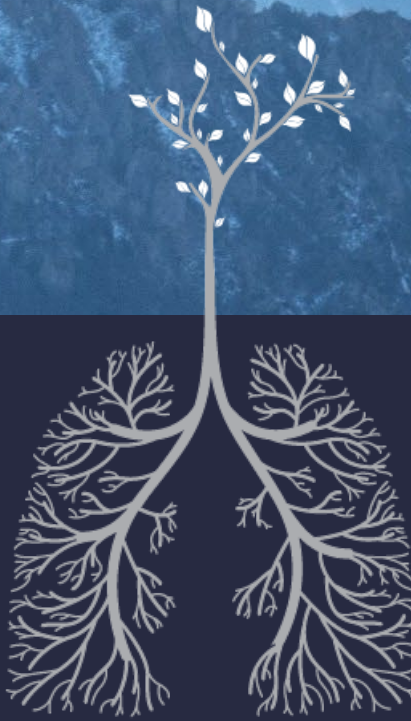
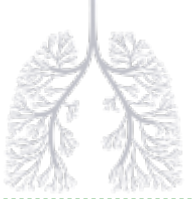


A stylized white graphic of a tree with a thin trunk and many small, leafless branches, positioned on the left side of the slide.

VICORE PHARMA

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

September 2026



Forward-Looking Statements

This presentation contains "forward-looking statements" within the meaning of the Private Securities Litigation Reform Act of 1995, as amended, that are based on the beliefs and assumptions and on information currently available to management of Vicore Pharma Holding AB (the "Company"). All statements other than statements of historical fact contained in this presentation are forward-looking statements. Forward-looking statements include, but are not limited to, statements concerning: the Company's planned and ongoing preclinical and clinical studies for buloxibutid and other product candidates, including the Phase 2b ASPIRE trial, and the potential advantages of those product candidates, including tolerability and efficacy; the initiation, enrollment, timing, progress, and release of data from, and results of those, clinical studies, including the expected timing of topline data from the ASPIRE trial and the pre-planned futility analysis; the Company's goals with respect to the development, regulatory pathway, and potential use, if approved, of its product candidates; the utility of prior preclinical, clinical, and synthetic control arm data in determining future clinical results; the timing or likelihood of regulatory filings and approvals; the Company's intellectual property position; the ability to commercialize buloxibutid; market opportunity estimates and competitive positioning; the Company's partnership with Nippon Shinyaku; and the sufficiency of the Company's cash position and financial resources to fund planned operations. In some cases, you can identify forward-looking statements by terminology such as "anticipates," "assumes," "believes," "can," "could," "estimates," "expects," "forecasts," "intends," "may," "might," "plans," "potential," "projects," "should," "targets," "will," "would," or in each case their negative, or other variations or comparable terminology.

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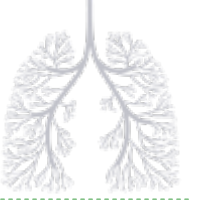
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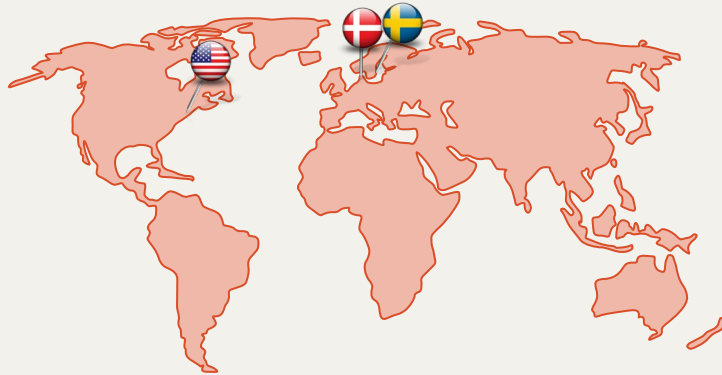
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Company overview



Vicore Vision

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



Locations

Stockholm, Sweden
Cambridge, Massachusetts, USA
Copenhagen, Denmark

Financials

Dual listing on Nasdaq Stockholm (VICO) and Nasdaq US (VCRE), and funded well past Phase 2b data

\$320m
market cap

As of September 3, 2026

\$93m
financial position

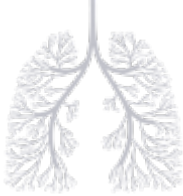
As of June 30, 2026



Shareholders

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

Pipeline

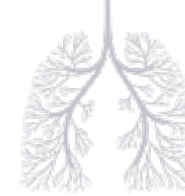


Vicore’s lead program, buloxibutid, is an oral small molecule AT2 receptor agonist, which has received Orphan Drug and Fast Track designation from FDA and is currently being investigated in a global 52-week Phase 2b trial in IPF, ASPIRE

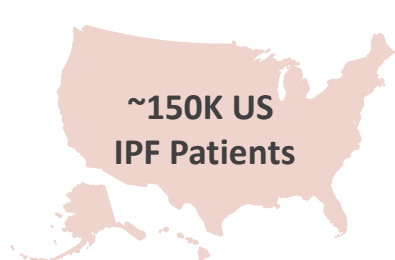
Compound	Indication	Preclinical	Phase 1	Phase 2	Phase 3	Comments	Rights
Buloxibutid	IPF					Phase 2b ongoing (NCT06588686)	Global ex-Japan rights
						Enrollment complete; targeting topline readout in mid-2027	NIPPON SHINYAKU CO., LTD.
New ATRAGs are in early-stage discovery							

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 treatment¹



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

10 months¹

The majority of the IPF market is not adequately addressed today

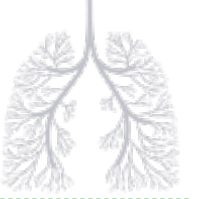
Limitations of available treatment options

Three FDA-approved therapies:



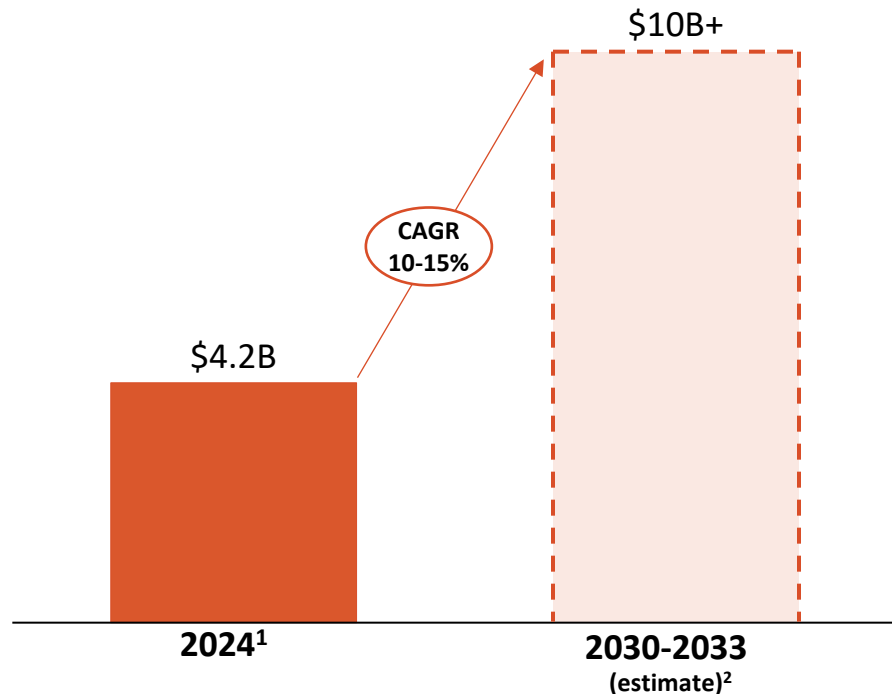
- Existing therapies offer only modest slowing of disease progression and have not demonstrated quality of life benefit
- Significant GI side effects limit uptake, often requiring dose reductions and contributing to high discontinuation rates
- Despite available treatments, the 5-year mortality rate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

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



Global IPF Market Size

Significant market expansion
expected over the next decade

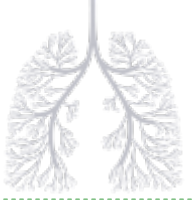


Drivers of Market Growth Today

- **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



Buloxibutid is positioned to potentially 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.

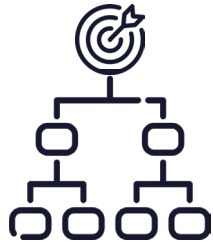
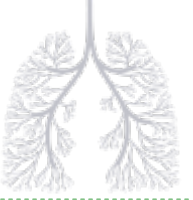
	 Buloxibutid	 Jascayd (nerandomilast)	 Tyvaso (treprostinil)	 Admilparant
MOA	AT2 receptor agonist Novel endogenous upstream and multi-modal mechanism	PDE4B antagonist	prostacyclin analog	LPA1 antagonist
Dosing & Administration	Oral BID Convenient oral dosing	Oral BID	Nebulized formulation, 48 (12x4) breaths per day	Oral BID
Tolerability¹	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
Efficacy²	Mean FVC improvement Observed +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 improved 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



(1) Tolerability based on clinical data generated to date; (2) Efficacy data observed in the Phase 2a AIR trial

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.

Buloxibutid is an AT2 receptor agonist with the potential to offer a differentiated treatment standard in IPF



Upstream MoA with strong preclinical data

- AT2 receptor expressed on alveolar progenitor cell (AEC2)
- Upstream mechanism is believed to drive alveolar repair, resolve fibrosis, and promote vascular function



Encouraging clinical data observed in the Phase 2a AIR trial

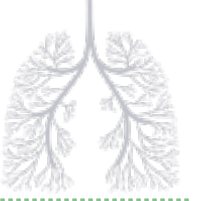
- Observed mean FVC change from baseline of +216 mL at 36 weeks, with benefit observed across all subgroups¹
- Synthetic control arm analysis reinforces the meaningful treatment effect observed with buloxibutid
- Good gastrointestinal tolerability and no treatment-related SAEs
- Biomarker data highly supportive of suggested MoA



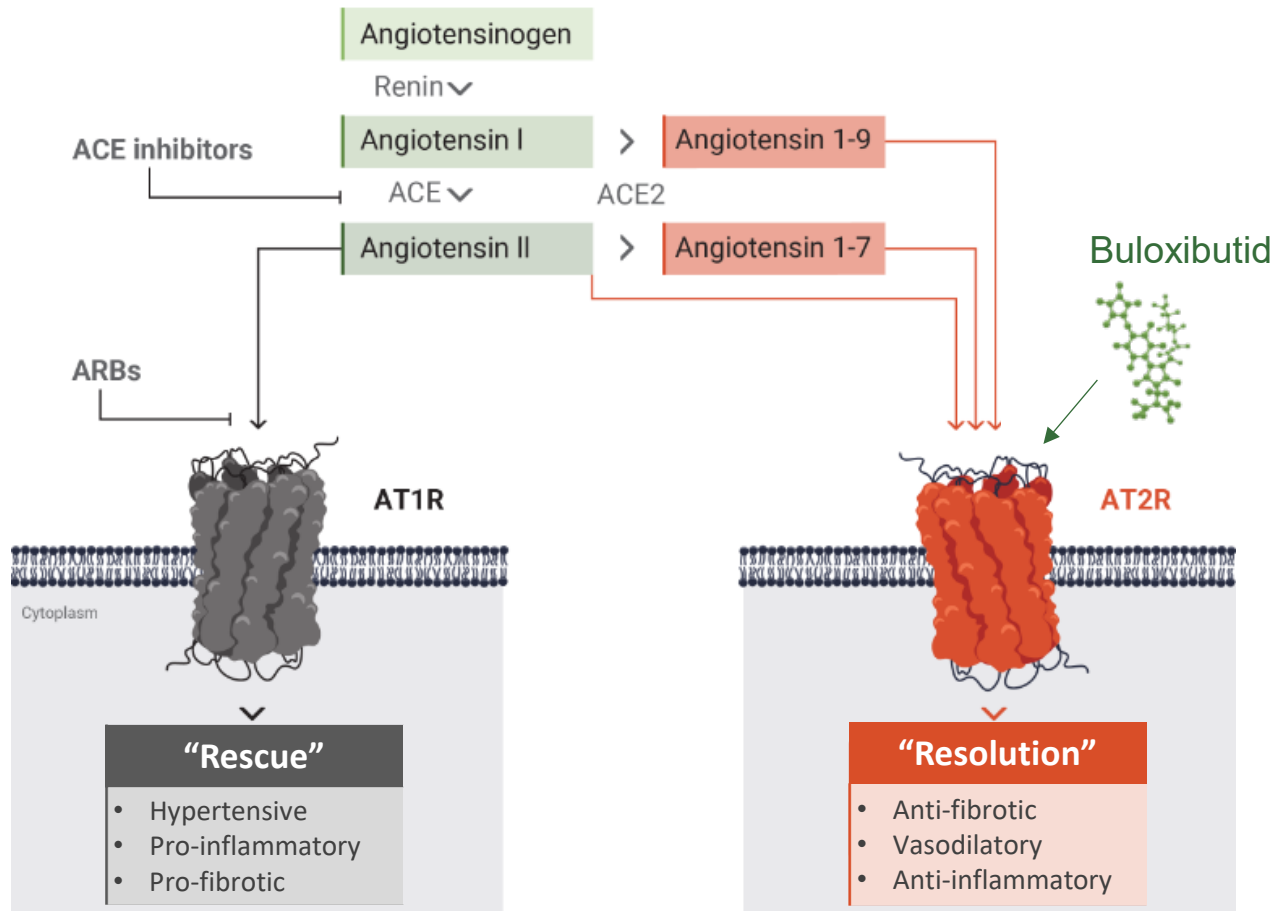
Phase 2b ASPIRE: confirming clinical activity in a randomized, placebo- controlled trial

