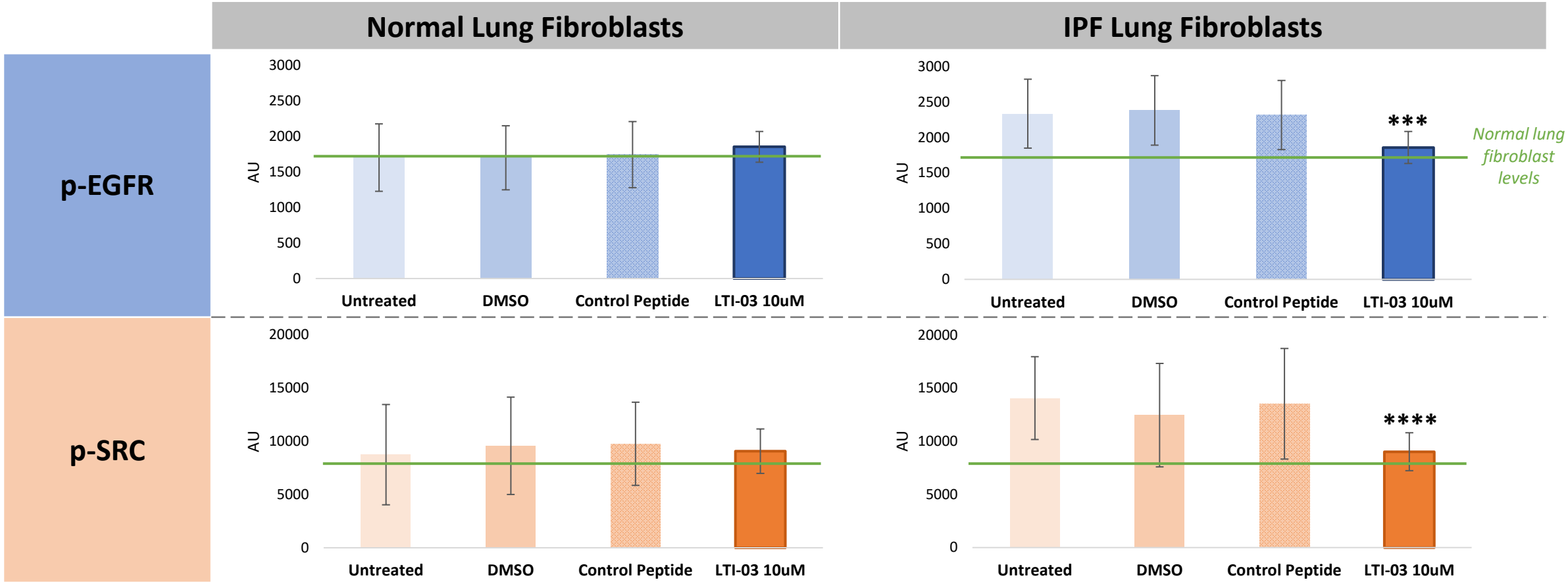
Simulation of Caveolin-1 Activity via CSD Peptide

- LTI-03 is a seven amino acid peptide encompassing a portion of the Cav1 CSD
- LTI-03 is dosed direct-to-lung by dry powder inhaler



For a review on CSD/CBD binding domain list, see: Byrne et. al. PLOS One 2012

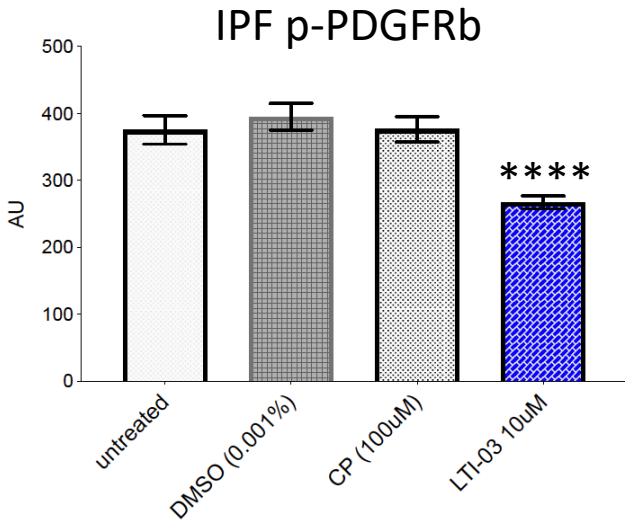
LTI-03 Attenuates Profibrotic Signaling in Vitro

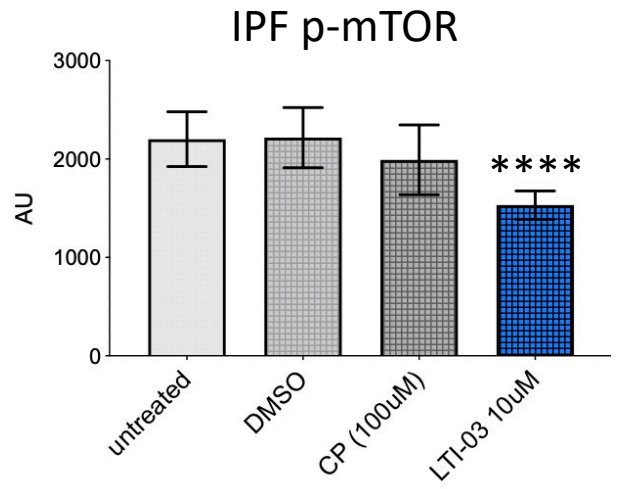


- Factors indicative of aberrant profibrotic signaling were significantly **down-regulated exclusively in fibroblasts derived from IPF patients,** with little to no effects on healthy donor fibroblast lines
- In IPF, LTI-03 appears to **reduce aberrant signaling** by **supplementing cells with the Cav1 signaling domain**

*RPPA assay, Shixia Huang; Baylor College of Medicine; Note(s) Data expressed as mean values and SDs. *p < .05; ** p < .01; ***p<.001; ****p < .0001*

Attenuates RTK and Metabolic Signaling in IPF Fibroblasts





RTK and associated signaling

ALK(D5F3)	*
p-ALK(3B4)(Y1586)	***
c-Jun	*
p-c-Myc(T58)	**
Herb2/ErbB3	***
p-EGFR(Y1173)(53A5)	***
p-MEK (1/2)	****
p44/42 MAPK (ERK1/2)	*
p-PDK1(S241)	****
p-PDGFRb(Y761)	****
p-RafB(S445)	****
p-Ret(Y905)	**
Stat5a	*
p-Stat5(Y694)	***
PI3Kp110a	**
PTEN	*
p-SRC	****
SRC-1	***
YAP	**

Metabolic signaling

AMPKa	****
p-AMPKb1(S108)	****
Deptor	**
LDHA	**
p-mTOR	****
p-Raptor	****
Raptor	**
p-Tuberin	****

