STIFEL

Galapagos NV

GLPG – NASDAQ; GLPG – NA

September 14, 2017

Biotechnology

BUY

COMPANY UPDATE

Positive KOL Feedback on GLPG1690 for IPF; Full Transcript Below; TP to $120

Financial Summary

Changes	Previous	Current
Rating	—	Buy
Target Price	$101.00	$120.00
FY17E EPS	—	€(2.83)
FY18E EPS	—	€(3.50)
FY17E Revenue	—	€99.7
FY18E Revenue	—	€78.2

Price (09/14/17):	$103.47
52-Week Range:	€104 - $54
Market Cap.(mm):	4,786.1
Shr.O/S-Diluted (mm):	46.3
Avg Daily Vol (3 Mo):	211,040
Dividend / Yield:	$0.00 / 0.0%

Revenue	2016A	2017E	2018E
Q1	€14.8	€39.9A	€NE
Q2	€33.9	€33.2A	€NE
Q3	€16.3	€13.3	€NE
Q4	€86.6	€13.3	€NE
FY (Dec)	€151.6A	€99.7	€78.2

EPS	2016A	2017E	2018E
Q1	€0.79	€(0.29)A	€NE
Q2	€(0.08)	€(0.74)A	€NE
Q3	€(0.53)	€(0.79)	€NE
Q4	€1.01	€(0.99)	€NE
EPS	€1.14A	€(2.83)	€(3.50)

Price Performance

Summary

We recently held a conference call with two idiopathic pulmonary fibrosis (IPF) KOLs to discuss Galapagos' recent positive P2a FLORA results (full transcript below), one of whom participated in the FLORA study. Our KOLs indicated that while GLPG1690 ('1690) safety and pharmacodynamics were the most important components of FLORA, they also felt that efficacy, while more speculative, "was as good as one could have hoped for." They noted that the concordance between serum LPA reductions, FVC stabilization, and functional respiratory imaging (FRI) parameters suggest that, "at this stage it is as nice a package that one could have hoped to deliver when the study was being designed." As a result of the positive KOL feedback, and consistent with their views, we are increasing our '1690 POS in our model to 15% (10% prior) – which results in our new $120 TP (from prior $101). Reiterate Buy.

Key Points

KOL overall FLORA impressions: According to our KOLs, the goal of the FLORA was to address early POC in IPF, and that explains the design of a small, short study - to get important info on safety, target engagement and more speculatively, efficacy. Neither perfenidone nor nintedanib, the two currently approved IPF products, went through similar, early studies - so it is difficult to compare FLORA data with those drugs. With regards to FLORA results, one KOL commented that, "the FLORA results were as good as one could have hoped - in that sense, it exceeded expectations," and while safety and pharmacodynamics were the important components, to see a signal is as good as one could have hoped for - very exciting and a very compelling rationale for taking the drug forward into larger, longer studies."

On the concordance observed across multiple measures: Serum LPA reductions ("very impressive"), FVC stabilization, and FRI results – "putting all the pieces of the jigsaw together, I think at this stage it is as nice a package that one could have hoped to deliver when the study was being designed."

On '1690 MOA (autotaxin inhibition): "So I am a little more skeptical with just 12 weeks (of data) as compared to longer-range studies, but very hopeful because I think the pathobiology of inhibiting autotaxin in this (disease) makes sense."

On future '1690 trials: "FDA's been quite straightforward about things – in the US, '1690 would be on top of existing therapy and that the only patients that would be on no background therapy would be patients who are unable to tolerate both approved drugs...~20-25% of patients can't tolerate existing therapies."

On potential for '1690 combo with existing therapies: "One of the benefits of this drug is that it doesn't seem to lead to the same sort of side effects of either of the existing therapies, so it fits neatly on top of both of them, arguably."

On quantifying '1690 ultimate approval chances: "10% to 20%, if not hopefully better, based on FLORA data and stage of development. I think for this stage of development, one couldn't hope for a better data package, but clearly, there are plenty of other things that could cause a hiccup between now and approval."

We are increasing '1690 POS to 15% from prior 10%: Based on what we view as strong KOL feedback on the FLORA study and the potential for '1690 in IPF, we are modestly increasing our POS. As a result, our TP is revised upward to $120, from $101 previously. We forecast FY25 unadjusted WW sales of $1.07B ($136M probability-adjusted at 15% 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 15% 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.

On FLORA imbalances in baseline characteristics: "I don't actually think any of those things are that important there's been a lot of work done using existing clinical trial sets to look for specific clinical factors that predict patients who are going to progress more rapidly or more slowly based on FVC - and actually none of the physiological measures, nor gender, nor duration of IPF have any impact on how patients' FVC progresses over time. So in that sense, the imbalance both in physiology and gender shouldn't actually have any impact on the anticipated rate of FVC decline over the three months of the study – and there's published data from the INPULSIS study that will very clearly show the lack of relationship between any of these factors and disease change over time."

On missing patient data in FLORA: "This issue of missing patient data has come up repeatedly in IPF clinical trials. At the end of the P3 studies, both nintedanib and perfenidone were missing about 20% of endpoint data. So in that sense, it's part of the course and it's less missing (in FLORA) than was encountered in those trials. I agree, it does introduce challenges but I think the fact that there were serial measurements mitigates, to a certain extent the challenges of missing those."

On functional respiratory imaging (FRI) data from FLORA: "The CT data is less well validated as an endpoint in IPF. It's sort of a very attractive endpoint to look at change in imaging markers but the reality is the tools are less well validated for use in clinical trials – but nonetheless, it gives a signal that supports the FVC signal as well, which again, adds a little bit of reassurance that we're not just looking at statistical noise."

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.

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.

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 next 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)

Stifel Biotech Analysts - Tom Shrader (FGEN - Buy) and Adam Walsh (GLPG – Buy)
Physicians – Dr. Toby Maher (Imperial College, London) and Dr. John Belperio (UCLA)
Topic: Emerging drugs for IPF: FGEN's pamrevlumab and GLPG's GLPG1690

DR. MAHER: I'm a clinician and an academic research scientist. I work at the Royal Brompton Hospital in London and Imperial College, London. So from a clinical point of view, together with four colleagues, I run a dedicated Pulmonary Fibrosis service where, at the moment, we see about 1,000 to 1,500 new IPF patients each year and I follow up about 3,000 patients with the disease. So we're by far, one of the biggest referral units in Europe. I've been using treatments with the current anti-fibrotic drugs; Pirfenidone since 2011 and Nintedanib since 2014.

And then from a trials perspective, I run a big trials unit focused on Fibrotic Lung Disease and have been involved with most of the landmark studies in the last decade and I'm also involved in running early proof of concept studies in Fibrotic Lung Disease.