- 52-week treatment
- Fully enrolled (n=378)
- IPF patients on stable nintedanib/SoC or not on SoC²
- Global footprint

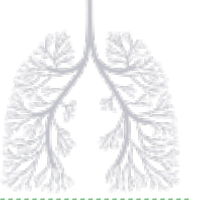




AT2R agonism is an upstream intervention driving tissue repair

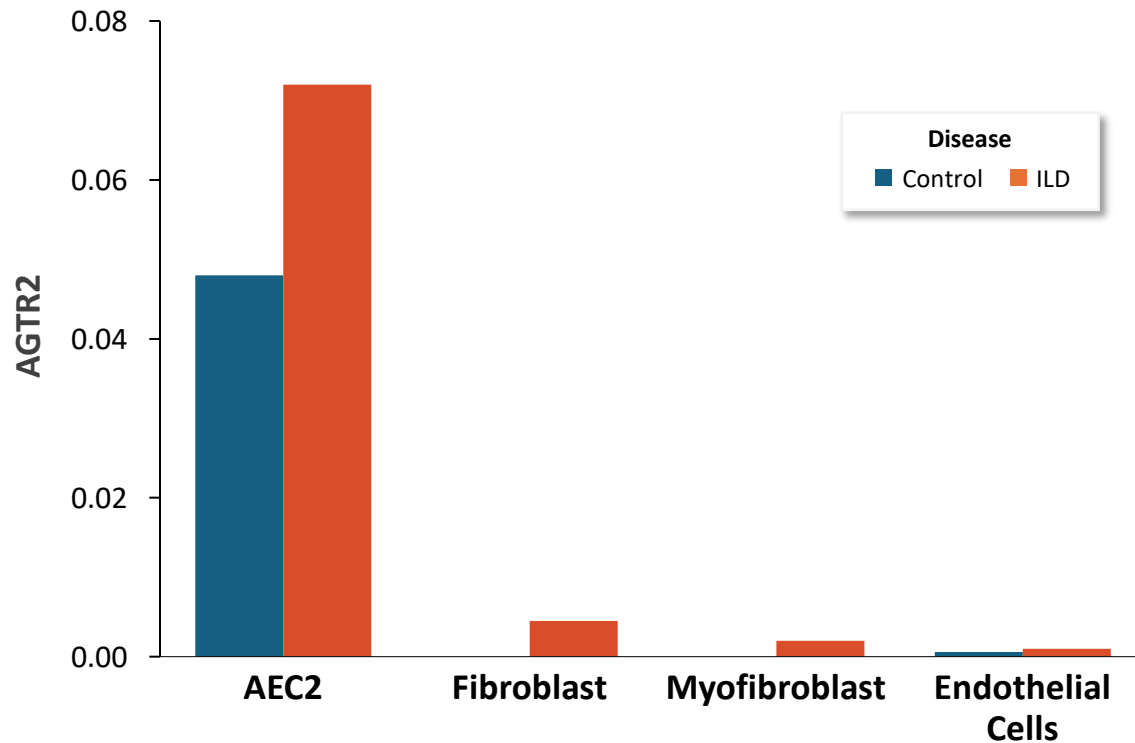


- AT2R is constitutively expressed in the lung, primarily on alveolar epithelial type 2 cells (AEC2) – the “alveolar repair cell”
- AT2R activation engages tissue-protective pathways via AEC2s, promoting inhibition of fibrotic progression and fibrosis resolution, anti-inflammatory effects, vasodilation, 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



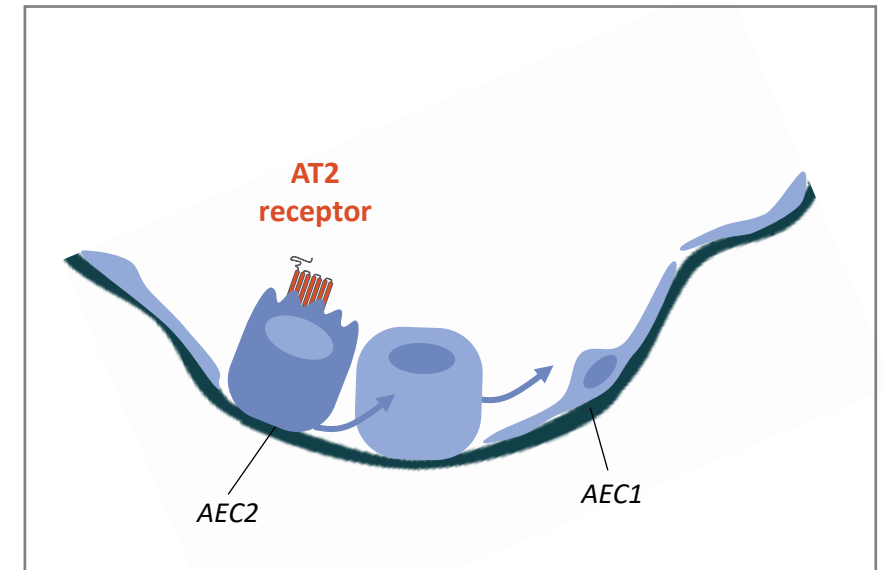
AT2R is highly expressed in human IPF lungs and on precursor AEC2s

AT2R Expression is Elevated in IPF Lung¹



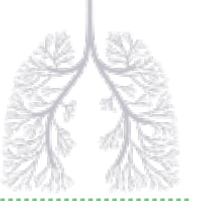
AT2R expression is highly upregulated in IPF lungs, particularly on AEC2s, and is also present on fibroblasts, myofibroblasts, and endothelial cells with higher expression in the diseased state compared with healthy tissue

AT2R is Highly Expressed on AEC2s

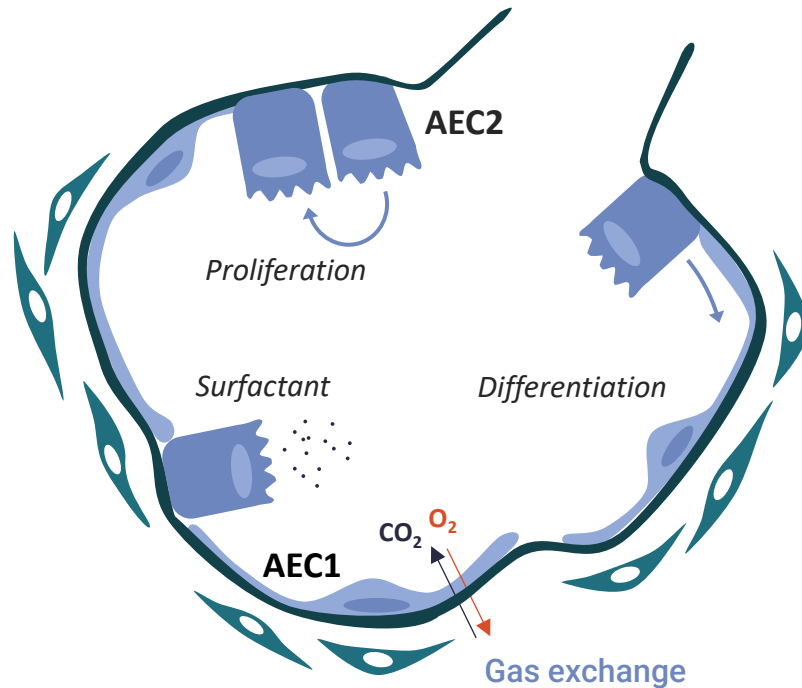


Single-cell analysis shows high AT2R expression on AEC2 in the lung, the progenitor cell that differentiates into AEC1 gas exchange cells

Alveolar epithelial cells are critical for healthy lung function



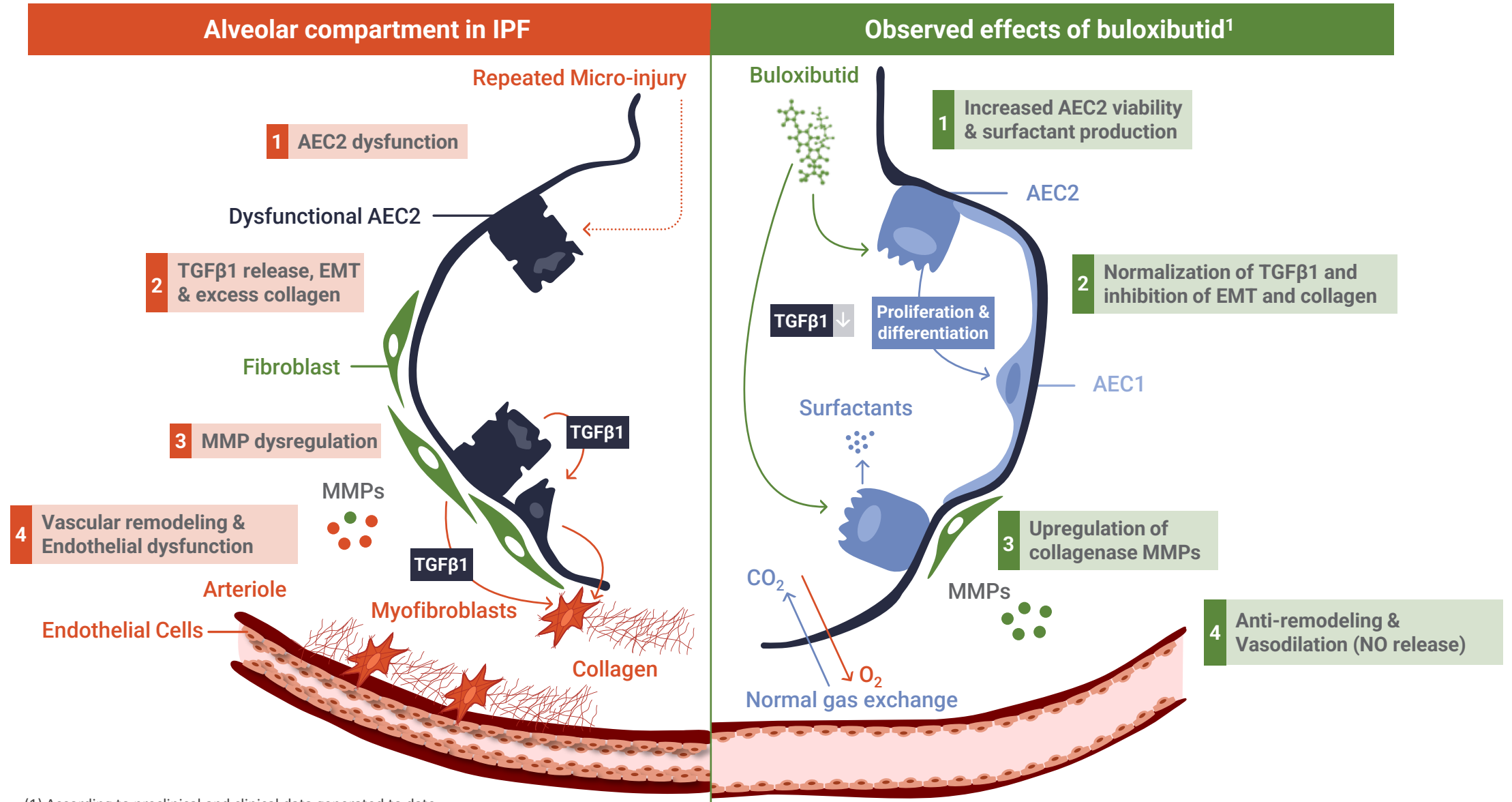
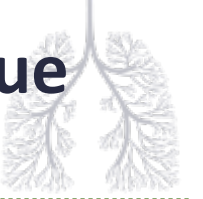
Healthy alveolus



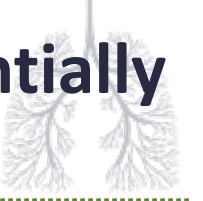
AEC – Alveolar Epithelial Cell

- 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 is associated with tissue repair via AEC2 precursor epithelial cells



Buloxibutid is designed to target key disease drivers in IPF, with a potentially disease-modifying mechanism through tissue repair

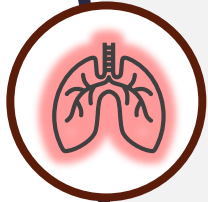


Effects Observed in Preclinical Studies



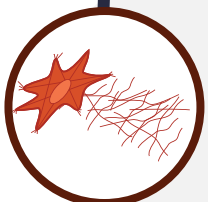
Tissue repair and regeneration

Buloxibutid designed to target precursor epithelial cells (AEC2) to drive tissue repair, offering a potentially disease-modifying mechanism of action



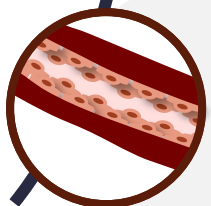
Anti-inflammatory

Buloxibutid associated with inhibition of pro-inflammatory cytokine release through inhibition of NF- κ B signaling



