VICORE PHARMA

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

May 2026

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Company overview

Vicore 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, USA
Copenhagen, Denmark

Financials

Publicly listed on Nasdaq Stockholm (VICO) and funded well past Phase 2b data

$341m
market cap
As of May 13, 2026

$111m
financial position
As of March 31, 2026

Shareholders

Vicore is backed by leading specialist investors in the US and Europe

Pipeline

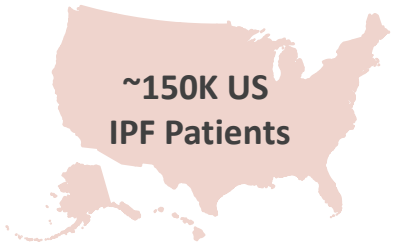
Vicore's lead program, buloxibutid, is a first-in-class oral small molecule AT2 receptor agonist, which has received Orphan Drug and Fast Track designation from FDA and is currently being investigated in a global 52-week Phase 2b trial in IPF, ASPIRE

Compound	Indication	Preclinical	Phase 1	Phase 2	Phase 3	Comments	Rights
Buloxibutid	**IPF**					Phase 2b ongoing (NCT06588686) Enrollment complete; targeting topline readout in mid-2027	Global ex-Japan rights Japan: NIPPON SHINYAKU CO., LTD.
New ATRAGs	**Multiple Indications**					Preclinical studies	Fully-owned

Unlocking the potential of a new class of drugs - Angiotensin II Type 2 Receptor Agonists (ATRAGs)

IPF is a progressive, fatal disease with significant unmet need despite available therapies

Orphan disease with high unmet need



Where only ~1/4 of US patients initiate treatment[1]

And the high discontinuation rate leads to an average time on treatment of only

10 months[1]

The majority of the IPF market is not adequately addressed today

Limitations of available treatment options

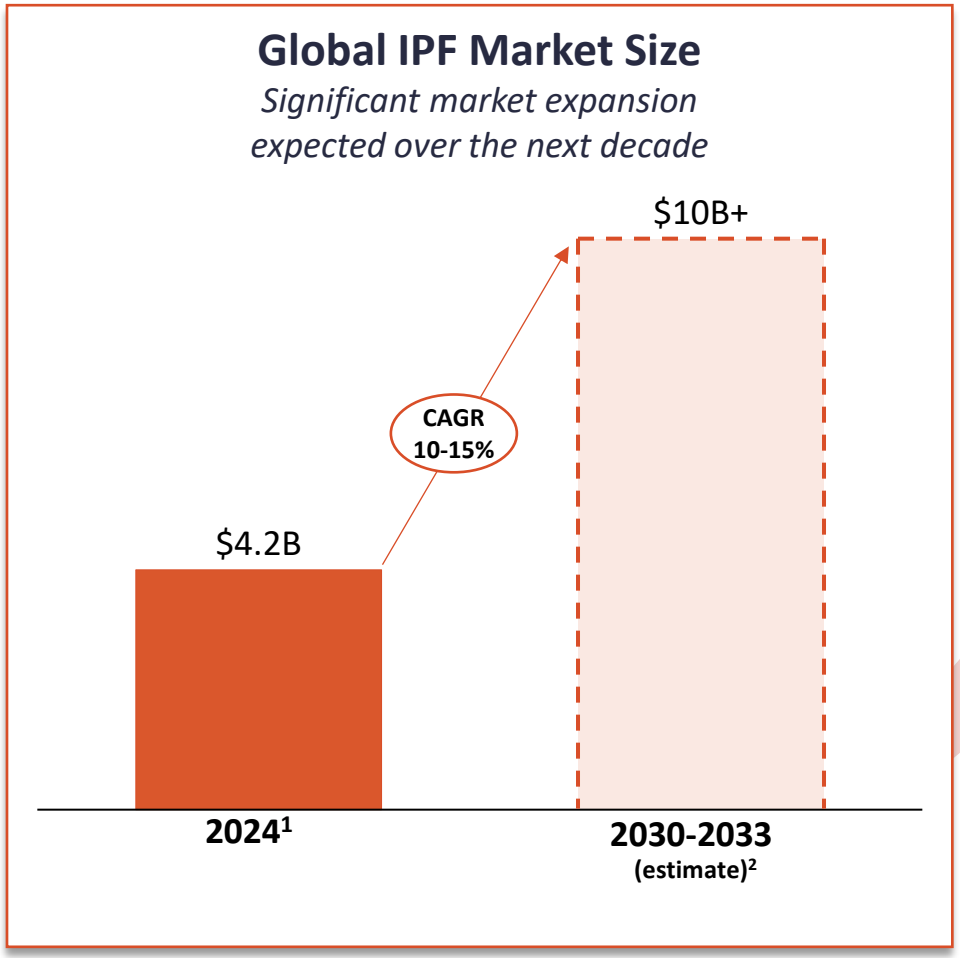
Three FDA-approved therapies:

OFEV® Esbriet® JASCAYD®

- Existing therapies offer only modest slowing of disease progression and have not demonstrated quality of life benefit
- Significant GI side effects limit uptake, often requiring dose reductions and contributing to high discontinuation rates
- Despite available treatments, the 5-year mortality rate is 80%[2]
- There are no approved disease-modifying therapies today

(1) Dempsey et al. Ann Am Thorac Soc 2021; (2) IPF Foundation

IPF is a large and growing market with urgent need for innovation



Drivers of Market Growth Today

- **New Approvals:** Jascayd
- **Disease Awareness:** Rising diagnosis rates and expected broader treatment adoption
- **Epidemiology:** Aging population and increasing disease prevalence

White Space for Market-Shifting Innovation

- **Disease-Modifying Efficacy:** Stabilization or improvement in lung function
- **Well-Tolerated & Combinable Therapies:** Expanding treatment pool, extending time on therapy, and reducing discontinuations

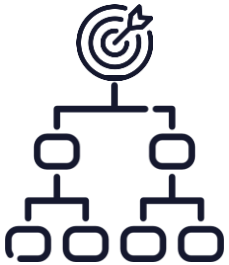
(1) Company reports (note: combined sales of marketed drugs Ofev and Esbriet where Ofev sales include SSc-ILD); (2) Global Data, The Insights Partners

Buloxibutid is positioned to transform the IPF landscape

Recently approved and late-stage IPF therapies have demonstrated modest reduction of lung function decline, with many associated with tolerability or administration challenges. Following these programs, buloxibutid is the most advanced IPF therapy in global development.

	Vicore pharma Buloxibutid	Boehringer Ingelheim Jascayd (nerandomilast)	United Therapeutics Tyvaso (treprostinil)	Bristol Myers Squibb Admilparant
MOA	**AT2 receptor agonist** Novel endogenous upstream and multi-modal mechanism	PDE4B antagonist	prostacyclin analog	LPA1 antagonist
Dosing & Administration	**Oral BID** Convenient oral dosing	Oral BID	Nebulized formulation, 48 (12x4) breaths per day	Oral BID
Tolerability	**Favorable and combinable tolerability profile;** mild-to-moderate hair loss	GI-tolerability issues, further exacerbated on top of SoC	Cough, throat irritation, headache, nausea and flushing	Favorable overall profile; transient reduction in blood pressure
Efficacy	**Unprecedented FVC improvement** +216mL vs. baseline at 36 weeks Potential to stabilize and improve lung function	Incremental efficacy +68.8mL vs. placebo at 52 weeks	Pooled efficacy: TETON-1&2 +111.8mL vs. placebo at 52 weeks	Incremental efficacy +45.5mL vs. placebo at 26 weeks
	Value Proposition	**Key Challenges**		
	Combination of unprecedented efficacy, favorable tolerability profile, and convenient dosing	Offers only incremental improvement in efficacy and tolerability	Inconvenient dosing and administration and difficult tolerability profile	Limited efficacy demonstrated at 26 weeks

Buloxibutid is a first-in-class AT2 receptor agonist with the potential to set a new standard in IPF



Upstream MoA with strong preclinical data

- AT2 receptor expressed on alveolar progenitor cell (AEC2)
- Upstream mechanism drives alveolar repair, resolves fibrosis, and promotes vascular function

Exceptional clinical data in the Phase 2a AIR trial

- Mean FVC change from baseline of +216 ml at 36 weeks, with benefit observed across all subgroups
- Synthetic control arm analysis confirms robust treatment effect observed with buloxibutid
- Excellent gastrointestinal tolerability and no treatment-related SAEs
- Biomarker data highly supportive of suggested MoA

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

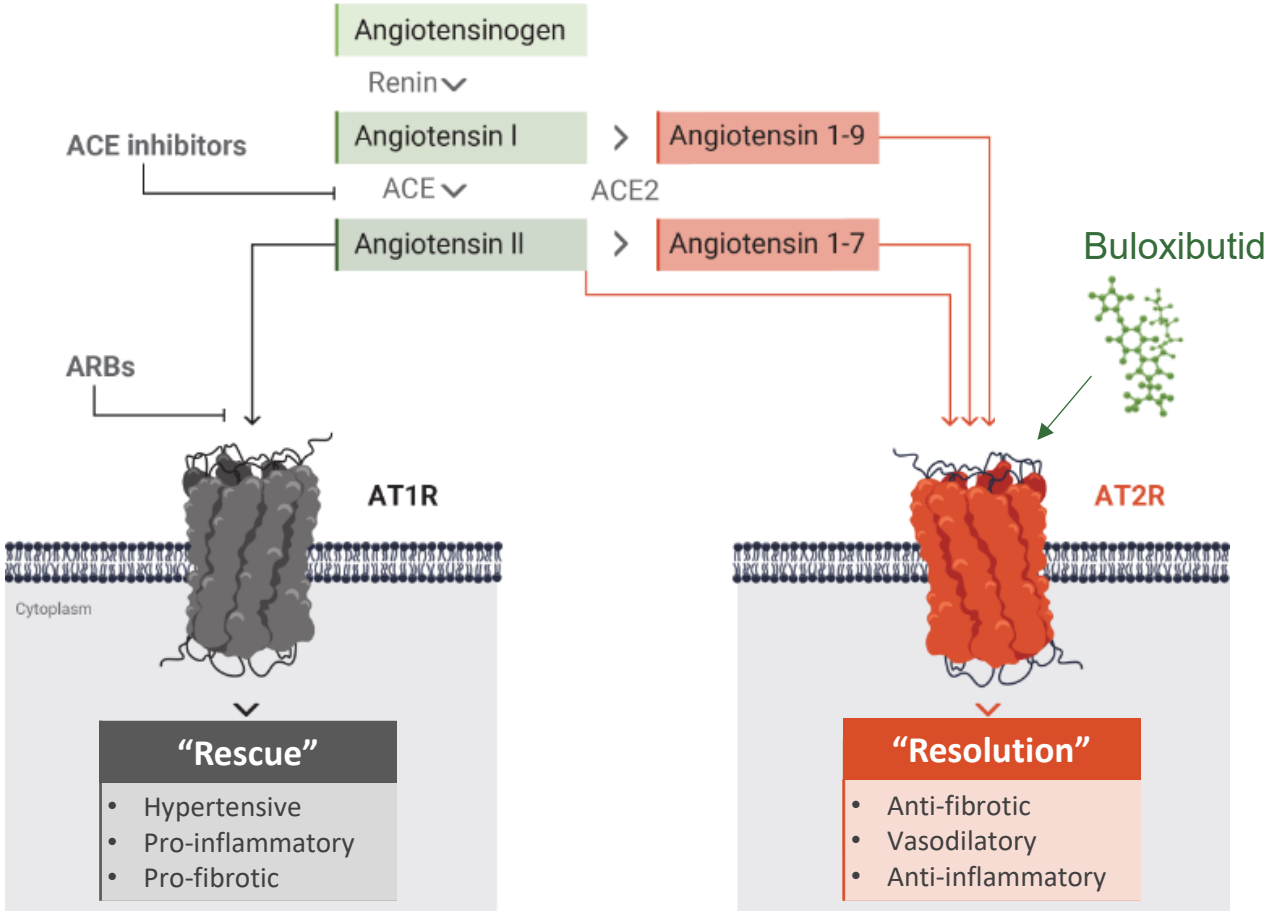
- 52-week treatment
- Fully enrolled (n=378)
- IPF patients on stable nintedanib/SoC or not on SoC[1]
- Global footprint

ASPIRE

vicore pharma

1. As nerandomilast is now approved in the United States, patients receiving stable background nerandomilast therapy will be eligible for enrollment in the trial, though this is anticipated to represent a small subset of the study population.