MR. SHRADER: I just wanted to spend maybe two to three minutes on the disease. So I think we're about to get small data sets with anecdotally or nearly anecdotal and very positive subsets. If you can give a sense; of your patients, is the disease relatively uniform? Are there still problems with diagnosis? And I guess the net is; how impressive is it for a patient to get better? Is that expected in the noise or is that truly unique if a patient shows a measurable increase and how big an increase would you really have to see to say; oh my goodness? Just a little bit on the disease?

DR. MAHER: So yeah, so first of all, untreated, it's a disease that gets eventually worse over three to four years with a median life expectancy being about three years from diagnosis. So it's a fairly deadly condition. I think, as with all diseases, there is a spectrum of sort of severity, with some patients going from diagnosis to death in a matter of months. Other patients having a slower form of the disease and often living up to ten years or so. That said, we have become much better at diagnosing an IPF over the last 15 to 20 years.

So the landmark studies for both approved drugs - but I think it's been one of the things that driven back to uniformity in achieving a diagnosis and better understanding around the strengths and weaknesses of current diagnostic tools. So I think current trials now contain a much more homogenous group of patients than they would have done 10-15 years ago.

As to the question of getting better, again, I think if we look at the published Phase 3 trials with Nintedanib and Pirfenidone, we do see a smaller proportion of patients -- I would say, just to recap the data, both drugs show a 50% reduction in rates of decline in disease over 12 months. But when you dig into the data with both drugs, about a 5th or a quarter of patients are stable over the 12 month treatment period and a smaller proportion, perhaps 5%, show subtle improvements in lung function.

So it's not beyond the realm of possibility, based on what we know from existing anti-fibrotic drugs, but it's certainly not been the norm from treatment.

MR. SHRADER: Dr. Maher, if I could just jump in here. Could you talk a little bit about the two current approved drugs? How good are they? Is their use warranted? And maybe some differences in the treatment paradigm using those drugs in the US versus the EU?

DR. MAHER: Yeah, so again, in the EU, we've had longer access to the drug. So we had perfenidone approved in 2011. It was only approved in late 2014 in the US. So we've got longer treatment experience. Just to give you my bias, I am much more upbeat than a lot of other pulmonologists. I think both drugs represent a massive step change for disease that hitherto has been uniformly fatal with a median survival of three years and to me, halving the rates declined is an enormous step forward. It's important, proof of concept, that the fibrosis is a treatable disease.

Both drugs have their challenges in so much as upwards of a quarter of patients do struggle to tolerate them due to side effects and of course, there remain treatment failures. But I think overall, if you sort of compare to similar field, like oncology or pulmonary hypertension where the death rates and survival are similar, it's very unusual to see such a sort of magnitude of treatments effect as we did with the existing anti-fibrotic drug. So I think they are important drugs.

Clearly, there are challenges. Clearly, the risk will improve things further. I think, based on modeling work that we published recently, patients who can tolerate both drugs might expect a two year improvement in life expectancy but that still means their life expectancy is 10 to 12 years shorter than an individual of the same age. So there is still a huge way to go before we really turn IPF into a chronic treatable disease as opposed to a progressive fatal disease.

MR. SHRADER: So Doctor, over the course of your practice, do you think you sense an impact to these drugs? I think

talking to Dr. Velperio, he would have said no but do you sense that people are around longer?

DR. MAHER: Yeah, for whatever reason, pulmonologists are pessimists and -- yeah, I know that both companies have had challenges, getting doctors to prescribe drugs, but I think we are gradually seeing the emergence of real world data that is demonstrating that the patients who do stay on drugs live longer. So there was an Australian registry paper published four months ago that showed unequivocally that patients who were on treatment had a much better life expectancy than untreated patients in the same country, enrolled into the database at the same time.

So for me, it's easy to see amongst my colleagues that leads them to think that drugs will show such a huge magnitude of effect in clinical trials are ineffective in clinical practice. I think it's just a lack of understanding around the needs of patients with a fatal disease and perhaps who have a sort of challenge that we have a lack of clinical measures of effectiveness. Essentially, you've got to follow someone on treatment for two to three years before you can decide the treatment is working for that individual. We don't have any short term measures of effectiveness but -- in clinical practice, it's something of an act of faith when it comes to treatment.

MR. SHRADER: Interesting and then just to finish up sort of the background section. You mentioned a quarter of the patients have significant side effects. What are the worst side effects and what side effects can't the two new drugs add to in order to be useful in combination? What's the worst thing you can see in the new drugs from a side effect profile?

DR. MAHER: Yeah, so I think with Pirfenidone, it's really around tolerability. I think with all the data we have now, we know the drug is actually safe and fortunately, we've never seen dangerous side effects. The two issues from a Pirfenidone point of view; one is a fairly sensitive rash. So about 10% of patients will get a rash going out into the sun but for the majority, that can be modified by; wearing a hat, wearing sun block. The one that tends to be more troublesome is gastrointestinal upset.

So the patient will often feel nauseous and they lose their appetites, they lose weight and that side effect accounts for the majority of people who discontinued the drug prematurely. It's certainly one that one would want to avoid with any new medication. And then with Nintedanib, the principle problem is diarrhea and it's not diarrhea like an oncology drug. So this is not people sort of sitting on the toilet all day. The biggest problem or the reason the patient stopped treatment with Nintedanib is they get diarrhea without warning.

So they'll be out of the house and then they have to go to the toilet. So when they have to go, they really have to go and that, of course, creates challenges for people trying to live a normal life. So again, I think diarrhea and the gastrointestinal upsets are the two side effects to avoid.

MR. SHRADER: Okay, perfect, and then Adam, do you want to dig into the Galapagos data?

MR. WALSH: Yeah, absolutely. So Dr. Maher, one of the reasons we were excited to have you on today's call is because you were quoted in the Galapagos Press Release and it seems you have some knowledge, perhaps some deep knowledge, of the Flora trial. So maybe you could start just by framing up the Flora study for us and give us your overall interpretation of the results and then I have some granular questions to follow.

DR. MAHER: Yeah, so I was involved with the Flora study but obviously I will stick to discussing the publically available data.

MR. WALSH: Of course.

DR. MAHER: So I think -- again, just the history of IPF drug development. The majority, both Pirfenidone and Nintendanib, actually didn't go through early phase studies in IPF. Essentially in both cases (BI and Intermune) as it were gambled by going to late phase studies and large numbers of patients and eventually InterMune with Pirfenidone went straight to phase 3.

So what we've hitherto lacked is a sort of development -- a model of the appropriate development pipeline for IPF and we've lacked a good model of a proof of concept study. So essentially with the Flora study, that is trying to address that sort of need. How do we conduct a study in a small number of patients to give us both important information on safety, important information on target engagement and perhaps, more speculatively, information on efficacy.

So the goal of the Flora study was to try and develop a study that would allow sort of quick answer on the safety and pharmacodynamics of the drugs while it's also being long enough to provide some information on efficacy. So that's sort of the genesis to the study design. And essentially, it was a three month study -- sorry.