Anti-fibrotic

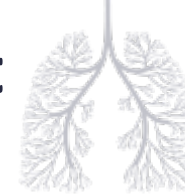
Buloxibutid associated with restoration of dysfunctional AEC2 and surfactant production, normalization of TGF β 1 levels, inhibition of EMT and collagen deposition, as well as break down of existing collagen build up



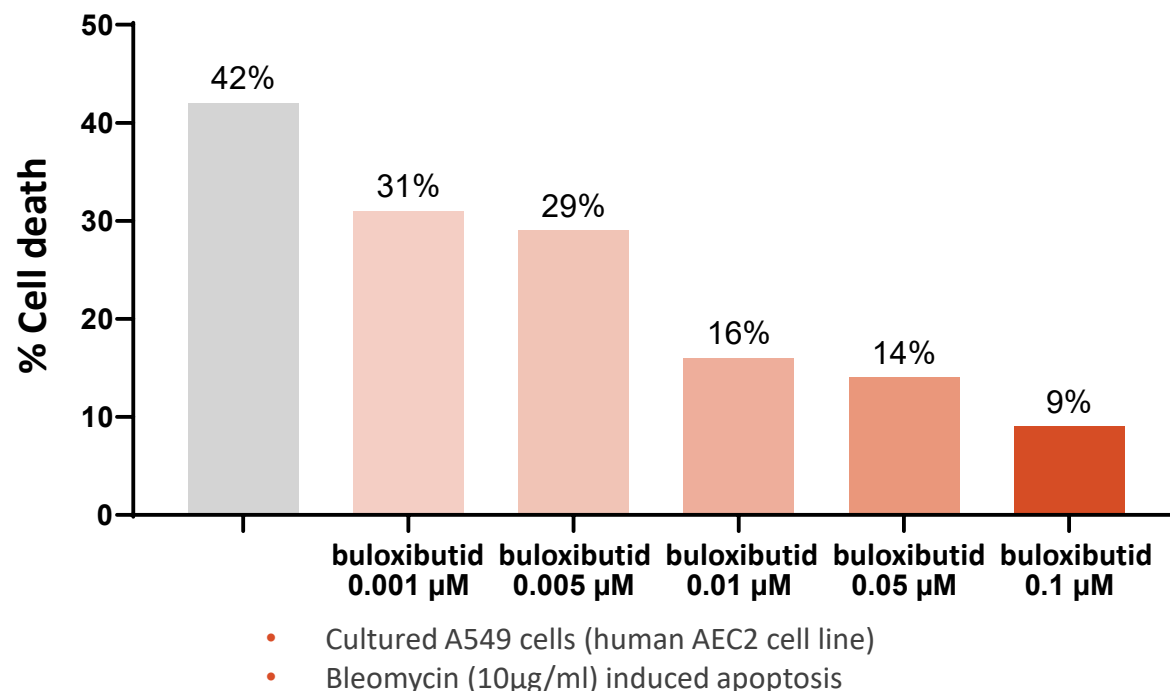
Reverses vascular remodeling

Buloxibutid associated with vasodilation and reversal of vascular remodeling through NO release

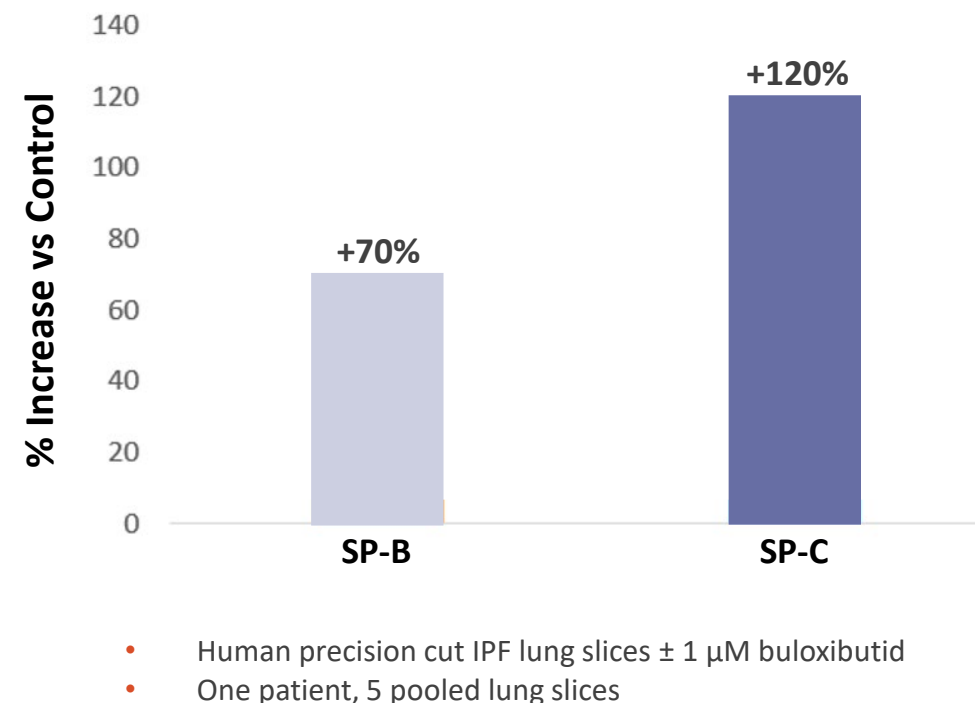
Buloxibutid is designed to protect AEC2s and drive increased surfactant production



Buloxibutid associated with reduced apoptosis of AEC2s¹

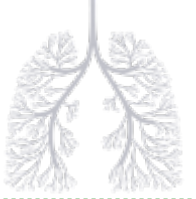


Increase in surfactant protein expression associated with buloxibutid in ex vivo human IPF precision cut lung slices²

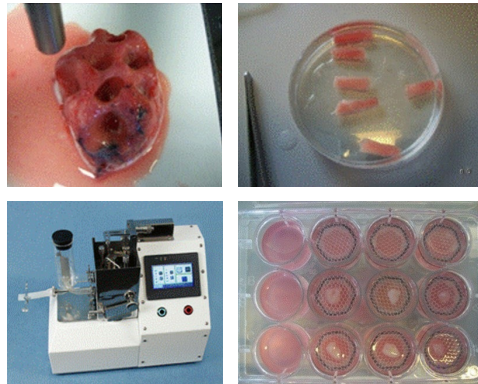


Treatment with buloxibutid is associated with protection of AEC2s, driving increased surfactant production to potentially address alveolar collapse

Buloxibutid is associated with TGFβ1 and collagen reduction in human IPF lung slices

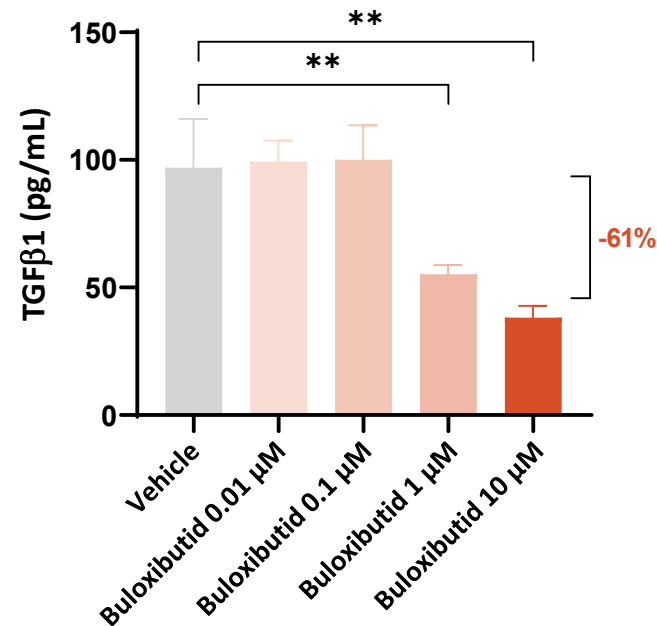


Human precision cut lung slices (PCLuS)

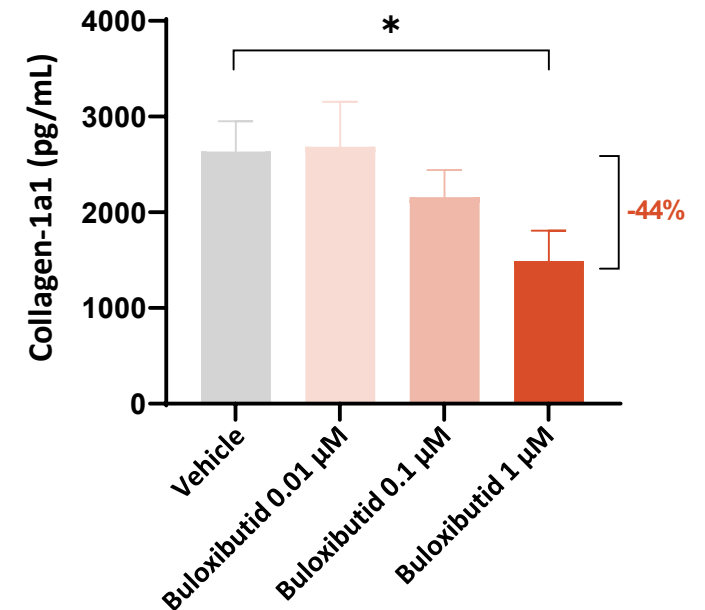


- Lung tissue collected from IPF patients undergoing transplant
- Intrinsic fibrosis, no stimuli added

TGFβ1 protein levels in PCLuS



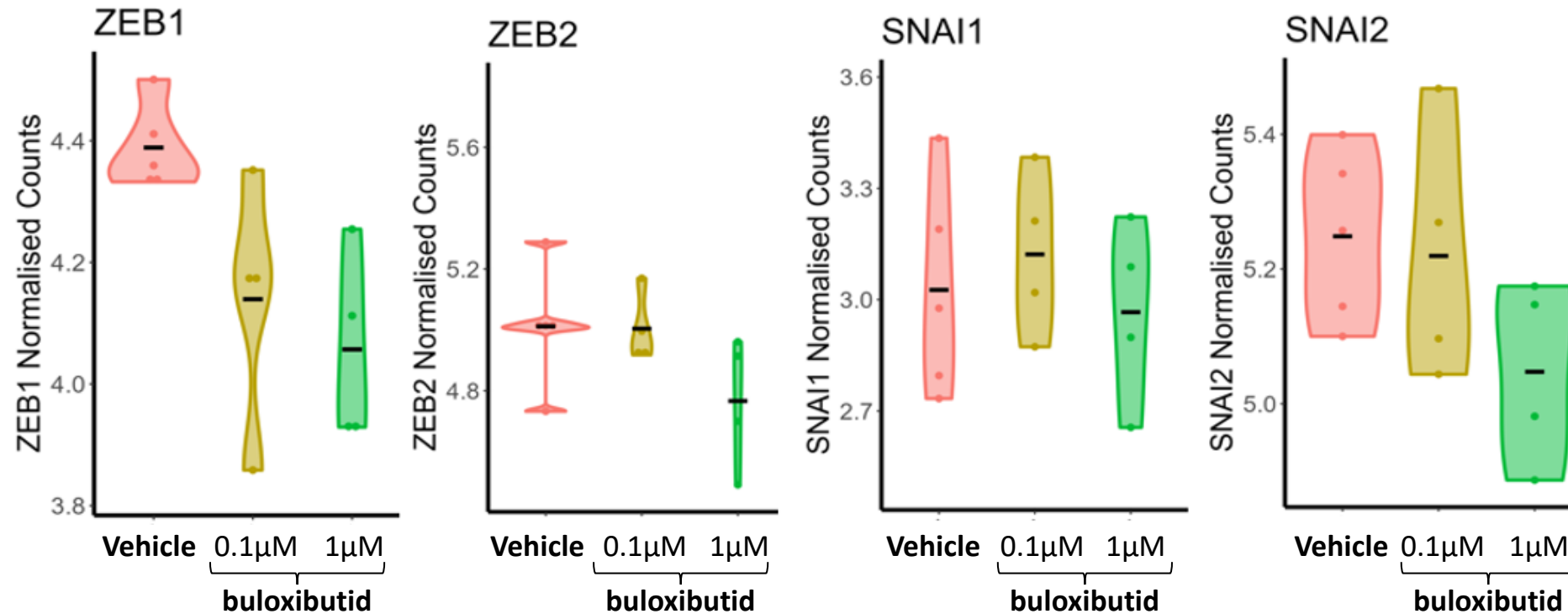
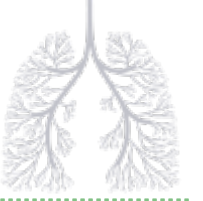
Collagen protein levels in PCLuS



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

Buloxibutid is associated with downregulated expression of EMT transcription factors in AEC2