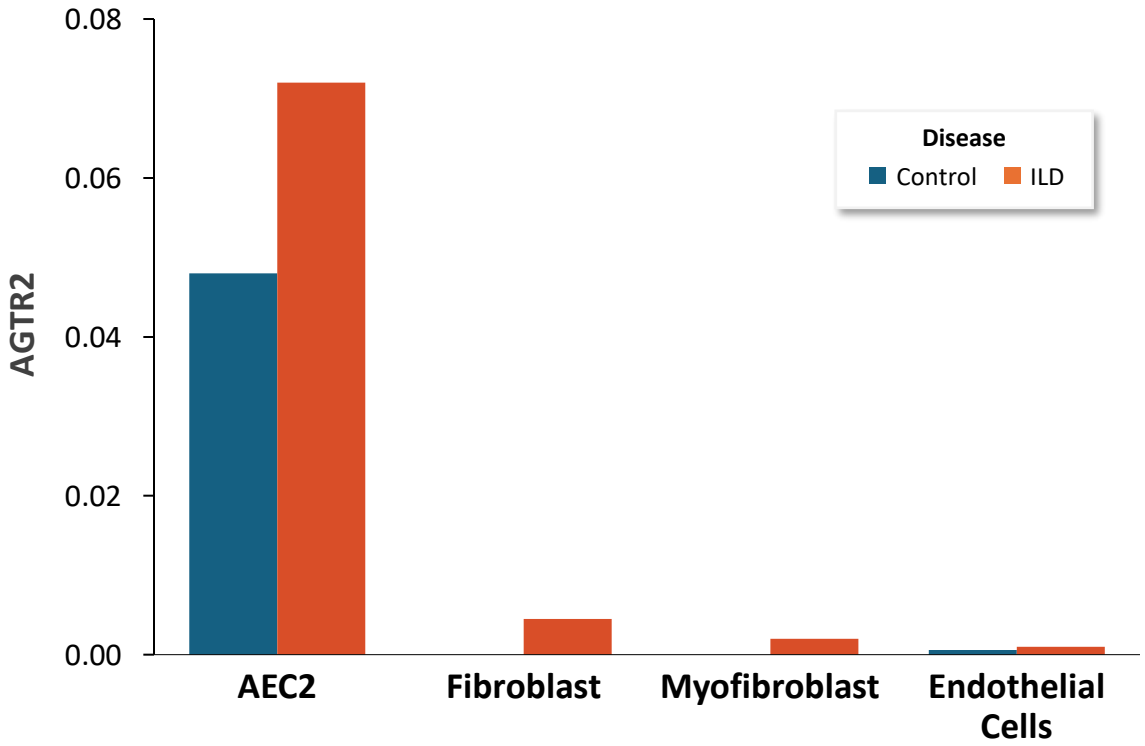
AT2R agonism is an upstream intervention driving tissue repair



- AT2R is constitutively expressed in the lung, primarily on alveolar epithelial type 2 cells (AEC2) – the "alveolar repair cell"
- AT2R activation engages tissue-protective pathways via AEC2s, promoting inhibition of fibrotic progression and fibrosis resolution, anti-inflammatory effects, vasodilation, and reversal of vascular remodeling
- Buloxibutid is an oral, selective AT2R agonist
- AT1R effects include increase in blood pressure, a key reason for ACE inhibitor and ARB development

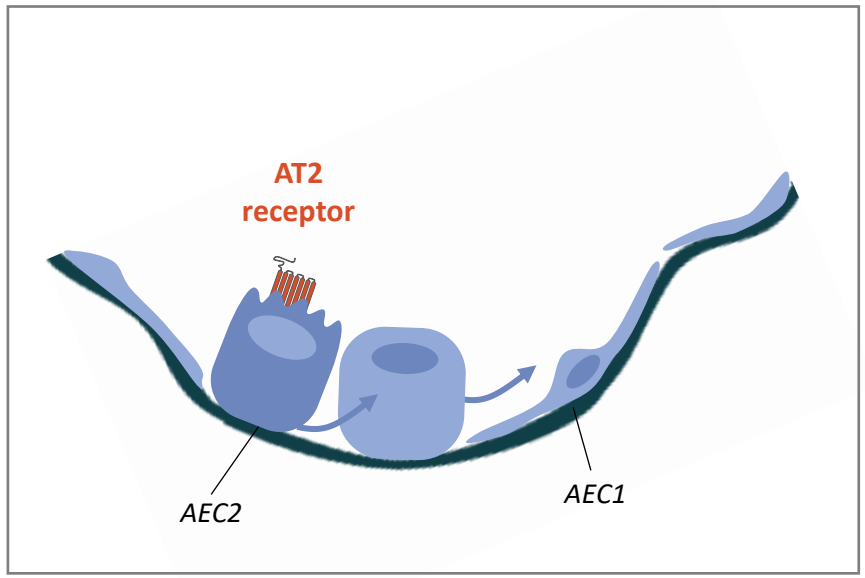
AT2R is highly expressed in human IPF lungs and on precursor AEC2s

AT2R Expression is Elevated in IPF Lung



AT2R expression is highly upregulated in IPF lungs, particularly on AEC2s, and is also present on fibroblasts, myofibroblasts, and endothelial cells with higher expression in the diseased state compared with healthy tissue

AT2R is Highly Expressed on AEC2s

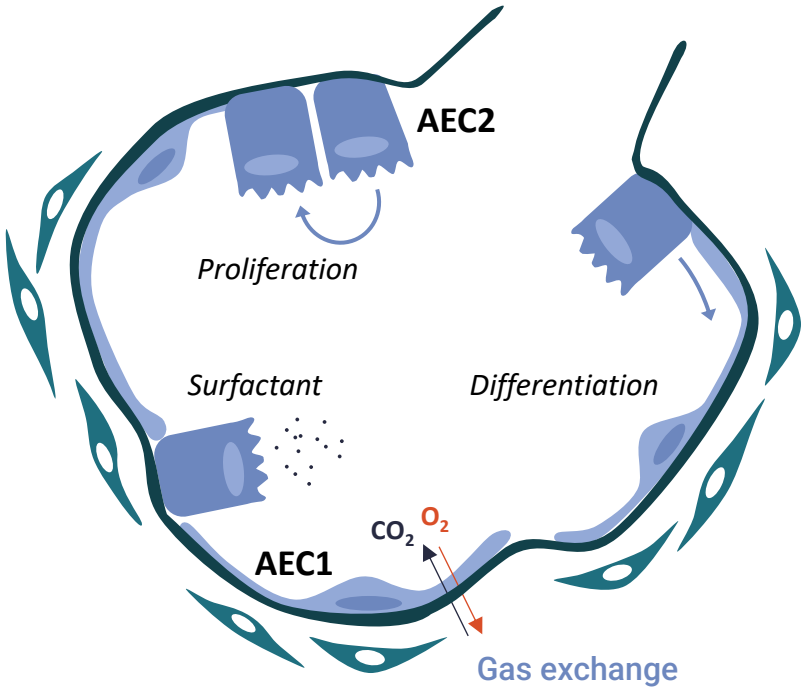


Single-cell analysis shows high AT2R expression on AEC2 in the lung, the progenitor cell that differentiates into AEC1 gas exchange cells

Source: Adapted from IPF Cell Atlas (Banovich/Kropski)

Alveolar epithelial cells are critical for healthy lung function

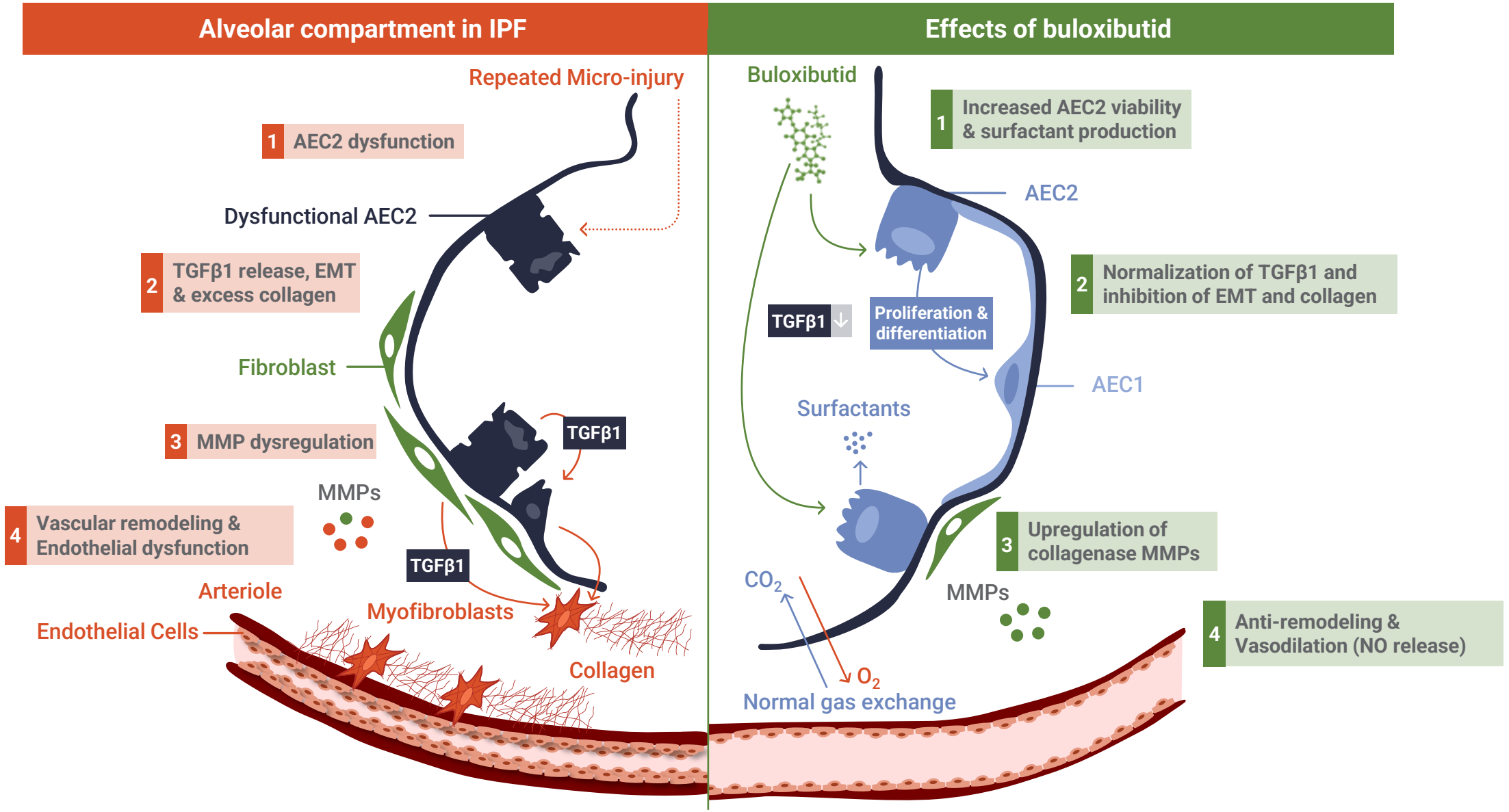
Healthy alveolus



AEC – Alveolar Epithelial Cell

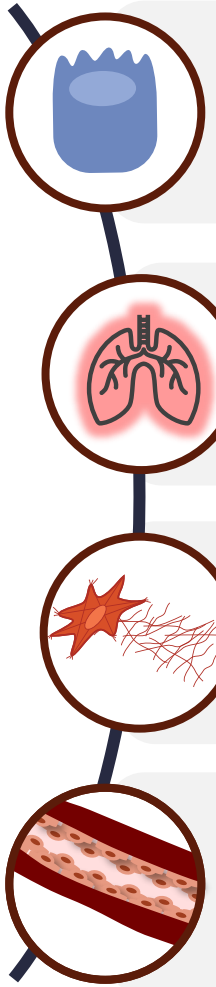
- The alveolar epithelium is exposed to damaging irritants in inhaled air
- AEC1 is the predominant alveolar cell type and is responsible for gas exchange
- AEC2 is a progenitor cell that is critical for alveolar integrity and function:
  - Proliferates to form new AEC2
  - Differentiates to AEC1 that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

