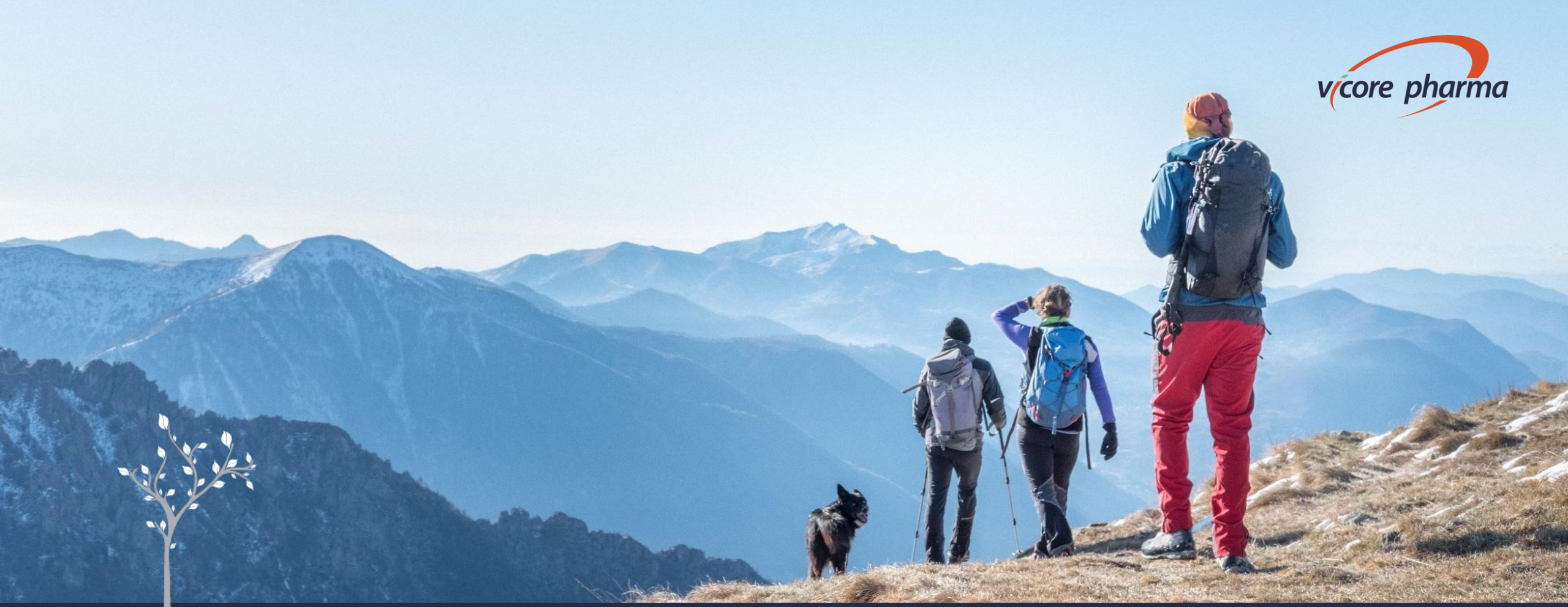

VICORE PHARMA

Unlocking the potential of a new class of drugs – Angiotensin II type 2 receptor agonists (ATRAGs)

November 2024

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Vicore at a glance

Unlocking the potential of a new drug class – ATRAGs

A powerful, upstream mechanism for idiopathic pulmonary fibrosis (IPF)

Unprecedented FVC improvement and excellent safety profile in 36-week Phase 2a IPF trial

Capitalizing on buloxibutid, while developing an ATRAG clinical program

Company overview

Vision

Transform the lives of patients where modulation of the AT2 (angiotensin II type 2) receptor can play a central role in halting and reversing disease pathology

Locations

Stockholm, Sweden, Cambridge, Massachusetts & Copenhagen, Denmark

Financials

Publicly listed (Nasdaq Stockholm: VICO) with 180 million USD market cap (October 31, 2024). Pro forma financial position, including gross proceeds from the share issues, as of September 30, 2024, amounts to approximately 123 million USD.

Key Shareholders

HealthCap, HBM Healthcare Investments, Sanofi, Capital Group, Invus, and Suvretta

Advancing a diversified pipeline

Molecular Therapies

Compound	Indication	Preclinical	Phase 1	Phase 2	Phase 3	Comments	Rights
Buloxibutid (C21)	**IPF**					Phase 2b ongoing (NCT06588686)	Global ex-Japan rights Japan: NIPPON SHINYAKU CO., LTD.
New ATRAGs	**Multiple Indications**					Preclinical studies	Fully-owned

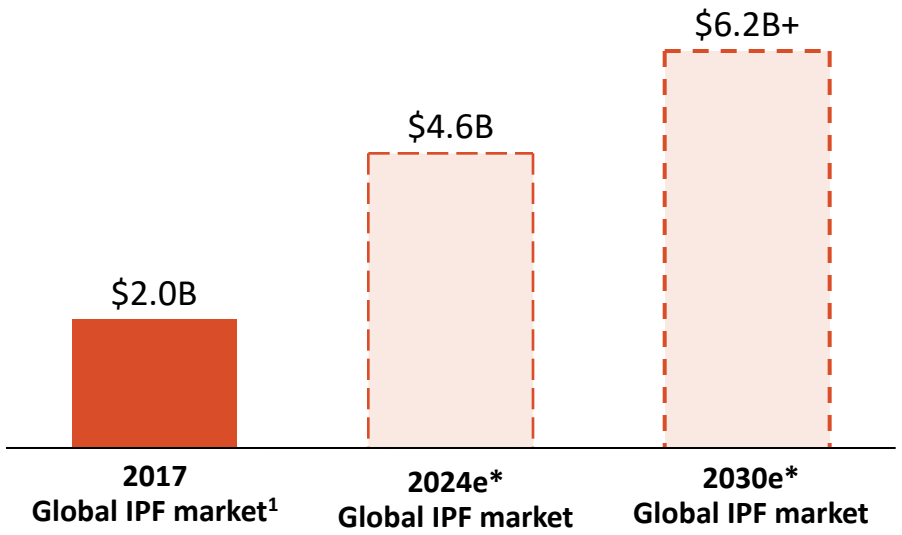
Digital Therapies

Compound	Indication	Preclinical	Phase 1	Phase 2	Phase 3	Comments	Rights
Almee™ DTx	**PF* Anxiety**					Pivotal study (NCT05330312) completed	Fully-owned

*PF = Pulmonary Fibrosis

IPF: A large and growing commercial opportunity

Large commercial market despite SoC shortcomings

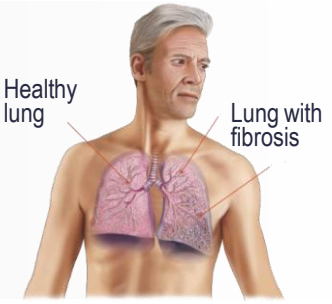


- Growth driven by increased diagnosis and treatment rate
- Limitations of current SoC – moderate deceleration of disease progression, but with significant side effects and no improvement in quality of life[1,2]
- Strong clinician and regulator desire for tolerable and combinable therapies

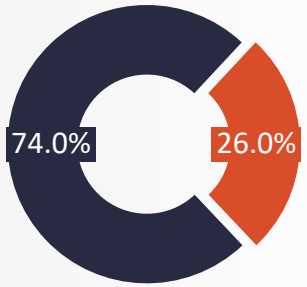
Majority of the market is not adequately addressed

Population in US and Europe

~250,000



Only ~26% of US patients initiate treatment[3]



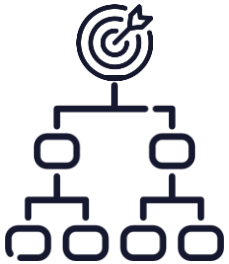
High discontinuation rate and short time on therapy[3]

Average duration of treatment: **10** months

*Estimated sales

(1) Evaluate Pharma, Mordor Intelligence. Note: Combined sales of marketed drugs Ofev and Esbriet where Ofev sales since 2019 includes the indication SSc-ILD; (2) Pulmonary Fibrosis Foundation, Resp Res. 21, Article no: 48 (2020); (3) Dempsey et al. Ann Am Thorac Soc 2021

Buloxibutid is a first-in-class AT2 receptor agonist with the potential to transform the IPF landscape



Upstream MoA with strong preclinical data

- AT2 receptor expressed on alveolar progenitor cell (AEC2)
- Upstream MoA drives antifibrosis, surfactant production and collagenase expression