Invasion associated markers

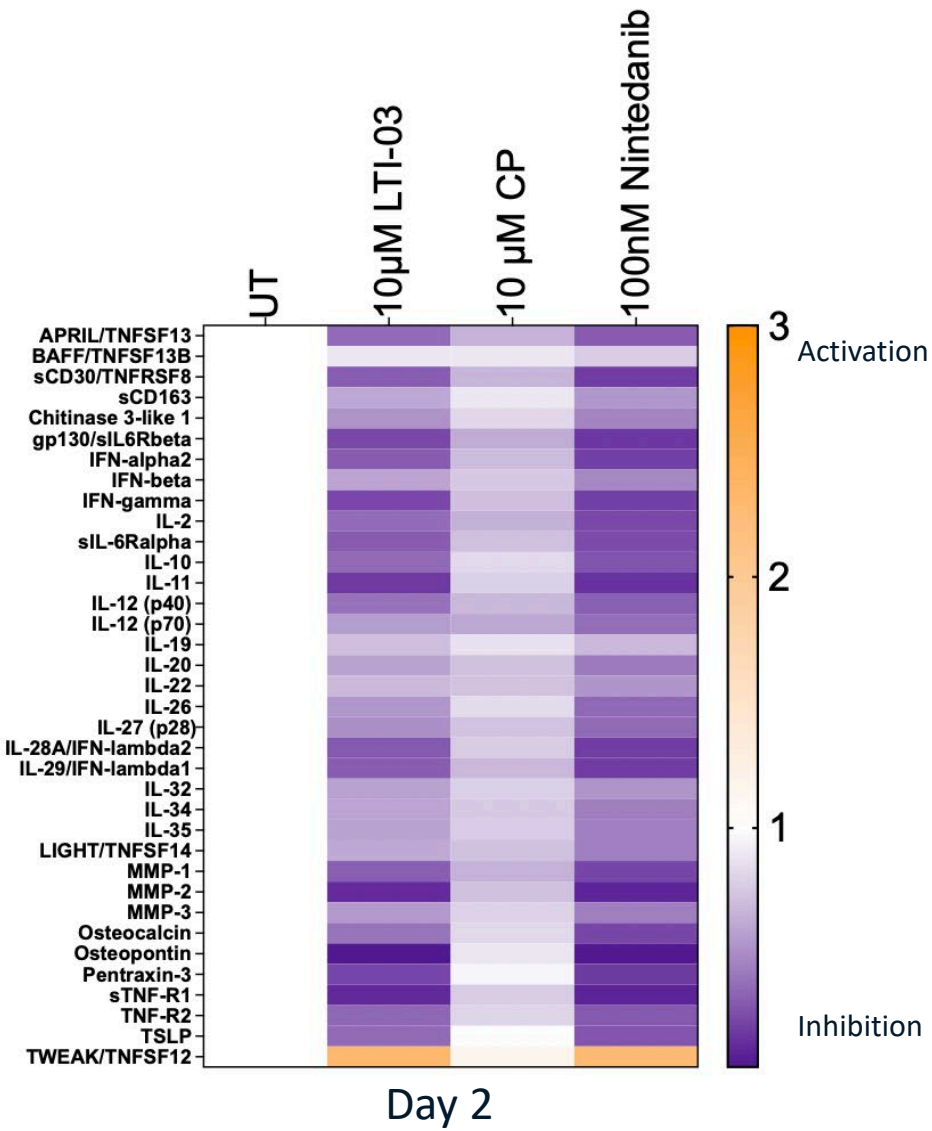
TWIST2	*
Wnt5ab	**

HDACs

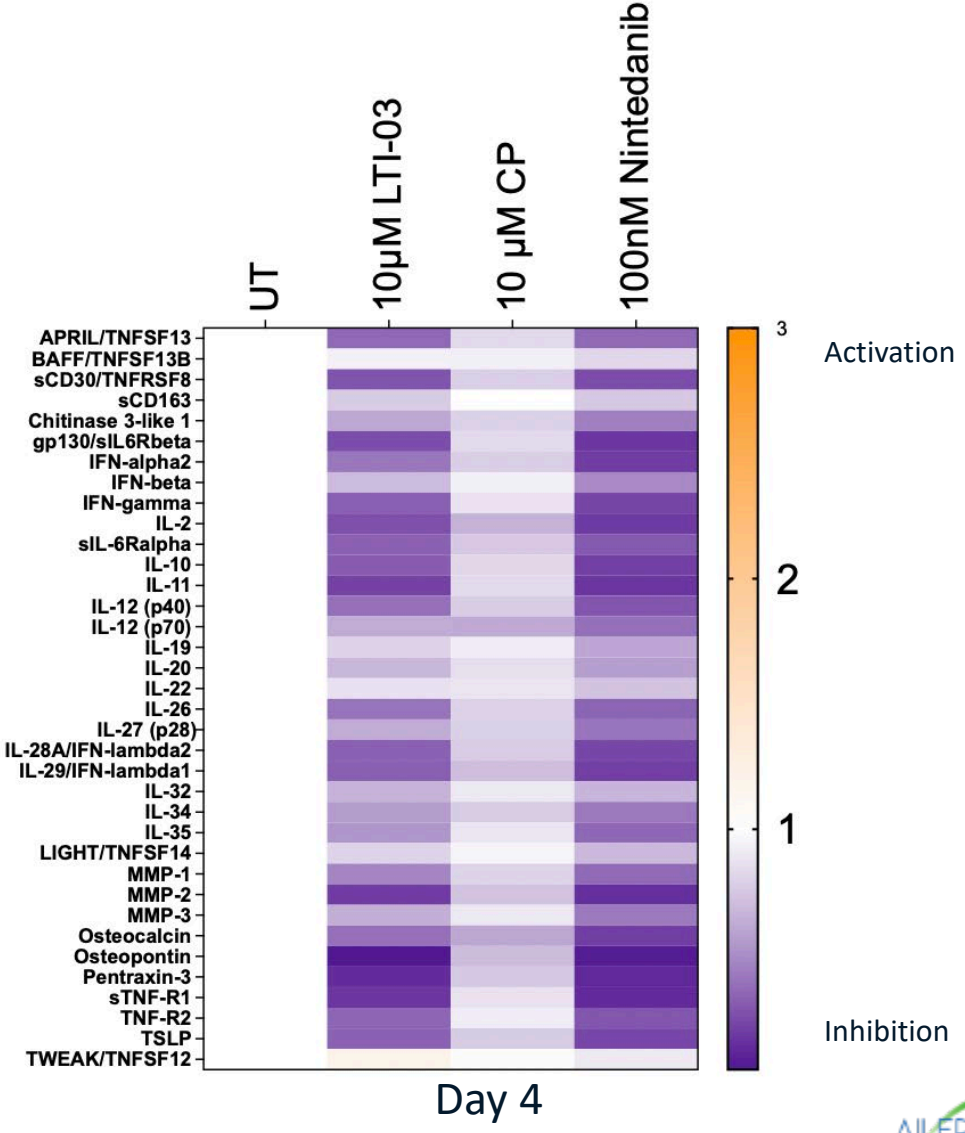
HDAC4	*
HDAC6	***

*RPPA assay, Shixia Huang; Baylor College of Medicine; Note(s) Data expressed as mean values and SDs. *p < .05; ** p < .01; ***p<.001; ****p < .0001*

Anti-fibrotic Activity at Physiologically Relevant Dose (Every 12hrs in Precision Cut Lung Slices (PCLS)—Single Patient Sample)

