STIFEL

Galapagos NV

GLPG – NASDAQ; GLPG – NA

August 11, 2017

Biotechnology

BUY

COMPANY UPDATE

Upgrading to Buy on Positive GLPG1690 P2 Results, Reduced CF Risk; TP to $101

Financial Summary

Changes	Previous	Current
Rating	Hold	Buy
Target Price	$83.00	$101.00
FY17E EPS	—	€(2.83)
FY18E EPS	—	€(3.50)
FY17E Revenue	—	€99.7
FY18E Revenue	—	€78.2

Price (08/10/17):	$81.06
52-Week Range:	$95 - $53
Market Cap.(mm):	3,749.5
Shr.O/S-Diluted (mm):	46.3
Avg Daily Vol (3 Mo):	194,193
Dividend / Yield:	$0.00 / 0.0%

Revenue	2016A	2017E	2018E
FY (Dec)	€151.6A	€99.7	€78.2
EPS	€1.14A	€(2.83)	€(3.50)

Price Performance

Summary

We are upgrading Galapagos to Buy from Hold and increasing our TP to $101 from $83 based on: 1) what we view as clearly positive early P2 POC results for wholly-owned GLPG1690 in idiopathic pulmonary fibrosis (IPF) – which compels us to include the drug in our model at a very reasonable 10% probability of success (POS), and; 2) reduced downside risk associated with Galapagos' CF triple combo - as we believe recently announced delay of P2 launch and positive Vertex triple results have brought Street views in line with our 15% POS. In addition, positive GLPG1690 results increase our confidence in the company's target discovery platform. With ~$1.5B in cash, Galapagos remains well-positioned to capitalize on its internal discovery efforts, in our view.

Key Points

FLORA study design: FLORA was a 23-patient (17 GLPG-1690 and 6 placebo), 12-week, exploratory, randomized, double-blind, placebo-controlled trial evaluating a 600mg daily oral dose of GLPG1690 vs. placebo. Primary endpoints were safety, tolerability, PK and PD. Secondary endpoints included FVC, changes in disease biomarkers, functional respiratory imaging (FRI), and quality of life. Baseline characteristics were in line with published data and mostly balanced between drug and placebo. Nintedanib or pirfenidone were discontinued at least 4 weeks prior to GLPG1690 treatment.

Compelling FVC results: Over the 12-weeks, GLPG1690 patients demonstrated an impressive mean FVC **increase** from baseline of 8 mL, while placebo patients showed a mean decrease of 87 mL (in line with prior studies). As a point of reference, currently approved treatments show a decrease of ~30mL over the same treatment period. We view the FVC increase in GLPG1690 patients as unique and compelling. While the result was not statistically significant, the trends were strong in a trial not powered for statistics. In addition, sensitive functional respiratory imaging (FRI) confirmed disease stabilization in the GLPG1690 arm, versus disease progression in the placebo arm, reaching statistical significance on two specific parameters.

Good safety: The drug was generally well tolerated, with one SAE in the drug arm leading to discontinuation - a patient who developed cancer two days into the trial - unrelated to treatment in our view. Overall rates of discontinuation due to AEs and SAEs were similar between GLPG1690 and placebo arms.

Biomarker reductions: GLPG1690-treated patients demonstrated steep reductions of serum LPA18:2, a biomarker for autotaxin inhibition - confirming target engagement and the drug's MOA.

Some caveats: While we view the GLPG-1690 results as clear POC in IPF, as with any exploratory trial there are many caveats - in this case, small patient numbers, short trial duration, minor imbalances in baseline characteristics, and a few excluded values (due to either administration of bronchodilator therapy too close to FVC measurement, or insufficient quality of FVC measurement). Nevertheless, we view the overall data as strongly supportive of POC.

Next steps: Full results are likely to be presented in May 2018 at ATS in San Diego. Subsequent trials will likely include two potentially pivotal one-year studies exploring multiple doses both as monotherapy vs. placebo as well as on-top of currently approved treatments. Further info on trial design and endpoints will be provided following meetings with regulators.

Adding GLPG1690 to model: Based on what we view as strong POC for GLPG1690 in IPF, we are adding the drug/indication to our model. We forecast FY25 unadjusted WW sales of $1.07B ($107M probability-adjusted at 10% POS).

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Investment Thesis

We are bullish on the prospects for key pipeline asset filgotinib in multiple diseases. While we remain cautious on CF triple combo, we believe Street expectations are now aligned with our 15% POS which limits downside risk. Recent positive POC data for GLPG1690 in IPF compel us to include it in our model with 10% POS. The rest of the pipeline is early and we await additional clinical data to assess its value. Galapagos is well financed with ~$1.5B cash on the balance sheet.

P1b MOR106 (anti-IL-17c mAb) results in atopic dermatitis expected 3Q17: The study is being conducted in two parts: a SAD and MAD. The SAD includes seven cohorts (n=42) of healthy males receiving IV MOR106 over four weeks vs. placebo (n=14). The MAD portion includes three cohorts of subjects with moderate to severe atopic dermatitis (n=18) dosed by IV over four weeks vs. placebo (n=6). The primary and secondary objectives include safety, tolerability, and PK. Exploratory objectives include Eczema Area & Severity Index (EASI), Scoring Atopic Dermatitis (SCORAD), Investigator Global Assessment (IGA), Dermatology Quality of Life Index (DLQI), and effect on Thymus & Activation-Regulated Chemokine (TARC).

