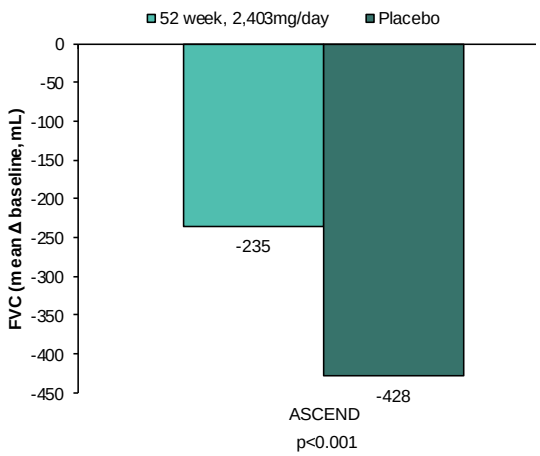
EXHIBIT 37: **Summary of clinical trials of Esbriet**

	Phase	Patients	Treatment	Comments on results
Open label trial	Phase 2	n=54	40mg/kg (max 3,600mg/day)	Study lacked placebo group; 1 and 2 year survival of 78% and 63%
Open label Japanese trial	Phase 2	n=10 with advanced IPF		No significant deterioration of the disease after 1 year; survival in 2 years not significantly changed
Multi-centre, randomised and placebo controlled trial	Phase 2	n=207 randomised 2:1 treatment:placebo	600mg 3 times per day or placebo	Did not meet 12m end point; terminated at 9m due to acute exacerbations in placebo; Slower decline of FVC in treatment group (-0.03L vs -0.13L, p=0.0366)
Double-blind, randomised, placebo-controlled Japanese trial	Phase 3	n=?? randomised 2:1:2 high dose:low dose:placebo	1,800mg per day, 1,200mg per day or placebo	Criticised for change of initial end point plus concerns about the handling of missing values in statistical analysis Met primary end point FVC change baseline to 52 weeks: high dose -0.09L vs placebo -0.16L (p=0.04) Progression free survival significantly longer for high dose (p=0.02)
CAPACITY-1	Phase 3	n=435 randomised 2:1:2 high dose:low dose:placebo	2,403mg/day, 1,197mg/day or placebo	Met primary end point change of FVC% predicted from baseline to wk72: high dose mean -8.0% vs placebo -12.4% (p=0.001)
CAPACITY-2	Phase 3	n=344	2,403mg/day or placebo	Did not meet primary end point change of FVC% predicted from baseline to wk72: high dose mean -9.0% vs placebo -9.6%; placebo group had greater proportion of patients with obstructive airway disease which is associated with reduced FVC decline. Significance was reached in 6MWT distance (absolute difference 31.8m)
CAPACITY pooled analysis				Reduced decline of FVC% predicted: high dose mean -8.5% vs placebo -11% (p<0.005); 31% reduced decline in 6MWT distance (p<0.001); 26% reduction in risk of death or disease progression (HR 0.74, p=0.025)
ASCEND	Phase 3	n=555 randomised 1:1 treatment:placebo	2,403mg/day or placebo	Met primary end point change of FVC% predicted from baseline to wk52: treatment mean -235mL vs placebo -428mL (p<0.001) Also reduced risk of death or disease progression, increased the proportion of patients who had no decline in FVC% predicted, reduced the decline of distance walked during 6MWT, improved progression-free survival

Source: Margaritopoulos et al (2016) Core Evidence (link)

EXHIBIT 38: **ASCEND FVC change**



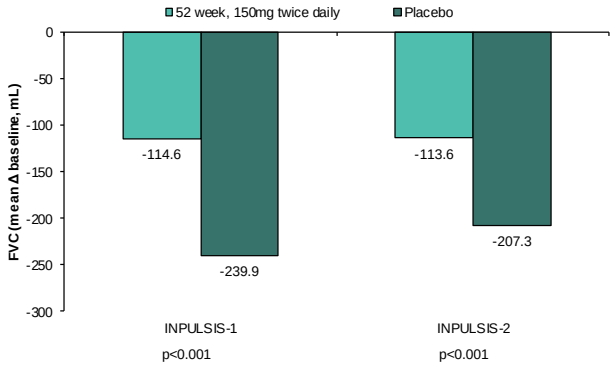
Source: Margaritopoulos et al (2016) Core Evidence (link)

EXHIBIT 39: **Summary of adverse events (ASCEND)**

	ASCEND	
	Pirfenidone (n=278)	Placebo (n=277)
Adverse events leading to discontinuation	40 (14.4)	30 (10.8)
Examples of common adverse events affecting a higher proportion of patients in the pirfenidone groups:		
Nausea	100 (36.0)	37 (13.4)
Rash	78 (28.1)	24 (8.7)
Dizziness	49 (17.6)	36 (13.0)
Dyspepsia	49 (17.6)	17 (6.1)
Anorexia	44 (15.8)	18 (6.5)
Vomiting	36 (12.9)	24 (8.7)
Weight loss	35 (12.6)	22 (7.9)
Gastroesophageal reflux	33 (11.9)	18 (6.5)
Insomnia	31 (11.2)	18 (6.5)
Grade 3 adverse events:		
Gastrointestinal	15 (5.4)	4 (1.4)
Skin	5 (1.8)	1 (0.4)
Serious adverse events*	55 (19.8)	69 (24.9)
Fatal adverse events	11 (4.0)	20 (7.2)