Exceptional clinical data in the Phase 2a AIR trial

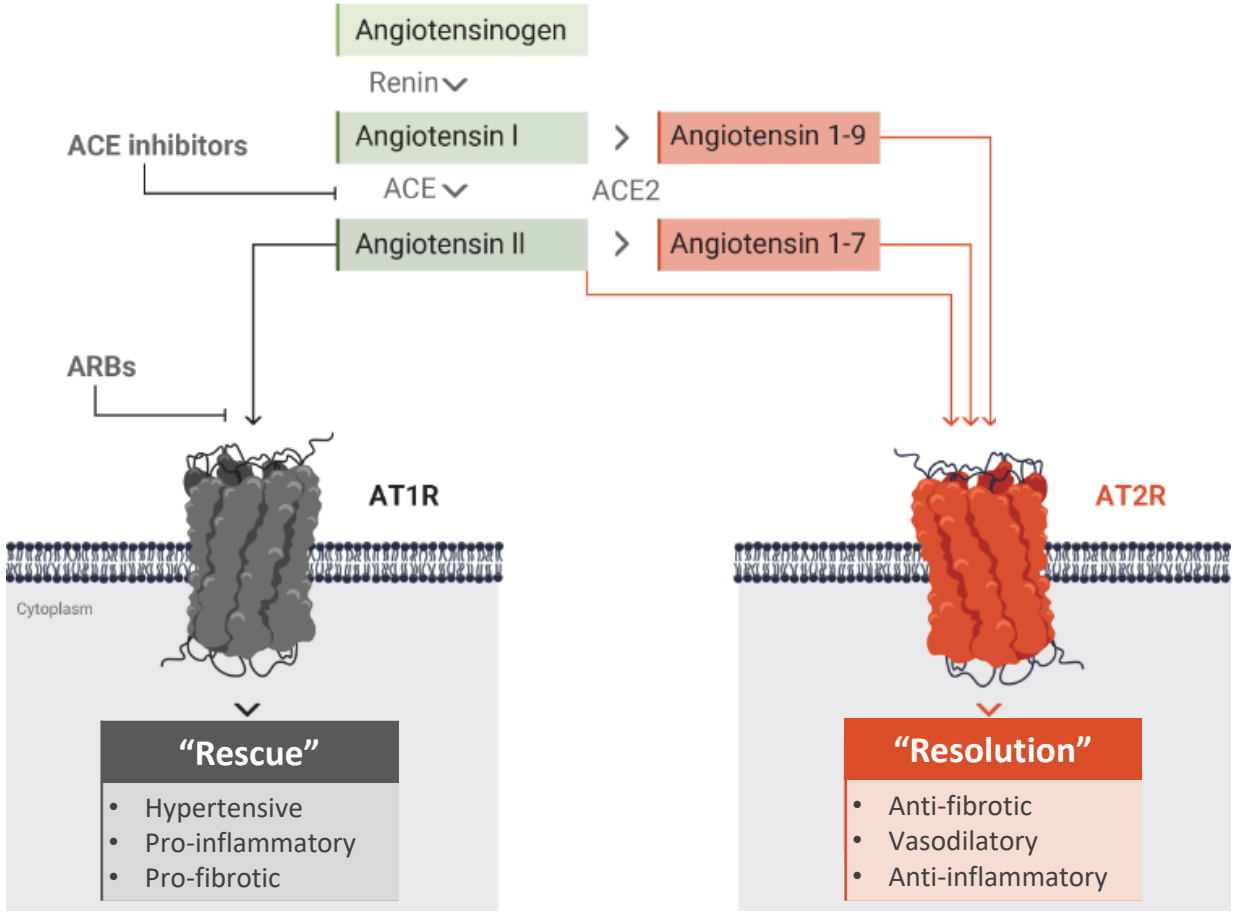
- Mean FVC change from baseline of +216 ml at 36 weeks
- All subgroups above baseline
- Excellent gastrointestinal tolerability and no treatment-related SAEs
- Biomarker data highly supportive of suggested MoA

Phase 2b ASPIRE: confirming the clinical activity in a randomized, placebo-controlled trial

- 52-week treatment
- N=270 (90 per arm)
- IPF patients on stable nintedanib/SoC or not on SoC
- Global footprint

ASPIRE

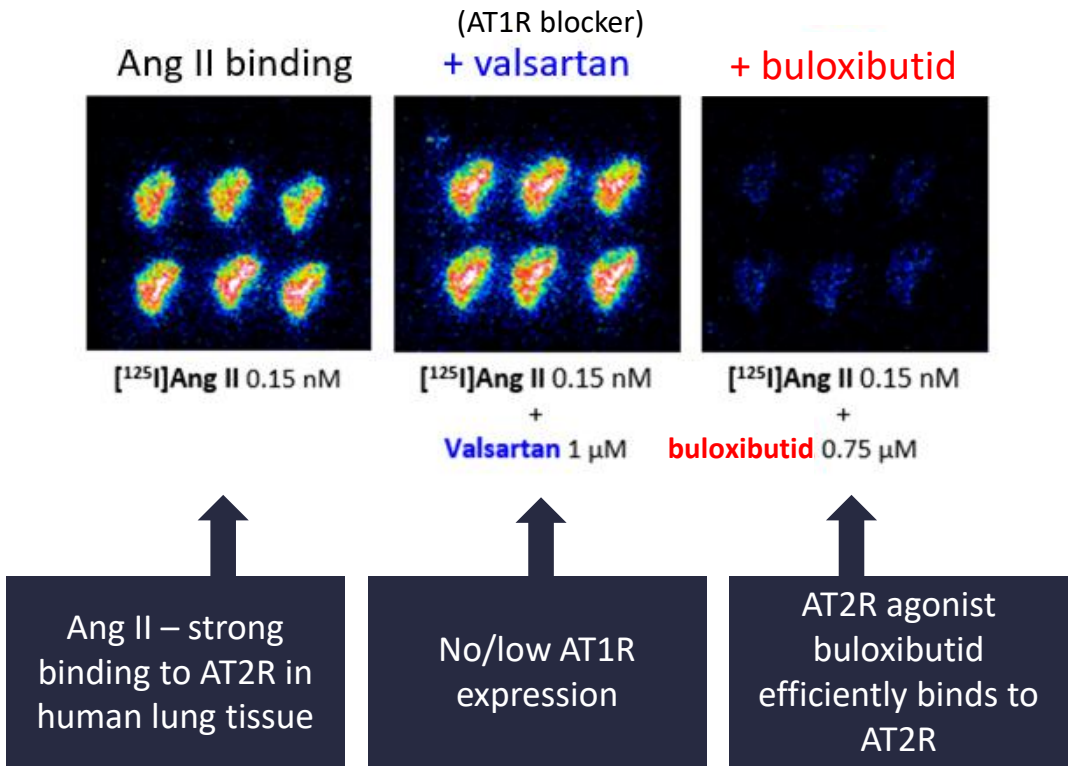
AT2R agonism is an upstream intervention driving tissue repair



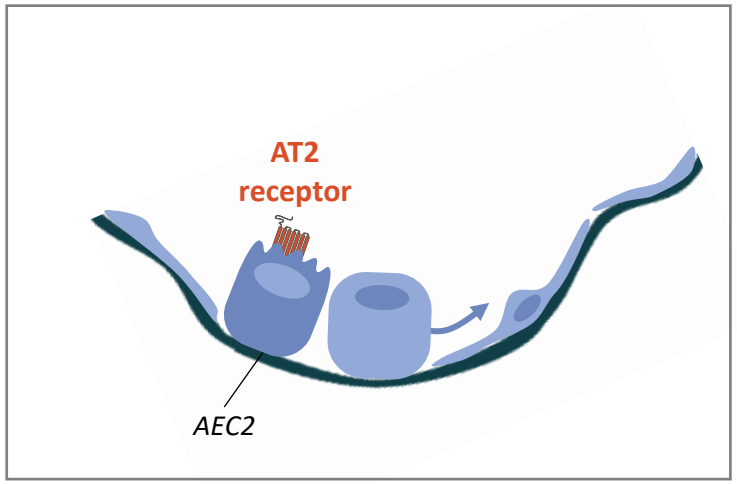
- The angiotensin II pathway is highly conserved with similar components across species
- Angiotensin II activates AT1R and AT2R
- AT1R is widely expressed, while AT2R is consistently expressed in the lung, primarily on alveolar epithelial type 2 cells (AEC2), and is upregulated at sites of disease/tissue injury
- AT1R effects include increase in blood pressure, a key reason for ACEi and ARB development
- AT2R activates tissue protective mechanisms including anti-fibrotic effects

AT2R is highly expressed in human lungs and specifically on precursor AEC2s

AT2R—but not AT1R—is expressed in the human lung[1]



AT2R is selectively expressed on AEC2s[2]