MR. WALSH: And then when you saw the results, at least, the way you were quoted in the press release, it appears that you saw something exciting there. Can you talk about your interpretation of what you saw?

DR. MAHER: Yeah, so I think, so having told you the study design, I think really the results were as good as one could ever have hoped and I suppose in that sense, it exceeded expectation because the safety and the pharmacodynamics was the important components. The efficacy was a small number of patients and was therefore a more speculative component to the study.

So to actually see -- perhaps robust is not the right word in a study of the size of the Flora study, but to see a signal as has been seen, to me, is as good as one could have hoped for and therefore, very exciting and a very compelling rationale for taking the drug forward into larger, longer studies.

MR. WALSH: Some of the questions that investors were asking the company on the conference call, one of them was about the minor imbalances and baseline characteristics and I understand from what you're saying, this is a small proof of concept study but we're all trying to interpret the results the best we can. Some of those included in the 1690 arm, there appears to have been more males than females, fewer smokers, and a slightly higher baseline FVC. But then alternatively, also in the 1690 arm in patients who had a longer duration of IPF at baseline, an average of 1.9 years versus just one year for placebo. How should we interpret these imbalances as we assess the data?

DR. MAHER: So again, given what was being measured is rate of FVC change over the three month change in false vital capacity, I don't actually think any of those things are that important. There's been a lot of work done using the existing clinical trial sets to look for specific clinical factors that predict patients who are going to progress more rapidly or more slowly based on the FCC and actually none of the physiological measures nor gender nor duration of IPF have any impact on how patients FCC progresses over time.

So in that sense, the imbalance both in physiology and gender shouldn't actually have any impact in the anticipated rate of false vital capacity decline over the three months of the study and there's published data from the INPLUSIS study that will very clearly show the lack of relationship between any of these factors and disease change over time.

MR. WALSH: So the magnitude of 1690 to FVC benefit slightly decline from weeks 4 to 8 and then from weeks 8 to 12. Is there anything to read into there?

DR. MAHER: So again, I think once you account for measurement noise, it becomes a little bit hard to try and break down each individual time point and say whether the gaps between placebo and treatment is more important or less important. I think for me it's important that the trend was sustained at each time point and for the duration of the study but I wouldn't want to dig into it further. Again, clearly, the caveats that one has to put on this is an early phase study with a small number of patients but as I say, beyond that, the data is very, very compelling.

MR. WALSH: Some patients were missing from the FVC analyses either due to poor quality measurement or if a patient had taken a bronchodilator too close to the FVC measurement. Does that impact your thinking about the results in any way?

DR. MAHER: Again, this issue of missing data is one that has come up repeatedly in IPF clinical trials. At the end of the Phase 3 studies, show that Nintedanib and Pirfenidone were missing about 20% endpoint data. So in that sense, it's part of the course and it's less missing than what was encountered in those trials. I agree, it does introduce challenges but I think the fact that there were serial measurements mitigates, to a certain extent, the challenge of missing those.

MR. WALSH: Do you think also -- go ahead, please.

DR. MAHER: I was going to say that sort of the second degree component of the CT data, as well. Again, the CT data is less well validated as an endpoint in IPF. It's sort of a very attractive endpoint to look at change in imaging markers but the reality is, the tools are less well validated for use in clinical trials but nonetheless, it gives a signal that supports the FCC signal, as well, which again, adds a little bit of reassurance that we're not just looking at statistical noise.

MR. WALSH: Can you talk a little bit more about functional respiratory imaging? I know you mentioned it's not validated. What is the strength of the current validation in terms of views amongst your colleagues? Is this something that is continuing to emerge and at what point can we put a lot of stock into that measurement?

DR. MAHER: Yeah, so on its own, I wouldn't put too much stock into it. Again, so CT imaging, the cross sectional imaging

of the lung is a very -- it's essentially the tool that we use for diagnosis of IPF and clearly, over time, one can recognize progression in fibrosis based on CT imaging. The challenge that we've had with that is how you quantify change and sort of very experienced human observers can detect change but there has to be quite a lot of change before they can reliably detect it and you get above the sort of measurement noise.

So there's been a drive in the last four or five years to develop computer algorithms that will eventually try and determine change and the functional respiratory imaging is one of four or five algorithms that have been developed and so we are at the point where the pros and cons of each of the imaging techniques is beginning to be understood and the strengths and limitations are beginning to be understood.

But I don't think we can honestly say we fully understand how noisy the measurement is, how robust specific changes over time are, and so on its own, I wouldn't -- it wouldn't give me confidence of a positive drug effect but I think, as a supported piece of evidence taken together with the false vital capacity, then it does add some reassurance that both measures of change are pointing in the same direction, if that makes sense.

MR. WALSH: So is it safe to say that the concordance on serum LPA reductions, FVC stabilization and FRI results, that the concordance between all those pointing in the same direction is something that allowed you to say that this is potentially a drug that can be very meaningful in this disease? It's not one component but when everything is pointing in the same way, that gives you more confidence. Is that safe to say?

DR. MAHER: Yes and I think the LPA data on its own -- and again, when sort of talking through what would have been the green lights when designing this study and thinking about what the important components were to sort of make a decision to move forward. I think the LPA data, which shows clear almost a dynamic engagement by the drug, is in itself very compelling. A very impressive knockdown of the LPA levels with therapy.

So as you say, putting all the pieces of the jigsaw together, I think at this stage, it's as nice a package that one could have hoped to deliver when the study was being designed.

MR. WALSH: Terrific. Tom, I just have a couple more quick ones and then I'll turn the floor completely over to you. Dr. Maher, what do you think the design of the next trial or trials should be, what do you think regulators are thinking about in terms of a mono-therapy trial or on top of current drugs; how do you think that's going to play out?

DR. MAHER: So my understanding of the FDA view is that they think quite straight forward about things. Another thing that's run in the US would be on top of existing therapy and that the only patients that would be on no background therapy were patients who were unable to tolerate both approved drugs. I think there are territories, including my own, where there are limitations on reimbursement.

So for instance in the United Kingdom, patients with a well preserved full-throttle capacity can't get access to either Pirfenidone or Nintedanib and so it is possible to recruit patients on no background therapy into trial. So if I was designing it, I would be looking at something that mirrored the studies conducted for nintedanib - if I was doing a Phase 2B study, I would probably design a six month round and a 12 month study to get clearly a rebound and I would anticipate that probably 80% of patients would be on background therapy and I would be trying to balance those patients to ensure that one could see if there were any important interactions with either existing treatment. I think one of the --

DR. MAHER: I was just going to say that one of the benefits of this drug, also, is that it doesn't seem to lead to the same sort of side effects as either of the existing therapies. So it fits neatly on top both of them, arguably.