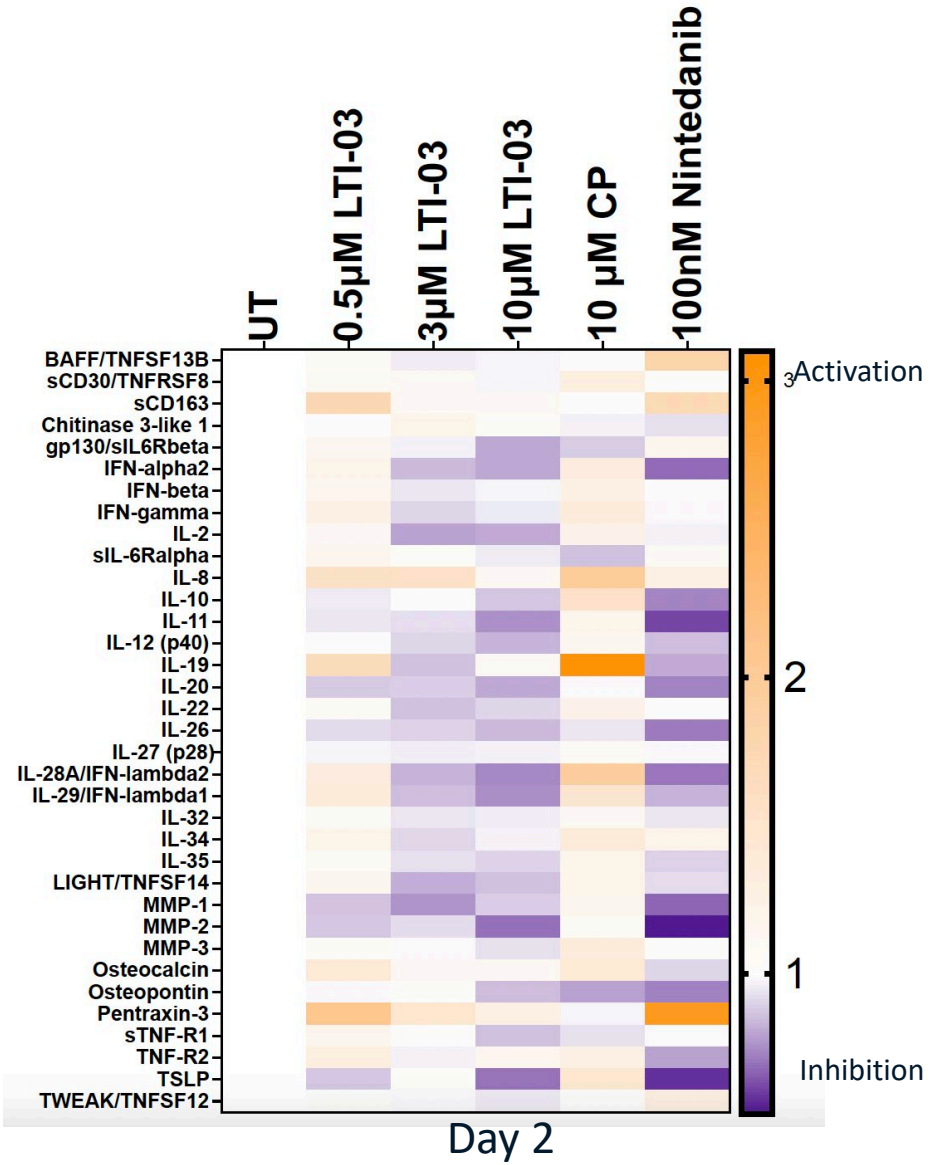


Day 2

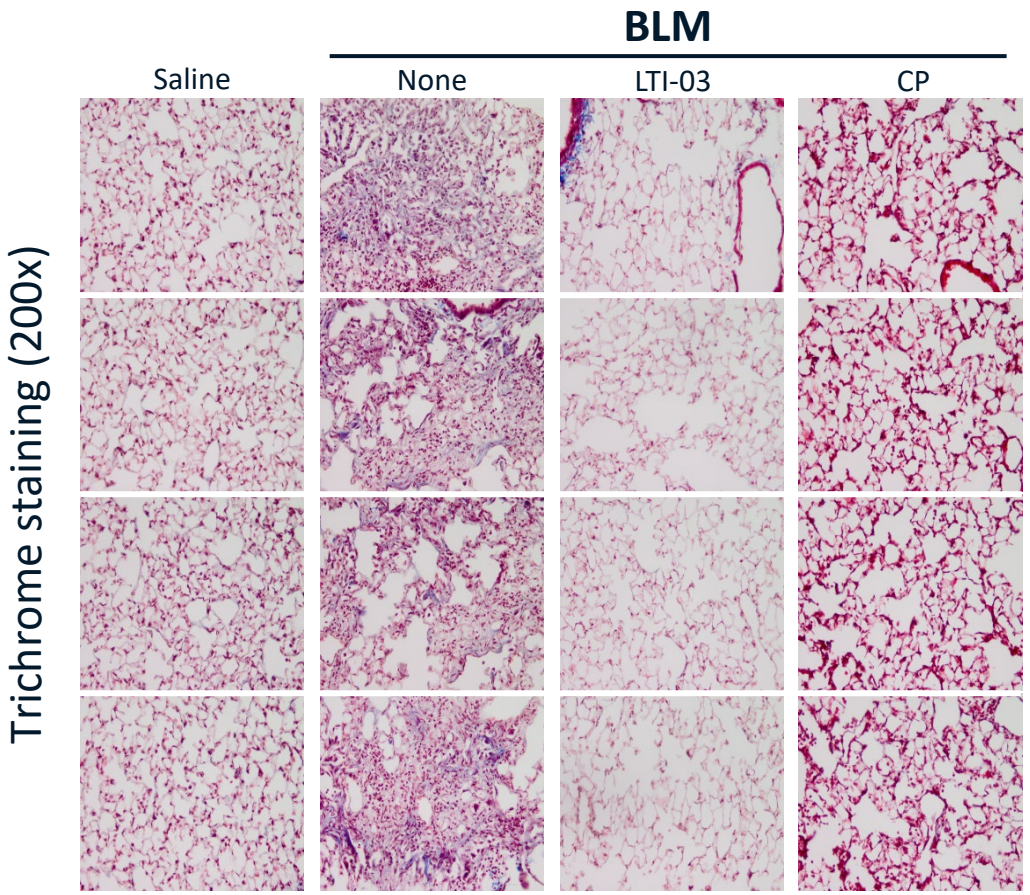


Day 4

Anti-Fibrotic Activity at Physiologically Relevant Dose (Every 12hrs in PCLS — Composite of Six Patient Samples)

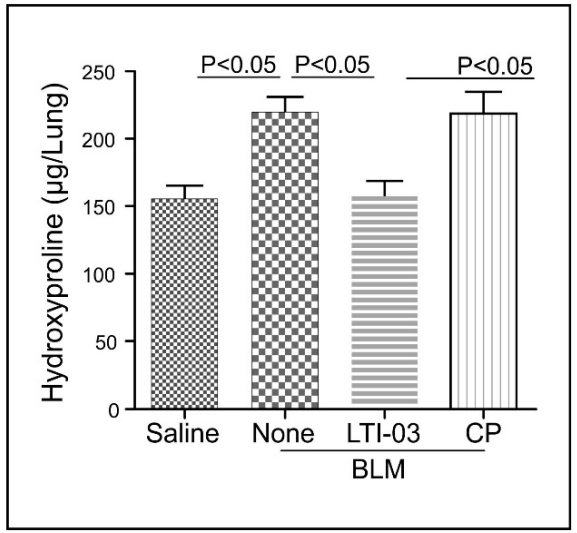


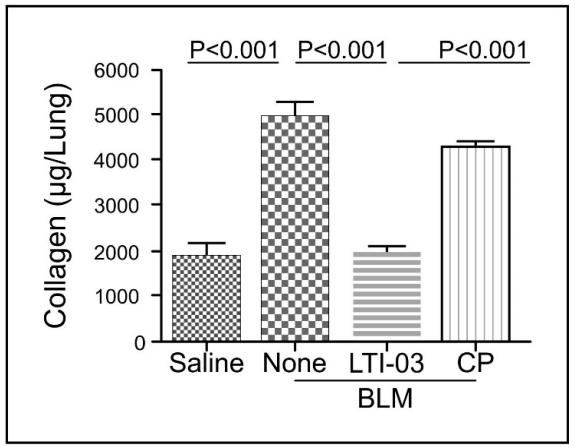
Demonstrated Anti-Fibrotic Properties in the 21-day Bleomycin Mouse Model of IPF



*The **bleomycin mouse model is an established murine model** for characterizing and assessing the impact of novel IPF therapies*

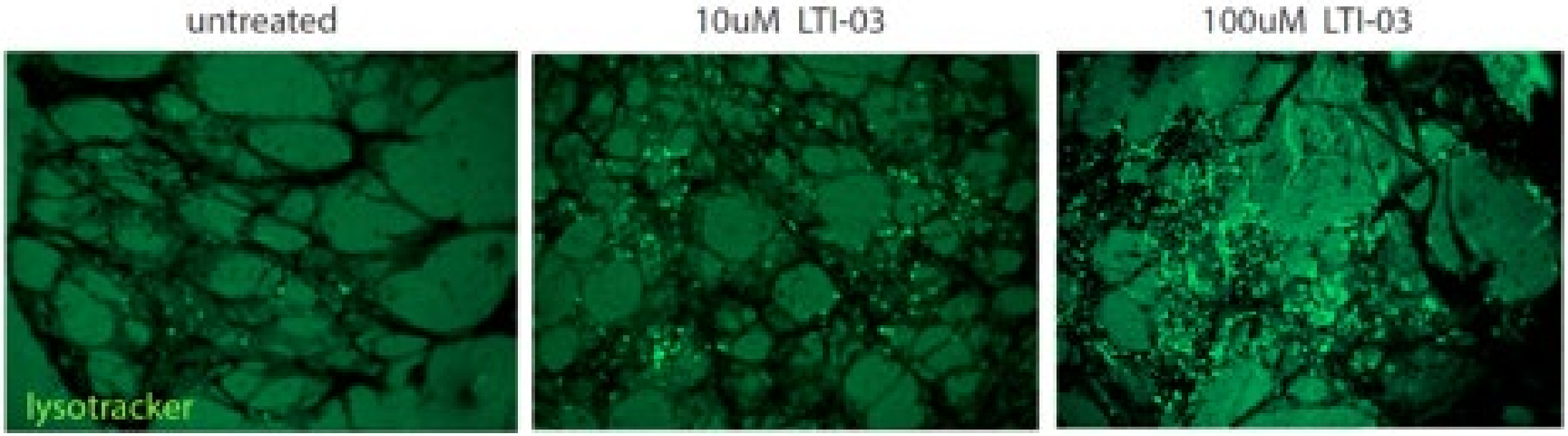
Fibrotic biomarkers





Shetty Lab, Manuscript in revision, Science Translational Medicine

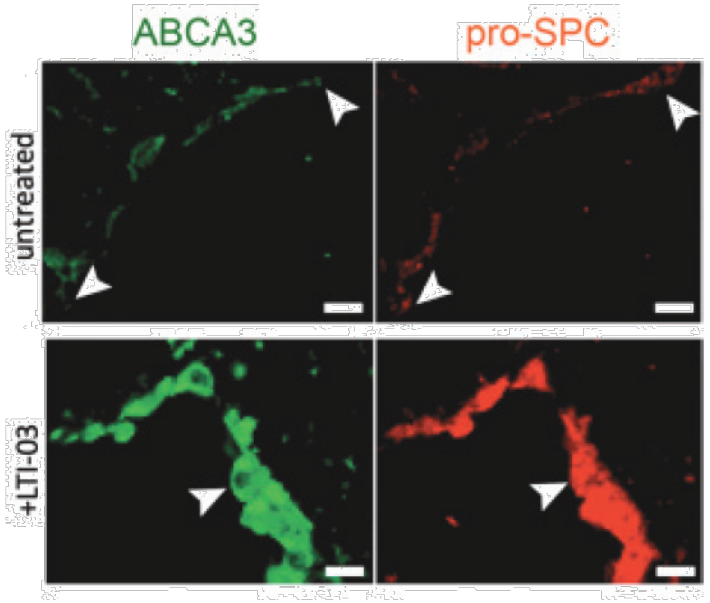
Dose Dependent Increase in Lysotracker Staining in Fibrotic PCLS Model (48hrs following single treatment)



LTI-03 Supports IPF Tissue Epithelium (Fibrotic PCLS Model)

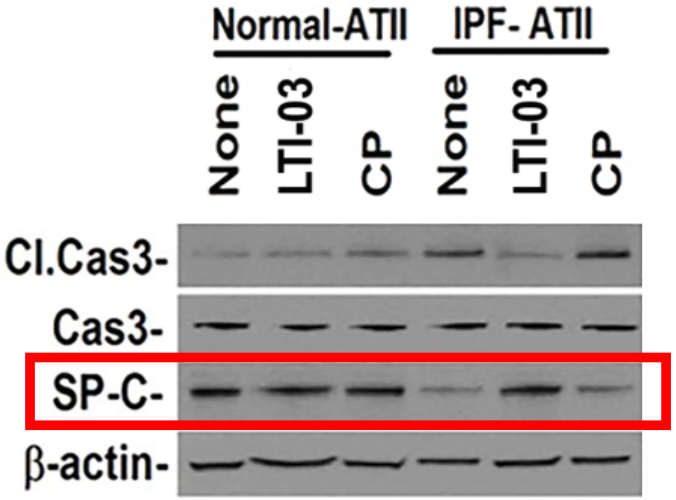
Immunofluorescent Staining for AEC2 Markers

Increases in lysotracker staining also correlated with increases in pro-SPC and ABCA3 gene (the pro-SPC transporter)