Source: King et al (2014) N Engl J Med. (link). Note serious adverse events includes worsening of IPF

EXHIBIT 40: **Primary endpoint: annual decline in forced vital capacity (FVC)**



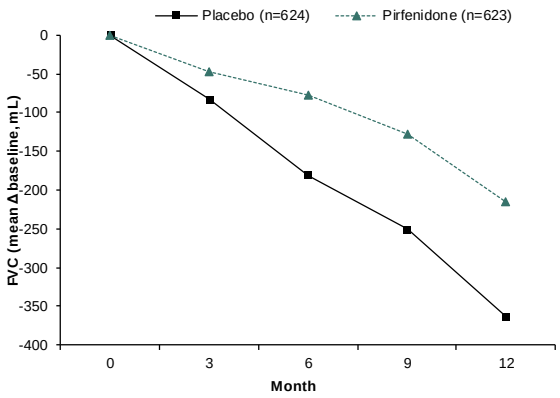
Source: Richeldi et al (2015) N Engl J Med (link)

EXHIBIT 41: **Adverse events: Ofev phase 3 clinical trials (number of patients, percent)**

	INPULSIS-1		INPULSIS-2	
	Nintedanib (n=309)	Placebo (n=204)	Nintedanib (n=329)	Placebo (n=219)
Any adverse event	298 (96.4)	181 (88.7)	311 (94.5)	198 (90.4)
Adverse events leading to discontinuation of treatment	65 (21.0)	22 (10.8)	58 (17.6)	33 (15.1)
Examples of common adverse events affecting a higher proportion of patients in the nintedanib groups:				
Diarrhea	190 (61.5)	38 (18.6)	208 (63.2)	40 (18.3)
Nausea	70 (22.7)	12 (5.9)	86 (26.1)	16 (7.3)
Decreased appetite	26 (8.4)	14 (6.9)	42 (12.8)	10 (4.6)
Vomiting	40 (12.9)	4 (2.0)	34 (10.3)	7 (3.2)
Weight loss	25 (8.1)	13 (6.4)	37 (11.2)	2 (0.9)
Severe adverse events	81 (26.2)	37 (18.1)	93 (28.3)	62 (28.3)
Serious adverse events	96 (31.3)	55 (27.0)	98 (29.8)	72 (32.9)
Fatal adverse events	12 (3.9)	10 (4.9)	8 (2.4)	18 (8.2)

Source: Adapted from Richeldi et al (2015) N Engl J Med (link)

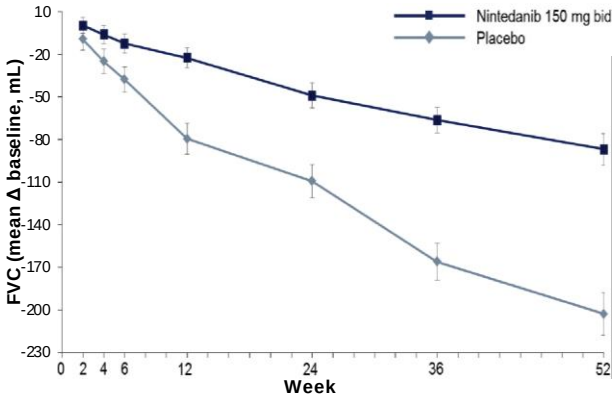
EXHIBIT 42: **Esbriet pooled analysis: ASCEND and CAPACITY studies**



Absolute difference (mL)	36	104	123	148
Relative difference %	43.5%	57.3%	49.1%	40.7%
Rank ANCOVA p-value	<0.001	<0.001	<0.001	<0.001

Source: Noble et al (2015) Eur Respir J, Bernstein analysis (link)