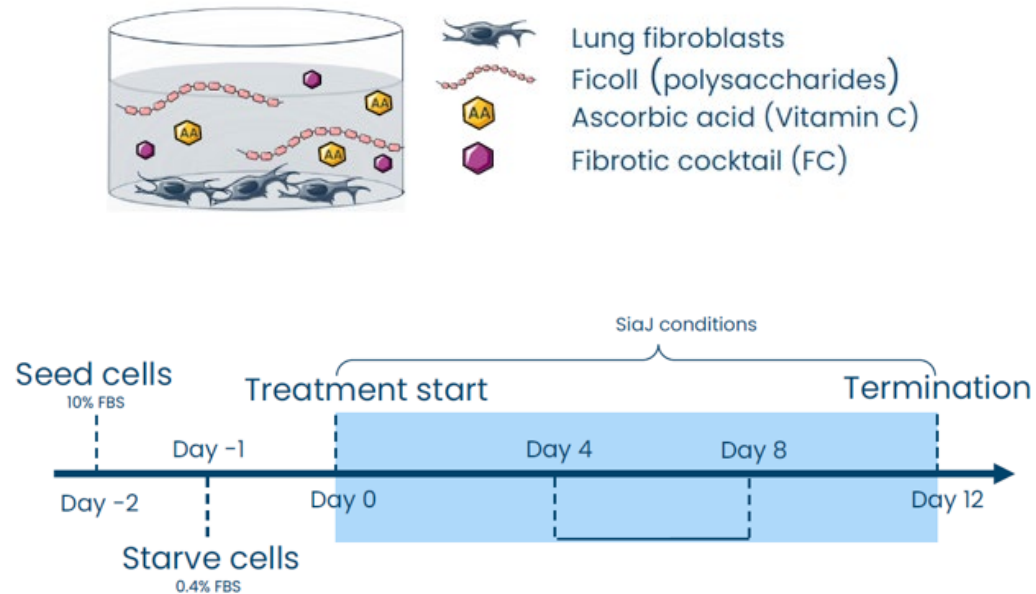


- Primary AEC2 cultures established from normal surgically resected human lung
- Buloxibutid treatment under baseline conditions with no stimuli added

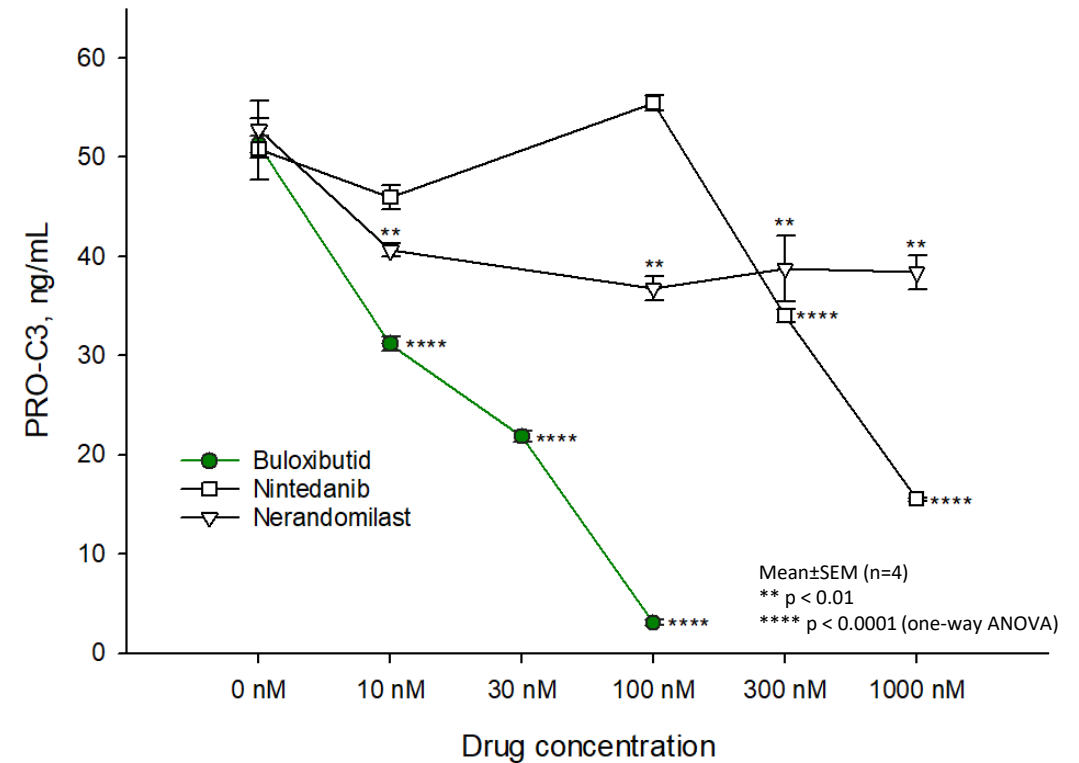
Collagen biomarker PRO-C3 inhibited in human lung fibroblast assay treated with buloxibutid, suggesting resolution of fibrosis



Human lung fibroblast assay methodology

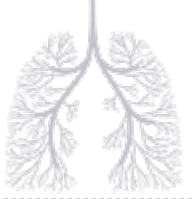


Impact on type III collagen biomarker PRO-C3



Buloxibutid associated with potent and dose-dependent inhibition of PRO-C3, suggesting inhibition of type III collagen formation and fibrotic activity. In a preclinical in vitro assay, buloxibutid showed potent inhibition of IPF biomarker PRO-C3 compared to nintedanib and nerandomilast, which may be indicative of its anti-fibrotic mechanism.

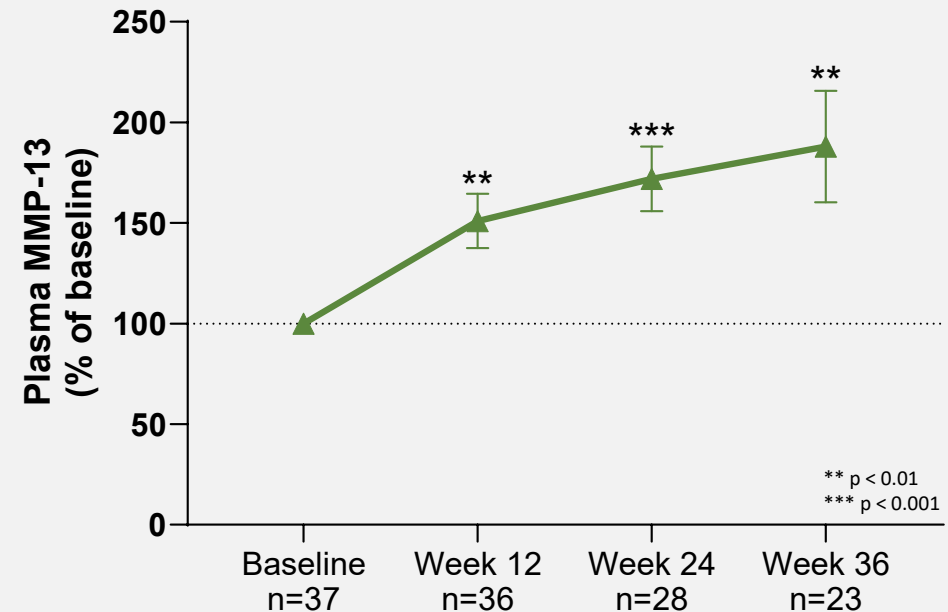
MMP-13 demonstrates antifibrotic activity and is crucial for lung repair in IPF



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

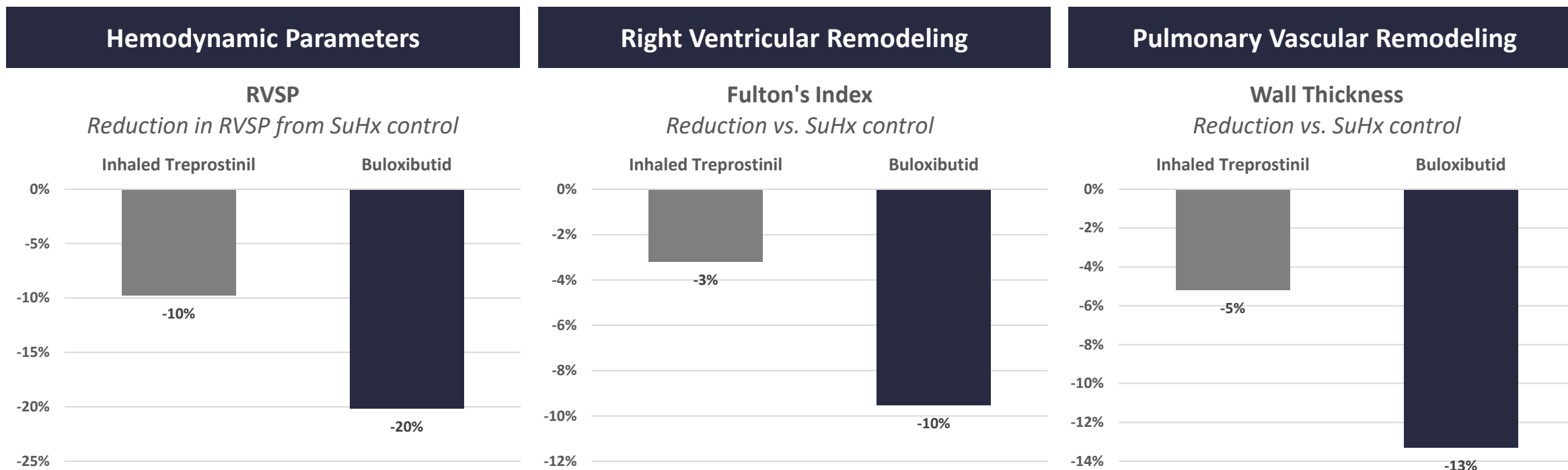


Patients treated with buloxibutid showed a significant increase in plasma levels of the collagenolytic collagenase MMP-13, suggesting that buloxibutid has the potential to degrade fibrosis

Buloxibutid demonstrated 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



Inhaled treprostinil: adapted from Corboz, et al., J. Pharmacol. Exp. Ther. 2022 – Dose: 65 µg/kg

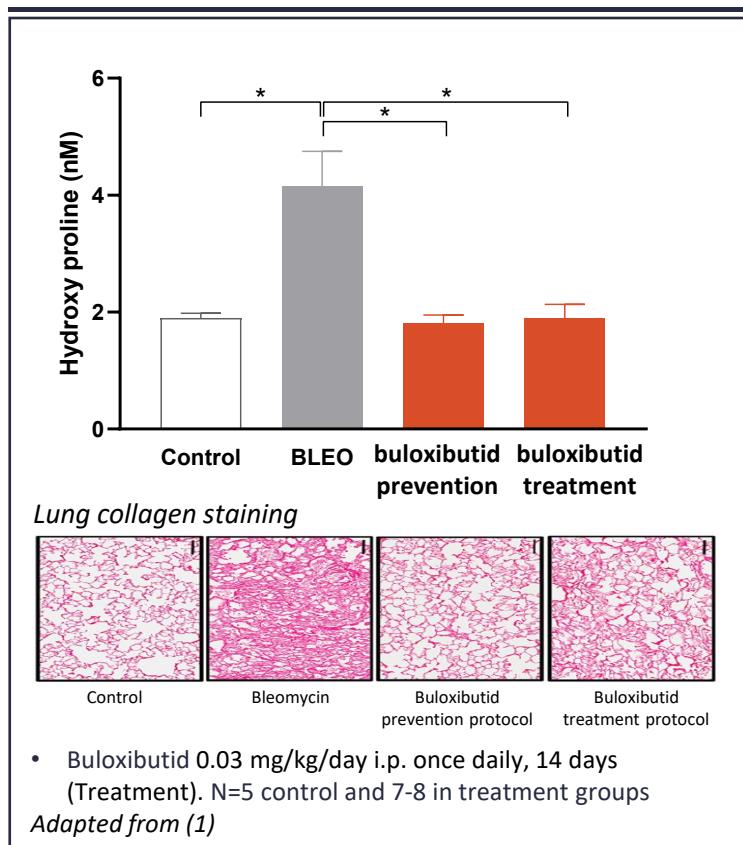
Buloxibutid: adapted from Tornling, et al., Int. J. Mol. Sci. 2023 – Dose: average result of 2 mg/kg and 20 mg/kg dose

Clinically relevant concentrations of buloxibutid demonstrated 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)