MR. WALSH: And my final question is, just give us a general sense, how you think the 1690 data stack up to those of other drugs in a similar stage of development. You mentioned there were no short term studies done but you can look at the Phase 3 data and kind of extrapolate to the three month time point. So that would be first, you impression there and then -- go ahead with that one and then I'll ask my final one.

DR. MAHER: Yes, so I always thought that three months was enough for an efficacy signal based on the previous two trials because you do see early separation of the FVC curves with both Pirfenidone and Nintedanib. So I think the challenge at three months is knowing how that magnitude of effect compares and I wouldn't want to try and draw too many conclusions on such a small number but I think -- I had always hoped that it would be possible to show effect at three months. The fact that it has been shown is reassuring. Beyond that, probably, I wouldn't want to say much more.

MR. WALSH: Final question, I'm going to ask you to put your financial analyst hat on here. Based on what you know, what do you think the probability of success of a drug that has shown these data is -- what do you think the probability of

success is that it will get ultimately to market based on what you know now? Would 10% be reasonable, 15, 25%?

DR. MAHER: I think -- clearly, if one looks at drug development and sort of the success rate for drugs that successfully get past 1B or 2A, I think there's as good a chance as any. So it's at least average for that sort of drug. So somewhere between 10 and 20% if not hopefully better but I think it -- as I said, I think for this stage of development, one couldn't hope for a better data package but clearly, there are plenty of other things that could cause a (ind) between now and approval.

MR. WALSH: Terrific, Tom -- thanks so much, Dr. Maher. Tom, the floor is yours.

MR. SHRADER: Thank you, Adam. Is Doctor Velperio on the line?

DR. VELPERIO: Yes, I'm on the line.

MR. SHRADER: Okay, very good, you've been very quiet. So have you been following, do you know where we are?

DR. VELPERIO: Yeah, absolutely.

MR. SHRADER: So maybe we can, really for both doctors -- maybe Doctor Verperio, you can start. How familiar are you with the FibroGen trial? I would just be curious on both of your senses of how exciting the data are and maybe, just based on the little bit we know, because we know much less about FibroGen, whether the data are as exciting, they could be as exciting, whether the data are not as exciting. Just your basic sense of how exciting what we know about the FibroGen drug, Pamrevlumab is. So Dr. Velperio, why don't you start?

DR. VELPERIO: Yeah, I think for the FibroGen data, that's an anti-body, essentially, against connective tissue growth factor, which has been known for a long time to be involved in fibroprolifative pathways. It's definitely downstream of TGF Beta, which is considered one of the master regulators of fibrosis. The problem with inhibiting TGF Beta, it's tough to inhibit, just the way it interacts with the matrix. Very difficult for antibodies to be able to truly neutralize the activity.

So here, you're going for something downstream with TGF Beta. TGF Beta, again, a master regulator. It's supposedly one of the mechanisms by which Pirfenidone, also known as Esbriet works and their trial, just from what's out there in the public domain, it looks like it's right up there from their results. Very similar data to Nintendanib, also known as Ofev as well as Pirfenidone also known as Esbriet.

You're seeing the same kind of reductions and fall and FVC as compared to those two trials from the data that's out there. The difference being, the other two drugs are pill form. One is a pill twice a day, Nintedanib. That's why a lot of people like that drug a bit better, versus Pirfenidone, which you have to take three times a day. This is a drug that's infusion therapy, it's done at least in the clinical trial, every three weeks you have your infusion. And side effects, at least from what's out there, do not seem to be very significant.

MR. SHRADER: Okay, and Dr. Maher, do you have familiarity with this trial? Your sense of this package? Hello?

DR. VELPERIO: If you look at the decline in the anti-TGF Beta group was about 150 CC's versus 300 CC decline in the patients that were in the control group. So this drug is showing very similar to other drugs. Most likely -- and with regard to other drugs in the US, I think they're being used a lot now.

I mean, I think early on, there was a lot of skepticism but they are two FDA approved drugs. People with Pulmonary Fibrosis want it whether they have mild disease, moderate disease or severe disease. So anybody who walks into our clinic with IPF are being put on these drugs. The problem is, expectations of patients are still very high that these drugs are going to actually make them feel better but we are very explicit with them up front that, you are not going to feel better with this drug.

This drug is not going to improve your cough, it's not going to improve your (ind) exertion. It does a theoretical benefit that we're going to slow the decline in your FVC. So we're very up front and that's what the two FDA approved drugs do and that is what seems to be the effect that we're going to be seeing with connective tissue growth factor blockade, very similar. Question is, will insurances allow combination therapy and I think that anti-connective tissue growth factor will be very good for combination therapy or as used as a standalone drug.

I think when the first two FDA drugs were approved, there were problems with regards to diarrhea with Ofev, Nintendanib, as well as the nausea with the Pirfenidone Esbriet group but we really don't see that because we don't titrate the drugs up very fast. We do a slow titration and really side effects that we're seeing are pretty much nil. We are in California where

we're at, so we do see a little bit more photosensitivity but as long as they use their sun block, they're okay from that perspective.

But realistically, the use of the drug, either one of them, is very easy as long as it's slow titration. All of the problems are in the first three months. So if you're slow on the first three months, you avoid all those problems and usually, the patients are okay. The biggest problem is, with the current form of drug that we have today, we don't even have stability with these drugs.

These patients are continuing to decline in front of our eyes, albeit at a slower rate, the problem is, it's not that much of a slower rate and we are already seeing some of the frustrations between doctors as well as patients that these drugs are not, quote un-quote, really working. So we do have a honeymoon period here. Of course, they're new drugs, it's an exciting time for a pulmonary fibrosis but I think there is some frustrations.

As our patients come into the office, we can't look at them and say, we're going to really be prolonging your quality of life or really prolonging even your survival for a significant period of time. So we don't hesitate on getting these patients on transplant lists as well as preparing them for potential demise. I mean, 5-15% of these patients will have an exacerbation annually, which really leads to a significant mortality, especially if they require a hospitalization. Usually 70% of those that have an exacerbation and wind up in the hospital, 70% are dead within 3 to 12 months after that exacerbation.

MR. SHRADER: Okay, Dr. Maher, did we lose you?

DR. MAHER: You did lose me for a while but I'm back.

MR. SHRADER: Okay, are you familiar at all with the FibroGen data set? Your basic sense of what you saw and how it might stack up to Galapagos based on limited data?

DR. MAHER: So I don't have a huge insight into that data set, I've only seen what's in the press release. Again, I think they have the advantage of it being a longer study, it's got more patients in it. So arguably, from that point of view, one can have slightly more confidence in the statistical robustness of the data seen. I think the FibroGen drug (ind) has its own challenges. It's a biologic, it's administered at a very high dose.