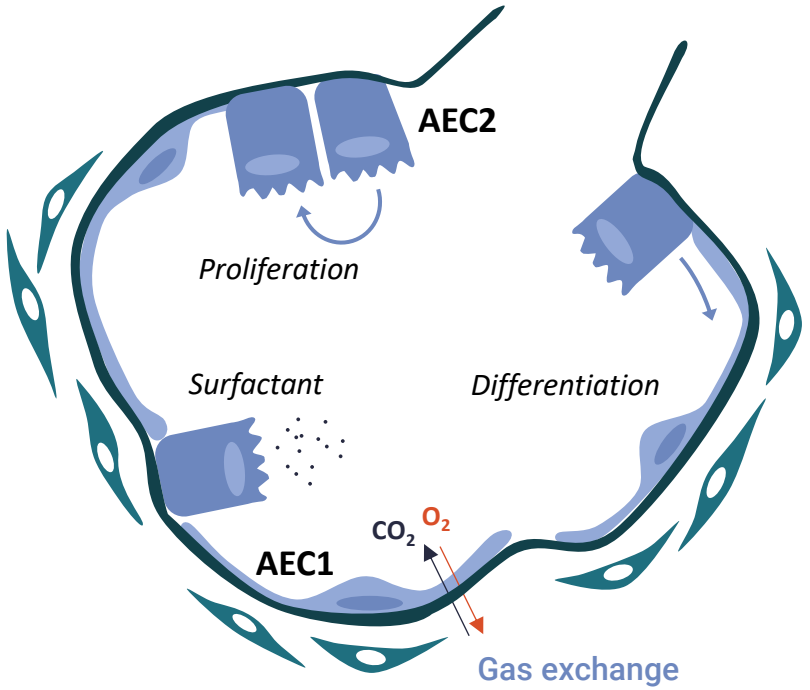
Single cell analysis shows AT2R expression selectively on AEC2 in the lung

(1) IPF atlas; (2) Vicore data on file

Alveolar epithelial cells are critical for healthy lung function

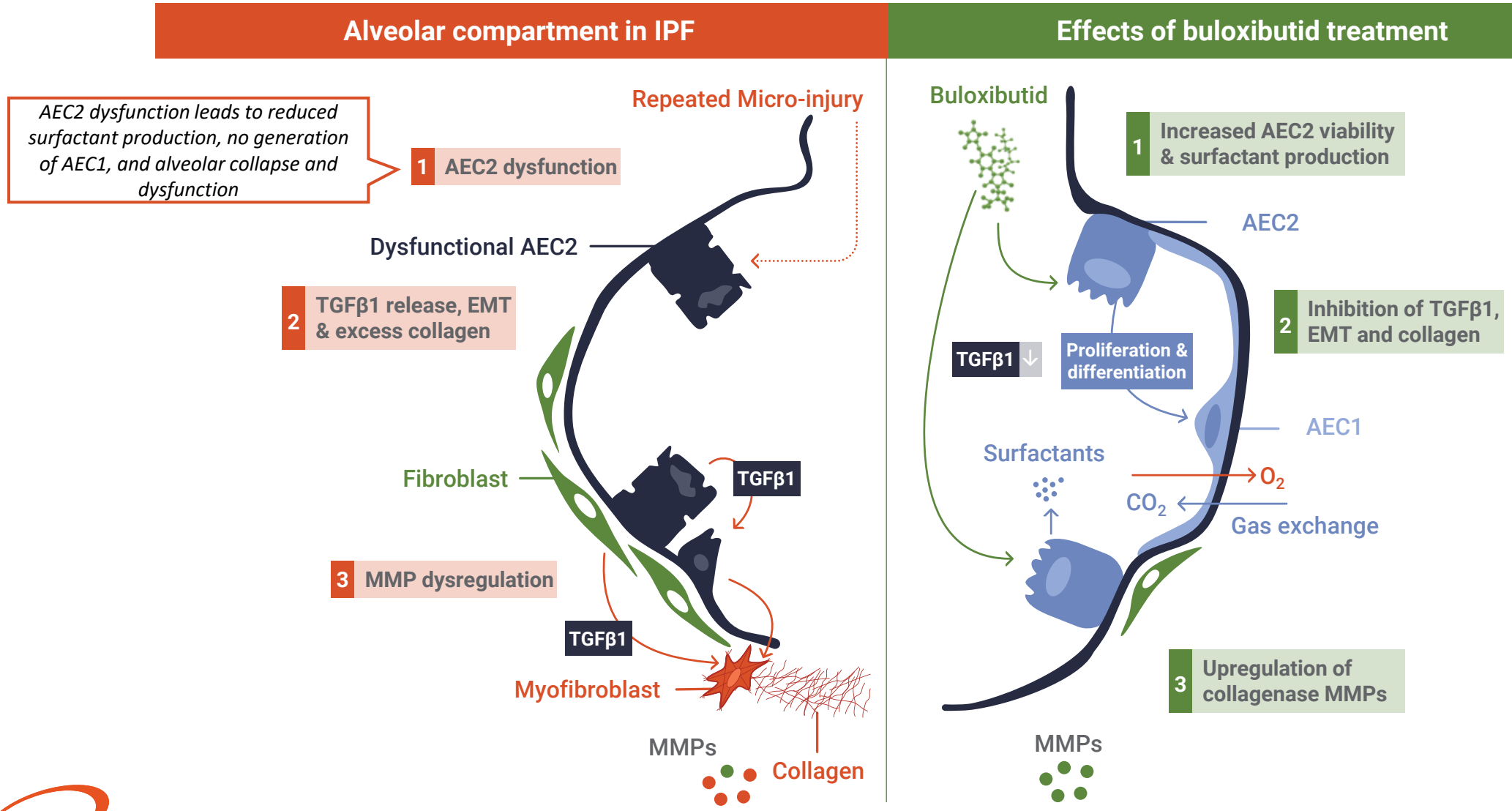
- The alveolar epithelium is constantly exposed to damaging irritants in inhaled air
- AEC1 is the predominant alveolar cell type and is responsible for gas exchange
- AEC2 is a progenitor cell that is critical for alveolar integrity and function:
  - Proliferates to form new AEC2
  - Differentiates to AEC1 that need to be replaced
  - Produces surfactant to maintain alveolar integrity
- AT2R selectively expressed on AEC2

Healthy alveolus



AEC – Alveolar Epithelial Cell

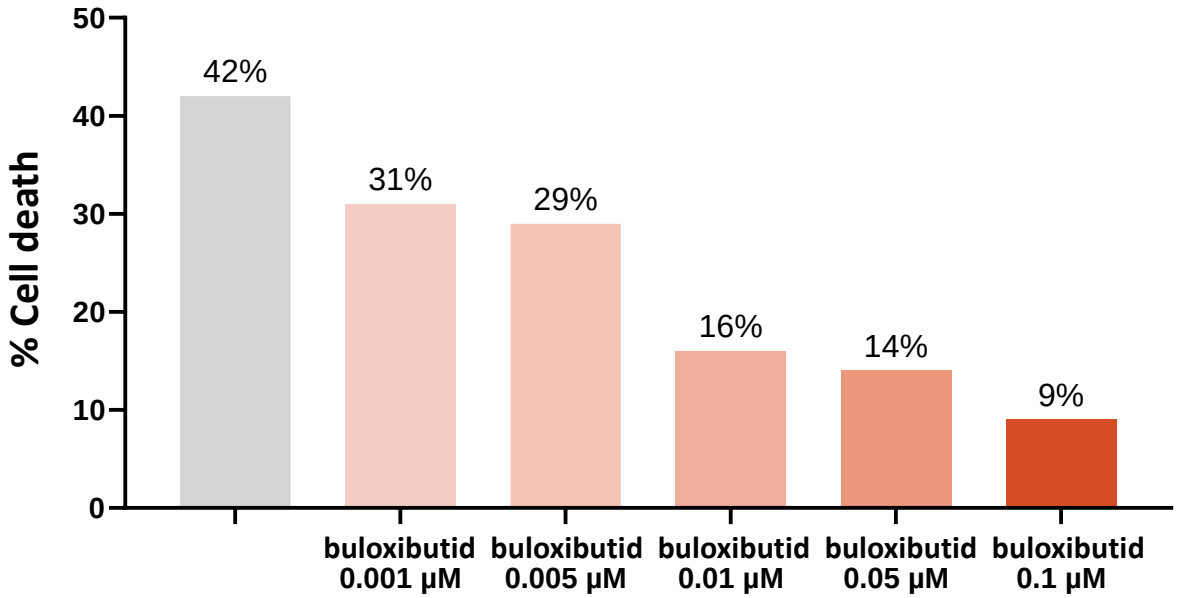
Buloxibutid is an oral, selective AT2R agonist that drives tissue repair via AEC2 precursor epithelial cells



EMT = Epithelial–mesenchymal transition; MMPs = Matrix metalloproteinases

Buloxibutid protects AEC2s and drives increased surfactant production

Buloxibutid protects AEC2 cells against apoptosis[1]



- Cultured A549 cells (human AEC2 cell line)
- Bleomycin (10µg/ml) induced apoptosis

Surfactant protein expression increased by buloxibutid in *ex vivo* human IPF precision cut lung slices[2]

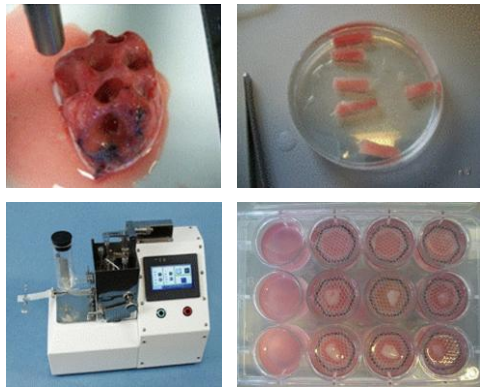
- Human precision cut IPF lung slices ± 1 µM buloxibutid
- One patient, 5 pooled lung slices