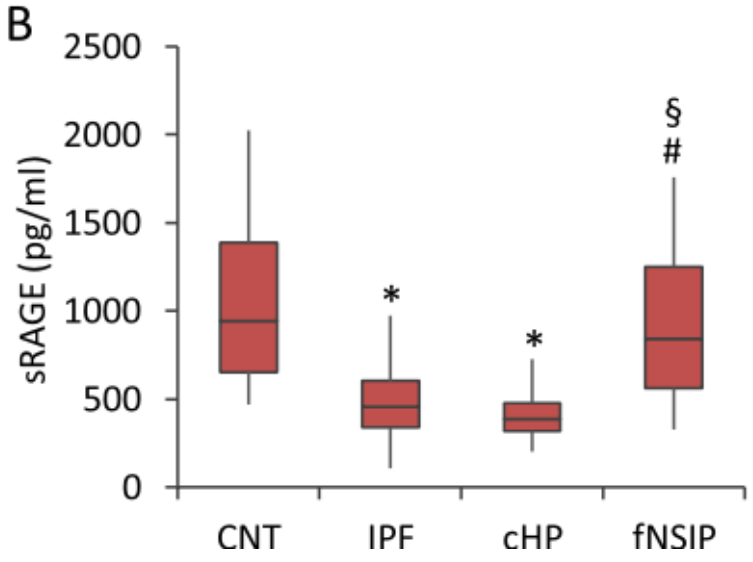
SPC is essential for lung function

LTI-03 also increases levels of SP-C in PCLS IPF Tissue



- In addition to producing AEC1s that make up the majority of the alveolar surface and are **important for proper gas exchange** and ion/water flux, AEC2s **also produce surfactant** that provides for **adequate lung expansion**

Soluble RAGE (sRAGE) is Decreased in Fibrotic Lungs[1]



Machahua *et al. Respiratory Research* (2016) 17:144
DOI 10.1186/s12931-016-0460-2

Respiratory Research

RESEARCH Open Access

Increased AGE-RAGE ratio in idiopathic pulmonary fibrosis

Carlos Machahua[1,2], Ana Montes-Worboys[1,2,3], Roger Llatjos[4], Ignacio Escobar[5], Jordi Dorca[1,2,3], Maria Molina-Molina[1,2,3*†] and Vanesa Vicens-Zygmunt[1,2†]

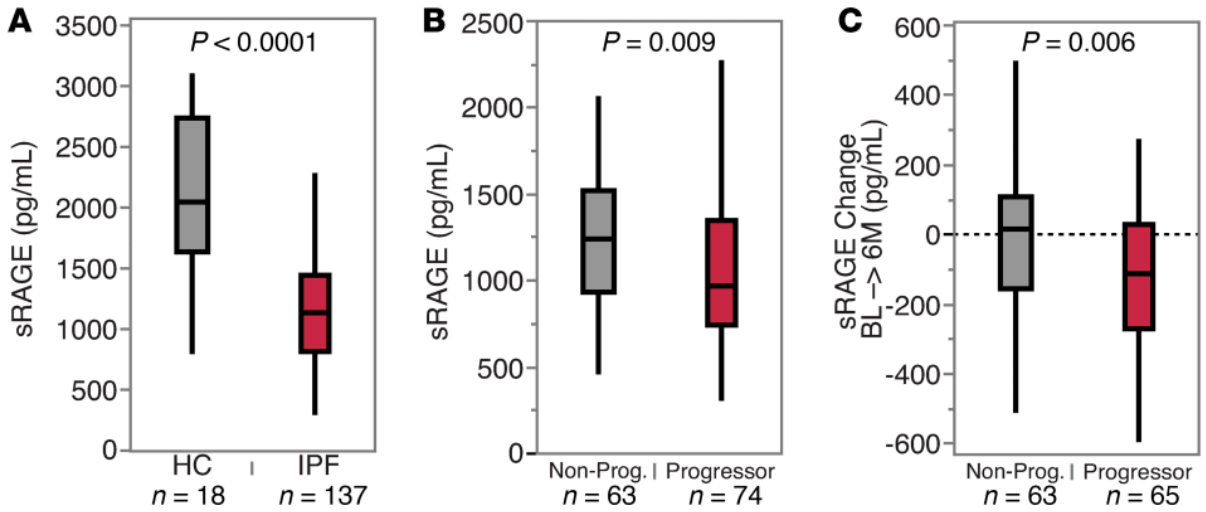


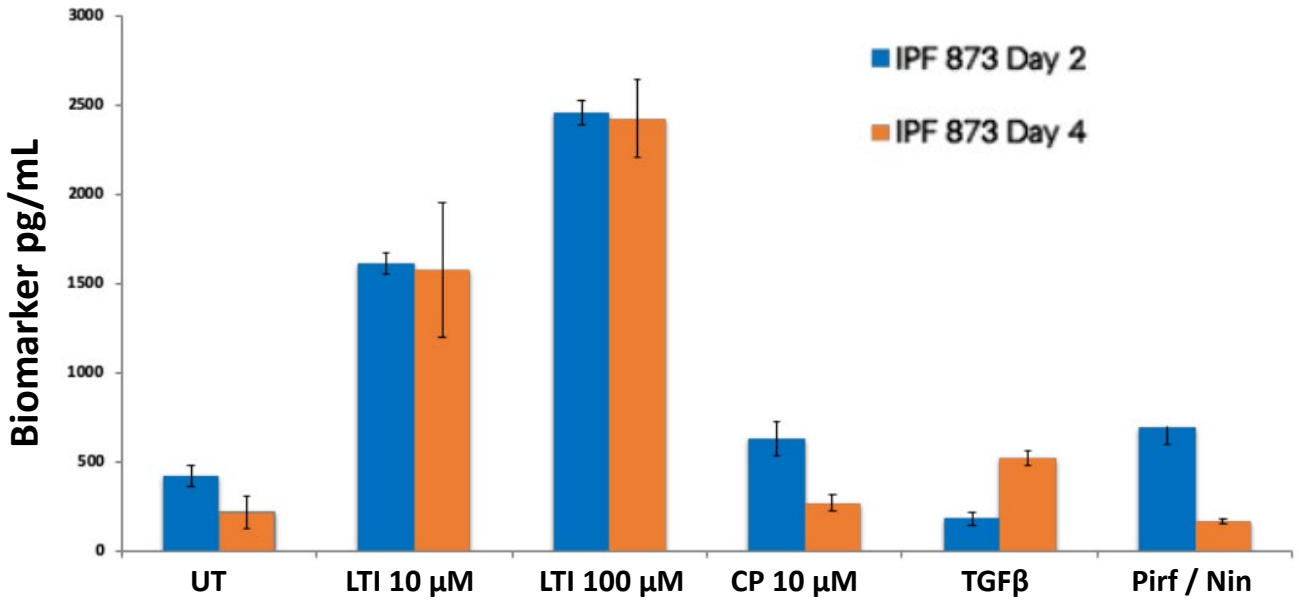
Figure 7. Decreased blood levels of RAGE were associated with more rapid disease progression in IPF patients. (**A**) Soluble RAGE levels in plasma from IPF patients and healthy controls. (**B**) Plasma sRAGE at baseline in IPF patients, dichotomized by disease progression (defined as loss of ≥10% predicted forced vital capacity [FVC] or death) from baseline to 1 year of follow-up. (**C**) Change in plasma sRAGE levels from baseline to 6 months in IPF progressors and nonprogressors. Statistical significance between the groups was determined by Wilcoxon rank sum test. BL, baseline.

[1]Machahua, C., Montes-Worboys, A., Llatjos, R. et al. Increased AGE-RAGE ratio in idiopathic pulmonary fibrosis. Respir Res 17, 144 (2016).

Novel Prognostic Biomarker Data Supports LTI-03 Protection of Epithelial Cells

Biomarker Correlates with LTI-03 Impact in PCLS

Administration of LTI-03 in the PCLS system increased the soluble protein biomarker, sRAGE, while currently approved therapies had negligible effects on sRAGE levels



***Low levels** of sRAGE at diagnosis predict poor survival in IPF*[1]

The increase in sRAGE** provides further evidence of increased AEC2 survival, leading to **greater AEC1 production** and thus **overall epithelial cell survival

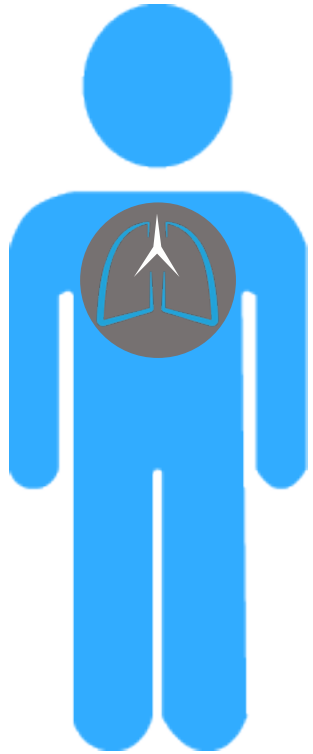
*Ability to **measure sRAGE in broncho-alveolar lavage fluid and blood** makes it a potentially **useful biomarker***

[1]Machahua, C., Montes-Worboys, A., Planas-Cerezales, L. et al. Serum AGE/RAGEs as potential biomarker in idiopathic pulmonary fibrosis. Respir Res 19, 215 (2018).