Strong preclinical in vivo evidence for buloxibutid in pulmonary fibrosis

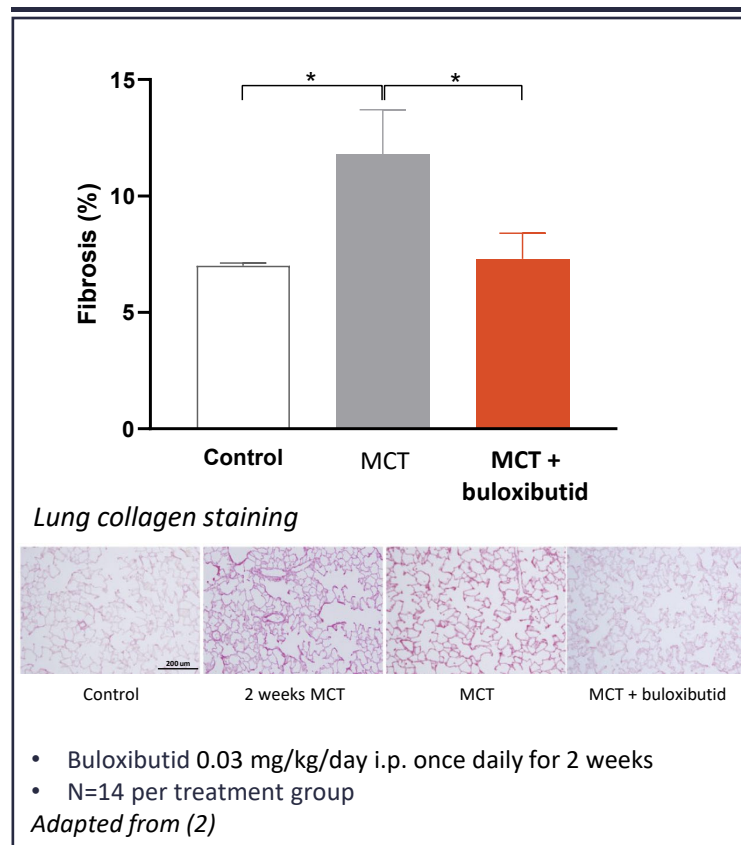


Bleomycin



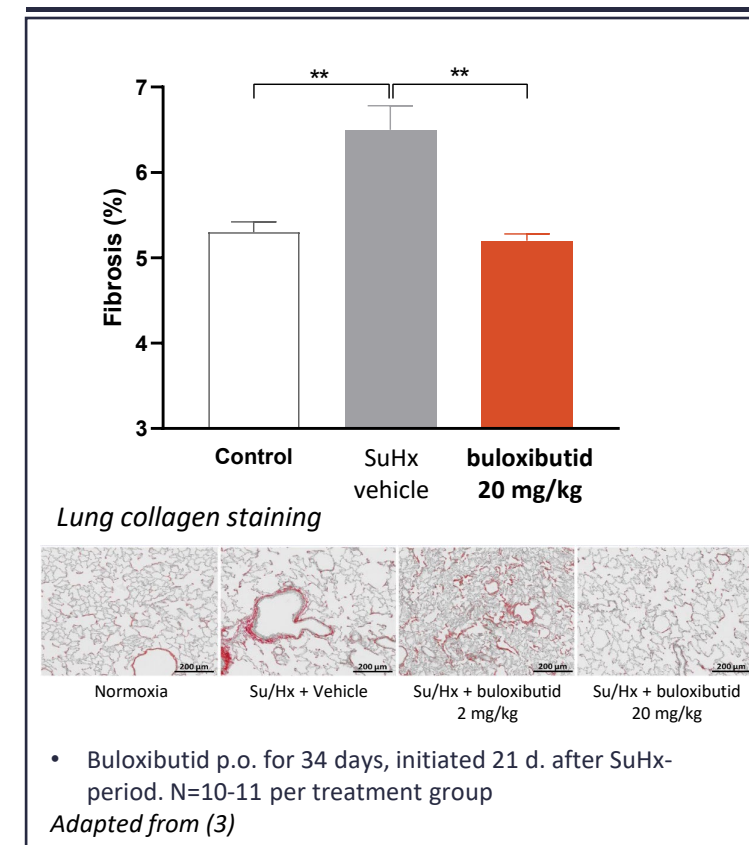
- Normalized collagen synthesis and attenuation of disrupted lung architecture

Monocrotaline



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

Sugen-Hypoxia



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

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

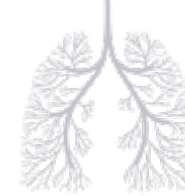
- Not including patients enrolled in the ongoing Phase 2b ASPIRE trial, a total of **366 trial participants** have been exposed to buloxibutid over the course of 9 completed clinical trials
- In the recently completed Phase 2a AIR trial, IPF patients were exposed to buloxibutid for **36 weeks**



No significant safety risks have been identified for buloxibutid to date based on current clinical dataset

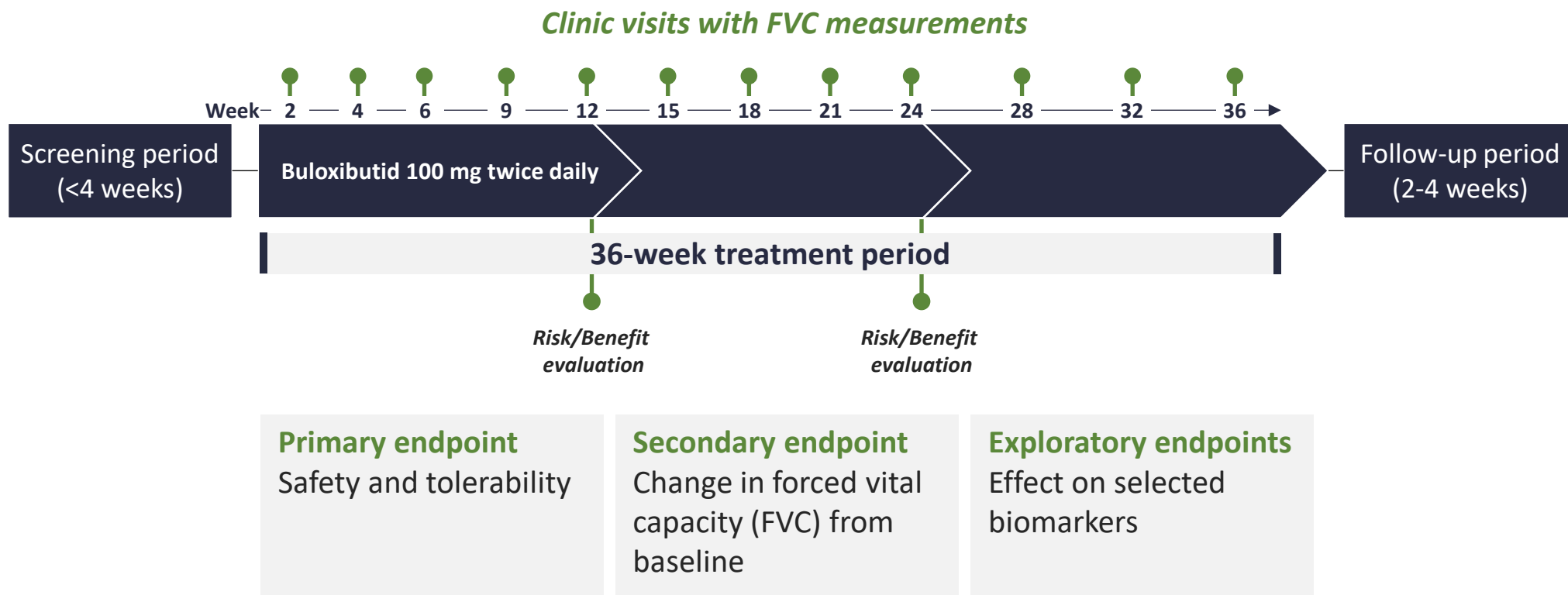
- The only identified risk of treatment with buloxibutid is mild to moderate hair loss, which was observed to be reversible, in 19% of participants in the Phase 2a AIR trial
- Across the robust safety dataset, there have been no treatment-related SAEs

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

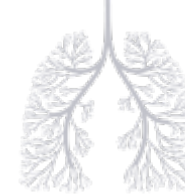


Patient population

Treatment-naïve IPF patients with centrally HRCT-confirmed diagnosis



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%

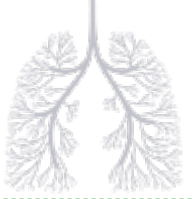
In line with other trials

Enrolled study population has disease severity comparable to global IPF study populations²

In line with other trials

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

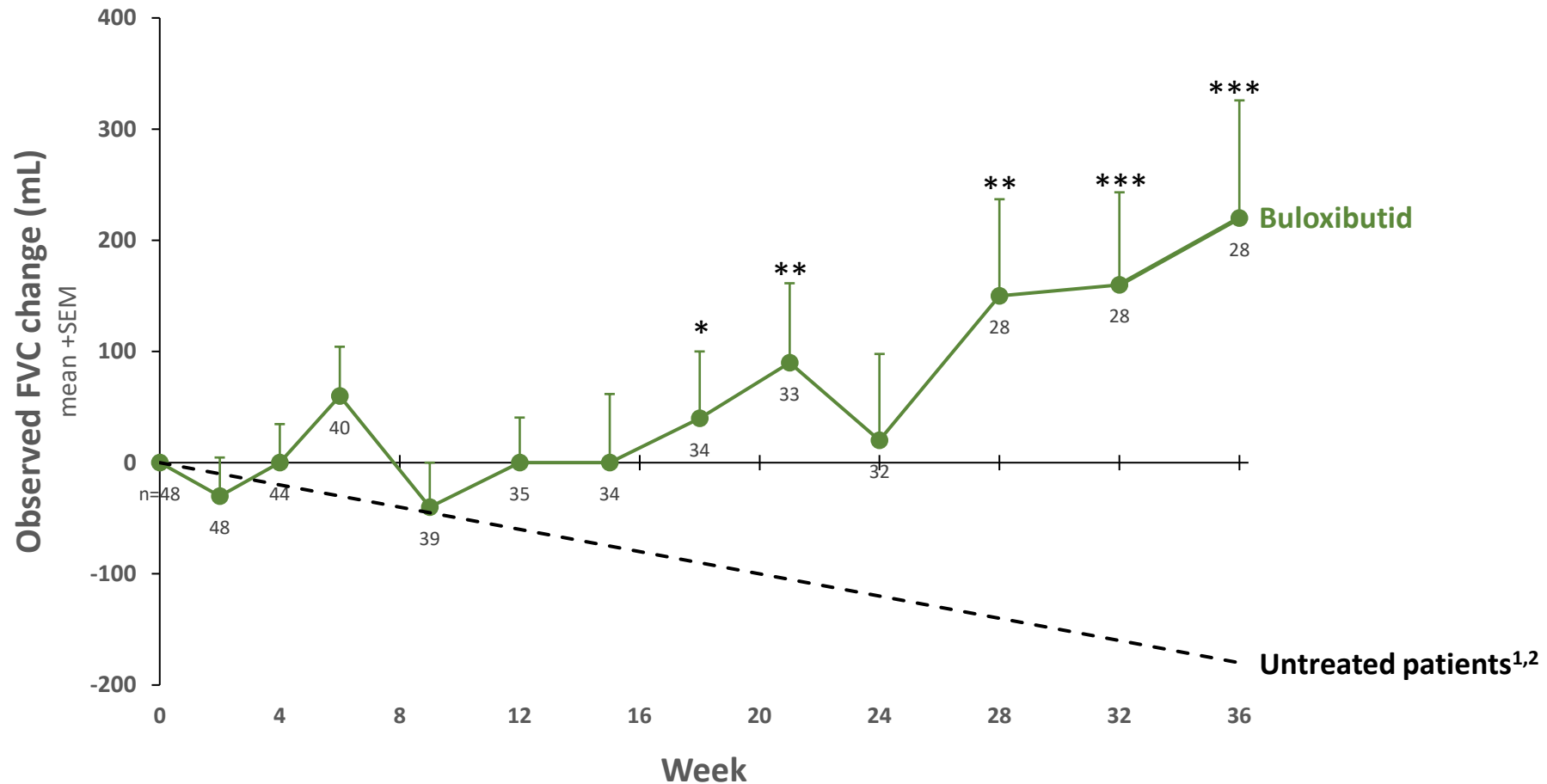
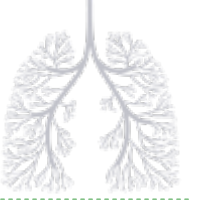
Treatment emergent adverse events: buloxibutid shows better tolerability than SoC



	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%	} Good GI tolerability
Nausea	23%	6%	4%	
Acute exacerbation of IPF	10%	10%	6%	} Low rate of exacerbations and cough worsening
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%	} No observed serious, severe, or fatal AEs related to buloxibutid
Severe AE	26%	18%	6%	
Serious AE	31%	27%	10%	

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

Buloxibutid was observed to stabilize and improve lung function over the 36-week Phase 2a AIR trial