I'm not certain we fully understand the dosing rationale yet. It's an intravenous infusion every three weeks, which makes it less convenient than an orally dosed molecule. So there are some down sides to the strategy and I think still some unanswered questions around how we (ind) genuinely target CTGF and which of the CTGF components, because there are lots of splashing variants, are the most important to go after.

MR. SHRADER: So you both commented on the IV nature of this drug. Is this particularly tough for this population? This is a pretty frail population.

DR. VELPERIO: You do lose a day every time you need an infusion, right, because you have to get to the infusion center, get your infusion, be monitored for a bit and then go home. So you do lose a day every three weeks. So I think that is definitely somewhat problematic versus doing your two to three pills a day where you really don't lose any time. So I think that is problematic.

MR. SHRADER: Okay and then I guess the same question that Dr. Maher answered. Do you expect Phase 3 would have to be a combination study in the US? Could you find patients that were Pirfenidone intolerant or do you think it almost has to be a combination study to get the patients you need in a reasonable time?

DR. VELPERIO: In the US, it's FDA approved for everyone with IPF's. Everyone with an IPF wants one or the other drug. So I think it's going to be a combination study for one or the other drug. Either one as background therapy and I think everybody will be on background therapy probably for three months before being placed on a combination with something like CTGF antibodies and that would have to be the study design.

The question is how low, how severe you would allow the disease to be to accrue patients very quickly to get the study done because if you look at the background therapies, it really seems that across the board, be it early, mild, moderate or severe disease, somewhat same effect with regard to these drugs. So it's going to be how aggressive they are and how quickly they want to do the trial to get patients in. But if you take patients with DLCO's 25% or above, I think the trial can be done very quickly.

MR. SHRADER: Okay and then last question before we open for audience questions. At the ERS meeting, we may see

HRCT data from maybe individual patients. Do you expect that to be very important, very compelling? I think, Dr. Maher, you spoke to that a little bit. Just your level of how definitive those data will be to see changes in those measurements for individual patients?

MR. VELPERIO: I think it's going to be helpful. I think it's an endpoint. Obviously, your primary endpoint is still going to have to be the FVC. I think that has always been for interstitial lung diseases, the ultimate endpoint besides mortality but I think it will be incredibly supportive to see changes on high resolution CAT scan at the pixel level.

MR. SHRADER: Dr. Maher, do you have?

DR. MAHER: I think I would concur with that viewpoint.

MR. SHRADER: Okay. So a handful of patients with high resolution data. Okay Adam, do you have anything or do you want to open it for questions? Operator, maybe we can see if we have audience questions?

MR. WALSH: So Operator, thanks for doing that. While we wait for the queue to fill up, I do have an e-mail question here and doctors, can you talk about what percentage of patients cannot tolerate the two currently approved drugs and what percentage can be immediately addressable -- will be the immediate addressable market for the two drugs from Galapagos and FibroGen. Maybe we'll start with Dr. Maher?

DR. MAHER: So I think in our practice, it's been about a quarter of patients who don't tolerate therapy. I think the question is really what the role of any future treatment would be, whether it would be failed patients or whether, as I would anticipate, it would be add-on therapy. I think Dr. Velperio has said, we still see patients inevitably getting worse on existing treatments.

So there is scope to try and get further anti-fibrotic effect by combining treatments and I think actually the advantage of both the FibroGen and the Galapagos drug is that they have distinct mechanisms of action that are different, both Nintedanib and Pirfenidone, and in that sense, they would both make-good add-on therapies to existing treatment.

MR. SHRADER: A related question. Do you believe the existing drugs are anti-fibrotic?

DR. MAHER: That's an interesting question. In the sense that they slow the progression of a measure that one can consider to be influenced primarily by fibrosis, then I would argue yes. If one wants to get into the biological semantics of exactly how they do that, then there is a school of thought that maybe they're having actions on things like epithelial cells but I'm not sure that's necessarily important ultimately when what we're doing is preventing the disease from getting worse.

MR. SHRADER: Yep, yep. Okay, Operator, do we have questions?

OPERATOR: There are no questions in queue.

MR. WALSH: I have another client e-mail question. At the upcoming ERS meeting, Celgene has some data on their JNK inhibitor that looks pretty interesting, per the abstract. Are you familiar with the Celgene data and also other drugs that may be coming down the pipeline and can you talk a little bit about the emerging competitive landscape for IPF?

DR. VELPERIO: I don't know anything about the Celgene data. I mean, those pathways are all kind of anti-inflammatory, anti-fibroproliferative. They all have a potential to work but the question is, which one is truly going to work? At least, which one is going to cause more stability if not reversal of the disease? That's going to be the question.

MR. WALSH: Dr. Maher, any view on the Celgene product or anything else?

DR. MAHER: No, I think based on the -- I don't know the study and I only know what was in the abstract from the ERS and again, that data looks interesting and certainly something to dig into in a bit more detail. IPF remains a pretty active field. There are other studies on game with integrin inhibitors, so JSK and Biogen have got Alpha B, Beta 6, different strategies for blocking Alpha B, Beta 6. Galecto, a small Danish biotech presented data at the ATS on the galectin 3 inhibitor.

There's still the anti-IL 13 approaches, they were still waiting data from Roche and Sanofi on their anti-IL 13 drugs and there are several other companies with sort of very early phase programs looking at a range of very different strategies in IPF. So it remains a very active field of interest for the pharmaceutical industry.

MR. SHRADER: Are you aware of the Prometic program? That's the question I have.

DR. MAHER: From a confidential perspective, yes, but nothing I can say out loud.

MR. SHRADER: Okay, well, it's made your radar screen. Adam?

MR. WALSH: Yeah, thanks, Tom. I have a couple more from clients. One of them wants to know your views on the odds of success for FibroGen's IPF drug reaching the market?

DR. VELPERIO: I think based off of what they're putting out there, it seems to be right in line with the other drugs and I think, if they do a combination therapy, obviously and we get FDA approved, then combination therapy -- I think a lot of patients will want this drug in combination with the two FDA approved drugs currently. I think unfortunately, this is a pretty open market. There are only two drugs for it. The two drugs are pretty equivocal from my perspective in using them.

So I think anything that gets FDA approved, especially as add-on and then as insurance companies will allow add-on therapy, I think you can really kind of get a niche in this market.

MR. WALSH: Dr. Maher?

DR. MAHER: So, probability of success. Again, not having seen the data, it's a bit hard to answer robustly on that one. I think one can work out the odds based on its progression down the drug development pipeline and I would just say that it's mitigated by challenges around manufacturing the drugs and the dosing regimen and the route required, which possibly offsets some of my expectations of success. So if you say it's 40% on average for a drug that's (ind), I might go a little bit lower than that.

MR. WALSH: And then I have another one from a client. Dr. Velperio, they specifically want to know your impression of Autotaxin inhibition?