EXHIBIT 43: **Ofev pooled analysis: TOMORROW and INPULSIS studies**



Source: Richeldi et al (2016) Resp Med (link). Bernstein analysis

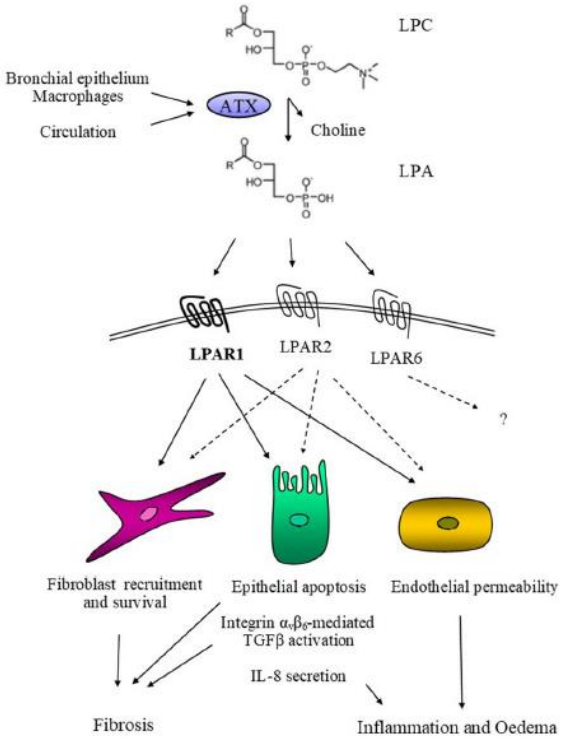
Galapagos' candidate drugs in IPF

Galapagos have a broad IPF portfolio of assets with differing mechanisms but only one asset (GLPG1690) has demonstrated proof of concept and that is where we focus our attention.

GLPG1690 is a selective autotaxin (ATX) inhibitor. In pulmonary fibrosis, ATX levels rise in the broncheoalveolar fluid, and increased ATX activity has been detected in a range of inflammatory and fibroproliferative diseases in the lung, kidney and skin. ATX is the enzyme responsible for generating lysophosphatidic acid (LPA), with LPA being formed locally in areas with increased ATX levels and acting locally via its receptors.

In the lung, LPA signalling via LPAR1, and possibly via LPAR2, activates G-protein-mediated signal transduction cascades (Exhibit 44). Apoptosis is triggered in epithelial cells, which in modelled pulmonary fibrosis is the initiating pathogenic event. Epithelial cells are also induced to secrete IL-8, which is both proinflammatory and stimulates permeability of endothelial cells, thus promoting pulmonary oedema. LPA has several effects on fibroblasts: it is a chemotactic factor that promotes fibroblast recruitment, while also being a stimulator of fibroblast activation (via TGF beta) and promotor of fibroblast survival.

EXHIBIT 44: **Schematic: role of autotaxin in pulmonary fibrosis**

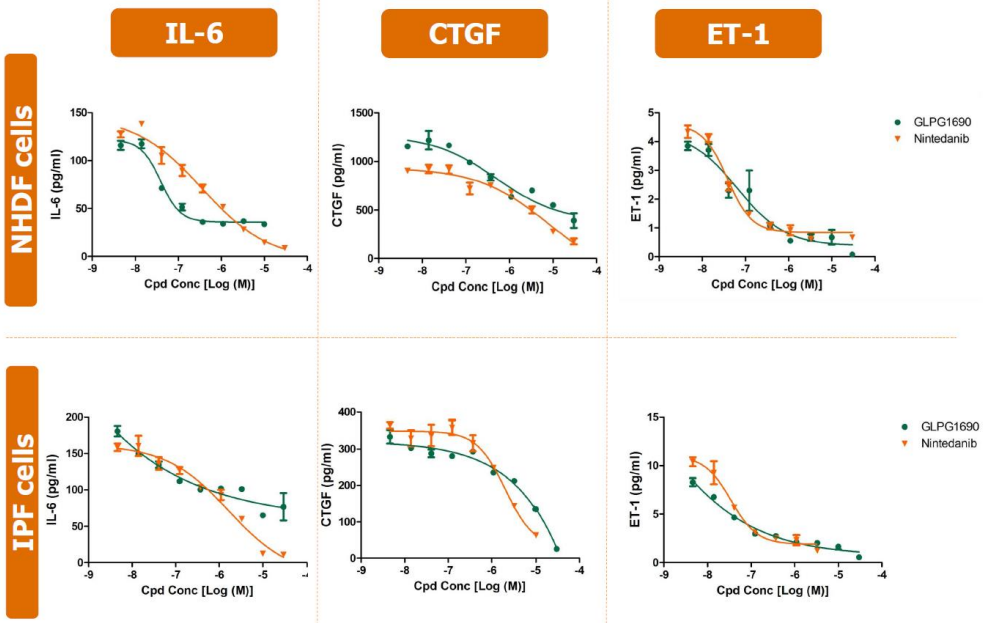


Source: Ninou et al (2018) Front. Med. (link)

It should be noted that LPA signals through at least six receptors (including LPAR1 and LPAR2) which are expressed differentially across a wide range of tissues and with overlapping specificities. While GLPG1690 targets autotaxin (thereby reducing LPA production more generally, and reducing the effects of LPA through any of its receptors), LPAR1 is also being considered as a potential target for IPF treatment (e.g., BMS-986020 is an LPA receptor antagonist being developed by BMS that has completed p2 trials for IPF, although perhaps unsuccessfully given results have not been released). The choice to target autotaxin might lead to unintended consequences; for example, LPAR2 is thought to protect against excessive innate immune responses to tissue injury, so targeting ATX might reduce this protective effect. However early clinical trials (discussed below) do not seem to suggest that GLPG1690 has unacceptably high rates of adverse events.