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



EMT = Epithelial–mesenchymal transition; MMPs = Matrix metalloproteinases

Buloxibutid addresses all main disease drivers in IPF and disease modification through tissue repair



Tissue repair and regeneration
Buloxibutid drives tissue repair by targeting precursor epithelial cells (AEC2), offering a disease-modifying mechanism of action

Anti-inflammatory
Buloxibutid inhibits release of pro-inflammatory cytokines through inhibition of NF-κB signaling

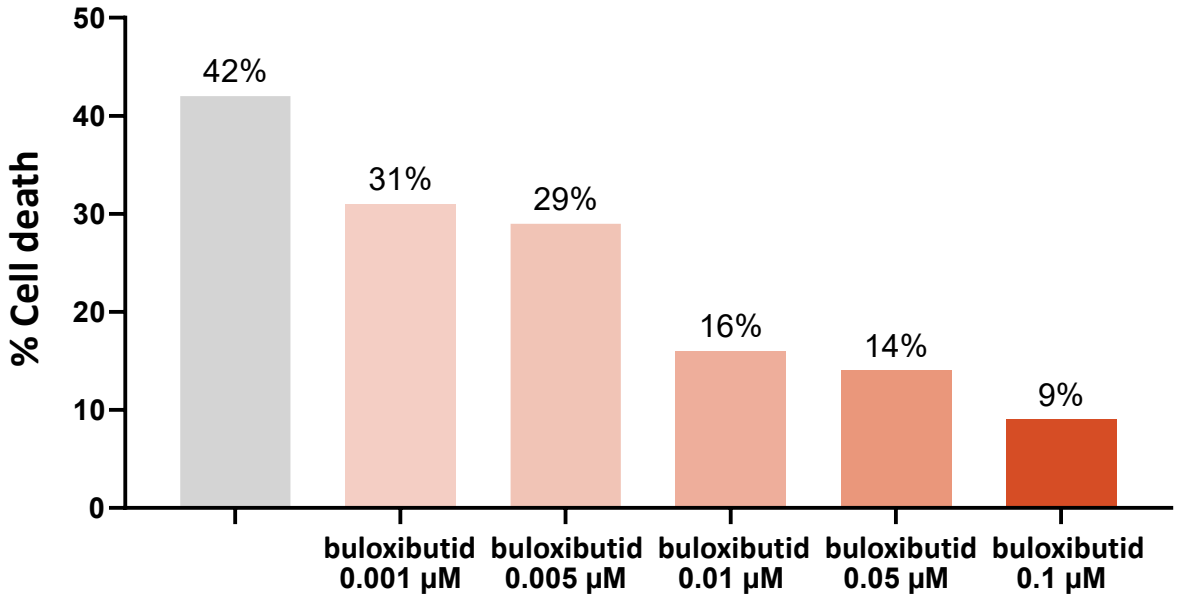
Anti-fibrotic
Buloxibutid restores dysfunctional AEC2 and surfactant production, normalizes TGFβ1 levels, inhibits EMT and collagen deposition, as well as breaks down existing collagen build up

Reverses vascular remodeling
Buloxibutid reverses vascular remodeling and drives vasodilation through NO release

EMT = Epithelial–mesenchymal transition

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]

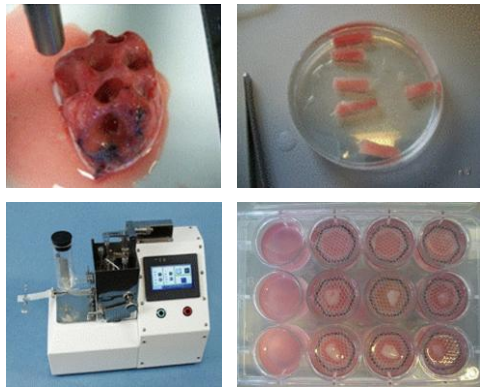
- Human precision cut IPF lung slices ± 1 µM buloxibutid
- One patient, 5 pooled lung slices

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

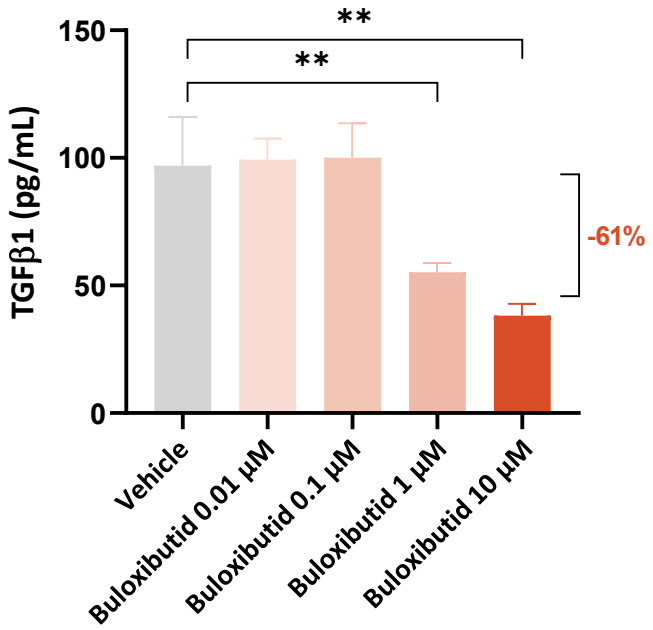
Buloxibutid reduces TGFβ1 and collagen in human IPF lung slices

Human precision cut lung slices (PCLuS)

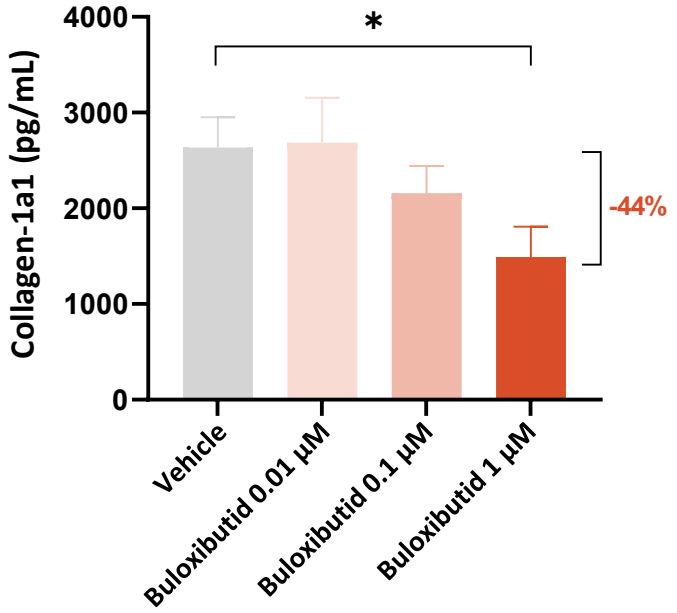


- Lung tissue collected from IPF patients undergoing transplant
- Intrinsic fibrosis, no stimuli added

TGFβ1 protein levels in PCLuS



Collagen protein levels in PCLuS

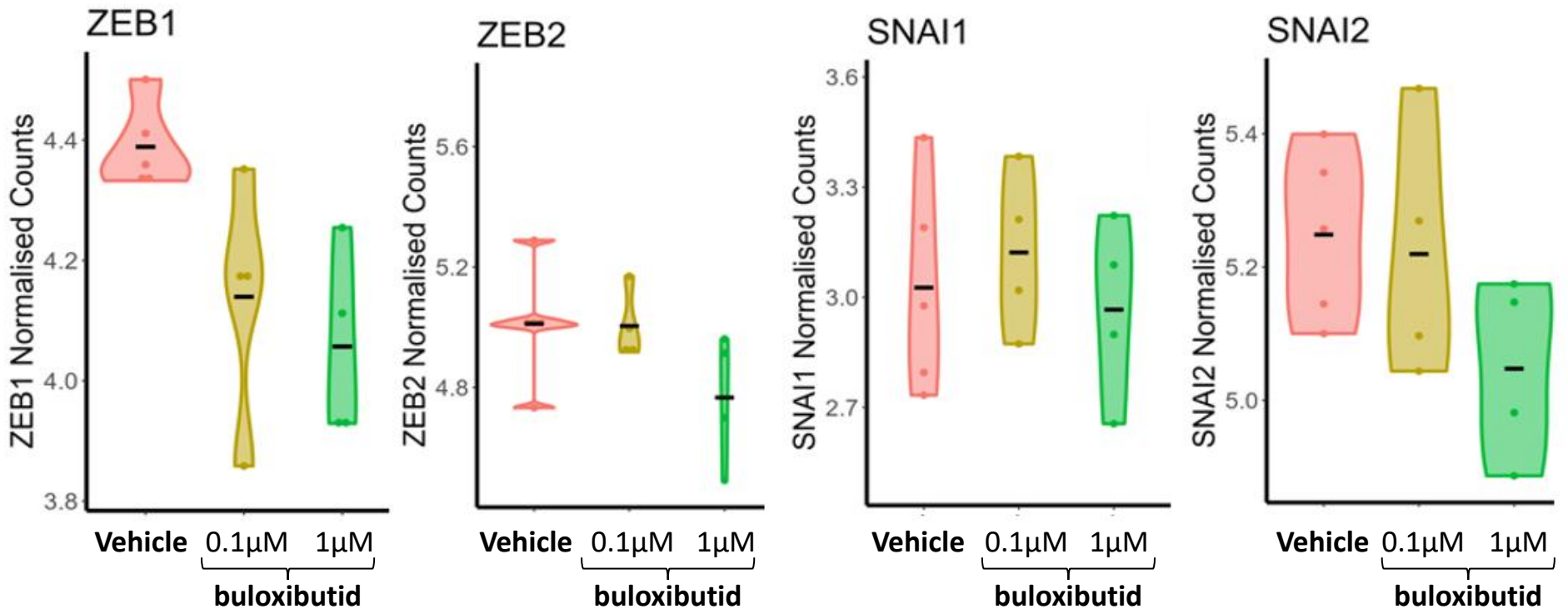


Dose-dependent reduction of TGFβ1 and Collagen-1a1 protein

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

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

Buloxibutid downregulates expression of EMT transcription factors in AEC2

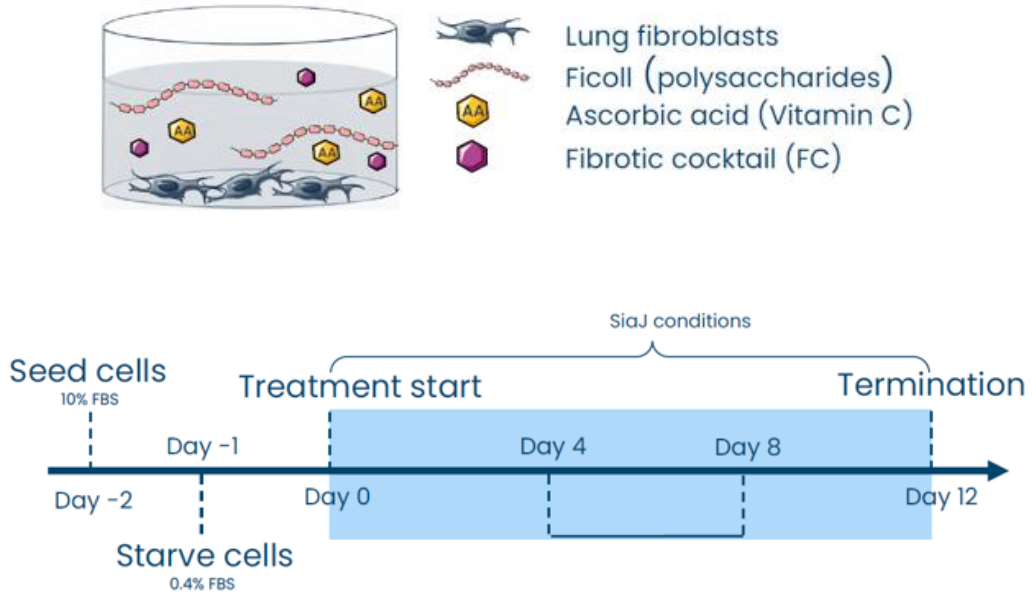


- Primary AEC2 cultures established from normal surgically resected human lung
- Buloxibutid treatment under baseline conditions with no stimuli added