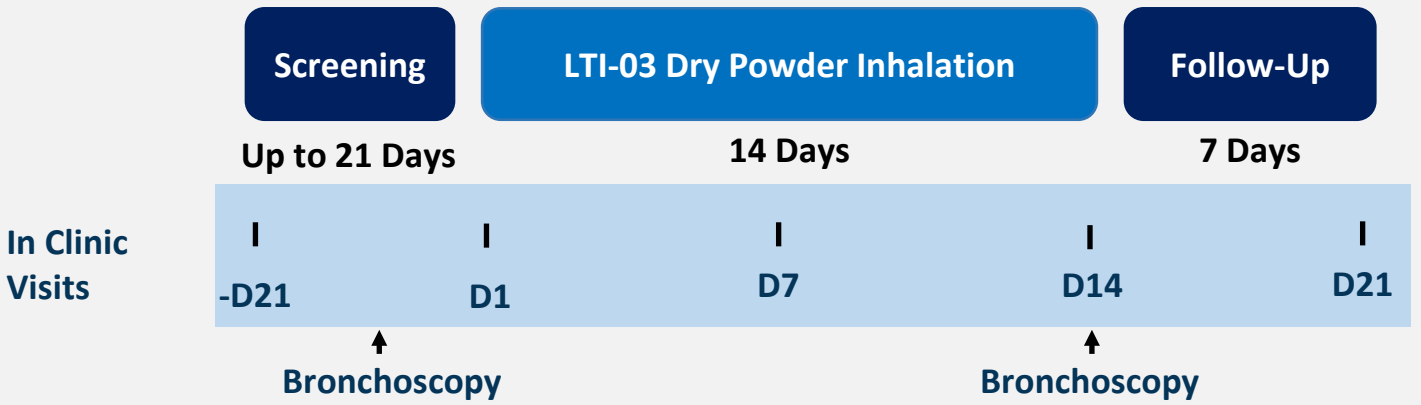
Phase 1a Clinical Trial Design (Status: Complete)

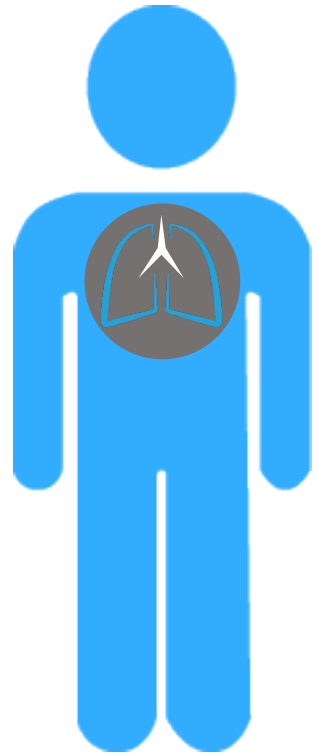
Healthy Human Volunteer Clinical Trial

- Objectives
  - Primary – Safety and Tolerability
  - Secondary – Pharmacokinetics
- Design
  - Single Ascending Dose (32 subjects / 3 doses)
    - Doses: 20mg, 40mg, 80mg
  - Multiple Ascending Dose (40 subjects / 5 doses)
    - Doses: 2.5mg, 5mg, 10mg, 20mg, 40mg



Phase 1b Clinical Trial Design (Status: In Process)





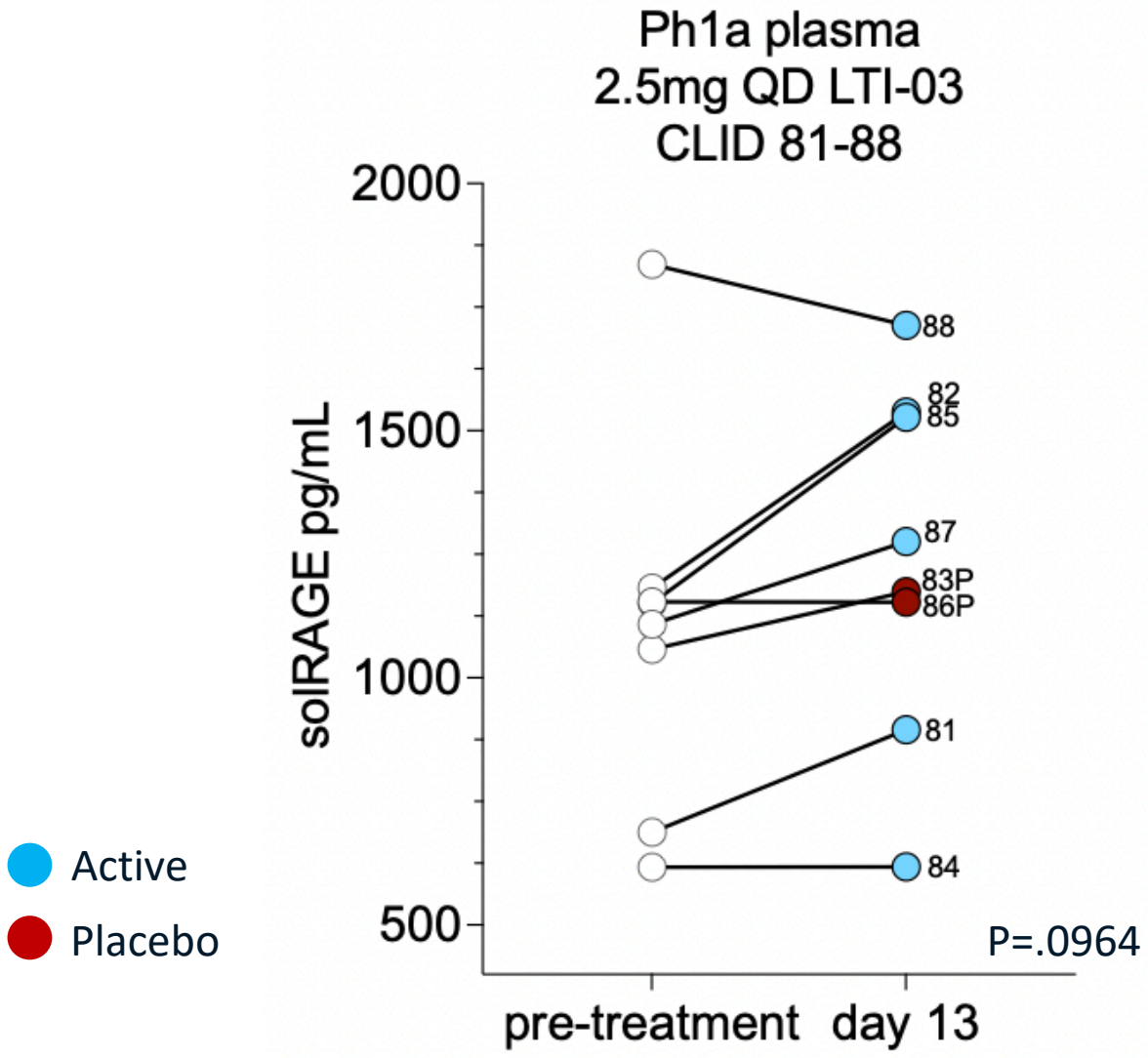
Study Design

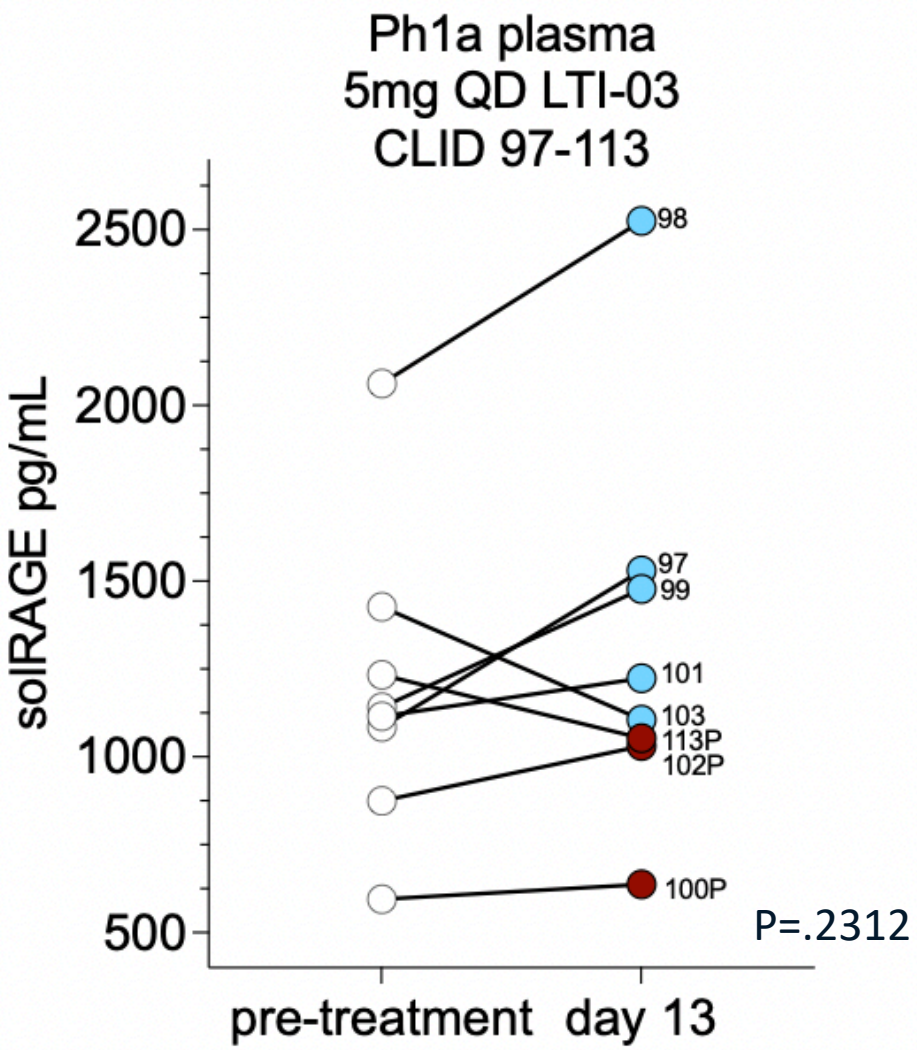
- IPF diagnosis ≤ 3 years; no previous antifibrotic therapy w/in 2 months of baseline
- 24 patients total (18 active, 6 placebo)
  - Low (2.5mg BID) and high (5mg BID) dose cohorts, sequential daily dosing for 14 days
- Bronchoscopy at screening and Day 14
- Primary endpoint: Safety/tolerability
- Key exploratory endpoint: Biomarkers (blood, BAL, brushings)