MOR106 is key potential driver: POC results due in 3Q17 provide significant optionality, in our view, as positive data would add a significant new leg to the story that we do not currently include in our model.

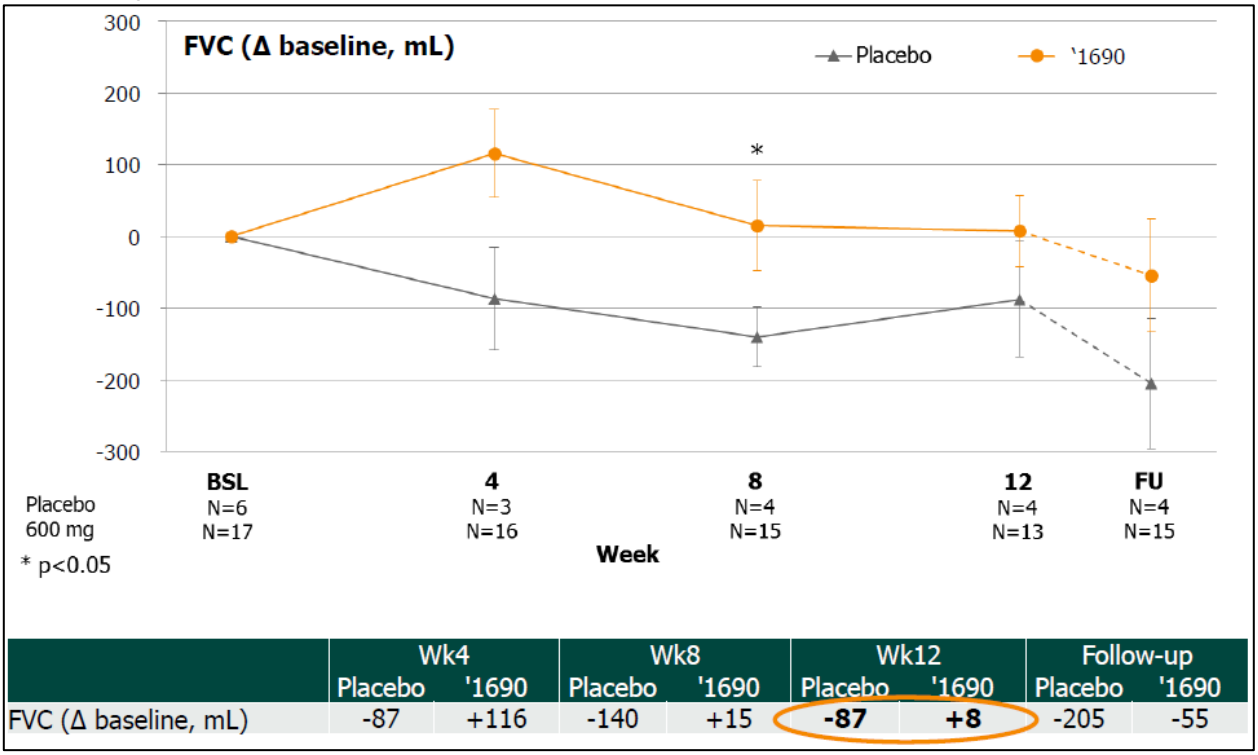
(continued below)

GLPG1690: Wholly-owned, novel approach to IPF

GLPG1690 is a first-in-class, oral, small-molecule, potent and selective inhibitor of autotaxin (ATX) enzyme in P2a development for idiopathic pulmonary fibrosis (IPF), a fatal lung disease affecting about 200,000 patients in the U.S. and EU. The Phase 2 top-line results (Aug 9th, 2017) showed that over the 12-week period, patients receiving GLPG1690 had an FVC increase of 8 mL, while patients on placebo arm showed an FVC reduction of 87 mL (mean from baseline). See Exhibit 1 for comparison of 12W data with the 2 marketed drugs, Nintedanib and Pirfenidone. In addition, sensitive functional respiratory imaging (FRI) confirmed disease stabilization in the GLPG1690 arm, in contrast to disease progression in the placebo arm (statistical significance on two specific parameters). Based on the mechanism of action of GLPG1690, patients on GLPG1690 treatment showed a clear reduction of serum LPA18:2, a biomarker for autotaxin inhibition. These positive data confirmed our view that GLPG1690 employs a novel mechanism of action and has the potential to make it a formidable competitor in the IPF space.