Treatment with buloxibutid protects AEC2s, driving increased surfactant production to address alveolar collapse

(1) Adapted from Nalbandyan et al. FASEB 2018; (2) Vicore data on file

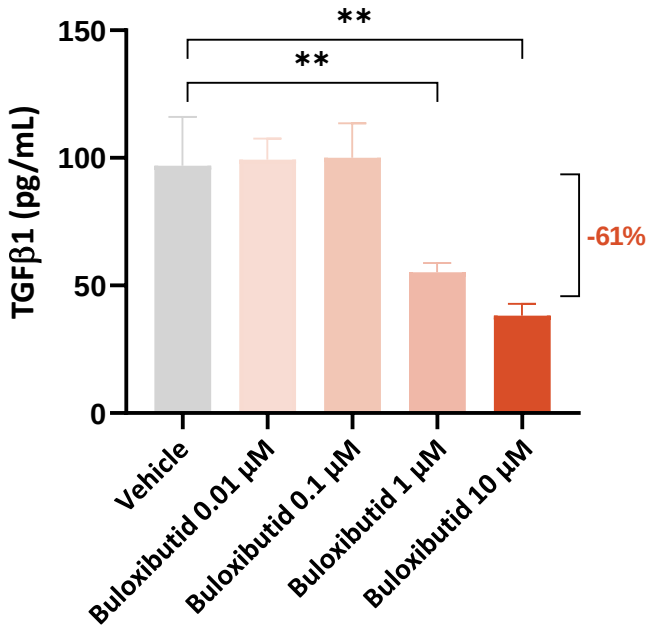
Buloxibutid reduces TGFβ1 and collagen in human IPF lung slices

Human precision cut lung slices (PCLuS)

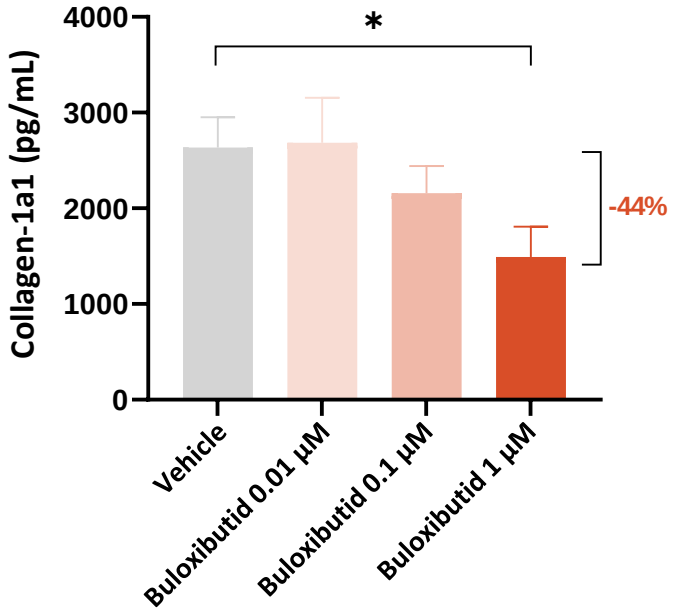


- Lung tissue collected from IPF patients undergoing transplant
- Intrinsic fibrosis, no stimuli added

TGFβ1 protein levels in PCLuS



Collagen protein levels in PCLuS

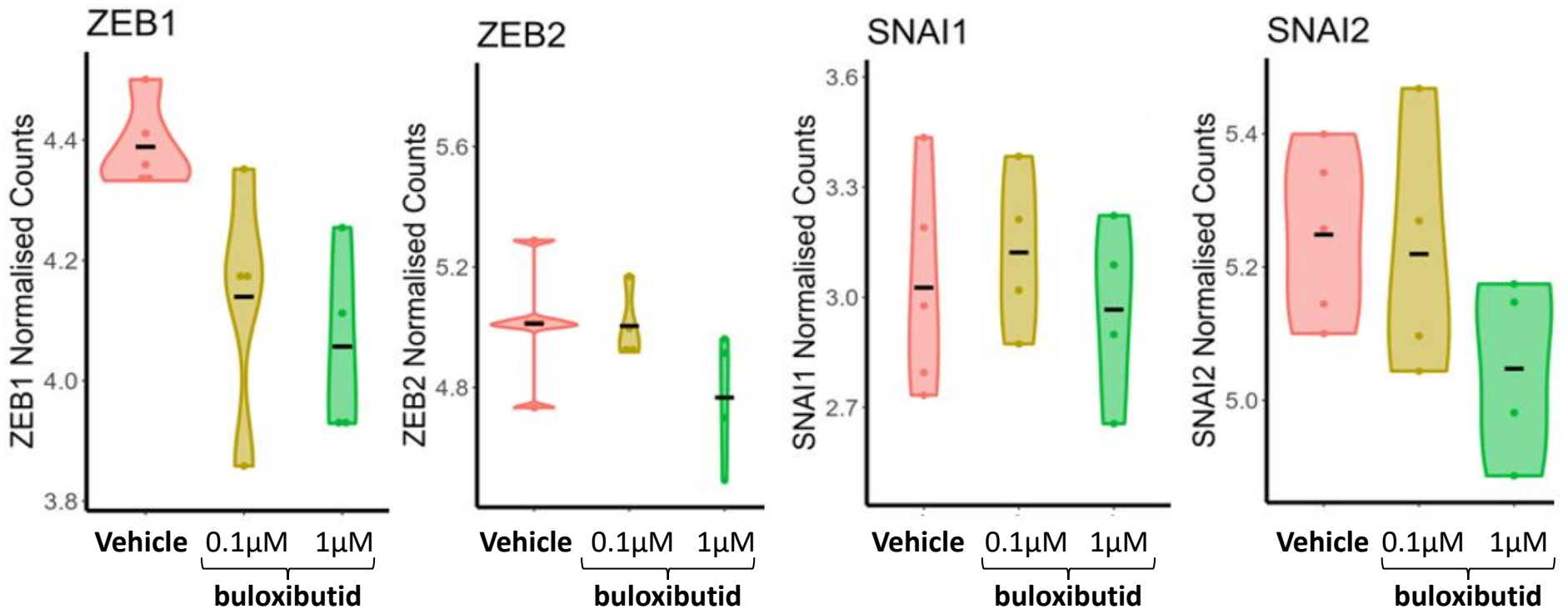


Dose-dependent reduction of TGFβ1 and Collagen-1a1 protein

Source: Vicore data on file
*p<0.05, **p<0.01, ***p<0.001; t-test versus vehicle

Data represent averages +/- SEM of Plus 5 separate tissue slices at each concentration, sampled after 144h exposure to buloxibutid or vehicle

Buloxibutid downregulates expression of EMT transcription factors in AEC2



- Primary AEC2 cultures established from normal surgically resected human lung
- Buloxibutid treatment under baseline conditions with no stimuli added

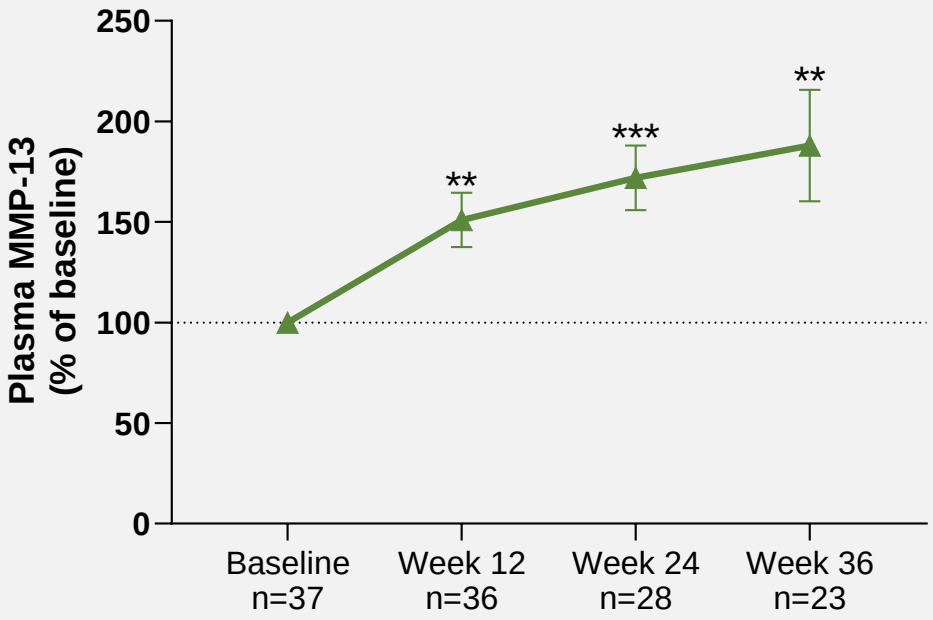
Source: Conforti et al. 2023, manuscript (Vicore collaboration with U Southampton)

