VICORE PHARMA

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

September 2025

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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

$324m
market cap

As of September 29, 2025

$100m
financial position

As of June 30, 2025

Shareholders

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

Pipeline

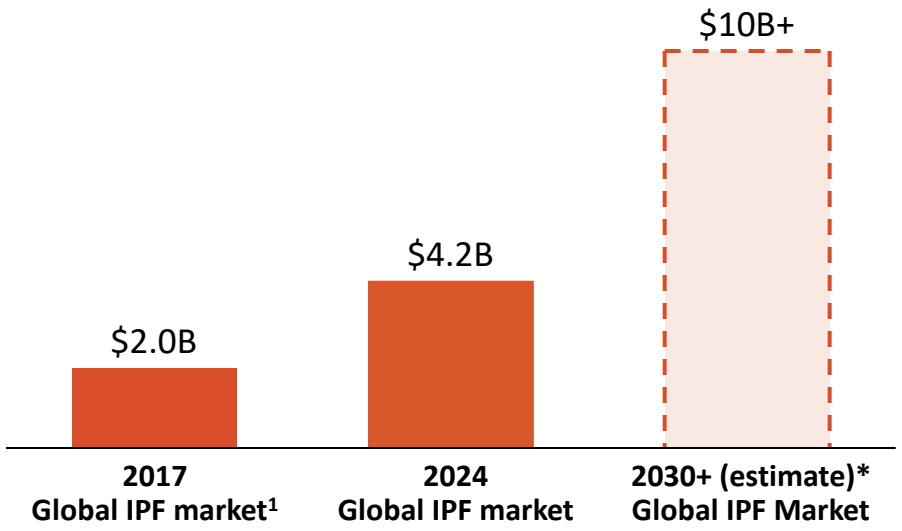
Vicore's lead program, buloxibutid, is a first-in-class oral small molecule AT2 receptor agonist, which has received Orphan Drug and Fast Track designation from FDA and is currently being investigated in a global 52-week Phase 2b trial in IPF, ASPIRE.

Compound	Indication	Preclinical	Phase 1	Phase 2	Phase 3	Comments	Rights
Buloxibutid	**IPF**					Phase 2b ongoing (NCT06588686) Targeting full enrollment by H1 2026	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: A large and growing commercial opportunity with high unmet need

Large commercial market despite SoC shortcomings

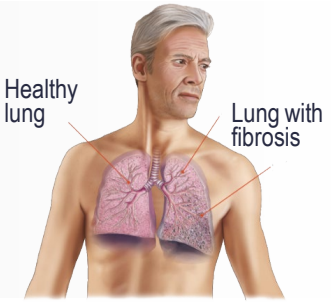


- Growth driven by increased diagnosis and treatment rate
- Limitations of current SoC – moderate impact on disease progression, but with significant side effects and no improvement in quality of life[1,2]
- Strong clinician and regulator desire for tolerable and combinable therapies

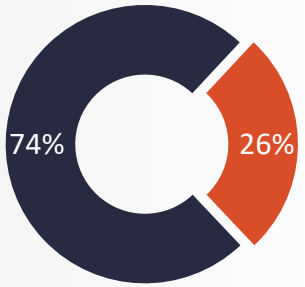
Majority of the market is not adequately addressed

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

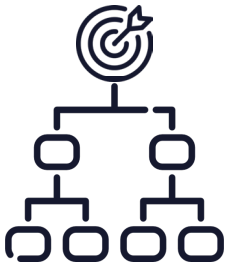
*Estimated sales (Global Data, The Insight Partners)
(1) Evaluate Pharma, Mordor Intelligence. Note: Combined sales of marketed drugs Ofev and Esbriet where Ofev sales since 2019 includes the indication SSc-ILD; (2) Pulmonary Fibrosis Foundation, Resp Res. 21, Article no: 48 (2020); (3) Dempsey et al.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

Buloxibutid is positioned to transform the IPF landscape

- **IPF therapies in Phase 3 are expected to moderately slow the decline of lung function, with many associated with tolerability or administration challenges**
- **Following the Phase 3 programs, buloxibutid is the leading IPF therapy in global development, poised to transform the IPF landscape with higher efficacy and better tolerability**

	Boehringer Ingelheim	United Therapeutics (A PUBLIC BENEFIT CORPORATION)	Bristol Myers Squibb™	vicore pharma
Name & Target	**Nerandomilast** PDE4B antagonist	**Inhaled treprostinil** prostacyclin analog	**Admilparant** LPA1 antagonist	**Buloxibutid** AT2 receptor agonist
Ease of Use & Tolerability	GI-tolerability issues, further exacerbated on top of SoC	48 (12x4) breaths per day, cough, throat irritation, headache, nausea and flushing	Favorable overall tolerability profile; transient reduction in blood pressure	**Favorable overall tolerability profile**
Efficacy	Incremental improvement over SoC, only slows lung function decline	Superiority over placebo for the change in FVC at 52 weeks by 95.6mL	Ph2 data reflects incremental impact, only slows lung function decline	**Potential to stabilize and improve lung function**
	Phase 3 (52-week)			***Phase 2b (52-week)***

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 mechanism drives alveolar repair, resolves fibrosis, and promotes vascular function

Exceptional clinical data in the Phase 2a AIR trial

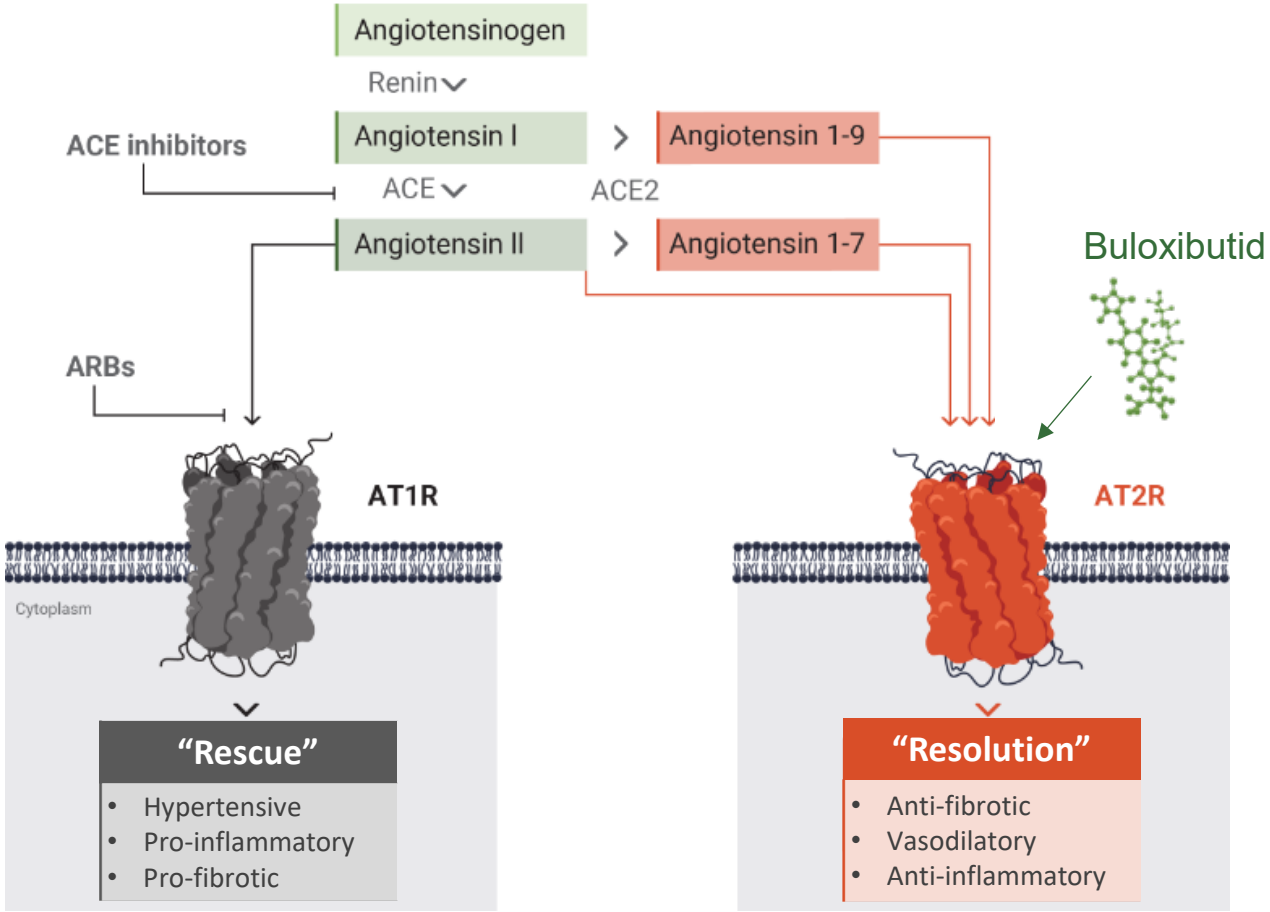
- Mean FVC change from baseline of +216 ml at 36 weeks
- All subgroups above baseline
- Excellent gastrointestinal tolerability and no treatment-related SAEs
- Biomarker data highly supportive of suggested MoA