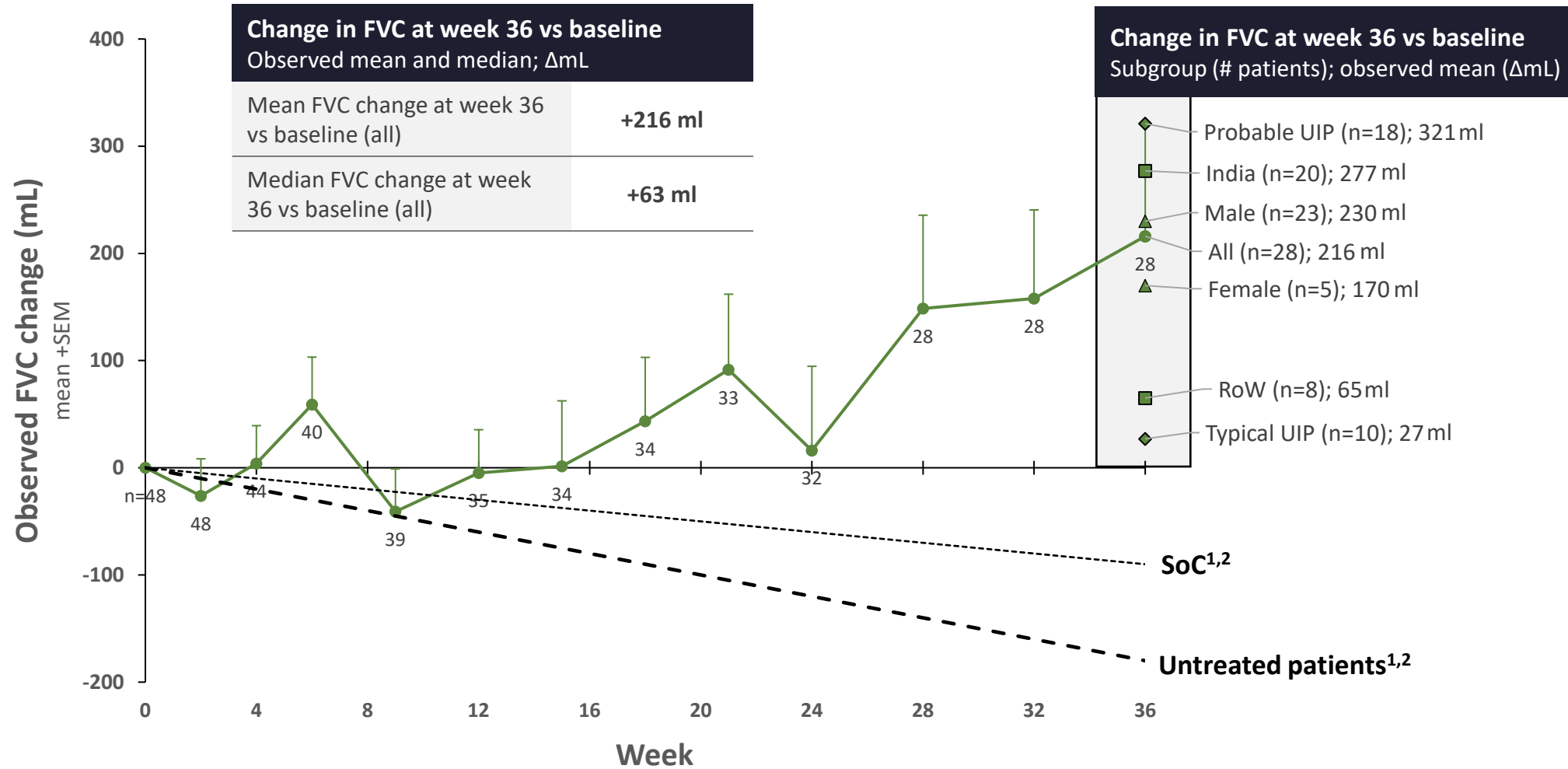
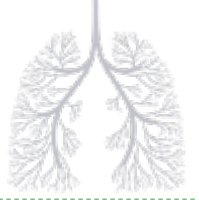


Note: N=48 patients with 2-week change from baseline FVC data. Observed values, no imputation.

*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; (2) Richeldi et al. N Engl J Med. 2014

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

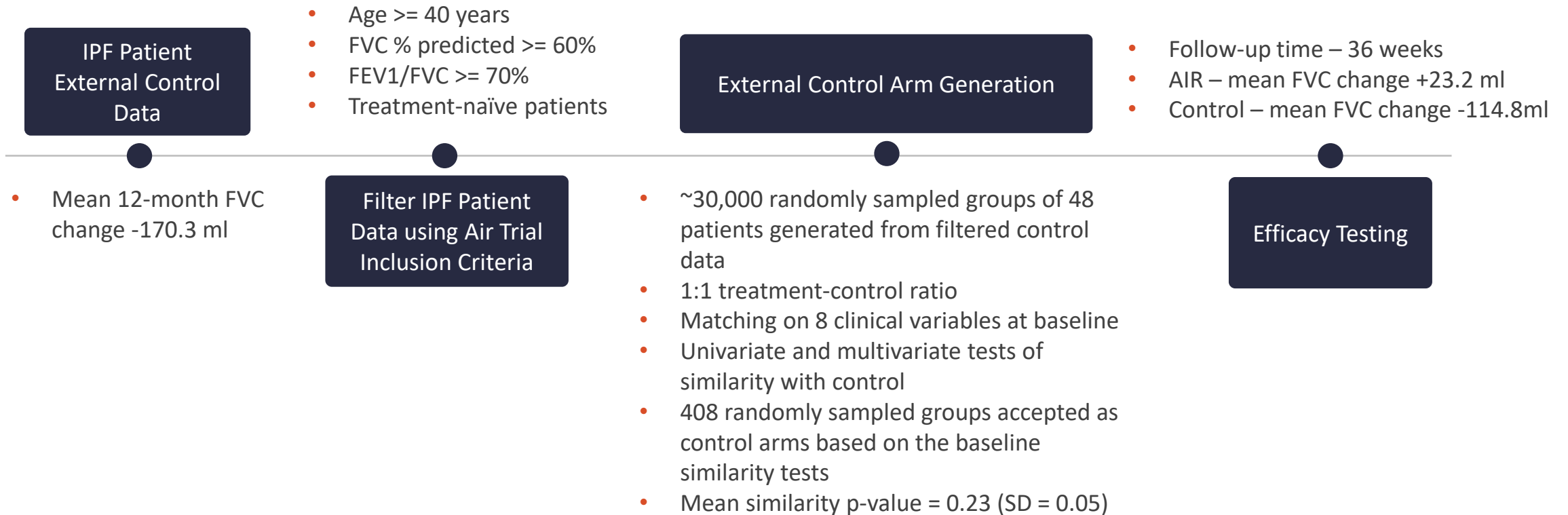
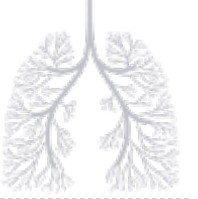


Note: N=48 patients with 2-week change from baseline FVC data. Observed values, no imputation.

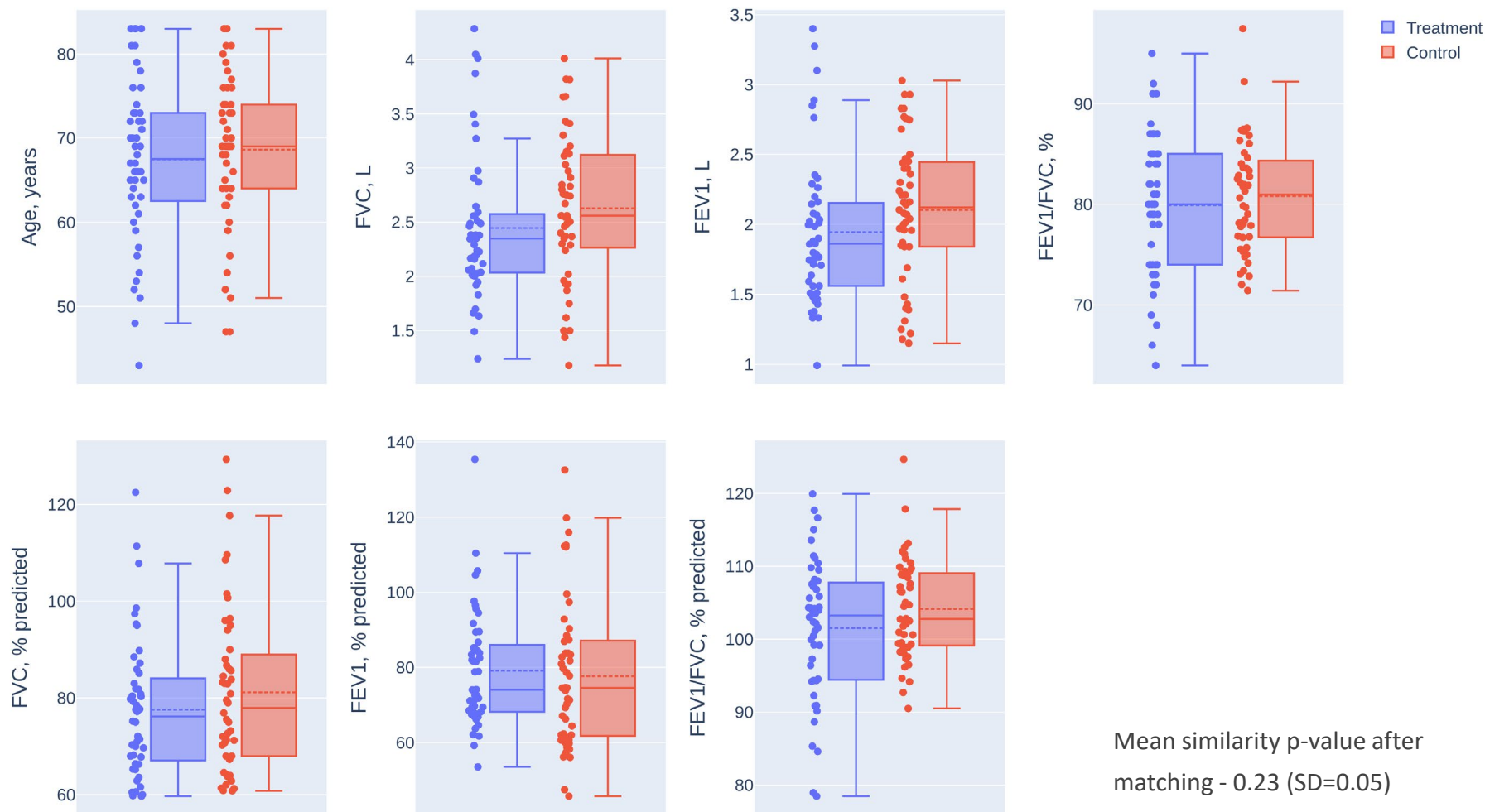
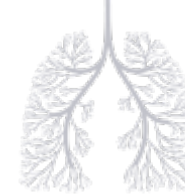
Note: Probable UIP and Typical UIP according to HRCT categorization at baseline.

(1) Noble et al. Eur Respir J. 2016; (2) Richeldi et al. N Engl J Med. 2014

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



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

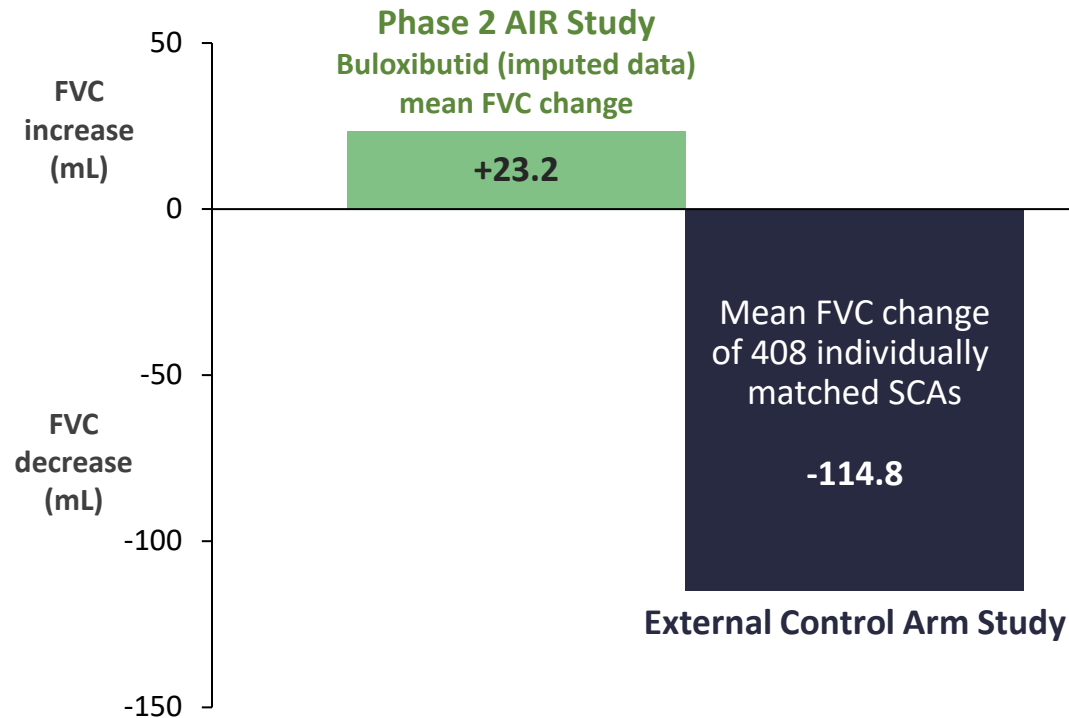


Mean similarity p-value after matching - 0.23 (SD=0.05)

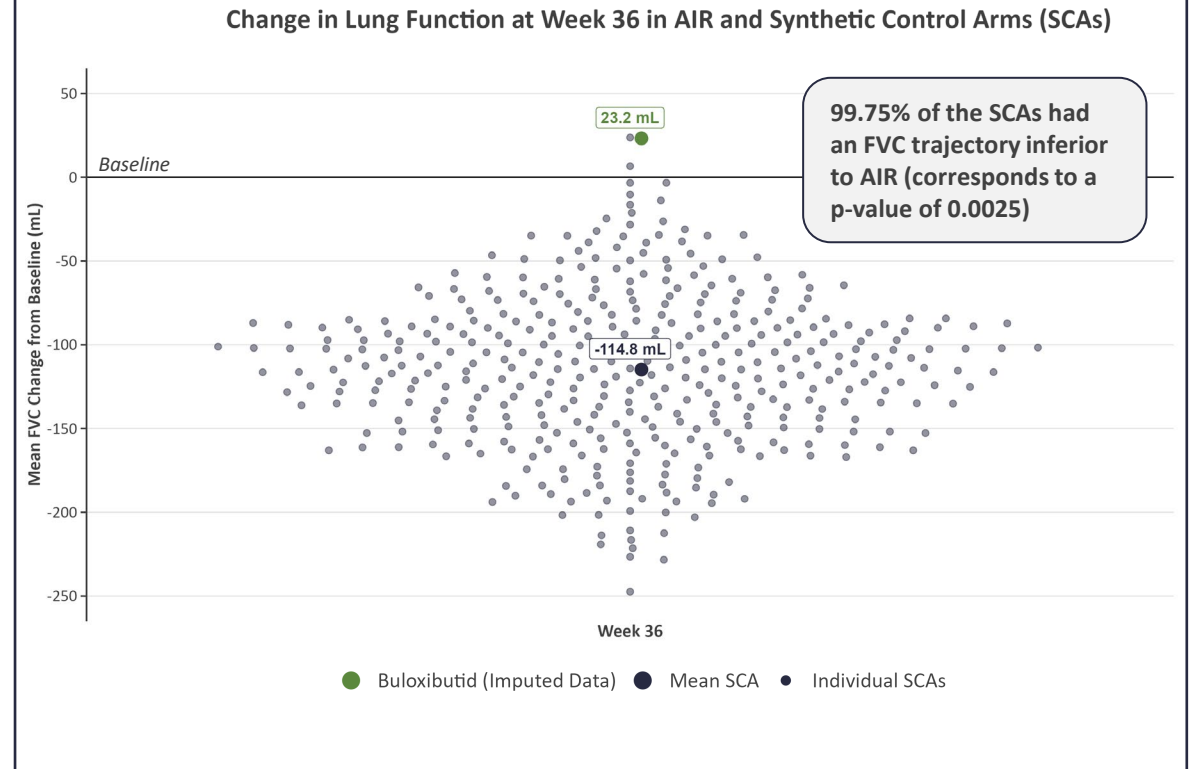


A Synthetic Control Arm analysis supports a clinically meaningful treatment effect