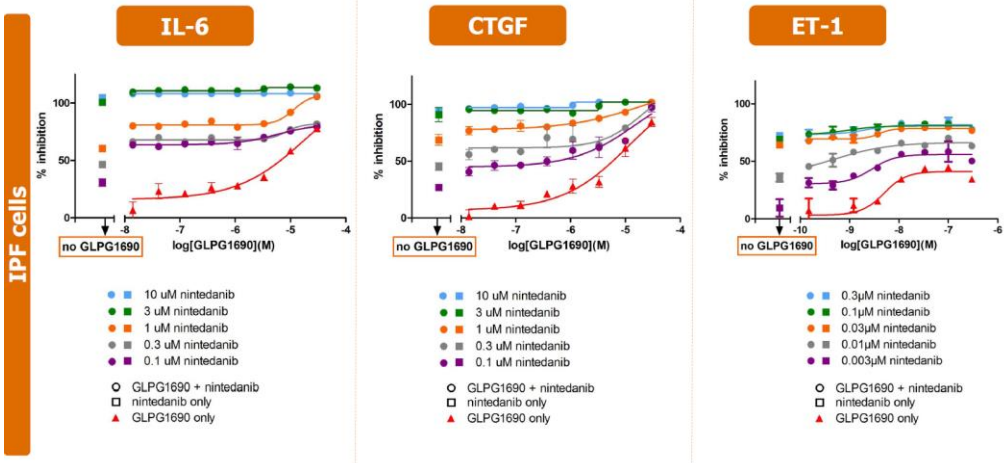
Earlier clinical and pre-clinical data was supportive for further testing. A couple of quick points that supported progress into p2. (i) Pre-clinical data demonstrates dose-dependent reductions of several TGF-beta induced pro-fibrotic mediators like ET-1, IL-6 and CTGF (Exhibit 45). When combined with Ofev, the added inhibitory effect can be seen (Exhibit 46). Please ask for more pre-clinical data if interested. (ii) p1 study demonstrated dose dependant reductions in plasma LPA18:2, a biomarker for autotaxin inhibition (Exhibit 47) with *in vivo* IC_{50} for reduction LPA18:2, in line with *ex vivo* IC_{50} (Exhibit 48; good correlation between PK and PD for LPA reduction).

EXHIBIT 45: **Effect of Ofev and GLPG1690 on TGF-beta induced IL-6, CTGF and ET-1**



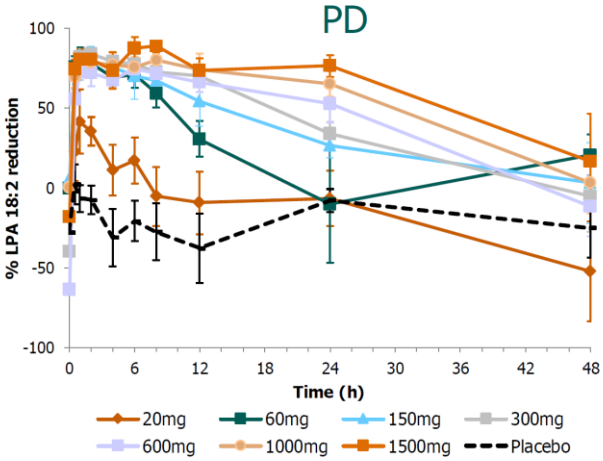
Source: Galapagos

EXHIBIT 46: **Combined effects of GLPG1690 and nintedanib**



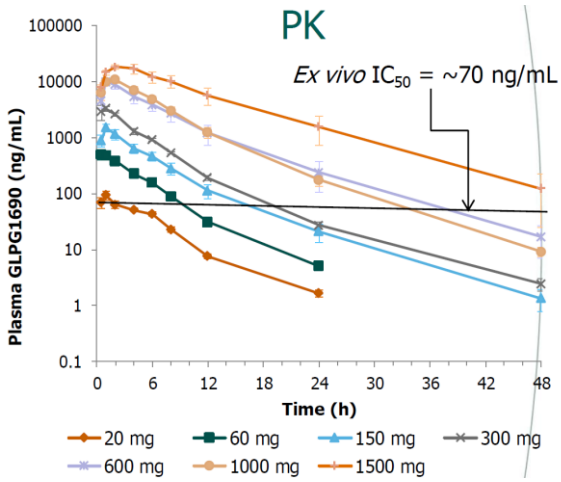
Source: Galapagos

EXHIBIT 47: **Dose-dependent reduction of LPA18:2 in plasma from healthy volunteers by GLPG1690**



Source: Galapagos

EXHIBIT 48: **60 mg dose is first dose with plasma concentrations durable above the ex vivo LPA18:2 IC50**



Source: Galapagos

Phase 2a FLORA efficacy results were encouraging (link), we just wish it were a little bigger. The 12-week study involved 23 IPF patients (centrally confirmed) who had not been receiving Ofev or Esbriet 4 weeks prior to entering the study and no exacerbations of IPF 6 weeks prior to entering the study (17 patient on 600mg GLPG1690 daily, 6 placebo). The baseline duration of IPF was higher in the drug group (1.9 years vs. 1 year) but baseline FVC was similar, albeit better in drug arm (2.8L vs. 2.7L, 75.3% of predicted normal vs. 69.7%). We asked physicians if this could possibly have skewed the data set and ultimately the answer was mixed (year 1 vs. year 2 is not a big deal but day 1 vs. year 3 would be).