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

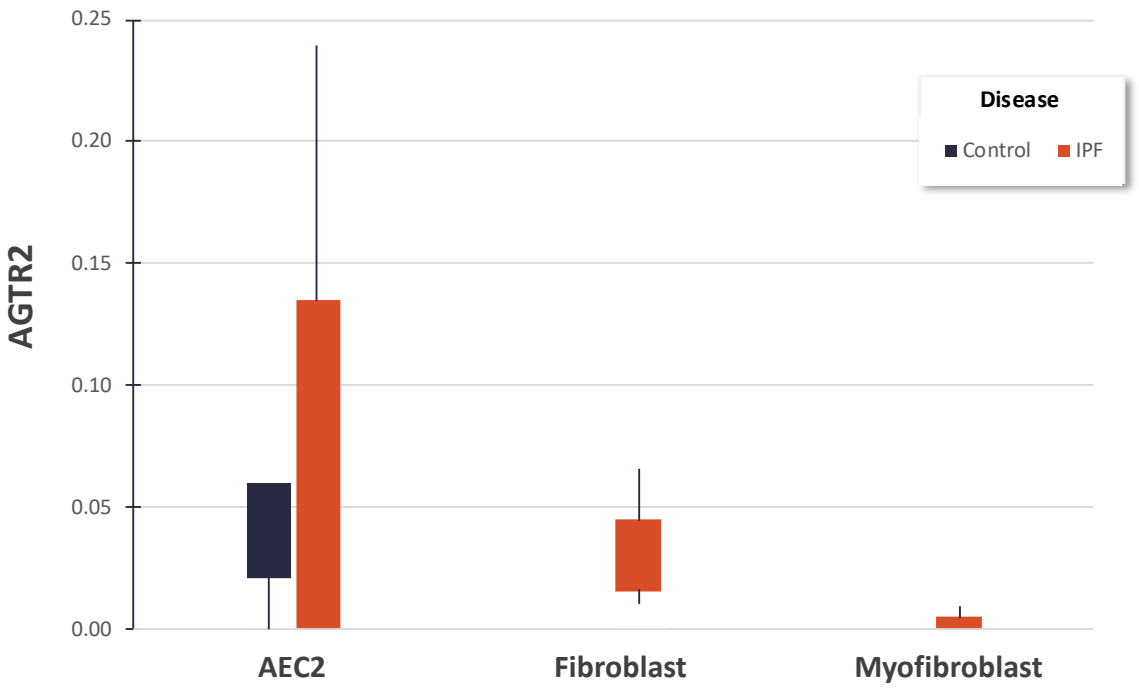
AT2R agonism is an upstream intervention driving tissue repair



- 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

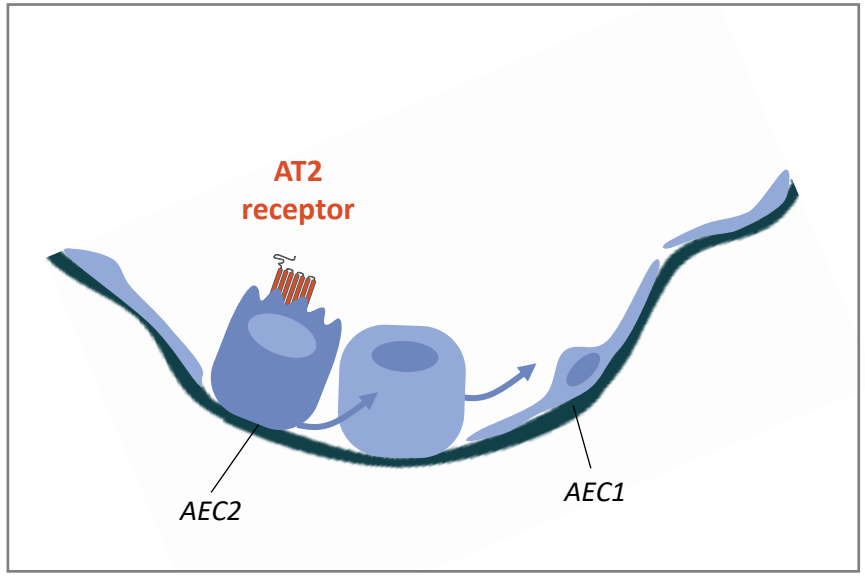
AT2R is highly expressed in human IPF lungs and on precursor AEC2s

AT2R Expression is Elevated in IPF Lung



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

AT2R is Highly Expressed on AEC2s

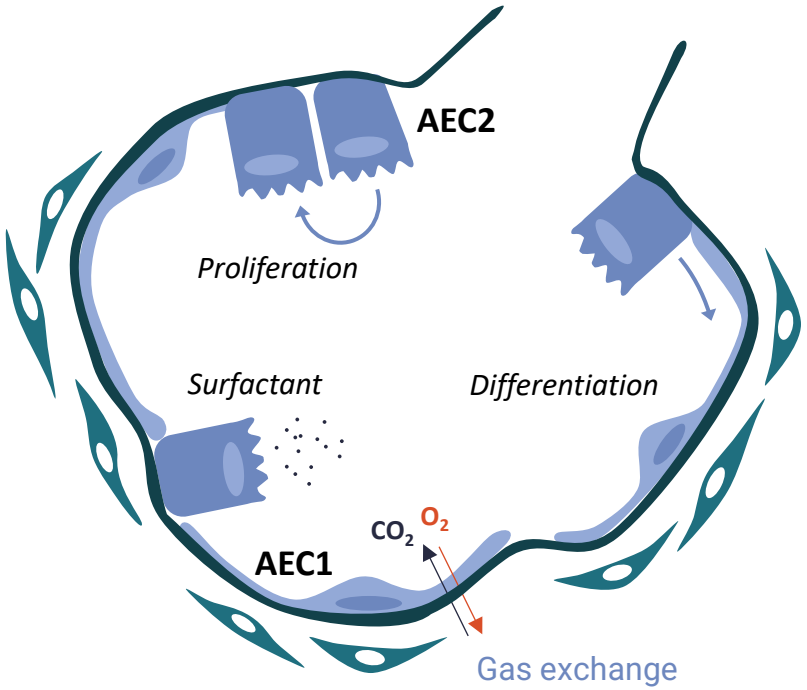


Single cell analysis shows high AT2R expression on AEC2 in the lung, the progenitor cell that differentiates into AEC1 gas exchange cells

Source: IPF Cell Atlas

Alveolar epithelial cells are critical for healthy lung function

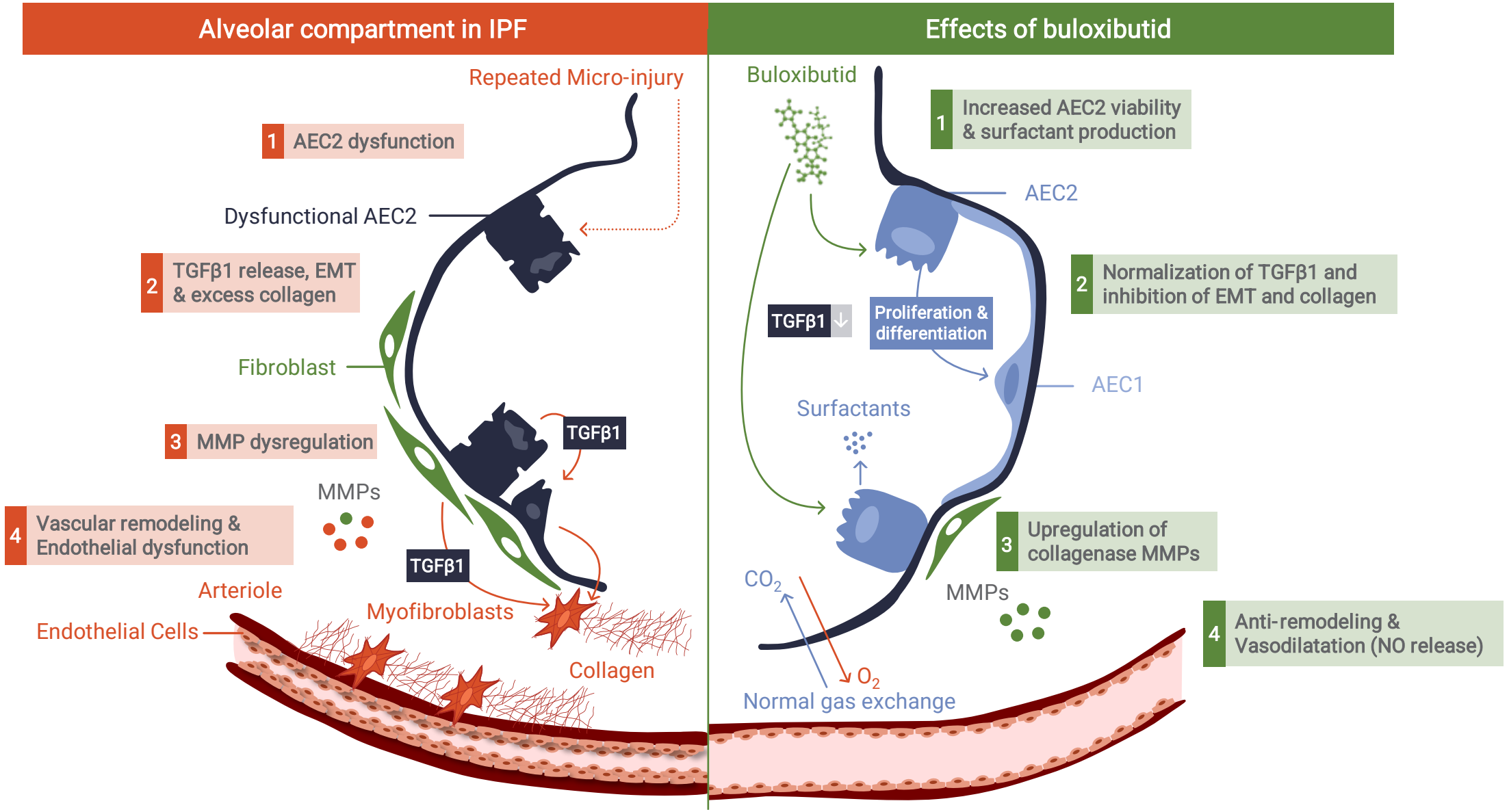
Healthy alveolus



AEC – Alveolar Epithelial Cell

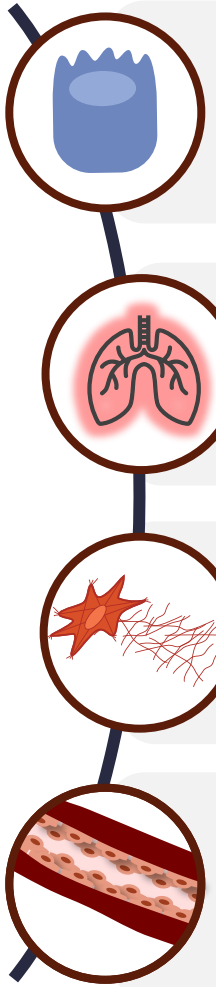
- The alveolar epithelium is exposed to damaging irritants in inhaled air
- AEC1 is the predominant alveolar cell type and is responsible for gas exchange
- AEC2 is a progenitor cell that is critical for alveolar integrity and function:
  - Proliferates to form new AEC2
  - Differentiates to AEC1 that 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