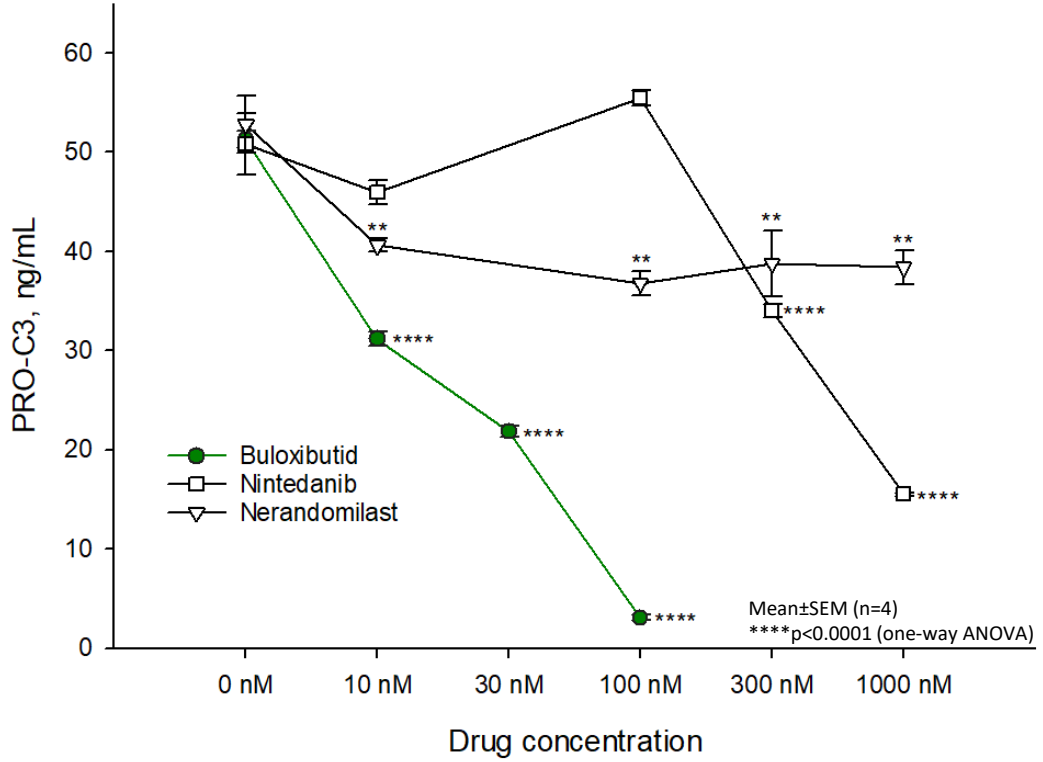
Source: Conforti et al. 2023, manuscript (Vicore collaboration with U Southampton)

Buloxibutid potently inhibits fibrosis in a human lung fibroblast assay

Human lung fibroblast assay methodology



Impact on type III collagen biomarker PRO-C3



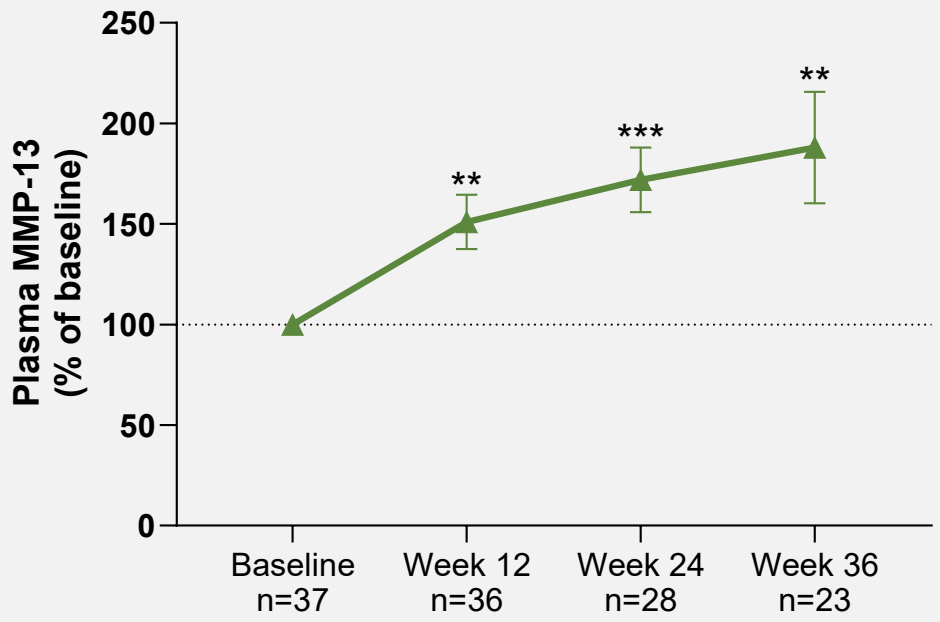
Buloxibutid potently and dose-dependently inhibited PRO-C3, reflecting inhibition of type III collagen formation and fibrotic activity. The superior in vitro performance of buloxibutid vs. nintedanib and nerandomilast on the IPF biomarker PRO-C3 reflecting fibrotic progression underscores its robust anti-fibrotic mechanism of action

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

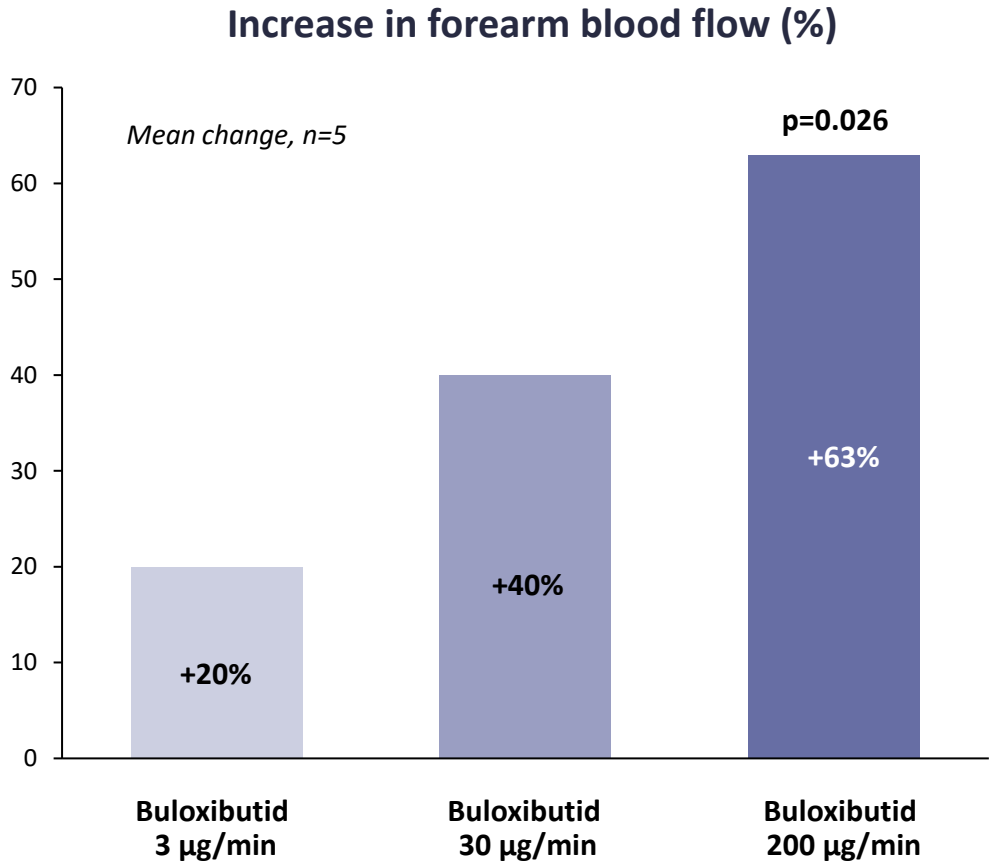
ECM = Extracellular Matrix; MMP = Matrix Metalloproteinases

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

Buloxibutid's vascular effects are clinically validated

Buloxibutid's vascular effects (vasodilation) are clinically validated in a forearm blood flow trial in healthy volunteers

- Buloxibutid shows dose-dependent increase in local blood flow
- Blood flow increased by 63% (p=0.026), without reducing systemic blood pressure or causing other side effects
- Local blood concentrations of buloxibutid in line with those reached with oral treatment
- No severe or serious TEAEs were reported



Buloxibutid addresses endothelial dysfunction and increases local blood flow, mediated by nitric oxide (NO) released from the endothelium

Source: Vicore data on file

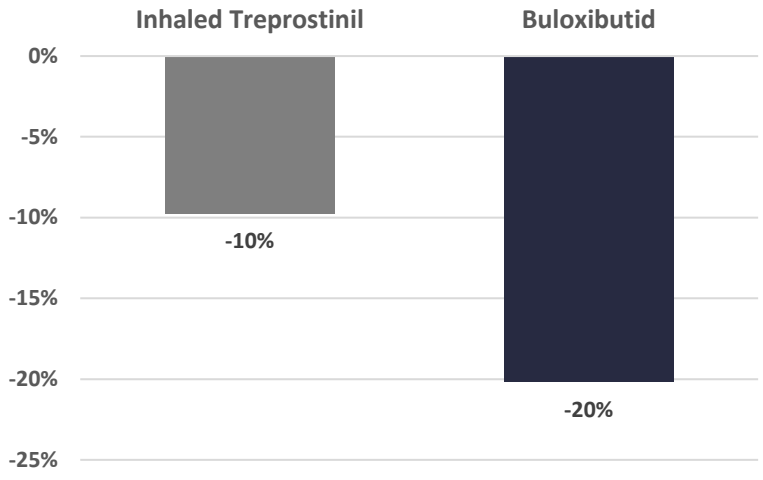
Buloxibutid shows greater reduction in key hemodynamic and vascular remodeling parameters compared to inhaled treprostinil in preclinical Sugen-Hypoxia rat model

Inhaled treprostinil and buloxibutid were evaluated in separate studies using the same study protocol