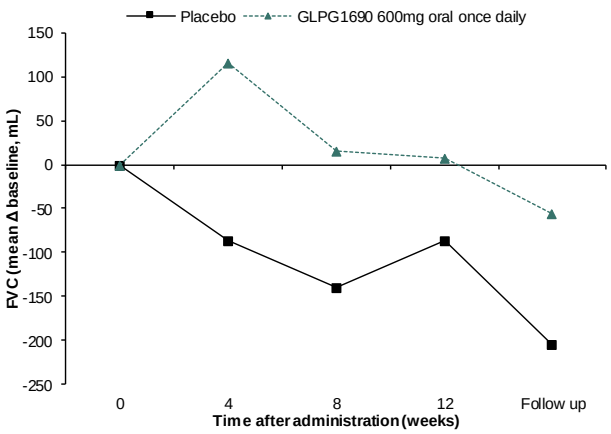
The mean time to C_{max} was 4.0 hours for GLPG1690 and while no formal analysis was done of the GLPG1690 trough, visual inspection showed week 1 sample concentrations consistent with previous studies where trough concentrations were established 4 days after the first dose. The FLORA study also examined reductions in plasma LPA 18:2, which supported the findings from earlier studies (Exhibit 50), with PK and PD data similar to healthy volunteers and target engagement demonstrated through plasma LPA 18:2 reduction.

Importantly, proof of concept was met. FVC increased 8mL with treatment at 12 weeks vs. a decrease of 87mL with placebo (Exhibit 49, observed-case analysis; comparable results for LOCF where FVC increased 25mL vs. a 70mL decrease with placebo). Functional respiratory imaging (FRI) confirmed disease stabilization. Mean change from specific airway volume was significantly lower in the treatment group (0.079mL/L vs. 3.038mL for placebo, p=0.0137).

There were no significant differences in quality of life as assessed by self-reported St George's Respiratory Questionnaire (SGRQ). A mean reduction of 5-8 points was taken to be a clinically important improvement (based on previous estimates of the minimum important difference in IPF). The mean changes from baseline to 12 weeks were: -5.45 in the symptom domain (vs. +2.90 for placebo), -2.32 in the activity domain (vs. +4.14 for placebo) and +3.22 in the impact domain (vs. -3.90 for placebo).

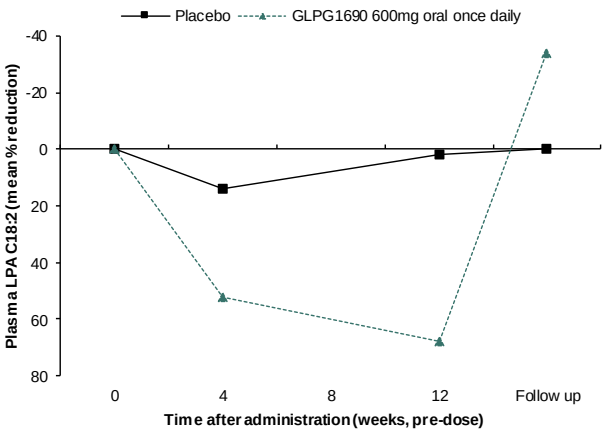
In short, a stabilisation of FVC at 12 weeks is a very good outcome, albeit from a very short, small population study.

EXHIBIT 49: **Stabilised FVC**



Note: Week 8 timepoint $p<0.05$; data is observed-case analysis
Source: Maher et al, The Lancet Respiratory Medicine 2018 (link), Bernstein analysis

EXHIBIT 50: **Sustained reduction in plasma LPA**



Source: Maher et al, The Lancet Respiratory Medicine 2018 (link), Bernstein analysis

Safety profile also encouraging. TEAEs were reported in 4 (67%) and 11 (65%) of patients in the placebo and treatment groups respectively, with most AE being mild to moderate in severity (Exhibit 51). Two patients had AEs deemed related to treatment, although it is not disclosed what these events were. Of the serious events, two affected patients were in the placebo group, and one affected patient in the treatment group had cholangiocarcinoma (bile duct cancer) symptoms that were noted 1 day after the first dose of treatment but which had also occurred during screening. No patients died or had acute IPF exacerbations. The most common types of adverse events are shown in Exhibit 52. The most common type of adverse event was infections and infestations, but these occurred in a similar proportion of patients in the treatment (41%) and placebo (50%) groups.