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

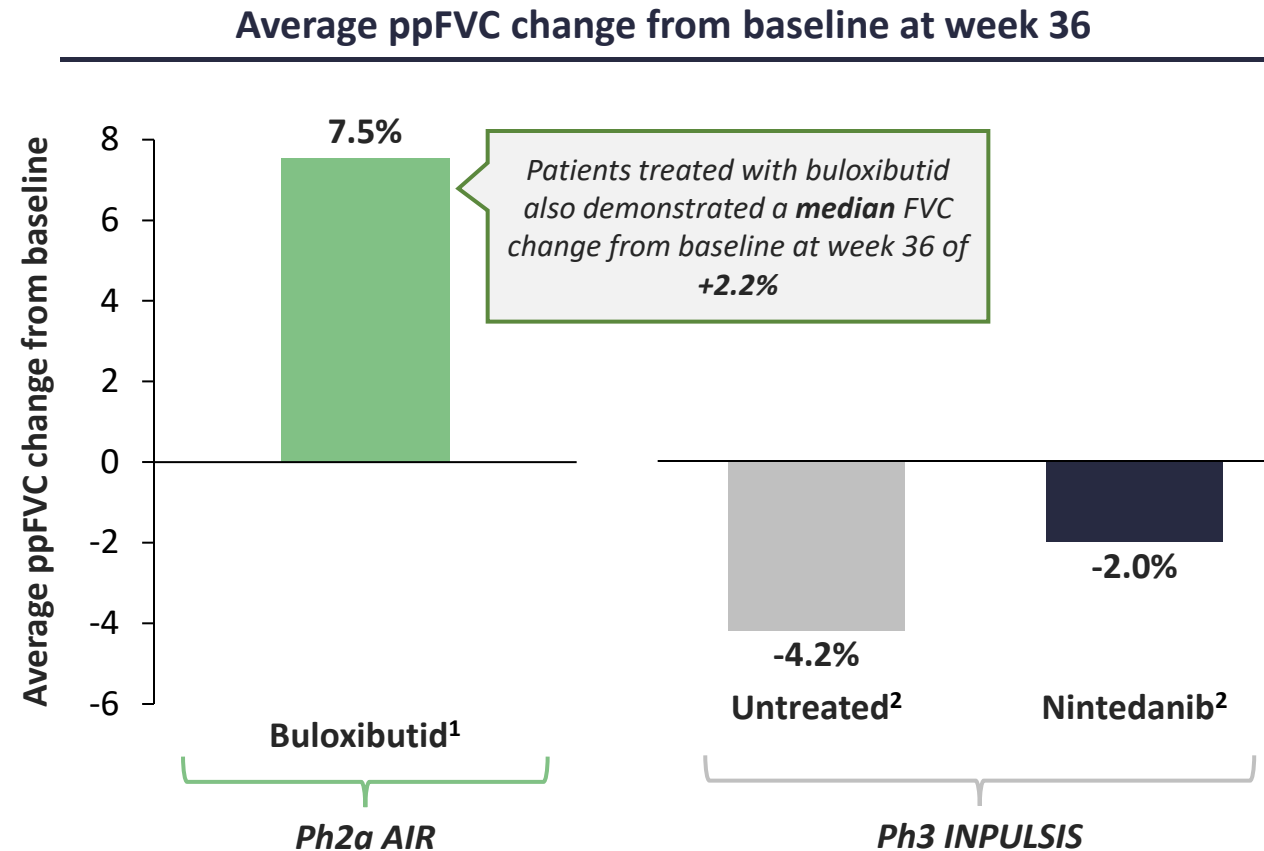
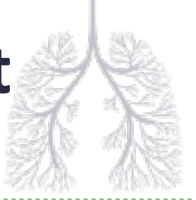


408 individual matched Synthetic Control Arms (SCAs) generated by Monte Carlo cross validation



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

Buloxibutid is associated with a significant increase in ppFVC, consistent with its observed impact on absolute FVC

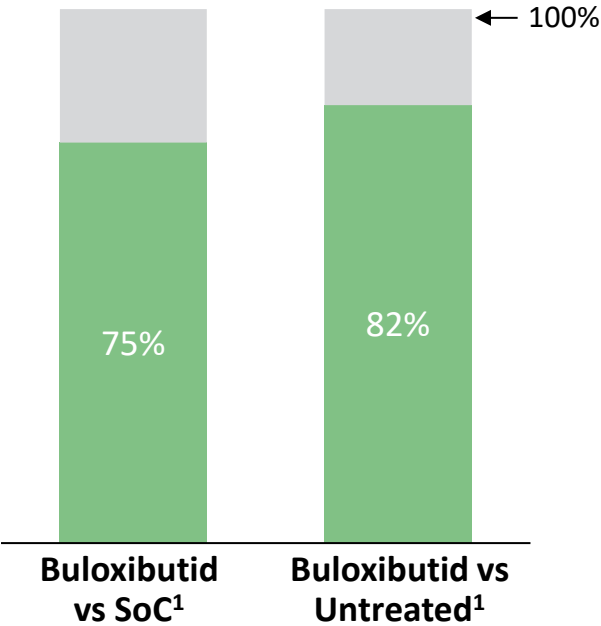


Average increase of 7.5 percentage points in ppFVC at 36 weeks from baseline

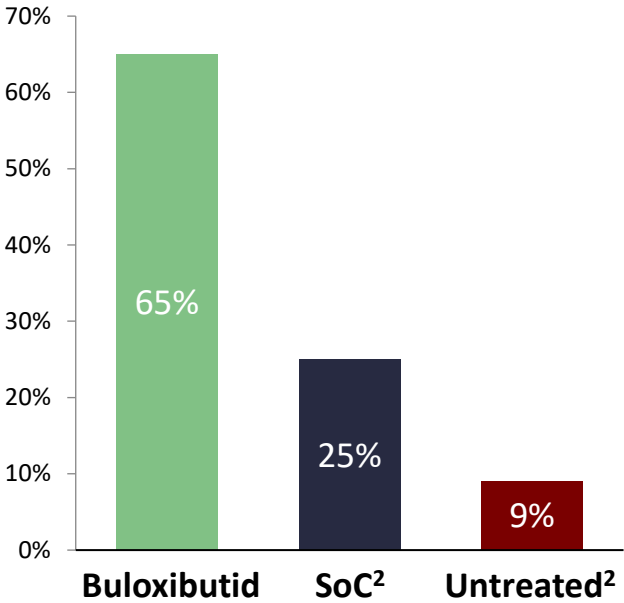
Buloxibutid showed favorable results compared to historical standard of care and untreated decline at 36 weeks



Percentage of patients showing more favorable expected Δ FVC



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

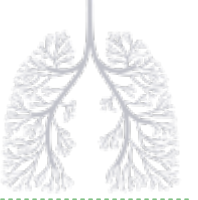


Patients treated with buloxibutid showed more favorable expected change in FVC compared with untreated patients and those treated with current standard of care at 36 weeks

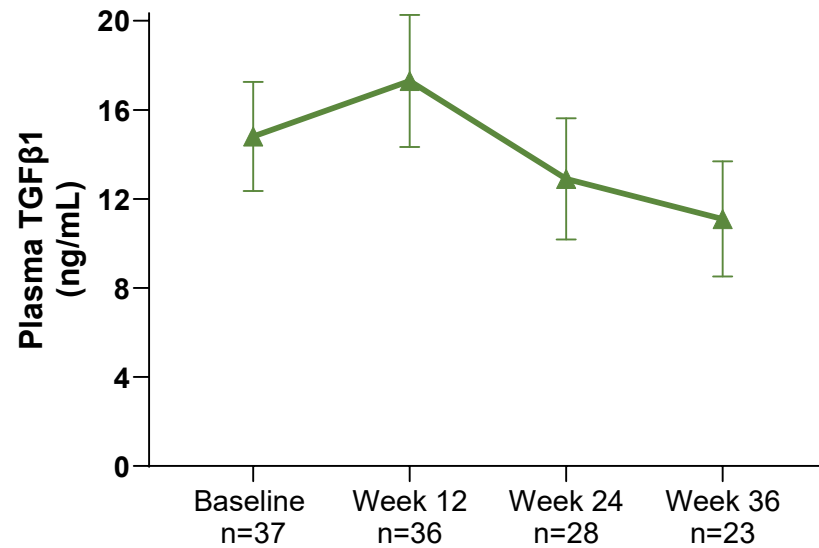
Most patients treated with buloxibutid were observed to have improved lung function at 36 weeks, showing more favorable results compared to historical SoC and untreated patients

(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%.
Analysis based on historical data and does not represent head-to-head study. Cross-trial comparisons are challenging due to differences in trial design, patient selection, and analysis method.

Buloxibutid is associated with increase of collagenase MMP-13 and decrease of TGF β 1 in the Phase 2a AIR trial

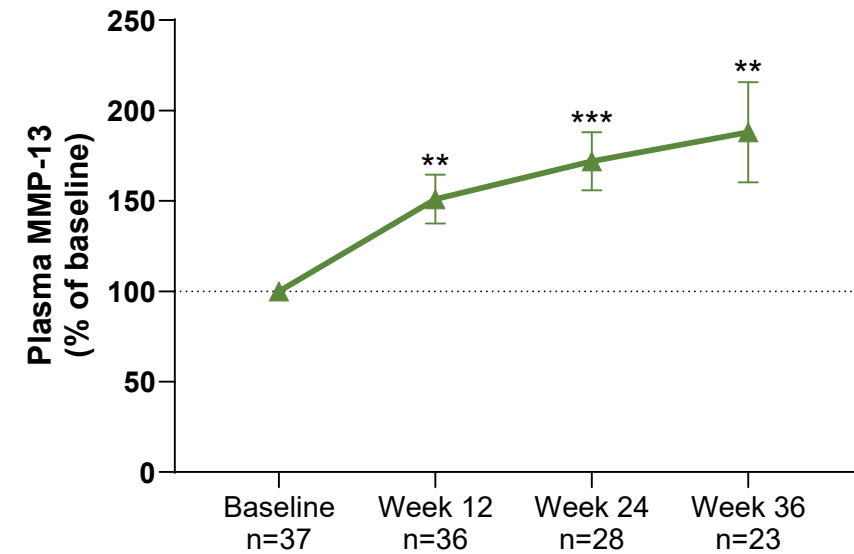


Plasma TGF β 1



TGF β 1 is a key fibrotic driver in IPF; reduced TGF β 1 is consistent with buloxibutid's mechanism of action and translational data

Plasma MMP-13

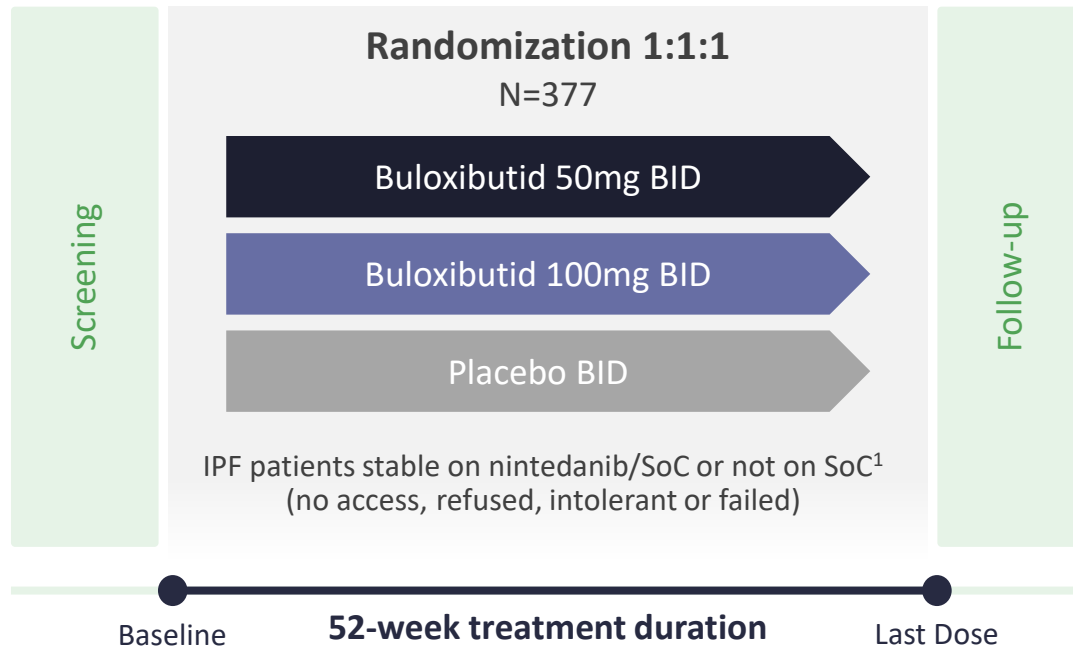


MMP-13 is an antifibrotic collagenase that plays a key role in fibrotic resolution