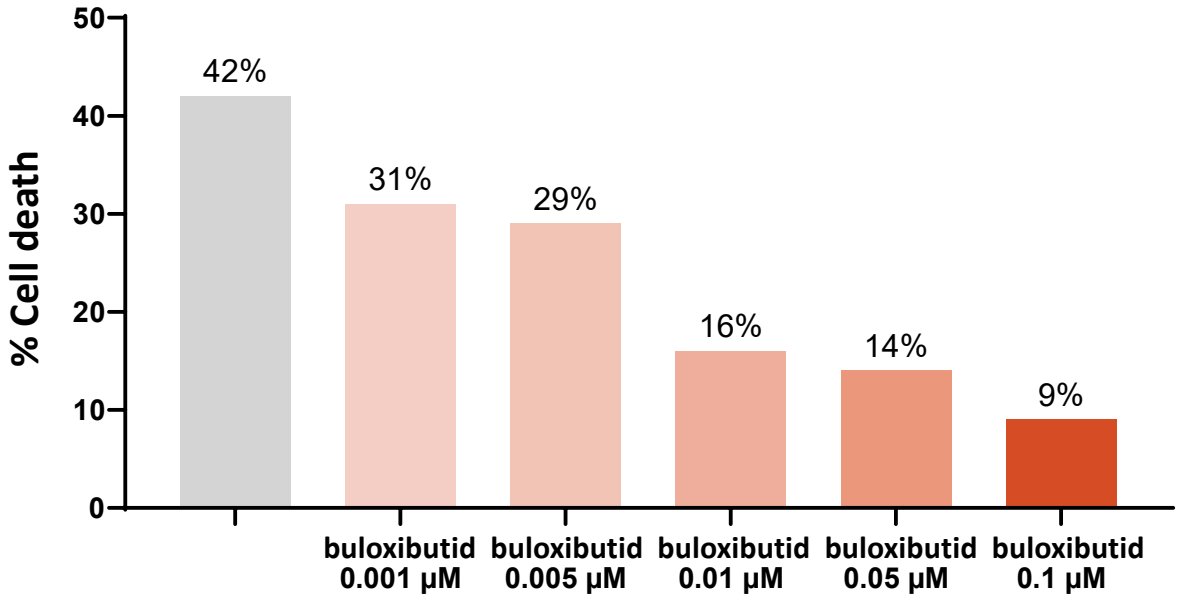
Anti-Fibrotic
Buloxibutid restores dysfunctional AEC2 and surfactant production, normalizes TGFβ1 levels, inhibits EMT and collagen deposition, as well as breaks down existing collagen build up

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]

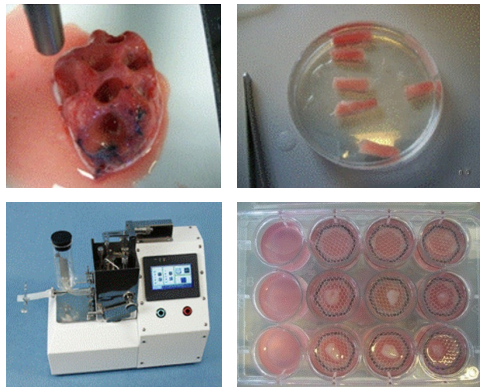
- Human precision cut IPF lung slices ± 1 µM buloxibutid
- One patient, 5 pooled lung slices

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

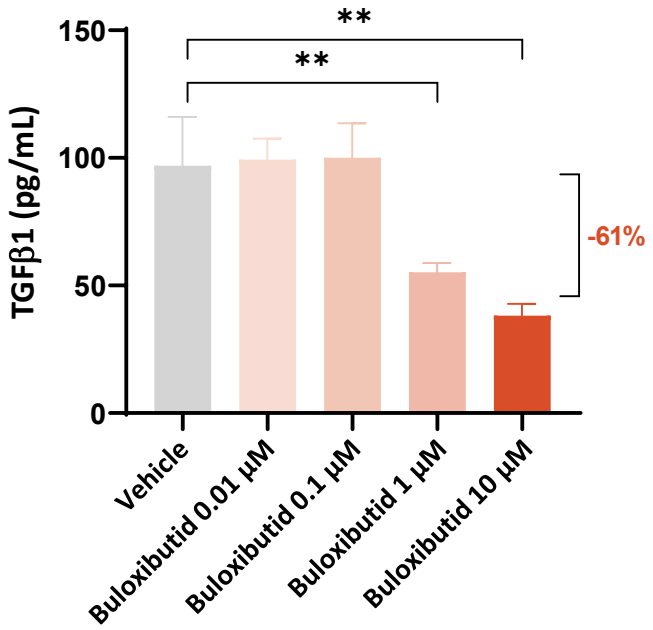
Buloxibutid reduces TGFβ1 and collagen in human IPF lung slices

Human precision cut lung slices (PCLuS)

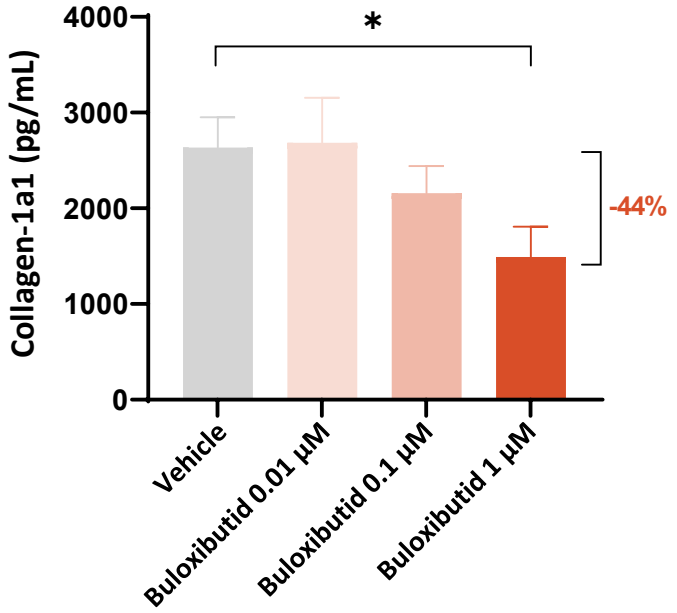


- Lung tissue collected from IPF patients undergoing transplant
- Intrinsic fibrosis, no stimuli added

TGFβ1 protein levels in PCLuS



Collagen protein levels in PCLuS

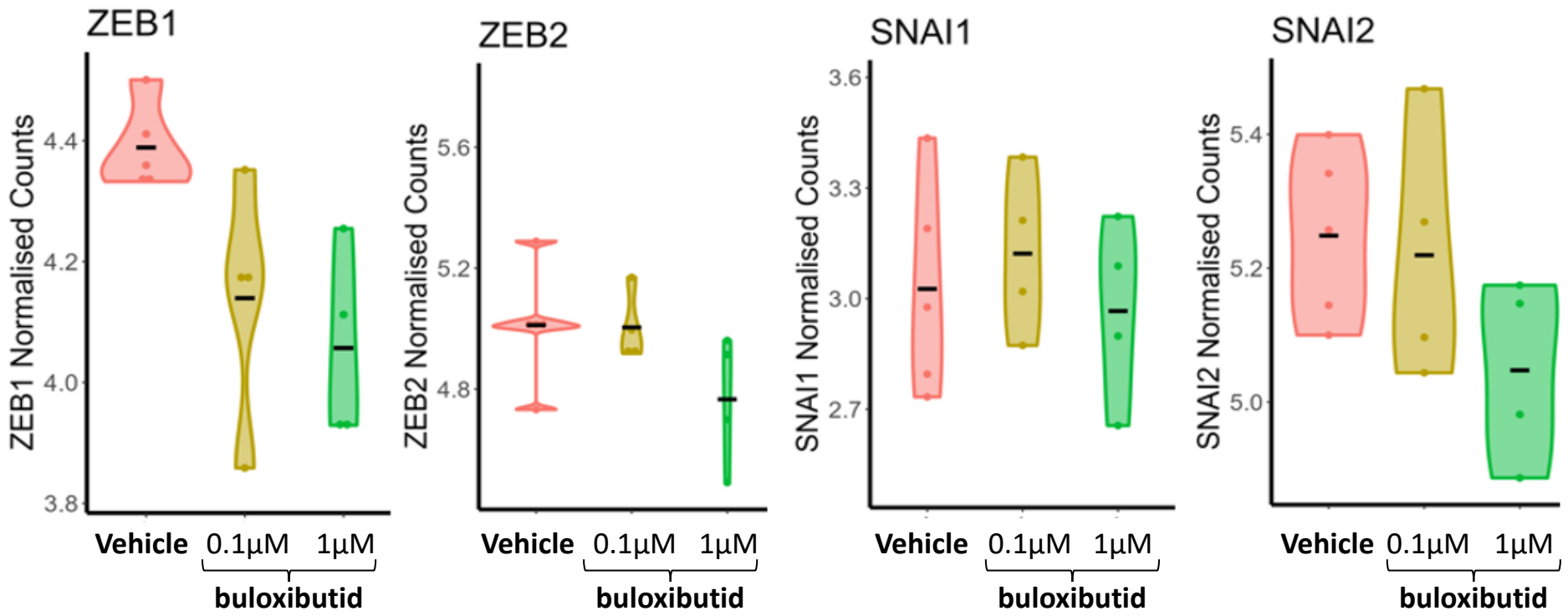


Dose-dependent reduction of TGFβ1 and Collagen-1a1 protein

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

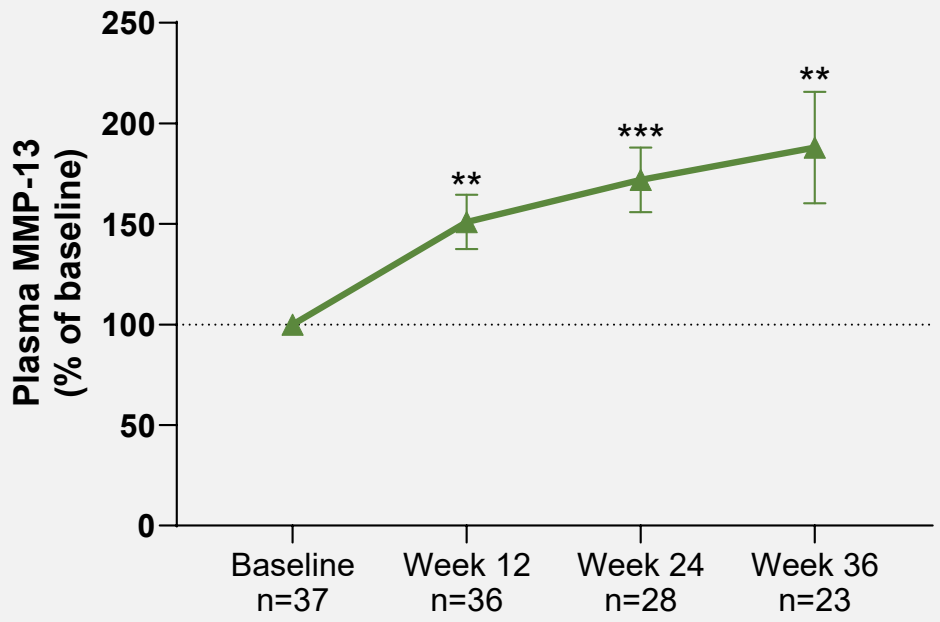
Source: Conforti et al. 2023, manuscript (Vicore collaboration with U Southampton)