Phase 1b Clinical Trial Biomarkers

Sample source/Indicator of	epithelial damage/repair	fibrosis	inflammation	thrombosis
Peripheral blood cells		p-AKT		
Platelet rich plasma (PRP)	CYFRA 21-1, SP-D, CA-19-9, KL-6, sRAGE, Galectin 7	MMP-7, Tenascin C (TNC), Periostin, IL-11, MYDGF, MMP-2	CCL18, CXCL13, sICAM1, IL-11, sCD163, CXCL7	PAI-1
Bronchoalveolar lavage	Galectin 7, surfactant protein C, sRAGE	MYDGF, MMP-2, TNC, MMP-7, periostin, IL-11	CCL18, CXCL13, sICAM1, IL-11, sCD163, CXCL7	PAI-1
Deep bronchial brushings		p-SMAD2/3		

sRAGE Preferentially Increased in Phase 1a HNV Plasma Analysis





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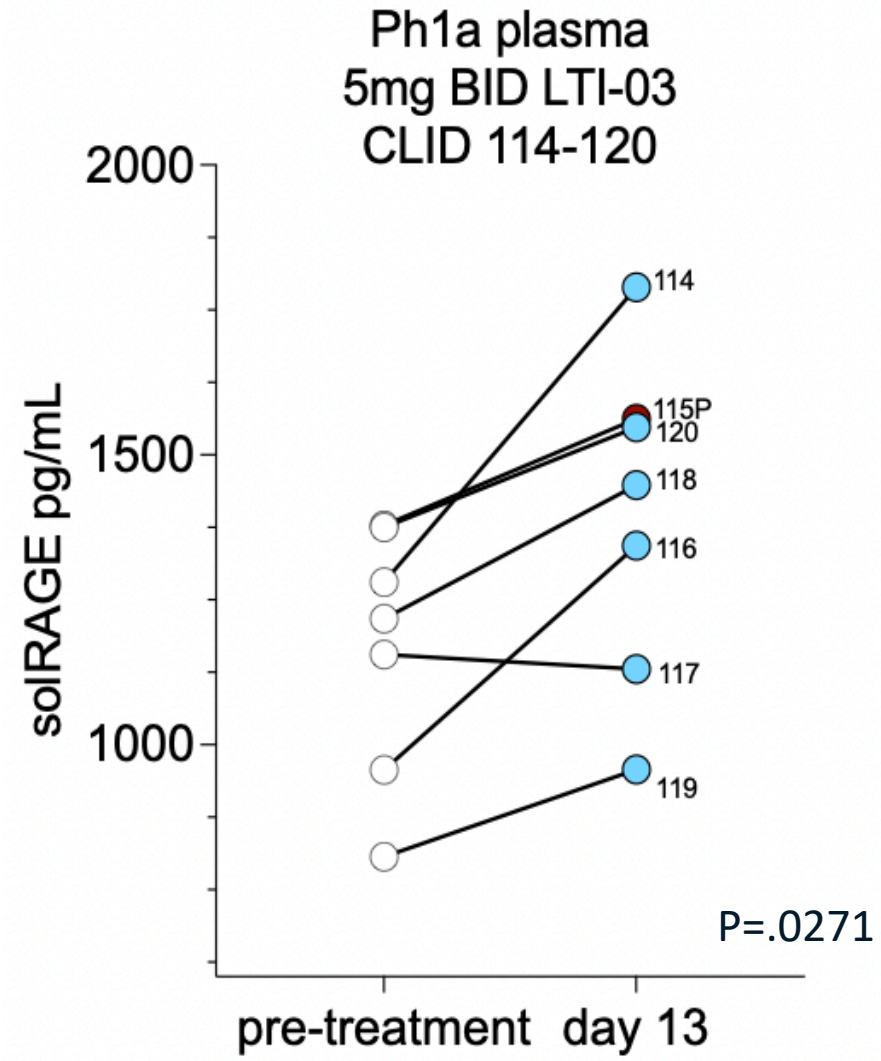
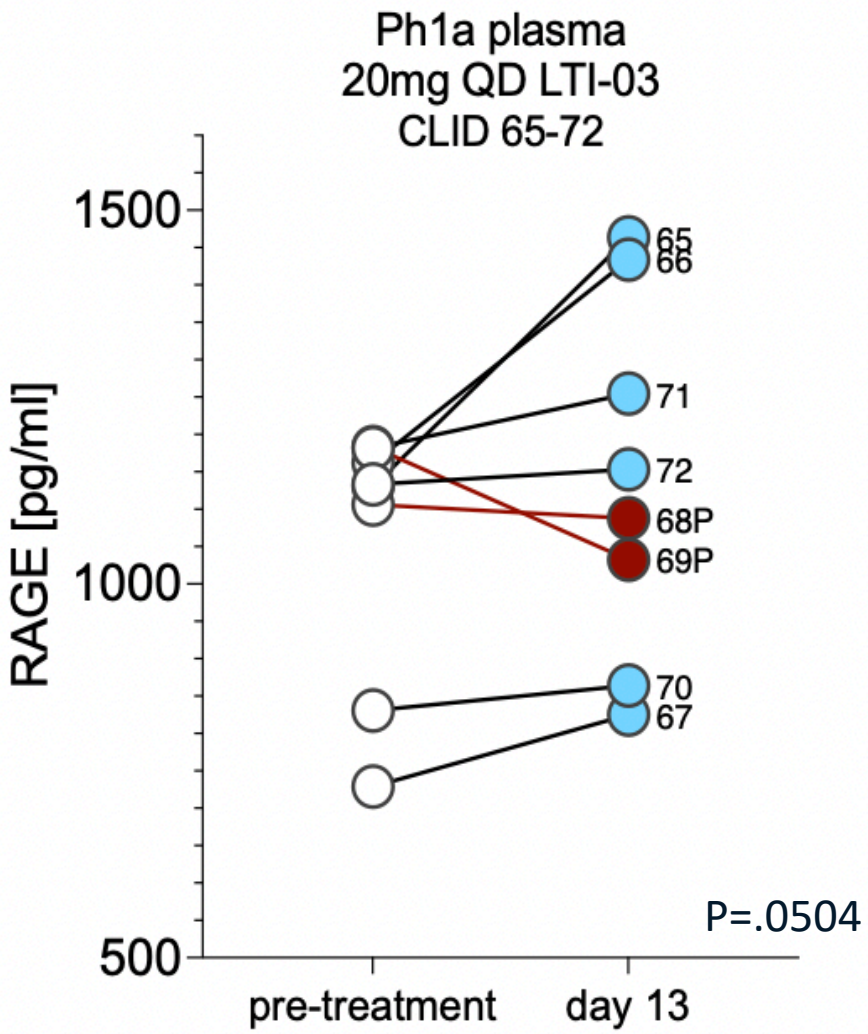
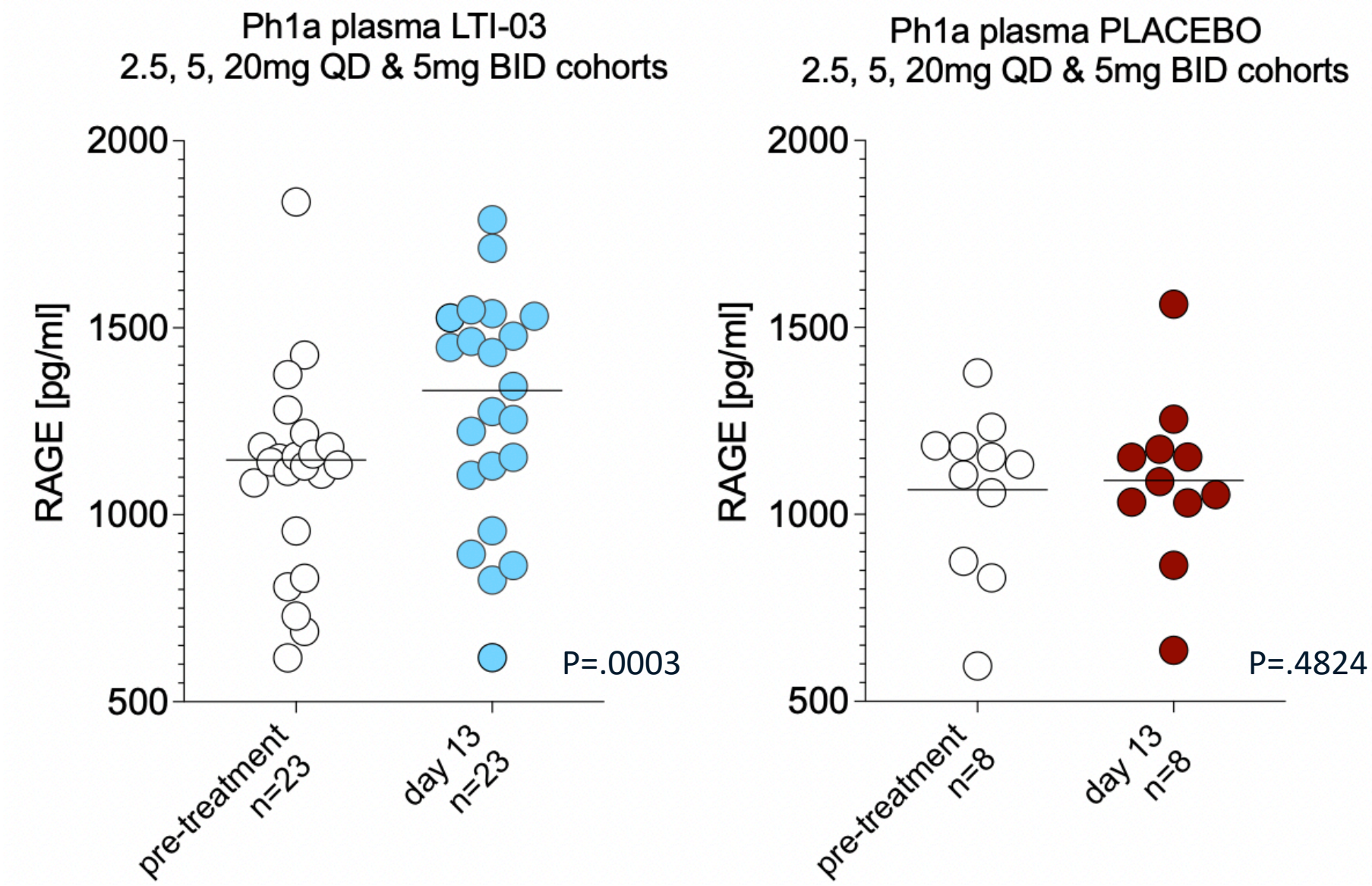