Hemodynamic Parameters

RVSP

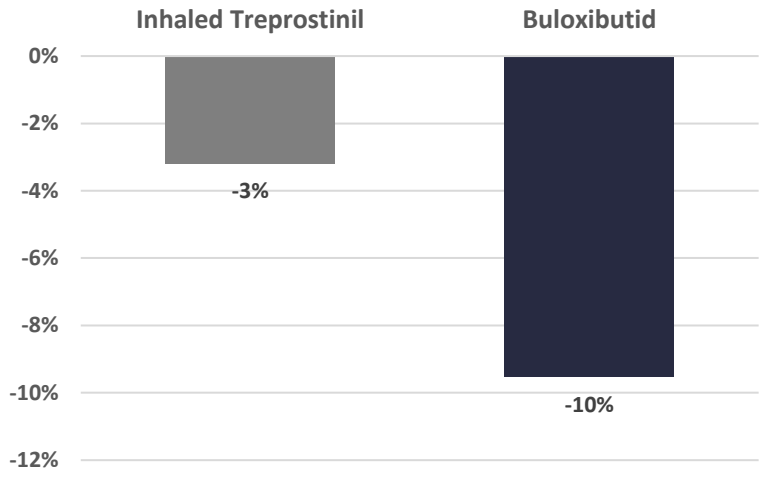
Reduction in RVSP from SuHx control



Right Ventricular Remodeling

Fulton's Index

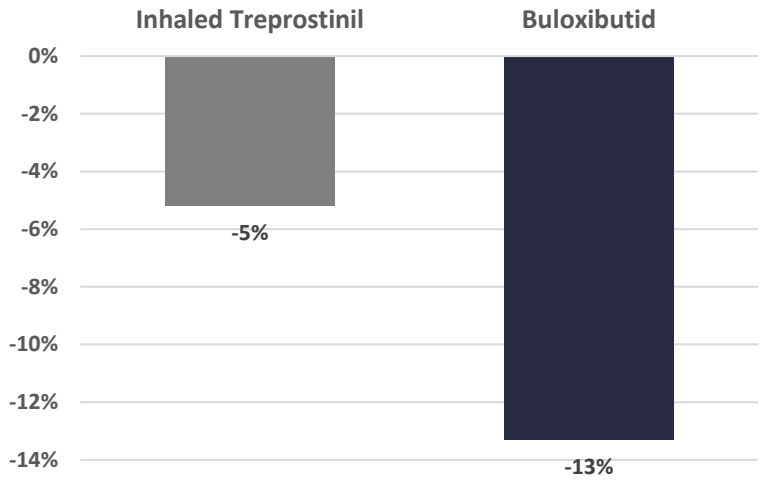
Reduction vs. SuHx control



Pulmonary Vascular Remodeling

Wall Thickness

Reduction vs. SuHx control



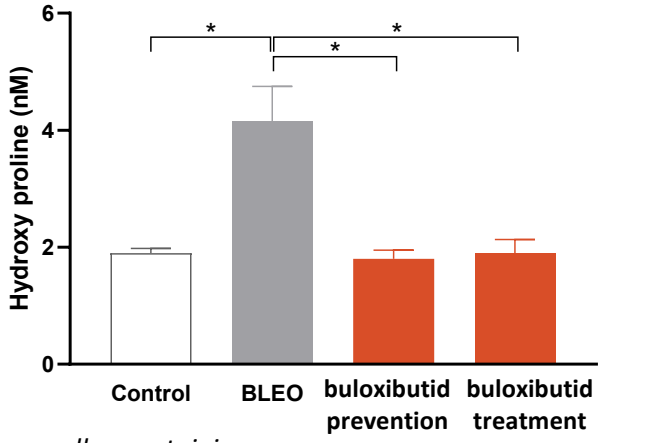
***Inhaled treprostinil**: adapted from Corboz, et al., J. Pharmacol. Exp. Ther. 2022 – Dose: 65 µg/kg*
***Buloxibutid**: adapted from Tornling, et al., Int. J. Mol. Sci. 2023 – Dose: average result of 2 mg/kg and 20 mg/kg dose*

Clinically relevant concentrations of buloxibutid show greater reduction compared to the Sugen-Hypoxia control than clinically relevant dose of inhaled treprostinil across key readouts, including RVSP, mPAP (data not shown), Fulton's index, wall thickness and muscularization (data not shown)

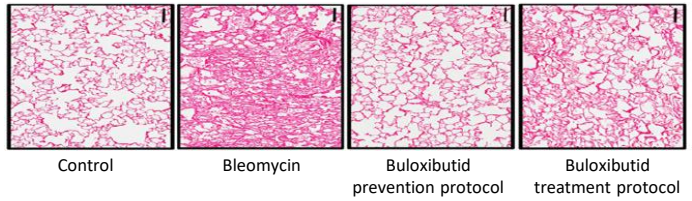
SuHx = Sugen-Hypoxia; **mPAP** = mean pulmonary artery pressure ; **RVSP** = right ventricular systolic pressure

Strong preclinical in vivo evidence for buloxibutid in pulmonary fibrosis

Bleomycin



Lung collagen staining

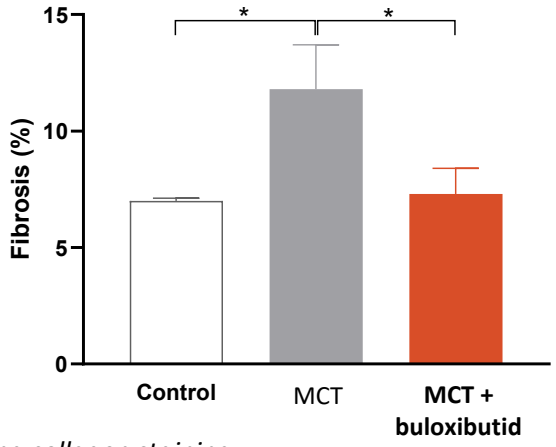


- Buloxibutid 0.03 mg/kg/day i.p. once daily, 14 days (Treatment). N=5 control and 7-8 in treatment groups

Adapted from (1)

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

Monocrotaline



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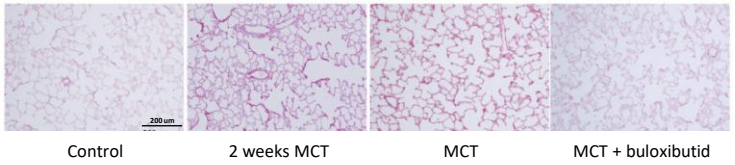
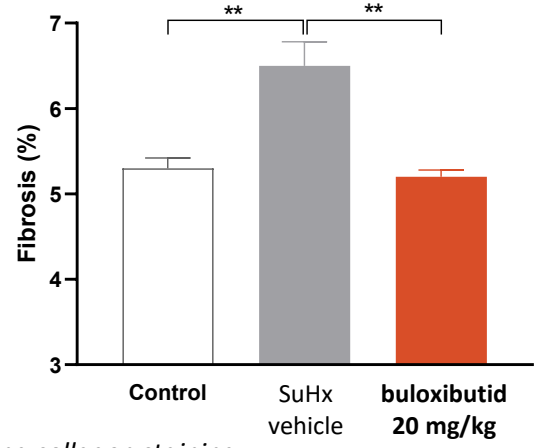


- Buloxibutid 0.03 mg/kg/day i.p. once daily for 2 weeks
- N=14 per treatment group

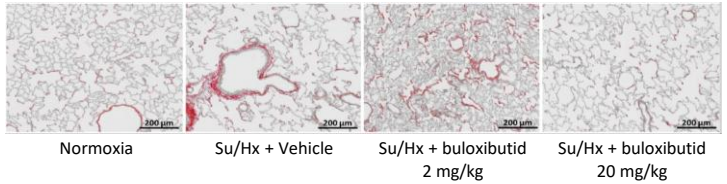
Adapted from (2)

- **Reversal of pulmonary fibrosis and prevention of right ventricular fibrosis**
- **Reversal of vascular remodeling and improved right heart function**

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**
- **Reversal of vascular remodeling**
- **Reduced RVSP and right ventricular hypertrophy**

(1) Rathinasabapathy et al. Front Physiol. 2018; (2) Bruce et al. Br J Pharmacol. 2015; (3) Tornling et al. Int. J. Mol. Sci. 2023;
RVSP = Right Ventricular Systolic Pressure

Buloxibutid has an extensive and robust safety database, with over 350 patients dosed across nine completed clinical trials

Buloxibutid has been tested extensively in the clinic, generating a robust safety database

- Not including patients enrolled in the ongoing Phase 2b ASPIRE trial, a total of **366 trial participants** have been exposed to buloxibutid over the course of 9 completed clinical trials
- In the recently completed Phase 2a AIR trial, IPF patients were exposed to buloxibutid for **36 weeks**

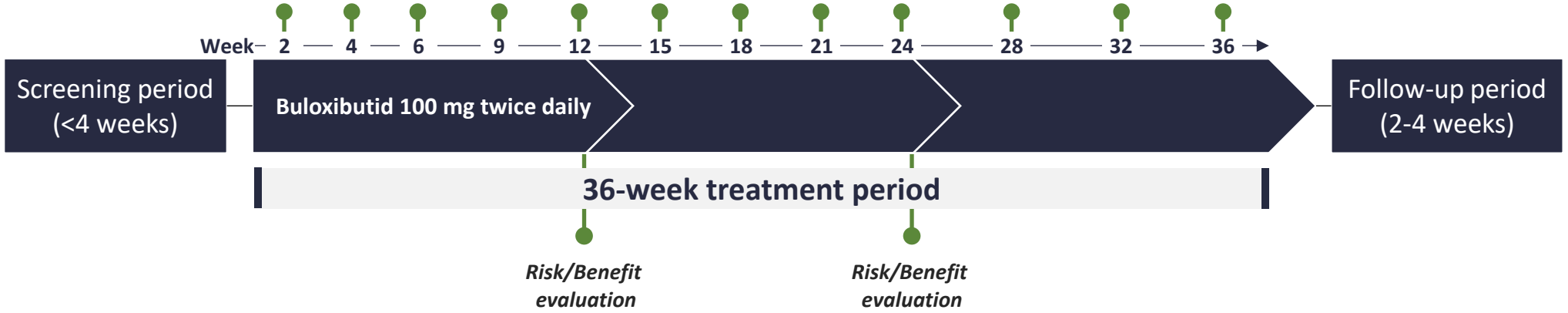
No significant safety risks have been identified for buloxibutid

- The only identified risk of treatment with buloxibutid is reversible, mild to moderate hair loss, observed in 19% of participants in the Phase 2a AIR trial
- Across the robust safety dataset, there have been no treatment-related SAEs

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)
Gender	Males	**77%**	80%
	Females	**23%**	20%
Ethnicity	White	**27%**	57%
	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)
% SoC	Pirfenidone	**0%**	0%
	Nintedanib	**0%**	0%

In line with other trials.

Enrolled study population has disease progression comparable to global IPF study populations.

In line with other trials.

As with the INPULSIS trials, AIR patients were treatment-naïve.

(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

	Comparison to SoC		Buloxibutid
	Ph3 INPULSIS-1 *52-week treatment*[1]		**Ph2a 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

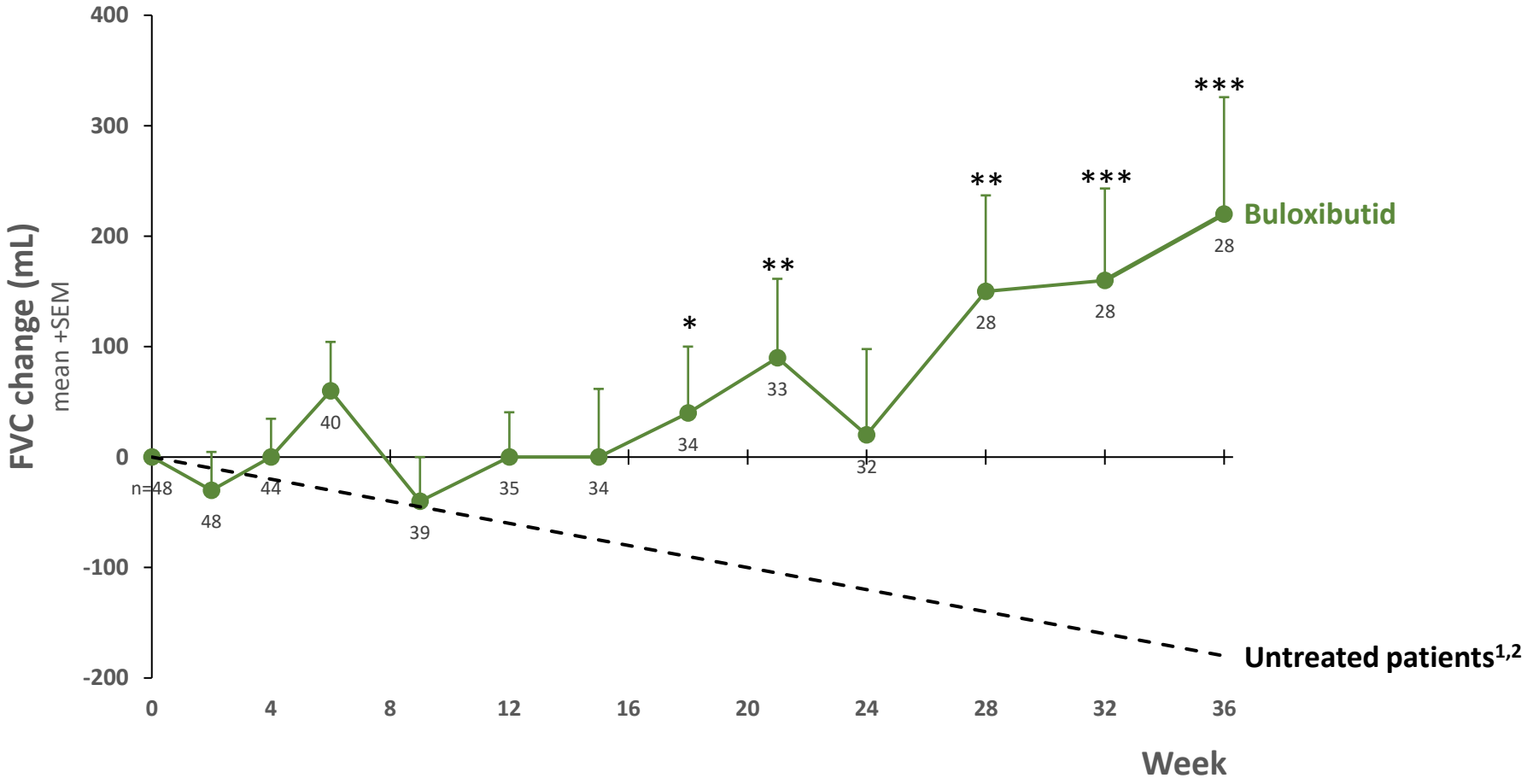
Low rate of exacerbations and cough worsening

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.