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



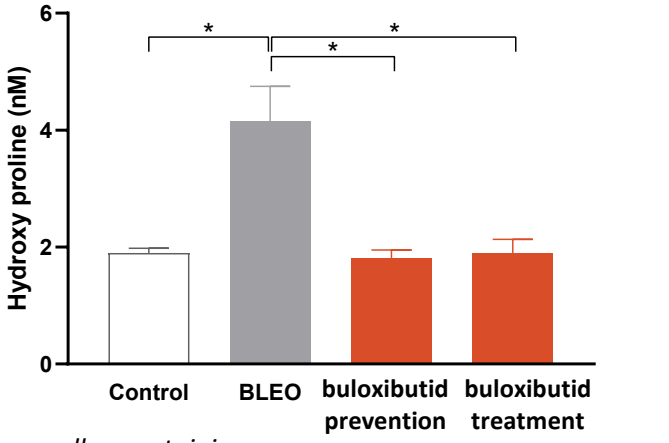
Buloxibutid significantly increased plasma levels of the fibrolytic collagenase MMP-13, indicating that buloxibutid has the potential to degrade fibrosis

ECM = Extracellular Matrix; MMP = Matrix Metalloproteases

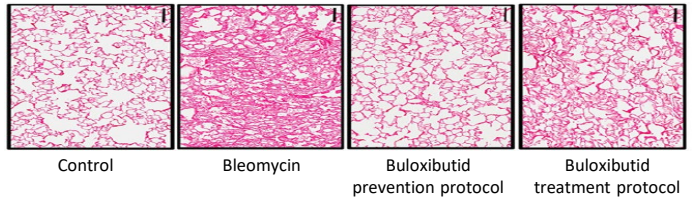
(1) Cabrera et al., 2019; (2) Nkyimbeng et al., 2013

Strong *in vivo* evidence for buloxibutid in pulmonary fibrosis

Bleomycin



Lung collagen staining

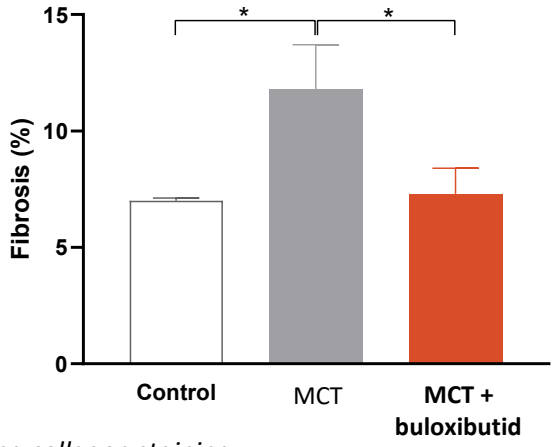


- Buloxibutid 0.03 mg/kg/day i.p. once daily, 14 days (Treatment). N=5 control and 7-8 in treatment groups

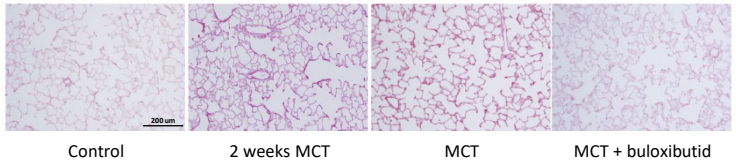
Adapted from (1)

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

Monocrotaline



Lung collagen staining

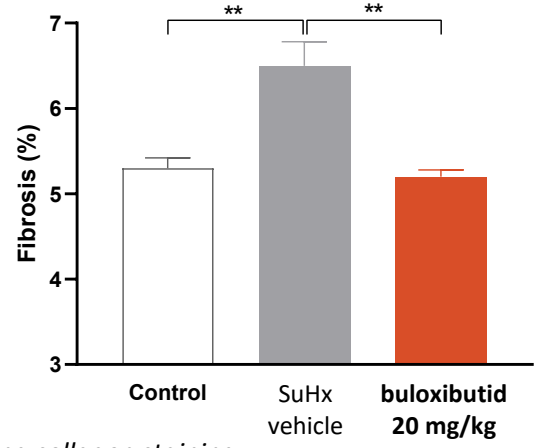


- Buloxibutid 0.03 mg/kg/day i.p. once daily for 2 weeks
- N=14 per treatment group

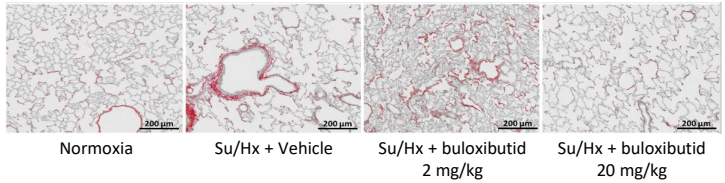
Adapted from (2)

- **Reversal of 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)

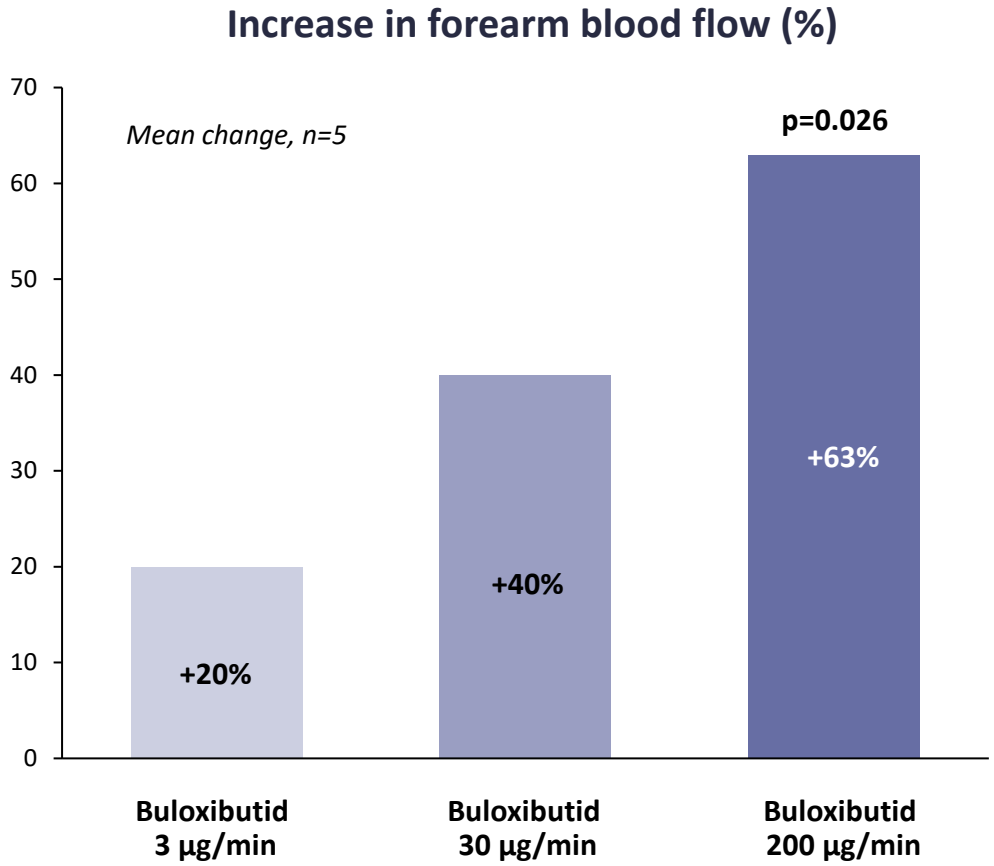
- **Reversal of fibrosis**
- **Reversal of vascular remodeling**
- **Reduced RVSP and right ventricular hypertrophy**

(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'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

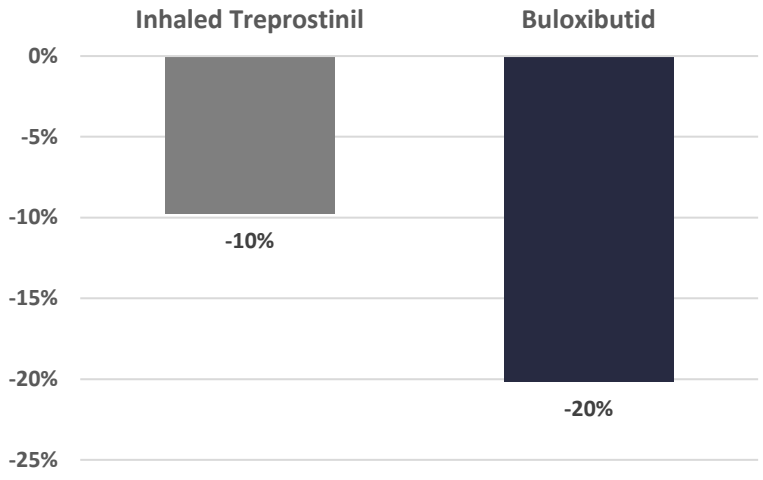
Buloxibutid shows greater reduction in key hemodynamic and vascular remodeling parameters compared to inhaled treprostinil in preclinical Sugen-Hypoxia rat model