Active

Placebo

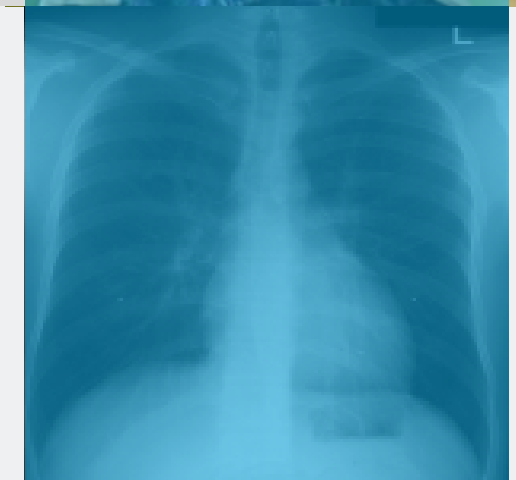
All Dose Groups – Stat Sig Increase in sRAGE from Day 0 to Day 13 Treatment



Active

Placebo

LTI-01: Enzyme Therapy for Loculated Pleural Effusions



LTI-01 is a PAI-1 Resistant Plasmin Activated Proenzyme for Loculated Pleural Effusions

- Hospital indication – significant population from pneumonia patients with an estimated population of over 60,000[1] patients in the US alone annually
- Current treatment options are surgery and/or off label fibrinolytic use
  - Surgery is expensive and invasive with longer hospital stays
  - Fibrinolytic use is used off label and has safety concerns
- LTI-01 has a potential safety benefit and dosing advantage over off label fibrinolytics
  - Improved drainage and fewer rescue treatments for patients treated with LTI-01 vs placebo in Phase 2a trial
- Physician market studies support the use of fibrinolytics and need for on-label treatment alternative to surgery
- Partnership with Taiho Pharma for development and commercialization rights in Japan

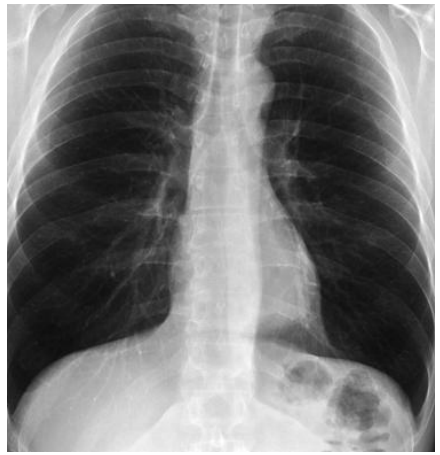
1 Management estimate.

There Are No Approved Drug Treatments for Loculated Pleural Effusion

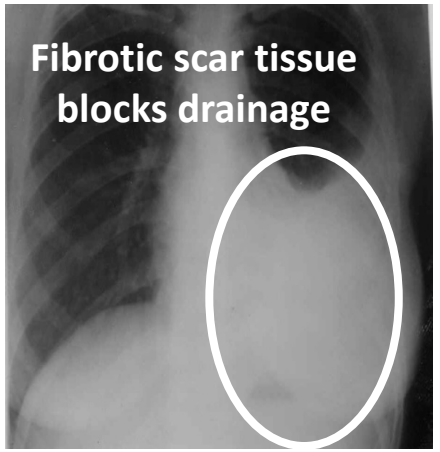
Disease Overview

- LPE occurs when fibrotic scar tissue forms in the pleural cavity, preventing effective drainage of fluid
- LPE is a frequent pneumonia complication in the elderly with a ~20% mortality rate
- LPE is managed with tPA/DNase (off-label) and/or surgery (costly and invasive)

Healthy Lungs



Loculated Pleural Effusion



Current treatment options for patients with LPE are limited

Surgery

91% Effective[1]

- Long hospital recovery (20-25 days)
- Risk of pain and complications
- Increased morbidity
- **Invasive and expensive**

Off-Label Fibrinolytics

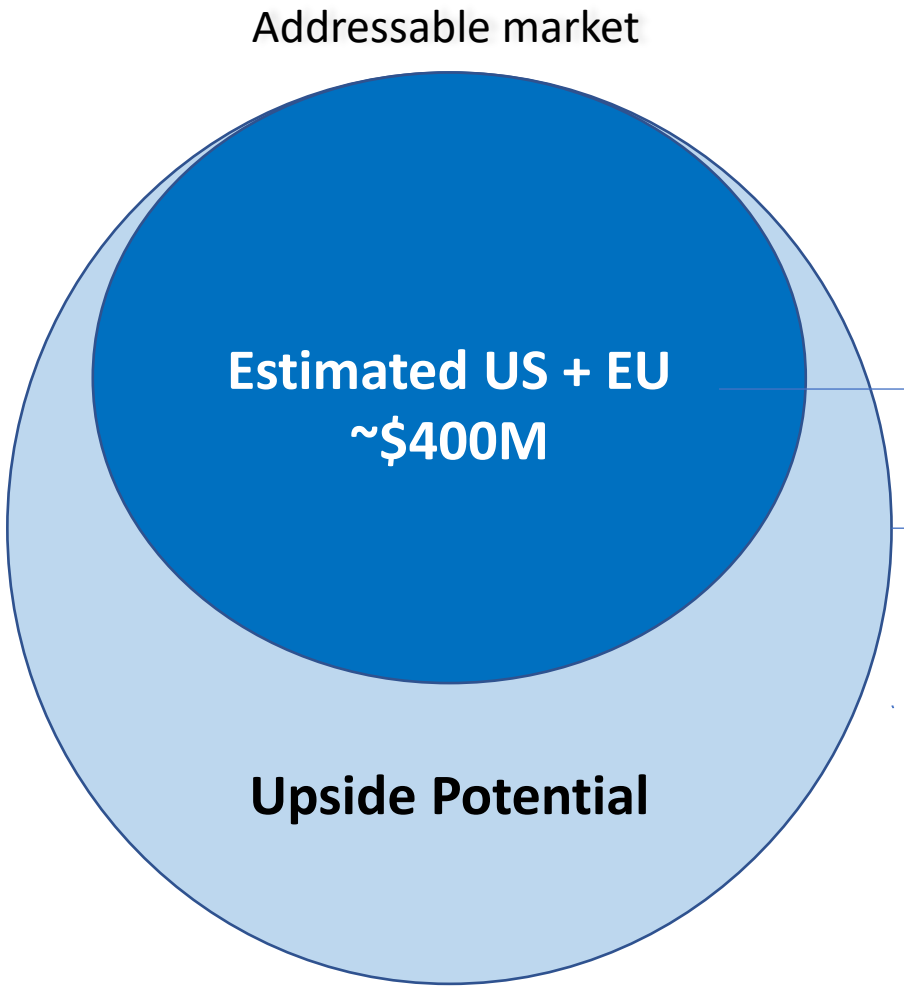
56% Effective[2]

- Less costly and risky than surgery
- Many patients still need surgery
- **Not FDA approved**

[1]Wait MA, Sharma S, Hohn J *et al.* A randomized trial of empyema therapy. *Chest* 1997; **111**: 1548– 51.

[2]Retrolysis Study raw data, Jason Akulian

Sizeable US and EU Commercial Opportunity with Potential Upside



Current US and EU Opportunity

- 30,000 US fibrinolytic patients
- Up to 30,000 additional US LPE patients
- tPA/DNase priced at $6,700 per patient in US
- Estimate similar EU market opportunity to US market

Key Catalyst: Substitution of tPA/DNase with on-label alternative

Upside Market Potential in the US and EU

- Premium Pricing
- Ability to drive beneficial clinical and economic outcomes

Key Catalyst: On-label therapy with clear efficacy, safety and dosing benefits

+ Japan partnership with TAIHO PHARMA

Source: Management estimates, industry publications and MME market access research study for Lung Tx.