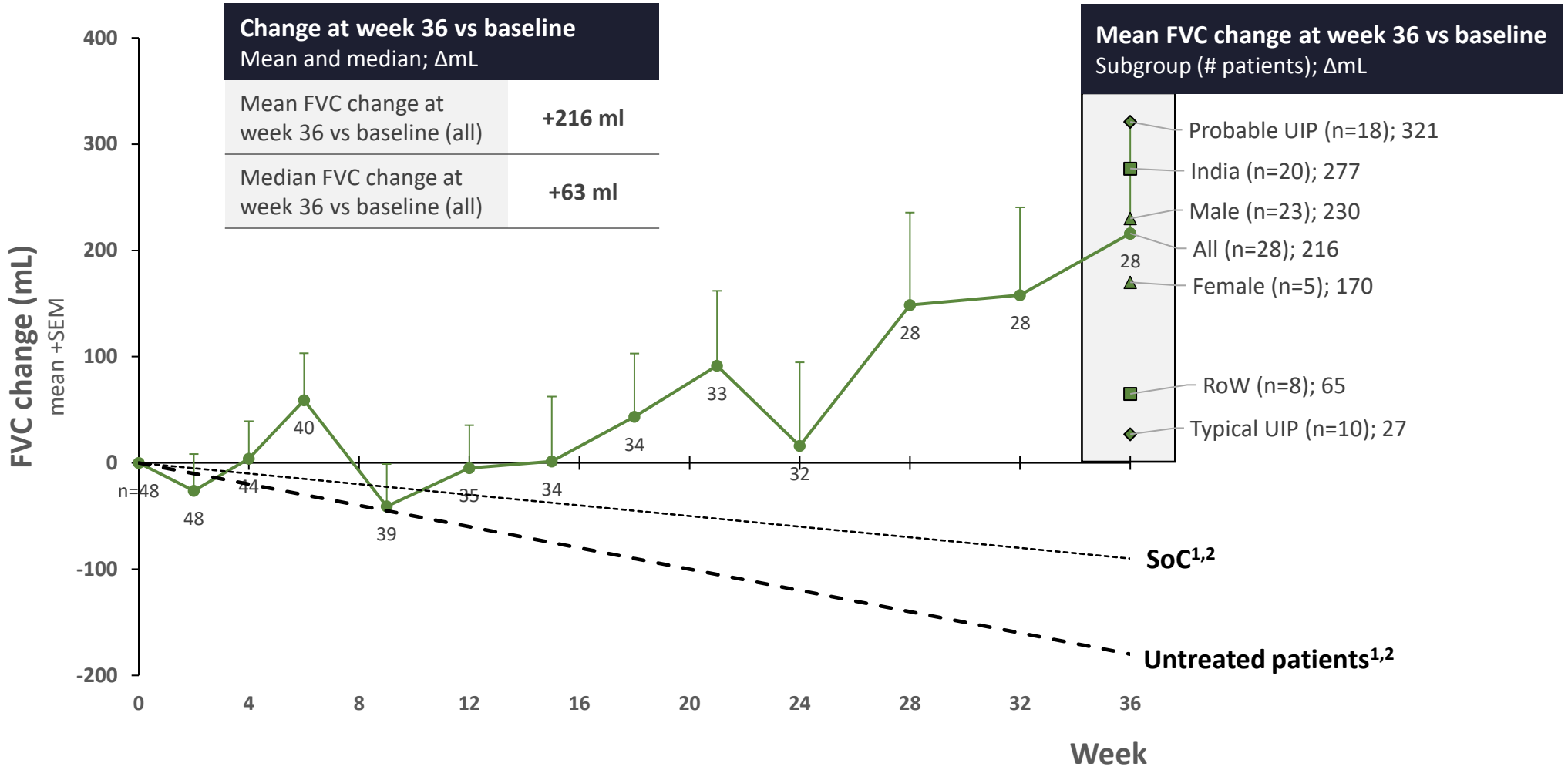
Buloxibutid stabilizes and improves lung function over the 36-week AIR trial



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. N 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. N Engl J Med 2014; 370:2071-2082

Development of a Synthetic Control Arm analysis to contextualize buloxibutid's effect in the AIR trial

IPF Patient External Control Data

- Over 10,000 patients
- Mean 12-month FVC change -170.3 ml

Filter IPF Patient Data using Air Trial Inclusion Criteria

- Age >= 40 years
- FVC % predicted >= 60%
- FEV1/FVC >= 70%
- Treatment-naïve patients

External Control Arm Generation

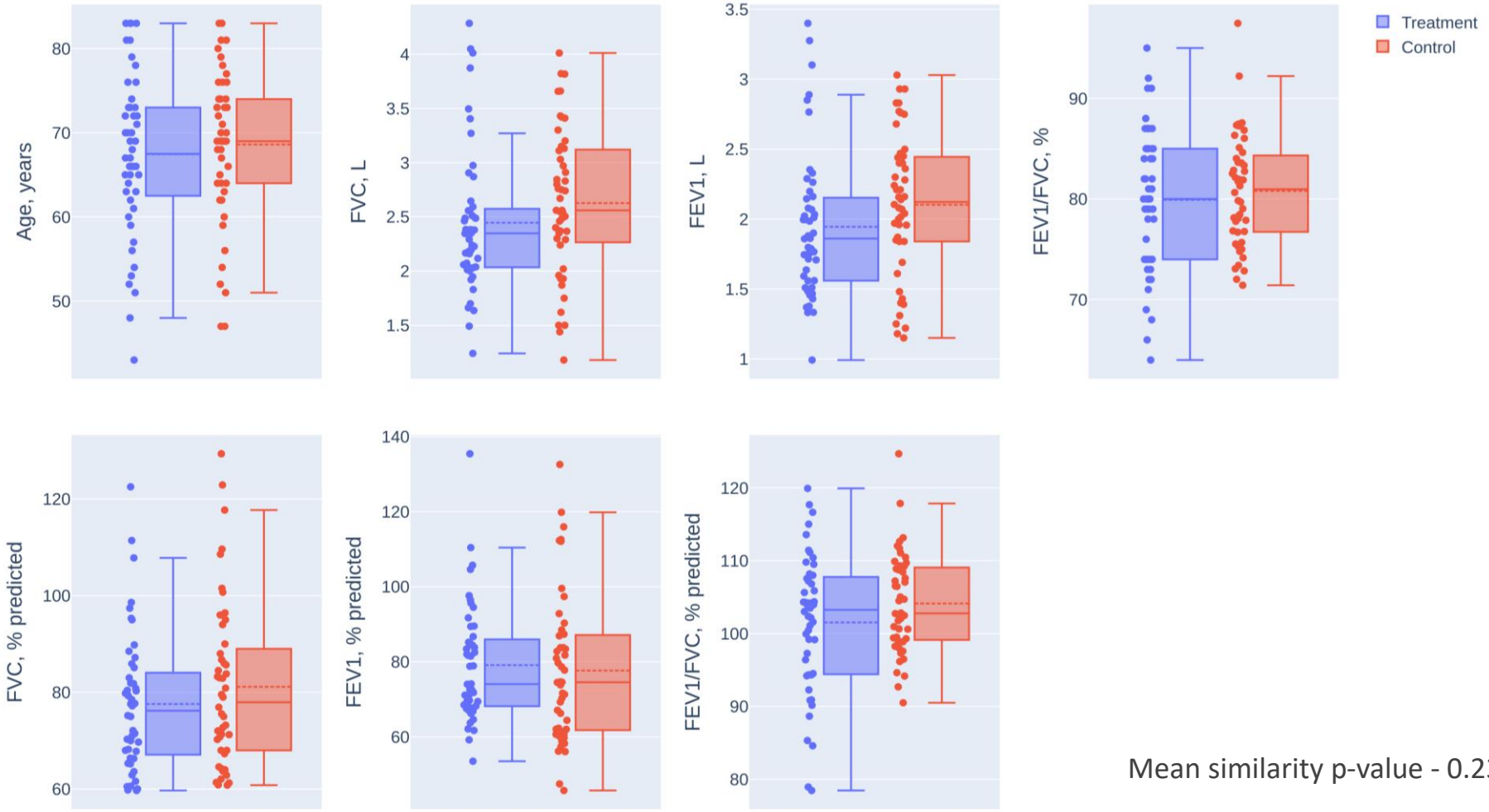
- ~30,000 randomly sampled groups of 48 patients generated from filtered control data
- 1:1 treatment-control ratio
- Matching on 8 clinical variables at baseline
- Univariate and multivariate tests of similarity with control
- 408 randomly sampled groups accepted as control arms based on the baseline similarity tests
- Mean similarity p-value = 0.23 (SD = 0.05)

Efficacy Testing

- Follow-up time – 36 weeks
- AIR – mean FVC change +23 ml
- Control – mean FVC change -114.8ml

Note: Qureight analysis

IPF patients selected for the Synthetic Control Arm analysis are highly matched to the Phase 2a AIR patient baseline characteristics

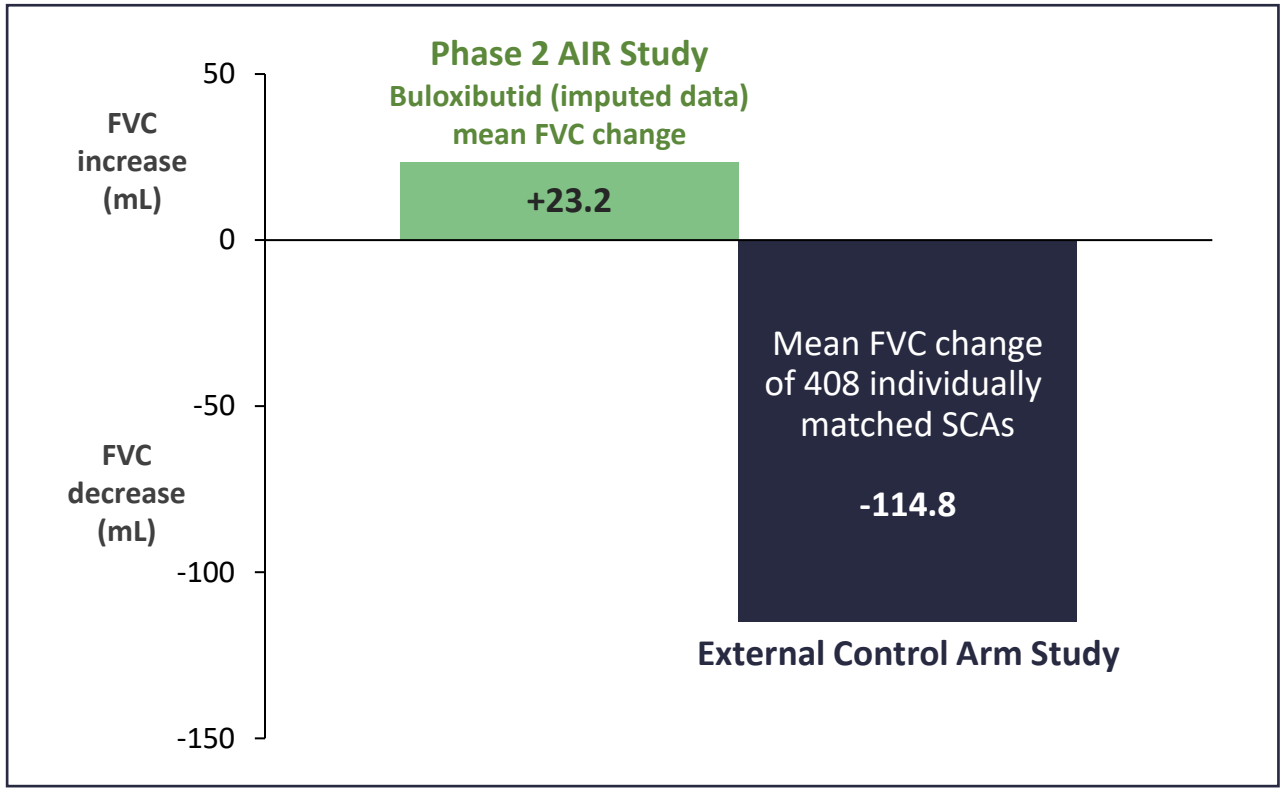


Mean similarity p-value - 0.23 (SD=0.05)