DR. VELPERIO: I think based off of the data that's out there and previously out there in the animal studies as well as human data, I think Autotaxin has multiple functions be it on inflammatory components, on T-cell components, all IL-13 as well as on fibroblasts. I think it's very exciting. So I think biologically, it makes sense.

The only problem with that is, the length of the study has been short and I agree, we've seen separation of curves early out as 12 weeks. But just based off of other studies where you have these early data, this really makes you worry about more long-term data and I think we've pushed the threshold back from 72 weeks to 52 weeks with these trials but I think you're going to want to see something. You still have to see something at least one year on these drugs.
So I'm a little more skeptical with just 12 weeks as compared to longer-range studies but very hopeful because I think the pathobiology of inhibiting Autotaxin in this pathobiology makes sense.

MR. WALSH: I have another -- Tom, do you have any? Operator, are there any in the queue?

MR. SHRADER: I guess the only question I have is -- and it's pretty vague. Do you have any sense of whether either mechanism makes more sense in combination with the existing drugs? Is there enough data around to even handicap -- relative to a question like that.

DR. VELPERIO: For me, to me, just more from a basic science view, I love the concept of CTGF. That's just -- for so many years, it's been implicated in all these fibrotic processes; liver, kidney, lung and there's just been an enormous amount of animal molecular data to say its importance. It's kind of nice to see some of that importance actually come to human trials and make an impact but that's just from my perspective.

MR. SHRADER: Okay.

MR. WALSH: So another client question real quick; are either of you familiar with the BMS Autotaxin inhibitor? It seems it's been out there for a while but not a lot of updates on data.

DR. VELPERIO: Yeah, I mean --

DR. MAHER: Yeah, so I have no direct -- sorry, sorry.

MR. SHRADER: Okay, no worries.

DR. VELPERIO: Yeah, they did a trial there and I thought that possibly -- yeah, I don't know what happened to that trial. I thought that it was stopped but never seen any data from that trial.

DR. MAHER: I'm told that if you dig into the annual reports, you can find that it was stopped for safety, I think, but that's my only knowledge.

MR. WALSH: That's right. Okay, good. Operator, any more questions in the queue?

OPERATOR: There are no questions in queue.

MR. WALSH: Tom, any questions from you?

MR. SHRADER: I'm good, I'm good. Thank you, everybody, enjoy your Friday afternoon.

MR. WALSH: Yeah, thank you so much for joining both of the doctors, Dr, Maher, Dr. Velperio, really appreciate it. On behalf of Stifel, thanks everyone for joining and I apologize again for the difficulty with the dial-in. Glad we made it through the call, have a great weekend.

DR. VELPERIO: Thank you guys, too.

DR. MAHER: All right, thanks all, bye.

OPERATOR: Thank you, ladies and gentlemen, this does conclude today's conference call. You may disconnect your phone lines at this time and have a wonderful day. Thank you for your participation.

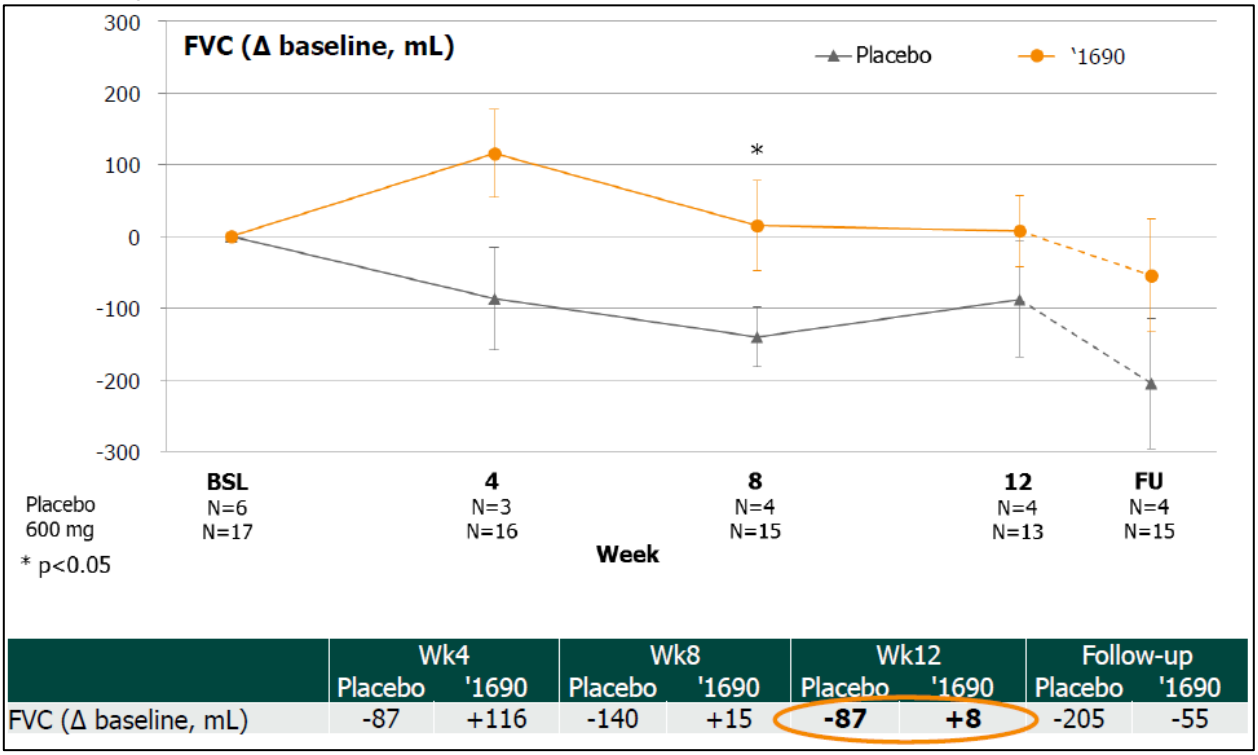
END

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.