EXHIBIT 51: **Treatment emergent adverse events (number of patients, percent)**

	FLORA	
	GLPG1690 (n=17)	Placebo (n=6)
Any adverse event	11 (65)	4 (67)
Mild	4 (24)	0
Moderate	6 (35)	3 (50)
Severe	1(6)	1 (17)
Serious events	1 (6)	2 (33)
Events resulting in death	0	0
Events related to treatment	2 (12)	0
Events leading to discontinuation of study drug:		
Temporary discontinuation	2 (12)	0
Permanent discontinuation	1 (6)	1 (17)

Source: Maher et al, The Lancet Respiratory Medicine 2018 (link), Bernstein analysis

EXHIBIT 52: **Most frequent adverse events**

	FLORA			
	GLPG1690 (n=17)		Placebo (n=6)	
	Patients (%)	*# events*	*Patients (%)*	*# events*
Infections and infestations	7 (41)	10	3 (50)	8
Respiratory, thoracic, and mediastinal disorders	4 (24)	8	2 (33)	4
Gastrointestinal disorders	2 (12)	2	2 (33)	2
Musculoskeletal and connective tissue disorders	1 (6)	1	2 (33)	6
Cardiac disorders	0	0	1 (17)	2
Renal and urinary disorders	0	0	1 (17)	3
Vascular disorders	0	0	1 (17)	1
General disorders and	2 (12)	2	1 (17)	1
Investigations	2 (12)	2	1 (17)	1

Source: Maher et al, The Lancet Respiratory Medicine 2018 (link), Bernstein analysis

The product compares well vs. approved products. As we have previously stated, cross trials comparisons in IPF are a challenge. Even more so given the fact that the GLPG1690 study (i) was over a shorter period of only 12 weeks vs. +52-weeks for Esbriet/Ofev, (ii) only recruited 17 drug treated patients vs. hundreds for Esbriet/Ofev, (iii) recruited a slightly different patient population, (iv) had different endpoints (Exhibit 53).

We have to take 17 patient data with a pinch of salt but looking at the 12-week data for both Esbriet and Ofev, (i) neither were able to demonstrate any form of improvement in FVC, which GLPG1690 did, (ii) both have inferior tolerability profiles, particularly GI, and (iii) both have inferior dosing schedules.

EXHIBIT 53: **Comparisons of FLORA study to Esbriet/Ofev studies**

	GLPG1690	Esbriet (pirfenidone)		Ofev (nintedanib)	
Study names	FLORA (P2)	CAPACITY-1 and 2	ASCEND	TOMORROW	INPULSIS-1 and 2
Phase	P2	P3	P3	P2	P3
Total patients	23	1,247		1,231	
Drug-treated patients	17	623		723	
Study duration	12 weeks	72 weeks	52 weeks	52 weeks	52 weeks
Primary endpoint(s)	Safety (adverse events), tolerability, PK & PD	Change in % predicted FVC	Absolute change in % predicted FVC	Rate of decline in FVC	Rate of decline in FVC
Summary of other endpoints	Changes in pulmonary function (spirometry), biomarkers, HRCT images, QoL measures	Absolute change in % predicted FVC, progression-free survival, 6MWT, SpO2, DLCO, dyspnea score, worsening of IPF	n/a	Absolute/relative changes in FVC% predicted and FVC, survival, SpO2, PaCO2, DLCO, dyspnea, 6MWT, FEV1/FVC, SGRQ scores, lung capacity measures, exacerbations, time to progression	As per TOMORROW, plus time to death or transplant and additional questionnaires (e.g., SOBQ, CASA-Q, PGI-C, EQ-5D)
Patient population:					
IPF diagnosis confirmation	Centrally confirmed	'Confident' local diagnosis	Centrally confirmed	Centrally confirmed	Centrally confirmed
IPF diagnosis duration	n/a	n/a	6-48 months	<5 years	<5 years
Patient age	≥40 years	Between 40 and 80	Between 40 and 80	>40 years	>40 years
% FVC	≥50%	≥50%	Between 50% and 90% inclusive	>50%	≥50%
% carbon monoxide diffusing capacity (% DLCO)	≥30%	Between 35% and 90% inclusive	Between 30% and 90% inclusive	Between 30% and 79% inclusive	Between 30% and 79% inclusive
FEV1/FVC ratio	≥0.7	n/a	≥0.8	n/a	≥0.7

Source: Company disclosure, Bernstein analysis and estimates

Phase 3 will start shortly. The ISABELA p3 trials (1 & 2) are expected to start recruiting before year end. The programme will consist of two identical trials: ISABELA 1 and ISABELA 2 with a total of 1,500 IPF patients. These patients will remain on their current standard of care (which may include Esbriet or Ofev) and randomised to one of two doses or placebo. The primary endpoint will be the change in FVC (in mL) at 52 weeks. The studies will also look at hospitalisations, mortality, quality of life, and safety/tolerability. All patients to be treated until last patient passes the 52-week milestone – meaning that for a subset of patients the study will collect longer term data. We will likely need to wait until 2020 to see the outcome from the study, although in interim is possible earlier.