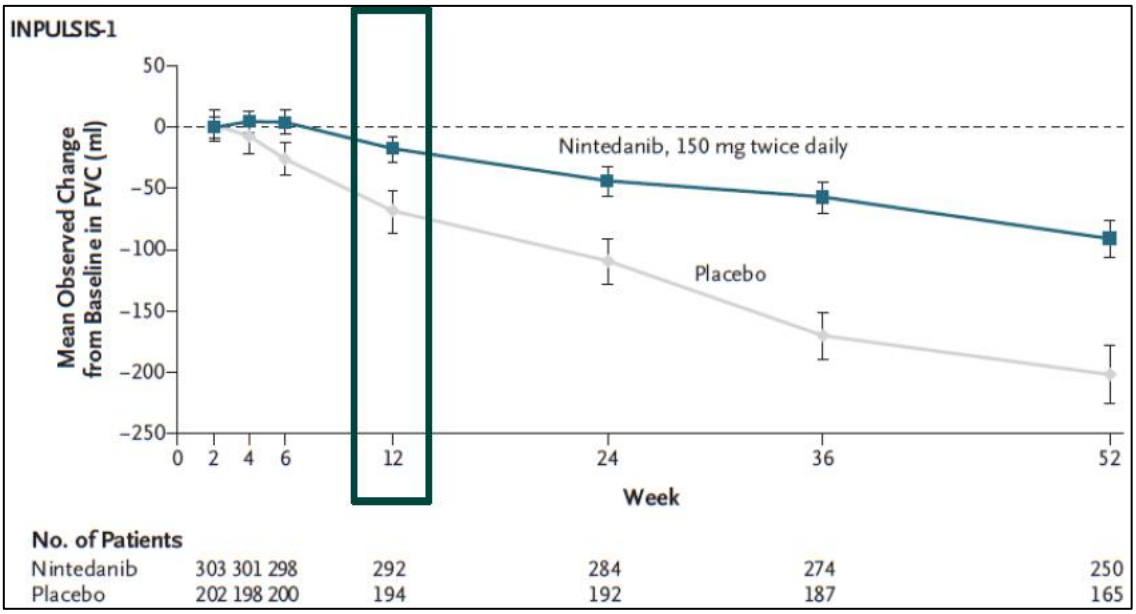
Exhibit 1: FVC data at week 12: GLPG1690 vs. Nintedanib vs. Pirfenidone

GLPG1690, Phase 2

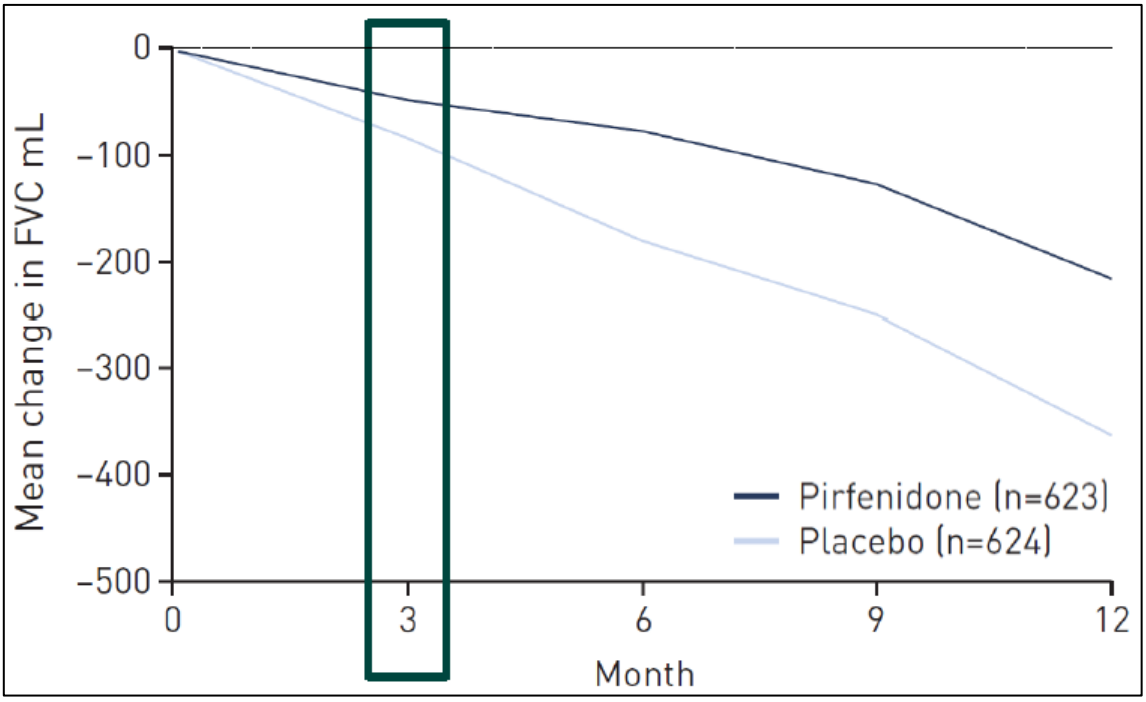


	Wk4		Wk8		Wk12		Follow-up	
	Placebo	'1690	Placebo	'1690	Placebo	'1690	Placebo	'1690
FVC (Δ baseline, mL)	-87	+116	-140	+15	**-87**	**+8**	-205	-55