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



Primary Endpoint:

Change from baseline in FVC at 52 weeks

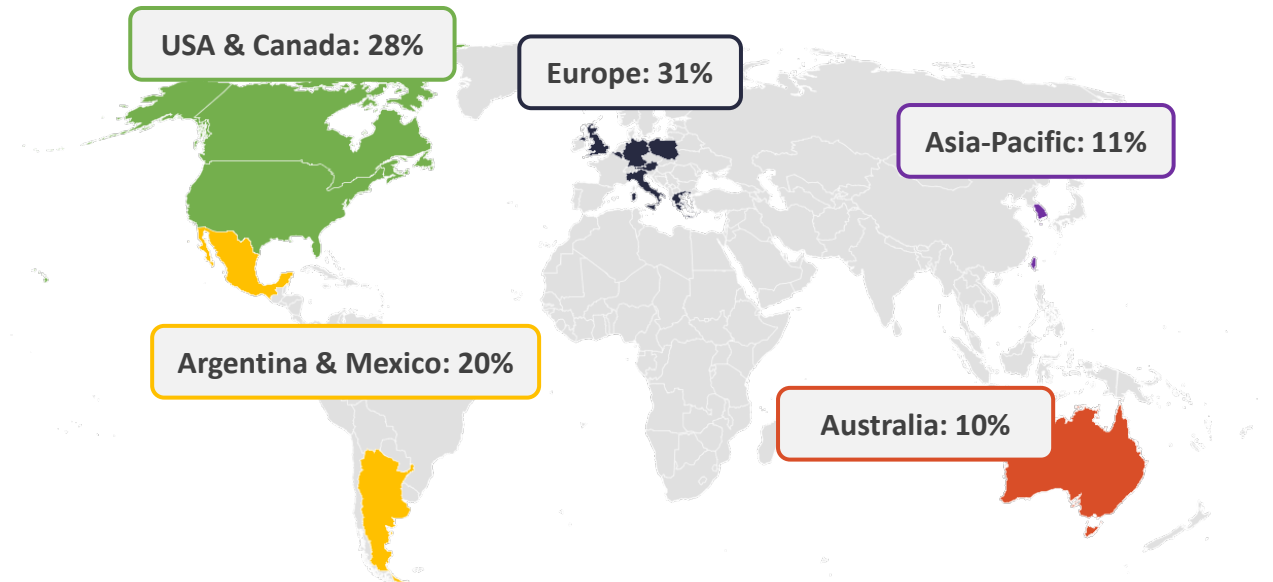
Key Secondary Endpoint:

Proportion of patients with disease progression at 52 weeks

Global Footprint

>100 sites across 14 countries

Proportion of Patients Randomized by Region*



*Preliminary data, reflecting 16 June 2026 database extract

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

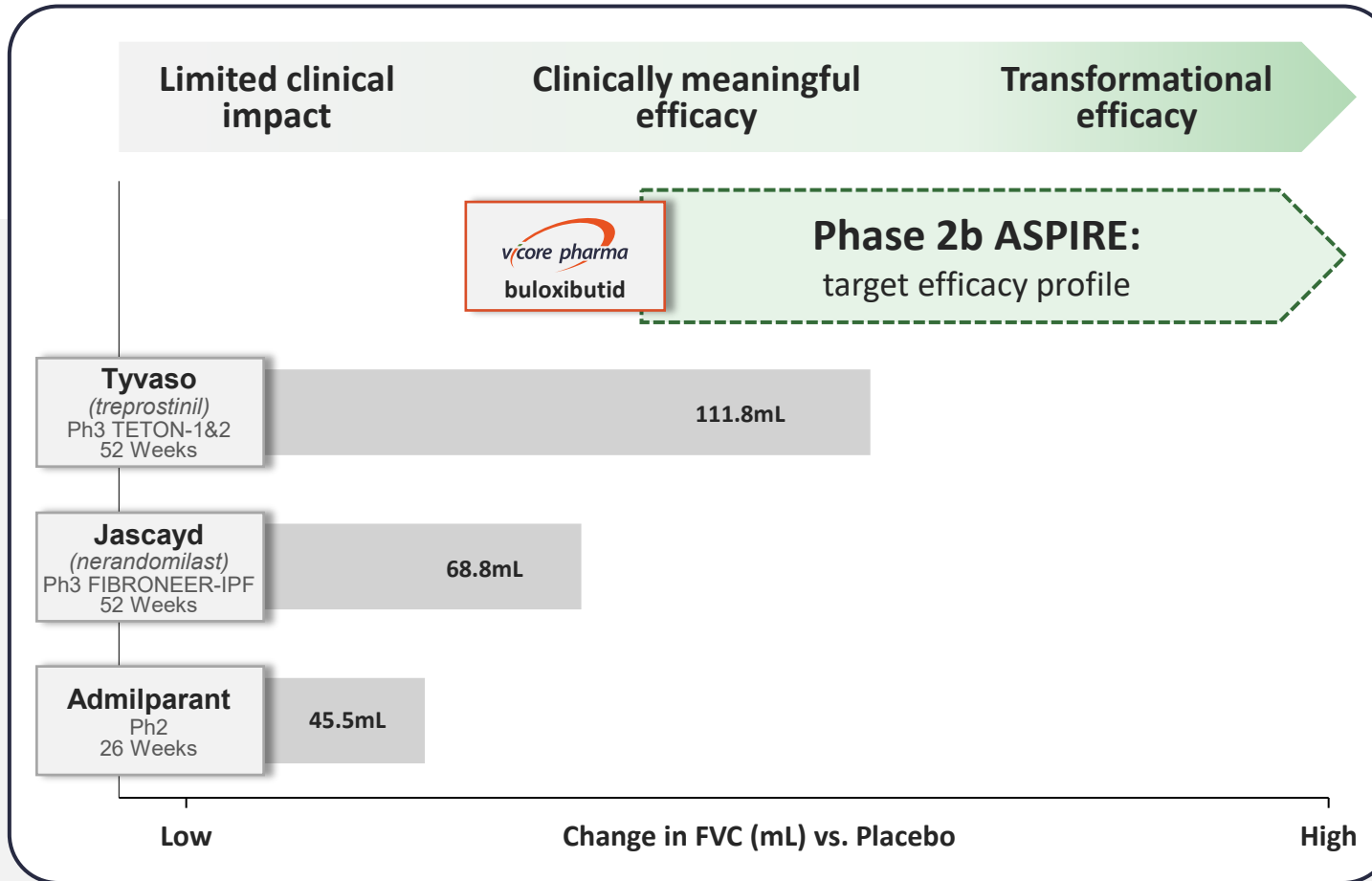
Method

- 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

- 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 potentially reshape the IPF treatment paradigm

ASPIRE baseline characteristics



Characteristics	ASPIRE (N=377*)
Age (years) – Mean (SD)	71.1 (7.5)
Gender	
Males	78.2%
Females	21.8%
Ethnicity	
White	86%
Asian	13%
American Indian or Alaska Native	1%
Black or Afro American	<1%
Time since diagnosis (years) – Mean (SD)	2.4 (1.7)
BMI (kg/m ²) – Mean (SD)	27.9 (4.8)
Smoking status	
Never smoked	36.9%
Former smoker	61.5%
Current smoker	1.6%
FVC (liter) – Mean (SD)	2.9 (0.8)
FVCpp – Mean (SD)	81.3% (17.6)
FEV1/FVC ratio – Mean (SD)	0.8 (0.06)
DLCOpp, Hb corrected – Mean (SD)	54.1% (15.7)
Oxygen saturation – Mean (range)	96% (86-100)
Background antifibrotic treatment at study start	
Not on antifibrotic treatment	40.3%
On antifibrotic treatment	59.7%

67.8% of the patients not on antifibrotic treatment have never been treated with an antifibrotic treatment (27.3% of all patients)

*Total randomized participants exposed to at least one dose of IMP

BMI: body mass index; DLCO: diffusing capacity of the lungs for carbon monoxide; FEV₁: forced expiratory volume in 1 second;

FVC: forced vital capacity; FVCpp: FVC percent predicted; Hb: haemoglobin; SD: standard deviation.

Preliminary data, reflecting 16 June 2026 database extract

ASPIRE baseline characteristics are in line with the latest IPF Ph3 trials



Characteristics	ASPIRE	FIBRONEER-IPF	TETON-1 & 2	ALOFT-IPF
	All patients (N=377*)	All patients (N=1,177)	All patients (N=1,191)	All patients (N=1,251)
Age (years) – Mean (SD)	71.1 (7.5)	70.2	72.3	71.0
Gender				
Males	78.2%	83.0%	78.7%	79.4%
Females	21.8%	17.0%	21.3%	20.6%
Ethnicity				
White	86%	68% [§]	87.2%	57.7%
Asian	13%	32%	8.0%	40.4%
American Indian or Alaska Native	1%	<1%	0.3%	-
Black or Afro American	<1%	<1%	1.0%	-
Other	-	0%	3.6% [†]	1.8% [‡]
Time since diagnosis (years) – Mean (SD)	2.4 (1.7)	3.5	3.7 (2.9)	2.1
Smoking status				
Never smoked	36.9%	29.0%	34.6%	-
Former smoker	61.5%	68.8%	63.7%	-
Current smoker	1.6%	2.2%	1.7%	-
FVC (liter) – Mean (SD)	2.9 (0.8)	2.8	2.7 (0.8)	2.7
FVCpp – Mean (SD)	81.3% (17.6)	78.2%	75.7% (17.4)	78.9%
DLCOpp, Hb corrected – Mean (SD)	54.1% (15.7)	50.9%	48.8% (18.8)	51.9%
Background antifibrotic treatment at study start				
Not on antifibrotic treatment	40.3%	22.3%	23.5%	35.9%
On antifibrotic treatment	59.7%	77.7%	76.5%	64.1%

*Total randomized participants exposed to at least one dose of IMP

BMI: body mass index; **DLCO:** diffusing capacity of the lungs for carbon monoxide; **FEV₁:** forced expiratory volume in 1 second; **FVC:** forced vital capacity; **FVCpp:** FVC percent predicted; **Hb:** haemoglobin; **SD:** standard deviation.

ASPIRE data: preliminary data, reflecting 16 June 2026 database extract

[†] includes Native Hawaiian or Other Pacific Islander, multiple or unknown,

[‡] American Indian, Alaska Native, Black or Afro American, unknown or not reported

[§] FIBRONEER-IPF ethnicity from prescribing information; all other FIBRONEER-IPF values pooled across arms (N=1177) from Richeldi L, et al, N Engl J Med, 2025.

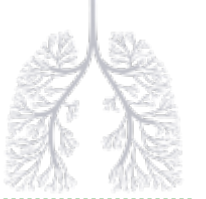
FIBRONEER-IPF: Richeldi L, et al. Nerandomilast in Patients with Idiopathic Pulmonary Fibrosis. N Engl J Med. 2025; JASCAYD® (nerandomilast) Prescribing Information.

Boehringer Ingelheim Pharmaceuticals, Inc. Revised August 2026.

TETON-1 & 2: Nathan SD, et al. Inhaled Treprostinil for Idiopathic Pulmonary Fibrosis. N Engl J Med. 2026

ALOFT-IPF: Maher TM, et al. Baseline Characteristics of Patients in the ALOFT-IPF Phase 3 Trial of Admiparant in IPF. Am J Respir Crit Care Med. 2026

Vicore's partnership with Nippon Shinyaku for buloxibutid in Japan



Partnership Overview

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

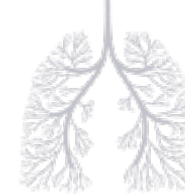
Financial Terms

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.

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.



HANS JEPSSON, PhD
CHIEF FINANCIAL OFFICER

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



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.



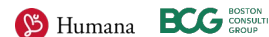
ROBERT SLACK, PhD, FRSB
CHIEF SCIENTIFIC OFFICER

Former VP of respiratory biology at GSK; Former CSO at Galecto. 25 years of experience in drug development.



MIKAEL NYGÅRD, PhD
CHIEF OPERATING OFFICER

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



HELEN BARKER
CHIEF TECHNOLOGY OFFICER

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



JIMMIE HOFMAN
CHIEF BUSINESS OFFICER

Business Development executive with extensive deal-making experience.



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.

ELISABETH BJÖRK, MD, PhD

Broad drug development experience, formerly 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.

PETER GUENTER

Nearly four decades of global pharmaceutical leadership experience (e.g., former CEO of Merck Healthcare, former CEO of Almirall).



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