The general consensus view from physicians is that Galapagos have an ambitious plan as it will highlight if there is any additional benefit when combined with existing treatment options. With a very heterogenous patient pool, sub-population analysis may not read well. Regardless, demonstrate benefit and the product will be used either alone or in combination with Esbriet or Ofev.

Galapagos has other less-developed candidates in IPF. Only GLPG1690 has been tested in clinical trials, however Galapagos do highlight some other molecules in their pipeline for IPF. Very limited information is available about their mechanism of action and performance in pre-clinical studies.

+ **GLPG1205** is a GPR84 inhibitor. G-protein coupled receptor 84 is a fatty acid receptor that is highly expressed on bone marrow cells, splenic T and B cells and circulating granulocytes, monocytes and macrophages, although in the latter cell types its expression depends on upregulation in response to inflammatory conditions. GPR84 is also expressed in many organs, including the lung. The role of GPR84 is not yet well characterised, however it is known to be upregulated by lipopolysaccharide and by Staphylococcus enterotoxin B, and to enhance the induction of IL-12 (which supports Th1 helper T cell responses) and IL-8 (a chemokine expressed by macrophages, epithelial cells, endothelial cells and airway smooth muscle cells). IL-8 is known to play a role in IPF: serum levels of IL-8 are elevated in patients with IPF and correlates with disease activity (link) and mediates fibrogenic mesenchymal progenitor cells (MPCs) in IPF (link).

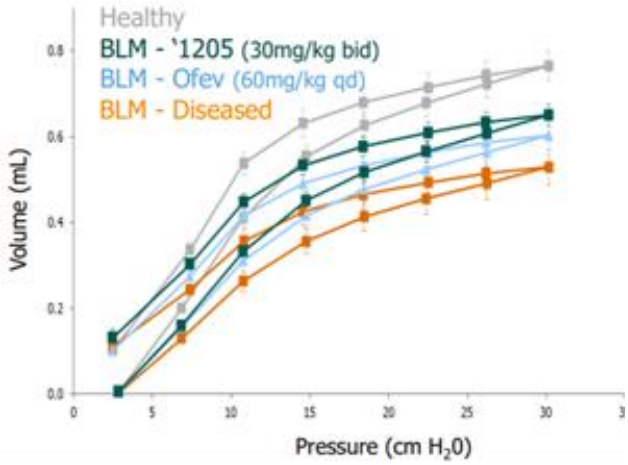
  GPR84 has typically been associated with metabolic and inflammatory disorders but studies in mouse models of fibrosis have shown that GPR84 also plays a role in fibrotic disease in a range of tissues. For example, *Gpr84* knockout mice have reduced kidney fibrosis in an adenine-induced nephropathy model, and treatment with PBI-4050 (known to be both an agonist of GPR40 and an inhibitor of GPR84) reduces lung fibrosis in a bleomycin mouse model (link).

Pre-clinical studies of GLPG1205 have been promising. In a bleomycin mouse model GLPG1205 seems to provide better improvement of respiratory capacity vs Ofev (Exhibit 54). Human trials of this candidate in ulcerative colitis demonstrated tolerability but no effect. The PINTA p2 trial (link, not yet listed) is expected to start shortly. It will test 100mg once daily oral for 26 weeks in 60 IPF patients. Galapagos seem excited by GLPG1205, suggesting it is a very potent and effective molecule.

+ **GLPG2384** has been nominated for an undisclosed indication but in early press releases Galapagos described it as a 'back up' for GLPG1205. Given GLPG2384 is also an antagonist for GPR84, it is possible that this might be an IPF candidate.

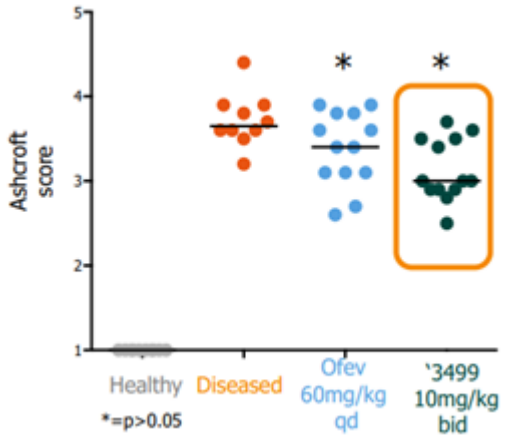
+ **GLPG3499** has been nominated for IPF but it has an undisclosed mechanism of action. Data in a bleomycin mouse model suggest reduction in fibrotic scores and numerical advantage over Ofev. It is expected to enter phase I in 2018.

EXHIBIT 54: **GLPG1205 - Inspiratory capacity**



Source: Galapagos

EXHIBIT 55: **GLPG3499 – Signs and symptoms score**



Source: Galapagos

Competitive pipeline in IPF is pretty full

The recent shift in understanding of the pathology of IPF as a disease driven by epithelial damage and dysregulated fibrotic repair (rather than driven predominantly by inflammation) has opened a new, wider range of targets for potential candidates (including those in Galapagos' pipeline). There are several competitor candidates (summarised in Exhibit 56) with varied mechanisms of action and with promising clinical results.