Nintedanib, Phase 3



Pirfendione, Phase 3



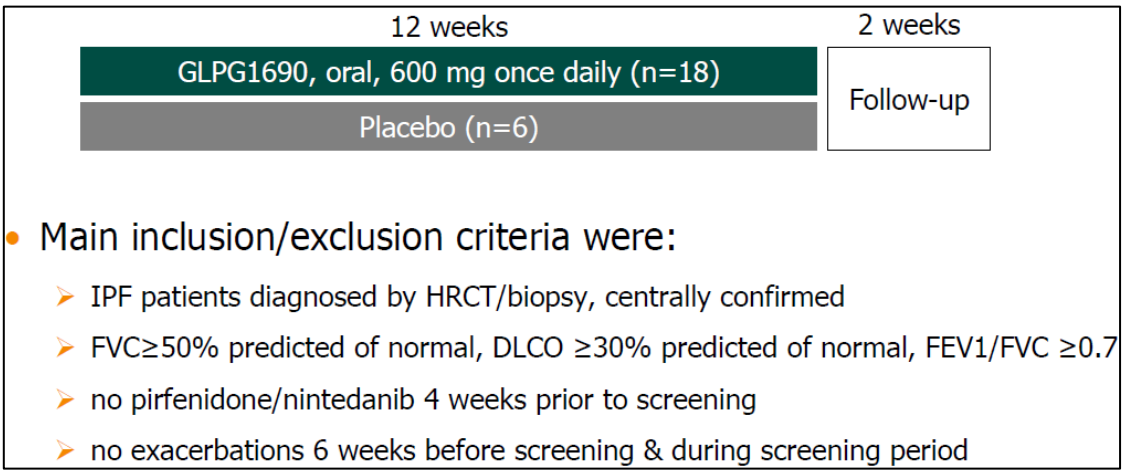
Source: Company reports

Of note, prior preclinical results in a mouse bleomycin model (predictive for IPF) demonstrated superiority to Esbriet (Pirfenidone) on both the Ashcroft fibrotic score and collagen content. Positive P1 results demonstrated target engagement and favorable safety and PK. GLPG1690 has US and EU orphan drug designation for IPF.

P2a FLORA Study

The study was conducted at 17 sites in U.K., Italy, and Ukraine. See Exhibit 2 for the study design and baseline characteristics. FLORA is a randomized, double-blind, placebo-controlled study investigating a once daily 600 mg oral dose of GLPG1690 administered for 12 weeks in 24 IPF patients diagnosed by centrally confirmed HRCT/biopsy. Patients taking pirfenidone or nintedanib four weeks prior to screening are excluded. Primary objectives include safety, tolerability, and PK/PD. Target engagement will be measured by LPA in plasma and bronchoalveolar lavage fluid (BALF), both at baseline and through 12 weeks of treatment. Other secondary endpoints include changes in pulmonary function assessed by spirometry, quality of life per the Saint George Respiratory Questionnaire, and exploration of the effect of GLPG1690 on functional respiratory imaging parameters derived from HRCT at baseline and week 12.

Exhibit 2: Flora trial design and baseline characteristics



Baseline disease characteristics (mean)	Placebo (N=6)	'1690 (N=17)	Total (N=23)
Duration of IPF (yrs)	1.0	1.9	1.7
DLCO (% predicted of normal)	40.6	37.8	38.6
Baseline FVC (L)	2.693	2.777	2.755
Baseline FVC (% predicted of normal)	69.7	75.3	73.8

Source: Company reports

Market opportunity and Competitive landscape

Until recently, treatments for IPF patients were limited to oxygen therapy, pulmonary rehabilitation, lung transplantation, and palliative care. In 2011, the EC approved Roche's Esbriet (pirfenidone), an oral drug with an unknown mechanism of action that has anti-fibrotic, anti-inflammatory, and antioxidant properties. In October 2014, the FDA simultaneously approved both Esbiret and Boehringer Ingelheim's Ofev (nintedanib) for IPF. Ofev is a small molecule that inhibits multiple receptor tyrosine kinases (FGFR, PDGFR, and VEGFR) that may be involved in the thickening or scarring of lung tissue in IPF patients. Ofev received EU approval in January 2015. Both drugs carry a list price of approximately $95,000/year.

Nevertheless, both drugs have limited efficacy, and the prognosis associated with the condition remains poor. Moreover, both drugs carry significant side-effects, primarily nausea and rash with Esbriet, and diarrhea and liver function abnormalities with Ofev. Due to these adverse events, discontinuation rates for both remain high (~25%). Given these shortcomings, we believe an attractive opportunity remains for novel IPF treatments that can demonstrate enhanced safety and better patient outcomes.

1H17 Esbriet sales reached CHF 314 million in the U.S. (up 19% y/y) and CHF 88 million in Europe (up 6% y/y). Worldwide sales were CHF 418 million (up 16% y/y). OFEV also reported strong growth in FY16 – net sales were up by 106% to €250 million. We estimate the IPF market will grow to $5 billion by 2025.

FibroGen's FG-3019 is a fully human mAb that targets connective tissue growth factor (CTGF), a key regulator of fibrosis in animal models. Preclinical data generated with FG-3019 appear remarkable, with significant reversal of fibrosis seen in lung tissue after short treatment exposures. These results have been followed with small clinical trials that also suggest lung fibrosis is being reversed in certain patients. In an exploratory open-label P2 trial in IPF patients, 35% of patients treated with FG-3019 showed stable or improved lung fibrosis at 48 weeks, as measured by HRCT. Furthermore, at both 24- and 48-week time points, improvements in lung fibrosis correlated with improvements in lung function. Based on these data, FibroGen is conducting a randomized placebo controlled P2 trial in IPF with 103 patients randomized (1:1) to receive either pamrevlumab or placebo for 48 weeks.