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

Hemodynamic Parameters

RVSP

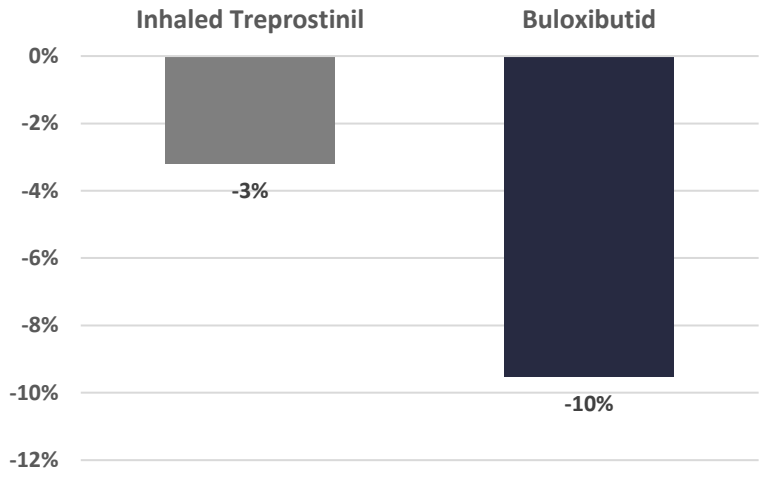
Reduction in RVSP from SuHx control



Right Ventricular Remodeling

Fulton's Index

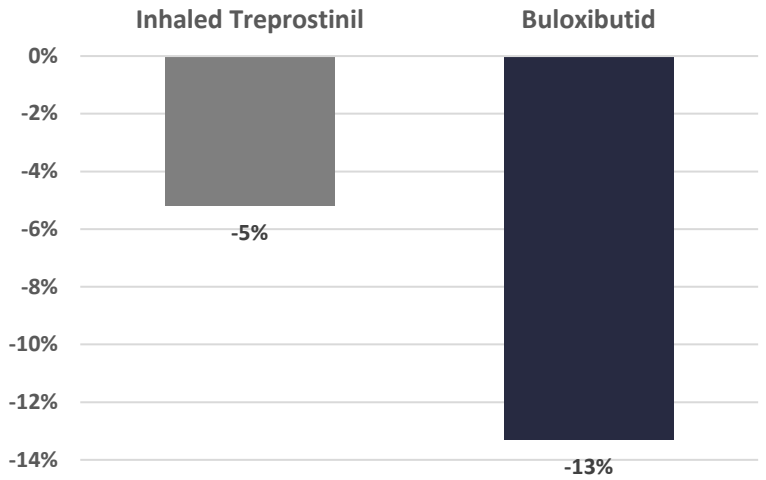
Reduction vs. SuHx control



Pulmonary Vascular Remodeling

Wall Thickness

Reduction vs. SuHx control



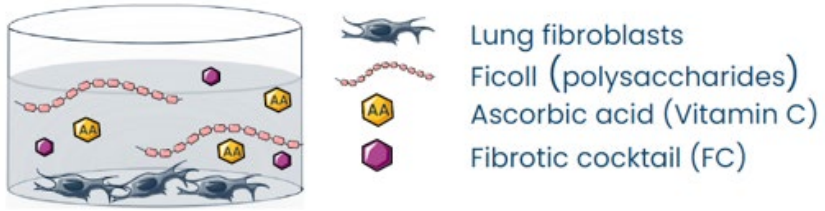
***Inhaled treprostinil**: adapted from Corboz, et al., J. Pharmacol. Exp. Ther. 2022 – Dose: 65 µg/kg*
***Buloxibutid**: adapted from Tornling, et al., Int. J. Mol. Sci. 2023 – Dose: average result of 2µM and 20µM dose*

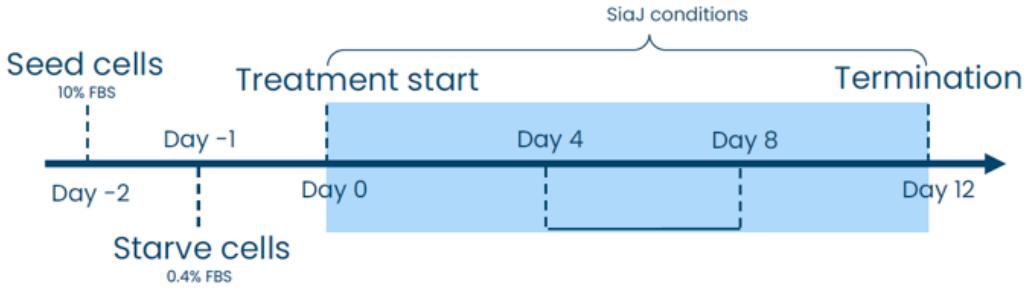
Clinically relevant doses of buloxibutid shows greater reduction compared to the Sugen-Hypoxia control than clinically relevant dose of inhaled treprostinil across key readouts, including RVSP, mPAP (data not shown), Fulton's index, wall thickness and muscularization (data not shown)

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

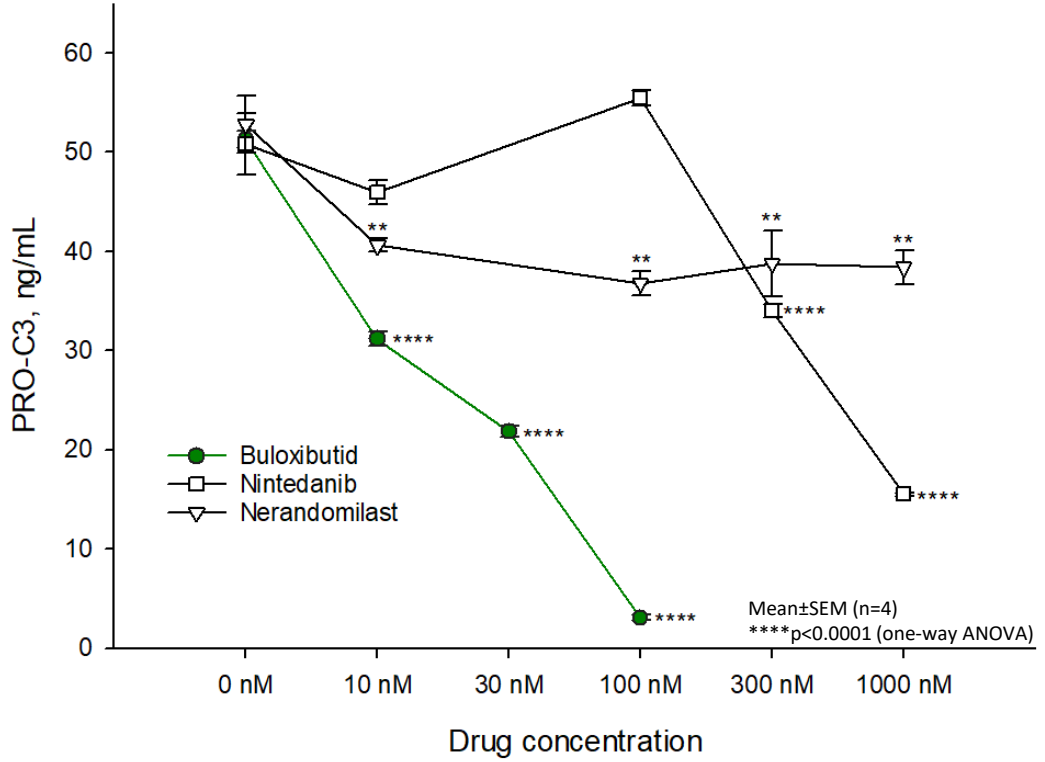
Buloxibutid potently inhibits fibrosis in a human lung fibroblast assay

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

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**

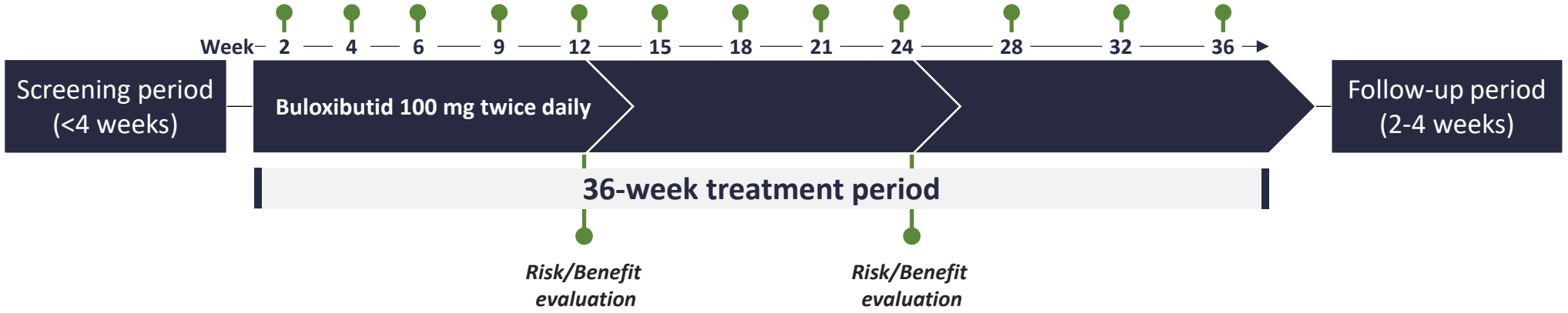
No significant safety risks have been identified for buloxibutid

- 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)	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