MMP-13 demonstrates antifibrotic activity and is crucial for lung repair in IPF

Collagenase MMP dysregulation contributes to IPF pathogenesis

- MMP-13 is an enzyme able to **cleave fibrillar collagens** and plays a significant role in the **degradation of the ECM**
- In mouse models, MMP-13 deficiency has been shown to[1,2]:
  1. Decrease collagenolytic activity
  2. Promote lung fibrosis
  3. Attenuate fibrosis resolution

Buloxibutid increased plasma MMP-13 in the Phase 2a AIR trial



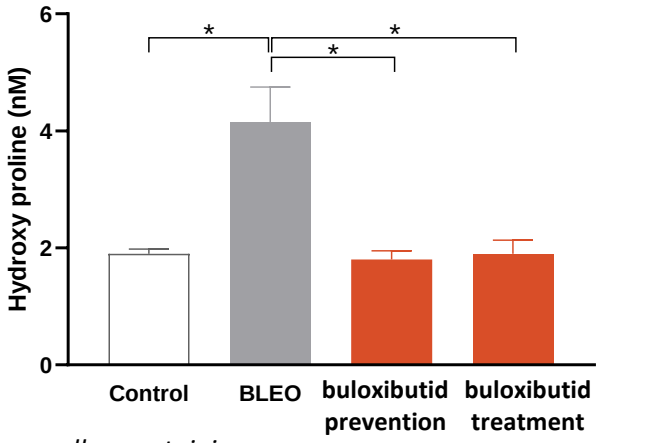
Buloxibutid significantly increased plasma levels of the fibrolytic collagenase MMP-13, indicating that buloxibutid has the potential to degrade fibrosis

ECM = Extracellular Matrix; MMP = Matrix Metalloproteases

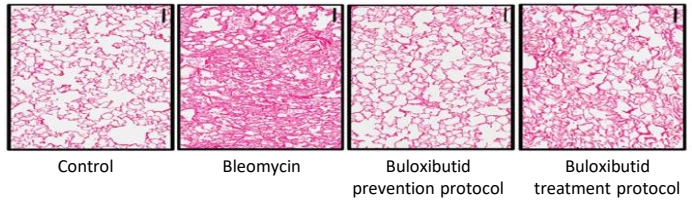
(1) Cabrera et al., 2019; (2) Nkyimbeng et al., 2013

Strong preclinical *in vivo* evidence for buloxibutid in pulmonary fibrosis

Bleomycin



Lung collagen staining

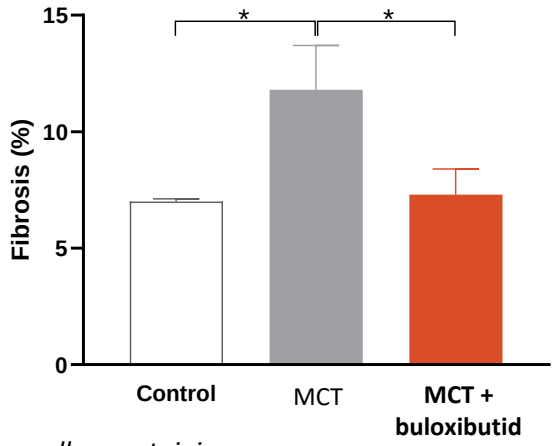


- Buloxibutid 0.03 mg/kg/day i.p. once daily, 14 days (Treatment). N=5 control and 7-8 in treatment groups

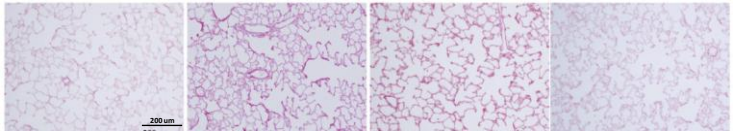
Adapted from (1)

➢ **Normalized collagen synthesis and attenuation of disrupted lung architecture**

Monocrotaline



Lung collagen staining

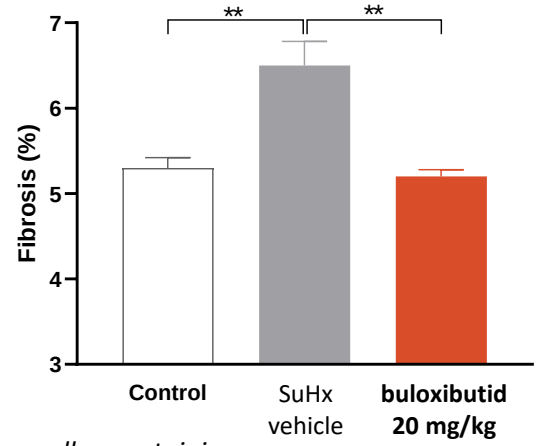


- Buloxibutid 0.03 mg/kg/day i.p. once daily for 2 weeks
- N=14 per treatment group

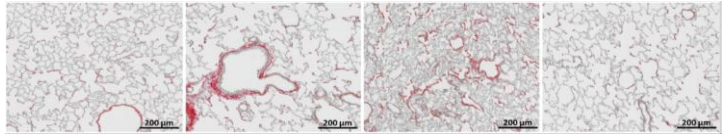
Adapted from (2)

➢ **Reversal of fibrosis**

Sugen-Hypoxia



Lung collagen staining



- Buloxibutid p.o. for 34 days, initiated 21 d. after SuHx-period. N=10-11 per treatment group

Adapted from (3)

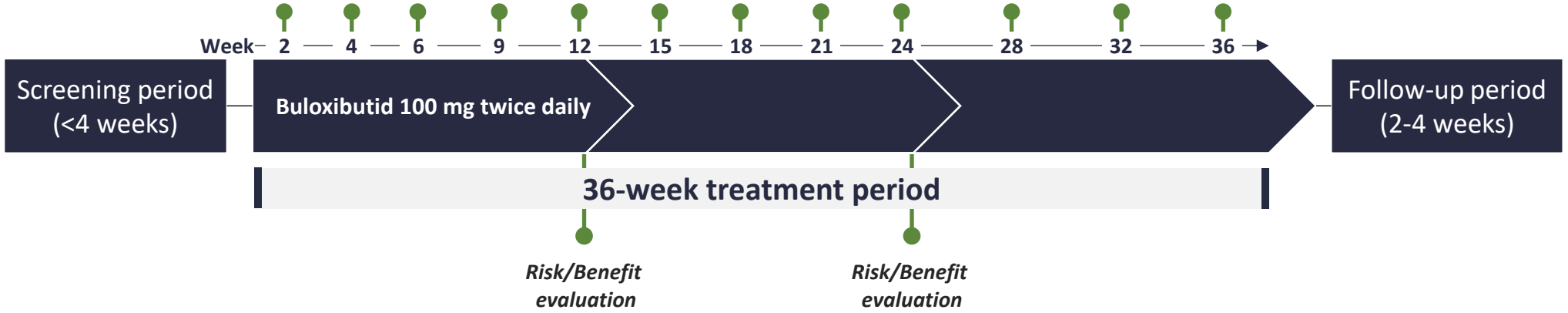
➢ **Reversal of fibrosis**

(1) Rathinasabapathy et al. Front Physiol. 2018; (2) Bruce et al. Br J Pharmacol. 2015; (3) Tornling et al. Int. J. Mol. Sci. 2023

AIR: An open-label Phase 2a trial of oral buloxibutid 100 mg BID for up to 36 weeks in treatment-naïve IPF patients

Patient population

Treatment-naïve IPF patients with centrally HRCT-confirmed diagnosis



Primary endpoint
Safety and tolerability

Secondary endpoint
Change in forced vital capacity (FVC) from baseline

Exploratory endpoints
Effect on selected biomarkers

AIR baseline patient characteristics are in line with other IPF trials

Key Characteristics

		AIR (N=52)	INPULSIS 1&2 (N=1,061)[1]	
Age (years) - Mean (SD)		**67 (9)**	67 (8)	In line with other trials.
Gender	Males	**77%**	80%	
	Females	**23%**	20%	
Ethnicity	White	**27%**	57%	Enrolled study population has disease progression comparable to global IPF study populations.
	Asian	**73%**	30%	
BMI (kg/m^2) – Mean (SD)		**24.6 (4.1)**	28 (4.6)	
FVC % predicted - Mean (SD)		**75.5 (14)**	79.7 (17)	In line with other trials.
% SoC	Pirfenidone	**0%**	0%	As with the INPULSIS trials, AIR patients were treatment-naïve.
	Nintedanib	**0%**	0%	

(1) INPULSIS 1&2 were the Phase 3 studies that led to the approval of nintedanib; Richeldi et al. N Engl J Med 2014; 370:2071-82

Treatment emergent adverse events: buloxibutid shows better tolerability than SoC