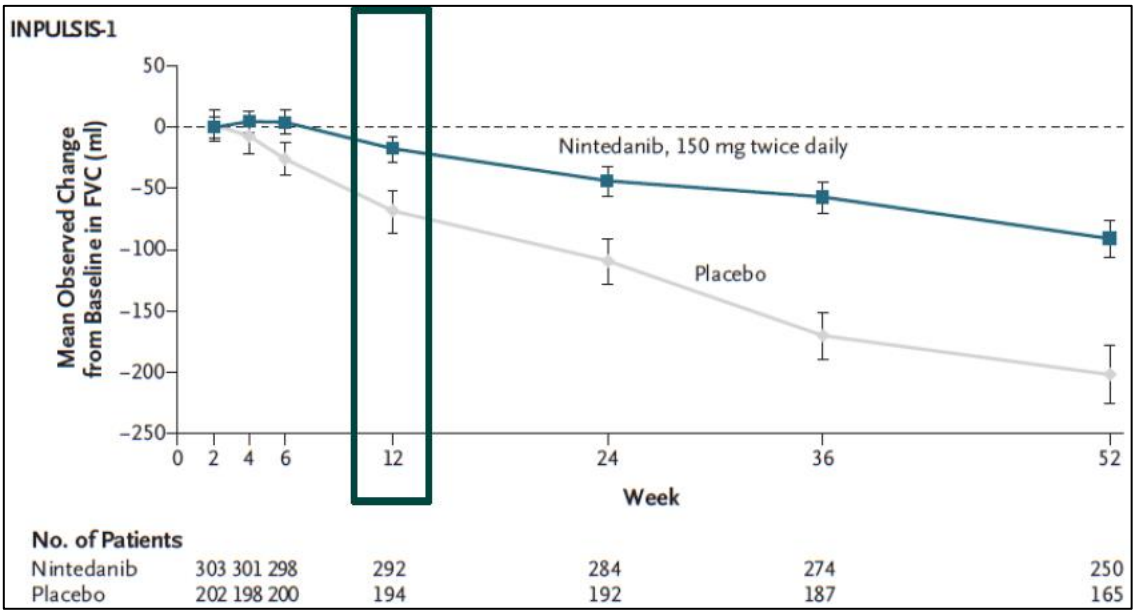
Exhibit 1: FVC data at week 12: GLPG1690 vs. Nintedanib vs. Pirfenidone

GLPG1690, Phase 2

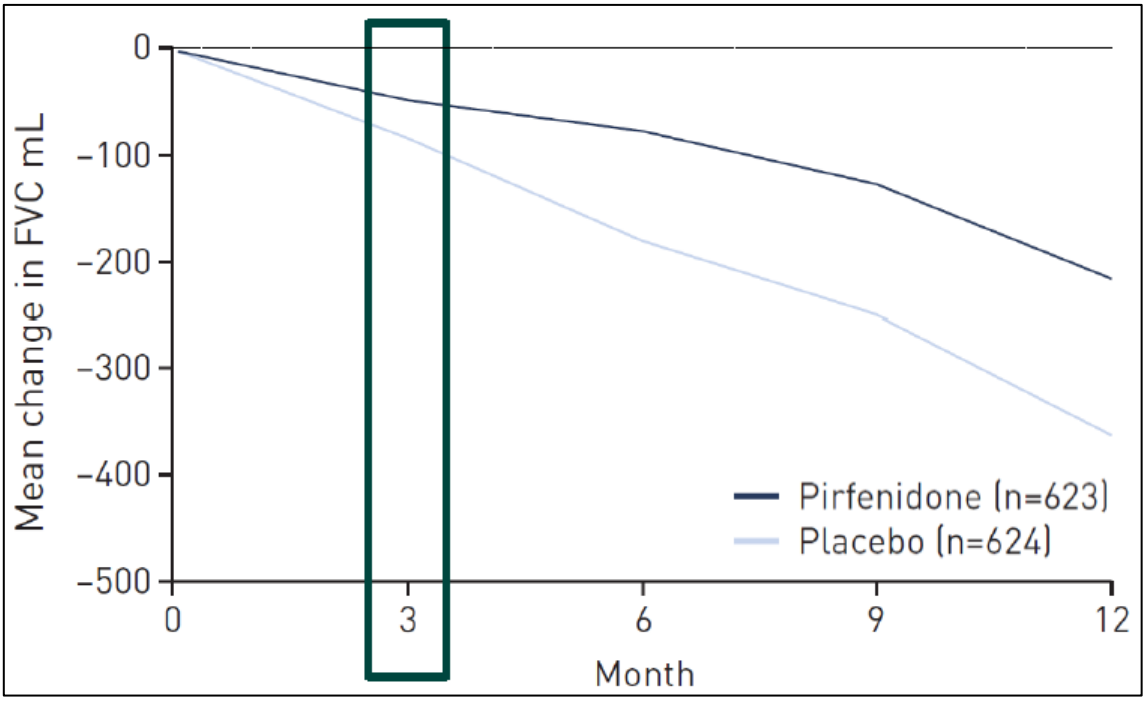


	Wk4		Wk8		Wk12		Follow-up	
	Placebo	'1690	Placebo	'1690	Placebo	'1690	Placebo	'1690
FVC (Δ baseline, mL)	-87	+116	-140	+15	**-87**	**+8**	-205	-55

Nintedanib, Phase 3



Pirfendione, Phase 3



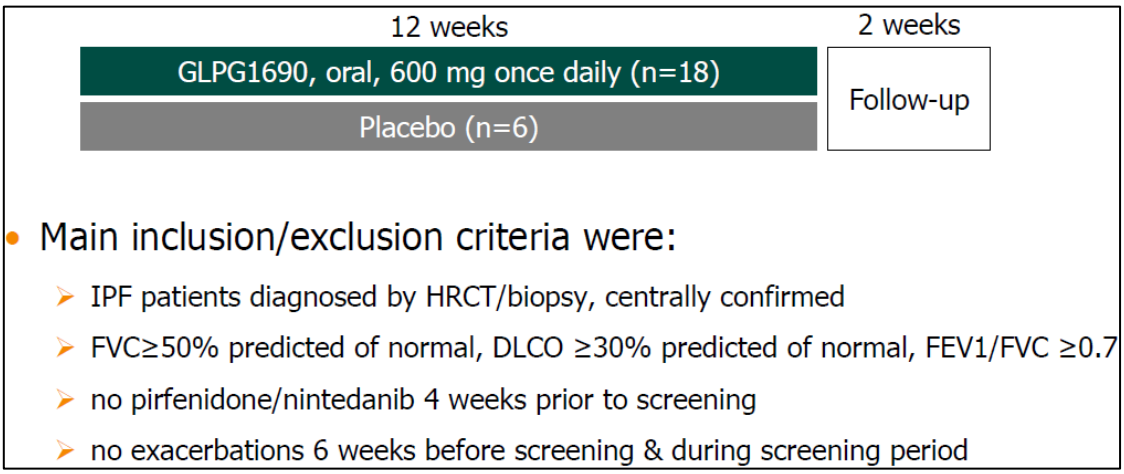
Source: Company reports

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).