	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

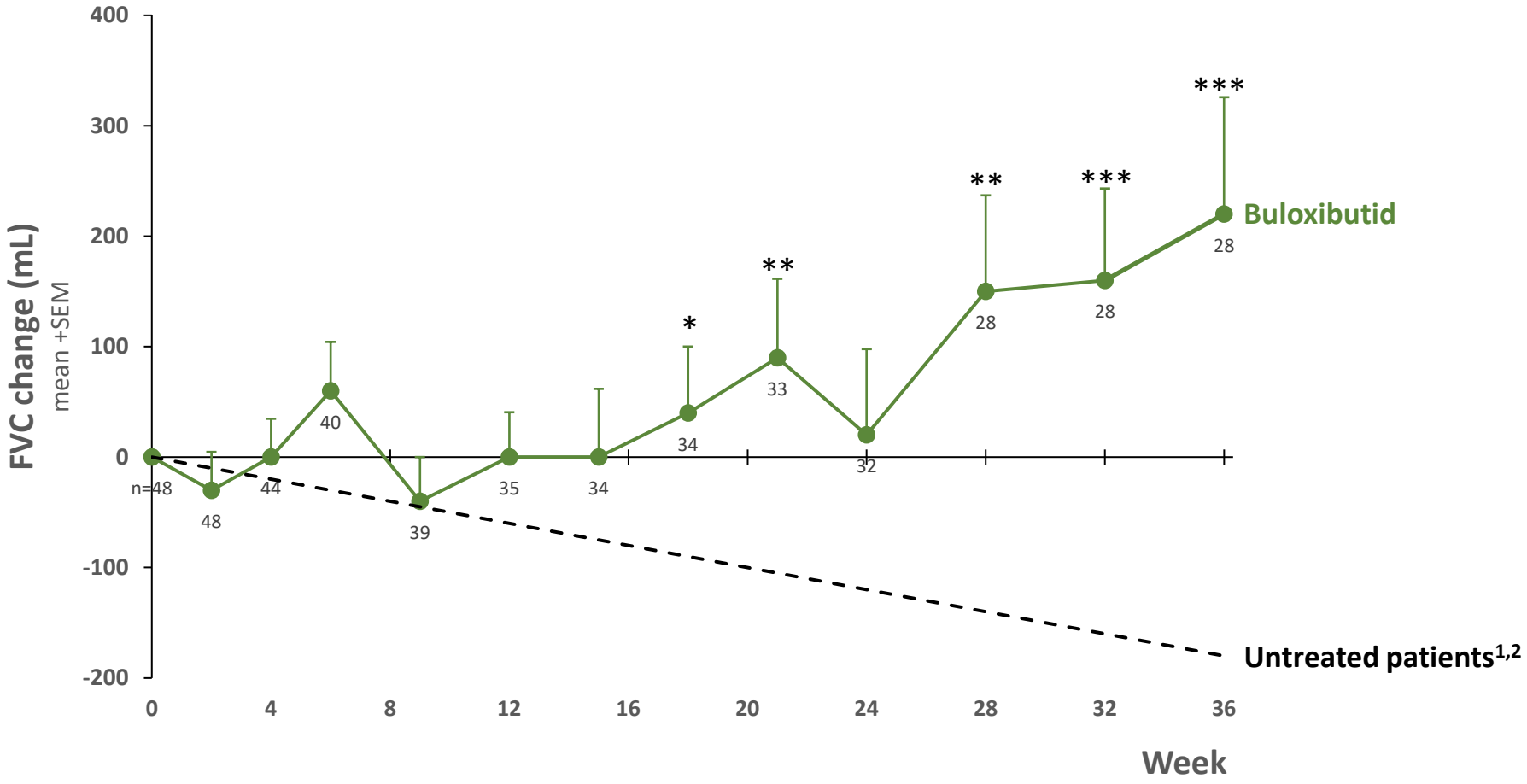
Low rate of exacerbations and cough worsening

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.

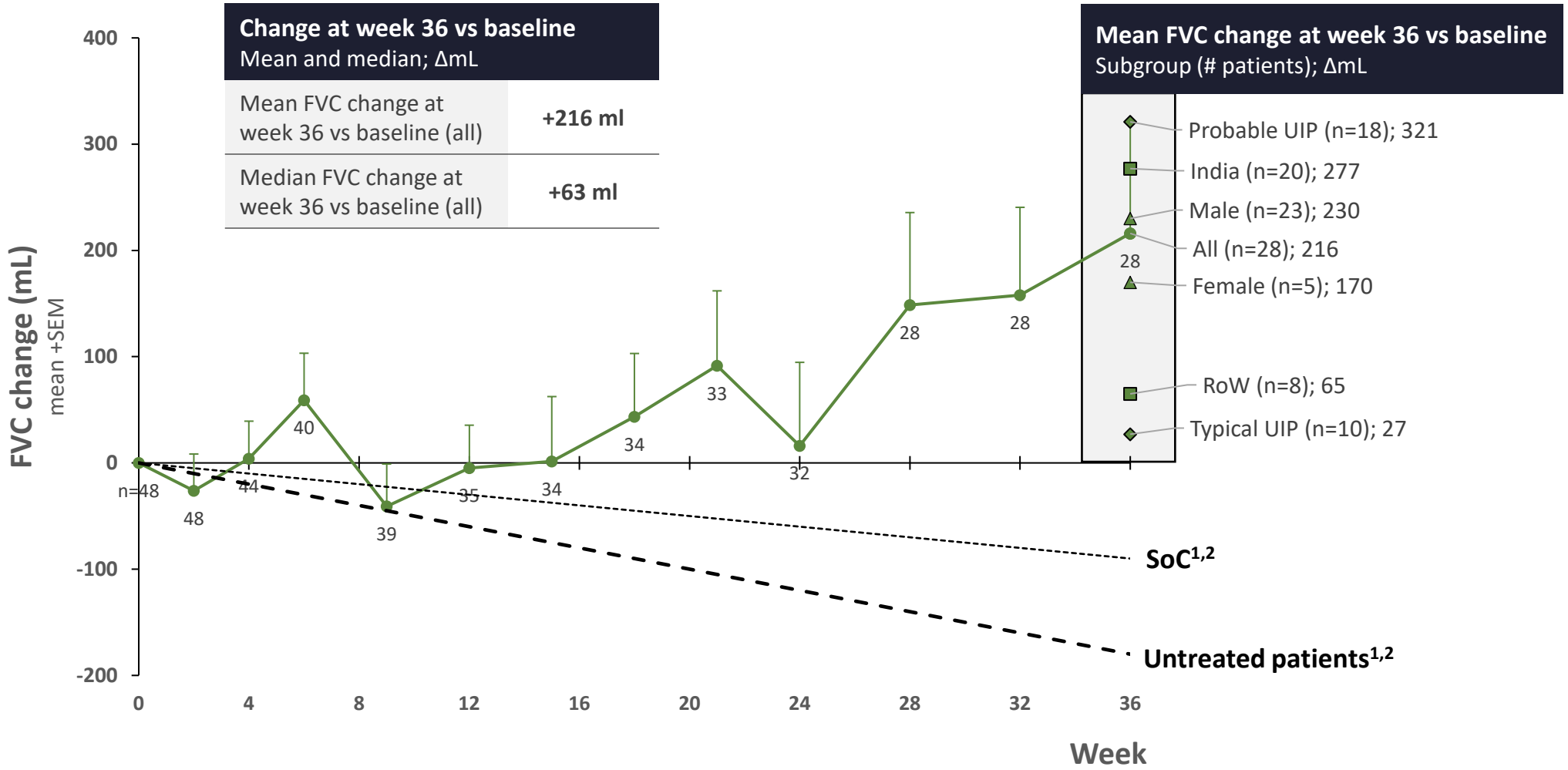
Buloxibutid stabilizes and improves lung function over the 36-week AIR study



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

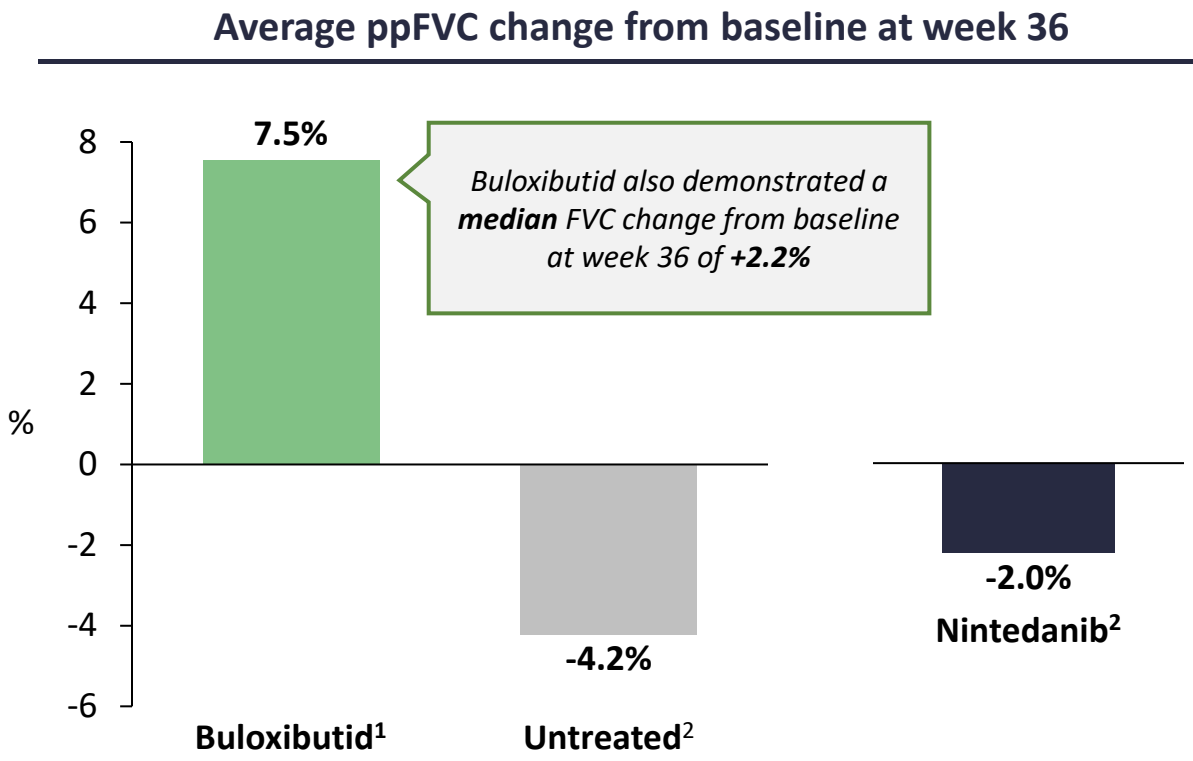
All subgroups show FVC stabilization and improvement over baseline at 36 weeks