On Aug. 7th, FibroGen announced positive topline data from the Phase 2 trial with Pamrevlumab for IPF, which contains a main placebo –controlled study, and two combo sub-studies with Pamrevlumab in combination with FDA approved IPF drugs, either pirfenidone or nintedanib. The main efficacy result was a reduction in FVC in treated patients of 2.85% (129 mL absolute drop) vs. 7.17% in placebo treated patients (308 mL absolute drop). The data for deaths (3 vs. 6) and hospitalizations (5 vs. 7) were both in favor of the treatment arm in a patient population with an FVC at baseline of about 70% of normal (considered moderate IPF). Bases on an extrapolated 52-week period (actual trial was 48 weeks), Pamrevlumab achieved a relative difference of 195 mL in FVC decline over placebo, which is clearly better that the 105 mL to 120 mL demonstrated in the pivotal trials for the two approved drugs. So, if these results are repeated in the pivotal Phase 3 trial, Pamrevlumab could be an important and valuable drug. This value is probably further underscored by Roche's acquisition of Intermune (pirfenidone's developer) for $8.3 billion (3.5x FibroGen's current valuation) after the drug reached about a $35 million quarterly sales rate. The company reported no detailed safety data other than to say that serious AEs favored the treatment group 7 to 3. Safety is potentially an important advantage for Pamrevlumab as existing IPF drugs are not particularly safe (pirfenidone has a liver toxicity and GI issues). In addition to the main trial, FibroGen reported data from two safety trials of Pamrevlumab in combination with the two currently marketed drugs, pirfenidone or nintedanib, and commented only that there were no surprises during the 24 weeks of these trials (and combined efficacy data were not collected).

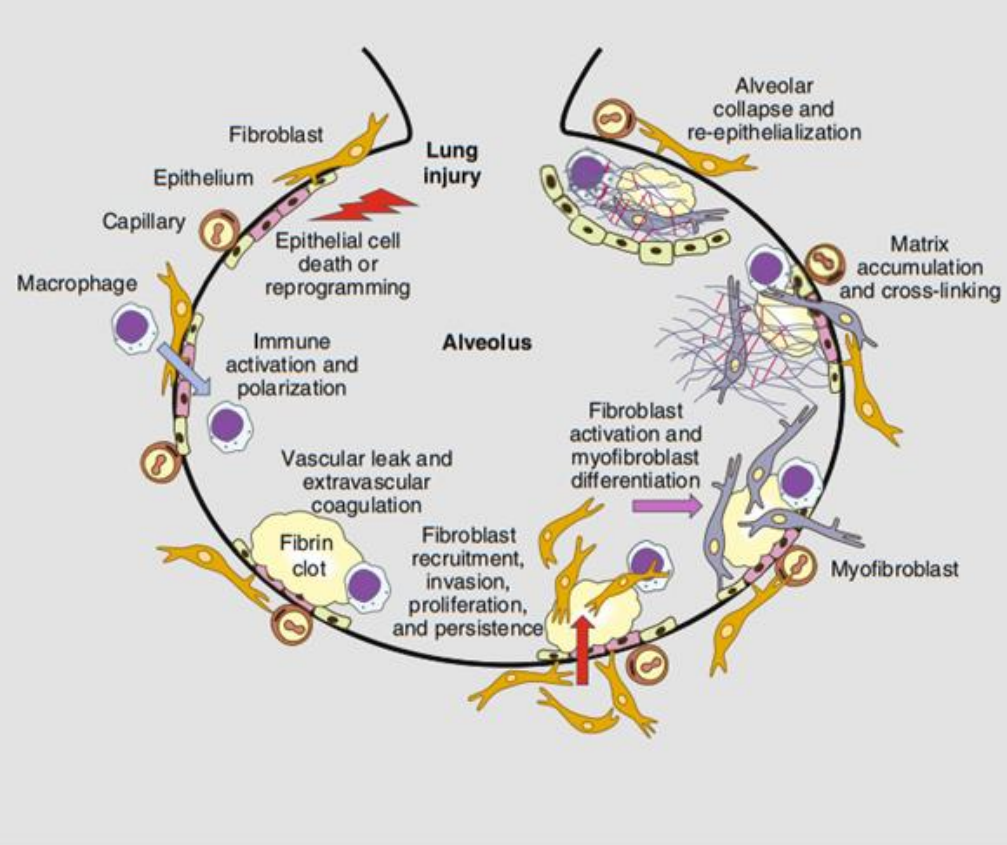
Idiopathic Pulmonary Fibrosis

IPF is a progressive, irreversible, chronic, incurable disease of the lungs of unknown cause characterized by tissue scarring (fibrosis). Current IPF models invoke a pathway in which genetically susceptible individuals undergo aberrant wound healing response as a result of repetitive alveolar insult. In turn, this alveolar epithelial cell injury drives fibroblast proliferation, myofibroblast differentiation, and finally collagen deposition. The resulting matrix of collagen and other extracellular proteins destroys the delicate alveolar structure required for efficient gas exchange in the lung. As the lung tissue thickens, less oxygen can pass from the alveoli into the capillary blood vessels that surround them, and the lungs become stiff with lower filling capacity. Over time, vital organs become oxygen starved. Unchecked, this process leads to progressive lung scarring, breathing inefficiency and death.