	INPULSIS 1 *52-week treatment*[1]		AIR *36-week treatment*
	Nintedanib	Placebo	Buloxibutid
	n=309	n=204	n=52
Any AE	96%	89%	71%
Common AEs (Non-exhaustive)			
Diarrhea	62%	19%	6%
Nausea	23%	6%	4%
Acute exacerbation of IPF	10%	10%	6%
Cough	15%	13%	8%
Vomiting	13%	2%	2%
COVID-19	n/a	n/a	6%
Hair loss[2]	n/a	n/a	19%
Fatal AE	4%	5%	4%
Severe AE	26%	18%	6%
Serious AE	31%	27%	10%

Good GI tolerability

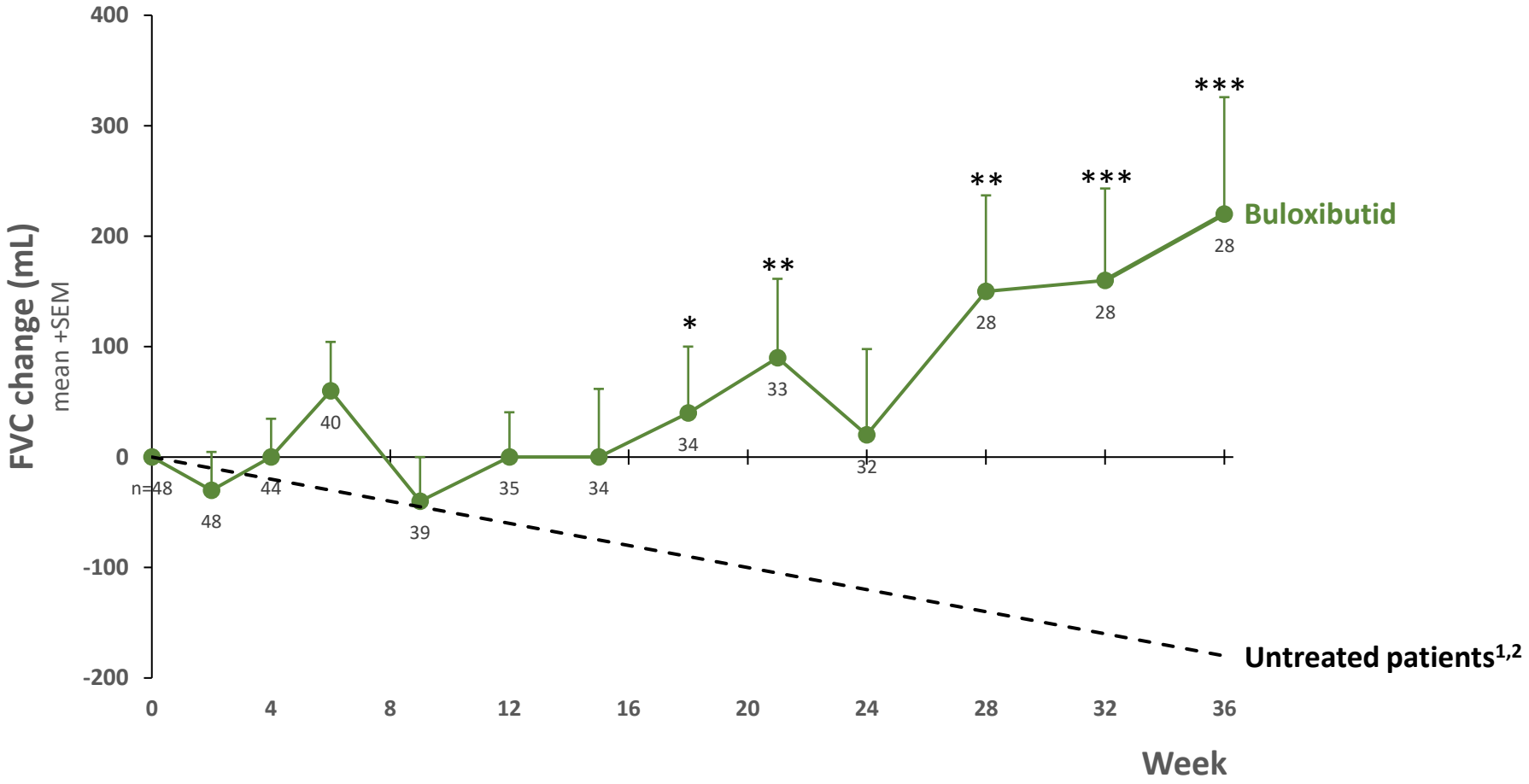
Low rate of exacerbations and cough worsening

No serious, severe, or fatal AEs related to buloxibutid

Buloxibutid has a favorable tolerability profile allowing it to be combined with other therapies for IPF

(1) N Engl J Med 2014;370:2071-82; (2) Hair loss was mild to moderate and reversible. One patient discontinued treatment due to hair loss.
Note: Rash, gastroesophageal reflux disease, elevated creatinine, and pyrexia were also reported at 8% in AIR trial.

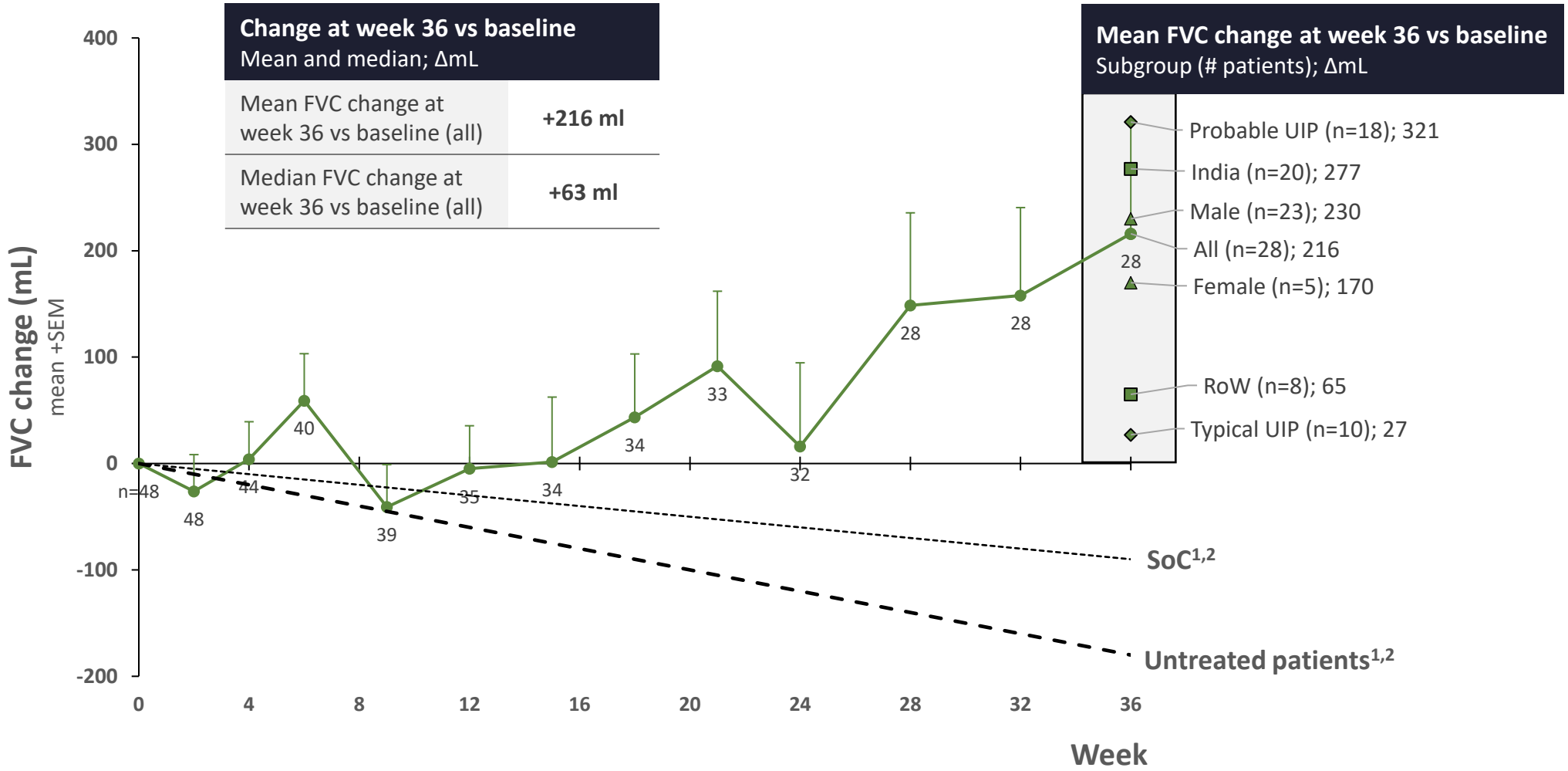
Buloxibutid stabilizes and improves lung function over the 36-week AIR study



Note: n=48 patients with 2-week FVC data. Observed values, no imputation.
*p<0.05, **p<0.01, ***p<0.001; t-test versus expected untreated decline corresponding to -120 mL/24 weeks

[1]*Noble et al. Eur Respir J. 2016 Jan; 47(1): 243–253*
[2]*Richeldi et al. Engl J Med 2014; 370:2071-2082*