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

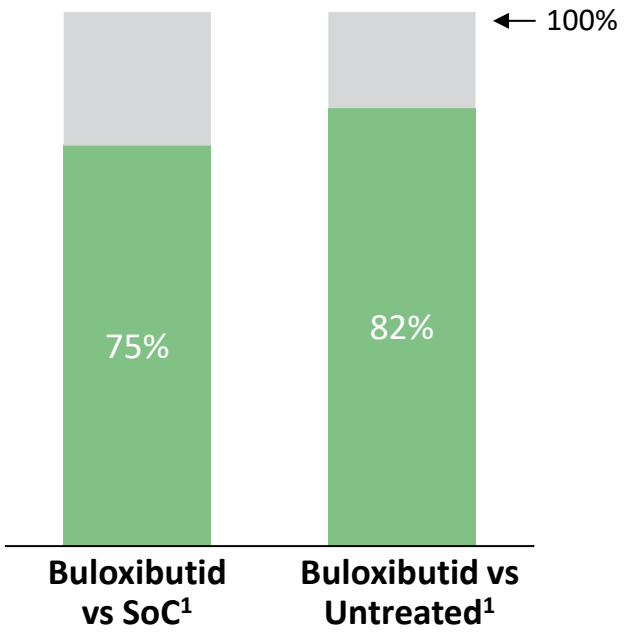


Average increase of 7.5% precent predicted FVC at 36 weeks from baseline

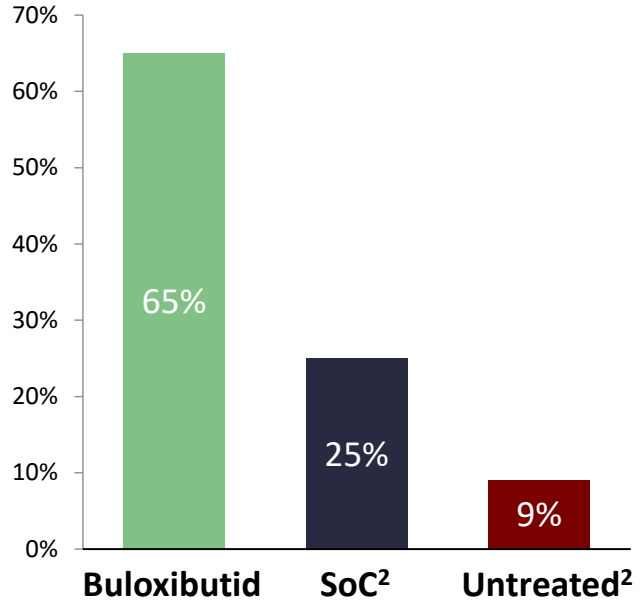
(1) Average ppFVC at 36 weeks (n=28); (2) 36 week estimated ppFVC for nintedanib and placebo, INPULSIS trial (Richeldi et al. N Engl J Med 2014; 370:2071-82)

Buloxibutid 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%.

Development of a Synthetic Control Arm analysis to contextualize buloxibutid's effect in the Phase 2a AIR study

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

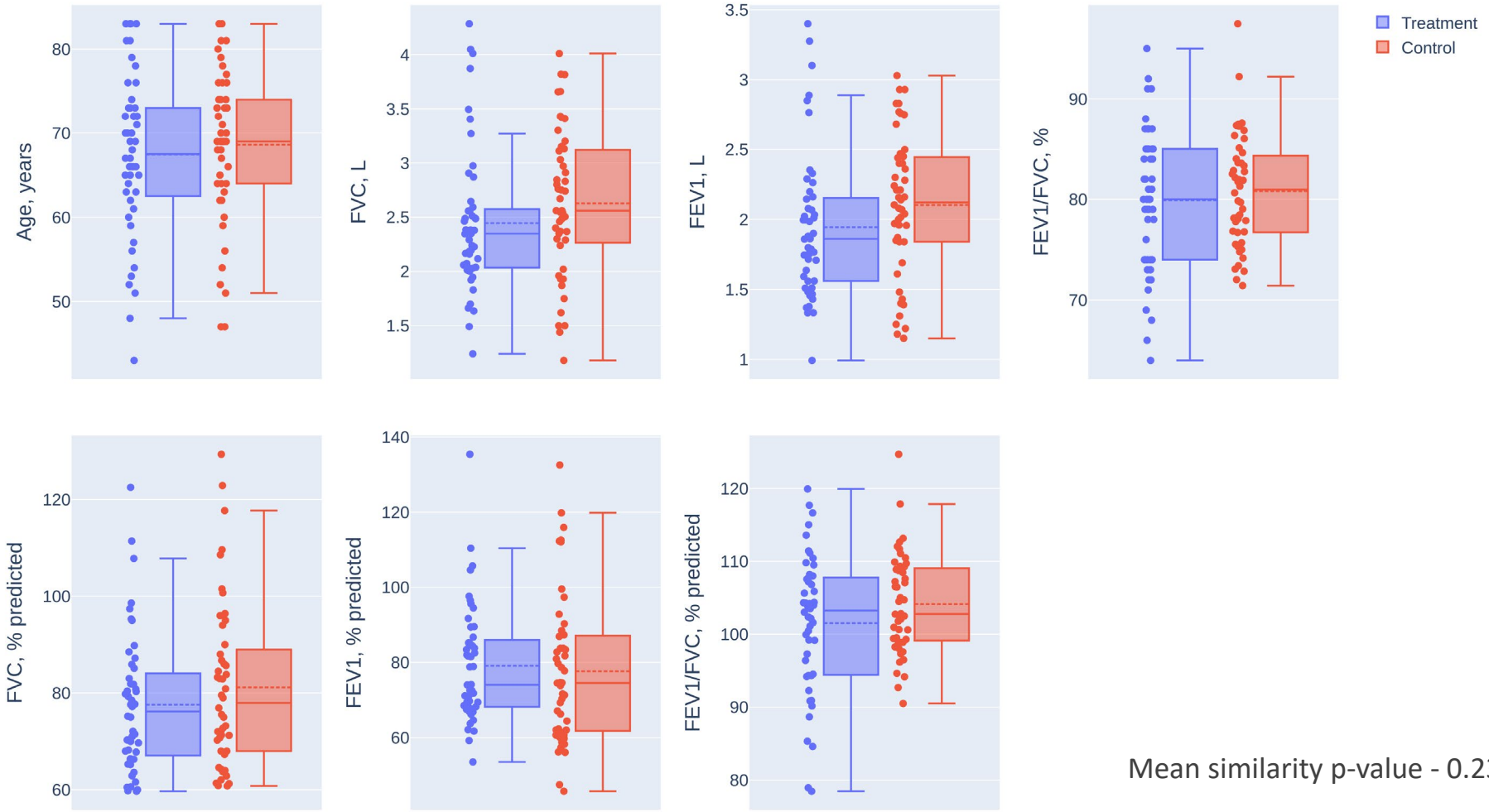
- 20,000 randomly sampled groups of 48 patients generated from filtered control data
- 1:1 treatment-control ratio
- Matching on 8 clinical variables at baseline
- Univariate and multivariate tests of similarity with control
- 408 randomly sampled groups accepted as control arms based on the baseline 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

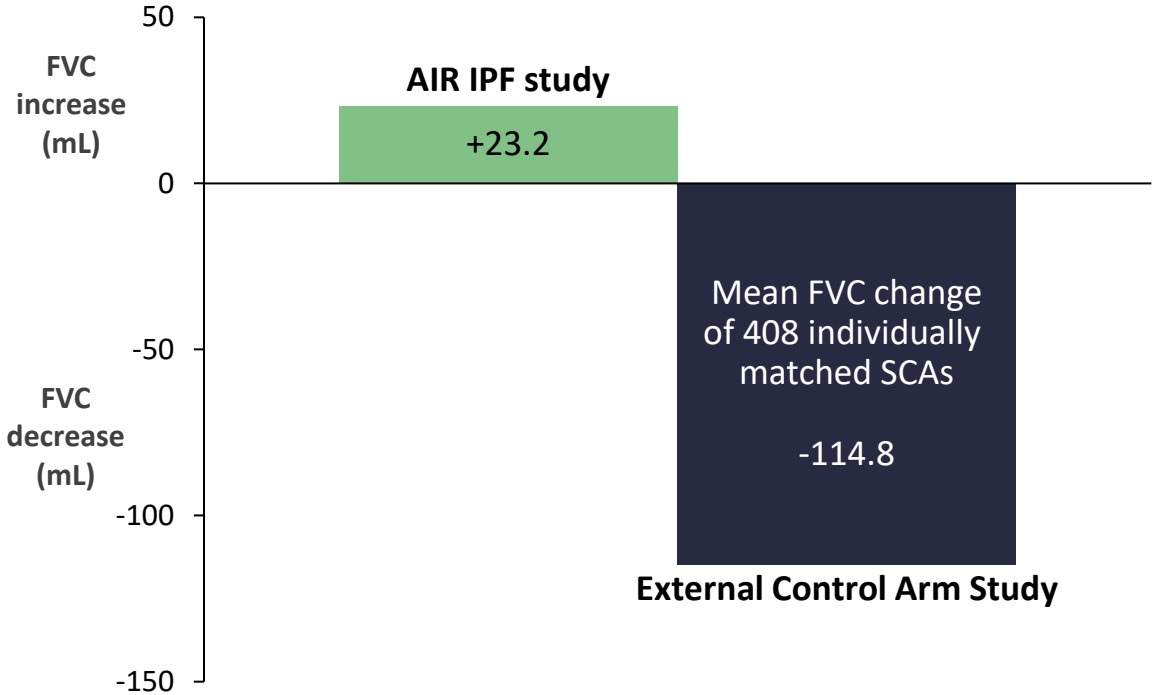


Mean similarity p-value - 0.23 (SD=0.05)