Pathogenesis of IPF

The clinical course with IPF is variable. Most patients gradually deteriorate, although some progress rapidly with episodes of acute exacerbations. Inevitably, the progressive lung scarring leads to death. The median survival for IPF patients is two to five years post-diagnosis, and the five-year survival rate is just 20% to 40%, which is worse than for many common cancers.

IPF typically occurs in adults over 50 years of age and affects more men than women. Risk factors include male gender, older age, smoking, exposure to certain pollutants, certain viruses, GERD, diabetes, cancer treatments (i.e., chest radiation, certain chemotherapies), and genetic factors. Symptoms include dry cough, shortness of breath, weight loss, severe fatigue, and clubbing of extremities. Over time, IPF can severely limit physical activity, self-care, and overall quality of life.



Source: American Thoracic Society

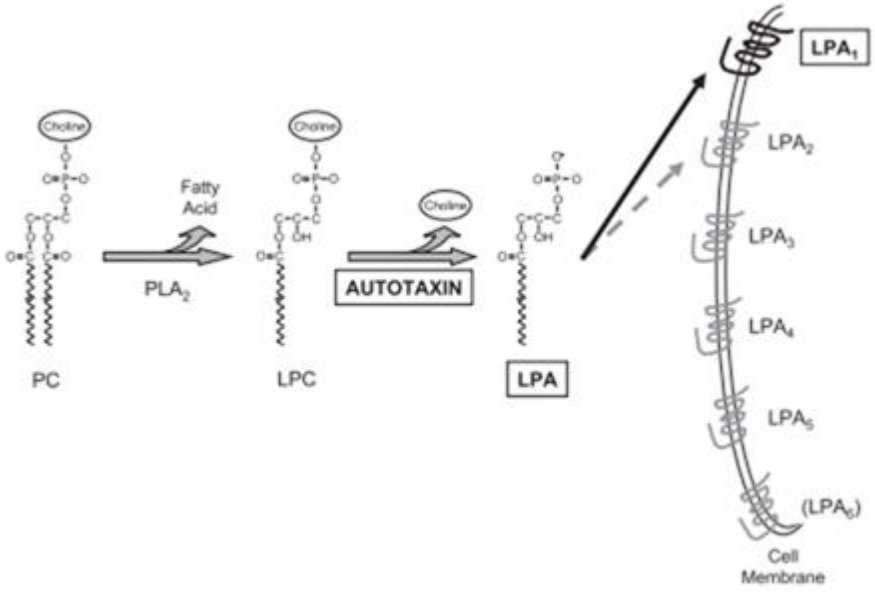
Diagnosis is based on the exclusion of known causes of interstitial lung diseases and presence of a typical pattern on high resolution computed tomography (HRCT) and/or on surgical biopsy. In IPF patients, both diagnostic methodologies show a characteristic pattern known as usual interstitial pneumonia (UIP).

Currently available pharmacologic treatments for IPF include the U.S./EU approved anti-fibrotic drugs Esbriet and Ofev. While these drugs have been shown to reduce the rate of loss of lung function, they do not reverse or cure the disease. Non-pharmacologic treatments include oxygen therapy, pulmonary rehabilitation, and lung transplant.

Autotaxin: A promising new target for IPF

Autotaxin (NPP2 or ENPP2) is an extracelluar enzyme responsible for the hydrolysis of LPC (lysophosphatidycholine) to the bioactive lipid signaling molecule LPA (lysophosphatidic acid). As shown in **Exhibit 3**, LPA acts through six specific cell surface G protein-coupled receptors, LPA1 to LPA6, to control a range of cell activities, including migration, contraction, and survival. LPA signaling has been shown to have pro-fibrotic effects on epithelial cells, endothelial cells, and fibroblasts. In the lung, locally produced LPA promotes epithelial cell apoptosis, induces vascular leak, and directs fibroblast recruitment, proliferation, and persistence. These processes are thought to drive the pathologic effects seen in IPF, including excessive production of extracellular matrix in the interstitial space, permanent scarring of the lung, and decline in lung function.

Exhibit 3: Autotaxin−lysophophatidic acid (ATX−LPA) signaling pathway



Source: Tager, Andrew M. "Autotaxin emerges as a therapeutic target for idiopathic pulmonary fibrosis: limiting fibrosis by limiting lysophosphatidic acid synthesis." American journal of respiratory cell and molecular biology 47.5 (2012): 563-565.

In a 2012 study published in the American Journal of Respiratory Cell and Molecular Biology by Oikonomou, et al., it was demonstrated that pulmonary autotaxin expression contributes to the pathogenesis of pulmonary fibrosis. Specifically, these investigators demonstrated genetic deletion or pharmacologic inhibition of autotaxin limited the development of lung fibrosis in the bleomycin mouse model, including reductions in lung collagen, bronchoalveolar lavage (BAL) cell counts, BAL total protein, as well as BAL levels of both LPA and TGF-β. Moreover, they also demonstrated increased levels of autotaxin in the lung tissue of IPF patients. In other studies, LPA levels have been shown to be increased in bronchoalveolar lavage fluid (BLAF) and in exhaled breath condensate of IPF patients. Taken together, these studies provided a sound rationale for the development of an autotaxin inhibitor for IPF, such as GLPG1690. Based on our review of the science, we believe Autotaxin inhibition with GLPG1690 holds the potential to broadly reduce LPA production and its downstream pro-fibrotic effects in the lungs of IPF patients.