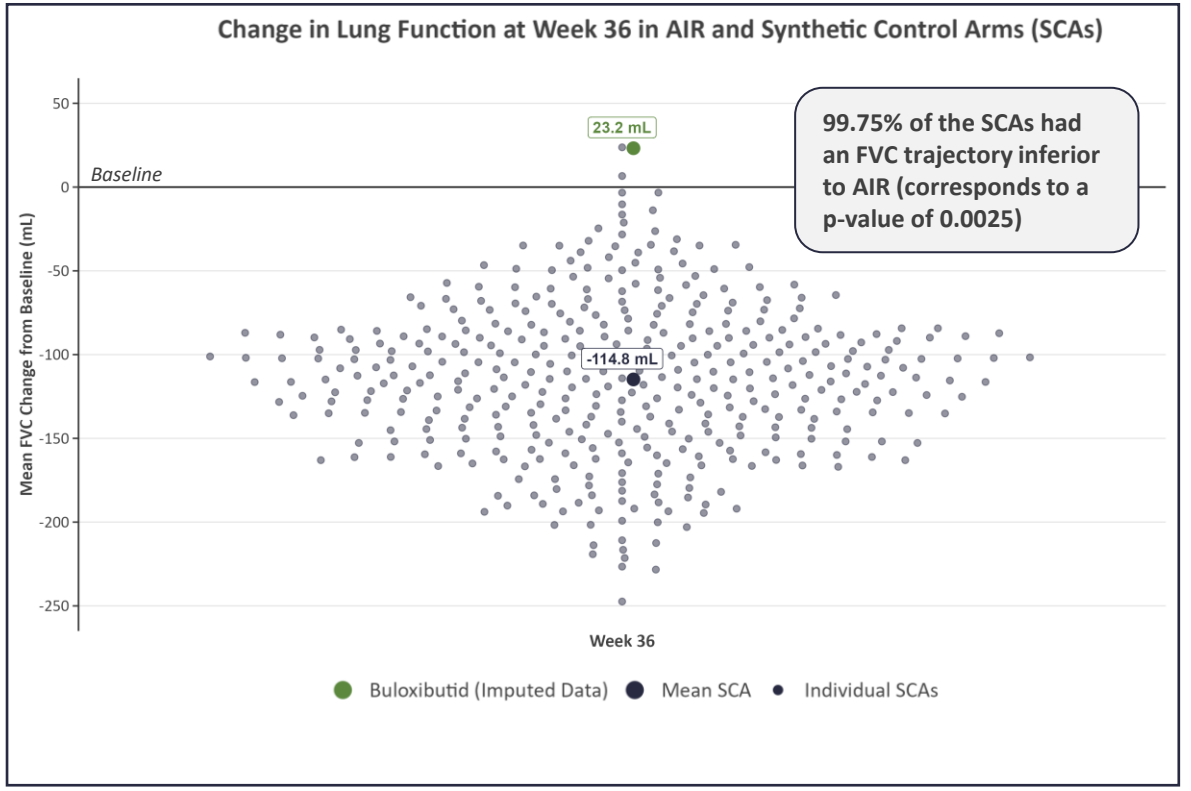
Note: Qureight analysis

A Synthetic Control Arm analysis demonstrates buloxibutid's robust treatment effect

Change in FVC in the Phase 2a AIR IPF trial compared to the external control arm study



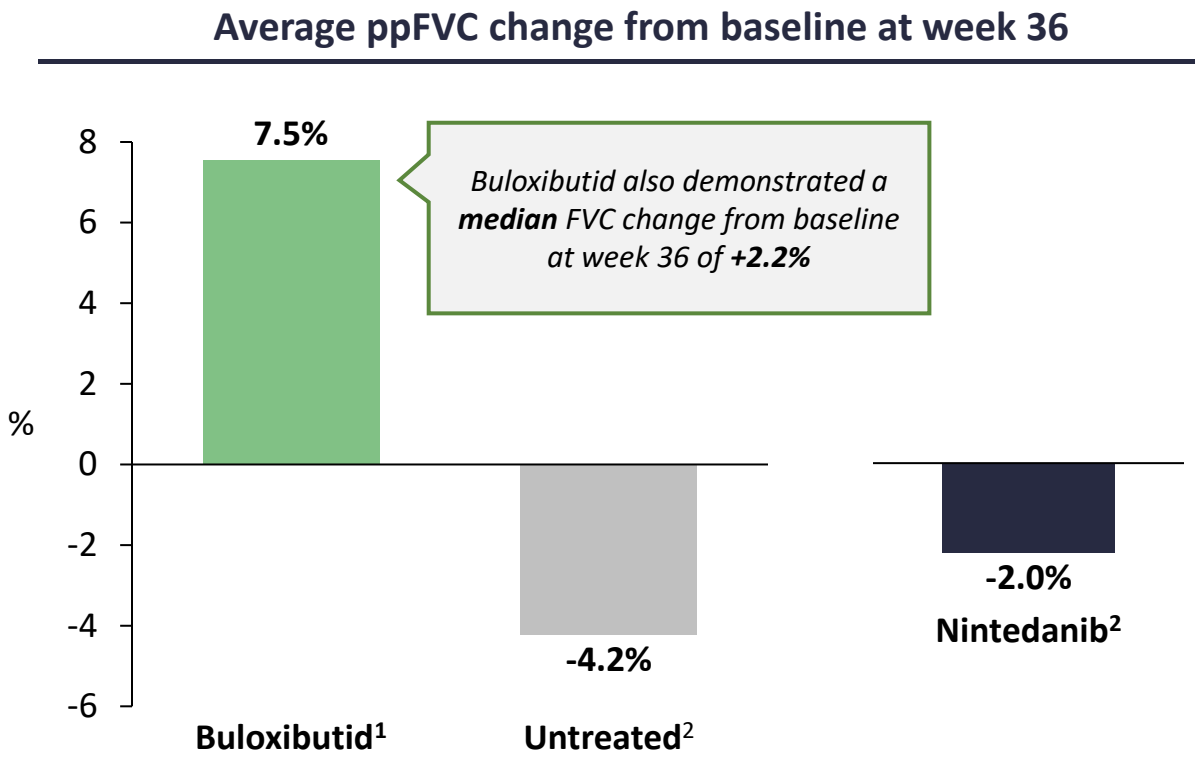
408 individual matched Synthetic Control Arms (SCAs) generated by Monte Carlo cross validation



The Monte Carlo approach demonstrates that in patients without significant differences in core baseline parameters, buloxibutid showed statistically significant treatment effect compared to control FVC distribution

Note: Qureight analysis

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

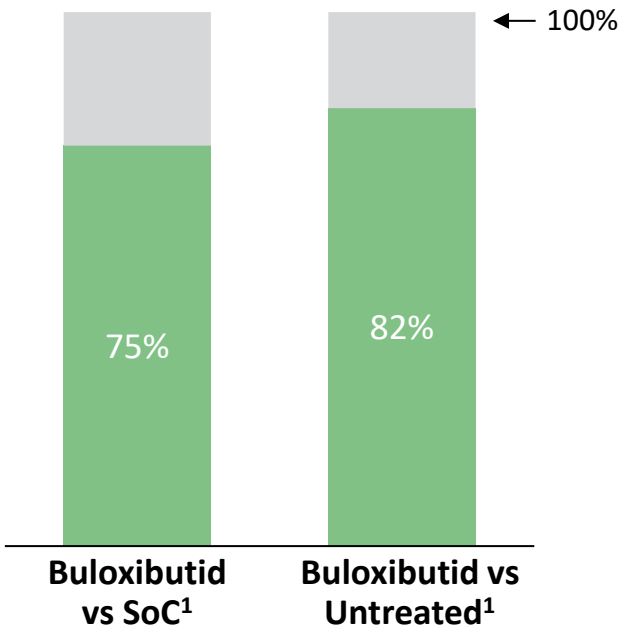


Average increase of 7.5% percent predicted FVC at 36 weeks from baseline

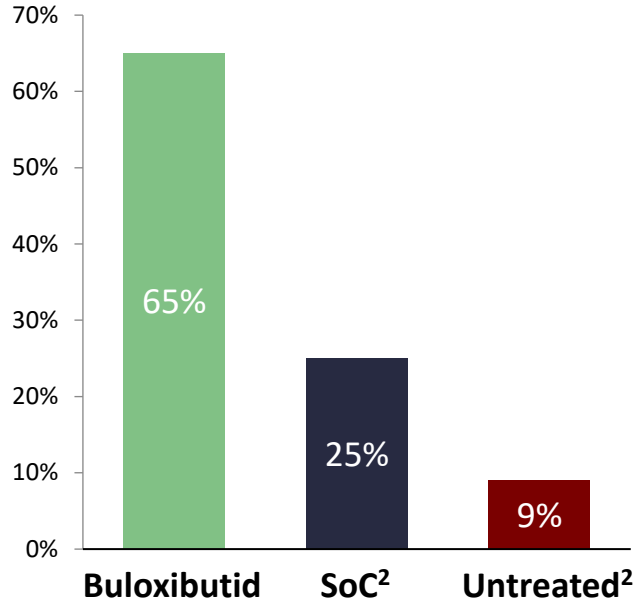
(1) Average ppFVC at 36 weeks (n=28); (2) 36 week estimated ppFVC for nintedanib and placebo, INPULSIS trial (Richeldi et al. N Engl J Med 2014; 370:2071-82)

Buloxibutid outperforms historical standard of care and untreated decline at 36 weeks

Percentage of patients outperforming expected ΔFVC



Percentage of patients with improved lung function (FVC) vs baseline



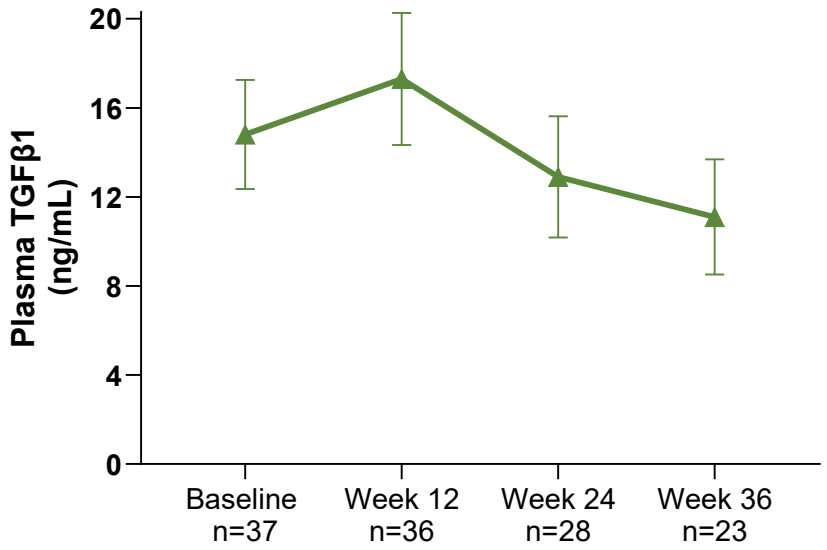
Buloxibutid outperforms expected change in FVC of untreated patients and those treated with current standard of care at 36 weeks

Most patients treated with buloxibutid experience improved lung function at 36 weeks, outperforming historical SoC and untreated patients

(1) Based on historical data (Richeldi et al., 2014); (2) Based on historical data (Flaherty et al., 2018)
Note: 25% of patients treated with nintedanib over 52 weeks showed improvement or no decline in FVC in the INPULSIS trials, and for untreated patients the number was 9%.