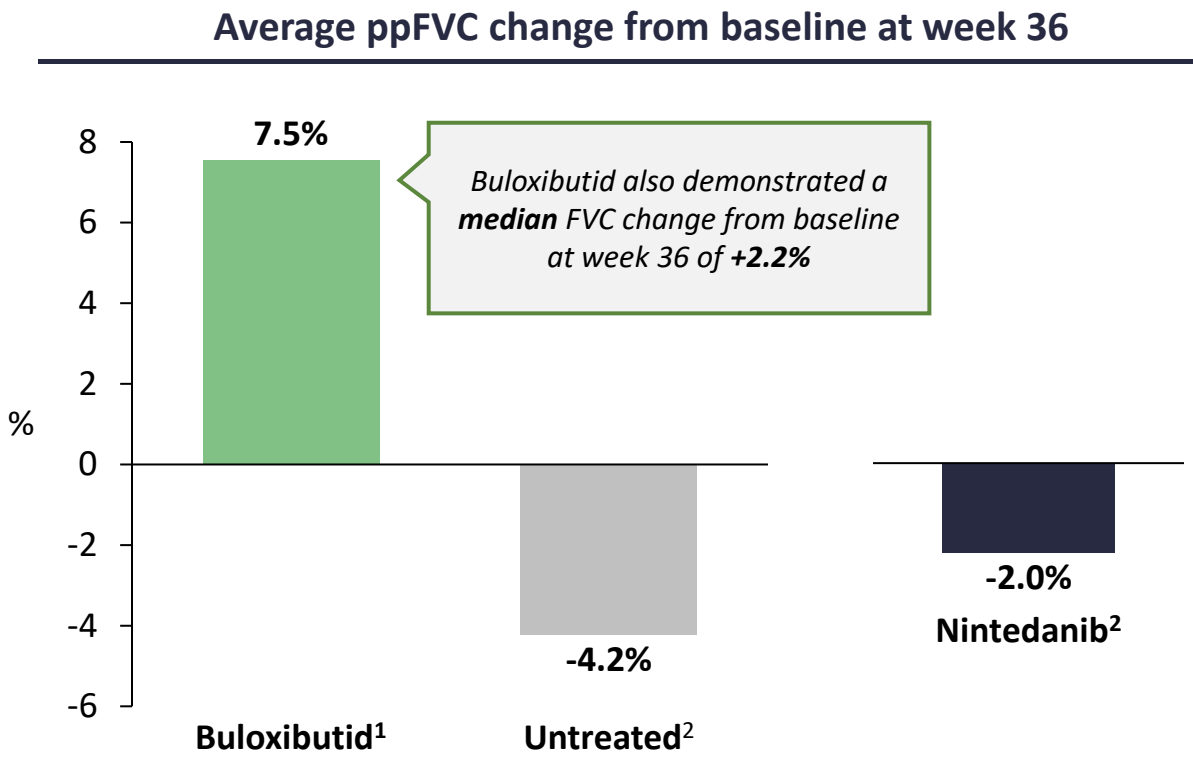
All subgroups show FVC stabilization and improvement over baseline at 36 weeks



Note: Probable UIP and Typical UIP according to HRCT categorization at baseline.

[1]Noble et al. Eur Respir J. 2016 Jan; 47(1): 243–253
[2]Richeldi et al. Engl J Med 2014; 370:2071-2082

Buloxibutid drives a significant increase in ppFVC, consistent with its impact on absolute FVC

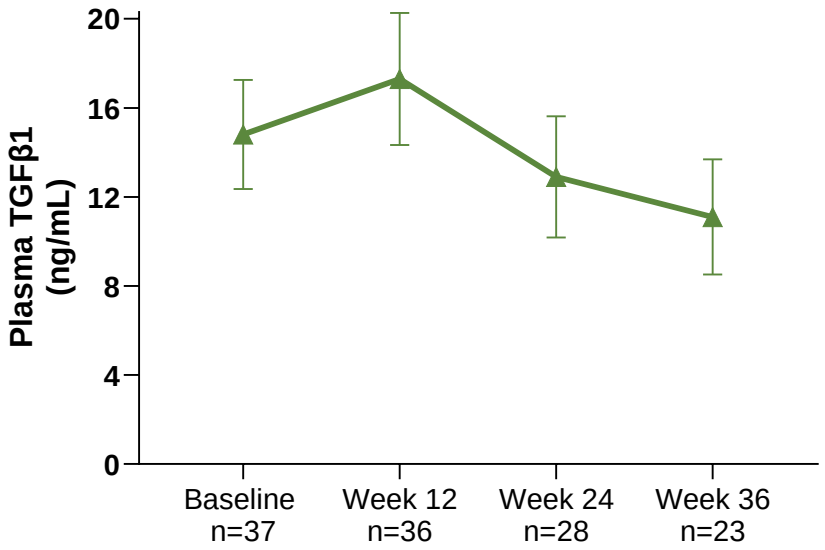


Average increase of 7.5% precent predicted FVC at 36 weeks from baseline

(1) Average ppFVC at 36 weeks (n=28); (2) 36 week estimated ppFVC for nintedanib and placebo, INPULSIS trial (Richeldi et al. N Engl J Med 2014; 370:2071-82)

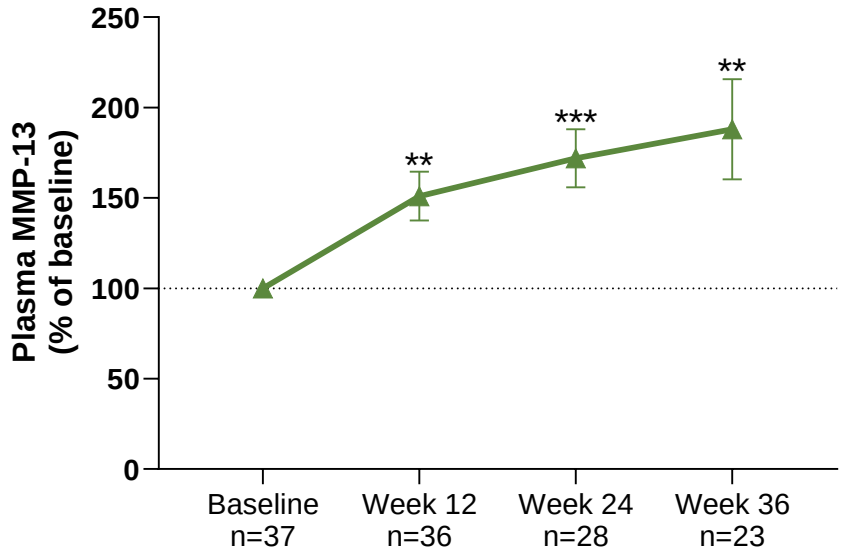
Buloxibutid increases collagenase MMP-13 drives a trend of decreased TGFβ1

Plasma TGFβ1



TGFβ1 is a key fibrotic driver in IPF; reduced TGFβ1 is consistent with buloxibutid's mechanism of action and translational data

Plasma MMP-13



MMP-13 is an antifibrotic collagenase that plays a key role in fibrotic resolution

Note: data shown as mean ±SEM
** p<0.01, ***p<0.001 paired t-test vs baseline

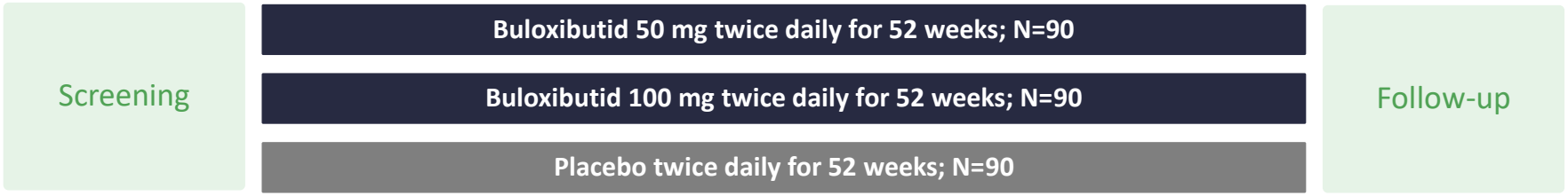
Phase 2b ASPIRE trial design

ASPIRE

Study Characteristics

- A randomized, double-blind, placebo-controlled, parallel-group, multicenter, dose-finding trial
- IPF patients on stable nintedanib/SoC or not on SoC (no access, refused, intolerant or failed)
- 52-week treatment duration; N=270 (90 per arm)
- Assessment of efficacy, safety, and pharmacokinetics at baseline as well as weeks 4, 12, 24, 36, and 52. Remote visits (by phone or video) to assess safety and compliance at weeks 8, 16, 20, 28, 32, 40, 44 and 48
- Primary endpoint is change from baseline in FVC at 52 weeks
- Key secondary efficacy endpoint - proportion of participants with disease progression at 52 weeks

Study Design



Vicore's partnership with Nippon Shinyaku for buloxibutid in Japan

NIPPON SHINYAKU CO., LTD.