While autotaxin appears to produce the majority of LPA in vivo, several other sources are known to exist. Therefore, a key question is whether the inhibition of autotaxin alone will be sufficient to reduce LPA to levels that slow or halt the progression of lung fibrosis.

Target Price Methodology/Risks

We arrive at our 12-month target price of $101 using a discounted cash flow (WACC 10%, terminal growth 1.5%). We probability-adjust our revenue projections for individual product candidates to reflect clinical, developmental, and regulatory risks. We use a 10% WACC, which is in line with industry peers, to reflect inherent risk in biotechnology drug development. Our 1.5% terminal growth rate reflects drug patent expirations, partially offset by assumed new drug approvals to sustain steady-state CF.

Risks include: development, clinical, regulatory, manufacturing, commercial, competitive, financing, political, and volatility inherent the sector.

Company Description

Galapagos is a clinical-stage biotechnology company specialized in the discovery and development of disease modifying, small molecule medicines with novel mechanisms of action. The pipeline includes clinical candidates focused on rheumatoid arthritis, inflammatory bowel disease, cystic fibrosis, idiopathic pulmonary fibrosis, osteoarthritis, and atopic dermatitis. Lead assets include filgotinib (partnered with Gilead) and a suite of CF potentiators and correctors (partnered with AbbVie). Multiple late stage trials are underway with filgotinib in RA and IBD, with results expected between mid-2018 and 2H19. The CF assets are progressing through multiple P1 and P2 trials, with the goal of launching a triple combo P2 trial around YE17, with results expected in mid-18. The Galapagos group, including fee-for-service subsidiary Fidelta, has approximately 460 employees, operating from its Mechelen, Belgium headquarters and facilities in The Netherlands, France and Croatia.

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Income Statement

Currency Converter as of 8/10/2017
Euro (€) to USD ($) 1.17
USD ($)to Euro (€) 0.85