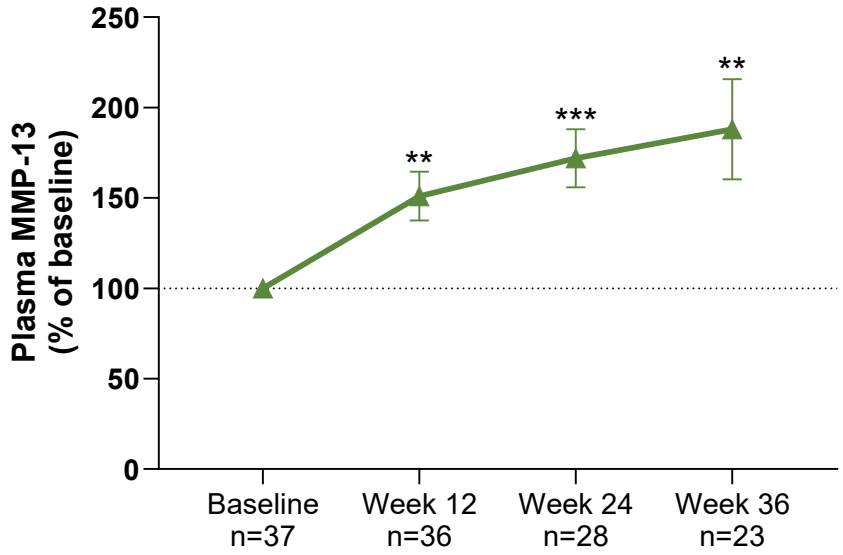
Buloxibutid increases collagenase MMP-13 and drives a trend of decreased TGFβ1

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: Robust randomized trial with global footprint

ASPIRE is a randomized, double-blind, placebo-controlled, parallel-group, multicenter, dose-finding trial

Trial Design

Screening

Randomization 1:1:1

n=378

- Buloxibutid 50mg BID
- Buloxibutid 100mg BID
- Placebo BID

IPF patients stable on nintedanib/SOC or not on SOC[1]
(no access, refused, intolerant or failed)

Follow-up

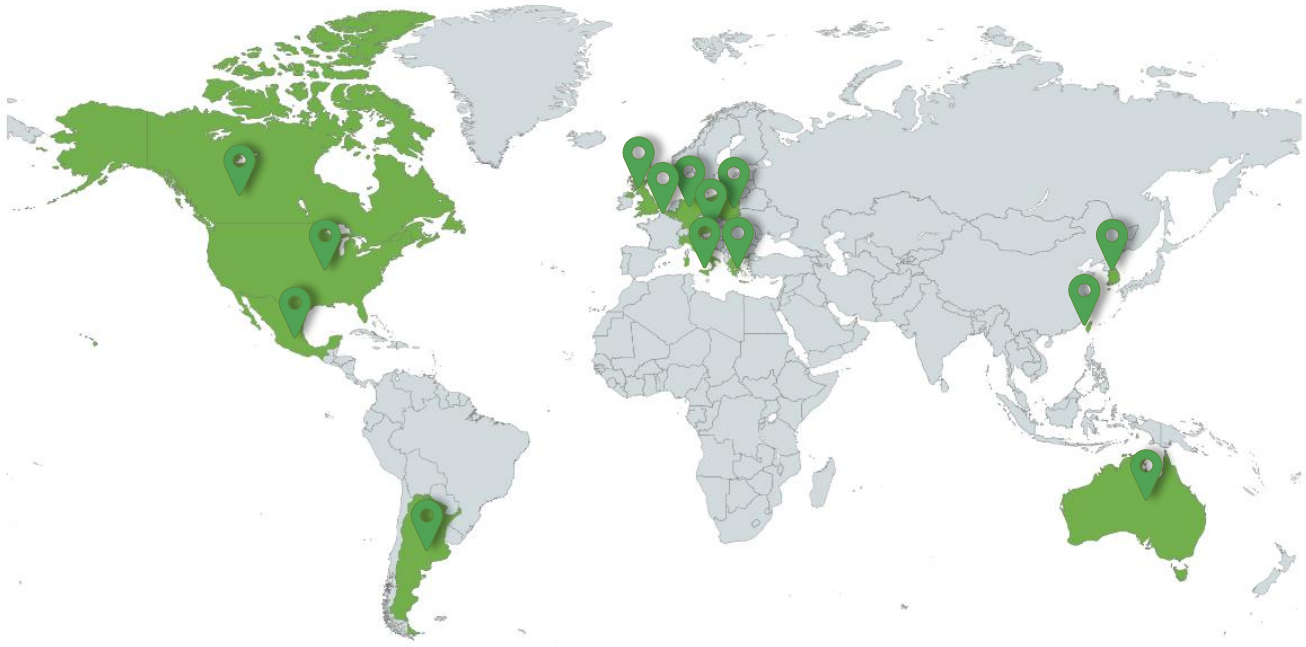
Baseline — **52-week treatment duration** — Last Dose

Primary Endpoint:
Change from baseline in FVC at 52 weeks

Key Secondary Endpoint:
Proportion of patients with disease progression at 52 weeks

Global Footprint

~120 sites across 14 countries



1. *As nerandomilast is now approved in the United States, patients receiving stable background nerandomilast therapy will be eligible for enrollment in the trial, though this is anticipated to represent a small subset of the study population.*

ASPIRE includes a pre-planned, independent futility analysis

An independent Data Monitoring Committee (iDMC) will conduct a pre-specified futility assessment once ~27% of patients have accrued data for the primary endpoint analysis.

Rationale

Purpose of the futility analysis:

Protect patients participating in the trial

Reduce risk of a negative readout at time of final analysis

Preserve trial integrity through a well-established futility approach consistent with regulatory expectations

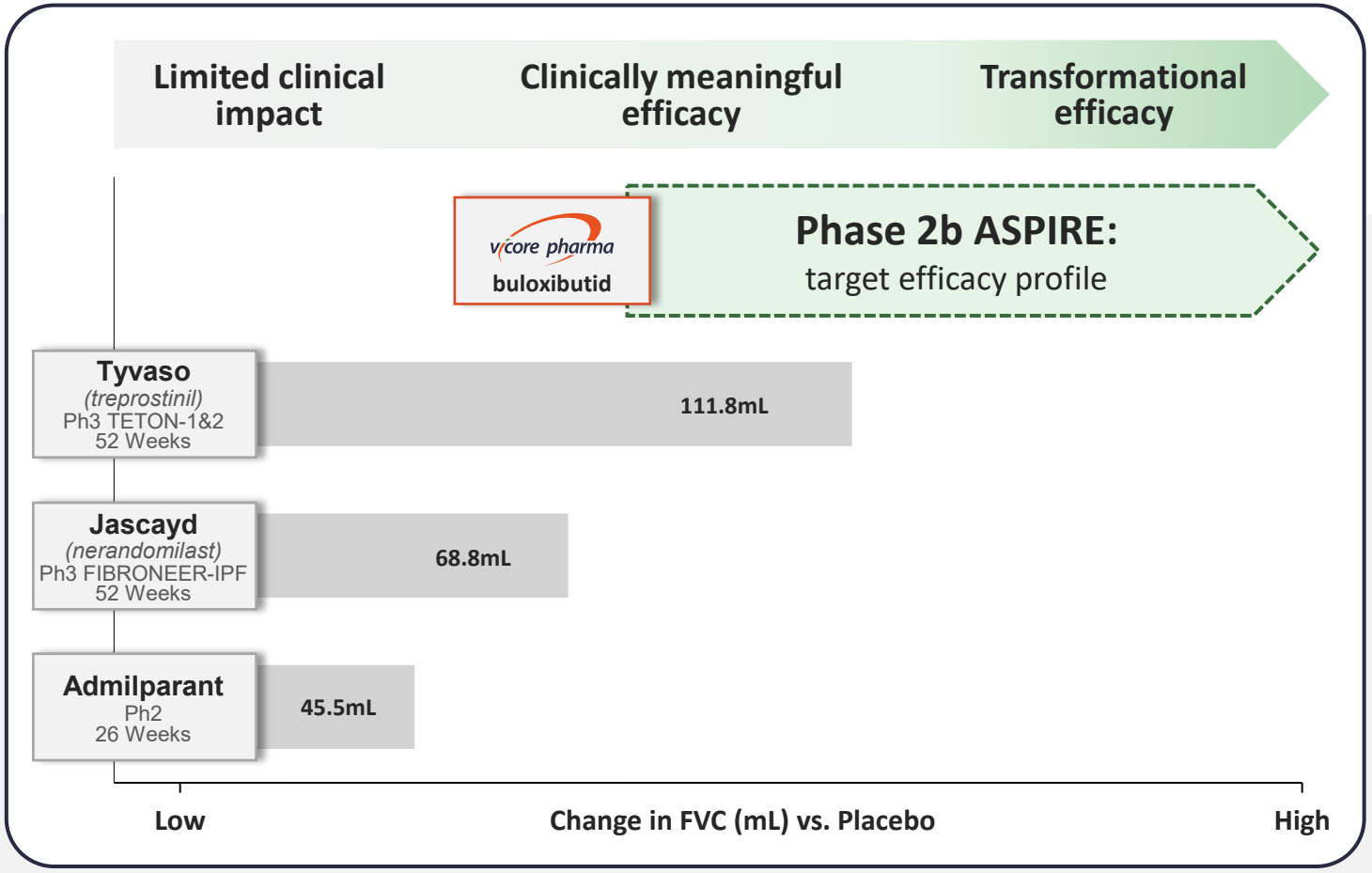
Method

- Based on efficacy data from the first **~100 randomized patients**
- Uses a **conditional power approach**, to estimate probability of success at final readout
- Stopping threshold set conservatively; stop for futility will be considered if the conditional power is **lower than 20%**

Timing and Communication

- Data cutoff expected late summer
- Results available after iDMC processing and review
- **Expected timing: Q4'26**
- Vicore will remain blinded; no effect size or trends will be disclosed to preserve trial integrity

Phase 2b ASPIRE trial: designed to position buloxibutid as a differentiated and highly competitive therapy for IPF in a multi-billion-dollar market



Defining Success in IPF

01 Efficacy

Clinically meaningful impact on FVC versus placebo

02 Tolerability

Safety and tolerability profile supportive of chronic use, adherence, and quality of life

03 Combinability

Mechanistic synergy and tolerability profile suitable for combination use

Buloxibutid combines the potential for disease-modifying efficacy with a favorable tolerability profile, positioning it to fundamentally reshape the IPF treatment paradigm

Graphic is not meant to represent head-to-head study. Cross-trial comparisons are challenging due to differences in trial design, patient selection, and analysis method.

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.

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 up to 10 years in the EU and Japan.
- Vicore has dosage form and method-of-use IP granted in the US and EU covering buloxibutid, with expiry in 2042 before considering 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

- 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 alone 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

HANS JEPPSSON, PhD
CHIEF FINANCIAL OFFICER
Cross-disciplinary background in finance and medicine. Former Danske Bank: Equity analyst.

Danske Bank
AstraZeneca

BERNT VAN DEN BLINK, MD, PhD
CHIEF MEDICAL OFFICER
Board-certified pulmonologist with 25+ years of experience in IPF and ILD drug development. Former senior clinical leader at Kinevant, Promedior, and Galapagos.

Erasmus MC
Promedior
Galápagos

JOHAN RAUD, MD, PhD
CHIEF SCIENTIFIC OFFICER
Ex AstraZeneca: Director of inflammation research. 25 years of experience in drug development.

AstraZeneca

MIKAEL NYGÅRD, PhD
CHIEF OPERATING OFFICER
Experienced healthcare Business Development executive, has led M&A and Corporate Development functions.

Humana
BCG BOSTON CONSULTING GROUP

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

Pfizer
ZIARCO
UNIVERSITY OF OXFORD

JIMMIE HOFMAN
CHIEF BUSINESS OFFICER
Business Development executive with extensive deal-making experience.

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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.

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