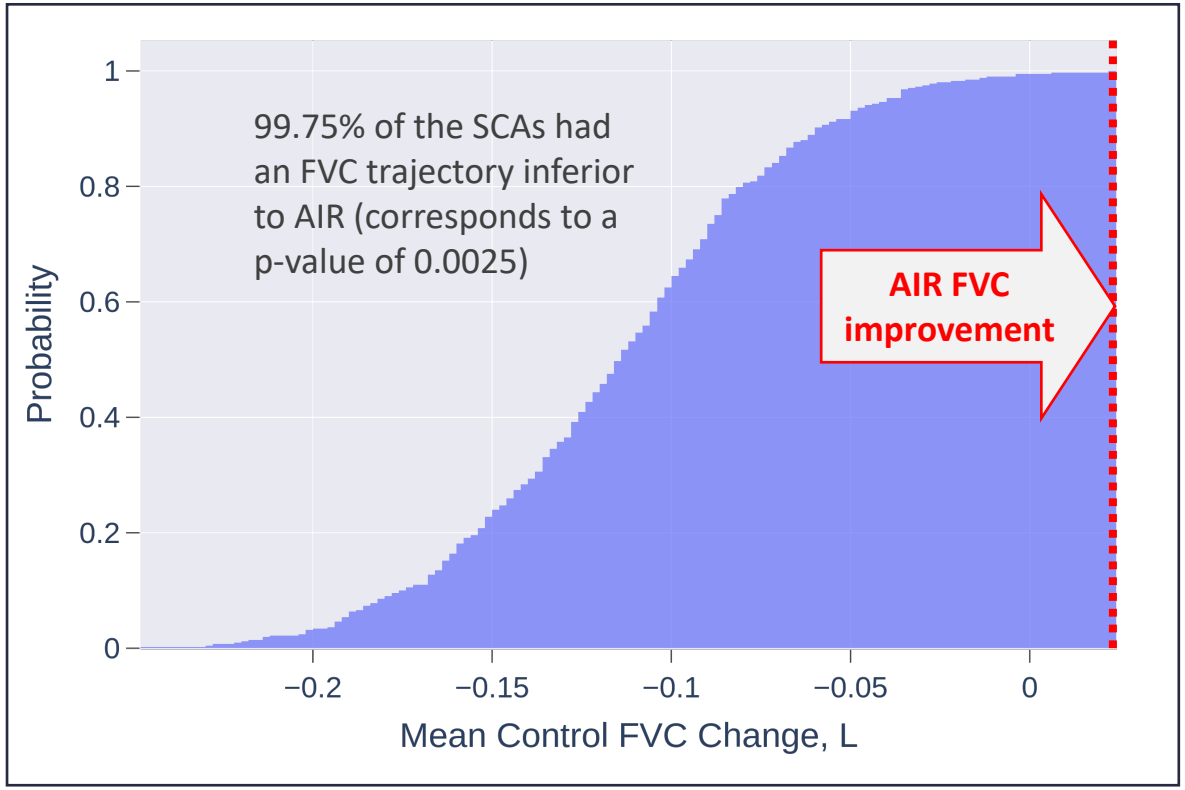
Note: Qureight analysis

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 (imputed data)



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

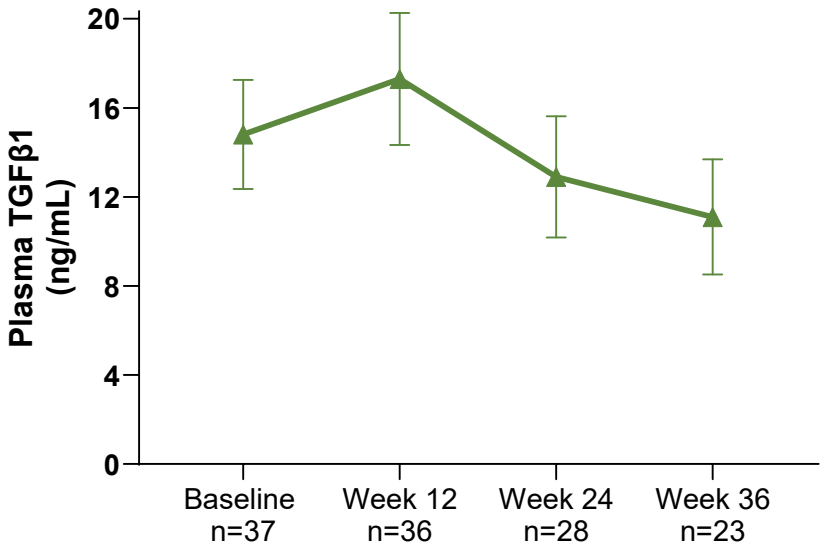


The Monte Carlo approach demonstrates that in patients without significant differences in core baseline parameters, buloxibutid showed statistically significant treatment effect compared to control FVC distribution

Note: Qureight analysis

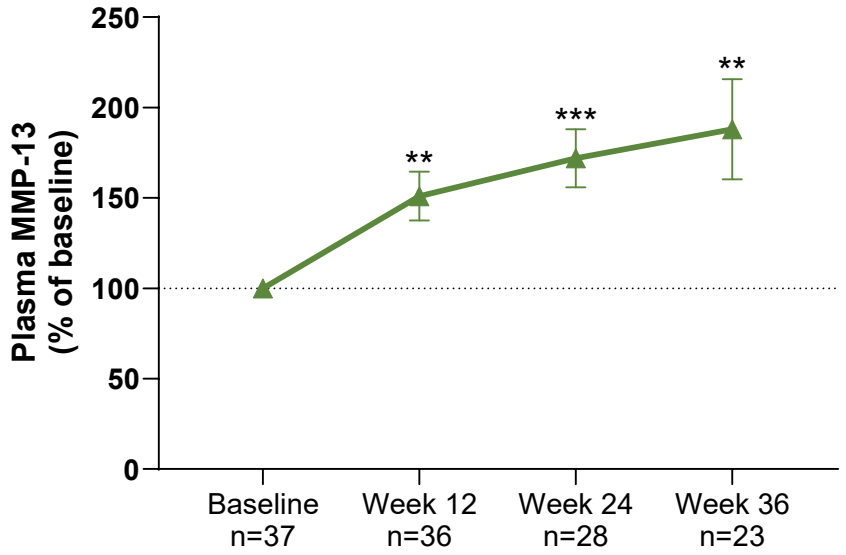
Buloxibutid increases collagenase MMP-13 drives a trend of decreased TGFβ1

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

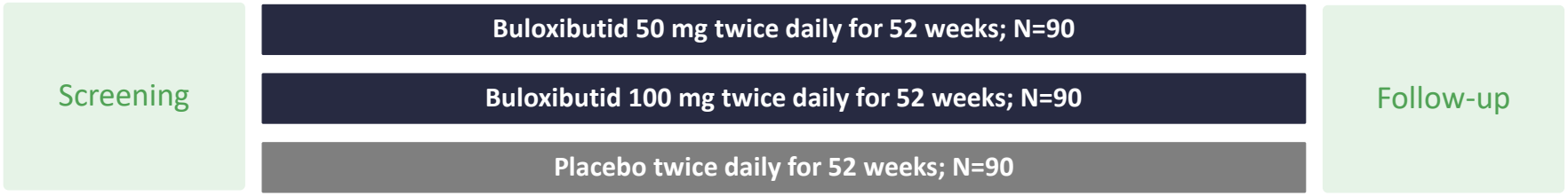
Phase 2b ASPIRE trial design

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

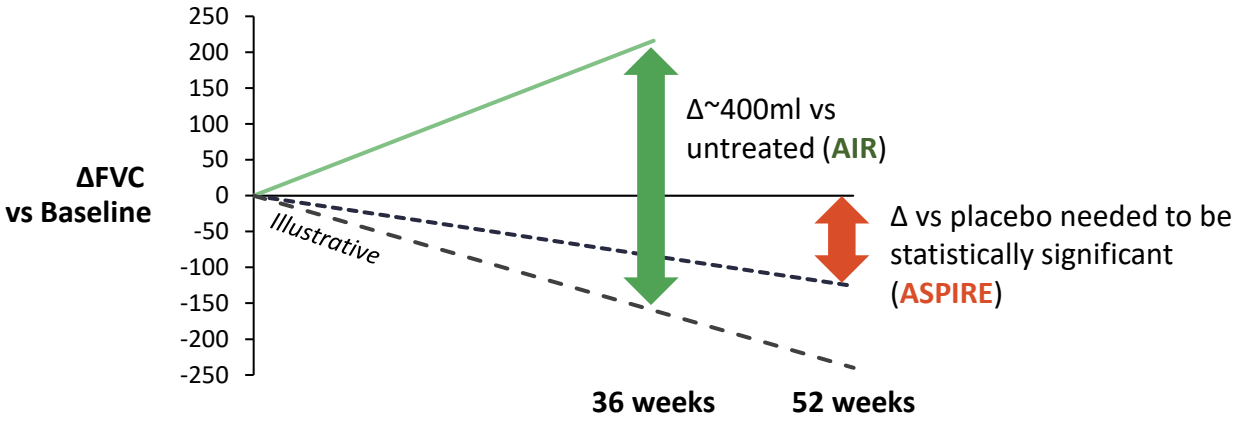
Study Design



ASPIRE is a global trial powered to demonstrate FVC stabilization

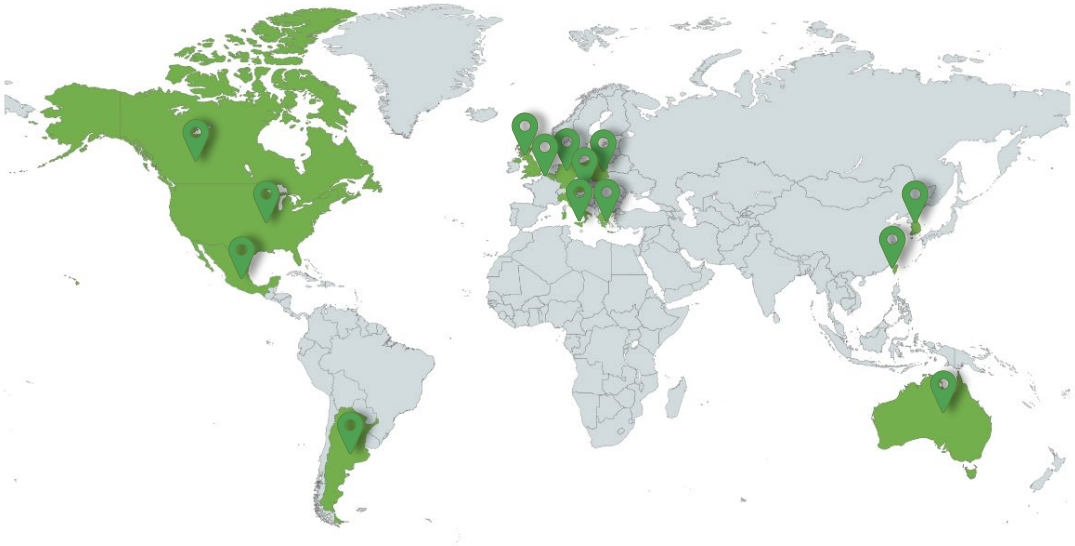
Large, conservatively powered study

- Powered to detect FVC stabilization at 52 weeks vs. conservative placebo arm assumption
- Transformative relative to standard care



Global footprint with a broad range of countries

- 101 sites across 14 countries



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. Ex Danske Bank: Equity analyst.

Danske Bank · 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

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

VP AND HEAD OF CMC

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

VP BUSINESS DEVELOPMENT

Business Development executive with extensive deal-making experience.

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vcore pharma

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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