(€ 000s, except per share data)	POS	FY 2014A	FY 2015A	Mar 1Q16A	Jun 2Q16A	Sep 3Q16A	Dec 4Q16A	FY 2016A	Mar 1Q17A	Jun 2Q17A	Sep 3Q17E	Dec 4Q17E	FY 2017E	FY 2018E	FY 2019E	FY 2020E	FY 2021E	FY 2022E	FY 2023E	FY 2024E	FY 2025E
Rheumatoid Arthritis (Filgotinib)	70%															8,868	44,084	105,540	178,729	225,116	251,566
Crohn's disease (Filgotinib)	50%															4,131	33,075	55,549	82,479	134,083	161,179
Ulcerative colitis (Filgotinib)	25%															893	7,163	12,373	18,878	31,569	38,921
Psoriatic arthritis (Filgotinib)	25%																633	3,149	7,539	12,766	16,080
Ankylosing spondylitis (Filgotinib)	25%															1,177	3,784	6,743	10,442	16,124	19,499
Cystic fibrosis (Triple)	15%																10,102	34,753	49,532	50,616	54,749
IPF (Autotaxin)	10%															15,647	29,292	43,589	58,562	74,237	90,639
Upfront/milestone pmts/cost reimbursements		90,021	60,579	14,817	33,947	16,276	86,572	151,612	39,863	33,168	13,317	13,317	**53,267**	**78,183**	**130,267**	**170,942**	**22,500**	**117,300**	-	-	**87,000**
Total Revenue €		**€ 90,021**	**€ 60,579**	**€ 14,817**	**€ 33,947**	**€ 16,276**	**€ 86,572**	**€ 151,612**	**€ 39,863**	**€ 33,168**	**€ 13,317**	**€ 13,317**	**€ 99,664**	**€ 78,183**	**€ 130,267**	**€ 201,658**	**€ 150,633**	**€ 378,996**	**€ 406,162**	**€ 544,512**	**€ 719,633**
Total Revenue $		**$109,583**	**$66,134**	**$16,774**	**$37,724**	**$18,297**	**$91,033**	**$163,826**	**$42,644**	**$38,807**	**$15,581**	**$15,581**	**$116,607**	**$91,475**	**$152,412**	**$235,940**	**$176,241**	**$443,425**	**$475,210**	**$637,079**	**$841,971**
COGS		-	-	-	-		-	-	-	-	-	-	-	-	-	1,565	2,929	4,359	5,856	7,424	9,064
Gross profit		90,021	60,579	14,817	33,947	16,276	86,572	151,612	39,863	33,168	13,317	13,317	99,664	78,183	130,267	200,094	147,704	374,637	400,306	537,088	710,569
R&D		111,110	129,714	27,818	34,594	34,327	42,834	139,573	44,930	47,983	45,312	54,541	192,766	223,971	157,294	142,375	106,782	110,480	119,319	125,285	131,549
SG&A		14,867	20,308	4,394	6,308	6,081	6,746	23,529	6,159	6,861	6,800	7,083	26,903	28,787	33,105	46,346	47,737	49,646	51,632	53,697	55,845
Income from co-promotion activities																744	6,175	16,933	39,405	62,501	85,500
Restructuring & integration costs		669	-	-	-	-	-														
Operating income (loss) €		**(€ 36,624)**	**(€ 89,444)**	**(€ 17,395)**	**(€ 6,955)**	**(€ 24,132)**	**€ 36,992**	**€ 11,491**	**(€ 11,226)**	**(€ 21,676)**	**(€ 38,795)**	**(€ 48,308)**	**(€ 120,005)**	**(€ 174,574)**	**(€ 60,132)**	**€ 12,116**	**(€ 639)**	**€ 231,444**	**€ 268,760**	**€ 420,607**	**€ 608,676**
Operating income (loss) $		**($44,582)**	**($97,646)**	**($19,692)**	**($7,729)**	**($27,128)**	**$38,898**	**($15,651)**	**($12,009)**	**($25,361)**	**($45,390)**	**($56,520)**	**($140,405)**	**($204,251)**	**($70,355)**	**$14,175**	**($748)**	**$270,789**	**$314,449**	**$492,110**	**$712,151**
Fair value share of subscription agreement		-	(30,632)	57,479	-	-	-	57,479	-	-	-	-	-	-	-	-	-	-	-	-	-
Financial income		2,291	1,987	626	1,455	561	7,308	9,950	894	1,425	816	787	3,921	3,017	2,566	2,494	1,989	2,334	2,696	3,622	5,581
Financial expense		(867)	(1,539)	(4,761)	1,755	(494)	1,808	(1,692)	(3,274)	(15,298)	-	-	(18,573)	-	-	-	-	-	-	-	-
Net income (loss) before taxes		**(35,201)**	**(119,627)**	**35,950**	**(3,745)**	**(24,065)**	**46,108**	**54,246**	**(13,606)**	**(35,549)**	**(37,979)**	**(47,521)**	**(€ 134,656)**	**(171,557)**	**(57,566)**	**14,609**	**1,349**	**233,778**	**271,456**	**424,228**	**614,257**
Income tax provision		2,103	(1,218)	-	24	(95)	(164)	(235)		(92)			-	-	-	-	-	15,897	18,459	28,848	41,769
Net income (loss) from continuing operations €		**(€ 37,303)**	**(€ 118,410)**	**€ 35,950**	**(€ 3,721)**	**(€ 24,160)**	**€ 45,944**	**€ 54,012**	**(€ 13,605)**	**(€ 35,642)**	**(€ 37,979)**	**(€ 47,521)**	**(€ 134,747)**	**(€ 171,557)**	**(€ 57,566)**	**€ 14,609**	**€ 1,349**	**€ 217,881**	**€ 252,997**	**€ 395,381**	**€ 572,487**
Net income (loss) from continuing operations$		**($45,409)**	**($129,268)**	**$40,697**	**($4,135)**	**($27,159)**	**$48,311**	**$57,714**	**($14,554)**	**($41,701)**	**($44,436)**	**($55,600)**	**($157,654)**	**($200,721)**	**($67,352)**	**$17,093**	**$1,579**	**$254,921**	**$296,007**	**$462,596**	**$669,810**
Net income from discontinued operations		70,514	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Translation differences, other				(382)	(191)	(362)	935	-	31	-	-	-	**31**	-	-	-	-	-	-	-	-
Total comprehensive income (loss) to owners of the parent €		**33,211**	**(118,410)**	**35,567**	**(3,912)**	**(24,522)**	**46,879**	**54,012**	**(13,574)**	**(35,642)**	**(37,979)**	**(47,521)**	**(134,716)**	**(171,557)**	**(57,566)**	**14,609**	**1,349**	**217,881**	**252,997**	**395,381**	**572,487**
EPS - continuing operations €		**(€ 1.24)**	**(€ 3.32)**	**€ 0.79**	**(€ 0.08)**	**(€ 0.53)**	**€ 0.96**	**€ 1.14**	**(€ 0.29)**	**(€ 0.74)**	**(€ 0.79)**	**(€ 0.99)**	**(€ 2.83)**	**(€ 3.50)**	**(€ 1.14)**	**€ 0.26**	**€ 0.02**	**€ 3.62**	**€ 4.01**	**€ 5.96**	**€ 8.22**
EPS - continuing operations $		**($1.51)**	**($3.62)**	**$0.89**	**($0.09)**	**($0.60)**	**$1.01**	**$1.22**	**($0.31)**	**($0.87)**	**($0.92)**	**($1.15)**	**($3.31)**	**($4.09)**	**($1.33)**	**$0.31**	**$0.03**	**$4.24**	**$4.69**	**$6.98**	**$9.62**
Shares outstanding		30,108	35,700	45,492	45,229	45,527	47,823	47,308	46,256	48,043	48,091	48,139	47,632	49,061	50,533	55,602	57,270	60,133	63,140	66,297	69,612

Source: Stifel estimates and reported company data

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Galapagos NV (GLPG) as of August 10, 2017 (in USD)

*Represents the value(s) that changed.
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