EXHIBIT 56: **Competitor drug candidates in IPF**

Candidate	Company	Mechanism	Potential indications	Development stage
FG-3019 (Pamrevlumab)	FibroGen	Anti-connective tissue growth factor antibody. CTGF is a mediator of fibrotic diseases in many tissues (lung, heart, liver, skin, kidney)	IPF, pancreatic cancer, Duchenne Muscular Dystrophy	Phase 2b showed significant slowing of IPF progression in terms of lung function (FVC) and fibrosis (QLF) Phase 3 not yet announced
PBI-4050	Prometic Life Sciences	Candidate activates GPR40 and suppresses GPR84 (note MoA overlap with GLPG1205)	IPF, Alström syndrome, metabolic syndrome and chronic kidney disease associated with type 2 diabetes	Open label Phase 2 demonstrated it can prevent lung fibrosis as monotherapy or in combination with nintedanib (but not pirfenidone due to likely drug-drug interactions) Phase 3 pivotal trial approved by FDA (Feb 2018)
PRM-151	Promedior	Recombinant human pentraxin 2 (serum amyloid P) reduces fibrosis by inhibiting innate immune responses	IPF, myelofibrosis, other systemic fibrotic diseases	Phase 2 showed significant slowing of decline in lung function (FVC % predicted) and reduced change in 6MWD.
KD025	Kadmon Therapy	Inhibits Rho-associated coiled coil kinase (ROCK2)	IPF, chronic graft-versus-host disease, psoriasis	Phase 2 open label showed reduction in FVC decline at 24 weeks (-68mL vs -173mL for placebo). Trial is still recruiting patients and patients can elect to extend to 96 weeks
CC-90001	Celgene	JNK1 inhibitor	IPF	Phase 1b showed mean improvements in FVC from baseline in 200/400mg groups; phase 2 recruiting, estimated SC Jun 2022
Bardoxolone Methyl	Reata Pharmaceuticals	Activates Nrf2, a regulator of oxidative damage, inflammation and fibrosis	IPF, pulmonary arterial hypertension, sarcoidosis	Phase 2 study of idiopathic lung disease (including 8 patients with IPF) showed improvement for IPF patients in 6MWD test; study did not examine lung function outcomes
RP5063	Reviva Pharmaceuticals	Modulates dopamine and serotonin receptors; with dysfunctional serotonin signalling associated with fibrosis in the lungs	IPF, PAH, schizophrenia and other neurological diseases	Phase 2 in IPF and pulmonary arterial hypertension (PAH) intended to be launched soon based on pre-clinical data in a bleomycin mouse model
SM04646	Samumed	Inhibits Wnt/beta-catenin pathway, which activtes proinflammatory cytokine response by alveolar epithelium	IPF	Pre-clinical data shows stronger effect than either pirfenidone or nintedanib; Phase 1 completed in healthy subjects; Phase 1 study in IPF patients given multiple ascending doses is ongoing
BG00011 (STX-100)	Biogen	Anti-avb6 integrin	IPF	Phase 2 just announced, SC expected Nov 2021
VAY736	Novartis	Anti-BAFF receptor	IPF	Phase 2 recruiting, SC expected Apr 2022
Tipelukast	MediciNova	Leukotriene antagonist; PDE4 and thromboxane A2 inhibitor	IPF	Phase 2 SC expected Dec 2018
TD139	Galecto Biotech	Galectin-3 inhibitor		Phase 1/2a study completed; phase 2b dose ranging study not yet registered
ART-123	Asahi Kasei	Human recombinant thrombomodulin inhibits coagulation by accelerating thrombin activation of protein C	Acute exacerbation of IPF Already approved in Japan for disseminated intravascular coagulation	Phase 3 study underway, but focusing on mortality post-acute exacerbations rather than impact on IPF disease progression

Source: Company websites, clinicaltrials.gov, press reports, Bernstein analysis

FibroGen's Pamrevlumab (FG-3019) is probably the name that comes up most during our discussions with investors and is considered the biggest threat to GLPG1690. The product is a fully human monoclonal antibody that inhibits connective tissue growth factor (CTGF). CTGF is a mediator of fibrotic disease, promoting the production of collagen and fibronectin while also inhibiting the metalloproteinases that normally mediate the breakdown of extracellular matrix components.

In a mouse model of fibrosis, FG-3019 treatment altered the expression of fibrosis-related genes in the lungs, including >3 fold reductions in the levels of fibronectin, CTGF, lysyl oxidase and collagen 1alpha1. This was then tested in the clinic and the p2a was published in 2016 with positive results (link). The challenge with the study being open label and lack of placebo arm control arm.

FibroGen followed this with the PRAISE (NCT01890265) p2b study where 103 patients received either 30mg/kg of pamrevlumab or placebo IV every 3 weeks. The study achieved the primary endpoint with treated patients having significantly