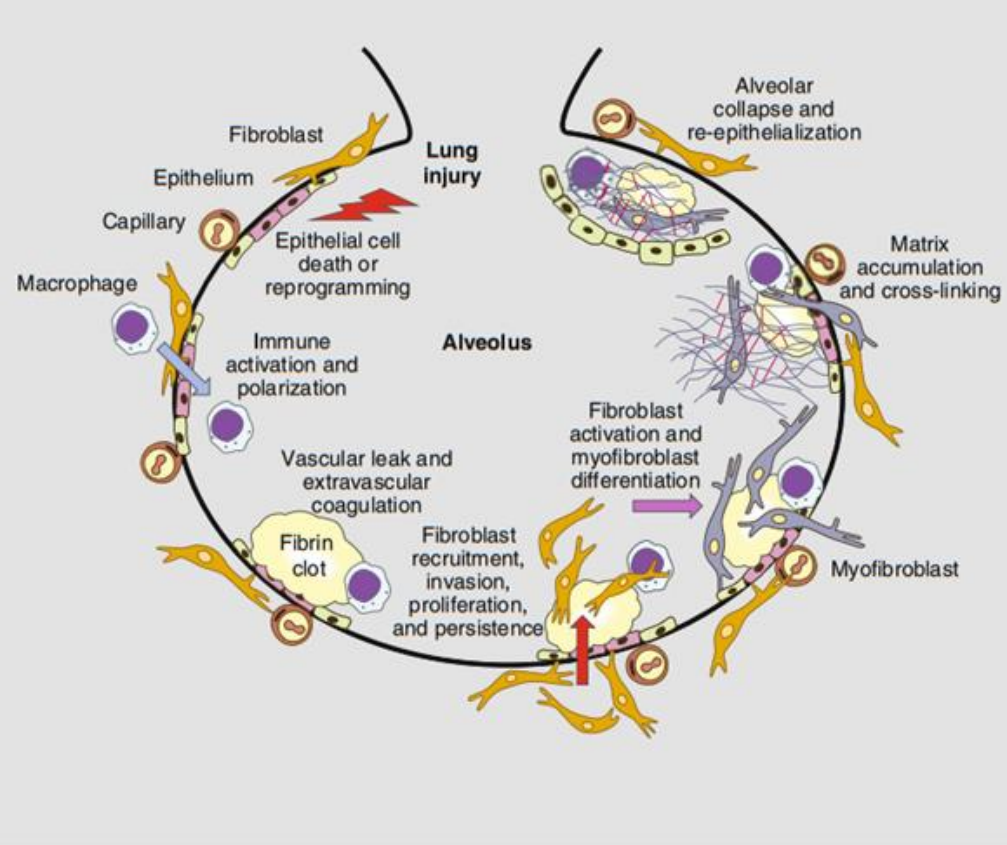
Idiopathic Pulmonary Fibrosis

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

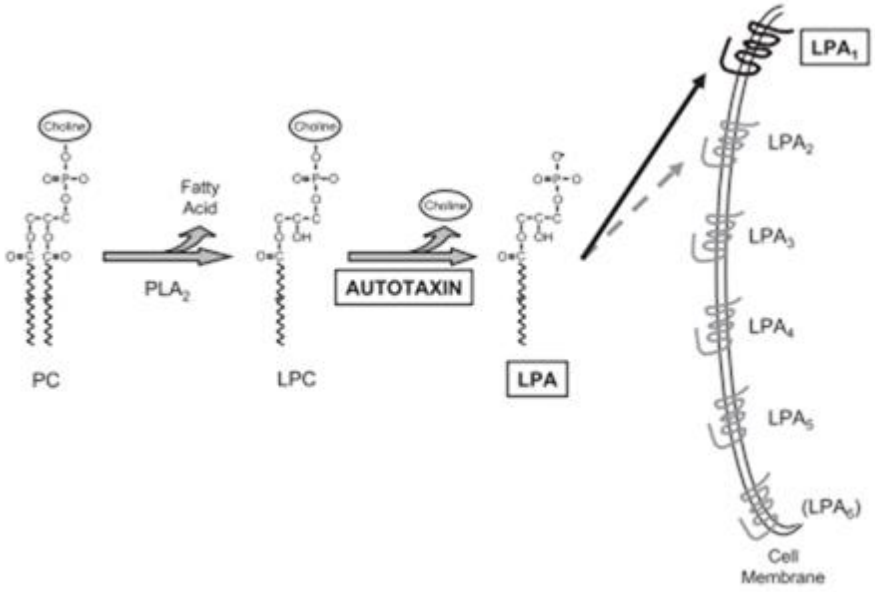
Diagnosis is based on the exclusion of known causes of interstitial lung diseases and presence of a typical pattern on high resolution computed tomography (HRCT) and/or on surgical biopsy. In IPF patients, both diagnostic methodologies show a characteristic pattern known as usual interstitial pneumonia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

Exhibit 4: DCF analysis

DCF valuation

(€ thousands)	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E	2026E	2027E	2028E	2029E	2030E	2031E	2032E	2033E	2034E
EBIT	(120,005)	(174,574)	(60,132)	19,157	12,542	251,059	295,113	454,014	649,463	737,833	767,420	938,305	950,816	1,008,121	845,390	716,938	667,170	628,642
EBIT x (1-t)	(120,005)	(174,574)	(60,132)	19,157	12,542	233,987	275,045	423,141	605,300	687,660	715,236	874,500	886,160	939,569	787,904	668,187	621,803	585,895
+ D&A	4,399	5,963	6,097	6,366	6,677	6,998	7,319	7,637	7,951	8,263	8,572	8,879	9,185	9,489	9,793	10,096	10,398	10,700
- Capex	(7,046)	(6,846)	(7,146)	(7,446)	(7,746)	(8,046)	(8,346)	(8,646)	(8,946)	(9,246)	(9,546)	(9,846)	(10,146)	(10,446)	(10,746)	(11,046)	(11,346)	(11,646)
- Change of WC	(51,257)	28,042	(19,876)	70,798	(27,645)	17,153	(50,148)	(50,149)	36,850	(50,152)	(153)	86,846	(50,155)	(50,157)	(50,158)	(50,159)	(50,161)	(50,162)
FCF	(71,394)	(203,499)	(41,305)	(52,721)	39,118	215,785	324,166	472,280	567,455	736,829	714,415	786,687	935,355	988,769	837,109	717,396	671,015	635,110
Terminal value	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	7,119,629

(Thousands except per share)

Discount rate	10%
Terminal Growth	1.5%
PV of FCF - Forecast	2,257,176
PV of FCF Terminal	1,304,083
Discounted cash flow	**3,561,259**
+ Net Debt	1,253,793
Equity Value	**4,815,052**
Share count	47,632
Value/Share (€)	**€ 101**

Euro to USD	1.19
Value/Share	**$120**

Terminal growth \ Discount Rate	7%	8%	9%	10%	11%	12%	13%
0.0%	$175	$150	$131	$116	$104	$94	$85
0.5%	$180	$154	$133	$118	$105	$94	$86
1.0%	$186	$157	$136	$119	$106	$95	$86
1.5%	$192	$161	$139	**$121**	$107	$96	$87
2.0%	$200	$166	$142	$123	$109	$97	$88
2.5%	$209	$172	$146	$126	$111	$99	$89
3.0%	$221	$179	$150	$129	$112	$100	$90

Source: Company reports, Stifel estimates

Target Price Methodology/Risks

We arrive at our 12-month target price of $120 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.