Partnership Overview

Vicore Pharma and Nippon Shinyaku have entered an exclusive license agreement to **develop and commercialize the drug candidate buloxibutid in Japan.**

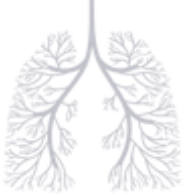
Financial Terms

Vicore has received an **upfront payment of USD 10 million** and is eligible for up to **USD 275 million in milestones**, plus tiered royalties on net sales in Japan up to the low 20s. In addition, Nippon Shinyaku will cover a portion of global non-clinical, CMC, and late-stage clinical development costs.

Strategic Benefits

The partnership leverages Nippon Shinyaku's **local expertise to address IPF**, a condition with limited treatment options in Japan, enhancing Vicore's global IPF strategy. Nippon Shinyaku is a **leader in the development of therapies for rare respiratory diseases** in Japan, including the discovery and development of Uptravi for PAH.

Almee™ is a digital therapy for anxiety in pulmonary fibrosis with the potential to increase commercial sales of molecular therapies



almee

Product description	▪ Almee™ is a digital companion to personalize pulmonary fibrosis treatment.
Clinically validated MoA	▪ COMPANION study demonstrated clinical validation. ▪ Behavior modifying MoA through digital Cognitive Behavioral Therapy (CBT). ▪ Significant reduction of anxiety symptoms (GAD-7) and improvement of Quality of Life (KBILD).
Opportunity	▪ Almee can be positioned to maximize the commercial value of a molecular asset by increasing the patients Quality of Life and optimizing therapy management. ▪ Almee's clinically validated MoA has the potential to increase treatment adherence, time-on-treatment, and treatment initiation for molecular assets.

Vicore is looking to partner Almee with pharma companies that have approved or late-stage IPF assets in development, enabling them to maximize the commercial opportunity for their molecular asset.

Almee™ provides multiple benefits for molecular assets

almee

Treats the psychological impact of living with PF

Designed to improve QoL for patients which current SoCs don't address

True patient engagement – designed to create trust and empowerment

Increased adherence, initiation, and time-on-treatment

Custom build for specific uptake issues.

Label & IP extension

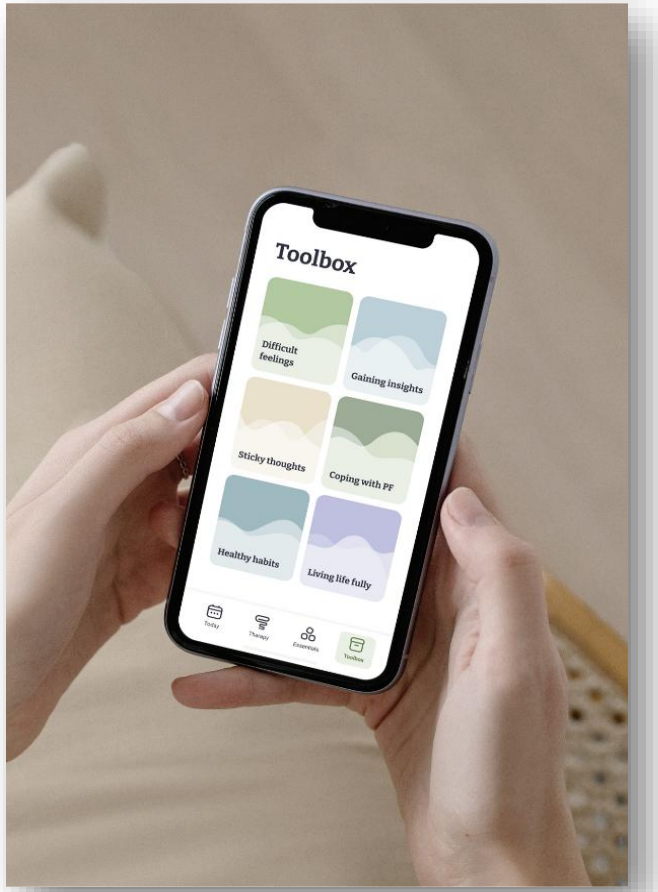
Expand the label of the molecular therapy through DTx/drug combinations. Opportunity for new IP.

Data access

Generate unique real-time data to optimize commercialization activities.

Differentiation

Differentiation versus emerging competition by leveraging a combined molecular and digital therapy approach.



Vicore has a platform of proprietary ATRAGs

Buloxibutid – a first-in-class drug for rare lung diseases

- Orphan drug status in IPF granted – Market exclusivity for 7 years in the US and 10 years in the EU.
- Vicore has formulation and method-of-use IP granted in the US and EU covering buloxibutid, with expiry in 2042 before considering any PTE or SPC*.

Follow-on compounds provide life-cycle-management optionality in IPF and complementary indications, as well as opportunities in a range of other diseases

- 7 novel proprietary classes developed.
- Optimized to drive differentiated biology and therapeutic activity in a range of potential diseases where the angiotensin II pathway can play a therapeutic role.
- Enable Vicore to significantly extend its AT2R franchise in respiratory diseases beyond buloxibutid, as well provide optionality to pursue a range of other diseases, either fully proprietary or in partnerships.

*PTE = Patent Term Extension; SPC = Supplementary Protection Certificate

Strong leadership team with extensive industry experience

AHMED MOUSA
CHIEF EXECUTIVE OFFICER

Experienced biotech executive with a background in molecular biology, law, and business development.

pieris · COVINGTON

JOHAN RAUD, MD, PhD
CHIEF SCIENTIFIC OFFICER

Ex AstraZeneca: Director of inflammation research.
25 years of experience in drug development.

AstraZeneca

PROF. BERTIL LINDMARK, MD, PhD
CHIEF MEDICAL OFFICER

Extensive industry experience in respiratory and inflammatory diseases. Ex-AstraZeneca: Led the development of global brands like Pulmicort and Symbicort.

almirall feel the science · AstraZeneca

NINA CARLÈN
CHIEF ADMINISTRATIVE OFFICER

More than 20 years of administration and communications experience. Responsible for HR and company administration.

BERGHS SCHOOL OF COMMUNICATION

JIMMIE HOFMAN
VP BUSINESS DEVELOPMENT

Business Development executive with extensive deal-making experience.

pieris

vicore pharma

HANS JEPPSSON, PhD
CHIEF FINANCIAL OFFICER

Cross-disciplinary background in finance and medicine.
Ex Danske Bank: Equity analyst.

Danske Bank · AstraZeneca

JOHANNA GRÄNS, PhD
PROGRAM DIRECTOR, EARLY DEVELOPMENT

Extensive experience in preclinical R&D. Project management and regulatory affairs. Research experience in drug metabolism.

UNIVERSITY OF GOTHENBURG

JESSICA SHULL, PhD
DIRECTOR OF DIGITAL HEALTH

More than 20 years of experience in the development and adoption of digital healthcare technologies.

World Health Organization · DIGITAL THERAPEUTICS ALLIANCE

HELEN BARKER
VP AND HEAD OF CMC

Pharmaceutical scientist and business leader, with over 25 years of experience delivering the technical and strategic development of novel compounds, devices and companies.

Pfizer · ZIARCO · UNIVERSITY OF OXFORD

MEGAN RICHARDS
VP IR, COMMUNICATIONS, PORTFOLIO STRATEGY

Extensive experience in commercial and portfolio strategy, corporate strategy, and communications.

ARTISAN · pieris

MIKAEL NYGÅRD, PhD
VP OPERATIONS AND CORPORATE STRATEGY

Experienced healthcare Business Development executive, has led M&A and Corporate Development functions.

Humana · BCG BOSTON CONSULTING GROUP

ÅSA MAGNUSSON
CHIEF ENGAGEMENT & COMMERCIAL OFFICER

More than 20 years of experience as a commercial executive in the pharmaceutical industry with focus on securing market access and launching rare disease medicines.

ALEXION · ACTELION

Board of Directors

HANS SCHIKAN, PharmD – CHAIRMAN
25 years management experience in global pharmaceuticals (e.g. CEO of Prosensa). Extensive board work experience from US Nasdaq-listed biotech firms.

ANN BARBIER, MD, PhD
More than 20 years of experience in drug discovery and development in rare diseases, including rare respiratory diseases.

MICHAEL BUSCHLE, PhD
More than 25 years of experience in basic research as well as biotech and pharma R&D. Extensive board work experience from US Nasdaq-listed biotech firms.

ELISABETH BJÖRK, MD, PhD
Broad drug development experience, currently leading global late-stage development activities in CVRM at AstraZeneca. Extensive board work experience in small and mid-size international life science companies.

JACOB GUNTERBERG
Experienced venture capitalist and life science sector financier.

HEIDI HUNTER
25 years in senior pharmaceutical development and commercialization positions.

YASIR AL-WAKEEL, BM BCH
A seasoned executive board member and strategic advisor with focus on strategic finance and business development in biotech companies.

Vicore is well positioned to unlock the potential of a new class of drugs - in rare lung diseases and beyond

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