LTI-01's Mechanism of Action Leads to Additional Inhibition Resistant Complexes Compared to tPA-DNase

Challenges	Solution	LTI-01 Potential Benefits

• Plasminogen activator inhibitor 1 (PAI-1) is increased in pleural injury leading to decreased fibrinolysis
 	
• PAI-1 is highly variable by patient and rapidly quenches activated fibrinolytics such as tPA and two-chain uPA
	• ***LTI-01 (scuPA) is a proenzyme that is uniquely PAI-1 resistant***
	
	• Confers potential advantages for both duration of activity and safety
		• **Improved Efficacy and Dosing**
		  • Longer fibrinolytic activity increases duration of fibrinolysis
		
		• **Improved Safety**
		  • Non-systemic
		  • Relatively slow onset of fibrinolytic activity
		  • Reduced bleeding and pain risk

Phase 2a Clinical Trial Measured LTI-01's Potential to Avoid Surgical Referral and Improve Outcomes

Placebo-controlled, Dose-ranging Study Evaluating LTI-01 in Patients With Loculated Pleural Effusions

Enrollment	*Start Date*	*Last Pt Date*
N = 40	Sept 2020	Feb 2022

Inclusion Criteria

- Clinical presentation of pleural complicated pleural effusion, empyema, or other pleural infection
- Presence pleural fluid that requires drainage and is infected
- Failure to adequately drain pleural fluid ≥3 hours post insertion of chest tube

QD x 3 Days Intrapleural Dosing

R

Treatment Arms

- 1,200,000 IU LTI-01
- 800,000 IU LTI-01
- 400,000 IU LTI-01
- Placebo

Endpoints

Primary

- **Incidence of Referral to Surgery or Death at 7 Days**

Secondary

- **Change in Pleural Opacity from Baseline**
- **Length of Hospital Stay**
- **Incidence of Pleural Hemorrhage**
- **Incidence of Pain**

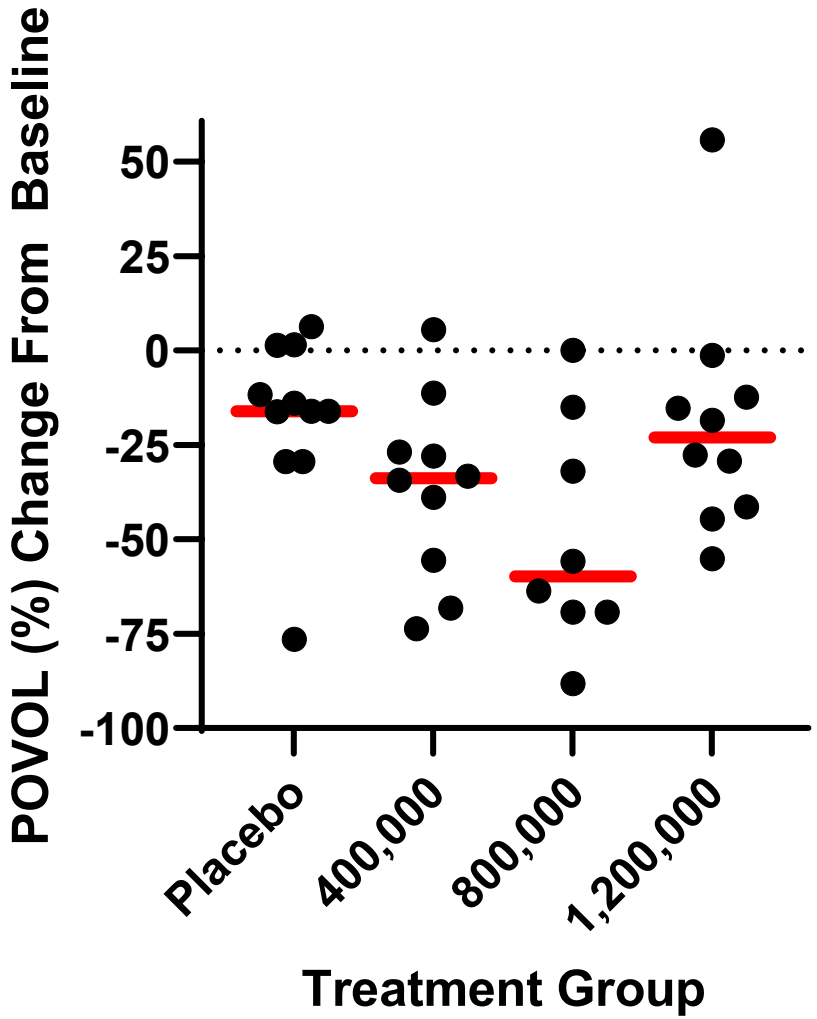
Demonstrated Drug Effect in Randomized, Placebo Controlled Trial

- Enrollment limited by Covid pandemic (treating physicians are interventional pulmonologists in hospital setting)

Treatment Success	LTI-01 (400,000 U) N=10	LTI-01 (800,000 U) N=9	LTI-01 (1,200,000 U) N=10	All LTI-01 N=29	Placebo N=11
No Rescue Therapy – mITT, n (%)	**6 (60%)**	**5 (55.5%)**	**4 (40%)**	**15 (51.7%)**	**3 (27.3%)**
Odds Ratio (OR)	0.26	0.31	0.49	0.33	
P value (vs Placebo)	0.147	0.221	0.463	0.161	
Primary end pt - Per Protocol, n (%)	7 (77.7%)	6 (66.6%)	5 (55.5%)	18 (66.6%)	7 (63.6%)
Primary end pt - mITT, n (%)	7 (70%)	6 (66.6%)	5 (50%)	18 (62.1%)	7 (63.6%)

The Modified Intent-to-Treat (ITT) population consists of all subjects who are randomized in the study, received any doses of study medication and have at least one post baseline efficacy assessment
Difference in primary end point and no rescue therapy is patients that did not meet criteria of treatment failure checklist but were deemed to need rescue by physician.

Meaningful Effect of Secondary Endpoint Pleural Opacity % Volume Change (POVAL)



Note: Red line is median

Note: 800,000 U outlier excluded with esophageal perforation

- 400,000 U and 800,000 U dose groups showed a significant difference from placebo ($p < 0.05$, rank based ANCOVA)
- Significant correlation between POVOL change and treatment failure defined by requiring rescue therapy (p-values <0.05) highlighting the clinical relevance of POVOL change.

Physician and Pharmacy Directors Research Supports LTI-01's Commercial Opportunity

Primary Research Reactions to LTI-01 Profile

(n = 20 Interventional Pulmonologists, 20 Hospital Pharmacy Directors)

Limited Perceived Utility — *High Perceived Utility*

Physician Perceived Advantages

LTI-01 Could Replace SOC	*LTI-01 likely to replace tPA/DNase as first line therapy if proven comparable/superior*
Favorable Safety	*The reduction in risk of bleeding and pain compared to off-label tPA/DNase was viewed a definite benefit*
Superior Dosing Convenience	*tPA and DNase are dosed twice daily, one hour apart, representing significant burden compared to LTI-01's once daily dosing*

"Its success rate, meaning the resolution of ongoing SIRS and pleural sepsis… that's outstanding. That's much greater than tPA alone… That's a pretty significant upside proposition."
– Hospital Pulmonologist

Pharmacy Director Perceived Advantages

Product Indicated Specifically for LPE	*Having an FDA approved drug for the particular diagnosis would be extremely important. Comparative data would be beneficial*
Reduction in Hospital Days	*Given the significant costs associated with each day in hospital, HPDs viewed even a 1-2 day reduction in hospital stay as meaningful and likely to drive formulary inclusion*

'If it was anything better than tPA/DNase and reducing hospital stay by like 1 or 2 days, I think that would be meaningful…"
-Hospital Pharmacy Director

Sources: Third party research from MME

Positioned for Commercial Success as the First Approved Therapy with Once-Daily Dosing and Premium Pricing Potential

Market Adoption

- Physicians prefer fibrinolytics to surgery, thus 50-60% off-label use
- At parity, **LTI-01 expected to replace off-label treatment** due to: first to approval and once daily dosing

Premium Pricing & Expanded Use

- LTI-01 is anticipated to have a **higher price & expanded use** compared to tPA/DNase
- **Superior clinical data** may lead to **further premium pricing** potential and expanded use

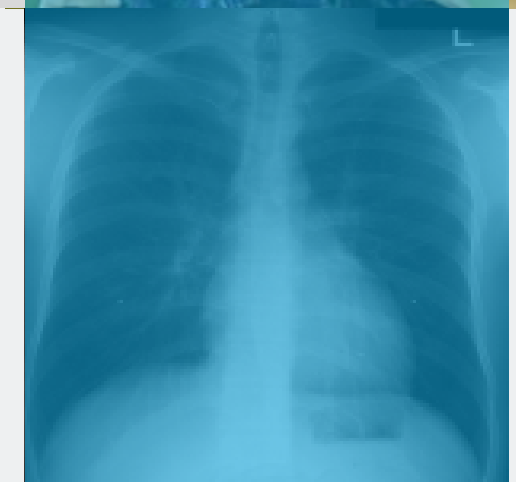
Highly Targeted Sales Efforts

- **Less than 5,000 US hospitals treating LPE**

LTI-01 is optimally positioned for commercial success given **1)** potential to be the preferred treatment option and **2)** likelihood of premium pricing over tPA/DNase

Sources: Third party research from MME

IP Summary



Strong IP Position for All Programs and Orphan Drug Designation for Both Clinical Assets

LTI-01

- Issued method of use US patent (7,332,469)
- Orphan Drug Designation in US and EU[1]
- First to file BLA market exclusivity of 12 years

LTI-03

- 4 issued US patents (composition and methods of use)
- 2 pending US patent apps
- 20 pending foreign patent apps
- Orphan Drug Designation in US[2]

FDA | ORPHAN PRODUCTS DEVELOPMENT | EUROPEAN MEDICINES AGENCY | EMA ORPHAN DRUG DESIGNATIONS

1 Orphan Drug Designation in the US and EU for the treatment of empyema.
2 Orphan Drug Designation in the US for the treatment of IPF.

NASDAQ: ALRN

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